



Policy Assessment for the Reconsideration of the Ozone National Ambient Air Quality Standards

External Review Draft

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Policy Assessment for the Reconsideration of the
Ozone National Ambient Air Quality Standards
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U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
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1 INTRODUCTION

This document, *Policy Assessment for the Reconsideration of the Ozone National Ambient Air Quality Standards, External Review Draft* (hereafter referred to as the draft PA), presents the draft policy assessment for the U.S. Environmental Protection Agency’s (EPA’s) reconsideration of the decision reached in the review of the ozone (O₃) national ambient air quality standards (NAAQS) completed in 2020.^{1, 2} This draft PA considers the key policy-relevant issues, drawing on those identified in the *Integrated Review Plan for the Ozone National Ambient Air Quality Standards* (IRP; [U.S. EPA, 2019]) in light of the available evidence assessed in the *Integrated Science Assessment for Ozone and Related Photochemical Oxidants* (ISA [U.S. EPA, 2020a]) and quantitative air quality, exposure and risk analyses based on that evidence, including any analyses updated for this reconsideration. Thus, this document will reassess the policy implications of the scientific evidence described in the 2020 ISA and related air quality, exposure and risk analyses. Accordingly, this document draws heavily on information presented in the 2020 PA (U.S. EPA, 2020b), with some updates to include more recent air quality information.

This document is organized into four chapters. Chapter 1 presents introductory information on the purpose of the PA in the context of NAAQS reviews, legislative requirements for NAAQS reviews, an overview of the history of the O₃ NAAQS, including background information on prior reviews, and a summary of the process for this reconsideration. Chapter 2 provides an overview of how photochemical oxidants, including O₃, are formed in the atmosphere, along with updated information on sources and emissions of important precursor chemicals, as well as updated ambient air monitoring data. Chapter 2 also summarizes key aspects of the ambient air monitoring requirements, and O₃ air quality, including model-based estimates of O₃ resulting from natural sources and anthropogenic sources outside the U.S. Chapters 3 focuses on policy-relevant aspects of the health effects evidence (as presented in the 2020 ISA) and exposure/risk information, identifying and summarizing key considerations related to review of the primary (health-based) standard. Similarly, Chapter 4 focuses on policy-

¹ The scope for this reconsideration, as for the 2020 decision on the O₃ NAAQS, focuses on the presence in ambient air of photochemical oxidants, a group of gaseous compounds of which ozone (the indicator for the current standards) is the most prevalent in the atmosphere and the one for which there is a very large, well-established evidence base of its health and welfare effects. The ozone standards that were established in 2015 (80 FR 65292, October 26, 2015) and retained in 2020 (85 FR 87256, December 31, 2020), are referred to in this document as the “current” or “existing” standards.

² On October 29, 2021, the Agency announced its decision to reconsider the 2020 O₃ NAAQS final action. This announcement is available at <https://www.epa.gov/ground-level-ozone-pollution/epa-reconsider-previous-administrations-decision-retain-2015-ozone>.

1 relevant aspects of the welfare effects evidence (as presented in the 2020 ISA) and air quality,
2 exposure and risk information, identifying and summarizing key considerations related to review
3 of the secondary (welfare -based) standard.

4 **1.1 PURPOSE**

5 Generally in each NAAQS review, the PA, when final, presents an evaluation, for
6 consideration by the EPA Administrator, of the policy implications of the available scientific
7 information, assessed in the ISA, any quantitative air quality, exposure or risk analyses based on
8 the ISA findings, and related limitations and uncertainties. Ultimately, a final decision on the
9 NAAQS will reflect the judgments of the Administrator. The role of the PA is to help “bridge the
10 gap” between the Agency’s scientific assessment and quantitative technical analyses, and the
11 judgments required of the Administrator in determining whether it is appropriate to retain or
12 revise the NAAQS.

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

18 The development of the PA is also intended to facilitate advice to the Agency and
19 recommendations to the Administrator from an independent scientific review committee, the
20 Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act
21 (CAA). The EPA generally makes available to the CASAC and the public one or more drafts of
22 the PA for CASAC review and public comment. As discussed below in section 1.2, the CASAC
23 is to advise on subjects including the Agency’s assessment of the relevant scientific information
24 and on the adequacy of the current standards, and to make recommendations as to any revisions
25 of the standards that may be appropriate. In its review of the draft PA, the CASAC also conveys
26 its advice on the standards.

27 In this draft PA for the reconsideration of the December 2020 O₃ NAAQS decision, we⁴
28 take into account the scientific evidence, as characterized in the 2020 ISA and the additional

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.

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

1 policy-relevant quantitative air quality, exposure and risk analyses described herein. Advice and
2 comments from the CASAC and the public on this draft PA will inform the final evaluation and
3 conclusions in the final PA.

4 The final PA is designed to assist the Administrator in considering the available scientific
5 and risk information and formulating judgments regarding the standards. Accordingly, the final
6 PA will inform the Administrator’s decision in this reconsideration. Beyond informing the
7 Administrator and facilitating the advice and recommendations of the CASAC, the final PA is
8 also intended to be a useful reference to all interested parties. In these roles, it is intended to
9 serve as a source of policy-relevant information that supports the Agency’s reconsideration of
10 the 2020 O₃ NAAQS decision, and it is written to be understandable to a broad audience.

11 **1.2 LEGISLATIVE REQUIREMENTS**

12 Two sections of the CAA govern the establishment and revision of the NAAQS. Section
13 108 (42 U.S.C. 7408) directs the Administrator to identify and list certain air pollutants and then
14 to issue air quality criteria for those pollutants. The Administrator is to list those pollutants
15 “emissions of which, in his judgment, cause or contribute to air pollution which may reasonably
16 be anticipated to endanger public health or welfare”; “the presence of which in the ambient air
17 results from numerous or diverse mobile or stationary sources”; and for which he “plans to issue
18 air quality criteria....” (42 U.S.C. § 7408(a)(1)). Air quality criteria are intended to “accurately
19 reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable
20 effects on public health or welfare which may be expected from the presence of [a] pollutant in
21 the ambient air....” (42 U.S.C. § 7408(a)(2)).

22 Section 109 [42 U.S.C. 7409] directs the Administrator to propose and promulgate
23 “primary” and “secondary” NAAQS for pollutants for which air quality criteria are issued [42
24 U.S.C. § 7409(a)]. Section 109(b)(1) defines primary standards as ones “the attainment and
25 maintenance of which in the judgment of the Administrator, based on such criteria and allowing
26 an adequate margin of safety, are requisite to protect the public health.”⁵ Under section
27 109(b)(2), a secondary standard must “specify a level of air quality the attainment and
28 maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite
29 to protect the public welfare from any known or anticipated adverse effects associated with the
30 presence of [the] pollutant in the ambient air.”⁶

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

1 In setting primary and secondary standards that are “requisite” to protect public health
2 and welfare, respectively, as provided in section 109(b), the EPA’s task is to establish standards
3 that are neither more nor less stringent than necessary. In so doing, the EPA may not consider the
4 costs of implementing the standards. See generally, *Whitman v. American Trucking Ass’ns*, 531
5 U.S. 457, 465-472, 475-76 (2001). Likewise, “[a]ttainability and technological feasibility are not
6 relevant considerations in the promulgation of national ambient air quality standards” (*American
7 Petroleum Institute v. Costle*, 665 F.2d 1176, 1185 [D.C. Cir. 1981], *cert. denied*, 455 U.S. 1034
8 [1982]; *accord Murray Energy Corp. v. EPA*, 936 F.3d 597, 623-24 [D.C. Cir. 2019]). At the
9 same time, courts have clarified the EPA may consider “relative proximity to peak background
10 ... concentrations” as a factor in deciding how to revise the NAAQS in the context of
11 considering standard levels within the range of reasonable values supported by the air quality
12 criteria and judgments of the Administrator (*American Trucking Ass’ns, v. EPA*, 283 F.3d 355,
13 379 [D.C. Cir. 2002], hereafter referred to as “*ATA III*”).

14 The requirement that primary standards provide an adequate margin of safety was
15 intended to address uncertainties associated with inconclusive scientific and technical
16 information available at the time of standard setting. It was also intended to provide a reasonable
17 degree of protection against hazards that research has not yet identified. See *Lead Industries
18 Ass’n v. EPA*, 647 F.2d 1130, 1154 (D.C. Cir 1980), *cert. denied*, 449 U.S. 1042 (1980);
19 *American Petroleum Institute v. Costle*, 665 F.2d at 1186; *Coalition of Battery Recyclers Ass’n v.
20 EPA*, 604 F.3d 613, 617-18 (D.C. Cir. 2010); *Mississippi v. EPA*, 744 F.3d 1334, 1353 (D.C. Cir.
21 2013). Both kinds of uncertainties are components of the risk associated with pollution at levels
22 below those at which human health effects can be said to occur with reasonable scientific
23 certainty. Thus, in selecting primary standards that include an adequate margin of safety, the
24 Administrator is seeking not only to prevent pollution levels that have been demonstrated to be
25 harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm,
26 even if the risk is not precisely identified as to nature or degree. The CAA does not require the
27 Administrator to establish a primary NAAQS at a zero-risk level or at background concentration
28 levels (see *Lead Industries v. EPA*, 647 F.2d at 1156 n.51, *Mississippi v. EPA*, 744 F.3d at 1351),
29 but rather at a level that reduces risk sufficiently so as to protect public health with an adequate
30 margin of safety.

31 In addressing the requirement for an adequate margin of safety, the EPA considers such
32 factors as the nature and severity of the health effects involved, the size of the sensitive
33 population(s), and the kind and degree of uncertainties. The selection of any particular approach

and deterioration of property, and hazards to transportation, as well as effects on economic values and on personal comfort and well-being.”

1 to providing an adequate margin of safety is a policy choice left specifically to the
2 Administrator’s judgment. See *Lead Industries Ass’n v. EPA*, 647 F.2d at 1161-62; *Mississippi v.*
3 *EPA*, 744 F.3d at 1353.

4 Section 109(d)(1) of the Act requires periodic review and, if appropriate, revision of
5 existing air quality criteria to reflect advances in scientific knowledge on the effects of the
6 pollutant on public health and welfare. Under the same provision, the EPA is also to periodically
7 review and, if appropriate, revise the NAAQS, based on the revised air quality criteria.⁷

8 Section 109(d)(2) addresses the appointment and advisory functions of an independent
9 scientific review committee. Section 109(d)(2)(A) requires the Administrator to appoint this
10 committee, which is to be composed of “seven members including at least one member of the
11 National Academy of Sciences, one physician, and one person representing State air pollution
12 control agencies.” Section 109(d)(2)(B) provides that the independent scientific review
13 committee “shall complete a review of the criteria...and the national primary and secondary
14 ambient air quality standards...and shall recommend to the Administrator any new...standards
15 and revisions of existing criteria and standards as may be appropriate....” Since the early 1980s,
16 this independent review function has been performed by the CASAC of the EPA’s Science
17 Advisory Board. A number of other advisory functions are also identified for the committee by
18 section 109(d)(2)(C), which reads:

19 Such committee shall also (i) advise the Administrator of areas in which
20 additional knowledge is required to appraise the adequacy and basis of existing,
21 new, or revised national ambient air quality standards, (ii) describe the research
22 efforts necessary to provide the required information, (iii) advise the
23 Administrator on the relative contribution to air pollution concentrations of
24 natural as well as anthropogenic activity, and (iv) advise the Administrator of any
25 adverse public health, welfare, social, economic, or energy effects which may
26 result from various strategies for attainment and maintenance of such national
27 ambient air quality standards.

28 As previously noted, the Supreme Court has held that section 109(b) “unambiguously bars cost
29 considerations from the NAAQS-setting process” (*Whitman v. American Trucking Ass’ns*, 531
30 U.S. 457, 471 [2001]). Accordingly, while some of the issues listed in section 109(d)(2)(C) as
31 those on which Congress has directed the CASAC to advise the Administrator are ones that are
32 relevant to the standard setting process, others are not. Issues that are not relevant to standard
33 setting may be relevant to implementation of the NAAQS once they are established.⁸

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.”

⁸ Because some of these issues are not relevant to standard setting, 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 the EPA to consider costs of implementation when reviewing and revising the

1 1.3 HISTORY OF THE O₃ NAAQS, REVIEWS AND DECISIONS

2 Primary and secondary NAAQS were first established for photochemical oxidants in
3 1971 (36 FR 8186, April 30, 1971) based on the air quality criteria developed in 1970 (U.S.
4 DHEW, 1970; 35 FR 4768, March 19, 1970). The EPA set both primary and secondary standards
5 at 0.08 parts per million (ppm), as a 1-hour average of total photochemical oxidants, not to be
6 exceeded more than one hour per year based on the scientific information in the 1970 air quality
7 criteria document (AQCD). Since that time, the EPA has reviewed the air quality criteria and
8 standards a number of times, with the most recent review being completed in 2020.

9 The EPA initiated the first periodic review of the NAAQS for photochemical oxidants in
10 1977. Based on the 1978 AQCD (U.S. EPA, 1978), the EPA published proposed revisions to the
11 original NAAQS in 1978 (43 FR 26962, June 22, 1978) and final revisions in 1979 (44 FR 8202,
12 February 8, 1979). At that time, the EPA changed the indicator from photochemical oxidants to
13 O₃, revised the level of the primary and secondary standards from 0.08 to 0.12 ppm and revised
14 the form of both standards from a deterministic (i.e., not to be exceeded more than one hour per
15 year) to a statistical form. With these changes, attainment of the standards was defined to occur
16 when the average number of days per calendar year (across a 3-year period) with maximum
17 hourly average O₃ concentration greater than 0.12 ppm equaled one or less (44 FR 8202,
18 February 8, 1979; 43 FR 26962, June 22, 1978).

19 Following the EPA's decision in the 1979 review, several petitioners sought judicial
20 review. Among those, the city of Houston challenged the Administrator's decision arguing that
21 the standard was arbitrary and capricious because natural O₃ concentrations and other physical
22 phenomena in the Houston area made the standard unattainable in that area. The U.S. Court of
23 Appeals for the District of Columbia Circuit (D.C. Circuit) rejected this argument, holding (as
24 noted in section 1.1 above) that attainability and technological feasibility are not relevant
25 considerations in the promulgation of the NAAQS (*American Petroleum Institute v. Costle*, 665
26 F.2d at 1185). The court also noted that the EPA need not tailor the NAAQS to fit each region or
27 locale, pointing out that Congress was aware of the difficulty in meeting standards in some
28 locations and had addressed this difficulty through various compliance related provisions in the
29 CAA (*id.* at 1184-86).

standards “it would be grounds for vacating the NAAQS” (*Whitman v. American Trucking Ass'ns*, 531 U.S. 457,
471 n.4 [2001]). At the same time, the CAA 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 The next periodic reviews of the criteria and standards for O₃ and other photochemical
2 oxidants began in 1982 and 1983, respectively (47 FR 11561, March 17, 1982; 48 FR 38009,
3 August 22, 1983). The EPA subsequently published the 1986 AQCD (U.S. EPA, 1986) and the
4 1989 Staff Paper (U.S. EPA, 1989). Following publication of the 1986 AQCD, a number of
5 scientific abstracts and articles were published that appeared to be of sufficient importance
6 concerning potential health and welfare effects of O₃ to warrant preparation of a supplement to
7 the 1986 AQCD (U.S. EPA, 1992). In August of 1992, the EPA proposed to retain the existing
8 primary and secondary standards based on the health and welfare effects information contained
9 in the 1986 AQCD and its 1992 Supplement (57 FR 35542, August 10, 1992). In March 1993,
10 the EPA announced its decision to conclude this review by affirming its proposed decision to
11 retain the standards, without revision (58 FR 13008, March 9, 1993).

12 In the 1992 notice of its proposed decision in that review, the EPA announced its
13 intention to proceed as rapidly as possible with the next review of the air quality criteria and
14 standards for O₃ and other photochemical oxidants in light of emerging evidence of health effects
15 related to 6- to 8-hour O₃ exposures (57 FR 35542, August 10, 1992). The EPA subsequently
16 published the AQCD and Staff Paper for that next review (U.S. EPA, 1996). In December 1996,
17 the EPA proposed revisions to both the primary and secondary standards (61 FR 65716,
18 December 13, 1996). With regard to the primary standard, the EPA proposed to replace the then-
19 existing 1-hour primary standard with an 8-hour standard set at a level of 0.08 ppm (equivalent
20 to 0.084 ppm based on the proposed data handling convention) as a 3-year average of the annual
21 third-highest daily maximum 8-hour concentration. The EPA proposed to revise the secondary
22 standard either by setting it identical to the proposed new primary standard or by setting it as a
23 new seasonal standard using a cumulative form. The EPA completed this review in 1997 by
24 setting the primary standard at a level of 0.08 ppm, based on the annual fourth-highest daily
25 maximum 8-hour average concentration, averaged over three years, and setting the secondary
26 standard identical to the revised primary standard (62 FR 38856, July 18, 1997).

27 On May 14, 1999, in response to challenges by industry and others to the EPA's 1997
28 decision, the D.C. Circuit remanded the O₃ NAAQS to the EPA, finding that section 109 of the
29 CAA, as interpreted by the EPA, effected an unconstitutional delegation of legislative authority
30 (*American Trucking Ass'ns v. EPA*, 175 F.3d 1027, 1034-1040 [D.C. Cir. 1999]). In addition, the
31 court directed that, in responding to the remand, the EPA should consider the potential beneficial
32 health effects of O₃ pollution in shielding the public from the effects of solar ultraviolet (UV)
33 radiation, as well as adverse health effects (*id.* at 1051-53). In 1999, the EPA sought panel
34 rehearing and for rehearing *en banc* on several issues related to that decision. The court granted
35 the request for panel rehearing in part and denied it in part but declined to review its ruling with
36 regard to the potential beneficial effects of O₃ pollution (*American Trucking Ass'ns v. EPA*, 195

1 F.3d 4, 10 [D.C Cir., 1999]). On January 27, 2000, the EPA petitioned the U.S. Supreme Court
2 for *certiorari* on the constitutional issue (and two other issues) but did not request review of the
3 ruling regarding the potential beneficial health effects of O₃. On February 27, 2001, the U.S.
4 Supreme Court unanimously reversed the judgment of the D.C. Circuit on the constitutional
5 issue (*Whitman v. American Trucking Ass 'ns*, 531 U.S. 457, 472-74 [2001], [holding that section
6 109 of the CAA does not delegate legislative power to the EPA in contravention of the
7 Constitution]). The Court remanded the case to the D.C. Circuit to consider challenges to the O₃
8 NAAQS that had not been addressed by that court's earlier decisions. On March 26, 2002, the
9 D.C. Circuit issued its final decision on the remand, finding the 1997 O₃ NAAQS to be "neither
10 arbitrary nor capricious," and so denying the remaining petitions for review. See *ATA III*, 283
11 F.3d at 379.

12 Specifically, in *ATA III*, the D.C. Circuit upheld the EPA's decision on the 1997 O₃
13 standard as the product of reasoned decision making. With regard to the primary standard, the
14 court made clear that the most important support for the EPA's decision to revise the standard
15 was the health evidence of insufficient protection afforded by the then-existing standard ("the
16 record [is] replete with references to studies demonstrating the inadequacies of the old one-hour
17 standard"), as well as extensive information supporting the change to an 8-hour averaging time
18 (*id.* at 378). The court further upheld the EPA's decision not to select a more stringent level for
19 the primary standard noting "the absence of *any* [emphasis in original] human clinical studies at
20 ozone concentrations below 0.08 [ppm]" which supported the EPA's conclusion that "the most
21 serious health effects of ozone are 'less certain' at low concentrations, providing an eminently
22 rational reason to set the primary standard at a somewhat higher level, at least until additional
23 studies become available" (*id.* at 379, internal citations omitted). The court also pointed to the
24 significant weight that the EPA properly placed on the advice it received from the CASAC (*id.* at
25 379). In addition, the court noted that "although relative proximity to peak background ozone
26 concentrations did not, in itself, necessitate a level of 0.08 [ppm], EPA could consider that factor
27 when choosing among the three alternative levels" (*id.* at 379).

28 Coincident with the continued litigation of the other issues, the EPA responded to the
29 court's 1999 remand to consider the potential beneficial health effects of O₃ pollution in
30 shielding the public from effects of UV radiation (66 FR 57268, Nov. 14, 2001; 68 FR 614,
31 January 6, 2003). The EPA provisionally determined that the information linking changes in
32 patterns of ground-level O₃ concentrations to changes in relevant patterns of exposures to UV
33 radiation of concern (UV-B) to public health was too uncertain, at that time, to warrant any
34 relaxation in 1997 O₃ NAAQS. The EPA also expressed the view that any plausible changes in
35 UV-B radiation exposures from changes in patterns of ground-level O₃ concentrations would
36 likely be very small from a public health perspective. In view of these findings, the EPA

1 proposed to leave the 1997 primary standard unchanged (66 FR 57268, Nov. 14, 2001). After
2 considering public comment on the proposed decision, the EPA published its final response to
3 this remand in 2003, re-affirming the 8-hour primary standard set in 1997 (68 FR 614, January 6,
4 2003).

5 The EPA initiated the fourth periodic review of the air quality criteria and standards for
6 O₃ and other photochemical oxidants with a call for information in September 2000 (65 FR
7 57810, September 26, 2000). In 2007, the EPA proposed to revise the level of the primary
8 standard within a range of 0.075 to 0.070 ppm (72 FR 37818, July 11, 2007). The EPA proposed
9 to revise the secondary standard either by setting it identical to the proposed new primary
10 standard or by setting it as a new seasonal standard using a cumulative form. Documents
11 supporting these proposed decisions included the 2006 AQCD (U.S. EPA, 2006) and 2007 Staff
12 Paper (U.S. EPA, 2007) and related technical support documents. The EPA completed the
13 review in March 2008 by revising the levels of both the primary and secondary standards from
14 0.08 ppm to 0.075 ppm while retaining the other elements of the prior standards (73 FR 16436,
15 March 27, 2008).

16 In May 2008, state, public health, environmental, and industry petitioners filed suit
17 challenging the EPA's final decision on the 2008 O₃ standards. On September 16, 2009, the EPA
18 announced its intention to reconsider the 2008 O₃ standards,⁹ and initiated a rulemaking to do so.
19 At the EPA's request, the court held the consolidated cases in abeyance pending the EPA's
20 reconsideration of the 2008 decision.

21 In January 2010, the EPA issued a notice of proposed rulemaking to reconsider the 2008
22 final decision (75 FR 2938, January 19, 2010). In that notice, the EPA proposed that further
23 revisions of the primary and secondary standards were necessary to provide a requisite level of
24 protection to public health and welfare. The EPA proposed to revise the level of the primary
25 standard from 0.075 ppm to a level within the range of 0.060 to 0.070 ppm, and to revise the
26 secondary standard to one with a cumulative, seasonal form. At the EPA's request, the CASAC
27 reviewed the proposed rule at a public teleconference on January 25, 2010 and provided
28 additional advice in early 2011 (Samet, 2010, Samet, 2011). Later that year, in view of the need
29 for further consideration and the fact that the Agency's next periodic review of the O₃ NAAQS
30 required under CAA section 109 had already begun (as announced on September 29, 2008),¹⁰ the
31 EPA decided to consolidate the reconsideration with its statutorily required periodic review.¹¹

⁹ The press release of this announcement is available at:

https://archive.epa.gov/epapages/newsroom_archive/newsreleases/85f90b7711acb0c88525763300617d0d.html.

¹⁰ The *Call for Information* initiating the new review was announced in the Federal Register (73 FR 56581, September 29, 2008).

¹¹ This rulemaking, completed in 2015, concluded the reconsideration process.

1 In light of the EPA’s decision to consolidate the reconsideration with the ongoing
2 periodic review, the D.C. Circuit proceeded with the litigation on the 2008 O₃ NAAQS decision.
3 On July 23, 2013, the court upheld the EPA’s 2008 primary standard, but remanded the 2008
4 secondary standard to the EPA (*Mississippi v. EPA*, 744 F.3d 1334 [D.C. Cir. 2013]). With
5 respect to the primary standard, the court first rejected arguments that the EPA should not have
6 lowered the level of the existing primary standard, holding that the EPA reasonably determined
7 that the existing primary standard was not requisite to protect public health with an adequate
8 margin of safety, and consequently required revision. The court went on to reject arguments that
9 the EPA should have adopted a more stringent primary standard. With respect to the secondary
10 standard, the court held that the EPA’s explanation for the setting of the secondary standard
11 identical to the revised 8-hour primary standard was inadequate under the CAA because the EPA
12 had not adequately explained how that standard provided the required public welfare protection.

13 At the time of the court’s decision, the EPA had already completed significant portions of
14 its next statutorily required periodic review of the O₃ NAAQS. This review had been formally
15 initiated in 2008 with a call for information in the *Federal Register* (73 FR 56581, September 29,
16 2008). In late 2014, based on the ISA, Risk and Exposure Assessments (REAs) for health and
17 welfare, and PA¹² developed for this review, the EPA proposed to revise the 2008 primary and
18 secondary standards by reducing the level of both standards to within the range of 0.070 to 0.065
19 ppm (79 FR 75234, December 17, 2014).

20 The EPA’s final decision in this review was published in October 2015, establishing the
21 now-current standards (80 FR 65292, October 26, 2015). In this decision, based on consideration
22 of the health effects evidence on respiratory effects of O₃ in at-risk populations, the EPA revised
23 the primary standard from a level of 0.075 ppm to a level of 0.070 ppm, while retaining all the
24 other elements of the standard (80 FR 65292, October 26, 2015). The EPA’s decision on the
25 level for the standard was based on the weight of the scientific evidence and quantitative
26 exposure/risk information. The level of the secondary standard was also revised from 0.075 ppm
27 to 0.070 ppm based on the scientific evidence of O₃ effects on welfare, particularly the evidence
28 of O₃ impacts on vegetation, and quantitative analyses available in the review.¹³ The other
29 elements of the standard were retained. This decision on the secondary standard also
30 incorporated the EPA’s response to the D.C. Circuit’s remand of the 2008 secondary standard in
31 *Mississippi v. EPA*, 744 F.3d 1344 (D.C. Cir. 2013). The 2015 revisions to the NAAQS were

¹² The final versions of these documents, released in August 2014, were developed with consideration of the comments and recommendations from the CASAC, as well as comments from the public on the draft documents (Frey, 2014a, Frey, 2014b, Frey, 2014c, U.S. EPA, 2014a, U.S. EPA, 2014b, U.S. EPA, 2014c).

¹³ These standards, set in 2015, are specified at 40 CFR 50.19.

1 accompanied by revisions to the data handling procedures, and the ambient air monitoring
2 requirements¹⁴ (80 FR 65292, October 26, 2015).¹⁵

3 After publication of the final rule, a number of industry groups, environmental and health
4 organizations, and certain states filed petitions for judicial review in the D.C. Circuit. The
5 industry and state petitioners argued that the revised standards were too stringent, while the
6 environmental and health petitioners argued that the revised standards were not stringent enough
7 to protect public health and welfare as the Act requires. On August 23, 2019, the court issued an
8 opinion that denied all the petitions for review with respect to the 2015 primary standard while
9 also concluding that the EPA had not provided a sufficient rationale for aspects of its decision on
10 the 2015 secondary standard and remanding that standard to the EPA (*Murray Energy Corp. v.*
11 *EPA*, 936 F.3d 597 [D.C. Cir. 2019]).

12 In the August 2019 decision, the court additionally addressed arguments regarding
13 considerations of background O₃ concentrations, and socioeconomic and energy impacts. With
14 regard to the former, the court rejected the argument that the EPA was required to take
15 background O₃ concentrations into account when setting the NAAQS, holding that the text of
16 CAA section 109(b) precluded this interpretation because it would mean that if background O₃
17 levels in any part of the country exceeded the level of O₃ that is requisite to protect public health,
18 the EPA would be obliged to set the standard at the higher nonprotective level (*id.* at 622-23).
19 Thus, the court concluded that the EPA did not act unlawfully or arbitrarily or capriciously in
20 setting the 2015 NAAQS without regard for background O₃ (*id.* at 624). Additionally, the court
21 denied arguments that the EPA was required to consider adverse economic, social, and energy
22 impacts in determining whether a revision of the NAAQS was “appropriate” under section
23 109(d)(1) of the CAA (*id.* at 621-22). The court reasoned that consideration of such impacts was
24 precluded by *Whitman*’s holding that the CAA “unambiguously bars cost considerations from the
25 NAAQS-setting process” (531 U.S. at 471, summarized in section 1.2 above). Further, the court
26 explained that section 109(d)(2)(C)’s requirement that CASAC advise the EPA “of any adverse
27 public health, welfare, social, economic, or energy effects which may result from various
28 strategies for attainment and maintenance” of revised NAAQS had no bearing on whether costs
29 are to be considered in setting the NAAQS (*Murray Energy Corp. v. EPA*, 936 F.3d at 622).

¹⁴ The current federal regulatory measurement methods for O₃ are specified in 40 CFR 50, Appendix D and 40 CFR part 53. Consideration of ambient air measurements with regard to judging attainment of the standards set in 2015 is specified in 40 CFR 50, Appendix U. The O₃ monitoring network requirements are specified in 40 CFR 58.

¹⁵ This decision additionally announced revisions to the exceptional events scheduling provisions, as well as changes to the air quality index and the regulations for the prevention of significant deterioration permitting program.

1 Rather, as described in *Whitman* and discussed further in section 1.2 above, most of that advice
2 would be relevant to implementation but not standard setting (*id.*).

3 **1.4 REVIEW COMPLETED IN 2020**

4 The EPA announced its initiation of the next periodic review of the air quality criteria for
5 photochemical oxidants and the O₃ NAAQS in June 2018, issuing a call for information in the
6 *Federal Register* (83 FR 29785, June 26, 2018). Two types of information were called for:
7 information regarding significant new O₃ research to be considered for the ISA for the review,
8 and policy-relevant issues for consideration in this NAAQS review. Based in part on the
9 information received in response to the call for information, the EPA developed a draft IRP
10 which was made available for consultation with the CASAC and for public comment (83 FR
11 55163, November 2, 2018; 83 FR 55528, November 6, 2018). Comments from the CASAC
12 (Cox, 2018) and the public were considered in preparing the final IRP (U.S. EPA, 2019).

13 Under the plan outlined in the IRP and consistent with revisions to the process identified
14 by the Administrator in his 2018 memo directing initiation of the review and completion within
15 the statutorily required timeframe, the O₃ NAAQS review completed in 2020 progressed on an
16 accelerated schedule (Pruitt, 2018).¹⁶ The EPA incorporated a number of changes in various
17 aspects of the review process, as summarized in the IRP, to support completion within the
18 required period (Pruitt, 2018). For example, rather than produce separate documents for the PA
19 and associated quantitative analyses, the human exposure and health risk analyses (that inform
20 the decision on the primary standard) and the air quality and exposure analyses (that inform the
21 decision on the secondary standard) were included in full as appendices in the PA, along with a
22 number of other technical appendices.

23 Drafts of the ISA and PA (including the associated quantitative and exposure analyses)
24 were reviewed by the CASAC and made available for public comment (84 FR 50836, September
25 26, 2019; 84 FR 58711, November 1, 2019).¹⁷ In a divergence from recent past practice, an O₃
26 panel was not assembled to assist the CASAC in its review. Rather, the CASAC was assisted in
27 its review by a pool of consultants with expertise in a number of fields (84 FR 38625, August 7,
28 2019).¹⁸ The approach employed by the CASAC in utilizing outside technical expertise

¹⁶ The Administrator's May 2018 direction to initiate this review of the O₃ NAAQS included further direction to the EPA staff to expedite the review, implementing an accelerated schedule aimed at completion of the review within the statutorily required period (Pruitt, 2018).

¹⁷ The draft ISA and draft PA were released for public comment and CASAC review on September 26, 2019 and October 31, 2019, respectively. The charges for the CASAC review summarized the overarching context for the document review (including reference to Pruitt [2018], and the CASAC's role under section 109(d)(2)(C) of the Act), as well as specific charge questions for review of each of the documents.

¹⁸ Rather than join with some or all of the CASAC members in a pollutant specific review panel as had been common in previous NAAQS reviews, the consultants comprised a pool of expertise that CASAC members drew

1 represented an additional modification of the process from past reviews. The CASAC discussed
2 its draft letters describing its advice and comments on the documents in a public teleconference
3 in early February 2020 (85 FR 4656; January 27, 2020). The letters to the Administrator
4 conveying the CASAC advice and comments on the draft PA and draft ISA were released later
5 that month (Cox, 2020a, Cox, 2020b). Comments from the CASAC and the public on the draft
6 ISA were considered by the EPA and led to a number of revisions in developing the final
7 document (ISA, Appendix 10, section 10.4.5). The ISA was completed and made available to the
8 public in April 2020 (85 FR 21849, April 20, 2020). The comments from CASAC and the public
9 were also considered in completing the PA and the advice regarding the standards was described
10 and considered in the final 2020 PA (85 FR 31182, May 22, 2020), and in the EPA’s decision-
11 making. On August 14, 2020, the EPA proposed to retain both the primary and secondary O₃
12 standards, without revision (85 FR 49830, August 14, 2020). In December 2020, the EPA issued
13 its final decision to retain the existing standards without revision (85 FR 87256, December 31,
14 2020).¹⁹

15 Following publication of the 2020 final action, three petitions were filed for review of the
16 EPA’s final decision in the D.C. Circuit and the court consolidated the cases. The EPA also
17 received two petitions for reconsideration of the 2020 action. On October 29, 2021, the Agency
18 filed a motion with the court explaining that it had decided to reconsider the 2020 O₃ NAAQS
19 final decision²⁰ and requested that the consolidated cases be held in abeyance until December 15,
20 2023. On December 21, 2021, the court ordered that the consolidated cases continue to be held in
21 abeyance pending further order of the court and directed the parties to file motions to govern by
22 December 15, 2023.

23 **1.5 RECONSIDERATION OF THE 2020 O₃ NAAQS DECISION**

24 On October 29, 2021, the EPA announced that it will reconsider the 2020 decision to
25 retain the 2015 O₃ standards. The EPA’s plans are to reconsider the decision based on the
26 existing scientific record and in a manner that adheres to rigorous standards of scientific integrity
27 and provides ample opportunities for public input and engagement.²¹ Consistent with the

on through the use of specific questions, posed in writing prior to the public meeting, regarding aspects of the documents being reviewed, as a means of obtaining subject matter expertise for its document review.

¹⁹ The decision on the secondary standard also considered and addressed the 2019 remand of the secondary standard by the D.C. Circuit such that that decision incorporated the EPA’s response to that remand.

²⁰ The Agency’s October 29, 2021 announcement is available at <https://www.epa.gov/ground-level-ozone-pollution/epa-reconsider-previous-administrations-decision-retain-2015-ozone>.

²¹ Information about the decision to reconsider the December 2020 O₃ NAAQS decision is available on this webpage: <https://www.epa.gov/ground-level-ozone-pollution/epa-reconsider-previous-administrations-decision-retain-2015-ozone>

1 commitment to rigorous standards of scientific integrity, the EPA will receive advice and
2 comments from a reestablished CASAC²² assisted by an expert O₃ Panel.²³ This reflects EPA’s
3 renewed commitment to a rigorous NAAQS review process, with a focus on protecting scientific
4 integrity.

5 Presentations and considerations to be included in the PA for reconsideration will be
6 based on the conclusions, studies and related information included in the air quality criteria for
7 the 2020 review. This includes the studies assessed in the 2020 ISA and PA and the integration
8 of the scientific evidence presented in them. The EPA has additionally provisionally considered
9 two sets of scientific studies on the health and welfare effects of O₃ that were not included in the
10 ISA (“ ‘new’ studies”) and that did not go through the comprehensive review process utilized in
11 review of the air quality criteria. With regard to the first set of studies, the EPA provisionally
12 considered a set of “new” scientific studies on the health and welfare effects of O₃ that were
13 raised and discussed in public comments on the July 2020 proposed decision (Luben et al.,
14 2020). In considering and responding to the comments, the EPA provisionally considered the
15 studies in the context of the findings of the ISA, as described in the December 2020 decision (85
16 FR 87262, December 31, 2020). The EPA concluded that, taken in context, the “new”
17 information and findings did not materially change any of the broad scientific conclusions
18 regarding the health and welfare effects of O₃ in ambient air made in the air quality criteria, and
19 accordingly, reopening the air quality criteria review was not warranted (Luben et al., 2020).²⁴
20 More recently, in the context of this reconsideration of the 2020 decision on the primary
21 standard, given the primary role of controlled human exposure studies in the most recent
22 decisions on the primary standard, the EPA has conducted a literature search for any “new”
23 controlled human exposure studies that may have been published since the literature cutoff date
24 for the 2020 ISA, and provisionally evaluated this small set of such newly identified studies
25 (Duffney et al., 2022). Based on this provisional evaluation, the EPA has concluded that, taken in
26 context, the “new” information and findings do not materially change any of the broad scientific

²² Consistent with his decision to reestablish the membership of the CASAC to “ensure the agency received the best possible scientific insight to support our work to protect human health and the environment,” after consideration of a candidate list based on public request for nominations (86 FR 17146-17147, April 1, 2021) the Administrator announced selection of the seven members to serve on the chartered CASAC on June 17, 2021 (<https://www.epa.gov/newsreleases/epa-announces-selections-charter-members-clean-air-scientific-advisory-committee>). The current CASAC membership is listed here: https://casac.epa.gov/ords/sab/f?p=105:29:1723269351020:::RP.29:P29_COMMITTEON:CASAC.

²³ The members of the O₃ CASAC panel are identified here: https://casac.epa.gov/ords/sab/f?p=113:14:11923922295141:::14:P14_COMMITTEON:2022%20CASAC%20zone%20Review%20Panel.

²⁴ As noted at that time, “new” studies may sometimes be of such significance that it is appropriate to delay a decision in a NAAQS review and to supplement the pertinent air quality criteria so the studies can be taken into account (58 FR at 13013– 13014, March 9, 1993).

1 conclusions regarding the health and welfare effects of O₃ in ambient air made in the air quality
2 criteria; thus, reopening the air quality criteria review is not warranted (Duffney et al., 2022).

3 This PA is being developed for consideration by the EPA Administrator in reaching his
4 decision on the reconsideration of the December 2020 decision to retain the existing O₃ NAAQS.
5 In assessing the policy implications of the available scientific information, this PA for the
6 reconsideration, as for the 2020 PA, is intended to help “bridge the gap” between the Agency’s
7 scientific assessment, presented in the 2020 ISA, and quantitative technical analyses, and the
8 judgments required of the Administrator in determining whether it is appropriate to retain or
9 revise the O₃ NAAQS. Accordingly, the PA for reconsideration will again address policy-
10 relevant questions based on those identified in the 2018 IRP. With regard to considerations
11 related to the primary standard, the PA for the reconsideration will focus on the evidence
12 described in the 2020 ISA,²⁵ and the exposure/risk analyses presented in the 2020 PA, which
13 will be included in full in this PA. With regard to considerations related to the secondary
14 standard, the PA for reconsideration will focus on the evidence documented in the 2020 ISA,
15 along with quantitative analyses presented in the 2020 PA and in subsequent technical memos,
16 which have been updated to reflect recent air quality data.

17 This draft PA for the reconsideration is being provided to the CASAC for review and
18 comment and made available for public comment. The CASAC advice and public comment on
19 this draft PA will inform completion of the final PA and development of the Administrator’s
20 proposed decision. The EPA is targeting the end of 2023 to complete decision-making in this
21 reconsideration.
22

²⁵ The ISA builds on evidence and conclusions from previous assessments, focusing on synthesizing and integrating the newly available evidence (ISA, section IS.1.1). Past assessments are generally cited when providing further, still relevant, details that informed the current assessment but are not repeated in the latest assessment.

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6

2 AIR QUALITY

This chapter begins with an overview of O₃ and other photochemical oxidants in the atmosphere (section 2.1). Subsequent sections summarize the sources and emissions of O₃ precursors (section 2.2), ambient air monitoring and data handling conventions for determining whether the standards are met (section 2.3), O₃ concentrations measured in the U.S. ambient air (section 2.4), and available evidence and information related to background O₃ in the U.S. (section 2.5). These focus primarily on tropospheric O₃ and surface-level concentrations occurring in ambient air¹.

2.1 O₃ AND PHOTOCHEMICAL OXIDANTS IN THE ATMOSPHERE

O₃ is one of many photochemical oxidants formed in the troposphere² by photochemical reactions of precursor gases in the presence of sunlight (ISA, Appendix 1, section 1.1)³ and is generally not directly emitted from specific sources. Tropospheric O₃ and other oxidants, such as peroxyacetyl nitrate (PAN) and hydrogen peroxide, form in polluted areas through atmospheric reactions involving two main classes of precursor pollutants: volatile organic compounds (VOCs) and nitrogen oxides (NO_x = NO and NO₂). The photolysis of the primary pollutant nitrogen dioxide (NO₂) results in products of NO and a singlet oxygen radical that can subsequently either form ozone or react with NO to reform the parent NO₂ compound. The reaction of the oxygen radical with NO to form NO₂ is disrupted by the presence of VOCs⁴ which leads to net ozone formation in the troposphere. Thus, NO_x, VOCs, CH₄ and CO are considered to be the primary precursors of tropospheric O₃ (ISA, Appendix 1, section 1.3.1)

The formation of O₃, other oxidants and oxidation products from these precursors is a complex, nonlinear function of many factors including (1) the intensity and spectral distribution of sunlight; (2) atmospheric mixing; (3) concentrations of precursors in the ambient air and the rates of chemical reactions of these precursors; and (4) processing on cloud and aerosol particles (ISA, Appendix 1, section 1.4; 2013 ISA, section 3.2). As a result, O₃ changes in a nonlinear fashion with the concentrations of its precursors rather than varying proportionally to emissions

¹ Ambient air means that portion of the atmosphere, external to buildings, to which the general public has access (see 40 CFR 50.1(e)).

² Ozone also occurs in the stratosphere, where it serves the beneficial role of absorbing the sun's harmful ultraviolet radiation and preventing the majority of this radiation from reaching the Earth's surface.

³ The only other appreciable source of O₃ to the troposphere is transport from the stratosphere, as described in section 2.5.1.1 below.

⁴ This reaction can also be disrupted by the radical that results from methane (CH₄) oxidation or a reaction between carbon monoxide (CO) and the hydroxyl radical (OH) in the atmosphere.

1 of its precursors (2013 ISA, section 3.2.4). In addition to the chemistry described above, NO can
2 also react with ozone directly such that emissions of NO_x lead to both the formation and
3 destruction of O₃, with the net formation or destruction depending on the local quantities of
4 NO_x, VOCs, radicals, and sunlight. O₃ chemistry is often described in terms of which precursors
5 most directly impact formation rates. A NO_x-limited regime indicates that O₃ concentrations will
6 decrease in response to decreases in ambient NO_x concentrations and vice-versa. These
7 conditions tend to occur when NO_x concentrations are generally low compared to VOC
8 concentrations and during warm, sunny conditions when NO_x photochemistry is relatively fast.
9 NO_x-limited conditions are more common during daylight hours, in the summertime, in
10 suburban and rural areas, and in portions of the country with high biogenic VOC emissions like
11 the Southeast. In contrast, NO_x-saturated conditions (also referred to as VOC-limited or radical-
12 limited) indicate that O₃ will increase as a result of NO_x reductions but will decrease as a result
13 of VOC reductions (2013 ISA, section 3.2; 2006 AQCD, chapter 2). NO_x-saturated conditions
14 occur at times when and at locations with lower levels of available sunlight, resulting in slower
15 photochemical formation of O₃, and when NO_x concentrations are in excess compared to VOC
16 concentrations. NO_x-saturated conditions are more common during nighttime hours, in the
17 wintertime, and in densely populated urban areas or industrial plumes. These varied relationships
18 between precursor emissions and O₃ chemistry result in localized areas in which O₃
19 concentrations are suppressed compared to surrounding areas, but which contain NO₂ that
20 contributes to subsequent O₃ formation further downwind (2013 ISA, section 3.2.4).
21 Consequently, O₃ response to reductions in NO_x emissions is complex and may include
22 decreases in O₃ concentrations at some times and locations and increases in O₃ concentrations at
23 other times and locations. Over the past decade, there have been substantial decreases in NO_x
24 emissions in the U.S. (see Figure 2-2) and many locations have transitioned from NO_x-saturated
25 to NO_x-limited (Jin et al., 2017) during times of year that are conducive to O₃ formation
26 (generally summer). As these NO_x emissions reductions have occurred, lower O₃ concentrations
27 have generally increased while the higher O₃ concentrations have generally decreased, resulting
28 in a compressed O₃ distribution, relative to historical conditions (ISA, Appendix 1, section 1.7).

29 Prior to 1979, the indicator for the NAAQS for photochemical oxidants was total
30 photochemical oxidants (36 FR 8186, April 30, 1971). Early ambient air monitoring indicated
31 similarities between O₃ measurements and the photochemical oxidant measurements, as well as
32 reduced precision and accuracy of the latter (U.S. EPA, 1978). To address these issues, the EPA
33 established O₃ as the indicator for the NAAQS for photochemical oxidants in 1979 (44 FR 8202,
34 February 8, 1979), and it is currently the only photochemical oxidant other than nitrogen dioxide
35 that is routinely monitored in a national ambient air monitoring network.

1 O₃ is present not only in polluted urban atmospheres, but throughout the troposphere,
2 even in remote areas of the globe. The same basic processes involving sunlight-driven reactions
3 of NO_x, VOCs, CH₄ and CO contribute to O₃ formation throughout the troposphere. These
4 processes also lead to the formation of other photochemical products, such as PAN, HNO₃, and
5 H₂SO₄, HCHO and other carbonyl compounds, as well as a number of organic particulate
6 compounds (ISA, Appendix 1, section 1.4; 2013 ISA, section 3.2).

7 As mentioned above, the formation of O₃ from precursor emissions is also affected by
8 meteorological parameters such as the intensity of sunlight and atmospheric mixing (2013 ISA,
9 section 3.2). Major episodes of high O₃ concentrations in the eastern U.S. are often associated
10 with slow-moving high-pressure systems which can persist for several days. High pressure
11 systems during the warmer seasons are associated with the sinking of air, resulting in warm,
12 generally cloudless skies, with light winds. The sinking of air results in the development of
13 stable conditions near the surface which inhibit or reduce the vertical mixing of O₃ precursors,
14 concentrating them near the surface. Photochemical activity involving these precursors is
15 enhanced because of higher temperatures and the availability of sunlight during the warmer
16 seasons. In the eastern U.S., concentrations of O₃ and other photochemical oxidants are
17 determined by meteorological and chemical processes extending typically over areas of several
18 hundred thousand square kilometers. Therefore, O₃ episodes are often regarded as regional in
19 nature, although more localized episodes often occur in some areas, largely the result of local
20 pollution sources during summer, e.g., Houston, TX (2013 ISA, section 2.2.1; Webster et al.,
21 2007). In addition, in some parts of the U.S. (e.g., Los Angeles, CA), mountain barriers limit O₃
22 dispersion and result in a higher frequency and duration of days with elevated O₃ concentrations
23 (2013 ISA, section 3.2).

24 More recently, high O₃ concentrations of up to 150 parts per billion (ppb)⁵ have been
25 measured during the wintertime in two western U.S. mountain basins (ISA, Appendix 1, section
26 1.4.1). Wintertime mountain basin O₃ episodes occur on cold winter days with low wind speeds,
27 clear skies, substantial snow cover, extremely shallow boundary layers driven by strong
28 temperature inversions, and substantial precursor emissions activity from the oil and gas sector.
29 The results of recent modeling studies suggest that photolysis of VOCs provides the source of
30 reactive chemical species (radicals) needed to initiate the chemistry driving these wintertime O₃
31 episodes. This mechanism is somewhat different from the chemistry driving summertime O₃
32 formation, which is initiated with the photolysis of NO₂ followed by the formation of the OH
33 radicals (ISA, Appendix 1, section 1.4.1).

⁵ Although the standards are specified in ppm (e.g., as described in Chapter 1), the units, ppb, are commonly used in describing O₃ concentrations throughout this document, with 0.070 ppm being equivalent to 70 ppb.

1 O₃ concentrations in a region are affected both by local formation and by transport of O₃
2 and its precursors from upwind areas. O₃ transport occurs on many spatial scales including local
3 transport within urban areas, regional transport over large regions of the U.S., and long-range
4 transport which may also include international transport. In addition, O₃ can be transferred into
5 the troposphere from the stratosphere, which is rich in naturally occurring O₃, through
6 stratosphere-troposphere exchange (STE). These intrusions usually occur behind cold fronts,
7 bringing stratospheric air with them and typically affect O₃ concentrations in higher elevation
8 areas (e.g., > 1500 m) more than areas at lower elevations, as discussed in section 2.5.3.2 (ISA,
9 Appendix 1, section 1.3.2.1; 2013 ISA, section 3.4.1.1).

10 **2.2 SOURCES AND EMISSIONS OF O₃ PRECURSORS**

11 Sources of emissions of O₃ precursor compounds can be divided into anthropogenic and
12 natural source categories, with natural sources further divided into emissions from biological
13 processes of living organisms (e.g., plants, microbes, and animals) and emissions from chemical
14 or physical processes (e.g., biomass burning, lightning, and geogenic sources). Anthropogenic
15 emissions associated with combustion processes, including mobile sources and power plants,
16 account for the majority of U.S. NO_x and CO emissions (Figure 2-1 and Figure 2-2). Emissions
17 of these chemicals have declined appreciably in the U.S. since 2002 (Figure 2-2). Anthropogenic
18 sources are also important for VOC emissions, though in some locations and times of the year
19 (e.g., southern states during summer) the majority of VOC emissions come from vegetation
20 (2013 ISA, section 3.2.1).⁶ In practice, the distinction between natural and anthropogenic sources
21 is often unclear, as human activities directly or indirectly affect emissions from what would have
22 been considered natural sources during the preindustrial era. Thus, precursor emissions from
23 plants, animals, and wildfires could be considered either natural or anthropogenic, depending on
24 whether emissions result from agricultural practices, forest management practices, lightning
25 strikes, or other types of events. There are additional challenges in distinguishing between ozone
26 resulting from natural versus anthropogenic sources because much O₃ results from reactions of
27 anthropogenic precursors with natural precursors (ISA, Appendix 1, section 1.8.1.2).

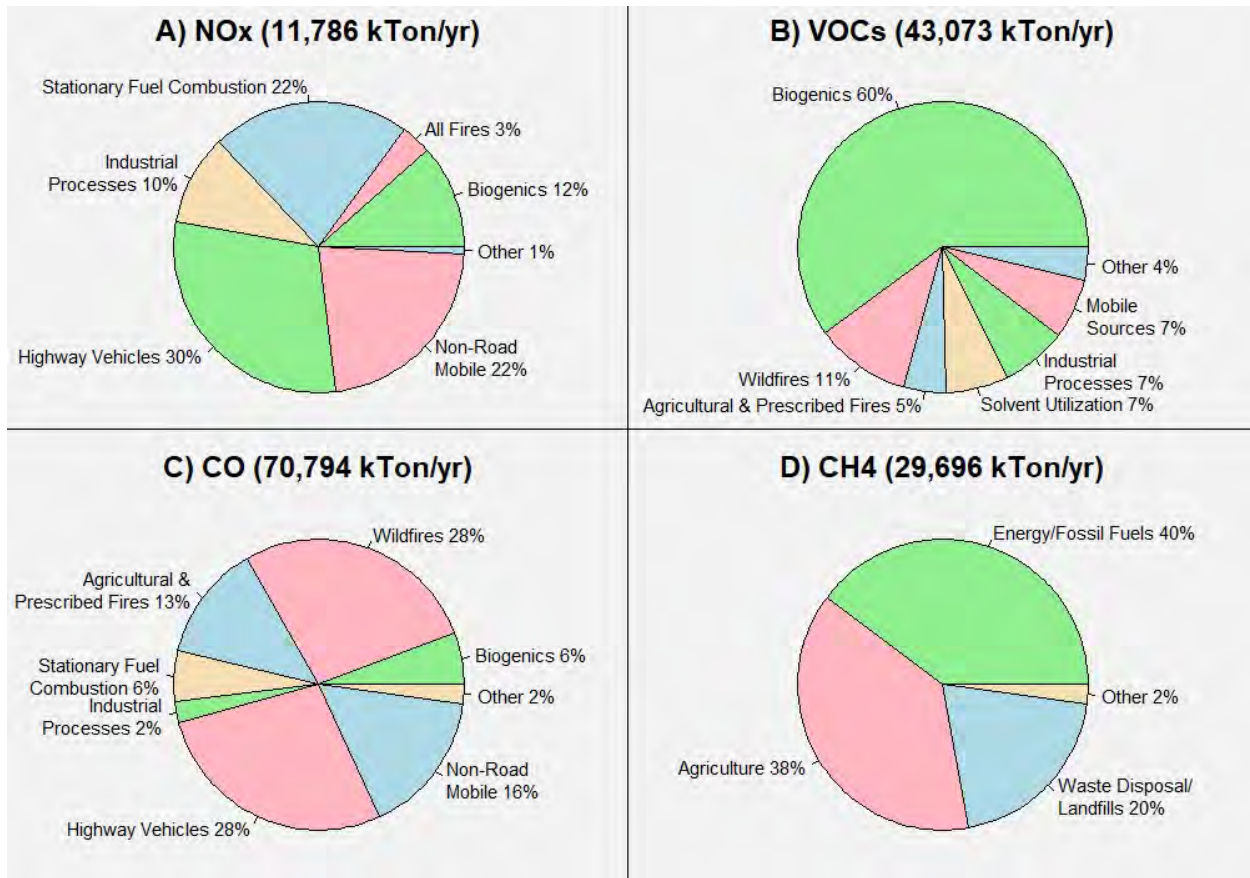
28 The National Emissions Inventory (NEI) is a comprehensive and detailed estimate of air
29 emissions of criteria pollutants, precursors to criteria pollutants, and hazardous air pollutants
30 from air emissions sources (U.S. EPA, 2021b). The NEI is released every three years based
31 primarily upon data provided by State, Local, and Tribal air agencies for sources in their

⁶ It should be noted that the definition of VOCs used in this section does not include CH₄ because it is excluded from the EPA's regulatory definition of VOCs in 40 CFR 51.100(s). More information about this regulatory definition of VOCs is available at <https://www.epa.gov/indoor-air-quality-iaq/technical-overview-volatile-organic-compounds>.

1 jurisdictions and supplemented by data developed by the US EPA. The NEI is built using the
2 EPA’s Emissions Inventory System (EIS) which collects data from State, Local, and Tribal air
3 agencies and blends that data with other data sources.⁷

4 Anthropogenic emissions of air pollutants result from a variety of sources such as power
5 plants, industrial sources, motor vehicles, and agriculture. The emissions from any individual
6 source typically vary in both time and space. For many of the thousands of sources that make up
7 the NEI, there is uncertainty in both of these factors. For some sources, such as power plants,
8 direct emission measurements enable more certain quantification of the magnitude and timing of
9 emissions than from sources without such direct measurements. However, for many source
10 categories emission inventories necessarily contain assumptions, interpolation and extrapolation
11 from a limited set of sample data (U.S. EPA, 2021b).

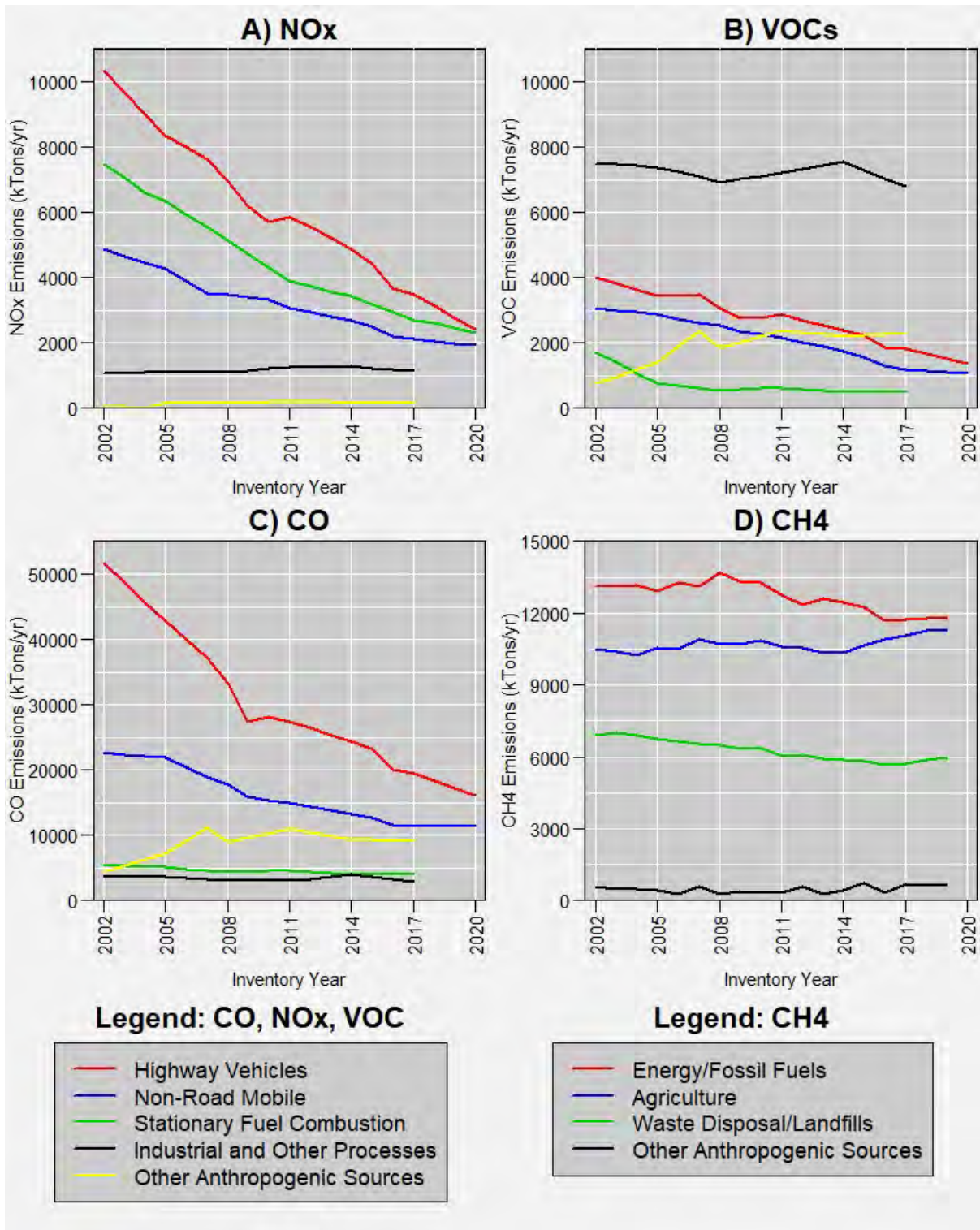
⁷ More details are available from: <https://www.epa.gov/enviro/nei-overview>.



1
 2 **Sources:** The 2017 National Emissions Inventory (U.S. EPA, 2021b) for panels A-C, and the *Inventory of U.S. Greenhouse Gas*
 3 *Emissions and Sinks: 1990-2019* (U.S. EPA, 2021a) for panel D. Categories contributing less than 2% each have been summed
 4 and are represented by the "other" category.

5 **Figure 2-1. U.S. O₃ precursor emissions by sector: A) NO_x; B) CO; C) VOCs; D) CH₄.**

6



1
 2 Sources: EPA's Air Pollutant Emissions Trends Data webpage (<https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data>) for panels A-C, and the *Inventory of U.S. Greenhouse Gas Emissions and Sinks: 1990-2019* (U.S. EPA, 2021a) for panel D.

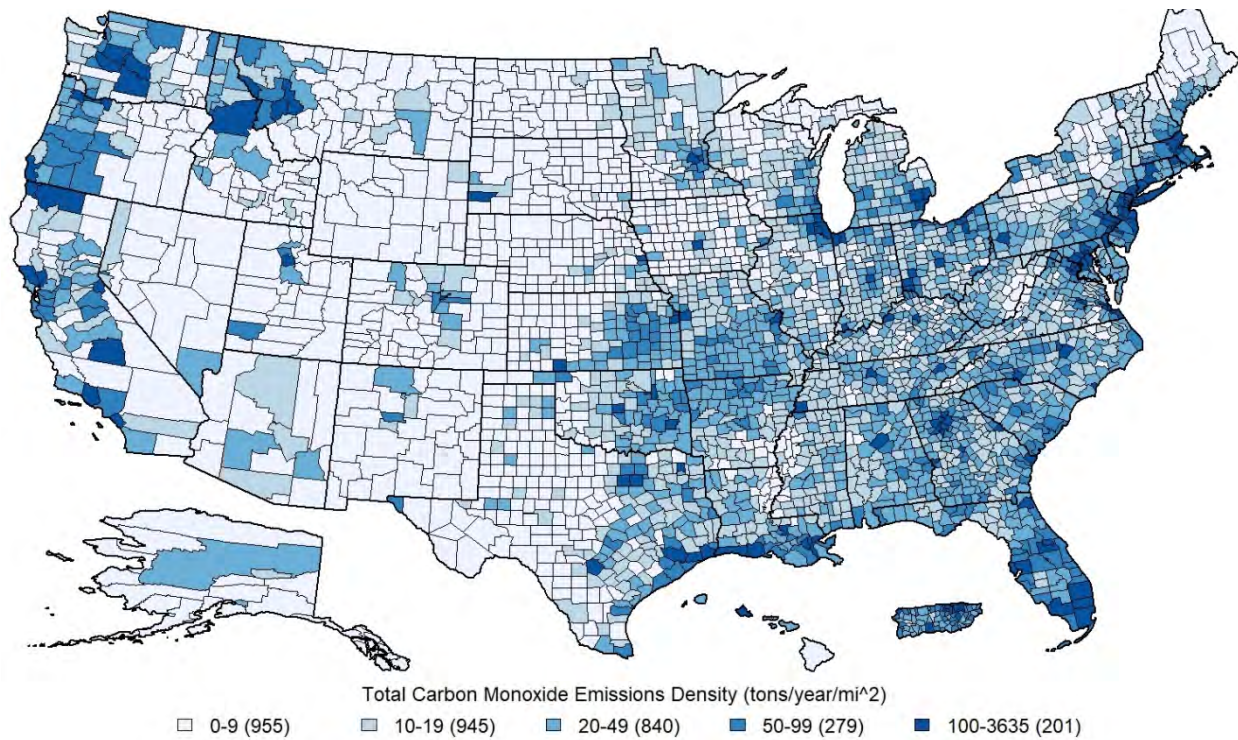
5 **Figure 2-2. U.S. anthropogenic O₃ precursor emission trends for: A) NO_x; B) CO; C)**
 6 **VOCs; and D) CH₄.**

1
2 Figure 2-3, Figure 2-4, and Figure 2-5 show county-level estimates of U.S. emissions
3 densities (in tons/year/mi²) for CO, NO_x, and VOCs, respectively. In general, CO and NO_x
4 emissions tend to be highest in urban areas which typically have the most anthropogenic sources,
5 however, CO emissions may be higher in some rural areas due to fires, and similarly, NO_x
6 emissions may be higher in some rural areas due to sources such as electricity generation, oil and
7 gas extraction, and traffic along major highways. While there are some significant anthropogenic
8 sources of VOC emissions in urban areas, in rural areas the vast majority of VOC emissions
9 come from plants and trees (biogenics), particularly in the southeastern U.S. In other areas of the
10 U.S., such as the Great Plains region and parts of the inter-mountain west, areas with higher
11 levels of VOC emissions are largely due to oil and gas extraction (U.S. EPA, 2021b).

12 It should be noted that O₃ levels in a given area are impacted by both local emissions that
13 form O₃ in the area as well as remote emissions that form O₃ that is then transported into the
14 area. Biogenic VOC emissions that lead to O₃ formation may vary greatly depending on the type
15 and amount of vegetation, which is generally much lower in urban areas than in rural areas.
16 However, biogenic VOC emissions that are upwind of an urban area can have a significant
17 impact on urban O₃ levels. Thus, while the county-level maps shown in Figure 2-3, Figure 2-4,
18 and Figure 2-5 illustrate the variability in precursor emissions in the U.S., it is not sufficient to
19 look only at the patterns in local emissions when considering the impact on O₃ concentrations.

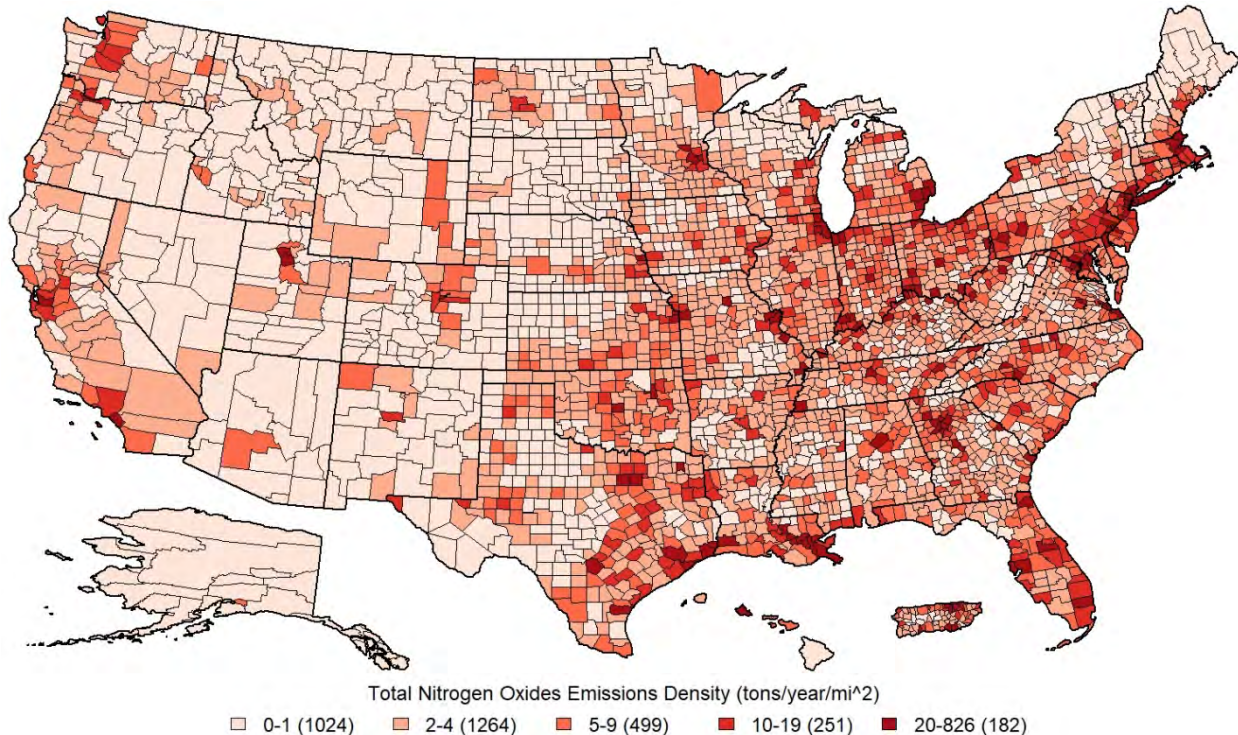
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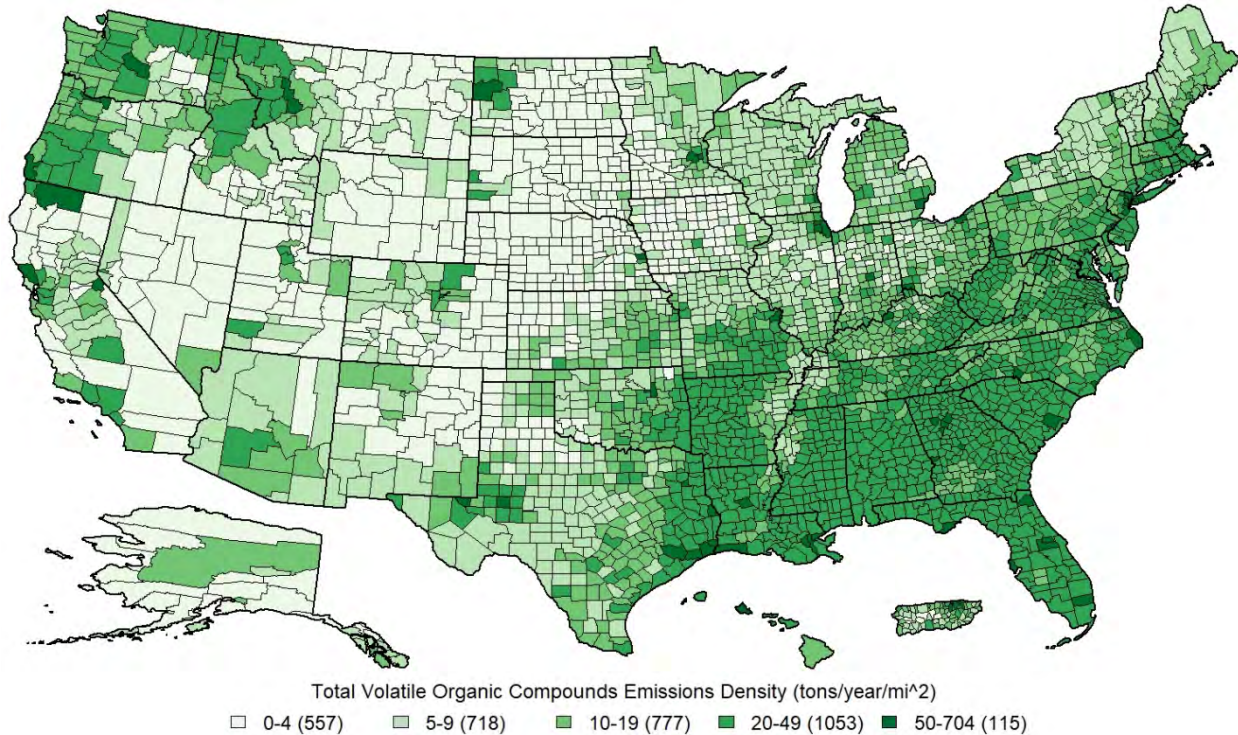
1
 2 Source: 2017 National Emissions Inventory, January 2021 Updated Release (U.S. EPA, 2021b; data downloaded from
 3 <https://www.epa.gov/air-emissions-inventories/2017-national-emissions-inventory-nei-data>)

4 **Figure 2-3. U.S. county-level CO emissions density estimates (tons/year/mi²) for 2017.**



5
 6 Source: 2017 National Emissions Inventory, January 2021 Updated Release (U.S. EPA, 2021b; data downloaded from
 7 <https://www.epa.gov/air-emissions-inventories/2017-national-emissions-inventory-nei-data>)

8 **Figure 2-4. U.S. county-level NO_x emissions density estimates (tons/year/mi²) for 2017.**



1
2 Source: 2017 National Emissions Inventory, January 2021 Updated Release (U.S. EPA, 2021b; data downloaded from
3 <https://www.epa.gov/air-emissions-inventories/2017-national-emissions-inventory-nei-data>)

4 **Figure 2-5. U.S. county-level VOC emissions density estimates (tons/year/mi²) for 2017.**

5 **2.3 AMBIENT AIR MONITORING AND DATA HANDLING**
6 **CONVENTIONS**

7 **2.3.1 Ambient Air Monitoring Requirements and Monitoring Networks**

8 State and local environmental agencies operate a network of O₃ monitors at state or local
9 air monitoring stations (SLAMS). The requirements for the SLAMS network depend on the
10 population and most recent O₃ design values⁸ in an area. The minimum number of O₃ monitors
11 required in a metropolitan statistical area (MSA) ranges from zero for areas with a population
12 less than 350,000 and no recent history of an O₃ design value greater than 85 percent of the level
13 of the standard, to four for areas with a population greater than 10 million and an O₃ design value
14 greater than 85 percent of the standard level.⁹ At least one monitoring site for each MSA must be
15 situated to record the maximum concentration for that particular metropolitan area. Siting criteria

⁸ 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.

⁹ The SLAMS minimum monitoring requirements to meet the O₃ design criteria are specified in 40 CFR Part 58, Appendix D. The minimum O₃ monitoring network requirements for urban areas are listed in Table D-2 of Appendix D to 40 CFR Part 58 (accessible at <https://www.ecfr.gov>).

1 for SLAMS includes horizontal and vertical inlet probe placement; spacing from minor sources,
2 obstructions, trees, and roadways; inlet probe material; and sample residence times.¹⁰ Adherence
3 to these criteria ensures uniform collection and comparability of O₃ data. Since the highest O₃
4 concentrations tend to be associated with a particular season for various locations, the EPA
5 requires O₃ monitoring during specific O₃ monitoring seasons (shown in Figure 2-6) which vary
6 by state from five months (May to September in Oregon and Washington) to all twelve months
7 (in 11 states), with the most common season being March to October (in 27 states).¹¹

8 Most of the state, local, and tribal air monitoring stations that report data to the EPA use
9 ultraviolet Federal Equivalent Methods (FEMs). The Federal Reference Method (FRM) was
10 revised in 2015 to include a new chemiluminescence by nitric oxide (NO-CL) method. The
11 previous ethylene (ET-CL) method, while still included in the CFR as an acceptable method, is
12 no longer used due to lack of availability and safety concerns with ethylene.¹² The NO-CL
13 method is beginning to be implemented in the SLAMS network.¹³

14 Ambient air quality data and associated quality assurance (QA) data are reported to the
15 EPA via the Air Quality System (AQS). Data are reported quarterly and must be submitted to
16 AQS within 90 days after the end of the quarterly reporting period. Each monitoring agency is
17 required to certify data that is submitted to AQS from the previous year. The data are certified,
18 taking into consideration any QA findings, and a data certification letter is sent to the EPA
19 Regional Administrator. Data must be certified by May 1st of the following year. Data collected
20 by FRM or FEM monitors that meet the QA requirements must be certified.¹⁴ To provide
21 decision makers with an assessment of data quality, the EPA's QA group derives estimates of
22 both precision and bias for O₃ and the other gaseous criteria pollutants from quality control (QC)
23 checks using calibration gas, performed at each site by the monitoring agency. The data quality
24 goal for precision and bias is 7 percent.¹⁵

¹⁰ The probe and monitoring path siting criteria for ambient air quality monitoring are specified in 40 CFR, Part 58, Appendix E.

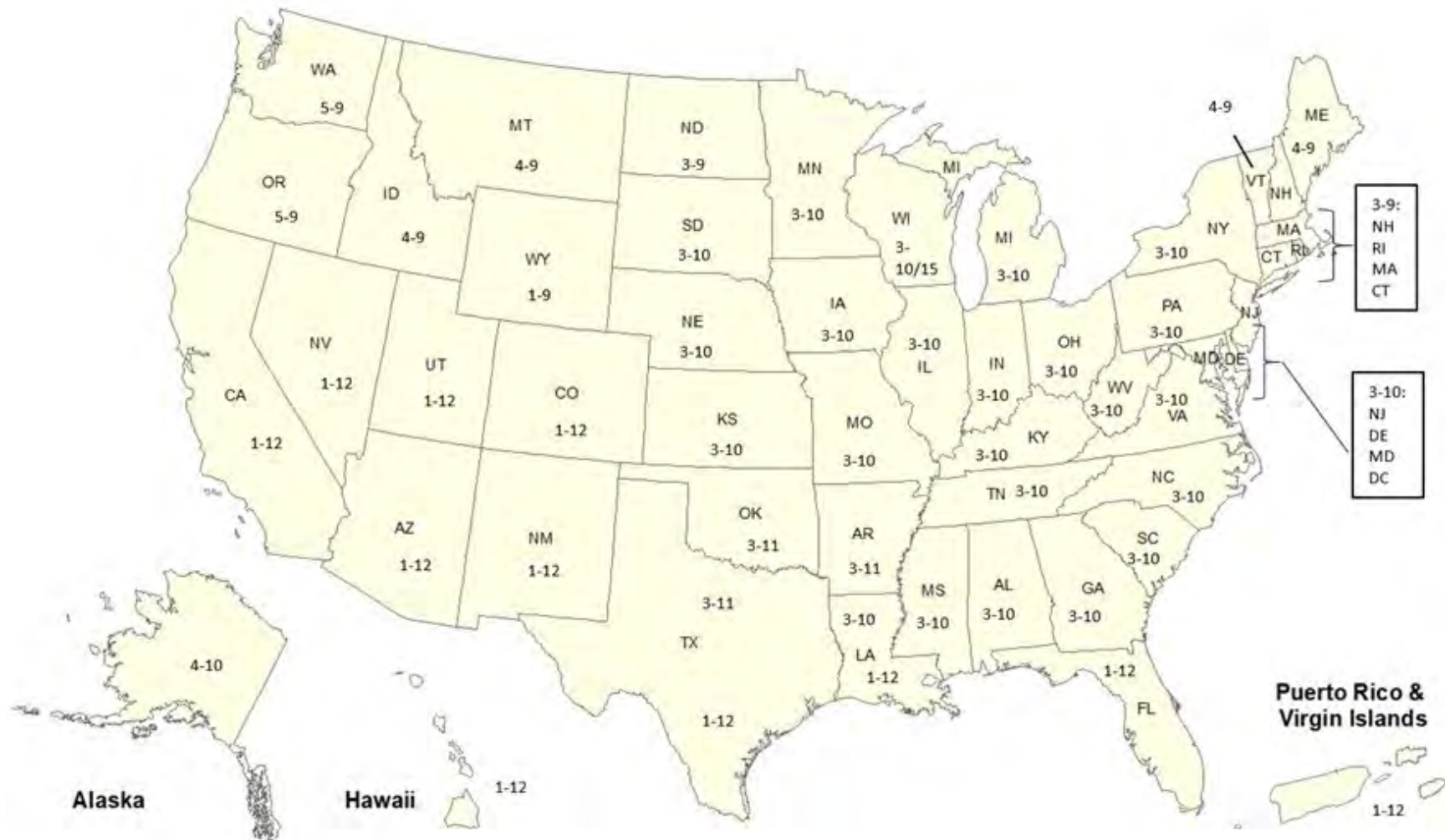
¹¹ The required O₃ monitoring seasons for each state are listed in 40 CFR Part 58, Appendix D, Table D-3.

¹² The current FRM for O₃ (established in 2015) is a chemiluminescence method, which is fully described in 40 CFR Part 50, Appendix D.

¹³ The EPA is currently participating in an international effort to implement a globally coordinated change in the parameter (the absorption cross-section value) used in the determination of atmospheric ozone for ozone monitoring, which will require an update of this parameter in the ozone monitoring regulations (40 CFR Part 50, Appendix D, section 4). The global implementation target date for this change is the beginning of the 2024 ozone season.

¹⁴ Quality assurance requirements for monitors used in evaluations of the NAAQS are provided in 40 CFR Part 58, Appendix A.

¹⁵ Annual summary reports of precision and bias can be obtained for each monitoring site at <https://www.epa.gov/outdoor-air-quality-data/single-point-precision-and-bias-report>.



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Figure 2-6. Current O₃ monitoring seasons in the U.S. Numbers in each state indicate the months of the year the state is required to monitor for O₃ (e.g., 3-10 means O₃ monitoring is required from March through October).

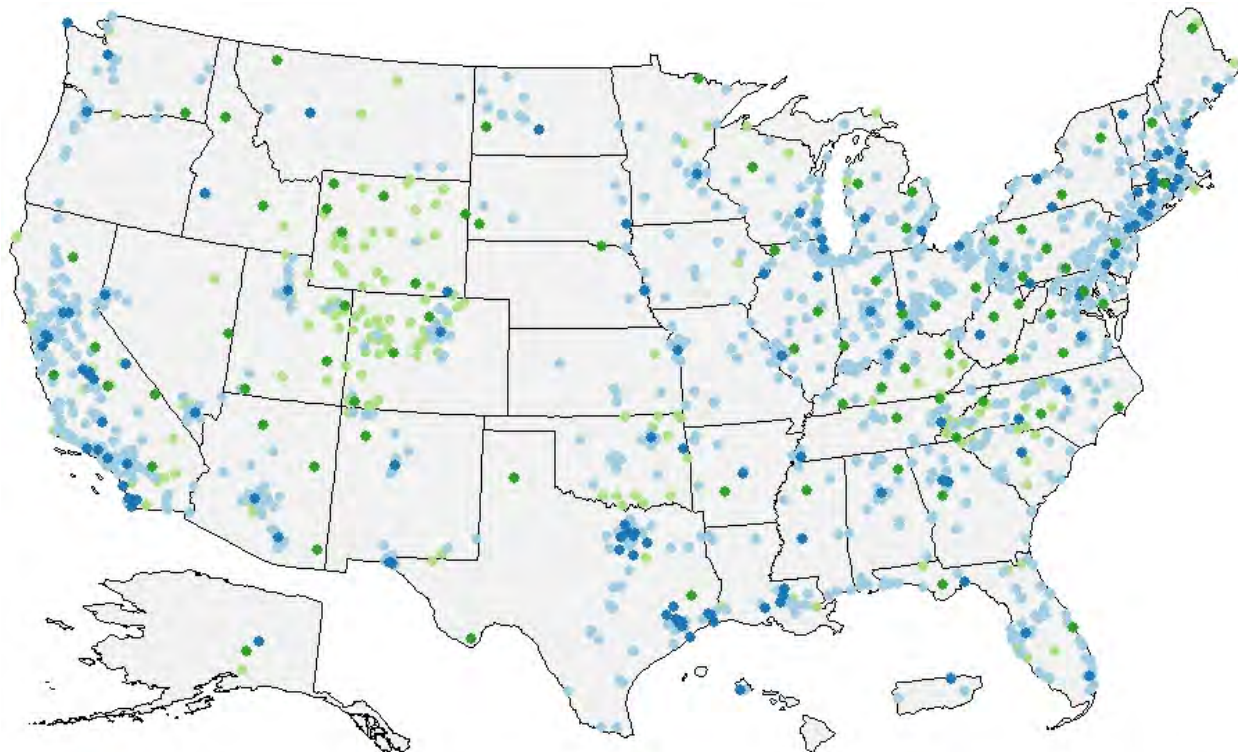
1 In 2020, there were over 1,300 federal, state, local, and tribal ambient air monitors
2 reporting O₃ concentrations to the EPA. Figure 2-7 shows the locations of such monitoring sites
3 that reported data to the EPA at any time during the 2018-2020 period. Nearly 80% of this
4 network are SLAMS monitors operated by state and local governments to meet regulatory
5 requirements and provide air quality information to public health agencies; these sites are largely
6 focused on urban and suburban areas.

7 Two important subsets of SLAMS sites separately make up the National Core (NCore)
8 multi-pollutant monitoring network and the Photochemical Assessment Monitoring Stations
9 (PAMS) network. Each state is required to have at least one NCore station, and O₃ monitors at
10 NCore sites are required to operate year-round. At each NCore site located in a MSA with a
11 population of 1 million or more (based on the most recent census), a PAMS network site is
12 required.¹⁶ In addition to reporting O₃ concentrations, the NCore and PAMS networks provide
13 data on O₃ precursor chemicals. The NCore sites feature co-located measurements of chemical
14 species such as nitrogen oxide and total reactive nitrogen, along with various meteorological
15 measurements. At a minimum, monitoring sites in the PAMS network are required to measure
16 certain O₃ precursors, such as NO_x and a target set of VOCs, during the months of June, July and
17 August, although some precursor monitoring may be required for longer periods of time to
18 improve the usefulness of data collected during an area's O₃ season (U.S. EPA, 2018a). The
19 enhanced monitoring at sites in these two networks informs our understanding of local O₃
20 formation.

21 While the SLAMS network has a largely urban and population-based focus, there are
22 monitoring sites in other networks that can be used to track compliance with the NAAQS in rural
23 areas. For example, the Clean Air Status and Trends Network (CASTNET) monitors are located
24 in rural areas. There were 84 CASTNET monitors operating in 2020, with most of the sites in the
25 eastern U.S. being operated by the EPA, and most of the sites in the western U.S. being operated
26 by the National Park Service (NPS). Finally, there are also a number of Special Purpose
27 Monitoring Stations (SPMs), which are not required but are often operated by air agencies for
28 short periods of time (less than 3 years) to collect data for human health and welfare studies, as
29 well as other types of monitoring sites, including monitors operated by tribes and industrial
30 sources. The SPMs are typically not used to assess compliance with the NAAQS.¹⁷

¹⁶ The requirements for PAMS, which were most recently updated in 2015, is fully described in section 5 of Appendix D to 40 CFR Part 58.

¹⁷ However, SPMs that use federal reference or equivalent methods, meet all applicable requirements in 40 CFR Part 58, and operate continuously for more than 24 months may be used to assess compliance with the NAAQS.



● SLAMS (961)
 ● NCORE/PAMS (126)
 ● CASTNET (84)
 ● SPM/OTHER (191)

1
2 **Figure 2-7. Map of U.S. ambient air O₃ monitoring sites reporting data to the EPA during**
3 **the 2018-2020 period.**

4 **2.3.2 Data Handling Conventions and Computations for Determining Whether the**
5 **Standards are Met**

6 To assess whether a monitoring site or geographic area (usually a county or urban area)
7 meets or exceeds a NAAQS, the monitoring data are analyzed consistent with the established
8 regulatory requirements for the handling of monitoring data for the purposes of deriving a design
9 value. A design value summarizes ambient air concentrations for an area in terms of the
10 indicator, averaging time and form for a given standard such that its comparison to the level of
11 the standard indicates whether the area meets or exceeds the standard. The procedures for
12 calculating design values for the current O₃ NAAQS (established in 2015) are detailed in
13 Appendix U to 40 CFR Part 50 and are summarized below.

14 Hourly average O₃ concentrations at the monitoring sites used for assessing whether an
15 area meets or exceeds the NAAQS are required to be reported in ppm to the third decimal place,
16 with additional digits truncated, consistent with the typical measurement precision associated
17 with most O₃ monitoring instruments. Monitored hourly O₃ concentrations flagged by the States
18 as having been affected by an exceptional event, having been the subject of a demonstration
19 submitted by the State, and having received concurrence from the appropriate EPA Regional

1 Office, are excluded from design value calculations consistent with 40 CFR 50.14.¹⁸ The hourly
2 concentrations are used to compute moving 8-hour averages, which are stored in the first hour of
3 each 8-hour period (e.g., the 8-hour average for the 7:00 AM to 3:00 PM period is stored in the
4 7:00 AM hour), and digits to the right of the third decimal place are truncated. Each 8-hour
5 average is considered valid if 6 or more hourly concentrations are available for the 8-hour period.

6 Next, the daily maximum 8-hour average (MDA8) concentration for each day is
7 identified as the highest of the 17 consecutive, valid 8-hour average concentrations beginning at
8 7:00 AM and ending at 11:00 PM (which includes hourly O₃ concentrations from the subsequent
9 day). MDA8 values are considered valid if at least 13 valid 8-hour averages are available for the
10 day, or if the MDA8 value is greater than the level of the NAAQS. Finally, the O₃ design value is
11 calculated as the 3-year average of the annual 4th highest MDA8 value¹⁹. An O₃ design value less
12 than or equal to the level of the NAAQS is considered to be valid if valid MDA8 values are
13 available for at least 90% of the days in the O₃ monitoring season (as defined for each state and
14 shown in Figure 2-6) on average over the 3 years, with a minimum of 75% data completeness in
15 any individual year. Design values greater than the level of the NAAQS are always considered to
16 be valid.

17 An O₃ monitoring site meets the NAAQS if it has a valid design value less than or equal
18 to the level of the standard, and it exceeds the NAAQS if it has a design value greater than the
19 level of the standard. A geographic area meets the NAAQS if all ambient air monitoring sites in
20 the area have valid design values meeting the standard. Conversely, if one or more monitoring
21 sites has a design value exceeding the standard, then the area exceeds the NAAQS.

22 **2.4 O₃ IN AMBIENT AIR**

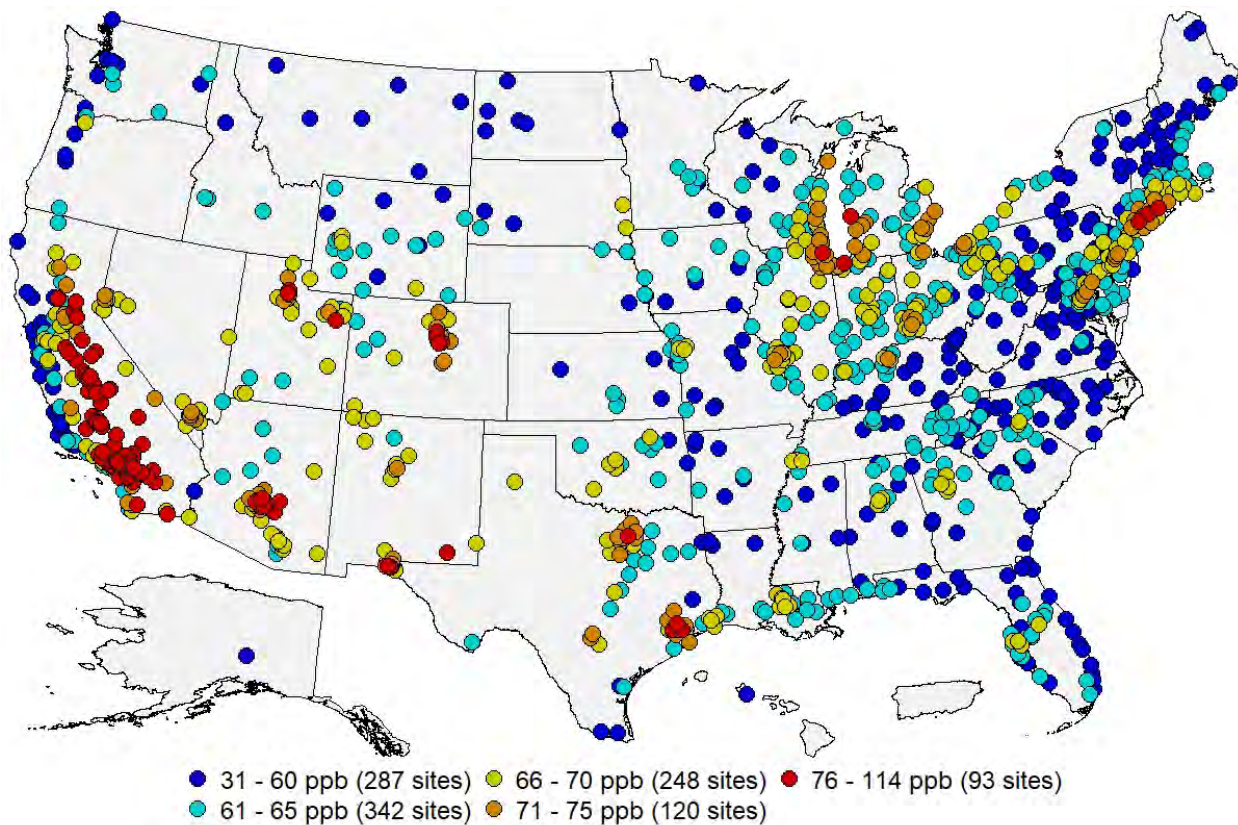
23 **2.4.1 Concentrations Across the U.S.**

24 Figure 2-8 below shows a map of the O₃ design values at U.S. ambient air monitoring
25 sites based on data from the 2018-2020 period. From the figure it is apparent that many
26 monitoring sites have recent design values exceeding the current NAAQS, and that most of these
27 sites are located in or near urban areas. The highest design values are located in California,
28 Texas, along the shoreline of Lake Michigan, and near large urban areas in the northeastern and
29 western U.S. There are also high design values associated with wintertime O₃ in the Uinta Basin
30 in Utah. The lowest design values are located in the north central region of the U.S., rural parts

¹⁸ A variety of resources and guidance documents related to identification and consideration of exceptional events in design value calculations are available at <https://www.epa.gov/air-quality-analysis/final-2016-exceptional-events-rule-supporting-guidance-documents-updated-faqs>.

¹⁹ Design values are reported in ppm to the third decimal place, with additional digits truncated. This truncation step also applies to the initially calculated 8-hour average concentrations (Appendix 2A, section 2A.1).

1 of New England and the southeastern U.S., and along the Pacific Ocean, including Alaska and
2 Hawaii.



3
4 **Figure 2-8. O₃ design values in ppb for the 2018-2020 period.**

5 **2.4.2 Trends in U.S. O₃ Concentrations**

6 Figure 2-9 shows a map of the site-level trends in the O₃ design values at U.S. monitoring
7 sites having complete data²⁰ from 2000-2002 through 2018-2020. The trends were computed
8 using the Thiel-Sen estimator (Sen, 1968; Thiel, 1950), and tests for significance were computed
9 using the Mann-Kendall test (Kendall, 1948; Mann, 1945). From this figure it is apparent that
10 design values have decreased significantly over most of the eastern U.S. during this period.
11 These decreases are in part due to EPA regulations aimed at reducing NO_x emissions from
12 EGUs, such as the Clean Air Interstate Rule and the Cross-State Air Pollution Rule, with the goal
13 of achieving broad, regional reductions in summertime NO_x emissions; as well as mobile
14 emission reductions from federal motor vehicle emissions and fuel standards, and; local controls
15 resulting from implementation of the existing O₃ standards. Other areas of the country have also

²⁰ The data completeness criteria for Figure 2-8 through Figure 2-14 are listed in Table 2A-1 of Appendix 2A.

1 experienced decreases in design values, most notably in California and near urban areas in the
2 intermountain west.

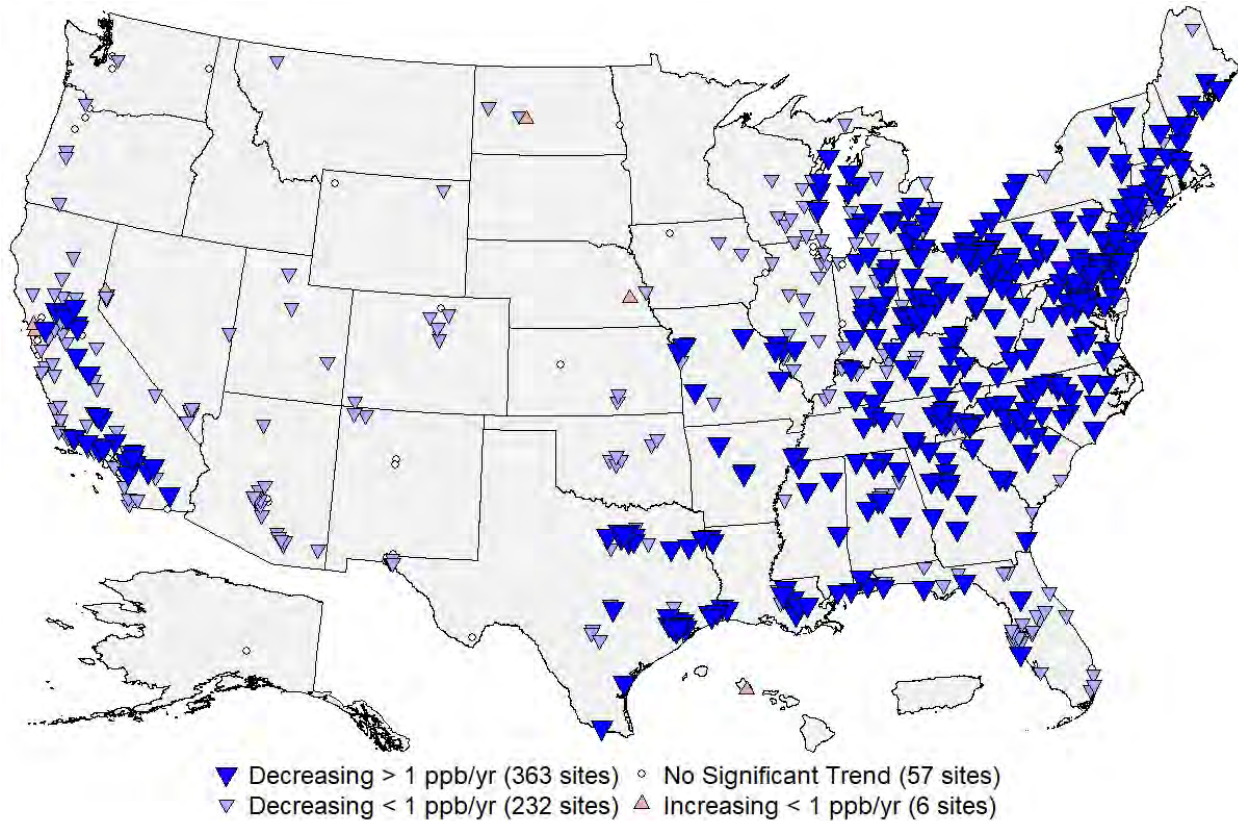
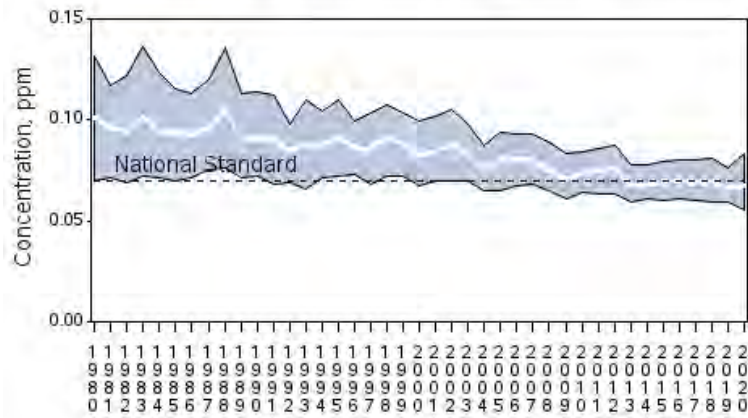


Figure 2-9. Trends in O₃ design values based on data from 2000-2002 through 2018-2020.

Figure 2-10 shows the national trend in the annual 4th highest MDA8 values based on 188 ambient air monitoring sites with complete data from 1980 to 2020. This figure shows that, on average, there has been a 33% decrease in U.S. annual 4th highest MDA8 levels since 1980. Since relatively few sites have been monitoring continuously since 1980, Figure 2-11 shows the national trend in the annual 4th highest MDA8 values and the design values based on the 822 monitoring sites with complete data from 2000 to 2020. The U.S. median annual 4th highest MDA8 values decreased by 25% nationally from 2002 (88 ppb) to 2013 (66 ppb), with some variability among individual years in this period which can partially be attributed to changes in meteorological conditions. Similarly, the U.S. median design value decreased by 20% from 2000-2002 (84 ppb) to 2013-2015 (67 ppb). The trend in the annual 4th highest MDA8 concentrations was relatively flat from 2013 to 2018, with decreases occurring in 2019 and 2020. The design values have been relatively constant since 2015, though there are slight decreases in 2019 and 2020. In general, the design value metric is more stable and therefore better reflects long-term changes in O₃ than the annual 4th highest MDA8 metric.

Ozone Air Quality, 1980 - 2020
 (Annual 4th Maximum of Daily Max 8-Hour Average)
 National Trend based on 188 Sites

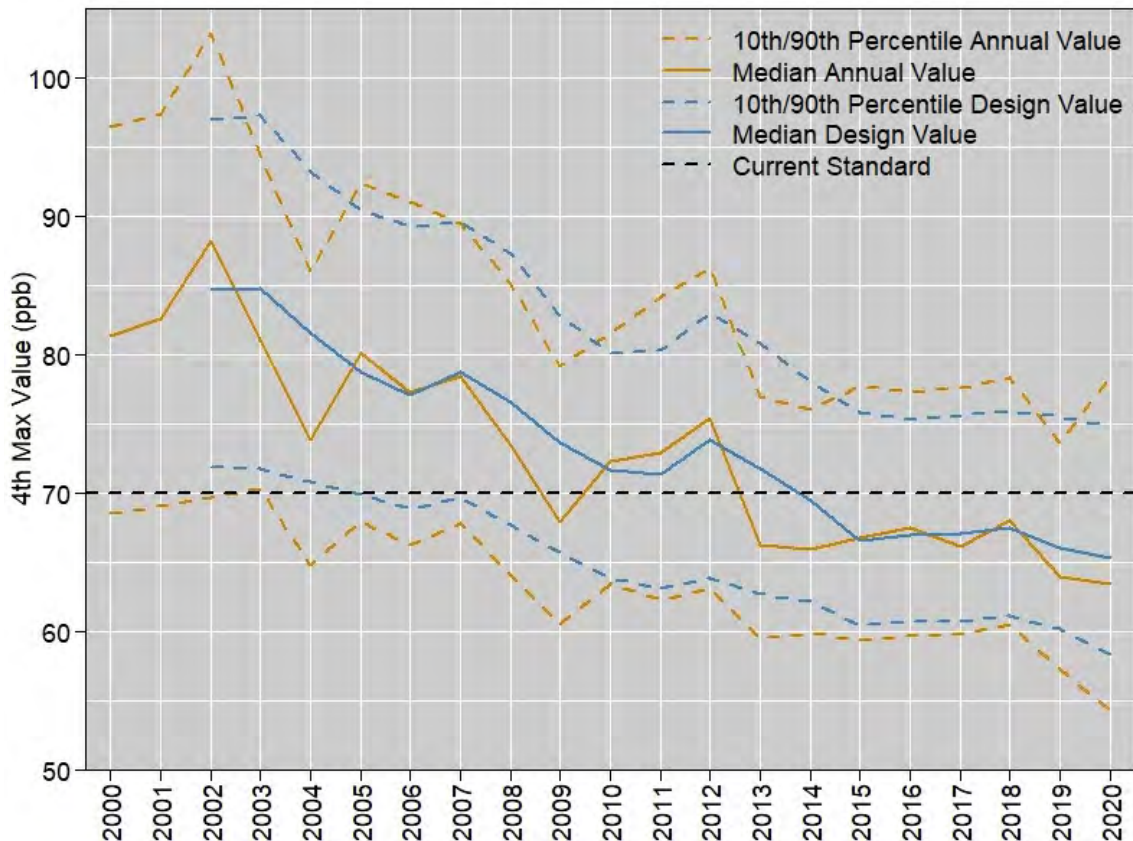


1980 to 2020 : 33% decrease in National Average

Source: EPA's Air Trends website (<https://www.epa.gov/air-trends/ozone-trends/>).

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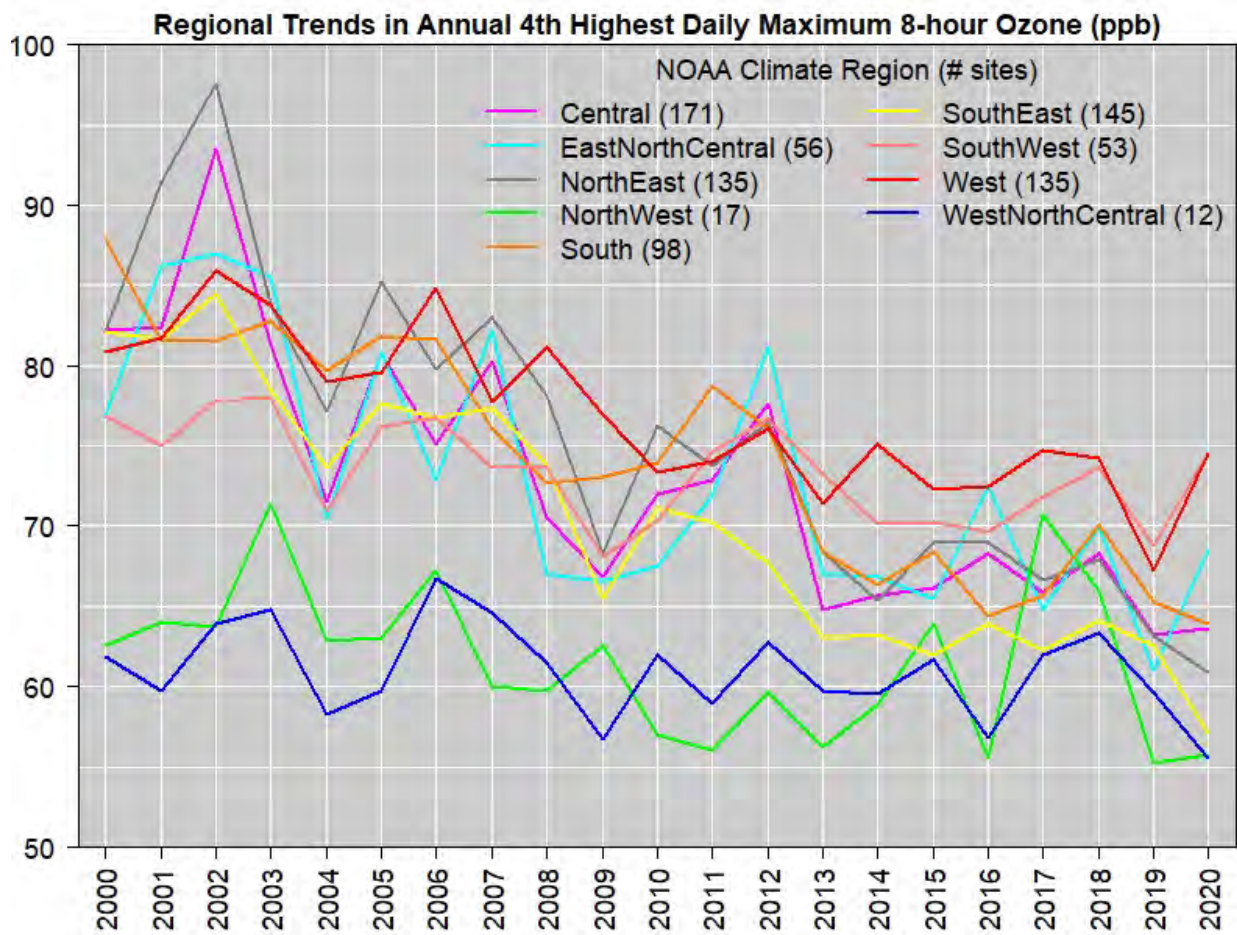
Figure 2-10. National trend in annual 4th highest MDA8 values, 1980 to 2020. The white center line is the average while the filled area represents the range between the 10th and 90th percentiles. The dotted line is the level of the standard.



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Figure 2-11. National trend in annual 4th highest MDA8 concentrations and O₃ design values in ppb, 2000 to 2020.

1 Figure 2-12 shows regional trends in the median annual 4th highest MDA8 values for the
 2 9 National Oceanic and Atmospheric Administration (NOAA) climate regions²¹ based on
 3 ambient air monitoring sites with complete O₃ monitoring data for 2000-2020. The five eastern
 4 U.S. regions (Central, East North Central, Northeast, Southeast, South) have all shown decreases
 5 of at least 10 ppb in median annual 4th highest MDA8 values since the early 2000's, with the
 6 Southeast region in particular showing the largest decrease of over 20 ppb. In contrast, the
 7 median annual 4th highest MDA8 values have changed by less than 10 ppb in each of the four
 8 western U.S. regions (Northwest, Southwest, West, West North Central). The large increase in
 9 the Northwest region in 2017 and 2018 correspond to years with historically high wildfire
 10 activity.



11
 12 **Figure 2-12. Regional trends in median annual 4th highest MDA8 concentrations, 2000 to**
 13 **2020.**

²¹ These regions are defined per Karl and Koss (1984) as illustrated in Appendix 2B, Figure 2B-1.

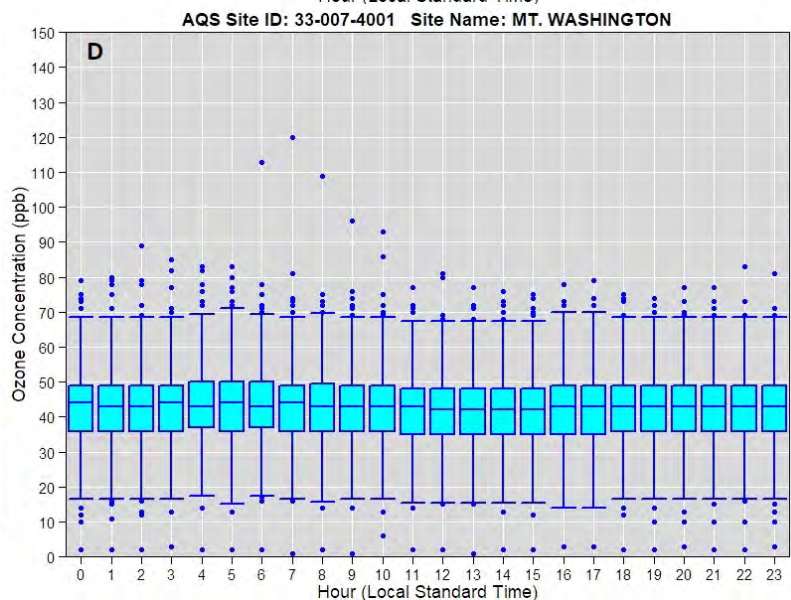
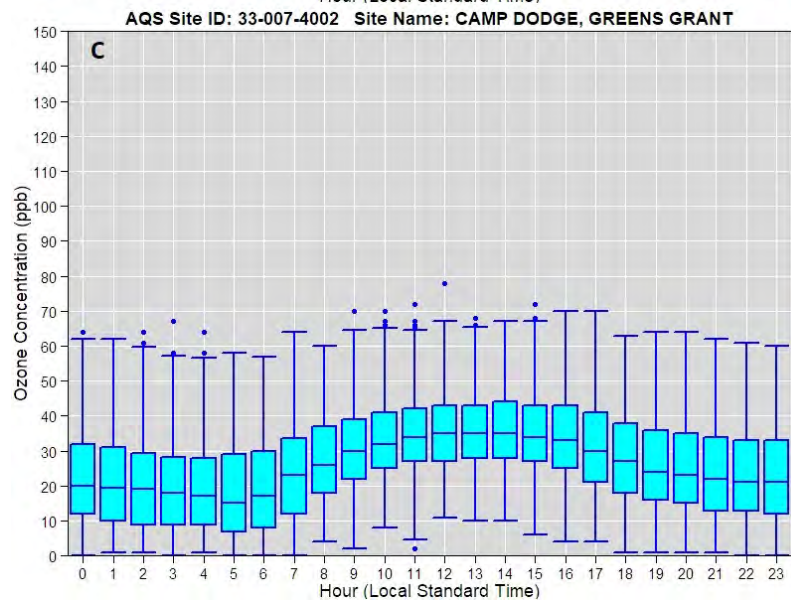
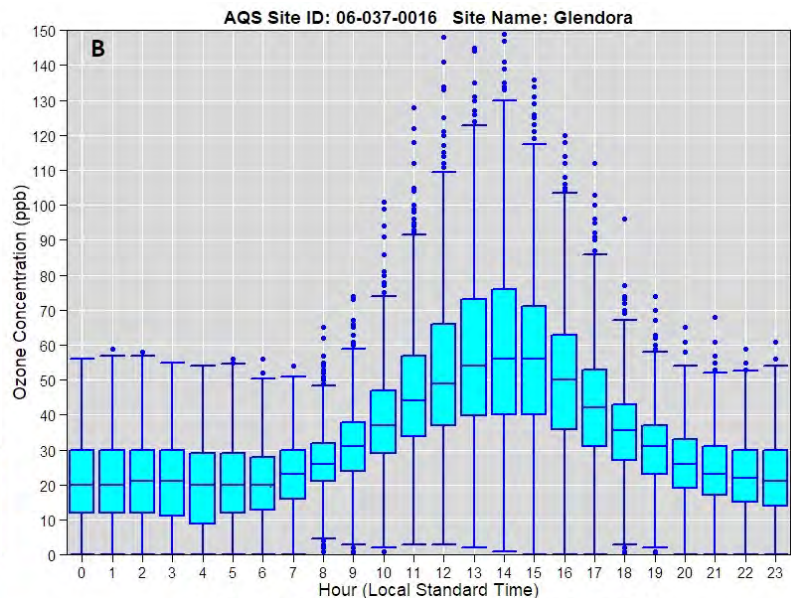
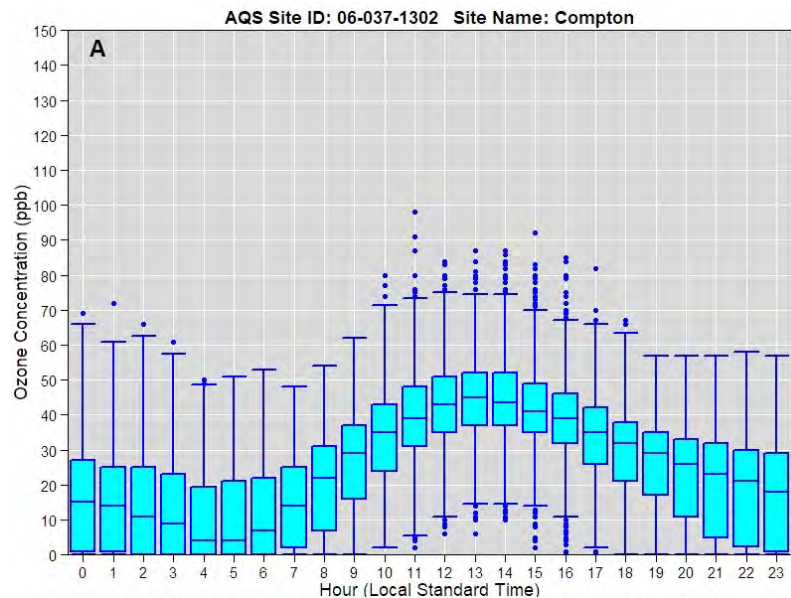
1 Trends presented in this section have focused on annual 4th high MDA8 concentrations
2 and design values. Additional information from the published literature has examined trends in
3 MDA8 concentrations across the distribution of high and low O₃ days. Simon et al., 2015) found
4 that, similar to results presented in this section for DVs and annual 4th high MDA8
5 concentrations, the 95th percentile of summertime MDA8 concentrations decreased significantly
6 at most sites across the U.S. between 1998 and 2013. In contrast, trends over that time period for
7 the 5th percentile, median and mean of MDA8 varied with location and time of year. Similarly,
8 Lefohn et al. (2017) reported that between 1980 and 2014 there was a compression of the
9 distribution of measured hourly O₃ values with extremely high and extremely low concentrations
10 becoming less common. As a result, O₃ metrics impacted by high hourly O₃ concentrations, such
11 as the annual 4th highest MDA8 value, decreased at most U.S. sites across this period.
12 Concurrently, metrics that are impacted by averaging longer time periods of hourly O₃
13 measurements, such as the 6-month (April-September) average of daytime (8am-7pm) O₃
14 concentrations, were more varied with only about half of the sites exhibiting decreases in this
15 metric and most other sites exhibiting no trend (Lefohn et al., 2017).

16 **2.4.3 Diurnal Patterns**

17 Tropospheric O₃ concentrations in most locations exhibit a diurnal pattern due to the
18 photochemical reactions that drive formation and destruction of O₃ molecules. Figure 2-13
19 shows boxplots of O₃ concentrations in ambient air, by hour of the day for four monitoring sites
20 that represent diurnal patterns commonly observed in the U.S. The boxes represent the 25th
21 percentile, median, and 75th percentiles and each box has “whiskers” which extend up to 1.5 times
22 the interquartile range (i.e., the 75th percentile minus the 25th percentile) from the box, and dots which
23 represent outlier values. The top panels show diurnal patterns, based on available data from 2015-
24 2017, at urban (panel A) and downwind suburban (panel B) monitoring sites in the Los Angeles
25 metropolitan area. Both sites generally measure their highest O₃ concentrations during the early
26 afternoon hours, and their lowest concentrations during the early morning hours, as is typical of
27 most urban and suburban areas in the U.S. However, higher levels of NO_x emissions near the
28 urban site may suppress O₃ formation throughout the day and increase the O₃ titration rate at
29 night, resulting in lower O₃ concentrations than those typically observed at the downwind site.

30 Ozone concentrations are generally lower in rural areas than in urban and suburban areas,
31 with less pronounced diurnal patterns. However, elevation and transport also play a larger role in
32 influencing concentrations in rural areas than in urban areas. The bottom panels in Figure 2-13
33 show diurnal patterns at low elevation (panel C) and high elevation (panel D) rural monitoring
34 sites in New Hampshire. The low elevation site experiences O₃ concentrations that are 10-20 ppb
35 lower, on average, than at the high elevation site. Ozone concentrations at the low elevation site

1 exhibit a slight diurnal pattern similar to that seen at the urban and suburban sites (generally
2 related to photochemical O₃ formation that increases concentrations in the late morning and
3 afternoon), while O₃ concentrations at the high elevation site do not exhibit any diurnal pattern.
4 The lack of a diurnal pattern observed at the high elevation site is typical of high elevation rural
5 sites throughout the U.S., suggesting that observed O₃ concentrations at such sites are primarily
6 driven by transport from upwind areas rather than being formed from local precursor emissions.
7 The presence of peak O₃ concentrations that are higher at the high elevation site than at the low
8 elevation site at all hours of the day indicates that the high elevation site may be influenced by
9 transport from the free troposphere to a greater extent than the low elevation site.
10



1
 2 **Figure 2-13. Diurnal patterns in hourly O₃ concentrations at selected monitoring sites: A) an urban site in Los Angeles; B) a**
 3 **downwind suburban site in Los Angeles; C) a low elevation rural site in New Hampshire; and D) a high elevation**
 4 **rural site in New Hampshire.**

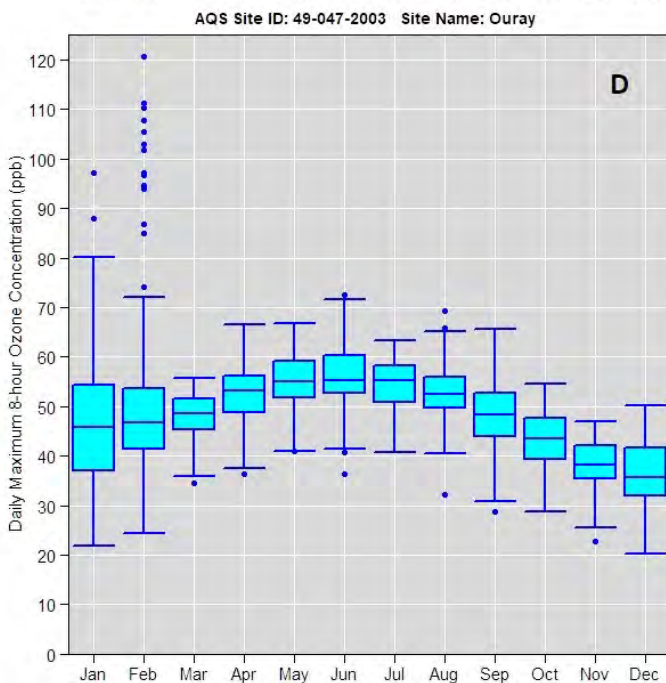
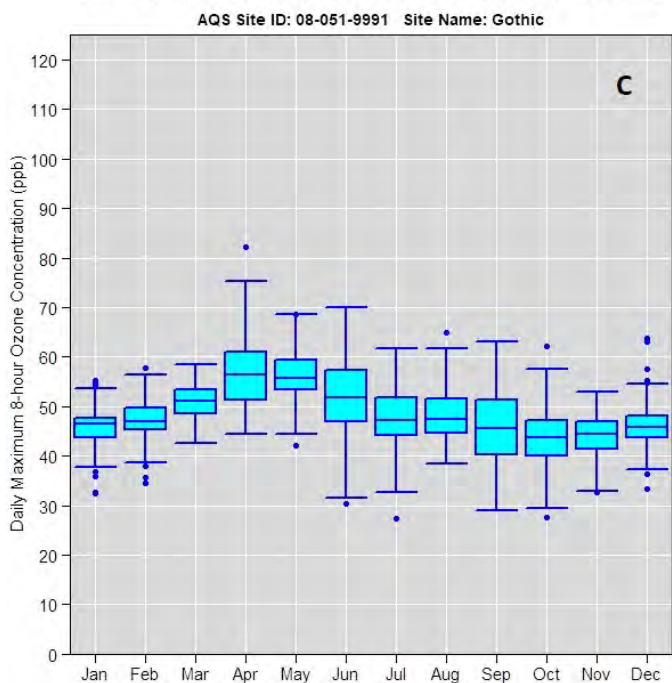
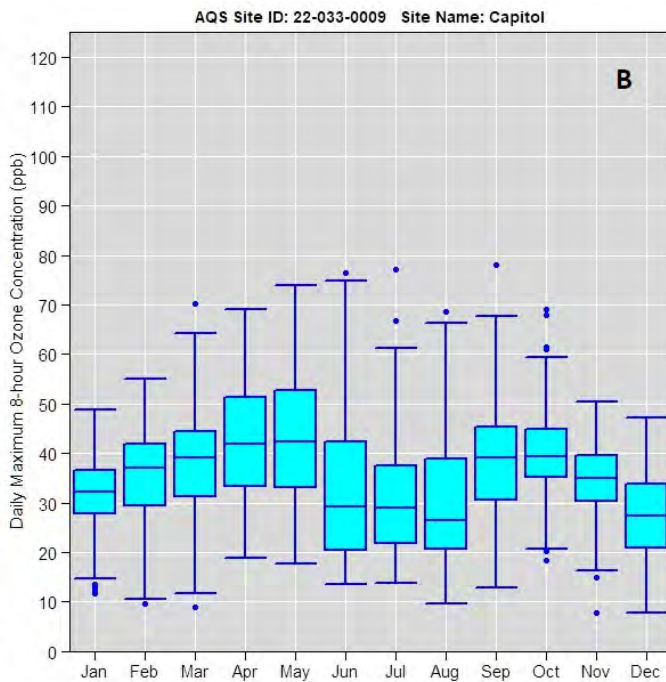
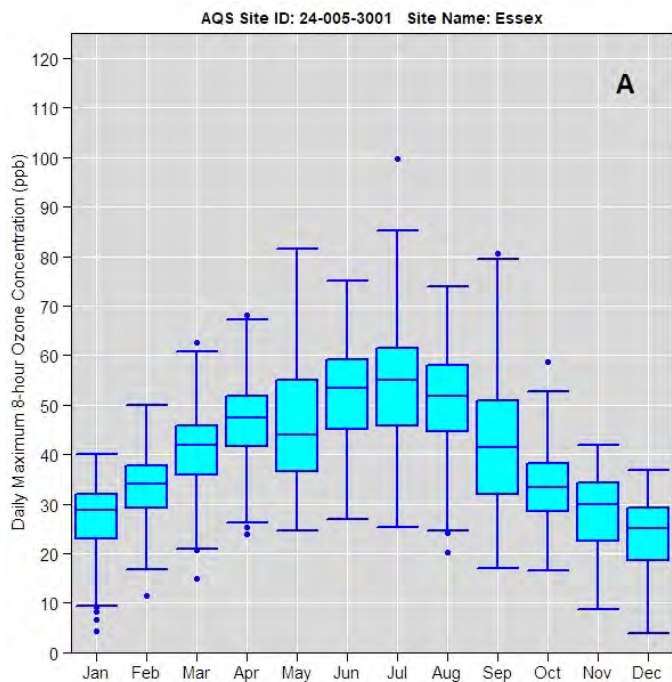
1 **2.4.4 Seasonal Patterns**

2 Tropospheric O₃ concentrations also tend to experience seasonal patterns due to seasonal
3 changes in meteorological conditions and the length and intensity of daylight. High O₃
4 concentrations are most commonly observed on hot, sunny, and stagnant days during the spring
5 and summer. Figure 2-14 shows boxplots of MDA8 O₃ concentrations by month of the year for
6 four monitoring sites that represent different kinds of seasonal patterns commonly observed in
7 the U.S. This figure is based on data from 2015-2017. The boxes represent the 25th percentile,
8 median, and 75th percentiles and each box has “whiskers” which extend up to 1.5 times the
9 interquartile range (i.e., the 75th percentile minus the 25th percentile) from the box, and dots which
10 represent outlier values. Panel A shows the seasonal pattern for an urban site in Baltimore, MD,
11 which reflects the typical seasonal pattern observed at many urban and suburban monitoring sites
12 across the U.S. The highest O₃ concentrations are observed during May to September, when the
13 days are the longest and solar radiation is strongest.

14 Panel B shows the seasonal pattern for an urban site in Baton Rouge, LA. In parts of the
15 southeastern U.S., the highest O₃ concentrations are often observed in April and May due to the
16 onset of warm temperatures combined with abundant emissions of biogenic VOCs at the start of
17 the growing season. This is often followed by lower concentrations during the summer months,
18 which is associated with high humidity levels that tend to suppress O₃ formation in the region
19 (Camalier et al., 2007). Some areas, particularly in the states bordering the Gulf of Mexico, may
20 experience a second peak in O₃ concentrations in September and October.

21 Panel C shows the seasonal pattern for a high elevation rural site in Colorado. The
22 highest O₃ concentrations in rural areas are typically observed in the spring. This can be due to
23 several factors, including those mentioned previously, and additionally, long-range transport
24 from Asia is most prevalent at this time of year. Stratospheric Tropospheric Exchange events,
25 which most often affect high elevation areas in the western U.S., are also most common during
26 the spring.

27 Finally, Panel D shows the seasonal pattern for a monitoring site in Utah where high
28 wintertime O₃ concentrations were observed. Over the past decade, high O₃ concentrations have
29 been observed in two mountain basins in the western U.S. during the winter months (December
30 to March). These wintertime O₃ episodes require a unique set of conditions, including a shallow
31 inversion layer, snow cover, calm or light winds, and pervasive local NO_x and VOC emissions
32 (in these cases, from oil and gas extraction). These conditions are relatively uncommon, and
33 elevated wintertime O₃ levels may not occur in some years.



2 **Figure 2-14. Seasonal patterns in MDA8 O₃ concentrations at selected monitoring sites**
 3 **(2015-2017): A) an urban site in Baltimore, MD; B) an urban site in Baton**
 4 **Rouge, LA; C) a rural site in Colorado; and D) a site in Utah experiencing**
 5 **high wintertime O₃.**

6

2.4.5 Variation in Recent Daily Maximum 1-hour Concentrations

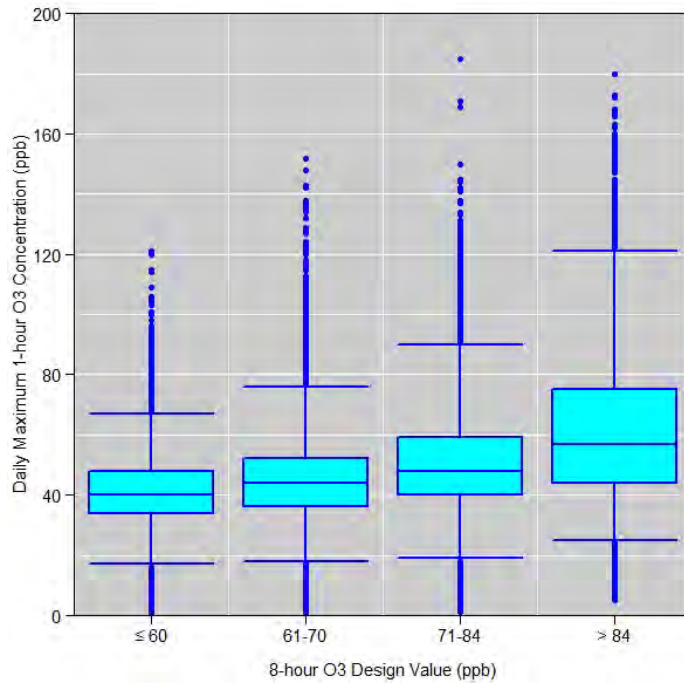
To provide a characterization of recent O₃ concentrations in the U.S. for periods shorter than 8 hours, this section presents recent O₃ monitoring data in terms of daily maximum 1-hour average (MDA1) concentrations, and their variation across monitoring sites that vary with regard to design values for the current O₃ standards.

Figure 2-15 shows boxplots of MDA1 values at U.S. monitoring sites based on 2018-2020 data stratified by each site's 8-hour O₃ design value. The boxes representing the 25th percentile, median, and 75th percentile MDA1 values increase slightly with higher design values. Although the overall range (minimum and maximum) of observed MDA1 values does not appear to change much, there is an increasing presence of higher MDA1 values extending up to around 160 ppb for the rightmost bin which includes only sites that exceed the current standards. The upper percentiles, including the 75th and the 99th percentiles (represented by top of box and upper whisker, respectively), in particular, are increased for the sites that do not meet the current standards (up to nearly 80 ppb and 120 ppb in the rightmost bin). In contrast, the boxplots show that there are only a small fraction of MDA1 values above 120 ppb for sites that meet the current standards.

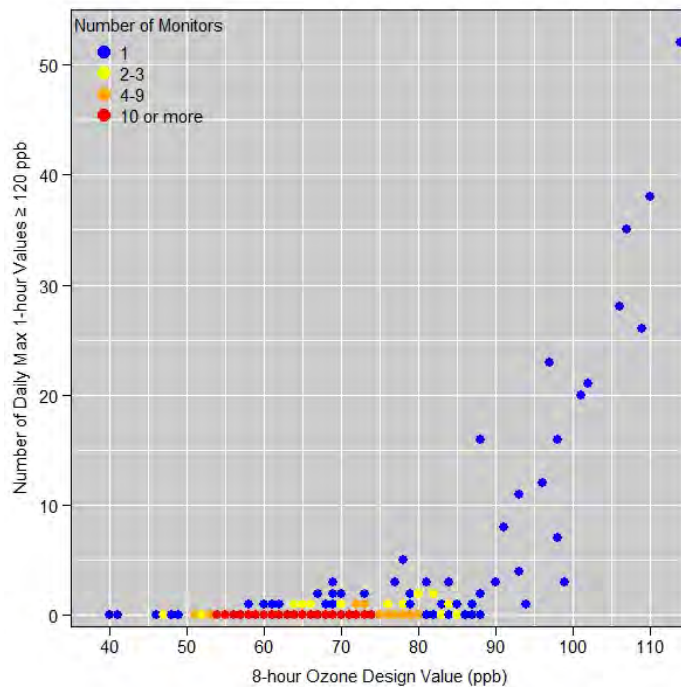
Figure 2-16 shows a scatter plot of the number of days at each monitoring site that have a MDA1 value of 120 ppb or greater based on 2018-2020 data compared to the site's 2018-2020 design value. According to the figure, a small proportion of O₃ monitoring sites in the U.S. observe MDA1 values at or above 120 ppb more than once per year, but these sites all exceed the current 8-hour standards. There are no sites that were meeting the current standards based on 2018-2020 data that had MDA1 values above 120 ppb more than three times over the same 3-year period (Appendix 2A, Table 2A-2).

Figure 2-17 shows the national trend in the annual 2nd highest MDA1 O₃ concentration, which was the metric used to track progress towards meeting the 1-hour O₃ NAAQS, originally set in 1979 and later replaced by the current 8-hour metric in 1997 (62 FR 38856, July 18, 1997).²² The monitoring sites represented in Figure 2-17 are the 834 sites with complete data from 2000 to 2020 (as summarized in Appendix 2A, Section 2A.2). The shapes of the trend lines in Figure 2-17 are similar to those shown for the annual 4th highest MDA8 values in Figure 2-11. The national median annual 2nd highest MDA1 value decreased by 27% from 2002 (105 ppb) to 2013 (77 ppb), which is comparable to the decrease observed in the national median annual 4th highest MDA8 value (25%) during the same period.

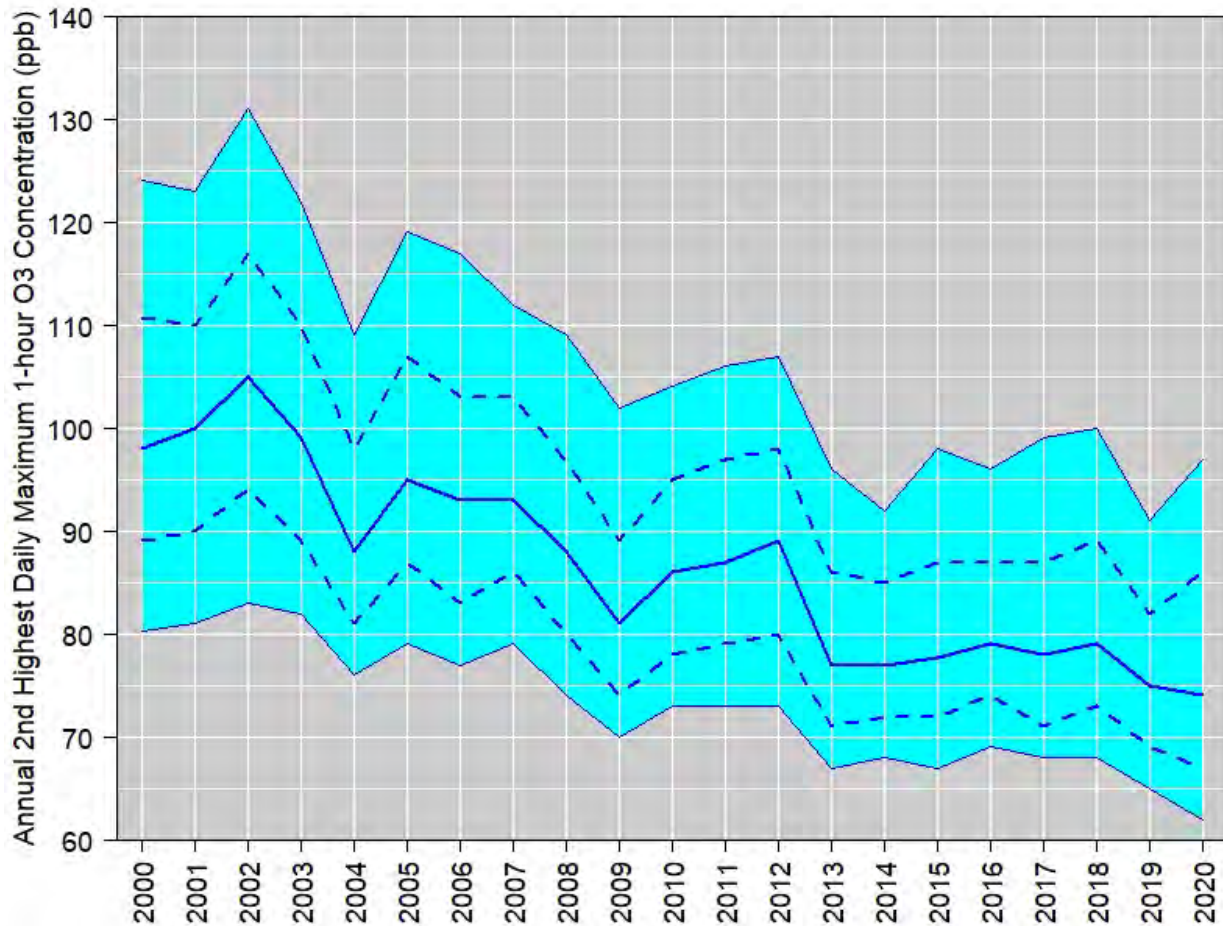
²² The 1-hour O₃ standards were formally revoked in 2005 (70 FR 44470, August 3, 2005).



1
 2 **Figure 2-15. Boxplots showing the distribution of MDA1 concentrations (2018-2020),**
 3 **binned according to each site's 2018-2020 design value.**



4
 5 **Figure 2-16. Number of days in 2018-2020 at each monitoring site with a MDA1**
 6 **concentration greater than or equal to 120 ppb compared to its 8-hour design**
 7 **value in ppb.**



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Figure 2-17. National trend in the annual 2nd highest MDA1 O₃ concentration, 2000 to 2020. The solid blue line represents the median value, dotted blue lines represent the 25th and 75th percentile values, and the light blue shaded area represents the range from the 10th to the 90th percentile values.

2.5 BACKGROUND O₃

There are a number of definitions of background O₃ used in various contexts that differ by the specific emissions sources and/or natural processes the definition includes (e.g., see ISA, Appendix 1, section 1.2.2). In this reconsideration, as in past reviews, the EPA generally characterizes O₃ concentrations that would exist in the absence of U.S. anthropogenic emissions as U.S. background (USB). An alternative phrasing for USB is the O₃ concentrations created collectively from global natural sources and from anthropogenic sources existing outside of the U.S. Such a definition helps distinguish the O₃ that can be controlled by precursor emissions reductions within the U.S. from O₃ originating from global natural and foreign precursor sources that cannot be controlled by U.S. regulations (ISA, section 1.2.2).

Because monitors cannot distinguish the origins of the O₃ they measure,²³ photochemical grid models have been widely used to estimate the contribution of background sources to observed surface O₃ concentrations. This section summarizes results of a state-of-the-science modeling analysis to estimate the magnitude of present-day USB and its various components. Conceptually, these USB estimates represent O₃ concentrations that occur as a result of global natural sources (or processes, see section 2.5.1 for more details) and those anthropogenic sources existing outside the U.S., i.e., the O₃ concentrations that would occur in the absence of any U.S. anthropogenic O₃ precursor emissions. Modeling results summarized in this section include average estimates of MDA8 USB concentrations for several temporal periods including seasons. Average USB estimates are also presented for days on which the total model-predicted MDA8 O₃ concentration was greater than either 60 ppb or 70 ppb, and for the days on which the 4th-highest MDA8 O₃ concentration was predicted to occur. Additionally, this modeling analysis investigated the contributions to USB of some specific groups of sources, such as international anthropogenic sources, and how those contributions vary by season and by location.

The section, which presents the information and analysis that were also presented in the parallel section of the 2020 PA, is organized as follows. Section 2.5.1 provides an overview of the various sources that contribute to USB, including currently available information on the magnitude, seasonal variability, and spatial variability of their contributions to USB. Section 2.5.2 summarizes the methodology for the modeling analyses used to quantify USB and component contributions. More detailed information about the modeling methodology is presented in Appendix 2B. Section 2.5.3 summarizes USB estimates using methodology

²³ Ozone concentrations that do not include contributions from U.S. anthropogenic emissions cannot be determined exclusively from O₃ measurements because even relatively remote monitoring sites in U.S. receive transport of U.S. anthropogenic O₃ from other locations.

1 described in section 2.5.2, including estimates specific to certain subgroups of sources. Section
2 2.5.4 summarizes key findings of the analyses.

3 **2.5.1 Summary of U.S. Background O₃ Sources**

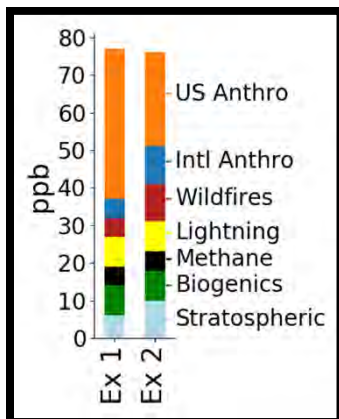
4 Jaffe et al. (2018) reviewed the literature on sources that contribute to USB. While the
5 term “background” may imply a low concentration well-mixed²⁴ environment, background
6 sources can create well-defined plumes and/or contribute to the well-mixed environment. The
7 USB definition, which is based on sources, includes both the well-mixed environment and more
8 well-defined plumes. Figure 2-18a (adapted from Jaffe et al. (2018)) illustrates sources of USB
9 O₃ (blue) and U.S. anthropogenic sources of O₃ (yellow). Figure 2-18b shows two theoretical
10 examples where background sources contribute to the total ground-level O₃. The first example
11 (Ex 1) highlights a typical monitoring site with lower USB, and the second example (Ex 2)
12 presents a scenario in which USB is a large contributor. Both examples oversimplify methane,
13 which has both natural and anthropogenic and both domestic and foreign contributions. Source
14 contributions to USB vary in space and time, and the stacked bar plot in this figure
15 oversimplifies the complex relationship between USB and total O₃. Even so, USB sources can
16 broadly be discussed as global natural sources (see sections 2.5.1.1 to 2.5.1.6) and international
17 anthropogenic sources (see section 2.5.1.7). In the simplest interpretation, the natural sources are
18 background regardless of where they occur, or which definition of background is being used
19 (e.g., USB or natural background²⁵). By contrast, ozone formed from anthropogenic emissions is
20 only considered as background when the emissions sources are not from sources within the focus
21 area. However, this paradigm is complicated by the fact that many sources of O₃ precursors are
22 the result of interactions between human and natural systems (for instance forest management
23 practices can impact both biogenic VOC emissions from trees and wildfires). In the context of
24 USB, anthropogenic background is synonymous with O₃ originating from international
25 anthropogenic emission sources. The relative contribution of international and natural
26 background sources can vary dramatically from place to place and are most notably larger at
27 locations near borders (international) or high elevation (natural). At non-border locations and
28 many border locations, the natural background is usually the dominant background source.
29

²⁴ We use the term “well-mixed” here to refer to conditions when the contributions from various types of sources are mixed due to chemistry or physical processes to the point where it is not possible to discern the contribution to O₃ from each individual source.

²⁵ Natural background is the O₃ that would exist in the absence of anthropogenic emission sources.



(a)



(b)

(a) U.S. O₃ sources shown with yellow boxes or arrows represent domestic sources. Sources shown with blue boxes or arrows represent USB sources. Note that locations for each process are not specific to any one region. The base map shows satellite-observed tropospheric NO₂ columns for 2014 from the Ozone Monitoring Instrument (OMI) onboard the NASA Aura satellite (Credit: NASA Goddard's Scientific Visualization Studio/T. Schindler). NO₂ column amounts are relative with red colors showing highest values, followed by yellow then blue. We use the OMI NO₂ columns as a proxy to show local O₃ precursor emission sources. (b) The bar chart shows two theoretical examples of USB O₃ contributions combine with domestic sources to produce elevated O₃ at a specific location on any given day. Each source varies daily and there are also nonlinear interactions between USB O₃ sources and anthropogenic sources that can further add to O₃ formation, e.g., wildfires and urban anthropogenic emissions (e.g., Singh et al., 2012). Minor adaptation from DOI: <https://doi.org/10.1525/elementa.309.f1>

1 **Figure 2-18. Conceptual models for O₃ sources: (a) in the U.S., and (b) at a single location.**

2 The natural and anthropogenic sources of background O₃ vary by location and by season.
 3 Emissions from anthropogenic sources largely occur in the same areas year after year. Natural
 4 sources of O₃ and precursors, on the other hand, vary both in magnitude and in location from day
 5 to day and year to year. As a result, certain types of natural sources may have large O₃
 6 contributions measured at a monitor at one point in time but not at other times. The combination
 7 of varying proximity and magnitude means that natural sources can contribute to background in
 8 the form of localized plumes of elevated O₃ that contribute to O₃ at monitoring sites on an
 9 episodic basis. In the absence of locally well-defined plumes, global natural and international
 10 anthropogenic sources are constantly contributing to the well-mixed background.

1 USB varies by location and by season due to both the nature of sources and the loss
2 processes. The nature of emission sources leads to seasonal and spatial patterns that will be
3 described further below. The contribution of these sources is modulated by transport patterns that
4 interact with deposition and chemical losses. For illustration, two emission sources of identical
5 magnitudes may have different contributions if one emits near the surface in summer and the
6 other emits in the free troposphere in spring. Warmer moister air in the summer at the surface
7 enhances O₃ chemistry losses and deposition of O₃ to the surface increases losses further. In
8 contrast, cooler, drier temperatures in the spring and free troposphere lengthen O₃ lifetimes and
9 faster winds in the free troposphere enable longer transport. The seasonality of temperature and
10 transport patterns gives O₃ USB a distinct seasonal cycle that results from both sinks and
11 sources.

12 The sections below summarize the state of the science estimates of USB contributions.
13 Each source type is described with respect to its seasonality as well as its local vs well-mixed
14 contribution potential. Jaffe et al. (2018) reviewed contributions of various sources to USB O₃
15 from modeling studies and the references therein are used to illustrate the range of O₃
16 contributions from each source. The literature-based estimate ranges provide context to the
17 estimates of USB that are reported in section 2.5.3.

18 **2.5.1.1 Stratosphere**

19 The only direct source of O₃ to the troposphere with appreciable contributions to O₃
20 concentrations is STE (other sources are indirect via precursors). STE occurs when stratospheric
21 air, which is relatively rich in O₃, is transported across the tropopause where it enhances
22 tropospheric concentrations. Most STE events create enhancements that do not immediately
23 reach the surface. Instead, STE-enhanced O₃ mixes into the free troposphere where it is
24 dispersed. In cases when the transported air reaches the surface before enough dispersion occurs,
25 it creates a localized plume of O₃ referred to as a Stratospheric Ozone Intrusion (SOI). The total
26 stratospheric contribution includes both the well-mixed contribution from the distant stratosphere
27 exchanges as well as any localized SOI plume.

28 The total global O₃ flux from the stratosphere to the troposphere is estimated at 510±90
29 teragrams per year (Tg/y) compared to 4620±600 Tg/y (post-2000 literature in Table 2 in Wu et
30 al., 2007) produced within the troposphere. The majority of the earth's surface is outside the U.S.
31 and only STE that take place over the U.S. are likely to create a large magnitude local
32 enhancement at a U.S. monitor.²⁶ A SOI that occurs outside the U.S. would likely be dispersed

²⁶ Recently methods have been developed for identifying and estimating SOIs that have clear localized contributions to O₃ concentrations with the potential to contribute to standards' exceedances. These are described in documents available at: <https://www.epa.gov/air-quality-analysis/guidance-preparation-exceptional-events-demonstrations-stratospheric-ozone>.

1 into the well-mixed background and reduced through chemical loss and deposition before it
2 reaches many monitors.

3 Modeling and observational studies show that SOI can episodically contribute large
4 amounts of O₃ at a subset of U.S. monitors, but stratospheric mixing more frequently contributes
5 smaller quantities of O₃. Modeling studies focused on seasons with frequent SOI find median
6 total stratospheric contributions to MDA8 are 10-22 ppb in the West and 3-13 ppb in the East
7 with episodic contributions up to 40 ppb mostly in the West (Table S2, Jaffe et al., 2018).
8 Because these studies focus on the most active season, these medians are expected to be upper
9 bounds for the annual average. Further, SOI are most common in the spring when MDA8 O₃
10 concentrations above 70 ppb are less common (ISA, section 1.3.2).

11 **2.5.1.2 Biogenic VOC**

12 Biogenic VOCs are the quintessential “natural” source of O₃ precursors. At global scales,
13 biogenic sources are the largest contributor to VOCs – even though local anthropogenic sources
14 of highly reactive VOCs can be very important in some areas. VOCs are also an important
15 source of carbon monoxide. Biogenic VOCs are emitted by various types of vegetation and
16 emissions peak in summer which is also when O₃ production is fast and O₃ lifetimes are short.

17 The large abundance of biogenic VOCs leads to NO_x-limited O₃ production in most of
18 the world. That is, concentrations of biogenic VOCs are in excess with respect to concentrations
19 of NO_x; therefore, O₃ production is controlled by the availability of NO_x. The methodologies²⁷
20 typically used by the air quality community estimate contribution based on sensitivity of O₃
21 production. As a result, the sensitivity-based contribution estimate of biogenic VOC sources to
22 O₃ shows relatively small contributions considering the large amount of emissions.

23 Estimates of biogenic VOC contributions in the literature are generally small compared to
24 NO_x. For example, Lapina et al. (2014) found that North American Background (NAB)²⁸ for
25 W126²⁹ O₃ was relatively insensitive to VOC (10.8% of NAB sensitivity) compared to NO_x
26 (79.8% of NAB sensitivity). This well-known global-scale sensitivity to NO_x would not exist if
27 concentrations of biogenic VOCs were a broadly limiting factor. Even though background O₃ is
28 not particularly sensitive to small changes in the biogenic VOC, natural sources of VOCs are a
29 critical component of all background O₃ estimates.

²⁷ Source apportionment techniques and derivative-normalization techniques use sensitivity to attribute concentrations to sources. When a concentration is insensitive to VOC sources, the contribution estimate solely from that source of VOC will be zero.

²⁸ North American Background is analogous to USB; but NAB is generally characterized as the O₃ concentrations that would exist in the absence of North American anthropogenic emissions.

²⁹ W126 is a daytime weighted average concentration where higher concentrations are given greater weight based on a sigmoidal curve (see Chapter 4).

2.5.1.3 Wildland Fires

Fires emit a complex mixture of nitrogen oxides, nitrogen reservoir species (e.g., PANs), and VOCs that are all precursors to O₃. In the northern hemisphere, the fire season generally starts in spring and extends into fall with the specific timing varying widely by region. Fires also exhibit significant year to year variability, with emissions varying by an order of magnitude between high and low fire years in some places (van der Werf et al., 2017). While smoke from fires affects most of the contiguous U.S. at some point during the year, the fire season in the western U.S. occurs primarily late in the summer. Fires across western states and parts of Canada can contribute both to regional background and episodic surface O₃ enhancements (McClure and Jaffe, 2018).³⁰

Ozone production in fire plumes depends on a range of factors including the type of fuel combusted, plume age, and interactions with other air masses (e.g. urban plumes) (Jaffe and Wigder, 2012). While some studies have estimated wildfire O₃ contributions to seasonal mean O₃ of up to several ppb during high fire years in the Western U.S. (Jaffe et al., 2018), O₃ production from individual fires varies substantially (Akagi et al., 2013). Several studies have shown that locations near large fires can even experience suppressed O₃ formation, perhaps due to titration from fresh NO emissions and/or reduced solar radiation resulting from high aerosol concentrations (McClure and Jaffe, 2018; Buysse et al., 2019). Large variability in O₃ precursor emissions from fires combined with complex in-plume dynamics and chemistry make accurately quantifying O₃ production from fires extremely difficult at both regional and local scales.³¹

New data from recent and upcoming field and aircraft campaigns³² are expected to provide new insights that expand current understanding of contributions from fires to O₃ concentrations in the U.S., both in the context of regional background concentrations and production during individual fire episodes.

2.5.1.4 Lightning Nitrogen Oxides

Lightning is an indirect natural O₃ precursor source. Lightning produces NO_x from molecular nitrogen and oxygen, similar to traditional combustion processes. Because NO_x is the

³⁰ Fires may occur on wildlands naturally or accidentally, or fires may be planned (prescribed) for various purposes and set intentionally. In the USB modeling work described in section 2.5.2.1 below, emissions associated with prescribed fires are categorized as anthropogenic emissions and are not included in estimating USB.

³¹ Recently methods have been developed for identifying and estimating wild or prescribed fire contributions to O₃ concentrations with the potential to contribute to standards' exceedances. These are described in documents available at <https://www.epa.gov/air-quality-analysis/final-2016-exceptional-events-rule-supporting-guidance-documents-updated-faqs>.

³² Western Wildfire Experiment for Cloud Chemistry, Aerosol Absorption and Nitrogen (WE-CAN, https://www.eol.ucar.edu/field_projects/we-can) in 2018 and Fire Influence on Regional to Global Environments and Air Quality (FIREX-AQ, <https://www.esrl.noaa.gov/csd/projects/firex-aq/>) in 2019.

1 globally limiting precursor for O₃ production and lightning emits where there are few other
2 sources, O₃ production is quite sensitive to this source. Over the U.S., lightning NO_x (LNO_x)
3 emissions peak in summer with convective activity and are characterized as having high
4 interannual variability (Murray, 2016). Allen et al. (2012) showed that the majority of LNO_x is
5 emitted in the free troposphere (i.e., troposphere above the planetary boundary layer). Thus,
6 LNO_x is produced in a NO_x-limited environment where any O₃ formed as a result will be
7 efficiently transported and loss pathways are limited.

8 The total NO_x created by lightning is highly uncertain (Murray, 2016). Murray (2016)
9 discusses the uncertainty in NO yield per flash rate and the role of large spatial gradients in the
10 yield. The effect of such uncertainties is evident in the range of global lightning emissions
11 (std/mean=0.4). Murray (2016) also discusses the uncertainty in the vertical distribution of NO
12 production and post-production redistribution.

13 Jaffe et al. (2018) reviewed contributions from lightning to surface USB O₃ based on
14 modeling studies using various flash rate yields, which shows large single day contributions to
15 modeled MDA8 O₃ (up to 46 ppb, Murray, 2016) and smaller contributions to annual means (1-6
16 ppb) and seasonal means (6-10 ppb). Lapina et al. (2014) showed that, in their modeling, W126
17 had a 15% contribution from lightning NO_x over the U.S.³³ A 15% contribution is consistent
18 with the annual and seasonal mean contributions to MDA8 reported by Zhang et al. (2014) and
19 Murray (2016). Lapina et al. (2014) also noted that 40% of the lightning NO_x sensitivity comes
20 from lightning strikes outside the U.S. The findings from these studies highlight the primary
21 importance of lightning NO_x as a contributor to the well-mixed background concentrations
22 (Murray, 2016).

23 **2.5.1.5 Natural and Agricultural Soil NO_x**

24 Nitrogen oxides from soils are a naturally occurring source that is enhanced by
25 anthropogenic activity. Truly natural soil NO_x is created as a byproduct of nitrogen fixation in
26 natural environments. The fixation and byproduct release are affected by flora composition,
27 nitrogen availability, and environmental conditions (e.g., humidity). Human activity affects the
28 amount and location of soil NO_x emissions by changing land cover and by increasing the
29 availability of nitrogen for fixation through the application of fertilizer to crop lands or additions

³³ The numbers shown in this report are derived from reported values in Lapina et al. (2014) which showed sensitivity of W126 to anthropogenic NO_x sources was 58% (of that, 80% US; 9% CAN; 4% MEX) and natural NO_x sources was 25%. The remaining 17% was attributed natural isoprene (1.3%), VOCs/CO from fires (Fig 9: ~3%) and international VOC/CO (Fig 9: ~14%). So non-North American anthropogenic NO_x (58% * 7% non-NA = 4%) and natural NO_x (25%) create a total NAB NO_x sensitivity of 29% and total NAB sensitivity of 35% (29% / 79.8%). Of the total sensitivity (parentheses contain percent of NAB NO_x sensitivity, see Fig 12), lightning was 15% (52.9%), soil NO_x was 8% (28.2%), fire NO_x was 1% (4.3%) and international anthropogenic NO_x was 4% (14.5%).

1 of nitrogen via deposition of emissions from other sources. The effect of human land cover
2 alteration is readily apparent in soil NO_x emission measurements. Steinkamp and Lawrence
3 (2011), highlight that soils in pristine natural ecosystems emit more NO_x compared to similar
4 ecosystems that have been disturbed by human activity. At the same time, human managed crop
5 lands emit more than natural ecosystems (pristine or disturbed) environments because of the
6 applied fertilizer.

7 Soil NO_x clearly has both anthropogenic and natural sources, but these are rarely
8 separated in the literature. First, Hudman et al., 2012 estimate that the majority (~80%) of soil
9 NO_x emissions are currently attributed to land surfaces without considering active fertilization or
10 deposition of anthropogenic nitrogen. Second, the emissions and attribution are relatively
11 uncertain. Finally, anthropogenic soil NO_x is associated with agricultural ammonia application
12 that is not directly regulated in the United States. As a result, the attribution of soil NO_x as a
13 “background” source is imperfect. In this assessment, no distinction is made between natural and
14 fertilizer-enhanced soil NO_x and instead we include both within “natural sources.”

15 Hudman et al. (2012) estimated the global soil NO_x emissions at 10.7 TgN/y. As noted
16 above, soil NO_x emissions are linked to nitrogen availability in the soil, which is increased by
17 anthropogenic activities. Hudman et al. (2012) attributed 1.8 TgN/y to anthropogenic soil
18 fertilization and 0.5 TgN/y to atmospheric deposition. Like lightning, most soil NO_x emissions
19 occur outside of the U.S. Unlike lightning, soil NO_x has a smaller long-range transport
20 component because it is emitted at the surface. For example, Lapina et al. (2014) calculated that
21 W126 had an 8% sensitivity to soil NO_x (see footnote 26) and noted that a small fraction (only
22 7%) was from emissions outside the U.S. The more local sensitivity is likely due to the emission
23 height and spatial distribution of soil NO_x.

24 **2.5.1.6 Post-Industrial Methane**

25 Like VOCs, CH₄ is a hydrocarbon that can form O₃ in the presence of NO_x and sunlight.
26 While some atmospheric methane is emitted naturally from wetlands, wildfires, geogenic
27 sources, and insects, significant global methane enhancements following the industrial revolution
28 are clearly associated with increased emissions from anthropogenic fossil fuel combustion
29 (Pachauri et al., 2015). Other human activities such as livestock cultivation, landfills and land
30 use modification (e.g., rice paddies) also release methane. More recently, changing climate
31 conditions have led to increased emissions from natural sources (e.g., permafrost melting) in
32 some areas (Reay et al., 2018), although the exact magnitude of these effects on global methane
33 concentrations, and consequently O₃ in the U.S., over longer time scales remains uncertain.

34 Due to its long atmospheric lifetime (~10 years), methane is well-mixed at seasonal and
35 annual time scales. As a result, isolating contributions to atmospheric methane concentrations

1 from individual geographic areas or specific emission sectors is very difficult (Turner et al.,
2 2017). However, sensitivity simulations with chemical transport models can be used to assess the
3 overall influence of global methane concentrations on regional O₃ budgets. For example, Lin et
4 al. (2017) used the GFDL-AM3 chemistry-climate model to estimate that increasing global
5 methane concentrations contributed ~20% to background MDA8 O₃ trends during boreal spring
6 and summer at several western U.S. sites during the period 1988 to 2012. In general, post-
7 industrial anthropogenic methane is estimated to contribute ~5 ppb to surface O₃ in the U.S., an
8 estimate that primarily comes from modeling studies (Jaffe et al., 2018 and references therein).

9 A major limitation with existing model-based estimates of the influence of global
10 methane on current U.S. O₃ concentrations is our limited understanding of historical methane
11 emissions. The U.S. and the rest of the world's anthropogenic methane emissions have not been
12 tracked quantitatively in detail until relatively recently. As a result, the pre-industrial methane
13 concentration is relatively unconstrained. Further, post-industrial methane can be attributed to
14 direct emissions and emissions from natural sources (e.g., permafrost). Many modeling studies,
15 including this one, do not explicitly track methane sources and sinks, further complicating
16 attribution in an air quality context. Therefore, the post-industrial methane contribution is
17 difficult to quantitatively attribute. The post-industrial enhancement of methane is clearly related
18 to direct anthropogenic emissions and alteration of natural emissions by human activity, which
19 includes both foreign and domestic contribution.

20 **2.5.1.7 International Anthropogenic Emissions**

21 International anthropogenic emissions are the only anthropogenic contribution to USB.
22 For the purposes of discussion, NO_x and VOCs will be discussed separately from methane
23 (methane is covered in section 2.5.1.6). NO_x and VOC emission estimates from outside the U.S.
24 are derived from international collaborative efforts like the Hemispheric Transport of Air
25 Pollutants (HTAP) task force of the United Nations Economic Commission for Europe
26 (Janssens-Maenhout et al. 2015). HTAP harmonized national emission databases from individual
27 countries with global estimates that cover areas without their own estimates. Collecting and
28 harmonizing these emission datasets requires coordination and technical expertise, which
29 recently occurred twice (HTAP Phase I and HTAP Phase II) and a new HTAP emission
30 inventory is currently underway. Global estimates that incorporate national information are
31 available (e.g., Community Emissions Data System and Emissions Database for Global
32 Atmospheric Research), but do not always have as much participation from individual countries.
33 This is particularly important because individual countries are most aware of regulations and
34 controls that have been promulgated within their borders.

1 International anthropogenic sources of O₃ include emissions within the borders of other
2 countries (e.g., onroad sources, power plants, etc.) as well as sources in international waters and
3 air space. Sources within the borders of other countries can be easily attributed to those countries
4 using geographical bounds based on emission source location. Some studies (e.g., Lin et al.,
5 2014), however, have done more complex analyses to spatially attribute emissions globally based
6 on the consumption of produced goods. For the purposes of this document, international
7 emissions are attributed based on the emission source location. Using emission source location,
8 maritime shipping and aircraft sources require more artificial distinctions. Typically, aircraft
9 takeoff and landing are assigned completely to the country where it occurs. Aircraft cruising
10 emissions are attributed based on geographic boundaries. This assumes that both inbound and
11 outbound flights change source type (domestic/international) when they cross a border.

12 **2.5.2 Approach for Quantifying U.S. Background Ozone**

13 Updating USB estimates is motivated by interannual variability, trends in international
14 anthropogenic emissions, and continual improvements in simulating processes affecting USB.
15 USB sources are expected to vary from year to year because natural emissions vary in response
16 to meteorology (e.g., temperature) and long-range transport patterns alter the efficiency of
17 transport from long-range USB sources (Lin et al., 2015). In addition, the scientific
18 characterization of background emission sources continues to evolve. As a result, we provide an
19 updated assessment of USB for 2016 using the latest stable version of the Community Multiscale
20 Air Quality (CMAQ) model applied at hemispheric to regional scales.

21 This assessment uses a firmly source-oriented definition of USB based on modeling. The
22 source composition of a model estimate can be quantified using tagging techniques or by
23 sensitivity analysis. By contrast, the source composition of measured O₃ is difficult to isolate. In
24 most areas at most times, measured O₃ concentrations are the result of contributions from a
25 variety of anthropogenic and non-anthropogenic sources. Measurements from locations
26 sometimes suggested to be representative of USB often have contributions from U.S.
27 anthropogenic sources. As a result, some researchers have filtered measurements to focus on
28 times when US contributions are minimized (e.g., based on wind direction or other indicators).
29 The measurement filtering approach is based on conceptual or quantitative models of source
30 contributions as a function of wind direction or another environmental variable. After correction,
31 the degree of contamination is minimized but not precisely known. Recently, urban
32 measurements have been paired with simplistic statistical models to estimate background
33 (Parrish et al., 2017). However, Jaffe et al. (2018) concluded that statistical adjustment cannot be
34 directly interpreted as “background” – even though the estimate is useful for bounding simulated
35 background. Due to the complications of quantifying background based on ambient air

1 measurements, the sources that contribute to background are most clearly defined using an air
2 quality model. Using separate nomenclature (baseline: monitors; background: models) helps to
3 clearly delineate between these approaches that each have their strengths and weaknesses.

4 This section quantifies O₃ from sources using a sensitivity approach. The multiscale
5 system is applied to predict total O₃ and then applied multiple times to predict O₃ without U.S.
6 anthropogenic emission sources. The difference between total O₃ and O₃ without the U.S.
7 anthropogenic emissions is used to characterize the USB.

8 **2.5.2.1 Methodology: USB Attribution**

9 This assessment attributes O₃ to USB sources using one of several available techniques.
10 Jaffe et al. (2018) reviewed the methods for identifying USB contributions. The methodologies
11 reviewed range in complexity from simply turning off U.S. anthropogenic (or specific sources)
12 emissions, to normalizing derivatives from instrumented models, to complex tagging techniques
13 (e.g., CAMx OSAT, APCA, or Grewe, 2013).³⁴ This analysis follows the zero-out approach for
14 simplicity of interpretation and consistency with previous EPA analyses. In urban areas, this
15 approach will estimate higher natural and USB contributions than total O₃ when NO_x titration is
16 present. The estimate, therefore, is an estimate of what concentrations could be without U.S.
17 anthropogenic emissions and not the fraction of observed O₃ that is USB.

18 This analysis is designed to quantify O₃ specifically and separately from global natural,
19 international anthropogenic, and U.S. anthropogenic sources. The precursors that this analysis
20 focuses on are NO_x and VOC because they have a response on timescales relevant to the
21 NAAQS planning schedules (i.e., not methane). Table 2-1 lists simulations and the sources they
22 exclude at the various spatial scales modeled (i.e., hemispheric – 108 km resolution, regional –
23 36 km resolution and regional – 12 km resolution). For international shipping and aviation, the
24 U.S. domain is either included (ZROW) or excluded (ZUSA). These simulations form the basis
25 for estimating the contributions of USB and its components. Given the long atmospheric lifetime
26 and attributability to U.S. sources, methane is not separately identified nor is it perturbed in any
27 simulations. This has the effect of attributing methane to natural processes, which are a
28 background source.

29
³⁴ For a discussion of methods and the effect on estimates, see (Jaffe et al., 2018).

1 **Table 2-1. Simulation names and descriptions for hemispheric-scale and regional-scale**
 2 **simulations.**

Simulation	Description
<i>Performed at Hemispheric^A and Regional^B Scales</i>	
BASE	All emission sectors are included
ZUSA	All U.S. anthropogenic emissions are removed including prescribed fires. ^C
ZROW	All international anthropogenic emissions are removed including prescribed fires where possible.
ZANTH	All anthropogenic emissions are removed including prescribed fires.
<i>Performed at Hemispheric Scale only</i>	
ZCHN	All Chinese anthropogenic emissions are removed.
ZIND	All India anthropogenic emissions are removed.
ZSHIP	Zero all near-U.S. commercial marine vessel category 3 and all global shipping.
ZFIRE	Zero all fire emissions (agricultural, prescribed, and wild).
^A Hemispheric-scale simulations use 108 km grid cells defined on a polar stereographic projection. ^B Regional-scale simulations use a nested 36 km and 12km simulation on a Lambert conformal projection. ^C Emissions estimated to be associated with intentionally set fires ("prescribed fires") are grouped with anthropogenic fires.	

3
 4 Table 2-2 describes the calculations that are used to derive contributions. It is important
 5 to note that contributions are not strictly additive. Large NO_x sources can create non-linear
 6 conditions that decrease O₃ concentrations due to titration which is most relevant at night and in
 7 the winter. In some cases, removing a source only increases the efficiency of other sources. In
 8 that case, some anthropogenic contribution exists unless all anthropogenic sources are removed.
 9 This residual anthropogenic contribution occurs in the model for both International and U.S.
 10 sources. The results presented in this section focus on Base, USB, International, Natural
 11 contributions. Some components of International and Natural were separately analyzed.
 12 Canada/Mexico are separately quantified at both hemispheric and regional scales. The India,
 13 China, Fire, and shipping contributions are analyzed only at the hemispheric scale and are
 14 presented in Appendix 2B. The analyses in Appendix 2B support the interpretation in the
 15 discussion below.

16

1 **Table 2-2. Expressions used to calculate contributions from specific sources.**

Label	Name	Description	Expression
BASE	Total	Total Concentration	BASE
USB	USB	U.S. Background	ZUSA
USA	USA	U.S. Contribution	BASE – ZUSA
Intl	International	Rest of the World Contribution	BASE – ZROW
Natural	Natural	Natural Contribution	ZANTH
Res-Anth		Anthropogenic contribution that is not attributed directly to either the U.S. or International due to non-linear chemistry	BASE - ZANTH - Intl – USA
IND	India	India Contribution	BASE – ZIND
CHN	China	China Contribution	BASE – ZCHN
Ship	Ship	Ship Contribution	BASE – ZSHIP
FIRE	Fire	Global fire contributions	BASE – ZFIRE

2 **2.5.2.2 Methodology: Strengths, Limitations and Uncertainties**

3 The model was evaluated to assess the accuracy of predictions and infer possible biases
 4 in USB estimates. Evaluations included comparison to satellite retrievals, O₃ sondes³⁵,
 5 CASTNET monitors, and AQS monitors. Results were also qualitatively compared to the
 6 Tropospheric Ozone Assessment Report (TOAR) database, which has global O₃ observations
 7 that have been well characterized³⁶ but Phase I, which was completed and available at the time of
 8 analysis, only extends through 2014. The evaluation of the hemispheric simulation that provides
 9 boundary conditions to the 36 km model simulation relies heavily upon the satellites, O₃ sondes
 10 and CASTNET monitors. Since the satellite data can be used to provide concentration estimates
 11 in areas without surface monitors, these data are particularly useful for evaluating O₃ column
 12 totals in the hemispheric modeling. The sonde data provide a means to evaluate predictions aloft
 13 which are important for understanding model performance of long-range transport. The regional
 14 evaluation analysis focuses on data measured at CASTNET and AQS monitors.³⁷ Evaluation
 15 using the AQS monitors provides information on how the model performs at urban/suburban O₃,
 16 which may exhibit large space/time gradients in O₃ concentration. CASTNET data are included

³⁵ O₃ sondes are balloon-borne instruments that ascend through the atmosphere taking O₃ and meteorological measurements. For more information, see <https://www.esrl.noaa.gov/gmd/ozwv/ozsondes/>.

³⁶ The TOAR database includes O₃ globally where each monitor has been consistently characterized as urban or rural. The global observations have been processed for several metrics (MDA8, W126, etc.) and gridded to 2-degree by 2-degree global fields for easy comparison to large-scale models.

³⁷ In the discussion here in section 2.5, the data for CASTNET sites are referred to as “CASTNET data” and data for all other sites in AQS are referred to as “AQS data” (even though data for many, if not all, CASTNET monitors are stored in AQS).

1 in the evaluation of both the hemispheric and regional models since monitoring sites in this
2 network are intended to represent O₃ concentrations across broad areas of the U.S. Model
3 performance evaluation results are summarized in this chapter and provided in more detail in
4 Appendix 2-B.

5 The evaluation using sonde data shows that the hemispheric model predictions of O₃ are
6 generally within 20% of the corresponding measurements throughout much of the free
7 troposphere. Near the tropopause, there is a low bias in the model that is most pronounced in the
8 spring. The low bias at the tropopause likely suggests an underestimate of stratospheric
9 exchange. Mean bias drops to below 20% in the middle troposphere (600-300 hPa). The low-bias
10 in the free troposphere may stem from underestimation of spring time stratospheric contribution
11 in some regions.

12 The acceptability of model performance was judged for the 2016 CMAQ O₃ performance
13 results considering the range of performance found in recent regional O₃ model applications
14 (NRC, 2002, Phillips et al., 2008, Simon et al., 2012, U.S. EPA, 2009, U.S. EPA, 2018b). The
15 model performance results, as described in this document, demonstrate the predictions from the
16 2016 modeling platform closely replicate the corresponding observed concentrations in terms of
17 the magnitude, temporal fluctuations, and spatial differences for 8-hour daily maximum O₃. At
18 CASTNET sites, the model performance is similarly good, but has a distinct seasonal pattern
19 (see Appendix 2B.3). The normalized mean bias increases from a low-bias in boreal Winter
20 (West: -16%; East: -14%) to relatively neutral in boreal Fall (West: 0%; East: 7%). These results
21 are consistent with the free troposphere bias seen in the comparison of model predictions to
22 sonde data. Despite the conceptual consistency, the low-bias in winter at CASTNET sites is also
23 influenced by local sources. For example, the Uinta Basin monitors have extremely high winter
24 observations that are underpredicted by the model. These are most likely due to underestimation
25 of O₃ formed from precursors emitted by local sources as well as the need for finer resolution
26 meteorological inputs to capture cold pool meteorology conditions that characterize these
27 events.³⁸

28 Model predictions have historically shown poor performance for capturing the impacts
29 from O₃ of wildfires and stratospheric intrusions. Wildfire contributions have been overpredicted
30 by models (Baker et al., 2016, Baker et al., 2018). Model predictions of O₃ from stratospheric
31 intrusions have ranged from underestimated to overestimated (e.g., Emery et al., 2012). Models
32 are not expected to perform well in capturing the contributions from wildfires and stratospheric

³⁸ The DIN431 CASTNET monitor, among others, is in the Uinta basin where wintertime O₃ can be caused by snow-cover enhanced photolysis combined with light VOC emissions from the oil and gas production. (see Ahmadov et al., 2015).

1 intrusions without a focused effort on properly characterizing the physical properties of
2 individual events.

3 This analysis uses an emission inventory with known issues in the fire inventory. The
4 “2016fe” inventory had double counting of some grassland fires.³⁹ To minimize the effects of
5 double counting, a filter is applied to the data to remove large episodic natural influences
6 including fires. The filter removes days where natural contributions deviate from the mean for
7 that grid cell by whichever is higher: 20 ppb or twice the standard deviation for that grid cell.
8 Using this approach, 0.1% of grid cell days were removed -- 71% of grid cells have no days
9 removed and fewer than 5% have more than 1% removed. Of the days that were removed, fewer
10 than 21% had MDA8 concentrations above 70 ppb.

11 This study does not directly quantify USB uncertainty. Jaffe et al. (2018) highlight that
12 uncertainties in USB and USB component estimates come from multi-model comparisons.
13 Dolwick et al., 2015) showed that multi-model estimates converged when applying bias
14 correction, indicating that differences in USB estimates are correlated with model performance.
15 No bias correction has been applied here, so in a limited manner bias in ambient predictions can
16 help set expectations for bias in USB. Based on hemispheric model evaluation, the stratospheric
17 component in spring is likely underestimated leading to a USB low bias in spring. As a single
18 estimate, this study relies upon the literature based ± 10 ppb for seasonal means and higher for
19 individual days (Jaffe et al., 2018). Further, differences between models that share
20 parameterizations may not fully quantify underlying uncertainty and the year-to-year variability
21 complicates comparing model simulations done for different years.

22 **2.5.3 Estimates of USB and Contributions to USB in 2016**

23 Background O₃ is known to vary seasonally, spatially, and with elevation (as discussed in
24 section 2.5.1, above). Seasonal variations are related to temporal changes in both sources and
25 sinks. Spatial variations are related to differential transport patterns and the proximity to sources
26 of background O₃. Elevation is important in determining USB because it relates to the proximity
27 to the free troposphere. In addition, the seasonality and spatial relationships of USB and USA
28 contributions are not always aligned. As a result, USB can be highest on days with lower total
29 O₃. For these reasons, estimates of USB and USB components (i.e., Natural and International)
30 contributions developed from the current modeling are summarized spatially, over time, and as a
31 function of total O₃.

32 All analyses of USB and components focus on model predictions over land within the
33 U.S. The U.S. and adjoining areas are represented in the modeling using grid cells. Only grid

³⁹ More information related to this issue is available on the fire working group wiki page
<http://views.cira.colostate.edu/wiki/wiki/9175#July-12-2018>.

1 cells in the U.S. are included in this analysis.⁴⁰ Grid cells with water as the dominant land use
2 (e.g., lake or ocean) were simply excluded from analysis to acknowledge the potential bias of
3 total O₃ over water bodies (U.S. EPA, 2018). The USB estimates provided here are all in terms
4 of a metric, MDA8, closely related to the form of the current O₃ standards, and do not directly
5 apply to other metrics.

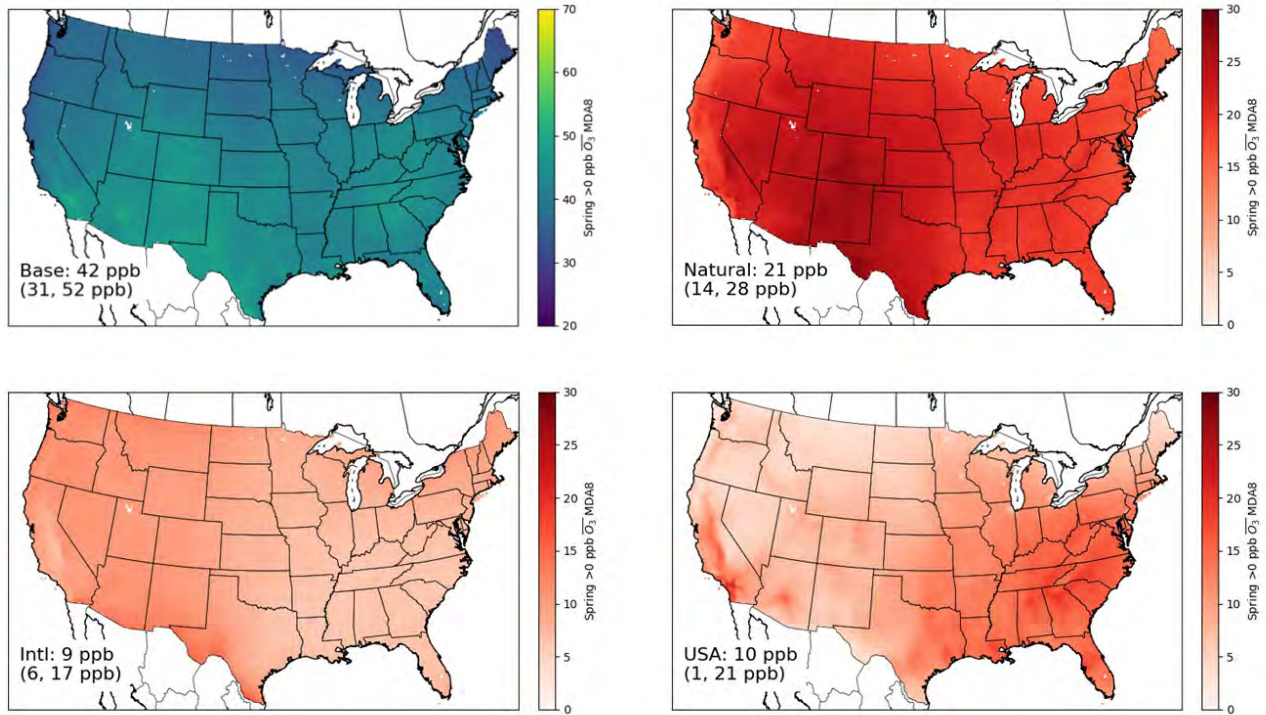
6 Section 2.5.3.1 characterizes the spatial variation of model-predicted MDA8 O₃
7 concentrations and contributions using maps of seasonal averages. Section 2.5.3.2 characterizes
8 the time variation of the predicted MDA8 O₃ and contributions using time series of spatial
9 averages. Section 2.5.3.3 characterizes the relationship between predicted USB components and
10 predicted total O₃. Section 2.5.3.4 summarizes USB predictions across regions and seasons.

11 **2.5.3.1 Spatial Characterization of O₃ Contributions**

12 Figure 2-19 and Figure 2-20 provide seasonally aggregated maps that show the spatial
13 distribution of total model-predicted MDA8 O₃ and contributions from natural, international, and
14 U.S. anthropogenic sources across the U.S.

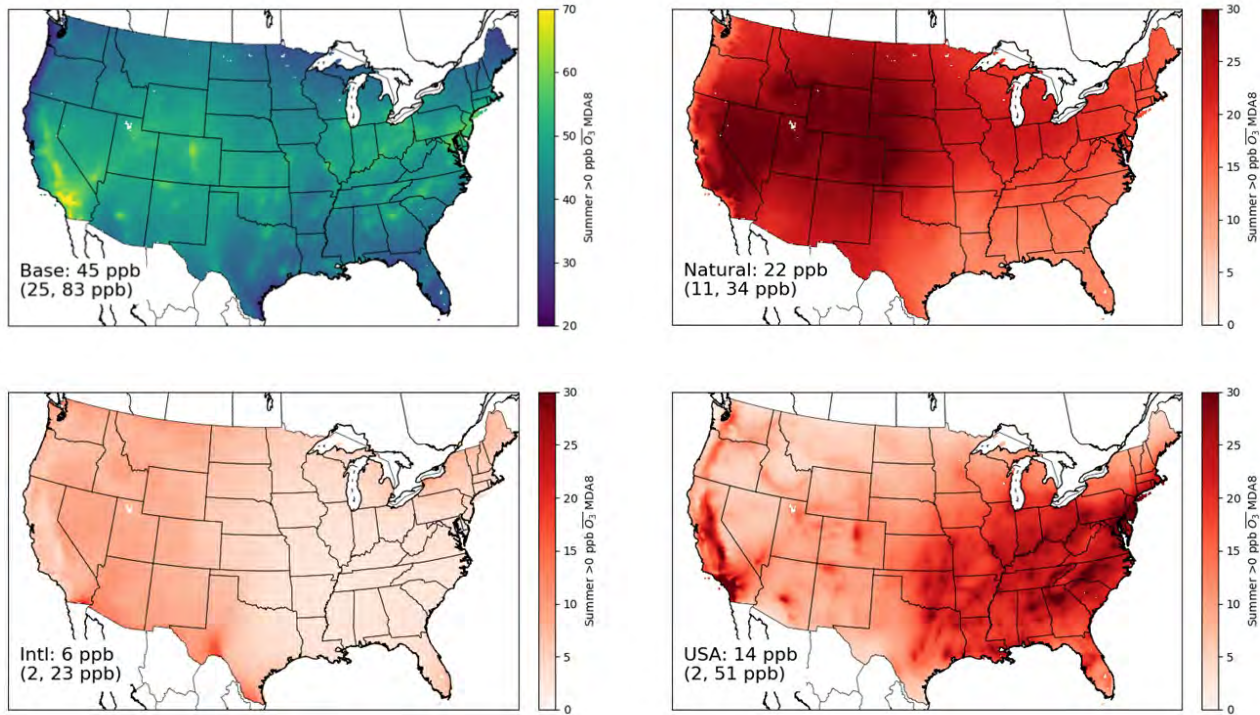
15 Figure 2-19 shows predicted MDA8 values for the 12 km domain averaged for spring
16 months (March, April, and May) for total O₃ and contributions from Natural, International, and
17 USA. Natural is a relatively large contributor to total O₃ in spring with a relatively small range of
18 values (ratio max:min = 2). International contributes less with a larger range (ratio max:min = 3).
19 There are spatial gradients primarily along parts of the Mexico border, and an overarching
20 general West-East gradient. The USA contribution, even in spring, has the largest variation (ratio
21 max:min > 20) with enhancements in some urban areas.

⁴⁰ Modeling grid cells are assigned to the U.S. based on the grid cell centers. For grid cells whose area covers the U.S. and an adjoining area, the grid cell is only assigned to the U.S. if the fraction of anthropogenic NO_x emissions contributed by the U.S. is greater than 80%. This is designed to remove grid cells from the analysis when the model cannot differentiate the border.



1 **Figure 2-19. Predicted MDA8 total O₃ concentration (top left), Natural (top right),**
 2 **International (bottom left), and USA (bottom right) contributions in spring**
 3 **(March, April, May). Each panel displays the simple spatial average and range**
 4 **(min, max) in ppb in the lower left-hand corner of the panel.**

5 Figure 2-20 shows the same type of information for the summer (June, July, August). The
 6 summer total concentrations are higher than spring due to increases in USA and Natural
 7 contributions. The international contribution spatial gradients have increased (reflecting shorter
 8 O₃ lifetimes), so that the maximum International contribution at the border is higher and the
 9 average contribution is lower compared to spring. Similarly, the West-East gradient of Natural,
 10 International, and USA contributions is enhanced in the summer. In addition, the USA
 11 contributions show distinct gradients in urban areas. Figure 2-20 highlights the increasingly near-
 12 border or high-elevation influence of international contribution during the summer when O₃
 13 concentrations are most likely to violate the NAAQS.
 14



1 **Figure 2-20. Predicted MDA8 total O₃ concentration (top left), Natural (top right),**
 2 **International (bottom left), and USA (bottom right) contributions in summer**
 3 **(June, July, Aug). Each contribution has the spatial average and range (min,**
 4 **max) in ppb in the lower left-hand corner of the panel.**

5 2.5.3.2 Seasonal and Geographic Variations in Ozone Contributions

6 Seasonal and geographic variations are an important part of background O₃. The
 7 geographic variation helps us to understand where USB contributes appreciably to O₃
 8 concentrations. The seasonal variation is particularly important as it determines whether high
 9 USB and MDA8 concentrations above 70 ppb are likely to occur at the same time. This section
 10 begins by characterizing the dependencies of predictions for different USB components on
 11 season and geography to define regions for further analysis. These dependencies are used to
 12 define regions for subsequent time series analysis.

13 **Seasonal dependence:** Comparing Figure 2-19 and Figure 2-20 highlights the seasonal
 14 differences in the predicted contributions from Natural, International, and USA sources. Between
 15 spring and summer, the International contribution decreases by 33%; the USA contribution
 16 increases by 40%; and the contribution from Natural sources shows a relatively small increase of
 17 5%. The differences in contributions between the spring and summer are due to a complex
 18 relationship between O₃ production, O₃ lifetime, and therefore transport efficiency. Cooler drier
 19 conditions increase the lifetime of O₃ in winter/spring compared to summer/fall (Liu et al.,
 20 1987). As a result, winter and spring have more efficient transport of O₃ compared to summer

1 and fall. Summer and fall, however, have warmer weather that promotes higher local O₃
2 production rates. Thus, summer and fall have locally fast O₃ production and relatively inefficient
3 transport, which combined increase the relative contribution of proximate sources.

4 **Border dependence:** In the summer, model-predicted gradients of International O₃ at the
5 borders are most obvious. As previously discussed, summer temperatures increase O₃ production
6 rates and decrease O₃ lifetimes. As a result, areas with locally high O₃ are evident near the border
7 in southern California and the Big Bend and lower Rio Grande areas of Texas. These local
8 enhancements generally occur within tens of kilometers from the border due to the short O₃
9 lifetime in summer as noted above.

10 **Topography dependence:** High elevation monitors are closer to the free troposphere; in
11 fact, at certain times of day and locations, the surface can sample free tropospheric air (Jaffe et
12 al., 2018). Complex topography can also enhance downward transport – for example, free
13 tropospheric air can “downwash” on the lee-side of high elevation mountains. Sites on the lee-
14 side can then be affected by this large-scale downwash. High elevation sites or sites influenced
15 by enhanced vertical transport may show higher contributions from more distant sources.

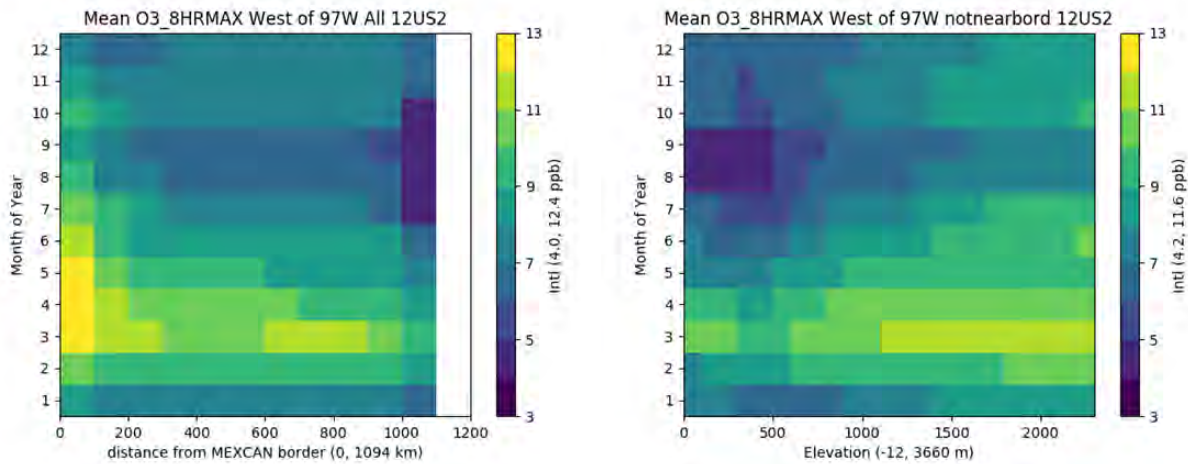
16 **Combined Seasonal and Geographic Dependence:** The simultaneous effects of
17 topography, proximity to international borders, and seasonal variations are highlighted by
18 Hovmoller diagrams (Figure 2-21). The Hovmoller diagram shows the average concentration as
19 a function of month (y-axis) and distance-to-border or elevation (x-axis). Due to the higher
20 magnitude of estimates of USB sources in the West than the East (Figure 2-19 and Figure 2-20),
21 the effects of distance and elevation are shown for the West. For the purposes of this analysis, we
22 use the 97W longitude line as a convenient way to separate the West from the East. The figures
23 show average estimated values and should not be used to estimate the international contribution
24 at any specific location. In addition, there are distinct gradients within the 100 m resolution of
25 the distance-to-border bins. For instance, the 0-100 km from the border grid cell values represent
26 a spatial average such that the locations directly adjacent to the border have Mexican
27 contributions higher than that average and the locations 100 km from the border have Mexican
28 contributions lower than that average.

29 Figure 2-21 shows that proximity to the border with Canada or Mexico is a good
30 indicator of the role of international contributions on USB predictions. In the spring, the average
31 international contribution can be as much as 12.4 ppb within 100 km of the border (62 miles). In
32 the early spring, large contributions persist further from the border because of the longer O₃
33 lifetimes. Near the borders the contributions also have much higher variability, both from day-to-
34 day and between locations on the border. The contribution from international sources drops
35 notably in the summer months when O₃ concentrations are highest. The day-to-day variability is
36 associated with the variations in wind direction, while the location variability is associated with

1 the proximity to an international population center. International contributions are highest in
2 near-border areas of the U.S. where there are emissions sources on the other side of the border.

3 To isolate the effect of elevation alone, Figure 2-21 shows the predicted international
4 contributions as a function of elevation after excluding border areas. In the spring, higher
5 international contributions are seen at all elevations. The international contribution at all
6 elevations decreases in summer compared to spring, but to lower contributions at lower elevation
7 and mostly slowly for the very high elevations (> 1500 m). This is consistent with findings from
8 Zhang et al. (2011) who used this elevation as a threshold.

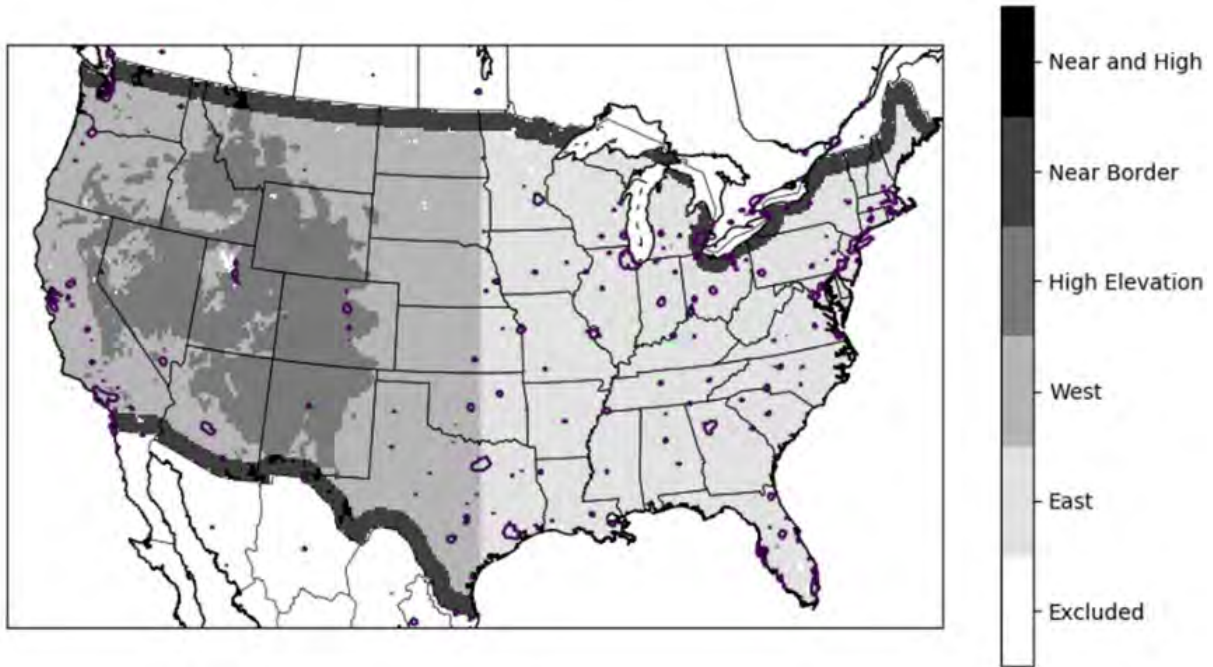
9



10 **Figure 2-21. Predicted contribution of International sources as a function of distance from**
11 **Mexico/Canada (left) and at “interior” locations (excluding border areas) by**
12 **elevation (right).**

13 **Timeseries Analysis:** The maps in Figure 2-19 and Figure 2-20 and the Hovmoller plots in
14 Figure 2-21 highlight the impact of season and location on predicted O₃ and contributions. To
15 further characterize the temporal variations in contributions, the contribution data are averaged
16 over West and East regions individually using 97W as a dividing line. The coarse “all-cells”
17 averaging of the data from individual grid cells ignores the major features of the relationship
18 between the sources and receptors on a sub-regional basis. For example, there are more grid cells
19 with high urban density and high anthropogenic NO_x in the East, so the USA contribution will
20 be higher in the East. Similarly, there are more high elevation areas in the West, so transported
21 O₃ from outside the U.S. will be higher there. Within the West, however, there are also urban
22 areas that have both high predicted contributions from international transport and anthropogenic
23 emissions in the U.S. An analysis using “all-cells” will highlight the general characteristics of the
24 region. To highlight the within region variability in the West, we also include analyses that focus
25 on urban cells at high-elevation, near borders, and elsewhere. Figure 2-22 shows regions (West

1 and East) with high-elevation and near border areas and urban areas highlighted by contours. As
 2 can be seen, all the high-elevation areas and Mexico/U.S. border are assigned to the West, the
 3 Canada/U.S. border extends across both East and West, and there are no high-elevation areas in
 4 the East.



5
 6 **Figure 2-22. Grid cell assignments to East (of 97W), West (of 97W), High Elevation (>**
 7 **1500m), Near Border (within 100 km), and Near and High (i.e., both High**
 8 **Elevation and Near Border). The purple outlines highlight grid cells with 20%**
 9 **or greater urban land use.** Near Border areas are in both the West and East, while
 10 High Elevation areas are exclusively in the West. Areas matching colors denoted
 11 East and West, are thus the Low Elevation/Interior areas.

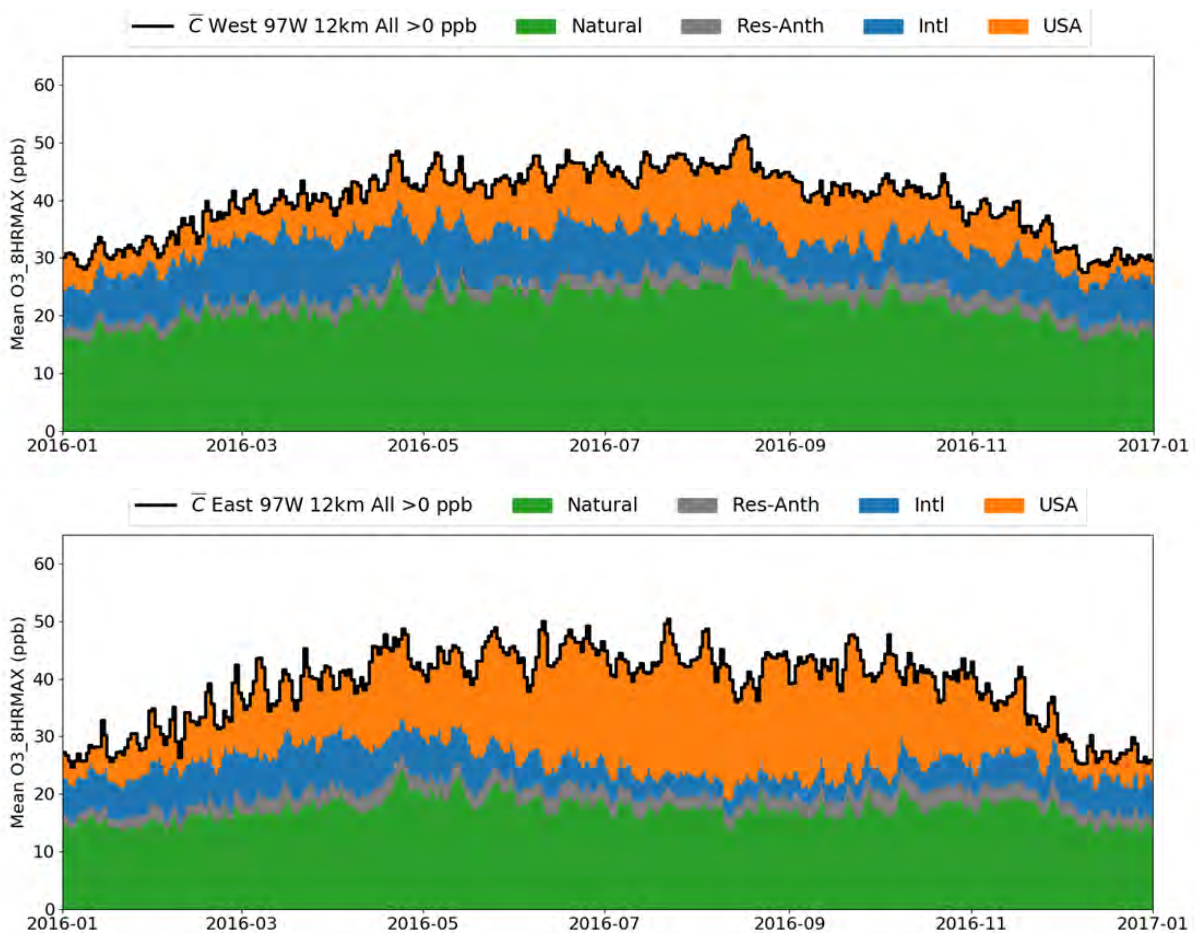
12 Figure 2-23 shows the time series of regional average (\bar{C}) MDA8 O₃ and O₃ contributions
 13 over the year for the West and East at “all-cells,” calculated using equation 2-1.

$$\bar{C} = \frac{\sum_x C_x}{N_x} \quad \text{Equation 2-1}$$

14
 15 where,
 16 N_x = number of grid cells (x) included
 17 C_x = concentration at each grid cell location (x)

18 The temporal pattern in the regional average clearly shows that the seasonality of MDA8
 19 predictions for each total O₃ component varies by region. The natural contribution has a single
 20 maximum in late summer in the West, whereas, in the East there is evidence of two peaks— the
 21 largest in late Spring and a second peak in early Fall. The somewhat lower MDA8 O₃ in summer

1 in the East requires further analysis but may be related to the lack of lightning emissions within
2 the regional domain. The seasonality international contribution predictions is more similar
3 between the two regions. The international contributions in both the West and East are greatest in
4 Spring, but the contribution in the West is larger both at its peak and its trough, compared to the
5 East. The total international contribution and the separately analyzed long-distance components
6 (e.g., China, India, international shipping) peak in spring when O₃ lifetimes favor long-range
7 transport (see Appendix 2B, Figure 2B-29). However, the Canada/Mexico component of
8 international contributions peaks in summer because of the relative proximity to the U.S.
9 receptors. The predicted USA contribution increases in the summer for both the West and the
10 East, but the USA contribution in the West is smaller than in the East. As mentioned previously,
11 this “all cells” average is disproportionately rural in the West. The following analysis looks
12 further at the different types of land in the West, including urban areas that are more
13 representative of population centers that behave differently than the “all cells” analysis.
14



15
16
17 **Figure 2-23. Annual time series of regional average predicted MDA8 total O₃ concentration**
18 **and contributions of each source (see legend) for the West (top), and the East**

1 **(bottom)**. Natural is global natural sources, Intl is international anthropogenic
2 sources, USA is U.S. anthropogenic sources, and Res-Anth is the residual
3 anthropogenic (see Table 2-2 for further descriptions).

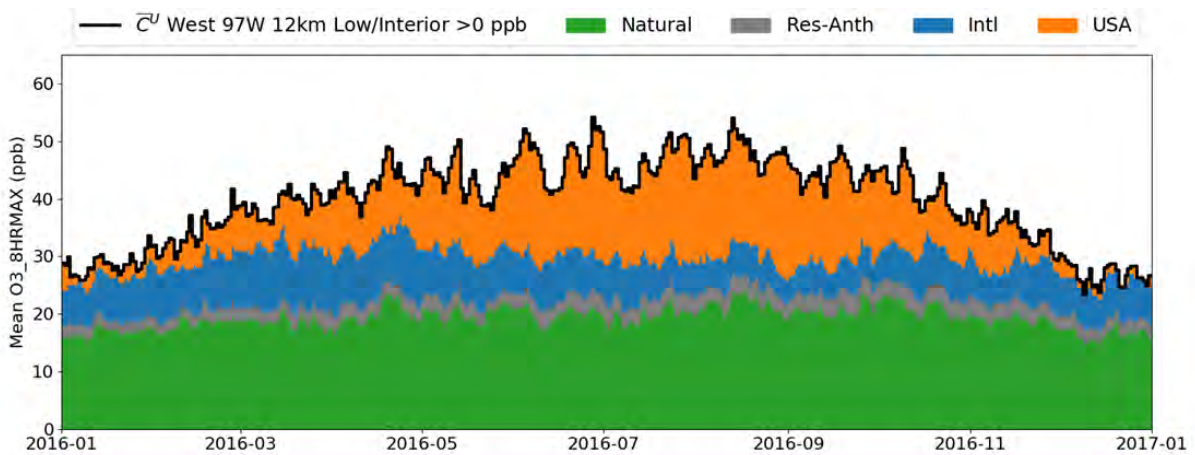
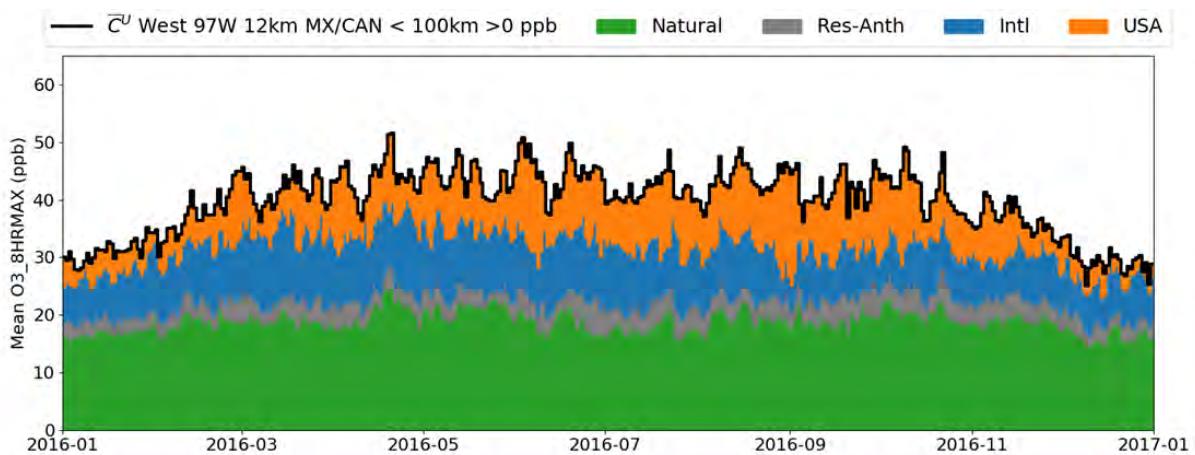
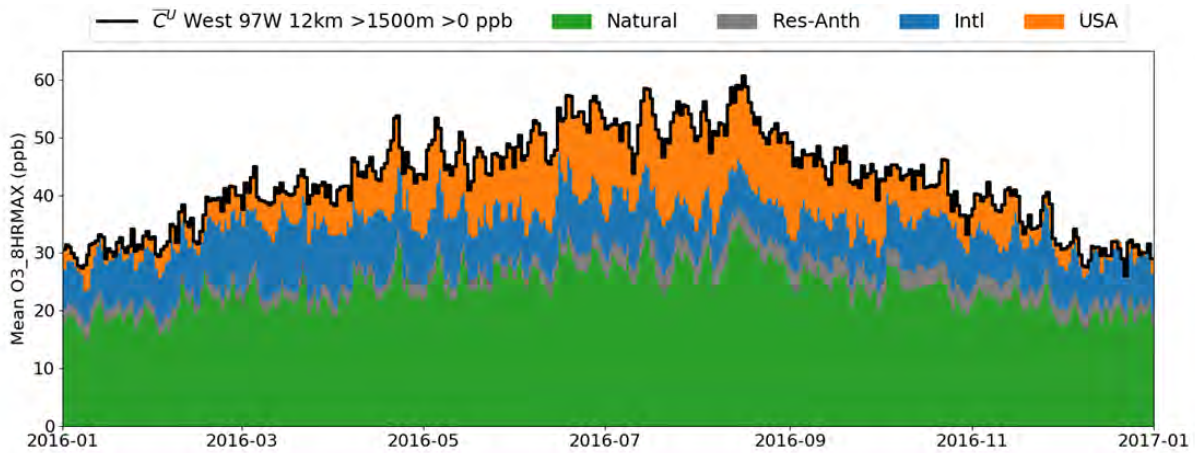
4 Figure 2-24 shows the predicted contributions to total O₃ in the West split into three
5 parts: the highest elevation areas, the near border areas, and Low/Interior areas with a weighted
6 average focusing on urban areas. Each of these subsets is illustrated in Figure 2-22, which shows
7 high elevation areas (exclusively in the West), near border areas (along the U.S./Mexico and
8 U.S./Canada borders), and dense urban areas. The Low/Interior areas are neither high elevation
9 nor near border. In each subset of cells, the purple outlines show the areas whose urban land use
10 is highest. The effect on O₃ contributions of the relative amount of urban land use can be
11 illustrated by computing an urban area weighted average contribution ($\overline{C^U}$), calculated using
12 equation 2-2.

$$\overline{C^U} = \frac{\sum_x A_x^U C_x}{\sum_x A_x^U} \qquad \text{Equation 2-2}$$

14 where,

15 A_x^U is the urban area in the grid cell x

16
17
18 The urban area weighted average gives a larger weight to data in those urban areas that have
19 dense emission sources (e.g., mobile). The urban area weighted average shows higher
20 contribution from USA while Natural and International are lower compared to Figure 2-23. The
21 differences between urban-weighted and non-weighted contributions are smaller in the East (not
22 shown) than in the West (compare Figure 2-23 top and Figure 2-24 bottom). Compared to the
23 West, the East has a larger fraction of land use that is urban (see Figure 2-22), which explains
24 this difference. Thus, the non-weighted regional average contributions in the East includes the
25 effects of urban areas much more so than the West. The seasonality of International is also
26 different between the highest elevation areas, near border areas, and urbanized areas. At
27 low/interior and at high-elevation sites, the simulated International contribution peaks earlier in
28 the year than at border sites. This earlier season peak is consistent with seasonality of O₃ lifetime
29 necessary for long-range transport and a smaller contribution of long-distance sources (India,
30 China, and global shipping, see Appendix 2B, Figure 2B-30). At near-border sites, the seasonal
31 cycle of predicted USB contributions from Canada/Mexico and from long-range transport
32 combine to create a maximum later in the spring or early summer that is dominated by
33 Canada/Mexico contributions (see Appendix 2B, Figure 2B-30, middle panel).



4 **Figure 2-24. Annual time series of regional urban area-weighted average predicted MDA8**
 5 **total O₃ concentration and contributions of each source (see legend) for the**
 6 **High-elevation West (top), near-border West (middle), and Low/Interior West**
 7 **(bottom). Natural is global natural sources, Intl is international anthropogenic**
 8 **sources, USA is U.S. anthropogenic sources, and Res-Anth is the residual**
 9 **anthropogenic (see Table 2-2 for further descriptions).**

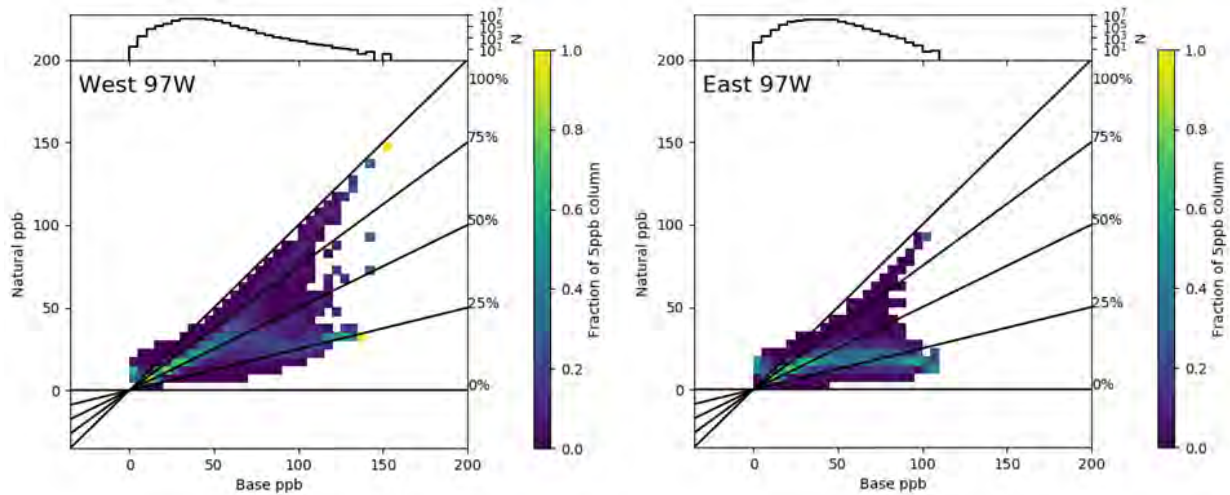
2.5.3.3 Ozone Source Contributions as a function of Total Ozone Concentration

Background contributions are also known to vary as a function of total O₃. To illustrate the relationship, specialized scatter density plots were created to show the contributions as a function of total O₃. Unlike the rest of this section, the scatter density plots do not apply the episodic natural filter described in section 2.5.2. Thus, episodic natural contributions including double counted fires are included in these presentations, and the effect of large events may be overestimated.⁴¹ In the scatter density plots (Figure 2-25 through Figure 2-27), each pixel represents a 5 ppb O₃ bin. In a traditional scatter density plot, the pixel color would represent the proportion of all points that fall within that pixel. However, in Figure 2-25 through Figure 2-27 the color represents the fraction of grid-cell-days within each 5 ppb total O₃ bin (i.e., the x-axis) that have a particular model-predicted contribution value (i.e., the y-axis). Brighter colors show where the most frequent model-predicted contribution (y-axis: Natural or International) lies within each 5-ppb bin of total O₃ value (x-axis). As a reference, percent contribution lines are overlaid on the plots to help contextualize the results.

Figure 2-25 shows the simulated daily Natural contribution as a function of total MDA8 concentration in the West and East for the whole year. In both regions the majority of total O₃ concentrations are under 40-50 ppb. At these low concentrations, the natural contribution correlates well with total O₃ and frequently contributes half of the total O₃. At low concentrations, natural contributions estimated by a zero-out approach can be larger than 100% of the total prediction. This is a result of NO_x-titration by local anthropogenic emissions, which reduces O₃ concentrations and is a well-known non-linearity of O₃ chemistry. Thus, removing the local NO_x source increases prediction concentrations. At higher concentrations, Figure 2-25 shows that predicted natural contributions in both regions have a bimodal distribution (or a fork in frequency of contributions). The lower mode represents a plateau of natural contributions with increasing total O₃, which represents enhancement by anthropogenic sources. The upper mode represents instances where natural contributions are correlated with total predicted O₃. In the West, the lower mode is less dominant than the East. This suggests, at least in the modeling, that there are more frequent model-predicted contributions from wildfires and/or stratospheric intrusions in the West. Wildfire emissions are known to be overestimated in this emission inventory and their contribution to O₃ concentrations are also often overestimated by CMAQ predictions. As a result, these predictions of very high natural contributions should be interpreted

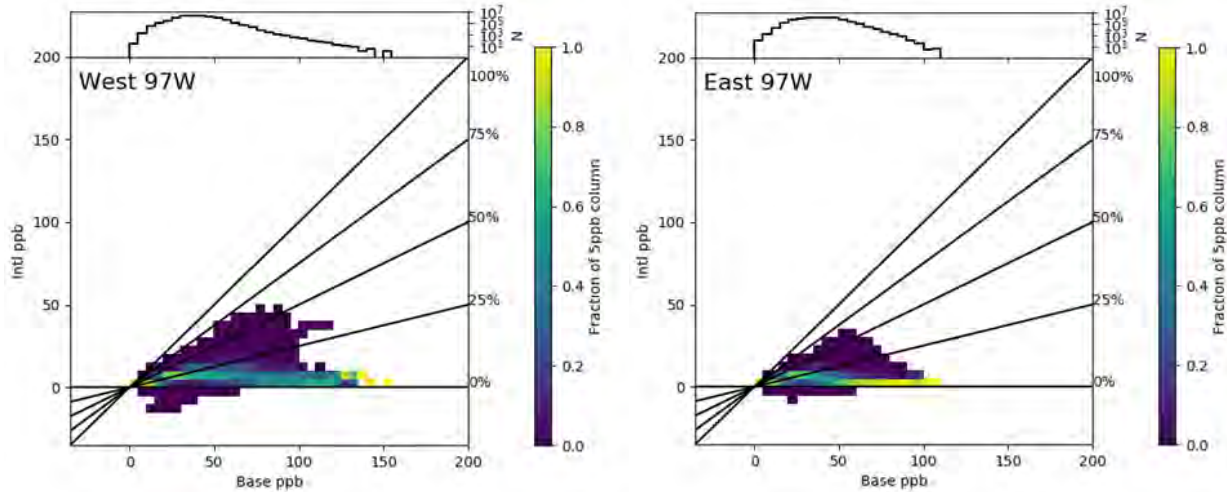
⁴¹ When episodic natural events contribute to elevated O₃ concentrations documented in air quality monitoring data to such an extent that they result in a regulatorily significant exceedance or violation of the NAAQS, they can be addressed via the Exceptional Events Rule (40 CFR 50.14).

1 qualitatively as simply indicating that such contributions can be appreciable, rather than as
2 providing accurate and precise quantitative predictions.
3



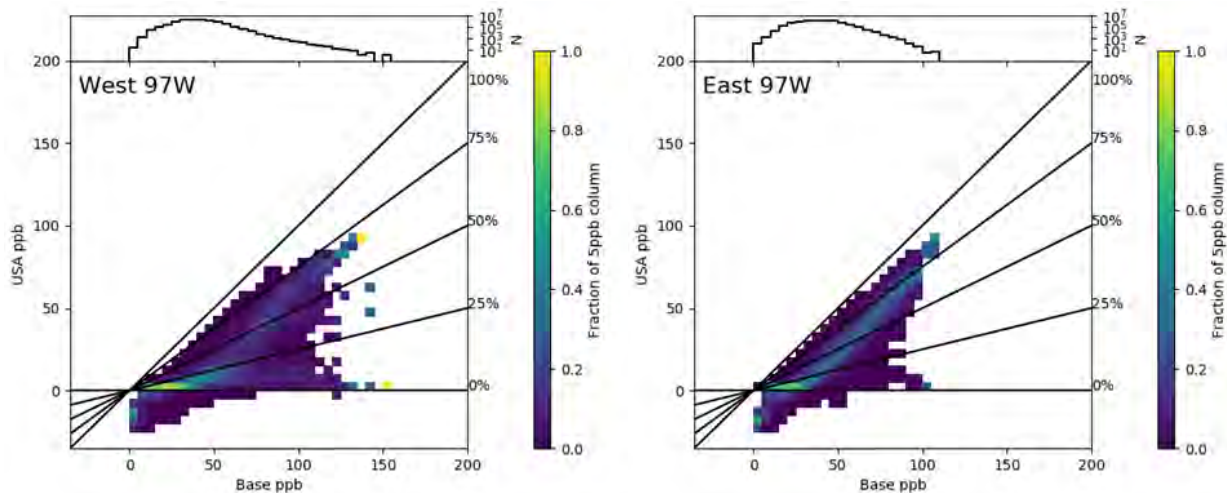
4 **Figure 2-25. Predicted contribution of Natural as a function of predicted total (Base)**
5 **MDA8 O₃ concentration in the West and East. Sloped lines show percent**
6 **contribution as a quick reference. The number of cells in each column is**
7 **identified using the probability density function above the plot, which is on a**
8 **log scale that highlights infrequent high concentrations.**

9 Figure 2-26 shows the predicted contribution in the West and East from international
10 anthropogenic sources. Unlike natural contributions, there is very little correlation between
11 international anthropogenic and total O₃. There are rare large model-predicted contributions,
12 which are more frequent in the West than in the East and rarely contribute more than 50% total
13 O₃ in either region. There are also negative contributions (up to -15 ppb), which arise from non-
14 linearities in chemistry. The largest negative contribution predictions are along the Mexico
15 border. These can either be NO_x-titration events or cases where chemistry associated with
16 international NO_x-sources remove precursors that would otherwise enhance O₃ from U.S.
17 sources. Negative international contributions tend to occur at relatively low total O₃
18 concentrations.



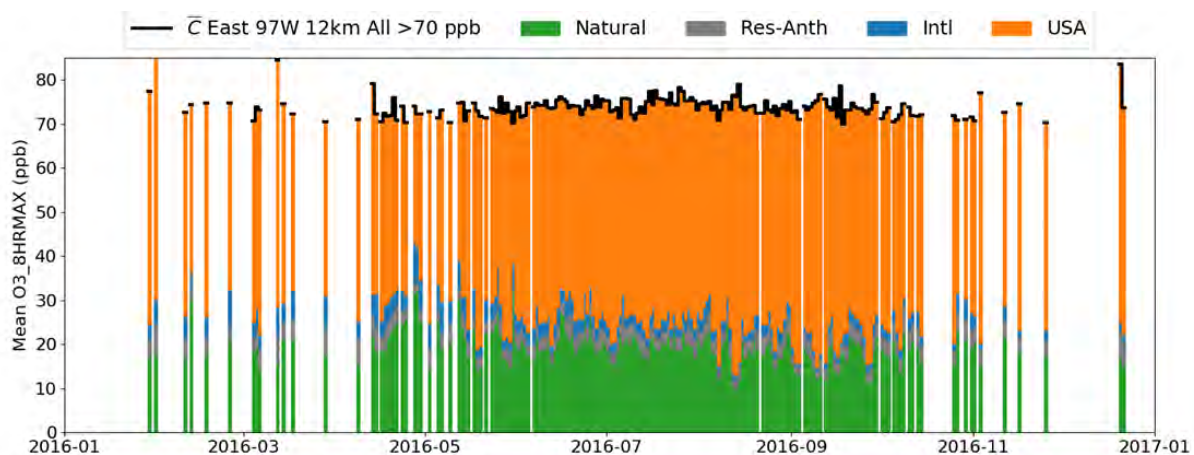
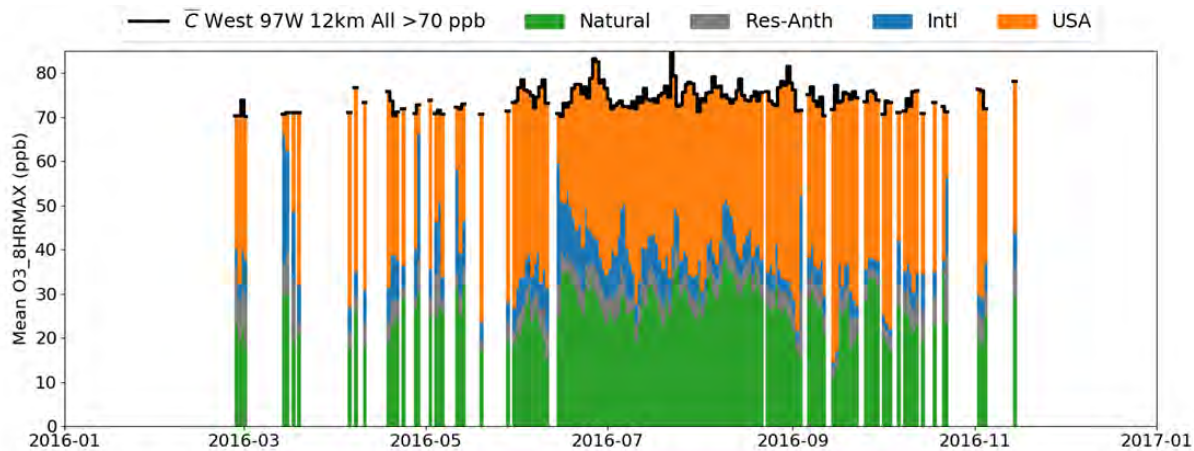
1 **Figure 2-26. Predicted contribution of International as a function of predicted total (Base)**
 2 **MDA8 O₃ concentration in the West and East. Sloped lines show percent**
 3 **contribution as a quick reference. The number of cells in each column is**
 4 **identified using the probability density function above the plot, which is on a**
 5 **log scale that highlights infrequent high concentrations.**

6 Figure 2-27 illustrates the relationship between predictions of U.S. anthropogenic sources
 7 and total O₃. Above 50 ppb, the predicted contribution from USA increases with total O₃ in both
 8 the West and the East. The relationship is stronger in the East, than the West, where near border
 9 contributions, fire contributions, and stratospheric exchange are smaller. Even so, the higher total
 10 O₃ in the West has a similar association of larger USA contributions at larger concentrations.
 11 This is consistent with previous findings (Henderson et al., 2012; U.S. EPA, 2014).
 12



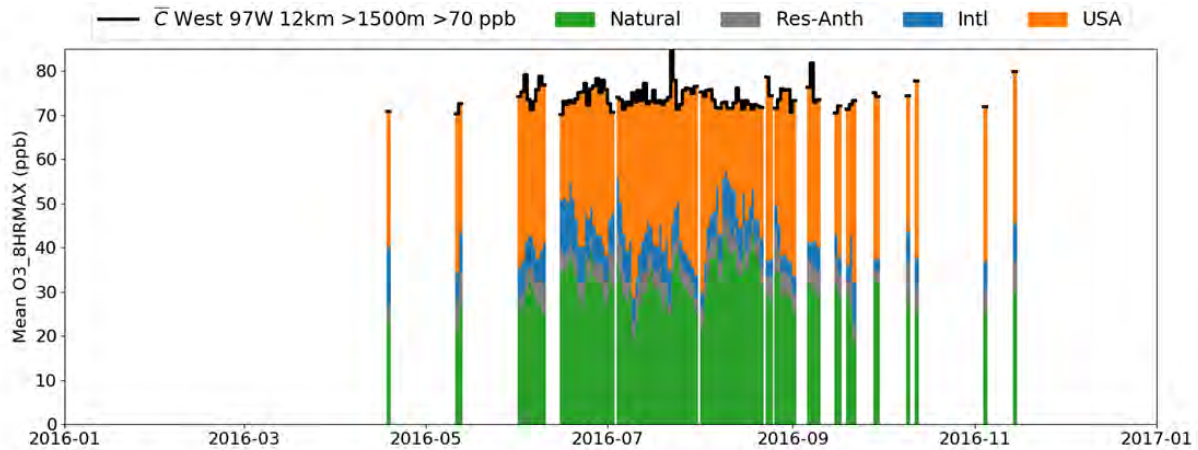
13 **Figure 2-27. Predicted contribution of USA as a function of predicted total (Base) MDA8**
 14 **O₃ concentration in the West and East. Sloped lines show percent contribution**
 15 **as a quick reference. The number of cells in each column is identified using the**
 16 **probability density function above the plot, which is on a log scale that**
 17 **highlights infrequent high concentrations.**

1 Another way of looking at the contributions is to restrict the time series to grid cells
2 where the concentration is above a threshold. Restricting to grid cells with high concentrations
3 implicitly weights the results toward urban areas where these high concentrations occur most
4 frequently. Figure 2-28 shows the seasonal and regional variation of USB (International
5 Anthropogenic and Natural) and USA (anthropogenic only) sources on high O₃ days (MDA8
6 >70 ppb). The largest magnitude differences between sources in the East and West come from
7 contributions predicted for Natural and USA sources. Recall that the West contains all the high-
8 elevation areas (>1500 m) and the full length of the U.S./Mexican border. Figure 2-29 includes
9 time series for high elevation, near Mexico border, and low-elevation interior areas separately.
10 Compared to the East, the low/interior sites in the West have 9 ppb larger contribution from
11 Natural and 2 ppb more from International. Compared to low/interior sites in the West, the high-
12 elevation sites have 7 ppb larger contributions from Natural and 4 ppb more from International.
13 For border areas, the International contribution is 13 ppb greater than in Low/Interior sites. As
14 previously noted, there are large gradients of predicted international contributions even within
15 the border areas, such that some locations within the 100 km of the border are predicted to
16 receive larger international contributions while others are predicted to receive substantially
17 smaller international contributions than noted above.
18

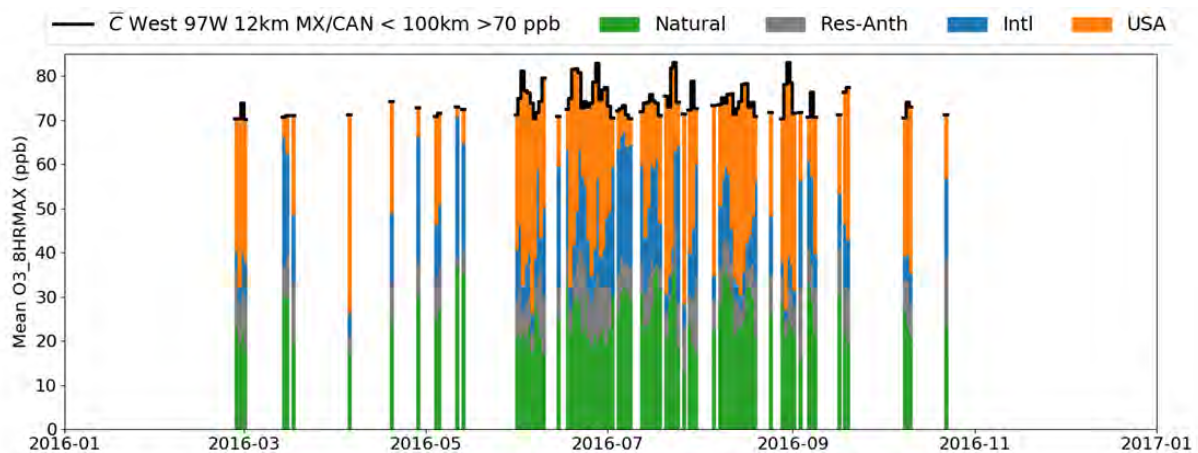


3 **Figure 2-28. Annual time series of regional average predicted MDA8 O₃ and contributions**
 4 **of each source to predicted MDA8 total O₃ (see legend) in the West (top) and**
 5 **East (bottom) including only those grid-cell days with MDA8 greater than 70**
 6 **ppb. Natural is global natural sources, Intl is international anthropogenic sources,**
 7 **USA is U.S. anthropogenic sources, and Res-Anth is the residual anthropogenic**
 8 **(see Table 2-2 for further descriptions).**

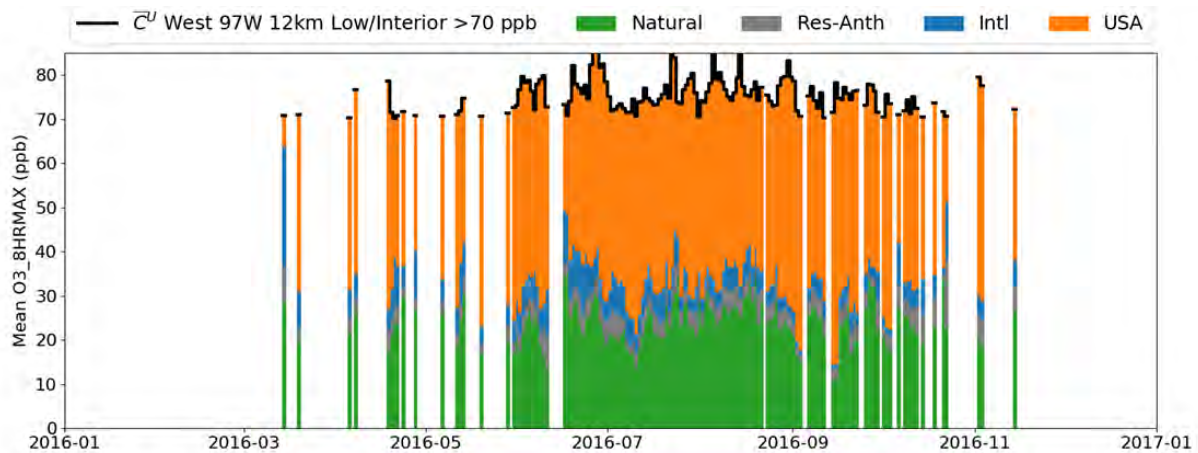
9



1



2



3

4 **Figure 2-29. Annual time series of regional average predicted MDA8 O₃ and contributions**
 5 **of each source to predicted MDA8 O₃ (see legend) in the high-elevation West**
 6 **(top), in the near-border West (middle), and in the Low/Interior West**
 7 **weighted toward urban areas (bottom) including only those grid-cell days with**
 8 **MDA8 O₃ greater than 70 ppb. Natural is global natural sources, Intl is**
 9 **international anthropogenic sources, USA is U.S. anthropogenic sources, and Res-**
 10 **Anth is the residual anthropogenic (see Table 2-2 for further descriptions).**

2.5.3.4 Predicted USB Seasonal Mean and USB on Peak O₃ Days

The analyses above describe the contributions from the components of USB to MDA8 O₃ over seasons and days. Jaffe et al. (2018) concluded that model predictions of seasonal means have more certainty than individual daily or episodic estimates of USB. However, from a policy perspective, it is also useful to understand the USB contributions for various regulatory-relevant metrics. In addition to reporting predicted USB using a seasonal average metric, we also examine predicted USB (1) on days with the highest predicted MDA8 total O₃ concentrations (top 10 days); (2) on days predicted to have the 4th highest MDA8 total O₃ concentrations in the year; and (3) on days when predicted MDA8 for total O₃ is above 60 ppb or above 70 ppb.

Figure 2-30 shows USB predicted by a single simulation with U.S. anthropogenic emissions zeroed-out. Similar to what was found for the seasonal average metric, the effect of topography and proximity to borders are readily evident for predicted MDA8 USB on the top 10 days and the 4th highest days. The differences in seasonal average contributions between the East and West are also evident with the top 10 days metric and 4th highest day metric. The speckled nature of the USB plot for the 4th highest day is due to the day or even season on which the 4th high is predicted to occur, which varies from grid cell to grid cell. The season in which the 4th highest day occurs influences the expected contribution from long-range international transport. The average USB contributions for the top 10 days exhibit a smoother spatial pattern because there is a tendency for high days to be grouped seasonally, even if the 4th highest is not. Because the USB contribution varies by season, the predicted USB contribution on the predicted 4th highest day is quite sensitive to model bias because bias may change the season on which the 4th highest predicted day occurs.

It is also important to highlight that areas with high predicted USB contributions do not always coincide with areas where MDA8 total O₃ concentrations are predicted to be above 70 ppb. On the 10 highest predicted MDA8 O₃ days, predicted USB is relatively constant over large areas (see Figure 2-30 middle left). Within these areas of relatively constant USB, Figure 2-30 shows that the locations having model-predicted MDA8 concentrations above 70 ppb are generally in or near urban areas (Figure 2-30 lower right).

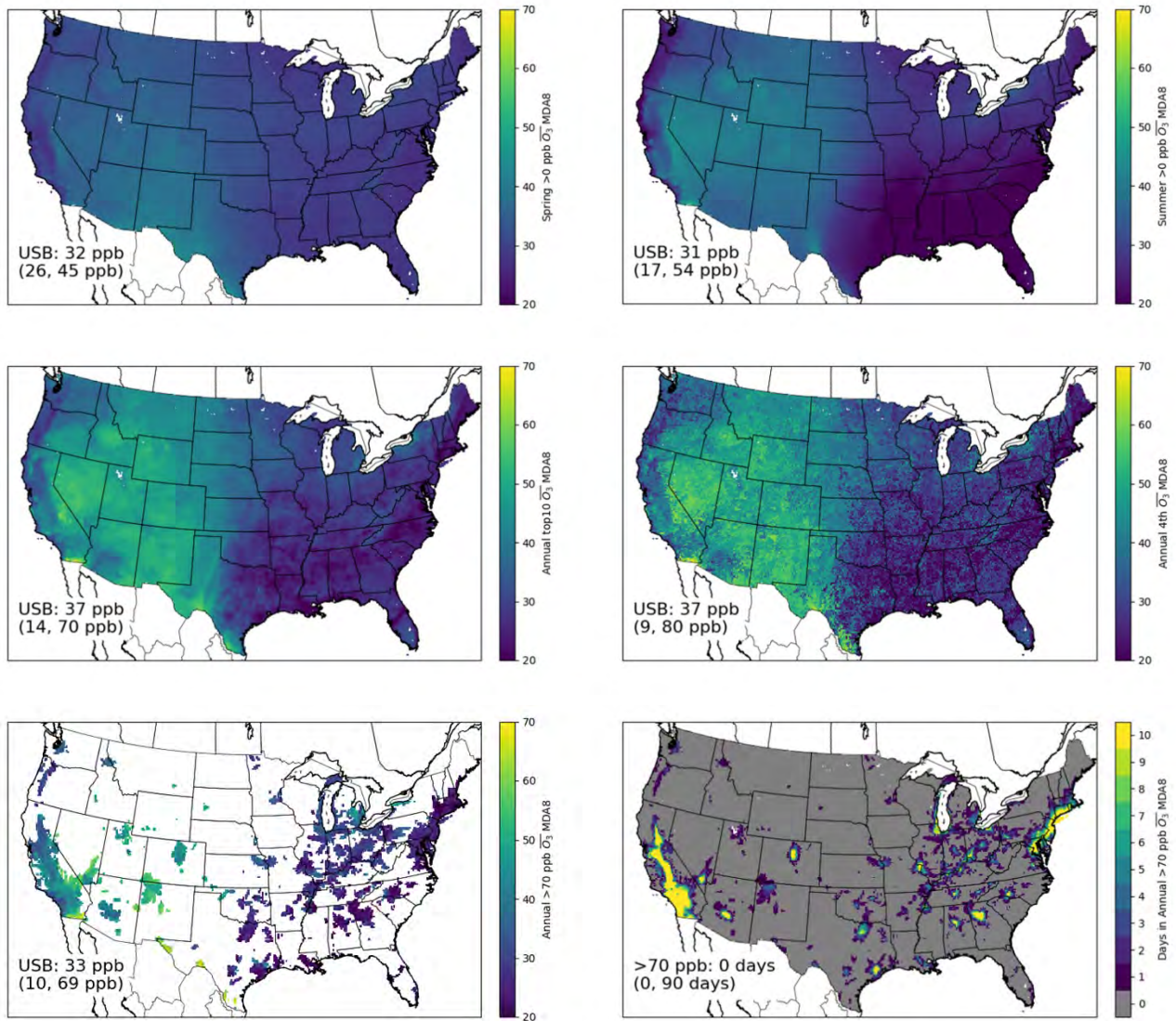
The USB contribution predicted in urban areas on the predicted top 10 days tends to be lower than in surrounding rural areas. This is due to the temporal anti-correlation of local contribution with natural and international contributions. In urban areas, MDA8 total O₃ concentrations above 70 ppb tend to occur in summer and fall when anthropogenic sources result in locally high increments of O₃. Also during these seasons, long-range transport is limited and USB from intercontinental transport is at its lowest. As a result, the predicted top 10 and 4th highest concentration days in urban areas tend to have lower predicted USB contributions than do such days in rural parts of the region even though rural areas have lower MDA8 O₃. As a

1 result, the areas with predicted top 10 days having MDA8 total O₃ above 70 ppb tend to have
2 lower percentage USB contributions than the surrounding areas.

3 Predicted USB contributions can be large on top 10 days near populated U.S./Mexico
4 border areas. In near-border areas with large anthropogenic emissions, international transport can
5 make a large contribution. For example, across the 4th highest days predicted for every grid cell
6 in this model simulation, the highest predicted MDA8 USB is 80 ppb (at a location immediately
7 adjacent to the border). Given the uncertainties associated with such single value predictions,
8 averaged predictions are important to consider. Compared to the maximum USB on the 4th high,
9 the maximum USB is 10 ppb lower for the average of top 10 days (Figure 2-30, middle left
10 panel) and 11 ppb lower the average of days with MDA8 above 70 ppb (Figure 2-30, lower left
11 panel). The very high USB values associated with international anthropogenic emissions are very
12 near the U.S./Mexico border and, to the extent that associated areas have been designated
13 nonattainment for the NAAQS, these areas may qualify under Clean Air Act section 179B, titled
14 “International border areas,” for specified regulatory relief upon submission of a satisfactory
15 demonstration.

16

1



2 **Figure 2-30. Map of predicted USB contributions by O₃ season for spring average (top left),**
3 **summer average (top right), top 10 predicted total O₃ days (center left), 4th**
4 **highest total O₃ simulated day (center right), and all days with total O₃ greater**
5 **than 70 ppb (bottom left), along with a map of the number of days with total**
6 **O₃ above 70 ppb (bottom right, where yellow pixels have 10+ days). Each**
7 **contribution has the spatial average and range (min, max) in the lower left-**
8 **hand corner of the panel.**

9

1 The maps in Figure 2-30 provide a detailed spatial representation of predicted USB but
2 may imply more precision than can be expected from a modeling system. For example, the
3 maximum USB on predicted fourth highest day reaches 80 ppb near the Mexico border. The
4 largest USB at nearby monitoring sites was 71 ppb.⁴² The observed 4th highs at those monitors
5 occurred in late February and early March, while the predicted 4th highs occurred in summer.
6 After selecting the 4th highs based on the observations and applying bias correction
7 proportionally to contributions, the new USB at these locations is 51 and 63 ppb. The USB
8 values for any given grid cell may be biased due to local features of topography, meteorology,
9 emissions bias, or model construct.

10 To complement the spatially resolved data and reduce bias associated with individual
11 daily model predictions, we also spatially aggregate the data by NOAA climate region. The
12 predicted USB values by climate region are provided in Table 2-3 to Table 2-6. Similar to the
13 figures, the tables separately quantify all grid cells (Table 2-3), high elevation (>1500 m) areas
14 (Table 2-4), near border areas (Table 2-5), and low-elevation (\leq 1500 m) interior areas (Table 2-
15 6). These tables show the spatial averages of USB within each climate region for the annual
16 average, seasonal averages, averages of days when MDA8 O₃ is greater than 60 or 70 ppb,
17 averages of each grid cell's top 10-days, and each cell's 4th highest day. Note that top 10-day
18 average and 4th high day for each grid cell may be from different times of the year compared to
19 the neighboring grid cells. As a result, grid cells with highest O₃ driven by transport in the Spring
20 are being mixed with grid cells with highest O₃ driven by local formation. Applying these
21 averages to interpret observations must, therefore, be done in the full context of time, space, and
22 concentration range.

⁴² Monitor 06-025-1003 measured 4th maximum value was 74 ppb on March 1, 2016. Monitor 06-073-1011
measured 4th maximum was 75 ppb on February 28, 2016. Predicted USB on predicted 4th high at both locations
was 71 ppb without bias correction in July and August.

1 **Table 2-3. Predicted USB for U.S. and U.S. regions based on averages for all U.S. grid**
 2 **cells.**

Regions ^A	Mean MDA8 for Seasons or Year					Mean MDA8 of Values in Subset			Annual 4 th highest MDA8
	DJF ^B	MAM ^C	JJA ^D	SON ^E	ANN ^F	>60ppb	>70ppb	Top10	
U.S.	26	32	31	29	30	38	33	37	37
West	28	35	36	32	33	47	43	44	44
East	24	29	24	25	26	28	27	28	28
NW	27	33	33	32	31	43	32	41	41
W	30	34	38	34	34	47	43	46	47
WNC	24	33	36	30	31	48	44	43	44
SW	31	38	39	35	36	51	48	49	49
S	27	33	26	27	28	34	29	33	33
ENC	21	30	28	26	26	31	34	32	33
C	24	30	25	26	26	28	28	28	28
SE	25	28	20	24	24	25	22	25	25
NE	25	29	27	27	27	29	26	28	27

^A U.S.=continental U.S, West= >97 degrees West longitude, East= <97 degrees West longitude, NW=Northwest, W=West, WNC=WestNorthCentral, SW=Southwest, S=South, ENC=EastNorthCentral, C=Central, SE=Southeast, and NE=Northeast.
^B Season defined as December, January and February.
^C Season defined as March, April and May.
^D Season defined as June, July and August.
^E Season defined as September, October and November.
^F Annual mean.

3

1 **Table 2-4. Predicted USB for high elevation locations (>1500 m).**

Regions ^A	Mean MDA8 for Seasons or Year					Mean MDA8 of values in subset			Annual 4th highest MDA8
	DJF ^B	MAM ^C	JJA ^D	SON ^E	ANN ^F	>60ppb	>70ppb	Top10	
U.S.	31	37	40	35	35	52	49	49	50
West	31	37	40	35	35	52	49	49	50
East	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
NW	29	35	38	33	34	52	42	47	48
W	32	36	42	36	36	53	47	51	52
WNC	28	35	39	34	34	52	48	48	49
SW	32	38	39	35	36	51	50	50	50
S	35	43	36	35	37	55	59	52	53
ENC	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
C	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
SE	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
NE	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A

^A U.S.=continental U.S, West= >97 degrees West longitude, East= <97 degrees West longitude, NW=Northwest, W=West, WNC=WestNorthCentral, SW=Southwest, S=South, ENC=EastNorthCentral, C=Central, SE=Southeast, and NE=Northeast.
^B Season defined as December, January and February.
^C Season defined as March, April and May.
^D Season defined as June, July and August.
^E Season defined as September, October and November.
^F Annual mean.

2 **Table 2-5. Predicted USB for locations within 100 km of Mexico or Canada Border.**

Regions ^A	Mean MDA8 for Seasons or Year					Mean MDA8 of values in subset			Annual 4th highest MDA8
	DJF ^B	MAM ^C	JJA ^D	SON ^E	ANN ^F	>60ppb	>70ppb	Top10	
U.S.	26	34	32	30	30	45	43	40	40
West	28	36	34	32	32	51	56	45	45
East	22	29	28	27	27	33	34	31	31
NW	27	32	30	31	30	46	N/A	38	38
W	30	35	41	36	36	46	51	51	51
WNC	21	33	34	29	29	49	N/A	42	42
SW	32	40	36	35	36	53	55	49	50
S	32	41	33	32	34	52	63	48	49
ENC	20	29	28	26	26	32	35	32	32
C	24	30	29	28	28	31	30	31	32
SE	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A	N/A
NE	24	29	28	27	27	34	41	30	30

^A U.S.=continental U.S, West= >97 degrees West longitude, East= <97 degrees West longitude, NW=Northwest, W=West, WNC=WestNorthCentral, SW=Southwest, S=South, ENC=EastNorthCentral, C=Central, SE=Southeast, and NE=Northeast.
^B Season defined as December, January and February.
^C Season defined as March, April and May.
^D Season defined as June, July and August.
^E Season defined as September, October and November.
^F Annual mean.

1 **Table 2-6. Predicted USB for low-elevation (≤ 1500 m) that are 100 km or farther from**
 2 **the border.**

Regions ^A	Mean MDA8 for Seasons or Year					Mean MDA8 of values in subset			Annual 4th highest MDA8
	DJF ^B	MAM ^C	JJA ^D	SON ^E	ANN ^F	>60ppb	>70ppb	Top10	
U.S.	25	31	28	28	28	33	30	34	34
West	27	34	34	31	31	43	39	41	41
East	24	29	24	25	26	27	27	28	28
NW	27	32	31	31	30	37	32	38	38
W	29	32	35	33	32	42	41	42	42
WNC	23	33	36	29	30	44	42	41	42
SW	29	37	38	33	34	49	43	47	47
S	26	32	26	27	28	32	26	32	32
ENC	21	30	28	26	26	31	33	32	33
C	24	30	25	26	26	28	28	28	28
SE	25	28	20	24	24	25	22	25	25
NE	25	29	26	27	27	28	25	27	26

^A U.S.=continental U.S, West= >97 degrees West longitude, East= <97 degrees West longitude, NW=Northwest, W=West, WNC=WestNorthCentral, SW=Southwest, S=South, ENC=EastNorthCentral, C=Central, SE=Southeast, and NE=Northeast.
^B Season defined as December, January and February.
^C Season defined as March, April and May.
^D Season defined as June, July and August.
^E Season defined as September, October and November.
^F Annual mean.

3 **2.5.4 Summary of USB**

4 Background O₃ results from a variety of sources, each of which has its own temporal
 5 pattern and spatial distribution. The location and timing of these sources impacts O₃ production,
 6 dispersion and loss and thus different background O₃ sources have unique seasonality and spatial
 7 patterns. The analysis presented here provides updated model-based estimates of magnitude,
 8 seasonality and spatial patterns of background O₃ contributions. The analysis separately
 9 characterizes the estimated magnitude and spatial/temporal patterns of MDA8 O₃ from three
 10 sources: natural, international anthropogenic, and USA anthropogenic.

11 The current analysis indicates that natural and USA O₃ contributions peak during the
 12 traditional O₃ season (May through September), while long-range intercontinental transport of
 13 international O₃ (i.e. contributions from China, India, etc.) peaks in the spring (February through
 14 May). The contributions from Canada/Mexico at near-border locations are associated with
 15 relatively short-range transport and the seasonality peaks during May through September, similar
 16 to USA anthropogenic O₃. The influence of Canada/Mexico, however, is indicated by the model
 17 predictions to have a stronger spatial gradient in summer, so Canada/Mexico contributions are
 18 most evident near the border. Of the three categories of contributions, the USA anthropogenic is
 19 best correlated with total O₃ at concentrations above 40-50 ppb in both the West and the East

1 suggesting that US anthropogenic emissions are usually the driving cause of high O₃ events in
2 the US. This is largely explained by temporal patterns of background O₃ influences in relation to
3 typical high O₃ events. There can be exceptions to this rule that are generally associated natural
4 contributions at high-elevation, during fires events, or at near-border sites.

5 This modeling analysis indicates the relationship between predicted international and
6 USA anthropogenic contributions depend upon the international sources and the location. Long-
7 range transport and USA anthropogenic contributions tend peak at different times of the year, so
8 the contribution of international is often at its minimum when local sources are the driving factor
9 for high total O₃ during the May through September O₃ season. Even in cases where O₃ formed
10 from international anthropogenic emissions does coincide seasonally with high O₃ periods, the
11 impact of those sources can have large spatial variation. For example, O₃ formed from
12 anthropogenic emissions in Canada and Mexico can peak in late spring or early summer when
13 total O₃ is high. During this time-period, there is a strong spatial variability not shown in the
14 regional mean. As a result, specific days at specific locations may experience larger or smaller
15 contributions from cross-border transport on an episodic basis that is not well characterized by
16 average seasonal contributions. Another example of spatial heterogeneity is exemplified by
17 wintertime O₃ events associated with emissions from local oil and gas production in the
18 Intermountain West. Even though these episodes can occur as early in the year as February,
19 international emissions do not contribute to them substantially. The conditions associated with
20 these events result in decoupling of the local air masses from the upper atmosphere, essentially
21 isolating air in the mountain valleys from the atmosphere above and reducing the influence of
22 long-range transport compared to other winter and early spring days. As a result, these unique
23 wintertime O₃ episodes have little relative influence from international emissions despite
24 occurring at a time of year when long-range transport from Asia is efficient. This highlights the
25 need to perform location specific analysis rather than relying on regional averages.

26 In addition to seasonal patterns, the ISA highlights interannual patterns in background O₃
27 as well as long-term trends (ISA, section IS.2.2.1). Natural emissions and international transport
28 are highly impacted by meteorological patterns which vary from year to year. One key ISA
29 finding is that decreasing East Asian NO_x emissions starting around 2010, which would suggest
30 decreasing contributions from East Asia in the future if those trends continue, and therefore
31 decreasing spring USB.

32 Assessments of background O₃ in the 2015 review reported regional variation in
33 background O₃ (2013 ISA; 2014 PA). Consistent with those assessments, modeling presented
34 here predicts that USB is higher in the West than in the East. In this analysis, we found that on
35 high O₃ days (greater than 70 ppb) the West-East differences are largely associated with
36 international contributions in near-border areas and natural contributions at high-elevation

1 locations. The Natural component of USB exhibits the largest magnitude difference between the
2 West and East. International contributions from intercontinental transport (e.g., Asia) are most
3 important at high elevations in the West, while international contributions from Canadian and
4 Mexican sources are most pronounced immediately adjacent to the borders.

5 The modeling performed for this assessment does not differentiate between natural
6 sources of ozone. For this analysis we did not attempt to separately quantify the contributions
7 from individual Natural sources (e.g., lightning, soil, fires, stratosphere) or to address exceptional
8 events beyond basic screening to remove very large fire plumes. Literature-based emissions
9 estimates and photochemical modeling studies can help to inform the likely contributors to
10 natural. In the northern hemisphere, the natural NO_x sources with the largest emissions estimates
11 are lightning (9.4 megatonN/yr), soils (5.5 megatonN/yr), and wildland fires (~2.2
12 megatonN/yr). Because NO_x is the limiting precursor at hemispheric scales, the emissions
13 estimates suggest that lightning and soils are most likely the largest contributors to Natural O₃,
14 except when impacted by specific fire episodes. As noted by Lapina et al. (2014), a large
15 contribution from lightning may be the result of lightning strikes outside the U.S. while the
16 contribution from soil NO_x tends to be largest from emissions within the U.S. The distant
17 lightning source is likely to have its effect as part of the well-mixed background. The local soil
18 NO_x emissions have a clear seasonal cycle and are known to have large local contributions. The
19 relative effect at any specific site would require further analysis, including identifying the portion
20 of the effect due to fertilizer.

21 The overall findings of this assessment are consistent with the 2014 PA, with the EPA's
22 Background Ozone whitepaper (U.S. EPA, 2015) and with the peer reviewed literature (e.g.,
23 Jaffe et al. 2018). The definition of USB is also consistent with the assessment in the 2014 PA
24 and includes global natural and international anthropogenic emission sources (NO_x and VOC).
25 Specific findings from the current analysis are summarized as:

- 26 • USB has important spatial variation that is related to geography, topography, and
27 international borders. The spatial variation is influenced by seasonal variation with long-
28 range international transport contributions peaking in the spring while US anthropogenic
29 contributions peak in summer.
- 30 • The West has higher predicted USB concentrations than the East, which includes higher
31 contributions from International and Natural sources. Within the West, high-elevation
32 and near-border areas stand out as having particularly high USB. The high-elevation
33 areas have more International and Natural contributions than low-interior areas in the
34 same region. The near-border areas in the West can have substantially more international
35 contribution than other parts of the West.
- 36 • The USA contributions that drive predicted MDA8 total O₃ concentrations above 70 ppb
37 are predicted to typically peak in summer. In this typical case, the predicted USB is
38 overwhelmingly from Natural sources. The most notable exception to the typical case is

1 reflected by predictions for an area near the Mexico border where the modeling indicates
2 that a combination of Natural and Canada/Mexico contributions can lead to predicted
3 MDA8 USB concentrations 60-80 ppb, on specific days, which is consistent with the O₃
4 PA prepared for the 2015 review (2014 PA, Section 2.4).⁴³

- 5 • Predicted international contributions, in most places, are lowest during the season with the
6 most frequent occurrence of MDA8 concentrations above 70 ppb. Except for the near-
7 border areas, the International contribution requires long-distance transport that is most
8 efficient in Spring.
- 9 • Days for which MDA8 total O₃ concentrations are predicted to be above 70 ppb tend to
10 have a substantially higher model-predicted USA (anthropogenic) contribution than other
11 days in both the West and the East.

⁴³ Uncertainties associated with such model predictions for individual days are recognized in section 2.5.3.4 above, along with observations of how they may differ from measurements at monitoring locations in the same area. It is also important to note that the modeling analyses presented here do not provide estimates of design values, which are derived from monitoring data (collected over three years) and used to assess exceedances of the O₃ standards. Additionally, as noted earlier, where such exceedances occur and are shown to be caused by USB, regulations for exceptional events may pertain.

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3 RECONSIDERATION OF THE PRIMARY STANDARD

This chapter presents and evaluates the policy implications of the key aspects of the scientific and technical information pertaining to this reconsideration of the 2020 decision on the O₃ primary standard. Specifically, the chapter presents key aspects of the available evidence of the health effects of O₃, as documented in the 2020 ISA, with support from the prior ISA and AQCDs, and associated public health implications.¹ It also presents key aspects of the quantitative risk and exposure analyses conducted for the 2020 review (and originally presented in the 2020 PA), with the details provided in Appendices 3C and 3D. Together this information provides the basis for our evaluation of the scientific information regarding health effects of O₃ 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 in this chapter is framed around key policy-relevant questions derived from the IRP (IRP, section 3.1.1), and also takes into account, as relevant, assessments of the evidence and quantitative exposure/risk analyses in prior reviews. In this way we identify key policy-relevant considerations and summary conclusions regarding the public health protection provided by the current standard for the Administrator’s consideration in this reconsideration of the 2020 decision on the primary O₃ standard.

Within this chapter, background information on the current standard is summarized in section 3.1. The general approach for considering the available information, 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 quantitative exposure and risk information, with associated uncertainties, is addressed in section 3.4. Section 3.5 summarizes the key evidence- and exposure/risk-based considerations identified in our evaluation, and also presents associated preliminary conclusions of this analysis. Key remaining uncertainties and areas for future research are identified in section 3.6.

¹ The ISA builds on evidence and conclusions from previous assessments, focusing on synthesizing and integrating the newly available evidence (ISA, section IS.1.1). Past assessments are generally cited when providing further, still relevant, details that informed the current assessment but are not repeated in the latest assessment.

3.1 BACKGROUND ON THE CURRENT STANDARD

The current primary O₃ standard of 0.070 ppm,² as the annual fourth-highest daily maximum 8-hour average concentration, averaged across three consecutive years, was set in 2015 and retained without revision in 2020 (80 FR 65292, October 26, 2015; 85 FR 87256, December 31, 2020). Establishment of this standard, and its retention in 2020, were based on the extensive body of evidence spanning several decades documenting the causal relationship between O₃ exposure and a broad range of respiratory effects, that had been augmented by evidence available since the 2008 review (80 FR 65292, October 26, 2015; 2013 ISA, p. 1–14). A key consideration driving the 2015 decision was the newly available evidence of adverse respiratory effects from controlled human exposure studies in healthy adults at an exposure concentration lower than had been previously studied (80 FR 65342–47 and 65362–66, October 26, 2015). While the study subjects in the vast majority of the controlled human exposure studies (and in all of these studies conducted at the lowest exposures) are healthy adults, the EPA’s establishment of the standard in 2015, and its retention in 2020, focused particularly on implications of these studies to insure protection of much less well studied at-risk populations,³ such as people with asthma, and particularly children with asthma (80 FR 65343, October 26, 2015; 85 FR 87305, December 31, 2020).

The 2020 review of the 2015 standard also considered differences in the health effects evidence since 2015 for effects other than respiratory effects. Specifically, the newly available evidence supported updated conclusions regarding metabolic effects, cardiovascular effects, and mortality (ISA, Table ES–1). For example, while the evidence available in the 2015 review was sufficient to conclude that the relationships for short-term O₃ exposure with cardiovascular health effects and mortality were likely to be causal, that conclusion was no longer supported by the more expansive evidence base which the 2020 ISA determines to be suggestive of, but not sufficient to infer, a causal relationship for these health effect categories (ISA, Appendix 4, section 4.1.17; Appendix 6, section 6.1.8). Further, newly available evidence since 2015 supports a new determination that the relationship between short-term O₃ exposure and metabolic effects

² Although ppm are the units in which the level of the standard is defined, the units, ppb, are more commonly used throughout this PA for greater consistency with their use in the more recent literature. The level of the current primary standard, 0.070 ppm, is equivalent to 70 ppb.

³ As used here and similarly throughout the document, the term population refers to persons having a quality or characteristic in common, such as, and including, a specific pre-existing illness or a specific age or lifestage. A lifestage refers to a distinguishable time frame in an individual’s life characterized by unique and relatively stable behavioral and/or physiological characteristics that are associated with development and growth. Identifying at-risk populations includes consideration of intrinsic (e.g., genetic or developmental aspects) or acquired (e.g., disease or smoking status) factors that increase the risk of health effects occurring with exposure to O₃ as well as extrinsic, nonbiological factors, such as those related to socioeconomic status, reduced access to health care, or exposure.

1 is likely to be causal (ISA, section IS.4.3.3). The basis for this conclusion is largely experimental
2 animal studies in which the exposure concentrations are well above those in the controlled
3 human exposure studies for respiratory effects as well as above those likely to occur in areas of
4 the U.S. that meet the current standard (85 FR 87270, December 31, 2020). Thus, while new
5 conclusions were reached in the 2020 review for these non-respiratory effect categories, they did
6 not lead to a change in focus for the standard, which continued to be protection of at-risk
7 populations from respiratory effects, as the effects causally related to O₃ at the lowest exposure
8 levels.

9 With regard to respiratory effects, the health effects evidence base available in the 2015
10 and 2020 reviews documents a broad range of effects associated with O₃ exposure (2013 ISA, p.
11 1-14; 2020 ISA, p. ES4-10). Such effects range from small, transient and/or reversible changes in
12 pulmonary function and pulmonary inflammation (documented in controlled human exposure
13 studies involving exposures ranging from 1 to 8 hours) to more serious health outcomes such as
14 emergency department visits and hospital admissions, which have been associated with ambient
15 air concentrations of O₃ in epidemiologic studies (2013 ISA, section 6.2; 2020 ISA, Appendix 3,
16 sections 3.1.5.1 and 3.1.5.2).⁴

17 Across the different study types, the controlled human exposure studies, which were
18 recognized to provide the most certain evidence indicating the occurrence of health effects in
19 humans following specific O₃ exposures, additionally document the roles of ventilation rate,⁵
20 exposure duration, and exposure concentration, in eliciting responses to O₃ exposure (80 FR
21 65343, October 26, 2015; 2014 PA, section 3.4). For example, the exposure concentrations
22 eliciting a given level of response in subjects at rest are higher than those eliciting a response in
23 subjects exposed while at elevated ventilation, such as while exercising (2013 ISA, section
24 6.2.1.1).⁶ Accordingly, of particular interest is the extent and magnitude of exposures during

⁴ In addition to extensive controlled human exposure and epidemiologic studies, the evidence base includes experimental animal studies that provide insight into potential modes of action for these effects, contributing to the coherence and robust nature of the evidence.

⁵ Ventilation rate (\dot{V}_E) is a specific technical term referring to breathing rate in terms of volume of air taken into the body per unit of time. A person engaged in different activities will exert themselves at different levels and experience different ventilation rates.

⁶ In the controlled human exposure studies, the magnitude or severity of the respiratory effects induced by O₃ is influenced by ventilation rate (in addition to exposure duration and exposure concentration), with physical activity increasing ventilation and potential for effects. In studies of generally healthy young adults exposed while at rest for 2 hours, 500 ppb is the lowest concentration eliciting a statistically significant O₃-induced reduction in group mean lung function measures, while a much lower concentration produces a statistically significant response in lung function when the ventilation rate of the group of study subjects is sufficiently increased with exercise (2013 ISA, section 6.2.1.1). For example, the lowest exposure concentration examined that elicited a statistically significant O₃-induced reduction in group mean lung function in an exposure of 2 hours or less was 120 ppb in a 1-hour exposure of trained cyclists who maintained a high exertion level throughout the exposure

1 periods of elevated ventilation, such as while exercising, under air quality conditions of interest.
2 Thus, key considerations in the establishment of the standard in 2015 and in its review in 2020
3 were the population exposure and risk assessments performed for air quality conditions
4 associated with just meeting the standard (and with alternative air quality scenarios). These
5 assessments, which included a focus on the at-risk populations of children and children with
6 asthma, analyzed the occurrence of exposures to O₃ concentrations of interest by individuals
7 breathing at elevated rates and characterized the associated risk.

8 The Administrator’s judgment in establishing the standard in 2015 was based primarily
9 on the extensive evidence of respiratory effects health effects evidence for O₃ with a focus on the
10 public health implications of the exposure and risk analyses conducted in that review. In the
11 review concluded in 2020, the Agency considered the health effects evidence base, including that
12 newly available since the 2015 decision, and the updated exposure/risk analyses. In 2020, the
13 Administrator reaffirmed judgments of the 2015 decision associated with establishment of the
14 different elements of the standard and made additional judgments reflecting the information
15 current to the review, concluding that the existing standard, set in 2015, continued to provide the
16 requisite public health protection with an adequate margin of safety (85 FR 87300-87306,
17 December 31, 2020). Key aspects of the health effects evidence and exposure and risk
18 information available in the 2020 review, as well as the associated judgments reflecting
19 consideration of associated limitations and uncertainties, are summarized below for each of the
20 four basic elements of the NAAQS (indicator, averaging time, form, and level), in turn.

21 In 1979, O₃ was established as the indicator for a standard meant to provide protection
22 against photochemical oxidants in ambient air (44 FR 8202, February 8, 1979). In setting the
23 current standard in 2015 and reviewing it in 2020, the Administrator considered the available
24 information presented in the ISA and PA, along with advice from the CASAC and public
25 comment. Both the 2013 and 2020 ISAs specifically noted that O₃ is the only photochemical
26 oxidant (other than nitrogen dioxide) that is routinely monitored and for which a comprehensive
27 database exists (2013 ISA, section 3.6; 80 FR 65347, October 26, 2015; 2020 ISA, p. IS-3; 85
28 FR 87301, December 31, 2020). The 2020 ISA further noted that “the primary literature
29 evaluating the health and ecological effects of photochemical oxidants includes ozone almost
30 exclusively as an indicator of photochemical oxidants” (2020 ISA, p. IS-3). In both reviews, the
31 CASAC indicated its support for O₃ as the appropriate indicator. Based on these considerations
32 and public comments, the Administrators in both reviews concluded that O₃ remains the most
33 appropriate indicator for a standard meant to provide protection against photochemical oxidants

period (2013 ISA, section 6.2.1.1; Gong et al., 1986) or after 2-hour exposure (heavy intermittent exercise) of young healthy adults (2013 ISA, section 6.2.1.1; McDonnell et al., 1983).

1 in ambient air, and they retained O₃ as the indicator for the primary standard (80 FR 65347,
2 October 26, 2015; 85 FR 87306; December 31, 2020).

3 The 8-hour averaging time for the primary O₃ standard was established in 1997 with the
4 decision to replace the then-existing 1-hour standard with an 8-hour standard (62 FR 38856, July
5 18, 1997). The decision in that review was based on newly available evidence from numerous
6 controlled human exposure studies in healthy adults of adverse respiratory effects resulting from
7 6- to 8-hour exposures, as well as quantitative analyses indicating the control provided by an 8-
8 hour averaging time of both 8-hour and 1-hour peak exposures and associated health risk (62 FR
9 38861, July 18, 1997; U.S. EPA, 1996). The 1997 decision was also consistent with advice from
10 the CASAC (62 FR 38861, July 18, 1997; 61 FR 65727, December 13, 1996). This averaging
11 time has been retained in each of the three NAAQS reviews since then (73 FR 16436, March 27,
12 2008; 80 FR 65292, October 26, 2015; 85 FR 87256, December 31, 2020). In the establishment
13 of the existing standard in 2015 and its review in 2020, the averaging time was retained in light
14 of both the strong evidence for O₃-associated respiratory effects following short-term exposures
15 and the available evidence related to effects following longer-term exposures (80 FR 65347-50,
16 October 26, 2015). The 2015 decision on a revised standard recognized that an 8-hour averaging
17 time is similar to the exposure periods evaluated in the more recent controlled human exposure
18 studies conducted at the lowest concentrations, and that other evidence, including that from
19 epidemiologic studies did not provide a strong basis of support for alternative averaging times
20 (80 FR 65348, October 26, 2015). Further, in 2015 the considerations on a revised standard also
21 included consideration of the extent to which the available evidence and exposure/risk
22 information suggested that a standard with an 8-hour averaging time can provide protection
23 against respiratory effects associated with longer-term exposures to ambient air O₃. Based on the
24 then-available evidence and information discussed in detail in the 2013 ISA, 2014 Health Risk
25 and Exposure Assessment (HREA), and 2014 PA, along with CASAC advice and public
26 comments, the Administrator concluded that a standard with an 8-hour averaging time (and
27 revised level) could effectively limit health effects attributable to both short- and long-term O₃
28 exposures and that it was appropriate to retain the 8-hour averaging time (80 FR 65350, October
29 26, 2015). The EPA reached similar conclusions in the 2020 review and retained the 8-hour
30 averaging time (85 FR 87306; December 31, 2020).

31 While giving foremost consideration to the adequacy of public health protection provided
32 by the combination of all elements of the standard, including the form, in 2015 the Administrator
33 placed considerable weight on the findings from prior reviews with regard to the use of the *n*-
34 high metric, as described below (80 FR 65350-65352, October 26, 2015). Based on these
35 findings and consideration of CASAC advice, the Administrator judged it appropriate to retain
36 the fourth-high form, more specifically the annual fourth-highest daily maximum 8-hour O₃

1 average concentration, averaged over 3 years (80 FR 65352, October 26, 2015). The EPA
2 reached similar conclusions in the 2020 review and retained the form of the annual fourth-
3 highest daily maximum 8-hour O₃ average concentration, averaged over 3 years (85 FR 87306;
4 December 31, 2020).

5 The concentration-based form (e.g., the *n*th-high metric) of the existing standard was
6 established in the 1997 review when it was recognized that such a form better reflects the
7 continuum of health effects associated with increasing O₃ concentrations than an expected
8 exceedance form,⁷ which had been the form of the standard prior to 1997. Unlike an expected
9 exceedance form, a concentration-based form gives proportionally more weight to years when 8-
10 hour O₃ concentrations are well above the level of the standard than years when 8-hour O₃
11 concentrations are just above the level of the standard. With regard to a specific concentration-
12 based form, the fourth-highest daily maximum was selected in 1997, recognizing that a less
13 restrictive form (e.g., fifth highest) would allow a larger percentage of sites to experience O₃
14 peaks above the level of the standard, and would allow more days on which the level of the
15 standard may be exceeded when the site attains the standard (62 FR 38868-38873, July 18,
16 1997), and there was not a basis identified for selection of a more restrictive form (62 FR 38856,
17 July 18, 1997). In subsequent reviews, the EPA also considered the potential value of a
18 percentile-based form, recognizing that such a statistic is useful for comparing datasets of
19 varying length because it samples approximately the same place in the distribution of air quality
20 values, whether the dataset is several months or several years long (73 FR 16474-75, March 27,
21 2008). However, the EPA concluded that, because of the differing lengths of the monitoring
22 season for O₃ across the U.S., a percentile-based statistic would not be effective in ensuring the
23 same degree of public health protection across the country.⁸ The importance of a form that
24 provides stability to ongoing control programs was also recognized.⁹ Advice from the CASAC in
25 the 2015 review supported this, stating that this concentration-based form that is averaged over
26 three years “provides health protection while allowing for atypical meteorological conditions that
27 can lead to abnormally high ambient ozone concentrations which, in turn, provides programmatic

⁷ The first O₃ standard, set in 1979 as an hourly standard, had an expected exceedance form, such that attainment was defined as when the expected number of days per calendar year, with maximum hourly average concentration greater than 0.12 ppm, was equal to or less than 1 (44 FR 8202, February 8, 1979).

⁸ Specifically, a percentile-based form would allow more days with higher air quality values (i.e., higher O₃ concentrations) in locations with longer O₃ seasons relative to locations with shorter O₃ seasons.

⁹ In the case of O₃, for example, it was noted that it was important to have a form that provides stability and insulation from the impacts of extreme meteorological events that are conducive to O₃ occurrence. Such events could have the effect of reducing public health protection, to the extent they result in frequent shifts in and out of attainment due to meteorological conditions because such frequent shifting could disrupt an area’s ongoing implementation plans and associated control programs (73 FR 16475, March 27, 2008).

1 stability” (Frey, 2014, p. 6; 80 FR 65352, October 26, 2015). Advice from the CASAC did not
2 raise objections with the indicator, averaging time and form of the existing standard (Cox, 2020).

3 In establishing the level of the standard in 2015 and in the decision to retain it in 2020,
4 the Administrator at each time carefully considered: (1) the assessment of the health effects
5 evidence and conclusions reached in the ISA; (2) the available quantitative exposure/risk
6 analyses, including associated limitations and uncertainties, described in detail in the HREA (in
7 the 2015 review) or appendices of the 2020 PA (in 2020); (3) considerations and staff
8 conclusions and associated rationales in the PA; (4) advice and comments from the CASAC;
9 and, (5) public comments (80 FR 65362, October 26, 2015; 85 FR 37300, December 31, 2020).
10 In weighing the health effects evidence and making judgments regarding the public health
11 significance of the quantitative estimates of exposures and risks allowed by the existing standard
12 and potential alternative standards considered, as well as judgments regarding margin of safety,
13 both of the decisions, in 2015 and 2020, considered the currently available information,
14 including EPA judgments in prior reviews, advice from the CASAC, statements of the American
15 Thoracic Society (ATS), an organization of respiratory disease specialists, and public comments.
16 In so doing, each decision recognized that the determination of what constitutes an adequate
17 margin of safety is expressly left to the judgment of the EPA Administrator. See *Lead Industries*
18 *Ass’n v. EPA*, 647 F.2d 1130, 1161-62 (D.C. Cir 1980); *Mississippi v. EPA*, 744 F.3d 1334, 1353
19 (D.C. Cir. 2013). In NAAQS reviews generally, evaluations of how particular primary standards
20 address the requirement to provide an adequate margin of safety include consideration of such
21 factors as the nature and severity of the health effects, the size of the sensitive population(s) at
22 risk, and the kind and degree of the uncertainties present. Consistent with past practice and long-
23 standing judicial precedent, in both the 2015 and 2020 decisions, the Administrator took into
24 account the need for an adequate margin of safety as an integral part of their decision-making.

25 The 2015 decision to set the level of the revised primary O₃ standard at 70 ppb placed the
26 greatest weight on the results of controlled human exposure studies and on quantitative analyses
27 based on information from these studies, particularly analyses comparing exposure estimates for
28 study area populations of children at elevated exertion to exposure benchmark concentrations
29 (exposures of concern), consistent with CASAC advice and interpretation of the scientific
30 evidence (80 FR 65362, October 26, 2015; Frey, 2014b).¹⁰ This weighting reflected the
31 recognition that controlled human exposure studies provide the most certain evidence indicating
32 the occurrence of health effects in humans following specific O₃ exposures, and, in particular,

¹⁰ The Administrator viewed the results of other quantitative analyses in this review – the lung function risk assessment, analyses of O₃ air quality in locations of epidemiologic studies, and epidemiologic-study-based quantitative health risk assessment – as being of less utility for selecting a particular standard level among a range of options (80 FR 65362, October 26, 2015).

1 that the effects reported in the controlled human exposure studies are due solely to O₃ exposures,
2 and are not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is
3 the case in epidemiologic studies) (80 FR 65362-65363, October 26, 2015).¹¹ With regard to this
4 evidence, the Administrator at that time recognized that: (1) the largest respiratory effects, and
5 the broadest range of effects, have been studied and reported following exposures to 80 ppb O₃
6 or higher (i.e., decreased lung function, increased airway inflammation, increased respiratory
7 symptoms, airway hyperresponsiveness, and decreased lung host defense¹²); (2) exposures to O₃
8 concentrations somewhat above 70 ppb¹³ have been shown to both decrease lung function and to
9 result in respiratory symptoms; and (3) exposures to O₃ concentrations as low as 60 ppb have
10 been shown to decrease lung function and to increase airway inflammation (80 FR 65363,
11 October 26, 2015). The Administrator also noted that 70 ppb was well below the O₃ exposure
12 concentration documented to result in the widest range of respiratory effects (i.e., 80 ppb), and
13 also below the lowest O₃ exposure concentration shown in 6.6-hour exposures with quasi-
14 continuous exercise to result in the combination of lung function decrements and respiratory
15 symptoms (80 FR 65363, October 26, 2015).

16 Consideration of the controlled human exposure study results and quantitative analyses
17 based on information from those studies focused primarily, both in 2015 and 2020, on the
18 exposure-based comparison-to-benchmarks analysis. This analysis characterizes the extent to
19 which individuals in at-risk populations could experience O₃ exposures, while engaging in their
20 daily activities, with the potential to elicit the effects reported in controlled human exposure
21 studies for concentrations at or above specific benchmark concentrations. The analysis conducted
22 for the 2020 review reflected a number of updates and improvements and provided estimates
23 with reduced uncertainty compared to those from the 2015 review (see section 3.4.1 below for
24 details). The results for analyses in both reviews are characterized through comparison of
25 exposure concentration estimates to three benchmark concentrations of O₃: 60, 70, and 80 ppb.
26 These are based on the three lowest concentrations targeted in studies of 6- to 6.6-hour exposures
27 of generally healthy adults engaging in quasi-continuous exercise (at a moderate level of
28 exertion), and that yielded different occurrences, of statistical significance, and severity of

¹¹ Other quantitative exposure/risk analyses (e.g., the lung function risk assessment, analyses of O₃ air quality in locations of epidemiologic studies, and epidemiologic-study-based quantitative health risk assessment) were viewed as providing information in support of the 2015 decision to revise the then-current standard level of 75 ppb, but of less utility for selecting a particular standard level among a range of options (80 FR 65362, October 26, 2015).

¹² Host defense refers to a decreased ability to repel pathogens and resist infection.

¹³ For the 70 ppb target exposure, the time weighted average concentration across the full 6.6-hour exposure was 73 ppb and the mean O₃ concentration during the exercise portion of the study protocol was 72ppb, based on O₃ measurements during the six 50-minute exercise periods (Schelegle et al., 2009).

1 respiratory effects (80 FR 65312, October 26, 2015; 85 FR 87277; December 31, 2020; 2020 PA,
2 section 3.3.3).¹⁴ A second exposure-based analysis provided population risk estimates of the
3 occurrence of days with O₃-attributable lung function reductions of varying magnitudes by using
4 the exposure-response (E-R) information in the form of E-R functions or other quantitative
5 descriptions of biological processes.¹⁵ These latter estimates were given less weight in the
6 Administrator's decisions in both the 2015 and 2020 reviews due to a recognition of relatively
7 greater uncertainty in interpretation of the results. Analyses in the 2020 PA quantitatively
8 illustrated this greater uncertainty associated with the lung function risk estimates related to their
9 greater reliance on estimation of responses at exposure levels below those that have been studied
10 (80 FR 65464, October 26, 2015; 85 FR 87277, December 31, 2020; 2020 PA, section 3.4.4).

11 In the 2015 decision to revise the standard level to 70 ppb (while retaining the existing
12 indicator, averaging time and form) and also the 2020 decision to retain that level (and all other
13 standard elements), without revision, the exposure analysis results for each of the three
14 benchmarks were considered in the context of the Administrator judgments concerning each
15 benchmark. Such judgments of the Administrator in setting the standard level of 70 ppb in 2015
16 are briefly summarized below. These are followed by a description of key aspects of the
17 considerations and judgments associated with the decision to retain this standard in 2020.

18 In the 2015 considerations of the degree of protection to be provided by a revised
19 standard, and the extent to which that standard would be expected to limit population exposures
20 to the broad range of O₃ exposures shown to result in health effects, the Administrator focused
21 particularly on the exposure analysis estimates of two or more exposures of concern. Placing the
22 most emphasis on a standard that limits repeated occurrences of exposures at or above the 70 and
23 80 ppb benchmarks, while at elevated ventilation, the Administrator noted that a standard of the
24 existing form and averaging time with a revised level of 70 ppb was estimated to eliminate the
25 occurrence of two or more days with exposures at or above 80 ppb and to virtually eliminate the
26 occurrence of two or more days with exposures at or above 70 ppb for all children and children
27 with asthma, even in the worst-case year and location evaluated (80 FR 65363-65364, October
28 26, 2015).¹⁶ The Administrator's consideration of exposure estimates at or above the 60 ppb
29 benchmark (focused most particularly on multiple occurrences), an estimated exposure to which

¹⁴ The studies given primary focus were those for which O₃ exposures occurred over the course of 6.6 hours during which the subjects engaged in six 50-minute exercise periods separated by 10-minute rest periods, with a 35-minute lunch period occurring after the third hour (e.g., Folinsbee et al., 1988 and Schelegle et al., 2009). Responses after O₃ exposure were compared to those after filtered air exposure.

¹⁵ The E-R information and quantitative models derived from it are based on controlled human exposure studies.

¹⁶ Under conditions just meeting an alternative standard with a level of 70 ppb across the 15 urban study areas, the estimate for two or more days with exposures at or above 70 ppb was 0.4% of children, in the worst year and worst area (80 FR 65313, Table 1, October 26, 2015).

1 the Administrator was less confident would result in adverse effects,¹⁷ was primarily in the
2 context of considering the extent to which the health protection provided by a revised standard
3 included a margin of safety against the occurrence of adverse O₃-induced effects (80 FR 65364,
4 October 26, 2015). In this context, the Administrator noted that a revised standard with a level of
5 70 ppb was estimated to protect the vast majority of children in urban study areas (i.e., about
6 96% to more than 99% of children in individual areas) from experiencing two or more days with
7 exposures at or above 60 ppb (while at moderate or greater exertion).¹⁸

8 Given the considerable protection provided against repeated exposures of concern for all
9 three benchmarks, including the 60 ppb benchmark, the Administrator in 2015 judged that a
10 standard with a level of 70 ppb would incorporate a margin of safety against the adverse O₃-
11 induced effects shown to occur in the controlled human exposure studies following exposures
12 (while at moderate or greater exertion) to a concentration somewhat higher than 70 ppb (80 FR
13 65364, October 26, 2015).¹⁹ The Administrator also judged the estimates of one or more
14 exposures (while at moderate or greater exertion) at or above 60 ppb to also provide support for
15 her somewhat broader conclusion that “a standard with a level of 70 ppb would incorporate an
16 adequate margin of safety against the occurrence of O₃ exposures that can result in effects that
17 are adverse to public health” (80 FR 65364, October 26, 2015).²⁰

¹⁷ The 2015 decision noted that “the Administrator is notably less confident in the adversity to public health of the respiratory effects that have been observed following exposures to O₃ concentrations as low as 60 ppb,” citing, among other considerations, “uncertainty in the extent to which short-term, transient population-level decrease in FEV₁ would increase the risk of other, more serious respiratory effects in that population” (80 FR 54363, October 26, 2015). Note: FEV₁ (a measure of lung function response) is the forced expiratory volume in one second.

¹⁸ The 2015 decision also noted the Administrator’s consideration of the extent to which she judged that adverse effects could occur following specific O₃ exposures related to each of the three benchmarks. The Administrator recognized the interindividual variability in responsiveness in her interpretation of the exposure analysis results noting “that not everyone who experiences an exposure of concern, including for the 70 ppb benchmark, is expected to experience an adverse response,” further judging “that the likelihood of adverse effects increases as the number of occurrences of O₃ exposures of concern increases.” And “[i]n making this judgment, she note[d] that the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O₃ may cause other more serious effects. Therefore, her decisions on the primary standard emphasize[d] the public health importance of limiting the occurrence of repeated exposures to O₃ concentrations at or above those shown to cause adverse effects in controlled human exposure studies” (80 FR 65331, October 26, 2015).

¹⁹ In so judging, she noted that the CASAC had recognized the choice of a standard level within the range it recommended based on the scientific evidence (which was inclusive of 70 ppb) to be a policy judgment (80 FR 65355, October 26, 2015; Frey, 2014b).

²⁰ While the Administrator was less concerned about single exposures, especially for the 60 ppb benchmark, she judged the HREA of one-or-more estimates informative to margin of safety considerations. In this regard, she noted that “a standard with a level of 70 ppb is estimated to (1) virtually eliminate all occurrences of exposures of concern at or above 80 ppb; (2) protect the vast majority of children in urban study areas from experiencing any exposures of concern at or above 70 ppb (i.e., ≥ about 99%, based on mean estimates; Table 1); and (3) to achieve substantial reductions, compared to the [then-]current standard, in the occurrence of one or more exposures of concern at or above 60 ppb (i.e., about a 50% reduction; Table 1)” (80 FR 65364, October 26, 2015).

1 The 2020 review of the 2015 standard also focused on the exposure-based analyses in the
2 context of results from the controlled human exposure studies of exposures from 60 to 80 ppb,
3 recognizing this information on exposure concentrations found to elicit respiratory effects in
4 exercising study subjects to be unchanged from what was available in the 2015 review (2020 PA,
5 section 3.3.1; 85 FR 87302, December 31, 2020).²¹ In considering the significance of responses
6 documented in these studies and in the full evidence base for the purposes of judging
7 implications of the available information on public health protection provided by the current
8 standard, several aspects, limitations and uncertainties of the evidence base were noted. For
9 example, as also recognized in 2015, the responses reported from exposures ranging from 60 to
10 80 ppb are transient and reversible in the study subjects who are largely healthy, adult subjects.
11 Such study data are lacking at these exposure levels for children and people with asthma, and the
12 evidence indicates that such responses, if repeated or sustained, particularly in people with
13 asthma, pose risks of effects of greater concern, including asthma exacerbation, as cautioned by
14 the CASAC (85 FR 87302, December 31, 2020).²²

15 As in 2015, the Administrator in 2020 also considered statements from the ATS, as well
16 as judgments made by the EPA in considering similar effects in previous NAAQS reviews (85
17 FR 87270-72, 87302-87305, December 31, 2020; 80 FR 65343, October 26, 2015). The ATS
18 statements included one newly available in the 2020 review (Thurston et al., 2017), which is
19 generally consistent with the prior statement (that was considered in the 2015 review) including
20 the attention that the prior statement gives to at-risk or vulnerable population groups, while also
21 broadening the discussion of effects, responses, and biomarkers to reflect the expansion of
22 scientific research in these areas (ATS, 2000; Thurston et al., 2017). The Administrator
23 recognized the role of such statements, as described by the ATS, as proposing principles or
24 considerations for weighing the evidence rather than offering “strict rules or numerical criteria”
25 (ATS, 2000, Thurston et al., 2017). In keeping with this intent of these statements (to avoid

²¹ With regard to the epidemiologic studies of respiratory effects, the Administrator recognized that, as a whole, these investigations of associations between O₃ and respiratory effects and health outcomes (e.g., asthma-related hospital admission and emergency department visits) provided strong support for the conclusions of causality but the studies were less informative regarding exposure concentrations associated with O₃ air quality conditions that meet the current standard. He noted that the evidence base in the 2020 review did not include new evidence of respiratory effects associated with appreciably different exposure circumstances than the evidence available in the 2015 review, including particularly any circumstances that would also be expected to be associated with air quality conditions likely to occur under the current standard.

²² The CASAC noted that “[a]rguably the most important potential adverse effect of acute ozone exposure in a child with asthma is not whether it causes a transient decrement in lung function, but whether it causes an asthma exacerbation” and that O₃ “has respiratory effects beyond its well-described effects on lung function,” including increases in airway inflammation which also have the potential to increase the risk for an asthma exacerbation. The CASAC further cautioned with regard to repeated episodes of airway inflammation, indicating that they have the potential to contribute to irreversible reductions in lung function (Cox, 2020, Consensus Responses to Charge Questions pp. 7–8).

1 specific criteria), the statements, in discussing what constitutes an adverse health effect, do not
2 comprehensively describe all the biological responses raised, e.g., with regard to magnitude,
3 duration or frequency of small pollutant-related changes in lung function.

4 The Administrator also recognized the limitations in the available evidence base with
5 regard to our understanding of these aspects of such changes that may be associated with
6 exposure concentrations of interest (e.g., as estimated in the exposure analysis). Notwithstanding
7 these limitations and associated uncertainties, he took note of the emphasis of the earlier ATS
8 statement on consideration of individuals with preexisting compromised function, such as that
9 resulting from asthma (an emphasis which is reiterated and strengthened in the current
10 statement), agreeing that these were important considerations in his judgment on the adequacy of
11 protection provided by the current standard for at-risk populations.

12 Among such important considerations, it was recognized that the controlled human
13 exposure studies, primarily conducted in healthy adults, on which the depth of our understanding
14 of O₃-related health effects is based, in combination with the larger evidence base, informs our
15 conceptual understanding of O₃ responses in people with asthma and in children. Aspects of the
16 EPA's understanding continue to be limited, however, including with regard to the risk of
17 particular effects and associated severity for these less studied population groups that may be
18 posed by 7-hour exposures with exercise to concentrations as low as 60 ppb that are estimated in
19 the exposure analyses for the 2020 review (85 FR 87303, December 31, 2020).

20 Collectively, these aspects of the evidence and associated uncertainties contributed to the
21 recognition that for O₃ in the 2020 review, as for other pollutants and other reviews, the available
22 evidence base in a NAAQS review generally reflects a continuum, consisting of levels at which
23 scientists generally agree that health effects are likely to occur, through lower levels at which the
24 likelihood and magnitude of the response become increasingly uncertain. As is the case in
25 NAAQS reviews in general, the 2020 decision regarding the primary O₃ standard depended on a
26 variety of factors, including science policy judgments and public health policy judgments. These
27 factors included judgments regarding aspects of the evidence and exposure/risk estimates, such
28 as judgments concerning the Administrator's interpretation of the different benchmark
29 concentrations, in light of the available evidence and of associated uncertainties, as well as
30 judgments on the public health significance of the effects that have been observed at the
31 exposures evaluated in the health effects evidence. These judgments are rooted in interpretation
32 of the evidence, which reflects a continuum of health-relevant exposures, with less confidence
33 and greater uncertainty in the existence of adverse health effects as one considers lower O₃
34 exposures. The factors relevant to judging the adequacy of the standards also included the
35 interpretation of, and decisions as to the relative weight to place on, different aspects of the
36 results of the exposure and risk assessment for the areas studied and the associated uncertainties.

1 Together, factors identified here informed the Administrator’s judgment about the degree of
2 protection that is requisite to protect public health with an adequate margin of safety, including
3 the health of sensitive groups, and, accordingly, his conclusion of the requisiteness of the
4 existing standard to protect public health with an adequate margin of safety (85 FR 87303,
5 December 31, 2020).

6 In placing greater weight and giving primary attention to the comparison-to-benchmarks
7 analysis, the Administrator recognized that, as noted in the 2020 PA, the comparison-to-
8 benchmarks analysis (newly updated in the 2020 review with a number of improvements over
9 the 2014 analysis, as described in section 3.4.1 below) provides for characterization of risk for
10 the broad array of respiratory effects documented in the controlled human exposure studies,
11 facilitating consideration of an array of respiratory effects, including but not limited to lung
12 function decrements (85 FR 87294, December 31, 2020). The Administrator recognized the three
13 benchmark concentrations (60, 70 and 80 ppb) to represent exposure conditions (during quasi-
14 continuous exercise) associated with different levels of respiratory response (both with regard to
15 the array of effects and severity of individual effects) in the subjects studied and to inform his
16 judgments on different levels of risk that might be posed to unstudied members of at-risk
17 populations. The highest benchmark concentration (80 ppb) represented an exposure where
18 multiple controlled human exposure studies involving 6.6-hour exposures during quasi-
19 continuous exercise demonstrate a range of O₃-related respiratory effects including inflammation
20 and airway responsiveness, as well as respiratory symptoms and lung function decrements in
21 healthy adult subjects. The second benchmark (70 ppb) represented an exposure level below the
22 lowest exposures that have reported both statistically significant lung function decrements²³ and
23 increased respiratory symptoms (reported at 73 ppb, Schelegle et al 2009) or statistically
24 significant increases in airway resistance and responsiveness (reported at 80 ppb, Horstman et
25 al., 1990). The lowest benchmark (60 ppb) represents still lower exposure, and a level for which
26 findings from controlled human exposure studies of largely healthy subjects have included:
27 statistically significant decrements in lung function (with mean decrements ranging from 1.7% to
28 3.5% across the four studies with average exposures of 60 to 63 ppb), but not respiratory
29 symptoms; and, a statistically significant increase in a biomarker of airway inflammatory
30 response relative to filtered air exposures in one study (Kim et al, 2011).

²³ The study group mean lung function decrement for the 73 ppb exposure was 6%, with individual decrements of 15% or greater (moderate or greater) in about 10% of subjects and decrements of 10% or greater in 19% of subjects. Decrements of 20% or greater were reported in 6.5% of subjects (Schelegle et al., 2009; 2020 PA, Table 3–2 and Appendix 3D, Table 3D–20). In studies of 80 ppb exposure, the percent of study subjects with individual FEV₁ decrements of this size ranged up to nearly double this (2020 PA, Appendix 3D, Table 3D–20).

1 In turning to the exposure/risk analysis results, the Administrator considered the
2 controlled human exposure evidence represented by these benchmarks noting that due to
3 differences among individuals in responsiveness, not all people experiencing exposures (e.g., to
4 73 ppb), experience a response, such as a lung function decrement, and among those
5 experiencing a response, not all will experience an adverse effect (85 FR 87304, December 31,
6 2020). Accordingly, the Administrator noted that not all people estimated to experience an
7 exposure of 7-hour duration while at elevated exertion above even the highest benchmark would
8 be expected to experience an adverse effect, even members of at-risk populations. With these
9 considerations in mind, he noted that while single occurrences could be adverse for some people,
10 particularly for the higher benchmark concentration where the evidence base is stronger, the
11 potential for adverse response and greater severity increased with repeated occurrences (as
12 cautioned by the CASAC). The Administrator also noted that while the exposure/risk analyses
13 provide estimates of exposures of the at-risk population to concentrations of potential concern,
14 they do not provide information on how many of such populations will have an adverse health
15 outcome. Accordingly, in considering the exposure/risk analysis results, while giving due
16 consideration to occurrences of one or more days with an exposure at or above a benchmark,
17 particularly the higher benchmarks, he judged multiple occurrences to be of greater concern than
18 single occurrences.

19 In this context, the Administrator considered the exposure risk estimates, focusing first on
20 the results for the highest benchmark concentration (80 ppb), which represents an exposure well
21 established to elicit an array of responses in sensitive individuals among study groups of largely
22 healthy adult subjects, exposed while at elevated exertion. Similar to judgments of past
23 Administrators, the Administrator in 2020 judged these effects in combination and severity to
24 represent adverse effects for individuals in the population group studied, and to pose a risk of
25 adverse effects for individuals in at-risk populations, most particularly people with asthma, as
26 noted above. Accordingly, he judged that the primary standard should provide protection from
27 such exposures. In considering the exposure/risk estimates, he focused on the results for children,
28 and children with asthma, given the higher frequency of exposures of potential concern for
29 children compared to adults, in terms of percent of the population groups. The exposure/risk
30 estimates indicated more than 99.9% to 100% of children and children with asthma, on average
31 across the three years, to be protected from one or more occasions of exposure at or above this
32 level; the estimate is 99.9% of children with asthma and of all children for the highest year and
33 study area (85 FR 87279, Table 2, December 31, 2020). Further, no children in the simulated
34 populations (zero percent) were estimated to be exposed more than once (two or more occasions)
35 in the 3-year simulation to 7-hr concentrations, while at elevated exertion, at or above 80 ppb (85
36 FR 87279, Table 2, December 31, 2020). These estimates indicated strong protection against

1 exposures of at-risk populations that have been demonstrated to elicit a wide array of respiratory
2 responses in multiple studies (85 FR 87304, December 31, 2020).

3 The Administrator next considered the results for the second benchmark concentration
4 (70 ppb), which is just below the lowest exposure concentration (73 ppb) for which a study has
5 reported a combination of a statistically significant increase in respiratory symptoms and
6 statistically significant lung function decrements in sensitive individuals in a study group of
7 largely healthy adult subjects, exposed while at elevated exertion (Schelegle et al., 2009).
8 Recognizing the lack of evidence for people with asthma from studies at 80 ppb and 73 ppb, as
9 well as the emphasis in the ATS statement on the vulnerability of people with compromised
10 respiratory function, such as people with asthma, the Administrator judged it appropriate that the
11 standard protect against exposure, particularly multiple occurrences of exposure, to somewhat
12 lower levels. In so doing, he noted that the exposure/risk estimates indicate more than 99% of
13 children with asthma, and of all children, to be protected from one or more occasions in a year,
14 on average, of 7-hour exposures to concentrations at or above 70 ppb, while at elevated exertion
15 (85 FR 87279, Table 2, December 31, 2020). The estimate is 99% of children with asthma for
16 the highest year and study area (85 FR 87279, Table 2, December 31, 2020). Further, he noted
17 that 99.9% of these groups were estimated to be protected from two or more such occasions, and
18 100% from still more occasions. These estimates also indicated strong protection of at-risk
19 populations against exposures similar to those demonstrated to elicit lung function decrements
20 and increased respiratory symptoms in healthy subjects, a response described as adverse by the
21 ATS (85 FR 87304, December 31, 2020).

22 In consideration of the exposure/risk results for the lowest benchmark (60 ppb), the
23 Administrator noted that the lung function decrements in controlled human exposure studies of
24 largely healthy adult subjects exposed while at elevated exertion to concentrations of 60 ppb,
25 although statistically significant, were much reduced from that observed in the next higher
26 studied concentration (73 ppb), both at the mean and individual level, and were not reported to
27 be associated with increased respiratory symptoms in healthy subjects.²⁴ In light of these results
28 and the transient nature of the responses, the Administrator did not judge these responses to
29 represent adverse effects for generally healthy individuals. However, he further considered these
30 findings specifically with regard to protection of at-risk populations, such as people with asthma.
31 In this regard, he noted that such data are lacking for at-risk groups, such as people with asthma,
32 and considered the evidence and comments from the CASAC regarding the need to consider
33 endpoints of particular importance for this population group, such as risk of asthma exacerbation

²⁴ The response for the 60 ppb studies is also somewhat lower than that for the 63 ppb study (Table 1; 2020 PA, Appendix 3D, Table 3D-20).

1 and prolonged inflammation. He took note of comments from the CASAC (and also noted in the
2 ATS statement) that small lung function decrements in this at-risk group may contribute to a risk
3 of asthma exacerbation, an outcome described by the CASAC as “arguably the most important
4 potential adverse effect” of O₃ exposure for a child with asthma. Thus, he judged it important for
5 the standard to provide protection that reduces such risks. With regard to the inflammatory
6 response, he noted the evidence indicating the role of repeated occurrences of inflammation in
7 contributing to severity of response. Thus, he found repeated occurrences of exposure events of
8 potential concern to pose greater risk than single events, leading him to place greater weight on
9 exposure/risk estimates for multiple occurrences (85 FR 87304-87305, December 31, 2020).

10 Thus, in this context, and given that the 70 ppb benchmark represents an exposure level
11 somewhat below the lowest exposure concentration for which both statistically significant lung
12 function decrements and increased respiratory symptoms have been reported in largely healthy
13 adult subjects, the Administrator considered the exposure/risk estimates for the third benchmark
14 of 60 ppb to be informative most particularly to his judgments on an adequate margin of safety.
15 In so doing, he took note that these estimates indicate more than 96% to more than 99% of
16 children with asthma to be protected from more than one occasion in a year (two or more), on
17 average, of 7-hour exposures to concentrations at or above this level (60 ppb), while at elevated
18 exertion (85 FR 87279, Table 2, December 31, 2020). Additionally, the analysis estimates more
19 than 90% of all children, on average across the three years, to be protected from one or more
20 occasions of exposure at or above this level. The Administrator found this to indicate an
21 appropriate degree of protection from such exposures (85 FR 87305, December 31, 2020).

22 The Administrator additionally considered whether it was appropriate to consider a more
23 stringent standard that might be expected to result in reduced O₃ exposures. As an initial matter,
24 he considered the advice from the CASAC. With regard to the CASAC advice, while part of the
25 Committee concluded the evidence supported retaining the current standard without revision,
26 another part of the Committee reiterated advice from the prior CASAC, which while including
27 the current standard level among the range of recommended standard levels, also provided policy
28 advice to set the standard at a lower level. In considering this advice in the 2020 review, as it was
29 raised by part of the then-current CASAC, the Administrator noted the slight differences of the
30 current exposure and risk estimates from the corresponding 2014 estimates for the lowest
31 benchmark, which were those considered by the CASAC in 2014 (85 FR 87280, Table 3,
32 December 31, 2020). For example, while the 2014 HREA estimated 3.3 to 10.2% of children, on
33 average, to experience one or more days with exposures at or above 60 ppb (and as many as
34 18.9% in a single year), the comparable estimates for the current analyses are lower (3.2 to 8.2%
35 on average and 10.6% in a single year), particularly with regard to the upper end of the range of
36 averages and the highest in a single year. While the estimates for two or more days with

1 occurrences at or above 60 ppb, on average across the assessment period, were more similar
2 between the two assessments, the 2020 estimate for the single highest year was much lower (9.2
3 versus 4.3%). The Administrator additionally recognized the 2020 PA finding that the factors
4 contributing to these differences, which includes the use of air quality data reflecting
5 concentrations much closer to the now-current standard than was the case in the 2015 review,
6 also contribute to a reduced uncertainty in the current estimates (85 FR 87275-87279, December
7 31, 2020; 2020 PA, sections 3.4 and 3.5). Thus, he noted that the exposure analysis estimates in
8 the 2020 review indicate the current standard to provide appreciable protection against multiple
9 days with a maximum exposure at or above 60 ppb. In the context of his consideration of the
10 adequacy of protection provided by the standard and of the CAA requirement that the standard
11 protect public health, including the health of at-risk populations, with an adequate margin of
12 safety, the Administrator concluded, “in light of all of the considerations raised here, that the
13 current standard provides appropriate protection, and that a more stringent standard would be
14 more than requisite to protect public health” (85 FR 87306; December 31, 2020).

15 Therefore, based on his consideration of the evidence and exposure/risk information,
16 including that related to the lowest exposures studied in controlled human exposure studies, and
17 the associated uncertainties, the Administrator judged that the current standard provides the
18 requisite protection of public health, including an adequate margin of safety, and thus should be
19 retained, without revision. Accordingly, he also concluded that a more stringent standard was not
20 needed to provide requisite protection and that the current standard provides the requisite
21 protection of public health under the Act (85 FR 87306, December 31, 2020).

22 **3.2 GENERAL APPROACH AND KEY ISSUES**

23 As is the case for primary NAAQS reviews, this reconsideration of the 2020 decision on
24 the primary O₃ standard is fundamentally based on using the Agency’s assessment of the
25 scientific evidence and associated quantitative analyses to inform the Administrator’s judgments
26 regarding a primary standard that is requisite to protect public health with an adequate margin of
27 safety. This approach builds on the substantial assessments and evaluations performed over the
28 course of O₃ NAAQS reviews to inform our understanding of the key-policy relevant issues in
29 this reconsideration of the 2020 decision.

30 The evaluations in the PA of the scientific assessments in the ISA (building on prior such
31 assessments), augmented by the quantitative risk and exposure analyses,²⁵ are intended to inform

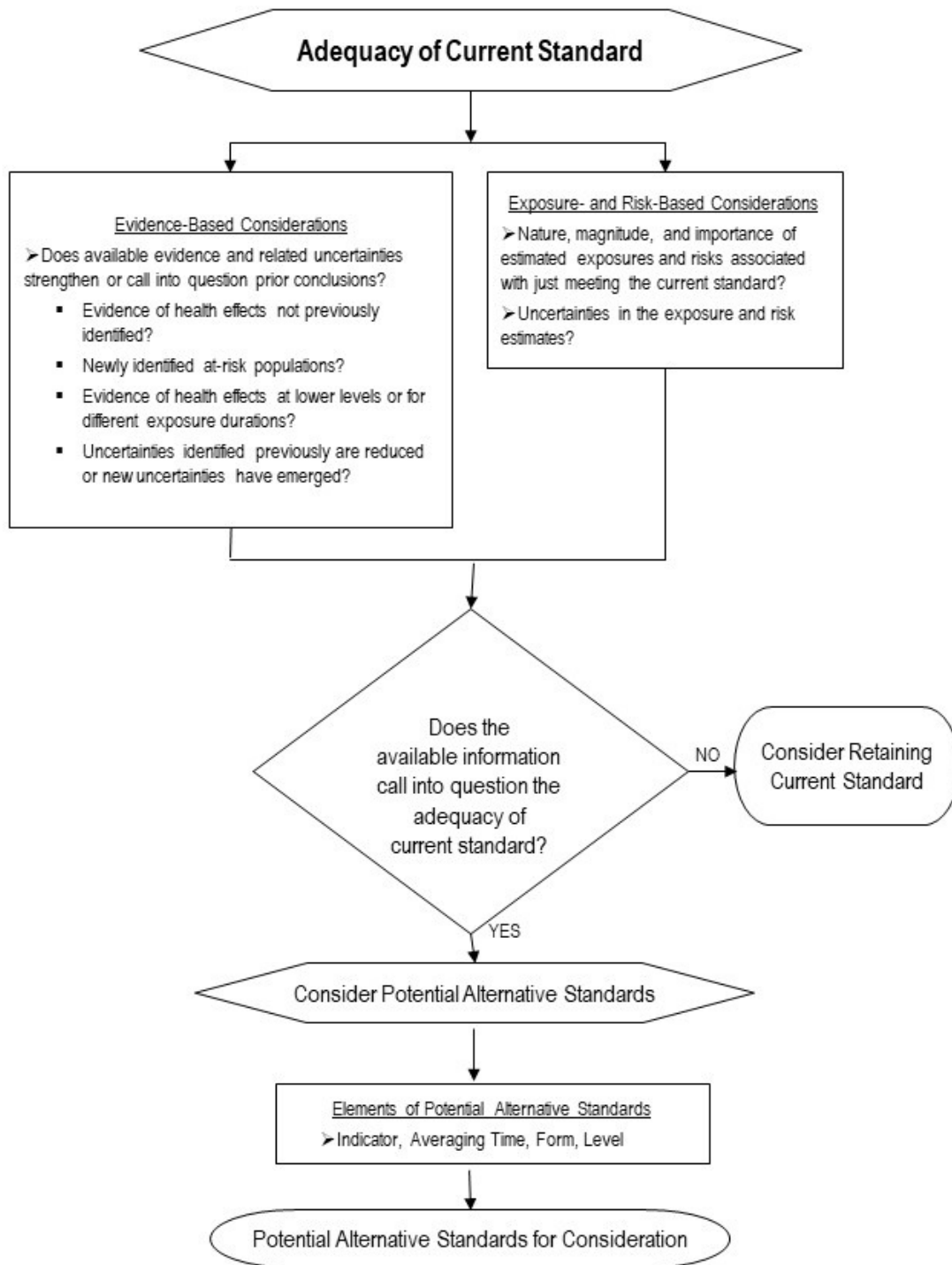
²⁵ The overarching purpose of the quantitative exposure and risk analyses is to inform the Administrator’s conclusions on the public health protection afforded by the current primary standard. An important focus is the assessment, based on current tools and information, of the potential for exposures and risks beyond those indicated by the information available at the time the standard was established.

1 the Administrator’s public health policy judgments and conclusions, including his decisions
2 regarding the O₃ standards. The PA considers the potential implications of various aspects of the
3 scientific evidence, the exposure/risk-based information, and the associated uncertainties and
4 limitations. Thus, the approach for this PA is to draw on the evaluation of the scientific and
5 technical information available in the 2020 review to address a series of key policy-relevant
6 questions using both evidence- and exposure/risk-based considerations. Together, consideration
7 of the available evidence and information will inform the answer to the following initial
8 overarching question:

- 9 • **Do the available scientific evidence and exposure-/risk-based information support or**
10 **call into question the adequacy of the public health protection afforded by the current**
11 **primary O₃ standard?**

12 In reflecting on this question, we will consider the body of scientific evidence, assessed
13 in the 2020 ISA and used as a basis for developing or interpreting exposure/risk analyses,
14 including whether it supports or calls into question the scientific conclusions reached in the 2020
15 review regarding health effects related to exposure to ambient air-related O₃. Information that
16 may be informative to public health judgments regarding significance or adversity of key effects
17 is also be considered. Additionally, the available exposure and risk information will be
18 considered, including with regard to the extent to which it may continue to support judgments
19 made in the 2020 review. Further, in considering this question with regard to the primary O₃
20 standard, as in all NAAQS reviews, we give particular attention to exposures and health risks to
21 at-risk populations.²⁶ Evaluation of the available scientific evidence and exposure/risk
22 information with regard to consideration of the current standard and the overarching question
23 above focuses on key policy-relevant issues by addressing a series of questions on specific
24 topics. Figure 3-1 summarizes, in general terms, the approach to considering the available
25 information in the context of policy-relevant questions pertaining to the primary standard.

²⁶ 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 lifestage. 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 O₃. *Vulnerability* refers to an increased risk of O₃-related health effects due to factors such as those related to socioeconomic status, reduced access to health care or exposure.



1
2 **Figure 3-1. Overview of general approach for the primary O₃ standard.**
3

1 The Agency’s approach with regard to the O₃ primary standard 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 these provisions. As discussed in section 1.2
4 above, 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 NAAQS reviews, the approach of the PA to informing these judgments is based on a
8 recognition that the available health effects evidence generally reflects continuums that include
9 ambient air exposures for which scientists generally agree that health effects are likely to occur
10 through lower levels at which the likelihood and magnitude of response become increasingly
11 uncertain. The CAA does not require the Administrator to establish a primary standard at a zero-
12 risk level or at background concentration levels, but rather at a level that reduces risk sufficiently
13 so as to protect public health, including the health of sensitive groups,²⁷ with an adequate margin
14 of safety.

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

24 **3.3 HEALTH EFFECTS EVIDENCE**

25 The health effects evidence on which this PA for the reconsideration of the 2020 decision
26 on the O₃ primary standard will focus is the evidence as assessed and described in the 2020 ISA
27 and prior ISAs or AQCDs. As described in section 1.5 above, the EPA has provisionally
28 considered more recently available studies that were raised in public comments in the 2020
29 review or were identified in a literature search that the EPA conducted for this reconsideration of
30 more recently available controlled human exposure studies (Luben et al., 2020; Duffney et al.

²⁷ 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 2022). The provisional consideration of these studies concluded that, taken in context, the
2 associated information and findings did not materially change any of the broad scientific
3 conclusions of the ISA regarding the health and welfare effects of O₃ in ambient air or warrant
4 reopening the air quality criteria for this review. Thus, the discussion below focuses on the health
5 effects evidence assessment, with associated conclusions, as described in the 2020 ISA.

6 **3.3.1 Nature of Effects**

7 The health effects evidence base for O₃ includes decades of extensive evidence that
8 clearly describes the role of O₃ in eliciting an array of respiratory effects and the more recent
9 evidence suggests the potential for relationships between O₃ exposure and other effects. As was
10 established in prior O₃ NAAQS reviews, the most commonly observed effects, and those for
11 which the evidence is strongest are transient decrements in pulmonary function and respiratory
12 symptoms, such as coughing and pain on deep inspiration, as a result of short-term exposures
13 particularly when breathing at elevated rates (ISA, section IS.4.3.1; 2013 ISA, p. 2-26). These
14 effects are demonstrated in the large, long-standing evidence base of controlled human exposure
15 studies²⁸ (1978 AQCD, 1986 AQCD, 1996 AQCD, 2006 AQCD, 2013 ISA, ISA). Lung function
16 effects are also positively associated with ambient air O₃ concentrations in epidemiologic panel
17 studies, available in past reviews, that describe these associations for outdoor workers and
18 children attending summer camps in the 1980s and 1990s (2013 ISA, section 6.2.1.2; ISA,
19 Appendix 3, section 3.1.4.1.3). Collectively, the epidemiologic evidence base documents
20 consistent, positive associations of O₃ concentrations in ambient air with lung function effects in
21 epidemiologic panel studies²⁹ and with more severe health outcomes in other epidemiologic
22 studies, including asthma-related emergency department visits and hospital admissions (2013
23 ISA, sections 6.2.1.2 and 6.2.7; ISA, Appendix 3, sections 3.1.4.1.3, 3.1.5.1 and 3.1.5.2).
24 Extensive animal toxicological evidence informs a detailed understanding of mechanisms
25 underlying the respiratory effects of short-term exposures, and studies in animal models also
26 provide evidence for effects of longer-term O₃ exposure on the developing lung (ISA, Appendix
27 3, sections 3.1.11 and 3.2.6).

²⁸ The vast majority of the controlled human exposure studies (and all of the studies conducted at the lowest exposures) involved young healthy adults (typically 18-35 years old) as study subjects (ISA, section 3.1.4; 2013 ISA, section 6.2.1.1). There are also some 1-8 hr controlled human exposure studies in older adults and adults with asthma, and there are still fewer controlled human exposure studies in healthy children (i.e., individuals aged younger than 18 years) or children with asthma (See, for example, Appendix 3A, Table 3A-3).

²⁹ Panel studies are a type of longitudinal epidemiologic study. The studies referenced here include a number of such past studies investigating O₃ and lung function measures in groups of children attending summer camp and respiratory symptoms in groups of children with asthma (ISA, sections 3.1.4.1.3 and 3.1.5.3; 2013 ISA, sections 6.2.1.2 and 6.2.4.1).

- 1 • **Does the available scientific evidence alter prior conclusions regarding the health**
2 **effects attributable to exposure to O₃?**

3 The available scientific evidence, as assessed in the ISA, continues to support the prior
4 conclusion that short-term O₃ exposure causes respiratory effects. Specifically, the full body of
5 evidence continues to support the conclusions of a causal relationship of respiratory effects with
6 short-term O₃ exposures and a likely causal relationship of respiratory effects with longer-term
7 exposures (ISA, sections IS.4.3.1 and IS.4.3.2). The evidence base described in the 2020 ISA
8 which is expanded from the evidence available in the 2015 review (and described in the 2013
9 ISA), also indicates a likely causal relationship between short-term O₃ exposure and metabolic
10 effects,³⁰ which were not evaluated as a separate category of effects in the 2015 review when less
11 evidence was available (ISA, section IS.4.3.3). The more recent evidence is primarily from
12 experimental animal research. For other types of health effects, recent evidence has led to
13 different conclusions from those reached previously. Specifically, the evidence base described in
14 the 2020 ISA, particularly in light of the additional controlled human exposure studies, is less
15 consistent than what was previously available and less indicative of O₃-induced cardiovascular
16 effects.³¹ This recent evidence has altered conclusions from the 2015 review with regard to
17 relationships between short-term O₃ exposures and cardiovascular effects and mortality, such
18 that likely causal relationships are no longer concluded.³² Thus, as discussed in the ISA,
19 conclusions have changed for some effects based on the recent evidence, and conclusions are
20 newly reached for an additional category of health effects. The prior conclusions on respiratory
21 effects, however, continue to be supported.

22 **3.3.1.1 Respiratory Effects**

23 The available evidence, as described in the 2020 ISA, continues to support the conclusion
24 of a causal relationship between short-term O₃ exposure and respiratory effects (ISA, section
25 IS.1.3.1). The strongest evidence for this comes from controlled human exposure studies

³⁰ The term “metabolic effects” is used in the ISA to refer metabolic syndrome (a collection of risk factors including high blood pressure, elevated triglycerides and low high density lipoprotein cholesterol), diabetes, metabolic disease mortality, and indicators of metabolic syndrome that include alterations in glucose and insulin homeostasis, peripheral inflammation, liver function, neuroendocrine signaling, and serum lipids (ISA, section IS.4.3.3).

³¹ As described in the ISA, “[t]he number of controlled human exposure studies showing little evidence of ozone induced cardiovascular effects has grown substantially” and “the plausibility for a relationship between short-term ozone exposure to cardiovascular health effects is weaker than it was in the previous review, leading to the revised causality determination” (ISA, p. IS-43).

³² The evidence for cardiovascular, reproductive and nervous system effects, as well as mortality, is “suggestive of, but not sufficient to infer” a causal relationship with short- or long-term O₃ exposures (ISA, Table IS-1). The evidence is inadequate to infer the presence or absence of a causal relationship between long-term O₃ exposure and cancer (ISA, section IS4.3.6.6).

1 demonstrating O₃-related respiratory effects in generally healthy adults.³³ The key evidence
2 comes from the body of controlled human exposure studies that document respiratory effects in
3 people exposed for short periods (6.6 to 8 hours) during quasi-continuous exercise.³⁴ The
4 potential for O₃ exposure to elicit health outcomes more serious than those assessed in the
5 experimental studies, particularly for children with asthma, continues to be indicated by the
6 epidemiologic evidence of associations of O₃ concentrations in ambient air with increased
7 incidence of hospital admissions and emergency department visits for an array of health
8 outcomes, including asthma exacerbation, COPD exacerbation, respiratory infection, and
9 combinations of respiratory diseases (ISA, Appendix 3, sections 3.1.5 and 3.1.6). The strongest
10 such evidence is for asthma-related outcomes and specifically asthma-related outcomes for
11 children, indicating an increased risk for people with asthma and particularly children with
12 asthma (ISA, Appendix 3, section 3.1.5.7).

13 Respiratory responses observed in human subjects exposed to O₃ for periods of 8 hours or
14 less, while intermittently or quasi-continuously exercising, include reduced lung function
15 decrements (e.g., based on forced expiratory volume in one second [FEV₁] measurements),³⁵
16 respiratory symptoms, increased airway responsiveness, mild bronchoconstriction (measured as a
17 change in specific airway resistance [sRaw]), and pulmonary inflammation, with associated
18 injury and oxidative stress (ISA, Appendix 3, section 3.1.4; 2013 ISA, sections 6.2.1 through
19 6.2.4). The available mechanistic evidence, discussed in greater detail in the ISA, describes
20 pathways involving the respiratory and nervous systems by which O₃ results in pain-related
21 respiratory symptoms and reflex inhibition of maximal inspiration (inhaling a full, deep breath),
22 commonly quantified by decreases in forced vital capacity (FVC) and total lung capacity. This
23 reflex inhibition of inspiration combined with mild bronchoconstriction contributes to the

³³ The phrases “healthy adults” or “healthy subjects” are used to distinguish from subjects with asthma or other respiratory diseases, because “the study design generally precludes inclusion of subjects with serious health conditions,” such as individuals with severe respiratory diseases (2013 ISA, p. lx).

³⁴ A quasi-continuous exercise protocol is common to these controlled exposure studies where, in the case of a 6.6-hour study, subjects complete six 50-minute periods of exercise, each followed by 10-minute periods of rest, in addition to a 30-minute lunch exposure period at rest (e.g., ISA, Appendix 3, section 3.1.4.1.1, and p. 3–11; 2013 ISA, section 6.2.1.1).

³⁵ The measure of lung function response most commonly considered across O₃ NAAQS reviews is changes in FEV₁. In considering controlled human exposure studies, an O₃-induced change in FEV₁ is typically the difference between the decrement observed with O₃ exposure ([post-exposure FEV₁ minus pre-exposure FEV₁] divided by pre-exposure FEV₁) and what is generally an improvement observed with filtered air (FA) exposure ([postexposure FEV₁ minus pre-exposure FEV₁] divided by pre-exposure FEV₁). As explained in the 2013 ISA, “[n]oting that some healthy individuals experience small improvements while others have small decrements in FEV₁ following FA exposure, investigators have used the randomized, crossover design with each subject serving as their own control (exposure to FA) to discern relatively small effects with certainty since alternative explanations for these effects are controlled for by the nature of the experimental design” (2013 ISA, pp. 6-4 to 6-5).

1 observed decrease in forced expiratory volume in one second (FEV₁), the most common metric
2 used to assess O₃-related pulmonary function effects. The evidence also indicates that the
3 additionally observed inflammatory response is correlated with mild airway obstruction,
4 generally measured as an increase in sRaw (ISA, Appendix 3, section 3.1.3). As described in
5 section 3.3.3 below, the prevalence and severity of respiratory effects in controlled human
6 exposure studies, including symptoms (e.g., pain on deep inspiration, shortness of breath, and
7 cough) increases, with increasing O₃ concentration, exposure duration, and ventilation rate of
8 exposed subjects (ISA, Appendix 3, sections 3.1.4.1 and 3.1.4.2).

9 Within the evidence base from controlled human exposure studies, the majority of studies
10 involve healthy adult subjects (generally 18 to 35 years old), although there are studies involving
11 subjects with asthma, and a limited number of studies, generally of durations shorter than four
12 hours, involving adolescents and adults older than 50 years. A summary of salient observations
13 of O₃ effects on lung function, based on the controlled human exposure study evidence reviewed
14 in the 1996 and 2006 AQCDs, and recognized in the 2013 ISA, continues to pertain to this
15 evidence base as it exists today “(1) young healthy adults exposed to ≥80 ppb O₃ develop
16 significant reversible, transient decrements in pulmonary function and symptoms of breathing
17 discomfort if minute ventilation (\dot{V}_E) or duration of exposure is increased sufficiently [i.e., as
18 measured by FEV₁ and/or FVC]; (2) relative to young adults, children experience similar
19 spirometric responses but lower incidence of symptoms from O₃ exposure; (3) relative to young
20 adults, ozone-induced spirometric responses are decreased in older individuals; (4) there is a
21 large degree of inter-subject variability in physiologic and symptomatic responses to O₃, but
22 responses tend to be reproducible within a given individual over a period of several months; and
23 (5) subjects exposed repeatedly to O₃ for several days experience an attenuation of spirometric
24 and symptomatic responses on successive exposures, which is lost after about a week without
25 exposure” (ISA, Appendix 3, section 3.1.4.1.1, p. 3-11).³⁶

26 The evidence is most well established with regard to the effects, reversible with the
27 cessation of exposure, that are associated with short-term exposures of several hours. For
28 example, the evidence indicates a rapid recovery from O₃-induced lung function decrements
29 (e.g., reduced FEV₁) and respiratory symptoms (2013 ISA, section 6.2.1.1). However, in some
30 cases, such as after exposure to higher concentrations such as 300 ppb, the recovery phase may
31 be slower and involve a longer time period (e.g., at least 24 hours [hrs]). Repeated daily exposure
32 studies at such higher concentrations also have found FEV₁ response to be enhanced on the
33 second day of exposure. This enhanced response is absent, however, with repeated exposure at

³⁶ A spirometric response refers to a change in the amount of air breathed out of the body (forced expiratory volumes) and the associated time to do so (e.g., FEV₁).

1 lower concentrations, perhaps as a result of a more complete recovery or less damage to
2 pulmonary tissues (2013 ISA, section pp. 6-13 to 6-14; Folinsbee et al., 1994).

3 With regard to airway inflammation and the potential for repeated occurrences to
4 contribute to further effects, O₃-induced respiratory tract inflammation “can have several
5 potential outcomes: (1) inflammation induced by a single exposure (or several exposures over
6 the course of a summer) can resolve entirely; (2) continued acute inflammation can evolve into a
7 chronic inflammatory state; (3) continued inflammation can alter the structure and function of
8 other pulmonary tissue, leading to diseases such as fibrosis; (4) inflammation can alter the
9 body’s host defense response to inhaled microorganisms, particularly in potentially at-risk
10 populations such as the very young and old; and (5) inflammation can alter the lung’s response to
11 other agents such as allergens or toxins” (2013 ISA, p. 6-76; ISA Appendix 3, section 3.1.5.6).
12 With regard to O₃-induced increases in airway responsiveness, the controlled human exposure
13 study evidence for healthy adults generally indicates a resolution within 18 to 24 hours after
14 exposure, with slightly longer persistence in some individuals (ISA, Appendix 3, section
15 3.1.4.3.1; 2013 ISA, p. 6–74; Folinsbee and Hazucha, 2000).

16 The extensive evidence base for O₃-related health effects, compiled over several decades,
17 continues to indicate respiratory responses to short exposures as the most sensitive effects of O₃.
18 This array of respiratory effects, including reduced lung function, respiratory symptoms,
19 increased airway responsiveness, and inflammation are of increased significance to people with
20 asthma given aspects of the disease that contribute to a baseline status that includes chronic
21 airway inflammation and greater airway responsiveness than people without asthma (ISA,
22 section 3.1.5). For example, O₃ exposure of a magnitude that increases airway responsiveness
23 may put such people at potential increased risk for prolonged bronchoconstriction in response to
24 asthma triggers (ISA, Appendix 3, p. 3–7, 3–28; 2013 ISA, section 6.2.9; 2006 AQCD, section
25 8.4.2). The increased significance of effects in people with asthma and risk of increased exposure
26 for children (from greater frequency of outdoor exercise as described in Section 3.3.2) is
27 illustrated by the epidemiological findings of positive associations between O₃ exposure and
28 asthma-related emergency department visits and hospital admissions for children with asthma.
29 Thus, the evidence indicates O₃ exposure to increase the risk of asthma exacerbation, and
30 associated outcomes, in children with asthma.

31 With regard to an increased susceptibility to infectious diseases, the experimental animal
32 evidence continues to indicate, as described in the 2013 ISA and past AQCDs, a potential role
33 for O₃ exposures through effects on defense mechanisms of the respiratory tract (2013 ISA,
34 section 6.2.5). Evidence regarding respiratory infections and associated effects has been
35 augmented by a number of epidemiologic studies reporting positive associations between short-

1 term O₃ concentrations and emergency department visits for a variety of respiratory infection
2 endpoints (ISA, Appendix 3, section 3.1.7; 2013 ISA, section 6.2.5).

3 Although the long-term exposure conditions that may contribute to further respiratory
4 effects are less well understood, the evidence-based conclusion remains that there is likely to be
5 a causal relationship for such exposure conditions with respiratory effects (ISA, section IS.4.3.2).
6 Most notably, experimental studies, including with nonhuman infant primates, have provided
7 evidence relating O₃ exposure to allergic asthma-like effects, and epidemiologic cohort studies
8 have reported associations of O₃ concentrations in ambient air with asthma development in
9 children (ISA, Appendix 3, section 3.2.4.1.3 and 3.2.6). The biological plausibility of such a role
10 for O₃ has been indicated by animal toxicological evidence on biological mechanisms (ISA,
11 Appendix 3, sections 3.2.3 and 3.2.4.1.2). Specifically, the animal evidence, including the
12 nonhuman primate studies of early life O₃ exposure, indicates that such exposures can cause
13 “structural and functional changes that could potentially contribute to airway obstruction and
14 increased airway responsiveness,” which are hallmarks of asthma (ISA, Appendix 3, section
15 3.2.6, p. 3-113).

16 Overall, the recent respiratory effects evidence is generally consistent with the evidence
17 base in the 2015 review (ISA, Appendix 3, section 3.1.4). A few recent studies provide insights
18 in previously unexamined areas, both with regard to human study groups and animal models for
19 different effects, while other studies confirm and provide depth to prior findings with updated
20 protocols and techniques (ISA, Appendix 3, sections 3.1.11 and 3.2.6). Thus, our current
21 understanding of the respiratory effects of O₃ is similar to that in the 2015 review.

22 One aspect of the evidence, augmented in the 2020 review as compared with the 2015
23 review, concerns pulmonary function in adults older than 50 years of age. Previously available
24 evidence in this age group indicated smaller O₃-related decrements in middle-aged adults (35 to
25 60 years) than in adults 35 years of age and younger (2006 AQCD, p. 6-23; 2013 ISA, p. 6-22;
26 ISA, Appendix 3, section 3.1.4.1.1.2). A recent multicenter study of 55- to 70-year old subjects
27 (average of 60 years), conducted for a 3-hour duration involving alternating 15-minute rest and
28 exercise periods and a 120 ppb exposure concentration, reported a statistically significant O₃
29 FEV₁ response (ISA, Appendix 3, section 3.1.4.1.1.2; Arjomandi et al., 2018). While there is not
30 a precisely comparable study in younger adults, the mean response for the 55- to 70-year olds,
31 1.2% O₃-related FEV₁ decrement, is lower than results for somewhat comparable exposures in
32 adults aged 35 or younger, suggesting somewhat reduced responses to O₃ exposure in this older
33 age group (ISA, Appendix 3, section 3.1.4.1.1.2; Arjomandi et al., 2018; Adams, 2000; Adams,

1 2006a).³⁷ Such a reduced response in middle-aged and older adults compared to young adults is
2 consistent with conclusions in the past (2013 ISA, section 6.2.1.1; 2006 AQCD, section 6.4).

3 The strongest evidence of O₃-related health effects continues to document the respiratory
4 effects of O₃ (ISA, section ES.4.1). There are no new studies, however, of 6.6-hour exposures
5 (with exercise) to O₃ concentrations below those previously studied.³⁸ Among the newly
6 available studies in the 2020 ISA, are several controlled human exposure studies that
7 investigated lung function effects of higher exposure concentrations (e.g., 100 to 300 ppb) in
8 healthy individuals younger than 35 years old, with findings generally consistent with previous
9 studies (ISA, Appendix 3, section 3.4.1.1.2, p. 3-17). The newly available animal toxicological
10 studies augment the previously available information concerning mechanisms underlying the
11 effects documented in experimental studies. Lastly, newly available epidemiologic studies of
12 hospital admissions and emergency department visits for a variety of respiratory outcomes
13 supplement the previously available evidence with additional findings of consistent associations
14 with O₃ concentrations across a number of study locations (ISA, Appendix 3, sections 3.1.4.1.3,
15 3.1.5, 3.1.6.1.1, 3.1.7.1 and 3.1.8). These studies include a number that report positive
16 associations for asthma-related outcomes, as well as a few for COPD-related outcomes. Together
17 these epidemiologic studies continue to indicate the potential for O₃ exposures to contribute to
18 such serious health outcomes, particularly for people with asthma.

19 **3.3.1.2 Other Effects**

20 As was the case for the evidence available previously, the evidence for health effects
21 other than those on the respiratory system is more uncertain than that for respiratory effects. For
22 some of these other categories of effects, the more recent evidence as described in the 2020 ISA
23 has contributed to changes to conclusions reached in the 2015 review. For example,
24 cardiovascular effects and mortality are no longer concluded to be likely causally related to O₃
25 exposures based on newly available evidence in combination with the uncertainties that had been
26 recognized for the previously available evidence. Additionally, newly available evidence also led

³⁷ For the same exposure concentration of 120 ppb, Adams (2006a) observed an average 3.2%, statistically significant, O₃-related FEV₁ decrement in young adults (average age 23 years) at the end of the third hour of an 8-hour protocol that alternated 30 minutes of exercise and rest, with the equivalent ventilation rate (EVR) averaging 20 L/min-m² during the exercise periods (versus 15 to 17 L/min-m² in Arjomandi et al., 2018)]. For the same concentration with a lower EVR during exercise (17 L/min-m²), although with more exercise, Adams (2000) observed a 4%, statistically significant, O₃-related FEV₁ decrement in young adults (average age 22 years) after the third hour of a 6.6-hour protocol (alternating 50 minutes exercise and 10 minutes rest).

³⁸ The 2020 ISA includes a newly available 3-hr study of subjects aged 55 years of age or older that involves a slightly lower target ventilation rate for the exercise periods. The exposure concentrations were 120 ppb and 70 ppb, only the former of which elicited a statistically significant FEV₁ decrement in this age group of subjects (ISA, Appendix 3, section 3.1.4.1.1.2).

1 to conclusions for another category, metabolic effects, for which formal causal determinations
2 were previously not articulated.

3 The ISA finds the evidence for metabolic effects sufficient to conclude that there is likely
4 to be a causal relationship with short-term O₃ exposures (ISA, section IS.4.3.3). The evidence of
5 metabolic effects of O₃ comes primarily from experimental animal study findings that short-term
6 O₃ exposure can impair glucose tolerance, increase triglyceride levels and elicit fasting
7 hyperglycemia and increase hepatic gluconeogenesis (ISA, Appendix 5, section 5.1.8, and Table
8 5-3). The exposure conditions from these studies generally involve much higher O₃
9 concentrations than those commonly occurring in areas of the U.S. where the current standard is
10 met. For example, the animal studies include 4-hour concentrations of 400 to 800 ppb (ISA,
11 Appendix 5, Tables 5-8 and 5-10). In addition, an epidemiologic study of a Taiwanese cohort
12 and 2002 air quality that was available in the 2015 review has reported positive associations of
13 multiday average O₃ concentrations in ambient air with changes in two indicators of glucose and
14 insulin homeostasis (ISA, Appendix 5, sections 5.1.3.1.1 and 5.1.8).

15 The ISA additionally concludes that the evidence is suggestive of, but not sufficient to
16 infer, a causal relationship between long-term O₃ exposures and metabolic effects (ISA, section
17 IS.4.3.6.2). As with metabolic effects and short-term O₃, the primary evidence is from
18 experimental animal studies in which the exposure concentrations are appreciably higher than
19 those commonly occurring in the U.S. For example, the animal studies include exposures over
20 several weeks to concentrations of 250 ppb and higher (ISA, Appendix 5, section 5.2.3.1.1). The
21 somewhat limited epidemiologic evidence related to long-term O₃ concentrations and metabolic
22 effects includes several studies reporting increased odds of being overweight or obese or having
23 metabolic syndrome and increased hazard ratios for diabetes incidence with increased O₃
24 concentrations (ISA, Appendix 5, sections 5.2.3.4.1, 5.2.5 and 5.2.9, Tables 5-12 and 5-15).

25 With regard to cardiovascular effects and total (nonaccidental) mortality and short-term
26 O₃ exposures, the conclusions in the ISA regarding the potential for a causal relationship have
27 changed from what they were in the 2015 review after integrating the previously available
28 evidence with the more recently available evidence. The relationships are now characterized as
29 suggestive of, but not sufficient to infer, a causal relationship (ISA, Appendix 4, section 4.1.17;
30 Appendix 6, section 6.1.8). This reflects several aspects of the evidence base: (1) a now-larger
31 body of controlled human exposure studies providing evidence that is not consistent with a
32 cardiovascular effect in response to short-term O₃ exposure; (2) a paucity of epidemiologic
33 evidence indicating more severe cardiovascular morbidity endpoints,³⁹ that would be expected if

³⁹ These include emergency department visits and hospital admission visits for cardiovascular endpoints including myocardial infarctions, heart failure or stroke (ISA, Appendix 6, section 6.1.8).

1 the impaired vascular and cardiac function (observed in animal toxicological studies) was the
2 underlying basis for cardiovascular mortality (for which epidemiologic studies have reported
3 some positive associations with O₃); and (3) the remaining uncertainties and limitations
4 recognized in the 2013 ISA (e.g., lack of control for potential confounding by copollutants in
5 epidemiologic studies) that still remain. Although there exists consistent or generally consistent
6 evidence for a limited number of O₃-induced cardiovascular endpoints in animal toxicological
7 studies and cardiovascular mortality in epidemiologic studies, there is a general lack of
8 coherence between these results and findings in controlled human exposure and epidemiologic
9 studies of cardiovascular health outcomes (ISA, section IS.1.3.1). Related to this updated
10 conclusion for cardiovascular effects, the evidence for short-term O₃ and mortality is also
11 updated (ISA, Appendix 6, section 6.1.8). While there remain consistent, positive associations
12 between short-term O₃ and total (nonaccidental), respiratory, and cardiovascular mortality (and
13 there are some studies reporting associations to remain after controlling for PM₁₀ and NO₂), the
14 full evidence base does not describe a continuum of effects that could lead to cardiovascular
15 mortality.⁴⁰ Therefore, because cardiovascular mortality is the largest contributor to total
16 mortality, the relatively limited biological plausibility and coherence within and across
17 disciplines for cardiovascular effects (including mortality) contributes to an accompanying
18 change in the causality determination for total mortality (ISA, section IS.4.3.5). Thus, the
19 evidence for cardiovascular effects and total mortality, as evaluated in the ISA, is concluded to
20 be suggestive of, but not sufficient to infer, a causal relationship with short-term (as well as long-
21 term) O₃ exposures (ISA, section IS.1.3.1).

22 For other health effect categories, EPA's conclusions, as described in the ISA, are largely
23 unchanged from those in the 2015 review. For example, the available evidence for reproductive
24 effects, as well as for effects on the nervous system, continue to be suggestive of, but not
25 sufficient to infer, a causal relationship (ISA, section IS.4.3.6). Additionally, the evidence is
26 inadequate to determine if a causal relationship exists between O₃ exposure and cancer (ISA,
27 section IS.4.3.6.6).

28 **3.3.2 Public Health Implications and At-risk Populations**

29 The public health implications of the evidence regarding O₃-related health effects, as for
30 other effects, are dependent on the type and severity of the effects, as well as the size of the
31 population affected. Such factors are discussed here in the context of our consideration of the

⁴⁰ Due to findings from controlled human exposure studies examining clinical endpoints (e.g., blood pressure) that do not indicate an O₃ effect and from epidemiologic studies examining cardiovascular-related hospital admissions and emergency department visits that do not find positive associations, a continuum of effects that could lead to cardiovascular mortality is not apparent (ISA, Appendices 4 and 6).

1 health effects evidence related to O₃ in ambient air. Additionally, we summarize the available
2 information related to judgments or interpretative statements developed by public health experts,
3 including particularly experts in respiratory health. This section also summarizes the current
4 information on population groups at increased risk of the effects of O₃ in ambient air.

5 With regard to O₃ in ambient air, the potential public health impacts relate most
6 importantly to the role of O₃ in eliciting respiratory effects, the category of effects that the ISA
7 concludes to be causally related to O₃ exposure. Controlled human exposure studies have
8 documented reduced lung function, respiratory symptoms, increased airway responsiveness, and
9 inflammation, among other effects, in largely healthy adults exposed while at elevated
10 ventilation, such as while exercising. Such effects, if of sufficient severity and in individuals
11 with compromised respiratory function, such as individuals with asthma, are plausibly related to
12 emergency department visits and hospital admissions for asthma which have been associated
13 with ambient air concentrations of O₃ in epidemiologic studies (as summarized in section 3.3.1
14 above; 2013 ISA, section 6.2.7; ISA, Appendix 3, sections 3.1.5.1 and 3.1.5.2).

15 The clinical significance of individual responses to O₃ exposure depends on the health
16 status of the individual, the magnitude of the changes in pulmonary function, the severity of
17 respiratory symptoms, and the duration of the response among other factors. While a particular
18 reduction in FEV₁ or increase in inflammation or airway responsiveness may not be of concern
19 for a healthy group,⁴¹ it may increase the risk of a more severe effect in a group with asthma. As
20 a more specific example, the same increase in inflammation or airway responsiveness in
21 individuals with asthma could predispose them to an asthma exacerbation event triggered by an
22 allergen to which they may be sensitized (e.g., ISA, Appendix 3, section 3.1.5.6.1; 2013 ISA,
23 sections 6.2.3 and 6.2.6). Duration and frequency of documented effects is also reasonably
24 expected to influence potential adversity and interference with normal activity. In summary,
25 consideration of differences in magnitude or severity, and also the relative transience or
26 persistence of the responses (e.g., FEV₁ changes) and respiratory symptoms, as well as pre-
27 existing sensitivity to effects on the respiratory system, and other factors, are important to
28 characterizing implications for public health effects of an air pollutant such as O₃ (ATS, 2000;
29 Thurston et al., 2017).

30 Decisions made in past reviews of the O₃ primary standard and associated judgments
31 regarding adversity or health significance of measurable physiological responses to air pollutants

⁴¹ For example, for most healthy individuals, moderate effects on pulmonary function, such as transient FEV₁ decrements smaller than 20% or transient respiratory symptoms, such as cough or discomfort on exercise or deep breath, would not be expected to interfere with normal activity, while larger pulmonary function effects (e.g., FEV₁ decrements of 20% or larger lasting longer than 24 hours) and/or more severe respiratory symptoms are more likely to interfere with normal activity for more of such individuals (e.g., 2014 PA, p. 3-53; 2006 AQCD, Table 8-2).

1 have been informed by guidance, criteria or interpretative statements developed within the public
2 health community, including the ATS, an organization of respiratory disease specialists, as well
3 as the CASAC. The ATS released its initial statement (titled *Guidelines as to What Constitutes*
4 *an Adverse Respiratory Health Effect, with Special Reference to Epidemiologic Studies of Air*
5 *Pollution*) in 1985 and updated it in 2000 (ATS, 1985; ATS, 2000). The ATS described its 2000
6 statement as being intended to “provide guidance to policy makers and others who interpret the
7 scientific evidence on the health effects of air pollution for the purposes of risk management”
8 (ATS, 2000). The statement further asserts that “principles to be used in weighing the evidence
9 and setting boundaries” and “the placement of dividing lines should be a societal judgment”
10 (ATS, 2000). The ATS explicitly states that it does “not attempt to provide an exact definition or
11 fixed list of health impacts that are, or are not, adverse,” providing instead “a number of
12 generalizable ‘considerations’” and that there “cannot be precise numerical criteria, as broad
13 clinical knowledge and scientific judgments, which can change over time, must be factors in
14 determining adversity” (ATS, 2000). A more recent ATS statement, while generally consistent
15 with the 2000 statement in the attention that statement gives to at-risk or vulnerable population
16 groups, broadens the discussion of effects, responses and biomarkers to reflect the expansion of
17 scientific research in these areas (Thurston, et al., 2017). The more recent statement additionally
18 notes that it does not offer “strict rules or numerical criteria, but rather proposes considerations to
19 be weighed in setting boundaries between adverse and nonadverse health effects,” providing a
20 general framework for interpreting evidence that proposes a “set of considerations that can be
21 applied in forming judgments” for this context (Thurston et al., 2017). Thus, the most recent
22 statement expands upon (with some specificity) and updates the prior statement by retaining
23 previously identified considerations, including, for example, its emphasis on consideration of
24 vulnerable populations, while retaining core consistency with the earlier ATS statement
25 (Thurston et al., 2017; ATS, 2000).

26 With regard to pulmonary function decrements, the earlier ATS statement concluded that
27 “small transient changes in forced expiratory volume in 1 s[econd] (FEV₁) alone were not
28 necessarily adverse in healthy individuals, but should be considered adverse when accompanied
29 by symptoms” (ATS, 2000). The more recent ATS statement continues to support this
30 conclusion and also gives weight to findings of such lung function changes in the absence of
31 respiratory symptoms in individuals with pre-existing compromised function, such as that
32 resulting from asthma (Thurston et al., 2017). More specifically, the recent ATS statement
33 expresses the view that when occurring in individuals with pre-existing compromised function,
34 such as asthma, the occurrence of “small lung function changes” “should be considered adverse
35 ... even without accompanying respiratory symptoms” (Thurston et al., 2017). In keeping with
36 the intent of these statements to avoid specific criteria, neither statement provides more specific

1 descriptions of such responses, such as with regard to magnitude, duration or frequency of small
2 pollutant-related lung function changes, for consideration of such conclusions. The earlier ATS
3 statement, in addition to emphasizing clinically relevant effects, also emphasized both the need
4 to consider changes in “the risk profile of the exposed population,” and effects on the portion of
5 the population that may have a diminished reserve that puts its members at potentially increased
6 risk if affected by another agent (ATS, 2000). In a similar vein, the more recent statement
7 emphasizes the distinction between population changes and individual changes in lung function
8 measures noting that for an exposed group of study subjects, while the mean change or reduction
9 may be small, some individual study group members will have larger reductions which in some
10 cases may have passed a threshold for clinical importance (Thurston et al., 2017). These
11 concepts, including the consideration of the magnitude of effects occurring in just a subset of
12 study subjects, continue to be recognized as important in the more recent ATS statement
13 (Thurston et al., 2017) and continue to be relevant to the evidence base for O₃.

14 • **Does the available evidence alter our prior understanding of populations that are**
15 **particularly at risk from O₃ exposures? What are important uncertainties in that**
16 **evidence?**

17 The newly available information regarding O₃ exposures and health effects among
18 sensitive populations, as thoroughly evaluated in the ISA, has not altered our understanding of
19 human populations at particular risk of health effects from O₃ exposures (ISA, section IS.4.4).
20 For example, the respiratory effects evidence, extending decades into the past and augmented by
21 new studies in this review, supports the conclusion that “individuals with pre-existing asthma are
22 at greater risk of ozone-related health effects based on the substantial and consistent evidence
23 within epidemiologic studies and the coherence with toxicological studies” (ISA, p. IS-57).
24 Numerous epidemiological studies document associations of O₃ with asthma exacerbation. Such
25 studies indicate the associations to be strongest for populations of children which is consistent
26 with their generally greater time outdoors while at elevated exertion. Together, these
27 considerations indicate people with asthma, including particularly children with asthma, to be at
28 relatively greater risk of O₃-related effects than other members of the general population (ISA,
29 sections IS.4.3.1 and IS.4.4.2, Appendix 3).⁴²

30 With respect to people with asthma, the limited evidence from controlled human
31 exposure studies (which are primarily in adult subjects) indicates similar magnitude of FEV₁
32 decrements as in people without asthma (ISA, Appendix 3, section 3.1.5.4.1). Across studies of
33 other respiratory effects of O₃ (e.g., increased respiratory symptoms, increased airway

⁴² Populations or lifestages can be at increased risk of an air pollutant-related health effect due to one or more factors. These factors can be intrinsic, such as physiological factors that may influence the internal dose or toxicity of a pollutant, or extrinsic, such as sociodemographic, or behavioral factors.

1 responsiveness and increased lung inflammation), the responses observed in study subjects
2 generally do not differ due to the presence of asthma, although the evidence base is more limited
3 with regard to study subjects with asthma (ISA, Appendix 3, section 3.1.5.7). However, the
4 features of asthma (e.g., increased airway responsiveness) contribute to a risk of asthma-related
5 responses, such as asthma exacerbation in response to asthma triggers, which may increase the
6 risk of more severe health outcomes (ISA, section 3.1.5). For example, a particularly strong and
7 consistent component of the epidemiologic evidence is the appreciable number of epidemiologic
8 studies that demonstrate associations between ambient air O₃ concentrations and hospital
9 admissions and emergency department visits for asthma (ISA, section IS.4.4.3.1).⁴³ We
10 additionally recognize that in these studies, the strongest associations (e.g., highest effect
11 estimates) or associations more likely to be statistically significant are those for childhood age
12 groups, which are, as recognized in section 3.4, the age groups most likely to spend time
13 outdoors during afternoon periods (when O₃ may be highest) and at activity levels corresponding
14 to those that have been associated with respiratory effects in the human exposure studies (ISA,
15 Appendix 3, sections 3.1.4.1 and 3.1.4.2).⁴⁴ The epidemiologic studies of hospital admissions
16 and emergency department visits are augmented by a large body of individual-level
17 epidemiologic panel studies that demonstrated associations of short-term ozone concentrations
18 with respiratory symptoms in children with asthma. Additional support comes from
19 epidemiologic studies that observed O₃-associated increases in indicators of airway inflammation
20 and oxidative stress in children with asthma (ISA, section IS.4.3.1). Together, this evidence
21 continues to indicate the increased risk of population groups with asthma (ISA, Appendix 3,
22 section 3.1.5.7).

23 Children and outdoor adult workers, are at increased risk largely due to their generally
24 greater time spent outdoors while at elevated exertion rates (including in summer afternoons and

⁴³ In addition to asthma exacerbation, the epidemiologic evidence also includes findings of positive associations of increased O₃ concentrations with hospital admissions or emergency department visits for COPD exacerbation and other respiratory diseases (ISA, Appendix 3, sections 3.1.6.1.3 and 3.1.8).

⁴⁴ Evaluations of activity pattern data indicate children to more frequently spend time outdoors during afternoon and early evening hours, while at moderate or greater exertion level, than other age groups (Appendix 3D, section 3D.2.5.3, including Figure 3D-9; 2014 HREA, section 5.4.1.5 and Appendix 5G, section 5G-1.4). For example, for days with some time spent outdoors, children spend, on average, approximately 2¼ hours of afternoon time outdoors, 80% of which is at a moderate or greater exertion level, regardless of their asthma status (Appendix 3D, section 3D.2.5.3). Adults, for days having some time spent outdoors, also spend approximately 2¼ hours of afternoon time outdoors regardless of their asthma status but the percent of afternoon time at moderate or greater exertion levels for adults (about 55%) is lower than that observed for children. Such analyses also note greater participation in outdoor events during the afternoon, compared to other times of day, for children ages 6 through 19 years old during the warm season months (ISA, Appendix 2, section 2.4.1, Table 2-1). Analyses of the limited activity pattern data by health status do not indicate asthma status to have appreciable impact (Appendix 3D, section 3D.2.5.3; 2014 HREA, section 5.4.1.5).

1 early evenings when O₃ levels may be higher).⁴⁵ This behavior makes them more likely to be
2 exposed to O₃ in ambient air under conditions contributing to increased dose, e.g., elevated
3 ventilation taking greater air volumes into the lungs⁴⁶ (ISA, section IS.4.4.2; 2013 ISA, section
4 5.2.2.7). Thus, in light of the evidence summarized in the prior paragraphs, children and outdoor
5 workers with asthma may be at increased risk of more severe outcomes, such as asthma
6 exacerbation. Further, with regard to children, there is experimental evidence from early life
7 exposures of nonhuman primates that indicates the potential for effects in childhood (through
8 adolescence) when human respiratory systems are under development (ISA, sections IS.4.4.2 and
9 IS.4.4.4.1). As noted in the ISA, “these experimental studies indicate that early-life ozone
10 exposure can cause structural and functional changes that could potentially contribute to airway
11 obstruction and increased airway responsiveness” (ISA, p. IS-52). Overall, the available
12 evidence, while not increasing our knowledge about susceptibility or at-risk status of these
13 population groups, is consistent with that in the 2015 review (ISA, section IS.4.4).

14 Evidence available in the 2020 ISA for older adults, a population identified as at risk in
15 the 2015 review, adds little to the evidence previously available (ISA, sections IS.4.4.2 and
16 IS.4.4.4.2; Table IS-10). The ISA notes, however, that “[t]he majority of evidence for older
17 adults being at increased risk of health effects related to ozone exposure comes from studies of
18 short-term ozone exposure and mortality evaluated in the 2013 Ozone ISA” (ISA, p. IS-52).
19 Such studies are part of the larger evidence base that is now concluded to be suggestive, but not
20 sufficient to infer a causal relationship of O₃ with mortality (ISA, sections IS.4.3.5 and
21 IS.4.4.4.2, Appendix 4, section 4.1.16.1 and 4.1.17).

22 The ISA also expressly considered the evidence regarding O₃ exposure and health effects
23 among populations with several other potential risk factors. As in the 2015 review, there is
24 suggestive evidence of low socioeconomic status (SES) as a factor associated with potentially
25 increased risk of O₃-related health effects (2013 ISA, section 8.3.3 and p. 8-37; ISA, section
26 IS.4.4). The 2013 ISA concluded that “[o]verall, evidence is suggestive of SES as a factor
27 affecting risk of O₃-related health outcomes based on collective evidence from epidemiologic
28 studies of respiratory hospital admissions but inconsistency among epidemiologic studies of

⁴⁵ More specifically regarding outdoor workers, in 2020 about 4% of civilian workers were required to spend more than two-thirds of their workday outdoors. Among construction, landscaping and groundskeeping workers, about 80-90% were required to spend more than two-thirds of their working day outside. Other employment sectors, including highway maintenance, protection services, extraction and other construction trades like engineers and equipment operators also had a high percentage of employees who spent most of their workday outdoors (Bureau of Labor Statistics, 2020). Such jobs often include physically demanding tasks and involve increased ventilation rates, increasing the potential for exposure to O₃.

⁴⁶ Additionally, compared to adults, children have higher ventilation rates relative to their lung volume which tends to increase the dose normalized to lung surface area (ISA, p. IS-60).

1 mortality and reproductive outcomes,” additionally stating that “[f]urther studies are needed to
2 confirm this relationship, especially in populations within the U.S.” (2013 ISA, p. 8-28). The
3 evidence in the 2020 ISA adds little to the evidence previously available in this area (ISA,
4 section IS.4.4.2 and Table IS-10). Regarding populations identified by race or ethnicity,
5 including American Indians or Native Americans, the evidence continued to be inadequate to
6 make a determination regarding a potential for increased risk (ISA, section IS.4.4, Table IS-10).

7 The ISA in the 2015 review additionally identified a role for dietary anti-oxidants such as
8 vitamins C and E in influencing risk of O₃-related effects, such as inflammation, as well as a role
9 for genetic factors to also confer either an increased or decreased risk (2013 ISA, sections 8.1
10 and 8.4.1). No recently available evidence was evaluated in the ISA that would inform or change
11 these prior conclusions (ISA, section IS.4.4 and Table IS-10).

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

14 The magnitude and characterization of a public health impact is dependent upon the size
15 and characteristics of the populations affected, as well as the type or severity of the effects. As
16 summarized above, children are an at-risk population and children under the age of 18 account
17 for 22.3% of the total U.S. population, with 6.0% of the total population being children under 5
18 years of age (U.S. Census Bureau, 2019). Further, as summarized above, a key population most
19 at risk of health effects associated with O₃ in ambient air is people with asthma. The National
20 Center for Health Statistics data for 2019 indicate that approximately 7.8% of the U.S.
21 population has asthma (Table 3-1; CDC, 2019). This is one of the principal populations that the
22 primary O₃ NAAQS is designed to protect (80 FR 65294, October 26, 2015). Table 3-1 below
23 considers the currently available information that helps to characterize key features of this
24 population.⁴⁷

25 The age group for which asthma prevalence documented by these data is greatest is
26 children aged five to 19, with 9.1% of children aged five to 14 and 7.4% of children aged 15-19
27 having asthma. In 2012 (the most recent year for which such an evaluation is available), asthma
28 was the leading chronic illness affecting children (Bloom et al., 2013). The prevalence is greater
29 for boys than girls (for those less than 18 years of age). Among populations of different races or
30 ethnicities, black non-Hispanic children have the highest prevalence, at 13.5%. Asthma
31 prevalence is also increased among populations in poverty. For example, 11.8% of people living
32 in households below the poverty level have asthma, compared to 7.2%, on average, of those

⁴⁷ Additionally, as part of the 2019 National Health Interview Survey, about 41% of people with asthma reported having had an asthma attack or asthma episode within the prior 12 months, with this percentage being slightly greater among children with asthma (44%) compared to adults with asthma (40%). A summary is available in Tables 5-1 and 6-1 of the survey (https://www.cdc.gov/asthma/most_recent_national_asthma_data.htm).

1 living above it. Populations groups with relatively greater asthma prevalence, such as
 2 populations in poverty and children, might be expected to have a relatively greater potential for
 3 O₃-related health impacts.⁴⁸

4
 5 **Table 3-1. National prevalence of asthma, 2019.**

Characteristic ^A	Number with Current Asthma (in thousands) ^B	Percent with Current Asthma
Total	25,131	7.8
Child (Age <18)	5,104	7.0
Adult (Age 18+)	20,026	8.0
All Age Groups		
0-4 years	517	2.6
5-14 years	3,725	9.1
15-19 years	1,529	7.4
20-24 years	2,092	9.9
25-34 years	3,574	8.0
35-64 years	9,594	7.8
65+ years	4,069	7.7
Child Age Group		
0-4 years	517	2.6
5-11 years	2,345	8.3
12-17 years	2,241	8.9
12-14 years	1,379	10.8
15-17 years	861	7.0
Sex		
Males	10,487	6.6
Boys (Age <18)	3,122	8.4
Men (Age 18+)	7,364	6.1
Females	14,643	8.9
Girls (Age <18)	1,981	5.5
Women (Age 18+)	12,662	9.8
Race/Ethnicity		
White NH ^C	15,094	7.7
Child (Age <18)	2,385	6.4
Adult (Age 18+)	12,701	8.1
Black NH	4,105	10.6
Child (Age <18)	1,289	13.5
Adult (Age 18+)	2,814	9.7
AI/AN ^E NH	349	10.7
Child (Age <18)	67	8.2

⁴⁸ As summarized in section 3.1 above, the current standard was set to protect at-risk populations, which include people with asthma. Accordingly, populations with asthma living in areas not meeting the standard would be expected to be at increased risk of effects.

Characteristic ^A	Number with Current Asthma (in thousands) ^B	Percent with Current Asthma
Adult (Age 18+)	281	11.6
Asian NH	697	3.8
Child (Age <18)	130	3.7
Adult (Age 18+)	567	3.8
Multiple ^D NH	867	12.6
Child (Age <18)	339	11.2
Adult (Age 18+)	527	13.7
Hispanic, all	3,874	6.6
Child (Age <18)	1,387	7.5
Adult (Age 18+)	2,486	6.1
Hispanic, Mexican ^F	1,933	5.3
Child (Age<18)	725	6.1
Adult (Age 18+)	1,207	5.0
Hispanic, Other ^F	1,929	8.5
Child (Age<18)	656	10.0
Adult (Age 18+)	1,273	7.9
Federal Poverty Threshold		
Below 100% of poverty level	4,814	11.8
100% to less than 250% of poverty level	7,837	8.5
250% to less than 450% of poverty level	6,345	7.3
450% of poverty level or higher	6,138	5.9
^A Numbers within selected characteristics may not sum to total due to rounding ^B Includes persons who answered "yes" to the questions "Have you EVER been told by a doctor or other health professional that you had asthma" and "Do you still have asthma?" ^C NH = non-Hispanic ^D Subcategory includes 'Other single and multiple races' for 2019 ^E AI/AN = American Indian/ Alaska Native ^F As a subset of Hispanic Adapted from 2019 National Health Interview Survey, Tables 3-1 and 4-1 (https://www.cdc.gov/asthma/most_recent_national_asthma_data.htm).		

1

2 **3.3.3 Exposure Concentrations Associated with Effects**

3 The extensive evidence base for O₃ health effects, compiled over several decades and

4 evaluated in the ISA, continues to indicate respiratory responses to short-term exposures as the

5 most sensitive effects. As at the time of the 2015 review, the EPA’s conclusions regarding

6 exposure concentrations of O₃ associated with respiratory effects reflect the extensive

7 longstanding evidence base of controlled human exposure studies of short-term O₃ exposures of

8 people with and without asthma.⁴⁹ These studies have documented an array of respiratory effects,

9 including reduced lung function, respiratory symptoms, increased airway responsiveness, and

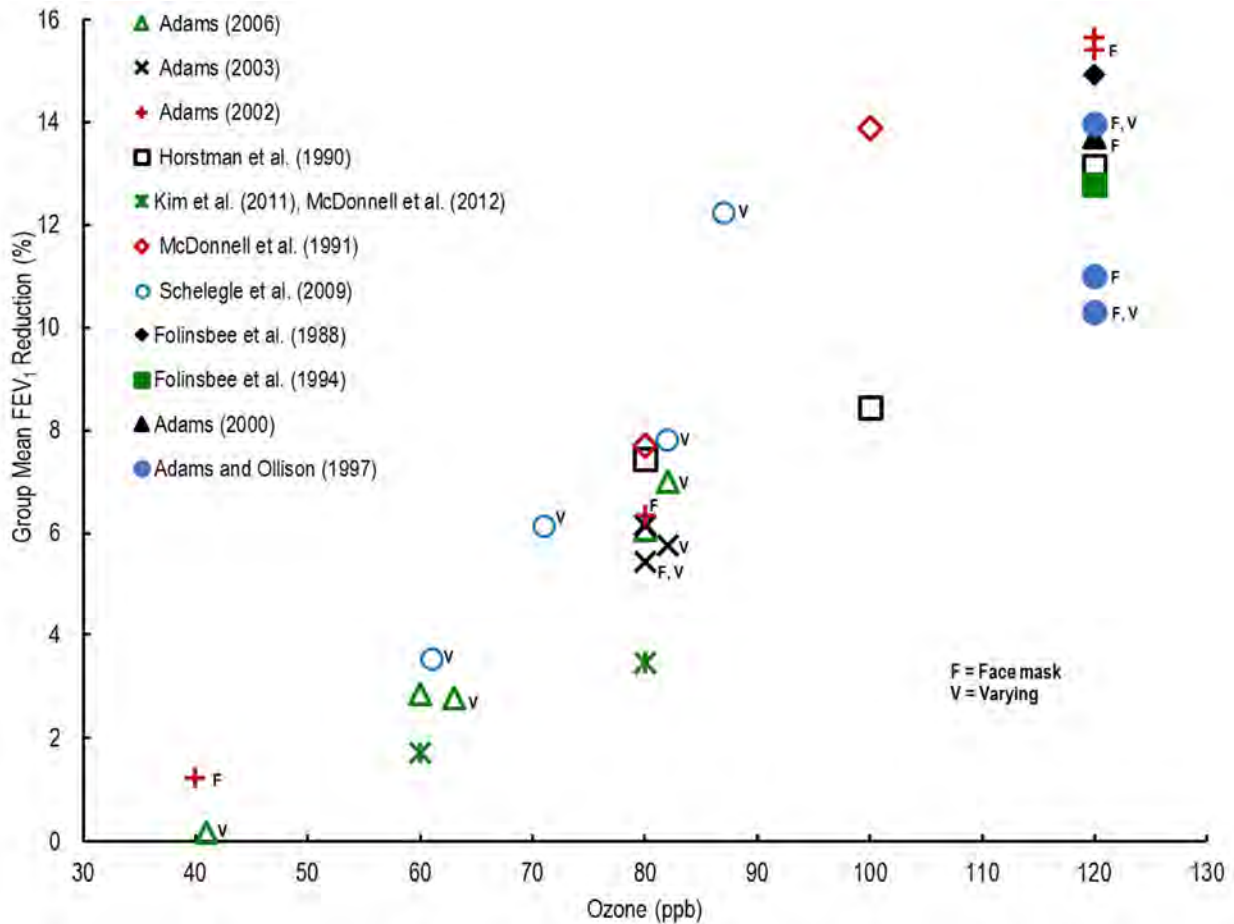
⁴⁹ As recognized elsewhere, the studies are largely conducted with adult subjects.

1 inflammation, in study subjects following 1- to 8-hour exposures, primarily while exercising.
2 The severity of observed responses, the percentage of individuals responding, and strength of
3 statistical significance at the study group level have been found to increase with increasing
4 exposure (ISA; 2013 ISA; 2006 AQCD). Factors influencing exposure include activity level or
5 ventilation rate, exposure concentration, and exposure duration (ISA; 2013 ISA; 2006 AQCD).
6 For example, evidence from studies with similar duration and exercise aspects (6.6-hour duration
7 with six 50-minute exercise periods) demonstrates an exposure-response relationship for O₃-
8 induced reduction in lung function (Figure 3-2).^{50,51} This specific evidence was integral to the
9 Administrator's judgments and decisions in 2015 and 2020 (80 FR 65292, October 26, 2015; 85

⁵⁰ For a subset of the studies included in Figure 3-2 (those with face mask rather than chamber exposures), there is no O₃ exposure during some of the 6.6-hr experiment (e.g., during the lunch break). Thus, while the exposure concentration during the exercise periods is the same for the two types of studies, the time-weighted average (TWA) concentration across the full 6.6-hr period differs slightly. For example, in the facemask studies of 120 ppb, the TWA across the full 6.6-hour experiment is 109 ppb (Appendix 3A, Table 3A-2).

⁵¹ The relationship also exists for size of FEV₁ decrement with alternative exposure or dose metrics, including total inhaled O₃ and intake volume averaged concentration.

2



3

4

5 **Figure 3-2. Group mean O₃-induced reduction in FEV₁ from controlled human exposure**
 6 **studies of healthy adults exposed for 6.6 hours with quasi-continuous exercise.**
 7 FEV₁ values plotted reflect group mean O₃-induced percent change in FEV₁, based
 8 on subtraction of the group mean filtered air percent change (post-pre exposure)
 9 from the group mean O₃ percent change in FEV₁ (adapted from Appendix 3A; ISA,
 10 Appendix 3, Figure 3-1). Concentrations are the time-weighted averages of target
 11 concentrations across full 6.6-hour period in chamber studies (or the average of
 12 target concentrations across the six exposures in face mask studies).

13

- 14 • **Does the available evidence alter prior conclusions regarding the exposure duration**
 15 **and concentrations associated with health effects? Does the available scientific**
 16 **evidence indicate health effects attributable to exposures to O₃ concentrations lower**
 17 **than previously reported?**

18 The available evidence, as documented in the ISA, including that newly available in the
 19 2020 review, does not alter our conclusions from the 2015 review on exposure duration and

1 concentrations associated with O₃-related health effects. These conclusions were largely based
2 on the body of evidence from the controlled human exposure studies. A limited number of newly
3 available controlled human exposure studies are described in the ISA, although none involve
4 lower exposure concentrations than those previously studied (e.g., Figure 3-2) or find effects not
5 previously reported (ISA, Appendix 3, section 3.1.4).⁵²

6 The extensive evidence base for O₃ health effects, compiled over several decades,
7 continues to indicate respiratory responses to short-term exposures as the most sensitive effects
8 of O₃. As summarized in section 3.3.1.1 above, an array of respiratory effects is well documented
9 in controlled human exposure studies of subjects exposed for 1 to 8 hours, primarily while
10 exercising. The risk of more severe health outcomes associated with such effects is increased in
11 people with asthma as illustrated by the epidemiological findings of positive associations
12 between O₃ exposure and asthma-related emergency department visits and hospital admissions.

13 The magnitude of respiratory response (e.g., size of lung function decrements and
14 magnitude of symptom scores) documented in the controlled human exposure studies is
15 influenced by ventilation rate, exposure duration, and exposure concentration. When performing
16 physical activities requiring elevated exertion, ventilation rate is increased, leading to greater
17 potential for health effects due to an increased internal dose (2013 ISA, section 6.2.1.1, pp. 6-5 to
18 6-11). Accordingly, the exposure concentrations eliciting a given level of response after a given
19 exposure duration is lower for subjects exposed while at elevated ventilation, such as while
20 exercising (2013 ISA, pp. 6-5 to 6-6). For example, in studies of generally healthy young adults
21 exposed while at rest for 2 hours, 500 ppb is the lowest concentration eliciting a statistically
22 significant O₃-induced group mean lung function decrement, while a 1- to 2-hour exposure to
23 120 ppb produces a statistically significant response in lung function when the ventilation rate of
24 the group of study subjects is sufficiently increased with exercise (2013 ISA, pp. 6-5 to 6-6).

25 The exposure conditions (e.g., duration and exercise) given primary focus in the past
26 several reviews are those of the 6.6-hour study design, which involves six 50-minute exercise
27 periods during which subjects maintain a moderate level of exertion to achieve a ventilation rate
28 of approximately 20 L/min per m² body surface area while exercising. The 6.6 hours of exposure
29 in these studies has generally occurred in an enclosed chamber and the study design includes
30 three hours in each of which is a 50-minute exercise period and a 10-minute rest period, followed
31 by a 35-minute lunch (rest) period, which is followed by three more hours of exercise and rest, as

⁵² No 6.6-hour studies are newly available (ISA, Appendix 3, section 3.1.4.1.1). The newly available studies are generally for exposures of three hours or less, and in nearly all instances involve exposure (while at elevated exertion) to concentrations above 100 ppb (ISA, Appendix 3, section 3.1.4).

1 before lunch.⁵³ Most of these studies performed to date involve exposure maintained at a
2 constant (unchanging) concentration for the full duration, although a subset of studies have
3 concentrations that vary (generally in a stepwise manner) across the exposure period and are
4 selected so as to achieve a specific target concentration as the exposure average (Appendix 3A,
5 Table 3A-2).⁵⁴

6 No studies of the 6.6-hour quasi-continuous exercise design are newly available since the
7 2015 review. The previously available studies of this design document statistically significant
8 O₃-induced reduction in lung function (FEV₁) and increased pulmonary inflammation in young
9 healthy adults exposed to O₃ concentrations as low as 60 ppb. Statistically significant group
10 mean changes in FEV₁, also often accompanied by statistically significant increases in
11 respiratory symptoms, become more consistent across such studies of exposures to higher O₃
12 concentrations, such as 70 ppb and 80 ppb (Table 3-2; Appendix 3A, Table 3A-1). The lowest
13 exposures concentration for which these studies document a statistically significant increase in
14 respiratory symptoms is somewhat above 70 ppb, at 73 ppb⁵⁵ (Schelegle et al., 2009; Appendix
15 3A, Table 3A-1). In the 6.6-hour studies, the group means of O₃-induced⁵⁶ FEV₁ reductions for
16 target exposure concentrations at or below 70 ppb are approximately 6% or lower (Figure 3-2,
17 Table 3-2). For example, the group means of O₃-induced FEV₁ decrements reported in these
18 studies that are statistically significantly different from the responses in filtered air are 6.1% for
19 the 70 ppb target (73 ppb time weighted average based on measurements) and 1.7% to 3.5% for
20 the 60 ppb target (Figure 3-2, Table 3-2).

21 The group mean O₃-induced FEV₁ decrements generally increase with increasing O₃
22 exposures, reflecting increases in both the number of the individuals affected and the magnitude

⁵³ A few studies have involved exposures by facemask rather than in a chamber. To date, there is little research differentiating between exposures conducted with a facemask and in a chamber since the pulmonary responses of interest do not seem to be influenced by the exposure mechanism. However, similar responses have been seen in studies using both exposure methods at higher O₃ concentrations (Adams, 2002; Adams, 2003). In the facemask designs, there is a short period of zero exposure, such that the total period of exposure is closer to 6 hours than 6.6 (Adams, 2000; Adams, 2002; Adams, 2003).

⁵⁴ In these studies, the exposure concentration changes for each of the six hours in which there is exercise and the concentration during the 35-minute lunch is the same as in the prior (third) hour with exercise. For example, in the study by Adams (2006b), the protocol for the 6.6-hour period is as follows: 60 minutes at 0.04 ppm, 60 minutes at 0.07 ppm, 95 minutes at 0.09 ppm, 60 minutes at 0.07 ppm, 60 minutes at 0.05 ppm and 60 minutes at 0.04 ppm.

⁵⁵ Measurements are reported in this study for each of the six 50-minute exercise periods, for which the mean is 72 ppb (Schelegle et al., 2009). Based on these data, the time-weighted average concentration across the full 6.6-hour duration was 73 ppb (Schelegle et al., 2009). The study design includes a 35-minute lunch period following the third exposure hour during which the exposure concentration remains the same as in the third hour.

⁵⁶ Consistent with the ISA and 2013 ISA, the phrase “O₃-induced” decrement or reduction in lung function or FEV₁ refers to the percent change from pre-exposure measurement of the O₃ exposure minus the percent change from pre-exposure measurement of the filtered air exposure (2013 ISA, p. 6-4).

1 of the FEV₁ reduction (Figure 3-2). For example, following 6.6-hour exposures to a lower
2 concentration (40 ppb), for which decrements were not statistically significant at the group mean
3 level, none of 60 subjects across two separate studies experienced an O₃-induced FEV₁ reduction
4 as large as 15% or more (Appendix 3D, Table 3D-19). Across the four experiments (with
5 number of subjects ranging from 30 to 59 subjects) that have reported results for 60 ppb target
6 exposure,⁵⁷ the number of subjects experiencing this magnitude of FEV₁ reduction (at or above
7 15%) varied (zero of 30, one of 59, two of 31 and two of 30 exposed subjects), while, together,
8 they represent 3% of all 150 subjects. The percentage of subjects (with reductions of 15% or
9 more) increased to 10% (three of 31 subjects) for the study at 73 ppb (70 ppb target
10 concentration) and is higher still (16%) in a variable exposure study at 80 ppb (Appendix 3D,
11 Tables 3D-19 and 3D-30; Schelegle et al., 2009). In addition to illustrating the E-R relationship,
12 these findings also illustrate the considerable variability in magnitude of responses observed
13 among study subjects (Table 3-2, Figure 3-2; ISA, Appendix 3, section 3.1.4.1.1; 2013 ISA, p. 6-
14 13).
15

⁵⁷ For these four experiments, the average concentration across the 6.6-hour period ranged from 60 to 63 ppb (Appendix 3A, Table 3A-2).

1
2

Table 3-2. Summary of 6.6-hour controlled human exposure study-findings, healthy adults.

Endpoint	O ₃ Target Exposure Concentration ^A	Statistically Significant Effect ^B	O ₃ -Induced Group Mean Response ^B	Study
FEV ₁ Reduction	120 ppb	Yes	-10.3% to -15.9% ^C	Horstman et al. 1990; Adams 2002; Folinsbee et al. (1988); Folinsbee et al. (1994); Adams, 2002; Adams 2000; Adams and Ollison 1997 ^D
	100 ppb	Yes	-8.5% to -13.9% ^C	Horstman et al., 1990; McDonnell et al., 1991 ^D
	87 ppb	Yes	-12.2%	Schelegle et al., 2009
	80 ppb	Yes	-7.5%	Horstman et al., 1990
			-7.7%	McDonnell et al., 1991
			-6.5%	Adams, 2002
			-6.2% to -5.5% ^C	Adams, 2003
			-7.0% to -6.1% ^C	Adams, 2006b
			-7.8%	Schelegle et al., 2009
		ND ^E	-3.5%	Kim et al., 2011 ^F
	70 ppb	Yes	-6.1%	Schelegle et al., 2009
	60 ppb	Yes ^G	-2.9%	Adams, 2006b; Brown et al., 2008
			-2.8%	
Yes		-1.7%	Kim et al., 2011	
40 ppb	No	-3.5%	Schelegle et al., 2009	
	No	-1.2%	Adams, 2002	
	No	-0.2%	Adams, 2006b	
Increased Respiratory Symptoms	120 ppb	Yes	Increased symptom scores	Horstman et al. 1990; Adams 2002; Folinsbee et al. 1988; Folinsbee et al. 1994; Adams, 2002; Adams 2000; Adams and Ollison 1997; Horstman et al., 1990; McDonnell et al., 1991; Schelegle et al., 2009; Adams, 2003; Adams, 2006b ^H
	100 ppb	Yes		
	87 ppb	Yes		
	80 ppb	Yes		
	70 ppb	Yes		
	60 ppb	No		
	40 ppb	No		
Airway Inflammation	80 ppb	Yes	Multiple indicators ^I	Devlin et al., 1991; Alexis et al., 2010
	60 ppb	Yes	Increased neutrophils	Kim et al., 2011
Increased Airway Resistance and Responsiveness	120 ppb	Yes	Increased	Horstman et al., 1990; Folinsbee et al., 1994 (O ₃ induced sRaw not reported)
	100 ppb	Yes		Horstman et al., 1990
	80 ppb	Yes		Horstman et al., 1990

^A This refers to the average concentration across the six exercise periods as targeted by authors. This differs from the time-weighted average concentration for the full exposure periods (targeted or actual). For example, as shown in Appendix 3A, Table 3A-2, in chamber studies implementing a varying concentration protocol with targets of 0.03, 0.07, 0.10, 0.15, 0.08 and 0.05 ppm, the exercise period average concentration is 0.08 ppm while the time weighted average for the full exposure period (based on targets) is 0.082 ppm due to the 0.6 hour lunchtime exposure between periods 3 and 4. In some cases this also differs from the exposure period average based on study measurements. For example, based on measurements reported in Schelegle et

al., (2009), the full exposure period average concentration for the 70 ppb target exposure is 73 ppb, and the average concentration during exercise is 72 ppb.

^B Statistical significance based on the O₃ compared to filtered air response at the study group mean (rounded here to decimal).

^C Ranges reflect the minimum to maximum FEV₁ decrements across multiple exposure designs and studies. Study-specific values and exposure details provided in the PA, Appendix 3A, Tables 3A-1 and 3A-2, respectively.

^D Citations for specific FEV₁ findings for exposures above 70 ppb are provided in PA, Appendix 3A, Table 3A-1.

^E ND (not determined) indicates these data have not been subjected to statistical testing.

^F The data for 30 subjects exposed to 80 ppb by Kim et al. (2011) are presented in Figure 5 of McDonnell et al. (2012).

^G Adams (2006) reported FEV₁ data for 60 ppb exposure by both constant and varying concentration designs. Subsequent analysis of the FEV₁ data from the former found the group mean O₃ response to be statistically significant ($p < 0.002$) (Brown et al., 2008; 2013 ISA, section 6.2.1.1). The varying-concentration design data were not analyzed by Brown et al., 2008.

^H Citations for study-specific respiratory symptoms findings are provided in the PA, Appendix 3A, Table 3A-1.

^I Increased numbers of bronchoalveolar neutrophils, permeability of respiratory tract epithelial lining, cell damage, production of proinflammatory cytokines and prostaglandins (ISA, Appendix 3, section 3.1.4.4.1; 2013 ISA, section 6.2.3.1).

1
2 For shorter exposure periods (e.g., from one to two hours), with heavy intermittent or
3 very heavy continuous exercise, higher exposure concentrations, ranging from 80 ppb to 400
4 ppb, have been studied (ISA, Appendix 3A, section 3.1, Table 3A-3; 2013 ISA, section 6.2.1.1;
5 2006 AQCD, Chapter 6). Across these shorter-duration studies (which involved ventilation rates
6 2-3 times greater than in the prolonged [6.6- or 8-hour] exposure studies),⁵⁸ the lowest exposure
7 concentration for which statistically significant respiratory effects were reported is 120 ppb, for a
8 1-hour exposure combined with continuous very heavy exercise and a 2-hour exposure with
9 intermittent heavy exercise. As recognized above the increased ventilation rate associated with
10 increased exertion increases the amount of O₃ entering the lung, where depending on dose and
11 the individual's susceptibility, it may cause respiratory effects (2013 ISA, section 6.2.1.1). Thus,
12 for exposures involving a lower exertion level, a comparable response would not be expected to
13 occur without a longer duration at this concentration (120 ppb), as is illustrated by the 6.6-hour
14 study results for this concentration (Appendix 3A, Table 3A-1).

15 With regard to epidemiologic studies reporting positive associations between O₃ exposure
16 concentrations and respiratory health outcomes such as asthma-related emergency department
17 visits and hospitalizations, these studies are generally primarily focused on investigating the
18 existence of a relationship between O₃ occurring in ambient air and specific health outcomes,
19 (*versus* detailing the specific exposure circumstances eliciting such effects). Accordingly, while
20 as a whole, this evidence base of epidemiologic studies provides strong support for the
21 conclusions of causality as summarized in section 3.3.1 above,⁵⁹ these studies provide less
22 information on details of the specific O₃ exposure circumstances that may be eliciting health
23 effects associated with such outcomes, and whether these occur under air quality conditions that

⁵⁸ A quasi-continuous exercise protocol is common to the prolonged exposure studies where study subjects complete six 50-minute periods of exercise, each followed by 10-minute periods of rest (2013 ISA, section 6.2.1.1).

⁵⁹ Combined with the coherent evidence from experimental studies, the epidemiologic studies “can support and strengthen determinations of the causal nature of the relationship between health effects and exposure to ozone at relevant ambient air concentrations” (ISA, p. ES-17).

1 meet the current standard.⁶⁰ For example, these studies generally do not measure personal
2 exposures of the study population or track individuals in the population with a defined exposure
3 to O₃ alone. Further, the vast majority of these studies were conducted in locations and during
4 time periods that would not have met the current standard. The extent to which reported
5 associations with health outcomes in the resident populations in these studies are influenced by
6 the periods of higher concentrations during times that did not meet the current standard is
7 unknown. While this does not lessen their importance in the evidence base documenting the
8 causal relationship between O₃ and respiratory effects, it means they are less informative in
9 considering O₃ exposure concentrations occurring under air quality conditions allowed by the
10 current standard. Notwithstanding this, we have considered the epidemiologic studies identified
11 in the ISA as to what they might indicate regarding O₃ exposure concentrations in this regard.

12 Consistent with the evaluation of the epidemiologic evidence of associations between O₃
13 exposure and respiratory health effects in the ISA, we focus on those studies conducted in the
14 U.S. and Canada as including populations and air quality characteristics that may be most
15 relevant to circumstances in the U.S. (ISA, Appendix 3, section 3.1.2). Among the epidemiologic
16 studies finding a statistically significant positive relationship of short- or long-term O₃
17 concentrations with respiratory effects, there are no single-city studies conducted in the U.S. in
18 locations with ambient air O₃ concentrations that would have met the current standard for the
19 entire duration of the study (see Appendix 3B, Table 3B-1; ISA, Appendix 3, Tables 3-13, 3-14,
20 3-39, 3-41, 3-42 and Appendix 6, Tables 6-5 and 6-6;). There are (among this large group of
21 studies) two single city studies conducted in western Canada that include locations for which the
22 highest-monitor design values⁶¹ fell just below 70 ppb, at 65 and 69 ppb (Appendix 3B, Table
23 3B-1; Kousha and Rowe, 2014; Villeneuve et al., 2007). These studies did not, however, include
24 analysis of correlations with other co-occurring pollutants or of the strength of the associations
25 when accounting for effects of copollutants in copollutant models (ISA, Tables 3-14 and 3-39).
26 Thus, these studies pose significant limitations with regard to informing conclusions regarding
27 specific O₃ exposure concentrations and elicitation of such effects. There are also a handful of
28 multicity studies conducted in the U.S. or Canada in which the O₃ concentrations in a subset of
29 the study locations and for a portion of the study period appear to have met the current standard
30 (Appendix 3B). Concentrations in other portions of the study area or study period, however, do
31 not meet the standard, or data were not available in some cities for the earlier years of the study

⁶⁰ For example, these studies generally do not measure personal exposures of the study population or track individuals in the population with a defined exposure to O₃ alone.

⁶¹ As described in chapter 2, a design value is the metric used to describe air quality in a given area relative to the level of the standard, taking the averaging time and form into account. For example, a design value of 70 ppb just meets the current primary standard.

1 period when design values for other cities in the study were well above 70 ppb. The extent to
2 which reported associations with health outcomes in the resident populations in these studies are
3 influenced by the periods of higher concentrations during times that did not meet the current
4 standard is unknown. Additionally, with regard to multicity studies, the reported associations
5 were based on the combined dataset from all cities, complicating interpretations regarding the
6 contribution of concentrations in the small subset of locations that would have met the current
7 standard compared to that from the larger number of locations that would have violated the
8 standard (Appendix 3B, Table 3B-1 and Table 3B-2).⁶² Further, given that populations in such
9 studies may have also experienced longer-term, variable and uncharacterized exposure to O₃ (as
10 well as to other ambient air pollutants), “disentangling the effects of short-term ozone exposure
11 from those of long-term ozone exposure (and vice-versa) is an inherent uncertainty in the
12 evidence base” (ISA, p. IS-87 [section IS.6.1]). While given the depth and breadth of the
13 evidence base for O₃ respiratory effects, such uncertainties do not change our conclusions
14 regarding the causal relationship between O₃ and respiratory effects.

15 With regard to the experimental animal evidence (largely rodent studies) and exposure
16 conditions associated with respiratory effects, the exposure concentrations in the animal studies
17 are generally much greater than those examined in the controlled human exposure studies
18 (summarized above) and higher than concentrations commonly occurring in ambient air in areas
19 of the U.S. where the current standard is met. This is also true for the small number of early life
20 studies in nonhuman primates (recognized in section 3.3.1.1 above) that reported O₃ to contribute
21 to allergic asthma-like effects in infant primates.⁶³ The exposures eliciting the effects in these
22 studies included multiple 5-day periods with O₃ concentrations of 500 ppb over 8-hours per day,
23 exposure conditions appreciably greater than occur in areas of the U.S. where the current
24 standard is met (ISA, Appendix 3, section 3.2.4.1.2).

25 With regard to short-term O₃ and metabolic effects, the category of nonrespiratory effects
26 for which the ISA concludes there to be a likely causal relationship with O₃, the evidence base is
27 comprised primarily of experimental animal studies, as summarized in section 3.3.1.2 above
28 (ISA, Appendix 5, section 5.1). The exposure conditions from these studies, however, generally
29 involve much higher O₃ concentrations than those examined in the controlled human exposure
30 studies for respiratory effects (and much higher than concentrations occurring in ambient air in

⁶² As recognized in the 2015 review, “multicity studies do not provide a basis for considering the extent to which reported O₃ health effects associations are influenced by individual locations with ambient [air] O₃ concentrations low enough to meet the current O₃ standard versus locations with O₃ concentrations that violate this standard” (80 FR 64344, October 26, 2015).

⁶³ These studies indicate that sufficient early-life O₃ exposure can cause structural and functional changes that could potentially contribute to airway obstruction and increased airway responsiveness (ISA, Table IS-10, p. 3-92 and p.3-113).

1 areas of the U.S. where the current standard is met). For example, the animal studies include 4-
2 hour concentrations of 400 to 800 ppb (ISA, Appendix 5, Table 5-8).⁶⁴ The two epidemiologic
3 studies reporting statistically significant positive associations of O₃ with metabolic effects (e.g.,
4 changes in glucose, insulin, metabolic clearance) are based in Taiwan and South Korea,
5 respectively.⁶⁵ Given the potential for appreciable differences in air quality patterns between
6 Taiwan and South Korea and the U.S., as well as differences in other factors that might affect
7 exposure (e.g., activity patterns), those studies are of limited usefulness for informing our
8 understanding of exposure concentrations and conditions eliciting such effects in the U.S. (ISA,
9 Appendix 5, section 5.1).

10 Thus, as in the 2015 review, the exposure to which we give greatest attention, particularly
11 with regard to considering O₃ exposures expected under air quality conditions that meet the
12 current standard, are those informed by the controlled human exposure studies. The full body of
13 evidence described in the current ISA continues to indicate respiratory effects as the effects
14 associated with lowest exposures, with conditions of exposure (e.g., duration, ventilation rate,
15 and concentration) influencing dose and associated response. Evidence for other categories of
16 effects does not indicate effects at comparably low exposures.

17 **3.3.4 Uncertainties in the Health Effects Evidence**

- 18 • **To what extent have previously identified uncertainties in the health effects evidence**
19 **been reduced or do important uncertainties remain?**

20 We have not identified any new uncertainties in the evidence since the 2015 review.
21 However, we continue to recognize important uncertainties that also existed at that time. This
22 array of important areas of uncertainty relates to the available health evidence, including that
23 newly available in the 2020 review, and is summarized below.

24 Although the evidence clearly demonstrates that short-term O₃ exposures cause
25 respiratory effects, as was the case in the last review, we continue to recognize uncertainties that
26 remain in several aspects of our understanding of these effects. Such uncertainties include those
27 associated with the severity and prevalence of responses to short (e.g., 6.6- to 8-hour) O₃
28 exposures at and below 60 ppb and responses of some population groups not well represented in
29 the evidence base of controlled human exposure studies (e.g., children and people with asthma).

⁶⁴ The exposure concentration in the single controlled human exposure study of metabolic effects (e.g., 300 ppb) are also well above those examined in the respiratory effect studies (ISA, Appendix 5, Table 5-7).

⁶⁵ Of the five epidemiologic studies discussed in the ISA that investigate associations between short-term O₃ exposure and metabolic effects, three are conducted in Asia or South America and two are conducted in the U.S. The two U.S. studies report either a null or negative association of metabolic markers with O₃ concentration, and while the South American study (focused on hospital admissions associated with diabetes complications) reported positive associations with 24-hr average concentrations for some subgroups, no associations were statistically significant (ISA, Appendix 5, Tables 5-6 and 5-9).

1 There are also uncertainties concerning the potential influence of exposure history and co-
2 exposure to other pollutants on the relationship between short-term O₃ exposure and respiratory
3 effects. With regard to the full health effects evidence base, we also recognize as an important
4 uncertainty the extent to which O₃ exposures are related to health effects other than respiratory
5 effects. The following discussion touches on each of these types of uncertainty.

6 The majority of the available studies have generally involved healthy young adult
7 subjects, although there are some studies involving subjects with asthma, and a limited number
8 of studies, generally of very short durations (i.e., less than four hours), involving adolescents and
9 adults older than 50. While there is evidence from short (6.6- to 8-hour) controlled exposure
10 studies of healthy adult subjects to concentrations as low as 40 ppb, the only controlled human
11 exposure study of such a duration (7.6 hours with quasi-continuous light exercise) conducted in
12 people with asthma was for an exposure concentration of 160 ppb (Appendix 3A, Table 3A-2).
13 Given the paucity of studies using subjects that have asthma, particularly those at exposure
14 concentrations likely to occur under conditions meeting the current standard, uncertainties
15 remain with regard to characterizing the response in people with asthma while at elevated
16 ventilation to lower exposure concentrations, e.g., below 80 ppb. The extent to which the
17 epidemiologic evidence, including that recently available, can inform this area of uncertainty
18 also may be limited.⁶⁶ As discussed in section 3.3.2 above, given the effects of asthma on the
19 respiratory system, exposures associated with relatively mild respiratory responses in largely
20 healthy people may pose an increased risk of more severe responses, including asthma
21 exacerbation, in people with asthma. Such considerations remain areas of uncertainty at this
22 time. Thus, uncertainty remains with regard to the extent to which the controlled human
23 exposure study evidence describes the responses of the populations, such as children with
24 asthma, that may be most at risk of O₃-related respiratory effects (e.g., through an increased
25 likelihood of severe responses, or greatest likelihood of response).

26 Other areas of uncertainty concerning the potential influence of O₃ exposure history and
27 co-exposure to other pollutants on the relationship between short-term O₃ exposures and
28 respiratory effects also remain in the evidence base. As in the epidemiologic evidence in the
29 2015 review, there is a limited number of studies that include copollutant analyses for a small set
30 of pollutants (e.g., PM or NO₂). Recent studies with such analyses suggest that observed
31 associations between O₃ concentrations and respiratory effects are independent of co-exposures

⁶⁶ Associations of health effects with O₃ that are reported in the epidemiologic analyses are based on air quality concentration metrics used as surrogates for the actual pattern of O₃ exposures experienced by study population individuals over the period of a particular study. Therefore, the studies are limited in what they can convey regarding the specific patterns of exposure circumstances (e.g., magnitude of concentrations over specific duration and frequency) that might be eliciting reported health outcomes.

1 to correlated pollutants or aeroallergens (ISA, sections IS.4.3.1 and IS.6.1; Appendix 3, sections
2 3.1.10.1 and 3.1.10.2). Despite the increased prevalence of copollutant modeling in recent
3 epidemiologic studies, however, uncertainty still exists with regard to the independent effect of
4 O₃ given the high correlations observed for some copollutants in some studies and the small
5 fraction of all atmospheric pollutants included in these analyses (ISA, section IS.4.3.1; Appendix
6 2, section 2.5). We also note that neither of the two epidemiologic studies of respiratory
7 outcomes conducting in Canadian areas that would have met the current standard included
8 copollutant modeling (as recognized in section 3.3.3 above).

9 Further, although there remains uncertainty in the evidence with regard to the potential
10 role of exposures to O₃ in eliciting health effects other than respiratory effects, the evidence has
11 been strengthened since the 2015 review with regard to metabolic effects. As noted in section
12 3.3.1.2 above, the ISA newly identifies metabolic effects as likely to be causally related to short-
13 term O₃ exposures. The evidence supporting this relationship is limited and not without its own
14 uncertainties. For example, as noted in section 3.3.1.2 above, the conclusion is based primarily
15 on animal toxicological studies conducted at much higher O₃ concentrations than those common
16 in ambient air in the U.S. A limited number of epidemiologic studies of short-term O₃
17 concentrations and metabolic effects are available, many of which did not control for
18 copollutants confounding; just two studies, both in Asia, report significant positive associations
19 with changes in markers of glucose homeostasis (ISA, Appendix 5; sections 5.1.8 and 5.3).

20 Uncertainty is increased with regard to a relationship between O₃ exposure and
21 cardiovascular effects and mortality, as discussed in section 3.3.1.2 above, including regarding a
22 now-larger body of controlled human exposure studies providing evidence that is not consistent
23 with a cardiovascular effect in response to short-term O₃ exposure; and a paucity of
24 epidemiologic evidence indicating more severe cardiovascular morbidity endpoints, that would
25 be expected if the impaired vascular and cardiac function (observed in animal toxicological
26 studies) was the underlying basis for cardiovascular mortality (for which epidemiologic studies
27 have reported some positive associations with O₃). Additionally, uncertainties and limitations
28 recognized in the 2013 ISA (e.g., lack of control for potential confounding by copollutants in
29 epidemiologic studies) still remain (ISA, section IS.1.3.1). As discussed in section 3.3.1.2, these
30 uncertainties also pertain to conclusions regarding short-term O₃ and mortality (ISA, Appendix
31 6, section 6.1.8). Uncertainties are unchanged with regard to other nonrespiratory categories of
32 effects (described in section 3.3.1.2 above) for which the evidence is either suggestive of, but not
33 sufficient to infer, a causal relationship or is inadequate to determine if a causal relationship
34 exists with O₃ (ISA, section IS.4.3).

35 In summary, while there are some changes with regard to limitations and uncertainties of
36 the health effects evidence base, some key uncertainties associated with the evidence for

1 respiratory effects that were identified in the 2015 review remain, including those related to the
2 extent of effects at concentrations below those evaluated in controlled human exposure studies,
3 and the potential for more severe impacts in individuals with asthma, including particularly
4 children, and in other at-risk populations.

5 **3.4 EXPOSURE AND RISK INFORMATION**

6 Our consideration of the scientific evidence, as in each review of the O₃ NAAQS, is
7 informed by results from quantitative analyses of estimated population exposure and consequent
8 risk. Estimates from the exposure-based analyses, particularly the comparison of daily maximum
9 exposures to benchmark concentrations, were most informative to the Administrator’s decision
10 in the 2015 review (as summarized in section 3.1 above). This largely reflected the EPA
11 conclusion that “controlled human exposure studies provide the most certain evidence indicating
12 the occurrence of health effects in humans following specific O₃ exposures,” and recognition that
13 “effects reported in controlled human exposure studies are due solely to O₃ exposures, and
14 interpretation of study results is not complicated by the presence of co-occurring pollutants or
15 pollutant mixtures (as is the case in epidemiologic studies)” (80 FR 65343, October 26, 2015).⁶⁷
16 Therefore, the quantitative analyses developed in the 2020 review focused on exposure-based
17 risk analyses, in reflection of the emphasis given to these types of analyses and the
18 characterization of their uncertainties in the 2015 review, along with the availability of new or
19 updated information, models, and tools that address those uncertainties (IRP, Appendix 5A).

20 This reconsideration of the 2020 decision will rely on the exposure-based risk analyses
21 performed in the 2020 review, which were first presented in the 2020 PA and considered in the
22 2020 decision. These analyses are also presented here and described in detail in the associated
23 Appendices 3C and 3D. In section 3.4.1, we summarize the conceptual model for the assessment,
24 as well as key aspects of the assessment design, including the study areas, populations simulated,

⁶⁷ In the 2015 review, the Administrator placed relatively less weight on the air quality epidemiologic-based risk estimates, in recognition of an array of uncertainties, including, for example, those related to exposure measurement error (80 FR 65346, October 26, 2015). In so doing, she recognized key uncertainties in utilizing the estimated air concentrations and epidemiologic study relationships (often called epidemiologic-based risk estimates) (80 FR 65316; 79 FR 75277-75279; 2014 HREA, sections 3.2.3.2 and 9.6). These included the heterogeneity in effect estimates between locations, the potential for exposure measurement errors, and uncertainty in the interpretation of the shape of concentration-response functions at lower O₃ concentrations, as well as uncertainties related to the public health importance of increases in relatively low O₃ concentrations following air quality adjustment. Lower confidence was also placed in the results of the epidemiologic-based risk assessment of respiratory mortality risks associated with long-term O₃ exposures in consideration of several factors. Importantly since that time, the causal determinations for short-term O₃ exposure with mortality in the current ISA differ from the 2013 ISA. The current determinations for both short-term and long-term O₃ exposure (as summarized in section 3.1 above) are that the evidence is “suggestive” but not sufficient to infer causal relationships for O₃ with mortality (ISA, Table IS-1).

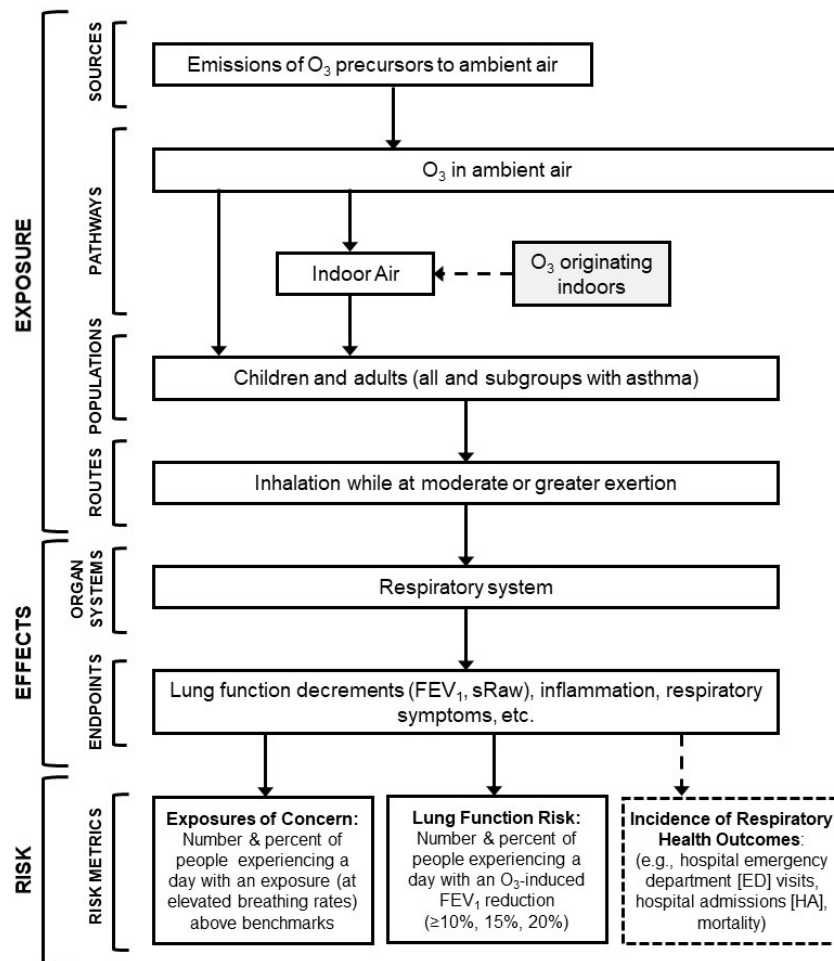
1 modeling tools, and exposure and risk metrics derived. Sections 3.4.2 and 3.4.3 summarize the
2 assessment results. Key limitations and uncertainties associated with the assessment estimates
3 are identified in section 3.4.4. and potential public health implications are discussed in section
4 3.4.5. An overarching consideration is whether the current exposure and risk information alters
5 overall conclusions reached in the 2015 review regarding the health risk associated with
6 exposure to O₃ in ambient air which formed an important foundation in the establishment at that
7 time of the existing standard.

8 **3.4.1 Conceptual Model and Assessment Approach**

9 The long-standing evidence base for O₃-related health effects is comprised of a large
10 assemblage of controlled human exposure studies, laboratory animal research studies, and air
11 quality epidemiologic studies. Together, these health effect studies lead to the strongly supported
12 conclusion that O₃ exposure causes respiratory effects (as summarized in section 3.3 above).
13 This conclusion is strongest with regard to short-term O₃ exposures, for which the ISA and
14 science assessments in prior reviews have determined there to be a causal relationship. The ISA
15 additionally determines the relationship between long-term exposure and respiratory effects, as
16 well as between short-term exposures and metabolic effects to be likely causal, recognizing that
17 associated uncertainties remain in the evidence. Given the relatively greater strength of the
18 evidence and understanding of the relevant exposure conditions, as well as availability of
19 appropriate data and modeling tools, the exposure and risk analysis is focused on respiratory
20 risks associated with short-term O₃ exposures.

21 The controlled human exposure studies document the occurrence of an array of
22 respiratory effects in humans in a variety of short-term exposure circumstances. These studies, in
23 combination with the laboratory animal studies, inform our understanding of the mode of action
24 for O₃-attributable effects, including those health outcomes associated with ambient air
25 concentrations in air quality epidemiologic studies (ISA, Appendix 3, section 3.1.3). Figure 3-3
26 below illustrates the conceptual model for O₃ in ambient air and respiratory effects, with a
27 particular focus on short-term exposures and including linkages with the risk metrics assessed in
28 the quantitative analyses described here.

29



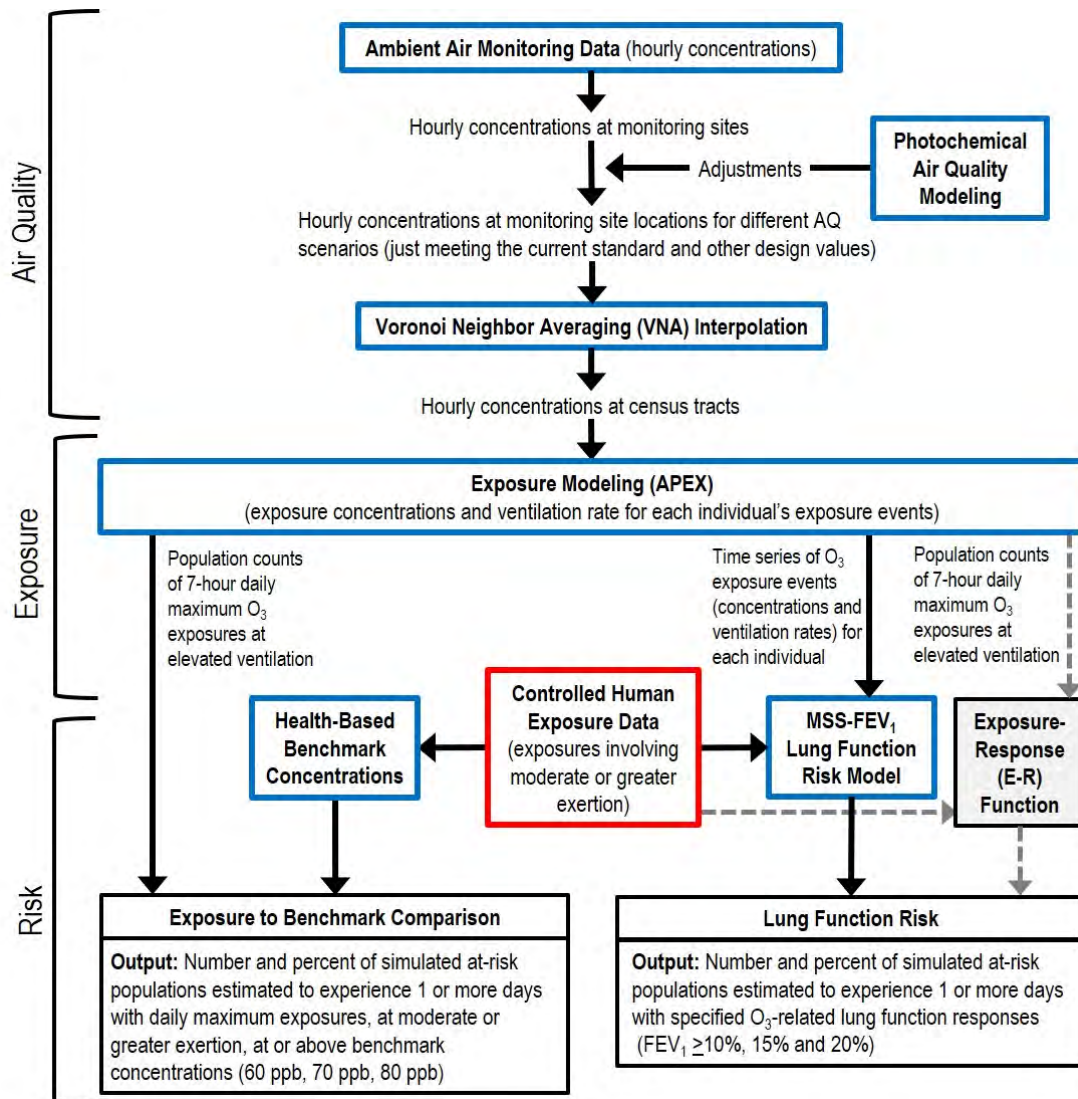
1
 2 **Figure 3-3. Conceptual model for exposure-based risk assessment.** Solid lines indicate
 3 processes explicitly modeled in the assessment. Dashed lines indicate relationships
 4 that are not explicitly modeled.

5 The exposure-based analyses, described in detail in Appendix 3D, were developed based
 6 on this conceptual model, in consideration of the information newly available in the 2020 review.
 7 In these analyses, we have estimated O₃ exposures and resulting risk for air quality conditions of
 8 interest, most particularly air quality conditions that just meet the current primary O₃ standard.
 9 These analyses inform our understanding of the protection provided by the current primary
 10 standard from effects that the health effects evidence indicates to be elicited in some portion of
 11 exercising people exposed for several hours to elevated O₃ concentrations.

12 The analysis approach employed is summarized in Figure 3-4 below and described in
 13 detail in Appendices 3C and 3D. This approach incorporates the use of an array of models and
 14 data to develop population exposure and risk estimates for a set of eight urban study areas.
 15 Ambient air O₃ concentrations were estimated in each study area using an approach that relies on
 16 a combination of ambient air monitoring data, atmospheric photochemical modeling and

1 statistical methods (described in detail in Appendix 3C). Population exposure and risk modeling
 2 is employed to characterize exposures and related lung function risk associated with the ambient
 3 air concentration estimates (described in detail in Appendix 3D). While the lung function risk
 4 analysis focuses only on the specific O₃ effect of FEV₁ reduction, the comparison-to-benchmark
 5 approach, with its use of multiple benchmark concentrations, provides for characterization of the
 6 risk of other respiratory effects, the type and severity of which increase with increased exposure
 7 concentration.

8



9

10 **Figure 3-4. Analysis approach for exposure-based risk analyses.** Dashed lines and gray box
 11 indicate the sole lung function risk approach used prior to 2014 HREA.

12 The analyses estimate exposure and risk for simulated populations in eight study areas in
 13 Atlanta, Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento and St. Louis. The eight
 14 study areas represent a variety of circumstances with regard to population exposure to short-term

1 concentrations of O₃ in ambient air. The eight study areas range in total population size from
2 approximately two to eight million and are distributed across the U.S. in seven of the nine
3 different NOAA climate regions: the Northeast, Southeast, Central, East North Central, South,
4 Southwest and West (Karl and Koss, 1984). Assessment of this set of study areas and the
5 associated exposed populations is intended to be informative to the EPA's consideration of
6 potential exposures and risks that may be associated with the air quality conditions that meet the
7 current primary standard.

8 This set of eight study areas represents a streamlined set as compared to the 15 study
9 areas in the 2015 review, with the areas chosen to ensure they reflect the full range of air quality
10 and exposure variation expected across major urban areas in the U.S. (2014 HREA, section 3.5).
11 As a specific example, while seven of the eight study areas were also included in the 2014
12 HREA, the eighth study area was not, and has been included in the more recent assessment to
13 insure representation of a large city in the southwest. Additionally, the years simulated reflect
14 more recent emissions and atmospheric conditions subsequent to data used in the 2014 HREA,
15 and therefore represent O₃ concentrations somewhat nearer the current standard than was the
16 case for study areas included in the HREA of the 2015 review (Appendix 3C, Table 3C and 2014
17 HREA, Table 4-1). Thus, the urban study areas (e.g., combined statistical areas that include
18 urban and suburban populations) the exposure and risk analyses discussed here reflect an array of
19 air quality, meteorological, and population exposure conditions.

20 Consistent with the health effects evidence (summarized in section 3.3 above), the focus
21 of the assessment is on short-term exposures of individuals in the population during times when
22 they are breathing at an elevated rate. Exposure and risk are characterized for four population
23 groups that include representation of key at-risk populations (children and people with asthma),
24 as described in section 3.3.2 above. Two of the four groups are populations of school-aged
25 children, aged 5 to 18 years:⁶⁸ all children and children with asthma. Two are populations of
26 adults: all adults and adults with asthma. Another population identified as at risk for O₃, outdoor
27 workers, was not included due to appreciable data limitations, a decision also made for past
28 exposure assessments.⁶⁹

⁶⁸ The child population group focuses on ages 5 to 18 in recognition of data limitations and uncertainties, including those related to accurately simulating activities performed, estimating physiological attributes, and also challenges in asthma diagnoses for children younger than 5 years old.

⁶⁹ Outdoor workers, due to the requirements of their job spend more time outdoors at elevated exertion. For a number of reasons, including the appreciable data limitations (e.g., related to specific durations of time spent outdoors and activity data), and associated uncertainties summarized in Table 3D-64 of Appendix 3D, this group was not simulated in this assessment. Limited exploratory analyses of a hypothetical outdoor worker population in the 2014 HREA (single study area, single year) for the 75 ppb air quality scenario estimated an appreciably greater portion of this population to experience exposures at or above benchmark concentrations than the full

1 Asthma prevalence estimates for each of the entire populations in the eight study areas
2 ranges from 7.7 to 11.2%; the rates for children in these study areas range from 9.2 to 12.3%
3 (Appendix 3D, section 3D.3.1). Spatial variation within each study area related to the population
4 distribution of age, sex, and family income was also taken into account.⁷⁰ For children, this
5 variation is greatest in the Detroit study area, with census tract level, age-specific asthma
6 prevalence estimates ranging from 6.4 to 13.2% for girls and from 7.7 to 25.5% for boys
7 (Appendix 3D, Table 3D-3).

8 Ambient air O₃ concentrations were estimated in each study area for the air quality
9 conditions of interest by adjusting hourly ambient air concentrations, from monitoring data for
10 the years 2015-2017, using a photochemical model-based approach and then applying a spatial
11 interpolation technique to produce air quality surfaces with high spatial and temporal resolution
12 (Appendix 3C).⁷¹ The photochemical modeling outputs included both modeled O₃ concentrations
13 and sensitivities of O₃ concentrations to changes in NO_x emissions for each hour in a single year
14 at all ambient air monitor locations (Appendix 3C, sections 3C.4 and 3C.5). Linear regression
15 was used with these single-year model outputs to create relationships between the sensitivities
16 and O₃ concentrations at each monitoring location for each hour of the day during each of the
17 four seasons. The relationships between hourly sensitivities and hourly O₃ for each season were
18 then used with three years of ambient air monitoring data at each location to predict hourly
19 sensitivities for the complete 3-year record at each monitoring location. From these, we
20 calculated hourly O₃ concentrations at each monitor location based on iteratively increasing NO_x
21 reductions to determine the adjustments necessary for the monitor location with the highest
22 design value in each study area to just meet the target value, e.g., 70 ppb for the current standard
23 scenario (Appendix 3C, section 3C.5). Hourly O₃ concentrations for all census tracts comprising
24 each study area were then derived from the model adjusted hourly concentrations at the ambient
25 air monitor locations using the Voronoi Neighbor Averaging (VNA) spatial interpolation
26 technique (Appendix 3C, section 3C.6). The final products were datasets of ambient air O₃
27 concentration estimates with high temporal and spatial resolution (hourly concentrations in 500

adult or child populations simulated, although there are a number of uncertainties associated with the estimates due to appreciable limitations in the data underlying the analyses (2014 HREA, section 5.4.3.2). It is expected that if an approach similar to that used in the 2014 HREA had been used for this assessment a generally similar pattern might be observed, although with somewhat lower overall percentages based on the comparison of current estimates with estimates from the 2014 HREA (Appendix 3D, section 3D.3.2.4).

⁷⁰ As described in Appendix 3D, section 3D.2.2.2, asthma prevalence in each study area is estimated based on combining regional national prevalence information from NHIS with U.S census tract level population data by linking demographic information related to age, sex, and family income. Then, further adjustments were made using state-level prevalence obtained from the U.S. Behavioral Risk Factor Surveillance System. See Appendix 3D, Attachment 1 for details.

⁷¹ A similar approach was used to develop the air quality scenarios for the 2014 HREA.

1 to 1700 census tracts) for each of the eight study areas (Appendix 3C, section 3C.7) representing
2 each of the three air quality scenarios assessed.⁷²

3 The photochemical modeling approach involved use of the Comprehensive Air Quality
4 Model with Extensions (CAMx), version 6.5, instrumented with the higher order decoupled
5 direct method (HDDM).⁷³ The CAMx-HDDM was run with emissions estimates and
6 meteorology data for calendar year 2016 to estimate the O₃ sensitivities,⁷⁴ and the linear
7 regressions of the modeled O₃ concentrations to their respective sensitivities were applied to
8 hourly O₃ concentrations reported at ambient air monitors for the 2015-2017 period to determine
9 the adjustments needed for each air quality scenario (Appendix 3C, sections 3C.4 and 3C.5). We
10 maximized the spatial representation of the monitoring data by using all available monitors
11 within each study area (between 12 and 30) in addition to those within 50 km of the study area
12 boundaries (yielding between 5 and 31 additional monitors per area). Because we selected study
13 areas having design values close to the level of the current standard, the levels of NO_x emissions
14 adjustments needed to meet the air quality scenarios of interest were generally lower than those
15 used in the 2014 HREA, thus reducing one of the important sources of uncertainty associated
16 with these air quality estimates.

17 Population exposures were estimated using the EPA's Air Pollutant Exposure model
18 (APEX) version 5, which probabilistically generates a large sample of hypothetical individuals
19 from demographic and activity pattern databases and simulates each individual's movements
20 through time and space to estimate their time-series of O₃ exposures occurring within indoor,
21 outdoor, and in-vehicle microenvironments (Appendix 3D, section 3D.2).⁷⁵ The APEX model
22 accounts for the most important factors that contribute to human exposure to O₃ from ambient
23 air, including the temporal and spatial distributions of people and ambient air O₃ concentrations
24 throughout a study area, the variation of ambient air-related O₃ concentrations within various
25 microenvironments in which people conduct their daily activities, and the effects of activities

⁷² For this assessment, high spatial and temporal resolution O₃ concentration datasets were created for conditions representing each area meeting the current standard of 70 ppb and two alternative air quality scenarios characterized by ozone concentrations that would result in design values of 75 and 65 ppb representing a level slightly above and a level slightly below the current standard.

⁷³ Details on the models, methods and input data used to estimate ambient air concentrations for the eight study areas are provided in Appendix 3C. The "higher order" aspect of the HDDM tool refers to the capability of capturing nonlinear response curves (Appendix 3C, section 3C.5.1).

⁷⁴ Sensitivities of O₃ refer to predicted incremental changes in O₃ concentrations in response to incremental changes in precursor emissions (e.g., NO_x emissions).

⁷⁵ The APEX model is a probabilistic model that estimates population exposure using a stochastic, event-based microenvironmental approach. This model has a history of application, evaluation, and progressive model development in estimating human exposure, dose, and risk for reviews of NAAQS for gaseous pollutants, including the 2015 review of the O₃ NAAQS (U.S. EPA, 2008; U.S. EPA, 2009; U.S. EPA, 2010; U.S. EPA, 2014; U.S. EPA, 2018).

1 involving different levels of exertion on breathing rate (or ventilation rate) for the exposed
2 individuals of different sex, age, and body mass in the study area (Appendix 3D, section 3D.2).
3 The APEX model generates each simulated person or profile by probabilistically selecting values
4 for a set of profile variables, including demographic variables, health status and physical
5 attributes (e.g., residence with air conditioning, height, weight, body surface area) and activity-
6 specific ventilation rate (Appendix 3D, section 3D.2).

7 By incorporating individual activity patterns⁷⁶ and estimating physical exertion for each
8 exposure event,⁷⁷ the model addresses an important determinant of individual's exposure (2013
9 ISA, section 4.4.1). This aspect of the exposure modeling is critical in estimating exposure,
10 ventilation rate, O₃ intake (dose), and health risk resulting from ambient air concentrations of
11 O₃.⁷⁸ Because of variation in O₃ concentrations among the different microenvironments in which
12 individuals are active, the amount of time spent in each location, as well as the exertion level of
13 the activity performed, will influence an individual's exposure to O₃ from ambient air and
14 potential for adverse health effects. Activity patterns vary both among and within individuals,
15 resulting in corresponding variations in exposure across a population and over time (2013 ISA,
16 section 4.4.1). For each exposure event, APEX tracks activity performed, ventilation rate,
17 exposure concentration, and duration for all simulated individuals throughout the assessment
18 period. This time-series of exposure events serves as the basis for calculating exposure and risk
19 metrics of interest.

20 The APEX model estimates of population exposures for simulated individuals breathing
21 at elevated rates⁷⁹ are used to characterize health risk based on information from the controlled
22 human exposure studies on the incidence of lung function decrements in study subjects who are
23 exposed over multiple hours while intermittently or quasi-continuously exercising (Appendix
24 3D, section 3D.2.8). In drawing on this evidence base for this purpose, the assessment gives

⁷⁶ To represent personal time-location-activity patterns of simulated individuals, the APEX model draws from the CHAD developed and maintained by the EPA (McCurdy, 2000; U.S. EPA, 2019). The CHAD is comprised of data from several surveys that collected activity pattern data at city, state, and national levels. Included are personal attributes of survey participants (e.g., age, sex), the locations visited, and activities performed by survey participants throughout a day, and the time-of-day activities occurred and their duration (Appendix 3D, section 3D.2.5.1).

⁷⁷ An exposure event occurs when a simulated individual inhabits a microenvironment for a specified time, while engaged at a constant exertion level and experiencing a particular pollutant concentration. If the microenvironmental concentration and/or activity/activity level changes, a new exposure event occurs (McCurdy and Graham, 2003).

⁷⁸ Indoor sources are generally minor in comparison to O₃ from ambient air (ISA, Appendix 2, section 2.4.3) and are not accounted for by the exposure modeling in this assessment.

⁷⁹ Based on minute-by-minute activity levels, and physiological characteristics of the simulated person, APEX estimates an equivalent ventilation rate (EVR), by normalizing the simulated individuals' activity-specific ventilation rate to their body surface area (Appendix 3D, section 3D.2.2.3.3).

1 primary focus to the well-documented controlled human exposure studies summarized in
2 Appendix 3A, Table 3A-1 for 6.6-hour average exposure concentrations ranging from 40 ppb to
3 120 ppb (Figure 3-2; ISA, Appendix 3, Figure 3-3). Health risk is characterized in two ways,
4 producing two types of risk metrics: one involving comparison of population exposures
5 involving elevated exertion to benchmark concentrations (that are specific to elevated exertion
6 exposures), and the second involving estimated population occurrences of ambient air O₃-related
7 lung function decrements (Figure 3-2). The first risk metric estimates population occurrences of
8 daily maximum 7-hour average exposure concentrations (during periods of elevated breathing
9 rates) at or above concentrations of potential concern (benchmark concentrations). The second
10 metric (lung function risk) uses E-R information for O₃ exposures and FEV₁ decrements to
11 estimate the portion of the simulated at-risk population expected to experience one or more days
12 with an O₃-related FEV₁ decrement of at least 10%, 15% and 20%. Both of these metrics are
13 used to characterize health risk associated with O₃ exposures among the simulated population
14 during periods of elevated breathing rates. Similar risk metrics were also derived in the HREA
15 for the 2015 review and the associated estimates informed the Administrator's 2015 decision on
16 the current standard (80 FR 65292, October 26, 2015).

17 The general approach and methodology for the exposure-based assessment is similar to
18 that used in the 2015 review although a number of updates and improvements, related to the air
19 quality, exposure and risk aspects of the assessment, have been implemented (Appendices 3C
20 and 3D). These are summarized here.

- 21 • The ambient air monitoring data used is from a more recent period (e.g., 2015-2017)
22 during which O₃ concentrations in the eight study areas are at or near the current standard
23 (Appendix 3C, Table 3C-1). This contrasts with the 2014 HREA use of 2006-2010 air
24 monitoring data, that for many study areas included design values (for unadjusted
25 concentrations) well above (e.g., by more than 10 ppb) the level of the then-existing
26 standard (2014 HREA, section 4.3.1.1, Table 4-1). The use of more recent ambient air
27 monitoring data in the current analysis allows for smaller adjustments to develop the air
28 quality conditions of interest, thus contributing to generally lesser uncertainty in the
29 concentrations estimated in each air quality scenario.
- 30 • The most recent CAMx model, with updates to the treatment of atmospheric chemistry
31 and physics within the model, is used to derive spatially and temporally varying
32 relationships between changes to emissions and modeled O₃ concentrations, which are
33 then used in adjusting ambient air concentrations to just meet the air quality scenarios.
34 Model inputs represent recent year emissions, meteorology, and international transport
35 (e.g., 2016). The 2016-based inputs were derived using updated methods for calculating
36 emissions, as well as updated meteorological and hemispheric photochemical models
37 (described in more detail in Appendix 3C).
- 38 • A significantly expanded CHAD, with now nearly 180,000 diaries, including over 25,000
39 for school-aged children is drawn on in the exposure modeling (Appendix 3D, section

1 3D.2.5.1), as are updated National Health and Nutrition Examination Survey data (2009-
2 2014), which are the basis for the age- and sex-specific body weight distributions used to
3 specify the individuals in the modeled populations (Appendix 3D, section 3D.2.2.3.1).

- 4 • Population exposure modeling inputs include the most recent U.S. Census demographics
5 and commuting data (i.e., 2010), meteorological data to reflect the assessment years
6 studied (e.g., 2015-2017), and updated estimates of asthma prevalence for all census
7 tracts in all study areas (e.g., 2013-2017). Regarding asthma prevalence, the more recent
8 information includes increased prevalence reported for adults and for children aged 10-17
9 years (Akinbami et al., 2016; CDC, 2016).⁸⁰
- 10 • The APEX equations used to estimate of ventilation rate (\dot{V}_E) and resting metabolic rate
11 have been updated such that the overall statistical model fit and predictability has been
12 improved (U.S. EPA, 2018, Appendix H).
- 13 • The approach for deriving population exposure estimates, both for comparison to
14 benchmark concentrations and for use in deriving lung function risk using the E-R
15 function, has been modified to provide for a better match of the simulated population
16 exposure estimates with the 6.6-hour duration of the controlled human exposure studies
17 and with the study subject ventilation rates (Appendix 3D, section 3D.2.8.1). The
18 modifications include deriving estimates for exposures of a duration and ventilation rate
19 more closely corresponding to the duration and average ventilation rate across the 6.6-
20 hour duration in the controlled human exposure studies (Appendix 3D, section 3D.2.8.1).
21 ⁸¹
- 22 • In addition to the E-R function, as updated in the 2014 HREA, an updated version of the
23 McDonnell Stewart Smith model (MSS-FEV₁ model, McDonnell et al., 2013) is used to
24 estimate individual-based lung function risk. Although the impact on risk estimates is
25 unclear, the updated MSS model has been described as better accounting for intra-subject
26 variability, yielding an improved model fit (McDonnell et al., 2013; Appendix 3D,
27 section 3D.2.8.2.2).

28 The comparison-to-benchmarks analysis characterizes the extent to which individuals in
29 at-risk populations could experience O₃ exposures, while engaging in their daily activities, with
30 the potential to elicit the effects reported in controlled human exposure studies for concentrations
31 at or above specific benchmark concentrations. Results are characterized through comparison of
32 exposure concentrations to three benchmark concentrations of O₃: 60, 70, and 80 ppb. These are
33 based on the three lowest concentrations targeted in studies of 6- to 6.6-hour exposures, with
34 quasi-continuous exercise (at moderate level of exertion), and that yielded different occurrences

⁸⁰ For more information, see <https://www.cdc.gov/nchs/products/databriefs/db239.htm>.

⁸¹ Estimated exposures for a 7-hour duration are used in the comparison to benchmark concentrations (that are based on the 6.6-hour exposure studies). The use of 7-hour exposure duration provides for a closer match of the duration for the benchmark concentrations to the duration of population exposure concentration estimates than the 8-hour exposure concentrations used in the last review. Additionally, an equivalent ventilation rate (EVR) of at least 17.3 L/min-m² is used to more closely correspond to the average across the 6.6 hours of the controlled human exposure studies (Appendix 3D, section 3D.2.8.1).

1 of statistical significance, and severity of respiratory effects (section 3.3.3 above; Appendix 3A,
2 section 3A.1; Appendix 3D, section 3D.2.8.1). The lowest benchmark, 60 ppb, represents the
3 lowest exposure concentration for which controlled human exposure studies have reported
4 statistically significant respiratory effects (as summarized in section 3.3.3 above). Exposure to
5 approximately 70 ppb⁸² averaged over a similar time resulted in a larger group mean lung
6 function decrement, as well as a statistically significant increase in prevalence of respiratory
7 symptoms over what was observed for 60 ppb (Figure 3-3; ISA, Appendix 3, section 3.1.4.1.1;
8 Schelegle et al., 2009). Studies of exposures to approximately 80 ppb have reported larger lung
9 function decrements at the study group mean than following exposures to 60 or 70 ppb, in
10 addition to an increase in airway inflammation, increased respiratory symptoms, increased
11 airway responsiveness, and decreased resistance to other respiratory effects (Figure 3-3 and
12 section 3.3.3, above; ISA, Appendix 3, sections 3.1.4.1-3.1.4.4).

13 The APEX-generated exposure concentrations for comparison to these benchmark
14 concentrations is the average of concentrations encountered by an individual while at an activity
15 level that elicits the specified elevated ventilation rate.⁸³ The incidence of such exposures at or
16 above the benchmark concentrations are summarized for each simulated population, study area,
17 and air quality scenario as discussed in sections 3.4.2 and 3.4.3 below (Appendix 3D).

18 The lung function risk analysis estimates (in two different ways) the extent to which
19 individuals in exposed populations could experience different sizes of O₃-induced lung function
20 decrements. The two different approaches utilize the evidence from the 6.6-hour controlled
21 human exposure studies in different ways.⁸⁴ One, the population-based E-R function, uses
22 quantitative descriptions of the E-R relationships for study group incidence of different

⁸² The design for the study on which the 70 ppb benchmark concentration is based, Schelegle et al. (2009), involved varying concentrations across the full exposure period. The study reported the average O₃ concentration measured during each of the six exercise periods. The mean concentration across these six values is 72 ppb. The 6.6-hr time weighted average based on the six reported measurements and the study design is 73 ppb (Schelegle et al., 2009). Other 6.6-hr studies generally report an exposure concentration precision at or below 3 ppb (e.g., Adams, 2006b).

⁸³ The model averages the ventilation rate (\dot{V}_E) for the exposed individual (based on the activities performed) over 7-hour periods. This is done based on the APEX estimates of \dot{V}_E and exposure concentration for every individual's time-series of exposure events. For the exposure duration of interest (e.g., 7 hours), the model derives and outputs the daily maximum average \dot{V}_E (and hence an equivalent ventilation rate or EVR) and simultaneously occurring exposure concentration for the specified duration for each simulated individual. To reasonably extrapolate the ventilation rate of the controlled human study subjects (i.e., adults having a specified body size and related lung capacity), who were engaging in quasi-continuous exercise during the study period, to individuals having varying body sizes (e.g., children with smaller size and related lung capacity), an equivalent ventilation rate (EVR) was calculated by normalizing the ventilation rate (L/min) by body surface area (m²). Seven-hour exposure concentrations associated with 7-hour average EVR at or above the target of 17.3 ± 1.2 L/min-m² (i.e., the value corresponding to average EVR across the 6.6-hour study duration in the controlled human exposure studies) are compared to the benchmark concentrations (Appendix 3D, section 3D.2.8.1).

⁸⁴ The two approaches also estimate responses associated with unstudied exposure circumstances and population groups in different ways.

1 magnitudes of lung function decrements based on the individual study subject observations. The
2 second, the individual-based MSS model, uses quantitative estimations of biological processes
3 identified as important in eliciting the different sizes of decrements at the individual level, with a
4 factor that also provides a representation of intra- and inter-individual response variability
5 (Appendix 3D, section 3D.2.8.2.2). The two approaches, described in detail in Appendix 3D,
6 utilize evidence from the 6.6-hour controlled human exposure studies in different ways, and
7 accordingly, differ in their strengths, limitations, and uncertainties.

8 The E-R function used for estimating the risk of lung function decrements was developed
9 from the individual study subject measurements of O₃-related FEV₁ decrements from the 6.6-
10 hour controlled human exposure studies targeting mean exposure concentrations from 120 ppb
11 down to 40 ppb (Appendix 3D, Table 3D-19; Appendix 3A, Figure 3A-1). The FEV₁ responses
12 reported in these studies have been summarized in terms of percent of study subjects
13 experiencing O₃-related decrements equal to at least 10%, 15% or 20%. Across the exposure
14 range from 40 to 120 ppb, the percentage of exercising study subjects with asthma estimated to
15 have at least a 10% O₃ related FEV₁ decrement increases from 0 to 7% (a statistically non-
16 significant response at exposures of 40 ppb) up to approximately 50 to 70% (at exposures of 120
17 ppb) (Appendix 3D, Section 3D.2.8.2.1, Table 3D-19). The E-R function relies on equations that
18 describe the fraction of the population experiencing a particular size decrement as a function of
19 the exposure concentration experienced while at the target ventilation rate.⁸⁵ This type of risk
20 model has been used in risk assessments since the 1997 O₃ NAAQS review. As used here, the
21 functions (fraction of the population having of a day or more per simulation period with at least
22 one decrement of one of the specified sizes) are applied to the APEX estimates of 7-hour average
23 exposure concentrations concomitant with the target ventilation level estimated by APEX, with
24 the results presented in terms of number of individuals in the simulated populations (and percent
25 of the population) estimated to experience a day (or more) with a lung function decrement at or
26 above 10%, 15% and 20%.

27 The MSS model, also used for estimating the risk of lung function decrements, was
28 developed using the extensive database from controlled human exposure studies that has been
29 compiled over the past several decades, and biological concepts based on that evidence
30 (McDonnell et al., 2012; McDonnell et al., 2013). The model mathematically estimates the
31 magnitude of FEV₁ decrement as a function of inhaled O₃ dose (based on concentration &
32 ventilation rate) over the time period of interest (Appendix 3D, section 3D.2.8.2.2). The
33 simulation of decrements is dynamic, based on a balance between predicted development of the

⁸⁵ This risk model was updated in the 2015 review to include the more recently available study data at that time (Appendix 3D, section 3D.2.8.2.1).

1 decrement in response to inhaled dose and predicted recovery (using a decay factor). Each
2 occurrence of decrements of interest (e.g., at or above 10%, 15% and 20%) is tallied. This model
3 was first applied in combination with the APEX model to generate lung function risk estimates
4 in the 2015 O₃ NAAQS review (80 FR 65314, October 26, 2015).⁸⁶

5 To generate risk estimates for lung function decrements, the model is applied to the
6 APEX estimates of exposure concentration and ventilation for every exposure event experienced
7 by a simulated individual. The model then utilizes its mathematical descriptions of dose
8 accumulation and decay, and relationship of dose to response, to estimate the magnitude of O₃
9 response associated with the sequence of exposure events in each individual's day. We report the
10 MSS model risk results using the same metrics as for the E-R function, i.e., number of
11 individuals in the simulated populations (and percent of the population) estimated to experience
12 a day (or more) per simulation period with a lung function decrement at or above 10%, 15% and
13 20%.

14 The comparison-to-benchmark analysis (involving comparison of 7-hour average
15 exposure concentrations that coincide with a 7-hour average elevated ventilation rates) provides
16 perspective on the extent to which the air quality being assessed could be associated with
17 discrete exposures to O₃ concentrations reported to result in an array of respiratory effects. For
18 example, estimates of such exposures can indicate the potential for O₃-related effects in the
19 exposed population, including effects for which we do not have E-R functions that could be used
20 in quantitative risk analyses (e.g., airway inflammation). Thus, the comparison-to-benchmark
21 analysis differs from the two lung function risk analyses with their specific focus on lung
22 function decrements and provides for a broader risk characterization with consideration of the
23 array of O₃-related respiratory effects.

24 **3.4.2 Population Exposure and Risk Estimates for Air Quality Just Meeting the Current** 25 **Standard**

26 In this section, we consider the exposure and risk estimates in the context of the
27 following questions.

- 28 • **What are the nature and magnitude of O₃ exposures and associated health risks for**
29 **air quality conditions just meeting the current standard? What portions of the**
30 **exposed populations are estimated to experience exposures of concern or lung**
31 **function decrements?**

32 To address these questions, we consider the estimates provided by the exposure and risk
33 simulations for the eight urban study areas with air quality conditions adjusted to just meet the

⁸⁶ As noted below, the MSS model used in the current assessment has been updated since the 2015 review based on the most recent study by its developers (McDonnell et al., 2013).

1 current standard (Appendix 3D, sections 3D.3.2 through 3D.3.3). In considering these estimates
2 here and their associated limitations, uncertainties and implications in greater depth in sections
3 3.4.5 and 3.5 below, we particularly focus on the extent of protection provided by the standard
4 from O₃ exposures of potential concern. As described in the prior section, the exposure and risk
5 analyses present two types of risk estimates for the 3-year simulation in each study area: (1) the
6 number and percent of simulated people experiencing exposures at or above the particular
7 benchmark concentrations of interest in a year, while breathing at elevated rates; and (2) the
8 number and percent of people estimated to experience at least one O₃-related lung function
9 decrement (specifically, FEV₁ reductions of a magnitude at or above 10%, 15% or 20%) in a
10 year and the number and percent of people estimated to experience multiple lung function
11 decrements.

12 As an initial matter regarding the objectives for the analysis approach, we note that the
13 analyses and the use of an urban case study approach (summarized in section 3.4.1 above) are
14 intended to provide assessments of air quality scenarios, including in particular one just meeting
15 the current standard, for a diverse set of areas and associated exposed populations. These
16 analyses are not intended to provide a comprehensive national assessment. Nor is the objective to
17 present an exhaustive analysis of exposure and risk in the areas that currently just meet the
18 current standard and/or of exposure and risk associated with air quality adjusted to just meet the
19 current standard in areas that currently do not meet the standard. Rather, the purpose is to assess,
20 based on current tools and information, the potential for exposures and risks beyond those
21 indicated by the information available at the time the standard was established. Accordingly, use
22 of this approach recognizes that capturing an appropriate diversity in study areas and air quality
23 conditions (that reflect the current standard scenario)⁸⁷ is an important aspect of the role of the
24 exposure and risk analyses in informing the Administrator's conclusions on the public health
25 protection afforded by the current standard.

26 Of the two types of risk metrics derived in the exposure and risk analyses, we turn first to
27 the results for the benchmark-based risk metric, which are summarized in terms of the percent of
28 the simulated populations of all children and children with asthma estimated to experience at

⁸⁷ A broad variety of spatial and temporal patterns of O₃ concentrations can exist when ambient air concentrations just meet the current standard. These patterns will vary due to many factors including the types, magnitude, and timing of emissions in a study area, as well as local factors, such as meteorology and topography. We focused our current assessment on specific study areas having ambient air concentrations close to conditions that reflect air quality that just meets the current standard. Accordingly, assessment of these study areas is more informative to evaluating the health protection provided by the current standard than would be an assessment that included areas with much higher and much lower concentrations.

1 least one day per year⁸⁸ with a 7-hour average exposure concentration at or above the different
2 benchmark concentrations while breathing at elevated rates under air quality conditions just
3 meeting the current standard (Table 3-3). The estimates for the adult populations, in terms of
4 percentages, are generally lower, due to the lesser amount and frequency of time spent outdoors
5 at elevated exertion (Appendix 3D, section 3D.3.2). Given the recognition of people with asthma
6 as an at-risk population and the relatively greater amount and frequency of time spent outdoors at
7 elevated exertion of children, we focus here on the estimates for children, including children with
8 asthma.

9 Under air quality conditions just meeting the current standard, less than 0.1% of any
10 study area's children with asthma, on average, were estimated to experience any days per year
11 with a 7-hour average exposure at or above 80 ppb, while breathing at elevated rates (Table 3-3).
12 With regard to the 70 ppb benchmark, the study areas' estimates for children with asthma range
13 up to 0.7 percent (0.6% for all children), on average across the 3-year period, and range up to
14 1.0% in a single year (Table 3-3). Approximately 3% to nearly 9% of each study area's
15 simulated children with asthma, on average across the 3-year period, are estimated to experience
16 one or more days per year with a 7-hour average exposure at or above 60 ppb (Table 3-3). This
17 range is very similar for the populations of all children (Table 3-3).

18 Regarding multiday occurrences, we see that no children are estimated to experience
19 more than a single day with a 7-hour average exposure at or above 80 ppb in any year simulated
20 in any study location (Table 3-3). For the 70 ppb benchmark, the estimate is less than 0.1% of
21 any area's children (on average across 3-year period), both those with asthma and all children
22 (Table 3-3, Figure 3-4). The estimates for the 60 ppb benchmark are slightly higher, with up to
23 3% of children estimated to experience more than a single day with a 7-hour average exposure at
24 or above 60 ppb, on average (and more than 4% in the highest year across all eight study area
25 locations) (Table 3-3).

26 These estimates are based on analyses that, while based on conceptually similar
27 approaches to those used in the 2014 HREA, reflect the updates and revisions to those
28 approaches that have been implemented since that time. Taking that into consideration, the
29 estimates for the 3-year period from the current assessment for air quality conditions simulated to
30 just meet the current standard are of a magnitude roughly similar, although slightly lower at the
31 upper end of the ranges, to the estimates for these same populations in the 2014 HREA. For

⁸⁸ The three years of ambient air O₃ concentrations analyzed in the exposure assessment analyses include concentrations during the O₃ seasons for that area. These seasons capture the times during the year when concentrations are elevated (80 FR 65419-65420, October 26, 2015). While the duration of an O₃ season for each year may vary across the study areas, for the purposes of the exposure and risk analyses, the O₃ season in each study area is considered synonymous with a year.

1 example, for air quality conditions just meeting the standard with a level of 70 ppb, the 2014
 2 HREA estimated 0.1 to 1.2% of children to experience at least one day with exposure at or above
 3 70 ppb, while at elevated ventilation (Appendix 3D, section 3D.3.2.4, Table 3D-38). There are a
 4 number of differences between the quantitative modeling and analyses performed in the current
 5 assessment and the 2014 HREA that likely contribute to the small differences in estimates
 6 between the two assessments (e.g., 2015-2017 vs. 2006-2010 distribution of ambient air
 7 concentrations, full statistical distribution of ventilation rates vs. a 5th percentile point estimate,
 8 7-hour vs. 8-hour exposure durations).

9 **Table 3-3. Percent and number of simulated children and children with asthma**
 10 **estimated to experience at least one or more days per year with a 7-hour**
 11 **average exposure at or above indicated concentration while breathing at an**
 12 **elevated rate in areas just meeting the current standard.**

Exposure Concentration (ppb)	One or more days		Two or more days		Four or more days	
	Average per year	Highest in a single year	Average per year	Highest in a single year	Average per year	Highest in a single year
<i>Children with asthma - percent of simulated population^A</i>						
≥ 80	0 ^B – <0.1 ^C	0.1	0	0	0	0
≥ 70	0.2 – 0.7	1.0	<0.1	0.1	0	0
≥ 60	3.3 – 8.8	11.2	0.6 – 3.2	4.9	<0.1 – 0.8	1.3
<i>- number of individuals^A</i>						
≥ 80	0 – 67	202	0	0	0	0
≥ 70	93 – 1145	1616	3 – 39	118	0	0
≥ 60	1517 – 8544	11776	282 – 2609	3977	23 – 637	1033
<i>All children - percent of simulated population^A</i>						
≥ 80	0 ^B – <0.1	0.1	0	0	0	0
≥ 70	0.2 – 0.6	0.9	<0.1	0.1	0 – <0.1	<0.1
≥ 60	3.2 – 8.2	10.6	0.6 – 2.9	4.3	<0.1 – 0.7	1.1
<i>- number of individuals^A</i>						
≥ 80	0 – 464	1211	0	0	0	0
≥ 70	727 – 8305	11923	16 – 341	757	0 – 5	14
≥ 60	14928 – 69794	96261	2601 – 24952	36643	158 – 5997	9554
^A Estimates for each study area were averaged across the 3-year assessment period. Ranges reflect the ranges of averages. ^B A value of zero (0) means that there were no individuals estimated to have the selected exposure in any year. ^C An entry of <0.1 is used to represent small, non-zero values that do not round upwards to 0.1 (i.e., <0.05).						

13
 14 In framing these same exposure estimates from the perspective of estimated protection
 15 provided by the current standard, these results indicate that, in the single year with the highest
 16 concentrations across the 3-year period, 99% of the population of children with asthma would
 17 not be expected to experience such a day with an exposure at or above the 70 ppb benchmark;

1 99.9% would not be expected to experience such a day with exposure at or above the 80 ppb
2 benchmark. The estimates, on average across the 3-year period, indicate that over 99.9%, 99.3%
3 and 91.2% of the population of children with asthma would not be expected to experience a day
4 with a 7-hour average exposure while at elevated ventilation that is at or above 80 ppb, 70 ppb
5 and 60 ppb, respectively (Table 3-3 above). Further, with regard to multiple days, more than
6 approximately 97% of all children or children with asthma (on average across a 3-year period),
7 are estimated to be protected against multiple days of exposures at or above 60 ppb. These
8 estimates indicate generally similar protection to that described in establishing the current
9 standard in 2015 (as summarized in section 3.1 above), with slightly greater level of protection
10 for occurrences at 70 ppb (see section 3.5.2 below, refer to Table 3-8).

11 With regard to lung function risk, the estimates for all children and for children with
12 asthma are again roughly similar, with the higher end of the ranges for the eight study areas
13 being just slightly higher in some cases for the children with asthma (Table 3-4). The lung
14 function risk estimates from the MSS model are appreciably higher than those based on the E-R
15 function (full results in Appendix 3D, section 3D.3.3). This difference relates to the fact, noted in
16 section 3.4.1 above, that the two lung function risk approaches are based on different aspects of
17 the controlled human exposure study evidence and differ in how they extrapolate beyond the
18 exposure study conditions and observations. Accordingly, uncertainties associated with the two
19 modeling approaches also differ (as discussed in section 3.4.4 below). The E-R function risk
20 approach conforms more closely to the circumstances of the 6.6-hour controlled human exposure
21 studies, such that the 7-hour duration and moderate or greater exertion level are necessary for
22 nonzero risk. This approach additionally, however, uses a continuous function which predicts
23 responses for exposure concentrations below those studied down to zero. As a result, exposures
24 below those studied in the controlled human exposures will result in a fraction of the population
25 being estimated by the E-R function to experience a lung function decrement (albeit to an
26 increasingly small degree with decreasing exposures). The MSS model, which has been
27 developed based on a conceptualization intended to reflect a broader set of controlled human
28 exposure studies (e.g., including studies of exposures to higher concentrations for shorter
29 durations), does not require a 7-hour exposure period for the model to generate an estimated
30 response, and lung function decrements are estimated for exertion below moderate or greater
31 levels, as well as for exposure concentrations lower than those that have been studied (Appendix
32 3D, section 3D.3.4.2; 2014 HREA section 6.3.3). These differences in the models, accordingly,
33 result in differences in the extent to which they produce estimates that reflect the particular

1 conditions of the available controlled human exposure studies and the frequency and magnitude
2 of the measured responses in those studies.⁸⁹

3 For example, the 6.6-hour controlled human exposure studies have reported
4 approximately 3% of subjects exposed to an average concentration of 60 ppb and 10% of
5 subjects exposed to 70 ppb to have at least a 15% FEV₁ decrement (Appendix 3D, Table 3D-20
6 and Figure 3D-11). Table 3-3 above shows that, at a maximum, approximately 11% and 1% of
7 children with asthma are estimated in a single year to have a day with daily maximum 7-hour
8 exposure at or above the 60 ppb and 70 ppb benchmarks, respectively, indicating that perhaps
9 10% (11% minus 1%) might be expected to have a day with an exposure at or above 60 ppb but
10 less than 70 ppb. If the simulated children had the same sensitivity as the controlled human
11 exposure study subjects, it might be expected that 0.3% (3% times 10%) of this group could have
12 a 15% (or larger) FEV₁ decrement resulting from concentrations at or above 60 ppb and less than
13 70 ppb and 0.1% (10% times 1%) of this group could have a 15% (or larger) decrement resulting
14 from concentrations at or above 70 ppb. Accordingly, this would yield an estimated lung
15 function risk for the simulated population of 0.4% for decrements of 15% or larger. This
16 contrasts with the estimates based on the E-R function, that are at most a 1% risk (Table 3-4),
17 and the MSS model estimates, that are at most an 8.7% risk (Table 3-4).

⁸⁹ The two models, their bases in the evidence and associated limitations and uncertainties are discussed in detail in Appendix 3D, sections 3D.2.8.2 and 3D.3.4.

1 **Table 3-4. Percent of simulated children and children with asthma estimated to**
 2 **experience at least one or more days per year with a lung function decrement**
 3 **at or above 10, 15 or 20% while breathing at an elevated rate in areas just**
 4 **meeting the current standard.**

Lung Function Decrement ^A	One or more days		Two or more days		Four or more days	
	Average per year	Highest in a single year	Average per year	Highest in a single year	Average per year	Highest in a single year
E-R Function						
	percent of simulated children with asthma ^A					
≥ 20%	0.2 – 0.3	0.4	0.1 – 0.2	0.2	<0.1 ^B – 0.1	0.1
≥ 15%	0.5 – 0.9	1.0	0.3 – 0.6	0.6	0.2 – 0.4	0.4
≥ 10%	2.3 – 3.3	3.6	1.5 – 2.4	2.6	0.9 – 1.7	1.8
	percent of all simulated children ^A					
≥ 20%	0.2 – 0.3	0.4	0.1 – 0.2	0.2	<0.1 – 0.1	0.1
≥ 15%	0.5 – 0.8	0.9	0.3 – 0.5	0.6	0.2 – 0.4	0.4
≥ 10%	2.2 – 3.1	3.3	1.3 – 2.2	2.4	0.8 – 1.6	1.7
MSS Model						
	percent of simulated children with asthma ^A					
≥ 20%	1.8 – 3.5	3.9	0.8 – 2.1	2.5	0.3 – 1.1	1.3
≥ 15%	4.5 – 8.2	8.7	2.2 – 4.9	5.3	1.1 – 2.9	3.3
≥ 10%	13.9 – 22	23.3	8.0 – 14.9	16	4.3 – 9.8	10.5
	percent of all simulated children ^A					
≥ 20%	1.7 – 3.1	3.6	0.8 – 1.7	2.0	0.3 – 0.9	1.1
≥ 15%	4.1 – 7.1	7.8	2.1 – 4.3	4.9	1.0 – 2.5	2.9
≥ 10%	13.2 – 20.4	21.8	7.4 – 13.6	14.8	3.9 – 8.8	9.7
^A Estimates for each urban case study area were averaged across the 3-year assessment period. Ranges reflect the ranges across urban study area averages. ^B An entry of <0.1 is used to represent small, non-zero values that do not round upwards to 0.1 (i.e., <0.05).						

5
 6 **3.4.3 Population Exposure and Risk Estimates for Additional Air Quality Scenarios**

7 In addition to estimating population exposure and risk for O₃ concentrations simulated to
 8 occur under air quality conditions when the current standard is just met, the exposure and risk
 9 analyses also estimated population exposure and risk in the eight study areas for two additional
 10 air quality scenarios. In these scenarios, the air quality conditions were adjusted such that the
 11 monitor location with the highest concentrations in each area had a design value just equal to
 12 either 75 ppb or 65 ppb.

13 The results for the comparison-to-benchmarks analysis for these additional air quality
 14 scenarios are summarized in Table 3-5 below for all three benchmark concentrations. The
 15 estimates for these two additional scenarios differ markedly from the results for air quality just
 16 meeting the current standard (summarized in Table 3-3 above). For simplicity, the summary of
 17 the comparison discussed here focuses on the 70 ppb benchmark concentration, which falls just

1 below the time-weighted exposure concentration for which there was a statistically significant
2 lung function decrement and also a statistically significant increase in respiratory symptom score
3 in one of the controlled human exposure studies, as noted in section 3.3.3 (ISA, Appendix 3,
4 section 3.1.4.1.1; Schelegle et al., 2009). The pattern is similar for the other two benchmarks,
5 although in general, the differences of the results for the additional scenarios from the results for
6 the current standard (presented in section 3.4.2) are somewhat greater for the higher benchmark
7 and slightly smaller for the lower benchmark.

8 Under air quality conditions in the 75 ppb scenario, estimated percentages of children
9 with asthma expected to experience at least one day per year with exposures at or above the
10 benchmark concentrations are two or more times higher than the estimates discussed in section
11 3.4.2 above for air quality conditions just meeting the current standard. For example, the
12 minimum and maximum percentages, on average per year across the study areas, of children
13 with asthma estimated to experience one or more days with exposures at or above the 70 ppb
14 benchmark are five and three times, respectively, greater than the corresponding percentages for
15 conditions associated with the current standard (Table 3-3 and Table 3-5). The highest estimated
16 percentage in a single year for the 70 ppb benchmark is more than twice as high for the 75 ppb
17 scenario compared to conditions associated with the current standard. The corresponding
18 estimate for two or more days per year is even greater for the 75 ppb scenario versus the current
19 standard scenario (Table 3-3 and Table 3-5).

20 In contrast, under air quality conditions in the 65 ppb scenario, the estimated percentages
21 of children with asthma expected to experience at least one day per year with exposures above
22 the benchmark concentrations are at most one third the estimates discussed in section 3.4.2 above
23 for air quality conditions just meeting the current standard (Table 3-3 and Table 3-5). The
24 highest estimated percentage of children expected to experience two or more days a year at or
25 above the 70 ppb benchmark drops to zero for the 65 ppb scenario compared to <0.1% for air
26 quality conditions just meeting the current standard (Table 3-3, Table 3-5).

27 As with the estimates for air quality just meeting the current standard, and as expected
28 given the various exposure and risk analysis updates implemented, the estimates discussed here
29 for the additional air quality scenarios are also slightly different from the estimates for such
30 scenarios that were derived in the 2015 review. However, the differences are not of such a
31 magnitude that the estimates for one air quality scenario in the current analyses are similar to
32 results for a different scenario in the 2015 review. For example, while the current estimates for
33 the 75 ppb air quality scenario are somewhat lower for some benchmarks than those for that
34 scenario in the 2015 review, they are still higher than the estimates from the 2015 review for the
35 air quality scenario just meeting the current standard.

36

1 **Table 3-5. Percent and number of simulated children and children with asthma**
 2 **estimated to experience one or more days per year with a daily maximum 7-**
 3 **hour average exposure at or above indicated concentration while breathing at**
 4 **an elevated rate – additional air quality scenarios.**

Exposure Concentration (ppb)	One or more days		Two or more days		Four or more days	
	Average per year	Highest in a single year	Average per year	Highest in a single year	Average per year	Highest in a single year
<i>Air quality scenario for 75 ppb</i>						
<i>Children with asthma - percent of simulated population^A</i>						
≥ 80	<0.1 ^B – 0.3	0.6	0 ^C – <0.1	<0.1	0	0
≥ 70	1.1 – 2.1	3.9	0.1 – 0.4	0.8	0 – <0.1	0.1
≥ 60	7.6 – 17.1	19.2	2.0 – 8.9	11.0	0.1 – 3.3	4.4
<i>- number of individuals^A</i>						
≥ 80	23 – 410	888	0 – 7	20	0	0
≥ 70	502 – 2480	4544	36 – 316	637	0 – 33	99
≥ 60	3538 – 14054	17673	1188 – 7232	8931	204 – 2708	3595
<i>All children - percent of simulated population^A</i>						
≥ 80	<0.1 ^B – 0.3	0.6	0 ^C – <0.1	<0.1	0	0
≥ 70	1.1 – 2.0	3.4	0.1 – 0.3	0.7	<0.1	<0.1
≥ 60	6.6 – 15.7	17.9	1.7 – 8.0	9.9	0.1 – 3.0	4.1
<i>- number of individuals^A</i>						
≥ 80	129 – 3127	6658	0 – 54	121	0	0
≥ 70	4915 – 19794	34981	414 – 2750	5775	3 – 141	368
≥ 60	34918 – 133400	162894	11087 – 67747	83660	1813 – 25773	34902
<i>Air quality scenario for 65 ppb</i>						
<i>Children with asthma - percent of simulated population^A</i>						
≥ 80	0 – <0.1	<0.1	0	0	0	0
≥ 70	0 – 0.2	0.3	0	0	0	0
≥ 60	0.5 – 2.5	4.3	<0.1 – 0.3	0.6	0 – <0.1	0.1
<i>- number of individuals^A</i>						
≥ 80	0 – 23	68	0	0	0	0
≥ 70	0 – 311	455	0	0	0	0
≥ 60	212 – 3542	5165	13 – 386	709	0 – 14	42
<i>All children - percent of simulated population^A</i>						
≥ 80	0 – <0.1	<0.1	0	0	0	0
≥ 70	0 – 0.2	0.2	0 – <0.1	<0.1	0	0
≥ 60	0.4 – 2.3	3.7	<0.1 – 0.3	0.5	0 – <0.1	<0.1
<i>- number of individuals^A</i>						
≥ 80	0 – 38	114	0	0	0	0
≥ 70	0 – 2495	3140	0 – 13	23	0	0
≥ 60	1832 – 29486	39772	83 – 3681	7188	0 – 179	354

^A Estimates for each study area were averaged across the 3-year assessment period. Ranges reflect the ranges of averages.
^B An entry of <0.1 is used to represent small, non-zero values that do not round upwards to 0.1 (i.e., <0.05).
^C A value of zero (0) means that there were no individuals estimated to have the selected exposure in any year.

1 Lung function risk estimated for children and children with asthma in air quality
2 scenarios with design values just above and below the current standard are presented in detail in
3 Appendix 3D, section 3D.3.3. The patterns of the estimates are, as expected, higher for the 75
4 ppb air quality scenario and lower for the 65 ppb scenario. For each scenario, the differences in
5 risk estimates between the two models is similar to that which occurs with the risk estimates for
6 air quality just meeting the current standard (as discussed in section 3.4.2 above). These
7 estimates (for both lung function risk approaches) are less different from those for the current
8 standard air quality scenario than are differences noted above for the comparison-to-benchmarks
9 estimates. This is due to the greater influence on the risk results of exposures associated with the
10 low O₃ concentrations that are less affected by air quality adjustments used to develop air
11 concentration surfaces for which the highest-concentration location has a design value just
12 meeting the different targets.

13 **3.4.4 Key Uncertainties**

14 In this section, we consider the uncertainties associated with the quantitative estimates of
15 exposure and risk, including those recognized by the characterization of uncertainty in Appendix
16 3D (section 3D.3.4). This characterization is based on an approach intended to identify and
17 compare the relative impact that important sources of uncertainty may have on the exposure and
18 risk estimates. The approach utilized is largely qualitative and is adapted from the World Health
19 Organization (WHO) approach for characterizing uncertainty in exposure assessment (WHO,
20 2008) augmented by several quantitative sensitivity analyses of key aspects of the assessment
21 approach (described in detail in Appendix 3D, section 3D.3.4). This characterization and
22 associated analyses build upon information generated from a previously conducted quantitative
23 sensitivity analysis of population-based O₃ exposure modeling (Langstaff, 2007), considering the
24 various types of data, algorithms, and models that together yield exposure and risk estimates for
25 the eight study areas. In this way, we considered the limitations and uncertainties underlying
26 these data, algorithms and models and the extent of their influence on the resultant exposure/risk
27 estimates using the general approach applied in past risk and exposure assessments for O₃,
28 nitrogen oxides, carbon monoxide and SO_x (U.S. EPA, 2008; U.S. EPA, 2010; U.S. EPA, 2014;
29 U.S. EPA, 2018).

30 The exposure and risk uncertainty characterization and quantitative sensitivity analyses,
31 presented in Appendix 3D, section 3D.3.4, involve consideration of the various types of inputs
32 and approaches that together result in the exposure and risk estimates for the eight study areas. In
33 this way the limitations and uncertainties underlying these inputs and approaches and the extent
34 of their influence on the resultant exposure/risk estimates are considered. Consistent with the
35 WHO (2008) guidance, the overall impact of the uncertainty is scaled by considering the extent

1 or magnitude of the impact of the uncertainty as implied by the relationship between the source
2 of the uncertainty and the exposure and risk output. The characterization in Appendix 3D also
3 evaluated the direction of influence, indicating how the source of uncertainty was judged, or
4 found, to quantitatively affect the exposure and risk estimates, e.g., likely to over- or under-
5 estimate (Appendix 3D, section 3D.3.4.1).

6 • **What are the important uncertainties associated with the exposure and risk**
7 **estimates?**

8 Based on the uncertainty characterization and associated analyses in Appendix 3D and
9 consideration of associated policy implications, we recognize several areas of uncertainty as
10 particularly important in our consideration of the exposure and risk estimates, while also
11 recognizing several areas where new or updated information reduced uncertainties in the
12 exposure and risk estimates compared to those in the 2015 review. In so doing, we note areas
13 that pertain to estimates for both types of risk metrics, as well as areas that pertain more to one
14 type of estimate versus the other. We also note differences in the uncertainties that pertain to
15 each of the two approaches used for the lung function risk metric.

16 An overarching and important area of uncertainty, remaining from the 2015 review and
17 important to our consideration of the exposure and risk analysis results, relates to the underlying
18 health effects evidence base. The quantitative analysis focuses on the evidence providing the
19 “strongest evidence” of O₃ respiratory effects (ISA, p. IS-1), the controlled human exposure
20 studies, and on the array of respiratory responses documented in those studies (e.g., lung function
21 decrements, respiratory symptoms, increased airway responsiveness and inflammation). The
22 comparison-to-benchmarks analysis is particularly focused on consideration of the potential for
23 exposures that pose a risk of experiencing this array of effects. We note, however, evidence is
24 lacking from controlled human exposure studies of 6.6-hour duration at the lower concentrations
25 (e.g., 60, 70 and 80 ppb) for children and for people of any age with asthma. While the limited
26 evidence informing our understanding of potential risk to people with asthma is uncertain, it
27 indicates the potential for this group, given their disease status, to be at risk (e.g., of asthma
28 exacerbation), as summarized in section 3.3.4 above. Such a conclusion is consistent with the
29 epidemiological study findings of positive associations of O₃ concentrations with asthma-related
30 emergency department visits and hospital admissions (and the higher effect estimates from these
31 studies), as referenced in section 3.3.1 above and presented in detail in the ISA. Thus, we
32 recognize uncertainty in interpretation of the exposure and risk estimates in the broader context
33 (e.g., as discussed in section 3.4.5 below).

34 Key uncertainties and limitations in data and tools that affect the quantitative estimates of
35 exposure and risk, particularly in their interpretation in the context of considering the current
36 standard, relate to each step in the assessment. These include uncertainty related to estimation of

1 the concentrations in ambient air for the current standard and the additional air quality scenarios;
2 lung function risk approaches that rely, to varying extents, on extrapolating from controlled
3 human exposure study conditions to lower exposure concentrations, lower ventilation rates, and
4 shorter durations; and characterization of risk for particular population groups that may be at
5 greatest risk, particularly for people with asthma, and particularly children with asthma. Areas in
6 which uncertainty has been reduced by new or updated information or methods include the use
7 of updated air quality modeling, with a more recent model version and model inputs, applied to
8 study areas with design values near the current standard, as well as updates to several inputs to
9 the exposure model, including changes to the exposure duration to better match those in the
10 controlled human exposure studies and an alternate approach to characterizing periods of activity
11 while at moderate or greater exertion for simulated individuals.

12 With regard to the analysis approach overall, two updates since the 2014 HREA reduce
13 uncertainty in the results. The first relates to identifying when simulated individuals may be at
14 moderate or greater exertion, with the new approach reducing the potential for overestimation of
15 the number of people achieving the associated ventilation rate, which was an important
16 uncertainty in the 2014 HREA. Additionally, the current analysis focus on exposures of 7 hours
17 duration better represents the 6.6-hour exposures from the controlled human exposure studies
18 (than the 8-hour exposure durations used for the 2014 HREA and prior assessments).

19 Additional aspects of the analytical design pertaining to both exposure-based risk metrics
20 include the estimation of ambient air O₃ concentrations for the air quality scenarios, and main
21 components of the exposure modeling. Uncertainties include the modeling approach used to
22 adjust ambient air concentrations to meet the air quality scenarios of interest and the method
23 used to interpolate monitor concentrations to census tracts. While the adjustment to conditions
24 near, just above, or just below the current standard is an important area of uncertainty, the size of
25 the adjustment needed to meet a given air quality scenario is minimized with the selection of
26 study areas for which recent O₃ design values were near the level of the current standard. Also,
27 more recent data are used as inputs for the air quality modeling, such as more recent O₃
28 concentration data (2015-2017), meteorological data (2016) and emissions data (2016), as well
29 as a recently updated air quality photochemical model which includes state-of-the-science
30 atmospheric chemistry and physics (Appendix 3C). Further, the number of ambient monitors
31 sited in each of the eight study areas provides a reasonable representation of spatial and temporal
32 variability for the air quality conditions simulated in those areas.

33 Among other key aspects, there is uncertainty associated with the simulation of study
34 area populations (and at-risk populations), including those with particular physical and personal
35 attributes. As also recognized in the 2014 HREA, exposures could be underestimated for some
36 population groups that are frequently and routinely outdoors during the summer (e.g., outdoor

1 workers, children.⁹⁰ In addition, longitudinal activity patterns do not exist for these and other
2 potentially important population groups (e.g., those having respiratory conditions other than
3 asthma), limiting the extent to which the exposure model outputs reflect information that may be
4 particular to these groups. Important uncertainties in the approach used to estimate energy
5 expenditure (i.e., metabolic equivalents of work or METs used to estimate ventilation rates),
6 include the use of longer-term average MET distributions to derive short-term estimates, along
7 with extrapolating adult observations to children. Both of these approaches are reasonable based
8 on the availability of relevant data and appropriate evaluations conducted to date, and
9 uncertainties associated with these steps are somewhat reduced in the current analyses (compared
10 to the 2014 HREA) because of the added specificity and use of redeveloped METs distributions
11 (based on newly available information), which is expected to more realistically estimate activity-
12 specific energy expenditure.

13 With regard to the exposure and risk modeling aspects of the two risk metrics, we
14 recognize that there are some uncertainties that apply to the estimation of lung function risk and
15 not the comparison-to-benchmarks analysis. For example, both lung function risk approaches
16 utilized in the risk analyses incorporate some degree of extrapolation beyond the exposure
17 circumstances evaluated in the controlled human exposure studies in recognition of the potential
18 for lung function decrements to be greater in unstudied population groups than is evident from
19 the available studies. For example, both models generate nonzero predictions for 7-hour
20 concentrations below the 6.6-hour concentrations investigated in the controlled human exposure
21 studies. In considering these risk estimates, we recognize that the uncertainty in the lung function
22 risk estimates increases with decreasing exposure concentration, and is particularly increased for
23 concentrations below those evaluated in controlled exposure studies (section 3.4.4 and Appendix
24 3D, section 3D.3.4). Further, the two lung function risk approaches differ in how they
25 extrapolate beyond the controlled human exposure study conditions and in the impact on the
26 estimates. The E-R function risk approach generates nonzero predictions from the full range of
27 potential nonzero concentrations for 7-hour average durations in which the average exertion
28 levels meets or exceeds the target. The MSS model, which draws on evidence-based concepts of
29 how human physiological processes respond to O₃, extrapolates beyond the controlled
30 experimental conditions, with regard to exposure concentration, exposure duration, and also,
31 ventilation rate (both magnitude and duration). The impact of this extrapolation, and the
32 difference between the two models in its extent beyond the studied exposure circumstances, is
33 illustrated by differences in the percent of the risk estimates derived on days for which the

⁹⁰ As described in section 3.4.1 above, the child populations modeled were school ages (ages 5 to 18), in recognition of limitations and uncertainties in the data for children younger than five years.

1 highest 7-hour average concentration is below the lowest 6.6-hour exposure concentration tested
2 (Table 3-6 and Table 3-7). For example, while 3 to 6% of the risk to children (based on single-
3 year estimates for three study areas) of experiencing at least one day with decrements greater
4 than 20% estimated by the E-R model is associated with exposure concentrations below 40 ppb
5 (the lowest exposure concentration studied, and at which no decrements of this severity occurred
6 in any study subjects), 25% to nearly 40% of MSS model estimates of decrements greater than
7 20% derive from exposures below 40 ppb (Table 3-6 and Table 3-7). Further, using ventilation
8 rates lower than those used for the E-R function risk approach (which are based on the controlled
9 human exposure study conditions) also contribute to relatively greater risks estimated by the
10 MSS model. Limiting the MSS model results to estimates for individuals with at least the same
11 exertion level achieved by study subjects (≥ 17.3 L/min-m²), reduces the risks of experiencing at
12 least one lung function decrement by an amount between 24 to 42% (Appendix 3D, Table 3D-
13 69).

14 The difference between the two models for risk contribution from low concentrations is
15 smaller for risk estimates for two or more days than the estimates for one or more days. This is
16 largely because the percent contribution to low-concentration risk for two or more decrement
17 days predicted by the E-R approach is, by design, greater than the corresponding contribution to
18 low-concentration risk for one or more days.⁹¹ This also occurs because the MSS model
19 estimates risk from a larger variety of exposure and ventilation conditions (Table 3-6, Table 3-7).
20 Further, many of the uncertainties previously identified as part of the 2014 HREA unique to the
21 MSS model remain as important uncertainties in the current assessment. For example, the
22 extrapolation of the MSS model age parameter down to age 5 (from the age range of 18- to 35-
23 year old study subjects to which the model was fit) is an important uncertainty given that
24 children are an at-risk population of particular interest in this assessment. Also, there is
25 uncertainty in estimating the frequency and magnitude of lung function decrements as a result of
26 the statistical form and parameters used for the MSS model inter- and intra-individual variability
27 terms. Each of these, among other newly identified MSS model uncertainties, are evaluated and
28 discussed in the current uncertainty characterization (Appendix 3D, section 3D.3.4). As a whole,
29 the differences between the two lung function risk approaches described above and the estimates
30 generated by these approaches indicate appreciably greater uncertainty associated with the MSS

⁹¹ The E-R function approach uses the daily maximum exposure concentration for the simulated population. By design, every individual would more than likely have a lower exposure on the second day than that experienced on the first day, and so on for each progressive day throughout the simulation period. Therefore, if any risk is estimated, the distribution of exposures would be shifted more so to lower concentrations for a greater proportion of the population.

1 model estimates than the E-R function estimates due to the significantly greater portion of
 2 relatively low concentrations contributing to risk.

3 **Table 3-6. Percent of risk estimated for air quality just meeting the current standard in**
 4 **three study areas using the E-R function approach on days where the daily**
 5 **maximum 7-hour average concentration is below specified values.**

Size of Lung Function Decrement	Percent of child population at risk of decrement from specific 7-hour concentrations ^A							
	Percent of one-or-more-days risk				Percent of two-or-more-days risk			
	< 30 ppb	< 40 ppb	< 50 ppb	< 60 ppb	< 30 ppb	< 40 ppb	< 50 ppb	< 60 ppb
≥ 20%	0.7 – 1%	3 – 6%	12 – 25%	39 – 70%	2 – 3%	7 – 12%	24 – 44%	67 – 93%
≥ 15%	2 – 3%	6 – 11%	19 – 34%	48 – 78%	4 – 5%	12 – 18%	34 – 54%	75 – 95%
≥ 10%	4 – 5%	11 – 16%	29 – 45%	61 – 86%	7 – 9%	18 – 25%	45 – 63%	83 – 97%

^A The ranges presented are based on 1-year simulations in three study areas (Atlanta, Dallas, and St Louis); the values presented here are rounded to whole numbers or at least one significant digit (full results are in Appendix 3D, section 3D.3.4.2, Table 3D-62).

6 **Table 3-7. Percent of risk estimated for air quality just meeting the current standard in**
 7 **three study areas using the MSS model approach on days where the daily**
 8 **maximum 7-hour average concentration is below specified values.**

Size of Lung Function Decrement	Percent of child population at risk of decrement from specified 7-hour concentrations ^A							
	Percent of one-or-more-days risk				Percent of two-or-more-days risk			
	< 30 ppb	< 40 ppb	< 50 ppb	< 60 ppb	< 30 ppb	< 40 ppb	< 50 ppb	< 60 ppb
≥ 20%	5 – 9%	25 – 38%	63 – 78%	88 – 96%	5 – 10%	28 – 42%	66 – 81%	90 – 98%
≥ 15%	11 – 18%	36 – 51%	72 – 84%	92 – 98%	11 – 19%	38 – 54%	74 – 87%	93 – 99%
≥ 10%	25 – 32%	57 – 67%	84 – 91%	96 – 99%	26 – 33%	57 – 68%	84 – 91%	96 – 99%

^A The ranges presented are based on 1-year simulations in three study areas (Atlanta, Dallas, and St Louis); the values presented here are rounded to whole numbers or at least one significant digit (full results are in Appendix 3D, section 3D.3.4.2, Table 3D-63).

9
 10 An additional area in which uncertainty has been reduced for the exposure estimates is
 11 related to the approach to identifying when simulated individuals may be at moderate or greater
 12 exertion. The approach used in the current assessment reduces the potential for overestimation of
 13 the number of people achieving the associated ventilation rate, an important uncertainty
 14 identified in the 2014 HREA. We also note that the exposure duration in the assessment was a 7-
 15 hour averaging time, which was selected to better represent the 6.6-hour exposures from the
 16 controlled human exposure studies, compared to the 8-hour exposure durations used in the model
 17 in the 2014 HREA and prior assessments.

18 In summary, among the multiple uncertainties and limitations in data and tools that affect
 19 the quantitative estimates of exposure and risk and their interpretation in the context of

1 considering the current standard, we recognize several here as particularly important, noting that
2 some of these uncertainties are similar to those recognized in the 2015 review. These include
3 uncertainty related to estimation of the concentrations in ambient air for the current standard and
4 the additional air quality scenarios; lung function risk approaches that rely, to varying extents, on
5 extrapolating from controlled human exposure study conditions to lower exposure
6 concentrations, lower ventilation rates, and shorter durations; and, characterization of risk for
7 particular population groups that may be at greatest risk, particularly for people with asthma,
8 particularly children. We also recognize several areas in which uncertainty has been reduced by
9 new or updated information or methods, including more refined air quality modeling based on
10 selection of study areas with design values near the current standard and more recent model
11 inputs, as well as updates to several inputs to the exposure model including changes to the
12 exposure duration to better match those in the controlled human exposure studies and an
13 alternate approach to characterizing periods of activity while moderate or greater exertion for
14 simulated individuals.

15 **3.4.5 Public Health Implications**

16 In considering public health implications of the quantitative exposure and risk estimates
17 that may inform the Administrator’s judgments in this area, this section discusses the information
18 pertaining to the following question.

- 19 • **To what extent are the estimates of exposures and risks to at-risk populations**
20 **associated with air quality conditions just meeting the current standard reasonably**
21 **judged important from a public health perspective?**

22 Several factors are important to the consideration of public health implications. These
23 include the magnitude or severity of the effects associated with the estimated exposures, as well
24 as their adversity at the individual and population scale. Other important considerations include
25 the size of the population estimated to experience such effects or to experience exposures
26 associated with such effects. Thus, the discussion here reflects consideration of the health
27 evidence, and exposure and risk estimates, as well as the consideration of potential public health
28 implications in previous NAAQS decisions and ATS policy statements (as also discussed in
29 section 3.3.2).

30 In considering the severity of responses associated with the exposure and risk estimates,
31 we take note of the health effects evidence for the different benchmark concentrations and
32 judgments made with regard to the severity of these effects in the 2015 review. We recognize the
33 greater prevalence of more severe lung function decrements among study subjects exposed to 80
34 ppb or higher concentrations (compared to the study findings for lower exposure concentrations),
35 as well as the prevalence of other effects such as respiratory symptoms; thus, such exposures (of

1 80 ppb and greater) are appropriately considered to be associated with adverse respiratory effects
2 consistent with past and recent ATS position statements and with EPA’s judgments in
3 establishing the current standard in 2015.⁹² Further, in the controlled human exposure study of an
4 average exposure level somewhat above 70 ppb (73 ppb), statistically significant increases in
5 transient lung function decrements (specifically reduced FEV₁) and respiratory symptoms have
6 been reported, leading EPA to also characterize these exposure conditions as being associated
7 with adverse responses, consistent with ATS statements as summarized in section 3.1 above
8 (e.g., 80 FR 65343, 65345, October 26, 2015; 85 FR 87304, December 31, 2020). Studies of
9 controlled human exposures to the lowest benchmark concentration of 60 ppb have found small
10 but statistically significant O₃-related decrements in lung function and airway inflammation
11 (without increased incidence of respiratory symptoms).

12 We additionally take note of the greater significance of estimates for multiple
13 occurrences of exposures at or above these benchmarks consistent with the evidence. This is
14 consistent with past O₃ NAAQS reviews in which it was recognized, using the example of effects
15 such as inflammation, that while isolated occurrences can resolve entirely, repeated occurrences
16 from repeated exposure could potentially result in more severe effects (2013 ISA, section 6.2.3
17 and p. 6-76). The ascribing of greater significance to repeated occurrences of exposures of
18 potential concern is also consistent with public health judgments in NAAQS reviews for other
19 pollutants, such as SO_x and carbon monoxide (84 FR 9900, March 18, 2019; 76 FR 54307,
20 August 31, 2011).

21 The exposure-based analyses include two types of metrics, one involving comparison-to-
22 benchmark concentrations corresponding to 6.6-hour exposure concentrations to which
23 exposures while at elevated ventilation have elicited lung function decrements, and the second
24 involving estimates of lung function risk with regard to such decrements of magnitudes at or
25 above 10%, 15% or 20%. Based on evidence base described in the 2020 ISA, which is largely
26 consistent with that available in the 2015 review (as summarized in section 3.3.1 above), the
27 quantitative exposure and risk analyses results in which we have the greatest confidence are
28 estimates from the comparison-to-benchmarks analysis, as discussed in section 3.4.4 above.

29 In light of the conclusions that people with asthma and children are at-risk populations
30 for O₃-related health effects (summarized in section 3.3.2 above) and the exposure and risk
31 analysis findings of higher exposures and risks for children (in terms of percent of that
32 population), we have focused the discussion here on children, and specifically children with

⁹² The ATS statements indicate that consideration of differences in magnitude or severity, and also the relative transience or persistence of the adverse responses (e.g., FEV₁ changes) and respiratory symptoms, as well as pre-existing sensitivity to effects on the respiratory system, and other factors, is important to characterizing implications for public health effects of an air pollutant such as O₃ (ATS, 2000; Thurston et al., 2017).

1 asthma. We recognize that the exposure and risk estimates indicate that in some areas of the U.S.
2 where O₃ concentrations just meet the current standard, on average across the 3-year period
3 simulated, just over 0.5%, and less than 0.1% of the simulated population of children with
4 asthma might be expected to experience a single day per year with a 7-hour exposure at or above
5 70 ppb and 80 ppb, respectively, while breathing at an elevated rate. With regard to the lowest
6 benchmark considered (60 ppb), the corresponding percentage is just over 8%, with higher
7 percentages in some individual years (Table 3-6). The corresponding estimates for the air quality
8 scenario with higher O₃ concentrations are notably higher (Table 3-5). For example, for the 75
9 ppb air quality scenario, 1.1% to 2.1% of children with asthma, on average across the 3-year
10 design period, are estimated to experience at least one day with exposure concentrations at or
11 above 70 ppb, while at moderate or greater exertion, with as many as 3.9% in a single year
12 (Table 3-5). For the 60 ppb benchmark, the single-day occurrence estimates for the 75 ppb
13 scenario range up to nearly 16%. Estimates for the 65 ppb scenario are appreciably lower.

14 With regard to estimates of lung function decrements, we focus on the E-R model
15 estimates as having less associated uncertainty, as discussed in section 3.4.4 above. The exposure
16 and risk analysis estimates 0.2 to 0.3% of children with asthma, on average across the 3-year
17 design period to experience one or more days with a lung function decrement at or above 20%,
18 and 0.5 to 0.9 % to experience one or more days with a decrement at or above 15% (Table 3-4
19 above). In a single year, the highest estimate is 1.0% of this at-risk population expected to
20 experience one or more days with a decrement at or above 15%. The corresponding estimate for
21 two or more days is 0.6% (Table 3-4 above). As discussed in section 3.4.3 above, the estimates
22 for the 75 ppb air quality scenario are notably higher, while the estimates for the 65 ppb scenario
23 are notably lower (Table 3-5). In reviewing the lung function risk estimates, we note the
24 uncertainties discussed in section 3.4.4 above, including the appreciable portion of these
25 estimates that are based on quantifying risk for exposure concentrations below those studied.

26 The size of the at-risk population (people with asthma, particularly children) in the U.S.
27 is substantial. As summarized in section 3.3.2, nearly 8% of the total U.S. population⁹³ and 7.0%
28 of U.S. children have asthma. The asthma prevalence in U.S. child populations (younger than 18
29 years) of different races or ethnicities ranges from 7.5% for all Hispanic children to 13.5% for
30 black non-Hispanic children (Table 3-1 above). This is well reflected in the exposure and risk
31 analysis study areas in which the asthma prevalence ranged from 7.7% to 11.2% of the total
32 populations and 9.2% to 12.3% of the children. In each study area, the prevalence varies among

⁹³ The number of people in the US with asthma is estimated to be about 25 million. As shown in Table 3-1 the estimated number of people with asthma was 25,131,000 in 2019.

1 census tracts, with the highest tract having a prevalence in boys of 25.5% and a prevalence in
2 girls of 17.1% (Appendix 3D, Table 3D-3).

3 The exposure and risk analyses inherently recognize that variability in human activity
4 patterns (where people go and what they do) is key to understanding the magnitude, duration,
5 pattern, and frequency of population exposures. For O₃ in particular, the amount and frequency
6 of afternoon time outdoors at moderate or greater exertion is an important factor for
7 understanding the fraction of the population that might experience O₃ exposures that have
8 elicited respiratory effects in controlled human exposure studies (2014 HREA, section 5.4.2). In
9 considering the available information regarding prevalence of behavior (time outdoors and
10 exertion levels) and daily temporal pattern of O₃ concentrations, we take note of the findings of
11 evaluations of the data in the CHAD. Based on these evaluations of human activity pattern data,
12 it appears that children and adults both, on average, spend about 2 hours of afternoon time
13 outdoors per day, but differ substantially in their participation in these events at elevated exertion
14 levels (rates of about 80% versus 60%, respectively) (2014 HREA, section 5.4.1.5), indicating
15 children are more likely to experience exposures that may be of concern. This is one basis for
16 their identification as an at-risk population for O₃-related health effects. The human activity
17 pattern evaluations have also shown there is little to no difference in the amount or frequency of
18 afternoon time outdoors at moderate or greater exertion for people with asthma compared with
19 those who do not have asthma (2014 HREA, section 5.4.1.5). Further, recent CHAD analyses
20 indicate that while 46 – 73% of people do not spend any afternoon time outdoors at moderate or
21 greater exertion, a fraction of the population (i.e., between 5.5 – 6.8% of children) spend more
22 than 4 hours per day outdoors at moderate or greater exertion and may have greater potential to
23 experience exposure events of concern than adults (Appendix 3D, section 3D.2.5.3 and Figure
24 3D-9). It is this potential that contributes importance to consideration of the exposure and risk
25 estimates.

26 In considering the public health implications of the exposure and risk estimates across the
27 eight study areas, we note the purpose for the study areas is to illustrate exposure circumstances
28 that may occur in areas that just meet the current standard, and not to estimate exposure and risk
29 associated with conditions occurring in those specific locations today. To the extent that
30 concentrations in the specific areas simulated may differ from others across the U.S., the
31 exposure and risk estimates for these areas are informative to consideration of potential
32 exposures and risks in areas existing across the U.S. that have air quality and population
33 characteristics similar to the study areas assessed, and that have ambient concentrations of O₃
34 that just meet the current standard today or that will be reduced to do so at some period in the
35 future. We note that numerous areas across the U.S. have air quality for O₃ that is near or above

1 the existing standard.⁹⁴ Thus, the air quality and exposure circumstances assessed in the eight
2 study areas are of particular importance in considering whether the available information calls
3 into question the adequacy of public health protection afforded by the current standard.

4 The exposure and risk estimates for the eight study areas reflect differences in exposure
5 circumstances among those areas and illustrate the exposures and risks that might be expected to
6 occur in other areas with such circumstances under air quality conditions that just meet the
7 current standard (or the alternate conditions assessed). Thus, the exposure and risk estimates
8 indicate the magnitude of exposure and risk that might be expected in many areas of the U.S.
9 with O₃ concentrations at or near the current standard. Although the methodologies and data used
10 to estimate population exposure and lung function risk in this assessment differ in several ways
11 from what was used in the 2015 review, the findings and considerations summarized here present
12 a pattern of exposure and risk that is generally similar to that considered in the last review (as
13 described in section 3.4.2 above), and indicate a level of protection generally consistent with that
14 described in the 2015 decision.

15 In summary, the considerations raised here are important to conclusions regarding the
16 public health significance of the exposure and risk results. We recognize that such conclusions
17 also depend in part on public health policy judgments that weigh in the Administrator's decision
18 regarding the protection afforded by the current standard. Such judgments that are common to
19 NAAQS decisions include those related to public health implications of effects of differing
20 severity (75 FR 355260 and 35536, June 22, 2010; 76 FR 54308, August 31, 2011; 80 FR 65292,
21 October 26, 2015). Such judgments also include those concerning the public health significance
22 of effects at exposures for which evidence is limited or lacking, as discussed in section 3.4.4
23 above, such as effects at the lower benchmark concentrations considered and lung function risk
24 estimates associated with exposure concentrations lower than those tested or for population
25 groups not included in the controlled exposure studies.

26 **3.5 KEY CONSIDERATIONS REGARDING THE CURRENT PRIMARY** 27 **STANDARD**

28 In considering what the available evidence and exposure/risk information indicate with
29 regard to the current primary O₃ standard, the overarching question we consider is:

⁹⁴ Based on data from 2016-2018, 142 counties have O₃ concentrations that exceed the current standard. Population size in these counties ranges from approximately 20,000 to more than ten million, with a total population of over 112 million living in counties that exceed the current standard. Air quality data are from Table 4. Monitor Status in the Excel file labeled *ozone_designvalues_20162018_final_06_28_19.xlsx* downloaded from <https://www.epa.gov/air-trends/air-quality-design-values>. Population sizes are based on 2017 estimates from the U.S. Census Bureau (<https://www.census.gov/programs-surveys/popest.html>).

- 1 • **Does the available scientific evidence- and exposure/risk-based information support**
2 **or call into question the adequacy of the protection afforded by the current primary**
3 **O₃ standard?**

4 To assist us in interpreting the available scientific evidence and the results of recent
5 quantitative exposure/risk analyses to address this question, we have focused on a series of more
6 specific questions, as detailed in sections 3.5.1 and 3.5.2 below. In considering the scientific and
7 technical information, we take into account the information available at the time of the 2015
8 review and information newly available in the 2020 review, which have been critically analyzed
9 and characterized in the 2013 ISA for the 2015 review and the ISA for the 2020 review,
10 respectively. In this context, a primary consideration is whether the available information alters
11 overall prior conclusions regarding health effects associated with photochemical oxidants,
12 including O₃, in ambient air.

13 **3.5.1 Evidence-based Considerations**

14 In considering the evidence with regard to the overarching question posed above
15 regarding the adequacy of the current standard, we address a series of more specific questions
16 that focus on policy-relevant aspects of the evidence. These questions begin with consideration
17 of the available evidence on health effects associated with exposure to photochemical oxidants,
18 and particularly O₃.

- 19 • **Is there evidence that indicates the importance of photochemical oxidants other than**
20 **O₃ with regard to abundance in ambient air, and potential for human exposures and**
21 **health effects?**

22 The 2020 ISA did not identify any newly available evidence regarding the importance of
23 photochemical oxidants other than O₃ with regard to abundance in ambient air, and potential for
24 health effects.⁹⁵ As summarized in section 2.1 above, O₃ is one of a group of photochemical
25 oxidants formed by atmospheric photochemical reactions of hydrocarbons with nitrogen oxides
26 in the presence of sunlight, with O₃ being the only photochemical oxidant other than nitrogen
27 dioxide that is routinely monitored in ambient air. Data for other photochemical oxidants are
28 generally derived from a few special field studies such that national scale data for these other
29 oxidants are scarce (ISA, Appendix 1, section 1.1; 2013 ISA, sections 3.1 and 3.6). Moreover,
30 few studies of the health impacts of other photochemical oxidants beyond O₃ have been
31 identified by literature searches conducted for other recent O₃ assessments (ISA, Appendix 1,
32 section 1.1). As stated in the ISA, “the primary literature evaluating the health...effects of

⁹⁵ Close agreement between past O₃ measurements and the photochemical oxidant measurements upon which the early photochemical oxidants NAAQS was based indicated the very minor contribution of other oxidant species in comparison to O₃ (U.S. DHEW, 1970).

1 photochemical oxidants includes ozone almost exclusively as an indicator of photochemical
2 oxidants” (ISA, section IS.1.1, p. IS-3). Thus, the evidence base for health effects of
3 photochemical oxidants does not indicate an importance of any other photochemical oxidants.
4 For these reasons, discussion of photochemical oxidants in this document focuses on O₃.

5 • **Does the available scientific evidence alter prior conclusions regarding the nature of**
6 **health effects attributable to human exposure to O₃ from ambient air?**

7 The evidence, as evaluated in the 2020 ISA, is largely consistent with the conclusion in
8 the last ISA (in the 2015 review) regarding the health effects causally related to O₃ exposures,
9 and most specifically regarding respiratory effects, which, as in the past, are concluded to be
10 causally related to short-term exposures to O₃. Also, as in the 2015 and prior reviews, respiratory
11 effects are concluded to be likely causally related to longer-term O₃ exposures (ISA, section
12 IS.1.3.1, Appendix 3). Further, while a causal determination was not made in the 2015 review
13 regarding metabolic effects, the 2020 ISA finds there to be sufficient evidence to conclude there
14 to likely be a causal relationship of short-term O₃ exposures and metabolic effects and finds the
15 evidence to be suggestive of, but not sufficient to infer, such a relationship between long-term O₃
16 exposure and metabolic effects (ISA, section IS.1.3.1). This is based on more recently available
17 evidence, largely from experimental animal studies, on these effects (ISA, Appendix 5).
18 Additionally, the EPA’s causal determinations regarding cardiovascular effects and mortality
19 have been updated from what they were in 2013 ISA based on more recently available evidence
20 in combination with uncertainties that had been identified in the previously available evidence
21 (ISA, Appendix 4, section 4.1.17 and Appendix 6, section 6.1.8). The EPA has concluded that
22 the evidence base is suggestive of, but not sufficient to infer, causal relationships between O₃
23 exposures (short- and long-term) and cardiovascular effects, mortality, reproductive and
24 developmental effects, and nervous system effects (ISA, section IS.1.3.1). As in the 2015 and
25 prior O₃ NAAQS reviews, the strongest evidence, including with regard to characterization of
26 relationships between O₃ exposure and occurrence and magnitude of effects, is for respiratory
27 effects, and particularly for effects such as lung function decrements, respiratory symptoms,
28 airway responsiveness, and respiratory inflammation.

29 • **Does the available evidence alter our prior understanding of populations that are**
30 **particularly at risk from O₃ exposures?**

31 The evidence, as evaluated in the 2020 ISA, does not alter our prior understanding of
32 populations at risk from health effects of O₃ exposures. As in the past, people with asthma, and
33 particularly children, are the at-risk population groups for which the evidence is strongest. In
34 addition to populations with asthma, groups with relatively greater exposures, particularly those
35 who spend more time outdoors during times when ambient air concentrations of O₃ are highest

1 and while engaged in activities that result in elevated ventilation, are recognized as at increased
2 risk. Such groups include outdoor workers and children. Other groups for which the evidence is
3 less clear include older adults, individuals with reduced intake of certain nutrients and
4 individuals with certain genetic variants. Recent evidence does not provide additional
5 information for these groups beyond the evidence available at the time of the 2015 review (ISA,
6 section IS.4.4).

- 7 • **Does the available evidence alter past conclusions regarding the exposure duration
8 and concentrations associated with health effects? To what extent does the scientific
9 evidence indicate health effects attributable to exposures to O₃ concentrations lower
10 than previously reported and what are important uncertainties in that evidence?**

11 The available evidence documented in the 2020 ISA regarding O₃ exposures associated
12 with health effects is largely similar to that available at the time of the 2015 review and does not
13 indicate effects attributable to exposures of shorter duration or lower concentrations than
14 previously understood. Respiratory effects continue to be the effects for which the experimental
15 information regarding exposure concentrations eliciting effects is well established, as
16 summarized in section 3.3.3 above. Such information allows for characterization of potential
17 population risk associated with O₃ in ambient air under conditions allowed by the current
18 standard. The more recently available controlled human exposure studies, as discussed in section
19 3.3.3 above, are conducted over shorter durations while at much higher concentrations than the
20 key set of 6.6-hour studies that have been the focus of the last several reviews. The respiratory
21 effects evidence includes support from a large number of epidemiologic studies. The positive
22 associations of O₃ with respiratory health outcomes (e.g., asthma-related hospital admissions and
23 emergency department visits) reported in these studies are coherent with findings from the
24 controlled human exposure and experimental animal studies. All but a few of these studies,
25 however, are conducted in areas during periods when the current standard is not met, making
26 them less useful with regard to indication of health effects of exposures allowed by the current
27 standard.

28 Within the evidence base for the recently identified category of metabolic effects, the
29 evidence derives largely from experimental animal studies of exposures appreciably higher than
30 those for the 6.6-hour human exposure studies along with a small number of epidemiologic
31 studies. As discussed in section 3.3.3 above, these studies do not prove to be informative to our
32 consideration of exposure circumstances likely to elicit health effects.

33 Thus, the 6.6-hour controlled human exposure studies of respiratory effects remain the
34 focus for our consideration of exposure circumstances associated with O₃ health effects. Based
35 on these studies, the exposure concentrations investigated range from as low as approximately 40
36 ppb to 120 ppb. This information on concentrations that have been found to elicit effects for 6.6-

1 hour exposures while exercising is unchanged from what was available in the 2015 review. The
2 lowest concentration for which lung function decrements have been found to be statistically
3 significantly increased over responses to filtered air remains approximately 60 ppb, at which
4 group mean decrements on the order of 2% to 4% have been reported (Table 3-2, Figure 3-2).
5 Respiratory symptoms were not increased with this exposure level.⁹⁶ Exposure to concentrations
6 slightly above 70 ppb, with quasi-continuous exercise, has been reported to elicit statistically
7 significant increases in both lung function decrements and respiratory symptom scores, as
8 summarized in section 3.3.3 above. Still greater group mean and individual responses in lung
9 function decrements and respiratory symptom scores, as well as inflammatory response and
10 airway responsiveness, are reported for higher exposure concentrations.

11 • **To what extent have previously identified uncertainties in the health effects evidence**
12 **been reduced or do important uncertainties remain?**

13 Uncertainties identified in the health effects evidence at the time of the 2015 review
14 generally remain. These include uncertainties related to the susceptibility of population groups
15 not studied, the potential for effects to result from exposures to concentrations below those
16 included in controlled human exposure studies, and the potential for increased susceptibility as a
17 result of prior exposures. We additionally recognize uncertainties associated with the
18 epidemiologic studies (e.g., the potential for copollutant confounding and exposure measurement
19 error). In this context, however, we note the appreciably greater strength in the epidemiologic
20 evidence in its support for determination of a causal relationship for respiratory effects than the
21 epidemiologic evidence related to other categories, such as metabolic effects, more recently
22 determined to have a likely causal relationship with short-term O₃ exposures (as summarized in
23 section 3.3.1 above).

24 **3.5.2 Exposure/risk-based Considerations**

25 Our consideration of the scientific evidence is informed by results from a quantitative
26 analysis of estimated population exposure and associated risk, as at the time of the 2015 review.
27 The overarching consideration in this section is whether the current exposure/risk information
28 alters overall conclusions of the 2015 review regarding health risk associated with exposure to
29 O₃ in ambient air. As in our consideration of the evidence in section 3.3.1 above, we have
30 focused the discussion regarding the exposure/risk information around key questions to assist us
31 in considering the exposure/risk analyses of at-risk populations living in a set of urban areas

⁹⁶A statistically significant increase in sputum neutrophils (a marker of increased airway inflammation) was observed in one controlled human exposure study following 6.6-hour exposures to 60 ppb (Table 3-2, Figure 3-2; Appendix 3A).

1 under air quality conditions simulated to just meet the existing primary O₃ standard. These
2 questions are as follows.

- 3 • **To what extent are the estimates of exposures and risks to at-risk populations**
4 **associated with air quality conditions just meeting the current standard reasonably**
5 **judged important from a public health perspective? What are the important**
6 **uncertainties associated with any exposure/risk estimates?**

7 The exposure and risk analyses conducted for the 2020 review, as described in section
8 3.4, provide exposure and risk estimates associated with air quality that might occur in an area
9 under conditions that just meet the current standard. These estimates illustrate the differences
10 likely to occur across various locations with such air quality as a result of area-specific
11 differences in emissions, meteorological and population characteristics. In understanding these
12 results, we note that the eight study areas provide a variety of circumstances with regard to
13 population exposure to concentrations of O₃ in ambient air. These study areas reflect different
14 combinations of different types of sources of O₃ precursor emissions, and also illustrate different
15 patterns of exposure to O₃ concentrations in a populated area in the U.S. (Appendix 3C, section
16 3C.2). In this way, the eight areas provide a variety of examples of exposure patterns that can be
17 informative to the EPA's consideration of potential exposures and risks that may be associated
18 with air quality conditions occurring under the current O₃ standard. While the same conceptual
19 air quality scenario is simulated in all eight study areas (i.e., conditions that just meet the existing
20 standard), variability in emissions patterns of O₃ precursors, meteorological conditions, and
21 population characteristics in the study areas contribute to variability in the estimated magnitude
22 of exposure and associated risk across study areas.

23 In considering the exposure and risk results, we focus first on estimates for the eight
24 study areas from the comparison-to-benchmarks analysis, the results in which we have the
25 greatest confidence, as discussed in section 3.4.4 above. These results for urban areas with air
26 quality that just meets the current standard indicate that up to 0.7% of children with asthma, on
27 average across the 3-year period, and up to 1.0% in a single year might be expected to
28 experience, while at elevated exertion, at least one day with a 7-hour average O₃ exposure
29 concentration at or above 70 ppb (Table 3-3). As noted earlier, this benchmark concentration
30 reflects the finding of statistically significant O₃-related decrements and increased respiratory
31 symptoms in a controlled human exposure study of individuals at elevated exertion. Less than
32 0.1% of this population group is estimated to have multiple days with an occurrence of this
33 exposure level (Table 3-3). For the benchmark concentration of 80 ppb (which reflects the
34 potential for more severe effects), a much lower percentage of children with asthma, <0.1% on
35 average across the 3-year period, with 0.1% in the highest single year, might be expected to
36 experience, while at elevated exertion, at least one day with such a concentration (Table 3-3).

1 There are no children with asthma estimated to experience more than a single day per year with a
2 7-hour average O₃ concentration at or above 80 ppb (Table 3-3). With regard to the lowest
3 benchmark concentration of 60 ppb, 8.8% of children with asthma, on average across the 3-year
4 period, might be expected to experience one or more days with a 7-hour average O₃ exposure
5 concentration at or above 60 ppb (the concentration associated with less severe effects), and just
6 over 11% in the highest single year (Table 3-3). Regarding multiple day occurrences, the
7 percentages for more than a single day occurrence are 3%, on average across the three years, and
8 just below 5% in the highest single year period (Table 3-3).

9 The estimates for the additional air quality scenarios differ as would be expected. For the
10 75 ppb air quality scenario, the percent of children with asthma that might be expected to
11 experience at least one day with a 7-hour average O₃ exposure concentration, while at elevated
12 exertion, at or above 70 ppb, is a factor of three or more higher than for the current standard
13 (Table 3-3, Table 3-5). The corresponding estimates for multiple days are a factor of four or
14 more higher than those for air quality just meeting the current standard. By comparison,
15 corresponding estimates for the 65 ppb scenario are approximately a third those for the current
16 standard scenario, with a correspondingly smaller incremental difference in absolute number of
17 children (Table 3-3, Table 3-5). With regard to the 80 ppb benchmark, the difference of the 75
18 ppb scenario from the current standard is a factor of three (for average across the 3-year period)
19 to six (for the highest in a single year) (Table 3-3, Table 3-5). In contrast, the estimates for the 80
20 ppb benchmark (which is associated with the more severe effects) in the 65 ppb air quality
21 scenario are nearly identical to those for the current standard (Table 3-3, Table 3-5).

22 With regard to the estimates of lung function risk, as an initial matter we note the
23 uncertainty associated with these estimates, as discussed in section 3.4.4 above. In this context,
24 we also recognize the lesser uncertainty associated with estimates derived using the E-R function
25 (in comparison to estimates based on MSS model). Accordingly, it is those estimates which we
26 consider here for air quality conditions just meeting the current standard. The E-R lung function
27 risk analysis for the eight study areas indicates that the percent of children with asthma in an
28 urban area that just meets the current standard that might be expected to experience one or more
29 days with a lung function decrement of at least 15% or 20% might range up 0.9% or 0.3%,
30 respectively, on average across the three years, and 1.0% or 0.4%, respectively, in a single-year
31 period (Table 3-4). The estimates for a day with a decrement of at least 10% might range up to
32 3.3%, on average across the three years, and just over 3.5% in a single-year period (Table 3-4).
33 With regard to multiple day occurrences, the percent of children with asthma that might be
34 expected to experience two or more days with a lung function decrement of at least 10% may be
35 as high as 2.4%, on average across the three years, and 2.6% in a single year (Table 3-4), with
36 much smaller percentages for larger decrements. For multiple days with a decrement of at least

1 15% or 20%, the corresponding percentages are much lower, 0.6% or 0.2%, respectively, on
2 average across the three years, and 0.6% or 0.2%, respectively, in a single year period (Table 3-
3 4).

4 We also consider the estimates from this assessment in light of the estimates from the
5 2014 HREA that were a focus of the decision on the standard in 2015. The estimates across all
6 study areas from this assessment are generally similar to those reported in the 2015 review across
7 all study areas included in that HREA, particularly for the two or more occurrences and for the
8 80 ppb benchmark (Table 3-8).⁹⁷ In our consideration here, we focus on the full array of study
9 areas (e.g., rather than limiting to areas common to the two assessments) given the purpose of the
10 assessments in providing estimates across a range of study areas to inform decision making with
11 regard to the exposures and risks that may occur across the U.S. in areas that just meet the
12 current standard. We note only slight differences, particularly for the lower benchmarks, and
13 most particularly in the estimates for the highest year. For example, for the 70 ppb benchmark,
14 the lower and higher end of the range of average per year percent of children with at least one
15 day above the benchmark from the 2014 HREA are both twice the corresponding values from the
16 current assessment (Table 3-8). Consideration of the percentage of children estimated to
17 experience a day or more with an exposure at or above 70 ppb across the three air quality
18 conditions in the two assessments, however, indicates that differences between air quality
19 scenarios in the current assessment remain appreciably larger than the slight differences in
20 estimates between the two assessments for a given scenario. The factors likely contributing to the
21 slight differences between the two assessments, such as for the lowest benchmark, include
22 greater variation in ambient air concentrations in some of the study areas in the 2014 HREA, as
23 well as the lesser air quality adjustments required in study areas for the current assessment due to
24 closer proximity of conditions to meeting the current standard (70 ppb).⁹⁸ Other important
25 differences between the two assessments are the updates made to the ventilation rates used for
26 identifying when a simulated individual is at moderate or greater exertion and the use of 7 hours
27 for the exposure duration. Both of these changes were made to provide closer linkages to the
28 conditions of the controlled human exposure studies which are the basis for the benchmark
29 concentrations. Thus, we recognize there to be reduced uncertainty associated with the current
30 estimates. Overall, particularly in light of differences in the assessments, we conclude the current

⁹⁷ For consistency with the estimates highlighted in the 2015 review, Table 3-8 focuses on the simulated population of all children (*versus* the simulated population for children with asthma that are the focus in section 3.4).

⁹⁸ The 2014 HREA air quality scenarios involved adjusting 2006-2010 ambient air concentrations, and some study areas had design values in that time period that were well above the then-existing standard (and more so for the current standard). Study areas included the current exposure analysis had 2015-2017 design values close to the current standard, requiring less of an adjustment for the current standard (70 ppb) air quality scenario.

1 estimates to be generally similar to those which were the focus in the 2015 decision on
 2 establishing the current standard.

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Table 3-8. Comparison of current assessment and 2014 HREA (all study areas) for percent of children estimated to experience at least one, or two, days with an exposure at or above benchmarks while at moderate or greater exertion.

Air Quality Scenario (DVC, ppb)	Estimated average % of simulated children with <u>at least one day</u> per year at or above benchmark (highest in single season)		Estimated average % of simulated children with <u>at least two days</u> per year at or above benchmark (highest in single season)	
	<i>Current PA^A</i>	<i>2014 HREA^B</i>	<i>Current PA^A</i>	<i>2014 HREA^B</i>
<i>Benchmark Exposure Concentration of 80 ppb</i>				
75	<0.1 ^A – 0.3 (0.6)	0 – 0.3 (1.1)	0 – <0.1 (<0.1)	0 (0.1)
70	0 – <0.1 (0.1)	0 – 0.1 (0.2)	0 (0)	0 (0)
65	0 – <0.1 (<0.1)	0 (0)	0 (0)	0 (0)
<i>Benchmark Exposure Concentration of 70 ppb</i>				
75	1.1 – 2.0 (3.4)	0.6 – 3.3 (8.1)	0.1 – 0.3 (0.7)	0.1 – 0.6 (2.2)
70	0.2 – 0.6 (0.9)	0.1 – 1.2 (3.2)	<0.1 (0.1)	0 – 0.1 (0.4)
65	0 – 0.2 (0.2)	0 – 0.2 (0.5)	0 – <0.1 (<0.1)	0 (0)
<i>Benchmark Exposure Concentration of 60 ppb</i>				
75	6.6 – 15.7 (17.9)	9.5 – 17.0 (25.8)	1.7 – 8.0 (9.9)	3.1 – 7.6 (14.4)
70	3.2 – 8.2 (10.6)	3.3 – 10.2 (18.9)	0.6 – 2.9 (4.3)	0.5 – 3.5 (9.2)
65	0.4 – 2.3 (3.7)	0 – 4.2 (9.5)	<0.1 – 0.3 (0.5)	0 – 0.8 (2.8)
^A For the current analysis, calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1" ^B For the 2014 HREA, calculated percent was rounded to the nearest tenth decimal using conventional rounding. Values that did not round upwards to 0.1 (i.e., <0.05) were given a value of "0". ^C The monitor location with the highest concentrations in each area had a design value just equal to the indicated value.				

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3.5.3 Preliminary Conclusions on the Primary Standard

This section describes our preliminary conclusions for the Administrator’s consideration with regard to the current primary O₃ standard. These preliminary conclusions are based on considerations described in the sections above, and in the discussion below regarding the available scientific evidence (as summarized in the 2020 ISA, and the ISA and AQCDs from prior reviews), and the risk and exposure information developed in the 2020 review and summarized in section 3.4 above. Taking into consideration the discussions above in this chapter, this section addresses the following overarching policy question.

- 1 • **Do the available scientific evidence- and exposure/risk-based information support or**
2 **call into question the adequacy of the protection afforded by the current primary O₃**
3 **standard?**

4 In considering this question, we recognize that, as is the case in NAAQS reviews in
5 general, the extent to which the protection provided by the current primary O₃ standard is judged
6 to be adequate will depend on a variety of factors, including science policy judgments and public
7 health policy judgments. These factors include public health policy judgments concerning the
8 appropriate benchmark concentrations on which to place weight, as well as judgments on the
9 public health significance of the effects that have been observed at the exposures evaluated in the
10 health effects evidence. The factors relevant to judging the adequacy of the standards also
11 include the interpretation of, and decisions as to the weight to place on, different aspects of the
12 results of the quantitative exposure risk analyses and the associated uncertainties. Thus, we
13 recognize that the Administrator’s conclusions regarding the adequacy of the current standard
14 will depend in part on public health policy judgments, science policy judgments, including those
15 regarding aspects of the evidence and exposure/risk estimates, and judgments about the degree of
16 protection that is requisite to protect public health with an adequate margin of safety.

17 Our response to the overarching question above takes into consideration the discussions
18 that address the specific policy-relevant questions in prior sections of this document (see section
19 3.2) and builds on the approach from previous reviews. We focus first on consideration of the
20 evidence, including that newly available in the 2020 ISA, including the extent to which it alters
21 prior key conclusions supporting the current standard. We then turn to consideration of the
22 quantitative exposure and risk estimates developed for the 2020 review, including associated
23 limitations and uncertainties. We consider what they indicate regarding the level of protection
24 from adverse effects provided by the current standard, as well as the extent to which
25 exposure/risk estimates may indicate differing conclusions regarding air quality conditions
26 associated with the current standard from those based on past assessments. We additionally
27 consider the key aspects of the evidence and exposure/risk estimates emphasized in establishing
28 the current standard, and the associated public health policy judgments and judgments about the
29 uncertainties inherent in the scientific evidence and quantitative analyses that are integral to
30 decisions on the adequacy of the current primary O₃ standard.

31 As an initial matter, we recognize the continued support in the available evidence for O₃
32 as the indicator for photochemical oxidants, as recognized in section 3.5.1 above. Of the
33 photochemical oxidants, O₃ is the only one other than nitrogen dioxide (for which there are
34 separate NAAQS) that is routinely monitored in ambient air. Further, as stated in the ISA, “the
35 primary literature evaluating the health and ecological effects of photochemical oxidants
36 includes ozone almost exclusively as an indicator of photochemical oxidants” (ISA, section

1 IS.1.1, p. IS-3). In summary, the evidence base for health effects of photochemical oxidants does
2 not indicate an importance of any other photochemical oxidants as it includes O₃ almost
3 exclusively as an indicator of photochemical oxidants, thus continuing to support the
4 appropriateness of O₃ as the indicator for photochemical oxidants.

5 In considering the extensive evidence base for health effects of O₃, we give particular
6 attention to the longstanding evidence of respiratory effects causally related to O₃ exposures.
7 This array of effects, and the underlying evidence base, was integral to the basis for setting the
8 current standard in 2015. As summarized in section 3.3.1 above and addressed in detail in the
9 ISA, the available evidence base does not include new evidence of respiratory effects associated
10 with appreciably different exposure circumstances, including any that would be expected to
11 occur under air quality conditions associated with the current standard. Thus, in considering the
12 information available at this time, we continue to focus on exposure circumstances associated
13 with the current standard as those of importance in this reconsideration.

14 Further, while the evidence base has been augmented somewhat since the 2015 review,
15 we note that the newly available evidence does not lead to different conclusions regarding the
16 respiratory effects of O₃ in ambient air or regarding exposure concentrations associated with
17 those effects; nor does it identify different populations at risk of O₃-related effects. For example,
18 as in the 2015 review, people of all ages with asthma, children, and outdoor workers, are
19 populations at increased risk of respiratory effects related to O₃ in ambient air. Children with
20 asthma, which number approximately five million in the U.S., may be particularly at risk (section
21 3.3.2 and Table 3.1).⁹⁹ In these ways, the health effects evidence is consistent with evidence
22 available in the 2015 review when the current standard was established. This strong evidence
23 base continues to demonstrate a causal relationship between short-term O₃ exposures and
24 respiratory effects, including in people with asthma. This conclusion is primarily based on
25 evidence from controlled human exposure studies that was available at the time the standard was
26 set that reported lung function decrements and respiratory symptoms in people exposed to O₃ for
27 6.6 hours during which they engage in five hours of exercise. Support is also provided by the
28 experimental animal and epidemiologic evidence that is coherent with the controlled exposure
29 studies. The epidemiologic evidence, including that recently available, includes studies reporting
30 positive associations for asthma-related hospital admissions and emergency department visits,
31 which are strongest for children, with short-term O₃ exposures. Based collectively on this
32 evidence, populations identified as at risk of such effects include people with asthma and
33 children.

⁹⁹ The size of the U.S. population with asthma is approximately 25 million.

1 As in the 2015 review, the most certain evidence of health effects in humans elicited by
2 exposures to specific O₃ exposure concentrations is provided by controlled human exposure
3 studies. This category of short-term studies includes an extensive evidence base of 1- to 3-hour
4 studies, conducted with continuous or intermittent exercise and generally involving relatively
5 higher exposure concentrations (e.g., greater than 120 ppb).¹⁰⁰ Given the lack of ambient air
6 concentrations of this magnitude in areas meeting the current standard (see section 2.4.1 above
7 and Appendix 2A), we continue to focus primarily on a second group of somewhat longer-
8 duration studies of much lower exposure concentrations. These studies employ a 6.6-hour
9 protocol that includes six 50-minute periods of exercise at moderate or greater exertion. There
10 are no new such studies with exercise available since the 2015 review. Thus, the newly available
11 evidence does not extend our understanding of the range of exposure concentrations that elicit
12 effects in such studies beyond what was understood previously.

13 Similarly, as in the 2015 review, 60 ppb remains the lowest exposure concentration
14 (target concentration, as average across exercise periods) at which statistically significant lung
15 function decrements have been reported in the 6.6-hour exposure studies. Two studies have
16 assessed exposure concentrations at the lower concentration of 40 ppb, with no statistically
17 significant finding of O₃-related FEV₁ decrements for the group mean in either study (which is
18 just above 1% in one study, and well below in the second). At 60 ppb, the group mean O₃-related
19 decrement in FEV₁ ranges from approximately 2 to 4%, with associated individual study subject
20 variability in decrement size. In the single study assessing the next highest exposure
21 concentration (just above 70 ppb, at 73 ppb),¹⁰¹ the group mean FEV₁ decrement (6%) was also
22 statistically significant, as were respiratory symptom scores. At higher exposure concentrations,
23 the incidence of both respiratory symptom scores and O₃-related lung function decrements in the
24 study subjects is increased. Other respiratory effects, such as inflammatory response and airway
25 resistance are also increased at higher exposures (ISA; 2013 ISA; 2006 AQCD).

26 In considering what may be indicated by the epidemiologic evidence with regard to
27 exposure concentrations eliciting effects, we recognize that of the numerous epidemiologic
28 studies of respiratory outcome associations with O₃ in ambient air, none were conducted in U.S.

¹⁰⁰ Table 3A-3 in Appendix 3 summarizes controlled human exposures to O₃ for 1 to 2 hours during continuous or intermittent exercise in contrast to similar exposure durations at rest. This table was adapted from Table 7-1 in the 1996 AQCD and Table AX6-1 in the 2006 CD, with additional studies from Table AX6-13 in the 2006 AQCD, as well as more recent studies from the 2013 ISA and the ISA.

¹⁰¹ As noted in sections 3.1.1 and 3.3.3 above, the 70 ppb target exposure comes from Schelegle et al. (2009). That study reported, based on O₃ measurements during the six 50-minute exercise periods, that the mean O₃ concentration during the exercise portion of the study protocol was 72 ppb. Based on the measurements for the six exercise periods, the time weighted average concentration across the full 6.6-hour exposure was 73 ppb (Schelegle et al., 2009).

1 locations during time periods when the current standard was met. In fact, the vast majority of
2 these studies were conducted in locations and during time periods that would not have met the
3 current standard, thus making them less useful for considering the potential for O₃ concentrations
4 allowed by the current standard to contribute to health effects. While there were a handful of
5 multi-city studies in which the O₃ concentrations in a subset of the study locations and for a
6 portion of the study period appear to have met the current standard, data were not available in
7 some cities for the earlier years of the study period when design values for other cities were well
8 above 70 ppb (as discussed in section 3.3.3). We recognize that the study analyses and
9 associations reported were based on the combined dataset across the full time period (and, for
10 multicity studies, from all cities), and the extent to which risk associated with exposures derived
11 from the concentrations in the subset of years (and locations) that would have met the current
12 standard compared to that from the years (and locations) that would have violated the standard
13 influenced the study findings is not clear. There were no studies conducted in U.S. locations with
14 ambient air O₃ concentrations that would meet the current standard for the entire duration of the
15 study (i.e., with design values¹⁰² at or below 70 ppb). Thus, the epidemiologic studies provide
16 limited insight regarding exposure concentrations associated with health outcomes that might be
17 expected under air quality conditions that meet the current standard (section 3.3.3 above). Thus,
18 the studies of 6.6-hour exposures with quasi-continuous exercise, and particularly those for
19 concentrations ranging from 60 to 80 ppb continue to provide an appropriate focus in this
20 reconsideration.

21 As in the 2015 review, we recognize some uncertainty, reflecting limitations in the
22 evidence base, with regard to the exposure levels eliciting effects as well as the severity of the
23 effects in some population groups not included in the available controlled human exposure
24 studies, such as children and individuals with asthma. Further, we note uncertainty in the extent
25 or characterization of effects at exposure levels below those studied. In this context, we
26 recognize that the controlled human exposure studies, primarily conducted in healthy adults, on
27 which the depth of our understanding of O₃-related health effects is based, provide limited, but
28 nonetheless important information with regard to responses in people with asthma or in children.
29 We also note that the evidence indicates that responses such as those observed in the controlled
30 human exposure studies, if repeated or sustained, particularly in people with asthma, can pose
31 risks of effects of greater concern, including asthma exacerbation. We also take note of
32 statements from the ATS, and judgments made by the EPA in considering similar effects in past
33 NAAQS reviews (80 FR 65343, October 26, 2015; 85 FR 87302, December 31, 2020). In so

¹⁰² As described in chapter 2, a design value is the metric used to describe air quality in a given area relative to the level of the standard, taking the averaging time and form into account. For example, a design value of 70 ppb just meets the current primary standard.

1 doing, we recognize the role of such statements in proposing principles or considerations for
2 weighing the evidence rather than offering “strict rules or numerical criteria” (ATS, 2000;
3 Thurston et al., 2017).

4 The more recent statement is generally consistent with the prior (2000) statement, that
5 was considered in the 2015 O₃ NAAQS review, including the attention that statement gives to at-
6 risk or vulnerable population groups, while also broadening the discussion of effects, responses,
7 and biomarkers to reflect the expansion of scientific research in these areas. One example of this
8 increased specificity is in the discussion of small changes in lung function (in terms of FEV₁) in
9 people with compromised function, such as people with asthma (Thurston et al., 2017). We note
10 that, in keeping with the intent of these statements to avoid specific criteria, the statements, in
11 discussing what constitutes an adverse health effect, do not comprehensively describe all the
12 biological responses raised, e.g., with regard to magnitude, duration or frequency of small
13 pollutant-related changes in lung function. These concepts, including the consideration of the
14 magnitude of effects occurring in just a subset of study subjects, continue to be recognized as
15 important in the more recent ATS statement (Thurston et al., 2017) and continue to be relevant to
16 the evidence base for O₃. In this context, we also recognize the limitations in the available
17 evidence base with regard to our understanding of these aspects (e.g. magnitude, duration and
18 frequency) of such changes (e.g., in lung function) that may be associated with exposure
19 concentrations of interest, including with regard to the exposure levels eliciting effects (as well
20 as the severity or magnitude of the effects) in some population groups not included in the
21 available controlled human exposure studies, such as children and individuals with asthma.
22 Notwithstanding these limitations, we recognize that the controlled human exposure studies,
23 primarily conducted in healthy adults, on which the depth of our understanding of O₃-related
24 health effects is based, in combination with the larger evidence base, inform our conceptual
25 understanding of O₃ responses in people with asthma and in children. Aspects of our
26 understanding continue to be limited, however, including with regard to the risk of particular
27 effects and associated severity for these less studied population groups that may be posed by 7-
28 hour exposures with exercise to concentrations as low as 60 ppb that are estimated in the
29 exposure analyses. Notwithstanding these limitations and associated uncertainties, we take note
30 of the emphasis of the ATS statement on consideration of effects in individuals with pre-existing
31 compromised function, such as that resulting from asthma (an emphasis which is reiterated and
32 strengthened in the current statement) Such considerations are important to the judgments on the
33 adequacy of protection provided by the current standard for at-risk populations. Collectively,
34 these aspects of the evidence and associated uncertainties contribute to a recognition that for O₃,
35 as for other pollutants, the available evidence base in a NAAQS review generally reflects a
36 continuum, consisting of exposure levels at which scientists generally agree that health effects

1 are likely to occur, through lower levels at which the likelihood and magnitude of the response
2 become increasingly uncertain.

3 As at the time the current standard was set in 2015, the exposure and risk estimates
4 developed from modeling exposures to O₃ derived from precursors emitted into ambient air are
5 critically important to consideration of the potential for exposures and risks of concern under air
6 quality conditions of interest, and consequently are critically important to judgments on the
7 adequacy of public health protection provided by the current standard. In turning to consideration
8 of the public health implications of estimated occurrences of exposures (while at increased
9 exertion) to the three benchmark concentrations (60, 70 and 80 ppb), we note the respiratory
10 effects reported for this range of concentrations in controlled human exposure studies during
11 quasi-continuous exercise. In this context, we recognize that the three benchmarks represent
12 exposure conditions associated with different levels of respiratory responses in the subjects
13 studied and can inform judgments on different levels of risk that might be posed to unstudied
14 members of at-risk populations. The highest benchmark concentration (80 ppb) represents an
15 exposure where multiple controlled human exposure studies, involving 6.6-hour exposures
16 during quasi-continuous exercise, demonstrate a range of O₃-related respiratory effects. These
17 respiratory effects include a statistically significant increase in multiple types of respiratory
18 inflammation indicators in multiple studies; statistically significantly increased airway resistance
19 and responsiveness; statistically significant FEV₁ decrements; and statistically significant
20 increases in respiratory symptoms (Table 3.2). In one variable exposure study for which 80 ppb
21 was the exposure period average concentration, the study subject group mean FEV₁ decrement
22 was nearly 8%, with individual decrements of 15% or greater (of moderate or greater size) in
23 16% of subjects and decrements of 10% or greater in 32% of subjects (Schelegle et al., 2009;
24 Table 3.2; Appendix 3D, Figure 3D-11 and Table 3D-20); the percentages of individual subjects
25 with decrements greater than 10 or 15% were lower in other studies for this exposure (Appendix
26 3D, Figure 3D-11 and Table 3D-20). The second benchmark (70 ppb) represents an exposure
27 level below the lowest exposures that have reported both statistically significant FEV₁
28 decrements¹⁰³ and increased respiratory symptoms (reported at 73 ppb, Schelegle et al., 2009) or
29 statistically significant increases in airway resistance and responsiveness (reported at 80 ppb,
30 Horstman et al., 1990). The lowest benchmark (60 ppb) represents still lower exposure, and a
31 level for which findings from controlled human exposure studies of largely healthy subjects have
32 included: statistically significant decrements in lung function (with group mean decrements

¹⁰³ The study group mean lung function decrement for the 73 ppb exposure was 6%, with individual decrements of 15% or greater (of moderate or greater size) in about 10% of subjects and decrements of 10% or greater in 19% of subjects. Decrements of 20% or greater were reported in 6.5% of subjects (Schelegle et al., 2009; Table 3-2; Appendix 3D, Figure 3D-11 and Table 3D-20).

1 ranging from 1.7% to 3.5% across the four studies with average exposures of 60 to 63 ppb¹⁰⁴),
2 but not respiratory symptoms; and, a statistically significant increase in a biomarker of airway
3 inflammatory response relative to filtered air exposures in one study (Kim et al, 2011; Table 3.2).

4 In this context, we additionally note that while not all people experiencing such
5 exposures experience a response (e.g. lung function decrement), as illustrated by the percentages
6 cited above, and among those individuals that experience a response, not all will experience an
7 adverse effect, the likelihood of adverse effects increase as the number of occurrences of O₃
8 exposures of concern increases (as recognized in the 2015 decision establishing the current
9 standard).¹⁰⁵ Thus, while single occurrences can be adverse for some people, particularly for the
10 higher benchmark concentration where the evidence base is stronger, the potential for adverse
11 response increases with repeated occurrences (particularly for people with asthma). Accordingly,
12 we recognize that the exposure/risk analyses provide estimates of exposures of the at-risk
13 population to concentrations of potential concern but are not yet able to provide information on
14 how many of such populations will have an adverse health outcome. Thus, in considering the
15 exposure/risk analysis results, while taking note of the extent of occurrences of one or more days
16 with an exposure at or above a benchmark, particularly the higher benchmarks, we additionally
17 recognize the potential for multiple occurrences to be of greater concern than single occurrences
18 (as was judged in establishing the current standard in 2015).

19 In the 2015 decision establishing the current standard, the controlled human exposure
20 study evidence as a whole provided context for consideration of the 2014 HREA results for the
21 exposures of concern (i.e., the comparison-to-benchmarks analysis) (80 FR 65363, October 26,
22 2015).¹⁰⁶ Similarly, in this reconsideration of the 2020 decision to retain the standard, the

¹⁰⁴ Among subjects in all four of these studies, individual FEV₁ decrements of at least 15% were reported in 3% of subjects, with 7% of subjects reported to have decrements at or above a lower value of 10% (Appendix 3D, Figure 3D-11 and Table 3D-20).

¹⁰⁵ The 2015 decision establishing the current standard stated for example, “the Administrator acknowledge[d] such interindividual variability in responsiveness in her interpretation of estimated exposures of concern.” In this 2015 decision context, the Administrator noted “that not everyone who experiences an exposure of concern, including for the 70 ppb benchmark, is expected to experience an adverse response,” judging “that the likelihood of adverse effects increases as the number of occurrences of O₃ exposures of concern increases.” In making this judgment, the Administrator noted that “the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O₃ may cause other more serious effects.” Therefore, the 2015 decision included her emphasis on “the public health importance of limiting the occurrence of repeated exposures to O₃ concentrations at or above those shown to cause adverse” (80 FR 65331, October 26, 2015).

¹⁰⁶ As summarized in section 3.1 above, the decision in the 2015 review considered the breadth of the O₃ respiratory effects evidence, recognizing the relatively greater significance of effects reported for exposures at and above 80 ppb as well as the greater array of effects elicited. The decision additionally emphasized consideration of the much less severe effects associated with lower exposures, such as 60 ppb, in light of the need for a margin of safety in setting the standard.

1 evidence base of 6.6-hour controlled human exposure studies, particularly those of exposures
2 from 60 to 80 ppb, which is little changed from the 2015 review, provides context for our
3 consideration of the public health implications of the results from the updated exposure/risk
4 analyses. In our consideration of these analyses, we first note several ways in which they differ
5 from and improve upon those available in the 2015 review. For example, we note the number of
6 improvements to input data and modeling approaches summarized in section 3.4.1 above. As in
7 past reviews, exposure and risk are estimated from air quality scenarios designed to just meet an
8 O₃ standard in all its elements. That is, the air quality scenarios are defined by the highest design
9 value in the study area, which is the location with the highest 3-year average of annual fourth
10 highest daily maximum 8-hour O₃ concentrations (e.g., equal to 70 ppb for the current standard
11 scenario). The risk and exposure analyses include air quality simulations based on more recent
12 ambient air quality data that include O₃ concentrations closer to the current standard than was the
13 case for the analyses in the 2015 review. As a result, much smaller reductions in precursor
14 emissions were needed in the photochemical modeling than was the case with the 2014 HREA.
15 Further, this modeling was updated to reflect the current state of the science. Additionally, the
16 approach for deriving population exposure estimates, both for comparison to benchmark
17 concentrations and for use in deriving lung function risk using the E-R function approach, has
18 been modified to provide for a better match of the simulated population exposure estimates with
19 the 6.6-hour duration of the controlled human exposure studies and with the study subject
20 ventilation rates. Together, these differences, as well as a variety of updates to model inputs, are
21 believed to reduce uncertainty associated with our interpretation of the analysis results.

22 As we consider the exposure and risk estimates, we also take note of the array of air
23 quality and exposure circumstances represented by the eight study areas. As summarized in
24 section 3.2.2 above, the areas fall into seven of the nine climate regions in the continental U.S.
25 The population sizes of the associated metropolitan areas range in size from approximately 2.4 to
26 8 million and vary in population demographic characteristics. While there are uncertainties and
27 limitations associated with the exposure and risk estimates, as noted in section 3.4.4 above, the
28 factors recognized here contribute to their usefulness in informing judgments relevant to the
29 Administrator's consideration of the current standard.

30 While there are more adults in the U.S. with asthma than children with asthma, the
31 exposure and risk analysis results in terms of percent of the simulated at-risk populations,
32 indicate higher frequency of exposures of potential concern and risks for children as compared to
33 adults. This finding relates to children's greater frequency and duration of outdoor activity, as
34 well as their greater activity level while outdoors (section 3.4.3 above). In light of these
35 conclusions and findings, we have focused our consideration of the exposure and risk analyses
36 here on children.

1 As can be seen by variation in exposure estimates across the study areas, the eight study
2 areas represent an array of exposure circumstances, including those contributing to relatively
3 higher and relatively lower exposures and associated risk. As recognized in Appendix 3D and in
4 section 3.4.3 above, the risk and exposure analyses are not intended to provide a comprehensive
5 national assessment. Rather, the analyses for this array of study areas and air quality patterns are
6 intended to indicate the magnitude of exposures and risks that may be expected in areas of the
7 U.S. that just meet the current standard but that may differ in ways affecting population
8 exposures of interest. In that way, the exposure and risk estimates are intended to be informative
9 to the EPA’s consideration of potential exposures and risks associated with the current standard
10 and the Administrator’s decision on the adequacy of protection provided by the current standard.

11 While we note reduced uncertainty in several aspects of the exposure and risk analysis
12 approach (as summarized above), we continue to recognize the relatively greater uncertainty
13 associated with the lung function risk estimates compared to the results of the comparison-to-
14 benchmarks analysis (and the greater uncertainty with the estimates derived using the MSS
15 model approach than the E-R approach). Thus, we focus primarily on the estimates of exposures
16 at or above different benchmark concentrations that represent different levels of significance of
17 O₃-related effects, both with regard to the array of effects and severity of individual effects.

18 Based on all of the above, and taking into consideration related information, limitations
19 and uncertainties, such as those recognized above, we address the extent to which the recently
20 available information supports or calls into question the adequacy of protection afforded by the
21 current standard. Focusing on the air quality scenario for the current standard, we note that
22 across all eight study areas, which provide an array of exposure situations, less than 1% of
23 children with asthma are estimated to experience, while breathing at an elevated rate, a daily
24 maximum 7-hour exposure per year at or above 70 ppb, on average across the 3-year period, with
25 a maximum of 1% for the study area with the highest estimates in the highest single year (as
26 summarized in section 3.4.2 above). Further, the percentage for at least one day with such an
27 exposure above 80 ppb is less than 0.1%, as an average across the 3-year period (and 0.1% or
28 less in each of the three years simulated across the eight study areas). No simulated individuals
29 were estimated to experience more than a single such day with an exposure at or above the 80
30 ppb benchmark. Although the exposure and risk analysis approaches have been updated since the
31 2015 review as summarized in section 3.4.1 above, these estimates are generally similar to the
32 comparable estimates for these benchmarks from the 2014 HREA considered at the time the
33 current standard was set,¹⁰⁷ with only slight differences observed, e.g., for the lowest benchmark.

¹⁰⁷ For example, in the 2015 decision to set the standard level at 70 ppb, the Administrator took note of several findings for the air quality scenarios for this level, noting that “a revised standard with a level of 70 ppb is

1 We take note, however, of the differences across air quality scenarios for both sets of estimates
2 which remain appreciably larger than the slight differences between the current and 2014
3 estimates. Thus, we observe that the current estimates of children and children with asthma that
4 might be expected to experience a day with an exposure while exercising at or above the three
5 benchmark concentrations are generally similar to those that were a primary focus of the decision
6 in establishing the current standard in 2015.

7 We additionally consider the estimates of 7-hour exposures, at elevated ventilation, at or
8 above 60 ppb. In so doing, we recognize that the role of this consideration in the 2015 decision
9 was in the context of the judgment of the Administrator at the time regarding an adequate margin
10 of safety for the new standard. We additionally recognize the greater significance of risk for
11 multiple occurrences of days at or above this benchmark, given the associated greater potential
12 for more lasting effects. The exposure analysis estimates indicate fewer than 1% to just over 3%
13 of children with asthma, on average across the 3-year period, to be expected to experience two or
14 more days with an exposure at or above 60 ppb, while at elevated ventilation. This finding of
15 about 97% to more than 99% of children protected from experiencing two or more days with
16 exposures at or above 60 ppb while at elevated exertion is quite similar to the characterization of
17 such estimates at the time of the 2015 decision establishing the current standard (as summarized
18 in section 3.1.2.4 above),¹⁰⁸ and half that indicated by the comparable estimates for air quality
19 just meeting the slightly higher design value of 75 ppb. In addition to this level of protection at
20 the lower exposure level (of 60 ppb), the current information also indicates more than 99% of
21 children with asthma, on average per year, to be protected from a day or more with an exposure
22 at or above 70 ppb. In light of public health judgments by the EPA in prior NAAQS reviews, and
23 related considerations, as well as ATS guidance, we recognize a greater concern for 7-hour
24 exposures generally at or above 70 and 80 ppb (while at elevated exertion) than such exposures
25 to O₃ concentrations below 70 ppb, and a greater concern for repeated (*versus* single)
26 occurrences of such exposures at concentrations at or above 60 ppb up to 70 ppb. With this in
27 mind, we find the current exposure and risk estimates to indicate that the current standard is

estimated to eliminate the occurrence of two or more exposures of concern to O₃ concentrations at or above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O₃ concentrations at or above 70 ppb for all children and children with asthma, even in the worst-case year and location evaluated” (80 FR 65363, October 26, 2015). This statement remains true for the results of the current assessment (Table 3-8).

¹⁰⁸ For example, with regard to the 60 ppb benchmark, for which the 2015 decision placed relatively greater weight on multiple (*versus* single) occurrences of exposures at or above it, the Administrator at that time noted the 2014 HREA estimates for the 70 ppb air quality scenario that estimated 0.5-3.5% of children to experience multiple such occurrences on average across the study areas, stating that the now-current standard “is estimated to protect the vast majority of children in urban study areas ... from experiencing two or more exposures of concern at or above 60 ppb” (80 FR 65364, October 26, 2015). The corresponding estimates, on average across the 3-year period in the current assessments, are remarkably similar at 0.6 -2.9% (Table 3-8).

1 likely to provide a high level of protection from O₃-related health effects to at-risk populations of
2 all children and children with asthma. We additionally recognize such protection to be generally
3 similar to what was estimated when the standard was set in 2015.

4 As recognized above, the protection afforded by the current standard stems from its
5 elements collectively, including the level of 70 ppb, the averaging time of eight hours and the
6 form of the annual fourth-highest daily maximum concentration averaged across three years. The
7 current evidence as considered in the ISA, the current air quality information as analyzed in
8 chapter 2 of this document, and the current risk and exposure information (presented in
9 Appendix 3D and summarized in section 3.4 above) provide continued support to these elements,
10 as well as to the current indicator, as discussed earlier in this section.

11 In summarizing the information discussed thus far, we reflect on the key aspects of the
12 2015 decision that established the current standard. As an initial matter, effects associated with
13 6.6-hour exposures with quasi-continuous exercise (in controlled human exposure studies) to 73
14 ppb O₃ (as a time-weighted average) included both lung function decrements and respiratory
15 symptoms, which the EPA recognized to be adverse; this judgment was based on consideration
16 of the EPA decisions in prior NAAQS reviews and CASAC advice, as well as ATS guidance (80
17 FR 65343, October 26, 2015). We note that the newly available information since the 2015
18 review includes an additional statement from ATS on assessing adverse effects of air pollution
19 which is generally consistent with the earlier statement (available at the time of the 2015
20 decision), e.g., continuing to emphasize potentially at-risk groups, including specific
21 consideration of effects in people with compromised lung function. While recognizing the
22 differences between the current and past exposure and risk analyses, as well as uncertainties
23 associated with such analyses, we note a rough consistency of the associated estimates when
24 considering the array of study areas in both reviews. Overall, the recent quantitative analyses
25 appear to comport with the conclusions reached in the 2015 review regarding control expected to
26 be exerted by the current standard on exposures of concern.

27 We additionally recognize that decisions regarding the adequacy of the current standard
28 depend in part on public health policy judgments, such as those identified above, and judgments
29 about when a standard is requisite to protect the public health, including the health of at-risk
30 populations, allowing for an adequate margin of safety. In this context, we take note of the long-
31 standing health effects evidence that documents the effects of 6.6-hour O₃ exposures on people
32 exposed while breathing at elevated rates and recognize that these effects have been reported in a
33 few individuals for the lowest concentration studied in exposure chambers (40 ppb). Thus, in
34 considering the exposure analysis estimates for 7-hour exposures at and above 60 ppb, we also
35 take note of the variability in the responses at low concentrations, including, for example, the
36 variation in average response to a 7-hour 60 ppb exposure with exercise (group mean FEV₁

1 decrement of 1.7 to 3.5% change), as well as the lack of statistically significant decrements in
2 lung function from such exposures at concentrations below 60 ppb. Consistent with the EPA’s
3 judgments in previous reviews, we also recognize the greater potential for health risk from
4 repeated (*versus* isolated single) occurrences In light of this, we note that the exposure estimates
5 indicate the current standard may be expected to protect more than 97% of populations of
6 children with asthma residing in areas just meeting the current standard from experiencing more
7 than a single day per year with an exposure at or above 60 ppb, on average over a 3-year period.
8 We additionally note the estimates that indicate protection of more than 99.9% of children with
9 asthma living in such areas from experiencing any days with a 7-hour exposure while at elevated
10 exertion of 80 ppb or higher in a 3-year period, on average. In light of ATS guidance, CASAC
11 advice and EPA judgments and considerations in past NAAQS reviews, these results indicate a
12 high level of protection of key at-risk populations from O₃-related health effects that is a
13 generally similar level of protection to what was articulated when the standard was set in 2015
14 and retained in 2020. Thus, the evidence and exposure/risk information, including that related to
15 the lowest exposures studied, lead us to conclude that the combined consideration of the body of
16 evidence and the quantitative exposure estimates including the associated uncertainties, do not
17 call into question the adequacy of the protection provided by the current standard. Rather, this
18 information continues to provide support for the current standard, and thus supports
19 consideration of retaining the current standard, without revision.

20 In reaching these conclusions, we recognize that the Administrator’s decisions in primary
21 standard reviews, in general, are largely public health judgments, as described above. We further
22 note that different public health policy judgments (e.g., from those made in both 2020 and 2015)
23 could lead to different conclusions regarding the extent to which the current standard provides
24 protection of public health with an adequate margin of safety. Such public health judgments
25 include those related to the appropriate degree of public health protection that should be afforded
26 to protect against risk of respiratory effects in at-risk populations, such as asthma exacerbation
27 and associated health outcomes in people with asthma, as well as with regard to the appropriate
28 weight to be given to differing aspects of the evidence and exposure/risk information, and how to
29 consider their associated uncertainties. For example, different judgments might give greater
30 weight to more uncertain aspects of the evidence or reflect a differing view with regard to margin
31 of safety. Such judgments are left to the discretion of the Administrator. In this context, we note
32 that the scientific evidence and quantitative exposure and risk information in the record on which
33 this reconsideration is based are largely unchanged. Staff conclusions regarding the adequacy of
34 the current standards thus remain unchanged from those reached in the 2020 PA.

35 In summary, the newly available health effects evidence, critically assessed in the 2020
36 ISA as part of the full body of evidence, reaffirms conclusions on the respiratory effects

1 recognized for O₃ in prior reviews. Further, we observe the general consistency of the more
2 recent evidence with the evidence that was available in the 2015 review with regard to key
3 aspects on which the current standard is based. We additionally note the quantitative exposure
4 and risk estimates for conditions just meeting the current standard that indicate a generally
5 similar level of protection for at-risk populations from respiratory effects, as that described in the
6 2015 review for the now-current standard. We also recognize limitations and uncertainties
7 associated with the available information, similar to those at the time of the 2015 review.
8 Collectively, these considerations (including those discussed above) provide the basis for the
9 preliminary conclusion that the available evidence and exposure/risk information does not call
10 into question the adequacy of protection provided by the existing standard or the scientific and
11 public health judgments that informed the 2020 decision to retain the current standard, which
12 was established in the 2015 review. Accordingly, we conclude it is appropriate in this
13 reconsideration of the 2020 decision that consideration be given to retaining the current primary
14 standard of 0.070 ppm O₃, as the fourth-highest daily maximum 8-hour concentration averaged
15 across three years, without revision. In light of this conclusion, we have not identified any
16 potential alternative standards for consideration.

17 **3.6 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH**

18 In this section, we highlight key uncertainties associated with reviewing and establishing
19 the primary O₃ standard, while additionally recognizing that research in these areas may be
20 informative to the development of more efficient and effective control strategies. The list in this
21 section includes key uncertainties and data gaps thus far highlighted in this review of the primary
22 standard. A critical aspect of our consideration of the evidence and the quantitative risk/exposure
23 estimates is our understanding of O₃ effects below the lowest concentrations studied in
24 controlled human exposure studies, for longer exposures and for different population groups,
25 particularly including people with asthma. Additional information in several areas would reduce
26 uncertainty in our interpretation of the available information for purposes of risk characterization
27 and, accordingly, reduce uncertainty in characterization of O₃-related health effects. In this
28 section, we highlight areas for future health-related research, model development, and data
29 collection activities to address these uncertainties and limitations in the current scientific
30 evidence. These areas are similar to those highlighted in past reviews.

31 Exposure and Risk Assessment Data and Tools:

- 32 • An important aspect of risk assessment and characterization to inform decisions regarding
33 the primary standard is our understanding of the exposure-response relationship for O₃-
34 related health effects in at-risk populations. Additional research is needed to more

1 comprehensively assess risk of respiratory effects in at-risk individuals exposed to O₃ in
2 the range of 40 to 80 ppb, and lower, for 6.6 hours while engaged in moderate exertion.

- 3 • Population- or cohort-based information on human exposure and associated health effects
4 for healthy adults and children and at-risk populations, including people with asthma, to
5 relevant levels and durations of O₃ concentrations in ambient air, including exposure
6 information in various microenvironments and at varying activity levels, is needed to
7 better evaluate current and future O₃ exposure and lung function risk models. Such
8 information across extended periods would facilitate evaluation of exposure models for
9 the O₃ season.
- 10 • Collection of time-activity data over longer time periods, and particularly for children
11 (including under the age of five), is needed to reduce uncertainty in the modeled exposure
12 distributions that form an important part of the basis for decisions regarding NAAQS for
13 O₃ and other air pollutants. Research addressing energy expenditure and associated
14 breathing rates in various population groups, particularly healthy children and children
15 with asthma, in various locations, across the spectrum of physical activity, including
16 sleep to vigorous exertion, is needed.

18 Health Effects Evidence Base:

- 19 • Epidemiologic studies assessing the influence of “long-term” or “short-term” O₃
20 exposures is complicated by a lack of knowledge regarding the exposure history of study
21 populations. Further, existing studies generally focus on either long-term or short-term
22 exposure separately, thereby making it difficult to assess whether a single short-term
23 high-level exposure versus a repeated long-term low-level exposure, or a combination of
24 both short-term high-level and repeated long-term low-level exposures, influence health
25 outcomes of the study subjects. Epidemiologic studies that include exposure
26 measurements across a longer-term assessment period and can simultaneously assess the
27 impact of these various elements of exposure (i.e., magnitude, frequency, durations, and
28 pattern) are needed.
- 29 • The extent to which the broad mix of photochemical oxidants as well as other copollutants
30 in the ambient air (e.g., PM, NO₂, SO₂, etc.) may play a role in modifying or contributing
31 to the observed associations between ambient air O₃ concentrations and reported health
32 outcomes continues to be an important research question. A better understanding of the
33 broader mixture of photochemical oxidants other than O₃ in ambient air, the associated
34 human exposures, and of the extent to which effects of the mixture may differ from those
35 of O₃, would be informative to future NAAQS reviews. Studies that examine and
36 improve analytical approaches to better understand the role of copollutants, as well as
37 temperature, in contributing to potential confounding or effect modification in
38 epidemiologic models would be helpful.
- 39 • Most epidemiologic study designs remain subject to uncertainty due to use of fixed-site
40 ambient air monitors serving as a surrogate for exposure measurements. The accuracy
41 with which measurements made at stationary outdoor monitors actually reflect subjects’
42 exposure is not yet fully understood. The degree to which discrepancies between

1 stationary monitor measurements and actual pollutant exposures introduces error into
2 statistical estimates of pollutant effects in epidemiologic studies needs to be investigated.

- 3 • For health endpoints reported in epidemiologic studies, such as respiratory hospital
4 admissions, emergency department visits, and premature mortality, a more
5 comprehensive characterization of the exposure circumstances (including ambient air
6 concentrations, as well as duration of exposure and activity levels of individuals) eliciting
7 such effects is needed
- 8 • Further research investigating additional uncertainties and factors that modify
9 epidemiologic associations, particularly for different population groups would improve
10 our understanding in these areas.
- 11 • The evidence base, expanded by evidence newly available for the 2020 review, indicates a
12 likely causal relationship between short-term O₃ exposure and metabolic effects. Further
13 research characterizing perturbations of glucose and insulin homeostasis by O₃ in
14 controlled human exposure studies at exertion and in animal toxicology studies at
15 concentrations closer to the current standard are needed inform decisions regarding the
16 primary standard. The collection of population-based information on clinical health
17 outcomes such as metabolic syndrome, diabetes, etc., as well as intermediate indicators
18 like insulin resistance is also needed for an array of populations and lifestages. Such
19 studies would provide an improved understanding of relationships between O₃ exposure
20 and metabolic-related health outcomes.

21 Air Quality:

- 22 • Advances in photochemical modeling representations of the atmosphere and in high
23 spatial and temporal resolution estimates of ozone precursor emissions will further reduce
24 uncertainties in photochemical modeling used in estimating O₃ concentrations for
25 different air quality scenarios.

26 A more robust ambient monitoring network is needed to better understand ozone concentration
27 gradients in urban areas. With the recent development of low-cost ozone sensors, this could be
28 achieved in the near future.

29

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4 RECONSIDERATION OF THE SECONDARY STANDARD

This chapter presents and evaluates the policy implications of the available scientific and technical information pertaining to this reconsideration of the 2020 decision on the O₃ secondary standard. Specifically, the chapter presents key aspects of the available evidence of the welfare effects of O₃, as documented in the 2020 ISA, with support from the prior ISA and AQCDs, and associated public welfare implications, as well as key aspects of quantitative analyses, including air quality and environmental exposure-related information that has been updated for this reconsideration using more recent air quality monitoring data, and is presented in detail in appendices 4D and 4F associated with this chapter. Together all of this information provides the foundation for our evaluation of the scientific information regarding welfare effects of O₃ in ambient air and the potential for welfare effects to occur under air quality conditions associated with the current standard (or any alternatives considered), 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 (IRP, section 3.2.1) and also takes into account, as relevant, prior assessments of the evidence and quantitative exposure/risk analyses. In light of all of these considerations, we will identify key policy-relevant considerations and summary conclusions regarding the public welfare protection provided by the current standard for the Administrator's consideration in this reconsideration.

Within this chapter, background information on the current standard, including considerations in its establishment in the 2015 review, is summarized in section 4.1. The general approach for considering the available information, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 4.2. Key aspects of the available welfare effects evidence and associated public welfare implications and uncertainties are addressed in section 4.3, and the current air quality and exposure information, with associated uncertainties, is addressed in section 4.4. Section 4.5 summarizes the key evidence- and air quality or exposure-based considerations identified in our evaluation, and also presents associated preliminary conclusions of this analysis. Key remaining uncertainties and areas for future research are identified in section 4.6.

4.1 BACKGROUND ON THE CURRENT STANDARD

As a result of the O₃ NAAQS review completed in 2015, the level of the secondary standard was revised to 0.070 ppm, in conjunction with retaining the indicator (O₃), averaging time (8 hours) and form (fourth-highest annual daily maximum 8-hour average concentration,

1 averaged across three years). The establishment of this standard in 2015, and its retention in
2 2020, is based primarily on consideration of the extensive welfare effects evidence base
3 compiled from more than fifty years of extensive research on the phytotoxic effects of O₃,
4 conducted both in and outside of the U.S., that documents the impacts of O₃ on plants and their
5 associated ecosystems (U.S. EPA, 1978, 1986, 1996, 2006, 2013). Key considerations in the
6 2015 decision were the scientific evidence and technical analyses available at that time, as well
7 as the Administrator's judgments regarding the available welfare effects evidence, the
8 appropriate degree of public welfare protection for the revised standard, and available air quality
9 information on seasonal cumulative exposures (in terms of the W126-based exposure index¹) that
10 may be allowed by such a standard (80 FR 65292, October 26, 2015).

11 The 2020 decision to retain the standard, without revision, additionally took into account
12 updates to the evidence base since the 2015 review, and associated conclusions regarding welfare
13 effects; updated and expanded quantitative analyses of air quality data, including the frequency
14 of cumulative exposures of potential concern and of elevated hourly concentrations in areas with
15 air quality meeting the standard; and also the August 2019 decision of the D.C. Circuit
16 remanding the 2015 secondary standard to the EPA for further justification or reconsideration, as
17 mentioned earlier in Section 1.3 (*Murray Energy Corp. v. EPA*, 936 F.3d 597 [D.C. Cir. 2019]).
18 In the August 2019 decision, the court held that EPA had not adequately explained its decision to
19 focus on a 3-year average for consideration of the cumulative exposure, in terms of W126,
20 identified as providing requisite public welfare protection, or its decision to not identify a
21 specific level of air quality related to visible foliar injury. The EPA's decision not to use a
22 seasonal W126 index as the form and averaging time of the secondary standard was also
23 challenged, but the court did not reach a decision on that issue, concluding that it lacked a basis
24 to assess the EPA's rationale because the EPA had not yet fully explained its focus on a 3-year
25 average W126 in its consideration of the standard. Accordingly, the 2020 decision included
26 discussion of these areas to address these aspects of the court's decision.

27 Among the updates to the welfare effects evidence considered in the 2020 decision was
28 the welfare effects evidence for two insect-related categories of effects with new determinations
29 in the 2020 ISA. Specifically, the 2020 ISA concluded the evidence sufficient to infer likely
30 causal relationships of O₃ with alterations of plant-insect signaling and insect herbivore growth
31 and reproduction. Uncertainties in the evidence for the effects, however, precluded a full
32 understanding of the effects, the air quality conditions that might elicit them, and the potential

¹ The W126 index is a cumulative seasonal metric described as the sigmoidally weighted sum of all hourly O₃ concentrations during a specified daily and seasonal time window, with each hourly O₃ concentration given a weight that increases from zero to one with increasing concentration (80 FR 65373-74, October 26, 2015). The units for W126 index values are ppm-hours (ppm-hrs). More detail is provided in section 4.3.3.1.1 below.

1 for impacts in a natural ecosystem. Together this resulted in a lack of clarity in the
2 characterization of these effects, and a lack of important quantitative information to consider
3 such effects in the context of reviewing the standard, such as in judging how particular ambient
4 air concentrations of O₃ relate to the degree of impacts on public welfare related to these effects.

5 With regard to the more well-established vegetation-related effects of O₃ in ambient air,
6 the extensive evidence base considered in the 2015 and 2020 decisions documents an array of
7 effects, ranging from the organism scale to larger-scale impacts, such as those on populations,
8 communities, and ecosystems. These categories of effects which the 2013 and 2020 ISAs
9 identified as causally or likely causally related to O₃ in ambient air include: reduced vegetation
10 growth, reproduction, crop yield, productivity and carbon sequestration in terrestrial systems;
11 alteration of terrestrial community composition, belowground biogeochemical cycles and
12 ecosystem water cycling; and visible foliar injury (2013 ISA, Appendix 9; 2020 ISA, Appendix
13 8).² Across the different types of studies, the strongest quantitative evidence available in both the
14 2015 and 2020 reviews for effects from O₃ exposure on vegetation comes from controlled
15 exposure studies of growth effects in a number of species (2013 ISA, p. 1-15). Of primary
16 importance in considering the appropriate level of protection for the standard, both in the 2015
17 decision establishing it and in its 2020 retention, were the studies of O₃ exposures that reduced
18 growth in tree seedlings from which E-R functions of seasonal relative biomass loss (RBL)³ have
19 been established (80 FR 65385-86, 65389-90, October 26, 2015). Consistent with advice from
20 the CASAC in both reviews, the Administrators considered the effects of O₃ on tree seedling
21 growth as a surrogate or proxy for the broader array of vegetation-related effects of O₃, ranging
22 from effects on sensitive species to broader ecosystem-level effects (80 FR 65369, 65406,
23 October 26, 2015; 85 FR 87319, 87399, December 31, 2020).

24 In their consideration of O₃ effects on tree seedling growth, the Administrators in both
25 the 2015 and 2020 decisions ascribed importance to the intended use of the natural resources and
26 ecosystems potentially affected. For example, the 2015 decision considered the available
27 evidence and quantitative analyses in the context of an approach for considering and identifying
28 public welfare objectives for the revised standard (80 FR 65403-65408, October 26, 2015). In
29 light of the extensive evidence base of O₃ effects on vegetation and associated terrestrial

² The 2020 ISA also newly determined the evidence sufficient to infer likely causal relationships of O₃ with increased tree mortality, although it does not indicate a potential for O₃ concentrations that occur in locations that meet the current standard to cause this effect (85 FR 87319, December 31, 2020; 2020 PA, section 4.3.1).

³ These functions were developed to quantify O₃-related reduced growth in tree seedlings relative to control treatments (without O₃). In this way, RBL is the percentage by which the O₃ treatment growth in a growing season differs from the control seedlings over the same period, and the functions provide a quantitative estimate of the reduction in a year's growth as a percentage of that expected in the absence of O₃ (2013 ISA, section 9.6.2; 2020 PA, Appendix 4A).

1 ecosystems, the Administrator, in both decisions, focused on protection against adverse public
2 welfare effects of O₃-related effects on vegetation, giving particular attention to such effects in
3 natural ecosystems, such as those in areas with protection designated by Congress, and areas
4 similarly set aside by states, tribes and public interest groups, with the intention of providing
5 benefits to the public welfare for current and future generations (80 FR 65405, October 26, 2015;
6 85 FR 87344, December 31, 2020).

7 Climate-related effects were also considered in both reviews (2013 ISA, Appendix 10,
8 Section 10.3; 2020 ISA, Appendix 9, Section 9.2 and 9.3). In 2020, as was the case when the
9 standard was set in 2015, the evidence documents tropospheric O₃ as a greenhouse gas causally
10 related to radiative forcing, and likely causally related to subsequent effects on variables such as
11 temperature and precipitation. In 2020, as in 2015, limitations and uncertainties in the evidence
12 base affected characterization of the extent of any relationships between ground-level O₃
13 concentrations in ambient air in the U.S. and climate-related effects and preclude quantitative
14 characterization of climate responses to changes in ground-level O₃ concentrations in ambient air
15 at regional or national (vs global) scales. As a result, the EPA recognized the lack of important
16 quantitative tools with which to consider such effects in its review of the standard. For example,
17 it was not feasible to relate different patterns of ground-level O₃ concentrations at the regional
18 (or national) scale in the U.S. with specific risks of alterations in temperature, precipitation, and
19 other climate-related variables. Thus, the available information did not provide a sufficient basis
20 for use in considering the adequacy of the secondary standard in either review (80 FR 65370,
21 October 26, 2015; 85 FR 87337-87339, December 31, 2020).

22 For quantifying effects on tree seedling growth as a surrogate or proxy for a broader array
23 of vegetation-related effects using the RBL metric, in 2015 and 2020 the evidence base provided
24 established E-R functions for seedlings of 11 tree species (80 FR 65391-92, October 26, 2015;
25 2014 PA, Appendix 5C; 85 FR 87307-9, 87313-4, December 31, 2020; 2020 PA, Appendix 4A).
26 Cumulative O₃ exposure was evaluated in terms of the W126 cumulative seasonal exposure
27 index, an index supported by the evidence in the 2013 and 2020 ISAs for this purpose and that
28 was consistent with advice from the CASAC in both reviews (2013 ISA, section 9.5.3, p. 9-99;
29 80 FR 65375, October 26, 2015; 2020 ISA, section 8.13; 85 FR 87307-8, December 31, 2020).
30 In judgments regarding effects that are adverse to the public welfare, the decision setting the
31 standard in 2015, and that retaining it in 2020, both utilized the RBL as a quantitative tool within
32 a larger framework of considerations pertaining to the public welfare significance of O₃ effects
33 (80 FR 65389, October 26, 2015; 73 FR 16496, March 27, 2008; 85 FR 87339-41, December 31,
34 2020).

35 Accordingly, in both the 2015 and 2020 decisions, consideration of the appropriate public
36 welfare protection objective for the secondary standard gave prominence to the estimates of tree

1 seedling growth impacts (in terms of RBL) for a range of W126 index values, developed from
2 the E-R functions for 11 tree species (80 FR 65391-92, Table 4, October 26, 2015; 85 FR 87339-
3 41, December 31, 2020). The Administrators also incorporated into their considerations the
4 broader evidence base associated with forest tree seedling biomass loss, including other less
5 quantifiable effects of potentially greater public welfare significance. That is, in drawing on
6 these RBL estimates, the Administrators noted they were not simply making judgments about a
7 specific magnitude of growth effect in seedlings that would be acceptable or unacceptable in the
8 natural environment. Rather, though mindful of associated uncertainties, the RBL estimates were
9 used as a surrogate or proxy for consideration of the broader array of related vegetation-related
10 effects of potential public welfare significance, which included effects on individual species and
11 extending to ecosystem-level effects (80 FR 65406, October 26, 2015; 85 FR 87304, December
12 31, 2020). This broader array of vegetation-related effects included those for which public
13 welfare implications are more significant but for which the tools for quantitative estimates were
14 more uncertain.

15 In the 2015 decision to revise the standard level to 70 ppb, and also the 2020 decision to
16 retain that standard, without revision, air quality analyses played an important role in the
17 Administrator’s judgments. Such judgments of the Administrator in setting the standard in 2015
18 are briefly summarized below. These are followed by a summary of additional key aspects of the
19 considerations and judgments associated with the decision to retain this standard in 2020.

20 In using the RBL estimates as a proxy, the Administrator in 2015 focused her attention on
21 a revised standard that would generally limit cumulative exposures to those for which the median
22 RBL estimate for seedlings of the 11 species with established E-R functions would be somewhat
23 below 6% (80 FR 65406-07, October 26, 2015).⁴ She noted that the median RBL estimate was
24 6% for a cumulative seasonal W126 exposure index of 19 ppm-hrs (80 FR 65391-92, Table 4,
25 October 26, 2015). Given the information on median RBL at different W126 exposure levels,
26 using a 3-year cumulative exposure index for assessing vegetation effects,⁵ the potential for

⁴ The Administrator noted the CASAC view regarding 6%, most particularly the CASAC’s characterization of this level of effect in the median studied species as “unacceptably high” (Frey, 2014, pp. iii, 13, 14). These comments were provided in the context of CASAC’s considering the significance of effects associated with a range of alternatives for the secondary standard (80 FR 65406, October 26, 2015).

⁵ Based on a number of considerations, the Administrator recognized greater confidence in judgments related to public welfare impacts based on a 3-year average metric than a single-year metric, and consequently concluded it to be appropriate to use a seasonal W126 index averaged across three years for judging public welfare protection afforded by a revised secondary standard. For example, she recognized uncertainties associated with interpretation of the public welfare significance of effects resulting from a single-year exposure, and that the public welfare significance of effects associated with multiple years of critical exposures are potentially greater than those associated with a single year of such exposure. She additionally concluded that use of a 3-year average metric could address the potential for adverse effects to public welfare that may relate to shorter exposure periods, including a single year (80 FR 65404, October 26, 2015).

1 single-season effects of concern, and CASAC comments on the appropriateness of a lower value
2 for a 3-year average W126 index, the Administrator concluded it was appropriate to identify a
3 standard that would restrict cumulative seasonal exposures to 17 ppm-hrs or lower, in terms of a
4 3-year W126 index, in nearly all instances (80 FR 65407, October 26, 2015). Based on such
5 information, available at that time, to inform consideration of vegetation effects and their
6 potential adversity to public welfare, the Administrator additionally judged that the RBL
7 estimates associated with marginally higher exposures in isolated, rare instances were not
8 indicative of effects that would be adverse to the public welfare, particularly in light of
9 variability in the array of environmental factors that can influence O₃ effects in different systems
10 and uncertainties associated with estimates of effects associated with this magnitude of
11 cumulative exposure in the natural environment (80 FR 65407, October 26, 2015).

12 Using these objectives, the 2015 decision regarding a standard revised from the then-
13 existing (2008) standard was based on extensive air quality analyses that included the most
14 recently available data as well as air monitoring data that extended back more than a decade (80
15 FR 65408, October 26, 2015; Wells, 2015). These analyses evaluated the cumulative seasonal
16 exposure levels in locations meeting different alternative levels for a standard of the existing
17 form and averaging time. These analyses supported the Administrator's judgment that a standard
18 with a revised level in combination with the existing form and averaging time could achieve the
19 desired level of public welfare protection, considered in terms of cumulative exposure, quantified
20 as the W126 index (80 FR 65408, October 26, 2015). Based on the extensive air quality analyses
21 and consideration of the W126 index value associated with a median RBL of 6%, and the W126
22 index values at monitoring sites that met different levels for a revised standard of the existing
23 form and averaging time, the Administrator additionally judged that a standard level of 70 ppb
24 would provide the requisite protection. The Administrator noted that such a standard would be
25 expected to limit cumulative exposures, in terms of a 3-year average W126 exposure index, to
26 values at or below 17 ppm-hrs, in nearly all instances, and accordingly, to eliminate or virtually
27 eliminate cumulative exposures associated with a median RBL of 6% or greater (80 FR 65409,
28 October 26, 2015).

29 The 2015 decision also took note of the well-recognized evidence for visible foliar injury
30 and crop yield effects. However, the RBL information available for seedlings of a set of 11 tree
31 species was judged to be more useful (particularly in a role as surrogate for the broader array of
32 vegetation-related effects) in informing judgments regarding the nature and severity of effects
33 associated with different air quality conditions and associated public welfare significance than
34 the available information on visible foliar injury and crop yield effects (80 FR 65405-06,
35 October 26, 2015). With regard to visible foliar injury, while the Administrator recognized the
36 potential for this effect to affect the public welfare in the context of affecting value ascribed to

1 natural forests, particularly those afforded special government protection, she also recognized
2 limitations in the available information that might inform consideration of potential public
3 welfare impacts related to this vegetation effect noting the significant challenges in judging the
4 specific extent and severity at which such effects should be considered adverse to public welfare
5 (80 FR 65407, October 26, 2015).⁶ Similarly, while O₃-related growth effects on agricultural and
6 commodity crops had been extensively studied and robust E-R functions developed for a number
7 of species, the Administrator found this information less useful in informing judgments
8 regarding an appropriate level of public welfare protection (80 FR 65405, October 26, 2015).⁷

9 In summary, the 2015 decision focused primarily on the information related to trees and
10 growth impacts in identifying the public welfare objectives for the revised secondary standard
11 (80 FR 65409-65410, October 26, 2015). In this context, the Administrator in 2015 judged that
12 the 70 ppb standard would protect natural forests in Class I and other similarly protected areas
13 against an array of adverse vegetation effects, most notably including those related to effects on
14 growth and productivity in sensitive tree species. She additionally judged that the new standard
15 would be sufficient to protect public welfare from known or anticipated adverse effects. These
16 judgments by the Administrator at that time appropriately recognized that the CAA does not
17 require that standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently
18 so as to protect the public welfare from known or anticipated adverse effects.

19 In 2020, as in 2015, the Administrator considered the available information regarding the
20 appropriate O₃ exposure metric to employ in assessing adequacy of air quality control in
21 protecting against RBL. In addition to finding it appropriate to continue to consider the seasonal
22 W126 index averaged over a 3-year period to estimate median RBL (as was concluded in 2015),
23 the Administrator in 2020 also judged it appropriate to also consider other metrics including peak
24 hourly concentrations⁸ (85 FR 87344, December 2020). With regard to these conclusions, his

⁶ These limitations included the lack of established E-R functions that would allow prediction of visible foliar injury severity and incidence under varying air quality and environmental conditions, a lack of consistent quantitative relationships linking visible foliar injury with other O₃-induced vegetation effects, such as growth or related ecosystem effects, and a lack of established criteria or objectives relating reports of foliar injury with public welfare impacts (80 FR 65407, October 26, 2015).

⁷ With respect to commercial production of commodities, the Administrator noted the difficulty in discerning the extent to which O₃-related effects on commercially managed vegetation are adverse from a public welfare perspective, given that the extensive management of such vegetation (which, as the CASAC noted, may reduce yield variability) may also to some degree mitigate potential O₃-related effects. Management practices are highly variable and are designed to achieve optimal yields, taking into consideration various environmental conditions. Further, changes in yield of commercial crops and commercial commodities, such as timber, may affect producers and consumers differently, complicating the assessment of overall public welfare effects still further (80 FR 65405, October 26, 2015).

⁸ Both the 2020 and 2013 ISAs reference the longstanding recognition of the risk posed to vegetation of peak hourly O₃ concentrations (e.g., “[h]igher concentrations appear to be more important than lower concentrations in

1 considerations included the extent of conceptual similarities of the 3-year average W126 index to
2 some aspects of the derivation approach for the established E-R functions, the context of RBL as
3 a proxy (as recognized above), and limitations associated with a reliance solely on W126 index
4 as a metric to control exposures that might be termed “unusually damaging”⁹ (85 FR 877339-40,
5 December 31, 2020).

6 With regard to the derivation and application of the established E-R functions, the 2020
7 review recognized several factors to contribute uncertainty and some resulting imprecision or
8 inexactitude to RBL estimated from single-year seasonal W126 index values (85 FR 49900-01,
9 August 14, 2020; 2020 PA sections 4.5.1.2 and 4.5.3).¹⁰ Additionally recognized was the
10 qualitative and conceptual nature of our understanding, in many cases, of relationships of O₃
11 effects on plant growth and productivity with larger-scale impacts, such as those on populations,
12 communities and ecosystems. From these considerations, it was judged that use of a seasonal
13 RBL averaged over multiple years, such as a 3-year average, is reasonable, and provides a more

eliciting a response” [ISA, p. 8-180]; “higher hourly concentrations have greater effects on vegetation than lower concentrations” [2013 ISA, p. 91-4] “studies published since the 2006 O₃ AQCD do not change earlier conclusions, including the importance of peak concentrations, ... in altering plant growth and yield” [2013 ISA, p. 9-117]). While the evidence does not indicate a particular threshold number of hours at or above 100 ppb (or another reference point for elevated concentrations), the evidence of greater impacts from higher concentrations (particularly with increased frequency) and the air quality analyses that document variability in such concentrations for the same W126 index value led the Administrator to judge such a multipronged approach to be needed to ensure appropriate consideration of exposures of concern and the associated protection from them afforded by the secondary standard (85 FR 87340, December 31, 2020).

⁹ In its discussion regarding the EPA’s use of a 3-year average W126 index, the 2019 court decision remanding the 2015 standard back to the EPA referenced advice from the CASAC in the 2015 review on protection against “unusually damaging years.” Use of this term occurs in the 2014 CASAC letter on the second draft PA (Frey, 2014). Most prominently, the CASAC defined as damage “injury effects that reach sufficient magnitude as to reduce or impair the intended use or value of the plant to the public, and thus are adverse to public welfare” (Frey, 2014, p. 9). We also note that the context for the CASAC’s use of the phrase “unusually damaging years” in the 2015 review is in considering the form and averaging time for a revised secondary standard in terms of a W126 index (Frey, 2014, p. 13), which as discussed below is relatively less controlling of high-concentration years (whether as a single year index or averaged over three years) than the current secondary standard and its fourth highest daily maximum 8-hour metric (85 FR 87327, December 31, 2020).

¹⁰ The E-R functions were derived mathematically from studies of different exposure durations (varying from shorter than one to multiple growing seasons) by applying adjustments so that they would yield estimates normalized to the same period of time (season). Accordingly, the estimates may represent average impact for a season, and have compatibility with W126 index averaged over multiple growing seasons or years (85 FR 87326, December 31, 2020; 2020 PA, section 4.5.1.2, Appendix 4A, Attachment 1). The available information also indicated that the patterns of hourly concentrations (and frequency of peak concentrations, e.g., at/above 100 ppb) in O₃ treatments on which the E-R functions are based differ from the patterns in ambient air meeting the current standard across the U.S. today (85 FR 87327, December 31, 2020). Additionally noted was the year-to-year variability of factors other than O₃ exposures that affect tree growth in the natural environment (e.g., related to variability in soil moisture, meteorological, plant-related and other factors), that have the potential to affect O₃ E-R relationships (ISA, Appendix 8, section 3.12; 2013 ISA section 9.4.8.3; PA, sections 4.3 and 4.5). All of these considerations contributed to the finding of a consistency of the use of W126 index averaged over multiple years with the approach used in deriving the E-R function, and with other factors that may affect growth in the natural environment (85 FR 87340, December 31, 2020).

1 stable and well-founded RBL estimate for its use as a proxy to represent the array of vegetation-
2 related effects identified above. More specifically, the Administrator concluded that the use of an
3 average seasonal W126 index derived from multiple years (with their representation of
4 variability in environmental factors) provides an appropriate representation of the evidence and
5 attention to the identified considerations. In so doing, he found that a sole reliance on single year
6 W126 estimates for reaching judgments with regard to magnitude of O₃ related RBL and
7 associated judgments of public welfare protection would ascribe a greater specificity and
8 certainty to such estimates than supported by the evidence. Rather, consistent with the judgment
9 of the prior Administrator, the Administrator in 2020 found it appropriate, for purposes of
10 considering public welfare protection from effects for which RBL is used as a proxy, to primarily
11 consider W126 index in terms of a 3-year average metric (85 FR 87339-87340, December 31,
12 2020).

13 With regard to the EPA's use of a 3-year average W126 index to assess protection from
14 RBL, the 2020 decision additionally took into account the 2019 court remand on this issue,
15 including the remand's reference to protection against "unusually damaging years." (85 FR
16 87325-87328, December 31, 2020). Accordingly, the EPA considered air quality analyses of
17 peak hourly concentrations in the context of considering protection against "unusually damaging
18 years." With regard to this caution, and in the context of controlling exposure circumstances of
19 concern (e.g., for growth effects, among others), the EPA considered air quality analyses that
20 investigated the annual occurrence of elevated hourly O₃ concentrations which may contribute to
21 vegetation exposures of concern (2020 PA, Appendix 2A, section 2A.2; Wells, 2020). These air
22 quality analyses illustrate limitations of the W126 index (whether in terms of a 3-year average or
23 a single year) for the purpose of controlling peak concentrations,¹¹ and also the strengths of the
24 current standard in this regard. The air quality analyses show that the form and averaging time of
25 the existing standard, in addition to controlling cumulative exposures in terms of W126 (as found
26 in the 2015 review), is much more effective than the W126 index in limiting peak concentrations
27 (e.g., hourly O₃ concentrations at or above 100 ppb)¹² and in limiting number of days with any
28 such hours (Wells, 2020, e.g., Figures 4, 5, 8, 9 compared to Figures 6, 7, 10 and 11).¹³ Thus, the
29 W126 index, by its very definition, and as illustrated by the air quality data analyses, does not

¹¹ The W126 index cannot, by virtue of its definition, always differentiate between air quality patterns with high peak concentrations and those without such concentrations.

¹² As described in section 4.3.3 below, the occurrence of high concentrations (including those at or above 100 ppb [e.g., Smith, 2012; Smith et al., 2012]), as well as cumulative exposures influence the effects of O₃ on plants.

¹³ With regard to the existing standard, historical air quality data extending back to 2000 additionally show the appreciable reductions in peak concentrations that have been achieved in the U.S. as air quality has improved under O₃ standards of the existing form and averaging time (Wells, 2020, Figures 12 and 13).

1 provide specificity with regard to year-to-year variability in elevated hourly O₃ concentrations
2 with the potential to contribute to the “unusually damaging years” that the CASAC had identified
3 for increased concern in the 2015 review. As a result, the 2020 decision found that a standard
4 based on a W126 index (either a 3-year or a single-year index) would not be expected to provide
5 effective control of the peak concentrations that may contribute to “unusually damaging years”
6 for vegetation.¹⁴ Based on all of the above, the 2020 decision concluded that control of such
7 years is a characteristic of the existing standard (the effectiveness of which is demonstrated by
8 the air quality analyses), and that that use of a seasonal W126 averaged over a 3-year period,
9 which is the design value period for the current standard, to estimate median RBL using the
10 established E-R functions, in combination with a broader consideration of air quality patterns,
11 such as peak hourly concentrations, is appropriate for considering the public welfare protection
12 provided by the standard (85 FR 87340-87341, December 31, 2020).

13 With regard to O₃ effects on crop yield for which there is long-standing evidence,
14 qualitative and quantitative, of the reducing effect of O₃ on the yield of many crops and a
15 potential for public welfare significance, the 2020 decision concluded that the existing standard
16 provides adequate protection of public welfare related to crop yield loss (85 FR 87342,
17 December 31, 2020). Key considerations in this conclusion included the established E-R
18 functions for 10 crops and the estimates of RYL derived from them (2020 ISA, 2020 PA,
19 Appendix 4A, section 4A.1, Table 4A-4), as well as the existence of a number of complexities
20 related to the heavy management of many crops to obtain a particular output for commercial
21 purposes, and related to other factors (85 FR 87341-87342, December 31, 2020). For example,
22 the Administrator considered the extensive management of agricultural crops that occurs to elicit
23 optimum yields (e.g., through irrigation and usage of soil amendments, such as fertilizer) to be
24 relevant in evaluating the extent of RYL estimated from experimental O₃ exposures that should
25 be judged adverse to the public welfare. With regard to the E-R functions for RYL for 10 crops,
26 the Administrator considered the air quality data with regard to the W126 index levels and
27 corresponding estimated RYL for the median species. He also took into consideration the
28 extensive management of agricultural crops, and the complexities associated with identifying
29 adverse public welfare effects for market-traded goods (where producers and consumers may be
30 impacted differently). Further, he noted that the secondary standard is not intended to protect
31 against all known or anticipated O₃-related effects, but rather those that are judged to be adverse
32 to the public welfare. The air quality data indicated that the current standard generally maintains

¹⁴ From these analyses, the Administrator concluded that the form and averaging time of the current standard is effective in controlling peak hourly concentrations and that a W126 index based standard would be much less effective in providing the needed protection against years with such elevated and potentially damaging hourly concentrations.

1 air quality at a W126 index below 17 ppm-hrs, with few exceptions, and would accordingly limit
2 the associated estimates of median RYL below 5.1% (based on experimental O₃ exposures), a
3 level which the Administrator judged would not constitute an adverse effect on public welfare.
4 Therefore, he concluded that the current standard provides adequate protection of public welfare
5 related to crop yield loss and did not need to be revised to provide additional protection against
6 this effect (85 FR 87342, December 31, 2020).

7 With regard to visible foliar injury, the Administrator considered the question of a level
8 of air quality that would provide protection against visible foliar injury related effects known or
9 anticipated to cause adverse effects to the public welfare. Based on the evidence and associated
10 quantitative analyses in this review, summarized in the 2020 PA, the Administrator's judgment
11 reflected his recognition of less confidence and greater uncertainty in the existence of adverse
12 public welfare effects with lower O₃ exposures (85 FR 87342-87344, December 31, 2020).

13 While recognizing there to be a paucity of established approaches for interpreting specific levels
14 of severity and extent of foliar injury in natural areas with regard to impacts on the public
15 welfare (e.g., related to recreational services), the Administrator recognized that injury to whole
16 stands of trees of a severity apparent to the casual observer (e.g., when viewed as a whole from a
17 distance) would reasonably be expected to affect recreational values and thus pose a risk of
18 adverse effects to the public welfare. He further noted that the available information did not
19 provide for specific characterization of the incidence and severity that would not be expected to
20 be apparent to the casual observer, nor for clear identification of the pattern of O₃ concentrations
21 that would provide for such a situation. In recognizing that quantitative analyses and evidence
22 are lacking that might support a more precise identification of a severity of visible foliar injury
23 and extent of occurrence that might be judged adverse to the public welfare, the Administrator
24 considered the USFS system for interpreting visible foliar injury impacts in surveys conducted at
25 biomonitoring sites (biosites) across the U.S. from 1994 through 2011. At these sites, the USFS
26 followed a national protocol that includes a scoring system with descriptors for biosite index
27 (BI)¹⁵ scores of differing magnitude for his purposes in this regard. More specifically, he
28 concluded that findings of BI scores categorized as "moderate to severe" injury by the USFS
29 scheme would be an indication of visible foliar injury occurrence that, depending on extent and
30 severity, may raise public welfare concerns. In this framework, the Administrator considered the
31 2020 PA evaluations of the available information and what that information indicated with
32 regard to patterns of air quality of concern for such an occurrence, and the extent to which they
33 are expected to occur in areas that meet the current standard. For example, the incidence of
34 nonzero BI scores, and particularly of relatively higher scores such as those above 15, classified

¹⁵ The BI is a measure of the severity of O₃-induced visible foliar injury observed at each biosite (Smith, 2012).

1 as indicative of “moderate to severe” injury in the USFS scheme appear to markedly increase
2 only with W126 index values above 25 ppm-hrs. He further took note of the multiple published
3 studies analyzing the USFS data across multiple years and multiple U.S. regions with regard to
4 metrics intended to quantify influential aspects of O₃ air quality, which indicated a potential role
5 for an additional metric related to the occurrence of days with relatively high hourly
6 concentrations (e.g., number of days with a 1-hour concentration at or above 100 ppb [2020 PA,
7 section 4.5.1.2]). In light of this evidence and the 2020 PA analyses of these data, the
8 Administrator judged that W126 index values at or below 25 ppm-hrs, when in combination with
9 infrequent occurrences of hourly concentrations at or above 100 ppb, would not be anticipated to
10 pose risk of visible foliar injury of an extent and severity so as to be adverse to the public welfare
11 (85 FR 87343, December 31, 2020).

12 With these conclusions in mind, the Administrator considered the available air quality
13 analyses (85 FR 87316-18, December 31, 2020; 2020 PA, Appendix 4C, section 4C.3; Appendix
14 4D; Wells, 2020). Together these analyses indicated that a W126 index above 25 ppm-hrs (either
15 as a 3-year average or in a single year) is not seen to occur at monitoring locations where the
16 current standard is met (including in or near Class I areas), and that, in fact, values above 17 or
17 19 ppm-hrs are rare and that days with any hourly concentrations at or above 100 ppb at
18 monitoring sites that meet the current standard are uncommon. Based on these findings, the
19 Administrator concluded that the current standard provides control of air quality conditions that
20 contribute to increased BI scores and to scores of a magnitude indicative of “moderate to severe”
21 foliar injury. Further, he noted the 2020 PA finding that the information from the USFS biosite
22 monitoring program, particularly in locations meeting the current standard or with W126 index
23 estimates likely to occur under the current standard, does not indicate a significant extent and
24 degree of injury (e.g., based on analyses of BI scores in the PA, Appendix 4C) or specific
25 impacts on recreational or related services for areas, such as wilderness areas or national parks,
26 thus giving credence to the associated 2020 PA conclusion that the evidence indicates that areas
27 that meet the current standard are unlikely to have BI scores reasonably considered to be impacts
28 of public welfare significance (85 FR 87344, December 31, 2020).

29 Before reaching a final decision on the standard, the Administrator, in returning to his
30 primary focus on RBL in its role as proxy for the broader array of vegetation-related effects of
31 O₃, further considered the available analyses of both the air quality data newly available in the
32 2020 review and of historical air quality at sites across the U.S., particularly including those sites
33 in or near Class I areas, for which the findings were consistent with the air quality analyses

1 available in the 2015 review.¹⁶ That is, in virtually all design value periods between 2000 and
2 2018 and all locations at which the current standard was met across the 19 years and 17 design
3 value periods (in more than 99.9% of such observations), the 3-year average W126 metric was at
4 or below 17 ppm-hrs. Further, in all such design value periods and locations the 3-year average
5 W126 index was at or below 19 ppm-hrs (85 FR 87344, December 31, 2020).

6 The Administrator additionally considered the protection provided by the current
7 standard from the occurrence of O₃ exposures within a single year with potentially damaging
8 consequences, including a significantly increased incidence of areas with visible foliar injury that
9 might be judged moderate to severe. He gave particular focus to BI scores above 15, termed
10 “moderate to severe injury” by the USFS categorization scheme (85 FR 87344, December 31,
11 2020; 2020 PA, sections 4.3.3.2, 4.5.1.2 and Appendix 4C). As discussed above, the incidence of
12 USFS sites with BI scores above 15 markedly increases with W126 index estimates above 25
13 ppm-hrs, a magnitude of W126 index indicated by the air quality analysis to be scarce at sites
14 that meet the current standard, with just a single occurrence across all U.S. sites with design
15 values meeting the current standard in the 19-year historical dataset dating back to 2000 (2020
16 PA, section 4.4, and Appendix 4D). Further, in light of the evidence indicating that peak short-
17 term concentrations (e.g., of durations as short as one hour) may also play a role in the
18 occurrence of visible foliar injury, the Administrator additionally took note of the air quality
19 analyses of hourly concentrations (2020 PA, Appendix 2A; Wells 2020). These analyses of data
20 from the past 20 years show a declining trend in 1-hour daily maximum concentrations mirroring
21 the declining trend in design values, supporting the 2020 PA conclusion that the form and
22 averaging time of the current standard provides appreciable control of peak 1-hour
23 concentrations. Furthermore, these analyses for the period from 2000 to 2018 indicate that sites
24 meeting the current standard had only a few days (up to just seven) with hourly concentrations at
25 or above 100 ppb (Wells, 2020). In light of these findings from the air quality analyses and
26 considerations in the 2020 PA, both with regard to 3-year average W126 index values at sites
27 meeting the current standard and the rarity of such values at or above 19 ppm-hrs, and with
28 regard to single-year W126 index values at sites meeting the current standard, and the rarity of
29 such values above 25 ppm-hrs, as well as with regard to the appreciable control of 1-hour daily
30 maximum concentrations, the Administrator judged that the current standard provides adequate
31 protection from air quality conditions with the potential to be adverse to the public welfare (85
32 FR 87344, December 31, 2020).

¹⁶ These data are distributed across all nine NOAA climate regions and 50 states, although some geographic areas within specific regions and states may be more densely covered and represented by monitors than others (2020 PA, Appendix 4D).

1 In reaching his conclusion on the current secondary O₃ standard, the Administrator
2 recognized, as is the case in NAAQS reviews in general, his decision depended on a variety of
3 factors, including science policy judgments and public welfare policy judgments, as well as the
4 available information. In the 2020 decision, the Administrator gave primary attention to the
5 principal effects of O₃ as recognized in the current ISA, the 2013 ISA and past AQCDs, and for
6 which the evidence is strongest (e.g., growth, reproduction, and related larger-scale effects, as
7 well as visible foliar injury). With regard to growth and the categories of effects identified above
8 for which RBL has been identified for use as a proxy, based on all of the identified
9 considerations, including the discussion of air quality immediately above, the Administrator
10 judged the current standard to provide adequate protection for air quality conditions with the
11 potential to be adverse to the public welfare. Further, with regard to visible foliar injury, the
12 Administrator concluded that the available information on visible foliar injury and with regard to
13 air quality analyses that may be informative to identification of air quality conditions associated
14 with appreciably increased incidence and severity of BI scores at USFS biomonitoring sites, and
15 with particular attention to Class I and other areas afforded special protection, indicated the
16 current standard to provide adequate protection from visible foliar injury of an extent or severity
17 that might be anticipated to be adverse to the public welfare.

18 In summary, the 2020 decision was based on consideration of the public welfare
19 protection afforded by the secondary O₃ standard from identified O₃-related welfare effects, and
20 from their potential to present adverse effects to the public welfare, and also on judgments
21 regarding what the available evidence, quantitative information, and associated uncertainties and
22 limitations (such as those identified above) indicate with regard to the protection provided from
23 the array of O₃ welfare effects. As a whole, the decision found that this information did not
24 indicate the current standard to allow air quality conditions with implications of concern for the
25 public welfare. Based on all of the identified considerations, as well as consideration of advice
26 from the CASAC¹⁷ and public comment, and including consideration of the available evidence
27 and quantitative exposure/risk information, the Administrator concluded the current secondary
28 standard to be requisite to protect the public welfare from known or anticipated adverse effects
29 of O₃ and related photochemical oxidants in ambient air, and thus that the standard should be
30 retained without revision (85 FR 87345, December 31, 2020).

¹⁷ Among other things, in the 2020 letter communicating the CASAC’s comments on the 2019 draft PA, the CASAC advised EPA that it “finds, in agreement with the EPA, that the available evidence does not reasonably call into question the adequacy of the current secondary ozone standard and concurs that it should be retained” (Cox, 2020, p. 1). It further stated that the approach described in the draft PA to considering the evidence for welfare effects “is laid out very clearly, thoroughly discussed and documented, and provided a solid scientific underpinning for the EPA conclusion leaving the current secondary standard in place” (85 FR 87318-87319, December 31, 2020).

4.2 GENERAL APPROACH AND KEY ISSUES

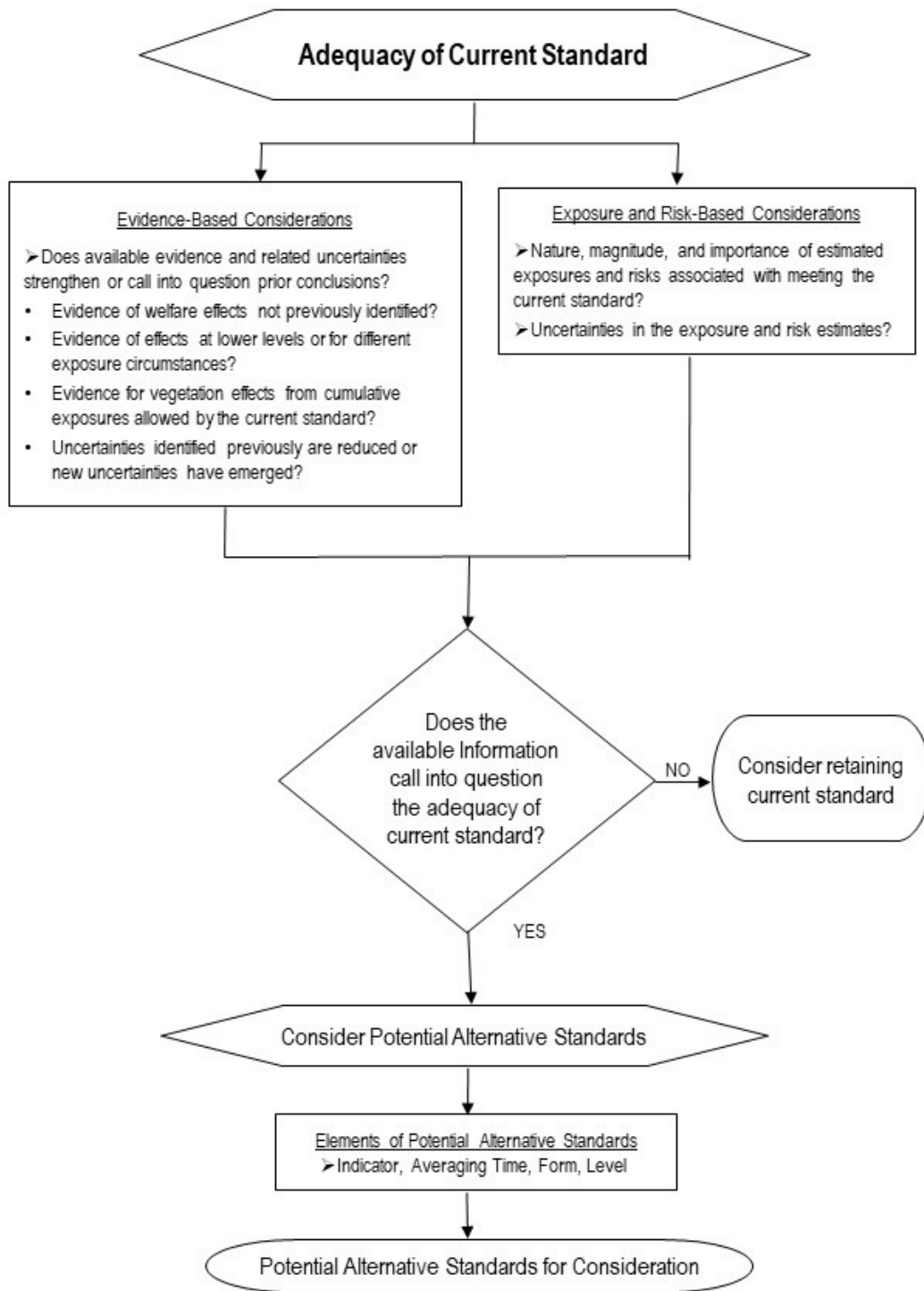
As in the case for secondary standard reviews, this reconsideration of the 2020 decision on the secondary standard is fundamentally based on using the Agency’s assessment of the scientific evidence and associated quantitative analyses to inform the Administrator’s judgments regarding a secondary standard that is requisite to protect the public welfare from known or anticipated adverse effects. This approach builds on the substantial assessments and evaluations performed over the course of O₃ NAAQS reviews to inform our understanding of the key-policy relevant issues in this reconsideration of the 2020 decision. As noted above, we are also considering the court’s 2019 decision on the O₃ secondary standard, particularly with regard to issues raised by the court in its remand of the standard (recognized in section 4.1.2 above) as was also done as part of the 2020 decision on the standard.

The evaluations in the PA, of the scientific assessments in the ISA (building on prior such assessments) augmented by quantitative air quality and exposure analyses, are intended to inform the Administrator’s public welfare policy judgments and conclusions, including his decisions regarding the O₃ standards. The PA considers the potential implications of various aspects of the scientific evidence, the air quality, exposure or risk-based information, and the associated uncertainties and limitations. Thus, the approach for this PA involves evaluating the available scientific and technical information to address a series of key policy-relevant questions using both evidence- and exposure/risk-based considerations. Together, consideration of the full set of evidence and information available will inform the answer to the following initial overarching question:

Do the available scientific evidence and exposure-/risk-based information support or call into question the adequacy of the public welfare protection afforded by the current secondary O₃ standard?

In reflecting on this question in the remaining sections of this chapter, we consider the body of scientific evidence assessed in the ISA, and considered as a basis for developing or interpreting air quality and exposure analyses, including whether it supports or calls into question the scientific conclusions reached in the 2020 review regarding welfare effects related to exposure to O₃ in ambient air. Information that may be informative to public policy judgments on the significance or adversity of key effects on the public welfare is also considered. Additionally, the available exposure and air quality information is considered, including with regard to the extent to which it may continue to support judgments made in previous reviews. Further, in considering this question with regard to the secondary O₃ standard, we give particular attention to exposures and risks for effects with the greatest potential for public welfare significance. Evaluation of the available scientific evidence and exposure/risk information with regard to consideration of the current standard and the overarching question above focuses on

1 key policy-relevant issues by addressing a series of questions on specific topics. Figure 3-1
2 summarizes, in general terms, the approach to considering the available information in the
3 context of policy-relevant questions pertaining to the secondary standard.
4



1
2 **Figure 4-1. Overview of general approach for the secondary O₃ standard.**

1 The Agency’s approach with regard to the O₃ secondary standard is consistent with the
2 requirements of the provisions of the CAA related to the review of NAAQS and with how the
3 EPA and the courts have historically interpreted these provisions. As discussed in section 1.2
4 above, these provisions require the Administrator to establish secondary standards that, in the
5 Administrator’s judgment, are requisite (i.e., neither more nor less stringent than necessary) to
6 protect the public welfare from known or anticipated adverse effects associated with the presence
7 of the pollutant in the ambient air. Consistent with the Agency’s approach across NAAQS
8 reviews, the approach of this PA to informing the Administrator’s judgments is based on a
9 recognition that the available evidence generally reflects continuums that include ambient air
10 exposures for which scientists generally agree that effects are likely to occur through lower
11 levels at which the likelihood and magnitude of response become increasingly uncertain. The
12 CAA does not require that standards be set at a zero-risk level, but rather at a level that reduces
13 risk sufficiently so as to protect the public welfare from known or anticipated adverse effects.

14 The Agency’s decisions on the adequacy of the current secondary standard and, as
15 appropriate, on any potential alternative standards considered in a review, are largely public
16 welfare policy judgments made by the Administrator. The four basic elements of the NAAQS
17 (i.e., indicator, averaging time, form, and level) are considered collectively in evaluating the
18 protection afforded by the current standard, or by any alternatives considered. Thus, the
19 Administrator’s final decisions in such reviews draw upon the scientific information and
20 analyses about welfare effects, environmental exposures and risks, and associated public welfare
21 significance, as well as judgments about how to consider the range and magnitude of
22 uncertainties that are inherent in the scientific evidence and analyses.

23 **4.3 WELFARE EFFECTS EVIDENCE**

24 The welfare effects evidence on which this PA for the reconsideration of the 2020
25 decision on the O₃ secondary standard will focus is the evidence described in the 2020 ISA and
26 prior ISAs or AQCDs. As described in section 1.5 above, the EPA has provisionally considered
27 more recently available studies that were raised in public comments in the 2020 review or were
28 identified in a literature search that the EPA conducted for this reconsideration of more recently
29 available controlled human exposure studies (Luben et al., 2020; Duffney et al. 2022). The
30 provisional consideration of these studies concluded that, taken in context, the associated
31 information and findings did not materially change any of the broad scientific conclusions of the
32 ISA regarding the health and welfare effects of O₃ in ambient air or warrant reopening the air
33 quality criteria for this review. Thus, the discussion below focuses on the welfare effects
34 evidence assessment, with associated conclusions, as described in the 2020 ISA.

1 4.3.1 Nature of Effects

2 The welfare effects evidence base includes more than fifty years of extensive research on
3 the phytotoxic effects of O₃, conducted both in and outside of the U.S., that documents the
4 impacts of O₃ on plants and their associated ecosystems (1978 AQCD, 1986 AQCD, 1996
5 AQCD, 2006 AQCD, 2013 ISA, 2020 ISA). As has been long established, O₃ can interfere with
6 carbon gain (photosynthesis) and allocation of carbon within the plant, making fewer
7 carbohydrates available for plant growth, reproduction, and/or yield (1996 AQCD, pp. 5-28 and
8 5-29). For seed-bearing plants, reproductive effects can include reduced seed or fruit production
9 or yield. The strongest evidence for effects from O₃ exposure on vegetation was recognized at
10 the time of the 2015 review to be from controlled exposure studies, which “have clearly shown
11 that exposure to O₃ is causally linked to visible foliar injury, decreased photosynthesis, changes
12 in reproduction, and decreased growth” in many species of vegetation (2013 ISA, p. 1-15). Such
13 effects at the plant scale can also be linked to an array of effects at larger spatial scales (and
14 higher levels of biological organization), with the evidence available in the 2015 review
15 indicating that “O₃ exposures can affect ecosystem productivity, crop yield, water cycling, and
16 ecosystem community composition” (2013 ISA, p. 1-15, Chapter 9, section 9.4). Beyond its
17 effects on plants, the evidence in the 2015 review also recognized O₃ in the troposphere as a
18 major greenhouse gas (ranking behind carbon dioxide and methane in importance), with
19 associated radiative forcing and effects on climate, with accompanying “large uncertainties in the
20 magnitude of the radiative forcing estimate ... making the impact of tropospheric O₃ on climate
21 more uncertain than the effect of the longer-lived greenhouse gases (2013 ISA, sections 10.3.4
22 and 10.5.1 [p. 10-30]).

- 23 • **Does the available evidence alter prior conclusions regarding the nature of welfare**
24 **effects attributable to O₃ in ambient air? Is there new evidence on welfare effects**
25 **beyond those identified in the 2015 review?**

26 The available evidence supports, sharpens, and expands somewhat on the conclusions
27 reached in the 2015 review (ISA, Appendices 8 and 9). Consistent with the previously available
28 evidence, the available evidence describes an array of O₃ effects on vegetation and related
29 ecosystem effects, as well as the role of tropospheric O₃ in radiative forcing and subsequent
30 climate-related effects. The ISA concludes there to be causal relationships between O₃ and
31 visible foliar injury, reduced vegetation growth and reduced plant reproduction,¹⁸ as well as
32 reduced yield and quality of agricultural crops, reduced productivity in terrestrial ecosystems,

¹⁸ The 2013 ISA did not include a separate causality determination for reduced plant reproduction. Rather, it was included with the conclusion of a causal relationship of O₃ with reduced vegetation growth (ISA, Table IS-12).

1 alteration of terrestrial community composition¹⁹, and alteration of belowground biogeochemical
2 cycles (ISA, section IS.5). The ISA also concludes there likely to be a causal relationship
3 between O₃ and alteration of ecosystem water cycling, reduced carbon sequestration in terrestrial
4 ecosystems, and with increased tree mortality (ISA, section IS.5). Additionally, newly available
5 evidence in the 2020 ISA augments more limited previously available evidence related to insect
6 interactions with vegetation, contributing to the ISA conclusion that the evidence is sufficient to
7 infer that there are likely to be causal relationships between O₃ exposure and alteration of plant-
8 insect signaling (ISA, Appendix 8, section 8.7) and of insect herbivore growth and reproduction
9 (ISA, Appendix 8, section 8.6). Thus, prior conclusions continue to be supported and conclusions
10 are also reached in the 2020 ISA for a few new areas based on the now expanded evidence.

11 As in the 2015 review, the strongest evidence and the associated findings of causal or
12 likely causal relationships with O₃ in ambient air, and the quantitative characterizations of
13 relationships between O₃ exposure and occurrence and magnitude of effects are for vegetation
14 effects. The scales of these effects range from the individual plant scale to the ecosystem scale,
15 with potential for impacts on the public welfare (as discussed in section 4.3.2 below). The
16 following summary addresses the identified vegetation-related effects of O₃ across these scales.

17 Visible foliar injury has long been used as a bioindicator of O₃ exposures, although it is
18 not always a reliable indicator of other negative effects on vegetation (ISA, sections IS.5.1.2 and
19 8.2, and Appendix 8, section 8.2; 2013 ISA, section 9.4.2; 2006 AQCD, 1996 AQCD, 1986
20 AQCD, 1978 AQCD). More specifically, ozone-induced visible foliar injury symptoms on
21 certain tree and herbaceous species, such as black cherry, yellow-poplar and common milkweed,
22 have long been considered diagnostic of exposure to elevated O₃ based on the consistent
23 association established with experimental evidence (ISA, Appendix 8, section 8.2; 2013 ISA, p.
24 1-10).²⁰ The available evidence, consistent with that in past reviews, indicates that “visible foliar
25 injury usually occurs when sensitive plants are exposed to elevated ozone concentrations in a
26 predisposing environment,” with a major factor for such an environment being the amount of soil
27 moisture available to the plant (ISA, Appendix 8, p. 8-23; 2013 ISA, section 9.4.2). The
28 significance of O₃ injury at the leaf and whole plant levels also depends on an array of factors
29 that include the amount of total leaf area affected, age of plant, size, developmental stage, and
30 degree of functional redundancy among the existing leaf area (ISA, Appendix 8, section 8.2;

¹⁹ The 2013 ISA concluded alteration of terrestrial community composition to be likely causally related to O₃ based on the then available information (ISA, Table IS-12).

²⁰ As described in the ISA, “[t]ypical types of visible injury to broadleaf plants include stippling, flecking, surface bleaching, bifacial necrosis, pigmentation (e.g., bronzing), and chlorosis or premature senescence and [t]ypical visible injury symptoms for conifers include chlorotic banding, tip burn, flecking, chlorotic mottling, and premature senescence of needles” (ISA, Appendix 8, p. 8-13).

1 2013 ISA, section 9.4.2). Such modifying factors contribute to the difficulty in quantitatively
2 relating visible foliar injury to other vegetation effects (e.g., individual tree growth, or effects at
3 population or ecosystem levels), such that visible foliar injury “is not always a reliable indicator
4 of other negative effects on vegetation” (ISA, Appendix 8, section 8.2; 2013 ISA, p. 9-39).²¹

5 Effects of O₃ on physiology of individual plants at the cellular level, such as through
6 photosynthesis and carbon allocation, can impact plant growth and reproduction (ISA, section
7 IS.5.1.2, Appendix 8, sections 8.3 and 8.4; 2013 ISA, p. 9-42). The available studies come from
8 a variety of different study types that cover an array of different species, effects endpoints, and
9 exposure methods and durations. In addition to studies on scores of plant species that have found
10 O₃ to reduce plant growth, the evidence accumulated over the past several decades documents O₃
11 alteration of allocation of biomass within the plant and plant reproduction (ISA, Appendix 8,
12 sections 8.3 and 8.4; 2013 ISA, p. 1-10). The biological mechanisms underlying the effect of O₃
13 on plant reproduction include “both direct negative effects on reproductive tissues and indirect
14 negative effects that result from decreased photosynthesis and other whole plant physiological
15 changes” (ISA, section IS.5.1.2). A newly available meta-analysis of more than 100 studies
16 published between 1968 and 2010 summarizes effects of O₃ on multiple measures of
17 reproduction (ISA, Appendix 8, section 8.4.1).

18 Studies involving experimental field sites have also reported effects on measures of plant
19 reproduction, such as effects on seeds (reduced weight, germination, and starch levels) that could
20 lead to a negative impact on species regeneration in subsequent years, and bud size that might
21 relate to a delay in spring leaf development (ISA, Appendix 8, section 8.4; 2013 ISA, section
22 9.4.3; Darbah et al., 2007, Darbah et al., 2008). A more recent laboratory study reported 6-hour
23 daily O₃ exposures of flowering mustard plants to 100 ppb during different developmental stages
24 to have mixed effects on reproductive metrics. While flowers exposed early *versus* later in
25 development produced shorter fruits, the number of mature seeds per fruit was not significantly
26 affected by flower developmental stage of exposure (ISA, Appendix 8, section 8.4.1; Black et al.,
27 2012). Another study assessed seed viability for a flowering plant in laboratory and field

²¹ Similar to the 2013 ISA, the 2020 ISA states the following (ISA, pp. 8-23 to 8-24).

Although visible injury is a valuable indicator of the presence of phytotoxic concentrations of ozone in ambient air, it is not always a reliable indicator of other negative effects on vegetation [e.g., growth, reproduction; U.S. EPA (2013)]. The significance of ozone injury at the leaf and whole-plant levels depends on how much of the total leaf area of the plant has been affected, as well as the plant’s age, size, developmental stage, and degree of functional redundancy among the existing leaf area (U.S. EPA, 2013). Previous ozone AQCDs have noted the difficulty in relating visible foliar injury symptoms to other vegetation effects, such as individual plant growth, stand growth, or ecosystem characteristics (U.S. EPA, 2006, 1996). Thus, it is not presently possible to determine, with consistency across species and environments, what degree of injury at the leaf level has significance to the vigor of the whole plant.

1 conditions, finding effects on seed viability of O₃ exposures (90 and 120 ppb) under laboratory
2 conditions but less clear effects under more field-like conditions (ISA, Appendix 8, section 8.4.1;
3 Landesmann et al., 2013).

4 With regard to agricultural crops, the current evidence base, as in the 2015 review, is
5 sufficient to infer a causal relationship between O₃ exposure and reduced yield and quality (ISA,
6 section IS.5.1.2). The evidence in the current ISA is augmented by new research in a number of
7 areas, including studies on soybean, wheat, and other non-soy legumes. The new information
8 assessed in the ISA remains consistent with the conclusions reached in the 2013 ISA (ISA,
9 section IS.5.1.2).

10 The evidence base for trees includes a number of studies conducted at the Aspen free-air
11 carbon-dioxide and ozone enrichment (FACE) experiment site in Wisconsin (that operated from
12 1998 through 2011) and also available in the 2015 review (ISA, IS.5.1 and Appendix 8, section
13 8.1.2.1; 2013 ISA, section 9.2.4). These studies, which occurred in a field setting (more similar
14 to natural forest stands than open-top-chamber studies), reported reduced tree growth when
15 grown in single or three species stands within 30-m diameter rings and exposed over one or more
16 years to elevated O₃ concentrations (hourly concentrations 1.5 times concentrations in ambient
17 air at the site) compared to unadjusted ambient air concentrations (2013 ISA, section 9.4.3;
18 Kubiske et al., 2006, Kubiske et al., 2007).²²

19 With regard to tree mortality, the 2013 ISA did not include a determination of causality
20 (ISA, Appendix 8, section 8.4). While the then-available evidence included studies identifying
21 ozone as a contributor to tree mortality, which contributed to the 2013 conclusion regarding O₃
22 and alteration of community composition (2013 ISA, section 9.4.7.4), a separate causality
23 determination regarding O₃ and tree mortality was not assessed (ISA, Appendix 8, section 8.4;
24 2013 ISA, Table 9-19). The evidence assessed in the 2013 ISA (and 2006 AQCD) was largely
25 observational, including studies that reported declines in conifer forests for which elevated O₃
26 was identified as contributor but in which a variety of environmental factors may have also
27 played a role (2013 ISA, section 9.4.7.1; 2006 AQCD, sections AX9.6.2.1, AX9.6.2.2,
28 AX9.6.2.6, AX9.6.4.1 and AX9.6.4.2). Since the 2015 review, three additional studies are now
29 available (ISA, Appendix 8, Table 8-9). Two of these are analyses of field observations, one of
30 which is set in the Spanish Pyrenees.²³ A second study is a large-scale empirical statistical

²² Seasonal (92-day) W126 index values for unadjusted O₃ concentrations over six years of the Aspen FACE experiments ranged from 2 to 3 ppm-hrs, while the elevated exposure concentrations (reflecting addition of O₃ to ambient air concentrations) ranged from somewhat above 20 to somewhat above 35 ppm-hrs (ISA, Appendix 8, Figure 8-17).

²³ The concentration gradient with altitude in the Spanish study, includes - at the highest site - annual average April-to-September O₃ concentrations for the 2004 to 2007 period that range up to 74 ppb (Diaz-de-Quijano et al., 2016), indicating O₃ concentrations likely to exceed the current U.S. secondary standard.

1 analysis of factors potentially contributing to tree mortality in eastern and central U.S. forests
2 during the 1971-2005 period, which reported O₃ (county-level 11-year [1996-2006] average 8
3 hour metric)²⁴ to be ninth among the 13 potential factors assessed²⁵ and to have a significant
4 positive correlation with tree mortality (ISA, section IS.5.2, Appendix 8, section 8.4.3; Dietze
5 and Moorcroft, 2011). A newly available experimental study also reported increased mortality in
6 two of five aspen genotypes grown in mixed stands under elevated O₃ concentrations (ISA,
7 section IS.5.1.2; Moran and Kubiske, 2013). Coupled with the plant-level evidence of
8 phytotoxicity discussed above, as well as consideration of community composition effects, this
9 evidence was concluded to indicate the potential for elevated O₃ concentrations to contribute to
10 tree mortality (ISA, section IS.5.1.2 and Appendix 8, sections 8.4.3 and 8.4.4). Based on the
11 available evidence, the ISA concludes there is likely to be a causal relationship between O₃ and
12 increased tree mortality (ISA, Table IS-2, Appendix 8, section 8.4.4).

13 A variety of factors in natural environments can either mitigate or exacerbate predicted
14 O₃-plant interactions and are recognized sources of uncertainty and variability. Such factors at
15 the plant level include multiple genetically influenced determinants of O₃ sensitivity, changing
16 sensitivity to O₃ across vegetative growth stages, co-occurring stressors and/or modifying
17 environmental factors (ISA, Appendix 8, section 8.12).

18 Ozone-induced effects at the scale of the whole plant have the potential to translate to
19 effects at the ecosystem scale, such as reduced productivity and carbon storage, and altered
20 terrestrial community composition, as well as impacts on ecosystem functions, such as
21 belowground biogeochemical cycles and ecosystem water cycling. For example, under the
22 relevant exposure conditions, O₃-related reduced tree growth and reproduction, as well as
23 increased mortality, could lead to reduced ecosystem productivity. Recent studies from the
24 Aspen FACE experiment and modeling simulations indicate that O₃-related negative effects on
25 ecosystem productivity may be temporary or may be limited in some systems (ISA, Appendix 8,
26 section 8.8.1). Previously available studies had reported impacts on productivity in some forest
27 types and locations, such as ponderosa pine in southern California and other forest types in the
28 mid-Atlantic region (2013 ISA, section 9.4.3.4). Through reductions in sensitive species growth,

²⁴ As indicated in Figures 2-11 and 2-12, annual fourth highest daily maximum 8-hour O₃ concentrations in these regions were above 80 ppb in the early 2000s and the median design values at national trend sites was nearly 85 ppb.

²⁵ This statistical analysis, which utilized datasets from within the 1971-2005 period, included an examination of the sensitivity of predicted mortality rate to 13 different covariates. On average across the predictions for 10 groups of trees (based on functional type and major representative species), the order of mortality rate sensitivity to the covariates, from highest to lowest, was: sulfate deposition, tree diameter, nitrate deposition, summer temperature, tree age, elevation, winter temperature, precipitation, O₃ concentration, tree basal area, topographic moisture index, slope and topographic radiation index (Dietze and Moorcroft, 2011).

1 and related ecosystem productivity, O₃ could lead to reduced ecosystem carbon storage (ISA,
2 IS.5.1.4; 2013 ISA, section 9.4.3). With regard to forest community composition, available
3 studies have reported changes in tree communities composed of species with relatively greater
4 and relatively lesser sensitivity to O₃, such as birch and aspen, respectively (ISA, section
5 IS.5.1.8.1, Appendix 8, section 8.10; 2013 ISA, section 9.4.3; Kubiske et al., 2007). As the ISA
6 concludes, “[t]he extent to which ozone affects terrestrial productivity will depend on more than
7 just community composition, but other factors, which both directly influence [net primary
8 productivity] (i.e., availability of N and water) and modify the effect of ozone on plant growth”
9 (ISA, Appendix 8, section 8.8.1). Thus, the magnitude of O₃ impact on ecosystem productivity,
10 as on forest composition, can vary among plant communities based on several factors, including
11 the type of stand or community in which the sensitive species occurs (e.g., single species *versus*
12 mixed canopy), the role or position of the species in the stand (e.g., dominant, sub-dominant,
13 canopy, understory), and the sensitivity of co-occurring species and environmental factors (e.g.,
14 drought and other factors).

15 The effects of O₃ on plants and plant populations also have implications for other
16 ecosystem functions. Two such functions, effects with which O₃ is concluded to be likely
17 causally or causally related, are ecosystem water cycling and belowground biogeochemical
18 cycles, respectively (ISA, Appendix 8, sections 8.11 and 8.9). With regard to the former, the
19 effects of O₃ on plants (e.g., *via* stomatal control, as well as leaf and root growth and changes in
20 wood anatomy associated with water transport) can affect ecosystem water cycling through
21 impacts on root uptake of soil moisture and groundwater as well as transpiration through leaf
22 stomata to the atmosphere (ISA, Appendix 8, section 8.11.1). These “impacts may in turn affect
23 the amount of water moving through the soil, running over land or through groundwater and
24 flowing through streams” (ISA, Appendix 8, section 8.11, p. 8-161). Evidence newly available
25 for the 2020 ISA is supportive of previously available evidence in this regard (ISA, Appendix 8,
26 section 8.11.6). This evidence, including that newly available, indicates the extent to which the
27 effects of O₃ on plant leaves and roots (e.g., through effects on chemical composition and
28 biomass) can impact belowground biogeochemical cycles involving root growth, soil food web
29 structure, soil decomposer activities, soil microbial respiration, soil carbon turnover, soil water
30 cycling and soil nutrient cycling (ISA, Appendix 8, section 8.9).

31 Additional vegetation- and insect-related effects with implications beyond individual
32 plants include the effects of O₃ on insect herbivore growth and reproduction and plant-insect
33 signaling (ISA, Table IS-12, Appendix 8, sections 8.6 and 8.7). With regard to insect herbivore
34 growth and reproduction, the evidence includes multiple effects in an array of insect species,
35 although without a consistent pattern of response for most endpoints (ISA, Appendix 8, Table 8-

1 11). As was also the case with the studies available at the time of the 2015 review,²⁶ in the newly
2 available studies the individual-level responses are highly context- and species-specific and not
3 all species tested showed a response (ISA, p. IS-64, Table IS-12, section IS.5.1.3 and Appendix
4 8, section 8.6). Evidence on plant-insect signaling comes from laboratory, greenhouse, open top
5 chambers (OTC) and FACE experiments (ISA, section IS.5.1.3 and Appendix 8, section 8.7).
6 The available evidence indicates a role for elevated O₃ in altering and degrading emissions of
7 chemical signals from plants and reducing detection of volatile plant signaling compounds
8 (VPSCs) by insects, including pollinators. Elevated O₃ concentrations degrade some VPSCs
9 released by plants, potentially affecting ecological processes including pollination and plant
10 defenses against herbivory. Further, the available studies report elevated O₃ conditions to be
11 associated with plant VPSC emissions that may make a plant either more attractive or more
12 repellant to herbivorous insects, and to predators and parasitoids that target phytophagous (plant-
13 eating) insects (ISA, section IS.5.1.3 and Appendix 8, section 8.7).

14 Ozone welfare effects also extend beyond effects on vegetation and associated biota due
15 to it being a major greenhouse gas and radiative forcing agent.²⁷ As in the 2015 review, the
16 available evidence, augmented since the 2013 ISA, continues to support a causal relationship
17 between the global abundance of O₃ in the troposphere and radiative forcing, and a likely causal
18 relationship between the global abundance of O₃ in the troposphere and effects on temperature,
19 precipitation, and related climate variables²⁸ (ISA, section IS.5.2 and Appendix 9; Myhre et al.,
20 2013). As was also true at the time of the 2015 review, tropospheric O₃ has been ranked third in
21 importance for global radiative forcing, after carbon dioxide and methane, with the radiative
22 forcing of O₃ since pre-industrial times estimated to be about 25 to 40% of the total warming
23 effects of anthropogenic carbon dioxide and about 75% of the effects of anthropogenic methane
24 (ISA, Appendix 9, section 9.1.3.3). Uncertainty in the magnitude of radiative forcing estimated
25 to be attributed to tropospheric O₃ is a contributor to the relatively greater uncertainty associated
26 with climate effects of tropospheric O₃ compared to such effects of the well mixed greenhouse
27 gases, such as carbon dioxide and methane (ISA, section IS.6.2.2).

²⁶ During the 2015 review, the 2013 ISA stated with regard to O₃ effects on insects and other wildlife that “there is no consensus on how these organisms respond to elevated O₃ (2013 ISA, section 9.4.9.4, p. 9-98).

²⁷ Radiative forcing is a metric used to quantify the change in balance between radiation coming into and going out of the atmosphere caused by the presence of a particular substance. The ISA describes it more specifically as “a perturbation in net radiative flux at the tropopause (or top of the atmosphere) caused by a change in radiatively active forcing agent(s) after stratospheric temperatures have readjusted to radiative equilibrium (stratospherically adjusted RF)” (ISA, Appendix 9, section 9.1.3.3).

²⁸ Effects on temperature, precipitation, and related climate variables were referred to as “climate change” or “effects on climate” in the 2013 ISA (ISA, p. IS-82; 2013 ISA, pp. 1-14, 10-31).

1 Lastly, the evidence regarding tropospheric O₃ and UV-B shielding was evaluated in the
2 2013 ISA and determined to be inadequate to draw a causal conclusion (2013 ISA, section
3 10.5.2). The current ISA concludes there to be no new evidence since the 2013 ISA relevant to
4 the question of UV-B shielding by tropospheric O₃ (ISA, IS.1.2.1 and Appendix 9, section
5 9.1.3.4).

6 **4.3.2 Public Welfare Implications**

7 The public welfare implications of the evidence regarding O₃ welfare effects are
8 dependent on the type and severity of the effects, as well as the extent of the effect at a particular
9 biological or ecological level of organization. We discuss such factors here in light of judgments
10 and conclusions made in NAAQS reviews regarding effects on the public welfare.

11 As provided in section 109(b)(2) of the CAA, the secondary standard is to “specify a
12 level of air quality the attainment and maintenance of which in the judgment of the
13 Administrator ... is requisite to protect the public welfare from any known or anticipated adverse
14 effects associated with the presence of such air pollutant in the ambient air.” The secondary
15 standard is not meant to protect against all known or anticipated O₃-related welfare effects, but
16 rather those that are judged to be adverse to the public welfare, and a bright-line determination of
17 adversity is not required in judging what is requisite (78 FR 3212, January 15, 2013; 80 FR
18 65376, October 26, 2015; see also 73 FR 16496, March 27, 2008). Thus, the level of protection
19 from known or anticipated adverse effects to public welfare that is requisite for the secondary
20 standard is a public welfare policy judgment made by the Administrator. The Administrator’s
21 judgment regarding the available information and adequacy of protection provided by an existing
22 standard is generally informed by considerations in prior reviews and associated conclusions.

- 23 • **Is there newly available information relevant to consideration of the public welfare**
24 **implications of O₃-related welfare effects?**

25 The categories of effects identified in the CAA to be included among welfare effects are
26 quite diverse,²⁹ and among these categories, any single category includes many different types of
27 effects that are of broadly varying specificity and level of resolution. For example, effects on
28 vegetation is a category identified in CAA section 302(h), and the ISA recognizes numerous
29 vegetation-related effects of O₃ at the organism, population, community, and ecosystem level, as
30 summarized in section 4.3.1 above (ISA, Appendix 8). The significance of each type of
31 vegetation-related effect with regard to potential effects on the public welfare depends on the

²⁹ Section 302(h) of the CAA states that language referring to “effects on welfare” in the CAA “includes, but is 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” (CAA section 302(h)).

1 type and severity of effects, as well as the extent of such effects on the affected environmental
2 entity, and on the societal use of the affected entity and the entity’s significance to the public
3 welfare. Such factors have been considered in the context of judgments and conclusions made in
4 some prior reviews regarding public welfare effects. For example, judgments regarding public
5 welfare significance in two prior O₃ NAAQS decisions gave particular attention to O₃ effects in
6 areas with special federal protections (such as Class I areas), and lands set aside by states, tribes
7 and public interest groups to provide similar benefits to the public welfare (73 FR 16496, March
8 27, 2008; 80 FR 65292, October 26, 2015).³⁰ In the 2015 review, the EPA recognized the “clear
9 public interest in and value of maintaining these areas in a condition that does not impair their
10 intended use and the fact that many of these lands contain O₃-sensitive species” (73 FR 16496,
11 March 27, 2008).

12 Judgments regarding effects on the public welfare can depend on the intended use for, or
13 service (and value) of, the affected vegetation, ecological receptors, ecosystems and resources
14 and the significance of that use to the public welfare (73 FR 16496, March 27, 2008; 80 FR
15 65377, October 26, 2015). Uses or services provided by areas that have been afforded special
16 protection can flow in part or entirely from the vegetation that grows there. Uses or services
17 provided by areas that have been afforded special protection can flow in part or entirely from the
18 vegetation that grows there. Ecosystem services range from those directly related to the natural
19 functioning of the ecosystem to ecosystem uses for human recreation or profit, such as through
20 the production of lumber or fuel (Costanza et al., 2017; ISA, section IS.5.1). Services of aesthetic
21 value and outdoor recreation depend, at least in part, on the perceived scenic beauty of the
22 environment. Additionally, public surveys have indicated that Americans rank as very important
23 the existence of resources, the option or availability of the resource and the ability to bequest or
24 pass it on to future generations (Cordell et al., 2008). The spatial, temporal, and social
25 dimensions of public welfare impacts are also influenced by the type of service affected. For
26 example, a national park can provide direct recreational services to the thousands of visitors that
27 come each year, but also provide an indirect value to the millions who may not visit but receive

³⁰ For example, the fundamental purpose of parks in the National Park System “is to conserve the scenery, natural and historic objects, and wild life in the System units and to provide for the enjoyment of the scenery, natural and historic objects, and wild life in such manner and by such means as will leave them unimpaired for the enjoyment of future generations” (54 U.S.C. 100101). Additionally, the Wilderness Act of 1964 defines designated “wilderness areas” in part as areas “protected and managed so as to preserve [their] natural conditions” and requires that these areas “shall be administered for the use and enjoyment of the American people in such manner as will leave them unimpaired for future use and enjoyment as wilderness, and so as to provide for the protection of these areas, [and] the preservation of their wilderness character ...” (16 U.S.C. 1131 (a) and (c)). Other lands that benefit the public welfare include national forests which are managed for multiple uses including sustained yield management in accordance with land management plans (see 16 U.S.C. 1600(1)-(3); 16 U.S.C. 1601(d)(1)).

1 satisfaction from knowing it exists and is preserved for the future (80 FR 65377, October 26,
2 2015).

3 The different types of effects on vegetation discussed in section 4.3.1 above differ with
4 regard to aspects important to judging their public welfare significance. For example, in the case
5 of crop yield loss, such judgments may consider aspects such as the heavy management of
6 agriculture in the U.S., while judgments for other categories of effects may generally relate to
7 considerations regarding natural areas, including specifically those areas that are not managed
8 for harvest. For example, effects on tree growth and reproduction, and also visible foliar injury,
9 have the potential to be significant to the public welfare through impacts in Class I and other
10 areas given special protection in their natural/existing state, although they differ in how they
11 might be significant.

12 In this context, it may be important to consider that O₃ effects on tree growth and
13 reproduction could, depending on severity, extent, and other factors, lead to effects on a larger
14 scale including reduced productivity, altered forest and forest community (plant, insect and
15 microbe) composition, reduced carbon storage and altered ecosystem water cycling (ISA, section
16 IS.5.1.8.1; 2013 ISA, Figure 9-1, sections 9.4.1.1 and 9.4.1.2). For example, the composition of
17 plants and other members of terrestrial communities can be affected through O₃ effects on
18 growth and reproductive success of sensitive plant species in the community, with the extent of
19 compositional changes dependent on factors such as competitive interactions (ISA, section
20 IS.5.1.8.1; 2013 ISA, sections 9.4.3 and 9.4.3.1). Impacts on some of these characteristics (e.g.,
21 forest or forest community composition) may be considered of greater public welfare
22 significance when occurring in Class I or other protected areas, due to value for particular
23 services that the public places on such areas.

24 Agriculture and silviculture provide ecosystem services with clear public welfare
25 benefits. With regard to agriculture-related effects, however, there are complexities in this
26 consideration related to areas and plant species that are heavily managed to obtain a particular
27 output (such as commodity crops or commercial timber production). In light of this, the degree to
28 which O₃ impacts on vegetation that could occur in such areas and on such species would impair
29 the intended use at a level that might be judged adverse to the public welfare has been less clear
30 (80 FR 65379, October 26, 2015; 73 FR 16497, March 27, 2008). While having sufficient crop
31 yields is of high public welfare value, important commodity crops are typically heavily managed
32 to produce optimum yields. Moreover, based on the economic theory of supply and demand,
33 increases in crop yields would be expected to result in lower prices for affected crops and their
34 associated goods, which would primarily benefit consumers. Analyses in past reviews have
35 described how these competing impacts on producers and consumers complicate consideration of

1 these effects in terms of potential adversity to the public welfare (2014 WREA, sections 5.3.2
2 and 5.7).

3 Other ecosystem services valued by people that can be affected by reduced tree growth,
4 productivity and associated forest effects include aesthetic value, food, fiber, timber, other forest
5 products, habitat, recreational opportunities, climate and water regulation, erosion control, air
6 pollution removal, and desired fire regimes, as summarized in Figure 4-2 (ISA, section IS.5.1;
7 2013 ISA, sections 9.4.1.1 and 9.4.1.2). In considering such services in past reviews, the Agency
8 the Agency has given particular attention to effects in natural ecosystems, indicating that a
9 protective standard, based on consideration of effects in natural ecosystems in areas afforded
10 special protection, would also “provide a level of protection for other vegetation that is used by
11 the public and potentially affected by O₃ including timber, produce grown for consumption and
12 horticultural plants used for landscaping” (80 FR 65403, October 26, 2015). For example,
13 locations potentially vulnerable to O₃-related impacts might include forested lands, both public
14 and private, where trees are grown for timber production. Forests in urbanized areas also provide
15 a number of services that are important to the public in those areas, such as air pollution removal,
16 cooling, and beautification. There are also many other tree species, such as various ornamental
17 and agricultural species (e.g., Christmas trees, fruit and nut trees), that provide ecosystem
18 services that may be judged important to the public welfare.

19 With its effect on the physical appearance of plants, visible foliar injury has the potential
20 to be significant to the public welfare, depending on its severity and spatial extent, by impacting
21 aesthetic or scenic values and outdoor recreation in Class I and other similarly protected areas
22 valued by the public.³¹ To assess evidence of injury to plants in forested areas on national and
23 regional scales, the U.S. Forest Service (USFS) conducted surveys of the occurrence and severity
24 of visible foliar injury on sensitive (bioindicator) species at biomonitoring sites across most of
25 the U.S., beginning in 1994 (in the eastern U.S.) and extending through 2011 (Smith, 2012;
26 Coulston et al., 2003). At these sites (biosites), a national protocol, including verification and
27 quality assurance procedures and a scoring system, was implemented. The resultant biosite index
28 (BI) scores may be described with regard to one of several categories ranging from little or no
29 foliar injury to severe injury. For example, BI scores of zero to five are described as “little or no

³¹ For example, although analyses specific to visible foliar injury are of limited availability, there have been analyses developing estimates of recreation value damages of severe impacts related to other types of forest effects, such as tree mortality due to bark beetle outbreaks (e.g., Rosenberger et al., 2013). Such analyses estimate reductions in recreational use when the damage is severe (e.g., reductions in the density of live, robust trees). Such damage would reasonably be expected to also reflect damage indicative of injury with which a relationship with other plant effects (e.g., growth and reproduction) would be also expected. Similarly, a couple of studies from the 1970s and 1980s indicated potential for differences in recreational use for areas with stands of pine in which moderate to severe injury was apparent from 30 or 40 feet (1996 AQCD, section 5.8.3).

1 foliar injury,” scores above five to 15 as “low” or “light to moderate” foliar injury, scores from
2 15 to 25 as “moderate foliar injury” and scores above 25 as “severe injury” (Campbell et al.,
3 2007; Smith et al., 2007; Smith, 2012).³² However, available information does not yet address or
4 describe the relationships expected to exist between some level of injury severity (e.g., little,
5 low/light, moderate or severe) and/or spatial extent affected and scenic or aesthetic values. This
6 gap impedes consideration of the public welfare implications of different injury severities, and
7 accordingly judgments on the potential for public welfare significance. That notwithstanding,
8 while minor spotting on a few leaves of a plant may easily be concluded to be of little public
9 welfare significance, some level of severity and widespread occurrence of visible foliar injury,
10 particularly if occurring in specially protected areas, where the public can be expected to place
11 value (e.g., for recreational uses), might reasonably be concluded to impact the public welfare.

12 The tropospheric O₃-related effects of radiative forcing and subsequent effects on
13 temperature, precipitation and related climate variables also have important public welfare
14 implications, although their quantitative evaluation in response to O₃ concentrations in the U.S.
15 is complicated by “[c]urrent limitations in climate modeling tools, variation across models, and
16 the need for more comprehensive observational data on these effects” (ISA, section IS.6.2.2). An
17 ecosystem service provided by forested lands is carbon sequestration or storage (ISA, section
18 IS.5.1.4 and Appendix 8, section 8.8.3; 2013 ISA, section 2.6.2.1 and p. 9-37),³³ which has an
19 extremely valuable role in counteracting the impact of greenhouse gases on radiative forcing and
20 related climate effects on the public welfare. Accordingly, the service of carbon storage can be of
21 paramount importance to the public welfare no matter in what location the trees are growing or
22 what their intended current or future use (e.g., 2013 ISA, section 9.4.1.2). The benefit exists as
23 long as the trees are growing, regardless of what additional functions and services it provides.

24 Categories of effects newly identified as likely causally related to O₃ in ambient air, such
25 as alteration of plant-insect signaling and insect herbivore growth and reproduction, also have
26 potential public welfare implications, e.g., given the role of the plant-insect signaling process in
27 pollination and seed dispersal (ISA, section IS.5.1.3). Uncertainties and limitations in the
28 evidence (e.g., as summarized in sections 4.3.3.3 and 4.3.4 below) preclude an assessment of the
29 extent and magnitude of O₃ effects on these endpoints, which thus also precludes an evaluation
30 of the potential for associated public welfare implications.

³² Authors of studies presenting USFS biomonitoring program data have suggested what might be considered “assumptions of risk” (e.g., for the forest resource) related to scores in these categories, e.g., none, low, moderate and high for BI scores of zero to five, five to 15, 15 to 25 and above 25, respectively (e.g., Smith et al., 2003; Smith et al., 2012). For example, maps of localized moderate to high risk areas may be used to identify areas where more detailed evaluations are warranted (Smith et al., 2012).

³³ While carbon sequestration or storage also occurs for vegetated ecosystems other than forests, it is relatively larger in forests given the relatively greater biomass for trees compared to some other plants.

1 In summary, several considerations are recognized as important to judgments on the
2 public welfare significance of the array of welfare effects of different O₃ exposure conditions.
3 These include uncertainties and limitations associated with the consideration of the magnitude of
4 key vegetation effects that might be concluded to be adverse to ecosystems and associated
5 services. Additionally, the presence of O₃-sensitive tree species may contribute to a vulnerability
6 of numerous locations to public welfare impacts from O₃ related to tree growth, productivity and
7 carbon storage and their associated ecosystems and services. Other important considerations
8 include the exposure circumstances that may elicit effects and the potential for the significance
9 of the effects to vary in specific situations due to differences in sensitivity of the exposed
10 species, the severity and associated significance of the observed or predicted O₃-induced effect,
11 the role that the species plays in the ecosystem, the intended use of the affected species and its
12 associated ecosystem and services, the presence of other co-occurring predisposing or mitigating
13 factors, and associated uncertainties and limitations.
14

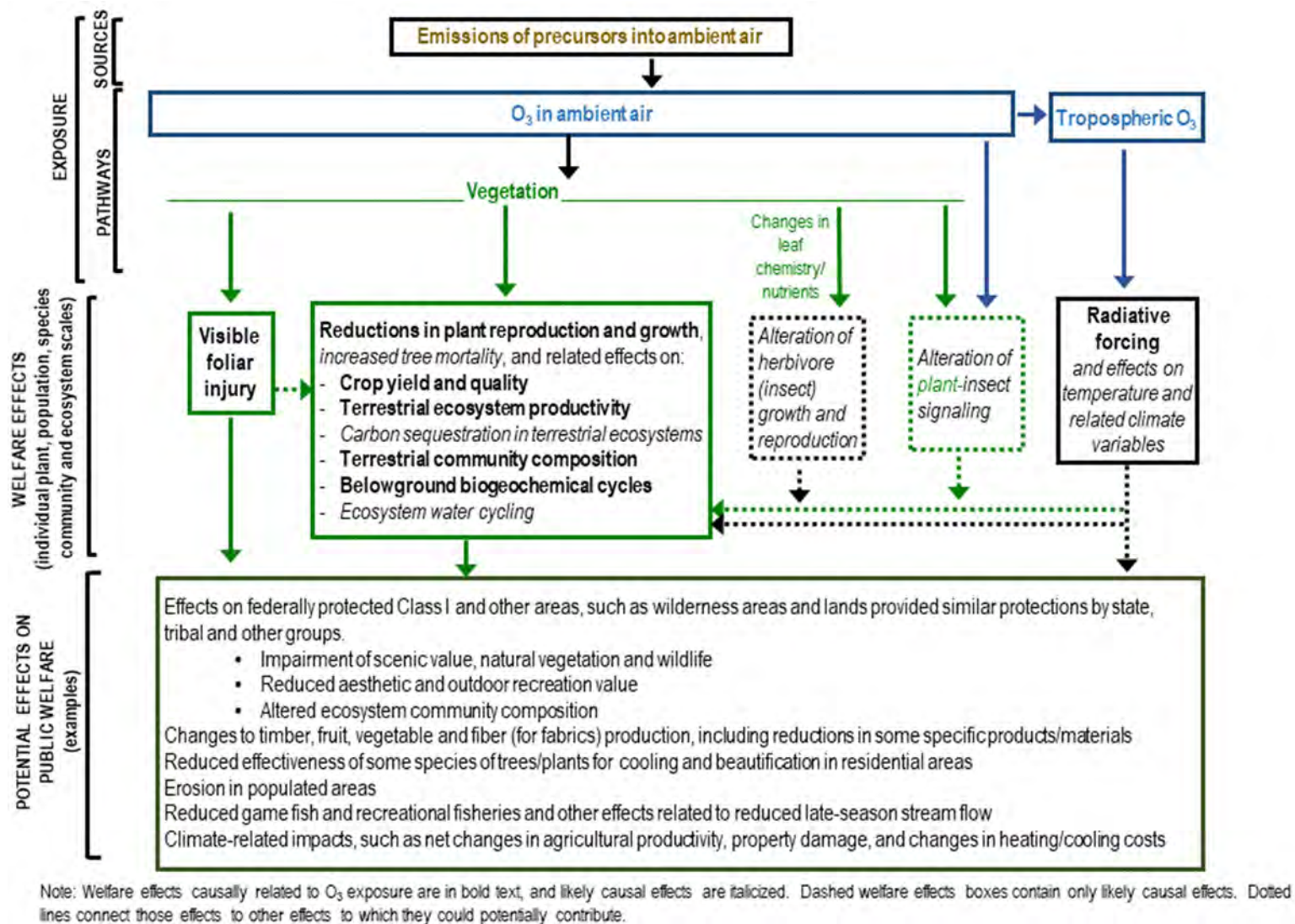


Figure 4-2. Potential effects of O₃ on the public welfare.

1 **4.3.3 Exposures Associated with Effects**

2 The types of effects identified in section 4.3.1 above vary widely with regard to the
3 extent and level of detail of the available information that describes the O₃ exposure
4 circumstances that may elicit them. The information on exposure metric and E-R relationships
5 for effects related to vegetation growth is long-standing, having been first described in the 1997
6 review, while such information is much less established for visible foliar injury. The evidence
7 base for other categories of effects is also lacking in information that might support
8 characterization of potential impacts of changes in O₃ concentrations. The discussion in this
9 section is organized in recognition of this variation. We focus first on growth and yield effects,
10 the category of effects for which the information on exposure metric and E-R relationships is
11 most advanced (section 4.3.3.1). Section 4.3.3.2 discusses the information regarding exposure
12 metrics and relationships between exposure and the occurrence and severity of visible foliar
13 injury. The availability of such information for other categories of effects is addressed in section
14 4.3.3.3.

15 **4.3.3.1 Growth-related Effects**

16 **4.3.3.1.1 Exposure Metrics**

17 The longstanding body of vegetation effects evidence includes a wealth of information on
18 aspects of O₃ exposure that influence effects on plant growth and yield, and that has been
19 described in the scientific assessments across the last several decades (1996 AQCD; 2006
20 AQCD; 2013 ISA; 2020 ISA). A variety of factors have been investigated, including
21 “concentration, time of day, respite time, frequency of peak occurrence, plant phenology,
22 predisposition, etc.” (2013 ISA, section 9.5.2). The importance of the duration of the exposure
23 and the relatively greater importance of higher concentrations over lower concentrations have
24 been consistently well documented (2013 ISA, section 9.5.3). For example, key conclusions of
25 the 1996 AQCD, that have been confirmed in the 2006 AQCD, 2013 ISA and 2020 ISA include
26 that “Ozone effects in plants are cumulative” and “Higher O₃ concentrations appear to be more
27 important than lower concentrations in eliciting a response” (2006 AQCD, p. E-27; 2013 ISA, p.
28 2-44; 2020 ISA, p. 8-180) These AQCDs and ISAs described several mathematical approaches
29 for a single metric or index that would, to some extent, reflect both conclusions.

30 The consideration of these different exposure metrics has primarily focused on their
31 ability to summarize ambient air concentrations of O₃ in a way that best correlates with effects
32 on vegetation, particularly growth-related effects. Metrics based on mean concentrations over
33 several hours (e.g., a seasonal average 12-hour concentration), have generally been considered to
34 be less robust as a metric relating exposure to growth effects (2020 ISA, p. 8-181). The
35 approaches that cumulate exposures over some specified period while weighting higher

1 concentrations more than lower had been evaluated for their predictiveness of growth responses
2 in a set of crop and tree species assessed in experimental O₃ exposure studies for which hourly
3 O₃ concentrations were available for analysis (2013 ISA, sections 9.5.2 and 9.5.3; ISA,
4 Appendix 8, section 8.2.2.2).

5 Along with the non-threshold concentration weighted W126 index, two other cumulative
6 indices that have received greatest attention across the past several O₃ NAAQS reviews have
7 been the threshold weighted indices, AOT60³⁴ and SUM06 (ISA, section IS.3.2).³⁵ Accordingly,
8 some studies of O₃ vegetation effects have reported exposures in terms of these metrics. Based
9 on extensive review of the published literature on different types of such E-R metrics, and
10 comparisons between metrics, and in the context of a single metric, the EPA has generally
11 focused on cumulative, concentration-weighted indices of exposure that reflect some
12 consideration of both concern for cumulative effects of O₃ exposure and for the greater
13 importance of higher concentrations than lower concentrations in vegetation effects (1996
14 AQCD; 2006 AQCD; 2013 ISA).³⁶ Quantifying exposure using such indices has been found to
15 improve the explanatory power of E-R models with regard to O₃ effects in studies of growth and
16 yield over that of indices based only on mean and peak exposure values (2013 ISA, section
17 2.6.6.1, p. 2-44).³⁷

18 The most well-studied datasets in this in this regard are two datasets established two
19 decades ago (referenced above and described further in section 4.3.3.1.2 below), one for growth
20 effects on seedlings of a set of 11 tree species and the second for quality and yield effects for a
21 set of 10 crops (e.g., Lee and Hogsett, 1996, Hogsett et al., 1997). These datasets, which include
22 growth and yield response information across a range of multiple seasonal cumulative exposures,
23 were used to develop quantitative E-R functions for reduced growth (termed relative biomass

³⁴ The AOT60 index is the seasonal sum of the difference between an hourly concentration above 60 ppb, minus 60 ppb (2006 AQCD, p. AX9-161). More recently, some studies have also reported O₃ exposures in terms of AOT40, which is conceptually similar but with 40 substituted for 60 in its derivation (ISA, Appendix 8, section 8.13.1).

³⁵ The SUM06 index is the seasonal sum of hourly concentrations at or above 0.06 ppm during a specified daily time window (2006 AQCD, p. AX9-161; 2013 ISA, section 9.5.2). This may sometimes be referred to as SUM60, e.g., when concentrations are in terms of ppb. There are also variations on this metric that utilize alternative reference points above which hourly concentrations are summed. For example, SUM08 is the seasonal sum of hourly concentrations at or above 0.08 ppm and SUM0 is the seasonal sum of all hourly concentrations.

³⁶ The Agency has focused its analyses in the last several reviews on metrics that characterize cumulative exposures over a season or seasons: SUM06 in the 1997 review (61 FR 65716, December 13, 1996; 62 FR 38856, July 18, 1997) and W126 in both the 2008 and 2015 reviews (72 FR 37818, July 11, 2007; 73 FR 16436, March 27, 2008; 80 FR 65373-65374, October 26, 2015). This approach to characterizing O₃ exposure concentrations with regard to potential vegetation effects, particularly growth, has been supported by CASAC in the past reviews (Henderson, 2006; Samet, 2010; Frey, 2014; Cox, 2020).

³⁷ As described in section 4.3.3.2 below, the W126 index and other similar cumulative exposure indices do not completely describe the relationship of O₃ to visible foliar injury in national surveys.

1 loss or RBL) in seedlings of the tree species and E-R functions for RYL for a set of common
2 crops (ISA, Appendix 8, section 8.13.2; 2013 ISA, section 9.6.2).

3 The EPA’s conclusions regarding cumulative exposure levels of O₃ associated with
4 vegetation-related effects at the time of the 2015 review were based primarily on these
5 established E-R functions and the W126 index, which is a cumulative, seasonal³⁸ concentration-
6 weighted index (80 FR 65404, October 26, 2015; ISA, section IS.3.2, Appendix 8, section 8.13).
7 This metric is a non-threshold approach described as the sigmoidally weighted sum of all hourly
8 O₃ concentrations observed during a specified daily and seasonal time window, where each
9 hourly O₃ concentration is given a weight that increases from zero to one with increasing
10 concentration (2013 ISA, p. 9-101).

11 Alternative methods for characterizing O₃ exposure to predict various plant responses
12 have, in recent years, included flux models (models that are based on the amount of O₃ that
13 enters the leaf). However, as was the case in the 2015 review, there remain a variety of
14 complications, limitations and uncertainties associated with this approach. For example, “[w]hile
15 some efforts have been made in the U.S. to calculate ozone flux into leaves and canopies, little
16 information has been published relating these fluxes to effects on vegetation” (ISA, section
17 IS.3.2). Further, as flux of O₃ into the plant under different conditions of O₃ in ambient air is
18 affected by several factors including temperature, vapor pressure deficit, light, soil moisture, and
19 plant growth stage, use of this approach to quantify the vegetation impact of O₃ would require
20 information on these various types of factors (ISA, section IS.3.2). In addition to these data
21 requirements, each species has different amounts of internal detoxification potential that may
22 protect species to differing degrees. The lack of detailed species- and site-specific data required
23 for flux modeling in the U.S. and the lack of understanding of detoxification processes continues
24 to make this technique less viable for use in risk assessments in the U.S. (ISA, section IS.3.2).

25 Among the studies newly available since the 2015 review, no new exposure indices for
26 assessing effects on vegetation growth or other physiological process parameters have been
27 identified. In the literature available since the 2013 ISA, the SUM06, AOTx (e.g., AOT60) and
28 W126 exposure metrics remain the metrics that are most commonly discussed (ISA, Appendix 8,
29 section 8.13.1). The ISA notes that “[c]umulative indices of exposure that differentially weight
30 hourly concentrations [which would include the W126 index] have been found to be best suited
31 to characterize vegetation exposure to ozone with regard to reductions in vegetation growth and
32 yield” (ISA, section ES.3). Accordingly, in this reconsideration of the 2020 decision, as in the
33 2015 and 2020 reviews, we use the seasonal W126-based cumulative, concentration-weighted

³⁸ In describing the form as “seasonal,” the EPA is referring generally to an index focused on a time period of a duration that may relate to that of a growing season for O₃-sensitive vegetation, not to the seasons of the year (spring, summer, fall, winter).

1 metric in interpreting quantitative exposure analyses, particularly related to growth effects of
2 cumulative O₃ exposures (as summarized in sections 4.3.3.2 and 4.4 below).

3 The first step in calculating the seasonal W126 index for a specific year is to sum the
4 weighted hourly O₃ concentrations in ambient air during daylight hours (defined as 8:00 a.m. to
5 8:00 p.m. local standard time) within each calendar month, resulting in monthly index values.
6 The monthly W126 index values are calculated from hourly O₃ concentrations as follows.³⁹

$$7 \quad \text{Monthly W126} = \sum_{d=1}^N \sum_{h=8}^{19} \frac{C_{dh}}{1+4403 \cdot \exp(-126 \cdot C_{dh})}$$

8 where,

9 *N* is the number of days in the month

10 *d* is the day of the month (*d* = 1, 2, ..., *N*)

11 *h* is the hour of the day (*h* = 0, 1, ..., 23)

12 *C_{dh}* is the hourly O₃ concentration observed on day *d*, hour *h*, in parts per million

13 The W126 index value for a specific year is the maximum sum of the monthly index values for
14 three consecutive months within a calendar year (i.e., January to March, February to April, ...
15 October to December). Three-year average W126 index values are calculated by taking the
16 average of seasonal W126 index values for three consecutive years (e.g., as described in
17 Appendix 4D, section 4D.2.2).

18 **4.3.3.1.2 Relationships Between Cumulative Concentration-weighted Exposure** 19 **Levels and Effects**

20 Across the array of O₃-related welfare effects, consistent and systematically evaluated
21 information on E-R relationships across multiple exposure levels is limited. Most prominent is
22 the information on E-R relationships for growth effects on tree seedlings and crops,⁴⁰ which has
23 been available for the past several reviews. The information on which these functions are based
24 comes primarily from the U.S. EPA's National Crop Loss Assessment Network (NCLAN)⁴¹
25 project for crops and the NHEERL-WED project for tree seedlings, projects implemented
26 primarily to define E-R relationships for major agricultural crops and tree species, thus
27 advancing understanding of responses to O₃ exposures (ISA, Appendix 8, section 8.13.2). These
28 projects and related studies included a series of experiments that used OTCs to investigate tree
29 seedling growth response and crop yield over a growing season under a variety of O₃ exposures

³⁹ In situations where data are missing, an adjustment is factored into the monthly index (as described in Appendix 4D, section 4D.2.2).

⁴⁰ The E-R functions estimate O₃-related reduction in a year's tree seedling growth or crop yield as a percentage of that expected in the absence of O₃ (Appendix 4A; ISA, Appendix 8, section 8.13.2).

⁴¹ The NCLAN program, which was undertaken in the early to mid-1980s, assessed multiple U.S. crops, locations, and O₃ exposure levels, using consistent methods, to provide the largest, most uniform database on the effects of O₃ on agricultural crop yields (1996 AQCD, 2006 AQCD, 2013 ISA, sections 9.2, 9.4, and 9.6; ISA, Appendix 8, section 8.13.2).

1 and growing conditions (2013 ISA, section 9.6.2; Lee and Hogsett, 1996). These experiments
2 assessed O₃ effects on tree seedling growth and crop yield for a variety of O₃ treatments and
3 growing conditions. The higher exposure levels in these datasets generally included numerous
4 hours at or above 100 ppb (Lefohn et al., 1997; Appendix 4A, Table 4A-6). Importantly, the
5 information on exposure includes hourly concentrations across the season (or longer) exposure
6 period which allowed for derivation of various seasonal metrics that were analyzed for
7 association with reduced growth. In the initial analyses of these data, exposure was characterized
8 in terms of several metrics, including seasonal SUM06 and W126 indices (Lee and Hogsett,
9 1996; 1997 Staff Paper, sections IV.D.2 and IV.D.3; 2007 Staff Paper, section 7.6), while use of
10 these functions in the 2015 review focused on their implementation in terms of seasonal W126
11 index (2013 ISA, section 9.6; 80 FR 65391-92, October 26, 2015).⁴²

12 The 11 species for which robust and well-established E-R functions for RBL were
13 derived black cherry, Douglas fir, loblolly pine, ponderosa pine, quaking aspen, red alder, red
14 maple, sugar maple, tulip poplar, Virginia pine, and white pine (Figure 4-3; Appendix 4A; 2020
15 ISA, Appendix 8, section 8.13.2; 2013 ISA, section 9.6).⁴³ While these 11 species represent only
16 a small fraction of the total number of native tree species in the contiguous U.S., this small
17 subset includes eastern and western species, deciduous and coniferous species, and species that
18 grow in a variety of ecosystems and represent a range of tolerance to O₃ (Appendix 4B; 2020
19 ISA, Appendix 8, section 8.13.2; 2013 ISA, section 9.6.2). The established E-R functions for
20 most of the 11 species were derived using data from multiple studies or experiments, many of
21 which employed open top chambers, an established experimental approach, involving a wide
22 range of exposure and/or growing conditions. For example, many of the experimental treatments
23 for exposures to elevated O₃ on which the established E-R functions for the 11 tree seedling
24 species are based, involved W126 index levels well above 20 ppm-hrs and had many (tens to

⁴² This underlying database for the exposure is a key characteristic that sets this set of studies (and their associated E-R analyses) apart from other available studies.

⁴³ A quantitative analysis of E-R information for an additional species was considered in the 2014 WREA. But the underlying study, rather than being an OTC controlled exposure study, involves exposure to ambient air along an existing gradient of O₃ concentrations in the New York City metropolitan area, such that O₃ and climate conditions were not controlled (2013 ISA, section 9.6.3.3). Based on comments from the CASAC on the WREA cautioning against placing too much emphasis on these data (e.g., saying that the eastern cottonwood response data from a single study “receive too much emphasis,” explaining that these “results are from a gradient study that did not control for ozone and climatic conditions and show extreme sensitivity to ozone compared to other studies” and that “[a]lthough they are important results, they are not as strong as those from other experiments that developed E-R functions based on controlled ozone exposure”) (Frey, 2014, p. 10), the EPA did not include the E-R function for eastern cottonwood among the set of tree seedling E-R functions given focus in the WREA, or relied on in decision-making for the 2015 review (80 FR 65292, October 26, 2015.)

1 more than a hundred) of hours of O₃ concentrations above 100 ppb (Appendix 4A, Table 4A-6;
2 Lefohn et al 1997).^{44 45}

3 From the available data, separate E-R functions were developed for each combination of
4 species and experiment⁴⁶ (2013 ISA, section 9.6.1; Lee and Hogsett, 1996). For the 11 species,
5 there are 51 separate “experiment-specific” E-R functions (Appendix 4A, section 4A.1.1; ISA,
6 section 8.1.2.1.2). For six of the 11 species, the species-specific function is based on just one or
7 two experimental datasets (e.g., red maple), while for other species there were as many as 11
8 datasets supporting 11 experiment-specific E-R functions (e.g., ponderosa pine). The exposure
9 durations varied from periods of 82 to 140 days in a single year to periods of 180 to 555 days
10 occurring across two years (Lee and Hogsett, 1996; Appendix 4A, Table 4A-5). The
11 experimental datasets for more than half the 11 species include exposures occurring across two
12 years. To account for potential for a delayed response, some datasets are for growth
13 measurements taken in the spring of the year after a prior year growing season exposure and
14 others are for growth measurements taken immediately after the exposure. From the separate
15 species-experiment-specific E-R functions, species-specific composite E-R functions were
16 developed (Appendix 4A). In order to be utilized in deriving a single species-specific function
17 and to produce species-specific E-R functions of consistent duration, the separate species-
18 experiment-specific E-R functions were derived first based on the exposure duration of the
19 experiment and then normalized to 3-month (seasonal) periods⁴⁷ (see Lee and Hogsett, 1996,
20 section I.3; Appendix 4A).

21 The 11 species-specific composite median functions are presented in Appendix 4A (see
22 section 4A.1.1). Biomass growth loss predictions using the function for aspen was evaluated in
23 the 2013 and 2020 ISAs based on a recent study for aspen (2013 ISA, section 9.6.2; ISA,

⁴⁴ Among the experiments on which the E-R functions are based, N100 values for exposure levels most common at U.S. sites that meet the current standard (e.g., W126 index less than 25 ppm-hrs for a single season), extend up above 10, to more than 40. Additionally, in a study that has reported the distributions of hourly concentrations, the 90th percentile in replicates for one of the elevated O₃ treatments ranged from 142 to 156 ppb, and the maximum ranged from 210 to 260 ppb (Appendix 4A, Table 4A-6; Lefohn et al 1992).

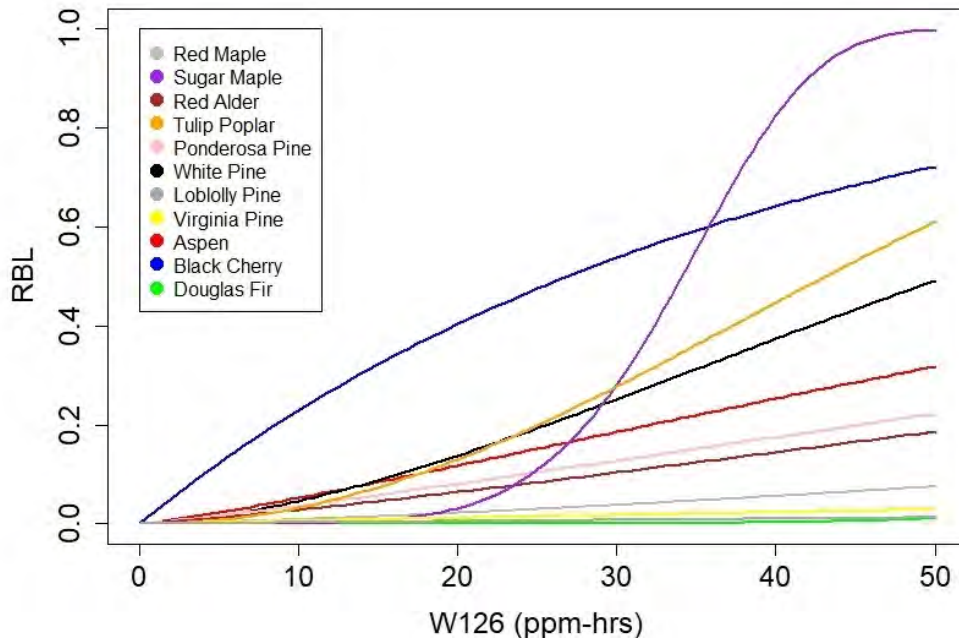
⁴⁵ Similarly, the experimental exposures in studies supporting some of the established E-R functions for 10 crop species also include many hours with hourly O₃ concentrations at or above 100 ppb (Lefohn and Foley, 1992).

⁴⁶ Use of the term, experiment, refers to each separate seedling response dataset (from each separate harvest), including, for example, a 2nd harvest in the spring that received the same growing season exposure as the response documented for seedlings in the 1st harvest immediately following the growing season. As an initial step in deriving species-specific E-R functions each of those response datasets were used to derive separate E-R functions (Appendix 4A, Attachment 1).

⁴⁷ Underlying the adjustment is a simplifying assumption of uniform W126 distribution across the exposure periods and of a linear relationship between duration of cumulative exposure in terms of the W126 index and plant growth response. Some functions for experiments that extended over two seasons were derived by distributing responses observed at the end of two seasons of varying exposures equally across the two seasons (e.g., essentially applying the average to both seasons).

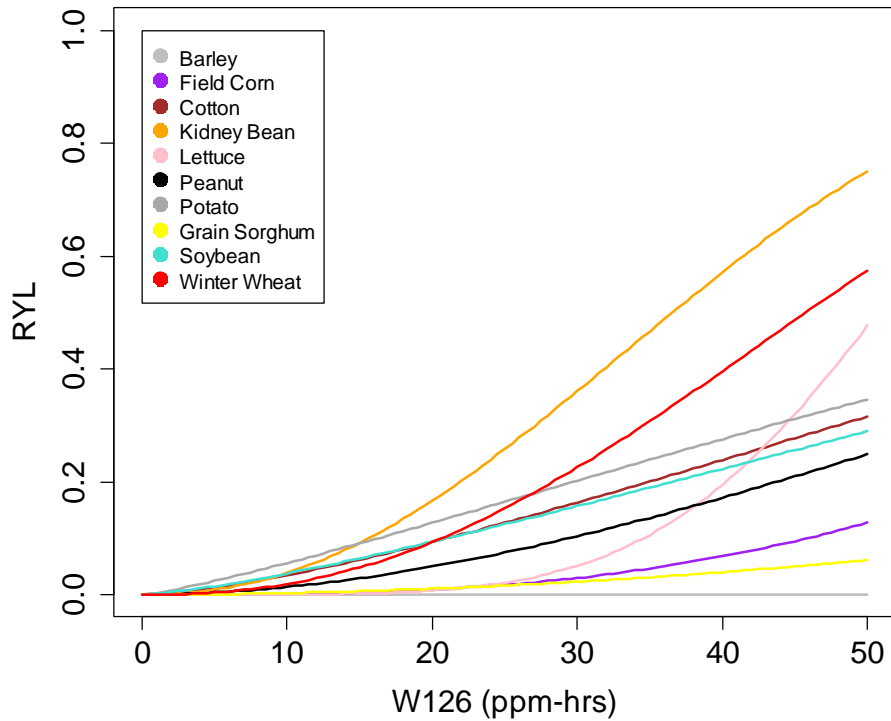
1 Appendix 8, section 8.13.2). The species-specific composite E-R functions developed from the
 2 experiment-specific functions indicate a wide variation in growth sensitivity of the studied tree
 3 species at the seedling stage (Appendix 4A, section 4A.1.1). A stochastic analysis performed for
 4 the 2014 WREA provides a sense of the variability and uncertainty associated with the estimated
 5 E-R relationships among and within species⁴⁸ (Appendix 4A, section 4A.1.1, Figure 4A-13).
 6 Further, based on the species-specific E-R functions, the studied tree species appear to vary
 7 widely in sensitivity to reduced growth at the seedling stage (Figure 4-3).

8 With regard to crops, established E-R functions are available for 10 crops: barley, field
 9 corn, cotton, kidney bean, lettuce, peanut, potato, grain sorghum, soybean, and winter wheat
 10 (Figure 4-4; Appendix 4A; ISA, Appendix 8, section 8.13.2). Since the 2015 review, new
 11 evidence is available for seven soybean cultivars that confirms the reliability of the soybean E-R
 12 functions developed from NCLAN data and indicates that they extend in applicability to recent
 13 cultivars (ISA, Appendix 8, section 8.13.2).



14
 15 **Figure 4-3. Established RBL functions for seedlings of 11 tree species.**

⁴⁸ The multiple functions derived for each species are derived from separate datasets, some of which may have the same exposure during the growing season but which reflect response derived from seedlings harvested in the spring subsequent to the growing season exposure (Lee and Hogsett, 1996). Accordingly, this analysis provides a sense of both uncertainty in experimental design and environmental and seedling response variability.



1
2 **Figure 4-4. Established RYL functions for 10 crops.**

3 Since the initial set of tree seedling studies were completed, several additional studies,
4 focused on aspen, have been published based on the Aspen FACE experiment in a planted forest
5 in Wisconsin; the findings were consistent with many of the OTC studies (ISA, Appendix 8,
6 section 8.13.2). Newly available studies that investigated growth effects of O₃ exposures are also
7 consistent with the existing evidence base, and generally involved particular aspects of the effect
8 rather than expanding the conditions under which plant species, particularly trees, have been
9 assessed (ISA, section IS.5.1.2). These publications include a compilation of previously available
10 studies on plant biomass response to O₃ (in terms of AOT40); the compilation reports linear
11 regressions conducted on the associated varying datasets (ISA, Appendix 8, section 8.13.2).
12 Based on these regressions, this study describes distributions of sensitivity to O₃ effects on
13 biomass across many tree and grassland species, including 17 species native to the U.S. and 65
14 introduced species (ISA, Appendix 8, section 8.13.2; van Goethem et al., 2013). Additional
15 information is needed to describe O₃ E-R relationships more completely for these species in the
16 U.S.⁴⁹ As was noted in the 2013 ISA, “[i]n order to support quantitative modeling of exposure-

⁴⁹ The studies compiled in this publication included at least 21 days exposure above 40 ppb O₃ (expressed as AOT40 [seasonal sum of the difference between an hourly concentration above 40 ppb and 40 ppb]) and had a maximum hourly concentration that was no higher than 100 ppb (van Goethem et al., 2013). The publication does not report study-specific exposure durations, details of biomass response measurements or hourly O₃ concentrations, making it less useful for describing E-R relationships that might support estimation of specific impacts associated with air quality conditions meeting the current standard (e.g., 2013 ISA, p. 9-118).

1 response relationships, data should preferably include more than three levels of exposure, and
2 some control of potential confounding or interacting factors should be present in order to model
3 the relationship with sufficient accuracy” (2013 ISA, p. 9-118). The 2013 ISA further discussed
4 the differences across available studies, recognizing that the majority of studies contrast only two
5 (or sometimes three with the addition of a carbon filtration) O₃ exposure levels. While such
6 studies can be important for verifying more extensive studies, they “do not provide exposure-
7 response information that is highly relevant to reviewing air quality standards” (2013 ISA, p. 9-
8 118).

9 **4.3.3.2 Visible Foliar Injury**

10 The evidence “continues to show a consistent association between visible injury and
11 ozone exposure,” while also recognizing the role of modifying factors such as soil moisture and
12 time of day (ISA, section IS.5.1.1). The ISA, in concluding that the newly available information
13 is consistent with conclusions of the 2013 ISA, also summarizes several recently available
14 studies that continue to document that O₃ elicits visible foliar injury in many plant species,
15 including a synthesis of previously published studies that categorizes studied species (and their
16 associated taxonomic classifications) as to whether or not O₃-related foliar injury has been
17 reported. Although this recent publication identifies many species in which visible foliar injury
18 has been documented to occur in the presence of elevated O₃,⁵⁰ it does not provide quantitative
19 information regarding specific exposure conditions or analyses of E-R relationships (ISA,
20 Appendix 8, section 8.3). Additionally, one recent study is identified as reporting visible foliar
21 injury in a non-native, yet established, and invasive tree species in a location with O₃
22 concentrations corresponding to a seasonal W126 index of 11.6 ppm-hrs (ISA, Appendix 8,
23 sections 8.2 and 8.2.1). The annual fourth highest 8-hour daily maximum concentration for the
24 study year and location of this study (monitoring site 42-027-9991) is 76 ppb. The design value
25 for the 3-year design period encompassing the year and location of this study exceeds 70 ppb
26 (monitoring site 42-027-9991 for 2011-2013 design period), indicating that the air quality
27 associated with the exposure would not have met the current secondary standard.⁵¹

28 As in the past, the available evidence, while documenting that elevated O₃ conditions in
29 ambient air generally result in visible foliar injury in sensitive species (when in a predisposing

⁵⁰ The publication identifies 245 species across 28 plant genera, many native to the U.S., in which O₃-related visible foliar injury has been reported (ISA, Appendix 8, section 8.3).

⁵¹ Ozone design values for this period are available at: <https://www.epa.gov/air-trends/air-quality-design-values>. The year 2011 is the first year for which data are available and adequate for use in deriving a design value at this monitoring site.

1 environment)⁵², it does not include a quantitative description of the relationship of incidence or
2 severity of visible foliar injury in sensitive species in natural areas of the U.S. with specific
3 metrics of O₃ exposure. Several studies of the extensive USFS field-based dataset of visible
4 foliar injury incidence in forests across the U.S.⁵³ illustrate the limitations of current
5 understanding of this relationship. For example, a study that was available in the 2015 review
6 presents a trend analysis of these data for sites located in 24 states of the northeast and north
7 central U.S. for the 16-year period from 1994 through 2009 that provides some insight into the
8 influence of changes in air quality and soil moisture on visible foliar injury and the difficulty
9 inherent in predicting foliar injury response under different air quality and soil moisture
10 scenarios (Smith, 2012, Smith et al., 2012; ISA, Appendix 8, section 8.2). This study, like prior
11 analyses of such data, shows the dependence of foliar injury incidence and severity on local site
12 conditions for soil moisture availability and O₃ exposure. For example, while the authors
13 characterize the ambient air O₃ concentrations to be the “driving force” behind incidence of
14 injury and its severity, they state that “site moisture conditions are also a very strong influence
15 on the biomonitoring data” (Smith et al., 2003). In general, the USFS data analyses have found
16 foliar injury prevalence and severity to be higher during seasons and sites that have experienced
17 the highest O₃ than during other periods (e.g., Campbell et al., 2007; Smith, 2012).

18 **4.3.3.2.1 Exposure Metrics**

19 Although studies of the incidence of visible foliar injury in national forests, wildlife
20 refuges, and similar areas have often used cumulative indices (e.g., SUM06) to investigate
21 variations in incidence of foliar injury, studies also suggest an additional role for metrics focused
22 on peak concentrations (ISA; 2013 ISA; 2006 AQCD; Hildebrand et al., 1996; Smith, 2012).
23 Other studies have indicated this uncertainty regarding a most influential metric(s), by
24 recognizing a research need. For example, a study of six years of USFS biosite data for three
25 western states found that the biosites with the highest cumulative O₃ exposure (SUM06 at or
26 above 25 ppm-hrs) had the highest percentage of biosites with injury and the highest mean
27 biosite index, with little discernable difference among the lower exposure categories; this study
28 also identified “better linkage between air levels and visible injury” as an O₃ research need

⁵² As noted in the 2013 and 2020 ISAs, visible foliar injury usually occurs when sensitive plants are exposed to elevated ozone concentrations in a predisposing environment, with a major modifying factor being the amount of soil moisture available to a plant. Accordingly, dry periods are concluded to decrease the incidence and severity of ozone-induced visible foliar injury, such that the incidence of visible foliar injury is not always higher in years and areas with higher ozone, especially with co-occurring drought (ISA, Appendix 8, p. 8-23; Smith, 2012; Smith et al., 2003).

⁵³ These data were collected as part of the U.S. Forest Service Forest Health Monitoring/Forest Inventory and Analysis (USFS FHM/FIA) biomonitoring network program (2013 ISA, section 9.4.2.1; Campbell et al., 2007, Smith et al., 2012).

1 (Campbell et al., 2007). More recent studies of the complete 16 years of data in 24 northeast and
2 north central states have suggested that a cumulative exposure index alone may not completely
3 describe the O₃-related risk of this effect (Smith et al., 2012; Smith, 2012). For example, Smith
4 (2012) observed there to be a declining trend in the 16-year dataset, “especially after 2002 when
5 peak ozone concentrations declined across the entire region” thus suggesting a role for peak
6 concentrations.

7 Some studies of visible foliar injury incidence data have investigated the role of peak
8 concentrations quantified by an O₃ exposure index that is a count of hourly concentrations (e.g.,
9 in a year or growing season) above a threshold 1-hour concentration of 100 ppb, N100 (e.g.,
10 Smith, 2012; Smith et al., 2012). For example, analyses of injury patterns over 16 years at USFS
11 biosites in 24 states in the Northeast and North Central regions, in the context of the SUM06
12 index and N100 metrics (although not in statistical combination), suggested that there may be a
13 threshold exposure needed for injury to occur,⁵⁴ and that the number of hours of elevated O₃
14 concentrations during the growing season (such as what is captured by a metric like N100) may
15 be more important than cumulative exposure in determining the occurrence of foliar injury
16 (Smith, 2012).⁵⁵ This finding is consistent with statistical analyses of seven years of visible foliar
17 injury data from a wildlife refuge in the mid-Atlantic (Davis and Orendovici, 2006). The latter
18 study investigated the fit of multiple models that included various metrics of cumulative O₃ (e.g.,
19 SUM06, SUM0, SUM08), alone and in combination with some other variables (Davis and
20 Orendovici, 2006). Among the statistical models investigated, the model with the best fit to the
21 visible foliar injury incidence data was found to be one that included N100 and W126 indices, as
22 well as drought index (Davis and Orendovici, 2006).⁵⁶

23 The established significant role of higher or peak O₃ concentrations, as well as pattern of
24 their occurrence, in plant responses has also been noted in prior ISAs or AQCDs. The evidence
25 has included studies that use indices to summarize the incidence of injury on bioindicator species
26 present at specific monitored sites, as well as experimental studies that assess the occurrence of
27 foliar injury in response to varying O₃ concentrations. In identifying support with regard to foliar

⁵⁴ Authors of the study observed that “injury is minimized when seasonal ozone concentrations, especially peak (N100) O₃ concentrations, drop below a certain threshold as in 2004 through 2009” (Smith et al., 2012).

⁵⁵ Although the ISA and past assessments have not described extensive evaluations of specific peak concentration metrics such as the N100 (that might assist in identifying one best suited for such purposes), in summarizing this study in the last review, the ISA observed that “[o]verall, there was a declining trend in the incidence of foliar injury as peak O₃ concentrations declined” (2013 ISA, p. 9-40).

⁵⁶ The models evaluated included several with cumulative exposure indices alone. These included SUM60 (i.e., SUM06 in ppb), SUM0, and SUM80 (SUM08 in ppb), but not W126. They did include a model with W126 that did not also include N100. Across all of these models evaluated, the model with the best fit to the data was found to be the one that included N100 and W126, along with the drought index (Davis and Orendovici, 2006).

1 injury as the response, the 2013 ISA and 2006 AQCD both cite studies that support the
2 “important role that peak concentrations, as well as the pattern of occurrence, plays in plant
3 response to O₃” (2013 ISA, p. 9-105; 2006 AQCD, p. AX9-169). For example, a study of
4 European white birch saplings reported that peak concentrations and the duration of the exposure
5 event were important determinants of foliar injury (2013 ISA, section 9.5.3.1; Oksanen and
6 Holopainen, 2001). This study also evaluated tree growth, which was found to be more related to
7 cumulative exposure (2013 ISA, p. 9-105).⁵⁷ A second study that was cited by both assessments
8 that focused on aspen, reported that “the variable peak exposures were important in causing
9 injury, and that the different exposure treatments, although having the same SUM06, resulted in
10 very different patterns of foliar injury (2013 ISA, p. 9-105; 2006 AQCD, p. AX9-169; Yun and
11 Laurence, 1999). As noted in the 2006 AQCD, the cumulative exposure indices (e.g., SUM06,
12 W126) were “originally developed and tested using only growth/yield data, not foliar injury” and
13 “[t]his distinction is critical in comparing the efficacy of one index to another” (2006 AQCD, p.
14 AX9-173). It is also recognized that where cumulative indices are highly correlated with the
15 frequency or occurrence of higher hourly average concentrations, they could be good predictors
16 of such effects (2006 AQCD, section AX9.4.4.3).

17 Dose modeling or flux models, discussed in section 4.3.3.1.1 above, have also been
18 considered for quantifying O₃ dose that may be related to plant injury. Among the newly
19 available evidence is a study examining relationships between short-term flux and leaf injury on
20 cotton plants that described a sensitivity parameter that might characterize the influence on the
21 flux-injury relationship of diel and seasonal variability in plant defenses (among other factors)
22 and suggested additional research might provide for such a sensitivity parameter to “function
23 well in combination with a sigmoidal weighting of flux, analogous to the W126 weighting of
24 concentration”, and perhaps an additional parameter (Grantz et al., 2013, p. 1710; ISA, Appendix
25 8, section 8.13.1). However, the ISA recognizes there is “much unknown” with regard to the
26 relationship between O₃ uptake and leaf injury, and relationships with detoxification processes
27 (ISA, Appendix 8, section 8.13.1 and p. 8-184). These uncertainties have made this technique
28 less viable for assessments in the U.S., precluding use of a flux-based approach at this time (ISA,
29 Appendix 8, section 8.13.1 and p. 8-184).

30 A study (by Wang et al. [2012], newly described in the 2020 ISA) involved a statistical
31 modeling analysis on a subset of the years of data that were described in Smith (2012). This
32 analysis, which involved 5,940 data records from 1997 through 2007 from the 24 northeast and
33 north central states, tested a number of models for their ability to predict the presence of visible

⁵⁷ The study authors concluded that “high peak concentrations were important for visible injuries and stomatal conductance, but less important for determining growth responses” (Oksanen and Holopainen, 2001).

1 foliar injury (a nonzero biosite score), regardless of severity, and generally found that the type of
2 O₃ exposure metric (e.g., SUM06 *versus* N100) made only a small difference, although the
3 models that included both a cumulative index (SUM06) and N100 had a just slightly better fit
4 (Wang et al., 2012). Based on their investigation of 15 different models, using differing
5 combinations of several types of potential predictors, the study authors concluded that they were
6 not able to identify environmental conditions under which they “could reliably expect plants to
7 be damaged” (Wang et al., 2012). This is indicative of the current state of knowledge, in which
8 there remains a lack of established quantitative functions describing E-R relationships that would
9 allow prediction of visible foliar injury severity and incidence under varying air quality and
10 environmental conditions.

11 **4.3.3.2.2 Exposure Levels Associated with Effects**

12 The available information related to O₃ exposures associated with visible foliar injury of
13 varying severity also includes the dataset developed by the EPA in the 2015 review from USFS
14 BI scores, collected during the years 2006 through 2010 at locations in 37 states (Appendix 4C).
15 In developing this dataset, the BI scores were combined with estimates of soil moisture⁵⁸ and
16 estimates of seasonal cumulative O₃ exposure in terms of W126 index⁵⁹ (Smith and Murphy,
17 2015; Appendix 4C). This dataset includes more than 5,000 records of which more than 80
18 percent have a BI score of zero (indicating a lack of visible foliar injury).⁶⁰ While the estimated
19 W126 index assigned to records in this dataset (described in Appendix 4C) ranges from zero to
20 somewhat above 50 ppm-hrs, more than a third of all the records (and also of records with BI
21 scores above zero or five)⁶¹ are at sites with W126 index estimates below 7 ppm-hrs and only 8%
22 of the records have W126 index values above 15 ppm-hrs. In an extension of analyses developed

⁵⁸ Soil moisture categories (dry, wet or normal) were assigned to each biosite record based on the NOAA Palmer Z drought index values obtained from the NCDC website for the April-through-August periods, averaged for the relevant year; details are provided in Appendix 4C, section 4C.2. There are inherent uncertainties in this assignment, including the substantial spatial variation in soil moisture and large size of NOAA climate divisions (hundreds of miles). Uncertainties and limitations in the dataset are summarized in Appendix 4C, section 4C.5).

⁵⁹ The W126 index values assigned to the biosite locations are estimates developed for 12 kilometer (km) by 12 km cells in a national-scale spatial grid for each year. The grid cell estimates were derived from applying a spatial interpolation technique to annual W126 values derived from O₃ measurements at ambient air monitoring locations for the years corresponding to the biosite surveys (details in Appendix 4C, sections 4.C.2 and 4C.5).

⁶⁰ In the scheme used by the USFS to categorize severity of biosite scores the lowest category encompasses BI scores from zero to just below 5; scores of this magnitude are described as “little or no foliar injury” (Smith et al., 2012). The next highest category encompasses scores from five to just below 15 and is described as “light to moderate foliar injury,” BI scores of 15 up to 25 are described as “moderate” and above 25 is described as “severe” (Smith, 2012; Smith et al., 2012)..

⁶¹ One third (33%) of scores above 15 are at sites with W126 below 7 ppm-hrs (Appendix 4C, Table 4C-3).

1 in the 2015 review, the presentation in the Appendix 4C⁶² describes the BI scores for the records
2 in the dataset in relation to the W126 index estimate for each record, using “bins” of increasing
3 W126 index values. The presentation utilizes the BI score breakpoints in the scheme used by the
4 USFS to categorize severity. This presentation indicates that, across the W126 bins, there is
5 variation in both the incidence of particular magnitude BI scores and in the average score per
6 bin. In general, however, the greatest incidence of records with BI scores above zero, five, or
7 higher – and the highest average BI score (as noted below) – occurs with the highest W126 bin
8 (i.e., the bin for W126 index estimates greater than 25 ppm-hrs), as seen in Figure 4-5 for records
9 in the normal soil moisture category⁶³ (see also Appendix 4C, Table 4C-6).

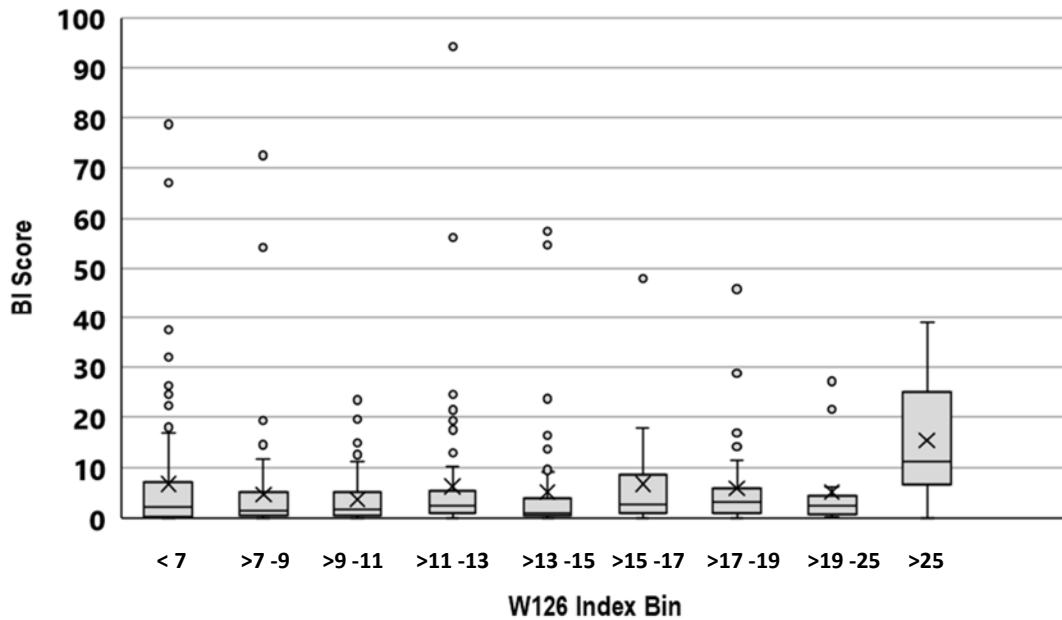
10 The average BI score per W126 index bin is also variable, although for records
11 categorized as normal soil moisture, the average BI score in the highest W126 bin is noticeably
12 greater than for lower W126 bin scores (Figure 4-5). For example, the average BI score for the
13 normal soil moisture category is 7.9 among records with W126 index estimates greater than 25
14 ppm-hrs, compared to 1.6 among records for W126 index estimates between 19 and 25 ppm-hrs.
15 For records categorized as wet soil moisture, the sample size for the W126 bins above 13 ppm-
16 hrs is quite small (including only 18 of the 1,189 records in that soil moisture category),
17 precluding meaningful interpretation.⁶⁴

18 While for BI scores above zero, the data may indicate a suggestion of increased incidence
19 among records in the W126 bins just below the highest (e.g., for the dry or normal soil moisture
20 categories), for BI scores above 5, there is little or no difference across the W126 bins except for
21 the highest bin, which is for W126 above 25 ppm-hrs (Appendix 4C, Table 4C-6). For example,
22 among records in the normal soil category, the proportion of records with BI above five
23 fluctuates between 5% and 13% across all but the highest W126 bin (>25 ppm-hrs) for which the
24 proportion is 41% (Appendix 4C, Table 4C-6). The same pattern is observed for BI scores above
25 15 at sites with normal and dry soil moisture conditions, albeit with lower incidences. For
26 example, the incidence of normal soil moisture records with BI score above 15 in the bin for
27 W126 index values above 25 ppm-hrs was 20% but fluctuates between 1% and 4% in the bin
28 with W126 index values at or below 25 ppm-hrs (Appendix 4C, Table 4C-6).

⁶² Beyond the presentation of a statistical analysis developed in the last review (Appendix 4C, section 4C.4.1), the PA presentations are primarily descriptive (as compared to statistical) in recognition of the limitations and uncertainties of the dataset (Appendix 4C, section 4C.5).

⁶³ The number of records per W126 bin in Figure 4-5 ranges from a low of 15 in the “>19-25” bin to 158 in the “<7” bin (Appendix 4C, Table 4C-4).

⁶⁴ In the full database for the wet soil moisture category, there are only 18 records at sites with a W126 index value above 13 ppm-hrs, with 9 or fewer (less than 1%) in each of them (Appendix 4C, Table 4C-3). Across the W126 bins in which at least 1% of the wet soil moisture records are represented, differences of incidence or average score of lower bins from the highest bin is less than a factor of two (Appendix 4C, section 4C.4.2).



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Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the arithmetic mean, and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

Figure 4-5. Distribution of nonzero BI scores at USFS biosites (normal soil moisture) grouped by assigned W126 index estimates.

Overall, the dataset described in Appendix 4C generally indicates the risk of injury, and particularly injury considered at least light, moderate or greater injury, to be higher at the highest W126 index values, with appreciable variability in the data for the lower bins. This appears to be consistent with the conclusions of the detailed quantitative analysis studies, summarized above, that the pattern is stronger at higher O₃ concentrations. A number of factors may contribute to the observed variability in BI scores and lack of a clear pattern with W126 index bin; among others, these may include uncertainties in assignment of W126 estimates and soil moisture categories to biosite locations, variability in biological response among the sensitive species monitored, and the potential role of other aspects of O₃ air quality not captured by the W126 index. Thus, the dataset has limitations affecting associated conclusions, and uncertainty remains regarding the tools for and the appropriate metric (or metrics) for quantifying influence of O₃ exposures, as well as perhaps for quantifying soil moisture conditions, with regard to their influence on extent and/or severity of injury in sensitive species in natural areas, as quantified via BI scores (Davis and Orendovici, 2006; Smith et al., 2012; Wang et al., 2012). Accordingly, the limitations recognized in the past remain in our ability to quantitatively estimate incidence and severity of visible foliar injury likely to occur in areas across the U.S. under different air quality conditions over a year, or over a multi-year period (Appendix 4C, section 4C.5).

1 4.3.3.3 Other Effects

2 With regard to radiative forcing and subsequent climate effects associated with the global
3 tropospheric abundance of O₃, the available evidence does not provide more detailed quantitative
4 information regarding O₃ concentrations at the national scale than was available in the 2015
5 review (ISA, Appendix 9). Rather, it is noted that “the heterogeneous distribution of ozone in the
6 troposphere complicates the direct attribution of spatial patterns of temperature change to ozone
7 induced [radiative forcing]” and there are “ozone climate feedbacks that further alter the
8 relationship between ozone [radiative forcing] and temperature (and other climate variables) in
9 complex ways” (ISA, Appendix 9, section 9.3.1, p. 9-19). Further, “precisely quantifying the
10 change in surface temperature (and other climate variables) due to tropospheric ozone changes
11 requires complex climate simulations that include all relevant feedbacks and interactions” (ISA,
12 section 9.3.3, p. 9-22). Yet, there are limitations in current climate modeling capabilities for O₃;
13 an important one is representation of important urban- or regional-scale physical and chemical
14 processes, such as O₃ enhancement in high-temperature urban situations or O₃ chemistry in city
15 centers where NO_x is abundant. Such limitations impede our ability to quantify the impact of
16 incremental changes in ground-level O₃ concentrations in the U.S. on radiative forcing and
17 subsequent climate effects.

18 With regard to tree mortality, the evidence available in the last several reviews included
19 field studies of pollution gradients that concluded O₃ damage to be an important contributor to
20 tree mortality although “several confounding factors such as drought, insect outbreak and forest
21 management” were identified as potential contributors (2013 ISA, p. 9-81, section 9.4.7.1).
22 Among the newly available studies, there is only limited experimental evidence that isolates the
23 effect of O₃ on tree mortality⁶⁵ and might be informative regarding O₃ concentrations of interest
24 in the review, and evidence is lacking regarding exposure conditions closer to those occurring
25 under the current standard and any contribution to tree mortality.

26 With regard to alteration of herbivore growth and reproduction, although “there are
27 multiple studies demonstrating ozone effects on fecundity and growth in insects that feed on
28 ozone-exposed vegetation”, “no consistent directionality of response is observed across studies
29 and uncertainties remain in regard to different plant consumption methods across species and the
30 exposure conditions associated with particular severities of effects” (ISA, pp. ES-18, IS-64, IS91
31 and Appendix 8, section 8.6.3). Such limitations and uncertainties in the evidence base for this
32 category of effects preclude broader characterization, as well as quantitative analysis related to

⁶⁵ Of the three new studies on tree mortality described in the ISA is another field study of a pollution gradient that, like such studies in prior reviews, recognizes O₃ exposures as one of several contributing environmental and anthropogenic stressors (ISA, p. 8-55).

1 air quality conditions meeting the O₃ standard. As characterized in the ISA, uncertainties remain
2 in the evidence; these relate to the different plant consumption methods across species and the
3 exposure conditions associated with particular responses, as well as variation in study designs
4 and endpoints used to assess O₃ response (ISA, IS.6.2.1 and Appendix 8, section 8.6). Thus,
5 while the evidence describes changes in nutrient content and leaf chemistry following O₃
6 exposure (ISA, p. IS-73), the effect of these changes on herbivores consuming the leaves is not
7 well characterized or clear.

8 The evidence for a second newly identified category of effects, alteration of plant-insect
9 signaling, draws on new research yielding clear evidence of O₃ modification of VPSCs and
10 behavioral responses of insects to these modified chemical signals (ISA, section IS.6.2.1). While
11 the evidence documents effects on plant production of signaling chemicals and on the
12 atmospheric persistence of signaling chemicals, as well as on the behaviors of signal-responsive
13 insects, it is limited with regard to characterization of mechanisms and the consequences of any
14 modification of VPSCs by O₃ (ISA, section IS.6.2.1). Further, the evidence includes a relatively
15 small number of plant species and plant-insect associations⁶⁶ and is limited to short, controlled
16 exposures, posing limitations for our purposes of considering the potential for associated impacts
17 to be elicited by air quality conditions that meet the current standard (ISA, section IS.6.2.1 and
18 Appendix 8, section 8.7).

19 For categories of vegetation-related effects that were recognized in past reviews, other
20 than growth and visible foliar injury (e.g., reduced plant reproduction, reduced productivity in
21 terrestrial ecosystems, alteration of terrestrial community composition and alteration of below-
22 ground biogeochemical cycles), the newly available evidence includes a variety of studies that
23 quantify exposures of varying duration in various countries using a variety of metrics (ISA,
24 Appendix 8, sections 8.4, 8.8 and 8.10). The ISA additionally describes publications that
25 summarize previously published studies in several ways. For example, a meta-analysis of
26 reproduction studies categorized the reported O₃ exposures into bins of differing magnitude,
27 grouping differing concentration metrics and exposure durations together, and performed
28 statistical analyses to reach conclusions regarding the presence of an O₃-related effect (ISA,
29 Appendix 8, section 8.4.1). While such studies continue to support conclusions of the ecological
30 hazards of O₃, they do not improve capabilities for characterizing the likelihood of such effects
31 under patterns of environmental O₃ concentrations occurring with air quality conditions that meet
32 the current standard (e.g., factors such as variation in exposure assessments and limitations in
33 response information preclude detailed analysis for such conditions).

⁶⁶ The available studies vary with regard to the experimental exposure circumstances in which the different types of effects have been reported; most of the studies have been carried out in laboratory conditions rather than in natural environments (ISA, section IS.6.2.1).

1 As at the time of the 2015 review, growth impacts, most specifically as evaluated by RBL
2 for tree seedlings and RYL for crops, remain the type of vegetation-related effects for which we
3 have the best understanding of exposure conditions likely to elicit them. Accordingly, as was the
4 case in the 2015 review, the quantitative analyses of exposures occurring under air quality that
5 meets the current standard (summarized in section 4.4 below) is focused primarily on the W126
6 index, given its established relationship with growth effects.

7 **4.3.4 Key Uncertainties**

8 The type of uncertainties for each category of effects generally tends to vary in relation to
9 the maturity of the associated evidence base from those associated with overarching
10 characterizations of the effects to those associated with quantification of the cause-and-effect
11 relationships. For example, given the longstanding nature of the evidence for many of the
12 vegetation effects identified in the ISA as causally or likely causally related to O₃ in ambient air,
13 the key uncertainties and limitations in our understanding of these effects relate largely to the
14 implications or specific aspects of the evidence, as well as to current understanding of the
15 quantitative relationships between O₃ concentrations in the environment and the occurrence and
16 severity (or relative magnitude) of such effects or understanding of key influences on these
17 relationships. For more newly identified categories of effects, the evidence may be less
18 extensive, thus precluding consideration of such details.

- 19 • **What are important uncertainties in the evidence? To what extent have important**
20 **uncertainties in the evidence identified in the past been reduced and/or have new**
21 **uncertainties been recognized?**

22 Among the categories of effects identified in past reviews, key uncertainties remain in the
23 evidence. The category of O₃ welfare effects for which current understanding of quantitative
24 relationships is strongest is reduced plant growth. As a result, this category was the focus of
25 decision-making on the standard in the 2015 review, with RBL in tree seedlings playing the role
26 of surrogate (or proxy) for the broader array of vegetation-related effects that range from the
27 individual plant level to ecosystem services. Limitations in the evidence base and associated
28 uncertainties recognized then remain and include a number of uncertainties that affect
29 characterization of the magnitude of cumulative exposure conditions that might be expected to
30 elicit growth reductions in U.S. forests. These limitations and uncertainties relate both to aspects
31 related to the extent and precision of the E-R evidence for the O₃ concentration patterns and
32 associated cumulative seasonal exposures common in areas of the U.S. that meet the current
33 standard, and with regard broader interpretation of RBL estimates with regard to longer term and
34 population and ecosystem scale impacts.

1 Uncertainties in RBL estimates for today’s O₃ air quality stem from limitations and
2 imprecision in our tools, and aspects of the underlying data. While the tree seedling E-R
3 relationships for the 11 species are long-established, there is large variation among the species
4 regarding the number of experimental datasets supporting each, and among the species and
5 experiments in the duration of the controlled exposures assessed. For example, the E-R function
6 for aspen (representing a mixture of wild type and four specific clones) is based on functions for
7 13 experimental datasets (for six different exposure studies), while the E-R functions for the red
8 maple and Virginia pine were each derived from a single experimental study (of 55 days for red
9 maple and 159 days for Virginia pine) (Appendix 4A, section 4A.2, Table 4A-6; 1996 AQCD,
10 Table 5-28; Lee and Hogsett, 1996).

11 Across these varied datasets, the controlled exposure periods vary in duration both within
12 and across years (e.g., from exposure periods of 82 to 140 days in a single year to periods of 180
13 to 555 days distributed across two years) and in whether measurements were made immediately
14 following exposure period or in the subsequent spring. The final set of E-R functions were
15 derived first for the exposure duration of the experiment and, then adjusted or normalized to 3-
16 month periods based on assumptions regarding relationships between duration, cumulative
17 exposure in terms of W126 index and plant growth response (Lee and Hogsett, 1996, section I.3;
18 Appendix 4A, Attachment 1). For example, while the functions are defined as describing a
19 seasonal response, some were derived by distributing responses observed at the end of two
20 seasons of varying exposures equally across the two seasons (essentially applying the average to
21 both seasons). Uncertainty associated with this variation in durations and assumptions inherent in
22 the adjustment step is contributed to RBL estimates derived through application of the resultant
23 functions.

24 Further, there is uncertainty associated with estimates of effects across multiple years
25 related to the limited availability of studies of seasonal growth effects on trees across multiple
26 years (particularly more than two) that have also reported detailed O₃ concentration data
27 throughout the exposure. This contributes uncertainty, and accordingly a lack of precision, to an
28 understanding of the quantitative impacts of seasonal O₃ exposure, including its year-to-year
29 variability, on tree growth and annual biomass accumulation. This uncertainty limits our
30 understanding of the extent to which tree biomass would be expected to appreciably differ at the
31 end of multi-year exposures for which the overall average exposure is the same, yet for which
32 the individual year exposures vary in different ways (e.g., as analyzed in Appendix 4D).⁶⁷

⁶⁷ Variation in annual W126 index values is described in Appendix 4D, indicating for the period, 2016-2018, that the amount by which annual W126 index values at a site differ from the 3-year average varies, but generally falls below 10 ppm-hrs across all sites and generally below 5 ppm-hrs at sites with design values at or below 70 ppb (Appendix 4D, Figure 4D-7).

1 One available study of multi-year growth effects for aspen, was summarized and assessed
2 in the 2020 and 2013 ISAs with regard the extent to which it confirmed O₃-related biomass
3 impacts estimated using the established E-R functions for aspen (King et al., 2005; 2013 ISA,
4 section 9.6.3.2; ISA, Appendix 8, section 8.13.2). The 2013 ISA applied the E-R functions to O₃
5 exposure (quantified as cumulative average seasonal W126 index) at each of six consecutive
6 years and compared the estimated aboveground biomass to estimates based on data reported for
7 each year by the study (2013 ISA, section 9.6.3.2). The conclusions reached were that the
8 experimental observations are “very close” to estimates based on the established E-R function
9 for aspen, and that “the function based on one year of growth was shown to be applicable to
10 subsequent years” (2013 ISA, p. 9-135; ISA, Appendix 8, p. 8-186). A similar assessment in the
11 2020 ISA that applied the E-R functions to O₃ exposure, quantified individually after a 92-day
12 season in each of six consecutive years similarly also concluded that predictions based on the E-
13 R functions generally agreed with the observations, given generally similar pattern and
14 magnitude of cumulative response (with some variation). In addition to indicating general
15 support for the E-R functions based on the cumulative W126 index, these assessments also
16 indicated uncertainty associated with the relative influences of individual seasonal exposures and
17 longer-term exposures, as represented by a cumulative average, given that either multiyear
18 average or single year W126 estimates provided general agreement with experimental
19 observations (Appendix 4A, section 4A.3.1; 2013 ISA, Figure 9-20; 2020 ISA, Appendix 8,
20 Figure 8-17).

21 Another area of important uncertainties relates to the extent to which the E-R functions
22 for reduced growth in tree seedlings are also descriptive of such relationships during later
23 lifestages, for which there is a paucity of established E-R relationships. Although such
24 information is limited with regard to mature trees, the analyses in the 2013 and 2020 ISAs
25 (summarized above) indicated that reported growth response of young aspen over six years was
26 similar to the reported growth response of seedlings (ISA, Appendix 8, section 8.13.2; 2013 ISA,
27 section 9.6.3.2). Evidence is lacking, however, on the shape of such relationships for older,
28 mature trees, or the extent to which these relationships in seedlings might also reflect responses
29 in older, mature trees

30 Additionally, there are uncertainties with regard to the extent to which various factors in
31 natural environments can either mitigate or exacerbate predicted O₃-plant interactions and
32 contribute variability in vegetation-related effects, including reduced growth. Such factors
33 include multiple genetically influenced determinants of O₃ sensitivity, changing sensitivity to O₃
34 across vegetative growth stages, co-occurring stressors and/or modifying environmental factors.
35 Such factors contribute uncertainties to interpretations of potential impacts in a season as well as
36 over multi-year periods. With regard to the latter, there is variability in ambient air O₃

1 concentrations from year to year, as well as year-to-year variability in environmental factors,
2 including rainfall and other meteorological factors that affect plant growth and reproduction,
3 such as through changes in soil moisture. These variabilities contribute uncertainties to estimates
4 of the occurrence and magnitude of O₃-related effects in any year, and to such estimates over
5 multi-year periods, as well as related effects in associated communities and ecosystems. All the
6 factors identified here contribute uncertainty and an associated imprecision or inexactitude to
7 estimates for trees in natural areas derived from the E-R functions and W126 index values in a
8 single year/season.

9 The uncertainties identified here are important for our interpretation of potential impacts
10 under air quality conditions that meet the current standard, which as described in section 4.4.2
11 below are generally associated with cumulative seasonal exposures lower than 20 or 25 ppm-hrs,
12 in terms of W126 index, and with quite low N100 values in a year. Such conditions are not
13 extensively represented in the datasets on which the tree seedling E-R functions are based. While
14 the functions have been concluded to provide a good fit to the underlying experimental datasets,
15 the datasets vary with regard to their representation of relatively lower O₃ treatment levels,⁶⁸ in
16 terms of W126 index (e.g., below 20 ppm-hrs). Additionally, the experimental datasets include
17 patterns of hourly concentrations that differ markedly from those common in areas meeting the
18 current standard (e.g., with greater prevalence of peak hourly concentrations). With regard to
19 W126 index level, the W126 index levels across the experiments range as high as 109.5 ppm-hrs
20 across a 121-day exposure (which, assuming a constant daily cumulative exposure would
21 correspond to 83 ppm-hrs across a 92-day season). Three of the eleven species include just one
22 of their treatment levels below a W126 index value of 20 ppm-hrs, with the other levels ranging
23 from 25.6 ppm-hrs (over 112 days) to 109.5 ppm-hrs (over 121 days), corresponding to 21 to 83
24 ppm-hrs for a 92-day season, based on assuming uniform cumulative exposure distribution
25 across the period (Appendix 4A, Table 4A-5).⁶⁹ With regard to peak concentrations, for the
26 experimental treatments with W126 index levels of a magnitude common at U.S. sites that meet
27 the current standard (e.g., less than 20 ppm-hrs for a single season), the values for N100 extend
28 up above 10, to more than 40 in one instance (Appendix 4A, Table 4A-6, black cherry and
29 aspen). Across the full set of treatments, values for N100 extend into the hundreds up to 515 in a
30 single treatment over 121 days. As discussed in section 4.4.1 below, such occurrences of
31 concentrations at or above 100 ppb are not common for U.S. sites that meet the current standard,

⁶⁸ As noted in Appendix 4A, section 4A.2, the baseline, untreated, ambient air was treated with O₃ to develop exposure levels for comparison to charcoal-filtered air and the baseline ambient air.

⁶⁹ For three of the five species in Table 4A-5 in Appendix 4A for which only one treatment exposure is for a W126 index below 20 ppm-hrs, there are three other treatments that range from a W126 index of 25 ppm-hrs up to one of 109.5 ppm-hrs (Appendix 4A, Table 4A-6).

1 at which N100 is virtually always less than 10 (and generally less than 5 [see Figure 4-7
2 below]).⁷⁰ Collectively, all of the factors identified above contribute uncertainty and an
3 associated imprecision or inexactitude to estimates based on the E-R functions for W126 index
4 levels at sites in the U.S. with air quality meeting the current standard.

5 We also note, as recognized in the 2015 review, uncertainties in the extent to which the
6 11 tree species for which there are established E-R functions encompass the range of O₃ sensitive
7 species in the U.S., and also the extent to which they represent U.S. vegetation as a whole. These
8 11 species include both deciduous and coniferous trees with a wide range of sensitivities and
9 species native to every NOAA climate region across the U.S. and in most cases are resident
10 across multiple states and regions. While recognizing this uncertainty, the available information
11 does not lead us to assume any difference in the range of sensitivity indicated by the species with
12 E-R functions.⁷¹

13 There are also uncertainties associated with our consideration of the magnitude of tree
14 growth effects, quantified as RBL, that might cause or contribute to adverse effects for trees,
15 forests, forested ecosystems, or the public welfare, these are related to various uncertainties or
16 limitations in the evidence base, including those associated with relating magnitude of tree
17 seedling growth reduction to larger-scale forest ecosystem impacts. Additionally, several factors
18 can also influence the degree to which O₃-induced growth effects in a sensitive species affect
19 forest and forest community composition and other ecosystem service flows (e.g., productivity,
20 belowground biogeochemical cycles, and terrestrial ecosystem water cycling) from forested
21 ecosystems. These include (1) the type of stand or community in which the sensitive species is
22 found (i.e., single species versus mixed canopy); (2) the role or position the species has in the
23 stand (i.e., dominant, sub-dominant, canopy, understory); (3) the O₃ sensitivity of the other co-
24 occurring species (O₃ sensitive or tolerant); and (4) environmental factors, such as soil moisture
25 and others. The lack of such established relationships with O₃ complicates consideration of the
26 extent to which different estimates of impacts on tree seedling growth would indicate
27 significance to the public welfare. Further, efforts to estimate O₃ effects on carbon sequestration
28 are handicapped by the large uncertainties involved in attempting to quantify the additional

⁷⁰ Among published studies of the datasets for the eleven E-R functions, the findings for at least one study (black cherry) reported statistical significance only for biomass effects observed for the highest O₃ exposure, which had a seasonal W126 index of 23 ppm-hrs and 77 hours with an O₃ concentration at or above 100 ppb (e.g., Appendix 4A, Table 4A-6, black cherry).

⁷¹ The CASAC in the 2015 review recognized this uncertainty, expressing the view that it should be anticipated that there are highly sensitive vegetation species for which we do not have E-R functions and others that are insensitive (Frey, 2014, p. 15), and concluding it to be more appropriate to assume that the sensitivity of species without E-R functions might be similar to the range of sensitivity for those species with E-R functions (Frey, 2014, p. 11).

1 carbon uptake by plants as a result of avoided O₃-related growth reductions. Such analyses
2 require complex modeling of biological and ecological processes with their associated sources of
3 uncertainty.

4 With regard to crop yield effects, as at the time of the 2015 review, we recognize the
5 potential for greater uncertainty in estimating the impacts of O₃ exposure on agricultural crop
6 production than that associated with O₃ impacts on vegetation in natural forests. This relates to
7 uncertainty in the extent to which agricultural management methods influence potential for O₃-
8 related effects and accordingly, the applicability of the established E-R functions for RYL in
9 current agricultural areas. Additionally, as changes in yield of commercial crops and commercial
10 commodities may affect producers and consumers differently, consideration of these effects in
11 terms of potential adversity to the public welfare impacts is limited.

12 With regard to visible foliar injury, for which longstanding evidence documents a causal
13 role for O₃, important uncertainties and limitations fall into two categories. The first category
14 relates to our understanding of the key aspects of O₃ concentrations - and other key variables
15 (e.g., soil moisture) - that have a direct bearing on the severity and incidence of vegetation
16 injury, while the second concerns the impacts on aesthetic and recreational values of various
17 severities and incidences of injury. With regard to the former, there is a lack of detailed
18 understanding of specific patterns of O₃ concentrations over a growing season and the key
19 aspects of those patterns (e.g., incidence of concentrations of particular magnitude) that
20 contribute to an increased incidence and severity of injury occurrence in the U.S. For example,
21 “the incidence of visible foliar injury is not always higher in years and areas with higher ozone,
22 especially with co-occurring drought” (ISA, Appendix 8, p. 8-24). Accordingly, there are no
23 established, quantitative E-R functions that document visible foliar injury severity and incidence
24 under varying air quality and environmental conditions (e.g., soil moisture). As discussed in
25 section 4.3.3.2 above, the available studies that have investigated the role of different variables,
26 including different metrics for characterizing O₃ concentrations over a growing season, do not
27 provide a basis for a single metric that would characterize the potential for different patterns of
28 O₃ concentrations to contribute to different incidences and severity of foliar injury in U.S.
29 forests. Further, while several studies of the USFS biosite dataset indicate a role for two metrics -
30 one reflecting cumulative, concentration-weighted exposures and a second that reflects peak
31 concentrations, statistical analyses of a number of models containing various metrics and
32 combinations of metrics have not been able to identify environmental conditions under which
33 visible foliar injury could be reliably expected (Smith, 2012; Wang et al., 2012). The second
34 category of uncertainties and limitations concerns the information that would support associated
35 judgments on the public welfare significance of different patterns of and severity of foliar injury,
36 such as the extent to which such effects in areas valued by the public for different uses may be

1 considered adverse to public welfare. In considering this issue, we note that some level of
2 severity of injury to a tree stand would be obvious to the casual observer (e.g., when viewing a
3 stand covering a hillside from a distance), and some level of severity of injury (e.g., leaf and
4 crown damage that appreciably affects overall plant physiology) would also be expected to affect
5 plant growth and reproduction. The extent to which recreational values are affected by lesser
6 levels of injury severity and incidence is not clear from the available information. Thus,
7 limitations and uncertainties in the available information, such as those described above,
8 complicate our ability to comprehensively estimate the potential for visible foliar injury, its
9 severity or extent of occurrence for specific air quality conditions, and associated public welfare
10 implications, thus affecting a precise identification of air quality conditions that might be
11 expected to provide a specific level of protection for this effect.

12 During the 2015 review, the 2013 ISA did not assess the evidence of O₃ exposure and
13 tree mortality with regard to its support for inference of a causal relationship. Evidence available
14 in the last several reviews included field studies of pollution gradients that concluded O₃ damage
15 to be an important contributor to tree mortality although several confounding factors such as
16 drought, insect outbreak and forest management were identified as potential contributors (2013
17 ISA, section 9.4.7.1). Since the 2015 review, three additional studies have been identified, as
18 summarized in section 4.3.1 above, contributing to the ISA conclusion of sufficient evidence to
19 infer a likely causal relationship for O₃ with tree mortality (ISA, Appendix 8, section 8.4). As
20 noted in the ISA, there is only limited evidence from experimental studies that isolate the effect
21 of O₃ on tree mortality, with the recently available Aspen FACE study of aspen survival
22 involving cumulative seasonal exposures above 30 ppm-hrs during the first half of the 11-year
23 study period (ISA, Appendix 8, Tables 8-8 and 8-9). Evidence is lacking regarding exposure
24 conditions closer to those occurring under the current standard and any contribution to tree
25 mortality.

26 In the case of the two newly identified categories of effects, the key uncertainties relate to
27 comprehensive characterization of the effects. For example, with regard to alteration of herbivore
28 growth and reproduction, although “there are multiple studies demonstrating ozone effects on
29 fecundity and growth in insects that feed on ozone-exposed vegetation”, “no consistent
30 directionality of response is observed across studies and uncertainties remain in regard to
31 different plant consumption methods across species and the exposure conditions associated with
32 particular severities of effects” (ISA, pp. ES-18, IS-64, IS91 and Appendix 8, section 8.6.3).
33 Such limitations and uncertainties in the evidence base for this category of effects preclude
34 broader characterization, as well as quantitative analysis related to air quality conditions meeting
35 the O₃ standard. As characterized in the ISA, uncertainties remain in the evidence; these relate to
36 the different plant consumption methods across species and the exposure conditions associated

1 with particular responses, as well as variation in study designs and endpoints used to assess O₃
2 response (ISA, IS.6.2.1 and Appendix 8, section 8.6). Thus, while the evidence describes
3 changes in nutrient content and leaf chemistry following O₃ exposure, the effect of these changes
4 on herbivores consuming the leaves is not well characterized or clear (ISA, p. IS-73).

5 The evidence for a second newly identified category of effects, alteration of plant-insect
6 signaling, draws on new research that has provided clear evidence of O₃ modification of VPSCs
7 and behavioral responses of insects to these modified chemical signals. Most of these studies,
8 however, have been carried out in laboratory conditions rather than in natural environments, and
9 involve a relatively small number of plant species and plant-insect associations. While the
10 evidence documents effects on plant production of signaling chemicals and on the atmospheric
11 persistence of signaling chemicals, as well as on the behaviors of signal-responsive insects, it is
12 limited with regard to characterization of mechanisms and the consequences of any modification
13 of VPSCs by O₃ (ISA, section IS.6.2.1). Further, the available studies vary with regard to the
14 experimental exposure circumstances in which the different types of effects have been reported
15 (most of the studies have been carried out in laboratory conditions rather than in natural
16 environments), and many of the studies involve quite short controlled exposures (hours to days)
17 to elevated concentrations, posing limitations for our purposes of considering the potential for
18 impacts associated with the studied effects to be elicited by air quality conditions that meet the
19 current standard (ISA, section IS.6.2.1 and Appendix 8, section 8.7).

20 With regard to radiative forcing and climate effects, “uncertainty in the magnitude of
21 radiative forcing estimated to be attributed to tropospheric ozone is a contributor to the relatively
22 greater uncertainty associated with climate effects of tropospheric ozone compared to such
23 effects of the well mixed greenhouse gases (e.g., carbon dioxide and methane)” (ISA, section
24 IS.6.2.2). With regard to O₃ effects on temperature, “the heterogeneous distribution of ozone in
25 the troposphere complicates the direct attribution of spatial patterns of temperature change to
26 ozone induced RF” and the existence of O₃ climate feedbacks “further alter the relationship
27 between ozone RF and temperature (and other climate variables) in complex ways” (ISA,
28 Appendix 9, section 9.3.1). Thus, various uncertainties “render the precise magnitude of the
29 overall effect of tropospheric ozone on climate more uncertain than that of the well-mixed
30 GHGs” (ISA, Appendix 9, section 9.3.3). Further, “[c]urrent limitations in climate modeling
31 tools, variation across models, and the need for more comprehensive observational data on these
32 effects represent sources of uncertainty in quantifying the precise magnitude of climate responses
33 to ozone changes, particularly at regional scales” (ISA, Appendix 9, section 9.3.3).

4.4 EXPOSURE AND AIR QUALITY INFORMATION

In general, decision-making in the 2015 review placed greatest weight on estimates of cumulative exposures to vegetation based on ambient air monitoring data and consideration of those estimates in light of E-R functions for O₃-related reduction in tree seedling growth (summarized in section 4.3.3 above). These analyses supported the consideration of the potential for O₃ effects on tree growth and productivity, as well as its associated impacts on a range of ecosystem services, including forest ecosystem productivity and community composition (80 FR 65292, October 26, 2015). These analyses were recognized as involving relatively reduced uncertainty (compared to the national or regional-scale modeling performed in the 2015 review) for the purposes of informing a characterization of cumulative O₃ exposure (in terms of the W126 index) associated with air quality just meeting the existing standard (IRP, section 5.2.2). The lesser uncertainty of these air quality monitoring-based analyses contributed to their being more informative in the 2015 review and to their being updated in the 2020 PA. A second set of air quality analyses was also considered in the 2020 decision; these analyses investigated the occurrence of peak concentrations at sites for which the O₃ concentrations meet different design values or contribute to different cumulative exposure levels in terms of the W126 index (Wells, 2020). Both sets of analyses have been updated for this reconsideration of the 2020 decision using the more recently available air quality data now available (Appendices 4D and 4F).

The first set of analyses are air quality and exposure analyses. They are an update of the analyses considered in the 2015 decision establishing the current standard, and in the 2020 decision to retain that standard. This set of analyses, in 2015 and 2020, as well as the current updated analyses presented here, evaluate W126-based cumulative exposure estimates at all U.S. monitoring locations, nationwide, and at the subset of sites in or near Class I areas, during 3-year periods that met the then-current standard and potential alternatives (80 FR 65485-86, Table 3, October 26, 2015; Wells, 2015; 2020 PA, section 4.4). For the 2015 and 2020 decisions, W126 index values⁷² occurring in locations with air quality meeting the then-current standard (or potential alternatives) were considered in the context of the magnitude of W126 exposure index associated with an estimate of 6% RBL in tree seedlings for the median tree species among the 11 species for which there are established E-R relationships (80 FR 65391-92, Table 4, October 26, 2015; 2020 PA, section 4.4). That magnitude of W126 index is 19 ppm-hrs (80 FR 65391-65392). This set of analyses also includes an evaluation of relationships between W126 index

⁷² Based on judgments in the last review, the W126 metric analyzed and considered in the 2015 decision was the 3-year average of consecutive year seasonal W126 index values (derived as described in section 4.3.3.1 above).

1 values and design values⁷³ based on the form and averaging time of the then-current secondary
2 standard (Wells, 2015; 2020 PA, section 4.4).

3 The second set of analyses (initially performed for consideration in the 2020 decision and
4 updated here) focus on the occurrence of peak concentrations, investigating the occurrence of
5 peak concentrations at sites for which the O₃ concentrations meet different design values or
6 contribute to different cumulative exposure levels in terms of the W126 index. The metrics used
7 for these analyses are the number of hours in a year for which the O₃ concentration was at or
8 above 100 ppb (N100), and the number of days in a year in which there was at least one hour
9 with an O₃ concentration at or above 100 ppb (D100). The value of 100 ppb is used here as it has
10 been in some studies focused on O₃ effects on vegetation (and discussed in section 4.3.3.2
11 above), simply as an indicator of elevated or peak hourly O₃ concentrations (e.g., Lefohn et al.,
12 1997, Smith, 2012; Davis and Orendovici, 2006; Kohut, 2007). Other values that have also been
13 considered in this way in other studies are 95 ppb and 110 ppb (2013 ISA, section 9.5.3.1). These
14 analyses provided additional information for the 2020 review beyond that provided by the first
15 set of analyses that focused only on W126 index.

16 Both sets of analyses described here have been performed with the expanded set of air
17 monitoring data now available,⁷⁴ which includes 1,578 monitoring sites with sufficient data for
18 derivation of design values (Appendix 4D, section 4D.2.2; Appendix 4F). Both sets of analyses
19 include a component based on data for the most recent periods, and a second component
20 considering data across the full historical period back to 2000, which is now expanded from that
21 previously available.⁷⁵ The most recent data analyzed are those for the design value period from
22 2018 to 2020. The first set of analyses include a focus on all sites in the U.S., as well as on the
23 subset of sites in or near Class I areas is described in detail in Appendix 4D. The second set of
24 analyses, which investigate the occurrence of peak concentrations at sites varying by design
25 value and W126 index, are described in detail in Appendix 4F.

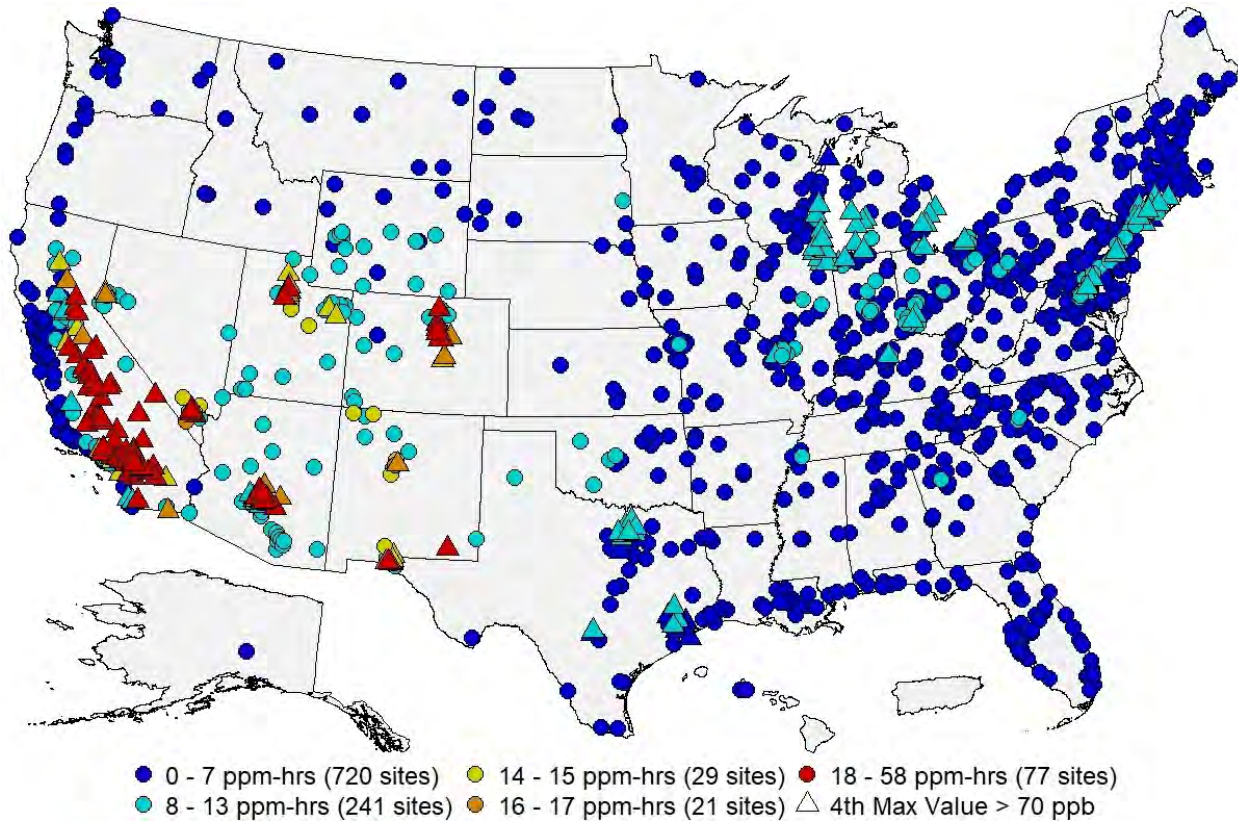
26 For all monitoring sites with valid design values for the recent period of 2018 through
27 2020, Figure 4-6 presents the 3-year average seasonal W126 index and also denotes whether
28 each site meets the current standard. Similarly, Figure 4-7 and Figure 4-8 present N100 and
29 D100 values, respectively, for these sites. Consideration of all three figures indicates that the

⁷³ As described in earlier chapters, a design value is a statistic that describes the air quality status of a given area relative to the level of the standard, taking the averaging time and form into account. For example, a design value of 75 would have indicated O₃ concentrations that just met the prior standard in a specific 3-yr period.

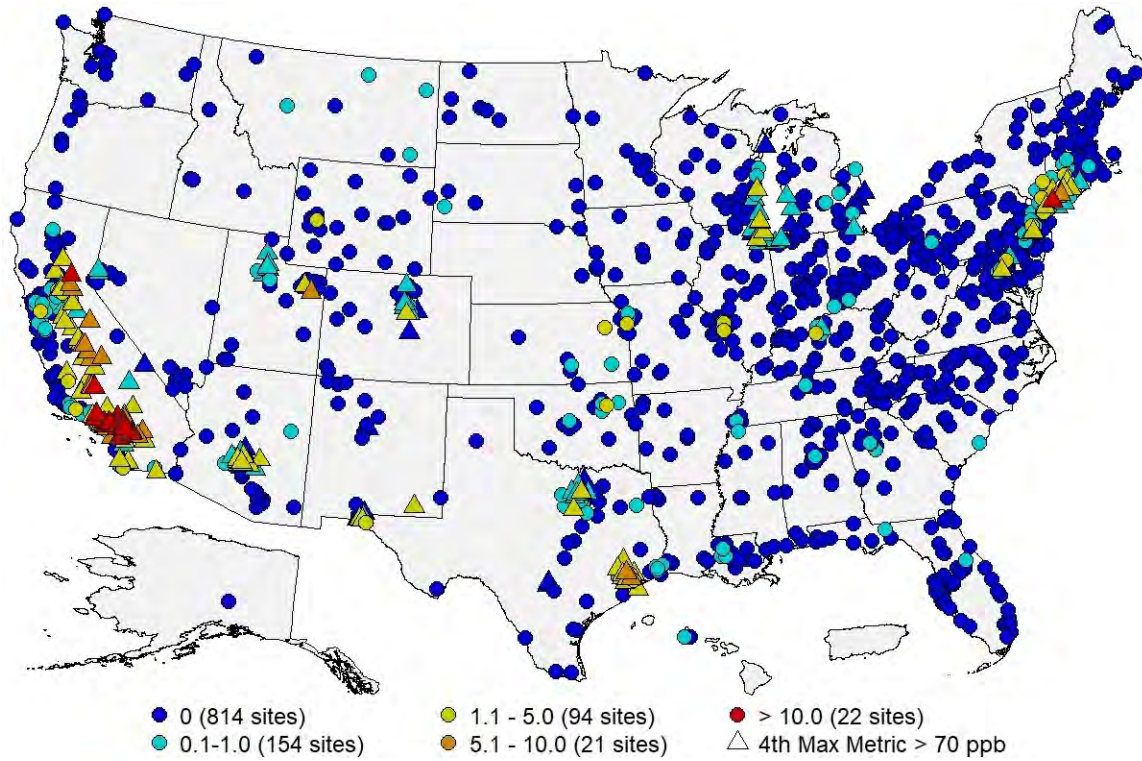
⁷⁴ In addition to being expanded with regard to data for more recent time periods than previously available, the current dataset also includes a small amount of newly available older data for some monitoring sites that are now available in the AQS.

⁷⁵ In the 2015 review, the dataset analyzed included data from 2000 through 2013 (Wells, 2015).

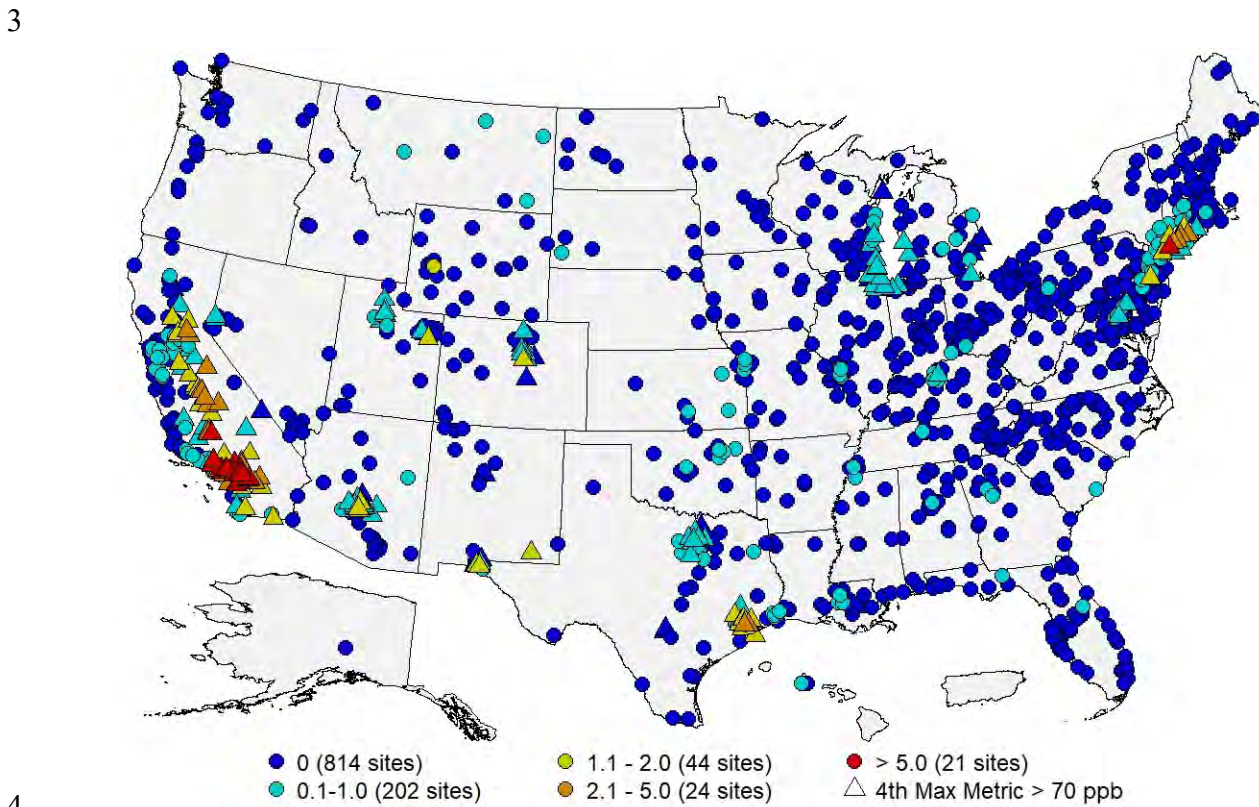
1 monitoring sites with design values above the level of the current standard have the higher W126
2 index values and also the higher values of N100 and D100 (compared to monitoring sites not
3 meeting the current standard, denoted with triangles). It can also be seen that there are some sites
4 that have relatively lower W126 index values, e.g., in the Northwest, Northeast and Midwest,
5 while recording N100 or D100 values of more than 5 (including some N100 values above 1010
6 and 5 respectively). The sections below summarize more completely the findings of all the air
7 quality analyses involving these three metrics.
8



10 **Figure 4-6. W126 index at monitoring sites with valid design values (2018-2020 average).**



1
2 **Figure 4-7. N100 values at monitoring sites with valid design values (2018-2020 average).**



4
5 **Figure 4-8. D100 values at monitoring sites with valid design values (2018-2020 average).**

1 **4.4.1 Influence of Form and Averaging Time of Current Standard on W126 Index and** 2 **Peak Concentration Metrics**

3 In revising the standard in 2015 to the now-current standard, the Administrator concluded
4 that, with revision of the standard level, the existing form and averaging time provided the
5 control needed to achieve the cumulative seasonal exposure circumstances identified for the
6 secondary standard (80 FR 65408, October 26, 2015). The focus on cumulative seasonal
7 exposure primarily reflected the evidence on E-R relationships for plant growth. The 2015
8 conclusion was based on the air quality data analyzed at that time (80 FR 65408, October 26,
9 2015). Analyses of the now expanded set of air monitoring data, which includes 1,578
10 monitoring sites with sufficient data for derivation of design values (Appendix 4D, section
11 4D.2.2), document similar findings as from the analysis of data from 2000-2013 described in the
12 2015 review, and the 2020 analysis of 2000-2018 data. The current (updated) analyses, which
13 now span 21 years and 19 3-year periods, are described in detail in Appendix 4D.

14 These analyses document the positive nonlinear relationship that is observed between
15 cumulative seasonal exposure, quantified using the W126 index, and design values, based on the
16 form and averaging time of the current standard. This is shown for both the average W126 index
17 across the 3-year design value period (Figure 4-9, left) and for annual index values within the
18 period (Figure 4-9, right). For both annual and 3-year average index values, it is clear that
19 cumulative seasonal exposures, assessed in terms of W126 index, are lower at monitoring sites
20 with lower design values. This is seen both for design values above the level of the current
21 standard (70 ppb), where the slope is steeper (due to the sigmoidal weighting of higher
22 concentrations by the W126 index function), as well as for lower design values that meet the
23 current standard (Figure 4-9; Appendix 4D). These presentations also indicate some regional
24 differences. For example, as shown in Figure 4-6 and Figure 4-9 for the 2018-2020 period, sites
25 meeting the current standard in the regions outside of the West and Southwest regions, all 3-year
26 average W126 index values (and virtually all annual values) are at or below 13 ppm-hrs. Ozone
27 concentrations, and W126 index values, are generally higher in the West and Southwest regions
28 (Figure 4-6). However, the positive relationship between the W126 index and the design value is
29 evident in all regions (Figure 4-9).

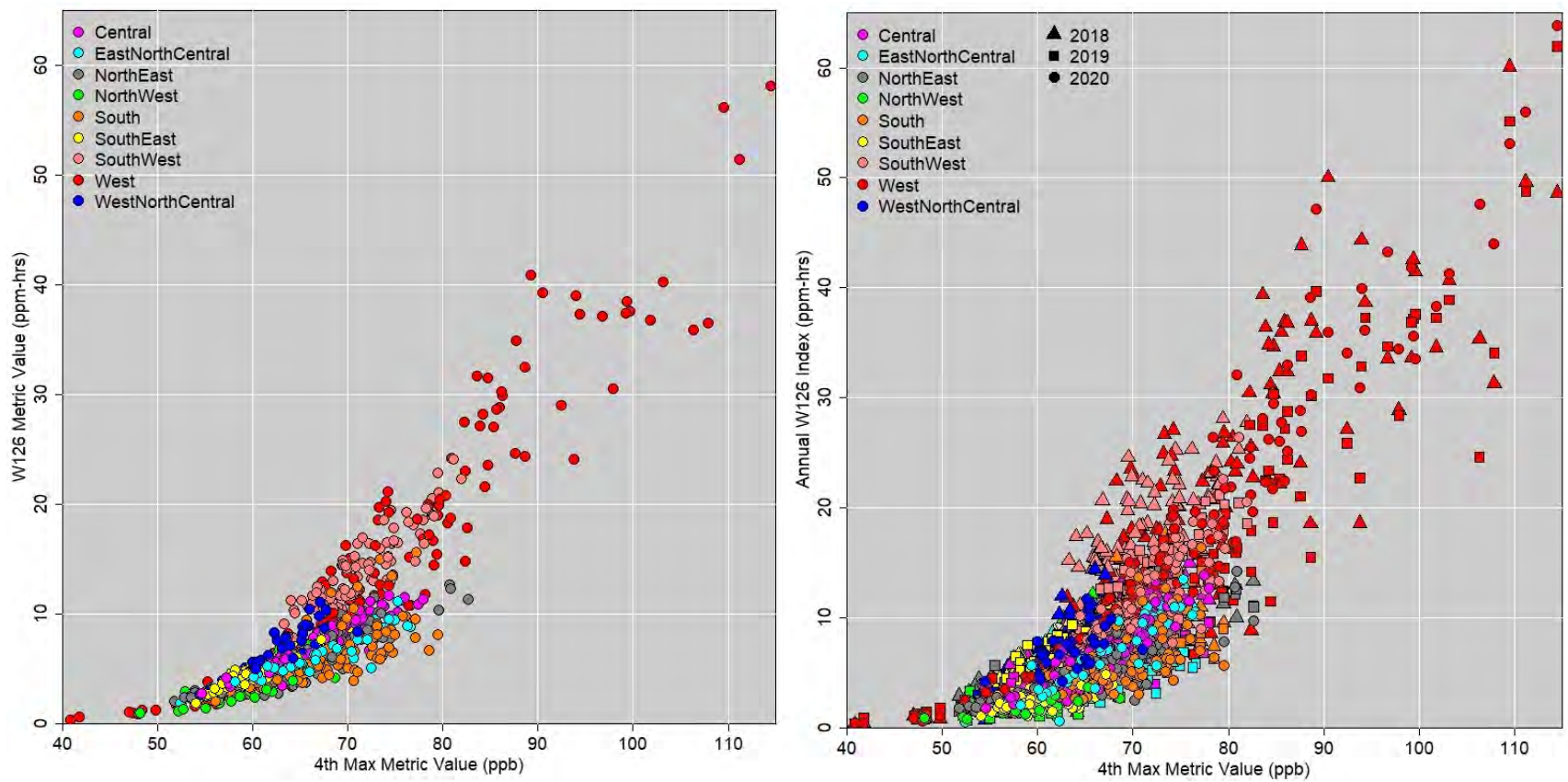


Figure 4-9. Relationship between the W126 index and design values for the current standard (2018-2020). The W126 index is analyzed in terms of averages across the 3-year design value period (left) and annual values (right).

1 An additional analysis, which was also performed in the 2015 review with the then-
2 available data, assesses the relationship between long-term changes in design value and long-
3 term changes in the W126 index (presented in detail in Appendix 4D, section 4D.3.2.3). Ozone
4 monitoring data have well documented reductions in O₃ design values in response to national
5 programs to control O₃ precursors (see section 2.4.2 above). The current analysis explores the
6 extent to which the W126 index has responded to these declines by focusing on the relationship
7 between changes (at each monitoring site) in the 3-year design value (termed “4th max” in
8 Appendix 4D, Figure 4-10 and Figure 4-10) across the 19 design value periods from 2000-2002
9 to 2018-2020 and changes in the W126 index over the same period.⁷⁶ This analysis, performed
10 using either the 3-year average W126 index or annual values, shows there to be a positive, linear
11 relationship between the changes in the W126 index and the changes in the design value at
12 monitoring sites across the U.S. (Figure 4-10). This means that a change in the design value at a
13 monitoring site was generally accompanied by a similar change in the W126 index (e.g., a
14 reduction in design value accompanied by a reduction in W126 index). Nationally, the W126
15 index (in terms of 3-year average) decreased by approximately 0.59 ppm-hrs per ppb decrease in
16 design value over the full period from 2000 to 2020. This relationship varies across the NOAA
17 climate regions, with the greatest change in the W126 index per unit change in design value
18 observed in the Southwest and West regions. Thus, the regions which had the highest W126
19 index values at sites meeting the current standard (Figure 4-10) also showed the greatest
20 improvement in the W126 index per unit decrease in their design values over the past 21 years
21 (Appendix 4D, Table 4D-12 and Figure 4D-12). This indicates that going forward as design
22 values are reduced in areas that are presently not meeting the current standard, the W126 index
23 in those areas would also be expected to decline (Appendix 4D, section 4D.3.2.3 and 4D.4).

24 Thus, the air quality analyses indicate control by the form and averaging time of the
25 current standard of W126 index exposures, both in terms of 3-year average and single-year
26 values. The overall trend showing reductions in the W126 index concurrent with reductions in
27 the design value metric for the current standard is positive whether the W126 index is expressed
28 in terms of the average across the 3-year design value period or the annual value (Appendix 4D,
29 section 4D.3.2.3). This similarity is consistent with the relationship between the W126 index and
30 the design value metric for the current standard summarized above, which shows a strong
31 positive relationship between those metrics (Figure 4-9, Appendix 4D, section 4D.3.1.2).

⁷⁶ At each site, the trend in values of a metric (W126 or 4th max), in terms of a per-year change in metric value, is calculated using the Theil-Sen estimator, a type of linear regression method that chooses the median slope among all lines through pairs of sample points. For example, if applying this method to a dataset with metric values for four consecutive years (e.g., W126₁, W126₂, W126₃, W126₄), the trend would be the median of the different per-year changes observed in the six possible pairs of values ([W126₄- W126₃]/1, [W126₃- W126₂]/1, [W126₂- W126₁]/1, [W126₄- W126₂]/2, [W126₃- W126₁]/2, [W126₄- W126₁]/3).

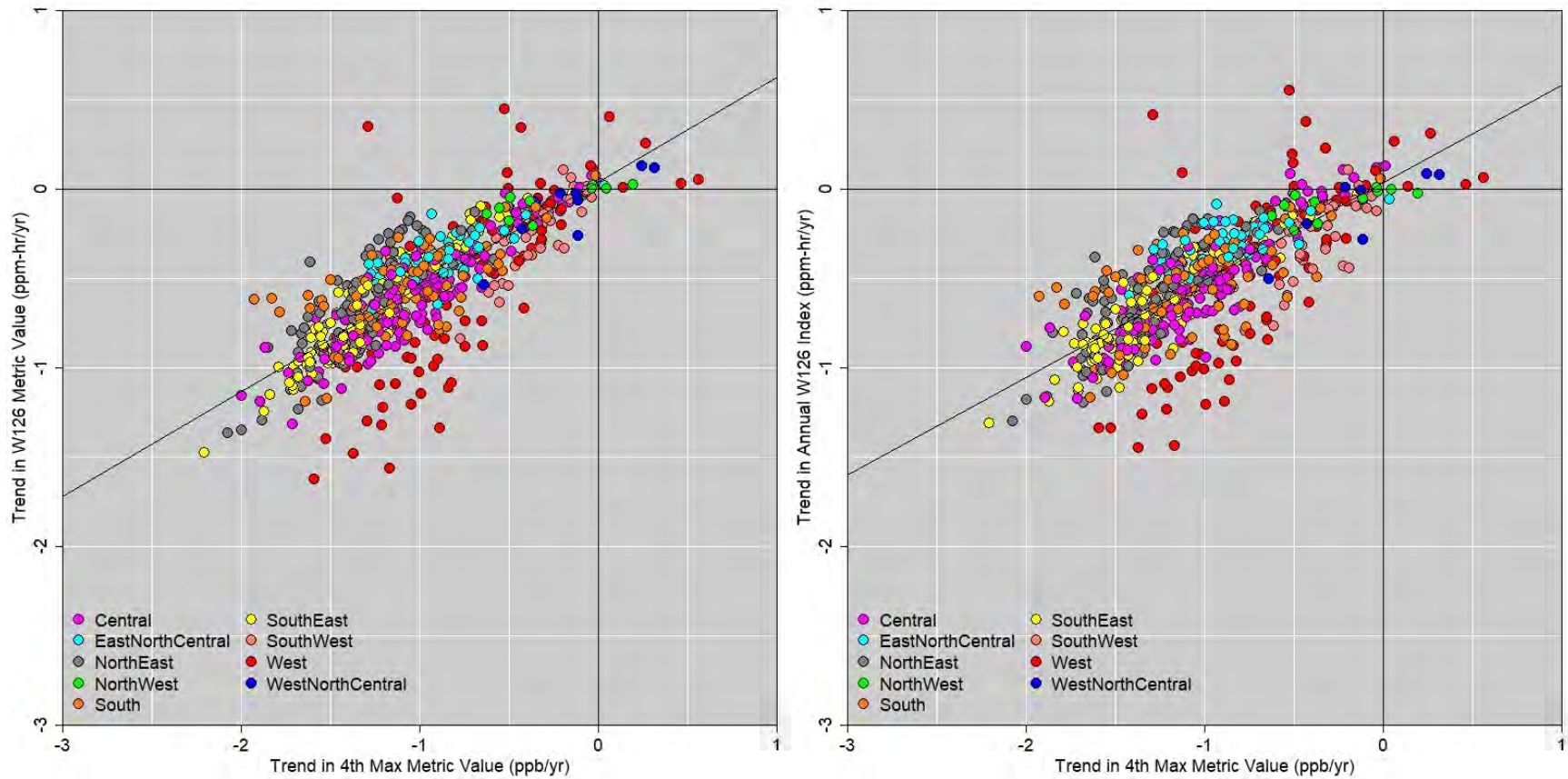
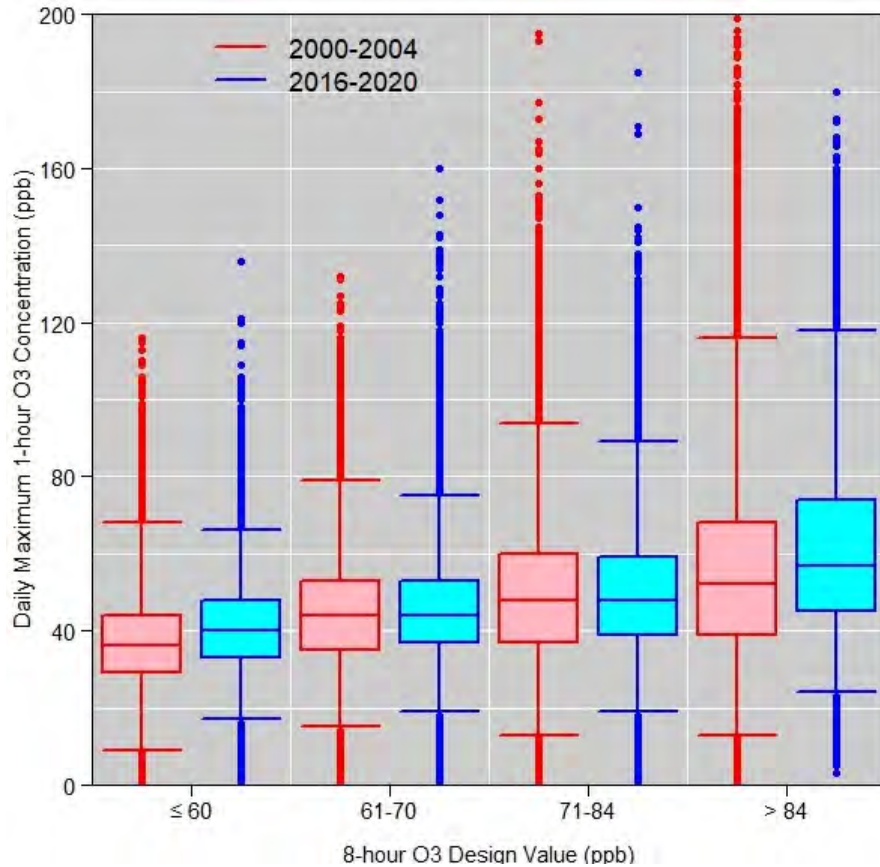


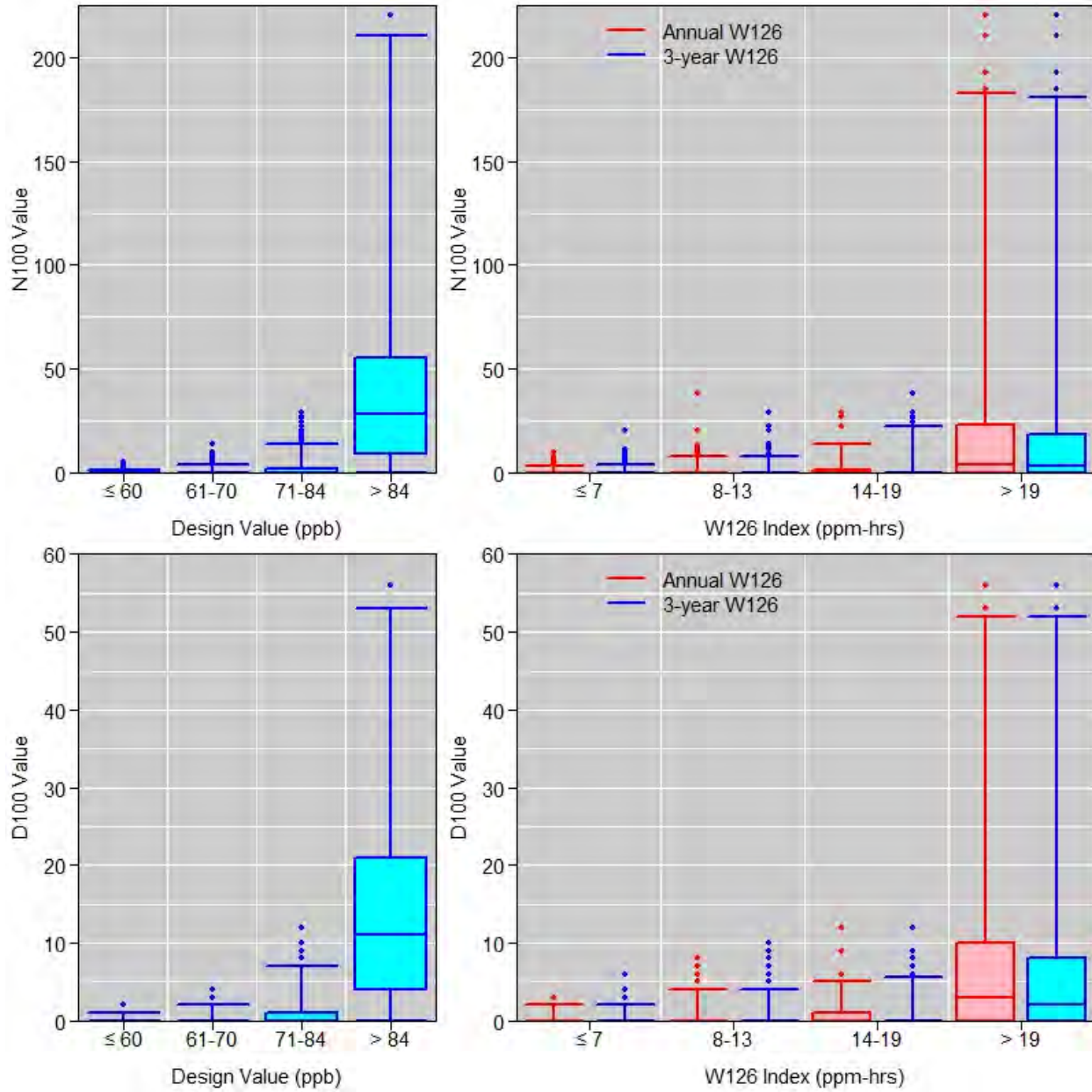
Figure 4-10. Relationship between trends in the W126 index and trends in design values across a 21-year period (2000-2020) at U.S. monitoring sites. W126 is analyzed in terms of averages across 3-year design value periods (left) and annual values (right).

1 In considering the control of the current form and averaging time on vegetation exposures
2 of potential concern, we additionally take note of the evidence discussed in section 4.3.3.2 above
3 regarding the potential for days with particularly high O₃ concentrations to play a contributing
4 role in vegetation effects. While the occurrence and severity of visible foliar injury indicates
5 some relationship with cumulative concentration-weighted indices such as SUM06 and W126,
6 the evidence also indicates a contributing role for occurrences of peak concentrations. We note
7 that the current standard's form and averaging time, by their very definition, limit such
8 occurrences. For example, the peak 8-hour average concentrations are lower at sites with lower
9 design values, as illustrated by the declining trends in annual fourth highest MDA8
10 concentrations that accompany the declining trend in design values described in chapter 2 (e.g.,
11 Figure 2-11). Additionally, peak hourly concentrations are also lower with lower design values.
12 As shown in Figure 4-11, the 99th through 25th percentile daily maximum 1-hour concentrations
13 (MDA1) are lower with lower design values. This is true both for the most recent three design
14 value periods and the three periods in 2000 through 2004. Additionally Figure 4-11 shows that
15 for sites with design values below the level of the standard (i.e., at or below 70 ppb) the 99th
16 percentile of daily maximum 1-hour ozone concentrations is less than 80 ppb. Further analyses
17 summarized in Appendix 2A document many fewer hourly concentrations at or above 100 ppb at
18 sites that meet the current standard compared to sites that do not. For example, the average
19 number of hours at or above 100 ppb per site in a 3-year period was well below one for sites
20 meeting the current standard compared to approximately 10 occurrences per site for sites not
21 meeting the current standard (Appendix 2A, Table 2A-2). This pattern also holds for hourly
22 concentrations at or above 120 or 160 ppb and is true for the recent air quality as well as past air
23 quality (Appendix 2A, Tables 2A-2 through 2A-4).



1
 2 **Figure 4-11. Distributions of MDA1 concentrations for the three design value periods in**
 3 **2000-2004 (red) and 2016-2020 (blue), binned by the design value at each**
 4 **monitoring site. Boxes represent the 25th, 50th and 75th percentiles; whiskers**
 5 **represent the 1st and 99th percentiles; and circles are outlier values.**

6 An additional investigation into the extent of control the current standard exerts on peak
 7 concentrations is described in the set of analyses presented in Appendix 4F. This investigation
 8 tallied the number of hours at or above 100 ppb (N100), and the number of days with an hour at
 9 or above 100 ppb (D100), at sites meeting different criteria with regard to seasonal W126 index,
 10 in a single year and as an average across three years, and also at sites with varying design values.
 11 The strong control of these peak concentration metrics exerted by the current standard is
 12 illustrated in Figure 4-12 by the low values common at sites meeting the current standard (design
 13 value of 70 ppb or lower). The parallel presentation for varying values of W126 index suggests
 14 that this metric has generally less potential for control of such peaks (Figure 4-12). For example,
 15 the distributions for N100 and D100 observed for monitoring sites meeting the current standard
 16 are more compressed and have lower maximum values than any of the W126 bins, with the
 17 lowest bins (for W126 index values at or below 7 ppm-hrs) being most similar (Figure 4-12).



1
 2 **Figure 4-12. Distributions of N100 (top panels) and D100 (bottom panels) values at**
 3 **monitoring sites differing by design values (left panels) and W126 index values**
 4 **(right panels) based on 2018-2020 monitoring data. The boxes represent the**
 5 **25th, 50th and 75th percentiles and the whiskers extend to the 1st and 99th.**

1 In considering the prevalence of peak concentrations occurring at monitoring sites, it can
 2 be seen that O₃ concentrations at or above 100 ppb occur at lower prevalence at sites that meet
 3 the current standard than at sites that meet a range of W126 index values. As shown in Table 4-1,
 4 during the highest year for the different N100 or D100 thresholds, the percentage of sites
 5 exceeding those thresholds is greater for the sites restricted to meet the different annual W126
 6 levels, with the exception of 7 ppm-hrs, than it is for sites meeting the current standard (design
 7 values [3-year 4th Max] at or below 70 ppb) for which the percentages are similar to those for the
 8 sites meeting a W126 of 7 ppm-hrs. This observation can also be made for the average
 9 percentages across the 3-year period. Further, in looking at the three most recent 3-year periods
 10 (extending from 2016 through 2020), a similar finding holds (Table 4-2).

11 **Table 4-1. Percent of monitoring sites during the 2018 to 2020 period with 4th max or**
 12 **W126 metrics at or below various thresholds that have N100 or D100 values**
 13 **above various thresholds.**

	Total Number of Sites	Number of sites where:			Number of sites where:		
		N100 > 0	N100 > 5	N100 > 10	D100 > 0	D100 > 2	D100 > 5
<i>Average percent of sites exceeding N100 or D100 threshold per year*</i>							
3-year 4 th Max ≤ 70	877	6%	0.4%	<0.1%	6%	0.3%	0%
Annual W126 ≤ 25	1134-1144	11%	1.7%	0.5%	11%	1.7%	0.3%
Annual W126 ≤ 19	1091-1129	10%	1.3%	0.3%	10%	1.3%	0.2%
Annual W126 ≤ 17	1067-1117	9.3%	1.3%	0.2%	9.3%	1.3%	0.2%
Annual W126 ≤ 15	1031-1091	9%	1.2%	0.2%	9%	1.2%	0.1%
Annual W126 ≤ 7	626-860	5.3%	0.4%	0%	5.3%	0.4%	0%
Annual 4 th Max ≤ 70	802-1000	3.7%	0%	0%	3.7%	0%	0%
<i>Percent of sites exceeding N100 or D100 threshold in maximum year of the three</i>							
3-year 4 th Max ≤ 70	See above	9%	0.6%	0.1%	9%	0.5%	0%
Annual W126 ≤ 25		15%	2%	0.6%	15%	2%	0.4%
Annual W126 ≤ 19		13%	2%	0.4%	13%	28%	0.3%
Annual W126 ≤ 17		13%	2%	0.3%	13%	2%	0.3%
Annual W126 ≤ 15		13%	2%	0.3%	13%	2%	0.3%
Annual W126 ≤ 7		8%	1%	0%	8%	1%	0%
Annual 4 th Max ≤ 70		4%	0%	0%	4%	0%	0%
* For the annual metrics, the entries for each N100 or D100 column may be for different years in the 3-year period. Thus the "Total Number of Sites" column presents the range in number of sites that meet the annual 4 th Max or W126 thresholds in each of the three years (as presented in Table 4F-2, Appendix 4F).							

14

1 **Table 4-2. Average percent of monitoring sites per year during 2016-2020 with 4th max or**
 2 **W126 metrics at or below various thresholds that have N100 or D100 values**
 3 **above various thresholds.**

	Total Number of Sites	Percent of sites where:			Percent of sites where:		
		N100 > 0	N100 > 5	N100 > 10	D100 > 0	D100 > 2	D100 > 5
<i>Average percent of sites exceeding N100 or D100 threshold per year (2016 – 2020)</i>							
3-year 4 th Max ≤ 70		5.1%	0.3%	0.01%	5.1%	0.2%	0%
Annual W126 ≤ 25		11.0%	1.7%	0.5%	11.0%	1.8%	0.4%
Annual W126 ≤ 19		10.0%	1.4%	0.3%	10.0%	1.4%	0.2%
Annual W126 ≤ 17		9.5%	1.2%	0.2%	9.5%	1.2%	0.1%
Annual W126 ≤ 15		9.1%	1.2%	0.2%	9.1%	1.1%	0.1%
Annual W126 ≤ 7		5.1%	0.4%	0%	5.1%	0.3%	0%
Annual 4 th Max ≤ 70		3.3%	0.02%	0%	3.3%	0.3%	0%

Drawn from Appendix 4F, Table 4F-3.

4
 5 These air quality analyses illustrate limitations of the W126 index for purposes of
 6 controlling peak concentrations, and also the strengths of the current standard in this regard. As
 7 discussed more fully in section 4.5.1.1 below, the W126 index cannot, by virtue of its definition,
 8 always differentiate between air quality patterns with high peak concentrations and those without
 9 such concentrations. This is demonstrated in the air quality analyses referenced above which
 10 indicate that the form and averaging time of the existing standard is much more effective than the
 11 W126 index in limiting peak concentrations (e.g., hourly O₃ concentrations at or above 100 ppb)
 12 and in limiting number of days with any such hours (e.g., Appendix 4F, Figures 4F-4, 4F-5, 4F-
 13 8, 4F-9 compared to Figures 4F-6, 4F-7, 4F-10 and 4F-11). A similar finding is evidenced in the
 14 historical data extending back to 2000. These data show the appreciable reductions in peak
 15 concentrations that have been achieved in the U.S. as air quality has improved under O₃
 16 standards of the existing form and averaging time (Appendix 4F, Figures 4F-12 and 4F-13).
 17 From the analyses, it can be seen that the form and averaging time of the current standard is
 18 effective in controlling peak hourly concentrations and that a W126 index-based standard would
 19 be much less effective in providing the needed protection against years with such elevated and
 20 potentially damaging hourly concentrations.

21 In summary, monitoring sites with lower O₃ concentrations as measured by the design
 22 value metric (based on the current form and averaging time of the secondary standard) have
 23 lower cumulative seasonal exposures, as quantified by the W126 index, and also lower short-
 24 term peak concentrations, thus indicating a level of control exerted by the current standard on
 25 these other metrics. As the form and averaging time of the secondary standard have not changed

1 since 1997, the decreasing trends in W126 index and in hourly and 8-hour daily maximum
2 concentrations over time also support the finding that a change in level (i.e., from 80 ppb in 1997
3 to 75 ppb in 2008 to 70 ppb in 2015) for a standard of the current form and averaging time
4 contributes to reductions in the level on cumulative seasonal exposures in terms of W126 index
5 (and on the magnitude of short-term peak concentrations). That is, that reductions in design
6 value, presumably associated with implementation of the revised standards, have been
7 accompanied by reductions in cumulative seasonal exposures in terms of W126 index, as well as
8 reductions in short-term peak concentrations. Further, the analyses focused on N100 and D100
9 metrics provide additional evidence of the control of the current standard on peak concentrations,
10 and also indicate a likely lesser effectiveness of the W126 index metric in providing such
11 control. Altogether, the analyses summarized here demonstrate the form and averaging time of
12 the current standards to be effective in controlling cumulative, concentration-weighted exposures
13 as well as peak hourly concentrations (e.g., concentrations at/above 100 ppb), two metrics that
14 have been found to be important to O₃ effects on vegetation (as discussed in section 4.3 above).

15 **4.4.2 Environmental Exposures in Terms of W126 Index**

16 Given the evidence indicating the W126 index to be strongly related to growth effects
17 and its use in the E-R functions for tree seedling RBL, exposure in the analyses described here is
18 quantified using the W126 metric (Figure 4-13). These analyses are intended to inform
19 conclusions regarding the magnitude of cumulative, concentration-weighted exposures, in terms
20 of W126 index, likely to occur in areas that meet the current standard. In light of the importance
21 placed on Class I areas in past secondary standard reviews and the greater public welfare
22 significance of O₃ related impacts in such areas, as discussed in section 4.3.2 above, a separate
23 evaluation is conducted on cumulative O₃ exposure at monitoring sites in or near Class I areas⁷⁷,
24 in addition to that at all monitoring sites nationwide. The potential for impacts of interest is
25 assessed through considering the magnitude of estimated exposure in light of current information
26 and, in comparison to levels given particular focus in the 2015 decision on the current standard
27 (80 FR 65292; October 26, 2015).⁷⁸

⁷⁷ Included are monitors sited within Class I areas or the closest monitoring site within 15 km of the area boundary.

⁷⁸ The W126 index values were rounded to the nearest unit ppm-hr for these comparisons to a specific whole-number W126 level (Appendix 4D, section 4D.2).

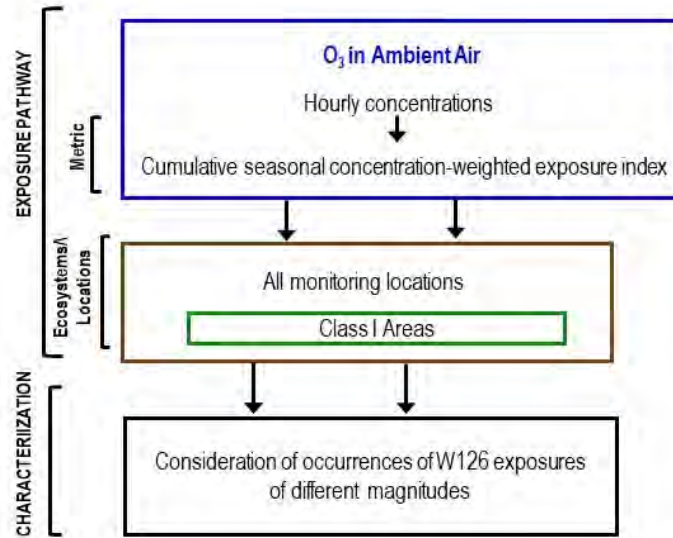


Figure 4-13. Analytical approach for characterizing vegetation exposure with W126 index.

The updated analyses discussed here and described in greater detail in Appendix 4D include assessment of all monitoring sites nationally and also a focused evaluation in Class I areas for which such monitoring data are available. The analyses include air quality monitoring data for the most recent 3-year period (2018 to 2020) for which data were available when the analyses were performed, and also all 3-year periods going back as far as the 2000-2002 period. Design values (3-year average annual fourth-highest 8-hour daily maximum concentration, also termed “4th max metric” in this analysis) and W126 index values (in terms of the 3-year average) were calculated at each site where sufficient data were available.⁷⁹ Across the nineteen 3-year periods from 2000-2002 to 2018-2020, the number of monitoring sites with sufficient data for calculation of valid design values and W126 index values ranged from a low of 992 in 2000-2002 to a high of 1,118 in 2015-2017. As specific monitoring sites differed somewhat across the 21 years, there were 1,578 sites with sufficient data for calculation of valid design values and W126 index values for at least one 3-year period between 2000 and 2020, and 510 sites had such data for all nineteen 3-year periods. The sections below discuss key aspects of these analyses and what they indicate with regard to protection from vegetation-related effects of potential public welfare significance.

The analyses of cumulative seasonal exposures included a focus on the W126 index in terms of the average seasonal index across the 3-year design value period, with additional analyses also characterizing the annual W126 index. Among the analyses performed is an evaluation of the variability of annual W126 index values across the 3-year period (Appendix

⁷⁹ Data adequacy requirements and methods for these calculations are described in Appendix 4D, section 4D.2.

1 4D, section 4D.3.1.2). This evaluation was performed for all monitoring sites in the most recent
2 3-year period, 2018 to 2020. This analysis indicates the extent to which single-year values within
3 the 3-year period deviate from the average for the period. Across the 877 sites (Appendix 4D,
4 Table 4D-1) meeting the current standard (design value at or below 70 ppb), 99% of single-year
5 W126 values in this subset differ from the 3-year average by no more than 5 ppm-hrs, and 78%
6 by no more than 2 ppm-hrs (Appendix 4D, Figure 4D-7).

7 The following discussion is framed by a key policy-relevant question based on those
8 identified in the IRP. The question considers all areas nationally, with particular focus on air
9 quality data for Class I areas.

- 10 • **What are the nature and magnitude of vegetation exposures associated with**
11 **conditions meeting the current standard at sites across the U.S., particularly in**
12 **specially protected areas, such as Class I areas, and what do they indicate regarding**
13 **the potential for O₃-related vegetation impacts?**

14 To address this question, we considered both recent air quality (2018-2020) and air
15 quality since 2000. These air quality analyses of cumulative seasonal exposures associated with
16 conditions meeting the current standard nationally provide conclusions generally similar to those
17 based on the data available at the time of the 2015 review when the current standard was set,
18 when the most recent data available for analysis were 2011 to 2013 (Wells, 2015). Cumulative
19 exposures vary across the U.S, with the highest W126 index values for sites that met the current
20 standard being located exclusively in the Southwest and West climate regions (Figure 4-6). In all
21 other NOAA climate regions, average W126 index values (for the 3-year period, 2018-2020) at
22 sites meeting the current standard are generally at or below 13 ppm-hrs (Figure 4-6). In the
23 Southwest and West, W126 index values at all sites meeting the current standard are at or below
24 17 ppm-hrs in the most recent 3-year period (Figure 4-6) and virtually all sites meeting the
25 current standard are at or below 17 ppm-hr across all of the nineteen 3-year periods in the full
26 dataset evaluated⁸⁰ (Table 4-3). Additionally, the historical dataset includes no occurrences of a
27 3-year average W126 index above 19 ppm-hrs at sites meeting the current standard, and just a
28 small number of occurrences (limited to eight [less than 0.08% of values], all but one from a
29 period prior to 2011) of a W126 index above 17 ppm-hrs, with the highest just equaling 19 ppm-
30 hrs (Table 4-3; Appendix 4D, section 4D.3.2.1).

⁸⁰ On over 99.9 percent of occasions across all sites with valid design values at or below 70 ppb during the 2000 to 2020 period, the W126 metric (seasonal W126, averaged over three years) was at or below 17 ppm-hrs (Table 4-1). All but one of the eight occasions when it was above 17 ppm-hrs (the highest was 19 ppm-hrs) occurred in the Southwest region during a period before 2011. The eighth occasion occurred at a site in the West region when the 3-year average W126 index value was 18 ppm-hrs. On more than 97 percent of occasions in the full dataset with valid design values at or below 70 ppb, the 3-year average W126 index was at or below 13 ppm-hrs (Appendix 4D, section 4D.3.2).

1 Given the recognition of more significant public welfare implications of effects in
2 protected areas, such as Class I areas (as discussed in section 4.3.2 above), we give particular
3 attention to Class I areas (Appendix 4D, section 4D.3.2.4). In so doing, we consider the updated
4 air quality analysis presented in Appendix 4D for 65 Class I areas. The findings for these sites,
5 which are distributed across all nine NOAA climate regions in the contiguous U.S., as well as
6 Alaska and Hawaii, mirror all U.S. sites. Among the Class I area sites meeting the current
7 standard (i.e., having a design value at or below 70 ppb) in the most recent period of 2018 to
8 2020, there are none with a W126 index (averaged over design value period) above 17 ppm-hrs
9 (Table 4-3). The historical dataset includes just seven occurrences (all dating from the 2000-2010
10 period) of a Class I area site meeting the current standard and having a 3-year average W126
11 index above 17 ppm-hrs, and no such occurrences above 19 ppm-hrs (Table 4-1). Additionally,
12 across the full 21-year dataset for 56 Class I areas with monitors meeting the current standard
13 during at least one or as many as nineteen 3-year periods since 2000, there are no more than 15
14 occurrences of a single-year W126 index above 19 ppm-hrs, the majority occurring during the
15 earlier years of the period (Appendix 4D, section 4D.3.2.4, Tables 4D-14 and 4D-16). For
16 example, the highest values were equal to 23 ppm-hrs, all occurring before 2012 (Appendix 4D,
17 4D-16).

18 Across the complete dataset (2000-2020), the W126 index, averaged over a 3-year
19 period, at sites with design values above 70 ppb (i.e., that would not meet the current standard)
20 ranges up to approximately 60 ppm-hrs (Appendix 4D, Table 4D-17). Focusing on the most
21 recent period, among all sites across the U.S. that do not meet the current standard in the 2018 to
22 2020 period, more than a quarter have average W126 index values above 19 ppm-hrs and more
23 than a third exceed 17 ppm-hrs (Table 4-3).⁸¹ A similar situation exists for Class I area sites
24 (Table 4-3). Thus, as at the time of the 2015 decision, the available quantitative information
25 continues to indicate appreciable control of seasonal W126 index-based cumulative exposure at
26 all sites with air quality meeting the current standard.

27

⁸¹ As described above and in detail in Appendix 4D, W126 index values were rounded to the nearest unit ppm-hr for comparisons to a specific whole-number W126 level.

1 **Table 4-3. Distribution of 3-yr average seasonal W126 index for sites in Class I areas and**
 2 **across U.S. that meet the current standard and for those that do not.**

3-year periods	Number of Occurrences or Site-DVs ^A							
	In Class I Areas				Across All Monitoring Sites (urban and rural)			
	Total	W126 (ppm-hrs)			Total	W126 (ppm-hrs)		
>19		>17	≤17	>19		>17	≤17	
	At sites that meet the current standard (design value at or below 70 ppb)							
2018-2020	47	0	0	47	877	0	0	877
All from 2000 to 2020	589	0	7	582	10,039	0	8	10,031
	At sites that exceed the current standard (design value above 70 ppb)							
2018-2020	10	7	8	2	213	58	77	136
All from 2000 to 2020	391	174	219	172	11,142	2,424	3,317	7,825

^A The counts presented here are drawn from Appendix D, Tables 4D-2, 4D-4, 4D-5, 4D-6, 4D-9, 4D-10, and 4D-14 through 17.

3 In summary, as discussed in section 4.3.3 above, the evidence available leads us to
 4 similar conclusions regarding exposure levels associated with effects as in the 2015 review.
 5 Based largely on this evidence in combination with the use of RBL as a surrogate or proxy for all
 6 vegetation-related effects, the value of 17 ppm-hrs, as an average W126 index (over three years)
 7 was generally identified as a target level for protection in the 2015 decision (80 FR 65393;
 8 October 26, 2015). The available information continues to indicate that average cumulative
 9 seasonal exposure levels at virtually all sites and 3-year periods with air quality meeting the
 10 current standard fall at or below this level of 17 ppm-hrs. Additionally, at sites meeting the
 11 current standard, single-year W126 index values are less than or equal to 19 ppm-hrs well over
 12 99% of the time (Appendix 4D, section 4D.3.2.1). In Class I area sites that meet the current
 13 standard for the most recent 3-year period, the average W126 index is below 17 ppm-hrs
 14 (Appendix 4D, Table 4D-16). Further, across the full 21-year dataset, with the exception of
 15 seven values that occurred prior to 2011, Class I area W126 index values (averages for each 3-
 16 year period) were no higher than 17 ppm-hrs during periods that met the current standard. This
 17 contrasts with the occurrence of much higher seasonal W126 index values in sites when the
 18 current standard was not met. For example, out of the 10 Class I area sites with design values
 19 above 70 ppb during the most recent period, seven had a W126 index (based on 3-year average)
 20 above 19 ppm-hrs (ranging up to 47 ppm-hrs) and eight sites had a W126 index above 17 ppm-
 21 hrs (Table 4-3; Appendix 4D, Table 4D-17). This same pattern is exhibited at all sites in the full
 22 dataset, as shown in Table 4-3, including both urban and rural sites.

4.4.3 Limitations and Uncertainties

- **What are the important uncertainties associated with any exposure estimates and associated characterization of potential for public welfare effects?**

The analyses described above in sections 4.1 and 4.2 are based primarily on the hourly air monitoring dataset that is available at O₃ monitoring sites nationwide. While there are inherent limitations in any air monitoring network, the monitors for O₃ are distributed across the U.S., covering all NOAA regions and all states (e.g., Figure 4-6).

That distribution notwithstanding, there is uncertainty about whether areas that are not monitored would show the same patterns of exposure as areas with monitors. There are limitations in the distributions of the monitors, such that some geographical areas are more densely covered than others. For example, only about 40% of all Federal Class I Areas have or have had O₃ monitors within 15 km with valid design values, thus allowing inclusion in the Class I area analysis. Even so, the dataset includes sites in 27 states distributed across all nine NOAA climatic regions across the contiguous U.S, as well as Hawaii and Alaska. Some NOAA regions have far fewer numbers of Class I areas with monitors than others. For instance, the Central, Northeast, East North Central, and South regions all have three or fewer Class I areas in the dataset. However, these areas also have appreciably fewer Class I areas in general when compared to the Southwest, Southeast, West, and West North Central regions, which are more well represented in the dataset. The West and Southwest regions are identified as having the largest number of Class I areas, and they have approximately a third of those areas represented with monitors, which include locations where W126 index values are generally higher, thus playing a prominent role in the analysis. We also recognize a limitation that accompanies any analysis, i.e., that it is based on information available at this time. Thus, it may or may not reflect conditions far out into the future as air quality and patterns of O₃ concentrations in ambient air continue to change in response to changing circumstances, such as changes in precursor emissions to meet the current standard across the U.S. That said, we note that for the air quality analyses (e.g., involving W126 index) that were also conducted in the 2015 review, the findings are largely consistent.

In considering the estimates of exposure represented by the W126 index, we note a limitation in this index in its ability to distinguish among air quality conditions with differing prevalence of peak concentrations (e.g., hourly concentrations at or above 100 ppb). As indicated in the analyses in Appendix 4F, summarized above in section 4.1.1, two different locations or years may have appreciably different patterns of hourly concentrations but the same W126 index value. To the extent that these concentrations influence vegetation responses, this may contribute an uncertainty to applications of the tree seedling E-R functions (as recognized by Lefohn et al., 1997).

1 Further, we note the discussion in section 4.4.1 above of how changes in O₃ patterns in
2 the past have affected the relationship between W126 index and the averaging time and form of
3 the current standard, as represented by design values (section 4.4.1, and Appendix 4D, section
4 4D.3.2.3). This analysis finds a positive, linear relationship between trends in design values and
5 trends in the W126 index (both in terms of single-year W126 index and averages over 3-year
6 design value period), as was also the case for similar analyses conducted for the data available at
7 the time of the 2015 review (Wells, 2015). While this relationship varies across NOAA regions,
8 the regions showing the greatest potential for exceeding W126 index values of interest (e.g., with
9 3-year average values above 17 and/or 19 ppm-hrs) also showed the greatest improvement in the
10 W126 index per unit decrease in design value over the historical period assessed (Appendix 4D,
11 section 4D.3.2.3). Thus, the available data and this analysis appear to indicate that as design
12 values are reduced to meet the current standard in areas that presently do not, W126 values in
13 those areas would also be expected to decline (Appendix 4D, section 4D.4).

14 **4.5 KEY CONSIDERATIONS REGARDING THE CURRENT** 15 **SECONDARY STANDARD**

16 In considering what the available evidence and exposure/risk information indicate with
17 regard to the current secondary O₃ standard, the overarching question we address is:

- 18 • **Does the available scientific evidence and air quality and exposure analyses support**
19 **or call into question the adequacy of the protection afforded by the current**
20 **secondary O₃ standard?**

21 To assist us in interpreting the available scientific evidence and the results of recent
22 quantitative analyses to address this question, we have focused on a series of more specific
23 questions. In considering the scientific and technical information, we consider both the
24 information available at the time of the 2015 review and information newly available since then
25 which has been critically analyzed and characterized in the current ISA, the 2013 ISA and prior
26 AQCDs. In this context, an important consideration is whether the newly available information
27 alters the EPA's overall conclusions from the 2015 review regarding welfare effects associated
28 with photochemical oxidants, including O₃, in ambient air. We also consider the available
29 quantitative information regarding environmental exposures, characterized by the pertinent
30 metric, likely to occur in areas of the U.S. where the standard is met. Additionally, we consider
31 the significance of these exposures with regard to the potential for O₃-related vegetation effects,
32 their potential severity, and any associated public welfare implications.

1 **4.5.1 Evidence and Exposure/Risk-based Considerations**

2 In considering first the available evidence with regard to the overarching question posed
3 above regarding the protection provided by the current standard from welfare effects, we address
4 a series of more specific questions that focus on policy-relevant aspects of the evidence. These
5 questions relate to three main areas of consideration: (1) the available evidence on welfare
6 effects associated with exposure to photochemical oxidants, and particularly O₃ (section 4.5.1.1);
7 (2) the risk management framework or approach for reaching conclusions on the adequacy of
8 protection provided by the secondary standard (section 4.5.1.2); and (3) findings from the air
9 quality and exposure analyses pertaining to public welfare protection under the current standard
10 (section 4.5.1.3).

11 **4.5.1.1 Welfare Effects Evidence**

- 12 • **Is there newly available evidence that indicates the importance of photochemical**
13 **oxidants other than O₃ with regard to abundance in ambient air, and potential for**
14 **welfare effects?**

15 No newly available evidence has been identified regarding the importance of
16 photochemical oxidants other than O₃ with regard to abundance in ambient air, and potential for
17 welfare effects.⁸² As summarized in section 2.1 above, O₃ is one of a group of photochemical
18 oxidants formed by atmospheric photochemical reactions of hydrocarbons with nitrogen oxides
19 in the presence of sunlight, with O₃ being the only photochemical oxidant other than nitrogen
20 dioxide that is routinely monitored in ambient air (ISA, Appendix 1, section 1.1).⁸³ Data for
21 other photochemical oxidants are generally derived from a few special field studies, such that
22 national scale data for these other oxidants are scarce (ISA, Appendix 1, section 1.1; 2013 ISA,
23 sections 3.1 and 3.6). Moreover, few studies of the welfare effects of other photochemical
24 oxidants beyond O₃ have been identified by literature searches conducted for the 2013 ISA and
25 prior AQCDs (ISA; Appendix 1, section 1.1). As stated in the current ISA, “the primary
26 literature evaluating the health and ecological effects of photochemical oxidants includes ozone
27 almost exclusively as an indicator of photochemical oxidants” (ISA, section IS.1.1). Thus, as was
28 the case for previous reviews, the evidence base for welfare effects of photochemical oxidants
29 does not indicate an importance of any other photochemical oxidants. For these reasons,
30 discussion of photochemical oxidants in this document focuses on O₃.

⁸² Close agreement between past ozone measurements and the photochemical oxidant measurements upon which the early NAAQS (for photochemical oxidants including O₃) was based indicated the very minor contribution of other oxidant species in comparison to O₃ (U.S. DHEW, 1970).

⁸³ Consideration of welfare effects associated with nitrogen oxides in ambient air is addressed in the review of the secondary NAAQS for ecological effects of oxides of nitrogen, oxides of sulfur and particulate matter (U.S. EPA, 2018).

1 • **Does the available evidence alter prior conclusions regarding the nature of welfare**
2 **effects attributable to O₃ in ambient air?**

3 The current evidence documented in the 2020 ISA, including that newly available,
4 supports, sharpens, and expands somewhat on the conclusions reached in the 2015 review (ISA,
5 sections IS.1.3.2 and IS.5 and Appendices 8 and 9). A wealth of scientific evidence, spanning
6 more than six decades, demonstrates effects on vegetation and ecosystems of O₃ in ambient air
7 (ISA, section IS.6.2.1; 2013 ISA, 2006 AQCD, 1997 AQCD, 1986 AQCD; U.S. DHEW, 1970).
8 Accordingly, consistent with the evidence in the 2015 review, the available evidence describes
9 an array of O₃ effects on vegetation and related ecosystem effects. The evidence also describes
10 climate effects of tropospheric O₃, through a role in radiative forcing and subsequent effects on
11 temperature, precipitation, and related climate variables. Evidence newly available in the 2020
12 ISA strengthens previous conclusions, provides further mechanistic insights and augments
13 current understanding of varying effects of O₃ among species, communities, and ecosystems
14 (ISA, section IS.6.2.1). The current evidence, including a wealth of longstanding evidence,
15 supports conclusions reached in the 2015 review of causal relationships between O₃ and visible
16 foliar injury, reduced yield and quality of agricultural crops, reduced vegetation growth and plant
17 reproduction,⁸⁴ reduced productivity in terrestrial ecosystems, and alteration of belowground
18 biogeochemical cycles. The current evidence, including that previously available, also supports
19 conclusions reached in the 2015 review of likely causal relationships between O₃ and reduced
20 carbon sequestration in terrestrial systems, and alteration of terrestrial ecosystem water cycling
21 (ISA, section IS.I.3.2). Additionally, as in the 2015 review, the current ISA determines there to
22 be a causal relationship between tropospheric O₃ and radiative forcing and a likely causal
23 relationship between tropospheric O₃ and temperature, precipitation, and related climate
24 variables (ISA, section IS.1.3.3). Further, the current evidence has led to an updated conclusion
25 on the relationship of O₃ with alteration of terrestrial community composition to causal (ISA,
26 sections IS.I.3.2). Lastly, the current ISA concludes the current evidence sufficient to infer likely
27 causal relationships of O₃ with three additional categories of effects (ISA, sections IS.I.3.2).
28 While previous recognition of O₃ as a contributor to tree mortality in a number of field studies
29 was a factor in the 2013 conclusion regarding composition, it has been separately assessed in the
30 current ISA, with the conclusion that the evidence is sufficient to infer a likely causal
31 relationship with O₃. Additionally, evidence newly available since the last ISA on two additional
32 plant-related effects augments more limited previously available evidence related to insect
33 interactions with vegetation, contributing to additional conclusions that the body of evidence is

⁸⁴ As noted in section 4.3.1 above, the 2020 ISA includes a causality determination specific to reduced plant reproduction, while this category of effects was considered in combination with reduced plant growth in the 2015 review (ISA, Table IS.13).

1 sufficient to infer likely causal relationships between O₃ and alterations of plant-insect signaling
2 and insect herbivore growth and reproduction (ISA, Appendix 8, sections 8.6 and 8.7).⁸⁵

3 As in the 2015 review, the strongest evidence and the associated findings of causal or
4 likely causal relationships with O₃ in ambient air, and quantitative characterizations of
5 relationships between O₃ exposure and occurrence and magnitude of effects, are for vegetation-
6 related effects, and particularly those identified in the 2015 review. The evidence base for the
7 newly identified category of increased tree mortality includes previously available evidence
8 largely comprised of field observations from locations and periods of O₃ concentrations higher
9 than are common today and three more recently available publications assessing O₃ exposures
10 not expected under conditions meeting the current standard. Among the three more recent
11 publications, one assessed survival of aspen clones across an 11-year period under O₃ exposures
12 that included single-year seasonal W126 index values ranging above 30 ppm-hrs during the first
13 four years, and the other two were analyses based on field observations during periods when O₃
14 concentrations were such that they would not be expected to meet the current standard, as
15 summarized in section 4.3.1 above (ISA, Appendix 8, section 8.4.3).

16 The information available regarding the newly identified categories of plant-insect
17 signaling and insect herbivore growth and reproduction does not provide for a clear
18 understanding of the specific environmental effects that may occur in the natural environment
19 under specific exposure conditions (as discussed in sections 4.3.1, 4.3.3.2 and 4.3.4 above). For
20 example, while the evidence base for effects on herbivore growth and reproduction is expanded
21 since the 2013 ISA, “there is no clear trend in the directionality of response for most metrics,”
22 such that some show an increased effect and some show reductions (ISA, p. IS-64; section
23 IS.5.1.3 and section 8.6). More specifically “no consistent directionality of response is observed
24 across the literature, and uncertainties remain in regard to different plant consumption methods
25 across species and the exposure conditions associated with particular severities of effects” (ISA,
26 p. IS-91). Additionally, while the available evidence documents effects of O₃ on some plant
27 VPSCs (e.g., changing the floral scent composition and reducing dispersion), and indicates
28 reduced pollinator attraction, decreased plant host detection and altered plant-host preference in
29 some insect species in the presence of elevated O₃ concentrations, characterization of such
30 effects is still “an emerging area of research with information available on a relatively small
31 number of insect species and plant-insect associations,” and with gaps remaining in the
32 consequences of modification of signaling compounds by O₃ in natural environments (ISA, p.

⁸⁵ As in the 2015 review, the 2020 ISA again concludes that the evidence is inadequate to determine if a causal relationship exists between changes in tropospheric ozone concentrations and UV-B effects (ISA, Appendix 9, section 9.1.3.4; 2013 ISA, section 10.5.2).

1 IS-91 and section IS.6.2.1). Accordingly, we focus on other vegetation effects described above,
2 rather than these two newly identified categories.

3 With regard to tropospheric O₃ and effects on climate, we recognize the strength of the
4 ISA conclusion that tropospheric O₃ is a greenhouse gas at the global scale, with associated
5 effects on climate (ISA, section 9.1.3.3). Accordingly, as indicated by the ISA causal
6 determinations, O₃ abundance in the troposphere contributes to radiative forcing and likely also
7 to subsequent climate effects. There is appreciable uncertainty, however, associated with
8 understanding quantitative relationships involving regional O₃ concentrations near the earth's
9 surface and climate effects of tropospheric O₃ on a global scale. As recognized in the ISA (and
10 summarized in sections 4.3.3.3 and 4.3.4 above), there are limitations in our modeling tools and
11 associated uncertainties in interpretations related to capabilities for quantitatively estimating
12 effects of regional-scale lower tropospheric O₃ concentrations on climate. Thus, while additional
13 characterizations of tropospheric O₃ and climate have been completed since the 2015 review,
14 uncertainties and limitations in the evidence that were also recognized in the 2015 review
15 remain. As summarized in sections 4.3.3.3 and 4.3.4 above, these affect our ability to make a
16 quantitative characterization of the potential magnitude of climate response to changes in O₃
17 concentrations in ambient air, particularly at regional (vs global) scales, and thus our ability to
18 assess the impact of changes in ambient air O₃ concentrations in regions of the U.S. on global
19 radiative forcing or temperature, precipitation, and related climate variables. Consequently, the
20 evidence in this area is not informative to our consideration of the adequacy of public welfare
21 protection of the current standard.

- 22 • **To what extent does the available evidence provide E-R information (e.g.,**
23 **quantitative E-R relationships) for O₃-related effects that can inform judgments on**
24 **the likelihood of occurrence of such effects in areas with air quality that meets the**
25 **current standard? Does the available evidence provide new or altered such**
26 **information since the 2015 review?**

27 In considering what the available information indicates with regard to exposures
28 associated with welfare effects and particularly in the context of what is indicated for exposures
29 associated with air quality conditions that meet the current standard, we focus particularly on the
30 availability of quantitatively characterized E-R relationships for key effects. While the ISA
31 describes additional studies of welfare effects associated with O₃ exposures since the 2015
32 review, the established E-R functions for tree seedling growth and crop yield that have been
33 available in the last several reviews continue to be the most robust descriptions of E-R
34 relationships for welfare effects. These well-established E-R functions for seedling growth
35 reduction in 11 tree species and yield loss in 10 crop species are based on response information
36 across multiple levels of cumulative seasonal exposure (estimated from extensive records of

1 hourly O₃ concentrations across the exposure periods). Studies of some of the same species,
2 conducted since the E-R function derivation, provide supporting information for these functions
3 (ISA, Appendix 8, section 8.13.2; 2013 ISA, sections 9.6.3.1 and 9.6.3.2). The E-R functions
4 provide for estimation of growth-related effects for a range of cumulative seasonal exposures.

5 The newly available evidence does not include new studies that assessed reductions in
6 tree growth or crop yield responses across multiple O₃ exposures and for which sufficient data
7 are available for analyses of the shape of the E-R relationship across the range of cumulative
8 exposure levels (e.g., in terms of W126 index) relevant to conditions associated with the current
9 standard. For example, among the newly available studies are several that summarize previously
10 available studies or draw from them, such as for linear regression analyses.⁸⁶ However, as
11 discussed in section 4.3.3.2 above, these do not provide robust E-R functions or cumulative
12 seasonal exposure levels associated with important vegetation effects that define the associated
13 exposure circumstances in a consistent manner, limiting their usefulness for our purposes here
14 with regard to considering the potential for occurrence of welfare effects in air quality conditions
15 that meet the current standard. Thus, robust E-R functions are not available for growth or yield
16 effects on any additional tree species or crops.

17 Based on these established E-R functions for tree seedling growth reductions in 11
18 species, the tree seedling RBL for the median tree species is 5.3% for a W126 index of 17 ppm-
19 hrs, rising to 5.7% for 18 ppm-hrs, 6.0% for 19 ppm-hrs and 6.4% for 20 ppm-hrs. Below 17
20 ppm-hrs, the median estimates include 4.9% for 16 ppm-hrs, 4.5% for 15 ppm-hrs, 4.2% for 14
21 ppm-hrs and 3.8% for 13 ppm-hrs (Appendix 4A, Table 4A-5). These RBL estimates are
22 unchanged from what was indicated by the evidence in the 2015 review. As summarized in
23 section 4.1 above, the RBL estimates were used in the 2015 decision as a surrogate or proxy for
24 the broader array of vegetation-related effects.

25 With regard to visible foliar injury, as in the 2015 review, we lack established E-R
26 relationships that would quantitatively describe relationships between visible foliar injury
27 (occurrence and incidence, as well as injury severity) and O₃ exposure, as well as factors
28 influential in those relationships, such as soil moisture conditions. As discussed in section
29 4.3.3.2 above, the available evidence continues to include both experimental studies that

⁸⁶ For example, among the newly available publications cited in the ISA is a publication on tree and grassland species that compiles EC₁₀ values (estimated concentration at which 10% lower biomass [compared to zero O₃] is predicted) derived using linear regression of previously published data on plant growth response and O₃ concentration quantified as AOT40. The data were from studies of various experimental designs, that involved various durations ranging up from 21 days, and involving various concentrations no higher than 100 ppb as a daily maximum hourly concentration. More detailed analyses of consistent, comparable E-R information across a relevant range of seasonal exposure levels, accompanied by detailed records of O₃ concentrations, that would support derivation of robust E-R functions for purposes discussed here are not available (ISA, Appendix 8, section 8.10.1.2).

1 document foliar injury in specific plants in response to O₃ exposures, and quantitative analyses
2 of the relationship between environmental O₃ exposures and occurrence of foliar injury. The
3 analyses involving environmental conditions, while often using cumulative exposure metrics to
4 quantify O₃ exposures (e.g., the W126 and SUM06 indices), have additionally reported there to
5 also be a role for a metric that quantifies the frequency or incidence of “high” O₃ days, such as
6 N100 (2013 ISA, p. 9-10; Smith, 2012; Wang et al., 2012). However, such analyses have not
7 resulted in the establishment of specific air quality metrics and associated quantitative functions
8 for describing the influence of ambient air O₃ on incidence and severity of visible foliar injury.

9 Multiple studies have involved quantitative analysis of data collected as part of the USFS
10 biosite biomonitoring program (e.g., Smith, 2012). These analyses continue to indicate the
11 limitations in capabilities for predicting the exposure circumstances under which visible foliar
12 injury would be expected to occur, as well as the circumstances contributing to increased injury
13 severity (Smith, 2012; Wang et al., 2012). As noted in section 4.3.3.2 above, expanded
14 summaries of the dataset compiled in the 2015 review from several years of USFS biosite
15 records does not clearly and consistently describe the shape of a relationship between incidence
16 of foliar injury or severity (based on individual site scores) and W126 index estimates (as a sole
17 representative of exposure). Overall, however, the dataset indicates that the proportion of records
18 having different levels of severity score is generally highest in the group of records for sites with
19 the highest W126 index (e.g., greater than 25 ppm-hrs for the normal and dry soil moisture
20 categories). Thus, the available evidence indicates increased occurrence and severity at the
21 highest category of exposures in the dataset (above 25 ppm-hrs in terms of a W126 index), but
22 does not provide for identification of air quality conditions, in terms of O₃ concentrations
23 associated with the relatively lower environmental exposures most common in the USFS dataset
24 that would correspond to a specific magnitude of injury incidence or severity scores across
25 locations.

26 Thus, based on considering the available information for the array of O₃ welfare effects,
27 we again recognize the E-R relationships available in the 2015 review for purposes of
28 considering O₃ exposure levels associated with growth-related impacts to be the most robust E-R
29 information available. The available evidence for growth-related effects, including that newly
30 available, does not indicate the occurrence of growth-related responses attributable to cumulative
31 O₃ exposures lower than was established at the time of the 2015 review. With regard to visible
32 foliar injury, the available information continues to be limited with regard to estimating
33 occurrence and severity (e.g., as quantified by BI score) across a range of air quality conditions
34 quantified by W126 index, such that a clear shape for a relationship between these variables is
35 not evident with the available data. Thus, the available information provides for only limited and
36 somewhat qualitative conclusions related to potential occurrence and/or severity under different

1 air quality conditions. The quantitative information for other effects is still more limited, as
2 recognized in sections 4.3.3 and 4.3.4 above. Thus, the newly available evidence does not
3 appreciably address key limitations or uncertainties needed to expand capabilities for estimating
4 welfare impacts that might be expected as a result of differing patterns of O₃ concentrations in
5 the U.S.

6 • **Does the evidence continue to support a cumulative, seasonal exposure index, such as**
7 **the W126 function, as a biologically relevant and appropriate metric for assessment**
8 **of vegetation-related effects of O₃ in ambient air?**

9 As in the 2015 review, the available evidence continues to support a cumulative, seasonal
10 exposure index as a biologically relevant and appropriate metric for assessment of the evidence
11 of exposure/risk information for vegetation, most particularly for growth-related effects. The
12 most commonly used such metrics are the SUM06, AOT40 (or AOT60) and W126 indices (ISA,
13 section IS.3.2).⁸⁷ The evidence for growth-related effects continues to support important roles for
14 cumulative exposure and for weighting higher concentrations over lower concentrations. Thus,
15 among the various such indices considered in the literature, the cumulative, concentration-
16 weighted metric, defined by the W126 function, continues to be best supported for purposes of
17 relating O₃ air quality to growth-related effects.

18 We additionally note that while in its approach to emphasizing higher concentrations, the
19 W126 index assigns greater weights to higher hourly concentrations, it cannot, given its
20 definition as an index that sums three months of weighted hourly concentrations into one, single
21 value, always differentiate between air quality patterns with frequent high peak concentrations
22 and those without such concentrations.⁸⁸ While the metric describes the pattern of varying
23 growth response observed across the broad range of cumulative exposures examined in the tree
24 seedling E-R studies (see Appendix 4A), given the way it is calculated the W126 index can
25 conceal peak concentrations that can be of concern. More specifically, one season or location

⁸⁷ While the evidence includes some studies reporting O₃-reduced soybean yield and perennial plant biomass loss using AOT40 (as well as W126) as the exposure metric, no newly available analyses are available that compare AOT40 to W126 in terms of the strength of association with such responses. Nor are studies available that provide analyses of E-R relationships for AOT with reduced growth or RBL with such extensiveness as the analyses supporting the established E-R functions for W126 with RBL and RYL.

⁸⁸ This is illustrated by the following two hypothetical examples. In the first example, two air quality monitors have a similar pattern of generally lower average hourly concentrations but differ in the occurrence of higher concentrations (e.g., hourly concentrations at or above 100 ppb). The W126 index describing these two monitors would differ. In the second example, one monitor has appreciably more hourly concentrations above 100 ppb compared to a second monitor; but the second monitor has higher average hourly concentrations than the first. In the second example, the two monitors may have the same W126 index, even though the air quality patterns observed at those monitors are quite different, particularly with regard to the higher concentrations, which have been recognized to be important in eliciting responses (as noted above).

1 could have few, or even no, hourly concentrations above 100 ppb⁸⁹ and the second could have
2 many such concentrations; yet (due to greater prevalence of more mid-range concentrations, e.g.,
3 contributing to a generally higher average hourly concentration in the second) each of the two
4 seasons or locations could have the identical W126 index (e.g., equal to 25 or 15 or 10 ppm-hrs,
5 or some other value), as discussed in section 4.4.1 above.

6 Accordingly, in our consideration of the potential for vegetation-related effects to occur
7 under air quality conditions associated with the current standard, we continue to focus on the
8 W126 index as the appropriate metric, while also being aware of the importance of considering
9 the occurrence and frequency of particularly high concentrations. We also recognize that this
10 metric may not well describe the key circumstances of O₃ exposure for occurrences of other
11 effects, particularly, visible foliar injury. As discussed in section 4.3.3.2 above, the evidence
12 indicates an important role for peak concentrations (e.g., N100) in influencing the occurrence
13 and severity of visible foliar injury. Thus, while we continue to recognize the W126 index as an
14 appropriate and biologically relevant focus for assessing air quality conditions with regard to
15 potential effects on vegetation growth and related effects, we also recognize the need for
16 attention to the pattern and magnitude of peak concentrations.

17 **4.5.1.2 General Approach for Considering Public Welfare Protection**

18 The general approach and risk management framework applied in 2015 for making
19 judgements and reaching conclusions regarding the adequacy of public welfare protection
20 provided by the newly established secondary standard is summarized in section 4.1 above. In
21 light of the available evidence and air quality information, we discuss here key considerations in
22 judging public welfare protection provided by the O₃ secondary standard in the context of a
23 series of questions.

- 24 • **Does the newly available information continue to support the use of tree seedling**
25 **RBL as a proxy for the broad array of vegetation-related effects?**

26 As summarized in section 4.3 above, the available evidence is largely consistent with that
27 available in the 2015 review and does not call into question conceptual relationships between
28 plant growth impacts and the broader array of vegetation effects. Rather, the ISA describes (or
29 relies on) conceptual relationships in considering causality determinations for ecosystem-scale
30 effects such as altered terrestrial community composition and reduced productivity, as well as
31 reduced carbon sequestration, in terrestrial ecosystems (ISA, Appendix 8, sections 8.8 and 8.10).

⁸⁹ As noted in section 4.4 above, the value of 100 ppb is used here as it has been in some studies focused on O₃ effects on vegetation, simply as an indicator of elevated or peak hourly O₃ concentrations (e.g., Lefohn et al., 1997, Smith, 2012; Davis and Orendovici, 2006; Kohut, 2007). Values of 95 ppb and 110 ppb have also been considered in this way (2013 ISA, section 9.5.3.1).

1 Thus, the evidence continues to support the use of tree seedling RBL as a proxy for a broad array
2 of vegetation-related effects, most particularly those conceptually related to growth.

3 Beyond these relationships of plant-level effects and ecosystem-level effects,⁹⁰ RBL can
4 be appropriately described as a scientifically valid surrogate of a variety of welfare effects based
5 on consideration of ecosystem services and the potential for adverse impacts on public welfare,
6 as well as conceptual relationships between vegetation growth-related effects (including carbon
7 allocation) and ecosystem-scale effects. Beyond tree seedling growth (on which RBL is
8 specifically based), two other vegetation effect categories with extensive evidence bases are crop
9 yield and visible foliar injury, both types of effects, their evidence bases and key considerations
10 with regard to protection afforded by the current standard (which go beyond a RBL target for
11 tree seedlings) are separately addressed in section 4.5.1.3 below.

- 12 • **To what extent does the available information alter our understanding of an**
13 **appropriate magnitude of RBL, in its role as a surrogate or proxy, reasonably**
14 **expected to be of public welfare significance?**

15 The available information does not differ from that available in the 2015 review with
16 regard to a magnitude of RBL in the median species appropriately considered a reference for
17 judgments concerning potential vegetation-related impacts to the public welfare. Based on the
18 available information, a 6% RBL median estimate from the established species-specific E-R
19 functions continues to be appropriate for such a reference point. We note this in the context of
20 RBL’s role as a surrogate or proxy of a larger array of vegetation effects for which it was judged
21 that isolated rare instances of cumulative exposures that correspond to 6% (as the median of the
22 11 E-R functions) were not indicative of adverse effects to the public welfare (80 FR 65409,
23 October 26, 2015). The available evidence continues to indicate conceptual relationships
24 between reduced growth and the broader array of vegetation-related effects (as discussed above).
25 Quantitative representations of such relationships have been used to study potential impacts of
26 tree growth effects on such larger-scale effects as community composition and productivity with
27 the results indicating the array of complexities involved (e.g., ISA, Appendix 8, section 8.8.4).
28 Given their purpose in exploring complex ecological relationships and their responses to
29 environmental variables, as well as limitations of the information available for such work, these
30 analyses commonly utilize somewhat general representations. This work indicates how

⁹⁰ As summarized in the ISA, O₃ can mediate changes in plant carbon budgets (affecting carbon allocation to leaves, stems, roots and other biomass pools) contributing to growth impacts, and altering ecosystem properties such as productivity, carbon sequestration and biogeochemical cycling. In this way, O₃ mediated changes in carbon allocation can “scale up” to population, community and ecosystem-level effects including changes in soil biogeochemical cycling, increased tree mortality, shifts in community composition, changes in species interactions, declines in ecosystem productivity and carbon sequestration and alteration of ecosystem water cycling (ISA, section 8.1.3).

1 established the existence of such relationships is, while also identifying complexities inherent in
2 quantitative aspects of such relationships and interpretation of estimated responses. Thus, the
3 currently available evidence, as characterized in the 2020 ISA, is little changed from the 2015
4 review with regard to informing identification of an RBL reference point reflecting ecosystem-
5 scale effects with public welfare impacts elicited through such linkages.

- 6 • **What does the available information indicate with regard to the roles of seasonal**
7 **cumulative and peak exposures on O₃ vegetation effects, and accordingly regarding**
8 **the uses of cumulative and peak exposure metrics in assessing air quality conditions**
9 **that may pose risk of harm to vegetation?**

10 As summarized in section 4.3.3, longstanding conclusions regarding O₃ effects on
11 vegetation recognize both the cumulative effect of O₃ on plants and the importance of higher
12 concentrations in eliciting responses (1996 and 2006 AQCDs; 2013 and 2020 ISAs). As a result,
13 there has been substantial research into identification of an air quality exposure-related metric
14 that might address both aspects of potentially harmful O₃ conditions. As discussed in section
15 4.3.3.1.1 above, the metrics explored have included, among others, those that sum the portion of
16 a concentration above a reference point (e.g., AOT06), those that sum only those concentrations
17 above a reference point (e.g., SUM06), and also, the W126 index, a non-threshold approach
18 described as the sigmoidally weighted sum of hourly O₃ concentrations (2013 ISA, p. 9-101).
19 These indices (designed to address both cumulative effects and the importance of higher
20 concentrations) have been analyzed with regard to the extent to which they may describe the
21 growth response of plants (e.g., crops and tree seedlings) in studies assessing multiple exposure
22 levels and have been found to improve the explanatory power of E-R models over those based
23 only on mean (e.g., seasonal mean of 7-hour daily means) or peak exposure values (e.g., seasonal
24 maximum of maximum daily 7-hour and/or 1-hour averages) (2020 ISA, p. IS-79; 2013 ISA, p.
25 2-44; 2006 AQCD 1996 AQCD).

26 The explanatory strength of these cumulative, concentration-weighted approaches with
27 regard to plant response to O₃ indicates the influence of the various dimensions of exposure (e.g.,
28 concentration, duration, frequency) on plant response. With regard to the role of concentrations,
29 the 2020 and 2013 ISAs and past AQCDs generally recognize higher O₃ concentrations to be
30 associated with relatively greater risk of vegetation damage, in terms growth-related effects
31 (and/or visible foliar injury, which is discussed more specifically in response to a question
32 below) and emphasize the risk posed to vegetation from higher hourly average O₃
33 concentrations.⁹¹ With regard to duration and cumulative effects, analyses of the controlled

⁹¹ For example, as stated in the 2020 and 2013 ISAs, “[h]igher concentrations appear to be more important than lower concentrations in eliciting a response” [ISA, p. 8-180]; “higher hourly concentrations have greater effects

1 exposure datasets also supported conclusions in the 1996 and 2006 AQCDs (retained in more
2 recent ISAs) that a model focused only on a peak-concentration based metric (found to be an
3 improvement over earlier use of a long-term average to summarize exposure), without
4 consideration of duration was less descriptive of response (e.g., 1996 AQCD, Volume II, section
5 5.5.1.1). Accordingly, metrics that cumulated concentrations, e.g., through summing, as is the
6 case for those identified above, were developed, with preference to those that emphasized higher
7 concentrations (1996 and 2006 AQCDs; 2020 ISA, IS 5.1.9).

8 As recognized across several past reviews, the strength of the cumulative, concentration-
9 weighted approaches, including the continuously weighted W126 index function, is in describing
10 variation in response documented in controlled exposure studies or crops and tree seedlings for
11 which extensive hourly O₃ datasets are available. We note that in these exposures studies, the
12 higher cumulative exposure levels (e.g., W126 index levels) were generally accompanied by an
13 appreciable prevalence of high concentrations (e.g., Appendix 4A, Table 4A-6; Lefohn et al
14 1997; Lefohn and Foley, 1992). While these were part of the patterns of O₃ concentrations to
15 which the plants were exposed, another exposure circumstance may have the same W126 index,
16 yet with a different pattern of peak concentrations that may contribute to differences in risk of
17 vegetation effects. In an example highlighted in the 2006 AQCD and 2013 ISA, a study by Yun
18 and Lawrence (1999) used exposure regimes constructed from 10 U.S. cities to demonstrate that
19 in regimes with similar values of cumulative, concentration-weighted metrics, differences in the
20 magnitude and occurrence of peak concentrations were influential with regard to injury in tree
21 seedlings (2006 AQCD, p. AX9-176; 2013 ISA, section 9.5.3.1; Yun and Lawrence, 1999).⁹²
22 Given this, we recognize that the seasonal cumulative metrics may not always differentiate
23 between air quality patterns that include particularly high peak concentrations and those without
24 or with relatively fewer such concentrations.

25 For example, while the W126 index preferentially weights higher hourly concentrations,
26 given its definition as an index that sums three months of weighted hourly concentrations into a
27 single value, it can estimate the same value for very different incidence of elevated O₃
28 concentrations. As described in section 4.5.1.1, at two sites with the same W126 index value, the
29 air quality patterns may differ such that one site may have appreciably more hourly

on vegetation than lower concentrations” [2013 ISA, p. 91-4] “studies published since the 2006 O₃ AQCD do not
change earlier conclusions, including the importance of peak concentrations, ... in altering plant growth and
yield” [2013 ISA, p. 9-117]).

⁹² The 2013 ISA, in examining trends (1970s through 1990s) in an areas of the San Bernardino Mountains in
California, noted the reductions in ponderosa pine growth impacts occurring with reductions in SUM06,
maximum peak concentration and hourly concentrations over 95 ppb. In observing that there had been little
change in mid-range O₃ concentrations over the same period, the 2013 ISA noted the lesser role indicated for the
mid-range concentration ranges compared to the higher values (2013 ISA, p. 9-106).

1 concentrations at or above 100 ppb compared to the other site. This is also supported by the
2 analyses of available air quality data summarized in section 4.4.1 (e.g., Appendix 4F, Figure 4F-
3 10). Focusing on the data for the most recent five years (2016 through 2020), the distribution of
4 N100 or D100 values at monitoring sites meeting different W126 index values also shows this
5 variability, which contrasts with the much lesser variability in N100 and D100 values for sites
6 meeting the current standard (see Figure 4-12, W126 index bins at/below 19 ppm-hrs compared
7 to design value bins for 70 ppb or lower). It can be seen that (1) there is little difference in D100
8 at sites with W126 index ranging from 8 to 19 ppm-hrs (single-year or 3-year average index);
9 and (2) the form and averaging time of the existing standard is much more effective than the
10 W126 index in limiting the number of hours with O₃ concentrations at or above 100 ppb (N100)
11 and in limiting the number of days with any such hours.⁹³

12 Given the considerations raised here, we recognize that focusing solely on W126 index
13 for considering the public welfare protection provided by the current standard would not be
14 considering all the relevant scientific information. Further, we note that such a sole focus, given
15 the damaging potential for repeated elevated hourly concentrations (e.g., at or above 100 ppb), as
16 discussed in sections 4.3.3 and 4.5.1.1 above (ISA, p. 8-180; 2013 ISA, section 9.5.3.1)⁹⁴, may
17 not give adequate attention to ensuring protection against “unusually damaging years.” As a
18 result, we find that focusing solely on the W126 index may not ensure protection is provided
19 from potentially damaging air quality, such as that associated with exposure patterns marked by
20 repeated occurrences of elevated concentrations. Thus, we conclude it is important to consider
21 both cumulative, concentration-weighted and peak exposure metrics in assessing air quality with
22 regard to the potential for specific exposure conditions that might be harmful to vegetation.”⁹⁵

⁹³ As one example contained in Table 4-1 above, across all sites that met the current standard during the recent period (2018-2020), few sites had more than 5 hours at or above 100 ppb in a year (0.6% in the highest year, Appendix 4F, Table 4F-2). Among the sites with any such hours, all had fewer than five days in any one year with any such concentrations (Table 4-1, Appendix 4F, Figure 4F-5). In comparison, across all sites with an annual W126 index below 15 ppm-hrs, 2% of them had more than 5 hours with a concentration at or above 100 ppb, and this included sites with as many as eight days with such a concentration (Table 4-1, Appendix 4F, Figure 4F-11). We note that we are not intending to ascribe specific significance to five days with an hour at or above 100 ppb or ten such hours, *per se*. Rather, these are used simply as reference points to facilitate comparison and to illustrate the point that such high concentrations, which based on toxicological principles, pose greater risk to biota than lower concentrations, are not necessarily limited at sites meeting particular W126 index values.

⁹⁴ The section of the 2013 ISA titled “Role of Concentration,” summarizes the experimental evidence base on which the significant role of peak O₃ concentrations was established (2013 ISA, section 9.5.3.1).

⁹⁵ With regard to air quality occurring under the current standard, we note analyses presented in section 4.4 above that show the current standard to provide control of both cumulative exposures and of peak concentrations indicating the potential to address both aspects of potentially harmful O₃ conditions noted here.

- 1 • **What does the available information indicate regarding the use of W126 index in a**
2 **single year or averaged over three years in considering cumulative seasonal exposure**
3 **protection objectives for the secondary standard?**

4 In setting the current standard in 2015, as described in section 4.1 above, the decision
5 focused on control of seasonal cumulative exposures in terms of a 3-year average W126 index
6 based on consideration of several factors.⁹⁶ We again consider here the extent to which the
7 available evidence supports the 3-year average W126 index as a reasonable metric for assessing
8 the level of protection provided by the current standard from cumulative seasonal exposures
9 related to RBL, or whether an alternate approach is more appropriate for use with the E-R
10 functions.

11 We first consider the evidence and information underlying the E-R functions and the
12 extent to which they can be said to better describe or predict growth reductions specific to single
13 season exposures, as compared to growth reductions generally reflecting an average seasonal
14 exposure. With regard to the established tree seedling E-R functions themselves, we note there
15 are aspects of the datasets and methodology on which the E-R functions are based which provide
16 support for a multiyear (e.g. 3-year) average approach. As summarized in section 4.3.4 above,
17 the E-R functions were derived from studies of durations that varied from shorter than 92 days to
18 as many as 140 days in a single year, and up to 555 days distributed across multiple years or
19 growing seasons, with the results normalized to the duration of a single 92-day seasonal period
20 (Appendix 4A, pp. 4A-31 to 4A- 32). Inherent in this approach is an assumption that the growth
21 impacts relate generally to the cumulative O₃ exposure across the full time period (which may
22 include multiple growing seasons), i.e., with little additional influence related to any seasonal or
23 year to year differences in the exposures. Consequently, given this step in their derivation
24 approach, the E-R functions cannot provide precise estimates of response from a single year's
25 seasonal exposure (e.g., *vs* averages over a period longer than 92 days or one that spans multiple
26 growing seasons). Thus, the use of a multiyear (e.g. 3-year) average in assessing RBL using the
27 established tree seedling E-R functions is reasonably described as compatible with the
28 normalization step taken to derive functions for a seasonal 90-day period from the underlying
29 data with its varying exposure durations.

⁹⁶ These factors include consideration of the strengths and limitations of the evidence and of the information on which to base judgments regarding adversity of effects on the public welfare (80 FR 65390, October 26, 2015). Also recognized was year-to-year variability, not just in O₃ concentrations, but also in environmental factors, including rainfall and other meteorological factors, that influence the occurrence and magnitude of O₃-related effects in any year (e.g., through changes in soil moisture), contributing uncertainties to projections of the potential for harm to public welfare based on a single year, particularly at the exposure levels of interest (80 FR 65404, October 26, 2015).

1 We also take note of aspects of the evidence that reflect variability in organism response
2 under different experimental conditions and the extent to which this variability is represented in
3 the available data, which might indicate an appropriateness of assessing environmental
4 conditions using a mean across seasons in recognition of the existence of such year-to-year
5 variability in conditions and responses. For example, among the species for which there are more
6 than two or three experimental datasets comprising the support for the species' E-R function (14
7 experimental datasets for aspen [seven for which the E-R function for wild aspen has been
8 derived and seven supporting a function for aspen clones] and 11 for ponderosa pine) illustrate
9 appreciable variability in response across experiments (Appendix 4A, Figure 4A-10).
10 Contributions to this variability may come from several factors, including variability in seasonal
11 response related to variability in non-O₃ related environmental influences on growth, such as
12 rainfall, temperature and other meteorological variables, as well as biological variability across
13 individual seedlings. An additional variability could also be due to influential aspects of the O₃
14 air quality on plant growth that are not completely captured by the W126 index, e.g., different
15 patterns of hourly concentrations that yield the same W126 index (see section 4.4.1 and below).
16 Such variability in the data underlying these E-R functions may further support a multiyear (e.g.
17 3-year) average approach.

18 An additional aspect of considering the evidence and information, is how well the data
19 underlying the E-R functions represent and reflect conditions that are currently being
20 experienced in the U.S., and most importantly, conditions that reflect current air quality patterns
21 when meeting the current standard. On a related note, it is also important to understand the extent
22 to which E-R predictions are extrapolated beyond the tested exposure conditions. As noted in
23 section 4.3.4 above, the O₃ concentrations and cumulative exposures for the experimental
24 datasets from which the tree seedling E-R functions were derived include conditions that do not
25 occur in ambient air at sites the meet the current standard (section 4.4; Appendix 4A, Table 4A-
26 6; section 4.4). A similar issue was discussed in a previously available publication that observed
27 appreciable differences between the prevalence of hourly concentrations at or above 100 ppb in
28 exposures on which the E-R functions are based and those common in ambient air at that time, a
29 difference which is in many ways only increased with today's air quality (Lefohn et al., 1997).⁹⁷

⁹⁷ For example, many of the experimental exposure of elevated O₃ on which the established E-R functions for the 11 tree seedling species are based, had hundreds of hours of O₃ concentrations above 100 ppb, far more than are common in (unadjusted) ambient air, including in areas that meet the current standard (Lefohn et al., 1997, Appendix 2A, section 2A.2, Appendix 4 F). To illustrate, in the most recent 2018-2020 design value period, the mean number of observations per site at or above 100 ppb was well below one. In contrast, across most of the O₃ treatments in the experiments comprising the E-R function database, well below half had an N100 value less than 20 hours through the exposure period (Appendix 4A, Table 4A-6). Similarly, the experimental exposures in studies supporting some of the established E-R functions for 10 crop species also include many hours with O₃ concentrations at or above 100 ppb (Lefohn and Foley, 1992)

1 This issue is also discussed in section 4.3.4 above, where it is noted that in the E-R tree seedling
2 datasets, the O₃ treatments for W126 index levels observed in areas that meet the current
3 standard had N100 counts ranging up above 40. And for many of the treatments, N100 values
4 range up to several hundred (Appendix 4A, Table 4A-6). We find it reasonable to interpret this
5 information, and its contribution to uncertainty in the application of the underlying E-R
6 functions, as arguing for a less precise interpretation, such as an average across multiple seasons.

7 In further considering the evidence and information and its support for use of a single or
8 multiple year W126 index, the concept of cumulative multiyear exposures and associated
9 impacts should be considered. In particular, we ask the question of whether applying the E-R
10 functions to a W126 index averaged over multiple years would over- or under-estimate
11 cumulative exposure response, whereas use of a single seasonal exposure metric would not. The
12 evidence relevant to this question, e.g., that allows for specific evaluation of the predictability of
13 growth impacts from single-year versus multiple-year average exposure estimates, is limited.
14 Multi-year studies reporting results for each year of the study are the most informative to the
15 question of plant annual and cumulative responses to individual years (high and low) over
16 multiple-year periods. However, as summarized in section 4.3.4 above, the evidence is quite
17 limited with regard to studies of O₃ effects that report seasonal observations across multi-year
18 periods and that also include detailed hourly O₃ concentration records (to allow for derivation of
19 cumulative exposure index values). One such study, which tracked exposures across six years, is
20 available for aspen (King et al., 2005; 2013 ISA, section 9.6.3.2; ISA, Appendix 8, section
21 8.13.2). This study is presented in the 2013 and 2020 ISAs in an evaluation of predicted growth
22 impacts compared to observations from the multiple years of the study.

23 For this evaluation, the ISAs considered the 6-year experimental dataset of O₃ exposures
24 and aspen growth effects with regard to correspondence of E-R function predictions with study
25 observations (2020 ISA, Appendix 8, section 8.13.2 and Figure 8-17; 2013 ISA, section 9.6.3.2,
26 Table 9-15, Figure 9-20). The analysis in the 2013 ISA compared observed reductions in growth
27 for each of the six years to those predicted by applying the established E-R function for Aspen to
28 cumulative multi-year average W126 index values (2013 ISA, section 9.6.3.2).^{98 99} The
29 evaluation in the 2020 ISA applied the E-R functions to the single-year W126 index for each
30 year rather than the cumulative multi-year W126 (2020 ISA, Appendix 8, Figure 8-17), with this

⁹⁸ Although not emphasized or explained in detail in the 2013 ISA, the W126 index estimates used to generate the predicted growth response were cumulative averages. For example, the growth impact estimate for year 1 used the W126 index for year 1; the estimate for year 2 used the average of W126 index in year 1 and W126 index in year 2; the estimate for year 3 used the average of W126 index in years 1, 2 and 3; and so on.

⁹⁹ One finding of this evaluation was that “the function based on one year of growth was shown to be applicable to subsequent years” (2013 ISA, p. 9-135).

1 approach indicating a somewhat less tight fit to the experimental observations (2020 ISA,
2 Appendix 8, p. 8-192),¹⁰⁰ Both ISAs reach similar conclusions regarding general support for the
3 E-R functions across a multiyear study of trees in naturalistic settings (ISA, Appendix 8, section
4 8.13.3 and p. 8-192; 2013 ISA, p. 9-135).¹⁰¹ We additionally note that an illustrative
5 mathematical exercise that explored estimates of above ground biomass of an aspen stand when a
6 multi-year O₃ exposure was quantified in terms of a single year varying W126 index or as a
7 repeated yearly exposure equal to the associated 3-year average. These analyses suggest that the
8 two approaches may yield generally similar total biomass estimates after multiple years'
9 exposure (Appendix 4A, section 4A.3).

10 Thus, while the E-R functions are based on strong evidence of cumulative seasonal O₃
11 exposure reducing tree growth, and while they provide for quantitative characterization of the
12 extent of such effects across cumulative seasonal O₃ exposure levels of appreciable magnitude,
13 there is uncertainty associated with the resulting RBL predictions that might be described as an
14 imprecision or inexactitude. Further, as summarized above, the evidence does not indicate
15 single-year seasonal exposure in combination with the established E-R functions to be a better
16 predictor of RBL than a seasonal exposure based on a multi-year average. Accordingly, it is
17 reasonable to conclude that the evidence provides support for use of a 3-year average in
18 assessing the level of protection provided by the current standard from cumulative seasonal
19 exposures related to RBL of concern based on the established E-R functions.¹⁰² The 3-year
20 average metric also appears to be reasonable for use in the context of the use of RBL as a proxy
21 to represent an array of vegetation-related effects. Accordingly, upon consideration of all of the
22 factors raised above, we find the use of a multiyear average, and more specifically a 3-year
23 average, W126 index in assessing protection for RBL based on the established tree seedling E-R
24 functions to be reasonable. We also note, as discussed in response to the prior question, the
25 importance of also considering an additional aspect of O₃ air quality, specifically the occurrence

¹⁰⁰ Based on information drawn from Figure 8-17 in the 2020 ISA, the correlation metric (r^2) for the percent difference (estimated vs observed biomass) and year of growth can be estimated to be approximately 0.7, while using values reported in Table 9-15 of the 2013 ISA (which are plotted in Figure 9-20), the r^2 for predicted O₃ impact *versus* observed impact is 0.99 and for the percent difference *versus* year is approximately 0.85.

¹⁰¹ For the 2013 ISA, the conclusions reached were that the agreement between the set of predictions and the Aspen FACE observations were “very close” (2013 ISA, p. 9-135). The results indicate that when considering O₃ impacts across multiple years, a multi-year average index yields predictions close to observed measurements (2013 ISA, section 9.6.3.2 and Figure 9-20; Appendix 4A, section 4.A.3). For the 2020 ISA, the conclusion reached was that results from the aspen study were “exceptionally close” to predictions from the E-R model (ISA, p. 8-192)

¹⁰² Three years (versus two or four years) was selected based on its compatibility with the multiyear duration often used in forms for NAAQS to account for year-to-year variability in air quality.

1 of elevated hourly concentrations that influence vegetation exposures of potential concern, in
2 reaching conclusions about the adequacy of the current standard.

3 • **What does the available information indicate for considering potential public welfare**
4 **protection from O₃-related visible foliar injury?**

5 In establishing the current secondary standard in 2015 and its underlying public welfare
6 protection objectives, as summarized in section 4.1, above, the Administrator focused primarily
7 on RBL in tree seedlings as a proxy or surrogate for the full array of vegetation related effects of
8 O₃ in ambient air, from sensitive species to broader ecosystem-level effects. At that time, the
9 Administrator also concluded the information regarding visible foliar injury to also provide
10 support for strengthening the standard at that time, taking note of the available analyses of USFS
11 biosite data (80 FR 65407-65408, October 26, 2015). She also concluded, however, that, due to
12 associated uncertainties and complexities, the evidence was not conducive to use for identifying
13 a quantitative public welfare protection objective focused specifically on visible foliar injury. In
14 reaching this conclusion, she recognized significant challenges in judging the specific extent and
15 severity at which such effects should be considered adverse to public welfare, in light of the
16 variability in the occurrence of visible foliar injury and the lack of clear quantitative relationships
17 for prediction of visible foliar injury severity and incidence or extent under varying air quality
18 and environmental conditions, as well as the lack of established criteria or objectives that might
19 inform consideration of potential public welfare impacts related to this vegetation effect (80 FR
20 65407, October 26, 2015).

21 As an initial matter, we note that, as recognized in the 2015 review, some level of visible
22 foliar injury can impact public welfare and thus might reasonably be judged adverse to public
23 welfare.¹⁰³ As summarized in section 4.3.2 above, depending on its spatial extent and severity,
24 there are many locations in which visible foliar injury can adversely affect the public welfare.
25 For example, significant, readily perceivable (or obvious) and widespread injury in national
26 parks and wilderness areas can adversely impact the perceived scenic beauty of these areas,
27 impacting the aesthetic experience for both outdoor enthusiasts and the occasional park visitor.¹⁰⁴

¹⁰³ As stated in the *Federal Register* notice for the 2015 decision: “[d]epending on the extent and severity, O₃-induced visible foliar injury might be expected to have the potential to impact the public welfare in scenic and/or recreational areas during the growing season, particularly in areas with special protection, such as Class I areas. (80 FR 65379, October 26, 2015); “[t]he Administrator also recognizes the potential for this effect to affect the public welfare in the context of affecting values pertaining to natural forests, particularly those afforded special government protection (80 FR 65407, October 26, 2015). The CASAC in the 2015 review also stated that visible foliar injury “can impact public welfare” (Frey, 2014, p. 10).

¹⁰⁴ In the discussion of the need for revision of the 1997 secondary standard, the 2008 decision noted that “[i]n considering what constitutes a vegetation effect that is adverse from a public welfare perspective, ... the Administrator has taken note of a number of actions taken by Congress to establish public lands that are set aside

1 Thus, as aesthetic value and outdoor recreation depend, at least in part, on the perceived scenic
2 beauty of the environment, judgments related to the extent of public welfare impacts of visible
3 foliar injury depend on the severity and extent of the injury, as well as the location where the
4 effects occur and the associated intended use. Beyond the limitations associated with the
5 evidence for descriptive quantitative relationships for O₃ concentrations and visible foliar injury
6 (as summarized in sections 4.3.3.2 and 4.3.4 above), there is little information clearly relating
7 differing severity and prevalence of injury to conditions in natural areas that would reasonably be
8 concluded to impact public use and enjoyment in a way that might suggest adversity to the public
9 welfare. The available information does not yet address or describe the relationships expected to
10 exist between some level of severity and/or extent of location affected and scenic or aesthetic
11 values (e.g., reflective of visitor enjoyment and likelihood of frequenting such areas). However,
12 while minor spotting on a few leaves of a plant may easily be concluded to be of little public
13 welfare significance, it might reasonably be expected that in cases of widespread and relatively
14 more severe injury during the growing season (particularly when sustained across multiple years
15 and accompanied by obvious impacts on the plant canopy), O₃-induced visible foliar injury could
16 adversely impact the public welfare in scenic and/or recreational areas, particularly in parks and
17 other areas with special protection, such as Class I areas.

18 In the face of the paucity of established approaches that might be informative to the
19 Administrator in judging severity and extent of visible foliar injury in a natural area that may be
20 appropriate to consider of public welfare significance, we take note of the USFS scheme,
21 summarized in section 4.3.2 above, for categorizing areas based on BI scores (e.g., Smith, 2012).
22 In this scheme, BI scores may be described with regard to one of several categories ranging from
23 little or no foliar injury to severe injury (e.g., Smith et al., 2003; Campbell et al., 2007; Smith et
24 al., 2007; Smith, 2012). However, the available information does not yet address or describe the
25 relationships expected to exist between some level of severity of foliar injury (e.g., little or
26 severe) and/or a spatial extent affected and scenic or aesthetic values. This gap impedes
27 consideration of the public welfare implications of different injury severities, and accordingly,
28 judgments on the potential for public welfare significance.

29 With regard to the USFS BI program, we further note that authors of studies presenting
30 USFS biomonitoring program data have suggested what might be “assumptions of risk” (e.g., for
31 the forest resource) related to scores in these categories, e.g., as described in section 4.3.2 above.

for specific uses that are intended to provide benefits to the public welfare, including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment of future generations” (73 FR 16496, March 27, 2008). This passage of the *Federal Register* notice announcing the 2008 decision clarified that “[s]uch public lands that are protected areas of national interest include national parks and forests, wildlife refuges, and wilderness areas” (73 FR 16496, March 27, 2008).

1 One suggestion has been that maps of localized moderate to high-risk areas may be used to
2 identify areas (for scores of 15 or higher) where more detailed evaluations are warranted (Smith
3 et al., 2012). While these are not explicitly related to consideration of the public values described
4 above (e.g., with regard to public aesthetic or recreational value), the description of the BI score
5 categories as well as these corresponding judgments related to risk for the forest resource may
6 both be informative for the Administrator’s purposes. For example, it might be reasonable to
7 conclude that a small discoloring on a single leaf of a plant that might yield a quite low, nonzero
8 BI score in the USFS system is not adverse to the public welfare. On the other hand, BI scores
9 corresponding to a high risk to the resource may reasonably be concluded to indicate the need for
10 attention and, perhaps a public welfare adversity potential. Thus, while the available evidence
11 does not include characterization of USFS biosite scores with regard to public perception and
12 potential impacts on public enjoyment, we find that they may be useful for the Administrator’s
13 purposes in considering the potential public welfare significance of different severities and
14 extents of visible foliar injury, as scored by BI. That notwithstanding, limitations remain in our
15 tools for characterizing the air quality conditions at sites that elicit scores of a particular severity
16 level, thus continuing to challenge our ability to precisely identify conditions that might provide
17 particular levels of public welfare protection for this effect.

18 In considering the available information regarding a relationship between W126 index
19 and the severity of visible foliar injury, we consider the presentation of USFS biosite data in
20 Appendix 4C, summarized in section 4.3.3.2.2 above. While recognizing limitations in the
21 dataset¹⁰⁵ and considering the records for the normal or dry soil moisture categories, for which
22 there is somewhat better representation of W126 index levels above 13 ppm-hrs,¹⁰⁶ we note the
23 lack of a clear trend in the percentage of USFS records recording visible foliar injury (of any
24 severity level) W126 index estimates below 17 ppm-hrs. Focusing on the magnitude of BI score,
25 we note that among records in the normal soil category, BI scores are noticeably increased in the
26 highest W126 index bin (above 25 ppm-hrs) compared to the others. The percentages of records
27 in the greater than 25 ppm-hrs bin that have BI scores above 15 (“moderate” and “severe” injury)
28 and above 5 (“light,” “moderate” and “severe” injury) are more than three times greater than
29 percentages for these score levels in any of the lower W126 bins. Additionally, the average BI of
30 7.9 in the greater-than-25-ppm-hrs bin is more than three times the average BI for the next

¹⁰⁵ For example, the majority of these data are records with W126 index estimates at or below 9 ppm-hrs, and fewer than 10% of the records have W126 estimates above 15 ppm-hrs. Additionally, the BI scores are quite variable across the full dataset, with even the bin for the lowest W126 index estimates (below 7 ppm-hrs) including BI scores well above 15 (Appendix 4C, section 4C.4.2).

¹⁰⁶ In the case of records in the wet soil moisture category, nearly 90% of the records are for W126 estimates at or below 9 ppm-hrs, limiting interpretations for higher W126 bins (Appendix 4C, Table 4C.4 and section 4C.6).

1 highest W126 index bin. The average BI in the next two lower W126 bins (which vary inversely
2 with W126 index) are just slightly higher than average BIs for the rest of the bins, and the
3 average BI for all bins at or below 25 ppm-hrs are well below 5. Among records in the dry soil
4 moisture category, the two highest W126 bins (which together include the W126 index estimates
5 above 19 ppm-hrs) exhibit percentages of records with BI above 15 or above 5 that are
6 appreciably greater than that for the lower W126 bins. With regard to average scores across all
7 dry soil moisture records, average BI for all W126 index bins is below 5, although the three
8 highest W126 index bins (above 17 ppm-hrs) are markedly greater than the lower bins (e.g.,
9 average BIs greater than *versus* less than 1).

10 Thus, the strongest conclusions that can be reached from the USFS dataset described in
11 Appendix 4C are that the incidence of sites with more severe injury (e.g., BI score above 15 or 5)
12 is also lower at sites with W126 index values below 25 ppm-hrs than at sites with higher W126
13 index values and that clear trends in such incidence related to increasing W126 index levels are
14 not evident across the bins for lower W126 index estimates (all of which are below 5%). As
15 discussed in section 4.3.3.2 above, variability in the data across sites, and uncertainty, with
16 regard to the role of peak O₃ concentrations as an influence on occurrence of visible foliar injury
17 separate from cumulative W126 index, lead to the conclusion that the available information does
18 not support precise conclusions as to the severity and extent of such injury associated with the
19 lower values of W126 index most common at USFS sites during the time of the dataset (2006-
20 2010). Notwithstanding this, records categorized as normal soil moisture indicate there to be an
21 appreciable difference in severity of injury between records with W126 index estimates above 25
22 ppm-hrs and those with estimates at or below 25 ppm-hrs (e.g., Appendix 4C, Figures 4C-5 and
23 4C-6 and Table 4C-5). The records categorized as dry soil moisture do not indicate such a clear
24 pattern. The records categorized as wet soil moisture are too limited (and variable) for W126
25 index estimates above 13 ppm-hrs to support a conclusion (Appendix 4C). Thus, we conclude,
26 based primarily on the BI scores records categorized as having normal soil moisture, that under
27 conditions that maintain W126 index values below 25 ppm-hrs a reduced severity (average BI
28 score below 5) and incidence of visible foliar injury, as quantified by biosite index scores, would
29 be expected. The observation of a lack of clear relationship between levels of a cumulative
30 seasonal index and BI scores until reaching a higher value is conceptually similar to findings of
31 the study by Campbell et al. (2007), identified in the 2013 ISA that focused on visible foliar
32 injury in west coast states. This study observed that both percentage of USFS biosites with injury
33 and the average BI were higher for sites with average cumulative O₃ concentrations above 25
34 ppm-hrs in terms of SUM06 as compared to groups of sites with lower average cumulative
35 exposure levels, with little difference apparent between the two lower exposure groups (80 FR

1 65395, October 26, 2015; Smith and Murphy, 2015; Campbell et al., 2007, Figures 27 and 28
2 and p. 30).¹⁰⁷

3 Such findings of variability in scores at lower values of a cumulative seasonal index and
4 a lack of clear relationship with exposure may relate to patterns of peak concentrations at sites
5 with similar cumulative seasonal index values. As discussed in section 4.3.3.2 above, several
6 studies of the USFS data have concluded that inclusion of a metric for quantifying peak
7 concentrations, in combination with one for cumulative seasonal exposures, may yield a more
8 predictive description of the relationship between O₃ air quality and the occurrence of visible
9 foliar injury. Similarly, a county-scale analysis of USFS biosite data in the 2007 Staff Paper
10 (from earlier years than those analyzed in the 2015 review) indicated a somewhat smaller
11 incidence of biosites with nonzero BI scores in counties with air quality meeting a fourth-high
12 metric of 74 ppb as compared to larger groups that also included sites with air quality meeting a
13 fourth-high metric up to 84 ppb (U.S. EPA 2007, pp. 7-63 to 7-64; 80 FR 65395, October 26,
14 2015). Given the control of the averaging time and form of the current standard on peak
15 concentrations (as discussed in section 4.4.1 above), this observation is consistent with a role for
16 peak concentrations in eliciting visible foliar injury. Although given that lower design values for
17 the current standard also yield lower W126 index values, the relative influence of peak
18 concentrations and cumulative seasonal exposures cannot be distinguished. With regard to the
19 control of the current standard on peak concentrations, however, we note the conceptual
20 similarity to the finding of the most recent and extensive USFS data analysis that reductions in
21 peak 1-hour concentrations have influenced the declining trend in visible foliar injury since 2002
22 (Smith, 2012).

23 In consideration of all of the above, we recognize the appreciable limitations of the
24 available information touched on above with regard to providing a foundation for judgments on
25 public welfare protection objectives specific to visible foliar injury. In light of such limitations
26 and in light of the above discussion, we recognize that while the evidence continues to show a
27 consistent association between the occurrence of visible injury and ozone, “visible foliar injury is
28 not always a reliable indicator of other negative effects on vegetation” (ISA, Appendix 8, section
29 8.2), and we do not have a precise understanding of the appropriate metrics for quantifying O₃ air
30 quality conditions for the purposes of informing the Administrator’s consideration of this
31 endpoint. Based on studies and analyses of the USFS biosite data, the conditions associated with
32 visible foliar injury in locations with sensitive species appear to relate to peak concentration

¹⁰⁷ In considering their findings, the authors expressed the view that “[a]lthough the number of sites or species with injury is informative, the average biosite injury index (which takes into account both severity and amount of injury on multiple species at a site) provides a more meaningful measure of injury” for their assessment at a statewide scale (Campbell et al., 2007).

1 (e.g., hours above a concentration such as 100 ppb) as well as sustained exposure to higher
2 concentrations over the growing season, such that cumulative exposure metrics may not well or
3 completely describe or predict the occurrence and severity of injury. Thus, in making judgments
4 regarding air quality conditions of concern and those providing protection with regard to impacts
5 associated with incidence and severity of visible foliar injury, we find it appropriate to consider
6 both cumulative concentration-weighted seasonal exposures and the occurrence of peak
7 concentrations. In this context, we note the control of these metrics achieved by the form and
8 averaging time of the current standard, as discussed in section 4.4 above. Lastly, we take note of
9 the USFS BI scheme as potentially useful to informing the Administrator’s consideration of the
10 potential public welfare significance of differing magnitudes of BI scores.

11 • **What does the available information indicate for considering potential public welfare**
12 **protection from O₃-related climate effects?**

13 In considering the available information for the effects of the global abundance of O₃ in
14 the troposphere on radiative forcing, and temperature, precipitation and related climate variables,
15 we note as an initial matter that, as summarized in section 4.3.3 above, there are limitations and
16 uncertainties in the associated evidence bases with regard to assessing potential for occurrence of
17 climate-related effects as a result of varying ground-level O₃ concentrations in ambient air of
18 locations in the U.S. Specifically, such limitations and uncertainties affect our ability to
19 characterize the extent of any relationships between O₃ concentrations in ambient air in the U.S.
20 and climate-related effects, thus precluding a quantitative characterization of climate responses
21 to changes in ground-level O₃ concentrations in ambient air at regional (vs global) scales that
22 might inform considerations related to the current standard. While the evidence supports a causal
23 relationship between the global abundance of O₃ in the troposphere and radiative forcing, and a
24 likely causal relationship between the global abundance of O₃ in the troposphere and effects on
25 temperature, precipitation, and related climate variables (ISA, section IS.5.2 and Appendix 9;
26 Myhre et al., 2013), the non-uniform distribution of O₃ (spatially and temporally) makes the
27 development of quantitative relationships between the magnitude of such effects and differing
28 ground-level O₃ concentrations in the U.S. challenging (ISA, Appendix 9). Additionally, “the
29 heterogeneous distribution of ozone in the troposphere complicates the direct attribution of
30 spatial patterns of temperature change to ozone induced [radiative forcing]” and there are “ozone
31 climate feedbacks that further alter the relationship between ozone [radiative forcing] and
32 temperature (and other climate variables) in complex ways” (ISA, Appendix 9, section 9.3.1, p.
33 9-19). Thus, various uncertainties “render the precise magnitude of the overall effect of
34 tropospheric ozone on climate more uncertain than that of the well-mixed GHGs” and “[c]urrent
35 limitations in climate modeling tools, variation across models, and the need for more
36 comprehensive observational data on these effects represent sources of uncertainty in quantifying

1 the precise magnitude of climate responses to ozone changes, particularly at regional scales”
2 (ISA, section IS.6.2.2, Appendix 9, section 9.3.3, p. 9-22).

3 As one example, current limitations in modeling tools include “uncertainties associated
4 with simulating trends in upper tropospheric ozone concentrations” (ISA, section 9.3.1, p. 9-19),
5 and uncertainties such as “the magnitude of [radiative forcing] estimated to be attributed to
6 tropospheric ozone” (ISA, section 9.3.3, p. 9-22). Further, “precisely quantifying the change in
7 surface temperature (and other climate variables) due to tropospheric ozone changes requires
8 complex climate simulations that include all relevant feedbacks and interactions” (ISA, section
9 9.3.3, p. 9-22). An important specific limitation in current climate modeling capabilities for O₃ is
10 representation of important urban- or regional-scale physical and chemical processes, such as O₃
11 enhancement in high-temperature urban situations or O₃ chemistry in city centers where NO_x is
12 abundant. Because of such limitations in the available information, we lack the ability to quantify
13 or judge the impact of incremental changes in ground-level O₃ concentrations in the U.S. on
14 radiative forcing and subsequent climate effects, thus precluding a consideration of potential
15 public welfare protection provided by the existing O₃ standard from O₃-related climate effects.¹⁰⁸

16 **4.5.1.3 Public Welfare Implications of Air Quality under the Current Standard**

17 Our consideration of the available scientific evidence in this reconsideration, as at the
18 time of the 2015 review, is informed by results from a quantitative analysis of air quality and
19 associated exposure. An overarching consideration is whether this information calls into question
20 the adequacy of protection provided by the current standard. As in our consideration of the
21 evidence above, we have organized the discussion regarding the information related to exposures
22 and potential risk around a key question to assist us in considering the quantitative analyses of air
23 quality at U.S. locations nationwide, particularly including those in Class I areas. We first
24 consider analyses particular to cumulative O₃ exposures, in terms of the W126 index, given the
25 established E-R relationships with growth-related effects, and specifically RBL as the identified
26 proxy or surrogate for the full array of such effects.

27 To understand the cumulative O₃ exposures likely occurring under the current standard
28 nationally, including in Class I areas, we consider the air quality analyses summarized in section
29 4.4 above. Nationwide in the most recent 3-year period, seasonal W126 index values are at or
30 below 17 ppm-hrs, as assessed by the 3-year average, when the current standard is met (Table 4-

¹⁰⁸ While these complexities inhibit our ability to analyze and quantitatively climate-related effects of O₃, such as radiative forcing, we note that our consideration of O₃ growth-related impacts on trees inherently encompasses consideration of the potential for O₃ to reduce carbon sequestration in terrestrial ecosystems (e.g., through reduced tree biomass as a result of reduced growth). That is, limiting the extent of O₃-related effects on growth would be expected to also limit reductions in carbon sequestration, a process that can reduce the tropospheric abundance of CO₂, the greenhouse gas ranked highest in importance (section 4.3.3.3 above; ISA, section 9.1.1).

1 3). With very few exceptions, this is also true across the full historical period. Further, such
2 exposures are generally well below 17 ppm-hrs across most of the U.S. Additionally, the overall
3 pattern for single-year seasonal W126 index values at monitors meeting the current standard in
4 the most recent period is generally similar, with few sites (about a dozen of the 877 sites
5 nationwide) having a single-year W126 index above 19 ppm-hrs (and under two dozen above 17
6 ppm-hrs).¹⁰⁹ The frequency of such higher single-year W126 index values at Class I area
7 monitors is also low during periods when the current standard is met. During the most recent
8 three years, the average seasonal W126 index is at or below 17 ppm-hrs at all Class I area
9 monitors meeting the current standard, just two single-year W126 index values above 17 ppm-
10 hrs and none above 19 ppm-hrs (Appendix 4D, Table 4D-16).¹¹⁰

11 Combining this information regarding likely W126-based exposure levels with the
12 established E-R functions for 11 tree seedling species indicates that based on monitoring data for
13 locations meeting the current standard during the most recent design period, the median species
14 RBL for tree seedlings, based on the 3-year average W126, would be at or below 5.3%, with
15 very few exceptions; the highest estimates are associated with W126 index values occurring in
16 areas that are not near or within Class I areas. Looking at the data over a longer time period
17 (2000-2018) confirms this general pattern for the bulk of the data, with some infrequent higher
18 occurrences, such that virtually all RBL estimates would be below 6%.¹¹¹ Further, given the
19 variability and uncertainty associated with the data underlying the E-R functions (as discussed in
20 section 4.5.1.2 above), the few higher single-year occurrences are reasonably considered to be of
21 less significance than 3-year average values.

22 With regard to visible foliar injury, as discussed earlier, the evidence is somewhat limited
23 and unclear with regard to the metric and quantitative approach that well describes a relationship
24 between incidence or severity of injury in U.S. forests across a broad range of air quality
25 conditions. However, we note several key findings of the evidence and quantitative analyses.
26 First, the increased incidence of BI scores associated with injury considered greater than “a
27 little” by the USFS scheme appears most consistently with higher W126 estimates, with greatest

¹⁰⁹ These highest W126 index values occur in the Southwest and West regions in which there are nearly 150 monitor locations meeting the current standard (Figure 4-6; Appendix 4D, Table 4D-1).

¹¹⁰ Across the full 21-year dataset for Class I area monitors meeting the current standard (57 monitors with at least one such period), there are 15 design value periods with single-year W126 index values above 19 ppm-hrs, all of which are prior to the 2013-2015 period (Appendix 4D, section 4D.3.2.4).

¹¹¹ Although potential for effects on crop yield was not given particular emphasis in the 2015 review (for reasons similar to those summarized earlier), we additionally note that combining the exposure levels summarized for areas across the U.S. where the current standard is met with the E-R functions established for 10 crop species indicates a median RYL across crops to be at or below 5.1%, on average, with very few exceptions. Further, estimates based on W126 index at the great majority of the areas are below 5%.

1 incidence for the highest exposure level (W126 index above 25 ppm-hrs), a magnitude not seen
2 to occur in Class I area monitoring sites, or in virtually any sites nationwide, that meet the
3 current standard (Appendix 4C, section 4C.3). Further, we note a decline in frequency of peak
4 hourly concentrations, including those at/above 100 ppb, at U.S. monitoring sites over the past
5 15 years. The analyses of hourly concentrations summarized in section 4.4.1 above, also
6 demonstrate substantial control of peak 1-hour concentrations by the current standard. Thus, we
7 lack an established metric or combination of metrics that well describes the relationship between
8 occurrence and severity of visible foliar injury across a broad range of O₃ concentration patterns
9 from those more common in the past to those in areas recently meeting today's standard, the
10 current information indicates air quality conditions of concern for this endpoint to generally
11 include cumulative seasonal exposures, in terms of seasonal single-year W126 index, at/above 25
12 ppm-hrs, in addition to appreciable occurrence of peak hourly concentrations at/above 100 ppb.
13 Based on this information, the available air quality information indicates that the exposure
14 conditions occurring at sites with air quality meeting the current standard are not those that might
15 reasonably be concluded to elicit the occurrence of significant foliar injury (with regard to
16 severity and extent).

- 17 • **Are such exposures (in terms of W126 index) that occur in areas that meet the**
18 **current standard indicative of welfare effects reasonably judged important from a**
19 **public welfare perspective? What are important associated uncertainties?**

20 Given the findings summarized in section 4.4 above regarding W126 index values in
21 areas where the current standard is met, we reflect on the potential public welfare significance of
22 vegetation-related effects that may be associated with such exposures. This consideration is
23 important to judgments regarding the secondary standard, which is not meant to protect against
24 all known or anticipated O₃-related welfare effects, but rather those that are judged to be adverse
25 to the public welfare (as noted in section 4.3.2 above). Accordingly, for the purposes of
26 informing that judgment, we consider here the exposures indicated to occur under conditions that
27 meet the current standard, the associated potential for effects and the potential public welfare
28 implications.

29 As an initial matter, we recognize the increased significance to the public welfare of
30 effects in areas that have been accorded special protection, such as Class I areas. In this context,
31 we note some general similarities of the exposure estimates in Class I areas for periods when the
32 current standard was met to such estimates at monitoring sites in other areas, as documented in
33 the larger air quality data analysis. Across both datasets, and extending back 21 years, the
34 cumulative exposure estimates, averaged over the design value period, for these air quality
35 conditions were virtually all at or below 17 ppm-hrs, with most of the W126 index values below
36 13 ppm-hrs (Appendix 4D, Table 4D-10), corresponding to median RBL estimates of 3.8% or

1 less (based on the established tree seedling E-R relationships detailed in Appendix 4A). We
2 additionally note that single-year W126 index values in Class I areas over the 21-year dataset
3 evaluated were generally at or below 19 ppm-hrs, particularly in the more recent years
4 (Appendix 4D, section 4D.3.2.4). Regarding the potential for effects associated with commonly
5 occurring exposures, we consider first the categories of effects for which the quantitative
6 information related to exposure and associated effects is most well developed. In this
7 reconsideration, as in the 2015 review, these are effects on plant growth. Based on the median of
8 RBL estimates derived from the established E-R functions for 11 tree species seedlings, W126
9 index values at or below 17 ppm-hrs correspond to median species tree seedling RBL estimates
10 at or below 5.3% (Appendix 4A, Table 4A-5). Judgments in the 2015 review (in the context of
11 the framework considered in section 4.5.1.2 above) concluded isolated rare occurrences of
12 exposures for which median RBL estimates might be at or just above 6% to not be indicative of
13 conditions adverse to the public welfare, particularly considering the variability in the array of
14 environmental factors that can influence O₃ effects in different systems, and the uncertainties
15 associated with estimates of effects in the natural environment.

16 In the 2015 review, the Administrator focused on cumulative exposure estimates derived
17 as the average W126 index over the 3-year design value period, concluding variations of single-
18 year W126 index from the average to be of little significance. This focus generally reflected the
19 judgment that estimates based on the average adequately, and appropriately, reflected the
20 precision of the current understanding of O₃-related growth reductions, given the various
21 limitations and uncertainties in such predictions. Additional analyses have been explored since
22 the 2015 to further examine this issue, as summarized in section 4.5.1.2 above. The current air
23 quality data indicate single-year W126 index values generally to vary by less than 5 ppm-hrs
24 from the 3-year average when the 3-year average is below 20 ppm-hrs (which is the case for
25 locations meeting the current standard). With such variation, year-to-year differences in tree
26 growth responding to each year's seasonal exposure from estimated response based on the 3-year
27 average of those seasonal exposures would, given the offsetting impacts of seasonal exposures
28 above and below the average, reasonably be expected to generally be small over tree lifetimes.
29 Additionally, we have also further considered the experimental data underlying the E-R
30 functions for estimating RBL, particularly those pertaining to cumulative exposures on the order
31 of 17 ppm-hrs and informing estimates of multiyear impacts. We note limitations in the evidence
32 base in these regards, as discussed further in section 4.5.1.2 above, that contribute to imprecision
33 or inexactitude to estimates of growth impacts associated with multi-year exposures in this range.
34 Further, the information available since 2015 does not appreciably address these limitations and
35 uncertainties to improve the certainty or precision in RBL estimates for such exposures.

1 With regard to visible foliar injury, as discussed in sections 4.3.3.2 and 4.5.1.2 above, a
2 quantitative description of the relationship between O₃ concentrations and visible foliar injury
3 extent or incidence, as well as severity, that would support estimation of injury under varying air
4 quality and environmental conditions (e.g., moisture), most particularly for locations that meet
5 the current standard is not yet established. In light of the potential role of peak O₃ concentrations
6 (e.g., hourly concentrations at or above 100 ppb) as an influence on visible foliar injury
7 occurrence and severity (that may not be fully captured by a focus on cumulative seasonal O₃
8 indices), we take note of analyses of peak concentrations summarized in section 4.4.1. These
9 indicate that the magnitude of daily maximum 1-hour concentrations has declined appreciably
10 since 2000. For example, the median annual 2nd highest MDA1 concentration across U.S. trend
11 monitoring sites declined by 27% from 2002 to 2013 (Figure 2-17 above), and the 99th percentile
12 MDA1 for all sites meeting the current standard in 2020 is below 80 ppb (Figure 4-11)). The
13 analysis in Appendix 2A of three recent design value periods (covering 2016 through 2020) and
14 three periods more than ten years prior (covering 2000 through 2004) show that the mean
15 number of observations per site at or above 100 ppb was well below one (0.22) for sites meeting
16 the current standards compared to well above one (10.04) for sites not meeting the current
17 standard. Further, the number of days with an hour at or above 100 ppb is below five at sites
18 meeting the current standard, and 99% are well below five (Figure 4-11, Appendix 2A, section
19 2A.2). These data and analyses indicate that the current standard provides appreciable control of
20 peak 1-hour concentrations, and thus, to the extent that such peak concentrations play a role in
21 the occurrence and severity of visible foliar injury, the current standard also provides appreciable
22 control.

23 In considering protection for visible foliar injury impacts provided by the standard, we
24 note, as discussed in section 4.3.2 above, that the public welfare implications associated with
25 visible foliar injury (when considered as an effect separate from effects on plant physiology)
26 relate largely to effects on scenic and aesthetic values. The available information does not yet
27 address or describe the relationships expected to exist for some level of visible foliar injury
28 severity (below that at which broader physiological effects on plant growth and survival might
29 also be expected) and/or extent of location or site injury (e.g., BI) scores with values held by the
30 public and associated impacts on public uses of the locations.¹¹² As discussed in section 4.3.2
31 above, this gap limits our ability to identify air quality conditions that might be expected to
32 provide a specific level of protection from public welfare effects of this endpoint (e.g., separate
33 from effects that might relate to plant growth and reproduction under conditions where foliar

¹¹² Information with some broadly conceptual similarity to this has been used for judging public welfare implications of visibility effects of PM in setting the PM secondary standard (78 FR 3086, January 15, 2012).

1 injury may also be severe).¹¹³ Thus, key considerations of this endpoint in past reviews have
2 related to qualitative consideration of potential impacts related to the plant’s aesthetic value in
3 protected forested areas and the somewhat general, nonspecific judgment that a more restrictive
4 standard is likely to provide increased protection. Nevertheless, while minor spotting on a few
5 leaves of a plant may easily be concluded to be of little public welfare significance, it is
6 reasonable to conclude that cases of widespread and relatively severe injury during the growing
7 season (particularly when sustained across multiple years and accompanied by obvious impacts
8 on the plant canopy) would likely impact the public welfare in scenic and/or recreational areas,
9 particularly in areas with special protection, such as Class I areas. In this context, we note the
10 potential usefulness of the USFS scheme for the purposes of informing the Administrator’s
11 judgments with regard to public welfare significance of such effects.

12 In light of the discussions here and in sections 4.3.3.2 and 4.5.1.2 (with consideration of
13 presentations in Appendix 4C and air quality analyses in Appendices 2A, 4D and 4F) we find
14 that the available information does not indicate that a situation of widespread and relatively
15 severe visible foliar injury is likely associated with air quality that meets the current standard.
16 More specifically, the air quality data for areas meeting the standard do not indicate conditions
17 associated with BI scores reasonably considered of concern in the context described above
18 (concerning potential for public welfare significance). For example, we note that the air quality
19 analyses indicate that virtually all seasonal W126 index values at locations meeting the current
20 standard are below 25 ppm-hr. Further, the average number of observations of 1-hour
21 concentrations at or above 100 ppb per site and design value period are well below one during
22 periods when the current standard is met. Thus, while the current evidence is limited for the
23 purposes of identifying public welfare protection objectives related to visible foliar injury in
24 terms of specific air quality metrics, the current information indicates that the occurrence of
25 injury categorized as more severe than “little” by the USFS categorization (i.e., a BI score above
26 5 or above 15) would be expected to be infrequent in areas that meet the current standard. Based
27 on the USFS dataset presentations as well as the air quality analyses of W126 index values and
28 frequency of 1-hour observations at or above 100 ppb, the prevalence of injury scores
29 categorized as severe, which, depending on spatial extent, might contribute to impacts of public
30 welfare significance do not appear likely to occur under air quality conditions that meet the
31 current standard.

32 With regard to other vegetation-related effects, including those at the ecosystem scale,
33 such as alteration in community composition or reduced productivity in terrestrial ecosystems, as

¹¹³ Further, no criteria have been established regarding a level or prevalence of visible foliar injury considered to be adverse to the affected vegetation as the current evidence does not provide for determination of a degree of leaf injury that would have significance to the vigor of the whole plant (ISA, Appendix 8, p. 8-24).

1 recognized in section 4.5.1.1, the available evidence is not clear with regard to the risk of such
2 impacts (and their magnitude or severity) associated with the environmental O₃ exposures
3 estimated to occur under air quality conditions meeting the current standard (e.g., W126 index
4 generally at or below 17 ppm-hrs). In considering effects on crop yield, the air quality analyses at
5 monitoring locations that meet the current standard indicate estimates of RYL for such
6 conditions to be at and below 5.1%, based on the median estimate derived from the established
7 E-R functions for 10 crops (Appendix 4A, Table 4A-5). We additionally recognize there to be
8 complexities involved in interpreting the significance of such small estimates in light of the
9 factors identified in section 4.3.2 above. These include the extensive management of crops in
10 agricultural areas that may to some degree mitigate potential O₃-related effects, as well as the use
11 of variable management practices to achieve optimal yields, while taking into consideration
12 various environmental conditions. We also recognize that changes in yield of commercial crops
13 and commercial commodities may affect producers and consumers differently, further
14 complicating consideration of these effects in terms of potential adversity to the public welfare
15 impacts. In light of these factors complicating conclusions regarding crop yield impacts, in
16 combination with the relatively low RYL estimates associated with W126 index values occurring
17 in areas meeting the current standard, as well as the relative scarcity of peak hourly
18 concentrations at or above 100 ppb, a situation which differs from the extensive occurrences
19 associated with the exposure treatments on which the established E-R functions for the 10 crop
20 species are based (e.g., Lefohn and Foley, 1992), the current information does not indicate
21 exposures occurring in areas meeting the current standard to be of public welfare significance
22 with regard to crop yield.

23 **4.5.2 Preliminary Conclusions**

24 This section describes preliminary conclusions for the Administrator’s consideration with
25 regard to the current secondary O₃ standard. These preliminary conclusions are based on
26 consideration of the assessment and integrative synthesis of the evidence (as summarized in the
27 ISA, and the 2013 ISA and AQCDs from prior reviews), and the information on quantitative
28 exposure and air quality analyses summarized above. Taking into consideration the discussions
29 above in this chapter, this section addresses the following overarching policy question.

- 30 • **Do the scientific evidence and air quality and exposure analyses support or call into**
31 **question the adequacy of the protection afforded by the current secondary O₃**
32 **standard?**

33 In considering this question, we first recognize what the CAA specifies with regard to
34 protection to be provided by the secondary standard. Under section 109(b)(2) of the CAA, a
35 secondary standard must “specify a level of air quality the attainment and maintenance of which,

1 in the judgment of the Administrator, based on such criteria, is requisite to protect the public
2 welfare from any known or anticipated adverse effects associated with the presence of [the]
3 pollutant in the ambient air.” Accordingly, as noted in section 4.3.2 above, the secondary
4 standard is meant to protect against O₃-related welfare effects that are judged to be adverse to the
5 public welfare (78 FR 8312, January 15, 2013; see also 73 FR 16496, March 27, 2008). Thus,
6 our consideration of the available information regarding welfare effects of O₃ is in this context,
7 while recognizing that the level of protection from known or anticipated adverse effects to public
8 welfare that is requisite for the secondary standard is a public welfare policy judgment made by
9 the Administrator.

10 As is the case in NAAQS reviews in general, the extent to which the protection provided
11 by the current secondary O₃ standard is judged to be adequate will depend on a variety of factors,
12 including science policy judgments and public welfare policy judgments. These factors include
13 public welfare policy judgments concerning the appropriate benchmarks on which to place
14 weight, as well as judgments on the public welfare significance of the effects that have been
15 observed at the exposures evaluated in the welfare effects evidence. The factors relevant to
16 judging the adequacy of the standard also include the interpretation of, and decisions as to the
17 weight to place on, different aspects of the quantitative analyses of air quality and cumulative O₃
18 exposure and any associated uncertainties. Thus, we recognize that the Administrator’s
19 conclusions regarding the adequacy of the current standard will depend in part on public welfare
20 policy judgments, science policy judgments regarding aspects of the evidence and exposure/risk
21 estimates, as well as judgments about the level of public welfare protection that is requisite under
22 the Clean Air Act.

23 As an initial matter, we recognize the continued support in the current evidence for O₃ as
24 the indicator for photochemical oxidants (as summarized in section 4.5.1.1 above). We note that
25 no newly available evidence has been identified since the 2015 decision regarding the
26 importance of photochemical oxidants other than O₃ with regard to abundance in ambient air,
27 and potential for welfare effects, and that, as stated in the current ISA, “the primary literature
28 evaluating the health and ecological effects of photochemical oxidants includes ozone almost
29 exclusively as an indicator of photochemical oxidants” (ISA, section IS.1.1). Thus, we recognize
30 that, as was the case for the 2015 and prior reviews, the evidence base for welfare effects of
31 photochemical oxidants does not indicate an importance of any other photochemical oxidants.
32 Thus, we conclude that the evidence continues to support O₃ as the indicator for the secondary
33 NAAQS for photochemical oxidants.

34 Our response to the overarching question above takes into consideration the discussions
35 that address the specific policy-relevant questions in prior sections of this document and the
36 approach described in section 4.2. We consider the evidence and the extent to which it alters key

1 conclusions supporting the current standard. We also consider the quantitative analyses,
2 including associated limitations and uncertainties, and what they may indicate regarding level of
3 protection provided by the current standard from adverse effects. We additionally consider the
4 key aspects of the evidence and air quality/exposure information emphasized in establishing the
5 now-current standard, and the associated public welfare policy judgments and judgments about
6 inherent uncertainties that are integral to decisions on the adequacy of the current secondary O₃
7 standard. Together these considerations contribute to our preliminary conclusion as to whether
8 the available scientific evidence and air quality and exposure analyses support or call into
9 question the adequacy of the protection afforded by the current secondary O₃ standard.

10 In considering the available evidence, we recognize the longstanding evidence base of the
11 vegetation-related effects of O₃, augmented in some aspects since the 2015 review. Consistent
12 with the evidence in the 2015 review, the existing evidence describes an array of effects on
13 vegetation and related ecosystem effects causally or likely causally related to O₃ in ambient air,
14 as well as the causal relationship of tropospheric O₃ with radiative forcing and subsequent likely
15 causally related effects on temperature, precipitation, and related climate variables. As was the
16 case in the 2015 review, a category of effects for which the evidence supports quantitative
17 description of relationships between air quality conditions and response is plant growth or yield.
18 The evidence base continues to indicate growth-related effects as sensitive welfare effects, with
19 the potential for ecosystem-scale ramifications. For this category of effects, there are established
20 E-R functions that relate cumulative seasonal exposure of varying magnitudes to various
21 incremental reductions in expected tree seedling growth (in terms of RBL) and in expected crop
22 yield (in terms of RYL). Many decades of research also recognize visible foliar injury as an
23 effect of O₃, although uncertainties continue to hamper efforts to quantitatively characterize the
24 relationship of its occurrence and relative severity with O₃ exposures. The evidence for these
25 categories of vegetation-related O₃ effects is discussed further below. But before focusing further
26 on these key vegetation-related effects, we address two endpoints newly identified in the 2020
27 ISA, as well as tropospheric O₃ effects related to climate.

28 With regard to categories of effects newly identified in the 2020 ISA as likely causally
29 related to O₃ in ambient air, such as alteration of plant-insect signaling and insect herbivore
30 growth and reproduction, we recognize that uncertainties limit our consideration of the
31 protection that might be provided by the current standard against these effects. Depending on a
32 number of factors, such effects may have a potential for adverse effects to the public welfare,
33 e.g., given the role of plant-insect signaling in such important ecological processes as pollination
34 and seed dispersal, as well as natural plant defenses against predation and parasitism (as
35 discussed in section 4.3.2 above Uncertainties in the evidence, however, preclude a sufficient
36 understanding to support a focus on such effects in considering protection provided by the

1 current standard. Areas of uncertainty and limitations in the evidence include key aspects of such
2 effects, the air quality conditions that might elicit them (and the magnitude or severity), the
3 potential for impacts in a natural ecosystem and, consequently, the potential for such impacts
4 under air quality conditions associated with meeting the current standard, as discussed in section
5 4.5.1.1 above. Thus, we do not find the evidence to provide sufficient information to support
6 judgments related to how particular patterns of O₃ concentrations in ambient air may relate to the
7 occurrence of such effects in natural systems or, accordingly, to any related impacts to the public
8 welfare.

9 We next recognize the strong evidence documenting tropospheric O₃ as a greenhouse gas
10 causally related to radiative forcing, and likely causally related to subsequent effects on variables
11 such as temperature and precipitation. In so doing, however, we take note of the limitations and
12 uncertainties in the evidence base that affect our ability to characterize the extent of any
13 relationships between O₃ concentrations in ambient air in the U.S. and climate-related effects,
14 thus precluding a quantitative characterization of climate responses to changes in O₃
15 concentrations in ambient air at regional (vs global) scales (as summarized in sections 4.3.3.3
16 and 4.3.4 above).¹¹⁴ As a result, we recognize the lack of important quantitative tools with which
17 to consider such effects in the context of protection provided by the current secondary O₃
18 standard, such that it is not feasible to relate different patterns of O₃ concentrations at the
19 regional (or national) scale in the U.S. with specific risks of alterations in temperature,
20 precipitation and other climate-related variables. We find these significant limitations and
21 uncertainties together to contribute to an insufficiency in the available information for the
22 purposes of supporting the Administrator's judgments particular to a secondary O₃ NAAQS and
23 protection of the public welfare from adverse effects linked to O₃ influence on radiative forcing,
24 and related climate effects.¹¹⁵ Thus, as is the case for the two newly identified categories of
25 insect-related effects discussed above, we conclude that the available evidence does not support a
26 focus on radiative forcing and related climate effects in considering the extent to which the
27 available evidence supports or calls into question the adequacy of protection afforded by the
28 current secondary standard.

29 Turning next to consideration of visible foliar injury, the available information has been
30 examined and analyzed as to what it indicates and supports with regard to adequacy of protection

¹¹⁴ With regard to radiative forcing and effects on temperature, precipitation, and related climate variables, while additional characterizations have been completed since the 2015 review, uncertainties and limitations in the evidence that were also recognized at that time remain.

¹¹⁵ Notwithstanding consideration of these effects, we note that a focus on the protection offered by the standard against vegetation-related effects is expected to also have positive implications for climate change protection through the protection of terrestrial ecosystem carbon storage.

1 provided by the current standard (e.g., as discussed in section 4.5.1 above). Visible foliar injury
2 is an effect for which an association with O₃ in ambient air is well documented. The public
3 welfare significance of visible foliar injury of vegetation in areas not closely managed for
4 harvest, particularly specially protected natural areas, has generally been considered in the
5 context of potential effects on aesthetic and recreational values, such as the aesthetic value of
6 scenic vistas in protected natural areas such as national parks and wilderness areas (e.g., 73 FR
7 16496, March 27, 2008). Accordingly, depending on its severity and spatial extent, as well as the
8 location(s) and the associated intended use, its effects on the physical appearance of the plant
9 have the potential to be significant to the public welfare. For example, while limited occurrences
10 (e.g., of severity or prevalence) may easily be concluded to be of little public welfare
11 significance, cases of widespread and relatively severe injury during the growing season
12 (particularly when sustained across multiple years and accompanied by obvious impacts on the
13 plant canopy) might reasonably be expected to have the potential to adversely impact the public
14 welfare in scenic and/or recreational areas, particularly in areas with special protection, such as
15 Class I areas.

16 In considering existing approaches for categorizing the severity of injury in natural areas,
17 we take note of the system developed by the USFS for its monitoring program¹¹⁶ to categorize BI
18 scores of visible foliar injury at biosites (sites with O₃-sensitive vegetation assessed for visible
19 foliar injury) in natural vegetated areas by severity levels (described in section 4.3.2 above). We
20 recognize, however, that quantitative analyses and evidence are lacking that might support a
21 precise conclusion - and associated judgment - as to a magnitude of BI score coupled with an
22 extent of occurrence that might be specifically identified as adverse to the public welfare. That
23 notwithstanding, we additionally note that the scale of the USFS biosite monitoring program's
24 objectives, which focus on natural settings in the U.S. and forests as opposed to individual
25 plants, may be informative to the Administrator with regard to his judgments concerning the
26 public welfare protection afforded by the current standard for such effects.

27 In considering the availability of established approaches that might be employed for
28 considering degrees of public welfare impacts related to the occurrence of visible foliar injury of
29 differing severity and extent (e.g., as summarized in sections 4.3.3.2 and 4.5.1.1 above), we note
30 the paucity of established approaches for interpreting specific levels of severity and extent of
31 foliar injury in protected forests with regard to impacts on public welfare effects (e.g., related to

¹¹⁶ During the period from 1994 (beginning in eastern U.S.) through 2011, the USFS conducted surveys of the occurrence and severity of visible foliar injury on sensitive species at sites across most of the U.S. following a national protocol (Smith, 2012).

1 recreational services).¹¹⁷ In this context, we recognize a potential usefulness of the USFS system,
2 including its descriptors for BI scores of differing magnitudes intended for that Agency’s
3 consideration in identifying areas of potential impact to forest resources. As described in section
4 4.3.2 above, very low BI scores (at or below 5) are described by the USFS scheme as “little or no
5 foliar injury” (Smith et al., 2007; Smith et al., 2012),¹¹⁸ and BI scores above 15 are categorized
6 as moderate to severe (and scores above 25 as severe). The lower categories of BI scores are
7 described by the USFS descriptions as indicative of injury of generally lesser risk to the natural
8 area, which we would suggest may also indicate lesser risk to public enjoyment. Accordingly, to
9 the extent that the USFS ranking system is of value to the Administrator’s judgments in this
10 context, it may be reasonable to conclude that occurrence of BI scores categorized as “moderate
11 to severe” injury by the USFS scheme would be an indication of visible foliar injury occurrence
12 that, depending on extent and severity, may be indicative of conditions of public welfare
13 significance. Thus, this framework may be informative to the Administrator’s consideration of
14 the evidence and analyses summarized in the sections above and what they indicate with regard
15 to patterns of air quality of concern for such an occurrence, and the extent to which they are
16 expected to occur in areas that meet the current standard.

17 We additionally consider the USFS biosite monitoring program studies of the occurrence,
18 extent, and severity of visible foliar injury in indicator species in defined plots or biosites in
19 natural areas across the U.S. Some of these studies, particularly those examining such data across
20 multiple years and multiple regions of the U.S., have reported that variation in cumulative O₃
21 exposure, in terms of metrics such as SUM06 or W126 index, does not completely explain the
22 patterns of occurrence and severity of injury observed. Although the availability of detailed
23 analyses that have explored multiple exposure metrics and other influential variables is limited,
24 multiple studies have indicated a potential role for an additional metric, one related to the
25 occurrence of days with relatively high concentrations (e.g., number of days with a 1-hour
26 concentration at or above 100 ppb), as summarized in section 4.5.1.2 above. Also noteworthy are
27 the publications related to the USFS biosite monitoring program that provide extensive evidence
28 of trends across the past nearly 20 years that indicate reductions in severity of visible foliar
29 injury that parallel reductions in peak concentrations that have been suggested to be influential in
30 the severity of visible foliar injury. For example, observations of such reductions in the incidence
31 of the higher BI scores over the 16-year period of the program (1994 through 2010), especially

¹¹⁷ This contrasts with another welfare effect, visibility, for which there is evidence relating to levels of visibility found to be acceptable by the public that was considered in judging the public welfare protection provided by the particulate matter secondary standard (78 FR 3226-3228, January 15, 2013).

¹¹⁸ Studies that consider such data for purposes of identifying areas of potential impact to the forest resource suggest this category corresponds to “none” with regard to “assumption of risk” (Smith et al., 2007; Smith et al., 2012).

1 after 2002, have led to researcher conclusions of a “declining risk of probable impact” on the
2 monitored forests over this period (e.g., Smith, 2012). These reductions parallel the O₃
3 concentration trend information nationwide that show clear reductions in cumulative seasonal
4 exposures, as well as in peak O₃ concentrations, both in terms of 8-hour and hourly
5 concentrations (e.g., Figures 2-11 and 2-17, and as summarized in section 4.4.1 above). . That is,
6 the extensive air quality evidence of trends across the past nearly 20 years indicate reductions in
7 peak concentrations that some studies have suggested to be influential in the severity of visible
8 foliar injury, as discussed in section 4.5.1 above.

9 In considering the available information that might inform the Administrator’s judgments
10 regarding visible foliar injury, we note a paucity of established approaches to inform the
11 Administrator’s judgment of a magnitude, severity or extent of visible foliar injury related effects
12 appropriately concluded to be known or anticipated to cause adverse effects to the public
13 welfare. However, some general conclusions or observations may be supported. For example,
14 based on the available evidence and associated quantitative analyses, we have less confidence
15 and greater uncertainty in the existence of adverse public welfare effects with lower O₃
16 exposures. More specifically, as discussed in the prior sections, the available information
17 suggests that O₃ air quality associated with W126 index values below 25 ppm-hrs (in a single
18 year), particularly when in combination with infrequent occurrences of hourly concentrations at
19 or above 100 ppb, is not likely to pose a risk of visible foliar injury in natural areas of an extent
20 and severity that might reasonably be considered to be of public welfare significance.

21 Support for this conclusion is seen in the air quality analyses that inform our
22 understanding of the occurrence and magnitude of cumulative seasonal exposures, in terms of
23 W126 index, and peak concentrations, in terms of the N100 and D100 metrics, in areas that meet
24 the current standard. These analyses indicate that virtually all W126 index values in a single year
25 are below 25 ppm-hrs at all monitoring locations (including in or near Class I areas) where the
26 current standard is met, and that, in fact, such values above 19 ppm-hrs are rare, as summarized
27 in section 4.4.2 above (Appendix 4D, sections 4D.3.1.24 and 4D.3.2.4). Thus, the analyses of air
28 quality since 2000 for areas that meet the current standard do not indicate the occurrence of
29 cumulative seasonal exposure, in terms of W126 index, of a magnitude that might be expected,
30 based on the available information (e.g., based on analyses of BI scores considered in sections
31 4.5.1.2 and 4.5.1.3 above), to contribute to a significant extent and degree of injury or specific
32 impacts on recreational or related services for areas, such as wilderness areas or national parks.
33 Further, we take note of the uncommonness of days with any hours at or above 100 ppb at
34 monitoring sites that meet the current standard, as well as the minimal number of hours on any
35 such days (as summarized in section 4.4.1). Based on these considerations, it would appear that
36 the current standard provides control of air quality conditions that contribute to increased BI

1 scores and to scores of a magnitude indicative of “moderate to severe” foliar injury. Thus, we
2 conclude that the evidence indicates that areas that meet the current standard are unlikely to have
3 BI scores reasonably considered to pose a risk of impacts of public welfare significance.
4 Accordingly, based on all of the considerations raised here, and in the sections above, we find it
5 reasonable to conclude that the available evidence and quantitative exposure information for
6 visible foliar injury do not call into question the adequacy of protection provided by the current
7 standard.

8 We turn now to consideration of the other vegetation-related effects, the evidence for
9 which as a whole is extensive, spans several decades, and supports the Agency’s conclusions of
10 causal or likely to be causal relationship for O₃ in ambient air with an array of effect categories
11 (as noted above). As an initial matter, we note the new ISA determination that the current
12 evidence is sufficient to infer likely causal relationships of O₃ with increased tree mortality,
13 while also noting that the evidence does not indicate a potential for O₃ concentrations that occur
14 in locations that meet the current standard to cause increased tree mortality, as summarized in
15 section 4.3.1 above.

16 As we turn our focus now to the more sensitive effect of vegetation growth and
17 conceptually related effects with a focus on RBL (described in section 4.5.1.2 above), we
18 recognize that public welfare policy judgments play an important role in decisions regarding a
19 secondary standard, just as public health policy judgments have important roles in primary
20 standard decisions. One type of public welfare policy judgment focuses on how to consider the
21 nature and magnitude of the array of uncertainties that are inherent in the scientific evidence and
22 analyses. These judgments are traditionally made with a recognition that current understanding
23 of the relationships between the presence of a pollutant in ambient air and associated welfare
24 effects is based on a broad body of information encompassing not only more established aspects
25 of the evidence but also aspects in which there may be substantial uncertainty. This may be true
26 even of the most robust aspect of the evidence base. In the case of the available evidence base, as
27 an example, we recognize increased uncertainty, and associated imprecision, at lower cumulative
28 exposures in application of the established and well-founded E-R functions, and in the current
29 understanding of aspects of relationships of such estimated effects with larger-scale impacts,
30 such as those on populations, communities, and ecosystems, as summarized in sections 4.5.1.3
31 and 4.3.4 above. Further, we recognize uncertainties in the details and quantitative aspects of
32 relationships between plant-level effects such as growth and reproduction, and ecosystem
33 impacts, the occurrence of which are influenced by many other ecosystem characteristics and
34 processes. These examples illustrate the role of public welfare policy judgments, both with
35 regard to the Administrator’s consideration of the extent of protection that is requisite and

1 concerning the weighing of uncertainties and limitations of the underlying evidence base and
2 associated quantitative analyses.

3 As summarized in section 4.1 above, the decisions that established the current standard in
4 2015, and retained it in 2020, involved a series of judgments contributing to the standard's
5 foundation with regard to growth-related effects. The first of these judgments relates to
6 consideration of the O₃ effect of reduced growth (quantified using the metric, RBL) as a proxy
7 for an array of other vegetation-related effects to the public welfare. The category of effects for
8 which the evidence is most certain with regard to quantitative functions describing relationships
9 between O₃ in ambient air and response continues to be reduced plant growth or yield. The
10 evidence base includes established E-R functions for seedlings of 11 tree species that relate
11 cumulative seasonal exposure of varying magnitudes to various incremental reductions in
12 expected tree seedling growth (in terms of RBL) and in expected crop yield. These functions are
13 well established and have been recognized across multiple O₃ NAAQS reviews. Uncertainties
14 related to use of the RBL estimates include the limited information regarding the extent to which
15 they reflect growth impacts in mature trees, and the fact that the 11 species represent a very small
16 portion of the tree species across the U.S.

17 While recognizing these and other uncertainties, RBL estimates based on the median of
18 the 11 species were used in the 2015 and 2020 decisions as a surrogate for comparable
19 information on other species and lifestages, as well as a proxy or surrogate for other vegetation-
20 related effects, including larger-scale effects. Use of this approach continues to appear to be a
21 reasonable judgment in this reconsideration of the 2020 decision. More specifically, the currently
22 available information continues to support (and does not call into question) the consideration of
23 RBL as a useful and evidence-based approach for consideration of the extent of protection from
24 the broad array of vegetation-related effects associated with O₃ in ambient air. As discussed in
25 section 4.5.1.2 above, these categories of effects include reduced vegetation growth,
26 reproduction, productivity, and carbon sequestration in terrestrial systems, and also alteration of
27 terrestrial community composition, belowground biogeochemical cycles, and ecosystem water
28 cycling. The current evidence base and available information (qualitative and quantitative), as in
29 the 2015 review, continue to support consideration of the potential for O₃-related vegetation
30 impacts in terms of the RBL estimates from established E-R functions as a quantitative tool
31 within a larger framework of considerations pertaining to the public welfare significance of O₃
32 effects. Such consideration would include effects that are associated with effects on vegetation,
33 and particularly those that conceptually relate to growth, and that are causally or likely causally
34 related to O₃ in ambient air, yet for which there are greater uncertainties affecting estimates of
35 impacts on public welfare. This approach to weighing the available information in reaching
36 judgments regarding the secondary standard additionally takes into account uncertainties

1 regarding the magnitude of growth impact that might be expected in mature trees, and of related,
2 broader, ecosystem-level effects for which the available tools for quantitative estimates are more
3 uncertain and those for which the policy foundation for consideration of public welfare impacts
4 is less well established. (80 FR 65389, October 26, 2015). The currently available evidence,
5 while somewhat expanded since the 2015 review, does not indicate an alternative metric for such
6 a use; nor is an alternative approach evident.

7 In considering tree growth effects, we take note of the other public welfare policy
8 judgments inherent in the Administrators' decisions in establishing the current standard in 2015,
9 and in retaining it in 2020. In addition to adoption of the median tree seedling RBL estimate for
10 the studied species as a surrogate for the broad array of vegetation related effects that extend to
11 the ecosystem scale, the decisions in 2015 and 2020 both incorporated the judgment that
12 cumulative seasonal exposures (in terms of the average W126 index across the 3-year design
13 period for the standard) associated with a median RBL somewhat below 6% is an appropriate
14 focus for considering target levels of protection for the secondary standard.

15 Decisions on the adequacy of secondary NAAQS require judgments on the extent to
16 which particular welfare effects (e.g., with regard to type, magnitude/severity, or extent) are
17 important from a public welfare perspective. In the case of O₃, such a judgment includes
18 consideration of the public welfare significance of small magnitude estimates of RBL and
19 associated unquantified potential for larger-scale related effects. In establishing the current
20 standard in 2015 with a focus on RBL as a proxy or surrogate for the broad array of vegetation
21 effects, the Administrator took note of the 2014 CASAC characterization of 6% RBL (in
22 seedlings of median tree species). As described in section 4.1 above, the rationale provided by
23 the CASAC with this characterization was primarily conceptual and qualitative, rather than
24 quantitative. The conceptual characterization recognized linkages between effects at the plant
25 scale and broader ecosystem impacts, with the CASAC recommending that the Administrator
26 consider RBL as a surrogate or proxy for the broader impacts that could be elicited by O₃. In the
27 2015 decision, the Administrator took note of this CASAC advice regarding use of RBL as a
28 proxy and set the standard with an underlying objective of limiting cumulative exposures (in
29 terms of W126 index, averaged over three years) "in nearly all instances to those for which the
30 median RBL estimate would be somewhat lower than 6%" (80 FR 65407, October 26, 2015).¹¹⁹
31 The information available in this reconsideration of the 2020 decision does not appear to call into
32 question such judgments, indicating them to continue to appear reasonable.

¹¹⁹ The 2015 decision additionally noted that "the Administrator does not judge RBL estimates associated with marginal higher exposures [at or above 19 ppm-hrs] in isolated, rare instances to be indicative of adverse effects to the public welfare" (80 FR 65407, October 26, 2015).

1 In considering what the available information indicates regarding the level of protection
2 for growth-related effects provided by the current standard, we recognize the importance of
3 considering the extent of both cumulative seasonal O₃ exposures and of elevated hourly
4 concentrations, as discussed in section 4.5.1.2 above. These aspects of O₃ air quality can
5 contribute to damaging conditions for vegetation. Thus, in considering the extent of protection
6 provided by the current standard, in addition to considering seasonal W126 index to estimate
7 median RBL using the established E-R functions, we also consider metrics that convey
8 information regarding peak hourly concentrations. While we recognize that the evidence does
9 not indicate a particular threshold number of hours at or above 100 ppb (or another reference
10 point for elevated concentrations), we take particular note of the evidence of greater impacts
11 from higher concentrations (particularly with increased frequency) and of the air quality analyses
12 that document variability in such concentrations for the same W126 index value. In light of these
13 factors, a multipronged approach is reasonably concluded to be appropriate for considering
14 exposures of concern and the protection from them that may be afforded by the secondary
15 standard.

16 The air quality analyses summarized in section 4.4 above describe the air quality
17 conditions that occur under the current standard and also the conditions in areas where the
18 standard is not met. We consider what is indicated regarding protection overall and protection
19 against “unusually damaging years” (an issue raised in the court remand of the 2015 decision on
20 the secondary standard). With regard to this issue, we take note of the air quality analyses
21 summarized in section 4.4.1, as also considered in section 4.5.1.2 above, that investigate the
22 annual occurrence of elevated hourly O₃ concentrations which may contribute to vegetation
23 exposures of concern (Appendix 2A, section 2A.2; Appendix 4F).¹²⁰ These air quality analyses
24 illustrate limitations of the W126 index for purposes of controlling peak concentrations, and also
25 the strengths of the current standard in this regard, showing that the form and averaging time of
26 the existing standard is much more effective than the W126 index in limiting peak concentrations
27 (e.g., hourly O₃ concentrations at or above 100 ppb) and in limiting number of days with any
28 such hours. As noted in prior sections, the W126 index, by virtue of its definition, does not
29 provide specificity with regard to year-to-year variability in elevated hourly O₃ concentrations
30 with the potential to contribute to the increased risk of vegetation effects, and the air quality
31 analyses illustrate this limitation. These analyses additionally document the control exerted by

¹²⁰ The ISA references the longstanding recognition of the risk posed to vegetation of peak hourly O₃ concentrations (e.g., “[h]igher concentrations appear to be more important than lower concentrations in eliciting a response” [ISA, p. 8-180]; “higher hourly concentrations have greater effects on vegetation than lower concentrations” [2013 ISA, p. 91-4] “studies published since the 2006 O₃ AQCD do not change earlier conclusions, including the importance of peak concentrations, ... in altering plant growth and yield” [2013 ISA, p. 9-117]).

1 the current standard, through all of its elements, on both cumulative seasonal O₃ exposures and
2 peak hourly concentrations.

3 In considering cumulative seasonal O₃ exposures occurring in areas that meet the current
4 standard with regard to growth-related effects represented by RBL (as discussed more fully
5 earlier, including in section 4.5.1.2), we focus, as was done in the 2015 decision, on a seasonal
6 W126 index, averaged across three years. In do so based on consideration of the extent of
7 conceptual similarities of the 3-year average W126 index with some aspects of the derivation
8 approach for the established E-R functions, the context of RBL as a proxy (as recognized above)
9 and other factors. With regard to the established E-R functions used to describe the relationship
10 of RBL with O₃ in terms of a seasonal W126 index, we recognize that the functions were derived
11 mathematically from studies of different exposure durations (varying from shorter than one to
12 multiple growing seasons) by applying adjustments so that they would yield estimates
13 normalized to the same period of time (season), such that the estimates may conceptually
14 represent average impact for a season. We note the compatibility of W126 index averaged over
15 multiple growing seasons or years with these adjustments. We also note that the exposure levels
16 represented in the data underlying the E-R functions are somewhat limited with regard to the
17 relatively lower cumulative exposure levels most commonly associated with the current standard
18 (e.g., at or below 17 ppm-hrs), with generally greater representation for higher exposures (e.g.,
19 ranging up to W126 index levels above 100 ppm-hrs), indicating additional uncertainty for
20 applications of the E-R functions to the lower cumulative exposure levels. We additionally note
21 the differing patterns of hourly concentrations of the elevated exposure levels (particularly with
22 regard to peak hourly concentrations, such as those at/above 100 ppb) in the datasets from which
23 the E-R functions from the patterns in ambient air meeting the current standard across the U.S.
24 today, as summarized in section 4.5.1.2 above. With these considerations regarding the E-R
25 functions and their underlying datasets in mind, we also take note of year-to-year variability of
26 factors other than O₃ exposures that affect tree growth in the natural environment (e.g., related to
27 variability in soil moisture, meteorological, plant-related and other factors), that have the
28 potential to affect O₃ E-R relationships, as noted in sections 4.3 and 4.5 above (ISA, Appendix 8,
29 section 3.12; 2013 ISA section 9.4.8.3). Thus, the use of the W126 index averaged over multiple
30 years has a compatibility with the approach used in deriving the E-R functions, and reflects
31 consideration of other aspects of the E-R function datasets and other factors that may affect
32 growth in the natural environment.

33 We additionally recognize the qualitative and conceptual nature of our understanding, in
34 many cases, of relationships of O₃ effects on plant growth and productivity with larger-scale
35 impacts, such as those on populations, communities and ecosystems. Based on these
36 considerations, use of a seasonal RBL averaged over multiple years, such as a 3-year average,

1 appears to be a reasonable approach, and provides a stable and well-founded RBL estimate for its
2 purposes as a proxy to represent the array of vegetation-related effects identified above. In light
3 of these considerations, we conclude there is support in the available information for use of an
4 average seasonal W126 index derived from multiple years (with their representation of
5 variability in environmental factors), and that the use of such averaging may provide an
6 appropriate representation of the evidence and attention to considerations summarized above.
7 Thus, we conclude that application of the multipronged approach referenced above would assess
8 anticipated exposures and protection afforded by the current secondary standard using a seasonal
9 W126 averaged over a 3-year period, which is the design value period for the current standard, to
10 estimate median RBL via the established E-R functions, in combination with a broader
11 consideration of air quality patterns, such as peak hourly concentrations.

12 In considering the quantitative analyses available in this review with regard to the control
13 of air quality conditions that might pose risks to the public welfare by the current standard, we
14 note the findings from the analysis of recent air quality at sites across the U.S., including in or
15 near 65 Class I areas, and also analyses of historical air quality. Findings from the analysis of the
16 air quality data from the most recent period and from the larger analysis of historical air quality
17 data extending back to 2000 are consistent with the air quality analysis findings that were part of
18 the basis for the current standard. That is, in virtually all design value periods and all locations at
19 which the current standard was met (more than 99.9% of the observations), the 3-year average
20 W126 metric was at or below 17 ppm-hrs, the target identified by the Administrator in
21 establishing the current standard and, in all such design value periods and locations, the W126
22 metric was at or below 19 ppm-hrs, as was also the case for the earlier and smaller dataset (80
23 FR 65404-65410, October 26, 2015). Additionally, across the full 21-year dataset for 56 Class I
24 areas with monitors meeting the current standard during at least one or as many as nineteen 3-
25 year periods since 2000, there are no more than 15 occurrences of a single-year W126 index
26 above 19 ppm-hrs, the majority occurring during the earlier years of the period (Appendix 4D,
27 section 4D.3.2.4, Tables 4D-14 and 4D-16). For example, the highest values were equal to 23
28 ppm-hrs, all occurring before 2012. Additionally, as emphasized in earlier sections, the current
29 standard better controls for peak concentrations (at or above 100 ppm-hrs), which may pose risks
30 of vegetation effects, than would be expected by either a single-year or three-year average
31 W126.¹²¹ Based on the evidence and air quality analyses described in sections 4.3 and 4.4 above,
32 as well as considerations summarized in section 4.5.1 above, the occurrences of 3-year average
33 W126 index values allowed by the current standard in Class I areas, including such infrequent

¹²¹ The historical dataset also shows the appreciable reductions in peak concentrations (via either the N100 or D100 metric) that have been achieved in the U.S. as air quality has improved under O₃ standards of the existing form and averaging time (Appendix 4F, Figures 4F-13 and 4F-14).

1 single-year deviations of the magnitude recognized here, above the average, can reasonably be
2 concluded not to raise concerns of adverse effects on the public welfare.

3 With regard to O₃ effects on crop yield, we take note of the long-standing evidence,
4 qualitative and quantitative, of the reducing effect of O₃ on the yield of many crops, as
5 summarized in the ISA and characterized in detail in past reviews (e.g., 2013 ISA, 2006 AQCD,
6 1997 AQCD, 2014 WREA). We also note the established E-R functions for 10 crops and the
7 estimates of RYL derived from them (Appendix 4A, section 4A.1, Table 4A-4), and the potential
8 public welfare significance of reductions in crop yield, as summarized in section 4.3.2 above. We
9 additionally recognize, however, that not every effect on crop yield will be judged adverse to
10 public welfare. In the case of crops in particular there are a number of complexities related to the
11 heavy management of many crops to obtain a particular output for commercial purposes, and
12 related to other factors, that are relevant to consider in evaluating potential O₃-related public
13 welfare impacts, as summarized in sections 4.3.2 and 4.5.1.3). For example, the extensive
14 management of agricultural crops that occurs to elicit optimum yields (e.g., through irrigation
15 and usage of soil amendments, such as fertilizer) is relevant to judgments concerning evaluation
16 of the extent of RYL estimated from experimental O₃ exposures reasonably considered to be
17 adverse to the public welfare. Such considerations include opportunities in crop management for
18 market objectives, as well as complications in judging relative adversity that relate to market
19 responses and their effects on producers and consumers in evaluating the potential impact on
20 public welfare of estimated crop yield losses.

21 In light of such complexities, uncertainties, and limitations, we have considered how
22 RYL estimates relate to RBL estimates identified above for evaluating protection provided by
23 the current standard. In this context, we note that W126 index values (3-year average) were at or
24 below 17 ppm-hrs in virtually all monitoring sites with air quality meeting the current standard.
25 Based on the established E-R functions, the median RYL estimate corresponding to 17 ppm-hrs
26 is 5.1%. In considering single-year index values, as discussed in section 4.4.2 above, the vast
27 majority are similarly low (with more than 99% less than or equal to 17 ppm-hrs), and the higher
28 values predominantly occur in urban areas. We additionally take note of the role of elevated
29 hourly concentrations in effects on vegetation growth and yield. In this context we also note the
30 extensive management of agricultural crops, and the complexities associated with identifying
31 adverse public welfare effects for market-traded goods (where producers and consumers may be
32 impacted differently). We also recognize that the current standard generally maintains air quality
33 at a W126 index below 17 ppm-hrs, with few exceptions, and accordingly would limit the
34 estimated RYL (based on experimental O₃ exposures) to this degree. In light of all of these
35 factors, we do not find the available information to call into question the adequacy of protection
36 afforded by the current standard for crop yield-related effects.

1 Thus, the available information leads us to conclude that the combined consideration of
2 the body of evidence and the quantitative air quality and exposure analyses, including associated
3 uncertainties, does not call into question the adequacy of the protection provided by the current
4 secondary standard. Rather, this information provides support for the current standard, and thus
5 supports consideration of retaining the current standard, without revision. In reaching these
6 conclusions, we recognize that the Administrator’s decisions in secondary standard reviews, in
7 general, are largely public welfare judgments, as described above. We further note that different
8 public welfare policy judgments (e.g., from those in both 2020 and 2015) could lead to different
9 conclusions regarding the extent to which the current standard provides the requisite protection
10 of the public welfare. Such public welfare judgments include those related to the appropriate
11 level of protection that should be afforded to protect against vegetation-related effects of public
12 welfare significance, as well as with regard to the appropriate weight to be given to differing
13 aspects of the evidence and air quality information, and how to consider their associated
14 uncertainties and limitations. For example, different judgments might give greater weight to
15 more uncertain aspects of the evidence or reflect a differing view with regard to public welfare
16 significance. Such judgments are left to the discretion of the Administrator. We note, however,
17 that the scientific evidence and quantitative air quality, exposure and risk information in the
18 record on which this reconsideration is based are largely unchanged. Staff conclusions regarding
19 the adequacy of the current standards thus remain unchanged from those reached in the 2020 PA.

20 In summary, the evidence characterized in the 2020 ISA is consistent with that available
21 in the 2015 review for the principal effects for which the evidence is strongest (e.g., plant
22 growth, reproduction, and related larger-scale effects, as well as visible foliar injury) and for key
23 aspects of the current standard. The evidence regarding RBL and air quality in areas meeting the
24 current standard does not appear to call into question the adequacy of public welfare protection
25 afforded by the standard. With regard to visible foliar injury, the currently available evidence for
26 forested locations across the U.S., such as studies of USFS biosites, does not indicate an
27 incidence of significant visible foliar injury that might reasonably be concluded to be adverse to
28 the public welfare under air quality conditions meeting the current standard. For the insect-
29 related effects that the ISA newly concludes likely to be causally related to O₃, the new
30 information does not support an understanding of the potential for the occurrence of such effects
31 in areas that meet the current standard to an extent that they might reasonably be judged
32 significant to public welfare. Thus, we do not find the current information for these newly
33 identified categories to call into question the adequacy of the current standard. Similarly, key
34 uncertainties recognized in the 2015 review remain in the evidence for O₃ contribution to
35 radiative forcing or effects on temperature, precipitation and related climate variables, including
36 specifically uncertainties that limit quantitative evaluations that might inform consideration of

1 these effects (as discussed above). Based on all of the above considerations, we conclude that the
2 currently available evidence and quantitative exposure/risk information does not call into
3 question the protection afforded by the current secondary standard, such that it is appropriate to
4 consider retaining the current standard without revision. In light of this conclusion, we have not
5 identified any potential alternative standards for consideration.

6 **4.6 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH**

7 In this section, we highlight key uncertainties associated with reviewing and establishing
8 the secondary O₃ standard and additionally recognize that research in these areas may
9 additionally be informative to the development of more efficient and effective control strategies.
10 The list in this section includes key uncertainties and data gaps thus far highlighted in this review
11 of the secondary standard. Additional information in several areas would reduce uncertainty in
12 our interpretation of the available information and, accordingly, reduce uncertainty in our
13 characterization of O₃-related welfare effects. For example, the items listed below generally
14 include uncertainties associated with the extrapolation to plant species and environments outside
15 of specific experimental or field study conditions and the assessment of ecosystem-scale impacts,
16 such as structure and function. Additional E-R studies in different species or for responses other
17 than reduced growth over multiple exposure conditions over growing seasons, that include
18 details on exposure circumstances (e.g., hourly concentrations throughout the exposure), and
19 exposure history, etc. would improve on and potentially expand characterizations of the potential
20 for and magnitude of the identified vegetation effects under different seasonal exposures.
21 Accordingly, in this section, we highlight areas for future welfare effects research, model
22 development, and data collection activities to address these uncertainties and limitations in the
23 current scientific evidence. These areas are similar to those highlighted in past reviews.

- 24 • While national visible foliar injury surveys have provided an extensive dataset on the
25 incidence of such effects at sites across the country that experienced differing cumulative
26 seasonal O₃ exposures and soil moisture conditions, there remain uncertainties in the
27 current understanding of the relationship between seasonal O₃ exposures (and other
28 influential factors, such as relative soil moisture) and the incidence and relative severity
29 of visible foliar injury. Further research investigating the role of peak concentrations, in
30 addition to cumulative seasonal exposures (particularly for W126 index values below 25
31 ppm) is also needed to improve consideration of the occurrence and variability of higher
32 hourly O₃ concentrations associated with vegetation effects. Research to better
33 characterize the relationship between O₃, soil moisture and foliar injury and specifically a
34 quantifiable relationship between these (and any other influential) factors. Additionally,
35 research would assist in interpreting connections between O₃-related foliar injury and
36 other physiological effects and ecosystem services. For example, research is needed on
37 the extent and severity of visible foliar injury that might impact ecosystem services (e.g.,
38 tourism), and the extent of impact it might have.

- 1 • Additional controlled exposure studies of effects, such as biomass impacts, that include
2 multiple exposure levels within the lower range of exposures associated with ambient air
3 quality conditions common today, extend over multiple years, and include the collection
4 of detailed O₃ concentration data over the exposure would reduce uncertainty in estimates
5 of effects across multiple-year periods and at the O₃ exposures common today. Also
6 needed is evaluation of such datasets with regard to the role of peak concentrations in
7 combination with that of cumulative seasonal exposures (e.g., as quantified by metrics
8 such as the W126 and SUM06 indices).
- 9 • Evidence newly available since the 2015 review includes studies on insect-plant
10 interactions that have established some statistically significant effects, but the evidence is
11 still limited with regard to discerning a pattern of responses in growth, reproduction, or
12 mortality, and a directionality of responses for most effects. More research is needed to
13 investigate the degree of response and directionalities of these relationships, and to
14 investigate potential effects on pollination. The evidence is also limited with regard to the
15 species represented (i.e., currently confined to three insect orders).
- 16 • Some evidence provides for linkages of effects on tree seedlings with larger trees and
17 similarities in results between exposure techniques. Uncertainties remain in this area as
18 well as uncertainties in extrapolating from O₃ effects on young trees (e.g., seedlings
19 through a few years of age) to mature trees and from trees grown in the open versus
20 those within the forest canopy.
- 21 • Uncertainties that remain in extrapolating individual plant response spatially or to higher
22 levels of biological organization, including ecosystems, could be informed by research
23 that explores and better quantifies the nature of the relationship between O₃, plant
24 response and multiple biotic and abiotic stressors, including those associated with the
25 affected ecosystem services (e.g., hydrology, productivity, carbon sequestration).
- 26 • Other uncertainties are associated with estimates of the effects of O₃ on the ecosystem
27 processes of water, carbon, and nutrient cycling, particularly at the stand and community
28 levels. These below- and above-ground processes include interactions of roots with the
29 soil or microorganisms, effects of O₃ on structural or functional components of soil food
30 webs and potential impacts on plant species diversity, changes in the water use of
31 sensitive trees, and if the sensitive tree species is dominant, potential changes to the
32 hydrologic cycle at the watershed and landscape level. Research on competitive
33 interactions under different O₃ exposures and any associated impacts on biodiversity or
34 genetic diversity would improve current understanding.
- 35 • Uncertainties related to characterizing the potential public welfare significance of O₃-
36 induced effects and impacts to associated ecosystem services could also be informed by
37 research. Research relating effects such as those on plant reproduction and propagation to
38 effects on production of non-timber forest products, and research to characterize public
39 preferences including valuation related to non-use and recreation for foliar injury, could
40 also help inform consideration of the public welfare significance of these effects.

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APPENDIX 2A

ADDITIONAL DETAILS ON DATA ANALYSIS PRESENTED IN SECTION 2.4

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2A.1 ANALYSES OF 8-HOUR CONCENTRATIONS

The analyses presented in section 2.4 of the main document are based on hourly O₃ concentration data from the EPA's Air Quality System (AQS) database (retrieved on August 12, 2021) for the years 2000 to 2020 for the sites meeting data completeness criteria as summarized in Table 2A-1 below. The daily maximum 8-hour (hr) average (MDA8) values, annual fourth highest MDA8 values, and design values (DVs) for the current standards were calculated according to Appendix U to 40 CFR Part 50. Those steps are generally as follows.

- 8-hr average concentrations are derived as the average of concentrations during eight consecutive hours for the:
 - o 8-hr periods which have at least six hourly concentrations;¹ and
 - o 8-hr periods which have fewer than six hourly concentrations and the sum of concentrations divided by eight, after truncation of the digits after the third decimal place, is greater than 0.070 parts per million (ppm)²
- The digits for the resultant 8-hr average concentration are truncated after the third decimal place.
- **MDA8 concentrations** are derived as the highest of the consecutive 8-hr averages beginning with the 8-hr period from 7am to 3pm and ending with the period from 11pm to 7am the following day for those days with:
 - o 8-hr concentrations for at least 13 of the 17 8-hr periods that begin with the 7am-to-3pm period and end with the 11pm-to-7am (next day) period, or
 - o 8-hr concentrations for fewer than 13 of the 17 8-hr periods if the maximum 8-hr concentration, after truncation of the digits after the third decimal place, is greater than 0.070 ppm.
- **Design Values** in ppm are derived as average of the annual 4th highest MDA8 concentrations in three consecutive years, with digits after the third decimal place truncated.
 - o Design values greater than 0.070 ppm are always considered valid.
 - o Design values less than or equal to 0.070 ppm must have MDA8 values for at least 90% of the days in the ozone monitoring season³, on average over the 3-year period, with a minimum of 75% of those days in any individual year.

¹ When there are at least six hours with a concentration reported, the 8-hr average is the average calculated using the number of hours with concentrations in the denominator.

² When there are fewer than six hours with a concentration reported, the 8-hr average is the average calculated using eight in the denominator and substituting zero for the missing hourly concentrations.

³ Ozone monitoring seasons are defined for each State in Table D-2 of Appendix D to 40 CFR Part 58.

1 **Table 2A-1. Summary of criteria describing the sites for which 8-hour metrics are**
 2 **presented in section 2.4 of main document.**

Presentation of 8-hour metrics in section 2.4	Time Period	Data included
Figure 2-8, DVs	2018-2020	Design values are presented for all sites with valid design values, which are sites having at least 75% data completeness in each of the three years and at least 90% completeness on average across the three years (per Appendix U)
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Figure 2-11, Trends	2000-2020	Annual fourth highest MDA8 values are based on all sites with at least 75% annual data completeness for at least 16 of the 21 years, with no more than two consecutive years having less than 75% complete data (n = 822 sites) Design values are presented for sites with valid DVs for at least 15 of the 19 3-year periods, with no more than two consecutive periods having invalid DVs (n = 658 sites)
Figure 2-12, Trends	2000-2020	
Figure 2-13, Diurnal Patterns	2015-2017	All hourly concentrations are presented for 2015-2017 for these four monitoring sites
Figure 2-14, Seasonal Pattern	2015-2017	All valid MDA8 values are presented for 2015-2017 for these four monitoring sites

3

4 **2A.2 ANALYSES OF 1-HOUR CONCENTRATIONS**

5 Figure 2-15 of Chapter 2 presents hourly concentrations available in AQS (at the time of
 6 the data query on August 12, 2021) from any site with such data during the 2018-2020 period.
 7 The daily maximum 1-hr (MDA1) values presented in section 2.4.5 and (summary statistics
 8 shown in Table 2A-2 below) were calculated according to Appendix H to 40 CFR Part 50 for all
 9 sites with valid 2018-2020 design values for the current 8-hour standards. Generally, MDA1
 10 values are derived (as the maximum 1-hr concentration during a day) for days for which at least
 11 18 hourly concentrations are available in AQS or for which at least one hourly concentration
 12 greater than 0.12 ppm has been reported in AQS. For this most recent design value period, the
 13 mean number of observations per site at or above 100 parts per billion (ppb) was well below one
 14 (0.22) for sites meeting the current standards compared to well above one (10.53) for sites not
 15 meeting the current standards.

16

1 **Table 2A-2. Summary statistics for MDA1 concentrations at sites with differing design**
 2 **values for 2018-2020.**

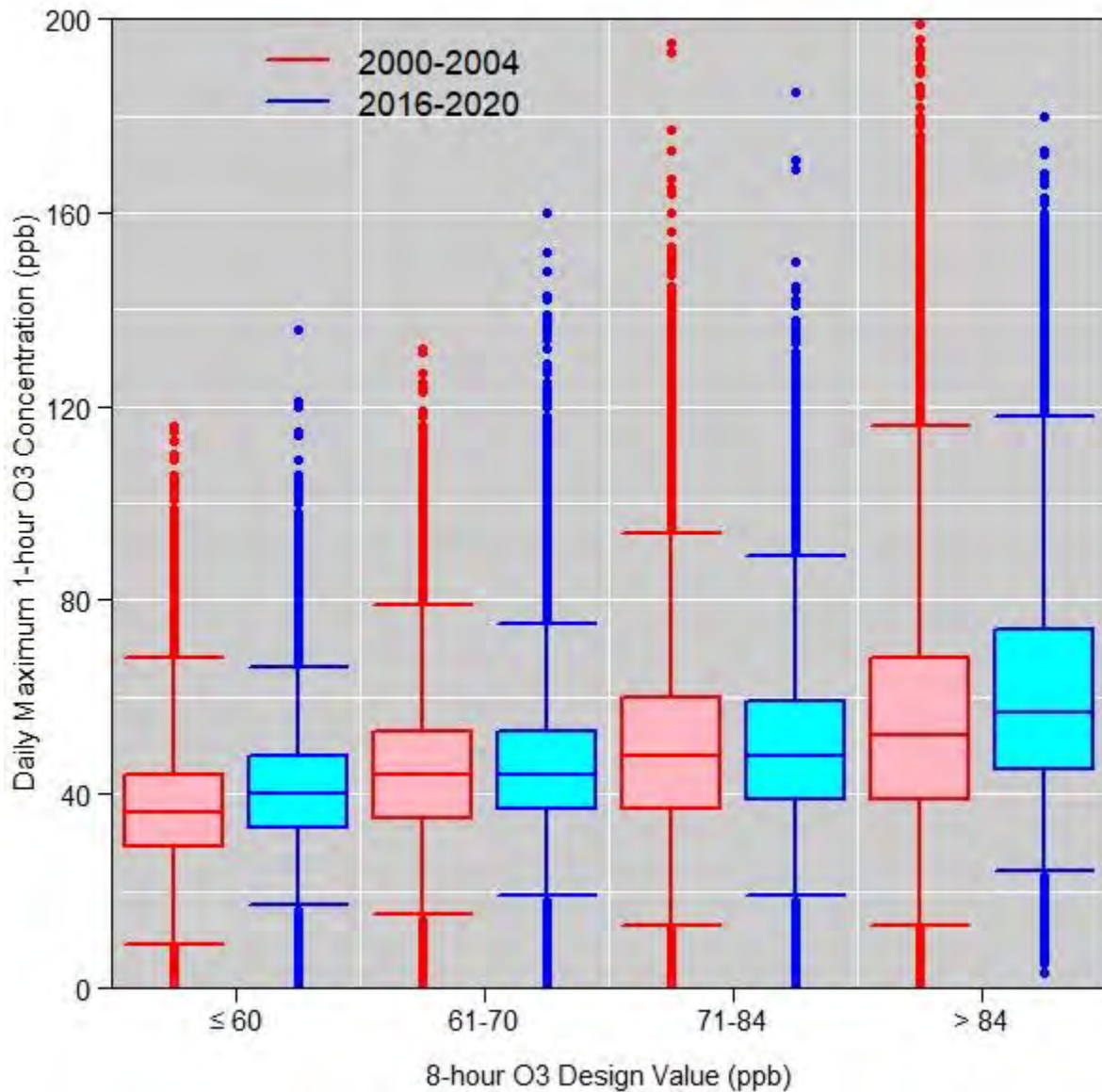
Statistic	Design Value (ppb)			
	31-60	61-70	71-84	85-114
Number of observations (obs)	261,302	554,712	164,988	27,958
Number of sites	287	590	170	26
25 th percentile concentration (ppb)	34	36	40	44
Median concentration (ppb)	40	44	48	57
Mean concentration (ppb)	40.7	44.5	49.7	61.2
75 th percentile concentration (ppb)	48	52	59	75
95 th percentile concentration (ppb)	58	65	76	101
99 th percentile concentration (ppb)	67	76	90	121
# of obs (# of sites) ≥ 240 ppb	0 (0)	0 (0)	0 (0)	0 (0)
# of obs (# of sites) ≥ 200 ppb	0 (0)	0 (0)	0 (0)	0 (0)
# of obs (# of sites) ≥ 160 ppb	0 (0)	0 (0)	4 (4)	14 (6)
# of obs (# of sites) ≥ 120 ppb	2 (2)	22 (17)	46 (29)	328 (21)
# of obs (# of sites) ≥ 100 ppb	15 (12)	180 (112)	526 (127)	1,538 (26)
Mean # of obs ≥ 100 ppb per site ^A	0.05	0.31	3.09	59.15

^A This is the number of obs at or above 100 ppb divided by the number of sites in this bin (column). For the two lowest bins combined (i.e., all sites with a design value ≤ 70 ppb), the mean is 0.22 obs ≥ 100 ppb per site, and for the two highest bins combined (i.e., all sites with a design value > 70 ppb), the mean is 10.53 obs ≥ 100 ppb per site.

3 The figures and tables presented below contain additional analyses based on the MDA1
 4 concentrations for years 2000-2004 and 2016-2020. Figure 2A-1 compares the distribution of
 5 MDA1 concentrations for each 8-hour design value bin between the earlier (2000-2004; red
 6 boxes) and latter (2016-2020; blue boxes) periods. The comparison shows a slight upward shift
 7 in the mid-range concentrations for the highest (≥ 85 ppb) and lowest (≤ 60 ppb) DV bins, while
 8 the two middle bins show little change. The range between the 1st and 99th percentiles as
 9 represented by the whiskers shrinks slightly between the earlier and latter periods in all four bins.
 10 Finally, the very highest concentrations (shown as dots above the top whisker) are reduced in the
 11 two highest DV bins. This is also reflected in Table 2A-3 and Table 2A-4, which show summary
 12 statistics similar to Table 2A-2 for the 2000-2004 and 2016-2020 periods, respectively. These
 13 tables show, as might be expected, that sites with higher design values have a larger number of
 14 days with MDA1 values at or above 100 ppb than sites with lower design values. This statistic is
 15 over 35 times higher for sites not meeting the current standard compared to sites meeting the
 16 current standard in 2000-2004, and over 45 times higher in 2016-2020. Across the three design
 17 value periods in 2016 to 2020, sites not meeting the current standards have on average over 9
 18 observations at or above 100 ppb per 3-year period, while the average for sites meeting the
 19 current standards is about 0.2.

20 Figure 2A-2 and Figure 2A-3 show maps of the average number of days where the
 21 MDA1 concentrations were greater than or equal to 100 ppb (also known as the D100 metric, see

1 Appendix 4F) for the 2000-2004 and 2016-2020 periods, respectively. These maps show that
2 nearly all sites in the U.S. have seen a large reduction in the number of days with high MDA1
3 concentrations since the beginning of the century. This is also reflected in the final rows of Table
4 2A-3 and Table 2A-4, which indicate a decrease of 83% in the total number MDA1 values
5 greater than or equal to 100 ppb between 2000-2004 and 2016-2020.



6
7 **Figure 2A-1. Boxplots comparing the distribution of MDA1 concentrations for 2000-2004**
8 **(red) to the distribution of MDA1 concentrations for 2016-2020 (blue),**
9 **binned by the 8-hour design value at each monitoring site. The boxes**
10 **represent the 25th, 50th and 75th percentiles and the whiskers represent the 1st and**
11 **99th percentiles. Outlier values are represented by circles.**

1 **Table 2A-3. Summary statistics for MDA1 concentrations at differing design values for**
 2 **2000-2004.**

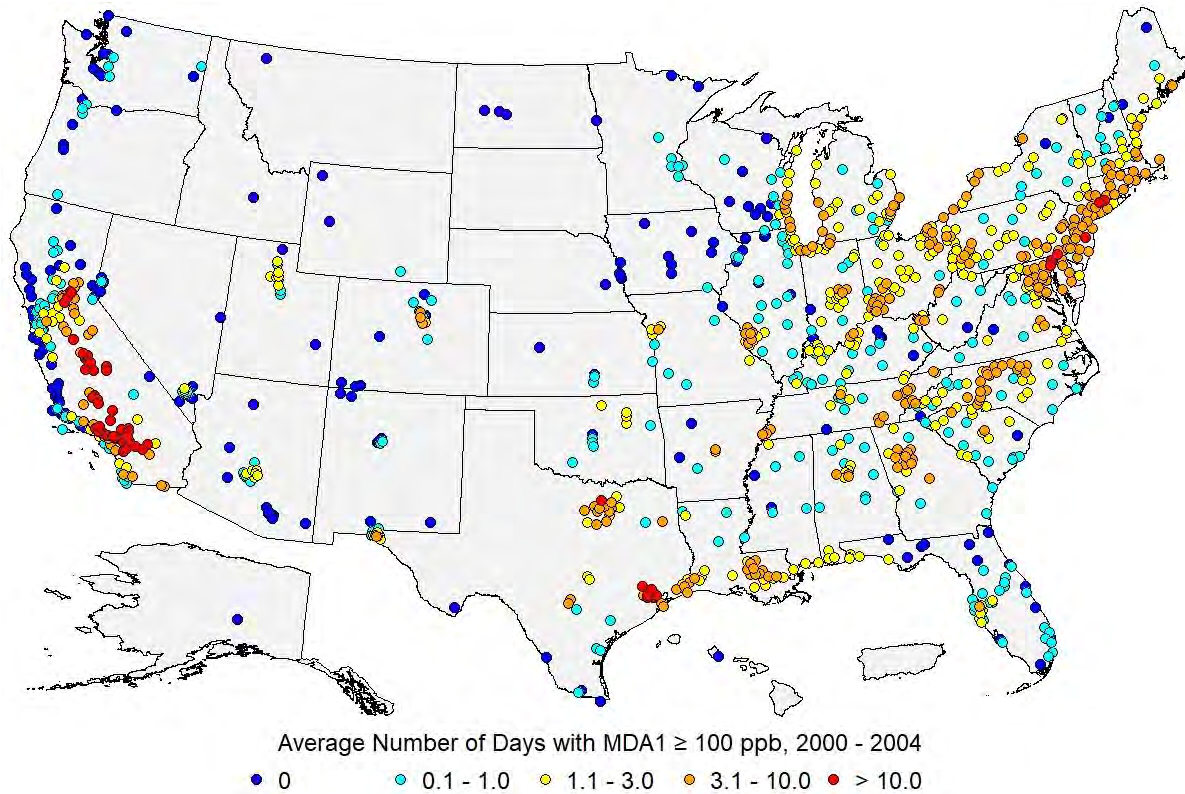
Statistic	Design Value (ppb)			
	35-60	61-70	71-84	85-131
Number of observations (obs)	117,848	288,396	1,312,716	912,178
Number of design values (DVs) ^A	130	313	1,518	1,151
25 th percentile concentration (ppb)	29	35	37	39
Median concentration (ppb)	36	44	48	52
Mean concentration (ppb)	36.5	44.3	49.3	54.8
75 th percentile concentration (ppb)	44	53	60	68
95 th percentile concentration (ppb)	56	68	79	95
99 th percentile concentration (ppb)	68	79	94	116
# of obs (# of DVs ^A) ≥ 240 ppb	0 (0)	0 (0)	0 (0)	0 (0)
# of obs (# of DVs ^A) ≥ 200 ppb	0 (0)	0 (0)	0 (0)	4 (4)
# of obs (# of DVs ^A) ≥ 160 ppb	0 (0)	0 (0)	15 (12)	252 (100)
# of obs (# of DVs ^A) ≥ 120 ppb	0 (0)	8 (6)	623 (339)	7,203 (940)
# of obs (# of DVs ^A) ≥ 100 ppb	26 (16)	161 (87)	7,078 (1,277)	32,133 (1,151)
Mean # of obs ≥ 100 ppb per DV ^B	0.20	0.51	4.66	27.92

^A Since this table covers three design value periods, individual sites may be counted up to three times.
^B This is the number of obs at or above 100 ppb divided by the number of site-DVs in this bin (column). For the two lowest bins combined (i.e., sites with a design value ≤ 70 ppb), the mean is 0.40 obs ≥ 100 ppb per site, and for the two highest bins combined (i.e., sites with a design value > 70 ppb), the mean is 14.69 obs ≥ 100 ppb per site.

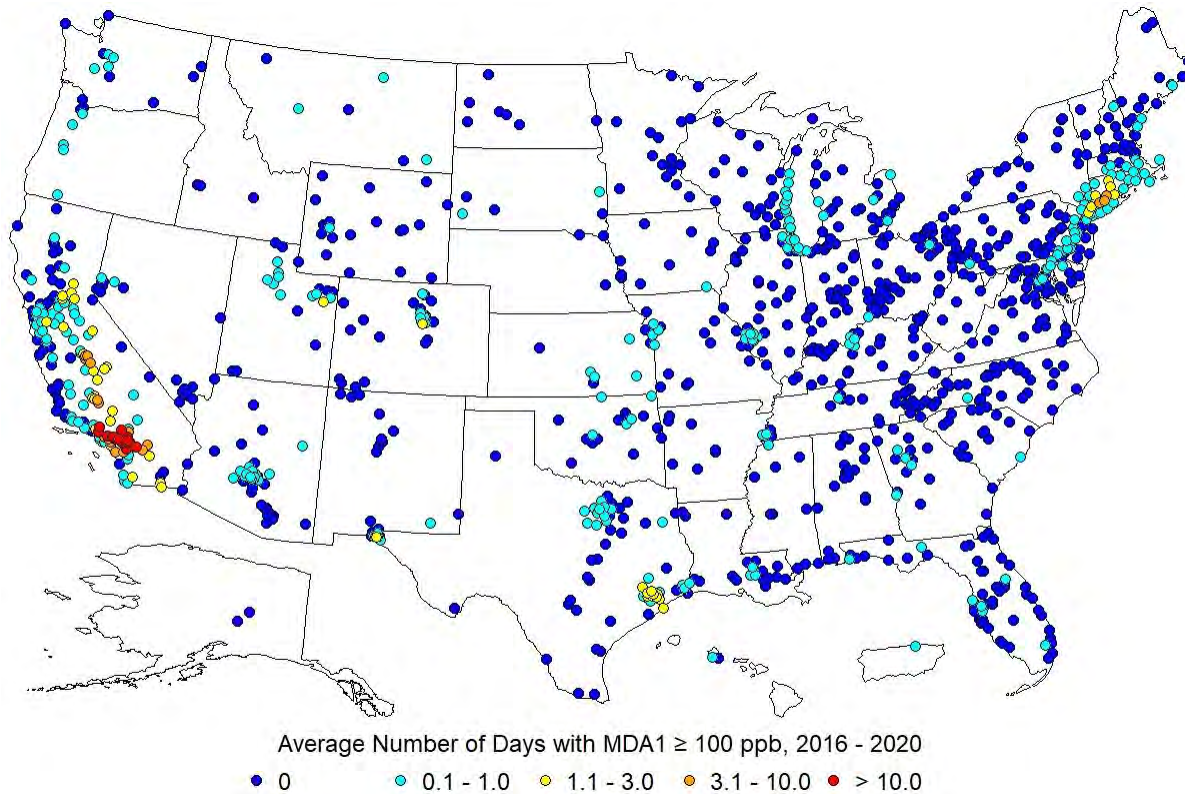
3
 4 **Table 2A-4. Summary statistics for MDA1 concentrations at differing design values for**
 5 **2016-2020.**

Statistic	Design Value (ppb)			
	29-60	61-70	71-84	85-114
Number of observations (obs)	582,220	1,824,438	558,927	99,742
Number of design values (DVs) ^A	637	1,969	579	93
25 th percentile concentration (ppb)	33	37	39	45
Median concentration (ppb)	40	44	48	57
Mean concentration (ppb)	40.6	44.8	49.2	60.6
75 th percentile concentration (ppb)	48	53	59	74
95 th percentile concentration (ppb)	58	65	76	99
99 th percentile concentration (ppb)	66	75	89	118
# of obs (# of DVs ^A) ≥ 240 ppb	0 (0)	0 (0)	0 (0)	0 (0)
# of obs (# of DVs ^A) ≥ 200 ppb	0 (0)	0 (0)	0 (0)	0 (0)
# of obs (# of DVs ^A) ≥ 160 ppb	0 (0)	1 (1)	4 (4)	15 (7)
# of obs (# of DVs ^A) ≥ 120 ppb	8 (6)	51 (42)	101 (77)	904 (69)
# of obs (# of DVs ^A) ≥ 100 ppb	41 (32)	486 (335)	1,591 (423)	4,761 (93)
Mean # of obs ≥ 100 ppb per DV ^B	0.06	0.25	2.75	51.19

^A Since this table covers three design value periods, individual sites may be counted up to three times.
^B This is the number of obs at or above 100 ppb divided by the number of site-DVs in this bin (column). For the two lowest bins combined (i.e., sites with a design value ≤ 70 ppb), the mean is 0.20 obs ≥ 100 ppb per site, and for the two highest bins combined (i.e., sites with a design value > 70 ppb), the mean is 9.45 obs ≥ 100 ppb per site.

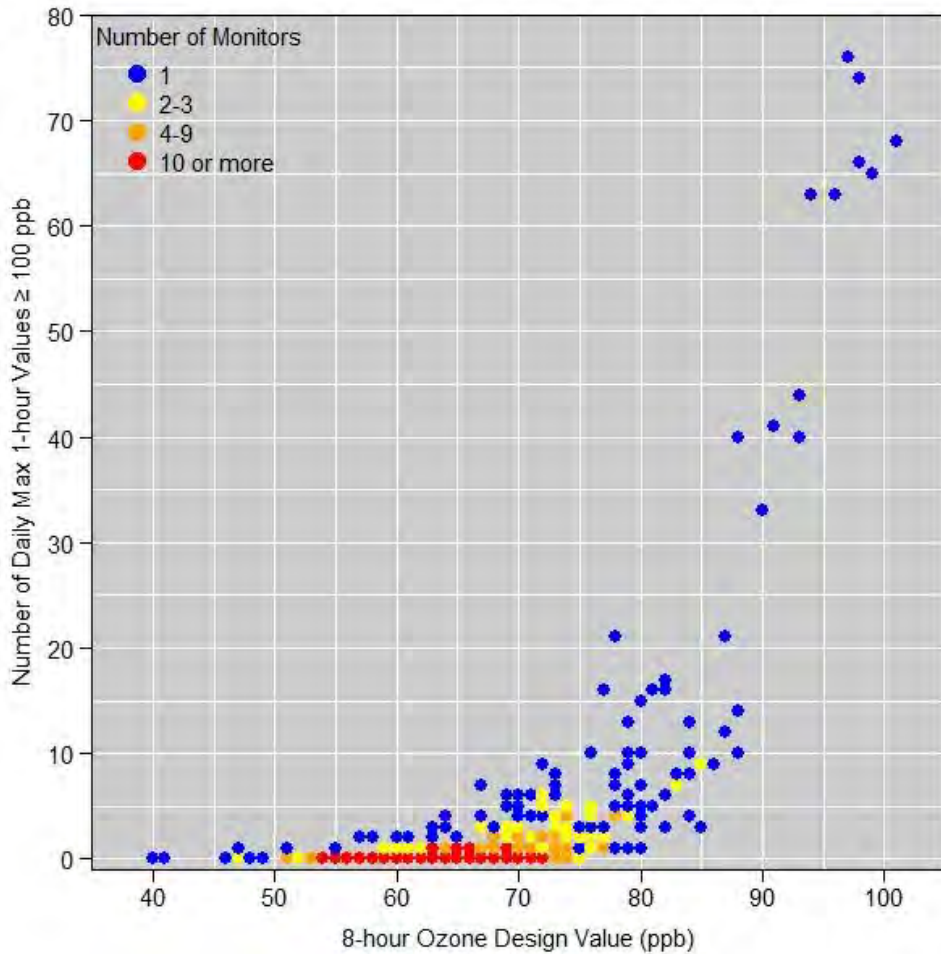


1
2 **Figure 2A-2. Map showing the average number of days with MDA1 \geq 100 ppb, 2000-2004.**



3
4 **Figure 2A-3. Map showing the average number of days with MDA1 \geq 100 ppb, 2016-2020.**

1 Figure 2A-4 below shows the number of days in 2018-2020 with an MDA1 concentration
2 at or above 100 ppb and 8-hour design values (similar to Figure 2-16), for all sites with a 2018-
3 2020 design value less than 102 ppb. All sites meeting the current standard had seven or fewer
4 (i.e., two or fewer per year) MDA1 values at or above 100 ppb, and all but eight sites meeting
5 the current standard had three or fewer (i.e., one or fewer per year) MDA1 values at or above
6 100 ppb.



7

8 **Figure 2A-4. Number of days in 2018-2020 at each monitoring site with a MDA1**
9 **concentration greater than or equal to 100 ppb and an 8-hour design value**
10 **less than 102 ppb.** Sites with higher design values had more days, up to a
11 maximum of 173 (at a site in southern CA with a design value of 114 ppb).

12

13

1 **APPENDIX 2B**

2 **ADDITIONAL DETAILS ON BACKGROUND OZONE**
3 **MODELING AND ANALYSIS**

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24 statistics for MDA8 O₃ by NOAA climate region, by season and monitoring
25 Network. 2B-15

26

27

28

29

1 This appendix for the background ozone (O₃) modeling and analysis, which presents the
2 analysis that was also presented in Appendix 2B of the 2020 PA (and is virtually identical to that
3 appendix), includes a description of the methodology for photochemical modeling, an evaluation
4 of the modeling, and a more detailed analysis of the predicted contributions from international
5 anthropogenic emissions. The methodology section includes a description of the modeling
6 platform and emissions. The evaluation section includes comparisons against surface, sondes and
7 satellite measurements. The international component analysis separately estimates O₃ impacts
8 from China, India, Canada/Mexico, and global shipping at the hemispheric scale.

9 **2B.1 PHOTOCHEMICAL MODELING METHODOLOGY**

10 **2B.1.1 Modeling Platform Overview**

11 A multiscale modeling system is applied at both hemispheric and regional scales with
12 consistent methodologies for emissions inputs, meteorological inputs, model chemistry, and
13 photochemical models. Consistency across spatial scales reduces the number of assumptions that
14 have to be made in integrating predictions from the global and the regional modeling. However,
15 methodological consistency does not address sources of uncertainty associated with individual
16 inputs used by the modeling system.

17 The modeling system uses one emission model, one meteorological model, and one
18 chemical transport model. The meteorological model is the Weather Research and Forecasting
19 model (WRF v3.8). The emissions model is the Sparse Matrix Operating Kernel for Emissions
20 (SMOKE v4.5). The chemical transport model is the Community Multiscale Air Quality model
21 (CMAQ) version 5.2.1 with the Carbon Bond mechanism (CB6r3) and the non-volatile aerosol
22 option (AE6). Each of these models is applied at hemispheric and regional scales. The regional
23 meteorology components of the modeling system are described in more detail in section 3C.4.1.4
24 of Appendix 3C, while emissions inputs are summarized here.

25 The models identified above are configured differently for the hemispheric and regional
26 scales as appropriate for the intended purpose. The hemispheric scale model uses a polar
27 stereographic projection at 108 kilometer (km) resolution to completely and continuously cover
28 the Northern Hemisphere. At the regional scale, the model employs a Lambert conic conformal
29 projection at 36 km resolution to cover North America and at 12 km resolution to cover the
30 lower 48 contiguous states. The hemispheric scale allows for long-range free tropospheric
31 transport with 44 layers between the surface and 50 hPa (~20 km asl). The 36 km and 12 km
32 regional modeling has 35 vertical layers between the surface and 50 hPa. The hemispheric
33 modeling system was initiated on May 1, 2015 and run continuously through December 31,
34 2016. The regional model was initialized using the hemispheric result on December 21, 2015 and
35 run continuously through December 31, 2016.

1 **2B.1.2 Emissions Overview**

2 The emissions inventories are summarized here and more information is available in the
3 Emissions Technical Support Documents (U.S. EPA, 2019a, U.S. EPA, 2019b) and in Appendix
4 3C. The emissions model inputs are discussed separately for natural and anthropogenic
5 emissions. The stratospheric fluxes (section 2.5.1.1 of main document) are not discussed here
6 because, although they are a source of ozone, they are not emissions. The regional inventories
7 over North America are based on the Inventory Collaborative 2016 emissions modeling platform
8 (<http://views.cira.colostate.edu/wiki/wiki/9169>), which was developed through the summer of
9 2019. Three versions of the 2016 inventory developed: “alpha” (also known as the 2016v7.1
10 platform) – which consisted of data closely related to the 2014 National Emissions Inventory
11 (NEI) version 2 and 2016-specific data for some sectors; “beta” (also known as the 2016v7.2
12 platform) – which incorporated data from state and local agencies and adjustments to better
13 represent the year 2016; and “version 1” (also known as the 2016v7.3 platform) – which has the
14 completed representation of 2016 and some elements from the 2017 NEI. For any regional
15 inventories, this analysis used the 2016 “alpha release” (specifically the modeling case
16 abbreviated 2016fe) that is publicly available from [https://www.epa.gov/air-emissions-](https://www.epa.gov/air-emissions-modeling/2016-alpha-platform)
17 [modeling/2016-alpha-platform](https://www.epa.gov/air-emissions-modeling/2016-alpha-platform). Any changes in the 2016 “beta” or “version 1” platforms are not
18 included in this modeling and therefore are not captured in the subsequent analysis.

19 **2B.1.2.1 Natural Emission Inventory**

20 The natural emission inventory databases cover all the sources discussed in section 2.5.1
21 except the International Anthropogenics. The databases that are available depend upon the scale.
22 At the global scale, lightning NO_x emissions are based on monthly climatological data; biogenic
23 VOC emissions have hourly and day-specific (MEGAN v2.1, Guenther et al., 2012) temporal
24 scales; soil NO_x also has hourly and day-specific temporal scales (Berkeley Dalhousie Soil NO_x
25 Parameterization, as implemented by Hudman et al., 2012); and fire emissions are based on day-
26 specific data (FINN v1.5, Wiedinmyer et al., 2011). Over our regional domain, regional
27 inventories supersede the biogenic VOCs, soil NO_x, and fire emissions using estimates
28 consistent with the 2016 collaborative emissions modeling platform ([https://www.epa.gov/air-](https://www.epa.gov/air-emissions-modeling/2016-alpha-platform)
29 [emissions-modeling/2016-alpha-platform](https://www.epa.gov/air-emissions-modeling/2016-alpha-platform)). The regional biogenic VOCs and soil NO_x are
30 derived from the Biogenic Emission Inventory System (BEIS v3.61). Of the natural inventories,
31 only fires are expected to change significantly in future versions of the 2016 emissions platform.
32 The biogenic VOC and NO_x changes will be minor due to small changes to the land use data
33 input to BEIS3.

1 Emissions of NO_x are of particular importance to this study and the natural inventory is
2 summarized here. The total natural NO_x emissions⁴ in this platform is 56 megatons NO_x
3 (reported as equivalent NO₂ mass) which is approximately 15.5 TgN. The contributors in order
4 of magnitude are lightning (55%), soil (33%), and wildfires (12%). Lightning is treated as a
5 climatological monthly mean contribution, while soils and wildfires are day-specific. It is
6 important to note that outside North America, prescribed fires are not identified distinctly from
7 wildfires. Therefore, all wildland fires outside North America are treated as natural. Though not
8 directly comparable, the lightning and soil magnitudes are consistent with the ranges reported by
9 (Lamarque et al., 2012). Consistent with previous regional modeling platforms, the lightning
10 emissions are not included in the emissions inputs to the regional modeling platform. At the
11 regional scale, the representation of lightning as a monthly mean rate would add lightning on
12 days where it may not have occurred. At the hemispheric scale, omitting lightning would remove
13 an important contribution to the well-mixed background O₃.

14 **2B.1.2.2 Anthropogenic Emission Inventory**

15 Anthropogenic emissions inputs include both domestic and international sources. The
16 domestic inventory includes a high-level of detail that is consistent with previous EPA emissions
17 platforms such as those used to model the year 2011 ([https://www.epa.gov/air-emissions-
18 modeling/2011-version-6-air-emissions-modeling-platforms](https://www.epa.gov/air-emissions-modeling/2011-version-6-air-emissions-modeling-platforms)). For the hemispheric emissions
19 modeling platform, there are over thirty anthropogenic sector of emission files. The traditional
20 regional platform covers North America including the U.S. sectors, Canadian sectors, and
21 Mexican sectors. In addition to the typical regional platform sectors, there are nine sectors based
22 on the Hemispheric Transport of Air Pollution Version 2 (EDGAR-HTAPv2) inventory and 15
23 sectors that represent emissions in China which together comprise the anthropogenic emissions
24 outside of North America. The international emission inventories are synthesized from the
25 EDGAR-HTAP v2 harmonized emission inventory and country specific databases where updates
26 were likely to be influential. Previous assessments like HTAP (2010, Phase 1) and HTAP (Phase
27 2) have shown that the anthropogenic portion of USB is most sensitive to emissions in Mexico,
28 Canada, and China. For Mexico and Canada, the hemispheric platform relies on the same
29 country-specific databases as the regional platform. For China, as mentioned above, the
30 hemispheric platform uses a new country specific database. The sources are detailed further
31 below.

32 The EDGAR-HTAP v2 inventories were projected to represent the year 2014. Projection
33 factors were calculated from the Community Emissions Data System (CEDS) inventory at a

⁴ We refer to wildfires and soil NO_x as natural for the purposes of this section even though both may be impacted to various degrees by human activity.

1 country-sector level. This allowed our inventory to evolve without the risks associated with
2 transitioning to a new inventory system. Especially because EDGAR-HTAP v2 is superseded for
3 critical counties, this was the optimal approach. Details of scaling factor development are
4 described in Section 2.1.5 of the 2016v7.1 Hemispheric Modeling Platform Technical Support
5 Document (U.S. EPA, 2019a).

6 Emissions estimates over Mexico are a combination of emissions supplied by the
7 Mexican government and emissions developed by the EPA. For the 2016 platform, emissions for
8 point, nonpoint, and nonroad sources were developed based on projections of Secretariat of
9 Environment and Natural Resources (SEMARNAT)-supplied data for the year 2008. For the
10 onroad mobile sources, the EPA developed year-specific inventories for 2014 and 2017 by
11 applying the MOVES-Mexico model and interpolating to the year 2016. More details are
12 available in the 2016v7.1 emissions platform TSD (U.S. EPA, 2019b).

13 Emissions for Canada were supplied by Canadian agencies and reprocessed by the EPA
14 for the domains and model years used in this analysis. Environment and Climate Change Canada
15 (ECCC) supplied data for four broad inventory sectors (point, on-road mobile, fugitive dust, and
16 area and non-road mobile sources, the latter including commercial marine vessels). The ECCC
17 emissions were interpolated to 2016 based on inventories from the years 2013 and 2025.

18 The China emission inventory was developed at Tsinghua University (THU) and
19 documented in Zhao et al., 2018 (see supplement). This inventory was extensively compared to
20 the EDGAR-HTAP v2 and EDGAR v4.3 inventories before use. The largest differences for NO_x
21 in 2016 occurred in individual emissions sectors rather than inventory totals. The SO₂ emissions
22 were more different than NO_x emissions between the two inventories because the THU
23 inventory applies controls to the metal industry that have been adopted by China. The difference
24 between emissions, primarily NO_x emissions, causes small decrease in the spring time surface
25 O₃ over the U.S. compared to using EDGAR-HTAP v2. Comparisons of this update are
26 summarized by Henderson et al.(2019).

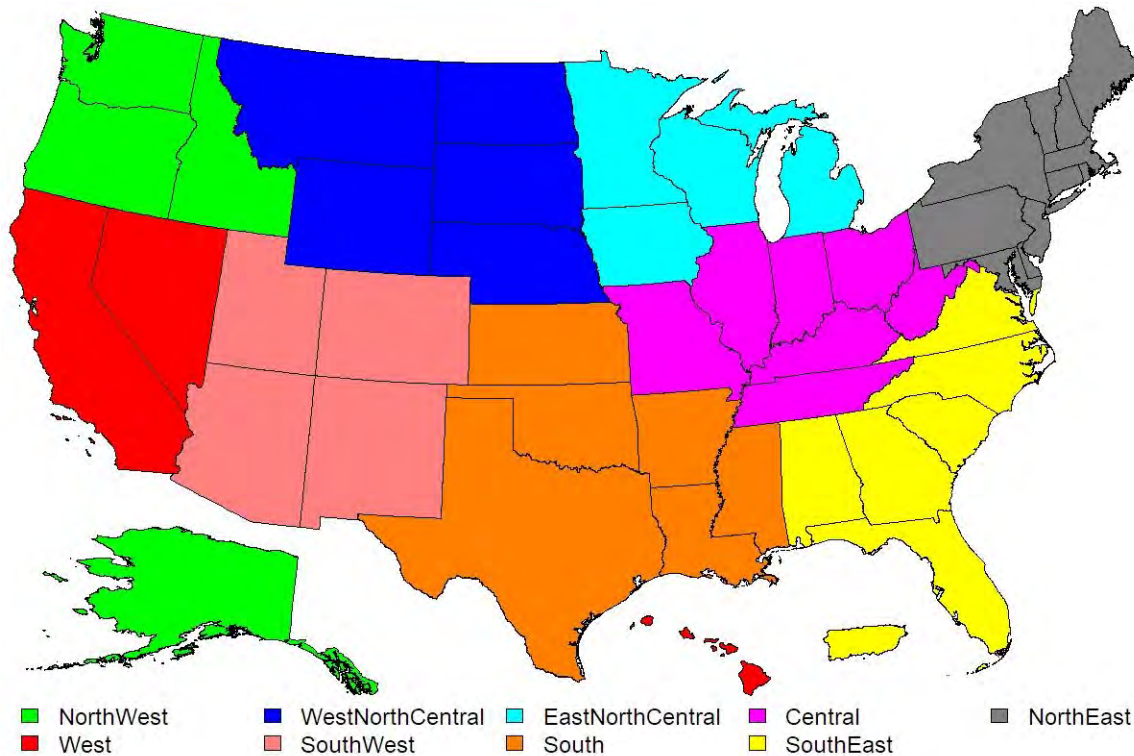
27 Emissions for the United States representing the year 2016 were developed using the
28 2014 National Emissions Inventory version 2 (2014NEIv2) as the starting point, although
29 emissions for some data categories were updated to better represent the year 2016. The point
30 source emission inventories for the platform are partially updated to represent 2016. Because
31 2016 is not a year for which a full NEI is compiled, states are only required to submit emissions
32 for their larger point sources. For units without 2016-specific emissions, the emissions were
33 carried forward from the 2014 NEIv2. For electric generating units, 2016-specific Continuous
34 Emissions Monitoring System (CEMS) data are used where the data can be matched to units in
35 the NEI. Point and nonpoint oil and gas emissions were projected from 2014 to 2016 using
36 factors based on historic production levels.

1 Other sectors are briefly summarized here and the reader is directed to the TSD for more
2 details (U.S. EPA, 2019a). Agricultural and wildland (including prescribed) fire emissions were
3 developed for the year 2016 using methods similar to those used to develop the 2014 NEI, except
4 that the input data relied on nationally-available data sets and did not benefit from state-
5 submitted data as are used for NEI year emissions. The assignment of wildland fires to wild or
6 prescribed is a complex process that is documented in the regional platform emissions TSD (U.S.
7 EPA, 2019b). Most area source sectors for this platform use unadjusted 2014 NEIv2 emissions
8 estimates except for commercial marine vehicles (CMV), fertilizer emissions, oil and gas
9 emissions, and onroad and nonroad mobile source emissions. For CMV, SO₂ emissions were
10 updated to reflect new rules for the North American Emission Control Area (regulation 13.6.1
11 and appendix VII of MARPOL Annex VI) on sulfur emissions that took effect in the year 2015.
12 For fertilizer ammonia emissions, a 2016-specific emissions inventory is used in this platform,
13 while animal ammonia emissions were the same as those in 2014 NEIv2. Onroad and nonroad
14 emissions were developed based on MOVES2014a outputs for the year 2016, and the activity
15 data used to compute the onroad emissions were projected from 2014 to 2016 based on distinct
16 state-specific factors for urban and rural roads. Emissions from 2014 NEIv2 were used directly
17 for residential wood combustion, fugitive dust, and other nonpoint sources, although
18 meteorological-based adjustments for dust sources and temporal allocation for residential wood
19 and agricultural ammonia sources were based on 2016 meteorology. Additional details on the
20 development of the U.S., Canada, and Mexico emissions are provided in the 2016v7.1 (U. S.
21 EPA, 2019b).

22 **2B.2 EVALUATION**

23 An operational model performance evaluation for O₃ was conducted for the 2016fe
24 simulation (as referred to in Section 2.5.2.2) using monitoring data, ozone sonde data, and
25 satellite data in order to estimate the ability of the CMAQv5.2.1 modeling system to replicate the
26 2016 base year O₃ concentrations for the 12 km continental U.S. domain and the 108 km
27 Northern Hemispheric domain. The purpose of this evaluation is to examine the ability of the
28 2016 air quality modeling platform to represent the magnitude and spatial and temporal
29 variability of measured (i.e., observed) O₃ concentrations within the modeling domain. The
30 model evaluation for O₃ focuses on comparisons of model-predicted 8-hour daily maximum
31 concentrations (MDA8) to the corresponding concentrations from monitoring data (for 2016)
32 collected at monitoring sites in the AQS. The evaluation divided these data into two datasets, one
33 limited to only CASTNET sites (described in section 2.3.1), and the second comprised of all
34 other sites. We refer to this second dataset as “AQS.”

1 Included in the evaluation are statistical measures of model performance based upon
 2 model-predicted versus observed MDA8 O₃ concentrations that were paired in space and time.
 3 Statistics were generated for each of the nine National Oceanic and Atmospheric Administration
 4 (NOAA) climate regions of the 12-km U.S. modeling domain (Figure 2B-1). The regions include
 5 the Northeast, Central, EastNorthCentral, Southeast, South, Southwest, WestNorthCentral,
 6 Northwest and West as were originally identified in Karl and Koss (1984). Note that most
 7 monitoring sites in the West region are located in California, therefore statistics for the West will
 8 be mostly representative of California O₃ model performance.



Source: <http://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php#references>

Figure 2B-1. NOAA U.S. climate regions.

12 For MDA8 O₃, model performance statistics were calculated for each climate region by
 13 season and for the May through September O₃ season of 2016. Seasons were defined as: winter
 14 (December-January-February), spring (March-April-May), summer (June-July-August), and fall
 15 (September-October-November). Observational data were excluded from the analysis and model
 16 evaluations for sites that did not meet a 75% completeness criterion.⁵ In addition to the
 17 performance statistics, several graphical presentations of model performance were prepared for
 18 MDA8 O₃ concentrations. These graphical presentations include:

⁵ Each monitoring site had to have 75% of MDA8 values within any seasonal subset to be included in that subset.
 Thus individual monitors may be included in one evaluation of season, but not another.

- 1 (1) density scatter plots of observations obtained from the AQS system excluding CASTNET
2 (hereafter AQS) and predicted MDA8 O₃ concentrations for May through September;
- 3 (2) regional maps that show the mean bias and error as well as normalized mean bias and
4 error calculated for MDA8 ≥ 60 ppb for May through September at individual AQS and
5 CASTNET monitoring sites;
- 6 (3) tile plots that show normalized mean bias (%) and mean bias (ppb) of MDA8 and MDA8
7 ≥ 60 ppb by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring
8 sites;
- 9 (4) O₃ sonde evaluations comparing vertically resolved ozone model predictions to ozone
10 sondes measurements from the World Ozone and Ultraviolet Data Centre (*woudc.org*).
- 11 (5) satellite evaluation comparing simulated tropospheric vertical column densities of O₃,
12 nitrogen dioxide, and formaldehyde to OMI retrievals.

13 The Atmospheric Model Evaluation Tool (AMET) was used to calculate the model
14 performance statistics used in this evaluation (Gilliam et al., 2005). For this evaluation of the O₃
15 predictions in the 2016fe CMAQ modeling platform, we have selected the mean bias, mean
16 error, normalized mean bias, and normalized mean error to characterize model performance,
17 statistics which are consistent with the recommendations in Simon et al. (2012) and the
18 photochemical modeling guidance (U.S. EPA, 2018).

19 Mean bias (MB) is used as average of the difference (predicted – observed) divided by
20 the total number of replicates (*n*). Mean bias is defined as:

21
$$MB = \frac{1}{n} \sum_1^n (P - O)$$
, where P = predicted and O = observed concentrations for every site
22 and day included in the evaluation.

23 Mean error (ME) calculates the absolute value of the difference (predicted - observed)
24 divided by the total number of replicates (*n*). Mean error is defined as:

25
$$ME = \frac{1}{n} \sum_1^n |P - O|$$

26 Normalized mean bias (NMB) is used as a normalization to facilitate a range of
27 concentration magnitudes. This statistic averages the difference (predicted - observed) over the
28 sum of observed values. NMB is a useful model performance indicator because it avoids
29 overinflating the observed range of values, especially at low concentrations. Normalized mean
30 bias is defined as:

1
$$\text{NMB} = \frac{\sum_1^n (P - O)}{\sum_1^n (O)} * 100, \text{ where } P = \text{predicted concentrations and } O = \text{observed}$$

2 Normalized mean error (NME) is also similar to NMB, where the performance statistic is
3 used as a normalization of the mean error. NME calculates the absolute value of the difference
4 (model - observed) over the sum of observed values. Normalized mean error is defined as

5
$$\text{NME} = \frac{\sum_1^n |P - O|}{\sum_1^n (O)} * 100$$

6
7 As described in more detail below, the model performance statistics indicate that the
8 MDA8 O₃ concentrations predicted by the 2016 CMAQ modeling platform closely reflect the
9 corresponding monitoring data-based MDA8 O₃ concentrations in space and time in each region
10 of the U.S. modeling domain. The acceptability of model performance was judged for the 2016
11 CMAQ O₃ performance results considering the range of performance found in recent regional O₃
12 model applications (NRC, 2002; Phillips et al., 2008; Simon et al., 2012; U.S. EPA, 2009; U.S.
13 EPA, 2018). These other modeling studies represent a wide range of modeling analyses that
14 cover various models, model configurations, domains, years and/or episodes, chemical
15 mechanisms, and aerosol modules. Overall, the 2016 CMAQ O₃ model performance results are
16 within the range found in other recent peer-reviewed and regulatory applications. The model
17 performance results, as described in this document, demonstrate the predictions from the 2016
18 modeling platform closely replicate the corresponding observed concentrations in terms of the
19 magnitude, temporal fluctuations, and spatial differences for 8-hour daily maximum O₃.

20 The model performance bias and error statistics for MDA8 O₃ predictions in each of the
21 nine NOAA climate regions and each season are provided in Table 2B-1. As noted above, seasons
22 were defined as: winter (December-January-February), spring (March-April-May), summer
23 (June-July-August), and fall (September-October-November). As indicated by the statistics in
24 Table 2-7, mean bias and error for 8-hour daily maximum O₃ are relatively low in each
25 subregion, not only in the summer when concentrations are highest, but also during other times of
26 the year. Generally, MB for MDA8 O₃ ≥ 60 ppb is less than ± 10 ppb. Generally, MDA8 O₃ at the
27 AQS sites in the summer and fall is over predicted except in the Southwest, with the greatest over-
28 prediction in the EastNorthCentral and WestNorthCentral. Likewise, MDA8 O₃ at the
29 CASTNET sites in the summer and fall is typically over predicted except in the West, Southwest
30 and WestNorthCentral where the bias shows an under-prediction. In the winter and spring,

1 MDA8 O₃ is under predicted at AQS and CASTNET sites in all the climate regions (with NMBs
2 less than approximately ± 25 percent in each subregion).

3 Figure 2B-2 and Figure 2B-3 are tile plots that summarize to provide an overview of
4 model performance by region and by season. Figure 2B-2 shows NMB (%) and MB (ppb) of
5 MDA8 by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites.
6 Likewise, Figure 2B-3 shows the NMB (%) and MB (ppb) of MDA8 ≥ 60 ppb by NOAA climate
7 region (y-axis) and by season (x-axis) at AQS monitoring sites. Figure 2B-2 shows that for the
8 majority of the nine climate regions throughout each year the NMB is within ± 10 percent. There
9 is greater over-prediction ($< 20\%$) during the fall in the South, EastNorthCentral (*aka* Upper
10 Midwest), and Central (*aka* Ohio Valley) regions and during the summer in the South, Southeast
11 and Central (*aka* Ohio Valley) regions. However, there is greater under-prediction (up to 30
12 percent) during the winter in the Northwest, Southwest, WestNorthCentral (*aka*
13 NRockiesPlains), EastNorthCentral (*aka* Upper Midwest), Central (*aka* Ohio Valley), and
14 Northeast regions as well during the spring in the Northwest.

15 The density scatterplots in Figure 2B-4 to Figure 2B-12 provide a qualitative comparison
16 of model-predicted and observed MDA8 O₃ concentrations for each climate region by season. In
17 these plots the intensity of the colors indicates the density of individual observed/predicted
18 paired values. The greatest number of individual paired values is denoted by locations in the plot
19 denoted in warmer colors. The plots indicate that the predictions correspond closely to the
20 observations in that a large number of observed/predicted paired values lie along or close to the
21 1:1 line shown on each plot. The model is more likely to over-predict the observed values at low
22 and mid-range concentrations generally < 60 ppb in each of the regions. There are some
23 relatively infrequent very large over predictions at high concentrations. Preliminary review of
24 these biases finds that some are related to fire impacts.

25 Spatial plots of the MB, ME, NMB and NME for individual monitors are shown in Figure
26 2B-13 through Figure 2B-16, respectively. The statistics shown in these two figures were
27 calculated over the May through September period, using data pairs on days with observed 8-hr
28 O₃ of greater than or equal to 60 ppb. Model bias at individual sites during the O₃ season is
29 similar to that seen on a sub-regional basis for the summer. Figure 2B-13 shows the mean bias
30 for 8-hr daily maximum O₃ greater than 60 ppb is under predicted overall, but generally within
31 ± 10 ppb across the AQS and CASTNET sites. The greatest exceptions are most evident at certain
32 near-coastal sites where, on average, the model over predicts MDA8 observed O₃ ≥ 60 ppb.
33 Likewise, the information in Figure 2B-15 indicates that the normalized mean bias for days with
34 observed 8-hr daily maximum O₃ greater than 60 ppb is within $\pm 10\%$ at the vast majority of
35 monitoring sites across the U.S. domain. Model error, as seen from Figure 2B-14 and Figure 2B-
36 16, is generally 2 to 10 ppb and 20 percent or less at most of the sites across the U.S. modeling

1 domain. Somewhat greater error is evident at sites in several areas most notably in the West,
2 WestNorthCentral, Northeast, EastNorthCentral, Southeast, and along portions of the Gulf Coast
3 and Great Lakes coastlines.

4 Sonde evaluations are shown for the 108 km Northern Hemisphere domain in Figure 2B-
5 18 through Figure 2B-21. The sondes used in this analysis and their release frequencies are
6 shown in Figure 2B-17. Figure 2B-18 shows that the annual mean prediction is generally within
7 20% of the measured sonde data, except for near the tropopause. Figure 2B-19 shows that the
8 performance of all sites is generally not as good in the spring (March, April, May) than in the
9 summer (June, July, August). The seasonal performance of each monitor is shown in Figure 2B-
10 20 for spring and Figure 2B-21 for summer. By comparison, Figure 2B-20 shows that low biases
11 extend deeper into the troposphere in spring than in summer. The structure of the bias seems to
12 suggest a stratospheric causal mechanism because the bias is near the tropopause.

13 Satellite evaluations in this analysis include tropospheric vertical columns of O₃, nitrogen
14 dioxide (an ozone precursor as described in chapter 2), and formaldehyde (a VOC reaction
15 product which is an indicator of VOCs and total reactivity of the atmosphere). At this time, only
16 formaldehyde comparison includes the application of the scattering weights and air mass factor
17 to the model, which are often used to create an averaging kernel. Similar processing for O₃ and
18 NO₂ was not available at the time this appendix was completed. Satellite evaluations focus
19 exclusively on the 108 km results over the Northern Hemisphere.

20 Simulated O₃ tropospheric vertical column densities are compared to the O₃ product
21 described and evaluated by Huang et al. (2017). Figure 2B-22 and Figure 2B-23 compares the
22 model to the retrieved column data without application of the averaging kernel. Omitting the
23 averaging kernel introduces some error into the comparison (Huang et al., 2017; see Figure 9 for
24 details). Even so, the comparison shows reasonable performance within the mid-latitudes. There
25 is a notable low bias in January mid-latitudes and near the north pole in April. In addition, high
26 biases are consistently seen near the corners of the domain in January and April. This cause of
27 this high-bias pattern will require further analysis. Within the mid latitudes, the model is
28 performing well with notable low biases in January and scattered high biases in Asia in July.
29 Given the limitations of the comparison, the performance is quite good.

30 Simulated nitrogen dioxide (NO₂) vertical columns are compared is the OMNO2d
31 (Krotkov et al., 2017, as processed by Lok Lamsal called OMNO2D_HR). Similar to O₃, the
32 averaging kernel is not being applied for NO₂. Figure 2B-24 and Figure 2B-25 show larger
33 relative biases for NO₂ than O₃, particularly in low NO₂ regions like over the oceans. Best
34 performance was over land during July. Model comparisons to NO₂ have commonly shown
35 biases and research in the broader community continues to resolve this issue.

1 Formaldehyde retrieval comparisons are shown in Figure 2B-26 and Figure 2B-27 using
2 the OMHCHO files, but using the recommended product described by González Abad et al.
3 (2015). The formaldehyde retrievals show a seasonal cycle in the evaluation with a low bias for
4 the northern-most retrievals in January and October. During April there are high biases that seem
5 to migrate northward by July. Though we note this bias feature, the main result is reasonable
6 spatial consistency between the satellite product and the model results. Future work should
7 explore this evaluation further.

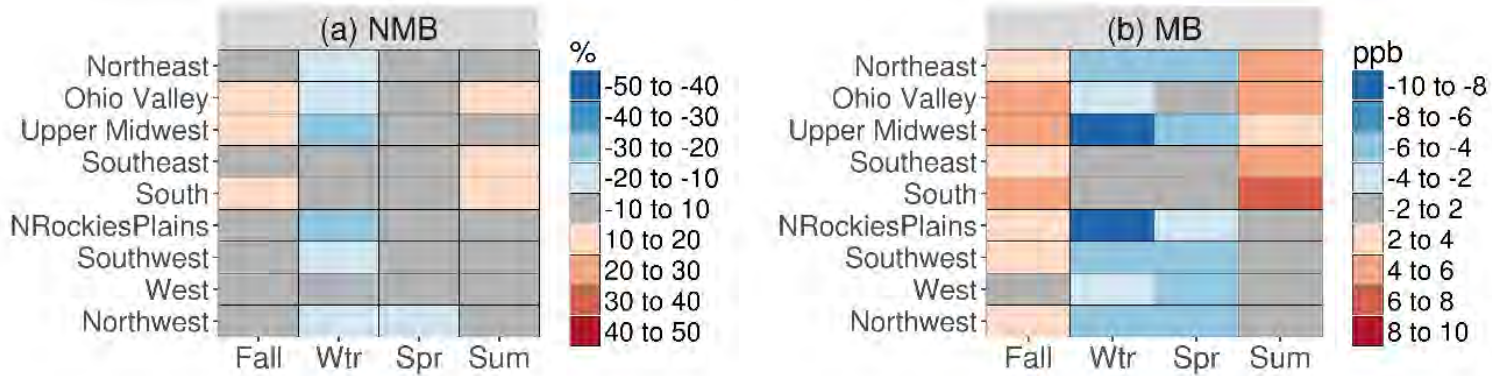
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1 **Table 2B-1. Summary of 12km resolution CONUS CMAQ 2016 model performance**
 2 **statistics for MDA8 O₃ by NOAA climate region, by season and monitoring**
 3 **Network.**

Climate region	Monitor Network	Season	No. of Obs	MB (ppb)	ME (ppb)	NMB (%)	NME (%)
Northeast	AQS	Winter	11,462	-5.9	6.9	-18.1	21.2
		Spring	15,701	-4.3	6.7	-9.8	15.2
		Summer	16,686	4.6	7.7	10.0	17.0
		Fall	13,780	3.3	5.8	9.5	16.9
	CASTNET	Winter	1,195	-6.7	7.3	-19.6	21.3
		Spring	1,246	-5.0	6.9	-11.0	15.2
		Summer	1,224	2.9	6.5	6.7	15.1
		Fall	1,215	3.4	5.6	9.9	16.5
Central	AQS	Winter	4,178	-3.8	5.7	-12.5	18.8
		Spring	15,498	-1.1	5.5	-2.5	12.1
		Summer	20,501	5.5	8.1	12.1	17.9
		Fall	14,041	4.9	6.1	12.6	15.7
	CASTNET	Winter	1,574	-3.1	5.4	-9.6	16.3
		Spring	1,600	-2.2	5.5	-4.8	12.0
		Summer	1,551	3.9	7.1	9.0	16.2
		Fall	1,528	2.7	5.1	6.9	12.8
EastNorthCentral	AQS	Winter	1,719	-8.5	9.2	-27.3	29.5
		Spring	6,892	-3.8	6.8	-8.4	15.2
		Summer	9,742	3.2	6.9	7.7	16.3
		Fall	6,050	5.6	3.4	17.6	20.2
	CASTNET	Winter	435	-9.6	10.1	-28.6	30.1
		Spring	434	-6.5	7.8	-14.4	17.4
		Summer	412	0.2	5.5	0.5	13.4
		Fall	426	2.9	5.1	9.2	16.0
Southeast	AQS	Winter	7,196	-1.4	5.0	-3.9	14.0
		Spring	14,569	-1.5	5.3	-3.2	11.3
		Summer	15,855	5.1	7.1	12.9	17.9
		Fall	12,589	3.4	5.4	8.4	13.3
	CASTNET	Winter	887	-3.5	5.3	-9.3	14.3
		Spring	947	-3.6	5.6	-7.5	11.7
		Summer	926	3.9	6.2	9.9	16.0
		Fall	928	1.7	5.0	4.0	11.9
South	AQS	Winter	11,342	-1.0	5.0	-3.1	15.0
		Spring	13,093	1.3	6.1	2.8	13.9
		Summer	12,819	6.0	7.8	15.7	20.4
		Fall	12,443	4.8	6.3	12.1	16.0

Climate region	Monitor Network	Season	No. of Obs	MB (ppb)	ME (ppb)	NMB (%)	NME (%)
	CASTNET	Winter	516	-1.7	5.0	-4.8	13.7
		Spring	532	-1.2	5.6	-2.6	12.3
		Summer	508	2.6	6.1	6.7	15.8
		Fall	520	3.5	5.0	9.0	12.9
Southwest	AQS	Winter	9,695	-4.2	6.2	-11.0	16.1
		Spring	10,608	-4.8	6.5	-9.4	12.7
		Summer	10,549	-1.2	6.0	-2.3	11.2
		Fall	10,298	2.5	4.9	6.0	12.0
	CASTNET	Winter	757	-8.1	8.5	-18.0	18.9
		Spring	810	-6.9	7.6	-13.1	14.5
		Summer	812	-2.8	5.5	-5.3	10.3
		Fall	791	-0.1	3.6	-0.3	8.3
WestNorthCentral	AQS	Winter	4,740	-9.3	9.6	-24.9	25.9
		Spring	5,066	-3.1	5.9	-7.2	13.5
		Summer	5,134	0.7	4.9	1.4	10.6
		Fall	4,940	3.3	5.2	9.8	15.3
	CASTNET	Winter	568	-9.1	9.8	-23.1	25.0
		Spring	607	-5.8	7.3	-12.4	15.6
		Summer	600	-1.8	4.6	-3.7	9.4
		Fall	505	1.7	4.8	4.4	12.8
Northwest	AQS	Winter	677	-5.7	7.5	-17.5	23.1
		Spring	1,288	-4.3	7.3	-10.5	18.2
		Summer	2,444	1.2	6.6	3.3	17.5
		Fall	1,236	2.8	5.9	9.0	18.7
	CASTNET	Winter	--	--	--	--	--
		Spring	--	--	--	--	--
		Summer	--	--	--	--	--
		Fall	--	--	--	--	--
West	AQS	Winter	14,550	-2.1	5.3	-6.1	15.3
		Spring	17,190	-4.0	6.1	-8.8	13.3
		Summer	18,046	0.6	8.1	1.2	15.2
		Fall	16,163	0.4	5.5	0.9	12.8
	CASTNET	Winter	506	-3.4	5.6	-8.7	14.1
		Spring	519	-5.7	6.6	-11.8	13.7
		Summer	526	-5.3	8.1	-8.7	13.3
		Fall	530	-2.2	4.7	-4.6	10.0

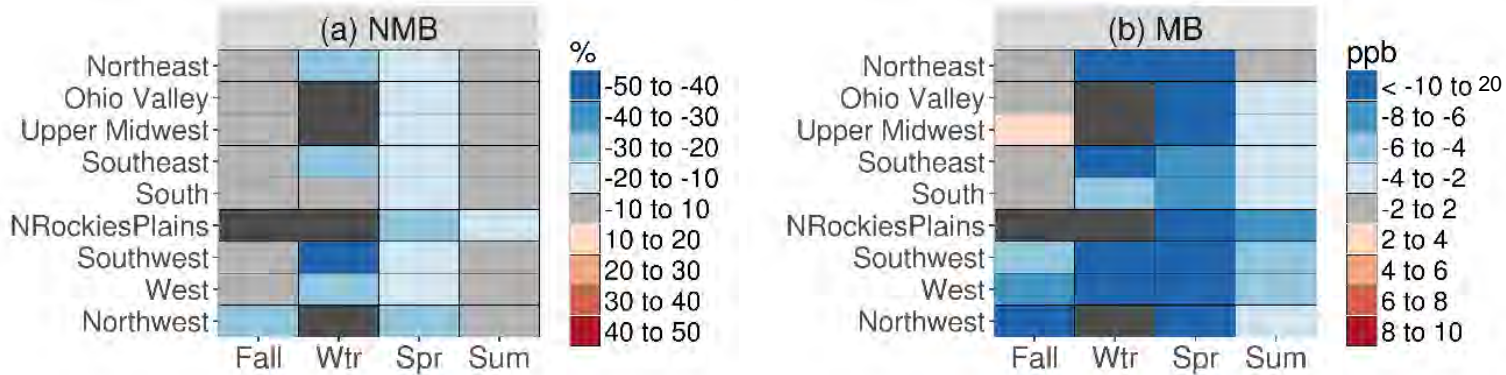
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3 **Figure 2B-2. (a) Normalized Mean Bias (%) and (b) Mean Bias (ppb) of maximum daily average 8-hr ozone (MDA8) by**
 4 **NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites.** In the text, alternative names are
 5 used: Ohio Valley is Central, Upper Midwest is EastNorthCentral, and NRockiesPlains is NorthWestCentral.

6



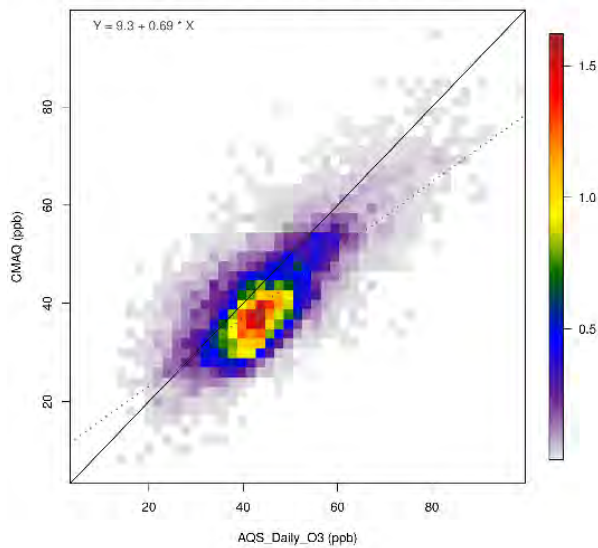
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8 **Figure 2B-3. NMB (a) and MB (b) of MDA8 O₃ greater than or equal to 60 ppb from the 12km resolution CONUS simulation**
 9 **by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites. Dark grey cells indicate**
 10 **missing values (i.e., no monitored days with MDA8 >= 60 ppb in that region).** In the text, alternative names are
 11 used: Ohio Valley is Central, Upper Midwest is EastNorthCentral, and NRockiesPlains is NorthWestCentral.

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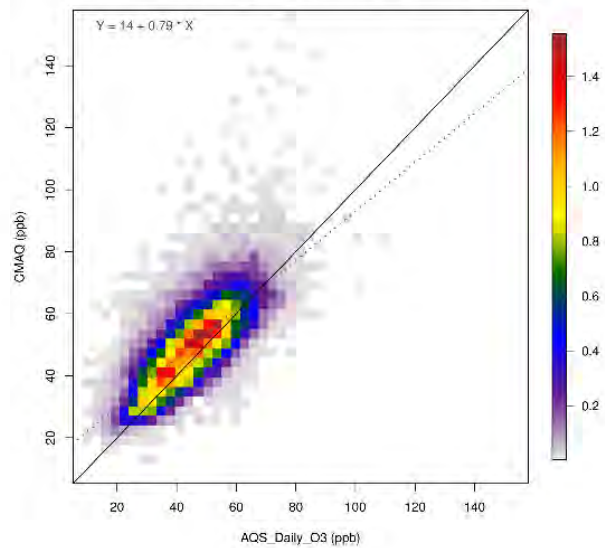
Spring

CMAQ_2016fe_cb6r3_16j_12US2 O3_8hrmax for Spring_Northeast



Summer

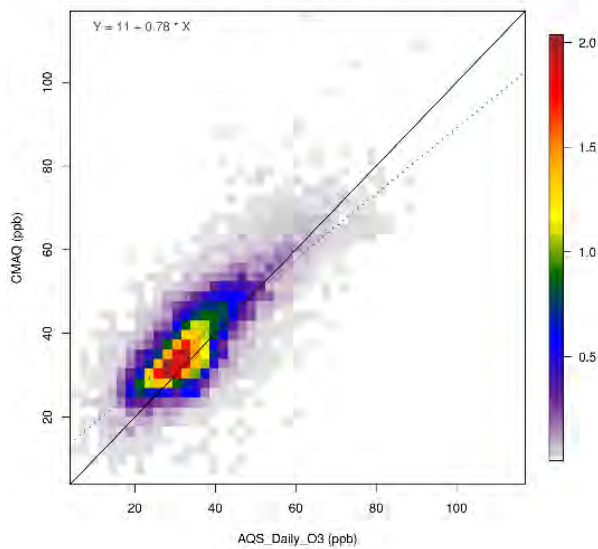
CMAQ_2016fe_cb6r3_16j_12US2 O3_8hrmax for Summer_Northeast



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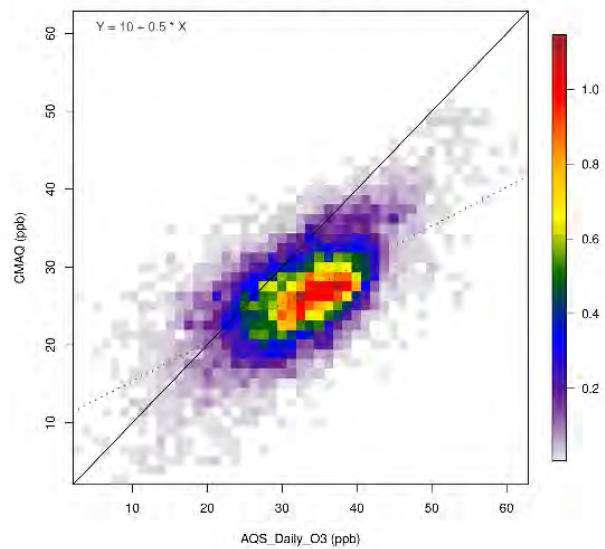
Fall

CMAQ_2016fe_cb6r3_16j_12US2 O3_8hrmax for Fall_Northeast



Winter

CMAQ_2016fe_cb6r3_16j_12US2 O3_8hrmax for Winter_Northeast

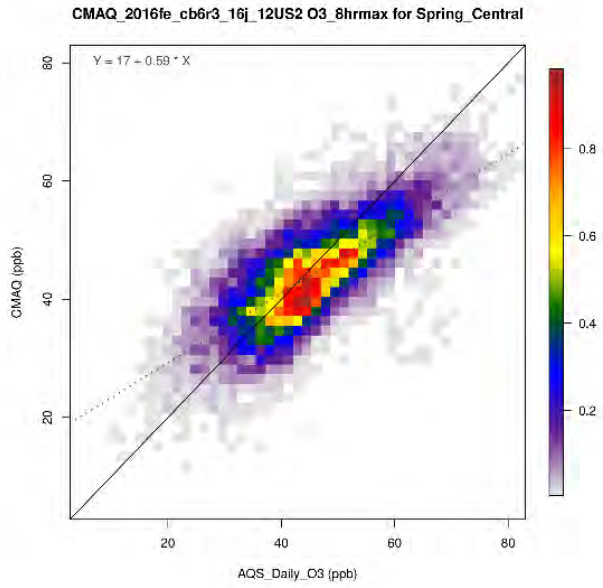


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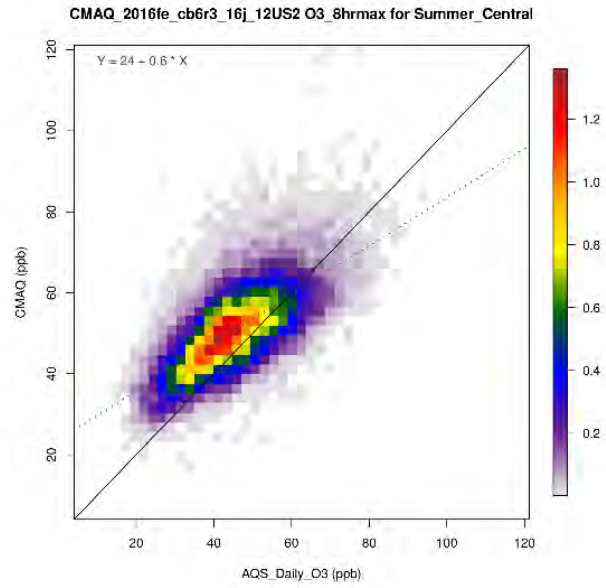
Figure 2B-4. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km resolution CONUS simulation for the Northeast region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

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Spring



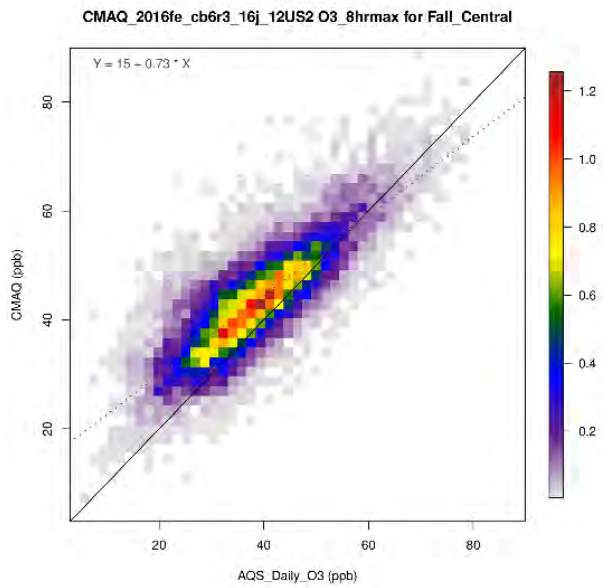
Summer



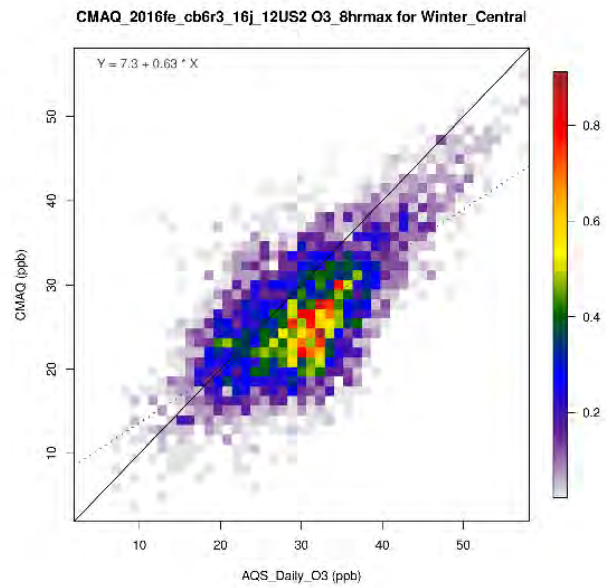
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Fall



Winter

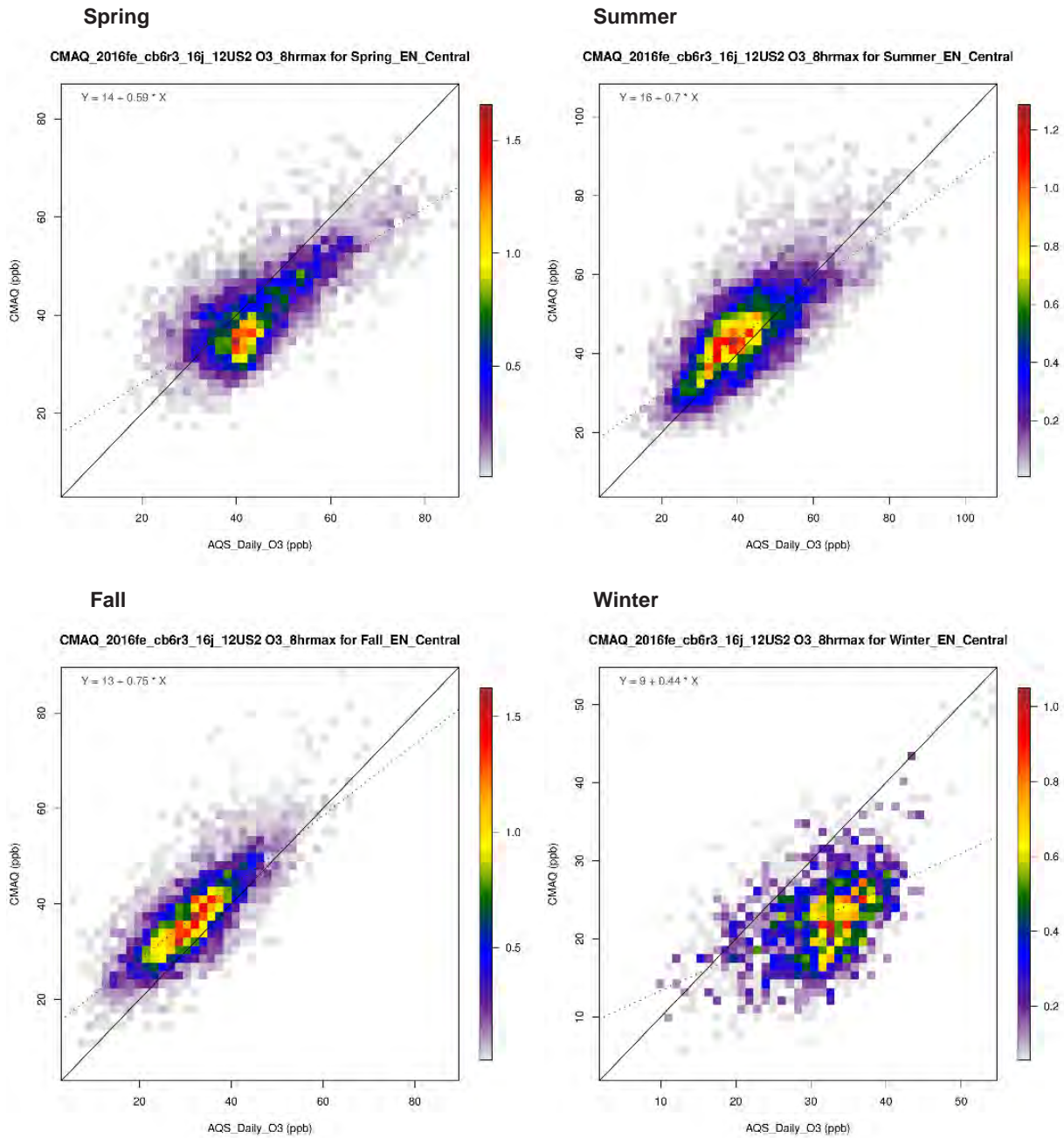


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5 **Figure 2B-5. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km**
 6 **resolution CONUS simulation for the Central region by season. Each plot has**
 7 **a separate scale that is shared for the x and y axes. The dashed line represents the**
 8 **best fit linear regression line.**

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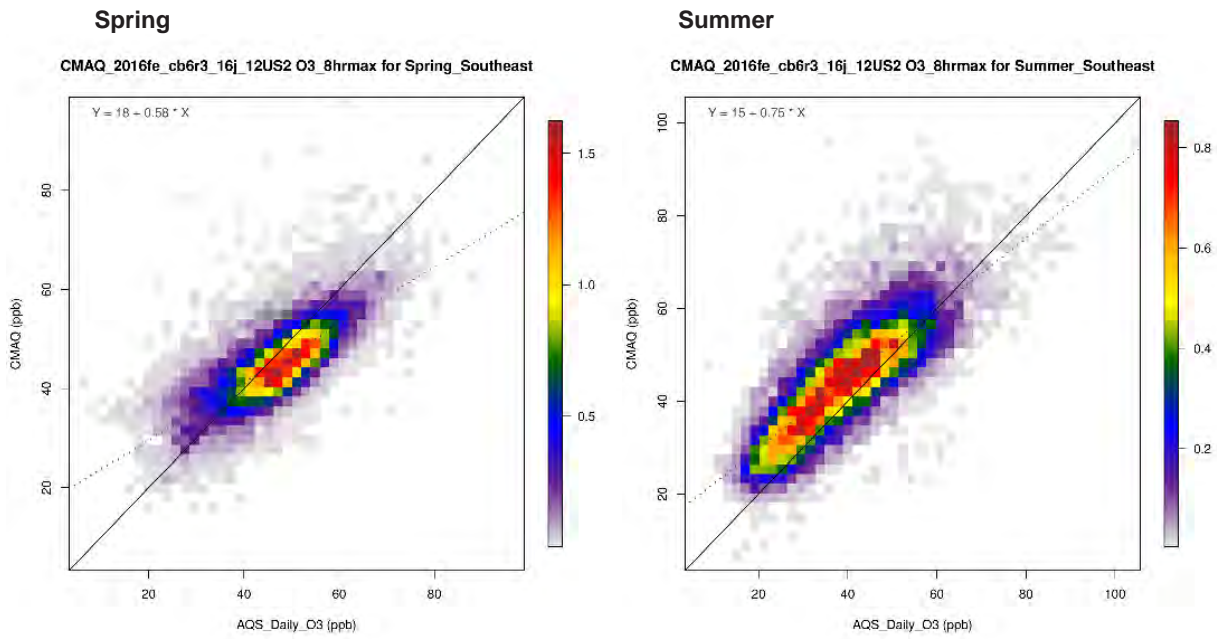
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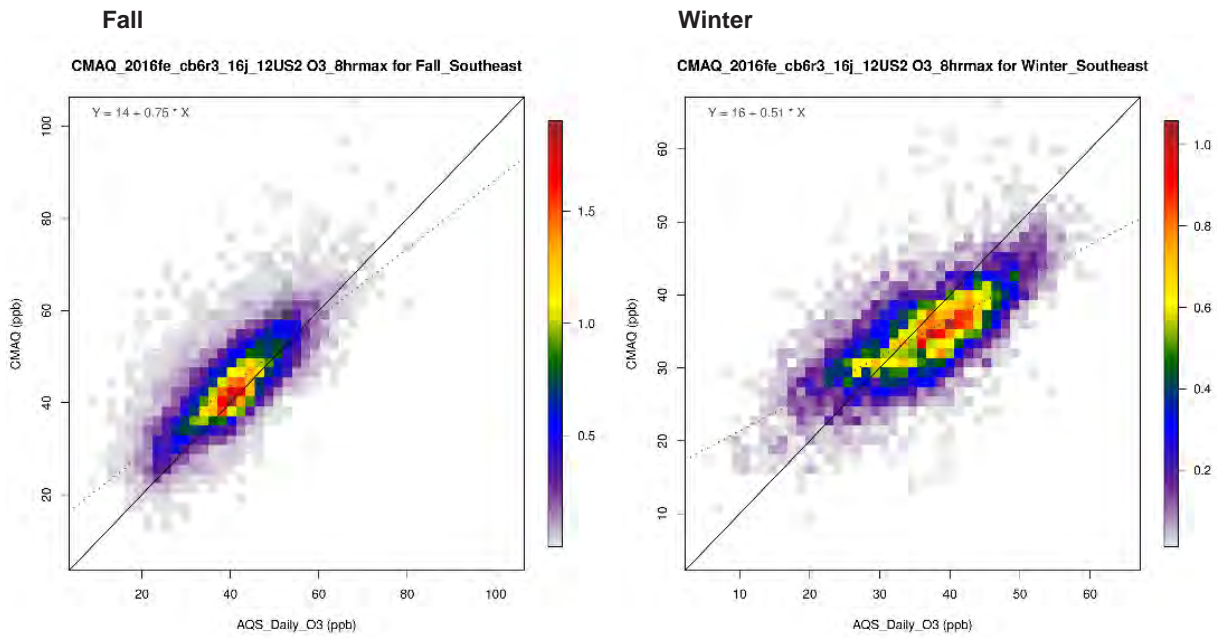
7 **Figure 2B-6. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km**
8 **resolution CONUS simulation for the EastNorthCentral region by season.**
9 Each plot has a separate scale that is shared for the x and y axes. The dashed line
10 represents the best fit linear regression line.

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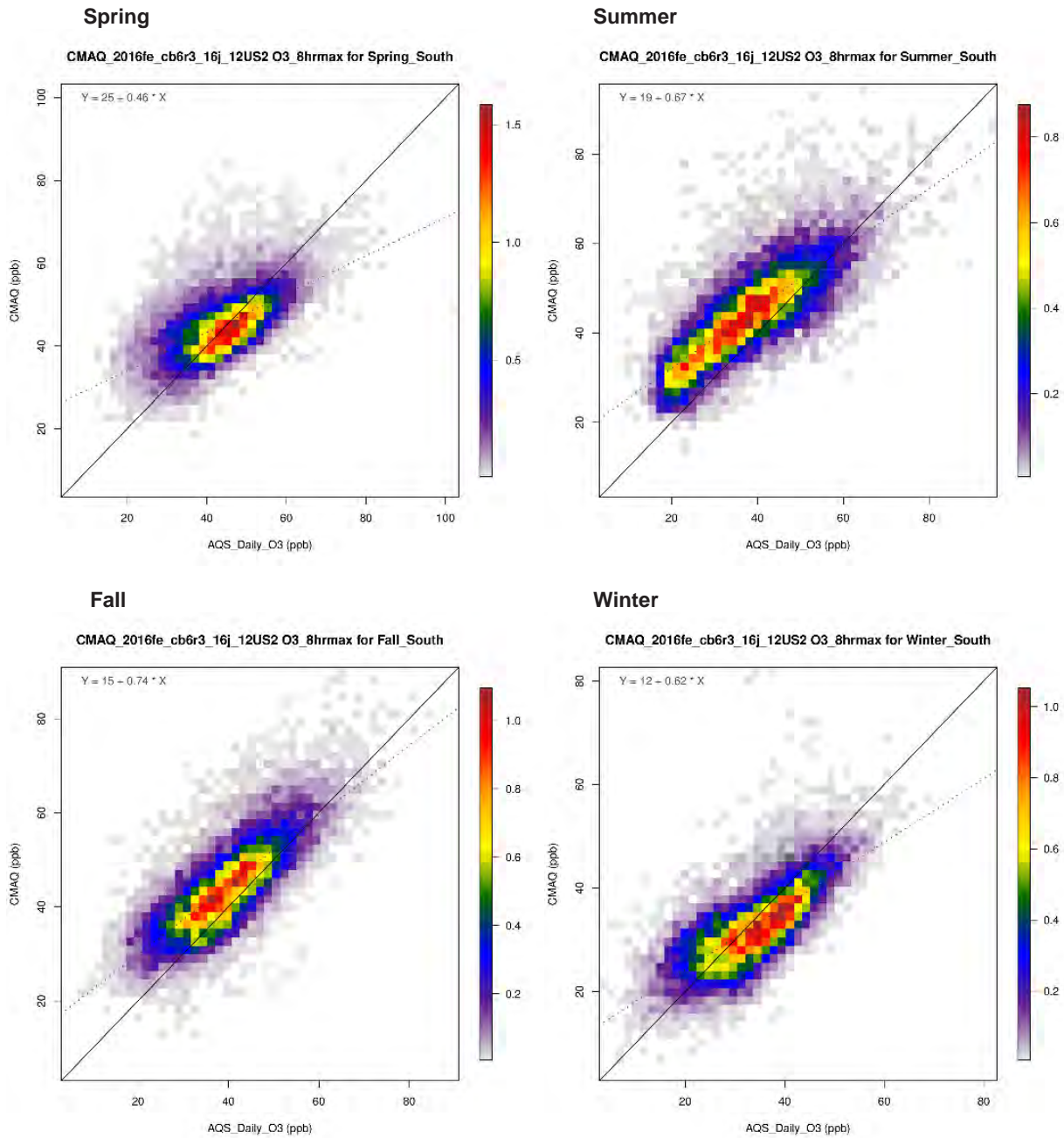
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Figure 2B-7. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km resolution CONUS simulation for the Southeast region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

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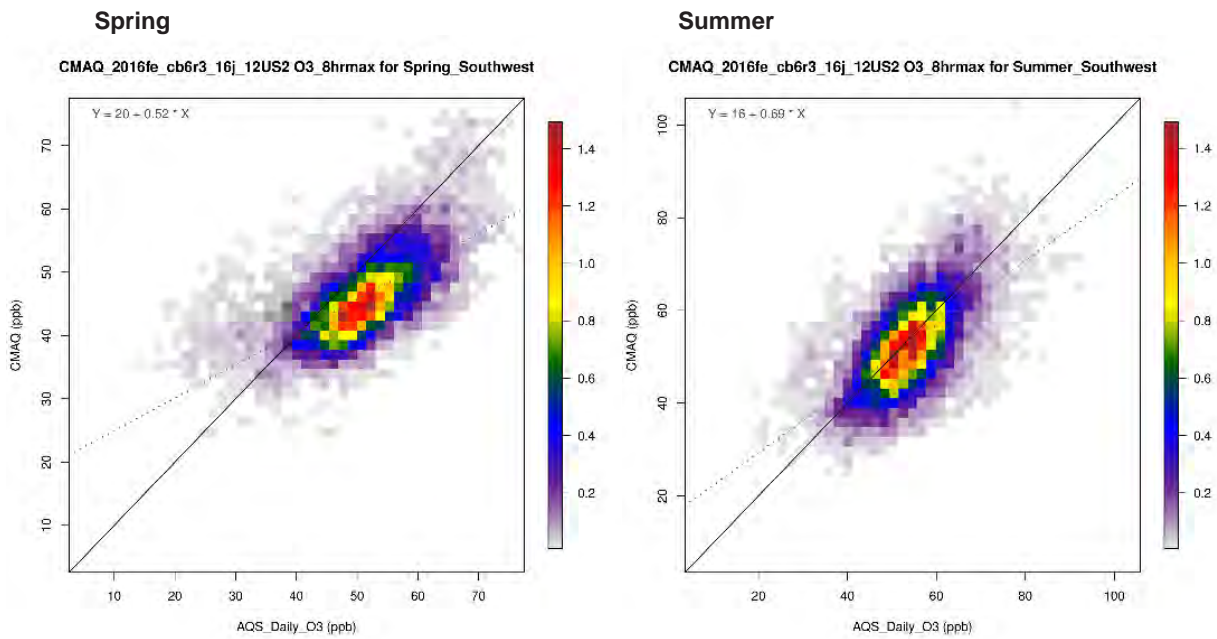
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7 **Figure 2B-8. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km**
8 **resolution CONUS simulation for the South region by season. Each plot has a**
9 **separate scale that is shared for the x and y axes. The dashed line represents the**
10 **best fit linear regression line.**

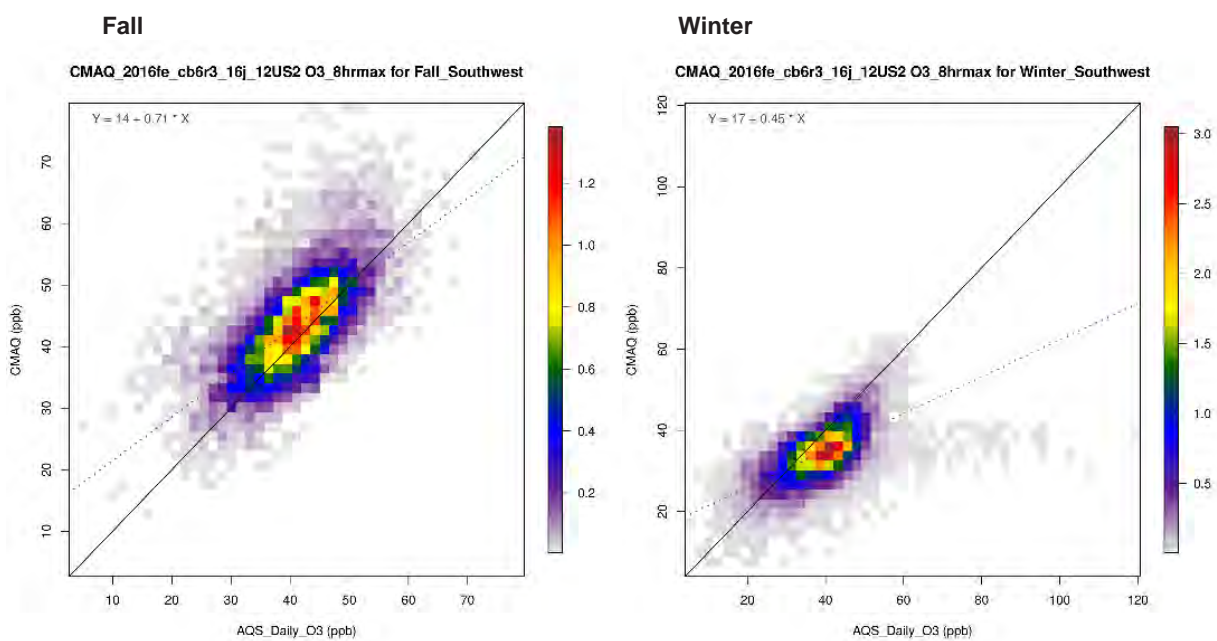
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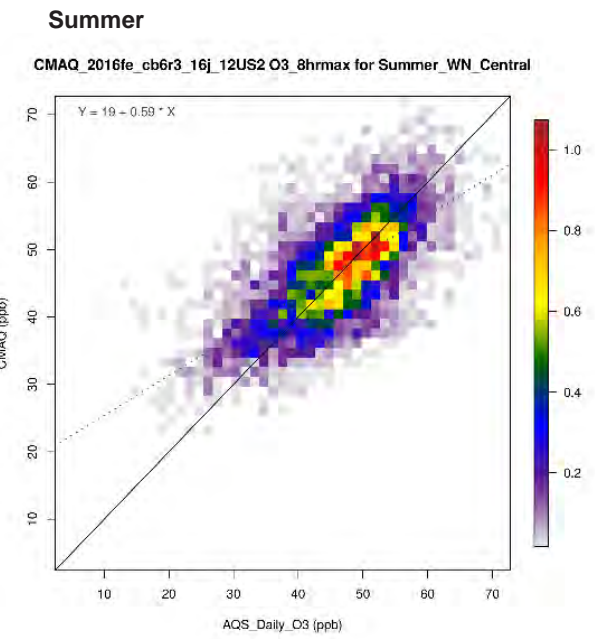
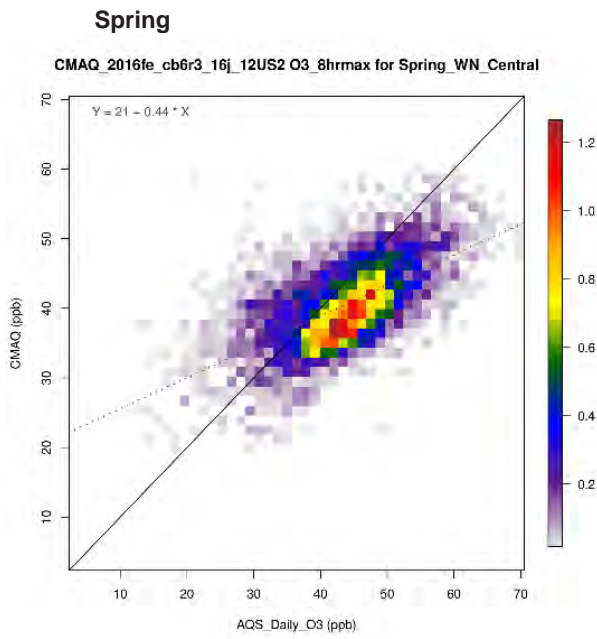
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5 **Figure 2B-9. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km**
 6 **resolution CONUS simulation for the Southwest region by season. Each plot**
 7 **has a separate scale that is shared for the x and y axes. The dashed line represents**
 8 **the best fit linear regression line.**

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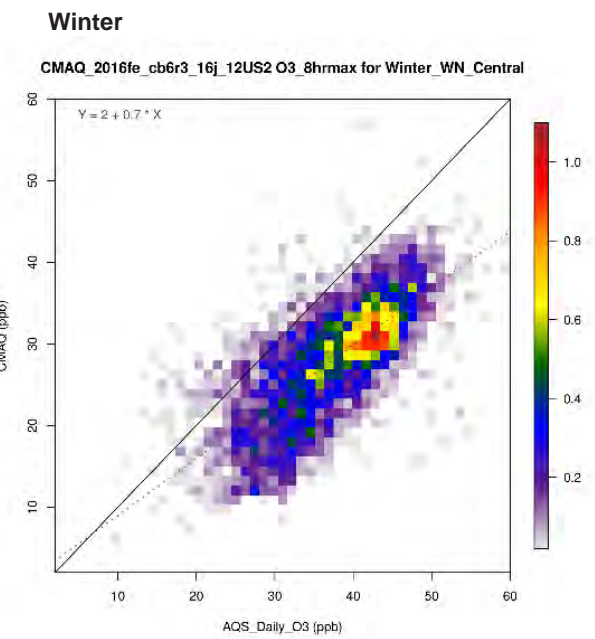
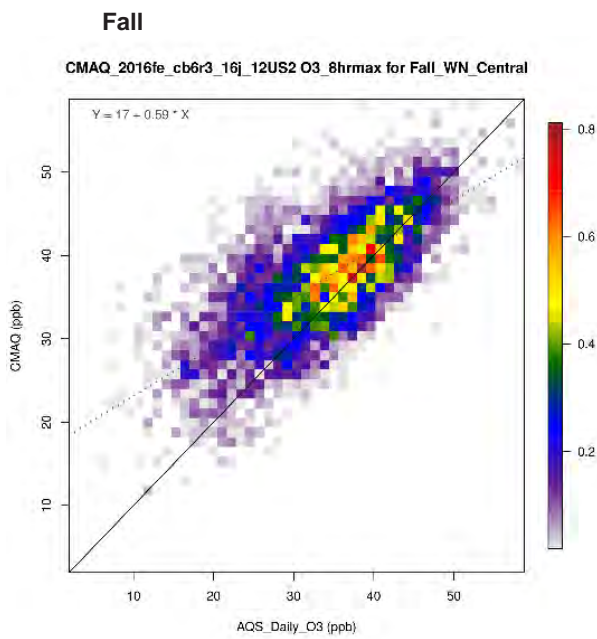
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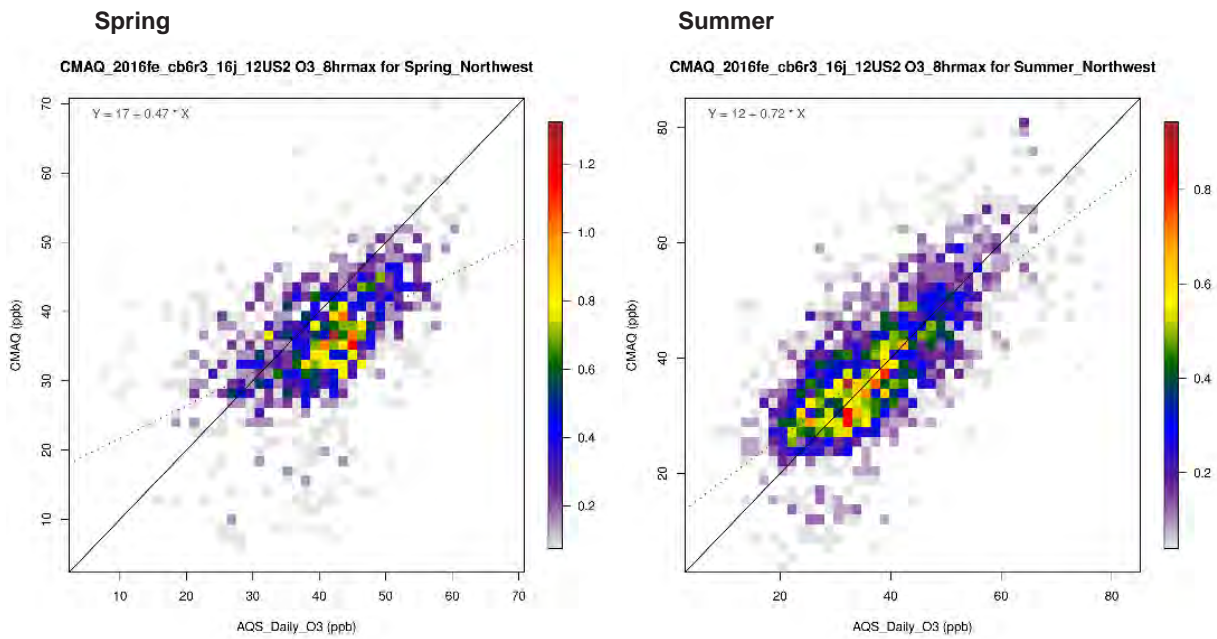
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5 **Figure 2B-10. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km**
 6 **resolution CONUS simulation for the WestNorthCentral region by season.**
 7 Each plot has a separate scale that is shared for the x and y axes. The dashed line
 8 represents the best fit linear regression line.

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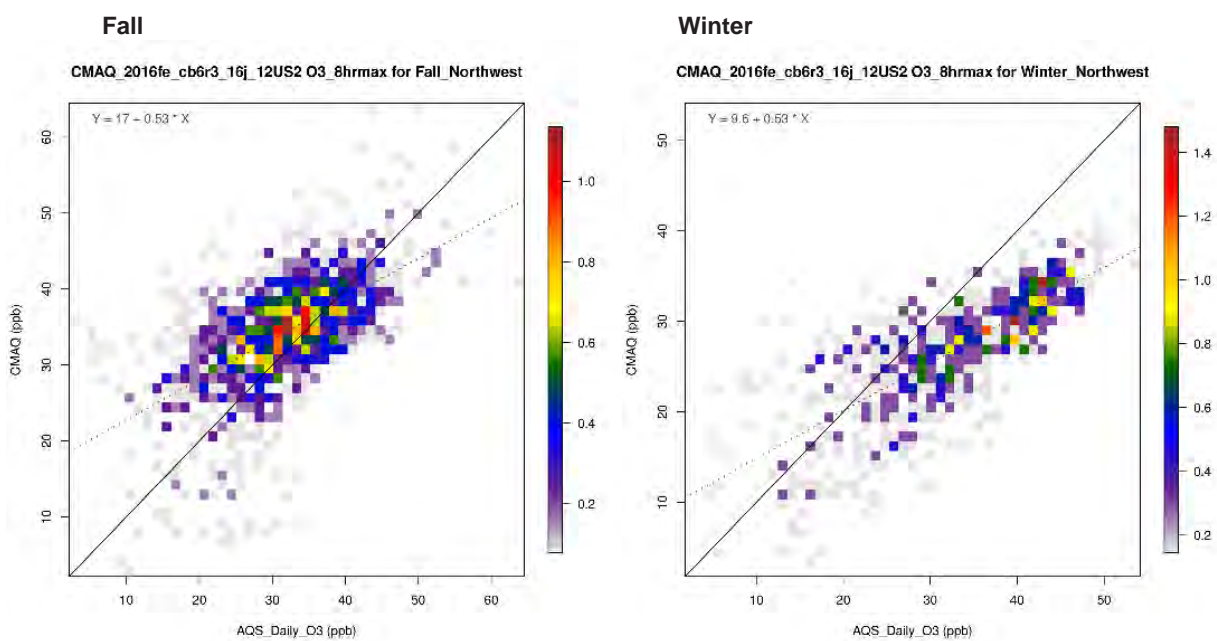
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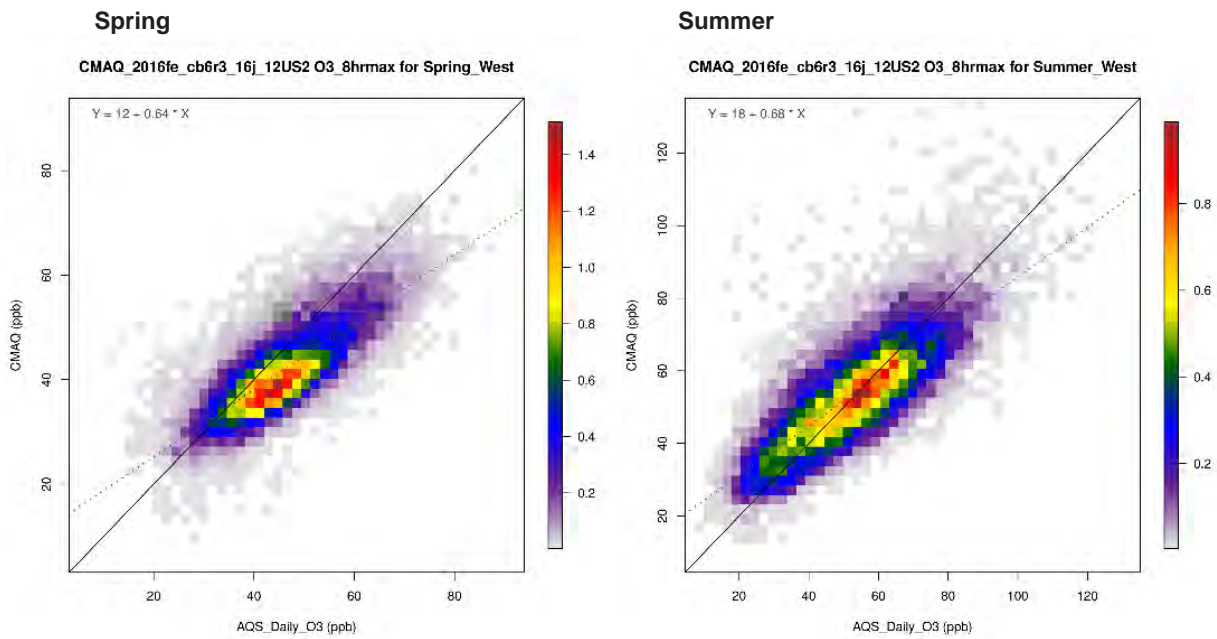
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5 **Figure 2B-11. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km**
 6 **resolution CONUS simulation for the Northwest region by season. Each plot**
 7 **has a separate scale that is shared for the x and y axes. The dashed line represents**
 8 **the best fit linear regression line.**

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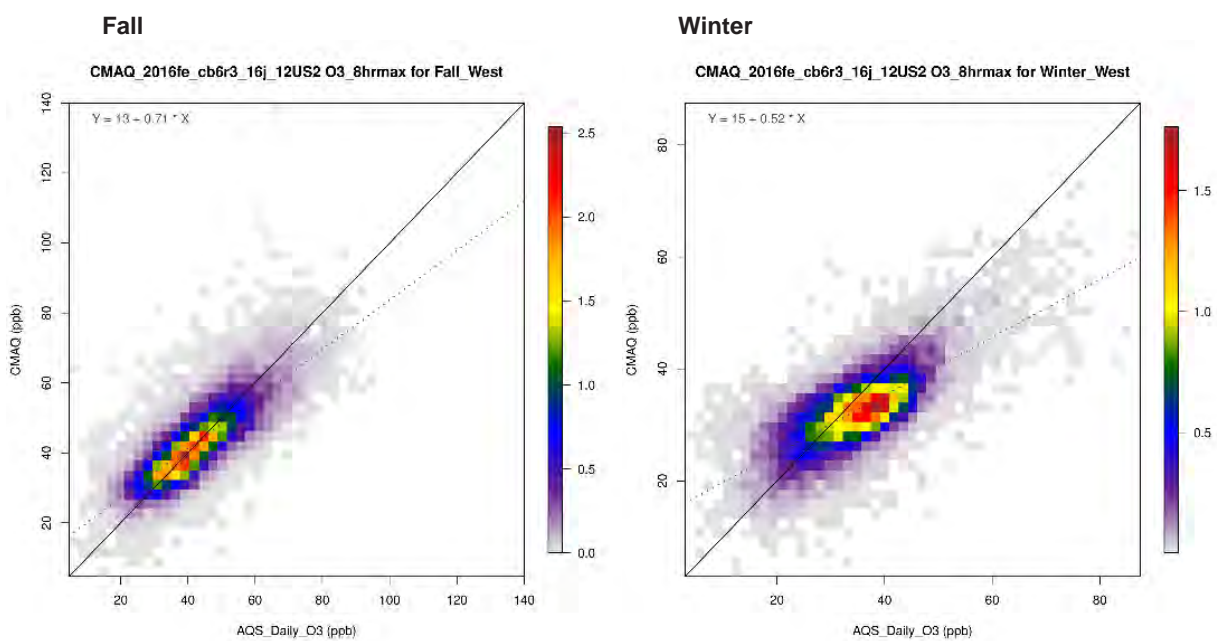
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5 **Figure 2B-12. Density scatter plots of observed versus predicted MDA8 O₃ from the 12km**
 6 **resolution CONUS simulation for the West region by season. Each plot has a**
 7 **separate scale that is shared for the x and y axes. The dashed line represents the**
 8 **best fit linear regression line.**

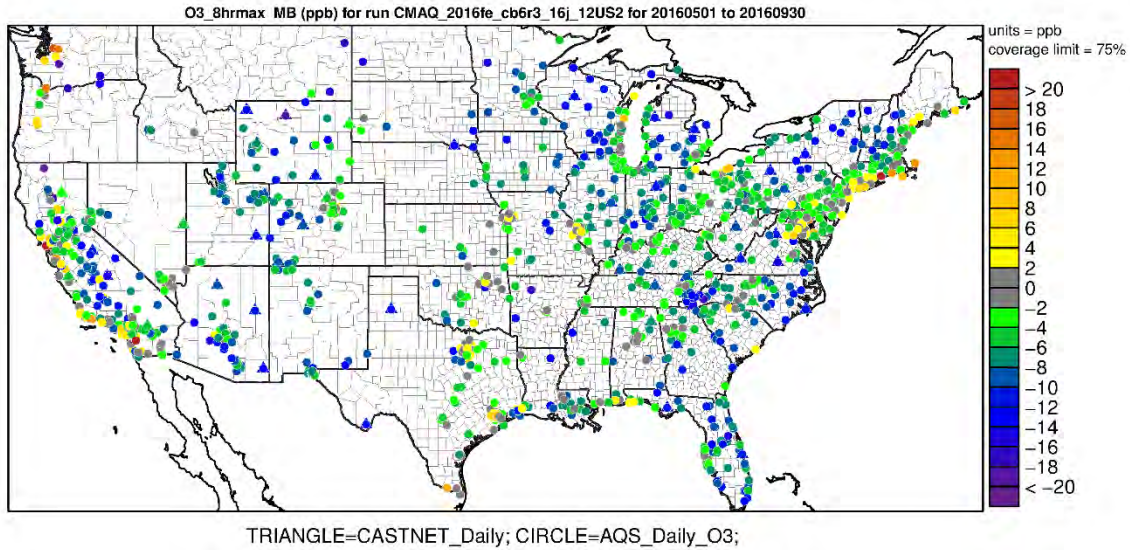
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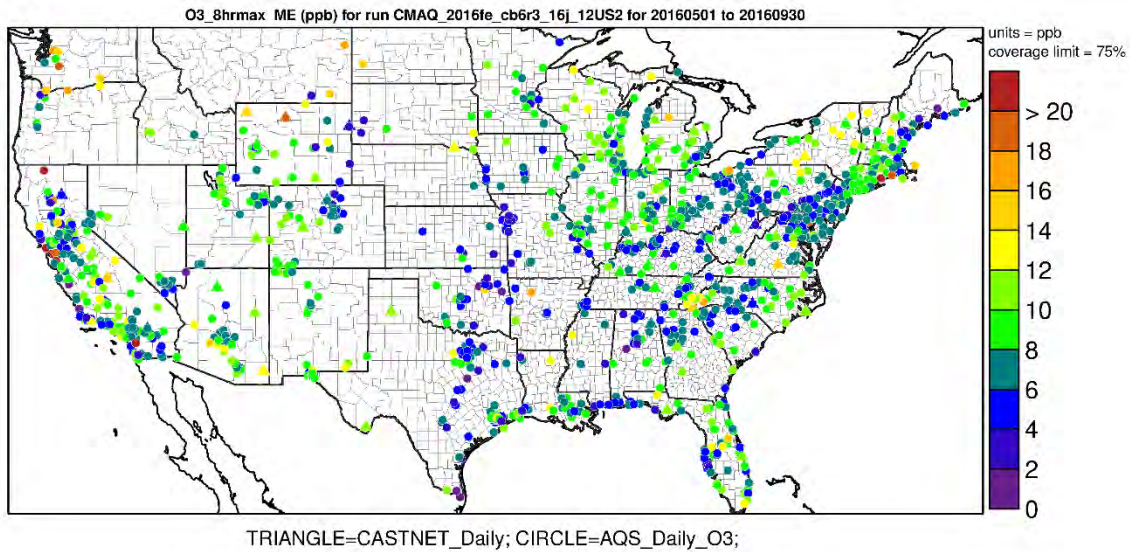
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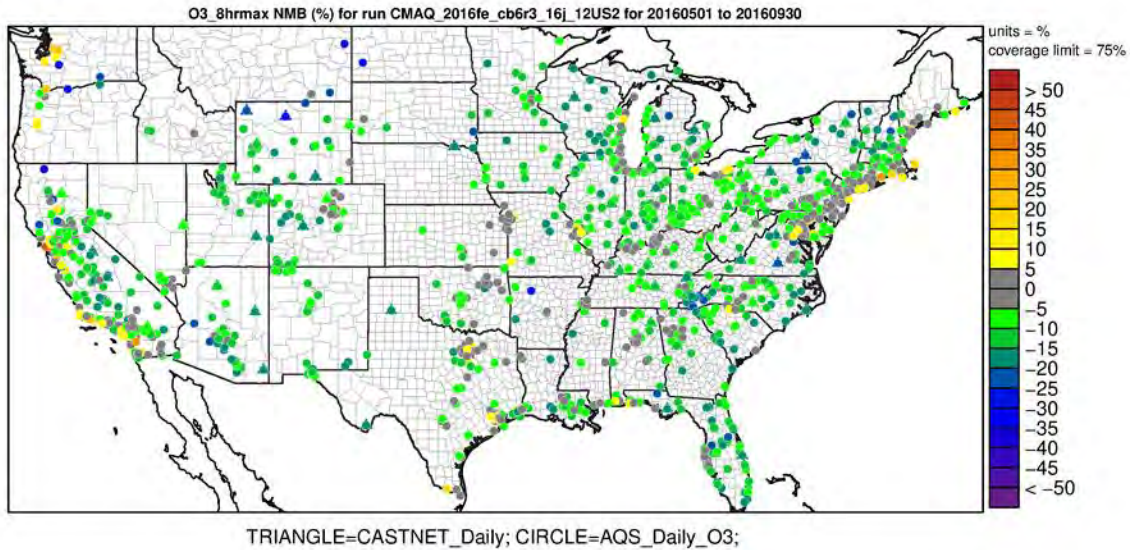
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Figure 2B-13. Mean Bias (ppb) from the 12km resolution CONUS simulation of MDA8 O₃ greater than or equal to 60 ppb over the period May through September 2016 at AQS and CASTNET monitoring sites in the continental U.S. modeling domain.

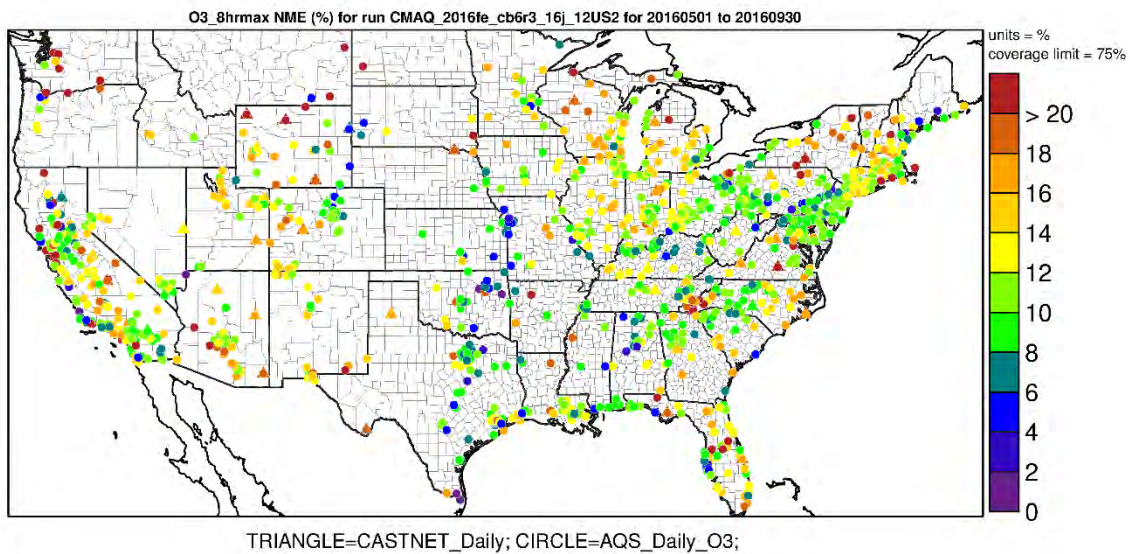


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Figure 2B-14. Mean Error (ppb) from the 12km resolution CONUS simulation of MDA8 O₃ greater than or equal to 60 ppb over the period May through September 2016 at AQS and CASTNET monitoring sites in the continental U.S. modeling domain.

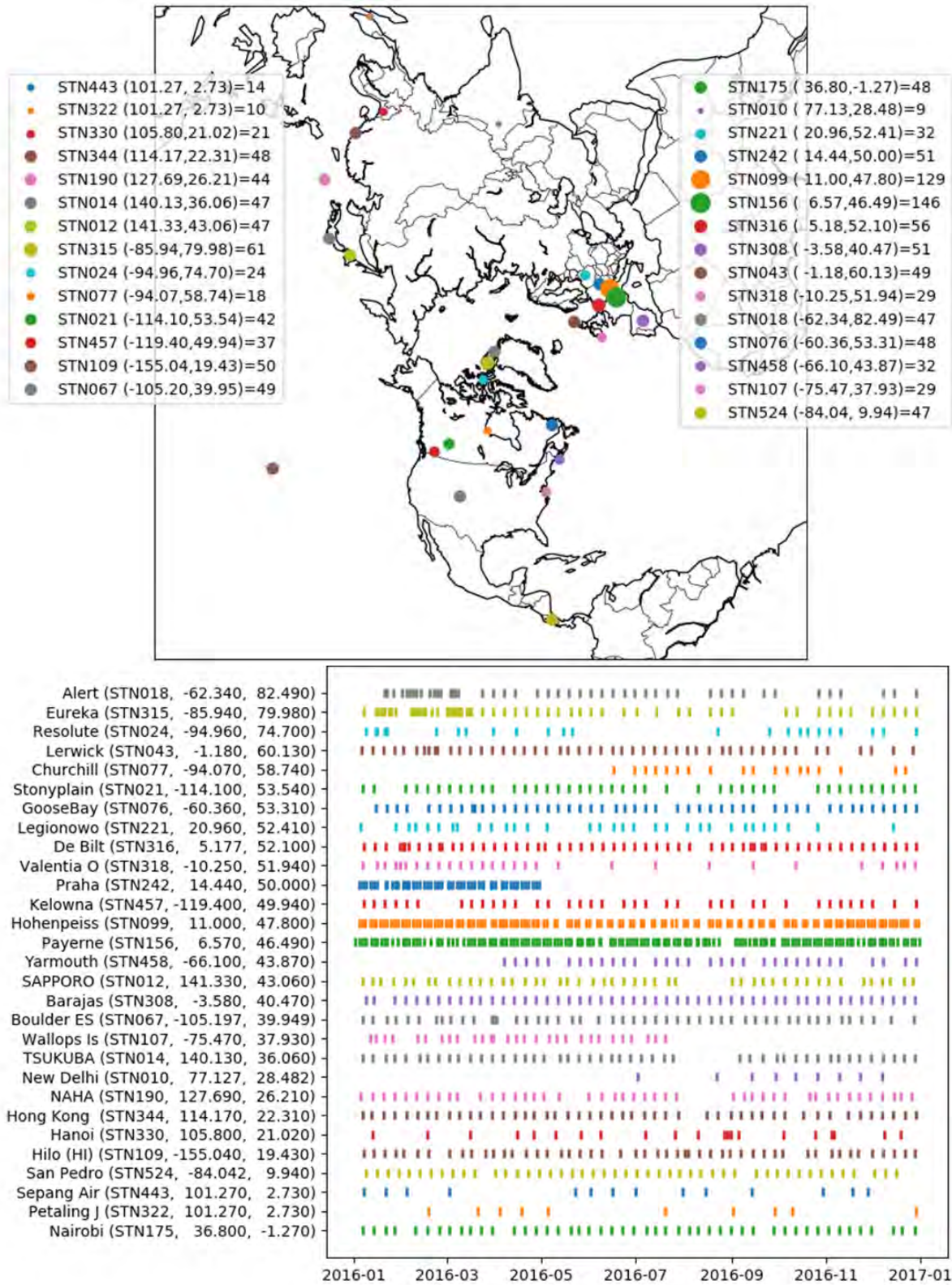


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2 **Figure 2B-15. NMB (%) from the 12km resolution CONUS simulation of MDA8 O₃ greater**
3 **than or equal to 60 ppb over the period May through September 2016 at AQS**
4 **and CASTNET monitoring sites in the continental U.S. modeling domain.**

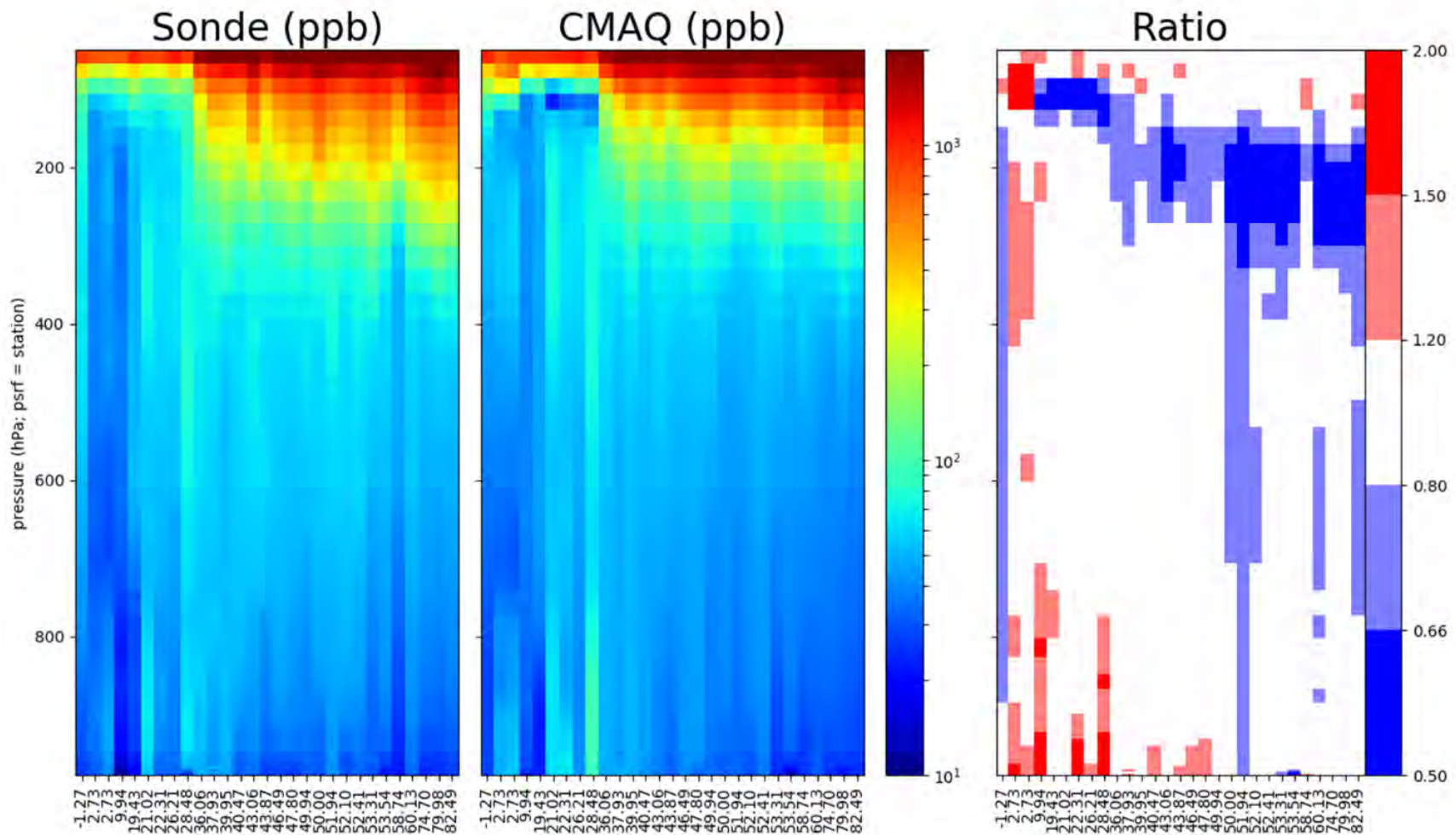


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6 **Figure 2B-16. NME (%) from the 12km resolution CONUS simulation of MDA8 O₃ greater**
7 **than or equal to 60 ppb over the period May through September 2016 at AQS**
8 **and CASTNET monitoring sites in the continental U.S. modeling domain.**

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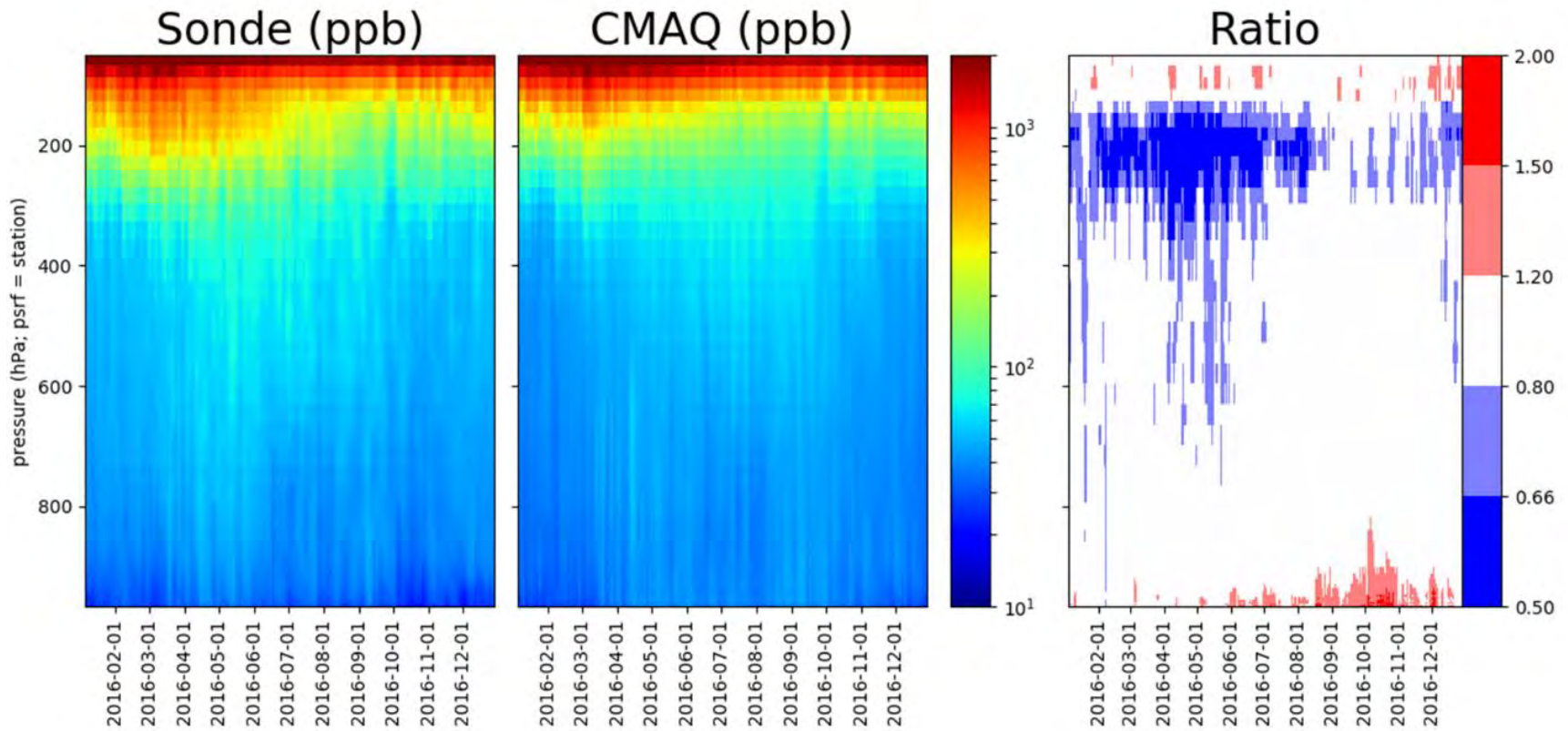


1 **Figure 2B-17. WUDC sonde locations and sampling frequency used in evaluation of**
 2 **hemispheric model simulation.**



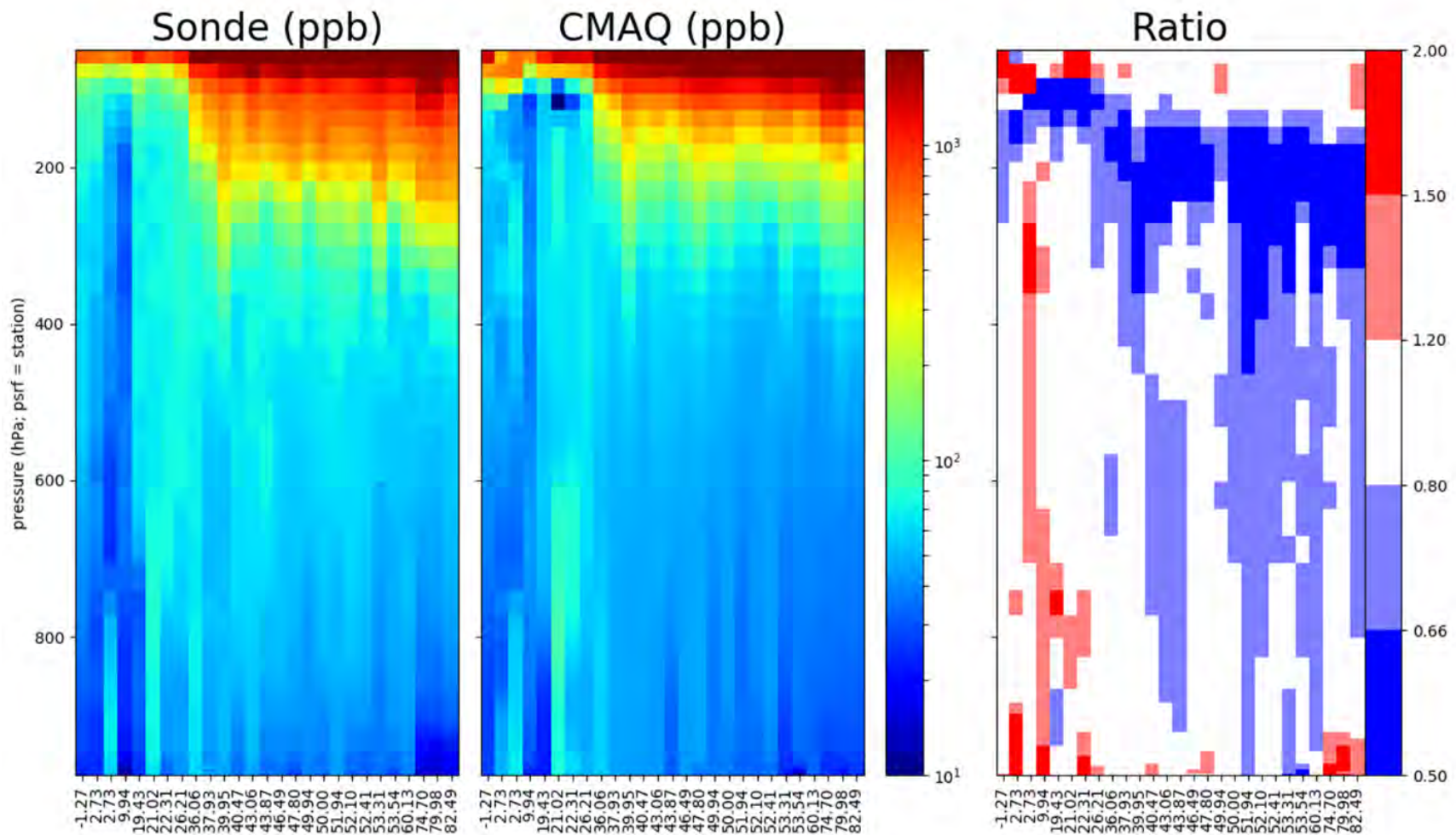
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 2 **Figure 2B-18. WOUDC sonde releases averaged by release location over 2016; observations (left), predictions from the**
 3 **hemispheric CMAQ simulation (middle), ratio (right). Observations are ordered with increasing latitude (South**
 4 **to North).**

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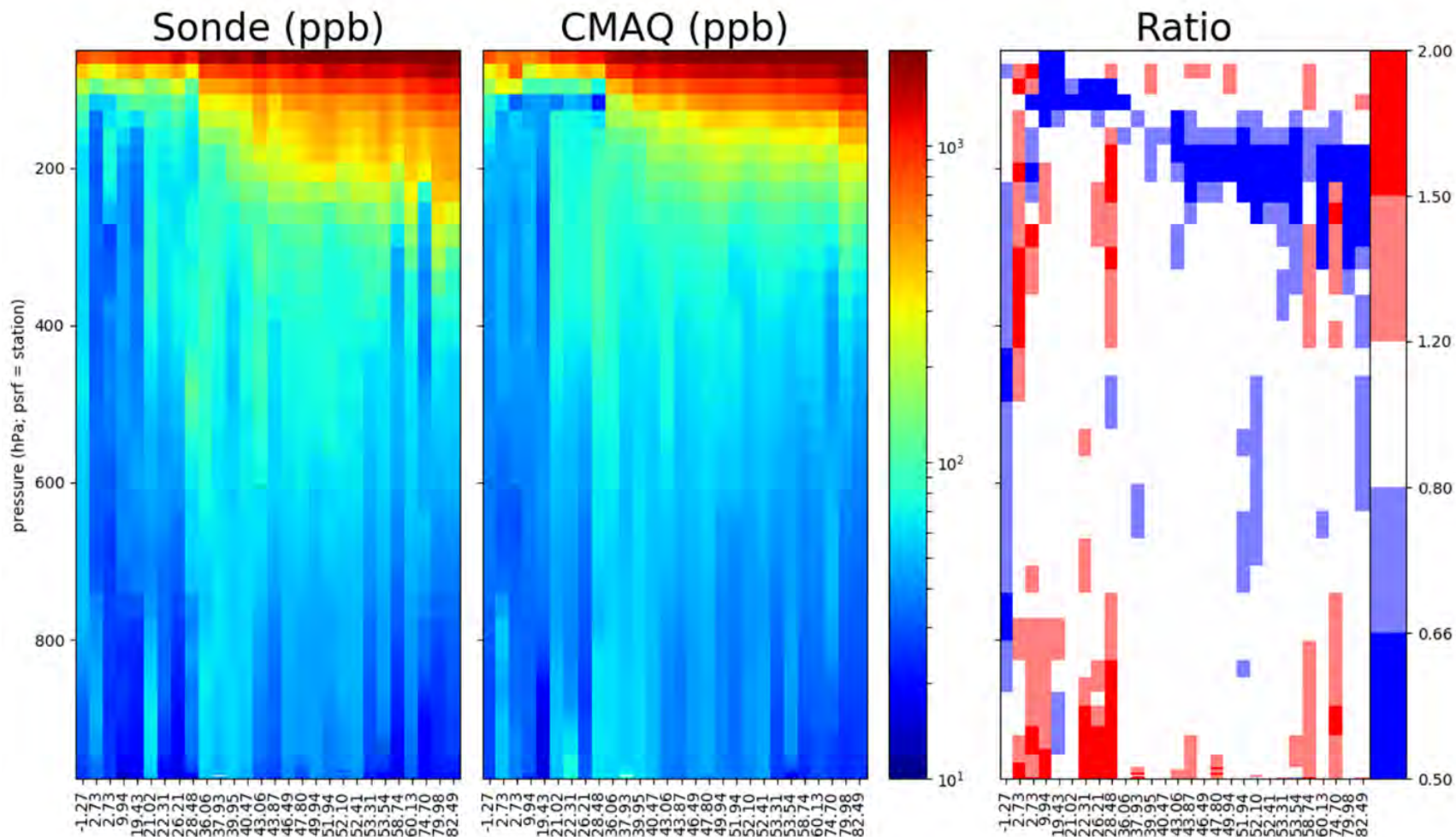


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3 **Figure 2B-19. Woudc sonde releases averaged by day with a 20-point moving average; observations (left), predictions from**
4 **the hemispheric CMAQ simulation (middle), ratio (right).**

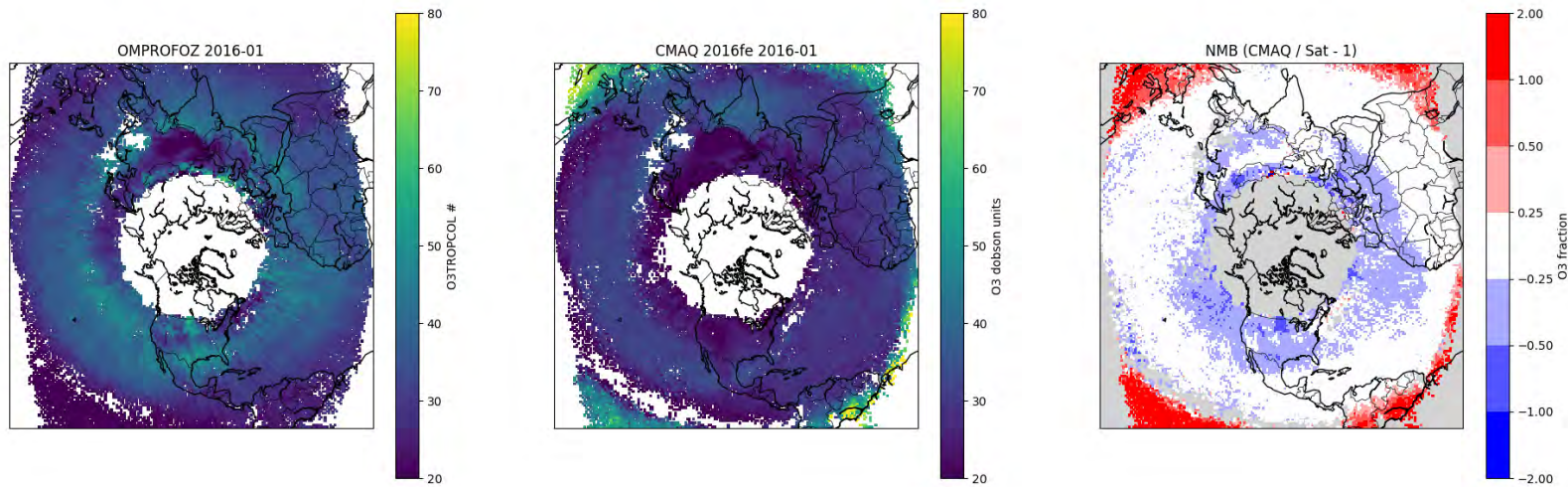


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 2 **Figure 2B-20. WOUDC sonde releases averaged by release location over March, April, May in 2016; observations (left),**
 3 **predictions from the hemispheric CMAQ simulation (middle), ratio (right).**

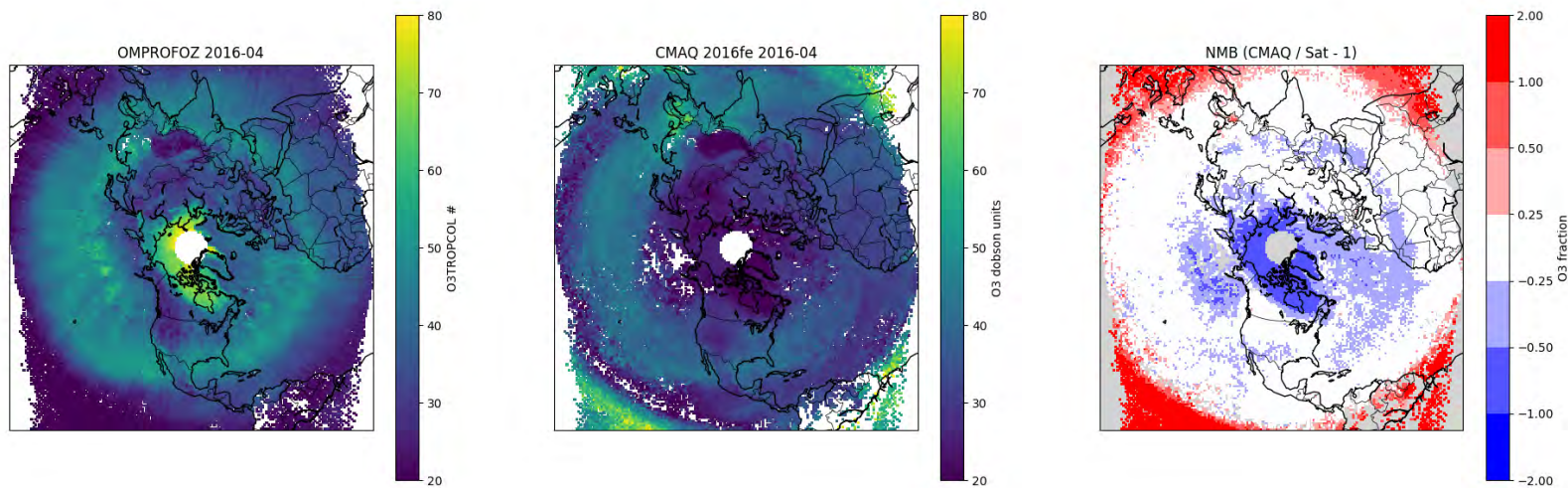


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 2 **Figure 2B-21. Woudc sonde releases averaged by release location over June, July, August in 2016; observations (left),**
 3 **predictions from the hemispheric CMAQ simulation (middle), ratio (right).**

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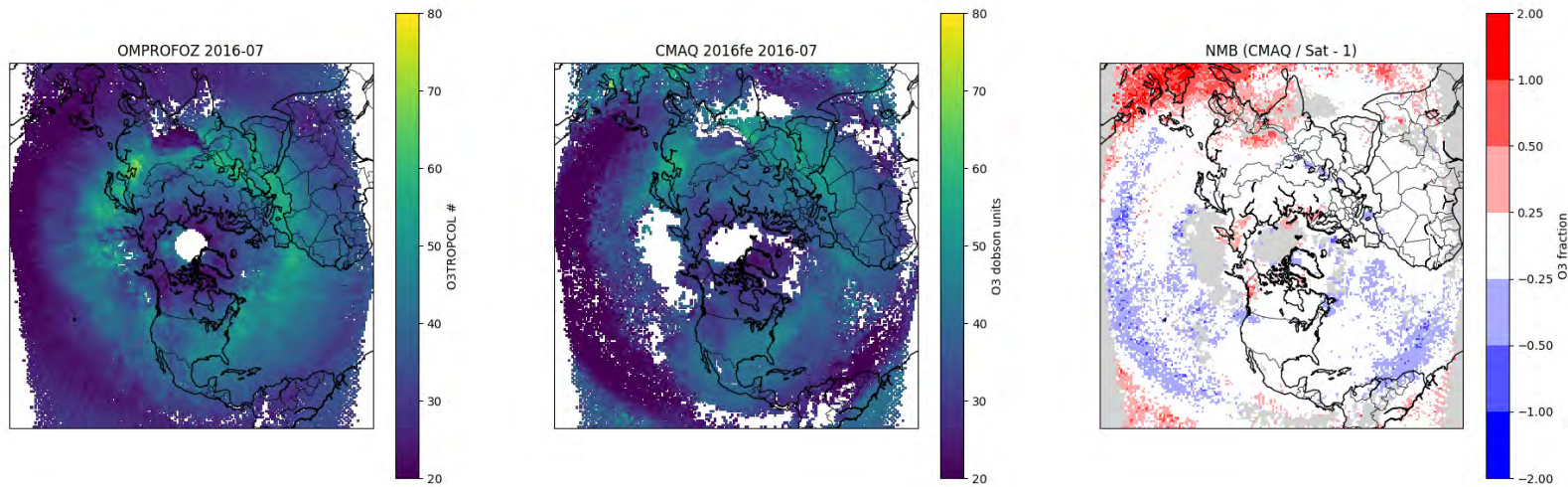


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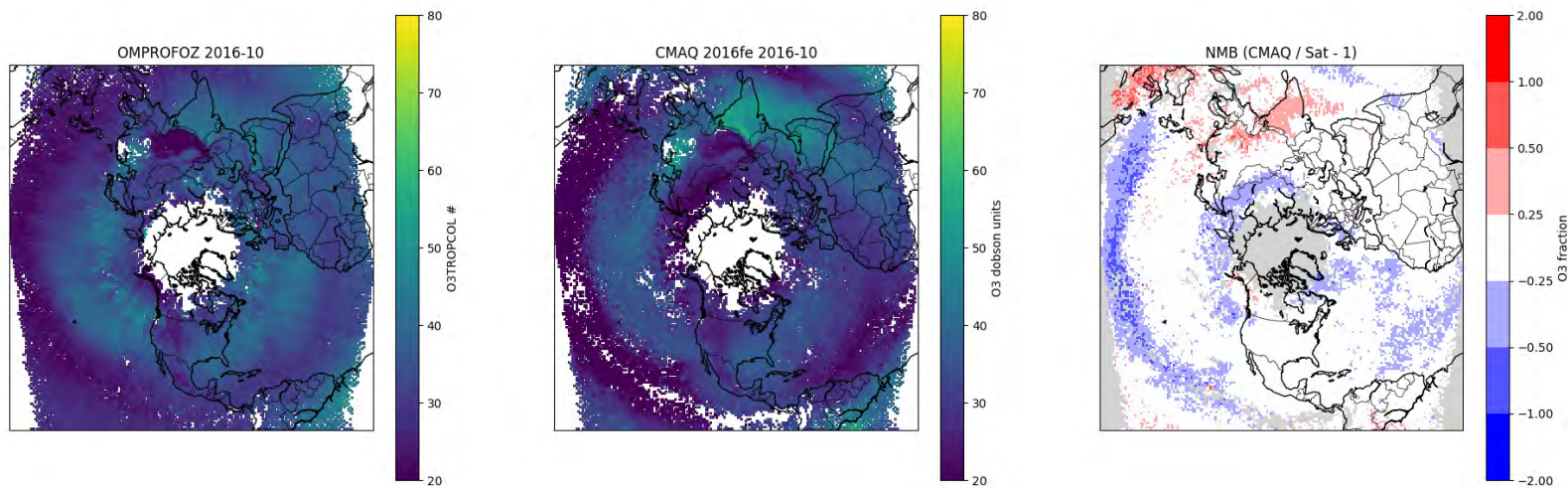


3 **Figure 2B-22. OMI O₃ (OMPROFOZ v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios**
4 **(right) of vertical column densities for January (top) and April (bottom).**

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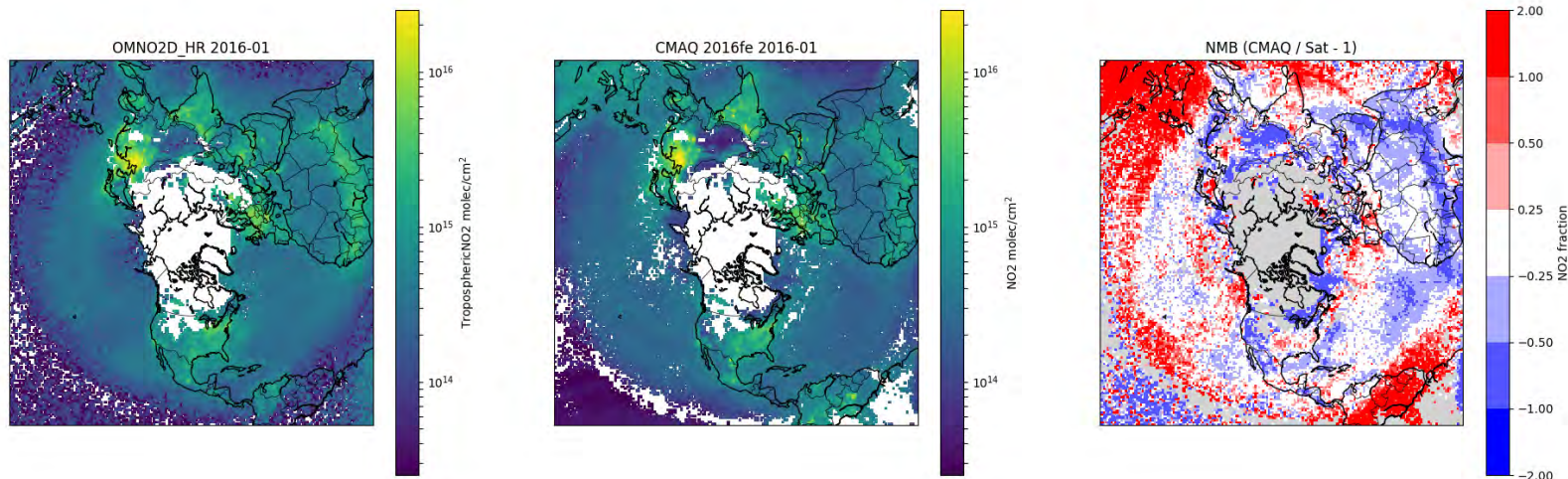


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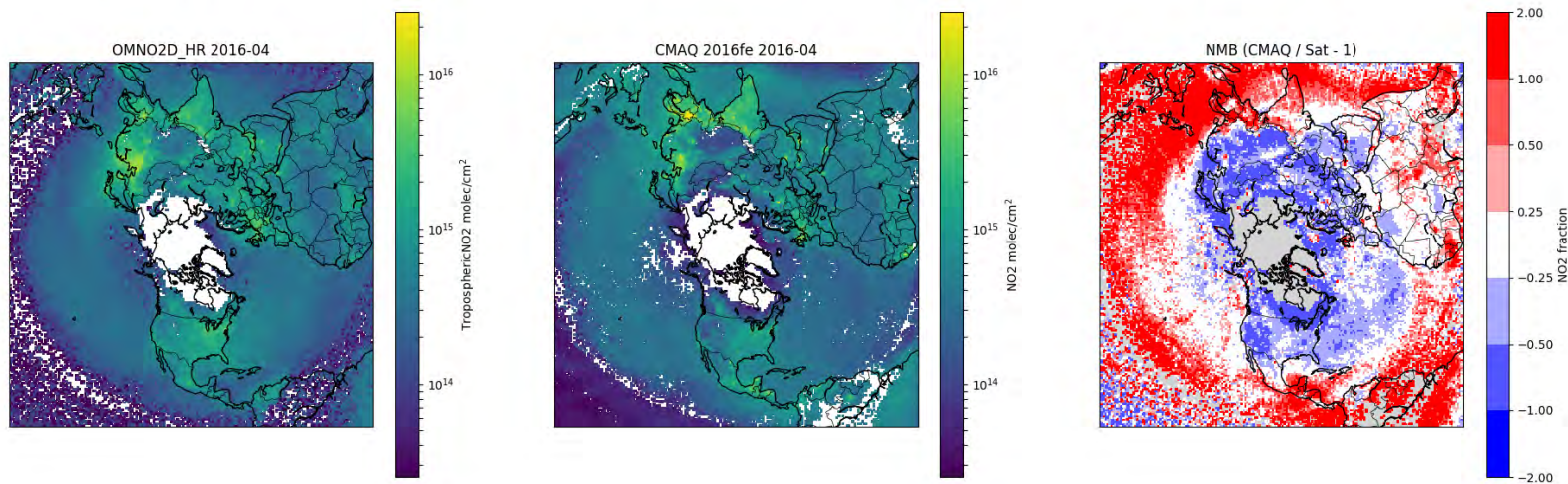


3 **Figure 2B-23. OMI O₃ (OMPROFOZ v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios**
4 **(right) of vertical column densities for July (top), and October (bottom).**

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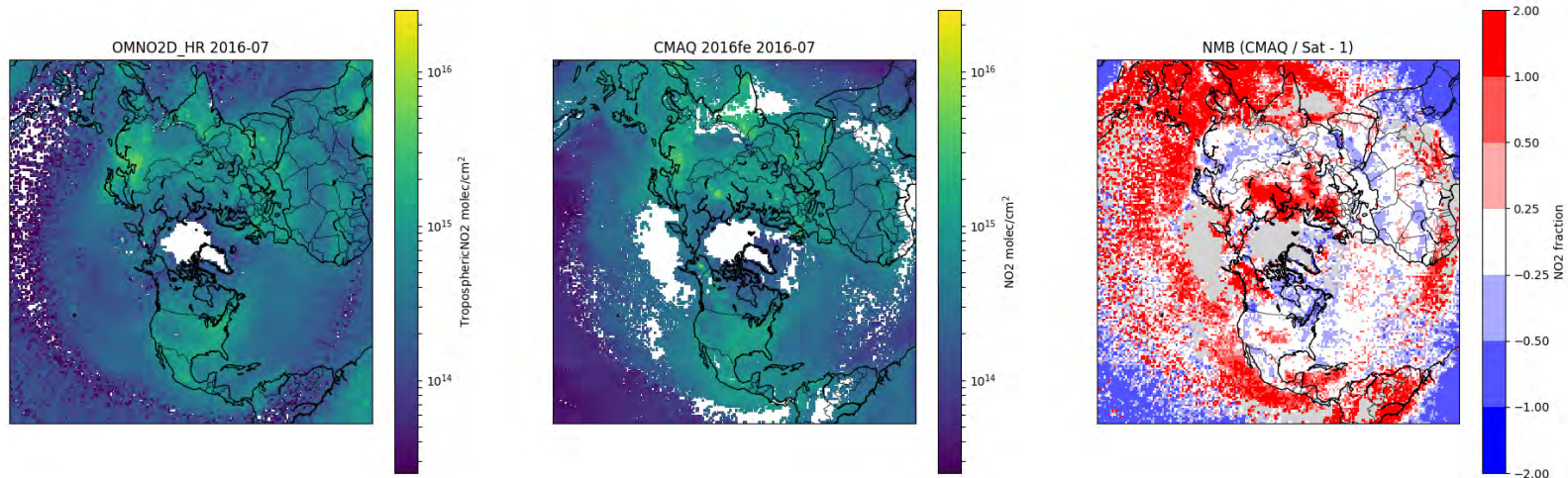


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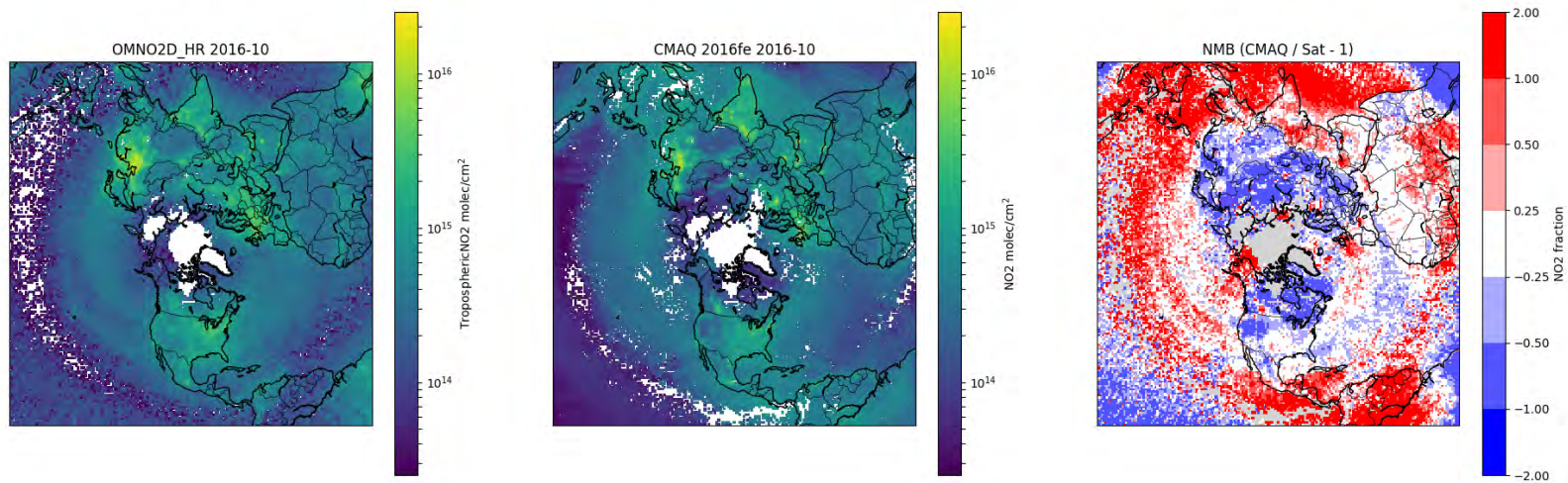


3 **Figure 2B-24. OMI Nitrogen Dioxide (OMNO2D_HR v003, left) compared to simulated (hemispheric CMAQ simulation,**
4 **center), and ratios (right) of vertical column densities for January (top) and April (bottom).**

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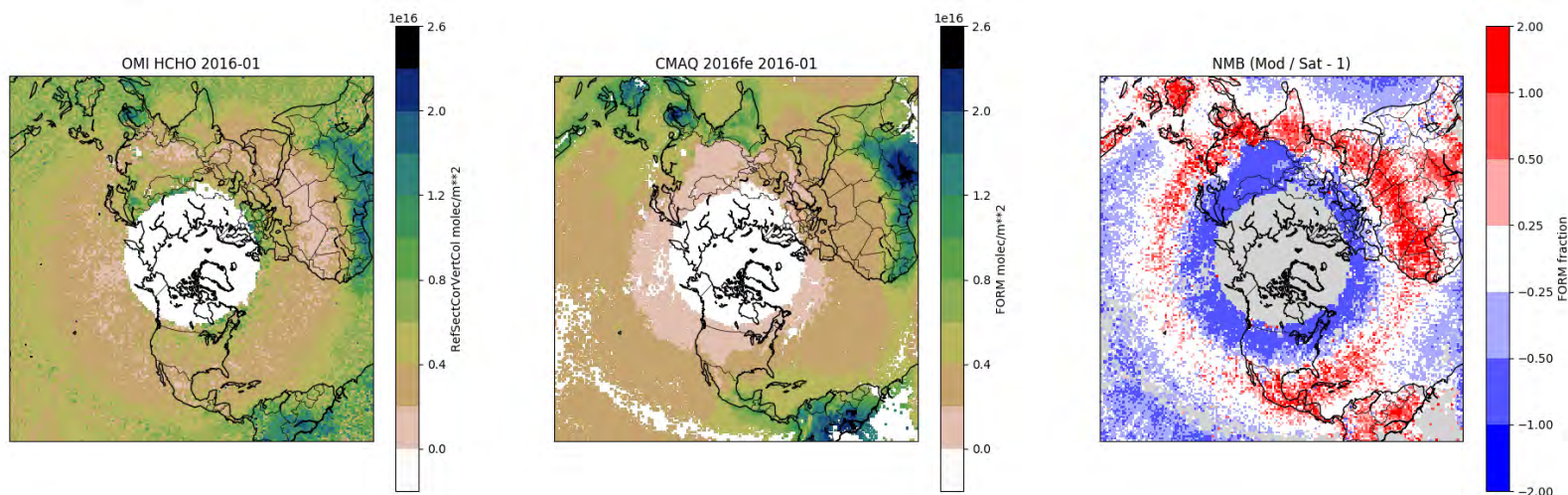


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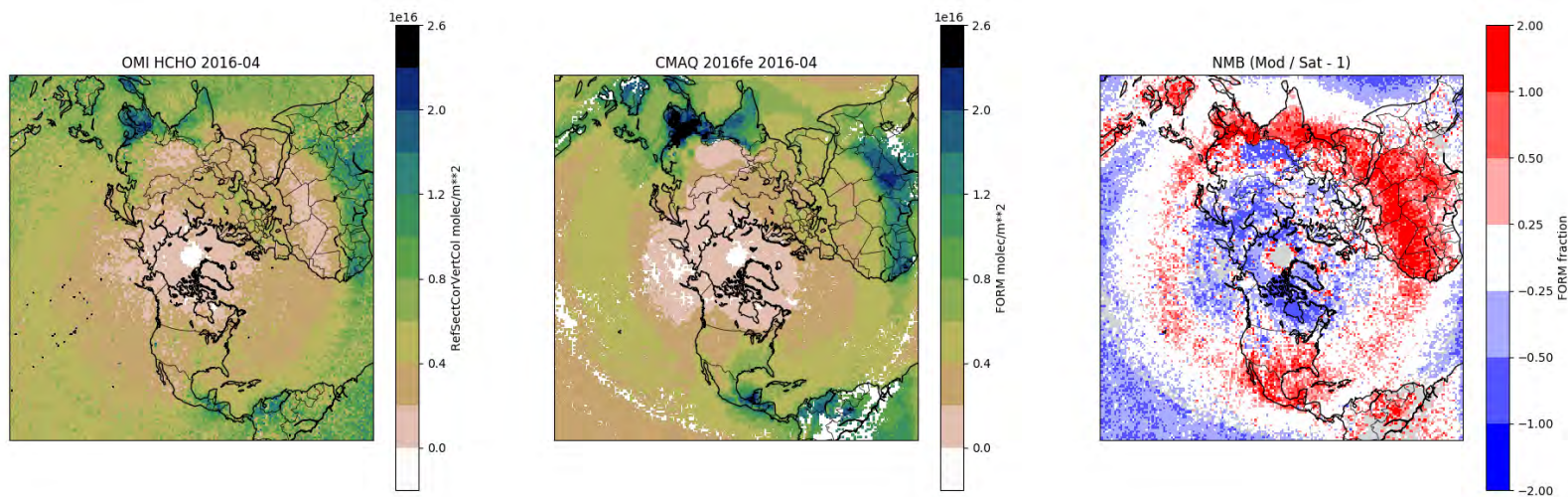


3 **Figure 2B-25. OMI Nitrogen Dioxide (OMNO2D_HR v003, left) compared to simulated (hemispheric CMAQ simulation,**
4 **center), and ratios (right) of vertical column densities for July (top) and and October (bottom).**

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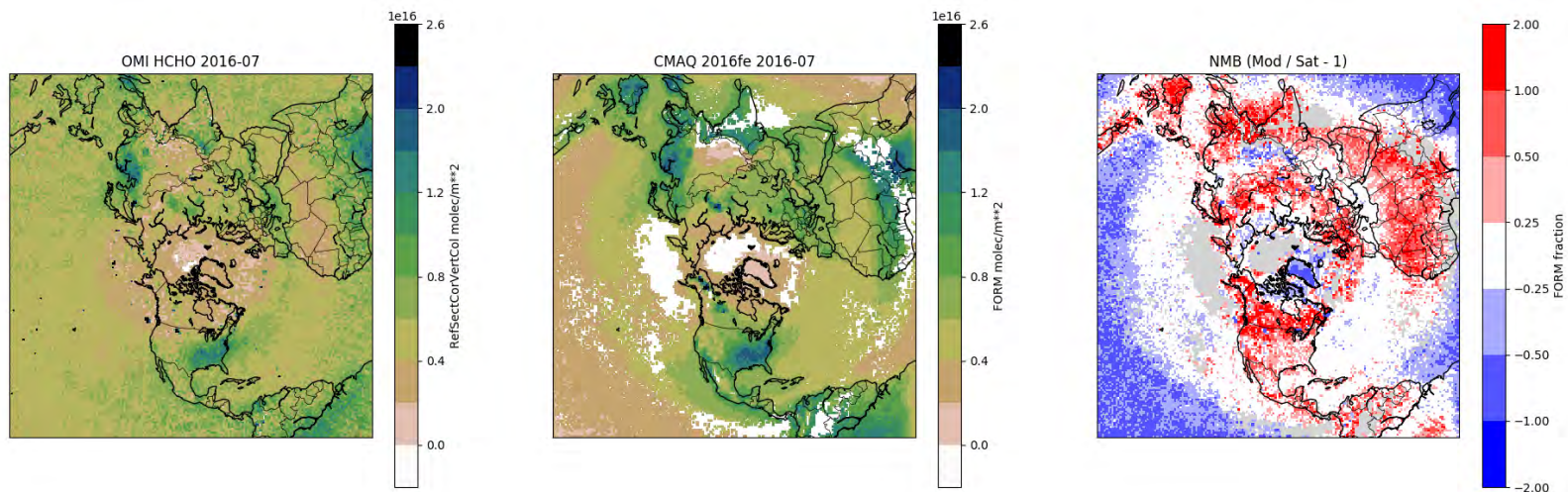
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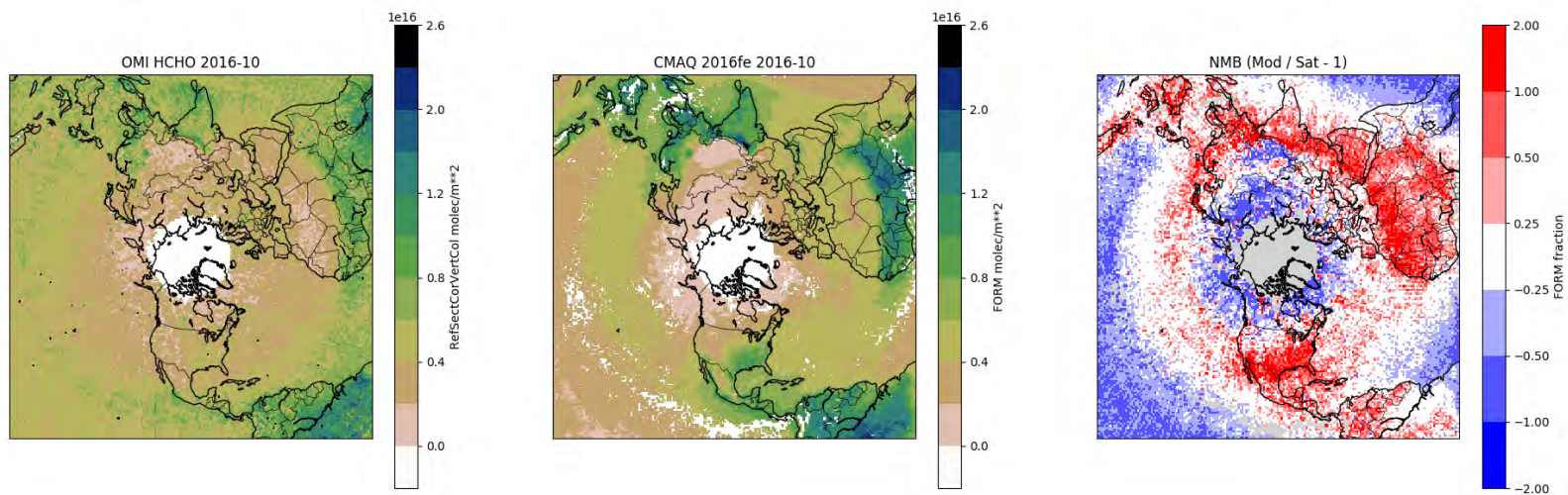
3 **Figure 2B-26. OMI Formaldehyde (OMHCHO v003, left) compared to simulated (hemispheric CMAQ simulation, center),**
4 **and ratios (right) of vertical column densities for January (top) and April (bottom).**

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3 **Figure 2B-27. OMI Formaldehyde (OMHCHO v003, left) compared to simulated (hemispheric CMAQ simulation, center),**
4 **and ratios (right) of vertical column densities for July (top), and October (bottom).**

2B.3 INTERNATIONAL CONTRIBUTIONS

This section characterizes the components of predicted international anthropogenic contributions to local O₃ concentrations and the sensitivities to model resolution. The main characterization of predicted O₃ contributions focused on results, based on simulations at a 12 km grid cell resolution, that separated Natural, International, and USA contributions to O₃. In this appendix, the International component is further characterized into some of its component parts. The component parts are only analyzed at the 108 km hemispheric resolution. First, the 108 km results are compared to the 12 km results to ensure general consistency to build confidence that, for large scale transport contributions, the 108 km characterization is relevant to the 12 km results.

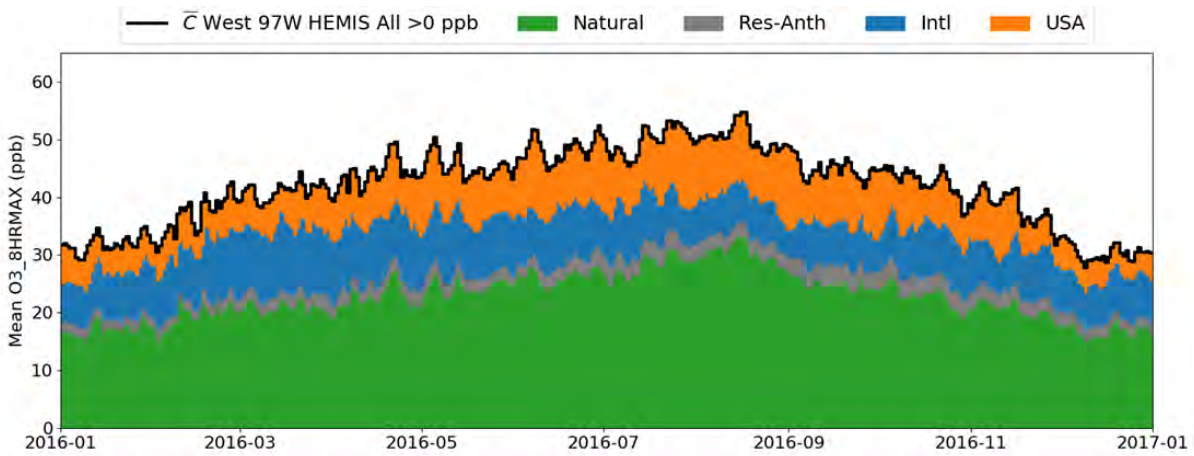
Figure 2B-28 shows the 108 km modeling averaged to the West (<97W) and East (>97W), which can be compared to the 12 km results in the main body. The results from the two modeling resolution are very consistent with very high correlation coefficients (r) for total O₃ ($r_{West}=0.987$; $r_{East}=0.989$), USA ($r_{West}=0.987$; $r_{East}=0.993$), International ($r_{West}=0.981$; $r_{East}=0.990$), and Natural ($r_{West}=0.959$; $r_{East}=0.814$). Within International, the Canada/Mexico component was separately estimated at both resolutions and agrees well for all grid cells ($r_{West}=0.966$; $r_{East}=0.935$), for high-elevation ($r_{West}=0.961$, $r_{East}=N/A$), and near-border ($r_{West}=0.961$, $r_{East}=0.947$). Since the coarser resolution model cannot resolve urban locations, the urban area weighted results have lower r (~0.8). While any particular grid cell may deviate due to local conditions, the averages across these large regions are quite consistent. The analysis is restricted to large scale averages when drawing conclusions from the 108 km analysis for the 12 km results.

Figure 2B-29 shows the predicted International contribution and some of its component parts: Canada/Mexico, China, India, and global shipping. This analysis did not attempt to quantify all International components separately, so the stacked bars generally account for only a portion of the total. However, the global shipping component of international is an overestimate as this sector includes some U.S. emissions. Global shipping includes O₃ produced within the U.S. Federal waters, which are also included in the USA contribution. As a result, the sum of components overstates shipping contributions to the total International contribution, but generally does not fully account for all components of the International contribution. The partial accounting is most obvious in the Winter and Spring when large-scale transport is most important. This suggests that during the summer, the selected components (China, India, Ships, Canada, Mexico) are a larger fraction of total International contribution. In both the East and the West, the International contribution peaks in Spring. The same seasonal signal can be seen for each International component except for Canada/Mexico. As a result, areas where

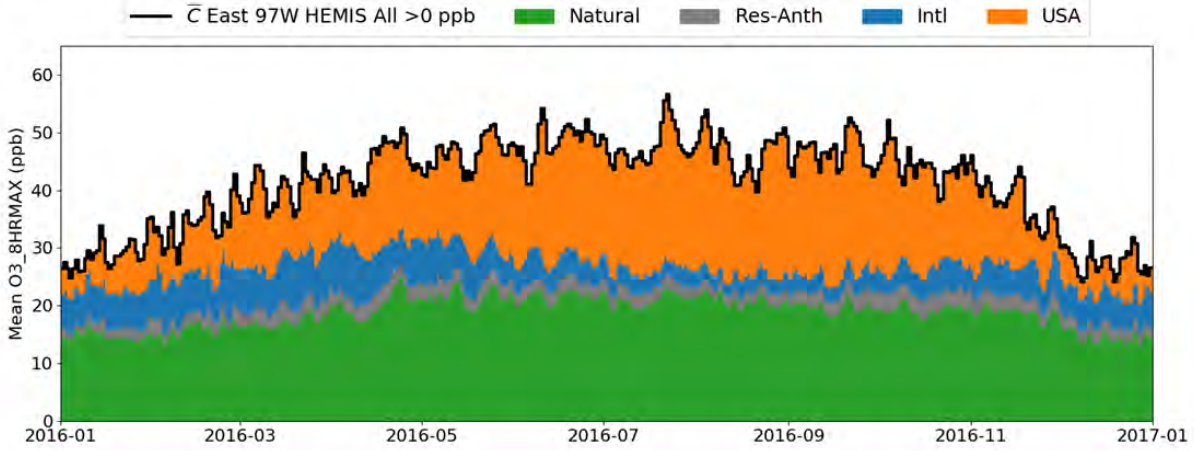
1 Canada/Mexico are more important will have a later peak of International than those influenced
 2 by the long-range components (e.g., India, China). The 108 km results cannot resolve the border
 3 well and will likely not fully capture the “near-border” effect.

4 Figure 2B-30 demonstrates the effect of International contribution on seasonality. Figure
 5 2B-30 shows the West broken out into high-elevation, near-border, and Low/Interior sites. The
 6 near-border areas have a larger Canada/Mexico component. The combination of long-range
 7 sources and Canada/Mexico create a peak International contribution at near-border sites that is
 8 one to two months later than at high-elevation or Low/Interior sites. Note that “near-border” sites
 9 are not well resolved by the 108 km simulations.

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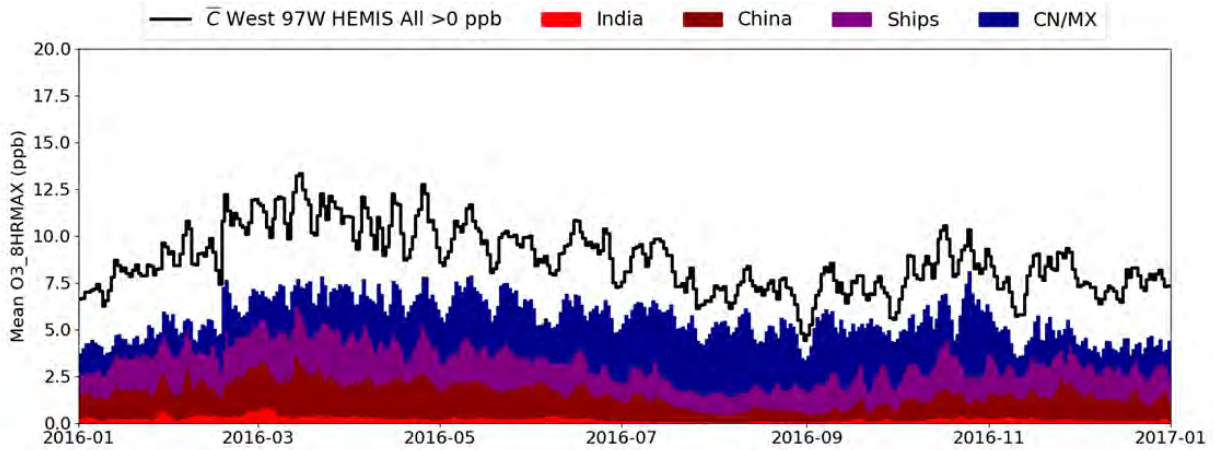


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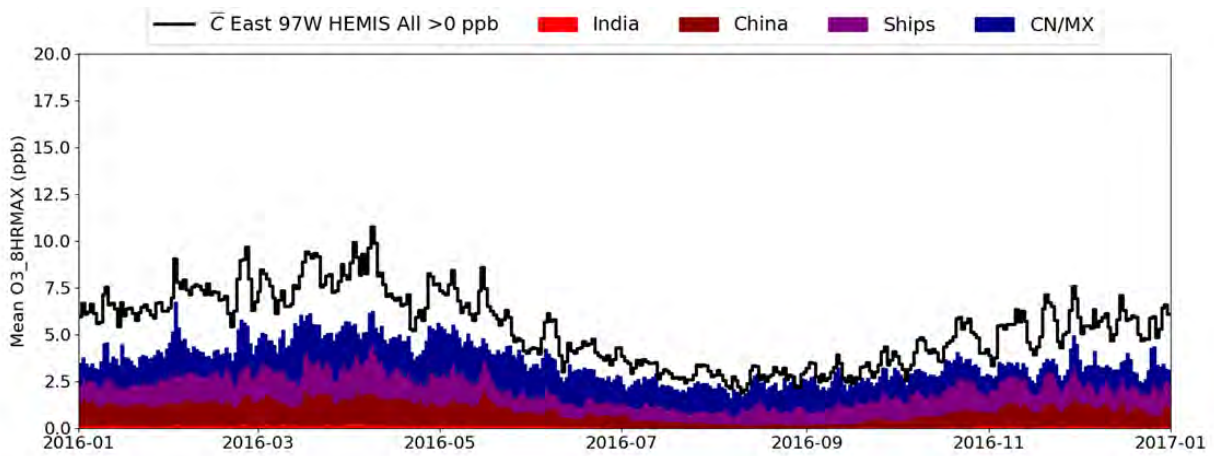
13 Average across all grid cells derived as $\bar{C} = \frac{1}{N_x} \sum_x C_x$

14 **Figure 2B-28. Total predicted MDA8 O₃ and contributions (see legend) over time in the**
 15 **West (top), and all East (bottom) averaged over all grid cells and days in the**
 16 **U.S.**

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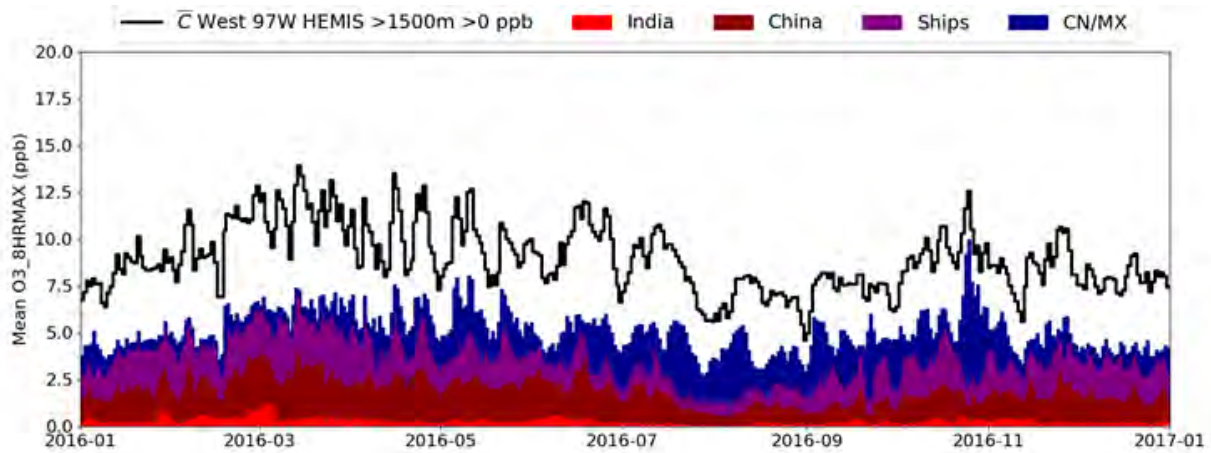
Average across all grid cells derived as $\bar{C} = \frac{1}{N_x} \sum_x C_x$

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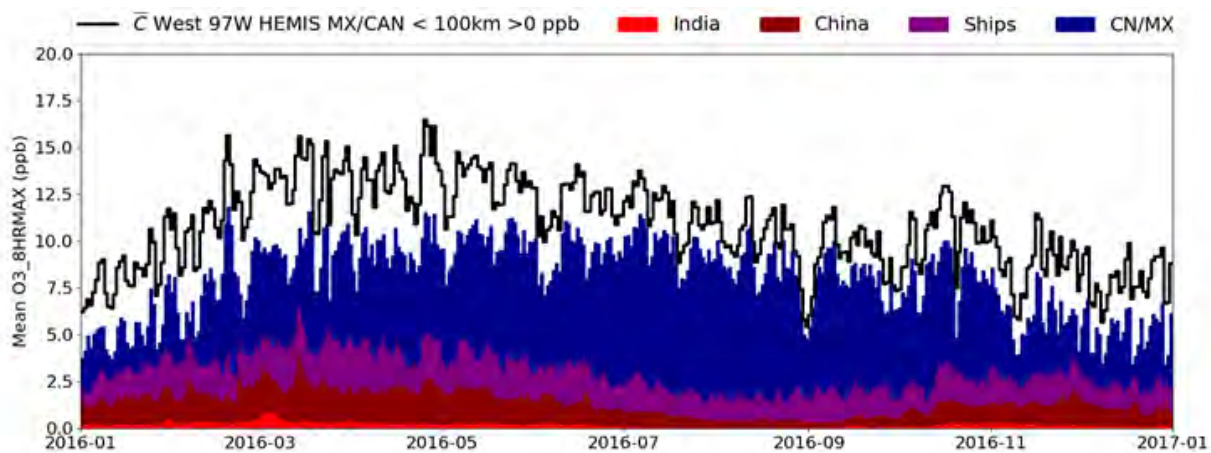
Figure 2B-29. International contribution (black line) to predicted MDA8 O₃ and components (see legend) over time in the West (top), and all East (bottom) averaged over all grid cells and days in the U.S.

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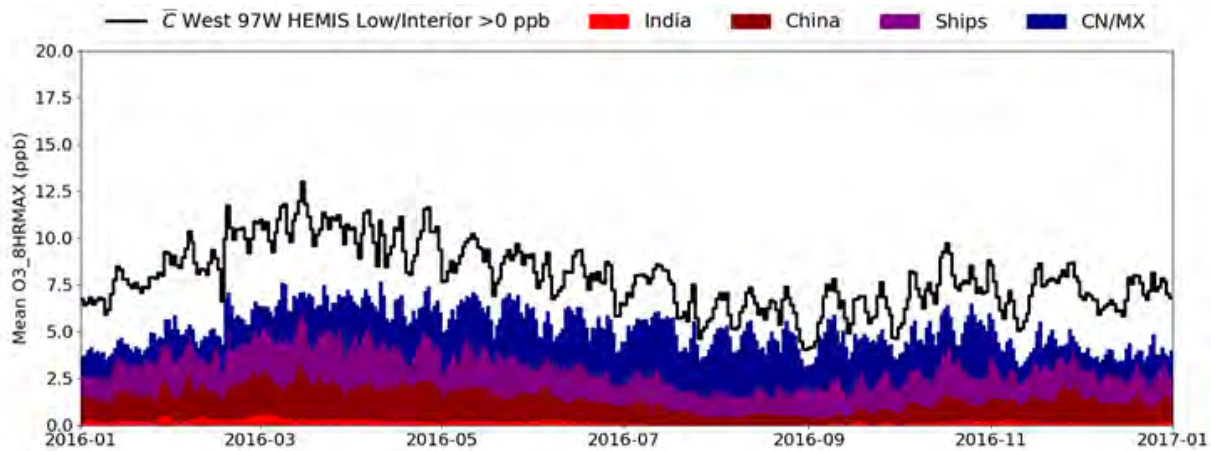
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$$\bar{C} = \frac{1}{N_x} \sum_x C_x$$

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Figure 2B-30. International contribution (black line) to predicted MDA8 O₃ and components (see legend) over time averaged over all grid cells in the West at high elevation (top), near-border sites (middle), and Low/Interior sites (bottom).

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APPENDIX 3A

DETAILS ON CONTROLLED HUMAN EXPOSURE STUDIES

3A.1. OVERVIEW

This appendix gives further study-specific details of the range of respiratory effects (with a particular focus on pulmonary function) in controlled human O₃ exposures during exercise. In these studies, the magnitude or severity of the respiratory effects induced by O₃ was influenced by ventilation rate, exposure duration, and exposure concentration. Because ventilation rates increase with increased physical activity level, the exposure concentrations eliciting a significant response in exercising subjects are lower than in subjects exposed while at rest (ISA, Appendix 3, section 3.1.4.2.1).

Table 3A-1 presents the O₃ induced change in forced expiratory volume in one second (FEV₁) in 6.6 to 8-hour controlled human exposure studies (involving quasi-continuous or intermittent exercise). The FEV₁ values presented are derived by subtracting the percent changes in mean FEV₁ in response to filtered air exposure with exercise from the corresponding percent changes in FEV₁ in response to O₃ exposure with exercise. The controlled human exposure studies presented involve exposures, with intermittent exercise, of duration 6 to 8 hours and target exposure concentrations ranging from 0.04 to 0.16 ppm O₃. Study design variables are also described in Table 3A-1 and include mode of exposure (chamber or facemask), whether the exposure concentration is constant or varying, exposure duration, exercise duration, and minute ventilation rate normalized by body surface area during exercise (equivalent ventilation rate,¹ or EVR). Table 3A-2 provides further details of individual study design protocols and subject characteristics for the studies summarized in Table 3A-1.

Table 3A-3 summarizes studies of controlled human exposure to O₃ for shorter durations (1 to 3 hours) during continuous or intermittent exercise in contrast to similar exposure durations at rest. The table presents reported effects related to pulmonary function, airway responsiveness, respiratory symptoms, inflammation and/or host defense. Key study design variables are also described and include exposure concentrations (ranging from 0.07 to 0.40 ppm O₃ for studies during exercise and 0.10 to 1.00 ppm for studies of subjects at rest), ventilation characteristics during exercise and subject characteristics (sex and health status). This table was adapted from Tables 7-1, 7-2 and 7-10 in the 1996 AQCD (U.S. EPA, 1996) and Table AX6-1 in the 2006 AQCD (U.S. EPA, 2006), with additional studies from Tables AX6-8 through AX6-13 in the 2006 AQCD, as well as more recent studies from the 2013 ISA (U.S. EPA, 2013) and 2020 ISA (U.S. EPA, 2020).

¹ The EVR is derived by dividing the minute ventilation rate (\dot{V}_E in L/min) by body surface area in m². Values reflect the study mean EVR across the six exercise periods except for R11, as described below.

1 **Table 3A-1. Cross-study comparison of mean O₃-induced FEV₁ decrements in 6.6 to 8-**
 2 **hour controlled human exposure studies (that include periods of exercise).**

Exposure Design ^C	Ref ^D	EVR ^E (L/min -m ²)	Δ FEV ₁ ^{A, B} (%)								
			Average Target Ozone Concentration During Exercise Periods (ppm) ^F								
			0.04	0.06	0.07	0.08	0.087	0.10	0.12	0.16	
6.6 Hour Chamber: Six 50-min exercise periods, each followed by 10 min rest; 35 min rest-lunch after 3rd hour.	[constant]	R1	20		-2.85*		-6.06*				
		R2	20		-1.71*		-3.46 [^]				
		R3	20				-7.45*		-8.45*	-13.14*	
		R4	20				-6.17*				
		R5	19							-15.65*	
		R6	22							-14.92*	
		R7	20				-7.71*		-13.88* ^G		
		R8	20							-12.79*	
	[varying]	R1	20	-0.17	-2.78		-6.99*				
	R4	20				-5.77*					
	R9	20		-3.52	-6.14*	-7.82*	-12.23*				
6.6 Hour with 6-hour facemask exposure: Six 60-min periods each consisting of 50 min of exercise and 10 min of rest, each followed by 3 min testing period without exposure; 24 min lunch without exposure after 3rd hour.	[constant]	R4	20				-6.14*				
		R5	20	-1.24			-6.35*			-15.41*	
		R10	17							-11.28*	
		R10	20							-13.69*	
		R10	23							-15.88*	
		R11	18 [#]							-11.00*	
	R11	20 ¹⁵⁻²³							-13.68*		
	[varying]	R4	20				-5.45*				
		R11	10 ⁹⁻¹²				0.80				
		R11	12 ⁷⁻¹¹							-3.50	
R11		18 ^{#, X}							-13.96*		
	R11	18 ^{#, Y}							-10.31*		
7.6 Hour Chamber: additional hour onto 6.6 hr protocol above.	[constant]	R12	15							-9.8*	
		R12 ^{As}	14							-19.4*	
8-Hour Chamber: Eight 30-min exercise periods, each followed by 30 min rest	[constant]	R13	20							-8.13*	
		R14	20							-4.07*	
	[varying] ^T	R13	20							-6.73*	
		R14	20							-5.62*	

^A Values reflect O₃-induced percent change in FEV₁ at the group mean level, based on subtraction of the filtered air percent change (post-pre exposure) from the O₃ % change in FEV₁. For studies R1, R2, R4, R5 and R9, Δ FEV₁ values were calculated from individual subject data provided by author. Δ FEV₁ values for R3, R6, R12 were calculated from individual subject data in publication; R7, R8, R10, R11, R13 and R14 Δ FEV₁ values were derived from group mean response provided in publication. Statistically significant findings are indicated by asterisk (*). A lack of statistical testing is indicated by (^). Unless indicated otherwise, all studies were in healthy adults.

^B In addition to Δ FEV₁, some studies reported respiratory symptoms scores (e.g. cough and pain on deep inspiration). The exposures with statistically significant increase in respiratory symptoms scores are indicated by orange shading (■). Blue shading (■) indicates symptom scores that were not statistically significant from filtered air.

^C Exposure designs with nonvarying exposure concentrations are indicated by [constant], while studies involving different O₃ concentrations for different periods of exposures are indicated by [varying]. [varying]^T denotes triangular wave exposure concentrations (0.07 ppm->0.16 ppm->0.10 ppm). Further details on concentrations are provided in Table 3A-2.

^D R1=Adams (2006b) and Brown et al. (2008); R2=Kim et al. (2011) and McDonnell et al., 2012; R3=Horstman et al. (1990); R4=Adams (2003); R5=Adams (2002); R6=Folinsbee et al. (1988); R7=McDonnell et al. (1991); R8=Folinsbee et al. (1994); R9=Schelegle et al. (2009). R10=Adams (2000); R11=Adams and Ollison (1997); R12=Horstman et al. (1995). R12^{As} refers to subjects with asthma; R13=Adams (2006a); R14=Hazucha et al. (1992).

^E The average mean EVR during exercise periods (calculated from study-reported information, see also Table 3A-2).

indicates value derived as average of reported mean hourly EVR (which included 50 minutes exercise and 10 minutes rest) (although study protocol indicated EVR of 20 L/min-m²).

¹⁵⁻²³ indicates hourly ventilation rate varied from 15-23 L/min-m²; value presented is the average mean EVR across the entire experimental period (including both exercise and rest periods).

⁹⁻¹² indicates hourly ventilation rate varied from 9-12 L/min-m²; value presented is average mean EVR across the entire experimental period (including both exercise and rest periods).

⁷⁻¹¹ indicates hourly ventilation rate varied from 7-11 L/min-m²; value presented is the average mean EVR across the entire experimental period (including both exercise and rest periods).

^X and ^Y refer to two different varying concentration protocols (Details on concentrations are provided in Table 3A-2.)

^F Author's target for average O₃ concentrations across the six exercise periods. This differs from the time-weighted average concentration (based on target or measurements) for full exposure period. For example, as shown in Table 3A-2 in chamber studies implementing a varying concentration protocol with targets of 0.03, 0.07, 0.10, 0.15, 0.08 and 0.05 ppm, the exercise period average concentration is 0.08 ppm while the TWA for the full exposure period (based on targets) is 0.82 ppm due to the 0.6 hour lunchtime exposure to 0.10 ppm between periods 3 and 4.

^G Results at 0.08 ppm for a subset of the study subjects that were exposed to 0.10 ppm.

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1 **Table 3A-2. Study-specific details of O₃ exposure protocols for 6.6 to- 8-hour controlled**
 2 **human exposure studies (that include periods of exercise).**

Ref ^A	EVR ^B during exercise (L/min-m ²)	Target Exposure Concentration ^C (ppm)		Number of Subjects ^E	Avg. Age (Range)	Reference
		Constant, (6.6-hr TWA) ^D	Varying (hourly concentrations), (6.6-hr TWA) ^D			
6.6-Hour Chamber Study: 50m+10m, 50m+10m, 50m+10m, 35m, 50m+10m, 50m+10m, 50m+10m						
Face Mask Exposure (FM): 50m+10m, 3m, 50m+10m, 3m, 50m+10m, 24m, 50m+10m, 3m, 50m+10m, 3m, 50m+10m						
<i>red=O₃ exposure, black = no exposure (i.e., no facemask) bold =exercise periods, non-bold=rest periods</i>						
R1	20	0.06 0.08	0.04 (0.03, 0.04, 0.05, 0.05, 0.04, 0.03), (0.041) 0.06 (0.04, 0.07, 0.09, 0.07, 0.05, 0.04), (0.063) 0.08 (0.03, 0.07, 0.10, 0.15, 0.08, 0.05), (0.082)	30 (15M,15F)	23 (21-29)	Adams (2006b) Brown et al. (2008)
R2	20	0.06 0.08		59 (27M,32F) 30 (15M,15F)	25 (19-35)	Kim et al. (2011) ^F
R3	20	0.08 0.10 0.12		22 (M)	25 (18-35)	Horstman et al. (1990)
R4	20	0.08 0.08 ^{FM} , (0.073)	0.08 (0.03, 0.07, 0.10, 0.15, 0.08, 0.05), (0.082) 0.08 ^{FM} (0.03, 0.07, 0.10, 0.15, 0.08, 0.05), (0.073)	30 (15M,15F)	22	Adams (2003)
R5	19-20	0.04 ^{FM} , (0.036) 0.08 ^{FM} , (0.073) 0.12 ^{FM} , (0.109) 0.12		30 (15M,15F)	22	Adams (2002)
R6	22	0.12		10 (M)	25 (18-33)	Folinsbee et al. (1988)
R7	20	0.08 0.08+0.10		38 (M) 10 (M)	25 (18-30)	McDonnell et al. (1991)
R8	18, 20	0.12		17 (M)	25	Folinsbee et al. (1994)
R9	20		0.06 (0.04, 0.07, 0.07, 0.09, 0.05, 0.04), (0.061) ^G 0.07 (0.05, 0.07, 0.08, 0.09, 0.08, 0.05), (0.071) ^G 0.08 (0.03, 0.07, 0.10, 0.15, 0.08, 0.05), (0.082) ^G 0.087 (0.04, 0.08, 0.09, 0.12, 0.10, 0.09), (0.087) ^G	31 (15M,16F)	21 (18-25)	Schelegle et al. (2009)
R10	17, 20, 23	0.12 ^{FM} , (0.109)		30(15M, 15F)	22	Adams (2000)
R11	10 ⁹⁻¹² , 11 ⁷⁻¹¹ , 18 [#] , 20 ¹⁵⁻²³	0.08 ^{FM} , (0.073) 0.12 ^{FM} , (0.109)	0.12 ^{FM} (0.07, 0.16, 0.10), (0.109) 0.12 ^{FM} (0.115, 0.115, 0.130, 0.130, 0.115, 0.115), (0.109)	12 (6M, 6F)	22	Adams and Ollison (1997)
7.6-hour Chamber: Additional hour on 6.6 hr chamber protocol above.						
R12	15-17	0.16		13 (NR) 17As(7M,10F)	25 (18-35)	Horstman et al. (1995)
8-hour Chamber: Eight 30-min exercise periods, each followed by 30 min rest						
R13	20	0.12	0.12 triangular* (0→0.24→0)	30 (15M,15F)	23 (21-29)	Adams (2006a)
R14	20	0.12	0.12 triangular* (0→0.24→0)	23 (M)	26 (20-35)	Hazucho et al. (1992)
^A R1-R14 matches study codes in Table 3A-1.						
^B EVR values are the study means during exercise periods except for R11, for which the EVRs are described below. ⁹⁻¹² indicates the study protocol varied the hourly ventilation rate from 9-12 L/min-m ² and value reflects the average mean EVR across the 6-hr experimental period which includes 50-min of exercise and 10 min of rest. ⁷⁻¹¹ indicates the study protocol varied the hourly ventilation rate from 7-11 L/min-m ² and the value reflects the average mean EVR across the 6-hr experimental period which includes 50-min of exercise and 10 min of rest.						

The study protocol describes the target exercise EVR as 20 L/min-m² but the actual mean EVR during exercise was not reported and could not be calculated from study data presented. The value was derived from the average of the mean hourly EVR which consisted of 50-min of exercise and 10-min of rest resulting in an EVR somewhat lower than the target of 20 L/min-m².

¹⁵⁻²³ indicates the study varied the hourly ventilation rate from 15-23 L/min-m²; and the value reflects the average mean EVR across the 6-hr experimental period which includes 50-min of exercise and 10 min of rest.

^c Unless marked by "F" (for face mask exposure), exposures were conducted in exposure chamber.

^d TWA (time weighted average) was calculated taking into account all exposure concentrations during experiment, including lunch and rest periods. The TWA concentrations for facemask exercise protocols (whether the exposure concentration was constant or varying) are lower than the target exposure concentrations because the subjects were not exposed to O₃ during the 3 minute rest and 24 minute lunch periods. Conversely, the TWA concentrations for varying exposure chamber protocols were higher than the targeted average exposure because of the sequence of concentrations, and their relative magnitude during the 35 minute lunch period.

^e All subjects were healthy adults unless marked by "As" for subjects with asthma. M=male, F=female, NR=sex not reported.

^f The 0.08 ppm data for the Kim study were reported in McDonnell et al., 2012.

* Triangular = steadily increasing concentration from 0 ppm to 0.24 ppm at hour 4, then back to 0 ppm.

^g While Schelegle et al. (2009) reported measured O₃ concentrations, the TWA target concentrations listed in the table for the four protocols are 0.061, 0.071, 0.082 and 0.087. Based on the O₃ concentration measurements taken during the 6 exercise periods, the average O₃ concentrations for the four protocols are 0.063 ppm, 0.072 ppm, 0.081 ppm and 0.088 ppm, while the 6.6-hour TWA concentrations are 0.063 ppm, 0.073 ppm, 0.083 ppm and 0.088 ppm

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Table 3A-3. Summary of controlled human exposures to O₃ for 1 to 3 hours during exercise or at rest.

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
Adult Subjects During Moderate to Heavy Exercise					
0.07	3 hr IE (6 ×15 min, EVR=15-17 L/min-m ²)	H ^{NS}	35M and 52F (55-70 yrs)	PF: No significant change in FEV ₁ SY: No significant change IF: No significant change	Arjomandi et al., 2018 Frampton et al., 2017; 2020 ISA U.S. EPA, 2020
0.08	1 hr CE (mean \dot{V}_E =57 L/min)	H ^{At}	42M and 8F (mean 26 yrs)	PF: No significant change in FEV ₁ SY: No significant change	Avol et al., 1984 ^F
0.08	2 hr IE (4×15 min, \dot{V}_E =68 L/min)	H	24M (18-33 yrs)	PF: No significant change in FEV ₁ SY: No significant change	Linn et al., 1986; 1996 AQCD, Table 7-1
0.10	0.5 hr (8 km time trial at 70% HR at 20°C and 31°C)	H ^{At}	9M (mean 24 yrs)	IF: NL 15 min postexposure showed no differences in inflammatory response between heat only or O ₃ only compared to control; significantly increased in nasal Club cells and glutathione after high-temperature O ₃ relative to lower temperature FA control.	Gomes et al., 2011 2020 ISA, p. 3-30, Table 3-9
0.10	1 hr IE (2 × 15 min, \dot{V}_E =27 L/min)	As ^M	12M and 9F (19-40 yrs)	PF/AR: No significant differences in FEV ₁ or FVC compared to FA and no exacerbation of exercise-induced asthma in a postexposure exercise challenge SY: No significant change	Weymer et al., 1994; 1996 AQCD, Table 7-2
0.10	2 hr Mild IE	H	12M and 10F; (mean 30 yrs)	PF: No significant change in FEV ₁ IF: Markers of exposure in exhaled breath condensate including markers of inflammation (8-isoprostane, TBARS and LTB ₄), and markers of oxidative stress (ROS-DNA interaction: 8-OHdG), increased in a sub-set of NQO1 wildtypes and GSTM1 null subjects	Corradi et al., 2002; 2006 AQCD, Table AX6-12
0.10	2 hr IE (4×15 min, \dot{V}_E =68 L/min)	H	24M (18-33 yrs)	PF: No significant change in FEV ₁ SY: No significant change	Linn et al., 1986; 1996 AQCD, Table 7-1
0.10	2 hr IE (4×15 min at either \dot{V}_E =30 L/min, \dot{V}_E =50 L/min or \dot{V}_E =70 L/min)	H	30M (three groups of 10) (19-28 yrs)	PF: No significant change in any of the three 10-male groups separately exposed via three ventilation rates	Folinsbee et al., 1978; ^G 1996 AQCD, p. 7-10
0.10	2 hr IE (4×14 min, \dot{V}_E =70 L/min)	H ^{NS}	20M (mean 25 yrs)	PF: No significant change AR: No significant change in sRAW SY: No significant change	Kulle et al., 1985; 1996 AQCD, Table 7-1
0.10	3 hr IE (6×15 min, EVR=25 L/min-m ²)	H ^{NS}	15M and 9F (18-40 yrs)	PF: No significant change SY: No significant change	Frampton et al., 2015; 2020 ISA, p. 3-15, Table 3-4

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.12	45 min IE ($\dot{V}_E=40-46$ L/min) (two sequential 10 min exposures to 0.1 and 0.25 ppm SO ₂); +/- 4 wk pre- treatment with antioxidant	As ^{SO2}	5M and 12F (19- 38 yrs)	PF: ↓ FEV ₁ * with no significant differences due to O ₃ between placebo and antioxidant supplement AR: No significant differences due to O ₃ in placebo vs, antioxidant pretreatment in bronchial hyperresponsiveness to 0.1 ppm SO ₂ .	Trenga et al., 2001; 2006 AQCD, p. 6-67 and Table AX6-7
0.12	1 hr CE (30 min warm up $\dot{V}_E=54$ L/min, 30 min competitive $\dot{V}_E=120$ L/min; overall mean $\dot{V}_E=87$ L/min)	H ^{Ath}	10M (19-29 yrs)	PF: No significant change in pulmonary function compared to FA SY: No significant symptoms	Schelegle and Adams, 1986; 1996 AQCD, p. 7-11, Table 7-1
0.12	1 hr CE (mean $\dot{V}_E=89$ L/min)	H ^{Ath}	15M and 2F (19-30 yrs)	PF: ↓ FEV ₁ ^J AR: > 20% increase in histamine responsiveness in one subject SY: Mild respiratory symptoms	Gong et al., 1986; 1996 AQCD, Tables 7-1 and 7-10; 2013 ISA, p. 6-6
0.12	1.5 hr IE (3×15 min, $\dot{V}_E=20$ L/min)	As ^A H ^{NAs}	5M and 5F 4M and 4F (18-41 yrs)	NL immediately and 24 hr after exposure PF: No change in lung or nasal function. IF: No change in PMN number	McBride et al., 1994; 2006 AQCD, Table AX6-12
0.12	3 hr IE (6×15 min, EVR=15-17 L/min/m ²)	H ^{NS}	35M and 52F (55-70 yrs)	PF: Small statistically significant attenuation of exercise-related increases FEV ₁ and FVC SY: No significant change IF: Significant increase in PMN independent of GSTM1 phenotype and significant increase in plasma CC16 (marker of airway epithelial injury) 4 hr and 22hr postexposure	Arjomandi et al., 2018 Frampton et al., 2017; 2020 ISA, p.3-30, Table 3-4
0.12	2 hr IE (4×15 min, EVR=20 L/min-m ²)	H ^{NS}	9M and 3F (mean 28 yrs)	PF: No changes in FEV ₁ or FVC IF: Increased percentage of vessels expressing P-selectin in bronchial biopsies 1.5 hr postexposure; no change in BAL markers, PMNs or expression of VCAM-1, E-selectin or ICAM-1 in vessel biopsies	Krishna et al., 1997; 2006 AQCD, Table AX6-12
0.12	2 hr IE (4×15 min, $\dot{V}_E=68$ L/min)	H	24M (18-33 yrs)	PF: No significant change in FEV ₁ SY: No significant change in respiratory symptoms	Linn et al., 1986; 1996 AQCD, Table 7-1
0.12	2.5 hr IE (4×15 min, $\dot{V}_E=68$ L/min)	H	22M (18-30 yrs)	PF: ↓ FVC*, ↓ FEV ₁ * and ↓ FEF ₂₅₋₇₅ * AR: No significant change in sRaw SY: Increased respiratory symptoms	McDonnell et al., 1983; 1996 AQCD, p. 7-15, Table 7-1
0.12	2.5 hr IE (4×15 min, EVR=25 L/min- m ²)	H	30M and 31F (18-35 yrs)	PF: ↓ FEV ₁ * compared with FA AR: No significant change in sRaw SY: No significant change	Seal et al., 1993; 1996 AQCD, p. 7-15, Table 7-1
0.125	3 hr IE (6×15 min, $\dot{V}_E=26$ L/min)	H As ^M	10M and 11F (mean 28 yrs) 5M and 10F (mean 30 yrs)	PF: No significant change in pulmonary function. IF: Small but significant neutrophil increases in As ^M subjects	Holz et al., 1999; 2006 AQCD, p. AX6-35 and Table AX6-3

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.125	3 hr IE (4×15 min, $\dot{V}_E=30$ L/min); 3 hr IE (4×15 min, $\dot{V}_E=30$ L/min) × 4 days; *challenged with allergen 20 hr following the last exposure and sputum collected 6-7 hr later	As ^A AI	6M and 5F (20- 53 yrs) 16M and 6F (19-48 yrs)	PF: Incidence and magnitude of early-phase FEV ₁ decrements to allergen were significantly greater in AI subjects exposed for 4 days. IF: Significant increase in sputum eosinophils in As ^A and AI subjects exposed for 4 days: increased sputum lymphocytes, mast cell tryptase, histamine, and LDH only in As ^A subjects exposed for 4 days.	Holz et al., 2002; 2006 AQCD, Tables AX6-3 and AX6-11
0.14	2 hr IE (4×15 min, $\dot{V}_E=68$ L/min)	H	24M (18-33 yrs)	PF: No significant change in FEV ₁ SY: No significant change in respiratory symptoms	Linn et al., 1986; 1996 AQCD, Table 7-1
0.15	2 hr IE (4×14 min, $\dot{V}_E=70$ L/min)	H ^{NS}	20M (mean 25 yrs)	PF: ↓ FEV ₁ * AR: 6 subjects with >15% decrease in sGaw SY: No significant change in respiratory symptoms	Kulle et al., 1985; 1996 AQCD, Table 7-1
0.16	1 hr CE (mean $\dot{V}_E=57$ L/min)	H ^{AI}	42M and 8F (mean 26 yrs)	PF: Small ↓ FEV ₁ * SY: ↑ in mild respiratory symptoms*	Avol et al., 1984; 1996 AQCD, Table 7-1
0.16	2 hr IE (4×15 min, $\dot{V}_E=68$ L/min)	H	24M (18-33 yrs)	PF: Small ↓ FEV ₁ * SY: No significant change in respiratory symptoms	Linn et al., 1986; 1996 AQCD, p. 7-11 and Table 7-1
0.18	1 hr CE (30 min warm up $\dot{V}_E=54$ L/min, 30 min competitive $\dot{V}_E=120$ L/min; overall mean $\dot{V}_E=87$ L/min)	H ^{AI}	10M (19-29 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * compared to FA; ↓ exercise time for subjects unable to complete simulation SY: ↑ respiratory symptoms*	Schelegle and Adams, 1986; 1996 AQCD, p. 7-11, Table 7-1
0.18	2 hr IE (4×15 min, EVR=35 L/min-m ²)	AI	26M with (18-30 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ * AR: ↑ sRaw* and increased reactivity to histamine* SY: ↑ respiratory symptoms*	McDonnell et al., 1987; 1996 AQCD, Table 7-2
0.18	2.5 hr IE (4×15 min, EVR=25 L/min- m ²)	H	32M and 32F (18-35 yrs)	PF: ↓ FEV ₁ * compared with FA AR: ↑ sRaw* compared with FA SY: ↑ respiratory symptoms* compared with FA	Seal et al., 1993; 1996 AQCD, p. 7-15, Table 7-1
0.18	2.5 hr IE (4×15 min, $\dot{V}_E=65$ L/min)	H	20M (18-30 yrs)	PF: ↓ FVC*, ↓ FEV ₁ * and ↓ FEF ₂₅₋₇₅ * AR: No significant change in sRaw SY: ↑ respiratory symptoms*	McDonnell et al., 1983; 1996 AQCD, p. 7-15, Table 7-1
0.20	30 to 80 min CE ($\dot{V}_E=33$ or 66 L/min)	H	8M (22-46 yrs)	PF: O ₃ effective dose significantly related to pulmonary function decrements (threshold for significant responses > 0.2 ppm) and exercise ventilatory pattern changes; O ₃ concentration accounted for the majority of the pulmonary function variance	Adams et al., 1981; 1996 AQCD, Table 7-1
0.20	1 hr CE ($\dot{V}_E=80$ L/min); 1 hr competitive simulation (30 min at $\dot{V}_E=52$ L/min, 30 min at $\dot{V}_E=100$ L/min; overall mean $\dot{V}_E=77.5$ L/min)	H ^{AI}	10M (19-31 yrs)	PF: ↓ FVC*, ↓ FEV ₁ * and ↓ FEF ₂₅₋₇₅ * compared to FA with both protocols; ↓ V _T * and ↑ f _R * with CE SY: ↑ respiratory symptoms*	Adams and Schelegle, 1983; 1996 AQCD, Table 7-1

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.20	1 hr CE ($\dot{V}_E=89$ L/min)	H ^{Ath}	15M and 2F (19-30 yrs)	PF: $\downarrow V_{E_{max}}$, $\downarrow VO_{2max}$, $\downarrow V_{Tmax}$, \downarrow work load*, \downarrow ride time*, \downarrow FVC*, and \downarrow FEV ₁ * compared with FA AR: > 20% increase in histamine responsiveness in nine subjects SY: \uparrow respiratory symptoms*	Gong et al., 1986; 1996 AQCD, Tables 7-1 and 7-10
0.20	1hr CE (mean $\dot{V}_E=60$ L/min); 2 exposures \times 24 hr apart	H ^{NS}	15M (mean 25 yrs)	PF: Consecutive days of exposure produced similar \downarrow FVC* and \downarrow FEV ₁ * on each day compared to FA SY: Consecutive days of exposure produced similar \uparrow respiratory symptoms*	Brookes et al., 1989; 2006 AQCD, Table AX6-9
0.20	2 hr IE (4 \times 15 min, 2 \times resting \dot{V}_E)	H ^{NS}	12M and 7F (21- 32 yrs)	AR: No change in sRaw to a 10-breath histamine (1.6%) aerosol challenge after O ₃ exposure.	Dimeo et al., 1981; 2006 AQCD, Table AX6-11
0.20	2 hr IE (4 \times 15 min, $\dot{V}_E=20$ L/min)	As ^A	4M and 5F (21-42 yrs)	PF: \downarrow FEV ₁ * but not FVC AR: No change in sRaw IF: 6 hr postexposure \uparrow PMNs* with no change in permeability markers; 24 hr postexposure PMNs decreased while albumin, total protein, myeloperoxidase and eosinophil cationic protein increased.	Newson et al., 2000; 2006 AQCD, Tables AX6-3, AX6-13
0.20	2 hr IE (4 \times 15 min, $\dot{V}_E=30$ L/min)	H ^{NS}	10M and 2F (mean 28 yrs)	IF: Significant increase in PMNs and epithelial cells, IL-8, Gro- α , and total protein in BAL fluid; % PMNs correlated positively with chemokine levels; significant decrease in the CD4 ⁺ /CD8 ⁺ ratio and % of activated CD4 ⁺ and CD8 ⁺ T cells in BAL fluid.	Krishna et al., 1998 2006 AQCD, Table AX6-13
0.20	2 hr IE (4 \times 15 min, EVR=20 L/min-m ²)	H ^{NS}	8M and 5F (20-31 yrs)	PF: \downarrow FVC*, \downarrow FEV ₁ *, and \downarrow FEF ₂₅₋₇₅ * IF: Spirometry responses did not predict inflammatory responses; increased adhesion molecule expression, submucosal mast cell numbers and alterations in lining fluid redox status; increase in human leukocyte antigen+ alveolar macrophages in BAL 1.5 hr postexposure.	Blomberg et al., 1999; 2006 AQCD, Tables AX6-1 and AX6-12
0.20	2 hr IE (4 \times 15 min, EVR=20 L/min-m ²)	H	10M and 12F (mean 24 yrs)	PF: \downarrow FEV ₁ * immediately postexposure but not significantly different from baseline 2 hr later. IF: Elevated CC16 levels remained high 6 hr postexposure but returned to baseline by 18 hr postexposure. No correlation between CC16 and FEV ₁ decrement.	Blomberg et al., 2003; 2006 AQCD, Table AX6-1
0.20	2 hr IE (4 \times 15 min, EVR=20 L/min-m ²) chronic inhaled corticosteroid	As	8M and 5F (mean 33 yrs)	PF: \downarrow FEV ₁ * and \downarrow FVC* AR: Significant increase sRaw IF: Significant increase in BAL neutrophils, but not eosinophils 18 hr postexposure; significant increase in mast cells in bronchial biopsy	Stenfors et al., 2010; 2013 ISA, p. 6-21

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.20	2 hr IE (4×15 min, EVR=20 L/min-m ²)	H ^{NAs} As ^M	6M and 9F (19-32 yrs); 9M and 6F (21-48 yrs)	PF: ↓FEV ₁ * (8%, H ^{NAs} ; 3% As ^M) and ↓FVC* in both groups with no significant difference between H ^{NAs} and As ^M IF: Significant increase in PMN in both groups with no significant difference between As ^M and H ^{NAs} 6 hr postexposure; no relationship between antioxidant levels and spirometric or cellular responses	Mudway et al., 2001; Stenfors et al., 2002; 2006 AQCD, Table AX6-1
0.20	2 hr IE (4×15 min, EVR=20 L/min-m ²)	H	8M and 5F (19-31 yrs) 6M and 9F (19-32 yrs) 16M and 15F (19-32 yrs)	IF: Postexposure bronchoscopy was performed at 1.5 hr, 6 hr, and 18 hr; significant correlations between lung PMNs and blood PMNs postexposure; significant increase in PMN at 6 hr in bronchial wash and BAL-fluid as well as in bronchial epithelium and submucosa biopsies; 18 hr, PMN increase persisted in both bronchial wash and BAL while PMN in biopsies tended slightly lower; significant decrease in blood PMNs in subjects 1.5 hr postexposure compared to FA that rebounded above FA levels at 6 hr and at 18 hr postexposure, there was no difference in PMN levels when compared to FA	Bosson et al., 2013; 2020 ISA, p. 3-29, p. 4-28
0.20	2 hr IE (4×15 min, EVR=20 L/min-m ²)	H ^{NAs} As ^M	6M and 6F (19-31 yrs) 9M and 6F (21-48 yrs)	IF: Significantly higher baseline expression of IL-4 and IL-5 in bronchial mucosal biopsies from As ^M vs. H ^{NAs} subjects 6 hr postexposure. Epithelial expression of IL-5, GM-CSF, ENA-78, and IL-8 increased significantly in As ^M vs. H ^{NAs} subjects.	Bosson et al., 2003; 2006 AQCD, Table AX6-12
0.20	2 hr IE (4×15 min, EVR=20 L/min-m ²)	H ^{NS}	8M and 5F (20-31 yrs)	IF: No neutrophils in NL 1.5 hr postexposure. 30% depletion of uric acid in NL during hr 2 of exposure with increase in plasma uric acid levels. No depletion of ascorbic acid, reduced glutathione, or extracellular superoxide dismutase.	Mudway et al., 1999; 2006 AQCD, Table AX6-12
0.20	2 hr IE (4×14 min, $\dot{V}_E=70$ L/min)	H ^{NS}	20M (mean 25 yrs)	PF: ↓FVC*, ↓FEV ₁ *, ↓FEF ₂₅₋₇₅ *, ↓IC* and ↓TLC* AR: ↓sGaw SY: ↑respiratory symptoms*	Kulle et al., 1985; 1996 AQCD, Table 7-1
0.20	3 hr IE (6×15 min, EVR=25 L/min-m ²)	H ^{NS}	15M and 9F (18-40 yrs)	PF: ↓FEV ₁ * and ↓FVC* SY: ↑respiratory symptoms*	Frampton et al., 2015; 2020 ISA, p. 3-15, Table 3-4
0.21	1 hr CE (75% VO _{2max})	H ^{Ath}	6M and 1F (18-27 yrs)	PF: ↓FVC*, ↓FEV ₁ *, ↓FEF ₂₅₋₇₅ *, and ↓MVV* compared to FA SY: ↑respiratory symptoms*	Folinsbee et al., 1984; 1996 AQCD, Table 7-1
0.21	1 hr CE ($\dot{V}_E=80$ L/min) followed by maximal sprint (peak $\dot{V}_E >140$ L/min) Pre-treatment with albuterol or placebo	H ^{Ath}	14M and 1F (16-34 yrs)	PF: ↓FVC*, ↓FEV ₁ *, ↓FEF ₂₅₋₇₅ *, and ↓V _{Emax} in both treatment groups. No difference in the effects of albuterol on exercise performance vs. placebo. AR: No significant differences in the effects of albuterol on airway reactivity to histamine challenge vs placebo.	Gong et al., 1988; 1996 AQCD, Table 7-1

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.22	2.25 hr IE (4×15 min, 6-8×resting \dot{V}_E)	H	83M and 55F (mean 22 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * AR: Increased airway responsiveness 1 day postexposure IF: Increased epithelial permeability 1 day postexposure; airway responsiveness and epithelial permeability 1 day postexposure did not correlate with FEV ₁ responses immediately following the O ₃ exposure	Que et al., 2011; 2013 ISA, p. 6-74
0.24	1 hr CE (mean \dot{V}_E =57 L/min)	H ^{Ath}	42M and 8F (mean 26 yrs)	PF: ↓ FEV ₁ * SY: ↑ respiratory symptoms*	Avol et al., 1984; 1996 AQCD, Table 7-1
0.24	1 hr competitive simulation at mean \dot{V}_E =87 L/min; (30 min at \dot{V}_E =54 L/min, 30 min at \dot{V}_E =120 L/min)	H ^{Ath}	10M (19-29 yrs)	PF: ↓ FVC*, ↓ FEV ₁ * and ↓ FEF ₂₅₋₇₅ * compared to FA; ↓ exercise time* for subjects unable to complete simulation SY: ↑ respiratory symptoms*	Schelegle and Adams, 1986; 1996 AQCD, p. 7-11, Table 7-1
0.24	1.5 hr IE (3×15 min, \dot{V}_E =20 L/min)	As ^A H ^{NAs}	5M and 5F 4M and 4F (18-41 yrs)	NL immediately and 24 hr after exposure PF: No change in pulmonary or nasal function. IF: Significant increase in PMNs (at both time points) and in epithelial cells (immediately after exposure) only in As ^A subjects	McBride et al., 1994; 2006 AQCD, Table AX6-12
0.24	2.5 hr IE (4×15 min, EVR=25 L/min-m ²)	H	31M and 33F (18-35 yrs)	PF: ↓ FEV ₁ * compared with FA AR: ↑ sRaw* compared with FA SY: ↑ respiratory symptoms* compared with FA	Seal et al., 1993; 1996 AQCD, p. 7-15, Table 7-1
0.24	2.5 hr IE (4×15 min, \dot{V}_E =65 L/min)	H	21M (18-30 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ * and ↓ V _T * and ↑ f* AR: ↑ sRaw* SY: ↑ respiratory symptoms*	McDonnell et al., 1983; 1996 AQCD, Table 7-1
0.25	1 hr IE (2×15 min, \dot{V}_E =27 L/min)	As ^M	12M and 9F (19-40 yrs)	PF/AR: No significant differences in FEV ₁ or FVC compared to FA and no exacerbation of exercise-induced asthma in a postexposure exercise challenge	Weymer et al., 1994; 2006 AQCD, Table AX6-11
0.25	1 hr CE (EVR=30 L/min-m ²)	H ^{NS}	5M and 2F (22-30 yrs)	PF: ↓ FEV ₁ * IF: ↑ substance P* and ↑ 8-epi-PGF _{2α} * in segmental washing but not BAL fluid	Hazbun et al., 1993; 1996 AQCD, Table 7-1
0.25	1 hr CE (\dot{V}_E =30L/min); Facemask exposure	H ^{NS}	32M and 28F (mean 23 yrs)	PF: ↓ FEV ₁ *; sex differences in FEV ₁ decrements not significant; Uptake of O ₃ greater in M vs. F, but uptake not correlated with significant differences in spirometric responses between M and F.	Ultman et al., 2004; 2006 AQCD, Table AX6-1
0.25	1 hr CE (mean \dot{V}_E =63 L/min)	H	19M and 7F (mean 21 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ * and ↓ MVV* compared to FA	Folinsbee et al., 1986; 1996 AQCD, Table 7-1
0.25	2 hr IE (2×30 min at \dot{V}_E =39 L/min) 4 consecutive days	H ^{NS}	5M and 3F 25-31 yrs	PF: Maximal mean ↓ FEV ₁ * and ↓ FVC* on day 2, negligible by day 4. AR/IF: Significant small airway function depression accompanied by significant PMN in BAL fluid one day following the end of O ₃ exposure; PMN number in BAL fluid on day 5 were significantly higher following O ₃ , compared to air exposures	Frank et al., 2001; AQCD 2006 Tables AX6-9, AX6-12

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.25	2 hr IE (4×14 min, $\dot{V}_E=70$ L/min)	H ^{NS}	20M (mean 25 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ *, ↓ IC* and ↓ TLC* AR: ↓ SGaw* SY: ↑ respiratory symptoms*	Kulle et al., 1985; 1996 AQCD, Table 7-1
0.25	3 hr IE (6×15 min, EVR=14 L/min-m ²)	H	15M and 3F (mean 44yrs)	IF: significant increase in 3 hr postexposure sputum PMN compared to pre-exposure sputum; Bimosiamose pretreatment reduced PMN after O ₃ exposure to approximately the pre-exposure baseline	Kirsten et al., 2011; 2020 ISA, p. 3-30 and Table 3-9
0.25	3 hr IE (6×15 min, $\dot{V}_E=30$ L/min)	As ^A Al H ^{NS}	13M and 11F (mean 26 yrs) 6M and 6F (mean 25 yrs) 5M and 5F (mean 23 yrs)	PF: O ₃ -induced FEV ₁ * decrements of 12.5, 14.1, and 10.2% in As ^M , Al and H ^{NS} , respectively (group differences not significant) AR: Methacholine responsiveness increased in As ^A subjects; allergen responsiveness increased significantly after O ₃ exposure in both As ^A and Al subjects; no change in H ^{NS} subjects; allergen or methacholine response not correlated with each other or lung function	Jorres et al., 1996; 2006 AQCD, Table AX6-11
0.25	3 hr IE (6×15 min, $\dot{V}_E=30$ L/min) *challenged with allergen 20 hr following the last exposure and sputum collected 6-7 hr later	As ^M Al	6M and 5F (20-53 yrs); 16M and 6F (19-48 yrs)	PF/AR: Significantly greater mean early-phase allergen FEV ₁ response and number of ≥20% reductions in FEV ₁ in Al subjects IF: Significant increase in sputum eosinophils (As ^M and Al) and lymphocytes, mast cell tryptase, histamine, and LDH (As ^M only).	Holz et al., 2002; 2006 AQCD, Tables AX6-3 and AX6-11
0.25	3 hr IE (6×15 min, EVR=20 L/min-m ²) four O ₃ exposures: screening, placebo, and two treatments (inhaled or oral corticosteroids)	H ^{NS}	14M and 4F (mean 31.4 yrs)	PF: Postexposure spirometry not significantly different from baseline. IF: Screening and placebo O ₃ exposures caused > 9-fold increase in sputum neutrophils relative to baseline levels; relative to placebo, inhaled or oral corticosteroids significantly reduced neutrophil levels	Holz et al., 2005 2006 AQCD, p. AX6-123 and Table AX6-13
0.25	3 hr IE (6×15 min, EVR=20 L/min-m ²)	H	12M and 12F (20-48 yrs)	IF/HD: Sputum neutrophils, sputum CD14+ cells, as well as concentrations of IL1B, IL6, IL8, MMP9, and TNFα in sputum supernatant significantly increased 3 hr postexposure	Holz et al., 2015; 2020 ISA, p.3-29 and Table 3-9
0.25	3 hr IE (6×15 min, EVR=20 L/min-m ²)	H	11M and 3F (mean 33 yrs)	IF: Increase in blood neutrophils, neutrophil activation and total leukocytes at 5 and 7 hr postexposure, but not 24 hr.	Biller et al., 2011; 2020 ISA, p. 4-28 and Table 3-4
0.25	3 hr IE (6×15 min, EVR=20 L/min-m ²)	H	11M and 3F (22-47 yrs)	PF: ↓ FVC*, and ↓ FEV ₁ * IF: PMN increased in the blood 5 hr after the start of a 3-hr exposure and returned to baseline 21 hr postexposure	Tank et al., 2011; 2020 ISA, p.3-29 and Table 3-4
0.25	3 hr IE (6×15 min, $\dot{V}_E=26$ L/min) and repeated 1 week later	H ^{NS} As ^M	10M and 11F (mean 28 yrs) 5M and 10F (mean 30 yrs)	PF/SY: Significant ↓ FVC* and ↓ FEV ₁ that tended to be greater in the As ^M ; no significant group differences in symptoms or spirometry. IF: Significant ↑ neutrophils that did not differ between groups.	Holz et al., 1999; 2006 AQCD, p. AX6-35 and Table AX6-3

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.27	2 hr IE (3×20 min, EVR=25 L/min-m ²)	As ^A	12 - sex not indicated (18-37 yrs)	PF/SY: ↓ FVC*, ↓ FEV ₁ * and ↓ VC* and significant increase in symptom scores 24 hr following allergen challenge compared to FA IF: Percentage of eosinophils, but not neutrophils, in induced sputum was higher 6 hr after O ₃ vs. FA exposure	Vagaggini et al., 2002; AQCD 2006 Table AX6-12
0.27	2 hr CE (EVR=25 L/min-m ²) FA and to O ₃ exposures before and after 4 wk of treatment with budesonide	As ^M	7M and 7F (20-50 yrs)	PF/SY: Significant ↓ FEV ₁ and symptom scores; no change in FEV ₁ decrements or symptom scores with budesonide IF: Significant O ₃ -induced increase in sputum PMN and IL-8 was significantly reduced by budesonide 6 hr postexposure.	Vagaggini et al., 2001; AQCD 2006 Table AX6-13
0.27	2 hr IE (3×20 min, EVR=25 L/min-m ²) repeated 4 days after prednisone or placebo	As ^A	8M and 1F (mean 25 yrs)	PF: Corticosteroid pretreatment did not prevent ↓ FEV ₁ * vs placebo. IF: Significant inflammatory response (PMN influx) was prevented by corticosteroid pretreatment in induced sputum 6 hr postexposure.	Vagaggini et al., 2007; 2013 ISA, p. 6-78
0.30	30 to 80 min CE (V̇ _E =33 or 66 L/min)	H	8M (22-46 yrs)	PF: Significant pulmonary function decrements and exercise ventilatory pattern changes; multiple regression analysis showed O ₃ effective dose is a better predictor of response than concentration, V̇ _E , or duration of exposure, and O ₃ concentration accounted for the majority of the pulmonary function variance	Adams et al., 1981; 1996 AQCD, Table 7-1
0.30	1 hr CE (EVR=15 L/min-m ²)	H ^{NS} S	17M and 13F (mean 25 yrs) 19M and 11F (mean 24 yrs)	PF: ↓ FEV ₁ * was similar in both groups; based on exhaled CO ₂ , only smokers showed a reduction in dead space (-6.1 ± 1.2%) and an increase in the alveolar slope	Bates et al., 2014; 2020 ISA, p. 3-18, Table 3-4
0.30	1 hr CE (V̇ _E =60 L/min)	H	5M	PF: ↓ FVC* and ↓ FEV ₁ * 1 hr postexposure AR: ↑ sRaw* 1 hr postexposure IF: ↑ PMNs* at 1 hr, 6 hr, and 24 hr postexposure compared with FA in first aliquot "bronchial" sample (peaked at 6 hr); ↑ PMNs* at 6 and 24 hr in pooled aliquots.	Schelegle et al., 1991; 1996 AQCD, Table 7-1
0.30	1 hr CE (V̇ _E =60 L/min) or 2hr IE (V̇ _E =45-47 L/min)	H	12M (mean 24 yrs)	PF: ↓ FEV ₁ * was equivalent for both protocols SY: Significant symptom scores only in CE protocol	McKittrick and Adams, 1995; 1996 AQCD, Table 7-1
0.30	2 hr CE (EVR=25 L/min-m ²)	As	13M and 10F (mean 33 yrs);	PF: 4% group mean FEV ₁ decrement; no baseline difference between responders (8 subjects with >10% FEV ₁ decrements) and nonresponders IF: Significant correlation between changes in FEV ₁ and changes in sputum neutrophils 6 hr postexposure compared to FA in responders; significant increase in eosinophils in nonresponders only; NQO1 wildtype and GSTM1 null genotypes (6 subjects) not associated with the changes in lung function or inflammatory responses	Vagaggini et al., 2010; 2013 ISA, p. 6-79-80

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.30	2 hr IE (4×15 min, EVR=25 L/min-m ²) at 22°C and 32.5°C	H ^{NS}	14M and 2F (20-36 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * compared to FA; no significant effect of temperature or O ₃ -temperature interaction IF: Significant decrease in PAI-1 and plasminogen levels 24 hr postexposure at 22°C, but a significant increase in these coagulation markers 24 hr postexposure at 32.5°C	Kahle et al., 2015; 2020 ISA, p. 4-26, Table 3-4
0.30	2 hr IE (4×15 min, EVR=25 L/min-m ²)	H	14M and 5F (18-35 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * IF: Significant relationship between FEV ₁ and plasma ferritin (larger FEV ₁ decrements in subjects with lower baseline plasma ferritin)	Ghio et al., 2014; 2020 ISA, p. 3-15, Table 3-4
0.30	2 hr IE (4×15 min, EVR=25 L/min-m ²)	H	20M and 3F (19-33 yrs)	IF: Significant increases in CRP, IL-1, and IL-8, but not TNF-α; significant decrease in PAI-1 immediately and 24 hr postexposure; metabolomics analysis of BALF samples concluded that 1 hr responses reflected oxidative stress and at 24 hr responses reflected tissue repair	Devlin et al., 2012; Cheng et al., 2018; 2020 ISA, p. 4-26, 4-28, Table 3-9
0.30	2 hr IE (4×15 min at either $\dot{V}_E=30$ L/min, $\dot{V}_E=50$ L/min or $\dot{V}_E=70$ L/min)	H	30M (three groups of 10) (19-26 yrs)	PF: ↓ FEV ₁ * and ↓ FVC* at all ventilation rates; ↓ MVV* only at highest \dot{V}_E . Note: additional exposure at 0.50 ppb resulted in ↓ FEV ₁ *, ↓ FVC*, ↓ MVV*, ↓ IC*, and ↓ TLC* at all ventilation rates.	Folinsbee et al., 1978 ^G 1996 AQCD p. 7-10
0.30	2.5 hr IE (4×15 min, $\dot{V}_E=65$ L/min)	H	20M (18-30 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ * and ↓ V _T *; and ↑ f _R * AR: ↑ sRaw* SY: ↑ respiratory symptoms*	McDonnell et al., 1983; 1996 AQCD, p. 7-15, Table 7-1
0.30	2.5 hr IE (4×15 min, EVR=25 L/min-m ²)	H	30M and 30F (18-35 yrs)	PF: ↓ FEV ₁ * compared with FA AR: ↑ sRaw* compared with FA SY: ↑ respiratory symptoms* compared with FA	Seal et al., 1993; 1996 AQCD, p. 7-15, Table 7-1
0.30	2 hr IE (4×15 min, EVR=25 L/min-m ²) 2 consecutive days	H	11M and 4F (23-36 yrs)	PF: 2 consecutive days of O ₃ exposure resulted in greater ↓ FEV ₁ * than the decrement immediately after the first day of O ₃ exposure	Madden et al., 2014; 2020 ISA, p. 3-15, Table 3-4
0.30	2 hr IE (4×15 min, EVR=25 L/min-m ²) for 2 days	H	11M and 4F (23-36 yrs)	PF/IF: ↓ FEV ₁ * positively correlated with significant decrease in the inflammatory cytokine IFN-γ in the blood	Stiegel et al., 2017; 2020 ISA, p. 3-15, Table 3-4
0.30	2 hr IE (2×20 min, EVR=25 L/min-m ²)	As	86M and 34F (mean 33 yrs)	PF/AR: Magnitude of O ₃ -induced FEV ₁ response increased with decreasing baseline FEV ₁ and lack of inhaled corticosteroid treatment; FEV ₁ response was unrelated to methacholine responsiveness	Bartoli et al., 2013; 2020 ISA, p. 3-17, p. 3-47, Table 3-16
0.32	1 hr CE (mean $\dot{V}_E=57$ L/min)	H ^{At}	42M and 8F (mean 26 yrs)	PF: ↓ FEV ₁ * SY: ↑ respiratory symptoms*	Avol et al., 1984; 1996 AQCD, Table 7-1
0.33	2 hr IE (4×15 min, bicycle at 600 kpm/min)	H ^{NS}	9M (mean 27 yrs)	PF: ↓ FVC*; post FA, normal gradient in ventilation which increased from apex to the base of the lung; post-O ₃ , ventilation shifted away from the lower-lung into middle and upper-lung regions; post-O ₃ increase in ventilation to mid-lung region correlated with decrease in midmaximal expiratory flow (r = 0.76, p < 0.05).	Foster et al., 1993; 2006 AQCD, Table AX6-1

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.35	50 min CE ($\dot{V}_E=60$ L/min) repeat exposures over 4 days	H ^{NS}	8M (19-26 yrs) (some known O ₃ -sensitive)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ * and ↓ V _T * compared to FA on days 1-4; largest ↓ FEV ₁ * on day 2; ↓ exercise performance time* on day 1 significantly less after the 4th day; ↑ f _R *, and ↓ VO _{2max} * on day 1, recovered by day 4.	Foxcroft and Adams, 1986; 2006 AQCD, Tables AX6-9, AX6-10
0.35	1 hr CE ($\dot{V}_E=80$ L/min) or 1 hr competitive simulation (30 min at $\dot{V}_E=52$ L/min, 30 at min $\dot{V}_E=100$ L/min; overall mean $\dot{V}_E =77.5$ L/min)	H ^{At}	10M (19-31 yrs)	PF: ↓ FVC*, ↓ FEV ₁ * and ↓ FEF ₂₅₋₇₅ * compared to FA with both protocols; ↓ V _T * and ↑ f _R * with CE; reduced exercise time in 3 subjects who were unable to complete CE and competitive protocols SY: ↑ respiratory symptoms*	Adams and Schelegle, 1983; 1996 AQCD, Table 7-1
0.35	1 hr CE (mean $\dot{V}_E=60$ L/min) Pretreatment: no drug, placebo, or indomethacin	H	14M (18-34 yrs)	PF: ↓ FVC* and ↓ FEV ₁ *; indomethacin significantly attenuated decreases in FVC and FEV ₁ compared to no drug and placebo; AR: ↑ sRaw* not affected by indomethacin	Schelegle et al., 1987; 1996 AQCD, Table 7-1
0.35/ 0.20	1 hr CE (mean $\dot{V}_E=60$ L/min); 2 exposures 24 hr apart	H ^{NS}	15M (mean 25 yrs)	PF: ↓ FVC*, ↓ FEV ₁ * responses on each day compared to FA with an increased response to 0.20 ppm on the second day SY: Consecutive exposures produced similar ↑ respiratory symptoms*	Brookes et al., 1989; 2006 AQCD. Table AX6-9
0.35	1 hr CE (mean $\dot{V}_E=60$ L/min); 2 exposures 24 hr apart	H ^{NS}	15M (mean 25 yrs)	PF: Significant ↓ FVC*, ↓ FEV ₁ * responses on each day compared to FA with an increased response to 0.35 ppm on the second day SY: Significant symptom responses were worse after second day of exposure to 0.35 ppm	Brookes et al., 1989; 2006 AQCD. Table AX6-9
0.35	1 hr CE ($\dot{V}_E=60$ L/min); two exposures for each subject separated by 24, 48, 72, or 120 hr	H ^{NS}	40M, 4 groups of 10 (19-35 yrs)	PF/AR: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ * and ↑ sRaw* for all exposures. Enhanced FEV ₁ * response after 24 hr repeat exposure and a trend toward an enhanced response at 48 hr. No differences between responses to exposures separated by 72 or 120 hr. Similar trends observed for sRaw.	Schonfeld et al., 1989; 2006 AQCD. Table AX6-9
0.35	70 min IE ($\dot{V}_E=40$ L/min)	H ^{NS}	18F (19-28 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ * and ↓ MVV* immediately postexposure. AR: ↑ sRaw* at 1 hr and 18 hr postexposure.	Folinsbee and Hazucha, 1989; 2006 AQCD, Table AX6-11
0.35	1.25 hr IE (2 × 30 min, $\dot{V}_E=40$ L/min)	H	19F (mean 22 yrs)	PF: ↓ FVC*, ↓ FEV ₁ * and ↓ FEF ₂₅₋₇₅ * 1 hr postexposure; Persistence of small effects on both inspired and expired spirometry past 18 hr. AR: ↑ sRaw* 1 hr and 18 hr postexposure but not 42 hr postexposure.	Folinsbee and Hazucha, 2000; 2006 AQCD, Table AX6-6
0.35	2.2 hr IE (2 × 30 min, $\dot{V}_E=50$ L/min; final 10 min rest)	H ^{NS}	15M (mean 25 yrs)	PF: ↓ FVC* and ↓ FEV ₁ *; pronounced slow phase in multi-breath nitrogen washouts post O ₃ exposure; washout delays not related to changes in ventilatory pattern or lung volume at FRC.	Foster et al., 1997; 2006 AQCD, Table AX6-1
0.37	2 hr IE (4×15 min, $\dot{V}_E=2.5$ × rest)	H	20M and 8F (19-29 yrs)	PF: ↓ FEF ₂₅ * and ↓ FEF ₅₀ * compared to FA Note: additional exposure at 0.50 and 0.75 ppb resulted in ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅ * and ↓ FEF ₅₀ * compared to FA	Silverman et al., 1976; 1996 AQCD, Table 7-1

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.40	1 hr IE (2.× 15 min, $\dot{V}_E=27$ L/min)	As ^M	6M and 6F (19-40 yrs)	PF: ↓ FEV ₁ * but no exacerbation of exercise-induced asthma in a postexposure exercise challenge SY: Significant increase in respiratory symptoms regardless of exercise induced asthma status (7 subjects)	Weymer et al., 1994; 2006 AQCD, Table AX6-11
0.40	1 hr CE (EVR=20 L/min-m ²)	H	22M (18-35 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEV ₁ /FVC*, and ↓ FEF ₂₅₋₇₅ ; half-width of an expired aerosol bolus was significantly increased, suggesting an O ₃ -induced change in small airway function.	Keefe et al., 1991; 1996 AQCD, Table 7-1
0.40	1 hr CE (EVR=20 L/min-m ²)	H	20M (18-35 yrs)	PF: 25% ↓ V _T and 9% ↓ O ₃ uptake efficiency in the lower respiratory tract	Gerrity et al., 1994; 1996 AQCD, Table 7-1
0.40	1 hr CE (EVR=30 L/min-m ²)	H ^{NS}	4 subjects (sex and age not indicated)	IF: Apoptotic cells in BAL fluid 6 hr postexposure HD: Alveolar macrophages from BAL fluid showed the presence of 4-HNE, protein adduct, 72-kD heat shock protein and ferritin.	Hamilton et al., 1998; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, cycle ergometry: 100W for M and 83W for F)	H ^{NS}	7M and 3F (23-41 yrs)	AR: Increase in airway responsiveness to methacholine challenge IF: Increase in percentage of PMN and PGF _{2α} ; increased TBX ₂ , and PGE ₂ concentrations in BAL fluid 3 hr postexposure vs FA	Seltzer et al., 1986; 1996 AQCD, Tables 7-1, 7-11
0.40	2 hr IE (4×15 min, $\dot{V}_E=30$ L/min) 3 day indomethacin pretreatment	H ^{NAs} As ^M	5M and 4F 6M and 7F (18-28 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * in both groups; significant reductions in mid-flows in both groups but were greater in As ^M vs. H ^{NAs} subjects; indomethacin pretreatment attenuated ↓ FVC* and ↓ FEV ₁ * responses to O ₃ in H ^{NAs} but not As ^M subjects.	Alexis et al., 2000; 2006 AQCD, Table AX6-1, AX6-13
0.40	2 hr IE (4×15 min, $\dot{V}_E=30-40$ L/min)	H ^{GSTM} + H ^{GSTM} -	6M and 13F 9M and 7F (mean 24 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * from baseline across groups; no difference in lung function response between groups IF: ↑ PMN* and increased expression of HLA-DR on airway macrophages and dendritic cells in GSTM1- subjects 24 hr postexposure; decreased macrophages in GSTM1-sufficient subjects 4-24 hr postexposure. Note: no FA control	Alexis et al., 2009; 2013 ISA, p. 6-80, p. 6-125
0.40	2 hr IE (4×15 min, $\dot{V}_E=30-40$ L/min)	H ^{NS}	4M and 5F (21-30 yrs)	IF/HD: Significant increase in sputum neutrophils; activation of monocytes and upregulation of cell surface molecules associated with antigen presentation (HLA-DR and CD86)	Lay et al., 2007; 2013 ISA, p. 5-44
0.40	2 hr IE (4×15 min, $\dot{V}_E=30-40$ L/min)	H ^{NAs} Al ^{NAs} As ^A	14M and 20F (mean 24 yrs) 7M and 7F (mean 25 yrs) 7M and 10F (mean 24 yrs)	IF/HD: Enhanced inflammatory response in As ^A with greater numbers of neutrophils, higher levels of cytokines (IL-6, IL-8, IL-18, and TNF-α) and greater macrophage cell-surface expression of TLR4 and IgE receptors in induced sputum compared with H ^{NAs} ; increase hyaluronan in Al ^{NAs} and As ^A compared with H ^{NAs} Note: no FA control	Hernandez et al., 2010; Hernandez et al., 2012; 2013 ISA, p. 6-130, p. 8-13; 2020 ISA, p. 3-29 p. 3-52, Table 3-20
0.40	2 hr IE (4×15 min, $\dot{V}_E=40$ L/min); Mouthpiece exposure	H	5M and 5F (mean 30 yrs)	IF: Significant increase in PMNs and decrease in macrophages in sputum 4 hr postexposure; IL-6, IL-8, and myeloperoxidase increased; possible relationship of IL-8 and PMN levels.	Fahy et al., 1995; 2006 AQCD, Table AX6-12

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.40	2 hr IE (4×15 min, EVR=18 L/min-m ²) Postexposure, H ^{WR} treated with naxlozone or saline and H ^{SR} treated with sufentanil or saline	H ^{WR} H ^{SR}	7M and 13F 21M and 21F (20-59 yrs)	PF/SY: ↓ spirometric lung function* across groups, young adults (<35 yrs) significantly more responsive than older individuals (>35 yrs). Sufentanil, a narcotic analgesic, largely abolished symptom responses and improved FEV ₁ in strong responders. Naloxone, an opioid antagonist, did not affect O ₃ effects in weak responders.	Passannante et al., 1998; 2006 AQCD, Table AX6-13
0.40	2 hr IE (4×15 min, EVR=20 L/min-m ²)	H ^{NAs} As ^A	5M and 1F (mean 29 yrs) 6M (mean 24 yrs)	PF: Similar ↓ FEV ₁ * in both groups AR: Maximal FEV ₁ response to methacholine increased similarly in both groups 12 hr postexposure IF: Significant increase in PMN in both groups	Hiltermann et al., 1995; 2006 AQCD, Table AX6-3
0.40	2 hr IE (4×15 min, EVR=20 L/min-m ²)	As ^M	1M and 5F (18-27 yrs)	PF: ↓ FEV ₁ * AR: Increased airway responsiveness to methacholine 16 hr postexposure; no effect of proteinase inhibitor (rALP)	Hiltermann et al., 1998; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, EVR=20 L/min-m ²)	As	10M and 6F (19-35 yrs)	IF: Levels of eosinophil cationic protein, IL-8 and percentage eosinophils highly correlated in sputum and BAL 16 hr postexposure.	Hiltermann et al., 1999; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, EVR=20 L/min-m ²) Apocynin or placebo	As ^M	1M and 6F (19-26 yrs)	AR/IF: Increased bronchial responsiveness to methacholine 16 hr postexposure; inhaled apocynin (an inhibitor of NADPH oxidase present in inflammatory cells) treatment significantly reduced O ₃ -induced airway responsiveness	Peters et al., 2001; 2006 AQCD, Table AX6-11,
0.40	2 hr IE (4×15 min, EVR=20 L/min-m ²)	H ^{NS} H ^{NS}	Placebo: 15M and 1F Antioxidant: 13M and 2F (mean 27 yrs)	AR: ↓ FVC*, and ↓ FEV ₁ * in both groups IF: no difference in PMNs and IL-6 levels in BAL fluid 1 hr postexposure between treatment groups.	Samet et al., 2001; Steck- Scott et al., 2004; 2006 AQCD, Tables AX6-1, AX6-13
0.40	2 hr IE (4×15 min, $\dot{V}_E=25$ L/min)	H ^{Wt} Ob	19F 19F (18-35 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * in both groups; ↓ FVC* was greater in obese women than in normal-weight women. AR/IF: Increase in airway responsiveness or increase in PMN after O ₃ exposure did not differ between normal-weight and obese women. SY: Symptoms in response to exposure did not differ between groups	Bennett et al., 2016; 2020 ISA, p. 3-57, p. 3-59, Tables 3-4, 3-8, 3-9, 3-31
0.40	2 hr IE (4×20 min of mild-moderate exercise) 2 wk pretreatment with budesonide or placebo	H ^{NAs}	6M and 9F (mean 31 yrs)	PF: ↓ FVC* and ↓ FEV ₁ * immediately postexposure; FVC and FEV ₁ decrements recovered 4 hr postexposure; AR: Small increased bronchial reactivity to methacholine IF: Increased PMNs and myeloperoxidase in 4 hr postexposure sputum; no protection from inhaled corticosteroid, budesonide.	Nightingale et al., 2000; 2006 AQCD, Table AX6-13

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.40	2 hr IE (4×20 min, 50W cycle ergometry, 10 min rest) 2 wk pretreatment with budesonide or placebo	H ^{NS}	4M and 5F (mean 30 yrs)	PF: Placebo-control: Immediately postexposure significant ↓ FVC and FEV ₁ relative to pre-exposure values; 3 hr postexposure FVC and FEV ₁ recovered to preexposure values. IF: Significant increases in 8-isoprostane at 4 hr postexposure; Budesonide for 2 wk prior to exposure did not affect responses.	Montuschi et al., 2002; 2006 AQCD, Table AX6-1
0.40	2 hr IE (4×15 min, $\dot{V}_E=50-75$ L/min)	H ^{NAs} A ^{INAs} A ^S ^A	5M and 8F 4M and 1F 3M and 8F (21-35 yrs)	PF/IF: FEV ₁ responses to O ₃ not differentiated by asthma; precent predicted FEV ₁ both before and after O ₃ exposure did not differ between inflammatory responders (>10% increase in PMN) and nonresponders	Fry et al., 2012; 2020 ISA, p. 3-29, p. 3-36, Table 3-17
0.40	2 hr IE (4×15 min, $\dot{V}_E=50-75$ L/min) Pretreatment: saline or atropine	H ^{NS}	8M (18-27yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ V _T *, and ↓ TLC*; and ↑ f _R *. Atropine pretreatment attenuated FEV ₁ and FEF ₂₅₋₇₅ response. AR: ↑ sRaw*; Atropine pretreatment abolished increase in sRaw	Beckett et al., 1985; 1996 AQCD, Table 7-1
0.40	2 hr IE (4×15 min, $\dot{V}_E=53-55$ L/min)	H ^{NAs} A SM	4M and 5F 4M and 5F (18-34 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, and ↓ FEF ₂₅₋₇₅ in both groups with a significantly greater percent ↓ in As compared to H ^{NAs} subjects AR: ↑ sRaw* in As; airway responsiveness (methacholine challenge) was not statistically different between H ^{NAs} and A SM subjects	Kreit et al., 1989; 2006 AQCD, Table AX6-11
0.40	2 hr IE (4×15 min, EVR=30 L/min-m ²) 4 day pretreatment with indomethacin or placebo	H ^{NS}	13M (18-31 yrs)	PF: Indomethacin pretreatment resulted in a significantly smaller FVC and FEV ₁ decrements than with O ₃ alone AR: airway hyperresponsiveness was not significantly affected by indomethacin pretreatment.	Ying et al., 1990; 1996 AQCD, Table 7-1
0.40	2 hr IE (4×15 min, $\dot{V}_E=66$ L/min)	H ^{NS}	8M (18-35 yrs)	IF: BAL fluid at 1 hr postexposure vs. 18 hr postexposure. At 1 hr, PMN's, total protein, LDH, α1-antitrypsin, fibronectin, PGE ₂ , thromboxane B ₂ , C3 _a , tissue factor, and clotting factor VII were increased; IL-6 and PGE ₂ were higher after 1 hr than 18 hr; fibronectin and tissue plasminogen activator higher after 18 hr. No time differences for PMN and protein.	Devlin et al., 1996; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, $\dot{V}_E=70$ L/min);	H	11M (18-35 yrs)	IF/HD: Macrophages 18 hr postexposure had changes in the rate of synthesis of 123 different proteins as assayed by computerized densitometry of two-dimensional gel protein profiles	Devlin and Koren, 1990; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, $\dot{V}_E=70$ L/min);	H	11M (18-35 yrs)	IF/HD: BAL fluid 18 hr postexposure contained increased levels of the coagulation factors, tissue factor, and factor VII; macrophages in the BAL fluid had elevated tissue factor mRNA	McGee et al., 1990; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, $\dot{V}_E=70$ L/min);	H	11M (18-35 yrs)	IF: NL done immediately before, immediately after, and 22 hr after exposure; increased PMNs at both postexposure times; increased levels of tryptase (marker of mast cell degranulation) immediately postexposure; increased levels of albumin 22 hr postexposure.	Graham and Koren, 1990; Koren et al., 1990; 2006 AQCD, Table AX6-12

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.40	2 hr IE (4×15 min, EVR=35 L/min-m ²)	H	11M (18-35 yrs)	PF/IF: Significant increase in PMNs, total protein, albumin, IgG, PGE ₂ , plasminogen activator, neutrophil elastase complement C3a, and fibronectin; no correlation between pulmonary function and inflammatory endpoints in BAL fluid 18 hr postexposure HD: decrease in percentage of macrophages compared to FA	Koren et al., 1989a; Koren et al., 1989b; 1996 AQCD, Table 7-1; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, EVR=35 L/min-m ²)	H	10M (18-35 yrs)	PF/IF: Increased PMN, protein, PGE ₂ , LDH, TXB ₂ , IL-6 α-1 anti-trypsin, and tissue factor in BAL fluid 1 hr postexposure compared to 18 hr; fibronectin and urokinase-type plasminogen activator higher 18 hr postexposure than 1 hr HD: Decreased phagocytosis of yeast by alveolar macrophages.	Koren et al., 1991; 1996 AQCD, Table 7-1; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, EVR=35 L/min-m ²)	H	8M (20-30 yrs)	PF: ↓ FVC* AR: ↑ sRaw* IF: Significantly increased clearance of ^{99m} Tc-DTPA from the lung indicating epithelial damage, and changes in permeability.	Kehrl et al., 1987; 2006 AQCD, Table AX6-13
0.40	2.5 hr IE (4×15 min, EVR=25 L/min-m ²)	H	30M and 30F (18-35 yrs)	PF: ↓ FEV ₁ * compared with FA AR: ↑ sRaw* compared with FA SY: ↑ Respiratory symptoms* compared with FA	Seal et al., 1993; 1996 AQCD, p. 7-15, Table 7-1
0.40	2.5 hr IE (4×15 min at V _E =64 L/min)	H	29M (18-30 yrs)	PF: ↓ FVC*, ↓ FEV ₁ *, ↓ FEF ₂₅₋₇₅ *, ↓ V _T * and ↑ f* AR: ↑ sRaw* SY: ↑ Respiratory symptoms*	McDonnell et al., 1983; 1996 AQCD, p. 7-15, Table 7-1
0.40	2 Hr IE (4×15 min, 2 × resting V _E) 2 Hr IE (4×15 min, 2 × resting V _E) × 3 days	H ^{NS}	12M and 7F (21-32 yrs)	AR: Significant increase in histamine airway responsiveness with progressive adaptation of the effect; after day 3 histamine responsiveness was not different from sham exposures	Dimeo et al., 1981; 2006 AQCD, Table AX6-11
0.40	IE (2×15 min, V _E =40 L/min-m ²) 2 h/day for 5 days, 2 h either 10 or 20 days later	H ^{NS}	16M (18-35 yrs)	PF: ↓ FEV ₁ * at each time point; FEV ₁ decrement was greatest on day 2 and was significantly attenuated by days 4 and 5. IF: BAL immediately after day 5 of exposure and again after exposure 10 or 20 days later. Most markers of inflammation (PMNs, IL-6, PGE ₂ , fibronectin) showed complete attenuation; markers of damage (LDH, IL-8, protein, 1-antitrypsin, elastase) did not. Reversal of attenuation was not complete for some markers, even after 20 days.	Devlin et al., 1997; 2006 AQCD, Tables AX6-9, and AX 6-12
0.40	3 hr/day (2 hr resting followed by 1 hr CE at 4-5 resting V _E) for 5 days,	H ^{NS}	13M and 11F (19-46 yrs)	AR: Enhanced airway response to methacholine after the first 3 days which normalized by day 5	Kulle et al., 1982; 1996 AQCD, Table 7-10

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.40	3 hr/day for 5 days: IE (6×15 min mild-moderate exercise, $\dot{V}_E=32$ L/min)	As ^M	8M and 2F (mean 31 yrs)	PF/SY: Significant ↓ FEV ₁ and increase in symptom response on O ₃ exposure days 1 and 2 that diminished with continued exposure; tolerance partially lost 4 and 7 days postexposure AR: bronchial reactivity to methacholine peaked after O ₃ exposure on day 1, but remained elevated with continued exposure	Gong et al., 1997; 2006 AQCD, Table AX6-11
Children During Moderate Exercise					
0.12	2.5 hr IE (4x15 min, EVR=35 L/min- m ²)	H	23 M (8-11 yrs)	PF: ↓ FEV ₁ * compared with clean air which persisted for 16-20 hr SY: No significant increase in severity of respiratory symptoms	McDonnell et al. (1985); 2006 AQCD
Adult Subjects at Rest					
0.10	2 hr	H	10M (18-28 yrs)	PF: No significant change in pulmonary function	Folinsbee et al., 1978; 1996 AQCD, p. 7-10
0.10	2 hr	H ^{NS}	13M and 1F (mean 24 yrs)	AR: No increased airway responsiveness to methacholine immediately after exposure.	Konig et al., 1980; 1996 AQCD, Table 7-10
0.12	1 hr Air-antigen/O ₃ -antigen	As ^A	4M and 3F (21-64 yrs)	PF: No change in baseline pulmonary function. AR: Increased allergen-specific airway responsiveness to inhaled ragweed or grass after O ₃ exposure compared to FA	Molfino et al., 1991; 1996 AQCD, Tables 7-2, 7- 10
0.12	1 hr	As ^A	10M and 5F (19-34 yrs)	PF: No significant change in pulmonary function to O ₃ alone. AR: No significant change in sRaw to O ₃ alone; no significant effect on airway responsiveness to grass allergen	Ball et al., 1996 2006 AQCD, Table AX6-11
0.12	1 hr (Air-Antigen)	As ^A	9M and 6F (18- 49 yrs)	AR: No effect of O ₃ on airway responsiveness to grass or ragweed allergen.	Hanania et al., 1998; 2006 AQCD, Table AX6-11
0.12	1 hr O ₃ at rest followed by 6 min maximal exercise	As ^A	7M and 8F (19- 45 yrs)	PF: No significant change in FEV ₁ AR: O ₃ pre-exposure did not affect the magnitude or time course of exercise-induced bronchoconstriction.	Fernandes et al., 1994 1996 AQCD, Table 7-2
0.20	2 hr	H ^{NS}	15 subjects	IF/HD: Increased numbers of CD3+, CD4+, and CD8+ T lymphocyte subsets, in addition to neutrophils, in BAL fluid 6 hr postexposure.	Blomberg et al., 1997; 2006 AQCD, Table AX6-12
0.25	2 hr	H	8M and 5F (21-22 yrs)	PF: No significant change in FVC compared with FA	Horvath et al., 1979; 1996 AQCD, Table 7-1
0.30	2 hr	H	10M (18-28 yrs)	PF: No significant change in pulmonary function	Folinsbee et al., 1978 ^G 1996 AQCD, p. 7-10
0.30	2 hr	H	9-11 subjects (18-35 yrs)	IF: Significantly elevated levels of pro-inflammatory oxysterols in BAL fluid compared to FA	Speen et al., 2016 2020 ISA, Table 3-9
0.32	2 hr	H ^{NS}	13M and 1F (mean 24 yrs)	AR: Increased airway responsiveness to methacholine immediately after exposure.	Konig et al., 1980; 1996 AQCD, Table 7-10

O ₃ ^A (ppm)	Exposure and Ventilation Characteristics During Exercise ^W	Subject Characteristics ^B		Reported Effects on Pulmonary Function (PF), Airway Resistance and/or Responsiveness (AR), Respiratory Symptoms (SY), Inflammation (IF) ^E and Host Defense (HD)	Reference AQCD/ISA
		Pop ^C	n ^D		
0.37	2 hr	H	20M and 8F (19-29 yrs)	PF: No significant change in FEV ₁ , FEF ₂₅ , and FEF ₅₀ compared with FA	Silverman et al., 1976; 1996 AQCD, Table 7-1
0.40	2 hr	As ^A	12 subjects (18-35 yrs)	IF: Release of early-onset mast cell-derived mediators into NL in response to allergen not enhanced after O ₃ exposure. No increase in neutrophil and eosinophil inflammatory mediators after O ₃ exposure or enhancement after allergen challenge. O ₃ increased eosinophil influx following allergen exposure.	Michelson et al., 1999 2006 AQCD, Table AX6-12
0.40	2 hr	As ^M	10 subjects (18-35 yrs)	IF: Increased response to allergen; significant increase in PMN and eosinophils after O ₃ plus allergen challenge; O ₃ alone increased nasal inflammation (PMN).	Peden et al., 1995; 2006 AQCD, Table AX6-12
0.40	2hr × 2 days during and out of grass pollen season	AI	5M and 5F (mean 28 yrs)	IF: Significant increase in nasal mucus total protein, albumin, PMNs, and eosinophils following O ₃ exposures during pollen season, but an allergen exaggerated the inflammatory response cannot be concluded because statistical tests were not performed across the seasons	Dokic and Trajkovska-Dokic, 2013; 2020 ISA, p. 3-51, Table 3-21
0.50	2 hr	H	10M (18-28 yrs)	PF: ↓ FEV ₁ * ^G , ↓ FVC* but no change in MVV	Folinsbee et al., 1978; ^G 1996 AQCD, Table 7-1
0.50	2 hr	H	8M and 5F (21-22 yrs)	PF: ↓ FVC* compared with FA	Horvath et al., 1979; 1996 AQCD, Table 7-1
0.50	2 hr	H	20M and 8F (19-29 yrs)	PF: No significant change in FEV ₁ , FEF ₂₅ , and FEF ₅₀ compared with FA	Silverman et al., 1976; 1996 AQCD, Table 7-1
0.60	2 hr	H ^{NS}	5M and 3F (22-30 yrs)	AR: 300% increase in histamine-induced ΔRaw 5 min after O ₃ exposure; 84 and 50% increases 24 hr and 1 week after exposure (p > 0.05), respectively. Two subjects had an increased response to histamine 1 week after exposure.	Golden et al., 1978; 1996 AQCD, Table 7-10;
0.75	2 hr	H	8M and 5F (21-22 yrs)	PF: ↓ FVC* compared with FA	Horvath et al., 1979; 1996 AQCD, Table 7-1
0.75	2hr	H	20M and 8F (19-29 yrs)	PF: ↓ FEV ₁ * ^G , ↓ FEF ₂₅ * ^G , and ↓ FEF ₅₀ * compared with FA	Silverman et al., 1976; 1996 AQCD, Table 7-1
1.00	2 hr	H ^{NS}	13M and 1F (mean 24 yrs)	AR: Increased airway responsiveness to methacholine immediately after exposure.	Konig et al., 1980 1996 AQCD, Table 7-10

Note: Newly added studies since the 2015 review are in blue font.

^A Reported target mean O₃ concentrations

^W Focused on O₃ exposures below 0.4 ppm during exercise and below 1.00 ppm at rest

^B Subject Characteristics are subdivided into subject population (Pop) and number (n) subjects.

^C Subject population included: healthy subjects (H), athletes included competitive endurance cyclists and runners (H^A), nonsmokers (H^{NS}), nonasthmatics (H^{NAs}), nonasthmatics with allergies (AI^{NAs}), asthmatics (As), mild asthmatics (As^M), SO₂-sensitive asthmatics (As^{SO2}), asthmatics with allergies (As^A), subjects with allergies (AI), smokers (S), healthy

subjects with the GSTM1 genotype (H^{GSTM+}) or null for the GSTM1 genotype (H^{GSTM-}), healthy subjects that have a weak O₃ response (H^{WR}) or have a strong O₃ response (H^{SR}), healthy weight subjects (H^W) and obese subjects (Ob).

^D Number is further characterized by sex, male (M) and female (F), and age range or mean age of the subjects.

^E For the purposes of this table the "IF" category includes reported effects on inflammation (the most commonly tested endpoint) as well as injury and oxidative stress responses because injury, inflammation, and oxidative stress responses are difficult to disentangle. Inflammation generally occurs as a consequence of injury and oxidative stress, but it can also lead to further oxidative stress and injury due to secondary production of reactive oxygen species (ROS) by inflammatory cells (2020 ISA section 3.1.3).

* Indicates statistical significance

^F Avol et al., 1984 reported O₃-induced effects for 0.08, 0.16, 0.24 and 0.32 ppm but only effects from 0.16, 0.24 and 0.32 ppm was referenced in 1996 AQCD, Table 7-1.

^G Folinsbee et al., 1978 reported data for subjects exposed to O₃ during exercise at 0.1 ppm and 0.3 ppm at 3 different ventilation rates and at rest at 0.1 ppm, 0.3 ppm and 0.5 ppm. Only the 0.5 ppm O₃ exposure to subjects at rest was referenced in 1996 AQCD, Table 7-1 (although the number of subjects was incorrectly identified for this exposure).

^J Subtracted from FA, the group mean decrement in FEV₁ was 9.7% (2006 AQCD and 2013 ISA).

Abbreviations: BAL, bronchoalveolar lavage; C3_a, complement protein fragment; CC16, protein secreted by Clara cells in the non-ciliated respiratory epithelium; CD86, surface costimulatory marker for T-cell activation; CE, continuous exercise; CRP, C-reactive protein; ENA-78, epithelial cell-derived neutrophil-activating peptide; FA, filtered air; FEF₂₅, (formerly designated as V_{25%VC}) instantaneous forced expiratory flow after 25% of forced vital capacity; FEF₂₅₋₇₅, forced expiratory flow over the middle half of forced vital capacity; FEF₅₀, (formerly designated as V_{50%VC}) instantaneous forced expiratory flow after 50% of forced vital capacity; FEV₁, forced expiratory volume in one second; f_R, respiratory frequency (also abbreviated as f); FRC, functional reserve capacity; FVC, forced vital capacity; GM-CSF, granulocyte-macrophage colony-stimulating factor; GSTM1, glutathione S-transferase M1 polymorphism; HLA-DR, human leukocyte antigens; 4-HNE, 4-hydroxynonenal; IC, inspiratory capacity; IE, intermittent exercise; IgE, immunoglobulin E; IgG, immunoglobulin G antibody; IL-6, IFN-γ, interferon-gamma; IL-1, interleukin 1 pro-inflammatory cytokine interleukin 6 pro-inflammatory cytokine; IL-8, interleukin 8 pro-inflammatory cytokine; IL-18, interleukin 18 pro-inflammatory cytokine; ISA, Integrated Science Assessment; LDH, lactate dehydrogenase; LTB₄, leukotriene; MMP9, metalloproteinase 9; MVV, maximal voluntary ventilation; NQO1, NAD(P)H:quinone oxidoreductase; NL, nasal lavage; 8-OHdG, 8-hydroxy-2'-deoxyguanosine; PAI-1, plasminogen activator fibrinogen inhibitor-1; PGE₂, prostaglandin E₂ a mediator of inflammation; PGE₂, bronchodilatory prostaglandin; PGF_{2α}, prostaglandin 2 alpha; PMN, polymorphonuclear neutrophils; ROS, reactive oxygen species; sGaw, specific airway conductance; sRaw, specific airway resistance; substance P, neuropeptide that act as a neurotransmitter and neuromodulator; TBARS, Thiobarbituric acid reactive substance, ^{99m}Tc-DTPA, radiolabelled diethylene triamine pentaacetic acid; TBX2, thromboxane B2; TLC, total lung capacity; TLR4, Toll-like receptor protein 4; TNF-α, tumor necrosis factor alpha; tPA, tissue plasminogen activator; VC, vital capacity; V_{Emax}, maximal expiratory volume; VO_{2max}, maximum rate of oxygen consumption during exercise; V_T, tidal volume; V_{Tmax}, peak tidal volume during exercise; W, watts; 8-epi-PGF_{2α}, prostaglandin 2 alpha; ^{99m}Tc-DTPA, technetium 99m-labelled diethylenetriamine penta-acetic acid used aerosol ventilation studies

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- 27

APPENDIX 3B

**AIR QUALITY INFORMATION FOR LOCATIONS OF
EPIDEMIOLOGIC STUDIES OF RESPIRATORY EFFECTS**

1 This appendix provides summary information about the O₃ concentrations in locations
2 and time periods of epidemiologic studies of associations between O₃ in ambient air and
3 respiratory health outcomes. Included here are studies conducted in the U.S. and Canada that
4 found associations between O₃ exposure and respiratory health effects such as emergency
5 department visits and hospital admissions, including studies that are newly available since the
6 2020 review, as well as those that were available at the time of the 2015 review, and that are
7 identified in the ISA. Information for studies identified in the ISA¹ as short-term are summarized
8 in Table 3B-1 and a subset of studies identified as long-term are summarized in Table 3B-2.

9 Air quality information for U.S.-based studies was obtained from the EPA's Air Quality
10 System (AQS) database.² For Canada-based studies, air quality information was obtained from
11 the National Air Pollutant Surveillance (NAPS) program.³ In Table 3B-1 and Table 3B-2, design
12 values (DVs)⁴ are presented as a range across all locations and time periods in the study.⁵
13 Detailed information about designs values for individual study locations and time periods are
14 available in the Attachment.⁶

¹ Single- and multi-city studies are included. Given the purpose of describing the air quality conditions in the cities studied, meta-analysis studies are not included; rather, the relevant underlying studies would be.

² Available at: <https://www.epa.gov/aqs>.

³ Available at: <https://www.canada.ca/en/environment-climate-change/services/air-pollution/monitoring-networks-data/national-air-pollution-program.html>.

⁴ The design value for the current standard is the 3-year average of the annual 4th highest daily maximum 8-hour average O₃ concentration.

⁵ For those locations with more than one monitor, the design values presented in Table 3B-1, Table 3B-2, and in the attachment for that location are for the highest monitor in that area.

⁶ In the attachment tables, blank cells indicate one of two situations: (1) monitoring data are unavailable for the specific time period or the entire period for the city, or (2) the available data do not meet the data requirements for the calculations.

1 **Table 3B-1. Epidemiologic studies of associations between short-term ozone concentrations and respiratory effects.**

Study Information									Ambient Air Quality
Study Area	Health Study Time Period	Air Quality Time Period	Study Reference ^A	Health Outcome	O ₃ Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects	Study-reported O ₃ Concentrations, in terms of study metric (ppb)		Design Values for Current NAAQS, across cities and study years (ppb) ^B
							Mean/median	Range	
U.S. Studies									
Single City Studies									
Indianapolis, IN	2007-2011	2007-2011	Byers et al., 2015	ED Visits for Asthma	8-hr daily maximums, moving average of lag 0-2	Distance and population-weighted daily average O ₃ concentration of 11 monitor values for the Indianapolis MSA (9 counties)	8-hr (WS): 48.5	NA	73-77
Atlanta, GA	1993-2004	1993-2004	Darrow et al., 2011	ED Visits for Aggregate Respiratory Diseases	1-hr and 8-hr daily maximums, previous day lag (lag 1)	Daily O ₃ concentration of single centrally located monitor in the Atlanta MSA	1-hr (WS): 62.0 8-hr (WS): 53.0	1-h Max: 180.0 8-hr Max: 148.0	91-121
Atlanta, GA	1993-2010	1993-2010	Darrow et al., 2014	ED Visits for Respiratory Infection	8-hr daily maximum, 3-day moving average of lag 0-2	Population-weighted daily average O ₃ concentration of 5 monitor values for the Atlanta MSA (20 counties)	8-hr (YR): 45.9	3.0-127.1	80-121
New Jersey	2004-2007	2004-2007	Gleason et al., 2014	ED Visits for Asthma	8-hr daily maximum, same day lag (lag 0)	Daily O ₃ concentration obtained from Bayesian spatio-temporal model assigned to study participants based on corresponding grid cells for geocoded residential addresses	NA	NA	92-93
New York, NY	1999-2009	1999-2009	Goodman et al., 2017a	HA for Asthma	8-hr daily maximum, average of lag 0-1	Daily average O ₃ concentrations of all monitors within 20-mile of the geographic center of NY city	8-hr (YR): 30.7	2.0-105.4	84-115
New York, NY	1999-2002	1999-2002	Ito et al., 2007	ED Visits for Asthma	8-hr daily maximum, average of lag 0-1	Average of 16 monitors within 20 miles of the geographic city center of NY city	8-hr (YR): 30.4 8-hr (WS): 42.7	5 th and 95 th percentiles: YR: 6.0-68.0 WS: 18.0-77.0	109-115
Atlanta, GA	1998-2007	1998-2007	Klemm et al., 2011	Respiratory Mortality	8-hr daily maximum, average of lag 0-1	Daily average O ₃ concentration of all monitors in four counties in Atlanta	8-hr (YR): 35.5	0.0-109.1	90-121

Study Information									Ambient Air Quality
Study Area	Health Study Time Period	Air Quality Time Period	Study Reference ^A	Health Outcome	O ₃ Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects	Study-reported O ₃ Concentrations, in terms of study metric (ppb)		Design Values for Current NAAQS, across cities and study years (ppb) ^B
							Mean/median	Range	
Atlanta, GA	2002-2008	2002-2008	O'Lenick et al., 2017	ED Visits for Asthma	8-hr daily maximum, 3-day moving average of lag 0-2	Daily O ₃ concentration obtained from spatio-temporal model assigned to study participants based on corresponding ZCTA for residential ZIP code	NA	NA	90-95
Little Rock, AR	2002-2012	2002-2012	Rodopoulou et al., 2015	ED Visits for Respiratory Infection	8-hr daily maximum, lag 2	Daily O ₃ concentration from one monitor in Little Rock, AR	8-hr (YR): 40.0	NA	70-83
Atlanta, GA	1999-2002	1999-2002	Sarnat et al., 2013	ED Visits for Asthma	24-hr daily average	Spatially resolved daily O ₃ concentration at ZIP code centroid assigned to participants based on residential ZIP code	8-hr (YR): 41.9	3.5-132.7	99-107
St. Louis, MO	2001-2003	2001-2004	Sarnat et al., 2015	ED Visits for Asthma	8-hr daily maximum, distributed lags (lags 0-2)	Daily O ₃ concentration from one monitor in St. Louis, MO.	8-hr (YR): 36.2	NA	92
New York, NY	2005-2011	2005-2012	Sheffield et al., 2015	ED Visits for Asthma	24-hr daily average	Daily average O ₃ concentration of seven monitors in NYC.	NA	NA	82-94
New York, NY	2005-2011	2005-2011	Shmool et al., 2016	ED Visits for Asthma	24-hr daily average, case-day	Near-residence exposure was determined by combining data from temporally- and spatially-refined estimates	Temporal estimates (WS): 30.4 Spatiotemporal estimates: 29.0	Temporal estimates: 5.0-60.0 Spatiotemporal estimates: 4.6-60.3	82-94
New York, NY	1999-2006	1999-2006	Silverman and Ito, 2010	HA for Asthma	8-hr daily maximum, average of lag 0-1	Average of 13 monitors within 20 miles of the geographic city center of NY city	8-hr (WS): 41.0	10 th and 90 th percentiles: 18.0-77.0	93-115
Atlanta, GA	2002-2010	2002-2010	Strickland et al., 2014	ED Visits for Asthma	8-hr daily maximum, 3-day moving average lag 0-2	Distance and population-weighted daily average of five monitor values for the Atlanta MSA (20 counties)	8-hr (YR): 42.2	NA	80-95
Atlanta, GA	1993-2004	1993-2004	Tolbert et al., 2007	ED Visits for Aggregate Respiratory Diseases	8-hr daily maximum, average of lag 0-1	Average of monitors in Atlanta city	8-hr (EC): 53.0	2.9-147.5	91-121

Study Information									Ambient Air Quality
Study Area	Health Study Time Period	Air Quality Time Period	Study Reference ^A	Health Outcome	O ₃ Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects	Study-reported O ₃ Concentrations, in terms of study metric (ppb)		Design Values for Current NAAQS, across cities and study years (ppb) ^B
							Mean/median	Range	
St. Louis, MO	2001-2007	2001-2007	Winqvist et al., 2012	HA for Asthma ED Visits for Asthma ED Visits for Respiratory Infection HA for Aggregate Respiratory Diseases ED Visits Aggregate Respiratory Diseases	8-hr daily maximum, distributed lags (lags 0-4)	Daily O ₃ concentration from one monitor in St. Louis, MO.	NA	NA	86-92
Atlanta, GA	1998-2004	1998-2004	Winqvist et al., 2014	ED Visits for Asthma	8-hr daily maximum, 3-day moving average of lag 0-2	Population-weighted daily average of five monitor values for the Atlanta MSA (20 counties)	8-hr (WS): 53.9	NA	91-121
Multi-city Studies									
3 U.S. cities	1993-2009	1993-2009	Alhanti et al., 2016	ED Visits for Asthma	8-hr maximum, 3-day moving average of lag 0-2	Population-weighted daily average of monitor values for each city	8-hr (YR) for 3 cities mean range: 37.3-43.7	NA	86-121
5 U.S. cities	2002-2008	2002-2008	Barry et al., 2018	ED Visits for Asthma ED Visits for Respiratory Infection ED Visits Aggregate Respiratory Diseases	8-hr maximum, 3-day moving average of lag 0-2	Daily O ₃ concentration obtained model simulations and monitor measurements were spatially averaged for each metropolitan area using population weighting	8-hr (YR) for 5 cities mean range: 37.5-42.2	Min Range: 3.9-9.4 Max Range: 80.2-106.3	83-95
3 metro areas in TX	2003-2011	2003-2011	Goodman et al., 2017b	HA for Asthma	8-hr maximum, same day lag (lag 0)	City-specific daily O ₃ concentrations were calculated using all monitors within each city: Dallas (8 monitors), Houston (44 monitors), Austin (6 monitors), then were averaged to obtain area-specific daily maximum 8-hr concentrations	8-hr (YR): 41.8	2.0-107.0	74-103
Nationwide (U.S.)	1987-1996	1987-1996	Katsouyanni et al., 2009	Respiratory Mortality	1-hr maximum, 2-day average of lag 0-1	Daily average of O ₃ concentrations from all monitors in each city	NA	NA	18-192

Study Information									Ambient Air Quality
Study Area	Health Study Time Period	Air Quality Time Period	Study Reference ^A	Health Outcome	O ₃ Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects	Study-reported O ₃ Concentrations, in terms of study metric (ppb)		Design Values for Current NAAQS, across cities and study years (ppb) ^B
							Mean/median	Range	
California	2005-2008	2005-2009	Malig et al., 2016	ED Visits for Asthma ED Visits for Respiratory Infection ED Visits Aggregate Respiratory Diseases	1-hr maximum, 2-day average of lag 0-1	Daily O ₃ concentration from nearest monitor within 20 km of population-weighted ZIP code centroid assigned to participants based on residential ZIP code	8-hr for 16 climatic zones mean range: (YR): 33.0-55.0 (WS): 31.0-75.0	NA	119-122
3 U.S. cities	2002-2008	2002-2008	O'Lenick et al., 2017	ED Visits Aggregate Respiratory Diseases	8-hr daily maximum, 3-day moving average of lag 0-2	Daily O ₃ concentration obtained from spatio-temporal model assigned to study participants based on corresponding ZCTA for residential ZIP code	8-hr (YR) for 3 cities mean ranges from 40.0-42.2	Min Range: 0.15-2.21 Max Range: 115-125	85-96
North Carolina	2006-2008	2006-2008	Sacks et al., 2014	ED Visits for Asthma	8-hr daily maximum, 3-day moving average of lag 0-2	O ₃ estimates from CMAQ model with Bayesian space-time approach assigned to census tract centroids and aggregated to county-level using area-weighted average of census tract centroids	8-hr (YR): 43.6 8-hr (WS): 50.1	Max:108.1	94
Georgia	2002-2008	2002-2008	Xiao et al., 2016	ED Visits for Asthma ED Visits for Respiratory Infection	8-hr daily maximum, 3-day moving average of lag 0-2	Daily O ₃ concentration obtained from spatio-temporal model assigned to study participants based on residential ZIP code	8-hr (YR): 42.1	5.4-106.1	91-95
48 U.S. cities	1989-2000	1989-2000	Zanobetti and Schwartz, 2008	Respiratory Mortality	8-hr daily average, same day lag (lag 0)	Daily average of O ₃ concentrations from all monitors in each city	8-hr (WS) for 40 U.S. cities mean range: 15.1-62.8	Min Range: 0.9-23.6 Max Range: 34.3-146.2	45-179
6 cities in TX	2001-2013	2001-2013	Zu et al., 2017	HA for Asthma	8-hr daily maximum, lag 0-3	City specific daily O ₃ concentrations were calculated using all monitors within each city: Dallas (15 monitors), Houston (44 monitors), Austin (6 monitors), El Paso (6 monitors), Fort Worth (9 monitors); then were averaged to obtain area-specific daily maximum 8-hr concentrations.	8-hr (YR): 32.2	1.0-82.8	71-103

Study Information									Ambient Air Quality
Study Area	Health Study Time Period	Air Quality Time Period	Study Reference ^A	Health Outcome	O ₃ Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects	Study-reported O ₃ Concentrations, in terms of study metric (ppb)		Design Values for Current NAAQS, across cities and study years (ppb) ^B
							Mean/median	Range	
Canadian Studies									
Single City Studies									
Edmonton, Canada	1992-2002	1992-2002	Kousha and Rowe, 2014	ED Visits for Respiratory Infection	8-hr daily maximum, same day lag (lag 0).	Daily average of O ₃ concentrations from three monitors in Edmonton, Canada	8-hr (YR): 18.6	NA	56-65
Windsor, Canada	2004-2010	2004-2010	Kousha and Castner, 2016	ED Visits for Respiratory Infection	8-hr daily maximum, same day lag (lag 0).	Daily average of O ₃ concentrations from monitors in Windsor, Canada	8-hr (YR): 25.3	NA	73-87
Alberta, Canada	1992-2002	1992-2002	Villeneuve et al., 2007	ED Visits for Asthma	8-hr daily maximum, lag 1.	Daily average of three monitors in census metropolitan of Edmonton, Alberta	8-hr (WS): 38.0 (Median)	NA	60-69
Multi-city Studies									
7 Canadian cities	1992-2003	1992-2003	Stieb et al., 2009	ED visits for Asthma	24-hr average, lag 1	Daily average of O ₃ concentrations from monitors in each city	24-hr (YR): Mean range: 10.3-22.1	NA	51-85
9 Canadian cities	2004-2011	2004-2011	Szyszkowicz et al., 2018	ED Visits for Asthma ED Visits for Respiratory Infection	24-hr daily average, lag 1.	Daily average of O ₃ concentrations from all monitors within 35 km of participants residential 3-digit postal codes	24-hr (YR) for 9 urban areas/districts mean range: 22.5-29.2	Min Range: 1.0-3.0 Max Range: 60.7-80.0	57-79
10 Canadian cities	1981-1999	1981-1999	Vanos et al., 2014	Respiratory Mortality	24-hr daily average, lag 1.	Daily average O ₃ concentrations from all monitors either downtown or at city airports located within 27 km of downtown	24-hr (YR): 19.3	NA	51-94
ED – emergency department; HA – hospital admission; WS – warm season; YR – year round; ZCTA – ZIP code tabulation area									
^A Studies investigating associations between short-term O ₃ exposure and respiratory mortality are summarized in the following tables and figures of Appendix 3 of the ISA (U.S. EPA, 2020): HA for asthma: Table 3-13, Figure 3-4; ED visits for asthma: Table 3-14, Figure 3-5; ED visits for respiratory infection: Table 3-39, Figure 3-6; Respiratory-related HA and ED: Figure 3-7; HA for aggregate respiratory diseases: Table 3-41; ED visits for aggregate respiratory diseases: Table 3-42.									
^B For those studies available at the time of the last review, design values were drawn from (Wells, 2012) and are presented in units of ppm. For those studies available since the time of the last review, design values were calculated based on data available from the EPA's Air Quality System (AQS) for U.S. studies and the National Air Pollutant Surveillance (NAPS) program for Canadian studies.									

1 **Table 3B-2. Subset of epidemiologic studies of associations between long-term ozone and respiratory effects.**

Study Information									Ambient Air Quality Data
Study Area	Health Study Time Period	Air Quality Time Period	Study Reference ^A	Health Outcome	O ₃ Concentration Metric Associated with Health Outcome	Assignment of Monitors to Study Subjects	Study-reported O ₃ Concentrations, in terms of study metric (ppb)		Design Values for Current NAAQS, across cities and study years (ppb) ^B
							Mean/median	Range	
U.S. Studies, multi-city									
Nationwide	1982-2000	1977-2000	Jerrett et al., 2009	Respiratory Mortality	Long-term warm-season average O ₃ value including year 1977-2000	Study participants assigned long-term O ₃ concentrations for MSA of residence ^C	Mean range for MSAs: 33.33-104.0	NA	59-248
California	1982-2000	1988-2002	Jerrett et al., 2013	Respiratory Mortality	Monthly average O ₃ value calculated using IDW from year 1988-2002	Study participants were assigned O ₃ concentration based on their residential address corresponding to the study site ^D	50.35	17.11-89.33	128-186
California (9 areas)	1993-2001, 1996-2004, 2006-2014	1993--2014	Garcia et al., 2019	Asthma diagnosis	Areawide annual mean O ₃ concentration (10am-6pm)	Community-specific annual mean concentrations for each year of each of the three cohorts.	-	26-76	65-165 [for 1993-2014]
Canadian Studies, multi-city									
Nationwide	1991-2011	2002-2009	Weichenhal et al., 2017	Respiratory Mortality	Monthly average O ₃ value calculated using pollutant-specific interpolation techniques to generate 21 km ² grid cell concentrations	Study participants were assigned O ₃ concentration from interpolation surface based on their residential postal code ^E	38.29	<1-60.46	35-98
Quebec	1999-2010	1999-2010	Tétreault et al., 2016	Asthma incidence	Average summer (June-Aug) concentration [8hr midday concentration per ISA]	Study participants were assigned concentration estimated for postal code centroid using interpolation based approach.	Mean: 32.07 Median: 32.19	12.18-43.12	49-79

^A Studies investigating associations between long-term O₃ exposure and respiratory mortality are summarized in the ISA, Appendix 6, Table 6-8 and Figure 6-9 (U.S. EPA, 2020).
^B For those studies available at the time of the last review, design values were drawn from (Wells, 2012). For those studies available since the time of the last review, design values were calculated based on data available from the EPA's Air Quality System (AQS) for U.S. studies and the National Air Pollution Surveillance (NAPS) program for Canadian studies.
^C Data for monitors were obtained for 1977-2000. Daily maximum 1-hour O₃ concentrations were used to calculate quarterly averages for each monitor. Averages for quarters 2 and 3 were then averaged to create a warm-season average O₃ concentration for each monitor. The warm-season O₃ concentrations for the time period 1977-2000 were computed for each year to form a single annual time series of O₃ measurements for 96 metropolitan areas.
^D Inverse distance weighted monthly average O₃ concentrations for all sites within a 50 km radius of operating monitors were calculated for the years 1988-2002.
^E A surface for average daily 8-hour maximum O₃ concentrations was generated for the months of May-October for years 2002-2009 using an air pollution-specific interpolation technique to generate a 21 km² grid value. The interpolation method incorporates modeled O₃ from the Canadian Hemispheric Regional Ozone and NO_x (CHRONOS) air quality forecast model with observations from Canada and the U.S.

2

ATTACHMENT

DESIGN VALUES FOR LOCATIONS AND TIME PERIODS ANALYZED IN EPIDEMIOLOGIC STUDIES

NOTE: Design values generally provided in parts per billion (ppb) rather than parts per million (ppm) in tables below for simplicity of presentation.

Alhanti et al., 2016 (3019562) - ED Visits for Asthma

Three U.S. cities. O₃: Atlanta (1993–2009), Dallas (2006–2009), St. Louis (2001–2007)

City	Census Area Name	dv.1993 1995	dv.1994 .1996	dv.1995 .1997	dv.1996 .1998	dv.1997 .1999	dv.1998 .2000	dv.1999 .2001	dv.2000 .2002	dv.2001 .2003	dv.2002 .2004	dv.2003 .2005	dv.2004 .2006	dv.2005 .2007	dv.2006 .2008	dv.2007 .2009
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	109	105	110	113	118	121	107	99	91	93	90	91	95	95	87

City	Census Area Name	dv.2006.2008	dv.2007.2009
Dallas, TX	Dallas-Fort Worth, TX-OK	91	86

City	Census Area Name	dv.2001. 2003	dv.2002. 2004	dv.2003. 2005	dv.2004. 2006	dv.2005. 2007
St. Louis	St. Louis-St. Charles-Farmington, MO-IL	92	89	86	86	89

Barry et al., 2018 (4829120) - ED Visits for Asthma, ED Visits for Aggregate Respiratory Diseases, ED Visit - Respiratory Infection

Five U.S. Cities: 20-co Atlanta (2002-2008), 7-co Birmingham (2002-2008), 12-co Dallas-Ft Worth (2006-2008), 3-co Pittsburgh (2002-2008), 16-co St Louis (2002-2007)

City	Census Area Name	dv.2002. 2004	dv.2003. 2005	dv.2004. 2006	dv.2005. 2007	dv.2006. 2008
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	93	90	91	95	95

City	Census Area Name	dv.2002. 2004	dv.2003. 2005	dv.2004. 2006	dv.2005. 2007	dv.2006. 2008
Birmingham, AL	Birmingham-Hoover-Talladega, AL	85	84	85	89	87

City	Census Area Name	dv.2006.2008
Dallas-Ft Worth	Dallas-Fort Worth, TX-OK	91

City	Census Area Name	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008
Pittsburgh, PA	Pittsburgh-New Castle-Weirton, PA-OH-WV	90	84	83	87	86

City	Census Area Name	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007
St. Louis	St. Louis-St. Charles-Farmington, MO-IL	89	86	86	89

Byers et al., 2015 (3019032) - ED Visits for Asthma

Indianapolis MSA (Marion and 8 surrounding counties), IN, U.S. O₃: 2007-2011

City	Census Area Name	dv.2007.2009	dv.2008.2010	dv.2009.2011
Indianapolis, IN	Indianapolis-Carmel-Muncie, IN	77	73	74

Cakmak et al., 2017 (4167344) - Long-Term Ozone and Respiratory Mortality

Nationwide, Canada. O₃: 2002-2009

Air quality data are not described for this study as it relied on O₃ concentrations for the years 2002–2009 as surrogates for study population annual O₃ concentrations during the 1984 to 2011 period (Cakmak, 2017).

Crouse et al., 2015 (3019335) - Long-Term Ozone and Respiratory Mortality

Nationwide, Canada. O₃: 2002-2009

Air quality data are not described for this study as it relied on O₃ concentrations for the years 2002–2009 as surrogates for study population annual O₃ concentrations during the 1984 to 2006 period (Crouse, 2015).

1 **Darrow et al., 2011** (202800) - ED Visits for Aggregate Respiratory Diseases

2 20-county Atlanta area, GA, U.S. O₃: 1993-2004

City	Census Area Name	dv1993_1995	dv1994_1996	dv1995_1997	dv1996_1998	dv1997_1999	dv1998_2000	dv1999_2001	dv2000_2002	dv2001_2003	dv2002_2004
Atlanta, GA	Atlanta-Sandy Springs-Marietta, GA	0.109	0.105	0.110	0.113	0.118	0.121	0.107	0.099	0.091	0.093

Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.

3
4 **Darrow et al., 2014** (2526768) - ED Visit - Respiratory Infection

5 20-county Atlanta area, GA, U.S. O₃: 1993-2010

City	Census Area Name	dv.1993.1995	dv.1994.1996	dv.1995.1997	dv.1996.1998	dv.1997.1999	dv.1998.2000	dv.1999.2001	dv.2000.2002
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	109	105	110	113	118	121	107	99
		dv.2001.2003	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010
		91	93	90	91	95	95	87	80

6
7 **Eckel et al., 2016** (3426159) - Long-Term Ozone and Respiratory Mortality

8 California, U.S. O₃: 1988-2011

State	dv.1988.1990	dv.1989.1991	dv.1990.1992	dv.1991.1993	dv.1992.1994	dv.1993.1995	dv.1994.1996	dv.1995.1997	dv.1996.1998	dv.1997.1999	dv.1998.2000
California	186	182	180	177	171	165	161	148	154	147	146
	dv.1999.2001	dv.2000.2002	dv.2001.2003	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010	dv.2009.2011
	129	128	131	127	127	121	122	119	118	112	107

9
10

1 **Garcia et al., 2019** (5119704) - Asthma Incidence
 2 Nine communities in Southern California, U.S. O₃: 1993-2014

City	Census Area Name	dv.1993 .1995	dv.1994 .1996	dv.1995 .1997	dv.1996 .1998	dv.1997 .1999	dv.1998 .2000	dv.1999. 2001	dv.2000 .2002	dv.2001 .2003	dv.2002 .2004
Long Beach, San Dimas	Los Angeles-Long Beach-Anaheim, CA (CBSA)	156	145	135	133	118	115	105	113	126	125
Lake Elsinore, Lake Gregory, Mira Loma, Riverside, Upland	Riverside-San Bernardino-Ontario, CA (CBSA)	165	161	148	154	147	146	129	128	131	127
Alpine	San Diego-Carlsbad, CA (CBSA)	108	104	99	102	99	100	94	95	93	89
Santa Maria	Santa Maria-Santa Barbara, CA (CBSA)	90	94	89	87	82	81	80	82	84	82

3

City	Census Area Name	dv.2003 .2005	dv.2004 .2006	dv.2005 .2007	dv.2006 .2008	dv.2007 .2009	dv.2008 .2010	dv.2009. 2011	dv.2010 .2012	dv.2011 .2013	dv.2012 .2014
Long Beach, San Dimas	Los Angeles-Long Beach-Anaheim, CA (CBSA)	120	112	110	107	108	103	97	96	99	97
Lake Elsinore, Lake Gregory, Mira Loma, Riverside, Upland	Riverside-San Bernardino-Ontario, CA (CBSA)	127	121	122	119	118	112	107	106	107	102
Alpine	San Diego-Carlsbad, CA (CBSA)	86	88	89	92	89	88	82	91	80	79
Santa Maria	Santa Maria-Santa Barbara, CA (CBSA)	78	75	75	73	77	76	73	68	65	68

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5

1 **Gleason et al., 2014** (2369662) - ED Visits for Asthma
 2 New Jersey (statewide), U.S. O₃: April-September, 2004-2007

State	dv.2004.2006	dv.2005.2007
New Jersey	93	92

3
 4 **Goodman et al., 2017a** (3859548) - Hospital Admissions for Asthma,
 5 New York City (20-mi radius from center), NY, U.S. O₃: 1999-2009

City	Census Area Name	dv.1999 .2001	dv.2000 .2002	dv.2001 .2003	dv.2002 .2004	dv.2003 .2005	dv.2004 .2006	dv.2005 .2007	dv.2006 .2008	dv.2007 .2009
New York, NY	New York-Newark, NY-NJ-CT-PA	109	115	109	102	94	93	94	89	84

6
 7 **Goodman et al., 2017b** (4169406) - Hospital Admissions for Asthma
 8 Houston, Dallas, and Austin, TX metro areas, U.S. O₃: 2003-2011

City	Census Area Name	dv.2003. 2005	dv.2004. 2006	dv.2005. 2007	dv.2006. 2008	dv.2007. 2009	dv.2008. 2010	dv.2009. 2011
Houston	Houston-The Woodlands, TX	103	103	96	91	84	84	89
Dallas	Dallas-Fort Worth, TX-OK	95	96	95	91	86	86	90
Austin	Austin-Round Rock, TX (CBSA ONLY)	82	82	80	77	75	74	75

9
 10 **Ito et al., 2007** (156594) - Emergency Department Visits for Asthma
 11 New York City, NY. O₃: 1999-2002

City	Census Area Name	dv.1999.2001	dv.2000.2002
New York, NY	New York-Northern New Jersey-Long Island, NY-NJ-PA	0.109	0.115
Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.			

12
 13
 14

1 **Jerrett et al., 2009 (194160) - Long-Term Ozone and Respiratory Mortality**
 2 Nationwide, U.S. O₃: 1977-2000

City	Census Area Name	dv1977_ 1979	dv1978_ 1980	dv1979_ 1981	dv1980_ 1982	dv1981_ 1983	dv1982_ 1984	dv1983_ 1985
Charleston, SC	Charleston-North Charleston-Summerville, SC		0.088	0.08	0.074	0.072	0.076	0.077
Charleston, WV	Charleston, WV	0.077	0.077	0.075	0.078	0.082	0.086	0.087
Charlotte, NC	Charlotte-Gastonia-Concord, NC-SC				0.1	0.099	0.097	0.098
Chattanooga, TN	Chattanooga, TN-GA		0.09	0.094	0.097	0.097	0.093	0.091
Chicago, IL	Chicago-Naperville-Joliet, IL-IN-WI	0.112	0.112	0.1	0.096	0.103	0.103	0.106
Cincinnati, OH	Cincinnati-Middletown, OH-KY-IN	0.119	0.109	0.104	0.101	0.1	0.1	0.097
Cleveland, OH	Cleveland-Elyria-Mentor, OH	0.108	0.101	0.094	0.092	0.096	0.098	0.1
Colorado Springs, CO	Colorado Springs, CO				0.06	0.06	0.063	0.062
Columbia, SC	Columbia, SC	0.078	0.109	0.091	0.087	0.088	0.084	0.082
Columbus, OH	Columbus, OH	0.098	0.103	0.091	0.093	0.092	0.094	0.093
Corpus Christi, TX	Corpus Christi, TX					0.079	0.086	0.084
Dallas/Ft Worth, TX	Dallas-Fort Worth-Arlington, TX		0.109	0.111	0.108	0.108	0.11	0.118
Dayton, OH	Dayton, OH	0.122	0.108	0.102	0.103	0.104	0.1	0.092
Denver, CO	Denver-Aurora-Broomfield, CO	0.091	0.089	0.088	0.084	0.089	0.087	0.082
Detroit, MI	Detroit-Warren-Livonia, MI	0.101	0.097	0.092	0.097	0.103	0.098	0.094
El Paso, TX	El Paso, TX					0.079	0.084	0.089
Evansville, IN	Evansville, IN-KY					0.096	0.094	0.092
Flint, MI	Flint, MI	0.082	0.086	0.082	0.085	0.088	0.087	0.08
Fresno, CA	Fresno, CA	0.101	0.103	0.123	0.123	0.116	0.114	0.11
Ft. Lauderdale, FL	Broward County, FL			0.074	0.075	0.071	0.069	0.069
City	Census Area Name	dv1977_ 1979	dv1978_ 1980	dv1979_ 1981	dv1980_ 1982	dv1981_ 1983	dv1982_ 1984	dv1983_ 1985
Gary, IN	Lake County, IN	0.105	0.098	0.087	0.09	0.095	0.097	0.095
Greely, CO	Greeley, CO					0.059	0.071	0.069
Greensboro, NC	Greensboro-High Point, NC			0.086	0.09	0.087	0.089	0.087
Greenville, SC	Greenville-Mauldin-Easley, SC			0.094	0.094	0.093	0.089	0.088
Harrisburg, PA	Harrisburg-Carlisle, PA		0.095	0.087	0.096	0.098	0.1	0.098
Houston, TX	Houston-Sugar Land-Baytown, TX	0.099	0.14	0.132	0.124	0.139	0.128	0.124

Huntington, WV	Huntington-Ashland, WV-KY-OH			0.088	0.09	0.095	0.097	0.097
Indianapolis, IN	Indianapolis-Carmel, IN	0.076	0.09	0.087	0.103	0.101	0.101	0.096
Jackson, MS	Jackson, MS	0.098	0.09	0.084	0.081	0.079	0.076	0.078
Jacksonville, FL	Jacksonville, FL	0.086	0.086	0.087	0.085	0.08	0.076	0.075
Jersey City, NJ	Hudson County, NJ							0.111
Johnstown, PA	Johnstown, PA	0.1	0.107	0.1	0.097	0.087	0.087	0.087
Kansas City, MO	Kansas City, MO-KS	0.074	0.081	0.097	0.089	0.089	0.094	0.096
Kenosha, WI	Kenosha County, WI				0.095	0.103	0.097	0.1
Knoxville, TN	Knoxville, TN					0.09	0.088	0.083
Lancaster, PA	Lancaster, PA	0.088	0.096	0.092	0.096	0.101	0.1	0.098
Lansing, MI	Lansing-East Lansing, MI			0.086	0.073	0.08	0.08	0.076
Las Vegas, NV	Las Vegas-Paradise, NV			0.074	0.085	0.085	0.08	0.079
Lexington, KY	Lexington-Fayette, KY		0.091	0.087	0.085	0.086	0.091	0.092
Little Rock, AR	Little Rock-North Little Rock-Conway, AR	0.098	0.107	0.1	0.085	0.082	0.083	0.087
Los Angeles, CA	Los Angeles-Long Beach-Santa Ana, CA	0.174	0.248	0.229	0.21	0.204	0.225	0.226
Madison, WI	Madison, WI	0.096	0.102	0.095	0.088	0.078	0.076	0.078
Memphis, TN	Memphis, TN-MS-AR	0.102	0.103	0.085	0.096	0.097	0.092	0.092
Milwaukee, WI	Milwaukee-Waukesha-West Allis, WI	0.114	0.11	0.11	0.106	0.111	0.104	0.105
Minneapolis, MN	Minneapolis-St. Paul-Bloomington, MN-WI			0.08	0.079	0.076	0.073	0.073
Nashville, TN	Nashville-Murfreesboro-Franklin, TN	0.092	0.085	0.077	0.083	0.083	0.09	0.095
Nassau, NY	Nassau County, NY							
New Haven, CT	New Haven-Milford, CT	0.135	0.127	0.118	0.121	0.13	0.136	0.128
New Orleans, LA	New Orleans-Metairie-Kenner, LA		0.087	0.087	0.085	0.083	0.099	0.089
City	Census Area Name	dv1977_1979	dv1978_1980	dv1979_1981	dv1980_1982	dv1981_1983	dv1982_1984	dv1983_1985
New York City, NY	New York-Northern New Jersey-Long Island, NY-NJ-PA	0.124	0.118	0.116	0.12	0.121	0.12	0.128
Newark, NJ	Essex County, NJ							
Norfolk, VA	Virginia Beach-Norfolk-Newport News, VA-NC	0.1	0.101	0.091	0.091	0.096	0.095	0.093
Oklahoma City, OK	Oklahoma City, OK	0.089	0.093	0.084	0.084	0.087	0.085	0.089
Orlando, FL	Orlando-Kissimmee, FL	0.078	0.08	0.077	0.078	0.078	0.078	0.074

Philadelphia, PA	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	0.126	0.136	0.127	0.125	0.114	0.122	0.119
Phoenix, AZ	Phoenix-Mesa-Scottsdale, AZ	0.076	0.078	0.081	0.085	0.09	0.093	0.096
Pittsburgh, PA	Pittsburgh, PA	0.111	0.123	0.109	0.104	0.106	0.099	0.099
Portland, ME	Portland-South Portland-Biddeford, ME					0.107	0.11	0.116
Portland, OR	Portland-Vancouver-Beaverton, OR-WA	0.084	0.088	0.082	0.082	0.081	0.074	0.076
Portsmouth, NH	Rockingham County, NH				0.097	0.094	0.082	0.077
Providence, RI	Providence-New Bedford-Fall River, RI-MA	0.121	0.124	0.124	0.121	0.115	0.121	0.121
Racine, WI	Racine, WI	0.093	0.112	0.108	0.109	0.113	0.112	0.111
Raleigh, NC	Raleigh-Cary, NC			0.088	0.091	0.089	0.085	0.087
Reading, PA	Reading, PA	0.098	0.105	0.109	0.114	0.106	0.102	0.1
Richmond, VA	Richmond, VA				0.084	0.098	0.098	0.099
Riverside, CA	Riverside-San Bernardino-Ontario, CA	0.239	0.245	0.235	0.217	0.21	0.209	0.211
Roanoke, VA	Roanoke, VA					0.083	0.086	0.084
Rochester, NY	Rochester, NY	0.093	0.091	0.084	0.086	0.09	0.091	0.09
Sacramento, CA	Sacramento-Arden Arcade-Roseville, CA			0.102	0.112	0.114	0.115	0.118
Salinas, CA	Salinas, CA		0.066	0.061	0.061	0.057	0.065	0.074
San Antonio, TX	San Antonio, TX		0.086	0.089	0.092	0.09	0.087	0.086
San Diego, CA	San Diego-Carlsbad-San Marcos, CA	0.115	0.118	0.141	0.137	0.13	0.126	0.132
San Francisco, CA	San Francisco-Oakland-Fremont, CA	0.085	0.092	0.086	0.091	0.089	0.091	0.096
San Jose, CA	San Jose-Sunnyvale-Santa Clara, CA	0.093	0.101	0.102	0.094	0.095	0.1	0.103
Seattle, WA	Seattle-Tacoma-Bellevue, WA	0.088	0.081	0.084	0.085	0.08	0.069	0.069
Shreveport, LA	Shreveport-Bossier City, LA				0.08	0.081	0.077	0.079
South Bend, IN	South Bend-Mishawaka, IN-MI		0.093	0.093	0.102	0.095	0.09	0.088
City	Census Area Name	dv1977_1979	dv1978_1980	dv1979_1981	dv1980_1982	dv1981_1983	dv1982_1984	dv1983_1985
Springfield, MA	Springfield, MA						0.1	0.112
St Louis, MO	St. Louis, MO-IL	0.122	0.117	0.109	0.101	0.107	0.111	0.113
Steubenville, OH	Weirton-Steubenville, WV-OH	0.098	0.099	0.088	0.083	0.073	0.071	0.064
Syracuse, NY	Syracuse, NY							
Tacoma, WA	Seattle-Tacoma-Bellevue, WA	0.088	0.081	0.084	0.085	0.08	0.069	0.069
Tampa, FL	Tampa-St. Petersburg-Clearwater, FL	0.09	0.088	0.087	0.087	0.089	0.09	0.087

Toledo, OH	Toledo, OH	0.108	0.104	0.102	0.1	0.101	0.09	0.087
Trenton, NJ	Trenton-Ewing, NJ					0.116	0.117	0.12
Tucson, AZ	Tucson, AZ	0.07	0.074	0.074	0.082	0.081	0.082	0.079
Vallejo, CA	Vallejo-Fairfield, CA	0.068	0.069	0.063	0.074	0.072	0.074	0.075
Ventura, CA	Ventura County, CA	0.13	0.13	0.109	0.104	0.098	0.112	0.113
Washington, DC	Washington-Arlington-Alexandria, DC-VA-MD-WV	0.112	0.101	0.101	0.113	0.113	0.112	0.11
Wichita, KS	Wichita, KS				0.074	0.078	0.079	0.081
Wilmington, DE	New Castle County, DE		0.083	0.088	0.093	0.106	0.112	0.116
Worcester, MA	Worcester, MA			0.102		0.092	0.096	0.099
York, PA	York-Hanover, PA	0.105	0.107	0.098	0.096	0.097	0.098	0.099
Youngstown, OH	Youngstown-Warren-Boardman, OH-PA					0.097	0.093	0.089

Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.

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Jerrett et al., 2009 (194160) - Long-Term Ozone and Respiratory Mortality (Continued)

City	Census Area Name	dv1984 _1986	dv1985 _1987	dv1986 _1988	dv1987 _1989	dv1988 _1990	dv1989 _1991	dv1990 _1992
Charleston, SC	Charleston-North Charleston-Summerville, SC	0.081	0.085	0.09	0.087	0.083	0.076	0.074
Charleston, WV	Charleston, WV	0.084	0.087	0.099	0.094	0.089	0.081	0.074
Charlotte, NC	Charlotte-Gastonia-Concord, NC-SC	0.094	0.102	0.112	0.104	0.101	0.092	0.091
Chattanooga, TN	Chattanooga, TN-GA	0.089	0.089	0.094	0.092	0.09	0.086	0.083
Chicago, IL	Chicago-Naperville-Joliet, IL-IN-WI	0.098	0.101	0.112	0.114	0.114	0.104	0.099
Cincinnati, OH	Cincinnati-Middletown, OH-KY-IN	0.093	0.098	0.109	0.106	0.107	0.102	0.095
Cleveland, OH	Cleveland-Elyria-Mentor, OH	0.094	0.092	0.104	0.105	0.104	0.093	0.09
Colorado Springs, CO	Colorado Springs, CO	0.062	0.06	0.061	0.063	0.065	0.066	0.063
Columbia, SC	Columbia, SC	0.081	0.084	0.069	0.091	0.091	0.081	0.084
Columbus, OH	Columbus, OH	0.089	0.089	0.093	0.097	0.095	0.089	0.092
Corpus Christi, TX	Corpus Christi, TX	0.078	0.083	0.086	0.089	0.085	0.079	0.077
Dallas/Ft Worth, TX	Dallas-Fort Worth-Arlington, TX	0.113	0.108	0.101	0.1	0.105	0.105	0.099
Dayton, OH	Dayton, OH	0.088	0.09	0.095	0.096	0.092	0.086	0.082
Denver, CO	Denver-Aurora-Broomfield, CO	0.079	0.081	0.088	0.087	0.086	0.08	0.074
Detroit, MI	Detroit-Warren-Livonia, MI	0.089	0.093	0.1	0.099	0.099	0.096	0.091
El Paso, TX	El Paso, TX	0.096	0.096	0.092	0.088	0.083	0.08	0.079

City	Census Area Name	dv1984 _1986	dv1985 _1987	dv1986 _1988	dv1987 _1989	dv1988 _1990	dv1989 _1991	dv1990 _1992
Evansville, IN	Evansville, IN-KY	0.09	0.094	0.099	0.1	0.099	0.091	0.088
Flint, MI	Flint, MI	0.077	0.079	0.09	0.091	0.09	0.085	0.081
Fresno, CA	Fresno, CA	0.117	0.118	0.121	0.115	0.11	0.108	0.108
Ft. Lauderdale, FL	Broward County, FL	0.073	0.073	0.077	0.076	0.079	0.075	0.073
Gary, IN	Lake County, IN	0.088	0.087	0.093	0.096	0.092	0.087	0.083
Greely, CO	Greeley, CO	0.067	0.068	0.07	0.072	0.074	0.075	0.072
Greensboro, NC	Greensboro-High Point, NC	0.089	0.089	0.1	0.097	0.1	0.088	0.085
Greenville, SC	Greenville-Mauldin-Easley, SC	0.085	0.089	0.091	0.09	0.085	0.075	0.075
Harrisburg, PA	Harrisburg-Carlisle, PA	0.091	0.096	0.103	0.103	0.098	0.094	0.091
Houston, TX	Houston-Sugar Land-Baytown, TX	0.127	0.127	0.118	0.117	0.119	0.119	0.116
Huntington, WV	Huntington-Ashland, WV-KY-OH	0.09	0.093	0.099	0.103	0.103	0.092	0.096
Indianapolis, IN	Indianapolis-Carmel, IN	0.09	0.091	0.096	0.098	0.095	0.091	0.089
Jackson, MS	Jackson, MS	0.077	0.076	0.077	0.075	0.079	0.076	0.076
Jacksonville, FL	Jacksonville, FL	0.075	0.081	0.084	0.086	0.084	0.081	0.079
Jersey City, NJ	Hudson County, NJ	0.104	0.109	0.117	0.118	0.115	0.107	0.104
Johnstown, PA	Johnstown, PA	0.085	0.087	0.097	0.097	0.093	0.086	0.083
Kansas City, MO	Kansas City, MO-KS	0.089	0.084	0.088	0.088	0.086	0.082	0.083
Kenosha, WI	Kenosha County, WI	0.089	0.098	0.111	0.114	0.114	0.104	0.099
Knoxville, TN	Knoxville, TN	0.094	0.087	0.097	0.093	0.094	0.086	0.089
Lancaster, PA	Lancaster, PA	0.09	0.091	0.097	0.097	0.093	0.09	0.09
Lansing, MI	Lansing-East Lansing, MI	0.073	0.077	0.09	0.089	0.087	0.081	0.082
Las Vegas, NV	Las Vegas-Paradise, NV	0.08	0.083	0.082	0.081	0.078	0.078	0.076
Lexington, KY	Lexington-Fayette, KY	0.092	0.094	0.099	0.099	0.096	0.085	0.078
Little Rock, AR	Little Rock-North Little Rock-Conway, AR	0.087	0.089	0.09	0.085	0.082	0.079	0.08
Los Angeles, CA	Los Angeles-Long Beach-Santa Ana, CA	0.222	0.217	0.205	0.192	0.186	0.179	0.177
Madison, WI	Madison, WI	0.075	0.079	0.09	0.091	0.079	0.081	0.079
Memphis, TN	Memphis, TN-MS-AR	0.093	0.096	0.1	0.095	0.095	0.089	0.091
Milwaukee, WI	Milwaukee-Waukesha-West Allis, WI	0.095	0.105	0.113	0.117	0.105	0.101	0.095
Minneapolis, MN	Minneapolis-St. Paul-Bloomington, MN-WI	0.071	0.073	0.077	0.08	0.079	0.075	0.071
Nashville, TN	Nashville-Murfreesboro-Franklin, TN	0.097	0.098	0.106	0.104	0.104	0.096	0.096

City	Census Area Name	dv1984 _1986	dv1985 _1987	dv1986 _1988	dv1987 _1989	dv1988 _1990	dv1989 _1991	dv1990 _1992
Nassau, NY	Nassau County, NY							
New Haven, CT	New Haven-Milford, CT	0.115	0.108	0.112	0.113	0.116	0.116	0.113
New Orleans, LA	New Orleans-Metairie-Kenner, LA	0.089	0.088	0.094	0.09	0.085	0.077	0.08
New York City, NY	New York-Northern New Jersey-Long Island, NY-	0.119	0.122	0.129	0.129	0.128	0.122	0.116
Newark, NJ	Essex County, NJ		0.086	0.092	0.105	0.098	0.088	0.086
Norfolk, VA	Virginia Beach-Norfolk-Newport News, VA-NC	0.087	0.089	0.095	0.093	0.091	0.084	0.086
Oklahoma City, OK	Oklahoma City, OK	0.087	0.084	0.085	0.087	0.087	0.086	0.084
Orlando, FL	Orlando-Kissimmee, FL	0.075	0.078	0.082	0.082	0.082	0.08	0.079
Philadelphia, PA	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	0.119	0.123	0.132	0.123	0.12	0.113	0.107
Phoenix, AZ	Phoenix-Mesa-Scottsdale, AZ	0.09	0.086	0.081	0.077	0.082	0.083	0.091
Pittsburgh, PA	Pittsburgh, PA	0.09	0.093	0.104	0.107	0.098	0.092	0.088
Portland, ME	Portland-South Portland-Biddeford, ME	0.112	0.112	0.112	0.117	0.115	0.109	0.105
Portland, OR	Portland-Vancouver-Beaverton, OR-WA	0.085	0.086	0.085	0.077	0.085	0.082	0.091
Portsmouth, NH	Rockingham County, NH	0.078	0.087	0.094	0.104	0.1	0.098	0.092
Providence, RI	Providence-New Bedford-Fall River, RI-MA	0.114	0.107	0.113	0.108	0.108	0.107	0.105
Racine, WI	Racine, WI	0.102	0.107	0.12	0.124	0.11	0.098	0.088
Raleigh, NC	Raleigh-Cary, NC	0.087	0.092	0.104	0.099	0.093	0.089	0.086
Reading, PA	Reading, PA	0.092	0.096	0.104	0.105	0.102	0.096	0.094
Richmond, VA	Richmond, VA	0.095	0.097	0.104	0.103	0.097	0.087	0.087
Riverside, CA	Riverside-San Bernardino-Ontario, CA	0.21	0.2	0.188	0.188	0.185	0.182	0.18
Roanoke, VA	Roanoke, VA	0.083	0.087	0.095	0.092	0.085	0.076	0.074
Rochester, NY	Rochester, NY	0.09	0.091	0.099	0.099	0.095	0.092	0.09
Sacramento, CA	Sacramento-Arden Arcade-Roseville, CA	0.118	0.114	0.114	0.114	0.107	0.105	0.105
Salinas, CA	Salinas, CA	0.071	0.071	0.068	0.072	0.07	0.07	0.071
San Antonio, TX	San Antonio, TX	0.085	0.083	0.084	0.085	0.085	0.082	0.079
San Diego, CA	San Diego-Carlsbad-San Marcos, CA	0.125	0.124	0.121	0.125	0.129	0.125	0.118
San Francisco, CA	San Francisco-Oakland-Fremont, CA	0.093	0.089	0.087	0.089	0.087	0.084	0.082
San Jose, CA	San Jose-Sunnyvale-Santa Clara, CA	0.097	0.092	0.092	0.097	0.088	0.082	0.083
Seattle, WA	Seattle-Tacoma-Bellevue, WA	0.075	0.077	0.074	0.076	0.079	0.078	0.086
Shreveport, LA	Shreveport-Bossier City, LA	0.082	0.085	0.086	0.087	0.088	0.084	0.086

City	Census Area Name	dv1984_1986	dv1985_1987	dv1986_1988	dv1987_1989	dv1988_1990	dv1989_1991	dv1990_1992
South Bend, IN	South Bend-Mishawaka, IN-MI	0.081	0.088	0.092	0.093	0.087	0.08	0.083
Springfield, MA	Springfield, MA	0.102	0.096	0.106	0.109	0.115	0.107	0.105
St Louis, MO	St. Louis, MO-IL	0.103	0.102	0.114	0.111	0.102	0.098	0.098
Steubenville, OH	Weirton-Steubenville, WV-OH	0.062	0.069	0.086	0.09	0.088	0.085	0.083
Syracuse, NY	Syracuse, NY		0.083	0.096	0.092	0.088	0.083	0.083
Tacoma, WA	Seattle-Tacoma-Bellevue, WA	0.075	0.077	0.074	0.076	0.079	0.078	0.086
Tampa, FL	Tampa-St. Petersburg-Clearwater, FL	0.088	0.091	0.09	0.086	0.085	0.079	0.081
Toledo, OH	Toledo, OH	0.079	0.083	0.097	0.102	0.099	0.086	0.082
Trenton, NJ	Trenton-Ewing, NJ	0.11	0.114	0.124	0.123	0.117	0.111	0.112
Tucson, AZ	Tucson, AZ	0.076	0.074	0.069	0.071	0.075	0.074	0.075
Vallejo, CA	Vallejo-Fairfield, CA	0.073	0.077	0.079	0.082	0.075	0.074	0.074
Ventura, CA	Ventura County, CA	0.116	0.114	0.131	0.132	0.13	0.126	0.117
Washington, DC	Washington-Arlington-Alexandria, DC-VA-MD-WV	0.104	0.11	0.116	0.115	0.107	0.1	0.1
Wichita, KS	Wichita, KS	0.077	0.076	0.08	0.08	0.081	0.075	0.074
Wilmington, DE	New Castle County, DE	0.102	0.106	0.114	0.114	0.115	0.107	0.101
Worcester, MA	Worcester, MA	0.091	0.086	0.088	0.091	0.091	0.089	0.091
York, PA	York-Hanover, PA	0.093	0.094	0.1	0.099	0.099	0.094	0.093
Youngstown, OH	Youngstown-Warren-Boardman, OH-PA	0.085	0.089	0.101	0.103	0.099	0.09	0.091
Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb								

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Jerrett et al., 2009 (194160) - Long-Term Ozone and Respiratory Mortality (Continued)

City	Census Area Name	dv1991_1993	dv1992_1994	dv1993_1995	dv1994_1996	dv1995_1997	dv1996_1998	dv1997_1999	dv1998_2000
Charleston, SC	Charleston-North Charleston-Summerville, SC	0.074	0.075	0.074	0.072	0.076	0.077	0.079	0.082
Charleston, WV	Charleston, WV	0.069	0.064	0.076	0.081	0.081	0.081	0.09	0.093
Charlotte, NC	Charlotte-Gastonia-Concord, NC-SC	0.091	0.092	0.094	0.094	0.097	0.103	0.104	0.104
Chattanooga, TN	Chattanooga, TN-GA	0.082	0.086	0.091	0.091	0.09	0.093	0.094	0.097
Chicago, IL	Chicago-Naperville-Joliet, IL-IN-WI	0.1	0.093	0.099	0.097	0.096	0.091	0.095	0.093
Cincinnati, OH	Cincinnati-Middletown, OH-KY-IN	0.091	0.091	0.098	0.099	0.095	0.092	0.095	0.094

Cleveland, OH	Cleveland-Elyria-Mentor, OH	0.092	0.093	0.098	0.1	0.099	0.098	0.099	0.095
Colorado Springs, CO	Colorado Springs, CO	0.062	0.061	0.061	0.059	0.056	0.059	0.062	0.065
Columbia, SC	Columbia, SC	0.085	0.087	0.086	0.081	0.08	0.087	0.092	0.096
Columbus, OH	Columbus, OH	0.09	0.086	0.09	0.092	0.092	0.093	0.097	0.095
Corpus Christi, TX	Corpus Christi, TX	0.078	0.079	0.082	0.083	0.083	0.08	0.081	0.083
Dallas/Ft Worth, TX	Dallas-Fort Worth-Arlington, TX	0.095	0.096	0.106	0.104	0.104	0.098	0.101	0.102
Dayton, OH	Dayton, OH	0.084	0.086	0.092	0.093	0.091	0.093	0.093	0.09
Denver, CO	Denver-Aurora-Broomfield, CO	0.071	0.074	0.081	0.081	0.079	0.084	0.083	0.086
Detroit, MI	Detroit-Warren-Livonia, MI	0.089	0.088	0.093	0.094	0.092	0.093	0.095	0.089
El Paso, TX	El Paso, TX	0.078	0.081	0.084	0.089	0.08	0.082	0.078	0.08
Evansville, IN	Evansville, IN-KY	0.087	0.089	0.094	0.095	0.093	0.093	0.094	0.091
Flint, MI	Flint, MI	0.077	0.071	0.075	0.082	0.084	0.086	0.089	0.086
Fresno, CA	Fresno, CA	0.111	0.107	0.108	0.107	0.111	0.115	0.113	0.111
Ft. Lauderdale, FL	Broward County, FL	0.076	0.079	0.074	0.069	0.069	0.072	0.075	0.075
Gary, IN	Lake County, IN	0.08	0.077	0.084	0.091	0.095	0.09	0.091	0.088
Greely, CO	Greeley, CO	0.068	0.066	0.068	0.071	0.07	0.071	0.071	0.071
Greensboro, NC	Greensboro-High Point, NC	0.083	0.084	0.088	0.086	0.085	0.089	0.092	0.094
Greenville, SC	Greenville-Mauldin-Easley, SC	0.082	0.081	0.082	0.081	0.083	0.087	0.09	0.09
Harrisburg, PA	Harrisburg-Carlisle, PA	0.091	0.089	0.092	0.087	0.088	0.088	0.094	0.093
Houston, TX	Houston-Sugar Land-Baytown, TX	0.104	0.11	0.114	0.116	0.117	0.116	0.118	0.112
Huntington, WV	Huntington-Ashland, WV-KY-OH	0.092	0.09	0.096	0.091	0.088	0.092	0.095	0.094
Indianapolis, IN	Indianapolis-Carmel, IN	0.087	0.09	0.094	0.098	0.097	0.098	0.097	0.095
City	Census Area Name	dv1991_ 1993	dv1992_ 1994	dv1993_ 1995	dv1994_ 1996	dv1995_ 1997	dv1996_ 1998	dv1997_ 1999	dv1998_ 2000
Jackson, MS	Jackson, MS	0.074	0.075	0.076	0.077	0.077	0.08	0.081	0.083
Jacksonville, FL	Jacksonville, FL	0.079	0.081	0.08	0.078	0.081	0.088	0.088	0.085
Jersey City, NJ	Hudson County, NJ	0.103	0.096	0.1	0.095	0.098	0.093	0.1	0.092
Johnstown, PA	Johnstown, PA	0.084	0.08	0.085	0.085	0.088	0.091	0.093	0.091
Kansas City, MO	Kansas City, MO-KS	0.082	0.082	0.09	0.092	0.094	0.093	0.091	0.089
Kenosha, WI	Kenosha County, WI	0.1	0.093	0.099	0.097	0.096	0.09	0.095	0.093

Knoxville, TN	Knoxville, TN	0.088	0.089	0.093	0.093	0.095	0.1	0.104	0.104
Lancaster, PA	Lancaster, PA	0.093	0.091	0.096	0.093	0.096	0.096	0.101	0.097
Lansing, MI	Lansing-East Lansing, MI	0.081	0.079	0.082	0.084	0.083	0.08	0.082	0.082
Las Vegas, NV	Las Vegas-Paradise, NV	0.075	0.079	0.079	0.08	0.079	0.08	0.077	0.085
Lexington, KY	Lexington-Fayette, KY	0.077	0.079	0.087	0.087	0.085	0.085	0.087	0.085
Little Rock, AR	Little Rock-North Little Rock-Conway, AR	0.078	0.077	0.08	0.08	0.081	0.08	0.082	0.087
Los Angeles, CA	Los Angeles-Long Beach-Santa Ana, CA	0.177	0.168	0.156	0.145	0.135	0.133	0.118	0.115
Madison, WI	Madison, WI	0.073	0.072	0.072	0.08	0.081	0.078	0.08	0.078
Memphis, TN	Memphis, TN-MS-AR	0.09	0.09	0.091	0.094	0.095	0.093	0.095	0.097
Milwaukee, WI	Milwaukee-Waukesha-West Allis, WI	0.09	0.084	0.092	0.097	0.098	0.093	0.097	0.092
Minneapolis, MN	Minneapolis-St. Paul-Bloomington, MN-WI	0.07	0.07	0.072	0.074	0.072	0.07	0.074	0.074
Nashville, TN	Nashville-Murfreesboro-Franklin, TN	0.095	0.096	0.099	0.099	0.099	0.101	0.102	0.1
Nassau, NY	Nassau County, NY								
New Haven, CT	New Haven-Milford, CT	0.108	0.097	0.105	0.101	0.107	0.1	0.103	0.096
New Orleans, LA	New Orleans-Metairie-Kenner, LA	0.081	0.086	0.084	0.085	0.083	0.084	0.086	0.091
New York City, NY	New York-Northern New Jersey-Long Island, NY-NJ-PA	0.108	0.1	0.106	0.104	0.108	0.104	0.107	0.107
Newark, NJ	Essex County, NJ	0.084	0.081	0.088	0.088	0.092	0.088	0.093	0
Norfolk, VA	Virginia Beach-Norfolk-Newport News, VA-NC	0.09	0.088	0.087	0.083	0.087	0.09	0.094	0.089
Oklahoma City, OK	Oklahoma City, OK	0.081	0.081	0.084	0.085	0.083	0.085	0.086	0.084
Orlando, FL	Orlando-Kissimmee, FL	0.078	0.082	0.079	0.079	0.078	0.084	0.085	0.085
Philadelphia, PA	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	0.106	0.099	0.104	0.101	0.11	0.107	0.11	0.106
Phoenix, AZ	Phoenix-Mesa-Scottsdale, AZ	0.088	0.086	0.089	0.09	0.092	0.091	0.088	0.088
City	Census Area Name	dv1991_1993	dv1992_1994	dv1993_1995	dv1994_1996	dv1995_1997	dv1996_1998	dv1997_1999	dv1998_2000
Pittsburgh, PA	Pittsburgh, PA	0.095	0.096	0.105	0.103	0.105	0.099	0.101	0.096
Portland, ME	Portland-South Portland-Biddeford, ME	0.102	0.095	0.096	0.092	0.096	0.092	0.092	0.084
Portland, OR	Portland-Vancouver-Beaverton, OR-WA	0.076	0.078	0.071	0.083	0.078	0.08	0.071	0.072
Portsmouth, NH	Rockingham County, NH	0.096	0.093	0.096	0.094	0.095	0.091	0.09	0.08
Providence, RI	Providence-New Bedford-Fall River, RI-MA	0.099	0.092	0.097	0.094	0.097	0.09	0.092	0.088

Racine, WI	Racine, WI	0.086	0.082	0.088	0.089	0.092	0.088	0.091	0.085
Raleigh, NC	Raleigh-Cary, NC	0.087	0.086	0.087	0.087	0.089	0.096	0.103	0.101
Reading, PA	Reading, PA	0.094	0.086	0.088	0.089	0.092	0.091	0.096	0.092
Richmond, VA	Richmond, VA	0.091	0.092	0.093	0.087	0.09	0.092	0.099	0.091
Riverside, CA	Riverside-San Bernardino-Ontario, CA	0.177	0.171	0.165	0.161	0.148	0.154	0.147	0.146
Roanoke, VA	Roanoke, VA	0.077	0.08	0.082	0.078	0.078	0.085	0.09	0.089
Rochester, NY	Rochester, NY	0.088	0.08	0.085	0.081	0.083	0.08	0.086	0.081
Sacramento, CA	Sacramento-Arden Arcade-Roseville, CA	0.11	0.104	0.106	0.106	0.099	0.103	0.103	0.107
Salinas, CA	Salinas, CA	0.069	0.07	0.069	0.067	0.065	0.066	0.062	0.064
San Antonio, TX	San Antonio, TX	0.079	0.082	0.087	0.087	0.087	0.085	0.088	0.086
San Diego, CA	San Diego-Carlsbad-San Marcos, CA	0.112	0.109	0.108	0.104	0.099	0.102	0.099	0.1
San Francisco, CA	San Francisco-Oakland-Fremont, CA	0.081	0.082	0.087	0.093	0.09	0.089	0.086	0.087
San Jose, CA	San Jose-Sunnyvale-Santa Clara, CA	0.08	0.08	0.083	0.088	0.085	0.086	0.082	0.082
Seattle, WA	Seattle-Tacoma-Bellevue, WA	0.077	0.074	0.071	0.076	0.078	0.081	0.074	0.075
Shreveport, LA	Shreveport-Bossier City, LA	0.085	0.086	0.083	0.08	0.082	0.084	0.089	0.092
South Bend, IN	South Bend-Mishawaka, IN-MI	0.089	0.087	0.089	0.094	0.094	0.092	0.092	0.088
Springfield, MA	Springfield, MA	0.1	0.095	0.094	0.092	0.097	0.096	0.099	0.089
St Louis, MO	St. Louis, MO-IL	0.091	0.091	0.098	0.104	0.1	0.095	0.095	0.094
Steubenville, OH	Weirton-Steubenville, WV-OH	0.085	0.08	0.087	0.086	0.085	0.084	0.087	0.083
Syracuse, NY	Syracuse, NY	0.087	0.081	0.082	0.079	0.079	0.077	0.082	0.08
Tacoma, WA	Seattle-Tacoma-Bellevue, WA	0.077	0.074	0.071	0.076	0.078	0.081	0.074	0.075
Tampa, FL	Tampa-St. Petersburg-Clearwater, FL	0.08	0.08	0.08	0.081	0.082	0.088	0.09	0.088
Toledo, OH	Toledo, OH	0.085	0.086	0.09	0.091	0.089	0.089	0.086	0.084
Trenton, NJ	Trenton-Ewing, NJ	0.111	0.105	0.104	0.1	0.101	0.097	0.104	0.102
City	Census Area Name	dv1991_1993	dv1992_1994	dv1993_1995	dv1994_1996	dv1995_1997	dv1996_1998	dv1997_1999	dv1998_2000
Tucson, AZ	Tucson, AZ	0.077	0.078	0.081	0.079	0.079	0.077	0.075	0.073
Vallejo, CA	Vallejo-Fairfield, CA	0.074	0.073	0.077	0.079	0.078	0.082	0.085	0.085
Ventura, CA	Ventura County, CA	0.115	0.112	0.117	0.119	0.115	0.112	0.106	0.105
Washington, DC	Washington-Arlington-Alexandria, DC-VA-MD-WV	0.101	0.096	0.098	0.094	0.1	0.101	0.106	0.101

Wichita, KS	Wichita, KS	0.068	0.065	0.07	0.072	0.074	0.078	0.08	0.08
Wilmington, DE	New Castle County, DE	0.098	0.099	0.103	0.098	0.099	0.095	0.1	0.097
Worcester, MA	Worcester, MA		0.095	0.095	0.089	0.087	0.087	0.094	0.088
York, PA	York-Hanover, PA	0.091	0.085	0.086	0.083	0.087	0.09	0.094	0.093
Youngstown, OH	Youngstown-Warren-Boardman, OH-PA	0.091	0.089	0.091	0.092	0.093	0.096	0.096	0.092
Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.									

1

2 **Jerrett et al., 2013** (2094363) - Long-Term Ozone and Respiratory Mortality

3

California, U.S. O₃: 1988-2002

State	dv.1988 .1990	dv.1989 .1991	dv.1990 .1992	dv.1991 .1993	dv.1992 .1994	dv.1993 .1995	dv.1994 .1996	dv.1995 .1997	dv.1996 .1998	dv.1997 .1999	dv.1998 .2000	dv.1999 .2001	dv.2000 .2002
California	186	182	180	177	171	165	161	148	154	147	146	129	128

4

5 **Katsouyanni et al., 2009** (199899) - Short-Term Ozone and Respiratory Mortality

6

Nationwide, U.S. O₃: 1987-1996

City	Census Area Name	dv1987_ 1989	dv1988_ 1990	dv1989_ 1991	dv1990_ 1992	dv1991_ 1993	dv1992_ 1994	dv1993_ 1995	dv1994_ 1996
Honolulu, HI	Honolulu, HI	0.020	0.018						
Lincoln, NE	Lincoln, NE	0.058	0.061	0.058	0.061	0.058	0.059	0.057	0.058
Colorado Springs,	Colorado Springs, CO	0.063		0.066	0.063	0.062	0.061	0.061	0.056
Des Moines, IA	Des Moines-West Des Moines, IA								0.062
Spokane, WA	Spokane, WA							0.064	0.066
Omaha, NE	Omaha-Council Bluffs, NE-IA	0.077	0.078	0.072	0.071	0.065	0.062	0.062	0.067
Albuquerque, NM	Albuquerque, NM	0.073	0.073	0.071	0.071	0.069	0.070	0.071	0.074
Wichita, KS	Wichita, KS	0.080	0.081	0.075	0.073	0.067	0.065	0.065	0.072
Mobile, AL	Mobile, AL	0.078	0.080	0.062	0.064	0.070	0.074	0.075	0.077
Minneapolis, MN	Minneapolis-St. Paul-Bloomington, MN-	0.080	0.079	0.068	0.070	0.069	0.070	0.072	0.074
Tucson, AZ	Tucson, AZ	0.068	0.074	0.069	0.072	0.077	0.078	0.081	0.079
Jackson, MS	Jackson, MS	0.075	0.079	0.076	0.076	0.074	0.075	0.076	0.077
Seattle, WA	Seattle-Tacoma-Bellevue, WA	0.076	0.079	0.078	0.086	0.077	0.074	0.071	0.076

City	Census Area Name	dv1987_ 1989	dv1988_ 1990	dv1989_ 1991	dv1990_ 1992	dv1991_ 1993	dv1992_ 1994	dv1993_ 1995	dv1994_ 1996
Tacoma, WA	Seattle-Tacoma-Bellevue, WA	0.076	0.079	0.078	0.086	0.077	0.074	0.071	0.076
Miami, FL	Miami-Fort Lauderdale-Pompano Beach,	0.083	0.079	0.075	0.073	0.076	0.080	0.080	0.074
Las Vegas, NV	Las Vegas-Paradise, NV	0.081	0.078	0.078	0.076	0.075	0.079	0.079	0.080
Madison, WI	Madison, WI	0.091	0.079	0.081	0.079	0.073	0.072	0.072	0.080
Portland, OR	Portland-Vancouver-Beaverton, OR-WA	0.077	0.085	0.082	0.091	0.076	0.078	0.058	0.083
Denver, CO	Denver-Aurora-Broomfield, CO	0.087	0.086	0.080	0.074	0.071	0.074	0.081	0.081
Little Rock, AR	Little Rock-North Little Rock-Conway, AR	0.085	0.082	0.079	0.080	0.078	0.077	0.080	0.080
Orlando, FL	Orlando-Kissimmee, FL	0.082	0.082	0.080	0.079	0.078	0.082	0.079	0.079
Salt Lake City, UT	Salt Lake City, UT	0.085	0.082	0.078	0.075	0.076	0.079	0.082	0.089
Jacksonville, FL	Jacksonville, FL	0.086	0.084	0.081	0.079	0.079	0.081	0.080	0.078
Corpus Christi, TX	Corpus Christi, TX	0.089	0.085	0.079	0.077	0.078	0.079	0.082	0.083
St. Petersburg, FL	Tampa-St. Petersburg-Clearwater, FL	0.086	0.085	0.079	0.081	0.080	0.080	0.080	0.081
Tampa, FL	Tampa-St. Petersburg-Clearwater, FL	0.086	0.085	0.079	0.081	0.080	0.080	0.080	0.081
Huntsville, AL	Huntsville, AL	0.087	0.083	0.077	0.082	0.085	0.083	0.080	0.078
El Paso, TX	El Paso, TX	0.088	0.083	0.080	0.079	0.078	0.081	0.084	0.089
San Antonio, TX	San Antonio, TX	0.085	0.085	0.082	0.079	0.079	0.082	0.087	0.087
New Orleans, LA	New Orleans-Metairie-Kenner, LA	0.090	0.085	0.077	0.080	0.081	0.086	0.084	0.085
Austin, TX	Austin-Round Rock, TX	0.084	0.086	0.084	0.084	0.081	0.082	0.084	0.084
Oklahoma City, OK	Oklahoma City, OK	0.087	0.087	0.086	0.084	0.081	0.081	0.084	0.085
Syracuse, NY	Syracuse, NY	0.092	0.088	0.083	0.083	0.087	0.081	0.082	0.079
Shreveport, LA	Shreveport-Bossier City, LA	0.087	0.088	0.084	0.086	0.085	0.086	0.083	0.080
San Jose, CA	San Jose-Sunnyvale-Santa Clara, CA	0.097	0.088	0.082	0.083	0.080	0.080	0.083	0.088
Kansas City, MO	Kansas City, MO-KS	0.088	0.086	0.082	0.083	0.082	0.082	0.090	0.092
Oakland, CA	San Francisco-Oakland-Fremont, CA	0.089	0.087	0.084	0.082	0.081	0.082	0.087	0.093
San Francisco, CA	San Francisco-Oakland-Fremont, CA	0.089	0.087	0.084	0.082	0.081	0.082	0.087	0.093
Phoenix, AZ	Phoenix-Mesa-Scottsdale, AZ	0.077	0.082	0.083	0.091	0.088	0.086	0.089	0.090
Lexington, KY	Lexington-Fayette, KY	0.099	0.096	0.085	0.078	0.077	0.079	0.087	0.087
Tulsa, OK	Tulsa, OK	0.089	0.090	0.087	0.087	0.082	0.083	0.088	0.091
Stockton, CA	Stockton, CA	0.093	0.090	0.087	0.088	0.088	0.087	0.086	0.085
Rochester, NY	Rochester, NY	0.099	0.095	0.092	0.090	0.088	0.080	0.085	0.081

City	Census Area Name	dv1987_ 1989	dv1988_ 1990	dv1989_ 1991	dv1990_ 1992	dv1991_ 1993	dv1992_ 1994	dv1993_ 1995	dv1994_ 1996
Dayton, OH	Dayton, OH	0.096	0.092	0.086	0.082	0.084	0.086	0.092	0.093
Greensboro, NC	Greensboro-High Point, NC	0.097	0.100	0.088	0.085	0.083	0.084	0.088	0.086
Ft. Wayne, IN	Fort Wayne, IN	0.094	0.092	0.087	0.085	0.085	0.088	0.089	0.093
Buffalo, NY	Buffalo-Niagara Falls, NY	0.100	0.095	0.089	0.088	0.086	0.083	0.087	0.086
Raleigh, NC	Raleigh-Cary, NC	0.099	0.093	0.089	0.086	0.087	0.086	0.087	0.087
Newark, NJ	Essex County, NJ	0.105	0.098	0.088	0.086	0.084	0.081	0.088	0.088
Toledo, OH	Toledo, OH	0.102	0.099	0.086	0.082	0.085	0.086	0.090	0.091
Knoxville, TN	Knoxville, TN	0.093	0.094	0.086	0.089	0.088	0.089	0.093	0.093
Columbus, OH	Columbus, OH	0.097	0.095	0.089	0.092	0.090	0.086	0.090	0.092
Birmingham, AL	Birmingham-Hoover, AL	0.094	0.093	0.084	0.088	0.089	0.092	0.096	0.096
Worcester, MA	Worcester, MA	0.091	0.091	0.089	0.091		0.095	0.095	0.089
Memphis, TN	Memphis, TN-MS-AR	0.095	0.095	0.089	0.091	0.090	0.090	0.091	0.094
Grand Rapids, MI	Grand Rapids-Wyoming, MI	0.105	0.103	0.096	0.090	0.085	0.081	0.086	0.089
Indianapolis, IN	Indianapolis-Carmel, IN	0.098	0.095	0.091	0.089	0.087	0.090	0.094	0.098
Madera, CA	Madera-Chowchilla, CA				0.091	0.096	0.091	0.093	0.093
Detroit, MI	Detroit-Warren-Livonia, MI	0.099	0.099	0.096	0.091	0.089	0.088	0.093	0.094
Baton Rouge, LA	Baton Rouge, LA	0.098	0.101	0.099	0.096	0.090	0.087	0.091	0.094
Modesto, CA	Modesto, CA	0.102	0.099	0.095	0.092	0.086	0.093	0.095	0.096
Charlotte, NC	Charlotte-Gastonia-Concord, NC-SC	0.104	0.101	0.092	0.091	0.091	0.092	0.094	0.094
Louisville, KY	Louisville/Jefferson County, KY-IN	0.098	0.099	0.096	0.092	0.094	0.094	0.100	0.094
Akron, OH	Akron, OH	0.112	0.109	0.099	0.093	0.094	0.088	0.090	0.089
Boston, MA	Boston-Cambridge-Quincy, MA-NH	0.105	0.101	0.098	0.092	0.096	0.093	0.096	0.094
Cleveland, OH	Cleveland-Elyria-Mentor, OH	0.105	0.104	0.093	0.090	0.092	0.093	0.098	0.100
Milwaukee, WI	Milwaukee-Waukesha-West Allis, WI	0.117	0.105	0.101	0.095	0.090	0.084	0.092	0.097
Pittsburgh, PA	Pittsburgh, PA	0.107	0.098	0.092	0.088	0.095	0.096	0.105	0.103
Cincinnati, OH	Cincinnati-Middletown, OH-KY-IN	0.106	0.107	0.102	0.095	0.091	0.091	0.098	0.099
Nashville, TN	Nashville-Murfreesboro-Franklin, TN	0.104	0.104	0.096	0.096	0.095	0.096	0.099	0.099
St Louis, MO	St. Louis, MO-IL	0.111	0.102	0.098	0.098	0.091	0.091	0.098	0.104
Dallas/Ft Worth, TX	Dallas-Fort Worth-Arlington, TX	0.100	0.105	0.105	0.099	0.095	0.096	0.106	0.104
Providence, RI	Providence-New Bedford-Fall River, RI-	0.108	0.108	0.107	0.105	0.099	0.092	0.097	0.094

City	Census Area Name	dv1987_ 1989	dv1988_ 1990	dv1989_ 1991	dv1990_ 1992	dv1991_ 1993	dv1992_ 1994	dv1993_ 1995	dv1994_ 1996
Washington, DC	Washington-Arlington-Alexandria, DC-VA-MD-WV	0.115	0.107	0.100	0.100	0.101	0.096	0.098	0.094
Chicago, IL	Chicago-Naperville-Joliet, IL-IN-WI	0.114	0.114	0.104	0.099	0.100	0.093	0.099	0.097
Jersey City, NJ	Hudson County, NJ	0.118	0.115	0.107	0.104	0.103	0.096	0.100	0.095
Atlanta, GA	Atlanta-Sandy Springs-Marietta, GA	0.113	0.107	0.104	0.105	0.101	0.101	0.109	0.105
Sacramento, CA	Sacramento-Arden Arcade-Roseville, CA	0.114	0.107	0.105	0.105	0.110	0.104	0.106	0.106
Baltimore, MD	Baltimore-Towson, MD	0.125	0.115	0.104	0.106	0.107	0.103	0.107	0.105
Philadelphia, PA	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	0.123	0.120	0.113	0.107	0.106	0.099	0.104	0.101
Fresno, CA	Fresno, CA	0.115	0.110	0.108	0.108	0.111	0.107	0.108	0.107
New York City, NY	New York-Northern New Jersey-Long	0.129	0.128	0.122	0.116	0.108	0.100	0.106	0.104
Houston, TX	Houston-Sugar Land-Baytown, TX	0.117	0.119	0.119	0.116	0.104	0.110	0.114	0.116
Bakersfield, CA	Bakersfield, CA	0.116	0.112	0.118	0.115	0.112	0.111	0.119	0.119
San Diego, CA	San Diego-Carlsbad-San Marcos, CA	0.125	0.129	0.125	0.118	0.112	0.109	0.108	0.104
Anaheim, CA	Orange County, CA	0.141	0.138	0.127	0.120	0.114	0.117	0.107	0.100
Los Angeles, CA	Los Angeles-Long Beach-Santa Ana, CA	0.192	0.186	0.179	0.177	0.177	0.168	0.156	0.145
Riverside, CA	Riverside-San Bernardino-Ontario, CA	0.188	0.185	0.182	0.180	0.177	0.171	0.165	0.161
San Bernardino, CA	San Bernardino County, CA	0.188	0.185	0.182	0.180	0.177	0.171	0.165	0.161
Anchorage, AK	Anchorage, AK								

Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.

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2 **Klemm et al., 2011** (1011160) - Short-Term Ozone and Respiratory Mortality
3 Atlanta (Fulton, DeKalb, Gwinnet & Cobb counties), GA, U.S. O₃: 8/1998 - 12/2007

City	Census Area Name	dv.1998. 2000	dv.1999. 2001	dv.2000. 2002	dv.2001. 2003	dv.2002. 2004	dv.2003. 2005	dv.2004.2 006	dv.2005. 2007
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	121	107	99	91	93	90	91	95

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5

1 **Kousha and Rowe, 2014** (2443421) - ED Visit - Respiratory Infection

2 Edmonton, Canada. O₃: 1992-2002

City	dv.1992. 1994	dv.1993. 1995	dv.1994. 1996	dv.1995. 1997	dv.1996. 1998	dv.1997. 1999	dv.1998. 2000	dv.1999. 2001	dv.2000. 2002
Edmonton	60	61	58	56	62	64	64	64	65

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4 **Kousha and Castner, 2016** (3160295) - ED Visit - Respiratory Infection

5 Windsor, Canada. O₃: 2004-2010

City	dv.2004.2006	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010
Windsor	80	87	84	80	73

6
7 **Malig et al., 2016** (3285875) - ED Visits for Asthma, ED Visits Aggregate Respiratory Diseases, ED Visit for Respiratory Infection
8 California (statewide), U.S. O₃: 2005-2008

State	dv.2005.2007	dv.2006.2008
California	122	119

9
10 **Nishimura et al., 2013** (1632336)
11 Four U.S. cities (Chicago, Houston, New York, San Francisco) and Puerto Rico.

12 This is a case control study with study participants, aged 8-21 years, identified during 2006-2011. Associations examined for annual
13 average O₃ concentration (1-h max; 8-h max, per ISA), averaged across first three years of life. Median birth year was 1996.

14
15 **O'Lenick et al., 2017** (3421578) - ED Visits for Asthma

16 20-county Atlanta metro area, GA, U.S. O₃: 2002-2008

City	Census Area Name	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008
Atlanta, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	93	90	91	95	95

17 **O'Lenick et al., 2017** (3859553) - ED Visits Aggregate Respiratory Diseases
18 20-co Atlanta, GA; 12-co Dallas, TX, and 16-co St. Louis, MO, U.S. O₃: 2002-2008

City	Census Area Name	dv.2002. 2004	dv.2003. 2005	dv.2004. 2006	dv.2005. 2007	dv.2006. 2008
Atlanta, GA	Atlanta--Athens-Clarke County--Sandy Springs, GA	93	90	91	95	95

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City	Census Area Name	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008
Dallas, TX	Dallas-Fort Worth, TX-OK	98	95	96	95	91

City	Census Area Name	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008
St. Louis, MO	St. Louis-St. Charles-Farmington, MO-IL	89	86	86	89	85

Rodopoulou et al., 2015 (2965674) - ED Visit for Respiratory Infection

Little Rock, AK, U.S. O₃: 2002-2012

City	Census Area Name	dv.2002 .2004	dv.2003 .2005	dv.2004 .2006	dv.2005 .2007	dv.2006 .2008	dv.2007 .2009	dv.2008 .2010	dv.2009 .2011	dv.2010 .2012
Little Rock, AK	Little Rock-North Little Rock, AR	78	77	80	83	80	73	70	74	77

Sacks et al., 2014 (2228782) - ED Visits for Asthma

North Carolina (Statewide), U.S. O₃: 2006-2008

State	dv.2006.2008
North Carolina	94

Sarnat et al., 2013 (1640373) - ED Visits for Asthma

Metro Atlanta area (186 zip codes), GA, U.S. O₃: 1999-2002

City	Census Area Name	dv.1999.2001	dv.2000.2002
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	107	99

1 **Sarnat et al., 2015** (2772940) - ED Visits for Asthma

2 St. Louis metro area, MO (8 MO counties, 8 IL counties), U.S. O₃: 2001-2003

City	Census Area Name	dv.2001.2003	dv.2002.2004
St. Louis	St. Louis-St. Charles-Farmington, MO-IL	92	92

3

4 **Sheffield et al., 2015** (3025138) - ED Visits for Asthma

5 New York City (all boroughs), NY, U.S. O₃: May-Sept. 2005-2011

City	Census Area Name	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010	dv.2009.2011	dv.2010.2012
New York, NY	New York-Newark, NY-NJ-CT-PA	94	89	84	82	84	85

6

7 **Shmool et al., 2016** (3288326) - ED Visits for Asthma

8 New York City, NY, U.S. O₃: June-Aug 2005-2011

City	Census Area Name	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010	dv.2009.2011
New York, NY	New York-Newark, NY-NJ-CT-PA	94	89	84	82	84

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10 **Silverman and Ito, 2010** (386252) HA for Asthma

11 New York, NY. O₃: 1999-2006

City	Census Area Name	dv.1999.2001	dv.2000.2002	dv.2001.2003	dv.2002.2004	dv.2003.2005	dv.2004.2006
New York City, NY	New York-Northern New Jersey-Long Island, NY-NJ-PA	0.109	0.115	0.109	0.102	0.094	0.093

Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.

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13 **Stieb et al., 2009** (195858) - ED Visits for Asthma

14 7 Canadian cities

O ₃ : 1992-2003City	dv1992_1994	dv1993_1995	dv1994_1996	dv1995_1997	dv1996_1998	dv1997_1999	dv1998_2000	dv1999_2001	dv2000_2002	dv2001_2003
Montreal						77	73	73	72	
Ottawa	64	64	63	66	65	69	63			
Edmonton	60	61	58	56	62	64	64	64	65	

Saint John	51	54	58						
Halifax									54
Toronto							79	81	85
Vancouver							52	54	57

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2 **Strickland et al., 2014** (2519636) - ED Visits for Asthma

3 20-county Atlanta area, GA, U.S. O₃: 2002-2010

City	Census Area Name	dv.2002. 2004	dv.2003. 2005	dv.2004. 2006	dv.2005. 2007	dv.2006. 2008	dv.2007. 2009	dv.2008. 2010
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	93	90	91	95	95	87	80

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5 **Szyszkowicz et al., 2018** (4245266) - ED Visits for Asthma, [ED Visit - Respiratory Infection]

6 Multicity (9), Canada. O₃: 2004-2011

City	dv.2004.2006	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010	dv.2009.2011
Algoma	67	70	68	65	62	59
Oakville	73	78	75	73	70	69
Burlington	70	74	73	70	68	66
Hamilton	73	75	73	72	70	69
London	69	72	71	68	65	64
Parkhill						
Longwoods						
Ottawa	64	69	66	64	62	57
Brampton	74	78	75	73	68	67
Mississauga	-	79	-	-	65	64
Toronto	74	79	76	74	73	70
Essex	-	79	74			
New Market	77	79	75	75	70	69
Stouffville						

Note: Some of the locations named as city in the study appear as Municipality in NAPS dataset from Canada and included few other cities within its boundary. In such instances, DV data (if available) were pulled for all the cities included within those municipalities, e.g., Halton included (Oakville, Burlington), Middlesex included (London, Parkhill, Longwoods), Peel included (Toronto, Brampton, Mississauga), York included (New Market, Stouffville).

1 **Tolbert et al., 2007** (90316) - ED Visits for Aggregate Respiratory Diseases

2 Atlanta, GA. O₃: 1993-2004

City	Census Area Name	dv1993_1995	dv1994_1996	dv1995_1997	dv1996_1998	dv1997_1999	dv1998_2000	dv1999_2001	dv2000_2002	dv2001_2003	dv2002_2004
Atlanta, GA	Atlanta-Sandy Springs-Marietta, GA	0.109	0.105	0.110	0.113	0.118	0.121	0.107	0.099	0.091	0.093

Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.

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4 **Tétreault et al., 2016** (3073711) – Asthma Incidence

5 Quebec Province, Canada. O₃: 1996-2011

City	dv.1996.1998	dv.1997.1999	dv.1998.2000	dv.1999.2001	dv.2000.2002	dv.2001.2003	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010	dv.2009.2011
Montreal	69	77	72	72	72	79	72	70	66	70	67	65	61	58
Quebec	61	65	59	60	63	70	64	59	56	63	60	58	55	54
Laval	72	75	67	68	68	75	68	67	62	65	62	61	59	60
Brossard	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Longueuil	70	76	70	70	68	74	71	68	64	65	62	61	60	60
Terrebonne	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Gatineau	-	75	72	73	69	71	67	66	66	-	-	-	59	55
Levis	-	-	-	-	-	-	-	-	-	-	59	57	52	50
Sherbrooke	-	-	-	-	-	-	-	-	63	63	60	59	57	56
Saguenay	-	-	-	-	-	-	-	54	54	57	56	54	52	51
Rouyn-Noranda	-	-	-	-	-	-	-	-	59	63	59	58	55	54
Trois-Rivieres	-	-	-	68	65	70	64	64	59	64	59	58	55	-
St. Zephirin-de-Courval (MUNI)	72	75	67	71	73	79	73	70	66	69	65	62	60	60
Forestville	55	-	-	-	-	-	-	-	-	-	-	-	-	-
Charette (MUNI)	70	71	62	65	64	68	63	61	58	62	61	61	58	55
Saint-Remi	67	-	-	-	-	-	-	-	-	-	-	-	-	-
Saint-Simon (MUNI)	66	71	65	65	64	70	66	62	58	59	58	56	55	55
Saint-Faustin-Lac-Carre (MUNI)	67	71	66	69	65	68	66	69	67	68	67	65	61	56
La Peche (MUNI)	-	71	72	74	72	73	68	66	64	67	66	63	-	54
Varennes	74	75	68	69	69	75	68	67	63	65	60	58	55	56
Temiscaming (MUNI)	-	-	-	-	-	-	-	-	-	-	63	60	58	57
Auclair (MUNI)	-	-	-	-	-	-	-	-	-	-	60	57	55	53
Causapscal	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Riviere-Eternite (MUNI)	-	-	-	-	-	-	-	-	-	-	-	-	-	-

City	dv.1996. 1998	dv.1997. 1999	dv.1998. 2000	dv.1999. 2001	dv.2000. 2002	dv.2001. 2003	dv.2002. 2004	dv.2003. 2005	dv.2004. 2006	dv.2005. 2007	dv.2006. 2008	dv.2007. 2009	dv.2008. 2010	dv.2009. 2011
La Dore (MUNI)	54	58	58	62	58	62	56	57	55	61	58	57	53	52
Deschambault (MUNI)	68	70	64	67	68	72	65	61	57	61	58	57	56	55
Saint-François	65	69	65	64	65	69	64	62	59	64	60	58	57	55
Notre-Dame-du-Rosaire (MUNI)	65	66	60	62	64	67	62	60	59	60	59	57	55	53
St-Hilaire-de-Dorset (MUNI)	67	70	66	67	69	73	71	67	65	65	65	63	59	57
Tingwick (MUNI)	69	73	66	66	66	72	70	67	62	63	-	60	61	57
Lac-Edouard (MUNI)	-	-	62	65	60	62	58	57	55	59	58	58	54	51
Montmorency (COUNTY)	-	-	-	-	-	-	-	-	-	-	-	-	-	-
Sutton	-	-	-	-	-	-	-	-	-	-	70	68	65	61
Chapais	-	-	-	-	-	-	-	-	-	59	56	56	56	55
Ste-Francoise (MUNI)	72	76	78	-	-	-	-	-	-	-	-	-	-	-
Saint-Anicet (MUNI)	74	79	76	75	74	79	75	73	69	70	68	-	67	63
L'Assomption (COUNTY)	-	76	70	70	67	71	60	60	58	67	64	-	64	59
La Patrie (MUNI)	-	68	66	67	71	73	72	68	65	63	63	62	59	55
Ferme Neuve (MUNI)	58	60	58	59	56	59	54	-	-	-	59	57	53	50
Senneterre	-	-	-	-	-	-	-	-	-	-	59	57	55	54
Lemieux (MUNI)	-	-	-	-	-	64	66	63	65	65	64	-	63	59
Saint-Jean-sur-Richelieu	-	-	-	-	68	73	67	65	61	64	62	59	57	56
Frelighsburg (MUNI)	-	-	-	-	-	-	-	-	-	68	68	66	63	59
Mingan (First Nations Reserce)	-	-	-	-	-	-	-	-	-	-	52	51	49	47

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Turner et al., 2016 (3060878) - Long-Term Ozone and Respiratory Mortality

Nationwide, U.S. O₃: 2002-2004

Air quality data are not described for this study as it relied on estimated O₃ concentrations for the years 2002–2004 as surrogates for study population O₃ concentrations during the 1982 to 2004 period (Turner et al., 2016).

1 **Vanos et al., 2014** (2231512) - Short-Term Ozone and Respiratory Mortality

2 10 Canadian cities, Canada. O₃: 1981 - 1999. The table below does not include design values prior to 1988 as data are not readily
 3 available for years prior to 1986.

City	dv.1986. 1988	dv.1987. 1989	dv.1988. 1990	dv.1989. 1991	dv.1990. 1992	dv.1991. 1993	dv.1992. 1994	dv.1993. 1995	dv.1994. 1996	dv.1995. 1997	dv.1996. 1998	dv.1997. 1999
Saint John		65	67	68	66	61	51	54	58	60	55	55
Toronto	90	89	85	81	78	75	70	72	73	77	80	84
Montreal	66	74	77	72	73	73	69	65	63	71	68	77
Ottawa	67	68	73	71	71	69	64	64	63	66	65	69
Windsor	94	94	91	82	79	79	78	85	90	86	86	86
Quebec						60	62.5	59	57.5			
Calgary	64	63	60	60	60	59	60	59	60	57	59	58
Edmonton	62	60	57	60	62	62	60	61	58	56	62	64
Winnipeg	62	64	63	58	53	53	54	54	54	56	56	62
Vancouver	73	70	74	61	60	55	55	65	63	59	61	58

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 5 **Villeneuve et al., 2007** (195859) - ED Visits for Asthma

6 Census Metropolitan of Edmonton, Alberta, Canada. 1992-2002

City	dv.1992. 1994	dv.1993. 1995	dv.1994. 1996	dv.1995. 1997	dv.1996. 1998	dv.1997. 1999	dv.1998. 2000	dv.1999. 2001	dv.2000. 2002
Census Metropolitan of Edmonton	60	67	69	63	61	64	64	63	64

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 8 **Weichenthal et al., 2017** (4165121) - Long-Term Ozone and Respiratory Mortality

9 Nationwide, Canada. O₃: 2002-2009

City	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008	dv.2007.2009
All cities (DV range)	45-98	43-93	42-85	36-89	35-86	37-83

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1 **Winqvist et al., 2012** (1668375) - Hospital Admissions for Asthma, ED Visits for Asthma, Hospital Admissions for Aggregate
 2 Respiratory, ED Visits for Aggregate Respiratory Diseases, ED Visits for Respiratory Infection
 3 St. Louis, MO (8 MO and 8 IL counties, 269 zip codes), U.S. O₃: 2001-2007

City	Census Area Name	dv.2001.2003	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007
St. Louis	St. Louis-St. Charles-Farmington, MO-IL	92	89	86	86	89

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 5 **Winqvist et al., 2014** (2347402) - ED Visits for Asthma
 6 Atlanta metro area, GA, U.S. O₃: 1998-2004

City	Census Area Name	dv.1998.2000	dv.1999.2001	dv.2000.2002	dv.2001.2003	dv.2002.2004
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	121	107	99	91	93

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 8 **Xiao et al., 2016** (3455927) - ED Visits for Asthma, ED Visit - Respiratory Infection
 9 Georgia (statewide), U.S. O₃: 2002-2008

State	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008
Georgia	93	93	91	95	95

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 11 **Zanobetti and Schwartz, 2008** (101596) - Short-Term Ozone and Respiratory Mortality
 12 48 U.S. cities

City	Census Area Name	dv1989 _1991	dv1990 _1992	dv1991 _1993	dv1992 _1994	dv1993 _1995	dv1994 _1996	dv1995 _1997	dv1996 _1998	dv1997 _1999	dv1998 _2000
Honolulu, HI	Honolulu, HI								0.045	0.048	0.047
Colorado Springs, CO	Colorado Springs, CO	0.066	0.063	0.062	0.061	0.061	0.056			0.062	0.065
Spokane, WA	Spokane, WA					0.064	0.066	0.066	0.068	0.067	0.067
Albuquerque, NM	Albuquerque, NM	0.071	0.071	0.069	0.070	0.071	0.074	0.069	0.073	0.071	0.075
Ft. Lauderdale, FL	Broward County, FL	0.075	0.073	0.076	0.079	0.074	0.069	0.069	0.072	0.075	0.075
Boulder, CO	Boulder, CO	0.076	0.073	0.073	0.071	0.072	0.071	0.071	0.078	0.078	0.078
Provo/Orem, UT	Provo-Orem, UT				0.069	0.068	0.071	0.076	0.082	0.082	0.086

City	Census Area Name	dv1989 _1991	dv1990 _1992	dv1991 _1993	dv1992 _1994	dv1993 _1995	dv1994 _1996	dv1995 _1997	dv1996 _1998	dv1997 _1999	dv1998 _2000
Miami, FL	Miami-Fort Lauderdale-Pompano Beach, FL	0.075	0.073	0.076	0.080	0.080	0.074	0.075	0.077	0.078	0.079
Seattle, WA	Seattle-Tacoma-Bellevue, WA	0.078	0.086	0.077	0.074	0.071	0.076	0.078	0.081	0.074	0.075
Denver, CO	Denver-Aurora-Broomfield, CO	0.080	0.074	0.071	0.074	0.081	0.081	0.079	0.084	0.083	0.086
Orlando, FL	Orlando-Kissimmee, FL	0.080	0.079	0.078	0.082	0.079	0.079	0.078	0.084	0.085	0.085
Salt Lake City, UT	Salt Lake City, UT	0.078	0.075	0.076	0.079	0.082	0.089	0.085	0.088	0.082	0.088
Tampa, FL	Tampa-St. Petersburg-Clearwater, FL	0.079	0.081	0.080	0.080	0.080	0.081	0.082	0.088	0.090	0.088
New Orleans, LA	New Orleans-Metairie-Kenner, LA	0.077	0.080	0.081	0.086	0.084	0.085	0.083	0.084	0.086	0.091
Oklahoma City,	Oklahoma City, OK	0.086	0.084	0.081	0.081	0.084	0.085	0.083	0.085	0.086	0.084
Terra Haute, IN	Terre Haute, IN	0.087	0.081	0.077	0.079	0.084	0.092	0.088	0.088	0.083	0.080
Austin, TX	Austin-Round Rock, TX	0.084	0.084	0.081	0.082	0.084	0.084	0.081	0.081	0.089	0.089
San Francisco, CA	San Francisco-Oakland-Fremont, CA	0.084	0.082	0.081	0.082	0.087	0.093	0.090	0.089	0.086	0.087
Greensboro, NC	Greensboro-High Point, NC	0.088	0.085	0.083	0.084	0.088	0.086	0.085	0.089	0.092	0.094
Tulsa, OK	Tulsa, OK	0.087	0.087	0.082	0.083	0.088	0.091	0.089	0.087	0.088	0.093
Kansas City, KS	Kansas City, MO-KS	0.082	0.083	0.082	0.082	0.090	0.092	0.094	0.093	0.091	0.089
Phoenix, AZ	Phoenix-Mesa-Scottsdale, AZ	0.083	0.091	0.088	0.086	0.089	0.090	0.092	0.091	0.088	0.088
Canton, OH	Canton-Massillon, OH	0.091	0.089	0.089	0.088	0.091	0.089	0.088	0.089	0.091	0.091
Columbus, OH	Columbus, OH	0.089	0.092	0.090	0.086	0.090	0.092	0.092	0.093	0.097	0.095
Detroit, MI	Detroit-Warren-Livonia, MI	0.096	0.091	0.089	0.088	0.093	0.094	0.092	0.093	0.095	0.089
Youngstown, OH	Youngstown-Warren-Boardman, OH-PA	0.090	0.091	0.091	0.089	0.091	0.092	0.093	0.096	0.096	0.092
Birmingham, AL	Birmingham-Hoover, AL	0.084	0.088	0.089	0.092	0.096	0.096	0.095	0.095	0.097	0.102
Boston, MA	Boston-Cambridge-Quincy, MA-NH	0.098	0.092	0.096	0.093	0.096	0.094	0.095	0.091	0.093	0.086
Milwaukee, WI	Milwaukee-Waukesha-West Allis, WI	0.101	0.095	0.090	0.084	0.092	0.097	0.098	0.093	0.097	0.092
Cincinnati, OH	Cincinnati-Middletown, OH-KY-IN	0.102	0.095	0.091	0.091	0.098	0.099	0.095	0.092	0.095	0.094
Cleveland, OH	Cleveland-Elyria-Mentor, OH	0.093	0.090	0.092	0.093	0.098	0.100	0.099	0.098	0.099	0.095
Charlotte, NC	Charlotte-Gastonia-Concord, NC-SC	0.092	0.091	0.091	0.092	0.094	0.094	0.097	0.103	0.104	0.104

City	Census Area Name	dv1989 _1991	dv1990 _1992	dv1991 _1993	dv1992 _1994	dv1993 _1995	dv1994 _1996	dv1995 _1997	dv1996 _1998	dv1997 _1999	dv1998 _2000
St Louis, MO	St. Louis, MO-IL	0.098	0.098	0.091	0.091	0.098	0.104	0.100	0.095	0.095	0.094
Chicago, IL	Chicago-Naperville-Joliet, IL-IN-WI	0.104	0.099	0.100	0.093	0.099	0.097	0.096	0.091	0.095	0.093
Pittsburgh, PA	Pittsburgh, PA	0.092	0.088	0.095	0.096	0.105	0.103	0.105	0.099	0.101	0.096
Nashville, TN	Nashville-Murfreesboro-Franklin, TN	0.096	0.096	0.095	0.096	0.099	0.099	0.099	0.101	0.102	0.100
Jersey City, NJ	Hudson County, NJ	0.107	0.104	0.103	0.096	0.100	0.095	0.098	0.093	0.100	0.092
Washington, DC	Washington-Arlington-Alexandria, DC-VA-MD-WV	0.100	0.100	0.101	0.096	0.098	0.094	0.100	0.101	0.106	0.101
Dallas/Ft Worth,	Dallas-Fort Worth-Arlington, TX	0.105	0.099	0.095	0.096	0.106	0.104	0.104	0.098	0.101	0.102
New Haven, CT	New Haven-Milford, CT	0.116	0.113	0.108	0.097	0.105	0.101	0.107	0.100	0.103	0.096
Sacramento, CA	Sacramento-Arden Arcade-Roseville, CA	0.105	0.105	0.110	0.104	0.106	0.106	0.099	0.103	0.103	0.107
Baltimore, MD	Baltimore-Towson, MD	0.104	0.106	0.107	0.103	0.107	0.105	0.107	0.104	0.109	0.107
Philadelphia, PA	Philadelphia-Camden-Wilmington, PA-NJ-DE-MD	0.113	0.107	0.106	0.099	0.104	0.101	0.110	0.107	0.110	0.106
San Diego, CA	San Diego-Carlsbad-San Marcos, CA	0.125	0.118	0.112	0.109	0.108	0.104	0.099	0.102	0.099	0.100
New York City, NY	New York-Northern New Jersey-Long Island, NY-NJ-PA	0.122	0.116	0.108	0.100	0.106	0.104	0.108	0.104	0.107	0.107
Atlanta, GA	Atlanta-Sandy Springs-Marietta, GA	0.104	0.105	0.101	0.101	0.109	0.105	0.110	0.113	0.118	0.121
Houston, TX	Houston-Sugar Land-Baytown, TX	0.119	0.116	0.104	0.110	0.114	0.116	0.117	0.116	0.118	0.112
Los Angeles, CA	Los Angeles-Long Beach-Santa Ana, CA	0.179	0.177	0.177	0.168	0.156	0.145	0.135	0.133	0.118	0.115
Note: Design values for this study were available in the last review (see Wells, 2012) and are presented in units of ppm, rather than ppb.											

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1 **Zu et al., 2017** (3859551) - Hospital Admissions for Asthma

2 6 Texas City Metro areas (Austin, Dallas, El Paso, Ft Worth, Houston, San Antonio), U.S. (*pooled, not individually assessed*)

3 O₃: 2001-2013

City	Census Area Name	dv.2001 .2003	dv.2002 .2004	dv.2003 .2005	dv.2004 .2006	dv.2005 .2007	dv.2006 .2008	dv.2007 .2009	dv.2008 .2010	dv.2009 .2011	dv.2010 .2012	dv.2011 .2013
Dallas and Fort Worth	Dallas-Fort Worth, TX-OK	100	98	95	96	95	91	86	86	90	87	87
El Paso	El Paso-Las Cruces, TX-NM	79	78	76	78	79	78	75	71	71	72	75
Houston	Houston-The Woodlands, TX	102	101	103	103	96	91	84	84	89	88	87
Austin	Austin-Round Rock, TX (CBSA only)	84	85	82	82	80	77	75	74	75	74	73
San Antonio	San Antonio-New Braunfels, TX (CBSA only)	89	91	86	87	82	78	74	75	75	80	81

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APPENDIX 3C

AIR QUALITY DATA USED IN POPULATION EXPOSURE AND RISK ANALYSES

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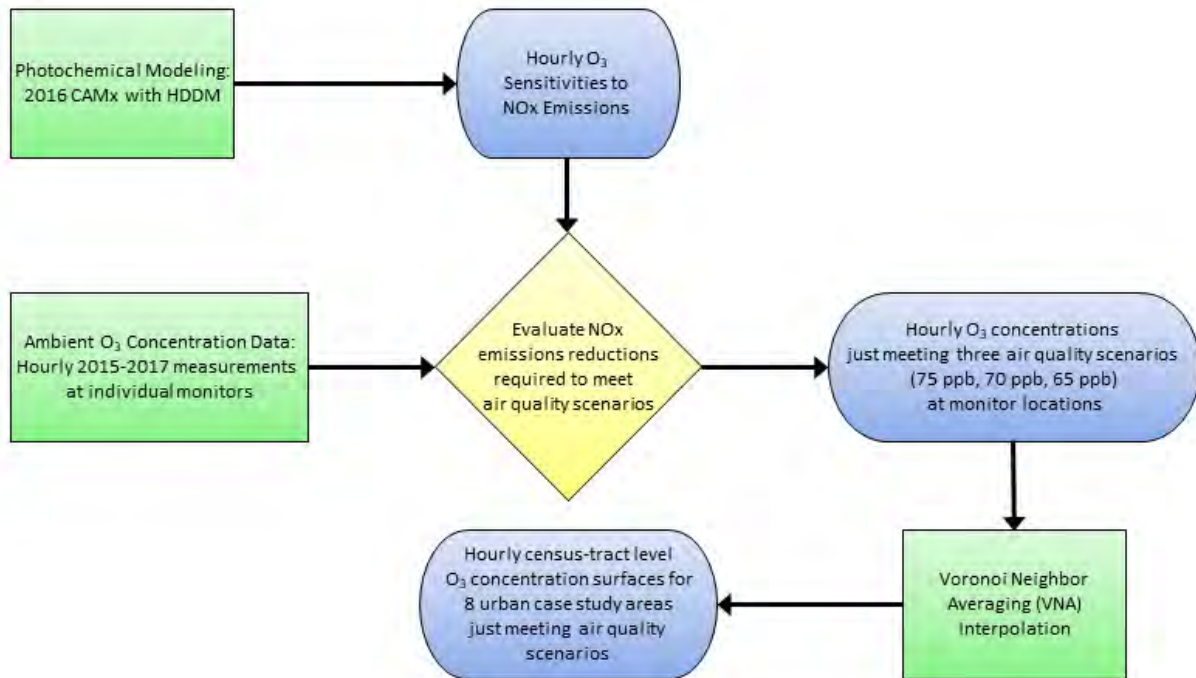
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1 **3C.1 OVERVIEW**

2 This appendix describes the development of the ozone (O₃) air quality estimates used in
3 the population exposure and risk modeling described in Appendix 3D. Figure 3C-1 below shows
4 a flowchart of the various data sources, processes and outputs involved in generating these
5 ambient O₃ concentration surfaces. This approach was used for eight urban study areas, which
6 are described further in section 3C.2.

7



8
9 **Figure 3C-1. Flowchart showing inputs, processes and outputs of the approach to**
10 **generate ambient air concentration estimates for use in the exposure and**
11 **risk modeling.**

12 Generation of the O₃ concentration surfaces for the exposure and risk modeling relied on
13 a combination of recent monitoring data and a model-based adjustment. Ambient hourly O₃
14 monitoring data for years 2015 through 2017 in each of the eight urban study areas was adjusted
15 using a model-based adjustment approach to create three different air quality scenarios. These
16 scenarios included conditions that just meet the current O₃ standard (design value of 70 ppb), as
17 well as conditions that just meet two alternative air quality scenarios having design values of 75
18 ppb and 65 ppb. Section 3C.3 provides additional information on the monitoring data. Section
19 3C.4 describes the air quality modeling that was used to perform the adjustments, as well as
20 results from the model evaluation that was performed to assess the accuracy of the modeled
21 concentrations. Section 3C.5 describes the model-based adjustment approach and its application
22 to the ambient air quality data to create the three air quality scenarios.

1 The final step in preparing the air quality input data for the exposure and risk modeling is
2 to interpolate the adjusted air quality data from the ambient air monitoring site locations to each
3 census tract in the eight urban study areas using Voronoi Neighbor Averaging (VNA), which is
4 described in section 3C.6. Finally, section 3C.7 provides various results from the model-based
5 adjustment procedure and the final air quality dataset used as inputs to the Air Pollutants
6 Exposure Model (APEX). The APEX model and its application to air quality in the eight urban
7 study areas is described in Appendix 3D.

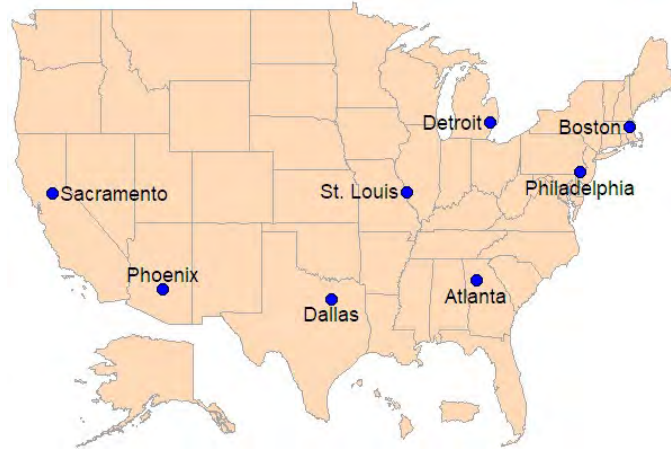
8 This appendix was developed in support of the risk and exposure analyses for the 2020
9 review. As outlined in section 1.5, the draft 2019 PA for the 2020 review, with a draft version of
10 this appendix, was made available for public comment and was reviewed and discussed by
11 CASAC in a public meeting (84 FR 50836, September 26, 2019; 84 FR 58711, November 1,
12 2019). In consideration of comments from the CASAC (Cox, 2020) and the public a number of
13 additional analyses and presentations were added to this appendix in the final 2020 PA (U.S.
14 EPA, 2020). These additions and clarifications included the following:

- 15 • Cites section in Appendix 3D for description of study area selection (section 3C.2);
- 16 • Summarizes differences in emissions between 2014 NEI and 2016 Platform used for
17 modeling in this assessment (section 3C.4.1.5);
- 18 • Adds clarifications regarding the model evaluation tables and figures presented in section
19 3C.4.2 (Figure 3C-12 to Figure 3C-47; Table 3C-5 to Table 3C-17);
- 20 • Provides rationale for choosing nitrogen oxides (NO_x) reductions only instead of the
21 combined NO_x and volatile organic compounds (VOC) reductions which were used in
22 the previous review (section 3C.5.2.2.3); and
- 23 • Adds a reference to a cross-validation analysis conducted in the last review, which
24 supports the use of the VNA technique for generating the air quality spatial fields (section
25 3C.6).

26 **3C.2 URBAN STUDY AREAS**

27 Eight urban study areas were chosen for analysis based on several criteria, including
28 geographic distribution, population, current air quality levels, availability of exposure model
29 inputs, air quality model performance, and ambient air monitoring network coverage. The
30 selection criteria and any other considerations in study area selection are described in Appendix
31 3D, section 3D.2.1. The eight urban study areas selected were: Atlanta, GA; Boston, MA; Dallas,
32 TX; Detroit, MI; Philadelphia, PA; Phoenix, AZ; Sacramento, CA; and St. Louis, MO. Figure
33 3C-2 shows a map of these eight study areas and Table 3C-1 provides summary information for
34 each area. The spatial extent of each study area was determined using the Combined Statistical

1 Area (CSA), with the exception of the Phoenix study area, which is not in a CSA. In that case,
 2 the Core Based Statistical Area (CBSA) was used as the area boundary.¹



3
 4 **Figure 3C-2. Map showing the location of the eight urban study areas.**

5
 6 **Table 3C-1. Summary information for the eight urban study areas.**

Study Area Name	CSA Name	Land Area (km ²)	Population (2010)	Number of O ₃ Monitors	2015-2017 DV (ppb)
Atlanta	Atlanta--Athens-Clarke County--Sandy Springs, GA	30,665	5,910,296	12	75
Boston	Boston-Worcester-Providence, MA-RI-NH-CT	25,117	7,893,376	23	73
Dallas	Dallas-Fort Worth, TX-OK	42,664	6,851,398	21	79
Detroit	Detroit-Warren-Ann Arbor, MI	16,884	5,318,744	13	73
Philadelphia	Philadelphia-Reading-Camden, PA-NJ-DE-MD	18,959	7,067,807	20	80
Phoenix	Phoenix-Mesa-Scottsdale, AZ ^A	34,799	4,192,887	30	76
Sacramento	Sacramento-Roseville, CA	18,871	2,414,783	21	86
St. Louis	St. Louis-St. Charles-Farmington, MO-IL	23,019	2,892,497	16	72

^A The Phoenix study area is not part of a CSA. The name listed in Table 3C-1 is the CBSA name.

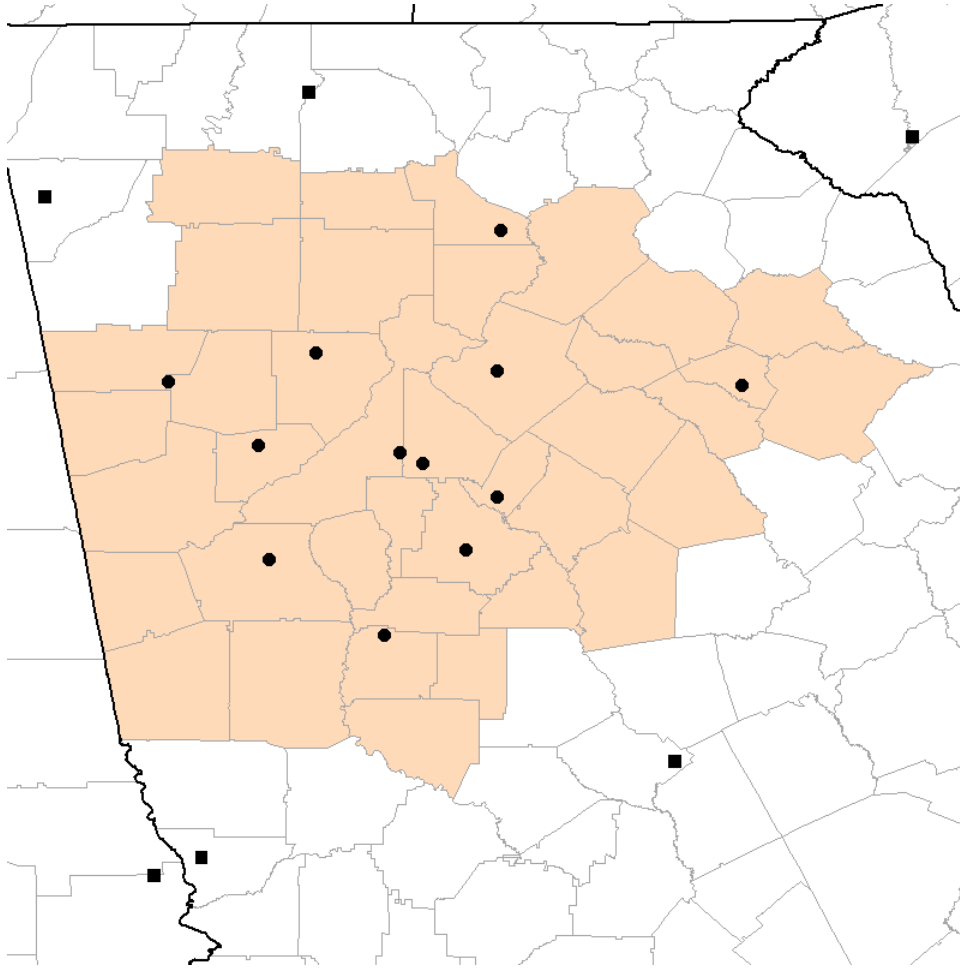
¹ CSA and CBSA boundaries are based on delineations promulgated by the Office of Management and Budget (OMB) in February of 2013. CBSA and CSA delineation files are available at <https://www.census.gov/geographies/reference-files/time-series/demo/metro-micro/delineation-files.html>.

3C.3 AMBIENT AIR OZONE MONITORING DATA

Hourly O₃ concentration data for all U.S. monitoring sites for 2015-2017 was retrieved from the EPA's Air Quality System (AQS) database in July of 2018. Design values² for 2015-2017 were calculated for each monitoring site according to the data handling requirements in Appendix U to 40 CFR Part 50. Monitors within the study area boundary for each urban study area were identified. These monitors were used to determine the NO_x emissions changes necessary to meet the current standard of 70 ppb, and the two alternative air quality scenarios having design values of 75 ppb and 65 ppb, following the model-based adjustment approach described in section 3C.5.

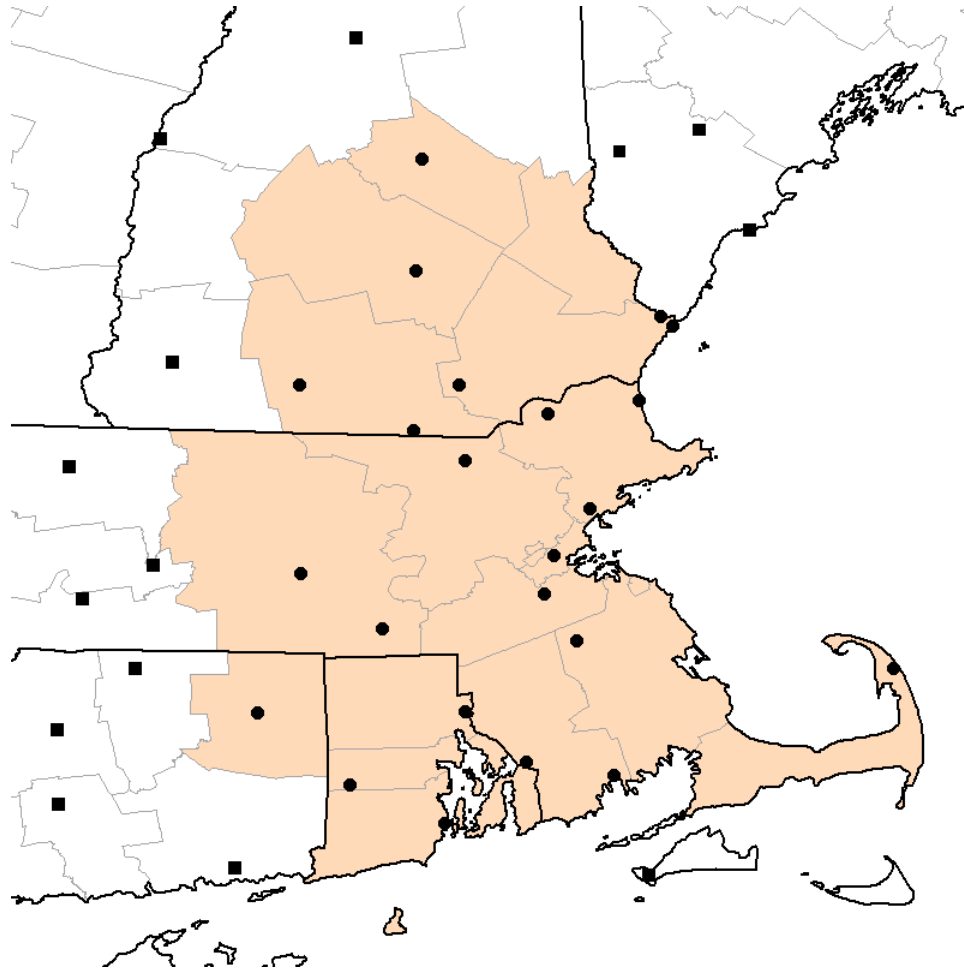
Additionally, monitors within 50 km of the study area boundary were identified as "buffer sites." Once the emissions changes required to meet the various air quality scenarios had been determined using the monitors within the CSA, these emissions changes were applied to both the CSA monitors and the buffer sites, as described in section 3C.5. The purpose of the buffer sites was to provide additional data for the spatial interpolation approach described in section 3C.6, providing improved estimates of air quality near the edges of the urban study area domain. Figure 3C-3 through Figure 3C-10 show maps of the boundaries for each urban study area, along with the locations of the monitoring sites used in the analysis. In each map, the shaded counties comprise the air quality domain of the urban study area used for estimating exposure and risk, the monitoring sites located inside the study area are denoted by black circles, and buffer sites are denoted by black squares.

² The design value is the 3-year average of the annual 4th highest daily maximum 8-hour average O₃ concentration. A monitoring site meets the current standard if its design value is less than or equal to 70 ppb.



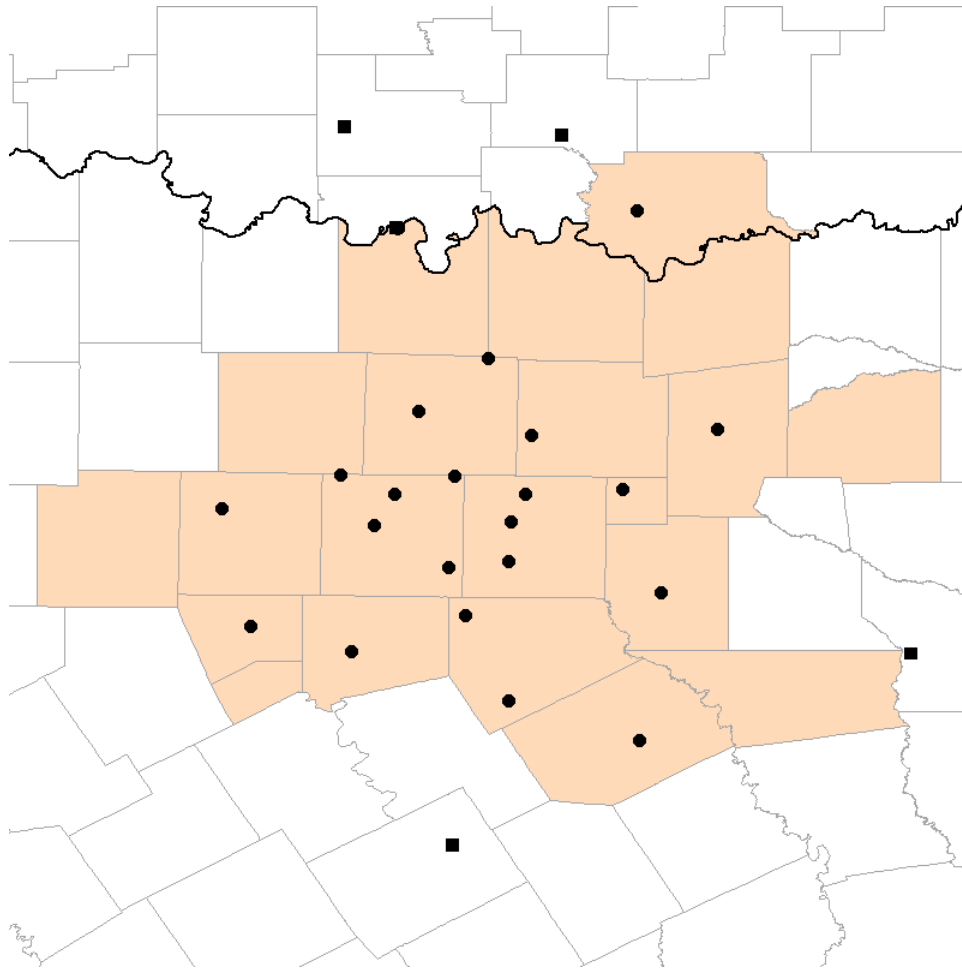
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Figure 3C-3. Map of the Atlanta study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.



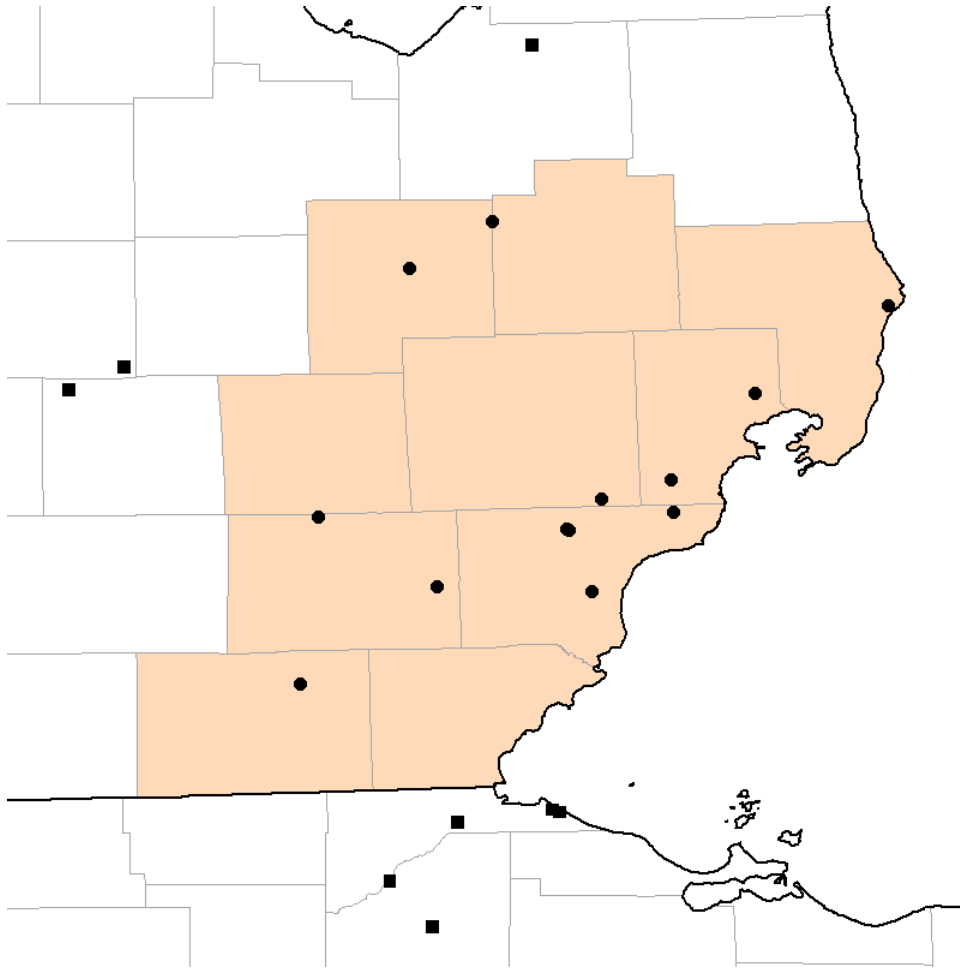
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Figure 3C-4. Map of the Boston study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.



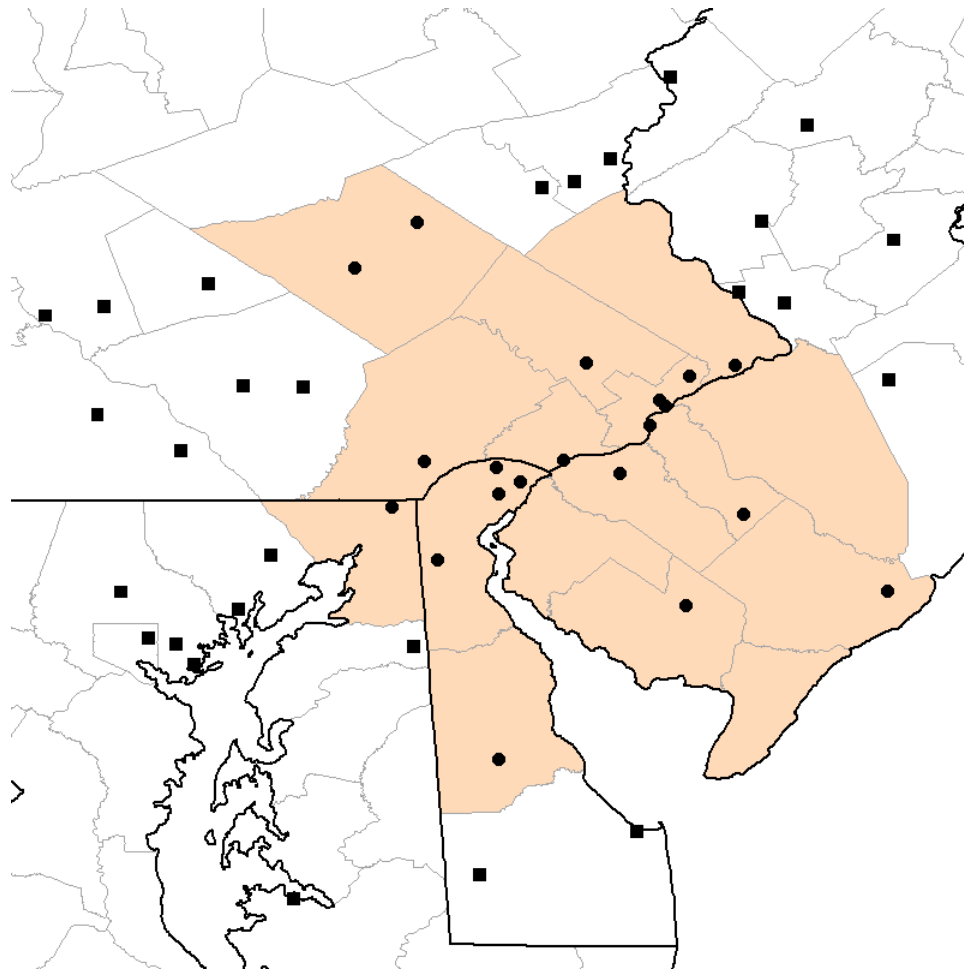
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Figure 3C-5. Map of the Dallas study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.



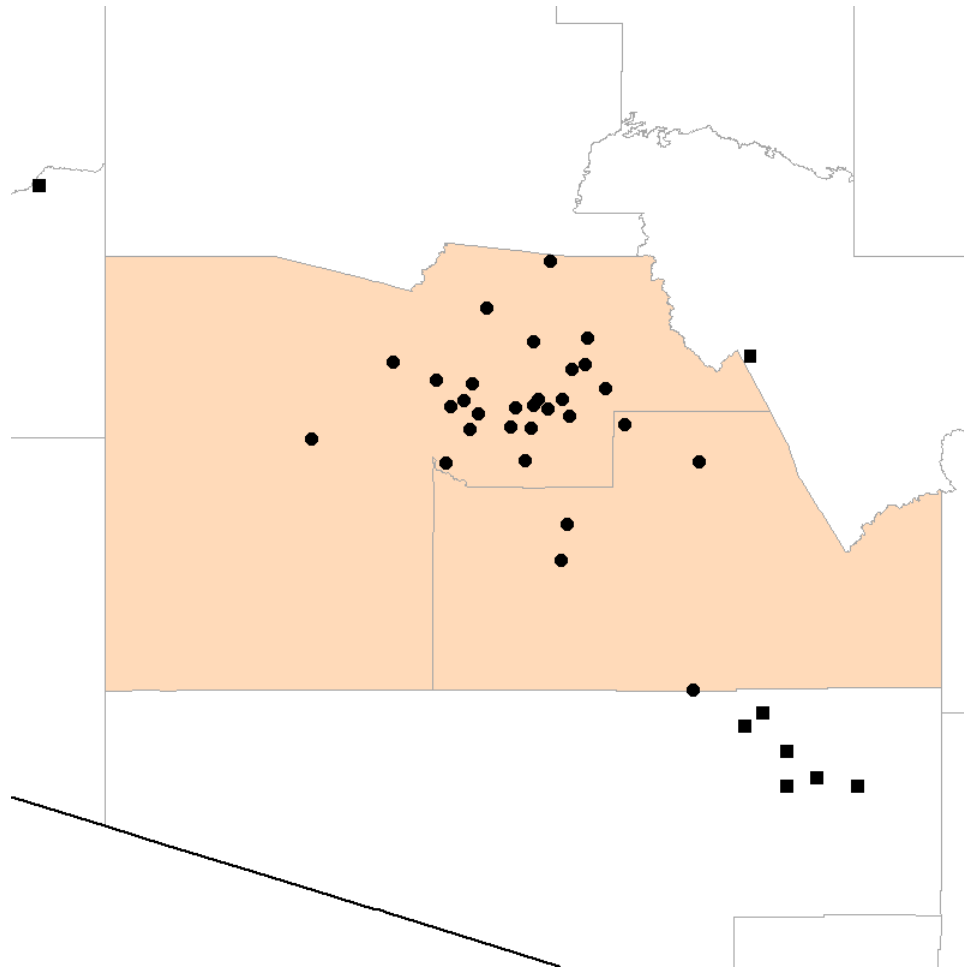
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Figure 3C-6. Map of the Detroit study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

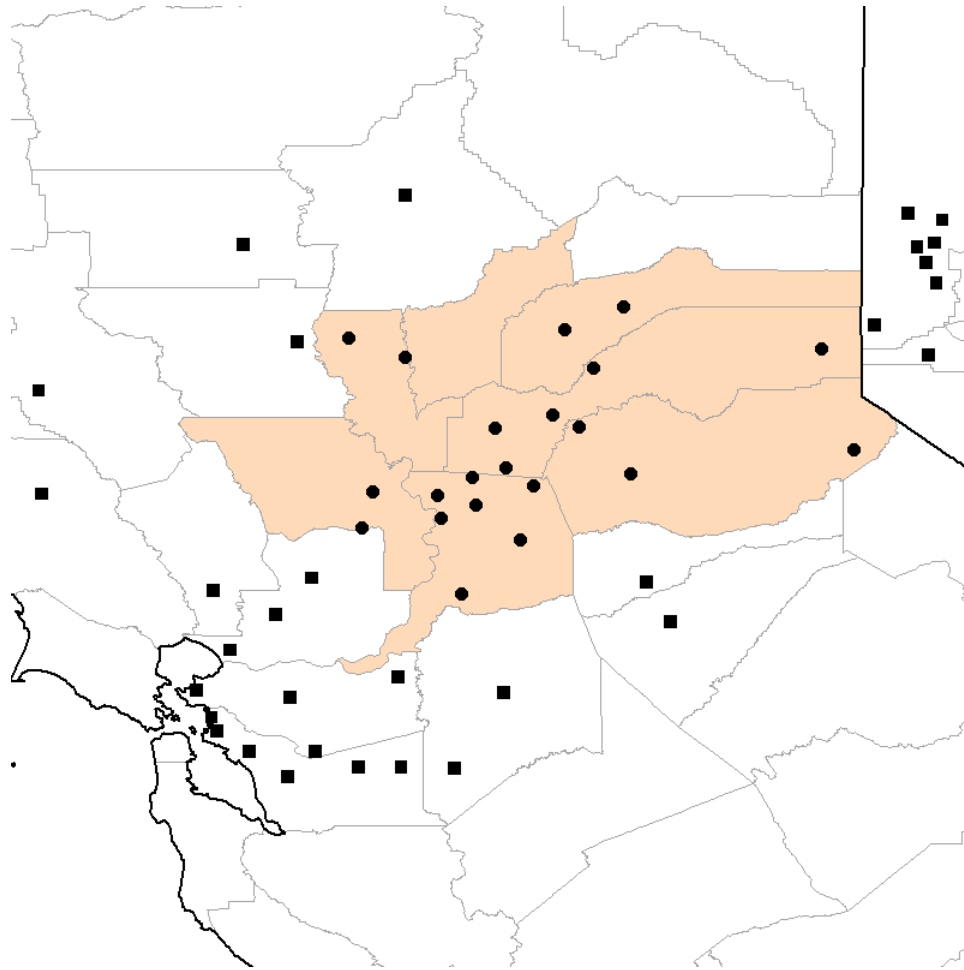


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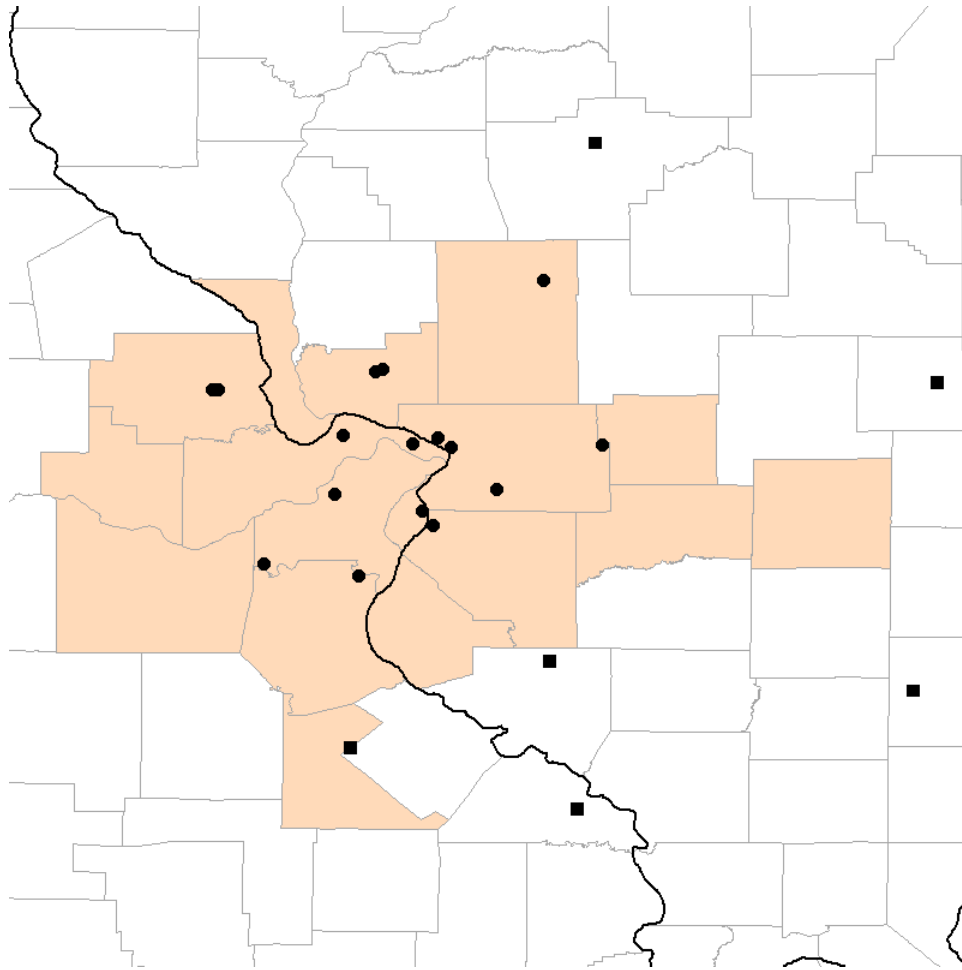
Figure 3C-7. Map of the Philadelphia study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.



1
2 **Figure 3C-8. Map of the Phoenix study area.** Counties in the CBSA are shaded, monitoring
3 sites in the CBSA are denoted by black circles, and buffer sites are denoted by
4 black squares.



1
2 **Figure 3C-9. Map of the Sacramento study area.** Counties in the CSA are shaded,
3 monitoring sites in the CSA are denoted by black circles, and buffer sites are
4 denoted by black squares.



1
 2 **Figure 3C-10. Map of the St. Louis study area.** Counties in the CSA are shaded, monitoring
 3 sites in the CSA are denoted by black circles, and buffer sites are denoted by
 4 black squares.

5 It is worth noting that for an area to show compliance with the current O₃ standard, all
 6 monitors within the urban area must have design values less than or equal to 70 ppb. According
 7 to Appendix U to 40 CFR Part 50, air quality monitors must also meet certain data completeness
 8 requirements to show compliance with the standard. However, any design value based on 3 years
 9 of monitoring data that exceeds the standard is not in compliance, regardless of data
 10 completeness. Therefore, when performing the air quality adjustments to create the three air
 11 quality scenarios, all monitors in each urban study area with data reported for each of the 3 years
 12 were included, regardless of data completeness.

13 Finally, per Appendix U to 40 CFR Part 50, data not meeting the ambient air monitoring
 14 requirements in 40 CFR Part 58, data reported using methods other than Federal Reference or
 15 Equivalent Methods, and data concurred by the appropriate EPA Regional Office as having been
 16 affected by an exceptional event were excluded from design value calculations. However, once
 17 the emissions changes required to determine compliance with the various air quality scenarios

1 had been determined, these values were included in the final adjustment and spatial interpolation.
2 In practice, fewer than 10,000 hourly concentrations out of more than 3 million (~0.3%) were
3 excluded from design value calculations in this manner.

4 **3C.4 AIR QUALITY MODELING DATA**

5 **3C.4.1 Comprehensive Air Quality Model with Extensions (CAMx)**

6 **3C.4.1.1 Model Set-up and Simulation**

7 The Comprehensive Air Quality Model with Extensions (CAMx) was used as the
8 modeling tool for this assessment. CAMx is a peer-reviewed model that simulates the formation
9 and fate of photochemical oxidants, aerosol concentrations, acid deposition, and air toxics, over
10 multiple scales for given input sets of meteorological conditions and emissions. CAMx is used
11 frequently for a range of scientific and regulatory applications related to the analysis of air
12 quality in the U.S. The Higher Order Direct Decoupled Method (HDDM) was implemented in
13 CAMx to estimate the model sensitivities to emissions changes as described in section 3C.5 of
14 this appendix. The CAMx-HDDM configuration tracks gas-phase species concentrations through
15 all modeled processes. However, HDDM implemented in CAMx does not track the effects of
16 aerosol and cloud processing on calculated O₃ sensitivities. Differences in predicted O₃
17 concentrations between the CAMx-HDDM configuration described here and a standard CAMx
18 v6.5 simulation with full treatment of aerosol-O₃ interactions did not influence O₃ predictions in
19 the urban study areas examined in this assessment. CAMx v6.5³ was run using the carbon bond
20 version 6 (CB06r4) gas-phase chemical mechanism (Yarwood et al., 2010; Gery et al., 1989) and
21 the AERO6 aerosol module which includes ISORROPIA for gas-particle partitioning of
22 inorganic species (Nenes et al., 1998) and secondary organic aerosol treatment as described in
23 Carlton et al. (2010).

24 **3C.4.1.2 Model Domain**

25 For this analysis, all CAMx runs were performed for a domain that covers the 48
26 contiguous states including portions of southern Canada and Northern Mexico with a 12 x 12 km
27 resolution (Figure 3C-11). The CAMx simulations were performed with 35 vertical layers with a
28 top layer at about 17,600 meters, or 50 millibars (mb). Table 3C-2 and Table 3C-3 provide some
29 basic geographic information regarding the CAMx domain and vertical layer structure,
30 respectively. Results from the lowest layer of the model were used for analyses to support the
31 risk and exposure analyses described in Appendix 3D.

32

³ For more information, see: http://www.camx.com/files/camxusersguide_v6-50.pdf.



1
2 **Figure 3C-11. Map of the CAMx modeling domain.**

3
4 **Table 3C-2. Geographic elements of domain used in the CAMx/HDDM modeling.**

Domain Element	CAMx Modeling Configuration Grid
Map Projection	Lambert Conformal Projection
Grid Resolution	12 km
True Latitudes	33 deg N and 45 deg N
Grid Dimensions	396 x 246 x 35
Vertical extent	35 Layers: Surface to 50 millibar level

1 **3C.4.1.3 Model Time Period**

2 The CAMx/HDDM modeling was performed for January 1 - December 31 of 2016. The
3 simulations included a 10-day spin-up period⁴ from December 22-31, 2015. The spin-up days
4 were not considered in the analysis for the HDDM results.

5 **3C.4.1.4 Model Inputs: Meteorology**

6 CAMx model simulations require inputs of meteorological fields, emissions, and initial
7 and boundary conditions. The gridded meteorological data for the entire year of 2016 at the 12
8 km continental U.S. scale domain were derived from version 3.8 of the Weather Research and
9 Forecasting Model (WRF), Advanced Research WRF (ARW) core (Skamarock et al., 2008). The
10 WRF Model is a mesoscale numerical weather prediction system developed for both operational
11 forecasting and atmospheric research applications.⁵ The 2016 WRF simulation included the
12 physics options of the Pleim-Xiu land surface model (LSM), Asymmetric Convective Model
13 version 2 planetary boundary layer (PBL) scheme, Morrison double moment microphysics,
14 Kain-Fritsch cumulus parameterization scheme and the RRTMG long-wave radiation (LWR)
15 scheme (Gilliam and Pleim, 2009). Additionally, lightning data assimilation was utilized to
16 suppress (force) deep convection where lightning was absent (present) in observational data.
17 This method is described by Heath et al. (2016) and was employed to help improve precipitation
18 estimates generated by the WRF model.

19 The WRF and CAMx simulations used the same map projection, a Lambert conformal
20 projection centered at (-97, 40) with true latitudes at 33 and 45 degrees north. The WRF and
21 CAMx simulations utilized 35 vertical layers with a surface layer of approximately 19 meters.
22 Table 3C-3 shows the vertical layer structure used in WRF to generate the CAMx meteorological
23 inputs.

24 The WRF meteorological outputs were processed to create model-ready inputs for CAMx
25 using the wrfcamx version 4.3 meteorological pre-processor (Ramboll Environ, 2014). The
26 specific meteorological inputs to CAMx include: horizontal wind components (i.e., speed and
27 direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each grid cell in
28 each vertical layer.

⁴ It is standard practice to allow chemical transport models to run for several days to weeks prior to the time period of interest in order to minimize the influence of initial conditions.

⁵ See: <http://wrf-model.org>

1 **Table 3C-3. Vertical layer structure for 2016 WRF and CAMx simulations.**

Layer Top Height (m)	Pressure (mb)	Model Layer
17,556	50	35
14,780	97.5	34
12,822	145	33
11,282	192.5	32
10,002	240	31
8,901	287.5	30
7,932	335	29
7,064	382.5	28
6,275	430	27
5,553	477.5	26
4,885	525	25
4,264	572.5	24
3,683	620	23
3,136	667.5	22
2,619	715	21
2,226	753	20
1,941	781.5	19
1,665	810	18
1,485	829	17
1,308	848	16
1,134	867	15
964	886	14
797	905	13
714	914.5	12
632	924	11
551	933.5	10
470	943	9
390	952.5	8
311	962	7
232	971.5	6
154	981	5
115	985.75	4
77	990.5	3
38	995.25	2
19	997.63	1

2

3 A detailed meteorological model performance evaluation was conducted for the 2016
 4 WRF simulations (U.S. EPA, 2017). The analysis included statistical evaluation of temperature,
 5 wind speed, and water vapor mixing ratios against observational data from airports, as well as
 6 evaluations of monthly precipitation compared to the Parameter-elevation Relationships on
 7 Independent Slopes Model (PRISM) and shortwave radiation compared to data from the Surface

1 Radiation Budget Measurement Network (SURFRAD) and the Solar Radiation Network
2 (SOLRAD).

3 **3C.4.1.5 Model Inputs: Emissions**

4 The emissions data used are based on the alpha version of the Inventory Collaborative
5 2016 emissions modeling platform.⁶ The modeling case used is abbreviated “2016fe” and is
6 publicly available.⁷

7 Emissions were processed to photochemical model inputs with the SMOKE modeling
8 system version 4.5 (Houyoux et al., 2000). For this analysis, emissions from wildfires and
9 prescribed burns were based on year 2016 nationally available fire datasets. Electric generating
10 unit (EGU) emissions are temporally allocated to hourly values based on patterns derived from
11 year 2016 Continuous Emissions Monitoring System (CEMS) data. In addition, U.S. emissions
12 are included from other point sources, area sources, agricultural sources (ammonia only),
13 anthropogenic fugitive dust sources, nonroad mobile sources, onroad mobile sources, and
14 biogenic sources. Emissions for onroad mobile sources were created using the EPA’s MOVES
15 2014a model,⁸ except that California emissions were adjusted to match the county total
16 emissions obtained directly from the California Air Resources Board. Biogenic emissions were
17 estimated using the Biogenic Emissions Inventory System version 3.61 (BEISv3.61) (Pouliot and
18 Bash, 2015). Other North American emissions from areas outside the U.S. are based on a 2013
19 Canadian inventory scaled to 2015, and projections of the 2008 Mexican inventory to the year
20 2016 along with the scaling of MOVES-Mexico emissions to year 2016 (ERG, 2017). The
21 construction of the emissions is described in more detail in the technical support document
22 Preparation of Emissions Inventories for the Version 7.1 2016 Regional Emissions Modeling
23 Platform (U.S. EPA, 2019). Emissions totals within the United States are summarized in Table
24 3C-4 for CO, NH₃, NO_x, PM₁₀, PM_{2.5}, SO₂, and VOC. Anthropogenic NO_x emissions in the
25 2016 platform are about 19% lower than those reported in the 2014 NEI due to both improved
26 inventory development methods and updates to specific components (e.g., cleaner vehicles
27 entering the onroad mobile fleet or EGUs transitioning from coal to natural gas).

28

⁶ <http://views.cira.colostate.edu/wiki/wiki/9169>

⁷ <https://www.epa.gov/air-emissions-modeling/2016-alpha-platform>

⁸ <https://www.epa.gov/moves>

1 **Table 3C-4. Summary of U.S. emissions totals by sector for the 12km CONUS domain (in**
 2 **thousand tons). “NA” indicates not applicable.**

Sector Abbrev.	Sector Description	CO	NH ₃	NO _x	PM ₁₀	PM _{2.5}	SO ₂	VOC
afdust_adj	Anthropogenic fugitive dust	NA	NA	NA	6,217	874	NA	NA
ag	Agricultural sources	NA	2,777	NA	NA	NA	NA	NA
ptagfire	Agricultural fires	593	80	18	96	68	6	36
cmv_c1c2	Category 1 and 2 Commercial Marine Vessels	47	NA	260	6	6	NA	5
cmv_c3	Ocean-going (Category 3) Commercial Marine Vessels	11	NA	108	4	4	4	5
nonpt	Nonpoint (area) sources not in other sectors	2,681	121	758	609	496	162	3,673
np_oilgas	Nonpoint oil and gas sources	642	NA	676	18	17	39	2,986
nonroad	Nonroad (off-road) equipment	12,189	2	1,207	122	115	2	1,465
onroad	Onroad mobile sources	20,446	101	4,046	273	130	27	1,962
ptfire	Wild and Prescribed Fires	23,642	388	333	2,415	2,046	181	5,581
ptegu	Point sources: electric generation units	672	25	1,289	171	141	1,545	33
ptnonipm	Point sources other than electric generating units	1,848	61	1,073	407	264	673	809
pt_oilgas	Oil and gas-related Point Sources	178	4	360	12	11	42	133
rail	Locomotive emissions	118	NA	673	21	19	1	35
rwc	Residential Wood Combustion emissions	2,099	15	30	314	314	8	338
Total anthro	Total US anthropogenic emissions (including wildfires)	65,167	3,576	10,832	10,685	4,507	2,689	17,241
beis	U.S. biogenic emissions	7,297	NA	979	NA	NA	NA	42,861
Total with biogenic	Total US emissions including biogenic emissions	72,463	3,576	11,812	10,685	4,507	2,689	60,102

3 **3C.4.1.6 Model Inputs: Boundary and Initial Conditions**

4 Initial and lateral boundary concentrations for the 12 km US2 domain are provided by the
 5 hemispheric version of the Community Multi-scale Air Quality model (H-CMAQ) v5.2.1. H-
 6 CMAQ was run for 2016 with a horizontal grid resolution of 108 km and 44 vertical layers up to
 7 50 hPa. The H-CMAQ predictions were used to provide one-way dynamic boundary conditions
 8 at one-hour intervals. An operational evaluation against sonde and satellite observations showed

1 that the 2016 H-CMAQ simulation reasonably captured general patterns of O₃ transport within
2 the northern Hemisphere that are relevant for the 12US2 domain (Henderson et al., 2018).

3 **3C.4.2 Evaluation of Modeled Ozone Concentrations**

4 In this section we present the results of an evaluation of the CAMx configuration used to
5 produce the air quality results described in Chapter 3. Specifically, we summarize the ability of
6 the CAMx model to reproduce the corresponding 2016 measured O₃ concentrations. This
7 operational evaluation shows that in general for most regions and seasons, the CAMx model
8 predictions for 2016 generally reproduce patterns of observed O₃. The notable exception to this
9 is a persistent underestimate in winter across almost all regions, particularly at higher latitude
10 sites.

11 In the following sections we present general model performance statistics and plots for
12 five regions of the U.S. We compare model predictions of maximum daily 8-hr average (MDA8)
13 O₃ concentrations to measurements reported in EPA’s AQS. We note that these comparisons are
14 based on MDA8 values calculated across all available modeled CAMx values and all observed
15 (AQS) concentrations, and that these comparisons include buffer sites. Model performance could
16 be different for comparisons without buffer sites, or using the modeled CAMx MDA8 values
17 only when the corresponding observed MDA8 values are available.

18 The model statistics presented here include mean bias, mean error, normalized mean bias,
19 and normalized mean error as calculated below, where *n* represents the total number of
20 observations:

21 Mean Bias:	$(\sum \text{modeled} - \text{observed})/n$
22 Mean Error:	$(\sum \text{modeled} - \text{observed})/n$
23 Normalized Mean Bias:	$(\sum \text{modeled} - \text{observed})/(\sum \text{observed})$
24 Normalized Mean Error	$(\sum \text{modeled} - \text{observed})/(\sum \text{observed})$

25 Our analysis focuses on regional model evaluation statistics from five US regions as well
26 as evaluations of the eight urban study areas included in the exposure and risk analysis – Atlanta,
27 Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento, and St. Louis.^{9,10} Statistics for
28 CAMx model performance in these regions and urban study areas are shown by season in Table
29 3C-5 through Table 3C-17 for observed days with MDA8 O₃ values ≥ 60 ppb, observed days

⁹ The five regions are defined as follows: Northeast (Connecticut, Delaware, District of Columbia, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, Vermont), Southeast (Alabama, Florida, Georgia, Kentucky, Mississippi, North Carolina, South Carolina, Tennessee, Virginia, West Virginia), Midwest (Illinois, Indiana, Michigan, Ohio, Wisconsin), Central (Arkansas, Iowa, Kansas, Louisiana, Minnesota, Missouri, Nebraska, Oklahoma, Texas), and West (Arizona, California, Colorado, Idaho, Nevada, New Mexico, Oregon, Utah, Wyoming).

¹⁰ Monitoring sites for each urban study area were selected based on core-based statistical area (CBSA) groupings.

1 with MDA8 O₃ < 60 ppb, and for all observed days. For each of the five regions listed above,
2 spatial plots are provided for each season showing Normalized Mean Bias (NMB) for MDA8 O₃
3 at individual sites. Summary NMB ranges are included at the bottom of each map showing the
4 min and max values for the season/region across all sites, as well as the 25th, 50th, and 75th
5 percentile values. Time series plots are provided for MDA8 O₃ in each urban study area for the
6 period from January-December 2016. Hourly time series plots are also provided for one month in
7 each season (January, April, July, October).¹¹

8 **3C.4.2.1 Operational Evaluation in the Northeastern U.S.**

9 Table 3C-5 shows that in the Northeast Region, model mean bias was generally less than
10 7 ppb and normalized mean bias was less than 15% in most cases. Errors were largest in the
11 winter, with underestimates also extending to the spring. Spatial maps of normalized mean bias
12 are shown in Figure 3C-12 through Figure 3C-15. During the O₃ season performance was best on
13 high O₃ days, particularly in the summer and fall. Two of the eight urban study areas evaluated
14 were in the Northeast: Boston and Philadelphia.

15 Model performance at the Boston study area monitoring sites (Table 3C-6) was similar to
16 that of the Northeast Region. The time series plots show that the model reasonably reproduces
17 the measured day-to-day variability in MDA8 O₃ concentrations (Figure 3C-16). The
18 underestimate in winter-spring observed in the Northeast region statistics is particularly
19 pronounced in Boston, likely due to its relatively northerly location where seasonal daylight and
20 temperature changes are more exaggerated. Variability of hourly daytime and nighttime O₃
21 concentrations is generally well modeled in all seasons, again noting the persistent underestimate
22 in January/April. Model characterization of hourly variability is particularly good in July,
23 although peak daytime O₃ is slightly overestimated. Nighttime O₃ is also consistently
24 overestimated in July/October (Figure 3C-17).¹²

25 Bulk model performance statistics for Philadelphia (Table 3C-7) are again similar to
26 those for the Northeast as a whole, with more moderate performance compared to Boston during
27 both winter (not as poor) and summer/fall (not as good). The spring underestimate present in the
28 Boston comparisons is much smaller for Philadelphia (Figure 3C-18, Figure 3C-19), again
29 suggesting that the winter-spring underestimate is more pronounced at more northerly sites.
30 Philadelphia also exhibits the nighttime overestimates in the July/October hourly comparisons
31 seen in Boston, with slightly higher overestimates of peak July daytime concentrations.

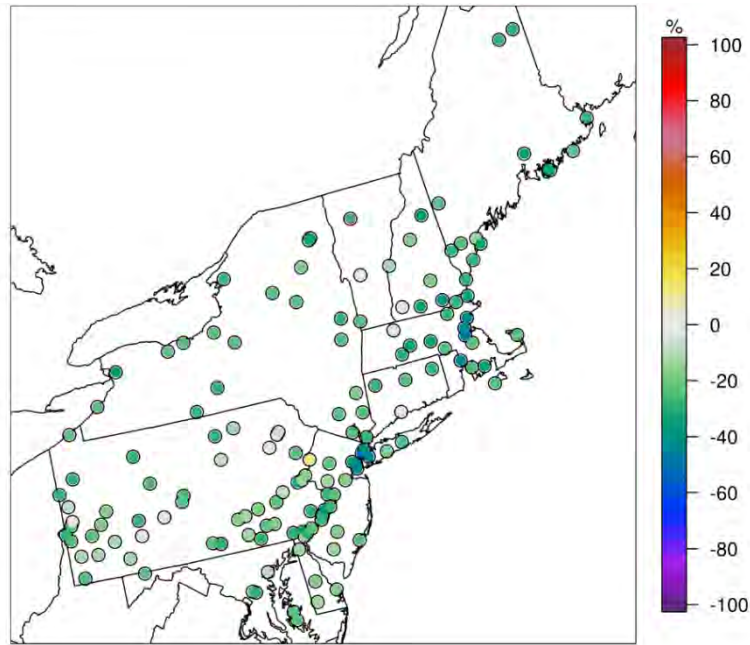
¹¹ Note that the MDA8 and hourly time series show average concentrations across all monitors within each urban study area. The number of monitors included in this average sometimes changes by season since different monitors within each study area take measurements over different periods of the year.

¹² Note that the Y-axis scale for the various time series are not consistent.

1 **Table 3C-5. CAMx model performance at monitoring sites in the Northeastern U.S.**
 2 **Statistics shown are mean bias (MB), normalized mean bias (NMB), mean**
 3 **error (ME), and normalized mean error (NME).**

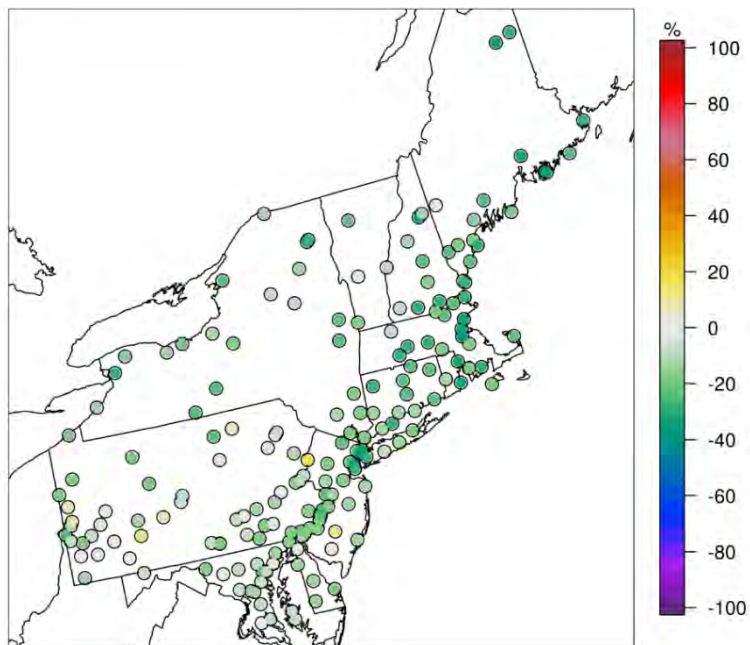
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	7056	-6.4	-21.0	7.3	23.8
	Days ≥ 60	1	-26.7	-42.4	26.7	42.4
	All Days	7057	-6.4	-21.0	7.3	23.8
Spring	Days < 60	7493	-6.2	-14.7	7.8	18.6
	Days ≥ 60	511	-5.1	-7.6	7.3	10.8
	All Days	8004	-6.1	-14.0	7.7	17.8
Summer	Days < 60	7385	5.0	11.8	7.7	18.1
	Days ≥ 60	870	0.8	1.2	6.7	10.2
	All Days	8255	4.5	10.1	7.6	16.9
Fall	Days < 60	7612	1.3	3.9	5.6	17.6
	Days ≥ 60	135	-0.9	-1.4	5.4	8.1
	All Days	7747	1.2	3.7	5.6	17.3

4
5



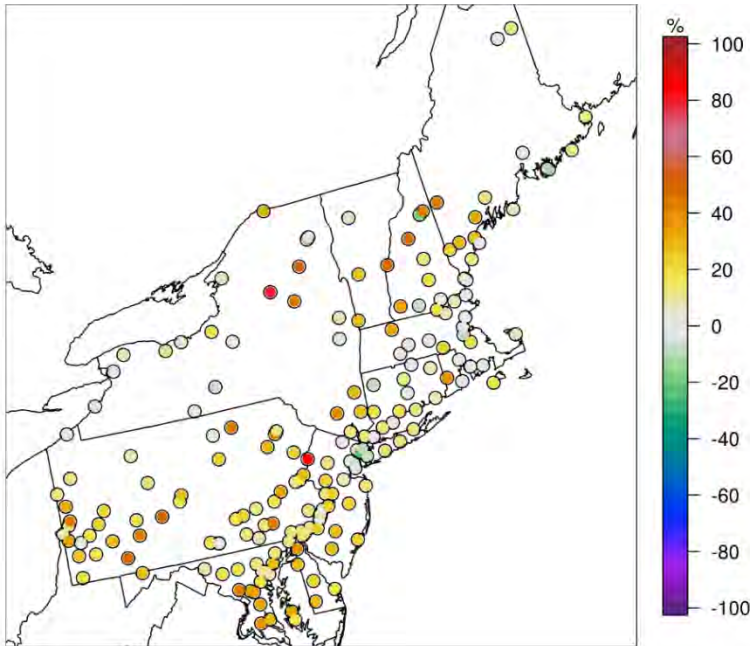
Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-55, -26, -22, -16, 14]

6
7 **Figure 3C-12. Normalized mean bias for MDA8 O₃ in the Northeastern U.S., winter 2016.**



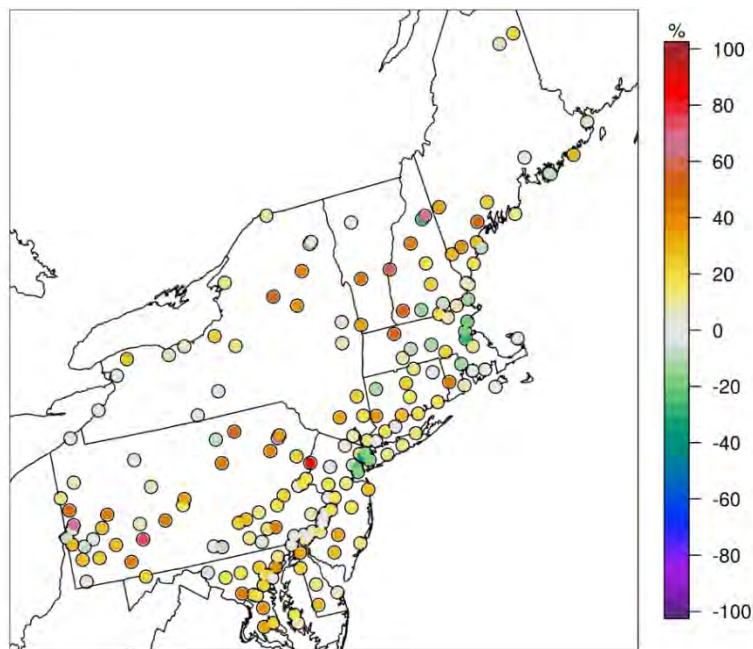
Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-43, -20, -14, -7.1, 23]

1
 2 **Figure 3C-13. Normalized mean bias for MDA8 O₃ in the Northeastern U.S., spring 2016.**
 3



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-21, 7.8, 17, 28, 85]

4
 5 **Figure 3C-14. Normalized mean bias for MDA8 O₃ in the Northeastern U.S., summer**
 6 **2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-37, 4.8, 15, 28, 93]

1

2 **Figure 3C-15. Normalized mean bias for MDA8 O₃ in the Northeastern U.S., fall 2016.**

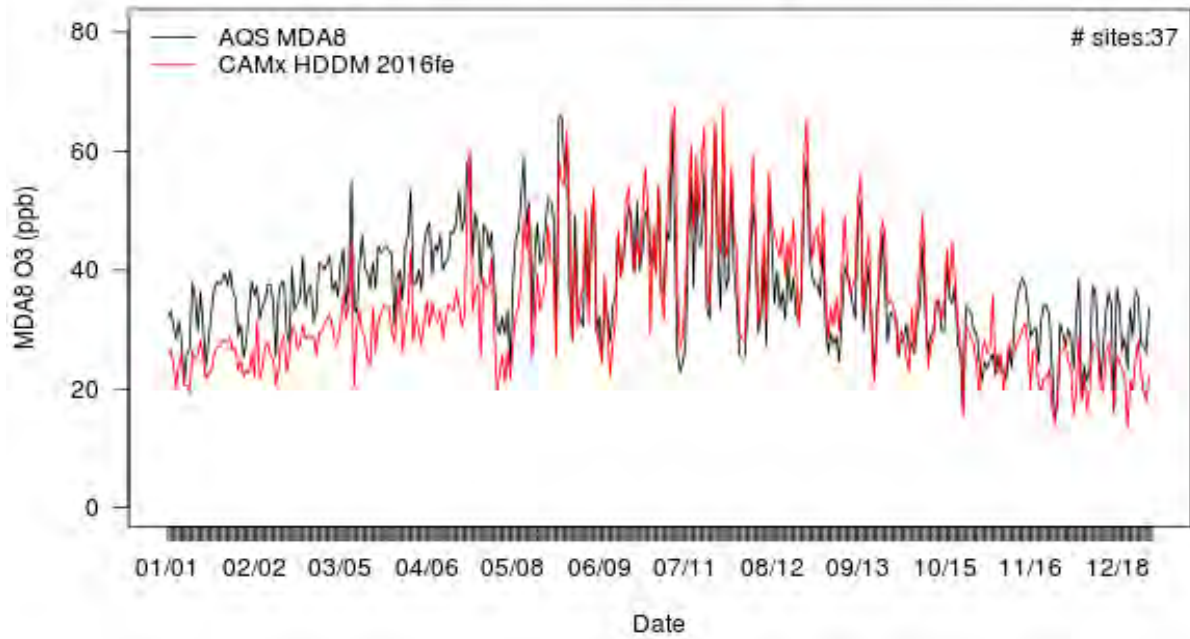
3

4 **Table 3C-6. CAMx model performance at monitoring sites in the Boston study area.**

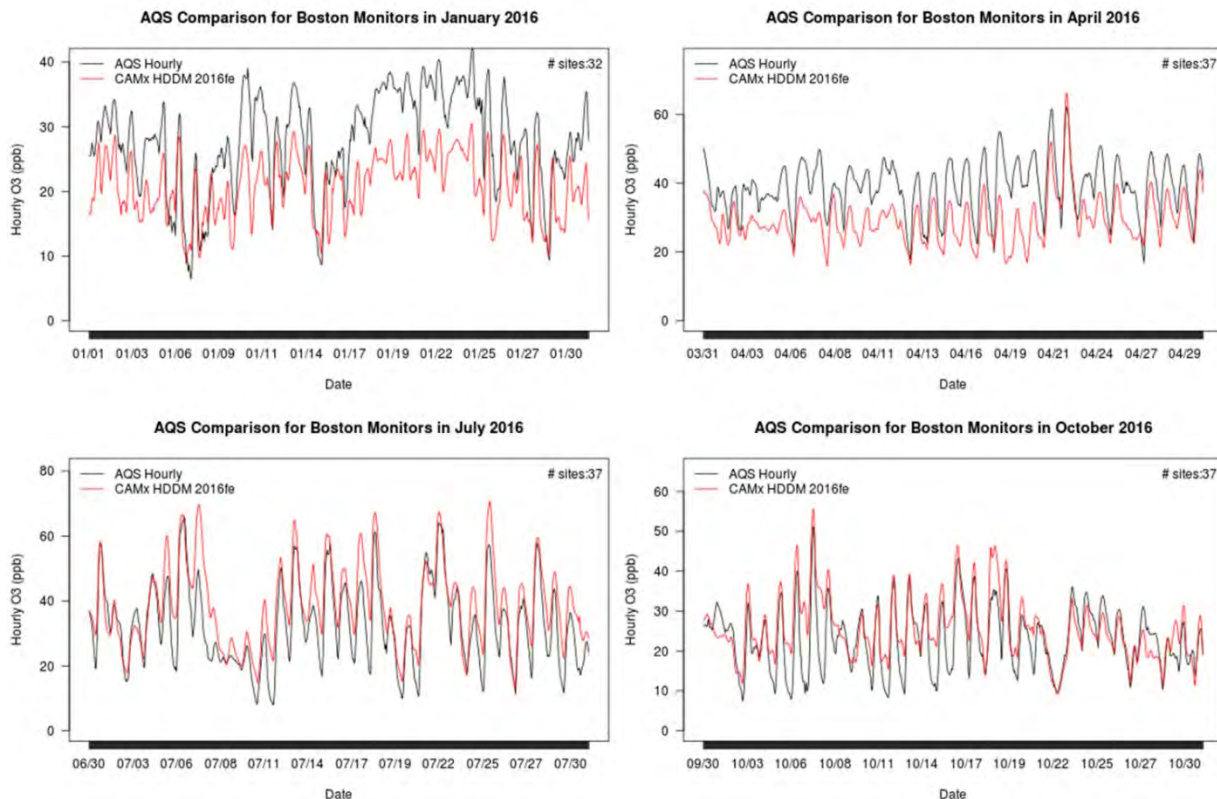
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	1346	-8.4	-25.6	8.9	27.2
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	1346	-8.4	-25.6	8.9	27.2
Spring	Days < 60	82	-9.1	-21.3	9.9	23.3
	Days ≥ 60	1476	-8.6	-12.6	10.4	15.2
	All Days	1558	-9.0	-20.6	9.9	22.6
Summer	Days < 60	1484	3.6	9.0	6.2	15.7
	Days ≥ 60	146	1.2	1.8	5.9	8.9
	All Days	1630	3.3	8.0	6.2	14.8
Fall	Days < 60	1482	-0.6	-1.8	5.4	17.4
	Days ≥ 60	8	0.3	0.43	5.4	8.4
	All Days	1490	-0.6	-1.8	5.4	17.3

5

AQS MDA8 Comparison for Boston Monitors in 2016



1
2 **Figure 3C-16. Time series of monitored (black) and modeled (red) MDA8 O₃ at Boston**
3 **monitoring sites in 2016.**
4



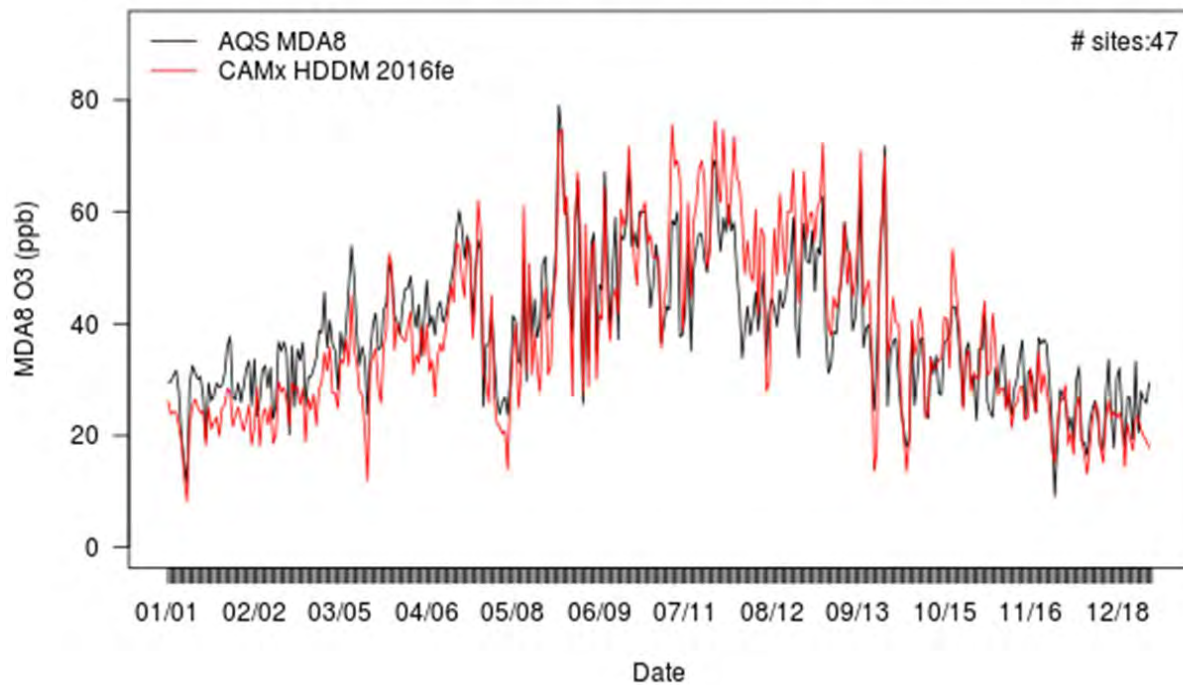
1
 2 **Figure 3C-17. Time series of monitored (black) and modeled (red) hourly O₃**
 3 **concentrations at Boston monitoring sites in January (top left), April (top**
 4 **right), July (bottom left), and October (bottom right) 2016.**

5
 6 **Table 3C-7. CAMx model performance at monitoring sites in the Philadelphia study area.**

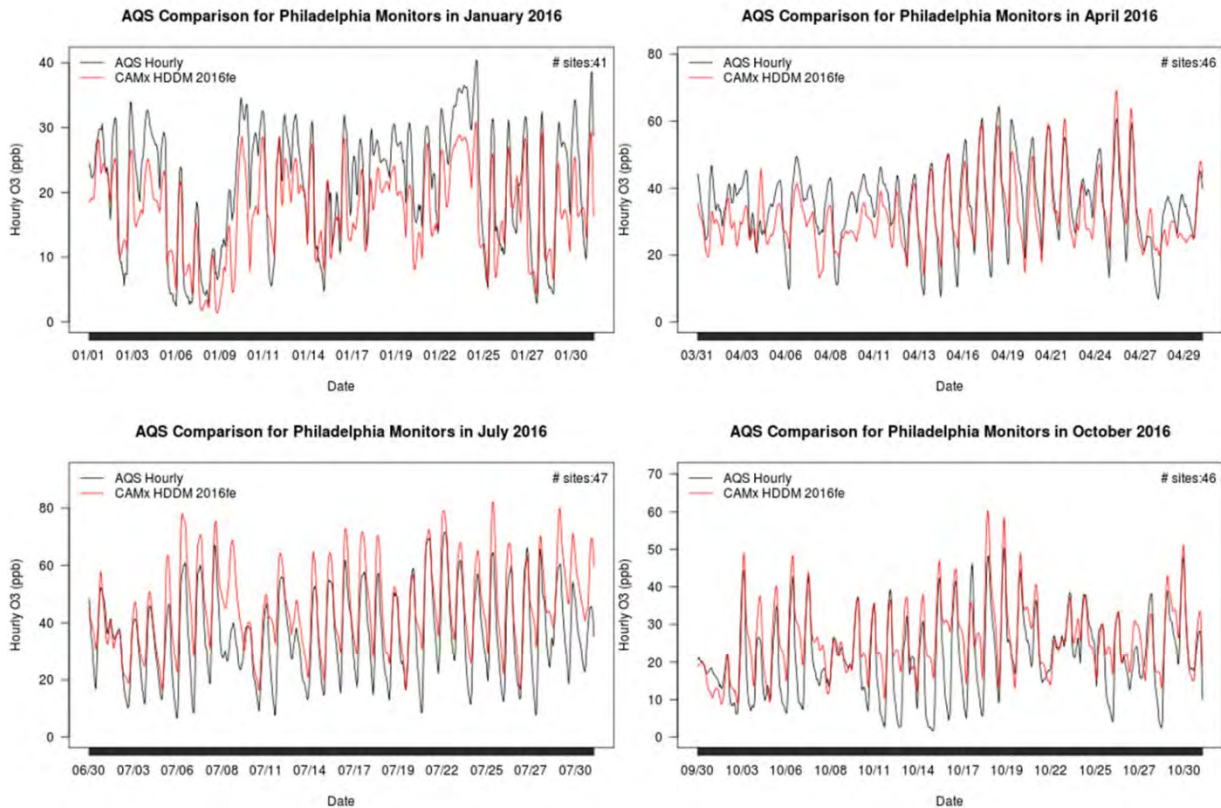
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	2151	-5.0	-17.9	6.1	21.7
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	2151	-5.0	-17.9	6.1	21.7
Spring	Days < 60	2328	-4.5	-10.9	6.6	16.0
	Days ≥ 60	150	-3.0	-4.4	5.1	7.5
	All Days	2478	-4.4	-10.3	6.5	15.2
Summer	Days < 60	2229	6.7	14.7	9.1	20.2
	Days ≥ 60	352	1.0	1.5	6.8	10.3
	All Days	2581	5.9	12.3	8.8	18.3
Fall	Days < 60	2333	1.9	5.9	5.7	17.7
	Days ≥ 60	71	-1.0	-1.4	5.2	7.7
	All Days	2404	1.8	5.5	5.7	17.1

7

AQS MDA8 Comparison for Philadelphia Monitors in 2016



1
2 **Figure 3C-18. Time series of monitored (black) and modeled (red) MDA8 O₃ at**
3 **Philadelphia monitoring sites in 2016.**



1
2 **Figure 3C-19. Time series of monitored (black) and modeled (red) hourly O₃**
3 **concentrations at Philadelphia monitoring sites for January (top left), April**
4 **(top right), July (bottom left), and October (bottom right) 2016.**

6 3C.4.2.2 Operational Evaluation in the Southeastern U.S.

7 In the Southeast region, mean bias for MDA8 O₃ was generally less than ~5 ppb at most
8 sites in all seasons, as indicated in Table 3C-8. The exception is winter, where there were only
9 four days with measured MDA8 > 60 ppb and all were largely underpredicted. Spatial maps of
10 normalized mean bias are shown in Figure 3C-20 through Figure 3C-23. Performance was best
11 in the spring (slightly underestimated) and on high O₃ days in the summer/fall. Atlanta was the
12 only one of the eight urban study areas located in the Southeast region.

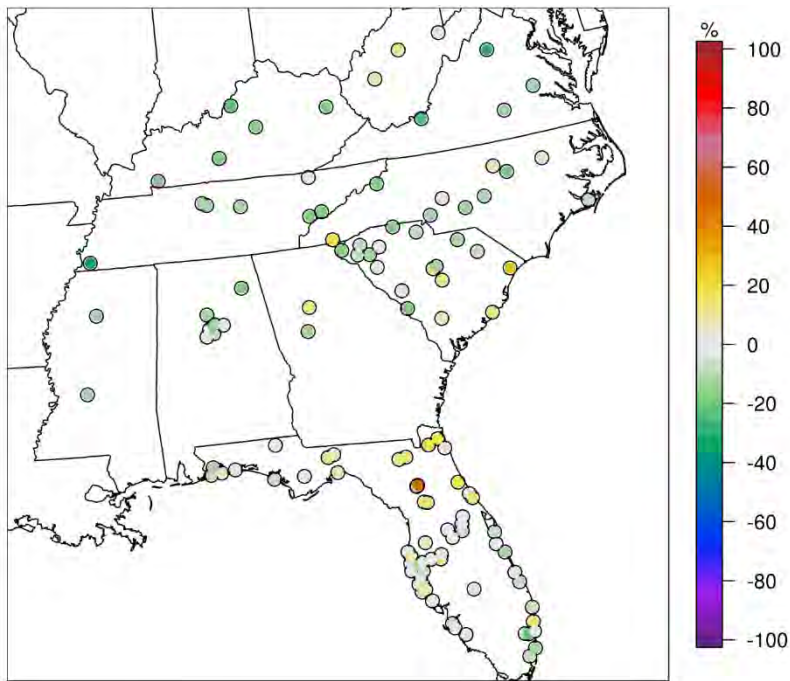
13 Mean bias and normalized mean bias at Atlanta sites for the spring, summer, and fall
14 months were typical of performance throughout the Southeast region, with much better
15 performance in winter. The MDA8 O₃ time series (Figure 3C-24) shows that the model
16 reasonably represents the variability occurring on high and low O₃ concentration days. The
17 hourly time series plots (Figure 3C-25) also show reasonable model performance during daytime
18 hours but some persistent overestimates of both nighttime and peak daytime O₃ occur, especially
19 in July.

1

2 **Table 3C-8. CAMx model performance at monitoring sites in the Southeastern U.S.**

Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	3775	-3.2	-9.2	5.3	15.4
	Days ≥ 60	4	-27.2	-40.6	27.2	40.6
	All Days	3779	-3.2	-9.2	5.3	15.4
Spring	Days < 60	7193	-0.6	-1.4	5.2	11.7
	Days ≥ 60	468	-2.6	-4.0	5.0	7.8
	All Days	7661	-0.7	-1.6	5.2	11.3
Summer	Days < 60	7825	5.2	13.9	7.6	20.2
	Days ≥ 60	396	0.4	0.6	6.2	9.5
	All Days	8221	5.0	12.8	7.5	19.3
Fall	Days < 60	6456	3.4	8.7	6.0	15.5
	Days ≥ 60	139	0.6	0.9	4.8	7.6
	All Days	6595	3.3	8.4	6.0	15.2

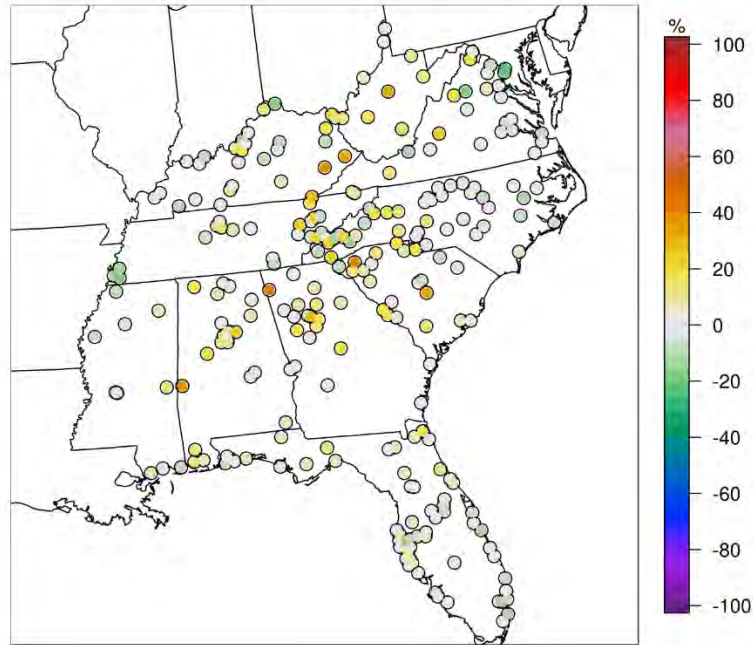
3



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-32, -9.6, -1.8, 7.6, 51]

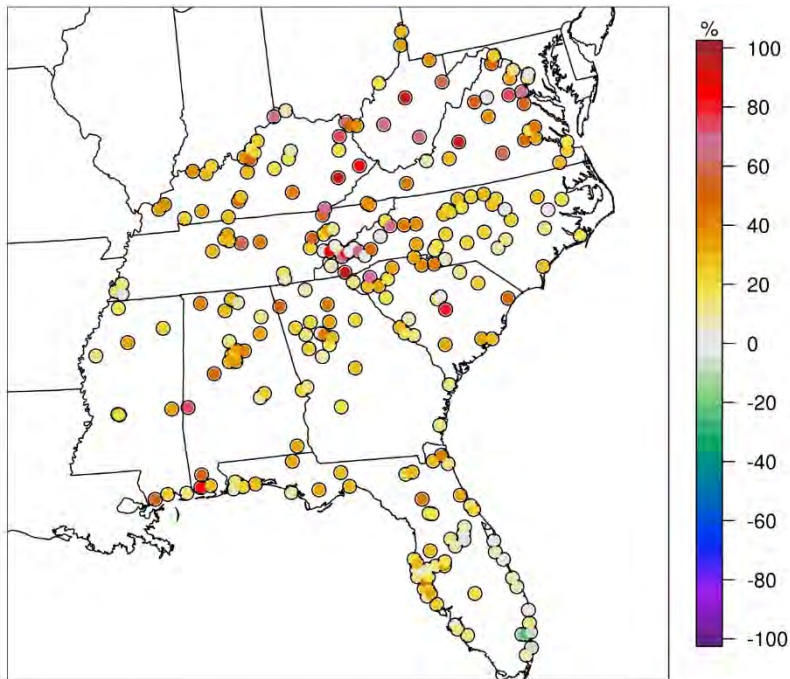
4

5 **Figure 3C-20. Normalized mean bias for MDA8 O₃ in the Southeastern U.S., winter 2016.**



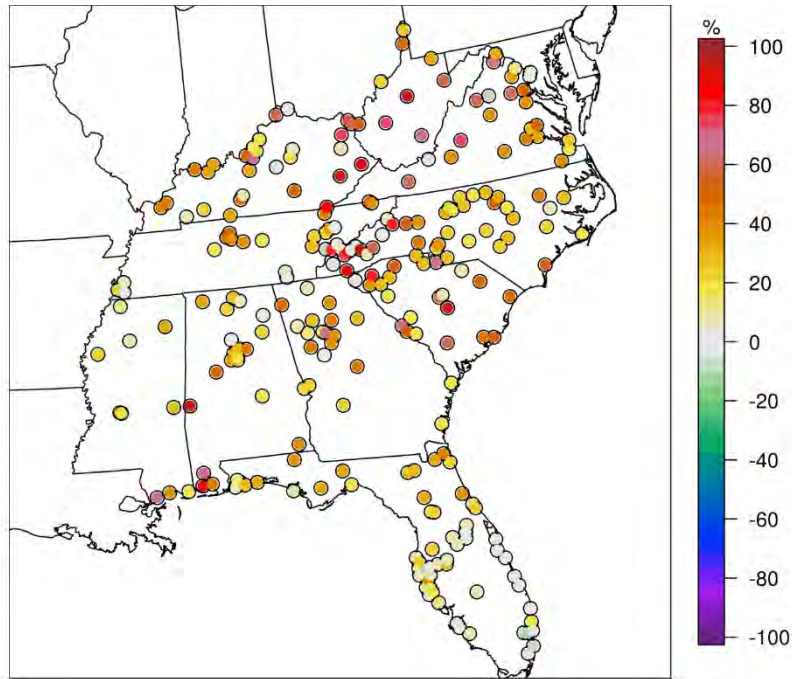
Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-18, -1.4, 5.1, 13, 44]

1
 2 **Figure 3C-21. Normalized mean bias for MDA8 O₃ in the Southeastern U.S., spring 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-22, 16, 27, 39, 130]

3
 4 **Figure 3C-22. Normalized mean bias for MDA8 O₃ in the Southeastern U.S., summer**
 5 **2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-12, 12, 29, 44, 120]

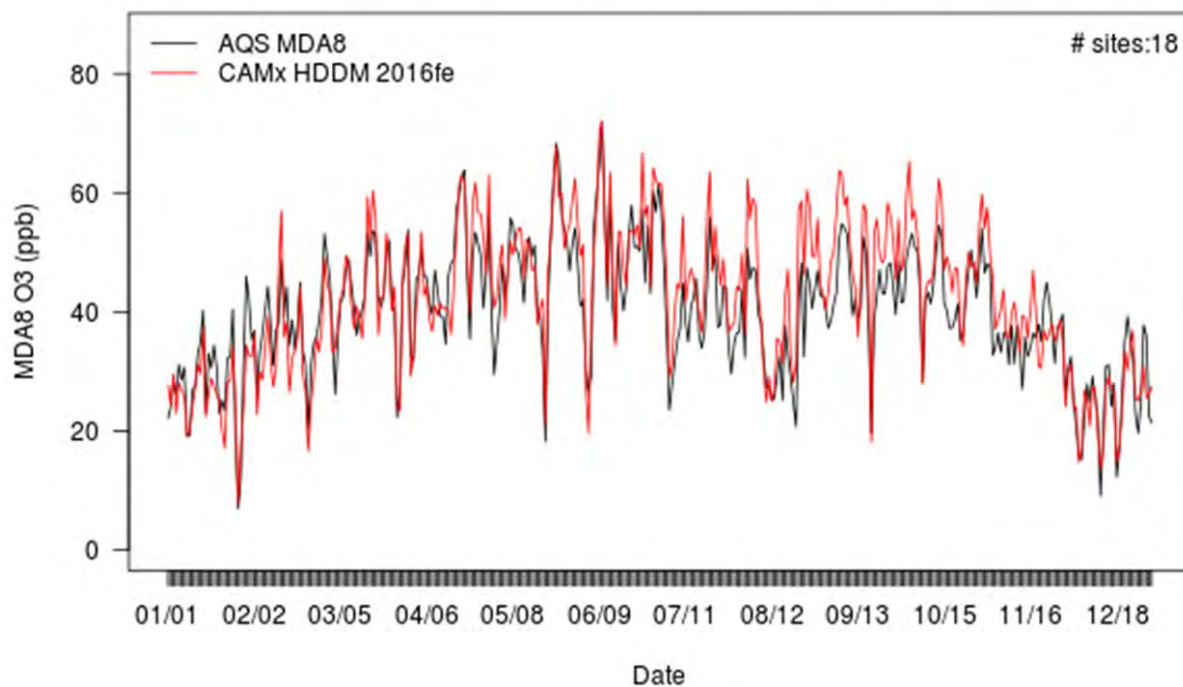
1
 2 **Figure 3C-23. Normalized mean bias for MDA8 O₃ in the Southeastern U.S., fall 2016.**

3
 4 **Table 3C-9. CAMx model performance at monitoring sites in the Atlanta study area.**

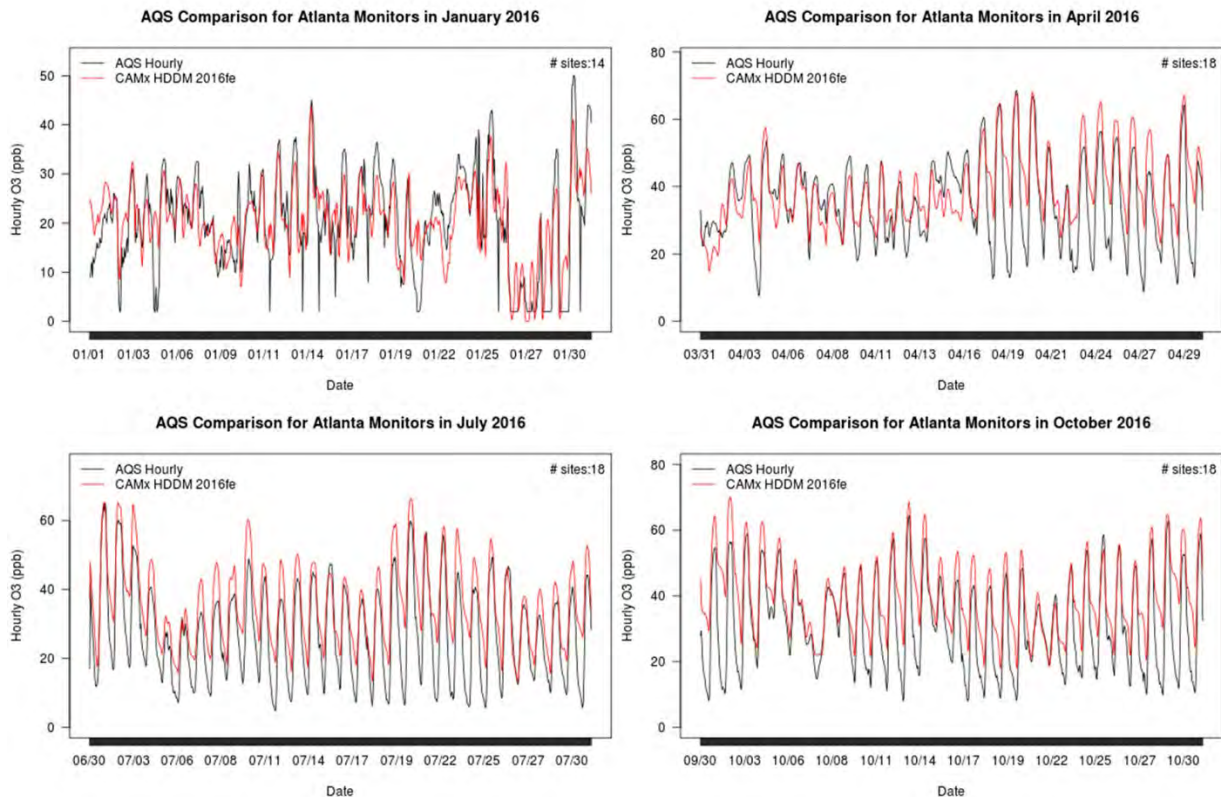
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	91	-0.9	-3.3	3.4	12.4
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	91	-0.9	-3.3	3.4	12.4
Spring	Days < 60	747	1.4	3.1	4.7	10.6
	Days ≥ 60	54	-1.4	-2.1	4.9	7.3
	All Days	801	1.2	2.6	4.7	10.3
Summer	Days < 60	717	5.4	13.4	6.9	17.1
	Days ≥ 60	93	-1.1	-1.6	6.0	8.9
	All Days	810	4.7	10.7	6.8	15.6
Fall	Days < 60	520	5.6	12.8	6.5	15.1
	Days ≥ 60	26	3.8	6.0	5.2	8.2
	All Days	546	5.5	12.4	6.5	14.6

5

AQS MDA8 Comparison for Atlanta Monitors in 2016



1
2 **Figure 3C-24. Time series of monitored (black) and modeled (red) MDA8 O₃ at Atlanta**
3 **monitoring sites in 2016.**



1
2 **Figure 3C-25. Time series of monitored (black) and modeled (red) hourly O₃**
3 **concentrations at Atlanta monitoring sites in January (top left), April (top**
4 **right), July (bottom left), and October (bottom right) 2016.**

6 **3C.4.2.3 Operational Evaluation in the Midwest U.S.**

7 Mean bias for MDA8 O₃ in the Midwest region was around 6 ppb or less at most sites for
8 all seasons (Table 3C-10), except for high O₃ days in spring. Normalized mean bias for MDA8
9 O₃ was less than 15%, except in the winter when it was somewhat higher (~20%). Normalized
10 mean error was lowest on high O₃ days in spring, summer, and fall, even though bias
11 performance was not notably better during these times. No distinct spatial patterns are apparent
12 from the maps of normalized mean bias (Figure 3C-26 through Figure 3C-29). Detroit was the
13 only one of the eight urban study areas located in the Midwest.

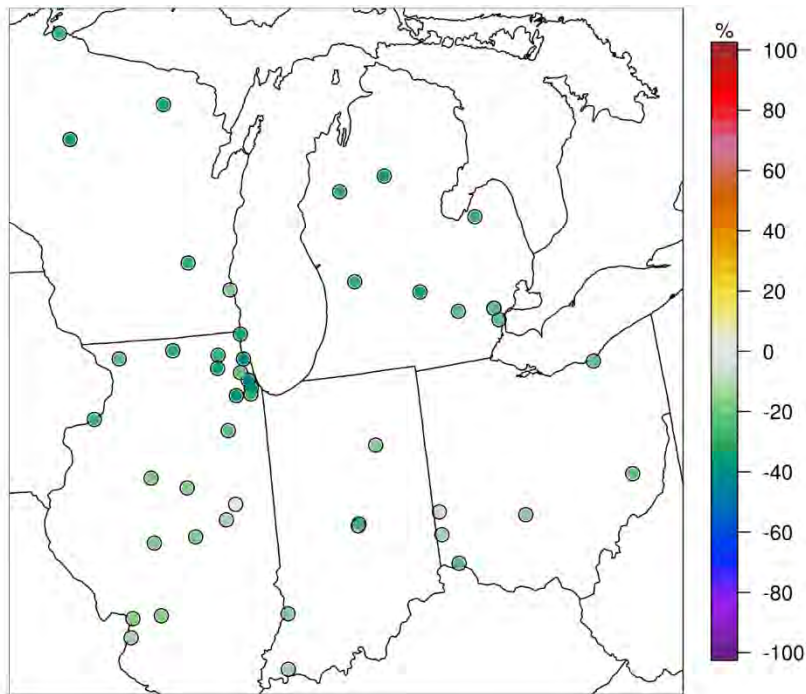
14 Detroit performance statistics for MDA8 O₃ were similar to those from the rest of the
15 Midwest. However, under-estimates on high O₃ days were more pronounced in Detroit than in
16 the rest of the region. The time series shows that the model accurately estimates both day and
17 nighttime hourly O₃ in Detroit in April and July and generally captures the variations in MDA8
18 O₃ throughout the year, although the persistent under-estimate in winter-spring is evident (Figure
19 3C-30, Figure 3C-31).

1
2

Table 3C-10. CAMx model performance at monitoring sites in the Midwest U.S.

Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	1775	-5.8	-20.2	6.4	22.4
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	1775	-5.8	-20.2	6.4	22.4
Spring	Days < 60	3635	-5.9	-14.1	7.6	18.1
	Days ≥ 60	370	-8.3	-12.5	9.2	14.0
	All Days	4005	-6.1	-13.9	7.8	17.6
Summer	Days < 60	4680	3.3	7.8	7.4	17.8
	Days ≥ 60	556	-4.9	-7.3	8.6	12.8
	All Days	5236	2.4	5.4	7.6	17.0
Fall	Days < 60	3439	2.2	6.7	5.1	15.3
	Days ≥ 60	51	3.3	5.1	5.6	8.6
	All Days	3490	2.3	6.7	5.1	15.1

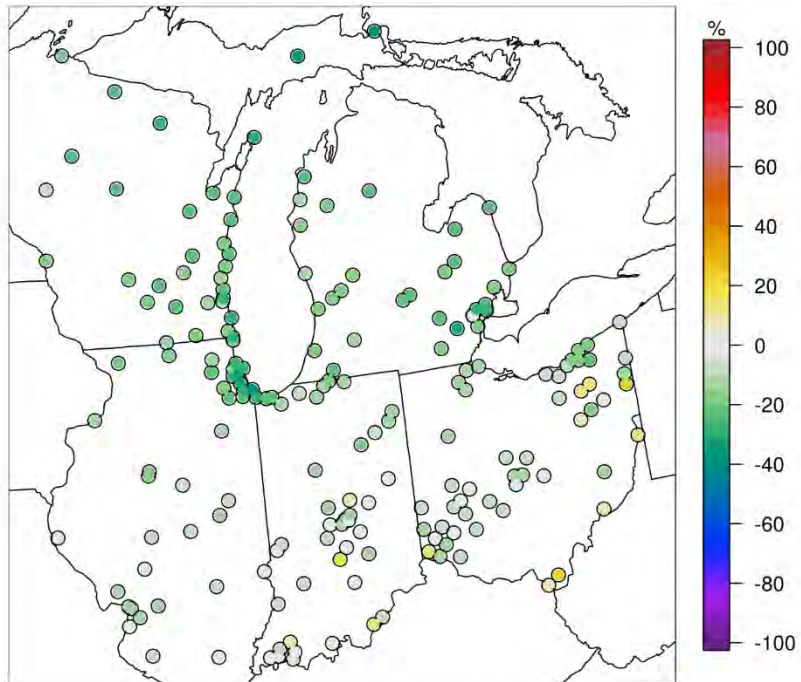
3



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-44, -27, -22, -16, -0.96]

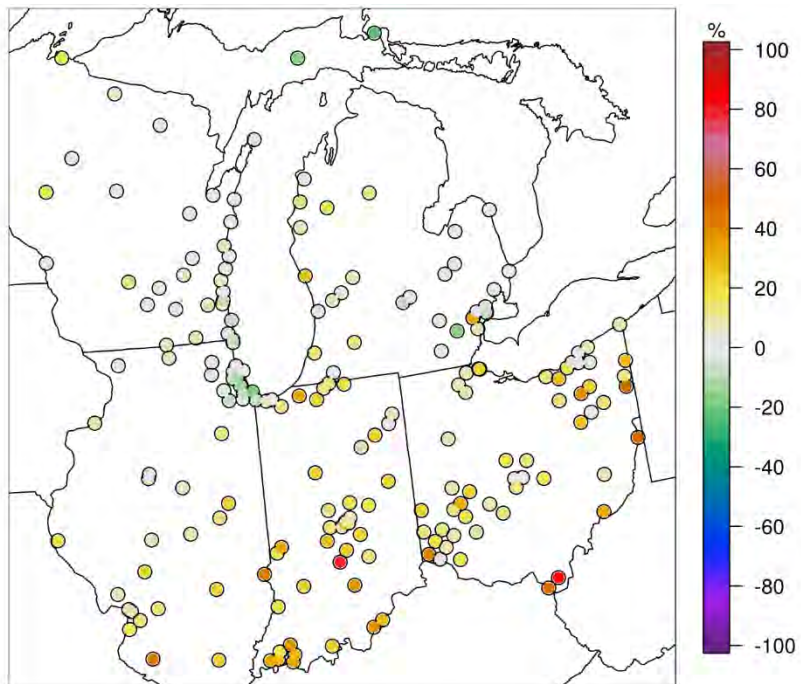
4

Figure 3C-26. Normalized mean bias for MDA8 O₃ in the Midwest U.S., winter 2016.



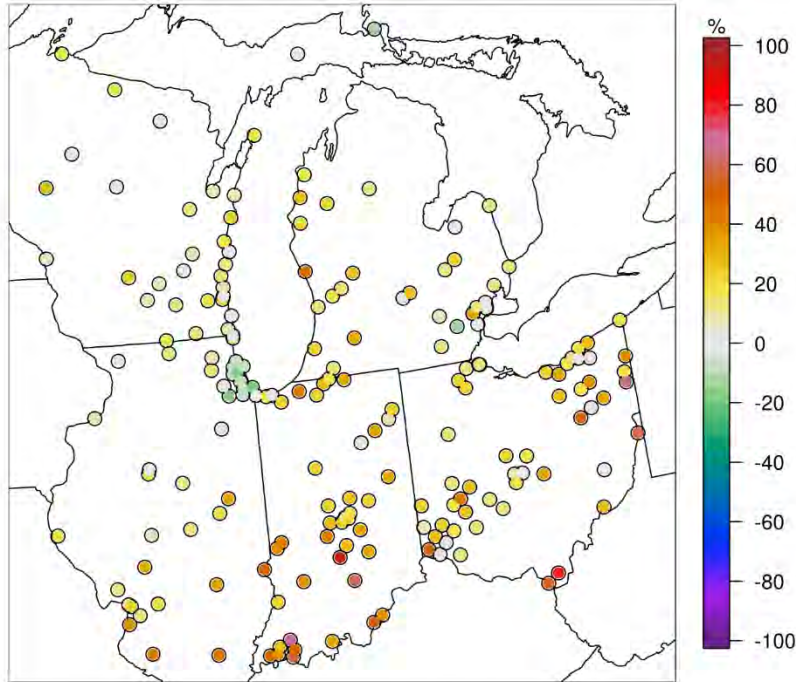
Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-38, -18, -11, -4, 27]

1
 2 **Figure 3C-27. Normalized mean bias for MDA8 O₃ in the Midwest U.S., spring 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-20, 4.4, 11, 22, 87]

3
 4 **Figure 3C-28. Normalized mean bias for MDA8 O₃ in the Midwest U.S., summer 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-14, 10, 20, 31, 130]

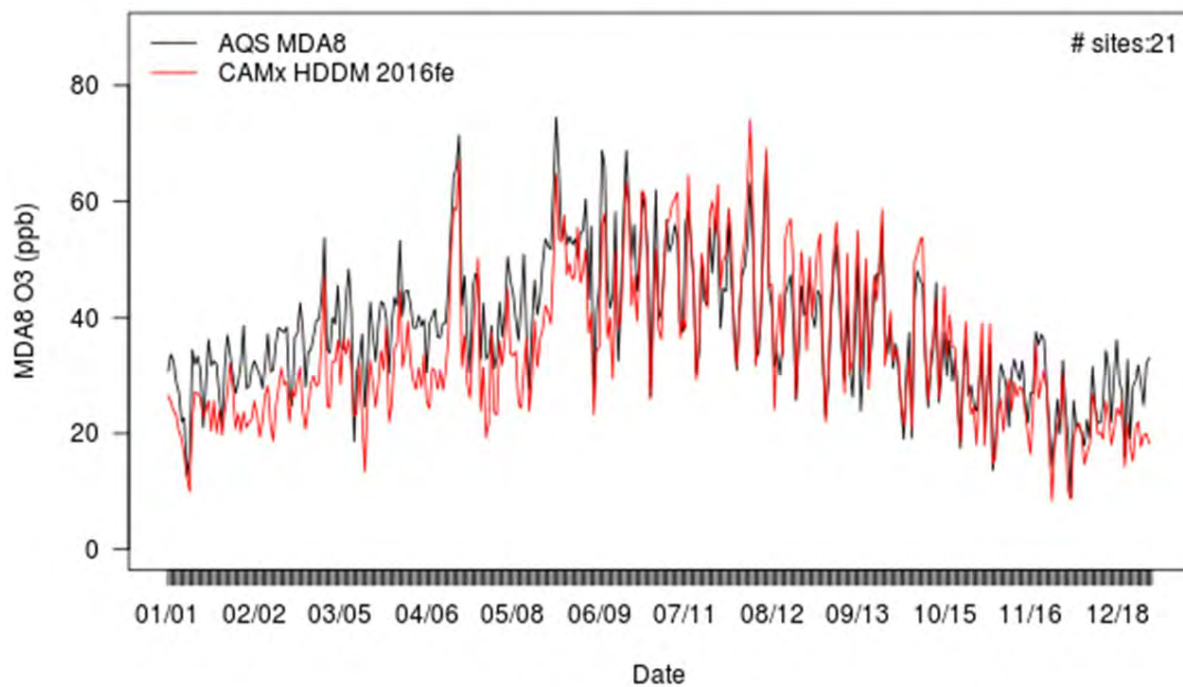
1
 2 **Figure 3C-29. Normalized mean bias for MDA8 O₃ in the Midwest U.S., fall 2016.**

3
 4 **Table 3C-11. CAMx model performance at monitoring sites in the Detroit study area.**

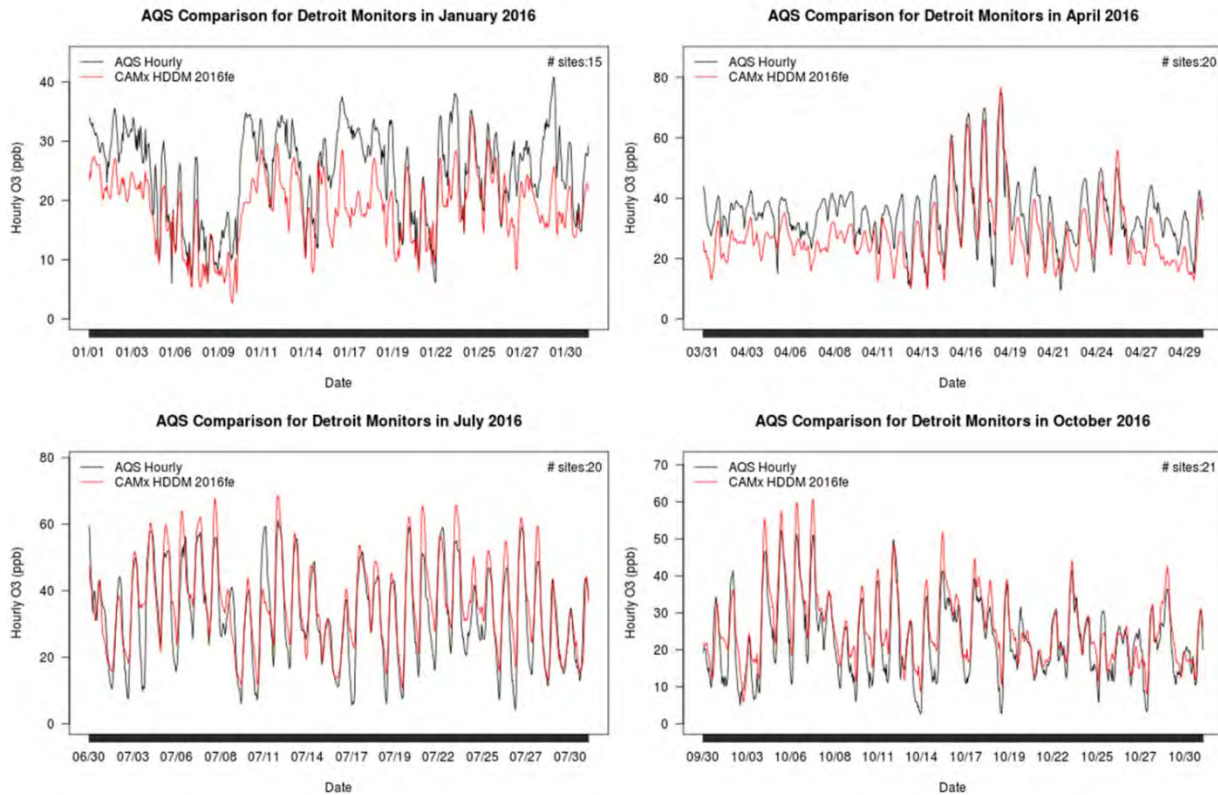
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	29	-4.1	-19.5	5.9	26.3
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	29	-4.1	-19.5	5.9	26.3
Spring	Days < 60	337	-6.5	-15.8	8.3	20.0
	Days ≥ 60	28	-9.4	-13.5	10.0	14.4
	All Days	365	-6.7	-15.5	8.4	19.3
Summer	Days < 60	485	2.0	4.7	6.8	16.1
	Days ≥ 60	59	-5.3	-8.1	7.9	12.1
	All Days	544	1.2	2.7	6.9	15.5
Fall	Days < 60	245	3.1	9.7	5.6	17.2
	Days ≥ 60	3	-4.1	-6.7	4.1	6.7
	All Days	248	3.0	9.3	5.5	17.0

5

AQS MDA8 Comparison for Detroit Monitors in 2016



1
2 **Figure 3C-30. Time series of monitored (black) and modeled (red) MDA8 O₃ at Detroit**
3 **monitoring sites in 2016.**



1
 2 **Figure 3C-31. Time series of monitored (black) and modeled (red) hourly O₃**
 3 **concentrations at Detroit monitoring sites in January (top left), April (top**
 4 **right), July (bottom left), and October (bottom right) 2016.**

6 **3C.4.2.4 Operational Evaluation in the Central U.S.**

7 Mean bias for MDA8 O₃ concentrations in the Central U.S. is within 4 ppb, except for
 8 high days in winter (-6 ppb) and spring (-7 ppb) (Table 3C-12). Normalized mean error is within
 9 15%, except for days < 60 ppb in winter and summer (~18%). Spatial maps of normalized mean
 10 bias are shown in Figure 3C-32 through Figure 3C-35. Overall performance is best on lower O₃
 11 days in spring and high O₃ days in summer and fall. St. Louis and Dallas were the only two of
 12 the eight study areas which are located in the Central U.S. region.

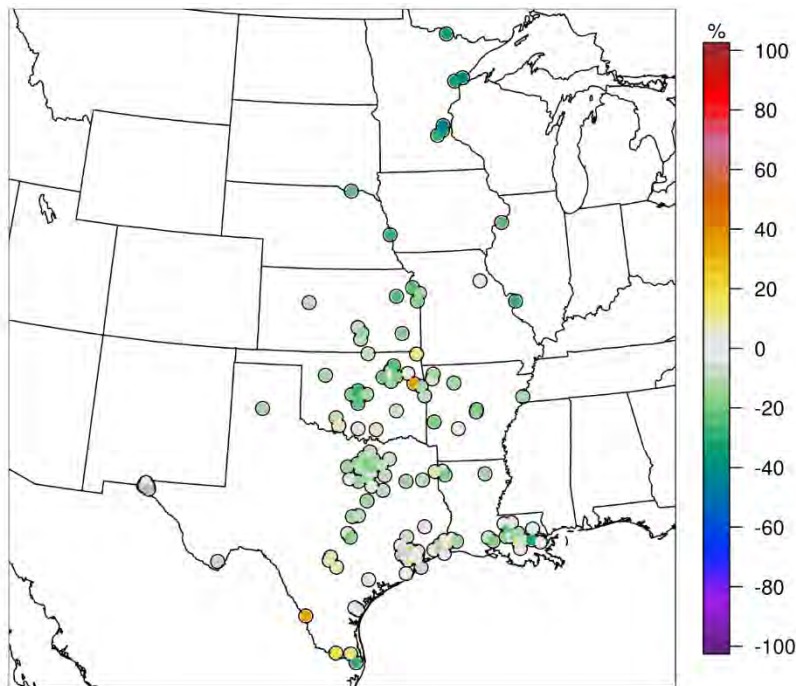
13 St. Louis mean bias for MDA8 was within 5 ppb for all days and seasons. A north-south
 14 gradient in NMB is apparent during both the winter and spring seasons in the maps shown in
 15 Figure 3C-32 and Figure 3C-33, with larger underestimates visible at higher latitude/more
 16 northerly monitors. Overall performance for St. Louis was best on high O₃ days in summer. The
 17 MDA8 time series shows reasonable agreement between CAMx and the monitor data for most of
 18 the year (Figure 3C-36), with underestimates in January and overestimates in July also apparent
 19 in the hourly time series (Figure 3C-37).

1 Performance statistics for MDA8 O₃ in Dallas were better than those for the broader
 2 region, with mean bias less than 5 ppb and normalized mean error just at or below 15% for all
 3 days and seasons. The MDA8 and hourly time series also show excellent model performance,
 4 with slightly underestimated peak day time O₃ in January (Figure 3C-38, Figure 3C-39).
 5 Overestimates of night-time O₃ in April and October, although these overpredictions are less
 6 pronounced in Dallas compared to many of the other urban study areas examined in the
 7 assessment.

8 **Table 3C-12. CAMx model performance at monitoring sites in the Central U.S.**

Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	4550	-4.0	-12.2	5.8	18.0
	Days ≥ 60	7	-5.7	-9.2	9.1	14.5
	All Days	4557	-4.0	-12.2	5.8	18.0
Spring	Days < 60	7086	-1.7	-3.9	6.2	14.4
	Days ≥ 60	324	-7.0	-10.9	7.8	12.2
	All Days	7410	-1.9	-4.3	6.2	14.3
Summer	Days < 60	8234	3.8	9.6	7.0	17.9
	Days ≥ 60	346	-2.7	-4.2	7.0	10.8
	All Days	8580	3.5	8.7	7.0	17.4
Fall	Days < 60	7109	2.6	7.4	5.1	14.6
	Days ≥ 60	124	-1.8	-2.8	5.3	8.2
	All Days	7233	2.5	7.1	5.1	14.4

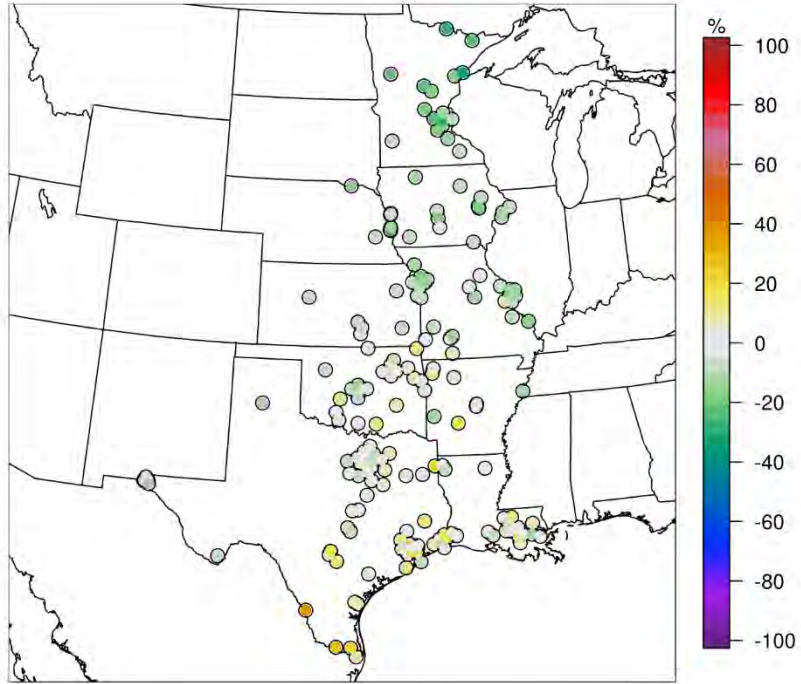
9



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-44, -13, -7.4, 1.6, 36]

10

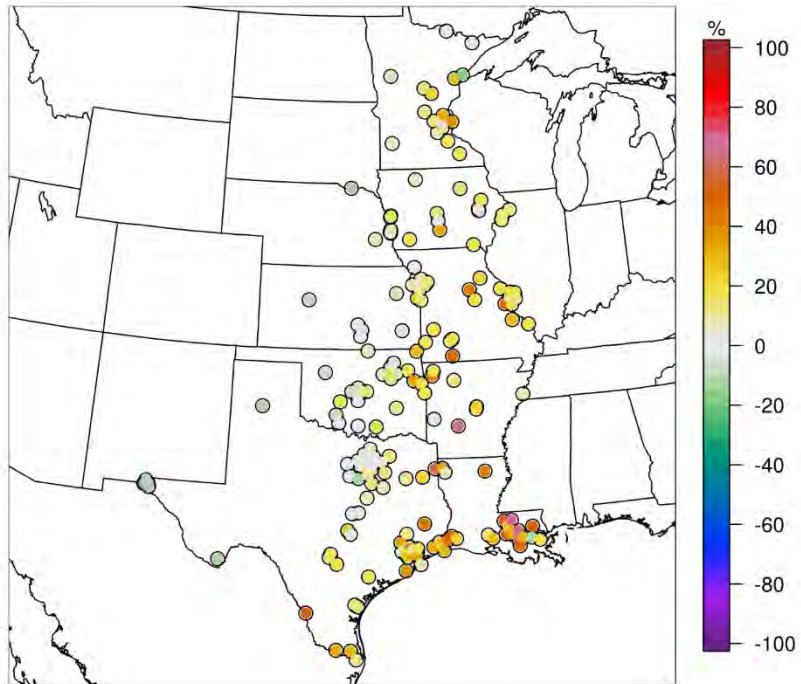
11 **Figure 3C-32. Normalized mean bias for MDA8 O₃ in the Central U.S., winter 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-28, -7.6, -0.93, 6.6, 41]

1

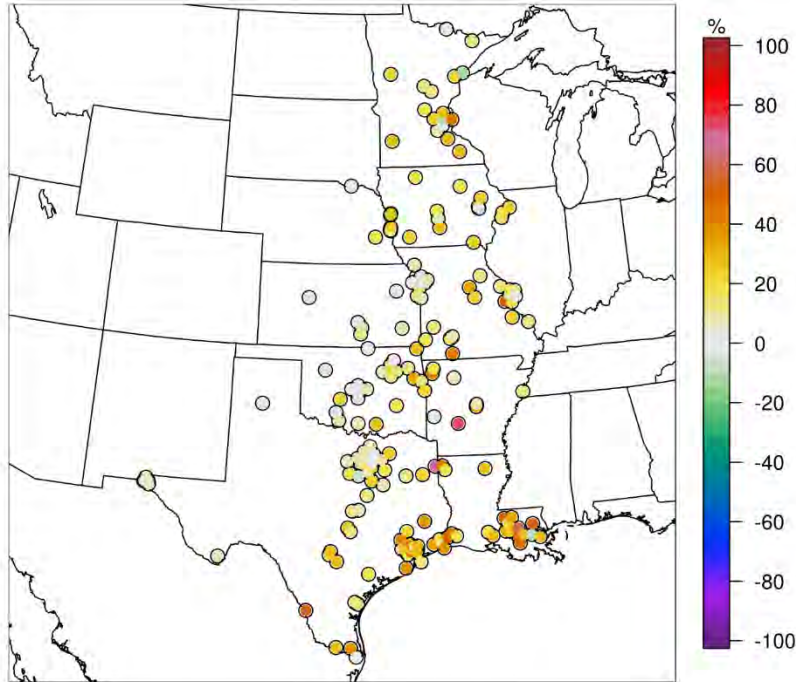
2 **Figure 3C-33. Normalized mean bias for MDA8 O₃ in the Central U.S., spring 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-21, 6.9, 16, 24, 72]

3

4 **Figure 3C-34. Normalized mean bias for MDA8 O₃ in the Central U.S., summer 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-9.1, 9.8, 19, 28, 75]

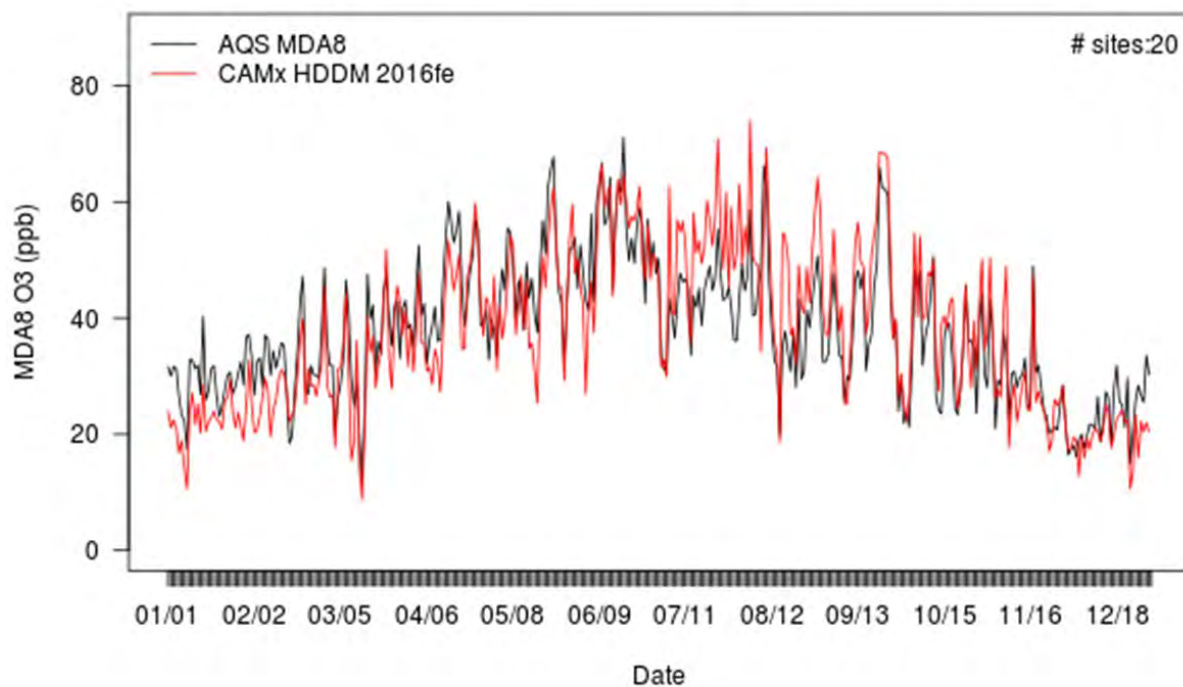
1
 2 **Figure 3C-35. Normalized mean bias for MDA8 O₃ in the Central U.S., fall 2016.**

3
 4 **Table 3C-13. CAMx model performance at monitoring sites in the Saint Louis study area.**

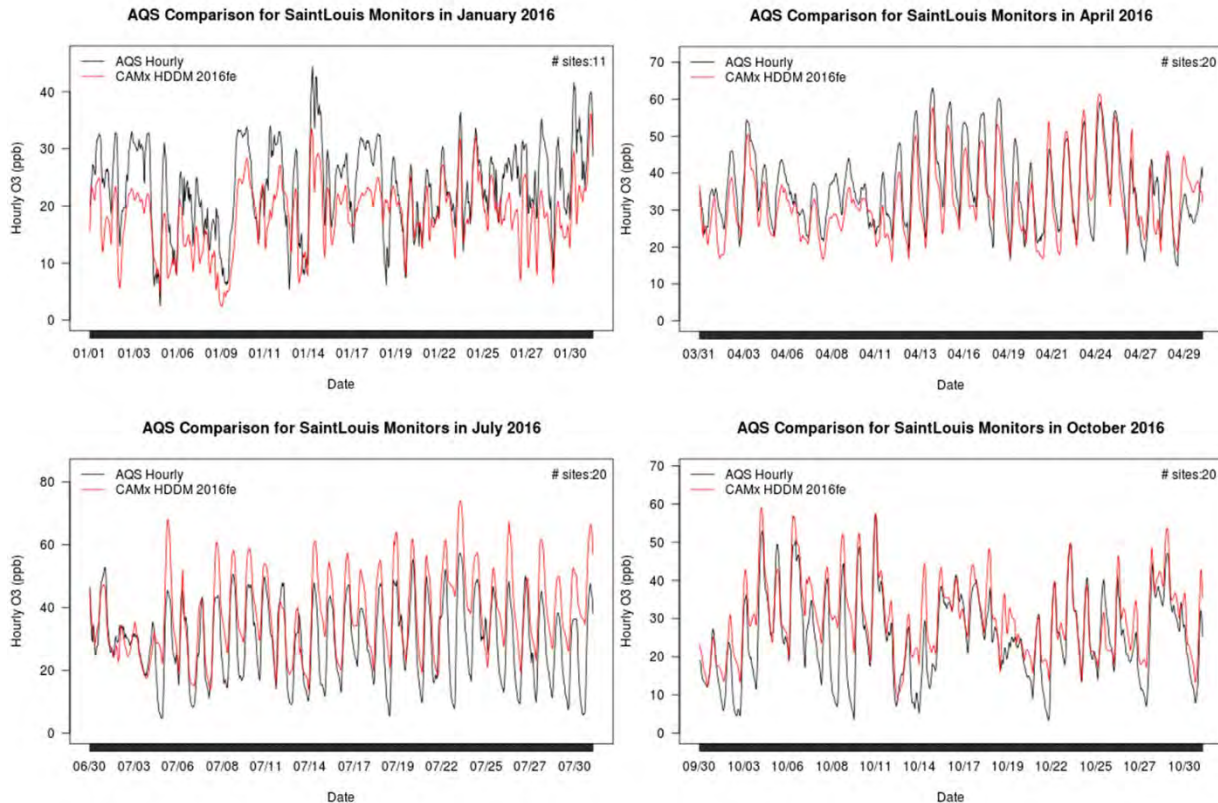
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	181	-5.9	-20.9	6.5	23.1
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	181	-5.9	-20.9	6.5	23.1
Spring	Days < 60	756	-3.5	-7.8	6.1	13.7
	Days ≥ 60	63	-7.2	-11.2	7.3	11.3
	All Days	819	-3.7	-8.1	6.2	13.4
Summer	Days < 60	1061	5.8	13.7	8.4	19.6
	Days ≥ 60	121	-1.1	-1.6	8.1	12.1
	All Days	1182	5.1	11.4	8.4	18.5
Fall	Days < 60	773	3.9	11.1	5.7	16.1
	Days ≥ 60	35	3.5	5.1	5.0	7.3
	All Days	808	3.9	10.6	5.7	15.4

5

AQS MDA8 Comparison for SaintLouis Monitors in 2016



1
2 **Figure 3C-36. Time series of monitored (black) and modeled (red) MDA8 O₃ at St. Louis**
3 **monitoring sites in 2016.**



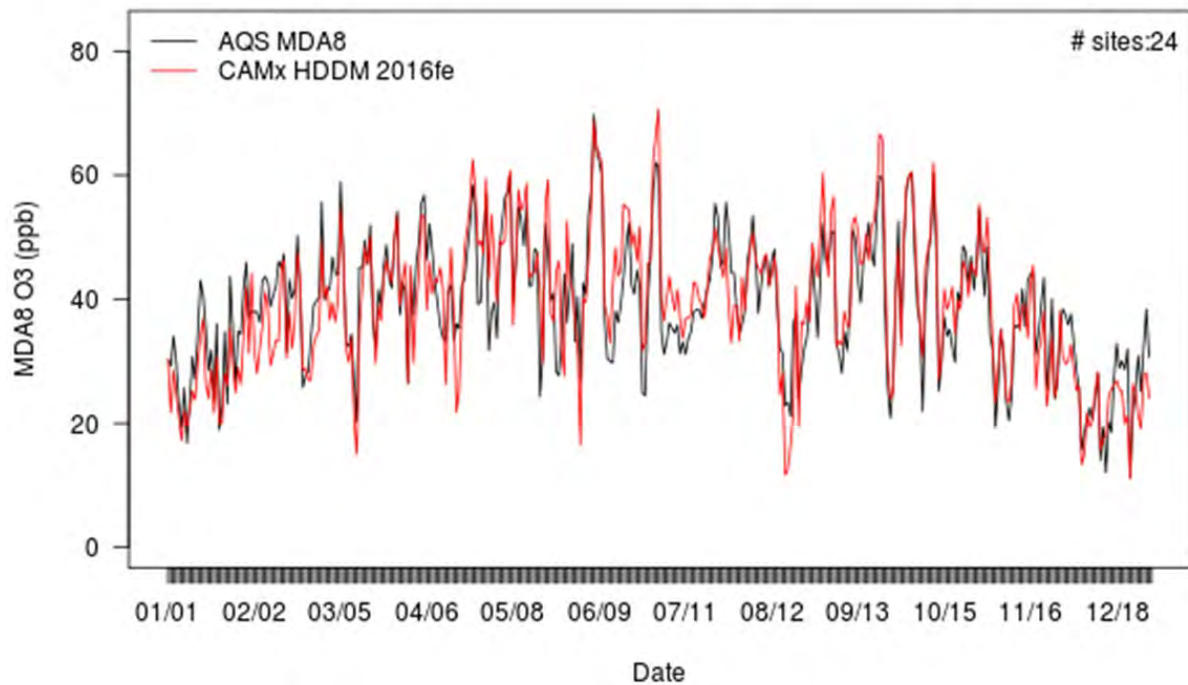
1
2 **Figure 3C-37. Time series of monitored (black) and modeled (red) hourly O₃**
3 **concentrations at St. Louis monitoring sites in January (top left), April (top**
4 **right), July (bottom left), and October (bottom right) 2016.**

5
6 **Table 3C-14. CAMx model performance at monitoring sites in the Dallas study area.**

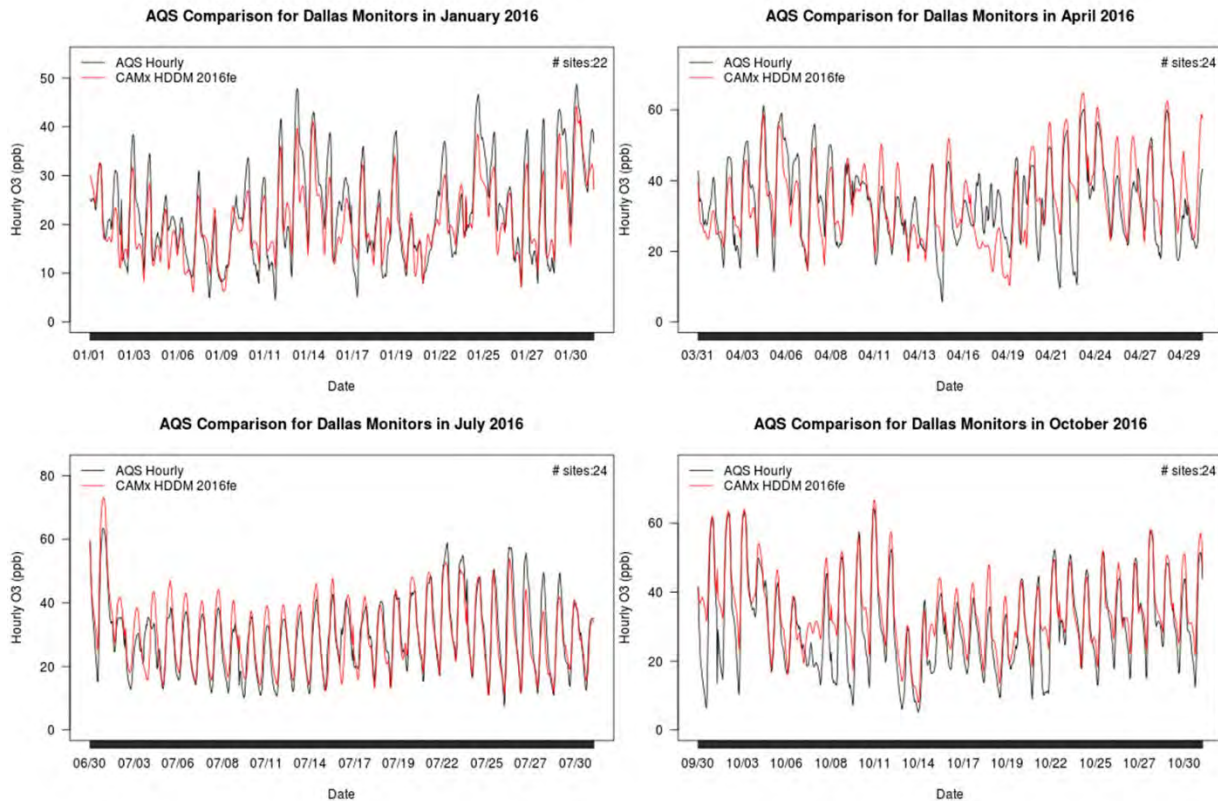
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	625	-3.2	-9.9	4.8	14.9
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	625	-3.2	-9.9	4.8	14.9
Spring	Days < 60	697	0.8	1.8	5.8	13.5
	Days ≥ 60	21	-4.9	-7.7	5.4	8.6
	All Days	718	0.6	1.4	5.7	13.3
Summer	Days < 60	700	2.1	5.4	5.9	15.4
	Days ≥ 60	25	-2.8	-4.0	6.5	9.4
	All Days	725	1.9	4.8	5.9	15.1
Fall	Days < 60	697	1.4	3.7	4.5	11.9
	Days ≥ 60	23	-3.6	-5.5	4.7	7.1
	All Days	720	1.3	3.2	4.5	11.6

7

AQS MDA8 Comparison for Dallas Monitors in 2016



1
2 **Figure 3C-38. Time series of monitored (black) and modeled (red) MDA8 O₃ at Dallas**
3 **monitoring sites in 2016.**



1
 2 **Figure 3C-39. Time series of monitored (black) and modeled (red) hourly O₃**
 3 **concentrations at Dallas monitoring sites in January (top left), April (top**
 4 **right), July (bottom left), and October (bottom right) 2016.**

5
 6 **3C.4.2.5 Operational Evaluation in the Western U.S.**

7 Model statistics for MDA8 O₃ in the Western U.S. are best on low O₃ days in summer
 8 and fall (Table 3C-15). High wintertime observations were substantially underestimated by the
 9 model with an average MB of -26 but likely for different reasons. The high days in Riverside
 10 California are probably due to traditionally understood O₃ formation that occurs on warm sunny
 11 days. The high O₃ concentrations in Wyoming are an example of wintertime O₃ formation that
 12 occurs during cold pool meteorology events which have substantial snow cover and extreme
 13 temperature inversions and are still an active area of research. Some spatial patterns in
 14 normalized mean bias are apparent in the winter and in the summer (Figure 3C-40 through
 15 Figure 3C-43), with overestimates on the West Coast and underestimates in the Intermountain
 16 West. Two urban study areas are located in the Western U.S. and are evaluated in this section:
 17 Sacramento and Phoenix.

18 The model performance for MDA8 O₃ values in the Sacramento study area was best on
 19 lower O₃ days in summer and fall (Figure 3C-44). In Sacramento there were no days during the

1 winter with measured MDA8 O₃ > 60 ppb. Normalized mean error is at or below 15% for all
 2 seasons except winter. Hourly time series show good agreement in Sacramento, except for winter
 3 when the model does not capture very much of the day to day variability in O₃ concentrations
 4 Figure 3C-45).

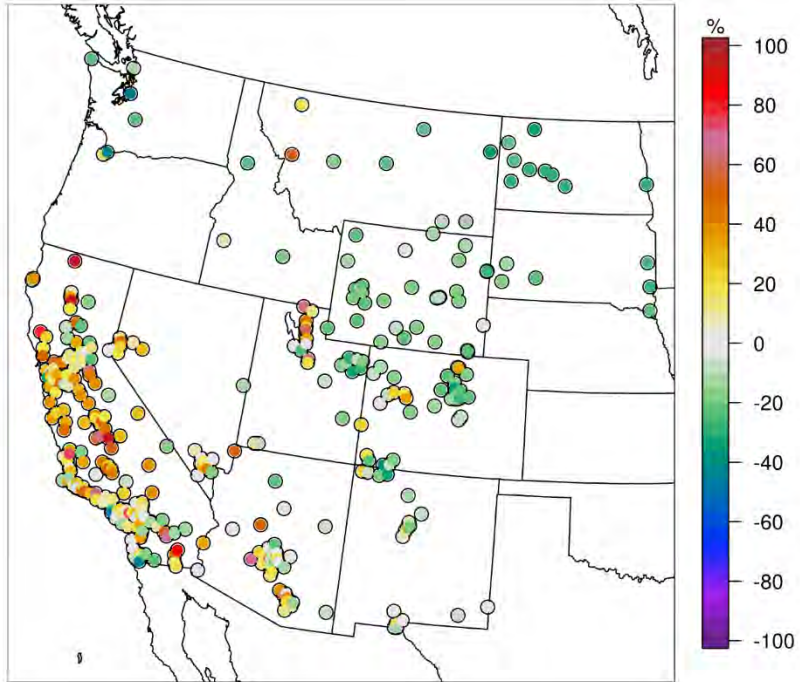
5 While normalized mean error was at or less than 15% in Phoenix on all days in all
 6 seasons, the MDA8 time series shows frequent underestimates in winter-spring as well as
 7 overestimates in summer-fall (Figure 3C-46). The hourly time series also show that though the
 8 model captures some of the overnight O₃ patterns in Phoenix, night time O₃ is significantly
 9 overestimated, particularly in January and October (Figure 3C-47).

10

11 **Table 3C-15. CAMx model performance at monitoring sites in the Western U.S.**

		No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	15888	-2.8	-8.2	6.0	18.1
	Days ≥ 60	113	-25.8	-35.7	25.8	35.7
	All Days	16001	-2.9	-8.7	6.2	18.4
Spring	Days < 60	15789	-4.6	-10.3	6.5	14.6
	Days ≥ 60	1471	-9.5	-14.7	10.0	15.4
	All Days	17260	-5.0	-10.8	6.8	14.7
Summer	Days < 60	13254	1.2	2.6	6.7	14.9
	Days ≥ 60	4461	-6.6	-9.5	9.5	13.7
	All Days	17715	-0.8	-1.6	7.4	14.5
Fall	Days < 60	15975	0.7	1.9	5.4	14.5
	Days ≥ 60	795	-9.2	-13.6	10.7	15.8
	All Days	16770	0.2	0.6	5.6	14.6

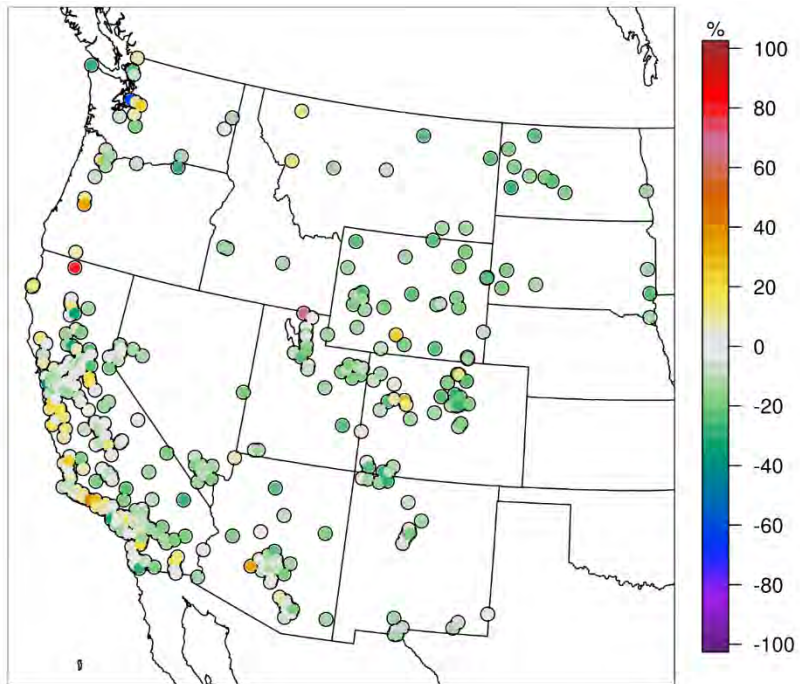
12



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-47, -12, 8.3, 27, 110]

1

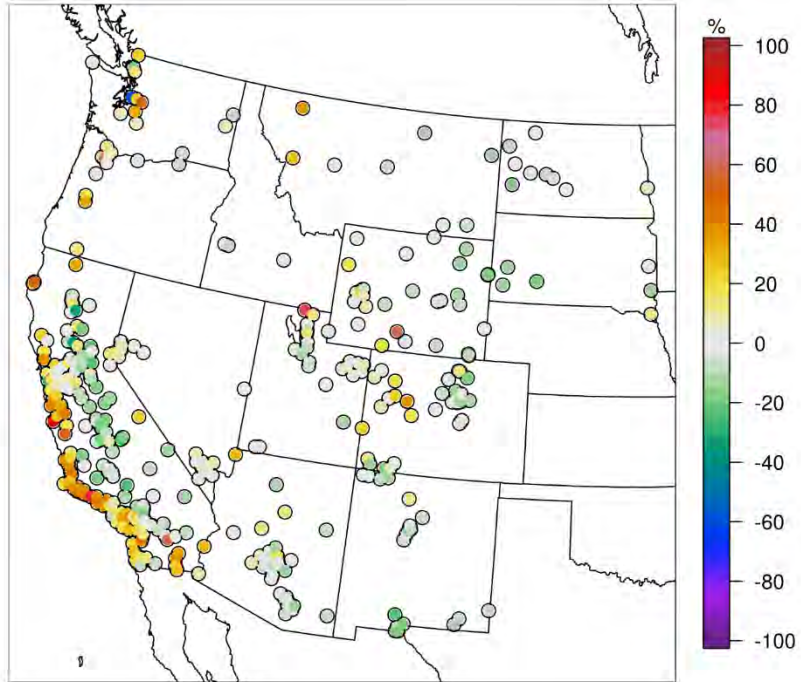
2 **Figure 3C-40. Normalized mean bias for MDA8 O₃ in the Western U.S., winter 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-60, -12, -5.7, 3.1, 82]

3

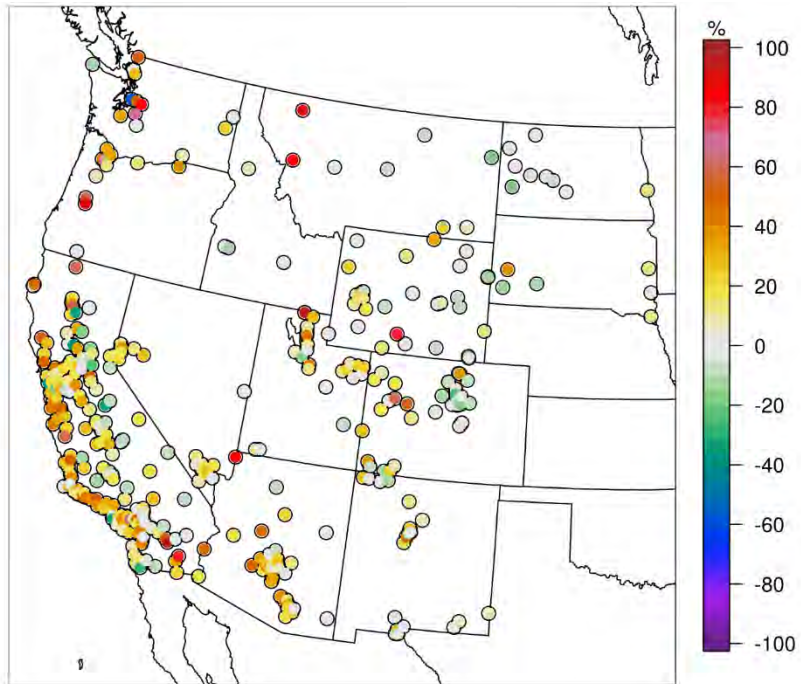
4 **Figure 3C-41. Normalized mean bias for MDA8 O₃ in the Western U.S., spring 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-55, -5.2, 3.2, 16, 90]

1

2 **Figure 3C-42. Normalized mean bias for MDA8 O₃ in the Western U.S., summer 2016.**



Bias Summary: [min, 25th %, 50th %, 75th %, max]
 [-57, 1.5, 16, 30, 120]

3

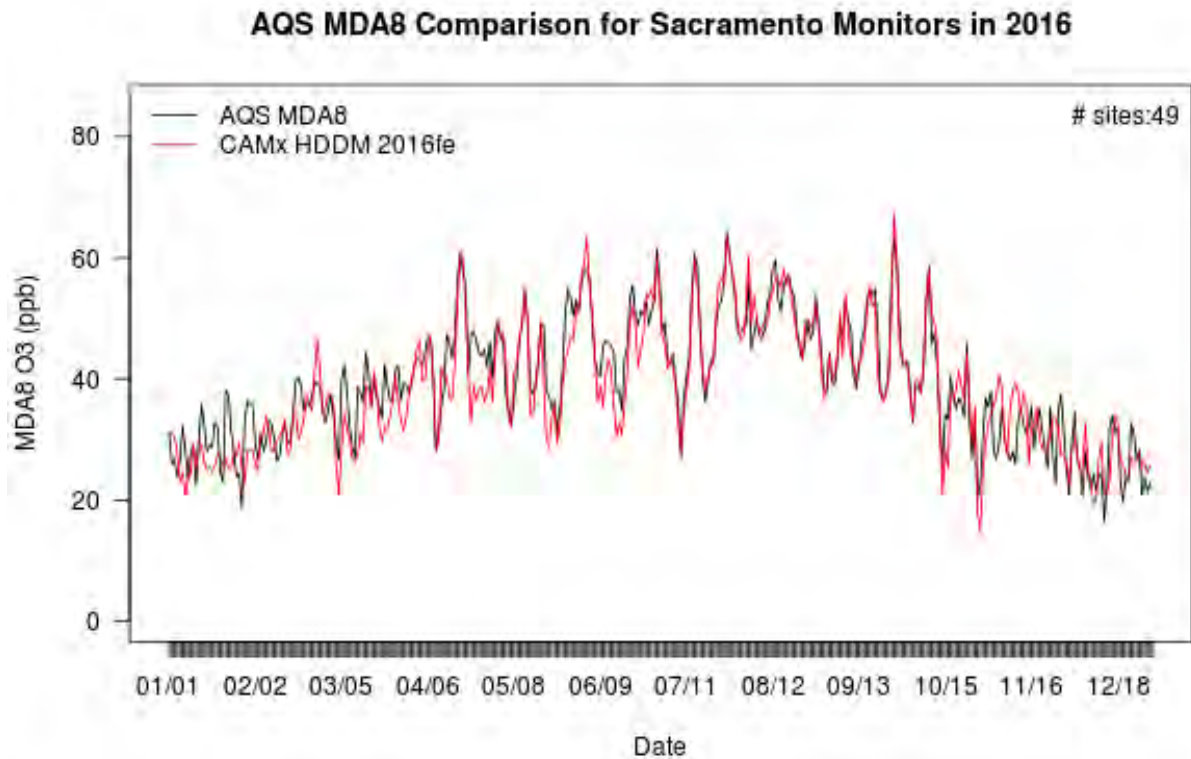
4 **Figure 3C-43. Normalized mean bias for MDA8 O₃ in the Western U.S., fall 2016.**

1
2

Table 3C-16. CAMx model performance at monitoring sites in the Sacramento study area.

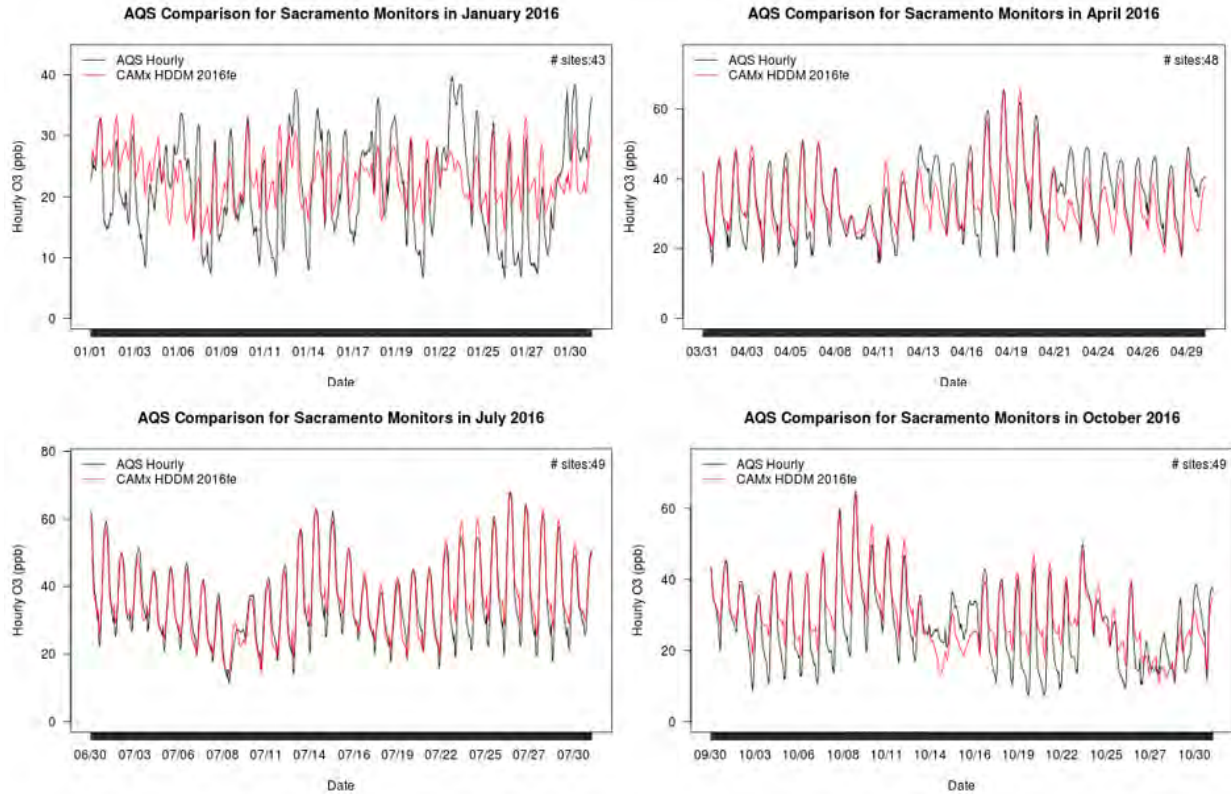
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	2359	-0.9	-3.2	5.5	18.9
	Days ≥ 60	0	NA	NA	NA	NA
	All Days	2359	-0.9	-3.2	5.5	18.9
Spring	Days < 60	2474	-3.2	-7.9	5.6	13.6
	Days ≥ 60	116	-8.1	-12.6	9.4	14.6
	All Days	2590	-3.5	-8.2	5.8	13.7
Summer	Days < 60	2157	0.6	1.3	5.8	13.7
	Days ≥ 60	628	-7.3	-10.8	8.8	13.0
	All Days	2785	-1.2	-2.5	6.5	13.5
Fall	Days < 60	2503	0.5	1.3	5.5	15.2
	Days ≥ 60	160	-7.7	-11.2	10.0	14.7
	All Days	2663	0.0	0.0	5.7	15.1

3



4
5
6

Figure 3C-44. Time series of monitored (black) and modeled (red) MDA8 O₃ at Sacramento monitoring sites in 2016.



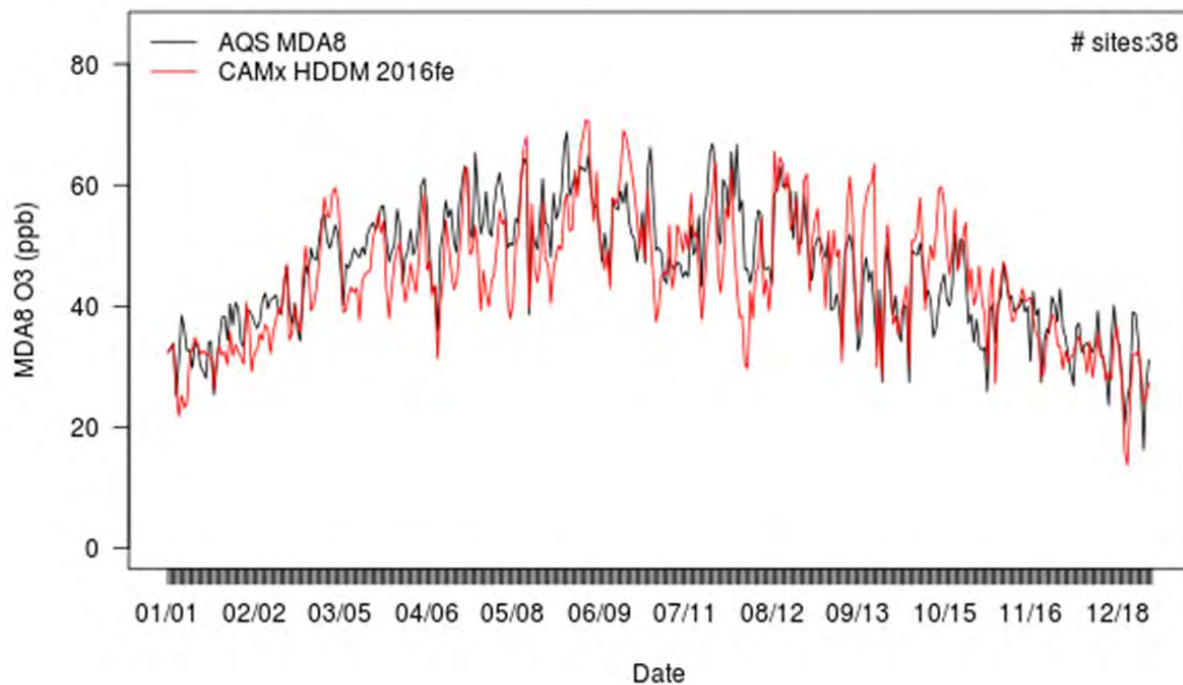
1
2 **Figure 3C-45. Time series of monitored (black) and modeled (red) hourly O₃**
3 **concentrations at Sacramento monitoring sites in January (top left), April**
4 **(top right), July (bottom left), and October (bottom right) 2016.**

5
6 **Table 3C-17. CAMx model performance at monitoring sites in the Phoenix study area.**

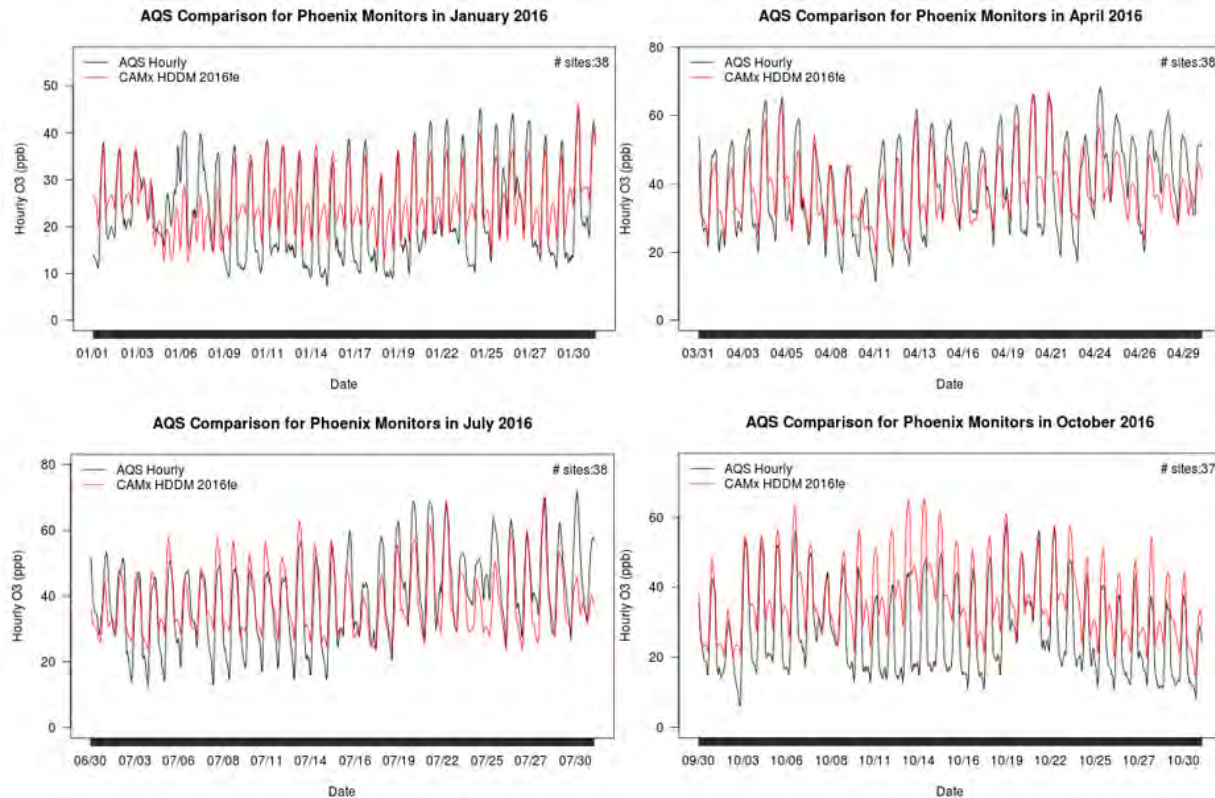
Season	MDA8 level (ppb)	No. of obs	MB (ppb)	NMB (%)	ME (ppb)	NME (%)
Winter	Days < 60	1292	-3.5	-9.8	5.3	15.0
	Days ≥ 60	3	-5.9	-9.7	5.9	9.7
	All Days	1295	-3.5	-9.8	5.3	14.9
Spring	Days < 60	265	-5.6	-10.9	6.8	13.3
	Days ≥ 60	1082	-8.5	-13.3	9.6	14.9
	All Days	1347	-6.2	-11.5	7.4	13.7
Summer	Days < 60	974	-2.1	-4.2	6.5	13.0
	Days ≥ 60	346	-4.7	-7.3	8.5	13.0
	All Days	1320	-2.8	-5.2	7.1	13.0
Fall	Days < 60	1278	2.6	6.7	6.1	15.4
	Days ≥ 60	5	-3.8	-6.2	5.4	8.7
	All Days	1283	2.6	6.6	6.1	15.4

7

AQS MDA8 Comparison for Phoenix Monitors in 2016



1
2 **Figure 3C-46. Time series of monitored (black) and modeled (red) MDA8 O₃ at Phoenix**
3 **monitoring sites in 2016.**



1
2 **Figure 3C-47. Time series of monitored (black) and modeled (red) hourly O₃**
3 **concentrations at Phoenix monitoring sites in January (top left), April (top**
4 **right), July (bottom left), and October (bottom right) 2016.**

5
6 **3C.5 AIR QUALITY ADJUSTMENT TO MEET CURRENT AND**
7 **ALTERNATIVE AIR QUALITY SCENARIOS**

8 **3C.5.1 Overview of the Higher Order Direct Decoupled Method (HDDM)**

9 In this section we present a model-based O₃ adjustment methodology that allows for
10 adjustments to observed hourly O₃ concentrations to reflect the expected impacts of changes in
11 NO_x emissions. This methodology uses the CAMx model, described above in section 3C.4,
12 instrumented with the Higher order Decoupled Direct Method (HDDM) - a tool that generates
13 modeled sensitivities of O₃ to emissions changes. The outputs of the HDDM are used to estimate
14 the distribution of O₃ concentrations associated with just meeting three air quality scenarios (O₃
15 monitor design values of 75 ppb, 70 ppb, and 65 ppb) within multiple urban study areas. The
16 HDDM sensitivities are applied to ambient air measurements of O₃ to estimate how O₃
17 concentrations would respond to changes in U.S. anthropogenic emissions. This approach, based
18 on Simon et al. (2013), was applied previously for the 2015 O₃ NAAQS review.

1 The CAMx photochemical modeling incorporates emissions from non-anthropogenic
2 sources and anthropogenic emissions from sources in the U.S and in the portions of Canada and
3 Mexico within the regional modeling domain. Pollution from sources in other locations within
4 and outside of North America is included as transport into the boundary of the modeling domain.

5 3C.5.1.1 Capabilities

6 Chemical transport models, such as CAMx, simulates physical and chemical processes in
7 the atmosphere to predict 3-dimensional (3-D) gridded pollutant concentrations. These models
8 account for the impacts of emissions, transport, chemistry, and deposition on spatially and
9 temporally varying pollutant concentrations. Required model inputs include time-varying
10 emissions and meteorology fields, time varying concentrations of pollutants at the boundaries of
11 the model domain (i.e. boundary conditions), and a characterization of the 3-D field of chemical
12 concentrations to initialize the model (i.e. initial conditions).

13 Beyond modeling the ambient air concentrations of O₃, chemical transport models can be
14 used to estimate the response of ambient air O₃ concentrations to changes in emissions. One
15 technique to simulate the response of O₃ to emissions changes, the brute force method, requires
16 the modeler to explicitly model this response by directly altering the emissions inputs in the
17 model simulation. This technique provides an estimate of the O₃ concentration at the altered
18 emission level, but often does not provide accurate information regarding the response of O₃ to
19 other levels of emissions since the chemistry for O₃ formation is nonlinear. Therefore, when
20 using only the brute force method, a new model simulation would need to be performed for
21 every emissions scenario under consideration.

22 Other analytical techniques have been developed to estimate the O₃ response to emission
23 perturbations without performing multiple simulations. One such method is termed the
24 Decoupled Direct Method (DDM) (Dunker, 1984). DDM, solves for sensitivity coefficients
25 which are defined as the partial derivative of the atmospheric diffusion equations that underly the
26 model calculations, Equations (3C-1) and (3C-2).

$$s_{ij}(t) = \frac{\partial C_i(t)}{\partial p_j}$$

27
28 Equation (3C-1)

$$S_{ij}(t) = \tilde{P}_j \frac{\partial C_i(t)}{\partial p_j} = \tilde{P}_j \frac{\partial C_i(t)}{\partial (\epsilon_j \tilde{P}_j)} = \frac{\partial C_i(t)}{\partial \epsilon_j}$$

29
30 Equation (3C-2)

1 Here, $S_{ij}(t)$, the sensitivity, gives the change in model concentration, C_i , (for instance O_3
 2 concentration) with an incremental change in any input parameter, p_j (in this case emissions).
 3 Equation (3C-2) allows us to normalize the sensitivity coefficient, $S_{ij}(t)$, so that it shows response
 4 in relative terms for the input rather than in absolute units. Therefore, $\tilde{P}_j(x,t)$ is the normalized
 5 input and ϵ_j is a scaling variable (Yang et al., 1997). In general terms, the sensitivity coefficient
 6 tells us how a model output (O_3 concentration) will change if a model input (emissions of NO_X
 7 or VOC) is perturbed. This first order sensitivity coefficient, $S_{ij}(t)$ is quite suitable for small
 8 perturbations, but gives a linear response which is unlikely to represent the results of large
 9 perturbations in very nonlinear chemical environments. Second (and third) order derivatives can
 10 be calculated to give higher order sensitivity coefficients (Hakami et al., 2003). Higher order
 11 sensitivity coefficients give the curvature and inflection points for the response curve and can
 12 capture the nonlinearities in the response of O_3 to emissions changes. Using Higher order DDM
 13 (HDDM) allows for the sensitivities to be more appropriately applied over larger emissions
 14 perturbations. Hakami et al. (2003) report that for an application in California, HDDM gave
 15 reasonable approximations of O_3 changes compared to that generated using brute force emissions
 16 reductions of up to 50% using the first three terms of the Taylor series expansion, Equation (3C-
 17 3).

$$C(+\Delta\epsilon) = C(0) + \Delta\epsilon S(0) + \frac{\Delta\epsilon^2}{2} S^2(0) + \dots + \frac{\Delta\epsilon^n}{n!} S^n(0) + R_{n+1}$$

18
 19 Equation (3C-3)

20 Here $\Delta\epsilon$ represents the relative change in emissions (for instance $\Delta\epsilon = -0.2$ would be equivalent
 21 to reducing emissions by 20%), $S^n(0)$ is the n^{th} order sensitivity coefficient, $C(0)$ is the
 22 concentration under baseline conditions (no perturbation in emissions) and R_{n+1} is a remainder
 23 term.

24 A variant of DDM called DDM-3D has been implemented into several chemical transport
 25 models, including CAMx, for both O_3 and particulate matter (PM) predictions (Cohan et al.,
 26 2005, Hakami et al., 2003, Napelenok et al., 2011, Dunker, 1984, Yang et al., 1997, Koo et al.,
 27 2007, Zhang et al., 2012). These implementations allow the modeler to define the parameters for
 28 which first and higher order sensitivities will be calculated. For instance, the sensitivity can be
 29 calculated for emissions from a specific source type, for emissions in a specific geographic
 30 region, and for emissions of a single O_3 precursor or for multiple O_3 precursors. In addition,
 31 sensitivities can be calculated to boundary conditions, initial conditions, and various other model
 32 inputs. Sensitivities to different sets of parameters can be calculated in a single model simulation

1 but computation time increases as the number of sensitivities increases. Outputs from an HDDM
2 simulation consist of time varying 3-D fields of first and second order sensitivities.

3 **3C.5.1.2 Limitations**

4 For the purposes of the O₃ NAAQS analysis, an HDDM-based approach is well-suited
5 given its ability to 1) capture the non-linearity of O₃ response to emissions changes, 2)
6 characterize different O₃ responses at different locations (downtown urban versus downwind
7 suburban) and at different times of day, allowing us to incorporate temporal and spatial
8 variations in response into the O₃ adjustment methodology, and 3) explicitly account for physical
9 and chemical processes influencing predicted sensitivities such as background O₃ sources.
10 However, in addition to the many potential benefits of using HDDM to understand and
11 characterize O₃ response to emissions changes, there are several limitations.

12 First, HDDM encompasses all of the uncertainties of the base photochemical model
13 formulation and inputs. So, uncertainties in how the physical and chemical processes are treated
14 in the model and in the model inputs propagate to the HDDM results. Also, HDDM can capture
15 response to larger emissions perturbations than DDM but it is still most accurate for small
16 perturbations. The larger the relative change in emissions, the less likely that the HDDM
17 sensitivities will properly capture the change in O₃ that would be predicted by using brute force
18 emission reductions. Several studies have reported reasonable performance of HDDM for O₃ up
19 to 50% emissions perturbations (Hakami et al., 2004, Hakami et al., 2003, Cohan et al., 2005),
20 but the magnitude of perturbation over which HDDM will give accurate estimates will depend on
21 the specific modeling episode, size of the model domain, emissions and meteorological inputs,
22 and the size of the emissions source to which the sensitivity is being calculated. In this work, we
23 applied sensitivities derived from model simulations done under varying NO_x levels (see section
24 3C.5.2.2) and found that using this technique we were able to replicate O₃ concentrations
25 estimated using brute force emission reductions with HDDM sensitivities for up to 90% NO_x
26 emissions reductions with a mean bias of less than 3 ppb and a mean error of less than 4 ppb.

27 **3C.5.2 Using CAMx/HDDM to Adjust Monitored Ozone Concentrations**

28 **3C.5.2.1 Conceptual Framework**

29 This section outlines the methodology in which we apply CAMx/HDDM to estimate
30 hourly O₃ concentrations that might result from just meeting three air quality scenarios (75 ppb,
31 70 ppb, and 65 ppb). These methods closely follow those documented in Simon et al. (2013) and
32 the risk and exposure assessment performed in the 2015 O₃ NAAQS review (U.S. EPA, 2014).
33 As part of the methodology, photochemical modeling results are not used in an absolute sense,

1 but instead are applied to modulate ambient air measurements, thus tying estimated O₃
2 distributions to measured values. The basic steps are outlined below and in Figure 3C-48.

3

4 **Step 1:** Run CAMx simulation with HDDM to determine hourly O₃ sensitivities to NO_x
5 emissions changes for the grid cells containing monitoring sites in an urban study area.

6 **Step 2:** For each monitoring site, season, and hour of the day use linear regression to relate first
7 order sensitivities of NO_x (S_{NO_x}) to modeled O₃ and second order sensitivities of NO_x ($S^2_{NO_x}$) to
8 the first order sensitivities.

9 **Step 3:** For each measured hourly O₃ value, calculate the first and second order sensitivities
10 based on monitoring site-, season-, and hour-specific functions calculated in Step 2.

11 **Step 4:** Adjust measured hourly 2015-2017 O₃ concentrations for incrementally increasing levels
12 of emissions reductions using assigned sensitivities, then recalculate 2015-2017 design values
13 until all monitors in the urban study area just meet the levels of the air quality scenario.

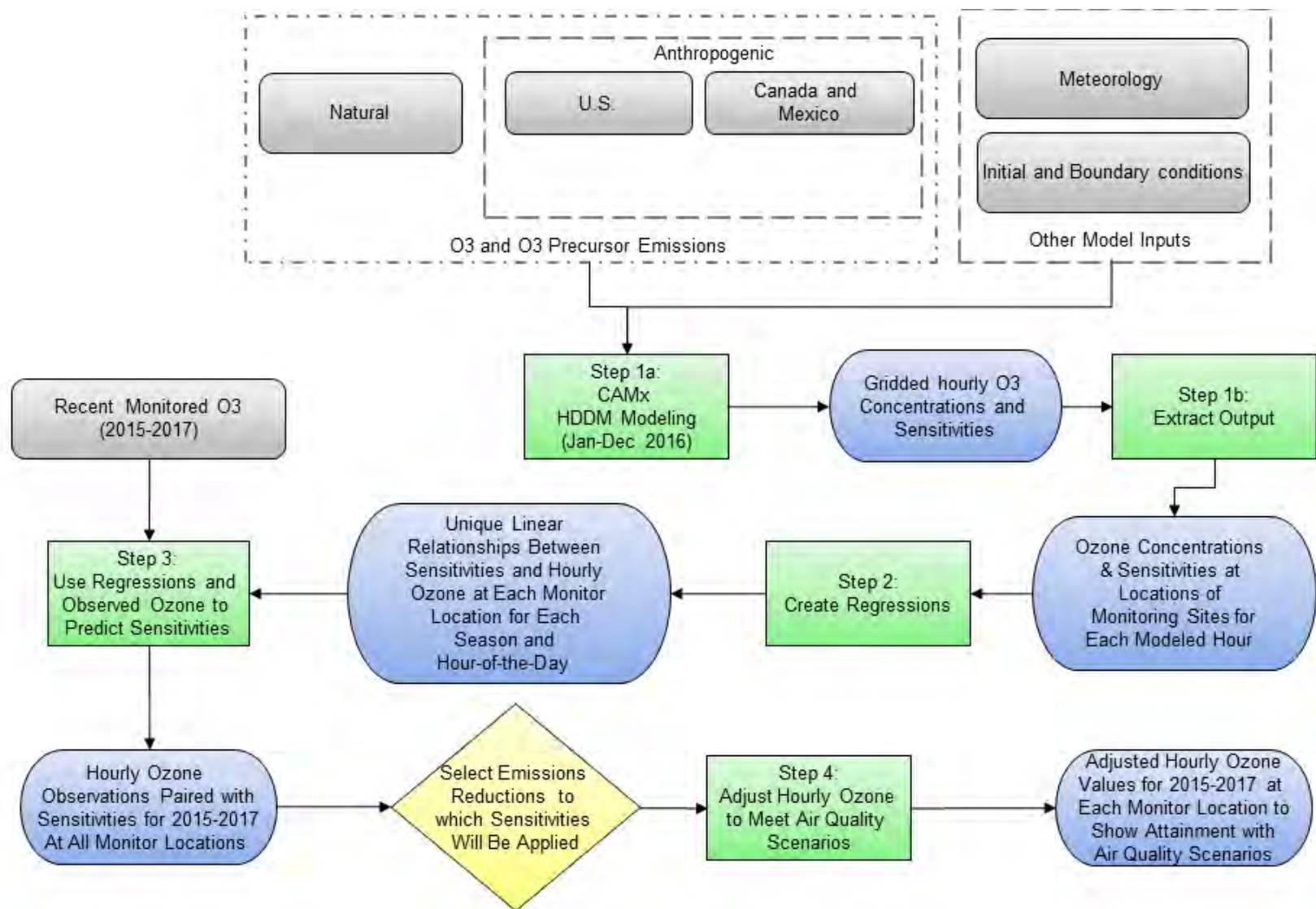


Figure 3C-48. Flow diagram demonstrating HDDM model-based O₃ adjustment approach.

1

2 **3C.5.2.2 Application to Measured O₃ Concentrations in Urban Study Areas**

3 The model-based adjustment approach described above was applied to the eight urban
4 study areas (Atlanta, Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento, and St. Louis)
5 for an air quality scenario adjusted to just meet the current standard of 70 ppb and two alternative
6 air quality scenarios having design values of 75 ppb and 65 ppb. The analysis used CAMx
7 photochemical modeling for January-December of 2016 and ambient air data for the years 2015-
8 2017. When running CAMx with HDDM, additional information is required to designate model
9 inputs for calculating sensitivities. In this analysis, HDDM was set up to calculate the sensitivity
10 of O₃ concentrations to U.S. anthropogenic NO_x emissions.¹³

11 U.S. anthropogenic emissions were defined as all emissions in the following sectors:
12 commercial marine, rail, residential wood combustion, agricultural fires, onroad mobile, offroad
13 mobile, EGU point sources, oil and natural gas point, non-EGU point, non-point oil and gas, and
14 non-point area. These anthropogenic sectors account for 10.5 million of the total CONUS-wide
15 11.8 million tons per year of NO_x emissions in 2016 (the remaining 1.3 million tons are from
16 biogenics and wildland fires, which included prescribed burns). Sensitivities were not calculated
17 for biogenic, wildland fire, Canadian, or Mexican emissions. In addition, sensitivities were not
18 calculated for any emissions originating from outside the domain (i.e., entering through the use
19 of boundary concentrations).

20 **3C.5.2.2.1 Multi-step Application of HDDM Sensitivities**

21 As discussed in section 3C.5.1.2 of this appendix, HDDM has been reported to
22 reasonably replicate brute force emissions reductions up to a 50% change in emissions. For this
23 analysis, it was desirable to have confidence that the HDDM sensitivities could replicate the
24 entire range of emissions reductions. Evaluations of the HDDM estimated O₃ concentrations
25 compared to that estimated from brute force emissions reduction model runs confirm that the
26 HDDM estimates of O₃ response to NO_x reductions are fairly comparable for a 50% change.
27 However, O₃ concentrations estimated from the HDDM sensitivities and the brute force method
28 begin to diverge in comparisons under larger emissions changes (90%). Consequently, two
29 additional CAMx/HDDM runs were performed under different levels of NO_x emissions
30 reductions in order to characterize O₃ sensitivities to NO_x reductions over a larger range of
31 emissions perturbations. One CAMx/HDDM simulation was performed with U.S. anthropogenic

¹³ Sensitivities were only assessed using U.S. emissions in the contiguous 48 states. We did not assess responses to VOC emission reductions in this analysis as a means to reduce computational costs because none of the urban study areas considered here required VOC emission reductions to achieve the lower design values in the air quality scenarios simulated in the 2014 HREA.

1 NO_X emissions reduced by 50%. A second additional simulation was performed with a 90%
 2 NO_X reduction. Emissions of other species were not modified from the base case in these two
 3 additional simulations. These additional HDDM simulations provide O₃ sensitivities to NO_X
 4 under chemical regimes with lower NO_X emissions. The sensitivities are used in a multistep
 5 adjustment approach, as described in the following sections.

6 Figure 3C-49 provides a conceptual picture of the multistep adjustment procedure using
 7 first-order sensitivities. Sensitivities from the base run are used to adjust O₃ concentrations for
 8 NO_X emissions reductions up to X%. Additional emission reductions beyond X% use
 9 sensitivities from the 50% NO_X cut run until reductions exceed (X+Y)%. Finally, sensitivities
 10 from the 90% NO_X emissions reduction run are applied for any emission reductions beyond
 11 (X+Y)%. In order to more closely approximate the non-linear O₃ response to any level of
 12 emissions reductions, 2nd order terms are added to the multistep approximation method in
 13 Equations (3C-4) through (3C-7). P represents the percentage NO_X cut for which the ΔO₃ values
 14 are being calculated, S and S² are the first and second order O₃ sensitivities to U.S. NO_X
 15 emissions, and X and Y are described above.

$$\Delta O_3 = -a \times S_{NOx_{base}} + \frac{a^2}{2} \times S_{NOx_{base}}^2 - b \times S_{NOx_{50\%cut}} + \frac{b^2}{2} \times S_{NOx_{50\%cut}}^2 - c \times S_{NOx_{90\%cut}} + \frac{c^2}{2} \times S_{NOx_{90\%cut}}^2$$

16
 17 Equation (3C-4)

$$a = \begin{cases} \frac{P}{100} & \text{for } P \leq X \\ \frac{X}{100} & \text{for } P > X \end{cases}$$

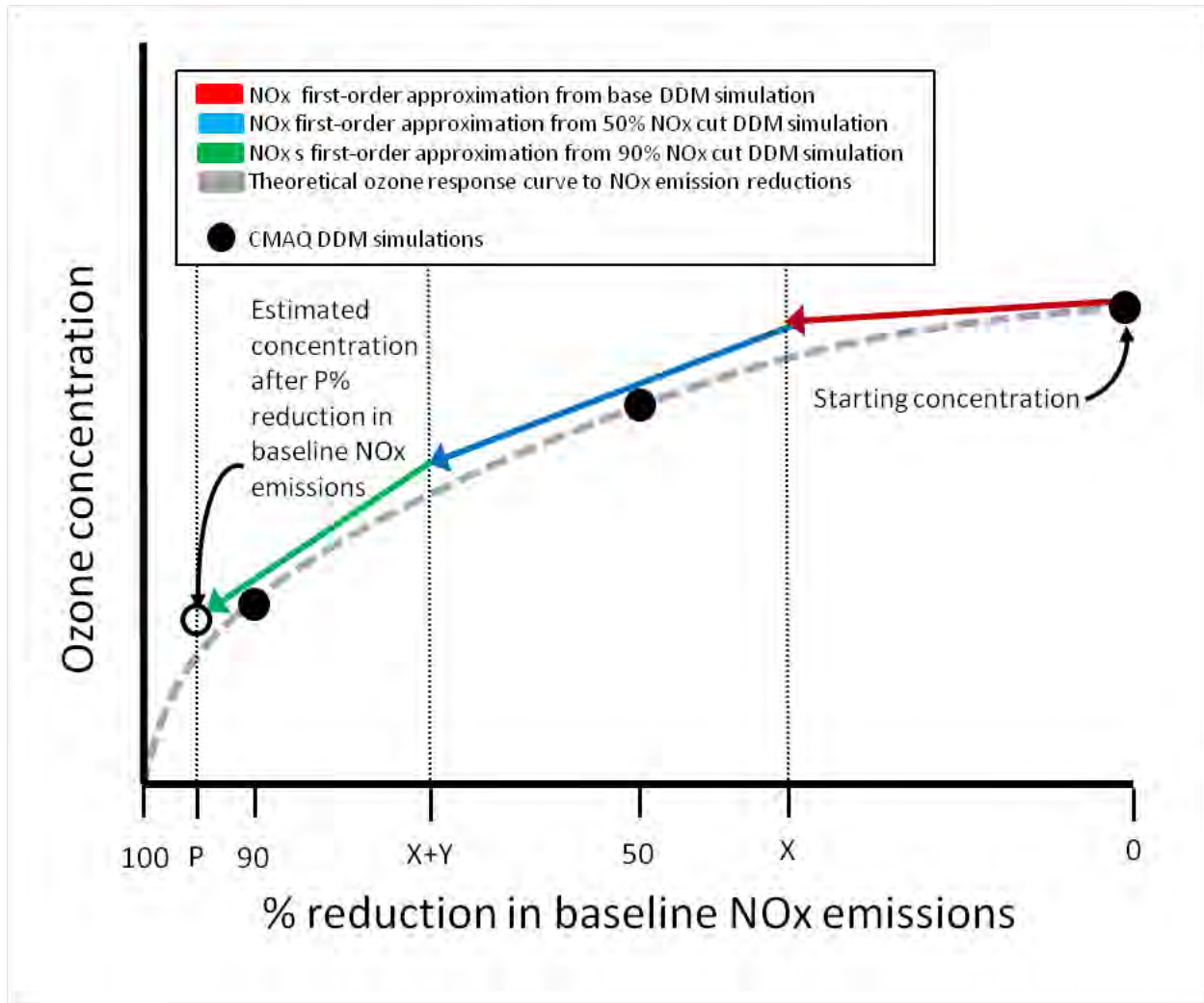
18
 19 Equation (3C-5)

$$b = \begin{cases} 0 & \text{for } P \leq X \\ \frac{2 \times (P - X)}{100} & \text{for } X < P \leq (X + Y) \\ \frac{2 \times Y}{100} & \text{for } P > (X + Y) \end{cases}$$

20
 21 Equation (3C-6)

$$c = \begin{cases} 0 & \text{for } P \leq (X + Y) \\ \frac{10 \times (P - (X + Y))}{100} & \text{for } 100 \geq P > (X + Y) \end{cases}$$

22
 23 Equation (3C-7)



1
2 **Figure 3C-49. Conceptual picture of 3-step application of HDDM sensitivities.**

3 The ideal value for equation transition points, X and Y, are determined by minimizing the
4 least square mean error between the adjusted concentrations using the multistep approach and
5 modeled concentrations from brute force NO_x emissions reduction runs. We first determined the
6 value of X which gave the lowest error compared to brute forces estimates at 50% NO_x
7 emissions reductions. Then holding X constant, we determined the value of Y which gave the
8 lowest error compared to brute force method O₃ concentration estimates using 90% NO_x
9 emissions reductions. This process was performed independently for each of the eight urban
10 study areas in this analysis.

11 Error in HDDM estimates of hourly O₃ is defined here as the difference between HDDM
12 estimated O₃ and O₃ estimated using the brute force method. Based on equations (3C-4) through
13 (3C-7), this can be calculated from Equations (3C-8) and (3C-9) for 50% NO_x emissions
14 reductions:

$$\varepsilon = \Delta Ozone_{HDDM,50} - \Delta Ozone_{BF,50}$$

Equation (3C-8)

$$\varepsilon = \frac{-X}{100} \times S_{NOx_{base}} + \frac{X^2}{2 \times 100^2} \times S_{NOx_{base}}^2 - \frac{2(50-X)}{100} \times S_{NOx_{zo}\%cut} + \frac{(2 \times (50-X))^2}{2 \times 100^2} \times S_{NOx_{zo}\%cut}^2 - \Delta Ozone_{BF,50}$$

Equation (3C-9)

Equation (3C-10) can be rearranged to appear in the form: $AX^2 + BX + C$:

$$\varepsilon = \left(\frac{S_{NOx_{base}}^2}{2 \times 100^2} + \frac{4 \times S_{NOx_{zo}\%cut}^2}{2 \times 100^2} \right) X^2 + \left(\frac{-S_{NOx_{base}}}{100} + \frac{2 \times S_{NOx_{zo}\%cut}}{100} - \frac{400 \times S_{NOx_{zo}\%cut}^2}{2 \times 100^2} \right) X + \left(-S_{NOx_{zo}\%cut} + \frac{S_{NOx_{zo}\%cut}^2}{2} - \Delta Ozone_{BF,50} \right)$$

Equation (3C-10)

$$A = \left(\frac{S_{NOx_{base}}^2}{2 \times 100^2} + \frac{4 \times S_{NOx_{zo}\%cut}^2}{2 \times 100^2} \right)$$

Equation (3C-11)

$$B = \left(\frac{-S_{NOx_{base}}}{100} + \frac{2 \times S_{NOx_{zo}\%cut}}{100} - \frac{400 \times S_{NOx_{zo}\%cut}^2}{2 \times 100^2} \right)$$

Equation (3C-12)

$$C = \left(-S_{NOx_{zo}\%cut} + \frac{S_{NOx_{zo}\%cut}^2}{2} - \Delta Ozone_{BF,50} \right)$$

Equation (3C-13)

Next, the error is squared, summed over all points (error can be calculated for each hourly O₃ value at each monitoring location), and the derivative is set to 0 to determine X which gives the least squares error (Equations (3C-14), (3C-15), and (3C-16)).

$$\varepsilon^2 = A^2 X^4 + 2ABX^3 + (2AC + B^2)X^2 + 2BCX + C^2$$

Equation (3C-14)

$$\sum \varepsilon^2 = (\sum A^2)X^4 + (\sum 2AB)X^3 + (\sum 2AC + B^2)X^2 + (\sum 2BC)X + \sum C^2$$

Equation (3C-15)

$$(\sum \varepsilon^2)' = (4 \sum A^2)X^3 + (3 \sum 2AB)X^2 + (2 \sum 2AC + B^2)X + (\sum 2BC) = 0$$

Equation (3C-16)

The value of X that gives the least squares error will occur at one of the three roots of the trinomial in Equation (3C-16) or at 0 or 50. All real roots, 0, and 50 were input into equation (3C-15) and X was set to the value which resulted in the lowest error in each city. An analogous procedure was followed to determine Y using the 90% NO_X emissions reduction brute force simulation and Equations (3C-17) through (3C-23).

$$\begin{aligned} \varepsilon = & \frac{-X}{100} \times S_{NOx_{base}} + \frac{X^2}{2 \times 100^2} \times S_{NOx_{base}}^2 - \frac{2Y}{100} \times S_{NOx_{90\%cut}} \\ & + \frac{2^2 Y^2}{2 \times 100^2} \times S_{NOx_{90\%cut}}^2 - \frac{10(90 - (X + Y))}{100} \times S_{NOx_{90\%cut}} \\ & + \frac{(10^2(90 - (X + Y)))^2}{2 \times 100^2} \times S_{NOx_{90\%cut}}^2 - \Delta Ozone_{BF,90} \end{aligned}$$

Equation (3C-17)

$$\varepsilon^2 = A^2 Y^4 + 2ABY^3 + (2AC + B^2)Y^2 + 2BCY + C^2$$

Equation (3C-18)

$$A = \left(\frac{4 \times S_{NOx_{90\%cut}}^2}{2 \times 100^2} + \frac{100 \times S_{NOx_{90\%cut}}^2}{2 \times 100^2} \right)$$

Equation (3C-19)

$$B = \left(\frac{-2 \times S_{NOx_{90\%cut}}}{100} + \frac{10 \times S_{NOx_{90\%cut}}}{100} - \frac{200 \times (90 - X) S_{NOx_{90\%cut}}^2}{2 \times 100^2} \right)$$

Equation (3C-20)

$$C = \left(\frac{-X}{100} S_{NOx_{base}} + \frac{X^2}{2 \times 100^2} S_{NOx_{base}}^2 - \frac{10 \times (90 - X)}{100} S_{NOx_{90\%cut}} + \frac{100 \times (90 - X)^2}{2 \times 100^2} S_{NOx_{90\%cut}}^2 - \Delta Ozone_{BF,90} \right)$$

Equation (3C-21)

$$\sum \varepsilon^2 = (\sum A^2)Y^4 + (\sum 2AB)Y^3 + (\sum 2AC + B^2)Y^2 + (\sum 2BC)Y + \sum C^2$$

Equation (3C-22)

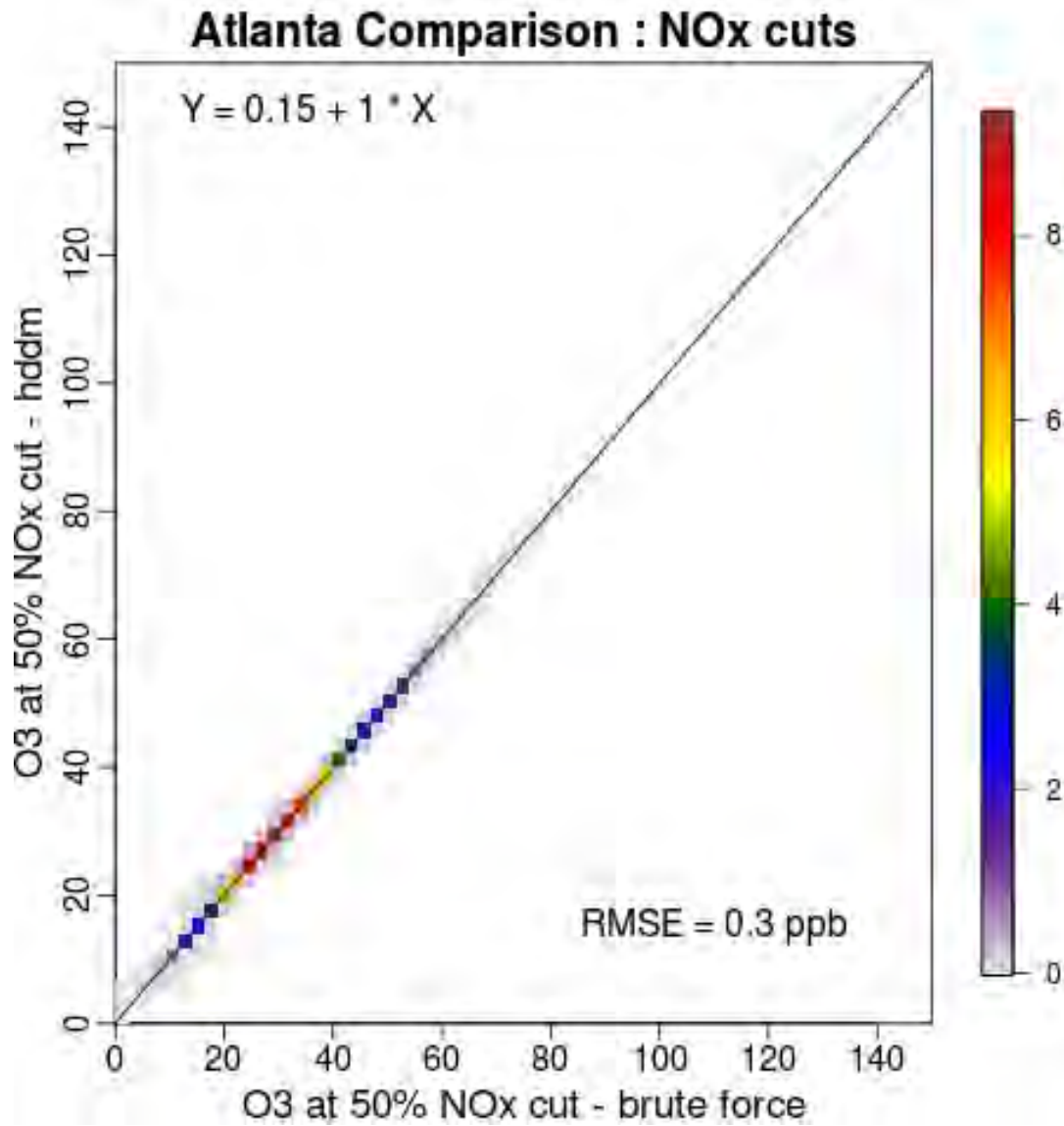
$$(\sum \epsilon^2)' = (4 \sum A^2)Y^2 + (3 \sum 2AB)Y^2 + (2 \sum 2AC + B^2)Y + (\sum 2BC) = 0$$

Equation (3C-23)

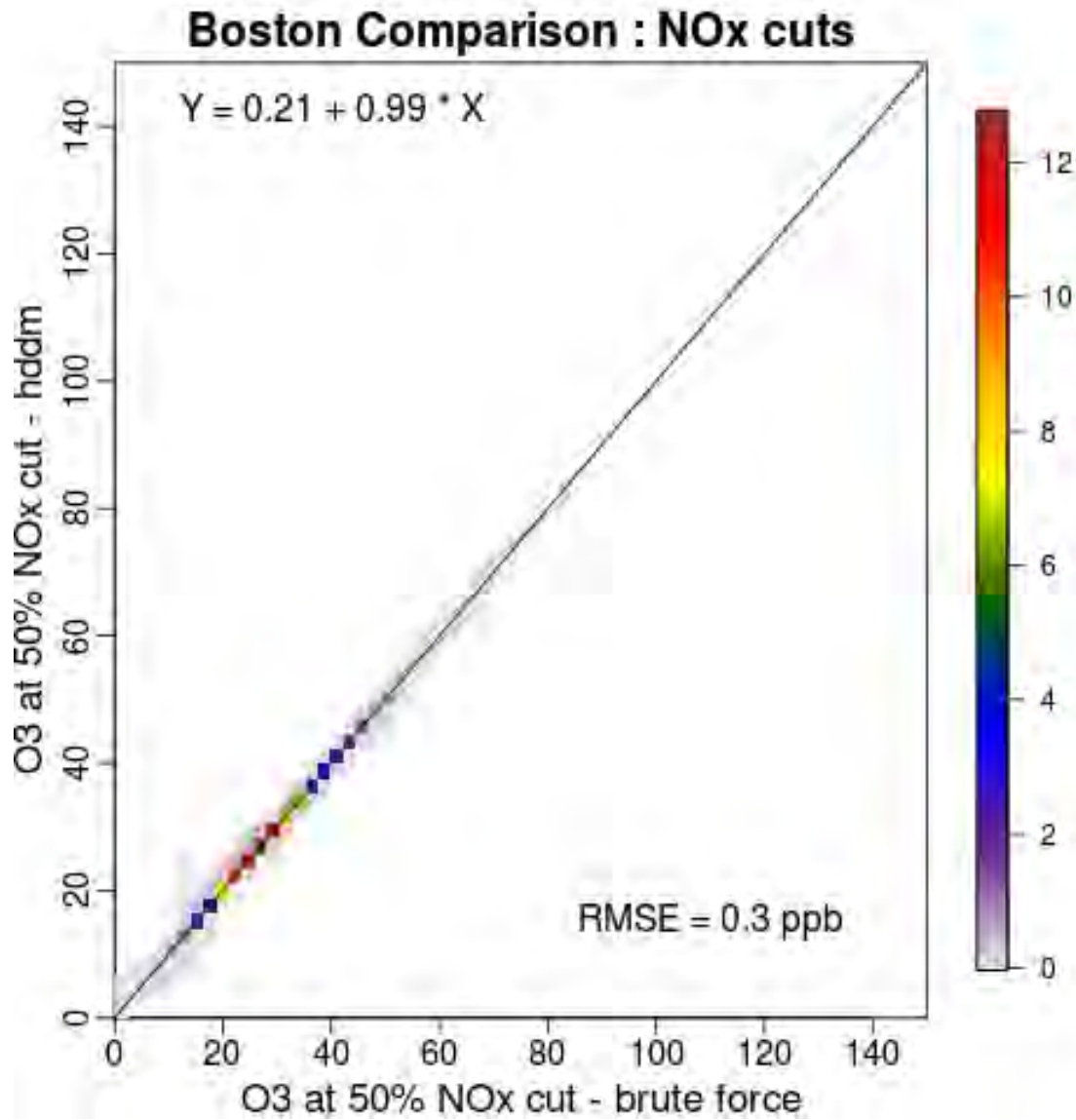
The X and Y cutpoints which have the least square error in each urban study area are shown in Table 3C-18. This 3-step adjustment methodology was shown to be a robust method for minimizing error in the HDDM applications for larger percentage changes in emissions by Simon et al. (2013). Figure 3C-50 through Figure 3C-65 are density scatter plots that compare hourly O₃ estimates from brute force with hourly O₃ estimates from the 3-step HDDM adjustments at all monitor locations in each of the eight urban study areas evaluated in this study. The colors in these plots depict the percentage of points falling at any one location. Mean error for the 50% and 90% 3-step HDDM adjustment NO_x emissions reductions cases compared to O₃ concentrations estimated using the brute force method are less than 0.5 ppb and 2 ppb respectively in all eight urban study areas.

Table 3C-18. X and Y cut-points used in Equations (3C-4) through (3C-7).

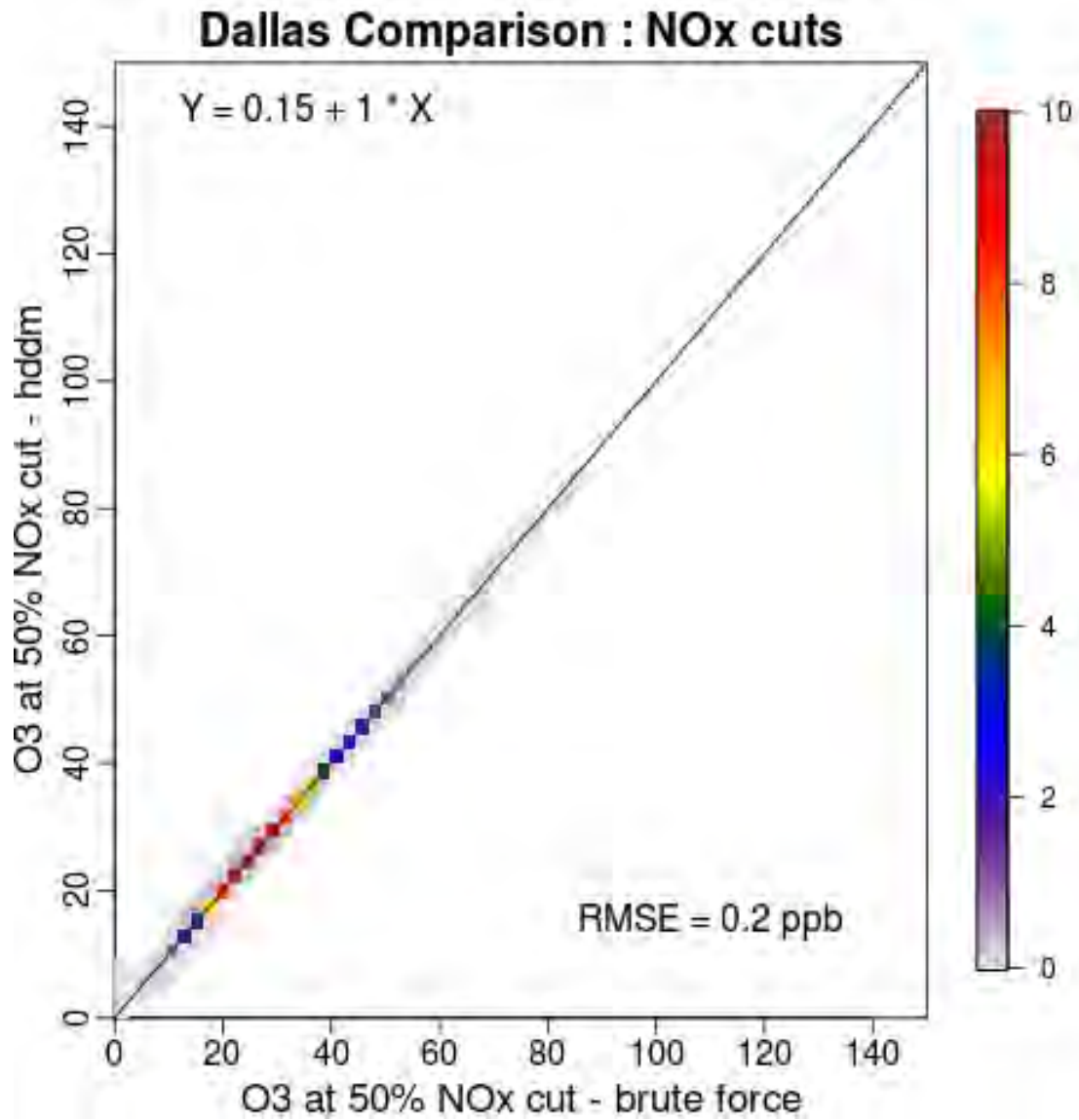
Urban Study Area	X	Y
Atlanta	37	48
Boston	38	45
Dallas	37	47
Detroit	37	45
Philadelphia	37	45
Phoenix	37	45
Sacramento	38	48
St. Louis	37	47



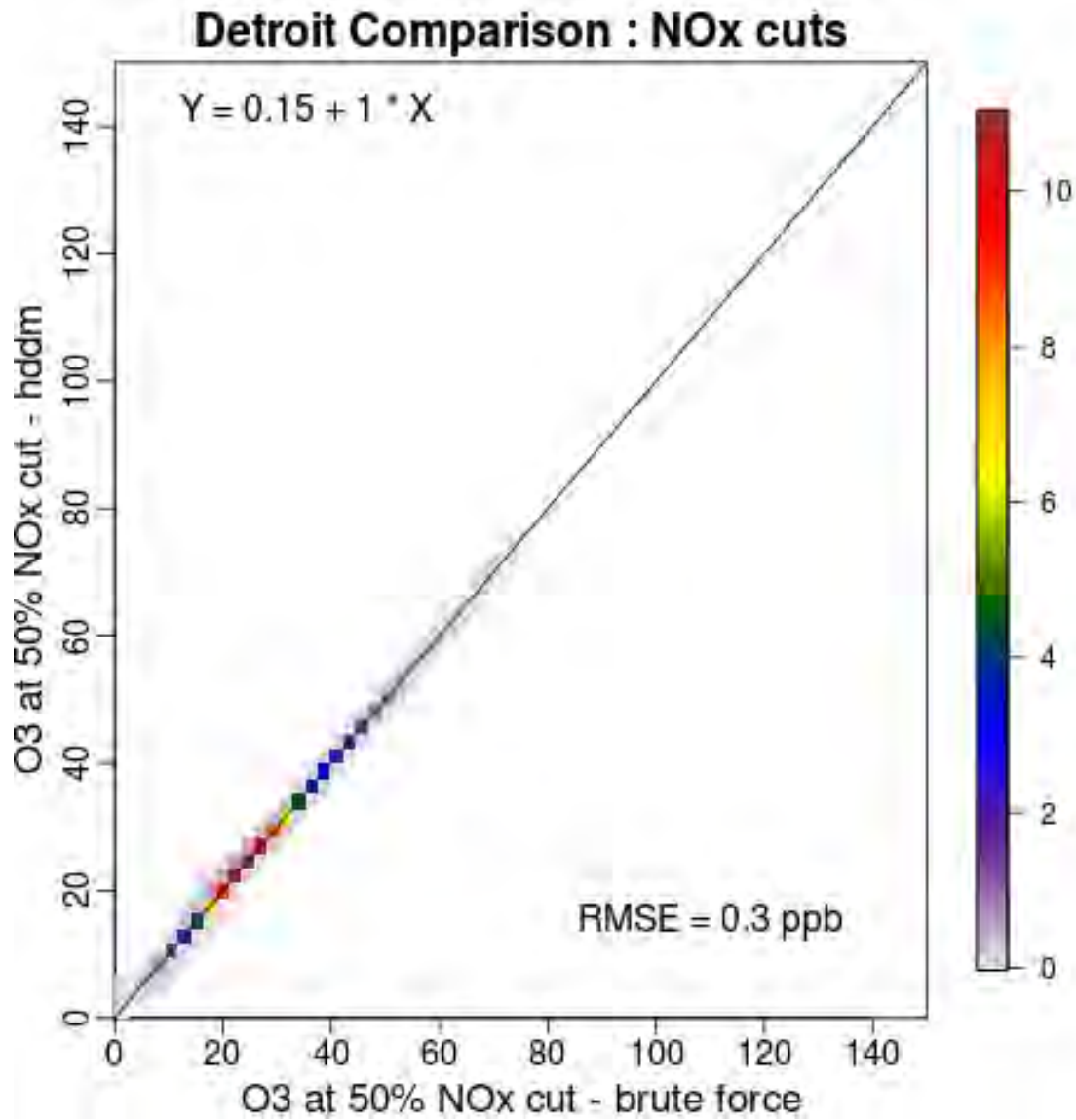
1
 2 **Figure 3C-50. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in Atlanta.**



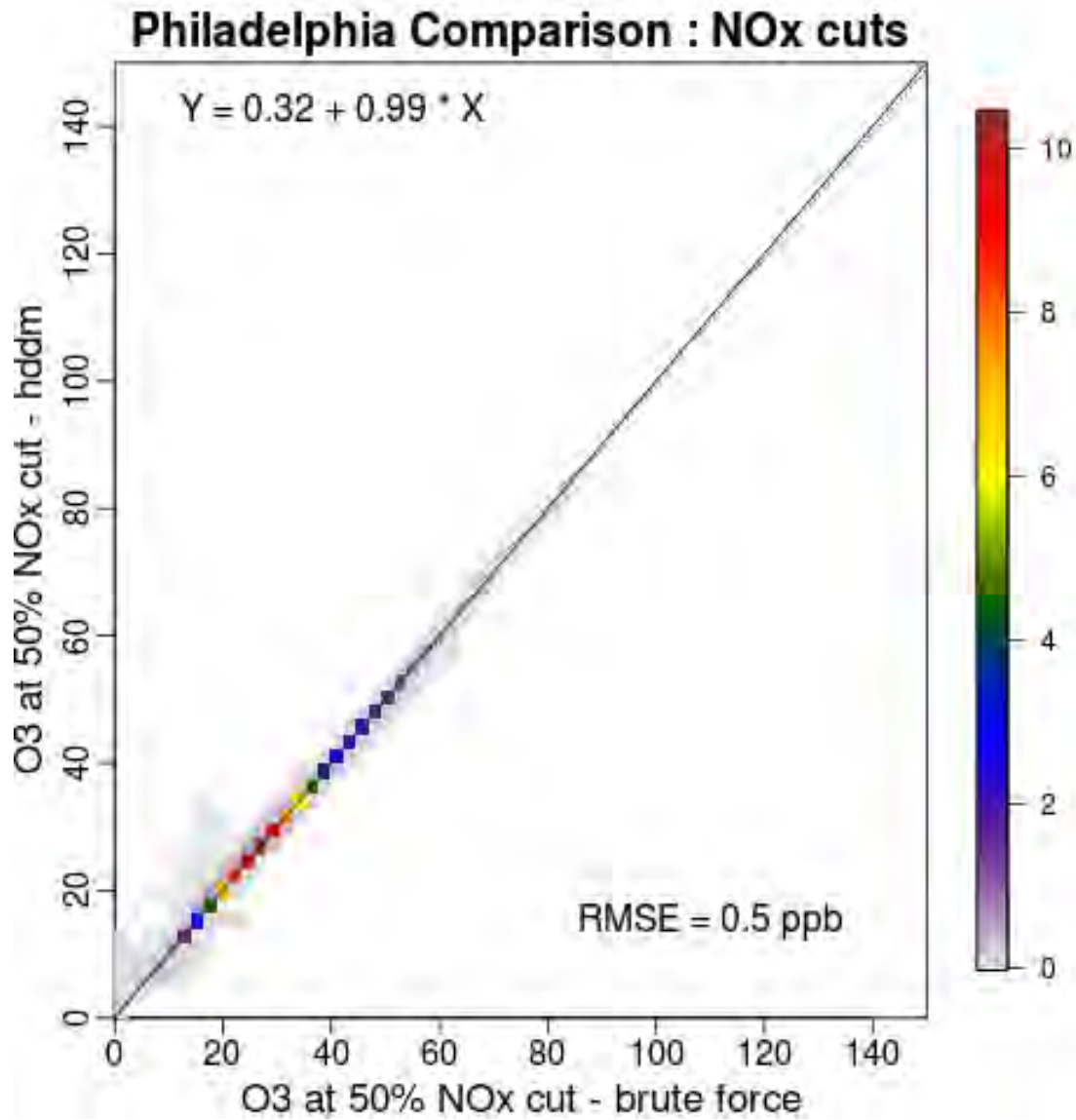
1
 2 **Figure 3C-51. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in Boston.**



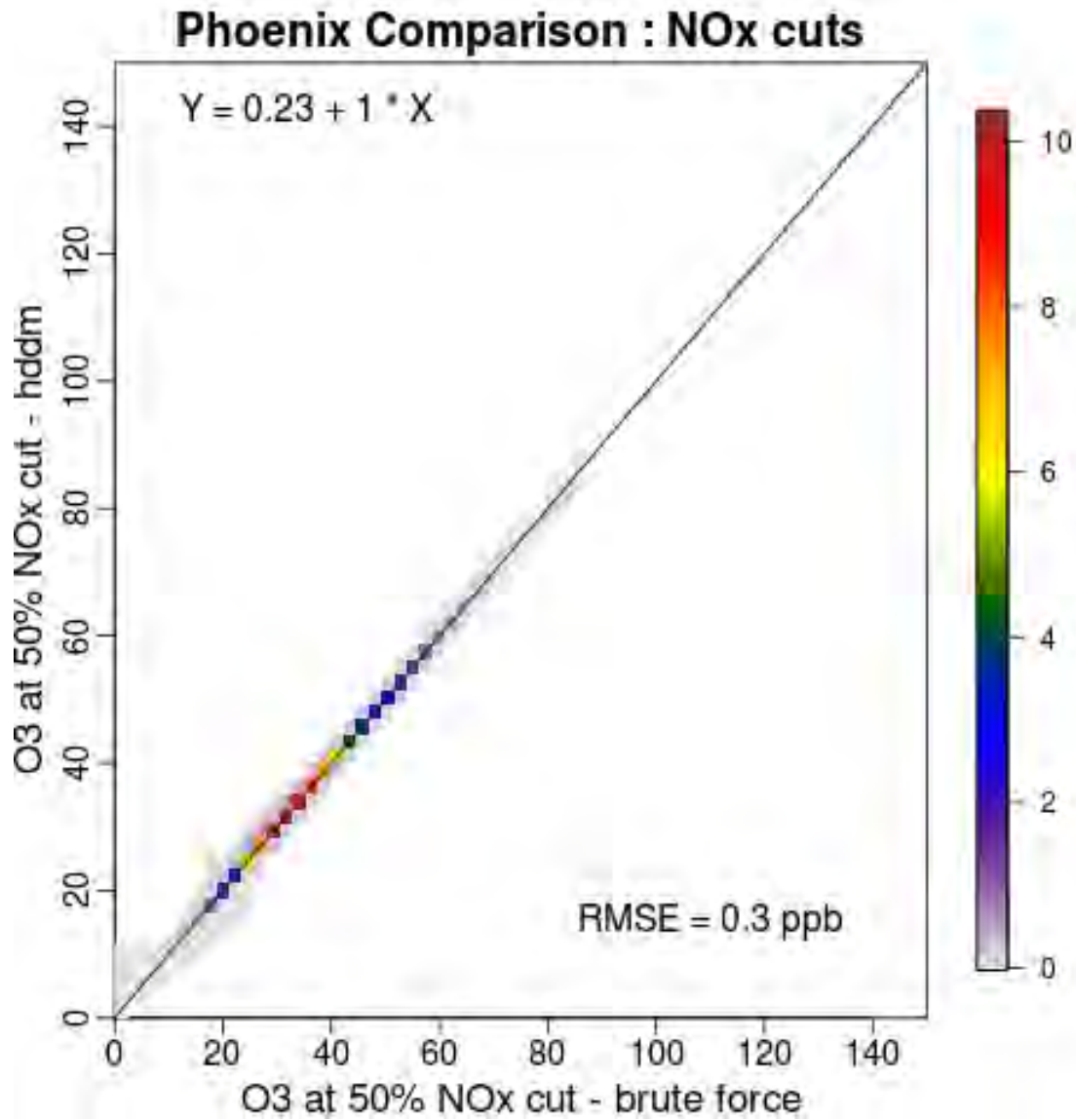
1
 2 **Figure 3C-52. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in Dallas.**



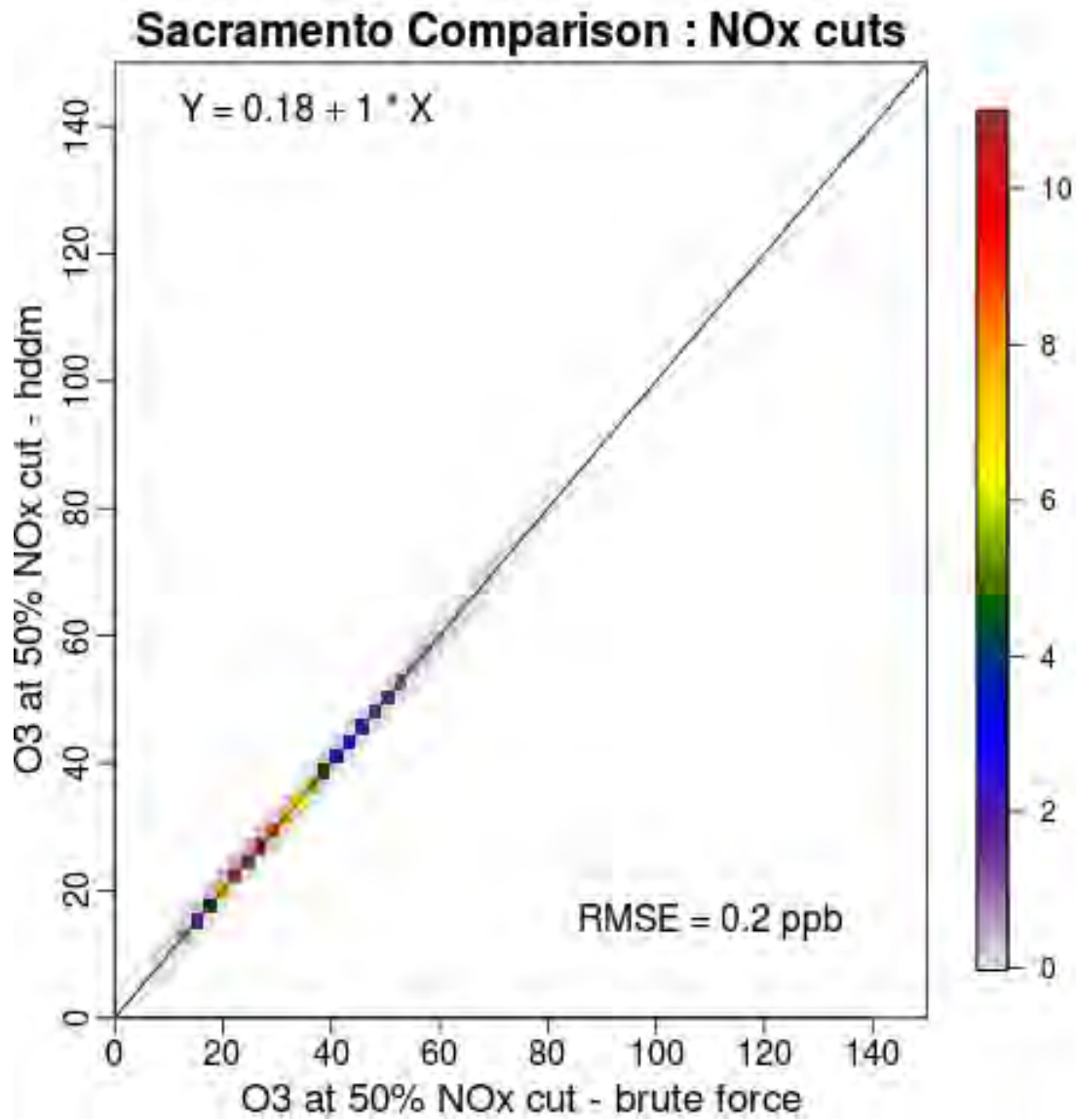
1
 2 **Figure 3C-53. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in Detroit.**



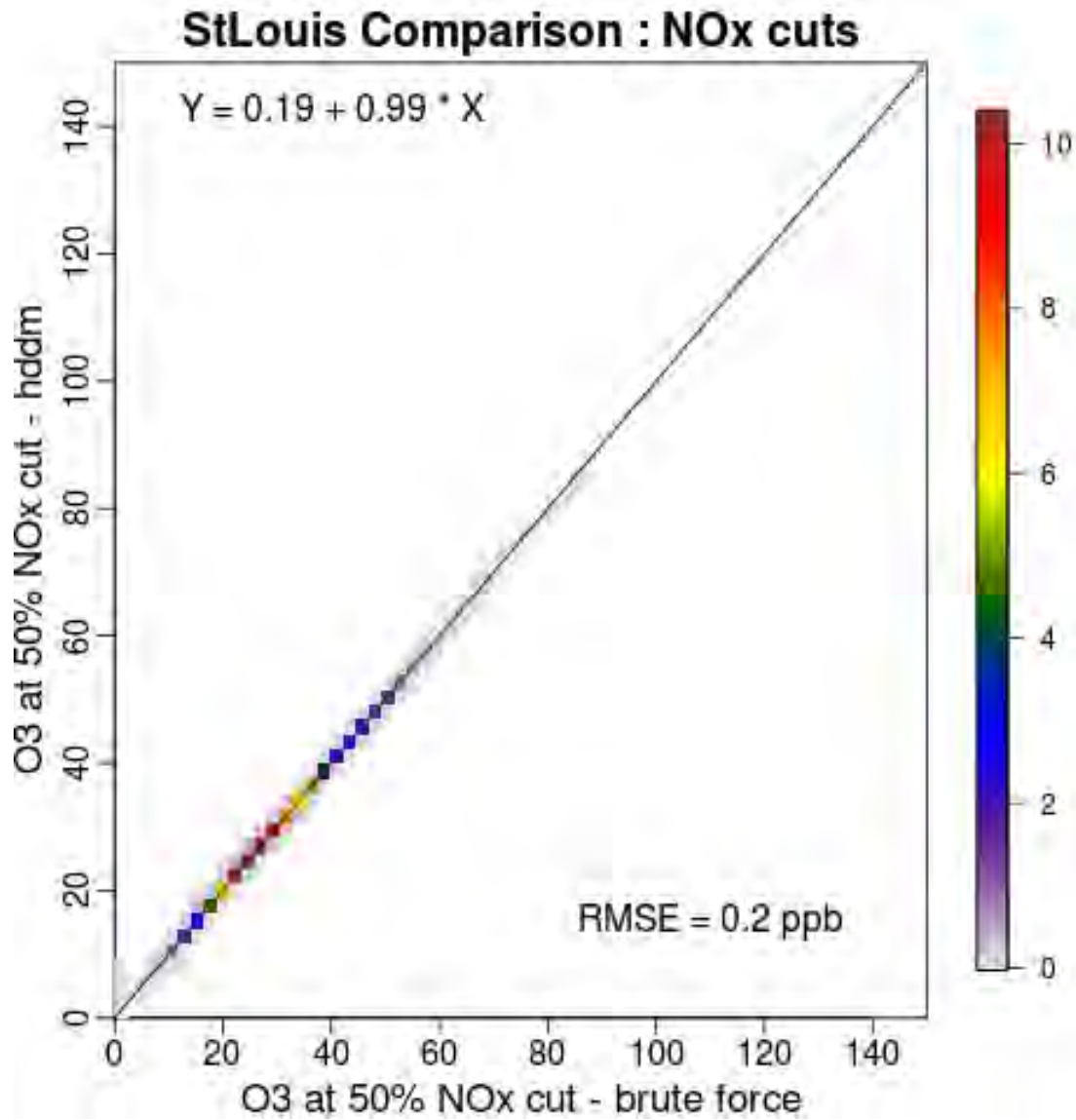
1
 2 **Figure 3C-54. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in Philadelphia.**



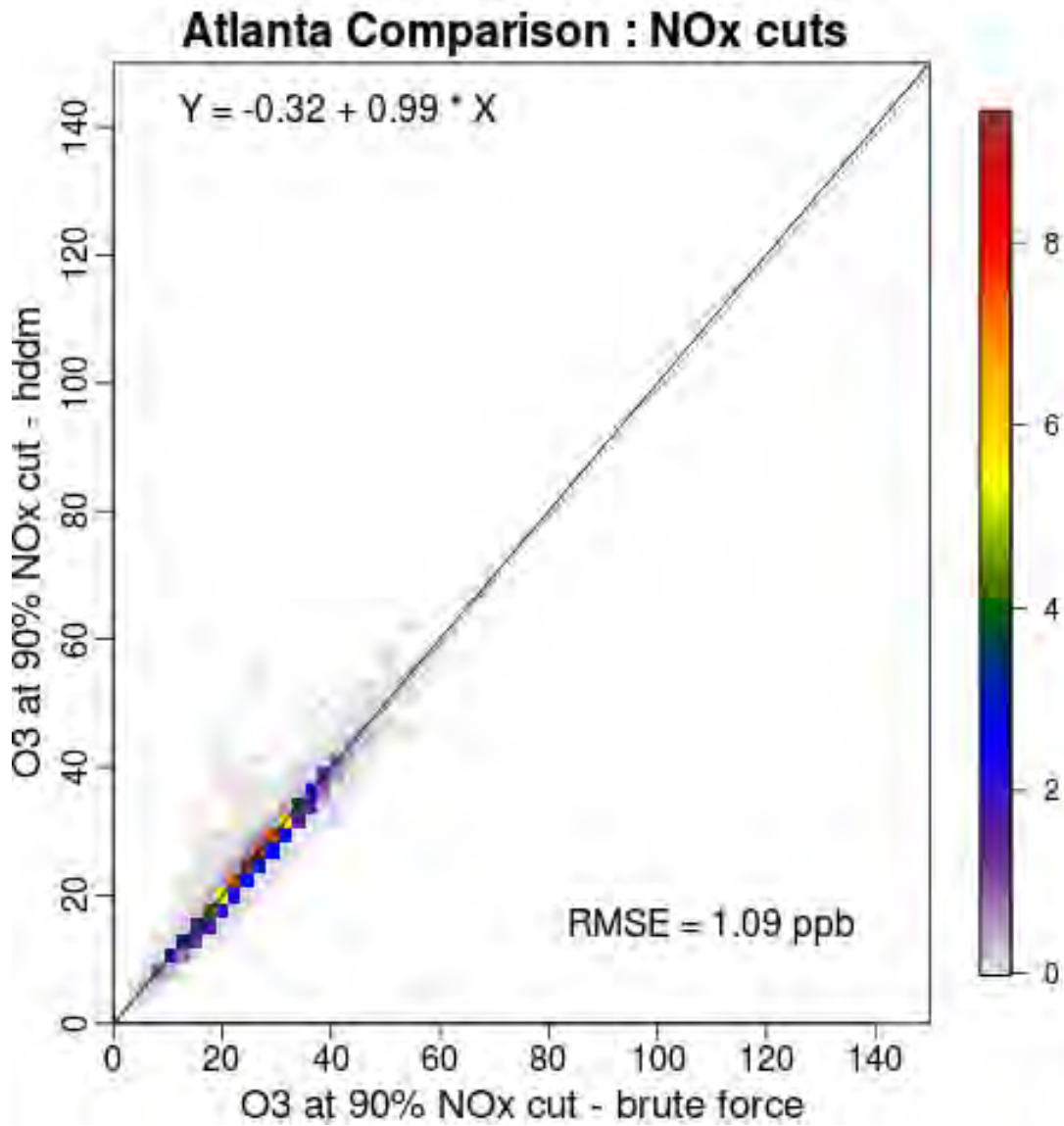
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 2 **Figure 3C-55. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in Phoenix.**



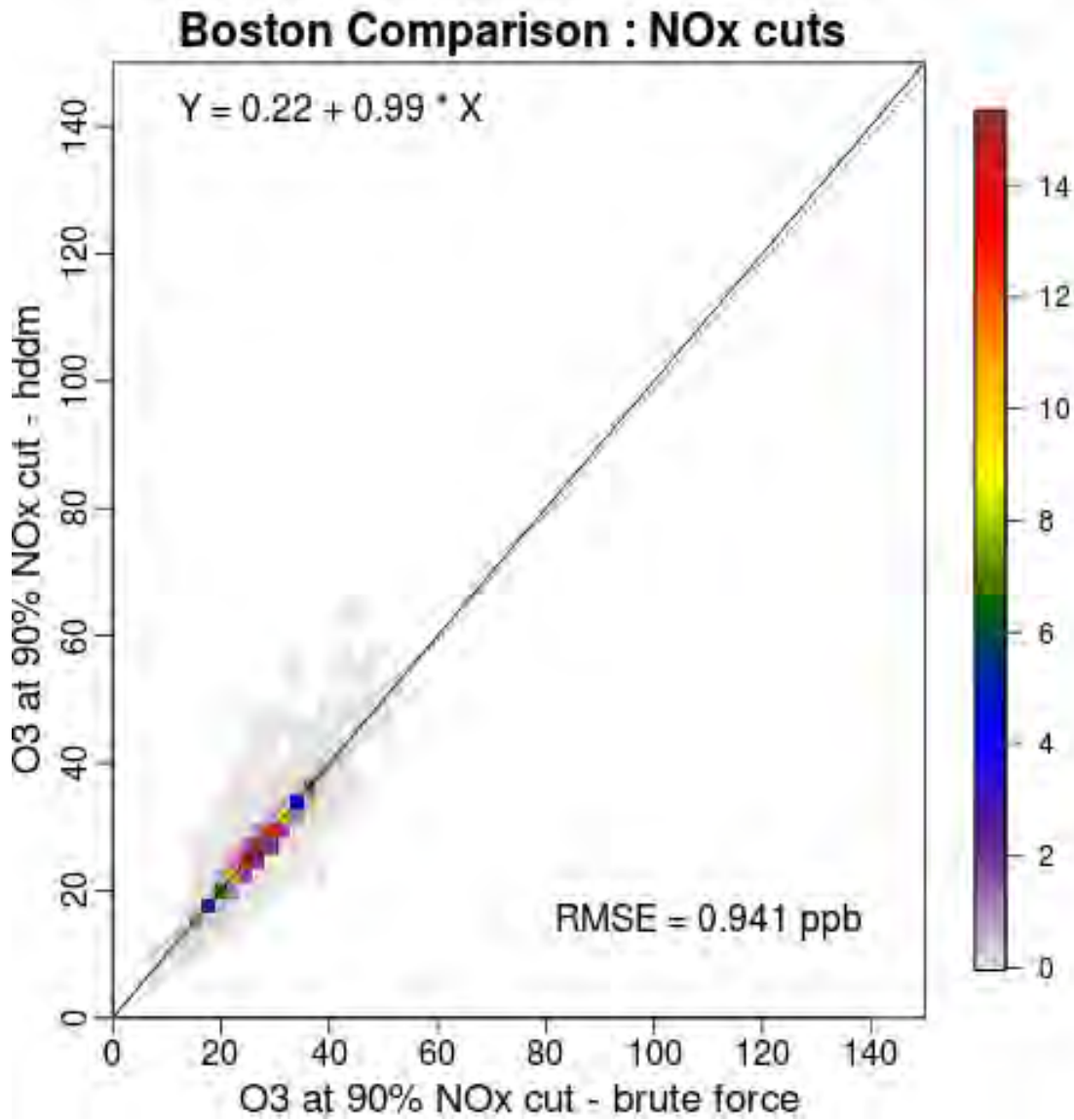
1
 2 **Figure 3C-56. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in Sacramento.**



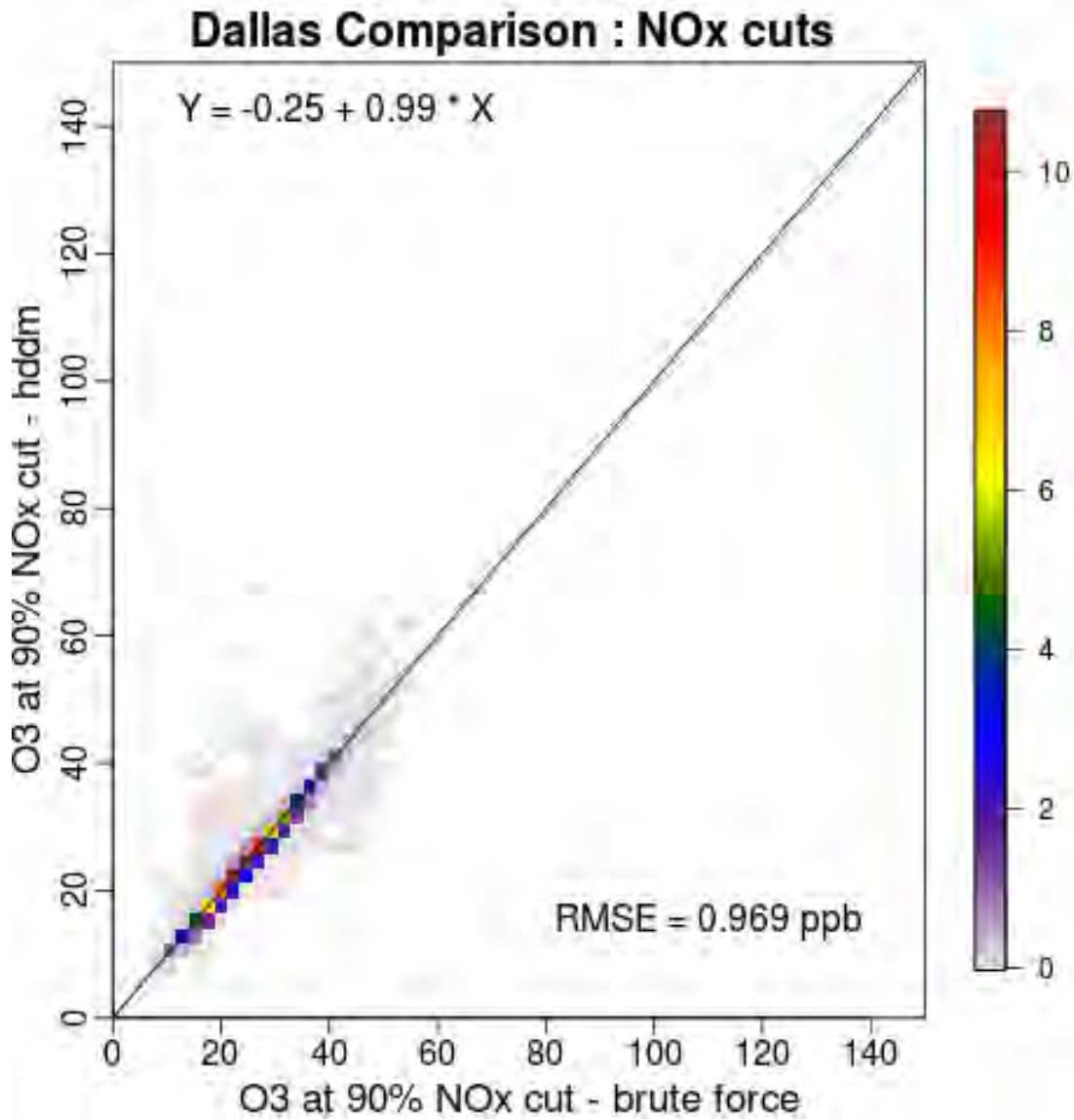
1
 2 **Figure 3C-57. Comparison of brute force and 3-step HDDM O₃ estimates for 50% NO_x**
 3 **cut conditions in St. Louis.**



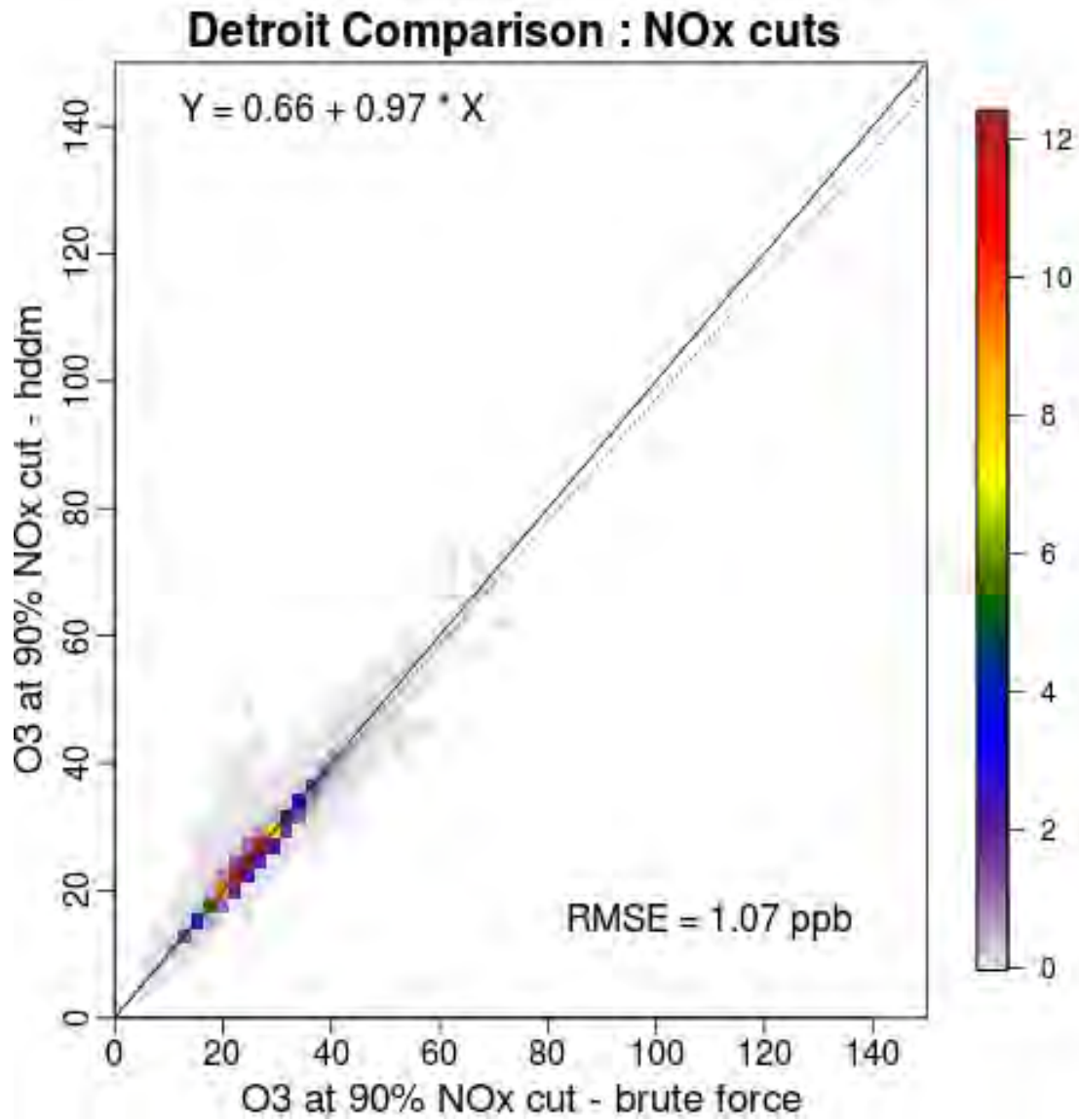
1
 2 **Figure 3C-58. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
 3 **cut conditions in Atlanta.**



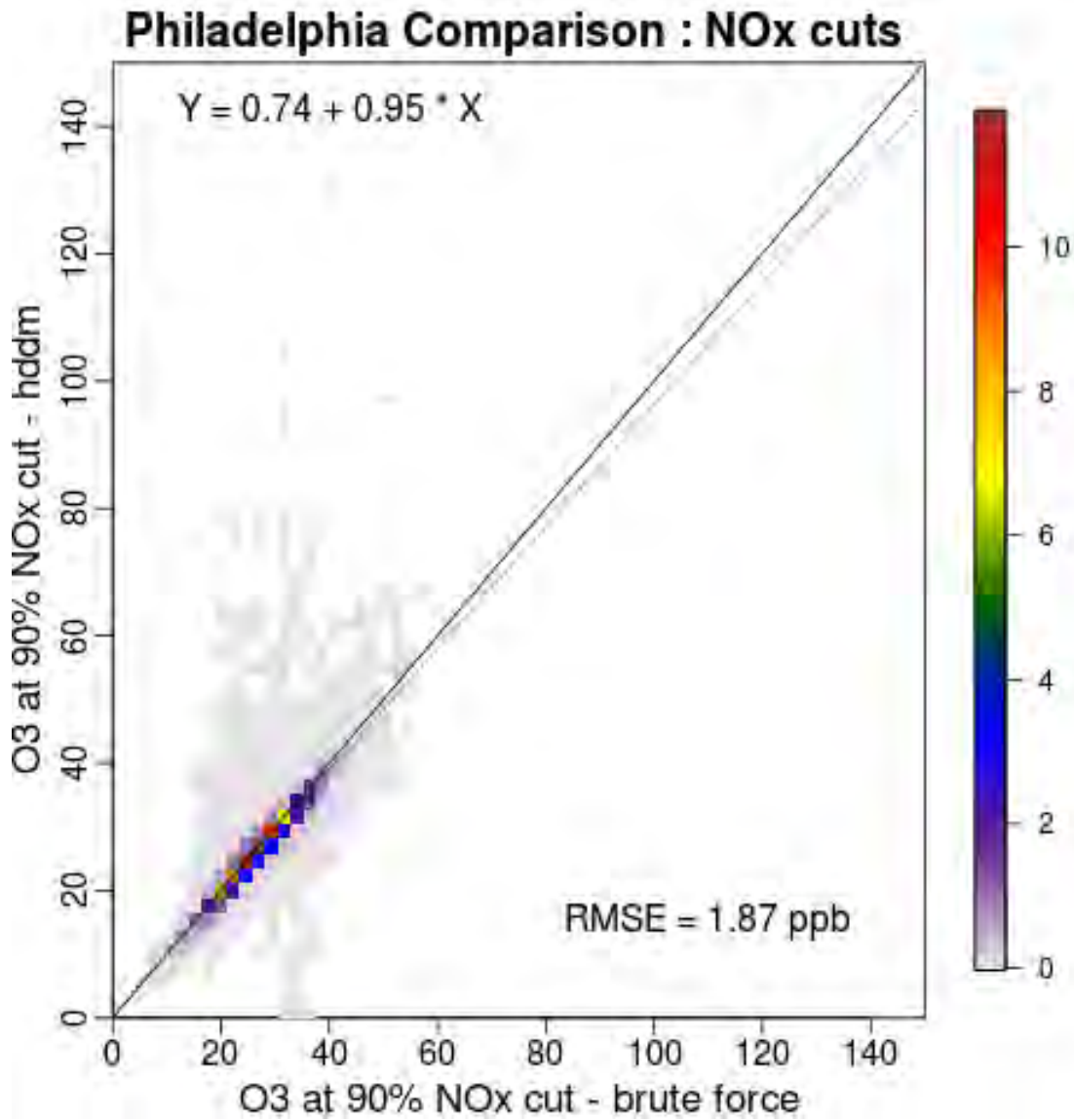
1
 2 **Figure 3C-59. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
 3 **cut conditions in Boston.**



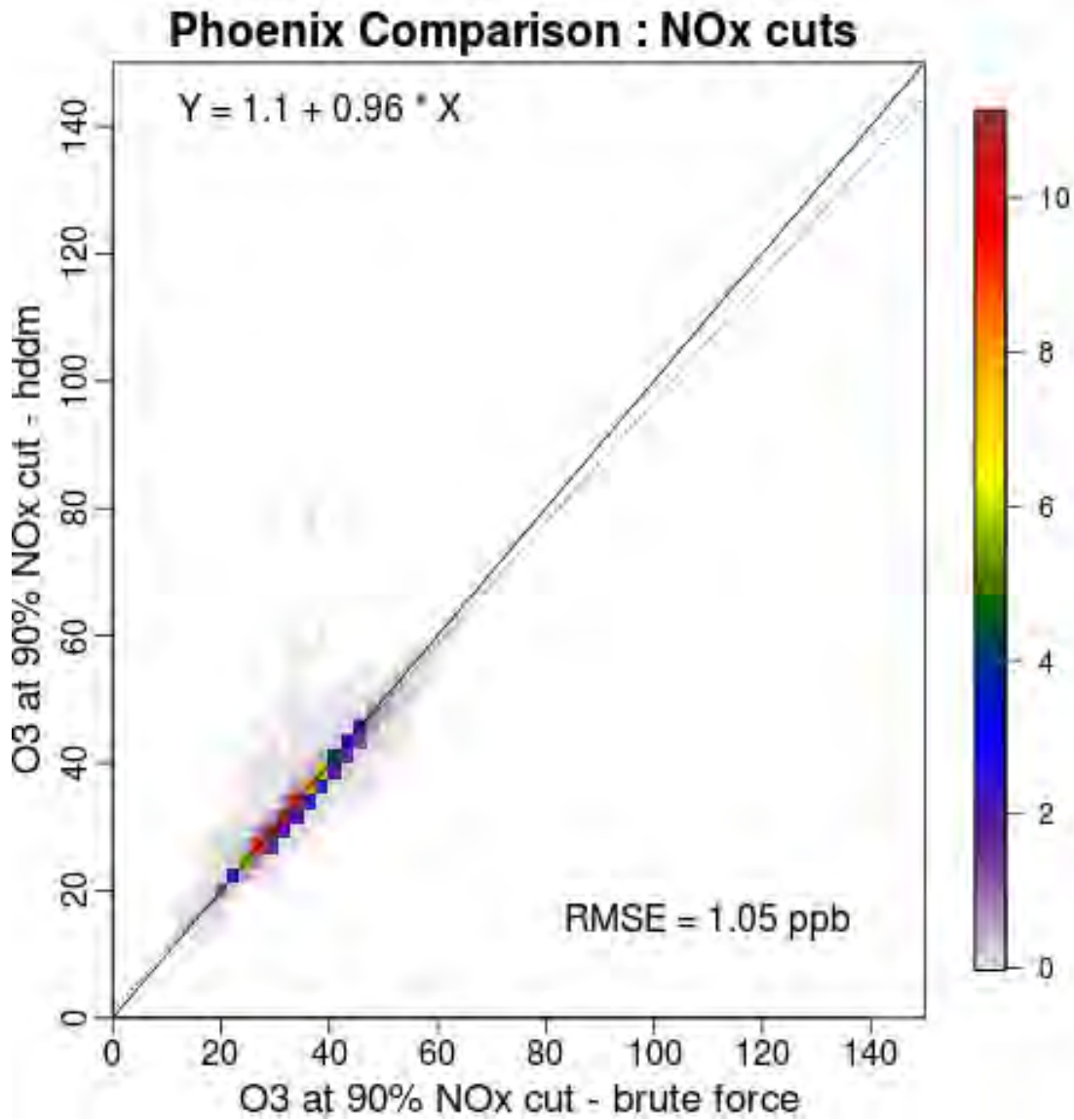
1
 2 **Figure 3C-60. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
 3 **cut conditions in Dallas.**



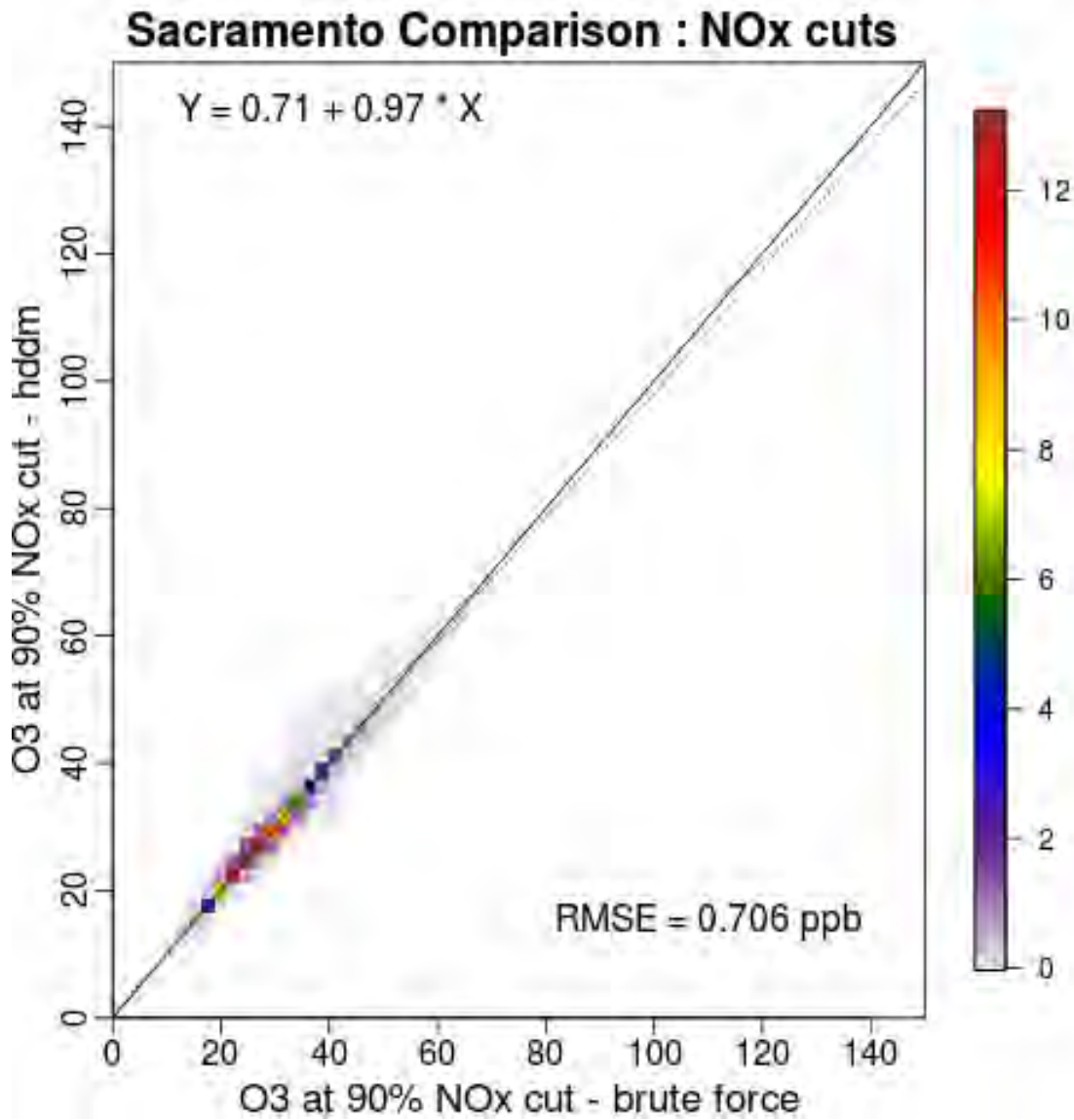
1
 2 **Figure 3C-61. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
 3 **cut conditions in Detroit.**



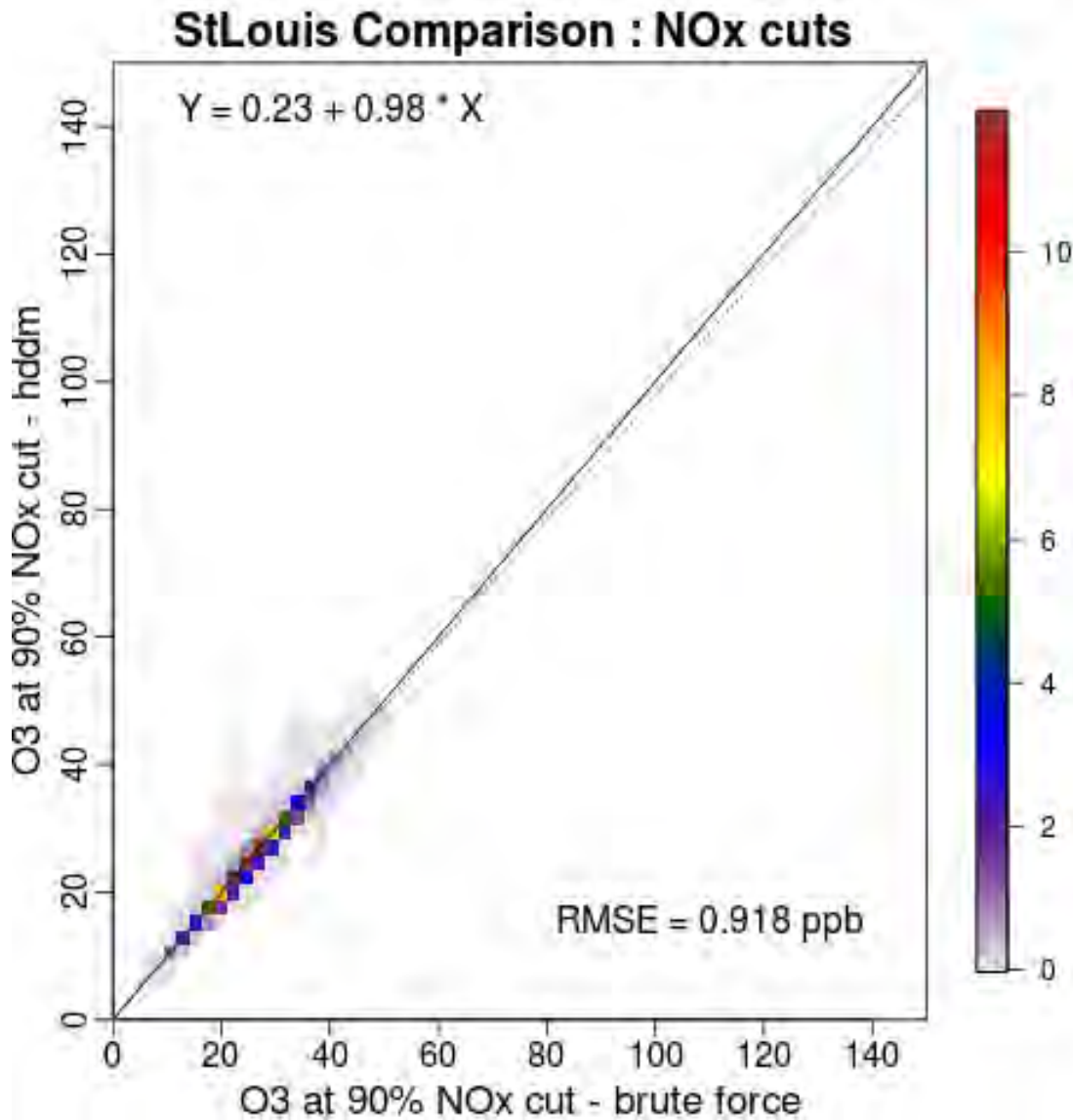
1
 2 **Figure 3C-62. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
 3 **cut conditions in Philadelphia.**



1
 2 **Figure 3C-63. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
 3 **cut conditions in Phoenix.**



1
 2 **Figure 3C-64. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
 3 **cut conditions in Sacramento.**



1
2 **Figure 3C-65. Comparison of brute force and 3-step HDDM O₃ estimates for 90% NO_x**
3 **cut conditions in St. Louis.**

4
5 **3C.5.2.2.2 Relationships between HDDM Sensitivities and Modeled O₃**
6 **Concentrations**

7 First and second order hourly O₃ sensitivities to NO_x emissions reductions were extracted
8 from the HDDM simulation for model grid cells that contained the O₃ monitors in the eight
9 urban study areas. Extracted data included modeled sensitivities at monitor locations for all
10 modeled hours in 2016. These sensitivities cannot be applied directly to observed values for two
11 reasons: 1) high modeled O₃ days/hours do not always occur concurrently with high observed O₃
12 days/hours and 2) the modeling time period includes only 2016 but the time period we are

1 analyzing in this assessment includes three full years of ambient air data, 2015-2017. As to the
2 first point, photochemical models are generally used in a relative sense for purposes of projecting
3 design values. In this manner, model predictions are “anchored” to ambient air measurements. In
4 general, the average response on high modeled days is used for this purpose. This allows for
5 more confidence in calculated results when “less than ideal model performance [occurs] on
6 individual days” (U.S. EPA, 2007). Similarly, for this analysis we believe it is appropriate to
7 account for the fact the model does not always perfectly agree with measurements and that
8 sensitivities from a low O₃ modeled day would not be appropriate to apply to a high O₃ measured
9 day (and vice-versa) even if they occur on the same calendar day. For this reason, a method was
10 developed to generalize the modeled site-, season-, and hour-specific sensitivities so that they
11 could be applied to ambient air data during 2015-2017.¹⁴

12 Simon et al. (2013) describe how first order sensitivities are generally well correlated
13 with hourly modeled O₃ concentrations and second order sensitivities are well correlated to first
14 order sensitivities. Based on their analysis, we create a separate linear regression for S_{NO_x} as a
15 function of hourly O₃ (i.e. $S_{NO_x} = m \times O_3 + b$) for every site, season¹⁵, and hour-of-the day
16 examined in this analysis. For instance, for summer 8-am hours at Detroit monitor site ID
17 260990009, S_{NO_x} and O₃ values from all 8-am hours in June-August 2016 are used to fit this
18 relationship. Similarly, S²_{NO_x} was calculated as a function of S_{NO_x}.

19 Comparisons between brute force and HDDM O₃ estimates shown in Figure 3C-50
20 through Figure 3C-65 demonstrate that for the vast majority of data points, HDDM replicates
21 brute force with minimal errors. These figures show a small number of instances, particularly for
22 Philadelphia, in which HDDM predicts very high hourly O₃ (> 100 ppb) while the brute force
23 emissions simulations for the 90% reduction show much lower O₃ (< 40 ppb). In these isolated
24 cases, base modeled O₃ is low due to NO_x titration and increases occur with reductions of NO_x.
25 The HDDM sensitivities for these few points appear to be too high to be applied over large
26 (>50%) emissions changes because of strongly nonlinear chemistry. However these extreme
27 cases are not relevant for this analysis, since the largest emissions reduction required for
28 Philadelphia was 53% to meet the air quality scenario for 65 ppb (Table 3C-19). The two urban
29 study areas requiring emission cuts larger than ~50%, Phoenix and Sacramento, both show much
30 better agreement between the 90% brute force and HDDM predictions (Figure 3C-63 and Figure
31 3C-64 respectively).

¹⁴ The 12 months modeled covered a variety of conditions such that we can use the results from this modeled time period in conjunction with the ambient data from the longer 3-year period for estimating responses and applying adjustments

¹⁵ Seasons are defined as follows: Winter = December, January, February; Spring = March, April, May; Summer = June, July, August; Fall = September, October, November.

1 For the 50% and 90% emissions reduction CAMx/HDDM simulation, regressions were
2 performed for first order NO_x sensitivities with modeled O₃ from the base HDDM simulation.
3 The regression technique was performed for the first and second order NO_x sensitivities from the
4 base run and the 50% emissions reduction and 90% emissions reduction simulations. The
5 sensitivities from the emissions reduction runs were fitted to hourly O₃ concentrations in the base
6 simulation. Simon et al. (2013) found that correlation coefficients using for sensitivities from
7 NO_x reduction simulations to base case O₃ concentrations were similar to those with O₃
8 concentrations from the NO_x reduction runs.

9 **3C.5.2.2.3 Application of Sensitivity Regressions to Ambient Air Data**

10 To apply the HDDM adjustments to observed data, sensitivities must be determined for
11 each hour from 2015-2017 at each site based on the linear relationship from the modeled data
12 and the observed O₃ concentration. The linear regression model also allows us to quantify the
13 standard error of each predicted sensitivity value at each hour and site.

14 Observed hourly O₃ from 2015-2017 at each monitor location was adjusted by applying
15 incrementally increasing emissions reductions using equations (3C-4) through (3C-8) and
16 recalculating MDA8 values for incrementally increasing emissions reductions until an emissions
17 level is reached for which all monitors in an urban study area achieved design values at the level of
18 the air quality scenario being evaluated (design values of 75, 70, or 65 ppb). Therefore, all
19 monitors within an urban study area were treated as responding to the same percentage reduction
20 in NO_x emissions.

21 The precursor reductions used to estimate spatial and temporal patterns of O₃
22 concentrations for the three air quality scenarios were NO_x-only reductions. We focused on
23 NO_x-only reductions in light of several key findings from analyses for the 2014 HREA that
24 explored the use of both NO_x and VOC reductions versus NO_x-only scenarios (2014 HREA,
25 Appendix 4D). There were several key findings from that comparison. First, in most of the urban
26 study areas, the NO_x/VOC scenario did not affect O₃ response at the monitor having the highest
27 design value in such a way to reduce the total required emissions cuts. Further, evidence in the
28 literature has shown that locations in the U.S. have gotten more NO_x-limited since 2007 (the
29 year modeled in the 2014 HREA) (Jin et al., 2017, Laughner and Cohen, 2019) and thus VOC
30 reductions would be expected to have less impact on resulting O₃ concentrations in our scenarios
31 for the 2016 modeling used here than they had in the previous analysis. Finally, the two areas
32 (Denver and Chicago) in which VOC emissions had the most impact in the 2014 HREA were not
33 included in the current analysis. For these reasons, NO_x-only reductions were determined to be

1 the most appropriate scenarios for this analysis. The final emissions reductions that were applied
2 in each urban study area are given in Table 3C-19 below.¹⁶

3

4 **Table 3C-19. Percent emissions changes used for each urban study area to just meet each**
5 **of the air quality scenarios evaluated.**

Urban Study Area	75 ppb	70 ppb	65 ppb
Atlanta	0%	25%	44%
Boston	+7%	14%	40%
Dallas	15%	32%	45%
Detroit	+18%	21%	47%
Philadelphia	23%	43%	53%
Phoenix	14%	49%	68%
Sacramento	45%	58%	72%
Saint Louis	+11%	13%	38%

6

7 The 2014 HREA included a thorough analysis of the standard error associated with the
8 predicted O₃ concentrations produced using the HDDM adjustment approach. This analysis
9 found that while the error in predicted values varied by site and air quality scenario being
10 evaluated, the magnitudes were small (<1.5 ppb in most cases). We did not repeat such an
11 analysis here given the small magnitude of the standard errors found in this previous assessment.

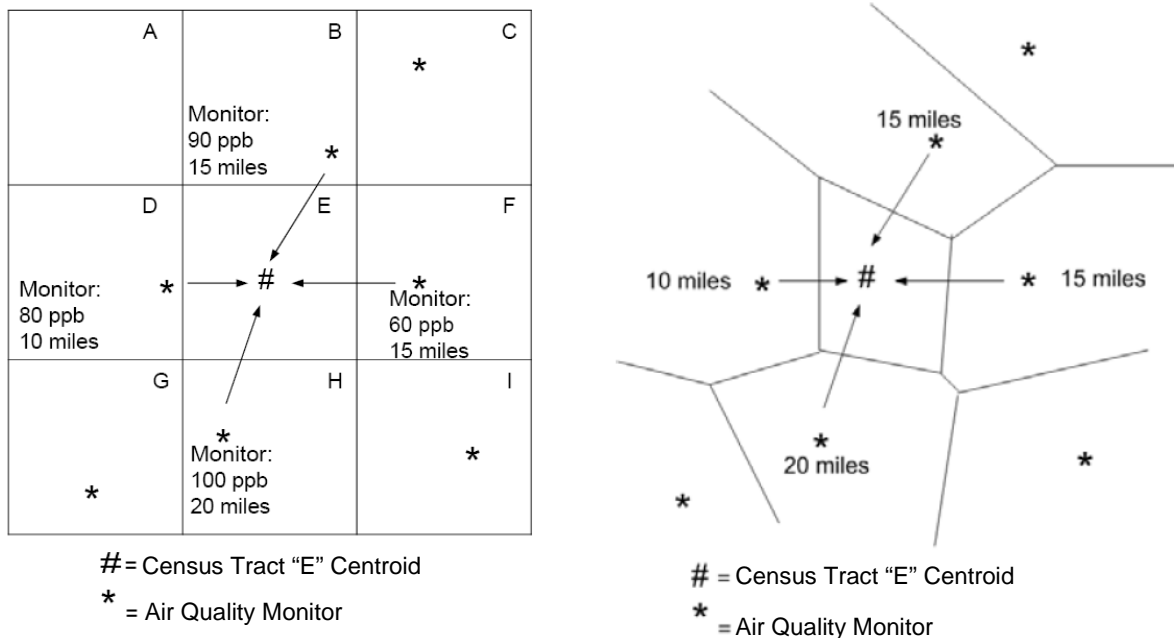
12 **3C.6 INTERPOLATION OF ADJUSTED AIR QUALITY USING** 13 **VORONOI NEIGHBOR AVERAGING**

14 The APEX exposure model uses spatial fields of ambient air quality concentrations at
15 variable spatial scales (e.g., 500 m regular grid, census tract centroid) as inputs, but requires that
16 there be no missing values. The final air quality data used as inputs to the APEX model were the
17 hourly O₃ concentrations at monitoring sites adjusted using CAMx/HDDM, then interpolated to
18 each census tract centroid in the eight urban study areas using the Voronoi Neighbor Averaging
19 (VNA; Gold et al., 1997; Chen et al., 2004) technique described below. A cross-validation
20 analysis supporting the use of the VNA technique for the creation of hourly O₃ spatial fields was
21 conducted in the 2015 review (U.S. EPA, 2014; Appendix 4A).

¹⁶ Note that these emissions reductions and broad nationwide emission cuts are not intended to represent recommended control scenarios since they would not be the most efficient method for achieving a particular standard in many areas.

1 The following paragraphs provide a numerical example of VNA used to estimate an O₃
 2 concentration value for census tract “E” in Figure 3C-66 below.

3 The first step in the VNA technique is to identify the set of nearest monitors for each
 4 census tract. The left-hand panel of Figure 3C-66 presents a numerical example with nine census
 5 tracts (squares) and seven monitoring sites (stars), with the focus on identifying the set of nearest
 6 neighboring sites to census tract “E” in the center of the panel. The Delaunay triangulation
 7 algorithm identifies the set of nearest neighboring monitors by drawing a set of polygons called
 8 the “Voronoi diagram” around the census tract “E” centroid and each of the monitoring sites.
 9 Voronoi diagrams have the special property that each edge of each of the polygons are the same
 10 distance from the two closest points, as shown in the right-hand panel of Figure 3C-66.
 11



12
 13 **Figure 3C-66. Numerical example of the Voronoi Neighbor Averaging (VNA) technique.**

14
 15 The VNA technique then chooses the monitoring sites whose polygons share a boundary
 16 with the census tract “E” centroid. These monitors are the “Voronoi neighbors”, which are used
 17 to estimate the concentration value for census tract “E”. The VNA estimate of the concentration
 18 value in census tract “E” is the inverse distance squared weighted average of the four monitored
 19 concentrations. The further the monitor is from the center of census tract “E”, the smaller the
 20 weight. For example, the weight for the monitor in census tract “D” 10 miles from the census
 21 tract “E” centroid is calculated as follows:

22

$$\frac{1/10^2}{1/10^2 + 1/15^2 + 1/15^2 + 1/20^2} = 0.4675$$

Equation (3C-24)

The weights for the other monitors are calculated in a similar fashion. The final VNA estimate for census tract “E” is calculated as follows:

$$VNA(E) = 0.4675 * 80 + 0.2078 * 90 + 0.2078 * 60 + 0.1169 * 100 = 80.3 \text{ ppb}$$

Equation (3C-25)

The adjusted hourly O₃ concentrations in the eight urban study areas were used to calculate VNA estimates for approximately 9,725 census tracts * 26,304 hours * 3 air quality scenarios ≈ 767 million values. The computations were executed using the R statistical computing program (R Core Team, 2018), with the Delaunay triangulation algorithm implemented in the “deldir” package (Turner, 2018).

3C.7 RESULTS FOR URBAN STUDY AREAS

3C.7.1 Design Values

Table 3C-20 through Table 3C-27 provide the design values for ambient monitoring sites in each of the eight urban study areas for 2015-2017 based on the observed data, and based on the adjusted O₃ concentrations for the three air quality scenarios (i.e., air quality meeting the current standard of 70 ppb, and air quality meeting two alternative levels of 75 ppb and 65 ppb). In each table, the highest design value for each scenario is displayed in bold text. The data in these tables demonstrate that high O₃ values at monitors within some urban study areas respond differently to reductions in NO_x emissions.

In five of the eight urban study areas, the monitor with the highest observed design value remained the highest when the air quality was adjusted in each of the three air quality scenarios. For example, Atlanta monitor 131210055 had the highest 2015-2017 design value of 75 ppb, as well as design values of 70 ppb and 65 ppb for the 70 ppb and 65 ppb scenarios, respectively. The other study areas where the same monitor had the highest design value in the observations as well as the 75 ppb, 70 ppb, and 65 ppb scenarios were Dallas (481210034), Detroit (261630019), Sacramento (060570005), and St. Louis (291831002).

Boston and Philadelphia saw shifts in the highest monitor as a result of the adjustments. In Boston, monitor 250051004 in Fall River, MA was highest in the observations and following the upward adjustment to meet 75 ppb. Monitor 250051004 and two other monitors (440090007 in Narragansett, RI and 440090007 east of Providence, RI) had design values of 70 ppb for the adjustment to meet the current standard. After the final adjustment for the 65 ppb scenario, the highest design value occurred at the Narragansett monitor. In Philadelphia, monitor 420170012 near Trenton, NJ was highest in the observations. However, following each of the adjustments to

1 75 ppb, 70 ppb and 65 ppb, the location of the highest monitor shifted slightly west to monitor
 2 421010024 (east of downtown Philadelphia).

3 The pattern for Phoenix was unique among the eight urban study areas. One monitor
 4 (040139997) was consistently high in the observations and for all adjusted levels. However, two
 5 other monitors were equally as high in the observations (040132005; 040131003 – also high at
 6 75 ppb) but responded more strongly to the applied NO_x reductions. While monitors 040132005
 7 and 040131003 are slightly removed from downtown Phoenix (near Pinnacle Peak to the
 8 northeast and Mesa to the southeast, respectively), monitor 040139997 is closer the center of the
 9 Phoenix metropolitan area. This location is likely near higher concentrations of urban NO_x
 10 sources, making this monitor slightly less responsive to the NO_x emissions adjustments.

11

12 **Table 3C-20. 2015-2017 design values for monitors in the Atlanta study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
130590002	64	64	59	54
130670003	67	67	62	57
130770002	63	63	59	54
130850001	65	65	61	56
130890002	71	71	66	59
130970004	69	69	64	58
131210055	75^A	75	70	65
131350002	71	71	66	60
131510002	71	71	65	59
132230003 ^B	N/A	N/A	N/A	N/A
132319991	67	67	62	56
132470001	69	69	64	57
^A Highest DV for each scenario is displayed in bold. ^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.				

13

14

1 **Table 3C-21. 2015-2017 design values for monitors in the Boston study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
090159991	70	72	68	61
250010002 ^B	N/A	N/A	N/A	N/A
250051004	73^A	75	70	63
250051006	69	71	68	62
250092006	66	68	65	61
250094005	65	67	64	59
250095005	62	64	61	56
250170009	64	66	62	57
250213003	70	72	68	62
250230005	68	70	65	60
250250042	61	62	61	58
250270015	65	67	64	59
250270024	66	68	64	59
330012004	59	61	57	53
330111011	62	64	61	57
330115001	67	65	65	60
330131007	63	64	61	56
330150014	63	65	61	57
330150016	66	68	65	59
330150018	65	67	64	59
440030002	72	74	70	63
440071010	70	72	68	62
440090007	71	73	70	65
^A Highest DV for each scenario is displayed in bold.				
^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.				

2
3

1 **Table 3C-22. 2015-2017 design values for monitors in the Dallas study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
400130380 ^B	N/A	N/A	N/A	N/A
480850005	74	72	67	63
481130069	74	72	68	63
481130075	74	72	68	63
481130087	64	62	58	54
481210034	79^A	75	70	65
481211032	74	71	66	62
481390016	65	63	60	56
481391044	64	61	58	55
482210001	67	65	61	58
482311006	62	60	56	53
482510003	73	70	65	60
482570005	61	59	56	53
483491051	63	61	58	56
483670081	70	67	63	59
483970001	66	63	60	57
484390075	71	69	65	60
484391002	72	70	67	62
484392003	73	71	67	62
484393009	75	73	69	64
484393011	67	65	61	57
^A Highest DV for each scenario is displayed in bold.				
^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.				

2

3 **Table 3C-23. 2015-2017 design values for monitors in the Detroit study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
260490021	67	70	65	60
260492001	67	71	65	59
260910007	66	70	64	58
260990009	71	73	69	63
260991003	66	68	65	61
261250001	70	72	68	63
261470005	71	74	69	64
261610008	67	69	65	60
261619991	69	72	66	59
261630001	66	69	65	60
261630019	73^A	75	70	65
261630093 ^B	N/A	N/A	N/A	N/A
261630094 ^B	N/A	N/A	N/A	N/A
^A Highest DV for each scenario is displayed in bold.				
^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.				

1

2 **Table 3C-24. 2015-2017 design values for monitors in the Philadelphia study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
100010002	66	62	57	53
100031007	67	64	59	55
100031010	74	70	65	60
100031013	71	67	63	58
100032004	72	68	63	58
240150003	74	70	64	59
340010006	64	60	55	51
340070002	77	74	68	63
340071001	68	64	60	56
340110007	66	62	56	53
340150002	74	70	68	60
420110006	66	63	57	53
420110011	70	67	61	58
420170012	80^A	75	69	64
420290100	73	69	63	58
420450002	71	69	64	60
420910013	72	69	64	59
421010004 ^B	N/A	N/A	N/A	N/A
421010024	78	75	70	65
421010048	76	72	67	63

^A Highest DV for each scenario is displayed in bold.
^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.

3

4

1 **Table 3C-25. 2015-2017 design values for monitors in the Phoenix study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
040130019	74	74	68	62
040131003	76	75	69	63
040131004	75	74	69	63
040131010	74	74	69	62
040132001	68	67	64	59
040132005	76^A	74	67	60
040133002	72	72	67	62
040133003	69	68	63	59
040134003	70	69	65	60
040134004	71	70	64	59
040134005 ^B	N/A	N/A	N/A	N/A
040134008	70	69	64	58
040134010	68	68	63	59
040134011	63	62	58	54
040135100 ^B	N/A	N/A	N/A	N/A
040137003	66	65	60	56
040137020	72	72	67	61
040137021	75	74	67	60
040137022	75	74	67	60
040137024	72	71	66	60
040139508	73	72	66	61
040139702	72	71	64	57
040139704	70	69	63	57
040139706	68	68	63	57
040139997	76	75	70	65
040213001	74	73	66	60
040213003	66	65	61	57
040213007	68	67	62	59
040217001	65	64	59	55
040218001	73	72	65	60
^A Highest DV for each scenario is displayed in bold.				
^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.				

2
3

1 **Table 3C-26. 2015-2017 design values for monitors in the Sacramento study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
060170010	83	71	65	59
060170012 ^B	N/A	N/A	N/A	N/A
060170020	80	69	63	56
060570005	86^A	75	70	65
060570007 ^B	N/A	N/A	N/A	N/A
060610003	84	72	66	58
060610004	77	67	62	56
060610006	79	71	65	58
060611004	64	61	60	58
060612002	75	67	61	54
060670002	78	70	65	58
060670006	77	71	66	59
060670010	69	63	59	54
060670011	68	61	56	50
060670012	82	72	66	59
060670014 ^B	N/A	N/A	N/A	N/A
060675003	78	69	63	57
061010003	64	56	52	47
061010004 ^B	N/A	N/A	N/A	N/A
061130004	63	55	52	47
061131003	69	60	55	50
^A Highest DV for each scenario is displayed in bold.				
^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.				

2
3

1 **Table 3C-27. 2015-2017 design values for monitors in the St. Louis study area.**

Monitor ID	Observed	75 ppb	70 ppb	65 ppb
170830117 ^B	N/A	N/A	N/A	N/A
170831001 ^B	N/A	N/A	N/A	N/A
171170002	65	68	63	57
171190008	69	72	67	62
171191009	68	71	66	61
171193007	70	73	68	62
171199991	67	70	65	58
171630010	68	71	67	61
290990019	68	71	66	59
291130003 ^B	N/A	N/A	N/A	N/A
291130004 ^B	N/A	N/A	N/A	N/A
291831002	72^A	75	70	65
291831004	70	73	67	62
291890005	65	67	63	58
291890014	69	72	67	62
295100085	66	69	65	61
^A Highest DV for each scenario is displayed in bold. ^B Monitor used to develop AQ surfaces but DVs not calculated because data were incomplete.				

2

3 **3C.7.2 Distribution of Hourly O₃ Concentrations**

4 Figure 3C-67 through Figure 3C-74 display diurnal boxplots of hourly O₃ concentrations
 5 for 2015-2017 at monitor locations in each urban study area. For each hour of the day, the
 6 rectangular box represents the 25th and 75th percentiles of the distribution, with a solid line
 7 representing the median of the distribution through the center. Each box has “whiskers” which
 8 extend up to 1.5 times the interquartile range (i.e., the 75th percentile minus the 25th percentile)
 9 from the box, and dots which represent outlier values. Black boxplots represent observed hourly
 10 O₃ concentrations, while blue boxplots represent hourly O₃ concentrations adjusted to meet the
 11 current standard of 70 ppb. Red boxplots represent hourly O₃ concentrations adjusted for the 75
 12 ppb¹⁷ scenario, and green boxplots represent hourly O₃ concentrations adjusted for the 65 ppb
 13 scenario.

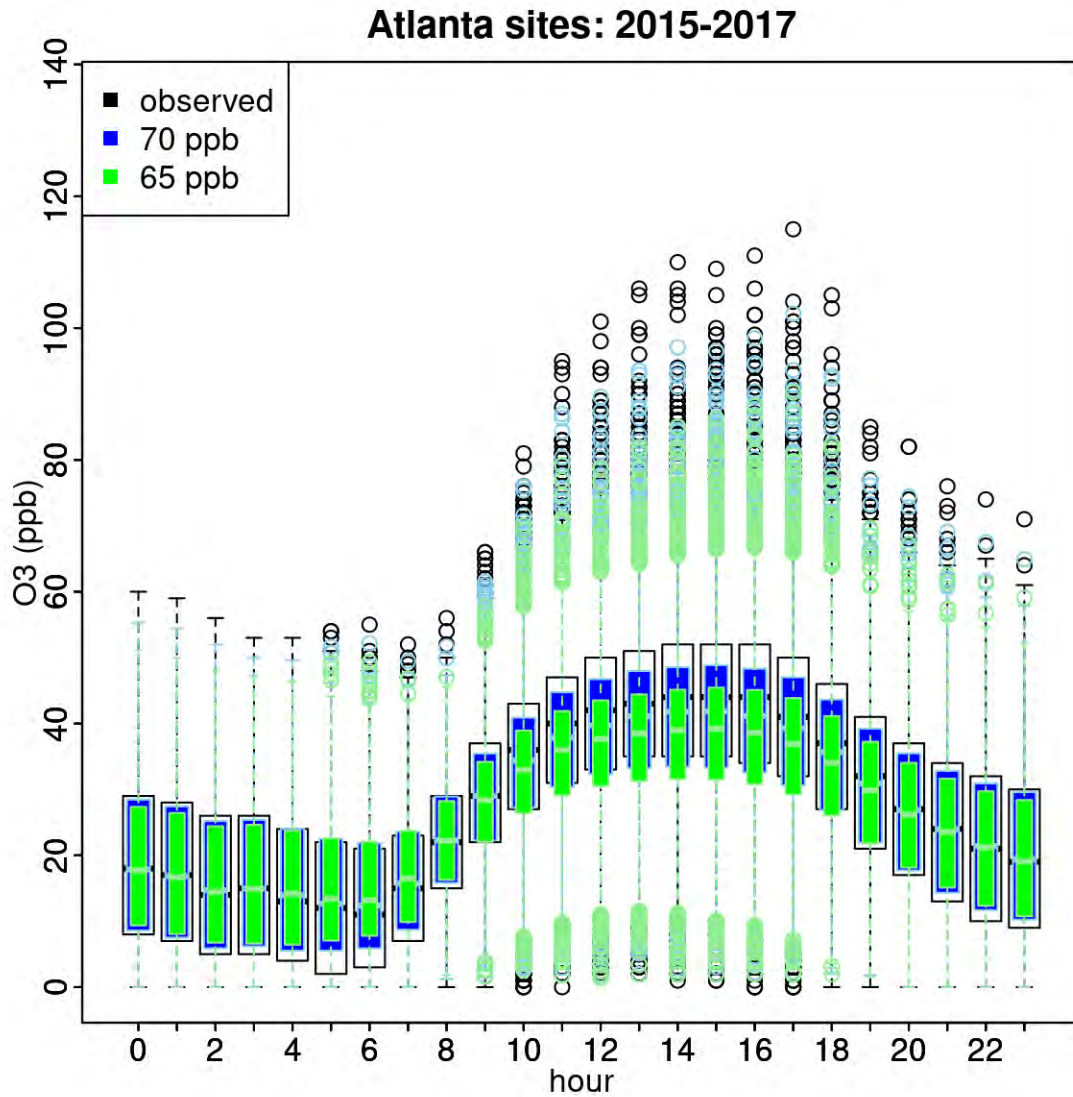
14 The boxplots include the observed O₃ concentrations as well as the concentrations
 15 adjusted to just meet the current standard and the two alternative air quality scenarios. Note that
 16 these plots include data from all sites in the study area, and thus the plots provide the overall
 17 distribution of O₃ at both the urban core sites and the downwind suburban sites. The hourly plots

¹⁷ No adjusted values are shown for the 75 ppb scenario for Atlanta because the observed design value was 75 ppb, and thus no adjustments were made to the hourly O₃ concentrations for that scenario.

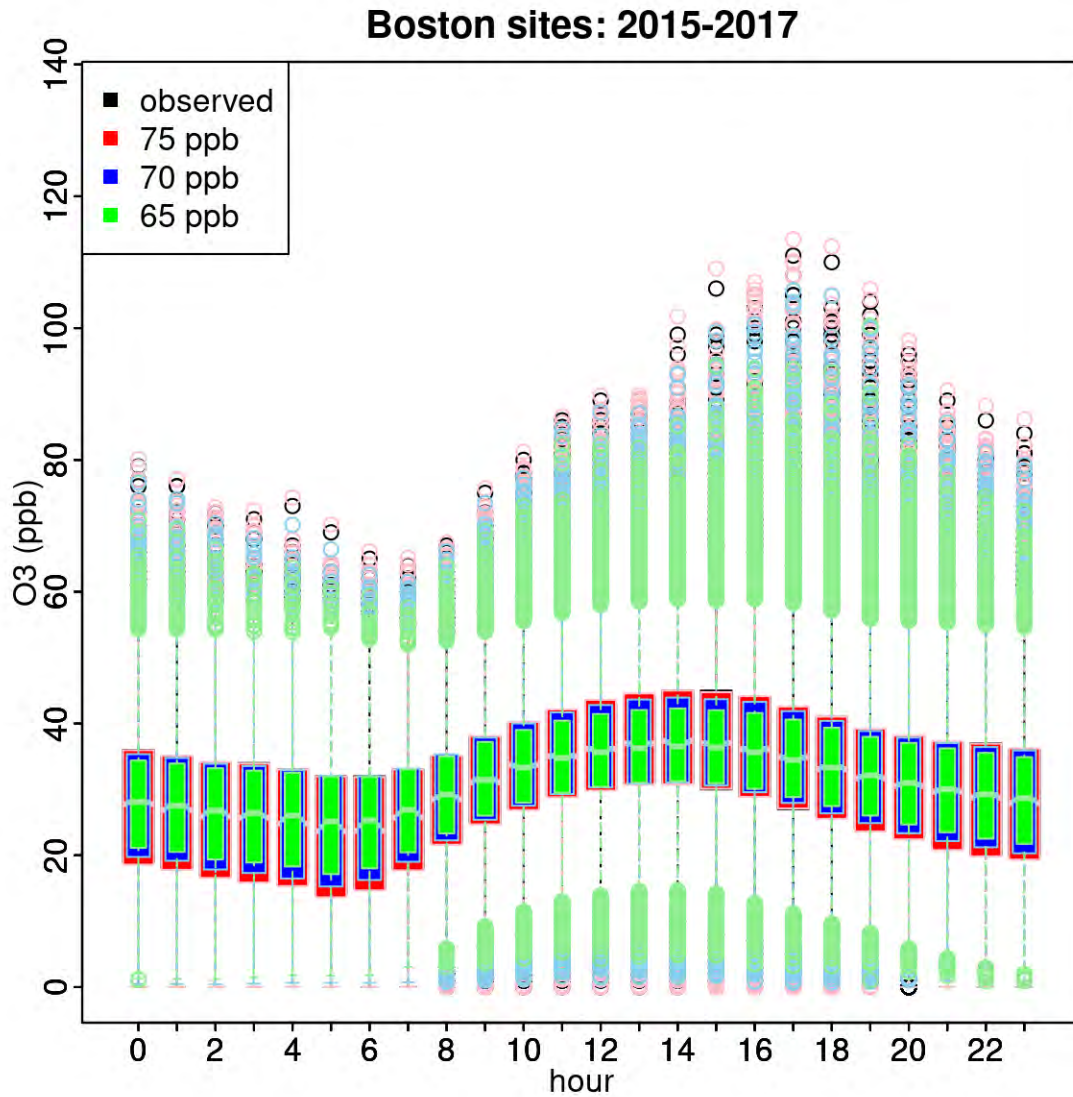
1 show similar patterns in most of the urban study areas. O₃ concentrations during daytime hours
2 decrease from observed values (black) to values adjusted to meet the current standard of 70 ppb
3 (blue) and decrease further under the alternative scenario of 65 ppb (green). These daytime
4 decreases are mainly seen on high O₃ days represented by outlier dots extending above the box
5 and whiskers. Some study areas had observed 2015-2017 design values already meeting the
6 alternative scenario of 75 ppb, therefore some plots show increases in O₃ concentrations while
7 other study areas show decreases in O₃ concentrations for the 75 ppb scenario.

8 In some urban study areas O₃ concentrations on the mid-range days, represented by the
9 25th – 75th percentile boxes, remained fairly constant (e.g. Boston) while in other urban study
10 areas O₃ on mid-range days decreased (e.g. Atlanta). Although daytime O₃ decreased,
11 concentrations during morning rush-hour period generally increase. These increases are
12 associated with VOC-limited and NO_x titration conditions near NO_x sources during rush-hour
13 periods. Reducing NO_x under these conditions results in less O₃ titration and thus increases O₃
14 concentrations. Nighttime increases in O₃ as a results of NO_x reductions are often seen to a lesser
15 extent than morning rush-hour period increases. Collectively these features generally lead to a
16 flattening of the diurnal O₃ pattern with smaller differences between daytime and nighttime
17 concentrations as NO_x emissions are reduced. Urban study areas that required more substantial
18 NO_x reductions for the 65 ppb scenario generally had more pronounced patterns of decreases in
19 daytime O₃ and increases in nighttime O₃ leading to a flatter diurnal O₃ pattern (e.g., Sacramento
20 in Figure 3C-73).

21 Figure 3C-75 through Figure 3C-82 display the same information as Figure 3C-67
22 through Figure 3C-74 but for monthly rather than diurnal distributions. Similar to the diurnal
23 plots, the seasonal distributions become flatter when adjusted to meet the 70 ppb and 65 ppb
24 scenarios, especially on the highest O₃ days. This is due to more O₃ decreases during summer
25 months and more O₃ increases in winter months. The O₃ increases in the winter are consistent
26 with the understanding that solar insolation rates are lower in the winter reducing total
27 photochemical activity and shifting the net effect of NO_x emissions on O₃ which can both create
28 O₃ through photochemical pathways and destroy O₃ through titration. In addition, the decreases
29 on the highest O₃ days and increases on the lowest O₃ days show a visible compression of the O₃
30 distribution in these plots, similar to what was seen in the diurnal plots.

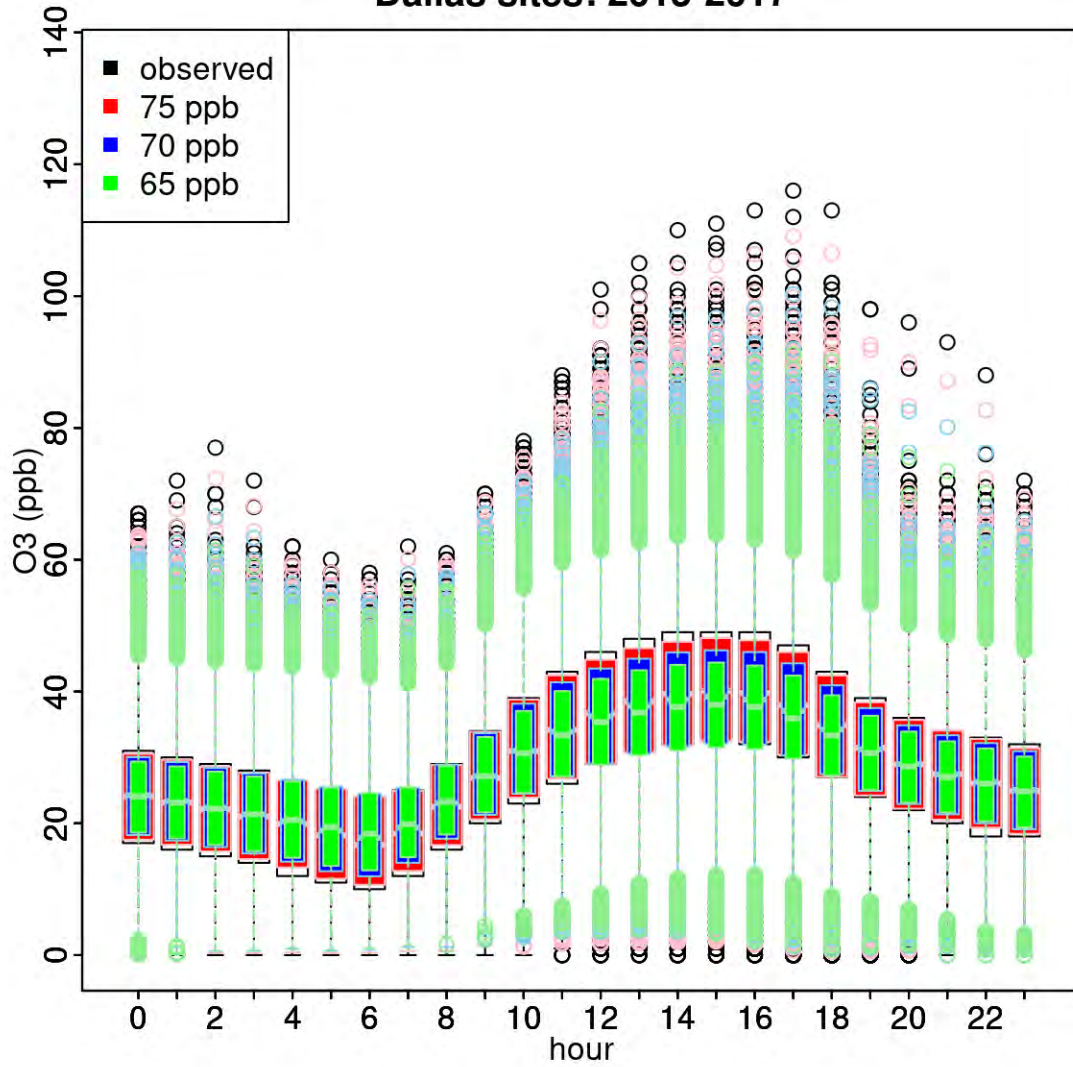


1
 2 **Figure 3C-67. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
 3 **Atlanta study area.** Note: Observed concentrations in this area have a design
 4 value of 75 ppb.

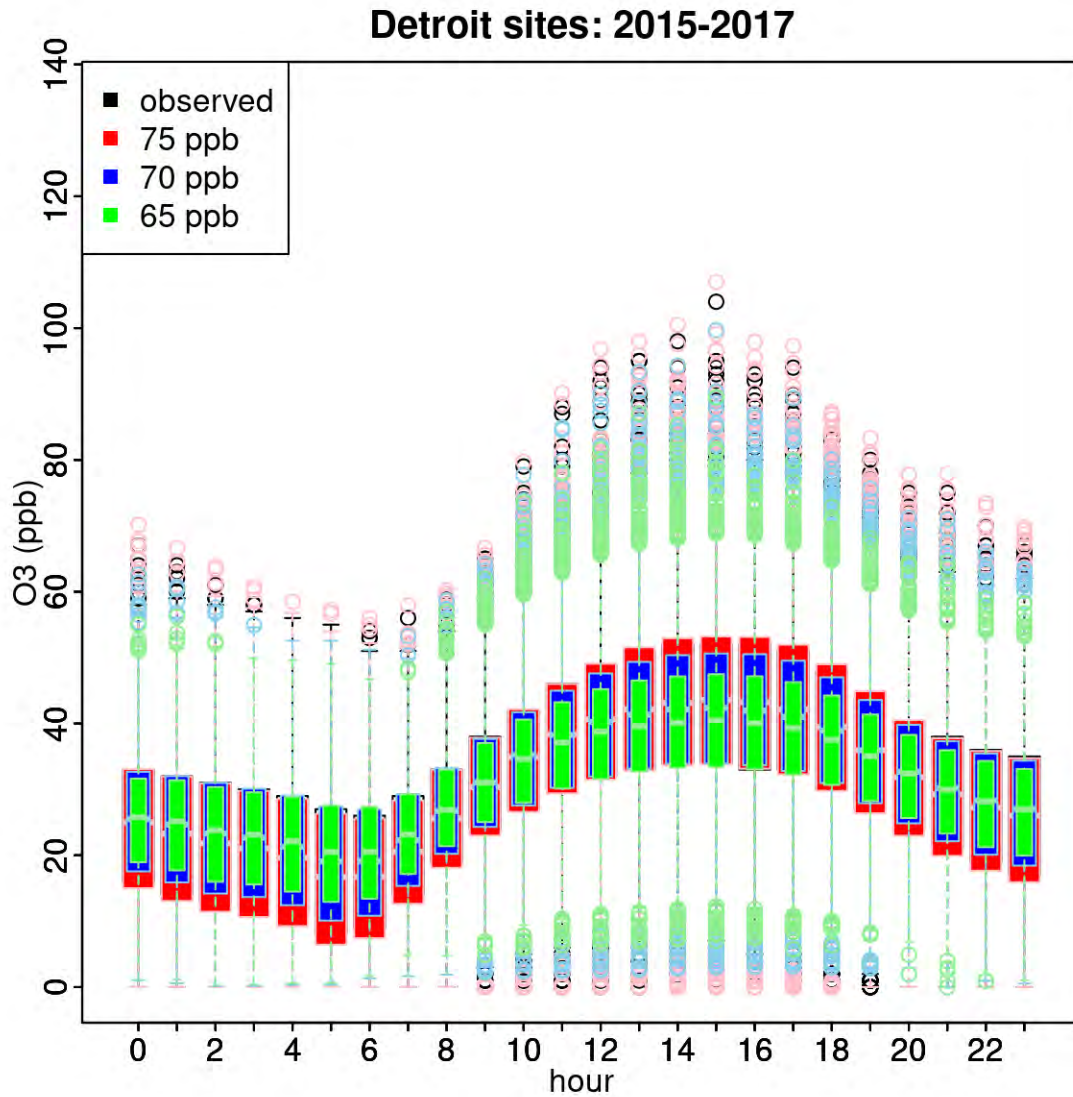


1
 2 **Figure 3C-68. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
 3 **Boston study area.**

Dallas sites: 2015-2017

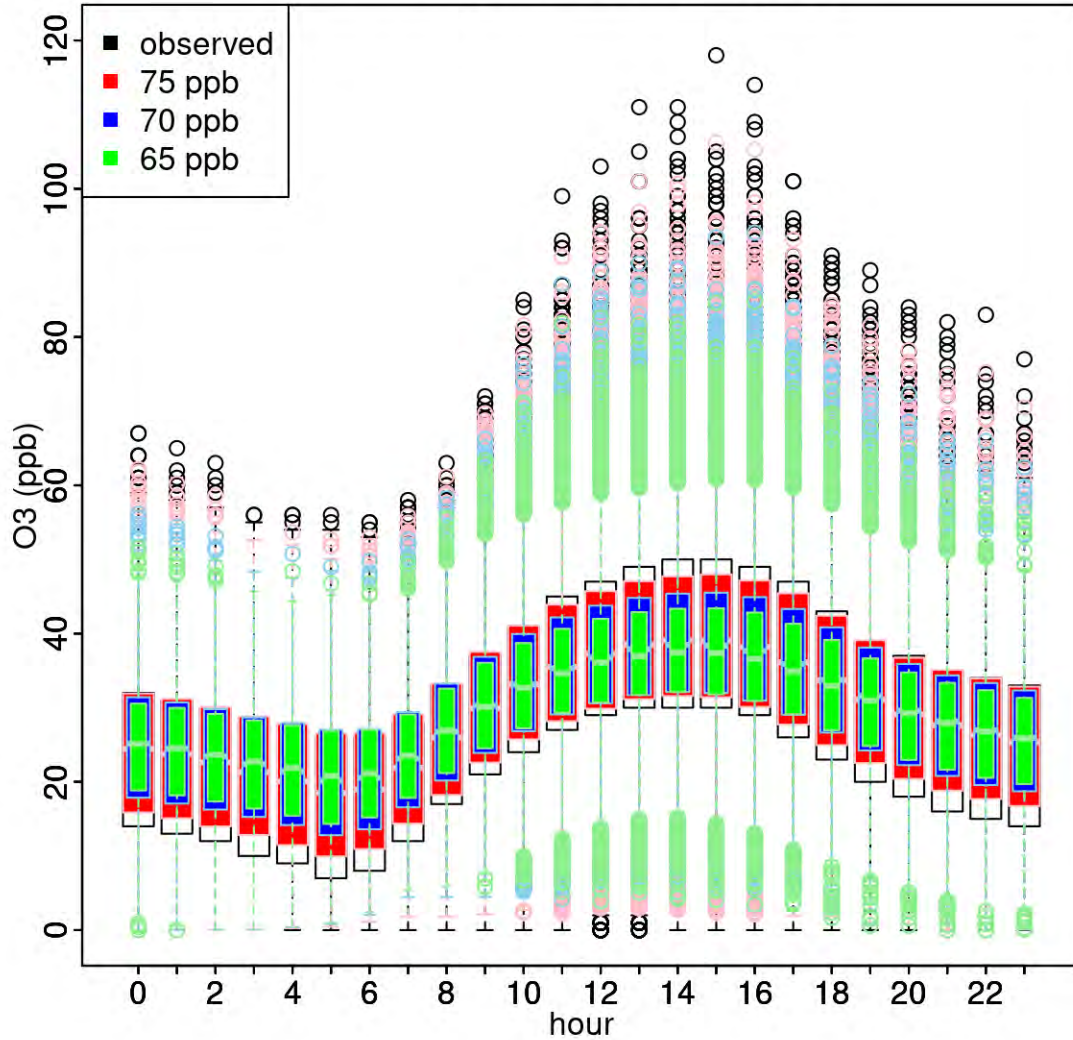


1
2 **Figure 3C-69. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Dallas study area.**



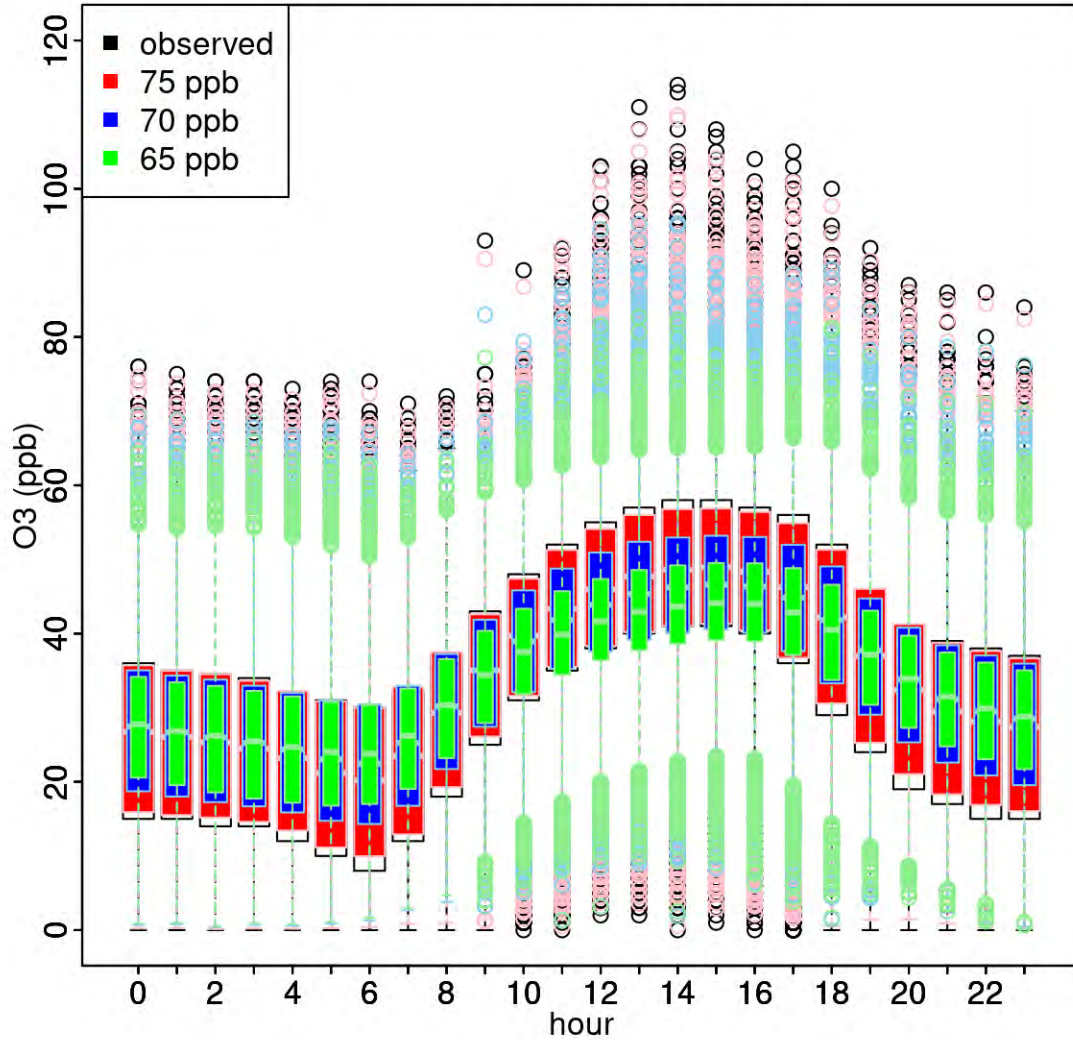
1
 2 **Figure 3C-70. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
 3 **Detroit study area.**

Philadelphia sites: 2015-2017



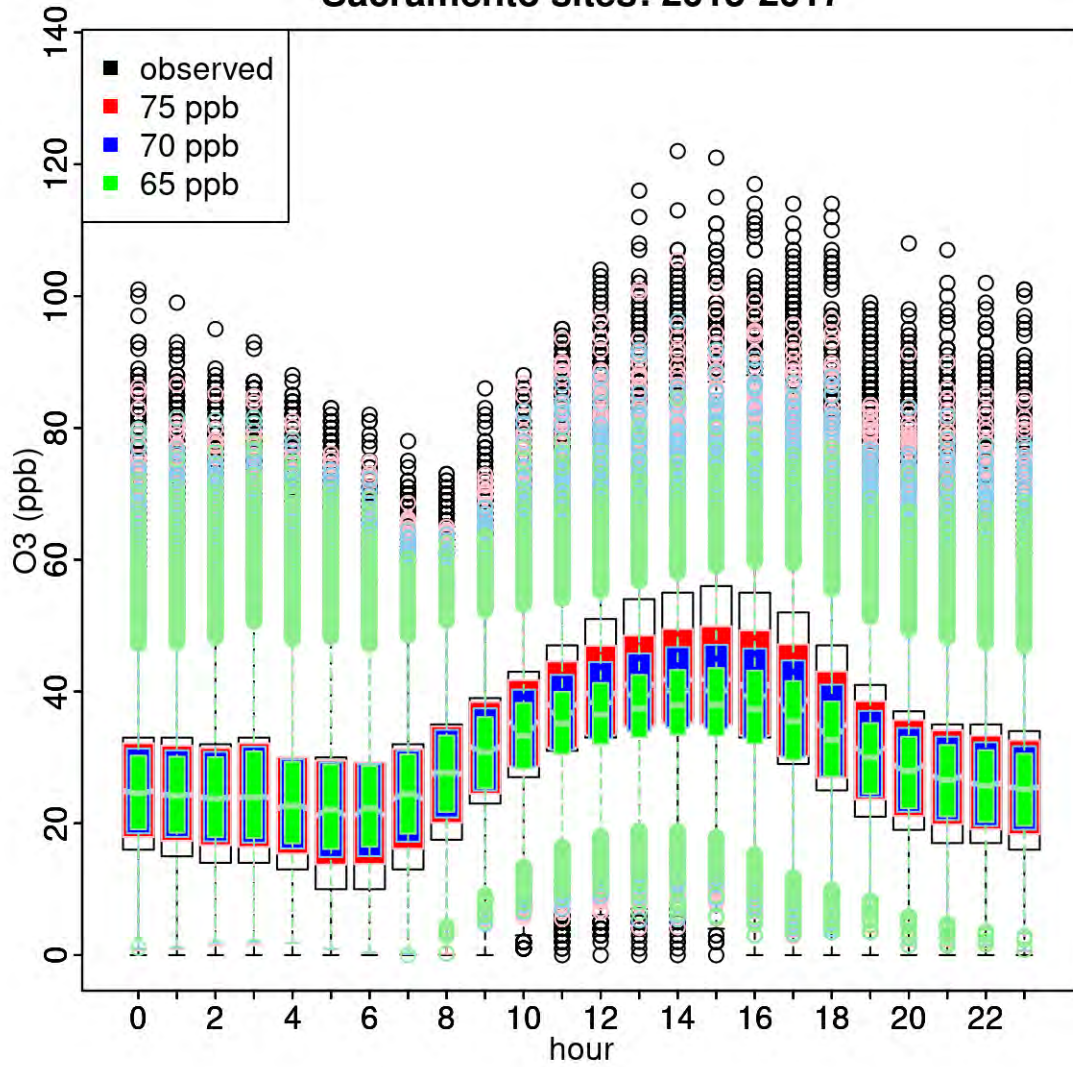
1
2 **Figure 3C-71. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Philadelphia study area.**

Phoenix sites: 2015-2017

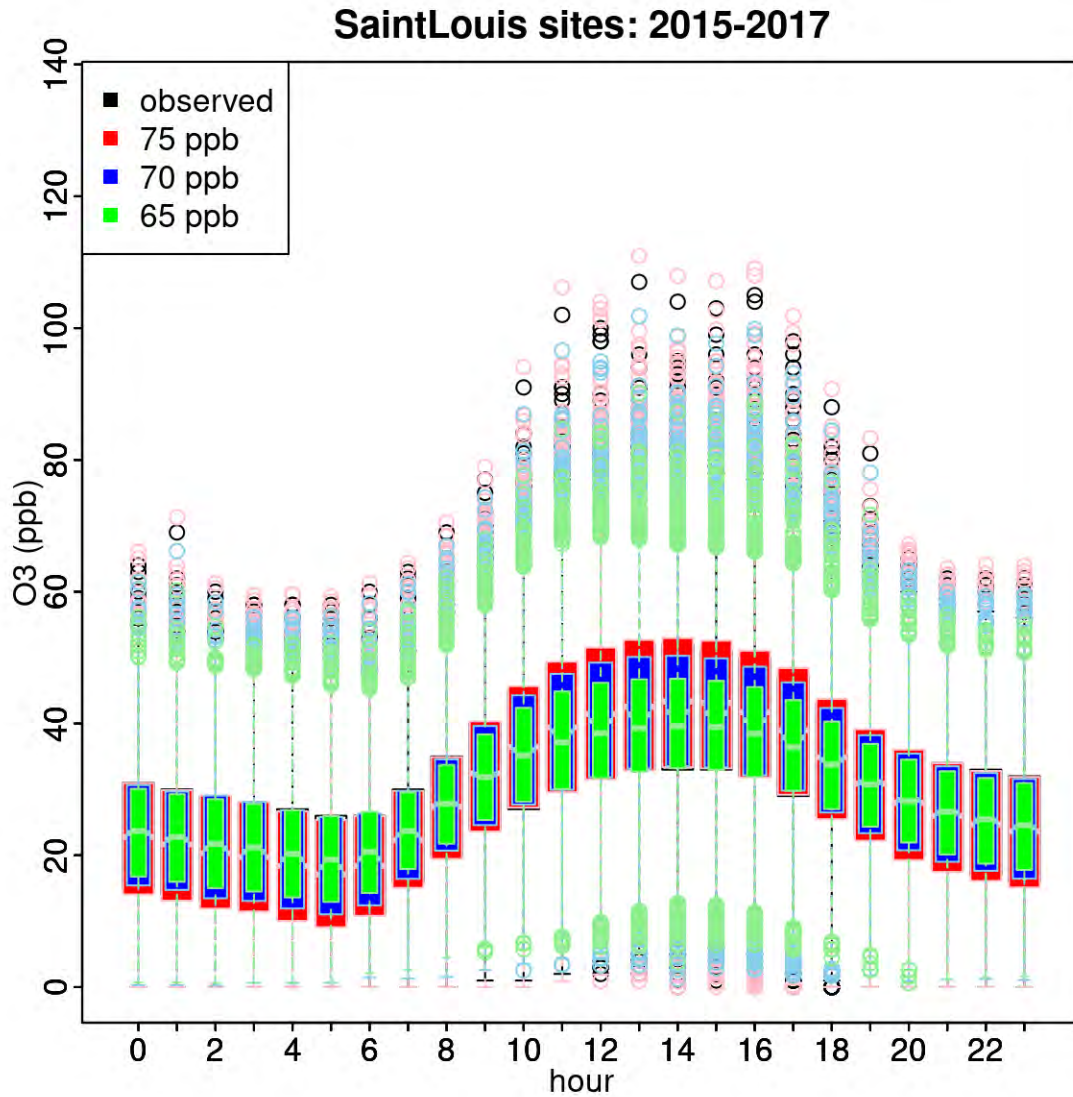


1
2 **Figure 3C-72. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Phoenix study area.**

Sacramento sites: 2015-2017

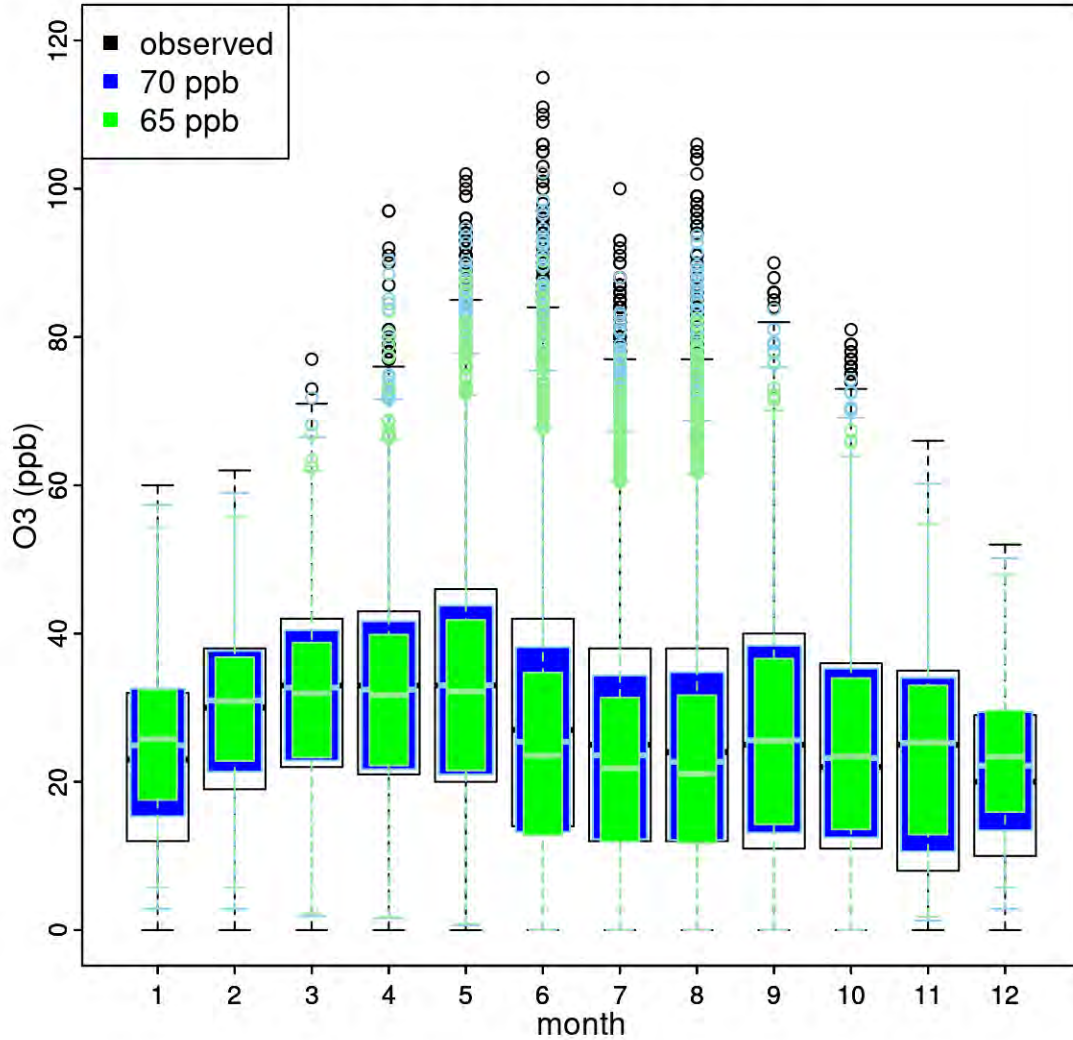


1
 2 **Figure 3C-73. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
 3 **Sacramento study area.**



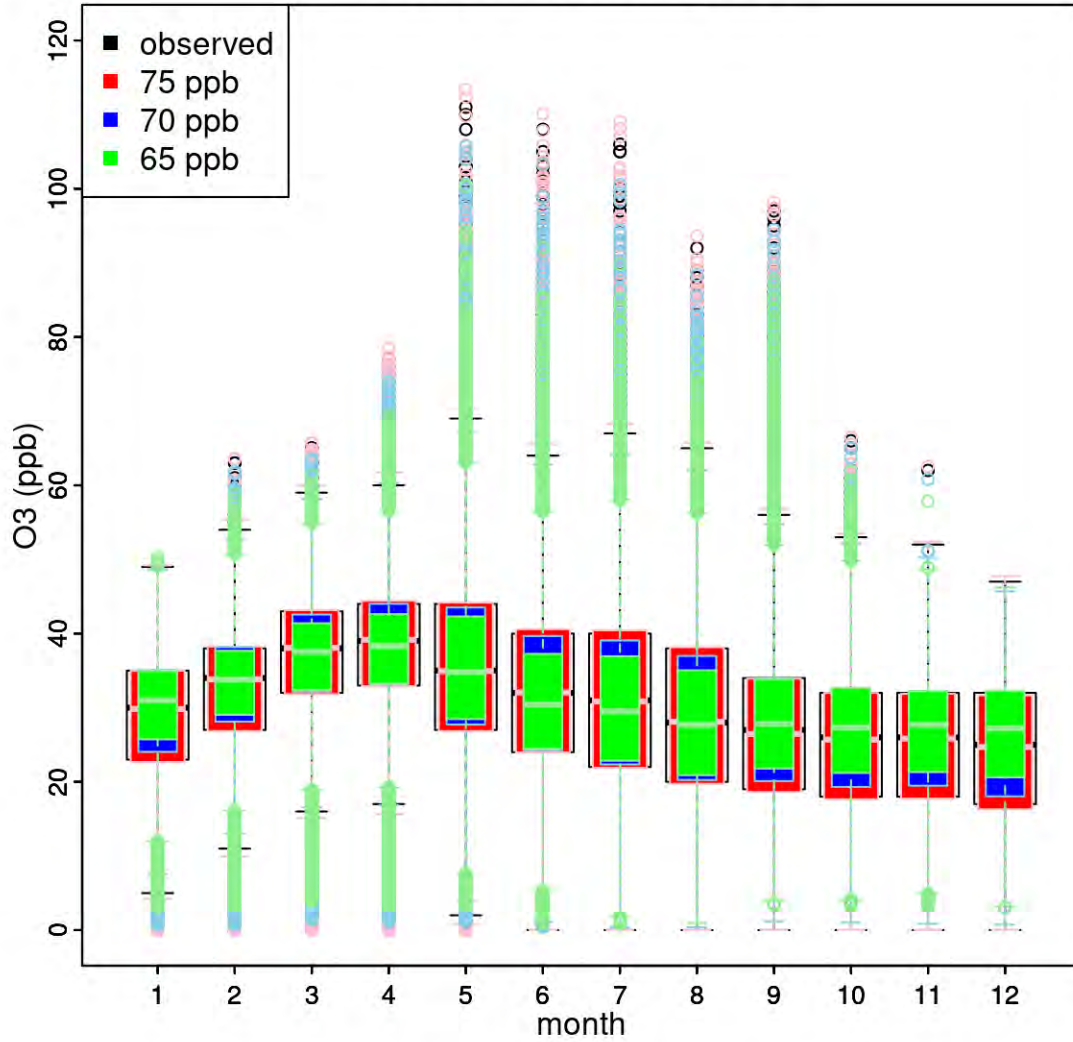
1
 2 **Figure 3C-74. Diurnal distribution of hourly O₃ concentrations at monitoring sites in the**
 3 **St. Louis study area.**

Atlanta sites: 2015-2017



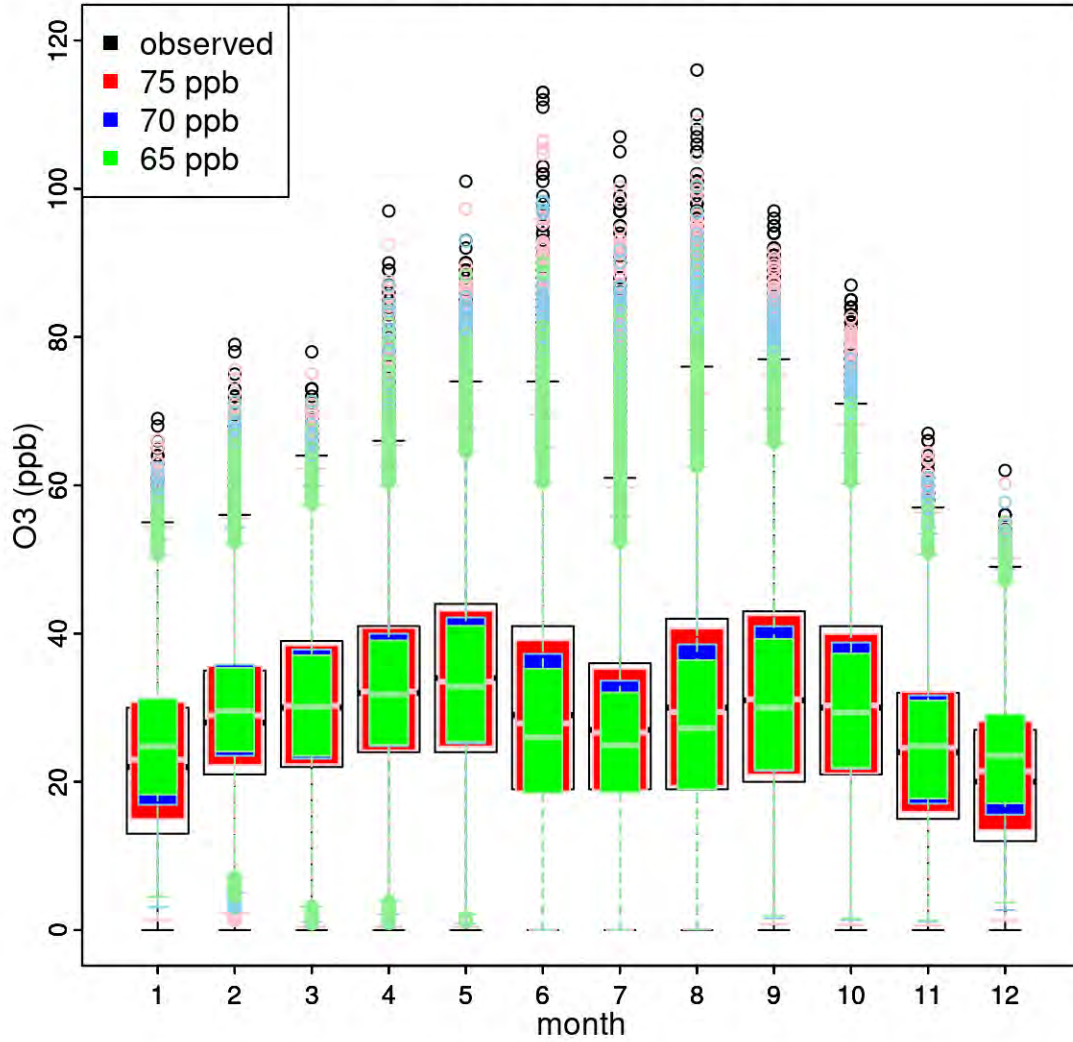
1
2 **Figure 3C-75. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Atlanta study area. Note: Observed concentrations in this area have a design**
4 **value of 75 ppb.**

Boston sites: 2015-2017



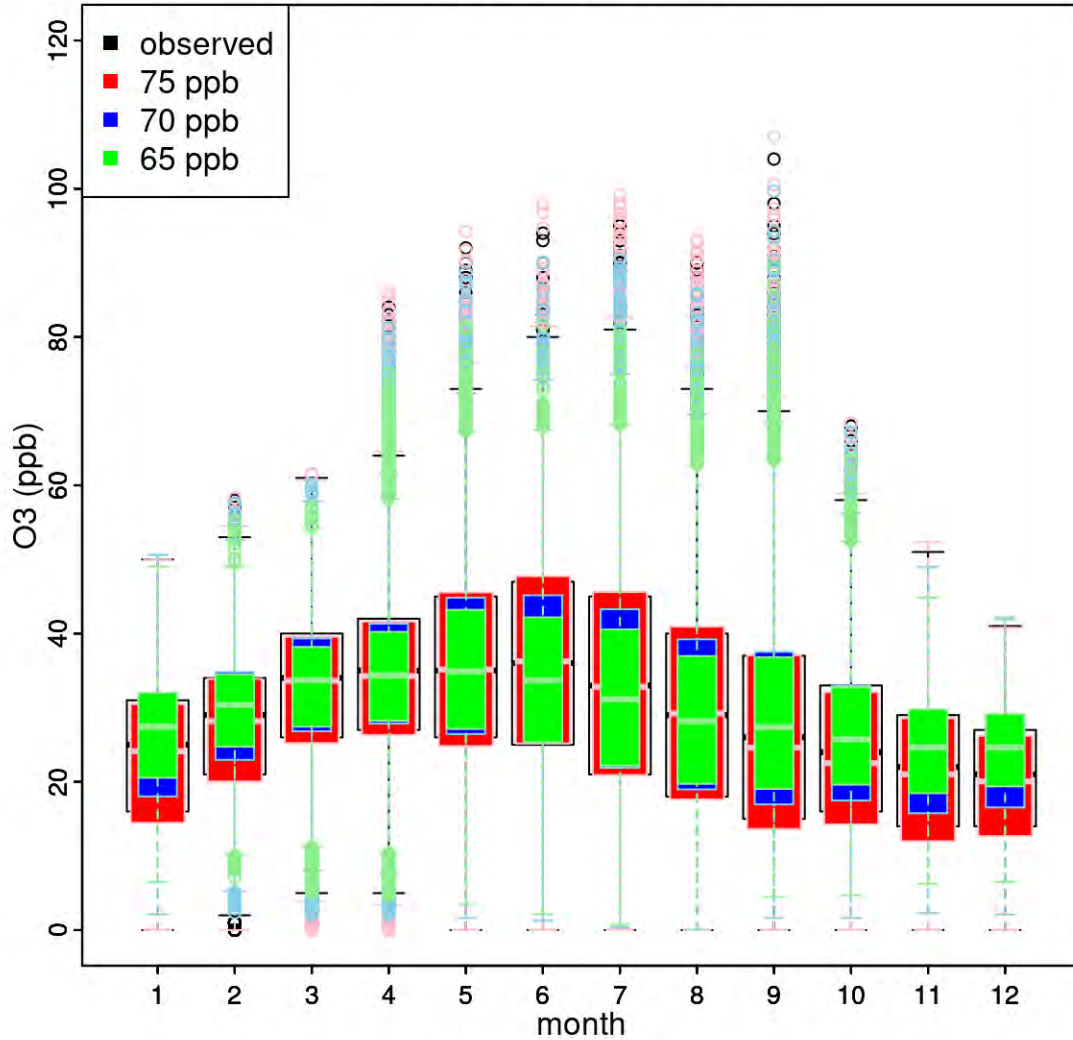
1
2 **Figure 3C-76. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Boston study area.**

Dallas sites: 2015-2017



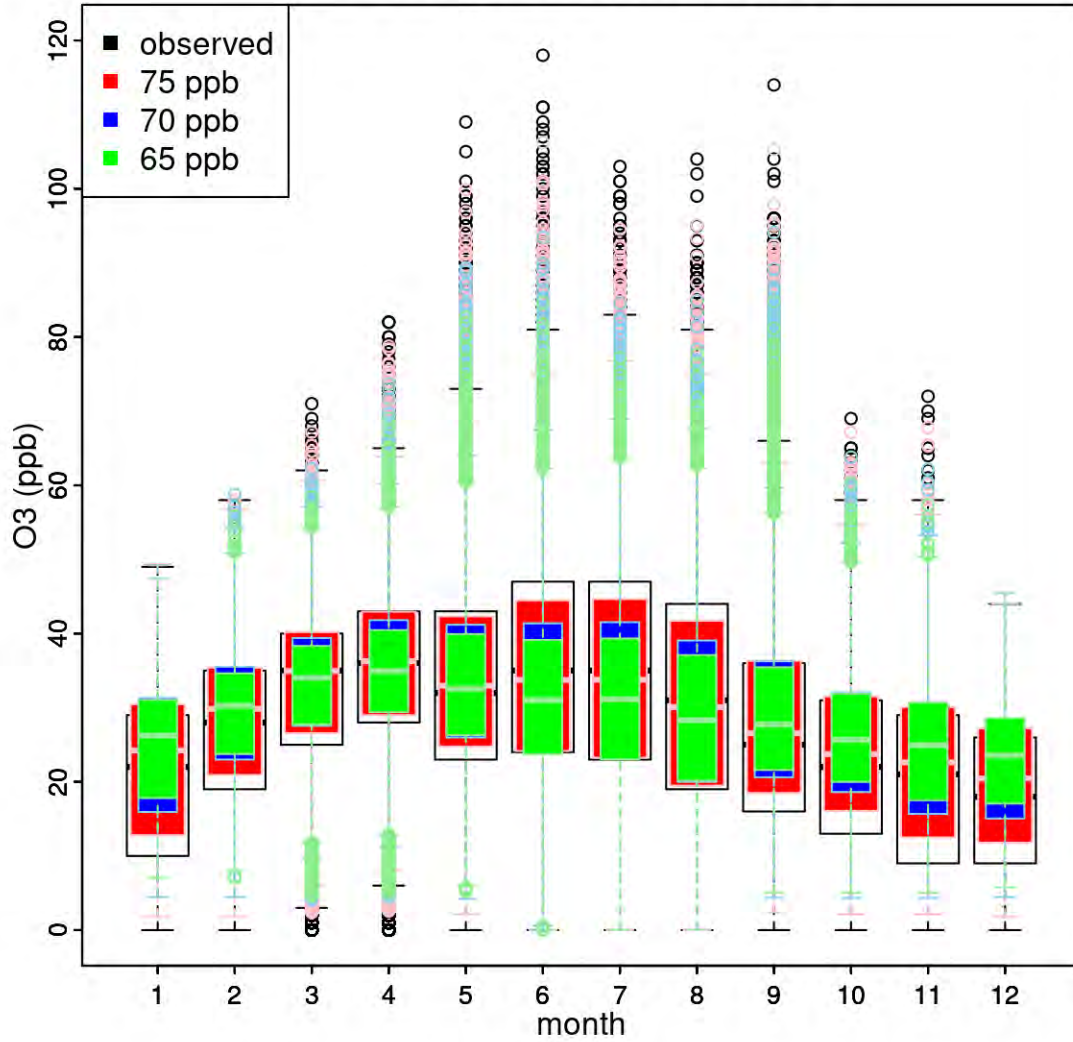
1
2 **Figure 3C-77. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Dallas study area.**

Detroit sites: 2015-2017



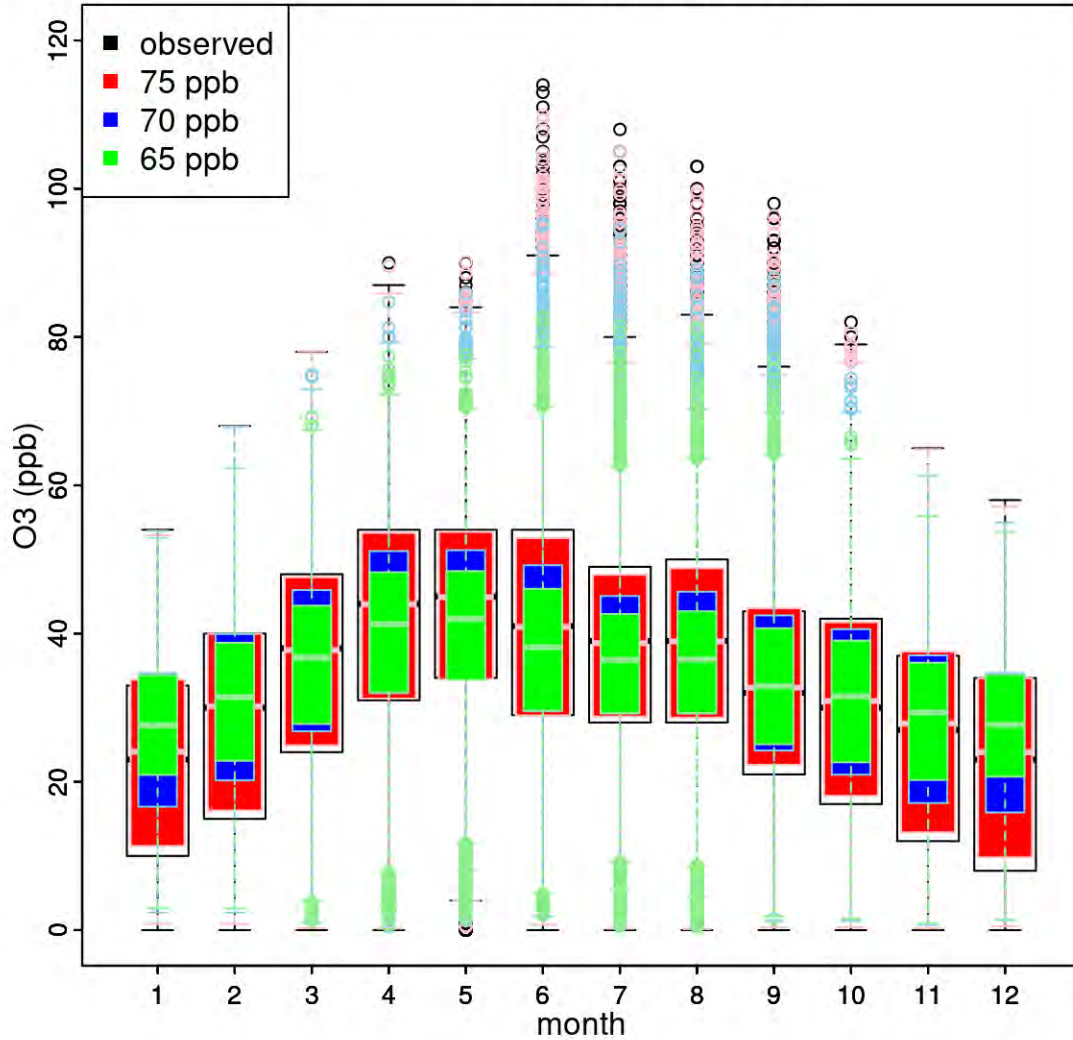
1
2 **Figure 3C-78. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Detroit study area.**

Philadelphia sites: 2015-2017



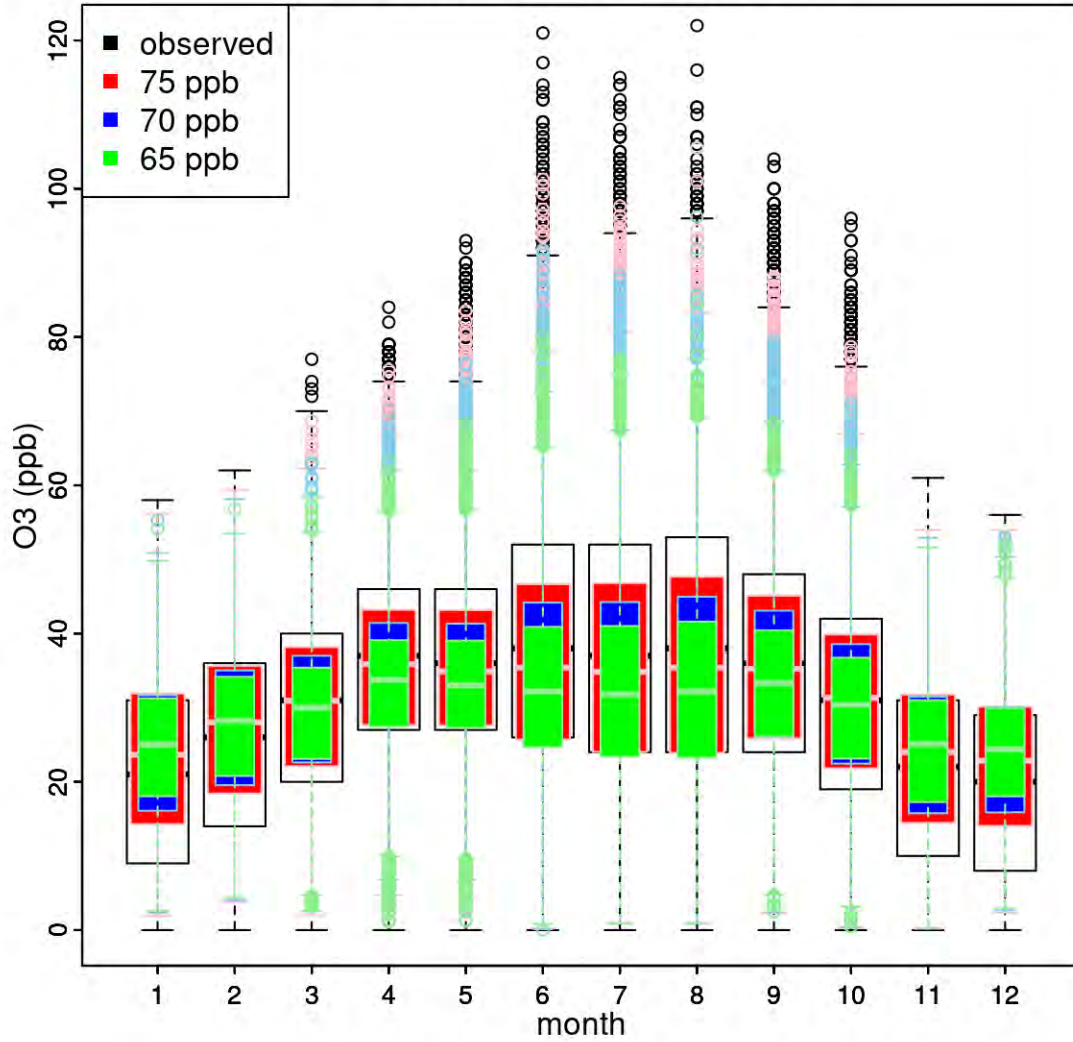
1
2 **Figure 3C-79. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Philadelphia study area.**

Phoenix sites: 2015-2017



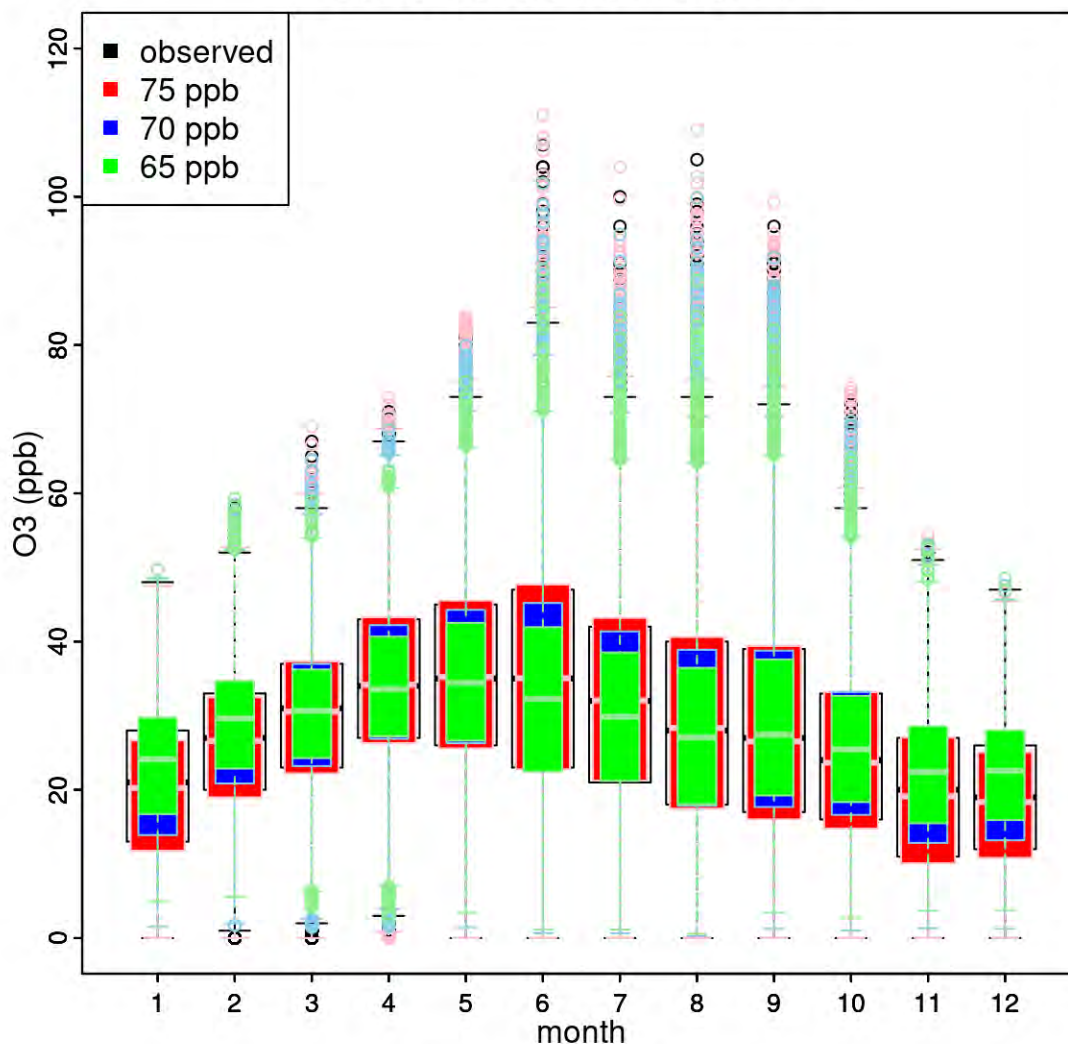
1
2 **Figure 3C-80. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Phoenix study area.**

Sacramento sites: 2015-2017



1
2 **Figure 3C-81. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **Sacramento study area.**

SaintLouis sites: 2015-2017



1
2 **Figure 3C-82. Monthly distribution of hourly O₃ concentrations at monitoring sites in the**
3 **St. Louis study area.**
4

5 **3C.7.3 Air Quality Inputs for the Exposure and Risk Analyses**

6 The air quality inputs for the exposure and risk analyses discussed in chapter 3 include
7 spatial surfaces of hourly O₃ concentrations estimated for each census tract in the eight urban
8 study areas using the VNA technique described in section 3C.6. In this section, we present three
9 types of figures which summarize the data from the hourly VNA surfaces for observed air

1 quality, and air quality adjusted to meet the current standard of 70 ppb, and air quality adjusted
2 to meet alternative scenarios of 75 ppb¹⁸ and 65 ppb.

3 The first set of figures (Figure 3C-83 through Figure 3C-90) shows density scatter plots
4 of the change in MDA8 O₃ concentrations versus the observed concentrations based on the
5 hourly VNA estimates in each study area. In each of these figures, the left-hand panel shows the
6 observed MDA8 values (x-axis) versus the change in those values that occur when air quality is
7 adjusted for the 75 ppb scenario (y-axis). The middle panel shows the MDA8 values for air
8 quality adjusted to meet the 75 ppb scenario (x-axis) versus the additional change in those values
9 that occur when air quality is adjusted to meet the current standard of 70 ppb (y-axis). Finally,
10 the right-hand panels show the corresponding changes from the current standard to the 65 ppb
11 scenario. Within each panel, the x and y values are rounded to the nearest integer and colored to
12 show the relative frequency of each 1 x 1 ppb square within the plot region. Values falling
13 outside of the plot region were set to the nearest value within the plot region, and frequencies
14 above the range in the color bar were set to the highest value within the color bar.

15 The second set of figures (Figure 3C-91 through Figure 3C-106) provides maps of the
16 adjusted design values (3-year average of the annual 4th highest MDA8 values) and May-
17 September average MDA8 values based on the ambient air data and the hourly VNA surfaces, as
18 well as difference maps showing the changes between these surfaces. For the difference maps,
19 the panels on the left show the changes in these values that occur when air quality is adjusted for
20 the 75 ppb scenario, the panels in the middle show the additional changes in these values that
21 occur when air quality is further adjusted to meet the current standard of 70 ppb, and the right-
22 hand panels show the additional changes that occur then air quality is further adjusted for the 65
23 ppb scenario. Within each panel, squares show values based on observed data at ambient air
24 monitoring sites while circles show values based on VNA estimates at census tract centroids.
25 While each panel shows both monitors in the study area for each selected urban study area as
26 well as some additional monitors located outside of the study area, only the monitors located
27 within the study area were used when determining the emissions reductions necessary to meet
28 the various standards.

29 The third set of figures (Figure 3C-107 through Figure 3C-114) shows changes in design
30 values (3-year average of the annual 4th highest MDA8 values) and May-September average
31 MDA8 values in the eight urban case study areas versus population and population density. The
32 total population and population density information for each census tract were obtained from the
33 U.S. Census Bureau based on the 2010 U.S. Census. Each panel shows a histogram of the total

¹⁸ Atlanta was already just meeting the 75 ppb scenario for the 2015-2017 period. Boston, Detroit, and St. Louis were below 75 ppb for 2015-2017; design values for these urban study areas were adjusted upward to just meet 75 ppb.

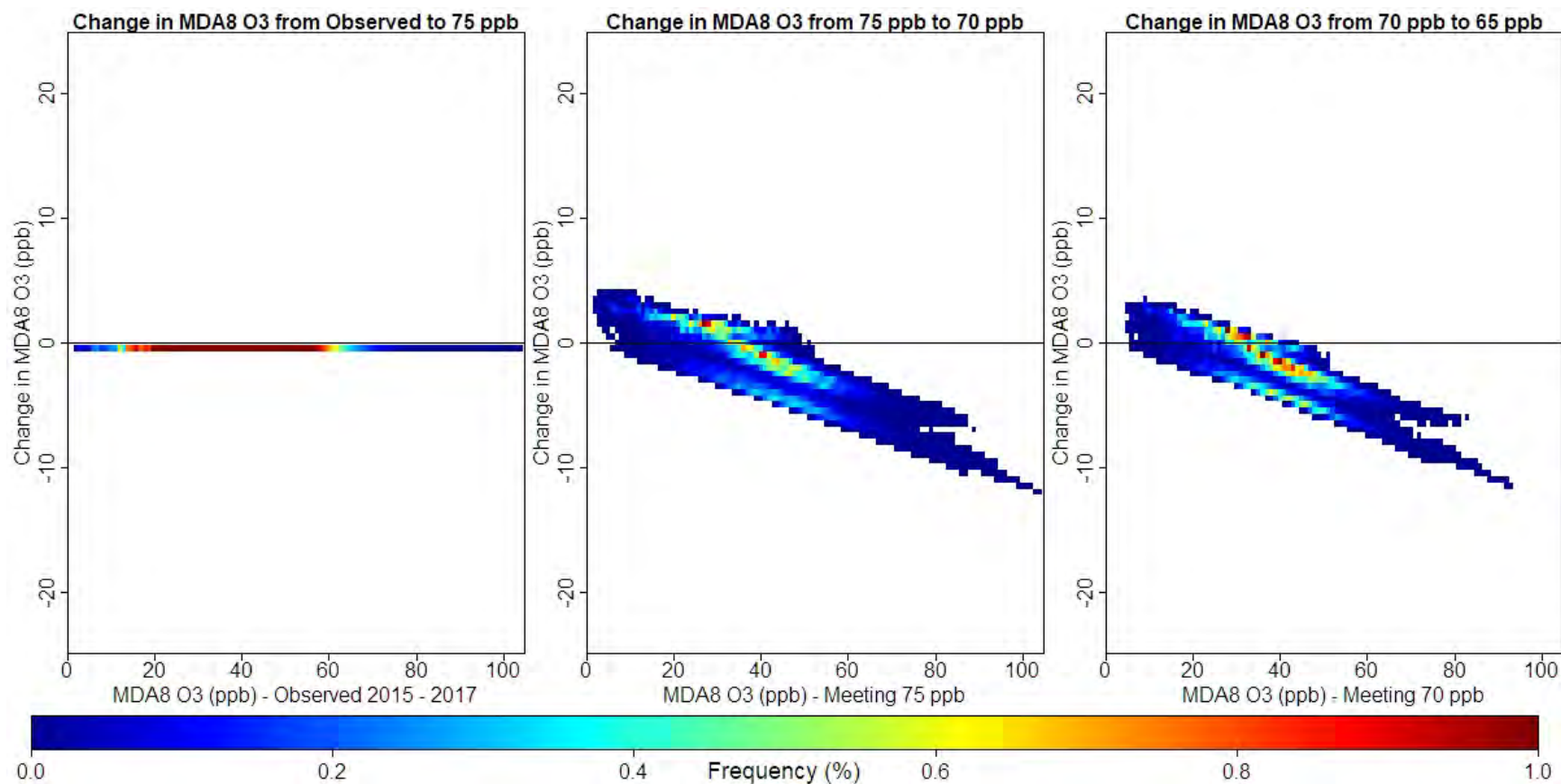
1 population stratified by the change in design value or seasonal average. The bars are also color-
2 coded by population density bin. Values falling outside of the plot region set to the nearest
3 values within the plot region.

4 In general, the density scatter plots show that the HDDM adjustment procedure predicts
5 increases in MDA8 O₃ at low ambient air concentrations and decreases in MDA8 O₃ at high
6 concentrations (Figure 3C-83 through Figure 3C-90). The vast majority of the increases in
7 MDA8 O₃ occur at ambient air concentrations below 50 ppb. The relationship between the
8 starting concentrations and the changes in these values based on the HDDM adjustments is fairly
9 linear with strong negative correlation in all eight urban study areas.¹⁹ In some study areas, such
10 as Philadelphia and Detroit, there is a bimodal pattern near the center of the distribution, which
11 may be indicative of differing behavior near the urban population center versus the surrounding
12 suburban areas.

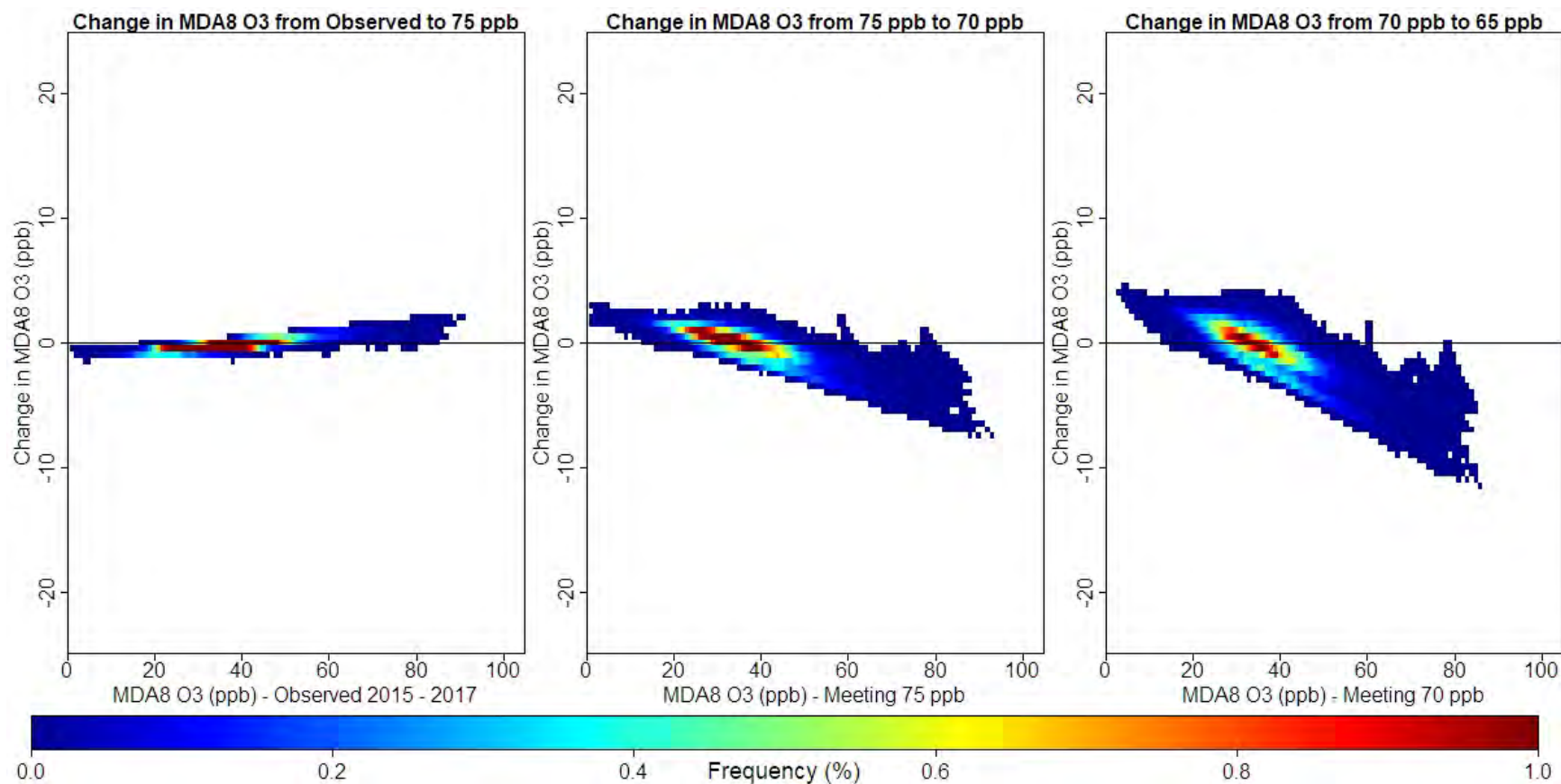
13 The maps reveal consistent spatial patterns of O₃ changes across the urban study areas.
14 The design values generally decreased when air quality was adjusted to meet the current standard
15 of 70 ppb²⁰ and continued to decrease when air quality was further adjusted for the 65 ppb
16 scenario (Figure 3C-91 through Figure 3C-106). The design values tend to decrease more
17 quickly in suburban and rural areas than in the urban population centers. The May-September
18 “seasonal” average MDA8 values also followed this trend to some extent, although the behavior
19 in the urban population centers varied slightly amongst the urban study areas (Figure 3C-107
20 through Figure 3C-114). In summary, these figures show that using the CAMx/HDDM
21 adjustment methodology, peak O₃ concentrations are reduced in urban study areas with large
22 domain-wide reductions in U.S. anthropogenic NO_x emissions.

¹⁹ Except for the “Observed - 75 ppb” changes for the three urban study areas where the design values were adjusted upwards: Boston, Detroit, and St. Louis.

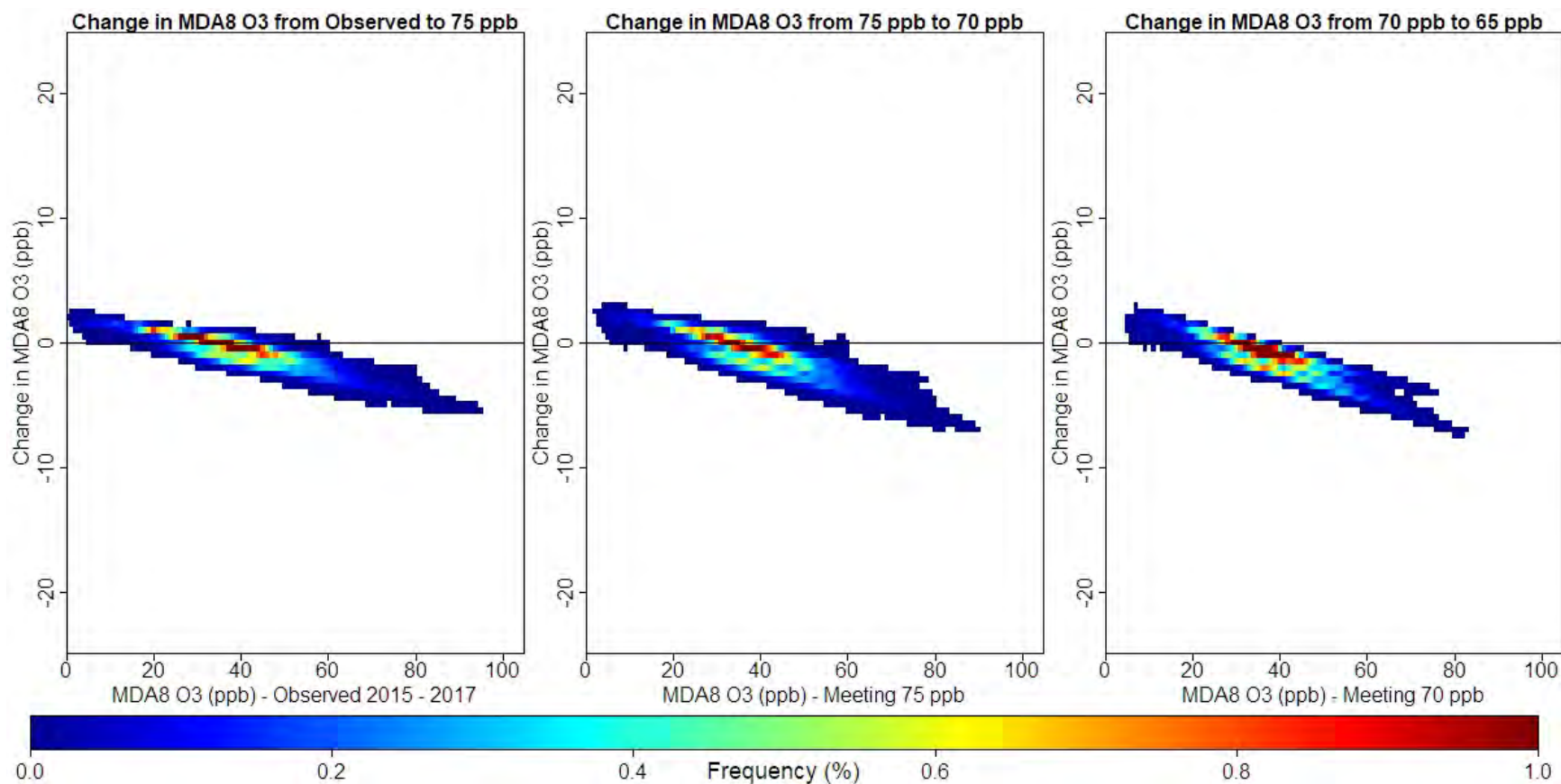
²⁰ All design values from the VNA surfaces decreased when going from recent conditions to the 75 ppb adjustment scenario, with the exceptions of study areas that required upward adjustments for the 75 ppb scenario: Boston, Detroit, and St. Louis.



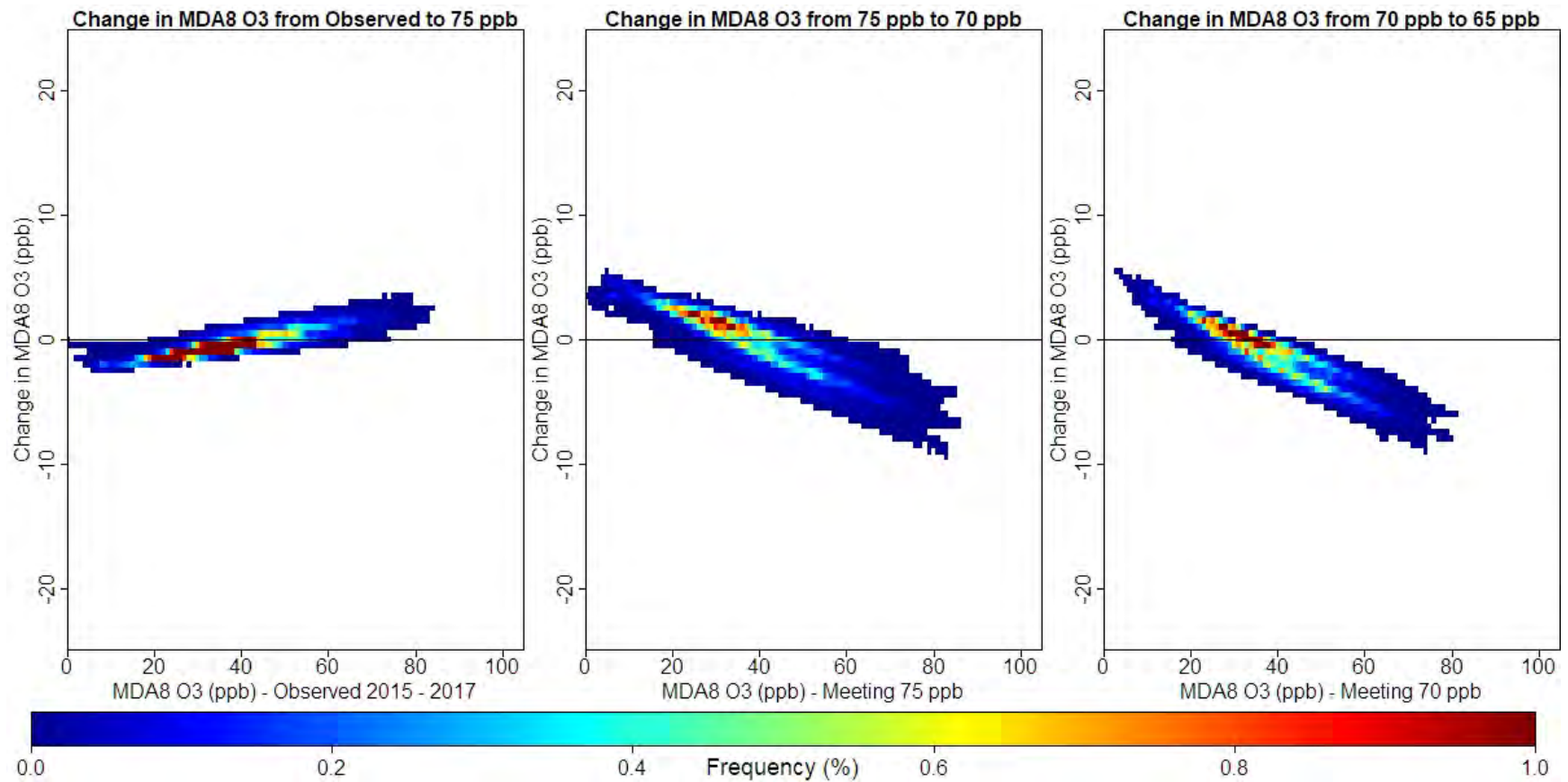
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2 **Figure 3C-83. Changes in MDA8 O₃ based on HDDM adjustments in the Atlanta study area.**



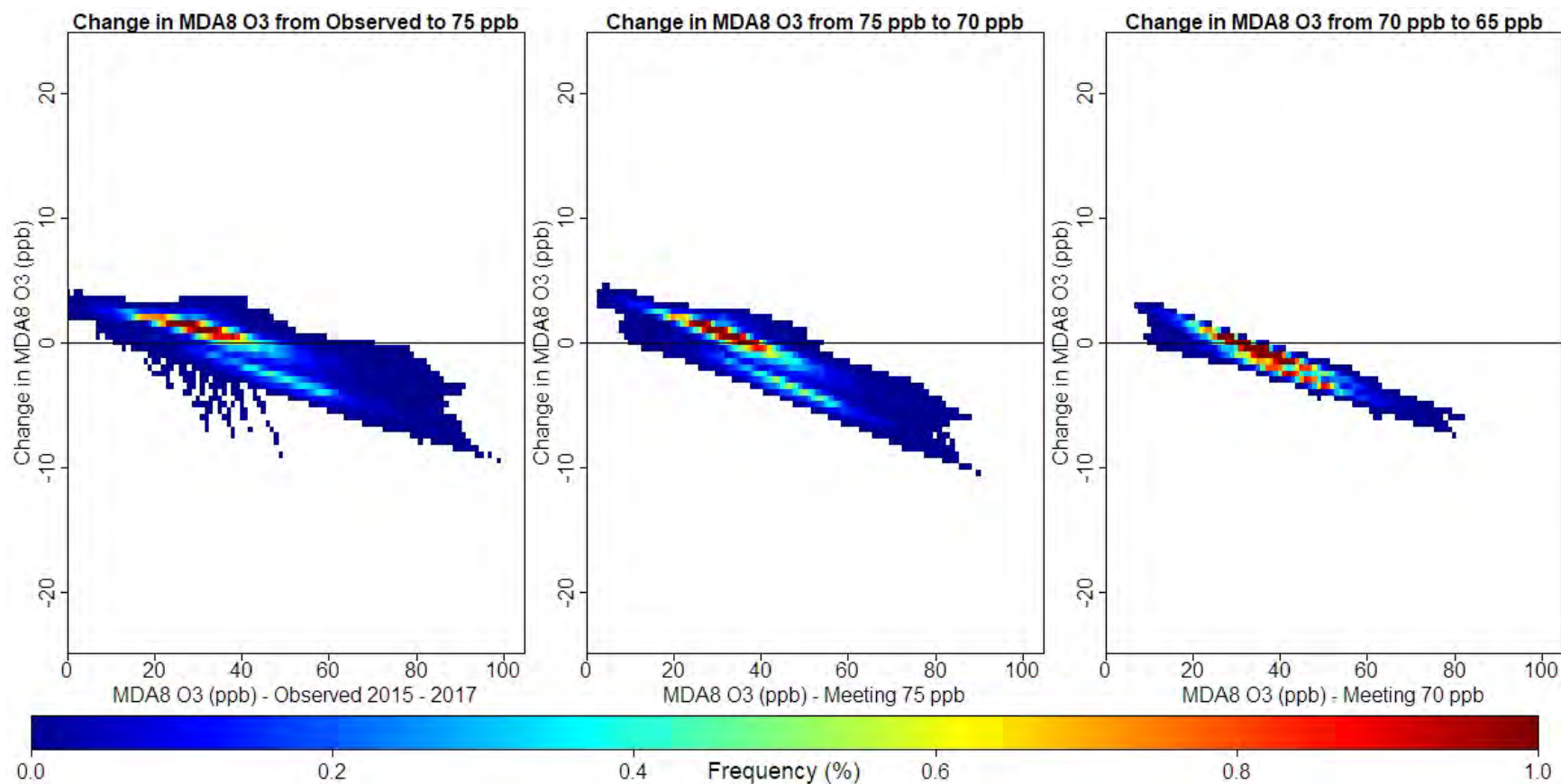
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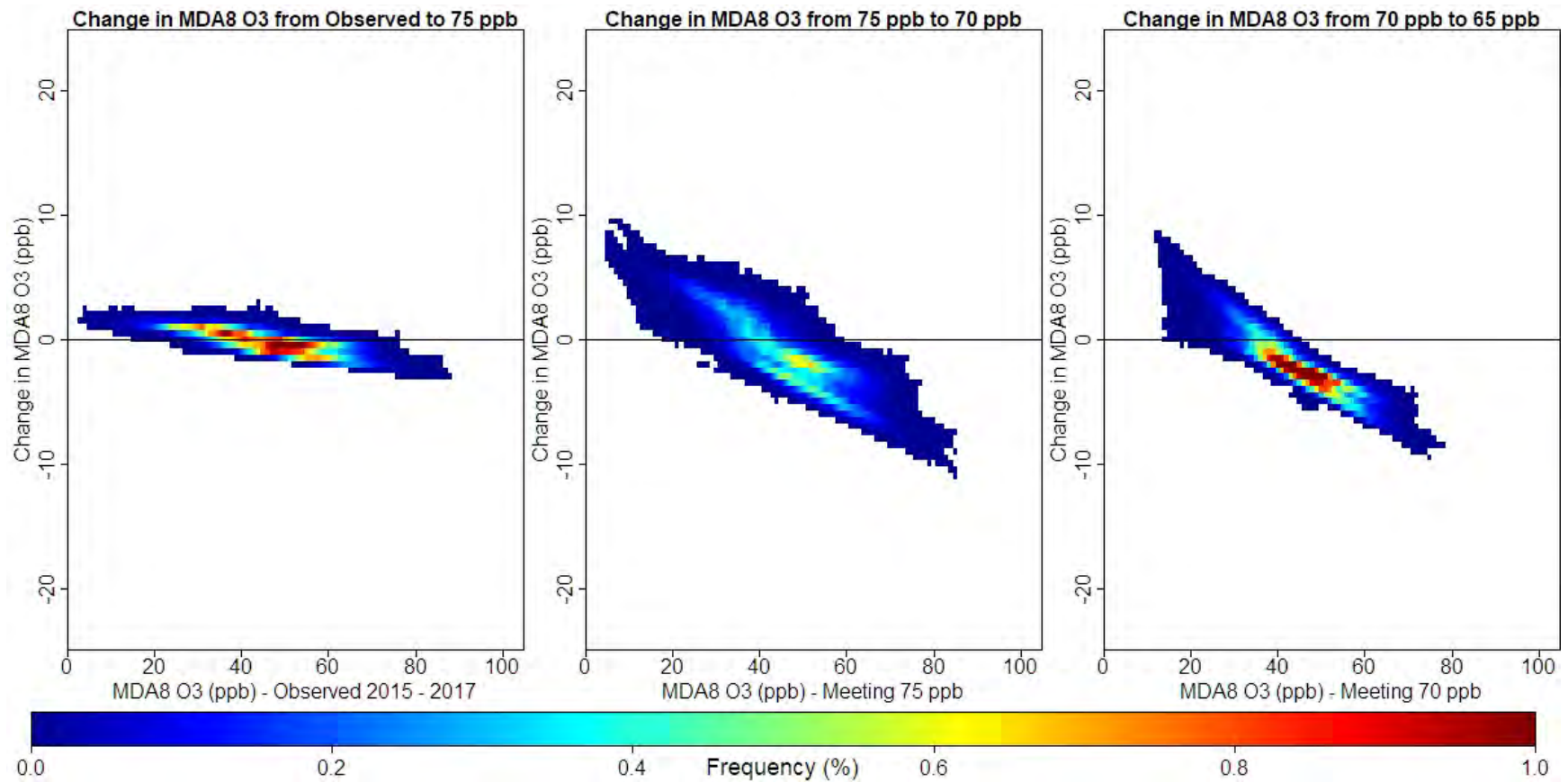
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2 **Figure 3C-85. Changes in MDA8 O₃ based on HDDM adjustments in the Dallas study area.**



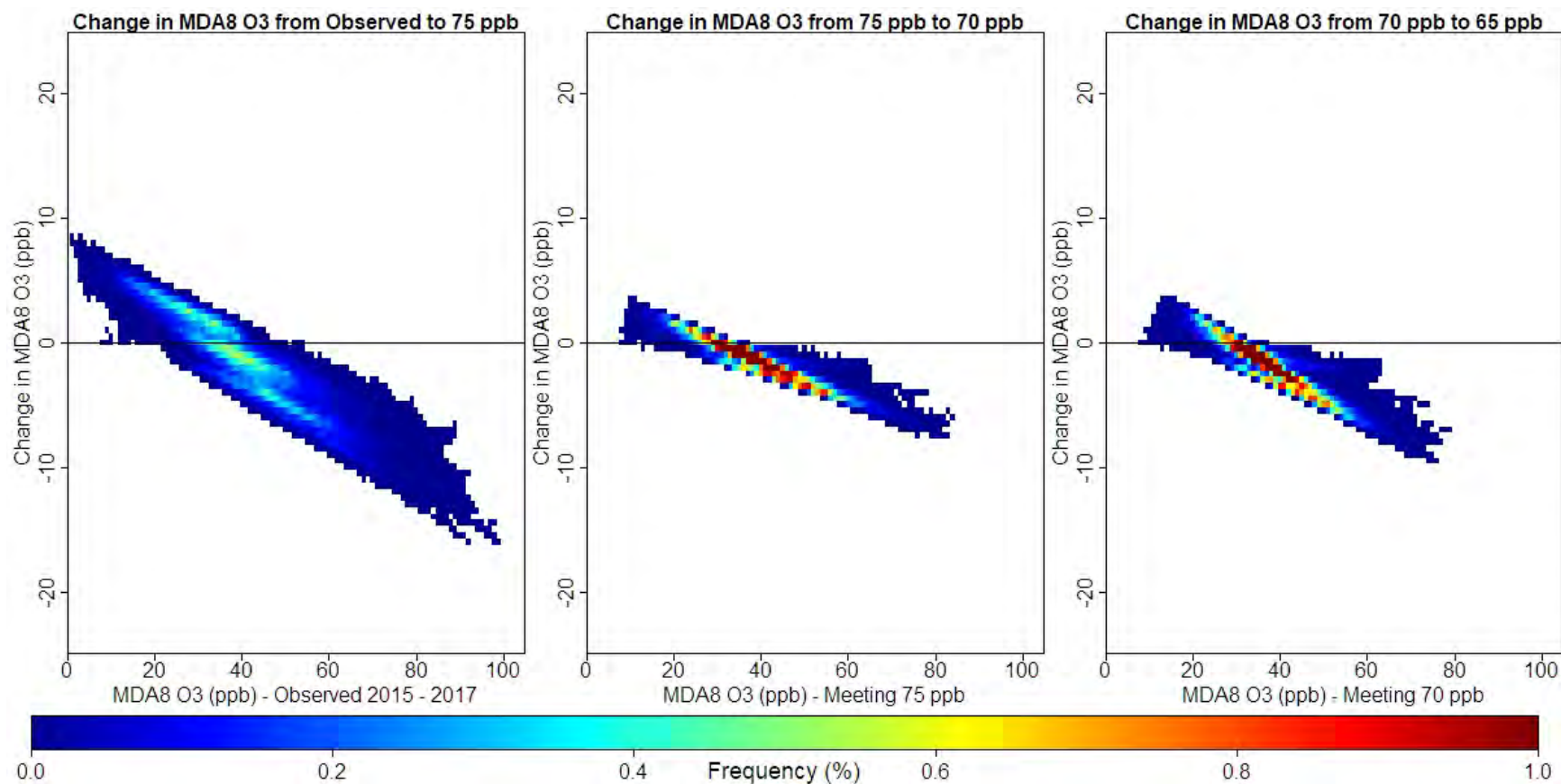
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2 **Figure 3C-86. Changes in MDA8 O₃ based on HDDM adjustments in the Detroit study area.**



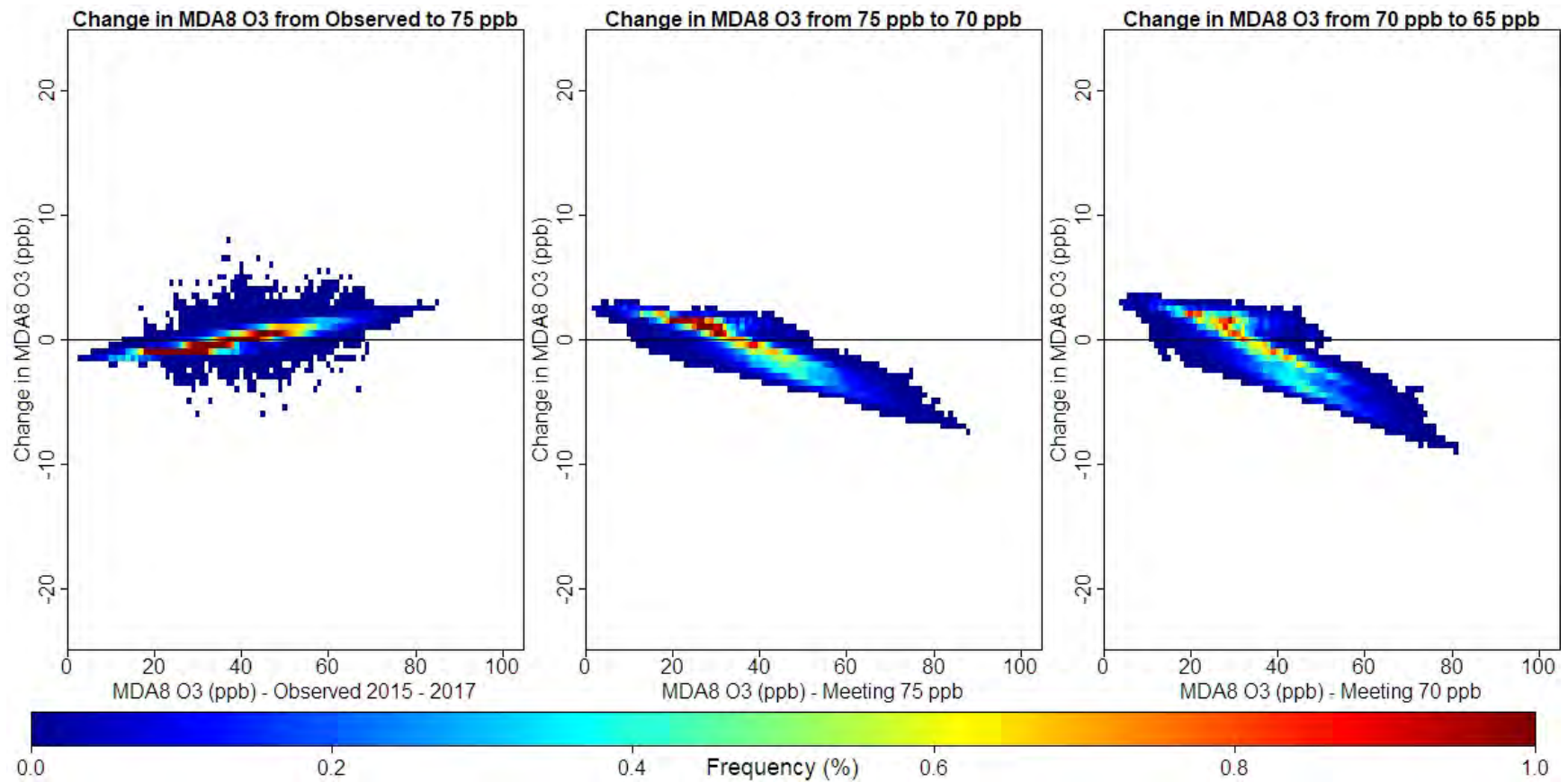
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2 **Figure 3C-87. Changes in MDA8 O₃ based on HDDM adjustments in the Philadelphia study area.**



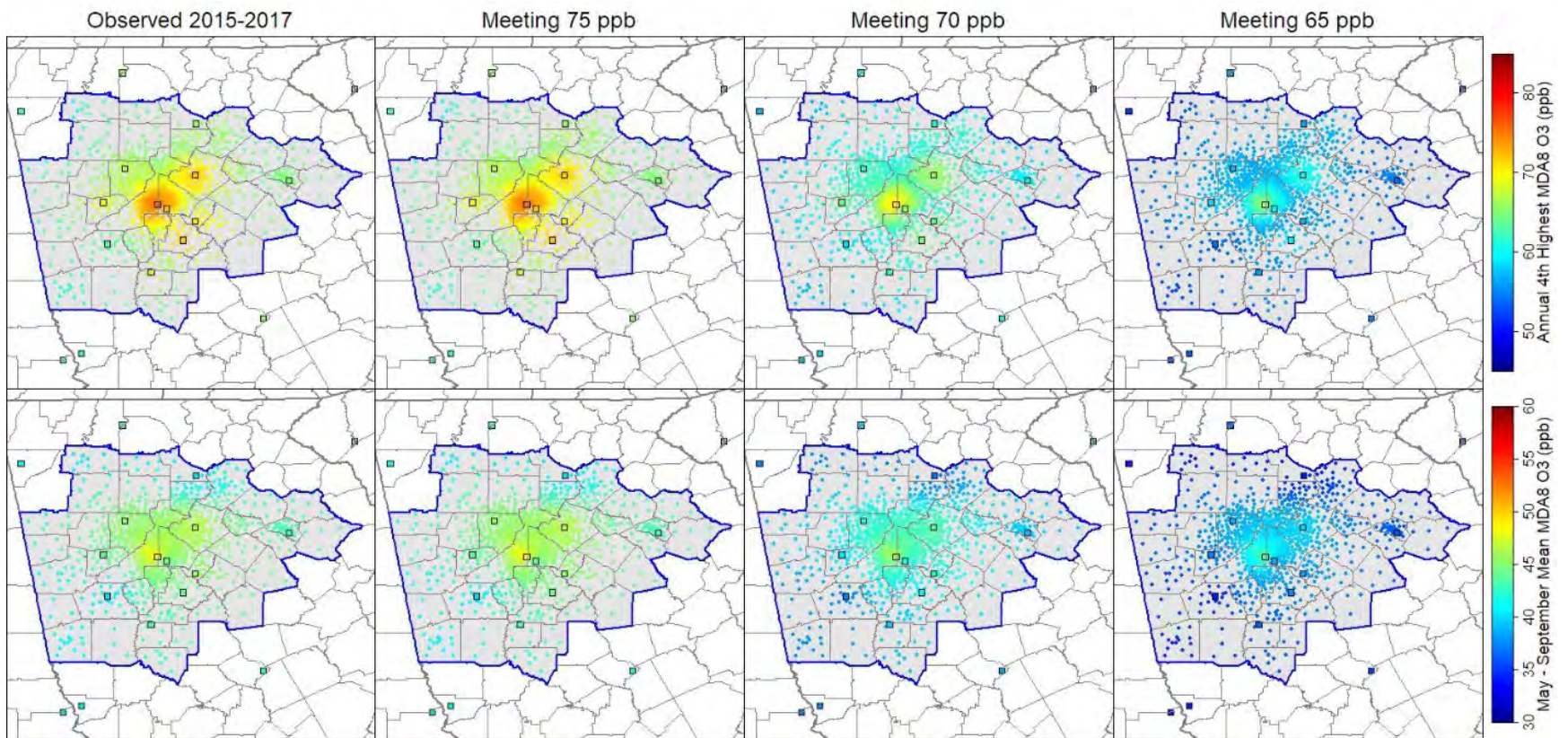
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2 **Figure 3C-88. Changes in MDA8 O₃ based on HDDM adjustments in the Phoenix study area.**



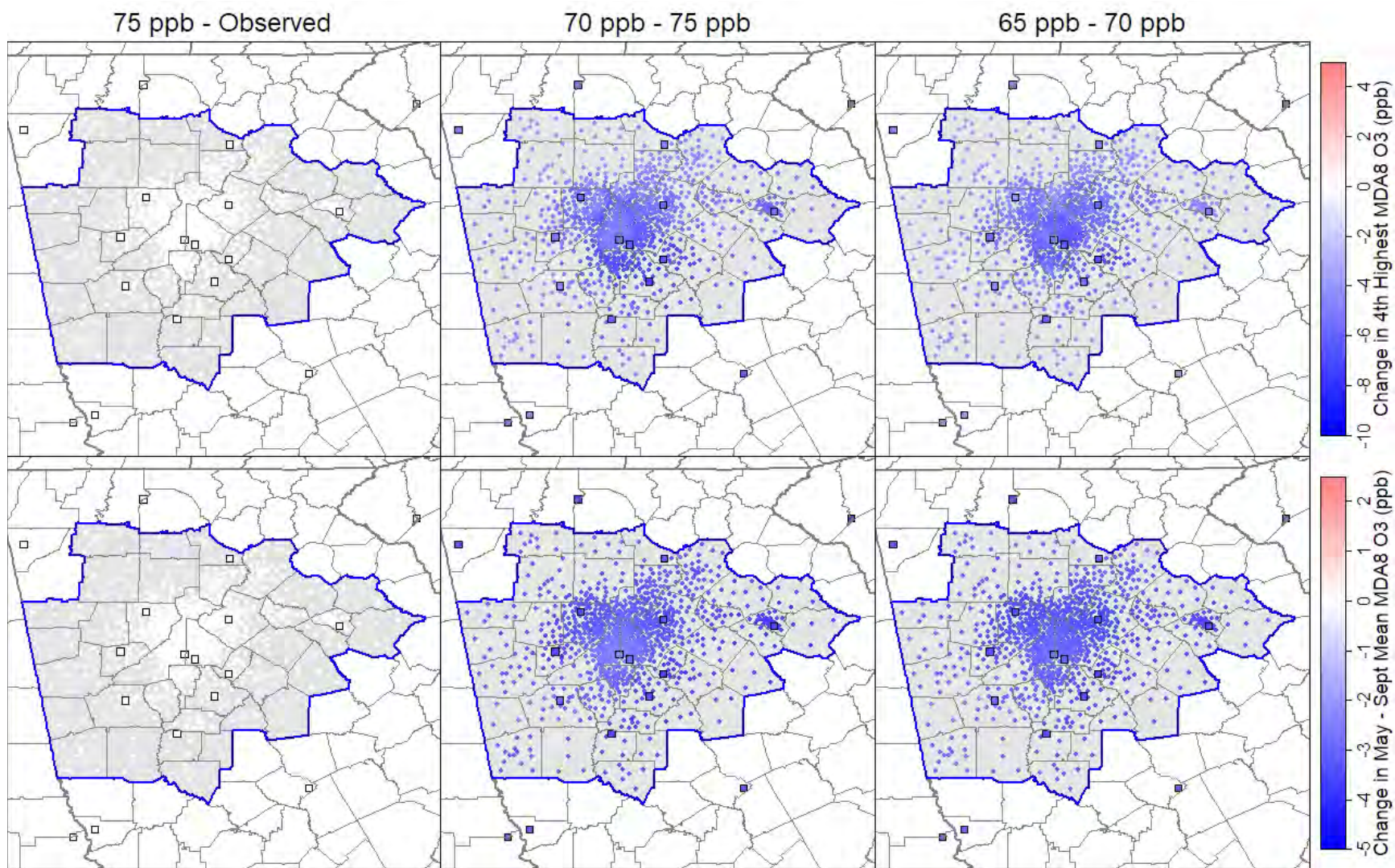
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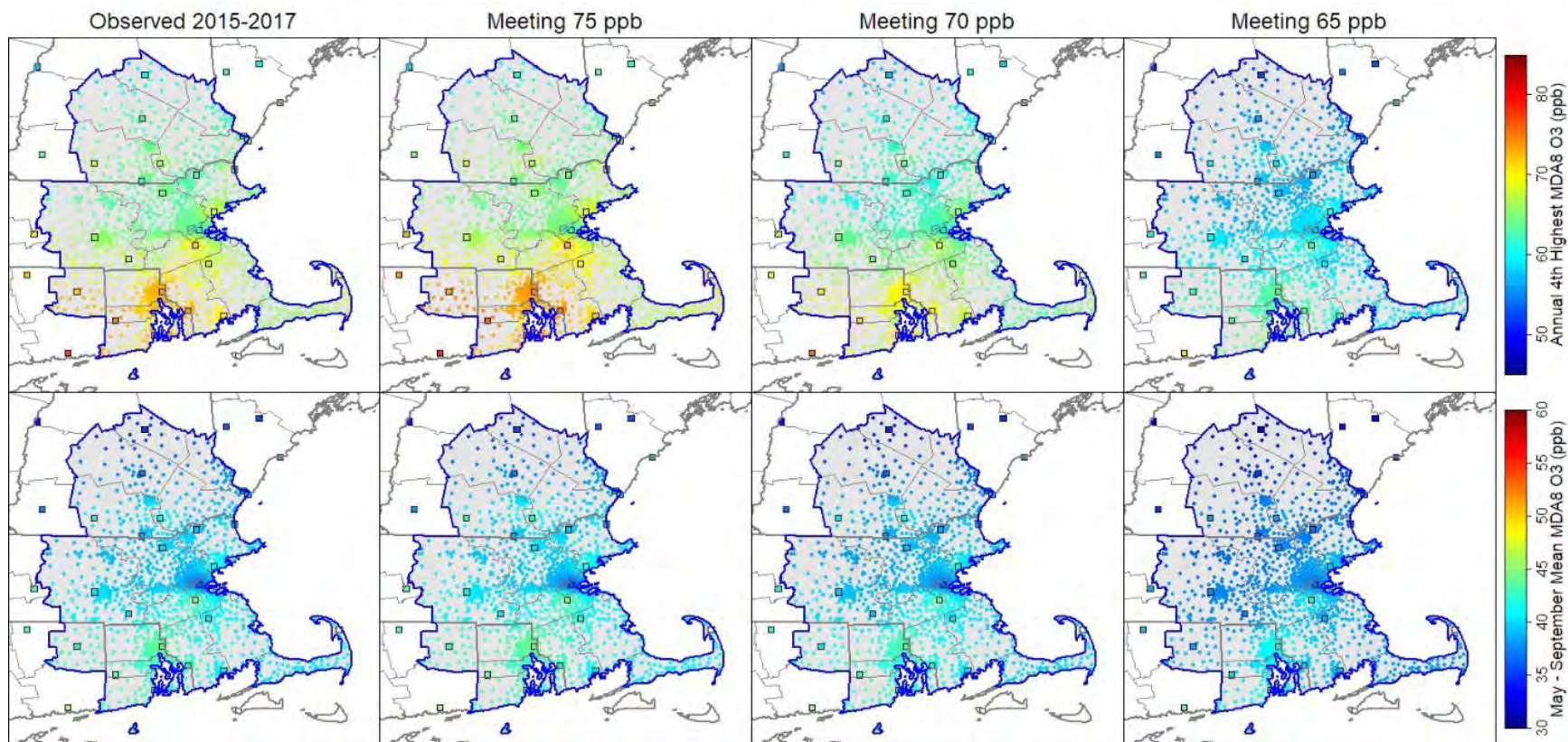
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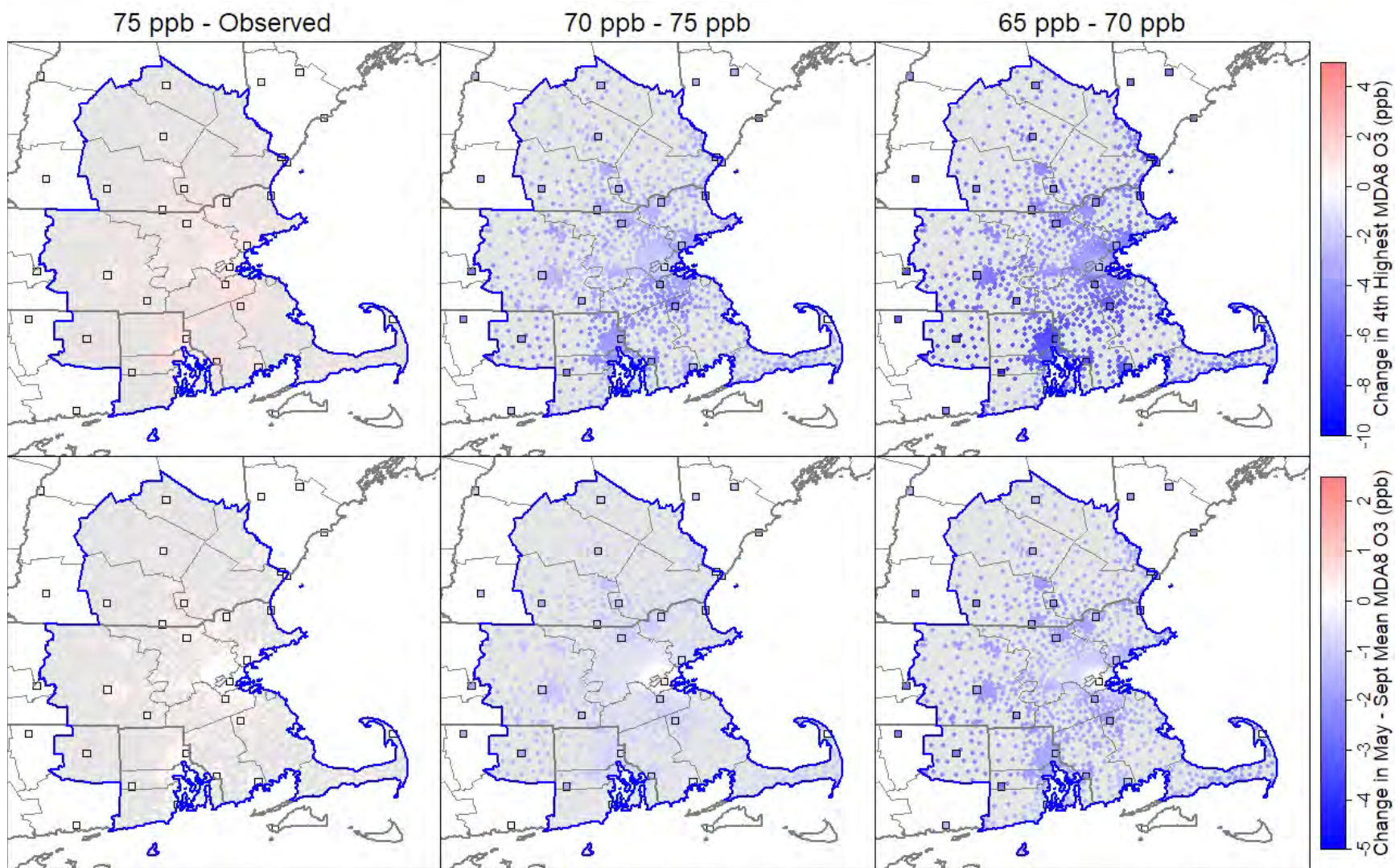
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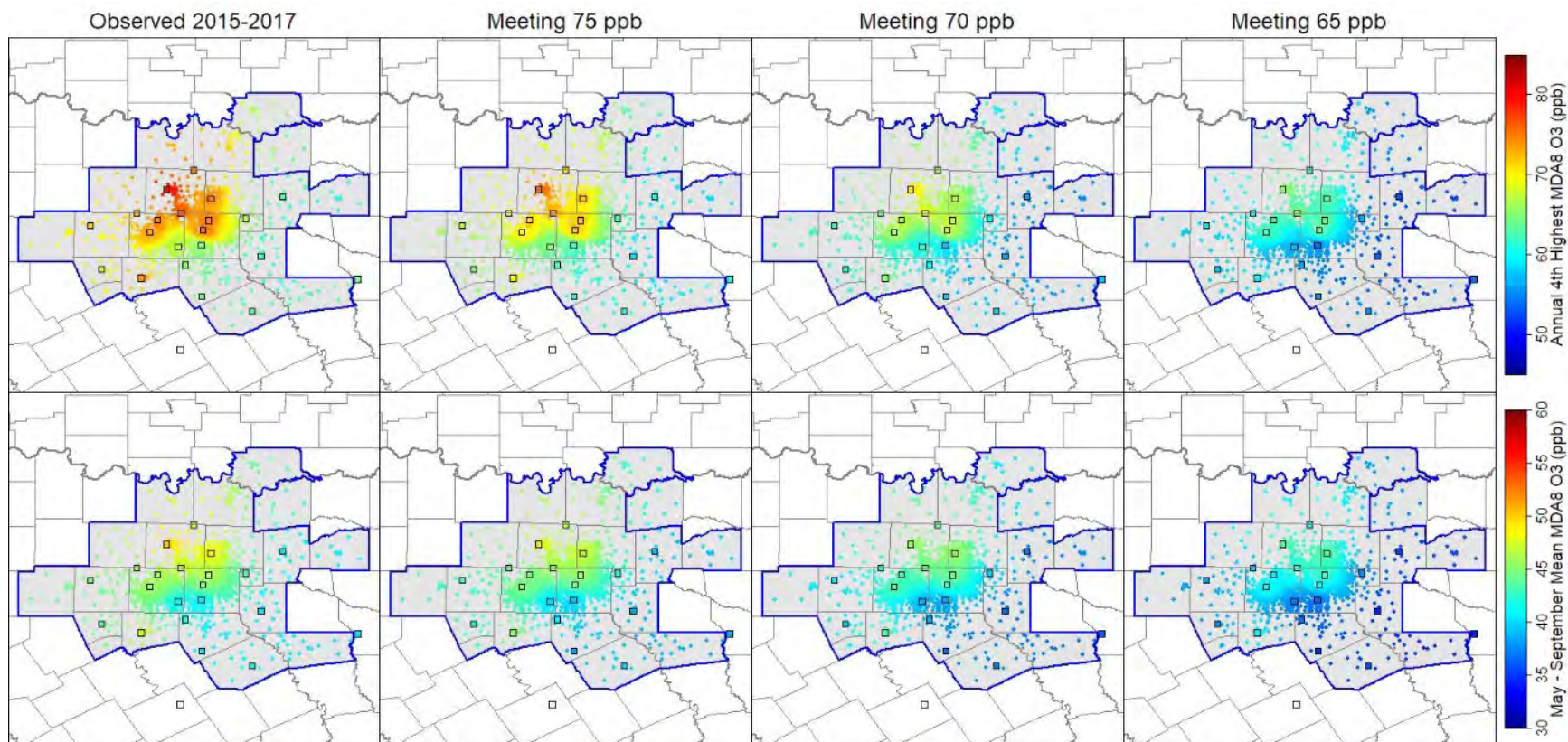
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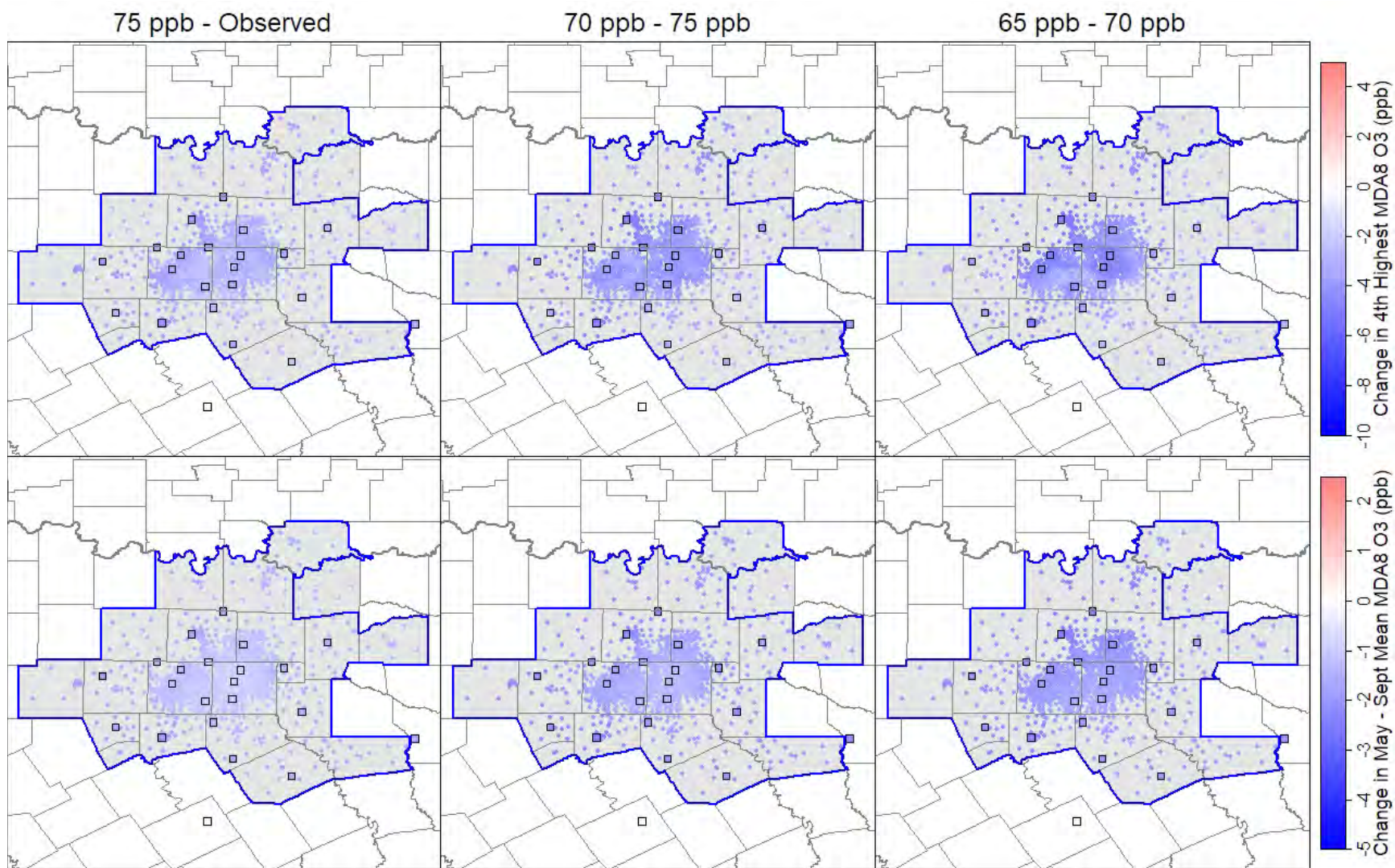
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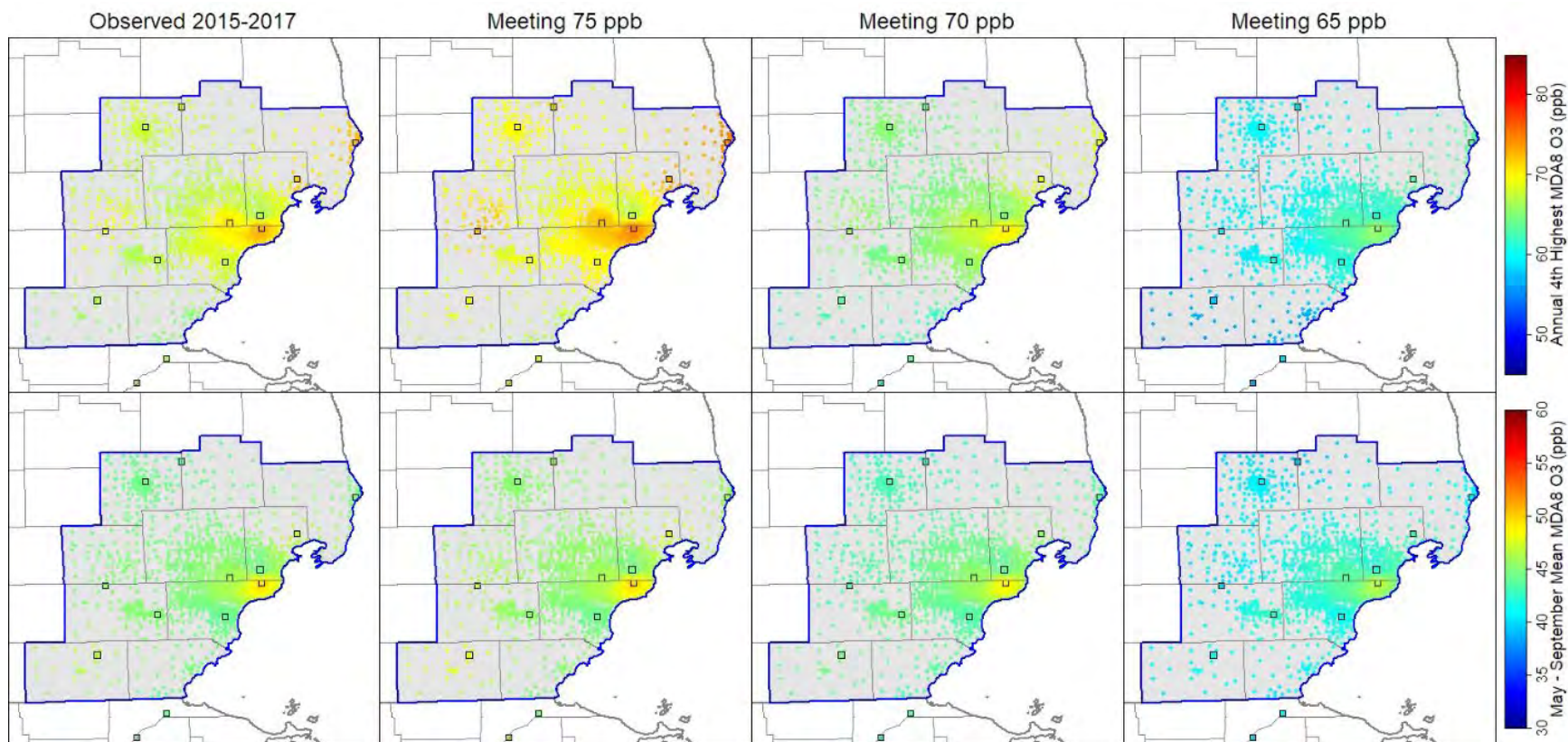
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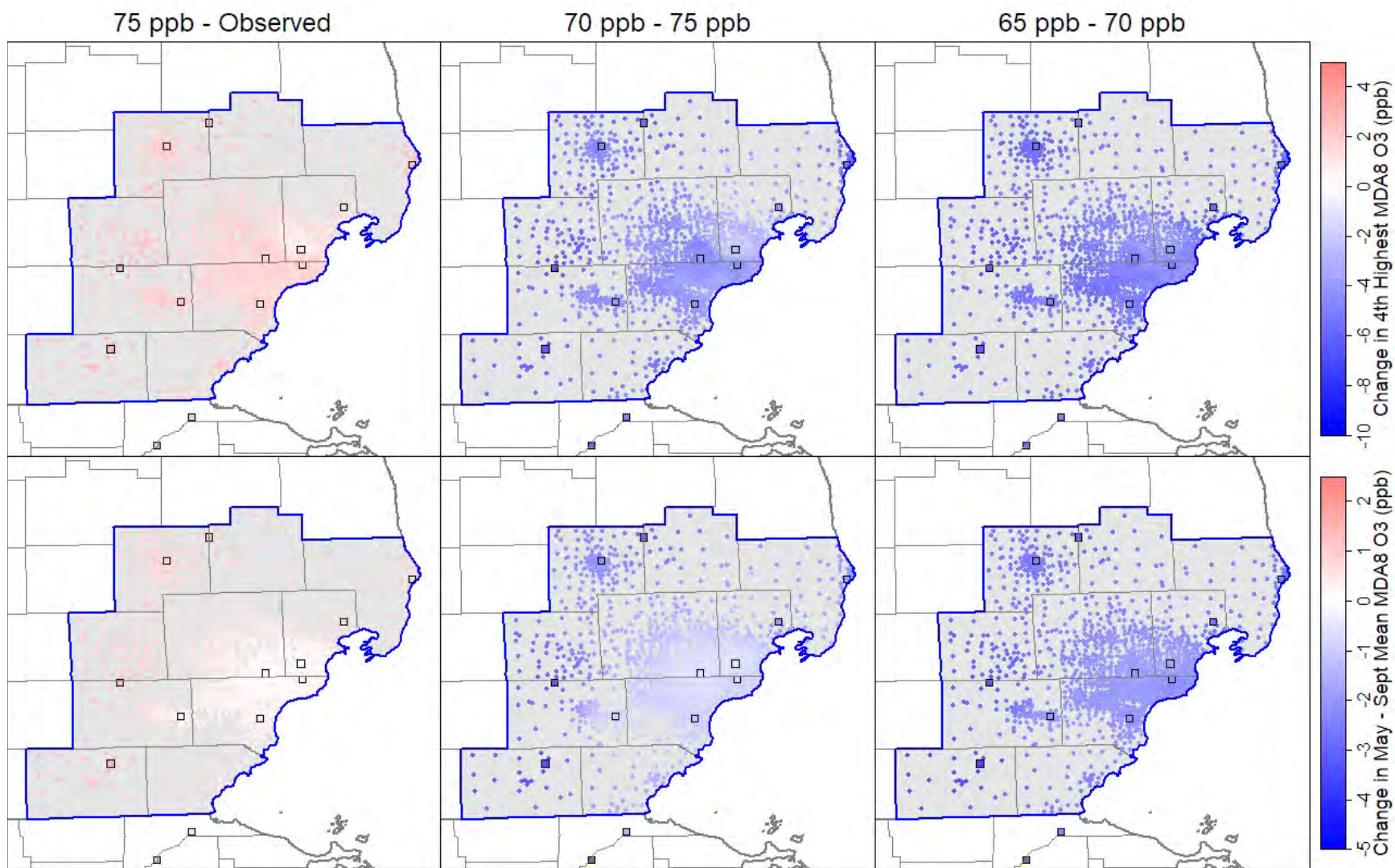
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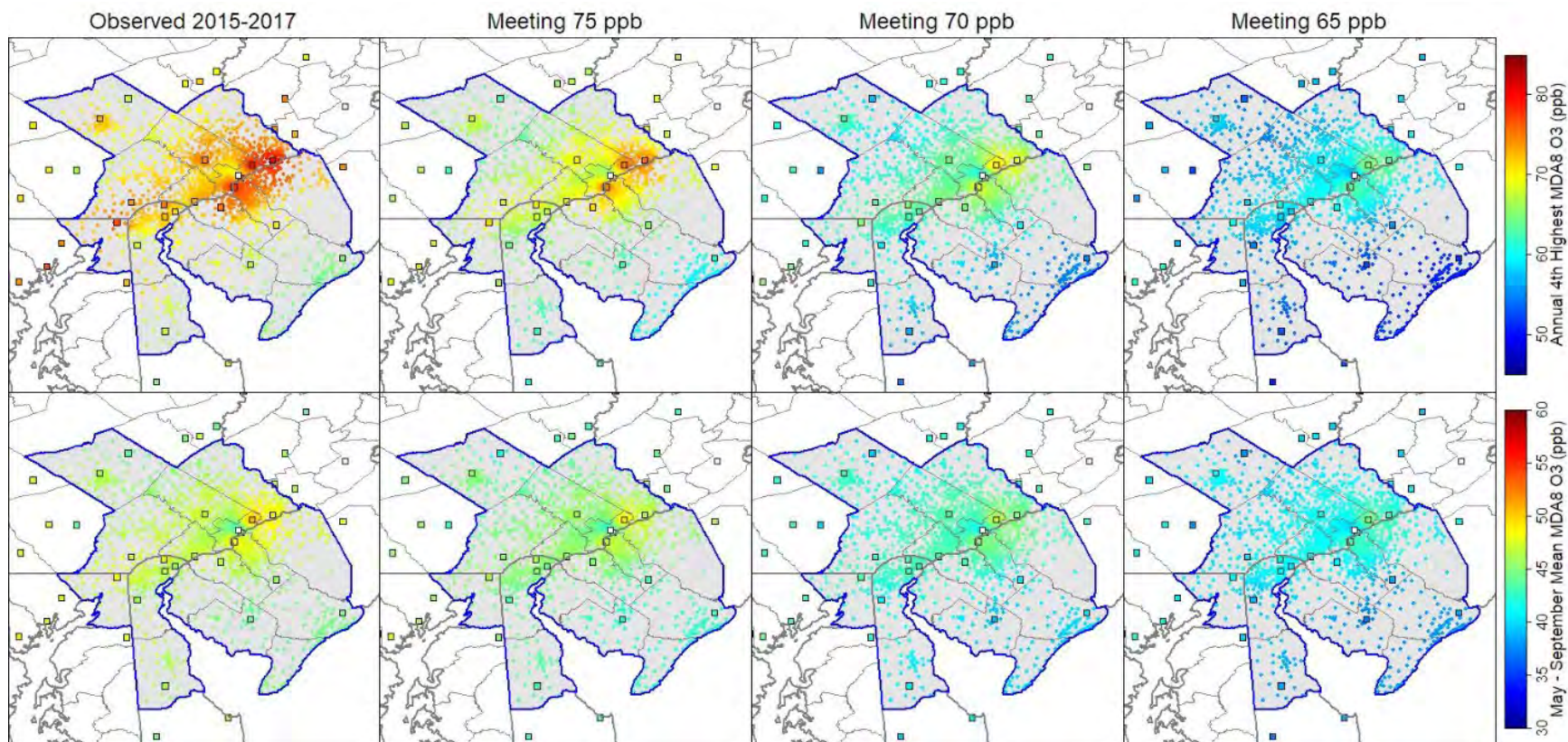
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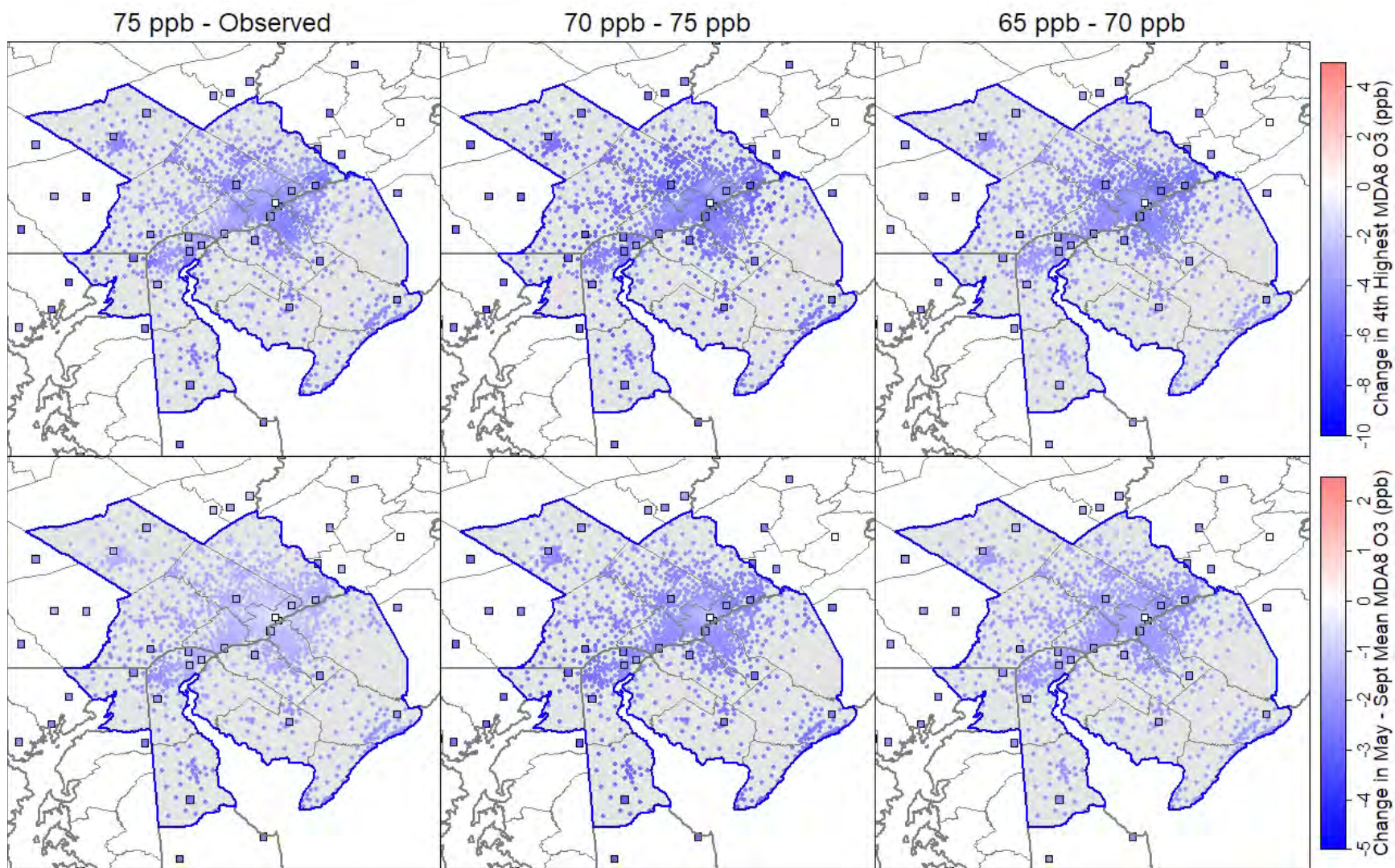
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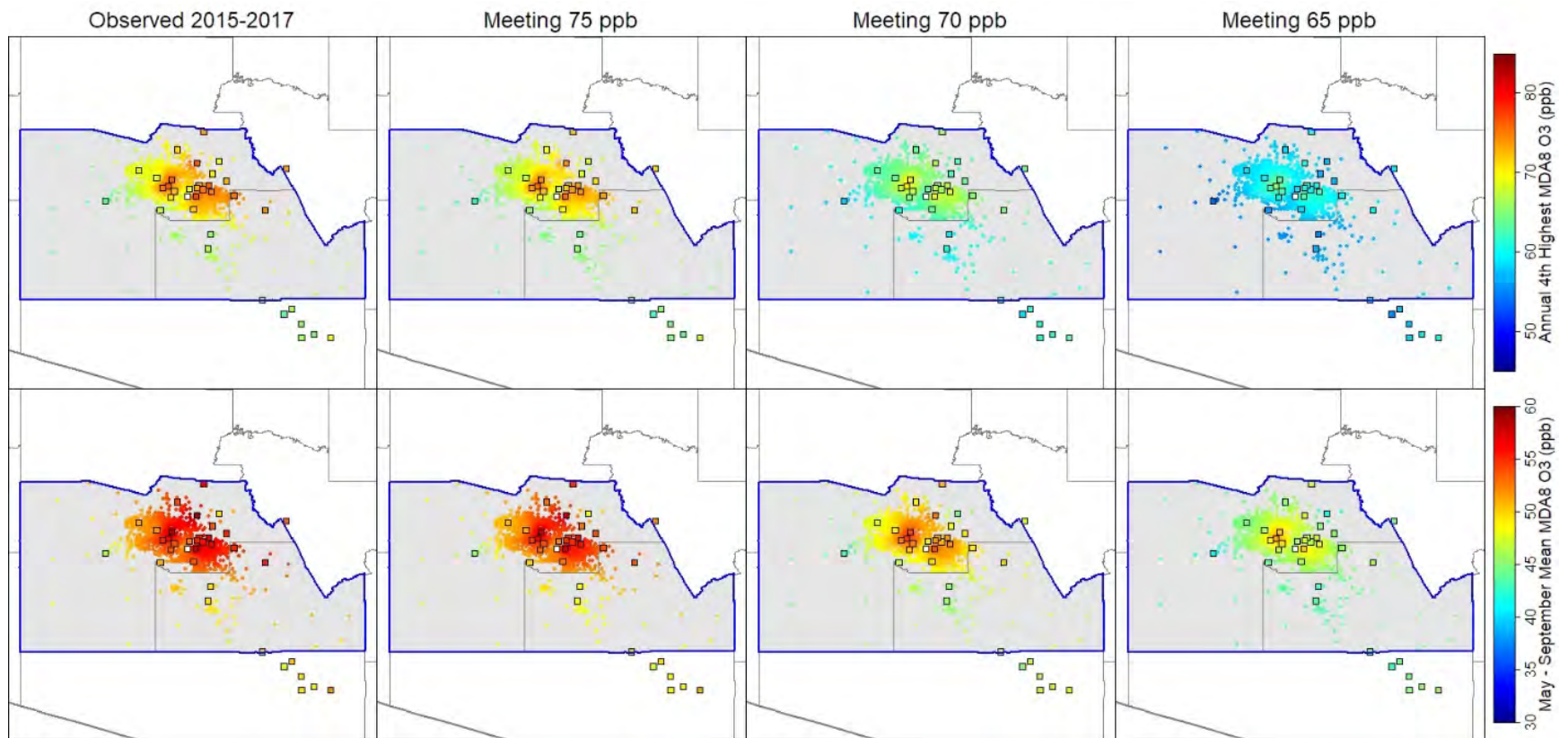
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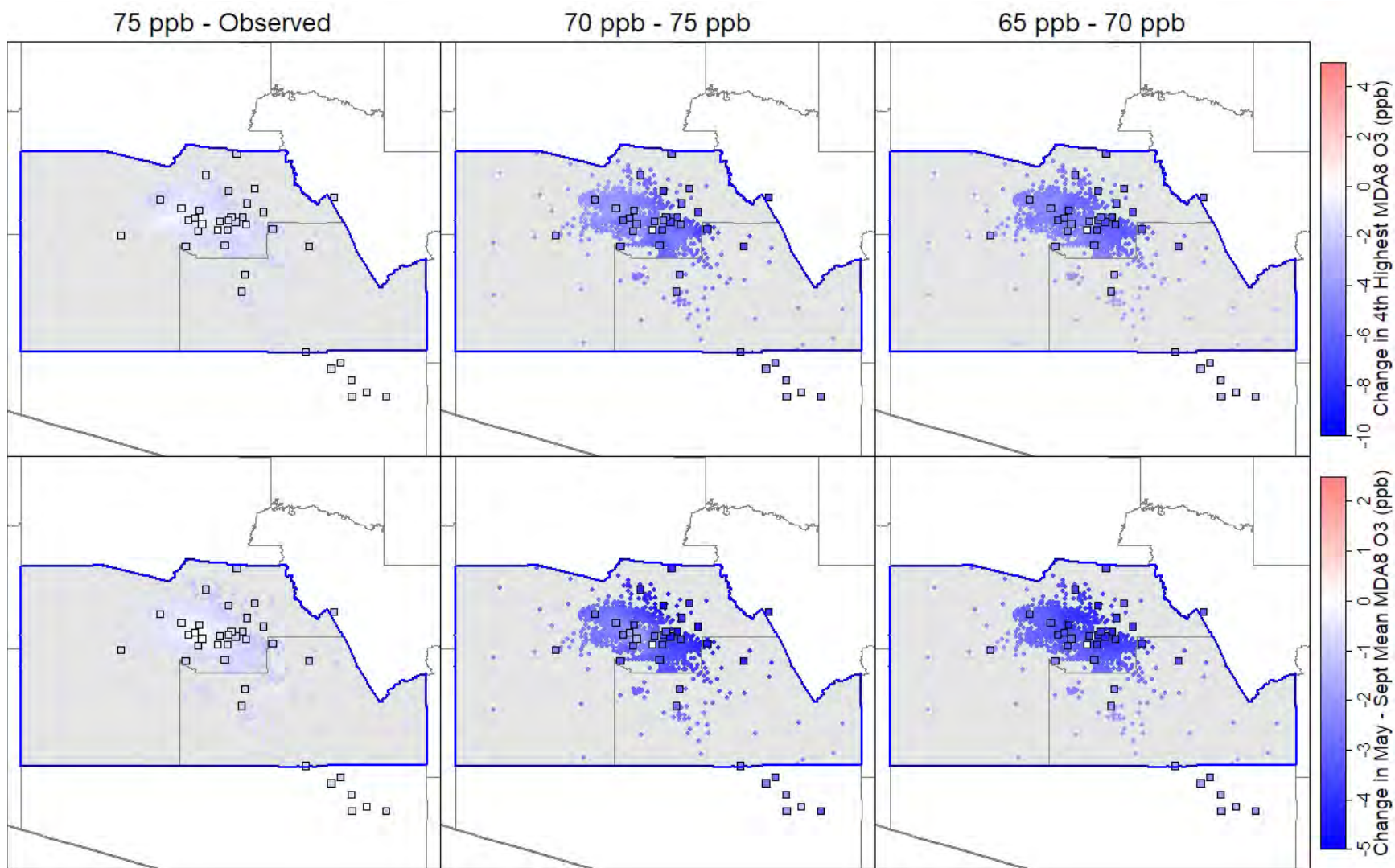
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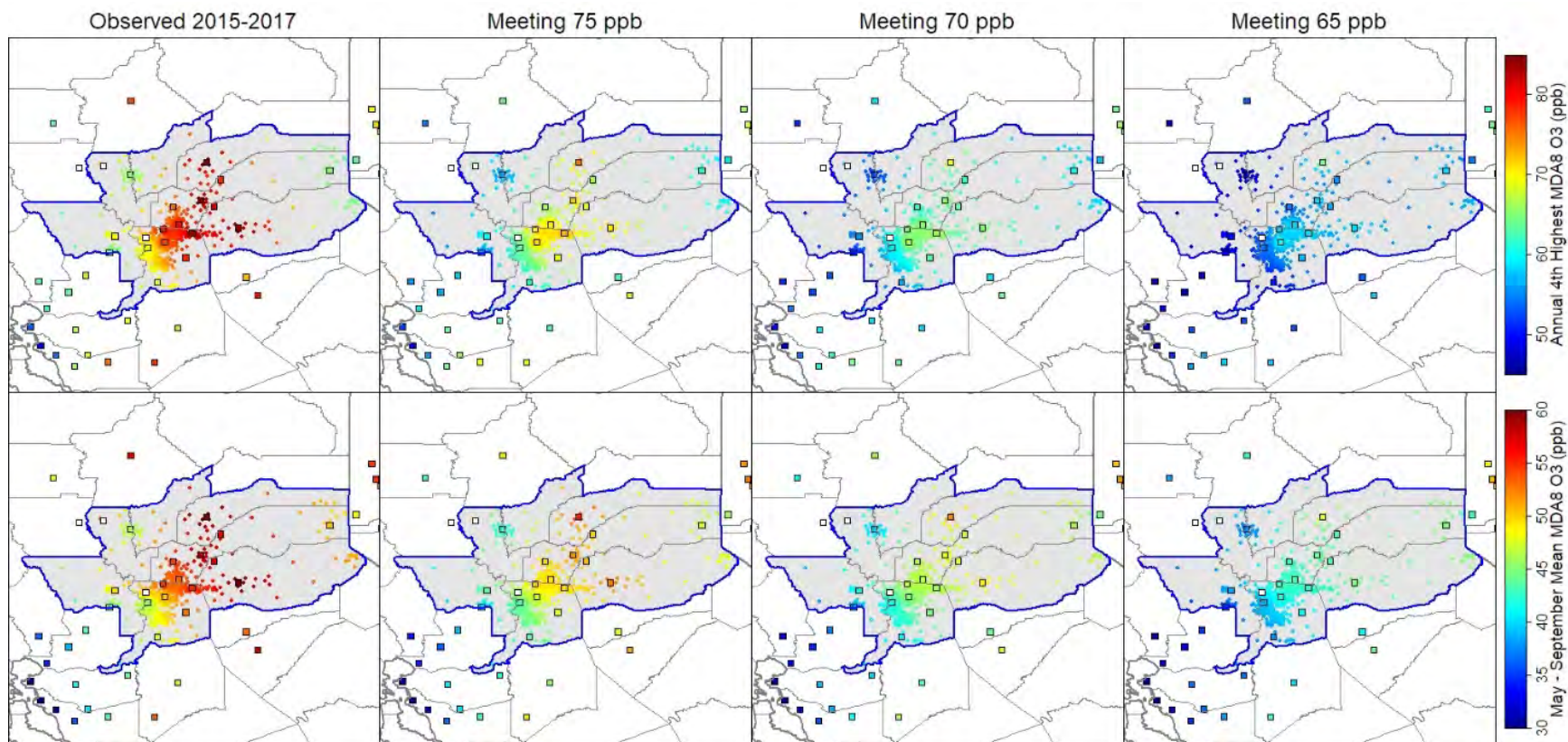
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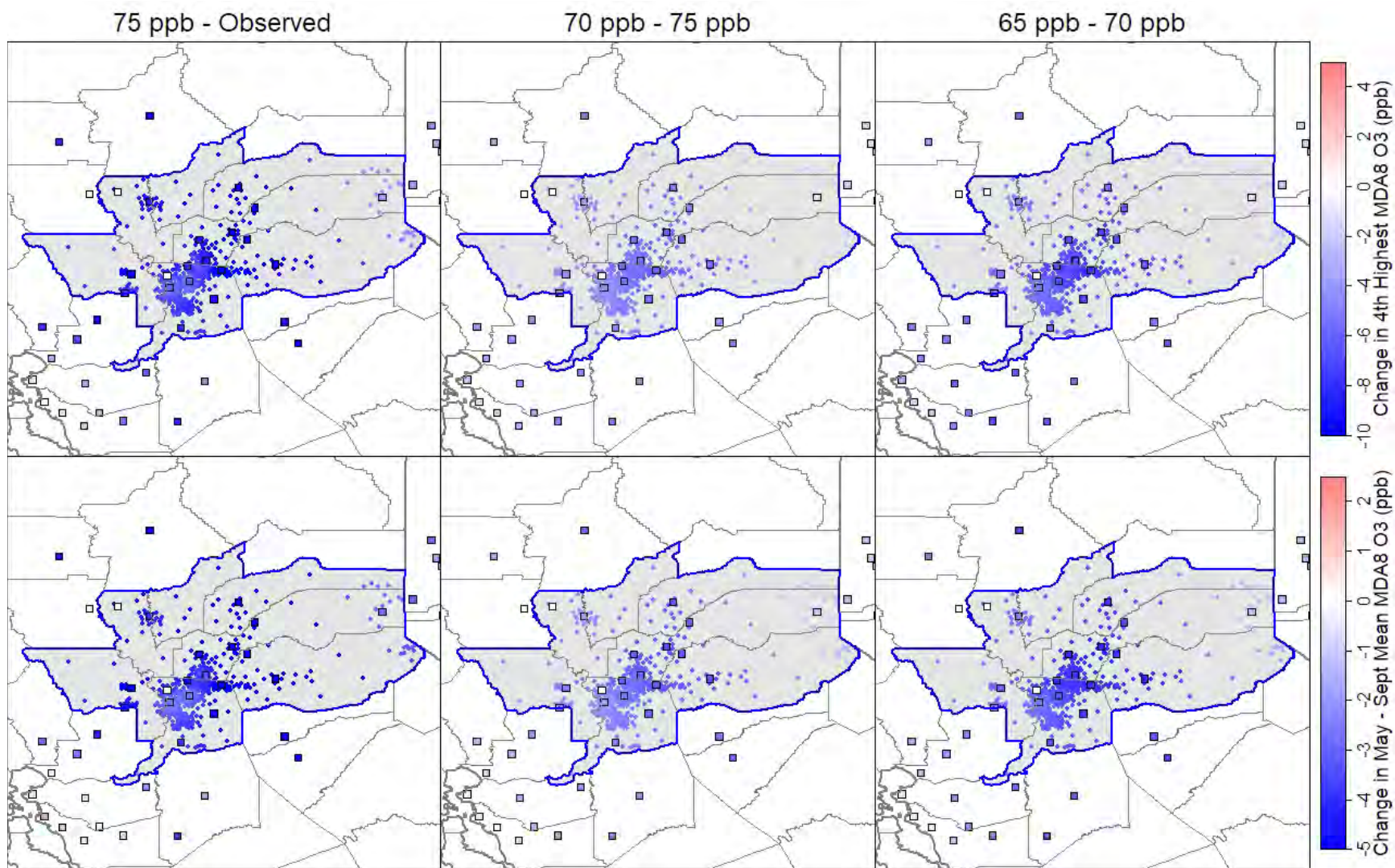
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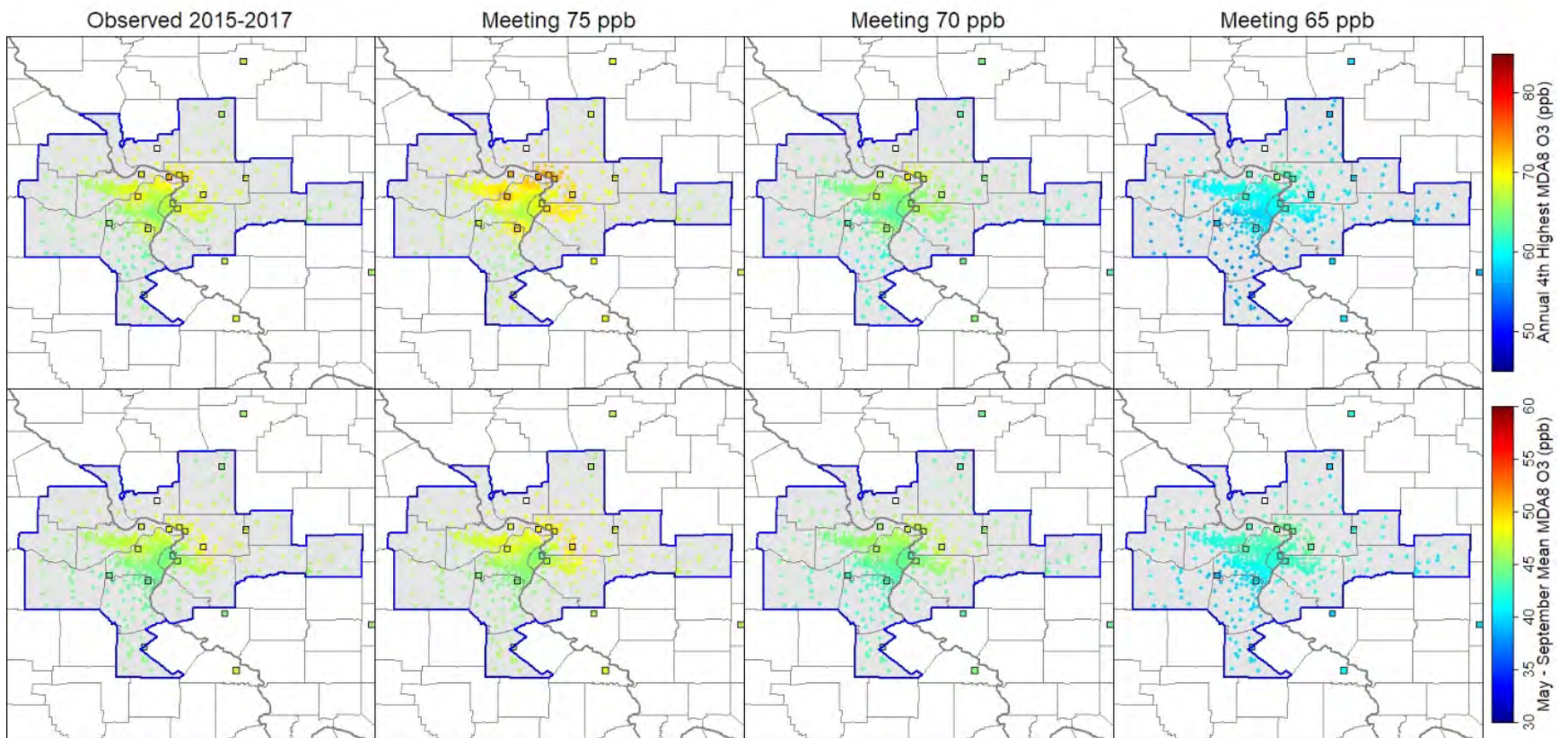
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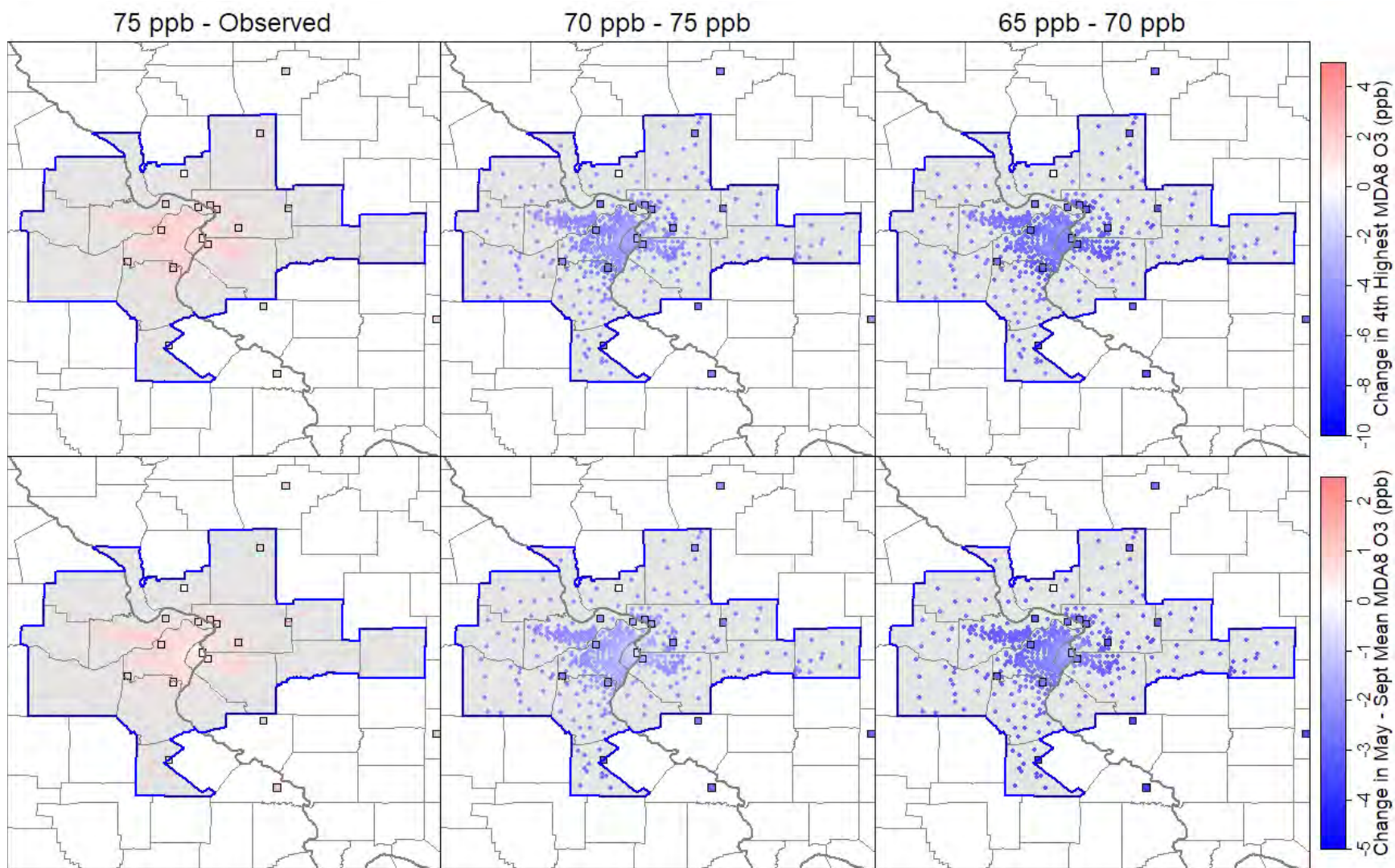
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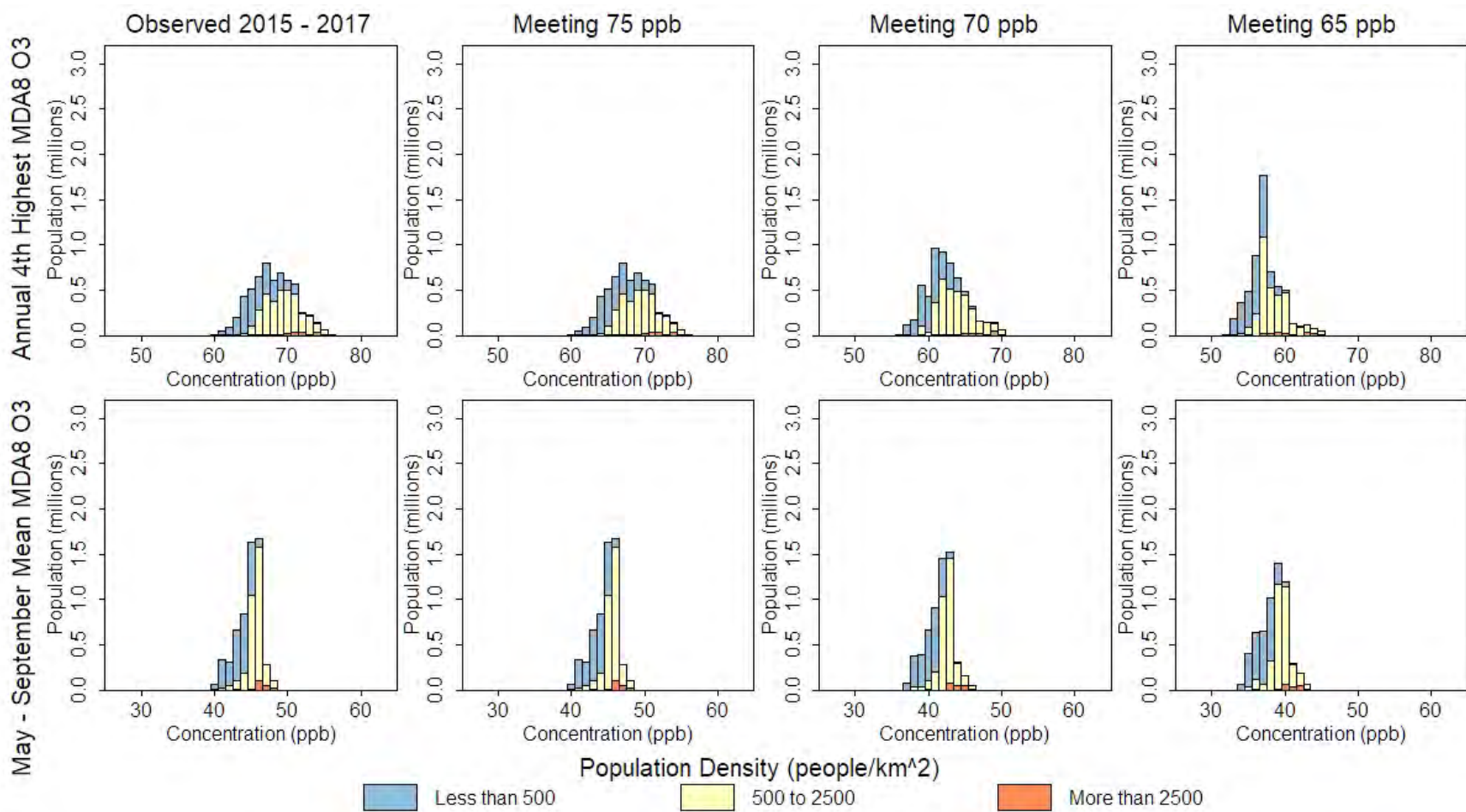
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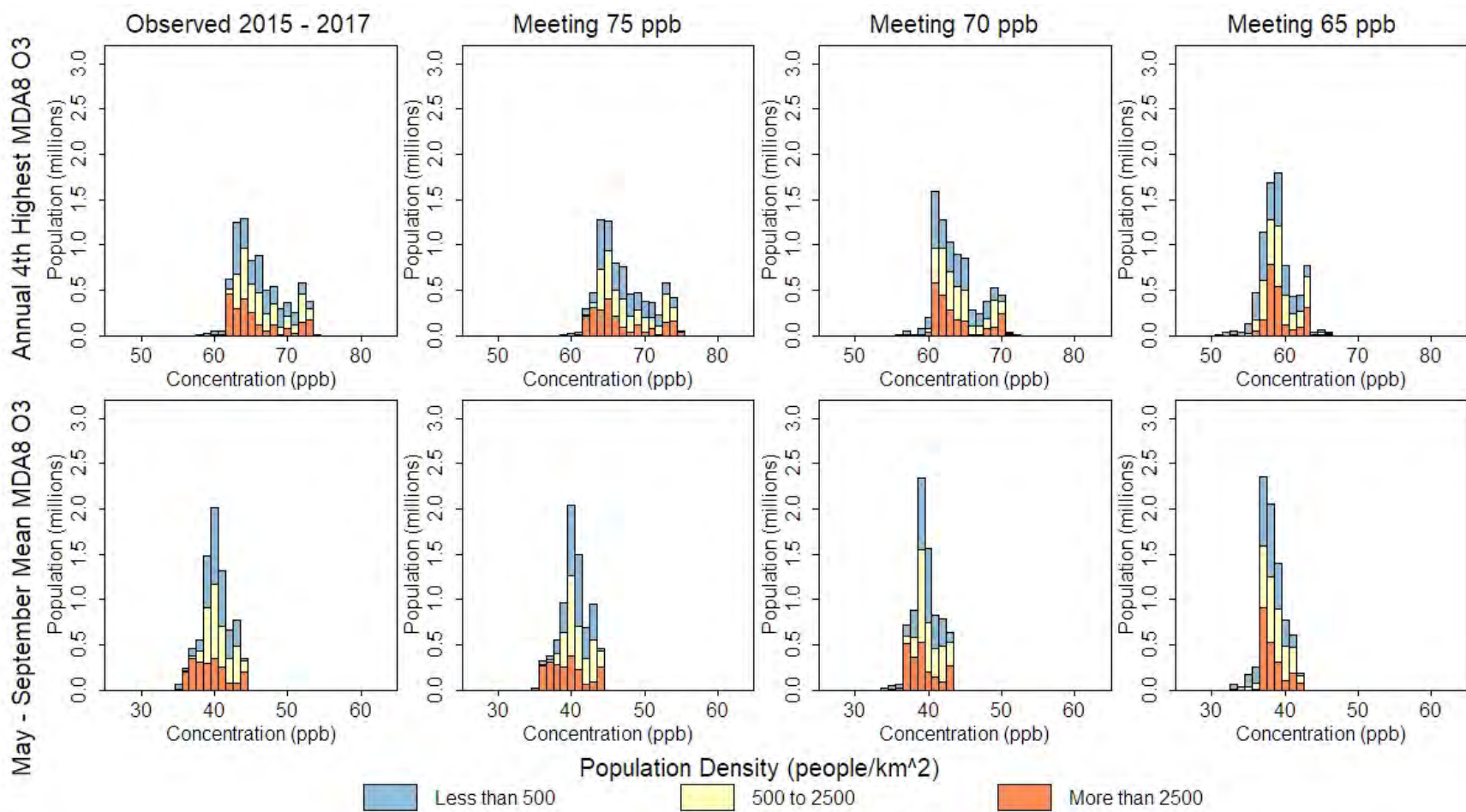
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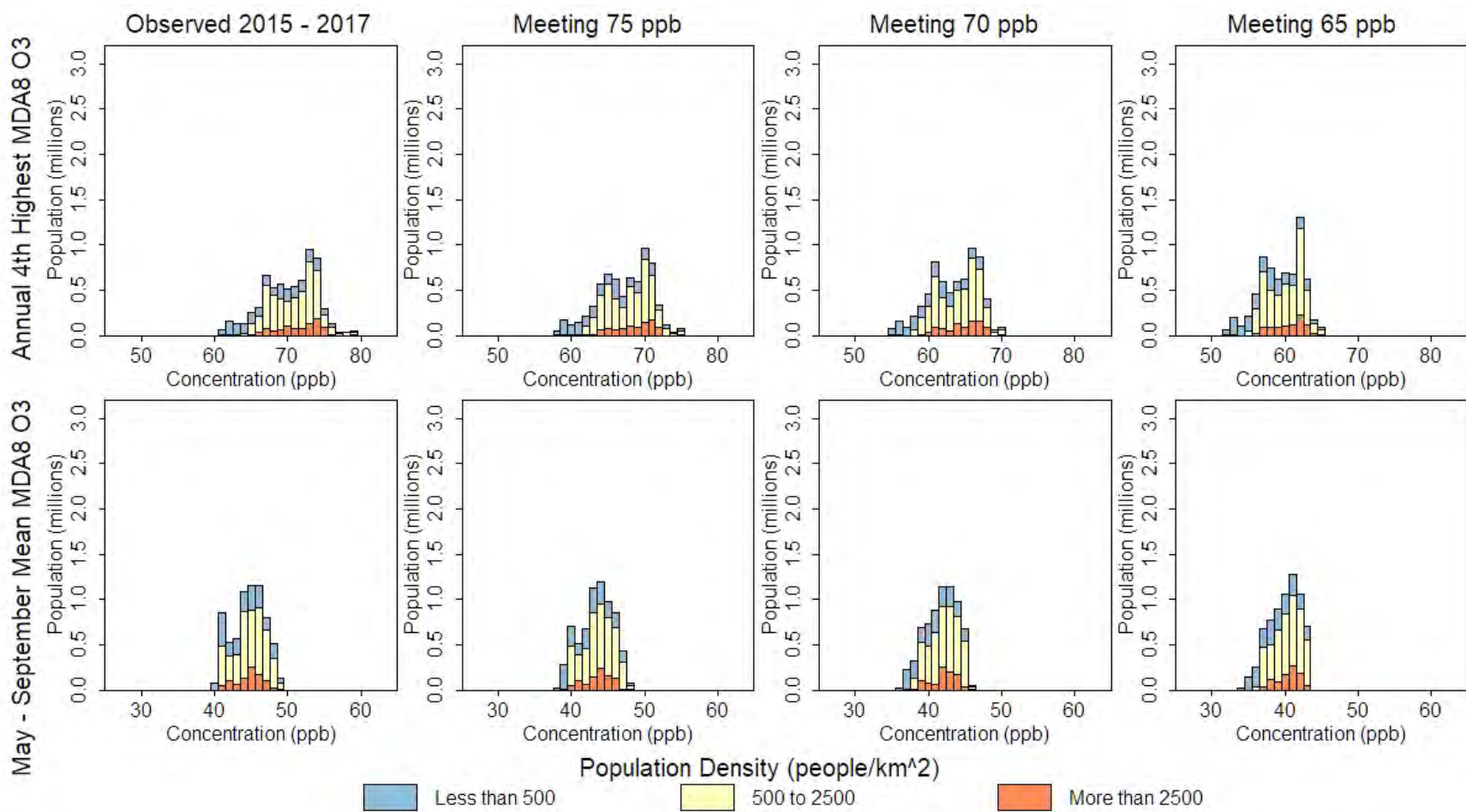
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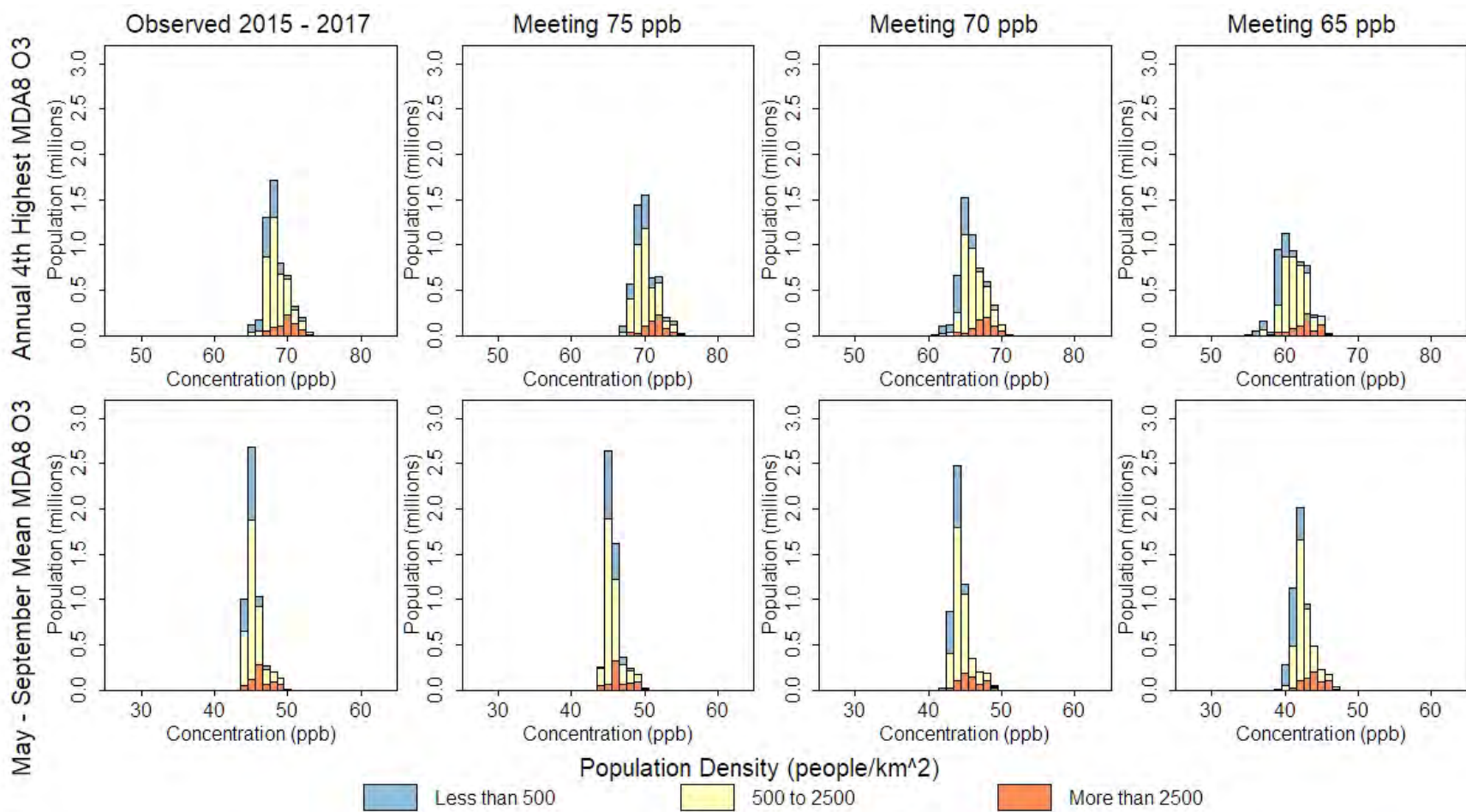
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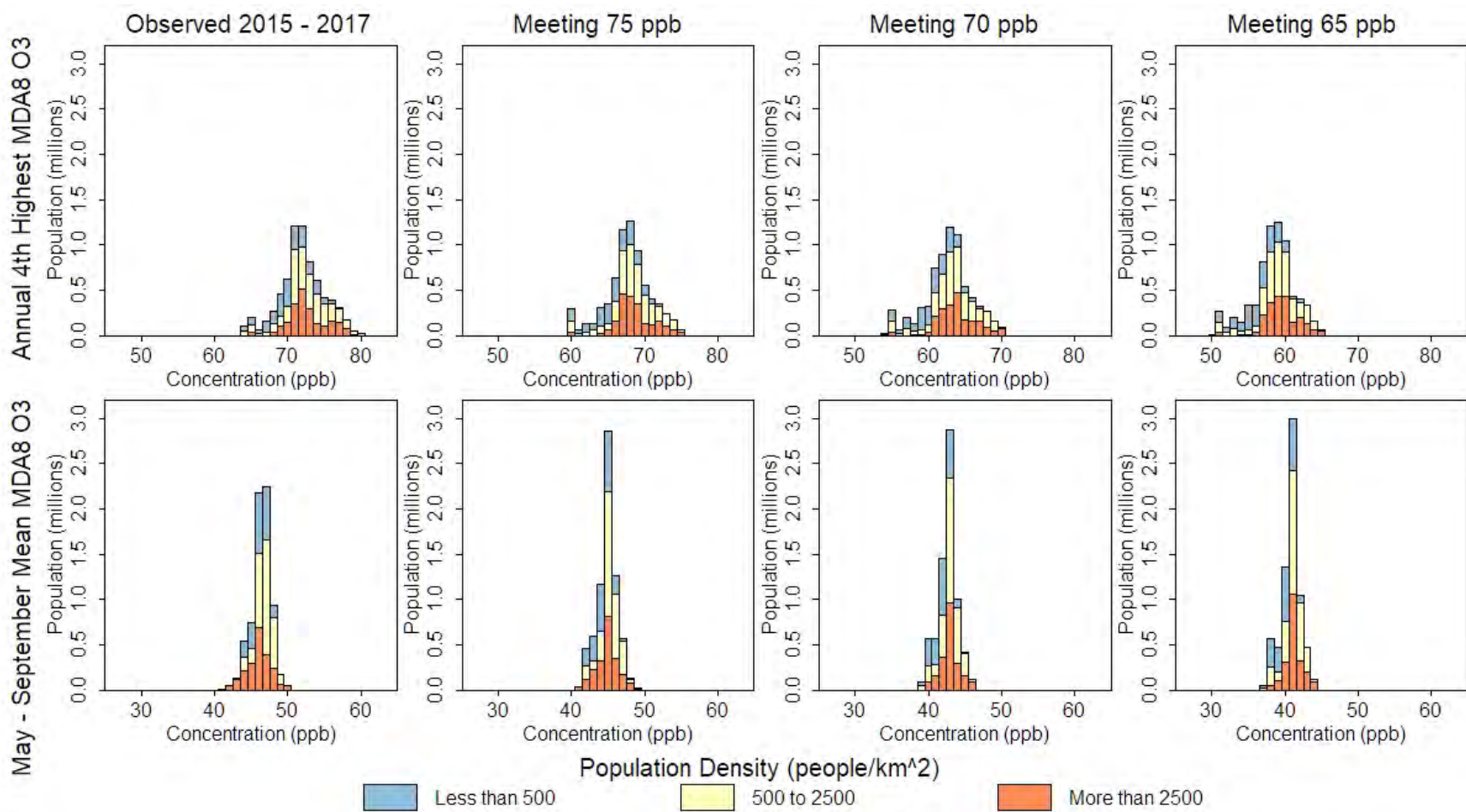
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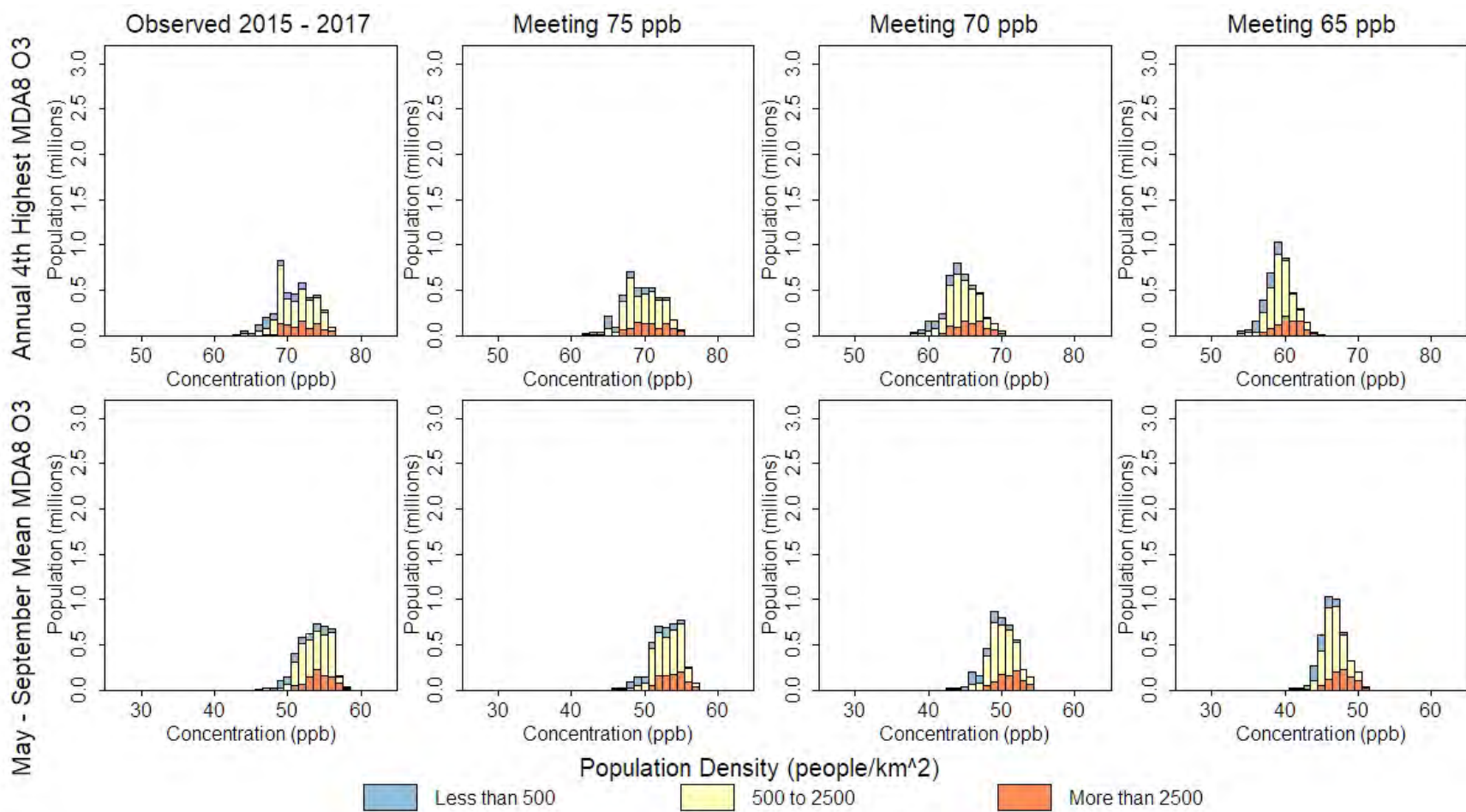
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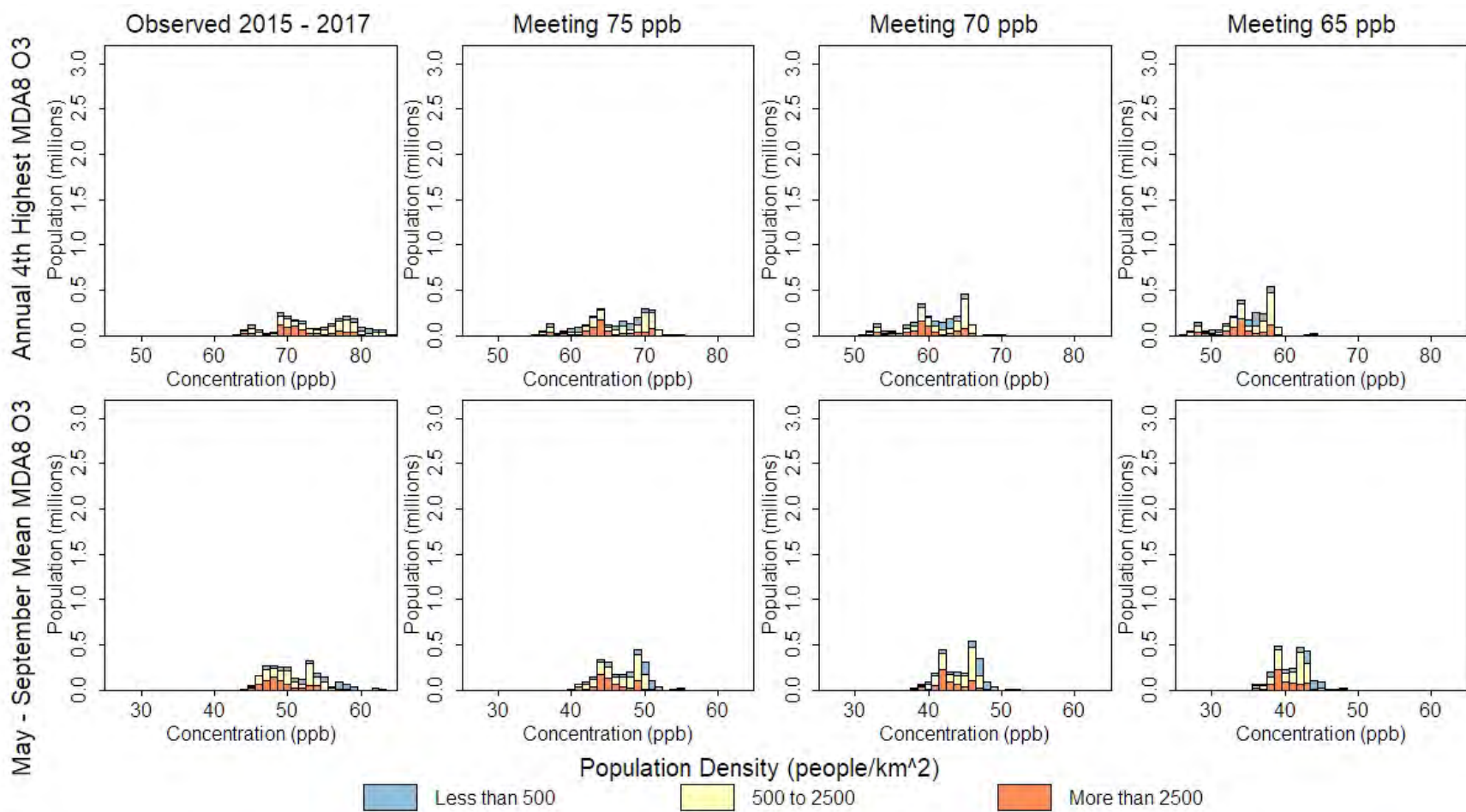
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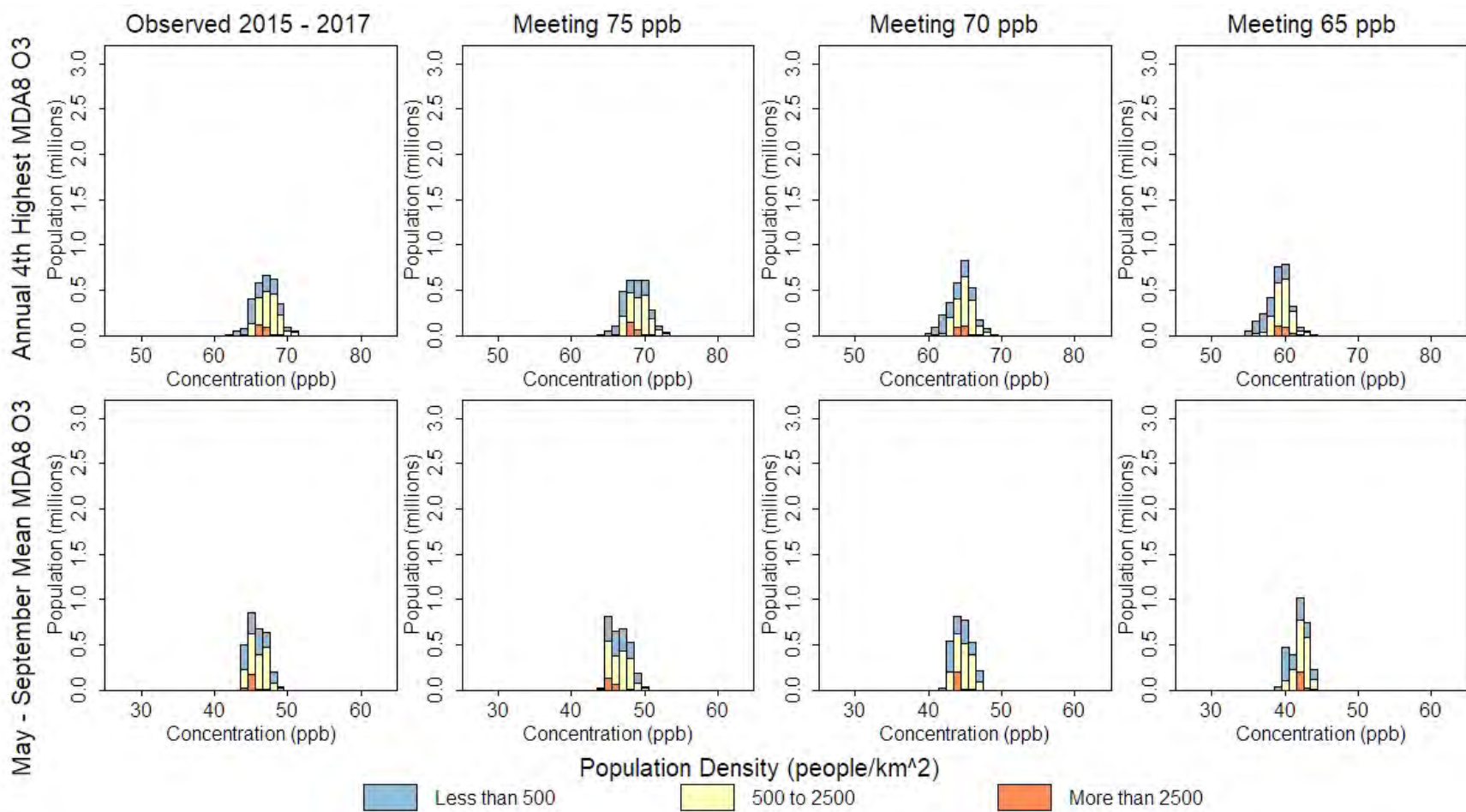
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1 **APPENDIX 3D**

2 **EXPOSURE AND RISK ANALYSIS FOR THE OZONE NAAQS REVIEW**

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3D.1 INTRODUCTION

This appendix summarizes the quantitative exposure and risk analysis performed for the 2020 O₃ NAAQS review. The analysis builds upon the methodology and lessons learned from the human exposure and risk analyses conducted in the 2015 O₃ review (2014 HREA; U.S. EPA, 2014), analysis plans outlined in the Integrated Review Plan (IRP; U.S. EPA, 2019d), and information provided in the 2020 O₃ Integrated Science Assessment (ISA; U.S. EPA, 2020a), which builds on the 2013 ISA (U.S. EPA, 2013).

Exposures and risks were modeled for people residing in eight U.S. urban study areas,¹ considering three hypothetical air quality scenarios developed from ambient air O₃ monitoring data adjusted based on a photochemical model-based approach for a single 3-year period (2015 to 2017), and based on health effects observed in controlled human exposure studies. The three air quality scenarios were for O₃ concentrations across the study area such that the location with the highest design value² just meets: (1) the current standard (i.e., a design value of 70 ppb), (2) a design value of 75 ppb, and (3) a design value of 65 ppb. The exposures and risks were estimated for (1) all school-age children (ages 5-18), (2) school-age children with asthma (ages 5-18), (3) all adults (ages 19-90),³ and (4) adults with asthma (ages 19-90),⁴ each while at moderate or greater exertion level at the time of exposure. The strong emphasis on children and people with asthma reflects the conclusion based on the currently available evidence that these are important at-risk groups, as summarized in section 3.3.2 above of the main document and described in the ISA (ISA, section IS.6.1).

Health risk is characterized in two ways in these analyses, producing two types of risk metrics: one involving comparison of population exposures, while at elevated exertion, to benchmark concentrations, and the second involving estimated population occurrences of ambient air O₃-related lung function decrements (Figure 3-3 of main document). The first risk

¹ For the 2014 HREA, controlled human exposure-based health risk was estimated in 15 urban study areas considering five air quality scenarios and two 3-year periods (2006-2008 and 2008-2010). In addition, an epidemiologic-based health risk approach was applied in 12 urban study areas also considering the same five air quality scenarios and for two single-year periods (2007 and 2009). Further, an epidemiologic-based health risk approach was applied to the continental U.S. considering a single air quality scenario (unadjusted, as is ambient air concentrations).

² The design value for these scenarios is the 3-year average of the annual 4th highest daily maximum 8-hr average O₃ concentration. For example, a monitoring site meets the current standard if the design value, derived from the data for that site, is less than or equal to 70 ppb.

³ For the 2014 HREA, older adults (ages 65-95) were simulated as a separate group. In the current assessment, older adults within this age group are included in the simulation of all adults. Additionally, the upper age limit in the current assessment is 90 years given data limitations since recognized in CHAD for older age entries.

⁴ For the 2014 HREA, adults with asthma (ages 19-95) were simulated, similar to the group simulated for the current assessment. Additionally, the upper age limit in the current assessment is 90 years given data limitations since recognized in CHAD for older age entries.

1 metric is based on comparison of estimated daily maximum 7-hour (7-hr) average exposures for
2 individuals breathing at elevated rates to concentrations of potential concern (benchmark
3 concentrations),⁵ and the second uses exposure-response (E-R) information for study subjects
4 experiencing FEV₁ decrements (specifically O₃-related decrement of 10% or more) to estimate
5 the portion of the simulated at-risk population expected to experience one or more days with an
6 O₃-related FEV₁ decrement of at least 10%, 15% and 20%.

7 A description of the exposure and risk modeling performed, including a summary of (1)
8 the ways in which scientific and public review of the current analysis occurred, and (2) the 2014
9 HREA and important updates in modeling tools and approaches that contributed to planning and
10 completion of the analyses presented in this document is provided in sections 3D.1.1 through
11 3D.1.4. The detailed description of the modeling tools, algorithms, input data and output metrics,
12 along with an assessment of how variability is addressed in the analysis is provided in section
13 3D.2. Finally, the exposure and risk results, including a characterization of uncertainties, are
14 found in section 3D.3.

15 **3D.1.1 Planning and Scientific/Public Review of the Analysis**

16 As described in section 1.4 of the main document, a consultation with the Clean Air
17 Scientific Advisory Committee (CASAC) was held in November 2018 on the draft IRP to
18 receive their input and comments from the public were also solicited on the draft IRP. Both
19 comments from the CASAC and the public were considered in shaping the analysis plans, which
20 were summarized in the final IRP.

21 This appendix was developed in support of the risk and exposure analyses for the 2020
22 review. As outlined in section 1.5 (of the main document) the draft 2019 PA for the 2020 review,
23 with a draft version of this appendix was made available for public comment and was reviewed
24 and discussed by CASAC in a public meeting (84 FR 50836, September 26, 2019; 84 FR 58711,
25 November 1, 2019). In consideration of comments from the CASAC (Cox, 2020) and the public
26 a number of additional analyses and presentations were added to this appendix in the final 2020
27 PA (U.S. EPA, 2020b). These analyses, investigations and/or clarifications of the available data
28 address a number of areas.

- 29 • Analyses of data on outdoor activity by different population groups including those
30 identified as at risk in this review (e.g., children with asthma and older adults) during
31 times of day when O₃ may be elevated (section 3D.2.5.3);

⁵ The exposure duration and approach for identifying simulated individuals at moderate or greater exertion have been updated from what was used in the 2014 HREA to more closely match the circumstances of the controlled human exposure studies, as described in section 3D.2.2.3.3 and 3D.2.8.1.

- 1 • Estimates for the comparison-to-benchmarks analysis additionally summarized in light
2 of the estimates from the last review (section 3D.3.2.4);
- 3 • Evaluation of risk characterization uncertainty related to its representation of
4 population groups having health conditions other than asthma, of older adults, and of
5 outdoor workers (section 3D.3.4.1);
- 6 • Evaluation of uncertainty in estimates for people with asthma that may be associated
7 with method for identifying individuals with asthma (section 3D.3.4.1);
- 8 • Evaluation of uncertainty with the E-R function and risk estimates (section 3D.3.4.1);
- 9 • Analyses investigating the sensitivity of the MSS model outputs to the value assigned
10 the individual variability parameter, and to low-level ventilation rates, as well as
11 overall model uncertainty in the MSS model (section 3D.3.4.1).

12 **3D.1.2 Overview**

13 Estimates of human exposure to O₃ can provide meaningful answers to policy-relevant
14 questions regarding exposures of concern and resulting risk estimates. This is particularly true
15 when the important elements of O₃ exposure, i.e., the frequency, magnitude, duration, and
16 pattern, are accounted for and when the exposures are estimated using policy-relevant ambient
17 air quality scenarios, i.e., ambient air conditions that either just meet the current O₃ standard or
18 other air quality scenarios. Further, the policy-relevance of these estimated O₃ exposures can be
19 extended when they are linked with adverse health outcome data obtained from controlled
20 human exposure studies to quantitatively estimate health risk. As a result, via the quantitative
21 relationships that exist between ambient air concentrations, exposures, and health effects, one
22 can estimate the impact varying air quality conditions have on public health.

23 Exposure to O₃ can be directly estimated by monitoring the concentration of O₃ in a
24 person's breathing zone (close to the nose/mouth) using a personal exposure monitor. Studies
25 employing this measurement approach have been reviewed in the current and 2013 O₃ ISAs and
26 in past O₃ Air Quality Criteria Documents (AQCDs; U.S. EPA, 1986, 1996, U.S. EPA, 2006).
27 Personal exposure measurements from these studies can be useful in describing a general range
28 of exposure concentrations (among other reported measurement data) and in identifying factors
29 that may influence varying exposure levels. However, these measurement studies of personal
30 exposure to O₃ are largely limited by the disparity between measurement sample durations and
31 durations of interest, and in appropriately capturing variability in population exposure occurring
32 over large geographic areas, particularly when considering both O₃ concentrations in ambient air
33 (e.g., spatial variability) and population (e.g., age, sex) attributes that greatly influence exposure.

34 Because of these limitations in personal exposure measurement data, more commonly
35 human exposure is estimated using sophisticated models that better account for physical (e.g.,
36 meteorology) or personal (e.g., age) attributes that may strongly influence variability in

1 exposures. These exposure models can combine information on ambient air O₃ concentrations in
2 various microenvironments, e.g., near roads, in schools, etc., with information on activity
3 patterns for individuals sampled from the general population or specific subpopulations, e.g.,
4 children with asthma. When integrating these varied data (among many others such as population
5 demographics and disease prevalence) and understanding the key factors affecting exposure,
6 exposure models can be more informative than the limited information given by measurement
7 data alone.

8 Ozone exposure is highly dependent on the ambient air concentrations in an urban area,
9 which vary spatially and temporally. An exposure model can reasonably estimate exposures for
10 any perceivable at-risk population (e.g., people with asthma living in a large urban area) and
11 considering any number of defined hypothetical air quality conditions (e.g., those in which
12 concentrations just meet a particular air quality standard) provided underlying data exist to
13 generate such estimates. Further, exposure models that account for variability in human
14 physiology can also realistically estimate pollutant intake dose by using activity-specific
15 ventilation rates. Each of these important features of O₃ exposure cannot realistically be
16 measured for a study group or population of interest over wide ranging temporal and spatial
17 scales, particularly when considering time, cost, and other constraints, and serve as the
18 justification for using a modeling approach to estimate exposure and health risks.

19 **3D.1.3 2014 Ozone Exposure and Risk Assessment**

20 The 2014 HREA included two types of risk analyses. The first type of risk analysis,
21 exposure-based risk, used health effect information obtained from controlled human exposure
22 studies (summarized in the IRP, section 5.1.1.1). The second type, epidemiologic-based risk,
23 used concentration-response functions derived from epidemiologic studies (IRP, section 5.1.1.2).
24 Because we used only the exposure-based risk analysis approach (see section 3D.1.4 below; IRP,
25 section 5.1.2), it is only these results that are succinctly summarized in this section.^{6,7}

⁶ Details regarding all of the risk analyses performed for the prior review can be found in chapters 5 (exposure-based health benchmark risk), 6 (exposure-based lung function risk), and 7 (epidemiologic-based risk) of the 2014 HREA.

⁷ We note that the CASAC comments on the draft PA included several related to development of risk estimates from epidemiological study results (Cox, 2020). Because an epidemiologic-based risk analysis was not performed for this review, the issues raised by those comments are not considered here.

1 For the 2014 HREA, two exposure-based risk analyses⁸ were performed in a set of 15
2 urban study areas⁹ and for five different air quality scenarios: unadjusted ambient air O₃
3 conditions, air quality adjusted to just meet the then-existing standard (75 ppb, annual 4th highest
4 daily maximum 8-hr average concentration, averaged over a 3-year period), and air quality
5 adjusted to just meet potential alternative O₃ standards having the same form and averaging
6 times, with levels of 70, 65 and 60 ppb.¹⁰ The scenarios were based on air quality from two 3-
7 year periods: 2006-2008 and 2008-2010. The first exposure-based risk analysis involved
8 comparison of population exposures, while at elevated exertion, to benchmark concentrations.
9 The exposure-to-benchmark comparison characterizes the extent to which individuals in at-risk
10 populations could experience exposures of concern (i.e., average exposure concentrations at or
11 above specific benchmarks while at moderate or greater exertion levels) while engaging in their
12 daily activities in study areas with air quality adjusted to just meet the then-existing standard and
13 other O₃ air quality conditions. Results were characterized using three benchmark concentrations
14 (60, 70, and 80 ppb O₃), exposures to which in controlled human exposure studies yielded
15 different occurrences and severity of respiratory effects in the human subjects (2014 HREA,
16 section 5.2.8). The second exposure-based risk analysis involves estimated population
17 occurrences of ambient air O₃-related lung function decrements. The lung function risk analysis
18 provides estimates of the extent to which populations in such areas could experience decrements
19 in lung function. Based on the range of health effects considered clinically relevant and the
20 potential for varied responses in healthy individuals versus people with asthma, the lung function
21 risk analysis reported estimates for risk of lung function decrement at or above three different
22 magnitudes, i.e., forced expiratory volume in one second (FEV₁) reductions of at least 10%,
23 15%, and 20% (2014 HREA, section 6.2.1).

24 Key observations and insights from the O₃ exposure-to-benchmark comparison and lung
25 function risks, in addition to important caveats and limitations, were addressed in Section II.B of
26 the Final Rule notice (80 FR 65312 to 65315, October 26, 2015). The exposure-based analyses in

⁸ For the primary analysis results in the 2014 HREA, population exposures were used to estimate health benchmark and lung function risks using an individual-based approach. In addition, a population-based E-R function approach was used to estimate lung function risk but done mainly for comparison with the individual-based approach and with prior review assessment results.

⁹ The 15 urban study areas assessed were Atlanta, Baltimore, Boston, Chicago, Cleveland, Dallas, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC.

¹⁰ These scenarios reflect air quality with design values that equal the level of the now-current standard and two others having levels just above and below the current standard. The air quality data were generated using a combined ambient monitor data and modeling approach similar to that used for the current assessment. These simulations were intended to be illustrative and do not reflect any consideration of specific control programs designed to meet the specified standards. Further, these simulations were not intended to represent predictions of when, whether, or how areas might meet a specified standard.

1 the 2014 HREA, and most particularly the exposure to benchmarks analysis were important
2 considerations in the 2015 decision on revisions to the primary O₃ standard (80 FR 65362-65365,
3 October 26, 2015).

4 **3D.1.4 Current Analysis**

5 As described in the IRP (section 5.1.2.2), the quantitative analyses for focus on the
6 comparison to benchmark exposure-based risk analysis approach, based on the controlled human
7 exposure studies. In part, this is because substantial updates to data, information, models, and
8 tools are available, ensuring that the new exposure and risk estimates are both improved and
9 appropriately targeted. Additionally, estimates from the exposure-based analyses, particularly the
10 comparison of daily maximum exposures to benchmark concentrations, were most informative to
11 the Administrator’s decision in the 2015 review (IRP, section 3.1.2). This largely reflected the
12 EPA conclusion that “controlled human exposure studies provide the most certain evidence
13 indicating the occurrence of health effects in humans following specific O₃ exposures,” and
14 recognition that “effects reported in controlled human exposure studies are due solely to O₃
15 exposures, and interpretation of study results is not complicated by the presence of co-occurring
16 pollutants or pollutant mixtures (as is the case in epidemiologic studies)” (80 FR 65343, October
17 26, 2015). In the 2015 review, the Administrator placed relatively less weight on the air quality
18 epidemiologic-based risk estimates, in recognition of an array of uncertainties, including, for
19 example, those related to exposure measurement error (80 FR 65346, October 26, 2015).

20 **3D.1.4.1 Aspects updated since 2014**

21 A number of aspects of the exposure-based risk analyses were updated since the 2014
22 HREA. The updates were based on important uncertainties characterized in the 2015 review and
23 having newly available data, information, models, and tools that could provide risk estimates in
24 which we have greater confidence that was the case for the risks estimated in the 2015 review, as
25 summarized in Appendix 5A of the IRP. These updates include:

- 26 • Air quality
 - 27 – More recent (2015-2017) ambient air monitoring data from US EPA’s Air Quality
 - 28 System (AQS) having unadjusted concentrations at or near the current standard
 - 29 (section 3D.2.3.2);
 - 30 – Updated photochemical model (CAMx version 6.5)¹¹ to adjust ambient air
 - 31 concentrations to just meet the air quality scenarios to be assessed (section
 - 32 3D.2.3.3).
- 33 • Exposure and risk model

¹¹ CAMx is the Comprehensive Air Quality Model with Extensions. This model is briefly described in Appendix 3C.
Additional information and model download can be found at <http://www.camx.com/>.

- 1 – More recent (2010) U.S. Census demographics and commuting data (section
2 3D.2.2.1);
- 3 – More recent (2013-2017) asthma prevalence for census tracts in all study areas
4 (section 3D.2.2.2);
- 5 – Updated equations to estimate resting metabolic rate (RMR) (section 3D.2.2.3.2)
6 and associated ventilation rate (\dot{V}_E) (section 3D.2.2.3.3);
- 7 – Improved matching of controlled human exposure study duration (6.6-hr) and
8 target ventilation rate to that estimated for simulated individuals (7-hr duration,
9 distribution accounting for resting ventilation) and used for benchmark
10 comparisons and population-based E-R lung function risk (section 3D.2.2.3.3 and
11 3D.2.8.1);
- 12 – More recent (2015-2017) meteorological data to reflect the assessment years
13 (section 3D.2.4)
- 14 – Increased number of diary-days and added new activity descriptions to activity
15 pattern data base (section 3D.2.5.1);
- 16 – Most recent MSS-FEV₁ model (McDonnell et al., 2013) to estimate individual
17 lung function risk (section 3D.2.8.2.2);
- 18 – New evaluations of important uncertainties (section 3D.3.4.2): form of E-R
19 function, E-R function risk confidence intervals, low exposure concentration
20 contribution to lung function risk, influence of ventilation rate on lung function
21 risk, influence of variability parameter settings in MSS-FEV₁ model.

22 **3D.2 POPULATION EXPOSURE AND RISK APPROACH**

23 This section describes the data, information, models, and tools used to characterize
24 exposure and health risk associated with O₃ in ambient air for three air quality scenarios. As
25 summarized above in section 3D.1.4, the overall analysis approach is based on linking the health
26 effects information observed in controlled human exposure studies to estimated population-based
27 exposures that reflect our current understanding of concentrations of O₃ in the ambient air.

28 Population exposures and risks were estimated using the EPA's Air Pollution Exposure
29 Model (APEX), version 5. APEX is a multipollutant, population-based, stochastic,
30 microenvironmental model that can be used to estimate human exposure via inhalation for
31 criteria and toxic air pollutants. APEX is designed to estimate human exposure to these
32 pollutants at the local, urban, and consolidated metropolitan level. In this analysis, we used
33 APEX to estimate exposure and risk in eight study areas, the details of which are provided in the
34 following subsections. Additional information not provided here regarding all of APEX modules,
35 algorithms, and modeling options can be found in the APEX User's Guide (U.S. EPA, 2019a;
36 U.S. EPA, 2019b).

37 Briefly, APEX calculates the exposure time-series for a user-specified duration and
38 number of individuals. Collectively and by design, these simulated individuals are intended to be

1 a representative random sample of the population in the chosen study area. To this end,
2 demographic data from the decennial census are used so that appropriate model sampling
3 probabilities can be derived considering personal attributes such as age and sex and used to
4 properly weigh the distribution of individuals in any given geographical area. For the exposure
5 and risk analyses performed here, the core demographic geographical units for estimating
6 exposure are census tracts. For each simulated person, the following general steps are performed:

- 7 • Select personal attribute variables and choose values to characterize the simulated
8 individual (e.g., age, sex, body weight, disease status);
- 9 • Construct an activity event sequence (a minute-by-minute time-series) by selecting a
10 sequence of appropriate daily activity diaries for the simulated individual (using
11 demographic and other influential variables);
- 12 • Calculate the pollutant concentrations in the microenvironments (MEs) that simulated
13 individuals visit;
- 14 • Calculate the simulated individual's exposure, and simultaneously, their breathing
15 rate for each exposure event and summarize for the selected exposure metric.

16 A simulated individual's complete time-series of exposures (i.e., *exposure profile*),
17 representing intra-individual variability in exposures, is combined with the exposure profiles for
18 all simulated individuals in each study area and summarized to generate the population
19 distribution of exposures, representing inter-individual variability in exposures. As described
20 above regarding air quality and in the sections that follow describing APEX model inputs and
21 approaches to estimating exposure, the overarching goal of the exposure and risk analysis is to
22 account for the most significant factors contributing to inhalation exposure and risk, i.e., the
23 temporal and spatial distribution of people and pollutant concentrations throughout the study area
24 and among the microenvironments. The population distributions of exposures are then combined
25 with the health effects information to characterize associated risk via two types of metrics: a
26 comparison to benchmark concentrations and lung function risk. The details of the model input
27 data and general approaches used for estimating exposure and risk are described in the sections
28 that follow.

29 **3D.2.1 Urban Study Areas**

30 To identify a list of urban areas for the current analysis, we first considered the list of 15
31 urban study areas evaluated in the 2014 HREA, which represented a range of geographic areas,
32 encompassing variability in air quality, climate, and population demographics. We also
33 considered other candidate study areas (e.g., Phoenix). As was done for the 2014 HREA, we
34 developed criteria to select urban study areas for the current exposure and risk analysis. Those
35 criteria are as follows:

- 1 • Have at least 10 ambient air monitors having complete year data for the 2015-2017
2 period;
- 3 • Combined statistical area (CSA)/metropolitan statistical area (MSA) ambient air
4 monitor design values are between 60-80 ppb, thus having minimal adjustment
5 needed to just meet the current 8-hr O₃ NAAQS;
- 6 • CSA/MSA population between 2 to 10 million;
- 7 • Anticipated reasonable air quality model performance¹²; and
- 8 • Reasonable geographic distribution across continental U.S.

9 Based on these selection criteria, we chose the eight study areas listed in Table 3D-1 (and
10 shown in Figure 3D-1) to develop our population exposure estimates. Included also are the nine
11 other study areas considered but not selected for the current exposure and risk analysis. We
12 recognize the Sacramento study area does not meet the design value criterion (i.e., 86 ppb is
13 outside the range of values considered), however we relaxed this criterion to include a study area
14 in the Pacific/West region of the U.S and because exposure and risk was evaluated in the 2014
15 HREA (as opposed to using Los Angeles which was also evaluated in the 2014 HREA but has a
16 2015-17 design value of 112 ppb).

17 We broadly defined the study areas using geographic coordinates to center the overall
18 exposure modeling domain for the APEX modeling (Table 3D-2). A wide city radius (i.e., 30
19 km) along with standard political/statistical county aggregations (e.g., whether in a CSA/MSA)
20 were then used to identify the specific counties that comprise each study area. As a result, 131
21 counties containing 9,725 census tracts were used to define the air quality domain in the eight
22 study areas.¹³ As done for prior exposure-based assessments, ambient air O₃ concentrations were
23 estimated to census tracts to capture spatial heterogeneity that may exist within each study area
24 (see Appendix 3C) and to link with the population input data sets (section 3D.2.2).

¹² While we expect air quality models to effectively capture relationships between ozone and its chemical precursors in most areas, there are known situations (e.g. documented influence of stratospheric ozone intrusions) that may be more challenging for air quality models to represent. We therefore excluded some of these more challenging areas from this analysis (see Table 3D-1).

¹³ The identification of specific counties and census tracts are provided in the APEX ambient air concentration input files for each study area. The approach used to estimate O₃ concentrations is summarized in section 3D.2.3 below and is described fully in the Appendix 3C of this PA.



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2

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Figure 3D-1. Locations of the eight study areas selected for the current O₃ exposure and risk analysis.

Table 3D-1. Criteria used to identify and select urban study areas for inclusion in the O₃ exposure and risk analyses.

Selected for Analysis?	Study Area	Census Division ^A	U.S. Climate Region ^B	CSA/MSA Population ^C (millions)	CSA/MSA Land Area ^D (Km ²)	Ambient Air Monitors (n)	Design Values ^E (ppb)	
							2017	2008, 2010
Yes	Atlanta	South Atlantic	Southeast	6.6	26,873	11	75	95, 80
	Boston	New England	Northeast	8.3	22,780	22	73	82, 76
	Dallas	West S Central	South	8.0	36,411	20	79	91, 86
	Detroit	East N Central	Upper Midwest	5.4	14,972	11	73	82, 75
	Philadelphia	Mid Atlantic	Northeast	7.2	15,391	19	80	92, 83
	Phoenix	Mountain	Southwest	4.9	37,725	28	76	81, 77
	Sacramento	Pacific	West	2.6	20,709	18	86	99, 99
	St. Louis	West N Central	Ohio Valley	2.9	23,504	12	72	82, 77
No	Baltimore	South Atlantic	Northeast	2.8	6,738	5	75	91, 89
	Chicago ^F	East N Central	Ohio Valley	9.9	21,941	21	78	78, 74
	Cleveland	East N Central	Ohio Valley	3.5	9,322	15	74	84, 77
	Denver ^F	Mountain	Southwest	3.6	33,824	10	79	86, 78
	Houston	West S Central	South	7.2	27,744	19	81	91, 84
	Los Angeles ^F	Pacific	West	18.8	87,943	41	112	119, 112
	New York ^F	Mid Atlantic	Northeast	23.5	30,544	36	83	89, 82
	Salt Lake City ^F	Mountain	Southwest	2.6	46,517	10	78	82, 74
	Washington DC	South Atlantic	Southeast	6.2	14,341	15	71	87, 81

^A U.S. Census Division data are found at: <https://www.ncdc.noaa.gov/monitoring-references/maps/us-census-divisions.php>.

^B U.S. Climate Region data are found at: <https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php>.

^C U.S. Census CSA/MSA population data are found at: <https://www.census.gov/data/tables/time-series/demo/popest/2010s-total-metro-and-micro-statistical-areas.html>.

^D U.S. Census land area data taken from "G001 Geographic Identifiers, 2010 SF1 100% data file" available at: <https://factfinder.census.gov/faces/nav/jsf/pages/searchresults.xhtml?refresh=t>.

^E Ozone ambient air monitor design values (see .xlsx sheet 'Table6. Monitor Trends') are found at: <https://www.epa.gov/air-trends/air-quality-design-values>.

^F Potential air quality modeling/adjustment issues: VOC-limited (Chicago, Denver), stratospheric O₃ issues (Denver), low monitor density (Salt Lake City), monitor issues (New York), and high DVs (Los Angeles).

1 **Table 3D-2. General description of ambient air quality domains for the eight study areas.**

CSA/MSA			Coordinates		Counties ^A (n)	Tracts (n)
			Longitude (degrees)	Latitude (degrees)		
Name	ID#	Abbrev.				
Atlanta-Athens-Clarke County- Sandy Springs, GA-AL	122	ATL	-84.3880	33.7490	39	1,077
Boston-Worcester-Providence, MA-RI-NH-CT	148	BOS	-71.0589	42.3601	19	1,753
Dallas-Fort Worth, TX-OK	206	DAL	-96.7970	32.7767	21	1,422
Detroit-Warren-Ann Arbor, MI	220	DET	-83.0458	42.3314	10	1,583
Philadelphia-Reading-Camden, PA-NJ-DE-MD	428	PHI	-75.1652	39.9526	16	1,725
Phoenix-Mesa, AZ	429	PHX	-112.0740	33.4484	2	988
Sacramento-Roseville, CA	472	SAC	-121.4944	38.5816	7	539
St. Louis-St. Charles-Farmington, MO-IL	476	STL	-90.2003	38.6303	17	638

^A Delineations promulgated by the Office of Management and Budget (OMB) in February of 2013 (see Appendix 3C, section 3C.2).

2

3 **3D.2.2 Simulated Populations**

4 APEX stochastically generates a user-specified number of simulated people to represent
5 the population in the study area. The number of simulated individuals can vary and is dependent
6 on the size of the population to be represented. For the current analysis, the number of simulated
7 individuals was set at 60,000 for each of the children and adult study groups (which includes
8 people with asthma for both of these study groups) to represent population residing within each
9 study area (i.e., between 2 and 10 million). Each simulated person is represented by a *personal*
10 *profile*. The personal profile includes specific attributes such as an age, a home tract, a work tract
11 (or is not employed), housing characteristics, physiological parameters, and so on. The profile
12 does not correspond to any particular individual that resides in the study area, but rather
13 represents a simulated person. Accordingly, while a single profile does not, in isolation, provide
14 information about the study population, a distribution of profiles represents a random sample
15 drawn from the study area population. As such, the statistical properties of the distribution of
16 simulated profiles are meant to reflect statistical properties of the population in the study area.

17 APEX generates population-based exposures using several population databases. Based
18 on the geographic boundaries defining the study areas and the study groups of interest, APEX
19 simulates representative individuals using appropriate geographic, demographic, and health
20 status information provided by existing population-based surveys. For the current exposure and
21 risk analysis, population input data sets are organized by U.S. census tracts.

1 Several updates were made to the APEX model inputs and algorithms for use in
2 simulating the populations of interest in this exposure and risk analysis and are described in the
3 following sections: population demographic data that are based on the 2010 census (section
4 3D.2.2.1), asthma prevalence rates based on the 2013-2017 National Health Interview Survey
5 (NHIS) that vary by age, sex and geographic location (section 3D.2.2.2), and data and equations
6 used to approximate personal attributes such as body weight, resting metabolic rate, and
7 breathing rate (section 3D.2.2.3).

8 **3D.2.2.1 Demographics**

9 As briefly described in section 3D.2.1 (and more fully in section 3D.2.3 below and in
10 Appendix 3C), ambient air concentrations were modeled to census tracts in each study area to
11 capture spatial heterogeneity in ambient air O₃ concentrations. Population data were generated
12 using the same spatial scale to also account for variability in population demographics. Tract-
13 level population counts were obtained from the 2010 Census of Population and Housing
14 Summary File 1.¹⁴ Summary File 1 contains what the Census program calls “the 100-percent
15 data,” which is the compiled information from the questions asked of all (100% of) people and
16 housing units in the U.S. Three national-based APEX input files¹⁵ are used for the current
17 exposure and risk analysis as follows.

- 18 • *Population_sectors_US_2010.txt*: census tract identifiers (IDs), latitudes and
19 longitudes in degrees.
- 20 • *Population_female_All_2010.txt*: census tract IDs, tract-level population counts for
21 females, stratified by 23 age groups.¹⁶
- 22 • *Population_male_All_2010.txt*: census tract IDs, tract-level population counts for
23 males, stratified by the same 23 age groups as done for females.

24 **3D.2.2.2 Asthma Prevalence**

25 The four population study groups included in this exposure assessment are adults (19 to
26 90 years old),¹⁷ children (5 to 18 years old),¹⁸ and those within each of the two groups having

¹⁴ Technical documentation - 2010 Census Summary File 1—Technical Documentation/prepared by the U.S. Census Bureau, Revised 2012 - available at: <http://www.census.gov/prod/cen2010/doc/sfl.pdf>.

¹⁵ The names of all APEX files are provided here to link the brief description with the appropriate APEX input file.

¹⁶ The age groups in this file are: 0-4, 5-9, 10-14, 15-17, 18-19, 20-20, 21-21, 22-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-61, 62-64, 65-66, 67-69, 70-74, 75-79, 80-84, >84.

¹⁷ The upper limit for adults was set to age 90 due to the limited information available in CHAD for modeling activity patterns and physiological processes for adults >90.

¹⁸ As in other NAAQS reviews, we do not estimate exposures and risk for children younger than 5 years old due to the more limited information contributing relatively greater uncertainty in modeling their activity patterns and physiological processes than children between the ages of 5 to 18.

1 asthma, based on their identification as an at-risk population (section 3.3.2 of the main
2 document; ISA, section IS.4.4.2). To best approximate the number (and percent) of individuals
3 comprising the latter two population groups in each study area, we considered several influential
4 variables that could affect asthma prevalence. It is widely recognized that there are significant
5 differences in asthma prevalence based on age, sex, U.S. region, and family income level, among
6 other factors.¹⁹ There is spatial heterogeneity in family income level across census geographic
7 areas (and also across age groups)²⁰ and spatial variability in local scale ambient air
8 concentrations of O₃ (e.g., Appendix 3C, Figures 3C-91 through 3C-106). Thus, we accounted
9 for these particular attributes of this study group and their spatial distribution across each of the
10 study areas to better estimate the variability in population-based O₃ exposures and risks for these
11 at-risk population groups.

12 With regard to asthma prevalence, the data are used to identify if a simulated individual
13 residing within a modeled census geographic area has asthma. The data are not used for selection
14 of any other personal attribute nor in the selection of activity pattern data. Thus, our primary
15 objective with these data was to generate census tract-level prevalence that reflect variability in
16 asthma prevalence contributed by several known influential attributes (i.e., age, sex, family
17 income level, geographic location). Two data sets were identified and linked together to estimate
18 asthma prevalence used for this exposure and risk analysis: asthma prevalence and population
19 data.

20 First, asthma prevalence data were obtained from the 2013-2017 National Health
21 Interview Survey (NHIS) and are stratified by NHIS defined regions (Midwest, Northeast, South,
22 and West), age, and sex.²¹ These asthma prevalence data are particularly useful given that age is
23 expressed as a continuous variable, a feature not found in other asthma prevalence data that are
24 available (e.g., state or county level data). We explored variables that were available in the NHIS
25 data set that contributed to variability in asthma prevalence and that could be used to extrapolate
26 the asthma prevalence to a finer geographic scale than the NHIS-provided four regions. The
27 linking variable had to be common with variables available in the population demographic data.
28 Based on this criterion, we selected family income level to poverty thresholds (i.e., whether the
29 family income was considered at/below or above a factor of 1.5 of the U.S. Census estimate of
30 poverty level for the given year) and used that as an additional variable to stratify the NHIS
31 asthma prevalence.

¹⁹ For example, see the Center for Disease Control report “National Surveillance of Asthma: United States, 2001–2010”, available at: https://www.cdc.gov/nchs/data/series/sr_03/sr03_035.pdf.

²⁰ For example, see the U.S. Census report “Income and Poverty in the United States: 2016”, available at: <https://www.census.gov/content/dam/Census/library/publications/2017/demo/P60-259.pdf>.

²¹ Information about the NHIS is available at: <http://www.cdc.gov/nchs/nhis.htm>.

1 Then, we obtained population data from the 2017 Census American Community Survey
2 (ACS) to estimate family income level to poverty thresholds at the census tract level and
3 stratified by several ages and age groups.²² By combining the NHIS and U.S. Census population
4 data sets, we developed census tract level asthma prevalence for children (by age in years) and
5 adults (by age groups), also stratified by sex (male, female) that were weighted by the individual
6 census tract population and family income level proportions. Finally, we adjusted the census
7 tract-level asthma prevalence data based on individual state-level prevalence data from the 2013-
8 2016 Behavioral Risk Factor Surveillance System (BRFSS).²³ This was done because overall, the
9 asthma prevalence data reported from BRFSS were consistently higher than that derived from
10 the NHIS data, particularly when considering adults, and thus resulted in an upward adjustment
11 to the initially derived NHIS census tract level data set. A detailed description of how the NHIS,
12 U.S. Census, and BRFSS data were processed and combined to create the data set used for input
13 to APEX is provided in Attachment 1. The national-based APEX input file is used for the current
14 exposure and risk analysis as follows:

- 15 • *asthma_prev_1317_tract_053119_adjusted.txt*: census tract IDs, tract-level asthma
16 prevalence (in fractional form) stratified by sex, 18 single year ages (for ages <18),²⁴
17 and 7 age groups (for ages > 17).

18 The asthma prevalence varies for the different ages and sexes of children and adults²⁵ that
19 reside in each census tract of each study area. We evaluated the spatial distribution of the asthma
20 prevalence using the tracts that comprise the air quality domain in each study area. We first
21 separated the estimates for children from those for adults and calculated the distribution of
22 asthma prevalence for the tracts, stratified by sex (Table 3D-3). These summary statistics
23 represent the range of age- and sex-specific probabilities for the census tracts comprising each
24 study area that are used by APEX to estimate the number of individuals that have asthma.
25

²² Census tract level data is the finest scale geographical unit having family income information. The family income/poverty ratio threshold used was 1.5, that is the surveyed person's family income was considered either ≤ or > than a factor of 1.5 of the U.S. Census estimate of poverty level for the given year.

²³ Table C2.1 (for each adults and children) was downloaded to obtain the 2013-2016 BRFSS current asthma prevalence by state and sex, available at: <https://www.cdc.gov/asthma/brfss/default.htm>. Table C1 was also downloaded to obtain the asthma prevalence for the two age groups not stratified by sex. Accessed 5/3/19.

²⁴ The census data only had children for single years up to and including age 17, after that age they are provided in groups. The upper portion of this age range differs from those considered as children in estimating exposures (i.e., in our exposure assessment children are considered upwards to 18 years old). To simulate the number of children with asthma age 18, estimated prevalence from the first adult group were used (i.e., individuals age 18-24).

²⁵ While prevalence was estimated for all ages of children (in single years 5-17), for adults they were estimated for seven age groups: 18-24 years, 25-34 years, 35-44 years, 45-54 years, 55-64 years, 65-74 years, and ≥75 years old (see Attachment 1 for more information).

1 **Table 3D-3. Descriptive statistics for children and adult asthma prevalence, using all**
 2 **census tracts within eight consolidated statistical areas (CSAs) in the APEX**
 3 **asthma prevalence file.**

CSA Name - ID# (# tracts) and Population group		Sex	Asthma Prevalence across all ages (or age groups) and census tracts ^A						
			Mean	Standard Deviation	Minimum	Median	95 th percentile	99 th percentile	Maximum
Atlanta-122 (1,077)	adult	female	11.1%	1.8%	7.7%	11.1%	14.0%	15.9%	20.9%
		male	5.5%	0.8%	4.3%	5.4%	7.1%	7.5%	7.9%
	child	female	9.7%	1.7%	6.5%	9.6%	12.9%	13.9%	15.0%
		male	14.1%	1.7%	10.6%	14.0%	16.8%	17.6%	18.3%
Boston-148 (1,753)	adult	female	13.8%	1.8%	10.5%	13.5%	17.3%	20.5%	28.9%
		male	7.6%	0.9%	5.4%	7.5%	9.1%	10.0%	12.9%
	child	female	9.4%	2.0%	5.6%	9.5%	12.4%	13.5%	17.1%
		male	15.4%	2.5%	8.7%	15.1%	19.5%	20.8%	23.4%
Dallas-206 (1,422)	adult	female	9.3%	1.5%	6.5%	9.3%	11.8%	13.5%	16.5%
		male	4.9%	0.7%	3.8%	4.9%	6.4%	6.8%	9.7%
	child	female	7.6%	1.3%	5.0%	7.4%	10.0%	10.9%	13.5%
		male	11.0%	1.4%	8.3%	11.0%	13.2%	13.8%	18.1%
Detroit-220 (1,583)	adult	female	13.3%	2.5%	7.8%	13.4%	17.8%	20.6%	25.6%
		male	7.9%	2.2%	1.0%	7.6%	12.4%	14.7%	19.0%
	child	female	8.6%	1.5%	6.4%	8.2%	11.6%	12.5%	13.2%
		male	13.3%	3.0%	7.7%	12.7%	19.9%	23.6%	25.5%
Philadelphia-428 (1,725)	adult	female	12.1%	2.3%	8.2%	12.0%	16.4%	19.8%	26.5%
		male	6.5%	0.9%	4.6%	6.4%	8.1%	9.0%	11.4%
	child	female	9.1%	1.9%	5.6%	9.2%	12.0%	13.1%	15.3%
		male	13.6%	2.4%	8.2%	13.3%	17.8%	19.2%	21.1%
Phoenix-429 (988)	adult	female	11.6%	1.6%	8.6%	11.7%	14.4%	16.0%	19.7%
		male	7.0%	1.5%	5.1%	7.1%	9.1%	11.7%	16.7%
	child	female	7.6%	1.5%	4.6%	8.0%	9.5%	9.6%	9.6%
		male	11.5%	1.8%	8.5%	11.6%	14.8%	15.9%	17.1%
Sacramento-472 (539)	adult	female	10.4%	1.4%	7.7%	10.5%	12.7%	14.0%	16.5%
		male	5.7%	1.1%	4.2%	5.9%	7.3%	9.0%	13.6%
	child	female	8.5%	1.7%	5.2%	9.0%	10.7%	10.9%	10.9%
		male	10.8%	1.7%	8.1%	10.9%	13.7%	14.8%	16.2%
St. Louis-476 (638)	adult	female	11.8%	2.1%	6.8%	11.9%	15.0%	17.4%	21.5%
		male	6.5%	1.8%	0.9%	6.5%	9.9%	11.8%	14.5%
	child	female	9.2%	2.0%	5.3%	9.1%	12.9%	14.2%	15.6%
		male	11.1%	2.4%	6.5%	10.7%	15.9%	19.3%	21.9%

^A Prevalence is based on single year ages (children) or age groups (adults) and sex derived from 2013-2017 CDC NHIS asthma prevalence and considering U.S. census tract level family income/poverty ratio data. Data presented are not population-weighted and represent the distribution of applied probabilities used by APEX for tracts having a non-zero population. Note, upper and lower percentiles could represent prevalence for a single year age/sex residing in a single tract within a study area.

1 In general and consistent with broadly defined national asthma prevalence (e.g., Table 3-
2 1 of the main document), male children have higher rates than female children²⁶ and adult
3 females have higher rates than adult males.²⁷ The overall asthma prevalence for children was
4 similar to that estimated for adults, largely the result of having a greater BRFSS adjustment
5 applied to adult females compared to that applied to children of either sex.²⁸ As described above,
6 and by design (i.e., in using age, sex, and family income variables) there is wide ranging spatial
7 variability in the estimated asthma prevalence. For instance, the Boston, Detroit, and
8 Philadelphia study areas have some of the highest asthma prevalence for boys and adult women
9 considering most of the descriptive statistics, with rates of 25% or higher in one or more census
10 tracts for a given year of age (Table 3D-3). In contrast, the Dallas study area exhibits some of the
11 lowest asthma prevalence (and low variability) for any of the four age/sex groups compared to
12 the other study areas.

13 There are other personal attributes shown to influence asthma prevalence, such as race,
14 ethnicity, obesity, smoking, health insurance, and activity level (e.g., Zahran and Bailey, 2013).
15 The set of variables chosen to stratify asthma prevalence for use in this exposure and risk
16 analysis (i.e., age, sex, and family income level) was based on maximizing the potential range in
17 asthma prevalence variability, maximizing the number of survey respondents comprising a
18 representative subset study group, and having the ability to link the set of attributes to variables
19 within the Census population demographic data sets. Many of the additional influential factors
20 identified here are not available in the census population data and/or have limited representation
21 in the asthma prevalence data (e.g., the survey participant does/does not have health insurance, or
22 they did/did not provide a response to a question regarding their body weight). Race is perhaps
23 the only attribute common to both the prevalence and population data sets that could be an
24 important influential factor and was not directly used to calculate asthma prevalence. However,
25 the use of race in calculating asthma prevalence, either alone or in combination with family
26 income level, would further stratify the NHIS analytical data set and appreciably reduce the
27 number of individuals of specific age, sex, race, and family income level, potentially reducing
28 the confidence in calculated asthma prevalence based on having so few data in a given

²⁶ Population weighted asthma prevalence, when not categorized by the eight study areas, is greater in boys (mean of 11.1%) than that of girls (mean of 7.3%). Nationally, asthma prevalence for boys is 9.5%, for girls is 7.3% (Table 3-1 of the PA).

²⁷ Population weighted asthma prevalence, when not categorized by the eight study areas, is greater in women (mean of 12.0%) than that of men (mean of 6.5%). Nationally, asthma prevalence for women is 9.8%, for men is 5.4% (Table 3-1 of the PA).

²⁸ Population weighted asthma prevalence, when not categorized by the eight study areas and sex, is similar for children (mean of 9.2%) and adults (mean of 9.3%). Nationally, asthma prevalence for children is 8.4% and for adults is 7.7% (Table 3-1 of the PA).

1 stratification. Because family income level already strongly influences asthma prevalence across
2 all races and stratifies the NHIS data into only two subgroups (i.e., above or below the poverty
3 threshold) in comparison to the larger number of subgroups a race variable might yield, family
4 income was chosen as the next most important variable beyond age and sex to rely on for
5 weighting the spatial distribution of asthma prevalence.

6 **3D.2.2.3 Personal Attributes**

7 In addition to using the above demographic information to construct the simulated
8 individuals, each modeled person is assigned anthropometric and physiological attributes by
9 APEX. All of these variables are treated probabilistically, accounting for interdependencies
10 where possible, and reflecting variability in the population. It is not the intention of this
11 document to provide detailed description of all the model inputs in each of the files and the data
12 used in their derivation, and where additional details exist, appropriate reference materials are
13 provided. We describe further a few APEX model inputs that have been recently updated and
14 that are available for use in this exposure and risk analysis. These are new statistical distributions
15 for estimating body weight, equations for estimating resting metabolic rate, and equations for
16 estimating activity-specific ventilation rate. Each of these data and algorithms are important,
17 particularly the ventilation rate (section 3D.2.2.3.3), because the health response observed in the
18 controlled human exposure studies is concomitant with elevated breathing rate. Brief
19 descriptions of the data used to develop these generalized (i.e., non-O₃ specific) input files are
20 provided in the sections below. For additional detail, see U.S. EPA (2018) Appendices G and H,
21 and the data within the APEX input files.

22 **3D.2.2.3.1 Body Weight and Surface Area**

23 Anthropometric attributes utilized by APEX in various assessments for estimating
24 exposures or doses can include height, body weight (BW), and body surface area (BSA). Two
25 key personal attributes determined for each individual in this assessment are BW and BSA, both
26 of which are used in the calculation of a number of other variables associated with estimating
27 exposures (e.g., ventilation rate).

28 Regarding the estimation of body weight, a new APEX input file was recently generated
29 using 2009-2014 National Health and Nutrition Examination Survey (NHANES) data.²⁹ Briefly,
30 body weight and height data for surveyed individuals were obtained and stratified by sex and
31 single years for ages 0 – 79; all ages above 80 were combined as a single age group. Statistical
32 form of the age- and sex-specific body weight and height distributions were evaluated using a

²⁹ NHANES questionnaire datasets for 2009-2010, 2011-2012, 2013-2014 are available at
<https://wwwn.cdc.gov/nchs/nhanes/Default.aspx>. Details regarding the data used and the derivation of the APEX
input file data distributions is found in U.S. EPA (2018), Appendix G.

1 log-likelihood statistic. Body weight was found to best fit a lognormal distribution; height was
2 found to best fit a normal distribution. Because height and body weight are not independent, the
3 joint distributions of height and logarithm of body weight were fit assuming a bivariate normal
4 distribution. Then, parameters defining the joint distributions³⁰ were smoothed using a natural
5 cubic spline to have them represent continuous functions of age rather than vary discontinuously.
6 In addition, having the smoothed parameters could be used to extrapolate information obtained
7 from the single age year distributions (ages 0 – 79) to approximate statistical distributions of
8 body weight for ages ≥ 80 . To do so, a linear function was fit to ages 70 and above to extrapolate
9 the parameter values (and hence the statistical distributions of body weight) up to age 100.

10 These body weight distributions are randomly sampled by APEX to estimate an age and
11 sex-specific body weight for each simulated individual. Comparison of the new distributions to
12 the body weight distributions previously used by APEX and developed from the 1999-2004
13 NHIS indicate, for both sexes and across all ages, simulated body weight is about two percent
14 greater using the updated distributions. This difference is expected given the consistent trend of
15 increasing body weight that has occurred in the U.S. population over the past few decades.

16 Age- and sex-specific body surface area, a variable used in conjunction with breathing
17 rate to approximate moderate or greater exertion (section 3D.2.2.3.3) is estimated for each
18 simulated individual (Equation 3D-1) and is based on an equation provided in Burmaster (1998):

$$19 \quad \text{BSA} = e^{-2.2781} \times \text{BW}^{0.6821} \quad \text{Equation 3D-1}$$

20 One standard APEX input file is used for the current O₃ exposure and risk analysis:

- 21 • *Physiology051619_Ufixed.txt*: Provides parameters for estimating body weight (log BW,
22 standard deviation of BW, lower and upper bounds of BW, by single age years 0-100 and
23 by two sexes) and regression coefficients used in estimating BSA for all sexes and ages.

24 **3D.2.2.3.2 Energy Expenditure and Oxygen Consumption Rates**

25 Energy expended by different individuals engaged in different activities can have an
26 important role in pollutant-specific exposure and/or dose. For example, energy expenditure is
27 related to ventilation rate, which is an important variable in estimating exposure and risk given
28 that the O₃-induced lung function response has been documented to occur under conditions of
29 elevated ventilation (section 3.3.1.1 of the main document). In addition, because we are also
30 interested in exposures that occur over relatively short durations (i.e., < 8 hours), estimating
31 activity-specific ventilation rate (\dot{V}_E) has always been an important motivation behind the
32 development of the algorithm used by APEX. The fundamental basis for \dot{V}_E algorithm is founded
33 in energy expenditure which, for our modeling purposes here, can be related to an individual's

³⁰ Five parameters were used for each age and sex: mean log(BW), standard deviation of log(BW), mean height, standard deviation of height, and body weight-height correlation coefficients.

1 resting metabolic rate (RMR) or the energy expended while an individual is at complete rest,
2 along with the energy expended while an individual performs activities involving greater
3 exertion, termed here as metabolic equivalents of work (METs) (McCurdy, 2000). The
4 approaches used by APEX for estimating RMR and METs are described below, beginning first
5 with the update to the equations used for estimating a simulated individual’s RMR.

6 Since the 2014 HREA,³¹ we have reviewed recent RMR literature and other published
7 sources containing individual data and have compiled the associated individual RMR
8 measurements, along with associated influential attributes such as age, sex, and body weight,
9 where available. Data from these individual studies were then combined with RMR data reported
10 in the Oxford-Brookes database (Henry, 2005; IOM, 2005) and screened for duplicate entries. In
11 addition, observations missing values for RMR, BW, age, or sex were deleted, resulting in a
12 dataset containing 16,254 observations (9,377 males and 6,877 females). Using this new RMR
13 dataset and having a goal of updating the previous RMR equations and reducing discontinuities
14 in RMR between age groups, new equations were developed.

15 Details regarding the data, the derivation, and performance evaluation of the new
16 equation that APEX uses to estimate RMR are provided in U.S. EPA (2018), Appendix H.
17 Briefly, the equations follow the general format of a multiple linear regression (MLR) model,
18 using age and body weight as independent variables to estimate each simulated individual’s
19 RMR, along with a residual error term (ϵ).³² It is known that RMR and BW, as well as RMR and
20 age, are not exactly linearly related; the algorithms developed here use BW (in kg), age (in
21 years), and the natural logarithms of BW and (age+1)³³ as follows in Equation 3D-2, with their
22 parameter estimates provided in Table 3D-4.

23
$$RMR = \beta_0 + \beta_1 BW + \beta_2 \log(BW) + \beta_3 Age + \beta_3 \log(Age) + \epsilon_i \quad \text{Equation 3D-2}$$

24 When comparing observed versus predicted values, the new RMR equations have a bias
25 of less than 0.5%, compared to the previously used APEX equations which had a bias of between
26 1-2%. Further, the discontinuities in RMR seen across particular age group boundaries using the

³¹ The algorithm used to estimate RMR for the 2014 HREA was based on analyses by Schofield (1985) who used clinical subject data from studies conducted as far back as 60 years prior to that publication. In addition, the Schofield (1985) RMR equations contained abrupt discontinuities at some of the equation boundaries (e.g., between age 59 and 60). As a result, we felt it was important to obtain newly available study data to develop RMR equations that better represent a more recent population and having fewer discontinuities.

³² The residual error term largely accounts for the estimation of inter-personal variability in RMR for individuals having the same body weight and age. There are other potentially influential sources of variability that are not explicitly accounted for by the equation (e.g., seasonal influences on RMR) and thus remain as an uncertainty.

³³ The “+1” modifier allows APEX to round age upwards instead of downwards to whole years, which is necessary to avoid undefined log(0) values.

1 previous equations have been reduced when using these updated equations in APEX. One
 2 standard APEX input file is used for the O₃ exposure and risk analysis:

- 3 • *Physiology051619_Ufixed.txt*: Regression coefficients used to estimate RMR (kcal day⁻¹)
 4 for two sexes and six age groups.

5 **Table 3D-4. Regression parameters used to estimate RMR by sex and age groups.**

Sex	Age Group	Subjects (n)	BW	log(BW)	Age	log(Age)	Intercept	Standard Deviation
male	0–5	625	13.19	270.2	-18.34	131.3	-208.5	69.10
	6–13	1355	10.21	260.2	13.04	-205.7	333.4	115.3
	14–24	4123	0.207	1078.0	115.1	-2794.0	3360.6	161.1
	25–54	2531	2.845	729.6	3.181	-191.6	-1067	178.2
	55–99	743	9.291	264.8	-5.288	181.5	-705.9	163.6
female	0–5	625	11.94	261.5	-22.31	120.9	-183.6	64.16
	6–13	1618	5.296	409.1	40.37	-524.9	392.7	99.43
	14–29	2657	0.968	676.9	40.89	-1002	772.7	143.1
	30–53	1346	4.935	355.4	16.28	-896.0	2225	145.3
	54–99	631	2.254	445.9	5.464	-489.9	944.2	124.5

Units: RMR = kilocalories/day; BW = kilograms; Age = years

6
 7 Following the estimation of an age- and sex-specific RMR for simulated individuals, the
 8 next variable used for estimating ventilation rate involved an approximation of the energy
 9 expended for activities an individual performs throughout their day. As mentioned above,
 10 activity-specific energy expenditure is highly variable and can be estimated using metabolic
 11 equivalents of work (METs), or the ratios of the rate of energy consumption for non-rest
 12 activities to the resting metabolic rate of energy consumption, as follows in Equation 3D-3:

13
$$EE = MET \times RMR$$
 Equation 3D-3

14 where,

- 15 EE = Energy expenditure (kcal/minute)
- 16 MET = Metabolic equivalent of work (unitless)
- 17 RMR = Resting metabolic rate (kcal/minute)

18
 19 Statistical distributions of METs were developed for simulated activities using the
 20 physical-activity compendium (Ainsworth et al., 2011; hereafter “the compendium”). The
 21 compendium contains a point value for the MET associated with each of several hundred
 22 different activities. Activity-specific MET distributions were developed by cross-walking the

1 activities described in the compendium with the descriptions of activities in the activity pattern
2 data base used by APEX (section 3D.2.5). The shape of the statistical distribution (e.g., normal,
3 lognormal, triangular, point) for each activity was assigned based on the number of
4 corresponding activities in the compendium and goodness-of-fit statistics. When simulating
5 individuals, APEX randomly samples from the activity-specific METs distributions to obtain
6 values for every activity performed. Two standard APEX input files are used for the current O₃
7 exposure and risk analysis:

- 8 • *MET_distributions_092915.txt*: MET distribution number, statistical form, distribution
9 parameters, lower and upper bounds, activity description
- 10 • *MET_mapping_071018.txt*: activity codes, age group (where applicable), occupation
11 group, MET distribution number, and activity description used to link of MET
12 distributions to activities performed

13 The rate of oxygen consumption ($\dot{V}O_2$, Liters min⁻¹) for each activity is then calculated
14 from the energy expended (kcal min⁻¹) using an energy conversion factor (ECF, Liters O₂ kcal⁻¹)
15 as follows in Equation 3D-4:

$$16 \quad \dot{V}O_2 = EE \times ECF \quad \text{Equation 3D-4}$$

17 The value of the ECF is randomly selected from a uniform distribution for each person,
18 U[0.20, 0.21] (Johnson, 2002, adapted from Esmail et al., 1995). One standard APEX input file
19 is used for the current O₃ exposure and risk analysis:

- 20 • *Physiology051619_Ufixed.txt*: Parameters of the uniform distribution representing the
21 ECF used for all ages and both sexes.

22 **3D.2.2.3.3 Ventilation Rate**

23 Human activities are variable over time, with a wide range of activities possible within
24 only a single hour of the day. The type of activity an individual performs, such as sleeping or
25 jogging (as well as individual-specific factors such as age, weight, RMR) will influence their
26 ventilation rate. APEX estimates minute-by-minute ventilation rates that account for the
27 expected variability in the activities performed by simulated individuals. Ventilation rate is
28 important in this assessment because the lung function responses associated with short-term O₃
29 exposures coincide with moderate or greater exertion (2013 ISA, Table 6-1). In our exposure
30 modeling approach, APEX generates the complete time-series of activity-specific ventilation
31 rates and the corresponding time-series of estimated O₃ exposures and is directly used for the
32 individual-based lung function risk (section 3D.2.8.2.2). APEX can then aggregate both the
33 ventilation rate and exposure concentration for the duration of interest (e.g., 7-hr average), and
34 they can be used for the benchmark comparison (section 3D.2.8.1) and estimating the
35 population-based lung function risk (section 3D.2.8.2.1). Thus, the model provides O₃ exposure

1 estimates for the simulated individuals that pertain to specific target levels for both ventilation
2 rate and exposure concentration. The approach to estimating activity-specific energy expenditure
3 and associated ventilation rate involves several algorithms and physiological variables, with
4 details found in the APEX User’s Guide (U.S. EPA, 2019a, U.S. EPA, 2019b).

5 Using the existing measurement \dot{V}_E dataset from Graham and McCurdy (2009), new \dot{V}_E
6 algorithms were developed for predicting activity specific \dot{V}_E in the individuals simulated by
7 APEX (Appendix H of U.S. EPA (2018)). The new \dot{V}_E algorithms do not directly employ
8 previously used variables to stratify the data (age groups, sex) and explain variability (age, body
9 weight, height) in ventilation rate, effectively simplifying and reducing the number of equations.
10 The new algorithms utilize a new variable, the maximum volume of oxygen consumed ($\dot{V}O_{2m}$)
11 as an input.³⁴ Body weight, height, and sex – as well as fitness level (which is often represented
12 by $\dot{V}O_{2m}$) - influence oxygen consumption for a particular activity. However, variability for
13 each of these influential variables are already captured in the algorithm used to estimate each
14 simulated individual’s RMR, and subsequently, the estimation of their activity-specific $\dot{V}O_2$.³⁵
15 Thus, the only input variables needed for the new \dot{V}_E algorithm are $\dot{V}O_2$ and $\dot{V}O_{2m}$,³⁶ both of
16 which are estimated by APEX.

17 Details for the derivation of and performance evaluation of the new equation that APEX
18 uses to estimate ventilation rate are provided in U.S. EPA (2018) Appendix H. Briefly, the \dot{V}_E
19 dataset contains 6,636 observations, with 4,565 males and 2,071 females. Similar to the earlier
20 ventilation equation by Graham and McCurdy (2009), a mixed-effects regression (MER) model
21 was fit because the MER separates residuals into within-person (e_w) and between-person (e_b)
22 effects, known as intrapersonal and interpersonal effects, respectively.³⁷ It was found that the
23 actual values of $\dot{V}O_2$ and $\dot{V}O_{2m}$ are less relevant than the fraction of maximum capacity,
24 represented by $f_1 = \dot{V}O_2/\dot{V}O_{2m}$. The variable f_1 may operate non-linearly (for example, $f_1 = 0.9$ is
25 likely *more* than twice as encumbering as $f_1 = 0.45$). A transformation regression approach

³⁴ Use of $\dot{V}O_{2m}$ as an explanatory variable in separate related research on metabolic equivalents of task (MET) values for persons with unusual maximum capacity for work suggests that their MET distributions are modified in a predictable way by their maximum MET (or, equivalently, by $\dot{V}O_{2m}$), thus providing support for use of this variable in the new \dot{V}_E algorithms. Details are provided in Appendix H of U.S. EPA (2018).

³⁵ Oxygen consumption associated with activities performed is based on the activity specific metabolic equivalents for work (METs), an individual’s estimated RMR, and an energy to oxygen conversion factor (Equations 3D-3 and 3D-4 above).

³⁶ Distributions of $\dot{V}O_{2m}$ used by APEX were derived from 20 published studies reporting individual data and grouped mean (and standard deviation) data obtained from 136 published studies. Details are provided in Isaacs and Smith, 2005 (and found in Appendix B of U.S. EPA (2009)).

³⁷ $N(0, e_b)$ is a normal distribution with mean zero and standard deviation $e_b = 0.09866$ meant to capture interpersonal variability, which is sampled once per person. $N(0, e_w)$ is an intrapersonal residual with standard deviation of $e_w = 0.07852$, which is sampled daily due to natural intrapersonal fluctuations in \dot{V}_E that occur daily.

1 (using PROC TRANSREG; SAS, 2017) was used to determine the most appropriate variable
2 transformation, indicating a power of 4 to 5 be used when only the log transformed $\dot{V}O_2$ was
3 used as the independent variable and described in Equation 3D-5.

$$4 \quad \dot{V}_E = e^{(3.300 + 0.8128 \times \ln(\dot{V}O_2) + 0.5126 \times (\dot{V}O_2 \div \dot{V}O_2m)^4 + N(0, e_b) + N(0, e_w))} \quad \text{Equation 3D-5}$$

5 In comparing the statistical fit of the new equation with the equations used by APEX
6 previously to estimate ventilation rate, the resulting coefficient of determination (R^2 values) for
7 the new equation ($R^2 = 0.94$) indicates an improved fit compared to that of the previous
8 equations ($R^2 = 0.89 - 0.92$). Further, because the data were not stratified by age groups (or any
9 other groupings), there are no discontinuities in predictions made across age boundaries as was
10 observed when employing the previous equations. Information used in estimating ventilation rate
11 is found in the following APEX two input files:

- 12 • *Physiology051619_Ufixed.txt*: parameters describing statistical distributions of
13 normalized maximum oxygen consumption rate ($N\dot{V}O_2m$) for two sexes by single age
14 years (0-100) (see, Isaacs and Smith, 2005).
- 15 • *Ventilation_062117.txt*: minimum and maximum age ranges, regression coefficients,
16 between and within error terms used to estimate individual activity-specific
17 ventilation.

18 To use this information to estimate health risks for children, the ventilation rates observed
19 for the adult controlled human exposure study subjects need to be converted into rates that best
20 reflect the different physiology of children. Consistent with prior REAs (U.S. EPA, 2009, 2014,
21 2018; Whitfield et al., 1996), we used an equivalent ventilation rate (EVR, L/min- m^2), which is
22 essentially an allometrically normalized ventilation rate (Equation 3D-6), to estimate instances
23 when any simulated individual reaches a ventilation rate as relatively as high as that of the study
24 subjects (i.e., termed here as moderate or greater exertion).

$$25 \quad EVR = \frac{\dot{V}_E}{BSA} \quad \text{Equation 3D-6}$$

26 Before discussing the value used to determine whether a simulated individual is at
27 moderate or greater exertion, a brief description of the controlled human exposure study protocol
28 is warranted. Most of the controlled human exposure studies evaluating O_3 health effects of
29 interest for our exposure benchmark analysis (e.g., Adams, 2006; Folinsbee et al., 1988) were
30 conducted over a 6.6-hr exposure period, thus, the most relevant exposures and associated
31 breathing rates for the exposure benchmark comparisons would be those occurring on average
32 over a 6.6-hr period (not an 8-hr period as was used in previous REAs). The typical protocol for
33 the 6.6-hr controlled human exposure studies employed a mixture of exercise and rest periods
34 varied across the duration of the study, with an expectation that the study subject achieves, on

1 average, a target EVR of 20 L/min-m² (i.e., a ventilation rate of ~35 L/min in females and ~40
2 L/min in males) while exercising using a treadmill or cycle ergometer (e.g., Schelegle et al.,
3 2009). Most researchers collected the ventilation data during periods of exertion and therefore
4 reported the exercise-only conditions (e.g., Horstman et al., 1990; Folinsbee et al., 1988).

5 More specifically, during the 6.6-hr study experiments, 5 hours were used for exercise
6 (i.e., six 50-minute (min) periods on a treadmill or cycle ergometer), with the remaining 1.6
7 hours comprised of a series of 10-min rest periods occurring immediately after the exercise along
8 with a 35-min lunch break before the fourth exercise period. As a result of these rest/lunch
9 periods, the study subject's actual ventilation rates (and hence EVRs) are expected to be less than
10 the target/observed exercise levels reported in the controlled human exposure studies. Note, the
11 simulated individuals used to estimate exposure and risk perform numerous activities throughout
12 the day, each having varied durations and exertion levels (e.g., jogging, sleeping, eating). As
13 such, when time-averaging across a simulated exposure period of interest, the period likely
14 would contain ventilation rates of varying duration and intensity. To better match the ventilation
15 information obtained from the controlled human exposure studies with that of the simulated
16 individuals, we accounted for the impact from the rest/lunch time ventilation rate along with that
17 attained during exercise to estimate an appropriate EVR for the study subjects.

18 Attachment 2 provides details regarding the data and approach used to estimate the EVR,
19 an APEX model variable used to identify when a simulated individual is at moderate or greater
20 exertion. Briefly, the controlled human exposure study data set available used to calculate EVR
21 was comprised of 177 study subjects, each evaluated for 2 or more exposure levels (i.e., totaling
22 485 experiments), and having multiple measurements for each exercise period, yielding 4,024
23 individual EVR data points. Of these six studies providing raw data,³⁸ only Schelegle et al.
24 (2009) mentioned resting \dot{V}_E (and hence a resting EVR), with an average value for males and
25 females estimated as 7.61 and 8.05 L/min-m², respectively and based on regression equations
26 provided by Aitken et al. (1986). We calculated total (exercise and rest) EVR for each person
27 across the 6.6-hr study period as a weighted average based on the observed EVR for the 5 hours
28 of exercise and the estimated EVR for 1.6 hours of rest/lunch. Descriptive statistics were
29 calculated and indicated the person-level EVR data were normally distributed, having a mean
30 value of 17.32 (L/min-m²) and a standard deviation of 1.25 (L/min-m²). To reflect variability
31 across simulated individuals, an EVR is probabilistically selected from this distribution once per
32 person and used for the duration of their simulation period. This new approach for assigning a
33 unique EVR to every simulated individual, one that accounts for rest and exercise periods and

³⁸ The six studies include Folinsbee et al. (1988), Folinsbee et al. (1994), Horstman et al. (1990), Kim et al. (2011), McDonnell et al. (1991), and Schelegle et al. (2009).

1 based on the distribution of ventilation rates achieved by all controlled human exposure study
2 subjects, more appropriately reflects the EVR variability expected to exist in the simulated
3 population compared to the approach used in the 2008 and 2015 reviews (e.g., U.S. EPA, 2007a;
4 U.S. EPA, 2007b; 2014 HREA) that assigned a single lower bound EVR value to all
5 individuals.³⁹

6 For practical and tractable modeling reasons, this individual-level EVR threshold is
7 applied to APEX simulated individuals using a 7-hr averaging time (representing the 6.6-hr
8 period rounded to whole numbers) in order to better represent the exposure study design than the
9 previously used 8-hr average. Then, once a simulated individual is identified as having surpassed
10 their personal 7-hr average EVR threshold in a given day, the level of their simultaneously
11 occurring 7-hr average O₃ exposure is recorded by APEX. Retained for each simulated
12 individual are the daily maximum 7-hr average exposure concentration(s) that occurred while at
13 moderate or greater exertion over the assessment period.

14 **3D.2.3 Ambient Air Concentrations**

15 Ambient air concentrations serve as a fundamental input used by APEX to estimate
16 exposure. There are two important attributes of ambient air concentrations to consider when
17 estimating population exposure and risk using APEX: spatial and temporal variability. This is
18 because there can be significant spatial and temporal heterogeneity in O₃ concentrations across
19 each of the study areas and there is substantial flexibility by APEX in handling ambient air
20 concentrations at varying scales, both temporally (e.g., hourly, daily) and spatially (e.g. 500-
21 meter grid, census tract).

22 For this exposure and risk analysis (as done for the 2015 review), we were interested in
23 having hourly O₃ concentrations at the census tract level. Having these temporally and spatially
24 resolved ambient air concentrations in each study area allows for better utilization of APEX
25 temporal and spatial capabilities in estimating exposure and risk (e.g., the population data
26 described in section 3D.2.2 are at a census tract level). Because APEX simulates where
27 individuals are located and what they are doing at specific times of the day, more realistic
28 exposure estimates are obtained in simulating the contact of individuals with these temporally
29 and spatially diverse concentrations.

³⁹ The EVR used in prior REAs (e.g., U.S. EPA, 2007b; U.S. EPA, 2007a; 2014 HREA) was based on a single lower bound EVR value of 13 L/min-m² selected from a range provided by Whitfield et al. (1996). For the current assessment approach, assigning randomly sampled values from an EVR distribution of $N\{17.32, 1.25\}$ still allows for some simulated individuals to be considered at elevated exertion when exceeding an EVR of ~13-14 L/min-m² (Appendix 3D, Attachment 2, Table 3) but overall, leads to fewer individuals achieving a moderate or greater exertion level when compared to simulations employing a single lower bound EVR value of 13 L/min-m².

1 Ambient air monitors for O₃ capture the temporal scale of interest (i.e., hourly) and can
2 provide general information regarding O₃ levels across an urban area. However, given their
3 limited spatial representativeness, i.e., tens of monitors extending across areas >10,000 km², the
4 monitors may not fully inform concentration variability that may exist at a finer spatial scale. In
5 addition, of interest are concentrations that represent a specific air quality scenario (e.g., ambient
6 air quality that just meets the current standard). In general, due to varying levels of precursor
7 emissions and meteorological conditions, most monitored 3-year periods do not have O₃
8 concentrations that just meet a specific air quality scenario of interest. Therefore, due to these
9 two realities, modeling methods are used to achieve the desired temporal and spatial scale along
10 with estimating ambient air O₃ concentrations that represent a specific air quality scenario.

11 The sections that follow briefly summarize the data and approaches used to estimate the
12 air quality concentrations used by APEX. A detailed description on the air quality data
13 collection, processing, adjustment, and evaluation is provided in Appendix 3C. First, section
14 3D.2.3.1 below provides information for the overall bounding of the modeling domains. The
15 identification of ambient air monitoring data used as a foundation for representing fine-scale
16 temporal and broad-scale spatial concentration variability is provided in section 3D.2.3.2. The
17 approach used to adjust concentrations to just meet air quality scenarios of interest is described
18 in section 3D.2.3.3. And finally, Section 3D.2.3.4 describes the technique used to interpolate the
19 concentrations from the monitor locations to the desired spatial scale (i.e., census tracts). It is
20 these estimated hourly census tract O₃ concentrations representing air quality scenarios that serve
21 as the basic ambient air concentrations from which each simulated individual's
22 microenvironmental concentrations and exposures are estimated (sections 3D.2.6 and 3D.2.7,
23 respectively). Multiple unique APEX input files are used for the current exposure and risk
24 analyses, one for each year and study area, and in the following two formats:

- 25 • *concsCSA[number]S[air quality scenario]Y[year].txt*: Tract IDs, hourly
26 concentrations (ppm), calendar date, by study area and year
- 27 • *districtsCSA[number]Y[year].txt*: Tract IDs, latitude, longitude, begin and end date

28 **3D.2.3.1 Spatial and Temporal Boundaries of Modeling Domains**

29 APEX has several options to select air quality data to use for estimating exposure and
30 risk. For this exposure and risk analysis, we used the list of counties that comprise each
31 CSA/MSA and their geographic boundaries to define the broad spatial characteristics of each
32 study area (0). As a result, simulated individuals residing within these counties would be part of
33 the exposure modeling domain and any ambient air concentrations estimated within these
34 counties would be used by APEX. Figure 3D-2 to Figure 3D-5 depict the spatial extent of the
35 exposure and risk modeling domain in each study area, along with a visualization of tract-level

1 population density and location of meteorological stations (see section 3D.2.4). The air radius for
2 APEX, a variable used to define the modeling domain, was set at 30 km to include all air quality
3 receptors (i.e., census tracts) within each county to model exposures and risks.

4 For each study area, three years of recent air quality were selected to estimate exposures.
5 The exposure periods are the O₃ seasons⁴⁰ for which routine hourly O₃ monitoring data were
6 available, and defined by 40 CFR part 58, Appendix D, Table D-3. These periods are designed to
7 reasonably capture variability in ambient air O₃ concentrations and meteorology and include the
8 high concentration events occurring in each area. Having this range of air quality data across
9 multiple years allows us to realistically estimate a range of exposures, rather than using a single
10 year of air quality. The number of O₃ monitors in operation did not vary from year to year, thus,
11 the overall spatial representation of each study area by the ambient air monitors (and that using
12 the statistically interpolated data) remained constant for each year over the simulation period.

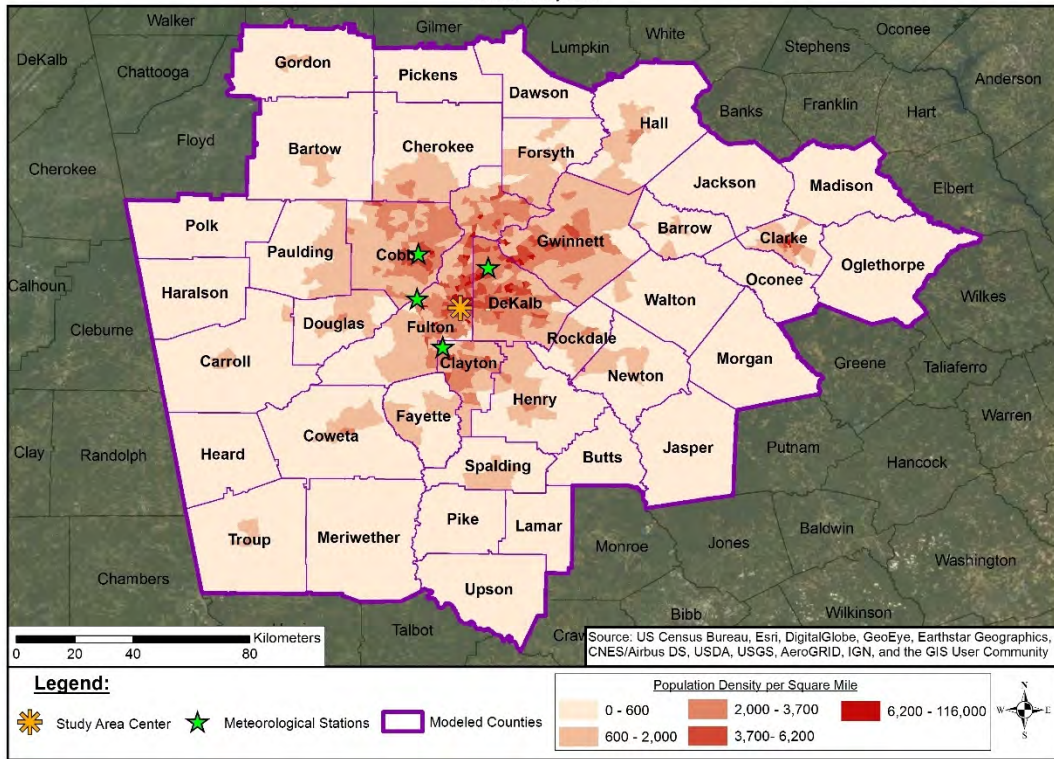
13

⁴⁰ In this current analysis and for practical purposes, even though there are different durations of monitoring data available across the study areas (i.e., some areas perform a full year of monitoring, others less than a full year), an O₃ season is considered to be synonymous with a year and exposure results are reported on a per year basis.

1 **Table 3D-5. List of states, counties, and O₃ seasons that define the air quality and**
 2 **exposure spatial and temporal modeling domain in each study area.**

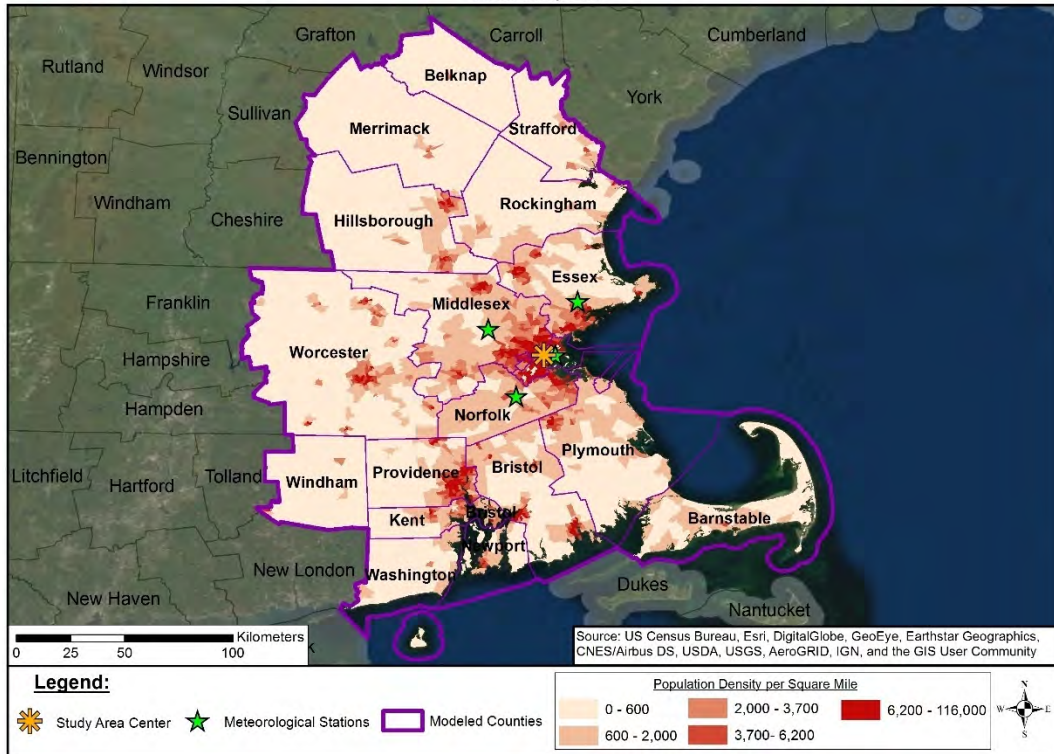
Study Area	State Abbreviation: County List ^A	O ₃ season ^B
Atlanta	GA: Barrow, Bartow, Butts, Carroll, Cherokee, Clarke, Clayton, Cobb, Coweta, Dawson, DeKalb, Douglas, Fayette, Forsyth, Fulton, Gordon, Gwinnett, Hall, Haralson, Heard, Henry, Jackson, Jasper, Lamar, Madison, Meriwether, Morgan, Newton, Oconee, Oglethorpe, Paulding, Pickens, Pike, Polk, Rockdale, Spalding, Troup, Upson, Walton.	March to October
Boston	CT: Windham. MA: Barnstable, Bristol, Essex, Middlesex, Norfolk, Plymouth, Suffolk, Worcester. NH: Belknap, Hillsborough, Merrimack, Rockingham, Strafford. RI: Bristol, Kent, Newport, Providence, Washington.	March to September
Dallas	TX: Bryan, Collin, Cooke, Dallas, Denton, Ellis, Fannin, Grayson, Henderson, Hood, Hopkins, Hunt, Johnson, Kaufman, Navarro, Palo Pinto, Parker, Rockwall, Somervell, Tarrant, Wise.	January to December
Detroit	MI: Genesee, Lapeer, Lenawee, Livingston, Macomb, Monroe, Oakland, St. Clair, Washtenaw, Wayne.	March to October
Philadelphia	DE: Kent, New Castle. MD: Cecil. NJ: Atlantic, Burlington, Camden, Cape May, Cumberland, Gloucester, Salem PA: Berks, Bucks, Chester, Delaware, Montgomery, Philadelphia.	March to October
Phoenix	AZ: Maricopa, Pinal.	January to December
Sacramento	CA: El Dorado, Nevada, Placer, Sacramento, Sutter, Yolo, Yuba.	January to December
St. Louis	IL: Bond, Calhoun, Clinton, Jersey, Macoupin, Madison, Marion, Monroe, St. Clair, MO: Franklin, Jefferson, Lincoln, St. Charles, St. Francois, St. Louis, Warren, St. Louis City.	March to October
^A Delineations promulgated by the Office of Management and Budget (OMB) in February of 2013 (see Appendix 3C, section 3C.2). ^B These are the regulatorily required monitoring seasons (see section 2.3.1 of the main document).		

Population Density - Year 2010
Atlanta Study Area



1

Population Density - Year 2010
Boston Study Area



2

3 **Figure 3D-2. County boundaries, census tract population densities, and meteorological**
4 **stations in the Atlanta (top) and Boston (bottom) study areas.**

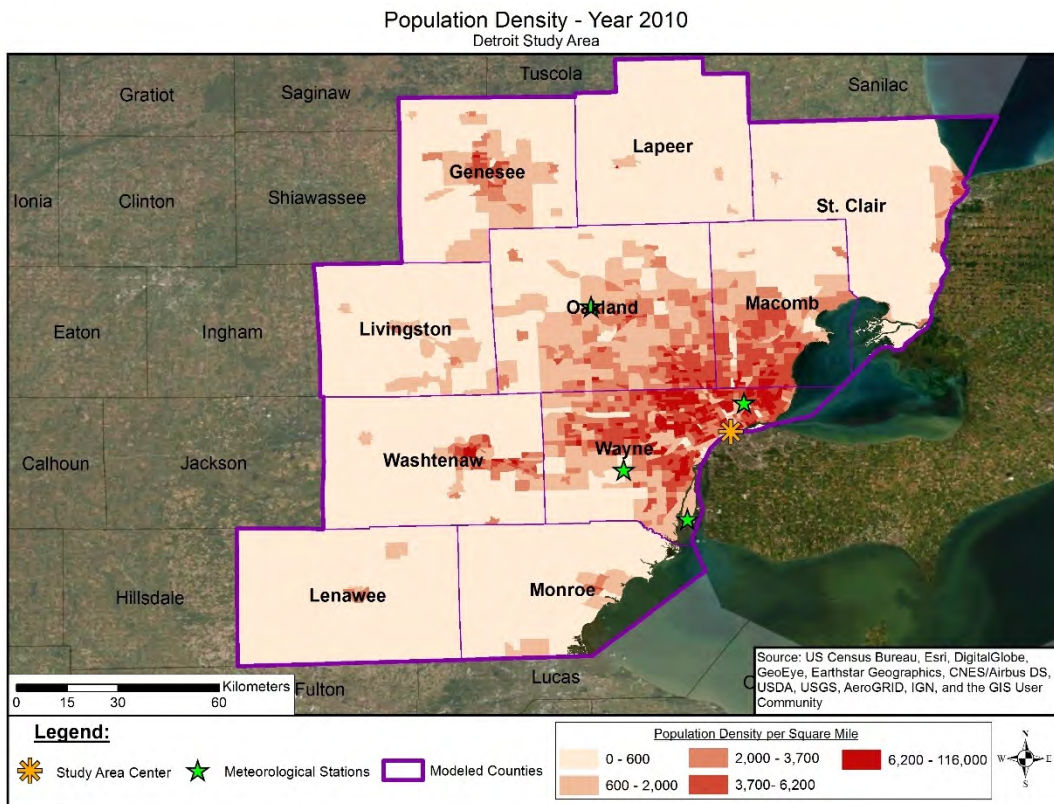
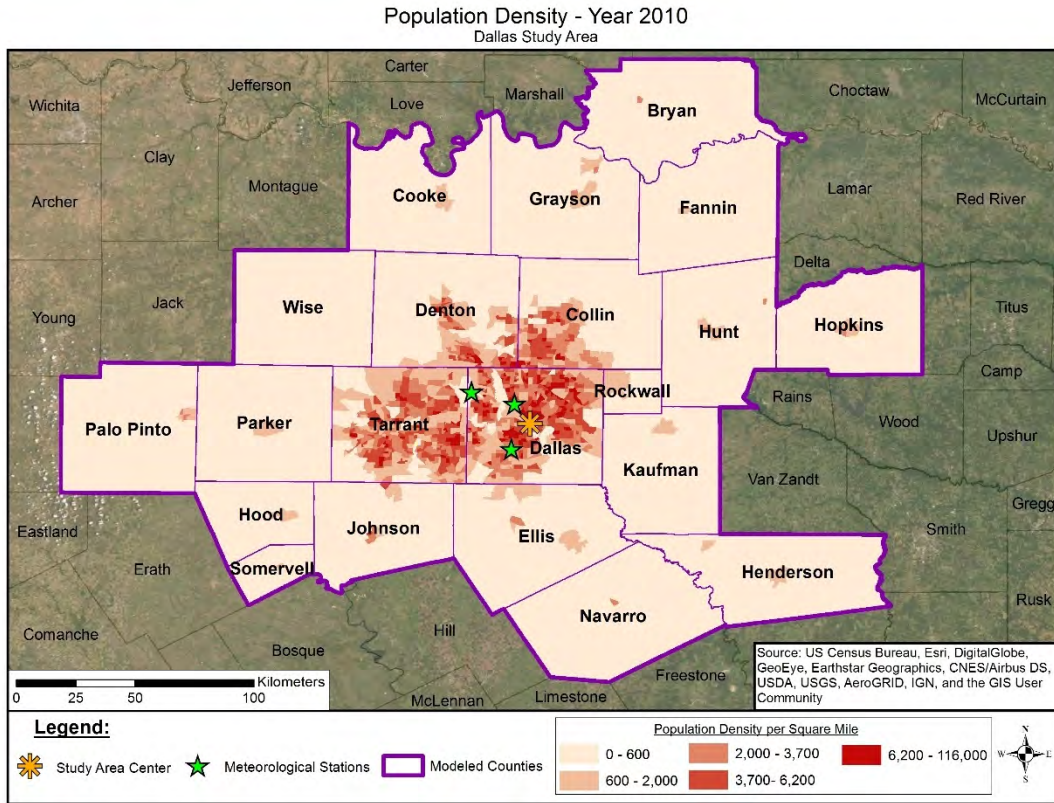


Figure 3D-3. County boundaries, census tract population densities, and meteorological stations in the Dallas (top) and Detroit (bottom) study areas.

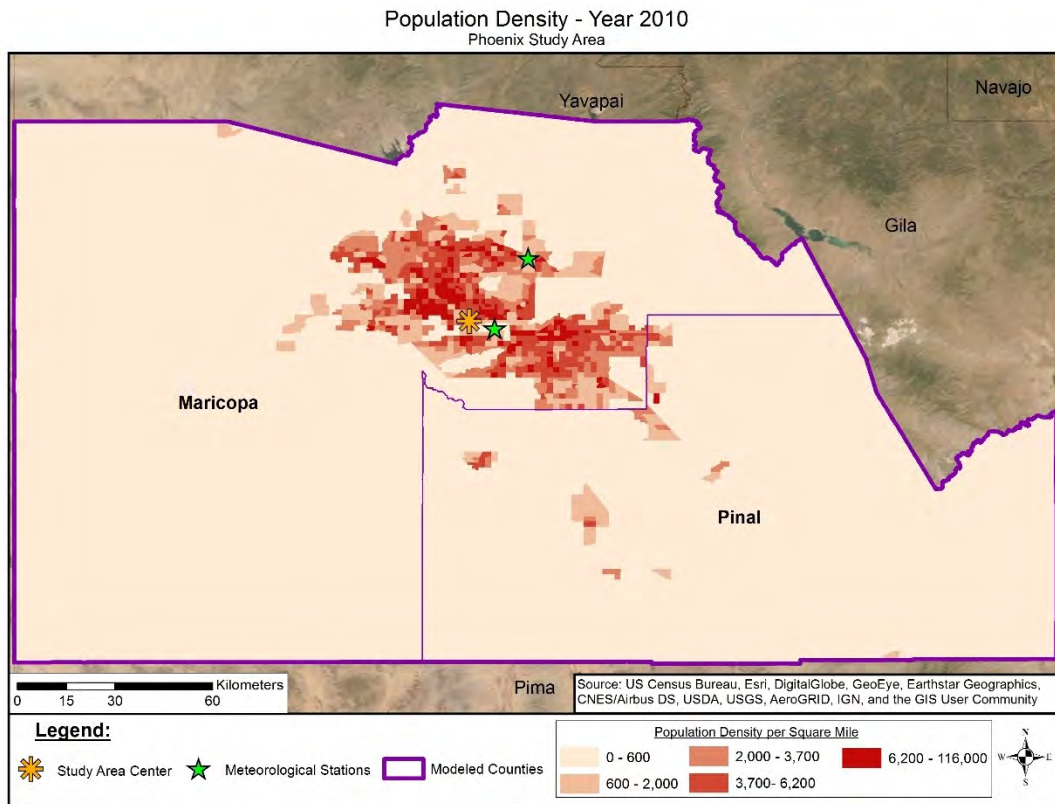
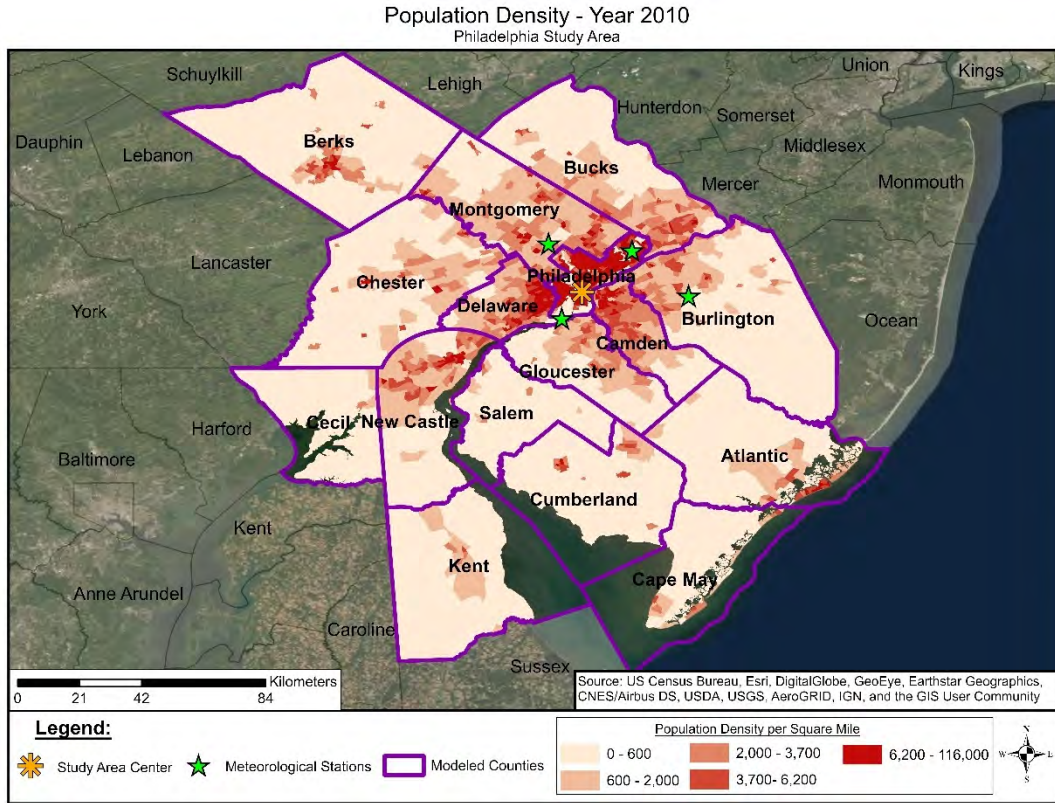


Figure 3D-4. County boundaries, census tract population densities, and meteorological stations in the Philadelphia (top) and Phoenix (bottom) study areas.

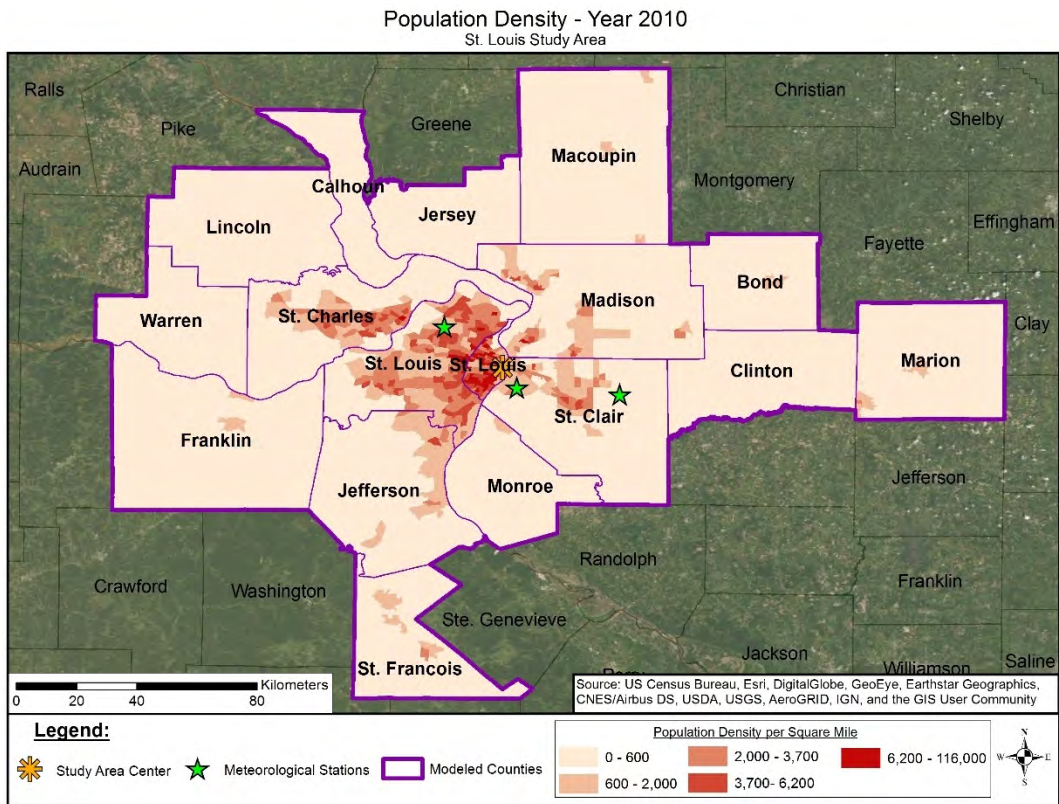
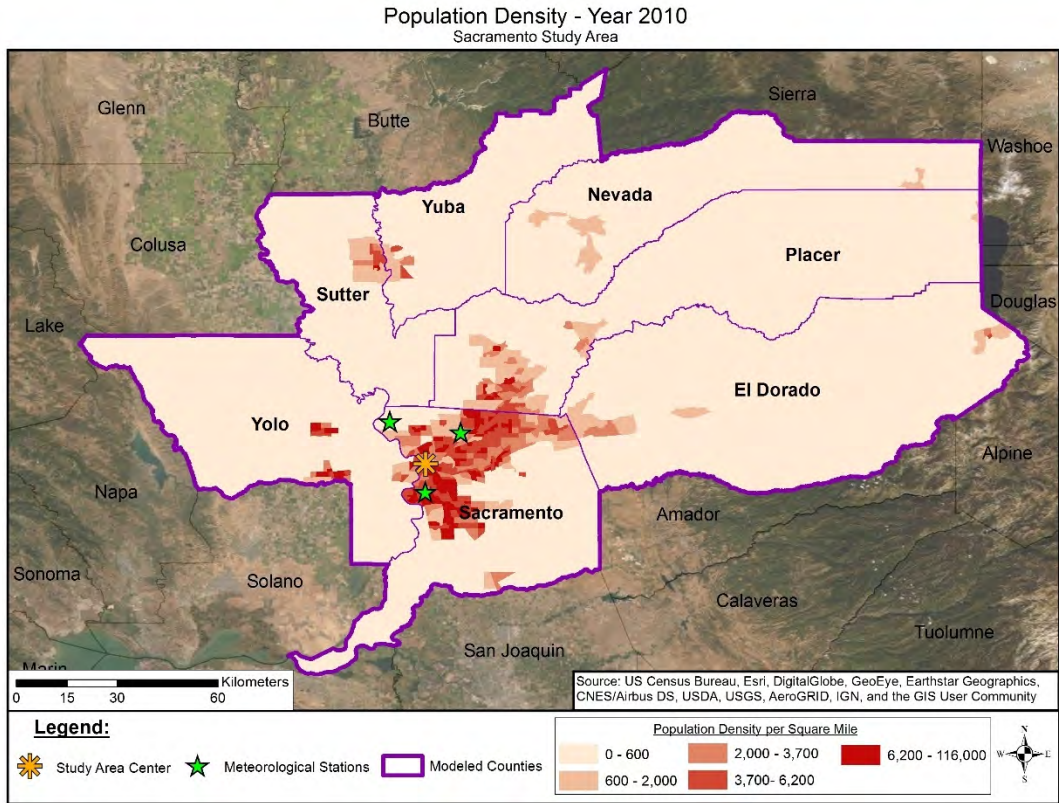


Figure 3D-5. County boundaries, census tract population densities, and meteorological stations in the Sacramento (top) and St. Louis (bottom) study areas.

1 **3D.2.3.2 Ambient Air Monitoring Data**

2 We used hourly O₃ concentrations from ambient air monitors in each study area for the
3 2015-2017 period to develop the air quality surface used for estimating exposure and risk (Table
4 3D-6; details in Appendix 3C, section 3C.3).⁴¹ Design values for monitors in each study area
5 were used to determine the direction and magnitude of adjustments needed to just meet the
6 current standard and the other two air quality scenarios (section 3D.2.3.3). The two other air
7 quality scenarios are O₃ concentrations for which the highest design value in the area is just
8 above or just below the current standard level: 75 ppb and 65 ppb. Ambient air monitors outside
9 each study area, but within 50 km, were also used to improve spatial interpolation of air quality
10 near the edges of the study areas (section 3D.2.3.4). All available ambient air O₃ monitor data
11 were used to develop the adjusted air quality surfaces, however design values were not
12 calculated for monitors having incomplete data.
13

⁴¹ Briefly, hourly O₃ concentration data for all U.S. monitoring sites for 2015-2017 were retrieved from the EPA’s Air Quality System (AQS) database. Monitors within the CSA boundary for each urban study area were identified and used to determine the NO_x emissions changes necessary to meet the air quality scenarios of interest (section 3D.2.3.3). Monitors within 50 km of the CSA boundary were identified to provide additional data for spatial interpolation (section 3D.2.3.4).

1 **Table 3D-6. List of ambient air monitor IDs, range of O₃ design values, and number of**
 2 **monitors in each study area.**

Study Area	State: Ambient Air Monitor IDs ^A	O ₃ Design Values (ppb) (# of monitors)
Atlanta	GA: 130590002, 130670003, 130770002, 130850001, 130890002, 130970004, 131210055 , 131350002, 131510002, <i>132230003</i> , 132319991, 132470001	63 – 75 (12)
Boston	CT: 090159991 MA: <i>250010002</i> , 250051004 , 250051006, 250092006, 250094005, 250095005, 250170009, 250213003, 250230005, 250250042, 250270015, 250270024 NH: 330012004, 330111011, 330115001, 330131007, 330150014, 330150016, 330150018 RI: 440030002, 440071010, 440090007	59 – 73 (23)
Dallas	OK: <i>400130380</i> TX: 480850005, 481130069, 481130075, 481130087, 481210034 , 481211032, 481390016, 481391044, 482210001, 482311006, 482510003, 482570005, 483491051, 483670081, 483970001, 484390075, 484391002, 484392003, 484393009, 484393011	61 – 79 (21)
Detroit	MI: 260490021, 260492001, 260910007, 260990009, 260991003, 261250001, 261470005, 261610008, 261619991, 261630001, 261630019 , <i>261630093</i> , <i>261630094</i>	66 – 73 (13)
Philadelphia	DE: 100010002, 100031007, 100031010, 100031013, 100032004 MD: 240150003 NJ: 340010006, 340070002, 340071001, 340110007, 340150002 PA: 420110006, 420110011, <i>421070004</i> , 420170012 , 420290100, 420450002, 420910013, 421010024 , 421010048	64 – 80 (20)
Phoenix	AZ: 040130019, 040131003, 040131004, 040131010, 040132001, 040132005, 040133002, 040133003, 040134003, 040134004, <i>040134005</i> , 040134008, 040134010, 040134011, <i>040135100</i> , 040137003, 040137020, 040137021, 040137022, 040137024, 040139508, 040139702, 040139704, 040139706, 040139997 , 040213001, 040213003, 040213007, 040217001, 040218001	63 – 76 (30)
Sacramento	CA: 060170010, <i>060170012</i> , 060170020, 060570005 , <i>060570007</i> , 060610003, 060610004, 060610006, 060611004, 060612002, 060670002, 060670006, 060670010, 060670011, 060670012, <i>060670014</i> , 060675003, 061010003, <i>061010004</i> , 061130004, 061131003	63 – 86 (21)
St. Louis	IL: <i>170830117</i> , <i>170831001</i> , 171170002, 171190008, 171191009, 171193007, 171199991, 171630010 MO: 290990019, <i>291130003</i> , <i>291130004</i> , 291831002 , 291831004, 291890005, 291890014, 295100085	65 – 72 (16)

^A **Bold font** indicates monitor(s) design value used to adjust ambient air concentrations to just meet selected air quality scenarios. From Appendix 3C, Tables 3C-20 to 3C-27. *Italic font* indicates monitor did not meet completeness criteria to calculate a design value.

3
 4 **3D.2.3.3 Model Adjusted Concentrations at Monitor Locations to Represent Air**
 5 **Quality Scenarios**

6 Details of the approach used to develop the three air quality scenarios (design values of
 7 70, 65 and 75 ppb) are provided in Appendix 3C, sections 3C.4 and 3C.5. Briefly, the ambient
 8 air concentrations described above in section 3D.2.3.2 were adjusted to just meet the current
 9 standard (70 ppb, annual 4th highest daily maximum 8-hr average concentration, averaged over a

1 3-year period) and two other air quality scenarios (75 and 65 ppb, annual 4th highest daily
2 maximum 8-hr average concentration, averaged over a 3-year period)⁴² using a model-based O₃
3 methodology that adjusts the observed hourly O₃ concentrations to reflect the expected spatially
4 and temporally varying impacts of changes in NO_x emissions. The methodology is similar to that
5 used for the 2014 HREA and employs a photochemical air quality model combined with a tool
6 that calculates modeled sensitivities of O₃ to precursor emission changes.

7 For the current analysis, the Comprehensive Air Quality Model with Extensions
8 (CAMx)⁴³ served as the chemical transport model,⁴⁴ with 2016 selected as the base year for
9 determining the adjustments needed for the 2015-2017 ambient air monitoring data. Model
10 inputs include meteorological data,⁴⁵ emissions,⁴⁶ and initial and boundary conditions.⁴⁷ The
11 evaluation of modeled versus observed O₃ concentrations for 2016 indicated CAMx generally
12 reproduced the observed spatial and temporal patterns, with the exception of concentration
13 underestimates occurring in winter across almost all regions (Appendix 3C, section 3C.4.2).

14 The CAMx model was instrumented with the Higher order Decoupled Direct Method
15 (HDDM) to calculate modeled nonlinear sensitivities of O₃ to emission changes (Appendix 3C,
16 section 3C.5). The photochemical modeling outputs included both modeled O₃ concentrations
17 and sensitivities of O₃ concentrations to changes in NO_x emissions for each hour in a single year
18 at all ambient air monitor locations (Appendix 3C, sections 3C.4 and 3C.5). Linear regression
19 was used with these single-year 2106 model outputs to create relationships between the
20 sensitivities and O₃ concentrations for each hour of each of the four seasons at each monitoring
21 location. The relationships between hourly sensitivities and hourly O₃ for each season were then
22 used with three years of ambient air monitoring data at each location to predict hourly
23 sensitivities for the complete 3-year record at each monitoring location. From these, we

⁴² In these scenarios, the air quality conditions were adjusted such that the monitor location with the highest concentrations in each area had a design value just equal to either 75 ppb or 65 ppb.

⁴³ The Comprehensive Air Quality Model with Extensions and associated documentation is found at www.camx.com.

⁴⁴ The 2014 HREA used the Community Multiscale Air Quality Modeling System (CMAQ) to model air quality.

⁴⁵ Horizontal wind components (i.e., speed and direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each 12 Km grid cell in each vertical layer was derived from version 3.8 of the Weather Research and Forecasting Model (WRF; <http://wrf-model.org>). For details, see PA, Appendix 3C, section 3C.4.1.4.

⁴⁶ Emissions from electric generating units, other point sources, area sources, agricultural sources (ammonia only), anthropogenic fugitive dust sources, nonroad mobile sources, onroad mobile sources, and biogenic sources are based on the alpha version of the Inventory Collaborative 2016 emissions modeling platform (<http://views.cira.colostate.edu/wiki/wiki/9169>). For details, see PA, Appendix 3C, section 3C.4.1.5.

⁴⁷ Initial and lateral boundary concentrations for the 12 km domain are provided by the hemispheric version of the Community Multi-scale Air Quality model (H-CMAQ) v5.2.1. The H-CMAQ model was run for 2016 with a horizontal grid resolution of 108 km and 44 vertical layers up to 50 hPa. For details, see PA, Appendix 3C, section 3C.4.1.6.

1 calculated hourly O₃ concentrations at each monitor location based on iteratively increasing NO_x
 2 reductions to determine the adjustments necessary for the monitor location with the highest
 3 design value in each study area to just meet the target value, e.g., 70 ppb for the current standard
 4 scenario (Appendix 3C, section 3C.5). For the 75 ppb air quality scenario, we note that three
 5 areas required an increase in NO_x emissions as their highest O₃ design values were below 75
 6 ppb. For the other five study areas and that same air quality scenario and for all study areas with
 7 the other two air quality scenarios (i.e., 65 and 70 ppb), emission reductions were required
 8 (Table 3D-7).

9 **Table 3D-7. Range of the percent NO_x emission changes needed to adjust air quality in the**
 10 **eight study areas for the three air quality scenarios.**

Design Value for each Air Quality Scenario	Range of NO _x Emission Changes Applied Across the Eight Study Areas
75 ppb	+18% to -45%
70 ppb	-13% to -58%
65 ppb	-38% to -72%
From Appendix 3C, Table 3C-19.	

11

12 **3D.2.3.4 Interpolation of Adjusted Monitor Concentrations to the Census Tracts**
 13 **Comprising Each Study Area**

14 As described above, model-based relationships between O₃ and NO_x emissions were
 15 used to adjust hourly O₃ concentrations at the ambient air monitor locations (section 3D.2.3.2) to
 16 represent conditions in which the study area just meets the selected air quality scenario (section
 17 3D.2.3.3). Simulated O₃ concentrations were then needed at a finer spatial scale than that given
 18 by the monitor sites to better represent the spatial heterogeneity in O₃ concentrations across
 19 locations frequented by the simulated population (and during the times frequented) across the
 20 study area. To accomplish this in each of the eight study areas, the adjusted hourly O₃
 21 concentrations at monitoring sites were interpolated to census tract centroids using the Voronoi
 22 Neighbor Averaging (VNA; Appendix 3C, section 3C.6). Nearby monitoring concentrations, for
 23 each hour, inform the estimation of O₃ for a given census tract using inverse distance weighting.
 24 In so doing, both spatial and temporal gaps in the desired air quality surface are filled
 25 simultaneously, resulting in a final dataset of ambient air O₃ concentration estimates with high
 26 temporal and spatial resolution (hourly concentrations in 500 to 1700 census tracts) for each of
 27 the eight study areas and for years 2015 to 2017 (Appendix 3C, section 3C.7).

3D.2.3.5 Evaluation of Temporal and Spatial Characteristics of the Simulated Air Quality Surfaces

We applied the above described approaches to simulate air quality surfaces that represent fine-scale temporal (i.e., hourly) and spatial (i.e., census tract) variability in O₃ concentrations for the three air quality scenarios in each study area. Then, characteristics of the simulated air quality surfaces were evaluated for trends and patterns that would be informative for interpreting the simulated exposure and risk results. For example, Figure 3D-6 illustrates the temporal variability across the three years of monitoring data, stratified by hour-of-day (left panel) and month (right panel), in Philadelphia for the ambient air measurements, and for the three simulated air quality scenarios (following the model-based adjustment at each monitor location).

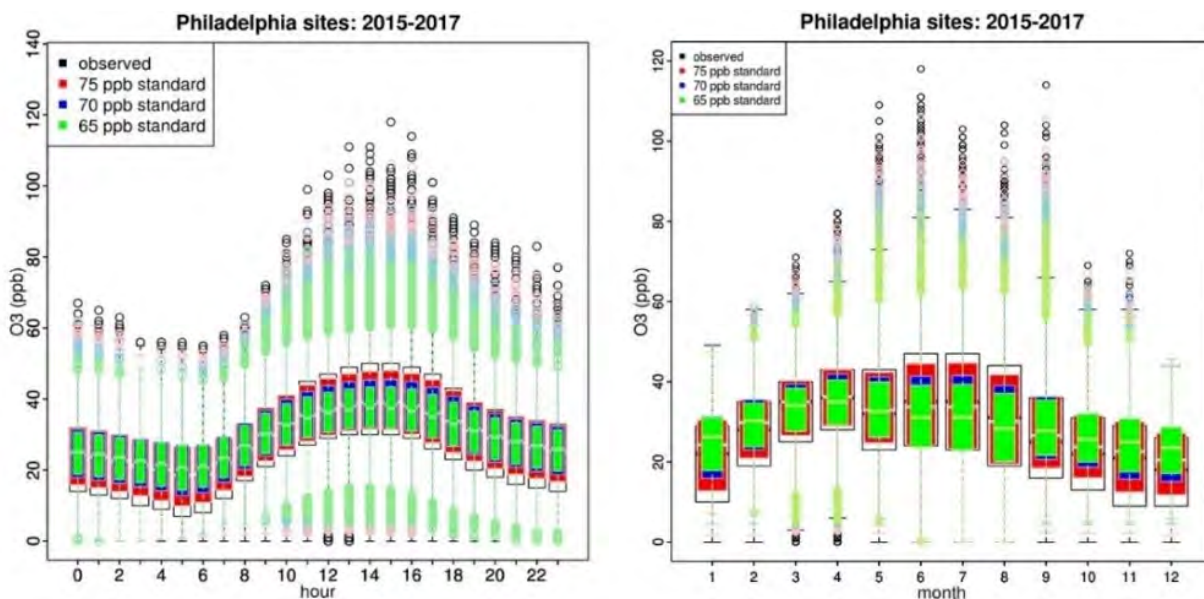


Figure 3D-6. Hourly O₃ distributions by hour-of-day (left panel) and month (right panel) at ambient air monitoring sites in Philadelphia for observed air quality (black), air quality adjusted to meet the current standard (70 ppb, blue) and two other design values (75 ppb, red; and 65 ppb, green). From Appendix 3C, Figures 3C-71 and 3C-79, respectively.

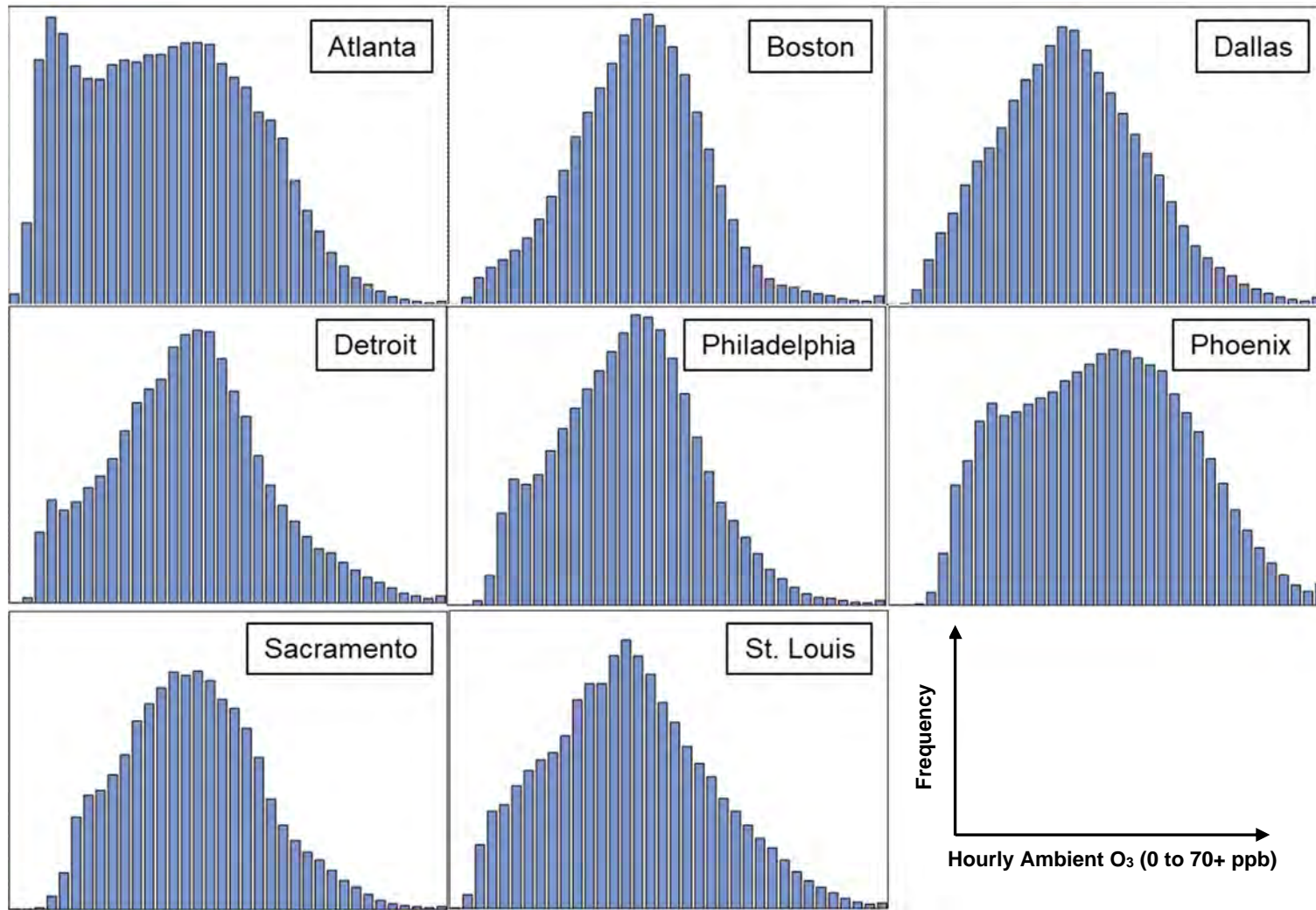
The diurnal and seasonal temporal patterns for the three air quality scenarios are similar to the monitor observations, with highest O₃ concentrations during the during late morning/afternoon hours and during spring/summer months. In addition, the upper end of the O₃ concentration distributions decrease from observed values (black) to values adjusted to meet the current standard of 70 ppb (blue) and decrease further when adjusted to meet a design value of 65 ppb (green). These decreases can be seen when evaluating the highest O₃ hours-of-the day and represented by the data points that extend beyond the whiskers of the boxplots. Further, the

1 overall pattern flattens when decreasing the level of the O₃ standard, considering both the diurnal
2 and monthly distributions. Regarding the diurnal pattern, O₃ increases during early morning
3 hours are associated with VOC-limited and NO_x titration conditions near NO_x sources during
4 rush-hour periods. Lower O₃ concentrations in the winter months result from lower solar
5 insolation rates and a reduction in total photochemical activity. See the accompanying Appendix
6 3C (Section 3C.7.2 and Figures 3C-67 through 3C-82) for details for temporal characteristics of
7 all eight study areas.

8 We also evaluated the hourly O₃ concentrations by considering the overall shape of the
9 concentration distribution using the census-tract resolution interpolated data. Even though both
10 the temporal and spatial attributes may be conflated in such a presentation, a histogram can be
11 useful in illustrating important features of the distribution (e.g., skewness, kurtosis, upper
12 percentile tails) that may be influential in estimated exposures and risks. For example, Figure
13 3D-7 illustrates the overall shape⁴⁸ of the hourly concentration distribution in each of the eight
14 study areas for the air quality scenario just meeting the current standard. The distribution for all
15 study areas are skewed to the right, generally representing a lognormal form.

16 There are notable differences across the collection of study areas. For example, the
17 distributions for Boston, Dallas, Philadelphia, and Sacramento are slender (i.e., leptokurtic),
18 showing much higher peaks around the mean value, relative to the other four study areas,
19 Atlanta, Detroit, Phoenix, and St. Louis which exhibit relatively flatter (i.e., platykurtic)
20 distributions, and the latter three of which, show an increased frequency of upper percentile
21 concentrations. Phoenix, in particular, exhibits the greatest right-most shift in the hourly O₃
22 concentration distribution and would reflect other areas of the U.S. having a similar distribution
23 of ambient air O₃ concentrations. Also, there are only limited instances of hourly O₃
24 concentrations >70 ppb in all study areas for the air quality scenario just meeting the current
25 standard (Figure 3D-7). This is consistent with recent (unadjusted) ambient air monitoring data,
26 whereas hourly O₃ concentrations are rarely at or above 100 ppb when design values are ≤70 ppb
27 (i.e., <0.02% frequency; see Appendix 2A, Table 2A-4). This is important to note because these
28 distinct features of the O₃ concentration distribution, along with the spatial and temporal
29 intersection of concentrations with population demographics and activity patterns, play an
30 important role in contributing to variation in the estimated population exposures and risks
31 presented in section 3D.3 below.

⁴⁸ Figure 3D-7 is intended to illustrate the differences in the shape of the distributions. All histograms have the exact same range of values for the x-axis, i.e., the midpoint concentrations range from 0 to 70 ppb, in 2 ppb increments (maximum value represents frequency of all hourly concentrations >70 ppb. Because there are varied distribution shapes, the range of values for the y-axis differ across the study areas. The actual value of the y-axis is unimportant in this context because of interest here are the relative differences that exist across the concentration distributions (e.g., frequency of high O₃ concentrations relative to the occurrence of low O₃ concentrations).



1

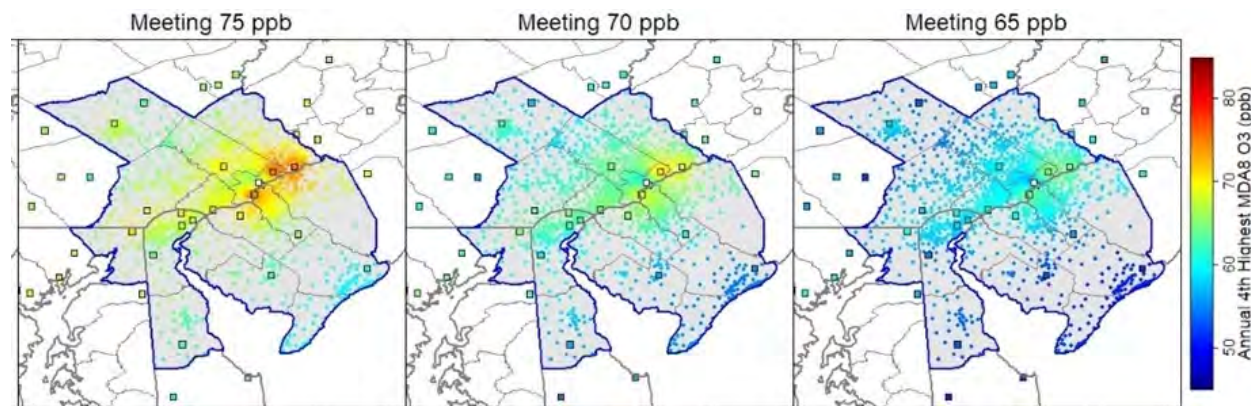
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4

Figure 3D-7. Histograms of hourly O₃ concentrations (ppb, x-axis) for the air quality scenario just meeting the current O₃ standard in the eight study areas. The x-axis midpoint concentrations range from 0 to 70 ppb, in 2 ppb increments (rightmost, maximum histogram bar for all study areas represents the frequency of all hourly concentrations >70 ppb).

1 Regarding spatial variability, Figure 3D-8 displays census tract design values for each of
2 the three air quality scenarios in Philadelphia. A decline in the highest ambient air O₃
3 concentrations is predicted across the study area when considering air quality scenarios at lower
4 design values.



6
7 **Figure 3D-8. Calculated design values for census tracts in the Philadelphia study area,**
8 **derived from a VNA interpolation of CAM_x/HDDM adjusted O₃**
9 **concentrations. Figure modified from Appendix 3C, Figure 3C-99.**

10 3D.2.4 Meteorological Data

11 Temperature data are used by APEX in selecting human activity data and in estimating
12 air exchange rates (AERs) for indoor residential microenvironments (MEs). When developing
13 profiles, APEX uses temperature data from the closest weather station to each Census tract.
14 Hourly surface temperature measurements were obtained from the National Oceanic and
15 Atmospheric Administration (NOAA) Integrated Surface Hourly (ISH) data files.⁴⁹ The weather
16 stations used for each study area are given in Table 3D-8, along with general locations provided
17 in Figure 3D-2 to Figure 3D-5.

18 In general, the occurrence of missing temperature data was limited to a few hours per
19 year. Missing hourly temperature data were estimated by the following procedure. Where there
20 were consecutive strings of missing values (data gaps) of 9 or fewer hours, missing values were
21 estimated by linear interpolation between the observed values at the ends of the gap. Remaining
22 missing values at a meteorological station were estimated by fitting linear regression models for
23 each hour of the day, with each of the other monitors, and choosing the model which maximizes
24 R², for each hour of the day, subject to the constraints that R² be greater than 0.40 and the
25 number of regression data values (days) is at least 100. If there no suitable regression models to
26 fill the missing values, for gaps of 12 or fewer hours, missing values were estimated by linear

⁴⁹ See: <ftp://ftp.ncdc.noaa.gov/pub/data/noaa/isd-lite/>

1 interpolation between the valid values at the ends of the gap. Any remaining missing values were
 2 replaced with the value at the closest station for that hour. Because there were limited instances
 3 of missing data, there were negligible differences between the statistically filled and the original
 4 temperature data with missing values.

5 **Table 3D-8. Study area meteorological stations, locations, and hours of missing data.**

Study Area	Station Name	WBAN ^A	Latitude	Longitude	Number of hours with missing temperature		
					2015	2016	2017
Atlanta	HARTSFIELD-JACKSON ATLANTA	13874	33.630	-84.442	6	4	5
	FULTON CO-BROWN FLD ARPT	03888	33.779	-84.521	34	84	220
	DEKALB-PEACHTREE AIRPORT	53863	33.875	-84.302	13	6	47
	DOBBINS AIR RESERVE BASE	13864	33.917	-84.517	171	142	58
Boston	LAURENCE G HANSCOM FLD	14702	42.470	-71.289	55	164	19
	BEVERLY MUNICIPAL AIRPORT	54733	42.584	-70.918	56	8	7
	GEN E L LOGAN INTERNATIONAL	14739	42.361	-71.010	5	4	5
	NORWOOD MEMORIAL AIRPORT	54704	42.191	-71.174	17	38	17
Dallas	DALLAS LOVE FIELD AIRPORT	13960	32.852	-96.856	5	5	5
	DALLAS/FT WORTH INTERNAT	03927	32.898	-97.019	5	5	5
	DALLAS EXECUTIVE AIRPORT	03971	32.681	-96.868	27	14	36
Detroit	DETROIT METRO WAYNE COUNTY	94847	42.231	-83.331	462	547	619
	GROSSE ILE MUNICIPAL AIRPORT	54819	42.099	-83.161	484	397	44
	DETROIT CITY AIRPORT	14822	42.409	-83.010	25	22	69
	OAKLAND CO. INTNL AIRPORT	94817	42.665	-83.418	16	11	17
Philadelphia	WINGS FIELD AIRPORT	64752	40.100	-75.267	150	241	324
	SOUTH JERSEY REGIONAL ARPT	93780	39.941	-74.841	<i>na</i>	90	69
	PHILADELPHIA INTERNATIONAL	13739	39.873	-75.227	5	6	5
	NE PHILADELPHIA AIRPORT	94732	40.079	-75.013	28	13	51
Phoenix	PHOENIX SKY HARBOR INTL	23183	33.428	-112.004	13	8	6
	SCOTTSDALE AIRPORT	03192	33.623	-111.911	9	19	10
Sacramento	SACRAMENTO EXECUTIVE	23232	38.507	-121.495	10	21	87
	SACRAMENTO MCCLELLAN AFB	23208	38.667	-121.400	366	368	89
	SACRAMENTO INTL AIRPORT	93225	38.696	-121.590	28	53	41
St. Louis	SCOTT AIR FORCE BASE/MIDAMER	13802	38.550	-89.850	110	49	45
	LAMBERT-ST LOUIS INTERNAT	13994	38.753	-90.374	11	7	7
	ST LOUIS DOWNTOWN AIRPORT	03960	38.571	-90.157	12	49	7

^A Weather Bureau Army Navy (WBAN) number of the meteorological stations.
 "na" is no data available

6

1 Multiple unique APEX input files are used for the current exposure and risk analyses, one
2 for each year and study area, and in the following two formats:

- 3 • *METdataCSA[number]Y[year].txt*: meteorological station IDs, hour of day, hourly
4 temperature (°F) for each meteorological station, by study area and year
- 5 • *METlocsCSA[number]Y[year].txt*: meteorological station IDs, latitudes and
6 longitudes, start and stop dates of temperature data

7 **3D.2.5 Construction of Human Activity Pattern Sequences**

8 Exposure models use human activity pattern data to estimate exposure to pollutants.
9 Different human activities, such as outdoor exercise, indoor reading, or driving a motor vehicle
10 can lead to different pollutant exposures, intakes and doses. This may be due to differences in the
11 pollutant concentration in the varied locations where different activities are performed as well as
12 to differences in the energy expended in performing the activities (because energy expended
13 influences inhalation and thus may influence pollutant intake). To model exposures to ambient
14 air pollutants, it is critical to have information on the locations where people spend time and the
15 activities performed in such locations. The following subsections describe the activity pattern
16 data, population commuting data, and the approaches used to simulate where individuals might
17 be and what they might be doing.

18 After the basic demographic variables are identified by APEX for a simulated individual
19 in the study area, values for the other variables are selected as well as the development of the
20 activity patterns that account for the places the simulated individual visits and the activities they
21 perform. The following subsections describe the population data we used in the assessment to
22 assign key features of the simulated individuals, and approaches used to simulate the basic
23 physiological functions important to the exposure estimates for this exposure and risk analysis.

24 **3D.2.5.1 Consolidated Human Activity Database**

25 The Consolidated Human Activity Database (CHAD) provides time series data on human
26 activities through a database system of collected human diaries, or daily time location activity
27 logs (U.S. EPA, 2019c). The purpose of CHAD is to provide a basis for conducting multi-route,
28 multi-media exposure assessments (McCurdy, 2000). The data contained within CHAD come
29 from multiple surveys with variable, study-specific structure (e.g., real time minute-by-minute
30 recording of diary events versus a recall method using time-block-averaging). Common to all of
31 the peer-reviewed studies, individuals provided information on their locations visited and
32 activities performed for each surveyed day. Personal attribute data for the surveyed individuals,
33 such as age and sex, are included in CHAD and are used as variables to link to the population
34 data. The latest version of CHAD contains data for nearly 180,000 individual diary days. Most of
35 the CHAD data are from studies conducted since 2000, several of which are newly included or

1 updated since the 2014 HREA.⁵⁰ Table 3D-9 provides the survey study information including the
 2 geographic coverage, year, and the number of diaries available for use by APEX.⁵¹

3
 4 **Table 3D-9. Overview of Studies Included in the APEX Activity Data Files.**

Study Name (abbreviation)	Geographic Coverage	Study Year	Number of Diary Days ^A		Age Range		Reference
			Ages 5-18	Any Age	min	max	
American Time Use Survey, Bureau of Labor Statistics (BLS)	Entire US	2003-11	7,559	123,932	15	85	US Bureau of Labor Statistics (2014)
Baltimore Retirement Home Study (BAL)	Baltimore County, MD	1997-98	0	390	72	93	Williams et al. (2000)
California Activity Pattern Studies (CAA, CAC, CAY)	California	CAA: 1987-88	36	1,570	18	94	Wiley et al. (1991a), Wiley et al. (1991b)
		CAC: 1989-90	680	1,197	0	11	
		CAY: 1987-88	182	182	12	17	
Cincinnati Activity Patterns Study (CIN)	Cincinnati, OH	1985	736	2,595	0	86	Johnson (1989)
Detroit Exposure and Aerosol Research Study (DEA)	Detroit, MI	2004-2007	5	336	18	74	Williams et al. (2009)
Denver, Colorado Personal Exposure Study (DEN)	Denver, CO	1982-1983	7	784	18	70	Johnson (1984); Johnson et al. (1986)
EPA Longitudinal Studies (EPA)	Central NC	1999-2000, 2002, 2006-08, 2012-2013	0	1,780	0	72	Isaacs et al. (2013)
Los Angeles Ozone Exposure Study: Elementary School/High School (LAE, LAH)	Los Angeles, CA	1989-1990	49	49	10	12	Roth Associates (1988); Spier et al. (1992)
			43	43	13	17	
National Human Activity Pattern Study (NHAPS): Air/Water (NHA, NHW)	48 states	1992-1994	659	4,723	0	93	Klepeis et al. (1995); Tsang and Klepeis (1996)
			713	4,663	0	93	

⁵⁰ CHAD updates since the 2014 HREA include expansion of activity codes, revision to the METs distributions, filling missing temperatures, characterizing ambiguous location entries, etc. See U.S. EPA, 2019c and Attachment 3.

⁵¹ Following stated updates to improve the CHAD diary information, some diaries in the CHAD master database remain unusable for exposure and risk modeling. Most commonly this is from having excessive missing or unknown location or activity data (e.g., ≥3 hours/day).

Study Name (abbreviation)	Geographic Coverage	Study Year	Number of Diary Days ^A		Age Range		Reference
			Ages 5-18	Any Age	min	max	
Population Study of Income Dynamics PSID I, II, III (ISR)	Whole US	I: 1997	3,302	5,327	0	13	University of Michigan, 2016
		II: 2002-2003	4,816	4,825	5	19	
		III:2007-2008	2,633	2,690	10	19	
National-scale Activity Study (NSA)	7 US metro areas	2009	0	6,820	35	92	Knowledge Networks (2009)
RTI Ozone Averting Behavior Study (OAB)	35 US metro areas	2002-2003	1,941	2,872	2	12	Mansfield et al. (2009)
RTP Particulate Matter Panel Study (RTP)	Wake and Orange Counties, NC	2000-2001	0	874	55	85	(Williams et al., 2003a, 2003b), Williams et al., 2001
Study of Use of Products and Exposure-related Behaviors (SUP)	California	2006-2010	1,293	8,831	1	88	Bennett et al. (2012)
Seattle Study (SEA)	Seattle, WA	1999-2001	317	1,645	6	91	Liu et al. (2003)
Valdez Air Health Study (VAL)	Valdez, AK	1990-1991	72	387	11	71	Goldstein et al. (1992)
Washington, DC Study (WAS)	Washington, DC	1982-1983	11	695	18	98	Hartwell et al. (1984); Johnson et al. (1986); Settergren et al. (1984)
All Studies, Areas, and Years (TOTAL):			25,054	177,210	0	98	

^A The APEX activity data file differs from that of the CHAD master database by removing what are considered as unusable diaries for our exposure and risk analyses (~2,000 diary days). The four criteria used to screen the CHAD master database are as follows: 1) Daily maximum temperature is missing, 2) daily average temperature is missing, 3) the day-of-week is missing, and 4) at least 3 hours of events have activity or location codes of "unknown" and/or "missing".

- 1
- 2 Three standard APEX input files are used for the current exposure and risk analyses to
- 3 create the activity pattern profiles for all simulated individuals.
- 4
- 5 • *CHADEvents_060419A.txt*: CHAD ID, clock hour (hhmm), duration of event
- 6 (minutes), CHAD activity code, and CHAD location code, serving as a daily
- 7 sequence of locations visited, activities performed, and their duration
- 8
- 9 • *CHADQuest_060419A.txt*: CHAD ID, day-of-week, sex, race, employment status,
- 10 age, maximum daily temperature, average temperature, occupation, missing time
- 11 (minutes), record count, commute time (see also section 3D.2.5.2)
- *CHADSTATSOutdoor_060419A.txt*: CHAD ID, total daily time spent outdoors (minutes) (see also section 3D.2.5.4)

3D.2.5.2 Commuting and Employment Data

Exposures can vary across a study area based on spatial heterogeneity in ambient air concentrations and how that corresponds with a simulated individual’s activity pattern and geographic location. APEX approximates home-to-work commuting flows between census designated areas for each employed individual, and thus accounts for differing ambient air concentrations that may occur in these geographic locations. APEX has a national commuting database originally derived from 2010 Census tract level data collected as part of the U.S. DOT Census Transportation Planning Package. The data used to generate the APEX commuting file are from the “Part 3-The Journey to Work” files. The Census files contain counts of individuals commuting from home to work locations at a number of geographic scales. These data have been processed to calculate fractions (and hence commute probabilities) for each tract-to-tract flow to create the national commuting data distributed with APEX. This database contains commuting data for each of the 50 states and Washington, D.C. This dataset does not differentiate people that work at home from those that commute within their home tract. A companion file to the commuting flow file is the commuting times file, i.e., an estimate of the usual amount of time in minutes it takes for commuters to get from home to work each day and tract-to-tract commuting distances. The commuting times file information is used to select CHAD activity pattern data from individuals having time spent inside vehicles similar to the census commute times and associated distances travelled. Two standard APEX input files are used for the current exposure and risk analysis, as listed here.

- *Commuting_times_US_2010.txt*: census block IDs, count of all employed individuals, count of employed individuals that do not work at home, 7 groups of block-level one-way commuting times (in minutes)
- *Commuting_flow_US_2010.txt*: census tract IDs, tract-to-tract commute cumulative probabilities (in fractional form), commute distance (km)

Another population-based file associated with commuting is the employment file. This APEX input file contains the probability of employment separately for males and females by age group (starting at age 16) and by census tract (the only census unit available for this type of data). The 2010 Census collected basic population counts and other data using the short form but collected more detailed socioeconomic data (including employed persons) from a relatively small subset of people using the 5-year American Community Survey (ACS).⁵² The ACS dataset

⁵² 2010 U.S. Census American FactFinder: <http://factfinder2.census.gov/>. For instance, to obtain the table ID B23001 “Sex by age by employment status for the population 16 years and over”, the following steps were performed. First, select the “guided search option”, choose “information about people” and select “employment

1 provides the number of people in the labor force, which were stratified by sex/age/tract,
2 considering both civilian workers and workers in the Armed Forces. The data were stratified by
3 sex and age group and were processed so that each sex-age group combination is given an
4 employment probability fraction (ranging from 0 to 1) within each census tract. Children under
5 16 years of age were assumed to be unemployed. One national-based APEX input file is used for
6 the current exposure and risk analyses as follows:

- 7 • *Employment_US_2010.txt*: census tract IDs, employment probabilities (in
8 fractional form), stratified by 13 age groups.⁵³

9 **3D.2.5.3 Assignment of Activity Pattern Data to Individuals**

10 Once APEX identifies the basic personal attributes of a simulated individual (section
11 3D.2.2) and daily air temperatures (section 3D.2.4), activity pattern data obtained from CHAD
12 (section 3D.2.5.1) are then selected based on age, sex, temperature category, and day of the
13 week. These attributes are considered first-order attributes in selecting CHAD diaries when
14 modeling human exposures (Graham and McCurdy, 2004). The particular locations people visit,
15 amount of time spent there, and frequency of these visits can also be influenced by local weather
16 conditions. When considering seasonal temperature ranges (i.e., cold/not cold during cool
17 months; hot/not hot during warm months), (Graham and McCurdy, 2004) found daily maximum
18 temperature (DMT) influences time spent outdoors. Participation rate and amount of time
19 outdoors was found lower on cold DMT days compared to the other three temperature
20 categories, while the participation rate on hot days was less than that on not hot days. Because of
21 these findings, we use a similar DMT range (<55, 55-83, ≥84 °F) to select activity pattern data
22 that best match each study area’s meteorological data for every day of the simulated individual’s
23 exposure profile. This information for the selecting of activity pattern data is found in the
24 following APEX input file, varying by study area and simulation year:

- 25 • *Functions_O3_CSA[number]_040219.txt*: probabilities and interval definitions
26 associated with a few input variables. For activity diary selection - day of week
27 intervals (weekend or weekday) by three temperature ranges.

28
29 While there may be other important attributes that may influence activity patterns (e.g.,
30 obesity, disease status), there are limits to our ability to link to all the possible personal attributes

(labor force) status”, “sex” and “age”. For geography type select “census tract - 140” for each state. Tables containing the employment numbers were downloaded and used to calculate the employment probabilities for each age group.

⁵³ The age groups in this file are: 16-19, 20-21, 22-24, 25-29, 30-34, 35-44, 45-54, 55-59, 60-61, 62-64, 65-69, 70-74, and >75.

1 that may be of interest in modeling an individual’s activities to the CHAD data. This is largely
2 because CHAD is a compilation of data collected from numerous individual activity pattern
3 studies conducted over several decades, many of which had a unique survey design. As a result,
4 there is a varying amount of missing personal attribute data for the surveyed individuals in
5 CHAD. For instance, there are only a limited number of CHAD diaries with survey-requested
6 health information (e.g., the health status of respondents). Specifically regarding whether or not a
7 survey participant had asthma, very few of the available diaries have either a ‘yes’ or ‘no’
8 response to this health condition. When considering the 177,210 diary days used by APEX, there
9 are only 4,935 diary days from individuals having asthma (of which 3,133 are children ages 5-
10 18),⁵⁴ representing a small fraction of the CHAD data. On its own, having approximately 5,000
11 diaries may appear to be a large number of diaries, however, following a grouping of the diaries
12 by their first-order attributes when developing simulated profiles (e.g., age, sex, day-of-week,
13 etc., daily temperature), would likely result in fewer than 100 diaries available for simulating a
14 single day for a particular individual. Accordingly, the selection of diaries to use for APEX-
15 simulated individuals does not consider health status (i.e., any diary is used, regardless of
16 whether the individual indicated they did or did not have asthma, or that information was
17 unknown).

18 This restriction in the number of diaries from individuals having asthma is not considered
19 to be a significant limitation for estimating exposures for simulated individuals with asthma. In
20 general, modeling people with asthma similarly to healthy individuals (i.e., using the same time-
21 location-activity profiles) is supported by the activity analyses reported by van Gent et al. (2007)
22 and Santuz et al. (1997). Other researchers, for example, Ford et al. (2003), have shown
23 significantly lower leisure time activity levels in asthmatics when compared with individuals
24 who have never had asthma. Based on these inconsistent findings, we evaluated this issue in the
25 2014 HREA and, using the available activity pattern data in the CHAD database, we compared
26 participation in afternoon outdoor activities at elevated exertion levels among people having
27 asthma, people not having asthma, and unknown health status (2014 HREA, Appendix G,
28 section 5G-1.4). The 2014 HREA analysis indicated health status had little to no impact on the
29 participation in afternoon activities at elevated exertion levels. A similar analysis was repeated
30 here to include the diary data currently used by APEX, not just those that would be included in
31 the simulations for the 2014 HREA (i.e. ~50,000 diaries).

32 Of interest in this current risk and exposure analysis are instances when individuals
33 experience their highest O₃ exposures. As shown in 2014 HREA, the highest exposures occur

⁵⁴ The American Time Use Survey, a study contributing the largest number of diaries (n=124,517) to CHAD, did not include a question for whether a surveyed individual has asthma.

1 when individuals spend time outdoors, particularly during the afternoon hours (2014 HREA,
2 Appendix 5G section 5G-2). To prepare the APEX activity dataset for analysis here, afternoon
3 hours were characterized as the time between 12 PM and 8 PM and only those persons that spent
4 some time outdoors were retained. As is done by APEX in simulating individuals, level of
5 exertion was estimated by sampling from the specific METs distributions assigned for each
6 person's activity performed. Then, we identified activities having a METs value of greater than
7 three as instances where a person was at moderate or greater exertion levels (U.S. DHHS, 1999).
8 Afternoon outdoor time was then stratified by exertion level, summed for two study groups of
9 interest (i.e., children and adults), and presented in percent form within Table 3D-10.

10 Regarding the diaries for children of interest for these exposure and risk analyses (ages 5-
11 18), about 13% are from an individual having asthma, 48% are from those who do not have
12 asthma, and the remaining portion of children's diaries have unknown health status. About 1% of
13 CHAD diaries for adults are from individuals with asthma and about 11% are from those who do
14 not have asthma. Far fewer children's diaries are from persons whose asthma status is unknown
15 (40%) compared to adults (88%), and the proportions are smaller still in terms of the total
16 available person-days. On average, about 42% of all children having known asthma status spent
17 some afternoon time outdoors, and the percent is actually higher for children with asthma
18 (48.4%) than for children not having asthma (40.5%). About half of the adults whose asthma
19 status was known spent afternoon time outdoors with a participation rate generally similar for
20 adults having asthma and adults not having asthma. Participation in outdoor events for children
21 having unknown asthma status varied little from that of persons with known asthma status.
22 Contrary to this, there were fewer adults with unknown asthma status that participated in outdoor
23 events (29%) when compared to those having known asthma status.

24 The amount of afternoon time spent outdoors by the persons that did so varied little
25 across the two study groups and two asthma classifications. Children, on average, spend
26 approximately 2¼ hours of afternoon time outdoors, 80% of which is at a moderate or greater
27 exertion level, regardless of their asthma status. For children whose asthma status is unknown,
28 slightly more afternoon time is spent outdoors (about 150 minutes) but the percent of afternoon
29 time at moderate or greater exertion levels is slightly lower (about 69%). As seen with children,
30 adults spend approximately 2¼ hours of afternoon time outdoors regardless of their asthma
31 status. However, the percent of afternoon time at moderate or greater exertion levels for adults
32 (about 55%) is lower than that observed for children.

33 Based on this updated analysis and additional comparisons of CHAD diary days with
34 literature reported values of outdoor time participation at varying activity levels (see 2014
35 HREA), there are strong similarities in outdoor time, outdoor event participation, and activity
36 levels achieved among the two study groups and with those reported in independent studies of

1 people with asthma. Thus, we conclude the use of any CHAD diary, regardless of
 2 known/unknown asthma status, is reasonable for purposes of simulating people with asthma in
 3 this exposure and risk analysis.

4 **Table 3D-10. Comparison of time spent outdoors and exertion level by asthma status for**
 5 **children and adult diaries used by APEX.**

Has Asthma?	CHAD: Children (5 to 18) ^A			CHAD: Adults (>18) ^B		
	Yes	No	Unknown	Yes	No	Unknown
Total Person Days (n)	3,133	11,948	9,973	1,279	16,323	127,377
Number of Person Days with Time Spent Outdoors (% participation)	1,517 (48.4%)	4,840 (40.5%)	4,054 (40.6%)	569 (44.5%)	7,900 (48.4%)	36,949 (29.0%)
Overall Percent of Afternoon Hours Spent Outdoors (%)	29.0%	27.3%	31.8%	28.3%	28.9%	27.2%
Overall Percent of Afternoon Time Outdoors at Moderate or Greater Exertion (%)	81.6%	81.1%	69.1%	55.4%	55.1%	62.3%

^A CHAD studies for where a survey questionnaire response of whether or not child had asthma include CIN, ISR, NHA, NHW, OAB, and SEA (see Table 3D-9 for study names).
^B CHAD studies for where survey a questionnaire response of whether or not adult had asthma include CIN, EPA, ISR, NHA, NHW, NSA, and SEA.

6
 7 We also evaluated how temperature influences the amount of afternoon time spent
 8 outdoors while at moderate or greater exertion by children (5-18 years) and adults (19-90 years).
 9 This differs from analyses in Graham and McCurdy (2004) in which all outdoor time at any
 10 exertion level was evaluated and the number of diary days available in CHAD was much less at
 11 that time (~23,000 diary days). Also, in this current analysis, each CHAD/APEX diary day was
 12 grouped by both DMT (<55, 55-83, or ≥84 °F) and day-type (weekday or weekend). Total
 13 available diary days for each of these groups is provided in Table 3D-11. Then, afternoon time
 14 outdoors (12:00 PM to 8:00 PM) was summed and place into one of five hourly groupings (0, 0-
 15 ≤½, ½-≤2, 2-≤4, and >4 hours per day) and the percent of diary days in each group was
 16 calculated, the results of which are provided in Figure 3D-9 for children and adults.

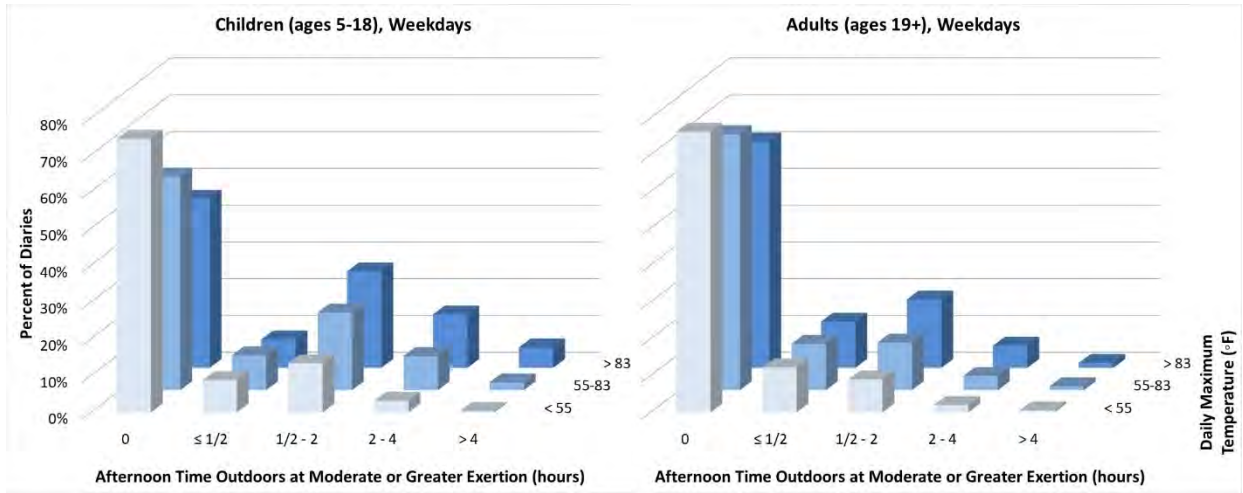
17 Overall, the greatest proportion of diary days would be characterized as not having any
 18 afternoon time spent outdoors at moderate or greater exertion (46 - 76%), with adults
 19 consistently having a greater frequency of not spending afternoon time outdoors than children
 20 (Figure 3D-9). Afternoon time outdoors at moderate or greater exertion for both children and
 21 adults is less likely to occur on cold days (DMT <55 °F), with progressively increased frequency
 22 of outdoor time with increasing temperatures for both day-types. Children are more frequently
 23 spending afternoon time outdoors at elevated exertion levels, particularly when considering the
 24 largest duration assessed (e.g., for durations of time outdoors ≥2 hours, the percent of child diary
 25 days is greater than adults by a factor of 1.3 to 2.7).

1 **Table 3D-11. Number of diary days in CHAD for children and adults, grouped by**
 2 **temperature and day-type categories.**

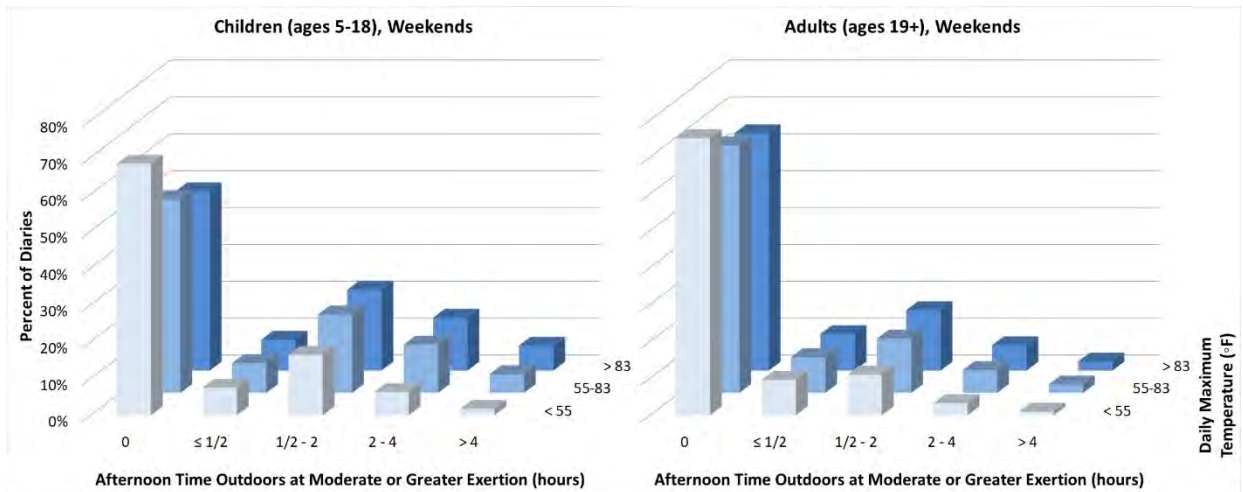
Daily Maximum Temperature (°F)	Children (5-18 years) Diary Days (n)		Adult (19-90 years) Diary Days (n)	
	Weekday	Weekend	Weekday	Weekend
<55	3,883	3,504	19,316	17,136
55-83	6,823	5,800	36,034	32,982
≥84	3,460	1,584	23,865	15,646

The number of diary days here can be used along with Figure 3D-9 to estimate the number of diaries available in each time/hour group. The total number of diary days for this analysis is 170,033 and differs from CHAD/APEX (n=177,210) because of the age range selected.

3



4



5

6 **Figure 3D-9. Percent of children (5-18 years) and adults (19-90 years) having afternoon**
 7 **time outdoors while at moderate or greater exertion, categorized by daily**
 8 **maximum temperature (°F) and time (hours/day) groups.**

1 **3D.2.5.4 Method for Longitudinal Activity Pattern Sequence**

2 In order to estimate population exposure over a full year, a year-long activity sequence
3 needed to be created for each simulated individual based on CHAD, which is largely a cross-
4 sectional activity database of 24-hr records. On average, the typical surveyed subject provided in
5 CHAD has about two days of diary data. For this reason, the construction of a season-long
6 activity sequence for each individual requires some combination of repeating the same data from
7 one subject and using data from multiple subjects. The best approach would reasonably account
8 for the day-to-day and week-to-week repetition of activities common to individuals and
9 recognizing even these diary sequences are not entirely correlated, while maintaining realistic
10 variability among individuals comprising each study group.

11 APEX provides three methods of assembling composite diaries: a basic method, a
12 diversity and autocorrelation (D&A) method, and a Markov-chain clustering (MCC) approach.
13 We have selected the diversity and autocorrelation (D&A) method for this assessment based on
14 our consideration of the assessment objectives, an evaluation of differences in results produced
15 by the three methods, and consideration of flexibility provided by each approach with regard to
16 specifying key variable values, as discussed below. First a brief description of each method is
17 provided below.

18 The basic method involves randomly selecting an activity diary for the simulated
19 individual from a user-defined diary pool (e.g., age, sex). While the method is adequate for
20 estimating a mean short-term exposure for a population as a whole, it is less useful for estimating
21 how often individuals in a population may experience peak O₃ exposures over a year.

22 The D&A method is a complex algorithm for assembling longitudinal diaries that
23 attempts to realistically simulate day-to-day (within-person correlations) and between-person
24 variation in activity patterns (and thus their exposures to the extent they are influenced by spatial
25 and temporal variability in ambient air and microenvironmental O₃ concentrations). This method
26 was designed to capture the tendency of individuals to repeat activities, based on reproducing
27 realistic variation in a key diary variable, which is a user selected function of diary variables. The
28 method targets two statistics: a population diversity statistic (*D*) and a within-person
29 autocorrelation statistic (*A*). The *D* statistic reflects the relative importance of within and
30 between-person variance in the key variable. The *A* statistic quantifies the lag-one (day-to-day)
31 key variable autocorrelation. Values of *D* and *A* for the key variable are selected by the model
32 user and set in the APEX parameters file, and the method algorithm constructs longitudinal
33 diaries that preserve these parameters. Further details regarding this methodology can be found
34 in Glen et al. (2008).

35 The Markov-chain clustering (MCC) approach is similarly complex in attempting to
36 recreate realistic patterns of day-to-day variability. First, cluster analysis is employed to divide

1 the daily activity pattern records into three groups based on time spent in, for example, five
2 microenvironments: indoor-residence, other indoors, outdoor-near roads, other outdoors, and
3 inside vehicles. For each simulated individual, a single time-activity record is randomly selected
4 from each cluster. Then the Markov process determines the probability of a given time-activity
5 pattern occurring on a given day based on the time-activity pattern of the previous day and
6 cluster-to-cluster transition probabilities (and are estimated from the available multi-day time-
7 activity records), thus constructing a long-term sequence for a simulated individual. Details
8 regarding the MCC method and supporting evaluations are provided in U.S. EPA (U.S. EPA,
9 2019a, U.S. EPA, 2019b).

10 Che et al. (2014) performed an evaluation of the impact of the three APEX methods on
11 PM_{2.5} exposure estimates. As expected, little difference was observed across the methods with
12 regard to estimates of the mean exposures of simulated individuals. Differences were observed,
13 however, in the number of multiday exposures exceeding a selected benchmark concentration.
14 With regard to the number of simulated individuals experiencing 3 or more days above
15 benchmark concentrations, the MCC method estimates were approximately 12-14% greater than
16 either the random or D&A methods. For the number of persons experiencing at least one
17 exposure of concern, however, the MCC method estimates were approximately 4% lower than
18 those of the other two methods. For additional context, we note that, using all methods, there is
19 an order of magnitude difference in the number of persons exposed at least once versus three or
20 more times, indicating that, overall, the occurrence of simulated multiday exposures are rare
21 events regardless of method selection.

22 Che et al. (2014) concludes that while the MCC method produces a higher number of
23 multiday exposures, there remains a question whether the MCC method has greater accuracy
24 relative to the other two methods. We note this conclusion applies to both the estimations of
25 single day and multiday exposures, as there is an inverse relationship between the two when
26 simulating exposures using APEX and a finite set of activity pattern data. Thus, the MCC
27 method produces a smaller number of single day exposures above benchmarks relative to the
28 other two methods, estimations also subject to a degree of uncertainty.

29 In the absence of having a robust data set (e.g., multiday/week diary data from a random
30 population) to better evaluate the accuracy of any of the methods, we considered selection of the
31 longitudinal approach for this assessment from a practical perspective, guided by a balancing of
32 the single day and multiday exposures that can be estimated by each method. In so doing, we
33 selected the D&A approach, recognizing that the D&A method allows for flexibility in the
34 selection of the key influential variable and its setting values, and also the ability to directly
35 observe the impact of changes to these values on model outputs.

1 The key variable selected for this exposure and risk analysis is the amount of time an
2 individual spends each day outdoors, as that is the most important determinants of exposure to
3 high levels of O₃ (2014 HREA, Appendix 5G, section 5G-2). In their evaluation, Che et al.
4 (2014) varied the values of *D* and *A* for this variable to determine the impact to estimated
5 exposures. Compared to their base level simulation (i.e., *D*=0.19 and *A*=0.22), increasing both *D*
6 and *A* by 100% increased the number of persons having at least three exposures above the
7 selected benchmark by about 4%, while also reducing the percent of persons experiencing at
8 least one day above benchmarks by less than 1% (Che et al., 2014). In recognizing uncertainty in
9 the parameterization of *D* and *A* (i.e., based on Xue et al., 2004) a limited field study of a small
10 subset of the population, children 7-12) and that the Che et al., 2014 base level simulation *D*&*A*
11 values produced a lower estimate of repeated exposures compared with the MCC method, we
12 have used values of 0.5 for *D* and 0.2 for *A* for all ages to potentially increase representation of
13 multiday exposures without significantly reducing the percent of the population experiencing at
14 least one day at or above benchmark concentrations.

15 **3D.2.6 Microenvironmental Concentrations**

16 In APEX, exposure of simulated individuals occurs in microenvironments (MEs) rather
17 than assuming people are exposed continuously and consistently to ambient air. To best estimate
18 personal exposures, it is important to maintain the spatial and temporal sequence of MEs people
19 inhabit and to appropriately represent the time series of concentrations that occur within them.
20 Two methods are available in APEX for calculating pollutant concentrations within MEs: a mass
21 balance model and a transfer factor approach. In both approaches, ME concentrations depend on
22 the ambient (outdoor) air O₃ concentrations and ambient air temperatures, as well as statistical
23 distributions to parameterize the variables used by each approach. Further, the statistical
24 distributions of some of the key variables depend on values of other variables in the model. For
25 example, the distribution of air exchange rates inside an individual's residence depends on the
26 type of heating and air conditioning present, which are also probabilistic inputs to the model. The
27 value of a variable can be set as a constant for the entire simulation (e.g., house volume remains
28 identical throughout the exposure period), or APEX can sample a new value hourly, daily, or
29 seasonally from user-specified statistical distributions. APEX also allows the user to specify
30 diurnal, weekly, or seasonal patterns for certain ME parameters. Details regarding the two
31 methods can be found in (U.S. EPA, 2019a, U.S. EPA, 2019b) and are briefly described below.

32 The mass balance method, used for the indoor MEs, assumes that an enclosed
33 microenvironment (e.g., a room within a home) is a single well-mixed volume in which the air
34 concentration is approximately spatially uniform (Figure 3D-10). The concentration of an air
35 pollutant in such a microenvironment is estimated using (1) inflow of air into the

1 microenvironment, (2) outflow of air from the microenvironment, (3) removal of a pollutant
 2 from the microenvironment due to deposition, filtration, and chemical degradation, and (4)
 3 emissions from sources of a pollutant inside the microenvironment (not used for this exposure
 4 and risk analysis). Considering the microenvironment as a well-mixed fixed volume of air, the
 5 mass balance equation for a pollutant in the microenvironment can be written in terms of
 6 concentration as follows in Equation 3D-7:

$$7 \quad \frac{dC(t)}{dt} = \dot{C}_{in} - \dot{C}_{out} - \dot{C}_{removal} \quad \text{Equation 3D-7}$$

8 where,

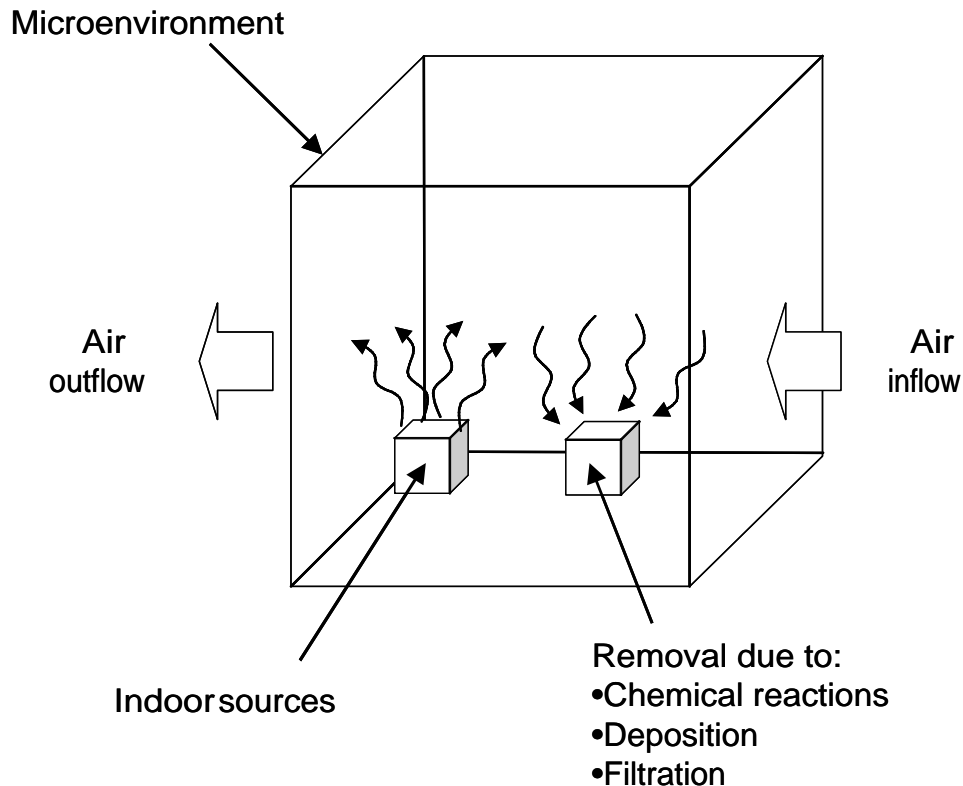
9 $C(t)$ = Concentration in the microenvironment at time t

10 \dot{C}_{in} = Rate of change in $C(t)$ due to air entering the microenvironment

11 \dot{C}_{out} = Rate of change in $C(t)$ due to air leaving the microenvironment

12 $\dot{C}_{removal}$ = Rate of change in $C(t)$ due to all internal removal processes

13



14

15 **Figure 3D-10. Illustration of the mass balance model used by APEX to estimate**
 16 **concentrations within indoor microenvironments.**

1 The factors model (used for the outdoor and inside vehicle MEs) is simpler than the mass
2 balance model. In this method, the value of the ME concentration is not dependent on the ME
3 concentration during the previous time step. Rather, this model uses Equation 3D-8 to calculate
4 the concentration in an ME from the ambient air quality data:

$$5 \quad C_{mean} = C_{ambient} \times f_{proximity} \times f_{pollutant} \quad \text{Equation 3D-8}$$

6 where,

7 C_{mean} = Mean concentration over the time step in a microenvironment (ppm)

8 $C_{ambient}$ = The concentration in the ambient (outdoor) air (ppm)

9 $f_{proximity}$ = Proximity factor (unitless)

10 $f_{pollutant}$ = fraction of ambient air pollutant entering microenvironment (unitless)

11
12 Based on findings from the 2014 HREA, we have specified seven MEs to simulate in this
13 assessment, largely based on two factors: the expectation of a particular ME leading to exposures
14 of interest and the availability of factors needed to reasonably model the ME. The 2014 HREA
15 indicated that high (≥ 50 ppb) 8-hr daily maximum O₃ exposures occurred while individuals spent
16 much larger amounts of afternoon time outdoors compared with those experiencing low (< 50
17 ppb) exposure levels (2014 HREA, Appendix 5G, Figure 5G-5). Given that finding and the
18 objective for the exposure assessment (i.e., understanding how often and where maximum O₃
19 exposures occur), we recognized the added efficiency of minimizing the number of MEs
20 compared to that done in the 2014 HREA (i.e., 28 microenvironments), particularly reducing the
21 number of lower-exposure indoor MEs that were parameterized and included at that time.

22 Accordingly, we aggregated the number of MEs to seven and estimate exposures of
23 ambient air origin that occur within a core group of indoor, outdoor, and inside vehicle MEs.
24 Four indoor MEs (indoor-residence, indoor-restaurant, indoor-school, and indoor-other⁵⁵) were
25 modeled based on having specific air exchange rate data available for each (section 3D.2.6.1).
26 All outdoor locations were assumed to have O₃ concentrations equivalent to ambient air,
27 however there were two MEs used to do so, distinguished by whether or not they occurred near
28 roads. The outdoor near road ME was modeled separately due to the expected decrease in
29 concentrations occurring in that ME relative to that of ambient air concentrations. And finally, an
30 inside-vehicle ME was modeled based on the expectation that it would lead to some instances of
31 relatively lower exposures compared with ambient air concentrations. Table 3D-12 lists the
32 seven microenvironments selected for this analysis and the exposure calculation method used for

⁵⁵ The indoor-other ME is comprised of all non-residential MEs, thus could include office buildings, stores, etc.

1 each. The variables and their associated parameters used to calculate ME concentrations are
2 summarized in subsequent sections below.

3 **Table 3D-12. Microenvironments modeled and calculation method used.**

Microenvironment (ME)	APEX ME	Calculation	Variables ^A
Indoor – Residence	1	Mass balance	AER & RM
Indoor – Restaurant	2	Mass balance	AER & RM
Indoor – School	3	Mass balance	AER & RM
Indoor – Other	4	Mass balance	AER & RM
Outdoor – General	5	Factors	None
Outdoor – Near road	6	Factors	PR
Inside – Vehicle	7	Factors	PE

^A AER = air exchange rate, RM = removal rate, PR = proximity factor, PE = fraction of pollutant entering microenvironment, None = ME concentration is equal to ambient air concentration.

4
5 The seven microenvironments were mapped to the 115 CHAD locations⁵⁶ because using
6 such a large number of MEs would go well beyond the practical scale needed for the exposure
7 and risk analyses. Note that the ambient air concentration used in calculating ME concentration
8 for each exposure event varies temporally and spatially. For example, commuters (i.e., employed
9 individuals who do not work at home) are assigned to either their home tract or work tract
10 concentration, depending on whether the population probabilities and commuting data base
11 produce either a home or work event. Additionally, depending on the particular ME (i.e., other
12 than home or work), the mapping of CHAD locations to the seven MEs also uses an identifier
13 that designates the relative location in the air quality surface from which the ambient air
14 concentration (used to calculate the ME concentration) is selected. For this assessment, such
15 locations would include the Census tract for a simulated individual’s home (H), work (W), near
16 work (NW), near home (NH), last (L, either NH or NW), other (O, average of all), or unknown
17 (U, last ME determined) location. Specific designations are provided in the APEX ME mapping
18 file, with selection based on known factors and professional judgement. For example, when an
19 individual is in their home, the ambient air concentration in the home tract is used to calculate
20 their ME concentration. When the individual is at work, the tract the individual commuted to is
21 used to calculate their ME concentration. Travel inside vehicles used the ambient air
22 concentration data from the tract used to calculate the prior ME concentration. Most other MEs
23 (both indoor and outdoor) use ambient air concentration data selected from near home tracts.

⁵⁶ The location codes indicate specific MEs that extend beyond simple aggregations of indoor, in-vehicle, and outdoor locations where people spend time. For example, CHAD has a location code for when individuals spent time inside their residence while in the kitchen.

1 Status attribute variables are also important in estimating ME concentrations, and can
2 include, but are not limited to, housing type, whether the house has air conditioning, and whether
3 the car has air conditioning. Because outdoor MEs are expected to contribute the most to an
4 individuals' highest O₃ exposure (and potential health risk) and the status attribute variables
5 pertain to indoor MEs, the setting of these particular variables will have limited impact to the
6 exposure and risk results generated here. In this assessment, a number of temperature ranges are
7 used in selecting the particular distribution for estimating air exchange rates (AERs). Maximum
8 daily temperature is also used in diary selection to best match the study area meteorological data
9 for the simulated individual (Graham and McCurdy, 2004) and air conditioning use.

10 Multiple APEX input files (the first and third in the list below), of the same general
11 format, are used for estimating ME concentrations in each study area. A single APEX ME
12 mapping file is used for all study areas. These ME input files contain the parameter settings for
13 all variables described in the subsections that follow.

- 14 • *ME_descriptions_O3_7MEs_CSA[number].txt*: defines ME calculation method,
15 conditional variables used (e.g., temperature categories – see functions file), distribution
16 type, distribution parameters (mean, standard deviation, minimum, maximum) for AERs,
17 decay rates, proximity factors, and PE fractions used to estimate O₃ in 7 MEs.
- 18 • *Microenvironment_mappings_07_MEs.txt*: maps 115 CHAD locations to the 7 APEX
19 MEs and assigns the tract-level ambient air concentrations to use for each location.
20 Contains CHAD location code, CHAD description, APEX ME number, and ambient air
21 concentration location identifier
- 22 • *Functions_O3_CSA[number]_040219.txt*: variables used for selecting AER - air
23 conditioning (A/C) prevalence (home has A/C, does not have A/C) by five temperature
24 ranges for air exchange rate (<50, 50-67, 68-76, 77-85, or >85 °F). (see section 3D.2.6.1)

25 **3D.2.6.1 Indoor Microenvironments**

26 As described above, all four indoor MEs (indoor-residential, indoor-restaurant, indoor-
27 school, and indoor-other) were modeled using a mass balance model. The three variables used to
28 calculate ME concentrations, air exchange rates (section 3D.2.6.1.1), air conditioning prevalence
29 (section 3D.2.6.1.2), and ozone removal rate (section 3D.2.6.1.3) are described below.

30 **3D.2.6.1.1 Air Exchange Rates**

31 Distributions of air exchange rates (AERs, hr⁻¹) for the indoor residential ME were
32 developed using data from several studies. The analysis of these data and the development of
33 most of the distributions used in the modeling were originally described in detail in the 2007
34 exposure analysis (U.S. EPA (2007a), Appendix A) and updated in the 2014 HREA (see
35 Appendix 5E). Briefly, AER distributions for the residential microenvironments depend on the
36 type of air conditioning (A/C) and on the outdoor temperature, among other variables for which
37 we do not have sufficient data to estimate. AER distributions were found vary greatly across

1 cities, A/C types, and temperatures, so that the selected AER distributions for the modeled cities
 2 should also depend on these attributes. For example, the mean AER for residences with A/C
 3 ranges from 0.38 in Research Triangle Park, NC at temperatures > 25 °C upwards to 1.244 in
 4 New York, NY considering the same temperature range (2014 HREA, Appendix 5E). For each
 5 combination of A/C type, city, and temperature with a minimum of 11 AER values, exponential,
 6 lognormal, normal, and Weibull distributions were fit to the AER values and compared.
 7 Generally, the lognormal distribution was the best-fitting of the four distributions, and so, for
 8 consistency, the fitted lognormal distributions are used for all the cases.

9 There were a number of limitations in generating study-area specific AER stratified by
 10 temperature and A/C type. For example, AER data and derived distributions were available only
 11 for selected cities, and yet the summary statistics and comparisons demonstrate that the AER
 12 distributions depend upon the city as well as the temperature range and A/C type. As a result,
 13 city-specific AER distributions were used where possible; otherwise staff selected AER data
 14 from a similar city. Another important limitation of the analysis was that distributions were not
 15 able to be fitted to all of the temperature ranges due to limited number of available measurement
 16 data in these ranges. A description of how these limitations were addressed can be found in the
 17 2014 HREA, Appendix 5E. The AER distributions used for the exposure modeling are given in
 18 Table 3D-13 (Residences with A/C) and Table 3D-14 (Residences without A/C).

19 **Table 3D-13. Air exchange rates (AER, hr⁻¹) for indoor residential microenvironments with**
 20 **A/C by study area and temperature.**

Study Area	Daily Mean Temperature (°C)	Lognormal Distribution GM, GSD, min, max (hr ⁻¹)	Original AER Study Data Used
Atlanta	< 10	0.962, 1.809, 0.1, 10	Research Triangle Park, NC
	10 - 20	0.562, 1.906, 0.1, 10	
	20 - 25	0.397, 1.889, 0.1, 10	
	> 25	0.380, 1.709, 0.1, 10	
Boston, Philadelphia	< 10	0.711, 2.108, 0.1, 10	New York, NY
	10 - 25	1.139, 2.677, 0.1, 10	
	> 25	1.244, 2.177, 0.1, 10	
Dallas, Phoenix	< 20	0.407, 2.113, 0.1, 10	Houston, TX
	20 - 25	0.467, 1.938, 0.1, 10	
	25 - 30	0.422, 2.258, 0.1, 10	
	> 30	0.499, 1.717, 0.1, 10	
Detroit	< 10	0.744, 1.982, 0.1, 10	Detroit, MI or New York, NY
	10 - 20	0.811, 2.653, 0.1, 10	
	20 - 25	0.785, 2.817, 0.1, 10	
	> 25	0.916, 2.671, 0.1, 10	

Study Area	Daily Mean Temperature (°C)	Lognormal Distribution GM, GSD, min, max (hr ⁻¹)	Original AER Study Data Used
Sacramento	< 25	0.503, 1.921, 0.1, 10	Sacramento
	>25	0.830, 2.353, 0.1, 10	
St. Louis	< 10	0.921, 1.854, 0.1, 10	St. Louis
	10 - 20	0.573, 1.990, 0.1, 10	
	20 - 25	0.530, 2.427, 0.1, 10	
	25 - 30	0.527, 2.381, 0.1, 10	
	> 30	0.609, 2.369, 0.1, 10	

1

2 **Table 3D-14. Air exchange rates (AER, hr⁻¹) for indoor residential microenvironments**
3 **without A/C by study area and temperature.**

Study Area	Daily Mean Temperature (°C)	Lognormal Distribution GM, GSD, min, max (hr ⁻¹)	Original AER Study Data Used
Atlanta, St. Louis	< 10	0.923, 1.843, 0.1, 10	St. Louis
	10 - 20	0.951, 2.708, 0.1, 10	
	> 20	1.575, 2.454, 0.1, 10	
Boston, Philadelphia	< 10	1.016, 2.138, 0.1, 10	New York, NY
	10 - 20	0.791, 2.042, 0.1, 10	
	> 20	1.606, 2.119, 0.1, 10	
Dallas, Phoenix	< 10	0.656, 1.679, 0.1, 10	Houston, TX
	10 - 20	0.625, 2.916, 0.1, 10	
	> 20	0.916, 2.451, 0.1, 10	
Detroit	< 10	0.791, 1.802, 0.1, 10	Detroit, MI or New York, NY
	10 - 20	1.056, 2.595, 0.1, 10	
	20 - 25	1.545, 2.431, 0.1, 10	
	>25	1.860, 2.437, 0.1, 10	
Sacramento	< 10	0.526, 3.192, 0.1, 10	Sacramento
	10 - 20	0.665, 2.174, 0.1, 10	
	20 - 25	1.054, 1.711, 0.1, 10	
	> 25	0.827, 2.265, 0.1, 10	

4

5 The AER distribution (hr⁻¹) used for indoor restaurants in all study areas is a fitted
6 lognormal distribution, having a geometric mean = 3.712, geometric standard deviation = 1.855
7 and bounded by the lower and upper values of the sample data set {1.46, 9.07}. This distribution
8 was developed using data from Bennett et al. (2012) who measured AER in restaurants (details
9 on derivation provided in the 2014 HREA, Appendix 5E). The AER distribution (hr⁻¹) used for

1 indoor schools in all study areas is a fitted Weibull distribution,⁵⁷ having a threshold (τ) = 0,
 2 shape (C) = 1.26, and scale (σ) = 1.75, bounded by a lower and upper range {0, 10}. This
 3 distribution was developed from Lagus Applied Technology, 1995, Shendell et al., 2004, and
 4 Turk et al., 1989 who measured AER in schools (raw data provided in Table 3D-15).
 5

6 **Table 3D-15. Individual air exchange rate data (hr⁻¹) obtained from three studies used to**
 7 **develop an AER distribution used for schools in all study areas.**

Individual Air Exchange Rate Data (hr ⁻¹)							
Lagus Applied Technology (1995)				Shendell et al. (2004)			Turk et al. (1989)
0.56	1.34	1.92	2.71	0.1	0.3	0.6	0.8
0.74	1.46	2.26	2.76	0.1	0.4	0.6	1.3
0.76	1.48	2.26	2.81	0.1	0.4	0.6	1.8
0.8	1.58	2.27	2.82	0.1	0.4	0.9	2
0.98	1.61	2.29	2.83	0.2	0.4	0.9	2.2
1.15	1.61	2.33	2.87	0.2	0.4	1.2	2.2
1.19	1.67	2.38	2.93	0.2	0.4	1.3	3
1.21	1.67	2.4	3.03	0.2	0.5	1.3	
1.22	1.73	2.53	3.23	0.2	0.5	1.4	
1.23	1.8	2.53	3.7	0.3	0.6	1.8	
1.23	1.84	2.57	4.38	0.3	0.6	2.9	
1.27	1.9	2.68	5.03	0.3	0.6	5.4	
1.33	1.91	2.71	8.72				

8
 9 The AER distribution (hr⁻¹) used for indoor other in all study areas is a fitted lognormal
 10 distribution, having a geometric mean = 0.949, geometric standard deviation = 1.857 and
 11 bounded by the lower and upper values of the sample data set {0.30, 4.02}. This distribution was
 12 developed using data from Bennett et al. (2012) who measured AER in non-residential buildings
 13 (details on derivation provided in the 2014 HREA, Appendix 5E).
 14

3D.2.6.1.2 Air Conditioning Prevalence

15 The selection of an AER distribution for the indoor residence ME is conditioned on the
 16 presence or absence of A/C. We assigned this housing attribute to indoor residential
 17 microenvironments using A/C prevalence data from the American Housing Survey (AHS).⁵⁸ The

⁵⁷ Of the three statistical distributions evaluated (lognormal, gamma, Weibull), results of a Cramer-von Mises goodness of fit test indicated the data distribution was not statistically different than a Weibull distribution.

⁵⁸ 2015 and 2017.xlsx files were downloaded from <https://www.census.gov/programs-surveys/ahs/data/interactive/ahstablecreator.html> for Atlanta, Boston, Dallas, Detroit, Philadelphia, and Phoenix (accessed on 3/4/2019). The most recent data available for Sacramento and St. Louis was 2011 and available at

1 A/C prevalence data were assigned to our study areas where the AHS data best matched our
 2 exposure simulation years and or study area. In all study areas and for each year, housing units
 3 containing either central or 3 or more room AC were summed, followed by the calculation of the
 4 A/C prevalence. If multiple years were available, these data were averaged to generate the final
 5 A/C prevalence (unitless) for each study area (Table 3D-16). For the other three indoor MEs
 6 (indoor-restaurant, indoor-school, and indoor-other) mechanical ventilation was assumed to be
 7 present in all buildings (i.e., A/C prevalence = 1.0).

8 **Table 3D-16. A/C prevalence from US Census American Housing Survey (AHS) data by**
 9 **study area.**

Study Area	Total Housing Units (×1,000)	Central AC (×1,000)	Room AC 3 or more (×1,000)	Year	AC Prevalence (unitless)	Mean AC Prevalence (unitless)	No AC Prevalence (unitless)
Atlanta	1982.8	1875.2	27.3	2015	0.96	0.96	0.04
	2109	2001	22.7	2017	0.96		
Boston	1838.4	649	311.9	2015	0.523	0.531	0.469
	1854	674.6	322.1	2017	0.538		
Dallas	2471.2	2323.1	49.9	2015	0.96	0.966	0.034
	2565	2444	46.7	2017	0.971		
Detroit	1709	1267.1	34	2015	0.761	0.761	0.239
	1723	1280	31.1	2017	0.761		
Philadelphia	2216.1	1395.4	295.9	2015	0.763	0.776	0.224
	2308	1516	303.1	2017	0.788		
Phoenix	1644	1591.3	7.4	2015	0.972	0.968	0.032
	1686	1619	6.7	2017	0.964		
Sacramento	783.7	677.5	4.6	2011	0.87	0.87	0.13
St. Louis	1115.2	1013.1	23.2	2011	0.929	0.929	0.071

10

11 **3D.2.6.1.3 Ozone Decay and Deposition Rates**

12 As done for the 2014 HREA, a distribution for combined O₃ decay and deposition rates
 13 was obtained from the analysis of measurements from a study by Lee et al. (1999). This study
 14 measured decay rates in the living rooms of 43 residences in Southern California. Measurements
 15 of decay rates in a second room were made in 24 of these residences. The 67 decay rates range
 16 from 0.95 to 8.05 hr⁻¹. A lognormal distribution was fit to the measurements from this study,

<https://www.census.gov/programs-surveys/ahs/data/2011/ahs-2011-summary-tables/ahs-metropolitan-summary-tables.html> (accessed on 4/2/2019).

1 yielding a geometric mean of 2.51 hr⁻¹ and a geometric standard deviation of 1.53 hr⁻¹. These
2 values are constrained to lie between 0.95 and 8.05 hr⁻¹. This combined O₃ decay and deposition
3 rate distribution was used for all four indoor microenvironments.

4 **3D.2.6.2 Outdoor Microenvironments**

5 As mentioned above, the two outdoor MEs (outdoor-general and outdoor-near road) used
6 the factors approach to estimate ME concentrations. The factors approach uses two variables in
7 combination with ambient air O₃ concentrations: a proximity factor and a factor expressing the
8 fraction of a pollutant entering (PE factor) an ME, and these are discussed below.

9 Proximity factors are used to adjust ambient air O₃ concentrations, based on the ME
10 location relative to that of the ambient air concentration. For the outdoor-general ME, there is no
11 adjustment used (proximity = 1.0); it is assumed that wherever an individual is outdoors, the
12 individual experiences the ambient air O₃ concentrations for the tract they are present in at that
13 time (e.g., at home, at work, or nearby census tract). For the outdoor-near road ME, a proximity
14 factor is used, recognizing that ambient air concentrations measured away from roadways tend to
15 increase with distance. As done for the 2014 HREA, we employed the distribution for local roads
16 (i.e., a normal distribution {0.755, 0.203}, bounded by 0.422 and 1.0) derived from the
17 Cincinnati Ozone Study (American Petroleum Institute, 1997, Appendix B; Johnson et al., 1995),
18 based on the assumption that most of the outdoors-near-road ozone exposures will occur
19 proximal to local roads (see Table 3D-17 and details below in section 3D.2.6.3).

20 PE factors are used to adjust for the percent of a pollutant entering a ME. PE factors for
21 the outdoor-general and outdoor-near road MEs, because they are effectively aligned with the
22 ambient air O₃ concentrations, are set equivalent to 1.

23 **3D.2.6.3 Inside-Vehicle Microenvironments**

24 As done for the 2014 HREA, for the in-vehicle ME, proximity and PE factor distributions
25 were obtained from the Cincinnati Ozone Study (American Petroleum Institute, 1997, Appendix
26 B; Johnson et al., 1995). This field study was conducted in the greater Cincinnati metropolitan
27 area in August and September 1994. Vehicle tests were conducted according to an experimental
28 design specifying the vehicle type, road type, vehicle speed, and ventilation mode. Vehicle types
29 were defined by the three study vehicles: a minivan, a full-size car, and a compact car. Road
30 types were interstate highways (interstate), principal urban arterial roads (urban), and local roads
31 (local). Nominal vehicle speeds (typically met over 1-min intervals within 5 mph) were at 35
32 mph, 45 mph, or 55 mph. Ozone concentrations were measured inside the vehicle, outside the
33 vehicle, and at six fixed-site monitors in the Cincinnati area. Table 3D-17 lists the parameters of
34 the normal distributions developed for proximity and PE factors (both are unitless) for in-vehicle
35 microenvironments used in this exposure and risk analysis.

1 A daily conditional variable was used to select the three proximity factor distributions to
 2 use in estimating the inside-vehicle ME concentrations. The 2015-2017 Vehicle Miles of Travel
 3 (VMT) data available from the U.S. Department of Transportation (DOT) were used to generate
 4 these daily conditional variables.⁵⁹ For local and interstate road types, the VMT for the same
 5 DOT categories were used. For urban roads, the VMT for all other DOT road types were
 6 summed (i.e., other freeways/expressways, other principal arterial, minor arterial, and collector).
 7 Table 3D-18 summarizes the conditional variables used for each study area to select for the
 8 proximity factor distribution used to estimate inside-vehicle ME concentrations.

9 **Table 3D-17. Parameter values for distributions of penetration and proximity factors used**
 10 **for estimating in-vehicle ME concentrations.**

ME Factor	Road Type	Arithmetic Mean (unitless)	Standard Deviation (unitless)	Lower Bound ^A (unitless)	Upper Bound (unitless)
PE	All	0.300	0.232	0.100	1.0
Proximity	Local	0.755	0.203	0.422	1.0
	Urban	0.754	0.243	0.355	1.0
	Interstate	0.364	0.165	0.093	1.0

^A A 5th percentile value estimated using a normal approximation as Mean - 1.64 × standard deviation.

11
 12 **Table 3D-18. VMT (2015-2017) derived conditional probabilities for interstate, urban, and**
 13 **local roads used to select inside-vehicle proximity factor distributions in each**
 14 **study area.**

Study Area	Conditional Probabilities for Vehicle Proximity Factors (unitless)		
	Interstate	Urban	Local
Atlanta	0.339	0.392	0.269
Boston	0.416	0.455	0.129
Dallas	0.496	0.453	0.051
Detroit	0.357	0.531	0.112
Philadelphia	0.361	0.523	0.116
Phoenix	0.364	0.542	0.094
Sacramento	0.456	0.433	0.111
St. Louis	0.460	0.363	0.177

⁵⁹ Data were downloaded (accessed on 3/13/2019) from U.S. Department of Transportation (DOT) Federal Highway Administration (FHA) Highway Statistics Series Publications. The three individual years (2015-2017) of data were downloaded from dropdown menu available at: <https://www.fhwa.dot.gov/policyinformation/statistics.cfm>.

3D.2.7 Estimating Exposure

APEX estimates the complete time series of exposure and breathing rate for every simulated individual. This is because APEX accounts for important factors that influence exposure and include the magnitude, duration, frequency of exposures, and the breathing rate of individuals at the time of exposure. APEX can summarize exposure data using standardized time metrics (e.g., hourly or daily average, daily maximum 7-hr average), as is needed for comparison to benchmark concentrations (section 3D.2.8.1) or can output the minute-by-minute exposure concentrations and simultaneous breathing rate, as is needed for the lung function risk modeling (section 3D.2.8.2.2). As a reminder, calculated exposures are distinct from that of ambient air concentrations by accounting for simulated individual's time-location-activity patterns and O₃ concentration decay/variation occurring within the occupied microenvironments. Further, exposures (and hence health risks) are estimated for four groups of individuals residing in each study area: children (individuals aged 5 to 18 years), children with asthma, adults (individuals older than 18 years), and adults with asthma.

3D.2.8 Estimating Risk

We derived two types of metrics to characterize potential population health risk: a comparison of simulated exposures to benchmark concentrations (section 3D.2.8.1) and by using simulated exposures to estimate lung function risk (section 3D.2.8.2). As done in the 2015 review, these two approaches are based on the body of evidence from the controlled human exposure studies reporting lung function decrements (as measured by changes in FEV₁)⁶⁰ along with supporting health evidence from O₃-related epidemiologic studies. As discussed in Appendix 3 of the ISA, there is a significant body of controlled human exposure studies reporting lung function decrements and respiratory symptoms in adults associated with 1- to 6.6-hr exposures to O₃, all but a few of which were available in the 2015 review and no new studies that included 6.6-hour exposures were available (ISA, Appendix 3, section 3.1.4.1.1; 2013 ISA, section 6.2.1.1). The exposure studies of greatest interest are those that have exposed subjects during exercise (ISA, Appendix 3; 2013 ISA, section 6.2.1.1). In general, the 1- to 2-hr exposure studies utilize an intermittent exercise protocol in which subjects rotate between periods of exercise and rest, though a limited number of these studies use a continuous exercise regime. A quasi-continuous exercise protocol is common to the 6.6-hr exposure studies where subjects

⁶⁰ There are other respiratory responses resulting from O₃ exposures that were measured in these studies, including increased lung inflammation, increased respiratory symptoms, increased airway responsiveness, and impaired host defenses. While the available quantitative information is inadequate to reasonably model these other health endpoints, nevertheless the observed responses remain informative in characterizing overall risks.

1 complete six 50-min periods of exercise followed by 10-min rest periods (along with a 35-min
2 lunch/rest period) (ISA, Appendix 3, section 3.1.4.1.1).

3 For lung function risk, we estimate risk of an O₃-related decrement at or above 10%, 15%
4 and 20%. These sizes of decrements have been used in the risk assessments for reviews
5 completed in 2015, 2008 and 1997 (2014 HREA; U.S. EPA, 2007a, U.S. EPA, 2007b; Whitfield
6 et al., 1996). In the 2015 review, the CASAC concurred with the EPA's use in the 2014 HREA
7 of estimated FEV₁ decrements of ≥15% as a scientifically relevant surrogate for adverse health
8 outcomes in active healthy adults, and an FEV₁ decrement of ≥10% as a scientifically relevant
9 surrogate for adverse health outcomes for people with asthma and lung disease (Frey, 2014, p.
10 3).

11 **3D.2.8.1 Comparison to Benchmark Concentrations**

12 For the comparison of simulated exposures to benchmark concentrations that reflect
13 observations from the 6.6-hr controlled human exposure studies, APEX estimates the daily
14 maximum 7-hr average O₃ exposure⁶¹ for every simulated individual, stratified by exertion level
15 at the time of exposure. This indicator was selected based on controlled human exposure studies
16 where reported adverse health responses were associated with exposure to O₃ and while the study
17 subject was exercising.⁶² A 7-hr average exposure concentration is more appropriate than using
18 an 8-hr average (as was done for the prior REAs) because it aligns more closely to the 6.6-hr
19 durations of the controlled human exposure studies on which the benchmark concentrations are
20 based.⁶³ The 7-hr average exposure concentrations experienced by simulated individuals while at
21 moderate or greater exertion (EVR ≥17.32 ± 1.25 L/min-m² body surface area; see above section
22 3D.2.2.3.3) are then compared to the benchmark concentrations.

23 Benchmark concentrations used in this assessment include O₃ exposure concentrations of
24 60, 70 and 80 ppb; the same benchmarks used for the 2014 HREA (based on there being no new
25 6.6-hr controlled human exposure studies that might inform consideration of alternatives).
26 Estimating the occurrence of ambient air-related 7-hr average O₃ exposures at and above these

⁶¹ Only the maximum 7-hr average O₃ exposure concentration is retained by APEX for each day simulated, per person.

⁶² Health responses observed in the controlled human exposure studies are from 6.6-hr exposures to O₃, that involved quasi-continuous exercise. Therefore, it is possible that the effects observed at benchmark levels identified using a 6.6-hr exposure could occur at slightly lower concentrations for a comparable 7-hr exposure and occur at still lower concentrations for a comparable 8-hr exposure. From a practical perspective, there would be a greater number of individuals estimated at or above a particular benchmark when averaging exposures across a 6.6-hr period than when compared to simulations using 7-hr or 8-hr averaging (the latter of which was used in the prior assessments and recognized specifically in the 2014 HREA, section 5.2.8, footnote 18).

⁶³ Note that the 8-hr averaging time for ambient air O₃ concentrations associated with the current standard remains the same as used in prior assessments. The only difference is that for the current exposure and risk analysis, 8-hr ambient air O₃ concentrations are now evaluated with a more appropriate exposure and risk metric (i.e., a 7-hr average exposure benchmark).

1 benchmark levels is intended to provide perspective on the potential for public health impacts of
2 O₃-related health effects observed in human clinical and toxicological studies, but for which
3 available data do not support development of E-R functions, precluding their evaluation in
4 quantitative risk assessments (e.g., lung inflammation, increased airway responsiveness, and
5 decreased resistance to infection), as well as lung function decrements which are currently
6 evaluated in quantitative risk assessments. The 80 ppb benchmark concentration represents an
7 exposure where multiple controlled human exposure studies (of the 6.6-hr, with exercise design)
8 demonstrate a range of O₃-related respiratory effects including lung inflammation and airway
9 responsiveness, as well as respiratory symptoms, in healthy adults. The 70 ppb benchmark
10 concentration reflects a study that found statistically significant decrements in lung function as
11 well as increased respiratory symptoms. The 60 ppb benchmark level represents the lowest
12 exposure level at which statistically significant decrements in lung function, but not respiratory
13 symptoms, have been observed in studies of healthy individuals (see Table 3-2 of the main
14 document).⁶⁴ This is summarized in Table 3D-19 below. Further details on the body of evidence
15 supporting the selection of these benchmark levels is described in the ISA, Appendix 3 and
16 summarized in the section 3.3 of the main document and Appendix 3A.

17 APEX then calculates two general types of exposure estimates for the population of
18 interest: the estimated number of people exposed to a specified O₃ concentration level and, the
19 number of days per year that they are so exposed, while at moderate or greater exertion. The
20 former highlights the number of individuals exposed *one or more* times per year (i.e., at least
21 once) at or above a selected benchmark level. The latter is expressed as *multiday* exposures, that
22 is, the number of times per year each simulated individual experiences a daily maximum
23 exposure at or above a benchmark. These same exposure results are also used in estimating
24 population-based lung function risk (section 3D.2.8.2.1).

25

⁶⁴ Prolonged exposure to 40 ppb O₃ results in a small decrease in group mean FEV₁ that is not statistically different from responses following exposure to filtered air (Adams, 2002; Adams, 2006).

1 **Table 3D-19. Responses reported in 6.6-hr controlled human exposure studies at a given**
 2 **benchmark concentration.**

Benchmark Concentration (ppb)	Responses Reported in Controlled Human Exposure Studies ^A	
	Decrements in Lung Function, and Other Effects	Respiratory Symptoms
≥80	Prolonged exposure to an average O ₃ concentration of 80 ppb, 100 ppb, or 120 ppb O ₃ results in statistically significant group mean decrements in FEV ₁ ranging from 6 ^B to 8%, 8 to 14%, and 10 to 16%, respectively. ^C Statistically significant increases in multiple inflammatory response indicators and in airway responsiveness.	Statistically significant increases in respiratory symptoms (ISA, section 3.1.4.2.1).
≥70	Prolonged exposure to an average O ₃ concentration of 70 ppb results in a statistically significant group mean decrement in FEV ₁ of about 6%. ^D	
≥60	Prolonged exposure to an average O ₃ concentration of 60 ppb results in group mean FEV ₁ decrements ranging from 1.7% to 3.5%. ^E Based on data from multiple studies, the weighted average group mean decrement was 2.5%. In some analyses, these group mean decrements in lung function were statistically significant ^F while in other analyses they were not. ^G Statistically significant increases in sputum neutrophils, an indicator of inflammatory response.	None of studies at this exposure concentration have observed a statistically significant increase in symptom scores (ISA, section 3.1.4.2.1).

^A Information is drawn from Table 3A-1 of Appendix 3A for 6.6-hr exposure protocol with exercise EVR of 20 L/min/m² (see also ISA, Figure 3-3). These studies have been performed with healthy adult subjects.
^B Measurements collected at 80 ppb exposure for 30 subjects as part of the Kim et al. (2011) study that were presented only in Figure 5 of McDonnell et al. (2012) indicate a group mean decrement of 3.5%.
^C Folinsbee et al. (1994), Horstman et al. (1990), McDonnell et al. (1991), Adams (2002), Adams (2006), Adams (2000), Adams and Ollison (1997), Schelegle et al. (2009).
^D Schelegle et al. (2009).
^E Adams (2002), Adams (2006), Schelegle et al. (2009), and Kim et al. (2011).
^F Brown et al. (2008), Kim et al. (2011). In an analysis of the Adams (2006) data, Brown et al. (2008) addressed the more fundamental question of whether there were statistically significant differences in responses before and after the 6.6-hr exposure period and found the study group average effect on FEV₁ at 60 ppb to be small, but statistically significant using several common statistical tests, even after removal of potential outliers.
^G Adams (2006), Schelegle et al. (2009).

3

4 **3D.2.8.2 Lung Function Risk**

5 We used two approaches to estimate health risk. As done for the lung function risk
 6 assessments conducted during prior O₃ NAAQS reviews, the first approach used a Bayesian
 7 Markov Chain Monte Carlo technique to develop probabilistic population-based Exposure-
 8 Response (E-R) functions. These population-based E-R functions were then combined with the
 9 APEX estimated population distribution of 7-hr maximum exposures for people at or above
 10 moderate exertion (EVR ≥17.32 ± 1.25 L/min-m² body surface area) to estimate the number of
 11 people expected to experience lung function decrements. The second approach is based on the
 12 McDonnell-Stewart-Smith (MSS) FEV₁ model (McDonnell et al., 2013). The MSS model uses
 13 the time-series of O₃ exposure and corresponding ventilation rates for each APEX simulated

1 individual to estimate their personal time-series of FEV₁ reductions, selecting the daily
2 maximum reduction for each person. As done for the exposure benchmark analysis, APEX
3 calculates, for the population of interest, the estimated number of simulated individuals expected
4 to experience an FEV₁ response at or above a selected level and the number of days per year that
5 may occur per person. A key difference between these approaches is that the population-based E-
6 R method directly approximates a population distribution of FEV₁ reductions while the MSS
7 model estimates FEV₁ reductions at the individual level (which are then aggregated to a
8 population level). Each of these approaches is discussed in detail below.

9 **3D.2.8.2.1 Population-based E-R function**

10 For developing the population-based E-R function, we used the exact same E-R function
11 as used for the 2014 HREA given CASAC advice on the approach used for the 2008 O₃ review
12 (Henderson, 2006) and that there were no new controlled human exposure study data to justify
13 the generating of a new E-R function for this current analysis. Briefly, data from several
14 controlled human exposure studies that evaluated 6.6-hr exposures at moderate exertion were
15 combined and used to estimate E-R functions. Considering the above discussion and as done in
16 the 2014 HREA, we separated the controlled human exposure study data into three lung function
17 decrement categories. The mid- to upper-end of the range of moderate levels of functional
18 responses and higher (i.e., FEV₁ decrements $\geq 15\%$ and $\geq 20\%$) are included to generally
19 represent potentially adverse lung function decrements in active healthy adults, while for people
20 with asthma or lung disease, a focus on moderate functional responses (FEV₁ decrements down
21 to 10%) may be appropriate (Table 3D-20 and Figure 3D-11).⁶⁵ The controlled human exposure
22 study data in this table were first corrected on an individual basis for study effects in clean
23 filtered air to remove any systemic bias that might be present in the data attributable to the
24 effects of the experimental procedures and extraneous responses (e.g., exercise, diurnal
25 variability, etc.) (2013 ISA, pp. 6-4 and 6-5). This is done by subtracting the FEV₁ decrement in
26 filtered air from the FEV₁ decrement (at the same time point) during exposure to O₃. An example
27 of this calculation is given in the 2014 HREA, Appendix 6D.

28

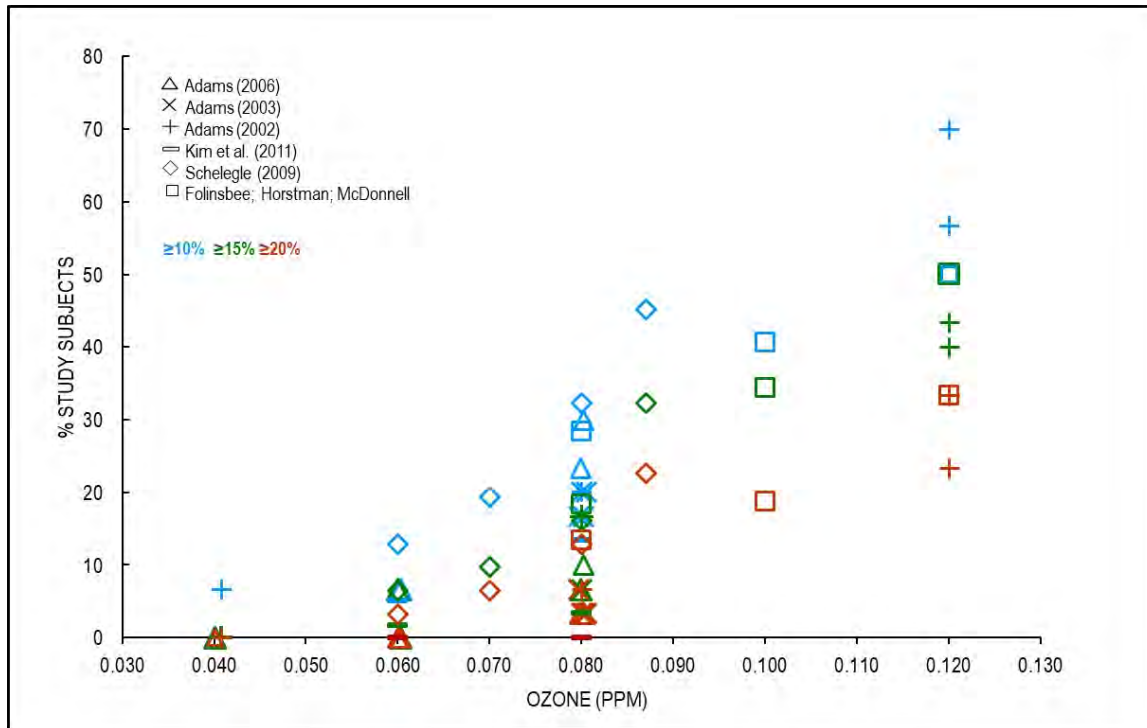
⁶⁵As in past reviews, the EPA has summarized study results with regard to multiple magnitudes of lung function decrement, including 10%, recognizing that 10% has been used in clinical settings to detect a FEV₁ change likely indicative of a response rather than intrasubject variability, e.g., for purposes of identifying subjects with responses to increased ventilation (Dryden, 2010).

1 **Table 3D-20. Summary of controlled human exposure study data stratified by**
 2 **concentration level and lung function decrements, corrected for individual**
 3 **response that occurred while exercising in clean air, ages 18-35.**

Study, Grouped by Average O ₃ Exposure	Protocol	Study Subjects (n)	Subjects Responding (n) ^A		
			ΔFEV ₁ ≥10%	ΔFEV ₁ ≥15%	ΔFEV ₁ ≥20%
0.040 ppm O₃					
Adams (2002)	Square-wave (constant level), face mask	30	2	0	0
Adams (2006)	Variable levels (exercise avg = 0.040 ppm)	30	0	0	0
0.060 ppm O₃					
Adams (2006)	Square-wave	30	2	0	0
	Variable levels (exercise avg = 0.060 ppm)	30	2	2	0
Kim et al. (2011)	Square-wave	59	3	1	0
Schelegle et al. (2009)	Variable levels (exercise avg =0.060 ppm)	31	4	2	1
0.070 ppm O₃					
Schelegle et al. (2009)	Variable levels (exercise avg= 0.070 ppm)	31	6	3	2
0.080 ppm O₃					
Adams (2002)	Square-wave, face mask	30	6	5	2
Adams (2003)	Square-wave, chamber	30	6	2	1
	Square-wave, face mask	30	5	2	2
	Variable levels (exercise avg=0.080 ppm), chamber	30	6	1	1
	Variable levels (exercise avg=0.080 ppm), face mask	30	5	1	1
Adams (2006)	Square-wave	30	7	2	1
	Variable levels (exercise avg=0.080 ppm)	30	9	3	1
F-H-M ¹	Square-wave	60	17	11	8
Kim et al. (2011)	Square-wave	30	4	1	0
Schelegle et al. (2009)	Variable levels (exercise avg=0.080 ppm)	31	10	5	4
0.0870 ppm O₃					
Schelegle et al. (2009)	Variable levels (exercise avg=0.087 ppm)	31	14	10	7
0.100 ppm O₃					
F-H-M ¹	Square-wave	32	13	11	6
0.120 ppm O₃					
Adams, 2002	Square-wave, chamber	30	17	12	10
	Square-wave, face mask	30	21	13	7
F-H-M ^B	Square-wave	30	18	15	10

^A Data from 2014 HREA, Table 6-3 and were originally compiled by Abt (2013). Individual subject responses were corrected using pre- and post-exposure observations.
^B F-H-M combines data from Folinsbee et al. (1988), Horstman et al. (1990), and McDonnell et al. (1991).

4
5



1
2 **Figure 3D-11. Controlled human exposure data for FEV₁ responses in individual study**
3 **subjects.**

4 A Bayesian Markov Chain Monte Carlo (BMCMC) approach (Lunn et al., 2012)
5 developed as part of an earlier O₃ exposure and risk analysis (U.S. EPA, 2007a, U.S. EPA,
6 2007b, section 3.1.2) was modified for the 2014 HREA and used to generate the population-
7 based E-R functions using the updated controlled human exposure study data (Abt, 2013).⁶⁶
8 Briefly, we considered both linear and logistic functional forms in estimating the E-R function
9 and chose a 90 percent logistic/10 percent piecewise-linear split using a BMCMC approach. For
10 each of the three measures of lung function decrement, we first assumed a 90 percent probability
11 that the E-R function has the following 3-parameter logistic form indicated by Equation 3D-9:⁶⁷

12
$$y(x; \alpha, \beta, \gamma) = \frac{\alpha e^\gamma (1 - e^{\beta x})}{(1 + e^\gamma)(1 + e^{\beta x + \gamma})}$$
 Equation 3D-9

⁶⁶ In some of the controlled human exposure studies, subjects were exposed to a given O₃ concentration more than once – for example, using a constant (square-wave) exposure pattern in one protocol and a variable (triangular) exposure pattern in another protocol. However, because there were insufficient data to estimate subject-specific response probabilities, we assumed a single response probability (for a given definition of response) for all individuals and treated the repeated exposures for a single subject as independent exposures in the binomial distribution.

⁶⁷ The 3-parameter logistic function is a special case of the 4-parameter logistic, in which the function is forced to go through the origin, so that the probability of response to 0.0 ppm is 0.

1 where x denotes the O₃ concentration (in ppm) to which the individual is exposed, y
2 denotes the corresponding response (decrement in FEV₁ \geq 10%, \geq 15% or \geq 20%), and α , β , and
3 γ are the three parameters whose values are estimated.

4 We then assumed a 10 percent probability that the E-R function has the following 2-piece
5 linear with threshold (hockey stick) form⁶⁸ indicated by Equation 3D-10:

$$6 \quad y(x; \alpha, \beta) = \begin{cases} \alpha + \beta x, & \text{for } \alpha + \beta x > 0 \\ 0, & \text{for } \alpha + \beta x < 0 \end{cases} \quad \text{Equation 3D-10}$$

7 The selection of the 90 percent logistic/10 percent piecewise-linear split was based
8 largely on the results of sensitivity analyses in the 2007 O₃ risk assessment combined with
9 CASAC advice on the model form (U.S. EPA, 2007b),⁶⁹ and from model fit determined in the
10 2014 HREA.⁷⁰ Therefore, as done for the 2014 HREA, we are using only the 90/10 E-R function
11 in the current analysis to estimate risk. Further, because there were no newly available controlled
12 human exposure study data for 6.6-hr duration exposures since the 2014 HREA, we used the
13 exact same 90/10 E-R function derived at that time, the overall approach of which is briefly
14 described below.

15 To generate the E-R functions, prior distributions needed to be specified to estimate the
16 posterior distribution for each of the unknown parameters (Box and Tiao, 1973). For the logistic
17 functional form, we assumed lognormal priors and used Max likelihood estimates (MLE) of the
18 means and variances for the 3 parameters. For the linear functional form, we assumed normal
19 priors using ordinary least square (OLS) estimates for the means and variances for the
20 parameters.

21 For each of the two functional forms (logistic and linear), we derived the posterior
22 distributions using the binomial likelihood function and prior distributions for each of the
23 unknown parameters. Specifically, we used three Markov chains (each chain corresponds to a set

⁶⁸ The 2-piece linear models estimate no occurrences below about 40 ppb for the 10% lung function decrement and below about 60 ppb for the 15% and 20% lung function decrements based on the limited available data at those exposure levels. Note that as these two-piece linear model forms are combined with a second model form (logistic) for the final model, their contribution to estimated responses is low.

⁶⁹ The 1997 risk assessment used a linear form consistent with the advice from the CASAC O₃ panel at the time that a linear model reasonably fit the available data at exposures of 0.08, 0.10, and 0.12 ppm. Following the addition of exposures data at 0.06 and 0.04 ppm in the 2007 assessment, a logistic model was found to provide a good fit to the data. The CASAC O₃ panel for that review noted that there are only limited data at the two lowest exposure levels and, as a result, a linear model could not entirely be ruled out, resulting in the combined model based on both the logistic and linear forms (U.S. EPA, 2007b).

⁷⁰ Analyses using the updated data available for the 2014 HREA determined that for each of the three E-R curves, the 90/10 logistic/linear mix has smaller error in fit (weighted RMSE) relative to the other two E-R curves evaluated: one having a 80/20 logistic/linear mix and the other having a 50/50 mix.

1 of initial parameter values) and for each chain we used 4,000 iterations as the “burn-in” period⁷¹
 2 followed by 96,000 iterations for the estimation. Each iteration corresponds to a set of estimates
 3 for the parameters of the (logistic or linear) exposure-response function. We then examined the
 4 outputs using the options WinBUGS provides to check convergence and auto-correlation (e.g.,
 5 trace plot, auto correlation). Finally, we combined 8,100 sets of values from the logistic model
 6 runs (the last 2,700 iterations from each chain) with 900 sets of values from the linear model runs
 7 (the last 300 iterations from each chain) to obtain a single combined distribution for each
 8 predicted value, reflecting the 90 percent/10 percent assumptions stated above (WinBUGS v
 9 1.4.3; Lunn et al., 2012).

10 We selected the median (50th percentile) E-R function from the 9,000 sets of functions to
 11 estimate the risk for changes in FEV₁ ≥10%, ≥15%, and ≥20% (Figure 3D-12). The original E-R
 12 data to which the curves were fit are also provided in the figure, along with the derived E-R
 13 function data used to combine with the daily maximum 7-hr exposures for the simulated
 14 population, while at moderate exertion (section 3D.2.8.1). The population at-risk is estimated by
 15 multiplying the expected response rate by the number of people exposed in the relevant
 16 population (and stratified by 7-hr average exposures, in 0.01 ppm increments), as shown in
 17 Equation 3D-11:

$$18 \quad R_k = \sum_{j=1}^N P_j x(RR_k | e_j) \quad \text{Equation 3D-11}$$

19 where:

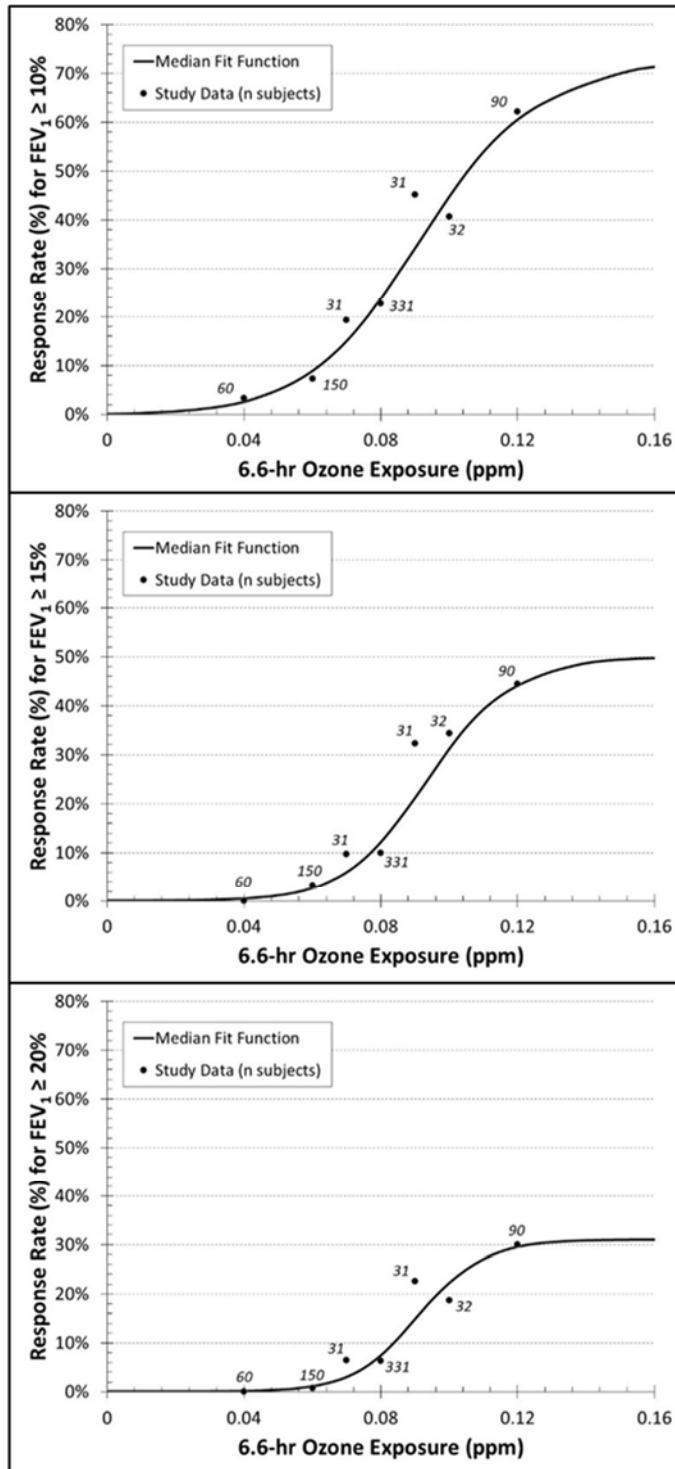
- 20 e_j = (the midpoint of) the j^{th} interval of personal exposure to O₃
- 21 P_j = fraction of the population with O₃ exposures of e_j ppm
- 22 $RR_k | e_j$ = k^{th} response rate at O₃ exposure concentration e_j
- 23 N = number of intervals (categories) of O₃ personal exposure concentration.

24
 25 The number of 0.01 ppm intervals was maximally set to 16 (Figure 3D-12), however,
 26 given the adjusted air quality scenarios, the midpoint values used in the risk calculation typically
 27 ranged from 0.05 to 0.095 ppm. Conventional rounding was applied to the sum of the calculated
 28 risk value.⁷²

⁷¹ Markov chain Monte Carlo (MCMC) simulations require an initial adaptive “burn-in” set of iterations, which are not used as part of the E-R curve output but allow the BMCMC sampling to stabilize.

⁷² For calculated risks (i.e., the summed number of people at each daily maximum 7-hr average exposure interval) where the tenths value was less than 0.5, data were rounded down to the next lowest integer. For calculated risks where the tenths value was greater than or equal to 0.5, data were rounded up to the next highest integer.

O ₃ (ppm)	FEV ₁ ≥10%	FEV ₁ ≥15%	FEV ₁ ≥20%
0	0	0	0
0.005	0.0008	0.0001	0
0.010	0.0019	0.0002	0
0.015	0.0035	0.0004	0.0001
0.020	0.0056	0.0007	0.0001
0.025	0.0084	0.0011	0.0002
0.030	0.0123	0.0018	0.0003
0.035	0.0176	0.0029	0.0006
0.040	0.0249	0.0045	0.0011
0.045	0.0362	0.0070	0.0019
0.050	0.0495	0.0109	0.0033
0.055	0.0665	0.0167	0.0060
0.060	0.0883	0.0260	0.0108
0.065	0.1160	0.0404	0.0180
0.070	0.1497	0.0595	0.0296
0.075	0.1905	0.0860	0.0476
0.080	0.2378	0.1212	0.0738
0.085	0.2894	0.1642	0.1083
0.090	0.3415	0.2115	0.1482
0.095	0.3948	0.2614	0.1879
0.100	0.4474	0.3116	0.2219
0.105	0.4961	0.3560	0.2493
0.110	0.5393	0.3922	0.2704
0.115	0.5756	0.4199	0.2853
0.120	0.6055	0.4408	0.2952
0.125	0.6292	0.4567	0.3012
0.130	0.6477	0.4695	0.3047
0.135	0.6639	0.4789	0.3068
0.140	0.6774	0.4867	0.3082
0.145	0.6893	0.4912	0.3089
0.150	0.6999	0.4941	0.3093
0.155	0.7084	0.4959	0.3096
0.160	0.7133	0.4968	0.3097



1
2 **Figure 3D-12. Median value of Bayesian fit population-based E-R function data (left panel)**
3 **and illustrative curves (right panel) for FEV₁ decrements ≥10% (top panel),**
4 **≥15 (middle panel), ≥20% (bottom panel).** Drawn from the 2014 HREA, Table
5 6A-1 with processing and model development described by Abt (2013).

6

1 From a practical perspective, the population-based E-R function risk approach takes into
2 account that there is a fraction of the population that could experience a lung function decrement
3 at any daily maximum 7-hr average exposure level (i.e., from the minimum to the maximum,
4 including the level of the exposure benchmarks), having a low probability of decrements
5 resulting from low exposures and higher probability at the highest exposures. That said, the
6 approach allows for decrements to occur at exposures below those tested/observed in the
7 controlled human exposure studies, albeit a small population fraction (e.g., see the response
8 frequency for exposures below 60 ppb in Figure 3D-12), recognizing there is potential for
9 variability in the degree of sensitivity between the controlled human exposure study subjects and
10 the simulated population. Note also that because there is a strict limit on attaining a particular
11 ventilation rate for the simulated individuals (i.e., 7-hr average exposures for individuals must
12 simultaneously occur at moderate or greater exertion, section 3D.2.2.3.3), there may be some
13 potential to underestimate lung function responses if they were to occur at the higher end of the
14 exposure distribution (i.e., where exposures are >60 ppb) that coincide with breathing rates just
15 below those specified by the moderate or greater exertion requirement.

16 **3D.2.8.2.2 The McDonnell-Stewart-Smith (MSS) Model**

17 The McDonnell-Stewart-Smith (MSS) model, a statistical model to estimate FEV₁
18 responses for individuals associated with short-term exposures to O₃, was developed using
19 controlled human exposure data⁷³ from studies using varying exposure durations and varying
20 exertion levels and breathing rates (McDonnell et al., 2007). Following the development of the
21 model by McDonnell et al., 2007), Schelegle et al. (2009) found a delay in response when
22 modeling FEV₁ decrements as a function of accumulated dose and estimated a threshold
23 associated with the delay. McDonnell et al. (2012) refit a 2010 version of the model that included
24 a body mass index (BMI) variable (McDonnell et al., 2010), adding data from eight additional
25 studies⁷⁴ and incorporating a threshold parameter into the model, which allows for modeling a
26 delay in response until accumulated dose (i.e., accounting for decreases over time according to
27 first order reaction kinetics) reaches a threshold value. The threshold is not a concentration
28 threshold and does not preclude responses at low concentration exposures.

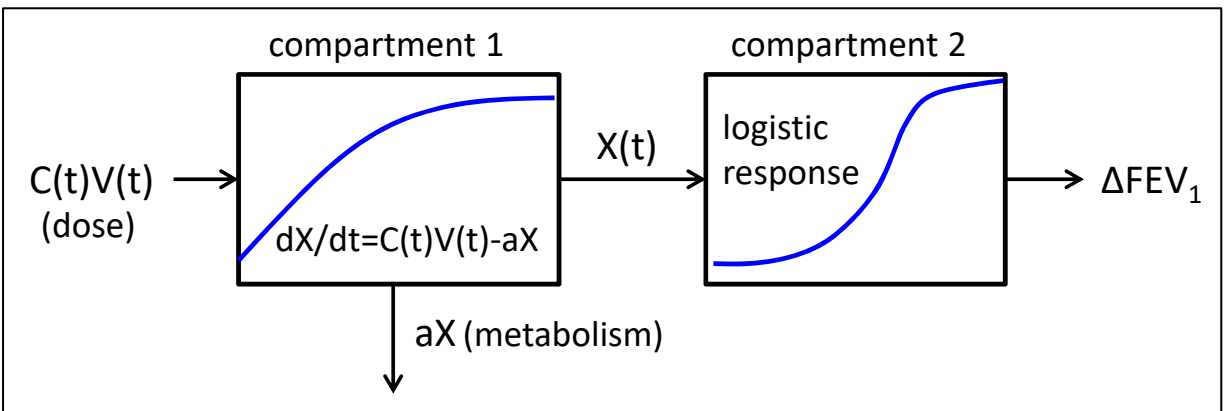
29 The MSS model was first used for estimating lung function risk in the 2014 HREA and
30 was based on the revised version of the model available at that time (McDonnell et al., 2012).
31 Another version of the MSS model has become available since the 2015 review, which differs
32 from the prior model in that it assumes that the intra-subject variance term (Var(ϵ)) increases

⁷³ Data were from 15 controlled human exposure studies that included 531 volunteers (ages 18 to 35), exposed to O₃ on a total of 864 occasions (McDonnell et al., 2007).

⁷⁴ Data from these eight additional studies included 201 individuals.

1 with the response (McDonnell et al., 2013).⁷⁵ Therefore, with a fixed ventilation rate, Var(ϵ) in
 2 this most recent version of the MSS model will be larger for higher exposure concentrations and
 3 smaller for lower exposure concentrations. The most recent version of the MSS model is the
 4 model described here and is the model used in this risk analysis.

5 The lung function model is conceptually a two-compartment model (Figure 3D-13). The
 6 accumulated amount O₃ (exposure concentration × ventilation rate, used to represent dose) is
 7 modeled in the first compartment and modified by an exponential decay factor to yield an
 8 intermediate quantity **X**. The response (FEV₁ reduction) of the individual to **X** is modeled in the
 9 second compartment as a sigmoid-shaped function of the net accumulated dose. A threshold
 10 parameter imposes the constraint that there is no response while the value of **X** is below the
 11 threshold value.



12
 13 **Figure 3D-13. Conceptual representation of the two-compartment model used by the MSS**
 14 **model. *C* is exposure concentration, *V* is ventilation rate, *t* is time, *X* is an**
 15 **intermediate quantity, *a* is a decay constant. Adapted from Figure 1 in**
 16 **McDonnell et al. (1999).**

17
 18 **X** is given by the solution of the differential Equation 3D-12:

19
$$\frac{dX}{dt} = C(t)V(t) - aX \quad \text{Equation 3D-12}$$

20 **X(t)** increases with “normalized dose” ($C \cdot V^{b_6}$) over time for an individual and allows for
 21 removal of “normalized dose” with a half-life of $1/a$ through the 2nd term in Equation 3D-12.

⁷⁵ The MSS model used for the 2014 HREA (McDonnell et al., 2012) assumed intra-subject variability was constant for all exposures and responses. It had been shown previously that FEV₁ response varies within individuals experiencing the same exposure and that the range of variation in response increases with higher exposure and response (McDonnell et al., 1983). Evaluations based on a goodness-of-fit test and visual inspection of observed versus predicted values indicate the most recent MSS model that better accounts for intra-subject variation is improved in its estimation capabilities when compared to the previous MSS model (McDonnell et al., 2013).

1 The response function \mathbf{M} is described in Equation 3D-13:

$$2 \quad \mathbf{M}_{ijk} = (\beta_1 + \beta_2 A_{ik} + \beta_8 B_{ik}) \left\{ \frac{1}{1 + \beta_4 e^{-\beta_3 T_{ijk}}} - \frac{1}{1 + \beta_4} \right\} \quad \text{Equation 3D-13}$$

3 where,

$$4 \quad T_{ijk} = \max\{0, X_{ijk} - \beta_9\} \quad \text{Equation 3D-14}$$

5 β_9 is a threshold parameter which allows \mathbf{X} to increase up to the threshold before the
6 median response is allowed to exceed zero. By construction, when $\mathbf{X} = 0$, then $\mathbf{M} = 0$. Because β_3
7 and β_4 are positive, when $\mathbf{X} > 0$ then $\mathbf{M} > 0$. Because \mathbf{X} is never negative, neither is \mathbf{M} . This
8 model calculates the percent FEV₁ decrement due to O₃ exposure (compartment 2) as:

$$9 \quad \% \Delta FEV_{1ijk} = e^{U_i} M_{ijk} + \varepsilon_{ijk} \quad \text{Equation 3D-15}$$

$$10 \quad Var(\varepsilon_{ijk}) = v_1 + v_2 e^{U_i} M_{ijk} \quad \text{Equation 3D-16}$$

11 Note that a positive value of $\% \Delta FEV_1$ means a decrease in effective lung volume or a
12 decrement in lung function. The above variance structure also allows for negative $\% \Delta FEV_1$
13 values or an increase in lung volume, i.e., an improvement in lung function. The indices i, j, k in
14 Equations 3D-12 to 3D-16 refer to the i^{th} subject at the j^{th} time for the k^{th} exposure protocol for
15 that subject, while the variables are defined as:

16 t = time (minutes)

17 t_0 = time at the start of the event

18 t_1 = time at the end of the event

19 $C(t)$ = O₃ exposure (ppm) at time t during the event

20 $V_E(t)$ = expired minute volume (L min⁻¹) at time t

21 BSA = body surface area (m²),

22 $V(t)$ = $V_E(t)/BSA$ (L/min-m²) at time t

23 A_{ik} = age (years) of the i^{th} subject in the k^{th} exposure protocol minus 23.8, the mean
24 age of the subjects

25 B_{ik} = the body mass index (BMI, kg/m²) of the i^{th} subject in the k^{th} exposure protocol
26 minus 23.1, the mean BMI of the subjects

27 U_i = subject-level zero-mean Gaussian random effect error/variability term (between-
28 individual variability not otherwise captured by the model)

29 ε_{ijk} = Gaussian error/variability term, which includes measurement error and within-
30 individual variability not otherwise captured by the model

$\nu_1, \nu_2 =$ constants used to parameterize the variance of ε_{ijk} . ν_1 captures the intra-individual noise in FEV₁ that is not due to ozone exposure, while ν_2 captures the remaining intra-individual variability in FEV₁.

β_1 to β_9 unitless fitted model parameters (constant for all simulated individuals)

In general, this model would be considered a non-linear random-effects model (Davidian and Giltinan, 2003). The best fit values (based on maximum likelihood) of the β s and the variances $\{\varepsilon_{ijk}\}$ were estimated from fits of the model to the clinical data (see McDonnell et al., 2013) and are provided in Table 3D-21.

Table 3D-21. Estimated coefficients for the MSS lung function model.

Values for MSS Model Coefficients Used in Equations 3D-12 to 3D-17 ^A										
β_1	β_2	β_3	β_4	β_5	β_6	β_8	β_9	ν_1	ν_2	$\text{var}(U)$ ^B
9.763	-0.4315	0.01281	30.92	0.002921	0.9525	0.4890	32.94	9.112	2.166	1.123

^A Based on "Model 3" from McDonnell et al. (2013).
^B The random sampling from the var(U) distribution was limited to ± 2 standard deviations.

As described above in estimating exposure, APEX uses activity pattern data to represent a sequence of events that simulate the movement of a modeled person through geographical locations and microenvironments during the simulation period. Each of these events are defined by a geographic location, start time, duration, microenvironment visited, and activity performed. Events in APEX are intervals of constant activity and exposure concentration, where an individual is in one microenvironment and can range in duration from 1 to 60 minutes. In APEX, because the exposure concentration $C(t)$, exertion level, and normalized ventilation rate $V(t)$ are constant over an event, Equation 3D-17 provides an analytic solution for each event:

$$X(t_1) = X(t_0)e^{-\beta_5(t_1-t_0)} + \frac{C(t_1)}{\beta_5}V(t_1)\beta_6(1 - e^{-\beta_5(t_1-t_0)}) \quad \text{Equation 3D-17}$$

Note that $C(t_1)$ and $V(t_1)$ denote the (constant) values of $C(t)$ and $V(t)$ during the event⁷⁶ from time t_0 to time t_1 . In APEX, values of U_i and ε_{ijk} are drawn from Gaussian distributions with mean zero and variances $\text{var}(U)$ and $\text{var}(\varepsilon)$, constrained to be within ± 2 standard deviations from the means (when sampled values fall outside of this range, they are discarded and resampled).

⁷⁶ Events in APEX are intervals of constant activity and concentration, where an individual is in one microenvironment. Events range in duration from one to 60 minutes. $C(t_1)$ and $V(t_1)$ denote the (constant) values of $C(t)$ and $V(t)$ during the event from time t_0 to time t_1 .

1 The values of U_i are chosen once for each individual and remain constant for individuals
2 throughout the simulation. Values for ε_{ijk} are sampled daily for each individual.

3 We are using this model to estimate lung function decrements for people ages 5 and
4 older. However, this model was developed using only data from individuals aged 18 to 35 and
5 the age adjustment term $[\beta_1 + \beta_2 (Age_{ik} - 23.8)]$ in the numerator of Equation 3D-13 is not
6 appropriate for all ages.⁷⁷ Clinical studies data for children which could be used to fit the model
7 for children are not available at this time. In the absence of data, we are extending the model to
8 ages 5 to 18 by holding the age term constant at the age 18 level. Since the response increases as
9 age decreases in the range 18 to 35, this trend may extend into ages of children, in which case the
10 responses of children could be underestimated. However, the slope of the age term in the MSS
11 model is estimated based on data for ages 18 to 35 and does not capture differences in age trend
12 within this range; in particular, we do not know at what age the response peaks, which could be
13 above or below age 18. The evidence from clinical studies indicates that the responsiveness of
14 children to O_3 is about the same as for young adults (ISA, Appendix 3, section 3.1.4.1.1) This
15 suggests that the age term for children should not be higher than the age term for young adults
16 (2014 HREA).⁷⁸

17 Because the responses to O_3 continuously declines from age 18 to 55 and for ages >55 the
18 response is generally considered minimal,⁷⁹ here we assume the MSS model age term for ages 35
19 to 55 linearly decreases to zero and set it to zero for ages >55 .⁸⁰ To extend the age term to ages
20 outside the range of ages the MSS model is based on (ages 18-35), we re-parameterized the age
21 term in the numerator of Equation 3D-13 by $[\beta_1 + \beta_2(\alpha_1 \text{ Age} + \alpha_2)]$, for different ranges of ages
22 (α_1 and α_2 depend on age), requiring that these terms match at each boundary to form a piecewise
23 linear continuous function of age. As a result, the values of α_1 and α_2 for four age ranges are
24 provided in Table 3D-22.

⁷⁷ Note that the effect of age is also accounted for by using age-specific ventilation rate and body surface area. In addition, APEX lung function risk for different age groups is also influenced by the time spent outdoors and the activities engaged in by those groups, which vary by age.

⁷⁸ See 2014 HREA Chapter 6 (sections 6.4.2 and 6.5.3) and Appendices 6D and 6E for details.

⁷⁹ There is a recent 3-hr controlled human exposure study (EVR = 15-17 L/min-m² during six 15-min exercise periods) performed on healthy adults (ages 59.9 ± 4.5) that found 3-hr O_3 exposures of 120 ppb yielded a statistically significant reduction FEV₁ when compared to the filtered air response (Arjomandi et al., 2018). How this relates to the magnitude and duration of exposures and ventilation rates of interest in this exposure and risk analysis remain uncertain at this time.

⁸⁰ "In healthy individuals, the fastest rate of decline in O_3 responsiveness appears between the ages of 18 and 35 years ... During the middle age period (35-55 years), O_3 sensitivity continues to decline, but at a much lower rate. Beyond this age (>55 years), acute O_3 exposure elicits minimal spirometric changes." (2013 ISA, p. 6-22)

1 **Table 3D-22. Age term parameters for application of the MSS model to all ages.**

Age Range	β_1	β_2	α_1	α_2
5 – 17	9.763	-0.4315	0	-5.8
18 – 35	9.763	-0.4315	1	-23.8
36 – 55	9.763	-0.4315	0.5714	-8.8
> 55	0	-0.4315	0	0
See Table 3D-21 for related MSS model coefficients.				

2
 3 As described above for the population-based E-R function risk approach (section
 4 3D.2.8.2.1), the individual-based MSS model risk approach also allows for decrements to occur
 5 at exposures below those tested/observed in the controlled human exposure studies, however, for
 6 this approach there is not a strict limit on the ventilation *per se*. Indeed, FEV₁ decrements are
 7 more likely to occur with high breathing rates (and concomitant with high exposures), but it is
 8 not necessary that an individual’s 7-hr average EVR reach their particular threshold (EVR
 9 $\geq 17.32 \pm 1.25$ L/min-m²) for an individual to experience an adverse response as is used for both
 10 the exposure benchmarks and the E-R function risk approach. The time-series of exposures,
 11 breathing rate, and FEV₁ will vary with each diary event, with FEV₁ non-linearly dependent on
 12 exposure levels/breathing rate from both the prior and current exposure/breathing events. That
 13 said in doing so, the MSS approach could overstate risk when including instances where both the
 14 exposures and ventilation rates are less than that tested/observed in the controlled human
 15 exposure studies.

16 **3D.2.9 Assessing Variability/Co-Variability and Characterizing Uncertainty**

17 An important issue associated with any population exposure and risk assessment is the
 18 assessment of variability and characterization of uncertainty. Variability refers to the inherent
 19 heterogeneity in a population or variable of interest (e.g., residential air exchange rates). The
 20 degree of variability cannot be reduced through further research, only better characterized with
 21 additional measurement. Uncertainty refers to the lack of knowledge regarding the values of
 22 model input variables (i.e., parameter uncertainty), the physical systems or relationships used
 23 (i.e., use of input variables to estimate exposure or risk or model uncertainty), and in specifying
 24 the scenario that is consistent with purpose of the assessment (i.e., scenario uncertainty).
 25 Uncertainty is, ideally, reduced to the maximum extent possible through improved measurement
 26 of key parameters and iterative model refinement.

27 Section 3D.2.9.1 summarizes how variability and co-variability are addressed in the
 28 current exposure and risk analysis and is based on the above described input data and model
 29 algorithms used. Section 3D.2.9.2 summarizes the overall approach used for the uncertainty

1 characterization. The outcome of the updated uncertainty characterization, which builds upon the
2 important uncertainties identified in the IRP (Appendix 5A) and addressed in this current
3 exposure and risk analyses, is discussed below in section 3D.3.4.

4 **3D.2.9.1 Variability and Co-variability Assessment**

5 The goal in addressing variability in this exposure and risk analysis is to ensure that the
6 estimates of exposure and risk reflect the variability of O₃ concentrations in ambient air,
7 population characteristics, associated O₃ exposures, physiological characteristics of simulated
8 individuals, and potential health risk across the study areas and for the simulated at-risk
9 populations. The details regarding many of the variability distributions used as model inputs are
10 described above, while details regarding the variability addressed within its algorithms and
11 processes are found in the APEX User Guides (U.S. EPA, 2019a, U.S. EPA, 2019b).

12 APEX is designed to account for variability in the model input data, including the
13 physiological variables that are important inputs to determining exertion levels and associated
14 ventilation rates. APEX simulates individuals and then calculates O₃ exposure and lung function
15 risk for each of these simulated individuals. This collection of probabilistically sampled
16 individuals represents the variability of the target population, and by accounting for several types
17 of variability, including demographic, physiological, and human behavior, APEX is able to
18 represent much of the variability in the exposure and risk estimates. For example, variability may
19 arise from differences in the population residing within census tracts (e.g., age distribution) and
20 the activities that may affect population exposure to O₃ (e.g., time spent outdoors, performing
21 moderate or greater exertion level activities outdoors). The range of exposure and associated risk
22 estimates are intended to reflect such sources of variability, although we note that the range of
23 values obtained reflects the input parameters, algorithms, and modeling system used, and may
24 not necessarily reflect the complete range of the true exposure or risk values.

25 We note also that correlations and non-linear relationships between variables input to the
26 model can result in the model producing inaccurate results if the inherent relationships between
27 these variables are not preserved. APEX is designed to account for co-variability, or linear and
28 nonlinear correlation among the model inputs, provided that enough is known about these
29 relationships to specify them. This is accomplished by providing inputs that enable the
30 correlation to be modeled explicitly within APEX. For example, there is a non-linear relationship
31 between the outdoor temperature and air exchange rate in homes. One factor that contributes to
32 this non-linear relationship is that windows tend to be closed more often when temperatures are
33 at either low or high extremes than when temperatures are moderate. This relationship is
34 explicitly modeled in APEX by specifying different probability distributions of air exchange
35 rates for different ambient air temperatures. Note that where possible, we identified and

1 incorporated the observed variability in input data sets rather than employing standard default
 2 assumptions and/or using point estimates to describe model inputs. In any event, APEX models
 3 variability and co-variability in two ways:

- 4 • **Stochastically**. The user provides APEX with probability distributions characterizing the
 5 variability of many input parameters. These are treated stochastically in the model and
 6 the estimated exposure distributions reflect this variability. For example, the rate of O₃
 7 decay in houses can depend on a number of factors which we are not able to explicitly
 8 model at this time, due to a lack of data. However, we can specify a distribution of
 9 removal rates that reflects observed variations in O₃ decay. APEX randomly samples
 10 from this distribution to obtain values that are used in the mass balance model. Further,
 11 co-variability can be modeled stochastically through the use of conditional distributions.
 12 If two or more parameters are related, conditional distributions that depend on the values
 13 of the related parameters are input to APEX. For example, the distribution of air
 14 exchange rates (AERs) in a house depends on the outdoor temperature and whether or not
 15 air conditioning (A/C) is in use. In this case, a set of AER distributions is provided to
 16 APEX for different ranges of temperatures and A/C use, and the selection of the
 17 distribution in APEX is driven by the temperature and A/C status at that time.
- 18 • **Explicitly**. For some variables used in modeling exposure, APEX models variability and
 19 co-variability explicitly and not stochastically. For example, the complete series of hourly
 20 ambient air O₃ concentrations and hourly temperatures are used in the exposure and risk
 21 calculations. These are input to the model continuously in the time period modeled at
 22 different spatial locations, and in this way the variability and co-variability of hourly O₃
 23 concentrations and hourly temperatures are modeled explicitly.

24 Important sources of the variability and co-variability accounted for by APEX and used
 25 for this exposure and risk analysis are provided in Table 3D-23 and Table 3D-24, respectively.

26
 27 **Table 3D-23. Summary of how variability was incorporated into the exposure and risk**
 28 **analysis.**

Component	Variability Source	Summary
Ambient Air Concentration Input (Appendix 3C)	CAMx Air Quality Modeling	Spatial: model results are output at 12 km spatial resolution for the full CONUS domain. Temporal: model results are calculated and archived at hourly resolution for the full 2016 calendar year.
	CAMx/HDDM estimates of 1-hr ambient air O ₃ concentrations	Spatial: simulations of O ₃ response to changes in emissions predicted to multiple monitors in eight geographically representative study areas. Temporal: hourly O ₃ for each of three years (2015-2017).
	Ambient air monitor hourly concentrations	Spatial: local ambient air monitor sites used to interpolate adjusted O ₃ concentrations to census tracts, including monitors outside of the study area. Temporal: pattern of hourly O ₃ concentrations at census tracts also informed by local ambient air monitors.

Component	Variability Source	Summary
Simulated Individuals	Population data	Individuals are randomly sampled from U.S. census tracts used in each study area, stratified by age (single years) and sex probabilities (U.S. Census Bureau, 2012).
	Employment	Work status is randomly generated from U.S. census tracts, stratified by age and sex employment probabilities (U.S. Census Bureau, 2012).
	Activity pattern data	Data diaries used to represent locations visited and activities performed by simulated individuals are randomly selected from CHAD (nearly 180,000 diaries) using six diary pools stratified by two day-types (weekday, weekend) and three temperature ranges (< 55.0 °F, between 55.0 and 83.9 °F, and ≥84.0 °F). CHAD diaries capture real locations that people visit and the activities they perform, ranging from 1-min to 1-hr in duration (U.S. EPA, 2019c).
	Commuting data	Employed individuals are probabilistically assigned ambient air concentrations originating from either their home or work block based on U.S. Census derived tract-level commuter data (U.S. DOT, 2012; U.S. Census Bureau, 2012).
	Longitudinal profiles	A sequence of diaries is linked together for each individual that preserves both the inter- and intra-personal variability in human activities (Glen et al., 2008).
	Asthma prevalence	Asthma prevalence is stratified by sex, single age years for children (5-17), seven adult age groups, (18-24, 25-34, 35-44, 45-54, 55-64, 65-74, and, ≥75), three regions (Midwest, Northeast, and South), and U.S. Census tract level poverty ratios (Attachment 1).
Physiological Factors Relevant to Ventilation Rate	Resting metabolic rate	Five age-group and two sex-specific regression equations, use body mass and age as independent variables (U.S. EPA (2018), Appendix H).
	Metabolic equivalents by activity	Randomly sampled from distributions developed for specific activities (some age-specific) (U.S. EPA, 2019c)
	Oxygen uptake per unit of energy expended	Randomly sampled from a uniform distribution to convert energy expenditure to oxygen consumption (U.S. EPA, 2019a, U.S. EPA, 2019b).
	Body mass	Randomly selected from population-weighted lognormal distributions with age- and sex-specific geometric mean (GM) and geometric standard deviation (GSD) derived from the National Health and Nutrition Examination Survey (NHANES) for the years 2009-2014 (U.S. EPA (2018), Appendix G).
	Body surface area	Sex-specific exponential equations using body mass as an independent variable (Burmester, 1998).
	Height	Randomly sampled from population-weighted normal distributions stratified by single age years and two sexes developed from 2009-2014 NHANES data (U.S. EPA (2018), Appendix G).
	Ventilation rate	Event-level activity-specific regression equation using oxygen consumption rate (VO ₂) and maximum VO ₂ as independent variables, and accounting for intra- and inter-personal variability (U.S. EPA (2018), Appendix H).
	Fatigue and EPOC	APEX approximates the onset of fatigue, controlling for unrealistic or excessive exercise events in an individual's activity time-series while also estimating excess post-exercise oxygen consumption (EPOC) that may occur following vigorous exertion activities using several equations and

Component	Variability Source	Summary
		input variable distributions (Isaacs et al., 2007; U.S. EPA, 2019a; U.S. EPA, 2019b).
	Equivalent ventilation rate	A randomly sampled value is selected for each simulated individual from a normal distribution derived from the controlled human exposure study data. This approach accounts for interpersonal variability in exertion level that occur during exposure events that include exercise and rest periods (Attachment 2).
Microenvironmental Approach	General	Seven total microenvironments are represented, including those expected to be associated with high exposure concentrations (i.e., outdoors and outdoor near-road). There is variability in particular microenvironmental algorithm inputs. This results in differential exposures for each individual (and event) because people spend varying amounts of time within each microenvironment and ambient air concentrations vary within and among study areas.
	Spatial Variability	Ambient air concentrations used in microenvironmental algorithms vary spatially within (i.e., census tracts) and among study areas (U.S. geographic regions).
	Temporal Variability	All exposure calculations are performed at the event-level when using either factors or mass balance approach (durations can be as short as one minute). For the indoor microenvironments, using a mass balance model accounts for O ₃ concentrations occurring during a previous hour (and of ambient air origin) to calculate a current event's indoor O ₃ concentrations.
	Air exchange rates	For residences, several lognormal distributions are sampled for up to five daily mean temperature ranges, study area region (2014 HREA Appendix 5E) and using study-area specific A/C prevalence rates from AHS survey data (U.S. Census Bureau, 2019). For restaurants, a lognormal distribution is sampled based on Bennett et al. (2012). For schools, a Weibull distribution is sampled based on data from Lagus Applied Technology (1995), Shendell et al. (2004), and Turk et al. (1989).
	Removal rates	Values randomly selected from a lognormal distribution for the three indoor microenvironments modeled (Lee et al., 1999).
	PE and PROX factors	Penetration and proximity factors randomly sampled from probability distributions for inside-vehicle and near-road microenvironments (American Petroleum Institute (1997), Appendix B; Johnson et al., 1995).
Lung Function Risk	Population-based Exposure Response Function	A continuous E-R function was derived using data from several controlled human exposure studies and a logit-linear modeling approach. The full distribution of population exposures was stratified by fine-scale bins (10 ppb) and linked to the continuous E-R function to estimate lung function risk.
	Individual-based MSS model	Calculation accounts for variability in age, body mass, and the continuous time-series of exposures and breathing rates. Residual terms (U and) addresses intra- and inter-variability in responses across the simulated population.

1
2

1 **Table 3D-24. Important components of co-variability in exposure modeling.**

Type of Co-variability	Modeled by APEX?	Treatment in APEX / Comments
Within-person correlations ^A	Yes	Sequence of activities performed, microenvironments visited, and general physiological parameters (body mass, height, ventilation rates).
Between-person correlations	No	Perhaps not important, assuming the same likelihood of the population of individuals either avoiding or experiencing an exposure event based on a social (group) activity.
Correlations between profile variables and microenvironment parameters	Yes	Profiles are assigned microenvironment parameters.
Correlations between demographic variables and activities	Yes	Census tract demographic variables, appropriately weighted and stratified by age and sex, are used in activity diary selection.
Correlations between activities and microenvironment parameters	No	Perhaps important, but do not have data. For example, frequency of opening windows when cooking or smoking tobacco products.
Correlations among microenvironment parameters in the same microenvironment	Yes	Modeled with joint conditional variables.
Correlations between demographic variables and air quality	Yes	Modeled with the spatially varying census tract demographic variables (age and sex) and census tract air quality data input to APEX.
Correlations between meteorological variables and activities	Yes	Daily varying temperatures are used in activity diary selection.
Correlations between meteorological variables and microenvironment parameters	Yes	The distributions of microenvironment parameters can be functions of temperature.
Correlations between drive times in CHAD and commute distances traveled	Yes	CHAD diary selection is weighted by commute times for employed persons during weekdays.
Consistency of occupation/school microenvironmental time and time spent commuting/busing for individuals from one working/school day to the next.	No	Simulated individuals are assigned activity diaries longitudinally without regard to occupation or school schedule (note though, longitudinal variable used to develop annual profile is time spent outdoors).
^A The term correlation is used to represent linear and nonlinear relationships.		

2

3 **3D.2.9.2 Approach for Uncertainty Characterization**

4 While it may be possible to capture a range of exposure or risk values by accounting for
5 variability inherent to influential factors, the true exposure or risk for any given individual within
6 a study area may be unknown, although it can be estimated. To characterize health risks,
7 exposure and risk assessors commonly use an iterative process of gathering data, developing
8 models, estimating exposures and risks, evaluating results for correctness and identifying areas
9 for potential improvement, given the goals of the assessment, scale and complexity of the
10 assessment performed, and limitations of the input data available. However, important

1 uncertainties often remain in any one of the data sets, tools, and approaches used and emphasis is
2 then placed on characterizing the nature of that uncertainty and its impact on exposure and risk
3 estimates.

4 The overall approach used for this exposure and risk generally follows that described by
5 WHO (2008) but varies in that a greater focus has been placed on evaluating the direction and
6 the magnitude of the uncertainty. This refers to qualitatively rating how the source of
7 uncertainty, in the presence of alternative information, may affect the estimated exposures and
8 health risk results. Following the identification of key uncertainties, we subjectively scale the
9 overall impact of the identified uncertainty by considering the relationship between the source of
10 uncertainty and the exposure concentrations (e.g., low, medium, or high potential impact). Also
11 to the extent possible, we include an assessment of the direction of influence, indicating how the
12 source of uncertainty may be affecting exposure or risk estimates (e.g., the uncertainty could lead
13 to over-estimates, under-estimates, or both directions). Further, and consistent with the WHO
14 (2008) guidance, we discuss the uncertainty in the knowledge-base (e.g., the accuracy of the data
15 used, recognition of data gaps) and, where possible, particular assessment design decisions (e.g.,
16 selection of particular model forms). The output of the uncertainty characterization is a summary
17 that describes, for each identified source of uncertainty, the magnitude of the impact and the
18 direction of influence the uncertainty may have on the exposure and risk results.

19 We further recognize that uncertainties associated with APEX exposure modeling have
20 been previously characterized in the REAs for nitrogen dioxide (NO₂), carbon monoxide (CO)
21 and sulfur dioxide (SO₂) conducted for recent primary NAAQS reviews, along with other
22 pollutant-specific issues (U.S. EPA, 2008, 2010, 2014, 2018), all complementary to quantitative
23 uncertainty characterizations conducted for the 2007 O₃ exposure assessment by Langstaff
24 (2007). Conclusions drawn from each of these characterizations are also considered here in light
25 of new information, data, tools, and approaches used in this exposure and risk analysis.

26 **3D.3 POPULATION EXPOSURE AND RISK RESULTS**

27 Exposure and risk results are presented here for simulated populations residing in the
28 eight study areas – Atlanta, Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento, and St.
29 Louis – for a three-year air quality scenario in which air quality conditions just meet the current
30 primary 8-hr O₃ standard (70 ppb, annual 4th highest daily maximum 8-hr average concentration,
31 averaged across 3-years) and two other air quality scenarios (i.e., design values of 75 and 65
32 ppb). Hourly concentrations of O₃ in ambient air for the three hypothetical air quality scenarios
33 are estimated at census tracts in each study area as described in section 3D.2 above. Population
34 exposure and risk associated with these concentrations is estimated using the APEX model
35 simulations (section 3D.2) and is briefly described with the following.

1 APEX uses the hourly air quality surface in each study area, along with U.S. census tract
2 population demographics, to estimate the number of days per year each simulated individual in a
3 particular study area experiences a daily maximum 7-hr average O₃ exposure at or above
4 benchmark levels of 60, 70, and 80 ppb (section 3D.2.8.1). These short-term exposures were
5 evaluated for children (5-18 years old), adults (>18 years old), and those with asthma within each
6 of these two study groups when the exposure corresponded with moderate or greater exertion
7 (i.e., the individual's EVR $\geq 17.32 \pm 1.25$ L/minute-m²).

8 Then, individuals expected to experience a lung function decrement (i.e., reduction in
9 FEV₁ $\geq 10\%$, $\geq 15\%$, $\geq 20\%$) were estimated using two approaches. The first approach linked the
10 population-based daily maximum 7-hr exposures while at moderate or greater exertion with an
11 exposure-response function derived from controlled human exposure study data (section
12 3D.2.8.2.1). The second lung function risk approach, considered an individual-based approach
13 here, used the McDonnell-Stewart-Smith (MSS) FEV₁ model (McDonnell et al., 2013) (section
14 3D.2.8.2.2). The MSS uses the time-series of O₃ exposure and corresponding ventilation rates for
15 each APEX simulated individual to estimate their personal time-series of FEV₁ reductions,
16 selecting the daily maximum reduction for each person. The number of individuals estimated to
17 experience decrements are then aggregated to the population level. Again, of interest for both of
18 these lung function risk approaches is the number of days per year each simulated individual in a
19 particular study area experiences a lung function decrement.

20 Study area characteristics and the composition of the simulated population are provided
21 in section 3D.3.1. Exposure results are presented in a series of tables that allow for simultaneous
22 comparison of the exposure and risk metrics across the eight study areas and three simulation
23 years. Two types of results are provided for each study area: the percent (and number) of the
24 simulated population exposed at or above selected benchmarks, stratified by the number of
25 occurrences (i.e., days) in a year (section 3D.3.2) and the percent (and number) of the simulated
26 population experiencing a reduction in FEV₁ $\geq 10\%$, $\geq 15\%$, $\geq 20\%$, also stratified by the number
27 of days in a year (section 3D.3.3). Tables summarizing all of the exposure and risk results for
28 each study area are provided in Attachment 4.

29 **3D.3.1 Characteristics of the Simulated Population and Study Areas**

30 The eight study areas differ in population, geographic size, and demographic features
31 (Table 3D-25). In each of the eight study areas, APEX simulated O₃ exposures and risks for
32 60,000 individuals,⁸¹ the demographic features of which were based on the information

⁸¹ While precisely 60,000 children and 60,000 adults were simulated as part of each APEX model run, the number of individuals estimated to be exposed are appropriately weighted to reflect the actual population residing within the census tracts that comprise each respective study area.

1 associated with the hundreds to thousands of census tracts within each area (as described in
2 section 3D.2.1 above).

3 Asthma prevalence in each modeling domain was estimated based on the 2013-2017
4 NHIS asthma prevalence data and the demographic characteristics for each study area (e.g., age,
5 sex and family income) using the methodology summarized in section 3D.2.2.2. Accordingly,
6 the percent of the simulated populations with asthma within the exposure modeling domain
7 varied by study area (Table 3D-25). The Dallas, Phoenix, and Sacramento study areas had the
8 lowest percent of children with asthma (9.2 to 9.6%), while Atlanta and Boston had the highest
9 percent of children with asthma (11.8 to 12.3%). The Dallas study area had the lowest percent of
10 adults with asthma (7.2%), while Boston and Detroit had the highest percent of adults with
11 asthma (both 10.9%). The statistics presented here are the aggregate of the study area as a whole,
12 within which asthma prevalence varied widely as the modeling approach fully accounted for the
13 variation in asthma prevalence across census tracts with demographic factors such as family
14 income to poverty ratios, age, and sex (and as described in section 3D.2.2.2).⁸² Nationally,
15 asthma prevalence is 7.9%; for children it is 8.4% and for adults it is 7.7% (Chapter 3, Table 3-
16 1). The asthma prevalence for children, adults, and the total population estimated for each of the
17 eight study areas are all greater than that of the national asthma prevalence, except for adults in
18 Dallas which has a slightly lower asthma prevalence. This suggests that overall, the at-risk
19 population simulated in the eight study areas could represent at-risk populations in other U.S.
20 urban areas that have a similarly above average asthma prevalence.

⁸² Representing the variation in asthma prevalence that occurs at the census tract level provides a level of resolution for identification of at-risk individuals that is directly compatible with the resolution of the spatially varying ambient air concentrations. In this way, the population in census tracts with higher concentrations is represented appropriately with regard to asthma prevalence and exposures of the at-risk individuals with asthma are not under-represented.

1 **Table 3D-25. Summary of study area features and the simulated population.**

Study Area (Land Area – km ²) ^A	Population Group (age range)	Simulated Population	Simulated Population with Asthma	% of Simulated Population with Asthma
Atlanta (30,655)	Children (5-18)	1,210,594	142,400	11.8
	Adults (19-90)	4,226,009	359,375	8.5
	All (5-90)	5,436,603	501,775	9.2
Boston (25,117)	Children (5-18)	1,365,267	167,617	12.3
	Adults (19-90)	5,870,125	642,224	10.9
	All (5-90)	7,235,392	809,841	11.2
Dallas (42,664)	Children (5-18)	1,418,728	130,421	9.2
	Adults (19-90)	4,688,180	336,898	7.2
	All (5-90)	6,106,908	467,319	7.7
Detroit (16,884)	Children (5-18)	1,040,588	116,899	11.2
	Adults (19-90)	3,932,484	427,221	10.9
	All (5-90)	4,973,072	544,119	10.9
Philadelphia (18,959)	Children (5-18)	1,309,547	146,982	11.2
	Adults (19-90)	5,228,541	503,305	9.6
	All (5-90)	6,538,088	650,287	9.9
Phoenix (34,799)	Children (5-18)	849,200	81,396	9.6
	Adults (19-90)	2,980,062	269,845	9.1
	All (5-90)	3,829,262	351,240	9.2
Sacramento (18,871)	Children (5-18)	465,845	45,208	9.7
	Adults (19-90)	1,715,065	138,253	8.1
	All (5-90)	2,180,910	183,461	8.4
St. Louis (23,019)	Children (5-18)	546,393	56,039	10.3
	Adults (19-90)	2,146,037	203,039	9.5
	All (5-90)	2,692,430	259,078	9.6
All Study Areas Combined	Children (5-18)	8,206,162	886,960	10.8
	Adults (19-90)	30,786,503	2,880,160	9.4
	All (5-90)	38,992,665	3,767,120	9.7

^A From Appendix 3C, Table 3C-1.

2 **3D.3.2 Exposures at or above Benchmark Concentrations**

3 The exposure to benchmark comparisons are presented in a series of tables focusing on
 4 the benchmark levels (i.e., people experiencing daily maximum 7-hr average O₃ exposures ≥60,
 5 70, and 80 ppb while at moderate or greater exertion). The full range of ambient air O₃
 6 concentrations for a 3-year O₃ season (2015-2017) were used by APEX, providing a range of
 7 estimated exposures. Adjusted air quality surfaces used to represent three air quality scenarios
 8 were developed using 2015-2017 design values modeled sensitivities to changes in precursor
 9 emissions (section 3D.2.3.3), and then interpolated to census tract centroids (section 3D.2.3.4).

1 Exposures were estimated for four study groups of interest (i.e., school-age children (5-18),
2 school-age children with asthma, adults (19-90), and adults with asthma).

3 In this exposure and risk analysis, we are primarily interested in O₃ exposures associated
4 with the ambient air quality adjusted to just meet the current standard (70 ppb, annual 4th highest
5 daily maximum 8-hr average concentration, averaged over a 3-year period). Provided are the
6 percent and number of people in each study group estimated to experience 7-hr exposures at or
7 above the benchmarks, while at moderate or greater exertion (section 3D.3.2.1). For each
8 exposure metric and study group, the occurrence of single-day (at least one day per year) and
9 multi-day (at least 2, 4, or 6 days per year) exposures are presented. Exposure results for the two
10 other adjusted air quality scenarios (the 75 ppb and 65 ppb scenarios) are presented in sections
11 3D.3.2.2 and 3D.3.2.3, respectively. These two sections present only the percent of each study
12 group estimated to experience exposures at or above benchmarks while at moderate or greater
13 exertion, for single-day and multiday exposures during a year, and not also the number of
14 simulated individuals in each study group. The complete exposure results associated with all
15 simulated years, air quality scenarios, the four study groups, and eight study areas are found in
16 Attachment 4.

17 In general, and for all air quality scenarios, the percent of children estimated to
18 experience exposures at or above any of the benchmarks is consistently higher than that
19 estimated for adults. This is expected because children spend a greater amount of time outdoors,
20 and at a greater frequency, while at moderate or greater exertion when compared to adults (2014
21 HREA, sections 5.4.1 and 5.4.2). Estimated exposures for healthy people are similar to people
22 with asthma when considered on a percent of population basis. This is because similar diary data
23 are used to simulate the activity patterns of each study group, justified by evaluations that
24 indicated similarities in time spent outdoors, participation rate, and exertion level for people with
25 asthma when compared to healthy individuals (section 3D.2.5.3). When considering the
26 estimated exposures in terms of population counts, while children comprise about 20% of the
27 simulated population (Table 3D-25), the number of children experiencing exposures at or above
28 the benchmarks is greater than that of adults. Again, this a direct result of the differences in time
29 spent outdoors performing activities at elevated exertion. And finally, Detroit, Phoenix, and St.
30 Louis have a higher percent of individuals at or above benchmark levels relative to the other
31 study areas, likely influenced by their having an hourly O₃ concentration distribution shape that,
32 overall, is more skewed to the right and/or has heavy tails at the uppermost percentiles (Figure
33 3D-7).

3D.3.2.1 Air Quality Just Meeting the Current Standard

With air quality adjusted to just meet the current standard, 0 to $\leq 0.1\%$ of people in all study groups were estimated to experience at least one daily maximum 7-hr exposure per year at or above the 80-ppb benchmark (Table 3D-26). The occurrence of 7-hr O₃ exposures at or above 70-ppb are also limited, even considering the worst year air quality in the three-year period, with 1% or fewer children (and children with asthma) in all study areas estimated to experience at least one daily maximum 7-hr exposure per year at or above the 70-ppb benchmark. For the same benchmark, 0.2% or fewer adults (and adults with asthma) were estimated to experience similar exposures when considering the worst air quality year. When considering the 60-ppb benchmark, on average, between about 3 to 9% of children (and children with asthma) experienced at least one daily maximum 7-hr exposure at or above that benchmark, while during the worst air quality year, the range in percent of children exposed extends slightly upwards (about 4 to 11%), indicating limited variability in ambient air concentrations across the three-year period. Again, there were fewer adults (and adults with asthma) exposed considering this same benchmark, on average ranging from 0.2 to 1.5% of this study group and the worst air quality year ranging from 0.2 to 1.8%.

The number of simulated people in each study group estimated to experience at least one 7-hr exposure per year at or above the benchmarks is provided in Table 3D-27. As noted above, there are few simulated people expected to experience a 7-hr exposure at or above the 80-ppb benchmark, at most about 1,200 children and 500 adults when considering the worst year in a single study area. Regarding the 70-ppb benchmark, on average, between about 700 to 8,300 children are estimated to experience at least one 7-hr exposure at or above that benchmark, while the range for adults is about half that of children (400 to about 3,700), the range of which considers the eight study areas. When considering the worst year, fewer than 12,000 children and 7,700 adults are estimated to experience at least one 7-hr exposure at or above the 70-ppb benchmark in each study area. On average, the number of children estimated to experience at least one 7-hr O₃ exposure at or above the 60-ppb benchmark could be as high as nearly 70,000 in a few study areas, while for adults the number is just below 45,000. During the worst air quality year, the estimated number of people experiencing at least one exposure at or above this same benchmark could be as high as about 100,000 for children and 63,000 for adults. As a whole, the patterns for people with asthma are similar though having smaller counts, the value of which is dictated by the asthma prevalence in each area (Table 3D-25). In general, the number of children with asthma at or above a benchmark would be about 10.8% of that estimated for all children, while the number adults with asthma at or above a benchmark is about 9.4% of that estimated for all adults.

1 Multiday exposures are limited when considering air quality adjusted to just meet the
2 current standard. For example, there are no children estimated to experience at least two days
3 with 7-hr O₃ exposures at or above the 80-ppb benchmark and ≤0.1% at or above the 70-ppb
4 benchmark (Table 3D-28 and Table 3D-29). When considering the worst air quality year, <5% of
5 children (and ≤0.4% of adults) are estimated to experience at least two days with 7-hr O₃
6 exposures at or above the 60-ppb benchmark. There are no people estimated to experience at
7 least four days with 7-hr O₃ exposures at or above the 70-ppb benchmark except in one study
8 area (Table 3D-30 and Table 3D-31), and ≤0.5% experience at least six days with 7-hr O₃
9 exposures at or above the 60-ppb benchmark (Attachment 4).
10

1 **Table 3D-26. Percent of people estimated to experience at least one exposure at or above**
 2 **benchmarks while at moderate or greater exertion, for air quality adjusted to**
 3 **just meet the current standard.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	3.3	1.4	5.2	0.4	0.1	0.8	<0.1	0	0.1
	Boston	4.4	3.4	6.0	0.6	0.4	0.9	<0.1	<0.1	<0.1
	Dallas	4.9	2.4	6.8	0.4	0.2	0.7	<0.1	0	<0.1
	Detroit	6.7	5.0	9.2	0.5	0.1	0.9	<0.1	0	<0.1
	Philadelphia	4.1	3.9	4.2	0.4	0.3	0.4	<0.1	0	<0.1
	Phoenix	8.2	6.0	10.6	0.2	<0.1	0.6	0	0	0
	Sacramento	3.2	2.3	3.9	0.2	0.1	0.3	0	0	0
St. Louis	6.0	4.1	8.7	0.4	0.1	0.9	<0.1	0	<0.1	
Children with Asthma	Atlanta	3.6	1.5	5.8	0.5	0.1	0.9	<0.1	0	0.1
	Boston	5.1	3.7	7.0	0.7	0.5	1.0	<0.1	0	0.1
	Dallas	5.3	2.2	7.4	0.4	0.3	0.7	<0.1	0	<0.1
	Detroit	7.3	5.4	10.0	0.5	0.1	0.9	0	0	0
	Philadelphia	4.3	4.1	4.4	0.4	0.4	0.4	0	0	0
	Phoenix	8.8	6.6	11.2	0.3	0	0.7	0	0	0
	Sacramento	3.3	2.6	4.0	0.2	0.1	0.3	0	0	0
St. Louis	6.0	3.9	9.0	0.3	<0.1	0.8	<0.1	0	<0.1	
Adults	Atlanta	0.5	0.2	0.8	0.1	<0.1	0.1	<0.1	0	<0.1
	Boston	0.5	0.3	0.8	0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.8	0.3	1.2	<0.1	<0.1	0.1	<0.1	0	<0.1
	Detroit	1.0	0.8	1.6	0.1	<0.1	0.2	0	0	0
	Philadelphia	0.5	0.5	0.5	<0.1	<0.1	<0.1	<0.1	0	<0.1
	Phoenix	1.5	1.1	1.8	<0.1	<0.1	0.1	0	0	0
	Sacramento	0.4	0.3	0.5	<0.1	<0.1	<0.1	0	0	0
St. Louis	0.9	0.5	1.3	<0.1	<0.1	0.1	0	0	0	
Adults with Asthma	Atlanta	0.4	0.2	0.6	<0.1	0	<0.1	0	0	0
	Boston	0.4	0.2	0.7	<0.1	0	0.1	<0.1	0	<0.1
	Dallas	0.6	0.2	0.9	<0.1	0	0.1	0	0	0
	Detroit	0.8	0.6	1.2	0.1	<0.1	0.2	0	0	0
	Philadelphia	0.4	0.3	0.5	<0.1	0	0.1	0	0	0
	Phoenix	1.3	1.0	1.5	<0.1	0	<0.1	0	0	0
	Sacramento	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
St. Louis	0.7	0.4	1.2	<0.1	0	0.1	0	0	0	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **Table 3D-27. Number of people estimated to experience at least one exposure at or above**
 2 **benchmarks while at moderate or greater exertion, for air quality adjusted to**
 3 **just meet the current standard.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (# per Year)			70 ppb Benchmark (7-hr) ^A (# per Year)			80 ppb Benchmark (7-hr) ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	39909	17291	63455	5199	1069	9947	464	0	1211
	Boston	59549	46465	81939	8305	5438	11923	372	91	592
	Dallas	69794	34499	96261	5864	3168	9718	173	0	284
	Detroit	69627	52203	95509	5093	1492	9487	29	0	52
	Philadelphia	53117	51116	54674	4656	4191	5151	44	0	87
	Phoenix	69569	50754	89775	1953	269	4784	0	0	0
	Sacramento	14928	10645	18378	727	272	1203	0	0	0
	St. Louis	32841	22320	47609	2331	446	4863	12	0	36
Children with Asthma	Atlanta	5152	2078	8333	666	141	1271	67	0	202
	Boston	8518	6166	11605	1145	796	1616	61	0	114
	Dallas	6952	2908	9813	576	355	946	8	0	24
	Detroit	8544	6209	11776	578	121	1110	0	0	0
	Philadelphia	6264	6024	6504	597	524	655	0	0	0
	Phoenix	7171	5336	9143	226	0	552	0	0	0
	Sacramento	1517	1157	1871	93	54	155	0	0	0
	St. Louis	3364	2195	4927	191	18	437	3	0	9
Adults	Atlanta	21318	9790	34160	2512	282	5001	117	0	352
	Boston	30362	19274	48429	3391	2152	5283	294	98	489
	Dallas	36646	14611	54461	2318	1328	4141	26	0	78
	Detroit	40920	30215	62264	3692	1049	7668	0	0	0
	Philadelphia	26375	25184	27973	1597	1481	1830	29	0	87
	Phoenix	44552	33178	54585	745	149	1788	0	0	0
	Sacramento	7318	4688	9176	400	229	600	0	0	0
	St. Louis	18981	11016	28185	942	72	2075	0	0	0
Adults with Asthma	Atlanta	1385	775	2113	70	0	141	0	0	0
	Boston	2544	1370	4207	294	0	685	65	0	98
	Dallas	2109	781	3047	104	0	234	0	0	0
	Detroit	3299	2425	5047	306	66	655	0	0	0
	Philadelphia	2179	1569	2614	87	0	261	0	0	0
	Phoenix	3377	2831	3973	50	0	99	0	0	0
	Sacramento	295	257	343	38	29	57	0	0	0
	St. Louis	1395	787	2325	72	0	179	0	0	0

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals exposed at the level).

1 **Table 3D-28. Percent of people estimated to experience at least two exposures at or above**
 2 **benchmarks while at moderate or greater exertion, for air quality adjusted to**
 3 **just meet the current standard.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	0.6	0.1	1.1	<0.1	0	<0.1	0	0	0
	Boston	0.8	0.5	1.4	<0.1	<0.1	<0.1	0	0	0
	Dallas	1.2	0.4	2.1	<0.1	<0.1	0.1	0	0	0
	Detroit	1.7	1.0	2.8	<0.1	<0.1	0.1	0	0	0
	Philadelphia	0.8	0.7	0.9	<0.1	<0.1	<0.1	0	0	0
	Phoenix	2.9	1.7	4.3	<0.1	0	<0.1	0	0	0
	Sacramento	0.6	0.3	0.9	<0.1	0	<0.1	0	0	0
	St. Louis	1.5	0.7	2.6	<0.1	<0.1	<0.1	0	0	0
Children with Asthma	Atlanta	0.7	0.1	1.2	<0.1	0	<0.1	0	0	0
	Boston	1.0	0.6	1.6	<0.1	0	<0.1	0	0	0
	Dallas	1.2	0.3	2.2	<0.1	0	0.1	0	0	0
	Detroit	1.9	1.1	2.9	<0.1	0	<0.1	0	0	0
	Philadelphia	0.9	0.8	0.9	<0.1	0	<0.1	0	0	0
	Phoenix	3.2	1.8	4.9	<0.1	0	0.1	0	0	0
	Sacramento	0.6	0.4	0.9	<0.1	0	<0.1	0	0	0
	St. Louis	1.3	0.6	2.2	<0.1	0	<0.1	0	0	0
Adults	Atlanta	<0.1	<0.1	0.1	0	0	0	0	0	0
	Boston	<0.1	<0.1	0.1	<0.1	0	<0.1	0	0	0
	Dallas	0.1	<0.1	0.1	<0.1	0	<0.1	0	0	0
	Detroit	0.1	0.1	0.2	<0.1	0	<0.1	0	0	0
	Philadelphia	<0.1	<0.1	<0.1	<0.1	0	<0.1	0	0	0
	Phoenix	0.3	0.2	0.4	<0.1	0	<0.1	0	0	0
	Sacramento	<0.1	<0.1	0.1	<0.1	0	<0.1	0	0	0
	St. Louis	0.1	<0.1	0.2	0	0	0	0	0	0
Adults with Asthma	Atlanta	<0.1	0	0.1	0	0	0	0	0	0
	Boston	<0.1	0	0.1	<0.1	0	<0.1	0	0	0
	Dallas	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Detroit	0.1	<0.1	0.1	0	0	0	0	0	0
	Philadelphia	<0.1	0	<0.1	0	0	0	0	0	0
	Phoenix	0.3	0.1	0.4	<0.1	0	<0.1	0	0	0
	Sacramento	<0.1	0	0.1	0	0	0	0	0	0
	St. Louis	0.1	<0.1	0.2	0	0	0	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4
5

1 **Table 3D-29. Number of people estimated to experience at least two exposures at or above**
 2 **benchmarks while at moderate or greater exertion, for air quality adjusted to**
 3 **just meet the current standard.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (# per Year)			70 ppb Benchmark (7-hr) ^A (# per Year)			80 ppb Benchmark (7-hr) ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	7365	1675	13801	155	0	282	0	0	0
	Boston	11317	6690	18477	341	91	660	0	0	0
	Dallas	17135	5226	29273	276	24	757	0	0	0
	Detroit	17829	10805	28894	243	69	520	0	0	0
	Philadelphia	10142	9210	11764	124	65	175	0	0	0
	Phoenix	24952	14153	36643	94	0	269	0	0	0
	Sacramento	2601	1281	4278	16	0	31	0	0	0
	St. Louis	8305	4071	14325	67	9	155	0	0	0
Children with Asthma	Atlanta	1002	202	1715	20	0	40	0	0	0
	Boston	1669	1047	2617	30	0	68	0	0	0
	Dallas	1600	378	2861	39	0	118	0	0	0
	Detroit	2180	1301	3469	11	0	17	0	0	0
	Philadelphia	1288	1113	1375	15	0	44	0	0	0
	Phoenix	2609	1444	3977	24	0	71	0	0	0
	Sacramento	282	179	396	5	0	8	0	0	0
	St. Louis	713	337	1211	3	0	9	0	0	0
Adults	Atlanta	1925	211	3592	0	0	0	0	0	0
	Boston	2446	1076	4794	98	0	196	0	0	0
	Dallas	3724	1250	6798	26	0	78	0	0	0
	Detroit	5178	2884	9438	44	0	131	0	0	0
	Philadelphia	1917	1656	2266	29	0	87	0	0	0
	Phoenix	8361	4718	11324	33	0	50	0	0	0
	Sacramento	572	257	972	10	0	29	0	0	0
	St. Louis	2587	858	4435	0	0	0	0	0	0
Adults with Asthma	Atlanta	94	0	211	0	0	0	0	0	0
	Boston	261	0	489	33	0	98	0	0	0
	Dallas	104	78	156	0	0	0	0	0	0
	Detroit	328	197	590	0	0	0	0	0	0
	Philadelphia	58	0	174	0	0	0	0	0	0
	Phoenix	745	397	1142	17	0	50	0	0	0
	Sacramento	38	0	86	0	0	0	0	0	0
	St. Louis	191	72	358	0	0	0	0	0	0

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals exposed at the level).

1 **Table 3D-30. Percent of people estimated to experience at least four exposures at or above**
 2 **benchmarks while at moderate or greater exertion, for air quality adjusted to**
 3 **just meet the current standard.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	<0.1	<0.1	0.1	0	0	0	0	0	0
	Boston	<0.1	<0.1	0.1	0	0	0	0	0	0
	Dallas	0.1	<0.1	0.3	0	0	0	0	0	0
	Detroit	0.2	0.1	0.3	0	0	0	0	0	0
	Philadelphia	0.1	<0.1	0.1	0	0	0	0	0	0
	Phoenix	0.7	0.3	1.1	<0.1	0	<0.1	0	0	0
	Sacramento	<0.1	<0.1	0.1	0	0	0	0	0	0
	St. Louis	0.2	<0.1	0.3	0	0	0	0	0	0
Children with Asthma	Atlanta	<0.1	0	0.1	0	0	0	0	0	0
	Boston	<0.1	0	0.1	0	0	0	0	0	0
	Dallas	0.2	<0.1	0.4	0	0	0	0	0	0
	Detroit	0.1	<0.1	0.2	0	0	0	0	0	0
	Philadelphia	<0.1	<0.1	0.1	0	0	0	0	0	0
	Phoenix	0.8	0.3	1.3	0	0	0	0	0	0
	Sacramento	0.1	0	0.2	0	0	0	0	0	0
	St. Louis	0.1	0	0.3	0	0	0	0	0	0
Adults	Atlanta	<0.1	0	<0.1	0	0	0	0	0	0
	Boston	<0.1	0	<0.1	0	0	0	0	0	0
	Dallas	<0.1	0	<0.1	0	0	0	0	0	0
	Detroit	<0.1	0	<0.1	0	0	0	0	0	0
	Philadelphia	<0.1	0	<0.1	0	0	0	0	0	0
	Phoenix	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	<0.1	0	<0.1	0	0	0	0	0	0
Adults with Asthma	Atlanta	0	0	0	0	0	0	0	0	0
	Boston	0	0	0	0	0	0	0	0	0
	Dallas	0	0	0	0	0	0	0	0	0
	Detroit	0	0	0	0	0	0	0	0	0
	Philadelphia	0	0	0	0	0	0	0	0	0
	Phoenix	<0.1	<0.1	0.1	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	0	0	0	0	0	0	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

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1 **Table 3D-31. Number of people estimated to experience at least four exposures at or above**
 2 **benchmarks while at moderate or greater exertion, for air quality adjusted to**
 3 **just meet the current standard.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (# per Year)			70 ppb Benchmark (7-hr) ^A (# per Year)			80 ppb Benchmark (7-hr) ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	538	61	1190	0	0	0	0	0	0
	Boston	471	137	865	0	0	0	0	0	0
	Dallas	1986	260	4422	0	0	0	0	0	0
	Detroit	1665	746	3035	0	0	0	0	0	0
	Philadelphia	662	349	1157	0	0	0	0	0	0
	Phoenix	5997	2633	9554	5	0	14	0	0	0
	Sacramento	158	8	411	0	0	0	0	0	0
	St. Louis	862	209	1803	0	0	0	0	0	0
Children with Asthma	Atlanta	67	0	101	0	0	0	0	0	0
	Boston	76	0	137	0	0	0	0	0	0
	Dallas	213	24	473	0	0	0	0	0	0
	Detroit	162	52	243	0	0	0	0	0	0
	Philadelphia	58	22	109	0	0	0	0	0	0
	Phoenix	637	212	1033	0	0	0	0	0	0
	Sacramento	23	0	70	0	0	0	0	0	0
	St. Louis	73	0	155	0	0	0	0	0	0
Adults	Atlanta	47	0	141	0	0	0	0	0	0
	Boston	33	0	98	0	0	0	0	0	0
	Dallas	104	0	234	0	0	0	0	0	0
	Detroit	109	0	262	0	0	0	0	0	0
	Philadelphia	29	0	87	0	0	0	0	0	0
	Phoenix	646	199	894	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	60	0	143	0	0	0	0	0	0
Adults with Asthma	Atlanta	0	0	0	0	0	0	0	0	0
	Boston	0	0	0	0	0	0	0	0	0
	Dallas	0	0	0	0	0	0	0	0	0
	Detroit	0	0	0	0	0	0	0	0	0
	Philadelphia	0	0	0	0	0	0	0	0	0
	Phoenix	83	50	149	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	0	0	0	0	0	0	0	0	0

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals exposed at the level).

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3D.3.2.2 Additional Air Quality Scenario: 75 ppb

When considering air quality adjusted so that the design value at the highest monitor location in each urban study area is equal to 75 ppb, there will be a greater percent and number of people estimated to experience 7-hr O₃ exposures at or above each of the benchmarks. For example, estimated exposures to O₃ concentrations at or above the 80-ppb benchmark are limited, but not insignificant. When considering the worst air quality year, upwards to 0.6% of children (and similarly for children with asthma) are estimated to experience at least one day with a 7-hr exposure at or above the 80-ppb benchmark, while on average, most study areas had at least 0.1% of children experiencing such an exposure (Table 3D-32). On average, between about 1 to 2% of children (and similarly for children with asthma) would experience at least one day with a 7-hr exposure at or above the 70-ppb benchmark, while for the worst air quality year upwards to 3.4% of children (and 3.9% children with asthma) would experience such an exposure. On average, between about 7 to 17% of children (and similarly for children with asthma) would experience at least one day with a 7-hr exposure at or above the 60-ppb benchmark, while for the worst year upwards to about 18% of children (and about 19% of children with asthma) would experience such an exposure.

Under the 75 ppb air quality scenario, multiday exposures to the 80 ppb benchmark are few, but not entirely eliminated as was shown with the exposure results considering air quality adjusted to just meet the current standard. A small percent (<0.1%) of children are estimated to experience at least two days with 7-hr exposures at or above the 80-ppb (Table 3D-33). On average, between 0.1 to 0.3% of children (and 0.1 to 0.4% of children with asthma) would experience at least two days with 7-hr exposures at or above the 70-ppb benchmark, while for the worst year upwards to 0.7% of children (and 0.8% of children with asthma) would experience such an exposure. When considering the worst air quality year, between about 3 to 10% of children (and 3 to 11% of children with asthma) and 0.2 to 1.2% of adults (and 0.1 to 1.1% of adults with asthma) are estimated to experience at least two days with 7-hr O₃ exposures at or above the 60-ppb benchmark. On average, all study areas (and study groups) have a small percent (<0.1%) estimated to experience at least four days with 7-hr O₃ exposures at or above the 70-ppb benchmark (Table 3D-34), and at most 2% of children (and 2.3% of children with asthma) are estimated experience at least six days with 7-hr O₃ exposures at or above the 60-ppb benchmark for the worst air quality year (Attachment 4).

1 **Table 3D-32. Percent of people estimated to experience at least one exposure at or above**
 2 **benchmarks while at moderate or greater exertion, for the 75 ppb air quality**
 3 **scenario.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	7.7	4.8	10.7	1.5	0.4	2.8	0.3	<0.1	0.6
	Boston	6.6	5.0	8.8	1.3	0.9	1.9	0.1	0.1	0.1
	Dallas	8.3	4.7	11.5	1.3	0.7	2.1	0.1	<0.1	0.1
	Detroit	11.0	8.6	13.9	1.9	0.9	3.4	0.1	<0.1	0.1
	Philadelphia	8.6	8.2	8.8	1.4	1.2	1.5	0.1	<0.1	0.1
	Phoenix	15.7	13.2	17.9	2.0	0.9	3.4	<0.1	0	0.1
	Sacramento	7.5	6.3	8.9	1.1	0.8	1.4	<0.1	<0.1	<0.1
	St. Louis	10.6	8.5	13.0	1.7	0.8	3.2	0.1	0	0.1
Children with Asthma	Atlanta	8.5	5.2	11.8	1.7	0.4	3.1	0.3	<0.1	0.6
	Boston	7.6	5.7	9.8	1.4	1.0	2.2	0.1	0.1	0.2
	Dallas	8.9	4.6	11.9	1.4	0.9	2.2	0.1	<0.1	0.1
	Detroit	12.0	9.6	15.0	2.1	1.1	3.9	<0.1	0	0.1
	Philadelphia	9.4	9.1	9.6	1.5	1.3	1.6	0.1	<0.1	0.1
	Phoenix	17.1	14.4	19.2	2.1	1.0	3.8	0.1	0	0.2
	Sacramento	7.8	6.9	9.3	1.1	0.9	1.5	0.1	<0.1	0.1
	St. Louis	10.6	8.4	13.2	1.6	0.6	3.2	0.1	0	0.1
Adults	Atlanta	1.3	0.8	1.8	0.2	0.1	0.4	<0.1	0	0.1
	Boston	0.9	0.5	1.3	0.1	0.1	0.2	<0.1	<0.1	<0.1
	Dallas	1.4	0.7	2.1	0.2	0.1	0.3	<0.1	0	<0.1
	Detroit	1.7	1.3	2.3	0.3	0.2	0.5	<0.1	0	<0.1
	Philadelphia	1.2	1.1	1.4	0.2	0.1	0.2	<0.1	<0.1	<0.1
	Phoenix	3.2	2.6	3.6	0.3	0.2	0.4	<0.1	0	<0.1
	Sacramento	1.1	0.9	1.3	0.1	0.1	0.2	<0.1	0	<0.1
	St. Louis	1.7	1.2	2.1	0.2	0.1	0.4	<0.1	0	<0.1
Adults with Asthma	Atlanta	0.9	0.6	1.1	0.2	0.1	0.3	<0.1	0	<0.1
	Boston	0.6	0.4	1.0	0.1	<0.1	0.1	<0.1	0	<0.1
	Dallas	1.1	0.5	1.5	0.1	<0.1	0.1	<0.1	0	<0.1
	Detroit	1.5	1.1	1.7	0.2	0.1	0.4	<0.1	0	<0.1
	Philadelphia	1.0	0.7	1.2	0.1	0.1	0.1	0	0	0
	Phoenix	2.7	2.3	3.0	0.2	0.1	0.4	0	0	0
	Sacramento	0.9	0.7	1.2	0.1	0.1	0.1	<0.1	0	<0.1
	St. Louis	1.3	1.0	1.8	0.2	<0.1	0.4	<0.1	0	<0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **Table 3D-33. Percent of people estimated to experience at least two exposures at or above**
 2 **benchmarks while at moderate or greater exertion, for the 75 ppb air quality**
 3 **scenario.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	2.5	1.1	4.0	0.2	<0.1	0.4	<0.1	0	<0.1
	Boston	1.7	1.1	2.6	0.1	<0.1	0.2	<0.1	0	<0.1
	Dallas	2.9	1.1	4.8	0.1	<0.1	0.3	<0.1	0	<0.1
	Detroit	4.0	2.5	5.8	0.2	<0.1	0.4	<0.1	0	<0.1
	Philadelphia	2.8	2.5	3.0	0.1	0.1	0.2	<0.1	0	<0.1
	Phoenix	8.0	6.0	9.9	0.3	0.1	0.7	<0.1	0	<0.1
	Sacramento	2.4	1.7	3.4	0.1	<0.1	0.2	0	0	0
	St. Louis	3.9	2.7	5.4	0.2	<0.1	0.4	<0.1	0	<0.1
Children with Asthma	Atlanta	2.7	1.1	4.2	0.2	<0.1	0.4	<0.1	0	<0.1
	Boston	2.0	1.3	3.0	0.1	<0.1	0.2	0	0	0
	Dallas	2.9	1.0	4.8	0.1	0	0.3	0	0	0
	Detroit	4.4	2.8	6.4	0.2	<0.1	0.4	0	0	0
	Philadelphia	3.0	2.8	3.1	0.1	0.1	0.1	0	0	0
	Phoenix	8.9	6.7	11.0	0.4	0.2	0.8	0	0	0
	Sacramento	2.6	1.9	3.8	0.1	<0.1	0.2	0	0	0
	St. Louis	3.6	2.5	4.9	0.1	0	0.3	0	0	0
Adults	Atlanta	0.2	0.1	0.4	<0.1	<0.1	<0.1	0	0	0
	Boston	0.1	0.1	0.2	<0.1	<0.1	<0.1	0	0	0
	Dallas	0.2	0.1	0.5	<0.1	<0.1	<0.1	0	0	0
	Detroit	0.3	0.2	0.5	<0.1	<0.1	<0.1	0	0	0
	Philadelphia	0.2	0.1	0.2	<0.1	<0.1	<0.1	0	0	0
	Phoenix	1.0	0.7	1.2	<0.1	<0.1	<0.1	0	0	0
	Sacramento	0.2	0.1	0.2	<0.1	<0.1	<0.1	0	0	0
	St. Louis	0.3	0.2	0.5	<0.1	<0.1	<0.1	0	0	0
Adults with Asthma	Atlanta	0.1	<0.1	0.2	<0.1	0	<0.1	0	0	0
	Boston	0.1	<0.1	0.1	<0.1	0	<0.1	0	0	0
	Dallas	0.1	<0.1	0.2	0	0	0	0	0	0
	Detroit	0.3	0.2	0.5	<0.1	0	<0.1	0	0	0
	Philadelphia	0.1	<0.1	0.2	0	0	0	0	0	0
	Phoenix	0.8	0.6	1.1	<0.1	0	<0.1	0	0	0
	Sacramento	0.1	0.1	0.2	0	0	0	0	0	0
	St. Louis	0.3	0.1	0.4	<0.1	0	<0.1	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

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Table 3D-34. Percent of people estimated to experience at least four exposures at or above benchmarks while at moderate or greater exertion, for the 75 ppb air quality scenario.

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	0.4	0.1	0.9	<0.1	0	<0.1	0	0	0
	Boston	0.1	0.1	0.2	<0.1	0	<0.1	0	0	0
	Dallas	0.6	0.1	1.2	<0.1	0	<0.1	0	0	0
	Detroit	0.7	0.3	1.2	<0.1	0	<0.1	0	0	0
	Philadelphia	0.4	0.3	0.6	<0.1	0	<0.1	0	0	0
	Phoenix	3.0	2.0	4.1	<0.1	<0.1	<0.1	0	0	0
	Sacramento	0.4	0.2	0.8	<0.1	0	<0.1	0	0	0
	St. Louis	0.8	0.4	1.2	<0.1	0	<0.1	0	0	0
Children with Asthma	Atlanta	0.5	0.1	0.9	<0.1	0	<0.1	0	0	0
	Boston	0.1	0.1	0.2	0	0	0	0	0	0
	Dallas	0.5	0.1	1.1	0	0	0	0	0	0
	Detroit	0.6	0.2	1.0	<0.1	0	<0.1	0	0	0
	Philadelphia	0.4	0.3	0.6	0	0	0	0	0	0
	Phoenix	3.3	2.2	4.4	<0.1	0	0.1	0	0	0
	Sacramento	0.4	0.2	0.8	0	0	0	0	0	0
	St. Louis	0.6	0.3	1.1	0	0	0	0	0	0
Adults	Atlanta	<0.1	0	<0.1	0	0	0	0	0	0
	Boston	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Dallas	<0.1	0	<0.1	0	0	0	0	0	0
	Detroit	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Philadelphia	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Phoenix	0.1	0.1	0.2	<0.1	0	<0.1	0	0	0
	Sacramento	<0.1	<0.1	<0.1	0	0	0	0	0	0
	St. Louis	<0.1	<0.1	<0.1	0	0	0	0	0	0
Adults with Asthma	Atlanta	0	0	0	0	0	0	0	0	0
	Boston	<0.1	0	<0.1	0	0	0	0	0	0
	Dallas	0	0	0	0	0	0	0	0	0
	Detroit	<0.1	0	<0.1	0	0	0	0	0	0
	Philadelphia	0	0	0	0	0	0	0	0	0
	Phoenix	0.1	0.1	0.1	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	<0.1	0	0.1	0	0	0	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **3D.3.2.3 Additional Air Quality Scenario: 65 ppb**

2 With increasing stringency (i.e., lowering) of the design value used to represent the air
3 quality scenario, there is a reduction in the percent and number of simulated individuals
4 experiencing 7-hr exposures at or above the benchmarks. Under the 65 ppb air quality scenario,
5 in 6 of the 8 study areas, there are no people estimated to experience at least one benchmark at or
6 above the 80-ppb benchmark (Table 3D-35). Exposures at or above the 70-ppb benchmark are
7 also limited, with at most 0.2% of children (and 0.3% of children with asthma) estimated
8 experience one such exposure during the worst air quality year. On average, between 0.4 to 2.3%
9 of children (and 0.5 to 2.5% of children with asthma) and between 0.1 to 0.4% of adults (and
10 <0.1 to 0.3% of adults with asthma) are estimated to experience at least one 7-hr O₃ exposure at
11 or above the 60-ppb benchmark, while during the worst air quality year, upwards to 3.7% of
12 children (and 4.3% of children with asthma) would experience such an exposure.

13 Multiday exposures at or above the 70-ppb benchmark are nearly eliminated under the 65
14 ppb air quality scenario, with only three study areas having at most, <0.1% of children (and no
15 children with asthma) estimated to experience 7-hr exposures at or above that benchmark for at
16 least two days (Table 3D-36). When considering the worst air quality year, ≤0.5% of children
17 (and 0.6% of children with asthma) and ≤0.1% of adults (and similarly for adults with asthma)
18 are estimated to experience at least two days with 7-hr O₃ exposures at or above the 60-ppb
19 benchmark. There are no people in any of the study areas estimated to experience at least four
20 days with 7-hr O₃ exposures at or above the 70-ppb benchmark (Table 3D-37), and there no
21 simulated individuals estimated to experience at least six days with 7-hr O₃ exposures at or
22 above the 60-ppb benchmark in all but two study areas (Attachment 4).

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Table 3D-35. Percent of people estimated to experience at least one exposure at or above benchmarks while at moderate or greater exertion, for the 65 ppb air quality scenario.

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	1.0	0.3	1.7	0.1	<0.1	0.1	<0.1	0	<0.1
	Boston	1.8	1.1	2.5	0.2	0.1	0.2	<0.1	0	<0.1
	Dallas	2.1	0.9	2.8	0.1	0.1	0.1	0	0	0
	Detroit	2.3	1.4	3.7	<0.1	<0.1	0.1	0	0	0
	Philadelphia	1.5	1.4	1.6	<0.1	<0.1	<0.1	0	0	0
	Phoenix	1.8	0.9	3.0	0	0	0	0	0	0
	Sacramento	0.4	0.3	0.6	0	0	0	0	0	0
	St. Louis	1.6	0.7	3.1	<0.1	0	<0.1	0	0	0
Children with Asthma	Atlanta	1.1	0.3	1.9	0.1	<0.1	0.2	<0.1	0	<0.1
	Boston	2.1	1.3	3.1	0.2	0.1	0.3	<0.1	0	<0.1
	Dallas	2.2	1.1	2.9	0.1	<0.1	0.1	0	0	0
	Detroit	2.5	1.5	4.3	<0.1	0	<0.1	0	0	0
	Philadelphia	1.6	1.3	1.9	<0.1	<0.1	<0.1	0	0	0
	Phoenix	2.1	1.0	3.4	0	0	0	0	0	0
	Sacramento	0.5	0.3	0.6	0	0	0	0	0	0
	St. Louis	1.5	0.6	3.0	<0.1	0	<0.1	0	0	0
Adults	Atlanta	0.1	<0.1	0.3	<0.1	0	<0.1	0	0	0
	Boston	0.2	0.1	0.3	<0.1	<0.1	<0.1	<0.1	0	<0.1
	Dallas	0.3	0.1	0.4	<0.1	<0.1	<0.1	0	0	0
	Detroit	0.4	0.2	0.6	<0.1	0	<0.1	0	0	0
	Philadelphia	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Phoenix	0.3	0.2	0.4	0	0	0	0	0	0
	Sacramento	0.1	<0.1	0.1	0	0	0	0	0	0
	St. Louis	0.2	0.1	0.4	<0.1	0	<0.1	0	0	0
Adults with Asthma	Atlanta	0.1	<0.1	0.1	0	0	0	0	0	0
	Boston	0.1	<0.1	0.2	<0.1	0	<0.1	0	0	0
	Dallas	0.2	<0.1	0.3	<0.1	0	<0.1	0	0	0
	Detroit	0.3	0.2	0.5	<0.1	0	<0.1	0	0	0
	Philadelphia	0.1	0.1	0.2	0	0	0	0	0	0
	Phoenix	0.2	0.1	0.4	0	0	0	0	0	0
	Sacramento	<0.1	<0.1	0.1	0	0	0	0	0	0
	St. Louis	0.2	<0.1	0.5	<0.1	0	<0.1	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

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Table 3D-36. Percent of people estimated to experience at least two exposures at or above benchmarks while at moderate or greater exertion, for the 65 ppb air quality scenario.

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	0.1	<0.1	0.2	<0.1	0	<0.1	0	0	0
	Boston	0.2	0.1	0.3	<0.1	0	<0.1	0	0	0
	Dallas	0.3	<0.1	0.5	0	0	0	0	0	0
	Detroit	0.3	0.1	0.5	<0.1	0	<0.1	0	0	0
	Philadelphia	0.1	0.1	0.2	0	0	0	0	0	0
	Phoenix	0.3	0.1	0.5	0	0	0	0	0	0
	Sacramento	<0.1	<0.1	<0.1	0	0	0	0	0	0
	St. Louis	0.2	<0.1	0.4	0	0	0	0	0	0
Children with Asthma	Atlanta	0.1	0	0.2	0	0	0	0	0	0
	Boston	0.2	0.1	0.3	0	0	0	0	0	0
	Dallas	0.3	<0.1	0.5	0	0	0	0	0	0
	Detroit	0.2	0.1	0.4	0	0	0	0	0	0
	Philadelphia	0.2	0.1	0.2	0	0	0	0	0	0
	Phoenix	0.3	0.1	0.6	0	0	0	0	0	0
	Sacramento	<0.1	0	<0.1	0	0	0	0	0	0
	St. Louis	0.1	<0.1	0.3	0	0	0	0	0	0
Adults	Atlanta	<0.1	0	<0.1	0	0	0	0	0	0
	Boston	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Dallas	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Detroit	<0.1	<0.1	0.1	0	0	0	0	0	0
	Philadelphia	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Phoenix	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Sacramento	<0.1	<0.1	<0.1	0	0	0	0	0	0
	St. Louis	<0.1	0	<0.1	0	0	0	0	0	0
Adults with Asthma	Atlanta	0	0	0	0	0	0	0	0	0
	Boston	<0.1	0	<0.1	0	0	0	0	0	0
	Dallas	0	0	0	0	0	0	0	0	0
	Detroit	<0.1	0	0.1	0	0	0	0	0	0
	Philadelphia	0	0	0	0	0	0	0	0	0
	Phoenix	<0.1	0	<0.1	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	<0.1	0	<0.1	0	0	0	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **Table 3D-37. Percent of people estimated to experience at least four exposures at or above**
 2 **benchmarks while at moderate or greater exertion, for the 65 ppb air quality**
 3 **scenario.**

Study Group	Study Area	60 ppb Benchmark (7-hr) ^A (% per Year)			70 ppb Benchmark (7-hr) ^A (% per Year)			80 ppb Benchmark (7-hr) ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	<0.1	0	<0.1	0	0	0	0	0	0
	Boston	<0.1	0	<0.1	0	0	0	0	0	0
	Dallas	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Detroit	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Philadelphia	<0.1	0	<0.1	0	0	0	0	0	0
	Phoenix	<0.1	<0.1	<0.1	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	<0.1	0	<0.1	0	0	0	0	0	0
Children with Asthma	Atlanta	<0.1	0	<0.1	0	0	0	0	0	0
	Boston	0	0	0	0	0	0	0	0	0
	Dallas	0	0	0	0	0	0	0	0	0
	Detroit	<0.1	0	<0.1	0	0	0	0	0	0
	Philadelphia	0	0	0	0	0	0	0	0	0
	Phoenix	<0.1	0	0.1	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	0	0	0	0	0	0	0	0	0
Adults	Atlanta	0	0	0	0	0	0	0	0	0
	Boston	0	0	0	0	0	0	0	0	0
	Dallas	0	0	0	0	0	0	0	0	0
	Detroit	0	0	0	0	0	0	0	0	0
	Philadelphia	0	0	0	0	0	0	0	0	0
	Phoenix	0	0	0	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	0	0	0	0	0	0	0	0	0
Adults with Asthma	Atlanta	0	0	0	0	0	0	0	0	0
	Boston	0	0	0	0	0	0	0	0	0
	Dallas	0	0	0	0	0	0	0	0	0
	Detroit	0	0	0	0	0	0	0	0	0
	Philadelphia	0	0	0	0	0	0	0	0	0
	Phoenix	0	0	0	0	0	0	0	0	0
	Sacramento	0	0	0	0	0	0	0	0	0
	St. Louis	0	0	0	0	0	0	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

5

1 **3D.3.2.4 Comparison with 2014 HREA Exposure Results**

2 We compared the exposure results for the current exposure and risk analysis with those
3 generated for the 2014 HREA. Table 3D-38 presents the percent of children experiencing at least
4 one exposure at or above the three benchmarks for the two assessments and Table 3D-39
5 presents the similar comparison for two or more exposures. Results are presented for all study
6 areas, and for the seven study areas common to both assessments. In general, the comparison
7 indicates similarity between the two assessments, particularly for the highest benchmark and
8 when focusing on the summary for all areas in each assessment. Such a focus is appropriate
9 given the purpose of the assessments in providing estimates across a range of study areas to
10 inform decision making with regard to the exposures and risks that may occur across the U.S. in
11 areas that just meet the current standard. For the lower benchmarks and particularly in comparing
12 for the seven areas common to both assessments, the current assessment estimates are slightly
13 lower than the 2014 HREA results, most notably for the highest single year, likely reflecting the
14 greater variation in ambient air concentrations in some study areas in the 2014 HREA. This is
15 supported by recent analyses that show changes to the distribution of ambient air O₃
16 concentrations over time occur primarily as reductions to the highest and lowest concentrations
17 (Downey et al., 2015; Simon et al., 2012).

18 In addition to generally lower baseline O₃ concentrations and lower variability in the
19 concentrations in the three air quality scenarios for the current assessment compared to 2014
20 HREA, there were also two important differences in the exposure modeling approach. The first is
21 the use, in the current assessment, of an EVR distribution (17.32 ± 1.25 L/minute-m²) to indicate
22 when a simulated individual is at moderate or greater exertion (section 3D.2.2.3.3) rather than
23 using a lower value for all simulated individuals (13 L/minute-m²; 5th percentile). The current
24 approach would be expected to result in far fewer individuals reaching the exertion level
25 concomitant with the exposure level of interest, thus reducing the percent of the population at or
26 above benchmarks. The second difference is the focus on 7-hr average exposures (compared to
27 the benchmarks) in this assessment rather than 8-hr averages. With this change, it would be
28 expected that there would be more simulated individuals at or above a given benchmark
29 concentration. While these two changes to the exposure modeling approach compete in their
30 overall influence on the exposure results, it would be expected that the change to using the EVR
31 distribution would have a greater impact.

32 As suggested above, the difference between the two assessments in the highest year
33 estimates is likely a function of the baseline ambient air concentrations in the study areas. As a
34 reminder, the 2014 HREA used air quality scenarios developed from adjusting 2006-2010
35 ambient air concentrations, and some study areas had design values in that time period that were
36 well above the then-existing standard (and more so for the current standard). In the current

1 exposure analysis, we selected study areas that had 2015-2017 design values close to the current
 2 standard, requiring less of an adjustment for the current standard (70 ppb) air quality scenario.

3

4 **Table 3D-38. Comparison of current assessment to 2014 HREA for percent of children**
 5 **estimated to experience at least one exposure at or above benchmarks while at**
 6 **moderate or greater exertion.**

Air Quality Scenario (DV, ppb)	Average Percent (%) of Simulated Children with at least One Day per Year at or above Specified Benchmark Exposure Concentration (highest in single season)			
	All areas ^A		7 common areas ^A	
	Current PA ^B	2014 HREA ^C	Current PA ^B	2014 HREA ^C
<i>Benchmark Exposure Concentration of 80 ppb</i>				
75	<0.1 ^B – 0.3 (0.6)	0 – 0.3 (1.1)	<0.1 – 0.3 (0.6)	0.1 – 0.3 (1.1)
70	0 – <0.1 (0.1)	0 – 0.1 (0.2)	0 – <0.1 (0.1)	0 ^B – 0.1 (0.2)
65	0 – <0.1 (<0.1)	0 (0)	0 – <0.1 (<0.1)	0 (0)
<i>Benchmark Exposure Concentration of 70 ppb</i>				
75	1.1 – 2.0 (3.4)	0.6 – 3.3 (8.1)	1.1 – 1.9 (3.4)	1.6 – 3.3 (8.1)
70	0.2 – 0.6 (0.9)	0.1 – 1.2 (3.2)	0.2 – 0.6 (0.9)	0.4 – 1.2 (3.2)
65	0 – 0.2 (0.2)	0 – 0.2 (0.5)	0 ^B – 0.2 (0.2)	0.1 – 0.2 (0.5)
<i>Benchmark Exposure Concentration of 60 ppb</i>				
75	6.6 – 15.7 (17.9)	9.5 – 17.0 (25.8)	6.6 – 11.0 (13.9)	10.3 – 16.3 (25.8)
70	3.2 – 8.2 (10.6)	3.3 – 10.2 (18.9)	3.2 – 6.7 (9.2)	5.8 – 10.2 (16.9)
65	0.4 – 2.3 (3.7)	0 – 4.2 (9.5)	0.4 – 2.3 (3.7)	2.4 – 3.9 (7.6)
^A Footnote 9 contains the names of the 15 study areas evaluated for the 2014 HREA. The seven study areas common to both include the eight evaluated in this assessment with exception of Phoenix. ^B For the current analysis, calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1" ^C For the 2014 HREA, calculated percent was rounded to the nearest tenth decimal using conventional rounding. Values that did not round upwards to 0.1 (i.e., <0.05) were given a value of "0".				

7

8

1 **Table 3D-39. Comparison of current assessment to 2014 HREA for percent of children**
 2 **estimated to experience at least two exposure at or above benchmarks while**
 3 **at moderate or greater exertion.**

Air Quality Scenario (DV, ppb)	Average Percent (%) of Simulated Children with at least Two Days per Year at or above Specified Benchmark Exposure Concentration (highest in single season)			
	All areas ^A		7 common areas ^A	
	Current PA ^B	2014 HREA ^C	Current PA ^B	2014 HREA ^C
<i>Benchmark Exposure Concentration of 80 ppb</i>				
75	0 – <0.1 (<0.1)	0 (0.1)	0 – <0.1 (<0.1)	0 (0.1)
70	0 (0)	0 (0)	0 (0)	0 (0)
65	0 (0)	0 (0)	0 (0)	0 (0)
<i>Benchmark Exposure Concentration of 70 ppb</i>				
75	0.1 – 0.3 (0.7)	0.1 – 0.6 (2.2)	0.1 – 0.2 (0.4)	0.2 – 0.6 (2.2)
70	<0.1 (0.1)	0 – 0.1 (0.4)	<0.1 (0.1)	0 – 0.1 (0.4)
65	0 – <0.1 (<0.1)	0 (0)	0 – <0.1 (<0.1)	0 (0)
<i>Benchmark Exposure Concentration of 60 ppb</i>				
75	1.7 – 8.0 (9.9)	3.1 – 7.6 (14.4)	1.7 – 4.0 (5.8)	3.7 – 7.0 (13.8)
70	0.6 – 2.9 (4.3)	0.5 – 3.5 (9.2)	0.6 – 1.7 (2.8)	1.5 – 3.2 (7.1)
65	<0.1 – 0.3 (0.5)	0 – 0.8 (2.8)	<0.1 – 0.3 (0.5)	0.3 – 0.7 (2.0)
^A Footnote 9 contains the names of the 15 study areas evaluated for the 2014 HREA. The seven study areas common to both include the eight evaluated in this assessment with exception of Phoenix. ^B For the current analysis, calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals exposed at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1" ^C For the 2014 HREA, calculated percent was rounded to the nearest tenth decimal using conventional rounding. Values that did not round upwards to 0.1 (i.e., <0.05) were given a value of "0".				

4

5 **3D.3.3 Lung Function Risk**

6 As described above, lung function risk was estimated using two approaches. The first, a
 7 population-based risk approach (i.e., using E-R functions, section 3D.2.8.2.1), combined the
 8 population distribution of daily maximum 7-hr exposures occurring while at moderate or greater
 9 exertion with continuous E-R functions derived from the controlled human exposure study data
 10 (Table 3D-20 and Figure 3D-12). Note that the E-R function risk approach uses the full
 11 distribution of daily maximum 7-hr exposures, from the minimum to the maximum exposures
 12 (i.e., not simply including the upper level exposures or benchmarks). It is, however, necessary
 13 that the daily maximum exposure did occur at a 7-hr EVR $\geq 17.32 \pm 1.25$ L/min-m². The results
 14 for the population-based (E-R function) risk approach, represented as percent (or counts) of the
 15 population estimated to experience lung function decrements (i.e., $\geq 10\%$, $\geq 15\%$, and $\geq 20\%$)

1 reduction in FEV₁) is provided in section 3D.3.3.1. A similar format to that provided for the
2 benchmark results above is followed, focusing largely on the air quality scenario for just meeting
3 the current standard and presenting the percent (and counts) of the population estimated to
4 experience lung function decrements while at elevated exertion.

5 The second risk approach, an individual-based risk approach (i.e., the MSS model,
6 section 3D.2.8.2.2), calculates the decrements in lung function continuously for each simulated
7 person using their unique time-series of O₃ exposures, simultaneously occurring breathing rates,
8 and personal attributes (e.g., age, body mass). Note that when using the MSS model risk
9 approach, the estimated reduction in FEV₁ considers the prior and current exposures/breathing
10 rates and has no hard restriction on either the exposure or exertion level. As such, lung function
11 decrements could also occur at exposures and/or breathing rates below that observed in the
12 controlled human exposure studies. The results for the individual-based (MSS model) risk
13 approach are found in section 3D.3.3.2. The complete results for both of the risk approaches can
14 be found in Attachment 4.

15 **3D.3.3.1 Population-based (E-R Function) Risk Approach**

16 As was observed with the exposure benchmarks and considering any of the air quality
17 scenarios, a smaller percent (and number) of adults are estimated to experience lung function
18 decrements when compared to children (Table 3D-40 to Table 3D-51). Again, this is driven
19 largely by the difference in time spent outdoors at elevated exertion. Even though there is limited
20 variability across the eight study areas, Detroit, Phoenix, and St. Louis generally exhibited higher
21 risk estimates relative to the other study areas for instances where risk estimates were above 1%
22 (e.g., where FEV₁ reductions $\geq 10\%$). This is expected given the observation made above
23 regarding the results for the exposure to benchmark comparison and its relationship with the
24 overall distribution of O₃ concentrations in ambient air (Figure 3D-7).

25 In general, when comparing E-R function risk estimates to the benchmark results, the
26 attenuation of the percent estimated to experience lung function decrements is at a lesser rate
27 than that observed for the percent of the population at or above the benchmark levels, with
28 increasing stringency of the design values, and when considering the number of times per year
29 either might occur. For example, while as much as 0.9% of children (and 1.0% of children with
30 asthma) are estimated to experience at least one FEV₁ reduction $\geq 15\%$ while at elevated exertion
31 with air quality just meeting the current standard (Table 3D-40), on average between 0.2 to 0.4%
32 of children (and similarly for children with asthma) in all 8 study areas are estimated to
33 experience at least four such decrements (Table 3D-44) when considering the same air quality
34 scenario. For comparison, while as much as 0.9% of children (and 1.0% of children with asthma)
35 are estimated to experience at least one exposure at or above the 70 ppb benchmark while at

1 elevated exertion for air quality just meeting the current standard (Table 3D-26), there are no
2 children (and similarly for children with asthma) estimated to experience at least four such
3 exposures in all but one study area (Table 3D-30) when considering the same air quality
4 scenario. This relative decreased rate of change observed for the E-R function risk results is
5 likely a function of the broader range (and low level) of exposures used in the calculation
6 compared to that represented by the exposure benchmarks.

7 The risks of lung function decrements in the 75 ppb air quality scenario, which allows
8 higher O₃ concentrations, are of course greater (Table 3D-46 through Table 3D-48) than those
9 for air quality adjusted to just meet the current standard (Table 3D-40 through Table 3D-45),
10 differing by at most a few tenths of a percentage point for both the 15% and 20% reduction in
11 FEV₁. A similar pattern is exhibited when comparing the lung function results for the current
12 standard to those for the 65 ppb air quality scenario (Table 3D-49 through Table 3D-51). A few
13 tenths of a percentage point lower risks are estimated for the lower design value scenario
14 compared to those estimated for the current standard.

15

1 **Table 3D-40. Percent of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for air quality adjusted to just meet**
 3 **the current standard, using the population-based (E-R function) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	2.2	1.9	2.5	0.5	0.4	0.6	0.2	0.1	0.2
	Boston	2.2	2.0	2.3	0.5	0.5	0.6	0.2	0.2	0.2
	Dallas	2.4	2.1	2.6	0.6	0.5	0.7	0.2	0.2	0.3
	Detroit	2.5	2.3	2.8	0.7	0.6	0.8	0.3	0.2	0.3
	Philadelphia	2.3	2.2	2.4	0.6	0.5	0.6	0.2	0.2	0.2
	Phoenix	3.1	2.9	3.3	0.8	0.7	0.9	0.3	0.3	0.4
	Sacramento	2.2	2.2	2.3	0.5	0.5	0.6	0.2	0.2	0.2
	St. Louis	2.5	2.3	2.8	0.7	0.6	0.8	0.2	0.2	0.3
Children with Asthma	Atlanta	2.3	2.0	2.6	0.6	0.5	0.7	0.2	0.1	0.3
	Boston	2.4	2.2	2.6	0.6	0.6	0.7	0.2	0.2	0.3
	Dallas	2.6	2.3	2.8	0.7	0.5	0.8	0.2	0.2	0.3
	Detroit	2.7	2.6	3.0	0.7	0.6	0.8	0.3	0.2	0.3
	Philadelphia	2.4	2.4	2.5	0.6	0.6	0.6	0.2	0.2	0.2
	Phoenix	3.3	3.1	3.6	0.9	0.8	1.0	0.3	0.3	0.4
	Sacramento	2.3	2.3	2.4	0.5	0.5	0.6	0.2	0.2	0.2
	St. Louis	2.6	2.3	2.8	0.7	0.6	0.8	0.2	0.2	0.3
Adults	Atlanta	0.6	0.6	0.7	0.1	0.1	0.2	<0.1	<0.1	0.1
	Boston	0.6	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.7	0.6	0.7	0.2	0.1	0.2	0.1	<0.1	0.1
	Detroit	0.6	0.6	0.7	0.2	0.1	0.2	0.1	<0.1	0.1
	Philadelphia	0.6	0.6	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.9	0.9	1.0	0.2	0.2	0.2	0.1	0.1	0.1
	Sacramento	0.6	0.6	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.7	0.6	0.7	0.1	0.1	0.2	0.1	<0.1	0.1
Adults with Asthma	Atlanta	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.4	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.6	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.5	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.7	0.7	0.8	0.2	0.2	0.2	0.1	0.1	0.1
	Sacramento	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.6	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **Table 3D-41. Number of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for air quality adjusted to just meet**
 3 **the current standard, using the population-based (E-R function) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (# per Year)			≥15% reduction in FEV ₁ ^A (# per Year)			≥20% reduction in FEV ₁ ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	26149	22779	29781	6369	5064	7768	2273	1634	2966
	Boston	29437	27715	31856	7433	6804	8442	2746	2457	3254
	Dallas	34128	30101	37100	8615	7070	9837	3153	2412	3760
	Detroit	26489	24402	28928	6978	6122	8030	2642	2220	3174
	Philadelphia	30134	28919	31014	7406	7050	7661	2655	2510	2750
	Phoenix	26169	24400	28193	6930	6199	7770	2614	2250	3029
	Sacramento	10458	10047	10800	2484	2321	2632	859	784	932
	St. Louis	13912	12540	15144	3594	3069	4143	1345	1093	1630
Children with Asthma	Atlanta	3322	2885	3793	814	646	989	289	202	383
	Boston	4027	3686	4323	1024	910	1160	387	341	455
	Dallas	3389	2956	3712	859	686	993	315	236	378
	Detroit	3208	2931	3503	844	728	971	318	260	382
	Philadelphia	3594	3448	3732	880	829	917	320	306	327
	Phoenix	2684	2463	2901	713	623	807	269	226	311
	Sacramento	1043	1009	1095	246	233	264	85	78	93
	St. Louis	1439	1302	1530	370	319	419	137	109	164
Adults	Atlanta	26671	24018	29934	5658	4789	6691	1808	1409	2254
	Boston	33036	30818	35514	7011	6261	7925	2218	1859	2642
	Dallas	32817	29848	35083	7215	6095	8126	2370	1875	2813
	Detroit	25452	23857	27527	5921	5309	6816	2054	1770	2491
	Philadelphia	32243	30936	33288	6826	6449	7146	2150	2004	2266
	Phoenix	28046	26622	29304	6639	6109	7102	2284	2036	2483
	Sacramento	10719	10490	10891	2239	2144	2315	677	629	715
	St. Louis	14271	12662	15165	3207	2683	3577	1073	858	1252
Adults with Asthma	Atlanta	1714	1550	1902	352	282	423	117	70	141
	Boston	2870	2544	3131	587	489	685	196	196	196
	Dallas	1953	1797	2110	443	391	469	130	78	156
	Detroit	2338	2163	2491	524	459	590	175	131	197
	Philadelphia	2585	2527	2701	552	523	610	174	174	174
	Phoenix	2020	1937	2086	480	447	497	166	149	199
	Sacramento	629	600	657	133	114	143	29	29	29
	St. Louis	1132	1037	1180	250	215	286	84	72	107

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals experiencing decrements at the level).

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1 **Table 3D-42. Percent of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for air quality adjusted to just**
 3 **meet the current standard, using the population-based (E-R function) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	1.4	1.3	1.6	0.3	0.3	0.4	0.1	0.1	0.1
	Boston	1.3	1.3	1.4	0.3	0.3	0.3	0.1	0.1	0.1
	Dallas	1.6	1.5	1.7	0.4	0.3	0.4	0.1	0.1	0.1
	Detroit	1.6	1.5	1.8	0.4	0.3	0.4	0.1	0.1	0.1
	Philadelphia	1.5	1.4	1.6	0.3	0.3	0.3	0.1	0.1	0.1
	Phoenix	2.2	2.1	2.4	0.5	0.5	0.6	0.2	0.2	0.2
	Sacramento	1.5	1.5	1.6	0.3	0.3	0.3	0.1	0.1	0.1
	St. Louis	1.7	1.5	1.8	0.4	0.3	0.4	0.1	0.1	0.1
Children with Asthma	Atlanta	1.6	1.4	1.7	0.3	0.3	0.4	0.1	0.1	0.1
	Boston	1.5	1.4	1.6	0.3	0.3	0.4	0.1	0.1	0.1
	Dallas	1.7	1.6	1.9	0.4	0.3	0.4	0.1	0.1	0.1
	Detroit	1.8	1.6	1.9	0.4	0.4	0.4	0.1	0.1	0.1
	Philadelphia	1.6	1.6	1.7	0.4	0.3	0.4	0.1	0.1	0.1
	Phoenix	2.4	2.2	2.6	0.6	0.5	0.6	0.2	0.2	0.2
	Sacramento	1.6	1.6	1.6	0.3	0.3	0.4	0.1	0.1	0.1
	St. Louis	1.7	1.5	1.8	0.4	0.3	0.4	0.1	0.1	0.1
Adults	Atlanta	0.4	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.4	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.4	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.6	0.6	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.4	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
Adults with Asthma	Atlanta	0.3	0.3	0.3	0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.3	0.2	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Dallas	0.3	0.3	0.4	0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.3	0.3	0.3	<0.1	<0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **Table 3D-43. Number of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for air quality adjusted to just**
 3 **meet the current standard, using the population-based (E-R function) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (# per Year)			≥15% reduction in FEV ₁ ^A (# per Year)			≥20% reduction in FEV ₁ ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	17291	15395	19450	3632	3047	4277	1110	868	1372
	Boston	18378	17430	19432	3891	3572	4278	1206	1069	1388
	Dallas	22897	20572	24757	5036	4256	5722	1624	1277	1939
	Detroit	17100	15973	18384	3896	3503	4370	1295	1127	1509
	Philadelphia	19992	18901	20909	4263	3972	4518	1324	1222	1419
	Phoenix	18937	17748	20310	4529	4076	5039	1566	1359	1797
	Sacramento	7200	6972	7415	1511	1436	1592	461	427	497
	St. Louis	9222	8351	9790	2076	1803	2295	680	565	783
Children with Asthma	Atlanta	2219	1977	2462	464	383	545	148	121	182
	Boston	2526	2321	2662	539	478	592	159	137	182
	Dallas	2278	2034	2483	496	402	567	158	118	189
	Detroit	2064	1890	2220	468	416	520	156	139	173
	Philadelphia	2416	2292	2532	517	480	546	160	153	175
	Phoenix	1948	1797	2109	467	410	524	161	142	184
	Sacramento	717	699	753	153	148	163	49	47	54
	St. Louis	941	856	1002	210	182	228	67	55	73
Adults	Atlanta	15542	14157	17468	2841	2465	3310	751	634	916
	Boston	18654	17904	19274	3326	3131	3522	848	783	881
	Dallas	19091	17893	19925	3542	3204	3829	990	859	1094
	Detroit	14135	13567	14747	2731	2556	2949	765	721	852
	Philadelphia	18939	18126	19607	3457	3224	3660	930	871	959
	Phoenix	17781	16986	18625	3708	3427	3973	1142	1043	1242
	Sacramento	6536	6489	6574	1182	1172	1201	305	286	314
	St. Louis	8203	7261	8870	1586	1323	1753	453	358	501
Adults with Asthma	Atlanta	1010	916	1127	188	141	211	70	70	70
	Boston	1631	1468	1761	261	196	294	98	98	98
	Dallas	1120	1094	1172	208	156	234	78	78	78
	Detroit	1311	1245	1376	262	262	262	66	66	66
	Philadelphia	1452	1394	1569	261	261	261	87	87	87
	Phoenix	1275	1192	1341	265	248	298	83	50	99
	Sacramento	391	372	400	67	57	86	29	29	29
	St. Louis	656	608	715	131	107	143	36	36	36

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals experiencing decrements at the level).

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Table 3D-44. Percent of people estimated to experience at least four lung function decrements at or above the indicated level, for air quality adjusted to just meet the current standard, using the population-based (E-R function) risk approach.

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	1.0	0.9	1.1	0.2	0.2	0.2	<0.1	<0.1	0.1
	Boston	0.8	0.8	0.9	0.2	0.1	0.2	<0.1	<0.1	<0.1
	Dallas	1.1	1.0	1.1	0.2	0.2	0.2	0.1	0.1	0.1
	Detroit	1.0	1.0	1.1	0.2	0.2	0.2	0.1	0.1	0.1
	Philadelphia	1.0	0.9	1.1	0.2	0.2	0.2	0.1	<0.1	0.1
	Phoenix	1.6	1.5	1.7	0.4	0.3	0.4	0.1	0.1	0.1
	Sacramento	1.1	1.0	1.1	0.2	0.2	0.2	0.1	0.1	0.1
	St. Louis	1.1	1.0	1.2	0.2	0.2	0.2	0.1	0.1	0.1
Children with Asthma	Atlanta	1.0	1.0	1.2	0.2	0.2	0.2	0.1	<0.1	0.1
	Boston	0.9	0.9	1.0	0.2	0.2	0.2	<0.1	<0.1	0.1
	Dallas	1.2	1.1	1.2	0.2	0.2	0.3	0.1	0.1	0.1
	Detroit	1.1	1.0	1.2	0.2	0.2	0.2	0.1	0.1	0.1
	Philadelphia	1.1	1.0	1.1	0.2	0.2	0.2	0.1	<0.1	0.1
	Phoenix	1.7	1.6	1.8	0.4	0.4	0.4	0.1	0.1	0.1
	Sacramento	1.1	1.1	1.1	0.2	0.2	0.2	0.1	0.1	0.1
	St. Louis	1.1	1.0	1.2	0.2	0.2	0.2	0.1	<0.1	0.1
Adults	Atlanta	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Boston	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Dallas	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Detroit	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Philadelphia	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Phoenix	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	St. Louis	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Adults with Asthma	Atlanta	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Boston	0.1	0.1	0.2	<0.1	<0.1	<0.1	0	0	0
	Dallas	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Detroit	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Philadelphia	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Phoenix	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	St. Louis	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

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1 **Table 3D-45. Number of people estimated to experience at least four lung function**
 2 **decrements at or above the indicated level, for air quality adjusted to just**
 3 **meet the current standard, using the population-based (E-R function) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (# per Year)			≥15% reduction in FEV ₁ ^A (# per Year)			≥20% reduction in FEV ₁ ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	11501	10371	12953	2179	1876	2542	592	484	726
	Boston	11476	11127	11673	2131	2025	2207	561	523	592
	Dallas	15259	14022	16197	3011	2648	3334	851	709	993
	Detroit	10816	10180	11429	2174	1994	2359	636	572	711
	Philadelphia	13227	12310	13969	2539	2314	2728	698	611	764
	Phoenix	13597	12823	14564	2972	2703	3284	948	835	1076
	Sacramento	4935	4814	5023	944	908	978	256	241	272
	St. Louis	6041	5446	6411	1214	1056	1302	352	291	382
Children with Asthma	Atlanta	1486	1352	1654	282	242	323	81	61	101
	Boston	1585	1502	1661	296	273	319	83	68	91
	Dallas	1529	1395	1632	299	260	331	87	71	95
	Detroit	1306	1197	1387	260	243	277	75	69	87
	Philadelphia	1593	1484	1702	306	284	327	80	65	87
	Phoenix	1406	1316	1500	311	283	340	99	85	113
	Sacramento	489	474	512	96	93	101	26	23	31
	St. Louis	619	565	674	124	109	137	33	27	36
Adults	Atlanta	8969	8382	10072	1455	1338	1690	329	282	423
	Boston	10534	10175	10762	1630	1565	1663	359	294	391
	Dallas	10965	10548	11252	1823	1719	1875	417	391	469
	Detroit	7865	7537	8127	1311	1245	1376	328	328	328
	Philadelphia	10922	10457	11241	1772	1656	1830	407	349	436
	Phoenix	11093	10629	11672	2069	1937	2235	563	497	646
	Sacramento	3992	3916	4059	648	629	657	143	143	143
	St. Louis	4626	4113	5043	775	680	858	179	143	215
Adults with Asthma	Atlanta	587	563	634	70	70	70	0	0	0
	Boston	913	783	978	131	98	196	0	0	0
	Dallas	651	625	703	78	78	78	0	0	0
	Detroit	721	655	786	131	131	131	0	0	0
	Philadelphia	813	784	871	116	87	174	0	0	0
	Phoenix	778	745	844	149	149	149	50	50	50
	Sacramento	229	229	229	29	29	29	0	0	0
	St. Louis	358	322	393	60	36	72	0	0	0

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals experiencing decrements at the level).

1 **Table 3D-46. Percent of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for the 75 ppb air quality scenario,**
 3 **using the population-based (E-R function) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	2.8	2.4	3.2	0.8	0.6	1.0	0.3	0.2	0.4
	Boston	2.4	2.3	2.7	0.7	0.6	0.8	0.3	0.2	0.3
	Dallas	2.8	2.4	3.1	0.8	0.6	0.9	0.3	0.2	0.4
	Detroit	3.0	2.7	3.4	0.9	0.8	1.0	0.4	0.3	0.5
	Philadelphia	2.9	2.7	3.0	0.8	0.8	0.8	0.3	0.3	0.3
	Phoenix	3.8	3.5	4.1	1.1	1.0	1.3	0.5	0.4	0.6
	Sacramento	2.8	2.7	2.9	0.8	0.7	0.8	0.3	0.3	0.3
	St. Louis	3.1	2.7	3.3	0.9	0.8	1.0	0.4	0.3	0.4
Children with Asthma	Atlanta	3.0	2.6	3.5	0.9	0.7	1.1	0.4	0.2	0.5
	Boston	2.7	2.5	3.0	0.8	0.6	0.9	0.3	0.2	0.4
	Dallas	3.0	2.6	3.3	0.8	0.7	1.0	0.3	0.3	0.4
	Detroit	3.3	3.0	3.6	1.0	0.8	1.1	0.4	0.3	0.5
	Philadelphia	3.1	2.9	3.1	0.9	0.8	0.9	0.3	0.3	0.4
	Phoenix	4.1	3.7	4.4	1.2	1.1	1.4	0.5	0.4	0.6
	Sacramento	2.9	2.8	2.9	0.8	0.7	0.8	0.3	0.3	0.3
	St. Louis	3.1	2.7	3.4	0.9	0.7	1.0	0.4	0.3	0.4
Adults	Atlanta	0.8	0.7	0.9	0.2	0.2	0.2	0.1	0.1	0.1
	Boston	0.6	0.6	0.7	0.1	0.1	0.2	<0.1	<0.1	0.1
	Dallas	0.8	0.7	0.9	0.2	0.2	0.2	0.1	0.1	0.1
	Detroit	0.7	0.7	0.8	0.2	0.2	0.2	0.1	0.1	0.1
	Philadelphia	0.7	0.7	0.8	0.2	0.2	0.2	0.1	0.1	0.1
	Phoenix	1.1	1.0	1.2	0.3	0.3	0.3	0.1	0.1	0.1
	Sacramento	0.7	0.7	0.8	0.2	0.2	0.2	0.1	0.1	0.1
	St. Louis	0.8	0.7	0.8	0.2	0.2	0.2	0.1	0.1	0.1
Adults with Asthma	Atlanta	0.6	0.5	0.7	0.1	0.1	0.2	<0.1	<0.1	0.1
	Boston	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.6	0.6	0.7	0.2	0.1	0.2	0.1	<0.1	0.1
	Detroit	0.6	0.6	0.7	0.2	0.1	0.2	0.1	<0.1	0.1
	Philadelphia	0.6	0.6	0.6	0.1	0.1	0.2	0.1	0.1	0.1
	Phoenix	0.9	0.8	0.9	0.2	0.2	0.2	0.1	0.1	0.1
	Sacramento	0.6	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.6	0.6	0.7	0.2	0.1	0.2	0.1	0.1	0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-47. Percent of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for the 75 ppb air quality scenario,**
 3 **using the population-based (E-R function) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	1.8	1.6	2.0	0.4	0.3	0.5	0.1	0.1	0.2
	Boston	1.5	1.4	1.6	0.3	0.3	0.4	0.1	0.1	0.1
	Dallas	1.9	1.6	2.1	0.4	0.4	0.5	0.2	0.1	0.2
	Detroit	1.9	1.7	2.1	0.5	0.4	0.5	0.2	0.1	0.2
	Philadelphia	1.9	1.8	1.9	0.4	0.4	0.5	0.2	0.1	0.2
	Phoenix	2.7	2.5	2.9	0.7	0.6	0.8	0.3	0.2	0.3
	Sacramento	1.9	1.8	1.9	0.4	0.4	0.5	0.1	0.1	0.2
	St. Louis	2.0	1.8	2.1	0.5	0.4	0.5	0.2	0.1	0.2
Children with Asthma	Atlanta	1.9	1.7	2.2	0.5	0.4	0.5	0.2	0.1	0.2
	Boston	1.7	1.5	1.8	0.4	0.3	0.4	0.1	0.1	0.2
	Dallas	2.0	1.8	2.2	0.5	0.4	0.6	0.2	0.1	0.2
	Detroit	2.0	1.9	2.2	0.5	0.5	0.6	0.2	0.2	0.2
	Philadelphia	2.0	1.9	2.1	0.5	0.4	0.5	0.2	0.1	0.2
	Phoenix	2.9	2.7	3.1	0.8	0.7	0.9	0.3	0.3	0.3
	Sacramento	1.9	1.9	2.0	0.5	0.4	0.5	0.1	0.1	0.2
	St. Louis	2.0	1.8	2.1	0.5	0.4	0.5	0.2	0.1	0.2
Adults	Atlanta	0.4	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.4	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.7	0.6	0.7	0.2	0.1	0.2	0.1	<0.1	0.1
	Sacramento	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.4	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
Adults with Asthma	Atlanta	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.3	0.2	0.3	0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.3	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.5	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	0.1
	Sacramento	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.4	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-48. Percent of people estimated to experience at least four lung function**
 2 **decrements at or above the indicated level, for the 75 ppb air quality scenario,**
 3 **using the population-based (E-R function) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	1.2	1.0	1.3	0.2	0.2	0.3	0.1	0.1	0.1
	Boston	0.9	0.9	0.9	0.2	0.2	0.2	<0.1	<0.1	0.1
	Dallas	1.2	1.1	1.3	0.3	0.2	0.3	0.1	0.1	0.1
	Detroit	1.2	1.1	1.3	0.3	0.2	0.3	0.1	0.1	0.1
	Philadelphia	1.2	1.1	1.3	0.3	0.2	0.3	0.1	0.1	0.1
	Phoenix	1.9	1.8	2.0	0.5	0.4	0.5	0.2	0.1	0.2
	Sacramento	1.3	1.2	1.3	0.3	0.2	0.3	0.1	0.1	0.1
Children with Asthma	Atlanta	1.3	1.2	1.4	0.3	0.2	0.3	0.1	0.1	0.1
	Boston	1.0	1.0	1.1	0.2	0.2	0.2	0.1	0.1	0.1
	Dallas	1.3	1.2	1.4	0.3	0.2	0.3	0.1	0.1	0.1
	Detroit	1.2	1.2	1.3	0.3	0.2	0.3	0.1	0.1	0.1
	Philadelphia	1.3	1.2	1.4	0.3	0.3	0.3	0.1	0.1	0.1
	Phoenix	2.0	1.9	2.2	0.5	0.4	0.6	0.2	0.2	0.2
	Sacramento	1.3	1.3	1.3	0.3	0.3	0.3	0.1	0.1	0.1
Adults	Atlanta	0.2	0.2	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Boston	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Dallas	0.3	0.2	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Detroit	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Philadelphia	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Phoenix	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.3	0.3	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Adults with Asthma	Atlanta	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Boston	0.1	0.1	0.2	<0.1	<0.1	<0.1	0	0	0
	Dallas	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Detroit	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	0	<0.1
	Philadelphia	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Phoenix	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
St. Louis	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	0	<0.1	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-49. Percent of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for the 65 ppb air quality scenario,**
 3 **using the population-based (E-R function) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	1.7	1.5	1.9	0.4	0.3	0.4	0.1	0.1	0.1
	Boston	1.8	1.7	1.9	0.4	0.4	0.4	0.1	0.1	0.2
	Dallas	2.0	1.8	2.1	0.5	0.4	0.5	0.2	0.1	0.2
	Detroit	2.0	1.9	2.2	0.5	0.4	0.5	0.2	0.1	0.2
	Philadelphia	1.9	1.8	2.0	0.4	0.4	0.4	0.1	0.1	0.1
	Phoenix	2.4	2.3	2.6	0.6	0.5	0.6	0.2	0.2	0.2
	Sacramento	1.7	1.7	1.7	0.3	0.3	0.4	0.1	0.1	0.1
St. Louis	2.0	1.8	2.1	0.4	0.4	0.5	0.1	0.1	0.2	
Children with Asthma	Atlanta	1.8	1.6	2.0	0.4	0.3	0.5	0.1	0.1	0.2
	Boston	2.0	1.9	2.1	0.4	0.4	0.5	0.1	0.1	0.2
	Dallas	2.2	2.0	2.3	0.5	0.4	0.5	0.2	0.1	0.2
	Detroit	2.2	2.1	2.4	0.5	0.5	0.6	0.2	0.2	0.2
	Philadelphia	2.0	2.0	2.1	0.5	0.4	0.5	0.1	0.1	0.1
	Phoenix	2.6	2.4	2.8	0.6	0.5	0.7	0.2	0.2	0.2
	Sacramento	1.7	1.7	1.8	0.4	0.3	0.4	0.1	0.1	0.1
St. Louis	2.0	1.8	2.2	0.5	0.4	0.5	0.1	0.1	0.2	
Adults	Atlanta	0.5	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.6	0.6	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.5	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.8	0.7	0.8	0.2	0.2	0.2	<0.1	<0.1	0.1
	Sacramento	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
St. Louis	0.6	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1	
Adults with Asthma	Atlanta	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.4	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.6	0.6	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
St. Louis	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-50. Percent of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for the 65 ppb air quality scenario,**
 3 **using the population-based (E-R function) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	1.1	1.0	1.3	0.2	0.2	0.2	0.1	<0.1	0.1
	Boston	1.1	1.1	1.2	0.2	0.2	0.2	0.1	0.1	0.1
	Dallas	1.4	1.3	1.5	0.3	0.2	0.3	0.1	0.1	0.1
	Detroit	1.4	1.3	1.4	0.3	0.3	0.3	0.1	0.1	0.1
	Philadelphia	1.3	1.2	1.4	0.3	0.2	0.3	0.1	0.1	0.1
	Phoenix	1.8	1.7	1.9	0.4	0.4	0.4	0.1	0.1	0.1
	Sacramento	1.2	1.2	1.2	0.2	0.2	0.2	0.1	0.1	0.1
	St. Louis	1.4	1.2	1.4	0.3	0.2	0.3	0.1	0.1	0.1
Children with Asthma	Atlanta	1.2	1.1	1.4	0.2	0.2	0.3	0.1	0.1	0.1
	Boston	1.3	1.2	1.3	0.2	0.2	0.3	0.1	0.1	0.1
	Dallas	1.5	1.4	1.6	0.3	0.3	0.3	0.1	0.1	0.1
	Detroit	1.5	1.4	1.5	0.3	0.3	0.3	0.1	0.1	0.1
	Philadelphia	1.4	1.3	1.5	0.3	0.3	0.3	0.1	0.1	0.1
	Phoenix	1.9	1.8	2.1	0.4	0.4	0.5	0.1	0.1	0.1
	Sacramento	1.2	1.2	1.3	0.2	0.2	0.2	0.1	0.1	0.1
	St. Louis	1.4	1.2	1.4	0.3	0.2	0.3	0.1	0.1	0.1
Adults	Atlanta	0.3	0.3	0.3	0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.3	0.3	0.3	<0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.4	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.3	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
Adults with Asthma	Atlanta	0.2	0.2	0.3	<0.1	<0.1	<0.1	<0.1	0	<0.1
	Boston	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Dallas	0.3	0.3	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Detroit	0.3	0.3	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Philadelphia	0.3	0.2	0.3	<0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	St. Louis	0.3	0.2	0.3	<0.1	<0.1	0.1	<0.1	<0.1	<0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-51. Percent of people estimated to experience at least four lung function**
 2 **decrements at or above the indicated level, for the 65 ppb air quality scenario,**
 3 **using the population-based (E-R function) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	0.8	0.7	0.9	0.1	0.1	0.2	<0.1	<0.1	<0.1
	Boston	0.7	0.7	0.7	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.9	0.9	1.0	0.2	0.2	0.2	<0.1	<0.1	0.1
	Detroit	0.9	0.8	0.9	0.2	0.2	0.2	<0.1	<0.1	<0.1
	Philadelphia	0.9	0.8	0.9	0.2	0.1	0.2	<0.1	<0.1	<0.1
	Phoenix	1.3	1.2	1.4	0.3	0.2	0.3	0.1	0.1	0.1
	Sacramento	0.8	0.8	0.9	0.1	0.1	0.2	<0.1	<0.1	<0.1
St. Louis	0.9	0.8	1.0	0.2	0.1	0.2	<0.1	<0.1	<0.1	
Children with Asthma	Atlanta	0.8	0.8	0.9	0.1	0.1	0.2	<0.1	<0.1	<0.1
	Boston	0.8	0.8	0.8	0.1	0.1	0.2	<0.1	<0.1	<0.1
	Dallas	1.0	0.9	1.1	0.2	0.2	0.2	<0.1	<0.1	0.1
	Detroit	0.9	0.9	1.0	0.2	0.2	0.2	<0.1	<0.1	<0.1
	Philadelphia	0.9	0.9	1.0	0.2	0.1	0.2	<0.1	<0.1	<0.1
	Phoenix	1.4	1.3	1.5	0.3	0.3	0.3	0.1	0.1	0.1
	Sacramento	0.9	0.9	0.9	0.2	0.2	0.2	<0.1	<0.1	<0.1
St. Louis	0.9	0.8	1.0	0.2	0.1	0.2	<0.1	<0.1	<0.1	
Adults	Atlanta	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Boston	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Dallas	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Detroit	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Philadelphia	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Phoenix	0.3	0.3	0.3	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Sacramento	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
St. Louis	0.2	0.2	0.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	
Adults with Asthma	Atlanta	0.1	0.1	0.1	<0.1	<0.1	<0.1	0	0	0
	Boston	0.1	0.1	0.1	<0.1	<0.1	<0.1	0	0	0
	Dallas	0.2	0.2	0.2	<0.1	<0.1	<0.1	0	0	0
	Detroit	0.2	0.1	0.2	<0.1	<0.1	<0.1	0	0	0
	Philadelphia	0.1	0.1	0.2	<0.1	<0.1	<0.1	0	0	0
	Phoenix	0.2	0.2	0.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
	Sacramento	0.1	0.1	0.1	<0.1	<0.1	<0.1	0	0	0
St. Louis	0.2	0.1	0.2	<0.1	<0.1	<0.1	0	0	0	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

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3D.3.3.2 Individual-based (MSS Model) Risk Approach

Lung function decrements estimated using the individual-based (MSS model) risk approach are about a factor of four or greater than those estimated using the population-based (E-R function) risk approach (Table 3D-52 through Table 3D-63). The estimated risk of at least one lung function decrement at or above 15% could be as high as 7.8% of children (and 8.7% of children with asthma) considering the worst year air quality and air quality just meeting the current standard, with the average across the 3-year period ranging from about 4.1% to 7.1% of children (and 4.5 to 8.2% of children with asthma) across the eight study areas (Table 3D-52). Recall that when using the E-R approach for the same air quality scenario, only about 1% of children were estimated to experience a decrement at or above 15% in the worst single year, worst area, and between 0.5 to 0.9% on average across the three years. This difference in estimated risks is generally similar to the comparison of the two approaches provided in the 2014 HREA (2014 HREA, Table 6-8) and is directly a result of the differences that exist between the approaches. While both of these risk approaches allow for exposures at and below that observed in the controlled human exposure studies, the MSS model does not have a strict restriction regarding the magnitude of the ventilation rate or its duration. The impact of these important model inputs (i.e., exposure, ventilation rate, and their duration) on the E-R and MSS risk results is discussed further in section 3D.3.4.

1 **Table 3D-52. Percent of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for air quality adjusted to just meet**
 3 **the current standard, using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	13.2	11.7	15.1	4.1	3.4	5.0	1.7	1.3	2.1
	Boston	13.2	12.4	14.1	4.4	4.0	5.0	1.9	1.6	2.3
	Dallas	14.6	13.1	15.7	4.9	4.0	5.4	2.1	1.6	2.5
	Detroit	15.6	14.4	16.9	5.4	4.8	6.1	2.4	2	2.7
	Philadelphia	14.5	13.6	15.0	4.6	4.3	4.8	1.9	1.8	1.9
	Phoenix	20.4	19.4	21.8	7.1	6.4	7.8	3.1	2.7	3.6
	Sacramento	14.3	13.8	14.7	4.4	4.3	4.7	1.8	1.7	2
St. Louis	15.4	14.0	16.3	5.2	4.5	5.9	2.2	1.9	2.7	
Children with Asthma	Atlanta	14.4	12.5	16.6	4.5	3.4	5.9	1.9	1.5	2.6
	Boston	13.9	12.9	14.7	4.8	4.4	5.4	2	1.7	2.4
	Dallas	15.7	13.6	16.9	5.4	4.5	5.9	2.5	1.8	2.8
	Detroit	16.8	15.3	18.4	6.2	5.7	6.9	2.7	2.3	3.3
	Philadelphia	15.2	15.0	15.5	4.8	4.6	5.3	1.9	1.8	2.1
	Phoenix	22.0	20.4	23.3	8.2	7.6	8.7	3.5	3	3.9
	Sacramento	14.7	14.2	15.0	4.5	4.3	4.8	1.8	1.6	2.1
St. Louis	15.8	14.5	16.5	5.4	4.7	5.8	2.4	2	2.8	
Adults	Atlanta	2.5	2.3	2.8	0.7	0.6	0.8	0.3	0.2	0.4
	Boston	2.3	2.1	2.5	0.6	0.6	0.7	0.3	0.2	0.3
	Dallas	2.9	2.6	3.1	0.8	0.7	1.0	0.3	0.3	0.4
	Detroit	2.6	2.5	2.8	0.8	0.7	0.9	0.3	0.3	0.4
	Philadelphia	2.5	2.4	2.6	0.7	0.7	0.7	0.3	0.3	0.3
	Phoenix	4.4	4.1	4.8	1.4	1.3	1.5	0.6	0.6	0.6
	Sacramento	2.6	2.6	2.6	0.7	0.6	0.7	0.3	0.3	0.3
St. Louis	2.7	2.3	2.9	0.8	0.7	0.9	0.3	0.3	0.4	
Adults with Asthma	Atlanta	2.3	2.2	2.4	0.6	0.6	0.7	0.2	0.1	0.3
	Boston	2.0	1.8	2.4	0.5	0.4	0.6	0.2	0.1	0.3
	Dallas	2.5	2.1	2.9	0.7	0.5	1.0	0.3	0.1	0.5
	Detroit	2.5	2.2	2.6	0.7	0.6	0.8	0.4	0.2	0.5
	Philadelphia	2.2	2.1	2.4	0.6	0.4	0.7	0.2	0.1	0.3
	Phoenix	3.5	3.1	3.8	1.1	1.0	1.2	0.5	0.4	0.6
	Sacramento	2.0	2.0	2.1	0.6	0.5	0.6	0.2	0.2	0.3
St. Louis	2.6	2.2	2.9	0.7	0.6	0.8	0.3	0.2	0.4	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

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1 **Table 3D-53. Number of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for air quality adjusted to just meet**
 3 **the current standard, using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (# per Year)			≥15% reduction in FEV ₁ ^A (# per Year)			≥20% reduction in FEV ₁ ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	159429	141680	182558	49769	40676	60328	20378	15233	25685
	Boston	179806	168747	192821	60125	55225	68218	26251	22368	31924
	Dallas	207221	185830	222622	68911	57317	76942	29588	22747	34853
	Detroit	162690	149480	176362	56695	50434	63632	24708	21037	28547
	Philadelphia	189412	178688	196192	60159	56856	62400	24692	23615	25449
	Phoenix	173383	164589	185182	60104	54688	65827	26306	22773	30302
	Sacramento	66574	64364	68293	20665	20024	21871	8473	7904	9286
St. Louis	84339	76632	89017	28337	24351	32356	12185	10309	14507	
Children with Asthma	Atlanta	20513	17776	23929	6356	4802	8434	2670	2078	3733
	Boston	23353	21366	24529	8002	7259	9056	3390	2844	3959
	Dallas	20485	17781	22298	7093	5911	7779	3208	2388	3689
	Detroit	19702	17603	21662	7226	6521	8151	3197	2653	3902
	Philadelphia	22393	21869	23135	7115	6701	7923	2859	2575	3099
	Phoenix	17885	16460	18994	6690	6114	7119	2854	2434	3199
	Sacramento	6625	6328	6972	2058	1941	2244	807	699	978
St. Louis	8852	8278	9234	3008	2650	3206	1327	1157	1512	
Adults	Atlanta	105509	97903	117483	29535	25779	35639	11692	8804	15284
	Boston	135567	121022	149395	37732	32286	41874	16110	12229	18295
	Dallas	133978	119705	144083	39563	33442	44694	15680	13127	19222
	Detroit	104123	97067	110175	30411	26872	33426	11994	10159	13764
	Philadelphia	131672	124004	135506	36048	35118	37123	14175	14030	14466
	Phoenix	131520	121636	143440	41440	40132	43658	17367	16887	18327
	Sacramento	43953	43734	44306	11643	10948	12291	4840	4802	4888
St. Louis	57287	48965	62593	17168	13985	19207	6974	5508	8226	
Adults with Asthma	Atlanta	8123	7677	8875	2278	2043	2395	751	493	1127
	Boston	12980	11447	15067	3229	2348	3816	1337	881	1663
	Dallas	8413	6876	9611	2344	1563	3360	912	391	1641
	Detroit	10508	9241	11273	3059	2359	3474	1529	852	1966
	Philadelphia	11241	10631	12026	2905	1917	3399	1220	436	1656
	Phoenix	9520	8543	10232	2980	2632	3328	1358	1093	1738
	Sacramento	2811	2716	2887	762	715	800	305	229	343
St. Louis	5258	4435	5687	1503	1288	1610	548	322	715	

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals experiencing decrements at the level).

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1 **Table 3D-54. Percent of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for air quality adjusted to just**
 3 **meet the current standard, using the individual-based (MSS model) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	7.7	6.7	9.1	2.1	1.7	2.6	0.8	0.6	1.0
	Boston	7.4	6.9	7.9	2.1	1.9	2.4	0.8	0.7	0.9
	Dallas	8.8	7.8	9.5	2.6	2.0	3.0	1.0	0.7	1.2
	Detroit	9.4	8.5	10.3	2.9	2.5	3.3	1.1	0.9	1.3
	Philadelphia	8.7	8.0	9.1	2.4	2.3	2.5	0.9	0.8	0.9
	Phoenix	13.6	12.8	14.8	4.3	3.8	4.9	1.7	1.5	2
	Sacramento	8.7	8.3	8.9	2.4	2.3	2.5	0.8	0.8	0.9
	St. Louis	9.3	8.2	10.0	2.8	2.3	3.1	1.1	0.9	1.2
Children with Asthma	Atlanta	8.3	6.9	10.2	2.2	1.7	3.3	0.8	0.6	1.2
	Boston	8.0	7.7	8.6	2.3	2.1	2.5	0.9	0.9	0.9
	Dallas	9.6	8.1	10.5	3.1	2.4	3.5	1.1	0.8	1.4
	Detroit	10.3	9.3	11.5	3.3	2.9	3.9	1.3	1.1	1.5
	Philadelphia	9.3	8.6	9.7	2.5	2.3	2.6	0.9	0.7	1
	Phoenix	14.9	13.7	16.0	4.9	4.4	5.3	2.1	1.8	2.5
	Sacramento	8.9	8.4	9.3	2.5	2.2	2.8	0.8	0.5	1.2
	St. Louis	9.4	8.5	9.9	2.9	2.5	3.1	1.1	0.9	1.4
Adults	Atlanta	1.2	1.0	1.4	0.3	0.2	0.4	0.1	0.1	0.1
	Boston	1.0	0.9	1.1	0.3	0.2	0.3	0.1	0.1	0.1
	Dallas	1.4	1.2	1.5	0.3	0.3	0.4	0.1	0.1	0.1
	Detroit	1.2	1.1	1.3	0.3	0.3	0.4	0.1	0.1	0.1
	Philadelphia	1.2	1.2	1.2	0.3	0.3	0.3	0.1	0.1	0.1
	Phoenix	2.4	2.3	2.6	0.7	0.7	0.7	0.3	0.2	0.3
	Sacramento	1.2	1.2	1.2	0.3	0.3	0.3	0.1	0.1	0.1
	St. Louis	1.3	1.0	1.4	0.3	0.3	0.4	0.1	0.1	0.1
Adults with Asthma	Atlanta	1.1	0.9	1.2	0.2	0.2	0.3	0.1	<0.1	0.2
	Boston	0.8	0.7	0.9	0.2	0.2	0.3	0.1	<0.1	0.1
	Dallas	1.1	0.9	1.4	0.3	0.1	0.4	0.1	<0.1	0.3
	Detroit	1.1	1.0	1.2	0.3	0.2	0.4	0.1	0.1	0.2
	Philadelphia	1.0	0.9	1.2	0.2	0.1	0.3	0.1	0.1	0.1
	Phoenix	2.0	1.8	2.1	0.6	0.5	0.7	0.2	0.2	0.2
	Sacramento	1.0	0.9	1.0	0.2	0.2	0.3	0.1	0.1	0.1
	St. Louis	1.1	0.9	1.3	0.3	0.2	0.4	0.1	<0.1	0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **Table 3D-55. Number of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for air quality adjusted to just**
 3 **meet the current standard, using the individual-based (MSS model) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (# per Year)			≥15% reduction in FEV ₁ ^A (# per Year)			≥20% reduction in FEV ₁ ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	92853	80585	110689	25537	20338	32040	9308	7042	11904
	Boston	100764	94590	107742	29058	25849	32471	10816	9079	12697
	Dallas	124935	109975	134637	36832	28658	42869	13738	10475	16410
	Detroit	97682	87982	107267	29946	26015	33819	11643	9729	13302
	Philadelphia	113291	104546	118929	31574	29508	32913	11226	10324	11677
	Phoenix	115472	108372	125399	36615	32468	41342	14766	12596	17210
	Sacramento	40342	38712	41406	10991	10559	11848	3822	3540	4371
	St. Louis	50799	44731	54612	15129	12704	17175	5795	4826	6775
Children with Asthma	Atlanta	11850	9725	14608	3208	2361	4681	1123	807	1715
	Boston	13433	12788	14267	3846	3390	4210	1517	1434	1570
	Dallas	12532	10570	13785	4051	3121	4587	1490	1017	1773
	Detroit	12036	10649	13510	3850	3295	4544	1515	1214	1821
	Philadelphia	13612	12572	14449	3660	3318	3841	1317	1091	1484
	Phoenix	12091	11025	13049	4015	3552	4331	1703	1429	1996
	Sacramento	4040	3773	4294	1121	994	1320	368	233	536
	St. Louis	5270	4863	5509	1627	1421	1739	619	501	747
Adults	Atlanta	48670	43528	58037	12091	10354	15495	4226	3029	6198
	Boston	59908	55473	63495	15426	12621	17513	5479	4305	6164
	Dallas	63629	57118	68916	16096	13908	18675	5912	5391	6720
	Detroit	48523	44634	52368	13130	11142	14681	4566	3867	5571
	Philadelphia	61406	60128	62830	14146	13856	14640	5025	4706	5316
	Phoenix	72697	68690	78624	21043	20513	21655	7665	7152	8642
	Sacramento	21086	21038	21152	5203	5088	5260	1906	1887	1944
	St. Louis	27183	21174	30545	6927	5437	7762	2528	1896	3040
Adults with Asthma	Atlanta	3804	3381	4367	845	634	986	305	141	563
	Boston	5316	4598	5870	1370	978	1859	424	196	587
	Dallas	3620	2891	4532	938	469	1485	416	156	859
	Detroit	4719	4064	5047	1442	983	1704	546	328	852
	Philadelphia	5170	4357	6013	1220	610	1569	378	261	436
	Phoenix	5281	4818	5612	1639	1341	1788	596	497	646
	Sacramento	1324	1258	1429	324	257	400	153	143	172
	St. Louis	2313	1860	2611	632	501	787	179	72	286

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals experiencing decrements at the level).

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1 **Table 3D-56. Percent of people estimated to experience at least four lung function**
 2 **decrements at or above the indicated level, for air quality adjusted to just**
 3 **meet the current standard, using the individual-based (MSS model) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	4.3	3.6	5.2	1.0	0.8	1.3	0.3	0.2	0.4
	Boston	3.9	3.7	4.0	1.0	0.9	1.1	0.3	0.2	0.3
	Dallas	5.1	4.4	5.4	1.3	1.0	1.5	0.4	0.3	0.5
	Detroit	5.2	4.7	5.7	1.4	1.2	1.5	0.5	0.4	0.5
	Philadelphia	4.8	4.3	5.1	1.2	1.0	1.3	0.4	0.3	0.4
	Phoenix	8.8	8.2	9.7	2.5	2.2	2.9	0.9	0.8	1.1
	Sacramento	5.0	4.8	5.2	1.2	1.1	1.3	0.4	0.3	0.4
	St. Louis	5.3	4.5	5.8	1.4	1.2	1.6	0.5	0.4	0.5
Children with Asthma	Atlanta	4.7	4.0	6.0	1.2	0.9	1.7	0.3	0.2	0.6
	Boston	4.3	4.1	4.4	1.1	0.9	1.3	0.3	0.3	0.4
	Dallas	5.7	4.9	6.2	1.4	1.1	1.7	0.5	0.3	0.6
	Detroit	5.8	5.4	6.3	1.6	1.4	1.8	0.5	0.4	0.7
	Philadelphia	5.1	4.9	5.6	1.3	1.2	1.5	0.4	0.3	0.5
	Phoenix	9.8	9.2	10.5	2.9	2.6	3.3	1.1	0.9	1.3
	Sacramento	5.1	4.9	5.3	1.2	1.1	1.5	0.5	0.3	0.7
	St. Louis	5.4	4.8	5.7	1.5	1.3	1.8	0.5	0.4	0.6
Adults	Atlanta	0.5	0.4	0.6	0.1	0.1	0.2	<0.1	<0.1	0.1
	Boston	0.4	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.6	0.5	0.7	0.1	0.1	0.1	<0.1	<0.1	0.1
	Detroit	0.5	0.5	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.5	0.5	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	1.3	1.2	1.4	0.3	0.3	0.4	0.1	0.1	0.1
	Sacramento	0.6	0.6	0.6	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.5	0.4	0.6	0.1	0.1	0.1	<0.1	<0.1	0.1
Adults with Asthma	Atlanta	0.5	0.4	0.6	0.1	<0.1	0.2	<0.1	0	0.1
	Boston	0.3	0.2	0.4	0.1	<0.1	0.1	<0.1	0	<0.1
	Dallas	0.5	0.3	0.8	0.2	0.1	0.3	<0.1	<0.1	0.1
	Detroit	0.5	0.3	0.6	0.1	<0.1	0.2	0.1	<0.1	0.1
	Philadelphia	0.5	0.3	0.6	0.1	<0.1	0.1	<0.1	0	0.1
	Phoenix	1.0	0.8	1.0	0.3	0.2	0.3	0.1	<0.1	0.1
	Sacramento	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	0.1
	St. Louis	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

1 **Table 3D-57. Number of people estimated to experience at least four lung function**
 2 **decrements at or above the indicated level, for air quality adjusted to just**
 3 **meet the current standard, using the individual-based (MSS model) risk**
 4 **approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (# per Year)			≥15% reduction in FEV ₁ ^A (# per Year)			≥20% reduction in FEV ₁ ^A (# per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	51699	44106	63455	12355	9967	15536	3780	2764	5206
	Boston	53018	51152	54929	13205	11832	14358	3921	3390	4323
	Dallas	71709	62140	77108	18302	14282	21352	6101	4540	7094
	Detroit	54058	48613	59001	14453	12574	16025	4839	3885	5584
	Philadelphia	62298	56943	66547	15358	13576	16544	4780	4038	5282
	Phoenix	74522	69479	81962	21334	18895	24698	7954	6893	9469
	Sacramento	23088	22182	24045	5531	5311	5955	1703	1522	1988
	St. Louis	28838	24579	31582	7625	6356	8587	2504	2158	2732
Children with Asthma	Atlanta	6699	5710	8636	1688	1291	2421	450	242	847
	Boston	7190	6849	7372	1821	1525	2230	508	432	592
	Dallas	7456	6432	8158	1852	1442	2128	702	426	851
	Detroit	6810	6226	7423	1902	1596	2151	613	468	780
	Philadelphia	7515	7093	8272	1906	1746	2183	597	458	786
	Phoenix	7973	7445	8534	2359	2081	2703	887	750	1033
	Sacramento	2290	2174	2453	561	481	683	207	140	303
	St. Louis	2996	2741	3151	859	747	965	289	228	319
Adults	Atlanta	21999	18947	26553	4625	3029	7325	1432	634	2395
	Boston	25307	22111	28079	5609	4403	6457	1859	1174	2544
	Dallas	28181	23519	31801	5730	4923	6563	1823	1485	2422
	Detroit	20689	17893	22743	4828	3801	5571	1486	1114	1704
	Philadelphia	27218	26840	27450	5810	5403	6100	1743	1394	1917
	Phoenix	38294	36555	41423	9553	8791	10828	3311	2732	4172
	Sacramento	9862	9547	10119	2201	1972	2344	610	486	715
	St. Louis	11708	9156	13520	2647	2182	3076	954	680	1109
Adults with Asthma	Atlanta	1714	1409	2113	376	141	704	164	0	423
	Boston	2218	1468	2739	391	294	489	131	0	196
	Dallas	1693	938	2657	521	313	938	156	78	234
	Detroit	2141	1376	2687	546	197	852	240	131	328
	Philadelphia	2382	1656	2789	436	174	610	116	0	349
	Phoenix	2599	2285	2781	679	497	844	232	50	397
	Sacramento	648	515	772	152	114	200	48	29	86
	St. Louis	978	751	1109	191	107	250	72	36	143

^A These values represent the population of individuals exposed in each study area. Values equal to zero are indicated by "0" (there are no individuals experiencing decrements at the level).

5

1 **Table 3D-58. Percent of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for the 75 ppb air quality scenario,**
 3 **using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	16.4	14.5	18.7	5.8	4.7	7.1	2.6	2.0	3.3
	Boston	14.7	13.6	15.9	5.2	4.7	5.9	2.4	2.0	2.9
	Dallas	16.7	14.9	18.2	6.0	5.0	6.8	2.7	2.1	3.2
	Detroit	17.8	16.2	19.5	6.7	5.9	7.7	3.1	2.6	3.7
	Philadelphia	17.5	16.6	18.1	6.2	5.9	6.4	2.8	2.7	2.9
	Phoenix	23.6	22.4	25.1	9.0	8.1	9.9	4.2	3.7	4.8
	Sacramento	17.2	16.6	17.7	6.0	5.7	6.3	2.7	2.5	2.9
St. Louis	17.8	16.2	18.8	6.6	5.7	7.5	3.0	2.5	3.6	
Children with Asthma	Atlanta	17.6	15.3	20.2	6.3	4.8	8.2	2.8	2.2	3.8
	Boston	15.6	14.2	16.8	5.6	4.9	6.4	2.6	2.1	3.2
	Dallas	17.9	15.6	19.7	6.7	5.6	7.5	3.1	2.4	3.6
	Detroit	19.1	17.5	20.9	7.6	6.7	8.8	3.5	2.9	4.3
	Philadelphia	18.4	18.0	18.7	6.7	6.5	6.9	2.9	2.8	3.1
	Phoenix	25.1	23.5	26.4	10.2	9.3	11.0	4.9	4.2	5.3
	Sacramento	17.5	16.7	18.2	6.2	6.0	6.4	2.6	2.4	2.8
St. Louis	18.1	16.5	19.0	6.8	6.0	7.3	3.1	2.6	3.6	
Adults	Atlanta	3.2	2.9	3.6	1.0	0.8	1.2	0.4	0.3	0.5
	Boston	2.6	2.3	2.9	0.7	0.6	0.8	0.3	0.3	0.4
	Dallas	3.3	2.9	3.6	1.0	0.9	1.2	0.4	0.4	0.5
	Detroit	3.1	2.8	3.3	1.0	0.8	1.1	0.4	0.3	0.5
	Philadelphia	3.1	2.9	3.3	0.9	0.9	1.0	0.4	0.4	0.4
	Phoenix	5.2	4.8	5.7	1.7	1.7	1.9	0.8	0.7	0.8
	Sacramento	3.2	3.1	3.2	0.9	0.9	1.0	0.4	0.4	0.4
St. Louis	3.1	2.7	3.4	1.0	0.8	1.1	0.4	0.3	0.5	
Adults with Asthma	Atlanta	2.9	2.7	3.3	0.9	0.8	1.0	0.4	0.3	0.4
	Boston	2.2	1.9	2.5	0.6	0.4	0.7	0.2	0.1	0.3
	Dallas	3.0	2.5	3.2	0.9	0.7	1.2	0.4	0.2	0.6
	Detroit	2.9	2.5	3.1	0.9	0.7	1.0	0.4	0.3	0.5
	Philadelphia	2.8	2.7	3.1	0.9	0.8	0.9	0.3	0.1	0.4
	Phoenix	4.1	3.7	4.4	1.4	1.3	1.5	0.7	0.6	0.7
	Sacramento	2.5	2.5	2.6	0.8	0.7	0.9	0.3	0.2	0.3
St. Louis	3.0	2.5	3.3	1.0	0.8	1.0	0.4	0.2	0.5	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-59. Percent of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for the 75 ppb air quality scenario,**
 3 **using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	10.0	8.7	11.8	3.1	2.5	3.9	1.3	1.0	1.6
	Boston	8.4	7.8	9.0	2.6	2.3	2.9	1.0	0.8	1.2
	Dallas	10.3	9.0	11.4	3.3	2.6	4.0	1.3	1.0	1.7
	Detroit	10.9	9.6	12.2	3.7	3.1	4.2	1.5	1.2	1.8
	Philadelphia	10.8	9.9	11.3	3.4	3.2	3.5	1.4	1.2	1.4
	Phoenix	16.1	15.2	17.5	5.6	5.0	6.3	2.5	2.1	2.9
	Sacramento	10.8	10.4	11.2	3.4	3.2	3.6	1.3	1.2	1.5
St. Louis	11.1	9.7	11.9	3.6	3.0	4.1	1.5	1.3	1.8	
Children with Asthma	Atlanta	10.8	9.2	13.1	3.4	2.6	4.6	1.3	1.0	1.9
	Boston	9.0	8.4	9.9	2.8	2.5	3.1	1.1	1.0	1.2
	Dallas	11.1	9.4	12.3	3.7	3.0	4.3	1.5	1.1	1.8
	Detroit	11.8	10.5	13.2	4.1	3.5	4.7	1.8	1.3	2.3
	Philadelphia	11.5	10.9	11.9	3.5	3.3	3.6	1.4	1.3	1.5
	Phoenix	17.4	16.1	18.8	6.4	5.9	6.8	2.9	2.4	3.4
	Sacramento	11.1	10.4	11.7	3.5	3.4	3.8	1.3	1.1	1.6
St. Louis	11.2	9.9	11.9	3.8	3.4	4.2	1.7	1.4	2.0	
Adults	Atlanta	1.5	1.4	1.8	0.4	0.3	0.5	0.2	0.1	0.2
	Boston	1.1	1.0	1.2	0.3	0.3	0.3	0.1	0.1	0.1
	Dallas	1.6	1.4	1.7	0.4	0.4	0.5	0.2	0.1	0.2
	Detroit	1.4	1.3	1.6	0.4	0.3	0.5	0.2	0.1	0.2
	Philadelphia	1.5	1.4	1.5	0.4	0.4	0.4	0.1	0.1	0.2
	Phoenix	2.9	2.7	3.1	0.9	0.9	1.0	0.4	0.3	0.4
	Sacramento	1.6	1.6	1.6	0.4	0.4	0.4	0.2	0.2	0.2
St. Louis	1.5	1.2	1.7	0.4	0.3	0.5	0.2	0.1	0.2	
Adults with Asthma	Atlanta	1.3	1.2	1.5	0.4	0.3	0.4	0.1	0.1	0.2
	Boston	0.9	0.8	1.1	0.2	0.2	0.3	0.1	<0.1	0.1
	Dallas	1.3	1.1	1.6	0.3	0.2	0.5	0.2	0.1	0.3
	Detroit	1.3	1.1	1.4	0.4	0.3	0.5	0.2	0.1	0.3
	Philadelphia	1.3	1.1	1.4	0.3	0.3	0.4	0.1	0.1	0.2
	Phoenix	2.3	2.1	2.5	0.7	0.6	0.8	0.3	0.2	0.4
	Sacramento	1.2	1.1	1.3	0.3	0.3	0.4	0.1	0.1	0.2
St. Louis	1.4	1.1	1.6	0.4	0.3	0.5	0.1	0.1	0.2	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-60. Percent of people estimated to experience at least four lung function**
 2 **decrements at or above the indicated level, for the 75 ppb air quality scenario,**
 3 **using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	5.8	4.9	7.1	1.6	1.3	2.1	0.6	0.4	0.7
	Boston	4.5	4.2	4.7	1.2	1.1	1.3	0.4	0.3	0.4
	Dallas	6.0	5.1	6.6	1.7	1.3	2	0.6	0.4	0.7
	Detroit	6.2	5.4	6.9	1.8	1.6	2.1	0.7	0.5	0.8
	Philadelphia	6.2	5.6	6.7	1.7	1.5	1.9	0.6	0.5	0.7
	Phoenix	10.6	9.8	11.7	3.4	3.1	3.9	1.4	1.2	1.7
	Sacramento	6.4	6.1	6.7	1.8	1.7	1.9	0.6	0.6	0.7
St. Louis	6.5	5.6	7.0	1.9	1.6	2.1	0.7	0.6	0.8	
Children with Asthma	Atlanta	6.4	5.3	8.0	1.8	1.4	2.6	0.6	0.4	1.0
	Boston	5.0	4.7	5.2	1.3	1.2	1.6	0.4	0.3	0.5
	Dallas	6.6	5.5	7.3	2.1	1.6	2.4	0.7	0.5	0.9
	Detroit	6.9	6.2	7.7	2.1	1.7	2.5	0.8	0.5	0.9
	Philadelphia	6.7	6.4	7.3	1.8	1.7	2.0	0.7	0.6	0.7
	Phoenix	11.6	10.7	12.6	4.0	3.6	4.5	1.6	1.4	2.0
	Sacramento	6.6	6.3	7.0	1.8	1.5	2.1	0.6	0.5	0.9
St. Louis	6.4	5.8	6.8	2.1	1.8	2.4	0.7	0.6	0.9	
Adults	Atlanta	0.7	0.6	0.9	0.2	0.1	0.2	0.1	<0.1	0.1
	Boston	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	0.1
	Dallas	0.7	0.6	0.8	0.2	0.1	0.2	0.1	<0.1	0.1
	Detroit	0.6	0.5	0.7	0.1	0.1	0.2	0.1	<0.1	0.1
	Philadelphia	0.7	0.7	0.7	0.2	0.1	0.2	<0.1	<0.1	0.1
	Phoenix	1.6	1.5	1.7	0.4	0.4	0.4	0.2	0.1	0.2
	Sacramento	0.7	0.7	0.7	0.2	0.2	0.2	0.1	0.1	0.1
St. Louis	0.7	0.5	0.8	0.2	0.1	0.2	0.1	<0.1	0.1	
Adults with Asthma	Atlanta	0.7	0.6	0.8	0.1	0.1	0.3	0.1	<0.1	0.1
	Boston	0.4	0.2	0.4	0.1	0.1	0.1	<0.1	0	<0.1
	Dallas	0.6	0.3	0.9	0.2	0.1	0.4	0.1	<0.1	0.1
	Detroit	0.5	0.4	0.7	0.2	0.1	0.2	0.1	<0.1	0.1
	Philadelphia	0.6	0.5	0.7	0.1	0.1	0.2	<0.1	<0.1	0.1
	Phoenix	1.2	1.1	1.2	0.4	0.3	0.4	0.1	<0.1	0.2
	Sacramento	0.6	0.5	0.7	0.2	0.1	0.3	0.1	0.1	0.1
St. Louis	0.6	0.4	0.7	0.1	0.1	0.1	0.1	<0.1	0.1	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-61. Percent of people estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for the 65 ppb air quality scenario,**
 3 **using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	10.3	9.2	11.7	2.8	2.3	3.4	1.0	0.8	1.3
	Boston	10.8	10.4	11.4	3.3	3.1	3.8	1.3	1.2	1.6
	Dallas	12.4	11.2	13.0	3.8	3.1	4.1	1.5	1.2	1.6
	Detroit	12.9	12.0	13.9	4.0	3.6	4.5	1.6	1.4	1.8
	Philadelphia	12.1	11.5	12.6	3.4	3.3	3.6	1.3	1.2	1.3
	Phoenix	16.9	16.0	18.1	5.2	4.7	5.7	2.1	1.8	2.4
	Sacramento	10.8	10.5	11.1	2.8	2.8	3.0	1.0	0.9	1.1
St. Louis	12.3	11.2	13.2	3.6	3.1	4.2	1.4	1.2	1.7	
Children with Asthma	Atlanta	11.2	9.6	13.2	3.1	2.5	4.0	1.1	0.8	1.6
	Boston	11.6	11.2	12.0	3.6	3.3	4.2	1.4	1.3	1.5
	Dallas	13.5	11.8	14.3	4.3	3.6	4.6	1.8	1.3	2.1
	Detroit	14.0	12.9	15.3	4.6	4.3	5.2	1.8	1.5	2.2
	Philadelphia	12.8	12.6	13.0	3.5	3.3	3.7	1.4	1.3	1.6
	Phoenix	18.5	17.1	19.7	6.0	5.4	6.4	2.4	2.1	2.9
	Sacramento	11.2	11.0	11.3	2.9	2.8	3.0	1.0	0.7	1.3
St. Louis	12.7	11.8	13.2	3.8	3.2	4.3	1.5	1.3	1.8	
Adults	Atlanta	1.9	1.8	2.1	0.5	0.4	0.6	0.2	0.1	0.2
	Boston	1.9	1.8	2.1	0.5	0.4	0.5	0.2	0.2	0.2
	Dallas	2.4	2.2	2.5	0.6	0.6	0.7	0.2	0.2	0.3
	Detroit	2.1	2.0	2.3	0.6	0.5	0.6	0.2	0.2	0.2
	Philadelphia	2.1	2.0	2.1	0.5	0.5	0.5	0.2	0.2	0.2
	Phoenix	3.6	3.3	3.9	1.0	1.0	1.0	0.4	0.4	0.4
	Sacramento	1.9	1.9	1.9	0.4	0.4	0.5	0.2	0.2	0.2
St. Louis	2.1	1.8	2.3	0.5	0.4	0.6	0.2	0.2	0.2	
Adults with Asthma	Atlanta	1.7	1.5	1.8	0.5	0.4	0.5	0.1	0.1	0.2
	Boston	1.6	1.5	1.9	0.4	0.3	0.5	0.2	0.1	0.2
	Dallas	2.0	1.7	2.5	0.6	0.4	0.8	0.3	0.1	0.5
	Detroit	2.0	1.8	2.1	0.5	0.4	0.6	0.3	0.2	0.3
	Philadelphia	1.9	1.7	2.0	0.4	0.3	0.5	0.2	0.1	0.2
	Phoenix	2.9	2.5	3.1	0.8	0.8	0.9	0.3	0.2	0.3
	Sacramento	1.6	1.5	1.7	0.4	0.3	0.4	0.1	0.1	0.2
St. Louis	2.0	1.8	2.2	0.5	0.4	0.6	0.2	0.1	0.3	

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-62. Percent of people estimated to experience at least two lung function**
 2 **decrements at or above the indicated level, for the 65 ppb air quality scenario,**
 3 **using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	5.7	5.0	6.8	1.4	1.1	1.6	0.4	0.3	0.5
	Boston	5.9	5.7	6.1	1.5	1.4	1.7	0.5	0.4	0.6
	Dallas	7.3	6.5	7.7	1.9	1.5	2.2	0.7	0.5	0.7
	Detroit	7.4	6.8	8.0	2.0	1.8	2.2	0.7	0.6	0.8
	Philadelphia	7.0	6.5	7.3	1.7	1.6	1.8	0.6	0.5	0.6
	Phoenix	11	10.2	12.0	3.1	2.8	3.5	1.1	1.0	1.3
	Sacramento	6.2	6.0	6.5	1.4	1.3	1.5	0.4	0.4	0.5
	St. Louis	7.1	6.2	7.7	1.8	1.6	2.1	0.6	0.5	0.7
Children with Asthma	Atlanta	6.2	5.3	7.5	1.4	1.1	2.0	0.4	0.3	0.7
	Boston	6.3	6.0	6.8	1.6	1.4	1.8	0.6	0.6	0.6
	Dallas	8.1	6.9	9.0	2.3	1.9	2.6	0.8	0.5	1.0
	Detroit	8.3	7.6	9.0	2.3	2.0	2.8	0.9	0.7	1.0
	Philadelphia	7.6	7.1	8.1	1.8	1.7	1.9	0.6	0.6	0.7
	Phoenix	12.1	11.2	12.9	3.7	3.4	4.0	1.3	1.1	1.5
	Sacramento	6.4	6.1	6.6	1.4	1.2	1.6	0.4	0.3	0.6
	St. Louis	7.3	6.7	7.6	2.0	1.7	2.2	0.7	0.6	0.7
Adults	Atlanta	0.8	0.8	1.0	0.2	0.1	0.3	0.1	<0.1	0.1
	Boston	0.8	0.8	0.9	0.2	0.2	0.2	0.1	0.1	0.1
	Dallas	1.1	1.0	1.2	0.3	0.2	0.3	0.1	0.1	0.1
	Detroit	1.0	0.9	1.0	0.2	0.2	0.3	0.1	0.1	0.1
	Philadelphia	0.9	0.9	1.0	0.2	0.2	0.2	0.1	0.1	0.1
	Phoenix	1.9	1.9	2.1	0.5	0.4	0.5	0.2	0.1	0.2
	Sacramento	0.9	0.9	0.9	0.2	0.2	0.2	0.1	0.1	0.1
	St. Louis	1.0	0.8	1.1	0.2	0.2	0.2	0.1	0.1	0.1
Adults with Asthma	Atlanta	0.7	0.6	0.9	0.2	0.1	0.3	0.1	<0.1	0.1
	Boston	0.7	0.6	0.8	0.2	0.1	0.2	0.1	<0.1	0.1
	Dallas	0.8	0.7	1.1	0.2	0.1	0.4	0.1	0	0.1
	Detroit	0.9	0.7	1.0	0.3	0.2	0.3	0.1	0.1	0.2
	Philadelphia	0.8	0.6	0.9	0.2	0.1	0.3	<0.1	<0.1	0.1
	Phoenix	1.5	1.5	1.6	0.4	0.3	0.5	0.1	0.1	0.2
	Sacramento	0.7	0.6	0.8	0.2	0.1	0.2	<0.1	<0.1	0.1
	St. Louis	0.9	0.7	1.0	0.2	0.1	0.3	<0.1	<0.1	0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

4

1 **Table 3D-63. Percent of people estimated to experience at least four lung function**
 2 **decrements at or above the indicated level, for the 65 ppb air quality scenario,**
 3 **using the individual-based (MSS model) risk approach.**

Study Group	Study Area	≥10% reduction in FEV ₁ ^A (% per Year)			≥15% reduction in FEV ₁ ^A (% per Year)			≥20% reduction in FEV ₁ ^A (% per Year)		
		Avg	Min	Max	Avg	Min	Max	Avg	Min	Max
Children	Atlanta	3.0	2.6	3.7	0.6	0.5	0.8	0.2	0.1	0.2
	Boston	3.0	2.9	3.0	0.6	0.6	0.7	0.2	0.2	0.2
	Dallas	4.0	3.6	4.3	0.9	0.7	1.0	0.3	0.2	0.3
	Detroit	4.0	3.6	4.3	0.9	0.8	1.0	0.3	0.2	0.3
	Philadelphia	3.7	3.5	4.0	0.8	0.7	0.9	0.2	0.2	0.2
	Phoenix	6.8	6.4	7.5	1.7	1.5	2.0	0.5	0.5	0.6
	Sacramento	3.4	3.3	3.5	0.6	0.6	0.7	0.2	0.1	0.2
	St. Louis	3.9	3.3	4.2	0.9	0.7	1.0	0.2	0.2	0.3
Children with Asthma	Atlanta	3.2	2.7	4.3	0.7	0.4	1.1	0.2	0.1	0.2
	Boston	3.4	3.3	3.5	0.8	0.6	0.9	0.2	0.2	0.2
	Dallas	4.6	4.0	4.9	1.1	0.8	1.3	0.3	0.2	0.4
	Detroit	4.5	4.2	4.7	1.1	0.9	1.1	0.3	0.2	0.3
	Philadelphia	3.9	3.6	4.1	0.9	0.8	1.0	0.2	0.2	0.3
	Phoenix	7.6	7.2	8.2	2.1	1.9	2.3	0.7	0.5	0.9
	Sacramento	3.6	3.3	3.7	0.7	0.6	0.9	0.2	0.1	0.3
	St. Louis	4.2	3.8	4.4	1.0	0.8	1.1	0.3	0.2	0.3
Adults	Atlanta	0.4	0.3	0.4	0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Boston	0.3	0.3	0.4	0.1	<0.1	0.1	<0.1	<0.1	<0.1
	Dallas	0.5	0.4	0.5	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Detroit	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Philadelphia	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	Phoenix	1.0	1.0	1.0	0.2	0.2	0.3	0.1	0.1	0.1
	Sacramento	0.4	0.4	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
	St. Louis	0.4	0.3	0.4	0.1	0.1	0.1	<0.1	<0.1	<0.1
Adults with Asthma	Atlanta	0.3	0.2	0.4	0.1	<0.1	0.2	<0.1	0	0.1
	Boston	0.3	0.2	0.3	<0.1	<0.1	<0.1	<0.1	0	<0.1
	Dallas	0.4	0.2	0.6	0.1	<0.1	0.3	<0.1	0	<0.1
	Detroit	0.4	0.3	0.5	0.1	<0.1	0.2	<0.1	<0.1	0.1
	Philadelphia	0.4	0.3	0.4	<0.1	0	0.1	<0.1	0	<0.1
	Phoenix	0.7	0.6	0.8	0.2	0.1	0.3	0.1	0	0.1
	Sacramento	0.3	0.3	0.4	0.1	<0.1	0.1	<0.1	0	<0.1
	St. Louis	0.3	0.3	0.4	0.1	<0.1	0.1	<0.1	0	<0.1

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

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3D.3.4 Uncertainty Characterization

While it may be possible to estimate a range of O₃ exposures or risks by accounting for variability inherent to influential factors, the true exposure or risk for any given individual residing within a study area is unknown. To characterize health risks, risk assessors commonly use an iterative process of gathering data, developing models, and estimating exposures and risks, which is based upon 1) the goals of the assessment, 2) evaluating results for correctness and identifying areas for potential improvement, 3) scale and complexity of the assessment performed, and 4) availability and limitations of the input data and information. Uncertainty can still remain following each iteration and emphasis is then placed on characterizing the nature and magnitude of that uncertainty and its impact on exposure and risk estimates. A summary of the overall characterization of uncertainty for the current O₃ exposure and risk analysis is provided in section 3D.3.4.1. The summary is followed by APEX sensitivity analyses in section 3D.3.4.2 that provide additional support to the uncertainty characterization regarding the influence a number of factors (e.g., contribution of low exposures) have on estimating lung function risk resulting from O₃ exposure.

3D.3.4.1 Summary of the Uncertainty Characterization

The REAs for previous reviews of the O₃, NO₂, SO₂, and CO NAAQS characterized uncertainty in exposure and risk modeling (Langstaff, 2007; U.S. EPA, 2008, 2009, 2010, 2014, 2018). The mainly qualitative approach used in this and other REAs, also informed by quantitative sensitivity analyses, is described by WHO (2008). Briefly, we identified key aspects of the assessment approach that may contribute to uncertainty in the exposure and risk estimates and provided the rationale for their inclusion. Then, we characterized the *magnitude* and *direction* of the influence on the assessment for each of these identified sources of uncertainty.

Consistent with the WHO (2008) guidance, we scaled the overall impact of the uncertainty by considering the degree of uncertainty as implied by the relationship between the source of uncertainty and the exposure and risk estimates. A qualitative characterization of *low*, *moderate*, and *high* was assigned to the magnitude of influence and knowledge base uncertainty descriptors, using quantitative observations relating to understanding the uncertainty, where possible. Where the magnitude of uncertainty was rated *low*, it was judged that large changes within the source of uncertainty would have only a small effect on the assessment results (e.g., an impact of few percentage points upwards to a factor of two). A designation of *medium* implies that a change within the source of uncertainty would likely have a moderate (or proportional) effect on the results (e.g., a factor of two or more). A characterization of *high* implies that a change in the source would have a large effect on results (e.g., an order of magnitude). We also included the direction of influence, whether the source of uncertainty was judged to potentially

1 over-estimate (“*over*”), under-estimate (“*under*”), or have an *unknown* impact to exposure/risk
2 estimates. A summary of the key findings of the prior uncertainty characterizations that are most
3 relevant to the current O₃ exposure and risk analysis are also provided in Table 3D-64.

1 **Table 3D-64. Characterization of key uncertainties in exposure and risk analyses using APEX.**

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
General Aspects of Assessment Design	Representation of population groups with asthma	Unknown	Low - Medium	Medium	Consistent with the ISA identification of people with asthma (and children with asthma in particular) as an important at-risk population for O ₃ in ambient air, risk estimates are developed for people with asthma and are reported separately for children and adults. Exposure and risk were not estimated for more targeted population groups with asthma based on additional personal attributes associated with increased asthma prevalence (e.g., obesity or African American or Hispanic ethnicity) generally due to limitations in the data needed to simulate these population groups. Such data limitations affect our ability to characterize O ₃ exposure and associated health risks for different population subgroups of children and adults with asthma, some of which may have higher exposure/risk and others lower.	Yes. Newly identified element of uncertainty
	Representation of population groups having health conditions other than asthma	Unknown	Unknown	Medium	Individuals having health conditions other than asthma have not been explicitly represented in this exposure and risk assessment as the evidence has not indicated any other population groups with a health condition that places them at increased risk (ISA, Table IS-11). Additionally, exposure/risk modeling for other such groups is hampered by data limitations in accurately defining the size of a particular population group, assigning appropriate activity pattern data, and estimating how responses observed in the controlled human exposure study data would quantitatively relate to simulated individuals. For example, the likelihood of individuals having a health condition such as chronic obstructive pulmonary disorder exercising for sufficient duration at a ventilation rate needed (e.g., EVR $\geq 17.32 \pm 1.25$ L/min-m ²) to receive a dose that would elicit a response is unknown.	Yes. Newly identified element of uncertainty
	Representation of older adults	Neither	Low	Low	In the current exposure and risk analysis, older adults (ages 65-95) were simulated as part of the all adult groups (ages 18-90) and not as a separate population subgroup. In the 2014 HREA, exposures and risks were estimated separately for older adults (2014 HREA, section 5.6). In those 2014 HREA results, the percent of older adults experiencing exposures at or above any benchmark tended to be lower than the comparable percentage for all adults or all adults with asthma, by a few percentage points or less. A similar pattern would be expected if this group were to have been included in the current analysis.	Yes. Newly identified element of uncertainty

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
	Representation of outdoor workers	Under	Low - Medium	Medium	In the current exposure and risk analysis, outdoor workers were not evaluated as a separate population subgroup. In the 2014 HREA, limited analyses were conducted for this subgroup because of appreciable data limitations and associated uncertainty. The exposures and risk estimates for this subgroup for the single study area and air quality scenario assessed indicated a greater percentage of outdoor workers experience single and multi-day exposures than that estimated for the full adult population, differing by about a factor of 5 or more depending on the benchmark level and number of days per year (2014 HREA, section 5.4.3.2). These limited results suggest that results for the full adult population would likely underestimate exposures and risks for outdoor workers. Important uncertainties exist in generating the simulated activity patterns for this group, including the limited number of CHAD diary days available for outdoor workers, assignment of diaries to proper occupation categories, approximating number of days/week and hours/day outdoors, etc.	Yes. Newly identified element of uncertainty
Ambient Air Monitor Concentrations	Ambient Air O ₃ Measurements	Both	Low	Low	Ozone measurements are assumed to be accurate to within ½ of the instrument's Method Detection Limit (MDL), which is 2.5 ppb for most instruments. The EPA requires that routine quality assurance checks are performed on all instruments. There is a known tendency for smoke produced from wildfires to cause interference in O ₃ instruments. Measurements collected by O ₃ analyzers were reported to be biased high by 5.1–6.6 ppb per 100 µg/m ³ of PM _{2.5} from wildfire smoke (U.S. EPA, 2007b). However, smoke concentrations high enough to cause significant interferences are infrequent and the overall impact is expected to be minimal.	No
	Air Quality System (AQS) Database Quality	Both	Low	Low	All ambient air pollutant measurements available from AQS are certified by both the monitoring agency and the corresponding EPA regional office. Monitor malfunctions sometimes occur causing periods of missing data or poor data quality. Monitoring data affected by malfunctions are usually flagged by the monitoring agency and removed from AQS. In addition, the AQS database managers run several routines to identify suspicious data for potential removal.	Yes. Recent year data used (2015 - 2017)
	Temporal Representation	Both	Low	Low	The temporal scale (hourly) is appropriate for analysis performed. Required O ₃ monitoring seasons are used to define the duration of the exposure and risk analyses in each study area. Monitor data are screened for temporal completeness and considered appropriate when calculating design values (and used for adjustments needed to meet air quality scenarios). While some monitoring data used in developing the air quality surface were not screened for temporal completeness, the inclusion of monitor data somewhat less than complete is considered a holistic approach that improves the filling of both temporal and spatial gaps that exist, where present.	No

Sources of Uncertainty		Uncertainty Characterization				Newly Characterized/ New Information?
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Spatial Representation	Both	Low - Medium	Low - Medium	Overall, the eight study areas have reasonably dense ambient monitoring networks but vary in size and geographic location. They are however considered adequate to capture spatial gradients in O ₃ concentrations that occur in urban areas.	No
Adjusted O ₃ Concentrations for Air Quality Scenarios	Modeled atmospheric state (CAMx)	Both	Medium	Medium	In the rollback adjustment framework applied in this assessment, the CAMx air quality model is used to calculate the chemical state of the atmosphere so that the Higher Decoupled Direct Method (HDDM) tool can archive O ₃ responsiveness to precursors at all times and locations within the model domain. Model predictions from CAMx, like all deterministic photochemical models, have both parametric and structural uncertainty associated with them. CAMx is regularly updated to include state-of-the-science parameterizations and processes relevant for atmospheric chemistry and physics. CAMx model performance is also routinely evaluated against available observational datasets (See Appendix 3C).	Yes. Recent year meteorology and emissions inputs used (2016)
	Ozone response sensitivities (HDDM)	Both	Medium	Medium	The HDDM approach allows for the approximation of O ₃ response to alternate emissions scenarios without re-running the model simulation multiple times using different emissions inputs. This approximation becomes less accurate for larger emissions perturbations especially under nonlinear chemistry conditions. However, even at 90% NO _x cut conditions, mean error in predicted O ₃ using HDDM sensitivities was within 2 ppb across all urban study areas compared to the brute force simulation (See Appendix 3C).	Yes. Recent year sensitivities used (from 2016 simulation)
	Voronoi Neighbor Averaging (VNA) spatial interpolation	Both	Low - Medium	Low - Medium	The VNA estimates are weighted based on distance from neighboring monitoring sites, thus the amount of uncertainty tends to increase with distance from the monitoring sites. Areas having a relatively less dense monitoring network (e.g., Atlanta, St. Louis) may have greater uncertainty in the air quality surface than areas with a denser network (e.g., Boston, Philadelphia).	No
APEX: General Input Databases	Population Demographics and Commuting	Both	Low	Low	The U.S. Census data are comprehensive and subject to quality control. Differences in 2010 population data versus modeled years (2015-2017) are likely small when estimating percent of population exposed. While population counts in most areas have likely increased (and thus total number exposed and at risk is likely underestimated), it is likely that there have not been substantive changes to the demographic distributions and commuting patterns in each study area, thus having minimal impact to the percent of the population exposed or experiencing lung function decrements.	Yes. Most recent year data used (2010)

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
	Activity Patterns (CHAD)	Both	Low - Medium	Low - Medium	The CHAD data are comprehensive and subject to quality control. The current version of CHAD contains an increased number of diaries used to estimate exposure from 2014 HREA. Previously, we evaluated trends and patterns in historical activity pattern data – no major issues noted with use of historical data to represent current patterns (2014 HREA, Appendix G, Figures 5G-1 and 5G-2). Compared outdoor event participation and outdoor time of the larger American Time Use Survey (ATUS) data with all other survey data. Participation rate in outdoor events by ATUS is lower, likely due to ATUS survey methods (i.e., a lack of distinction of time spent inside or outside of residence). This finding would primarily apply to adults (ATUS subjects are ages 16 and older). Comparison of activity data (outdoor events and exertion level) for people with asthma generally similar to individuals without asthma (section 3D.2.5.3, Table 3D-10) (see also 2014 HREA, Appendix G, Tables 5G2 to 5G-5). There is little indication of differences in time spent outdoors comparing activity patterns across U.S. regions, though sample size may be a limiting factor in drawing significant conclusions (2014 HREA, section 5.4.1.6). Remaining uncertainty exists for other influential factors that cannot be accounted for (e.g., SES, region/local participation in outdoor events and associated amount of time).	Yes. New data added to CHAD (ATUS 2003-2013) (U.S. EPA, 2019c, Attachment 3)
	Meteorological Data	Both	Low	Low	The NOAA ISH data are comprehensive and subject to quality control, having very few missing values. Limited use in selecting CHAD diaries for simulated individuals and AERs that may vary with temperature. However, while using three years of varying meteorological conditions, the 2015-2017 MET data set may not reflect the full suite of conditions that could exist in future hypothetical air quality scenarios or across periods greater than 3-years.	Yes. Recent year data used (2015-2017) (section 3D.2.4)

Sources of Uncertainty		Uncertainty Characterization				Newly Characterized/ New Information?
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Asthma Prevalence: Selection of "Still" Rather than "Ever" Questionnaire Response	Under	Low	Low	One of the two datasets used to estimate asthma prevalence is 2013-2017 NHIS data. The NHIS dataset includes several categories describing whether a surveyed individual has asthma based on a series of questions (Attachment 1). The first question inquires whether a doctor has "Ever" told the individual they have asthma. This is followed by a question as to whether they "Still" have asthma. In all instances, those responding "Yes" to the "Still" question is a subset of those responding "Yes" to the "Ever" question. For estimating asthma prevalence for the simulated populations, we focused on the dataset for those answering they "Still" have asthma, consistent with the characterization of asthma prevalence in the ISA (ISA, Table IS-11), and concluding that this approach would provide us with the most appropriate estimate of the population of individuals that have asthma and accordingly (based on the at-risk status of this population group) would likely be at increased risk of response to O ₃ exposures. To the extent that some individuals answering "No" to the "Still" question are at increased risk, our approach would underestimate the at-risk group. The answers to subsequent questions in the NHIS dataset (regarding whether the respondent had an asthma attack or asthma-related ER visit during past year) indicate that the extent to which focusing on "Still" have asthma may underestimate this population group is likely small. This conclusion is based on the findings that nearly 95% of those answering "No" to the "Still" have question also did not have an asthma attack or asthma-related ER visit in the past year, while nearly half of those answering "Yes" did have such an experience, as well as the fact that nearly 95% of the survey respondents that indicated they had had an asthma attack or asthma-related ER visit over the past year are captured by the "Still" have asthma category (Attachment 1). Thus, while it is likely that using the response for the "Still" question underestimates asthma prevalence for those not having a physician determined diagnosis, the magnitude of underestimation is likely quite small.	Yes. Newly identified element of uncertainty

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
	Asthma Prevalence: Weighted by Family Income	Both	Low	Low - Medium	Data used are from peer-reviewed quality-controlled sources. Use of these data accounts for variability in important influential variables (poverty status, as well as age, sex, and region). Regional prevalence from NHIS were adjusted to reflect state-level prevalence from BRFSS, improving local representation. It is possible however that variability in microscale prevalence is not entirely represented when considering other potentially influential variables such as race and obesity, two attributes that can influence asthma prevalence and can vary spatially (U.S. EPA, 2018, section 4.1.2). Family income level was used to represent spatial variability in asthma prevalence and may, in some instances, capture spatial variability in race and obesity (Ogden et al., 2010), and thus to some extent, reasonably represent the potential influence race and obesity have on asthma prevalence. However, instances where these influential variables are not fully represented in simulating the at-risk population, and where populations identified by such variables are associated with increased asthma prevalence that may spatially intersect with the highest ambient air concentrations, could lead to uncertainty in estimated exposures and health risk. Further characterization could be appropriate by comparing with local prevalence rates stratified by a similar collection of influential variables, where such data exist.	Yes. Recent year data used (2013-2017) (Attachment 1)
APEX: Microenvironmental Concentrations	Outdoor Near-road and Vehicle PE and PROX Factors	Both	Low	Low - Medium	Uncertainty in mean PROX value used is approximately 15 percentage points (Figure 10 and Table 7 of Langstaff (2007)). Factor may be of greater importance in certain study areas or under varying conditions, though even with this mean difference, in-vehicle penetration/decay decreases exposures and hence the importance of in-vehicle microenvironments. Further, considering that the exposures of interest need to be concomitant with elevated exertion, the accurate estimation of exposures occurring inside vehicles is considered relatively unimportant. This uncertainty could be important for exposure events that occur outdoors near roads (i.e., PE factor = 1) and when simulated individuals might be at elevated exertion for long durations. That said, the frequency of these specific events is likely low, but nevertheless unquantified at this time.	No
	Indoor: Air Exchange Rates	Both	Low	Medium	Uncertainty due to random sampling variation via bootstrap distribution analysis indicated the AER geometric mean (GM) and standard deviation (GSD) uncertainty for a given study area tends to range from ± 1.0 GM and ± 0.5 GSD hr ⁻¹ (Langstaff, 2007). Some of the eight study areas used AER from a geographically similar city. Non-representativeness remains an important issue as city-to-city variability can be wide ranging (GM/GSD pairs can vary by factors of 2-3) and data available for city-specific evaluation are limited (Langstaff, 2007). The restaurant and school AER distributions are derived from small samples and may not be representative of all possible types of restaurants and schools, in general. That said, indoor microenvironments are considered less likely to contribute to an individual's daily maximum 7-hr average O ₃ exposure while at elevated exertion levels and likely does not contribute substantially to uncertainty in the exposure and risk estimates.	Yes. New distribution used for restaurant and school ME (section 3D.2.6.1.1).

Sources of Uncertainty		Uncertainty Characterization				Newly Characterized/ New Information?
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Indoor-Residence: A/C Prevalence	Both	Low	Low	Data were obtained from a reliable source, are comprehensive, and subject to quality control (U.S. Census Bureau, 2019). For six of the of the eight study areas, A/C prevalence was available for 2015 and 2017, while for the remaining two study areas (Sacramento and St. Louis), the most recent year data available was 2011. There is uncertainty associated with the use of an A/C prevalence derived from a different year than the years simulated in the two study areas due to changes in housing stock that may occur over time. That said, indoor microenvironments are considered less likely to contribute to an individual's daily maximum 7-hr average O ₃ exposure while at elevated exertion levels and likely do not contribute substantially to uncertainty in the exposure and risk estimates.	No
	Indoor: Removal Rate	Both	Low	Medium	Greatest uncertainty in the input distribution regarded representativeness, though estimated as unbiased but correct to within 10% (Langstaff, 2007).	No
APEX: Simulated Activity Profiles	Longitudinal Profiles	Under	Low - Medium	Medium	The magnitude of potential influence for this uncertainty would be mostly directed toward estimates of multiday exposures. Simulations indicate the number of single day and multiday exposures of interest can vary based on the longitudinal approach selected (Che et al., 2014). As discussed in section 3D.2.5.4, the D&A method provides a reasonable balance of this exposure feature. Note however, long-term diary profiles (i.e., monthly, annual) do not exist for a population, thus limiting the evaluation. Further, the general population-based modeling approach used for main body results does not assign rigid schedules, for example explicitly representing a 5-day work week for employed people.	No
	Commuting	Both	Low	Medium	Method used in this assessment (and used previously in the 2014 HREA) is designed to link Census commute distances with CHAD vehicle drive times. This is considered an improvement over the former approach that did not match commute distance and activity time. While vehicle time is accounted for through diary selection, it is not rigidly scheduled. However, accurate estimation of exposures occurring while inside vehicles is considered relatively unimportant because it is unlikely to occur while at elevated exertion.	No
	Activity Patterns for Simulated At-Risk Population	Both	Low	Low - Medium	Analyses of activity patterns of people with asthma are similar to that of individuals not having asthma regarding participation rate in outdoor activities and exertion level (section 3D.2.5.3; see also 2014 HREA, Appendix G, Tables 5G-2 to 5G-5).	No
APEX: Physiological Processes	Body Weight (NHANES)	Unknown	Low	Low	Comprehensive and subject to quality control, appropriate years selected for simulated population, though possible small regional variation is possibly not well-represented by national data (U.S. EPA, 2018, Appendix G.)	Yes. Recent year data used (2009-2014) (U.S. EPA (2018), Appendix G)
	NVO _{2max}	Unknown	Low	Low	Upper bound control for unrealistic activity levels rarely used by model, thus likely not very influential.	No

Sources of Uncertainty		Uncertainty Characterization				Newly Characterized/ New Information?
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Resting Metabolic Rate (RMR)	Unknown	Low	Low	New, improved algorithm used for the current O ₃ exposure and risk analysis (U.S. EPA 2018, Appendix H). Comprehensive literature review resulted in construction of large data base used to derive new RMR equations. Equations consider variables most influential to RMR (i.e., age, body weight, and sex). There are other factors that could affect intra-personal variability in RMR such as time-of-day (Haugen et al., 2003) or seasonal/temperature influences (van Ooijen et al., 2004; Leonard et al., 2014). Variability from these and other potentially influential factors may be indirectly accounted for by the residual error term used in the RMR Equation 3D-2 depending on the extent to which these influential factors varied across the clinical study data that were used to create the RMR analytical data set. However, because there is inadequate information regarding the presence of multiple RMR measurements for individual study subjects, we could not estimate intra-individual variability nor could we use these influential factors, other than age and sex, as explanatory variables in the RMR equation. Therefore, any influences on spatial variability in RMR, both within and among the eight study areas, would largely be driven by the spatial distribution of age and sex.	Yes. Recent data and new equations (U.S. EPA (2018), Appendix H).
	METS Distributions	Over	Low - Medium	Medium	In a prior characterization of uncertainty in METs, APEX estimated daily mean METs range from about 0.1 to 0.2 units (between about 5-10%) higher than independent literature reported values (Table 15 of Langstaff, 2007). Some of the diary activities in CHAD encompassed broad categories (e.g., 'play sports', 'travel, general') and as such METs distributions were developed using multiple activities, some of which that could vary greatly in magnitude. Since the 2014 HREA, the list of CHAD activities (and corresponding METs values) were expanded from 142 to 320, by evaluating available diary comment details and disaggregating the originally assigned broad activities to more specific activities (see Attachment 3 and U.S. EPA, 2019c. New distributions were developed using METs estimates provided by Ainsworth et al. (2011). It is expected that the added specificity and redevelopment of METs distributions would more realistically estimate activity-specific energy expenditure. Two important uncertainties remain: the application of literature provided longer-term average METs values to short-term events and the extrapolation of METs data provided for adults to children. However, shorter-term values are of greater importance in this assessment, thus METs could be better characterized where short-term METS data are available.	Yes. New activity codes and MET distributions (U.S. EPA, 2019a, U.S. EPA, 2019b)

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
	Ventilation Rates	Unknown	Low	Low - Medium	Predictions made using the prior algorithm showed excellent agreement with independent measurement data, particularly when considering simulated study group (Graham and McCurdy, 2009; 2014 HREA Figure 5-23 and Figure 5-24). New algorithm derived using the same data observed to have improved predictability (U.S. EPA, 2018, Appendix H). However, a shorter-term comparison (a single hour rather than daily) of predicted versus measured ventilation rates, while more informative, cannot be performed due to lack of ventilation rate data at this duration and considering influential factors (e.g., age, particular activity performed).	Yes. New equation (U.S. EPA, 2018, Appendix H).
Exposure-based risk	EVR Characterization of Moderate or Greater Exertion	Both	Low	Low - Medium	The 2014 HREA recognized that the simulated number of people achieving this level of exertion could be moderately overestimated, affecting the results for comparison to benchmarks and the population-based E-R approach used to estimate lung function risk. A new approach to identifying when individuals may be at moderate or greater exertion was developed to better address inter-personal variability observed in the controlled human exposure study subjects (Attachment 2). Uncertainty remains in the extrapolation of the observations made from adults and proportionally applied to children.	Yes. New distribution-based approach (Attachment 2).
	Benchmark Concentration Levels for Population Study Groups	Under	Low	Medium	There is only very limited evidence from controlled human exposure studies of population groups potentially at greater risk. Compared to the healthy young adults included in the controlled human exposure studies, members of some populations (e.g., children with asthma) are considered more likely to experience adverse effects following exposures to lower O ₃ concentrations (80 FR 65322, 65346, October 26, 2015; Frey, 2014, p. 7). Although not directly characterized in the 2014 HREA, the benchmark levels derived from the controlled human exposure studies may not be entirely representative of effects likely to be exhibited by the simulated population and could underestimate the size of the population at risk and/or the magnitude of adverse effects.	No
	Exposure Duration	Under	Low	Low	The exposure duration for the studies from which the benchmark concentrations are drawn is 6.6-hr (six 50-min exercise periods separated by 10-min rest periods, and with a 35-min lunch after 3 rd hour). For practical reasons, daily maximum exposures were time averaged over 7 hours (rather than 8 hours previously used) to better relate to the concentrations used for the controlled human exposure study subjects. The whole number 7, was used (rather than 6.6) due to logistical and timeline constraints on implementation of a 6.6-hr duration in the exposure model. Use of 7 hours, while more accurately reflecting the exposure duration in the controlled human exposure studies, would likely underestimate risk relative to directly using 6.6 hours, albeit to a limited extent. Use of 7 hours reduces the magnitude of risk underestimation compared to use of 8 hours (as was done in the prior REAs).	Yes. A 7-hr duration for averaging exposure concentrations was used to better represent 6.6-hr exposures (section 3D.2.8.1)

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
Lung Function Risk Estimation	Contribution to Risk of Exposures at or Below 40 ppb	Over	Low - Medium	Low - Medium	While there is limited support for O ₃ being causally linked to lung function responses at the lowest tested exposure level (i.e., 40 ppb exposures), there are no observations at lower exposures. Data available at 40 ppb are limited to two studies, in one of which O ₃ was administered by facemask and had the only positive response. Because the lung function risk analysis assumes there is an exposure response relationship at exposures below 40 ppb, the influence of this source of uncertainty could possibly contribute to the overestimation of risk when including risk resulting from low exposures. The magnitude of influence appears to be greater for the MSS model estimates when compared to the E-R function estimates.	Yes. New evaluation of the contribution of risk from low exposures. (section 3D.3.4.3)
	Extrapolation of E-R Data from Healthy Subjects to Simulated People with Asthma	Under	Low	Low - Medium	Subjects with asthma in controlled human exposure studies appear to be at least as sensitive to acute effects of O ₃ in terms of FEV ₁ and inflammatory responses as healthy non-asthmatic subjects (2013 ISA, section 8.2.2). Note however, study subjects with asthma are typically characterized as having a "mild" condition, thus, there is uncertainty in how others expressing a more severe condition would respond to similar O ₃ exposures. In addition, many epidemiologic studies report greater risk of health effects among individuals with asthma. Considering each of these elements, a direct extrapolation could understate the at-risk population.	No
	Extrapolation of E-R Data from Adults 18-35) to Children and to Older Adults	Both	Low - Medium	Low	Because the vast majority of controlled human exposure studies investigating lung function responses were conducted with adult subjects, the lung function risk estimates for children, ages 5-18, is based on E-R data from adult subjects to estimate responses in children aged 5-18. However, the few available studies of O ₃ -related lung function decrement in children indicate that children's FEV ₁ responses are similar to those observed in adults 18-35 years old (e.g. McDonnell et al., 1985). Regarding older adults, the evidence indicates a decline in responsiveness with increasing age 18 to 35, followed by a rate of decrease that dampens for ages 36 to 55, and ultimately leads to limited responsiveness in adults >55 years old (2013 ISA, p. 6-22). A newly available study, Arjomandi et al., 2018, found a statistically significant reduction in FEV ₁ (group mean of 1.7%) for older adults (mean age 59.9) following 3-hr exposures of 120 ppb O ₃ with exercise (EVR of 15-17 L/min-m ²), although statistical significance was not found for 3-hr exposures of 70 ppb. Given the 7-hr focus of the current assessment as well as the fact that the air quality scenario for the current standard includes no hours with an ambient air concentration at or above 120 ppb (Appendix 3C, Figures 3C-67 to 3C-74), the setting of the age term at zero for older adults appears to remain appropriate for the simulated exposure conditions.	No

Sources of Uncertainty		Uncertainty Characterization				Newly Characterized/ New Information?
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty	Comments	
Category	Element	Direction	Magnitude			
	Assumed No Interaction of other Co-pollutants on O ₃ -related Lung Function Responses	Under	Low	Medium	There are a few studies regarding the potential for an increased response to O ₃ when exposure is in the presence of other common pollutants such as particulate matter (potentially including particulate sulfur compounds), nitrogen dioxide, and sulfur dioxide, although the studies are limited (e.g., with regard to relevance to ambient air exposure concentrations) and/or provide inconsistent results.	No
	Statistical model for E-R Function	Both	Low	Low	The selection of statistical model to best reflect the E-R relationship can influence risk estimates, particularly for instances when large proportions of the simulated population are exposed to low-level concentrations. The 90/10 logit/linear model (section 3D-2.8.2.1) yielding an E-R function similar in shape to an E-R function developed using a probit link (a commonly used fitting method), would tend to estimate lower risk than a function based on a logistic fit (which would have a relatively higher response at low level exposures). Overall, the relatively low contribution of low-level exposures to risk when using the E-R function approach indicates the selection of the 90/10 logit/linear fit to have a limited impact on uncertainty in risk estimates.	Yes. Section 3D.3.4.2.1
	Statistical Uncertainty in E-R Function	Both	Low - Medium	Low	A BMCMC approach was used to iteratively generate 9,000 unique E-R functions section 3D.2.8.2.1). We used the median (50 th percentile) function for generating population-based (E-R function) lung function risk in the main body results. A 95% confidence interval for risk estimates was generated using the 2.5 th and 97.5 th percentile E-R functions. Overall, the range of risk estimates using the confidence intervals was small, on the order of a few percentage points, but increased in relative magnitude when considering the larger lung function decrements.	Yes. section 3D.3.4.2.2
	Contribution of Low-level Ventilation Rate in MSS model Estimated Risk	Over	Low - Medium	Low - Medium	We evaluated the role of ventilation rate in estimating risk with the MSS model approach (section 3D.2.8.2.2) by comparing risk generated using either of two model conditions: risk for when simulated individuals experienced decrements at any ventilation rate, or risk for when ventilation rate was at moderate or greater exertion (the latter reflects the E-R function risk approach). The MSS model risk estimates were about 20-40% lower when selecting for simulated individuals at moderate or greater exertion compared with MSS model risk estimates using individuals at any ventilation rate (Table 3D-69). Even when including only individuals at higher exertion rates, the MSS model risk estimates are still a factor of three or more higher than risks estimated using the E-R function risk approach. Given that the controlled human exposure studies indicate an importance of elevated ventilation in combination with the studied exposure concentrations, the MSS model likely overestimates risk.	Yes. section 3D.3.4.2.4

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
	Variability Parameter Setting in MSS Model	Over	Medium	Low - Medium	<p>The value of the MSS model variable U (Equations 3D-15 and 3D-16) is randomly assigned from a distribution to simulated individuals and is meant to address inter-individual variability not accounted for by the other MSS model variables. The influence of U was qualitatively evaluated by examining example time series for two children simulated with different values for U and for which similar-sized lung function decrements are predicted. While both children had similar exposure profiles in terms of duration exposed to elevated concentrations, the ventilation duration at peak concentrations differed. The difference observed (Figure 3D-18) suggests that random assignment of high U values leads to simulated individuals being predicted to experience lung function decrements at relatively lower time-averaged breathing rates as those with a lower U value. Given the difference of these exposure conditions from those in which such decrements are observed in controlled human exposure studies, it is likely that the risk is overestimated, and the amount of overestimation may be similar to that described for ventilation rate in the preceding entry. A second variable v_1, a constant, is used by the MSS model to address intra-individual variability (Equation 3D-16). Because the v_1 is described as representing the non-ozone related contribution to response variability in the study observations (McDonnell et al., 2013), a non-zero setting may contribute to over estimates in risk. We found estimated risks using v_1 set to zero to be about 20-35% lower than when using the default parameter setting (the setting used for the main results in section 3D.3.3.2).</p>	Yes. section 3D.3.4.2.5

Sources of Uncertainty		Uncertainty Characterization			Newly Characterized/ New Information?	
		Influence of Uncertainty on Exposure Risk Estimates		Knowledge-base Uncertainty		Comments
Category	Element	Direction	Magnitude			
	Statistical and Model Uncertainty in MSS model	Both	Low	Low	<p>Glasgow and Smith (2017) evaluated statistical uncertainty in the MSS model employed by APEX. Multiple sets of lung function risk results were generated using random draws of the MSS model coefficients (considering their standard errors) and performing APEX simulations for children ages 5-17 and for 2010 air quality just meeting a design value of 75 ppb in Atlanta. Calculated bounds on the risk estimates could extend to as low as 0% and >35% of children experiencing at least one decrement $\geq 10\%$ (Glasgow and Smith (2017), Figure 1). While the bounds were wide ranging (and affecting mostly the lowest decrement size), the reported median risk estimate (18.1%) is similar to that estimated in the 2014 HREA. Note, these central tendency risk values are based on using the best estimates of the MSS model coefficients and are derived from the existing controlled human exposure study data. It is possible that with new controlled human study observations, these model coefficients (and associated standard errors) could possibly change, resulting in a shift of central tendency risk estimates in either direction (greater or lower frequency of lung function decrements) while also changing the outer bounds (increasing or decreasing the confidence intervals). Even so, the outer bounds of any risk estimates, based on the current MSS model or a newly derived MSS model, are generated by using a distribution of coefficient values, the bounds of which have a lesser probability of occurrence compared to those generated using the central tendency values. Further, Glasgow and Smith (2017) also evaluated MSS model uncertainty using two different parameterizations (one including BMI as an explanatory variable, the other not). Comparison of median risk values for the two parameterizations ranged from fractions to a few percentage points, with the largest difference reported for the lowest decrement and overall, lower values were reported for the MSS model that includes a BMI variable. We note the risk results generated in our assessment are based on the MSS model that includes a BMI variable. While uncertainty in the MSS model risk estimates could be further characterized (e.g., including the type of analyses reported by Glasgow and Smith, 2017), that would not be expected to change the overall conclusion that there is relatively greater uncertainty associated with the MSS model estimates than with the E-R model estimates.</p>	Yes (Glasgow and Smith (2017))

3D.3.4.2 Targeted Evaluations of Lung Function Risk Models

The intent of the following targeted evaluations is to provide insight into a few of the important uncertainties identified in section 3D.3.4.1 concerning the lung function risk estimates. Analyses were designed to inform how the uncertainties may influence the exposure and risks reported in section 3D.3. Results or estimates generated in these targeted evaluations do not replace (nor supplement) the results in section 3D.3, nor do they address all aspects of the exposure and risk assessment. Further, because the main body results indicated children were estimated to experience lung function decrements more frequently than adults, we focused these targeted evaluations on simulations with children.

Briefly, we performed five targeted evaluations with each discussed in the following sections. The first section discusses the statistical model used to represent the E-R function (section 3D.3.4.2.1). The next section discusses the development and interpretation of confidence intervals for the lung function risk estimates generated using the population-based E-R function (section 3D.3.4.2.2). This is followed by a section describing an evaluation of the contribution of low-level exposures to risk estimated using the population-based E-R function and the individual MSS model lung function risk approaches (section 3D.3.4.2.3). Section 3D.3.4.2.4 evaluates the role moderate or greater ventilation has in estimating risk using the MSS model. And finally, a discussion and evaluation of variability parameters used in the MSS model is presented in section 3D.3.4.2.5.

3D.3.4.2.1 Statistical Model Used for the E-R Function

There are several approaches available to fit data to a continuous E-R function, for example, regression models (linear, logistic) and use of curve smoothing techniques (moving averages, polynomial splines). Logistic regression is commonly used for concentration-, exposure-, and dose-response relationships when study observations contain a binary dependent variable (e.g., either yes/no response). A logistic regression can be fit using a varied linking approach, such as logit or probit, the selection of which can depend on assumptions made regarding the distribution of responses (logistic or inverse normal, respectively)⁸³ among other factors (e.g., model fit statistics).

The statistical model selected for the E-R function and used to estimate the frequency of lung function decrements in this exposure and risk analysis is the same as that used in the 2014 and 2007 REAs and was based on combining logistic (with a logit link) and two-piece linear

⁸³ For example, regarding the development of an E-R function describing lung function decrements associated with exposure to SO₂ for the risk assessment performed in the 2012 review of the SO₂ NAAQS (U.S. EPA, 2009), the CASAC for that review (Samet, 2009) suggested that the distribution of individual response thresholds supported use of a probit function rather than a logistic function (pp. 14 and 60-63).

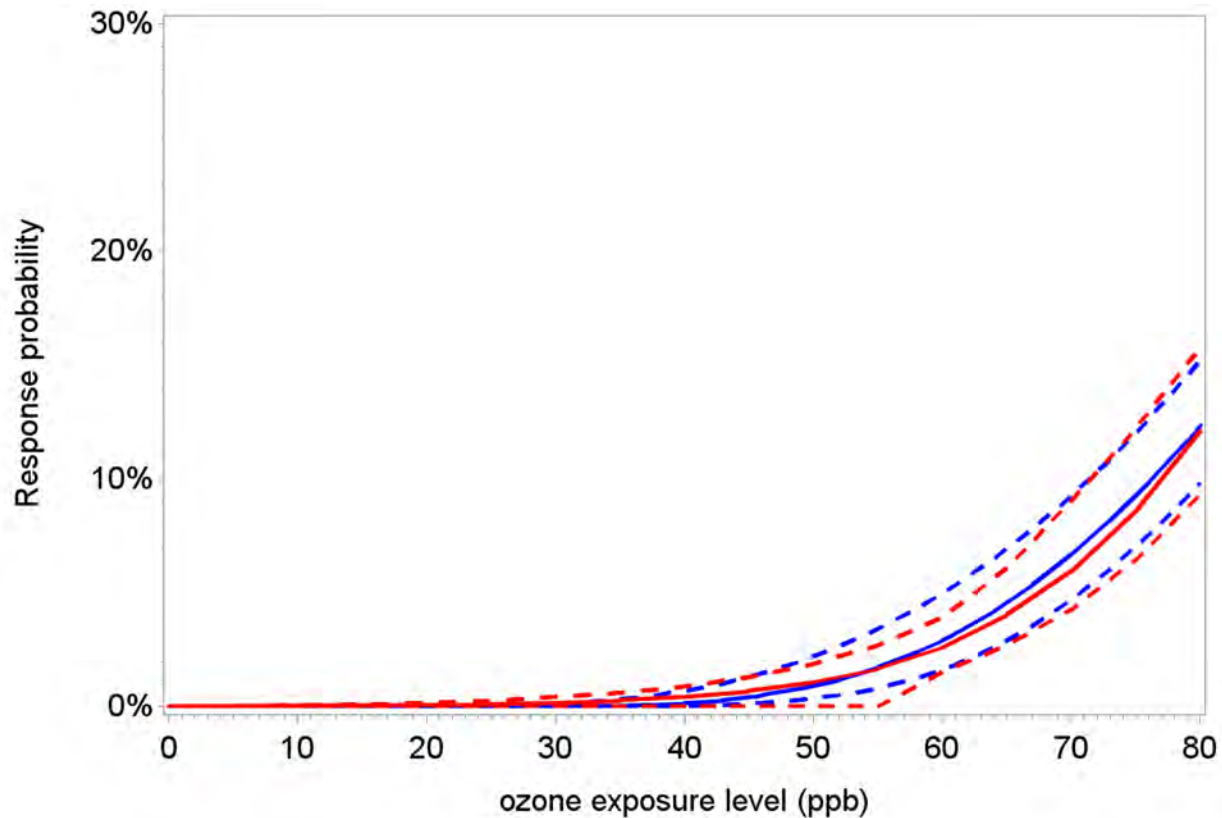
1 forms in a 90/10 percent proportion, respectively, using a Bayesian Markov Chain Monte Carlo
2 (BMCMC) modeling approach (section 3D.2.8.2.1). The selection of this model was based on
3 advice received from the CASAC review in the 2008 O₃ NAAQS review (Henderson, 2006) and
4 evaluation of the curve fit statistics for this function (U.S. EPA, 2007a; 2014 HREA).

5 Of practical importance for this assessment is how the response curve is extrapolated
6 from the lowest observed exposure to zero exposure/response. For general context, in comparing
7 a probit to a logit link in a logistic regression, the probit link would yield a relatively lower
8 response at the lowest level exposures. The two-piece linear model used in part for developing
9 the current E-R function resembles a hockey stick, with the paddle representing a zero response
10 for the lowest exposures, and the handle representing the increased response that coincides with
11 increasing exposures, beginning at the junction between the paddle and the stick.⁸⁴ Based on this
12 statistical form, when combining the logit linked logistic model with a hockey stick type model
13 (as done for this assessment), it was assumed the 90/10 percent proportion logit/linear E-R form
14 would have a response curve shape for low-level exposures similar to that using a probit link. To
15 better evaluate these E-R functions, we fit the controlled human exposure study data (Figure 3D-
16 12) using a probit link and compared that with the 90/10 logit/linear curve.

17 As an example, Figure 3D-14 illustrates the E-R functions fit from these two approaches,
18 using the $\geq 15\%$ lung function decrement observations. Plotted for the probit approach is the
19 curve derived from the best estimate of the model coefficients, along with 95% confidence
20 intervals derived from the model coefficient variability. For the 90/10 logit/linear approach, the
21 median (50th percentile) function is plotted, along with 95% confidence intervals derived from
22 the 2.5th and 97.5th percentile E-R functions obtained from the 9,000 BMCMC model iterations.
23 As expected, the probit curve is very similar to the 90/10 logit/linear curve, albeit with the
24 former having a response just below the latter for the lower exposures. The opposite occurs for
25 exposures above 55 ppb; for those higher exposures, a relatively greater response is indicated
26 using the 90/10 logit/linear E-R function. Based on there being little difference between the two
27 curves and only slight off-setting of the response at different exposure levels, it is likely that
28 applying a probit fit for the E-R function to the population distribution of daily maximum 7-hr
29 exposures would result in little to no difference from the risk estimates derived with the 90/10
30 logit/linear E-R function.⁸⁵

⁸⁴ The combined two-piece linear/logistic E-R function is used, as described in section 3D.2.8.2.1 above, because of the limited controlled human exposure study data, and associated uncertainty regarding the response, at low level exposures (i.e., <60 ppb). Note, the two-piece linear model has a lower percent contribution (10%) compared to that of the non-threshold logistic model (90%) in deriving the combined E-R function.

⁸⁵ Evaluation of the E-R functions fit for the 10% decrement indicated that the 90/10 logit/linear curve had a somewhat higher response than the probit curve at the low-level exposures (and lower response at exposures >55



1
 2 **Figure 3D-14. Comparison of a probit function curve (blue line) with the Bayesian**
 3 **logistic/linear function curve (red) in estimating the probability of lung**
 4 **function decrements $\geq 15\%$ (based on data in Table 3D-20). Confidence**
 5 **intervals for the probit model reflect variability in the regression model**
 6 **coefficients.**

7 **3D.3.4.2.2 Confidence Intervals for the Population-based E-R Function**
 8 **and Effect on Lung Function Risk Estimates**

9 To estimate lung function risk using the population-based E-R function approach, the
 10 results of which are presented in the main body of this document (section 3D.3.3.1), we selected
 11 the median (50th percentile) E-R function originally developed as part of the 2014 HREA. This
 12 E-R function was derived from Bayesian Markov Chain Monte Carlo (BMCMC) approach that
 13 iteratively combined logistic and linear E-R functions fit to the controlled human exposure study
 14 data in Table 3D-20 (section 3D.2.8.2.1). The selection of the median E-R function to estimate
 15 risk in the current assessment generally assumes the simulated at-risk population is comprised of

ppb). For the 20% decrement, the probit curve was similar to the 90/10 logit/linear curve at low-level exposures, but slightly higher for exposures between 50 and 70 ppb. Given the smallness of the difference and limited contribution of the lower exposures to the risk estimates (Table 3D-66), these finding does not imply significant uncertainty or support generation of new simulations and risk estimates using the probit E-R function.

1 individuals that have a similar response frequency as that of the general collection of controlled
2 human exposure study test subjects.

3 Because there were two or more studies reporting observed responses at most of the
4 exposure levels and the BMCMC approach generates numerous E-R functions, statistical
5 uncertainty in the E-R function can be used to approximate lower and upper bounds to the lung
6 function risk estimates. To evaluate such bounds here, a 95% confidence interval for lung
7 function risk was estimated by combining the population distribution of daily maximum 7-hr
8 exposures (occurring while at moderate or greater exertion) for simulated children in each study
9 area with the 2.5th and 97.5th percentile population-based E-R functions (2014 HREA, Appendix
10 6A, Table 6A-1). Lung function risk estimates based on these lower and upper percentile
11 functions and those based on the median function (for air quality just meeting the existing
12 standard) are presented, in terms of the minimum and maximum year results, in Table 3D-65 for
13 each of the three lung function decrements (i.e., FEV₁ decrements ≥ 10 , 15, and 20%). The
14 estimates for the median E-R function are drawn from Table 3D-40. The estimates for the best
15 and worst air quality years yield the minimum and maximum estimates for each of the three
16 functions providing a range for estimates based on each of the particular E-R functions.

17 The range of values for the estimated risk generated by each of the E-R functions as a
18 result of using different air quality years (i.e., the distance in estimated risk between the
19 minimum and maximum values) is small, on the order of a few tenths of a percentage point, with
20 the smallest range of values associated with the largest lung function decrement (Table 3D-65).
21 In general, the range of the overall 95% confidence interval (i.e., the distance in estimated risk
22 between the 2.5th and 97.5th values) is also small when considering percentage points. For
23 example, the lower bound percent of children estimated to experience at least one lung function
24 decrement $\geq 10\%$ for the Atlanta study area is about 1.5% and the upper bound value is about
25 4.0% (median E-R function value $\sim 2.4\%$). With increasing magnitude of the decrement, the
26 range of percentage points becomes smaller (e.g., a $\geq 20\%$ decrement has a range of about 0.6
27 percentage points for Atlanta). In terms of relative magnitude, one might consider this range
28 large (i.e., a factor of 6 or more), but because there are so few children estimated to experience
29 these large lung function decrements, this interpretation of the confidence interval would be
30 inappropriate. Further, it would be unreasonable to simply assume that use of the lower and
31 upper bounds of the E-R functions would appropriately estimate lower and upper bounds of risk
32 without additional context regarding the controlled human exposure study data, the interpretation
33 of the bounds on the E-R function, and how these might relate to statistical uncertainty in the risk
34 estimates.

1 **Table 3D-65. Percent of children estimated to experience at least one lung function**
 2 **decrement at or above the indicated level, for air quality adjusted to just meet**
 3 **the current standard, using the population-based (E-R function) risk**
 4 **approach.**

FEV ₁ Decrement	Study Area	Percent of Children Estimated to Experience at Least One Decrement per Year using Specified E-R Functions ^A					
		Lower Bound (2.5%) E-R Function		Median (50%) E-R Function ^B		Upper Bound (97.5%) E-R Function	
		min ^C	max ^C	min	max	min	max
≥10%	Atlanta	1.0	1.5	1.9	2.5	3.1	4.0
	Boston	1.2	1.4	2.0	2.3	3.3	3.8
	Dallas	1.2	1.6	2.1	2.6	3.5	4.3
	Detroit	1.4	1.8	2.3	2.8	3.9	4.5
	Philadelphia	1.3	1.4	2.2	2.4	3.6	3.9
	Phoenix	1.8	2.2	2.9	3.3	4.8	5.4
	Sacramento	1.2	1.4	2.2	2.3	3.6	3.8
	St. Louis	1.4	1.8	2.3	2.8	3.8	4.5
≥15%	Atlanta	>0.1	0.2	0.4	0.6	0.7	1.1
	Boston	0.1	0.2	0.5	0.6	0.8	1.0
	Dallas	0.1	0.2	0.5	0.7	0.8	1.1
	Detroit	0.1	0.3	0.6	0.8	1.0	1.2
	Philadelphia	0.1	0.1	0.5	0.6	0.9	1.0
	Phoenix	0.2	0.3	0.7	0.9	1.2	1.5
	Sacramento	0.1	0.1	0.5	0.6	0.9	1.0
	St. Louis	0.1	0.3	0.6	0.8	0.9	1.2
≥20%	Atlanta	>0.1	0.1	0.1	0.2	0.4	0.7
	Boston	>0.1	0.1	0.2	0.2	0.5	0.7
	Dallas	>0.1	0.1	0.2	0.3	0.5	0.8
	Detroit	0.1	0.1	0.2	0.3	0.7	0.9
	Philadelphia	>0.1	>0.1	0.2	0.2	0.6	0.6
	Phoenix	0.1	0.1	0.3	0.4	0.8	1.1
	Sacramento	>0.1	>0.1	0.2	0.2	0.5	0.6
	St. Louis	>0.1	0.1	0.2	0.3	0.6	0.9

^A Calculated percent is rounded to the nearest tenth decimal using conventional rounding. Values equal to zero are designated by "0" (there are no individuals experiencing decrements at that level). Small, non-zero values that do not round upwards to 0.1 (i.e., <0.05) are given a value of "<0.1".

^B The median function is used to generate E-R function risk estimates reported in the main body results. Note, these are identical to the results reported in Table 3D-40.

^C The minimum (min) are results for the best air quality year and the maximum (max) are results for the worst air quality year of the three years simulated.

5
 6 As a reminder, while the controlled human exposure study subjects are volunteers (and
 7 assumed to be selected at random), it is important to note there are important fundamental biases
 8 in their collective composition: none of the individuals have known preexisting health conditions

1 (e.g., cardiovascular disease, asthma) and all of the subjects are required to be physically fit
2 enough to meet a study's exercise target levels. Clearly, not every member of the simulated
3 population has these attributes, but the risk approach does select for when simulated individuals
4 are at moderate or greater exertion while exposed, as was done for the controlled human
5 exposure study subjects. Therefore, representation of potentially sensitive individuals (i.e., those
6 with pre-existing health conditions) in the study data and thus, in the derived E-R functions, is
7 absent.

8 In addition, use of this type of statistical approach to estimate lower and upper bounds of
9 lung function risk does not suggest that the range of functions could be equally applied to the
10 simulated population as a whole (e.g., that the entire population could have a risk as low as X or
11 as high as Y , based on the 2.5th and 97.5th percentile functions selected, respectively) nor does the
12 range of E-R functions likely represent individuals that are least sensitive, or more importantly
13 (given the NAAQS review context for these analyses), those most sensitive to O₃ exposure. The
14 variability in the observed response in study subjects at given O₃ exposures can be due to many
15 factors (e.g., uncertainties in exposure conditions, response/concentration measurements, study
16 subject sensitivity for healthy individuals, number of subjects per study, etc.). When used in such
17 an analysis here, one might suggest the lower and upper bounds account for some of these
18 uncertainties, however, they would be bounded by their collective representativeness and actual
19 weighting of these uncertainties present in the study observations. In its application, it would be
20 assumed that the distribution of the features of the study subjects are similarly reflected in the
21 simulated population, which as described above, is not entirely the case.

22 Further, the range of functions used to represent lower and upper bounds is derived from
23 a distribution of functions. If there were a perfect matching of the study subject attributes with
24 those of the simulated population, the risks estimated using either end of the 95% interval for the
25 E-R function would certainly have a much smaller likelihood and better apply to a smaller
26 proportion of the population, than those estimated from using the median E-R function. That
27 said, even in the absence of perfectly matching the attributes of the controlled human exposure
28 study subjects with those of the simulated population, the median E-R function may be most
29 appropriate to estimate lung function risk for the simulated population as a whole. Still, the
30 median E-R function may be underestimating the number/percent of individuals experiencing
31 decrements to the extent that the general population includes individuals that would experience
32 greater decrements than experienced by individuals represented in the controlled human
33 exposure studies. Further, as recognized in Chapter 3 of the main document, similarly sized
34 decrements in individuals with compromised respiratory function or in individuals with asthma
35 may be more likely to elicit other, perhaps more significant, health outcomes.

3D.3.4.2.3 Contribution of Low-Level Exposures to Lung Function Risk Estimates

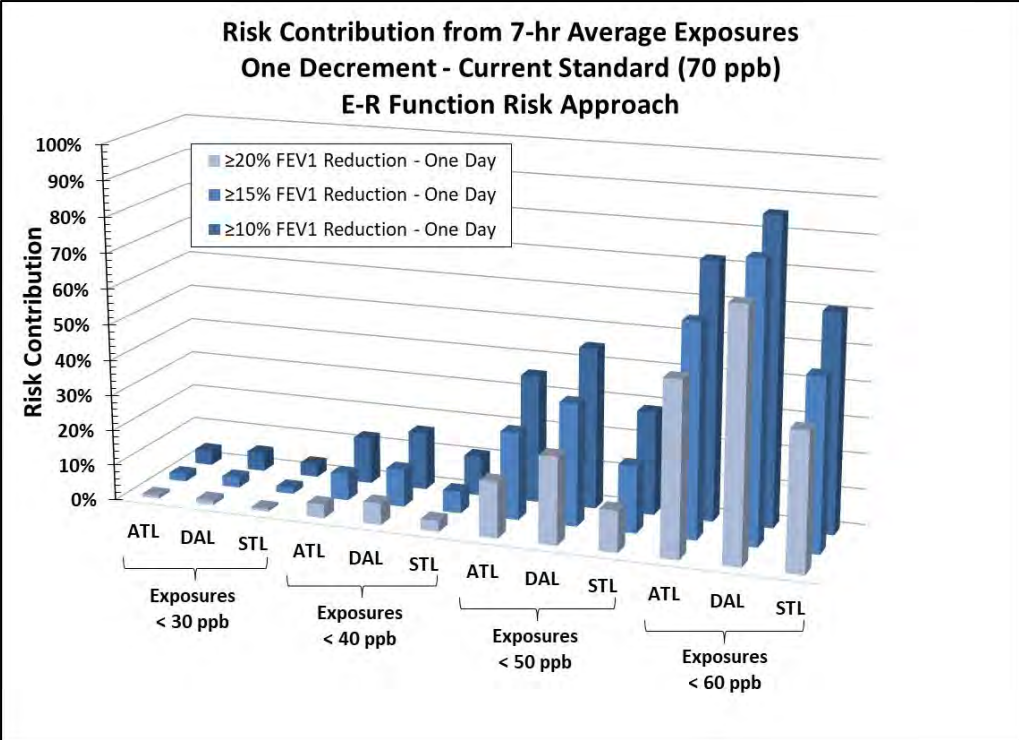
The two approaches used to estimate lung function risk were evaluated to better understand how the distribution of exposures influences the estimated risk. For the first approach that used the population-based E-R function to estimate risk, we evaluated the risk contribution resulting from each of the daily maximum 7-hr exposure levels that occur while at elevated risk. Because the continuous function used is extrapolated from the lowest observed exposure (40 ppb) in the controlled human exposure studies to zero, of particular interest here were the contributions from low exposures to the estimated risk where there are no controlled human study data available (i.e., O₃ exposures <40 ppb, 6.6-hrs). Further, because there were only two studies that included exposures of 40 ppb (one that elicited decrements between 10 and 15% in two study subjects, with no statistical significance at the group mean level, the other eliciting no decrement of at least 10% in any subjects), we also evaluated the contribution to estimated risk resulting from exposures ≥ 50 ppb and ≥ 60 ppb.

The APEX exposure output for the E-R function approach that were the basis for the main results reported in section 3D.3.3 are in a format useful for calculating the risk contribution from each 7-hr average exposure bin (0 to 160 ppb, in 10 ppb increments), thus no new APEX simulations were needed for this evaluation. However, given the objectives for this evaluation, time limitations on it, and that new simulations were required to evaluate the MSS model approach (see below), we focused on three of the eight study areas for this evaluation. These areas were selected at random (i.e., Atlanta, Dallas, and St. Louis), and simulations were performed for a single year (2016). The results for this evaluation are provided in Table 3D-66 for the three study areas, three air quality scenarios using 2016 data, and focusing on the risk contribution to lung function decrements occurring at least one and two days per year. Figure 3D-15 illustrates the same results, but for air quality just meeting the current standard.

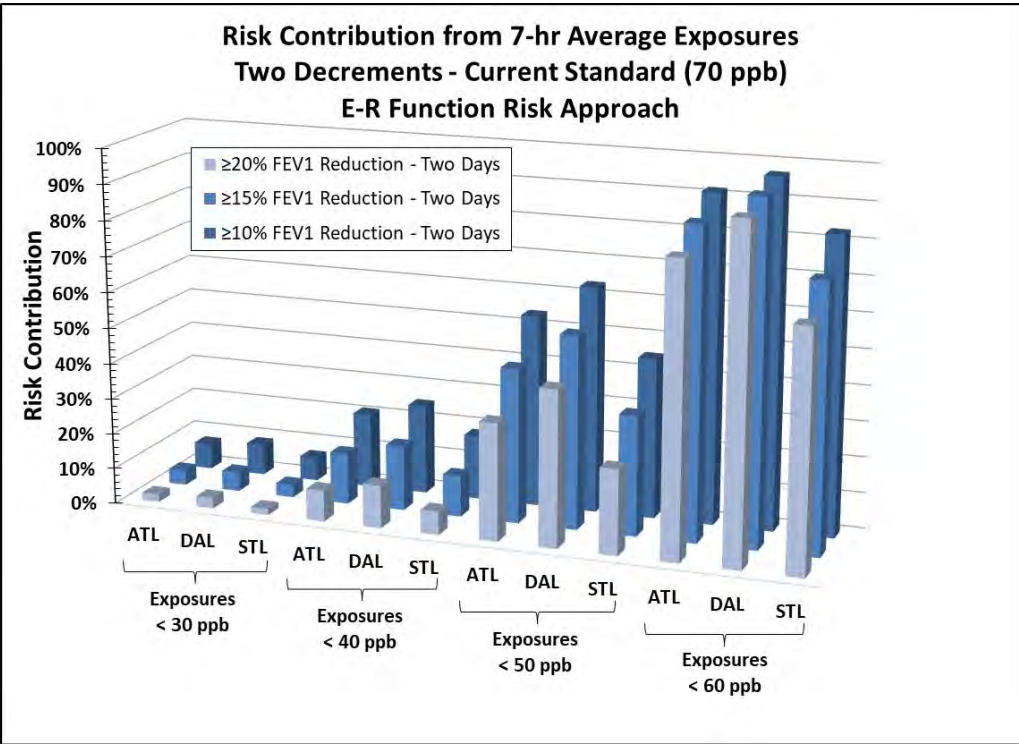
There is variability in the risk contribution across the three study areas, variability which increases with increasing magnitude of the lung function decrement and increasing O₃ exposures across the three air quality scenarios. The risk estimated from 7-hr average exposures below 40 ppb is generally low and is lower for higher magnitudes of the lung function decrement and higher air quality scenario design value. That said, the majority of risk (84 to 98%) is attributed to 7-hr average exposures ≥ 40 ppb for any of the air quality scenarios. The risk contribution attributed to 7-hr average exposures ≥ 60 ppb varies greatly across the study areas, the magnitude of the decrements, and the air quality scenarios. For example, on average about 37% of the risk is contributed by 7-hr average exposures ≥ 60 ppb. But in Dallas, the contribution from these exposures is much less (on average about 22%), while in St. Louis, the contribution is much more (on average about 50%).

1 **Table 3D-66. Estimated lung function risk contribution resulting from selected 7-hr**
 2 **average O₃ exposures in children, using the E-R function risk approach, 2016.**

Air Quality Scenario	7-hr Exposure	Study Area	Risk Contribution from Indicated 7-hr Exposure, E-R Function Approach					
			One Decrement/FEV ₁ Reduction			Two Decrements/FEV ₁ Reduction		
			≥10%	≥15%	≥20%	≥10%	≥15%	≥20%
65 ppb	<30 ppb	Atlanta	6.2%	3.5%	1.8%	10.3%	6.7%	3.8%
		Dallas	6.7%	3.9%	2.1%	10.8%	7.1%	4.2%
		St. Louis	5.4%	2.9%	1.4%	9.1%	5.6%	3.0%
	<40 ppb	Atlanta	19.8%	13.4%	8.1%	30.4%	23.3%	16.2%
		Dallas	20.9%	14.7%	9.2%	31.3%	24.3%	17.2%
		St. Louis	16.5%	10.6%	6.1%	25.6%	18.5%	12.0%
	<50 ppb	Atlanta	54.3%	43.1%	32.2%	75.4%	67.7%	58.2%
		Dallas	58.1%	48.1%	37.6%	76.6%	69.5%	60.6%
		St. Louis	43.4%	32.7%	23.3%	62.6%	53.1%	42.7%
	<60 ppb	Atlanta	88.7%	81.9%	74.4%	98.5%	97.4%	95.9%
		Dallas	94.2%	90.2%	85.6%	99.8%	99.6%	99.4%
		St. Louis	83.3%	75.2%	67.7%	96.7%	94.5%	91.8%
Current Standard (70 ppb)	<30 ppb	Atlanta	4.2%	2.0%	0.9%	7.3%	4.2%	2.1%
		Dallas	5.3%	2.8%	1.4%	8.8%	5.4%	2.9%
		St. Louis	3.7%	1.7%	0.7%	6.6%	3.6%	1.7%
	<40 ppb	Atlanta	12.9%	7.5%	3.9%	21.0%	14.4%	8.8%
		Dallas	16.3%	10.5%	6.0%	25.1%	18.2%	11.9%
		St. Louis	11.1%	6.1%	3.1%	18.0%	11.5%	6.6%
	<50 ppb	Atlanta	35.9%	24.5%	15.7%	53.9%	43.3%	32.9%
		Dallas	45.3%	34.4%	24.5%	63.4%	54.0%	43.7%
		St. Louis	29.0%	18.7%	11.6%	44.9%	33.7%	23.8%
	<60 ppb	Atlanta	72.3%	59.7%	48.7%	91.7%	86.6%	81.2%
		Dallas	85.8%	77.9%	69.8%	97.0%	95.0%	92.6%
		St. Louis	61.1%	48.3%	38.5%	82.9%	74.5%	66.5%
75 ppb	<30 ppb	Atlanta	2.9%	1.2%	0.4%	5.2%	2.6%	1.2%
		Dallas	4.3%	2.1%	0.9%	7.4%	4.3%	2.2%
		St. Louis	2.8%	1.2%	0.4%	5.2%	2.6%	1.1%
	<40 ppb	Atlanta	8.6%	4.3%	1.9%	14.7%	8.8%	4.7%
		Dallas	13.1%	7.7%	4.0%	21.0%	14.4%	8.8%
		St. Louis	8.4%	4.1%	1.9%	14.1%	8.1%	4.2%
	<50 ppb	Atlanta	23.6%	13.7%	7.6%	37.6%	26.5%	17.6%
		Dallas	35.6%	24.6%	16.0%	52.9%	42.4%	32.2%
		St. Louis	21.6%	12.5%	7.0%	34.6%	23.5%	15.1%
	<60 ppb	Atlanta	52.8%	37.7%	27.0%	75.8%	64.9%	55.2%
		Dallas	75.1%	63.3%	52.7%	92.0%	87.2%	82.1%
		St. Louis	47.4%	33.6%	24.4%	69.3%	57.2%	47.3%



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3 **Figure 3D-15. Estimated lung function risk contribution resulting from selected 7-hr**
 4 **average O₃ exposures in children, using the E-R function risk approach and**
 5 **air quality adjusted to just meet the current standard, for one decrement (top**
 6 **panel) and two decrements (bottom panel), 2016.**

7

1 As was done with the E-R function results, we evaluated the influence exposure level has
 2 on risks estimated using the MSS model. New APEX simulations were performed to estimate the
 3 continuous hourly time-series of O₃ exposures and FEV₁ decrements. All simulation conditions
 4 remained the same as done for the main body risk results except that for this evaluation, a single
 5 year of air quality (2016) was used and fewer children were simulated to maintain a tractable
 6 analysis (10,000 rather than the 60,000 done for the main body results). Note, there is little
 7 difference in risks estimated when varying the total number of simulated children (Table 3D-67).
 8 Because the risk estimated using the MSS model is calculated from a cumulative time-series of
 9 O₃ exposures (and EVR, along with contributions from other variables used by the MSS model),
 10 we calculated the 7-hr average O₃ exposure occurring just prior to the FEV₁ decrements to allow
 11 for reasonable comparison with the above E-R function risk contribution results.

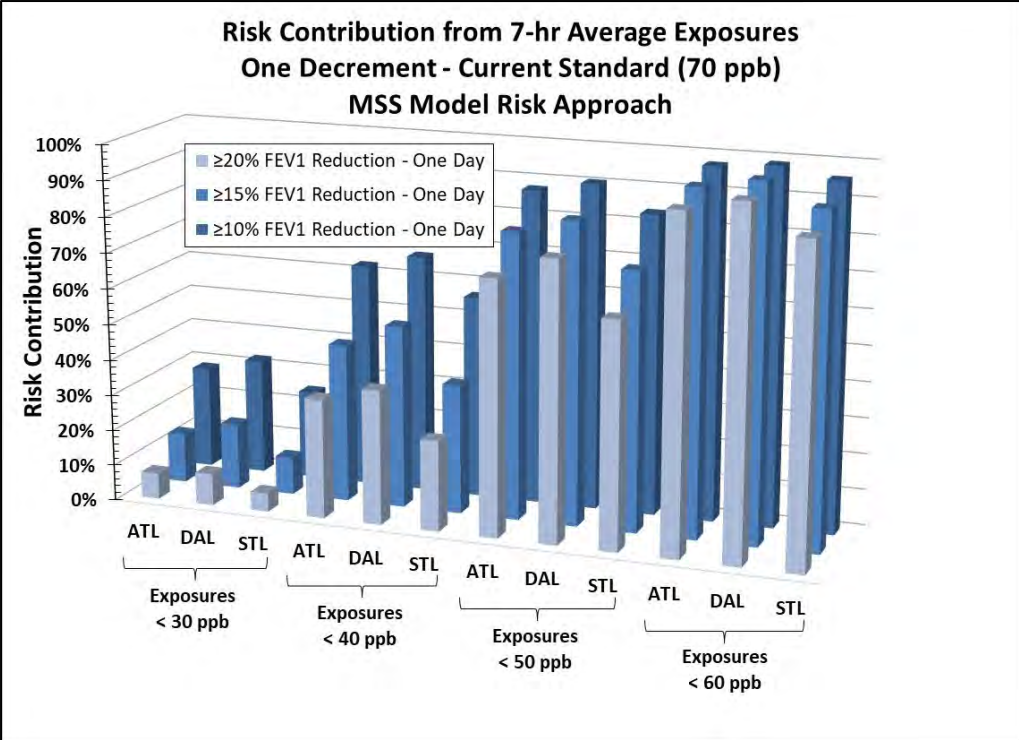
12 Table 3D-68 and Figure 3D-16 present the risk contribution resulting from selected 7-hr
 13 average O₃ exposures that occur prior to a lung function decrement of interest, estimated using
 14 the MSS model. While the general pattern in the risk contributions across the air quality
 15 scenarios, study areas, and decrements are similar to that described above using the E-R function
 16 approach, there are noteworthy differences between the two risk approaches. First, there is less
 17 variability in the risk contribution values across the study areas and decrements when using the
 18 MSS model risk approach. For example, the overall coefficient of variation (COV; standard
 19 deviation/mean) ranges from 1 to 31% (mean 11%) across study areas when evaluating the MSS
 20 model risk contributions, while the COV ranges from 6 to 49% (mean 26%) for the same
 21 evaluation using the E-R function. Second, the MSS model consistently calculates a greater
 22 percent of lung function decrements that result from low O₃ exposures (Table 3D-68) relative to
 23 that estimated when using the E-R function (Table 3D-66). While the majority of risk (84 to
 24 98%, mean 91%) using the E-R function risk approach was attributed to 7-hr average exposures
 25 ≥ 40 ppb, when using the MSS model, between 33 to 75% (mean 54%) of risk is attributed to 7-hr
 26 average exposures ≥ 40 ppb when considering the three air quality scenarios and all three
 27 decrements. Based on this evaluation, the MSS model more frequently predicts responses to
 28 occur at lower O₃ exposures than does the E-R function approach.

29 **Table 3D-67. MSS model risk estimates from varying the number of simulated children.**

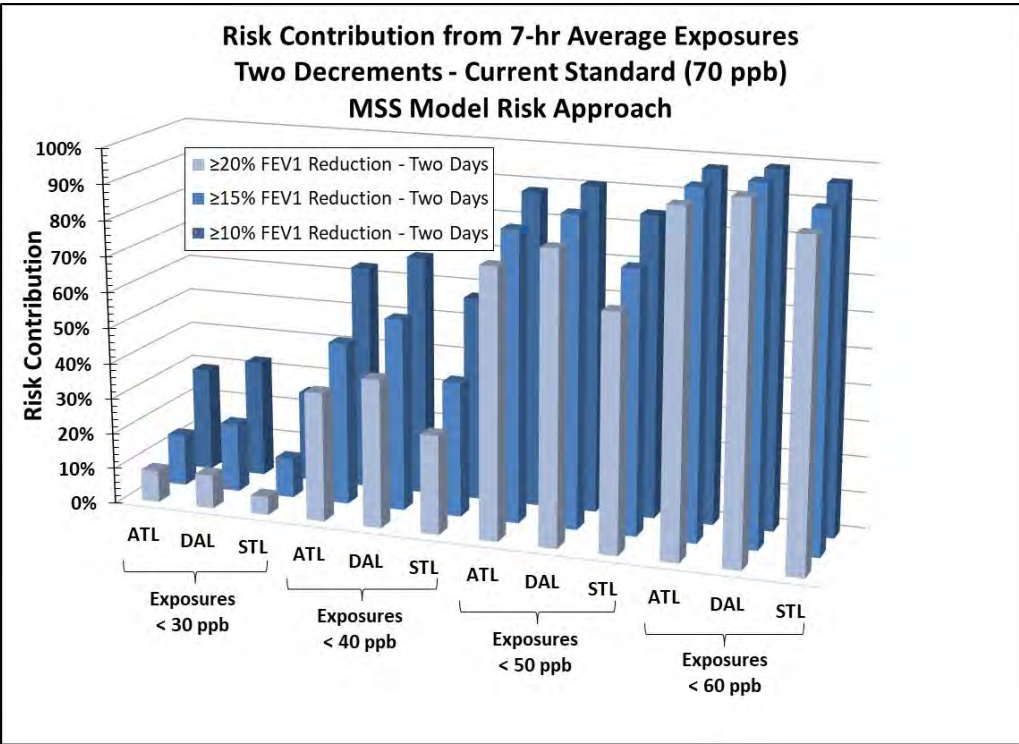
Study Area (2016 AQ)	APEX Simulation, 70 ppb AQ Scenario (number of simulated children)	% of Children Experiencing at least One Decrement		
		FEV ₁ $\geq 10\%$	FEV ₁ $\geq 15\%$	FEV ₁ $\geq 20\%$
Atlanta (worst year)	Sensitivity (n = 10,000)	14.6%	5.1%	2.1%
	Main Results, Table 3D-52 (n = 60,000)	15.1%	5.0%	2.1%
Dallas (best year)	Sensitivity (n = 10,000)	13.3%	4.1%	1.7%
	Main Results, Table 3D-52 (n = 60,000)	13.1%	4.0%	1.6%
St. Louis (worst year)	Sensitivity (n = 10,000)	16.3%	5.8%	2.5%
	Main Results, Table 3D-52 (n = 60,000)	16.3%	5.9%	2.7%

1 **Table 3D-68. Estimated lung function risk contribution resulting from selected 7-hr**
 2 **average O₃ exposures in children, using the MSS model risk approach, 2016.**

Air Quality Scenario	7-hr Exposure	Study Area	Risk Contribution from Indicated 7-hr Exposure, MSS Model Approach					
			One Decrement/FEV ₁ Reduction			Two Decrements/FEV ₁ Reduction		
			≥10%	≥15%	≥20%	≥10%	≥15%	≥20%
65 ppb	< 30 ppb	Atlanta	33.5%	16.2%	10.1%	33.9%	17.4%	10.4%
		Dallas	36.0%	19.6%	8.8%	36.9%	20.6%	9.1%
		St. Louis	29.6%	13.8%	9.0%	30.3%	15.1%	9.4%
	<40 ppb	Atlanta	70.9%	52.9%	41.6%	71.6%	55.4%	44.0%
		Dallas	71.7%	57.2%	38.9%	72.6%	60.6%	42.1%
		St. Louis	64.9%	46.4%	35.1%	65.5%	49.3%	40.3%
	<50 ppb	Atlanta	93.0%	86.8%	81.3%	93.6%	88.9%	84.9%
		Dallas	93.7%	89.2%	81.6%	94.0%	90.9%	86.0%
		St. Louis	89.7%	82.3%	70.9%	90.1%	84.5%	76.5%
	<60 ppb	Atlanta	99.1%	97.9%	96.6%	99.3%	98.0%	96.5%
		Dallas	99.5%	98.7%	97.9%	99.6%	99.1%	99.4%
		St. Louis	98.4%	97.2%	95.9%	98.5%	97.7%	96.6%
Current Standard (70 ppb)	< 30 ppb	Atlanta	28.6%	13.8%	7.3%	29.3%	14.4%	8.9%
		Dallas	32.4%	18.4%	8.8%	33.0%	19.4%	9.5%
		St. Louis	24.8%	10.5%	5.2%	25.5%	11.1%	5.0%
	<40 ppb	Atlanta	62.7%	44.2%	33.4%	63.3%	45.8%	36.2%
		Dallas	66.7%	51.0%	37.5%	67.6%	54.0%	41.5%
		St. Louis	56.6%	36.1%	25.3%	57.4%	37.9%	27.7%
	<50 ppb	Atlanta	87.7%	79.7%	70.9%	88.3%	81.4%	75.1%
		Dallas	90.6%	84.0%	77.5%	91.1%	86.7%	81.1%
		St. Louis	83.5%	72.0%	62.9%	84.4%	73.5%	65.8%
	<60 ppb	Atlanta	97.5%	95.2%	92.5%	97.8%	96.2%	94.6%
		Dallas	98.8%	98.0%	96.0%	99.0%	98.6%	97.8%
		St. Louis	95.9%	91.9%	87.8%	96.2%	92.8%	89.7%
75 ppb	< 30 ppb	Atlanta	25.1%	11.7%	6.5%	25.6%	12.2%	7.2%
		Dallas	29.7%	16.7%	9.0%	30.3%	17.7%	10.2%
		St. Louis	21.9%	9.4%	4.9%	22.4%	10.0%	5.0%
	<40 ppb	Atlanta	55.9%	38.1%	28.2%	56.6%	39.1%	30.0%
		Dallas	62.1%	46.3%	33.0%	63.2%	48.9%	38.0%
		St. Louis	51.9%	32.2%	22.0%	52.6%	33.7%	23.1%
	<50 ppb	Atlanta	81.4%	70.5%	62.9%	82.2%	72.2%	66.1%
		Dallas	87.0%	78.8%	71.2%	87.9%	81.5%	75.8%
		St. Louis	78.3%	63.7%	53.2%	79.1%	66.1%	56.3%
	<60 ppb	Atlanta	94.6%	90.6%	87.2%	95.0%	92.1%	90.4%
		Dallas	97.7%	95.5%	93.0%	98.0%	96.7%	94.7%
		St. Louis	93.1%	87.1%	83.0%	93.6%	88.9%	85.6%



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Figure 3D-16. Lung function risk contribution resulting from selected 7-hr average O₃ exposures in children, using the MSS model risk approach and air quality adjusted to just meet the current standard, for one decrement (top panel) and two decrements (bottom panel), 2016.

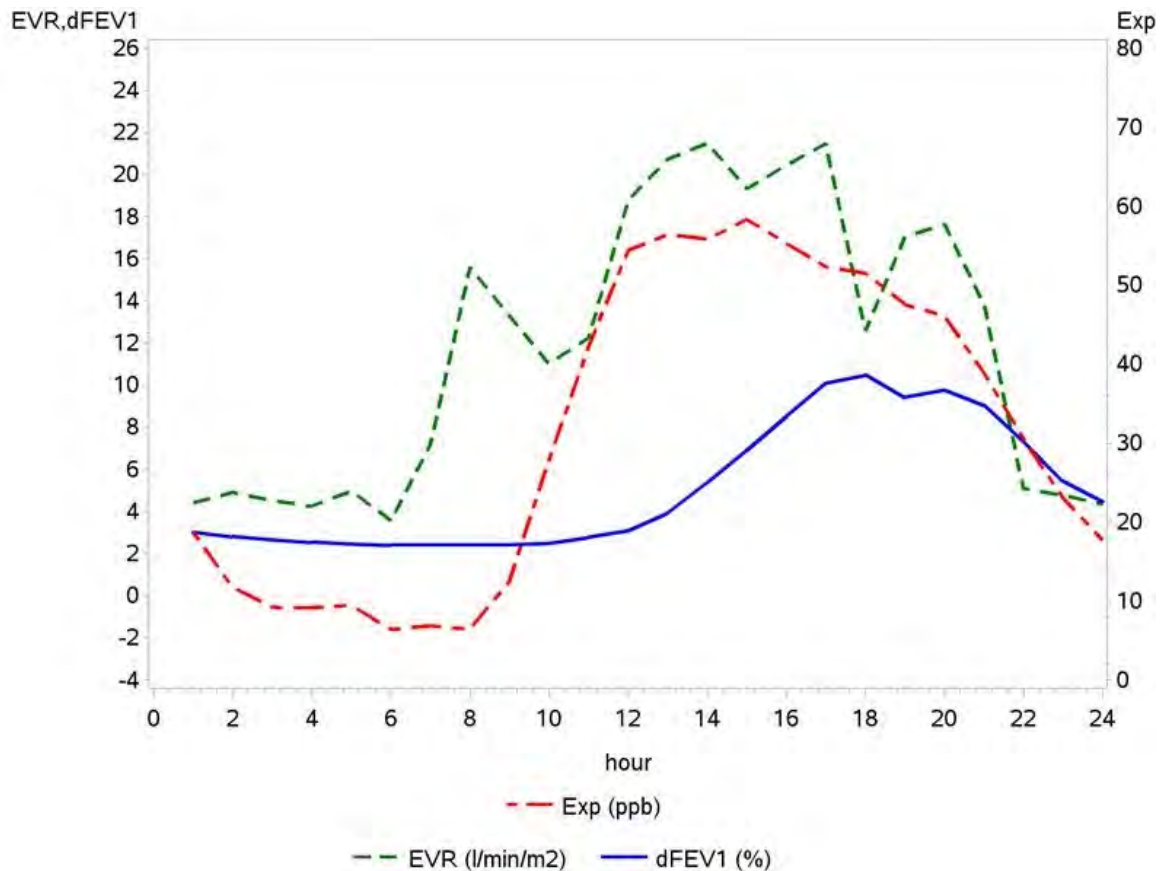
7

3D.3.4.2.4 Influence of Ventilation Rate in Lung Function Risk Estimates

A second important variable used to estimate lung function risk in both the E-R function and MSS model is the ventilation rate. Recall that the E-R function approach uses a threshold value for EVR to designate whether an individual is at moderate or greater exertion ($EVR \geq 17.32 \pm 1.25 \text{ L/min-m}^2$). Technically, while any 7-hr average O_3 exposure can potentially lead to a lung function decrement using the E-R function approach, a lung function decrement is only calculated when individuals are at or above their designated EVR value and when it occurs simultaneously with their daily maximum 7-hr average O_3 exposure. This is not the case with the MSS model lung function risk approach; both O_3 exposure and ventilation rate are considered cumulatively over time (among other influential MSS model variables) and neither of which have a designated level or duration to attain.

Because of this notable difference in the MSS model approach, we first visually evaluated the relationship between the time-series of O_3 exposure and ventilation rate (as represented by EVR), along with the simultaneous occurrence of lung function decrements calculated by the MSS model. Of particular interest to this evaluation was whether the pattern of these variables was correlated, and more importantly, how increases in both exposure and ventilation rates eventually corresponded to increases in the magnitude of the FEV_1 decrement. As was done above to evaluate the risk contribution from selected O_3 exposure levels, we used the same APEX simulation of 10,000 children (and 2016 air quality) which output the hourly time series of O_3 exposure, EVR, and MSS model calculated FEV_1 decrements for each simulated individual. The initial goal was to observe how the MSS model functions and see if there were general patterns in the O_3 exposure, EVR, and FEV_1 reductions.

Figure 3D-17 illustrates an example of the estimated hourly time-series of O_3 exposure, EVR, and FEV_1 decrement for a child considering 2016 air quality adjusted to just meet the current standard in the Atlanta study area. As shown here (and among all other visualizations of children we reviewed that had a lung function decrement of interest), the O_3 exposure and EVR are well correlated with subsequent occurrence of a lung function decrement. With increasing O_3 exposures and breathing rates, there is an increase in the magnitude of the FEV_1 reduction and, following a continuous episode of high exposure along with elevated breathing rate, a lung function decrement of interest is attained (Figure 3D-17).



1
 2 **Figure 3D-17. Example time-series of O₃ exposures, EVR, and FEV₁ reductions estimated**
 3 **using MSS model for a simulated child in the Atlanta study area, based on a**
 4 **day in a year (2016) of the current standard air quality scenario.**

5 When considering the influence of EVR in isolation, we can discern how, in many
 6 instances, the MSS model risk estimates are greater than those estimated using the E-R function
 7 approach when both use a generally similar O₃ exposure profile (i.e., any level, though using
 8 different averaging times). Recall, that the E-R function risk is only estimated for those attaining
 9 moderate or greater exertion levels $EVR \geq 17.32 \pm 1.25$ L/min-m². While there is likely a
 10 minimum EVR in the MSS model, considering both the level and duration, that would lead to
 11 lung function decrements, that minimum is not explicitly defined as it is in the E-R function risk
 12 approach.

13 Note, the E-R method is a fairly direct translation of the controlled human exposure study
 14 data to exposure-dependent response probabilities, particularly considering the strict adherence
 15 to exertion level needed for a response. As described above (section 3D.3.4.2.2), there is low
 16 statistical uncertainty associated with the risk estimates. We already know that relatively lower
 17 ventilation rates substantially influence MSS model risk estimates based on analyses described in
 18 the 2014 HREA (Chapter 6, Tables 6-9 and 6-10). In that assessment, when restricting the MSS

1 results to when an 8-hr EVR of at least 13 L/min-m² was not achieved by simulated individuals
2 (at that time, the threshold for moderate exertion threshold), about 40 to 50% fewer simulated
3 individuals were estimated to experience a lung function decrement, a result better aligned with
4 the E-R function risk results.

5 As a second evaluation of the influence of EVR, a similar evaluation of the degree to
6 which low-level EVRs influence MSS risk estimates was performed here. We limited the
7 evaluation to a single year (2016) of air quality adjusted to just meet the current standard in the
8 three study areas and using the same simulation of 10,000 children described above for
9 generating the hourly data for the MSS model lung function risks. We identified the days when
10 children were exercising at moderate or greater exertion, i.e., 7-hr average EVR ≥ 17.3 L/min-m²
11 and calculated the percent of children experiencing one or more lung function decrements of
12 interest (i.e., $\geq 10\%$, $\geq 15\%$, and $\geq 20\%$). Results for each the main body MSS model approach and
13 the MSS model restricted to children at moderate or greater exertion are presented, along with
14 results using the E-R function risk approach (Table 3D-69).

15 The pattern of risk estimates was consistent across the three study areas. Using the
16 Atlanta study area results as an example, the E-R function risk approach predicts the percent of
17 children experiencing one or more FEV₁ decrements $\geq 10\%$ to be 2.5%, while the main body
18 MSS model risk approach predicts 14.6% of children experience the same decrement (Table 3D-
19 69). When using the MSS model and restricting the risk results to children at moderate or greater
20 exertion, 8.5% of children experiencing one or more FEV₁ decrements $\geq 10\%$. Even with this
21 adjustment for moderate or greater exertion, this indicates an uncertainty in the MSS model
22 estimates such that the MSS model is potentially overpredicting risks for children by about a
23 factor of three or more, particularly when considering the larger lung function decrements.

24 Note that the MSS model used an age-term that extends information developed for 18-
25 year olds to estimate lung function risks in the simulated children (ages 5 to 18). The age term at
26 age 18 is at a maximum value and progressively decreases in value (and hence risk) through age
27 35 adults (the age range of study subjects in the controlled human exposure studies). Therefore,
28 use of this extrapolation might also contribute to some of the noted differences in the two risk
29 approaches because this approach uses the maximum possible observed value. However, the
30 2013 ISA indicates children's responses to O₃ exposure are similar to those for young adults
31 (2013 ISA, section 8.3.1.1), which lends credence to use of the age-term extrapolation in the
32 MSS model and, overall, supports the application of E-R risk approach for children.

33

1 **Table 3D-69. Percent of children experiencing one or more FEV₁ decrements ≥10, 15, 20%,**
 2 **2016 air quality adjusted to just meet the current standard, considering**
 3 **influence of moderate or greater exertion level in the MSS model and E-R**
 4 **function risk approaches.**

Study Area (2016 AQ)	Lung Function Risk Approach	Exertion Level (L/min-m ²)	% of Children Experiencing at least One Decrement		
			FEV ₁ ≥10%	FEV ₁ ≥15%	FEV ₁ ≥20%
Atlanta (worst year)	E-R function ^A	≥17.32 ± 1.25	2.5%	0.6%	0.2%
	MSS model ^B	Any	14.6%	5.1%	2.1%
	MSS model ^C	≥17.3	8.5%	3.5%	1.6%
Dallas (best year)	E-R function	≥17.32 ± 1.25	2.1%	0.5%	0.2%
	MSS model	Any	13.3%	4.1%	1.7%
	MSS model	≥17.3	7.9%	2.9%	1.3%
St. Louis (worst year)	E-R function	≥17.32 ± 1.25	2.8%	0.8%	0.3%
	MSS model	Any	16.3%	5.8%	2.5%
	MSS model	≥17.3	9.7%	3.9%	1.9%

^A The median (50th percentile) E-R function used to generate the main body results (Table 3D-40).

^B Sensitivity results for 10,000 children simulation (Table 3D-67).

^C Screened sensitivity results for only those children achieving moderate or greater exertion level.

3D.3.4.2.5 Influence of MSS Model Variability Parameter Settings

In this evaluation, we considered how the values for two MSS model variables, U and ν_1 , influenced the calculated lung function decrements. These variables are used to account for inter- and intra-individual variability, respectively, in the estimated lung function decrements. Both of these variables are in the 2012 MSS model (McDonnell et al., 2012; and used in the 2014 HREA to estimate lung function risk) and the 2013 MSS model (McDonnell et al. (2013); and used for the current assessment). However, because the 2013 MSS model adjusted the structure of the intra-individual variability to now include two explanatory variables, ν_1 and ν_2 , the interpretation of ν_1 has changed (McDonnell et al. (2013)).⁸⁶ Each of these variables is discussed in greater detail below.

The first variable is U , a random variable meant to address inter-individual variability not accounted for by the other MSS model variables. The impact of the values assigned to U is apparent simply from its roles in the MSS model calculations, as an exponent to the natural logarithm used in estimating the base ΔFEV_1 (Equation 3D-15) and within the calculation of an intra-individual variance term ε (Equation 3D-16). Based on these roles, it is likely that high

⁸⁶ Effectively, in McDonnell et al. (2012), intra-personal variability (ε) was solely represented by ν_1 . In McDonnell et al. (2013), the intra-personal variability (ε) is represented by $\nu_1 + \nu_2 \times (e^{U_i} \times M_{ijk})$ (see Equation 3D-16). According to McDonnell et al. (2013), this was done such that “individuals experiencing small effects either because exposure was low, or because of demographics (e.g. older age) or because baseline value of responsiveness (U_i) was small would be expected to exhibit less variability in response than those with larger mean responses.”

1 values for U would likely yield high lung function decrements, particularly for instances of high
2 O_3 exposures that occur simultaneously with high ventilation rates over a few to several hours.
3 Note that when comparing the variance of U in the 2012 MSS model versus the 2013 MSS
4 model, its standard error is greater (0.917 versus 1.123) in the most recent model.

5 For this evaluation, we used the same APEX simulation (as described in the prior section)
6 of 10,000 children (and 2016 air quality), which output the hourly time series of O_3 exposure,
7 EVR, and FEV_1 decrements for each simulated individual. We screened the output data for
8 simulated individuals having experienced each of the three FEV_1 decrements of interest (i.e.,
9 $\geq 10\%$, $\geq 15\%$, and $\geq 20\%$) and occurring on separate days. We recognize there are a limited
10 number of children experiencing lung function decrements on multiple days per year (e.g., Table
11 3D-57), particularly when considering the highest lung function decrement, but we were
12 interested in controlling for the influence personal variables might have on the magnitude of each
13 of the decrements. We identified a few simulated individual children having multiple decrements
14 at each level of interest, and first visually compared how variation in the value assigned the U
15 variable appeared to influence the magnitude of FEV_1 reduction for the subset of these simulated
16 children that had similar time-series of O_3 exposure and ventilation rate.

17 As an example, Figure 3D-18 illustrates the estimated hourly time-series of O_3 exposure,
18 EVR, and FEV_1 decrement for two simulated children (top and bottom panels) that differ in the
19 value they were assigned for the U variable (both runs used the 2016 year for the current
20 standard air quality scenario for the Atlanta study area). In both cases, the O_3 exposure and EVR
21 are well correlated for each child prior to the occurrence of a lung function decrement, consistent
22 with the controlled human exposure study data. With increasing magnitude of the FEV_1
23 decrement (Figure 3D-18, from left to right panels) there is also progressively higher exposures
24 and breathing rates, each occurring as peak events that continue over a few to several hours just
25 prior to eliciting the indicated FEV_1 decrement of interest. In general, for each of the three
26 magnitudes of FEV_1 decrement, the time-series of O_3 exposures appears similar for the two
27 simulated children – a consistently high exposure maintained across multiple hours for all of the
28 instances where a lung function decrement occurred, with the highest decrement achieved when
29 exposures were also highest. There is however a recognizable difference in the EVR time-series
30 for the two simulated children. For the first child, with the lower value for the U variable (top
31 panels, Figure 3D-18), the peak of the EVR time-series is broader, that is, longer in duration,
32 than it is for the peak EVR for the second child (bottom panels, Figure 3D-18). The peak EVR
33 for the second child (that has the higher value for the U variable) is similar in magnitude to that
34 for the first child, but it does not persist over as long a duration. The figure illustrates this
35 difference for three magnitudes of decrement (10%, 15% and 20%) in vertical pairs of panels
36 from left to right, with the pairs of upper and lower panels differing only by the value of

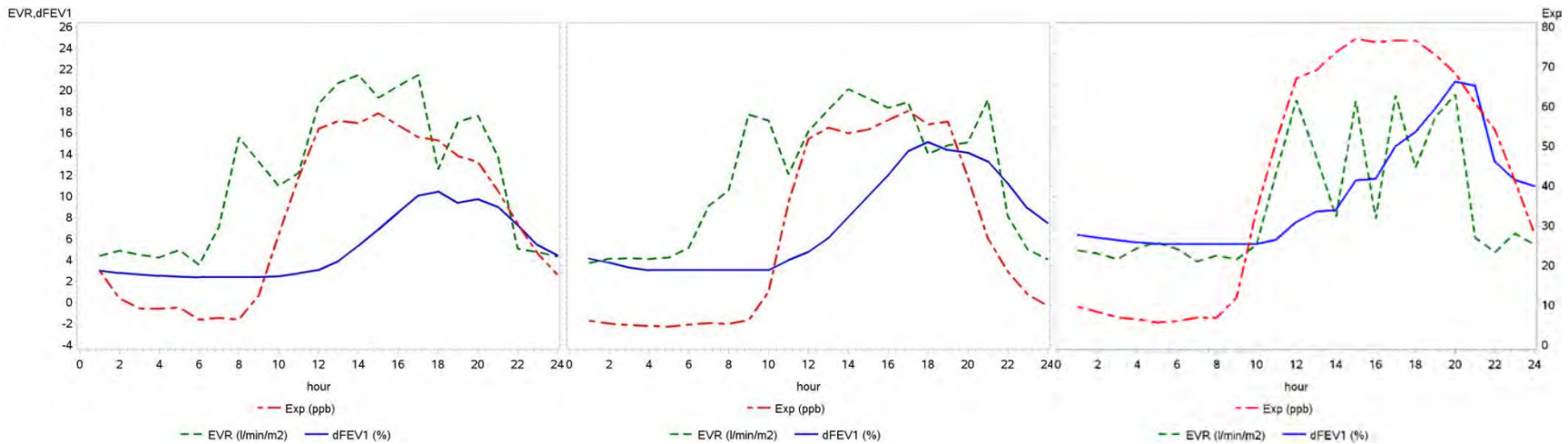
1 parameter U . Specifically, the lower panel child achieves the same decrement as the upper panel
2 child but while having a lower average EVR for the event.

3 The first simulated child (upper panel) has a U value of 0.963, which falls within one
4 standard deviation of the distribution of U (i.e., U has a standard error of 1.123, Table 3D-21).
5 The second (lower panel) simulated child has a U value of 1.78, within the U variable
6 parameterization (i.e., within 2 standard deviations), but is nearly twice that of the first child.
7 Specifically, while the second child has a lower overall “normalized dose” (i.e., $C \times V^{\beta_6}$ in
8 Equation 3D-12) over a similar exposure duration as the first child, the similar risk result is
9 likely a result of the second child being assigned a higher value for U . This higher value of U
10 yielded lung function decrements for the second child similar in magnitude to that predicted for
11 the first simulated child even though the second child had relatively lower doses than the first
12 child for each of the three days.

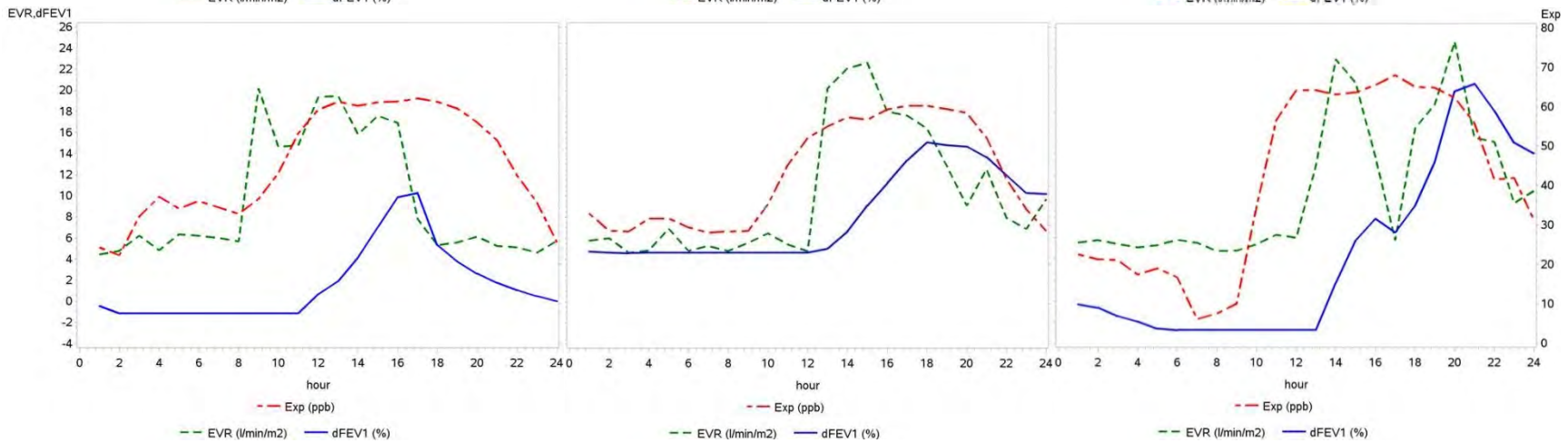
13 The second variable, ν_l , a constant, is used on the calculation of the intra-individual
14 variance term ε (Equations 3D-16). In evaluating the MSS model parameters used for this
15 assessment, McDonnell et al. (2013) notes the estimate of ν_l is consistent with intra-subject
16 FEV₁ variability observed in the forced air trials and below threshold O₃ exposures. The variable
17 ν_l could be interpreted to represent a separate, non-ozone related contribution to response
18 variability in the study observations. This suggests the use of non-zero values for ν_l , as is
19 provided by McDonnell et al. (2013) in MSS model applications (and as was done for the current
20 risk analysis), could lead to a greater number of simulated individuals at or above the lung
21 function decrements (in particular the lowest decrement) and a greater portion of that risk would
22 be attributed to relatively lower exposure levels and ventilation rates, when compared to
23 simulation results having ν_l set as zero.

24 We evaluated the influence that the value of ν_l has on risk estimates. A new APEX
25 simulation was required for this evaluation. All model settings were the same as was done for
26 generating the main assessment results reported in section 3D.3.3.2, except for varying the value
27 of ν_l (the MSS model default ν_l value is 9.112, a new simulation had ν_l set as zero) Again, both
28 simulations were performed for 10,000 children in three study areas (Atlanta, Dallas, and St.
29 Louis) for a simulated year using 2016 air quality adjusted just meet the current standard. Results
30 for this evaluation are presented in Table 3D-70.

1



2



3 **Figure 3D-18. Time-series of O₃ exposures, EVR, and FEV₁ reductions of 10% (left panel), 15% (middle panel), and 20%**
 4 **(right panel) estimated using MSS model for two simulated children (interpersonal variability parameter $U =$**
 5 **0.963, top panel; $U = 1.78$, bottom panel) in the Atlanta study area on three days in a year (2016) of the current**
 6 **air quality scenario.**

1 For each value of ν_1 , there were small differences in estimated risk across the three study
 2 areas. However, setting the ν_1 to zero (compared to the value reported by McDonnell et al.,
 3 2013) resulted in a decrease in the percent of children experiencing lung function decrements of
 4 $\geq 10\%$, $\geq 15\%$, and $\geq 20\%$ of about 35, 22, and 20% (regardless of study area). This reduction in risk is
 5 similar in magnitude to that resulting from excluding the contribution from low-level exposures
 6 (section 3D.3.4.2.3) and not using ventilation rates below moderate or greater exertion (section
 7 3D.3.4.2.4) when estimating lung function decrements using the MSS model.

8 **Table 3D-70. Percent of children experiencing one or more FEV₁ decrements $\geq 10\%$, 15, 20%,**
 9 **2016 air quality adjusted to just meet the current standard, considering the**
 10 **setting of variability parameter, ν_1 , in the MSS model.**

Study Area	MSS Model Parameter Setting ^A	Decrement (FEV ₁ Reduction)		
		$\geq 10\%$	$\geq 15\%$	$\geq 20\%$
Atlanta	$\nu_1 = 9.112$ (default)	15%	5.1%	2.1%
	$\nu_1 = 0$	9.7%	3.9%	1.7%
Dallas	$\nu_1 = 9.112$ (default)	13%	4.1%	1.7%
	$\nu_1 = 0$	7.9%	3.2%	1.3%
St. Louis	$\nu_1 = 9.112$ (default)	16%	5.8%	2.5%
	$\nu_1 = 0$	11%	4.6%	2.1%

^A See Table 3D-21 and Equation 3D-16.

11
12

3D.4 REFERENCES

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APPENDIX 3D, ATTACHMENT 1: ESTIMATING U.S. CENSUS TRACT LEVEL ASTHMA PREVALENCE (2013-2017)

OVERVIEW

This attachment describes the development of the 2013-2017 census tract-level asthma prevalence file used by EPA's Air Pollution Exposure Model (APEX) to identify individuals with asthma during exposure model simulations. The approach used to estimate the APEX file four basic steps: 1) processing National Health Interview Survey (NHIS) regional asthma prevalence data, 2) processing U.S. Census poverty/income status data, and 3) combining the two sets considering variables known to influence asthma (e.g., age, sex, poverty status, U.S. region) to estimate asthma prevalence stratified by age and sex for all U.S. Census tracts, and 4) the NHIS regionally derived data were adjusted to account for state level asthma prevalence data obtained from the Behavioral Risk Factor Surveillance System (BRFSS). Details regarding the data sets and the processing approaches used are provided below.

GENERAL HISTORY

The current NHIS data processing approach is in part based on work originally performed by Cohen and Rosenbaum (2005) and then revised and extended by U.S. EPA (2014, 2018). Briefly, Cohen and Rosenbaum (2005) calculated asthma prevalence for children aged 0 to 17 years for each age, sex, and four U.S. regions using 2003 NHIS survey data.¹ The regions defined by the NHIS were 'Midwest', 'Northeast', 'South', and 'West'. The asthma prevalence was defined as the probability of a 'Yes' response to the question "EVER been told that [the child] had asthma?"² among those persons that responded either 'Yes' or 'No' to this question.³ The responses were weighted to take into account the complex survey design of the NHIS.⁴ Standard errors and confidence intervals for the prevalence were calculated using a logistic model (PROC SURVEY LOGISTIC). A scatterplot technique (LOESS smoother) was applied to smooth the prevalence curves across ages and used to compute the standard errors and

¹ The National Health Interview Survey (NHIS) is the principal source of information on the health of the civilian noninstitutionalized population of the United States and is one of the major data collection programs of the National Center for Health Statistics (NCHS) which is part of the Centers for Disease Control and Prevention (CDC). See <https://www.cdc.gov/nchs/nhis/data-questionnaires-documentation.htm> for data and documentation.

² The response was recorded as variable "CASHMEV" in the downloaded dataset. Data and documentation are available at http://www.cdc.gov/nchs/nhis/quest_data_related_1997_forward.htm.

³ If there were another response to this variable other than "yes" or "no" (i.e., refused, not ascertained, don't know, and missing), the NHIS surveyed individual was excluded from the analysis data set.

⁴ In the SURVEY LOGISTIC procedure, the variable "WTF_SC" was used for weighting, "PSU" was used for clustering, and "STRATUM" was used to define the stratum.

confidence intervals for the smoothed prevalence estimates. Logistic analysis of the raw and smoothed prevalence curves showed statistically significant differences in prevalence by gender and region, supporting their use as stratification variables in the final data set (Cohen and Rosenbaum, 2005). These smoothed prevalence estimates were then used as an input to APEX to estimate air pollutant exposure in children with asthma (U.S. EPA 2007; 2008; 2009).

For the 2014 O₃ REA (U.S. EPA, 2014), we updated the asthma prevalence database used by APEX by combining several years of NHIS survey data (2006-2010). Asthma prevalence for children (by age year) was estimated as described above and, for this update, we also included an estimate of asthma prevalence for adults. In addition, two sets of asthma prevalence for each adults and children were estimated. The first data set, as was done previously, was based on responses to the question “EVER been told that [the child/adult] had asthma”. A second data set was developed using the probability of a ‘Yes’ response to a question that followed those that answered ‘Yes’ to the first question regarding ever having asthma, specifically, do those persons “STILL have asthma?”.⁵ Further, in addition to the nominal variables region and sex, the asthma prevalence were stratified by a income/poverty threshold (i.e., whether the family income was below or at/above the US Census estimate of poverty level for the given year). These 2006-2010 asthma prevalence data were then linked to 2000 U.S. Census tract level income/poverty threshold probabilities, also stratified by age (section 5C-5 of Appendix 5C, US EPA, 2014). Staff considered the variability in population exposures to be better represented when accounting for and modeling these newly refined attributes of this at-risk population. This is was done because of the 1) significant observed differences in asthma prevalence by age, sex, region, and poverty status, 2) the variability in the spatial distribution of poverty status across census tracts, stratified by age, and 3) the potential for spatial variability in local scale ambient concentrations.

And finally, asthma prevalence files used by APEX for the most recent SO₂ REA (Appendix E of U.S. EPA, 2018) were updated in a similar manner using data that reasonably bounded the exposure assessment period of interest (2011-2015) and, as was done for the 2014 O₃ REA, linked the asthma prevalence to the 2010 U.S census tract income to poverty ratio probabilities. The approach to update the asthma prevalence used for the current O₃ REA analyses follows the same approach used previously, although now employs an adjustment to account for local more asthma prevalence information at the state level, rather than relying solely on the regional data. This is described in the four steps that follow below.

Step 1: NHIS Data Set Description and Processing

⁵ The response was recorded as variable “CASSTILL” for children and “AASSTILL” for adults in the respective downloaded datasets. Ultimately, the asthma prevalence used by APEX was based on this variable rather than those using the data for those individuals responding “Yes” to “Ever” having asthma.

The objective of this processing step is to estimate asthma prevalence for children and adults considering several influential variables. First, raw 2013-2017 data and associated documentation were downloaded from the Center for Disease Control (CDC) and Prevention's NHIS website.⁶ The 'Sample Child' and 'Sample Adult' files were selected because of the availability of person-level attributes of interest within these files, i.e., age in years ('age_p'), sex ('sex'), U.S. geographic region ('region'), coupled with the response to questions of whether or not the surveyed individual ever had and still has asthma. In total, five years of survey data were used, comprising nearly 60,000 children and 165,000 adults for years 2013-2017 (Table 1).

Information regarding personal and family income and poverty ranking are also provided by the NHIS in additional survey files. Data files ('INCIMPx.dat') are available for every survey year, each containing either the actual response for the desired financial variable (where provided by survey participant) or the imputed value.⁷ For this current analysis, the ratio of family income-to-poverty was provided as a continuous variable ('POVRATI3') and used to develop a nominal variable for this evaluation: either the survey participant was below or above a selected family income-to-poverty ratio threshold. This was done to be consistent with data generated as part of the next data set processing step, i.e., developing a database containing the census tract level family income-to-poverty ratio probabilities, stratified by age (see Step 2 below).

When considering the number of stratification variables used in the development of the asthma prevalence file (i.e., age years and sex), the level of asthma prevalence (8%, on average), and the distribution of family income-to-poverty ratios among the surveyed population (12%, on average), sample size was an important motivation for aggregating the adult data into age groups. When considering the adult data, there were insufficient numbers of persons available to stratify the data by single age years (for some ages there were no survey persons). Therefore, the adult survey data were grouped into the following age groups: ages 18-24, 25-34, 35-44, 45-54, 55-64, 65-74, and, ≥ 75 .⁸ To increase the number of persons within the age, sex, and four region groupings of our characterization of 'below poverty', the family income-to-poverty ratio threshold was selected as <1.5 , thus including persons that were within 50% above the threshold. For individuals containing the imputed family income information, typically there were 5 estimated values. If the mean of the 5 imputed values were <1.5 , the person's family income was

⁶ Data and documentation are available at http://www.cdc.gov/nchs/nhis/quest_data_related_1997_forward.htm (for 2013-2015, accessed April 11, 2017; for 2016-2017 accessed March 11, 2019).

⁷ Financial information was not collected from all persons; therefore, the NHIS provides imputed data. Details into the available variables and imputation method are provided with each year's data set. For example, see "Multiple Imputation of Family Income and Personal Earnings in the National Health Interview Survey: Methods and Examples" at <https://www.cdc.gov/nchs/data/nhis/tecdoc15.pdf>.

⁸ These same age groupings were used to create the companion file containing the census tract level family income-to-poverty ratio probabilities (Step 2).

categorized ‘below’ the poverty threshold; if the mean of the 5 values were ≥ 1.5 , the person’s family income was categorized ‘above’ the poverty threshold.

These processed person-level income files were then merged with the ‘Sample Adult’ and ‘Sample Child’ files using the ‘HHX’ (a household identifier), ‘FMX’ (a family identifier), and ‘FPX’ (an individual identifier) variables. Note, all persons within the ‘Sample Adult’ and ‘Sample Child’ files had corresponding financial survey data.

As was done for previous asthma prevalence data analysis, two asthma survey response variables were of interest in this analysis and were used to develop the two separate prevalence data sets for each children and adults. The response to the first question “Have you EVER been told by a doctor or other health professional that you [or your child] had asthma?” was recorded as variable name ‘CASHMEV’ for children and ‘AASMEV’ for adults. Only persons having responses of either ‘Yes’ or ‘No’ to this question were retained to estimate the asthma prevalence. This assumes that the exclusion of those responding otherwise, i.e., those that ‘refused’ to answer, instances where it was “not ascertained’, or the person ‘does not know’, does not affect the estimated prevalence rate if either ‘Yes’ or ‘No’ answers could actually be given by these persons. There were very few persons providing an unusable response (Table 1), thus the above assumption is reasonable. A second question was asked as a follow to persons responding “Yes” to the first question, specifically, “Do you STILL have asthma?” and noted as variables ‘CASSTILL’ and ‘AASSTILL’ for children and adults, respectively. Again, while only persons responding ‘Yes’ and ‘No’ were retained for further analysis, the representativeness of the screened data set is assumed unchanged from the raw survey data given the few persons in each survey year having unusable data.

Table 1. Number of total surveyed persons from NHIS (2013-2017) sample adult and child files and the number of those responding to asthma survey questions.

Children	2013	2014	2015	2016	2017	TOTAL
All Children	12,860	13,380	12,291	11,107	8,845	58,483
Yes/No to Ever Have Asthma	12,851	13,366	12,281	11,098	8,832	58,428
Yes/No to Still Have Asthma	12,844	13,359	12,269	11,087	8,823	58,382
Adults						
All Adults	34,557	36,697	33,672	33,028	26,742	164,696
Yes/No to Ever Have Asthma	34,525	36,667	33,641	33,007	26,720	164,560
Yes/No to Still Have Asthma	34,498	36,615	33,614	32,959	26,681	164,367

Logistic Models

As described in the previous section, four person-level analytical data sets were created from the raw NHIS data files, generally containing similar variables: a ‘Yes’ or ‘No’ asthma

response variable (either ‘EVER’ or ‘STILL’), an age (or age group for adults), their sex (‘male’ or ‘female’), US geographic region (‘Midwest’, ‘Northeast’, ‘South’, and ‘West’), and poverty status (‘below’ or above’). One approach to calculate prevalence rates and their uncertainties for a given sex, region, poverty status, and age is to calculate the proportion of ‘Yes’ responses among the ‘Yes’ and ‘No’ responses for that demographic group, appropriately weighting each response by the survey weight. This simplified approach was initially used to develop ‘raw’ asthma prevalence rates however this approach may not be completely appropriate. The two main issues with such a simplified approach are that the distributions of the estimated prevalence rates would not be well approximated by normal distributions and that the estimated confidence intervals based on a normal approximation would often extend outside the [0, 1] interval. A better approach for such survey data is to use a logistic transformation and fit the model:

$$Prob (asthma) = \exp(beta) / (1 + \exp(beta)),$$

where beta may depend on the explanatory variables for age, sex, poverty status, or region. This is equivalent to the model:

$$Beta = \text{logit} \{prob (asthma)\} = \log \{prob (asthma) / [1 - prob (asthma)]\}.$$

The distribution of the estimated values of *beta* is more closely approximated by a normal distribution than the distribution of the corresponding estimates of *Prob (asthma)*. By applying a logit transformation to the confidence intervals for *beta*, the corresponding confidence intervals for *Prob (asthma)* will always fall within [0, 1]. Another advantage of the logistic modeling is that it can be used to compare alternative statistical models, e.g., as models where the prevalence probability depends upon age, region, poverty status, and sex, or on age, region, poverty status but not sex.

In earlier analyses using the NHIS asthma prevalence data, a variety of logistic models were developed and evaluated for use in estimating asthma prevalence, where the transformed probability variable beta is a given function of age, gender, poverty status, and region (Cohen and Rosenbaum, 2005; U.S. EPA, 2014). The SAS procedure SURVEYLOGISTIC was used to fit the various logistic models, taking into account the NHIS survey weights and survey design (using both stratification and clustering options), as well as considering various combinations of the selected explanatory variables.

As an example, Table 2 lists the models fit and their log-likelihood goodness-of-fit measures using the ‘Sample Child’ data set and for the “STILL” asthma response variable using the 2013-2017 NHIS data. A total of 32 logistic models were fit, depending on the inclusion of selected explanatory variables and how age was considered in the model. The ‘Strata’ column lists the eight possible stratifications: no stratification, stratified by sex, by region, by poverty status, by region and sex, by region and poverty status, by sex and poverty status, and by region,

gender and poverty status. For example, “5. region, sex” indicates that separate prevalence estimates were made for each combination of region and gender. As another example, “2. sex” means that separate prevalence estimates were made for each sex, so that for each sex, the prevalence is assumed to be the same for each region. Note the prevalence estimates are independently calculated for each stratum. The ‘Description’ column of Table 2 indicates how beta depends upon the age:

<i>Linear in age</i>	<i>Beta = $\alpha + \beta \times \text{age}$, where α and β vary with strata</i>
<i>Quadratic in age</i>	<i>Beta = $\alpha + \beta \times \text{age} + \gamma \times \text{age}^2$ where α, β and γ vary with strata</i>
<i>Cubic in age</i>	<i>Beta = $\alpha + \beta \times \text{age} + \gamma \times \text{age}^2 + \delta \times \text{age}^3$ where α, β, γ, and δ vary with the strata</i>
<i>f(age)</i>	<i>Beta = arbitrary function of age, with different functions for different strata</i>

The category *f(age)* is equivalent to making age one of the stratification variables, and is also equivalent to making beta a polynomial of degree 17 in age (since the maximum age for children is 17), with coefficients that may vary with the strata. The fitted models are listed in order of complexity, where the simplest model (model 1) is a non-stratified linear model in age and the most complex model (model 32) has a prevalence that is an arbitrary function of age, sex, poverty status, and region. Model 32 is equivalent to calculating independent prevalence estimates for each of the 288 combinations of age, sex, poverty status, and region.

Table 2 also includes the -2 Log Likelihood statistic, a goodness-of-fit measure, and the associated degrees of freedom (DF), which is the total number of estimated parameters. Any two models can be compared using their -2 Log Likelihood values: models having lower values are preferred. If the first model is a special case of the second model, then the approximate statistical significance of the first model is estimated by comparing the difference in the -2 Log Likelihood values with a chi-squared random variable having *r* degrees of freedom, where *r* is the difference in the DF (hence a likelihood ratio test). For all pairs of models from Table 2, all the differences in the -2 Log Likelihood statistic are at least 50,000 and thus are significant at p-values well below 1 percent. Based on its having the lowest -2 Log Likelihood value, the last model fit (model 32: retaining all explanatory variables and using *f(age)*) was preferred and used to estimate the asthma prevalence in the prior analyses⁹ as well as employed for this 2013-2017 NHIS data analysis.

⁹ Similar results were obtained when estimating prevalence using the ‘EVER’ have asthma variable as well as when investigating model fit using the adult data sets. In the Cohen and Rosenbaum (2005) analysis, adult data were not used and the family income-to-poverty ratio was not a variable in their models. Also, because age was a

Table 2. Logistic models and model fit statistics for estimating child asthma prevalence using the “STILL” asthma response variable from 2013-2017 NHIS data.

Model	Description	Strata	- 2 Log Likelihood	DF
1	1. logit(prob) = linear in age	1. none	209411405	2
2	1. logit(prob) = linear in age	2. gender	208645067	4
3	1. logit(prob) = linear in age	3. region	209056169.8	8
4	1. logit(prob) = linear in age	4. poverty	208433518.7	4
5	1. logit(prob) = linear in age	5. region, gender	208230032	16
6	1. logit(prob) = linear in age	6. region, poverty	207999872.9	16
7	1. logit(prob) = linear in age	7. gender, poverty	207630301.3	8
8	1. logit(prob) = linear in age	8. region, gender, poverty	207046731.4	32
9	2. logit(prob) = quadratic in age	1. none	207554776.3	3
10	2. logit(prob) = quadratic in age	2. gender	206754508.8	6
11	2. logit(prob) = quadratic in age	3. region	207092990.7	12
12	2. logit(prob) = quadratic in age	4. poverty	206568831.2	6
13	2. logit(prob) = quadratic in age	5. region, gender	206177195.9	24
14	2. logit(prob) = quadratic in age	6. region, poverty	205966568.6	24
15	2. logit(prob) = quadratic in age	7. gender, poverty	205719195.5	12
16	2. logit(prob) = quadratic in age	8. region, gender, poverty	204888997.5	48
17	3. logit(prob) = cubic in age	1. none	207244848.3	4
18	3. logit(prob) = cubic in age	2. gender	206429982.6	8
19	3. logit(prob) = cubic in age	3. region	206770493.7	16
20	3. logit(prob) = cubic in age	4. poverty	206240699	8
21	3. logit(prob) = cubic in age	5. region, gender	205817245.3	32
22	3. logit(prob) = cubic in age	6. region, poverty	205532902.7	32
23	3. logit(prob) = cubic in age	7. gender, poverty	205380882.1	16
24	3. logit(prob) = cubic in age	8. region, gender, poverty	204406907.3	64
25	4. logit(prob) = f(age)	1. none	206929745.9	18
26	4. logit(prob) = f(age)	2. gender	205902376.7	36
27	4. logit(prob) = f(age)	3. region	205961955.1	72
28	4. logit(prob) = f(age)	4. poverty	205783757.8	36
29	4. logit(prob) = f(age)	5. region, gender	204430849.5	144
30	4. logit(prob) = f(age)	6. region, poverty	204133603.6	144
31	4. logit(prob) = f(age)	7. gender, poverty	204565028.6	72
32	4. logit(prob) = f(age)	8. region, gender, poverty	201725493.2	288

categorical variable in the adult data sets in U.S. EPA (2014, 2018) and analyses conducted here, it could only be evaluated using $f(\text{age_group})$.

The SURVEYLOGISTIC procedure produces estimates of the beta values and their 95% confidence intervals for each combination of age, region, poverty status, and gender. By applying the inverse logit transformation,

$$\text{Prob (asthma)} = \exp(\text{beta}) / (1 + \exp(\text{beta})),$$

one can convert the beta values and associated 95% confidence intervals into predictions and 95% confidence intervals for the prevalence. The standard error for the prevalence was estimated as:

$$\text{Std Error } \{\text{Prob (asthma)}\} = \text{Std Error (beta)} \times \exp(-\text{beta}) / (1 + \exp(\text{beta}))^2,$$

which follows from the delta method (i.e., a first order Taylor series approximation).

Estimated asthma prevalence using this approach and termed here as ‘unsmoothed’ are provided in the supplement at the end of this document. Graphical representation is provided in a series of figures incorporating the following variables:

- Region
- Gender
- Age (in years) or Age_group (age categories)
- Poverty Status
- Prevalence = predicted prevalence
- SE = standard error of predicted prevalence
- LowerCI = lower bound of 95% confidence interval for predicted prevalence
- UpperCI = upper bound of 95% confidence interval for predicted prevalence

A series of plots are provided per figure that vary by the four regions and two income-to-poverty ratios. Historically, we have used the prevalence results based on the ‘STILL’ have asthma variable. Supplemental Figures S-1 through S-4 show the estimated prevalence for children and adults by age (or age-group), stratified by gender. Data used for each figure/plot (as well as plots for the ‘EVER’ variable) can be provided upon request.

Loess Smoother

The estimated prevalence curves show that the prevalence is not necessarily a smooth function of age. The linear, quadratic, and cubic functions of age modeled by SURVEYLOGISTIC were identified as a potential method for smoothing the curves, but they did not provide the best fit to the data. One reason for this might be due to the attempt to fit a global regression curve to all the age groups, which means that the predictions for age *A* are

affected by data for very different ages. A local regression approach that separately fits a regression curve to each age A and its neighboring ages was used, giving a regression weight of 1 to the age A , and lower weights to the neighboring ages using a tri-weight function:

$$\text{Weight} = \{1 - [|\text{age} - A| / q]^3\}, \text{ where } |\text{age} - A| \leq q.$$

The parameter q defines the number of points in the neighborhood of the age A . Instead of calling q the smoothing parameter, SAS defines the smoothing parameter as the proportion of points in each neighborhood. A quadratic function of age to each age neighborhood was fit separately for each gender and region combination. These local regression curves were fit to the beta values, the logits of the asthma prevalence estimates, and then converted them back to estimated prevalence rates by applying the inverse logit function $\exp(\text{beta}) / (1 + \exp(\text{beta}))$. In addition to the tri-weight variable, each beta value was assigned a weight of $1 / [\text{std error}(\text{beta})]^2$, to account for their uncertainties.

In this application of LOESS, weights of $1 / [\text{std error}(\text{beta})]^2$ were used such that $\sigma^2 = 1$. The LOESS procedure estimates σ^2 from the weighted sum of squares. Because it is assumed $\sigma^2 = 1$, the estimated standard errors are multiplied by $1 / \text{estimated } \sigma$ and adjusted the widths of the confidence intervals by the same factor.

There are several potential values that can be selected for the smoothing parameter; the optimum value was determined by evaluating three regression diagnostics: the residual standard error, normal probability plots, and studentized residuals. To generate these statistics, the LOESS procedure was applied to estimated smoothed curves for beta, the logit of the prevalence, as a function of age, separately for each region, gender, and poverty classification. For the children data sets, curves were fit using the choices of 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, and 1.0 for the smoothing parameter. This selected range of values was bounded using the following observations. With only 18 points (i.e., the number of single year ages for children), a smoothing parameter of 0.2 cannot be used because the weight function assigns zero weights to all ages except age A , and a quadratic model cannot be uniquely fit to a single value. A smoothing parameter of 0.3 also cannot be used because that choice assigns a neighborhood of 5 points only ($0.3 \times 18 = 5$, rounded down), of which the two outside ages have assigned weight zero, making the local quadratic model fit exactly at every point except for the end points (ages 0, 1, 16 and 17). Usually one uses a smoothing parameter below 1 so that not all the data are used for the local regression at a given x value. Note also that a smoothing parameter of 0 can be used to generate the raw, unsmoothed, prevalence. The selection of the smoothing parameter used for the adult curves would follow a similar logic, although the lower bound could effectively be extended only to 0.9 given the number of age groups. This limits the selection of smoothing

parameter applied to the two adult data sets to a value of 0.9, though values of 0.8 – 1.0 were nevertheless compared for good measure.

The first regression diagnostic used was the residual standard error, which is the LOESS estimate of σ . As discussed above, the true value of σ equals 1, so the best choice of smoothing parameter should have residual standard errors as close to 1 as possible. For children ‘EVER’ having asthma and when considering the best models (of the 112 possible, those having $0.95 < RSE < 1.05$) using this criterion, the best choice varies with gender, region, and poverty status between smoothing parameters of 0.5 and 0.6 (Table 3). For the ‘STILL’ data set, a value of 0.7 or 0.8 would be slightly preferred. Both the ‘EVER’ and ‘STILL’ adult data sets had, at best, only one model with an RSE within the set criterion, and could be smoothed using a value of 0.8.

Table 3. Top model smoothing fits where residual standard error at or a value of 1.0.

Study Group	Asthma Question	Smoothing Parameter						
		0.4	0.5	0.6	0.7	0.8	0.9	1.0
Children	EVER	1	4	4	3	2	3	4
	STILL	3	3	3	4	4	3	2
Adults	EVER	n/a ^A	n/a	n/a	n/a	1	0	1
	STILL	n/a	n/a	n/a	n/a	1	0	1

^A n/a is not available.

The second regression diagnostic was developed from an approximate studentized residual. The residual errors from the LOESS model were divided by standard error (beta) to make their variances approximately constant. These approximate studentized residuals should be approximately normally distributed with a mean of zero and a variance of $\sigma^2 = 1$. To test this assumption, normal probability plots of the residuals were created for each smoothing parameter, combining all the studentized residuals across genders, regions, poverty status, and ages. The results for the children data indicate little distinction or affect by the selection of a particular smoothing parameter (e.g., see Figure 1), although linearity in the plotted curve is best expressed with smoothing parameters generally between 0.6 and 0.8. When considering the adult data sets, the appropriate value would generally be 0.9.

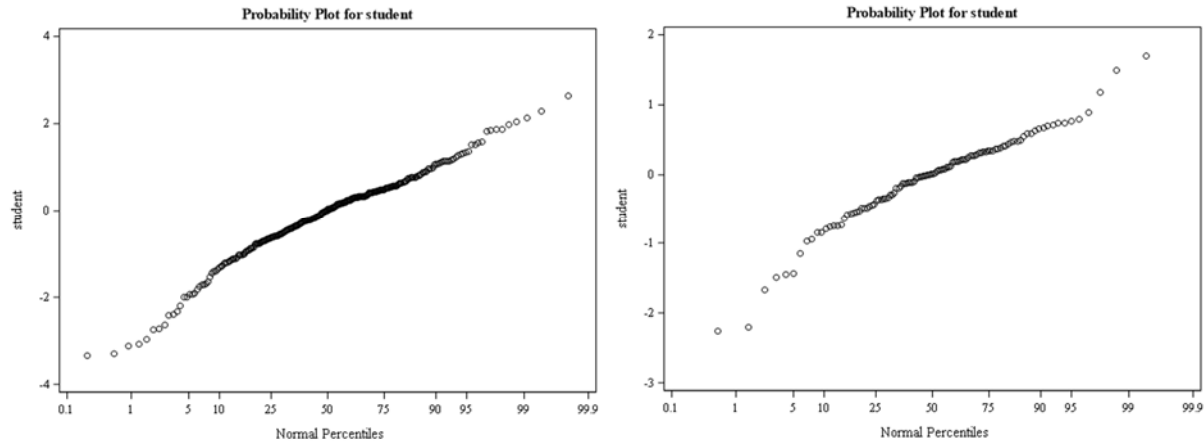


Figure 1. Normal probability plot of studentized residuals generated using logistic model, ‘STILL’ prevalence data, with smoothing set to 0.7 and 0.9 for children (left) and adults (right), respectively.

The third regression diagnostic are plots of the studentized residuals against the smoothed beta values. All the studentized residuals for a given smoothing parameter are plotted together within the same graph. Also plotted is a LOESS smoothed curve fit to the same set of points, with SAS’s optimal smoothing parameter choice, to indicate the typical pattern. Ideally there should be no obvious pattern and an average studentized residual close to zero with no regression slope (e.g., see Figure 2). For the children data sets, these plots generally indicate no unusual patterns, and the results for smoothing parameters 0.4 through 0.6 indicate a fit LOESS curve closest to the studentized residual equals zero line. When considering the adult data sets, 0.8 to 0.9 appears to be appropriate values.

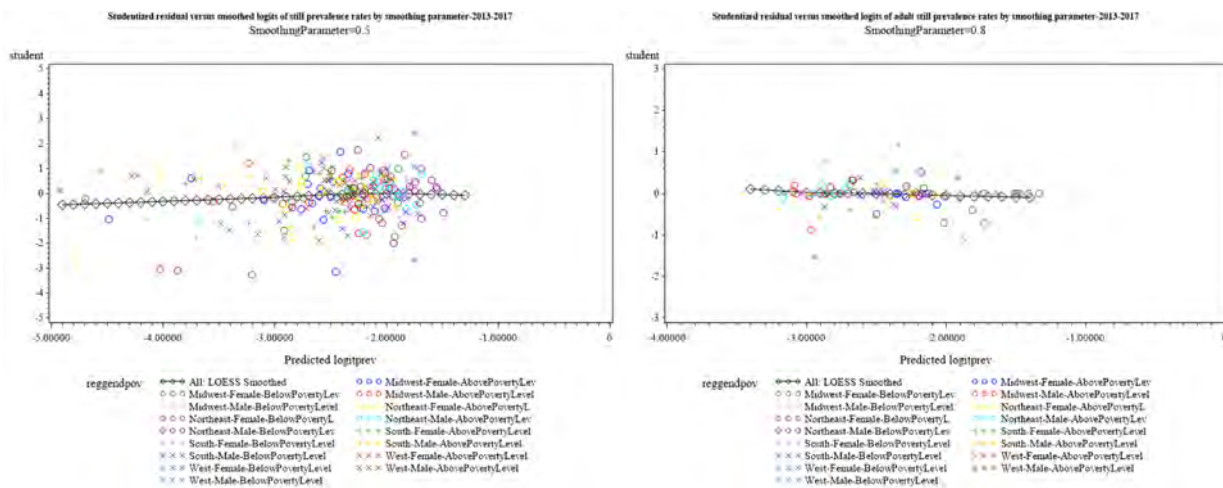


Figure 2. Studentized residuals versus model predicted betas generated using a logistic model and the ‘STILL’ prevalence data, smoothing set to 0.5 and 0.8 for children (left) and adults (right), respectively.

When considering both children asthma prevalence responses evaluated, the residual standard error (estimated values for sigma) suggests the choice of smoothing parameter as varied, ranging from 0.7 to 0.8. The normal probability plots of the studentized residuals suggest preference for smoothing at or above 0.6. The plots of residuals against smoothed predictions suggest the choices of 0.4 through 0.6. We therefore chose the final value of 0.6 to use for smoothing the children's asthma prevalence. For the adults, there were small differences in the statistical metrics used to evaluate the smoothing. A value of 0.9 was selected for smoothing based on the above findings and to remain consistent with what was used in the prior analysis (U.S. EPA, 2014; 2018).

The smoothed asthma prevalence and associated graphical presentation are provided in Supplemental Figures S-5 through S-8. A similar format to that presented using the non-smoothed asthma prevalence was followed, and again, only providing the results for children and adults that reported 'STILL' having asthma.

Step 2: U.S. Census Tract Poverty Ratio Data Set Description and Processing

This section briefly describes the approach used to generate census tract level poverty ratios for all U.S. census tracts, stratified by age and age groups where available. The following steps were performed using data from the 2017 U.S. Census 5-year American Community Survey (ACS)¹⁰ and modified SAS data processing files.¹¹

First, ACS internal point latitudes and longitudes were obtained from the 2017 Gazetteer files.¹² Next, the individual state level ACS sequence files (SF-56) were downloaded,¹³ retaining the number of persons across the variable "B17024" for each state considering the appropriate logical record number.¹⁴ The data provided by the B17024 variable is stratified by age or age groups (ages <5, 5, 6-11, 12-14, 15, 16-17, 18-24, 25-34, 35-44, 45-54, 55-64, 65-74, and ≥75)

¹⁰ <https://www.census.gov/newsroom/press-kits/2018/acs-5year.html>.

¹¹ ACS file processing code was adapted from ACS 2012 SAS programs and from ACS 2012 SAS Macros available at http://www2.census.gov/acs2012_5yr/summaryfile/UserTools/SF20125YR_SAS.zip and http://www2.census.gov/acs2012_5yr/summaryfile/UserTools/SF_All_Macro.sas. These were the same processing files used for updating the 2011-2015 asthma prevalence data set (US EPA, 2018).

¹² Data available at: <https://www.census.gov/geographies/reference-files/time-series/geo/gazetteer-files>.

¹³ We used the summary tables (B17024), giving census tract populations by poverty income ratio and age group downloaded from https://www2.census.gov/programs-surveys/acs/summary_file/2017/data/5_year_by_state/. Each state's ACS2017 5-yr table compressed file was unzipped with the sequence file 56 (SF-56; *e20175[state abbreviation]0056000.txt*) and appropriate geography file (*g20175[state abbreviation].txt*) retained.

¹⁴ Variable names (2017 Code List.pdf) are available at <https://www.census.gov/programs-surveys/acs/technical-documentation/summary-file-documentation.html>, along with the file for the appropriate logical record number (*ACS_2017_SF_5YR_Appendices.xls*).

and income/poverty ratios, given in increments of 0.25. We calculated two new variables for each age using the number of persons from the B17024 stratifications; the fraction of those persons having poverty ratios < 1.5 and ≥ 1.5 by summing the appropriate B17024 variable and dividing by the total number of persons in that age/age group. Then, the individual state level geographic data (g20175[xx].txt files) were screened for tract level information using the “sumlev” variable equal to ‘140’. Also identified was the US Region for each state, consistent with that used for the NHIS asthma prevalence data.¹⁵

Finally, the poverty ratio data were combined with the above described census tract level geographic data using the “stusab” and “logrecno” variables. Because APEX requires the input data files to be entirely complete (no missing values), additional processing of the poverty probability file was needed. For where there was missing tract level poverty information,¹⁶ we substituted an age-specific value using the average for the particular county the tract was located within, or the state-wide average. The percent of tracts substituted using county averaged values varied by age group though, on average, was approximately 1.6% of the total tracts (Table 4). Few tracts in six of the age groups were substituted using state averaged values (in total only 9 tracts had a substitution using state values for one of the age groups). The final output was a single file containing relevant tract level poverty probabilities (pov_acs2017_5yr.sas7bdat) by age groups for all U.S. census tracts.

Table 4. Percent of tracts substituted with county average or state average poverty status.

Percent Substituted	Age Groups (years)										
	≤5	6-11	12-17	18-24	25-34	35- 44	45-54	55-64	65-74	≥75	all
Filled using County Average	1.9%	2.0%	1.9%	1.5%	1.4%	1.4%	1.3%	1.3%	1.6%	1.9%	1.6%
Filled using State Average	<0.1%	<0.1%	<0.1%	none	<0.1%	none	none	none	none	none	<0.1%

Step 3: Combining Census Tract Poverty Ratios with the NHIS Regional Asthma Prevalence Data

The two data sets were merged considering the region identifier and stratified by age and sex. The Census tract-level asthma prevalence data set was calculated using the following weighting scheme:

¹⁵ <https://www2.census.gov/geo/pdfs/maps-data/maps/reference/> (using file *us_regdiv.pdf*)

¹⁶ Whether there were no data collected by the Census for poverty status or there were no people in an age group is relatively inconsequential to estimating the exposed people with asthma, particularly considering latter case as no people in that age group would be modeled by APEX when using the same Census population data set.

$Asthma\ prevalence = round((pov_prob * prev_belowpov) + ((1 - pov_prob) * prev_abovepov), 0.0001);$

whereas each U.S. census tract contains a tract-specific poverty-weighted asthma prevalence, stratified by ages (children 0-17), age groups (adults), and two sexes.

To evaluate the overall accuracy of the Census tract-level estimated asthma prevalence, we first compared these values with the NHIS national summary data for asthma prevalence reported for 2013 to 2017.¹⁷ According to the CDC, the NHIS are the principal source of national asthma prevalence data for the US. Note also, the NHIS 2013-2017 raw data was used to estimate the asthma prevalence for four U.S. regions in step 1 above. The NHIS national summary data are stratified by two age groups (children and adults) and for the two sexes (male and female) and were simply averaged across the five years of data available for the comparison. The Census tract-level estimated asthma prevalence were population-weighted using 2010 U.S. Census tract population data and aggregated to generate a similar national summary metric (and also considered data from 2013-2017 in their initial development). Table 5 show reasonable agreement between the two data sets: where present, the differences between the two data sets were generally small (≤ 0.1 percentage points) with the greatest percentage point difference found for adult females (~ 0.4 percentage points). The adult asthma prevalence estimated for both sexes using the Census tract-level was lower than the NHIS reported value, while the children's asthma prevalence data were generally similar between the two data sets. Overall, this degree of agreement was expected given that the 2013-2017 NHIS regional asthma prevalence (stratified by age, sex, and family income) served as the source for extrapolating asthma prevalence to the census tract level.

Step 4: Adjusting NHIS Regionally-derived Prevalence Data to Reflect State-level Asthma Prevalence

We then compared the NHIS Regionally-derived census tract-level estimated asthma prevalence to the Behavioral Risk Factor Surveillance System (BRFSS),¹⁸ an independent source providing state (and national) data about U.S. residents regarding their chronic health conditions such as asthma (among other health issues). For this comparison, the BRFSS asthma prevalence data were available for 2013-2016 and averaged across those four years to obtain a national summary metric. This BRFSS metric is similar to that calculated using the Census tract-level and

¹⁷ Downloaded was Table 4-1, the 2013-2017 NHIS current asthma prevalence percents by age groups and sex available at https://www.cdc.gov/asthma/nhis/default.htm#anchor_1524067853614. Accessed 5/7/19.

¹⁸ Downloaded was table C2.1 (for each adults and children), the 2013-2016 BRFSS current asthma prevalence percents by state and sex available at <https://www.cdc.gov/asthma/brfss/default.htm>. Table C1 was also downloaded to obtain the asthma prevalence for the two age groups not stratified by sex. Accessed 5/3/19.

NHIS asthma prevalence data sets and is provided in Table 5. The asthma prevalence data reported from BRFSS are consistently greater than that calculated using the Census tract-level data, particularly when considering adults. Overall, the BRFSS adult asthma prevalence is 1.6 percentage points greater than that estimated using the Census tract-level estimated prevalence, with the greatest difference observed for the two data sets of 2.8 percentage points observed for adult females. Asthma prevalence for the two data sets were closer when considering children, though the Census-tract level estimated data were still consistently lower than the BRFSS reported values (~0.2 to 0.4 percentage points).

Table 5. Asthma prevalence stratified by two age groups and sex using Census tract-level estimates, NHIS and BRFSS reported data.

Data Set (years of data)	All Ages, Both Sexes	Children (<18 years old)			Adults (≥ 18 years old)		
		all	female	male	all	female	male
NHIS (2013-2017)	7.8%	8.4%	7.2%	9.6%	7.6%	9.6%	5.5%
Census tract-level estimate	7.6%	8.5%	7.2%	9.7%	7.3%	9.2%	5.3%
BRFSS (2013-2016) ^A	n/a	8.8%	7.4%	10.1%	8.9%	11.4%	6.3%

^A The BRFSS does not have any data for some states, and where represented, not all four years of data were available for those state. n/a is not available.

It is unlikely that additional data are available for meaningful comparison, certainly not to the extent to which the NHIS Regionally-derived Census tract-level asthma prevalence is stratified and also not without inconsistencies in methodology used in their collection and reporting, if these data do exist at a local level (e.g., county health department data across all US counties). However, we were concerned with the potential for underestimating asthma prevalence that is indicated by the comparison of the NHIS Regionally-derived census tract-level asthma prevalence with the BRFSS data. Note, we used the NHIS 2013-2017 raw data set in Step 1 to serve as the basis for the census tract-level estimated asthma prevalence given its large sample size for both children and adults and because of the stratification of important influential variables (i.e., age, sex, family income). Contrary to this, the NHIS data are aggregated to four US regions and could account for less spatial variability than that provided by the individual state-level data obtained from BRFSS. With that in mind, we chose to adjust the NHIS-Census tract-level data (upwards or downwards) based on the percent difference observed between a population weighted state level aggregate of the census tract level data and the BRFSS state-level asthma prevalence (Table 6) and was calculated as follows:

$$\text{State Adjustment Factor} = (\text{NHIS_Census}_{\text{regional prevalence}} - \text{BRFSS}_{\text{state prevalence}}) / \text{BRFSS}_{\text{state prevalence}}$$

Table 6. Factors used to adjust NHIS Regionally-derived census tract-level asthma prevalence and based on BRFSS state level data.

State	Adjustment Factor – Children ^A		Adjustment Factor – Adults ^A	
	male	female	male	female
Alabama	0.510	0.413	0.356	0.399
Alaska	0	0	0.076	0.296
Arizona ^B	0.157	0.058	0.199	0.237
Arkansas	0	0	0.343	0.299
California	0.099	0.199	-0.023	0.108
Colorado	0	0	0.165	0.179
Conneticut	0.114	0.153	0.220	0.365
Delaware	0	0	0.286	0.476
Florida	-0.124	-0.11	0.155	0.136
Georgia	0.234	0.015	0.183	0.320
Hawaii	0.59	1.002	0.277	0.355
Idaho	0	0	0.182	0.171
Illinois	-0.016	-0.151	0.044	0.134
Indiana	-0.107	0.030	0.239	0.388
Iowa	0	0	0.044	0.049
Kansas	0.140	0.035	0.111	0.176
Kentucky	0.076	-0.016	0.701	0.628
Lousiana	-0.051	-0.174	0.250	0.130
Maine	-0.021	-0.104	0.494	0.478
Maryland	0.200	0.218	0.399	0.399
Massachusetts	0.257	0.061	0.328	0.479
Michigan	0.169	0.036	0.414	0.38
Minnesota	-0.228	-0.059	-0.014	0.069
Mississippi	0.127	-0.026	0.151	0.120
Missouri	0.003	0.226	0.264	0.301
Montana	-0.137	0.107	0.154	0.173
Nebraska	-0.180	-0.210	0.030	0
Nevada	-0.143	0.068	-0.070	0.129
New Hampshire	-0.031	0.009	0.276	0.502
New Jersey	-0.094	0.009	0.052	0.078
New Mexico	0.141	0.208	0.340	0.302
New York	-0.040	-0.024	0.285	0.237
North Carolina	0.171	0.416	0.154	0.225
North Dakota	0	0	0.254	0.132
Ohio	0.024	0.016	0.233	0.332
Oklahoma	0.298	0.065	0.549	0.365
Oregon	-0.047	0.237	0.400	0.443
Pennsylvania	0.137	0.003	0.172	0.357

State	Adjustment Factor – Children ^A		Adjustment Factor – Adults ^A	
	male	female	male	female
Puerto Rico ^C	0	0	0	0
Rhode Island	0.083	0.136	0.376	0.447
South Carolina	0	0	0.240	0.252
South Dakota	0	0	0.040	-0.043
Tennessee	0.116	-0.111	0.256	0.368
Texas	-0.034	-0.210	0.068	0.111
Utah	-0.079	-0.032	0.239	0.160
Vermont	-0.114	0.131	0.333	0.453
Virginia	0	0	0.218	0.333
Wash DC	0.389	0.436	0.656	0.577
Washington	-0.108	0.091	0.246	0.294
West Virginia	0.041	-0.032	0.561	0.581
Wisconsin	-0.097	0.232	0.279	0.284
Wyoming	0	0	0.146	0.190

^A Values of zero indicate there were no BRFSS data were available, therefore no adjustment was made.
^B Data reported for Arizona children in the 2013 BRFSS were atypical: prevalence for females were greater than that of male, having rates almost opposite that expected. These data were not used to calculate the adjustment factor.
^C The NHIS-Census regional data was not used for estimating asthma prevalence for Puerto Rico, therefore only BRFSS data for the two age groups and sexes were used.

The adjustment factor was applied to the census tract estimated asthma prevalence considering the state level information as follows:

$$Prevalence_{Adjusted} = NHIS/Census_{prevalence} + (Adjustmen\ Factor \times NHIS/Census_{prevalence})$$

By design, the adjustment has better aligned the estimated NHIS Regionally-derived census tract-level asthma prevalence with the BRFSS reported values at the state and national level (Table 7). These BRFSS-adjusted census tract-level asthma prevalence data are used for the APEX simulations and are found within the *asthma_prev_1317_tract_051319_adjusted.txt* file. For brevity, data are shown only for a few states most relevant to the study areas of interest in the current O₃ exposure and risk analysis.

Table 7. Population-weighted state level asthma prevalence stratified by two age groups and sex: Original census tract-level estimates based on 2013-2017 NHIS regional prevalence and US Census family income data, 2013-2016 BRFSS reported prevalence, and BRFSS-adjusted census tract-level estimates used for the APEX asthma prevalence file.

State	Related Study Area ^A	Sex	Child Asthma Prevalence			Adult Asthma Prevalence		
			Census tract-level estimate	BRFSS state reported data	Adjusted APEX prevalence file	Census tract-level estimate	BRFSS state reported data	Adjusted APEX prevalence file
Georgia	Atlanta	female	7.9%	8.1%	8.0%	8.7%	11.4%	11.4%
		male	10.0%	12.4%	12.3%	4.7%	5.6%	5.5%
Massachusetts ²	Boston	female	7.7%	8.2%	8.1%	9.5%	14.0%	14.0%
		male	10.9%	13.7%	13.6%	5.8%	7.7%	7.6%
Texas ²	Dallas	female	7.9%	6.2%	6.3%	8.6%	9.5%	9.5%
		male	10.0%	9.6%	9.6%	4.7%	5.0%	5.0%
Michigan	Detroit	female	7.1%	7.4%	7.4%	9.7%	13.4%	13.4%
		male	9.9%	11.6%	11.6%	5.8%	8.2%	8.2%
Pennsylvania	Philadelphia	female	8.0%	8.0%	8.0%	9.6%	13.0%	13.0%
		male	11.0%	12.6%	12.5%	5.8%	6.8%	6.8%
Arizona ^B	Phoenix	female	5.9%	6.2%	6.2%	9.5%	11.7%	11.7%
		male	8.6%	9.9%	9.9%	5.6%	6.8%	6.7%
California	Sacramento	female	5.9%	7.1%	7.0%	9.4%	10.4%	10.4%
		male	8.6%	9.4%	9.4%	5.6%	5.5%	5.5%
Missouri	St. Louis	female	7.0%	8.5%	8.5%	9.7%	12.6%	12.6%
		male	9.7%	9.7%	9.7%	5.8%	7.3%	7.3%
All US States		female	7.2%	7.4%	7.4%	9.2%	11.4%	11.4%
		male	9.7%	10.1%	10.1%	5.3%	6.3%	6.3%
		both	8.5%	8.8%	8.8%	7.3%	8.9%	8.9%

^A Each study area is defined by a Consolidated Statistical Area (CSA) may involve counties from more than one US state. This information is added for relevance to the spatial scale and not meant to be absolute in defining the prevalence for any of the study areas.

^B Data for children were only available for the following years in a few states: 2016 (Arizona), 2015 and 2016 (Massachusetts), 2013-2015 (Texas). Adults based on 2013-2016.

The asthma prevalence estimates vary for the different ages and sexes of children and adults that reside in each census tract of each study area. We evaluated the spatial distribution of the asthma prevalence using the specific census tracts that comprise the consolidated statistical area (CSA) that generally define each study area. We first separated data for children from those for adults and calculated simple descriptive statistics of asthma prevalence for the tracts, stratified by sex (Table 8). Consistent with broadly defined national asthma prevalence (e.g., Table 3-1 of the draft PA), on average, children have higher estimated rates than adults, male children have higher rates than female children, and adult females have higher rates than adult males.

By using age, sex, and family income variables to develop the tract level prevalence, we also observe that there is spatial variability in the estimated prevalence both within and across the CSAs. Atlanta, Boston, Detroit, and Philadelphia have some of the highest asthma prevalence for male children considering most of the statistics with rates as high as 25.5% in one or more census tracts for males of a given year of age. The Dallas study area exhibits some of the lowest asthma prevalence when considering adults (both sexes) with rates as low as 3.8% in one or more tracts for males within a given age group. These summary statistics represent the range of age- and sex-specific values for the census blocks used in each APEX simulation to estimate the number of individuals that have asthma.

Table 8. Descriptive statistics for non-population weighted asthma prevalence for children (ages 5-17) and adults (age >17) using all census tracts from 8 consolidated statistical areas (CSAs) in the APEX asthma prevalence file (2013-2017).

CSA Name - ID# (# tracts) and Population group		Sex	Asthma Prevalence across all ages (or age groups) and census tracts ^A						
			Mean	Standard Deviation	Minimum	Median	95 th percentile	99 th percentile	Maximum
Atlanta-122 (1,077)	adult	female	11.1%	1.8%	7.7%	11.1%	14.0%	15.9%	20.9%
		male	5.5%	0.8%	4.3%	5.4%	7.1%	7.5%	7.9%
	child	female	9.7%	1.7%	6.5%	9.6%	12.9%	13.9%	15.0%
		male	14.1%	1.7%	10.6%	14.0%	16.8%	17.6%	18.3%
Boston-148 (1,753)	adult	female	13.8%	1.8%	10.5%	13.5%	17.3%	20.5%	28.9%
		male	7.6%	0.9%	5.4%	7.5%	9.1%	10.0%	12.9%
	child	female	9.4%	2.0%	5.6%	9.5%	12.4%	13.5%	17.1%
		male	15.4%	2.5%	8.7%	15.1%	19.5%	20.8%	23.4%
Dallas-206 (1,422)	adult	female	9.3%	1.5%	6.5%	9.3%	11.8%	13.5%	16.5%
		male	4.9%	0.7%	3.8%	4.9%	6.4%	6.8%	9.7%
	child	female	7.6%	1.3%	5.0%	7.4%	10.0%	10.9%	13.5%
		male	11.0%	1.4%	8.3%	11.0%	13.2%	13.8%	18.1%
Detroit-220 (1,583)	adult	female	13.3%	2.5%	7.8%	13.4%	17.8%	20.6%	25.6%
		male	7.9%	2.2%	1.0%	7.6%	12.4%	14.7%	19.0%
	child	female	8.6%	1.5%	6.4%	8.2%	11.6%	12.5%	13.2%
		male	13.3%	3.0%	7.7%	12.7%	19.9%	23.6%	25.5%
Philadelphia-428 (1,725)	adult	female	12.1%	2.3%	8.2%	12.0%	16.4%	19.8%	26.5%
		male	6.5%	0.9%	4.6%	6.4%	8.1%	9.0%	11.4%
	child	female	9.1%	1.9%	5.6%	9.2%	12.0%	13.1%	15.3%
		male	13.6%	2.4%	8.2%	13.3%	17.8%	19.2%	21.1%
Phoenix-429 (988)	adult	female	11.6%	1.6%	8.6%	11.7%	14.4%	16.0%	19.7%
		male	7.0%	1.5%	5.1%	7.1%	9.1%	11.7%	16.7%
	child	female	7.6%	1.5%	4.6%	8.0%	9.5%	9.6%	9.6%
		male	11.5%	1.8%	8.5%	11.6%	14.8%	15.9%	17.1%
Sacramento-472 (539)	adult	female	10.4%	1.4%	7.7%	10.5%	12.7%	14.0%	16.5%
		male	5.7%	1.1%	4.2%	5.9%	7.3%	9.0%	13.6%
	child	female	8.5%	1.7%	5.2%	9.0%	10.7%	10.9%	10.9%
		male	10.8%	1.7%	8.1%	10.9%	13.7%	14.8%	16.2%
St. Louis-476 (638)	adult	female	11.8%	2.1%	6.8%	11.9%	15.0%	17.4%	21.5%
		male	6.5%	1.8%	0.9%	6.5%	9.9%	11.8%	14.5%
	child	female	9.2%	2.0%	5.3%	9.1%	12.9%	14.2%	15.6%
		male	11.1%	2.4%	6.5%	10.7%	15.9%	19.3%	21.9%

^A As described in the text, prevalence is based on single year ages (children) or age group (adults) and sex derived from 2013-2017 CDC NHIS asthma prevalence and considering U.S. census tract level family income/poverty ratio data. Data presented are not population-weighted and represent the distribution of applied probabilities used by APEX for tracts having a non-zero population. Note also, upper and lower percentiles could represent prevalence for a single-year age/sex group residing in a single tract within a study area.

Evaluation of Additional Asthma Prevalence Questions and Responses

To estimate asthma prevalence, we used responses to the question of whether an NHIS study participant responded ‘Yes’ to the survey question of ‘STILL’ having asthma rather than using the responses to the question of ‘EVER’ having asthma (with the former being a subset of the latter group). According to the CDC, lifetime asthma is defined by responding ‘Yes’ to “Have you ever been told by a doctor {nurse or other health professional} that you have asthma?”, while current asthma is defined as responding ‘Yes’ to both the aforementioned and this subsequent question “Do you still have asthma?”.¹⁹ Because the exposure and risk analyses in this review reflect a generally current actualized hypothetical single-year scenario that is not covering the lifetime of the simulated individuals, the prevalence estimate based on those participants responding as currently (‘STILL’) having asthma was deemed most appropriate. We note that the response of survey participants who stated they do not still have asthma does not reflect a doctor’s/health professional’s diagnosis, thus it is possible there may be individuals in this group that might actually still have asthma and experience asthma-related health effects, potentially leading to an underestimate in the asthma prevalence used in our exposure and risk simulations. Because we used the responses to the “STILL” having asthma question to estimate prevalence in this assessment, we evaluated additional related questions in the NHIS data to estimate the magnitude of this potential underestimate in asthma prevalence.

There are two additional questions related to asthma prevalence that are asked of NHIS survey participants who responded ‘Yes’ to the ‘EVER’ having asthma question that could provide insight into the likelihood that people could ‘STILL’ have asthma but did not respond ‘Yes’ to that latter question. The first additional asthma question is, “DURING THE PAST 12 MONTHS, have you had an episode of asthma or an asthma attack?” (i.e., variable ‘CASHYR’ or ‘AASHYR’ for children and adults, respectively); the second is, “DURING THE PAST 12 MONTHS, have you had to visit an emergency room or urgent care center because of asthma?” (i.e., variable ‘CASERYR1’ or ‘AASERYR1’). We evaluated the responses to all four of these asthma questions using children’s 2017 data set as an example, the results of which are presented in Table 9.

Most survey participants responded either yes or no to the ‘EVER’ having asthma question; those not providing a response were removed from the analysis. There were few individuals not responding to the question (13 of 8,845), thus it was assumed there would be no bias to the overall conclusions following their removal. Of the remaining children surveyed, 13.2% (i.e., 1,168 of 8,832) had a doctor/health professional diagnose them as having asthma at some time in their life, with a majority of those ‘EVER’ having asthma (63.3%) responding

¹⁹ <https://www.cdc.gov/asthma/brfss/default.htm>.

‘Yes’ to ‘STILL’ having asthma. Based on these responses to the ‘STILL’ having asthma question, the overall asthma prevalence for children would be estimated as 8.4% (i.e., 739 of 8,832). As mentioned above, it is possible that prevalence is underestimated due to the nature of the diagnosis (i.e., self assessment) and at most, could be underestimated by a factor of 1.6 (i.e., 13.2/8.4) if assuming ‘EVER’ having asthma response was appropriate to use in this assessment. We suggest solely using this ‘EVER’ having asthma response would likely bias the prevalence high based on the below analysis of responses to the two additional asthma questions.

Table 9. Children’s responses to four questions regarding their asthma status, 2017 NHIS.

Diagnosed by a Doctor as EVER Having Asthma?	Participant Reported as STILL Having Asthma?	Participant Reported in Past 12 Months Did You Have:		Survey Participants (n)
		Asthma Attack?	Asthma-related ER Visit?	
Did not respond	-	-	-	13
No	-	-	-	7,664
Yes (n=1,168)	No (n=420)	No	No	396
		No	Yes	5
		Yes	No	15
		Yes	Yes	4
	I don't know (n=9)	No	No	5
		Yes	No	4
	Yes (n=739)	No	No	336
		No	Yes	22
		Yes	No	248
		Yes	Yes	131
		I don't know	No	1
	I don't know	I don't know	1	
Sum of EVER (Y/N), all ages				8,832

There were a few participants (6.5%, 28 of 429) who reported they did not or did not know they ‘STILL’ have asthma (note also, an unprofessional diagnosis), but also reported they had an asthma attack and/or had to be treated by a doctor because of asthma. Based on these data, asthma prevalence estimated using the response for the ‘STILL’ having asthma question alone might be underestimated by about 0.3 percentage points (i.e., 28/8832, the number reporting asthma attack or ER visit but also reporting “no” for still having asthma divided by total respondents), such that the overall asthma prevalence for children might be 8.7% rather than 8.4%. This would be with the assumption that the individual has accurately self-diagnosed an asthma attack, a perhaps reasonable assumption given they had been diagnosed with asthma at some time in their life. When considering the participants that stated they ‘STILL’ have asthma,

approximately 54% reported they had an asthma attack and/or had to be treated by a doctor because of asthma (i.e., 401 of 739). This clearly indicates that when survey participants reported they ‘STILL’ have asthma, they are more likely to have asthma attacks/ER visits than those who do not state they ‘STILL’ have asthma. An alternative hypothesis is also possible, in that they could have indicated they still have asthma as a result of the asthma attack/ER visit. Regardless, the health condition and the adverse response appear to be interrelated.

Additionally, we could assume that all participants that ‘EVER and ‘STILL’ have asthma (100% rather than the 54% estimated above) would have an asthma attack/ER visit at some time in their life (and perhaps not just within 12 months). Applying that information to survey participants who stated they did not ‘STILL’ have asthma and also report they have experienced an asthma attack/ER visit, implies that the asthma prevalence derived without these individuals (i.e., 0.3 percentage points) might be underestimated by a factor of about two. Thus, based on this analysis and including assumptions made using the responses to the additional questions, it is possible that asthma prevalence estimated using the ‘STILL’ variable alone (as was done for this assessment) could be underestimated by about 0.6 percentage points (i.e., an overall ‘current’ asthma prevalence for children would be about 9.0% rather than the 8.4% used in the simulations).

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SUPPLEMENTAL FIGURES S-1 to S-4, ASTHMA PREVALENCE NON-SMOOTHED

Figure S-1. Non-smoothed asthma prevalence for children that still have asthma. Above (left panels) and below poverty level (right panels) for Midwest (top panels) and Northeast (bottom panels) regions.

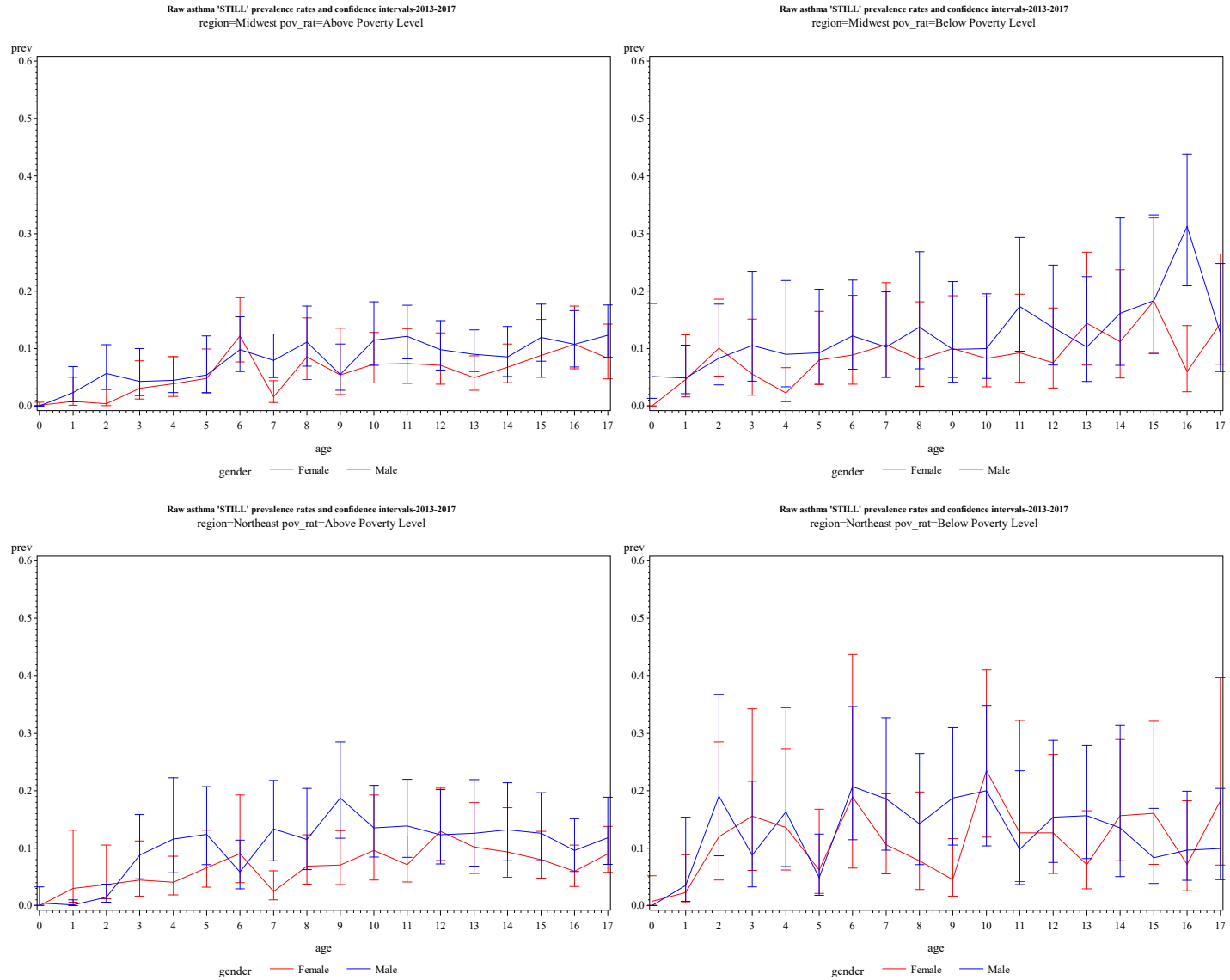


Figure S-2. Non-smoothed asthma prevalence for children that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.

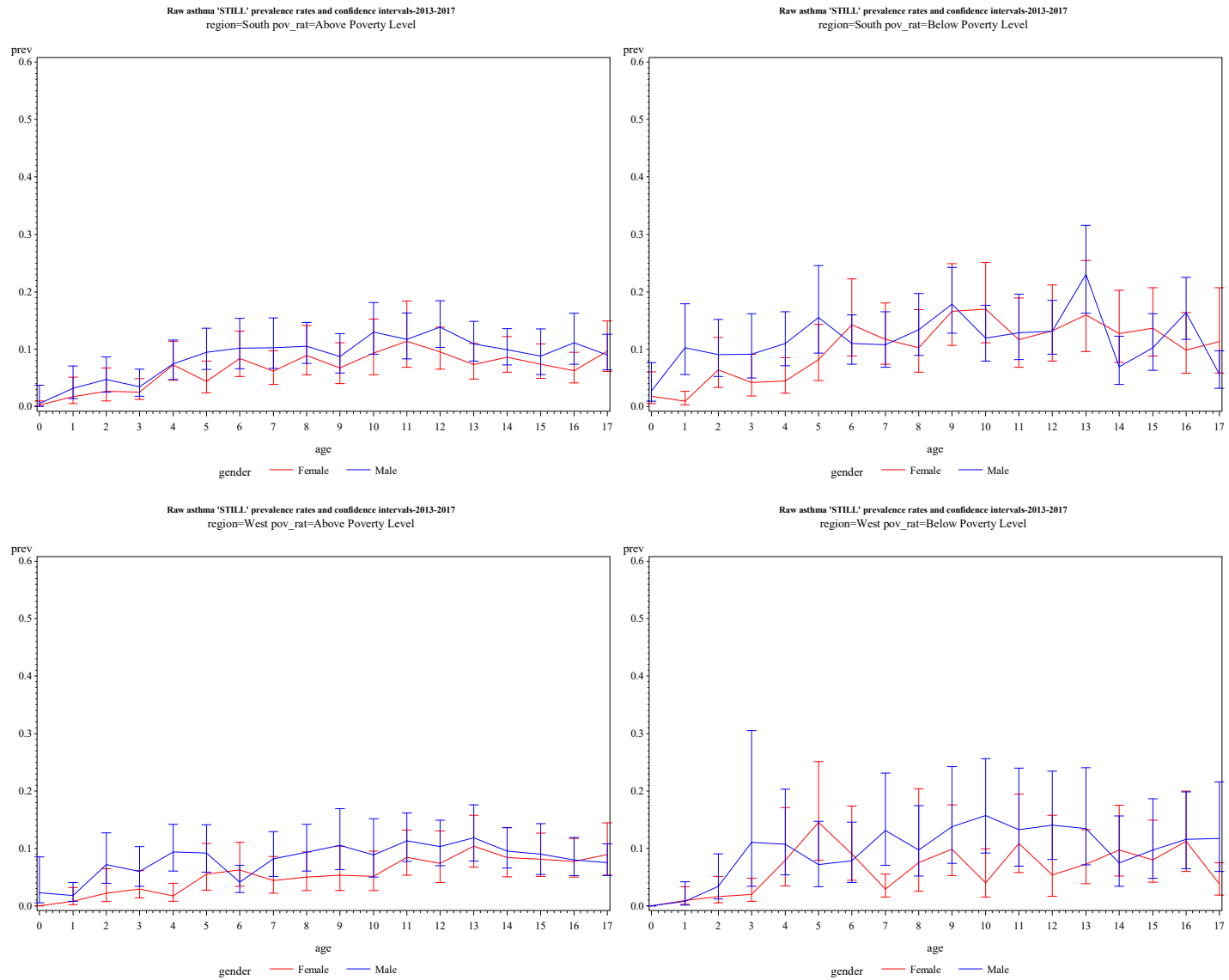


Figure S-3. Non-smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for Midwest (top panels) and Northeast (bottom panels) regions.

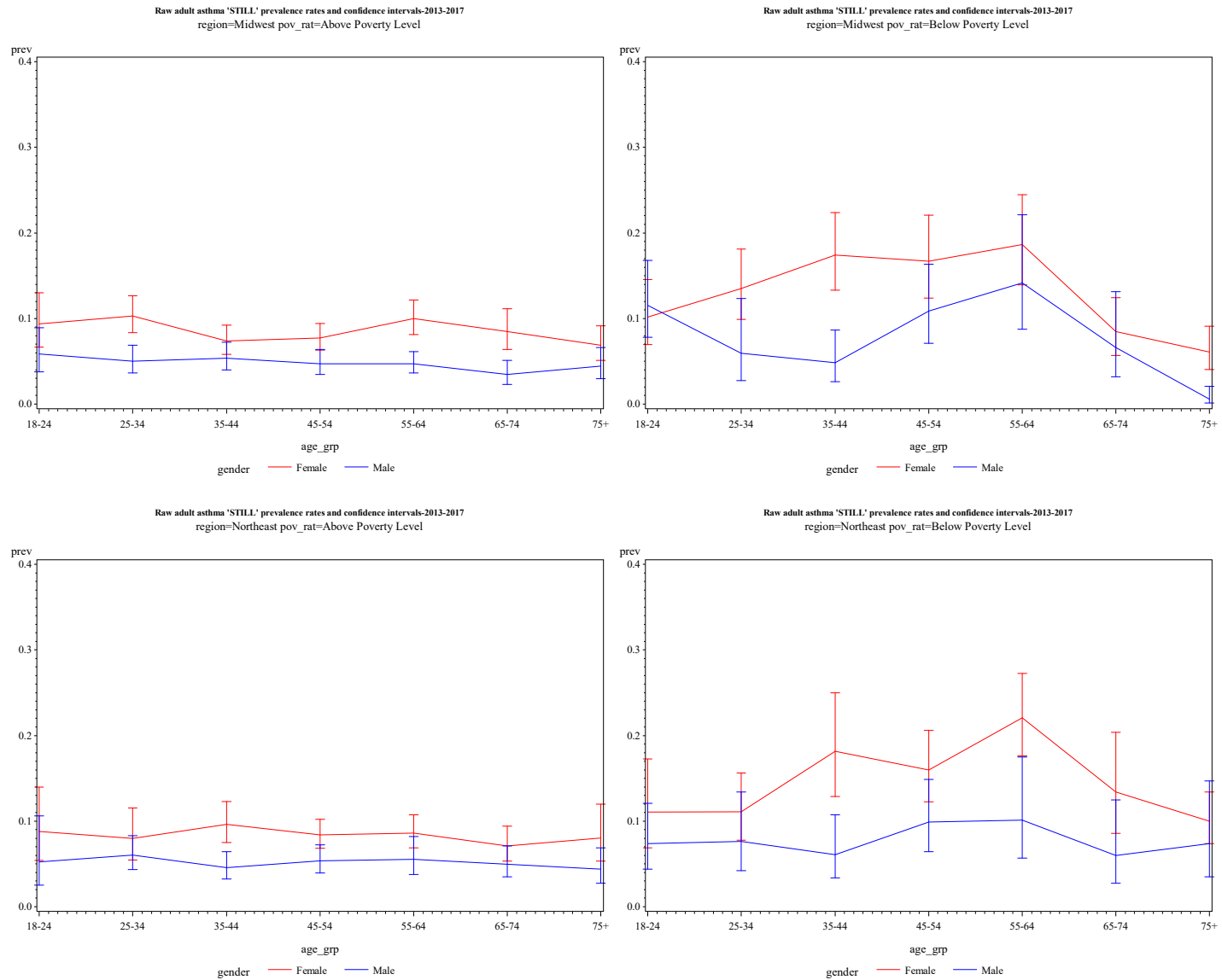
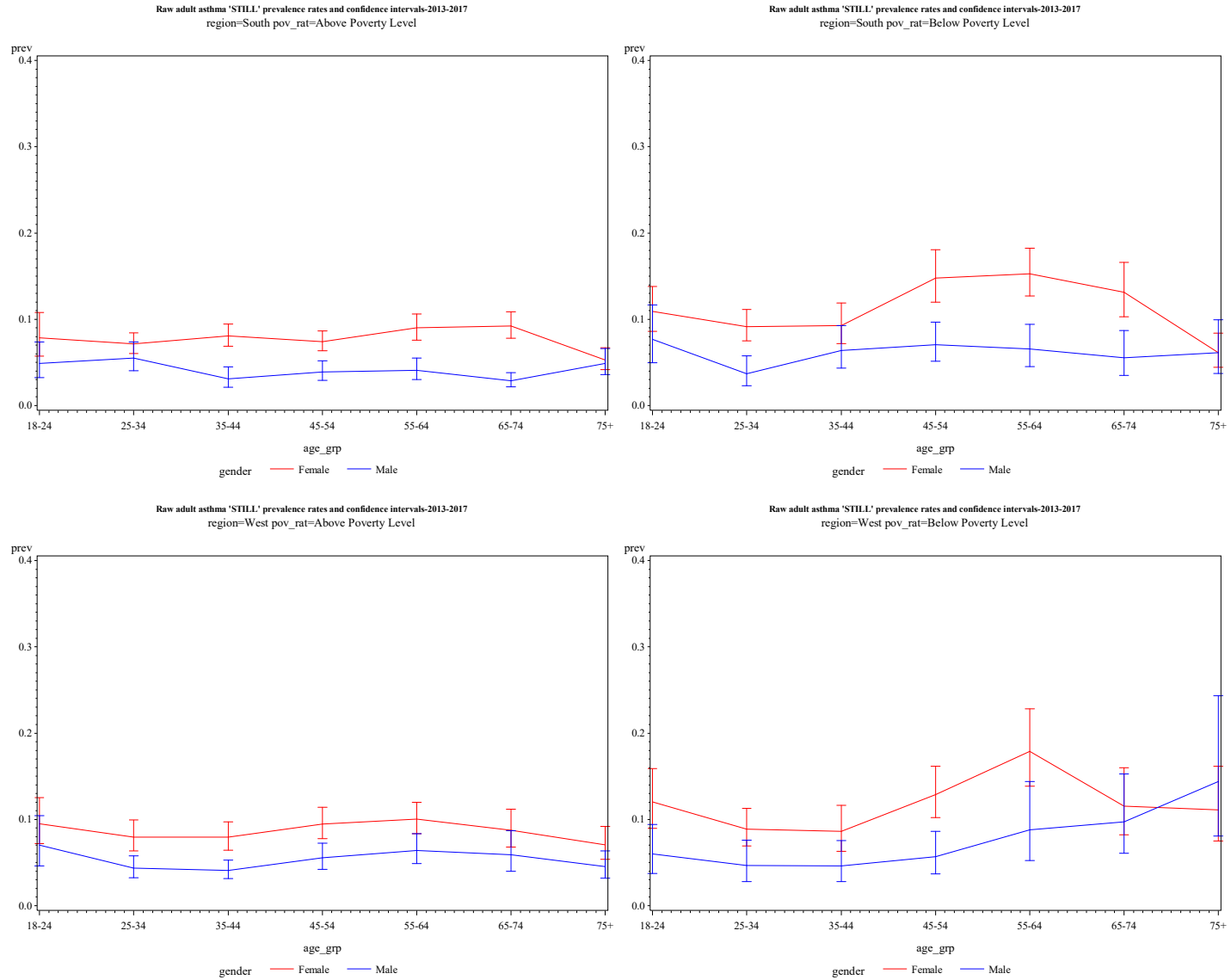


Figure S-4. Non-smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.



SUPPLEMENTAL FIGURES S-5 to S-8, ASTHMA PREVALENCE SMOOTHED

Figure S-5. Smoothed asthma prevalence for children that still have asthma. Above (left panels) and below poverty level (right panels) for Midwest (top panels) and Northeast (bottom panels) regions.

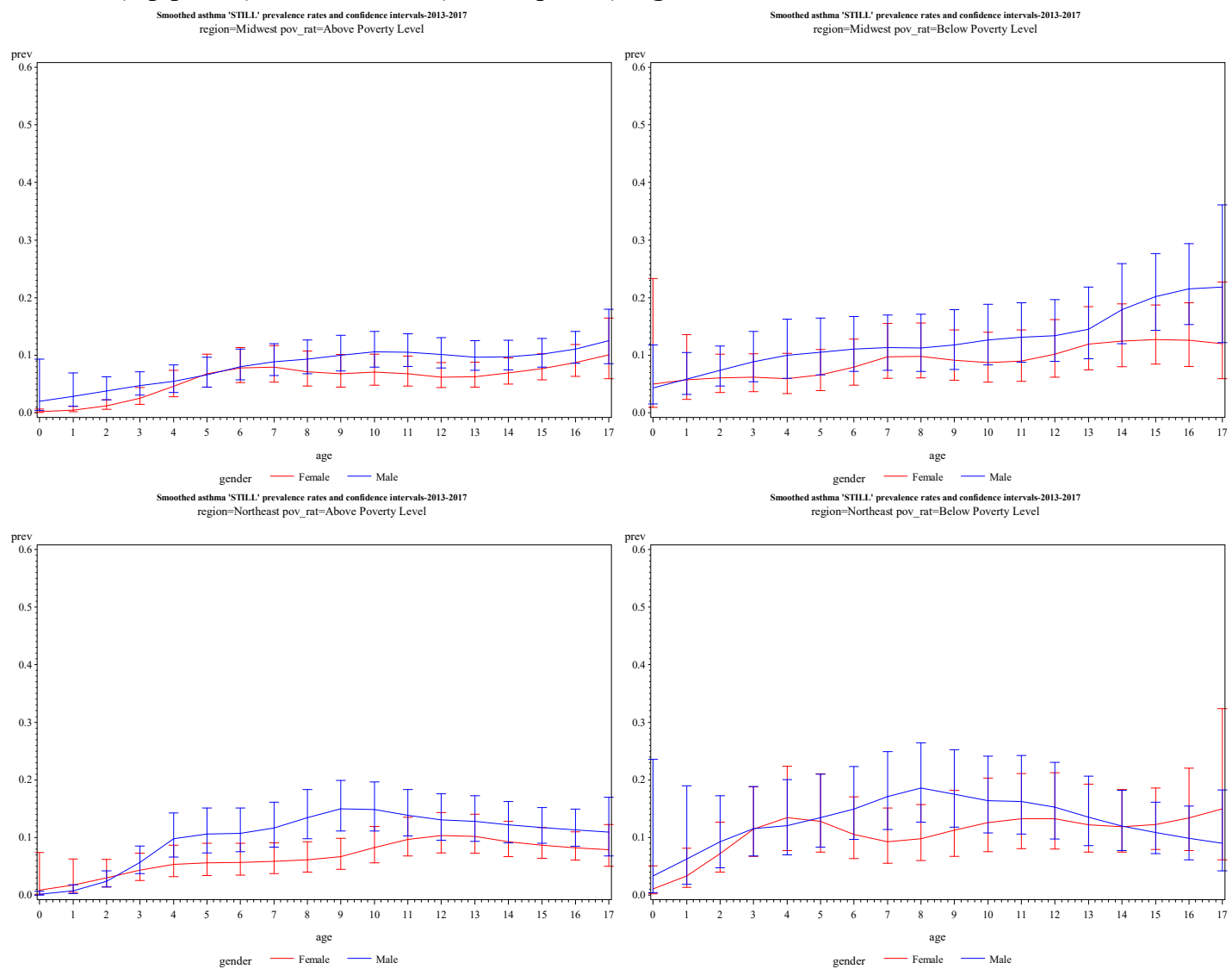


Figure S-6. Smoothed asthma prevalence for children that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.

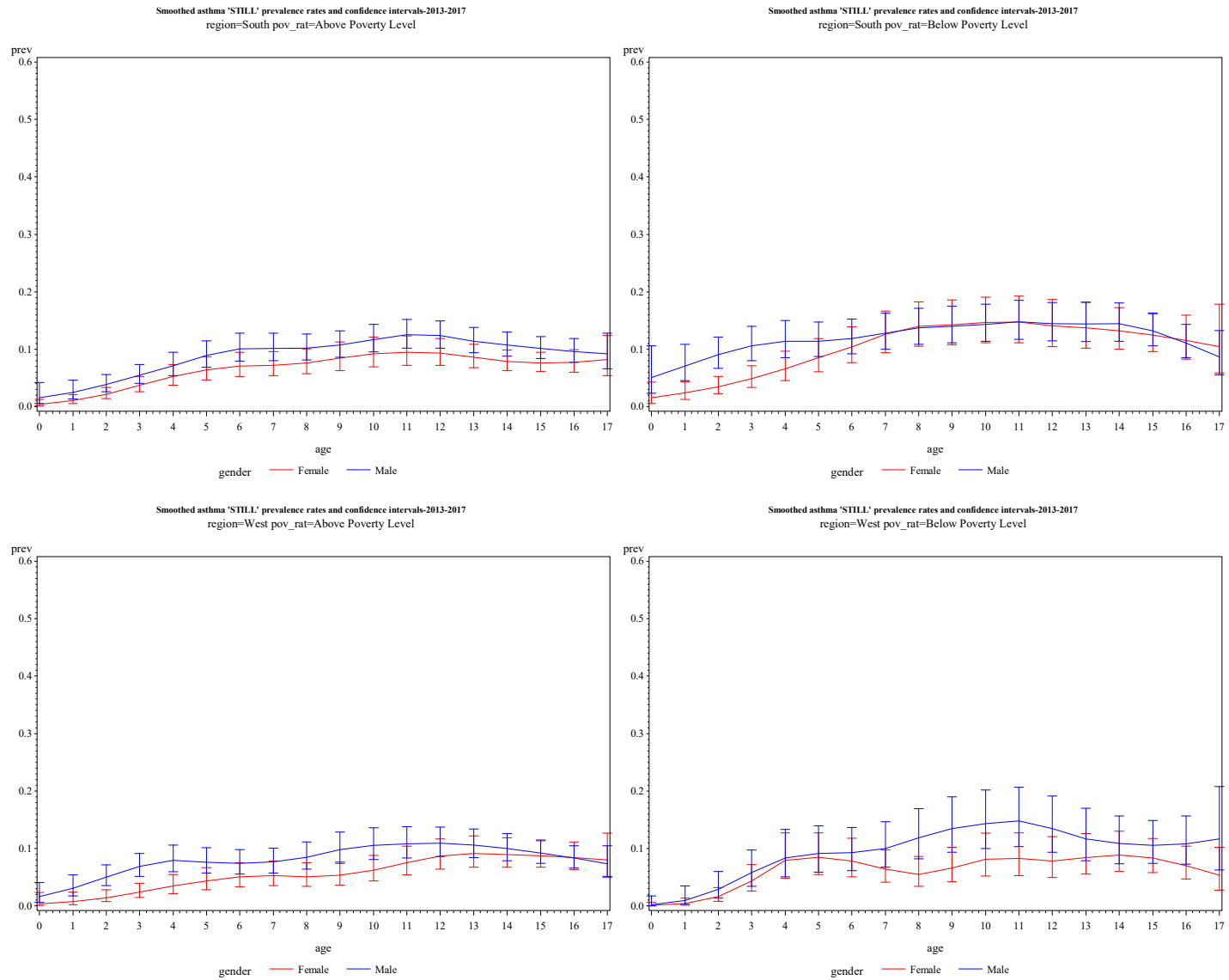


Figure S-7. Smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for Midwest (top panels) and Northeast (bottom panels) regions.

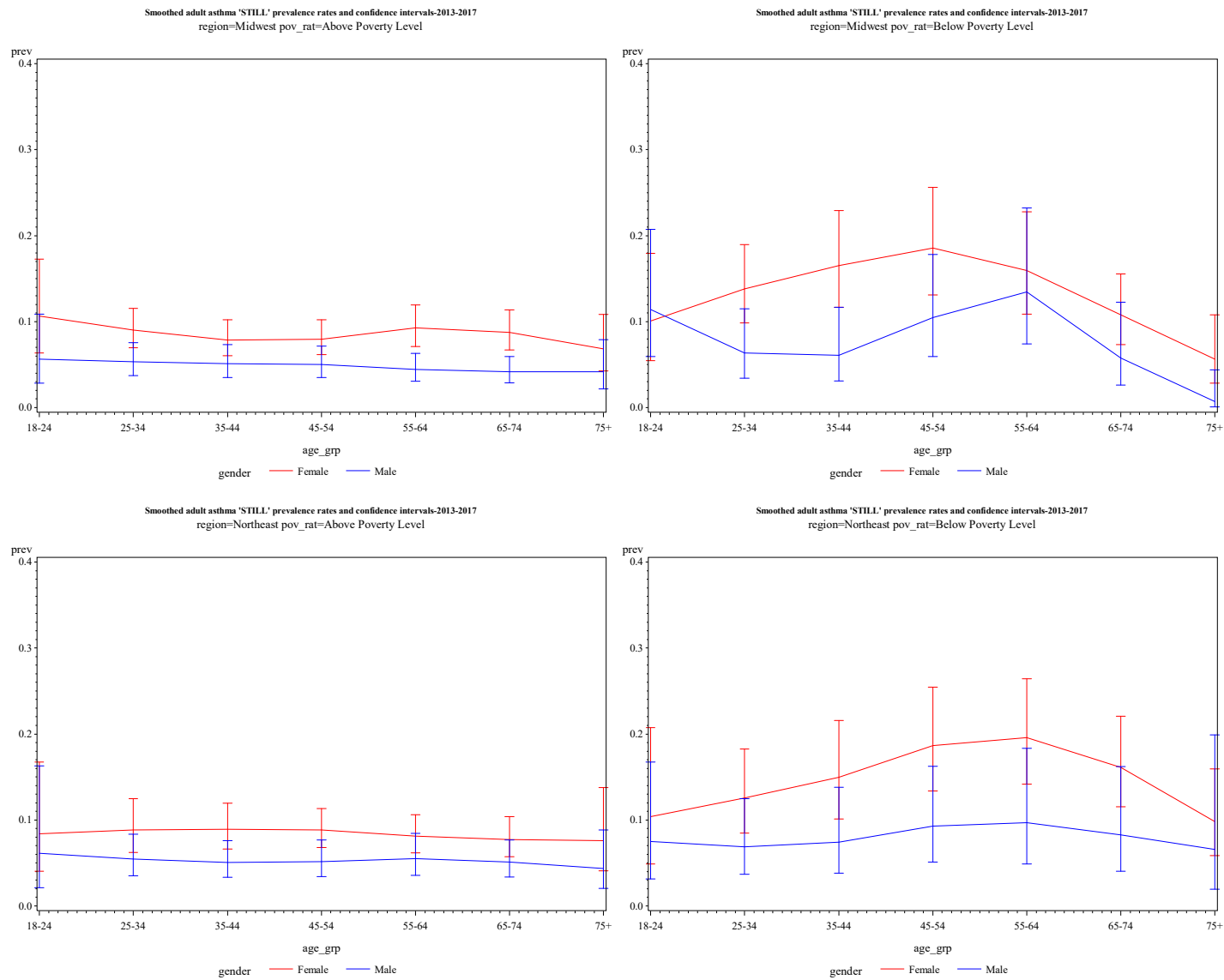
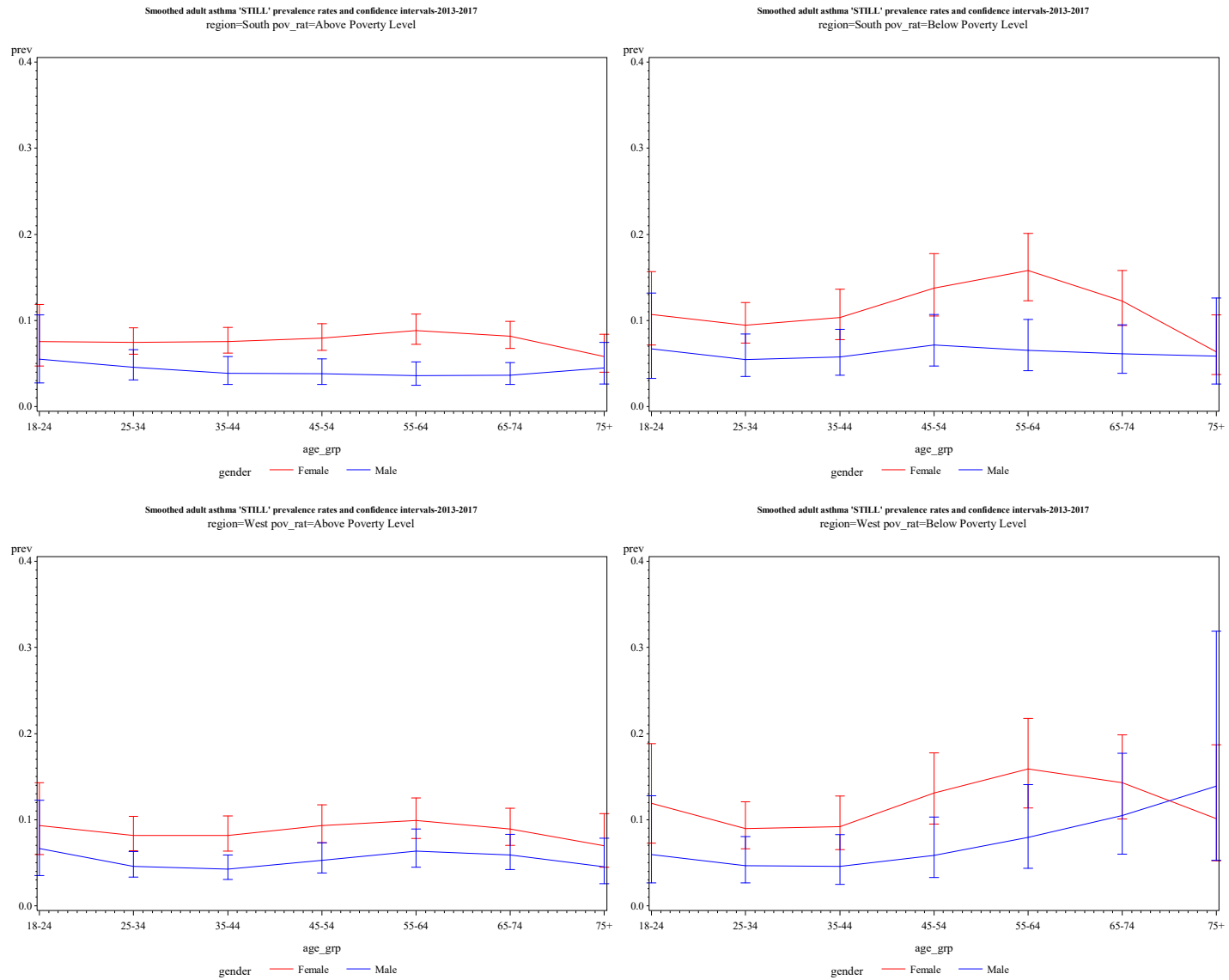


Figure S-8. Smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.



**APPENDIX 3D, ATTACHMENT 2:
ICF TECHNICAL MEMO: IDENTIFICATION OF SIMULATED INDIVIDUALS AT
MODERATE EXERTION**

MEMORANDUM

To: John Langstaff and Stephen Graham, EPA
From: Jeanne Luh, Graham Glen, and Chris Holder, ICF
Date: March 26, 2019
Re: Identification of Simulated Individuals at Moderate Exertion

1. Introduction

Under Work Assignment 4-55 of U.S. Environmental Protection Agency (EPA) Contract EP-W-12-010, the EPA Work Assignment Manager (WAM) asked ICF (hereafter “us”, “we”, etc.) to evaluate the approach used in the Air Pollutants Exposure Model (APEX; U.S. EPA, 2017a and 2017b) to identify when simulated individuals are at moderate exertion on average during any 8-hour exposure period. APEX uses the **ModEVR8** parameter, where EVR is equivalent ventilation rate, to define the threshold EVR for moderate exertion. EVR, calculated as ventilation rate divided by body surface area (V_e/BSA), values at or above **ModEVR8** (but below **HeavyEVR8**, the threshold for heavy exertion) are classified as moderate exertion. The **ModEVR8** value typically used in regulatory runs of APEX is 13 L/min-m², which was developed by Whitfield (1996) using clinical data from McDonnell et al. (1991). In McDonnell et al., study participants were required to maintain a V_e of 40 L/min while exposed to ozone and performing activities classified as moderate exertion over a 6.6-hour period. Using this data, Whitfield (1996) defined the EVR range to be 13–27 L/min-m² for 8-hour-average exposures at moderate exertion.

The approach used to define moderate exertion was noted in public comments in the last review of EPA’s Health Risk and Exposure Assessment for Ozone in 2014 (U.S. EPA, 2014). The bullets below summarize two critiques that some public commenters had about the **ModEVR8** value of 13 L/min-m².

- A **ModEVR8** value of 13 L/min-m² was too low and resulted in an overstatement of the number of exposures. This, in turn, resulted in an overestimation of the lung function decrement risk when exposure-response functions were used to estimate risk.
- The strenuous nature of the exercise performed in the clinical studies to achieve an EVR of 20 L/min-m² was not comparable to the activities and range of actual 8-hour EVRs in the populations of interest. They suggested that use of the clinical studies data may not be reasonable in defining **ModEVR8**.

Due to the lack of available controlled studies for human exposure to ozone, we focused on evaluating how **ModEVR8** is defined and we performed our analyses using an expanded dataset of clinical studies provided by the EPA WAM where the target EVR under moderate exertion was 20 L/min-m².

2. Data Sources

In Table 1 we list the clinical studies with data available on V_e and EVR for individuals undergoing moderate exertion during 6.6-hour exposure to filtered air and ozone. Adult study participants were required to maintain an EVR of 20 L/min-m² while undergoing intermittent moderate exercise, which consisted of six periods of 50-minute exercise on the treadmill or cycle ergometer, each followed by a 10-minute break, and with a 35-minute lunch after the third period.

Table 1. Clinical Studies with 6.6-hour Moderate Exertion

Reference	No. Subjects / Gender	Age Range (years)	O ₃ Exposure (ppm)
Folinsbee et al. (1998)	10 Males	18–33	0, 0.12
Horstman et al. (1990)	22 Males	18–35	FA, 0.08, 0.10, 0.12
McDonnell et al. (1991)	28 Males	18–30	0, 0.08
McDonnell et al. (1991)	10 Males	18–30	0, 0.08, 0.1
Folinsbee et al. (1994)	17 Males	25±4	FA, 0.12
Schelegle et al. (2009)	15 Males, 16 Females	18–25	Mean: FA, 0.06, 0.07, 0.08, 0.087 Max: n/a, 0.09, 0.09, 0.15, 0.12
Kim et al. (2011)	27 Males, 32 Females	19–35	FA, 0.06

Notes: No. = number; O₃ = ozone; ppm = parts per million; FA = filtered air; max = maximum; n/a = not available.

3. Equivalent Ventilation Rates

3.1. Original EVR Threshold

The **ModEVR8** of 13 L/min-m² typically used in regulatory runs of APEX was based on the range of 13–27 L/min-m² defined by Whitfield (1996) for 8-hour exposures. However, details were not available on how this range was obtained from the McDonnell et al. (1991) data. We analyzed the data to determine

- if the mean EVR was calculated based on all data points or based on the person-averaged EVR values, and
- the number of standard deviations away from the mean that would result in the range of values reported.

The EPA WAM provided a SAS data file with 4,024 individual EVR data points corresponding to 485 experiments. The McDonnell et al. (1991) data were provided as two separate datasets with Study IDs of “Ozi-2” and “Pokoz”, which were identified within the SAS dataset as OZI and POK, respectively. Using the McDonnell et al. (1991) OZI and POK datasets individually and combined, we calculated the mean, standard deviation, and upper and lower bounds (defined as mean ± 3 standard deviations) using (i) all individual EVR data points and (ii) person-averaged EVR values. The person-averaged EVRs are the average over time, resulting in one person-averaged EVR per unique subject and experiment, which is more consistent with how APEX evaluates whether a profile is at moderate exertion (by calculating the profile’s 8-hour-average EVR). In Table 2 we present the results of this analysis, which suggest that the range of 13–27 L/min-m² used by Whitfield (1996) was obtained using individual EVR data from the OZI dataset and three standard deviations away from the mean (see gray-shaded cells in the table).

Table 2. EVR Metrics for Individual EVR Data Points and Person-averaged EVRs, during Intermittent Moderate Exercise

	McDonnell et al. (1991) Datasets		
	OZI	POK	OZI + POK Superset
Individual EVR Data Points (L/min-m²)			
Mean	20.29	20.22	20.26
Standard Deviation	2.30	1.95	2.14
Lower Bound	13.37	14.38	13.83
Upper Bound	27.20	26.06	26.69
Person-averaged EVRs (L/min-m²)			
Mean	20.29	20.22	20.26
Standard Deviation	2.05	1.61	1.85
Lower Bound	14.15	15.39	14.72
Upper Bound	26.43	25.06	25.80

Notes: EVR = equivalent ventilation rate; L/min-m² = liters per minute per square meter; lower bound = mean - 3 standard deviations; upper bound = mean + 3 standard deviations.

Cells shaded in gray indicate metrics lining up with the 13–27 L/min-m² range of moderate-exertion EVRs defined by Whitfield (1996) for 8-hour exposures based on the McDonnell et al. (1991) data.

3.2. EVR Threshold from All Clinical Studies

ModEVR8 can be re-calculated for the expanded dataset following the original approach of three standard deviations away from the mean. In Table 3 we present the mean, median, standard deviation, and upper- and lower-bound EVRs using person-averaged EVR values from all datasets listed in Table 1.

The EVRs measured in the studies were collected during periods of exertion and represent exercise-only conditions. However, during the 6.6-hour experiment, only 5 hours were used for exercise (i.e., six 50-minute periods of treadmill or cycle ergometer), with the remaining 1.6 hours for rest or lunch. During resting times/lunch, EVR values are expected to drop. As discussed below, we estimated the impact on EVRs from incorporating rest time.

Of the studies in Table 1, only Schelegle et al. (2009) mentioned resting V_e (and, by default, resting EVR), which was estimated using regression equations derived from the data of Aitken et al. (1986). For college-age males, this was $V_e = 7.61 \times \text{BSA}$, and for college-age females, this was $V_e = 8.05 \times \text{BSA}$. These resting EVR values, 7.61 and 8.05 L/min-m² for college-age males and females respectively, are consistent with expected resting EVR values. For example, Adams (2006) reported group-mean-total and exercise-only V_e , which can be used with their reported BSAs to estimate a resting EVR of 6.38 L/min-m² for that study. In our analysis, we used those college-age male and female values to calculate resting EVR for each study, as the weighted average based on the number of males and females in the study. We then calculated total (exercise and rest) EVR as a weighted average based on 5 hours of exercise and 1.6 hours of rest/lunch. As expected, the values in Table 3 show that total (exercise and rest) EVRs are lower than exercise-only EVRs.

Table 3. EVR Metrics Derived from All Clinical Studies in Table 1, during Intermittent Moderate Exercise

	Person-averaged EVRs (L/min-m ²)	
	Exercise Only	Exercise + Rest
Mean	20.39	17.32
Standard Deviation	1.65	1.25
Lower Bound	15.44	13.57
Upper Bound	25.34	21.08
Median	20.35	17.31

Notes: EVR = equivalent ventilation rate; L/min-m² = liters per minute per square meter; lower bound = mean - 3 standard deviations; upper bound = mean + 3 standard deviations.

3.3. Parameters for Distribution Sampling

An alternative to setting **ModEVR8** to a single value is to allow it to be sampled from a distribution for each person. This introduces variability in **ModEVR8** and reflects the variability across individuals in V_e , and thus EVR, when performing moderate-exertion activities.

We modified the APEX code to allow for sampling **ModEVR8** from a distribution. The distribution parameters are specified in the modified physiology input file, where users can specify the distribution shape and corresponding parameters. For each profile, the APEX code samples **ModEVR8** from the distribution. EVR values at or above this sampled **ModEVR8** (but below **HeavyEVR8**) are classified as being at moderate exertion. The sampled **ModEVR8** values are then written to the *Profile Summary* output file.

4. Comparison of Approaches in Defining Moderate Exertion

4.1. APEX Runs

We conducted four APEX runs, listed in Table 4, to compare how different **ModEVR8** values (including dynamic sampling of values from a distribution) would affect the exposure outcomes. We used internal version APEX5.04, modified on December 20, 2018 to allow sampling of **ModEVR8** from a distribution. (A more updated version will be provided to the EPA WAM soon following this memorandum, containing additional model updates unrelated to EVR). The simulations were for the Los Angeles area, time period of January 1 to December 31, 2007, for 10,000 profiles, and for both children (ages 5 to 18 years) and total population (ages 5 years and up). We calculated the **ModEVR8** values listed in Table 4 from exercise-only data.

Table 4. Model Runs

Run Name	ModEVR8 (L/min-m ²)	Comments
EVR13	13	Original ModEVR8 value, calculated as: <ul style="list-style-type: none"> ■ Three standard deviations below the mean (see shaded lower-bound value in Table 2) ■ Using the OZI group of McDonnell et al. (1991) data ■ From individual EVR data points (instead of person-averaged EVRs)
EVR16	15.4	Updated ModEVR8 value, calculated as: <ul style="list-style-type: none"> ■ Three standard deviations below the mean (see lower-bound exercise-only value in Table 3) ■ Using the data specified in Table 1 ■ From person-averaged EVRs (instead of individual EVR data points)
EVR_Med	20.4	Median value using person-averaged EVRs from the data specified in Table 1 (see median exercise-only value in Table 3)
DIST20_1	varies	ModEVR8 sampled for each profile from a distribution. <ul style="list-style-type: none"> ■ Distribution parameters calculated using person-averaged EVRs from the data specified in Table 1 ■ Normal distribution; mean = 20.4; standard deviation = 1.7; upper truncation = 25.3; lower truncation = 15.4 (see exercise-only column in Table 3)

Notes: L/min-m² = liters per minute per square meter; EVR = equivalent ventilation rate; ModEVR8 = the model parameter for the threshold of moderate-exertion EVR for an 8-hour period.

4.2. Simulated Population Results

Across the test runs, for all profiles and children only, we compared the percent of the profiles reaching moderate exertion at least once and the person-day counts at moderate exertion. Results for both metrics and profile groups, presented in Table 5 to Table 8 and graphically in Figure 1 and Figure 2, show that as the **ModEVR8** value increases, the metrics decrease as expected (EVR13 > EVR15 > EVR_Med).

Table 5. Percent of Modeled Profiles Reaching Moderate Exertion (Ages 5 Years and Up)

Level (ppm)	Run Name (see Table 4)			
	EVR13	EVR15	EVR_Med	DIST20_1
0	86.7	66.3	18.5	20.8
0.01	84.8	64.3	17.2	19.4
0.02	83.9	63.3	16.3	18.5
0.03	82.2	61.0	14.4	16.8
0.04	79.8	57.3	12.1	14.3
0.05	76.3	51.7	9.1	11.2
0.06	69.8	43.2	6.0	7.9
0.07	57.3	31.2	3.4	4.8
0.08	38.3	18.4	1.5	2.1
0.09	18.3	7.3	0.46	0.74
0.10	3.5	1.3	0.04	0.06
0.11	0.36	0.07	0	0
0.12	0	0	0	0
0.13	0	0	0	0
0.14	0	0	0	0
0.15	0	0	0	0
0.16	0	0	0	0

Notes: ppm = parts per million.

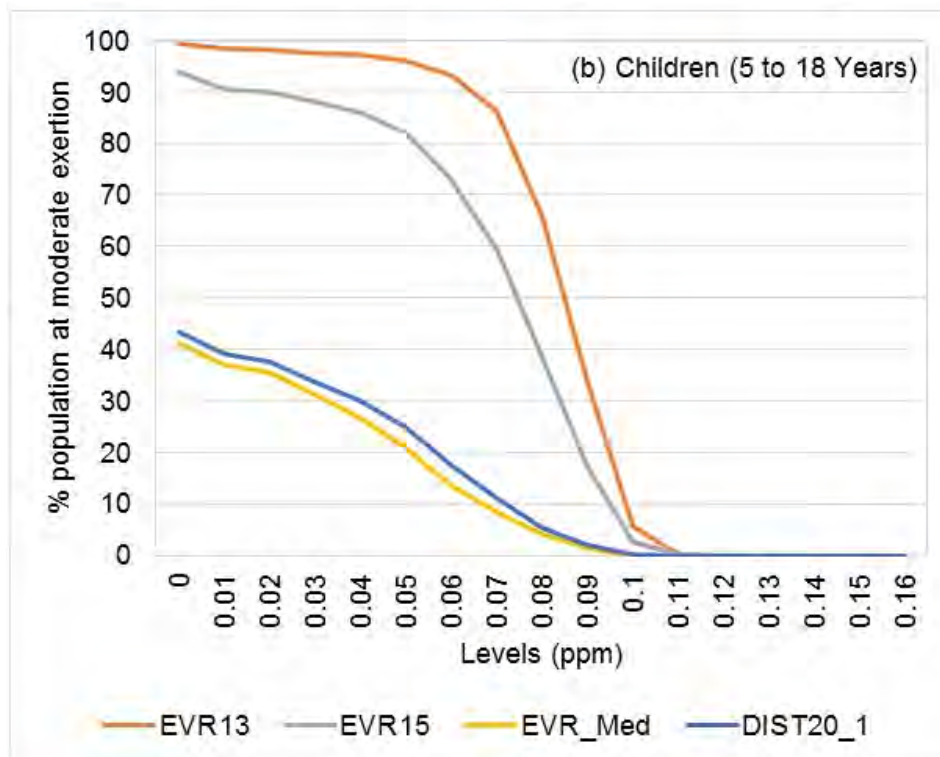
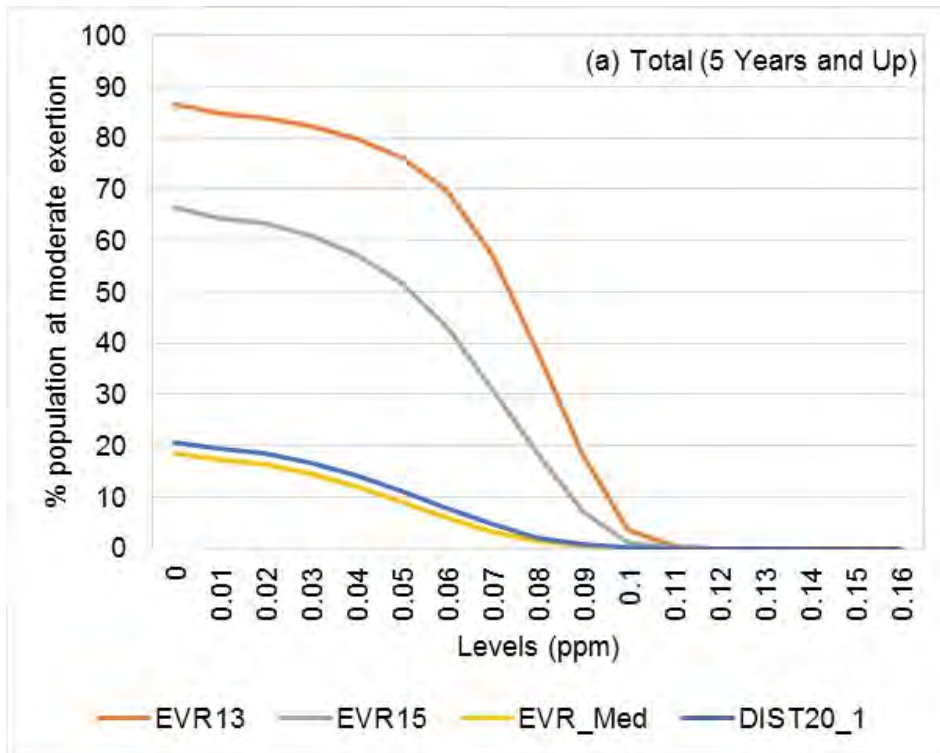
Shading indicates relative magnitude of values (reds and oranges are higher values; yellows and greens are lower values).

Table 6. Percent of Modeled Child Profiles (Ages 5 to 18 Years) Reaching Moderate Exertion

Level (ppm)	Run Name (see Table 4)			
	EVR13	EVR15	EVR_Med	DIST20_1
0	99.4	93.7	41.2	43.3
0.01	98.4	90.7	37.1	39.2
0.02	98.2	89.8	35.4	37.6
0.03	97.6	88.0	31.2	33.7
0.04	97.1	85.8	26.7	29.9
0.05	96.0	82.1	20.8	24.7
0.06	93.4	72.9	13.6	17.6
0.07	86.3	59.4	8.3	11.2
0.08	65.7	38.6	4.0	5.4
0.09	33.8	17.2	1.3	2.1
0.10	5.8	2.6	0	0.1
0.11	0.3	0.1	0	0
0.12	0	0	0	0
0.13	0	0	0	0
0.14	0	0	0	0
0.15	0	0	0	0
0.16	0	0	0	0

Notes: ppm = parts per million.

Shading indicates relative magnitude of values (reds and oranges are higher values; yellows and greens are lower values).



Notes: ppm = parts per million.

Legend entries are the run names specified in Table 4.

Figure 1. Percent of Modeled Profiles Reaching Moderate Exertion for (a) All Profiles and (b) Children Only

Table 7. Number of Modeled Person-days Reaching Moderate Exertion (Ages 5 Years and Up)

Run Name (see Table 4)				
Level (ppm)	EVR13	EVR15	EVR_Med	DIST20_1
0	1.7E+06	7.4E+05	4.9E+04	8.1E+04
0.01	1.5E+06	6.5E+05	4.1E+04	6.9E+04
0.02	1.3E+06	5.3E+05	3.2E+04	5.4E+04
0.03	9.1E+05	3.6E+05	2.2E+04	3.6E+04
0.04	5.0E+05	1.9E+05	1.1E+04	1.8E+04
0.05	2.3E+05	8.6E+04	4.8E+03	8.0E+03
0.06	9.3E+04	3.4E+04	1.9E+03	3.2E+03
0.07	3.4E+04	1.3E+04	6.8E+02	1.1E+03
0.08	1.1E+04	3.9E+03	2.0E+02	3.2E+02
0.09	2.7E+03	9.6E+02	4.8E+01	8.7E+01
0.10	4.0E+02	1.3E+02	4.0E+00	6.0E+00
0.11	3.8E+01	8.0E+00	0	0
0.12	0	0	0	0
0.13	0	0	0	0
0.14	0	0	0	0
0.15	0	0	0	0
0.16	0	0	0	0

Notes: ppm = parts per million.

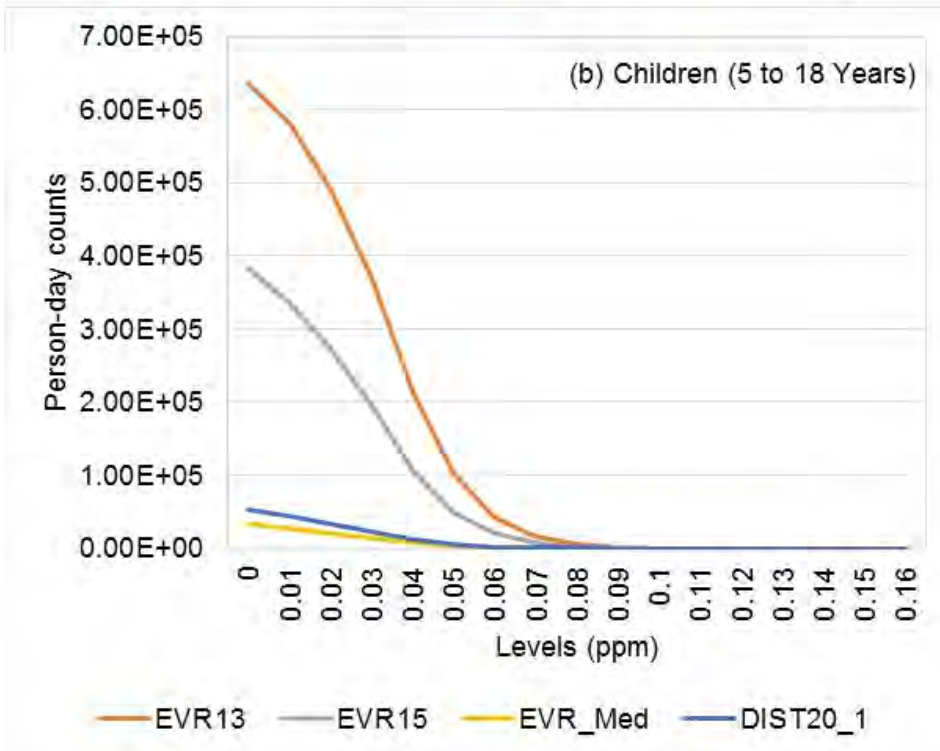
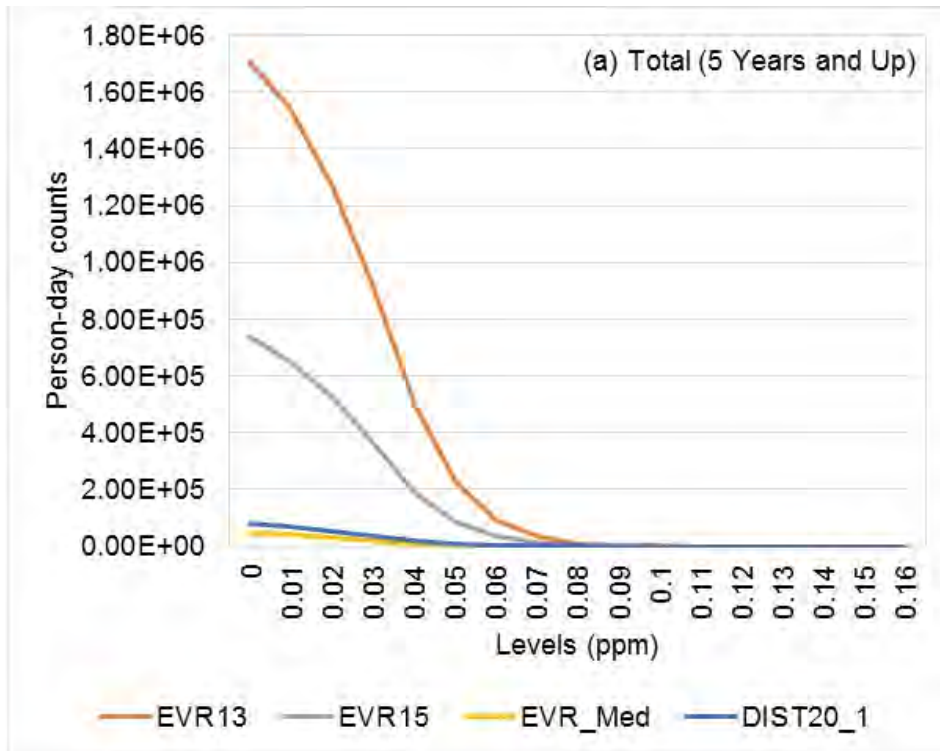
Shading indicates relative magnitude of values (reds and oranges are higher values; yellows and greens are lower values).

Table 8. Number of Modeled Person-days Reaching Moderate Exertion (Ages 5 to 18 Years)

Run Name (see Table 4)				
Level (ppm)	EVR13	EVR15	EVR_Med	DIST20_1
0	6.4E+05	3.8E+05	3.4E+04	5.2E+04
0.01	5.8E+05	3.4E+05	2.8E+04	4.4E+04
0.02	4.9E+05	2.7E+05	2.1E+04	3.4E+04
0.03	3.7E+05	2.0E+05	1.4E+04	2.3E+04
0.04	2.1E+05	1.1E+05	7.1E+03	1.2E+04
0.05	1.0E+05	4.9E+04	3.1E+03	5.2E+03
0.06	4.3E+04	2.0E+04	1.2E+03	2.1E+03
0.07	1.6E+04	7.3E+03	4.4E+02	7.3E+02
0.08	5.0E+03	2.2E+03	1.2E+02	2.0E+02
0.09	1.2E+03	5.3E+02	2.9E+01	5.6E+01
0.10	1.4E+02	6.0E+01	0	2.0E+00
0.11	7.0E+00	4.0E+00	0	0
0.12	0	0	0	0
0.13	0	0	0	0
0.14	0	0	0	0
0.15	0	0	0	0
0.16	0	0	0	0

Notes: ppm = parts per million.

Shading indicates relative magnitude of values (reds and oranges are higher values; yellows and greens are lower values).



Notes: ppm = parts per million.

Legend entries are the run names specified in Table 4.

Figure 2. Number of Modeled Person-days Reaching Moderate Exertion for (a) All Profiles and (b) Children Only

The alternative method where one **ModEVR8** value per person is sampled from a distribution resulted in higher metrics as compared to setting the **ModEVR8** equal to the median of the distribution (DIST20_1 > EVR_Med). These results are expected because sampling from the distribution allows the selection of **ModEVR8** values lower than the median value. Lower **ModEVR8** values will result in more profiles reaching “moderate exertion” in the modeling. Specifically, for person-day counts, sampling **ModEVR8** from a distribution results in counts that are more than 50 percent greater than when **ModEVR8** is set to the median value. While the sampling also allows the selection of higher **ModEVR8** values (resulting in fewer profiles reaching “moderate exertion”), profiles reach lower EVRs much more commonly than higher EVRs, so much so that using lower **ModEVR8** values brings many more profiles into the “moderate exertion” pool than are excluded when higher **ModEVR8** values are used.

However, sampling from a distribution still gives metrics that are much lower than when setting the **ModEVR8** value to three standard deviations below the mean (DIST20_1 < EVR15). As an example, for an exposure level of 0.05 parts per million, DIST20_1 results in 40 percent fewer profiles overall reaching moderate exertion at least once (11.2 percent with DIST20_1 versus 51.7 percent with EVR15), and 57 percent fewer children (82.1 percent with DIST20_1 versus 24.7 percent with EVR15). When considering person-day counts, in general, DIST20_1 counts were nearly an order of magnitude lower than EVR15 counts.

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**APPENDIX 3D, ATTACHMENT 3:
ICF TECHNICAL MEMO: UPDATES TO THE METEOROLOGY DATA AND
ACTIVITY LOCATIONS WITHIN CHAD**

MEMORANDUM

To: John Langstaff and Stephen Graham, U.S. EPA-OAQPS

From: John Hader, Graham Glen, Caroline Foster, Samuel Kovach, Delaney Reilly, Chris Holder, River Williams, Anna Stamatogiannakis, and George Agyeman-Badu, ICF

Date: June 18, 2019

Re: Updates to the Meteorology Data and Activity Locations within CHAD

1. Introduction

In the November 1, 2016 version of CHAD, approximately 18 percent (32,723 out of 179,912) of diary-days are missing values for daily-maximum temperature (Tmax) and thus cannot be used by APEX. The temperature data currently in CHAD originate from a variety of sources, including from the original studies and from EPA or contractors who encoded the study data into CHAD. As discussed in Section 2, we used a methodical process to replace most of these missing values. As part of this exercise, for diary-days without county-location information, we identified county locations for over 10,000 diary-days based on respondent zip code and for over 6,000 diary-days based on the metropolitan locations of several of the studies. Some of the diary-days that received repaired county locations were not missing temperature data; nonetheless, we made the repairs as part of a “cleaning up” of the diary data. After this process, only 0.3 percent (565) of diary-days have missing values for Tmax and remain unusable by APEX.

In the same version of CHAD, six studies have at least 200 minutes per day (on average) of time spent in locations that are not sufficiently clear (they are ambiguous). Unspecified and missing location codes are ambiguous, as are those taking place at a residence or a place of employment without specifying whether they are in the three broad microenvironments (MEs) of indoors, outdoors, or in-vehicle. If studies have an apparent bias (via ambiguity) in time spent in the three broad MEs, then the APEX-modeled exposures will also be biased. As discussed in Section 3, we used paired activity-location information from the other 15 studies in CHAD to derive frequency distributions of location codes used per each activity code, with different distributions intended for reassigning unspecified/missing locations, ambiguous residential locations, and ambiguous workplace locations. For the six targeted studies, for a diary event with an ambiguous location code, we reassigned the location code based on the activity by sampling from these frequency distributions. After this process, the time spent per day in ambiguous locations dropped substantially for the six studies, though one study still had more than 200 minutes per day spent in ambiguous locations. These location-code reassignments will substantially reduce bias in APEX exposure estimates, particularly given that one of the six studies constitutes more than half of all CHAD diary-days.

These modifications do not impact the official EPA CHAD-Master database, which remains unchanged. Instead, the modifications are specific to the version of the diary data used for APEX modeling.

2. Temperature Data

2.1. Overview and Objectives

The current CHAD questionnaire file includes Tmax and daily-average temperature (Tavg; °F) as well as daily precipitation (inches) and daily number of hours with precipitation. Only Tmax is typically used by APEX modelers, and it is used to help select a set of diaries that have similar temperature values as those experienced by a simulated profile at his/her location on a given modeling day. Diary-days without values for Tmax cannot be selected for use by any simulated profile.

As shown in Table 2-1, approximately 18 percent of diary-days are currently unusable by APEX on the basis of missing Tmax. Less than 1 percent of those are missing all indicators of respondent location (state, county, and zip code) and are not from studies of a single metropolitan area; it will not be possible to identify reasonable temperature data for those diary-days. Most of the remaining diary-days have only state information (no information on county or zip code).

Table 2-1. Information on Diary-days Missing Daily-maximum Temperature Values

	Count	Percent of All Diary-Days	Percent of Diary-days Missing Tmax
Missing Tmax	32,723	18%	100%
→ From the 1980s	14	0.008%	0.04%
From the 1990s	1,230	0.7%	4%
From the 2000s	25,512	14%	78%
From the 2010s	5,967	3%	18%
Missing All Location Information (state, county, zip code; is not a single-metropolitan study)	111	0.06%	0.3%
Is a Study of a Single Metropolitan Area	0	0%	0%
Has State Location but not County (and is not a single-metropolitan study)	30,895	17%	94%
→ Has Zip Code	30	0.02%	0.09%

Notes: Studies limited to one metropolitan area were put into CHAD without county or zip-code information.
Tmax = daily-maximum temperature

The objective of this task is to use historical meteorological records to identify reasonable temperature values for diary-days currently missing those values. Identifying these values relies on knowing or estimating the geographic location of each diary-day. Since most of the target diary-days identify the respondent's state but not county or zip code, in most cases we have made assumptions about respondent locations within the state.

A structured methodology of identifying appropriate temperature data allows us to identify reasonable temperature values for nearly all diary-days, not just those currently missing temperature data. While we will generally not update temperature data in CHAD that are not already missing (unless we believe the current values are erroneous), we can compare current and "new" temperatures as part of quality control (QC). With this in mind, as detailed in Section 2.2, we developed a hierarchy to assign a county location to nearly all diary-days. Then, as detailed in Section 2.3, we matched county locations to the five closest meteorological stations from the historical records, thus enabling the assignment of temperature values.

2.2. Assigning County Locations to Diary-days

Matching diary-days with nearby meteorological stations requires knowing (or estimating) where the diary-days took place. County is the primary indicator of diary location, though zip codes are also available for some diaries, and assigning temperature data on a county basis is reasonable given the typical spatial resolution of counties and typical temperature gradients.

About 43 percent (77,811) of all diary-days already had county designations. For these diary-days, we “cleaned up” the county names to be more consistent with the names provided by the U.S. Census Bureau. While the county and state locations of diary-days are not used in APEX, creating consistent location designations (and use of the more reliable state-county FIPS designations) made the temperature-assignment process more reliable.

The remaining 57 percent (102,101) of all diary-days had no county locations. As indicated in Table 2-2, 111 had no location information at all and they were not from studies located in a single metropolitan area. We could not assign counties to these 111 diary-days, and thus we could not replace missing temperature data if needed.

Table 2-2. Information on Diary-days Without County Designations

		Count	Percent of All Diary-Days	How County Locations Were Determined (showing counts of diary-days)		
				Metropolitan Study Location	Zip Code	State's Population Distribution
Missing All Location Information (state, county, zip code; is not a single-metropolitan study)		111	0.06%	0	0	0
Is a Study of a Single Metropolitan Area		6,150	2%	6,150	0	0
Has State Location but not County (and is not a single-metropolitan study)		95,840	55%	0	0	84,141 (14 from 1980s; 6,139 from 1990s; 64,046 from 2000s; 13,942 from 2010s)
→	Has Zip Code	11,699	7%	0	11,635	64 (1 from 1980s; 62 from 1990s; 1 from 2000s; 0 from 2010s)

Note: Studies limited to one metropolitan area were put into CHAD without county or zip-code information.

For the other 101,990 diary-days without county designations, a small amount (6,150) were from studies located within a single metropolitan area. Diary-days from these studies were originally put into CHAD without county or zip-code information. We made the assumption that all such respondents lived in the primary county associated with the area, as listed below.

- Hamilton County, Ohio for the Cincinnati Activity Patterns Study (CIN)
- Wayne County, Michigan for the Detroit Exposure and Aerosol Research Study (DEA)
- Denver County, Colorado for the Denver, Colorado Personal Exposure Study (DEN)
- King County, Washington for the Seattle Study (SEA)
- District of Columbia for the Washington, DC Study (WAS)

Additionally, a small amount (11,635) of diary-days without county designations had reliable zip codes that we geocoded to their most likely counties, following the process listed below. Note that we used geospatial files representing the year 2000 because most of the CHAD diary-days (129,569 diary-days, which is 72 percent of all diary-days) were from the 2000s, and county boundaries have remained unchanged through the last few decades for nearly all U.S. counties.

- Use GIS software to convert the year-2000 county polygons¹ to centroid points (one centroid per county).
- Use GIS software to identify the county centroid (year 2000) closest to each zip-code centroid (also year 2000; from the zip-code tabulation areas file.² These centroid-proximity matches were restricted to within the same state (e.g., a zip-code centroid located in California could only be matched to a county in California).
- A small number of zip codes (145) could not be identified in the Gazetteer files. We identified the county locations of 85 such zip codes with reasonable confidence using Internet searches, leaving 60 zip codes unmatched to counties.

For the remaining 84,205 diary-days without county designations (which includes 64 diary-days that could not be reliably matched to counties via zip code), we assigned them to counties within the state based on population distributions. We used U.S. Census data to calculate the population distributions within each state. Since such distributions change over time, we did this on a decadal basis, covering the decades represented by the CHAD diary-days (the 1980s through 2010s), as indicated below. The majority of such population-based assignments were for diary-days in the 2000s decade (as indicated in Table 2-2).

- **2000s and 2010s:** We queried decadal census data from the U.S. Census Bureau (filtering by Population Total, the 2010 or 2000 year, and All Counties within United States).³ The SF1 100% datasets were employed.
- **1980s and 1990s:** We used intercensal data from the U.S. Census Bureau's State and County Intercensal Datasets websites for 1980 to 1989 and 1990 to 1999.⁴ The county

¹ From the U.S. Census cartographic boundary files available at <https://www.census.gov/geographies/mapping-files/time-series/geo/cartographic-boundary-file.2000.html>.

² From the U.S. Census Gazetteer files available at <https://www.census.gov/geographies/reference-files/time-series/geo/gazetteer-files.2000.html>.

³ The American FactFinder website, used at the time of these analyses were performed, has been decommissioned as of March 30, 2020. Similar data queries can be made at <https://data.census.gov/cedsci/>.

⁴ Data available at <https://www.census.gov/data/tables/time-series/demo/popest/1980s-county.html> and <https://www.census.gov/data/datasets/time-series/demo/popest/intercensal-1990-2000-state-and-county-characteristics.html>.

populations were partitioned by demographics, which we aggregated to county-total population values.

2.3. Assigning Temperature Data to Diary-days

The National Centers for Environmental Information (NCEI) distributes several databases of land-based meteorology station data. We utilized the Global Historical Climatology Network–Daily (GHCND), as it provided QCed daily temperature data at a relatively high spatial resolution across the U.S.⁵ We narrowed the GHCND database based on the criteria listed below.

- Stations must be located 24–50° N and 126–66° W (for contiguous U.S.), 51–72° N and 179.999–129° W (for Alaska; we did not use any stations in the far-western Aleutian Islands), and 18.5–22.5° N and 160.5–154.5° W (for Hawaii). Note that these boundaries may extend somewhat into neighboring countries.
- Stations must include Tmax and daily-minimum temperature (Tmin) as typically reported parameters (requiring Tavg was too restrictive; we elected to calculate Tavg as the average of Tmax and Tmin).
- On a decadal basis, stations must report data for the entirety of that decade (or for 2010–2014 for the 2010s).

Some of the GHCND stations were of ‘higher quality’ than others, as they are part of the U.S. Historical Climatology Network (HCN), the U.S. Climate Reference Network (CRN) and/or the Global Climate Observing System Surface Network (GSN). We preferred data from these stations in our temperature assignments.

In Table 2-3, we indicate the number of meteorological stations per decade, including the number of higher-quality stations, that meet all the selection criteria listed above. In Figure 2-1 and Figure 2-2, for the 1980s and 2010s respectively, we show examples of the geographic spread of meteorology stations (with higher-quality stations differentiated) in North and South Carolina.

Table 2-3. Number of GHCND Meteorological Stations Meeting Selection Criteria, per Decade and U.S. Region

Year	Number of Meteorological Station Counts (higher-quality Stations) ^a		
	Contiguous U.S.	Alaska	Hawaii
1980	6,621 (1,225)	230 (19)	54 (2)
1990	7,207 (1,233)	251 (19)	56 (2)
2000	7,813 (1,151)	341 (21)	72 (2)
2010	8,445 (1,210)	388 (29)	85 (4)

^a Note that a small number of stations included here may be across the U.S. border in other countries.

⁵ <https://www.ncdc.noaa.gov/data-access/land-based-station-data/land-based-datasets>.

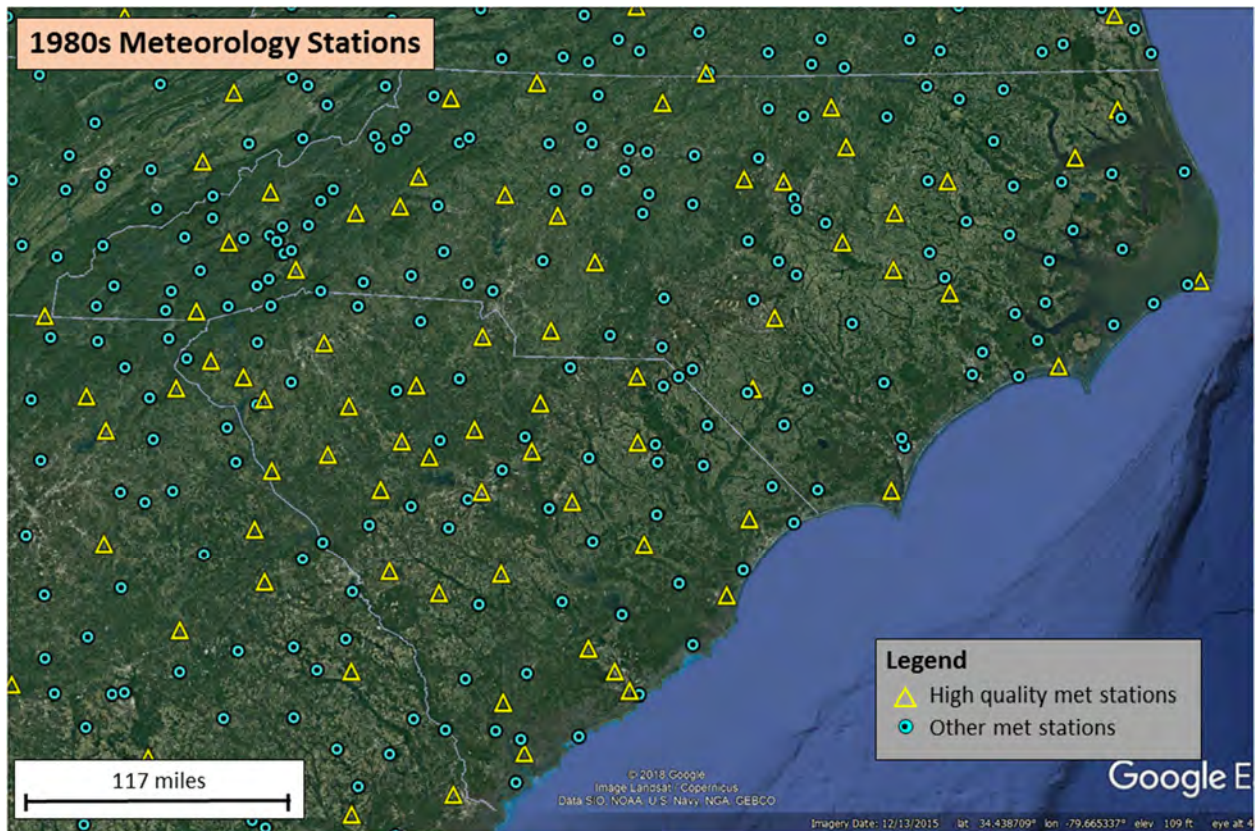


Figure 2-1. GHCND Meteorological Stations from the 1980s Meeting Selection Criteria, in the North and South Carolina Region

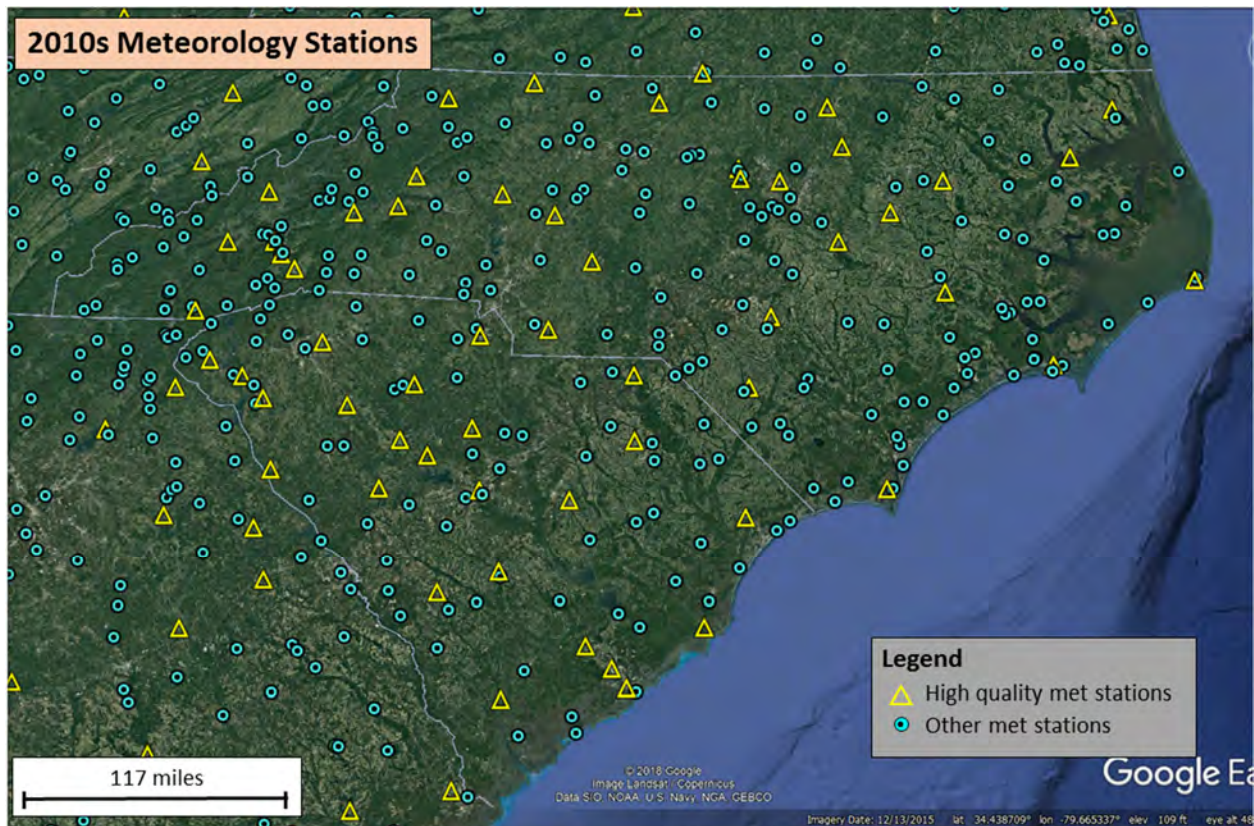


Figure 2-2. GHCND Meteorological Stations from the 2010s Meeting Selection Criteria, in the North and South Carolina Region

By decade (with county locations fixed at the year-2000 definitions), we used ArcMap’s “Generate Near Table” tool to map each U.S. county to its five closest meteorological stations from the GHCND dataset. The stations were initially sorted by closest proximity to the county centroid. Then, we resorted the matches to ensure that the closest higher-quality within 30 miles of the county centroid was the preferred station of the five stations.

The median distance from county centroid to the preferred meteorological station was 19 km—only in Alaska were some county centroids more than 100 km from the preferred station, and a few counties in Arizona, California, Nevada, and Texas were 50–70 km from the preferred station. The median distance from county centroid to the fifth selected station was 42 km.

Based on the county location and decade of the diary-day, and the five meteorological stations selected for that county and decade, we identified Tmax and Tmin from the preferred station. If the preferred station’s Tmax and Tmin values were missing, then we used the values from the second station, and so on until we identified non-missing values. If none of the five stations supplied non-missing Tmax and Tmin values, then the values were left missing.

Using the method above, 178,893 diary-days (> 99 percent) were matched with new Tmax and Tavg values, leaving 1,019 diary-days (0.6 percent) without matched values. As a QC check, we compared the newly matched temperature values (“new” temperatures) to the existing temperature values where available (“old” temperatures). Using Tmax, there were 146,735 diary-days (82 percent) available for comparison. In Table 2-4, we indicate how many diary-days were negligibly different ($\leq 5^\circ$), 5–10° different, 10–20° different, or > 20° different.

Table 2-4. Comparison of Old (in Current CHAD-Master) and New (Identified Here) Daily-maximum Temperatures

Difference between Old Tmax and New Tmax	Number of Diary-days	Percent of Diary-days Available for Comparison
≤ 5 °F	101,507	69.2%
5–10 °F	24,604	16.8%
10–20 °F	16,032	10.9%
> 20 °F	4,592	3.1%

During this QC check, we further examined the 4,592 diary-days (3 percent) where the Tmax values were > 20° different. During this step, we discovered that most of these diary-days were from the American Time Use Survey by the Bureau of Labor Statistics (BLS). In 2,431 of the 4,592 diary-days with differences over 20°, they were from the BLS study *and* the old Tmax was equivalent to the old Tavg. This indicated a systematic error in the old BLS temperatures.

Using a similar approach, we compared the old and new Tavg values. The results are indicated in Table 2-5. The results comparing the old and new Tavg values were similar to those for Tmax.

Table 2-5. Comparison of Old and New Average Temperatures

Difference between Old Tmax and New Tmax	Number of Diary-days	Percent of Diary-days Available for Comparison
≤ 5 °F	109,632	74.7%
5–10 °F	24,430	16.6%
10–20 °F	10,271	7.0%
> 20 °F	2,363	1.6%

We further examined the 2,363 diary-days (1.3%) where differences in Tavg values were > 20°. For 1,569 of these diary-days, they were from the BLS study *and* the old Tavg was equivalent to the old Tmax, again indicating a systematic error in the old BLS temperatures.

As an additional check, we examined the mean Tmax and mean Tavg across all diary-days. The mean Tmax and mean Tavg for the old values were 68.0° and 58.4°, respectively. For the new data, the mean Tmax and mean Tavg were 68.4° and 57.8° respectively. The consistency between the two was expected and provides additional assurance.

At the direction of EPA, and given the errors found in the temperatures of the BLS study, we developed a diary dataset using a combination of the old and new temperatures. To create this dataset, we replaced all the old temperatures (maximum and average) of the BLS diary-days. Next, we replaced all previously missing values where new values were available (across all studies). Following these rules, we replaced values for 125,581 diary-days, such that the new diary dataset now has Tmax and Tavg values for 179,347 diary-days. Temperatures remain missing for 565 diary-days, while 53,766 diary-days retained their old temperatures.

In addition to the new temperature data, we updated the dataset with information that was used as intermediate to this process, with fields indicated in Table 2-6.

Table 2-6. Updated or Added Fields in the CHAD Dataset

Field Name	Description
county	Values updated to include newly georeferenced data
state	Values updated to include newly georeferenced data
FIPS	Field added to provide a unique ID to every state-county

Field Name	Description
old_avgtemp	Field renamed to identify the temperatures (°F) in the November 2016 CHAD
old_maxtemp	Field renamed to identify the temperatures (°F) in the November 2016 CHAD
FIPSfromZip	Field added: TRUE or FALSE—if the county originally was missing, did we identify by zip code?
FIPSfromStudy	Field added: TRUE or FALSE—if the county originally was missing, did we identify by study location?
FIPSfromCountyRandom	Field added: TRUE or FALSE—if the county originally was missing, did we identify by county population distributions in the state?
new_avgtemp	Field added to provide new temperatures (°F) queried in this task
new_maxtemp	Field added to provide new temperatures (°F) queried in this task
ReplacedMaxTemp	Field added to provide the final temperatures (°F) to use in future applications (either the old or new value, depending on the study and other criteria as discussed in this memorandum)
ReplacedAvgTemp	Field added to provide the final temperatures (°F) to use in future applications (either the old or new value, depending on the study and other criteria as discussed in this memorandum)

3. CHAD Activity Locations

3.1. Introduction

Each diary-day reports a series of “events” covering 24 hours. Event durations vary, but each event has one location code and one activity code. To use diaries in APEX, the location codes are mapped to APEX MEs, each of which has a method for determining its air quality. While the number of MEs is flexible, generally all APEX runs distinguish between time spent in three basic MEs: indoor, outdoor, and in-vehicle. Yet six of the location codes are ambiguous, even at that coarse level of defining MEs (i.e., they do not distinguish between the three basic MEs). CHAD is composed of 21 originally separate studies, and some of these studies use these ambiguous codes, but others do not.

These six ambiguous location codes are shown below, and in Table 3-1 we show the average amount of time spent in ambiguous locations (by study).

- Residential:
 - ◆ 30000 (Residence, general)
 - ◆ 30010 (Your residence)
 - ◆ 30020 (Other’s residence)
- Workplace:
 - ◆ 33400 (At work: no specific location, moving among locations)

- Unknown:
 - ◆ U (Uncertain)
 - ◆ X (Missing)

Table 3-1. Average Amount of Ambiguous Time by Study

Study	Average Ambiguous Time (minutes per day)
BAL: Baltimore Retirement Home Study	3
BLS: American Time Use Survey (ATUS), Bureau of Labor Statistics	498
CAA: California Adults Activity Pattern Studies	67
CAC: California Children Activity Pattern Studies	0
CAY: California Youth Activity Pattern Studies	101
CIN: Cincinnati Activity Patterns Study	2
DEA: Detroit Exposure and Aerosol Research Study	1,186
DEN: Denver, Colorado Personal Exposure Study	16
EPA: EPA Longitudinal Studies	333
ISR: Population Study of Income Dynamics I, II, III	58
LAE: Los Angeles Ozone Exposure Study: Elementary School	34
LAH: Los Angeles Ozone Exposure Study: High School	2
NHA: National Human Activity Pattern Study: Air	18
NHW: National Human Activity Pattern Study: Water	18
NSA: National-scale Activity Study	154
OAB: RTI Ozone Averting Behavior Stud	121
RTP: RTP Particulate Matter Panel Study	1,081
SEA: Seattle Study	1,205
SUP: Study of Use of Products and Exposure-related Behaviors	804
VAL: Valdez Air Health Study	2
WAS: Washington, DC Study	16

Note: Bolded studies have relatively large average amounts of ambiguous time.

APEX assigns MEs based only on the location code (not the activity code), and furthermore, APEX uses a deterministic mapping (that is, the same location code maps to the same ME throughout that APEX run). But this rule may lead to an unavoidable bias if applied to certain diary studies. We examined the CHAD activity code that is paired with each location code (on the event level), to determine the likely place of occurrence of each event. Since this is not always a certainty, part of this exercise is to probabilistically assign specific locations to events with ambiguous location codes, based on the paired activity.

3.2. Methods

The starting point is the November 2016 version of CHAD. It has 179,912 diary-days. Two of those (EPA002171 and EPA002172) have been deleted because they each contained 24 hours of missing data.

For our purposes, we divided all location codes into six general MEs and temporarily related them to the location codes shown as shown below, which are unambiguous. The codes are typical examples of the categories shown. For example, 31110 is a car; while not all vehicular travel is in a car, it is reasonable that the air quality in a car would be similar to that found in other types of vehicles.

- IH (indoors at a residence) → Code 30120 (Your residence, indoor)

- IO (indoors elsewhere) → Code 32000 (Other, indoor general)
- OH (outdoors at a residence) → Code 30200 (Residence, outdoor)
- OV (outdoors near traffic) → Code 35200 (Public garage / parking lot)
- O (outdoors elsewhere) → Code 35000 (Other outdoor, general)
- V (in an enclosed vehicle) → Code 31110 (Motorized travel by car)

The six ambiguous location codes had more than one mapping option for a location category, as shown below. They were reassigned location codes based on activity (and occupation where applicable), as discussed later.

- Codes 30000 (residence, general), 30010 (your residence), 30020 (other's residence)
 - ◆ Could be either IH or OH; occasionally V or OV
- Code 33400 (at work; no specific location, moving among locations)
 - ◆ Could be any, but depends on occupation
 - Occupation TRANS (transportation and material moving)
 - V (specifically 31120, travel by truck)
 - Occupation FARM (farming, forestry, and fishing)
 - O
 - Occupation HSHLD (private household)
 - IH
 - Activity code ≥ 18000 (travel)
 - V
 - Activity codes 17700–17823 (active-leisure activities; exercise activities)
 - OV
 - All others
 - IO
- Codes U (uncertain), X (missing)
 - ◆ Could be any

For analysis purposes, we divided CHAD into two parts. The “bad” part consisted of the six studies with at least 200 minutes per day on average spent in ambiguous locations (see Table 3-1; the studies were BLS, DEA, EPA [EPA Longitudinal Studies], RTP [RTP Particulate Matter Panel Study], SEA, and SUP [Study of Use of Products and Exposure-related Behaviors]). The “good” part consisted of the 15 studies with an average of fewer than 200 minutes per day of ambiguous time.

For the purposes of replacing location codes U and X in the “bad” part of CHAD, we analyzed the “good” part to determine the time fractions in each of the six location categories for each activity code (except activity codes U and X). We excluded any time in ambiguous locations. For example, the “eating” code (14400) divided as IH = 76 percent, IO = 21 percent, OH = 2 percent, O = 1 percent, and OV and V = less than 1 percent. A few activity codes did not have examples in the “good” part of CHAD, and so we mapped them to similar activities. These cases occurred extremely rarely in the “bad” part of CHAD, as well. The number of such cases increased if we stratified CHAD by age group, and for most activities the allocation to the six location categories was not very different between age groups. Therefore, we did not treat age groups separately. We linked the time-fraction distributions to the activities in the six studies in the “bad” part of CHAD. We reassigned U and X locations by activity (excluding activity codes U and X), following these distributions from the “good” part of CHAD.

For the purposes of replacing ambiguous residential location codes (30000 – Residence, general; 30010 – Your residence; and 30020 – Other’s residence), we made separate time-fraction determinations (also from the “good” part of CHAD) where we generally restricted time to three categories: IH, OH, and OV. We used the last of these (OV) for time in the garage or working on cars. We made an exception for selected travel activity codes over 18000, which indicate that the person was in a vehicle. For example, we assigned 18031 (drive a motor vehicle) and similar codes to V. We linked these refined time-fraction determinations to the activities in the six studies in the “bad” part of CHAD, for all events with location codes 30000, 30010, or 30020. We reassigned these locations by activity (for activities other than U and X), following these distributions of time spent. We made an exception for the DEA study, where it was clear that the residential codes up to 30020 were used only for indoor events. Note the before the location reassignments, the DEA study averaged 83 minutes in OH locations but only 29 minutes in IH locations.

In many cases, the same diary had the same activity code for several consecutive events with ambiguous location codes. For example, the person might be sleeping for several hours, but the location is not clear. It would not make sense for them to be relocated part way through, so for such consecutive events we determined the reassignment (from the activity’s distribution across the six location categories) only for the first of such events, and then subsequent events received the same new location reassignment.

3.3. Discussion

As shown in Table 3-2, five of the six studies where we reassigned location codes now have fewer than 200 minutes per day of ambiguous location time. The exception is the SUP study, in which most diaries were shorter than 24 hours and were padded with missing activities and locations to fill out the day. Many of the SUP diaries were previously rejected by APEX, and might continue to be, but most of the other diaries will now be acceptable. In particular, the BLS diaries constitute more than half of CHAD, and they have gone from 498 ambiguous minutes to just 10 such minutes per diary-day.

Table 3-2. Minutes per Day in the Six Location Categories, Before (“Old”) and After (“New”) Location Reassignments, For the Six Studies With 200 Minutes per Day or More of Time Spent in Ambiguous Locations

Location Category	BLS		DEA		EPA		RTP		SEA		SUP	
	Old	New	Old	New	Old	New	Old	New	Old	New	Old	New
IH	754	1,049	29	1,157	677	903	90	973	0.04	1,121	327	787
IO	79	228	48	95	246	346	131	170	139	145	175	176
OH	22	47	83	83	50	55	36	77	16	73	22	47
O	17	23	19	19	23	23	17	17	24	25	45	45
OV	0.3	1.7	3.3	3.4	24	24	5.8	6.8	1.0	2.1	5.0	5.1
V	70	81	72	72	87	87	80	80	54	54	61	61
Ambiguous	498	10	1,186	10.3	333	2.4	1,081	116	1,205	21	804	317
Indoor Total	833	1,277	78	1,252	923	1,249	220	1,143	139	1,265	503	963
Outdoor Total	39	72	105	106	96	102	59	101	41	99	72	98

Several questions remained, as listed below. We discussed these questions with EPA in May 2019, with decisions noted below.

1. Should the “good” part of CHAD be defined differently?
 - a. No, keep it as-is.
2. Should other location codes be deemed ambiguous?
 - a. Not at this time.
3. Should this method be applied to the ambiguous events in “good” CHAD?
 - a. No.

The last question is perhaps the most important. The CAY, NSA, and OAB studies average over 100 minutes of ambiguous time per diary, which is significant. The same method could be applied there, and might significantly reduce the ambiguous time in those studies. One reason not to apply this method is that the time percentages would then be applied to some of the same studies used to derive the percentages, and this presents the appearance of circular reasoning. It is not exactly circular because we excluded ambiguous time when deriving the percentages, but even so, there may be a correlation between the choice of location code and choice of activity code within a single study. For example, there may be a reason particular to the given study for why some eating events were assigned specific location codes, and others were assigned location X. Hence, it is not clear whether general percentages for all eating events should apply to those (relatively few) coded with location X. This is less of a concern when most or all eating events are paired with location X.

4. Diagram of Processing

In Figure 4-1, we indicate the input and output files for the temperature and location-code updates discussed above, as well as the processing programs and ancillary files. We briefly discuss these files and programs below the figure.

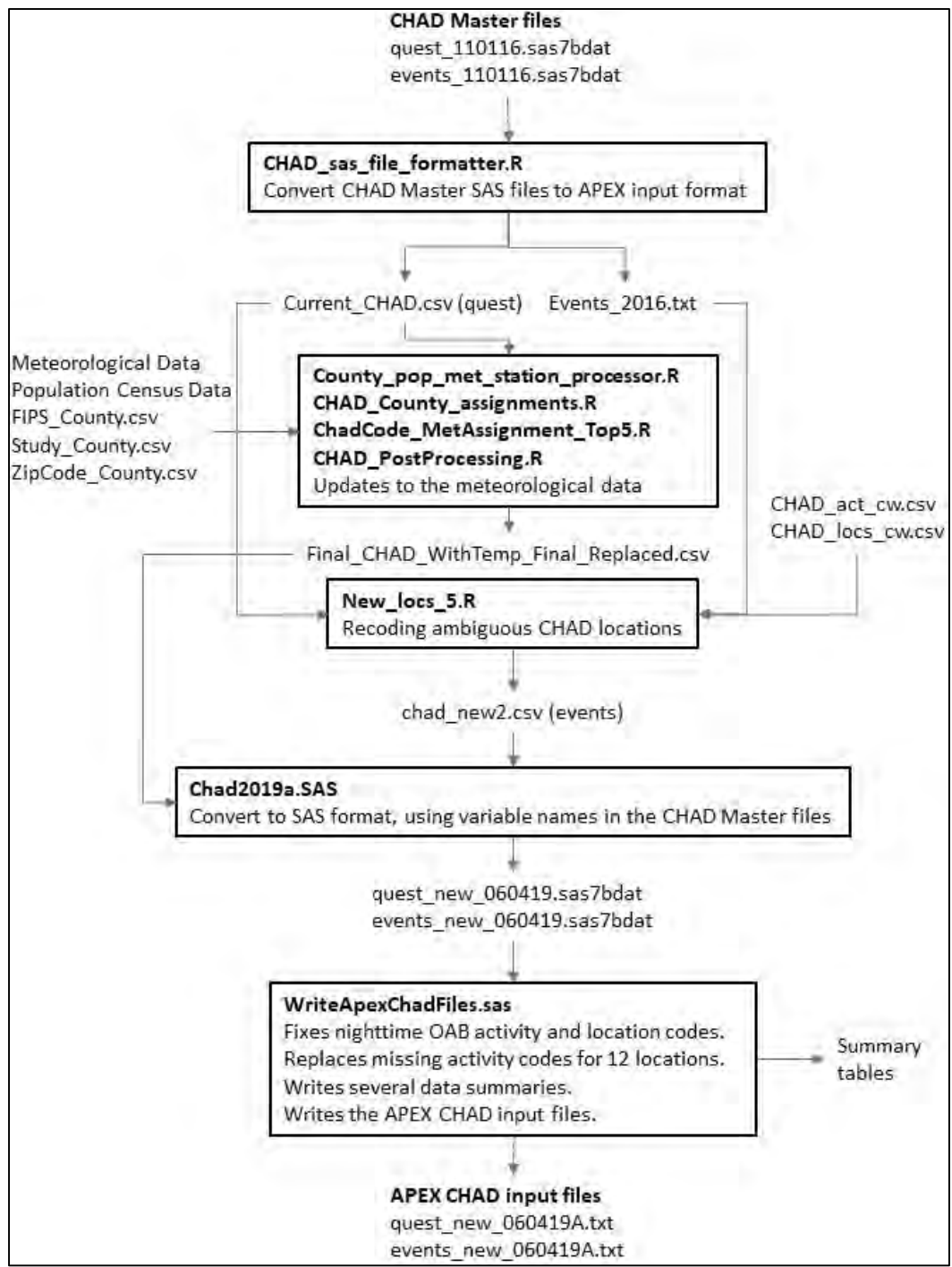


Figure 4-1. Files and Processing Programs Used in this Task

Both the temperature and location-code tasks began with the November 2016 version of the CHAD-master files (*quest_110116.sas7bdat* and *events_110116.sas7bdat*), which we converted to text or CSV files (*Current_CHAD.csv* for the questionnaire file; *Events_2016.txt* for the events file) for easier processing in R programs.

We used four different R scripts to modify temperatures and county designations in the questionnaire file. *County_pop_met_station_processor.R* reformatted GIS data, outputting the

ranking of up to five meteorology stations for every county, by decade and reorganized based on distance and station quality. *CHAD_County_assignments.R* filled in missing location data, based on zip code, study, and random assignment based on population density. *ChadCode_MetAssignment_Top5.R* combined the outputs of the previous two scripts to assign temperatures (and other intermediate details) the questionnaire file. *CHAD_PostProcessing.R* cleaned the data of unnecessary fields and reformatted the data for processing back into a SAS dataset. The resulting updated questionnaire file was *Final_CHAD_WithTemp_Final_Replaced.csv*.

The location-code reassignments were made by *New_locs_5.R* (where 5 is the version number of the script). The output events file was *chad_new2.csv*.

The new questionnaire and events files were not directly suitable as input to APEX because they contains extra variables, including both the old and new location codes, details about county reassignments and meteorological stations, etc. The program *Chad2019a.sas* converted the files to SAS format and utilized field names conforming to those of CHAD-Master, producing *quest_new_060419.sas7bdat* and *events_new_060419.sas7bdat*.

Finally, the EPA WAM's program (*WriteApexChadFiles.sas*) processed the above-mentioned SAS datasets in various ways, most importantly producing the APEX-ready diary files (*quest_new_060419A.txt* and *events_new_060419A.txt*).

**APPENDIX 3D, ATTACHMENT 4:
DETAILED EXPOSURE AND RISK RESULTS**

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Atlanta	S65	2015	0	782548	702709	652006	613670	581448	553463
All Children	Atlanta	S65	2015	10	737635	650392	594402	553161	518598	488576
All Children	Atlanta	S65	2015	20	638508	531491	470336	424273	390699	360596
All Children	Atlanta	S65	2015	30	498684	383940	321655	279970	246820	222104
All Children	Atlanta	S65	2015	40	321736	210321	154209	118800	93619	74875
All Children	Atlanta	S65	2015	50	110083	37448	14991	6335	2805	1352
All Children	Atlanta	S65	2015	60	11501	585	101	20	0	0
All Children	Atlanta	S65	2015	70	424	20	0	0	0	0
All Children	Atlanta	S65	2015	80	0	0	0	0	0	0
All Children	Atlanta	S65	2015	90	0	0	0	0	0	0
All Children	Atlanta	S65	2015	100	0	0	0	0	0	0
All Children	Atlanta	S65	2016	0	790356	714553	665988	626301	592142	564218
All Children	Atlanta	S65	2016	10	752243	669035	614820	572006	537181	508631
All Children	Atlanta	S65	2016	20	662941	562038	500480	454800	419047	390195
All Children	Atlanta	S65	2016	30	534497	422316	358800	315319	284086	259612
All Children	Atlanta	S65	2016	40	357993	246719	191476	156167	129352	107985
All Children	Atlanta	S65	2016	50	139824	55465	25887	13720	6981	3773
All Children	Atlanta	S65	2016	60	21165	1917	202	61	0	0
All Children	Atlanta	S65	2016	70	1473	20	0	0	0	0
All Children	Atlanta	S65	2016	80	81	0	0	0	0	0
All Children	Atlanta	S65	2016	90	0	0	0	0	0	0
All Children	Atlanta	S65	2016	100	0	0	0	0	0	0
All Children	Atlanta	S65	2017	0	787491	708984	655456	615486	582962	554835
All Children	Atlanta	S65	2017	10	743406	655093	596157	553746	519042	489362
All Children	Atlanta	S65	2017	20	640061	533004	470074	424596	389488	360696
All Children	Atlanta	S65	2017	30	493943	379743	317155	274704	244822	220005
All Children	Atlanta	S65	2017	40	295708	189377	137604	105160	82462	64767
All Children	Atlanta	S65	2017	50	70477	18744	6638	2320	868	504
All Children	Atlanta	S65	2017	60	3955	40	0	0	0	0
All Children	Atlanta	S65	2017	70	40	0	0	0	0	0
All Children	Atlanta	S65	2017	80	0	0	0	0	0	0
All Children	Atlanta	S65	2017	90	0	0	0	0	0	0
All Children	Atlanta	S65	2017	100	0	0	0	0	0	0
All Children	Atlanta	S70	2015	0	782548	702709	652006	613670	581448	553463
All Children	Atlanta	S70	2015	10	741489	655557	599325	558225	524187	494528
All Children	Atlanta	S70	2015	20	653358	549630	486417	441806	406941	376999
All Children	Atlanta	S70	2015	30	530462	416666	353433	311526	277266	250391
All Children	Atlanta	S70	2015	40	382185	267884	208121	169826	141680	119163
All Children	Atlanta	S70	2015	50	191173	94729	54598	32787	19470	11985
All Children	Atlanta	S70	2015	60	38981	6618	1493	363	81	20
All Children	Atlanta	S70	2015	70	4580	182	20	0	0	0
All Children	Atlanta	S70	2015	80	182	0	0	0	0	0
All Children	Atlanta	S70	2015	90	0	0	0	0	0	0
All Children	Atlanta	S70	2015	100	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Atlanta	S70	2016	0	790356	714553	665988	626301	592142	564218
All Children	Atlanta	S70	2016	10	755774	673776	620167	577675	543436	513857
All Children	Atlanta	S70	2016	20	676823	578381	517468	472071	435531	407264
All Children	Atlanta	S70	2016	30	564036	453105	391244	346311	313302	286891
All Children	Atlanta	S70	2016	40	415839	301135	242926	204631	177513	153927
All Children	Atlanta	S70	2016	50	226825	124368	77559	51148	35349	24131
All Children	Atlanta	S70	2016	60	63455	13801	3914	1190	343	101
All Children	Atlanta	S70	2016	70	9947	282	20	0	0	0
All Children	Atlanta	S70	2016	80	1211	0	0	0	0	0
All Children	Atlanta	S70	2016	90	121	0	0	0	0	0
All Children	Atlanta	S70	2016	100	0	0	0	0	0	0
All Children	Atlanta	S70	2017	0	787491	708984	655456	615486	582962	554835
All Children	Atlanta	S70	2017	10	747340	660036	602573	559436	524914	495012
All Children	Atlanta	S70	2017	20	654730	549529	486094	440414	406356	376495
All Children	Atlanta	S70	2017	30	528767	412591	348328	305534	273877	248333
All Children	Atlanta	S70	2017	40	359082	248031	191112	155198	130058	108005
All Children	Atlanta	S70	2017	50	147228	62003	31597	17029	9887	5770
All Children	Atlanta	S70	2017	60	17291	1675	343	61	0	0
All Children	Atlanta	S70	2017	70	1069	0	0	0	0	0
All Children	Atlanta	S70	2017	80	0	0	0	0	0	0
All Children	Atlanta	S70	2017	90	0	0	0	0	0	0
All Children	Atlanta	S70	2017	100	0	0	0	0	0	0
All Children	Atlanta	S75	2015	0	782548	702709	652006	613670	581448	553463
All Children	Atlanta	S75	2015	10	744495	658725	602654	561595	528021	498018
All Children	Atlanta	S75	2015	20	664192	562724	500016	454921	419471	389953
All Children	Atlanta	S75	2015	30	557055	442896	379057	335698	301862	274260
All Children	Atlanta	S75	2015	40	427219	312717	251642	210442	180197	156651
All Children	Atlanta	S75	2015	50	269115	159395	108792	77417	55808	41201
All Children	Atlanta	S75	2015	60	92348	28126	10573	4136	1897	807
All Children	Atlanta	S75	2015	70	16202	1412	141	40	20	0
All Children	Atlanta	S75	2015	80	2320	40	0	0	0	0
All Children	Atlanta	S75	2015	90	141	0	0	0	0	0
All Children	Atlanta	S75	2015	100	0	0	0	0	0	0
All Children	Atlanta	S75	2016	0	790356	714553	665988	626301	592142	564218
All Children	Atlanta	S75	2016	10	758457	677267	623819	581610	547632	517852
All Children	Atlanta	S75	2016	20	687718	591173	529352	485186	449635	419975
All Children	Atlanta	S75	2016	30	588470	478528	415052	370825	336464	309327
All Children	Atlanta	S75	2016	40	460429	344999	283178	243773	215123	191617
All Children	Atlanta	S75	2016	50	299642	189660	136837	101831	78265	60913
All Children	Atlanta	S75	2016	60	129312	48424	22053	10431	4984	2603
All Children	Atlanta	S75	2016	70	33957	4257	666	182	20	0
All Children	Atlanta	S75	2016	80	6658	121	0	0	0	0
All Children	Atlanta	S75	2016	90	1069	0	0	0	0	0
All Children	Atlanta	S75	2016	100	161	0	0	0	0	0
All Children	Atlanta	S75	2017	0	787491	708984	655456	615486	582962	554835
All Children	Atlanta	S75	2017	10	749862	663668	606407	563350	529070	498906
All Children	Atlanta	S75	2017	20	666311	563209	499612	453852	419027	388580

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Atlanta	S75	2017	30	553786	439062	374881	331198	297625	270003
All Children	Atlanta	S75	2017	40	408475	292762	233120	194845	167001	144848
All Children	Atlanta	S75	2017	50	222527	120656	76106	51168	34885	24373
All Children	Atlanta	S75	2017	60	57846	12832	3793	1211	424	222
All Children	Atlanta	S75	2017	70	5387	262	20	0	0	0
All Children	Atlanta	S75	2017	80	404	0	0	0	0	0
All Children	Atlanta	S75	2017	90	0	0	0	0	0	0
All Children	Atlanta	S75	2017	100	0	0	0	0	0	0
All Children	Boston	S65	2015	0	862212	765847	702953	654281	613301	578919
All Children	Boston	S65	2015	10	828103	724661	659401	605997	564037	530315
All Children	Boston	S65	2015	20	719792	597213	524695	471381	429331	395632
All Children	Boston	S65	2015	30	564197	430947	358701	309347	271005	242494
All Children	Boston	S65	2015	40	352717	224814	164424	124171	95705	74976
All Children	Boston	S65	2015	50	124717	41686	16383	6667	2822	1456
All Children	Boston	S65	2015	60	22754	1456	114	0	0	0
All Children	Boston	S65	2015	70	3095	0	0	0	0	0
All Children	Boston	S65	2015	80	114	0	0	0	0	0
All Children	Boston	S65	2015	90	0	0	0	0	0	0
All Children	Boston	S65	2015	100	0	0	0	0	0	0
All Children	Boston	S65	2016	0	865716	770989	706503	658309	617829	583447
All Children	Boston	S65	2016	10	832676	731123	663975	612618	571000	535344
All Children	Boston	S65	2016	20	725617	603038	527539	472769	430036	396747
All Children	Boston	S65	2016	30	564310	430924	357654	309278	273554	244315
All Children	Boston	S65	2016	40	359884	232050	168383	128995	100393	79754
All Children	Boston	S65	2016	50	137300	47739	19887	8988	3572	1502
All Children	Boston	S65	2016	60	15314	1160	114	0	0	0
All Children	Boston	S65	2016	70	1251	0	0	0	0	0
All Children	Boston	S65	2016	80	0	0	0	0	0	0
All Children	Boston	S65	2016	90	0	0	0	0	0	0
All Children	Boston	S65	2016	100	0	0	0	0	0	0
All Children	Boston	S65	2017	0	862462	763776	699927	649867	610434	576484
All Children	Boston	S65	2017	10	825941	722226	656693	605155	564288	526902
All Children	Boston	S65	2017	20	718654	597805	519871	464532	421185	386393
All Children	Boston	S65	2017	30	563013	424871	353672	302247	265089	235008
All Children	Boston	S65	2017	40	377155	243518	175369	131475	100142	77706
All Children	Boston	S65	2017	50	173912	69924	29968	12925	5006	2162
All Children	Boston	S65	2017	60	33631	4323	432	46	0	0
All Children	Boston	S65	2017	70	3140	23	0	0	0	0
All Children	Boston	S65	2017	80	0	0	0	0	0	0
All Children	Boston	S65	2017	90	0	0	0	0	0	0
All Children	Boston	S65	2017	100	0	0	0	0	0	0
All Children	Boston	S70	2015	0	862212	765847	702953	654281	613301	578919
All Children	Boston	S70	2015	10	828717	725776	660493	606952	565312	532022
All Children	Boston	S70	2015	20	727164	605655	532318	479391	437682	402185
All Children	Boston	S70	2015	30	585199	452449	378338	328256	289505	259810
All Children	Boston	S70	2015	40	396041	265772	201468	157802	127061	104761
All Children	Boston	S70	2015	50	184288	81643	41686	21867	12287	7031

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Boston	S70	2015	60	46465	6690	1479	410	114	46
All Children	Boston	S70	2015	70	7554	91	0	0	0	0
All Children	Boston	S70	2015	80	592	0	0	0	0	0
All Children	Boston	S70	2015	90	0	0	0	0	0	0
All Children	Boston	S70	2015	100	0	0	0	0	0	0
All Children	Boston	S70	2016	0	865716	770989	706503	658309	617829	583447
All Children	Boston	S70	2016	10	833541	732397	665204	614120	571842	536391
All Children	Boston	S70	2016	20	735037	612709	537597	482440	438205	405166
All Children	Boston	S70	2016	30	587497	453519	380659	330122	292144	262336
All Children	Boston	S70	2016	40	409466	275238	209227	166836	134365	111315
All Children	Boston	S70	2016	50	208021	97139	52836	30969	18067	10672
All Children	Boston	S70	2016	60	50242	8783	1661	137	46	23
All Children	Boston	S70	2016	70	5438	273	0	0	0	0
All Children	Boston	S70	2016	80	91	0	0	0	0	0
All Children	Boston	S70	2016	90	0	0	0	0	0	0
All Children	Boston	S70	2016	100	0	0	0	0	0	0
All Children	Boston	S70	2017	0	862462	763776	699927	649867	610434	576484
All Children	Boston	S70	2017	10	826737	723250	657877	605951	564652	527835
All Children	Boston	S70	2017	20	726527	605655	528700	473475	429217	393493
All Children	Boston	S70	2017	30	585313	446647	372081	321020	282178	251300
All Children	Boston	S70	2017	40	418909	284977	213687	168428	134274	108038
All Children	Boston	S70	2017	50	238512	116890	63735	35497	20320	10922
All Children	Boston	S70	2017	60	81939	18477	4369	865	68	23
All Children	Boston	S70	2017	70	11923	660	23	0	0	0
All Children	Boston	S70	2017	80	432	0	0	0	0	0
All Children	Boston	S70	2017	90	0	0	0	0	0	0
All Children	Boston	S70	2017	100	0	0	0	0	0	0
All Children	Boston	S75	2015	0	862212	765847	702953	654281	613301	578919
All Children	Boston	S75	2015	10	828581	725457	659970	606952	565107	531203
All Children	Boston	S75	2015	20	729826	609137	535981	482758	440799	404847
All Children	Boston	S75	2015	30	594164	461028	387895	336902	297446	267524
All Children	Boston	S75	2015	40	417408	285637	219558	174731	143012	120235
All Children	Boston	S75	2015	50	218625	107765	60845	34974	21730	12970
All Children	Boston	S75	2015	60	68559	14677	3823	1069	296	91
All Children	Boston	S75	2015	70	12788	341	0	0	0	0
All Children	Boston	S75	2015	80	1047	0	0	0	0	0
All Children	Boston	S75	2015	90	23	0	0	0	0	0
All Children	Boston	S75	2015	100	0	0	0	0	0	0
All Children	Boston	S75	2016	0	865716	770989	706503	658309	617829	583447
All Children	Boston	S75	2016	10	833905	732921	665295	614165	571933	536027
All Children	Boston	S75	2016	20	737950	616577	541215	486103	441982	407760
All Children	Boston	S75	2016	30	598032	463827	389966	338950	301337	270255
All Children	Boston	S75	2016	40	434087	297651	229729	184379	152000	126583
All Children	Boston	S75	2016	50	244815	127971	76705	48649	31629	20684
All Children	Boston	S75	2016	60	82553	20957	5643	1479	387	137
All Children	Boston	S75	2016	70	12356	819	0	0	0	0
All Children	Boston	S75	2016	80	865	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Boston	S75	2016	90	0	0	0	0	0	0
All Children	Boston	S75	2016	100	0	0	0	0	0	0
All Children	Boston	S75	2017	0	862462	763776	699927	649867	610434	576484
All Children	Boston	S75	2017	10	826806	723091	658263	605701	564447	527357
All Children	Boston	S75	2017	20	729462	609432	531726	475659	431902	395450
All Children	Boston	S75	2017	30	596212	457569	381296	329712	289983	258604
All Children	Boston	S75	2017	40	440594	303226	233188	184425	150452	124285
All Children	Boston	S75	2017	50	275738	145651	85557	52267	32425	20206
All Children	Boston	S75	2017	60	119711	35724	11400	2890	887	182
All Children	Boston	S75	2017	70	26532	3322	455	23	0	0
All Children	Boston	S75	2017	80	1957	23	0	0	0	0
All Children	Boston	S75	2017	90	0	0	0	0	0	0
All Children	Boston	S75	2017	100	0	0	0	0	0	0
All Children	Dallas	S65	2015	0	931702	848139	790894	748592	711327	679098
All Children	Dallas	S65	2015	10	886185	790965	727382	681320	641596	608800
All Children	Dallas	S65	2015	20	783658	667299	593194	540819	500811	466643
All Children	Dallas	S65	2015	30	636205	503034	429047	377902	341606	310985
All Children	Dallas	S65	2015	40	459431	325149	255276	210066	176632	151780
All Children	Dallas	S65	2015	50	231229	118748	69021	44146	28635	19366
All Children	Dallas	S65	2015	60	39772	7188	1537	213	24	24
All Children	Dallas	S65	2015	70	1017	0	0	0	0	0
All Children	Dallas	S65	2015	80	0	0	0	0	0	0
All Children	Dallas	S65	2015	90	0	0	0	0	0	0
All Children	Dallas	S65	2015	100	0	0	0	0	0	0
All Children	Dallas	S65	2016	0	933499	848896	794440	751618	716860	687137
All Children	Dallas	S65	2016	10	888573	794582	736249	689313	651291	619157
All Children	Dallas	S65	2016	20	783895	670018	601091	547203	505729	473737
All Children	Dallas	S65	2016	30	627078	496176	424507	375443	338036	310016
All Children	Dallas	S65	2016	40	418005	287411	220423	177081	145372	120497
All Children	Dallas	S65	2016	50	152608	62873	29486	16292	7992	4635
All Children	Dallas	S65	2016	60	12343	307	47	24	0	0
All Children	Dallas	S65	2016	70	946	0	0	0	0	0
All Children	Dallas	S65	2016	80	0	0	0	0	0	0
All Children	Dallas	S65	2016	90	0	0	0	0	0	0
All Children	Dallas	S65	2016	100	0	0	0	0	0	0
All Children	Dallas	S65	2017	0	938796	854973	800967	757435	720619	688911
All Children	Dallas	S65	2017	10	899119	806003	744430	698038	658219	624146
All Children	Dallas	S65	2017	20	798578	682053	612749	560587	518214	484850
All Children	Dallas	S65	2017	30	647318	516937	444204	394312	357307	328081
All Children	Dallas	S65	2017	40	455577	319970	251659	205101	171737	144072
All Children	Dallas	S65	2017	50	218200	102243	54645	31094	18278	11208
All Children	Dallas	S65	2017	60	36343	3547	497	24	0	0
All Children	Dallas	S65	2017	70	922	0	0	0	0	0
All Children	Dallas	S65	2017	80	0	0	0	0	0	0
All Children	Dallas	S65	2017	90	0	0	0	0	0	0
All Children	Dallas	S65	2017	100	0	0	0	0	0	0
All Children	Dallas	S70	2015	0	931702	848139	790894	748592	711327	679098

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Dallas	S70	2015	10	887982	792738	729415	683567	643653	611708
All Children	Dallas	S70	2015	20	793802	677797	603290	551578	510955	477284
All Children	Dallas	S70	2015	30	657793	526466	449311	398261	360357	329169
All Children	Dallas	S70	2015	40	499298	363786	292211	244069	209664	181810
All Children	Dallas	S70	2015	50	304601	181905	122294	87063	63748	47386
All Children	Dallas	S70	2015	60	96261	29273	10759	4422	1773	780
All Children	Dallas	S70	2015	70	9718	757	24	0	0	0
All Children	Dallas	S70	2015	80	236	0	0	0	0	0
All Children	Dallas	S70	2015	90	0	0	0	0	0	0
All Children	Dallas	S70	2015	100	0	0	0	0	0	0
All Children	Dallas	S70	2016	0	933499	848896	794440	751618	716860	687137
All Children	Dallas	S70	2016	10	890677	797491	738992	692008	654436	622774
All Children	Dallas	S70	2016	20	792218	680233	611377	557111	515755	483219
All Children	Dallas	S70	2016	30	648784	518356	444606	395872	358158	328294
All Children	Dallas	S70	2016	40	459006	327111	259414	214512	180108	154405
All Children	Dallas	S70	2016	50	218271	109455	63086	39062	23929	15157
All Children	Dallas	S70	2016	60	34499	5226	1301	260	71	47
All Children	Dallas	S70	2016	70	3168	24	0	0	0	0
All Children	Dallas	S70	2016	80	284	0	0	0	0	0
All Children	Dallas	S70	2016	90	0	0	0	0	0	0
All Children	Dallas	S70	2016	100	0	0	0	0	0	0
All Children	Dallas	S70	2017	0	938796	854973	800967	757435	720619	688911
All Children	Dallas	S70	2017	10	901176	808509	747575	701017	660796	627007
All Children	Dallas	S70	2017	20	807256	691914	622893	571369	528996	494900
All Children	Dallas	S70	2017	30	668552	537958	465106	414576	377145	346217
All Children	Dallas	S70	2017	40	494308	359222	288215	242106	207323	177838
All Children	Dallas	S70	2017	50	279820	153388	95693	62471	40859	28185
All Children	Dallas	S70	2017	60	78621	16907	4469	1277	402	95
All Children	Dallas	S70	2017	70	4705	47	0	0	0	0
All Children	Dallas	S70	2017	80	0	0	0	0	0	0
All Children	Dallas	S70	2017	90	0	0	0	0	0	0
All Children	Dallas	S70	2017	100	0	0	0	0	0	0
All Children	Dallas	S75	2015	0	931702	848139	790894	748592	711327	679098
All Children	Dallas	S75	2015	10	888668	794204	730645	684867	644859	612559
All Children	Dallas	S75	2015	20	799950	685009	610975	559002	517670	483904
All Children	Dallas	S75	2015	30	674652	543042	466099	412732	374260	343048
All Children	Dallas	S75	2015	40	528287	393106	319687	269700	233121	204250
All Children	Dallas	S75	2015	50	362036	231938	164691	123760	95859	75405
All Children	Dallas	S75	2015	60	162894	67602	32725	16670	9293	4942
All Children	Dallas	S75	2015	70	29912	4280	828	95	24	0
All Children	Dallas	S75	2015	80	2081	24	0	0	0	0
All Children	Dallas	S75	2015	90	47	0	0	0	0	0
All Children	Dallas	S75	2015	100	0	0	0	0	0	0
All Children	Dallas	S75	2016	0	933499	848896	794440	751618	716860	687137
All Children	Dallas	S75	2016	10	891623	799217	740623	693663	656138	624311
All Children	Dallas	S75	2016	20	798129	686570	618211	563873	522518	489579
All Children	Dallas	S75	2016	30	664745	534600	459692	409871	370808	341890

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Dallas	S75	2016	40	489508	355391	285992	241160	206283	178736
All Children	Dallas	S75	2016	50	273507	151662	96852	65734	45399	32347
All Children	Dallas	S75	2016	60	66964	15819	5131	1726	520	189
All Children	Dallas	S75	2016	70	9860	166	24	24	0	0
All Children	Dallas	S75	2016	80	1419	0	0	0	0	0
All Children	Dallas	S75	2016	90	71	0	0	0	0	0
All Children	Dallas	S75	2016	100	0	0	0	0	0	0
All Children	Dallas	S75	2017	0	938796	854973	800967	757435	720619	688911
All Children	Dallas	S75	2017	10	902051	809810	749372	702791	662901	629040
All Children	Dallas	S75	2017	20	813262	699267	631098	577730	535806	502064
All Children	Dallas	S75	2017	30	683118	554250	479837	427557	390008	359151
All Children	Dallas	S75	2017	40	522257	387313	316022	267714	232411	202500
All Children	Dallas	S75	2017	50	328932	197936	133644	94251	67768	49017
All Children	Dallas	S75	2017	60	125037	38755	13998	5344	2317	851
All Children	Dallas	S75	2017	70	16126	804	71	0	0	0
All Children	Dallas	S75	2017	80	71	0	0	0	0	0
All Children	Dallas	S75	2017	90	0	0	0	0	0	0
All Children	Dallas	S75	2017	100	0	0	0	0	0	0
All Children	Detroit	S65	2015	0	658727	585868	537967	501043	471369	444747
All Children	Detroit	S65	2015	10	631377	552916	501598	463617	433093	405847
All Children	Detroit	S65	2015	20	556680	464033	407303	366963	334324	307407
All Children	Detroit	S65	2015	30	448043	347730	291521	253903	225322	202412
All Children	Detroit	S65	2015	40	314483	214118	161863	128912	106001	87409
All Children	Detroit	S65	2015	50	142110	62349	31096	16129	9261	5064
All Children	Detroit	S65	2015	60	14932	1179	121	17	0	0
All Children	Detroit	S65	2015	70	87	0	0	0	0	0
All Children	Detroit	S65	2015	80	0	0	0	0	0	0
All Children	Detroit	S65	2015	90	0	0	0	0	0	0
All Children	Detroit	S65	2015	100	0	0	0	0	0	0
All Children	Detroit	S65	2016	0	666184	595199	547679	510322	479989	454772
All Children	Detroit	S65	2016	10	639771	562646	512264	474179	442545	416842
All Children	Detroit	S65	2016	20	566774	476190	421542	379832	347903	320883
All Children	Detroit	S65	2016	30	464831	363269	306783	267657	238139	215645
All Children	Detroit	S65	2016	40	336769	231982	177941	145318	120205	100521
All Children	Detroit	S65	2016	50	178287	88571	50625	28321	16580	9903
All Children	Detroit	S65	2016	60	38276	5498	884	87	17	0
All Children	Detroit	S65	2016	70	815	17	0	0	0	0
All Children	Detroit	S65	2016	80	0	0	0	0	0	0
All Children	Detroit	S65	2016	90	0	0	0	0	0	0
All Children	Detroit	S65	2016	100	0	0	0	0	0	0
All Children	Detroit	S65	2017	0	661623	588886	542771	505899	475913	448996
All Children	Detroit	S65	2017	10	635990	558553	509212	469409	439267	411813
All Children	Detroit	S65	2017	20	565681	473381	417328	376519	344695	316963
All Children	Detroit	S65	2017	30	458899	357563	301129	261274	232675	209019
All Children	Detroit	S65	2017	40	326727	225721	174316	140133	116008	96948
All Children	Detroit	S65	2017	50	159730	72980	40427	22199	12695	7770
All Children	Detroit	S65	2017	60	17725	1353	139	35	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Detroit	S65	2017	70	330	0	0	0	0	0
All Children	Detroit	S65	2017	80	0	0	0	0	0	0
All Children	Detroit	S65	2017	90	0	0	0	0	0	0
All Children	Detroit	S65	2017	100	0	0	0	0	0	0
All Children	Detroit	S70	2015	0	658727	585868	537967	501043	471369	444747
All Children	Detroit	S70	2015	10	632365	553732	502535	464501	433630	406766
All Children	Detroit	S70	2015	20	562663	471733	415056	374352	341677	314708
All Children	Detroit	S70	2015	30	464952	364032	306557	269044	239890	215835
All Children	Detroit	S70	2015	40	348025	246394	192491	158048	132675	112609
All Children	Detroit	S70	2015	50	197330	106868	65470	41398	26882	17499
All Children	Detroit	S70	2015	60	52203	10805	2549	746	191	17
All Children	Detroit	S70	2015	70	1492	69	17	0	0	0
All Children	Detroit	S70	2015	80	0	0	0	0	0	0
All Children	Detroit	S70	2015	90	0	0	0	0	0	0
All Children	Detroit	S70	2015	100	0	0	0	0	0	0
All Children	Detroit	S70	2016	0	666184	595199	547679	510322	479989	454772
All Children	Detroit	S70	2016	10	640326	564016	513287	475132	443204	417831
All Children	Detroit	S70	2016	20	573121	483076	429069	386908	354823	327855
All Children	Detroit	S70	2016	30	481740	380855	323623	283994	253244	228825
All Children	Detroit	S70	2016	40	369669	264361	208569	172027	146203	125478
All Children	Detroit	S70	2016	50	235329	136820	90965	62626	43653	30697
All Children	Detroit	S70	2016	60	95509	28894	9764	3035	1041	399
All Children	Detroit	S70	2016	70	9487	520	0	0	0	0
All Children	Detroit	S70	2016	80	52	0	0	0	0	0
All Children	Detroit	S70	2016	90	0	0	0	0	0	0
All Children	Detroit	S70	2016	100	0	0	0	0	0	0
All Children	Detroit	S70	2017	0	661623	588886	542771	505899	475913	448996
All Children	Detroit	S70	2017	10	636528	559455	510460	470120	440203	412610
All Children	Detroit	S70	2017	20	572202	479763	423866	383092	351372	323467
All Children	Detroit	S70	2017	30	475115	374508	316980	277005	246619	223015
All Children	Detroit	S70	2017	40	359315	255586	202498	166078	140705	120517
All Children	Detroit	S70	2017	50	221142	123379	79327	52602	35710	23934
All Children	Detroit	S70	2017	60	61169	13788	3885	1214	451	156
All Children	Detroit	S70	2017	70	4301	139	0	0	0	0
All Children	Detroit	S70	2017	80	35	0	0	0	0	0
All Children	Detroit	S70	2017	90	0	0	0	0	0	0
All Children	Detroit	S70	2017	100	0	0	0	0	0	0
All Children	Detroit	S75	2015	0	658727	585868	537967	501043	471369	444747
All Children	Detroit	S75	2015	10	631446	552691	501182	463409	432347	405430
All Children	Detroit	S75	2015	20	564398	473589	417102	376173	343221	315784
All Children	Detroit	S75	2015	30	473277	372357	314154	274733	245596	222148
All Children	Detroit	S75	2015	40	366634	263165	208204	172269	146047	125998
All Children	Detroit	S75	2015	50	234982	137375	91242	63424	44329	31738
All Children	Detroit	S75	2015	60	89387	26223	8776	3018	1041	572
All Children	Detroit	S75	2015	70	9296	416	52	0	0	0
All Children	Detroit	S75	2015	80	69	0	0	0	0	0
All Children	Detroit	S75	2015	90	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Detroit	S75	2015	100	0	0	0	0	0	0
All Children	Detroit	S75	2016	0	666184	595199	547679	510322	479989	454772
All Children	Detroit	S75	2016	10	639615	563132	512056	473970	442059	416513
All Children	Detroit	S75	2016	20	575254	485244	430665	388035	355812	328756
All Children	Detroit	S75	2016	30	489926	389666	332034	291174	260667	234965
All Children	Detroit	S75	2016	40	387931	282450	226328	188052	160615	139526
All Children	Detroit	S75	2016	50	271472	167587	118610	86698	65436	49879
All Children	Detroit	S75	2016	60	144191	60701	28061	12921	5983	2792
All Children	Detroit	S75	2016	70	34981	4561	607	69	0	0
All Children	Detroit	S75	2016	80	1249	17	0	0	0	0
All Children	Detroit	S75	2016	90	0	0	0	0	0	0
All Children	Detroit	S75	2016	100	0	0	0	0	0	0
All Children	Detroit	S75	2017	0	661623	588886	542771	505899	475913	448996
All Children	Detroit	S75	2017	10	635452	558501	509160	468629	438036	410859
All Children	Detroit	S75	2017	20	573607	481029	425965	384601	352482	324178
All Children	Detroit	S75	2017	30	483544	381740	323935	282971	252655	229450
All Children	Detroit	S75	2017	40	376519	271472	216928	179536	153695	132606
All Children	Detroit	S75	2017	50	253262	154180	105880	74298	53070	39248
All Children	Detroit	S75	2017	60	109747	38432	14950	6157	2549	989
All Children	Detroit	S75	2017	70	15106	694	35	0	0	0
All Children	Detroit	S75	2017	80	746	0	0	0	0	0
All Children	Detroit	S75	2017	90	0	0	0	0	0	0
All Children	Detroit	S75	2017	100	0	0	0	0	0	0
All Children	Philadelphia	S65	2015	0	844309	758097	699407	656192	618084	586655
All Children	Philadelphia	S65	2015	10	815062	724070	661496	615312	577248	545077
All Children	Philadelphia	S65	2015	20	730465	621184	550272	501426	463187	430295
All Children	Philadelphia	S65	2015	30	600755	476806	407466	359296	323393	294801
All Children	Philadelphia	S65	2015	40	413773	289279	226901	184974	153894	130169
All Children	Philadelphia	S65	2015	50	163999	72811	37955	21040	12419	7268
All Children	Philadelphia	S65	2015	60	20975	2204	437	44	0	0
All Children	Philadelphia	S65	2015	70	393	0	0	0	0	0
All Children	Philadelphia	S65	2015	80	0	0	0	0	0	0
All Children	Philadelphia	S65	2015	90	0	0	0	0	0	0
All Children	Philadelphia	S65	2015	100	0	0	0	0	0	0
All Children	Philadelphia	S65	2016	0	846469	759581	703030	656803	619197	588532
All Children	Philadelphia	S65	2016	10	817310	724070	664639	616928	580064	548831
All Children	Philadelphia	S65	2016	20	729047	618826	551276	500487	459957	429095
All Children	Philadelphia	S65	2016	30	593814	471022	401660	353730	317609	287009
All Children	Philadelphia	S65	2016	40	401660	274416	211776	169674	139489	116200
All Children	Philadelphia	S65	2016	50	155531	63644	30556	14754	7508	3623
All Children	Philadelphia	S65	2016	60	21171	1702	109	0	0	0
All Children	Philadelphia	S65	2016	70	175	0	0	0	0	0
All Children	Philadelphia	S65	2016	80	0	0	0	0	0	0
All Children	Philadelphia	S65	2016	90	0	0	0	0	0	0
All Children	Philadelphia	S65	2016	100	0	0	0	0	0	0
All Children	Philadelphia	S65	2017	0	843414	754867	696046	650277	613959	581897
All Children	Philadelphia	S65	2017	10	813403	717894	655690	609703	572185	540101

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Philadelphia	S65	2017	20	721168	607914	538857	488286	449153	417418
All Children	Philadelphia	S65	2017	30	575524	450462	381231	332298	296896	268042
All Children	Philadelphia	S65	2017	40	380860	254227	193049	151602	122268	99875
All Children	Philadelphia	S65	2017	50	139991	52447	23550	10651	5042	2335
All Children	Philadelphia	S65	2017	60	18203	1484	109	22	0	0
All Children	Philadelphia	S65	2017	70	480	0	0	0	0	0
All Children	Philadelphia	S65	2017	80	0	0	0	0	0	0
All Children	Philadelphia	S65	2017	90	0	0	0	0	0	0
All Children	Philadelphia	S65	2017	100	0	0	0	0	0	0
All Children	Philadelphia	S70	2015	0	844309	758097	699407	656192	618084	586655
All Children	Philadelphia	S70	2015	10	816219	725838	663744	617080	578973	547085
All Children	Philadelphia	S70	2015	20	738344	629521	559853	509807	471240	439047
All Children	Philadelphia	S70	2015	30	621140	497715	427087	378590	342272	312021
All Children	Philadelphia	S70	2015	40	460066	332145	266624	223933	192023	165156
All Children	Philadelphia	S70	2015	50	238250	129711	80493	52426	35947	25165
All Children	Philadelphia	S70	2015	60	54674	11764	3405	1157	349	65
All Children	Philadelphia	S70	2015	70	4627	131	0	0	0	0
All Children	Philadelphia	S70	2015	80	44	0	0	0	0	0
All Children	Philadelphia	S70	2015	90	0	0	0	0	0	0
All Children	Philadelphia	S70	2015	100	0	0	0	0	0	0
All Children	Philadelphia	S70	2016	0	846469	759581	703030	656803	619197	588532
All Children	Philadelphia	S70	2016	10	818467	725860	666472	618717	581286	550795
All Children	Philadelphia	S70	2016	20	736598	627993	560268	509698	469429	437323
All Children	Philadelphia	S70	2016	30	614963	490753	422089	372719	336204	305888
All Children	Philadelphia	S70	2016	40	448913	319726	253114	209986	177160	151296
All Children	Philadelphia	S70	2016	50	229476	119234	69777	44743	28068	18115
All Children	Philadelphia	S70	2016	60	53560	9210	1899	480	44	22
All Children	Philadelphia	S70	2016	70	5151	65	0	0	0	0
All Children	Philadelphia	S70	2016	80	0	0	0	0	0	0
All Children	Philadelphia	S70	2016	90	0	0	0	0	0	0
All Children	Philadelphia	S70	2016	100	0	0	0	0	0	0
All Children	Philadelphia	S70	2017	0	843414	754867	696046	650277	613959	581897
All Children	Philadelphia	S70	2017	10	814844	719487	657436	611187	573996	542196
All Children	Philadelphia	S70	2017	20	728807	616753	547303	497584	458232	425974
All Children	Philadelphia	S70	2017	30	597263	471153	401594	352203	314640	285001
All Children	Philadelphia	S70	2017	40	426148	296481	233143	188531	156796	132133
All Children	Philadelphia	S70	2017	50	205599	99984	55787	31080	18705	11589
All Children	Philadelphia	S70	2017	60	51116	9451	2073	349	87	0
All Children	Philadelphia	S70	2017	70	4191	175	0	0	0	0
All Children	Philadelphia	S70	2017	80	87	0	0	0	0	0
All Children	Philadelphia	S70	2017	90	0	0	0	0	0	0
All Children	Philadelphia	S70	2017	100	0	0	0	0	0	0
All Children	Philadelphia	S75	2015	0	844309	758097	699407	656192	618084	586655
All Children	Philadelphia	S75	2015	10	816895	726187	664508	617932	579431	547740
All Children	Philadelphia	S75	2015	20	745307	637357	568758	518013	478247	446403
All Children	Philadelphia	S75	2015	30	640718	517162	446206	396422	359711	328784
All Children	Philadelphia	S75	2015	40	503608	373701	306281	261167	226835	199815

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Philadelphia	S75	2015	50	316932	197130	138681	102494	78267	60239
All Children	Philadelphia	S75	2015	60	113669	38850	16784	8185	3536	1550
All Children	Philadelphia	S75	2015	70	20036	1964	393	87	0	0
All Children	Philadelphia	S75	2015	80	1550	22	0	0	0	0
All Children	Philadelphia	S75	2015	90	22	0	0	0	0	0
All Children	Philadelphia	S75	2015	100	0	0	0	0	0	0
All Children	Philadelphia	S75	2016	0	846469	759581	703030	656803	619197	588532
All Children	Philadelphia	S75	2016	10	819165	726668	666821	620201	581963	551101
All Children	Philadelphia	S75	2016	20	742906	637357	568496	518100	477985	445028
All Children	Philadelphia	S75	2016	30	635960	512098	442671	391424	354189	323305
All Children	Philadelphia	S75	2016	40	491582	362504	293993	246675	212605	186654
All Children	Philadelphia	S75	2016	50	310210	189142	129994	94506	69100	52076
All Children	Philadelphia	S75	2016	60	115153	36493	13139	4845	1986	829
All Children	Philadelphia	S75	2016	70	18377	1113	87	0	0	0
All Children	Philadelphia	S75	2016	80	240	0	0	0	0	0
All Children	Philadelphia	S75	2016	90	0	0	0	0	0	0
All Children	Philadelphia	S75	2016	100	0	0	0	0	0	0
All Children	Philadelphia	S75	2017	0	843414	754867	696046	650277	613959	581897
All Children	Philadelphia	S75	2017	10	815477	720513	658811	612170	574171	542982
All Children	Philadelphia	S75	2017	20	736511	624305	554484	505180	465871	433176
All Children	Philadelphia	S75	2017	30	617211	492259	421696	371388	333476	302069
All Children	Philadelphia	S75	2017	40	467094	337296	271076	225482	191303	164588
All Children	Philadelphia	S75	2017	50	281509	162624	105091	70846	48104	33830
All Children	Philadelphia	S75	2017	60	107950	33306	11677	4060	1353	371
All Children	Philadelphia	S75	2017	70	16173	1310	109	0	0	0
All Children	Philadelphia	S75	2017	80	633	0	0	0	0	0
All Children	Philadelphia	S75	2017	90	0	0	0	0	0	0
All Children	Philadelphia	S75	2017	100	0	0	0	0	0	0
All Children	Phoenix	S65	2015	0	573408	527665	497306	475283	455327	438640
All Children	Phoenix	S65	2015	10	554797	505302	472721	448448	427020	409314
All Children	Phoenix	S65	2015	20	507326	445844	408479	379182	355687	336241
All Children	Phoenix	S65	2015	30	435880	362750	321153	291530	268772	249707
All Children	Phoenix	S65	2015	40	332632	254859	213489	185041	164306	147209
All Children	Phoenix	S65	2015	50	173265	100206	66323	46720	33572	25094
All Children	Phoenix	S65	2015	60	11323	2066	510	142	71	14
All Children	Phoenix	S65	2015	70	0	0	0	0	0	0
All Children	Phoenix	S65	2015	80	0	0	0	0	0	0
All Children	Phoenix	S65	2015	90	0	0	0	0	0	0
All Children	Phoenix	S65	2015	100	0	0	0	0	0	0
All Children	Phoenix	S65	2016	0	573705	529561	500023	476840	457549	440310
All Children	Phoenix	S65	2016	10	555165	506718	475722	451067	429483	411494
All Children	Phoenix	S65	2016	20	508459	447599	410036	382565	359905	340812
All Children	Phoenix	S65	2016	30	435611	365510	323602	294276	271418	253543
All Children	Phoenix	S65	2016	40	327140	251561	210064	182904	161574	145383
All Children	Phoenix	S65	2016	50	145100	77178	47230	30996	21683	15526
All Children	Phoenix	S65	2016	60	7982	722	142	42	42	28
All Children	Phoenix	S65	2016	70	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Phoenix	S65	2016	80	0	0	0	0	0	0
All Children	Phoenix	S65	2016	90	0	0	0	0	0	0
All Children	Phoenix	S65	2016	100	0	0	0	0	0	0
All Children	Phoenix	S65	2017	0	575177	529830	499174	476614	457889	440961
All Children	Phoenix	S65	2017	10	557655	508204	475637	451548	432045	414169
All Children	Phoenix	S65	2017	20	512337	452510	414792	387080	363727	343572
All Children	Phoenix	S65	2017	30	443282	373436	332504	303263	280420	261327
All Children	Phoenix	S65	2017	40	347408	270767	230416	202747	180993	164080
All Children	Phoenix	S65	2017	50	187234	113524	80164	59147	44456	34378
All Children	Phoenix	S65	2017	60	25334	4388	1076	354	71	14
All Children	Phoenix	S65	2017	70	0	0	0	0	0	0
All Children	Phoenix	S65	2017	80	0	0	0	0	0	0
All Children	Phoenix	S65	2017	90	0	0	0	0	0	0
All Children	Phoenix	S65	2017	100	0	0	0	0	0	0
All Children	Phoenix	S70	2015	0	573408	527665	497306	475283	455327	438640
All Children	Phoenix	S70	2015	10	555674	506336	473953	449439	428379	410532
All Children	Phoenix	S70	2015	20	511742	452100	414608	385636	362071	342256
All Children	Phoenix	S70	2015	30	449213	376988	334967	305627	282005	262318
All Children	Phoenix	S70	2015	40	361405	283817	240508	212003	190192	173803
All Children	Phoenix	S70	2015	50	236842	159409	121322	95521	77334	63633
All Children	Phoenix	S70	2015	60	68177	24061	10912	5803	3284	1713
All Children	Phoenix	S70	2015	70	807	0	0	0	0	0
All Children	Phoenix	S70	2015	80	0	0	0	0	0	0
All Children	Phoenix	S70	2015	90	0	0	0	0	0	0
All Children	Phoenix	S70	2015	100	0	0	0	0	0	0
All Children	Phoenix	S70	2016	0	573705	529561	500023	476840	457549	440310
All Children	Phoenix	S70	2016	10	556084	507807	476911	452737	430714	412626
All Children	Phoenix	S70	2016	20	512988	453869	416518	388339	366189	347903
All Children	Phoenix	S70	2016	30	448279	379621	337543	308231	284595	265955
All Children	Phoenix	S70	2016	40	357598	281694	239913	212144	190277	173067
All Children	Phoenix	S70	2016	50	218768	142326	103645	79598	62388	49961
All Children	Phoenix	S70	2016	60	50754	14153	5576	2633	1189	594
All Children	Phoenix	S70	2016	70	269	14	0	0	0	0
All Children	Phoenix	S70	2016	80	0	0	0	0	0	0
All Children	Phoenix	S70	2016	90	0	0	0	0	0	0
All Children	Phoenix	S70	2016	100	0	0	0	0	0	0
All Children	Phoenix	S70	2017	0	575177	529830	499174	476614	457889	440961
All Children	Phoenix	S70	2017	10	558575	509647	476911	452963	433616	415655
All Children	Phoenix	S70	2017	20	517517	458568	421076	393434	371256	350437
All Children	Phoenix	S70	2017	30	456587	386556	346176	316992	293823	274858
All Children	Phoenix	S70	2017	40	375955	298579	258256	230586	208026	190546
All Children	Phoenix	S70	2017	50	254335	175954	136834	111104	93058	78240
All Children	Phoenix	S70	2017	60	89775	36643	18074	9554	5392	3326
All Children	Phoenix	S70	2017	70	4784	269	42	14	0	0
All Children	Phoenix	S70	2017	80	0	0	0	0	0	0
All Children	Phoenix	S70	2017	90	0	0	0	0	0	0
All Children	Phoenix	S70	2017	100	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Phoenix	S75	2015	0	573408	527665	497306	475283	455327	438640
All Children	Phoenix	S75	2015	10	555575	506251	473372	449198	428167	410065
All Children	Phoenix	S75	2015	20	513992	455016	417170	388849	365198	344931
All Children	Phoenix	S75	2015	30	456728	385763	343332	313567	289308	270527
All Children	Phoenix	S75	2015	40	379408	301664	258553	229539	207007	189471
All Children	Phoenix	S75	2015	50	276655	198147	157470	130678	110382	94134
All Children	Phoenix	S75	2015	60	136778	69054	41186	25844	16899	11535
All Children	Phoenix	S75	2015	70	13191	1613	226	28	0	0
All Children	Phoenix	S75	2015	80	198	0	0	0	0	0
All Children	Phoenix	S75	2015	90	0	0	0	0	0	0
All Children	Phoenix	S75	2015	100	0	0	0	0	0	0
All Children	Phoenix	S75	2016	0	573705	529561	500023	476840	457549	440310
All Children	Phoenix	S75	2016	10	555830	507850	476882	452241	430403	412173
All Children	Phoenix	S75	2016	20	515309	456742	419590	391241	368765	350026
All Children	Phoenix	S75	2016	30	456303	387915	346219	316171	292153	273230
All Children	Phoenix	S75	2016	40	376564	301268	258058	229256	207134	189584
All Children	Phoenix	S75	2016	50	264469	186329	145666	117175	97856	82726
All Children	Phoenix	S75	2016	60	111769	50527	27599	16574	9794	6171
All Children	Phoenix	S75	2016	70	8025	863	113	28	0	0
All Children	Phoenix	S75	2016	80	0	0	0	0	0	0
All Children	Phoenix	S75	2016	90	0	0	0	0	0	0
All Children	Phoenix	S75	2016	100	0	0	0	0	0	0
All Children	Phoenix	S75	2017	0	575177	529830	499174	476614	457889	440961
All Children	Phoenix	S75	2017	10	558717	509322	476755	452836	433899	415358
All Children	Phoenix	S75	2017	20	519710	461611	424289	396152	374469	353734
All Children	Phoenix	S75	2017	30	464880	396180	355121	325612	302244	282543
All Children	Phoenix	S75	2017	40	393434	317233	276132	247315	224868	206228
All Children	Phoenix	S75	2017	50	294871	215216	174185	147237	127295	111274
All Children	Phoenix	S75	2017	60	151653	83660	53358	34902	24400	16956
All Children	Phoenix	S75	2017	70	29255	5775	1302	368	85	14
All Children	Phoenix	S75	2017	80	1062	14	0	0	0	0
All Children	Phoenix	S75	2017	90	0	0	0	0	0	0
All Children	Phoenix	S75	2017	100	0	0	0	0	0	0
All Children	Sacramento	S65	2015	0	311348	285237	266859	252643	241284	230547
All Children	Sacramento	S65	2015	10	297194	266813	246867	231750	219397	208582
All Children	Sacramento	S65	2015	20	261494	224025	200368	183411	170259	159218
All Children	Sacramento	S65	2015	30	207977	166113	141516	125033	113239	103596
All Children	Sacramento	S65	2015	40	127797	85902	64349	50218	40886	33494
All Children	Sacramento	S65	2015	50	32718	9930	3439	1359	551	225
All Children	Sacramento	S65	2015	60	1599	78	8	0	0	0
All Children	Sacramento	S65	2015	70	0	0	0	0	0	0
All Children	Sacramento	S65	2015	80	0	0	0	0	0	0
All Children	Sacramento	S65	2015	90	0	0	0	0	0	0
All Children	Sacramento	S65	2015	100	0	0	0	0	0	0
All Children	Sacramento	S65	2016	0	311681	285283	268039	253940	242146	232146
All Children	Sacramento	S65	2016	10	297411	267612	247962	233093	220857	210849
All Children	Sacramento	S65	2016	20	260516	223901	201493	184847	171633	160949

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Sacramento	S65	2016	30	207891	165950	141718	125763	114039	104101
All Children	Sacramento	S65	2016	40	132176	88394	66608	52679	42400	34744
All Children	Sacramento	S65	2016	50	41716	16374	7430	3867	1840	908
All Children	Sacramento	S65	2016	60	2632	163	8	0	0	0
All Children	Sacramento	S65	2016	70	0	0	0	0	0	0
All Children	Sacramento	S65	2016	80	0	0	0	0	0	0
All Children	Sacramento	S65	2016	90	0	0	0	0	0	0
All Children	Sacramento	S65	2016	100	0	0	0	0	0	0
All Children	Sacramento	S65	2017	0	311363	284484	266269	252263	240694	230686
All Children	Sacramento	S65	2017	10	297395	267574	247418	231797	219157	209382
All Children	Sacramento	S65	2017	20	262069	225112	201657	185616	172409	161159
All Children	Sacramento	S65	2017	30	210352	167906	144746	128286	115755	105964
All Children	Sacramento	S65	2017	40	132277	88759	66965	52881	43230	35396
All Children	Sacramento	S65	2017	50	33564	9643	3408	1413	590	272
All Children	Sacramento	S65	2017	60	1266	8	0	0	0	0
All Children	Sacramento	S65	2017	70	0	0	0	0	0	0
All Children	Sacramento	S65	2017	80	0	0	0	0	0	0
All Children	Sacramento	S65	2017	90	0	0	0	0	0	0
All Children	Sacramento	S65	2017	100	0	0	0	0	0	0
All Children	Sacramento	S70	2015	0	311348	285237	266859	252643	241284	230547
All Children	Sacramento	S70	2015	10	298141	268063	248078	233218	220803	209863
All Children	Sacramento	S70	2015	20	266067	229289	206012	189405	175631	164645
All Children	Sacramento	S70	2015	30	220384	178318	154109	137067	124357	114676
All Children	Sacramento	S70	2015	40	156811	112890	90684	74869	63207	54558
All Children	Sacramento	S70	2015	50	71810	36313	21398	13354	8735	5544
All Children	Sacramento	S70	2015	60	10645	1281	186	8	0	0
All Children	Sacramento	S70	2015	70	707	31	0	0	0	0
All Children	Sacramento	S70	2015	80	0	0	0	0	0	0
All Children	Sacramento	S70	2015	90	0	0	0	0	0	0
All Children	Sacramento	S70	2015	100	0	0	0	0	0	0
All Children	Sacramento	S70	2016	0	311681	285283	268039	253940	242146	232146
All Children	Sacramento	S70	2016	10	298125	268816	249002	234320	222169	212060
All Children	Sacramento	S70	2016	20	265446	229320	207006	190546	177068	166136
All Children	Sacramento	S70	2016	30	219522	178846	154567	136772	124311	114396
All Children	Sacramento	S70	2016	40	159016	115079	91375	75630	63984	54892
All Children	Sacramento	S70	2016	50	81841	44434	28331	18960	13137	9340
All Children	Sacramento	S70	2016	60	18378	4278	1219	411	179	47
All Children	Sacramento	S70	2016	70	1203	16	0	0	0	0
All Children	Sacramento	S70	2016	80	0	0	0	0	0	0
All Children	Sacramento	S70	2016	90	0	0	0	0	0	0
All Children	Sacramento	S70	2016	100	0	0	0	0	0	0
All Children	Sacramento	S70	2017	0	311363	284484	266269	252263	240694	230686
All Children	Sacramento	S70	2017	10	298413	268730	248971	233155	220616	210655
All Children	Sacramento	S70	2017	20	266782	230213	207480	191090	177681	166788
All Children	Sacramento	S70	2017	30	222519	180049	156175	139909	127587	117144
All Children	Sacramento	S70	2017	40	161167	117308	93736	78184	66849	57881
All Children	Sacramento	S70	2017	50	76748	39247	23634	15381	10482	7050

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Sacramento	S70	2017	60	15761	2244	435	54	8	0
All Children	Sacramento	S70	2017	70	272	0	0	0	0	0
All Children	Sacramento	S70	2017	80	0	0	0	0	0	0
All Children	Sacramento	S70	2017	90	0	0	0	0	0	0
All Children	Sacramento	S70	2017	100	0	0	0	0	0	0
All Children	Sacramento	S75	2015	0	311348	285237	266859	252643	241284	230547
All Children	Sacramento	S75	2015	10	298661	268715	248854	233823	221564	210624
All Children	Sacramento	S75	2015	20	268893	232806	209677	193077	179544	167999
All Children	Sacramento	S75	2015	30	228280	186167	162091	144901	131679	121694
All Children	Sacramento	S75	2015	40	173395	128783	105413	89838	77462	68425
All Children	Sacramento	S75	2015	50	99838	59015	39775	28479	20878	15699
All Children	Sacramento	S75	2015	60	29457	7919	2562	901	326	93
All Children	Sacramento	S75	2015	70	3603	202	23	0	0	0
All Children	Sacramento	S75	2015	80	116	0	0	0	0	0
All Children	Sacramento	S75	2015	90	0	0	0	0	0	0
All Children	Sacramento	S75	2015	100	0	0	0	0	0	0
All Children	Sacramento	S75	2016	0	311681	285283	268039	253940	242146	232146
All Children	Sacramento	S75	2016	10	298878	269383	249646	234996	222922	212767
All Children	Sacramento	S75	2016	20	268404	232798	210329	193955	180352	169389
All Children	Sacramento	S75	2016	30	226626	186734	162487	144334	131298	120731
All Children	Sacramento	S75	2016	40	173760	130157	106562	90226	78029	68565
All Children	Sacramento	S75	2016	50	109147	66243	46724	35094	26763	20342
All Children	Sacramento	S75	2016	60	41445	15878	7438	3766	1964	1040
All Children	Sacramento	S75	2016	70	6499	761	116	8	0	0
All Children	Sacramento	S75	2016	80	217	0	0	0	0	0
All Children	Sacramento	S75	2016	90	0	0	0	0	0	0
All Children	Sacramento	S75	2016	100	0	0	0	0	0	0
All Children	Sacramento	S75	2017	0	311363	284484	266269	252263	240694	230686
All Children	Sacramento	S75	2017	10	298739	269421	249670	233885	221331	211385
All Children	Sacramento	S75	2017	20	269841	233528	211059	194863	180996	170189
All Children	Sacramento	S75	2017	30	230050	187821	164086	147417	134466	123930
All Children	Sacramento	S75	2017	40	176990	133340	109505	93076	81010	71406
All Children	Sacramento	S75	2017	50	106570	64162	44465	32632	24713	19123
All Children	Sacramento	S75	2017	60	33851	9464	3230	1328	575	248
All Children	Sacramento	S75	2017	70	4643	280	16	0	0	0
All Children	Sacramento	S75	2017	80	54	0	0	0	0	0
All Children	Sacramento	S75	2017	90	0	0	0	0	0	0
All Children	Sacramento	S75	2017	100	0	0	0	0	0	0
All Children	St. Louis	S65	2015	0	355693	320478	297229	278469	263525	250485
All Children	St. Louis	S65	2015	10	338755	300106	275045	255985	239703	226653
All Children	St. Louis	S65	2015	20	297047	249820	221043	199980	183269	170010
All Children	St. Louis	S65	2015	30	235887	183552	155822	136744	121463	109297
All Children	St. Louis	S65	2015	40	159583	108140	82360	64911	52636	43356
All Children	St. Louis	S65	2015	50	65585	26864	12348	5828	2978	1439
All Children	St. Louis	S65	2015	60	4034	200	9	0	0	0
All Children	St. Louis	S65	2015	70	0	0	0	0	0	0
All Children	St. Louis	S65	2015	80	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	St. Louis	S65	2015	90	0	0	0	0	0	0
All Children	St. Louis	S65	2015	100	0	0	0	0	0	0
All Children	St. Louis	S65	2016	0	359080	325268	301645	283232	267933	254646
All Children	St. Louis	S65	2016	10	344483	306763	281083	260903	244975	231552
All Children	St. Louis	S65	2016	20	305898	258790	228693	206910	190855	177541
All Children	St. Louis	S65	2016	30	248399	195764	166413	146461	131635	119815
All Children	St. Louis	S65	2016	40	174308	122456	95655	77469	64037	54257
All Children	St. Louis	S65	2016	50	87059	42300	23049	13141	7522	4435
All Children	St. Louis	S65	2016	60	16847	2204	392	109	18	0
All Children	St. Louis	S65	2016	70	55	0	0	0	0	0
All Children	St. Louis	S65	2016	80	0	0	0	0	0	0
All Children	St. Louis	S65	2016	90	0	0	0	0	0	0
All Children	St. Louis	S65	2016	100	0	0	0	0	0	0
All Children	St. Louis	S65	2017	0	355702	320669	297356	279526	264764	252179
All Children	St. Louis	S65	2017	10	342115	304487	279471	260520	244875	231352
All Children	St. Louis	S65	2017	20	305215	259682	231033	210498	194106	180801
All Children	St. Louis	S65	2017	30	251869	200053	171804	151988	137309	125425
All Children	St. Louis	S65	2017	40	179900	128548	101711	83753	70503	60222
All Children	St. Louis	S65	2017	50	77387	35270	19178	11028	6693	3943
All Children	St. Louis	S65	2017	60	5764	464	27	0	0	0
All Children	St. Louis	S65	2017	70	146	0	0	0	0	0
All Children	St. Louis	S65	2017	80	0	0	0	0	0	0
All Children	St. Louis	S65	2017	90	0	0	0	0	0	0
All Children	St. Louis	S65	2017	100	0	0	0	0	0	0
All Children	St. Louis	S70	2015	0	355693	320478	297229	278469	263525	250485
All Children	St. Louis	S70	2015	10	339492	301063	276193	256868	240896	227636
All Children	St. Louis	S70	2015	20	301664	255257	225533	205134	188351	175019
All Children	St. Louis	S70	2015	30	246742	194862	166531	146433	131271	119023
All Children	St. Louis	S70	2015	40	181020	128612	101556	83106	69893	59484
All Children	St. Louis	S70	2015	50	102157	54403	32711	20781	13660	8751
All Children	St. Louis	S70	2015	60	22320	4071	883	209	36	18
All Children	St. Louis	S70	2015	70	446	9	0	0	0	0
All Children	St. Louis	S70	2015	80	0	0	0	0	0	0
All Children	St. Louis	S70	2015	90	0	0	0	0	0	0
All Children	St. Louis	S70	2015	100	0	0	0	0	0	0
All Children	St. Louis	S70	2016	0	359080	325268	301645	283232	267933	254646
All Children	St. Louis	S70	2016	10	345411	307656	282367	262223	246469	232745
All Children	St. Louis	S70	2016	20	310497	264108	234567	212611	196091	183033
All Children	St. Louis	S70	2016	30	259664	206792	177141	156888	141279	129195
All Children	St. Louis	S70	2016	40	195627	143046	114843	96156	82004	71568
All Children	St. Louis	S70	2016	50	120880	70330	46462	32274	22429	16064
All Children	St. Louis	S70	2016	60	47609	14325	5036	1803	519	182
All Children	St. Louis	S70	2016	70	4863	155	9	0	0	0
All Children	St. Louis	S70	2016	80	0	0	0	0	0	0
All Children	St. Louis	S70	2016	90	0	0	0	0	0	0
All Children	St. Louis	S70	2016	100	0	0	0	0	0	0
All Children	St. Louis	S70	2017	0	355702	320669	297356	279526	264764	252179

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All Children	St. Louis	S70	2017	10	343053	305771	280855	261850	246314	232782
All Children	St. Louis	S70	2017	20	309878	264991	236688	216326	199579	186092
All Children	St. Louis	S70	2017	30	262851	211363	182614	162597	147025	134686
All Children	St. Louis	S70	2017	40	202211	149748	122019	103132	89763	78763
All Children	St. Louis	S70	2017	50	119123	69201	45870	32410	23859	17685
All Children	St. Louis	S70	2017	60	28595	6520	1949	574	219	46
All Children	St. Louis	S70	2017	70	1685	36	0	0	0	0
All Children	St. Louis	S70	2017	80	36	0	0	0	0	0
All Children	St. Louis	S70	2017	90	0	0	0	0	0	0
All Children	St. Louis	S70	2017	100	0	0	0	0	0	0
All Children	St. Louis	S75	2015	0	355693	320478	297229	278469	263525	250485
All Children	St. Louis	S75	2015	10	339574	301336	276420	257105	241196	227846
All Children	St. Louis	S75	2015	20	303931	257825	228420	207748	190864	177468
All Children	St. Louis	S75	2015	30	253353	201200	172569	152708	137054	124186
All Children	St. Louis	S75	2015	40	193851	140742	113149	94144	80119	69246
All Children	St. Louis	S75	2015	50	123958	73544	49458	34760	24742	18186
All Children	St. Louis	S75	2015	60	46635	14662	5245	2031	729	291
All Children	St. Louis	S75	2015	70	4162	255	27	0	0	0
All Children	St. Louis	S75	2015	80	0	0	0	0	0	0
All Children	St. Louis	S75	2015	90	0	0	0	0	0	0
All Children	St. Louis	S75	2015	100	0	0	0	0	0	0
All Children	St. Louis	S75	2016	0	359080	325268	301645	283232	267933	254646
All Children	St. Louis	S75	2016	10	345867	308029	282950	262851	246778	233301
All Children	St. Louis	S75	2016	20	313065	266849	237608	215935	199151	185846
All Children	St. Louis	S75	2016	30	266685	213503	183834	163372	147171	134914
All Children	St. Louis	S75	2016	40	208531	155148	126526	107166	92805	81522
All Children	St. Louis	S75	2016	50	141707	89590	63491	46871	35661	27447
All Children	St. Louis	S75	2016	60	70922	29259	13223	6675	3215	1539
All Children	St. Louis	S75	2016	70	17730	2113	310	73	9	0
All Children	St. Louis	S75	2016	80	555	9	0	0	0	0
All Children	St. Louis	S75	2016	90	0	0	0	0	0	0
All Children	St. Louis	S75	2016	100	0	0	0	0	0	0
All Children	St. Louis	S75	2017	0	355702	320669	297356	279526	264764	252179
All Children	St. Louis	S75	2017	10	343545	306290	281420	262369	246815	233392
All Children	St. Louis	S75	2017	20	312455	267942	240076	219768	202757	189143
All Children	St. Louis	S75	2017	30	268880	218348	189107	168644	152963	140642
All Children	St. Louis	S75	2017	40	215106	161833	133420	114788	100673	88780
All Children	St. Louis	S75	2017	50	143109	91931	66141	50159	39022	30835
All Children	St. Louis	S75	2017	60	55623	19743	8351	3843	1730	829
All Children	St. Louis	S75	2017	70	6484	565	64	9	0	0
All Children	St. Louis	S75	2017	80	492	0	0	0	0	0
All Children	St. Louis	S75	2017	90	0	0	0	0	0	0
All Children	St. Louis	S75	2017	100	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2015	0	96464	87526	81735	77135	73342	70214
Asthma Children	Atlanta	S65	2015	10	91420	81271	74714	69912	66119	62689
Asthma Children	Atlanta	S65	2015	20	79899	67854	60409	54961	50825	47072
Asthma Children	Atlanta	S65	2015	30	63011	49392	41705	36277	32061	28953

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
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Asthma Children	Atlanta	S65	2015	40	41221	27259	20358	15617	12570	10330
Asthma Children	Atlanta	S65	2015	50	14164	5206	2219	928	565	323
Asthma Children	Atlanta	S65	2015	60	1654	101	20	0	0	0
Asthma Children	Atlanta	S65	2015	70	40	0	0	0	0	0
Asthma Children	Atlanta	S65	2015	80	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2015	90	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2015	100	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2016	0	99390	90068	83935	78951	74875	71627
Asthma Children	Atlanta	S65	2016	10	94850	84600	77680	72595	68156	64948
Asthma Children	Atlanta	S65	2016	20	84278	71586	63899	58209	53549	50139
Asthma Children	Atlanta	S65	2016	30	67733	53549	45619	40313	37105	33755
Asthma Children	Atlanta	S65	2016	40	46043	31637	24333	20035	16747	14124
Asthma Children	Atlanta	S65	2016	50	18260	6961	3087	1735	908	484
Asthma Children	Atlanta	S65	2016	60	2724	222	40	20	0	0
Asthma Children	Atlanta	S65	2016	70	222	0	0	0	0	0
Asthma Children	Atlanta	S65	2016	80	20	0	0	0	0	0
Asthma Children	Atlanta	S65	2016	90	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2016	100	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2017	0	96827	88757	82361	77922	73786	70416
Asthma Children	Atlanta	S65	2017	10	91904	82502	75521	70860	66562	62810
Asthma Children	Atlanta	S65	2017	20	80605	68621	60792	54921	50462	46850
Asthma Children	Atlanta	S65	2017	30	63092	49190	41059	35914	32182	29175
Asthma Children	Atlanta	S65	2017	40	38073	25261	18623	13982	11178	9180
Asthma Children	Atlanta	S65	2017	50	9100	2219	807	282	101	61
Asthma Children	Atlanta	S65	2017	60	424	0	0	0	0	0
Asthma Children	Atlanta	S65	2017	70	20	0	0	0	0	0
Asthma Children	Atlanta	S65	2017	80	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2017	90	0	0	0	0	0	0
Asthma Children	Atlanta	S65	2017	100	0	0	0	0	0	0
Asthma Children	Atlanta	S70	2015	0	96464	87526	81735	77135	73342	70214
Asthma Children	Atlanta	S70	2015	10	91985	82119	75218	70376	66764	63254
Asthma Children	Atlanta	S70	2015	20	81574	69952	62325	56938	52963	49049
Asthma Children	Atlanta	S70	2015	30	67047	53085	45660	40333	36257	32747
Asthma Children	Atlanta	S70	2015	40	49049	34744	27480	22396	18724	15879
Asthma Children	Atlanta	S70	2015	50	24515	12913	7627	4620	2805	1634
Asthma Children	Atlanta	S70	2015	60	5044	1090	303	101	20	0
Asthma Children	Atlanta	S70	2015	70	585	40	0	0	0	0
Asthma Children	Atlanta	S70	2015	80	0	0	0	0	0	0
Asthma Children	Atlanta	S70	2015	90	0	0	0	0	0	0
Asthma Children	Atlanta	S70	2015	100	0	0	0	0	0	0
Asthma Children	Atlanta	S70	2016	0	99390	90068	83935	78951	74875	71627
Asthma Children	Atlanta	S70	2016	10	95254	85004	78346	73281	69125	65675
Asthma Children	Atlanta	S70	2016	20	85871	73665	65977	60489	56010	52197
Asthma Children	Atlanta	S70	2016	30	71385	57423	49453	44187	40373	37569
Asthma Children	Atlanta	S70	2016	40	52802	38396	31254	26411	23021	19934
Asthma Children	Atlanta	S70	2016	50	28691	15919	10028	6557	4540	2926
Asthma Children	Atlanta	S70	2016	60	8333	1715	504	101	20	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Atlanta	S70	2016	70	1271	20	0	0	0	0
Asthma Children	Atlanta	S70	2016	80	202	0	0	0	0	0
Asthma Children	Atlanta	S70	2016	90	20	0	0	0	0	0
Asthma Children	Atlanta	S70	2016	100	0	0	0	0	0	0
Asthma Children	Atlanta	S70	2017	0	96827	88757	82361	77922	73786	70416
Asthma Children	Atlanta	S70	2017	10	92187	83067	76207	71385	67349	63738
Asthma Children	Atlanta	S70	2017	20	82381	71022	62830	57100	52742	49090
Asthma Children	Atlanta	S70	2017	30	66966	53125	44731	39425	35813	32726
Asthma Children	Atlanta	S70	2017	40	45821	32424	24898	21105	17554	14608
Asthma Children	Atlanta	S70	2017	50	19127	7990	4156	1997	1211	686
Asthma Children	Atlanta	S70	2017	60	2078	202	81	0	0	0
Asthma Children	Atlanta	S70	2017	70	141	0	0	0	0	0
Asthma Children	Atlanta	S70	2017	80	0	0	0	0	0	0
Asthma Children	Atlanta	S70	2017	90	0	0	0	0	0	0
Asthma Children	Atlanta	S70	2017	100	0	0	0	0	0	0
Asthma Children	Atlanta	S75	2015	0	96464	87526	81735	77135	73342	70214
Asthma Children	Atlanta	S75	2015	10	92368	82522	75884	70719	67208	63818
Asthma Children	Atlanta	S75	2015	20	82704	71264	63778	58653	54335	50542
Asthma Children	Atlanta	S75	2015	30	70537	56575	48626	43400	39385	35874
Asthma Children	Atlanta	S75	2015	40	54941	40494	32747	27541	23486	20802
Asthma Children	Atlanta	S75	2015	50	34522	21185	14628	10270	7808	6134
Asthma Children	Atlanta	S75	2015	60	12025	3834	1614	807	404	161
Asthma Children	Atlanta	S75	2015	70	2119	202	20	20	0	0
Asthma Children	Atlanta	S75	2015	80	282	0	0	0	0	0
Asthma Children	Atlanta	S75	2015	90	0	0	0	0	0	0
Asthma Children	Atlanta	S75	2015	100	0	0	0	0	0	0
Asthma Children	Atlanta	S75	2016	0	99390	90068	83935	78951	74875	71627
Asthma Children	Atlanta	S75	2016	10	95718	85488	78608	73523	69710	66260
Asthma Children	Atlanta	S75	2016	20	87344	75299	67430	62144	57584	54093
Asthma Children	Atlanta	S75	2016	30	74774	60610	52540	47274	43158	39829
Asthma Children	Atlanta	S75	2016	40	58209	43783	36298	31112	27541	24979
Asthma Children	Atlanta	S75	2016	50	37912	23808	17412	13135	10088	7788
Asthma Children	Atlanta	S75	2016	60	17009	5972	2764	1311	605	242
Asthma Children	Atlanta	S75	2016	70	4439	565	101	20	20	0
Asthma Children	Atlanta	S75	2016	80	888	20	0	0	0	0
Asthma Children	Atlanta	S75	2016	90	182	0	0	0	0	0
Asthma Children	Atlanta	S75	2016	100	20	0	0	0	0	0
Asthma Children	Atlanta	S75	2017	0	96827	88757	82361	77922	73786	70416
Asthma Children	Atlanta	S75	2017	10	92368	83672	76711	71808	67934	64141
Asthma Children	Atlanta	S75	2017	20	83773	72373	64726	58835	54356	50805
Asthma Children	Atlanta	S75	2017	30	70114	56837	48323	42794	38739	35269
Asthma Children	Atlanta	S75	2017	40	52277	38073	30547	26129	22376	19491
Asthma Children	Atlanta	S75	2017	50	29054	15899	9685	6477	4459	3067
Asthma Children	Atlanta	S75	2017	60	7425	1614	464	182	61	40
Asthma Children	Atlanta	S75	2017	70	545	61	20	0	0	0
Asthma Children	Atlanta	S75	2017	80	61	0	0	0	0	0
Asthma Children	Atlanta	S75	2017	90	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Atlanta	S75	2017	100	0	0	0	0	0	0
Asthma Children	Boston	S65	2015	0	110791	99209	91427	85625	79800	75522
Asthma Children	Boston	S65	2015	10	106673	94181	86421	79072	73474	69310
Asthma Children	Boston	S65	2015	20	93157	78526	69333	62484	57000	52677
Asthma Children	Boston	S65	2015	30	73633	57273	47921	41595	36748	33153
Asthma Children	Boston	S65	2015	40	47307	30218	22572	16861	13129	10331
Asthma Children	Boston	S65	2015	50	17066	5689	2275	910	319	182
Asthma Children	Boston	S65	2015	60	3163	319	0	0	0	0
Asthma Children	Boston	S65	2015	70	455	0	0	0	0	0
Asthma Children	Boston	S65	2015	80	68	0	0	0	0	0
Asthma Children	Boston	S65	2015	90	0	0	0	0	0	0
Asthma Children	Boston	S65	2015	100	0	0	0	0	0	0
Asthma Children	Boston	S65	2016	0	115661	104420	96593	90403	85056	80756
Asthma Children	Boston	S65	2016	10	112270	99710	91609	84647	79049	74430
Asthma Children	Boston	S65	2016	20	99209	83486	73815	65988	59935	55498
Asthma Children	Boston	S65	2016	30	78526	60845	50924	43939	39274	35474
Asthma Children	Boston	S65	2016	40	51220	33859	24916	18954	14972	11946
Asthma Children	Boston	S65	2016	50	19705	7031	3163	1456	683	250
Asthma Children	Boston	S65	2016	60	2298	205	23	0	0	0
Asthma Children	Boston	S65	2016	70	91	0	0	0	0	0
Asthma Children	Boston	S65	2016	80	0	0	0	0	0	0
Asthma Children	Boston	S65	2016	90	0	0	0	0	0	0
Asthma Children	Boston	S65	2016	100	0	0	0	0	0	0
Asthma Children	Boston	S65	2017	0	112976	101257	93612	86990	82075	77684
Asthma Children	Boston	S65	2017	10	108607	96661	88469	81962	76364	71540
Asthma Children	Boston	S65	2017	20	96843	81529	71244	63235	57819	52927
Asthma Children	Boston	S65	2017	30	76341	57592	48763	42073	37340	33176
Asthma Children	Boston	S65	2017	40	50697	33563	24734	18067	13903	11286
Asthma Children	Boston	S65	2017	50	23915	9716	4460	1752	592	250
Asthma Children	Boston	S65	2017	60	5165	523	46	0	0	0
Asthma Children	Boston	S65	2017	70	387	0	0	0	0	0
Asthma Children	Boston	S65	2017	80	0	0	0	0	0	0
Asthma Children	Boston	S65	2017	90	0	0	0	0	0	0
Asthma Children	Boston	S65	2017	100	0	0	0	0	0	0
Asthma Children	Boston	S70	2015	0	110791	99209	91427	85625	79800	75522
Asthma Children	Boston	S70	2015	10	106764	94203	86763	79140	73770	69492
Asthma Children	Boston	S70	2015	20	94249	79754	70380	63235	57956	53632
Asthma Children	Boston	S70	2015	30	76341	59867	50378	43893	39069	35269
Asthma Children	Boston	S70	2015	40	52904	35429	27669	21617	17453	14495
Asthma Children	Boston	S70	2015	50	25804	11286	5552	3004	1547	865
Asthma Children	Boston	S70	2015	60	6166	1047	250	91	23	23
Asthma Children	Boston	S70	2015	70	1024	0	0	0	0	0
Asthma Children	Boston	S70	2015	80	114	0	0	0	0	0
Asthma Children	Boston	S70	2015	90	0	0	0	0	0	0
Asthma Children	Boston	S70	2015	100	0	0	0	0	0	0
Asthma Children	Boston	S70	2016	0	115661	104420	96593	90403	85056	80756
Asthma Children	Boston	S70	2016	10	112452	99892	91541	84783	79185	74612

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Boston	S70	2016	20	100370	84738	75363	67603	61346	56954
Asthma Children	Boston	S70	2016	30	81893	63781	54269	46851	41345	37545
Asthma Children	Boston	S70	2016	40	57887	39456	30559	24552	19842	16747
Asthma Children	Boston	S70	2016	50	29968	14176	8146	4892	2822	1889
Asthma Children	Boston	S70	2016	60	7782	1343	250	0	0	0
Asthma Children	Boston	S70	2016	70	796	68	0	0	0	0
Asthma Children	Boston	S70	2016	80	0	0	0	0	0	0
Asthma Children	Boston	S70	2016	90	0	0	0	0	0	0
Asthma Children	Boston	S70	2016	100	0	0	0	0	0	0
Asthma Children	Boston	S70	2017	0	112976	101257	93612	86990	82075	77684
Asthma Children	Boston	S70	2017	10	108744	96775	88674	81939	76318	71813
Asthma Children	Boston	S70	2017	20	97344	82417	72200	64486	58843	53678
Asthma Children	Boston	S70	2017	30	79140	60504	51015	44144	39297	35019
Asthma Children	Boston	S70	2017	40	56386	39365	29945	23665	18590	15382
Asthma Children	Boston	S70	2017	50	32812	16247	8624	4778	2731	1502
Asthma Children	Boston	S70	2017	60	11605	2617	455	137	0	0
Asthma Children	Boston	S70	2017	70	1616	23	0	0	0	0
Asthma Children	Boston	S70	2017	80	68	0	0	0	0	0
Asthma Children	Boston	S70	2017	90	0	0	0	0	0	0
Asthma Children	Boston	S70	2017	100	0	0	0	0	0	0
Asthma Children	Boston	S75	2015	0	110791	99209	91427	85625	79800	75522
Asthma Children	Boston	S75	2015	10	106809	94021	86603	79094	73588	69333
Asthma Children	Boston	S75	2015	20	94454	80232	70880	63417	58251	53928
Asthma Children	Boston	S75	2015	30	77274	61050	51584	45008	40071	36384
Asthma Children	Boston	S75	2015	40	55430	38318	29695	23938	20183	16725
Asthma Children	Boston	S75	2015	50	29968	14654	8374	5074	3254	1684
Asthma Children	Boston	S75	2015	60	9375	2184	614	182	68	23
Asthma Children	Boston	S75	2015	70	1752	68	0	0	0	0
Asthma Children	Boston	S75	2015	80	137	0	0	0	0	0
Asthma Children	Boston	S75	2015	90	0	0	0	0	0	0
Asthma Children	Boston	S75	2015	100	0	0	0	0	0	0
Asthma Children	Boston	S75	2016	0	115661	104420	96593	90403	85056	80756
Asthma Children	Boston	S75	2016	10	112430	100006	91609	84715	79185	74430
Asthma Children	Boston	S75	2016	20	100666	85147	75704	67968	61915	57341
Asthma Children	Boston	S75	2016	30	82940	65283	55566	48239	42596	38546
Asthma Children	Boston	S75	2016	40	60777	42824	33130	27146	22618	18954
Asthma Children	Boston	S75	2016	50	35383	18795	11013	7486	5097	3459
Asthma Children	Boston	S75	2016	60	12674	3163	933	205	46	0
Asthma Children	Boston	S75	2016	70	1684	137	0	0	0	0
Asthma Children	Boston	S75	2016	80	137	0	0	0	0	0
Asthma Children	Boston	S75	2016	90	0	0	0	0	0	0
Asthma Children	Boston	S75	2016	100	0	0	0	0	0	0
Asthma Children	Boston	S75	2017	0	112976	101257	93612	86990	82075	77684
Asthma Children	Boston	S75	2017	10	108789	96547	88674	81848	76364	71677
Asthma Children	Boston	S75	2017	20	97935	82894	72632	64691	59184	53792
Asthma Children	Boston	S75	2017	30	80528	62120	52335	45145	40412	36271
Asthma Children	Boston	S75	2017	40	59435	41572	32653	26168	21002	17703

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Boston	S75	2017	50	37932	20274	11696	7122	4505	2958
Asthma Children	Boston	S75	2017	60	16315	4915	1343	319	114	46
Asthma Children	Boston	S75	2017	70	3641	387	68	0	0	0
Asthma Children	Boston	S75	2017	80	319	0	0	0	0	0
Asthma Children	Boston	S75	2017	90	0	0	0	0	0	0
Asthma Children	Boston	S75	2017	100	0	0	0	0	0	0
Asthma Children	Dallas	S65	2015	0	90113	82594	76635	73254	69872	66775
Asthma Children	Dallas	S65	2015	10	86330	77250	71267	67248	63488	60249
Asthma Children	Dallas	S65	2015	20	77155	66373	59350	54408	50247	46463
Asthma Children	Dallas	S65	2015	30	63181	50649	43177	38258	35161	32229
Asthma Children	Dallas	S65	2015	40	46014	32844	26246	21967	18373	15984
Asthma Children	Dallas	S65	2015	50	23196	11846	6834	4043	2601	1702
Asthma Children	Dallas	S65	2015	60	3878	709	95	0	0	0
Asthma Children	Dallas	S65	2015	70	71	0	0	0	0	0
Asthma Children	Dallas	S65	2015	80	0	0	0	0	0	0
Asthma Children	Dallas	S65	2015	90	0	0	0	0	0	0
Asthma Children	Dallas	S65	2015	100	0	0	0	0	0	0
Asthma Children	Dallas	S65	2016	0	90420	83208	78266	74318	71315	68501
Asthma Children	Dallas	S65	2016	10	86164	78361	73325	68879	65427	62069
Asthma Children	Dallas	S65	2016	20	77273	67106	60367	55212	51145	48142
Asthma Children	Dallas	S65	2016	30	62022	49466	42846	37880	33955	31212
Asthma Children	Dallas	S65	2016	40	41427	28895	22038	17592	14802	12059
Asthma Children	Dallas	S65	2016	50	14991	5817	2743	1584	662	355
Asthma Children	Dallas	S65	2016	60	1395	47	24	0	0	0
Asthma Children	Dallas	S65	2016	70	47	0	0	0	0	0
Asthma Children	Dallas	S65	2016	80	0	0	0	0	0	0
Asthma Children	Dallas	S65	2016	90	0	0	0	0	0	0
Asthma Children	Dallas	S65	2016	100	0	0	0	0	0	0
Asthma Children	Dallas	S65	2017	0	91035	83563	78645	74341	70724	68028
Asthma Children	Dallas	S65	2017	10	87819	79023	72592	68241	64623	61218
Asthma Children	Dallas	S65	2017	20	78196	67153	60509	55661	51263	48119
Asthma Children	Dallas	S65	2017	30	63252	51192	43933	39133	35634	32583
Asthma Children	Dallas	S65	2017	40	44974	31638	25301	20524	17427	14282
Asthma Children	Dallas	S65	2017	50	21588	10735	5675	3003	1608	1040
Asthma Children	Dallas	S65	2017	60	3476	402	47	0	0	0
Asthma Children	Dallas	S65	2017	70	118	0	0	0	0	0
Asthma Children	Dallas	S65	2017	80	0	0	0	0	0	0
Asthma Children	Dallas	S65	2017	90	0	0	0	0	0	0
Asthma Children	Dallas	S65	2017	100	0	0	0	0	0	0
Asthma Children	Dallas	S70	2015	0	90113	82594	76635	73254	69872	66775
Asthma Children	Dallas	S70	2015	10	86401	77368	71362	67461	63606	60532
Asthma Children	Dallas	S70	2015	20	77935	67082	60130	55401	51074	47740
Asthma Children	Dallas	S70	2015	30	65285	52706	44903	40150	36556	33931
Asthma Children	Dallas	S70	2015	40	50176	37052	29912	25395	21659	18869
Asthma Children	Dallas	S70	2015	50	30668	18467	12698	8654	6006	4564
Asthma Children	Dallas	S70	2015	60	9813	2861	1064	473	213	71
Asthma Children	Dallas	S70	2015	70	946	118	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Dallas	S70	2015	80	24	0	0	0	0	0
Asthma Children	Dallas	S70	2015	90	0	0	0	0	0	0
Asthma Children	Dallas	S70	2015	100	0	0	0	0	0	0
Asthma Children	Dallas	S70	2016	0	90420	83208	78266	74318	71315	68501
Asthma Children	Dallas	S70	2016	10	86259	78598	73727	69116	65640	62471
Asthma Children	Dallas	S70	2016	20	77817	67933	61384	56087	52375	49041
Asthma Children	Dallas	S70	2016	30	64245	51665	44761	40079	35894	32702
Asthma Children	Dallas	S70	2016	40	45707	32867	25608	21446	18373	15677
Asthma Children	Dallas	S70	2016	50	21494	10144	5746	3689	2270	1513
Asthma Children	Dallas	S70	2016	60	2908	378	118	24	24	0
Asthma Children	Dallas	S70	2016	70	426	0	0	0	0	0
Asthma Children	Dallas	S70	2016	80	0	0	0	0	0	0
Asthma Children	Dallas	S70	2016	90	0	0	0	0	0	0
Asthma Children	Dallas	S70	2016	100	0	0	0	0	0	0
Asthma Children	Dallas	S70	2017	0	91035	83563	78645	74341	70724	68028
Asthma Children	Dallas	S70	2017	10	88008	79236	72899	68525	64883	61502
Asthma Children	Dallas	S70	2017	20	79047	68052	61407	56773	52446	49183
Asthma Children	Dallas	S70	2017	30	65238	52942	46085	41261	37525	34570
Asthma Children	Dallas	S70	2017	40	49159	35137	28469	24378	20974	18089
Asthma Children	Dallas	S70	2017	50	28114	15913	9907	6503	4209	2767
Asthma Children	Dallas	S70	2017	60	8134	1561	402	142	24	0
Asthma Children	Dallas	S70	2017	70	355	0	0	0	0	0
Asthma Children	Dallas	S70	2017	80	0	0	0	0	0	0
Asthma Children	Dallas	S70	2017	90	0	0	0	0	0	0
Asthma Children	Dallas	S70	2017	100	0	0	0	0	0	0
Asthma Children	Dallas	S75	2015	0	90113	82594	76635	73254	69872	66775
Asthma Children	Dallas	S75	2015	10	86519	77604	71504	67650	63748	60627
Asthma Children	Dallas	S75	2015	20	78621	67815	60840	56134	52115	48568
Asthma Children	Dallas	S75	2015	30	66798	54077	46487	41782	37951	35043
Asthma Children	Dallas	S75	2015	40	52824	40055	32536	28044	24095	21210
Asthma Children	Dallas	S75	2015	50	36580	23622	16859	12958	10357	7425
Asthma Children	Dallas	S75	2015	60	15724	6313	2956	1490	969	520
Asthma Children	Dallas	S75	2015	70	2956	355	47	0	0	0
Asthma Children	Dallas	S75	2015	80	166	0	0	0	0	0
Asthma Children	Dallas	S75	2015	90	0	0	0	0	0	0
Asthma Children	Dallas	S75	2015	100	0	0	0	0	0	0
Asthma Children	Dallas	S75	2016	0	90420	83208	78266	74318	71315	68501
Asthma Children	Dallas	S75	2016	10	86282	78739	73774	69352	65876	62708
Asthma Children	Dallas	S75	2016	20	78408	68525	62046	56655	53131	49916
Asthma Children	Dallas	S75	2016	30	65971	53250	46345	41592	37431	34286
Asthma Children	Dallas	S75	2016	40	48781	35847	28611	23764	20572	18136
Asthma Children	Dallas	S75	2016	50	27311	15015	9104	6313	4327	3121
Asthma Children	Dallas	S75	2016	60	6006	1348	331	142	24	0
Asthma Children	Dallas	S75	2016	70	1111	0	0	0	0	0
Asthma Children	Dallas	S75	2016	80	47	0	0	0	0	0
Asthma Children	Dallas	S75	2016	90	0	0	0	0	0	0
Asthma Children	Dallas	S75	2016	100	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Dallas	S75	2017	0	91035	83563	78645	74341	70724	68028
Asthma Children	Dallas	S75	2017	10	88127	79401	73277	68595	65214	61715
Asthma Children	Dallas	S75	2017	20	79685	68737	61951	57151	52989	49679
Asthma Children	Dallas	S75	2017	30	66822	54763	47598	42373	38826	35657
Asthma Children	Dallas	S75	2017	40	52091	38164	31236	26578	23196	20666
Asthma Children	Dallas	S75	2017	50	32560	19508	13596	10144	7236	5249
Asthma Children	Dallas	S75	2017	60	13100	3831	1064	473	213	47
Asthma Children	Dallas	S75	2017	70	1371	95	0	0	0	0
Asthma Children	Dallas	S75	2017	80	24	0	0	0	0	0
Asthma Children	Dallas	S75	2017	90	0	0	0	0	0	0
Asthma Children	Dallas	S75	2017	100	0	0	0	0	0	0
Asthma Children	Detroit	S65	2015	0	77853	70222	64551	60129	56729	53504
Asthma Children	Detroit	S65	2015	10	74853	66806	60319	55550	51995	48960
Asthma Children	Detroit	S65	2015	20	66285	55741	49168	44242	39872	36542
Asthma Children	Detroit	S65	2015	30	53486	41467	34652	30246	26656	23847
Asthma Children	Detroit	S65	2015	40	38155	25911	19632	15366	12435	10024
Asthma Children	Detroit	S65	2015	50	16927	7267	3711	1890	1041	468
Asthma Children	Detroit	S65	2015	60	1717	173	0	0	0	0
Asthma Children	Detroit	S65	2015	70	0	0	0	0	0	0
Asthma Children	Detroit	S65	2015	80	0	0	0	0	0	0
Asthma Children	Detroit	S65	2015	90	0	0	0	0	0	0
Asthma Children	Detroit	S65	2015	100	0	0	0	0	0	0
Asthma Children	Detroit	S65	2016	0	80871	72806	67187	62747	58741	55706
Asthma Children	Detroit	S65	2016	10	78079	68627	62956	58204	54180	51249
Asthma Children	Detroit	S65	2016	20	69008	58065	51492	46393	42508	38987
Asthma Children	Detroit	S65	2016	30	56244	44034	37496	32796	29136	26223
Asthma Children	Detroit	S65	2016	40	40323	28287	21488	17777	13996	11655
Asthma Children	Detroit	S65	2016	50	21904	10857	6053	3018	1682	850
Asthma Children	Detroit	S65	2016	60	5064	486	104	17	0	0
Asthma Children	Detroit	S65	2016	70	52	0	0	0	0	0
Asthma Children	Detroit	S65	2016	80	0	0	0	0	0	0
Asthma Children	Detroit	S65	2016	90	0	0	0	0	0	0
Asthma Children	Detroit	S65	2016	100	0	0	0	0	0	0
Asthma Children	Detroit	S65	2017	0	79917	71558	66060	62140	58516	54718
Asthma Children	Detroit	S65	2017	10	76847	67968	62123	57215	53885	50451
Asthma Children	Detroit	S65	2017	20	68609	57649	50937	45959	41762	38033
Asthma Children	Detroit	S65	2017	30	56122	43236	36663	31998	28443	25442
Asthma Children	Detroit	S65	2017	40	39820	28079	21540	17222	14169	11377
Asthma Children	Detroit	S65	2017	50	19806	8914	5238	2636	1422	954
Asthma Children	Detroit	S65	2017	60	2012	173	35	0	0	0
Asthma Children	Detroit	S65	2017	70	35	0	0	0	0	0
Asthma Children	Detroit	S65	2017	80	0	0	0	0	0	0
Asthma Children	Detroit	S65	2017	90	0	0	0	0	0	0
Asthma Children	Detroit	S65	2017	100	0	0	0	0	0	0
Asthma Children	Detroit	S70	2015	0	77853	70222	64551	60129	56729	53504
Asthma Children	Detroit	S70	2015	10	74905	66910	60406	55723	51977	49064
Asthma Children	Detroit	S70	2015	20	67205	56469	50139	45005	40756	37635

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Detroit	S70	2015	30	55793	43288	36369	31825	28200	25668
Asthma Children	Detroit	S70	2015	40	41762	29518	23396	18904	15730	12938
Asthma Children	Detroit	S70	2015	50	24107	12366	7544	4613	3035	1960
Asthma Children	Detroit	S70	2015	60	6209	1301	243	52	0	0
Asthma Children	Detroit	S70	2015	70	121	0	0	0	0	0
Asthma Children	Detroit	S70	2015	80	0	0	0	0	0	0
Asthma Children	Detroit	S70	2015	90	0	0	0	0	0	0
Asthma Children	Detroit	S70	2015	100	0	0	0	0	0	0
Asthma Children	Detroit	S70	2016	0	80871	72806	67187	62747	58741	55706
Asthma Children	Detroit	S70	2016	10	78218	68852	63112	58446	54215	51336
Asthma Children	Detroit	S70	2016	20	69771	58897	52498	47555	43583	40115
Asthma Children	Detroit	S70	2016	30	58325	45994	39334	34998	31148	28096
Asthma Children	Detroit	S70	2016	40	44624	32137	25390	21020	17690	14672
Asthma Children	Detroit	S70	2016	50	28477	16372	10753	7267	4839	3313
Asthma Children	Detroit	S70	2016	60	11776	3469	1110	243	139	69
Asthma Children	Detroit	S70	2016	70	1110	17	0	0	0	0
Asthma Children	Detroit	S70	2016	80	0	0	0	0	0	0
Asthma Children	Detroit	S70	2016	90	0	0	0	0	0	0
Asthma Children	Detroit	S70	2016	100	0	0	0	0	0	0
Asthma Children	Detroit	S70	2017	0	79917	71558	66060	62140	58516	54718
Asthma Children	Detroit	S70	2017	10	76986	68193	62297	57371	53954	50538
Asthma Children	Detroit	S70	2017	20	69321	58533	51544	46792	42681	38918
Asthma Children	Detroit	S70	2017	30	57770	45526	38242	33993	30229	27125
Asthma Children	Detroit	S70	2017	40	43878	31270	25113	20500	17222	14655
Asthma Children	Detroit	S70	2017	50	27055	15314	9712	6348	4492	2792
Asthma Children	Detroit	S70	2017	60	7648	1769	572	191	139	35
Asthma Children	Detroit	S70	2017	70	503	17	0	0	0	0
Asthma Children	Detroit	S70	2017	80	0	0	0	0	0	0
Asthma Children	Detroit	S70	2017	90	0	0	0	0	0	0
Asthma Children	Detroit	S70	2017	100	0	0	0	0	0	0
Asthma Children	Detroit	S75	2015	0	77853	70222	64551	60129	56729	53504
Asthma Children	Detroit	S75	2015	10	74732	66823	60146	55602	51821	48873
Asthma Children	Detroit	S75	2015	20	67465	56885	50399	45318	41086	37947
Asthma Children	Detroit	S75	2015	30	56573	44346	37652	32605	28980	26258
Asthma Children	Detroit	S75	2015	40	43878	31235	24957	20621	17308	14499
Asthma Children	Detroit	S75	2015	50	28911	16615	10631	7249	4891	3208
Asthma Children	Detroit	S75	2015	60	11030	3243	798	277	52	35
Asthma Children	Detroit	S75	2015	70	1214	35	0	0	0	0
Asthma Children	Detroit	S75	2015	80	0	0	0	0	0	0
Asthma Children	Detroit	S75	2015	90	0	0	0	0	0	0
Asthma Children	Detroit	S75	2015	100	0	0	0	0	0	0
Asthma Children	Detroit	S75	2016	0	80871	72806	67187	62747	58741	55706
Asthma Children	Detroit	S75	2016	10	78061	68748	62817	58412	54163	51232
Asthma Children	Detroit	S75	2016	20	69962	59140	52671	47763	43722	40132
Asthma Children	Detroit	S75	2016	30	59504	47277	40097	35536	31825	28894
Asthma Children	Detroit	S75	2016	40	46861	34010	27680	23084	19442	16597
Asthma Children	Detroit	S75	2016	50	32744	20066	14169	10094	7353	5654

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Detroit	S75	2016	60	17673	7492	3261	1231	520	225
Asthma Children	Detroit	S75	2016	70	4544	520	104	17	0	0
Asthma Children	Detroit	S75	2016	80	69	0	0	0	0	0
Asthma Children	Detroit	S75	2016	90	0	0	0	0	0	0
Asthma Children	Detroit	S75	2016	100	0	0	0	0	0	0
Asthma Children	Detroit	S75	2017	0	79917	71558	66060	62140	58516	54718
Asthma Children	Detroit	S75	2017	10	76899	68037	62175	57284	53781	50243
Asthma Children	Detroit	S75	2017	20	69598	58689	51787	47121	42890	38883
Asthma Children	Detroit	S75	2017	30	58828	46306	38970	34548	30992	27801
Asthma Children	Detroit	S75	2017	40	45925	33229	26726	22130	18696	16233
Asthma Children	Detroit	S75	2017	50	30853	18991	12955	8828	6452	4717
Asthma Children	Detroit	S75	2017	60	13458	4683	1821	763	364	208
Asthma Children	Detroit	S75	2017	70	1682	87	0	0	0	0
Asthma Children	Detroit	S75	2017	80	52	0	0	0	0	0
Asthma Children	Detroit	S75	2017	90	0	0	0	0	0	0
Asthma Children	Detroit	S75	2017	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2015	0	100049	90817	84248	78616	73924	70432
Asthma Children	Philadelphia	S65	2015	10	97016	87172	79315	73575	69493	66198
Asthma Children	Philadelphia	S65	2015	20	87631	75146	66612	60872	56703	52709
Asthma Children	Philadelphia	S65	2015	30	73073	58428	50243	43848	39505	35620
Asthma Children	Philadelphia	S65	2015	40	50352	35314	27675	22284	18552	15605
Asthma Children	Philadelphia	S65	2015	50	20102	9058	4954	2794	1441	808
Asthma Children	Philadelphia	S65	2015	60	2248	218	44	0	0	0
Asthma Children	Philadelphia	S65	2015	70	44	0	0	0	0	0
Asthma Children	Philadelphia	S65	2015	80	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2015	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2015	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2016	0	98522	89180	82632	77241	72745	69144
Asthma Children	Philadelphia	S65	2016	10	95400	85666	78442	72331	67856	64233
Asthma Children	Philadelphia	S65	2016	20	85775	72854	64975	58973	54237	50767
Asthma Children	Philadelphia	S65	2016	30	69581	55590	47711	42036	38348	35030
Asthma Children	Philadelphia	S65	2016	40	47515	33110	25711	21040	16937	14230
Asthma Children	Philadelphia	S65	2016	50	18421	7923	3667	1724	851	327
Asthma Children	Philadelphia	S65	2016	60	2750	262	0	0	0	0
Asthma Children	Philadelphia	S65	2016	70	22	0	0	0	0	0
Asthma Children	Philadelphia	S65	2016	80	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2016	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2016	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2017	0	98958	89180	82501	76958	73051	69515
Asthma Children	Philadelphia	S65	2017	10	95728	85426	77743	72505	68140	64561
Asthma Children	Philadelphia	S65	2017	20	85688	72593	64408	58820	54477	50614
Asthma Children	Philadelphia	S65	2017	30	68620	53910	45812	40094	35947	32761
Asthma Children	Philadelphia	S65	2017	40	46052	30185	23528	18465	15191	12506
Asthma Children	Philadelphia	S65	2017	50	16937	6308	3012	1179	655	306
Asthma Children	Philadelphia	S65	2017	60	1964	218	0	0	0	0
Asthma Children	Philadelphia	S65	2017	70	65	0	0	0	0	0
Asthma Children	Philadelphia	S65	2017	80	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Philadelphia	S65	2017	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S65	2017	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2015	0	100049	90817	84248	78616	73924	70432
Asthma Children	Philadelphia	S70	2015	10	97125	87325	79533	73858	69668	66394
Asthma Children	Philadelphia	S70	2015	20	88635	75910	67791	61767	57402	53691
Asthma Children	Philadelphia	S70	2015	30	75212	60741	52491	46423	41578	37562
Asthma Children	Philadelphia	S70	2015	40	56114	40574	33001	27282	23397	19840
Asthma Children	Philadelphia	S70	2015	50	28897	15562	9953	6766	4845	3274
Asthma Children	Philadelphia	S70	2015	60	6264	1375	437	109	22	22
Asthma Children	Philadelphia	S70	2015	70	611	0	0	0	0	0
Asthma Children	Philadelphia	S70	2015	80	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2015	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2015	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2016	0	98522	89180	82632	77241	72745	69144
Asthma Children	Philadelphia	S70	2016	10	95510	85841	78682	72593	68009	64473
Asthma Children	Philadelphia	S70	2016	20	86561	73880	66285	60021	55437	51487
Asthma Children	Philadelphia	S70	2016	30	72374	57860	49806	44241	40290	37017
Asthma Children	Philadelphia	S70	2016	40	53189	38413	30578	25776	21717	18399
Asthma Children	Philadelphia	S70	2016	50	27326	14885	8425	5020	3318	2030
Asthma Children	Philadelphia	S70	2016	60	6504	1113	175	44	0	0
Asthma Children	Philadelphia	S70	2016	70	655	44	0	0	0	0
Asthma Children	Philadelphia	S70	2016	80	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2016	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2016	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2017	0	98958	89180	82501	76958	73051	69515
Asthma Children	Philadelphia	S70	2017	10	95946	85557	77896	72636	68424	64779
Asthma Children	Philadelphia	S70	2017	20	86517	73553	65368	59672	55481	51858
Asthma Children	Philadelphia	S70	2017	30	71567	56420	48584	42429	37890	34485
Asthma Children	Philadelphia	S70	2017	40	51400	35532	28177	22786	19490	16326
Asthma Children	Philadelphia	S70	2017	50	24248	12463	7224	3907	2401	1288
Asthma Children	Philadelphia	S70	2017	60	6024	1375	284	22	0	0
Asthma Children	Philadelphia	S70	2017	70	524	0	0	0	0	0
Asthma Children	Philadelphia	S70	2017	80	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2017	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S70	2017	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S75	2015	0	100049	90817	84248	78616	73924	70432
Asthma Children	Philadelphia	S75	2015	10	97147	87434	79642	73989	69733	66547
Asthma Children	Philadelphia	S75	2015	20	89638	76783	68642	62575	58078	54477
Asthma Children	Philadelphia	S75	2015	30	77569	62946	54739	48519	44154	39963
Asthma Children	Philadelphia	S75	2015	40	61461	46074	37824	31778	27588	24248
Asthma Children	Philadelphia	S75	2015	50	38261	23768	16850	12572	9800	7552
Asthma Children	Philadelphia	S75	2015	60	13859	4474	2095	939	393	196
Asthma Children	Philadelphia	S75	2015	70	2183	175	44	0	0	0
Asthma Children	Philadelphia	S75	2015	80	218	0	0	0	0	0
Asthma Children	Philadelphia	S75	2015	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S75	2015	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S75	2016	0	98522	89180	82632	77241	72745	69144

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Philadelphia	S75	2016	10	95619	85884	78529	72702	68184	64473
Asthma Children	Philadelphia	S75	2016	20	87085	75059	67027	61265	56441	52731
Asthma Children	Philadelphia	S75	2016	30	74950	60392	52120	46598	42386	38981
Asthma Children	Philadelphia	S75	2016	40	58275	43564	35380	30098	26082	23157
Asthma Children	Philadelphia	S75	2016	50	36798	23354	15976	11524	8272	6068
Asthma Children	Philadelphia	S75	2016	60	14078	4562	1375	480	196	87
Asthma Children	Philadelphia	S75	2016	70	2270	196	0	0	0	0
Asthma Children	Philadelphia	S75	2016	80	65	0	0	0	0	0
Asthma Children	Philadelphia	S75	2016	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S75	2016	100	0	0	0	0	0	0
Asthma Children	Philadelphia	S75	2017	0	98958	89180	82501	76958	73051	69515
Asthma Children	Philadelphia	S75	2017	10	95946	85710	78049	72767	68577	64779
Asthma Children	Philadelphia	S75	2017	20	87434	74208	65914	60501	56398	52578
Asthma Children	Philadelphia	S75	2017	30	74077	58886	50876	44852	40050	36624
Asthma Children	Philadelphia	S75	2017	40	55961	40552	32957	27304	23463	20342
Asthma Children	Philadelphia	S75	2017	50	33088	19512	13205	9080	6155	4212
Asthma Children	Philadelphia	S75	2017	60	13292	4103	1724	567	262	44
Asthma Children	Philadelphia	S75	2017	70	1942	131	22	0	0	0
Asthma Children	Philadelphia	S75	2017	80	87	0	0	0	0	0
Asthma Children	Philadelphia	S75	2017	90	0	0	0	0	0	0
Asthma Children	Philadelphia	S75	2017	100	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2015	0	58411	53655	50924	48659	46819	44993
Asthma Children	Phoenix	S65	2015	10	56727	51745	48532	46196	44158	42488
Asthma Children	Phoenix	S65	2015	20	51886	46253	42502	39658	36742	34548
Asthma Children	Phoenix	S65	2015	30	44923	37520	33260	30274	27641	25844
Asthma Children	Phoenix	S65	2015	40	34619	26778	22306	19107	17126	15243
Asthma Children	Phoenix	S65	2015	50	18541	10643	7034	4897	3468	2633
Asthma Children	Phoenix	S65	2015	60	1500	226	14	0	0	0
Asthma Children	Phoenix	S65	2015	70	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2015	80	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2015	90	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2015	100	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2016	0	56995	52580	49848	47584	45701	44130
Asthma Children	Phoenix	S65	2016	10	55127	50329	47456	45163	43026	41512
Asthma Children	Phoenix	S65	2016	20	50442	44654	41186	38384	36360	34548
Asthma Children	Phoenix	S65	2016	30	43380	37266	33133	30161	27910	26014
Asthma Children	Phoenix	S65	2016	40	33289	25518	21414	18725	16630	15073
Asthma Children	Phoenix	S65	2016	50	15059	7982	5081	3354	2364	1571
Asthma Children	Phoenix	S65	2016	60	778	71	0	0	0	0
Asthma Children	Phoenix	S65	2016	70	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2016	80	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2016	90	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2016	100	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2017	0	58340	53684	50457	48121	46437	44894
Asthma Children	Phoenix	S65	2017	10	56925	51518	48150	45645	44031	42375
Asthma Children	Phoenix	S65	2017	20	52325	46338	42460	39488	37252	35341
Asthma Children	Phoenix	S65	2017	30	45772	38058	33855	30939	28901	27160

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Phoenix	S65	2017	40	35907	27953	24146	21103	18697	16956
Asthma Children	Phoenix	S65	2017	50	19348	12002	8464	6199	4671	3637
Asthma Children	Phoenix	S65	2017	60	2788	481	127	42	0	0
Asthma Children	Phoenix	S65	2017	70	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2017	80	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2017	90	0	0	0	0	0	0
Asthma Children	Phoenix	S65	2017	100	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2015	0	58411	53655	50924	48659	46819	44993
Asthma Children	Phoenix	S70	2015	10	56826	51801	48659	46281	44257	42573
Asthma Children	Phoenix	S70	2015	20	52339	46819	42984	40210	37520	35256
Asthma Children	Phoenix	S70	2015	30	46310	39148	34789	31788	29085	27203
Asthma Children	Phoenix	S70	2015	40	37351	29566	24896	22192	19716	17946
Asthma Children	Phoenix	S70	2015	50	24995	16588	12738	10063	8209	6610
Asthma Children	Phoenix	S70	2015	60	7034	2406	1203	665	354	170
Asthma Children	Phoenix	S70	2015	70	127	0	0	0	0	0
Asthma Children	Phoenix	S70	2015	80	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2015	90	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2015	100	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2016	0	56995	52580	49848	47584	45701	44130
Asthma Children	Phoenix	S70	2016	10	55269	50414	47612	45347	43224	41639
Asthma Children	Phoenix	S70	2016	20	50782	45305	41823	38922	36983	35100
Asthma Children	Phoenix	S70	2016	30	44427	38440	34435	31519	29283	27089
Asthma Children	Phoenix	S70	2016	40	35978	28717	24542	21584	19475	17777
Asthma Children	Phoenix	S70	2016	50	22192	14734	10714	8591	6680	5308
Asthma Children	Phoenix	S70	2016	60	5336	1444	623	212	127	85
Asthma Children	Phoenix	S70	2016	70	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2016	80	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2016	90	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2016	100	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2017	0	58340	53684	50457	48121	46437	44894
Asthma Children	Phoenix	S70	2017	10	57010	51674	48362	45871	44173	42559
Asthma Children	Phoenix	S70	2017	20	52806	46947	43040	40210	38030	35949
Asthma Children	Phoenix	S70	2017	30	46961	39445	35270	32425	30331	28505
Asthma Children	Phoenix	S70	2017	40	38624	30868	26877	23877	21640	19857
Asthma Children	Phoenix	S70	2017	50	26453	18343	14351	11507	9822	8308
Asthma Children	Phoenix	S70	2017	60	9143	3977	2109	1033	609	382
Asthma Children	Phoenix	S70	2017	70	552	71	0	0	0	0
Asthma Children	Phoenix	S70	2017	80	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2017	90	0	0	0	0	0	0
Asthma Children	Phoenix	S70	2017	100	0	0	0	0	0	0
Asthma Children	Phoenix	S75	2015	0	58411	53655	50924	48659	46819	44993
Asthma Children	Phoenix	S75	2015	10	56769	51730	48518	46267	44243	42602
Asthma Children	Phoenix	S75	2015	20	52495	47046	43295	40549	37818	35511
Asthma Children	Phoenix	S75	2015	30	47145	40139	35681	32737	29977	27995
Asthma Children	Phoenix	S75	2015	40	39191	31307	26637	23877	21626	19659
Asthma Children	Phoenix	S75	2015	50	28844	20480	16404	13814	11450	9780
Asthma Children	Phoenix	S75	2015	60	14451	7402	4161	2774	1840	1161

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Phoenix	S75	2015	70	1302	170	28	0	0	0
Asthma Children	Phoenix	S75	2015	80	14	0	0	0	0	0
Asthma Children	Phoenix	S75	2015	90	0	0	0	0	0	0
Asthma Children	Phoenix	S75	2015	100	0	0	0	0	0	0
Asthma Children	Phoenix	S75	2016	0	56995	52580	49848	47584	45701	44130
Asthma Children	Phoenix	S75	2016	10	55269	50428	47555	45234	43054	41526
Asthma Children	Phoenix	S75	2016	20	51108	45489	42078	39191	37195	35270
Asthma Children	Phoenix	S75	2016	30	45078	39077	35185	32524	30090	27981
Asthma Children	Phoenix	S75	2016	40	37605	30628	26495	23466	21202	19447
Asthma Children	Phoenix	S75	2016	50	26835	19135	14847	12172	10091	8733
Asthma Children	Phoenix	S75	2016	60	11634	5364	2802	1755	1019	580
Asthma Children	Phoenix	S75	2016	70	807	142	14	0	0	0
Asthma Children	Phoenix	S75	2016	80	0	0	0	0	0	0
Asthma Children	Phoenix	S75	2016	90	0	0	0	0	0	0
Asthma Children	Phoenix	S75	2016	100	0	0	0	0	0	0
Asthma Children	Phoenix	S75	2017	0	58340	53684	50457	48121	46437	44894
Asthma Children	Phoenix	S75	2017	10	56953	51773	48291	45942	44215	42403
Asthma Children	Phoenix	S75	2017	20	53033	47272	43338	40323	38172	36204
Asthma Children	Phoenix	S75	2017	30	47470	40479	36233	33331	30996	29184
Asthma Children	Phoenix	S75	2017	40	40507	32425	28618	25787	23636	21810
Asthma Children	Phoenix	S75	2017	50	30670	22504	18145	15187	13064	11620
Asthma Children	Phoenix	S75	2017	60	15668	8931	5789	3595	2562	1897
Asthma Children	Phoenix	S75	2017	70	3128	637	113	99	14	0
Asthma Children	Phoenix	S75	2017	80	156	0	0	0	0	0
Asthma Children	Phoenix	S75	2017	90	0	0	0	0	0	0
Asthma Children	Phoenix	S75	2017	100	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2015	0	30691	27958	26095	24744	23595	22454
Asthma Children	Sacramento	S65	2015	10	29286	26297	24239	22733	21382	20334
Asthma Children	Sacramento	S65	2015	20	25536	21941	19620	17935	16600	15637
Asthma Children	Sacramento	S65	2015	30	20691	16569	14006	12384	11258	10202
Asthma Children	Sacramento	S65	2015	40	12904	8735	6491	5124	4185	3540
Asthma Children	Sacramento	S65	2015	50	3362	1017	435	163	70	31
Asthma Children	Sacramento	S65	2015	60	217	16	8	0	0	0
Asthma Children	Sacramento	S65	2015	70	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2015	80	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2015	90	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2015	100	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2016	0	31786	28968	27198	25738	24597	23463
Asthma Children	Sacramento	S65	2016	10	30280	27190	25101	23587	22267	21351
Asthma Children	Sacramento	S65	2016	20	26281	22461	20233	18548	17166	16126
Asthma Children	Sacramento	S65	2016	30	20668	16763	14185	12741	11421	10489
Asthma Children	Sacramento	S65	2016	40	13137	8851	6755	5443	4270	3463
Asthma Children	Sacramento	S65	2016	50	4262	1770	784	342	155	78
Asthma Children	Sacramento	S65	2016	60	295	23	0	0	0	0
Asthma Children	Sacramento	S65	2016	70	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2016	80	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2016	90	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Sacramento	S65	2016	100	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2017	0	30598	27990	26118	24698	23448	22500
Asthma Children	Sacramento	S65	2017	10	29340	26281	24302	22632	21320	20412
Asthma Children	Sacramento	S65	2017	20	25893	22190	19721	18215	16918	15885
Asthma Children	Sacramento	S65	2017	30	20497	16219	13773	12423	11328	10350
Asthma Children	Sacramento	S65	2017	40	12702	8595	6530	5132	4177	3432
Asthma Children	Sacramento	S65	2017	50	3253	1002	318	148	85	47
Asthma Children	Sacramento	S65	2017	60	124	0	0	0	0	0
Asthma Children	Sacramento	S65	2017	70	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2017	80	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2017	90	0	0	0	0	0	0
Asthma Children	Sacramento	S65	2017	100	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2015	0	30691	27958	26095	24744	23595	22454
Asthma Children	Sacramento	S70	2015	10	29379	26390	24356	22920	21561	20458
Asthma Children	Sacramento	S70	2015	20	26080	22454	20125	18463	17112	16126
Asthma Children	Sacramento	S70	2015	30	21910	17834	15249	13502	12399	11452
Asthma Children	Sacramento	S70	2015	40	15753	11320	9092	7648	6475	5582
Asthma Children	Sacramento	S70	2015	50	7221	3781	2337	1374	893	598
Asthma Children	Sacramento	S70	2015	60	1157	179	39	0	0	0
Asthma Children	Sacramento	S70	2015	70	70	8	0	0	0	0
Asthma Children	Sacramento	S70	2015	80	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2015	90	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2015	100	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2016	0	31786	28968	27198	25738	24597	23463
Asthma Children	Sacramento	S70	2016	10	30389	27291	25194	23727	22430	21491
Asthma Children	Sacramento	S70	2016	20	26786	22989	20839	19146	17741	16693
Asthma Children	Sacramento	S70	2016	30	21864	17966	15668	13874	12539	11553
Asthma Children	Sacramento	S70	2016	40	15994	11592	9200	7710	6467	5427
Asthma Children	Sacramento	S70	2016	50	8269	4705	2958	1918	1343	924
Asthma Children	Sacramento	S70	2016	60	1871	396	132	70	23	0
Asthma Children	Sacramento	S70	2016	70	155	8	0	0	0	0
Asthma Children	Sacramento	S70	2016	80	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2016	90	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2016	100	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2017	0	30598	27990	26118	24698	23448	22500
Asthma Children	Sacramento	S70	2017	10	29449	26367	24434	22811	21483	20513
Asthma Children	Sacramento	S70	2017	20	26421	22609	20280	18688	17547	16491
Asthma Children	Sacramento	S70	2017	30	21763	17539	14860	13533	12492	11514
Asthma Children	Sacramento	S70	2017	40	15621	11196	8975	7469	6328	5551
Asthma Children	Sacramento	S70	2017	50	7283	3750	2182	1312	939	668
Asthma Children	Sacramento	S70	2017	60	1522	272	31	0	0	0
Asthma Children	Sacramento	S70	2017	70	54	0	0	0	0	0
Asthma Children	Sacramento	S70	2017	80	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2017	90	0	0	0	0	0	0
Asthma Children	Sacramento	S70	2017	100	0	0	0	0	0	0
Asthma Children	Sacramento	S75	2015	0	30691	27958	26095	24744	23595	22454
Asthma Children	Sacramento	S75	2015	10	29426	26468	24418	22958	21654	20536

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	Sacramento	S75	2015	20	26359	22811	20497	18805	17485	16336
Asthma Children	Sacramento	S75	2015	30	22578	18587	16041	14286	13121	12190
Asthma Children	Sacramento	S75	2015	40	17306	13044	10575	9006	7811	7050
Asthma Children	Sacramento	S75	2015	50	10000	5940	4162	2943	2174	1685
Asthma Children	Sacramento	S75	2015	60	3059	846	318	93	39	16
Asthma Children	Sacramento	S75	2015	70	427	23	8	0	0	0
Asthma Children	Sacramento	S75	2015	80	23	0	0	0	0	0
Asthma Children	Sacramento	S75	2015	90	0	0	0	0	0	0
Asthma Children	Sacramento	S75	2015	100	0	0	0	0	0	0
Asthma Children	Sacramento	S75	2016	0	31786	28968	27198	25738	24597	23463
Asthma Children	Sacramento	S75	2016	10	30435	27337	25280	23836	22531	21553
Asthma Children	Sacramento	S75	2016	20	27066	23370	21103	19534	18044	16965
Asthma Children	Sacramento	S75	2016	30	22586	18696	16413	14527	13207	12151
Asthma Children	Sacramento	S75	2016	40	17345	13207	10808	9270	7950	6863
Asthma Children	Sacramento	S75	2016	50	10947	6778	4860	3540	2717	2065
Asthma Children	Sacramento	S75	2016	60	4301	1739	769	349	163	78
Asthma Children	Sacramento	S75	2016	70	675	78	23	0	0	0
Asthma Children	Sacramento	S75	2016	80	23	0	0	0	0	0
Asthma Children	Sacramento	S75	2016	90	0	0	0	0	0	0
Asthma Children	Sacramento	S75	2016	100	0	0	0	0	0	0
Asthma Children	Sacramento	S75	2017	0	30598	27990	26118	24698	23448	22500
Asthma Children	Sacramento	S75	2017	10	29496	26452	24503	22873	21522	20606
Asthma Children	Sacramento	S75	2017	20	26646	22920	20629	19030	17850	16801
Asthma Children	Sacramento	S75	2017	30	22586	18455	15668	14247	13082	12065
Asthma Children	Sacramento	S75	2017	40	17260	12772	10536	9061	7756	6724
Asthma Children	Sacramento	S75	2017	50	10388	6242	4363	3106	2236	1724
Asthma Children	Sacramento	S75	2017	60	3253	978	357	171	70	16
Asthma Children	Sacramento	S75	2017	70	404	8	0	0	0	0
Asthma Children	Sacramento	S75	2017	80	23	0	0	0	0	0
Asthma Children	Sacramento	S75	2017	90	0	0	0	0	0	0
Asthma Children	Sacramento	S75	2017	100	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2015	0	37965	34049	31527	29587	27775	26391
Asthma Children	St. Louis	S65	2015	10	36016	31764	29150	27101	25161	23586
Asthma Children	St. Louis	S65	2015	20	31490	26200	23049	20690	18942	17557
Asthma Children	St. Louis	S65	2015	30	24642	19333	16292	14288	12594	11247
Asthma Children	St. Louis	S65	2015	40	16647	11256	8360	6584	5400	4471
Asthma Children	St. Louis	S65	2015	50	6793	2477	1093	474	228	91
Asthma Children	St. Louis	S65	2015	60	328	9	0	0	0	0
Asthma Children	St. Louis	S65	2015	70	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2015	80	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2015	90	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2015	100	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2016	0	36882	32765	30461	28595	27037	25826
Asthma Children	St. Louis	S65	2016	10	35133	30880	28367	26327	24697	23440
Asthma Children	St. Louis	S65	2016	20	30871	26027	23194	21045	19361	17913
Asthma Children	St. Louis	S65	2016	30	25025	19743	16665	14525	13050	11702
Asthma Children	St. Louis	S65	2016	40	17785	12230	9243	7549	6211	5409

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					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	St. Louis	S65	2016	50	8779	4216	2176	1166	729	483
Asthma Children	St. Louis	S65	2016	60	1657	173	36	0	0	0
Asthma Children	St. Louis	S65	2016	70	27	0	0	0	0	0
Asthma Children	St. Louis	S65	2016	80	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2016	90	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2016	100	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2017	0	37656	33967	31427	29505	27966	26864
Asthma Children	St. Louis	S65	2017	10	36344	32419	29596	27647	25926	24533
Asthma Children	St. Louis	S65	2017	20	32501	27529	24497	22329	20490	19087
Asthma Children	St. Louis	S65	2017	30	26737	21073	18013	16292	14552	13259
Asthma Children	St. Louis	S65	2017	40	19370	13897	10682	8597	7249	6101
Asthma Children	St. Louis	S65	2017	50	8251	3643	1849	965	483	310
Asthma Children	St. Louis	S65	2017	60	510	36	9	0	0	0
Asthma Children	St. Louis	S65	2017	70	9	0	0	0	0	0
Asthma Children	St. Louis	S65	2017	80	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2017	90	0	0	0	0	0	0
Asthma Children	St. Louis	S65	2017	100	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2015	0	37965	34049	31527	29587	27775	26391
Asthma Children	St. Louis	S70	2015	10	36135	31873	29305	27165	25316	23723
Asthma Children	St. Louis	S70	2015	20	32000	26855	23577	21291	19442	17913
Asthma Children	St. Louis	S70	2015	30	25808	20399	17230	15317	13696	12230
Asthma Children	St. Louis	S70	2015	40	18741	13268	10409	8469	7121	6065
Asthma Children	St. Louis	S70	2015	50	10582	5309	3114	2013	1229	738
Asthma Children	St. Louis	S70	2015	60	2195	337	82	0	0	0
Asthma Children	St. Louis	S70	2015	70	18	0	0	0	0	0
Asthma Children	St. Louis	S70	2015	80	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2015	90	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2015	100	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2016	0	36882	32765	30461	28595	27037	25826
Asthma Children	St. Louis	S70	2016	10	35242	30962	28504	26445	24879	23531
Asthma Children	St. Louis	S70	2016	20	31390	26582	23795	21519	19861	18368
Asthma Children	St. Louis	S70	2016	30	26136	20936	17785	15691	13951	12676
Asthma Children	St. Louis	S70	2016	40	19697	14234	11128	9407	8014	6939
Asthma Children	St. Louis	S70	2016	50	12121	6967	4562	3096	2067	1457
Asthma Children	St. Louis	S70	2016	60	4927	1211	455	155	64	9
Asthma Children	St. Louis	S70	2016	70	437	0	0	0	0	0
Asthma Children	St. Louis	S70	2016	80	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2016	90	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2016	100	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2017	0	37656	33967	31427	29505	27966	26864
Asthma Children	St. Louis	S70	2017	10	36454	32556	29769	27748	26063	24688
Asthma Children	St. Louis	S70	2017	20	32993	28230	25180	22912	21073	19643
Asthma Children	St. Louis	S70	2017	30	27839	22184	19087	17129	15572	14243
Asthma Children	St. Louis	S70	2017	40	21482	16000	12959	10855	9307	8050
Asthma Children	St. Louis	S70	2017	50	12631	7130	4617	3269	2340	1748
Asthma Children	St. Louis	S70	2017	60	2969	592	173	64	36	9
Asthma Children	St. Louis	S70	2017	70	118	9	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Children	St. Louis	S70	2017	80	9	0	0	0	0	0
Asthma Children	St. Louis	S70	2017	90	0	0	0	0	0	0
Asthma Children	St. Louis	S70	2017	100	0	0	0	0	0	0
Asthma Children	St. Louis	S75	2015	0	37965	34049	31527	29587	27775	26391
Asthma Children	St. Louis	S75	2015	10	36153	31946	29350	27147	25371	23750
Asthma Children	St. Louis	S75	2015	20	32201	27165	23914	21619	19634	18213
Asthma Children	St. Louis	S75	2015	30	26436	20927	17822	15909	14370	12767
Asthma Children	St. Louis	S75	2015	40	19980	14716	11629	9535	8241	7012
Asthma Children	St. Louis	S75	2015	50	12968	7495	4954	3506	2359	1712
Asthma Children	St. Louis	S75	2015	60	4808	1402	492	155	46	9
Asthma Children	St. Louis	S75	2015	70	364	0	0	0	0	0
Asthma Children	St. Louis	S75	2015	80	0	0	0	0	0	0
Asthma Children	St. Louis	S75	2015	90	0	0	0	0	0	0
Asthma Children	St. Louis	S75	2015	100	0	0	0	0	0	0
Asthma Children	St. Louis	S75	2016	0	36882	32765	30461	28595	27037	25826
Asthma Children	St. Louis	S75	2016	10	35315	30980	28522	26500	24888	23622
Asthma Children	St. Louis	S75	2016	20	31645	26919	24069	21892	20135	18723
Asthma Children	St. Louis	S75	2016	30	26855	21637	18477	16355	14416	13177
Asthma Children	St. Louis	S75	2016	40	21027	15426	12303	10345	9125	8050
Asthma Children	St. Louis	S75	2016	50	14142	8788	6138	4526	3379	2586
Asthma Children	St. Louis	S75	2016	60	7212	2705	1102	619	346	137
Asthma Children	St. Louis	S75	2016	70	1767	191	27	0	0	0
Asthma Children	St. Louis	S75	2016	80	82	0	0	0	0	0
Asthma Children	St. Louis	S75	2016	90	0	0	0	0	0	0
Asthma Children	St. Louis	S75	2016	100	0	0	0	0	0	0
Asthma Children	St. Louis	S75	2017	0	37656	33967	31427	29505	27966	26864
Asthma Children	St. Louis	S75	2017	10	36454	32592	29806	27729	26145	24715
Asthma Children	St. Louis	S75	2017	20	33266	28494	25517	23203	21400	19898
Asthma Children	St. Louis	S75	2017	30	28540	22967	19616	17621	16182	14853
Asthma Children	St. Louis	S75	2017	40	22812	17239	14088	12157	10491	9234
Asthma Children	St. Louis	S75	2017	50	15181	9607	6812	5081	3943	2969
Asthma Children	St. Louis	S75	2017	60	5719	1931	856	301	155	100
Asthma Children	St. Louis	S75	2017	70	610	55	18	0	0	0
Asthma Children	St. Louis	S75	2017	80	18	0	0	0	0	0
Asthma Children	St. Louis	S75	2017	90	0	0	0	0	0	0
Asthma Children	St. Louis	S75	2017	100	0	0	0	0	0	0
All Adults	Atlanta	S65	2015	0	1444098	1258787	1143840	1058756	992690	936765
All Adults	Atlanta	S65	2015	10	1250476	1029174	893026	791391	713280	645875
All Adults	Atlanta	S65	2015	20	845906	585443	443590	360197	299694	252363
All Adults	Atlanta	S65	2015	30	523391	319838	230388	178478	139388	111848
All Adults	Atlanta	S65	2015	40	282368	140022	81703	49867	31836	20074
All Adults	Atlanta	S65	2015	50	72265	15073	3663	986	141	0
All Adults	Atlanta	S65	2015	60	5564	0	0	0	0	0
All Adults	Atlanta	S65	2015	70	211	0	0	0	0	0
All Adults	Atlanta	S65	2015	80	0	0	0	0	0	0
All Adults	Atlanta	S65	2015	90	0	0	0	0	0	0
All Adults	Atlanta	S65	2016	0	1444309	1253434	1141445	1056925	990647	933878

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Atlanta	S65	2016	10	1277311	1066293	940498	843089	769556	704335
All Adults	Atlanta	S65	2016	20	912325	650946	510502	413304	346181	293637
All Adults	Atlanta	S65	2016	30	591148	379707	281170	222218	182282	151291
All Adults	Atlanta	S65	2016	40	336390	181437	113398	76843	54234	37682
All Adults	Atlanta	S65	2016	50	93254	20919	5916	1197	493	141
All Adults	Atlanta	S65	2016	60	10706	352	0	0	0	0
All Adults	Atlanta	S65	2016	70	704	0	0	0	0	0
All Adults	Atlanta	S65	2016	80	0	0	0	0	0	0
All Adults	Atlanta	S65	2016	90	0	0	0	0	0	0
All Adults	Atlanta	S65	2017	0	1447972	1257872	1140248	1056854	990154	935568
All Adults	Atlanta	S65	2017	10	1256111	1034034	900210	806111	722577	650665
All Adults	Atlanta	S65	2017	20	844568	581569	446055	357379	296877	251729
All Adults	Atlanta	S65	2017	30	521560	320120	236868	184254	147910	121216
All Adults	Atlanta	S65	2017	40	269972	128189	76491	46345	29934	19017
All Adults	Atlanta	S65	2017	50	45218	7536	1761	352	70	0
All Adults	Atlanta	S65	2017	60	1057	0	0	0	0	0
All Adults	Atlanta	S65	2017	70	0	0	0	0	0	0
All Adults	Atlanta	S65	2017	80	0	0	0	0	0	0
All Adults	Atlanta	S65	2017	90	0	0	0	0	0	0
All Adults	Atlanta	S70	2015	0	1444098	1258787	1143840	1058756	992690	936765
All Adults	Atlanta	S70	2015	10	1262379	1040162	907888	807590	731240	663554
All Adults	Atlanta	S70	2015	20	894224	627281	478596	389356	325051	274831
All Adults	Atlanta	S70	2015	30	567835	353224	254194	200665	160166	128893
All Adults	Atlanta	S70	2015	40	348998	185944	119737	81421	56347	39795
All Adults	Atlanta	S70	2015	50	140022	43950	17397	5705	2043	493
All Adults	Atlanta	S70	2015	60	20003	1972	211	0	0	0
All Adults	Atlanta	S70	2015	70	2254	0	0	0	0	0
All Adults	Atlanta	S70	2015	80	0	0	0	0	0	0
All Adults	Atlanta	S70	2015	90	0	0	0	0	0	0
All Adults	Atlanta	S70	2016	0	1444309	1253434	1141445	1056925	990647	933878
All Adults	Atlanta	S70	2016	10	1290834	1078759	955078	861120	785615	722718
All Adults	Atlanta	S70	2016	20	956275	697925	549804	451760	379143	323783
All Adults	Atlanta	S70	2016	30	638761	415205	308851	245320	201792	168406
All Adults	Atlanta	S70	2016	40	405767	231656	155869	111708	85084	64940
All Adults	Atlanta	S70	2016	50	168547	60784	25779	10917	3663	1479
All Adults	Atlanta	S70	2016	60	34160	3592	282	141	0	0
All Adults	Atlanta	S70	2016	70	5001	0	0	0	0	0
All Adults	Atlanta	S70	2016	80	352	0	0	0	0	0
All Adults	Atlanta	S70	2016	90	0	0	0	0	0	0
All Adults	Atlanta	S70	2017	0	1447972	1257872	1140248	1056854	990154	935568
All Adults	Atlanta	S70	2017	10	1267098	1048332	915494	824847	741242	672499
All Adults	Atlanta	S70	2017	20	885560	621998	481061	386821	322163	272507
All Adults	Atlanta	S70	2017	30	565299	351956	261308	204187	164533	138261
All Adults	Atlanta	S70	2017	40	331178	174464	112694	76209	53389	37752
All Adults	Atlanta	S70	2017	50	106566	26624	9649	3522	1127	423
All Adults	Atlanta	S70	2017	60	9790	211	70	0	0	0
All Adults	Atlanta	S70	2017	70	282	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Atlanta	S70	2017	80	0	0	0	0	0	0
All Adults	Atlanta	S70	2017	90	0	0	0	0	0	0
All Adults	Atlanta	S75	2015	0	1444098	1258787	1143840	1058756	992690	936765
All Adults	Atlanta	S75	2015	10	1269634	1047980	918030	816395	740608	674189
All Adults	Atlanta	S75	2015	20	929088	662286	510079	414853	345124	292088
All Adults	Atlanta	S75	2015	30	610517	383651	277156	217217	173971	143332
All Adults	Atlanta	S75	2015	40	397386	221654	148544	104312	77688	57333
All Adults	Atlanta	S75	2015	50	207286	85577	40147	20285	9931	4649
All Adults	Atlanta	S75	2015	60	55220	9368	1479	352	141	0
All Adults	Atlanta	S75	2015	70	7396	282	0	0	0	0
All Adults	Atlanta	S75	2015	80	1057	0	0	0	0	0
All Adults	Atlanta	S75	2015	90	0	0	0	0	0	0
All Adults	Atlanta	S75	2016	0	1444309	1253434	1141445	1056925	990647	933878
All Adults	Atlanta	S75	2016	10	1297737	1086648	961699	870487	796039	733987
All Adults	Atlanta	S75	2016	20	989943	732720	588049	485287	408796	348716
All Adults	Atlanta	S75	2016	30	680951	443661	334629	263633	217499	183197
All Adults	Atlanta	S75	2016	40	455775	271521	189677	140867	109947	84731
All Adults	Atlanta	S75	2016	50	240530	107341	55502	29652	15777	8029
All Adults	Atlanta	S75	2016	60	75153	15214	3522	563	141	70
All Adults	Atlanta	S75	2016	70	15495	916	0	0	0	0
All Adults	Atlanta	S75	2016	80	2817	0	0	0	0	0
All Adults	Atlanta	S75	2016	90	211	0	0	0	0	0
All Adults	Atlanta	S75	2017	0	1447972	1257872	1140248	1056854	990154	935568
All Adults	Atlanta	S75	2017	10	1274353	1055516	925073	834566	750046	684332
All Adults	Atlanta	S75	2017	20	917607	655524	510150	414219	343011	291735
All Adults	Atlanta	S75	2017	30	604812	380059	280818	219119	179183	149601
All Adults	Atlanta	S75	2017	40	382595	212920	145868	100931	75434	55995
All Adults	Atlanta	S75	2017	50	167984	60291	25004	12608	7184	3240
All Adults	Atlanta	S75	2017	60	33456	2888	211	0	0	0
All Adults	Atlanta	S75	2017	70	3099	70	0	0	0	0
All Adults	Atlanta	S75	2017	80	0	0	0	0	0	0
All Adults	Atlanta	S75	2017	90	0	0	0	0	0	0
All Adults	Boston	S65	2015	0	1850655	1592858	1438181	1324300	1230183	1160719
All Adults	Boston	S65	2015	10	1661832	1370968	1193299	1069439	961624	873475
All Adults	Boston	S65	2015	20	1148099	795108	600318	478709	398190	333325
All Adults	Boston	S65	2015	30	699132	430672	313269	244295	194399	159765
All Adults	Boston	S65	2015	40	359447	173462	97151	56745	36199	23481
All Adults	Boston	S65	2015	50	76214	14675	2152	1076	391	98
All Adults	Boston	S65	2015	60	8707	98	0	0	0	0
All Adults	Boston	S65	2015	70	1174	0	0	0	0	0
All Adults	Boston	S65	2015	80	196	0	0	0	0	0
All Adults	Boston	S65	2015	90	0	0	0	0	0	0
All Adults	Boston	S65	2016	0	1865134	1607632	1447964	1340345	1253076	1178330
All Adults	Boston	S65	2016	10	1689128	1394253	1216877	1087441	986279	895977
All Adults	Boston	S65	2016	20	1166883	813501	616461	491036	404647	341446
All Adults	Boston	S65	2016	30	701969	434389	315226	246447	203302	169060
All Adults	Boston	S65	2016	40	378819	186670	113098	72007	44711	30231

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Boston	S65	2016	50	101553	19469	4500	1174	294	0
All Adults	Boston	S65	2016	60	7533	98	98	0	0	0
All Adults	Boston	S65	2016	70	685	0	0	0	0	0
All Adults	Boston	S65	2016	80	0	0	0	0	0	0
All Adults	Boston	S65	2016	90	0	0	0	0	0	0
All Adults	Boston	S65	2017	0	1856329	1605871	1448453	1339367	1248478	1172460
All Adults	Boston	S65	2017	10	1682867	1396698	1219323	1092430	987062	896075
All Adults	Boston	S65	2017	20	1182635	811251	617341	494460	407582	335086
All Adults	Boston	S65	2017	30	715373	438694	318846	247915	195573	155754
All Adults	Boston	S65	2017	40	392614	189312	110848	67213	42852	27981
All Adults	Boston	S65	2017	50	135502	26024	6457	1663	587	98
All Adults	Boston	S65	2017	60	15360	978	98	0	0	0
All Adults	Boston	S65	2017	70	1468	0	0	0	0	0
All Adults	Boston	S65	2017	80	0	0	0	0	0	0
All Adults	Boston	S65	2017	90	0	0	0	0	0	0
All Adults	Boston	S70	2015	0	1850655	1592858	1438181	1324300	1230183	1160719
All Adults	Boston	S70	2015	10	1662713	1371555	1191929	1069830	964559	875725
All Adults	Boston	S70	2015	20	1173144	816143	621744	497493	411300	345848
All Adults	Boston	S70	2015	30	730244	448967	324129	249969	200367	164559
All Adults	Boston	S70	2015	40	408463	206726	124251	79149	51755	35612
All Adults	Boston	S70	2015	50	122490	31601	7925	2152	881	196
All Adults	Boston	S70	2015	60	19274	1076	196	98	0	0
All Adults	Boston	S70	2015	70	2152	0	0	0	0	0
All Adults	Boston	S70	2015	80	294	0	0	0	0	0
All Adults	Boston	S70	2015	90	0	0	0	0	0	0
All Adults	Boston	S70	2016	0	1865134	1607632	1447964	1340345	1253076	1178330
All Adults	Boston	S70	2016	10	1689911	1397188	1219714	1091061	985594	896662
All Adults	Boston	S70	2016	20	1196429	838352	642290	512071	423236	355045
All Adults	Boston	S70	2016	30	738364	450826	327846	257307	209074	174930
All Adults	Boston	S70	2016	40	425388	219738	140100	92552	63202	45004
All Adults	Boston	S70	2016	50	159570	44026	15654	6261	2837	1370
All Adults	Boston	S70	2016	60	23383	1468	98	0	0	0
All Adults	Boston	S70	2016	70	2739	98	98	0	0	0
All Adults	Boston	S70	2016	80	98	0	0	0	0	0
All Adults	Boston	S70	2016	90	0	0	0	0	0	0
All Adults	Boston	S70	2017	0	1856329	1605871	1448453	1339367	1248478	1172460
All Adults	Boston	S70	2017	10	1684237	1396601	1221182	1093702	987649	896662
All Adults	Boston	S70	2017	20	1209833	837080	639550	511484	421768	347805
All Adults	Boston	S70	2017	30	748441	455913	332347	254568	202813	161135
All Adults	Boston	S70	2017	40	440846	226782	137557	89226	60267	40308
All Adults	Boston	S70	2017	50	188235	48331	14675	4794	1859	391
All Adults	Boston	S70	2017	60	48429	4794	98	0	0	0
All Adults	Boston	S70	2017	70	5283	196	0	0	0	0
All Adults	Boston	S70	2017	80	489	0	0	0	0	0
All Adults	Boston	S70	2017	90	0	0	0	0	0	0
All Adults	Boston	S75	2015	0	1850655	1592858	1438181	1324300	1230183	1160719
All Adults	Boston	S75	2015	10	1659582	1368130	1187820	1064351	961429	871029

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Boston	S75	2015	20	1183319	827101	630451	503755	415996	350642
All Adults	Boston	S75	2015	30	742082	455815	328140	251339	201443	164364
All Adults	Boston	S75	2015	40	426954	220619	135209	88150	59288	40993
All Adults	Boston	S75	2015	50	152819	44124	14480	4598	2152	587
All Adults	Boston	S75	2015	60	31014	3131	489	391	0	0
All Adults	Boston	S75	2015	70	4305	98	0	0	0	0
All Adults	Boston	S75	2015	80	489	0	0	0	0	0
All Adults	Boston	S75	2015	90	0	0	0	0	0	0
All Adults	Boston	S75	2016	0	1865134	1607632	1447964	1340345	1253076	1178330
All Adults	Boston	S75	2016	10	1686878	1394253	1216290	1088223	982659	893433
All Adults	Boston	S75	2016	20	1209148	849994	649334	521463	430476	362480
All Adults	Boston	S75	2016	30	756072	459044	332347	257992	210542	175615
All Adults	Boston	S75	2016	40	444271	234414	149982	102825	70931	49896
All Adults	Boston	S75	2016	50	188627	58897	23089	10958	5087	2348
All Adults	Boston	S75	2016	60	44124	4403	489	98	98	0
All Adults	Boston	S75	2016	70	5283	196	98	0	0	0
All Adults	Boston	S75	2016	80	391	0	0	0	0	0
All Adults	Boston	S75	2016	90	0	0	0	0	0	0
All Adults	Boston	S75	2017	0	1856329	1605871	1448453	1339367	1248478	1172460
All Adults	Boston	S75	2017	10	1682182	1393470	1218638	1091256	984909	892357
All Adults	Boston	S75	2017	20	1218834	847646	647279	517843	424801	352403
All Adults	Boston	S75	2017	30	765758	465697	336652	256231	204085	161918
All Adults	Boston	S75	2017	40	462468	240773	148318	98031	68289	47255
All Adults	Boston	S75	2017	50	213379	63495	21817	7925	3620	1565
All Adults	Boston	S75	2017	60	78366	10762	1272	196	0	0
All Adults	Boston	S75	2017	70	10664	783	98	0	0	0
All Adults	Boston	S75	2017	80	685	0	0	0	0	0
All Adults	Boston	S75	2017	90	0	0	0	0	0	0
All Adults	Dallas	S65	2015	0	1659225	1460056	1340038	1250572	1177202	1118443
All Adults	Dallas	S65	2015	10	1471229	1236976	1093362	983580	895364	818634
All Adults	Dallas	S65	2015	20	1054684	754797	588992	480070	401621	341925
All Adults	Dallas	S65	2015	30	663456	419670	307232	241598	194638	158304
All Adults	Dallas	S65	2015	40	400527	210734	133613	87903	59462	42506
All Adults	Dallas	S65	2015	50	162836	50867	18206	7345	3047	1250
All Adults	Dallas	S65	2015	60	20472	1016	234	0	0	0
All Adults	Dallas	S65	2015	70	547	0	0	0	0	0
All Adults	Dallas	S65	2015	80	0	0	0	0	0	0
All Adults	Dallas	S65	2015	90	0	0	0	0	0	0
All Adults	Dallas	S65	2016	0	1670711	1470213	1353634	1261824	1191110	1126335
All Adults	Dallas	S65	2016	10	1483731	1252057	1103051	994754	906147	830667
All Adults	Dallas	S65	2016	20	1043745	748390	588210	481789	400918	344581
All Adults	Dallas	S65	2016	30	658142	424437	315827	246676	200654	166899
All Adults	Dallas	S65	2016	40	367866	191590	120096	76574	50085	34927
All Adults	Dallas	S65	2016	50	97749	21253	4766	938	313	156
All Adults	Dallas	S65	2016	60	4923	78	78	0	0	0
All Adults	Dallas	S65	2016	70	234	0	0	0	0	0
All Adults	Dallas	S65	2016	80	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Dallas	S65	2016	90	0	0	0	0	0	0
All Adults	Dallas	S65	2017	0	1672743	1467478	1349961	1259792	1182828	1124460
All Adults	Dallas	S65	2017	10	1490841	1258698	1109380	1003349	920446	848014
All Adults	Dallas	S65	2017	20	1069061	774097	611886	502182	418420	359662
All Adults	Dallas	S65	2017	30	683693	438345	329735	264101	214875	179323
All Adults	Dallas	S65	2017	40	396464	214328	135254	92748	62509	45553
All Adults	Dallas	S65	2017	50	141974	37662	11720	3438	1172	469
All Adults	Dallas	S65	2017	60	17659	547	0	0	0	0
All Adults	Dallas	S65	2017	70	156	0	0	0	0	0
All Adults	Dallas	S65	2017	80	0	0	0	0	0	0
All Adults	Dallas	S65	2017	90	0	0	0	0	0	0
All Adults	Dallas	S70	2015	0	1659225	1460056	1340038	1250572	1177202	1118443
All Adults	Dallas	S70	2015	10	1476308	1241664	1099691	990769	902162	825432
All Adults	Dallas	S70	2015	20	1082423	783317	617668	505151	420686	357474
All Adults	Dallas	S70	2015	30	703071	445221	324188	255115	207452	170025
All Adults	Dallas	S70	2015	40	440533	241598	159007	110563	77433	54539
All Adults	Dallas	S70	2015	50	226674	87044	39928	18753	9064	4610
All Adults	Dallas	S70	2015	60	54461	6798	1328	234	78	0
All Adults	Dallas	S70	2015	70	4141	0	0	0	0	0
All Adults	Dallas	S70	2015	80	0	0	0	0	0	0
All Adults	Dallas	S70	2015	90	0	0	0	0	0	0
All Adults	Dallas	S70	2016	0	1670711	1470213	1353634	1261824	1191110	1126335
All Adults	Dallas	S70	2016	10	1485997	1258698	1110317	1001317	912867	839028
All Adults	Dallas	S70	2016	20	1070468	772846	610088	501635	418108	357864
All Adults	Dallas	S70	2016	30	687756	445143	331611	261757	212453	177838
All Adults	Dallas	S70	2016	40	410606	221438	144865	99546	69229	47819
All Adults	Dallas	S70	2016	50	148459	43131	14924	5470	1953	625
All Adults	Dallas	S70	2016	60	14611	1250	313	0	0	0
All Adults	Dallas	S70	2016	70	1328	78	0	0	0	0
All Adults	Dallas	S70	2016	80	78	0	0	0	0	0
All Adults	Dallas	S70	2016	90	0	0	0	0	0	0
All Adults	Dallas	S70	2017	0	1672743	1467478	1349961	1259792	1182828	1124460
All Adults	Dallas	S70	2017	10	1496155	1263386	1120084	1012491	929197	856530
All Adults	Dallas	S70	2017	20	1095940	801601	638061	525467	440923	377867
All Adults	Dallas	S70	2017	30	715026	461551	345441	276915	227377	191200
All Adults	Dallas	S70	2017	40	439595	247380	161430	114470	83684	61571
All Adults	Dallas	S70	2017	50	195810	65088	27348	10314	4297	1406
All Adults	Dallas	S70	2017	60	40865	3125	313	78	78	0
All Adults	Dallas	S70	2017	70	1485	0	0	0	0	0
All Adults	Dallas	S70	2017	80	0	0	0	0	0	0
All Adults	Dallas	S70	2017	90	0	0	0	0	0	0
All Adults	Dallas	S75	2015	0	1659225	1460056	1340038	1250572	1177202	1118443
All Adults	Dallas	S75	2015	10	1476152	1240414	1099534	991238	902084	827307
All Adults	Dallas	S75	2015	20	1104535	803085	635561	520388	434204	368647
All Adults	Dallas	S75	2015	30	733153	466396	339346	265117	214484	176588
All Adults	Dallas	S75	2015	40	471787	265429	174010	124315	90169	63916
All Adults	Dallas	S75	2015	50	276290	115720	58055	32505	16721	10001

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Dallas	S75	2015	60	98139	21097	5157	1406	313	156
All Adults	Dallas	S75	2015	70	12971	625	234	0	0	0
All Adults	Dallas	S75	2015	80	781	0	0	0	0	0
All Adults	Dallas	S75	2015	90	0	0	0	0	0	0
All Adults	Dallas	S75	2016	0	1670711	1470213	1353634	1261824	1191110	1126335
All Adults	Dallas	S75	2016	10	1487481	1258464	1111177	1002567	913648	840903
All Adults	Dallas	S75	2016	20	1087970	792381	626653	515856	432172	367397
All Adults	Dallas	S75	2016	30	709243	463427	341768	268398	220579	183855
All Adults	Dallas	S75	2016	40	440455	241676	161742	115095	82278	59618
All Adults	Dallas	S75	2016	50	192059	67588	27895	11252	4923	2969
All Adults	Dallas	S75	2016	60	31489	3125	625	0	0	0
All Adults	Dallas	S75	2016	70	3751	78	0	0	0	0
All Adults	Dallas	S75	2016	80	313	0	0	0	0	0
All Adults	Dallas	S75	2016	90	78	0	0	0	0	0
All Adults	Dallas	S75	2017	0	1672743	1467478	1349961	1259792	1182828	1124460
All Adults	Dallas	S75	2017	10	1498186	1264793	1122819	1014053	932635	861062
All Adults	Dallas	S75	2017	20	1111724	821525	655720	540469	454519	391150
All Adults	Dallas	S75	2017	30	737294	478429	357474	285276	234018	196904
All Adults	Dallas	S75	2017	40	470224	270821	181667	129863	97670	74620
All Adults	Dallas	S75	2017	50	237847	92045	43600	20472	10158	5391
All Adults	Dallas	S75	2017	60	66416	8282	1485	234	78	78
All Adults	Dallas	S75	2017	70	6329	156	0	0	0	0
All Adults	Dallas	S75	2017	80	0	0	0	0	0	0
All Adults	Dallas	S75	2017	90	0	0	0	0	0	0
All Adults	Detroit	S65	2015	0	1188134	1019169	920988	847385	790298	745861
All Adults	Detroit	S65	2015	10	1064065	871635	763557	679533	612353	558740
All Adults	Detroit	S65	2015	20	746779	519153	395805	320497	267343	226708
All Adults	Detroit	S65	2015	30	467441	292380	217008	170604	138358	114697
All Adults	Detroit	S65	2015	40	278682	147009	93396	62789	43519	31198
All Adults	Detroit	S65	2015	50	98771	27790	10159	3474	1311	655
All Adults	Detroit	S65	2015	60	9700	393	0	0	0	0
All Adults	Detroit	S65	2015	70	0	0	0	0	0	0
All Adults	Detroit	S65	2015	80	0	0	0	0	0	0
All Adults	Detroit	S65	2015	90	0	0	0	0	0	0
All Adults	Detroit	S65	2016	0	1181777	1016416	916465	845615	790167	742060
All Adults	Detroit	S65	2016	10	1065703	876551	765851	685891	623364	564311
All Adults	Detroit	S65	2016	20	773126	542814	420383	344027	285367	241782
All Adults	Detroit	S65	2016	30	494838	303588	224414	180763	148189	121841
All Adults	Detroit	S65	2016	40	295526	155005	99951	67835	47780	32836
All Adults	Detroit	S65	2016	50	128068	39128	14026	5374	1901	655
All Adults	Detroit	S65	2016	60	24578	2228	131	0	0	0
All Adults	Detroit	S65	2016	70	786	0	0	0	0	0
All Adults	Detroit	S65	2016	80	0	0	0	0	0	0
All Adults	Detroit	S65	2016	90	0	0	0	0	0	0
All Adults	Detroit	S65	2017	0	1196917	1026247	926100	854791	800588	755102
All Adults	Detroit	S65	2017	10	1074813	885268	774830	694477	630377	572832
All Adults	Detroit	S65	2017	20	764016	539471	418089	337079	279075	236342

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Detroit	S65	2017	30	489267	307913	228805	179846	145109	121514
All Adults	Detroit	S65	2017	40	293888	156185	102638	70654	49287	36048
All Adults	Detroit	S65	2017	50	108864	28707	10356	3670	1114	721
All Adults	Detroit	S65	2017	60	8520	328	0	0	0	0
All Adults	Detroit	S65	2017	70	262	0	0	0	0	0
All Adults	Detroit	S65	2017	80	0	0	0	0	0	0
All Adults	Detroit	S65	2017	90	0	0	0	0	0	0
All Adults	Detroit	S70	2015	0	1188134	1019169	920988	847385	790298	745861
All Adults	Detroit	S70	2015	10	1064917	873274	763688	680910	614516	561559
All Adults	Detroit	S70	2015	20	764737	538750	413566	334458	279469	237456
All Adults	Detroit	S70	2015	30	487497	307586	225725	176437	142880	119679
All Adults	Detroit	S70	2015	40	311912	170998	114173	78781	58332	42471
All Adults	Detroit	S70	2015	50	147337	52630	23923	11076	5374	2884
All Adults	Detroit	S70	2015	60	30215	3212	590	0	0	0
All Adults	Detroit	S70	2015	70	1049	0	0	0	0	0
All Adults	Detroit	S70	2015	80	0	0	0	0	0	0
All Adults	Detroit	S70	2015	90	0	0	0	0	0	0
All Adults	Detroit	S70	2016	0	1181777	1016416	916465	845615	790167	742060
All Adults	Detroit	S70	2016	10	1065769	876944	765524	687791	626183	566736
All Adults	Detroit	S70	2016	20	791806	564246	439455	359495	300573	254104
All Adults	Detroit	S70	2016	30	521906	323054	236736	189087	154219	127740
All Adults	Detroit	S70	2016	40	326789	179387	119154	86121	61806	45355
All Adults	Detroit	S70	2016	50	176175	67377	31919	15861	7341	3867
All Adults	Detroit	S70	2016	60	62264	9438	1966	262	66	66
All Adults	Detroit	S70	2016	70	7668	131	0	0	0	0
All Adults	Detroit	S70	2016	80	0	0	0	0	0	0
All Adults	Detroit	S70	2016	90	0	0	0	0	0	0
All Adults	Detroit	S70	2017	0	1196917	1026247	926100	854791	800588	755102
All Adults	Detroit	S70	2017	10	1075469	885923	775420	694083	629787	574143
All Adults	Detroit	S70	2017	20	781843	559068	435260	350843	290545	246042
All Adults	Detroit	S70	2017	30	514500	322136	239488	188038	151597	126560
All Adults	Detroit	S70	2017	40	325216	179059	121055	84483	62854	46534
All Adults	Detroit	S70	2017	50	160576	57283	25299	11732	5964	2425
All Adults	Detroit	S70	2017	60	30280	2884	262	66	0	0
All Adults	Detroit	S70	2017	70	2359	0	0	0	0	0
All Adults	Detroit	S70	2017	80	0	0	0	0	0	0
All Adults	Detroit	S70	2017	90	0	0	0	0	0	0
All Adults	Detroit	S75	2015	0	1188134	1019169	920988	847385	790298	745861
All Adults	Detroit	S75	2015	10	1059804	866064	754840	673110	606127	552842
All Adults	Detroit	S75	2015	20	769259	539865	416122	335703	279600	235621
All Adults	Detroit	S75	2015	30	496607	310994	226577	176503	141832	117778
All Adults	Detroit	S75	2015	40	326462	181091	121710	86318	63379	47845
All Adults	Detroit	S75	2015	50	174733	70785	35065	18155	9307	5768
All Adults	Detroit	S75	2015	60	51450	9372	1966	524	0	0
All Adults	Detroit	S75	2015	70	5964	197	0	0	0	0
All Adults	Detroit	S75	2015	80	0	0	0	0	0	0
All Adults	Detroit	S75	2015	90	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Detroit	S75	2016	0	1181777	1016416	916465	845615	790167	742060
All Adults	Detroit	S75	2016	10	1060591	870783	760739	679992	618580	560510
All Adults	Detroit	S75	2016	20	798950	570931	446665	364541	303326	256529
All Adults	Detroit	S75	2016	30	536129	331574	239685	189546	154022	127806
All Adults	Detroit	S75	2016	40	344879	189939	126495	92544	67573	51581
All Adults	Detroit	S75	2016	50	203309	86711	44109	24119	13764	7275
All Adults	Detroit	S75	2016	60	91627	20252	5702	1573	131	131
All Adults	Detroit	S75	2016	70	21170	1180	0	0	0	0
All Adults	Detroit	S75	2016	80	1049	0	0	0	0	0
All Adults	Detroit	S75	2016	90	0	0	0	0	0	0
All Adults	Detroit	S75	2017	0	1196917	1026247	926100	854791	800588	755102
All Adults	Detroit	S75	2017	10	1069242	878517	765655	685432	621398	565688
All Adults	Detroit	S75	2017	20	785776	561690	438144	353006	290545	243290
All Adults	Detroit	S75	2017	30	526560	327445	241192	188235	151597	124922
All Adults	Detroit	S75	2017	40	338194	187186	126560	89398	67377	49746
All Adults	Detroit	S75	2017	50	189087	73931	37948	19597	10290	5505
All Adults	Detroit	S75	2017	60	62526	10093	1966	328	197	0
All Adults	Detroit	S75	2017	70	6161	131	0	0	0	0
All Adults	Detroit	S75	2017	80	328	0	0	0	0	0
All Adults	Detroit	S75	2017	90	0	0	0	0	0	0
All Adults	Philadelphia	S65	2015	0	1659016	1438982	1297637	1196813	1118472	1057385
All Adults	Philadelphia	S65	2015	10	1497106	1244393	1091719	979829	887632	810337
All Adults	Philadelphia	S65	2015	20	1079694	764064	597709	487039	408175	348918
All Adults	Philadelphia	S65	2015	30	694176	443903	330444	262821	212627	181779
All Adults	Philadelphia	S65	2015	40	406170	219512	142303	96641	67622	48800
All Adults	Philadelphia	S65	2015	50	121128	29280	9150	3137	523	174
All Adults	Philadelphia	S65	2015	60	8714	261	0	0	0	0
All Adults	Philadelphia	S65	2015	70	174	0	0	0	0	0
All Adults	Philadelphia	S65	2015	80	0	0	0	0	0	0
All Adults	Philadelphia	S65	2015	90	0	0	0	0	0	0
All Adults	Philadelphia	S65	2016	0	1662589	1436019	1305567	1206137	1125879	1063572
All Adults	Philadelphia	S65	2016	10	1504338	1256506	1104791	992726	899222	826981
All Adults	Philadelphia	S65	2016	20	1083702	774870	603025	492964	413490	351794
All Adults	Philadelphia	S65	2016	30	688512	441115	329311	263431	218814	182563
All Adults	Philadelphia	S65	2016	40	388568	206440	126356	85138	59693	41480
All Adults	Philadelphia	S65	2016	50	104571	23267	6884	1656	784	174
All Adults	Philadelphia	S65	2016	60	8714	174	0	0	0	0
All Adults	Philadelphia	S65	2016	70	87	0	0	0	0	0
All Adults	Philadelphia	S65	2016	80	0	0	0	0	0	0
All Adults	Philadelphia	S65	2016	90	0	0	0	0	0	0
All Adults	Philadelphia	S65	2017	0	1653788	1437500	1306351	1209797	1131369	1064182
All Adults	Philadelphia	S65	2017	10	1503554	1249883	1099649	985493	896259	815042
All Adults	Philadelphia	S65	2017	20	1059477	755437	585248	474490	396933	338722
All Adults	Philadelphia	S65	2017	30	675353	425952	311708	245654	199382	168272
All Adults	Philadelphia	S65	2017	40	371575	191713	116771	74681	51065	33898
All Adults	Philadelphia	S65	2017	50	88624	16121	3747	523	174	87
All Adults	Philadelphia	S65	2017	60	8976	349	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
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All Adults	Philadelphia	S65	2017	70	174	0	0	0	0	0
All Adults	Philadelphia	S65	2017	80	0	0	0	0	0	0
All Adults	Philadelphia	S65	2017	90	0	0	0	0	0	0
All Adults	Philadelphia	S70	2015	0	1659016	1438982	1297637	1196813	1118472	1057385
All Adults	Philadelphia	S70	2015	10	1501463	1249447	1097906	984621	896172	818528
All Adults	Philadelphia	S70	2015	20	1111849	794390	626466	511351	430135	367828
All Adults	Philadelphia	S70	2015	30	728074	468303	349092	274586	222736	189535
All Adults	Philadelphia	S70	2015	40	455667	260120	175592	123916	90802	68232
All Adults	Philadelphia	S70	2015	50	190842	64224	24836	10719	5316	2440
All Adults	Philadelphia	S70	2015	60	27973	2266	349	87	0	0
All Adults	Philadelphia	S70	2015	70	1481	0	0	0	0	0
All Adults	Philadelphia	S70	2015	80	87	0	0	0	0	0
All Adults	Philadelphia	S70	2015	90	0	0	0	0	0	0
All Adults	Philadelphia	S70	2016	0	1662589	1436019	1305567	1206137	1125879	1063572
All Adults	Philadelphia	S70	2016	10	1509393	1263564	1111675	1000830	907588	835957
All Adults	Philadelphia	S70	2016	20	1115074	809465	632218	516144	433620	369309
All Adults	Philadelphia	S70	2016	30	724763	464730	344648	275806	230056	193717
All Adults	Philadelphia	S70	2016	40	442160	246961	162608	113808	81740	60128
All Adults	Philadelphia	S70	2016	50	176550	54638	21350	8191	4183	1743
All Adults	Philadelphia	S70	2016	60	25968	1830	87	0	0	0
All Adults	Philadelphia	S70	2016	70	1481	0	0	0	0	0
All Adults	Philadelphia	S70	2016	80	0	0	0	0	0	0
All Adults	Philadelphia	S70	2016	90	0	0	0	0	0	0
All Adults	Philadelphia	S70	2017	0	1653788	1437500	1306351	1209797	1131369	1064182
All Adults	Philadelphia	S70	2017	10	1509218	1258423	1106098	991854	905845	823931
All Adults	Philadelphia	S70	2017	20	1093114	783584	613569	497321	418283	355367
All Adults	Philadelphia	S70	2017	30	708119	447040	326610	258028	209926	176812
All Adults	Philadelphia	S70	2017	40	424035	234413	146661	102654	71544	50455
All Adults	Philadelphia	S70	2017	50	145005	40434	12548	4444	2091	959
All Adults	Philadelphia	S70	2017	60	25184	1656	261	0	0	0
All Adults	Philadelphia	S70	2017	70	1830	87	0	0	0	0
All Adults	Philadelphia	S70	2017	80	0	0	0	0	0	0
All Adults	Philadelphia	S70	2017	90	0	0	0	0	0	0
All Adults	Philadelphia	S75	2015	0	1659016	1438982	1297637	1196813	1118472	1057385
All Adults	Philadelphia	S75	2015	10	1500853	1248053	1099475	984447	898612	821665
All Adults	Philadelphia	S75	2015	20	1137295	822362	651999	533660	450177	384733
All Adults	Philadelphia	S75	2015	30	763628	493487	365649	287047	232932	197639
All Adults	Philadelphia	S75	2015	40	501417	291578	203565	148142	112588	84615
All Adults	Philadelphia	S75	2015	50	268573	113546	58734	31458	17254	9934
All Adults	Philadelphia	S75	2015	60	71195	12461	3224	523	0	0
All Adults	Philadelphia	S75	2015	70	8017	349	0	0	0	0
All Adults	Philadelphia	S75	2015	80	523	0	0	0	0	0
All Adults	Philadelphia	S75	2015	90	87	0	0	0	0	0
All Adults	Philadelphia	S75	2016	0	1662589	1436019	1305567	1206137	1125879	1063572
All Adults	Philadelphia	S75	2016	10	1509654	1262344	1111762	1003008	909679	838048
All Adults	Philadelphia	S75	2016	20	1142349	839878	659232	540893	453750	386215
All Adults	Philadelphia	S75	2016	30	763106	488259	362512	287395	239293	199643

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Philadelphia	S75	2016	40	492877	281906	190232	136378	102392	77731
All Adults	Philadelphia	S75	2016	50	259946	103699	50891	25968	13768	7756
All Adults	Philadelphia	S75	2016	60	62742	8889	1830	436	261	0
All Adults	Philadelphia	S75	2016	70	6884	87	0	0	0	0
All Adults	Philadelphia	S75	2016	80	87	0	0	0	0	0
All Adults	Philadelphia	S75	2016	90	0	0	0	0	0	0
All Adults	Philadelphia	S75	2017	0	1653788	1437500	1306351	1209797	1131369	1064182
All Adults	Philadelphia	S75	2017	10	1509480	1260253	1106621	995078	904712	825848
All Adults	Philadelphia	S75	2017	20	1119343	810947	639538	519107	436932	370529
All Adults	Philadelphia	S75	2017	30	742191	470394	339245	269008	218727	183086
All Adults	Philadelphia	S75	2017	40	469174	266481	173239	125485	91848	68407
All Adults	Philadelphia	S75	2017	50	215329	78341	32766	14814	7407	4270
All Adults	Philadelphia	S75	2017	60	57340	7494	871	87	0	0
All Adults	Philadelphia	S75	2017	70	8627	261	0	0	0	0
All Adults	Philadelphia	S75	2017	80	261	0	0	0	0	0
All Adults	Philadelphia	S75	2017	90	0	0	0	0	0	0
All Adults	Phoenix	S65	2015	0	1055538	935690	867248	816686	771836	738559
All Adults	Phoenix	S65	2015	10	968570	838738	758227	701705	654670	614787
All Adults	Phoenix	S65	2015	20	749734	578529	479840	411100	357756	315539
All Adults	Phoenix	S65	2015	30	520915	360886	287079	241633	206816	181635
All Adults	Phoenix	S65	2015	40	347724	219531	162215	123623	98988	80611
All Adults	Phoenix	S65	2015	50	144881	57168	27516	14106	7251	3725
All Adults	Phoenix	S65	2015	60	7947	546	50	0	0	0
All Adults	Phoenix	S65	2015	70	0	0	0	0	0	0
All Adults	Phoenix	S65	2015	80	0	0	0	0	0	0
All Adults	Phoenix	S65	2015	90	0	0	0	0	0	0
All Adults	Phoenix	S65	2016	0	1041681	923074	850410	799650	758078	722913
All Adults	Phoenix	S65	2016	10	962858	825875	749386	691921	644041	606989
All Adults	Phoenix	S65	2016	20	741439	571079	474873	405735	356217	315638
All Adults	Phoenix	S65	2016	30	515302	360339	288321	239498	206369	180194
All Adults	Phoenix	S65	2016	40	345141	218985	161668	124616	100974	82051
All Adults	Phoenix	S65	2016	50	115179	41522	17930	8096	3775	1738
All Adults	Phoenix	S65	2016	60	4818	199	0	0	0	0
All Adults	Phoenix	S65	2016	70	0	0	0	0	0	0
All Adults	Phoenix	S65	2016	80	0	0	0	0	0	0
All Adults	Phoenix	S65	2016	90	0	0	0	0	0	0
All Adults	Phoenix	S65	2017	0	1040042	922379	855427	801637	762201	726489
All Adults	Phoenix	S65	2017	10	957742	828358	756290	699172	655067	616724
All Adults	Phoenix	S65	2017	20	755098	587271	492555	421728	371713	326366
All Adults	Phoenix	S65	2017	30	528911	371862	296119	245358	210939	185906
All Adults	Phoenix	S65	2017	40	361978	234630	176668	138821	114136	94418
All Adults	Phoenix	S65	2017	50	148457	63277	31887	17483	10579	6457
All Adults	Phoenix	S65	2017	60	12814	745	99	0	0	0
All Adults	Phoenix	S65	2017	70	0	0	0	0	0	0
All Adults	Phoenix	S65	2017	80	0	0	0	0	0	0
All Adults	Phoenix	S65	2017	90	0	0	0	0	0	0
All Adults	Phoenix	S70	2015	0	1055538	935690	867248	816686	771836	738559

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Phoenix	S70	2015	10	970308	839533	760810	701954	656359	616773
All Adults	Phoenix	S70	2015	20	767267	601079	501197	431116	378220	334115
All Adults	Phoenix	S70	2015	30	547338	382243	301086	250872	214862	186850
All Adults	Phoenix	S70	2015	40	383137	247196	185757	146222	118060	97994
All Adults	Phoenix	S70	2015	50	216402	106637	64717	40827	25678	17036
All Adults	Phoenix	S70	2015	60	45893	9040	2285	894	497	199
All Adults	Phoenix	S70	2015	70	298	50	0	0	0	0
All Adults	Phoenix	S70	2015	80	0	0	0	0	0	0
All Adults	Phoenix	S70	2015	90	0	0	0	0	0	0
All Adults	Phoenix	S70	2016	0	1041681	923074	850410	799650	758078	722913
All Adults	Phoenix	S70	2016	10	963603	827961	750876	693361	645730	608926
All Adults	Phoenix	S70	2016	20	761456	592983	498167	428036	375786	333121
All Adults	Phoenix	S70	2016	30	542669	378170	300639	249233	215856	188340
All Adults	Phoenix	S70	2016	40	380107	248835	186502	149649	121736	101272
All Adults	Phoenix	S70	2016	50	189482	91736	53244	31042	19619	12020
All Adults	Phoenix	S70	2016	60	33178	4718	1043	199	0	0
All Adults	Phoenix	S70	2016	70	149	0	0	0	0	0
All Adults	Phoenix	S70	2016	80	0	0	0	0	0	0
All Adults	Phoenix	S70	2016	90	0	0	0	0	0	0
All Adults	Phoenix	S70	2017	0	1040042	922379	855427	801637	762201	726489
All Adults	Phoenix	S70	2017	10	959928	829848	757333	702152	657054	619207
All Adults	Phoenix	S70	2017	20	775710	611856	515501	446314	393070	347227
All Adults	Phoenix	S70	2017	30	560301	393865	313503	258024	221319	194846
All Adults	Phoenix	S70	2017	40	397640	264630	201552	161370	135642	113441
All Adults	Phoenix	S70	2017	50	221071	118159	75942	50711	33923	23691
All Adults	Phoenix	S70	2017	60	54585	11324	2881	844	348	199
All Adults	Phoenix	S70	2017	70	1788	50	0	0	0	0
All Adults	Phoenix	S70	2017	80	0	0	0	0	0	0
All Adults	Phoenix	S70	2017	90	0	0	0	0	0	0
All Adults	Phoenix	S75	2015	0	1055538	935690	867248	816686	771836	738559
All Adults	Phoenix	S75	2015	10	965242	833772	753906	695993	648859	610168
All Adults	Phoenix	S75	2015	20	771439	609274	508597	439609	385272	340472
All Adults	Phoenix	S75	2015	30	561891	391729	309181	256534	217495	188886
All Adults	Phoenix	S75	2015	40	401315	261500	198472	154417	126404	105296
All Adults	Phoenix	S75	2015	50	257726	139815	91786	60843	43310	30595
All Adults	Phoenix	S75	2015	60	101769	30148	11175	4619	2334	993
All Adults	Phoenix	S75	2015	70	6854	397	50	0	0	0
All Adults	Phoenix	S75	2015	80	50	0	0	0	0	0
All Adults	Phoenix	S75	2015	90	0	0	0	0	0	0
All Adults	Phoenix	S75	2016	0	1041681	923074	850410	799650	758078	722913
All Adults	Phoenix	S75	2016	10	959977	823391	745810	687053	640117	602171
All Adults	Phoenix	S75	2016	20	769353	602022	505319	436132	381299	338734
All Adults	Phoenix	S75	2016	30	557967	387656	308287	255044	218637	189780
All Adults	Phoenix	S75	2016	40	398335	261252	197926	158937	131123	110014
All Adults	Phoenix	S75	2016	50	237064	127050	81852	55081	38244	26175
All Adults	Phoenix	S75	2016	60	78276	19470	6308	2086	894	199
All Adults	Phoenix	S75	2016	70	4718	99	50	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Phoenix	S75	2016	80	0	0	0	0	0	0
All Adults	Phoenix	S75	2016	90	0	0	0	0	0	0
All Adults	Phoenix	S75	2017	0	1040042	922379	855427	801637	762201	726489
All Adults	Phoenix	S75	2017	10	956451	826818	752615	698080	652137	614141
All Adults	Phoenix	S75	2017	20	783309	621244	524590	455651	400073	356713
All Adults	Phoenix	S75	2017	30	576294	406331	322890	264431	225640	197975
All Adults	Phoenix	S75	2017	40	418649	278139	212280	171503	143490	121438
All Adults	Phoenix	S75	2017	50	268007	153970	103358	74502	55777	40728
All Adults	Phoenix	S75	2017	60	105991	35711	13708	6010	3179	1192
All Adults	Phoenix	S75	2017	70	12318	993	99	50	0	0
All Adults	Phoenix	S75	2017	80	397	0	0	0	0	0
All Adults	Phoenix	S75	2017	90	0	0	0	0	0	0
All Adults	Sacramento	S65	2015	0	580206	508974	467127	437713	414074	393207
All Adults	Sacramento	S65	2015	10	515920	432254	384489	350559	324776	301709
All Adults	Sacramento	S65	2015	20	358935	262777	211525	178367	153584	134604
All Adults	Sacramento	S65	2015	30	232591	155328	120626	96844	81094	68917
All Adults	Sacramento	S65	2015	40	115252	58341	34759	22239	14607	10205
All Adults	Sacramento	S65	2015	50	19323	2658	657	57	29	0
All Adults	Sacramento	S65	2015	60	715	29	0	0	0	0
All Adults	Sacramento	S65	2015	70	0	0	0	0	0	0
All Adults	Sacramento	S65	2015	80	0	0	0	0	0	0
All Adults	Sacramento	S65	2015	90	0	0	0	0	0	0
All Adults	Sacramento	S65	2016	0	579921	510346	467641	437456	414474	392607
All Adults	Sacramento	S65	2016	10	512176	432311	385690	351274	324490	300336
All Adults	Sacramento	S65	2016	20	360993	263348	210753	176452	152241	132975
All Adults	Sacramento	S65	2016	30	232992	155528	118797	95415	78750	66516
All Adults	Sacramento	S65	2016	40	112337	55111	31614	20266	12949	8918
All Adults	Sacramento	S65	2016	50	24668	5031	1286	457	114	57
All Adults	Sacramento	S65	2016	60	1172	86	0	0	0	0
All Adults	Sacramento	S65	2016	70	0	0	0	0	0	0
All Adults	Sacramento	S65	2016	80	0	0	0	0	0	0
All Adults	Sacramento	S65	2016	90	0	0	0	0	0	0
All Adults	Sacramento	S65	2017	0	578434	512118	468527	437942	412988	393893
All Adults	Sacramento	S65	2017	10	513891	434740	389434	355247	328549	305825
All Adults	Sacramento	S65	2017	20	359678	263720	211039	176537	151040	131860
All Adults	Sacramento	S65	2017	30	232791	157100	120941	98673	83381	71976
All Adults	Sacramento	S65	2017	40	114824	57426	35216	21981	14549	9747
All Adults	Sacramento	S65	2017	50	19094	2801	515	114	29	0
All Adults	Sacramento	S65	2017	60	1315	29	0	0	0	0
All Adults	Sacramento	S65	2017	70	0	0	0	0	0	0
All Adults	Sacramento	S65	2017	80	0	0	0	0	0	0
All Adults	Sacramento	S65	2017	90	0	0	0	0	0	0
All Adults	Sacramento	S70	2015	0	580206	508974	467127	437713	414074	393207
All Adults	Sacramento	S70	2015	10	518121	435255	388176	354504	328406	305567
All Adults	Sacramento	S70	2015	20	375142	279470	226074	191287	163760	144094
All Adults	Sacramento	S70	2015	30	249713	167419	130173	105505	89241	75692
All Adults	Sacramento	S70	2015	40	147410	83810	56368	39132	28785	21181

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Sacramento	S70	2015	50	49280	14292	5202	2115	715	343
All Adults	Sacramento	S70	2015	60	4688	257	0	0	0	0
All Adults	Sacramento	S70	2015	70	372	0	0	0	0	0
All Adults	Sacramento	S70	2015	80	0	0	0	0	0	0
All Adults	Sacramento	S70	2015	90	0	0	0	0	0	0
All Adults	Sacramento	S70	2016	0	579921	510346	467641	437456	414474	392607
All Adults	Sacramento	S70	2016	10	514834	435369	388920	354304	328206	303538
All Adults	Sacramento	S70	2016	20	376342	277155	225017	187885	162645	141836
All Adults	Sacramento	S70	2016	30	250628	168248	128115	104390	86011	72662
All Adults	Sacramento	S70	2016	40	143837	78493	49851	34530	24926	18237
All Adults	Sacramento	S70	2016	50	56654	18551	8147	3544	1744	772
All Adults	Sacramento	S70	2016	60	9176	972	143	0	0	0
All Adults	Sacramento	S70	2016	70	600	29	0	0	0	0
All Adults	Sacramento	S70	2016	80	0	0	0	0	0	0
All Adults	Sacramento	S70	2016	90	0	0	0	0	0	0
All Adults	Sacramento	S70	2017	0	578434	512118	468527	437942	412988	393893
All Adults	Sacramento	S70	2017	10	516921	437570	392864	359449	332151	310198
All Adults	Sacramento	S70	2017	20	375885	279556	227303	188771	160644	140864
All Adults	Sacramento	S70	2017	30	249085	168105	130745	106105	89755	77550
All Adults	Sacramento	S70	2017	40	146438	83609	56140	40161	28785	21781
All Adults	Sacramento	S70	2017	50	54253	16150	6374	3087	1601	772
All Adults	Sacramento	S70	2017	60	8089	486	0	0	0	0
All Adults	Sacramento	S70	2017	70	229	0	0	0	0	0
All Adults	Sacramento	S70	2017	80	0	0	0	0	0	0
All Adults	Sacramento	S70	2017	90	0	0	0	0	0	0
All Adults	Sacramento	S75	2015	0	580206	508974	467127	437713	414074	393207
All Adults	Sacramento	S75	2015	10	519550	436884	389891	356533	330064	307368
All Adults	Sacramento	S75	2015	20	385747	289703	236507	200491	172364	151555
All Adults	Sacramento	S75	2015	30	264034	177166	136891	110622	94128	80036
All Adults	Sacramento	S75	2015	40	167848	98588	68946	50566	38818	30299
All Adults	Sacramento	S75	2015	50	75006	28384	12806	6632	3487	1601
All Adults	Sacramento	S75	2015	60	15779	1887	314	57	0	0
All Adults	Sacramento	S75	2015	70	1515	57	0	0	0	0
All Adults	Sacramento	S75	2015	80	114	0	0	0	0	0
All Adults	Sacramento	S75	2015	90	0	0	0	0	0	0
All Adults	Sacramento	S75	2016	0	579921	510346	467641	437456	414474	392607
All Adults	Sacramento	S75	2016	10	516206	436941	390206	355504	329578	305453
All Adults	Sacramento	S75	2016	20	386347	288731	233849	196203	169477	148325
All Adults	Sacramento	S75	2016	30	264663	176480	134947	109821	90270	77264
All Adults	Sacramento	S75	2016	40	165447	93385	62714	44677	33444	25554
All Adults	Sacramento	S75	2016	50	81351	32872	16865	9804	5202	2830
All Adults	Sacramento	S75	2016	60	22382	4259	943	372	86	57
All Adults	Sacramento	S75	2016	70	2687	114	29	0	0	0
All Adults	Sacramento	S75	2016	80	200	0	0	0	0	0
All Adults	Sacramento	S75	2016	90	0	0	0	0	0	0
All Adults	Sacramento	S75	2017	0	578434	512118	468527	437942	412988	393893
All Adults	Sacramento	S75	2017	10	517978	439200	394779	361679	334238	312056

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	Sacramento	S75	2017	20	387948	290532	236850	198519	169220	147467
All Adults	Sacramento	S75	2017	30	262005	177138	136891	111622	94128	81294
All Adults	Sacramento	S75	2017	40	165447	99245	69403	51795	39446	30585
All Adults	Sacramento	S75	2017	50	80837	32186	15579	8918	5174	3402
All Adults	Sacramento	S75	2017	60	18008	2172	457	57	0	0
All Adults	Sacramento	S75	2017	70	2944	114	0	0	0	0
All Adults	Sacramento	S75	2017	80	0	0	0	0	0	0
All Adults	Sacramento	S75	2017	90	0	0	0	0	0	0
All Adults	St. Louis	S65	2015	0	677754	588086	534292	493660	463115	438972
All Adults	St. Louis	S65	2015	10	598923	495985	435109	388898	350841	319652
All Adults	St. Louis	S65	2015	20	414328	288034	224154	179588	148935	125865
All Adults	St. Louis	S65	2015	30	257882	161167	117531	90849	73538	60983
All Adults	St. Louis	S65	2015	40	143999	72608	44244	27577	18778	13305
All Adults	St. Louis	S65	2015	50	42849	9121	2361	715	179	0
All Adults	St. Louis	S65	2015	60	1931	0	0	0	0	0
All Adults	St. Louis	S65	2015	70	0	0	0	0	0	0
All Adults	St. Louis	S65	2015	80	0	0	0	0	0	0
All Adults	St. Louis	S65	2015	90	0	0	0	0	0	0
All Adults	St. Louis	S65	2016	0	676395	585725	533433	493839	461291	436754
All Adults	St. Louis	S65	2016	10	603394	501100	442298	398018	361643	330025
All Adults	St. Louis	S65	2016	20	433285	304523	237852	194216	161847	138992
All Adults	St. Louis	S65	2016	30	280737	177871	130622	104011	85913	70748
All Adults	St. Louis	S65	2016	40	168965	89061	56298	38629	27434	19600
All Adults	St. Louis	S65	2016	50	65740	20352	6367	2146	1001	465
All Adults	St. Louis	S65	2016	60	9049	429	72	0	0	0
All Adults	St. Louis	S65	2016	70	107	0	0	0	0	0
All Adults	St. Louis	S65	2016	80	0	0	0	0	0	0
All Adults	St. Louis	S65	2016	90	0	0	0	0	0	0
All Adults	St. Louis	S65	2017	0	675465	586905	532217	492587	461505	436397
All Adults	St. Louis	S65	2017	10	608080	508754	448021	403062	368117	337643
All Adults	St. Louis	S65	2017	20	442334	318293	249978	202979	169895	145823
All Adults	St. Louis	S65	2017	30	288499	185096	138527	110378	90777	76399
All Adults	St. Louis	S65	2017	40	180196	101400	67743	45997	32942	23893
All Adults	St. Louis	S65	2017	50	60554	18134	6688	2253	1288	501
All Adults	St. Louis	S65	2017	60	2897	179	0	0	0	0
All Adults	St. Louis	S65	2017	70	36	0	0	0	0	0
All Adults	St. Louis	S65	2017	80	0	0	0	0	0	0
All Adults	St. Louis	S65	2017	90	0	0	0	0	0	0
All Adults	St. Louis	S70	2015	0	677754	588086	534292	493660	463115	438972
All Adults	St. Louis	S70	2015	10	600676	498131	438686	391652	355062	322943
All Adults	St. Louis	S70	2015	20	429887	303736	235921	191248	158556	133662
All Adults	St. Louis	S70	2015	30	273799	170431	124470	96607	78616	64488
All Adults	St. Louis	S70	2015	40	168249	89847	59016	39308	27076	19922
All Adults	St. Louis	S70	2015	50	72965	23106	8906	4149	1896	465
All Adults	St. Louis	S70	2015	60	11016	858	179	0	0	0
All Adults	St. Louis	S70	2015	70	72	0	0	0	0	0
All Adults	St. Louis	S70	2015	80	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	St. Louis	S70	2015	90	0	0	0	0	0	0
All Adults	St. Louis	S70	2016	0	676395	585725	533433	493839	461291	436754
All Adults	St. Louis	S70	2016	10	605755	504140	444909	402167	365184	333530
All Adults	St. Louis	S70	2016	20	448164	321262	251086	208094	173757	148649
All Adults	St. Louis	S70	2016	30	298478	189638	139099	110664	92029	75684
All Adults	St. Louis	S70	2016	40	192857	106944	71785	51898	38164	28471
All Adults	St. Louis	S70	2016	50	96715	37591	17490	8119	4006	2289
All Adults	St. Louis	S70	2016	60	28185	4435	572	143	0	0
All Adults	St. Louis	S70	2016	70	2075	0	0	0	0	0
All Adults	St. Louis	S70	2016	80	0	0	0	0	0	0
All Adults	St. Louis	S70	2016	90	0	0	0	0	0	0
All Adults	St. Louis	S70	2017	0	675465	586905	532217	492587	461505	436397
All Adults	St. Louis	S70	2017	10	610583	511901	452420	406996	372302	342901
All Adults	St. Louis	S70	2017	20	457177	335819	266037	216356	182055	155910
All Adults	St. Louis	S70	2017	30	307134	197900	147504	117281	96464	80655
All Adults	St. Louis	S70	2017	40	203945	120107	82658	60018	44781	33872
All Adults	St. Louis	S70	2017	50	100220	39952	19279	9764	4936	2611
All Adults	St. Louis	S70	2017	60	17741	2468	393	36	36	0
All Adults	St. Louis	S70	2017	70	680	0	0	0	0	0
All Adults	St. Louis	S70	2017	80	0	0	0	0	0	0
All Adults	St. Louis	S70	2017	90	0	0	0	0	0	0
All Adults	St. Louis	S75	2015	0	677754	588086	534292	493660	463115	438972
All Adults	St. Louis	S75	2015	10	600712	498989	439294	391866	355026	323944
All Adults	St. Louis	S75	2015	20	438292	312928	243683	196291	164208	137668
All Adults	St. Louis	S75	2015	30	285852	177120	129370	100041	81514	66956
All Adults	St. Louis	S75	2015	40	181912	100184	66313	45889	32942	24429
All Adults	St. Louis	S75	2015	50	93102	35445	17347	8548	4650	2253
All Adults	St. Louis	S75	2015	60	25967	3577	644	107	0	0
All Adults	St. Louis	S75	2015	70	1753	36	0	0	0	0
All Adults	St. Louis	S75	2015	80	0	0	0	0	0	0
All Adults	St. Louis	S75	2015	90	0	0	0	0	0	0
All Adults	St. Louis	S75	2016	0	676395	585725	533433	493839	461291	436754
All Adults	St. Louis	S75	2016	10	606434	504891	446304	401774	366257	334424
All Adults	St. Louis	S75	2016	20	457893	329739	260314	215641	180625	153871
All Adults	St. Louis	S75	2016	30	311605	197292	144321	115528	95177	78795
All Adults	St. Louis	S75	2016	40	206556	116530	80119	58623	44351	33836
All Adults	St. Louis	S75	2016	50	117424	50968	27398	14879	8370	4900
All Adults	St. Louis	S75	2016	60	45961	10802	2361	644	143	0
All Adults	St. Louis	S75	2016	70	8226	179	36	0	0	0
All Adults	St. Louis	S75	2016	80	250	0	0	0	0	0
All Adults	St. Louis	S75	2016	90	0	0	0	0	0	0
All Adults	St. Louis	S75	2017	0	675465	586905	532217	492587	461505	436397
All Adults	St. Louis	S75	2017	10	611585	513117	453386	408892	373053	345154
All Adults	St. Louis	S75	2017	20	467443	346549	276481	226800	190461	163170
All Adults	St. Louis	S75	2017	30	320403	207915	153084	121716	100220	83552
All Adults	St. Louis	S75	2017	40	217930	129585	91528	67529	51111	40024
All Adults	St. Louis	S75	2017	50	124148	56584	30116	17276	10194	6295

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Adults	St. Louis	S75	2017	60	37270	7583	1538	429	250	107
All Adults	St. Louis	S75	2017	70	3291	215	0	0	0	0
All Adults	St. Louis	S75	2017	80	179	0	0	0	0	0
All Adults	St. Louis	S75	2017	90	0	0	0	0	0	0
Asthma Adults	Atlanta	S65	2015	0	99029	84802	77054	70433	65362	60221
Asthma Adults	Atlanta	S65	2015	10	84309	67898	58037	49867	44091	39936
Asthma Adults	Atlanta	S65	2015	20	55149	38738	29371	23525	20144	16974
Asthma Adults	Atlanta	S65	2015	30	35146	22257	15284	11692	9297	7325
Asthma Adults	Atlanta	S65	2015	40	18665	9579	5353	3592	2254	1761
Asthma Adults	Atlanta	S65	2015	50	5423	1197	282	70	0	0
Asthma Adults	Atlanta	S65	2015	60	423	0	0	0	0	0
Asthma Adults	Atlanta	S65	2015	70	0	0	0	0	0	0
Asthma Adults	Atlanta	S65	2015	80	0	0	0	0	0	0
Asthma Adults	Atlanta	S65	2016	0	99029	86211	75998	69236	64306	60643
Asthma Adults	Atlanta	S65	2016	10	86985	72476	62474	55079	49867	45570
Asthma Adults	Atlanta	S65	2016	20	61700	41767	33033	25567	20637	17538
Asthma Adults	Atlanta	S65	2016	30	37964	23595	17397	13101	10635	8241
Asthma Adults	Atlanta	S65	2016	40	21905	11762	6269	4226	2747	1902
Asthma Adults	Atlanta	S65	2016	50	4860	845	70	0	0	0
Asthma Adults	Atlanta	S65	2016	60	211	0	0	0	0	0
Asthma Adults	Atlanta	S65	2016	70	0	0	0	0	0	0
Asthma Adults	Atlanta	S65	2016	80	0	0	0	0	0	0
Asthma Adults	Atlanta	S65	2017	0	103255	88535	79519	72194	67194	62615
Asthma Adults	Atlanta	S65	2017	10	86704	69940	59939	52543	47050	43035
Asthma Adults	Atlanta	S65	2017	20	57262	37752	28807	22680	19087	16622
Asthma Adults	Atlanta	S65	2017	30	34372	20496	15707	11833	9297	7889
Asthma Adults	Atlanta	S65	2017	40	16411	8100	5212	3240	1690	1338
Asthma Adults	Atlanta	S65	2017	50	2676	352	70	0	0	0
Asthma Adults	Atlanta	S65	2017	60	70	0	0	0	0	0
Asthma Adults	Atlanta	S65	2017	70	0	0	0	0	0	0
Asthma Adults	Atlanta	S65	2017	80	0	0	0	0	0	0
Asthma Adults	Atlanta	S70	2015	0	99029	84802	77054	70433	65362	60221
Asthma Adults	Atlanta	S70	2015	10	85365	69166	58953	50994	44796	40851
Asthma Adults	Atlanta	S70	2015	20	57967	41344	31061	25286	21553	18031
Asthma Adults	Atlanta	S70	2015	30	38245	24159	16693	13030	10706	8170
Asthma Adults	Atlanta	S70	2015	40	22891	12326	7748	4930	3451	2606
Asthma Adults	Atlanta	S70	2015	50	8804	3310	1479	634	282	70
Asthma Adults	Atlanta	S70	2015	60	1268	70	0	0	0	0
Asthma Adults	Atlanta	S70	2015	70	70	0	0	0	0	0
Asthma Adults	Atlanta	S70	2015	80	0	0	0	0	0	0
Asthma Adults	Atlanta	S70	2016	0	99029	86211	75998	69236	64306	60643
Asthma Adults	Atlanta	S70	2016	10	88394	73040	63601	56347	50853	46627
Asthma Adults	Atlanta	S70	2016	20	64588	45782	35710	28526	23313	19651
Asthma Adults	Atlanta	S70	2016	30	40922	25567	18947	14861	11762	9720
Asthma Adults	Atlanta	S70	2016	40	25708	14580	8804	6550	4719	3310
Asthma Adults	Atlanta	S70	2016	50	10072	3029	845	211	70	0
Asthma Adults	Atlanta	S70	2016	60	2113	211	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Atlanta	S70	2016	70	141	0	0	0	0	0
Asthma Adults	Atlanta	S70	2016	80	0	0	0	0	0	0
Asthma Adults	Atlanta	S70	2017	0	103255	88535	79519	72194	67194	62615
Asthma Adults	Atlanta	S70	2017	10	88042	71208	60855	54304	48599	44303
Asthma Adults	Atlanta	S70	2017	20	59516	40570	30779	25004	20637	17890
Asthma Adults	Atlanta	S70	2017	30	37259	22609	16622	13594	11058	8875
Asthma Adults	Atlanta	S70	2017	40	21130	11340	7466	5212	3944	2747
Asthma Adults	Atlanta	S70	2017	50	6480	1338	563	70	0	0
Asthma Adults	Atlanta	S70	2017	60	775	0	0	0	0	0
Asthma Adults	Atlanta	S70	2017	70	0	0	0	0	0	0
Asthma Adults	Atlanta	S70	2017	80	0	0	0	0	0	0
Asthma Adults	Atlanta	S75	2015	0	99029	84802	77054	70433	65362	60221
Asthma Adults	Atlanta	S75	2015	10	86281	69729	59657	51909	46204	41344
Asthma Adults	Atlanta	S75	2015	20	59868	42964	33526	26624	22539	19228
Asthma Adults	Atlanta	S75	2015	30	41415	26060	18594	14157	11692	9368
Asthma Adults	Atlanta	S75	2015	40	26835	15284	9931	6902	4789	3451
Asthma Adults	Atlanta	S75	2015	50	12889	5564	2888	1690	916	352
Asthma Adults	Atlanta	S75	2015	60	3310	775	70	0	0	0
Asthma Adults	Atlanta	S75	2015	70	423	0	0	0	0	0
Asthma Adults	Atlanta	S75	2015	80	0	0	0	0	0	0
Asthma Adults	Atlanta	S75	2016	0	99029	86211	75998	69236	64306	60643
Asthma Adults	Atlanta	S75	2016	10	89028	73744	63954	56629	51487	47754
Asthma Adults	Atlanta	S75	2016	20	66630	48529	38457	30498	25074	21553
Asthma Adults	Atlanta	S75	2016	30	44655	27187	20567	15848	12889	11128
Asthma Adults	Atlanta	S75	2016	40	29159	16904	10635	7959	6339	4930
Asthma Adults	Atlanta	S75	2016	50	15566	5987	2817	986	282	141
Asthma Adults	Atlanta	S75	2016	60	4156	704	70	0	0	0
Asthma Adults	Atlanta	S75	2016	70	1057	70	0	0	0	0
Asthma Adults	Atlanta	S75	2016	80	70	0	0	0	0	0
Asthma Adults	Atlanta	S75	2017	0	103255	88535	79519	72194	67194	62615
Asthma Adults	Atlanta	S75	2017	10	88605	71842	61348	54727	49163	45077
Asthma Adults	Atlanta	S75	2017	20	61911	42894	32681	26624	22327	19017
Asthma Adults	Atlanta	S75	2017	30	40147	24088	17890	14791	12115	9931
Asthma Adults	Atlanta	S75	2017	40	24863	13735	9861	6550	5423	3874
Asthma Adults	Atlanta	S75	2017	50	10072	3310	1479	704	563	141
Asthma Adults	Atlanta	S75	2017	60	2254	70	0	0	0	0
Asthma Adults	Atlanta	S75	2017	70	211	0	0	0	0	0
Asthma Adults	Atlanta	S75	2017	80	0	0	0	0	0	0
Asthma Adults	Boston	S65	2015	0	171995	144601	128654	117011	105564	99107
Asthma Adults	Boston	S65	2015	10	152525	122783	105564	90498	80225	72692
Asthma Adults	Boston	S65	2015	20	100673	67506	50679	38449	32384	25535
Asthma Adults	Boston	S65	2015	30	58310	33949	24459	19078	13990	11251
Asthma Adults	Boston	S65	2015	40	27296	11642	6164	3424	2152	1468
Asthma Adults	Boston	S65	2015	50	4990	1076	0	0	0	0
Asthma Adults	Boston	S65	2015	60	489	0	0	0	0	0
Asthma Adults	Boston	S65	2015	70	196	0	0	0	0	0
Asthma Adults	Boston	S65	2015	80	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
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Asthma Adults	Boston	S65	2016	0	177180	150275	133448	122588	112902	105369
Asthma Adults	Boston	S65	2016	10	157711	127382	109674	97444	86878	79247
Asthma Adults	Boston	S65	2016	20	104097	69952	52048	42265	35123	29546
Asthma Adults	Boston	S65	2016	30	62125	39428	28764	21915	17610	14675
Asthma Adults	Boston	S65	2016	40	34145	16632	9588	6457	4011	2739
Asthma Adults	Boston	S65	2016	50	8414	1565	294	196	98	0
Asthma Adults	Boston	S65	2016	60	196	0	0	0	0	0
Asthma Adults	Boston	S65	2016	70	0	0	0	0	0	0
Asthma Adults	Boston	S65	2016	80	0	0	0	0	0	0
Asthma Adults	Boston	S65	2017	0	174636	148416	131589	120925	110750	103216
Asthma Adults	Boston	S65	2017	10	156145	127284	109086	97151	87171	77486
Asthma Adults	Boston	S65	2017	20	105369	71029	54103	44613	36199	29448
Asthma Adults	Boston	S65	2017	30	62713	39526	28372	21719	15947	12621
Asthma Adults	Boston	S65	2017	40	33949	17415	9392	5479	3913	2250
Asthma Adults	Boston	S65	2017	50	11251	2348	489	294	0	0
Asthma Adults	Boston	S65	2017	60	1565	98	0	0	0	0
Asthma Adults	Boston	S65	2017	70	98	0	0	0	0	0
Asthma Adults	Boston	S65	2017	80	0	0	0	0	0	0
Asthma Adults	Boston	S70	2015	0	171995	144601	128654	117011	105564	99107
Asthma Adults	Boston	S70	2015	10	152428	122979	105662	90987	80421	72594
Asthma Adults	Boston	S70	2015	20	103314	69757	51951	39526	33362	26318
Asthma Adults	Boston	S70	2015	30	61343	35319	25731	19763	14675	11447
Asthma Adults	Boston	S70	2015	40	31014	14871	8120	5185	3131	1859
Asthma Adults	Boston	S70	2015	50	8512	1957	685	0	0	0
Asthma Adults	Boston	S70	2015	60	1370	0	0	0	0	0
Asthma Adults	Boston	S70	2015	70	196	0	0	0	0	0
Asthma Adults	Boston	S70	2015	80	98	0	0	0	0	0
Asthma Adults	Boston	S70	2016	0	177180	150275	133448	122588	112902	105369
Asthma Adults	Boston	S70	2016	10	158200	128751	110163	97346	86780	79345
Asthma Adults	Boston	S70	2016	20	107228	71713	53516	43830	36395	30622
Asthma Adults	Boston	S70	2016	30	65550	40895	29448	23187	18491	14969
Asthma Adults	Boston	S70	2016	40	39036	20154	11740	8218	5283	3718
Asthma Adults	Boston	S70	2016	50	12621	3816	1663	587	196	98
Asthma Adults	Boston	S70	2016	60	2055	489	0	0	0	0
Asthma Adults	Boston	S70	2016	70	0	0	0	0	0	0
Asthma Adults	Boston	S70	2016	80	0	0	0	0	0	0
Asthma Adults	Boston	S70	2017	0	174636	148416	131589	120925	110750	103216
Asthma Adults	Boston	S70	2017	10	155950	126990	109282	97444	87269	77583
Asthma Adults	Boston	S70	2017	20	108010	73474	56255	45493	36982	30916
Asthma Adults	Boston	S70	2017	30	65843	40700	29351	22698	16730	13697
Asthma Adults	Boston	S70	2017	40	38743	20643	11447	7631	5381	3522
Asthma Adults	Boston	S70	2017	50	16241	4403	783	294	98	0
Asthma Adults	Boston	S70	2017	60	4207	294	0	0	0	0
Asthma Adults	Boston	S70	2017	70	685	98	0	0	0	0
Asthma Adults	Boston	S70	2017	80	98	0	0	0	0	0
Asthma Adults	Boston	S75	2015	0	171995	144601	128654	117011	105564	99107
Asthma Adults	Boston	S75	2015	10	152428	122392	105662	90400	79834	71909

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Boston	S75	2015	20	104488	70833	52538	41091	33851	26611
Asthma Adults	Boston	S75	2015	30	62419	35514	26024	19665	15164	11447
Asthma Adults	Boston	S75	2015	40	32775	16241	9392	6261	3620	2446
Asthma Adults	Boston	S75	2015	50	10664	3033	1076	98	0	0
Asthma Adults	Boston	S75	2015	60	2250	196	0	0	0	0
Asthma Adults	Boston	S75	2015	70	391	0	0	0	0	0
Asthma Adults	Boston	S75	2015	80	196	0	0	0	0	0
Asthma Adults	Boston	S75	2016	0	177180	150275	133448	122588	112902	105369
Asthma Adults	Boston	S75	2016	10	158004	128458	109674	96661	86584	79149
Asthma Adults	Boston	S75	2016	20	108402	72203	54005	44319	37373	31307
Asthma Adults	Boston	S75	2016	30	66724	41287	29448	23285	18687	15262
Asthma Adults	Boston	S75	2016	40	40895	20937	12719	8903	6164	4109
Asthma Adults	Boston	S75	2016	50	16045	5283	2837	1468	391	196
Asthma Adults	Boston	S75	2016	60	3033	587	98	98	98	0
Asthma Adults	Boston	S75	2016	70	294	0	0	0	0	0
Asthma Adults	Boston	S75	2016	80	0	0	0	0	0	0
Asthma Adults	Boston	S75	2017	0	174636	148416	131589	120925	110750	103216
Asthma Adults	Boston	S75	2017	10	155558	126599	109282	97542	86878	77290
Asthma Adults	Boston	S75	2017	20	108499	73768	56745	45591	37569	31992
Asthma Adults	Boston	S75	2017	30	67702	41776	29644	22600	17317	13599
Asthma Adults	Boston	S75	2017	40	41580	21817	12621	8414	5674	3913
Asthma Adults	Boston	S75	2017	50	18784	6066	1174	391	196	98
Asthma Adults	Boston	S75	2017	60	6164	881	0	0	0	0
Asthma Adults	Boston	S75	2017	70	783	98	0	0	0	0
Asthma Adults	Boston	S75	2017	80	98	0	0	0	0	0
Asthma Adults	Dallas	S65	2015	0	102827	89701	81887	75558	70401	66650
Asthma Adults	Dallas	S65	2015	10	90091	74933	64853	57430	51492	46726
Asthma Adults	Dallas	S65	2015	20	63759	44538	34536	27660	22972	18440
Asthma Adults	Dallas	S65	2015	30	39693	24457	17034	12814	10001	7814
Asthma Adults	Dallas	S65	2015	40	23675	11564	7110	4610	3360	2422
Asthma Adults	Dallas	S65	2015	50	9064	1875	703	234	0	0
Asthma Adults	Dallas	S65	2015	60	938	0	0	0	0	0
Asthma Adults	Dallas	S65	2015	70	0	0	0	0	0	0
Asthma Adults	Dallas	S65	2015	80	0	0	0	0	0	0
Asthma Adults	Dallas	S65	2016	0	106500	93217	84700	77668	72432	67510
Asthma Adults	Dallas	S65	2016	10	94154	78605	66807	59696	54539	50007
Asthma Adults	Dallas	S65	2016	20	64853	46726	36255	28754	23753	20784
Asthma Adults	Dallas	S65	2016	30	42115	26098	19143	14143	11486	9454
Asthma Adults	Dallas	S65	2016	40	21956	10158	6720	4376	2422	1641
Asthma Adults	Dallas	S65	2016	50	5079	1328	313	78	78	78
Asthma Adults	Dallas	S65	2016	60	156	0	0	0	0	0
Asthma Adults	Dallas	S65	2016	70	78	0	0	0	0	0
Asthma Adults	Dallas	S65	2016	80	0	0	0	0	0	0
Asthma Adults	Dallas	S65	2017	0	102046	89701	82668	75870	70870	66103
Asthma Adults	Dallas	S65	2017	10	91732	77042	65400	59618	54227	50007
Asthma Adults	Dallas	S65	2017	20	61884	45241	35161	29614	25160	21800
Asthma Adults	Dallas	S65	2017	30	39381	25551	19534	16096	13049	10548

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Asthma Adults	Dallas	S65	2017	40	23285	12814	7970	5626	3829	2813
Asthma Adults	Dallas	S65	2017	50	8908	2578	469	78	0	0
Asthma Adults	Dallas	S65	2017	60	859	0	0	0	0	0
Asthma Adults	Dallas	S65	2017	70	0	0	0	0	0	0
Asthma Adults	Dallas	S65	2017	80	0	0	0	0	0	0
Asthma Adults	Dallas	S70	2015	0	102827	89701	81887	75558	70401	66650
Asthma Adults	Dallas	S70	2015	10	90638	75402	65400	57899	52351	47429
Asthma Adults	Dallas	S70	2015	20	65400	45944	35943	29301	23753	19065
Asthma Adults	Dallas	S70	2015	30	41959	25941	18362	13830	11252	8361
Asthma Adults	Dallas	S70	2015	40	26254	13596	8204	5782	3751	2969
Asthma Adults	Dallas	S70	2015	50	13439	3907	1953	703	313	234
Asthma Adults	Dallas	S70	2015	60	3047	156	0	0	0	0
Asthma Adults	Dallas	S70	2015	70	234	0	0	0	0	0
Asthma Adults	Dallas	S70	2015	80	0	0	0	0	0	0
Asthma Adults	Dallas	S70	2016	0	106500	93217	84700	77668	72432	67510
Asthma Adults	Dallas	S70	2016	10	94389	79074	67432	59931	54930	50398
Asthma Adults	Dallas	S70	2016	20	66963	47585	37974	30161	25082	21566
Asthma Adults	Dallas	S70	2016	30	44147	27738	20315	15002	11642	9923
Asthma Adults	Dallas	S70	2016	40	25472	12189	7970	5782	3516	2266
Asthma Adults	Dallas	S70	2016	50	8048	2344	859	313	156	78
Asthma Adults	Dallas	S70	2016	60	781	78	0	0	0	0
Asthma Adults	Dallas	S70	2016	70	78	0	0	0	0	0
Asthma Adults	Dallas	S70	2016	80	0	0	0	0	0	0
Asthma Adults	Dallas	S70	2017	0	102046	89701	82668	75870	70870	66103
Asthma Adults	Dallas	S70	2017	10	92123	77199	66494	59852	55242	50242
Asthma Adults	Dallas	S70	2017	20	64462	47194	36412	30708	26332	22972
Asthma Adults	Dallas	S70	2017	30	41334	26723	20315	16956	13908	11330
Asthma Adults	Dallas	S70	2017	40	25082	15237	9689	7267	5235	3751
Asthma Adults	Dallas	S70	2017	50	11720	4610	1563	469	156	0
Asthma Adults	Dallas	S70	2017	60	2500	78	0	0	0	0
Asthma Adults	Dallas	S70	2017	70	0	0	0	0	0	0
Asthma Adults	Dallas	S70	2017	80	0	0	0	0	0	0
Asthma Adults	Dallas	S75	2015	0	102827	89701	81887	75558	70401	66650
Asthma Adults	Dallas	S75	2015	10	90404	75402	65322	57899	52508	47507
Asthma Adults	Dallas	S75	2015	20	66728	47819	36802	29848	24613	19534
Asthma Adults	Dallas	S75	2015	30	44381	27191	19456	14768	11564	8986
Asthma Adults	Dallas	S75	2015	40	27504	15002	9064	6642	4610	3204
Asthma Adults	Dallas	S75	2015	50	16565	6251	2813	1485	703	313
Asthma Adults	Dallas	S75	2015	60	5313	859	156	0	0	0
Asthma Adults	Dallas	S75	2015	70	469	0	0	0	0	0
Asthma Adults	Dallas	S75	2015	80	0	0	0	0	0	0
Asthma Adults	Dallas	S75	2016	0	106500	93217	84700	77668	72432	67510
Asthma Adults	Dallas	S75	2016	10	94389	78996	67432	60399	55086	50554
Asthma Adults	Dallas	S75	2016	20	68057	48679	38756	30864	25629	21566
Asthma Adults	Dallas	S75	2016	30	45163	29223	21019	15315	12033	10158
Asthma Adults	Dallas	S75	2016	40	27348	13518	8517	6798	4376	2969
Asthma Adults	Dallas	S75	2016	50	11095	3907	1406	547	234	156

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Dallas	S75	2016	60	1719	78	0	0	0	0
Asthma Adults	Dallas	S75	2016	70	234	0	0	0	0	0
Asthma Adults	Dallas	S75	2016	80	78	0	0	0	0	0
Asthma Adults	Dallas	S75	2017	0	102046	89701	82668	75870	70870	66103
Asthma Adults	Dallas	S75	2017	10	92123	77433	66416	60087	55242	50476
Asthma Adults	Dallas	S75	2017	20	65400	48288	37427	32114	27035	23128
Asthma Adults	Dallas	S75	2017	30	42741	27817	21175	17659	14299	11799
Asthma Adults	Dallas	S75	2017	40	26566	16799	10705	7814	5938	4219
Asthma Adults	Dallas	S75	2017	50	14221	6485	3047	1328	547	234
Asthma Adults	Dallas	S75	2017	60	4454	469	0	0	0	0
Asthma Adults	Dallas	S75	2017	70	156	0	0	0	0	0
Asthma Adults	Dallas	S75	2017	80	0	0	0	0	0	0
Asthma Adults	Detroit	S65	2015	0	117319	100672	87825	81337	75569	71178
Asthma Adults	Detroit	S65	2015	10	104145	83762	72751	64034	56497	50729
Asthma Adults	Detroit	S65	2015	20	70850	47452	35458	27921	23202	19859
Asthma Adults	Detroit	S65	2015	30	42799	25823	19138	14812	12125	9831
Asthma Adults	Detroit	S65	2015	40	24709	12781	7931	4916	3408	2294
Asthma Adults	Detroit	S65	2015	50	9045	2884	1049	197	131	66
Asthma Adults	Detroit	S65	2015	60	655	0	0	0	0	0
Asthma Adults	Detroit	S65	2015	70	0	0	0	0	0	0
Asthma Adults	Detroit	S65	2015	80	0	0	0	0	0	0
Asthma Adults	Detroit	S65	2016	0	116467	99557	89923	82189	74717	69867
Asthma Adults	Detroit	S65	2016	10	103883	84745	73210	64296	57611	51450
Asthma Adults	Detroit	S65	2016	20	73406	49877	38735	31001	26020	21563
Asthma Adults	Detroit	S65	2016	30	46141	27658	20383	15861	13108	10093
Asthma Adults	Detroit	S65	2016	40	26741	13567	7996	5374	4195	3015
Asthma Adults	Detroit	S65	2016	50	10552	3408	1311	459	131	66
Asthma Adults	Detroit	S65	2016	60	2032	262	0	0	0	0
Asthma Adults	Detroit	S65	2016	70	66	0	0	0	0	0
Asthma Adults	Detroit	S65	2016	80	0	0	0	0	0	0
Asthma Adults	Detroit	S65	2017	0	116205	99885	89464	81534	75504	70195
Asthma Adults	Detroit	S65	2017	10	104670	85269	73406	64689	57087	52302
Asthma Adults	Detroit	S65	2017	20	72882	51188	39325	32115	25889	21498
Asthma Adults	Detroit	S65	2017	30	46403	29100	22153	17762	14091	12256
Asthma Adults	Detroit	S65	2017	40	28117	15861	10093	7078	4785	3343
Asthma Adults	Detroit	S65	2017	50	10749	2949	1442	328	197	131
Asthma Adults	Detroit	S65	2017	60	721	0	0	0	0	0
Asthma Adults	Detroit	S65	2017	70	66	0	0	0	0	0
Asthma Adults	Detroit	S65	2017	80	0	0	0	0	0	0
Asthma Adults	Detroit	S70	2015	0	117319	100672	87825	81337	75569	71178
Asthma Adults	Detroit	S70	2015	10	104407	84286	73079	64099	56431	50991
Asthma Adults	Detroit	S70	2015	20	72161	49484	36900	29231	24119	20842
Asthma Adults	Detroit	S70	2015	30	44634	27527	19990	15402	12846	10356
Asthma Adults	Detroit	S70	2015	40	28248	15009	9569	6685	4588	3539
Asthma Adults	Detroit	S70	2015	50	13108	4653	2294	655	393	131
Asthma Adults	Detroit	S70	2015	60	2425	197	66	0	0	0
Asthma Adults	Detroit	S70	2015	70	66	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Detroit	S70	2015	80	0	0	0	0	0	0
Asthma Adults	Detroit	S70	2016	0	116467	99557	89923	82189	74717	69867
Asthma Adults	Detroit	S70	2016	10	103818	85204	72948	64427	58004	52040
Asthma Adults	Detroit	S70	2016	20	74914	52105	40374	32574	26741	22612
Asthma Adults	Detroit	S70	2016	30	48763	29494	21301	16582	13698	11208
Asthma Adults	Detroit	S70	2016	40	29756	15664	9897	7013	5112	3998
Asthma Adults	Detroit	S70	2016	50	15206	5637	2622	1507	524	197
Asthma Adults	Detroit	S70	2016	60	5047	590	66	0	0	0
Asthma Adults	Detroit	S70	2016	70	655	0	0	0	0	0
Asthma Adults	Detroit	S70	2016	80	0	0	0	0	0	0
Asthma Adults	Detroit	S70	2017	0	116205	99885	89464	81534	75504	70195
Asthma Adults	Detroit	S70	2017	10	104473	85204	73144	64362	57349	52433
Asthma Adults	Detroit	S70	2017	20	74324	53089	40898	33098	27134	22743
Asthma Adults	Detroit	S70	2017	30	48566	31067	23005	18221	14747	12453
Asthma Adults	Detroit	S70	2017	40	31919	18286	12387	8586	6226	4457
Asthma Adults	Detroit	S70	2017	50	14878	5571	2818	1114	590	262
Asthma Adults	Detroit	S70	2017	60	2425	197	0	0	0	0
Asthma Adults	Detroit	S70	2017	70	197	0	0	0	0	0
Asthma Adults	Detroit	S70	2017	80	0	0	0	0	0	0
Asthma Adults	Detroit	S75	2015	0	117319	100672	87825	81337	75569	71178
Asthma Adults	Detroit	S75	2015	10	104670	83696	71244	62854	55579	50008
Asthma Adults	Detroit	S75	2015	20	73013	49353	37096	30018	23923	20646
Asthma Adults	Detroit	S75	2015	30	45617	28052	20646	15468	12912	9831
Asthma Adults	Detroit	S75	2015	40	29559	15795	10356	7210	4981	3801
Asthma Adults	Detroit	S75	2015	50	15730	6030	3080	1245	524	459
Asthma Adults	Detroit	S75	2015	60	4719	721	197	0	0	0
Asthma Adults	Detroit	S75	2015	70	524	66	0	0	0	0
Asthma Adults	Detroit	S75	2015	80	0	0	0	0	0	0
Asthma Adults	Detroit	S75	2016	0	116467	99557	89923	82189	74717	69867
Asthma Adults	Detroit	S75	2016	10	103031	84286	72489	63510	57349	51253
Asthma Adults	Detroit	S75	2016	20	75438	53351	41029	32902	26938	22808
Asthma Adults	Detroit	S75	2016	30	50467	30018	21629	16844	13764	11273
Asthma Adults	Detroit	S75	2016	40	31329	16975	10945	7799	5899	4653
Asthma Adults	Detroit	S75	2016	50	17631	7144	3867	2228	1245	590
Asthma Adults	Detroit	S75	2016	60	7472	1966	393	131	0	0
Asthma Adults	Detroit	S75	2016	70	1835	66	0	0	0	0
Asthma Adults	Detroit	S75	2016	80	0	0	0	0	0	0
Asthma Adults	Detroit	S75	2017	0	116205	99885	89464	81534	75504	70195
Asthma Adults	Detroit	S75	2017	10	104211	84155	72161	63706	56693	52105
Asthma Adults	Detroit	S75	2017	20	74848	53351	41291	33033	26872	21956
Asthma Adults	Detroit	S75	2017	30	49943	31788	23333	18548	14550	12584
Asthma Adults	Detroit	S75	2017	40	33295	18614	12256	9241	7013	4850
Asthma Adults	Detroit	S75	2017	50	17893	7013	3998	2228	1180	655
Asthma Adults	Detroit	S75	2017	60	6620	786	262	0	0	0
Asthma Adults	Detroit	S75	2017	70	459	0	0	0	0	0
Asthma Adults	Detroit	S75	2017	80	131	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2015	0	137336	115725	101608	91238	85487	80868

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Philadelphia	S65	2015	10	120866	97077	84005	75117	67187	60738
Asthma Adults	Philadelphia	S65	2015	20	84964	58995	46708	37297	30761	25533
Asthma Adults	Philadelphia	S65	2015	30	55945	36426	25794	20217	16557	14378
Asthma Adults	Philadelphia	S65	2015	40	33114	17603	12113	7843	5403	3747
Asthma Adults	Philadelphia	S65	2015	50	10370	2876	610	87	0	0
Asthma Adults	Philadelphia	S65	2015	60	871	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2015	70	0	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2015	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2016	0	131672	112152	101434	93242	85748	79474
Asthma Adults	Philadelphia	S65	2016	10	119298	97077	84790	75291	67448	61958
Asthma Adults	Philadelphia	S65	2016	20	85487	61087	45053	36600	31110	26927
Asthma Adults	Philadelphia	S65	2016	30	55161	33898	24661	19258	16034	13246
Asthma Adults	Philadelphia	S65	2016	40	30587	14814	8279	4531	2701	1830
Asthma Adults	Philadelphia	S65	2016	50	6536	1394	436	87	87	0
Asthma Adults	Philadelphia	S65	2016	60	436	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2016	70	0	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2016	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2017	0	136726	115725	102654	95247	88972	82088
Asthma Adults	Philadelphia	S65	2017	10	121999	98122	85835	77121	69627	63004
Asthma Adults	Philadelphia	S65	2017	20	81740	58908	45837	37645	32504	28234
Asthma Adults	Philadelphia	S65	2017	30	54900	33986	24923	20043	15773	12636
Asthma Adults	Philadelphia	S65	2017	40	29367	14988	7930	4009	2701	2091
Asthma Adults	Philadelphia	S65	2017	50	7581	697	87	0	0	0
Asthma Adults	Philadelphia	S65	2017	60	871	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2017	70	0	0	0	0	0	0
Asthma Adults	Philadelphia	S65	2017	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2015	0	137336	115725	101608	91238	85487	80868
Asthma Adults	Philadelphia	S70	2015	10	121041	97599	84702	75378	68058	61610
Asthma Adults	Philadelphia	S70	2015	20	87752	61348	48625	38778	32766	27276
Asthma Adults	Philadelphia	S70	2015	30	58473	37907	27101	21698	17603	14814
Asthma Adults	Philadelphia	S70	2015	40	36251	20827	14466	10283	7146	5664
Asthma Adults	Philadelphia	S70	2015	50	16470	5141	1830	174	87	87
Asthma Adults	Philadelphia	S70	2015	60	2614	174	0	0	0	0
Asthma Adults	Philadelphia	S70	2015	70	0	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2015	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2016	0	131672	112152	101434	93242	85748	79474
Asthma Adults	Philadelphia	S70	2016	10	119995	98209	85487	75727	67971	63091
Asthma Adults	Philadelphia	S70	2016	20	88101	63265	47493	38517	32853	28757
Asthma Adults	Philadelphia	S70	2016	30	57688	35467	25620	20653	17167	14117
Asthma Adults	Philadelphia	S70	2016	40	35293	17777	12026	6971	4793	3224
Asthma Adults	Philadelphia	S70	2016	50	12113	3573	1220	523	261	174
Asthma Adults	Philadelphia	S70	2016	60	1569	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2016	70	0	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2016	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2017	0	136726	115725	102654	95247	88972	82088
Asthma Adults	Philadelphia	S70	2017	10	122958	98994	86097	77731	70585	63527
Asthma Adults	Philadelphia	S70	2017	20	84702	61435	48190	39563	33986	29193

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Philadelphia	S70	2017	30	56817	35380	26753	20914	16383	14030
Asthma Adults	Philadelphia	S70	2017	40	33811	18387	11503	6884	4357	2963
Asthma Adults	Philadelphia	S70	2017	50	12461	3137	523	0	0	0
Asthma Adults	Philadelphia	S70	2017	60	2353	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2017	70	261	0	0	0	0	0
Asthma Adults	Philadelphia	S70	2017	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S75	2015	0	137336	115725	101608	91238	85487	80868
Asthma Adults	Philadelphia	S75	2015	10	121041	98558	84964	75030	68145	62307
Asthma Adults	Philadelphia	S75	2015	20	89757	63091	49235	40783	33898	29106
Asthma Adults	Philadelphia	S75	2015	30	59954	40347	28408	22134	18300	15511
Asthma Adults	Philadelphia	S75	2015	40	39563	23877	16034	12200	8627	6449
Asthma Adults	Philadelphia	S75	2015	50	22657	9150	4270	1656	610	349
Asthma Adults	Philadelphia	S75	2015	60	5926	784	87	0	0	0
Asthma Adults	Philadelphia	S75	2015	70	697	0	0	0	0	0
Asthma Adults	Philadelphia	S75	2015	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S75	2016	0	131672	112152	101434	93242	85748	79474
Asthma Adults	Philadelphia	S75	2016	10	120431	98122	85574	76075	67797	63265
Asthma Adults	Philadelphia	S75	2016	20	90367	65270	49933	40085	33550	29803
Asthma Adults	Philadelphia	S75	2016	30	60477	37297	27276	21350	17603	14378
Asthma Adults	Philadelphia	S75	2016	40	40173	20740	13943	8540	6361	4793
Asthma Adults	Philadelphia	S75	2016	50	19346	6884	3050	1481	871	349
Asthma Adults	Philadelphia	S75	2016	60	3573	436	0	0	0	0
Asthma Adults	Philadelphia	S75	2016	70	523	0	0	0	0	0
Asthma Adults	Philadelphia	S75	2016	80	0	0	0	0	0	0
Asthma Adults	Philadelphia	S75	2017	0	136726	115725	102654	95247	88972	82088
Asthma Adults	Philadelphia	S75	2017	10	123481	99517	86009	77818	70062	63614
Asthma Adults	Philadelphia	S75	2017	20	87578	63004	49845	40783	35206	30238
Asthma Adults	Philadelphia	S75	2017	30	58821	37123	27711	21960	17254	14291
Asthma Adults	Philadelphia	S75	2017	40	38081	21176	13681	9324	6361	3921
Asthma Adults	Philadelphia	S75	2017	50	17603	5839	2091	436	174	174
Asthma Adults	Philadelphia	S75	2017	60	5141	174	0	0	0	0
Asthma Adults	Philadelphia	S75	2017	70	610	0	0	0	0	0
Asthma Adults	Philadelphia	S75	2017	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S65	2015	0	81554	71968	65512	60297	56472	53790
Asthma Adults	Phoenix	S65	2015	10	74055	63475	55826	49817	46588	42565
Asthma Adults	Phoenix	S65	2015	20	55578	42615	34420	28907	25132	22152
Asthma Adults	Phoenix	S65	2015	30	39386	26076	20066	17036	14205	12367
Asthma Adults	Phoenix	S65	2015	40	25231	14950	10877	8245	6357	5215
Asthma Adults	Phoenix	S65	2015	50	10579	3775	1490	944	447	447
Asthma Adults	Phoenix	S65	2015	60	596	50	0	0	0	0
Asthma Adults	Phoenix	S65	2015	70	0	0	0	0	0	0
Asthma Adults	Phoenix	S65	2015	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S65	2016	0	78177	68243	61687	58012	54734	51853
Asthma Adults	Phoenix	S65	2016	10	71621	60247	53095	48327	44751	42118
Asthma Adults	Phoenix	S65	2016	20	52697	39237	31837	26970	23493	21109
Asthma Adults	Phoenix	S65	2016	30	35413	23840	19519	16589	14155	12516
Asthma Adults	Phoenix	S65	2016	40	23940	15347	10977	8146	6755	5612

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Phoenix	S65	2016	50	8593	2930	1341	447	199	149
Asthma Adults	Phoenix	S65	2016	60	248	0	0	0	0	0
Asthma Adults	Phoenix	S65	2016	70	0	0	0	0	0	0
Asthma Adults	Phoenix	S65	2016	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S65	2017	0	77233	66604	60942	56323	52797	50214
Asthma Adults	Phoenix	S65	2017	10	69882	58757	52598	48277	45098	43012
Asthma Adults	Phoenix	S65	2017	20	53293	39833	33377	28807	23989	21506
Asthma Adults	Phoenix	S65	2017	30	35661	24983	20562	16539	14702	13410
Asthma Adults	Phoenix	S65	2017	40	24884	16539	12715	10281	8195	6804
Asthma Adults	Phoenix	S65	2017	50	10430	4818	2384	1242	497	248
Asthma Adults	Phoenix	S65	2017	60	944	0	0	0	0	0
Asthma Adults	Phoenix	S65	2017	70	0	0	0	0	0	0
Asthma Adults	Phoenix	S65	2017	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S70	2015	0	81554	71968	65512	60297	56472	53790
Asthma Adults	Phoenix	S70	2015	10	74104	63277	55926	49717	46787	43360
Asthma Adults	Phoenix	S70	2015	20	56969	44204	36108	30049	26324	23443
Asthma Adults	Phoenix	S70	2015	30	40728	28162	21158	17533	14553	12914
Asthma Adults	Phoenix	S70	2015	40	27814	17036	12665	10033	7996	6606
Asthma Adults	Phoenix	S70	2015	50	15397	7301	4073	2334	1440	993
Asthma Adults	Phoenix	S70	2015	60	3328	695	199	149	50	50
Asthma Adults	Phoenix	S70	2015	70	50	50	0	0	0	0
Asthma Adults	Phoenix	S70	2015	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S70	2016	0	78177	68243	61687	58012	54734	51853
Asthma Adults	Phoenix	S70	2016	10	71819	60396	53095	48674	45098	42218
Asthma Adults	Phoenix	S70	2016	20	54535	41026	33675	28559	24784	22400
Asthma Adults	Phoenix	S70	2016	30	36853	25082	20016	17284	15049	13063
Asthma Adults	Phoenix	S70	2016	40	26771	17135	13212	10480	8146	6804
Asthma Adults	Phoenix	S70	2016	50	14006	6258	3824	2334	1341	695
Asthma Adults	Phoenix	S70	2016	60	2831	397	99	50	0	0
Asthma Adults	Phoenix	S70	2016	70	0	0	0	0	0	0
Asthma Adults	Phoenix	S70	2016	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S70	2017	0	77233	66604	60942	56323	52797	50214
Asthma Adults	Phoenix	S70	2017	10	70329	58906	52598	48625	45049	42963
Asthma Adults	Phoenix	S70	2017	20	55081	41522	34817	30347	26125	22897
Asthma Adults	Phoenix	S70	2017	30	37499	26175	21655	17682	15149	13559
Asthma Adults	Phoenix	S70	2017	40	27218	18526	14056	11920	10083	8344
Asthma Adults	Phoenix	S70	2017	50	15198	8593	5712	3924	2583	1788
Asthma Adults	Phoenix	S70	2017	60	3973	1142	99	50	0	0
Asthma Adults	Phoenix	S70	2017	70	99	0	0	0	0	0
Asthma Adults	Phoenix	S70	2017	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S75	2015	0	81554	71968	65512	60297	56472	53790
Asthma Adults	Phoenix	S75	2015	10	73856	62879	55032	49419	46290	42814
Asthma Adults	Phoenix	S75	2015	20	57168	44651	36456	30943	26970	23940
Asthma Adults	Phoenix	S75	2015	30	41622	28311	21953	18029	14801	13112
Asthma Adults	Phoenix	S75	2015	40	29403	18029	13659	10381	8394	6953
Asthma Adults	Phoenix	S75	2015	50	18725	9338	6159	3973	2682	1689
Asthma Adults	Phoenix	S75	2015	60	7599	1887	646	298	248	50

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Phoenix	S75	2015	70	298	50	50	0	0	0
Asthma Adults	Phoenix	S75	2015	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S75	2016	0	78177	68243	61687	58012	54734	51853
Asthma Adults	Phoenix	S75	2016	10	71770	59850	52946	48128	44999	41622
Asthma Adults	Phoenix	S75	2016	20	55131	41622	33824	29304	25678	22648
Asthma Adults	Phoenix	S75	2016	30	37896	25579	20761	17433	15347	13112
Asthma Adults	Phoenix	S75	2016	40	27913	18129	14106	11424	8791	7450
Asthma Adults	Phoenix	S75	2016	50	17135	8791	5861	3675	2483	1738
Asthma Adults	Phoenix	S75	2016	60	6159	1589	447	149	99	0
Asthma Adults	Phoenix	S75	2016	70	447	0	0	0	0	0
Asthma Adults	Phoenix	S75	2016	80	0	0	0	0	0	0
Asthma Adults	Phoenix	S75	2017	0	77233	66604	60942	56323	52797	50214
Asthma Adults	Phoenix	S75	2017	10	69982	58509	52201	48128	45049	42764
Asthma Adults	Phoenix	S75	2017	20	55677	42317	35810	30744	26870	23890
Asthma Adults	Phoenix	S75	2017	30	38592	27168	21854	17880	15546	13808
Asthma Adults	Phoenix	S75	2017	40	28509	19768	14751	12566	10877	9238
Asthma Adults	Phoenix	S75	2017	50	18178	11175	7251	5364	4222	3129
Asthma Adults	Phoenix	S75	2017	60	7947	2930	993	397	99	50
Asthma Adults	Phoenix	S75	2017	70	1043	50	0	0	0	0
Asthma Adults	Phoenix	S75	2017	80	0	0	0	0	0	0
Asthma Adults	Sacramento	S65	2015	0	36274	31357	28213	26012	24583	23096
Asthma Adults	Sacramento	S65	2015	10	31643	25726	22439	20324	18694	16979
Asthma Adults	Sacramento	S65	2015	20	21467	14978	12063	10233	8833	7546
Asthma Adults	Sacramento	S65	2015	30	13921	9004	7003	5431	4745	4059
Asthma Adults	Sacramento	S65	2015	40	7289	3373	2344	1486	800	572
Asthma Adults	Sacramento	S65	2015	50	1343	200	29	0	0	0
Asthma Adults	Sacramento	S65	2015	60	57	0	0	0	0	0
Asthma Adults	Sacramento	S65	2015	70	0	0	0	0	0	0
Asthma Adults	Sacramento	S65	2015	80	0	0	0	0	0	0
Asthma Adults	Sacramento	S65	2016	0	36960	31586	28127	26040	24383	22896
Asthma Adults	Sacramento	S65	2016	10	31443	25526	22667	20409	18894	17236
Asthma Adults	Sacramento	S65	2016	20	21181	15350	12406	10319	9033	7775
Asthma Adults	Sacramento	S65	2016	30	13806	8947	6717	5460	4602	4030
Asthma Adults	Sacramento	S65	2016	40	6660	3373	1972	1229	772	543
Asthma Adults	Sacramento	S65	2016	50	1658	286	0	0	0	0
Asthma Adults	Sacramento	S65	2016	60	57	0	0	0	0	0
Asthma Adults	Sacramento	S65	2016	70	0	0	0	0	0	0
Asthma Adults	Sacramento	S65	2016	80	0	0	0	0	0	0
Asthma Adults	Sacramento	S65	2017	0	36588	31614	28413	25983	23725	22267
Asthma Adults	Sacramento	S65	2017	10	31814	25926	22582	19723	17665	16265
Asthma Adults	Sacramento	S65	2017	20	21067	14578	11177	9290	7804	6689
Asthma Adults	Sacramento	S65	2017	30	12663	8204	6632	5374	4316	3802
Asthma Adults	Sacramento	S65	2017	40	6117	3144	1801	1201	829	629
Asthma Adults	Sacramento	S65	2017	50	943	143	0	0	0	0
Asthma Adults	Sacramento	S65	2017	60	86	0	0	0	0	0
Asthma Adults	Sacramento	S65	2017	70	0	0	0	0	0	0
Asthma Adults	Sacramento	S65	2017	80	0	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Sacramento	S70	2015	0	36274	31357	28213	26012	24583	23096
Asthma Adults	Sacramento	S70	2015	10	31700	25955	22696	20524	18780	17494
Asthma Adults	Sacramento	S70	2015	20	22296	16207	13006	11005	9376	8032
Asthma Adults	Sacramento	S70	2015	30	14864	9747	7746	6060	5059	4402
Asthma Adults	Sacramento	S70	2015	40	9347	5117	3201	2458	1829	1401
Asthma Adults	Sacramento	S70	2015	50	3259	972	429	57	0	0
Asthma Adults	Sacramento	S70	2015	60	257	0	0	0	0	0
Asthma Adults	Sacramento	S70	2015	70	57	0	0	0	0	0
Asthma Adults	Sacramento	S70	2015	80	0	0	0	0	0	0
Asthma Adults	Sacramento	S70	2016	0	36960	31586	28127	26040	24383	22896
Asthma Adults	Sacramento	S70	2016	10	31529	25755	22839	20724	19123	17465
Asthma Adults	Sacramento	S70	2016	20	21953	16122	13235	11119	9433	8261
Asthma Adults	Sacramento	S70	2016	30	14892	9776	7318	5860	5088	4230
Asthma Adults	Sacramento	S70	2016	40	8347	4688	3030	2230	1629	1172
Asthma Adults	Sacramento	S70	2016	50	3716	1201	343	143	57	29
Asthma Adults	Sacramento	S70	2016	60	286	86	0	0	0	0
Asthma Adults	Sacramento	S70	2016	70	29	0	0	0	0	0
Asthma Adults	Sacramento	S70	2016	80	0	0	0	0	0	0
Asthma Adults	Sacramento	S70	2017	0	36588	31614	28413	25983	23725	22267
Asthma Adults	Sacramento	S70	2017	10	31929	26298	22868	20266	18094	16608
Asthma Adults	Sacramento	S70	2017	20	22124	15436	11948	9804	8318	7175
Asthma Adults	Sacramento	S70	2017	30	14035	9090	7175	5688	4716	4059
Asthma Adults	Sacramento	S70	2017	40	7918	4602	3287	2144	1629	1372
Asthma Adults	Sacramento	S70	2017	50	3030	772	143	57	29	0
Asthma Adults	Sacramento	S70	2017	60	343	29	0	0	0	0
Asthma Adults	Sacramento	S70	2017	70	29	0	0	0	0	0
Asthma Adults	Sacramento	S70	2017	80	0	0	0	0	0	0
Asthma Adults	Sacramento	S75	2015	0	36274	31357	28213	26012	24583	23096
Asthma Adults	Sacramento	S75	2015	10	31729	26098	22982	20781	18894	17637
Asthma Adults	Sacramento	S75	2015	20	23039	16893	13578	11405	9719	8432
Asthma Adults	Sacramento	S75	2015	30	15836	10262	8089	6317	5260	4574
Asthma Adults	Sacramento	S75	2015	40	10490	6088	4202	3116	2315	1915
Asthma Adults	Sacramento	S75	2015	50	4888	1829	915	400	257	114
Asthma Adults	Sacramento	S75	2015	60	972	143	0	0	0	0
Asthma Adults	Sacramento	S75	2015	70	86	0	0	0	0	0
Asthma Adults	Sacramento	S75	2015	80	29	0	0	0	0	0
Asthma Adults	Sacramento	S75	2016	0	36960	31586	28127	26040	24383	22896
Asthma Adults	Sacramento	S75	2016	10	31643	25869	22896	20867	19180	17551
Asthma Adults	Sacramento	S75	2016	20	22953	16722	13663	11777	9976	8575
Asthma Adults	Sacramento	S75	2016	30	15693	10147	7661	6146	5260	4431
Asthma Adults	Sacramento	S75	2016	40	9576	5517	3830	2858	2230	1658
Asthma Adults	Sacramento	S75	2016	50	5145	2001	972	515	286	114
Asthma Adults	Sacramento	S75	2016	60	1629	286	0	0	0	0
Asthma Adults	Sacramento	S75	2016	70	114	0	0	0	0	0
Asthma Adults	Sacramento	S75	2016	80	0	0	0	0	0	0
Asthma Adults	Sacramento	S75	2017	0	36588	31614	28413	25983	23725	22267
Asthma Adults	Sacramento	S75	2017	10	31986	26526	23153	20495	18208	16665

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Sacramento	S75	2017	20	23296	16236	12491	10090	8718	7603
Asthma Adults	Sacramento	S75	2017	30	14950	9519	7575	5888	4888	4202
Asthma Adults	Sacramento	S75	2017	40	9004	5460	3887	2773	2144	1744
Asthma Adults	Sacramento	S75	2017	50	4345	1744	829	429	200	114
Asthma Adults	Sacramento	S75	2017	60	972	143	0	0	0	0
Asthma Adults	Sacramento	S75	2017	70	114	0	0	0	0	0
Asthma Adults	Sacramento	S75	2017	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S65	2015	0	59231	50503	45496	41812	38843	36447
Asthma Adults	St. Louis	S65	2015	10	51719	41562	35767	31439	28256	25431
Asthma Adults	St. Louis	S65	2015	20	34873	23463	18062	14378	11696	9693
Asthma Adults	St. Louis	S65	2015	30	20602	12805	9013	6975	5544	4793
Asthma Adults	St. Louis	S65	2015	40	11231	5687	3434	2075	1538	1037
Asthma Adults	St. Louis	S65	2015	50	3505	715	215	72	36	0
Asthma Adults	St. Louis	S65	2015	60	143	0	0	0	0	0
Asthma Adults	St. Louis	S65	2015	70	0	0	0	0	0	0
Asthma Adults	St. Louis	S65	2015	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S65	2016	0	56262	47570	43028	39094	36053	33764
Asthma Adults	St. Louis	S65	2016	10	49824	40524	34945	30402	27398	25180
Asthma Adults	St. Louis	S65	2016	20	34337	23249	17669	14307	11624	9657
Asthma Adults	St. Louis	S65	2016	30	21496	12876	9693	7762	6259	5437
Asthma Adults	St. Louis	S65	2016	40	13019	6903	4149	3076	1967	1431
Asthma Adults	St. Louis	S65	2016	50	5294	1645	572	72	36	0
Asthma Adults	St. Louis	S65	2016	60	930	72	36	0	0	0
Asthma Adults	St. Louis	S65	2016	70	36	0	0	0	0	0
Asthma Adults	St. Louis	S65	2016	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S65	2017	0	58694	50897	45746	42062	39094	36769
Asthma Adults	St. Louis	S65	2017	10	52435	43243	37556	34158	30760	27577
Asthma Adults	St. Louis	S65	2017	20	37198	26217	20495	16524	13556	11338
Asthma Adults	St. Louis	S65	2017	30	23249	15165	10694	8405	6832	5437
Asthma Adults	St. Louis	S65	2017	40	14271	7368	4686	3112	2253	1717
Asthma Adults	St. Louis	S65	2017	50	4328	1180	644	286	179	107
Asthma Adults	St. Louis	S65	2017	60	72	0	0	0	0	0
Asthma Adults	St. Louis	S65	2017	70	0	0	0	0	0	0
Asthma Adults	St. Louis	S65	2017	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S70	2015	0	59231	50503	45496	41812	38843	36447
Asthma Adults	St. Louis	S70	2015	10	52006	42134	36125	31547	28578	25896
Asthma Adults	St. Louis	S70	2015	20	36232	24679	18849	15487	12411	10337
Asthma Adults	St. Louis	S70	2015	30	22211	13949	9621	7475	5830	5007
Asthma Adults	St. Louis	S70	2015	40	13341	7153	4542	3040	2003	1610
Asthma Adults	St. Louis	S70	2015	50	5794	1717	644	215	107	0
Asthma Adults	St. Louis	S70	2015	60	787	72	72	0	0	0
Asthma Adults	St. Louis	S70	2015	70	0	0	0	0	0	0
Asthma Adults	St. Louis	S70	2015	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S70	2016	0	56262	47570	43028	39094	36053	33764
Asthma Adults	St. Louis	S70	2016	10	50432	40632	35195	30796	27791	25287
Asthma Adults	St. Louis	S70	2016	20	35588	24358	18635	15559	12197	10194
Asthma Adults	St. Louis	S70	2016	30	22784	13913	9979	8012	6545	5615

Study Group	Study Area	AQ Scenario	Year	Benchmark (ppb)	Number of People with 7-hr Exposure at or above Benchmark					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	St. Louis	S70	2016	40	14665	8262	5580	3899	2683	1860
Asthma Adults	St. Louis	S70	2016	50	7618	2826	1431	501	286	143
Asthma Adults	St. Louis	S70	2016	60	2325	358	36	0	0	0
Asthma Adults	St. Louis	S70	2016	70	179	0	0	0	0	0
Asthma Adults	St. Louis	S70	2016	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S70	2017	0	58694	50897	45746	42062	39094	36769
Asthma Adults	St. Louis	S70	2017	10	52685	43672	37985	34372	31082	28256
Asthma Adults	St. Louis	S70	2017	20	38307	27684	21997	17383	14378	12447
Asthma Adults	St. Louis	S70	2017	30	24894	16203	11660	8835	7511	5973
Asthma Adults	St. Louis	S70	2017	40	16453	9478	6009	4221	2790	2325
Asthma Adults	St. Louis	S70	2017	50	7332	2754	1431	823	501	358
Asthma Adults	St. Louis	S70	2017	60	1073	143	36	0	0	0
Asthma Adults	St. Louis	S70	2017	70	36	0	0	0	0	0
Asthma Adults	St. Louis	S70	2017	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S75	2015	0	59231	50503	45496	41812	38843	36447
Asthma Adults	St. Louis	S75	2015	10	52041	42420	36518	31726	28757	25896
Asthma Adults	St. Louis	S75	2015	20	36876	25538	19600	15809	12912	10802
Asthma Adults	St. Louis	S75	2015	30	23499	14522	10015	7762	6009	5115
Asthma Adults	St. Louis	S75	2015	40	14593	8048	5222	3434	2718	1896
Asthma Adults	St. Louis	S75	2015	50	7618	2861	1180	537	215	72
Asthma Adults	St. Louis	S75	2015	60	2039	250	72	36	0	0
Asthma Adults	St. Louis	S75	2015	70	36	0	0	0	0	0
Asthma Adults	St. Louis	S75	2015	80	0	0	0	0	0	0
Asthma Adults	St. Louis	S75	2016	0	56262	47570	43028	39094	36053	33764
Asthma Adults	St. Louis	S75	2016	10	50360	40846	35481	30831	27720	25395
Asthma Adults	St. Louis	S75	2016	20	36626	24787	19493	15809	12805	10444
Asthma Adults	St. Louis	S75	2016	30	24071	14450	10373	8298	6653	5723
Asthma Adults	St. Louis	S75	2016	40	15416	8870	6080	4614	3291	2432
Asthma Adults	St. Louis	S75	2016	50	9264	3863	2218	1109	537	322
Asthma Adults	St. Louis	S75	2016	60	3577	787	143	0	0	0
Asthma Adults	St. Louis	S75	2016	70	858	72	36	0	0	0
Asthma Adults	St. Louis	S75	2016	80	36	0	0	0	0	0
Asthma Adults	St. Louis	S75	2017	0	58694	50897	45746	42062	39094	36769
Asthma Adults	St. Louis	S75	2017	10	52685	43815	38343	34623	31082	28328
Asthma Adults	St. Louis	S75	2017	20	39237	28542	22855	18456	14986	13019
Asthma Adults	St. Louis	S75	2017	30	26003	16846	12089	9156	7690	6224
Asthma Adults	St. Louis	S75	2017	40	17204	10122	6617	4757	3398	2861
Asthma Adults	St. Louis	S75	2017	50	9550	4256	2182	1252	930	680
Asthma Adults	St. Louis	S75	2017	60	2539	644	107	107	107	36
Asthma Adults	St. Louis	S75	2017	70	107	0	0	0	0	0
Asthma Adults	St. Louis	S75	2017	80	36	0	0	0	0	0

Study Group	Study Area	AQ Scenario	Year	FEV ₁ (percent)	Number of People at or above FEV ₁ Decrement					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Atlanta	S65	2015	10	119728	64262	44045	33433	25947	20943
All Children	Atlanta	S65	2015	15	31879	15718	9806	6739	4802	3612
All Children	Atlanta	S65	2015	20	12812	4903	2663	1897	1291	1069
All Children	Atlanta	S65	2016	10	141680	82320	57846	44510	36156	29619
All Children	Atlanta	S65	2016	15	40636	19955	12994	9261	7122	5649
All Children	Atlanta	S65	2016	20	15213	6457	3914	2583	1957	1392
All Children	Atlanta	S65	2017	10	111112	60772	41019	31072	24373	19713
All Children	Atlanta	S65	2017	15	28368	13357	8535	6134	4600	3470
All Children	Atlanta	S65	2017	20	9443	3975	2119	1372	989	646
All Children	Atlanta	S70	2015	10	154048	87284	61922	47536	38457	31536
All Children	Atlanta	S70	2015	15	48303	24232	16444	11561	8777	6941
All Children	Atlanta	S70	2015	20	20217	8979	4903	3369	2441	1856
All Children	Atlanta	S70	2016	10	182558	110689	80424	63455	51955	43561
All Children	Atlanta	S70	2016	15	60328	32040	21125	15536	12187	9765
All Children	Atlanta	S70	2016	20	25685	11904	7607	5206	3914	3087
All Children	Atlanta	S70	2017	10	141680	80585	57443	44106	35228	28711
All Children	Atlanta	S70	2017	15	40676	20338	13720	9967	7647	5972
All Children	Atlanta	S70	2017	20	15233	7042	4358	2764	1957	1372
All Children	Atlanta	S75	2015	10	192081	114563	83491	65170	53266	44631
All Children	Atlanta	S75	2015	15	68560	36136	24736	18482	14668	11682
All Children	Atlanta	S75	2015	20	29680	14890	9624	6335	4822	3632
All Children	Atlanta	S75	2016	10	226744	142527	107238	85791	70739	60489
All Children	Atlanta	S75	2016	15	85367	47516	32484	25039	19309	15798
All Children	Atlanta	S75	2016	20	39385	19370	12590	9019	6840	5306
All Children	Atlanta	S75	2017	10	175435	105584	75824	59057	47839	40252
All Children	Atlanta	S75	2017	15	56918	30103	20580	15294	11803	9221
All Children	Atlanta	S75	2017	20	23728	11561	7445	5145	3632	2744
All Children	Boston	S65	2015	10	142102	77729	54110	40116	31196	25713
All Children	Boston	S65	2015	15	41823	18909	11855	7850	5529	4210
All Children	Boston	S65	2015	20	15746	5825	3368	2207	1365	956
All Children	Boston	S65	2016	10	144536	79345	54884	40594	32061	25804
All Children	Boston	S65	2016	15	41686	20138	12606	8851	6508	4960
All Children	Boston	S65	2016	20	16474	6713	3868	2435	1752	1206
All Children	Boston	S65	2017	10	155777	83827	56295	41117	32152	26259
All Children	Boston	S65	2017	15	51380	23141	13994	9352	6508	4505
All Children	Boston	S65	2017	20	21435	7668	3868	2480	1661	1115
All Children	Boston	S70	2015	10	168747	94590	66762	51152	40685	33267
All Children	Boston	S70	2015	15	55225	25849	16998	11832	8738	6553
All Children	Boston	S70	2015	20	22368	9079	5234	3390	2480	1616
All Children	Boston	S70	2016	10	177849	99960	70061	52972	41709	34086
All Children	Boston	S70	2016	15	56932	28853	18454	13425	9830	7691
All Children	Boston	S70	2016	20	24461	10672	6030	4050	2890	2230
All Children	Boston	S70	2017	10	192821	107742	73019	54929	42187	34223
All Children	Boston	S70	2017	15	68218	32471	20502	14358	10331	7850
All Children	Boston	S70	2017	20	31924	12697	6485	4323	2981	2048

Study Group	Study Area	AQ Scenario	Year	FEV ₁ (percent)	Number of People at or above FEV ₁ Decrement					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
All Children	Boston	S75	2015	10	186109	106309	75021	57409	45736	38023
All Children	Boston	S75	2015	15	64054	30969	20502	14654	10786	8328
All Children	Boston	S75	2015	20	26827	11491	7031	4278	3095	2321
All Children	Boston	S75	2016	10	199238	114159	80437	62506	48990	39661
All Children	Boston	S75	2016	15	68673	35156	23050	16656	12265	9602
All Children	Boston	S75	2016	20	30514	13857	8214	5484	3891	2822
All Children	Boston	S75	2017	10	217328	123124	84783	63849	49354	39547
All Children	Boston	S75	2017	15	80960	39820	25417	17384	13107	9989
All Children	Boston	S75	2017	20	39684	16702	9375	5893	3937	2662
All Children	Dallas	S65	2015	10	184931	109857	77865	59799	48355	40103
All Children	Dallas	S65	2015	15	58735	30621	20146	14258	10853	8489
All Children	Dallas	S65	2015	20	23362	10451	6242	4091	2885	2270
All Children	Dallas	S65	2016	10	158708	92406	66018	50956	41261	33600
All Children	Dallas	S65	2016	15	43957	20832	14400	10428	8087	6573
All Children	Dallas	S65	2016	20	16717	7236	3902	2790	1844	1395
All Children	Dallas	S65	2017	10	183891	109739	78621	61100	50128	41238
All Children	Dallas	S65	2017	15	57482	29084	19247	13738	10617	8796
All Children	Dallas	S65	2017	20	23196	10499	6857	4469	3074	2365
All Children	Dallas	S70	2015	10	222622	134637	98578	77108	62660	52209
All Children	Dallas	S70	2015	15	76942	42869	28587	21352	16812	13123
All Children	Dallas	S70	2015	20	34853	16410	10215	7094	5013	4067
All Children	Dallas	S70	2016	10	185830	109975	80300	62140	50625	42207
All Children	Dallas	S70	2016	15	57317	28658	19484	14282	11232	9174
All Children	Dallas	S70	2016	20	22747	10475	6597	4540	3310	2246
All Children	Dallas	S70	2017	10	213211	130192	95173	75878	61833	51760
All Children	Dallas	S70	2017	15	72473	38968	25986	19271	14707	11752
All Children	Dallas	S70	2017	20	31165	14329	9198	6668	4753	3760
All Children	Dallas	S75	2015	10	257783	161049	118582	94062	77628	65380
All Children	Dallas	S75	2015	15	96899	56182	38637	28753	22794	18183
All Children	Dallas	S75	2015	20	45612	23527	15393	10428	7921	6195
All Children	Dallas	S75	2016	10	211343	127142	93305	72828	60367	50861
All Children	Dallas	S75	2016	15	70274	37005	25230	18562	14873	11941
All Children	Dallas	S75	2016	20	29770	13785	8796	6171	4635	3570
All Children	Dallas	S75	2017	10	240285	148494	111110	88718	73230	61927
All Children	Dallas	S75	2017	15	86992	48000	32252	24449	19035	15393
All Children	Dallas	S75	2017	20	39819	19295	12154	8938	7023	5391
All Children	Detroit	S65	2015	10	124524	70240	49393	37392	29483	24523
All Children	Detroit	S65	2015	15	37045	18592	11707	7943	6018	4527
All Children	Detroit	S65	2015	20	14117	6174	3399	2012	1474	1075
All Children	Detroit	S65	2016	10	144399	83143	58585	44364	35710	29778
All Children	Detroit	S65	2016	15	46514	23205	14828	10510	7995	6174
All Children	Detroit	S65	2016	20	18696	8186	4891	3278	2255	1544
All Children	Detroit	S65	2017	10	133039	77108	54561	42525	34322	28079
All Children	Detroit	S65	2017	15	40548	20829	13649	9920	7718	5931
All Children	Detroit	S65	2017	20	16979	7683	4475	2896	2168	1665

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All Children	Detroit	S70	2015	10	149480	87982	63337	48613	38883	32067
All Children	Detroit	S70	2015	15	50434	26015	17326	12574	9348	7458
All Children	Detroit	S70	2015	20	21037	9729	5689	3885	2706	1908
All Children	Detroit	S70	2016	10	176362	107267	76709	59001	47659	40028
All Children	Detroit	S70	2016	15	63632	33819	22928	16025	12504	9816
All Children	Detroit	S70	2016	20	28547	13302	8394	5584	4041	3052
All Children	Detroit	S70	2017	10	162228	97798	69650	54561	44121	36715
All Children	Detroit	S70	2017	15	56018	30004	19910	14759	11533	9053
All Children	Detroit	S70	2017	20	24541	11897	7458	5047	3659	2567
All Children	Detroit	S75	2015	10	168471	100070	73101	56157	45734	37531
All Children	Detroit	S75	2015	15	61048	31929	21922	16129	12123	9747
All Children	Detroit	S75	2015	20	26587	12574	7562	5134	3746	2740
All Children	Detroit	S75	2016	10	202568	126674	91797	71749	58082	48387
All Children	Detroit	S75	2016	15	80143	44017	29986	22130	17100	13649
All Children	Detroit	S75	2016	20	38415	18991	12106	8221	6001	4665
All Children	Detroit	S75	2017	10	185519	113355	82189	64621	52342	43601
All Children	Detroit	S75	2017	15	68679	38727	25703	18522	14516	11811
All Children	Detroit	S75	2017	20	32119	16112	10389	7197	5238	3885
All Children	Philadelphia	S65	2015	10	164741	96099	67878	51749	41556	34419
All Children	Philadelphia	S65	2015	15	46620	23834	15693	11197	8447	6526
All Children	Philadelphia	S65	2015	20	17351	7595	4496	2968	2030	1484
All Children	Philadelphia	S65	2016	10	162035	94266	65084	49872	40749	33612
All Children	Philadelphia	S65	2016	15	45921	23244	15060	10760	8294	6810
All Children	Philadelphia	S65	2016	20	17526	7923	4365	2837	1899	1462
All Children	Philadelphia	S65	2017	10	150445	85099	60021	45267	36493	29356
All Children	Philadelphia	S65	2017	15	42648	20691	12877	9123	6701	5347
All Children	Philadelphia	S65	2017	20	16348	6831	3841	2532	1724	1331
All Children	Philadelphia	S70	2015	10	196192	118929	85841	66547	53888	44568
All Children	Philadelphia	S70	2015	15	62400	32913	22437	16544	12550	10258
All Children	Philadelphia	S70	2015	20	25449	11677	7159	5282	3754	2619
All Children	Philadelphia	S70	2016	10	193355	116397	82458	63404	52076	43586
All Children	Philadelphia	S70	2016	15	61221	32302	21935	15955	12681	9800
All Children	Philadelphia	S70	2016	20	25012	11677	7312	5020	3579	2488
All Children	Philadelphia	S70	2017	10	178688	104546	74775	56943	46358	37955
All Children	Philadelphia	S70	2017	15	56856	29508	19119	13576	10280	7857
All Children	Philadelphia	S70	2017	20	23615	10324	5653	4038	2903	2248
All Children	Philadelphia	S75	2015	10	237072	147695	109936	87085	71720	59824
All Children	Philadelphia	S75	2015	15	84204	46162	32499	24423	18923	15584
All Children	Philadelphia	S75	2015	20	37278	18814	11873	8599	6482	5085
All Children	Philadelphia	S75	2016	10	232750	145032	106575	83855	67987	57904
All Children	Philadelphia	S75	2016	15	83440	46074	32084	24161	18552	15016
All Children	Philadelphia	S75	2016	20	37628	18574	11742	8425	6242	4889
All Children	Philadelphia	S75	2017	10	216861	129929	94615	73793	59759	49828
All Children	Philadelphia	S75	2017	15	77525	42058	27042	19927	15453	12070
All Children	Philadelphia	S75	2017	20	35358	16326	9647	6548	4933	3776

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All Children	Phoenix	S65	2015	10	141180	90695	69549	55764	47541	40592
All Children	Phoenix	S65	2015	15	43974	25150	18145	14196	11436	9398
All Children	Phoenix	S65	2015	20	17423	9115	6015	4529	3482	2760
All Children	Phoenix	S65	2016	10	135773	87001	66676	54349	45390	39106
All Children	Phoenix	S65	2016	15	39856	23424	16970	13035	10601	9143
All Children	Phoenix	S65	2016	20	15484	8166	5463	4161	3298	2831
All Children	Phoenix	S65	2017	10	153889	101649	78027	63633	54306	47187
All Children	Phoenix	S65	2017	15	48588	29524	21230	16927	13899	11860
All Children	Phoenix	S65	2017	20	20041	11054	7459	5308	4232	3453
All Children	Phoenix	S70	2015	10	170378	112646	87680	72125	61256	52933
All Children	Phoenix	S70	2015	15	59798	36034	26070	20409	16956	14535
All Children	Phoenix	S70	2015	20	25844	14493	9851	7501	5959	4869
All Children	Phoenix	S70	2016	10	164589	108372	83873	69479	58906	50980
All Children	Phoenix	S70	2016	15	54688	32468	23990	18895	15781	13417
All Children	Phoenix	S70	2016	20	22773	12596	8860	6893	5746	4572
All Children	Phoenix	S70	2017	10	185182	125399	98889	81962	70399	61921
All Children	Phoenix	S70	2017	15	65827	41342	31251	24698	21060	17932
All Children	Phoenix	S70	2017	20	30302	17210	12384	9469	7530	6128
All Children	Phoenix	S75	2015	10	197312	132787	104721	87482	74135	65219
All Children	Phoenix	S75	2015	15	76117	47484	34930	27712	22872	19645
All Children	Phoenix	S75	2015	20	36105	20607	14351	11153	8747	7218
All Children	Phoenix	S75	2016	10	190306	128668	99908	83137	71389	62869
All Children	Phoenix	S75	2016	15	69167	42771	31619	25901	21456	18399
All Children	Phoenix	S75	2016	20	31576	18130	12894	10006	8152	6949
All Children	Phoenix	S75	2017	10	213036	148242	118449	99271	85684	75848
All Children	Phoenix	S75	2017	15	84439	53528	40889	33374	28080	24188
All Children	Phoenix	S75	2017	20	40549	24499	17762	14012	11337	9568
All Children	Sacramento	S65	2015	10	48758	27826	19938	15808	12896	10645
All Children	Sacramento	S65	2015	15	12919	6250	3804	2873	2205	1755
All Children	Sacramento	S65	2015	20	4193	1716	1040	714	528	373
All Children	Sacramento	S65	2016	10	51701	30303	21444	16406	13269	10986
All Children	Sacramento	S65	2016	15	13828	6972	4441	3253	2601	2112
All Children	Sacramento	S65	2016	20	4985	2244	1328	916	637	458
All Children	Sacramento	S65	2017	10	50614	29030	20132	15559	12640	10536
All Children	Sacramento	S65	2017	15	12896	6367	3898	2896	2073	1623
All Children	Sacramento	S65	2017	20	4255	1677	1017	637	435	334
All Children	Sacramento	S70	2015	10	64364	38712	28059	22182	18649	15862
All Children	Sacramento	S70	2015	15	20024	10559	7267	5311	4169	3269
All Children	Sacramento	S70	2015	20	7904	3540	2213	1599	1188	916
All Children	Sacramento	S70	2016	10	68293	41406	30466	24045	19767	16638
All Children	Sacramento	S70	2016	15	21871	11848	7896	5955	4705	3797
All Children	Sacramento	S70	2016	20	9286	4371	2756	1988	1467	1157
All Children	Sacramento	S70	2017	10	67066	40909	29030	23036	18975	15901
All Children	Sacramento	S70	2017	15	20101	10567	7275	5326	4169	3292
All Children	Sacramento	S70	2017	20	8230	3556	2135	1522	1149	815

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All Children	Sacramento	S75	2015	10	77253	48300	35521	28432	23649	20520
All Children	Sacramento	S75	2015	15	26693	14992	10497	7958	6165	5008
All Children	Sacramento	S75	2015	20	11755	5769	3758	2694	2073	1568
All Children	Sacramento	S75	2016	10	82439	51996	38867	31118	25746	21623
All Children	Sacramento	S75	2016	15	29504	16685	11871	8929	6995	5629
All Children	Sacramento	S75	2016	20	13354	6980	4410	3284	2547	2065
All Children	Sacramento	S75	2017	10	81057	50839	37586	29667	24511	20947
All Children	Sacramento	S75	2017	15	27539	15311	10567	8036	6250	5194
All Children	Sacramento	S75	2017	20	11957	5893	3711	2702	2026	1514
All Children	St. Louis	S65	2015	10	61187	34086	23668	18195	14543	11702
All Children	St. Louis	S65	2015	15	17066	8542	5555	4062	2951	2249
All Children	St. Louis	S65	2015	20	6338	2832	1776	1157	892	583
All Children	St. Louis	S65	2016	10	71851	42218	30070	23149	18696	15508
All Children	St. Louis	S65	2016	15	22839	11584	7750	5418	3980	2941
All Children	St. Louis	S65	2016	20	9243	3779	2195	1357	1020	719
All Children	St. Louis	S65	2017	10	69365	40633	28822	21901	17621	14680
All Children	St. Louis	S65	2017	15	19716	10081	6548	4908	3816	3023
All Children	St. Louis	S65	2017	20	7668	3542	2268	1421	1047	838
All Children	St. Louis	S70	2015	10	76632	44731	31691	24579	19861	16574
All Children	St. Louis	S70	2015	15	24351	12704	8587	6356	4963	3843
All Children	St. Louis	S70	2015	20	10309	4826	3078	2158	1557	1175
All Children	St. Louis	S70	2016	10	89017	54612	40087	31582	25517	21382
All Children	St. Louis	S70	2016	15	32356	17175	11729	8587	6739	5282
All Children	St. Louis	S70	2016	20	14507	6775	4180	2732	2013	1475
All Children	St. Louis	S70	2017	10	87368	53055	38393	30352	24742	20390
All Children	St. Louis	S70	2017	15	28303	15508	10409	7932	6047	4918
All Children	St. Louis	S70	2017	20	11738	5783	3907	2623	1994	1548
All Children	St. Louis	S75	2015	10	88406	53228	38621	30452	24697	20526
All Children	St. Louis	S75	2015	15	31172	16610	11319	8515	6739	5282
All Children	St. Louis	S75	2015	20	13724	6848	4462	3087	2176	1694
All Children	St. Louis	S75	2016	10	102931	65203	48420	38430	31627	26600
All Children	St. Louis	S75	2016	15	40724	22557	15417	11638	9143	7422
All Children	St. Louis	S75	2016	20	19433	9744	6338	4380	3151	2486
All Children	St. Louis	S75	2017	10	101192	63691	46890	37055	30625	25726
All Children	St. Louis	S75	2017	15	36080	20289	13833	10536	8405	6739
All Children	St. Louis	S75	2017	20	15845	8269	5409	3779	2941	2359
Asthma Children	Atlanta	S65	2015	10	15415	8030	5306	3975	2905	2058
Asthma Children	Atlanta	S65	2015	15	3712	1574	1009	625	424	282
Asthma Children	Atlanta	S65	2015	20	1271	363	222	182	161	101
Asthma Children	Atlanta	S65	2016	10	18966	10835	7869	6154	5044	4298
Asthma Children	Atlanta	S65	2016	15	5811	2825	1937	1513	1130	948
Asthma Children	Atlanta	S65	2016	20	2300	1009	545	343	282	202
Asthma Children	Atlanta	S65	2017	10	13619	7465	5165	3773	3127	2522
Asthma Children	Atlanta	S65	2017	15	3612	1796	1150	807	605	343
Asthma Children	Atlanta	S65	2017	20	1190	464	262	202	202	101

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Asthma Children	Atlanta	S70	2015	10	19834	11218	7607	5750	4661	3652
Asthma Children	Atlanta	S70	2015	15	5831	2583	1776	1291	948	706
Asthma Children	Atlanta	S70	2015	20	2199	807	484	242	161	141
Asthma Children	Atlanta	S70	2016	10	23929	14608	10673	8636	7203	5972
Asthma Children	Atlanta	S70	2016	15	8434	4681	3127	2421	1876	1614
Asthma Children	Atlanta	S70	2016	20	3733	1715	1130	847	625	484
Asthma Children	Atlanta	S70	2017	10	17776	9725	7183	5710	4580	3672
Asthma Children	Atlanta	S70	2017	15	4802	2361	1836	1352	1049	747
Asthma Children	Atlanta	S70	2017	20	2078	847	484	262	242	222
Asthma Children	Atlanta	S75	2015	10	24393	14446	10593	8272	6658	5468
Asthma Children	Atlanta	S75	2015	15	8615	4298	2845	2058	1634	1291
Asthma Children	Atlanta	S75	2015	20	3390	1453	928	686	444	262
Asthma Children	Atlanta	S75	2016	10	28994	18764	14083	11501	9584	8071
Asthma Children	Atlanta	S75	2016	15	11723	6638	4661	3672	2986	2583
Asthma Children	Atlanta	S75	2016	20	5448	2724	1876	1392	1090	888
Asthma Children	Atlanta	S75	2017	10	21730	12994	9241	7445	6134	5044
Asthma Children	Atlanta	S75	2017	15	6820	3672	2562	1997	1574	1251
Asthma Children	Atlanta	S75	2017	20	3087	1554	928	565	424	282
Asthma Children	Boston	S65	2015	10	18499	10353	7418	5438	4323	3663
Asthma Children	Boston	S65	2015	15	5416	2366	1707	1069	728	592
Asthma Children	Boston	S65	2015	20	2184	910	432	341	250	159
Asthma Children	Boston	S65	2016	10	19751	10217	7463	5939	4665	3823
Asthma Children	Boston	S65	2016	15	5643	3095	2162	1502	1001	683
Asthma Children	Boston	S65	2016	20	2298	956	523	319	182	137
Asthma Children	Boston	S65	2017	10	20047	11286	7463	5552	4278	3322
Asthma Children	Boston	S65	2017	15	6963	2822	1707	1320	887	592
Asthma Children	Boston	S65	2017	20	2457	1024	569	319	296	228
Asthma Children	Boston	S70	2015	10	21366	12788	9125	6849	5598	4665
Asthma Children	Boston	S70	2015	15	7259	3390	2298	1525	1251	933
Asthma Children	Boston	S70	2015	20	2844	1434	796	432	341	228
Asthma Children	Boston	S70	2016	10	24165	13243	9375	7350	6075	4847
Asthma Children	Boston	S70	2016	15	7691	4210	2913	2230	1684	1229
Asthma Children	Boston	S70	2016	20	3368	1570	887	501	364	228
Asthma Children	Boston	S70	2017	10	24529	14267	9898	7372	5734	4619
Asthma Children	Boston	S70	2017	15	9056	3937	2366	1707	1365	1092
Asthma Children	Boston	S70	2017	20	3959	1547	887	592	410	296
Asthma Children	Boston	S75	2015	10	23483	13835	10331	7759	6235	5347
Asthma Children	Boston	S75	2015	15	8146	4119	2640	1934	1525	1092
Asthma Children	Boston	S75	2015	20	3413	1661	1001	546	455	319
Asthma Children	Boston	S75	2016	10	26827	14972	10626	8920	6986	5552
Asthma Children	Boston	S75	2016	15	9284	5006	3527	2708	2116	1570
Asthma Children	Boston	S75	2016	20	4187	2002	1229	751	523	364
Asthma Children	Boston	S75	2017	10	27965	16429	11332	8669	6599	5438
Asthma Children	Boston	S75	2017	15	10626	5165	3004	2071	1616	1388
Asthma Children	Boston	S75	2017	20	5256	2048	1229	774	478	341

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Asthma Children	Dallas	S65	2015	10	18869	11042	8441	6361	5155	4398
Asthma Children	Dallas	S65	2015	15	6030	3050	2010	1348	1111	969
Asthma Children	Dallas	S65	2015	20	2530	1111	686	426	378	307
Asthma Children	Dallas	S65	2016	10	15393	8985	6881	5249	4280	3452
Asthma Children	Dallas	S65	2016	15	4753	2435	1537	1040	828	567
Asthma Children	Dallas	S65	2016	20	1750	615	284	236	142	95
Asthma Children	Dallas	S65	2017	10	18443	11539	8087	6361	5533	4540
Asthma Children	Dallas	S65	2017	15	5911	3405	2270	1726	1419	1159
Asthma Children	Dallas	S65	2017	20	2719	1301	828	520	355	307
Asthma Children	Dallas	S70	2015	10	22298	13785	10309	8158	6715	5509
Asthma Children	Dallas	S70	2015	15	7779	4587	2979	1986	1655	1301
Asthma Children	Dallas	S70	2015	20	3689	1679	1040	851	567	473
Asthma Children	Dallas	S70	2016	10	17781	10570	7921	6432	5438	4374
Asthma Children	Dallas	S70	2016	15	5911	3121	2104	1442	1111	875
Asthma Children	Dallas	S70	2016	20	2388	1017	615	426	260	142
Asthma Children	Dallas	S70	2017	10	21376	13241	9647	7779	6597	5557
Asthma Children	Dallas	S70	2017	15	7590	4445	2932	2128	1797	1513
Asthma Children	Dallas	S70	2017	20	3547	1773	1230	828	567	473
Asthma Children	Dallas	S75	2015	10	25915	16221	12438	9624	8110	6857
Asthma Children	Dallas	S75	2015	15	9931	5651	4138	3192	2365	1821
Asthma Children	Dallas	S75	2015	20	4682	2317	1466	993	851	733
Asthma Children	Dallas	S75	2016	10	20406	12319	9080	7212	6242	5202
Asthma Children	Dallas	S75	2016	15	7354	3902	2696	2104	1537	1230
Asthma Children	Dallas	S75	2016	20	3168	1395	875	615	402	260
Asthma Children	Dallas	S75	2017	10	23835	15015	11184	9056	7567	6479
Asthma Children	Dallas	S75	2017	15	8914	5084	3783	2956	2246	1892
Asthma Children	Dallas	S75	2017	20	4398	2341	1537	1111	899	686
Asthma Children	Detroit	S65	2015	10	14811	8741	6417	4856	3642	3087
Asthma Children	Detroit	S65	2015	15	4908	2237	1492	1075	798	590
Asthma Children	Detroit	S65	2015	20	1700	780	451	260	173	87
Asthma Children	Detroit	S65	2016	10	17985	10649	7475	5567	4422	3850
Asthma Children	Detroit	S65	2016	15	6122	3295	2046	1353	1058	798
Asthma Children	Detroit	S65	2016	20	2567	1197	746	382	243	191
Asthma Children	Detroit	S65	2017	10	16441	9782	6868	5376	4301	3469
Asthma Children	Detroit	S65	2017	15	5238	2549	1734	1283	1041	746
Asthma Children	Detroit	S65	2017	20	2133	1058	624	382	277	225
Asthma Children	Detroit	S70	2015	10	17603	10649	8134	6226	4839	3989
Asthma Children	Detroit	S70	2015	15	6521	3295	2046	1596	1127	902
Asthma Children	Detroit	S70	2015	20	2653	1214	798	468	382	156
Asthma Children	Detroit	S70	2016	10	21662	13510	9938	7423	5827	5047
Asthma Children	Detroit	S70	2016	15	8151	4544	3052	2151	1682	1335
Asthma Children	Detroit	S70	2016	20	3902	1821	1145	780	468	382
Asthma Children	Detroit	S70	2017	10	19841	11949	8672	6781	5723	4683
Asthma Children	Detroit	S70	2017	15	7007	3711	2497	1960	1613	1179
Asthma Children	Detroit	S70	2017	20	3035	1509	1006	590	468	347

Study Group	Study Area	AQ Scenario	Year	FEV ₁ (percent)	Number of People at or above FEV ₁ Decrement					
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Asthma Children	Detroit	S75	2015	10	20083	12088	9157	7128	5706	4613
Asthma Children	Detroit	S75	2015	15	7666	4006	2688	1942	1509	1162
Asthma Children	Detroit	S75	2015	20	3295	1509	989	607	503	347
Asthma Children	Detroit	S75	2016	10	24575	15539	11672	9070	7249	5845
Asthma Children	Detroit	S75	2016	15	10319	5584	4024	3000	2203	1925
Asthma Children	Detroit	S75	2016	20	5064	2740	1630	1075	798	572
Asthma Children	Detroit	S75	2017	10	22355	13857	9972	7995	6799	5567
Asthma Children	Detroit	S75	2017	15	8654	4856	3330	2428	1942	1578
Asthma Children	Detroit	S75	2017	20	3972	1942	1422	989	624	486
Asthma Children	Philadelphia	S65	2015	10	19294	12135	8294	6111	4780	4038
Asthma Children	Philadelphia	S65	2015	15	5478	2706	2008	1528	1135	960
Asthma Children	Philadelphia	S65	2015	20	2139	1069	611	415	284	175
Asthma Children	Philadelphia	S65	2016	10	18683	10804	7181	5195	4169	3558
Asthma Children	Philadelphia	S65	2016	15	4758	2488	1659	1179	851	611
Asthma Children	Philadelphia	S65	2016	20	1877	808	437	262	196	175
Asthma Children	Philadelphia	S65	2017	10	18355	10411	7377	5937	4693	3710
Asthma Children	Philadelphia	S65	2017	15	5195	2706	1659	1244	982	786
Asthma Children	Philadelphia	S65	2017	20	2357	917	567	371	153	109
Asthma Children	Philadelphia	S70	2015	10	23135	14449	10651	8272	6439	5173
Asthma Children	Philadelphia	S70	2015	15	7923	3841	2859	2183	1812	1375
Asthma Children	Philadelphia	S70	2015	20	2903	1375	960	786	589	349
Asthma Children	Philadelphia	S70	2016	10	22175	13816	9669	7093	5435	4649
Asthma Children	Philadelphia	S70	2016	15	6722	3318	2292	1790	1310	1048
Asthma Children	Philadelphia	S70	2016	20	2575	1091	698	458	306	240
Asthma Children	Philadelphia	S70	2017	10	21869	12572	9320	7181	6002	4867
Asthma Children	Philadelphia	S70	2017	15	6701	3820	2488	1746	1484	1157
Asthma Children	Philadelphia	S70	2017	20	3099	1484	720	546	393	327
Asthma Children	Philadelphia	S75	2015	10	27850	17766	13510	10826	8796	7159
Asthma Children	Philadelphia	S75	2015	15	10280	5347	3841	2925	2401	2117
Asthma Children	Philadelphia	S75	2015	20	4343	2226	1484	1069	917	742
Asthma Children	Philadelphia	S75	2016	10	26911	17199	12593	9669	7639	6395
Asthma Children	Philadelphia	S75	2016	15	9712	4823	3427	2575	2052	1637
Asthma Children	Philadelphia	S75	2016	20	4147	1855	1310	873	502	458
Asthma Children	Philadelphia	S75	2017	10	26191	15889	11677	9276	7508	6199
Asthma Children	Philadelphia	S75	2017	15	9516	5282	3470	2510	2183	1746
Asthma Children	Philadelphia	S75	2017	20	4452	2204	1353	939	720	524
Asthma Children	Phoenix	S65	2015	10	15215	9992	7685	6086	5223	4444
Asthma Children	Phoenix	S65	2015	15	4982	2972	2066	1656	1373	1118
Asthma Children	Phoenix	S65	2015	20	1882	1090	722	538	467	368
Asthma Children	Phoenix	S65	2016	10	13785	9058	7119	5817	4982	4119
Asthma Children	Phoenix	S65	2016	15	4331	2717	1840	1514	1189	1005
Asthma Children	Phoenix	S65	2016	20	1727	920	580	396	340	269
Asthma Children	Phoenix	S65	2017	10	16064	10502	8166	6666	5732	4925
Asthma Children	Phoenix	S65	2017	15	5223	3255	2392	1882	1557	1373
Asthma Children	Phoenix	S65	2017	20	2335	1231	977	736	637	538

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Asthma Children	Phoenix	S70	2015	10	18201	12200	9539	7940	6695	5746
Asthma Children	Phoenix	S70	2015	15	6836	4161	2930	2293	1911	1571
Asthma Children	Phoenix	S70	2015	20	2930	1684	1104	878	708	566
Asthma Children	Phoenix	S70	2016	10	16460	11025	8846	7445	6298	5506
Asthma Children	Phoenix	S70	2016	15	6114	3552	2689	2081	1783	1543
Asthma Children	Phoenix	S70	2016	20	2434	1429	991	750	651	538
Asthma Children	Phoenix	S70	2017	10	18994	13049	10346	8534	7331	6341
Asthma Children	Phoenix	S70	2017	15	7119	4331	3354	2703	2307	2024
Asthma Children	Phoenix	S70	2017	20	3199	1996	1429	1033	934	807
Asthma Children	Phoenix	S75	2015	10	20735	14139	11294	9539	8294	7289
Asthma Children	Phoenix	S75	2015	15	8492	5378	3935	3114	2548	2137
Asthma Children	Phoenix	S75	2015	20	4331	2307	1613	1203	934	793
Asthma Children	Phoenix	S75	2016	10	18937	12964	10473	8648	7530	6610
Asthma Children	Phoenix	S75	2016	15	7473	4727	3482	2873	2349	2052
Asthma Children	Phoenix	S75	2016	20	3383	1953	1401	1146	948	764
Asthma Children	Phoenix	S75	2017	10	21513	15328	12214	10219	8874	7813
Asthma Children	Phoenix	S75	2017	15	8931	5506	4444	3666	3057	2604
Asthma Children	Phoenix	S75	2017	20	4303	2802	2052	1656	1288	1132
Asthma Children	Sacramento	S65	2015	10	4891	2725	1988	1630	1328	1126
Asthma Children	Sacramento	S65	2015	15	1250	567	303	248	194	124
Asthma Children	Sacramento	S65	2015	20	334	124	70	31	23	16
Asthma Children	Sacramento	S65	2016	10	5256	3075	2189	1731	1429	1211
Asthma Children	Sacramento	S65	2016	15	1413	745	528	427	365	280
Asthma Children	Sacramento	S65	2016	20	590	272	163	124	78	47
Asthma Children	Sacramento	S65	2017	10	5039	2896	1933	1460	1219	986
Asthma Children	Sacramento	S65	2017	15	1242	551	349	287	248	194
Asthma Children	Sacramento	S65	2017	20	419	194	140	70	54	31
Asthma Children	Sacramento	S70	2015	10	6328	3773	2686	2174	1871	1638
Asthma Children	Sacramento	S70	2015	15	1988	1048	714	520	435	280
Asthma Children	Sacramento	S70	2015	20	699	233	155	140	101	70
Asthma Children	Sacramento	S70	2016	10	6972	4294	3113	2453	2096	1731
Asthma Children	Sacramento	S70	2016	15	2244	1320	831	683	551	466
Asthma Children	Sacramento	S70	2016	20	978	536	373	303	225	163
Asthma Children	Sacramento	S70	2017	10	6576	4053	2873	2244	1794	1506
Asthma Children	Sacramento	S70	2017	15	1941	994	629	481	388	311
Asthma Children	Sacramento	S70	2017	20	745	334	225	179	163	132
Asthma Children	Sacramento	S75	2015	10	7477	4635	3432	2795	2376	2057
Asthma Children	Sacramento	S75	2015	15	2733	1530	1040	792	582	474
Asthma Children	Sacramento	S75	2015	20	1064	551	311	210	155	101
Asthma Children	Sacramento	S75	2016	10	8440	5427	4006	3238	2725	2244
Asthma Children	Sacramento	S75	2016	15	2950	1747	1250	955	784	637
Asthma Children	Sacramento	S75	2016	20	1312	745	520	396	334	303
Asthma Children	Sacramento	S75	2017	10	7772	5047	3758	2927	2368	1980
Asthma Children	Sacramento	S75	2017	15	2686	1522	978	691	543	466
Asthma Children	Sacramento	S75	2017	20	1149	505	349	264	225	194

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Asthma Children	St. Louis	S65	2015	10	6730	3843	2659	2158	1794	1412
Asthma Children	St. Louis	S65	2015	15	1821	993	665	464	346	282
Asthma Children	St. Louis	S65	2015	20	729	337	219	146	109	73
Asthma Children	St. Louis	S65	2016	10	7149	4171	3133	2431	2022	1758
Asthma Children	St. Louis	S65	2016	15	2340	1202	920	610	474	301
Asthma Children	St. Louis	S65	2016	20	1002	410	237	137	100	91
Asthma Children	St. Louis	S65	2017	10	7422	4235	2987	2441	2031	1685
Asthma Children	St. Louis	S65	2017	15	2204	1120	738	528	410	319
Asthma Children	St. Louis	S65	2017	20	838	355	246	164	127	82
Asthma Children	St. Louis	S70	2015	10	8278	4863	3624	2741	2249	1958
Asthma Children	St. Louis	S70	2015	15	2650	1421	956	747	583	455
Asthma Children	St. Louis	S70	2015	20	1157	501	346	228	191	164
Asthma Children	St. Louis	S70	2016	10	9043	5437	4034	3151	2705	2277
Asthma Children	St. Louis	S70	2016	15	3206	1721	1275	965	829	647
Asthma Children	St. Louis	S70	2016	20	1512	747	474	319	200	182
Asthma Children	St. Louis	S70	2017	10	9234	5509	3961	3096	2614	2222
Asthma Children	St. Louis	S70	2017	15	3169	1739	1166	865	610	519
Asthma Children	St. Louis	S70	2017	20	1311	610	446	319	219	173
Asthma Children	St. Louis	S75	2015	10	9371	5628	4289	3315	2705	2349
Asthma Children	St. Louis	S75	2015	15	3442	1921	1320	1029	783	592
Asthma Children	St. Louis	S75	2015	20	1503	783	455	337	246	209
Asthma Children	St. Louis	S75	2016	10	10400	6511	4763	3743	3160	2668
Asthma Children	St. Louis	S75	2016	15	4025	2277	1603	1311	1065	865
Asthma Children	St. Louis	S75	2016	20	1994	1084	738	501	346	264
Asthma Children	St. Louis	S75	2017	10	10591	6602	4836	3770	3151	2759
Asthma Children	St. Louis	S75	2017	15	3934	2186	1585	1211	938	701
Asthma Children	St. Louis	S75	2017	20	1785	956	628	419	328	246
All Adults	Atlanta	S65	2015	10	75293	32047	19440	12960	8663	5846
All Adults	Atlanta	S65	2015	15	19228	6480	2676	1831	1268	1057
All Adults	Atlanta	S65	2015	20	7325	2324	1127	704	423	211
All Adults	Atlanta	S65	2016	10	89662	43387	26835	18665	14016	11340
All Adults	Atlanta	S65	2016	15	24088	10565	6550	4015	3099	2465
All Adults	Atlanta	S65	2016	20	9649	3522	2043	1409	1127	916
All Adults	Atlanta	S65	2017	10	74941	32118	20355	13101	8522	6691
All Adults	Atlanta	S65	2017	15	17608	6198	3029	1902	1127	845
All Adults	Atlanta	S65	2017	20	5564	1550	634	211	141	141
All Adults	Atlanta	S70	2015	10	101142	43528	27046	18947	13594	9861
All Adults	Atlanta	S70	2015	15	27187	10354	5494	3029	1831	1268
All Adults	Atlanta	S70	2015	20	10988	3451	1620	1268	986	704
All Adults	Atlanta	S70	2016	10	117483	58037	37752	26553	20426	16693
All Adults	Atlanta	S70	2016	15	35639	15495	9790	7325	5212	4015
All Adults	Atlanta	S70	2016	20	15284	6198	3592	2395	1550	1338
All Adults	Atlanta	S70	2017	10	97903	44444	27539	20496	13735	10283
All Adults	Atlanta	S70	2017	15	25779	10424	5423	3522	1902	1268
All Adults	Atlanta	S70	2017	20	8804	3029	1338	634	141	141

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All Adults	Atlanta	S75	2015	10	129598	60361	37048	25849	19228	14791
All Adults	Atlanta	S75	2015	15	39513	16200	9156	5635	3522	2113
All Adults	Atlanta	S75	2015	20	16974	5283	2536	1690	1338	986
All Adults	Atlanta	S75	2016	10	153404	76491	49726	36414	27610	22116
All Adults	Atlanta	S75	2016	15	49796	22116	14298	9790	7536	5635
All Adults	Atlanta	S75	2016	20	22046	9790	6128	3733	2606	1761
All Adults	Atlanta	S75	2017	10	121286	57333	35498	26342	19017	14791
All Adults	Atlanta	S75	2017	15	35146	14650	8522	5705	3592	2465
All Adults	Atlanta	S75	2017	20	13805	5212	2606	1550	634	493
All Adults	Boston	S65	2015	10	104195	46374	26611	17904	13599	10958
All Adults	Boston	S65	2015	15	25535	9881	5577	2642	1761	1272
All Adults	Boston	S65	2015	20	9294	2935	1370	587	391	294
All Adults	Boston	S65	2016	10	114272	50287	32286	21426	16828	12523
All Adults	Boston	S65	2016	15	31209	14186	7729	4598	2935	2055
All Adults	Boston	S65	2016	20	13893	4892	1957	978	783	489
All Adults	Boston	S65	2017	10	123175	51951	30818	21426	15849	12229
All Adults	Boston	S65	2017	15	30427	11740	7142	4990	3326	2348
All Adults	Boston	S65	2017	20	12816	4500	2739	1663	881	587
All Adults	Boston	S70	2015	10	121022	55473	34047	22111	16339	12816
All Adults	Boston	S70	2015	15	32286	12621	6946	4403	2544	1957
All Adults	Boston	S70	2015	20	12229	4305	2152	1174	881	587
All Adults	Boston	S70	2016	10	136285	60756	39917	28079	21622	16436
All Adults	Boston	S70	2016	15	39036	17513	10958	6457	4109	2642
All Adults	Boston	S70	2016	20	18295	6164	3131	1859	881	685
All Adults	Boston	S70	2017	10	149395	63495	37275	25731	19763	15752
All Adults	Boston	S70	2017	15	41874	16143	9784	5968	4696	3424
All Adults	Boston	S70	2017	20	17806	5968	3620	2544	1468	881
All Adults	Boston	S75	2015	10	133448	60169	36884	23774	18882	15262
All Adults	Boston	S75	2015	15	37667	14969	8120	5087	3033	2152
All Adults	Boston	S75	2015	20	14773	5381	2544	1370	881	587
All Adults	Boston	S75	2016	10	152036	66430	43830	31405	23676	18882
All Adults	Boston	S75	2016	15	44319	19763	12229	7631	5577	3424
All Adults	Boston	S75	2016	20	21132	7925	4109	2446	1468	587
All Adults	Boston	S75	2017	10	169060	72203	41776	29448	22502	17708
All Adults	Boston	S75	2017	15	49603	18491	11447	7142	5185	4109
All Adults	Boston	S75	2017	20	20741	7435	4305	3326	2055	1272
All Adults	Dallas	S65	2015	10	118845	55008	36177	23832	17346	12033
All Adults	Dallas	S65	2015	15	33677	13752	7110	4766	3360	2266
All Adults	Dallas	S65	2015	20	12346	3751	2032	1094	703	469
All Adults	Dallas	S65	2016	10	101734	46804	29067	18675	14611	10861
All Adults	Dallas	S65	2016	15	26488	10548	6095	4141	3125	2188
All Adults	Dallas	S65	2016	20	10001	3907	2110	938	625	391
All Adults	Dallas	S65	2017	10	118142	52898	33286	22972	16721	12814
All Adults	Dallas	S65	2017	15	31020	11173	6329	4454	2735	1797
All Adults	Dallas	S65	2017	20	11408	3751	1875	1328	781	469

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All Adults	Dallas	S70	2015	10	144083	68916	45397	31801	23363	17424
All Adults	Dallas	S70	2015	15	44694	18675	10627	6563	5235	3672
All Adults	Dallas	S70	2015	20	19222	6720	3360	2422	1406	781
All Adults	Dallas	S70	2016	10	119705	57118	34380	23519	17815	13986
All Adults	Dallas	S70	2016	15	33442	13908	7579	4923	3907	2735
All Adults	Dallas	S70	2016	20	13127	5391	2891	1485	1172	781
All Adults	Dallas	S70	2017	10	138145	64853	41569	29223	20862	16174
All Adults	Dallas	S70	2017	15	40553	15705	8048	5704	3594	2500
All Adults	Dallas	S70	2017	20	14690	5626	3516	1563	1094	781
All Adults	Dallas	S75	2015	10	170650	81652	54617	38912	29457	22347
All Adults	Dallas	S75	2015	15	56024	24457	13752	9064	6642	5079
All Adults	Dallas	S75	2015	20	25394	9142	4844	3047	2188	1250
All Adults	Dallas	S75	2016	10	137364	65635	40631	28520	21956	16956
All Adults	Dallas	S75	2016	15	40475	16721	9376	6095	5001	3125
All Adults	Dallas	S75	2016	20	17190	6954	3594	2032	1485	1094
All Adults	Dallas	S75	2017	10	156663	77511	49617	35474	26410	19768
All Adults	Dallas	S75	2017	15	49226	19768	10939	7657	5001	3438
All Adults	Dallas	S75	2017	20	19065	7110	3907	2266	1719	1250
All Adults	Detroit	S65	2015	10	77732	34344	20580	14222	10487	8717
All Adults	Detroit	S65	2015	15	19662	8127	3998	2491	1639	1376
All Adults	Detroit	S65	2015	20	6751	2491	1311	918	655	459
All Adults	Detroit	S65	2016	10	88481	40767	24644	16582	12387	9372
All Adults	Detroit	S65	2016	15	24119	10159	5505	3736	2097	1639
All Adults	Detroit	S65	2016	20	9569	3343	1966	1049	852	524
All Adults	Detroit	S65	2017	10	85663	38407	24185	16451	11863	9176
All Adults	Detroit	S65	2017	15	22284	9438	5243	3212	2556	1639
All Adults	Detroit	S65	2017	20	8651	3605	1573	786	393	197
All Adults	Detroit	S70	2015	10	97067	44634	26020	17893	13829	11339
All Adults	Detroit	S70	2015	15	26872	11142	5833	3801	2491	1835
All Adults	Detroit	S70	2015	20	10159	3867	1966	1114	852	590
All Adults	Detroit	S70	2016	10	110175	52368	33098	22743	17106	13829
All Adults	Detroit	S70	2016	15	33426	14681	7996	5571	3932	2818
All Adults	Detroit	S70	2016	20	13764	5571	2622	1704	1311	786
All Adults	Detroit	S70	2017	10	105128	48566	30739	21432	16320	12518
All Adults	Detroit	S70	2017	15	30936	13567	7472	5112	3605	2687
All Adults	Detroit	S70	2017	20	12060	4260	2359	1639	852	590
All Adults	Detroit	S75	2015	10	109454	50860	30477	20252	15664	12191
All Adults	Detroit	S75	2015	15	31722	13370	7210	4391	3212	2228
All Adults	Detroit	S75	2015	20	12846	4785	2622	1704	918	786
All Adults	Detroit	S75	2016	10	131476	61806	39915	27986	20383	15599
All Adults	Detroit	S75	2016	15	43192	18286	10290	6816	4916	3932
All Adults	Detroit	S75	2016	20	18745	7603	3605	2097	1639	1114
All Adults	Detroit	S75	2017	10	122300	56431	35917	25168	18745	14878
All Adults	Detroit	S75	2017	15	37162	16451	9045	6358	4260	3212
All Adults	Detroit	S75	2017	20	16582	5505	3080	2163	1180	852

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All Adults	Philadelphia	S65	2015	10	109538	48364	29628	20914	15947	12374
All Adults	Philadelphia	S65	2015	15	27711	10719	6884	4009	3050	1917
All Adults	Philadelphia	S65	2015	20	9760	3573	1917	1220	523	349
All Adults	Philadelphia	S65	2016	10	109712	49671	30326	22221	15773	12461
All Adults	Philadelphia	S65	2016	15	26317	10544	6100	4270	2527	1743
All Adults	Philadelphia	S65	2016	20	8889	3834	1743	1046	959	610
All Adults	Philadelphia	S65	2017	10	103874	49235	30761	21263	15250	11241
All Adults	Philadelphia	S65	2017	15	25620	10370	6361	3573	2266	1656
All Adults	Philadelphia	S65	2017	20	9499	2963	1656	871	436	261
All Adults	Philadelphia	S70	2015	10	135506	61261	37994	26840	20217	16208
All Adults	Philadelphia	S70	2015	15	37123	14640	9586	6100	4531	2963
All Adults	Philadelphia	S70	2015	20	14030	5054	2963	1917	1046	436
All Adults	Philadelphia	S70	2016	10	135506	62830	38691	27363	21524	16557
All Adults	Philadelphia	S70	2016	15	35903	13943	7669	5926	3921	2876
All Adults	Philadelphia	S70	2016	20	14030	5316	2701	1917	1394	1133
All Adults	Philadelphia	S70	2017	10	124004	60128	37820	27450	20653	15250
All Adults	Philadelphia	S70	2017	15	35118	13856	8714	5403	3311	2527
All Adults	Philadelphia	S70	2017	20	14466	4706	2440	1394	784	436
All Adults	Philadelphia	S75	2015	10	172542	78951	49410	35990	26578	20478
All Adults	Philadelphia	S75	2015	15	50194	20740	12723	8801	6013	4531
All Adults	Philadelphia	S75	2015	20	20827	8017	4967	2614	1917	1220
All Adults	Philadelphia	S75	2016	10	165658	80084	50107	35380	27450	22134
All Adults	Philadelphia	S75	2016	15	50804	21263	11677	7843	5926	4444
All Adults	Philadelphia	S75	2016	20	20043	7930	4531	3311	2179	1656
All Adults	Philadelphia	S75	2017	10	151541	74594	48103	34944	26666	20827
All Adults	Philadelphia	S75	2017	15	46883	19694	11241	7669	4706	3311
All Adults	Philadelphia	S75	2017	20	20304	7320	3660	1830	1220	959
All Adults	Phoenix	S65	2015	10	104253	55181	37151	28857	23046	18228
All Adults	Phoenix	S65	2015	15	28758	14255	9089	6407	4867	3377
All Adults	Phoenix	S65	2015	20	11225	4619	2881	1689	993	646
All Adults	Phoenix	S65	2016	10	99584	55429	37549	28708	22897	18774
All Adults	Phoenix	S65	2016	15	29254	13261	8543	6258	4520	3775
All Adults	Phoenix	S65	2016	20	10480	4420	2781	1788	1540	894
All Adults	Phoenix	S65	2017	10	114931	61240	42665	30595	23989	19470
All Adults	Phoenix	S65	2017	15	31042	15645	10530	7500	5811	4271
All Adults	Phoenix	S65	2017	20	12566	5960	3129	2235	1341	1093
All Adults	Phoenix	S70	2015	10	129484	70776	48029	36555	29354	24983
All Adults	Phoenix	S70	2015	15	40529	20960	13212	8791	6506	5066
All Adults	Phoenix	S70	2015	20	16887	7152	4222	2732	1838	1242
All Adults	Phoenix	S70	2016	10	121636	68690	48227	36903	29552	24437
All Adults	Phoenix	S70	2016	15	40132	20513	12814	9040	6655	5414
All Adults	Phoenix	S70	2016	20	16887	7202	4172	3030	2285	1838
All Adults	Phoenix	S70	2017	10	143440	78624	55230	41423	32234	26423
All Adults	Phoenix	S70	2017	15	43658	21655	14205	10828	8046	6308
All Adults	Phoenix	S70	2017	20	18327	8642	5761	4172	2583	1987

Study Group	Study Area	AQ Scenario	Year	FEV ₁ (percent)	Number of People at or above FEV ₁ Decrement					
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All Adults	Phoenix	S75	2015	10	152530	82945	57267	44552	35016	29453
All Adults	Phoenix	S75	2015	15	50115	26274	17533	12119	9238	7251
All Adults	Phoenix	S75	2015	20	22052	10579	6457	4172	3129	2235
All Adults	Phoenix	S75	2016	10	142298	80462	57118	44204	35214	28311
All Adults	Phoenix	S75	2016	15	49270	25728	16738	11771	8990	7301
All Adults	Phoenix	S75	2016	20	22152	9785	6059	4073	3278	2334
All Adults	Phoenix	S75	2017	10	169218	93574	65611	50462	40579	32979
All Adults	Phoenix	S75	2017	15	56075	28807	18675	13311	10728	8543
All Adults	Phoenix	S75	2017	20	24735	11771	7947	5712	4222	2881
All Adults	Sacramento	S65	2015	10	32672	14692	8775	6803	5231	4116
All Adults	Sacramento	S65	2015	15	7661	3316	1887	1315	1000	800
All Adults	Sacramento	S65	2015	20	2916	1086	715	457	172	114
All Adults	Sacramento	S65	2016	10	32329	14864	9118	6031	4516	3516
All Adults	Sacramento	S65	2016	15	7804	2973	1801	1258	743	515
All Adults	Sacramento	S65	2016	20	2773	858	372	257	143	114
All Adults	Sacramento	S65	2017	10	31843	15235	9747	6660	5174	4002
All Adults	Sacramento	S65	2017	15	7546	3287	2144	1229	829	600
All Adults	Sacramento	S65	2017	20	3001	1058	343	257	200	57
All Adults	Sacramento	S70	2015	10	43734	21152	13263	9547	7375	6117
All Adults	Sacramento	S70	2015	15	11691	5260	3287	2287	1658	1229
All Adults	Sacramento	S70	2015	20	4802	1887	1172	715	515	343
All Adults	Sacramento	S70	2016	10	44306	21038	13863	9919	7232	5260
All Adults	Sacramento	S70	2016	15	12291	5088	3087	1972	1401	1058
All Adults	Sacramento	S70	2016	20	4831	1887	1000	486	314	200
All Adults	Sacramento	S70	2017	10	43820	21067	13578	10119	7175	6003
All Adults	Sacramento	S70	2017	15	10948	5260	3201	2344	1801	1229
All Adults	Sacramento	S70	2017	20	4888	1944	1029	629	457	200
All Adults	Sacramento	S75	2015	10	53196	26641	17008	12034	9204	7718
All Adults	Sacramento	S75	2015	15	15350	7060	4316	2887	2258	1829
All Adults	Sacramento	S75	2015	20	6460	2744	1572	1172	743	572
All Adults	Sacramento	S75	2016	10	54939	26984	17608	12834	9662	7546
All Adults	Sacramento	S75	2016	15	17008	6717	4145	2944	2287	1744
All Adults	Sacramento	S75	2016	20	6975	3001	1486	943	657	486
All Adults	Sacramento	S75	2017	10	54739	26669	17465	12720	9833	7746
All Adults	Sacramento	S75	2017	15	15407	7318	4545	3116	2258	1801
All Adults	Sacramento	S75	2017	20	6460	2830	1601	943	657	457
All Adults	St. Louis	S65	2015	10	38915	16453	9836	6653	4864	4006
All Adults	St. Louis	S65	2015	15	9192	3612	2325	1466	1001	715
All Adults	St. Louis	S65	2015	20	3326	1180	537	322	215	72
All Adults	St. Louis	S65	2016	10	48465	23034	13162	8906	6402	5007
All Adults	St. Louis	S65	2016	15	13127	5079	3004	1860	1431	1037
All Adults	St. Louis	S65	2016	20	5329	1717	930	680	393	179
All Adults	St. Louis	S65	2017	10	47535	22283	14200	9514	7118	5472
All Adults	St. Louis	S65	2017	15	12697	4972	2861	1967	1395	1073
All Adults	St. Louis	S65	2017	20	4578	1824	930	644	501	286

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All Adults	St. Louis	S70	2015	10	48965	21174	12948	9156	6903	5759
All Adults	St. Louis	S70	2015	15	13985	5437	3076	2182	1466	1037
All Adults	St. Louis	S70	2015	20	5508	1896	1073	680	358	179
All Adults	St. Louis	S70	2016	10	62593	30545	19100	12447	9121	6975
All Adults	St. Louis	S70	2016	15	19207	7762	4435	2683	1896	1466
All Adults	St. Louis	S70	2016	20	8226	3040	1574	1073	823	644
All Adults	St. Louis	S70	2017	10	60304	29830	19207	13520	9943	8262
All Adults	St. Louis	S70	2017	15	18313	7583	4471	3076	2075	1610
All Adults	St. Louis	S70	2017	20	7189	2647	1502	1109	715	537
All Adults	St. Louis	S75	2015	10	57442	26074	15273	10766	8620	6832
All Adults	St. Louis	S75	2015	15	17776	7082	3934	2826	1931	1431
All Adults	St. Louis	S75	2015	20	7225	2504	1538	1037	572	286
All Adults	St. Louis	S75	2016	10	72965	36840	23070	15702	11839	9192
All Adults	St. Louis	S75	2016	15	24536	10301	5866	3756	2504	1753
All Adults	St. Louis	S75	2016	20	10659	4149	2361	1574	966	751
All Adults	St. Louis	S75	2017	10	70927	35624	23392	16381	12519	10194
All Adults	St. Louis	S75	2017	15	23893	10229	6188	3612	2826	2039
All Adults	St. Louis	S75	2017	20	9586	3827	2182	1538	1180	894
Asthma Adults	Atlanta	S65	2015	10	5494	2324	1479	845	493	282
Asthma Adults	Atlanta	S65	2015	15	1409	493	70	70	70	0
Asthma Adults	Atlanta	S65	2015	20	282	70	70	0	0	0
Asthma Adults	Atlanta	S65	2016	10	6691	3381	2254	1550	1197	986
Asthma Adults	Atlanta	S65	2016	15	1690	916	775	563	423	282
Asthma Adults	Atlanta	S65	2016	20	775	493	282	211	211	211
Asthma Adults	Atlanta	S65	2017	10	5635	2254	1620	845	563	423
Asthma Adults	Atlanta	S65	2017	15	1831	352	211	70	70	70
Asthma Adults	Atlanta	S65	2017	20	282	141	70	0	0	0
Asthma Adults	Atlanta	S70	2015	10	7677	3381	2113	1620	986	634
Asthma Adults	Atlanta	S70	2015	15	2043	986	282	141	141	0
Asthma Adults	Atlanta	S70	2015	20	634	141	70	70	70	0
Asthma Adults	Atlanta	S70	2016	10	8875	4367	3240	2113	1690	1550
Asthma Adults	Atlanta	S70	2016	15	2395	916	775	704	563	493
Asthma Adults	Atlanta	S70	2016	20	1127	563	493	423	211	211
Asthma Adults	Atlanta	S70	2017	10	7818	3663	2324	1409	986	634
Asthma Adults	Atlanta	S70	2017	15	2395	634	282	282	141	70
Asthma Adults	Atlanta	S70	2017	20	493	211	141	0	0	0
Asthma Adults	Atlanta	S75	2015	10	10142	4296	2747	2113	1479	1057
Asthma Adults	Atlanta	S75	2015	15	2817	1409	493	211	211	70
Asthma Adults	Atlanta	S75	2015	20	1338	423	70	70	70	0
Asthma Adults	Atlanta	S75	2016	10	11903	5494	3733	2958	2113	1902
Asthma Adults	Atlanta	S75	2016	15	3803	1479	986	916	704	563
Asthma Adults	Atlanta	S75	2016	20	1479	775	704	423	352	211
Asthma Adults	Atlanta	S75	2017	10	9649	4226	3099	2183	1620	1197
Asthma Adults	Atlanta	S75	2017	15	2747	916	634	423	282	211
Asthma Adults	Atlanta	S75	2017	20	1057	352	141	141	70	0

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Asthma Adults	Boston	S65	2015	10	9490	3816	1957	1468	1076	978
Asthma Adults	Boston	S65	2015	15	1859	881	587	294	0	0
Asthma Adults	Boston	S65	2015	20	685	98	0	0	0	0
Asthma Adults	Boston	S65	2016	10	9784	4403	3229	1957	1370	1174
Asthma Adults	Boston	S65	2016	15	2642	1370	881	196	196	196
Asthma Adults	Boston	S65	2016	20	978	489	294	98	98	0
Asthma Adults	Boston	S65	2017	10	11838	4892	2837	1761	1468	1076
Asthma Adults	Boston	S65	2017	15	3131	881	391	294	294	196
Asthma Adults	Boston	S65	2017	20	1272	391	196	196	0	0
Asthma Adults	Boston	S70	2015	10	11447	4598	2250	1468	1174	978
Asthma Adults	Boston	S70	2015	15	2348	978	587	294	196	98
Asthma Adults	Boston	S70	2015	20	881	196	0	0	0	0
Asthma Adults	Boston	S70	2016	10	12425	5479	3522	2739	1761	1370
Asthma Adults	Boston	S70	2016	15	3522	1859	881	489	294	196
Asthma Adults	Boston	S70	2016	20	1468	587	294	196	98	98
Asthma Adults	Boston	S70	2017	10	15067	5870	3326	2446	1957	1370
Asthma Adults	Boston	S70	2017	15	3816	1272	489	391	391	294
Asthma Adults	Boston	S70	2017	20	1663	489	196	196	98	98
Asthma Adults	Boston	S75	2015	10	12425	5381	2348	1565	1370	1272
Asthma Adults	Boston	S75	2015	15	2642	978	685	489	196	98
Asthma Adults	Boston	S75	2015	20	783	294	98	0	0	0
Asthma Adults	Boston	S75	2016	10	14186	5870	3620	2935	2152	1468
Asthma Adults	Boston	S75	2016	15	3718	2152	1468	881	587	294
Asthma Adults	Boston	S75	2016	20	1859	783	489	196	98	98
Asthma Adults	Boston	S75	2017	10	16143	6653	3522	2739	2250	1370
Asthma Adults	Boston	S75	2017	15	4403	1565	783	587	391	391
Asthma Adults	Boston	S75	2017	20	2055	489	294	196	98	98
Asthma Adults	Dallas	S65	2015	10	6407	2422	1797	1328	1172	547
Asthma Adults	Dallas	S65	2015	15	1797	859	313	156	78	78
Asthma Adults	Dallas	S65	2015	20	547	0	0	0	0	0
Asthma Adults	Dallas	S65	2016	10	5782	2266	1016	625	391	391
Asthma Adults	Dallas	S65	2016	15	1172	313	313	313	156	156
Asthma Adults	Dallas	S65	2016	20	391	234	234	156	78	78
Asthma Adults	Dallas	S65	2017	10	8439	3516	2657	2110	1719	1485
Asthma Adults	Dallas	S65	2017	15	2813	1250	1016	859	469	313
Asthma Adults	Dallas	S65	2017	20	1641	391	156	156	78	0
Asthma Adults	Dallas	S70	2015	10	8751	3438	2188	1485	1172	1016
Asthma Adults	Dallas	S70	2015	15	2110	859	547	313	234	156
Asthma Adults	Dallas	S70	2015	20	703	156	78	78	78	0
Asthma Adults	Dallas	S70	2016	10	6876	2891	1250	938	469	391
Asthma Adults	Dallas	S70	2016	15	1563	469	313	313	234	156
Asthma Adults	Dallas	S70	2016	20	391	234	234	156	156	156
Asthma Adults	Dallas	S70	2017	10	9611	4532	3282	2657	2110	1953
Asthma Adults	Dallas	S70	2017	15	3360	1485	1094	938	625	469
Asthma Adults	Dallas	S70	2017	20	1641	859	625	234	78	78

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Asthma Adults	Dallas	S75	2015	10	10861	3985	2735	1641	1406	1250
Asthma Adults	Dallas	S75	2015	15	2735	938	625	547	313	156
Asthma Adults	Dallas	S75	2015	20	1250	313	156	78	78	0
Asthma Adults	Dallas	S75	2016	10	8361	3672	1641	1094	781	469
Asthma Adults	Dallas	S75	2016	15	2266	547	313	313	313	156
Asthma Adults	Dallas	S75	2016	20	547	234	234	234	156	156
Asthma Adults	Dallas	S75	2017	10	10705	5313	3829	3125	2344	2110
Asthma Adults	Dallas	S75	2017	15	4141	1797	1328	1250	859	547
Asthma Adults	Dallas	S75	2017	20	1875	1016	703	391	313	234
Asthma Adults	Detroit	S65	2015	10	7603	3146	1507	1114	655	590
Asthma Adults	Detroit	S65	2015	15	1770	721	262	131	131	131
Asthma Adults	Detroit	S65	2015	20	655	262	131	131	0	0
Asthma Adults	Detroit	S65	2016	10	8979	3932	2163	1770	1507	1376
Asthma Adults	Detroit	S65	2016	15	2425	1376	852	721	393	262
Asthma Adults	Detroit	S65	2016	20	1376	655	328	66	66	0
Asthma Adults	Detroit	S65	2017	10	8914	4260	2753	2097	1507	1114
Asthma Adults	Detroit	S65	2017	15	2687	1311	590	393	393	328
Asthma Adults	Detroit	S65	2017	20	1442	393	328	262	66	0
Asthma Adults	Detroit	S70	2015	10	9241	4064	1901	1376	983	852
Asthma Adults	Detroit	S70	2015	15	2359	983	524	197	197	131
Asthma Adults	Detroit	S70	2015	20	852	328	131	131	131	66
Asthma Adults	Detroit	S70	2016	10	11011	5047	2949	2359	1901	1770
Asthma Adults	Detroit	S70	2016	15	3343	1704	1180	852	786	721
Asthma Adults	Detroit	S70	2016	20	1770	852	459	262	262	131
Asthma Adults	Detroit	S70	2017	10	11273	5047	3212	2687	1901	1573
Asthma Adults	Detroit	S70	2017	15	3474	1639	918	590	459	393
Asthma Adults	Detroit	S70	2017	20	1966	459	459	328	262	197
Asthma Adults	Detroit	S75	2015	10	10356	4653	2622	1507	1114	918
Asthma Adults	Detroit	S75	2015	15	2753	1114	590	262	262	131
Asthma Adults	Detroit	S75	2015	20	1180	393	262	197	131	131
Asthma Adults	Detroit	S75	2016	10	12977	6095	3736	2556	2097	1770
Asthma Adults	Detroit	S75	2016	15	4326	1901	1311	918	786	721
Asthma Adults	Detroit	S75	2016	20	1966	1114	655	393	393	328
Asthma Adults	Detroit	S75	2017	10	13239	6161	3998	2949	2163	1901
Asthma Adults	Detroit	S75	2017	15	4195	2097	1180	786	524	459
Asthma Adults	Detroit	S75	2017	20	2359	655	459	393	328	262
Asthma Adults	Philadelphia	S65	2015	10	10370	4619	2701	1917	1220	959
Asthma Adults	Philadelphia	S65	2015	15	2614	1046	784	349	349	261
Asthma Adults	Philadelphia	S65	2015	20	1133	349	174	174	87	87
Asthma Adults	Philadelphia	S65	2016	10	8453	3137	1917	1394	1133	697
Asthma Adults	Philadelphia	S65	2016	15	1394	349	174	0	0	0
Asthma Adults	Philadelphia	S65	2016	20	349	174	0	0	0	0
Asthma Adults	Philadelphia	S65	2017	10	9760	4357	3224	2179	1569	1133
Asthma Adults	Philadelphia	S65	2017	15	2440	1307	436	261	174	87
Asthma Adults	Philadelphia	S65	2017	20	1133	174	0	0	0	0

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					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Philadelphia	S70	2015	10	12026	5141	3050	2701	1830	1220
Asthma Adults	Philadelphia	S70	2015	15	3399	1481	959	610	436	349
Asthma Adults	Philadelphia	S70	2015	20	1569	436	349	349	87	87
Asthma Adults	Philadelphia	S70	2016	10	10631	4357	2701	1656	1394	959
Asthma Adults	Philadelphia	S70	2016	15	1917	610	261	174	174	0
Asthma Adults	Philadelphia	S70	2016	20	436	261	87	0	0	0
Asthma Adults	Philadelphia	S70	2017	10	11067	6013	3660	2789	2091	1656
Asthma Adults	Philadelphia	S70	2017	15	3399	1569	959	523	261	174
Asthma Adults	Philadelphia	S70	2017	20	1656	436	174	0	0	0
Asthma Adults	Philadelphia	S75	2015	10	15773	7233	4183	3311	2353	1743
Asthma Adults	Philadelphia	S75	2015	15	4531	1917	1220	784	523	436
Asthma Adults	Philadelphia	S75	2015	20	1917	784	349	349	174	87
Asthma Adults	Philadelphia	S75	2016	10	13594	5403	3660	2440	2004	1394
Asthma Adults	Philadelphia	S75	2016	15	3921	1569	784	349	174	87
Asthma Adults	Philadelphia	S75	2016	20	697	261	87	87	0	0
Asthma Adults	Philadelphia	S75	2017	10	13507	6710	4706	3573	2527	2266
Asthma Adults	Philadelphia	S75	2017	15	4444	1743	1133	959	436	261
Asthma Adults	Philadelphia	S75	2017	20	1917	610	349	87	0	0
Asthma Adults	Phoenix	S65	2015	10	8444	4321	2583	2036	1639	1291
Asthma Adults	Phoenix	S65	2015	15	2334	1440	844	497	397	248
Asthma Adults	Phoenix	S65	2015	20	844	397	298	199	99	0
Asthma Adults	Phoenix	S65	2016	10	6953	3973	2483	1589	1291	1192
Asthma Adults	Phoenix	S65	2016	15	2086	795	447	298	199	99
Asthma Adults	Phoenix	S65	2016	20	596	149	50	0	0	0
Asthma Adults	Phoenix	S65	2017	10	8046	4172	2732	2086	1738	1391
Asthma Adults	Phoenix	S65	2017	15	2235	1341	993	695	497	497
Asthma Adults	Phoenix	S65	2017	20	894	546	298	248	149	99
Asthma Adults	Phoenix	S70	2015	10	10232	5612	3626	2781	1987	1738
Asthma Adults	Phoenix	S70	2015	15	3328	1788	1142	695	546	447
Asthma Adults	Phoenix	S70	2015	20	1738	646	397	248	149	50
Asthma Adults	Phoenix	S70	2016	10	8543	4818	3526	2285	1788	1440
Asthma Adults	Phoenix	S70	2016	15	2632	1341	795	497	248	248
Asthma Adults	Phoenix	S70	2016	20	1093	497	149	50	50	50
Asthma Adults	Phoenix	S70	2017	10	9785	5414	3924	2732	2036	1788
Asthma Adults	Phoenix	S70	2017	15	2980	1788	1142	844	646	596
Asthma Adults	Phoenix	S70	2017	20	1242	646	447	397	248	199
Asthma Adults	Phoenix	S75	2015	10	11871	6705	4420	3328	2483	2086
Asthma Adults	Phoenix	S75	2015	15	4023	2185	1540	1093	745	596
Asthma Adults	Phoenix	S75	2015	20	1937	1043	546	298	149	149
Asthma Adults	Phoenix	S75	2016	10	10182	5612	4222	2881	2285	1838
Asthma Adults	Phoenix	S75	2016	15	3526	1689	1093	745	546	397
Asthma Adults	Phoenix	S75	2016	20	1589	646	348	99	50	50
Asthma Adults	Phoenix	S75	2017	10	11473	6308	4470	3328	2682	2185
Asthma Adults	Phoenix	S75	2017	15	4073	2136	1391	1142	944	695
Asthma Adults	Phoenix	S75	2017	20	1738	894	646	546	397	298

Study Group	Study Area	AQ Scenario	Year	FEV ₁ (percent)	Number of People at or above FEV ₁ Decrement					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	Sacramento	S65	2015	10	2287	858	515	457	343	343
Asthma Adults	Sacramento	S65	2015	15	543	172	86	86	86	29
Asthma Adults	Sacramento	S65	2015	20	257	29	29	29	0	0
Asthma Adults	Sacramento	S65	2016	10	2172	1143	715	543	400	343
Asthma Adults	Sacramento	S65	2016	15	515	200	143	86	86	57
Asthma Adults	Sacramento	S65	2016	20	172	57	57	57	57	29
Asthma Adults	Sacramento	S65	2017	10	2115	915	486	400	314	314
Asthma Adults	Sacramento	S65	2017	15	457	257	172	57	57	29
Asthma Adults	Sacramento	S65	2017	20	172	86	0	0	0	0
Asthma Adults	Sacramento	S70	2015	10	2887	1286	858	657	543	400
Asthma Adults	Sacramento	S70	2015	15	715	314	286	114	114	29
Asthma Adults	Sacramento	S70	2015	20	343	143	86	29	29	29
Asthma Adults	Sacramento	S70	2016	10	2830	1429	1029	772	543	400
Asthma Adults	Sacramento	S70	2016	15	772	400	343	200	114	86
Asthma Adults	Sacramento	S70	2016	20	343	172	114	86	57	29
Asthma Adults	Sacramento	S70	2017	10	2716	1258	743	515	400	314
Asthma Adults	Sacramento	S70	2017	15	800	257	200	143	86	57
Asthma Adults	Sacramento	S70	2017	20	229	143	86	29	0	0
Asthma Adults	Sacramento	S75	2015	10	3544	1744	1172	800	657	572
Asthma Adults	Sacramento	S75	2015	15	1058	429	343	200	143	86
Asthma Adults	Sacramento	S75	2015	20	400	143	86	86	57	29
Asthma Adults	Sacramento	S75	2016	10	3602	1744	1258	943	657	457
Asthma Adults	Sacramento	S75	2016	15	1229	600	372	372	257	114
Asthma Adults	Sacramento	S75	2016	20	457	229	143	114	86	57
Asthma Adults	Sacramento	S75	2017	10	3430	1544	972	657	486	400
Asthma Adults	Sacramento	S75	2017	15	943	400	314	200	114	86
Asthma Adults	Sacramento	S75	2017	20	314	200	86	86	0	0
Asthma Adults	St. Louis	S65	2015	10	3684	1466	894	537	322	322
Asthma Adults	St. Louis	S65	2015	15	894	250	107	72	36	0
Asthma Adults	St. Louis	S65	2015	20	179	36	0	0	0	0
Asthma Adults	St. Louis	S65	2016	10	4185	1931	1073	715	572	429
Asthma Adults	St. Louis	S65	2016	15	1145	501	358	215	143	143
Asthma Adults	St. Louis	S65	2016	20	537	179	107	72	36	0
Asthma Adults	St. Louis	S65	2017	10	4435	1896	1252	751	537	322
Asthma Adults	St. Louis	S65	2017	15	1001	429	286	143	36	36
Asthma Adults	St. Louis	S65	2017	20	358	72	0	0	0	0
Asthma Adults	St. Louis	S70	2015	10	4435	1860	1145	751	537	465
Asthma Adults	St. Louis	S70	2015	15	1288	501	215	107	72	36
Asthma Adults	St. Louis	S70	2015	20	322	72	36	36	36	0
Asthma Adults	St. Louis	S70	2016	10	5687	2611	1574	1073	787	608
Asthma Adults	St. Louis	S70	2016	15	1610	787	429	250	143	143
Asthma Adults	St. Louis	S70	2016	20	715	286	215	143	107	36
Asthma Adults	St. Louis	S70	2017	10	5651	2468	1610	1109	787	608
Asthma Adults	St. Louis	S70	2017	15	1610	608	358	215	107	72
Asthma Adults	St. Louis	S70	2017	20	608	179	107	36	0	0

Study Group	Study Area	AQ Scenario	Year	FEV ₁ (percent)	Number of People at or above FEV ₁ Decrement					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
Asthma Adults	St. Louis	S75	2015	10	5079	2253	1431	858	680	537
Asthma Adults	St. Louis	S75	2015	15	1717	608	322	179	107	72
Asthma Adults	St. Louis	S75	2015	20	465	143	36	36	36	0
Asthma Adults	St. Louis	S75	2016	10	6545	3219	2039	1395	1073	787
Asthma Adults	St. Louis	S75	2016	15	1967	1001	465	286	215	143
Asthma Adults	St. Louis	S75	2016	20	1001	358	250	215	143	72
Asthma Adults	St. Louis	S75	2017	10	6724	2933	1931	1109	1073	823
Asthma Adults	St. Louis	S75	2017	15	2146	823	465	215	179	107
Asthma Adults	St. Louis	S75	2017	20	823	250	179	72	36	36

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APPENDIX 4A

**EXPOSURE-RESPONSE FUNCTIONS FOR 11 TREE SPECIES AND
TEN CROPS**

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Attachment

Derivation of Composite Median Equations (parameterized models) in Lee and Hogsett (1996)

1 **4A.1 BACKGROUND**

2 Air quality criteria documents (AQCDs) for prior ozone (O₃) reviews have presented
3 exposure-response functions derived in the 1980s through mid-1990s from the results of a series
4 of studies on the growth effects of a range of seasonal O₃ exposure levels. These studies included
5 research conducted by the National Crop Loss Assessment Network (NCLAN) on commercial
6 crop species and by the EPA’s National Health and Environmental Effects Laboratory Western
7 Ecology Division (NHEERL/WED) on seedlings of 11 tree species¹. These studies also included
8 documentation of hourly concentrations across the full exposures, and multiple exposure
9 scenarios per experiment, which has resulted in their being the focus of work to characterize
10 exposure-response (E-R) relationships for growth impacts on crops and tree species.

11 The experimental study results were analyzed to define a quantitative model that would
12 well describe the E-R relationships of seasonal O₃ exposure and first impaired tree seedling
13 growth and crop yield.² Those studies, which used several different metrics to quantify exposure
14 (e.g., SUM06, W126), concluded that for the use of a single metric, such as W126 index, a three-
15 parameter Weibull model form provides the most appropriate model for the response of absolute
16 yield and growth to O₃ exposure because of the interpretability of its parameters, its flexibility
17 (given the small number of parameters), and its tractability for estimation (2013 ISA, section
18 9.6.2). This three-parameter Weibull model is presented in equation 4A-1.

$$Y = \alpha e^{-\left(\frac{W126}{\eta}\right)^\beta}$$

Equation 4A-1

19
20 where:

- 21 Y = total yield or biomass;
22 W126 = O₃ exposure (e.g., 3-month sum of daily cumulative W126 from 8am to 8pm);
23 and,
24 η and β are species-specific variables
25

26 With removal of the intercept term, α, the model estimates relative yield or biomass
27 without any further reparameterization. In order to compare E-R functions and associated
28 estimates across species, genotypes, or experiments (of same species/genotype) for which

¹ These programs and the research conducted under them is described in detail in the 1996 AQCD (sections 5.5 and 5.6), summarized in the 2006 AQCD (section 9.5), 2013 ISA (section 9.6), and the 2020 ISA (Appendix 8, section 8.13).

² Examples of these analyses include Lee et al. (1994), Gumpertz and Rawlings (1992), Heck et al. (1984), Hogsett et al. (1997), Lee and Hogsett (1999), Lee et al. (1987), Lee et al. (1988), Lee et al. (1989), Lesser et al. (1990), Rawlings and Cure (1985).

1 absolute values of the response may vary greatly, the model is reformulated in terms of relative
 2 annual yield (or biomass) or relative yield (or biomass) loss (yield loss= [1-relative yield]). The
 3 resultant 2-parameter model of relative yield was presented in the 1996 and 2006 AQCDs and
 4 2013 and 2020 ISA as basis for deriving common models for multiple species, multiple
 5 genotypes within species and multiple experimental locations (2013 ISA, section 9.6.2; 2020
 6 ISA, Appendix 8, section 8.13.2). The models presented in the AQCDs were in terms of SUM06
 7 over a 3-month season; those models were updated for 12-hour W126 over a 3-month season in
 8 the 2013 ISA (2013 ISA, section 9.6.2). The 2-parameter model structure, for relative biomass
 9 loss (RBL) or relative yield loss (RYL) as a function of W126 is described in equation 4A-2.

10
$$\text{RBL} = 1 - \exp[-(\text{W126}/\eta)\beta] \quad \text{Equation 4A-2}$$

11 Based on this model structure, functions for estimating RBL from seasonal W126 index,
 12 parameterized for each of eleven tree species, are presented and discussed in section 4A.1.1
 13 below, and RYL functions for the 10 crop species are presented in section 4A.1.2.

14 **4A.1.1 Tree Species Seedling E-R Functions**

15 The RBL functions for each of 11 common tree species were derived as median
 16 composite functions from response estimates based on functions derived for each study or
 17 experiment for which data were collected for that species (Lee and Hogsett, 1996, Tables 12 and
 18 13). The eleven species-specific (composite median) functions, based on Lee and Hogsett
 19 (1996),³ are presented in Table 4A-1.

20 **Table 4A-1. RBL functions for tree species.**

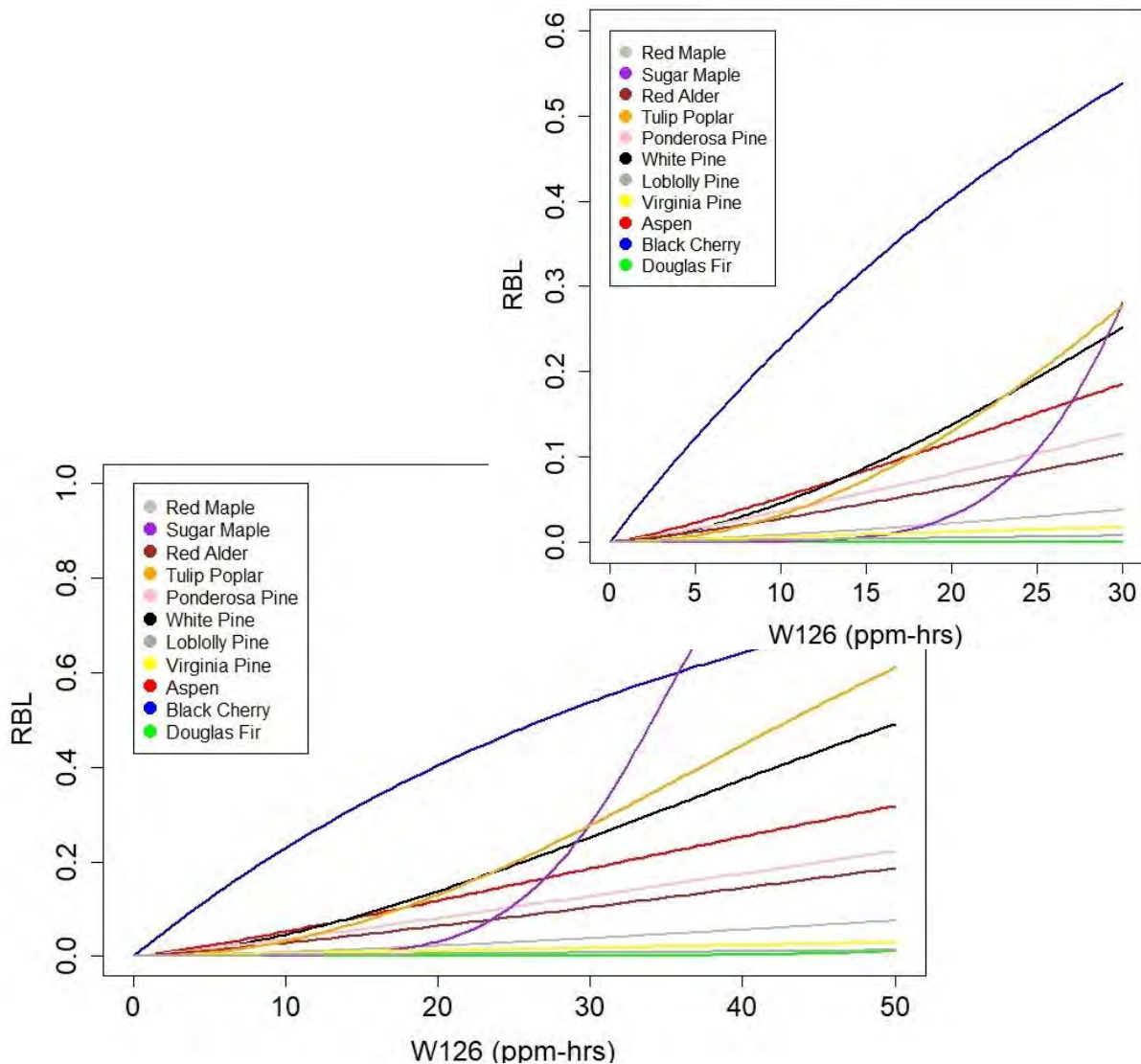
Species	RBL Function	η (ppm)	β
Red Maple (<i>Acer rubrum</i>)	$1 - \exp[-(\text{W126}/\eta)\beta]$	318.12	1.3756
Sugar Maple (<i>Acer saccharum</i>)		36.35	5.7785
Red Alder (<i>Alnus rubra</i>)		179.06	1.2377
Tulip Poplar (<i>Liriodendron tulipifera</i>)		51.38	2.0889
Ponderosa Pine (<i>Pinus ponderosa</i>)		159.63	1.1900
Eastern White Pine (<i>Pinus strobus</i>)		63.23	1.6582
Loblolly Pine (<i>Pinus taeda</i>)		1,021.63	0.9954
Virginia Pine (<i>Pinus virginiana</i>)		1,714.64	1.0000
Quaking Aspen (<i>Populus tremuloides</i>), wild		109.81	1.2198
Black Cherry (<i>Prunus serotina</i>)		38.92	0.9921
Douglas Fir (<i>Pseudotsuga menziesii</i>)		106.83	5.9631

Source: These functions are those presented in Lee and Hogsett (1996), Table 13 or, for loblolly pine, as presented in Table 8-24 of Appendix 8 of the ISA.

21

³ The functions presented in Table 4A-1 reflect the median composite response functions presented in Table 13 of Lee and Hogsett (1996), with the addition of the response curve for loblolly pine from Table 8-24 of Appendix 8 of the 2020 ISA. The process for deriving the composite functions is described in Lee and Hogsett (1996).

1 Figure 4A-1 presents species-specific E-R functions for the tree seedlings. The figures
 2 illustrate how the values of the two parameters affect the shape of the resulting curves. The value
 3 of η in the RBL function affects the point of the curve where the slope appreciably changes, and
 4 β affects the steepness of the curve. The response functions with smaller values of β (e.g.,
 5 Virginia Pine) or with η values that are above the range shown for of W126 index values (e.g.,
 6 functions for ponderosa pine and red alder) exhibit smaller slopes that have less change across
 7 this W126 range. These functions describe a more constant rate of change in RBL over the range
 8 of O₃ exposure shown (e.g., up to 30 ppm-hrs). In contrast, the response functions with larger β
 9 values (e.g., the function for Sugar Maple) exhibit a threshold-like behavior, with large changes
 10 in RBL over a small range of W126 index values and relatively small changes at other index
 11 values. In these cases, the “threshold” is determined by the η parameter of the model.

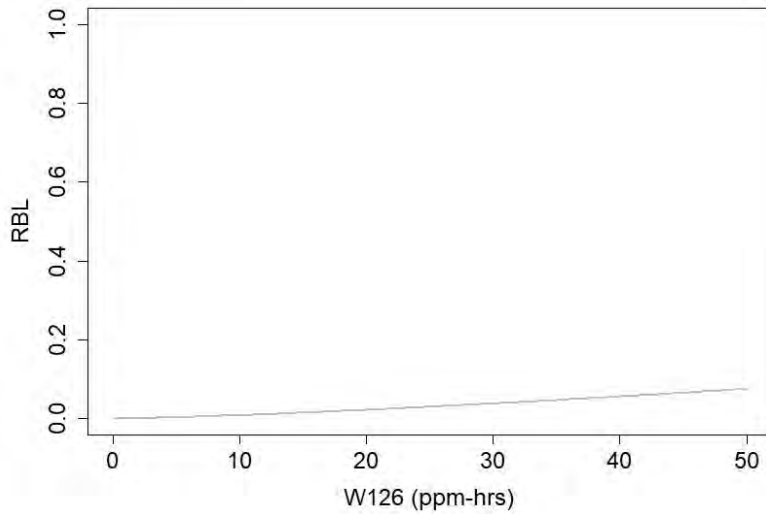


38 **Figure 4A-1. RBL functions for seedlings of 11 tree species.**

1 The shape of curves presented in Figure 4A-1 also illustrate how sensitive the RBL value
2 is to changes in O₃. Two species, Loblolly Pine (dark grey line) and Virginia Pine (yellow line)
3 have E-R functions that approach linearity within the W126 range represented on the x-axis,
4 meaning that any 1 percent change in W126 produces the same change in RBL. Black Cherry
5 (blue line) has an E-R function that exhibits a declining slope with increasing W126 (each
6 successive equal change in W126 produces a smaller change in RBL), with the appearance of
7 leveling off (Figure 4A-1). The functions for the remaining species appear to be somewhat
8 linear, e.g., each 1% change in W126 across the W126 range produces an identical (or somewhat
9 similar) percent change in RBL.

10 As mentioned above, the species-specific functions were derived from median estimates
11 based on the functions from the individual experiments for each species. Figure 4A-2 through
12 Figure 4A-12 present the species-specific functions along with the functions derived from the
13 experiments available for that species.⁴ These figures provide a sense of the across-experiment
14 variability for each species, where such information is available.

15

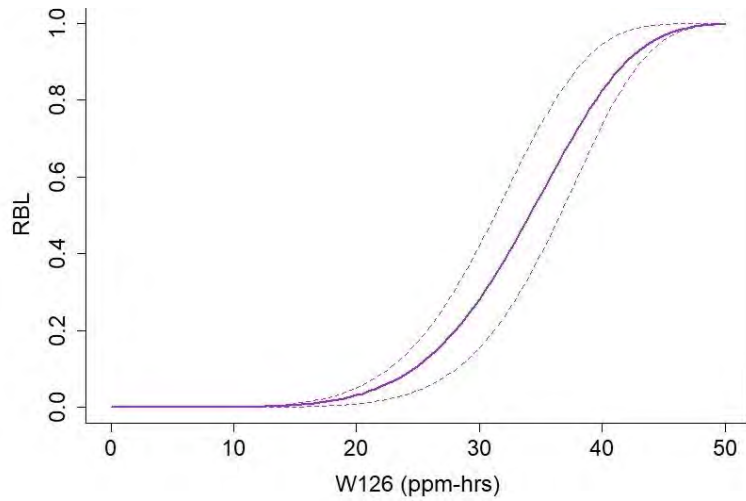


16

17 **Figure 4A-2. RBL functions for Red Maple (*Acer rubrum*).**

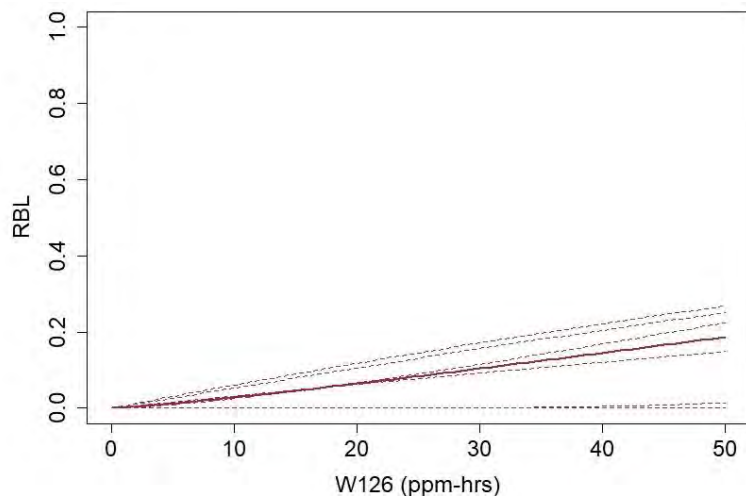
18

⁴ For aspen, the dark (red) line shown in Figure 4A-1 is the median composite for wild (vs clonal genotype) studies.



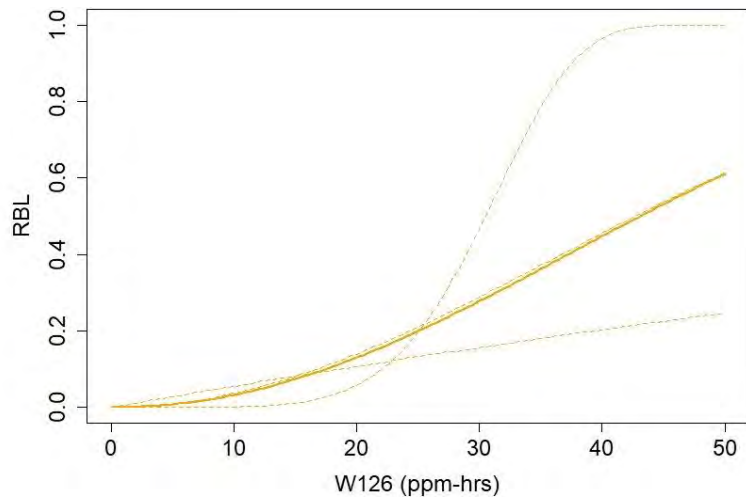
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2 **Figure 4A-3. RBL functions for Sugar Maple (*Acer saccharum*).**



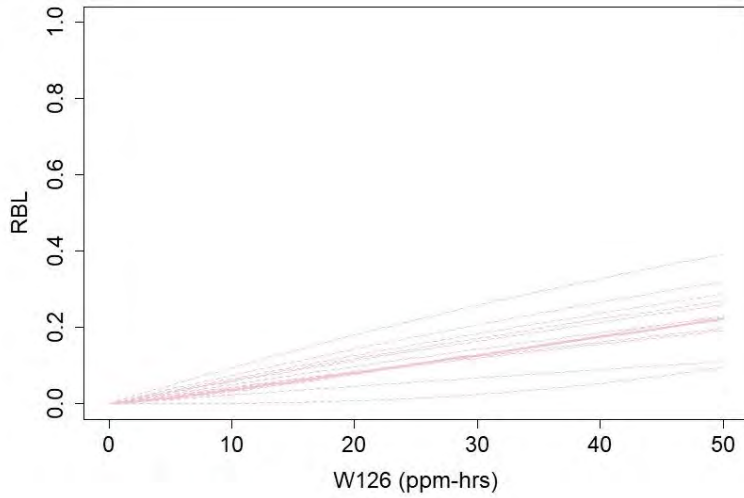
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4 **Figure 4A-4. RBL functions for Red Alder (*Alnus rubra*).**



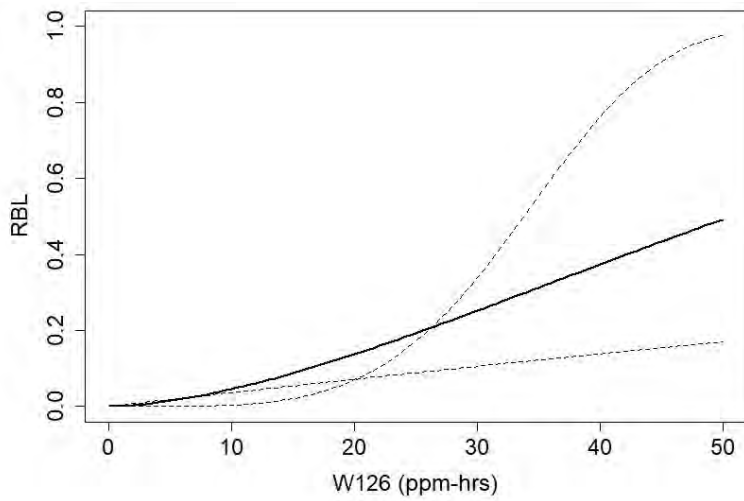
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6 **Figure 4A-5. RBL functions for Tulip Poplar (*Liriodendron tulipifera*).**



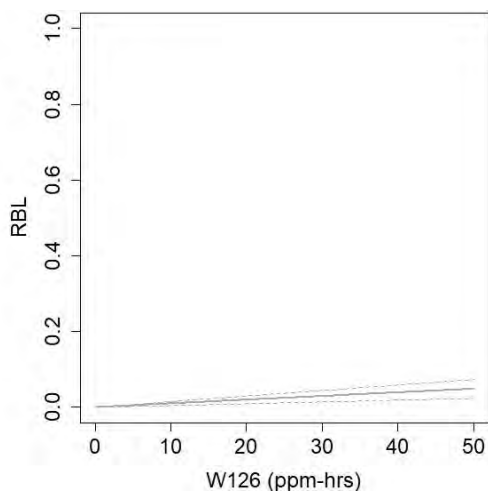
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2 **Figure 4A-6. RBL functions for Ponderosa Pine (*Pinus ponderosa*).**



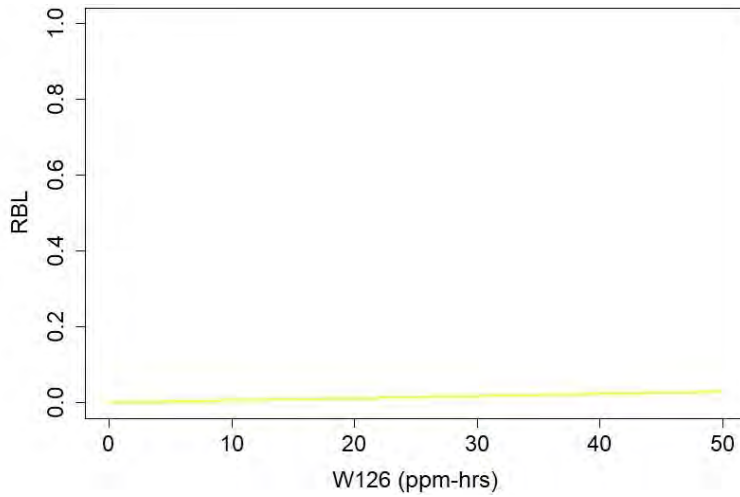
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4 **Figure 4A-7. RBL functions for White Pine (*Pinus strobus*).**



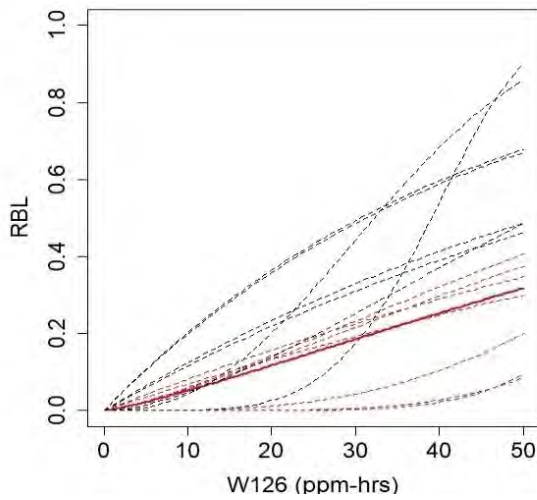
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6 **Figure 4A-8. RBL functions for Loblolly Pine (*Pinus taeda*).**



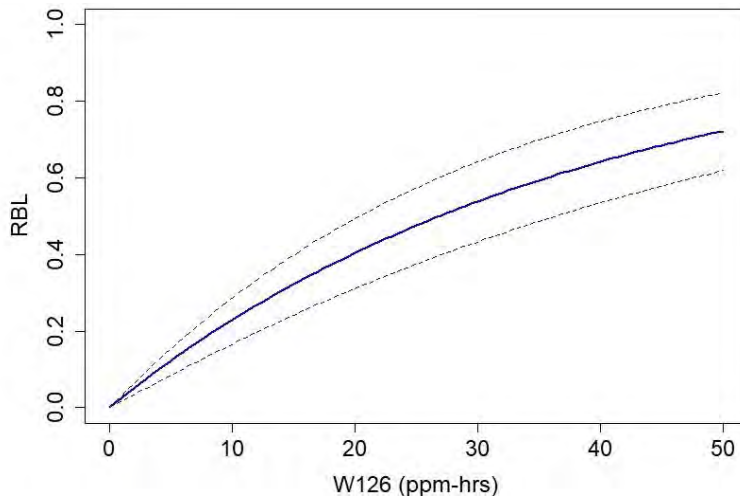
1

2 **Figure 4A-9. RBL functions for Virginia Pine (*Pinus virginiana*).**



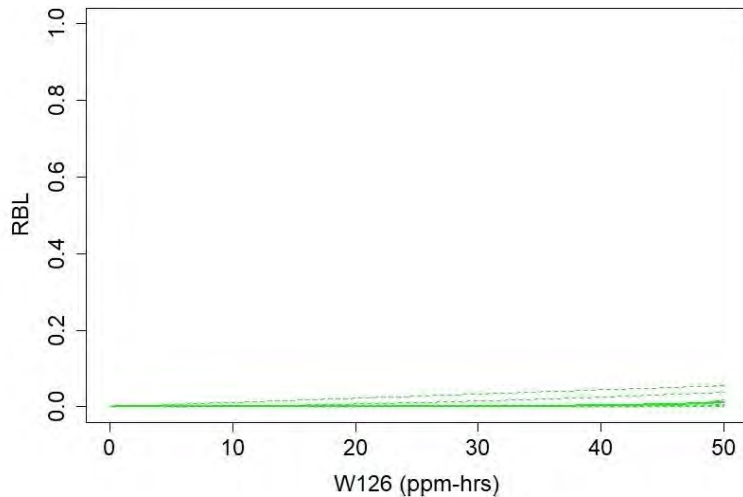
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4 **Figure 4A-10. RBL functions for Aspen (*Populus tremuloides*). Red lines = wild, black=clone.**



5

6 **Figure 4A-11. RBL functions for Black Cherry (*Prunus serotina*).**



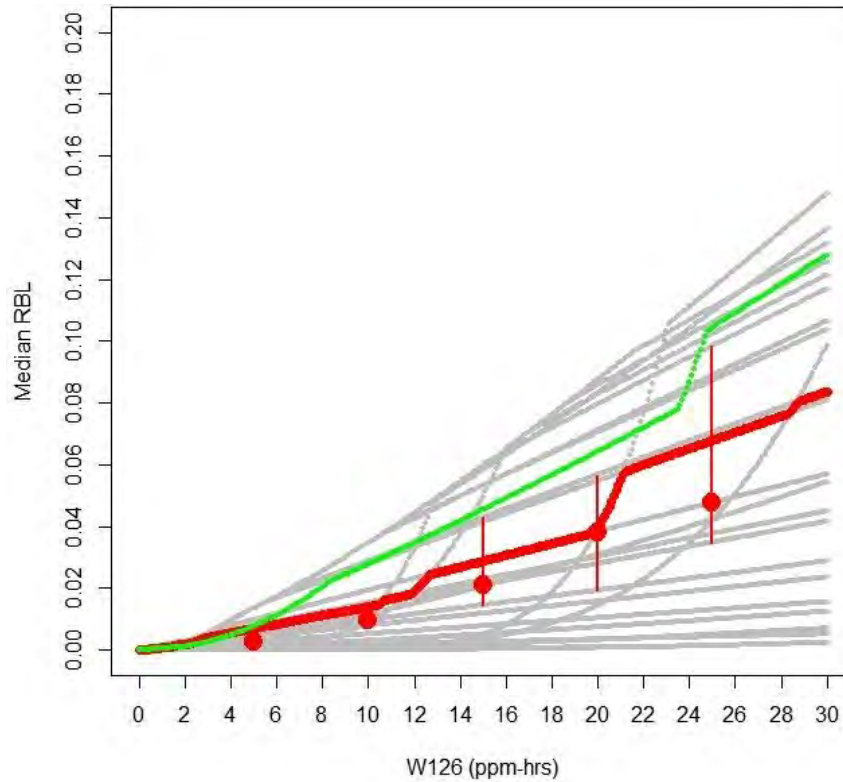
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2 **Figure 4A-12. RBL functions for Douglas Fir (*Pseudotsuga menziesii*).**

3 In the 2015 review, consideration of the E-R functions for the seedlings of 11 tree species
 4 focused on the median estimate across the 11 species-specific functions. Recognizing the extent
 5 to which experimental variation contributes to uncertainty in the species-specific E-R functions,
 6 a stochastic analysis was performed in the quantitative exposure/risk assessment for the 2015
 7 review as an approach to investigating the impact of uncertainty and variability in the E-R
 8 function dataset; an update of this analysis is presented in Figure 4A-13. This figure illustrates
 9 different approaches to estimating a median E-R function from the functions from the individual
 10 experiments. In this figure, each grey curve is the median across 11 species-specific functions
 11 where the species-specific functions are represented by a random draw from the experiment-
 12 specific functions available for each species.⁵ The red points are the median across the random
 13 draws at that W126 value and the whiskers extend to the 75th and 25th percentiles of those draws.
 14 For reference, the green line is the median across the 11 species-specific functions, and the red
 15 line is the median across the 51 experiments (regardless of species).⁶

⁵ For example, there are seven separate experiment-specific E-R functions for ponderosa pine (Lee and Hogsett, 1996). In each iteration, one of the seven is drawn. This is performed for all eleven species. Each iteration of these random draws is represented by a single grey line that plots the median of the 11 RBLs derived from the 11 functions for each W126 index level across the range of W126 presented in the figure. At different parts of the W126 range, different species' E-R functions will produce the median estimate. As a result, the grey line for each iteration of the random draws has an area of rapid change over a particular range of W126 levels (when the E-R function producing the median estimate switches to a different species) and then a smoothing (as the median estimates are being produced by the same E-R function). That is, since there are 11 species (i.e., an odd number), each point on each grey line in the figure comes from the curve for the species' function that predicts the 6th highest (or lowest) RBL for that W126 index value.

⁶ Both the green and red lines include two step-like changes along the W126 index range from 8 to 26 ppm-hrs. These steps reflect the influence on the median of the functions of species with inflection points that differ from the others (that can be seen in Figure 4A-1). For example, on the green curve (for the median across the species-specific functions), from a W126 index of approximately 8 ppm-hrs to 23 ppm-hrs, the curve largely follows the response function for red alder (which is somewhat centrally located among the functions over that W126 range



1
2 **Figure 4A-13. Stochastic analyses of median E-R function across 11 species.**

3
4 **4A.1.2 Crop Species E-R Functions**

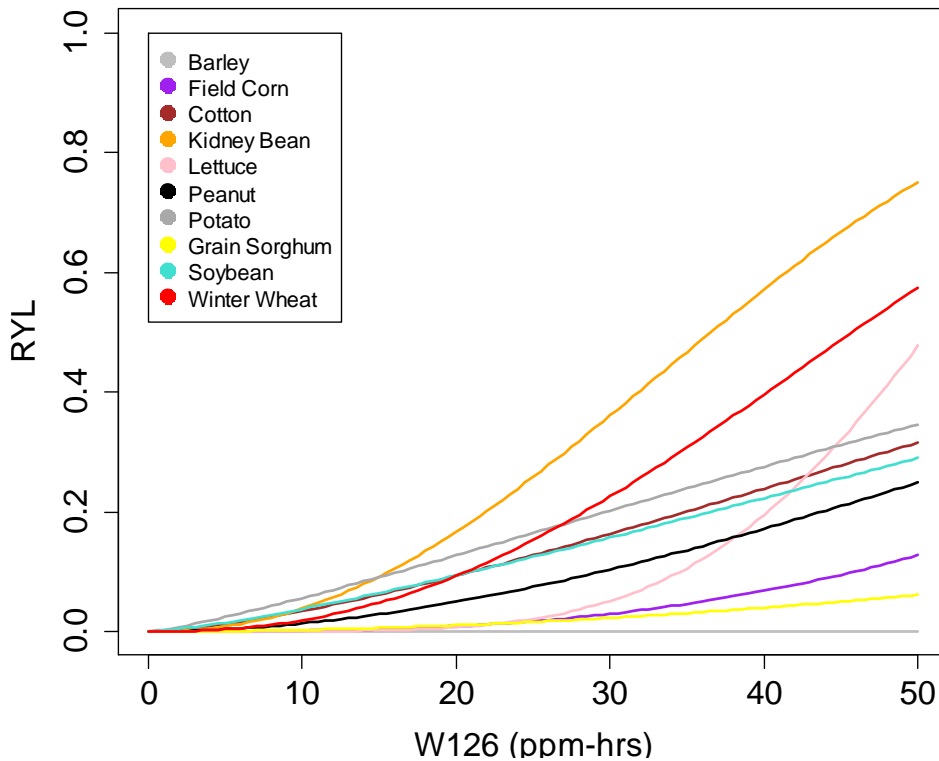
5 The RYL functions for the 10 crop species are presented in Table 4A-2, and Figure 4A-
6 14 presents the functions graphically.

7 **Table 4A-2. RYL functions for crop species**

Species	RYL Function	η (ppm)	β
Barley	$1 - \exp[-(W126/\eta)^\beta]$	6,998.5	1.388
Field Corn		97.9	2.968
Cotton		96.1	1.482
Kidney Bean		43.1	2.219
Lettuce		54.6	4.917
Peanut		96.8	1.890
Potato		99.5	1.242
Grain Sorghum		205.3	1.957
Soybean		110.2	1.359
Winter Wheat		53.4	2.367
Source: These functions are derived from those presented in Lee and Hogsett (1996).			

in Figure 4A-1). The step between 23 and 24 ppm-hrs is driven by the rapid changes of the response-function for sugar maple and above that level of W126, the response-function for ponderosa pine is central and represented by the median.

1



2

3 **Figure 4A-14. RYL functions for crop species.**

4

5 **4A.1.3 Summary Tables for Tree Species and Crops**

6 Table 4A-3 and Table 4A-4 below provide estimates of the relative loss for tree biomass
7 and crop yield, respectively, at various W126 index values using the composite E-R functions for
8 each species for each integer W126 index value between 7 ppm-hrs and 30 ppm-hrs. The cross-
9 species median of the species-specific composite functions is calculated for all 11 tree species.

10 These tables also provide estimates of the number of species for trees and crops respectively that
11 would be below various reference values (e.g., 2% RBL for trees) at various W126 index values.
12 Table 4A-5 summarizes the median values for each integer W126 index value between 7 ppm-
13 hrs and 23 ppm-hrs.

14

1 **Table 4A-3. Relative biomass loss for eleven individual tree seedlings and median at various W126 index values.**

W126	Douglas Fir	Loblolly	Virginia Pine	Red maple	Sugar maple	Red Alder	Ponderosa Pine	Aspen	Tulip Poplar	Eastern White Pine	Black Cherry	Median (11 species)	Number of Species ≤ 2%	Number of Species ≤ 5%	Number of Species ≤ 10%	Number of Species ≤ 15%
30	0.1%	0.8%	1.7%	3.8%	28.1%	10.4%	12.8%	18.6%	27.7%	25.2%	53.8%	12.8%	3	4	4	6
29	0.0%	0.7%	1.7%	3.6%	23.7%	10.0%	12.3%	17.9%	26.1%	24.0%	52.6%	12.3%	3	4	5	6
28	0.0%	0.7%	1.6%	3.5%	19.9%	9.6%	11.8%	17.2%	24.5%	22.8%	51.4%	11.8%	3	4	5	6
27	0.0%	0.7%	1.6%	3.3%	16.4%	9.2%	11.4%	16.5%	23.0%	21.6%	50.1%	11.4%	3	4	5	6
26	0.0%	0.7%	1.5%	3.1%	13.4%	8.8%	10.9%	15.8%	21.4%	20.5%	48.8%	10.9%	3	4	5	7
25	0.0%	0.6%	1.4%	3.0%	10.9%	8.4%	10.4%	15.2%	19.9%	19.3%	47.5%	10.4%	3	4	5	7
24	0.0%	0.6%	1.4%	2.8%	8.7%	8.0%	10.0%	14.5%	18.4%	18.2%	46.2%	8.7%	3	4	7	8
23	0.0%	0.6%	1.3%	2.7%	6.9%	7.6%	9.5%	13.8%	17.0%	17.1%	44.8%	7.6%	3	4	7	8
22	0.0%	0.6%	1.3%	2.5%	5.3%	7.2%	9.0%	13.1%	15.6%	15.9%	43.3%	7.2%	3	4	7	8
21	0.0%	0.5%	1.2%	2.4%	4.1%	6.8%	8.6%	12.4%	14.3%	14.9%	41.9%	6.8%	3	5	7	10
20	0.0%	0.5%	1.2%	2.2%	3.1%	6.4%	8.1%	11.8%	13.0%	13.8%	40.3%	6.4%	3	5	7	10
19	0.0%	0.5%	1.1%	2.1%	2.3%	6.0%	7.6%	11.1%	11.8%	12.7%	38.8%	6.0%	3	5	7	10
18	0.0%	0.5%	1.0%	1.9%	1.7%	5.7%	7.2%	10.4%	10.6%	11.7%	37.2%	5.7%	5	5	7	10
17	0.0%	0.4%	1.0%	1.8%	1.2%	5.3%	6.7%	9.8%	9.4%	10.7%	35.6%	5.3%	5	5	9	10
16	0.0%	0.4%	0.9%	1.6%	0.9%	4.9%	6.3%	9.1%	8.4%	9.7%	33.9%	4.9%	5	6	10	10
15	0.0%	0.4%	0.9%	1.5%	0.6%	4.5%	5.8%	8.4%	7.4%	8.8%	32.2%	4.5%	5	6	10	10
14	0.0%	0.4%	0.8%	1.4%	0.4%	4.2%	5.4%	7.8%	6.4%	7.9%	30.4%	4.2%	5	6	10	10
13	0.0%	0.3%	0.8%	1.2%	0.3%	3.8%	4.9%	7.1%	5.5%	7.0%	28.6%	3.8%	5	7	10	10
12	0.0%	0.3%	0.7%	1.1%	0.2%	3.5%	4.5%	6.5%	4.7%	6.2%	26.7%	3.5%	5	8	10	10
11	0.0%	0.3%	0.6%	1.0%	0.1%	3.1%	4.1%	5.9%	3.9%	5.4%	24.8%	3.1%	5	8	10	10
10	0.0%	0.3%	0.6%	0.9%	0.1%	2.8%	3.6%	5.2%	3.2%	4.6%	22.9%	2.8%	5	9	10	10
9	0.0%	0.2%	0.5%	0.7%	0.0%	2.4%	3.2%	4.6%	2.6%	3.9%	20.9%	2.4%	5	10	10	10
8	0.0%	0.2%	0.5%	0.6%	0.0%	2.1%	2.8%	4.0%	2.0%	3.2%	18.8%	2.0%	5	10	10	10
7	0.0%	0.2%	0.4%	0.5%	0.0%	1.8%	2.4%	3.4%	1.5%	2.6%	16.7%	1.5%	7	10	10	10

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1 **Table 4A-4. Relative yield loss for ten individual crop species and median at various W126 index values.**

W126	Barley	Lettuce	Field Corn	Grain Sorghum	Peanut	Cotton	Soybean	Winter Wheat	Potato	Kidney Bean	Median (10 species)	Number of Species ≤ 5%	Number of Species ≤ 10%	Number of Species ≤ 20%	Number of Species > 5% and ≤ 10%	Number of Species > 10% and ≤ 20%
30	0.1%	5.1%	2.9%	2.3%	10.4%	16.3%	15.7%	22.5%	20.2%	36.1%	13.0%	3	4	7	1	3
29	0.0%	4.4%	2.7%	2.1%	9.7%	15.6%	15.0%	21.0%	19.4%	34.0%	12.4%	4	5	8	1	3
28	0.0%	3.7%	2.4%	2.0%	9.1%	14.9%	14.4%	19.5%	18.7%	31.9%	11.8%	4	5	9	1	4
27	0.0%	3.1%	2.2%	1.9%	8.6%	14.1%	13.7%	18.0%	18.0%	29.8%	11.2%	4	5	9	1	4
26	0.0%	2.6%	1.9%	1.7%	8.0%	13.4%	13.1%	16.6%	17.2%	27.8%	10.6%	4	5	9	1	4
25	0.0%	2.1%	1.7%	1.6%	7.4%	12.7%	12.5%	15.3%	16.5%	25.8%	10.0%	4	5	9	1	4
24	0.0%	1.7%	1.5%	1.5%	6.9%	12.0%	11.8%	14.0%	15.7%	23.9%	9.4%	4	5	9	1	4
23	0.0%	1.4%	1.3%	1.4%	6.4%	11.3%	11.2%	12.7%	15.0%	22.0%	8.8%	4	5	9	1	4
22	0.0%	1.1%	1.2%	1.3%	5.9%	10.6%	10.6%	11.5%	14.2%	20.1%	8.2%	4	5	9	1	4
21	0.0%	0.9%	1.0%	1.1%	5.4%	10.0%	10.0%	10.4%	13.5%	18.4%	7.7%	4	7	10	3	3
20	0.0%	0.7%	0.9%	1.0%	5.0%	9.3%	9.4%	9.3%	12.7%	16.6%	7.1%	5	8	10	3	2
19	0.0%	0.6%	0.8%	0.9%	4.5%	8.7%	8.8%	8.3%	12.0%	15.0%	6.4%	5	8	10	3	2
18	0.0%	0.4%	0.7%	0.8%	4.1%	8.0%	8.2%	7.3%	11.3%	13.4%	5.7%	5	8	10	3	2
17	0.0%	0.3%	0.6%	0.8%	3.7%	7.4%	7.6%	6.4%	10.5%	11.9%	5.1%	5	8	10	3	2
16	0.0%	0.2%	0.5%	0.7%	3.3%	6.8%	7.0%	5.6%	9.8%	10.5%	4.4%	5	9	10	4	1
15	0.0%	0.2%	0.4%	0.6%	2.9%	6.2%	6.4%	4.8%	9.1%	9.2%	3.9%	6	10	10	4	0
14	0.0%	0.1%	0.3%	0.5%	2.6%	5.6%	5.9%	4.1%	8.4%	7.9%	3.3%	6	10	10	4	0
13	0.0%	0.1%	0.2%	0.5%	2.2%	5.0%	5.3%	3.5%	7.7%	6.8%	2.8%	6	10	10	4	0
12	0.0%	0.1%	0.2%	0.4%	1.9%	4.5%	4.8%	2.9%	7.0%	5.7%	2.4%	8	10	10	2	0
11	0.0%	0.0%	0.2%	0.3%	1.6%	3.9%	4.3%	2.3%	6.3%	4.7%	2.0%	9	10	10	1	0
10	0.0%	0.0%	0.1%	0.3%	1.4%	3.4%	3.8%	1.9%	5.6%	3.8%	1.6%	9	10	10	1	0
9	0.0%	0.0%	0.1%	0.2%	1.1%	2.9%	3.3%	1.5%	4.9%	3.0%	1.3%	10	10	10	0	0
8	0.0%	0.0%	0.1%	0.2%	0.9%	2.5%	2.8%	1.1%	4.3%	2.4%	1.0%	10	10	10	0	0
7	0.0%	0.0%	0.0%	0.1%	0.7%	2.0%	2.3%	0.8%	3.6%	1.8%	0.8%	10	10	10	0	0

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1 **Table 4A-5. Tree seedling RBL and CYL estimated for seasonal W126 O₃ exposure.**

W126 index value for exposure period	Tree seedling biomass loss ^A		Crop yield loss ^C	
	Median Value ^B	Individual Species	Median Value ^D	Individual Species
23 ppm-hrs	Median species w. 7.6% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤ 10% loss: 8/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 8.8 % loss ^D	≤ 5% loss: 4/10 species >5,<10% loss: 1/10 species >10,<20% loss: 4/10 species >20: 1/10 species
22 ppm-hrs	Median species w. 7.2% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 8.2 % loss ^D	≤ 5% loss: 4/10 species >5,<10% loss: 1/10 species >10,<20% loss: 4/10 species >20: 1/10 species
21 ppm-hrs	Median species w. 6.8% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 7.7 % loss ^D	≤ 5% loss: 4/10 species >5,<10% loss: 3/10 species >10,<20% loss: 3/10 species
20 ppm-hrs	Median species w. 6.4% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 7.1 % loss ^D	≤ 5% loss: 5/10 species >5,<10% loss: 3/10 species >10,<20% loss: 2/10 species
19 ppm-hrs	Median species w. 6.0% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 6.4 % loss ^D	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10,<20% loss: 2/10 species
18 ppm-hrs	Median species w. 5.7% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 5.7 % loss ^D	≤ 5% loss: 5/10 species >5,<10% loss: 3/10 species >10,<20% loss: 2/10 species
17 ppm-hrs	Median species w. 5.3% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 9/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 5.1 % loss ^D	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10,<20% loss: 2/10 species
16 ppm-hrs	Median species w. 4.9% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 6/11 species ≤ 10% loss: 10/11 species >30% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 5/10 species >5,<10% loss: 4/10 species >10,<20% loss: 1/10 species

W126 index value for exposure period	Tree seedling biomass loss ^A		Crop yield loss ^C	
	Median Value ^B	Individual Species	Median Value ^D	Individual Species
15 ppm-hrs	Median species w. 4.5% loss ^B	\leq 2% loss: 5/11 species \leq 5% loss: 6/11 species \leq 10% loss: 10/11 species >30% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: 6/10 species >5, <10% loss: 4/10 species
14 ppm-hrs	Median species w. 4.2% loss ^B	\leq 2% loss: 5/11 species \leq 5% loss: 6/11 species \leq 10% loss: 10/11 species >30% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: 6/10 species >5,<10% loss: 4/10 species
13 ppm-hrs	Median species w. 3.8% loss ^B	\leq 2% loss: 5/11 species <5% loss: 7/11 species <10% loss: 10/11 species >20% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: 6/10 species >5, <10% loss: 4/10 species
12 ppm-hrs	Median species w. 3.5% loss ^B	\leq 2% loss: 5/11 species \leq 5% loss: 8/11 species \leq 10% loss: 10/11 species >20% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: 8/10 species >5,<10% loss: 2/10 species
11 ppm-hrs	Median species w. 3.1% loss ^B	\leq 2% loss: 5/11 species \leq 5% loss: 8/11 species \leq 10% loss: 10/11 species >20% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: 9/10 species >5, <10% loss: 1/10 species
10 ppm-hrs	Median species w. 2.8% loss ^B	\leq 2% loss: 5/11 species \leq 5% loss: 9/11 species <10% loss: 10/11 species >20% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: 9/10 species >5,<10% loss: 1/10 species
9 ppm-hrs	Median species w. 2.4% loss ^B	\leq 2% loss: 5/11 species \leq 5% loss: 10/11 species >20% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: all species
8 ppm-hrs	Median species w. 2.0% loss ^B	\leq 2% loss: 5/11 species \leq 5% loss: 10/11 species >15% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: all species
7 ppm-hrs	Median species w. \leq 2% loss ^B	\leq 2% loss: 7/11 species \leq 5% loss: 10/11 species >15% loss: 1/11 species	Median species w. \leq 5% loss ^D	\leq 5% loss: all species

A Estimates here are based on the 11 E-R functions for tree seedlings described in section 4A.1.
 B This median value is the median of the composite E-R functions for 11 tree species in Table 4A-3.
 C Estimates here are based on the 10 E-R functions for crops described in section 4A.1.
 D This median value is the median of the composite E-R functions for 10 crops in Table 4A-4.

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1 **4A.2 TREE SEEDLING RBL STUDIES**

2 The experimental cases on which the 11 species-specific E-R functions are based are
3 listed in Table 4A-6 below. As summarized in section 4A.1.1 (and described more fully in
4 Attachment 1 below), 51 E-R functions were derived, one for each of row in Table 4A-6 (e.g.,
5 Lee and Hogsett, 1996, Table 12 and 1996 AQCD, Table 5-28). As indicated by the rows in
6 Table 4A-6, the cases are defined by the species, the exposure (e.g., year), and harvest time (e.g.,
7 immediately after exposure or the subsequent spring) of the dataset used to derive each of the 51
8 functions. Thus, the eleven species-specific functions for the eleven tree species are represented
9 by the 51 cases. As described in section 4A.1 above, species-specific (composite) functions were
10 derived for each species, and Table 4A-5 above presents median RBL estimates from the 11
11 species-specific functions.

12 The O₃ exposure studies represented by the 51 cases were conducted from 1988 to 1992
13 at the U.S. Environmental Protection Agency research laboratory in Corvallis, Oregon, Michigan
14 Technological University’s Ford Forestry Center in Alberta, Michigan and by researchers from
15 Appalachian State University at Great Smoky Mountains National Park near Gatlinburg,
16 Tennessee (Hogsett et al 1995; Hogsett et al., 1997; Neufeld et al 2000; Neufeld et al., 1995;
17 Lefohn et al., 1991; Karnosky et al., 1996; Anderson et al., 1997). Similar experimental
18 protocols were used to expose seedlings to O₃ in 3-meter diameter, 2.4-meter tall modified open-
19 top chambers (Heagle et al., 1973). Experiments used a common standard operating procedure
20 developed by the US EPA to ensure federal guidelines for data quality were met (Hogsett et al.,
21 1985). For all studies at all sites, the experimental design was a single-factor nested experiment
22 with a range of O₃ treatment levels including charcoal-filtered air (control), a baseline O₃ profile
23 (1.0x ambient) and several modified O₃ profiles (e.g., 0.5x, 1.5x, 2.0x ambient air O₃), with
24 multiple replicate chambers for each treatment. For experiments described in Karnosky et al
25 (1996), the “baseline ambient” is a modified profile intended to reflect 6-year averages of
26 Pinkerton and Lefohn (1987).

27 Based on archived datasets at U.S. EPA U.S. EPA, Center for Public Health and
28 Environmental Assessment, Pacific Ecological Systems Division, Corvallis, WA, for some of the
29 exposures, O₃ treatments across the exposure periods of various dates and durations are given in
30 Table 4A-6 in terms of W126, SUM06 and N100. Additionally, SUM06 exposures previously
31 reported in Hogsett et al. (1995) are also presented as available.

1 **Table 4A-6. Individual tree seedling experimental cases for which E-R functions were derived in Lee and Hogsett (1996).**

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Study ID ^A	Species	Site	Year exp'd	Exposure Period (days) ^B	Exposure (derived from hourly O ₃ concentrations over the identified exposure periods). Values are averages of replicates; N100 values are rounded to whole numbers. W126 and SUM06 are for 12-hr periods 8am-8pm. ^C								Harvest ^D	Study/Source and notes, with SUM06 (ppm-hr) ^E reported for full exposure period, e.g., per Hogsett et al 1997, Table 2 (which does not specify whether 12 or 24 hrs SUM06).
<p><i>Rows specify individual experimental datasets (uniquely defined by 1st four columns and harvest) for E-R functions in Lee & Hogsett, 1996 (e.g., Table 12), as described in Attachment 1 to this Appendix (and also presented in 1996 AQCD, Table 5-28).</i></p>														
1	Aspen - wild	OR	1989	6/6-9/18 (105) ^F	SUM06	0		0.7	76.6	62.9	103.9	104.4	1	
1	Aspen - wild	OR			:	0	6.8	48.5	56.0	92.3	97.9	2		
					W126:	0	0	5	228	82	472			
2	Aspen - wild	OR	1991	6/5-9/11 (99) ^F	SUM06	0			13.6	25.8	77.7		1	
2	Aspen - wild	OR			:	0		11	23.4	70.1		2		
					W126:	0		18	71	296				
3	Aspen - wild	OR	1990	6/5-9/19 (107) ^F	SUM06	0			15.1	62	86		1	Hogsett (unpublished) cited in Hogsett et al., 1997 Hogsett et al., 1995, SUM06: 0.2, 16.1, 72.1, 102.8
3	Aspen - wild	OR			W126:	0		12.1	54.7	76.6		2		
4	Aspen - 216	MI	1990	6/20-9/10 (82)	SUM06	0	6.8	7.3	8	26.1			1	May be reported in Karnosky et al., 1996, where detailed description has similarities (all 4 genotypes, 5 exposures in 1990), but with dates as June 20 to Sept 16 in 1990 (which tally to ~88 days), vs recovered ORD dataset dates that match 82 days in Lee & Hogsett 1996 & 1996 AQCD. This experiment is not listed in the Hogsett 1995 & 1997 papers. Karnosky et al 1996 presents N100 of 0, 0, 4, 42, 79 (for 1990) and 0, 24, 38, 45, 84 (for 1991).
4	Aspen 253	MI			W126:	0	5.7	6.5	7	22.5			1	
4	Aspen 259	MI			N100:	0	1	7	6	60			1	
4	Aspen - 271	MI												
5	Aspen - 216	MI	1991	6/9-9/14 (98)	SUM06	0							1	Karnosky et al (1995, in press) cited in Hogsett et al., 1995 Hogsett et al., 1997 for 259, 271, WT, which reported SUM06: 0.0, 11.5, 24.5, 32.4, 40.3, 60.5. Published as Karnosky et al., 1996, who report exposure of clones 216, 259 and 271 (and also WT seedlings) June 9-Sept 14, 1991, via 5 exposures (0, 0.5x, 1x, 1.5x and 2x). The N100 reported for these exposures are: 0, 24, 38, 45, and 84. Across clone average had statistically significant total biomass loss at highest exposures (as did 216).
5	Aspen - 259	MI			W126:	0		19.2		36.3			1	
5	Aspen - 271	MI			N100:	0		16.6		31.5		86	1	

Study ID ^A	Species	Site	Year exp'd	Exposure Period (days) ^B	Exposure (derived from hourly O ₃ concentrations over the identified exposure periods). Values are averages of replicates; N100 values are rounded to whole numbers. W126 and SUM06 are for 12-hr periods 8am-8pm. ^C								Harvest ^D	Study/Source and notes, with SUM06 (ppm-hr) ^E reported for full exposure period, e.g., per Hogsett et al 1997, Table 2 (which does not specify whether 12 or 24 hrs SUM06).
Rows specify individual experimental datasets (uniquely defined by 1 st four columns and harvest) for E-R functions in Lee & Hogsett, 1996 (e.g., Table 12), as described in Attachment 1 to this Appendix (and also presented in 1996 AQCD, Table 5-28).														
6	Aspen-wild	MI	1991	6/9-9/14 (98)	SUM06	0	14.2	19.2	32.0	36.3			1	Karnosky et al (1995, in press) cited in Hogsett et al., 1995 Hogsett et al., 1997 for 259, 271, WT, which reported SUM06: 0.0, 11.5, 24.5, 32.4, 40.3, 60.5 Published as Karnosky et al., 1996, who report exposure of WT seedlings June 9-Sept 14, 1991, via 5 exposures (0, 0.5x, 1x, 1.5x and 2x). The N100 reported for these exposures are: 0, 24, 38, 45, and 84.
7	Douglas Fir	OR	1989	6/7-9/27 (113)	SUM06	0.1			16.4	66.4	91.6	110.4	1	
7	Douglas Fir	OR			W126:	0			13.3	59.2	82.8	103.4	2	
7	Douglas Fir	OR	Plus 1990	6/5-10/3 (121) 2-yr total =234 days	SUM06	0.1			16.6	69.0	95.1	117.1	3	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997, SUM06: 0.1, 33.4, 147.2, 207.2, 261.5 (for full 234 days)
7	Douglas Fir	OR			W126:	0			13.5	61.5	85.8	109.5	4	
8	Douglas Fir	OR	1991	6/5-9/30 (118)	SUM06	0			16.1	30.6	66.8	91.7	1	
8	Douglas Fir	OR			W126:	0			13	27.7	59.8	82.6	2	
8	Douglas Fir	OR	Plus 1992	6/2-9/21 (112) 2-yr total =230 days	SUM06	0.1			14.7	28.1	63.9	88.2	3	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997, SUM06: 0.1, 30.4, 60.6, 143.0, 202.9 (for full 230 days)
9	Ponderosa Pine	OR	1989	6/7-9/27 (111)	SUM06	0			0.7		83.2	113.0	1	
9	Ponderosa Pine	OR			W126:	0			7.3		53.8	100.4	2	
10	Ponderosa Pine	OR	1989	6/7-9/27 (113)	SUM06	0.1			16.4	66.4	91.6	110.4	1	May be described in Andersen et al., 1997 (although only 2 treatments plus control are reported): Seedlings exposed to O ₃ for two growing seasons were statistically significant smaller than CF-exposed seedlings (SUM00 greater than 253). Total biomass reduced 58% at highest exposure.
10	Ponderosa Pine	OR			W126:	0			13.3	59.2	82.8	103.4	2	
10	Ponderosa Pine	OR	Plus 1990	6/5-10/3 (121) 2-yr total =234 days	SUM06	0.1			16.6	69.0	95.1	117.1	3	
10	Ponderosa Pine	OR			W126:	0			13.5	61.5	85.8	109.5	4	
11	Ponderosa Pine	OR	1991		SUM06	0			16.1	30.6	66.8	91.7	1	

Study ID ^A	Species	Site	Year exp'd	Exposure Period (days) ^B	Exposure (derived from hourly O ₃ concentrations over the identified exposure periods). Values are averages of replicates; N100 values are rounded to whole numbers. W126 and SUM06 are for 12-hr periods 8am-8pm. ^C				Harvest ^D	Study/Source and notes, with SUM06 (ppm-hr) ^E reported for full exposure period, e.g., per Hogsett et al 1997, Table 2 (which does not specify whether 12 or 24 hrs SUM06).			
Rows specify individual experimental datasets (uniquely defined by 1 st four columns and harvest) for E-R functions in Lee & Hogsett, 1996 (e.g., Table 12), as described in Attachment 1 to this Appendix (and also presented in 1996 AQCD, Table 5-28).													
11	Ponderosa Pine	OR		6/5–9/30 (118)	W126: 0 N100: 0			13.0 24	27.7 84	59.8 244	82.6 384	2	Lee and Hogsett, 1999, who statistically significant biomass loss at the 2 highest exposures (12-hr W126 greater than 59)
11	Ponderosa Pine	OR	Plus 1992	6/2–9/21 (112) 2-yr total =230 days	SUM06: 0.1 W126: 0.1 N100: 1			14.7 11.8 19	28.1 25.6 78	63.9 56.9 234	88.2 79.4 340	3	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997, 0.1, 30.4, 60.6, 143.0, 202.9 (for full 230 days)
12	Ponderosa Pine	OR	1992	140								1	
13	Ponderosa Pine	OR	1991	84								1	
14	Red Alder	OR	1990	6/5–10/3 (121)	SUM06: 0.1 W126: 0 N100: 0			16.5 13.5 25	69.0 61.5 253	95.1 85.8 355	117.1 109.5 515	1	
15	Red Alder	OR	1989	6/7–9/27 (113)	SUM06: 0.1			16.4	66.4	91.6	110.4	1	
15	Red Alder	OR			W126: 0 N100: 0			13.3 25	59.2 241	82.8 351	103.4 491	2	
16	Red Alder	OR	1991	6/5–9/30 (118)	SUM06: 0			16.1	30.6	66.8	91.7	1	
16	Red Alder	OR			W126: 0 N100: 0			13 24	27.7 84	59.8 244	82.6 384	2	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997, SUM06: 0.0, 16.0, 31.8, 73.4, 103.6
17	Red Alder	OR	1992	6/2–9/21 (112)	SUM06: 0.1 W126: 0.1 N100: 1			14.7 11.8 19	28.1 25.6 78	63.9 56.9 234	88.2 79.4 340	1	Hogsett (unpublished) per Hogsett et al., 1995 Hogsett et al., 1997, SUM06: 0.1, 14.5, 29.1, 70.1, 99.9.
18	Black Cherry	SM NP G	1989	6/14–8/28 (76)	SUM06: 0 W126: 0 N100: 0	1.9 1.9 0		13.5 11.1 10		25.8 23 77		1	Neufeld et al., 1995 cited in Hogsett et al., 1995 Hogsett et al., 1997, SUM06: 0.0, 1.9, 17.1, 40.6. Also Neufeld and Renfro, 1993. [Statistically significant reduction in highest treatment group]
19	Black Cherry	SM NP	1992	5/20–10/6 (140)	SUM06: 0 W126: 0 N100: 0	0.9 0 0	1.6 1.4 0	18.6 15.1 5		45.6 39.5 103		1	Neufeld, pers comm in Hogsett et al., 1995 Hogsett et al., 1997, SUM06: 0.0, 00, 0.8, 18.1, 50.2. Described in Neufeld et al., 1995, Neufeld and Renfro, 1993 [Statistically significant reduction in highest treatment]
20	Red Maple	SM NP	1988	7/1-8/24 (55)	SUM06: 0 W126: 0 N100: 0		2.8 2.4 0	15.7 12.0 3		64.4 59.8 300		1	Neufeld (pers comm) cited in Hogsett et al., 1995 Hogsett et al., 1997. SUM06: 9.2, 12, 47, 125.4

Study ID ^A	Species	Site	Year exp'd	Exposure Period (days) ^B	Exposure (derived from hourly O ₃ concentrations over the identified exposure periods). Values are averages of replicates; N100 values are rounded to whole numbers. W126 and SUM06 are for 12-hr periods 8am-8pm. ^C							Harvest ^D	Study/Source and notes, with SUM06 (ppm-hr) ^E reported for full exposure period, e.g., per Hogsett et al 1997, Table 2 (which does not specify whether 12 or 24 hrs SUM06).	
Rows specify individual experimental datasets (uniquely defined by 1 st four columns and harvest) for E-R functions in Lee & Hogsett, 1996 (e.g., Table 12), as described in Attachment 1 to this Appendix (and also presented in 1996 AQCD, Table 5-28).														
21	Tulip Poplar	SM NP	1990	6/30–9/12 (75)	SUM06	0.1	0.1	0.9	13.3	30.1			1	
					W126:	0.1	0.1	1.5	11.2	39.7				
					N100:	0	0	0	12	51				
21	Tulip Poplar	SM NP	plus-1991	5/3–8/19 (109) 2-yr total =184 days	SUM06	0	0.3	0.7	22.7	54.2			3	Neufeld (pers comm) cited in Hogsett et al., 1995 Hogsett et al., 1997, SUM06: 0.1, 0.5, 1.4, 34.5, 88.7 (for full 184 days)
					W126:	0	0.4	1.5	18.7	45.3				
					N100:	0	1	0	8	102				
22	Tulip Poplar	SM NP	1992	5/20–10/8 (142)	SUM06	0	0	0.9	18.7	45.9			1	
					W126:	0	0	1.4	15.2	39.7				
					N100:	0	0	0	5	103				
23	Loblolly GAKR 15-23	AL	1988-89	5/23/88-11/28/89 (555)									3	Qiu et al., 1992 and Lefohn et al., 1992 (cited by Hogsett et al., 1995 Hogsett et al., 1997; SUM06: 4.9, 58.5, 301.5, 507)
23	Loblolly GAKR 15-91	AL			W126: (24hr)	6.7			50.8	267.8	486		3	Statistically significant reductions at highest treatment for GARK15-91 only (90 th percentile for 2 nd highest treatment ranges 142-156 ppb across replicates; maximum ranges 210-260 ppb)
24	Sugar Maple	MI	1990	(83)	SUM06	0	0.60.	6.8	7.3	8	26.1		1	
					W126:	0	8	5.7	6.5	7	22.5			
					N100:	0	0	1	7	6	60			
24	Sugar Maple	MI	Plus 1991	(97) 2-yr total =180 days									3	Karnosky (pers. comm.) cited by Hogsett et al., 1995 Hogsett et al., 1997 (SUM06: 0.0, 25.2, 27.8, 49.8, 67.6, 94.4, for full 180 days). May be described in Rebbeck and Loats, 1997, who reported no statistically significant treatment effects in any of the seedlings exposed to O ₃ between two individual seasons or after exposure to 304 ppm (SUM00 index) over two growing seasons (total of 225 days).
25	E. White Pine	MI	1990	6/20–9/10 (83)	SUM06	0	0.60.	6.8	7.3	8	26.1		1	Karnosky (pers. comm.) cited by Hogsett et al., 1995 Hogsett et al., 1997 (SUM06: 0.0, 25.2, 27.7, 49.8, 64.2, 94.4, for full 180 days)
					W126:	0	8	5.7	6.5	7	22.5			
					N100:	0	0	1	7	6	60			
25	E. White Pine	MI	Plus 1990	(97)									3	May be described in Isebrands et al., 2000 pg 170 which reported no statistically significant difference in height,

Study ID ^A	Species	Site	Year exp'd	Exposure Period (days) ^B	Exposure (derived from hourly O ₃ concentrations over the identified exposure periods). Values are averages of replicates; N100 values are rounded to whole numbers. W126 and SUM06 are for 12-hr periods 8am-8pm. ^C								Harvest ^D	Study/Source and notes, with SUM06 (ppm-hr) ^E reported for full exposure period, e.g., per Hogsett et al 1997, Table 2 (which does not specify whether 12 or 24 hrs SUM06).
Rows specify individual experimental datasets (uniquely defined by 1 st four columns and harvest) for E-R functions in Lee & Hogsett, 1996 (e.g., Table 12), as described in Attachment 1 to this Appendix (and also presented in 1996 AQCD, Table 5-28).														
				2-yr total =180 days									stem, root or current year needle biomass in response to O ₃	
26	Virginia Pine	SM NP	1992	5/4–10/9 (159)	SUM06	0	2	2.9		24.6	56.1	1	Neufeld (pers. comm.) cited in Hogsett et al., 1995 Hogsett et al., 1997 (SUM06: 0.0, 0.0, 1.9, 21.7, 51.6) May be described in Neufeld et al. (2000), who reported no statistically significant treatment effects on biomass from 152-day duration (SUM06 up to 56.2).	
<p>A Study ID as in Lee and Hogsett (1996), Table 12 (and 1996 AQCD).</p> <p>B Duration corresponds to length in days of the first year of exposure for Harvests 1 and 2 and to the total length of the first and second years' exposure periods for Harvest 3.</p> <p>C Exposure metric values derived from recently recovered datasets associated with Lee and Hogsett research at U.S. EPA, Center for Public Health and Environmental Assessment, Pacific Ecological Systems Division, Corvallis, WA.</p> <p>D Harvest 1 occurs immediately following end of first year of exposure. Harvest 2 occurs in spring following first year of exposures. Harvest 3 occurs immediately following end of second year of exposures. Harvest 4 occurs in spring following second year of exposures.</p> <p>E First SUM06 treatment value corresponds to charcoal-filtered exposure (Hogsett et al., 1997 Table 2).</p> <p>F For the three Oregon exposures of aspen in 1989, 1990 and 1991, the durations of exposure reported in Lee and Hogsett, 1996, Hogsett et al 1995 and Hogsett et al 1997 (84, 118 and 112 days, respectively) differ from the number of days of exposure data for each in the recovered dataset for the research described in footnote C above (105, 99 and 107 days, respectively). Based on review of the information by staff from USEPA, CPHEA, PSAD (including coauthor on Lee and Hogsett, 1996), the values for numbers of days duration in the 1995-1997 publications are presumed to reflect typographic errors. Accordingly, the exposure metric values reported in this table are concluded to reflect the study exposures and the dataset from which the E-R functions were derived in Lee and Hogsett, 1996.</p> <p>G SMNP = Smoky Mountains National Park.</p> <p>H The duration of exposure reported in Lee and Hogsett, 1996, Hogsett et al 1995 and Hogsett et al 1997 for the 1992 tulip poplar and Virginia pine exposures (81 and 98 days, respectively) differs from the number of days of exposure data in the recovered dataset for the research (142 and 159 or 152 days). The lead investigator on these studies has affirmed that the values for number of days duration in the 1995-1997 publications are erroneous. Accordingly, the exposure metric values reported in this table are concluded to reflect the study exposures and the dataset from which the E-R functions were derived in Lee and Hogsett, 1996.</p>														

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1 **4A.3 ANALYSIS OF RBL ACROSS MULTIPLE YEARS**

2 There are few studies of multiple year O₃ exposures that provide detailed O₃
3 concentrations during the exposures and quantify growth for each year that might be analyzed
4 with regard to the influence of each season's O₃ exposures across a multiple-year period. In
5 section 4A.3.1, one such study that has been analyzed in the 2013 and 2020 ISAs is described.
6 Section 4A.3.2 includes a somewhat basic set of example calculations as a theoretical illustration
7 that considers potential impacts of different patterns of annual cumulative O₃ exposures across
8 multiple years.

9 **4A.3.1 Comparison of Predicted and Observed O₃ Growth Impacts in the 2013 and 2020** 10 **ISAs**

11 The 2013 and 2020 ISAs present comparisons of aspen stand growth observations from
12 an Aspen FACE multiyear O₃ exposure study with predictions derived through the application of
13 a median composite E-R function for wild aspen and aspen clones⁷ to seasonal W126 index
14 values (2013 ISA, section 9.6.3.2; 2020 ISA, Appendix 8, Figure 8-17). The Aspen FACE study
15 monitored growth of aspen stands annually from 1997 through 2003 (King et al., 2005).⁸ Growth
16 was monitored for stands grown in ambient air and under elevated O₃ conditions. The elevated
17 O₃ treatment involved increasing hourly concentrations by approximately 1.5 times over the O₃
18 concentrations occurring in ambient air at the site (King et al., 2005).

19 For the ISA comparisons of growth impacts predicted using the aspen E-R function
20 (described in section 4A.1.1 above) to those observed in the study, hourly O₃ measurements were
21 obtained from the authors (for both the “ambient” and “elevated” treatments) and used to
22 calculate seasonal W126 index. For the 2013 ISA, a cumulative (multiyear) seasonal average
23 W126 index was related to growth response and for the 2020 ISA the single-year seasonal W126
24 index was used. The values for “observed” above ground total biomass for the aspen stands were
25 derived from measurements obtained from the authors and allometric equations (2013 ISA,
26 section 9.6.3.2; King et al., 2005 and associated Corrigendum).⁹

⁷ The median composite function used in the ISAs “was developed from NHEERL/WED data for 11 studies of wild-type seedlings of aspen as well as four clonally propagated genotypes” (2013 ISA, p. 9-133).

⁸ Other studies have involved observations involving the same aspen stand extended out to 2008 (e.g., Talhelm et al., 2014; Zak et al., 2011). Complications associated with performing similar types of comparisons over this longer time period relate to variation in both the tree measurements taken over the extended period (e.g., diameter measurements at varying tree heights), and the O₃ treatments (e.g., the difference in single-year W126 index ranged from approximately 20 to 30 ppm-hrs through 2003 and then dropped to 10 ppm-hrs for four of the last five years), as well as changing growth patterns associated with aging trees.

⁹ The publication by King et al., (2005) reports on measurements for the years 1997 through 2003.

1 Both the 2013 ISA comparison of observed biomass to predicted biomass based on
2 application of the E-R function to W126 in terms of cumulative (multiyear) seasonal average¹⁰
3 and the 2020 ISA comparison using W126 in terms of single year seasonal index indicate the E-
4 R function to describe generally similar O₃ impacts on Aspen biomass. Based on the 2013
5 analysis (presented in the Tables 9-14 and 9-15, and Figure 9-20 of the 2013 ISA), the 2013 ISA
6 concludes “the agreement between predictions ... and observations was very close” and “the
7 function based on one year of growth was shown to be applicable to subsequent years.” (p 9-
8 135).¹¹ The 2020 ISA also notes a closeness of predictions to observations (2020 ISA, Appendix
9 8, p. 8-192 and Figure 8-17). The variation in the comparisons of predictions to observations in
10 the two presentations illustrate the variability inherent in the magnitude of growth impacts of O₃
11 and also the quantitative relationship of O₃ exposure and RBL, while also supporting ISA
12 conclusions of a general agreement of model predictions using either multiyear or single year
13 W126 estimates with experimental observations (2013 ISA, Figure 9-20; 2020 ISA, Appendix 8,
14 Figure 8-17).

15 **4A.3.2 Example Calculations Comparing Estimated Impacts of Constant and Annually** 16 **Varying Seasonal Exposure**

17 This section explores estimates of aspen growth affected by multiple years of O₃
18 exposure based on application of the E-R function for aspen described in section 4A.1.1 above.
19 Estimates of aspen absolute biomass are calculated in response to O₃-related RBL for several
20 scenarios with different ways of expressing single-year O₃ exposure conditions that all have the
21 same 3-year average W126 index. In this way, absolute biomass of aspen is estimated across
22 multiple years in response to O₃ exposures expressed as a constant annual W126 index value and
23 compared to estimated absolute biomass in response to O₃ exposures expressed as the same
24 W126 index value in terms of a 3-year average but with varying annual values. Several different
25 scenarios with varying annual W126 are included, with all meeting the 3-year average limit of
26 the constant W126 index scenario (Figure 4A-15), with the extent of the variation reflecting what
27 is shown to be common at U.S. monitoring locations, although with the highest single-year value
28 somewhat higher than that occurring in U.S. monitoring locations that meet the existing NAAQS
29 (e.g., Appendix 4D, section 4D.3.1.2).

¹⁰ The cumulative seasonal average for each year is calculated as the average of the seasonal W126 index values for that year and all of the preceding years.

¹¹ Using the values reported in Table 9-15 of the 2013 ISA (which are plotted in Figure 9-20), to derive correlation coefficients related to that analysis, the r^2 for predicted O₃ impact versus observed impact is 0.99 and for the percent difference versus year is approximately 0.85. This indicates a strong correlation for the 2013 ISA analysis of the experimental observations with predictions based on a cumulative multiyear W126 index, and a good fit for the exposure metric reflecting cumulative multiyear exposure.

1 This analysis is not intensive or elaborate; rather, it is a mathematical exercise intended to
 2 provide an illustration of concepts associated with application of the E-R functions described in
 3 section 4A.1 using data from a study with aspen of the effects of a six-year exposure on
 4 accumulating biomass (King et al., 2005), which is also utilized in a different type of analysis in
 5 the 2013 ISA, that is summarized in the ISA and also in section 4A.3.1 above (2020 ISA,
 6 Appendix 8, Figure 8-17; 2013 ISA, section 9.6.3.2).

7 **Description of Analysis:** The analysis presented here is intended to simply illustrate the
 8 application of the tree seedling E-R function for aspen over a multi-year period using two types
 9 of air quality scenarios: (1) one in which the O₃ concentrations are limited such that each year's
 10 W126 is no higher than 17 ppm-hrs, and (2) a second in which the O₃ concentrations are allowed
 11 to vary each year as long as the 3-year average is no higher than 17 ppm-hrs. More specifically,
 12 the two scenarios are (1) repeated years of W126=17 ppm-hrs and (2) repeated 3-year cycles of
 13 the same varying W126 (e.g., 10, 17 and 24 ppm-hrs).

	O ₃ exposure in terms of single year W126 index (ppm-hrs)					
Scenario	Year 1	Year 2	Year 3	Year 4	Year 5	Year 6
Constant	17	17	17	17	17	17
Varying	10	24	17	10	24	17
Varying	24	17	10	24	17	10
Varying	24	10	17	24	10	17
Varying	10	17	24	10	17	24

15 This analysis is intended to inform consideration of potential magnitude of an over or
 16 under estimation of growth reduction when the target W126 value was calculated from a 3-year
 17 average or for each individual year. In this analysis, above-ground tree biomass is estimated for
 18 each year through a six-year period.¹² The example for this analysis uses aspen, beginning with a
 19 seedling, and utilizes data on growth rates (annual biomass increases) for the control treatment in
 20 a study by King et al., 2005. Based on the annual measurements,¹³ we derived the following
 21 linear model ($r^2 = 0.4137$):

$$\text{W126 scenario annual growth} = 0.2395 * \text{Previous Year Biomass} + 215.05$$

¹² In order to avoid extrapolation baseline growth beyond that presented in King et al. (2005), the analysis is limited to the six-year time period. While other Aspen FACE studies have followed the same stand for additional years, there are aspects of the longer dataset (e.g., different height of tree measurements) that would contribute uncertainties that lead to the decision to limit the analysis to this duration.

¹³ Individual tree growth measurements from Aspen FACE (1997-2008) research, including annual biomass increases in King et al. (2005), received from researchers (Ozone NAAQS Docket, EPA-HQ-OAR-2018-0279).

1 This function was then used to estimate each year’s annual growth prior to application of the O₃
2 growth effect which was estimated by applying the established E-R function for aspen. In our
3 analysis, above ground biomass loss¹⁴ was calculated using the estimated growth rate (yearly
4 biomass production) and the relative biomass loss (RBL) for the pertinent W126 value based on
5 the aspen E-R function. This biomass loss was calculated for the 3-year average W126 of 17
6 ppm and for each of the three individual year values of 10, 17 and 24 ppm (Table 4A-7).

7 The above ground biomass of the aspen stand across the six years of growth was
8 compared across the two exposure scenarios (Figure 4A-15; Table 4A-7). The difference
9 between the two scenarios in total above ground biomass for the stand varied from year to year.
10 After the first year, this difference in the year’s total above ground biomass (not to be confused
11 with annual growth in biomass, to which RBL is applied) was always less than 3%. In summary,
12 the estimated impact of O₃ on absolute biomass of aspen following multiple years of exposure
13 does not differ appreciably whether the E-R function is applied to annual growth with a single-
14 year W126 index varying across a 3-year period or with a W126 index for each year set equal to
15 the average across the three years. In summary, and consistent with the analysis described above,
16 the estimated impact of O₃ using different annual measurements does not differ appreciably
17 across the six years of growth.

18 **Summary of Analysis Limitations, Assumptions and Uncertainties:** Given the limited
19 availability of multiple year O₃ exposure studies providing detailed O₃ concentrations during the
20 exposures and quantified annual growth, as well as the simply conceptual or illustrative nature of
21 the analysis, there are multiple inherent assumptions, limitations and uncertainties.

- 22 • Consistent with the general concept that a tree’s annual growth is related to the size of the
23 tree going into the growing season, the analysis derives estimates of annual growth as a
24 function of prior year biomass (with the function derived from the annual biomass
25 measurements from the Aspen FACE research).¹⁵ There is uncertainty in the resulting
26 estimates from a number of sources including that the function used did not account for
27 influences other than tree size on annual growth. The impact of these uncertainties
28 (including direction and magnitude) on this analysis are unknown.
- 29 • Variables other than O₃ that can affect growth in a given year (e.g., precipitation,
30 temperature, community competition) are represented in the current analysis only through
31 their effects on the annual measurements provided by the “control” from the aspen study
32 by King et al. (2005) on which the annual growth function is based.

¹⁴ Above-ground growth (foliage and wood) is used consistent with 2013 and 2020 ISA analyses described in section 4A.3.1 above).

¹⁵ This differs from the approach of the analysis in the 2020 PA.

- 1 • Additionally, this analysis is based on aspen, and the specific pattern of differences
2 between the two scenarios might be expected to vary for species with different biomass
3 growth rates (and E-R functions). However, while many multi-year tree growth studies
4 may exist, datasets of tree growth that investigate the impact of O₃ across multiple-year
5 periods (providing annual growth measurements and also detailed records of hourly
6 concentrations that support derivation of W126 index metrics) such as that available for
7 aspen in the study by King et al. (2005) are not prevalent.
- 8 • This example analysis includes a W126 index value of 24 ppm-hrs every third year. Yet,
9 the frequency of such a value is quite rare, as can be seen from the air quality analyses in
10 Appendix 4D, which show that across the period from 2000 through 2020 for even just
11 the subset of sites meeting the current standard but with design value closest to 70 ppb
12 (66-70 ppb), the 99th percentile is below 20 ppm-hrs (PA, Appendix 4D, Figure 4D-8).
13 Focusing just on Class I areas for the full period from 2000 to 2020, there are no more
14 than 15 occurrences of a single-year W126 index value above 19 ppm-hrs, all of which
15 date prior to 2013 (85 FR 49904, August 14, 2020). Thus, this example includes as one of
16 the three years, a magnitude of W126 index that has been quite rarely observed in areas
17 that meet the current standard since 2000. W126 index values below 17 ppm-hrs are more
18 common.
- 19 • The shape of the E-R curve for aspen species is generally linear, as are eight of the other
20 species with E-R curves (see section 4A.1.1 above), but we recognize that varying shapes
21 of curves may have the potential to influence the differences in the comparison analyzed.
22 Although EPA does not have growth information to complete an analysis such as this one
23 for another of the 11 species, uncertainties related to the shape of the two species with
24 less linear E-R curves were considered. Black cherry has an E-R function with a
25 declining slope with increasing W126, with the appearance of leveling off, which
26 produces a smaller change in RBL relative to the change in W126. This slope presents the
27 opposite pattern to that of sugar maple. Sugar maple has a small slope at or below 19
28 ppm-hrs (RBL estimates associated with its E-R function in this range are appreciably
29 lower than those for the aspen¹⁶) and does not have a large change in slope until at or
30 above 26 ppm-hrs (the highest W126 values observed at U.S. ambient air monitoring
31 sites), Furthermore, the geographic range of sugar maple is generally limited to the
32 northeast and upper midwest of the U.S., areas with among the relatively lower W126
33 index levels across the U.S. (see Appendix 4D, Figure 4D-2).¹⁷
- 34 • Additionally, while the availability of multi-year experimental data that can be examined
35 with regard to this issue for the range of exposures investigated here is limited, a multi-
36 year study available in the 2015 review (King et al., 2005) is discussed in section 4A.3.1
37 above (2013 ISA section 9.6.3.2). As summarized in section 4A.3.1, the multi-year

¹⁶ At sites and time periods during 2000 through 2018 in which the current standard was met, and focusing on the higher values of W126 index observed at sites with design values closest to the current standard (e.g., 66 -70 ppb), the sugar maple RBL estimated for the 75th percentile is less than 1% (PA, Appendix 4A, Table 4A-4 and Appendix 4D, Figure 4D-8).

¹⁷ A noteworthy uncertainty regarding the shape of this E-R function is that a W126 index of 22.5 ppm-hrs may be the highest experimental exposure level in the first season of the two season's exposure on which the sugar maple E-R function is based (see Table 4A-6 above).

1 experimental dataset from King et al. (2005) was assessed in the 2013 ISA and is also
2 discussed in the 2020 ISA with regard to growth effects and correspondence of E-R
3 function predictions with study observations (2020 ISA, Appendix 8, section 8.13.2 and
4 Figure 8-17; 2013 ISA, section 9.6.3.2, Table 9-15, Figure 9-20). The analysis in the
5 2013 ISA, which focused on the six years for which the aspen study reported data,
6 compared observed reductions in growth for each year of a 6-year period to those
7 predicted by applying the established E-R function for Aspen to cumulative multi-year
8 average W126 index values (2013 ISA, section 9.6.3.2).¹⁸ One finding of this evaluation
9 was that “the function based on one year of growth was shown to be applicable to
10 subsequent years” (2013 ISA, p. 9-135), indicating that the approach employed in the
11 illustrative analysis presented here -for the initial six years- may be reasonable for the
12 circumstances examined here.¹⁹

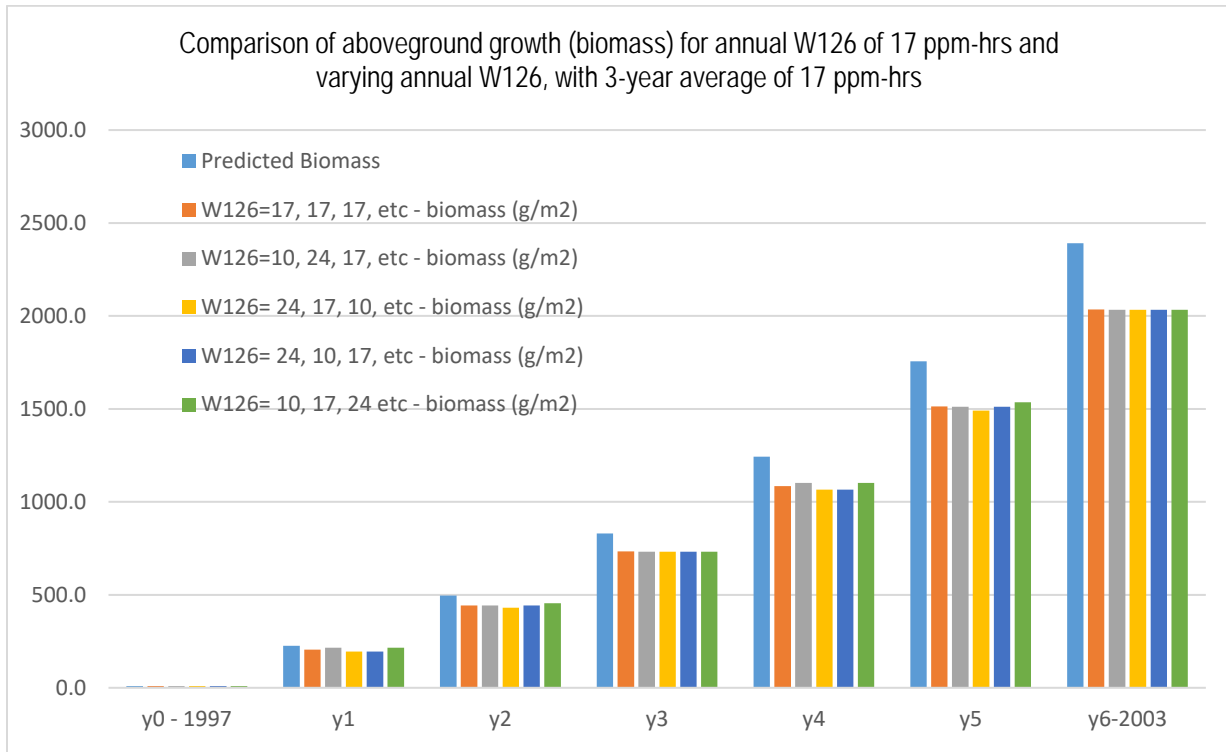
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14

¹⁸ For example, the growth impact estimate for year 1 used the W126 index for year 1; the estimate for year 2 used the average of W126 index in year 1 and W126 index in year 2; the estimate for year 3 used the average of W126 index in years 1, 2 and 3; and so on.

¹⁹ In the 2020 ISA, an evaluation slightly different from that in the 2013 ISA was performed, applying the E-R functions to the W126 index for each year rather than the cumulative multi-year W126 (2020 ISA, Appendix 8, Figure 8-17). This approach, while indicating a just slightly less tight fit to the observations (than the 2013 ISA approach) in the later years, was similarly concluded to be “exceptionally close” to the experimental observations (2020 ISA, Appendix 8, p. 8-192), indicating the aspen E-R functions to predict the yearly findings generally reliably from the six years of exposures of the Aspen FACE experiment.

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Figure 4A-15. Estimated aboveground biomass of aspen with different patterns of annual seasonal W126 index using annual growth as a function of prior year absolute biomass for trees in the same scenario.

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Table 4A-7. Comparison of total aspen above ground biomass estimated for different patterns of varying annual exposures and constant exposures equal to 3-year average (17 ppm-hrs) using annual growth as a function of prior year absolute biomass for trees in the same scenario.

Year	Predicted Biomass*	Growth - % increase	W126=17, biomass (g/m2)	W126=10, 24, 17, etc - biomass (g/m2)	W126=24, 17, 10, etc - biomass (g/m2)	W126=24, 10, 17, etc - biomass (g/m2)	W126=10, 17, 24 etc - biomass (g/m2)	% difference in total tree biomass of W126 10-17-24 vs 17	% difference in total tree biomass of W126 10-24-17 vs 17	% difference in total tree biomass of W126 24-17-10 vs 17	% difference in total tree biomass of W126 24-10-17 vs 17
y0 - 1997	9.1		9.1	9.1	9.1	9.1	9.1				
y1	226.3	2387.14%	205.0	215.0	194.8	194.8	215.0	4.9%	4.9%	-5.0%	-5.0%
y2	495.6	118.97%	443.3	442.9	430.9	442.9	455.5	2.7%	-0.1%	-2.8%	-0.1%
y3	829.3	67.34%	733.1	732.6	732.6	732.6	732.6	-0.1%	-0.1%	-0.1%	-0.1%
y4	1243.0	49.88%	1085.4	1102.8	1066.5	1066.5	1102.8	1.6%	1.6%	-1.7%	-1.7%
y5	1755.8	41.25%	1513.8	1512.5	1490.8	1512.5	1535.0	1.4%	-0.1%	-1.5%	-0.1%
y6-2003	2391.3	36.20%	2034.8	2033.2	2033.2	2033.2	2033.2	-0.1%	-0.1%	-0.1%	-0.1%

* The value in the first row of this and other columns is the total absolute biomass measurement from King et al. 2005, Table 3 (foliage plus wood). The subsequent rows of the first column utilize the function (above) to derive current year biomass as function of prior year biomass. In the other columns, the annual increment derived with the function is reduced by predicted RBL for the applicable W126 index value. The W126-RBL E-R function used is $1 - \exp[-W126/109.81]^{1.2198}$.

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1

2

3

Attachment to Appendix 4A

1 **Derivation of Composite Median Equations (parameterized models)**
2 **in Lee and Hogsett (1996)**
3

4 *The following describes the methodology used to produce the sets of parameters in Tables 2, 12,*
5 *and 13 of Lee and Hogsett (1996), which have been used in some form in AQCDs and ISAs since*
6 *1996. “Regression”, “parameter estimation”, “model estimation” and “model fitting” all refer*
7 *to the same statistical procedure of using nonlinear ordinary least square regression to obtain*
8 *values for model parameters from a dataset.*
9

- 10 1) Tables 12, 13, and 2 in Lee and Hogsett (1996) primarily summarize parameter values
11 estimated through regression from 51 controlled exposure studies of tree seedlings conducted
12 by NHEERL/WED. In those studies, 11 species of trees were exposed to a set of ozone
13 concentrations for durations varying from 55 to 234 days or up to 555 days (in the case of
14 one species).
- 15 2) The model fitted to the data from each of the 51 individual studies (in Table 12) is a three-
16 parameter Weibull model with the following parameterization: $Predicted\ Biomass = A \exp(-[exposure/B]^c)$. When removing the intercept A , this model gives biomass relative to no
17 exposure, and the resulting two-parameter equations all have the same 0-1 range of relative
18 biomass response and can thus be compared or aggregated across studies with different
19 ranges of absolute biomass. $Predicted\ Relative\ Biomass = \exp(-[exposure/B]^c)$ and $Predicted$
20 $Relative\ Biomass\ Loss = 1 - \exp(-[exposure/B]^c)$. When estimating each set of three
21 parameters for each separate study, the ozone exposure was quantified using the 12-hour
22 daytime W126 index, summed over the duration of each study.
23
- 24 a. Table 12 gives parameter values for 51 models, one per study, that reflect W126
25 over each study duration. This table also presents the W126 index estimated for a
26 92-day duration for RBLs of 10% and 20%.
- 27 b. Table 13 presents parameter values for the 51 models given in Table 12, as well
28 as parameter values for composite models for the 11 tree species included in those
29 51 studies, two sets of values per species (one for the median and the second for
30 the 75th percentile). This table also presents W126 index estimates for RBLs of
31 10%, 20% and 30%. These three estimates for the composite models are estimates
32 for a 92-day duration.
- 33 c. Table 2 presents values for composite models for all experiments for all species
34 aggregated.
- 35 3) The median composite models, one per species (table 13) are derived as follows. For each of
36 the studies for a given species, the predicted relative biomass loss is first generated at six
37 values of exposure: 10, 20, 30, 40, 50, and 60 ppm-hr, using the study-specific two-parameter
38 equation. This is done in a way to obtain six values of exposure for 12-hour daytime
39 exposures summed over 92 days.²⁰ All but the median of the relative biomass loss estimates at

²⁰ Since the W126 index is cumulative, and the duration of exposure varied between studies, the calculated values of exposure at which some given percent loss is expected were prorated to 92 days using simple linear scaling. For example, the duration of the first ponderosa pine study in Table 12 is 111 days. To derive the 92day RBL for 10 ppm-hrs, a factor of 92/111 is applied to 10 ppm-hrs before it is input to the experiment-specific equation to derive an RBL estimate for 10 ppm-hrs over a 92-day exposure.

1 each value of exposure are then discarded, and the two-parameter model for relative biomass
2 loss is fitted to the remaining six median points. For example, Ponderosa Pine was the subject
3 of 11 studies; 11 sets of parameters were estimated through regression (see item 2 above); 66
4 values of predicted relative biomass loss were then computed, 11 at each of the six exposure
5 values. All but the median of those 11 relative biomass loss estimates were discarded at each
6 of the six levels of exposure, and the two-parameter model fitted to the six remaining points,
7 giving the Ponderosa Pine median composite equation for a 92-day exposure.

8 4) The all-species, median composite models (table 2) were estimated using the same
9 aggregation method but applied to all 51 studies at once. The 51 equations in Table 12 were
10 used to compute 306 values of relative biomass loss, six values each for the 51 sets of *B* and *C*
11 parameters, with those six values of exposure generated in a way to obtain 12-hour daytime
12 exposures summed over 92 days. The two-parameter model was then fitted to the 75th
13 percentile and the median as in item 3 above. Table 2 also includes the results of the same
14 method using other exposure indices besides the 12-hour W126 index.

15 5) For every equation in the tables, values of exposure at which some given percent loss is
16 expected relative to no exposure, or any other exposure, can be back-calculated using:
17 $Exposure = B * (-\ln(1 - predicted\ relative\ biomass\ loss))^{1/C}$. Some of those expected values of
18 exposure are presented for various loss percentages in tables 2, 12, and 13 for the all-species
19 median composite model, the 51 studies, and the 11 species-level median composite,
20 respectively. In the case of single-study calculations in Table 12, the value of exposure for a
21 given loss percentage was first calculated based on the respective duration of each study, then
22 simply prorated. For example, the duration of Study 1 Harvest 1 in table 12 was 84 days and
23 the exposure at which 10% loss is expected over that duration is 13.71 ppm-hr. The prorated
24 exposure value for a 10% loss over 92 days is calculated as $13.71 * 92 / 84 = 15.01$ ppm-hr.
25 Median models for species in Table 13 or for all species in Table 2 were parameterized with
26 92-day durations and the exposure values for the various loss percentages did not therefore
27 require prorating.

28

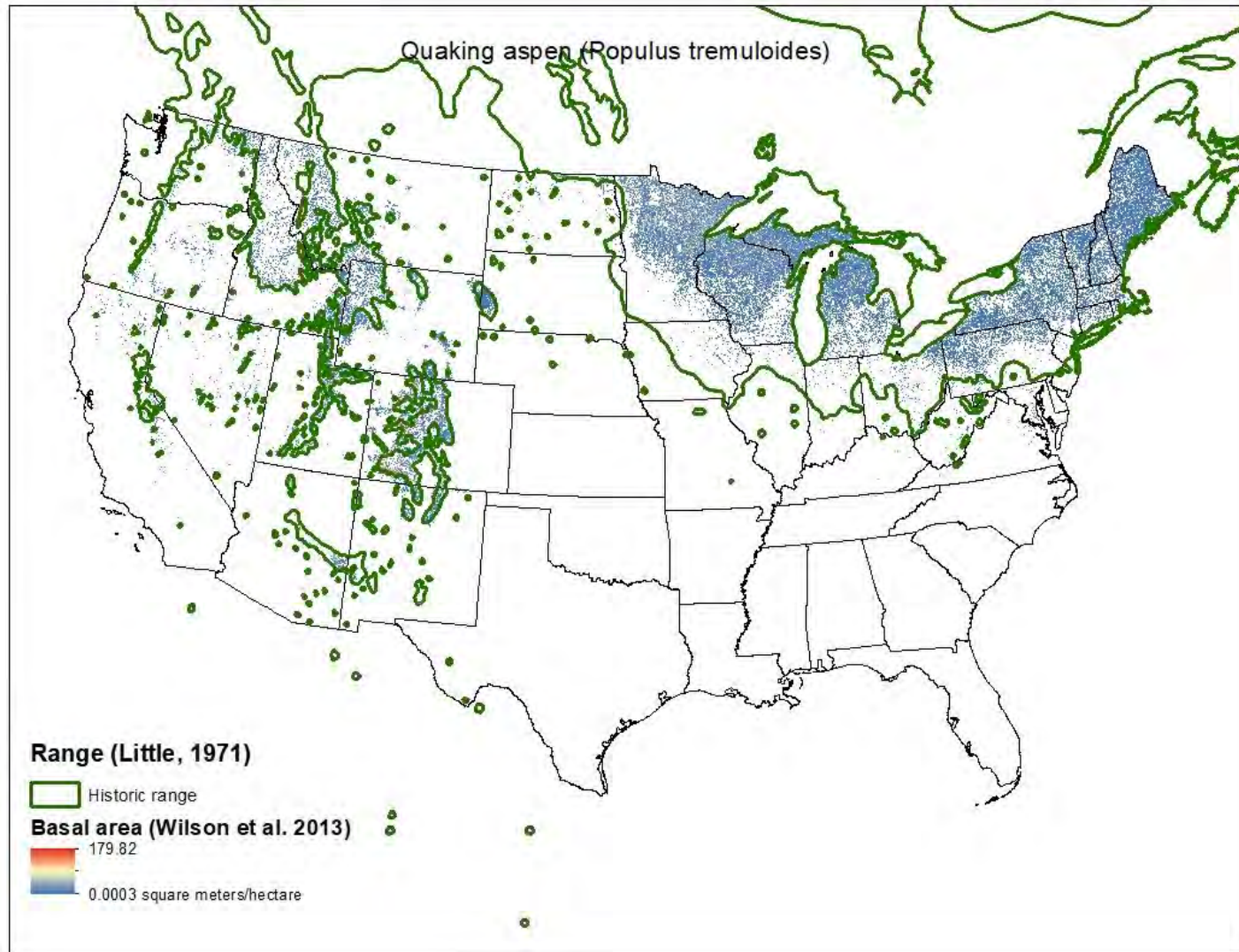
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APPENDIX 4B

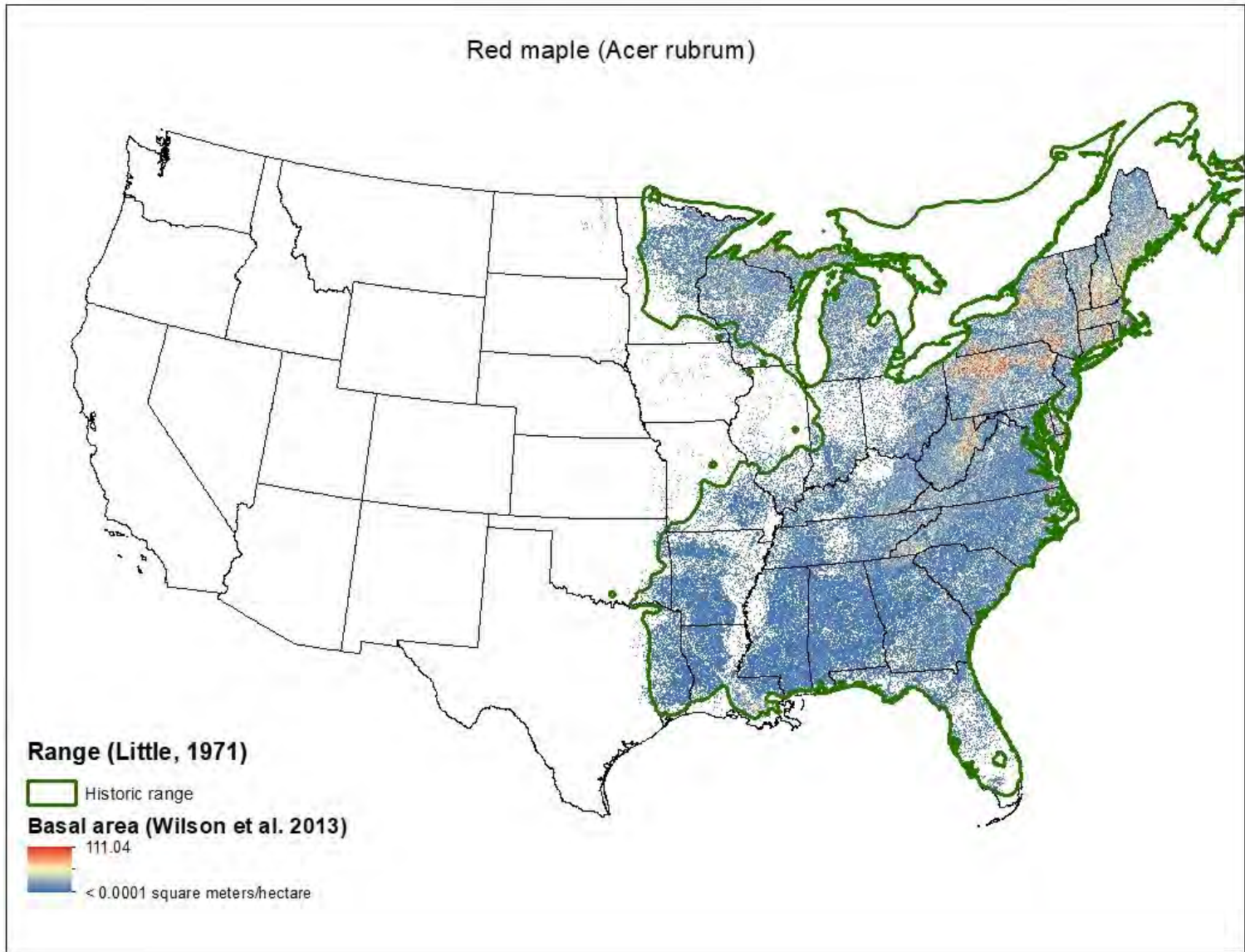
U.S. DISTRIBUTION OF 11 TREE SPECIES

1 **4B.1. DESCRIPTION**

2 This appendix presents maps of the distribution across the U.S. of 11 tree species for
3 which there are established exposure-response (E-R) functions, as described in Appendix 4A.
4 Historical ranges were based on Little (1971, 1976, 1977, and 1978) and basal area of each
5 species was taken from Wilson et. al (2013) raster data to show present range and estimated
6 density. Basal area is computed at the stand level as the sum of the basal area values for each
7 individual tree (in sq. ft.), which is summed across all of the basal area per tree in the
8 hectare. The map construction consists of tree species abundance, distribution, and basal area at
9 a 250-meter (m) pixel size for the contiguous United States (Wilson 2013).



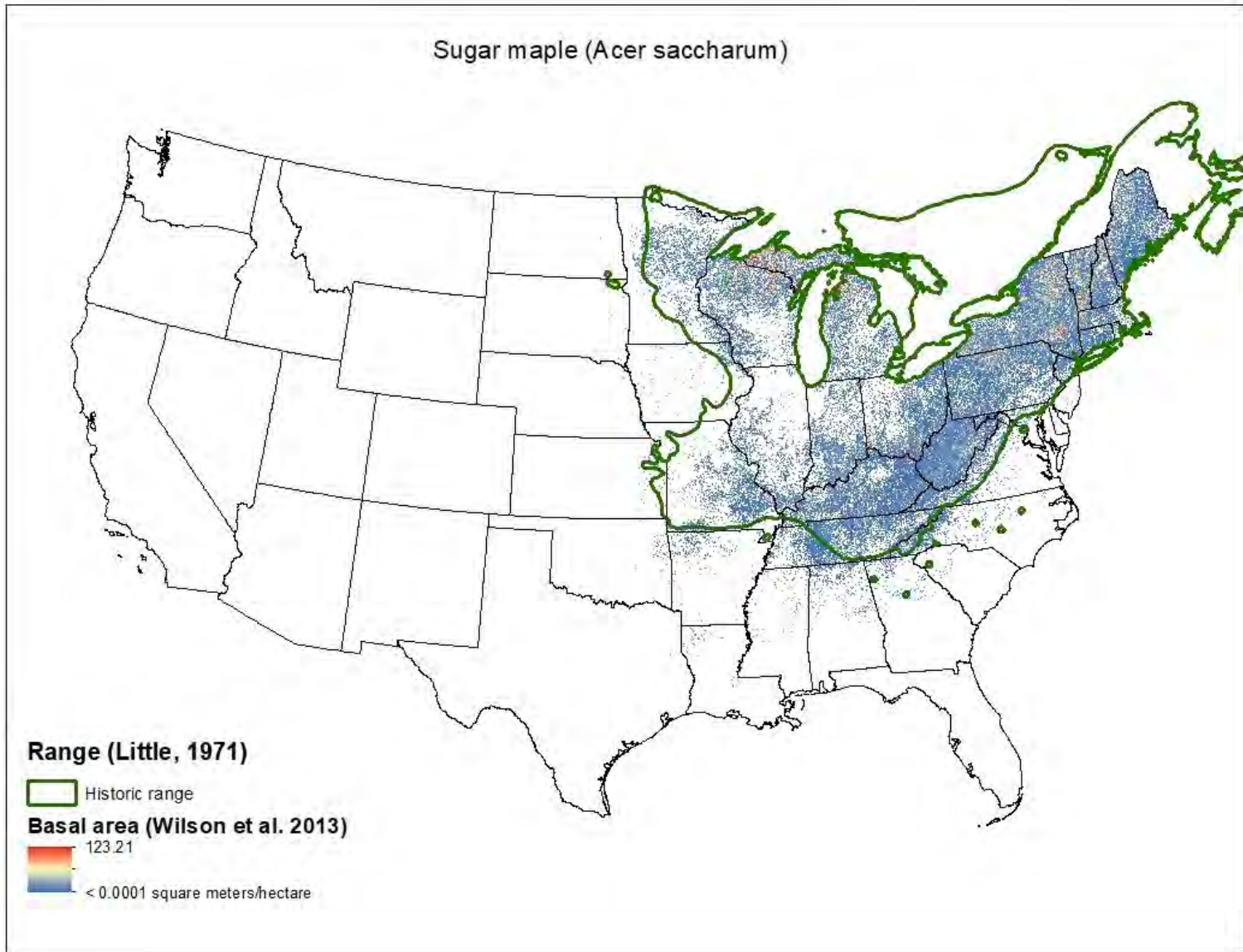
1
 2 **Table 4B-1. Distribution of quaking aspen (*Populus tremuloides*) in the continental U.S.**



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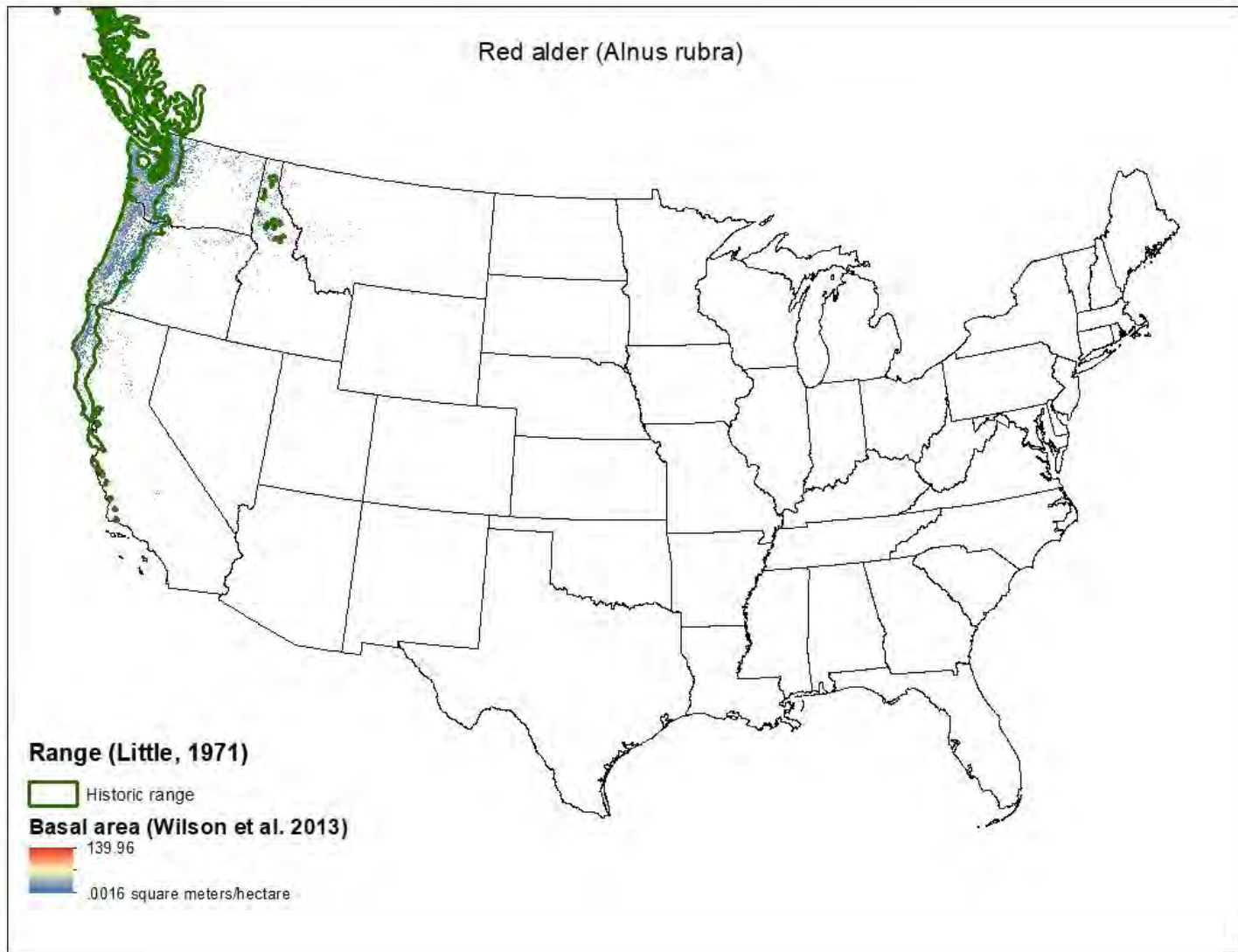
Table 4B-2. Distribution of red maple (*Acer rubrum*) in the continental U.S.



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Table 4B-3. Distribution of sugar maple (*Acer saccharum*) in the continental U.S.

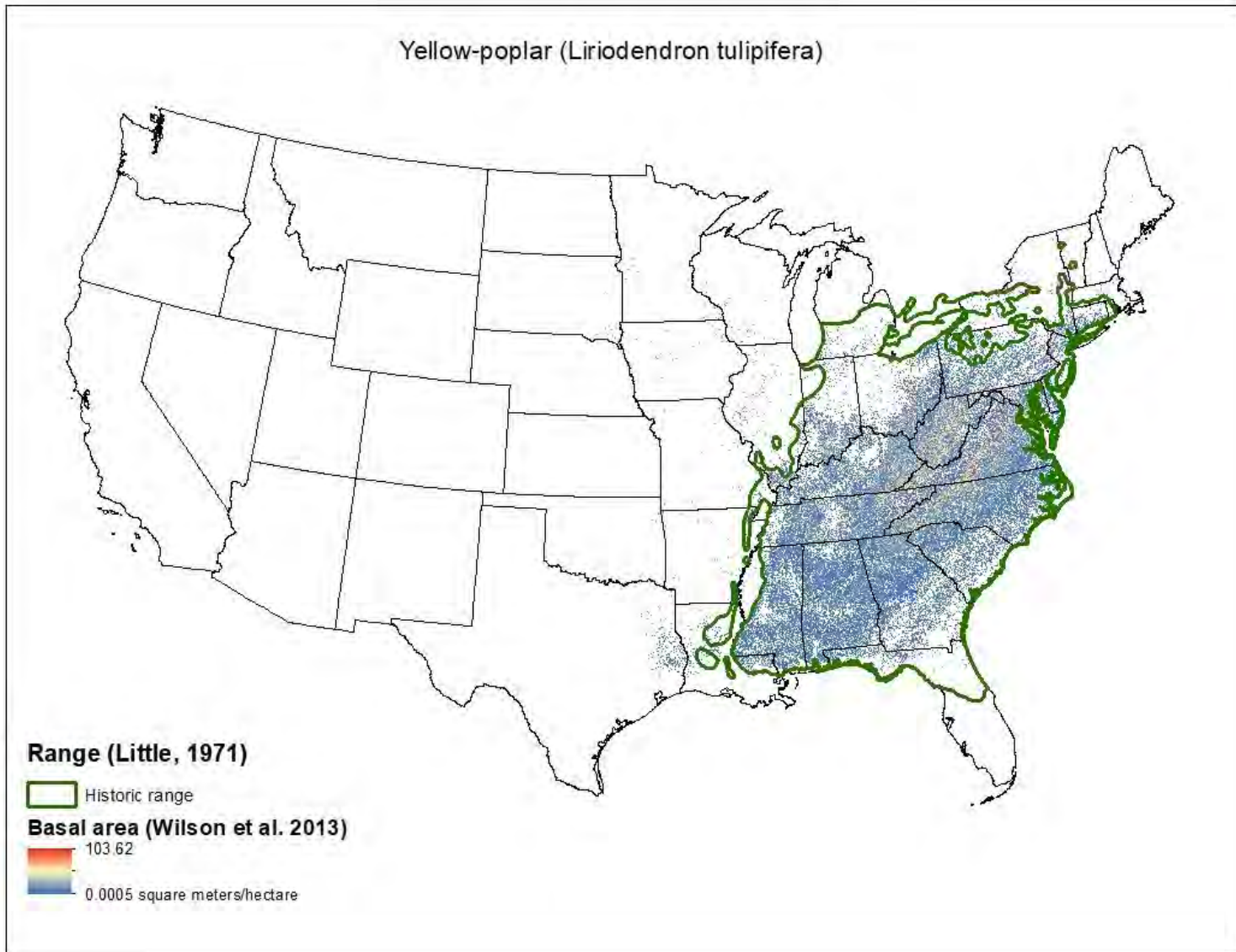
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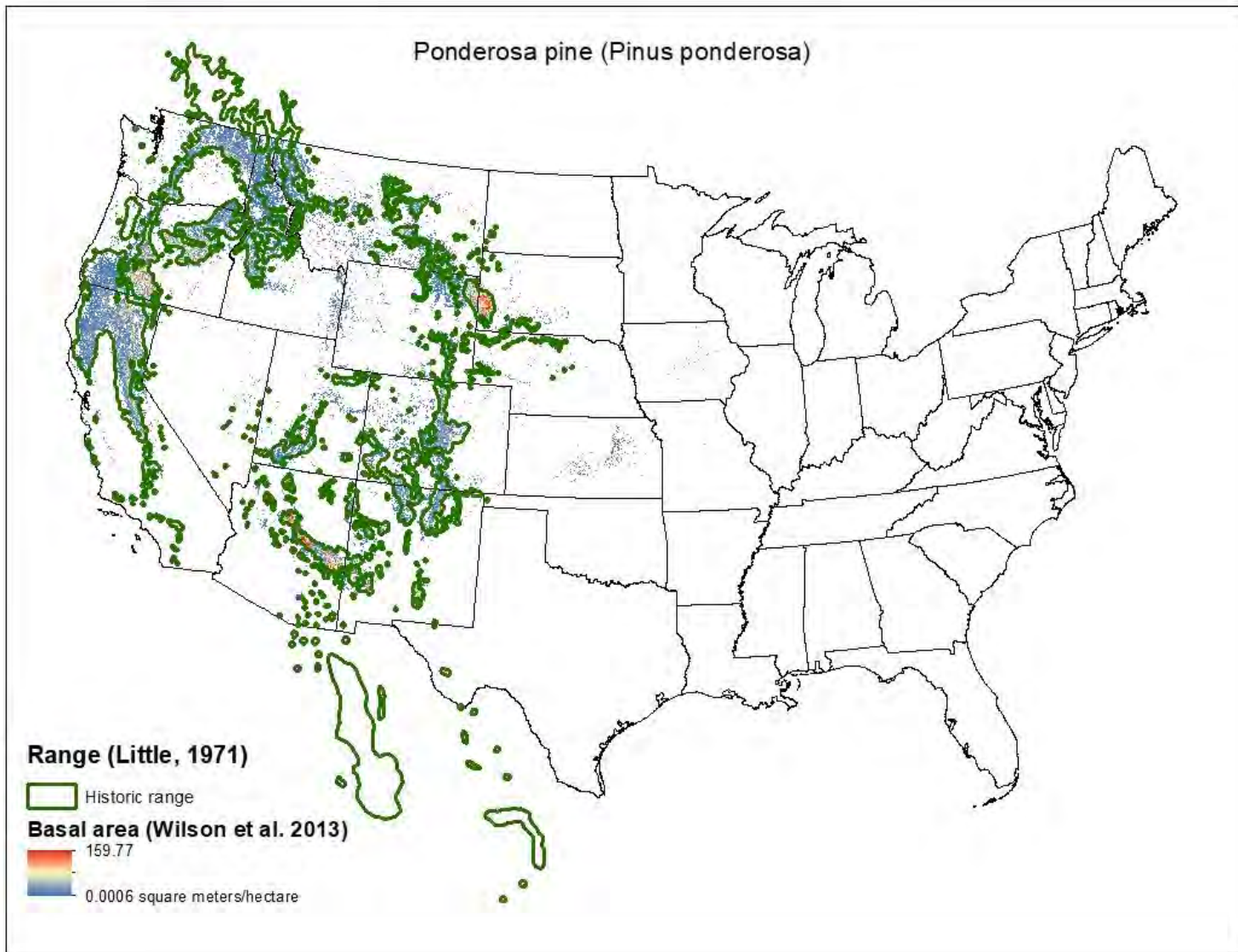
3

Table 4B-4. Distribution of red alder (*Alnus rubra*) in the continental U.S.



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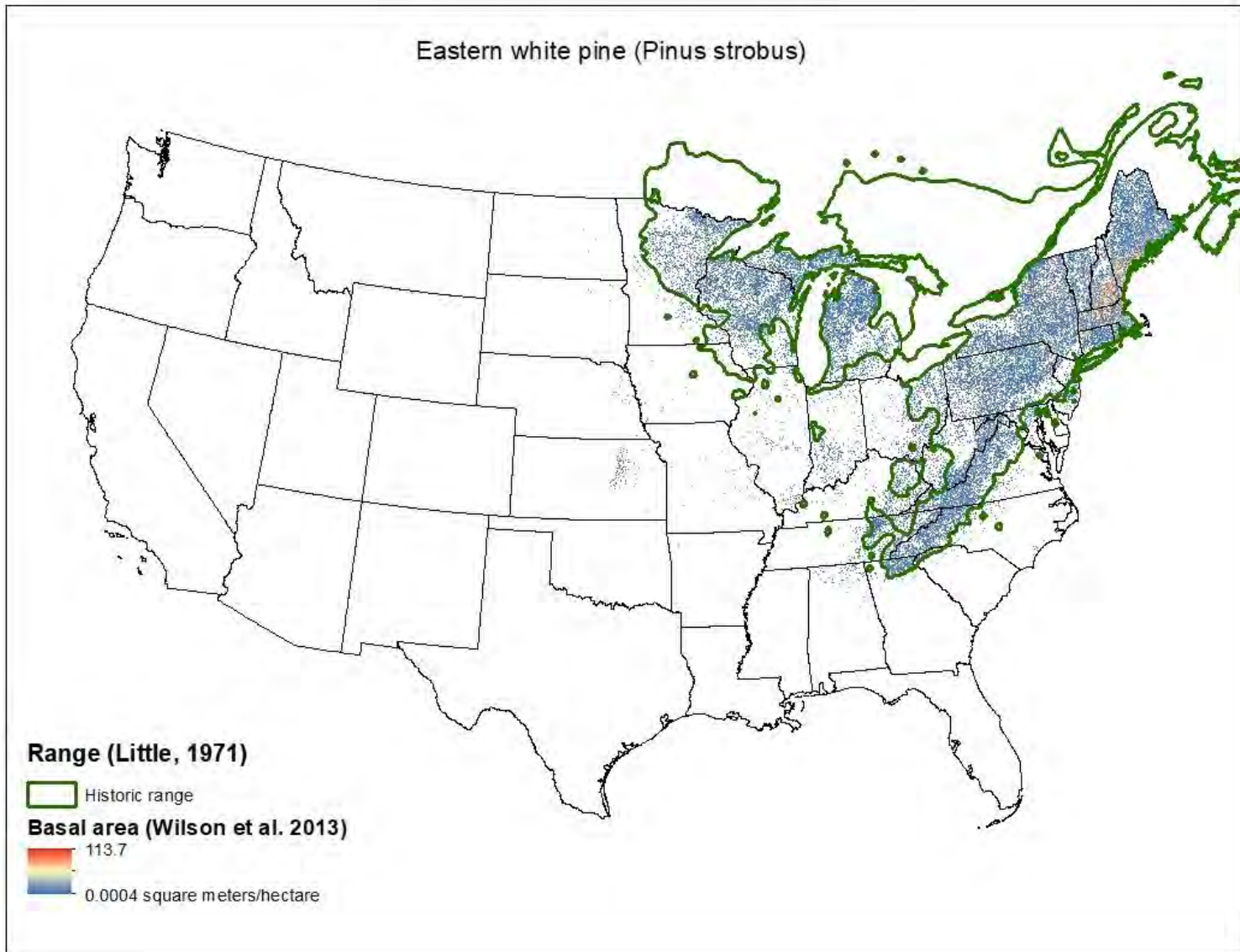
Table 4B-5. Distribution of tulip poplar (*Liriodendron tulipifera*) in the continental U.S.



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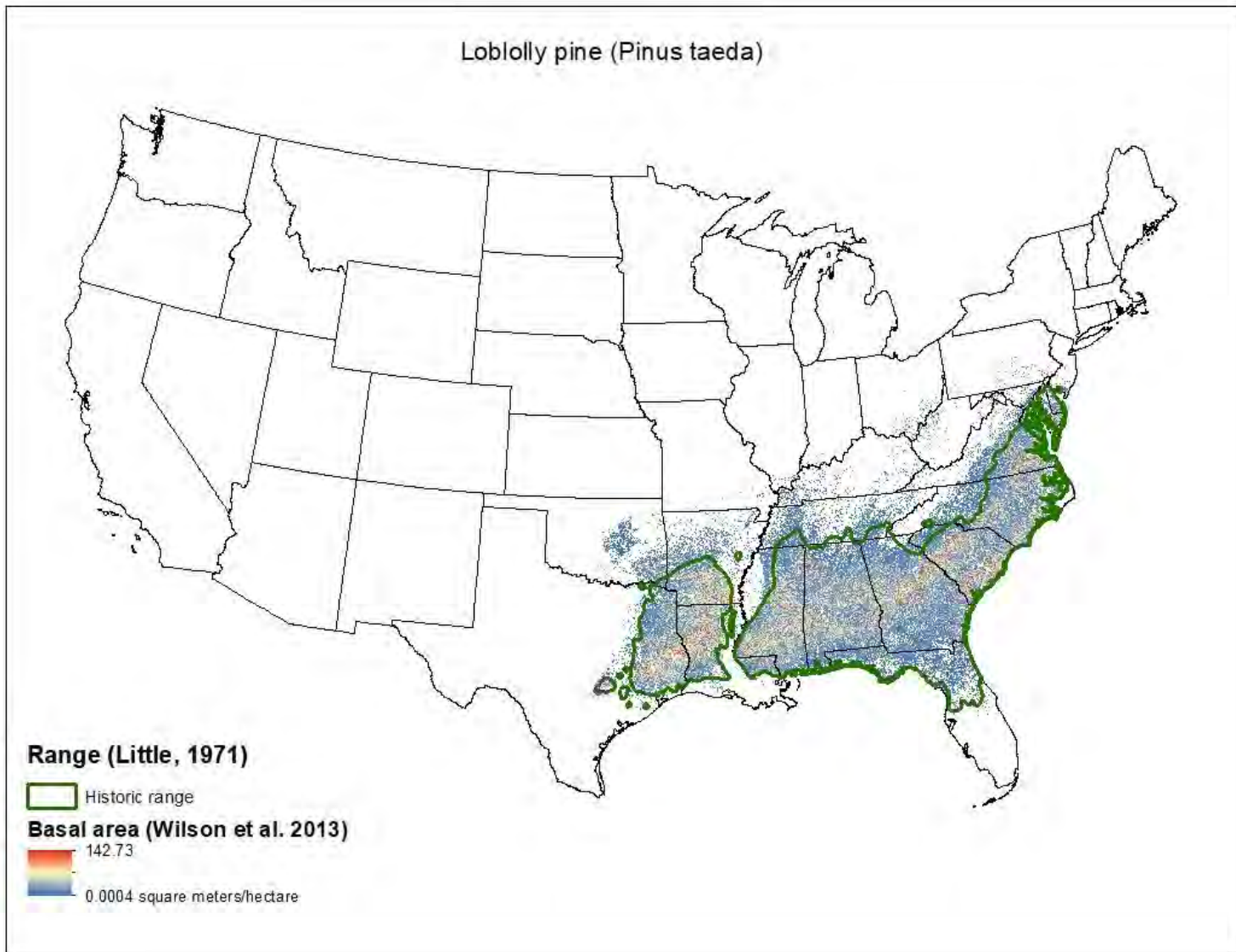
Table 4B-6. Distribution of ponderosa pine (*Pinus ponderosa*) in the continental U.S.



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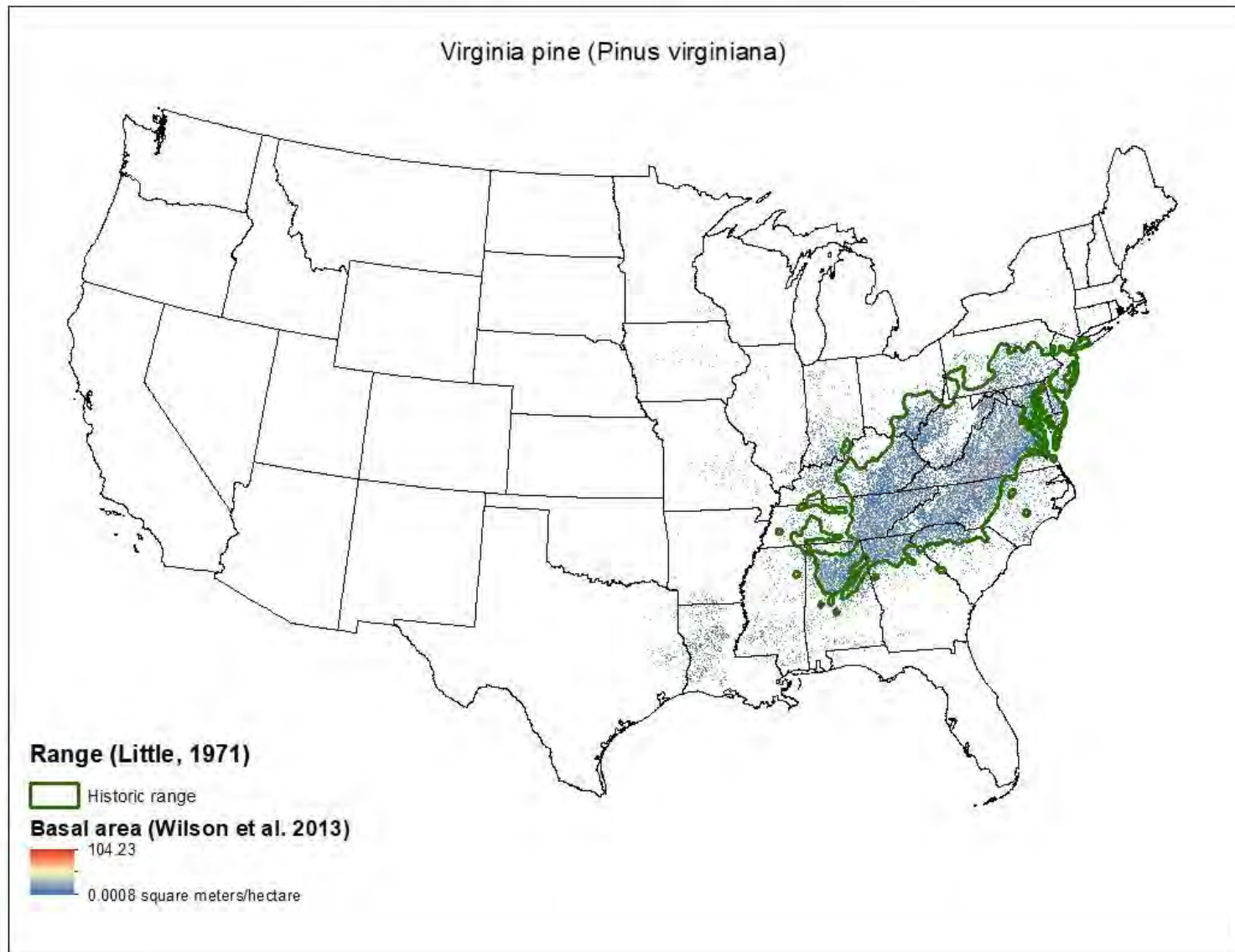
Table 4B-7. Distribution of eastern white pine (*Pinus strobus*) in the continental U.S.



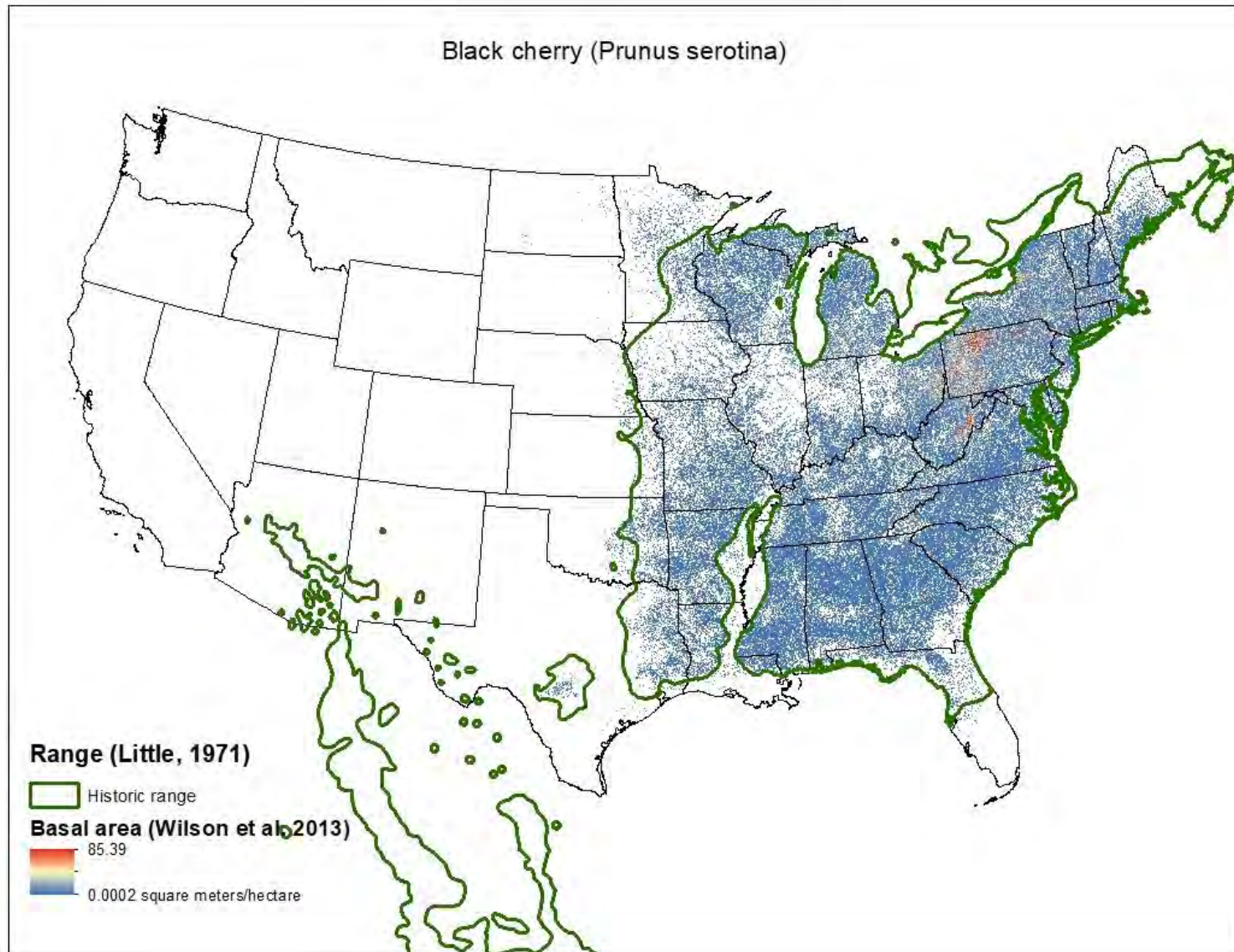
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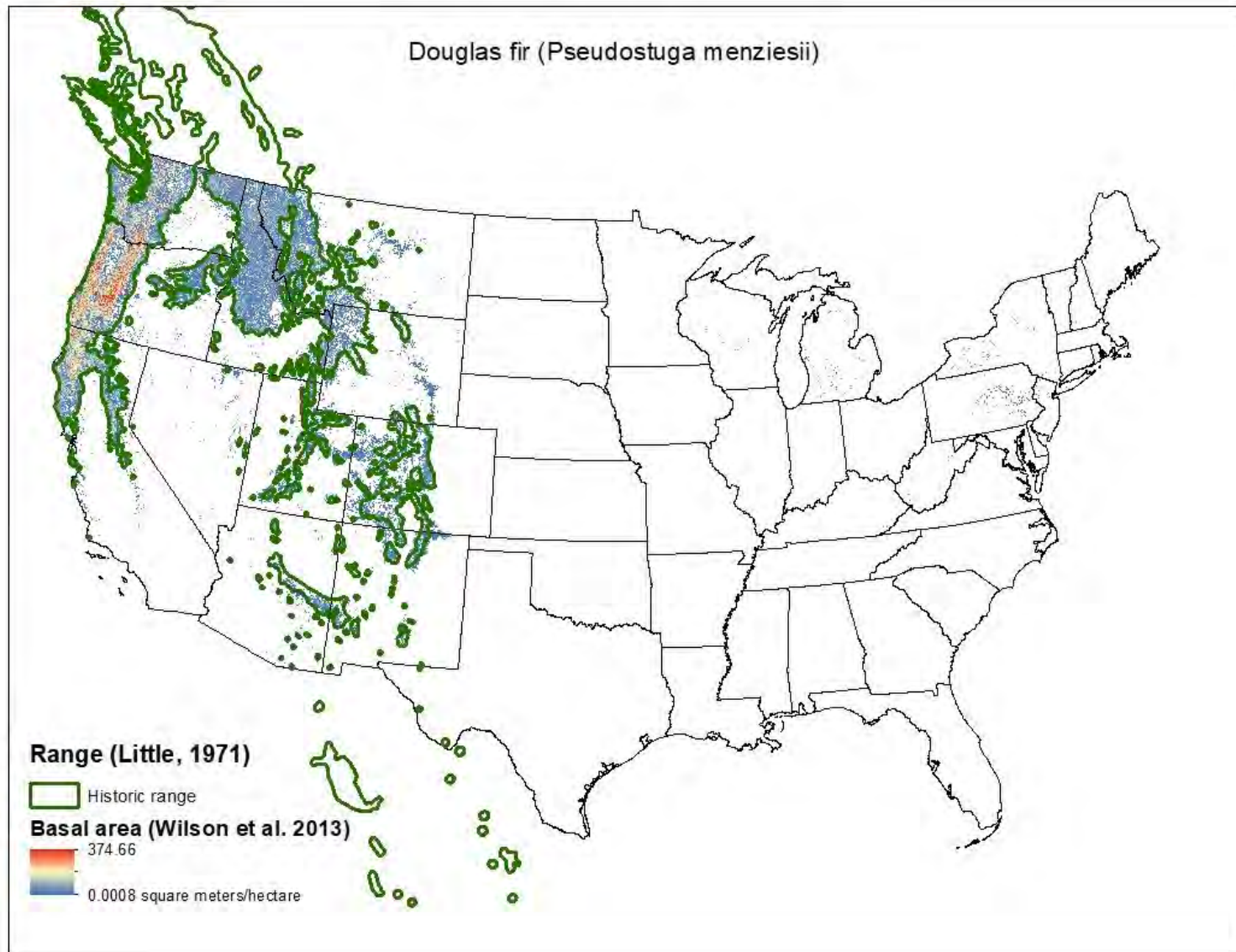
Table 4B-8. Distribution of loblolly pine (*Pinus taeda*) in the continental U.S.



1
 2 **Table 4B-9. Distribution of Virginia pine (*Pinus virginiana*) in the continental U.S.**



1
 2 **Table 4B-10. Distribution of black cherry (*Prunus serotina*) in the continental U.S.**



1

2

Table 4B-11. Distribution of Douglas fir (*Pseudotsuga menziesii*) in the continental U.S.

1 **REFERENCES**

2 Little, E.L., Jr., 1971, Atlas of United States trees, volume 1, conifers and important hardwoods:
3 U.S. Department of Agriculture Miscellaneous Publication 1146, 9 p., 200 maps.

4
5 Little, E.L., Jr., 1976, Atlas of United States trees, volume 3, minor Western hardwoods: U.S.
6 Department of Agriculture Miscellaneous Publication 1314, 13 p., 290 maps.

7
8 Little, E.L., Jr., 1977, Atlas of United States trees, volume 4, minor Eastern hardwoods: U.S.
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11 Little, E.L., Jr. 1978, Atlas of United States trees, volume 5, Florida: U.S. Department of
12 Agriculture Miscellaneous Publication 1361, 262 maps.

13
14 Wilson, Barry Tyler; Lister, Andrew J.; Riemann, Rachel I.; Griffith, Douglas M. 2013. Live tree
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16 USDA Forest Service, Rocky Mountain Research Station. [https://doi.org/10.2737/RDS-](https://doi.org/10.2737/RDS-2013-0013)
17 2013-0013

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APPENDIX 4C

**VISIBLE FOLIAR INJURY SCORES AT U.S. FOREST SERVICE
BIOSITES (2006-2010)**

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1 **4C.1 INTRODUCTION**

2 It has long been recognized that elevated ozone (O₃) can cause visible foliar injury in
3 some plants (ISA, Appendix 8, section 8.2). As discussed in the current and past ISAs as well as
4 past Air Quality Criteria Documents, the severity and extent of visible foliar injury can vary with
5 a variety of environmental variables (e.g., climatic variables as well as pollutant exposure) as
6 well as variation in genetic factors within the same plant population (ISA, Appendix 8, section
7 8.2). Visible foliar injury “occurs only when sensitive plants are exposed to elevated O₃
8 concentrations in a predisposing environment,” and “a major modifying factor is the amount of
9 soil moisture available to a plant during the year when assessed” (U.S. EPA, 2013 [2013 ISA], p.
10 9-39).

11 In recognition of the long-standing evidence regarding O₃ and visible foliar injury in
12 susceptible species, the U.S. Forest Service (USFS) and U.S. Park Service have used plant
13 species with this susceptibility in their biomonitoring programs. A number of publications have
14 focused on findings from biomonitoring surveys in the USFS-Forest Health Monitoring (FHM)
15 and Forest Inventory and Analyses (FIA) programs. From the mid 1990s through 2010, this
16 survey work included collecting information on the presence of visible foliar injury at the
17 biomonitoring sites (biosites). Data on visible foliar injury incidence and severity data were
18 collected each year at biosites in forested areas at states across the U.S. and summarized in terms
19 of a biosite index (BI). The BI is a measure of the severity of O₃-induced visible foliar injury
20 observed at each biosite.

21 Data from the multi-year USFS survey were used in analyses developed in the 2015 O₃
22 NAAQS review (80 FR 65292, October 26, 2015). These analyses utilized a dataset that had
23 been developed by merging biosite data collected as part of the USFS FHM/FIA Network during
24 the years 2006 through 2010, with NOAA soil moisture index values (as a surrogate for soil
25 moisture measurements) and W126 estimates of seasonal O₃ exposure for those sites based on
26 ambient air monitoring data for those 5 years (Smith and Murphy, 2015; U.S. EPA, 2014 [2014
27 WREA]) The resultant combined dataset included a BI score, soil moisture index value and a
28 W126 index estimate each for 5,284 records at locations in 37 states for 1 or more of the years in
29 the 5-year period from 2006-2010. This appendix brings forward key presentations developed
30 from the combined dataset for the 2015 O₃ review and also includes additional presentations of
31 key aspects of the dataset and the variables represented within it.

32 **4C.2 DATASET PREPARATION**

33 The combined dataset was developed from three datasets: (1) the national-scale
34 FIA/FHM dataset of BI scores, (2) the NOAA’s National Climatic Data Center national dataset
35 of monthly drought indices and (3) national surfaces of estimated seasonal W126 index

1 developed by the EPA for the WREA in the last O₃ NAAQS review and further analyzed in a
2 subsequent technical memo (Smith and Murphy, 2015). These individual datasets and how they
3 were used to create the combined dataset, are described below.

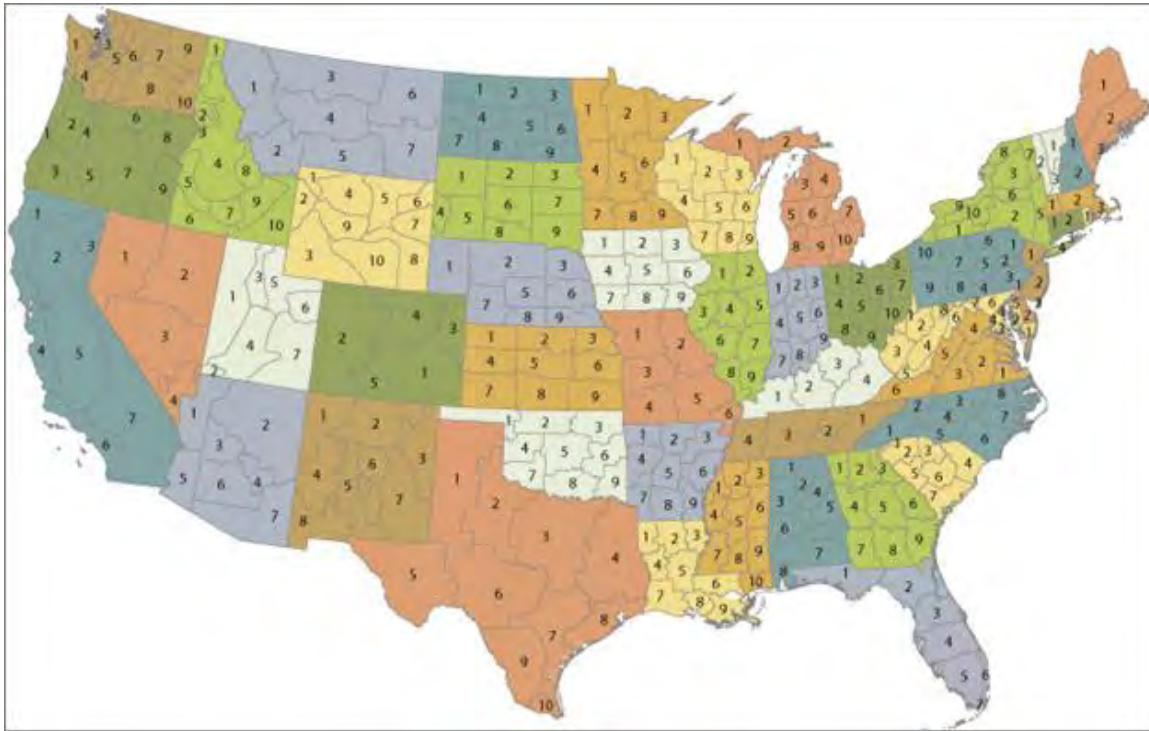
4 Biosite Index: The USFS O₃ biomonitoring program has developed a national-scale data
5 set focused on visible foliar injury and that includes BI scores at biosites in U.S. forests (Smith,
6 2012). The field methods, sampling procedures, and analytical techniques are consistent across
7 biosites and years. The BI is calculated from species-specific scores based on a combination of
8 the proportion of leaves affected on individual bioindicator plants and the severity of symptoms
9 on injured foliage using an established scale (Horsfall and Cowling, 1978; Smith, 2012). Each
10 site is sampled until 30 plants of at least two species have been evaluated (Smith et al., 2007).
11 The site BI is the average score for each species averaged across all species on the biosite
12 multiplied by 1,000 (Smith, 2012). The BI score ranges from zero to greater than 25, with a score
13 of zero indicating no presence of foliar injury symptoms and scores increasingly greater than
14 zero indicating increasingly greater severity of symptoms (Smith, 2012). Categories that have
15 been used in publications include little or very light injury (BI greater than 0 up to 5), light injury
16 (BI greater than 5 up to 15), moderate (BI greater than 15 up to 25) and heavy/severe (BI above
17 25) (Smith, 2012; Coulston et al., 2003).

18 The biosite data (BI scores) were obtained from the USFS for the years 2006 to 2010.
19 While including most states in the contiguous U.S., the data obtained did not include records for
20 most of the western states (Montana, Idaho, Wyoming, Nevada, Utah, Colorado, Arizona, New
21 Mexico, Oklahoma, and portions of Texas) because biosite data were not available for those
22 states during the 2006-2010 period (Smith et al., 2012).

23 Soil Moisture Index: The NOAA Palmer Z drought index is a monthly moisture anomaly
24 index that is derived from measurements such as precipitation and temperature. This index
25 represents the difference between monthly soil moisture and long-term average soil moisture
26 (Palmer, 1965). The Palmer Z index is derived each month for each of 344 climate region
27 divisions within the contiguous U.S. by the National Climatic Data Center (NCDC).¹ The index
28 values typically range from -4 to +4, with positive values representing more wetness than normal
29 and negative values representing more dryness than normal. For the combined dataset, index
30 values for April through August in the years 2006-2010 were obtained from the NCDC website
31 (NOAA, 2012). These monthly values were then averaged to create a single growing season
32 index for each year in each division. Moisture categories were then assigned consistent with

¹ There are 344 climate divisions in the continental U.S. For each climate division, monthly station temperature and precipitation values are computed from the daily observations as described on the website for the National Climatic Data Center of the U.S. National Atmospheric and Oceanic Administration: <https://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-divisions.php>

1 NOAA’s Palmer Z drought index, with index values less than -1.25 identified as “dry”, values
2 greater than or equal to 1 identified as “wet”, and index values between -1.25 and 1 identified as
3 “normal.” Values beyond the range from -2.75 to +3.5 could be interpreted as extreme drought
4 and extremely moist, respectively (NCDC, 2012c). The NCDC climate divisions with Palmer Z
5 data are shown in Figure 4C-1.



6
7 **Figure 4C-1. Climate divisions for which there are Palmer Z soil moisture index values.**

8 W126 Index Estimates: Estimates of seasonal W126 exposure index for the years 2006
9 through 2010 were developed for 12 kilometer (km) by 12 km grid cells in a national-scale
10 spatial surface. The estimates at this scale were derived from applying a spatial interpolation
11 technique to annual W126 values derived from O₃ measurements at ambient air monitoring
12 locations. Specifically, the Voronoi Neighbor Averaging (VNA) spatial interpolation technique
13 was applied to the monitor-location W126 index values to derive an W126 index estimates for
14 each grid cell (U.S. EPA, 2014, Appendix 4A).²

15 Combined Dataset: To create the dataset that relates the grid cells with W126 index
16 estimates to grid cells with BI scores, the EPA provided a file with the national-scale surface of
17 grid cells (a “shape” file) to USFS staff, who assigned the BI scores (with sampling year
18 specified) to grid cells for all but three states. Having this step performed by the USFS ensured

²The VNA application step used to estimate W126 indices at the centroid of every 12 km x 12 km grid cell, rather than only at each monitor location (described in Appendix 4A of the WREA), can result in a lowering of the highest values in each region (80 FR 65374-65375; October 26, 2015).

1 that the precise and accurate geographic coordinates for each biosite were used in this step,
2 which allowed the most accurate matching of Palmer Z and W126 index values as possible with
3 these datasets.³ For three states (California, Oregon, and Washington) the EPA downloaded
4 biosite indices from the public website and assigned them to the grid cells in which the biosite
5 was located based on the publicly available geographic coordinates.⁴ The EPA overlaid the
6 Palmer Z dataset for each year on the national surface of W126 index estimates for that year to
7 assign a Palmer Z index to each grid cell in each year’s national surface. The completed dataset
8 (Smith and Murphy, 2015, Appendix) includes the following variables: identifier, year, W126
9 index, BI score, Palmer Z index, state and soil moisture category (dry, wet, normal)⁵.

10 **4C.3 DATASET CHARACTERISTICS**

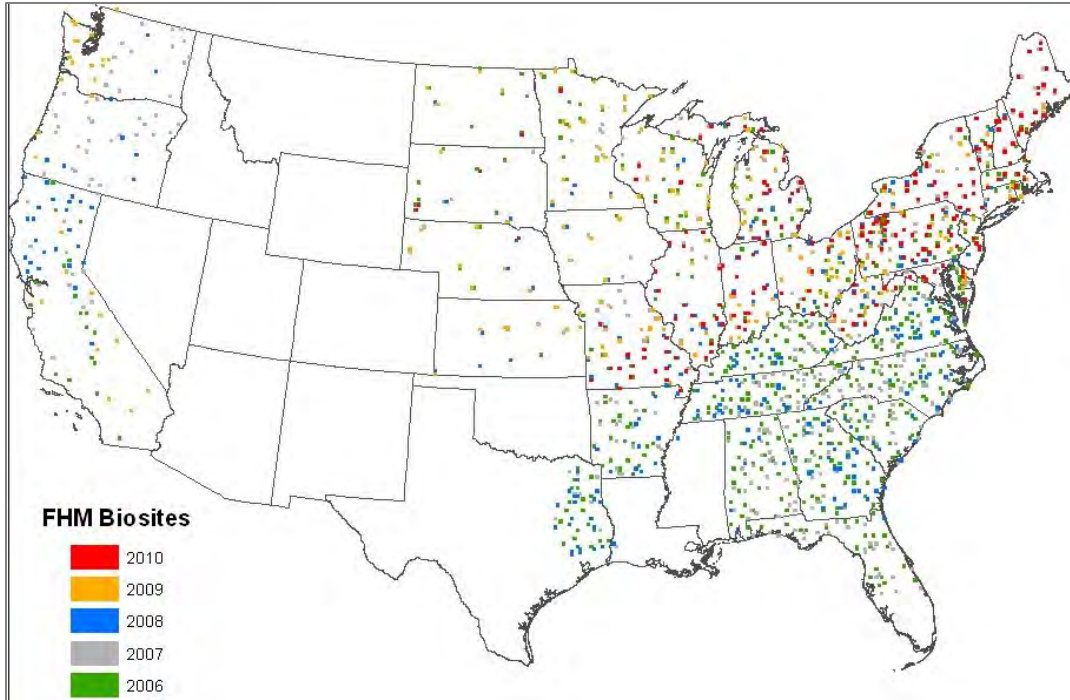
11 The dataset for the analyses included 5,284 biosite records distributed across the 37
12 different states and the five years from 2006 – 2010 (Smith and Murphy, 2015, Appendix).
13 Figure 4C-2, reprinted from 2014 WREA, indicates the distribution of sites across the
14 continental U.S. Table 4C-2 summarizes the biosite index values for each year. The “Damage”
15 categories used follow the USFS risk categories with the exception of including a separate
16 category for a biosite index of zero (Smith, 2008, 2012). The zero category was defined and used
17 as a measure of the presence or absence of any level of visible foliar injury. Across all of the
18 sites, over 81 percent of the observations recorded no foliar injury. This percentage was similar
19 across all of the years, with a low value of 78 percent and a high value of 85 percent. Across the
20 5,284 records in the dataset, only 998 had BI scores greater than zero.

21

³ This step was taken because the publicly available USFS BI dataset includes location coordinates that have been slightly altered to avoid specifying the true biosite location for privacy considerations of some property owners.

⁴ As a result, there is a potential for the biosites for these states to be matched with the W126 index estimate for an adjacent grid cell rather than the one in which the biosite is truly located.

⁵ As described earlier in the section on “Soil Moisture Index,” all index values less than -1.25 were categorized as “dry” and all index values greater than or equal to 1 were categorized as “wet.”



1
2 **Figure 4C-2. USFS biomonitoring sites for visible foliar injury (“Biosites”).**

3
4 **Table 4C-1. Summary of biosite index scores for 2006 to 2010 USFS biomonitoring sites.**

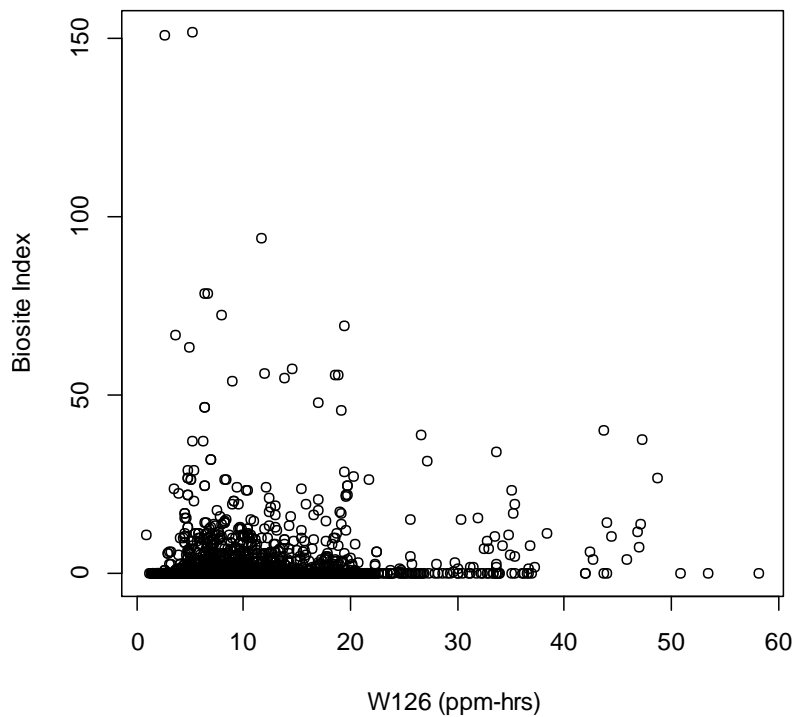
Biosite Index	Damage	2006	2007	2008	2009	2010	Total
0	None	744	769	796	902	1,075	4,286
< 5	Very Light	139	131	98	135	183	686
5 to 15	Light	41	29	29	61	65	225
15 to 25	Moderate	15	6	8	6	12	47
> 25	Heavy	12	4	4	8	12	40
Total		951	939	935	1,112	1,347	5,284

5
6 **4C.4 RELATIONSHIPS OF BIOSITE INDEX SCORES WITH W126**
7 **ESTIMATES AND SOIL MOISTURE CATEGORIES**

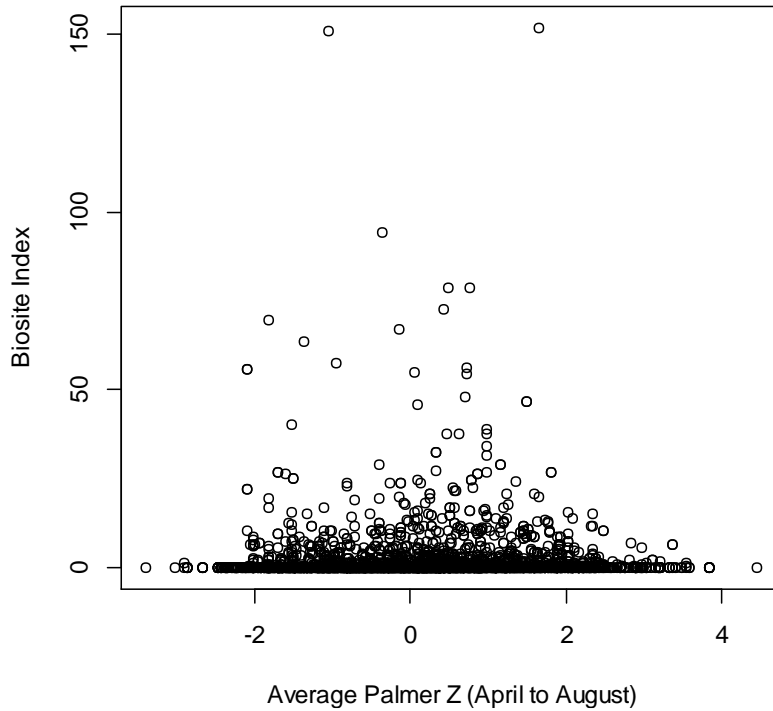
8 **4C.4.1 Relationships Examined in Full Dataset**

9 Scatterplots of the full dataset show no clear relationship between O₃ and biosite index
10 (Figure 4C-3), as well as no clear relationship between O₃ and the Palmer Z drought index,
11 measured as an average value of the months from April to August (Figure 4C-4). The lack of a
12 clear relationship is partly due to the high number of observations with no foliar injury (see
13 Table 4C-1 above and also the distribution of records by soil moisture category and W126
14 summarized in section 4C.4.2 below) and may also reflect, in part, differing spatial resolutions of

1 the O₃ exposure surface, NCDC climate divisions, and the biosites. To investigate the strength of
2 any relationship in light of the high percentage of zero values, a censored regression was
3 conducted using a threshold of zero (i.e., including only the non-zero observations). The results
4 of the regression (Table 4C-2) are consistent with the evaluation of the evidence in the ISA (and
5 prior ISA and AQCDs), indicating a significant relationship between foliar injury and both O₃
6 and moisture (as measured by Palmer Z), and also a significant interaction between O₃ and
7 moisture. The censored regression does not provide a “goodness of fit” statistic as easily
8 interpreted as the r-squared value associated with a standard regression, so the results are more
9 difficult to interpret. Thus, while higher O₃ corresponds to higher BI score, the parameters
10 describing such a relationship in predictive quantitative terms are unresolved.
11



12
13 **Figure 4C-3. Scatter plot of biosite index score *versus* W126 index (ppm-hrs).**



1
 2 **Figure 4C-4. Scatter plot of biosite index score versus Palmer Z (April to August).**

3
 4 **Table 4C-2. Statistics from censored regression.**

Coefficient	Intercept Estimate	Standard Error	t-value	p
Intercept	-22.5967	0.8934	-25.293	< 0.0001
W126	0.7307	0.0613	11.919	<0.0001
Palmer Z (Apr-Aug)	1.8357	0.4850	3.785	0.0002
W126: Palmer Z	0.1357	0.0437	3.104	0.0019
	Marginal Effect			
W126	0.1178	0.0099	11.918	<0.0001
Palmer Z (Apr-Aug)	0.2960	0.0777	3.812	0.0001
W126: Palmer Z	0.0219	0.0070	3.093	0.0020

5
 6 An exploration (in the 2014 WREA) of the use of regression coefficients to calculate
 7 estimated biosite index values did not accurately predict the observed values, likely due in part to
 8 the large number of non-injury observations. It is also of note that the W126 index bin with the
 9 largest percentage of records of each category of BI score (e.g., all, zero, above zero, above 5,
 10 above 15) is that for the lowest W126 index values (0).

1 **Table 4C-3. Cumulative percentage of records with specified BI score.**

< 7 ppm-hrs	>7 -9 ppm-hrs	>9 – 11 ppm-hrs	>11 -13 ppm-hrs	>13 -15 ppm-hrs	>15 -17 ppm-hrs	>17 - 19 ppm-hrs	>19 - 25 ppm-hrs	>25 ppm-hrs
<i>Cumulative Percentage of Records (percent of records in bin plus all bins to its left)</i>								
Of All Records								
42%	59%	73%	82%	88%	92%	96%	98%	100%
Of Records with BI=0 (total in dataset =4286)								
43%	60%	73%	83%	88%	93%	97%	99%	100%
Of Records with BI>0 (total in dataset =998)								
37%	53%	69%	78%	84%	87%	93%	96%	100%
Of Records with BI>5 (total in dataset =310)								
36%	49%	64%	73%	78%	82%	88%	91%	100%
Of Records with BI > 15 (total in dataset =85)								
33%	45%	49%	59%	64%	69%	78%	86%	100%

2

3 **4C.4.2 Examination of Relationships in Dataset Stratified by Soil Moisture Category**

4 The following tables and figures describe the data in this dataset with a focus on
 5 consideration of potential trends with W126 index for the different soil moisture categories. The
 6 W126 index estimates were rounded to integer values for consistency with Appendix 4D
 7 analyses and associated clarity in binning of the values.⁶ Additionally, consistent with USFS
 8 publications (e.g., Campbell et al., 2007), the BI scores⁷ are rounded to one decimal place. Table
 9 4C-4 presents the counts of records in total and stratified by soil moisture category and W126
 10 index bin. Table 4C-5 presents average BI scores by soil moisture category and W126 bin, and
 11 Table 4C-6 presents the fraction of records with BI scores of differing severity levels
 12 (corresponding to the USFS severity scheme), in the full dataset and also in the subsets by soil
 13 moisture category.

14 The distribution of records across W126 bins are presented in Table 4C-4 and Figure 4C-
 15 5, and the distribution of scores per bin is presented in Figure 4C-6 through Figure 4C-11. These
 16 figures show that even the lowest W126 index bin (for estimates below 7 ppm-hrs) includes
 17 scores well above 5, and several above 15. Further, zero scores comprise more than half the dry
 18 and normal soil category record scores in every bin, including the highest bin (>25 ppm hrs), as

⁶ The presentations here are not precise statistical analyses. Rather, they are intended to generally inform conclusions regarding ability of available datasets to discern air quality conditions contributing to visible foliar injury occurrences of potential concern. In this light, binning was used to explore the potential for clear differences in BI scores among sites with differing W126 estimates across the range of interest while also maintaining reasonable sample sizes.

⁷ Two records with estimated W126 index below 7 ppm-hrs and BI scores just over 150 are omitted from presentations in this section as the next highest BI score in this dataset for any W126 index was below 100.

1 seen by the median lines merged with the zero line in Figure 4C-6 and Figure 4C-8. This is also
2 the case for all but the two highest bins for the wet soil moisture category records, which,
3 however, contain just a total of 9 records, limiting the extent to which they provide a basis for
4 interpretation of patterns across W126 bins. The wet soil moisture records have quite limited
5 sample sizes for the higher W126 index bins, e.g., the number of samples in bins for W126 index
6 estimates above 13 ppm-hrs represent no more than 1 percent of the total number of wet soil
7 moisture records (Figure 4C-10).

8 Focusing on the distribution of scores for records in the normal soil moisture category, it
9 can be seen that scores are noticeably increased in the highest W126 bin, index estimates greater
10 than 25 ppm-hrs, over those for the lower bins (Figure 4C-6 and Figure 4C-7). This is also for
11 the average BI scores per bin, where the highest W126 bin (>25 ppm-hrs) has an average BI
12 appreciably higher than the others (Table 4C-5). The average BI in this highest bin is 7.9 *versus*
13 averages of 1.6 (for W126 >19 to 25 ppm-hrs) and 2.3 (for W126 >17 to 19 ppm-hrs) in the next
14 lower bins and varying from 0.8 to 1.2 in all the others. Among the records with nonzero scores,
15 the highest average BI is also in the highest W126 index bin (>25 ppm-hrs); in this case the BI is
16 approximately 15, more than double the next highest average BI scores for any of the other
17 W126 index bins (for which no other trend is exhibited). The incidence of records with BI scores
18 categorized by the USFS as “moderate” or “severe” injury (BI score above 15) is also greatest in
19 the bin for the highest W126 index estimates (> 25 ppm-hrs), with 20% of those records in this
20 bin having such a BI score compared to only 2 to 4% of the records in each of the lower bins. A
21 similar pattern holds for the records with BI scores above 5, while there is much more variability
22 across the bins for records with any nonzero score (Table 4C-6).

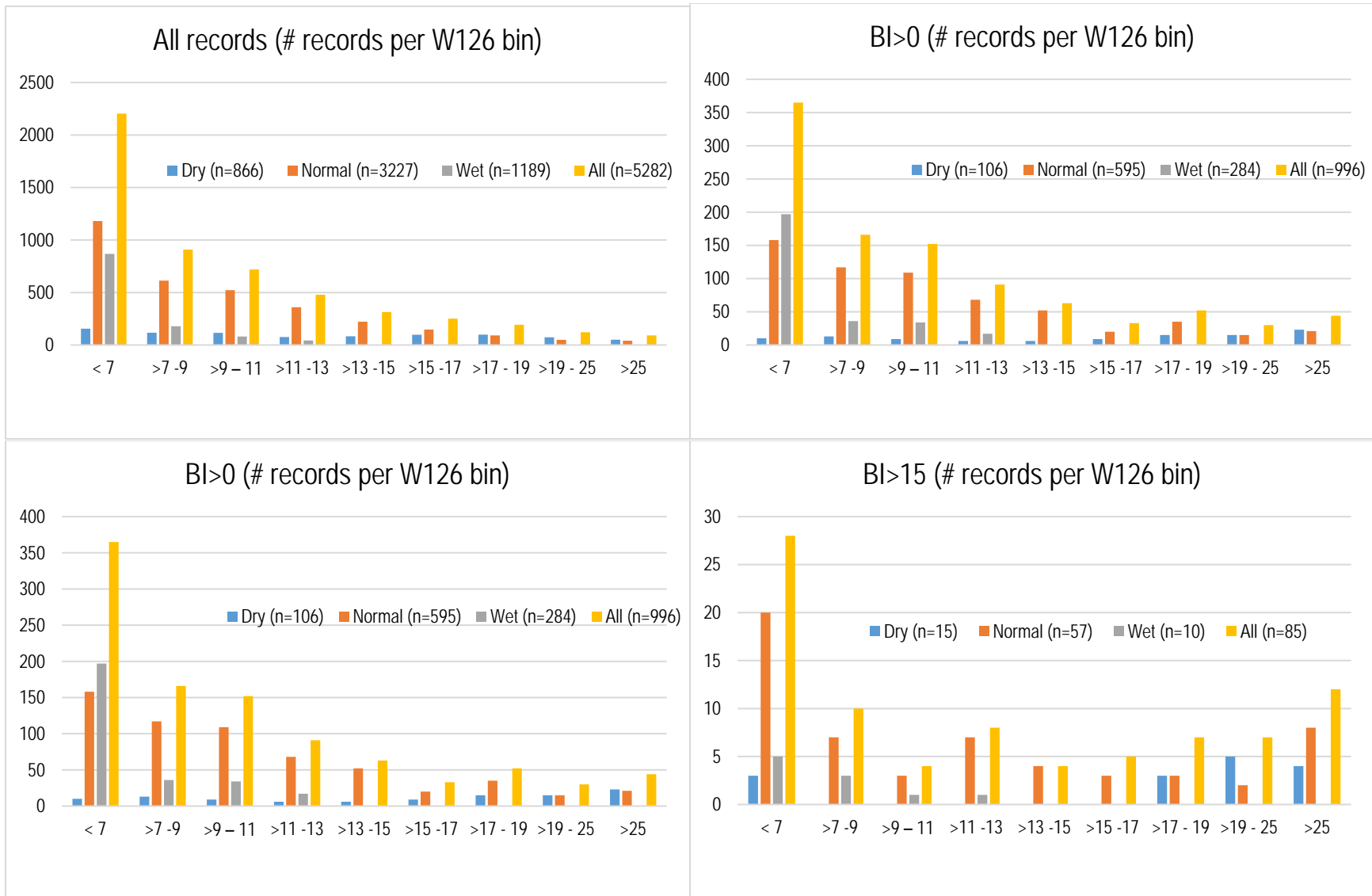
23 With regard to the dry soil moisture category, there is a suggestion of an increased
24 incidence of the highest severity scores in the highest two W126 bins. For example, the
25 proportion of dry soil moisture category records with BI scores categorized by the USFS as
26 “moderate” or “severe” injury (BI score above 15), is 7 and 8% in the bin for the two highest
27 W126 index estimates (>19 to 25 and > 25 ppm-hrs, respectively), compared to 0 to 3% in each
28 of the lower bins. It is noteworthy, however, that the percentages of 7 and 8% reflect no more
29 than 4 or 5 individual records with this severity score.

30 As noted above, sample size for the wet soil moisture category is particularly limited for
31 the W126 index bins above 13 ppm-hrs. In the lower W126 bins, the proportion of such records
32 with BI scores above 15 varies from 1 to 2%. For BI scores above 5 or above 0, there is a
33 suggestion of an increased incidence in the relatively higher *versus* lower W126 index bins,
34 although it is not known if the significant reduction in sample size that also occurs in comparing
35 across these bins (see Table 4C-4) and associated variability is playing a role (Table 4C-6).

1 **Table 4C-4. Number of biosite records in different W126 index bins.**

	< 7 ppm-hrs	>7 -9 ppm-hrs	>9 – 11 ppm-hrs	>11 -13 ppm-hrs	>13 -15 ppm-hrs	>15 -17 ppm-hrs	>17 - 19 ppm-hrs	>19 - 25 ppm-hrs	>25 ppm-hrs
All Records (n=5282^A)									
Dry (n=866)	155	117	116	76	83	97	99	73	50
Normal (n=3227)	1181	613	522	360	222	147	92	49	41
Wet (n=1189)	868	179	81	43	9 ^B	7 ^B	2 ^B	0 ^B	0 ^B
All	2204	909	719	479	314	251	193	122	91
Records with BI > 15 (total in dataset =85)									
Dry	3	0	0	0	0	0	3	5	4
Normal	20	7	3	7	4	3	3	2	8
Wet	5	3	1	1	0 ^B	2 ^B	1 ^B	0 ^B	0 ^B
All	28	10	4	8	4	5	7	7	12
Records with BI>5 (total in dataset =310)									
Dry	6	3	5	3	4	1	5	8	10
Normal	56	30	28	18	11	8	12	3	17
Wet	49	9	13	6	1 ^B	2 ^B	2 ^B	0 ^B	0 ^B
All	111	42	46	27	16	11	19	11	27
Records with BI>0 (total in dataset =998)									
Dry	10	13	9	6	6	9	15	15	23
Normal	158	117	109	68	52	20	35	15	21
Wet	197	36	34	17	5 ^B	4 ^B	2 ^B	0 ^B	0 ^B
All	365	166	152	91	63	33	52	30	44
Records with BI=0 (total in dataset =4286)									
Dry	145	104	107	70	77	88	84	58	27
Normal	1023	496	413	292	170	127	57	34	20
Wet	671	143	47	26	4 ^B	3 ^B	0 ^B	0 ^B	0 ^B
All	1839	743	567	388	251	218	141	92	47
^A As noted in the beginning of section 4C.1.2, this count reflects the omission of two outlier values.									
^B Sample size for this W126 bin is below 1% of all samples assigned this soil moisture category.									

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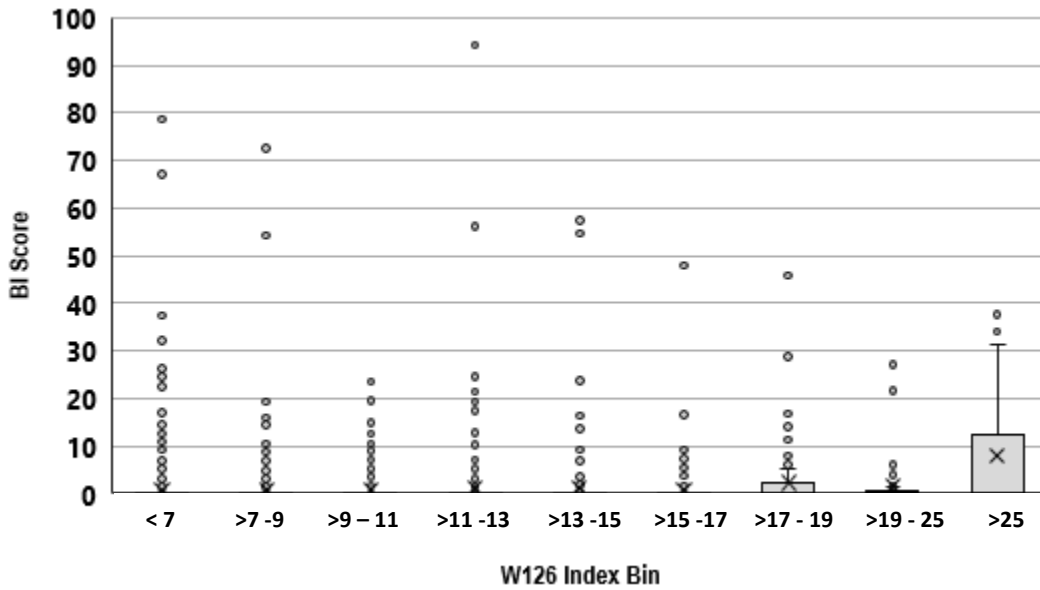


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3 **Figure 4C-5. Distribution of biosite records by W126 bin and soil moisture type.**

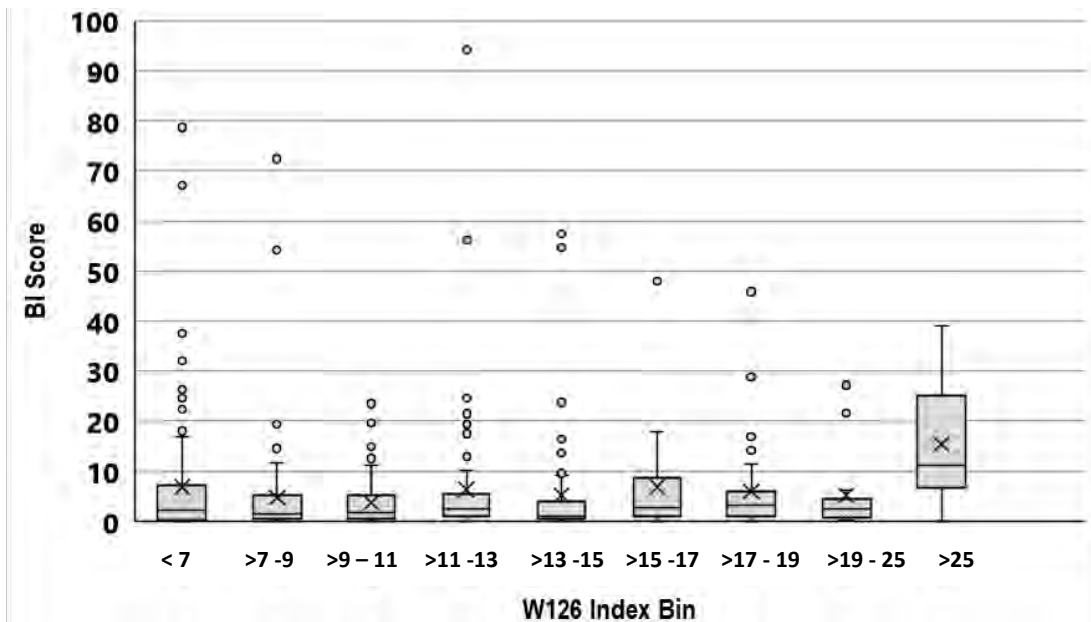
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Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

Figure 4C-6. Distribution of BI scores (including zeros) at USFS biosites (normal soil moisture) grouped by W126 index values.

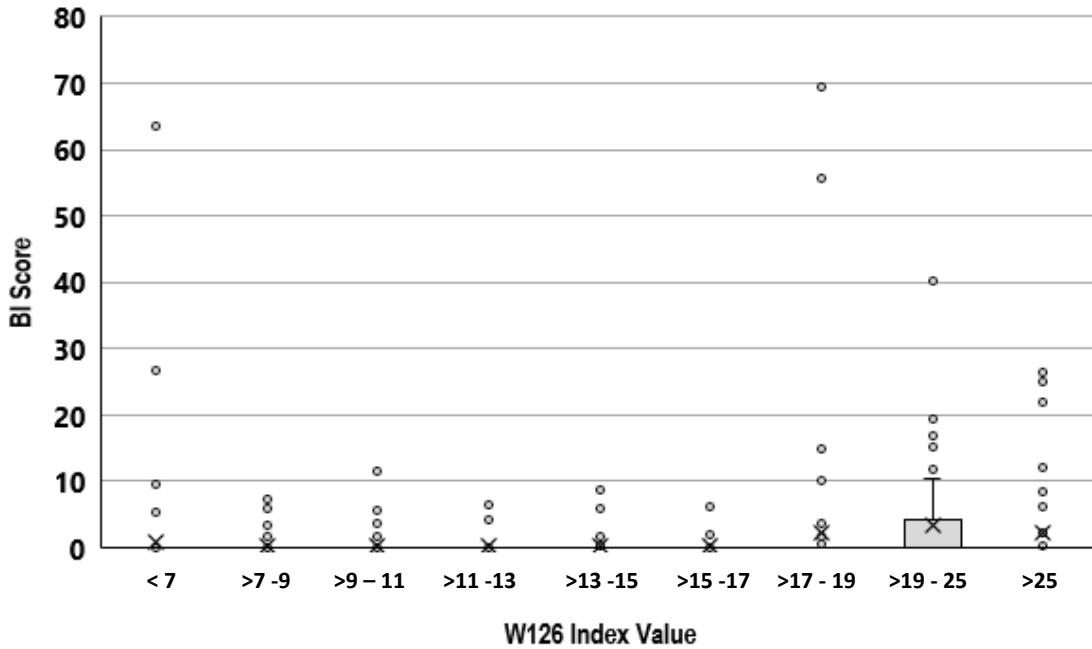
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Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

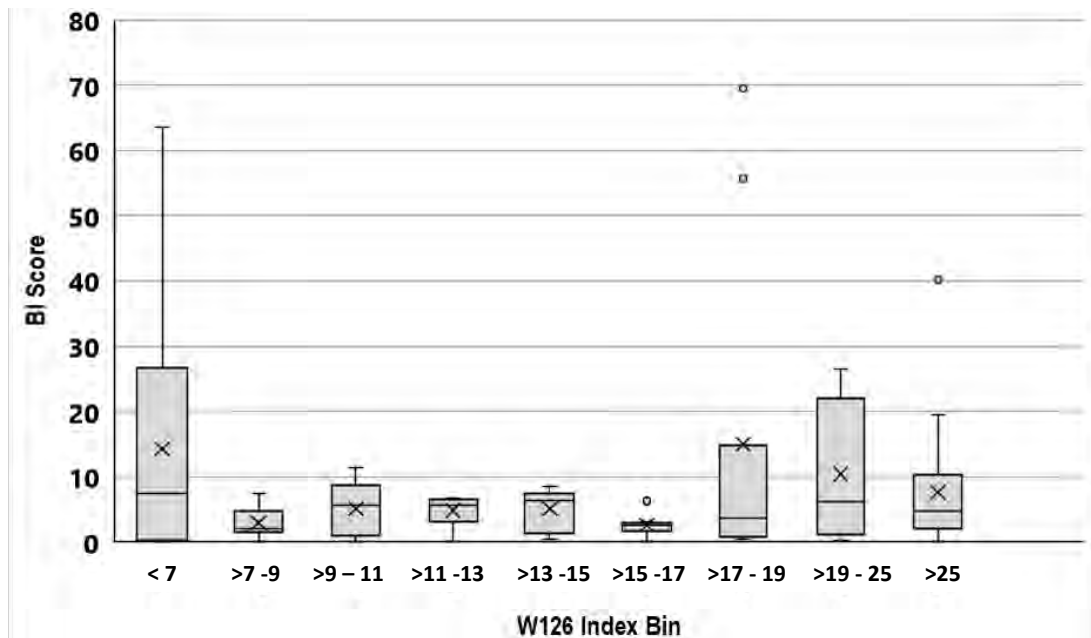
Figure 4C-7. Distribution of nonzero BI scores at USFS biosites (normal soil moisture) grouped by W126 index values.

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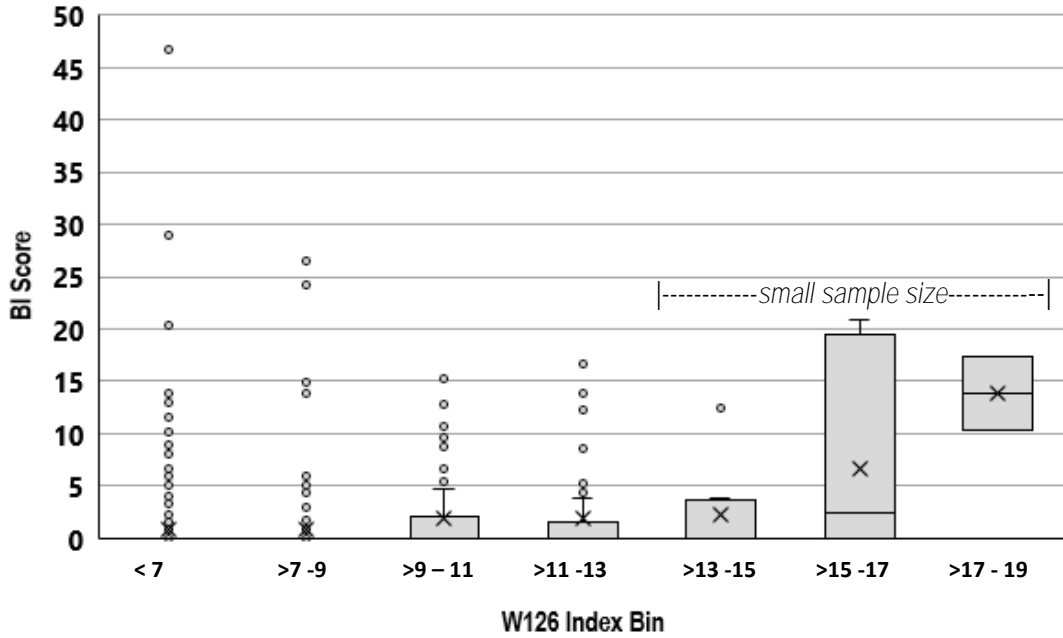
Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

Figure 4C-8. Distribution of BI scores (including zeros) at USFS biosites (dry soil moisture) grouped by W126 index values.



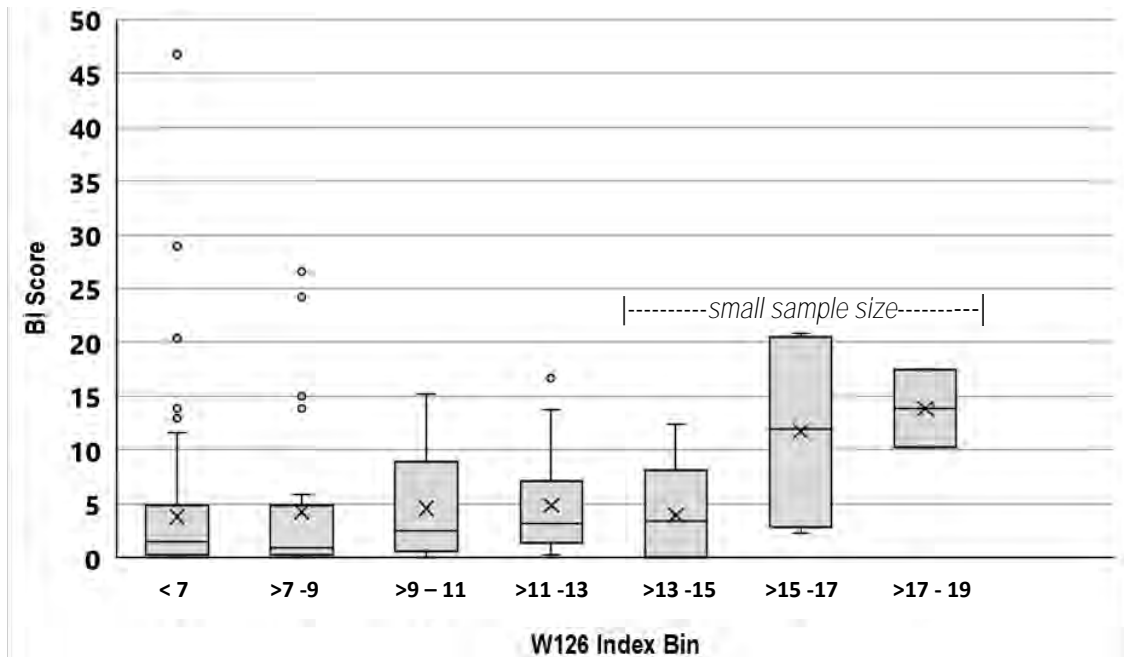
Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus (or 25th percentile minus) 1.5 times the interquartile range (75th minus 25th percentile). Circles show still higher scores

Figure 4C-9. Distribution of nonzero BI scores at USFS biosites (dry soil moisture) grouped by W126 index values.



Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

Figure 4C-10. Distribution of BI scores (including zeros) at USFS biosites (wet soil moisture) grouped by W126 index values.



Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus (and the 25th minus) 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores above that.

Figure 4C-11. Distribution of nonzero BI scores at USFS biosites (wet soil moisture) grouped by W126 index values.

1 **Table 4C-5. Average BI scores of the records in each W126 index bin.**

Soil Moisture	≤ 7 ppm-hrs	>7 -9 ppm-hrs	>9 - 11 ppm-hrs	>11 -13 ppm-hrs	>13 -15 ppm-hrs	>15 -17 ppm-hrs	> 17 - 19 ppm-hrs	> 19 - 25 ppm-hrs	> 25 ppm-hrs
Average BI (all records)									
Dry	0.9	0.3	0.4	0.4	0.4	0.2	2.3	2.1	3.5
Normal	0.9	0.9	0.8	1.2	1.2	0.9	2.3	1.6	7.9
Wet ^c	0.9	0.9	1.9	1.9	[2.2]	[6.7]	[13.9]	-	-
All	0.9	0.8	0.8	1.1	1.0	0.8	2.4	1.9	5.5
Average BI (records with BI > 0)									
Dry	14.2	3.0	5.1	4.2	5.1	2.6	15.0	10.40	7.60
Normal	6.8	4.7	3.7	6.3	5.2	6.9	6.0	5.19	15.42
Wet ^c	3.8	4.3	4.6	4.9	[4.0]	[11.8]	[13.9]	-	-
All	5.4	4.4	4.0	6.0	5.1	6.3	9.0	7.8	11.3
Average BI (records with BI >5)									
Dry	23.6	6.9	8.1	6.6	7.1	6.3	41.1	18.4	14.2
Normal	17.0	14.3	10.5	19.0	19.7	15.1	13.8	18.3	18.5
Wet ^c	11.4	14.2	9.7	10.4	[12.5]	[20.2]	[13.9]	-	-
All	14.9	13.7	10.0	15.7	16.1	15.2	21.0	18.4	16.9
Average BI (records with BI >15)									
Dry	39	-	-	-	-	-	60.3	24.1	22.9
Normal	32.0	31.2	22.2	36.0	38.0	27.5	30.5	24.4	27.9
Wet ^c	34.4	25.8	15.2	16.7	-	[20.2]	[17.4]	-	-
All	33.2	29.6	20.4	33.6	38.0	24.6	41.4	24.2	26.3
^A Brackets indicate bins in which total sample size for that bin is below 1% of all for that soil moisture category (i.e., 0 to 9 samples).									

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1 **Table 4C-6. Proportion of records in each W126 index bin with specified BI score.**

Soil Moisture	≤ 7 ppm-hrs	>7 -9 ppm-hrs	>9 - 11 ppm-hrs	>11 -13 ppm-hrs	>13 -15 ppm-hrs	>15 -17 ppm-hrs	> 17 - 19 ppm-hrs	> 19 – 25 ppm-hrs	>25 ppm-hrs
Proportion of Records with BI >15 (USFS categories of “moderate” and “severe”)									
Dry	0.02	0.00	0.00	0.00	0.00	0.00	0.03	0.07	0.08
Normal	0.02	0.01	0.01	0.02	0.02	0.02	0.03	0.04	0.20
Wet ^A	0.01	0.02	0.01	0.02	[0.00]	[0.29 (2)]	[0.50 (1)]	[0.00]	[0.00]
All	0.01	0.01	0.01	0.02	0.01	0.02	0.04	0.06	0.13
Proportion of Records with BI >5 (USFS categories of “low,” “moderate” and “severe”)									
Dry	0.04	0.03	0.04	0.04	0.05	0.01	0.05	0.11	0.20
Normal	0.05	0.05	0.05	0.05	0.05	0.05	0.13	0.06	0.41
Wet ^A	0.06	0.05	0.16	0.14	[0.11 (1)]	[0.29 (2)]	[1.00 (2)]	[0.00]	[0.00]
All	0.05	0.05	0.06	0.06	0.05	0.04	0.10	0.09	0.30
Proportion of Records with BI >5 & ≤15 (USFS category of “low”)									
Dry	0.02	0.03	0.04	0.04	0.05	0.01	0.02	0.04	0.12
Normal	0.03	0.04	0.05	0.03	0.03	0.03	0.10	0.02	0.22
Wet ^A	0.05	0.03	0.15	0.12	[0.11 (1)]	[0.00]	[0.50 (1)]	[0.00]	[0.00]
All	0.04	0.04	0.06	0.04	0.04	0.02	0.06	0.03	0.16
Proportion of Records with BI >0 & ≤5 (USFS category of “little”)									
Dry	0.03	0.09	0.03	0.04	0.02	0.08	0.10	0.10	0.26
Normal	0.09	0.14	0.16	0.14	0.18	0.08	0.25	0.24	0.10
Wet ^A	0.17	0.15	0.26	0.26	[0.44 (4)]	[0.29 (2)]	0.00	[0.00]	0.00
All	0.12	0.14	0.15	0.13	0.15	0.09	0.17	0.16	0.19
Proportion of Records with BI >0 (USFS categories of “little,” “low,” “moderate” and “severe”)									
Dry	0.	0.11	0.08	0.08	0.07	0.09	0.15	0.21	0.46
Normal	0.13	0.19	0.21	0.19	0.23	0.14	0.38	0.31	0.51
Wet ^A	0.23	0.20	0.42	0.40	[0.56 (5)]	[0.57 (4)]	[1.00 (2)]	[0.00]	[0.00]
All	0.17	0.18	0.21	0.19	0.20	0.13	0.27	0.25	0.48
Proportion of Records with BI =0 (USFS category of no injury)									
Dry	0.94	0.89	0.92	0.92	0.93	0.91	0.85	0.79	0.54
Normal	0.87	0.81	0.79	0.81	0.77	0.86	0.62	0.69	0.49
Wet ^A	0.77	0.80	0.58	0.60	[0.44 (4)]	[0.43 (3)]	[0.00]	[0.00]	[0.00]
All	0.83	0.82	0.79	0.81	0.80	0.87	0.73	0.75	0.52
^A Brackets indicate bins in which total sample size for that bin is below 1% of all for that soil moisture category (i.e., 0 to 9 samples). Additionally, for these entries the value in parenthesis is the number of records in specified BI bin.									

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1 The observations of visible foliar injury for the highest W126 bin compared to the others
2 is generally consistent with the evidence regarding visible foliar injury as an indicator of O₃
3 exposure (e.g., ISA, Appendix 8, section 8.2; 2013 ISA, section 9.4.2; U.S. EPA, 2006 [2006
4 AQCD], p. AX9-22). The evidence indicates a generally greater extent and severity of visible
5 foliar injury with higher O₃ exposure levels and an influence for soil moisture conditions (ISA,
6 Appendix 8, Section 8.2). Further, consistent with this evidence, the censored regression of the
7 USFS dataset described in section 4C.1.1 above found a significant relationship between visible
8 foliar injury and both O₃ and moisture, as measured by Palmer Z.

9 A study cited in the current and 2013 ISAs, which analyzed trends in the incidence and
10 severity of foliar injury, observed a declining trend in the incidence of foliar injury as peak O₃
11 concentrations declined (2013 ISA, p. 9-40; Smith, 2012). Another study, also available in the
12 last review, that focused on O₃-induced visible foliar injury in west coast forests observed that
13 both percentage of biosites with injury and average BI were higher for sites with average
14 cumulative O₃ concentrations above 25 ppm-hrs in terms of SUM06⁸ as compared to groups of
15 biosites with lower average cumulative exposure concentrations, with much less clear differences
16 between the two lower exposure groups (Campbell et al., 2007, Figures 27 and 28 and p. 30). A
17 similar finding was reported in the 2007 Staff Paper which reported on an analysis that showed a
18 smaller percentage of biosites with injury among the group of biosites with O₃ exposures aot or
19 below a SUM06 metric of 15 ppm-hrs or a 4th high metric of 74 ppb as compared to larger
20 groups that also included biosites with SUM06 values up to 25 ppm-hrs or 4th high metric up to
21 84 ppb, respectively (U.S. EPA, 2007 [2007 Staff Paper], pp. 7-63 to 7-64).

22 The observations described here have a general consistency with the extensive evidence
23 base on foliar injury, which indicates that visible foliar injury prevalence and severity are
24 generally higher at higher (compared to lower) O₃ concentrations. As the FIA/FHM biosites vary
25 in the type of vegetation and species that are present and the vegetation types and species vary in
26 sensitivity, BI scores would be expected to differ even between two biosites identical in all
27 environmental characteristics when there are different species present. Therefore, limitations in
28 the biosite dataset can affect patterns and relationships observed in the BI scores. Additionally,
29 various environmental and genetic factors influence the exposure-response relationship, with the
30 most well understood being soil moisture conditions (ISA, Appendix 8, Section 8.2). Our
31 understanding of specific aspects of these influences on the relationship between O₃ exposures,
32 the most appropriate exposure metrics, and the occurrence or severity of visible foliar injury is,
33 however, still incomplete.

⁸ Based on an approach used in the 2007 Staff Paper (and the associated temporal patterns of O₃ concentrations in data available at that time), a SUM06 index value of 25 ppm-hrs would be estimated to correspond to a W126 index of approximately 21 ppm-hrs (2007 Staff Paper, Appendix 7B, p. 7B-2).

1 4C.5 LIMITATIONS AND UNCERTAINTIES

2 The purpose of the analyses and presentations summarized above was to investigate the
3 potential relationship between BI scores at USFS biosites and O₃ in terms of the seasonal W126
4 index. The lack of a clear relationship (across W126 bins below 25 ppm-hrs) in the presentations
5 above may relate to inherent limitations and uncertainties in the different aspects of the dataset.
6 The limitations and uncertainties associated with aspects of the dataset developed for the 2014
7 WREA, and further investigated above, are presented here. In summarizing these below, they are
8 grouped into four areas: 1) biosite scores, 2) soil moisture categorization, 3) W126 index
9 estimates, and 4) combining of datasets.

10 **Biosite data:** Site selection, availability, and species presence also contribute to
11 uncertainty within the dataset and analysis. Data are lacking from many western states including
12 Montana, Idaho, Wyoming, Nevada, Utah, Colorado, Arizona, New Mexico, Oklahoma, and
13 portions of Texas. Furthermore, in certain states (California, Washington, and Oregon) exact
14 locations of sampled sites were not available, and these sites were assigned to the grid based on
15 publicly available geographic coordinates, increasing the level of uncertainty. Because the grid
16 sizes are relatively small, limiting the geographic skew of estimated location (7 km in any
17 direction), it is likely that these locations were at least assigned to adjacent grid cells. While the
18 extent of such differences and magnitude of any effect on the resultant dataset are unknown, it
19 may have relatively small difference and low magnitude of influence on the dataset (2014
20 WREA, p. 7-60).

21 **Soil moisture categories:** The use of the Palmer Z soil moisture index contributes
22 uncertainty of unknown directionality and magnitude. Short-term estimates of soil moisture can
23 be highly variable from month to month within a single year. Using averages contributes to a
24 potential temporal mismatch between soil moisture and injury. Soil moisture is also substantially
25 spatially variable, and the soil moisture data can be hundreds of miles wide in climate regions.
26 There is much diversity within regions, and some vegetation, such as that along riverbanks, may
27 experience sufficient soil moisture during periods of drought to exhibit foliar injury. All of these
28 factors contribute uncertainty to this categorization (2014 WREA, p. 7-61).

29 **W126 index estimates:** Ambient air quality measurements have some inherent
30 uncertainties (considered low [2014 WREA, p. 4-39]) associated with them. These uncertainties
31 relate to monitoring network design, O₃ monitoring seasons, monitor malfunctions, wildlife and
32 wildfire/smoke impacts, and interpolations of missing data. There is likely somewhat greater
33 uncertainty associated with the assignment of W126 index estimates to all biosites due to the
34 need for interpolating between monitor sites to estimate concentrations in unmonitored areas

1 (2014 WREA, sections 4A.2.1).⁹ Accordingly, there is relatively greater uncertainty associated
2 with sites at some distance from monitoring sites and lesser uncertainty in densely monitored
3 areas (2014 WREA, p. 4-40). Unfortunately, which sites are which is unknown.

4 **Combining datasets:** Uncertainty is associated with the combination of data types of
5 different spatial resolution. For example, the biosite scores are available at a much finer spatial
6 resolution than the W126 index estimates, which represent a much small spatial area than that
7 represented by the soil moisture categorization. Yet, as recognized above, soil moisture may vary
8 on much finer scales. To avoid losing resolution of the finest-scale dataset (the biosite scores),
9 the finest spatial resolution available was used (e.g., rather than averaging the BI scores across
10 the grids for which W126 index was estimated or across the climate regions for which the soil
11 moisture scores area available), although this approach contributes its own uncertainty.

12 There is also uncertainty in the combination step associated with the differing temporal
13 scales or time-of-year represented by the three types of data.

14 Overall, we recognize a number of limitations and uncertainties that may be affecting our
15 ability to identify a relationship between O₃, as quantified by seasonal W126 index, and visible
16 foliar injury at USFS locations (based on BI scores), particularly at sites with W126 index
17 estimates at or below 25 ppm-hrs.

18 **4C.6 SUMMARY AND KEY OBSERVATIONS**

19 The following are key observations concerning the dataset presented in this appendix,
20 which includes the subset of USFS biosite data for the years 2006 through 2010, and for which
21 limitations and uncertainties are recognized in section 4C.5 above.

22 **Full Dataset:**

- 23 • The combined dataset includes more than 5,000 records, each of which documents a biotic
24 index scores, soil moisture index value and W126 index estimate, for USFS biosites in 37
25 states in one or more years from 2006 to 2010.
- 26 • The majority of the records are for W126 index estimates at or below 9 ppm-hrs, with
27 fewer than 10% of records assigned W126 index estimates above 15 ppm-hrs.
- 28 • The BI scores (in all soil moisture categories) are quite variable, with at least half the
29 scores in nearly all bins being zero, and even the bin for the lowest W126 index estimates
30 (below 7 ppm-hrs) having at least one scores above 5 and 15.
- 31 • With regard to soil moisture conditions most of the dataset (61% of all records) are for
32 soil moisture conditions categorized as normal. The remainder include somewhat more

⁹ Evaluations of the VNA interpolation technique describe correlations with monitoring data and indicate more accurate prediction of monitoring data by the VNA method than use of an air quality model (2014 WREA, section 4.A.3.1).

1 records for wet soil moisture conditions than dry, with 23% of all records categorized as
2 wet soil moisture conditions and 16% as dry soil moisture.

3 **Records in Wet Soil Moisture Category:**

- 4 • The wet soil moisture records are concentrated in the two lower W126 index bins which
5 contain nearly 90% of all records for this soil moisture category.
 - 6 – Accordingly, interpretations of patterns across W126 bins for this soil moisture
7 category are limited by small sample size across the bins. For example, the number of
8 records in each of the W126 bins above 13 ppm-hrs (ranging from zero to 9)
9 comprise less than 1% of the records in this soil moisture category.

10 **Records in Normal Soil Moisture Category:**

- 11 • Among records in the normal soil moisture category, BI scores are noticeably increased in
12 the highest W126 index bin (index estimates above 25 ppm-hrs), averaging 7.9.
 - 13 – The percentages of records in this W126 bin with scores above 15 or above 5 are
14 more than three times greater than percentages for these score magnitudes in any of
15 the lower W126 index bins.
 - 16 – The average BI score for records in the highest W126 bin is also appreciably greater
17 than scores for records in the other bins. The average scores in the next two highest
18 W126 bins are 1.6 and 2.3, respectively, which are only slightly higher than average
19 scores for the rest of the bins, which vary from 0.8 to 1.2 without a clear relationship
20 to estimated W126 index.
 - 21 – Among the records in this category with nonzero scores, the highest average BI is
22 also in the highest W126 index bin (>25 ppm-hrs); in this case the BI is
23 approximately 15, more than double the next highest average scores across the other
24 W126 index bins (for which no other trend is exhibited). The proportion of records
25 with any injury is also highest in the highest W126 index bin; it is also slightly
26 increased in the next lower W126 bins compared to the rest (the bins at or below 17
27 ppm-hrs) across which there is little evident pattern.

28 **Records in Dry Soil Moisture Category:**

- 29 • Dry soil moisture category records in the two highest W126 bins (>19 and > 25 ppm-hrs)
30 exhibit the greatest percentages of records with BI above 15 and above 5. For scores
31 above 15, the percentages are 7 and 8% compared to 0 to 3 % in the other bins, and for
32 scores above 5, the scores are 11 and 20% compared to 1 to 5% in the other bins.

33 In summary, the observations described here are generally consistent with the extensive
34 evidence base on foliar injury and O₃, which indicates that foliar injury prevalence and severity
35 are generally higher at higher (compared to lower) O₃ concentrations. The presentations here of
36 USFS data do not indicate clear trends in BI across the full range of W126 index estimates.
37 Rather, they indicate increased BI for the highest estimates, with the increase in both incidence
38 of higher scores and in average score being most clear for W126 index estimates above 25 ppm-
39 hrs, with a suggestion of slight increase for some records with W126 index estimates above 17 or

1 19 ppm-hrs (dry soil moisture category). Variability as well as sample size limitations contribute
2 to the lack of more precise conclusions. Additionally, as indicated in the evidence summarized in
3 the ISA and prior scientific assessments, various environmental and genetic factors influence the
4 exposure-response relationship. Our understanding of specific aspects of these influences on the
5 relationship between O₃ exposures, the most appropriate exposure metrics, and the occurrence or
6 severity of visible foliar injury is, however, still incomplete.

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8

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APPENDIX 4D

ANALYSIS OF THE W126 O₃ EXPOSURE INDEX AT U.S. AMBIENT AIR MONITORING SITES

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1 **4D.1 OVERVIEW**

2 This appendix presents various analyses of ambient air monitoring data for ozone (O₃)
3 concentrations in the U.S. relating to the W126-based cumulative exposure index. These
4 analyses focus on the annual maximum 3-month sum of daytime hourly weighted O₃
5 concentrations, averaged over 3 consecutive years, hereafter referred to as the “W126 metric,”
6 calculated as described in section 2 below. These analyses examine spatial and temporal patterns
7 in the W126 metric using monitoring data from 2000 to 2020 and make various comparisons
8 between the W126 metric and design values for the current O₃ standard (the annual 4th highest
9 daily maximum 8-hour O₃ concentration, averaged over 3 consecutive years; hereafter referred to
10 as the “4th max metric”). Additional analyses assess the relative variability between the W126
11 metric and its constituent annual index values and the magnitude of W126 index values at
12 monitoring sites in or near federally protected ecosystems known as Class I areas. These
13 analyses are largely parallel to analyses that were completed for the last review of the O₃
14 NAAQS (79 FR 75331, December 17, 2014; 80 FR 65385, October 26, 2015; U.S. EPA, 2014a,
15 Wells, 2014, Wells, 2015).

16 **4D.2 DATA HANDLING**

17 **4D.2.1 Data Retrieval and Preparation**

18 Hourly O₃ concentration data were retrieved from the EPA’s Air Quality System (AQS,
19 <https://www.epa.gov/aqs>) database for 2,021 ambient air monitoring sites which operated
20 between 2000 and 2020. These data were used to calculate W126 and 4th max metric values for
21 each 3-year period from 2000-2002 to 2018-2020. Before calculating these metrics, some initial
22 processing was done on the hourly data. First, data collected using monitoring methods other
23 than federal reference or equivalent methods, and data collected at monitoring sites not meeting
24 EPA’s quality assurance or other criteria in 40 CFR part 58 were removed from the analysis.
25 Second, data collected by multiple monitoring instruments operating at the same location were
26 combined according to Appendix U to 40 CFR Part 50. Finally, data were combined across 108
27 pairs of monitoring sites approved for such combination by the EPA Regional Offices. The final
28 hourly O₃ concentration dataset contained 1,808 monitoring sites.

29 **4D.2.2 Derivation of the 4th Max and W126 Metrics**

30 The 4th max metric values were calculated according to the data handling procedures in
31 Appendix U to 40 CFR part 50. First, moving 8-hour averages were calculated from the hourly
32 O₃ concentration data for each site. For each 8-hour period, an 8-hour average value was
33 calculated if there were at least 6 hourly O₃ concentrations available. Each 8-hour average was
34 stored in the first hour of the period (e.g., the 8-hour average from 12:00 PM to 8:00 PM is

1 stored in the 12:00 PM hour). Daily maximum 8-hour average values were found using the 8-
2 hour periods beginning from 7:00 AM to 11:00 PM each day. These daily maximum values were
3 used if at least 13 of the 17 possible 8-hour averages were available, or if the daily maximum
4 value was greater than 70 parts per billion (ppb). Finally, the annual 4th highest daily maximum
5 value was found for each year, then averaged across each consecutive 3-year period to obtain the
6 final set of 4th max metric values in units of ppb. Any decimal digits in these values were
7 truncated for applications requiring direct comparison to a 4th max level (e.g., Table 4D-2),
8 otherwise, all decimal digits were retained. The 4th max metric values were considered valid if
9 daily maximum values were available for at least 90% of the days in the O₃ monitoring season
10 (defined in Appendix D to 40 CFR part 58) on average across the three years, with a minimum of
11 75% of the days in the O₃ monitoring season in any calendar year. In addition, 4th max metric
12 values were considered valid if they were greater than the 4th max levels to which they were
13 being compared.

14 The W126 metric values were calculated using the hourly O₃ concentration data in parts
15 per million (80 FR 65374, October 26, 2015). For daytime hours (defined as the 12-hour period
16 from 8:00 AM to 8:00 PM Local Standard Time each day), the hourly concentration values at
17 each O₃ monitoring site were weighted using the following equation:

18
$$\text{Weighted O}_3 = \text{O}_3 / (1 + 4403 * \exp (-126 * \text{O}_3)).$$

19 These weighted values were summed over each calendar month, then adjusted for
20 missing data (e.g., if 80% of the daytime hourly concentrations were available, the sum would be
21 multiplied by $1/0.8 = 1.25$) to obtain the monthly W126 index values. Monthly W126 index
22 values were not calculated for months where fewer than 75% of the possible daytime hourly
23 concentrations were available. Next, moving 3-month sums were calculated from the monthly
24 index values, and the highest of these 3-month sums was determined to be the annual W126
25 index. Three-month periods spanning multiple years (e.g., November to January, December to
26 February) were not considered in these calculations. The annual W126 index values were
27 averaged across each consecutive 3-year period to obtain the final W126 metric values, with
28 units in parts per million-hours (ppm-hrs). The W126 metric values were rounded to the nearest
29 unit ppm-hr for applications requiring direct comparison to a W126 level (e.g., Table 4D-3),
30 otherwise, all decimal digits were retained. For consistency with the 4th max metric calculations,
31 the W126 metric values were considered valid if hourly O₃ concentration values were available
32 for at least 90% of the daytime hours during the O₃ monitoring season on average across the
33 three years, with a minimum of 75% of the daytime hours during the O₃ monitoring season in
34 any calendar year. Also, for consistency with the 4th max metric calculations, the W126 metric

1 values were considered valid if they were greater than the W126 levels to which they were being
2 compared.

3 In summary, the “4th max metric” refers to the average of the 4th highest daily maximum
4 8-hour averages in three consecutive years and the “W126 metric” refers to the average of annual
5 W126 index values (“annual” or “single-year” W126 index) over three years. In the final dataset,
6 1,578 of the 1,808 O₃ monitoring sites had sufficient data to calculate valid 4th max and W126
7 metric values for at least one 3-year period between 2000-2002 and 2018-2020. The number of
8 sites with valid 4th max and W126 metric values ranged from a low of 992 in 2000-2002 to a
9 high of 1,118 in 2015-2017, and 510 sites had valid 4th max and W126 metric values for all
10 nineteen 3-year periods.

11 **4D.2.3 Derivation of Temporal Trends**

12 Site-level trends for the W126 metric and annual W126 index values were computed in a
13 similar manner to the site-level trends for the 4th max metric presented in Chapter 2. Specifically,
14 for the annual W126 index, a site must have at least 75% annual data completeness for at least 16
15 of the 21 years, with no more than two consecutive years having less than 75% data
16 completeness in order to be included in the analysis. For the W126 metric, a site must have a
17 valid W126 metric value (according to the data completeness criteria presented in the previous
18 section) in at least 15 of the 19 3-year periods, and no more than two consecutive 3-year periods
19 that do not have valid W126 metric values. There were 822 sites meeting these criteria for the
20 annual W126 index and 666 sites meeting these criteria for the W126 metric. The national
21 median, 10th percentile, and 90th percentile values of these site-level trends are presented in
22 Figure 4D-9.

23 Other analyses presented in Section 4D.3.2.2 use trends in the 4th max and W126 metrics
24 as well as the annual W126 index calculated with non-parametric regression methods. These
25 trends were computed using the Theil-Sen estimator (Sen, 1968; Theil, 1950), a type of
26 regression method that chooses the median slope among all lines crossing through each possible
27 pair of sample points¹. These trends are reported in units of ppb/yr for the 4th max metric or ppm-
28 hr/yr for the W126 metric and annual W126 index. The data completeness criteria described in
29 the previous paragraph were also applied to site for which these trends were calculated.²

¹ For example, if applying this method to a dataset with W126 metric values for four consecutive years (e.g., W126₁, W126₂, W126₃, and W126₄), the trend would be the median of the per-year changes observed in the six possible pairs of values (e.g., the median of [W126₄-W126₃]/1, [W126₃-W126₂]/1, [W126₂-W126₁]/1, [W126₄-W126₂]/2, [W126₃-W126₁]/2, and [W126₄-W126₁]/3).

² For the 4th max metric, the data completeness criteria used were valid 4th max metric values (as defined in section 4D.2.2) in 15 of the 19 3-year periods, and no more than two consecutive periods that do not have valid 4th max

1 Statistical tests for significance of the Theil-Sen estimator were computed using the non-
2 parametric Mann-Kendall test (Kendall, 1948; Mann; 1945).

3 **4D.2.4 Identification of O₃ Monitoring Sites in Federal Class I Areas**

4 The Clean Air Act (section 162) designated certain federally areas as Class I areas. These
5 areas are federally mandated to preserve certain air quality values. Class I designation allows the
6 least amount of deterioration of existing air quality. Areas designated as Class I include all
7 international parks, national wilderness areas which exceed 5,000 acres in size, national
8 memorial parks which exceed 5,000 acres in size, and national parks which exceed 6,000 acres in
9 size, provided the park or wilderness area was in existence on August 7, 1977. There are 158
10 such areas (e.g., 44 FR 69122, November 30, 1979). Other areas may, and have been,
11 subsequently designated as Class I consistent with the CAA (section 162). As of January 2022,
12 six Class II areas on Tribal lands have been re-designated as Class I.³

13 To identify which O₃ monitoring sites represented air quality in federal Class I areas,
14 shapefiles (i.e., files that specify area boundaries) for all 158 mandated federal Class I areas⁴
15 were downloaded from EPA’s Environmental Dataset Gateway (EDG; <https://edg.epa.gov/>) and
16 augmented with the six tribal areas redesignated as Class I. These boundaries were matched to
17 the 1,808 O₃ monitoring sites in the hourly O₃ concentration dataset described in section 4D.2.1.
18 Since Class I areas include federally designated wilderness areas in which permanent structures
19 such as air monitoring trailers are prohibited, if there was no monitor located within the area
20 boundary, the matching was expanded to include the nearest monitoring site within 15 km of the
21 boundary. For each Class I area and 3-year period, if a 4th max or W126 metric value was not
22 available for the nearest monitor, the values from the next nearest monitor within 15 km were
23 used, where applicable. In addition, if a Class I area had multiple monitors inside the boundary,
24 we used the monitor with the highest 4th max metric value in each 3-year period. These monitors
25 were extracted from the 4th max and W126 dataset described in section 4D.2.2, yielding a final
26 Class I areas dataset with a total of 980 records that had valid 4th max and W126 metric values at
27 78 O₃ monitoring sites representing 65 Class I areas (out of 164 total Class I areas).

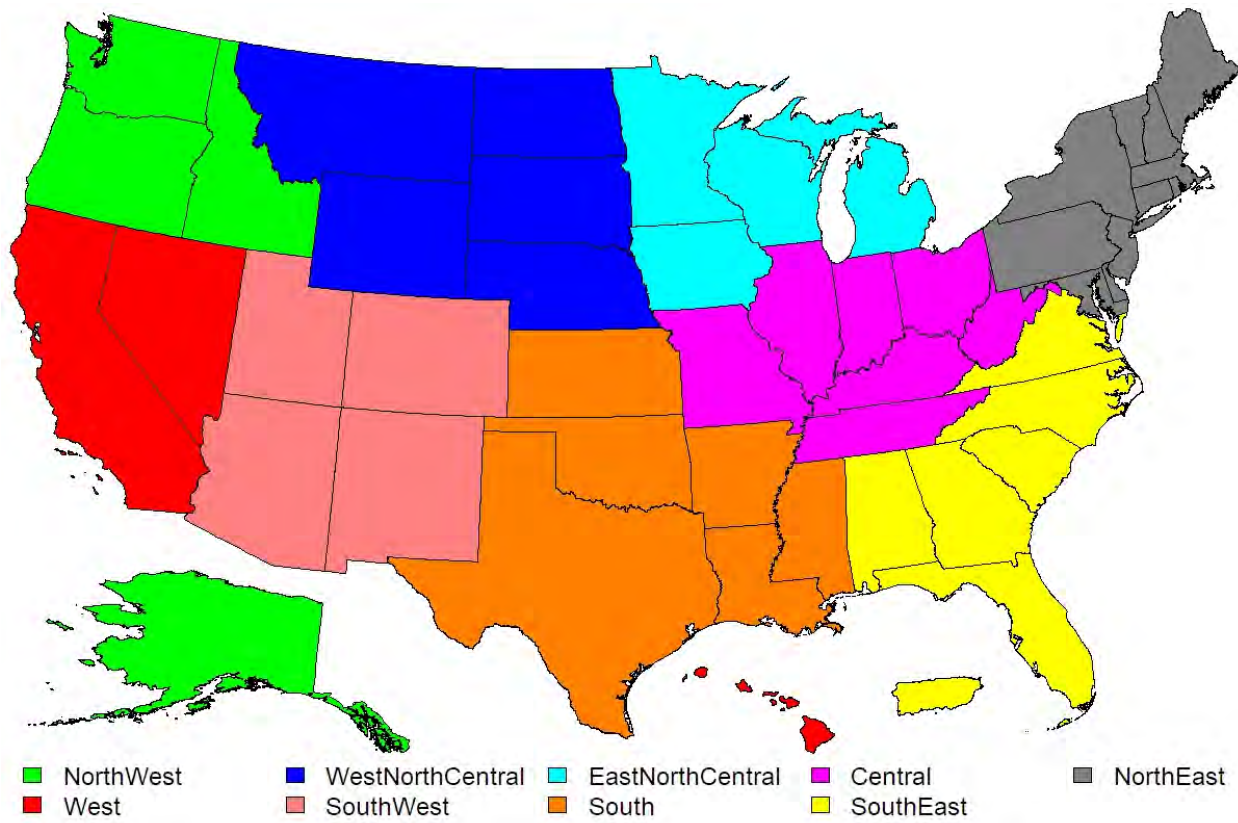
metric values. There were 658 sites meeting these criteria, and all these sites also met the data completeness criteria for the W126 metric and the annual W126 index.

³ The Class I areas on Tribal lands as of December 2020 are listed at: <https://www.nps.gov/subjects/air/tribalclass1.htm>. Since then, one additional area has been designated Class I on Tribal lands (84 FR 34306, July 18, 2019).

⁴ The set of Class I areas identified in 1977 are referred to here as “mandated.”

1 **4D.2.5 Assignment of Monitoring Sites to NOAA Climate Regions**

2 In order to examine regional differences, many of the further analyses were stratified into
3 the nine NOAA climate regions (Karl and Koss, 1984), which are shown in Figure 4D-1. Since
4 the NOAA climate regions only cover the contiguous U.S., Alaska was added to the Northwest
5 region, Hawaii was added to the West region, and Puerto Rico was added to the Southeast
6 region.



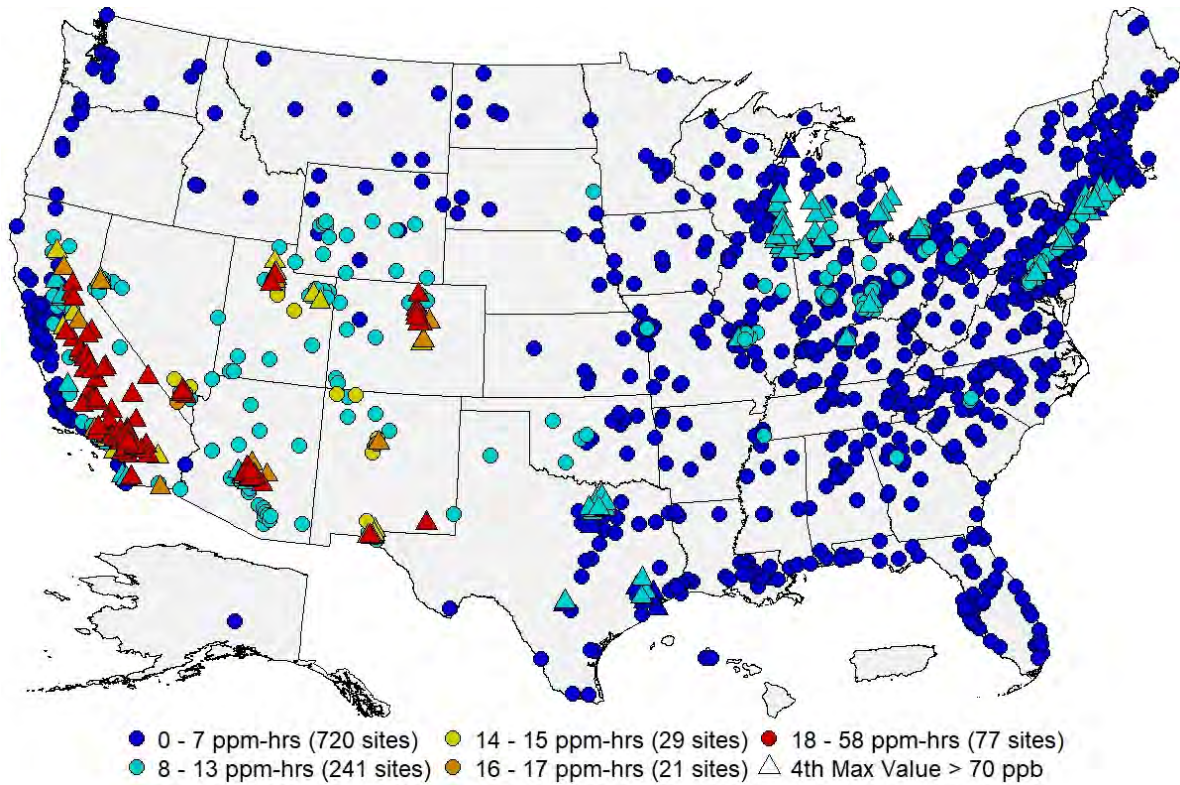
7
8 **Figure 4D-1. Map of the nine NOAA climate regions.**

9 **4D.3 RESULTS**

10 **4D.3.1 National Analysis Using Recent Air Quality Data**

11 This section presents various results based on the 4th max and W126 metrics for the 2018-
12 2020 period. Figure 4D-2 shows a map of the observed W126 metric values based on 2018-2020
13 data. From this figure, it is apparent that W126 metric values are generally at or below 13 ppm-
14 hrs in the eastern and northwestern U.S. In the U.S. as a whole, about 66% of all monitoring sites
15 recorded W126 metric values at or below 7 ppm-hrs, and about 93% of all monitoring sites
16 recorded W126 metric values at or below 17 ppm-hrs. The highest W126 metric values occur in
17 the southwestern U.S. where there are numerous monitoring sites with W126 metric values
18 above 17 ppm-hrs, however, none of these sites meet the current standard. Table 4D-1 shows the

1 number of sites in each NOAA climate region that have a valid 2018-2020 design value meeting
 2 the current standard and the number of sites in each region that have a 2018-2020 design value
 3 not meeting the current standard.



4
 5 **Figure 4D-2. Map of W126 metric values at U.S. O₃ monitoring sites based on 2018-2020**
 6 **data.** Circles indicate monitoring sites with 4th max metric values less than or
 7 equal to 70 ppb, while triangles indicate monitoring sites with 4th max metric
 8 values greater than 70 ppb.

9 **Table 4D-1. Number of O₃ monitoring sites with valid 2018-2020 design values in each**
 10 **NOAA climate region**

NOAA Climate Region	Total # of Sites	# of Sites with Design Value ≤ 70 ppb	# of Sites with Design Value > 70 ppb
Central	203	179	24
EastNorthCentral	78	62	16
NorthEast	179	160	19
NorthWest	23	23	0
South	130	105	25
SouthEast	157	157	0
SouthWest	106	59	47
West	170	88	82
WestNorthCentral	44	44	0
National	1,090	877	213

1 **4D.3.1.1 Comparison of the 4th Max and W126 Metrics**

2 The following analyses make several comparisons between the 4th max and W126 metric
 3 values (both of which are 3-year average metrics) based on 2018-2020 data. Table 4D-2 shows
 4 the number of sites with 4th max metric values greater than each 4th max level, and the number of
 5 sites with 4th max metric values less than or equal to each 4th max level. Table 4D-3 shows the
 6 number of sites with W126 metric values greater than each W126 level, and the number of sites
 7 with W126 metric values less than or equal to each W126 level.

8 The 4th max and W126 metric values were also compared to each combination of 4th max
 9 and W126 levels based on 2018-2020 data. Table 4D-4 shows the number of sites with 4th max
 10 metric values greater than each 4th max level, and W126 metric values less than or equal to each
 11 W126 level (e.g., 127 sites had 4th max metric values greater than 70 ppb and W126 metric
 12 values less than or equal to 13 ppm-hrs). Table 4D-5 shows the number of sites with 4th max
 13 metric values less than or equal to each 4th max level, and W126 metric values greater than each
 14 W126 level (e.g., 13 sites with a 4th max metric value at or below 70 ppb had a W126 metric
 15 value greater than 13 ppm-hrs). Finally, Table 4D-6 shows the number of sites with 4th max
 16 metric values greater than each 4th max level, and W126 metric values greater than each W126
 17 level.

18 **Table 4D-2. Number of sites with 4th max metric values greater than various 4th max levels**
 19 **based on 2018-2020 data.**

4 th Max Level (ppb)	75	70	65
# of Sites > Level	93	213	468
# of Sites ≤ Level	989	877	629
Total # of Sites ^A	1,082	1,090	1,097

^A For each 4th max level, a site with a 4th max metric value less than or equal to the level is counted only if it meets the data completeness criteria described in section 4D.2.2, whereas a site with a 4th max metric value greater than the level is counted regardless of data completeness. Therefore, the total number of sites may differ among the columns.

20 **Table 4D-3. Number of sites with W126 metric values greater than various W126 levels**
 21 **based on 2018-2020 data.**

W126 Level (ppm-hrs)	19	17	15	13	11	9	7
# of Sites > Level	58	77	98	127	170	245	379
# of Sites ≤ Level	1,027	1,009	991	963	921	850	722
Total # of Sites ^A	1,085	1,086	1,089	1,090	1,091	1,095	1,101

^A For each W126 level, a site with a W126 metric value less than or equal to the level is counted only if it meets the data completeness criteria described in section 4D.2.2, whereas a site with a W126 metric value greater than the level is counted regardless of data completeness. Therefore, the total number of sites may differ among the columns.

1 **Table 4D-4. Number of sites with 4th max metric values greater than various 4th max levels**
 2 **and W126 metric values less than or equal to various W126 levels based on**
 3 **2018-2020 data.**

# Sites > 4 th Max Level AND ≤ W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4 th Max Level (ppb)	75	37	25	19	14	11	4	1
	70	146	128	112	95	81	52	13
	65	393	375	357	329	287	225	114

4 **Table 4D-5. Number of sites with 4th max metric values less than or equal to various 4th**
 5 **max levels and W126 metric values greater than various W126 levels based on**
 6 **2018-2020 data.**

# Sites ≤ 4 th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4 th Max Level (ppb)	75	3	9	21	44	83	147	272
	70	0	0	2	13	41	83	172
	65	0	0	0	0	0	9	25

7 **Table 4D-6. Number of sites with 4th max metric values greater than various 4th max levels**
 8 **and W126 metric values greater than various W126 levels based on 2018-2020**
 9 **data.**

# Sites > 4 th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4 th Max Level (ppb)	75	55	68	74	79	82	89	92
	70	58	77	96	114	129	159	199
	65	58	77	98	127	170	234	349

10
 11 According to Table 4D-2, 9% of U.S. O₃ monitoring sites had 2018-2020 4th max metric
 12 values greater than 75 ppb, 19% of sites had 4th max metric values greater than 70 ppb, and 43%
 13 of sites had 4th max metric values greater than 65 ppb. According to Table 4D-3, 7% of U.S. O₃
 14 monitoring sites had 2018-2020 W126 metric values greater than 17 ppm-hrs, 12% of sites had
 15 W126 metric values greater than 13 ppm-hrs, and 34% of sites had W126 metric values greater
 16 than 7 ppm-hrs. According to Table 4D-5, there were no monitoring sites with a 4th max metric
 17 value less than or equal to 70 ppb and a W126 metric value greater than 17 ppm-hrs, only two
 18 monitoring sites with a 4th max less than or equal to 70 ppb and a W126 greater than 15 ppm-hrs
 19 in the 2018-2020 period.

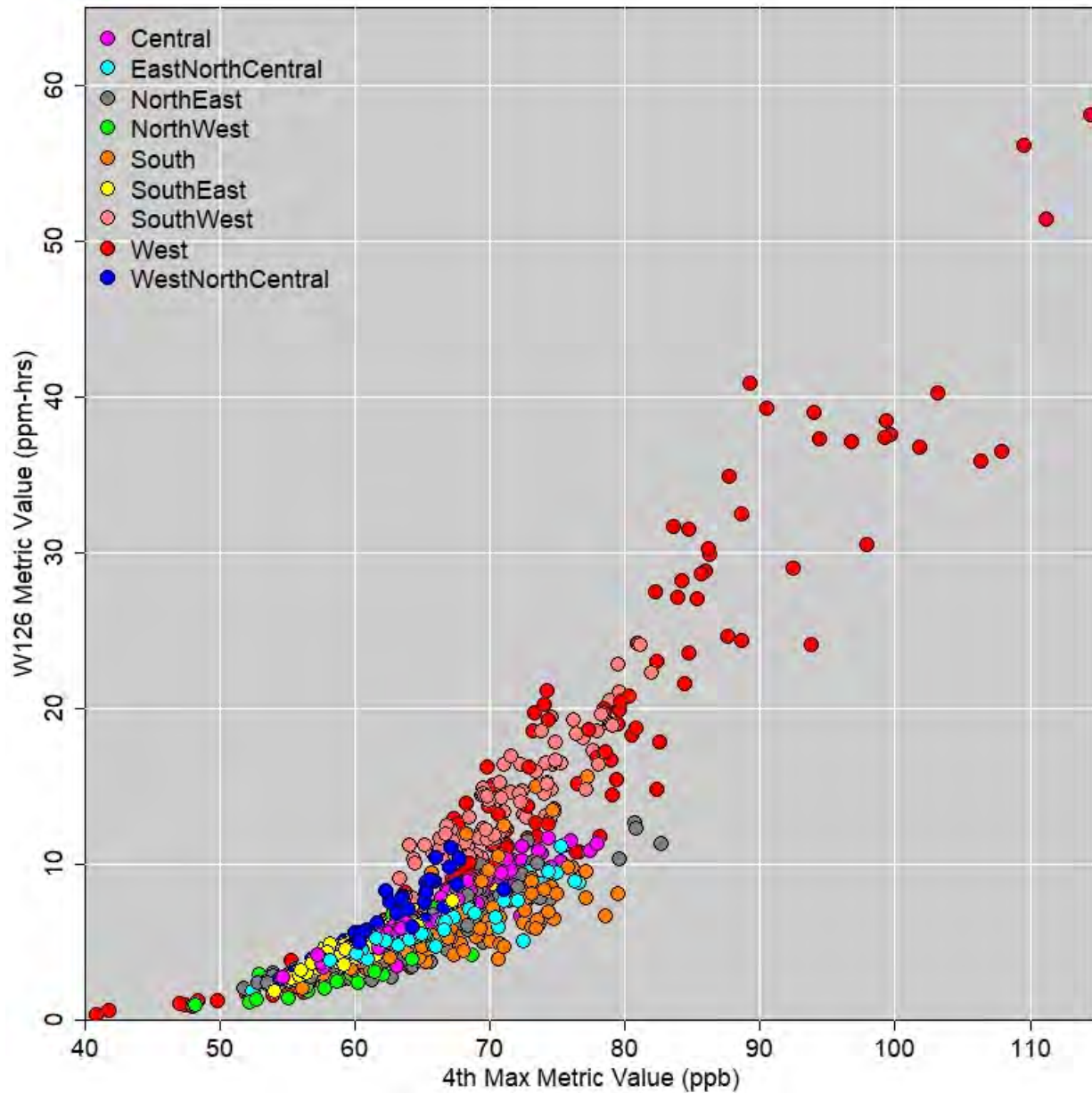
1 **4D.3.1.2 Relationships Between Metrics and the Annual W126 Index**

2 Figure 4D-3 shows a scatter plot comparing the 4th max (x-axis) and W126 (y-axis)
3 metric values (both 3-year averages) based on 2018-2020 data, with points colored by NOAA
4 climate region. This figure indicates that there is a strong, positive, non-linear relationship
5 between the 4th max and W126 metrics. The amount of variability in the relationship between the
6 4th max and W126 metrics appears to increase as the metric values themselves increase. The
7 relationship between the 4th max and W126 metrics also appears to vary across regions. In
8 particular, the Southwest and West regions (i.e., the southwestern U.S.) appear to have higher
9 W126 metric values relative to their respective 4th max metric values than the rest of the U.S.

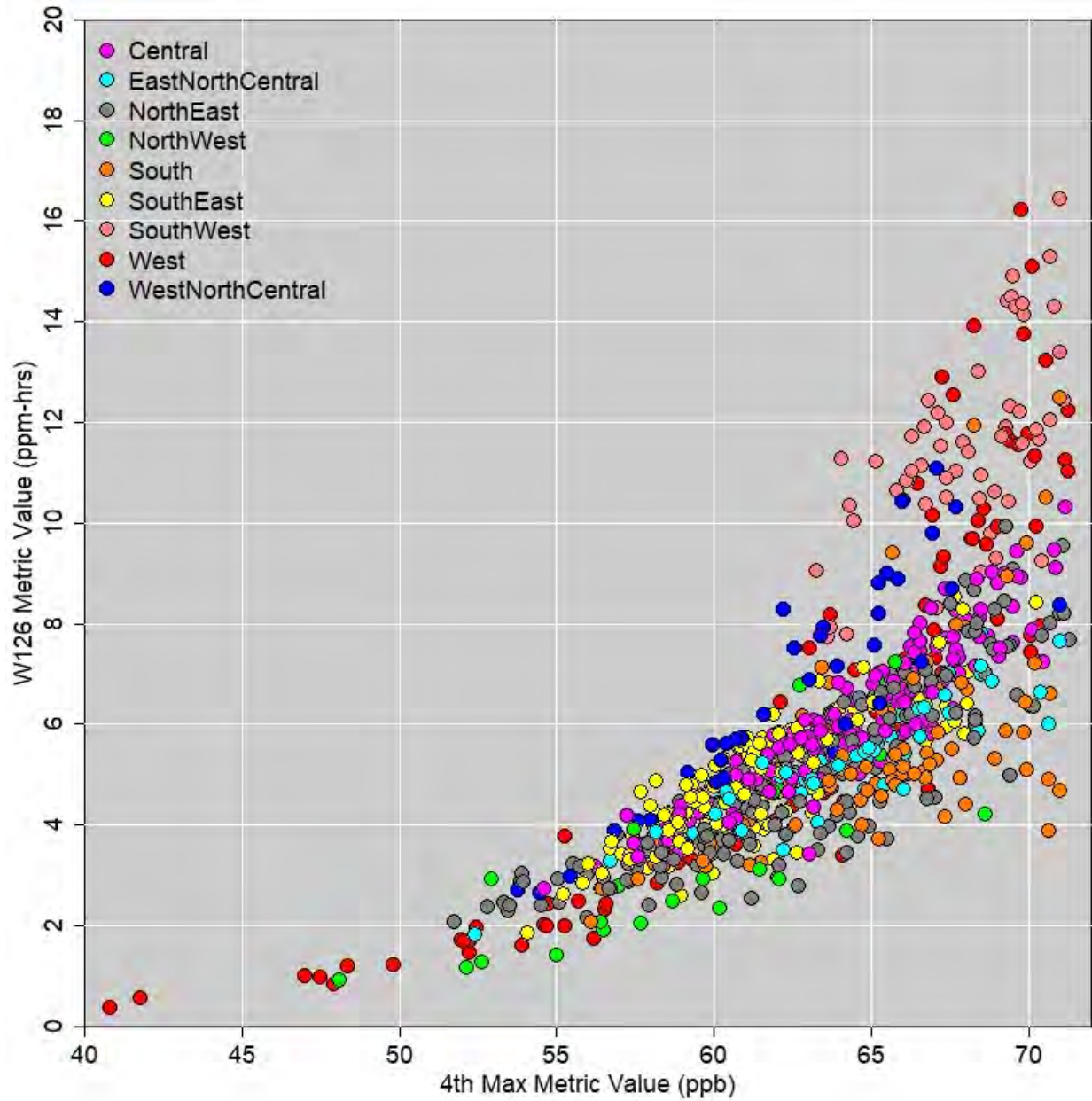
10 Figure 4D-4 shows the same information as Figure 4D-3, but only for monitoring sites
11 meeting the current standard. This figure shows that all monitoring sites meeting the current
12 standard have W126 metric values of 16 ppm-hrs or less, and all sites outside the Southwest and
13 West climate regions have W126 metric values of 13 ppm-hrs or less.

14 Finally, Figure 4D-5 shows a scatter plot comparing the 4th max metric values (x-axis) to
15 the annual W126 index values (y-axis) based on 2018-2020 data, with points colored by NOAA
16 climate region. This figure shows that the annual W126 index values have a similar positive,
17 non-linear relationship with the 4th max metric values as the W126 metric values. As might be
18 expected, there is generally more variability in the relationship between the annual W126 index
19 values and the 4th max metric values than between the W126 metric values and the 4th max
20 metric values.

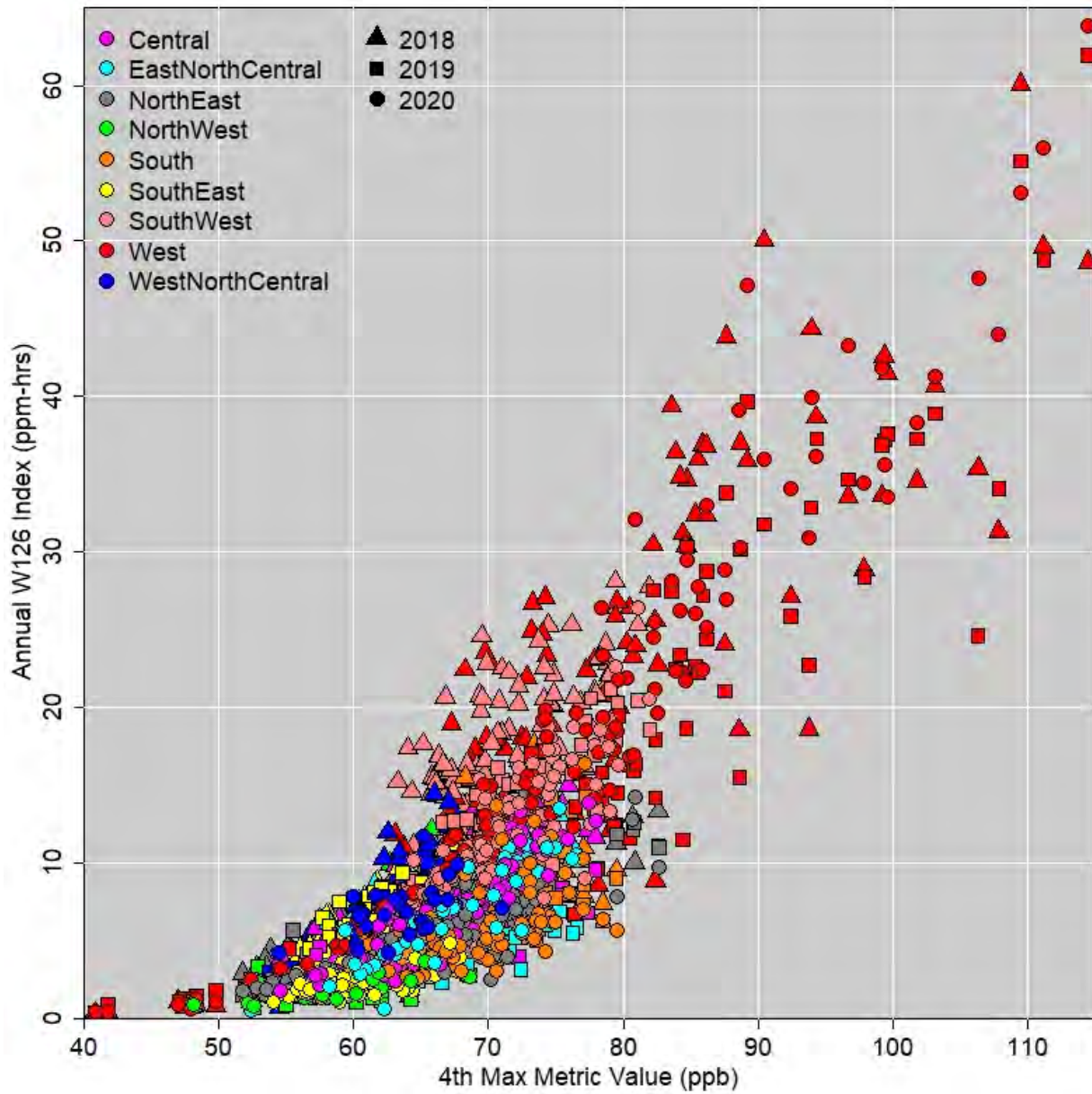
21 Figure 4D-6 shows a scatter plot of the deviations in the 2018, 2019, and 2020 annual
22 W126 index values (y-axis) from the 2018-2020 average W126 metric values (x-axis). This
23 figure shows that the magnitude of the annual W126 index deviations from the 3-year average
24 tend to increase as the W126 metric value increases. About 40% of the annual W126 index
25 values are within +/- 1 ppm-hr of the 3-year average value, about 73% are within +/- 2 ppm-hrs
26 of the 3-year average value, and about 96% are within +/- 5 ppm-hrs of the 3-year average value.
27 Figure 4D-7 also presents the deviations in the 2018, 2019, and 2020 annual W126 index values
28 from their respective 2018-2020 averages for the sites meeting the current standard. For these
29 sites, 42% of annual W126 index values are within 1 ppm-hr of the 3-yr average, 78% are within
30 2 ppm-hrs, and 99% are within 5 ppm-hrs (Figure 4D-7). From these two figures it can be seen
31 that lower 4th max metric values generally correspond to smaller inter-annual variation within
32 W126 metric values, especially for sites meeting the current standard.



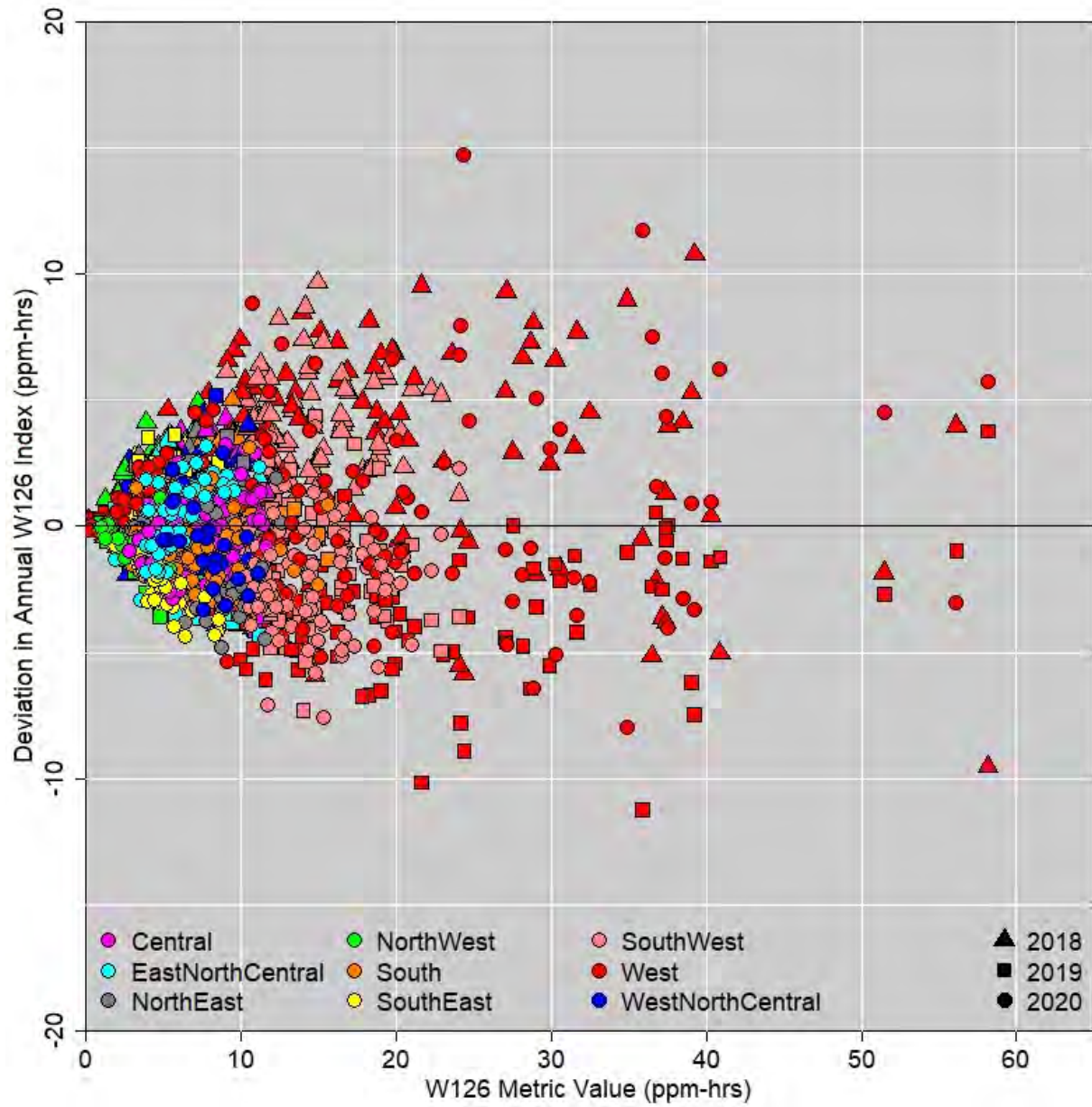
1
 2 **Figure 4D-3. Scatter plot of W126 metric values versus 4th max metric values (design**
 3 **values) based on 2018-2020 monitoring data.**



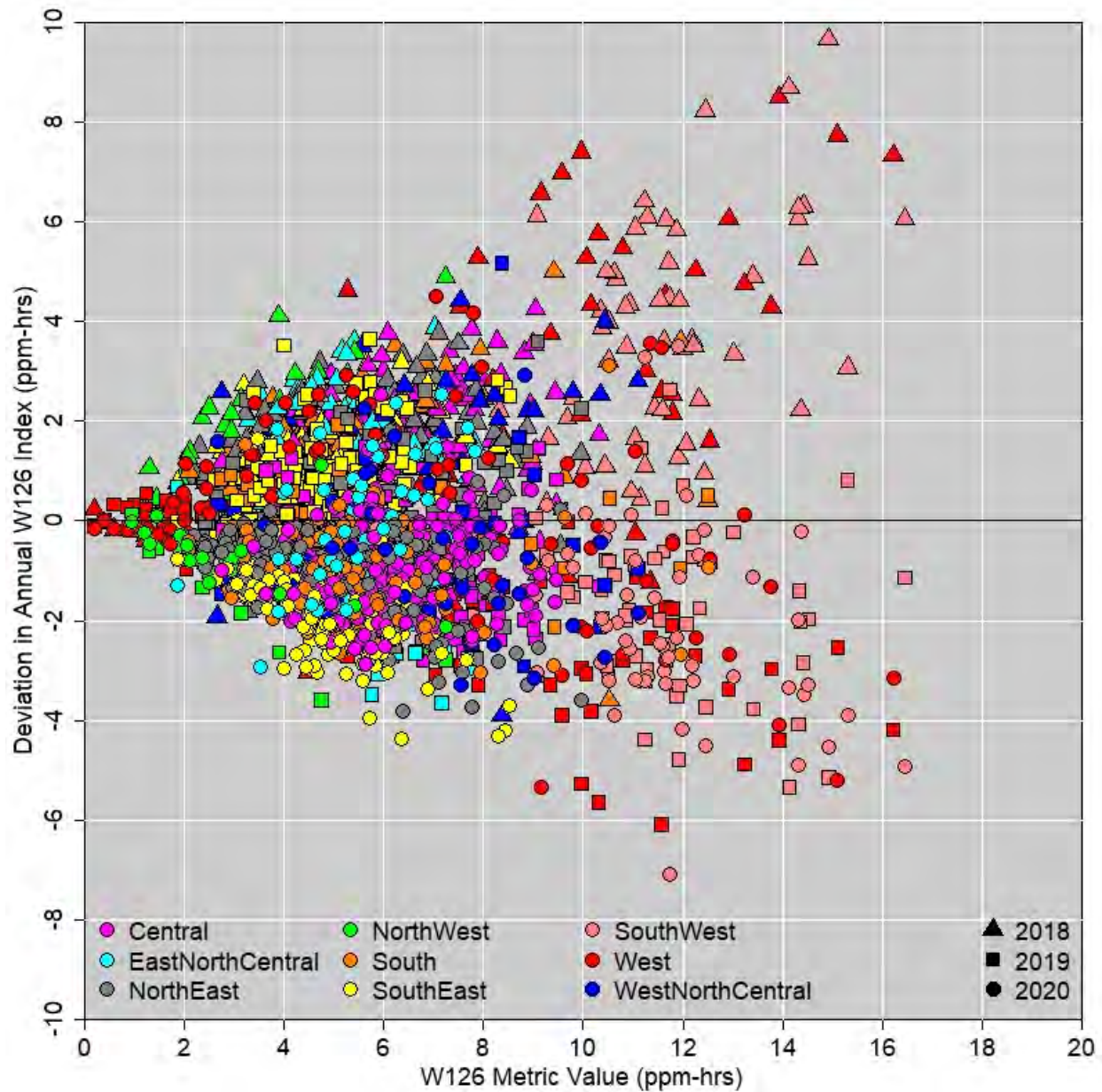
1
 2 **Figure 4D-4. Scatter plot of W126 metric values versus 4th max metric values (design**
 3 **values) at monitoring sites meeting the current standard based on 2018-2020**
 4 **monitoring data.**



1
 2 **Figure 4D-5. Scatter plot of annual W126 index values versus 4th max metric values**
 3 **(design values) based on 2018-2020 monitoring data.**
 4



1
 2 **Figure 4D-6. Deviation in annual W126 index values from their respective 3-year averages**
 3 **for all U.S. monitoring sites in 2018-2020.**



1
 2 **Figure 4D-7. Deviation in annual W126 index values from their respective 3-year averages**
 3 **for all U.S. monitoring sites with 4th max metric values at or below 70 ppb in**
 4 **2018-2020.**

5 **4D.3.2 National Analysis Using Historical Air Quality Data**

6 This section presents various results based on the 4th max and W126 metrics for the full
 7 19-year period spanning years 2000 to 2020. Comparisons similar to those shown in section
 8 4D.3.1 are shown in section 4D.3.2.1, trends in W126 are shown in section 4D.3.2.2, and several
 9 comparisons of the trends in the 4th max and W126 metrics are shown in section 4D.3.2.3.

1 **4D.3.2.1 Comparison of the 4th Max and W126 Metrics**

2 Table 4D-7 to Table 4D-11 present similar information to Table 4D-2 to Table 4D-6,
3 respectively, except that the values shown in each cell contain the number of occurrences
4 summed over all 19 consecutive 3-year periods (2000-2002 to 2018-2020) instead of just the
5 2018-2020 period. For example, Table 4D-10 shows that over all 19 consecutive 3-year periods,
6 there were 276 occurrences where sites had 4th max metric values less than or equal to 70 ppb
7 and W126 metric values greater than 13 ppm-hrs. In general, the relative magnitudes of the
8 numbers shown in Table 4D-7 to Table 4D-11 compare well to their respective counterparts in
9 Table 4D-2 to Table 4D-6. According to Table 4D-10, there have been no occurrences over the
10 entire 21-year period where a site has had a 4th max metric value less than or equal to 70 ppb and
11 a W126 metric value greater than 19 ppm-hrs.⁵

12 Figure 4D-8 shows the distribution of annual W126 index values observed at sites during
13 3-year periods with different 4th max metric values (which are the design values for the current
14 standard). These distributions are illustrated by box-and-whisker plots with boxes showing the
15 25th, 50th, and 75th percentile of the annual W126 index values occurring with 4th max metric
16 values within each bin, whiskers extending to the 1st and 99th percentiles of the annual W126
17 index values, and points occurring outside the 1st and 99th percentiles represented by dots. This
18 figure shows that for the bin with the highest 4th max metric values meeting the current standard,
19 66-70 ppb, the 99th percentile of the annual W126 index values was about 19 ppm-hours, or in
20 other words, for sites meeting the current standard, annual W126 index values were less than or
21 equal to 19 ppm-hrs about 99% of the time.

22

⁵ There was a single occurrence of a site with a 4th max of 70 ppb and a W126 that when rounded, just equaled 19 ppm-hrs.

1 **Table 4D-7. Total number of 4th max metric values greater than various 4th max levels**
 2 **based on all 17 consecutive 3-year periods (2000-2002 to 2018-2020).**

4 th Max Level (ppb)	75	70	65
Values > Level	6,848 (33%)	11,142 (53%)	15,947 (74%)
Values ≤ Level	14,059 (67%)	10,039 (47%)	5,622 (26%)
Total # of Values ^A	20,907	21,181	21,569

^A For each 4th max level, a site with a 4th max metric value less than or equal to the level is counted only if it meets the data completeness criteria described in section 4D.2.2, whereas a site with a 4th max metric value greater than the level is counted regardless of data completeness. Therefore, the total number of values may differ among the columns.

3 **Table 4D-8. Total number of W126 metric values greater than various W126 levels based**
 4 **on all 17 consecutive 3-year periods (2000-2002 to 2018-2020).**

W126 Level (ppm-hrs)	19	17	15	13	11	9	7
Values > Level	2,424	3,329	4,579	6,262	8,315	10,860	13,748
Values ≤ Level	18,303	17,438	16,233	14,628	12,693	10,282	7,587
Total # of Values ^A	20,727	20,767	20,812	20,890	21,008	21,142	21,335

^A For each W126 level, a site with a W126 metric value less than or equal to the level is counted only if it meets the data completeness criteria described in section 4D.2.2, whereas a site with a W126 metric value greater than the level is counted regardless of data completeness. Therefore, the total number of values may differ among the columns.

5 **Table 4D-9. Total number of 4th max metric values greater than various 4th max levels and**
 6 **W126 metric values less than or equal to various W126 levels based on all 17**
 7 **consecutive 3-year periods (2000-2002 to 2018-2020).**

Values > 4 th Max Level AND ≤ W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4 th Max Level (ppb)	75	4,290	3,603	2,719	1,716	856	280	41
	70	8,228	7,372	6,228	4,837	3,254	1,529	408
	65	12,650	11,785	10,580	8,975	7,056	4,781	2,340

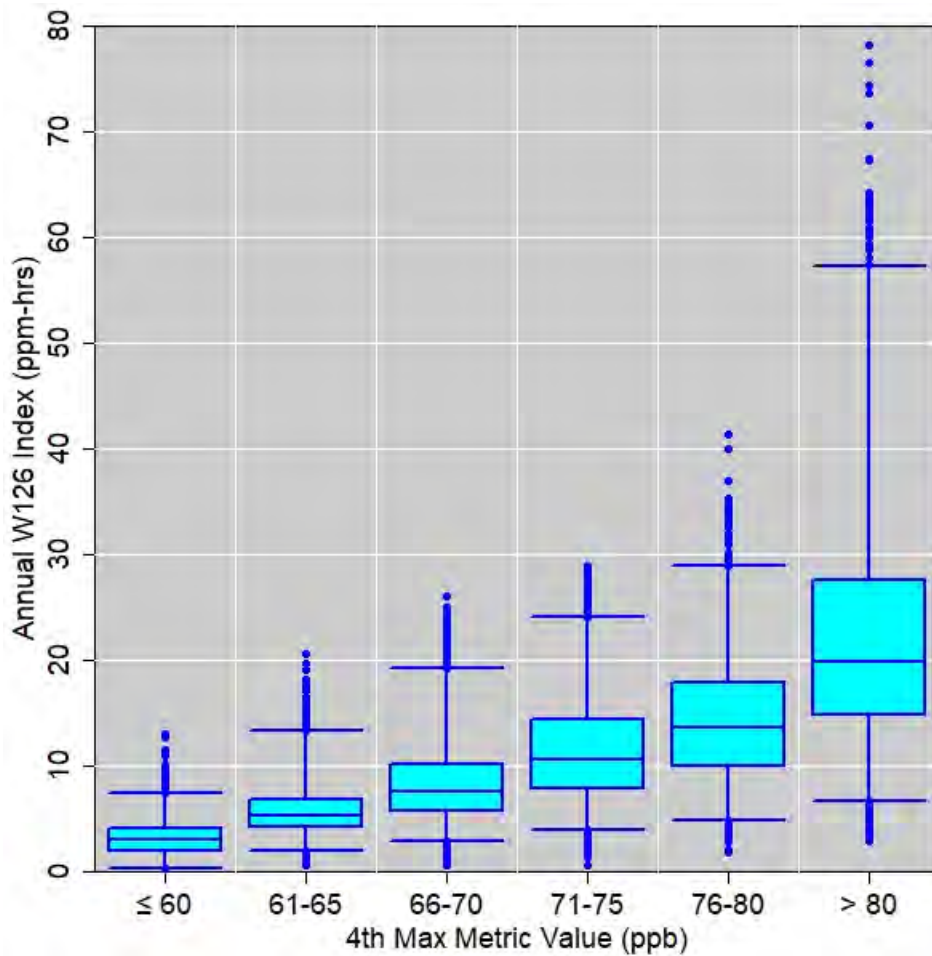
8 **Table 4D-10. Total number of 4th max metric values less than or equal to various 4th max**
 9 **levels and W126 metric values greater than various W126 levels based on all**
 10 **17 consecutive 3-year periods (2000-2002 to 2018-2020).**

Values ≤ 4 th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4 th Max Level (ppb)	75	95	267	585	1,181	2,251	4,069	6,518
	70	0	8	68	276	625	1,304	2,879
	65	0	0	0	0	16	150	400

1 **Table 4D-11. Total number of 4th max metric values greater than various 4th max levels and**
 2 **W126 metric values greater than various W126 levels based on all 17**
 3 **consecutive 3-year periods (2000-2002 to 2018-2020).**

Values > 4 th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4 th Max Level (ppb)	75	2,318	3,032	3,940	4,982	5,895	6,506	6,761
	70	2,424	3,317	4,500	5,946	7,615	9,420	10,611
	65	2,424	3,329	4,579	6,262	8,295	10,685	13,286

4



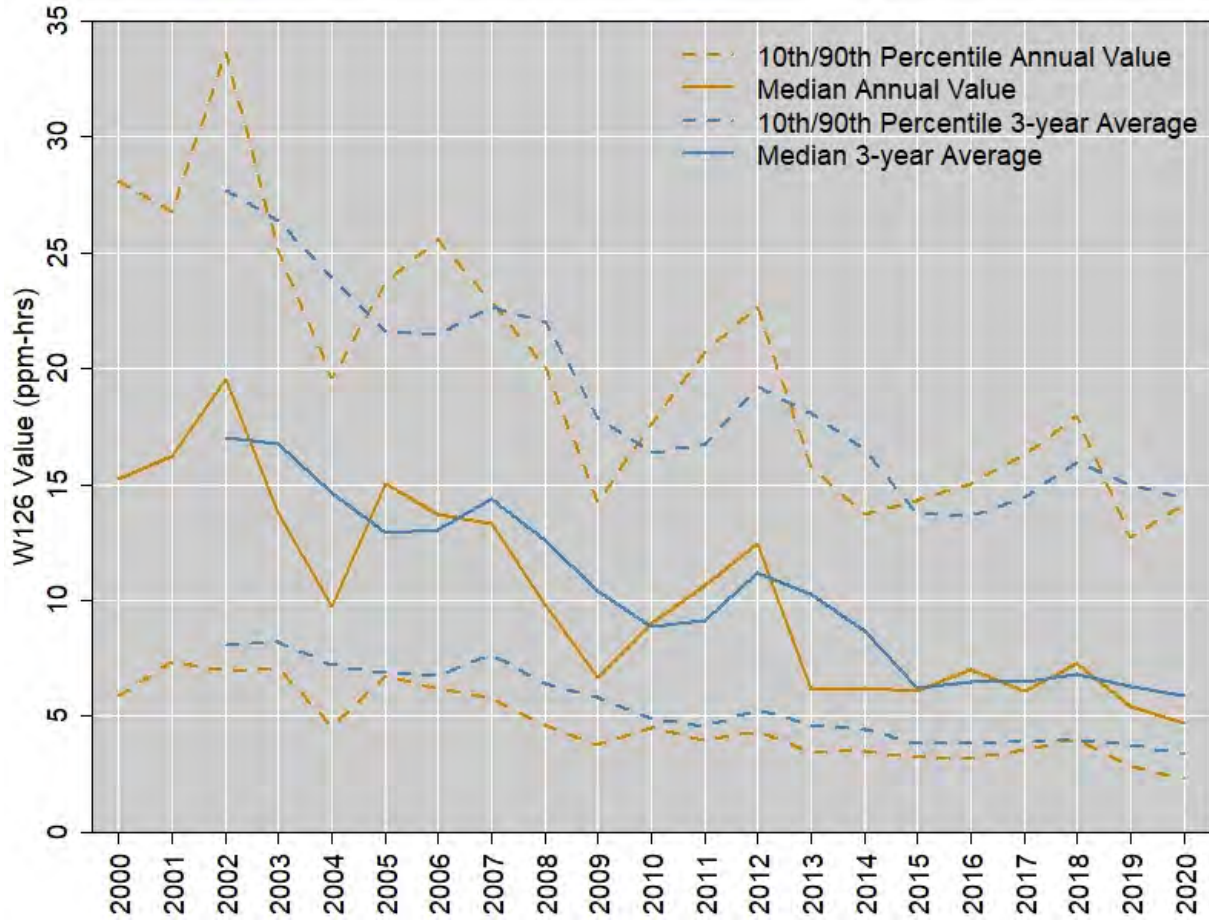
5

6 **Figure 4D-8. Annual W126 index values in ppm-hrs binned by 4th max metric values based**
 7 **on monitoring data for years 2000-2020.** Boxes show 25th, 50th, and 75th
 8 percentiles, whiskers extend to the 1st and 99th percentiles, and points below the 1st
 9 percentile or above the 99th percentile are represented by dots.

10 **4D.3.2.2 Trends in W126 Metric**

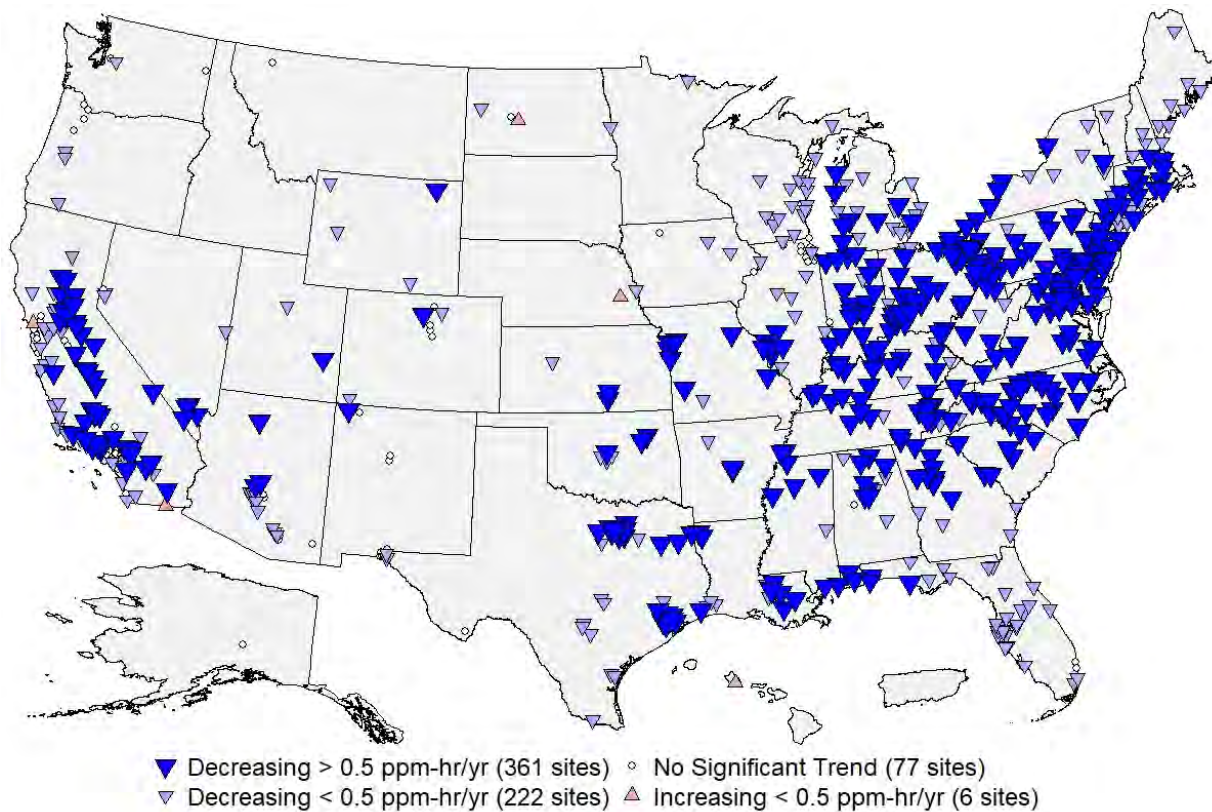
11 Figure 4D-9 below shows national trends in both the annual W126 index and the 3-year
 12 W126 metric based on the monitoring sites reporting data for the full period. Most notably, the

1 figure shows decreasing trends in W126 metric values, with the median value decreasing by
 2 about 65% from 2002 to 2020. The annual W126 index shows considerable year-to-year
 3 variability, with the median value sometimes increasing or decreasing by up to a factor of two
 4 from one year to the next, while the 3-year average is less impacted by this inter-annual
 5 variability, resulting in a smoother trend line.



6
 7 **Figure 4D-9. National trends in annual W126 index values (2000-2020) and W126 metric**
 8 **values (2002-2020).**

9 Figure 4D-10 shows a map of the site-level trends in the W126 metric values from 2000-
 10 2002 to 2018-2020. According to Figure 4D-10, nearly 88% of U.S. monitoring sites
 11 experienced significant decreases in W126 over this period, especially in the eastern U.S. and
 12 California where many O₃ monitoring sites saw decreases of 0.5 ppm-hr/yr or more. Many
 13 locations in the western U.S. experienced little or no change over this period. Only six monitors
 14 in disparate locations showed significant increasing trends in the W126 metric during the 2002-
 15 2020 period.



1
2 **Figure 4D-10. Map of trends in W126 metric values at U.S. O₃ monitoring sites from 2000-**
3 **2002 to 2018-2020.**

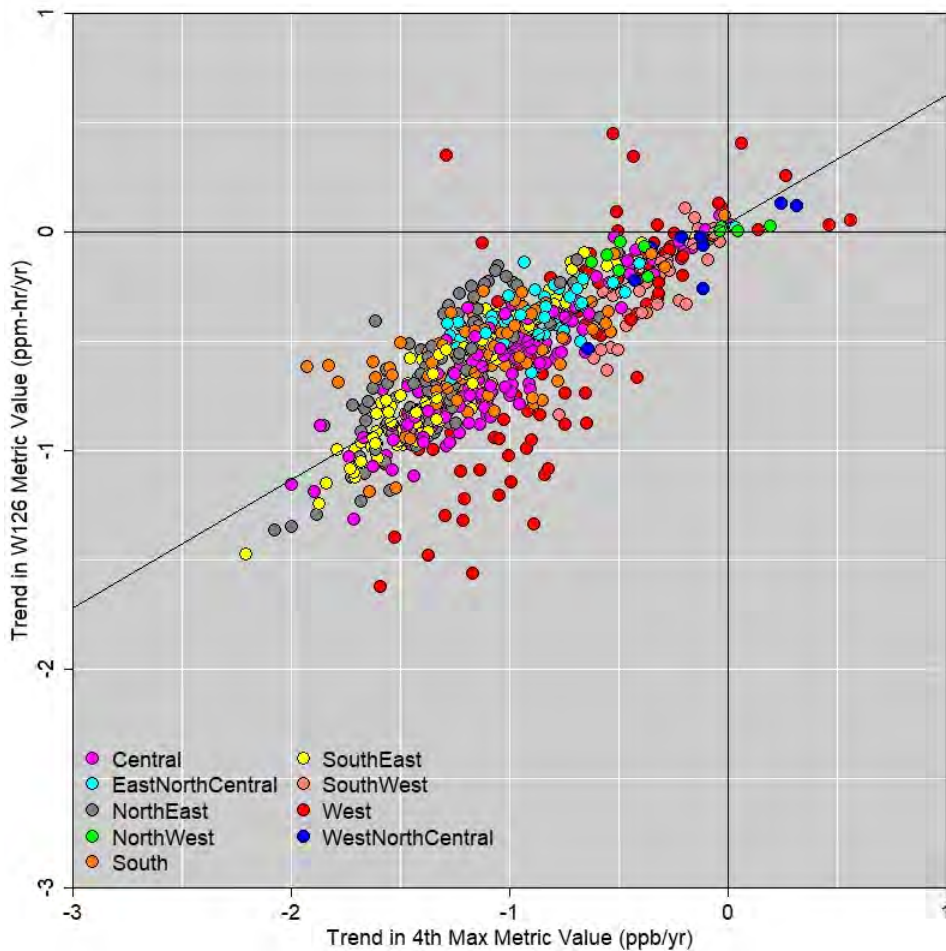
4 **4D.3.2.3 Comparison of Trends in the 4th Max and W126 Metrics**

5 Figure 4D-11 shows a scatter plot comparing the trends in the 4th max metric values (x-
6 axis, ppb/yr) to the trends in the W126 metric values (y-axis, ppm-hr/yr). These trends are
7 calculated using the Thiel-Sen estimator as in Figure 4D-10. The relationship between the trends
8 in the two metrics was linear and positive (Pearson correlation coefficient $R = 0.81$), meaning a
9 decrease in the 4th max metric is usually accompanied by a decrease in the W126 metric. The
10 slope of the regression line shown in Table 4D-12 indicates that, on average, there was a change
11 of approximately 0.59 ppm-hr in the W126 metric values per unit ppb change in the 4th max
12 metric values.

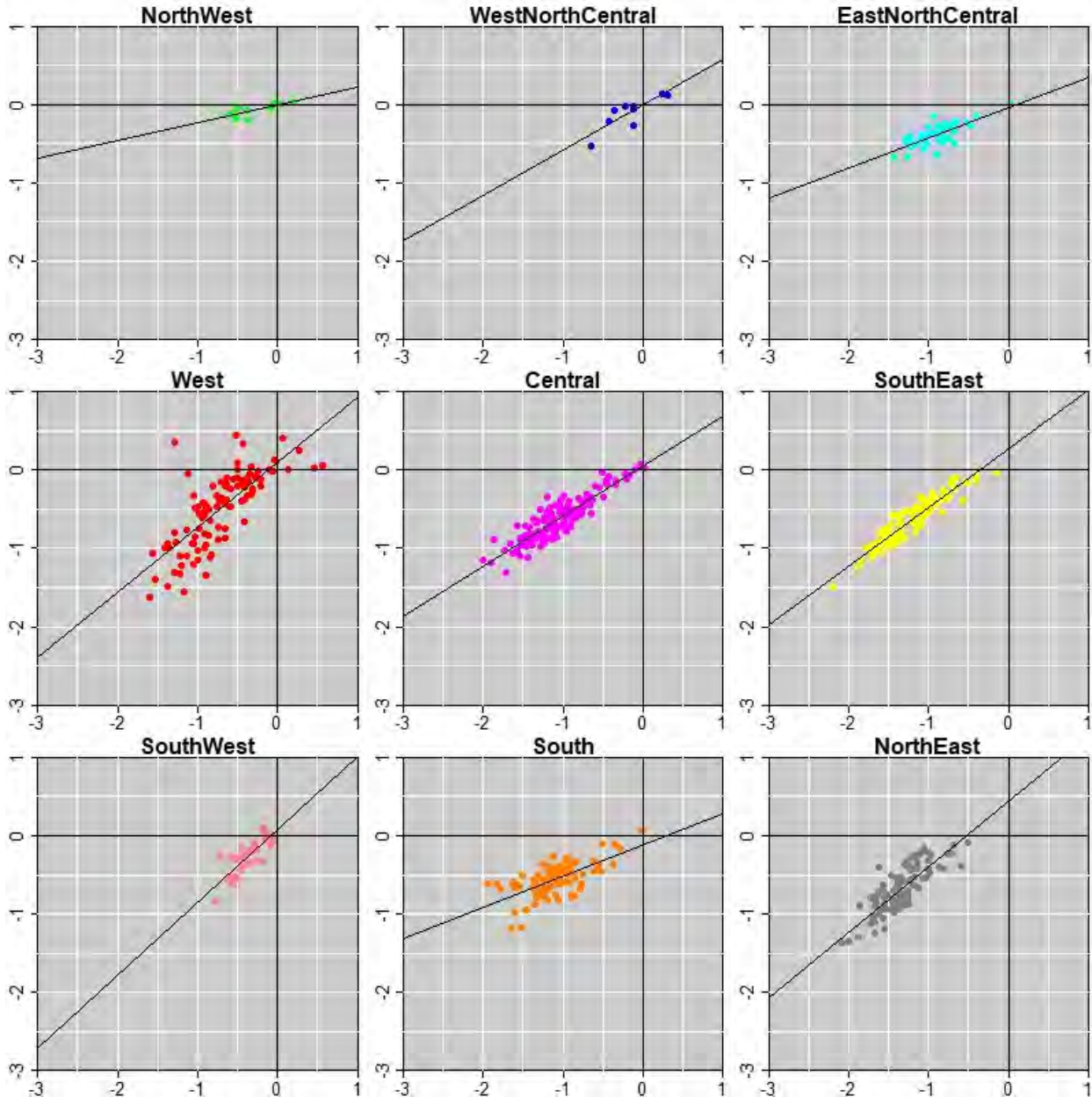
13 Figure 4D-12 shows scatter plots comparing the trends in the 4th max metric values (x-
14 axis, ppb/yr) to the trends in the W126 metric values (y-axis, ppm-hr/yr) in each NOAA climate
15 region and the associated regression lines fit using the sites within each region. Table 4D-12
16 provides some summary statistics based on the regional trends comparisons. Figure 4D-12 and
17 Table 4D-12 show that the positive, linear relationship between the trends in the 4th max metric
18 values and the trends in the W126 metric values persists within each region, with Pearson
19 correlation coefficients ranging from 0.65 to 0.94. The regression lines shown in Figure 4D-12

1 with slopes listed in Table 4D-12 indicate that the Southwest region, which had the greatest
2 potential for sites having higher W126 metric values relative to their 4th max metric values, also
3 exhibited the greatest response in the W126 metric values per unit change in the 4th max metric
4 values. In Figure 4D-11 and Figure 4D-13 (as well as the West region panels in Figure 4D-12
5 and Figure 4D-14), there appear to be three sites in the West region with an increasing trend in
6 the W126 metric (slope > 0.3) and a decreasing trend in the 4th max metric (slope < -0.4). These
7 three sites are all located downwind of Los Angeles, CA and generally have 4th max metric
8 values of 100 ppb or greater, along with W126 metric values in the 30-50 ppm-hr range.

9 Figure 4D-13, Figure 4D-14 and Table 4D-13 present information similar to that shown
10 in Figure 4D-11, Figure 4D-12 and Table 4D-12, respectively, except that trends in annual W126
11 index values are presented instead of W126 metric values. The figures show that the same
12 general pattern occurs when comparing annual W126 index values to the 4th max metric values
13 as was seen for the W126 metric values. There is slightly more variability in the relationship, as
14 can be seen from the slight increase in scatter in the figures and the slightly lower correlation
15 values shown in Table 4D-12 as compared to Table 4D-11.



1 **Figure 4D-11. Scatter plot comparing trends in 4th max metric values (x-axis) to trends in**
2 **W126 metric values (y-axis).**



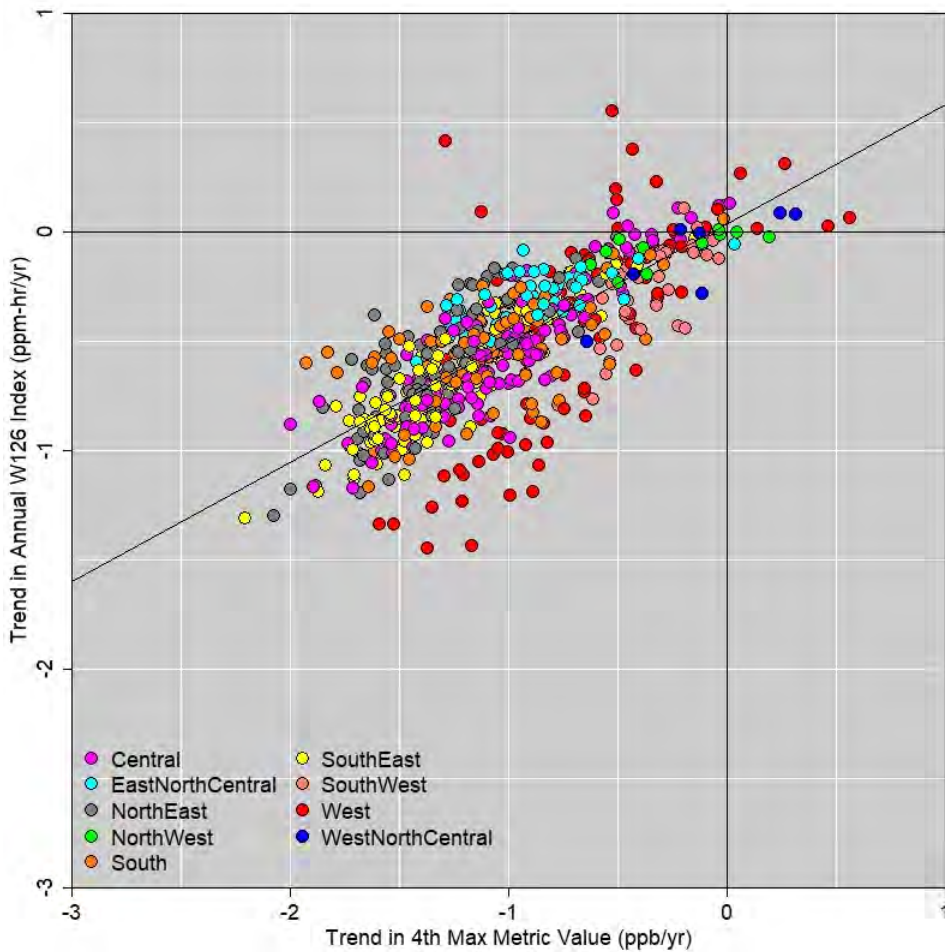
3
4 **Figure 4D-12. Scatter plots comparing the trends in 4th max metric values (x-axis, ppb) and**
5 **W126 metric values (y-axis, ppm-hrs) based on O₃ monitoring sites within**
6 **each of the nine NOAA climate regions.**

7

1 **Table 4D-12. Summary statistics based on regional comparisons of trends in 4th max metric**
 2 **values to trends in W126 metric values.**

NOAA Climate Region	Number of O ₃ Sites	Mean Trend in 4th Max Metric Value (ppb/yr)	Mean Trend in W126 Metric Value (ppm-hr/yr)	Regression Slope	Pearson Correlation Coefficient
Central	149	-1.06	-0.63	0.64	0.90
East North Central	45	-0.92	-0.38	0.39	0.75
Northeast	104	-1.33	-0.67	0.84	0.81
Northwest	12	-0.25	-0.06	0.23	0.79
South	83	-1.09	-0.55	0.40	0.65
Southeast	115	-1.22	-0.64	0.75	0.94
Southwest	39	-0.35	-0.24	0.93	0.83
West	102	-0.70	-0.49	0.83	0.76
West North Central	9	-0.16	-0.16	0.58	0.85
National	658	-1.00	-0.55	0.59	0.81

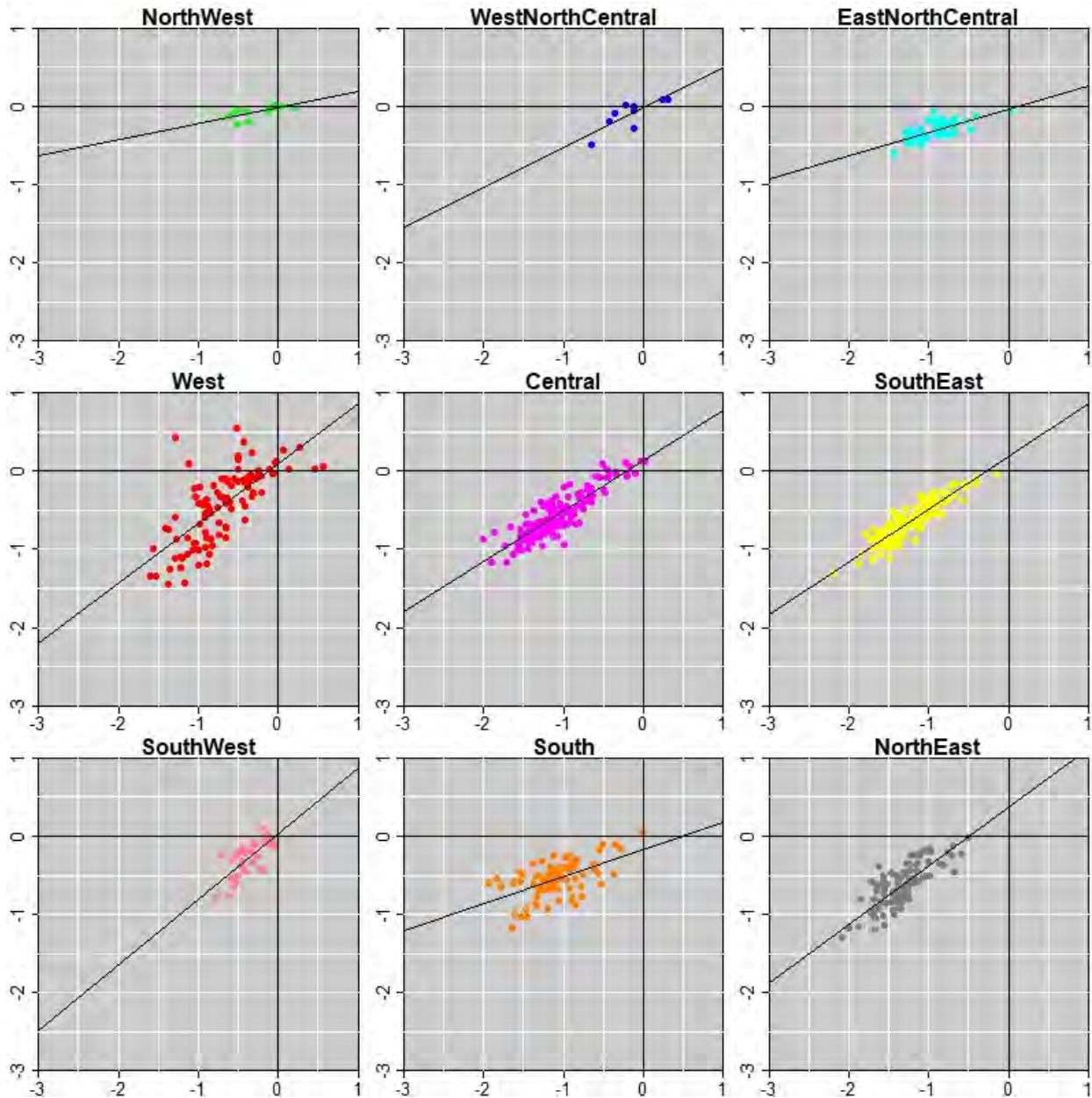
3



4

5 **Figure 4D-13. Scatter plot comparing trends in 4th max metric values (x-axis) and trends in**
 6 **annual W126 index values (y-axis).**

1



2

3 **Figure 4D-14. Scatter plots comparing trends in 4th max metric values (x-axis, ppb) to**
4 **trends in annual W126 index values (y-axis, ppm-hrs) based on O₃ monitoring**
5 **sites within each of the nine NOAA climate regions.**

6

1 **Table 4D-13. Summary statistics based on regional comparisons of trends in 4th max metric**
 2 **values and trends in annual W126 index values.**

NOAA Climate Region	Number of O ₃ Sites	Mean Trend in 4 th Max Metric Value (ppb/yr)	Mean Trend in Annual W126 Index Value (ppm-hr/yr)	Regression Slope	Pearson Correlation Coefficient
Central	149	-1.06	-0.56	0.64	0.88
East North Central	45	-0.92	-0.29	0.30	0.70
Northeast	104	-1.33	-0.61	0.75	0.79
Northwest	12	-0.25	-0.09	0.20	0.69
South	83	-1.09	-0.54	0.35	0.55
Southeast	115	-1.22	-0.61	0.67	0.91
Southwest	39	-0.35	-0.23	0.84	0.74
West	102	-0.70	-0.45	0.77	0.72
West North Central	9	-0.16	-0.15	0.50	0.80
National	658	-1.00	-0.50	0.55	0.78

3 **4D.3.2.4 W126 Metric Values in Federal Class I Areas**

4 Table 4D-14 below lists the 65 federal Class I areas for which we have monitoring data
 5 available for at least one 3-year period within the 2000-2020 period from a monitor located
 6 either within the area boundaries or within 15 km of the boundary. This summary table indicates
 7 the number of three-year periods for which the two metrics are available, the number of periods
 8 where 4th max metric values were at or below 70 ppb (i.e., when the current standard was met)
 9 and the range of the W126 metric values (which are also 3-year averages) during those periods.
 10 In total, the table is summarizing the 980 combinations of Class I areas and 3-year periods of
 11 which 589 have a 4th max metric value at or below 70 ppb and 391 have a 4th max metric value
 12 above 70 ppb. In the most recent period (2018-2020), of the 56 areas for which we have
 13 monitors, 47 sites have 4th max metric values at or below 70 ppb.

14 Table 4D-15 lists the Class I areas with the highest W126 metric values when the 4th max
 15 metric value is at or below 70 ppb. Among areas with a 4th max metric value at or below 70 ppb
 16 during any of the 3-year periods from 2000 to 2020, five areas (all located in the Southwest
 17 region) had one or more W126 metric values above 17 ppm-hrs, with the highest W126 metric
 18 values equal to 19 ppm-hrs and the highest annual W126 index values equal to 23 ppm-hrs, when
 19 rounded. All seven instances where a Class I area observed a 4th max metric value at or below 70
 20 ppb and a W126 metric value above 17 ppm-hrs occurred prior to 2011. This contrasts with the
 21 much higher values observed in Class I areas when the current standard is not met (Table 4D-
 22 17). In the 2018-2020 period, the W126 metric values range up to 41 ppm-hrs at sites in Class I
 23 areas when the standard is not met, with higher values in the historical Class I dataset.

24 Figure 4D-15 shows the distribution of annual W126 index values in Class I areas during
 25 3-year periods with different 4th max metric values. The full distribution of annual W126 index

1 values, including the minimum and maximums, increase with increasing 4th max metric values.
2 For example, the 99th percentile increases from about 20 ppm-hrs or lower to higher than 25
3 ppm-hrs for 4th max metric values at and below 70 ppb compared to 4th max metric values above
4 70 ppb. As indicated by Table 4D-15, the 3-year periods with the highest W126 metric values
5 occurring for 4th max metric values at or below 70 ppb occurred in the earlier years of the dataset
6 (2000-2010).

7 Table 4D-16 summarizes the occurrence of relatively higher annual W126 index values
8 in Class I areas during 3-year periods when the 4th max metric value is at or below 70 ppb. This
9 figure summarizes the W126 metric (i.e., 3-year average of annual W126 index values), as well
10 as maximum annual W126 index values in each 3-year period meeting the current standard. For
11 all instances of an area and 3-year period with a maximum annual W126 index value above 19
12 ppm-hrs, Figure 4D-16 illustrates the variation among the annual W126 index values and the
13 extent to which they differ from the 3-year average.

14 Finally, Table 4D-17 further documents the ranges of W126 metric values occurring
15 during periods when the 4th max metric value was above 70 ppb, indicating the extent to which
16 the current standard appears to be controlling the W126 metric.

17

1 **Table 4D-14. W126 metric values in Class I areas with 4th max metric values at or below 70**
 2 **ppb (2000-2020).**

NOAA Region (number of Class I areas ¹ , number of states ¹ with an area in region)	State	Area Name ²	Number of 3- year periods with data	Number of 3- year periods with 4 th max ≤ 70 ppb	Range of W126 Metric Values when 4 th max ≤ 70 ppb
Central (7, 4)	Kentucky	Mammoth Cave National Park ^{RM, VP}	19	9	5-11
	Tennessee	Great Smoky Mountains National Park ^{3 SM, YP, LP, VP, RM, BC, WP}	19	8	6-10
	West Virginia	Otter Creek Wilderness ^{VP, YP, RM, SM, BC, LP, WP}	18	11	4-8
EastNorthCentral (6, 3)	Michigan	Seney Wilderness Area ^{* QA, RM, SM, BC, WP}	17	9	4-6
	Minnesota	Boundary Waters Canoe Area Wilderness Area ^{* SM, QA, WP}	6	6	2-4
		Voyageurs National Park ^{QA, RM, WP}	15	15	2-6
NorthEast (6, 4)	Maine	Acadia National Park ^{RM, QA, SM, WP}	19	8	4-5
	New Hampshire	Great Gulf Wilderness Area ^{* WP}	16	16	3-8
	New Jersey	Brigantine Wilderness Area ^{* BC}	18	7	4-8
NorthWest (29, 4)	Idaho	Craters of the Moon Wilderness Area ^{* DF, QA}	13	13	6-13
	Washington	Alpine Lakes Wilderness ^{* DF, PP}	19	17	2-6
		Mount Rainer National Park, ^{DF}	17	16	2-6
		North Cascades National Park ^{* PP, DF, RA}	3	3	1-2
		Olympic National Park ^{DF, RA}	8	8	1-6
Alaska	Denali National Park ^{QA} (Formerly Mt. McKinley Nat Pk)	19	19	2-4	
South (6, 4)	Arkansas	Caney Creek Wilderness Area [*]	14	8	4-7
		Upper Buffalo Wilderness Area ^{* SM}	19	13	3-8
	Texas	Big Bend National Park ^{QA, DF, PP}	18	15	6-13
SouthEast (16, 6)	Alabama	Sipsey Wilderness ^{* WP, RM, SM, YP, LP, VP}	6	1	11
	Florida	St. Marks Wilderness Area [*]	17	12	4-11
	Georgia	Cohutta Wilderness Area ^{* WP, VP, YP}	19	8	4-6
	North Carolina	Great Smoky Mountains National Park ^{* SM, YP, LP, VP, RM, BC, WP}	See Tennessee for monitor with highest design value (4 th max)		
		Linville Gorge Wilderness Area ^{* VP, WP, RM, YP}	19	15	5-11
		Shining Rock Wilderness Area [*]	19	12	5-10
	South Carolina	Cape Romain Wilderness [*]	17	10	3-8
Virginia	James River Face Wilderness ^{* WP}	19	13	3-10	
	Shenandoah National Park ^{WP, VP, QA, BC, RM, SM, YP}	19	8	5-11	
SouthWest (38, 4)	Arizona	Chiricahua National Monument ^{DF, PP}	19	11	11-17
		Grand Canyon National Park ^{DF, PP, QA}	19	11	10-19
		Mazatzal Wilderness Area ^{DF, PP}	19	2	15

NOAA Region (number of Class I areas ¹ , number of states ¹ with an area in region)	State	Area Name ²	Number of 3- year periods with data	Number of 3- year periods with 4 th max ≤ 70 ppb	Range of W126 Metric Values when 4 th max ≤ 70 ppb
		Petrified Forest National Park	11	11	11-17
		Saguaro Wilderness Area* 2 DF, PP	19	7	12-15
		Superstition Wilderness Area* PP	19	1	13
		Yavapai Reservation* OA, PP, DF	4	4	10-15
	Colorado	Maroon Bells-Snowmass Wilderness. Area* OA, DF	14	14	11-19
		Mesa Verde National Park* PP, DF	19	17	11-18
		Rocky Mountain National Park* DF, PP, OA	19	5	13-15
		Weminuche Wilderness Area* DF, PP	8	3	13-18
	New Mexico	San Pedro Parks Wilderness* PP, DF	5	5	11-14
	Utah	Canyonlands National Park PP, DF	18	12	10-15
Zion National Park* DF, PP, OA		13	8	11-18	
West (32, 3)	California	Agua Tibia Wilderness* DF	6	0	-
		Cucamonga Wilderness Area* DF, PP	19	0	-
		Desolation Wilderness Area* PP	8	4	8-13
		Joshua Tree Wilderness Area*	19	0	-
		Kaiser Wilderness Area*	1	0	-
		Lassen Volcanic National Park DF, PP	19	13	7-14
		Pinnacles Wilderness Area*	19	10	7-10
		San Gabriel Wilderness Area* DF, PP	19	0	-
		San Geronimo Wilderness Area* PP, OA	15	0	-
		San Jacinto Wilderness Area* PP	19	0	-
		San Rafael Wilderness Area*	19	11	5-9
		Sequoia National Park PP, OA, DF	19	0	-
		Ventana Wilderness Area*	19	19	2-4
	Yosemite National Park DF, PP, OA	19	0	-	
Hawaii	Hawaii Volcanoes National Park	2	2	0	
WestNorthCentral (26, 4)	Montana	Gates of the Mountain Wilderness Area*	8	8	3-6
		Glacier National Park OA, PP, DF	19	19	2-3
		Northern Cheyenne Reservation*	9	9	3-5
	North Dakota	Lostwood Wilderness*	15	15	4-5
		Theodore Roosevelt National Park ² PP	18	18	4-7
	South Dakota	Badlands Wilderness*	13	13	3-12
Wind Cave National Park PP		12	12	5-15	

NOAA Region (number of Class I areas ¹ , number of states ¹ with an area in region)	State	Area Name ²	Number of 3- year periods with data	Number of 3- year periods with 4 th max ≤ 70 ppb	Range of W126 Metric Values when 4 th max ≤ 70 ppb
	Wyoming	Bridger Wilderness*	15	14	9-16
		Grand Teton National Park ^{DF, OA}	7	7	5-8
		Yellowstone National Park ^{DF, OA}	19	19	6-11

*The monitoring site is outside of the area but within 15 km of the area boundary.

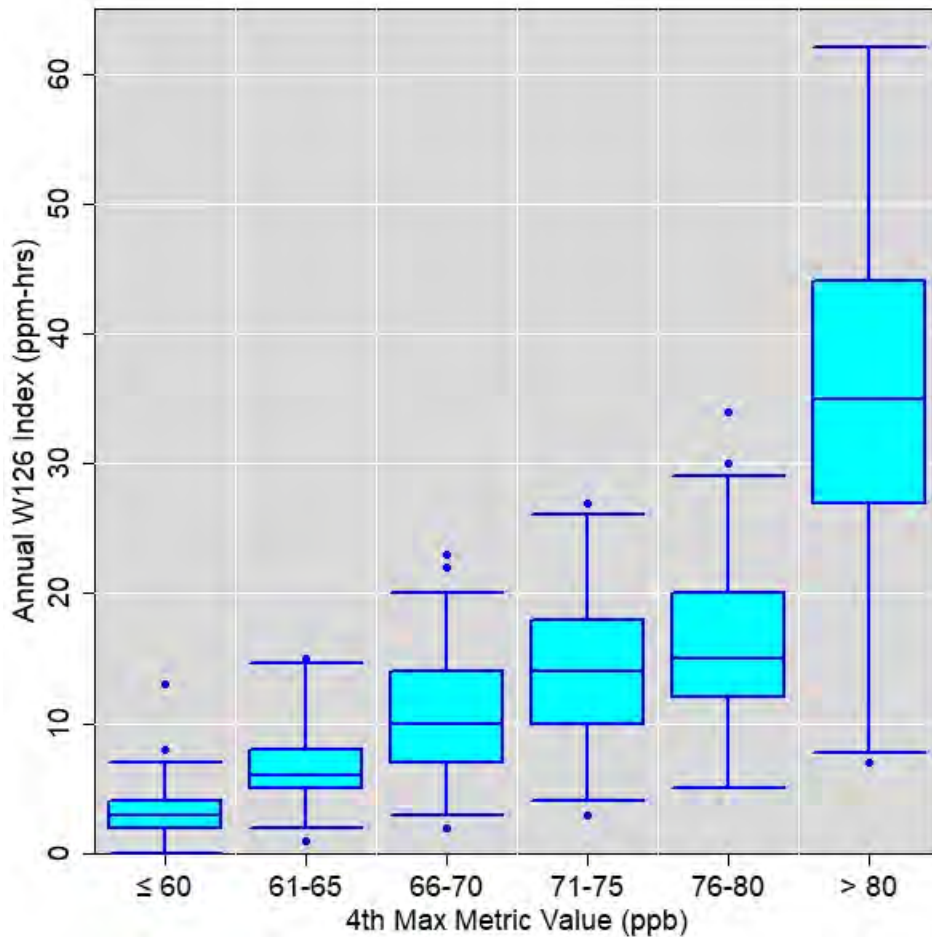
¹ Areas are counted in all regions and states with a Class I area.

² The 2-letter superscripts associated with some area names are abbreviations for species documented to be present in the area for which there is an established exposure-response function described in Appendix 4A: OA=Quaking Aspen, BC=Black Cherry, C=Cottonwood, DF=Douglas Fir, LP=Loblolly Pine, PP=Ponderosa Pine, RM=Red Maple, SM=Sugar Maple, VP=Virginia Pine, YP=Yellow (Tulip) Poplar. Sources include www.NPS.gov, www.inaturalist.org/guides, www.fs.usda.gov, www.msjnha.org/trees, www.wilderness.net

³ This area has two monitors; it is represented by the one with consistently higher values.

1
2 **Table 4D-15. Highest W126 metric values occurring in Class I areas when the 4th max**
3 **metric value is at or below 70 ppb (2000-2020).**

Class I Area	State/County	4 th max Range (ppb)	3-year Periods	W126 Metric Range (ppm-hrs)
Areas with W126 metric values above 17				
Grand Canyon National Park	AZ/Coconino	70	2006-2008	19
Maroon Bells-Snowmass Wilderness	CO/Gunnison	70	2000-2002, 2001-2003, 2002-2004	18-19
Mesa Verde National Park	CO/Montezuma	70	2006-2008	18
Weminuche Wilderness ^A	CO/LaPlata	70	2006-2008	18
Zion National Park ^B	UT/Washington	70	2008-2010	18
Areas with W126 metric values at or below 17 and above 15				
Bridger Wilderness	WY/Sublette	70	2001-2003	16
Canyonlands National Park	UT/San Juan	70	2006-2008	17
Chiricahua National Monument	AZ/Cochise	69	2006-2008	17
Maroon Bells-Snowmass Wilderness	CO/Gunnison	68	2003-2005, 2004-2006, 2005-2007	16
Mesa Verde National Park	CO/Montezuma	67-70	2000-2002, 2001-2003 2002-2004, 2003-2005 2011-2013	16-17
Petrified Forest National Park	AZ/Navajo	70	2011-2013, 2012-2014	16-17
Zion National Park	UT/Washington	70	2007-2009, 2009-2011 2012-2014	16-17
^A Monitoring site is 15.0 km from area.				
^B Monitoring site is 3.4 km from area.				



1
 2 **Figure 4D-15. Range of annual W126 index values in ppm-hrs observed at monitoring sites**
 3 **in Class I areas based on 2000-2020 monitoring data. Values are binned**
 4 **according to 4th max metric values in ppb.** Boxes show 25th, 50th, and 75th
 5 percentiles, whiskers extend to 1st and 99th percentiles, and points below the 1st
 6 percentile or above the 99th percentile are represented by dots.

7 **Table 4D-16. Summary of Class I area W126 index values when 4th max is at/below 70 ppb.**

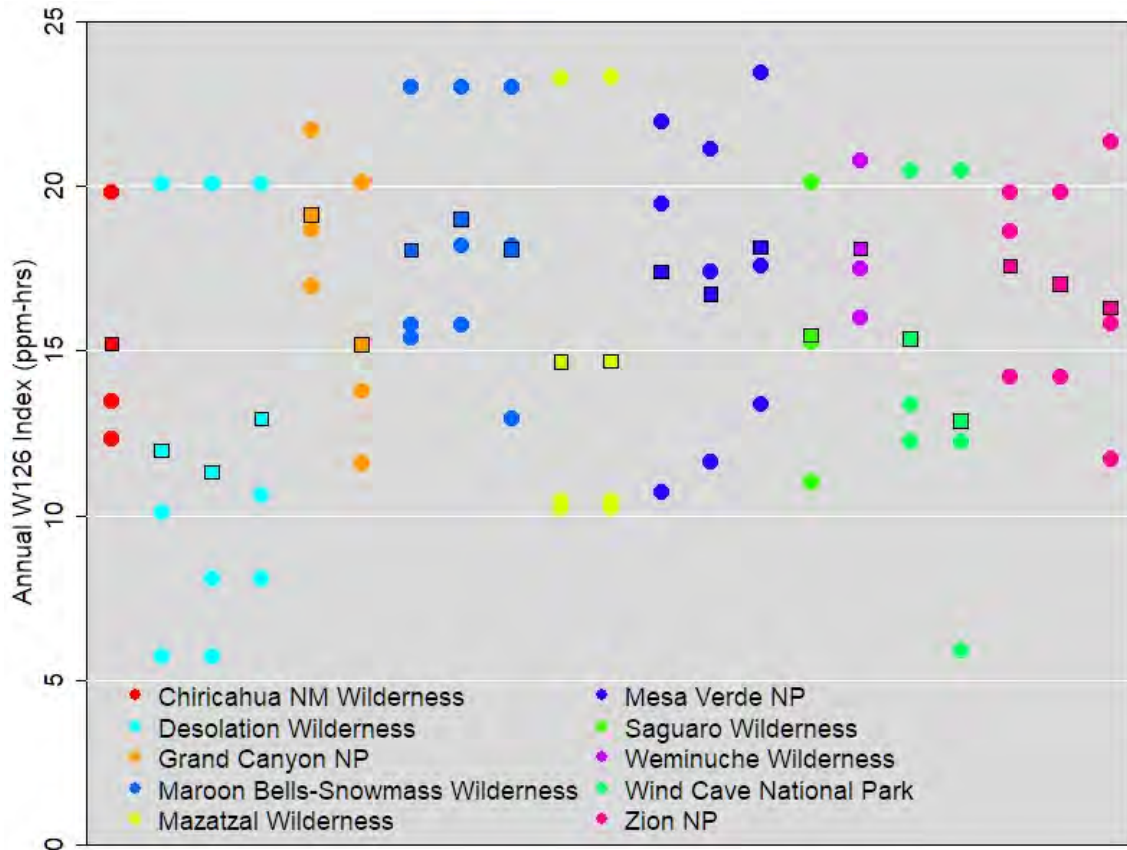
Time period	Total number of Area-DVs in time period (Number of areas)	Among areas with design values (DVs) ≤ 70 ppb					
		Number of area-DVs with W126 metric... (Number of areas)			Number of Area-DVs with maximum annual W126 index... (Number of areas)		
		>19	>17	≤ 17	>19	>17	≤ 17
2018-2020	57 (56)	0	0	47 (46)	0	2 (2) ^A	45 (44)
2000-2020	980 (65)	0	7 (5) ^A	589 (56)	15 (10) ^B	39 (18) ^C	531 (55)

^A These areas are all in the Southwest Region.

^B All but two of these areas are in Southwest Region; the other two are in West and West North Central Regions. The highest maximum annual W126 index value in dataset is 23 ppm-hrs of which there are four occurrences, all from prior to 2012 in SW. The most recent maximum annual W126 index value above 19 ppm-hrs is during 2012-2014 period (in 2012) when there are three (20, 20 and 21 ppm-hrs).

^C All but eight of these areas are in Southwest Region; the others are in West, South, Central and West North Central Regions.

1



2

3 **Figure 4D-16. Range of annual W126 index values observed in each 3-year period where a**
 4 **site in a Class I area had a design value meeting the current standard and had**
 5 **at least one annual W126 index value greater than 19 ppm-hrs. Each vertical**
 6 **column is one such 3-year period. Dots show annual W126 index values and**
 7 **squares show the W126 metric value.**

8

1 **Table 4D-17. W126 values in Class I areas with 4th max metric values above 70 ppb (2000-2020).**

NOAA Region	W126 metric >19			W126 metric >17			W126 metric >15			W126 metric ≤15		
	Number of areas	W126 metric range (ppm-hrs)	Annual W126 index range (ppm-hrs)	Number of areas	W126 metric range (ppm-hrs)	Annual W126 index range (ppm-hrs)	Number of areas	W126 metric range (ppm-hrs)	Annual W126 index range (ppm-hrs)	Number of areas	W126 metric range (ppm-hrs)	Annual W126 index range (ppm-hrs)
2018-2020												
Central	0	-	-	0	-	-	0	-	-	0	-	-
EastNorthCentral	0	-	-	0	-	-	0	-	-	0	-	-
NorthEast	0	-	-	0	-	-	0	-	-	0	-	-
NorthWest	0	-	-	0	-	-	0	-	-	0	-	-
South	0	-	-	0	-	-	0	-	-	0	-	-
SouthEast	0	-	-	0	-	-	0	-	-	0	-	-
SouthWest	0	-	-	0	-	-	2	17	11-21	0	-	-
West	7	20-41	14-47	8	19-41	12-47	8	19-41	12-47	0	-	-
WestNorthCentral	0	-	-	0	-	-	0	-	-	0	-	-
2000-2020												
Central	1	20-31	9-37	2	19-31	9-37	2	16-31	9-37	3	9-15	6-22
EastNorthCentral	0	-	-	0	-	-	0	-	-	1	6-8	4-11
NorthEast	1	20	17-24	1	20	17-24	1	17-20	9-24	2	5-14	4-18
NorthWest	0	-	-	0	-	-	0	-	-	2	6-7	3-10
South	0	-	-	0	-	-	0	-	-	3	7-14	5-18
SouthEast	1	22	18-25	1	22	18-25	3	16-22	10-25	8	7-15	5-22
SouthWest	8	20-33	9-39	10	18-33	9-39	10	16-33	9-39	5	11-15	7-24
West	12	20-61	14-74	13	18-61	12-74	13	16-61	12-74	4	10-15	8-20
WestNorthCentral	0	-	-	0	-	-	1	17	14-19	0	-	-

2

1 **4D.4 KEY LIMITATIONS AND UNCERTAINTIES**

2 This section summarizes key limitations and uncertainties associated with aspects of the
3 datasets analyzed in the preceding sections. The first section summarizes key limitations and
4 uncertainties associated with complete dataset monitoring sites in all U.S. locations (urban and
5 rural), which focus on patterns and relationships across all monitoring sites. The second section
6 concentrates on the Class I area sites. Overall, we recognize that while the datasets analyzed are
7 quite extensive (e.g., more than 1,100 sites covering all 50 states in the most recent 3-year
8 period), there are limitations and uncertainties associated with the spatial representation of O₃
9 monitoring sites in rural areas and Class I areas, specifically.

10 **Analyses of data for all U.S. monitoring sites:** Given that there has been a longstanding
11 emphasis on urban areas in the EPA's monitoring regulations, urban areas are generally well
12 represented in the U.S. dataset, with the effect being that the current dataset is more
13 representative of locations where people live than of complete spatial coverage for all areas in
14 the U.S., (i.e., the current dataset is more population weighted than geographically weighted).
15 Thus, the spatial coverage of the current O₃ monitoring network may be less representative of
16 natural areas which tend to be more sparsely populated. As O₃ precursor sources are also
17 generally more associated with urban areas, one impact of this may be a greater representation of
18 relatively higher concentration sites. One method that has been suggested to create a more
19 geographically representative dataset is the use of photochemical air quality modeling to estimate
20 concentrations. However, this approach has been found to present its own uncertainties with
21 regard to estimating annual W126 index values (U.S. EPA, 2014b, Appendix 4A), making it less
22 useful for the current analyses.

23 **Dataset for Class I monitoring sites:** A limitation of this dataset is that it includes sites
24 in only 65 of the 164 Class I areas in the U.S. The representation of states containing Class I
25 areas is somewhat greater, with monitoring sites in Class I areas in 29 of the 36 states that have
26 such an area. All nine NOAA climate regions are represented. As can be seen from Figure 4D-2,
27 sites outside of Class I areas in the states not represented (LA, MO, NV, OK, OR, VT, WI) have
28 W126 metric values at or below 13 ppm-hrs during the recent 3-year period (2018-2020). Across
29 the states represented in the dataset, the fraction of a given state's Class I areas included in the
30 dataset generally ranges from about a third to 100%. An exception to that is New Mexico, for
31 which a monitoring site is in or near only one of the nine Class I areas in the state. This contrasts
32 with neighboring Arizona, also in the Southwest region and for which more than half the Class I
33 areas are represented in the dataset.

1 **4D.5 SUMMARY**

2 The preceding sections present analyses based on 21 years of O₃ concentration data
3 reported at monitoring sites across the U.S. These analyses, intended to inform the review of the
4 current O₃ secondary standard, investigate spatial and temporal patterns in the W126 metric
5 using monitoring data from 2000 to 2020 and the extent of relationships between the W126
6 metric, annual W126 index values and design values for the current secondary O₃ standard (i.e.,
7 the 4th max metric). Further analyses of O₃ concentrations in or near federally protected
8 ecosystems known as Class I areas focus on examining the levels and distributions of levels of
9 the W126 metric and the annual W126 index occurring in such areas when the current secondary
10 standard is met and also when the current secondary standard is not met.

11 The analyses based on recent (2018-2020) data showed that about one in five U.S. sites
12 had 4th max metric values greater than the current standard level of 70 ppb. By contrast, only
13 about 1 in 14 U.S. sites had W126 metric values greater than 17 ppm-hrs, and about 1 in 9 U.S.
14 sites had W126 metric values greater than 13 ppm-hrs. There were O₃ monitors exceeding the
15 current standard level of 70 ppb in 6 of 9 climate regions, while only two regions, the West and
16 Southwest, had O₃ monitors with W126 metric values exceeding 13 ppm-hrs.

17 When examining the 4th max and W126 metrics in combination, the 2018-2020 data
18 showed that there were many U.S. O₃ sites with 4th max metric values exceeding the current
19 standard that had W126 metric values less than or equal to 17 ppm-hrs (128) and 13 ppm-hrs
20 (95). By contrast, there were relatively few sites meeting the current standard that had W126
21 metric values greater than 13 ppm-hrs (13); and there were no sites that had a W126 metric value
22 above 17 ppm-hrs. The 13 sites that met the current standard and had W126 metric values greater
23 than 13 ppm-hrs were located exclusively in the Southwest and West climate regions, whereas
24 the 95 sites that exceeded the current standard and had W126 metric values less than or equal to
25 13 ppm-hrs had a much broader geographic distribution.

26 Among O₃ monitoring sites in Federal Class I areas, few areas since 2000 have had 4th
27 max metric values meeting the current standard and W126 metric values above 17 ppm-hrs, the
28 most recent of which occurred during the 2012-2014 period. These instances are all in or near
29 Class I areas in the Southwest region, with the highest (19 ppm-hrs) occurring during the 2006-
30 2008 period.

31 The analysis of inter-annual variability shows that the distribution of annual W126 index
32 deviations from their respective 3-year averages generally increase with increasing W126 metric
33 values. For sites with W126 metric values below 20 ppm-hrs (e.g., focusing on W126 metric
34 values that have occurred with 4th max metric values at or below 70 ppb), the annual deviation
35 was generally within 5 ppm-hrs. Additionally, well over 99% of 4th max metric values meeting

1 the current standard were associated with annual W126 index values of less than or equal to 19
2 ppm-hrs.

3 The trends analysis showed that both W126 metric values and annual W126 index values
4 have generally decreased since 2000, with U.S. median W126 metric values decreasing by over
5 65%, from 17 ppm-hrs in 2002 to less than 6 ppm-hrs in 2020. A substantial number of U.S. sites
6 have experienced decreases of over 10 ppm-hrs in the past decade, particularly in the eastern
7 U.S.

8 The analysis comparing trends in the 4th max metric values and to trends in the W126
9 metric values based on data from 2000-2020 showed that there was a positive, linear relationship
10 between the per-year changes in the 4th max and W126 metrics. Nationally, the W126 metric
11 values decreased by approximately 0.6 ppm-hr per unit ppb decrease in the 4th max metric
12 values. This relationship varied across the NOAA climate regions. The Southwest and West
13 regions which showed the greatest potential for exceeding only the W126 levels of interest also
14 showed the greatest improvement in the W126 metric values per unit decrease in 4th max metric
15 values. This analysis indicates that W126 metric values in those areas not meeting the current
16 standard would be expected to decline as the 4th max metric values are reduced to meet the
17 current standard, consistent with the relationship shown in Figure 4D-11.

18

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APPENDIX 4E

2

**OZONE WELFARE EFFECTS AND RELATED ECOSYSTEM
SERVICES AND PUBLIC WELFARE ASPECTS**

3

1 **Table 4E-1. Ecosystem services and aspects of public welfare potentially affected by the**
 2 **different types of O₃ welfare effects.**

O ₃ Effect ^A	Aspect of Public Welfare Potentially Affected (Examples) ^B	Ecosystem Services ^C
Visible foliar injury	<ul style="list-style-type: none"> • Appearance and scenic beauty of forests wilderness areas, including federal, tribal, state, municipally protected areas • Quality of specific agricultural crops, plant leaf products • Appearance of plants in residential/commercial areas (ornamentals) 	Cultural Recreation Provisioning
Reduced vegetation growth	<ul style="list-style-type: none"> • Food, raw material, and unique biological material and product production • Shade provision • Quality of plants of cultural importance to Native American Tribes • Changes to national yield and prices 	Cultural Provisioning
Reduced plant reproduction		
Reduced yield and quality of agricultural crops		
Reduced productivity in terrestrial ecosystems		
<i>Reduced carbon sequestration in terrestrial systems</i>	<ul style="list-style-type: none"> • Regulation/control of climatological features and meteorological phenomena • Changes in pollution removal in urban areas 	Regulating Supporting
<i>Increased tree mortality</i>	<ul style="list-style-type: none"> • Regulation/control of wildfires • Regulation of erosion and soil stability • Decline of ecosystem services provided by trees (see Table 4E-2) 	Regulating Cultural Supporting Provisioning
<i>Alteration of terrestrial community composition</i>	<ul style="list-style-type: none"> • Intrinsic value of areas specially protected from anthropogenic degradation • Production of preferred species of timber • Preservation of unique or endangered ecosystems or species • Species diversity in protected areas 	Cultural Provisioning Supporting
Alteration of belowground biogeochemical cycles	<ul style="list-style-type: none"> • Soil quality • Soil nutrient cycling, decomposition, and availability • Carbon storage • Regulation of soil fauna and microbial communities • Water quality and resource management • Regulation of hydraulic flow 	Supporting Regulating
<i>Alteration of ecosystem water cycling</i>	<ul style="list-style-type: none"> • Water quality and resource management • Regulation of hydraulic flow 	Provisioning Regulating Supporting
<i>Altered of herbivore growth and reproduction</i>	<ul style="list-style-type: none"> • Food sources, habitat, and protection for native fauna 	Supporting Regulating
<i>Alteration of plant insect signaling</i>	<ul style="list-style-type: none"> • Plant-pollinator interactions • Timber and agricultural plant resistance to insect pest damage 	Supporting Provisioning
Radiative forcing and related climate effects	<ul style="list-style-type: none"> • Regulation/control of meteorological phenomena 	Regulating

NOTE: Sources include ISA (Appendix 8, Figure 8-1 and Table 8-1) and 2014 WREA (Section 5).

^A Effects identified as causally or *likely causally* related to O₃ (draft ISA, Appendices 8 and 9).

^B Examples provided in Costanza et al., 2017) and 2014 WREA, Section 5 (U.S. EPA, 2014)

^C Description of Ecosystem Services in 2013 ISA, Section 9.4.1.2 and in the 2014 WREA, Section 5.1:

- *Regulating*: Services of importance for human society such as carbon sequestration, climate and water regulation, protection from natural hazards such as floods, avalanches, or rock-fall, water and air purification, and disease and pest regulation.
- *Supporting*: The services needed by all the other ecosystem services, either indirectly or directly, such biomass production, production of atmospheric O₂, soil formation and retention, nutrient cycling, water cycling, biodiversity, and provisioning of habitat.
- *Provisioning*: Services that include market goods, such as food, water, fiber, and medicinal and cosmetic products
- *Cultural*: services that satisfy human spiritual and aesthetic appreciation of ecosystems and their components including recreational and other nonmaterial benefits

1 **Table 4E-2. Ecosystem services and specific uses of the 11 tree species with robust E-R**
 2 **functions for reduced growth.**

Tree Species	O ₃ Effect	Role in Ecosystems and Public Uses
Black Cherry <i>Prunus serotina</i>	Biomass loss, Visible foliar injury	Cabinets, furniture, paneling, veneers, crafts, toys; Cough remedy, tonic, sedative; Flavor for rum and brandy; Wine making and jellies; Food and habitat for songbirds, game birds, and mammals
Eastern White Pine <i>Pinus strobus</i>	Biomass loss	Commercial timber, furniture, woodworking, and Christmas trees; Medicinal uses as expectorant and antiseptic; Food and habitat for songbirds and mammals; Used to stabilize strip mine soils
Quaking Aspen <i>Populus tremuloides</i>	Biomass loss, Visible foliar injury	Commercial logging for pulp, flake-board, pallets, boxes, and plywood; Products including matchsticks, tongue depressors, and ice cream sticks; Valued for its white bark and brilliant fall color; Important as a fire break Habitat for variety of wildlife; Traditional native American use as a food source
Yellow (Tulip) Poplar <i>Liriodendron tulipifera</i>	Biomass loss, Visible foliar injury	Furniture stock, veneer, and pulpwood; Street, shade, or ornamental tree – unusual flowers; Food and habitat for wildlife; Rapid growth for reforestation projects
Ponderosa Pine <i>Pinus ponderosa</i>	Biomass loss, Visible foliar injury	Lumber for cabinets and construction; Ornamental and erosion control use; Recreation areas; Food and habitat for many bird species, including the red-winged blackbird, chickadee, finches, and nuthatches
Red Alder <i>Alnus rubra</i>	Biomass loss, Visible foliar injury	Commercial use in products such as furniture, cabinets, and millwork; Preferred for smoked salmon; Dyes for baskets, hides, moccasins; Medicinal use for rheumatic pain, diarrhea, stomach cramps – the bark contains salicin, a chemical similar to aspirin; Roots used for baskets; Food and habitat for mammals and birds – dam and lodge construction for beavers; Conservation and erosion control
Red Maple[^] <i>Acer rubrum</i>	Biomass loss	One of the most abundant and widespread trees in eastern U.S. Used for revegetation, especially in riparian buffers and landscaping, where it is valued for its brilliant fall foliage, some lumber and syrup production; Important wildlife browse food, especially for elk and white-tailed deer in winter, also leaves are important food source for some species of butterflies and moths.
Virginia Pine <i>Pinus virginiana</i>	Biomass loss, Visible foliar injury	Pulpwood, stabilization of strip mine spoil banks and severely eroded soils; Nesting for woodpeckers, food and habitat for songbirds and small mammals
Sugar Maple <i>Acer saccharum</i>	Biomass loss	Commercial syrup production; Native Americans used sap as a candy, beverage – fresh or fermented into beer, soured into vinegar and used to cook meat; Valued for its fall foliage and as an ornamental; Commercial logging for furniture, flooring, paneling, and veneer; Woodenware, musical instruments; Food and habitat for many birds and mammals
Loblolly Pine[*]	Biomass loss, visible foliar injury	Most important and widely cultivated timber species in the southern U.S.; Furniture, pulpwood, plywood, composite boards, posts, poles, pilings, crates, boxes, pallets. Also planted to stabilize eroded or damaged soils. It can be used for shade or ornamental trees, as well as bark mulch; Provides habitat, food and cover for white-tailed deer, gray squirrel, fox squirrel, bobwhite quail and wild turkey, red-cockaded woodpeckers, and a variety of other birds and small mammals. Standing dead trees are frequently used for cavity nests by woodpeckers.
Douglas Fir <i>Pseudotsuga menziesii</i>	Biomass loss	Commercial timber and used for Christmas trees; Medicinal uses, spiritual and cultural uses for several Native American tribes; Spotted owl habitat; Food and habitat for mammals including antelope and mountain sheep

Sources: 2014 WREA, USDA-NRCS (2013); Burns and Honkala, 1990).

[^]Red maple information from https://www.srs.fs.usda.gov/pubs/misc/ag_654/volume_2/silvics_v2.pdf

^{*}Loblolly pine use information from

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APPENDIX 4F

ANALYSIS OF THE N100 AND D100 OZONE CONCENTRATION METRICS AT U.S. AMBIENT AIR MONITORING SITES

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1 **4F.1 OVERVIEW**

2 This technical memorandum presents various analyses of ambient air monitoring data for
3 ozone (O₃) concentrations in the U.S. relating to the form and averaging time of the current
4 secondary standard and some metrics reported in environmental assessments. These metrics
5 include the W126-based cumulative exposure index, the N100 (number of hours at or above 100
6 ppb), and D100 (number of days with one or more hours at or above 100 ppb). The calculation of
7 these metrics is described in Section 4F.2 below. These analyses describe relationships between
8 the three environmental metrics and the design values for the current standard (the annual 4th
9 highest daily maximum 8-hour O₃ concentration, averaged over 3 consecutive years; hereafter
10 referred to as the “4th max metric”). The analyses presented here are an extension of analyses
11 that are presented Section 2.4.5, Appendix 2A, and Appendix 4D of the Policy Assessment for
12 the review (U.S. EPA, 2022).

13 **4F.2 DATA HANDLING**

14 **4F.2.1 Data Retrieval and Preparation**

15 Hourly O₃ concentration data were retrieved from the EPA’s Air Quality System (AQS,
16 <https://www.epa.gov/aqs>) database for 2,021 ambient air monitoring sites which operated
17 between 2000 and 2020. These data were used to calculate W126 and 4th max metric values for
18 each 3-year period from 2000-2002 to 2018-2020. Before calculating these metrics, some initial
19 processing was done on the hourly data. First, data collected using monitoring methods other
20 than federal reference or equivalent methods and data collected at monitoring sites not meeting
21 EPA’s quality assurance or other criteria in 40 CFR part 58 were removed from the analysis.
22 Second, data collected by multiple monitoring instruments operating at the same location were
23 combined according to Appendix U to 40 CFR Part 50. Finally, data were combined across pairs
24 of monitoring sites approved for such combination by the EPA Regional Offices. The final
25 hourly O₃ concentration dataset contained 1,808 monitoring sites.

26 **4F.2.2 Derivation of the Metrics**

27 The 4th max metric values were calculated according to the data handling procedures in
28 Appendix U to 40 CFR part 50. First, moving 8-hour averages were calculated from the hourly
29 O₃ concentration data for each site. For each 8-hour period, an 8-hour average value was
30 calculated if there were at least 6 hourly O₃ concentrations available. Each 8-hour average was
31 stored in the first hour of the period (e.g., the 8-hour average from 12:00 PM to 8:00 PM is
32 stored in the 12:00 PM hour). Daily maximum 8-hour average values were found using the 8-
33 hour periods beginning from 7:00 AM to 11:00 PM each day. These daily maximum values were
34 used if at least 13 of the 17 possible 8-hour averages were available, or if the daily maximum

1 value was greater than 70 parts per billion (ppb). Finally, the annual 4th highest daily maximum
2 value was found for each year, then averaged across each consecutive 3-year period to obtain the
3 final set of 4th max metric values in units of ppb. Any decimal digits in these values were
4 truncated for applications requiring direct comparison to a 4th max level (e.g., Table 1),
5 otherwise, all decimal digits were retained. The 4th max metric values were considered valid if
6 daily maximum values were available for at least 90% of the days in the O₃ monitoring season
7 (defined in Appendix D to 40 CFR part 58) on average across the three years, with a minimum of
8 75% of the days in the O₃ monitoring season in any calendar year. In addition, 4th max metric
9 values were considered valid if they were greater than the 4th max levels to which they were
10 being compared.

11 The W126 metric values were calculated using the hourly O₃ concentration data in parts
12 per million (80 FR 65374, October 26, 2015). For daytime hours (defined as the 12-hour period
13 from 8:00 AM to 8:00 PM Local Standard Time each day), the hourly concentration values at
14 each O₃ monitoring site were weighted using the following equation:

$$15 \quad \text{Weighted O}_3 = \text{O}_3 / (1 + 4403 * \exp(-126 * \text{O}_3)).$$

16 These weighted values were summed over each calendar month, then adjusted for
17 missing data (e.g.; if 80% of the daytime hourly concentrations were available, the sum would be
18 multiplied by 1/0.8 = 1.25) to obtain the monthly W126 index values. Monthly W126 index
19 values were not calculated for months where fewer than 75% of the possible daytime hourly
20 concentrations were available. Next, moving 3-month sums were calculated from the monthly
21 index values, and the highest of these 3-month sums was determined to be the annual W126
22 index. Three-month periods spanning multiple years (e.g., November to January, December to
23 February) were not considered in these calculations. The annual W126 index values were
24 averaged across each consecutive 3-year period to obtain the final W126 metric values, with
25 units in parts per million-hours (ppm-hrs). The W126 metric values were rounded to the nearest
26 unit ppm-hr for applications requiring direct comparison to a W126 level (e.g., Table 1),
27 otherwise, all decimal digits were retained. For consistency with the 4th max metric calculations,
28 the W126 metric values were considered valid if hourly O₃ concentration values were available
29 for at least 90% of the daytime hours during the O₃ monitoring season on average across the
30 three years, with a minimum of 75% of the daytime hours during the O₃ monitoring season in
31 any calendar year. For consistency with the 4th max metric calculations, the W126 metric values
32 were considered valid if they were greater than the W126 levels to which they were being
33 compared.

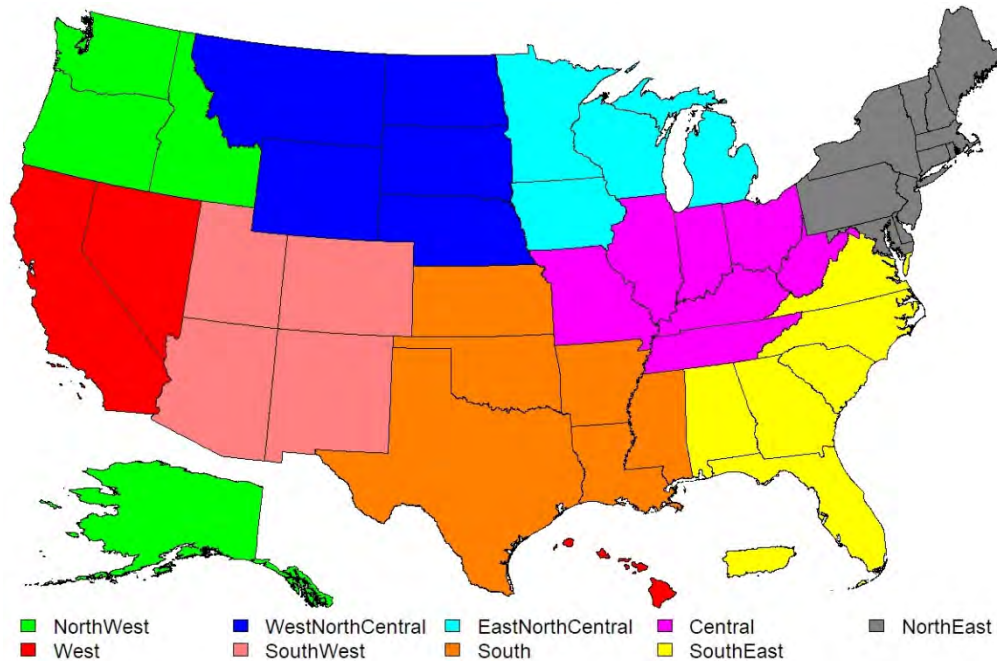
34 The N100 metric was calculated as the maximum number of hours with an hourly O₃
35 concentration of 100 ppb or greater in the three consecutive calendar months yielding the highest

1 number in a given year. Similarly, the D100 metric was calculated as the maximum number of
2 days with at least one hourly O₃ concentration of 100 ppb or greater in the three consecutive
3 calendar months yielding the highest number in a given year. These metrics were considered
4 valid if the annual data completeness rate for the O₃ monitoring season was at least 75 percent.

5 In summary, the “4th max metric” refers to the average of the 4th highest daily maximum
6 8-hour averages in three consecutive years and the “W126 metric” refers to the average of annual
7 W126 index values (“annual” or “single-year” W126 index) over three years. Where a single-
8 year value is intended, it is referred to as annual or single-year. In the final dataset, 1,757 of the
9 1,808 O₃ monitoring sites had sufficient data to calculate valid annual 4th max, W126, N100 and
10 D100 values for at least one year between 2000 and 2020. The number of sites with valid annual
11 metric values ranged from 1,102 in 2000 to 1,225 in 2014, and 586 sites had valid annual metric
12 values in all 21 years. Additionally, 1,578 of the 1,808 O₃ monitoring sites had sufficient data to
13 calculate valid 4th max and W126 metric values for at least one 3-year period between 2000-
14 2002 and 2018-2020. The number of sites with valid 4th max and W126 metric values ranged
15 from a low of 992 in 2000-2002 to a high of 1,118 in 2015-2017, and 510 sites had valid 4th max
16 and W126 metric values for all nineteen 3-year periods.

17 **4F.2.3 Assignment of Monitoring Sites to NOAA Climate Regions**

18 In order to examine regional differences, many of the further analyses were stratified into
19 the nine NOAA climate regions (Karl and Koss, 1984), which are shown in Figure 4F-1. Since
20 the NOAA climate regions only cover the contiguous U.S., Alaska was added to the Northwest
21 region, Hawaii was added to the West region, and Puerto Rico was added to the Southeast
22 region.



1

2 **Figure 4F-1. Map of the nine NOAA climate regions.**

3 **4F.3 RESULTS**

4 **4F.3.1 National Analysis Using Recent Air Quality Data**

5 This section presents various results based on the annual 4th max, W126, N100, and D100
 6 metrics as well as the 3-year average 4th max and W126 metrics¹ for the 2018-2020 period.
 7 Figure 4F-2 and Figure 4F-3 show maps of the average annual N100 and D100 values,
 8 respectively, at sites with valid 4th max metric values (design values) for 2018-2020. About 74%
 9 of the O₃ monitoring sites did not have any hourly concentrations at or above 100 ppb in 2018-
 10 2020, and an additional 18% of the sites had an average of one day or less per year where hourly
 11 O₃ concentrations reached 100 ppb or more. Sites with more than one day per year where hourly
 12 O₃ concentrations reached 100 ppb or more were generally located near large urban areas, with
 13 the most extreme values located downwind of Los Angeles, CA.

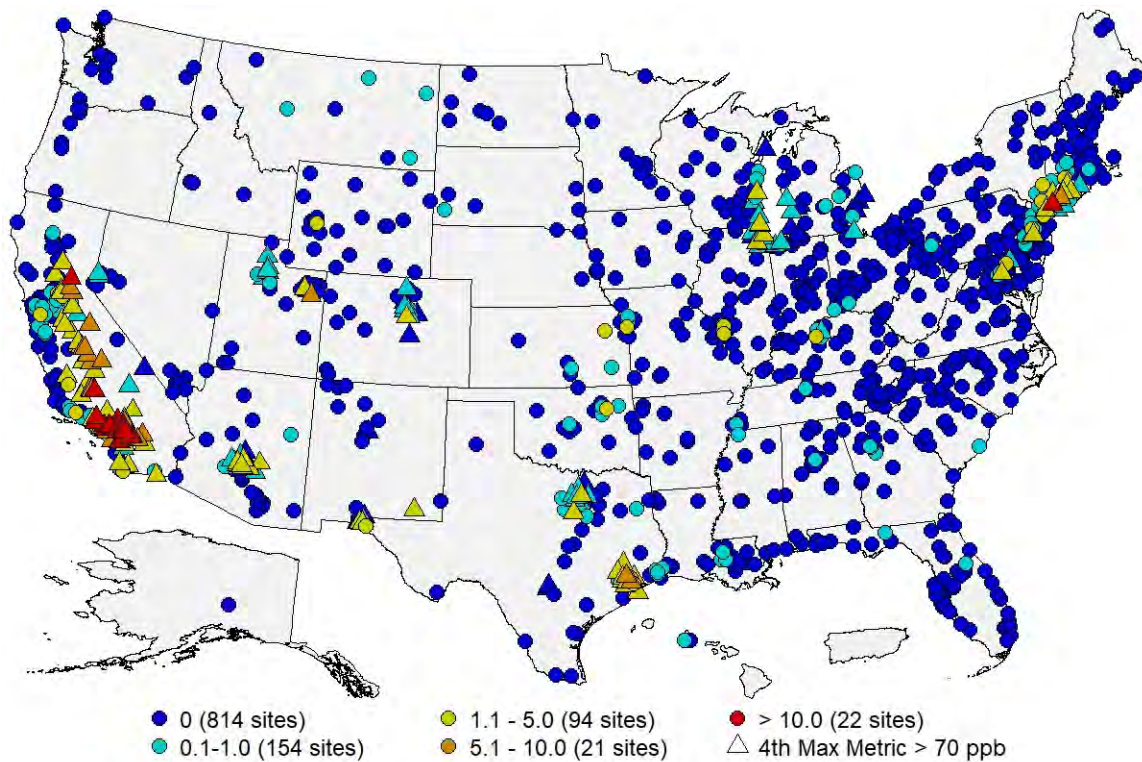
14 Figure 4F-4 and Figure 4F-5 show scatter plots comparing the 4th max metric values (x-
 15 axis) at each O₃ monitoring site for the 2018-2020 period to their respective N100 and D100
 16 values (y-axis) for 2018, 2019, and 2020. Similarly, Figure 4F-6 and Figure 4F-7 show scatter
 17 plots comparing W126 metric values (x-axis) at each O₃ monitoring site for the 2018-2020
 18 period to their respective N100 and D100 values (y-axis) for 2018, 2019, and 2020. For sites
 19 meeting the current standard (i.e., 4th max metric value ≤ 70 ppb), the hourly O₃ concentrations

¹ As defined in section 4F.2.2 above, the term “W126 metric” refers to the 3-year average W126 index. The term “annual W126” is used in reference to single-year W126 index values.

1 reached 100 ppb or more for at most ten hours across four distinct days. By contrast, it was only
 2 at sites with W126 metric values of 7 ppm-hrs or lower where at most ten total hourly
 3 concentrations reached 100 ppb or higher, occurring on no more than four distinct days. Sites
 4 with W126 metric values of 10 ppm-hrs or lower had as many as ten days with at least one hour
 5 at or above 100 ppb. Focusing on sites with W126 metric values below 20 ppm-hrs, several sites
 6 had N100 values of ten or greater and D100 values of five or greater, with individual sites having
 7 as many as 29 hours on up to 12 distinct days with concentrations of 100 ppb or greater.

8 Figure 4F-8 and Figure 4F-9 show scatter plots (similar to Figure 4F-4 and Figure 4F-5)
 9 that compare sites having different 2018, 2019, and 2020 annual 4th max values (x-axis) with
 10 regard to the 2018, 2019, and 2020 N100 and D100 values (y-axis), respectively. As can be seen
 11 from these figures, sites where the annual 4th max value was at or below 70 ppb generally had at
 12 most five hours on two distinct days where the O₃ concentrations reached 100 ppb or more.

13 Figure 4F-10 and Figure 4F-11 show similar scatter plots comparing sites having different 2018,
 14 2019, and 2020 annual W126 values (x-axis) with regard to the 2018, 2019, and 2020 N100 and
 15 D100 values (y-axis), respectively. There were sites that had five or more hours at or above 100
 16 ppb on up to three distinct days at annual W126 levels as low as 5 ppm-hrs. Focusing on sites
 17 where the annual W126 values were below 20 ppm-hrs, several sites had ten or more hours on
 18 five or more distinct days where O₃ concentrations reached 100 ppb or more.



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20 **Figure 4F-2. Map of 2018-2020 Average N100 Values at sites with valid design values.**

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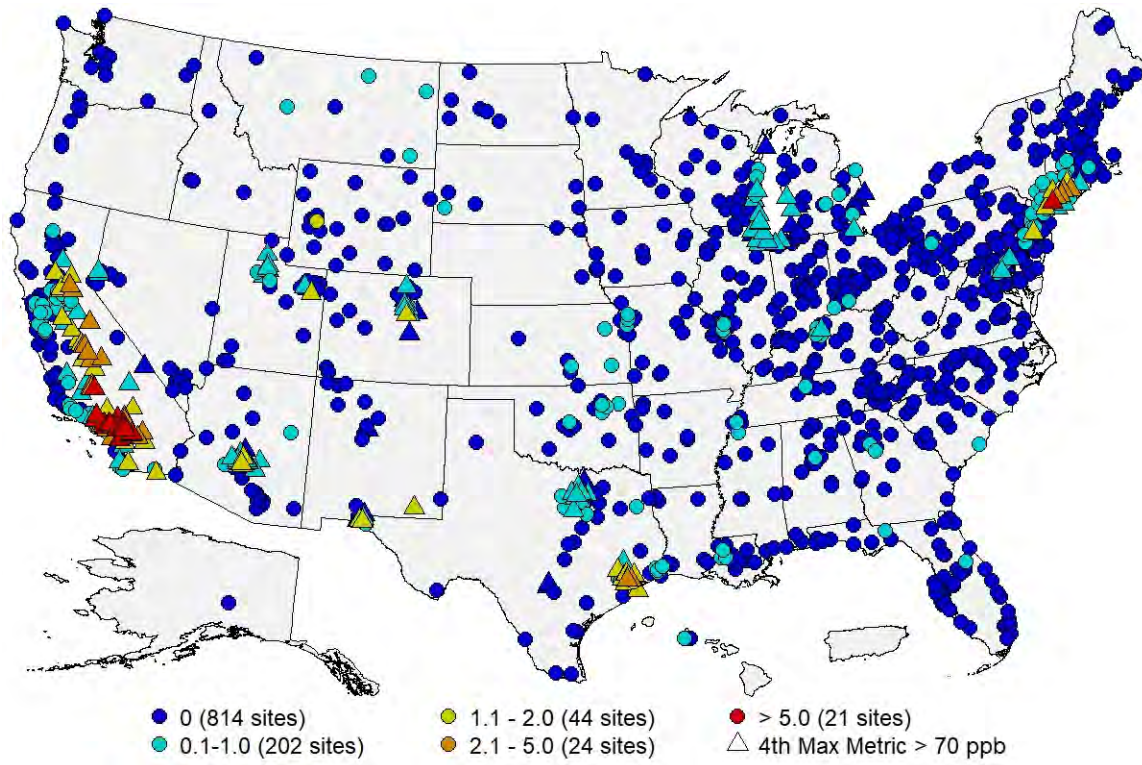
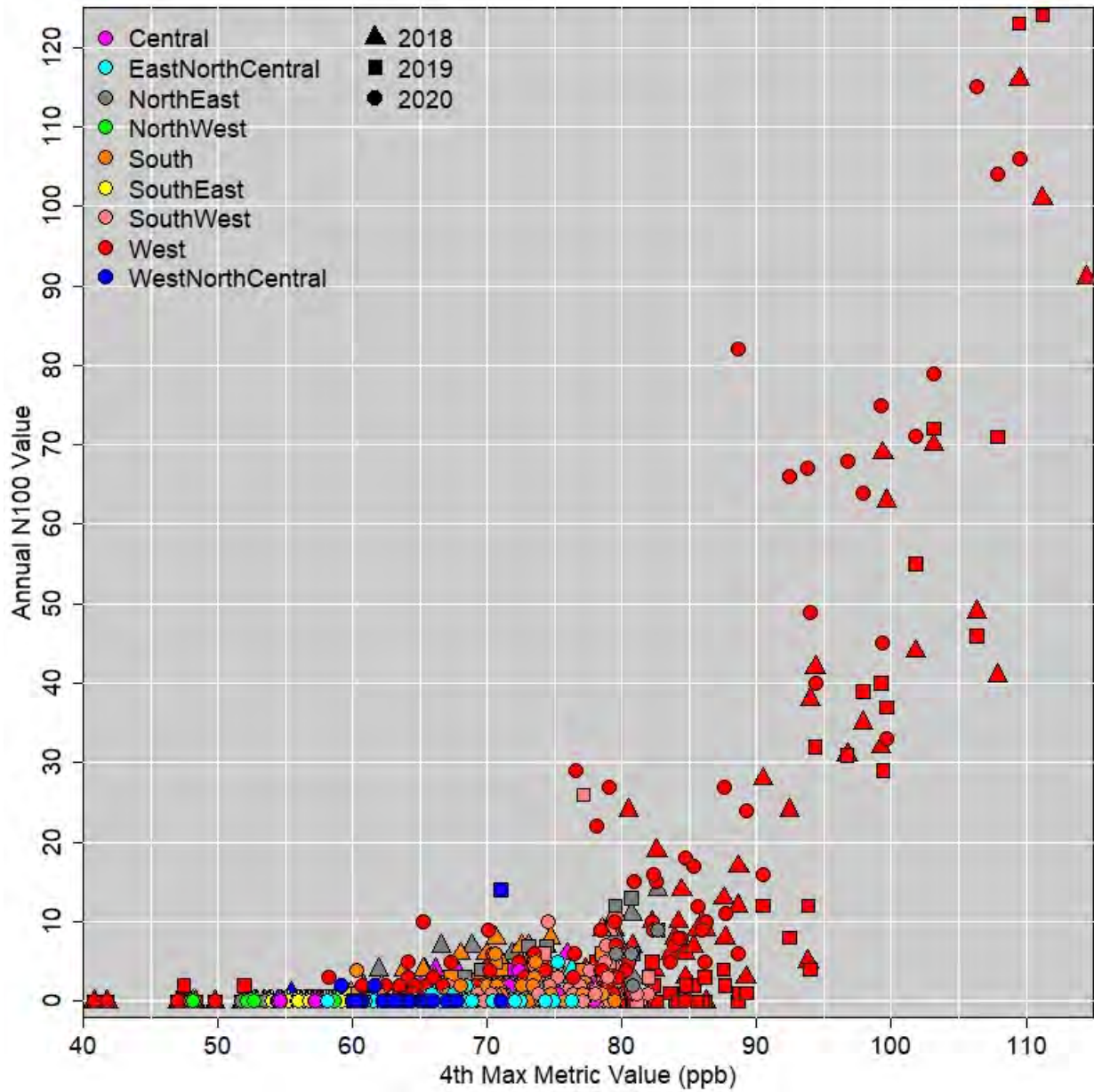
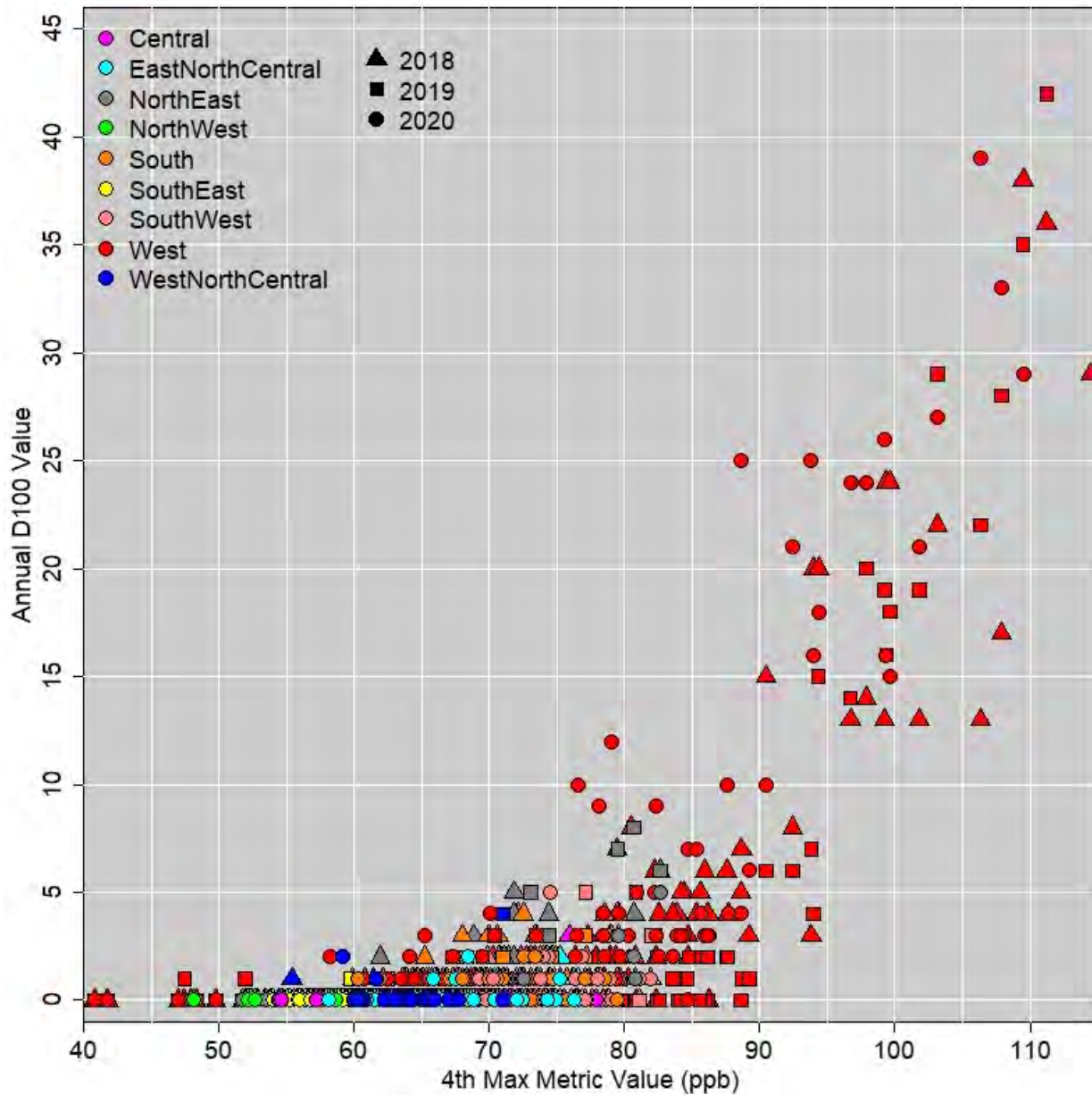


Figure 4F-3. Map of 2018-2020 Average D100 Values at sites with valid design values.



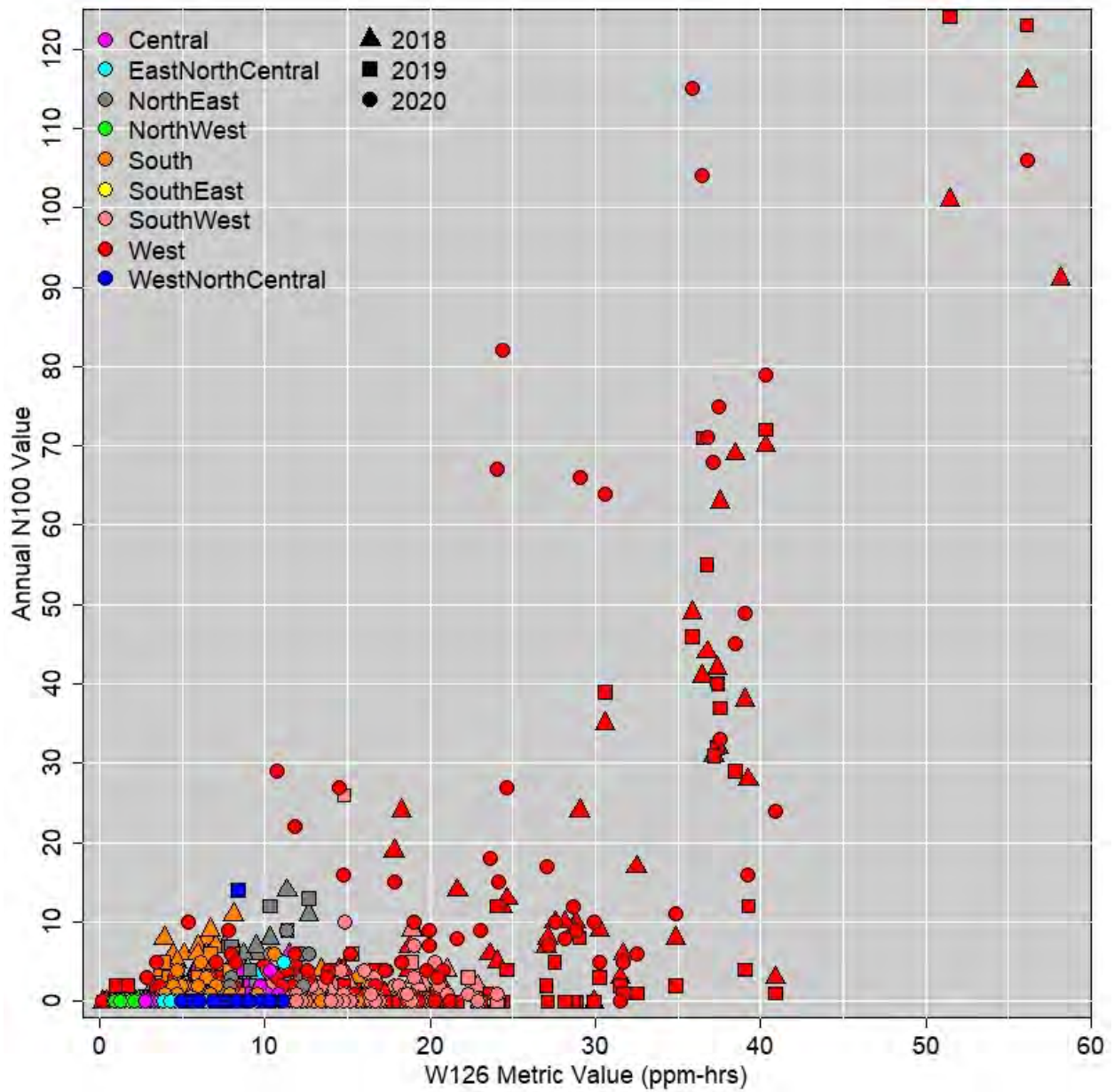
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Figure 4F-4. Scatter plot of annual N100 values (y-axis) versus 4th max metric values (design values, x-axis) based on 2018-2020 monitoring data.



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 2 **Figure 4F-5. Scatter plot of annual D100 values (y-axis) versus 4th max metric values**
 3 **(design values, x-axis) based on 2018-2020 monitoring data.**

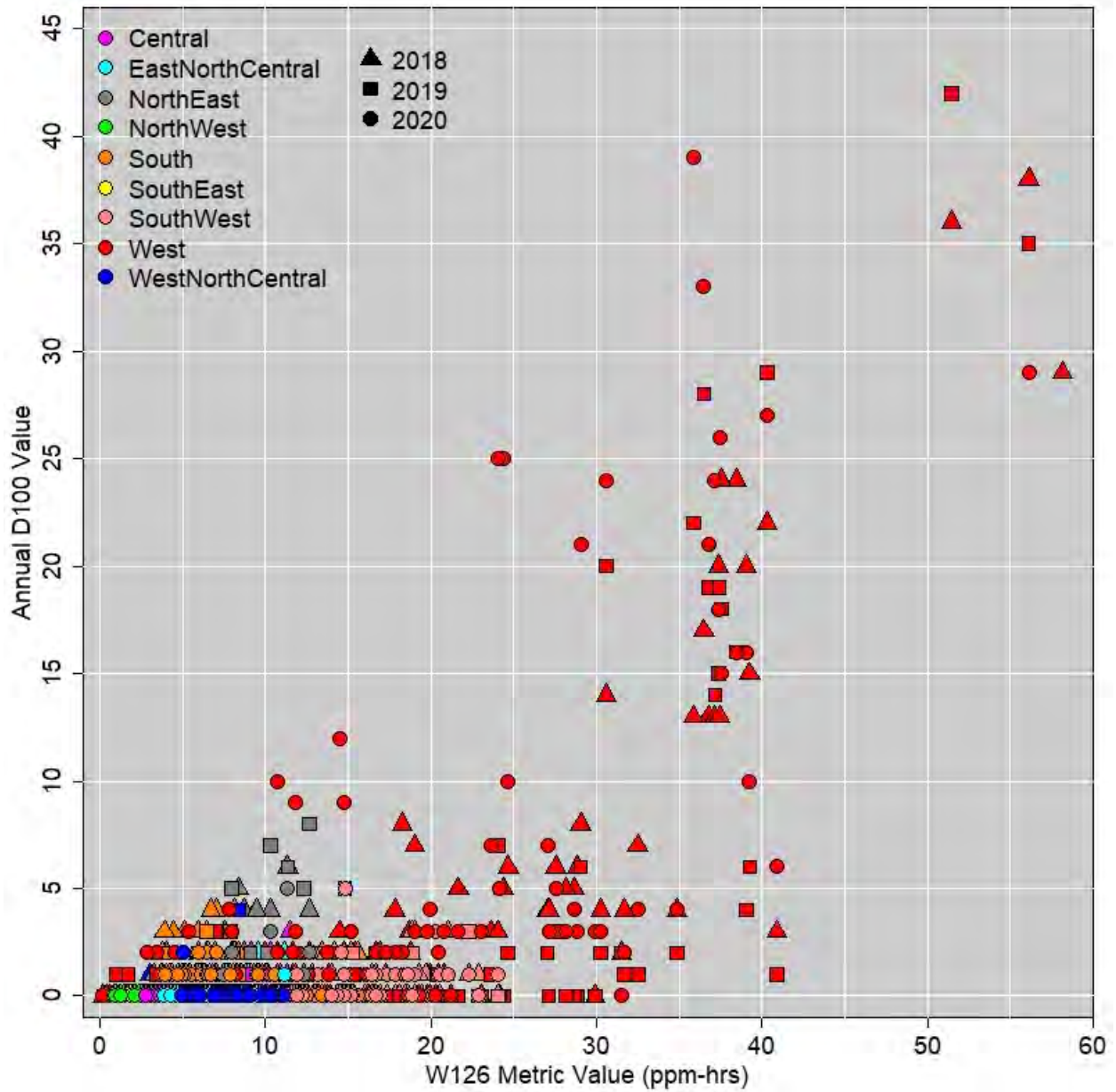
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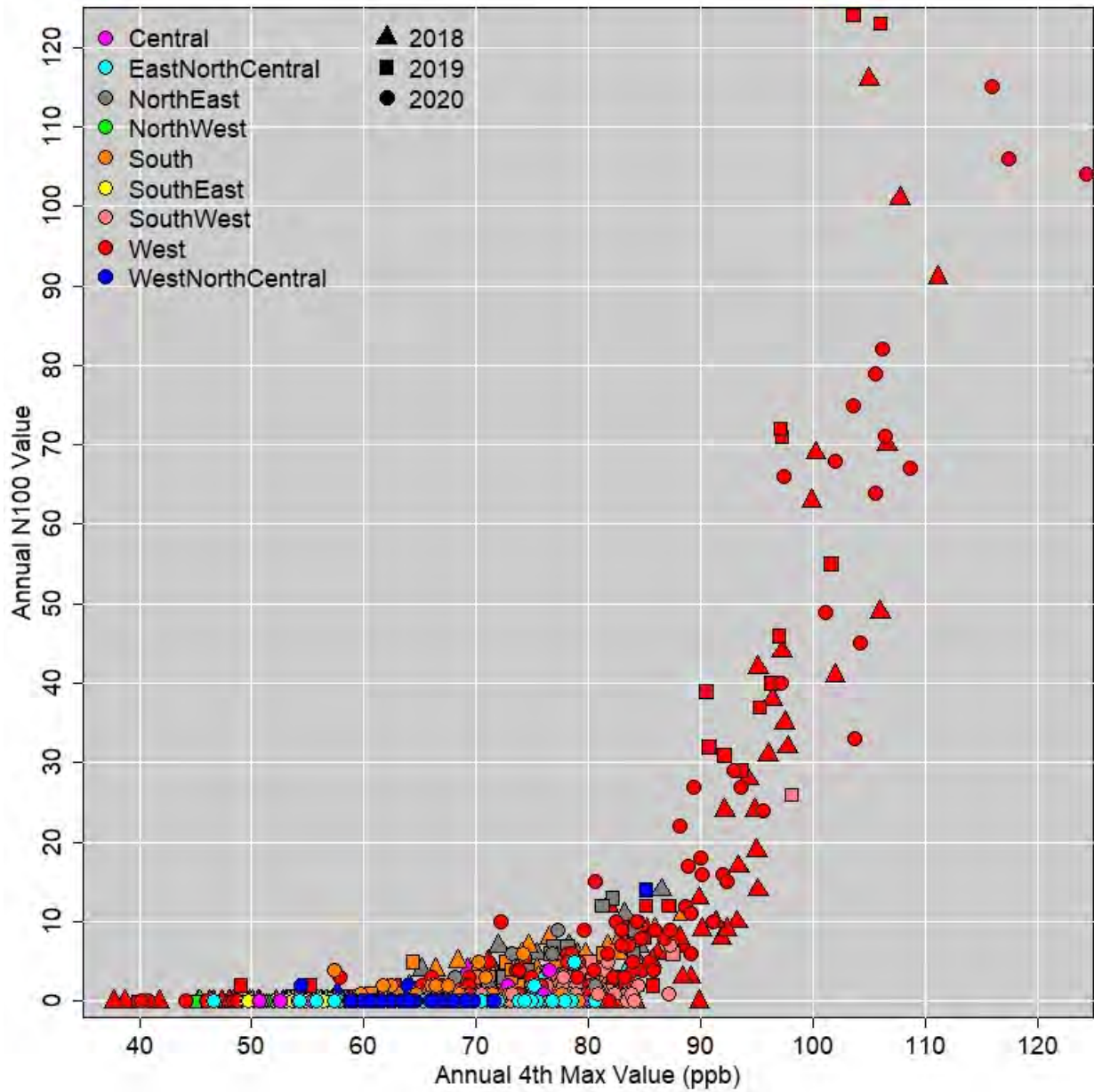
2 **Figure 4F-6. Scatter plot of annual N100 values (y-axis) versus W126 metric values (x-**
 3 **axis) based on 2018-2020 monitoring data.**

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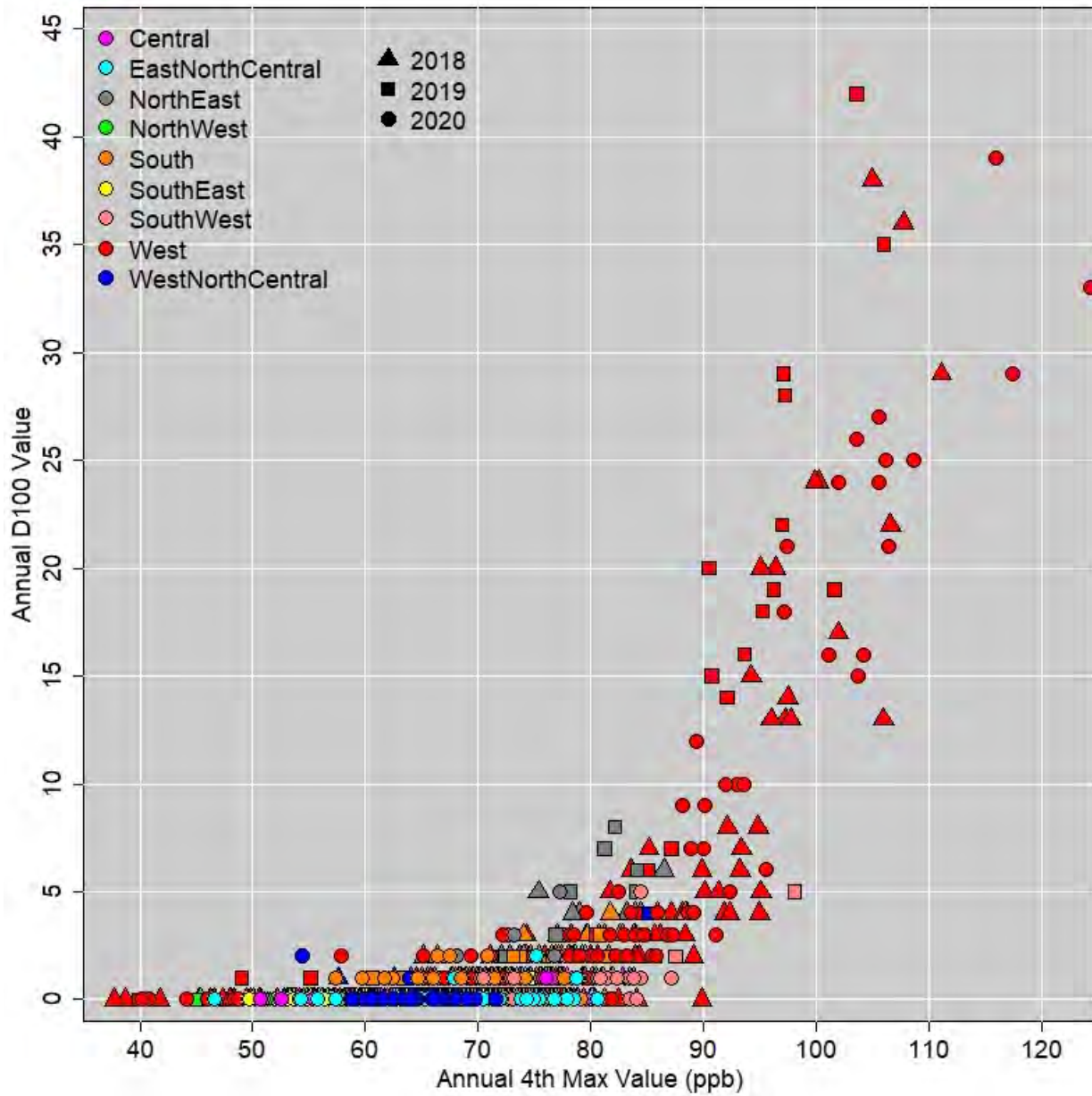
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Figure 4F-7. Scatter plot of annual D100 values (y-axis) versus W126 metric values (x-axis) based on 2018-2020 monitoring data.



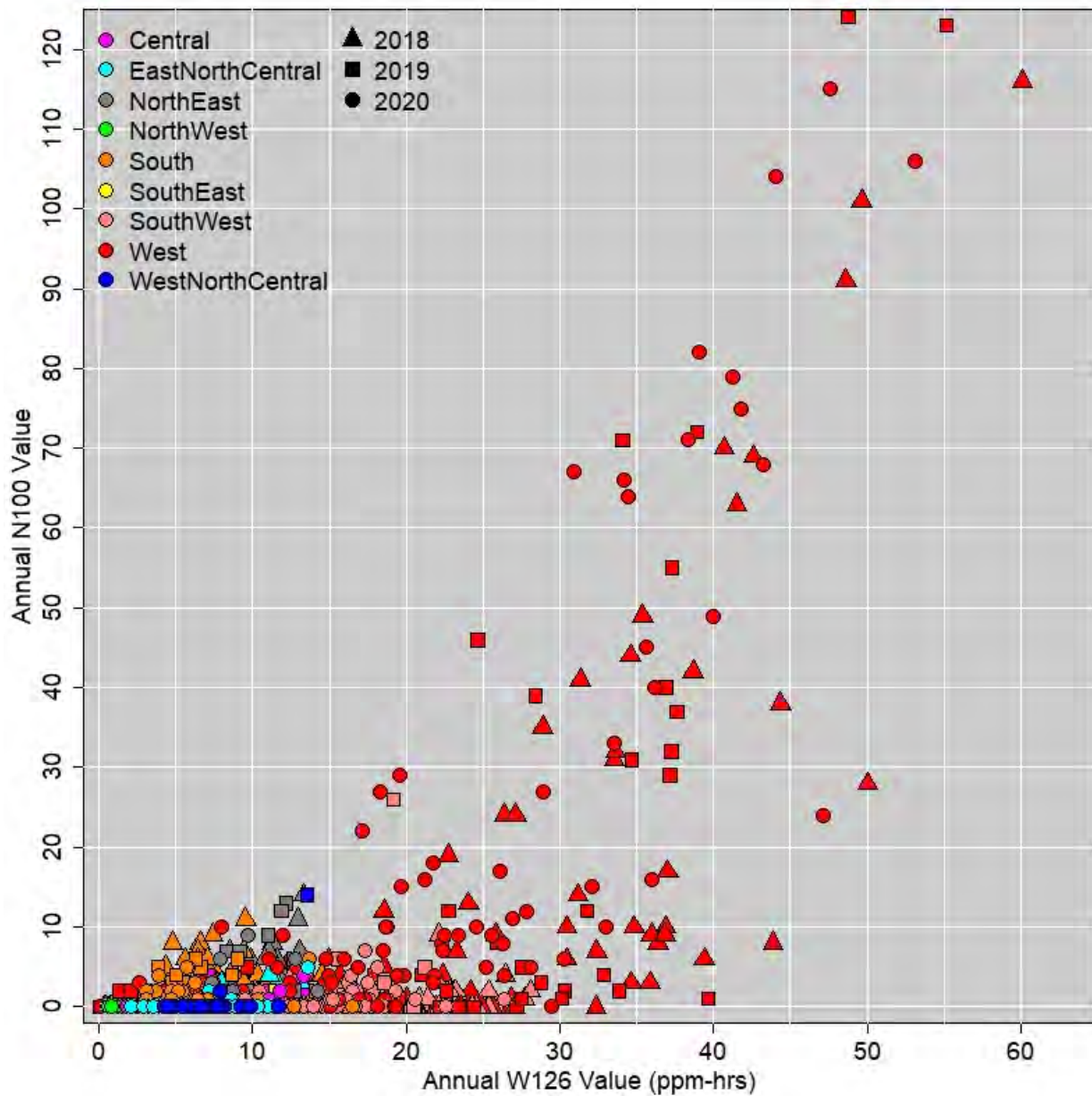
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Figure 4F-8. Scatter plot of annual N100 values (Y-axis) versus annual 4th max values (x-axis), based on 2018-2020 monitoring data.



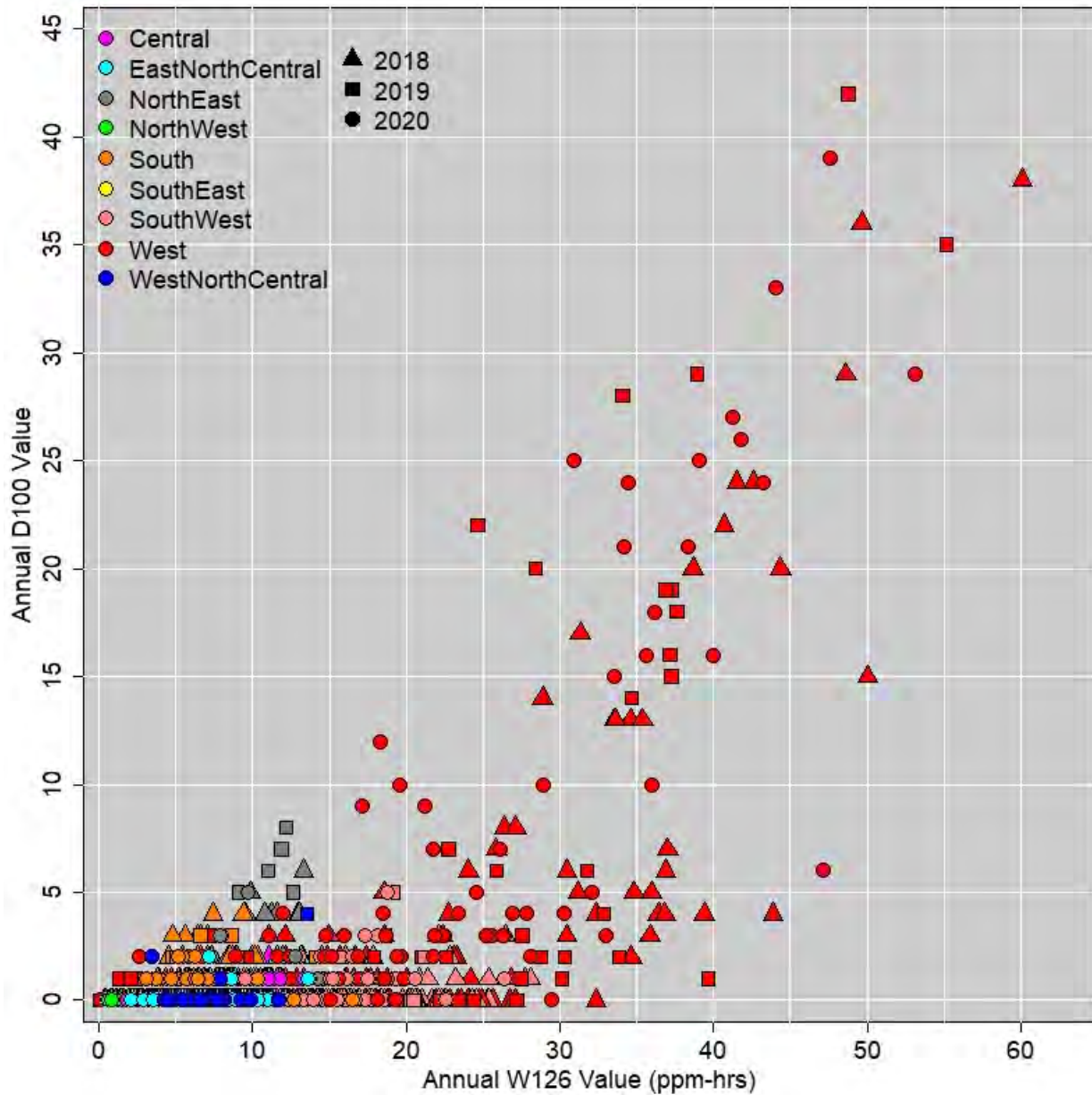
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 2 **Figure 4F-9. Scatter plot of annual D100 values (Y-axis) versus annual 4th max values (x-**
 3 **axis), based on 2018-2020 monitoring data.**

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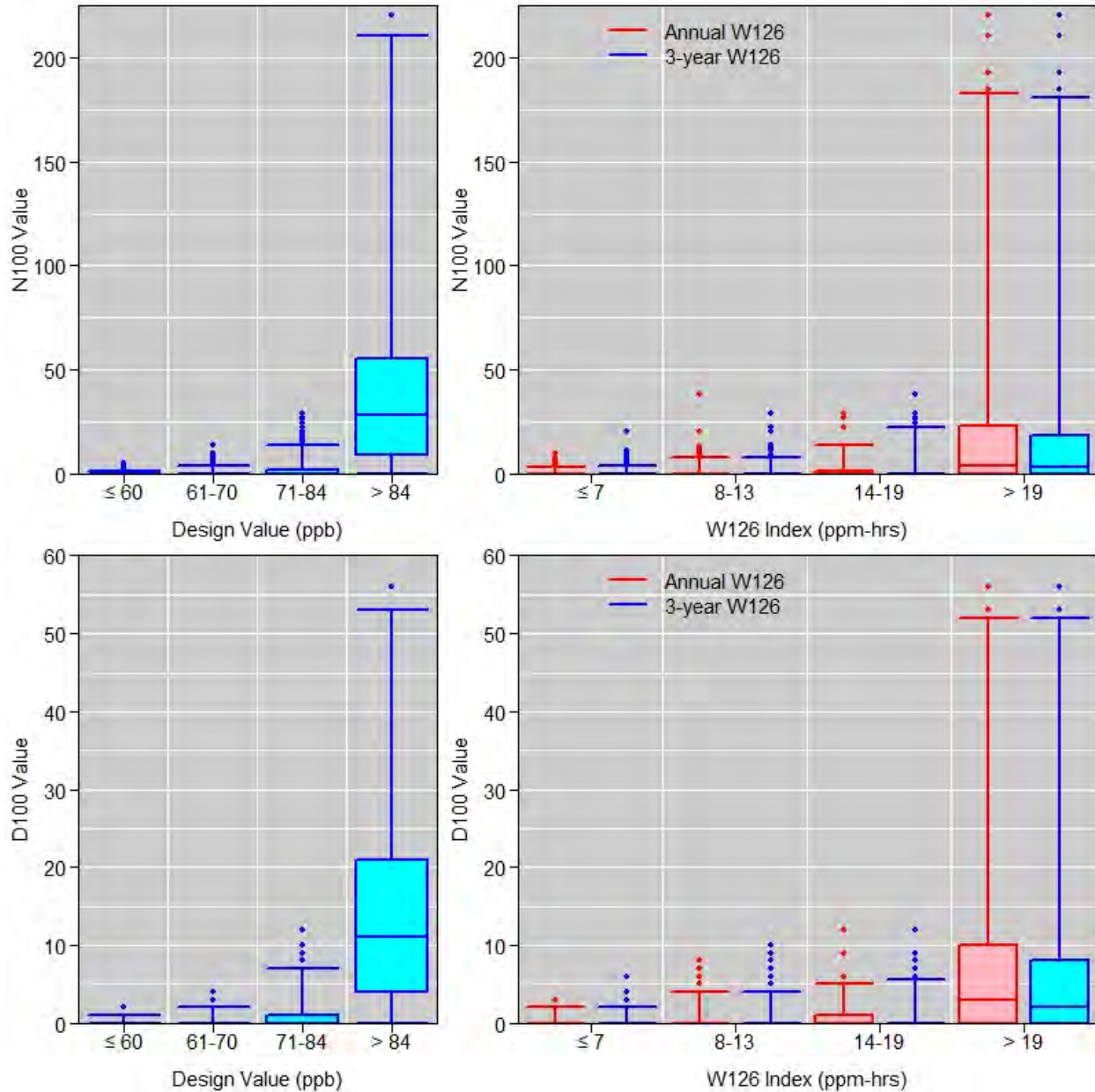


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Figure 4F-10. Scatter plot of annual N100 values (Y-axis) versus annual W126 values (x-axis), based on 2018-2020 monitoring data.



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 2 **Figure 4F-11. Scatter plot of annual D100 values (Y-axis) versus annual W126 values (x-**
 3 **axis), based on 2018-2020 monitoring data.**



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Figure 4F-12. Boxplots showing distribution of N100 values (top panels) and D100 values (bottom panels) based on 2016-2020 data binned according to design values (left panels) and W126 values (right panels, annual W126 in red, 3-year W126 in blue). The boxes represent the 25th, 50th and 75th percentiles and the whiskers extend to the 1st and 99th percentiles. Outlier values are represented by circles.

Table 4F-1 below shows the number of sites where the 2018-2020 4th max metric values meet the current standard or the number of instances (i.e., site-years) where the 2018, 2019, and 2020 annual 4th max values are at or below the level of the current standard and the 2018, 2019,

1 and 2020 N100 or D100 values are above various thresholds. The table also shows number of
2 sites where the 2018-2020 W126 metric values are at or below specific W126 levels or the
3 number of instances where the 2018, 2019, and 2020 annual W126 values are at or below
4 specific W126 levels and the 2018, 2019, and 2020 N100 or D100 values are above various
5 thresholds. The number of sites or instances where the N100 and D100 values were nonzero are
6 always equal, because having at least one hour where the concentration is at or above 100 ppb
7 guarantees having at least one day where the maximum hourly concentration is at least 100 ppb.
8 The number of sites or instances where the D100 values exceeded 2 and 5 were generally similar
9 to the number of sites or instances where the N100 values exceeded 5 and 10, respectively.

10 With regard to sites at or below specific annual 4th max and W126 values in any of the
11 three years, according to Table 4F-1, there were no instances out of over 2,700 site-years where
12 the N100 value exceeded 5 for sites during a year where the annual 4th max value was at or
13 below the level of the current standard. Additionally, there were only ten sites out 877 (about
14 1%) that met the current standard based on 2018-2020 data and also had N100 values exceeding
15 5 in one or more years. By contrast, there were 47 instances out of over 3,300 (1.4%) where the
16 N100 value exceeded 5 for sites that had an annual W126 value at or below 19 ppm-hrs; and
17 additionally, 37 sites out of over 1,000 (more than 3%) that had a 2018-2020 W126 metric value
18 was at or below 17 ppm-hrs and a N100 value exceeding 5 in one or more years. Even when
19 looking at sites at or below a W126 level of 7 ppm-hrs, there were nearly as many sites (9) with
20 N100 values exceeding 5 than for sites meeting the current standard (10).

21 Table 4F-2 shows the same statistics as in Table 4F-1 for the annual 4th max and annual
22 W126 values broken out into individual years, with the maximum annual value across the three
23 years for each combination of 4th max/W126 and N100/D100 thresholds highlighted in light
24 blue. This table shows that while there is considerable inter-annual variation in the 4th max and
25 W126 values across years, the annual W126 values always have a higher proportion of sites
26 below the threshold and above the N100 or D100 thresholds compared to those of the annual 4th
27 max values. Further, during the highest year for the different N100 and D100 thresholds, the
28 proportion of sites exceeding those thresholds is greater for the sites at/below the different annual
29 W126 levels than it is for sites with design values at/below 70 ppb. This is also evident in
30 comparing Figure 4F-5 to Figure 4F-11 and Figure 4F-4 to Figure 4F-10.

1 **Table 4F-1. Number of instances where 4th max or W126 values are at or below various**
 2 **thresholds and N100 or D100 values are above various thresholds based on O₃**
 3 **monitoring data from recent years (2018-2020).**

	Total*	Number of instances where:			Number of instances where:		
		N100 > 0	N100 > 5	N100 > 10	D100 > 0	D100 > 2	D100 > 5
Number of sites exceeding threshold in one or more years							
3-year Total**	1,073	278 (26%)	80 (7%)	39 (4%)	278 (26%)	83 (8%)	34 (3%)
3-year 4 th Max ≤ 70	877	125 (14%)	10 (1%)	1 (0.1%)	125 (14%)	9 (1%)	0 (0%)
3-year W126 ≤ 19	1,027	233 (23%)	43 (4%)	12 (1%)	233 (23%)	41 (4%)	9 (0.9%)
3-year W126 ≤ 17	1,009	218 (22%)	37 (4%)	10 (1%)	218 (22%)	34 (3%)	7 (0.7%)
3-year W126 ≤ 15	991	207 (21%)	37 (4%)	10 (1%)	207 (21%)	34 (3%)	7 (0.7%)
3-year W126 ≤ 7	722	100 (14%)	11 (2%)	0 (0%)	100 (14%)	9 (1%)	0 (0%)
Average number of sites exceeding threshold per year							
3-year Total**	1,073	145.3 (14%)	44.7 (4%)	24.7 (2%)	145.3 (14%)	46.7 (4%)	22.7 (2%)
3-year 4 th Max ≤ 70	877	49 (6%)	3.3 (0.4%)	0.3 (<0.1%)	49 (6%)	3 (0.3%)	0 (0%)
3-year W126 ≤ 19	1,027	107.7 (10%)	17.7 (2%)	4.7 (0.5%)	107.7 (10%)	16.3 (2%)	3.3 (0.3%)
3-year W126 ≤ 17	1,009	100 (10%)	15 (1%)	3.7 (0.4%)	100 (10%)	13.7 (1%)	2.7 (0.3%)
3-year W126 ≤ 15	991	94.7 (10%)	15 (2%)	3.7 (0.4%)	94.7 (10%)	13.7 (1%)	2.7 (0.3%)
3-year W126 ≤ 7	722	43 (6%)	4 (0.6%)	0 (0%)	43 (6%)	3.3 (0.5%)	0 (0%)
Total number of instances (site/years) exceeding threshold							
Annual Total***	3,522	473 (13%)	143 (4%)	77 (2%)	473 (13%)	149 (4%)	70 (2%)
Annual 4 th Max ≤ 70	2,743	96 (3%)	0 (0%)	0 (0%)	96 (3%)	0 (0%)	0 (0%)
Annual W126 ≤ 25	3,421	375 (11%)	64 (2%)	19 (0.6%)	375 (11%)	66 (2%)	12 (0.4%)
Annual W126 ≤ 19	3,336	333 (10%)	47 (1%)	10 (0.3%)	333 (10%)	47 (1%)	6 (0.2%)
Annual W126 ≤ 17	3,285	309 (9%)	41 (1%)	7 (0.2%)	309 (9%)	38 (1%)	5 (0.2%)
Annual W126 ≤ 15	3,196	281 (9%)	37 (1%)	6 (0.2%)	281 (9%)	35 (1%)	4 (0.1%)
Annual W126 ≤ 7	2,319	115 (5%)	9 (0.4%)	0 (0%)	115 (5%)	8 (0.3%)	0 (0%)

	Total*	Number of instances where:			Number of instances where:		
		N100 > 0	N100 > 5	N100 > 10	D100 > 0	D100 > 2	D100 > 5
		Number of sites exceeding threshold in one or more years					
<p>* Total number of sites where the 3-year 4th max or W126 value is at or below the threshold, or the total number of instances (i.e., site/years) where the annual 4th max or W126 value is at or below the threshold.</p> <p>** First column shows the number of sites with sufficient data to calculate valid 3-year 4th max and W126 values. Subsequent columns tally the subset of those sites where the N100 or D100 value exceeds the threshold in one or more years.</p> <p>*** First column shows the number of instances where a site had sufficient data to calculate valid annual 4th max and W126 values. Subsequent columns tally the subset of those instances where the N100 or D100 value exceeds the threshold.</p>							

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1 **Table 4F-2. Number of instances where annual 4th max or W126 values are at or below**
 2 **various thresholds and N100 or D100 values are above various thresholds based on O₃**
 3 **monitoring data from 2018-2020**

	Total Number of Sites*	Number of sites where:			Number of sites where:		
		N100 > 0	N100 > 5	N100 > 10	D100 > 0	D100 > 2	D100 > 5
<i>Number of sites exceeding threshold in the maximum year of the three</i>							
3-year 4 th Max ≤ 70	877	75 (9%)	5 (0.6%)	1 (0.1%)	75 (9%)	4 (0.5%)	0 (0%)
Annual 4 th Max ≤ 70	See Below	39 (4%)	0 (0%)	0 (0%)	39 (4%)	0 (0%)	0 (0%)
Annual W126 ≤ 25		166 (15%)	26 (2%)	7 (0.6%)	166 (15%)	26 (2%)	5 (0.4%)
Annual W126 ≤ 19		146 (13%)	21 (2%)	4 (0.4%)	146 (13%)	20 (28%)	3 (0.3%)
Annual W126 ≤ 17		139 (13%)	20 (2%)	3 (0.3%)	139 (13%)	18 (2%)	3 (0.3%)
Annual W126 ≤ 15		131 (13%)	20 (2%)	3 (0.3%)	131 (13%)	18 (2%)	3 (0.3%)
Annual W126 ≤ 7		47 (8%)	8 (1%)	0 (0%)	47 (8%)	6 (1%)	0 (0%)
<i>Number of sites exceeding threshold in individual years</i>							
2020 Total**	1,172	165 (14%)	56 (5%)	32 (3%)	165 (14%)	56 (5%)	27 (2%)
2019 Total**	1,163	101 (9%)	27 (2%)	19 (2%)	101 (9%)	31 (3%)	19 (2%)
2018 Total**	1,187	207 (17%)	60 (5%)	26 (2%)	207 (17%)	62 (5%)	24 (2%)
2020 4 th Max ≤ 70	941	39 (4%)	0 (0%)	0 (0%)	39 (4%)	0 (0%)	0 (0%)
2019 4 th Max ≤ 70	1,000	25 (3%)	0 (0%)	0 (0%)	25 (3%)	0 (0%)	0 (0%)
2018 4 th Max ≤ 70	802	32 (4%)	0 (0%)	0 (0%)	32 (4%)	0 (0%)	0 (0%)
2020 W126 ≤ 25	1,134	131 (12%)	26 (2%)	7 (0.6%)	131 (12%)	26 (2%)	5 (0.4%)
2019 W126 ≤ 25	1,144	78 (7%)	13 (1%)	6 (0.5%)	78 (7%)	15 (1%)	5 (0.4%)
2018 W126 ≤ 25	1,143	166 (15%)	25 (2%)	6 (0.5%)	166 (15%)	25 (2%)	2 (0.2%)
2020 W126 ≤ 19	1,116	114 (10%)	15 (1%)	2 (0.2%)	114 (10%)	14 (1%)	2 (0.2%)
2019 W126 ≤ 19	1,129	73 (6%)	11 (1%)	4 (0.4%)	73 (6%)	13 (1%)	3 (0.3%)
2018 W126 ≤ 19	1,091	146 (13%)	21 (2%)	4 (0.4%)	146 (13%)	20 (2%)	1 (0.1%)
2020 W126 ≤ 17	1,101	103 (9%)	11 (1%)	1 (0.1%)	103 (9%)	9 (0.9%)	1 (0.1%)
2019 W126 ≤ 17	1,117	67 (6%)	10 (0.9%)	3 (0.3%)	67 (6%)	11 (1%)	3 (0.3%)
2018 W126 ≤ 17	1,067	139 (13%)	20 (27%)	3 (0.3%)	139 (13%)	18 (2%)	1 (0.1%)
2020 W126 ≤ 15	1,074	85 (8%)	8 (0.7%)	0 (0%)	85 (8%)	6 (0.6%)	0 (0%)
2019 W126 ≤ 15	1,091	65 (6%)	9 (0.8%)	3 (0.3%)	65 (6%)	11 (1%)	3 (0.3%)
2018 W126 ≤ 15	1,031	131 (13%)	20 (2%)	3 (0.3%)	131 (13%)	18 (2%)	1 (0.1%)
2020 W126 ≤ 7	833	34 (4%)	0 (0%)	0 (0%)	347 (4%)	0 (0%)	0 (0%)
2019 W126 ≤ 7	860	34 (4%)	1 (0.1%)	0 (0%)	34 (4%)	2 (0.2%)	0 (0%)
2018 W126 ≤ 7	626	47 (8%)	8 (1%)	0 (0%)	47 (8%)	6 (1%)	0 (0%)
* Total number of sites where the annual 4 th max or W126 value is at or below the threshold.							
** First column represents the number of sites with sufficient data to calculate a valid annual 4 th max value. Subsequent columns tally the subset of those sites where the N100 or D100 value exceeds the threshold in one or more years.							

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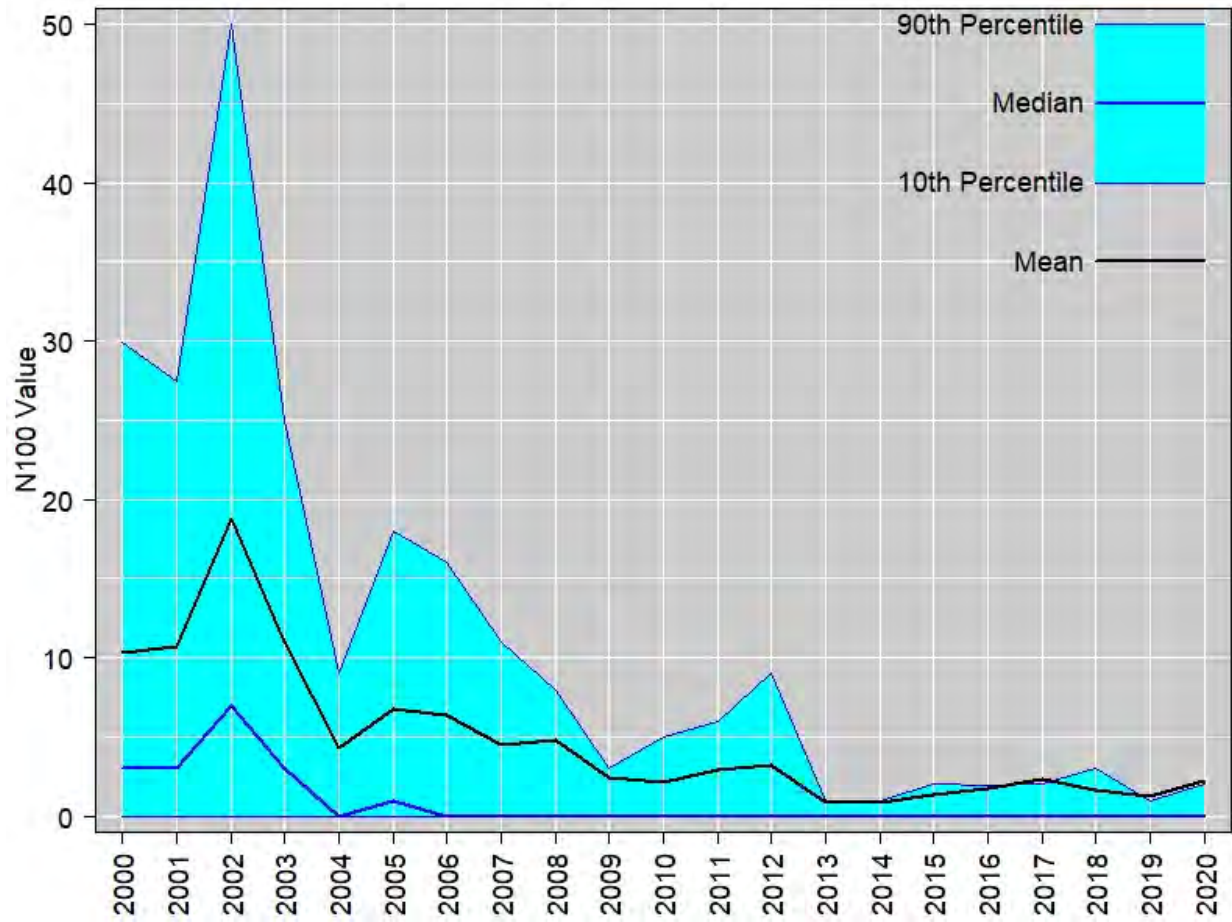
Table 4F-3. Average % of monitoring sites per year during 2016-2020 with 4th max or W126 metrics at or below various thresholds that have N100 or D100 values above various thresholds.

	Percent of sites where:			Percent of sites where:		
	N100 > 0	N100 > 5	N100 > 10	D100 > 0	D100 > 2	D100 > 5
<i>Average percent of sites exceeding N100 or D100 threshold per year (2016 – 2020)</i>						
3-year 4 th Max ≤ 70	5.1	0.3	0.01	5.1	0.2	0
3-year W126 ≤ 19	10.1	1.4	0.4	10.1	1.4	0.2
3-year W126 ≤ 17	9.7	1.4	0.3	9.7	1.3	0.2
3-year W126 ≤ 15	9.4	1.3	0.3	9.4	1.2	0.2
3-year W126 ≤ 7	6.1	0.5	0.04	6.1	0.3	0.01
Annual W126 ≤ 25	11.0	1.7	0.5	11.0	1.8	0.4
Annual W126 ≤ 19	10.0	1.4	0.3	10.0	1.4	0.2
Annual W126 ≤ 17	9.5	1.2	0.2	9.5	1.2	0.1
Annual W126 ≤ 15	9.1	1.2	0.2	9.1	1.1	0.1
Annual W126 ≤ 7	5.1	0.4	0	5.1	0.3	0
Annual 4 th Max ≤ 70	3.3	0.02	0	3.3	0.02	0

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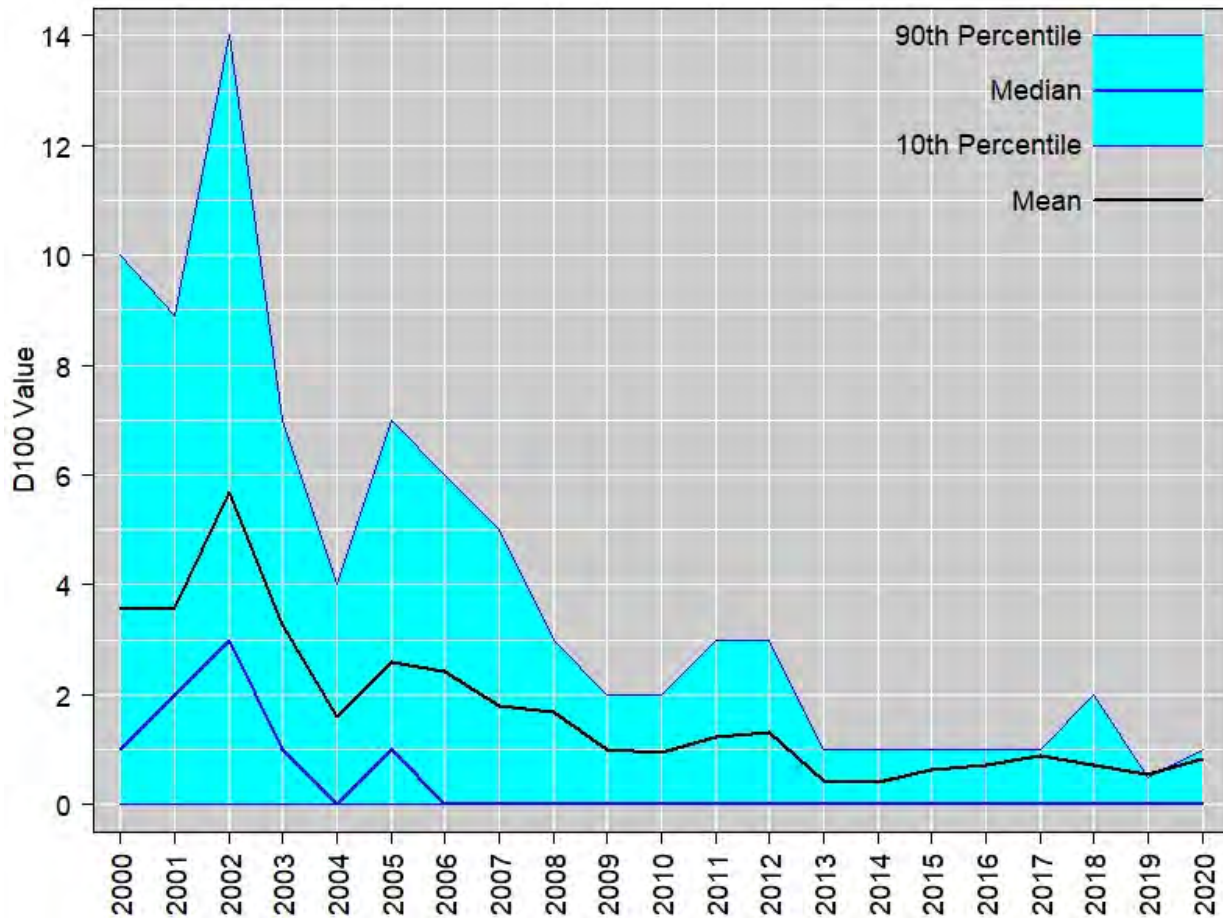
4F.3.2 National Analysis Using Historical Air Quality Data

Figure 4F-13 and Figure 4F-14 show the trend in national 10th percentile, median, 90th percentile and mean N100 and D100 values, respectively, based on 822 U.S. O₃ monitoring sites with complete data for 2000 to 2020. A site must have 75% annual data completeness in terms of the 4th max metric (see section 4F.2.2) for at least 16 of the 21 years, with no more than two consecutive years missing to be included in the trend. As can be seen from the figures, the median N100 and D100 values in the U.S. have been zero since 2006, meaning over half of the monitoring sites have N100 and D100 values of zero. The mean N100 value has decreased from more than ten in 2000-2002 to less than two in recent years, a decline of more than 80%. Similarly, the mean D100 value has decreased from four or more in 2000-2002 to less than one in recent years, also a decline of more than 80%. The 90th percentile values of both metrics have decreased at an even faster rate.



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Figure 4F-13. Trend in N100 values from 2000 to 2020 based on data from 808 U.S. O₃ monitoring sites



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2 **Figure 4F-14. Trend in D100 values from 2000 to 2020 based on data from 808 U.S. O₃**
3 **monitoring sites**

4
5 Table 4F-4 below shows the number of instances (site-years) where a site had an annual
6 4th max value or 4th max metric value at or below the level of the current standard and an annual
7 N100 or D100 value above various thresholds based on the full dataset spanning years 2000 to
8 2020. The table also shows number of instances (site-years) where a site had an annual W126
9 value or W126 metric value at or below specific W126 levels and N100 or D100 values above
10 various thresholds based on the full 2000-2020 dataset. The numbers in Table 4F-4 are generally
11 proportionally similar to those shown previously in Table 4F-1.

12 According to Table 4F-4, there were only 8 instances where the N100 value exceeded 5 at
13 a site with an annual 4th max value at or below the level of the current standard, and only 107
14 instances out of over 10,000 (about 1%) that met the current standard and also had N100 values
15 exceeding 5 in one or more of the three years of the design value period. By contrast, there were
16 over 1,500 instances where the annual W126 value was less than or equal to 19 ppm-hrs and the
17 N100 value in that year exceeded 5, and over 2,600 instances (more than 15%) where the W126

1 metric value was at or below 17 ppm-hrs and the N100 value exceeded 5 in one or more years of
 2 the 3-year period. Even when looking at sites at or below a W126 level of 7 ppm-hrs, there were
 3 more instances with N100 values exceeding 5 (170) than for sites meeting the current standard
 4 (107).
 5

6 **Table 4F-4. Number of instances where 4th max or W126 values are at or below various**
 7 **thresholds and N100 or D100 values are above various thresholds based on data from**
 8 **all years (2000-2020)**

	Total*	Number of instances where:			Number of instances where:		
		N100 > 0	N100 > 5	N100 > 10	D100 > 0	D100 > 2	D100 > 5
		Number of instances where site exceeds threshold in one or more years					
3-year Total**	20,483	10,103 (49%)	4,942 (24%)	3,213 (16%)	10,103 (49%)	4,920 (24%)	2,486 (12%)
3-year 4 th Max ≤ 70	10,026	1,638 (16%)	107 (1%)	16 (0.2%)	1,638 (16%)	89 (0.9%)	7 (0.1%)
3-year W126 ≤ 19	18,292	7,994 (44%)	3,178 (17%)	1,695 (9%)	7,994 (44%)	3,095 (17%)	1,054 (6%)
3-year W126 ≤ 17	17,427	7,255 (42%)	2,664 (15%)	1,328 (8%)	7,255 (42%)	2,576 (15%)	768 (4%)
3-year W126 ≤ 15	16,222	6,307 (39%)	2,076 (13%)	951 (6%)	6,307 (39%)	1,997 (12%)	522 (3%)
3-year W126 ≤ 7	7,576	1,427 (19%)	170 (2%)	40 (0.5%)	1,427 (19%)	152 (2%)	23 (0.3%)
		Total number of instances (site/years) exceeding threshold					
Annual Total***	24,987	7,908 (32%)	3,652 (15%)	2,327 (9%)	7,908 (32%)	3,609 (14%)	1,715 (7%)
Annual 4 th Max ≤ 70	12,402	563 (5%)	8 (0.1%)	0 (0%)	563 (5%)	3 (<0.1%)	0 (0%)
Annual W126 ≤ 25	23,482	6,504 (28%)	2,444 (10%)	1,274 (5%)	6,504 (28%)	2,370 (10%)	709 (3%)
Annual W126 ≤ 19	21,660	5,121 (24%)	1,587 (7%)	736 (3%)	5,121 (24%)	1,503 (7%)	344 (2%)
Annual W126 ≤ 17	20,600	4,427 (21%)	1,226 (6%)	530 (3%)	4,427 (21%)	1,162 (6%)	234 (1%)
Annual W126 ≤ 15	19,225	3,663 (19%)	885 (5%)	324 (2%)	3,663 (19%)	839 (4%)	144 (0.8%)
Annual W126 ≤ 7	10,427	770 (7%)	62 (0.6%)	4 (<0.1%)	770 (7%)	50 (0.5%)	2 (<0.1%)

* Total number of sites where the 3-year 4th max or W126 value is at or below the threshold, or the total number of instances (i.e., site/years) where the annual 4th max or W126 value is at or below the threshold.

** First column shows the number of sites with sufficient data to calculate valid 3-year 4th max and W126 values. Subsequent columns tally the subset of those sites where the N100 or D100 value exceeds the threshold in one or more years.

*** First column shows the number of instances where a site had sufficient data to calculate valid annual 4th max and W126 values. Subsequent columns tally the subset of those instances where the N100 or D100 value exceeds the threshold.

4F.4 SUMMARY

The presentation here shows various analyses of ambient air monitoring data for O₃ concentrations in the U.S. relating to the form and averaging time of the current secondary standard, the W126-based cumulative exposure index, the N100 metric (number of hours at or above 100 ppb) and D100 metric (number of days with one or more hours at or above 100 ppb).

- About 74% of the O₃ monitoring sites with valid design values in 2018-2020 did not have any hourly concentrations at or above 100 ppb, and another 18% had only a single day where hourly O₃ concentrations reached 100 ppb or more (Figure 4F-2 and Figure 4F-3).
- Based on data from 2018-2020, sites where the current standard was met (4th max metric value was at or below 70 ppb) had a maximum annual N100 count of 10 and D100 count of 4 (Figure 4F-4 and Figure 4F-5). Sites with W126 metric values as low as 7 ppm-hrs also had a maximum annual N100 count of 10 and D100 count of 4. At sites with W126 metric values below 20 ppm-hrs, several sites had N100 values of ten or greater and D100 values of five or greater, with individual sites having as many as 29 hours on up to 12 distinct days with concentrations of 100 ppb or greater (Figure 4F-6 and Figure 4F-7).
- In 2018-2020, sites where the annual 4th max value was at or below 70 ppb had a maximum annual N100 count of 5 and D100 count of 2 (Figure 4F-8 and Figure 4F-9). Sites with annual W126 values as low as 5 ppm-hrs had a maximum N100 count of 8 and D100 count of 3. At sites with annual W126 values below 20 ppm-hrs, several sites had ten or more hours on five or more distinct days where O₃ concentrations reached 100 ppb or more (Figure 4F-10 and Figure 4F-11).
- Based on data from 2018-2020, about 1% of sites that met the current standard had an N100 value exceeding 5 in one or more years. By comparison, more than 3% of sites where the W126 metric value was at or below 17 ppm-hrs had an N100 value exceeding 5 (Table 4F-1). There were no sites with N100 values exceeding 5 among sites with annual 4th max values at or below the level of the current standard compared with between 11 and 21 sites per year with N100 values exceeding 5 among sites with annual W126 values at or below 19 ppm-hrs (Table 4F-2).
- Based on data from 2000-2020, about 1% of design values that met the current standard had N100 values exceeding 5 in one or more years of the 3-year period. By comparison, about 15% of W126 metric values at or below 17 ppm-hrs had N100 values exceeding 5 in one or more years of the 3-year period (Table 4F-4).
- Since 2000-2002, the national mean N100 and D100 values have decreased by more than 80% (Figure 4F-13 and Figure 4F-14).

1 **4F.5 REFERENCES**

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