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

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

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1 INTRODUCTION

This document, *Policy Assessment for the Review of the Ozone National Ambient Air Quality Standards* (hereafter referred to as the PA), presents the policy assessment for the U.S. Environmental Protection Agency's (EPA's) current review of the ozone (O_3) national ambient air quality standards (NAAQS).[1] The overall plan for this review was presented in the *Integrated Review Plan for the Ozone National Ambient Air Quality Standards* (IRP; [U.S. EPA, 2019]). The IRP also identified key policy-relevant issues to be addressed in this review and discussed in the main documents that generally inform NAAQS reviews, including an Integrated Science Assessment (ISA), and a Policy Assessment (PA).

This document is organized into four chapters. Chapter 1 presents introductory information on the purpose of the PA, legislative requirements for reviews of the NAAQS, an overview of the history of the O_3 NAAQS, including background information on prior reviews, and a summary of the progress to date for the current review. Chapter 2 provides an overview of how photochemical oxidants, including O_3, are formed in the atmosphere, along with current information on sources and emissions of important precursor chemicals. Chapter 2 also summarizes key aspects of the ambient air monitoring requirements, and current O_3 air quality, including estimates of O_3 resulting from natural sources and anthropogenic sources outside the U.S. Chapters 3 and 4 focus on policy-relevant aspects of the currently available health and welfare effects evidence and exposure/risk information, identifying and summarizing key considerations related to this review of the primary (health-based) and secondary (welfare-based) standard, respectively.

1.1 PURPOSE

The PA, when final, presents an evaluation, for consideration by the EPA Administrator, of the policy implications of the currently available scientific information, assessed in the ISA, any quantitative air quality, exposure or risk analyses based on the ISA findings, and related limitations and uncertainties. Ultimately, a final decision on the O_3 NAAQS will reflect the judgments of the Administrator. The role of the PA is to help "bridge the gap" between the Agency's scientific assessment and quantitative technical analyses, and the judgments required of the Administrator in determining whether it is appropriate to retain or revise the O_3 NAAQS.

[1] This review 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 standards that are the focus of this review were set in 2015 (80 FR 65292, October 26, 2015) and are referred to in this document as the "current" or "existing" standards.

In evaluating the question of adequacy of the current standards and whether it may be appropriate to consider alternative standards, the PA focuses on information that is most pertinent to evaluating the standards and their basic elements: indicator, averaging time, form, and level.[2] These elements, which together serve to define each standard, must be considered collectively in evaluating the public health and public welfare protection the standards afford.

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

In this PA, we take into account the available scientific information, as assessed in the *Integrated Science Assessment for Ozone and Related Photochemical Oxidants* (ISA [U.S. EPA, 2020]) and additional policy-relevant quantitative air quality, exposure and risk analyses.[3] Thus, the PA is based on the final ISA and the evaluation and conclusions in this document have also been informed by the advice received from the CASAC in its reviews of the draft PA and draft IRP, and also by public comment received thus far in the review.

The PA is designed to assist the Administrator in considering the currently available scientific and risk information and formulating judgments regarding the standards. Accordingly, the PA will inform the Administrator's decision in this review. Beyond informing the Administrator and facilitating the advice and recommendations of the CASAC, the PA is also intended to be a useful reference to all parties interested in the review of the O_3 NAAQS. In these roles, it is intended to serve as a source of policy-relevant information that supports the Agency's review of the O_3 NAAQS, and it is written to be understandable to a broad audience.

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

[3] The terms "staff," "we" and "our" throughout this document refer to the staff in the EPA's Office of Air Quality Planning and Standards (OAQPS).

1.2 LEGISLATIVE REQUIREMENTS

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

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

In setting primary and secondary standards that are "requisite" to protect public health and welfare, respectively, as provided in section 109(b), the EPA's task is to establish standards that are neither more nor less stringent than necessary. In so doing, the EPA may not consider the costs of implementing the standards. See generally, *Whitman v. American Trucking Ass'ns*, 531 U.S. 457, 465-472, 475-76 (2001). Likewise, "[a]ttainability and technological feasibility are not relevant considerations in the promulgation of national ambient air quality standards" (*American Petroleum Institute v. Costle*, 665 F.2d 1176, 1185 [D.C. Cir. 1981], *cert. denied*, 455 U.S. 1034 [1982]; *accord Murray Energy Corp. v. EPA*, 936 F.3d 597, 623-24 [D.C. Cir. 2019]). At the same time, courts have clarified the EPA may consider "relative proximity to peak background

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

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

… concentrations" as a factor in deciding how to revise the NAAQS in the context of considering standard levels within the range of reasonable values supported by the air quality criteria and judgments of the Administrator (*American Trucking Ass'ns, v. EPA*, 283 F.3d 355, 379 [D.C. Cir. 2002], hereafter referred to as "*ATA III*").

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

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

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

Section 109(d)(2) addresses the appointment and advisory functions of an independent scientific review committee. Section 109(d)(2)(A) requires the Administrator to appoint this

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

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

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

As previously noted, the Supreme Court has held that section 109(b) "unambiguously bars cost considerations from the NAAQS-setting process" (*Whitman v. American Trucking Ass'ns,* 531 U.S. 457, 471 [2001]). Accordingly, while some of the issues listed in section 109(d)(2)(C) as those on which Congress has directed the CASAC to advise the Administrator, are ones that are relevant to the standard setting process, others are not. Issues that are not relevant to standard setting may be relevant to implementation of the NAAQS once they are established.[7]

1.3 HISTORY OF THE O_3 NAAQS, REVIEWS AND DECISIONS

Primary and secondary NAAQS were first established for photochemical oxidants in 1971 (36 FR 8186, April 30, 1971) based on the air quality criteria developed in 1970 (U.S.

[7] 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 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).

DHEW, 1970; 35 FR 4768, March 19, 1970). The EPA set both primary and secondary standards at 0.08 parts per million (ppm), as a 1-hour average of total photochemical oxidants, not to be exceeded more than one hour per year based on the scientific information in the 1970 air quality criteria document (AQCD). Since that time, the EPA has reviewed the air quality criteria and standards a number of times, with the most recent review being completed in 2015.

The EPA initiated the first periodic review of the NAAQS for photochemical oxidants in 1977. Based on the 1978 AQCD (U.S. EPA,1978), the EPA published proposed revisions to the original NAAQS in 1978 (43 FR 26962, June 22, 1978) and final revisions in 1979 (44 FR 8202, February 8, 1979). At that time, the EPA changed the indicator from photochemical oxidants to O_3, revised the level of the primary and secondary standards from 0.08 to 0.12 ppm and revised the form of both standards from a deterministic (i.e., not to be exceeded more than one hour per year) to a statistical form. With these changes, attainment of the standards was defined to occur when the average number of days per calendar year (across a 3-year period) with maximum hourly average O_3 concentration greater than 0.12 ppm equaled one or less (44 FR 8202, February 8, 1979; 43 FR 26962, June 22, 1978).

Following the EPA's decision in the 1979 review, several petitioners sought judicial review. Among those, the city of Houston challenged the Administrator's decision arguing that the standard was arbitrary and capricious because natural O_3 concentrations and other physical phenomena in the Houston area made the standard unattainable in that area. The U.S. Court of Appeals for the District of Columbia Circuit (D.C. Circuit) rejected this argument, holding (as noted in section 1.1 above) that attainability and technological feasibility are not relevant considerations in the promulgation of the NAAQS (*American Petroleum Institute v. Costle*, 665 F.2d at 1185). The court also noted that the EPA need not tailor the NAAQS to fit each region or locale, pointing out that Congress was aware of the difficulty in meeting standards in some locations and had addressed this difficulty through various compliance related provisions in the CAA (*id.* at 1184-86).

The next periodic reviews of the criteria and standards for O_3 and other photochemical oxidants began in 1982 and 1983, respectively (47 FR 11561, March 17, 1982; 48 FR 38009, August 22, 1983). The EPA subsequently published the 1986 AQCD (U.S. EPA, 1986) and the 1989 Staff Paper (U.S. EPA, 1989). Following publication of the 1986 AQCD, a number of scientific abstracts and articles were published that appeared to be of sufficient importance concerning potential health and welfare effects of O_3 to warrant preparation of a supplement to the 1986 AQCD (U.S. EPA, 1992). In August of 1992, the EPA proposed to retain the existing primary and secondary standards based on the health and welfare effects information contained in the 1986 AQCD and its 1992 Supplement (57 FR 35542, August 10, 1992). In March 1993,

the EPA announced its decision to conclude this review by affirming its proposed decision to retain the standards, without revision (58 FR 13008, March 9, 1993).

In the 1992 notice of its proposed decision in that review, the EPA announced its intention to proceed as rapidly as possible with the next review of the air quality criteria and standards for O_3 and other photochemical oxidants in light of emerging evidence of health effects related to 6- to 8-hour O_3 exposures (57 FR 35542, August 10, 1992). The EPA subsequently published the AQCD and Staff Paper for that next review (U.S. EPA, 1996). In December 1996, the EPA proposed revisions to both the primary and secondary standards (61 FR 65716, December 13, 1996). With regard to the primary standard, the EPA proposed to replace the then-existing 1-hour primary standard with an 8-hour standard set at a level of 0.08 ppm (equivalent to 0.084 ppm based on the proposed data handling convention) as a 3-year average of the annual third-highest daily maximum 8-hour concentration. The EPA proposed to revise the secondary standard either by setting it identical to the proposed new primary standard or by setting it as a new seasonal standard using a cumulative form. The EPA completed this review in 1997 by setting the primary standard at a level of 0.08 ppm, based on the annual fourth-highest daily maximum 8-hour average concentration, averaged over three years, and setting the secondary standard identical to the revised primary standard (62 FR 38856, July 18, 1997).

On May 14, 1999, in response to challenges by industry and others to the EPA's 1997 decision, the D.C. Circuit remanded the O_3 NAAQS to the EPA, finding that section 109 of the CAA, as interpreted by the EPA, effected an unconstitutional delegation of legislative authority (*American Trucking Ass'ns v. EPA*, 175 F.3d 1027, 1034-1040 [D.C. Cir. 1999]). In addition, the court directed that, in responding to the remand, the EPA should consider the potential beneficial health effects of O_3 pollution in shielding the public from the effects of solar ultraviolet (UV) radiation, as well as adverse health effects (*id.* at 1051-53). In 1999, the EPA sought panel rehearing and for rehearing *en banc* on several issues related to that decision. The court granted the request for panel rehearing in part and denied it in part but declined to review its ruling with regard to the potential beneficial effects of O_3 pollution (*American Trucking Ass'ns v. EPA*,195 F.3d 4, 10 [D.C Cir., 1999]). On January 27, 2000, the EPA petitioned the U.S. Supreme Court for *certiorari* on the constitutional issue (and two other issues) but did not request review of the ruling regarding the potential beneficial health effects of O_3. On February 27, 2001, the U.S. Supreme Court unanimously reversed the judgment of the D.C. Circuit on the constitutional issue (*Whitman v. American Trucking Ass'ns*, 531 U.S. 457, 472-74 [2001], [holding that section 109 of the CAA does not delegate legislative power to the EPA in contravention of the Constitution]). The Court remanded the case to the D.C. Circuit to consider challenges to the O_3 NAAQS that had not been addressed by that court's earlier decisions. On March 26, 2002, the D.C. Circuit issued its final decision on the remand, finding the 1997 O_3 NAAQS to be "neither

arbitrary nor capricious," and so denying the remaining petitions for review. See *ATA III*, 283 F.3d at 379.

Specifically, in *ATA III*, the D.C. Circuit upheld the EPA's decision on the 1997 O_3 standard as the product of reasoned decision making. With regard to the primary standard, the court made clear that the most important support for the EPA's decision to revise the standard was the health evidence of insufficient protection afforded by the then-existing standard ("the record [is] replete with references to studies demonstrating the inadequacies of the old one-hour standard"), as well as extensive information supporting the change to an 8-hour averaging time (*id.* at 378). The court further upheld the EPA's decision not to select a more stringent level for the primary standard noting "the absence of *any* [emphasis in original] human clinical studies at ozone concentrations below 0.08 [ppm]" which supported the EPA's conclusion that "the most serious health effects of ozone are 'less certain' at low concentrations, providing an eminently rational reason to set the primary standard at a somewhat higher level, at least until additional studies become available" (*id.* at 379, internal citations omitted). The court also pointed to the significant weight that the EPA properly placed on the advice it received from the CASAC (*id.* at 379). In addition, the court noted that "although relative proximity to peak background ozone concentrations did not, in itself, necessitate a level of 0.08 [ppm], EPA could consider that factor when choosing among the three alternative levels" (*id.* at 379).

Coincident with the continued litigation of the other issues, the EPA responded to the court's 1999 remand to consider the potential beneficial health effects of O_3 pollution in shielding the public from effects of UV radiation (66 FR 57268, Nov. 14, 2001; 68 FR 614, January 6, 2003). The EPA provisionally determined that the information linking changes in patterns of ground-level O_3 concentrations to changes in relevant patterns of exposures to UV radiation of concern (UV-B) to public health was too uncertain, at that time, to warrant any relaxation in 1997 O_3 NAAQS. The EPA also expressed the view that any plausible changes in UV-B radiation exposures from changes in patterns of ground-level O_3 concentrations would likely be very small from a public health perspective. In view of these findings, the EPA proposed to leave the 1997 primary standard unchanged (66 FR 57268, Nov. 14, 2001). After considering public comment on the proposed decision, the EPA published its final response to this remand in 2003, re-affirming the 8-hour primary standard set in 1997 (68 FR 614, January 6, 2003).

The EPA initiated the fourth periodic review of the air quality criteria and standards for O_3 and other photochemical oxidants with a call for information in September 2000 (65 FR 57810, September 26, 2000). In 2007, the EPA proposed to revise the level of the primary standard within a range of 0.075 to 0.070 ppm (72 FR 37818, July 11, 2007). The EPA proposed to revise the secondary standard either by setting it identical to the proposed new primary

standard or by setting it as a new seasonal standard using a cumulative form. Documents supporting these proposed decisions included the 2006 AQCD (U.S. EPA, 2006) and 2007 Staff Paper (U.S. EPA, 2007) and related technical support documents. The EPA completed the review in March 2008 by revising the levels of both the primary and secondary standards from 0.08 ppm to 0.075 ppm while retaining the other elements of the prior standards (73 FR 16436, March 27, 2008).

In May 2008, state, public health, environmental, and industry petitioners filed suit challenging the EPA's final decision on the 2008 O_3 standards. On September 16, 2009, the EPA announced its intention to reconsider the 2008 O_3 standards,[8] and initiated a rulemaking to do so. At the EPA's request, the court held the consolidated cases in abeyance pending the EPA's reconsideration of the 2008 decision.

In January 2010, the EPA issued a notice of proposed rulemaking to reconsider the 2008 final decision (75 FR 2938, January 19, 2010). In that notice, the EPA proposed that further revisions of the primary and secondary standards were necessary to provide a requisite level of protection to public health and welfare. The EPA proposed to revise the level of the primary standard from 0.075 ppm to a level within the range of 0.060 to 0.070 ppm, and to revise the secondary standard to one with a cumulative, seasonal form. At the EPA's request, the CASAC reviewed the proposed rule at a public teleconference on January 25, 2010 and provided additional advice in early 2011 (Samet, 2010, Samet, 2011). Later that year, in view of the need for further consideration and the fact that the Agency's next periodic review of the O_3 NAAQS required under CAA section 109 had already begun (as announced on September 29, 2008),[9] the EPA decided to consolidate the reconsideration with its statutorily required periodic review.[10]

In light of the EPA's decision to consolidate the reconsideration with the current review, the D.C. Circuit proceeded with the litigation on the 2008 O_3 NAAQS decision. On July 23, 2013, the court upheld the EPA's 2008 primary standard, but remanded the 2008 secondary standard to the EPA (*Mississippi v. EPA*, 744 F.3d 1334 [D.C. Cir. 2013]). With respect to the primary standard, the court first rejected arguments that the EPA should not have lowered the level of the existing primary standard, holding that the EPA reasonably determined that the existing primary standard was not requisite to protect public health with an adequate margin of safety, and consequently required revision. The court went on to reject arguments that the EPA should have adopted a more stringent primary standard. With respect to the secondary standard,

[8] The press release of this announcement is available at: *https://archive.epa.gov/epapages/newsroom_archive/newsreleases/85f90b7711acb0c88525763300617d0d.html*.

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

[10] This rulemaking, completed in 2015, concluded the reconsideration process.

the court held that the EPA's explanation for the setting of the secondary standard identical to the revised 8-hour primary standard was inadequate under the CAA because the EPA had not adequately explained how that standard provided the required public welfare protection.

At the time of the court's decision, the EPA had already completed significant portions of its next statutorily required periodic review of the O_3 NAAQS. This review had been formally initiated in 2008 with a call for information in the *Federal Register* (73 FR 56581, September 29, 2008). In late 2014, based on the ISA, Risk and Exposure Assessments (REAs) for health and welfare, and PA[11] developed for this review, the EPA proposed to revise the 2008 primary and secondary standards by reducing the level of both standards to within the range of 0.070 to 0.065 ppm (79 FR 75234, December 17, 2014).

The EPA's final decision in this review was published in October 2015, establishing the now-current standards (80 FR 65292, October 26, 2015). In this decision, based on consideration of the health effects evidence on respiratory effects of O_3 in at-risk populations, the EPA revised the primary standard from a level of 0.075 ppm to a level of 0.070 ppm, while retaining all the other elements of the standard (80 FR 65292, October 26, 2015). The EPA's decision on the level for the standard was based on the weight of the scientific evidence and quantitative exposure/risk information. The level of the secondary standard was also revised from 0.075 ppm to 0.070 ppm based on the scientific evidence of O_3 effects on welfare, particularly the evidence of O_3 impacts on vegetation, and quantitative analyses available in the review.[12] The other elements of the standard were retained. This decision on the secondary standard also incorporated the EPA's response to the D.C. Circuit's remand of the 2008 secondary standard in *Mississippi v. EPA*, 744 F.3d 1344 (D.C. Cir. 2013). The 2015 revisions to the NAAQS were accompanied by revisions to the data handling procedures, and the ambient air monitoring requirements[13] (80 FR 65292, October 26, 2015).[14]

After publication of the final rule, a number of industry groups, environmental and health organizations, and certain states filed petitions for judicial review in the D.C. Circuit. The industry and state petitioners argued that the revised standards were too stringent, while the

[11] 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).

[12] These standards, set in 2015, are specified at 40 CFR 50.19.

[13] The current federal regulatory measurement methods for O_3 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 O_3 monitoring network requirements are specified in 40 CFR 58.

[14] 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.

environmental and health petitioners argued that the revised standards were not stringent enough to protect public health and welfare as the Act requires. On August 23, 2019, the court issued an opinion that denied all the petitions for review with respect to the 2015 primary standard while also concluding that the EPA had not provided a sufficient rationale for aspects of its decision on the 2015 secondary standard and remanding that standard to the EPA (*Murray Energy Corp. v. EPA*, 936 F.3d 597 [D.C. Cir. 2019]).

In the August 2019 decision, the court additionally addressed arguments regarding considerations of background O_3 concentrations, and socioeconomic and energy impacts. With regard to the former, the court rejected the argument that the EPA was required to take background O_3 concentrations into account when setting the NAAQS, holding that the text of CAA section 109(b) precluded this interpretation because it would mean that if background O_3 levels in any part of the country exceeded the level of O_3 that is requisite to protect public health, the EPA would be obliged to set the standard at the higher nonprotective level (*id.* at 622-23). Thus, the court concluded that the EPA did not act unlawfully or arbitrarily or capriciously in setting the 2015 NAAQS without regard for background O_3 (*id.* at 624). Additionally, the court denied arguments that the EPA was required to consider adverse economic, social, and energy impacts in determining whether a revision of the NAAQS was "appropriate" under section 109(d)(1) of the CAA (*id.* at 621-22). The court reasoned that consideration of such impacts was precluded by *Whitman's* holding that the CAA "unambiguously bars cost considerations from the NAAQS-setting process" (531 U.S. at 471, summarized in section 1.2 above). Further, the court explained that section 109(d)(2)(C)'s requirement that CASAC advise the EPA "of any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance" of revised NAAQS had no bearing on whether costs are to be considered in setting the NAAQS (*Murray Energy Corp. v. EPA*, 936 F.3d at 622). Rather, as described in *Whitman* and discussed further in section 1.2 above, most of that advice would be relevant to implementation but not standard setting (*id.*).

1.4 CURRENT O_3 NAAQS REVIEW

In May 2018, the Administrator directed his Assistant Administrators to initiate this review of the O_3 NAAQS (Pruitt, 2018). In conveying this direction, the Administrator further directed the EPA staff to expedite the review, implementing an accelerated schedule aimed at completion of the review within the statutorily required period (Pruitt, 2018). Accordingly, the EPA took immediate steps to proceed with the review. In June 2018, the EPA announced the initiation of the current periodic review of the air quality criteria for photochemical oxidants and the O_3 NAAQS and issued a call for information in the *Federal Register* (83 FR 29785, June 26, 2018). Two types of information were called for: information regarding significant new O_3

research to be considered for the ISA for the review, and policy-relevant issues for consideration in this NAAQS review. Based in part on the information received in response to the call for information, the EPA developed a draft IRP which was made available for consultation with the CASAC and for public comment (83 FR 55163, November 2, 2018; 83 FR 55528, November 6, 2018). Comments from the CASAC (Cox, 2018) and the public were considered in preparing the final IRP (U.S. EPA, 2019).

Under the plan outlined in the IRP and consistent with revisions to the process identified by the administrator in his 2018 memo directing initiation of the review, the current review of the O_3 NAAQS is progressing on an accelerated schedule (Pruitt, 2018). The EPA is incorporating a number of efficiencies in various aspects of the review process, as summarized in the IRP, to support completion within the statutorily required period (Pruitt, 2018). As one example of such an efficiency, rather than produce two separate documents, the exposure and risk analyses for the primary standard are included as an appendix in the PA, along with a number of other technical appendices. The draft PA (including these analyses as appendices) was reviewed by the CASAC and made available for public comment while the draft ISA was also being reviewed by the CASAC and was available for public comment (84 FR 50836, September 26, 2019; 84 FR 58711, November 1, 2019).[15] The CASAC was assisted in its review by a pool of consultants with expertise in a number of fields (84 FR 38625, August 7, 2019). The approach employed by the CASAC in utilizing outside technical expertise represents an additional modification of the process from past reviews. Rather than join with some or all of the CASAC members in a CASAC review panel as has been common in other NAAQS reviews in the past, in this O_3 NAAQS review (and also in the recent CASAC review of the PA for the particulate matter NAAQS), the consultants comprised a pool of expertise that CASAC members drew on through the use of specific questions, posed in writing prior to the public meeting, regarding aspects of the documents being reviewed, obtaining subject matter expertise for its document review in a focused, efficient and transparent manner.

The CASAC discussed its review of both the draft ISA and the draft PA over three days at a public meeting in December 2019 (84 FR 58713, November 1, 2019).[16] The CASAC discussed its draft letters describing its advice and comments on the documents in a public

[15] 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.

[16] While simultaneous reviews of first drafts of both documents has not been usual in past reviews, there have been occurrences of the CASAC review of a draft PA (or draft REA when process involved policy assessment being included within the REA document) simultaneous with review of a second (or later) draft ISA (e.g., 73 FR 19835, April 11, 2008; 73 FR 34739, June 18, 2008; 77 FR 64335, October 19, 2020; 78 FR 938, January 7, 2013).

teleconference in early February 2020 (85 FR 4656; January 27, 2020). The letters to the Administrator conveying the CASAC advice and comments on the draft PA and draft ISA were released later that month (Cox, 2020a, Cox, 2020b).

The letters from the CASAC and public comment on the draft ISA and draft PA have informed completion of the final documents and further inform development of the Administrator's proposed decision in the review. Comments from the CASAC on the draft ISA have been considered by the EPA and led to a number of revisions in developing the final document. The CASAC review and the EPA's consideration of CASAC comments are described in Appendix 10, section 10.4.5 of the final ISA. As noted by Administrator Wheeler noted, in his response to the CASAC letter conveying its review, "for those comments and recommendations that are more significant or cross-cutting and which were not fully addressed, the Agency will develop a plan to incorporate these changes into future Ozone ISAs as well as ISAs for other criteria pollutant reviews." The ISA was completed and made available to the public in April 2020 (85 FR 21849, April 20, 2020).

The CASAC comments additionally provided a number of comments intended to improve the PA. For example, it recommended that the process followed in the current review, including its distinctions from prior reviews, be clearly summarized, as has been done in the presentation in this section of the PA. Further, the CASAC and public comment also provided comments on improving the clarity and other aspects of the presentations of air quality information in Chapter 2, the scientific evidence of health and welfare effects in Chapters 3 and 4, and the quantitative exposure and risk analyses, presented in detail in Appendices 3C and 3D. These comments have been considered in completing these sections of this document. For example, the summary of the health effects evidence has been strengthened, consistent with the final ISA, including further recognition of evidence of O_3-related inflammatory response and susceptibility of people with asthma. Additions to the quantitative exposure and risk analyses are summarized in Appendix 3D, section 3D.1. And, additional data presentations on O_3 precursors trends and regional emissions patterns have been added to Chapter 2.

The CASAC advice to the Administrator regarding the O_3 standards has also been described and considered in this document. Advice on the primary standard is summarized in section 3.5.3 and considered in the conclusions discussed in section 3.5.4. For the secondary standard, the CASAC advice is summarized in section 4.5.2. and considered in the PA conclusions discussed in section 4.5.3.

The current timeline for the review of the standards projects a proposed decision near the middle of 2020. Materials upon which this proposed decision is based, including the documents

described above, will be made available to the public in the docket for the review.[17] Following a public comment period on the proposed decision, a final decision in the review is projected for late in 2020.

[17] The docket for the current O_3 NAAQS review is identified as EPA-HQ-OAR-2018-0279. This docket has incorporated the ISA docket (EPA-HQ-ORD-2018-0274) by reference. Both dockets are publicly accessible at www.regulations.gov.

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2 AIR QUALITY

This chapter begins with an overview of O_3 and other photochemical oxidants in the atmosphere (section 2.1). Subsequent sections summarize the sources and emissions of O_3 precursors (section 2.2), ambient air monitoring and data handling conventions for determining whether the standards are met (section 2.3), O_3 concentrations measured in the U.S. ambient air (section 2.4), and available evidence and information related to background O_3 in the U.S. (section 2.5). These focus primarily on tropospheric O_3 and surface-level concentrations occurring in ambient air[1].

2.1 O_3 AND PHOTOCHEMICAL OXIDANTS IN THE ATMOSPHERE

O_3 is one of a group of photochemical oxidants formed in the troposphere[2] 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 O_3 and other oxidants, such as peroxyacetyl nitrate (PAN) and hydrogen peroxide, form in polluted areas by atmospheric reactions involving two main classes of precursor pollutants: volatile organic compounds (VOCs) and nitrogen oxides (NO_X). This occurs especially during the summer, as a result of the photolysis of primary pollutants such as nitrogen dioxide (NO_2). The reaction is disrupted by the presence of VOCs, the radical that results from methane (CH_4) oxidation; or a reaction between carbon monoxide (CO) and the hydroxyl radical (OH) in the atmosphere. Thus, the substances NOx, VOC, CH_4 and CO are considered to be the primary precursors of tropospheric O_3. The formation of O_3, 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).

Rather than varying directly with emissions of its precursors, O_3 changes in a nonlinear fashion with the concentrations of its precursors (2013 ISA, section 3.2.4). Emissions of NO_X lead to both the formation and destruction of O_3, depending on the local quantities of NO_X, VOCs, radicals, and sunlight. O_3 chemistry is often described in terms of which precursors most

[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 O_3 to the troposphere is transport from the stratosphere, as described in section 2.5.1.1 below.

directly impact formation rates. A NOx-limited regime indicates that O_3 concentrations will decrease in response to decreases in ambient NOx concentrations and vice-versa. These conditions tend to occur when NOx concentrations are generally low compared to VOC concentrations and during warm, sunny conditions when NOx photochemistry is relatively fast. NOx-limited conditions are more common during daylight hours, in the summertime, in suburban and rural areas, and in portions of the country with high biogenic VOC emissions like the Southeast. In contrast, NOx-saturated conditions (also referred to as VOC-limited or radical-limited) indicate that O_3 will increase as a result of NOx reductions but will decrease as a result of VOC reductions (2013 ISA, section 3.2; 2006 AQCD, chapter 2). NOx-saturated conditions occur at times when and at locations with lower levels of available sunlight, resulting in slower photochemical formation of O_3, and when NOx concentrations are in excess compared to VOC concentrations. NOx-saturated conditions are more common during nighttime hours, in the wintertime, and in densely populated urban areas or industrial plumes. These varied relationships between precursor emissions and O_3 chemistry result in localized areas in which O_3 concentrations are suppressed compared to surrounding areas, but which contain NO_2 that contributes to subsequent O_3 formation further downwind (2013 ISA, section 3.2.4). Consequently, O_3 response to reductions in NO_X emissions is complex and may include decreases in O_3 concentrations at some times and locations and increases in O_3 concentrations at other times and locations. Over the past decade, there have been substantial decreases in NOx emissions in the U.S. (see Figure 2-2) and many locations have transitioned from NOx-saturated to NOx-limited (Jin et al., 2017) during times of year that are conducive to O_3 formation (generally summer). As these NO_X emissions reductions have occurred, lower O_3 concentrations have generally increased while the higher O_3 concentrations have generally decreased, resulting in a compressed O_3 distribution, relative to historical conditions (ISA, Appendix 1, section 1.7).

Prior to 1979, the indicator for the NAAQS for photochemical oxidants was total photochemical oxidants (36 FR 8186, April 30, 1971). Early ambient air monitoring indicated similarities between O_3 measurements and the photochemical oxidant measurements, as well as reduced precision and accuracy of the latter (U.S. EPA, 1978). To address these issues, the EPA established O_3 as the indicator for the NAAQS for photochemical oxidants in 1979 (44 FR 8202, February 8, 1979), and it is currently the only photochemical oxidant other than nitrogen dioxide that is routinely monitored in a national ambient air monitoring network.

O_3 is present not only in polluted urban atmospheres, but throughout the troposphere, even in remote areas of the globe. The same basic processes involving sunlight-driven reactions of NO_X, VOCs, and CO contribute to O_3 formation throughout the troposphere. These processes also lead to the formation of other photochemical products, such as PAN, HNO_3, and H_2SO_4, and

to other gaseous compounds, such as HCHO and other carbonyl compounds, as well as a number of particulate compounds (ISA, Appendix 1, section 1.4; 2013 ISA, section 3.2).

As mentioned above, the formation of O_3 from precursor emissions is also affected by meteorological parameters such as the intensity of sunlight and atmospheric mixing (2013 ISA, section 3.2). Major episodes of high O_3 concentrations in the eastern U.S. are often associated with slow-moving high-pressure systems which can persist for several days. High pressure systems during the warmer seasons are associated with the sinking of air, resulting in warm, generally cloudless skies, with light winds. The sinking of air results in the development of stable conditions near the surface which inhibit or reduce the vertical mixing of O_3 precursors, concentrating them near the surface. Photochemical activity involving these precursors is enhanced because of higher temperatures and the availability of sunlight during the warmer seasons. In the eastern U.S., concentrations of O_3 and other photochemical oxidants are determined by meteorological and chemical processes extending typically over areas of several hundred thousand square kilometers. Therefore, O_3 episodes are often regarded as regional in nature, although more localized episodes often occur in some areas, largely the result of local pollution sources during summer, e.g., Houston, TX (2013 ISA, section 2.2.1; Webster et al., 2007). In addition, in some parts of the U.S. (e.g., Los Angeles, CA), mountain barriers limit O_3 dispersion and result in a higher frequency and duration of days with elevated O_3 concentrations (2013 ISA, section 3.2).

More recently, high O_3 concentrations of up to 150 parts per billion (ppb)[4] have been measured during the wintertime in two western U.S. mountain basins (ISA, Appendix 1, section 1.4.1). Wintertime mountain basin O_3 episodes occur on cold winter days with low wind speeds, clear skies, substantial snow cover, extremely shallow boundary layers driven by strong temperature inversions, and substantial precursor emissions activity from the oil and gas sector. The results of recent modeling studies suggest that photolysis of VOCs provides the source of reactive chemical species (radicals) needed to initiate the chemistry driving these wintertime O_3 episodes. This mechanism is markedly different from the chemistry driving summertime O_3 formation, which is initiated with the photolysis of NO_2 followed by the formation of the OH radicals (ISA, Appendix 1, section 1.4.1).

O_3 concentrations in a region are affected both by local formation and by transport of O_3 and its precursors from upwind areas. O_3 transport occurs on many spatial scales including local transport within urban areas, regional transport over large regions of the U.S., and long-range transport which may also include international transport. In addition, O_3 can be transferred into

[4] Although the standards are specified in ppm (e.g., as described in Chapter 1), the units, ppb, are commonly used in describing O_3 concentrations throughout this document, with 0.070 ppm being equivalent to 70 ppb.

the troposphere from the stratosphere, which is rich in naturally occurring O_3, through stratosphere-troposphere exchange (STE). These intrusions usually occur behind cold fronts, bringing stratospheric air with them and typically affect O_3 concentrations in higher elevation areas (e.g. > 1500 m) more than areas at lower elevations, as discussed in section 2.5.3.2 (ISA, Appendix 1, section 1.3.2.1; 2013 ISA, section 3.4.1.1).

2.2 SOURCES AND EMISSIONS OF O_3 PRECURSORS

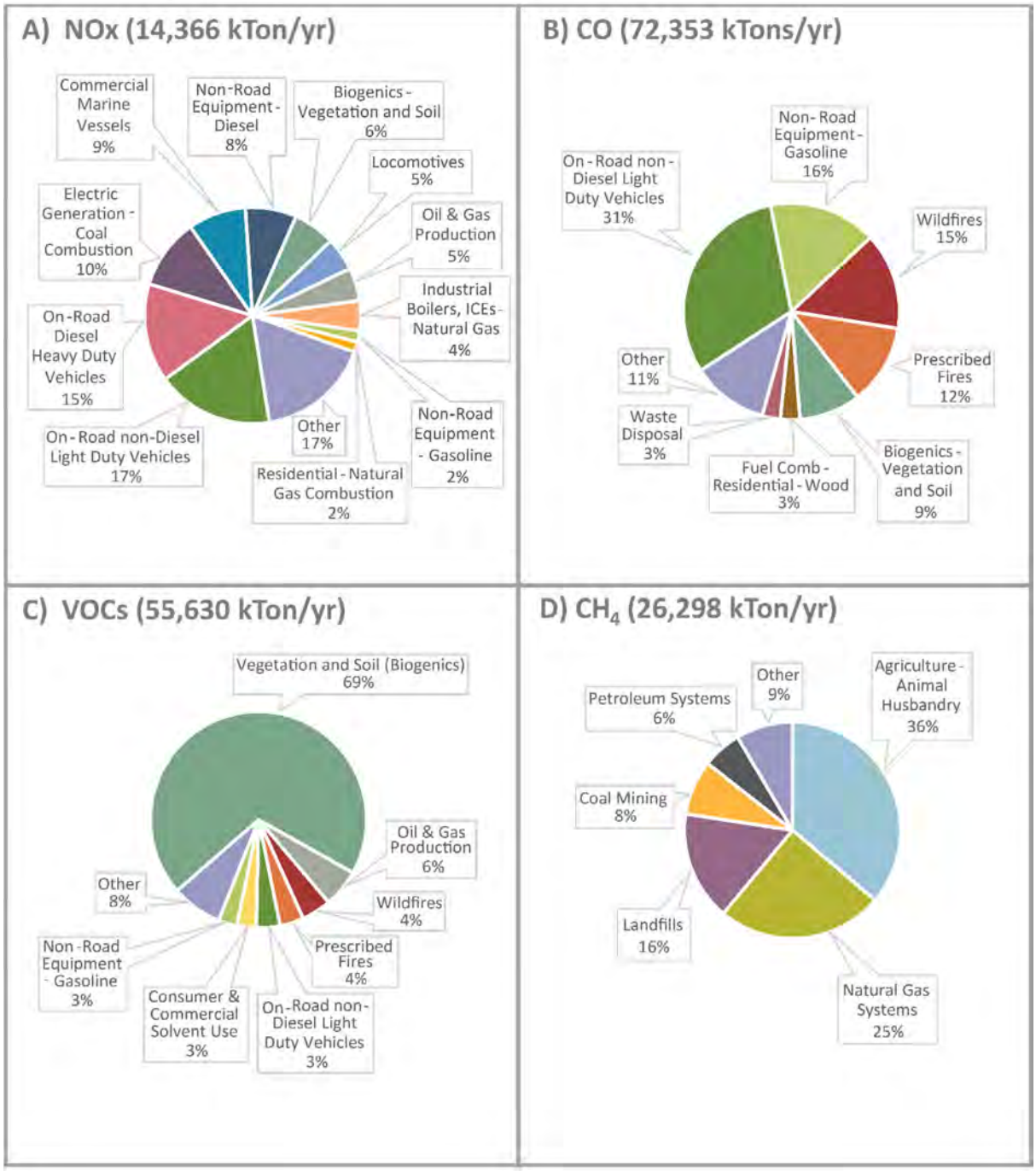
Sources of emissions of O_3 precursor compounds can be divided into anthropogenic and natural source categories, with natural sources further divided into emissions from biological processes of living organisms (e.g., plants, microbes, and animals) and emissions from chemical or physical processes (e.g., biomass burning, lightning, and geogenic sources). Anthropogenic emissions associated with combustion processes, including mobile sources and power plants, account for the majority of U.S. NO_X and CO emissions (Figure 2-1 and Figure 2-2). Emissions of these chemicals from mobile sources have declined appreciably since 2002 (Figure 2-2). Anthropogenic sources are also important for VOC emissions, though in some locations and times of the year (e.g., southern states during summer) the majority of VOC emissions come from vegetation (2013 ISA, section 3.2.1)[5]. In practice, the distinction between natural and anthropogenic sources is often unclear, as human activities directly or indirectly affect emissions from what would have been considered natural sources during the preindustrial era. Thus, precursor emissions from plants, animals, and wildfires could be considered either natural or anthropogenic, depending on whether emissions result from agricultural practices, forest management practices, lightning strikes, or other types of events. Additional challenges are presented because much O_3 results from reactions between anthropogenic and natural precursors (ISA, Appendix 1, section 1.8.1.2).

The National Emissions Inventory (NEI) is a comprehensive and detailed estimate of air emissions of criteria pollutants, precursors to criteria pollutants, and hazardous air pollutants from air emissions sources (U.S. EPA, 2018c). The NEI is released every three years based primarily upon data provided by State, Local, and Tribal air agencies for sources in their jurisdictions and supplemented by data developed by the US EPA. The NEI is built using the EPA's Emissions Inventory System (EIS) first to collect the data from State, Local, and Tribal air agencies and then to blend that data with other data sources.[6]

[5] It should be noted that the definition of VOCs used in this section does not include CH_4 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*.

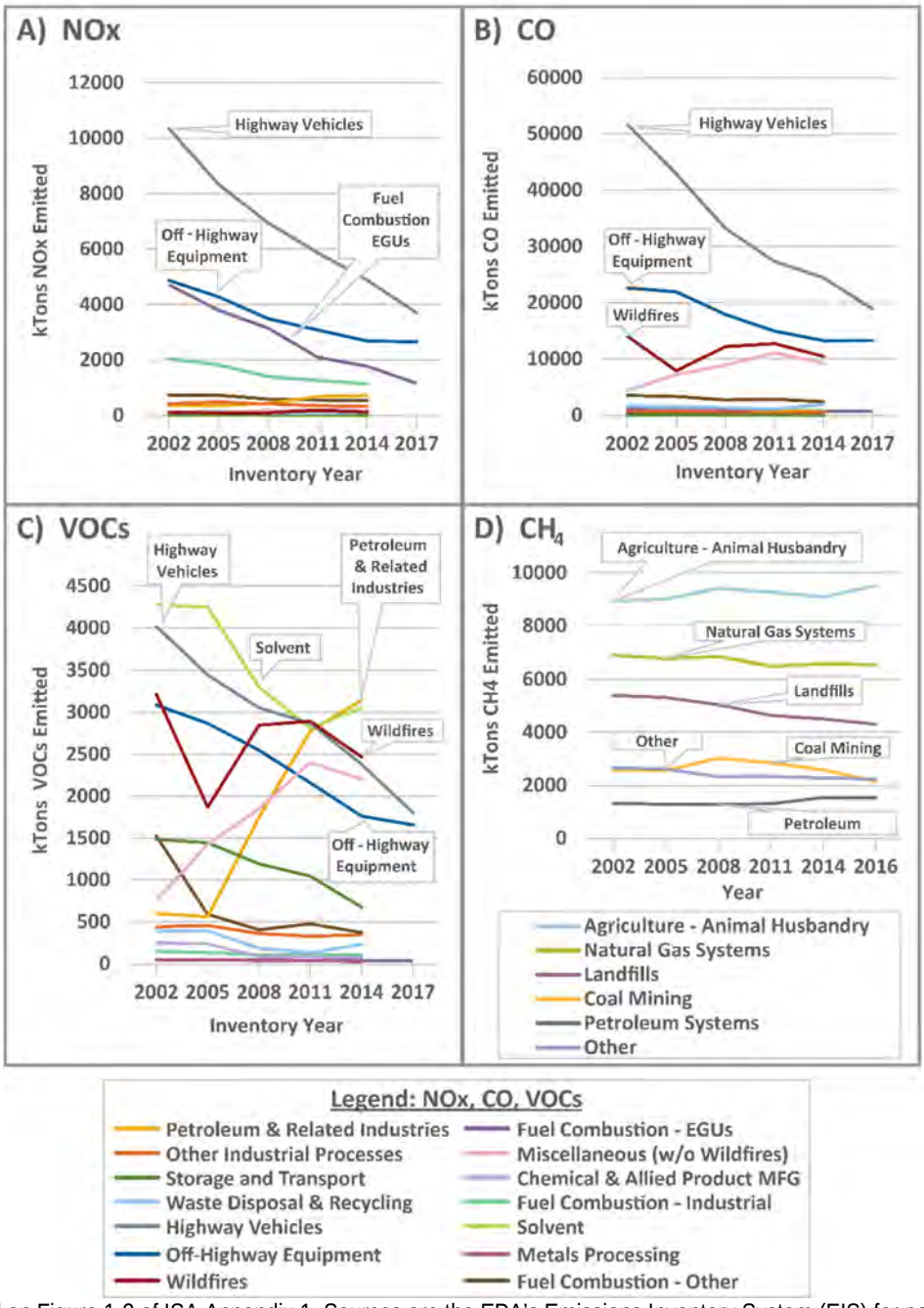
[6] More details are available from: *https://www.epa.gov/enviro/nei-overview*.

Accuracy in an emissions inventory reflects the extent to which the inventory represents the actual emissions that occurred. Anthropogenic emissions of air pollutants result from a variety of sources such as power plants, industrial sources, motor vehicles and agriculture. The emissions from any individual source typically varies in both time and space. For the thousands of sources that make up the NEI, there is uncertainty in one or both of these factors. For some sources, such as power plants, direct emission measurements enable the emission factors derived from them to be more certain than sources without such direct measurements. However, it is not practically possible to directly monitor each of the emission sources individually and, therefore, emission inventories necessarily contain assumptions, interpolation and extrapolation from a limited set of sample data (U.S. EPA, 2018c).



Source: Based on Figure 1-2 of ISA Appendix 1. Sources are the 2014 National Emissions Inventory, version 2 (U.S. EPA, 2018c) for panels A-C, and the *Inventory of U.S. Greenhouse Gas Emissions and Sinks: 1990-2016* (U.S. EPA, 2018b) for panel D. Categories contributing less than 2% each have been summed and are represented by the "other" category.

Figure 2-1. U.S. O_3 precursor emissions by sector: A) NO_X; B) CO; C) VOCs; D) CH_4.

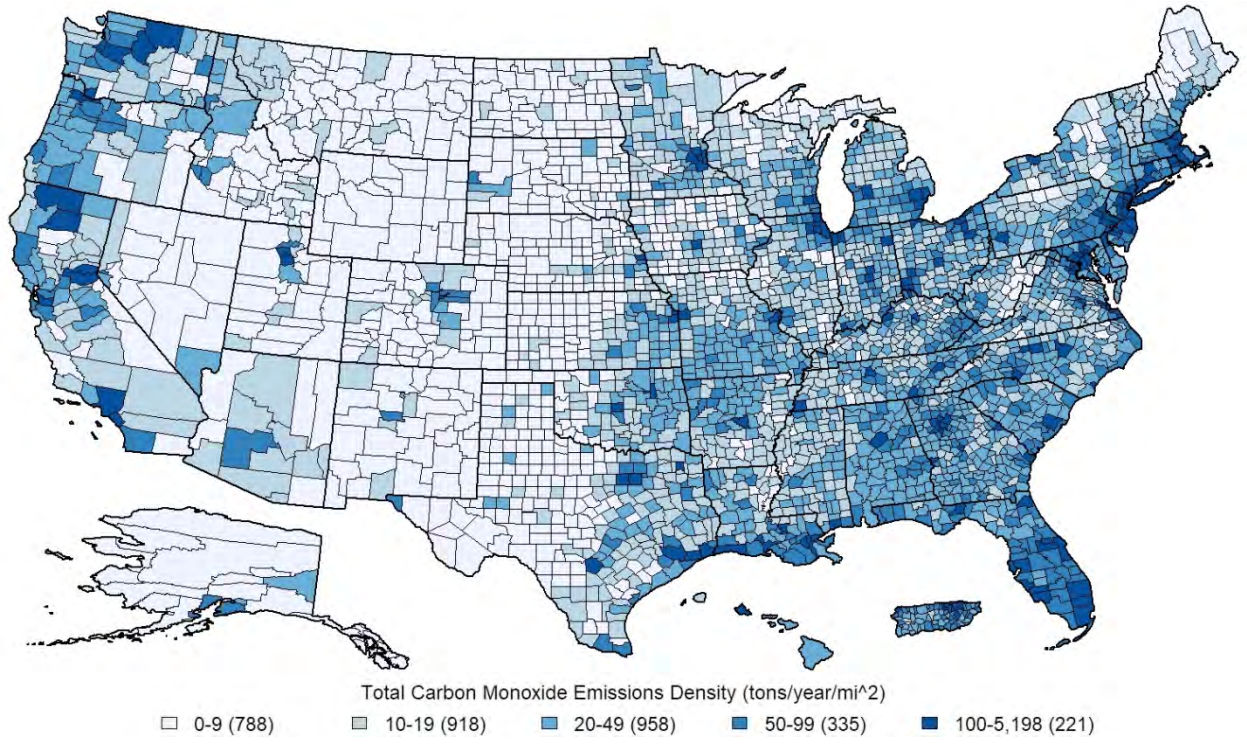


Source: Based on Figure 1-3 of ISA Appendix 1. Sources are the EPA's Emissions Inventory System (EIS) for panels A-C, and the *Inventory of U.S. Greenhouse Gas Emissions and Sinks: 1990-2016* (U.S. EPA, 2018a) for panel D. Estimates for 2017 come from air pollutant emissions trends estimates available on the EPA's website (*https://www.epa.gov/air-emissions-inventories/air-pollutant-emissions-trends-data*). Categories contributing less than 2% each have been summed and are represented by the "other" category. Sources shown generate 90% or more of the estimated U.S. anthropogenic emissions.

Figure 2-2. U.S. anthropogenic O_3 precursor emission trends for: A) NO_X; B) CO; C) VOCs; and D) CH_4.

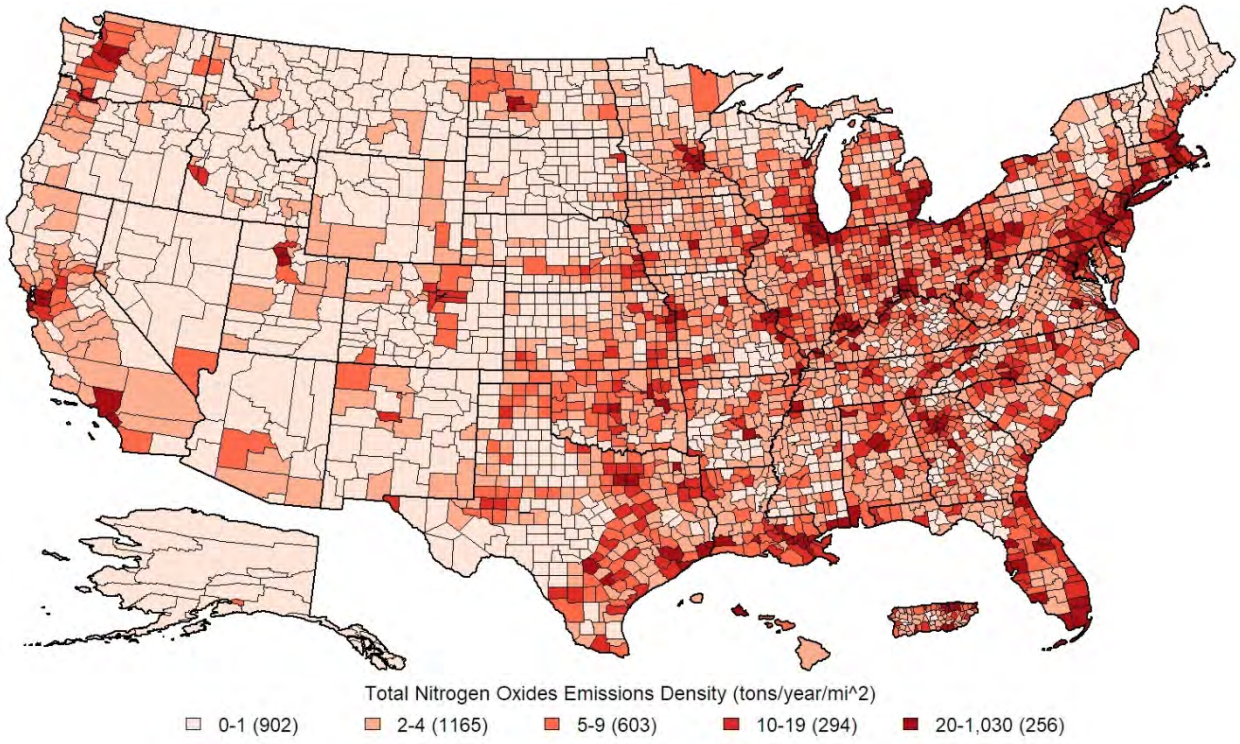
Figure 2-3, Figure 2-4 and Figure 2-5 show county-level estimates of U.S. emissions densities (in tons/year/mi^2) for CO, NO_X, and VOCs, respectively. In general, CO and NOx emissions tend to be highest in urban areas which typically have the most anthropogenic sources, however, CO emissions may be higher in some rural areas due to fires, and similarly NO_X emissions may be higher in some rural areas due to sources such as electricity generation, oil and gas extraction, and traffic along major highways. While there are some significant anthropogenic sources of VOC emissions in urban areas, in rural areas the vast majority of VOC emissions come from plants and trees (biogenics), particularly in the southeastern U.S. In other areas of the U.S., such as the Great Plains region and parts of the inter-mountain west, areas with higher levels of VOC emissions are largely due to oil and gas extraction (U.S. EPA, 2018c).

It should be noted that O_3 levels in a given area are impacted by both local emissions that form O_3 in the area as well as remote emissions that form O_3 which is then transported into the area. Biogenic VOC emissions that lead to O_3 formation may vary greatly depending on the type and amount of vegetation, which is generally much lower in urban areas than in rural areas. However, biogenic VOC emissions that are upwind of an urban area can have a significant impact on urban O_3 levels. Thus, while the county-level maps shown in Figure 2-3, Figure 2-4 and Figure 2-5 illustrate the variability in precursor emissions in the U.S., it is not sufficient to look only at the patterns in local emissions when considering the impact on O_3 concentrations.



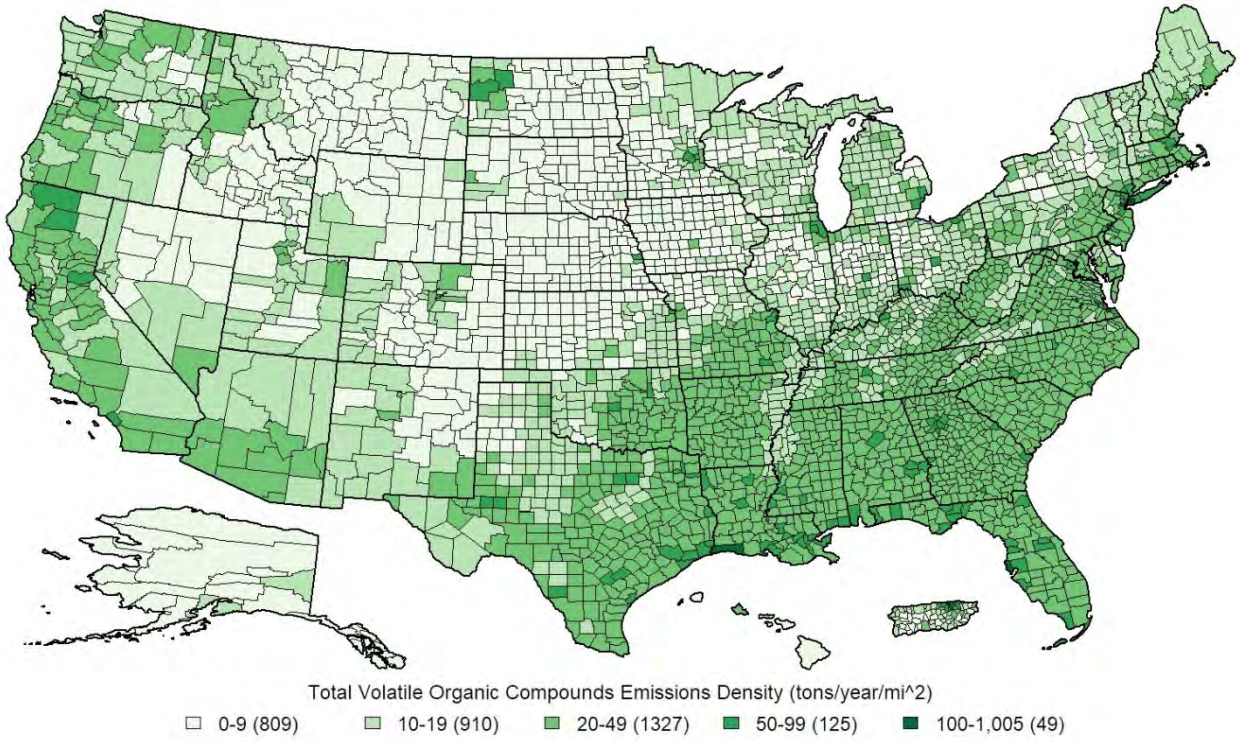
Source: 2014 National Emissions Inventory, version 2 (US EPA, 2018c; data downloaded from *https://edap.epa.gov/public/extensions/nei_report_2014/dashboard.html*)

Figure 2-3. U.S. county-level CO emissions density estimates (tons/year/mi^2) for 2014.



Source: 2014 National Emissions Inventory, version 2 (US EPA, 2018c; data downloaded from *https://edap.epa.gov/public/extensions/nei_report_2014/dashboard.html*)

Figure 2-4. U.S. county-level NO_X emissions density estimates (tons/year/mi^2) for 2014.



Source: 2014 National Emissions Inventory, version 2 (US EPA, 2018c; data downloaded from *https://edap.epa.gov/public/extensions/nei_report_2014/dashboard.html*)

Figure 2-5. U.S. county-level VOC emissions density estimates (tons/year/mi^2) for 2014.

2.3 AMBIENT AIR MONITORING AND DATA HANDLING CONVENTIONS

2.3.1 Ambient Air Monitoring Requirements and Monitoring Networks

State and local environmental agencies operate O_3 monitors at state or local air monitoring stations (SLAMS) as part of the SLAMS network. The requirements for the SLAMS network depend on the population and most recent O_3 design values[7] in the area. The minimum number of O_3 monitors required in a metropolitan statistical area (MSA) ranges from zero for areas with a population less than 350,000 and no recent history of an O_3 design value greater than 85 percent of the level of the standard, to four for areas with a population greater than 10 million and an O_3 design value greater than 85 percent of the standard level.[8] Within an O_3 monitoring network, at least one site for each MSA must be designed to record the maximum concentration for that particular metropolitan area. Siting criteria for SLAMS includes horizontal and vertical inlet probe placement; spacing from minor sources, obstructions, trees, and roadways; inlet probe material; and sample residence times.[9] Adherence to these criteria ensures uniform collection and comparability of O_3 data. Since the highest O_3 concentrations tend to be associated with a particular season for various locations, the EPA requires O_3 monitoring during specific O_3 monitoring seasons (shown in Figure 2-6) which vary by state from five months (May to September in Oregon and Washington) to all twelve months (in 11 states), with the most common season being March to October (in 27 states).[10]

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

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[7] 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.

[8] The SLAMS minimum monitoring requirements to meet the O_3 design criteria are specified in 40 CFR Part 58, Appendix D. The minimum O_3 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).

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

[10] The required O_3 monitoring seasons for each state are listed in Table D-3 of Appendix D to 40 CFR Part 58.

[11] The current FRM for O_3 (established in 2015) is a chemiluminescence method, which is fully described in Appendix D to 40 CFR Part 50.

Ambient air quality data and associated quality assurance (QA) data are reported to the EPA via the Air Quality System (AQS). Data are reported quarterly and must be submitted to AQS within 90 days after the end of the quarterly reporting period. Each monitoring agency is required to certify data that is submitted to AQS from the previous year. The data are certified, taking into consideration any QA findings, and a data certification letter is sent to the EPA Regional Administrator. Data must be certified by May 1st of the following year. Data collected by FRM or FEM monitors that meet the QA requirements must be certified.[12] To provide decision makers with an assessment of data quality, the EPA's QA group derives estimates of both precision and bias for O_3 and the other gaseous criteria pollutants from quality control (QC) checks using calibration gas, performed at each site by the monitoring agency. The data quality goal for precision and bias is 7 percent.[13]

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

[13] 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*.

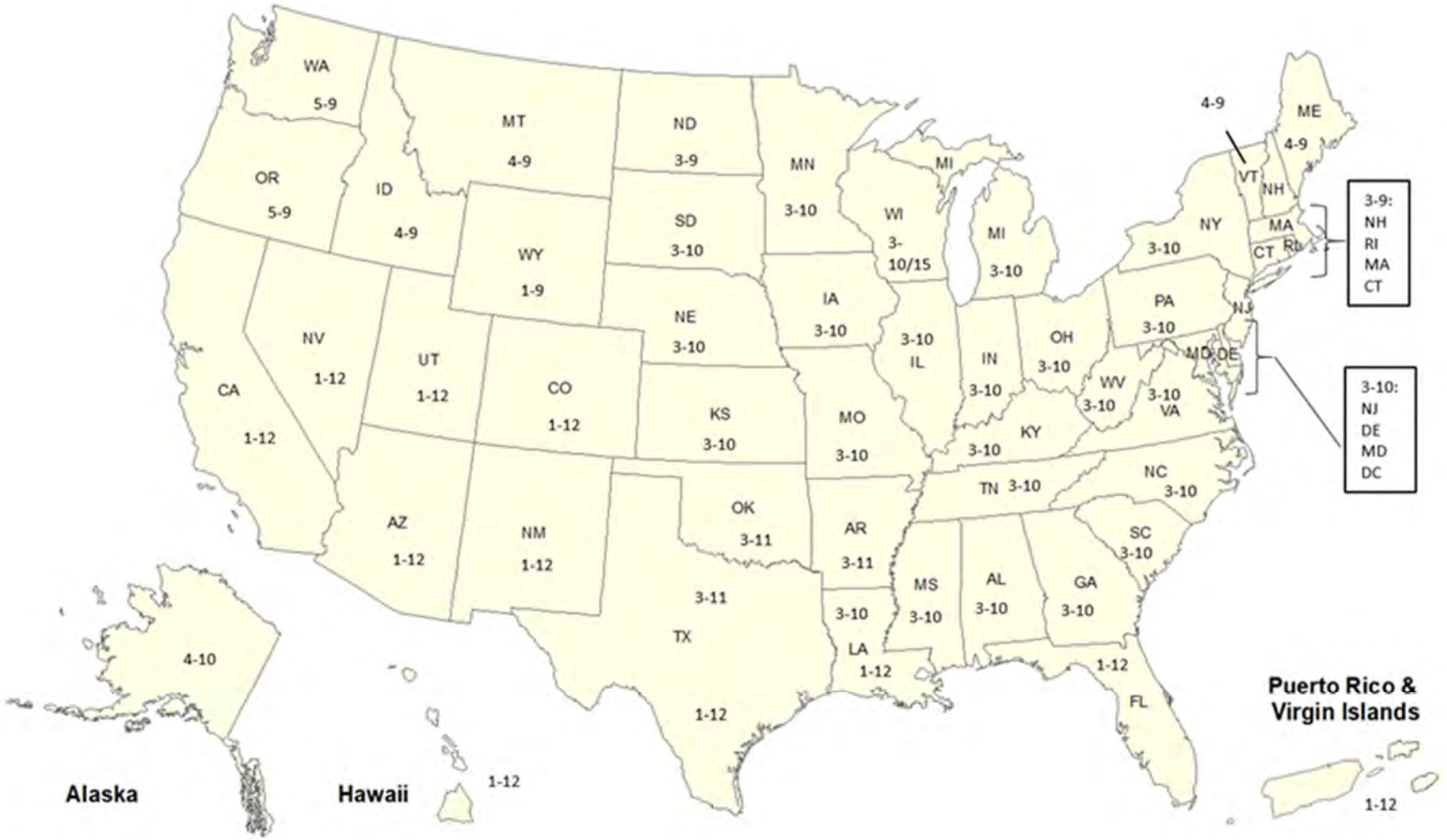


Figure 2-6. Current O_3 monitoring seasons in the U.S. Numbers in each state indicate the months of the year the state is required to monitor for O_3 (e.g., 3-10 means O_3 monitoring is required from March through October).

In 2018, there were over 1,300 federal, state, local, and tribal ambient air monitors reporting O_3 concentrations to the EPA. Figure 2-7 shows the locations of such monitoring sites that reported data to the EPA at any time during the 2016-2018 period. Nearly 80% of this network are SLAMS monitors operated by state and local governments to meet regulatory requirements and provide air quality information to public health agencies; these sites are largely focused on urban and suburban areas.

Two important subsets of SLAMS sites separately make up the National Core (NCore) multi-pollutant monitoring network and the Photochemical Assessment Monitoring Stations (PAMS) network. Each state is required to have at least one NCore station, and O_3 monitors at NCore sites are required to operate year-round. At each NCore site located in a MSA with a population of 1 million or more (based on the most recent census), a PAMS network site is required.[14] At a minimum, monitoring sites in the PAMS network are required to measure certain O_3 precursors during the months of June, July and August, although some precursor monitoring may be required for longer periods of time to improve the usefulness of data collected during an area's O_3 season (U.S. EPA, 2018a).

In addition to reporting O_3 concentrations, the NCore and PAMS networks provide data on O_3 precursor chemicals. The NCore sites feature co-located measurements of chemical species such as nitrogen oxide and total reactive nitrogen, along with various meteorological measurements. The additional data collected at the PAMS sites include measurements of NO_X, and a target set of VOCs. The enhanced monitoring at sites in these two networks informs our understanding of local O_3 formation.

While the SLAMS network has a largely urban and population-based focus, there are monitoring sites in other networks that can be used to track compliance with the NAAQS in rural areas. For example, the Clean Air Status and Trends Network (CASTNET) monitors are located in rural areas. There were 77 CASTNET sites operating in 2018, with most of the sites in the eastern U.S. being operated by the EPA, and most of the sites in the western U.S. being operated by the National Park Service (NPS). Finally, there are also a number of Special Purpose Monitoring Stations (SPMs), which are not required but are often operated by air agencies for short periods of time (less than 3 years) to collect data for human health and welfare studies, as well as other types of monitoring sites, including monitors operated by tribes and industrial sources. The SPMs are typically not used to assess compliance with the NAAQS.[15]

[14] 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.

[15] However, SPMs that use federal reference or equivalent methods, meet all applicable requirements in 40 CFR Part 58, and operate continuously for at least 3 years may be used to assess compliance with the NAAQS.

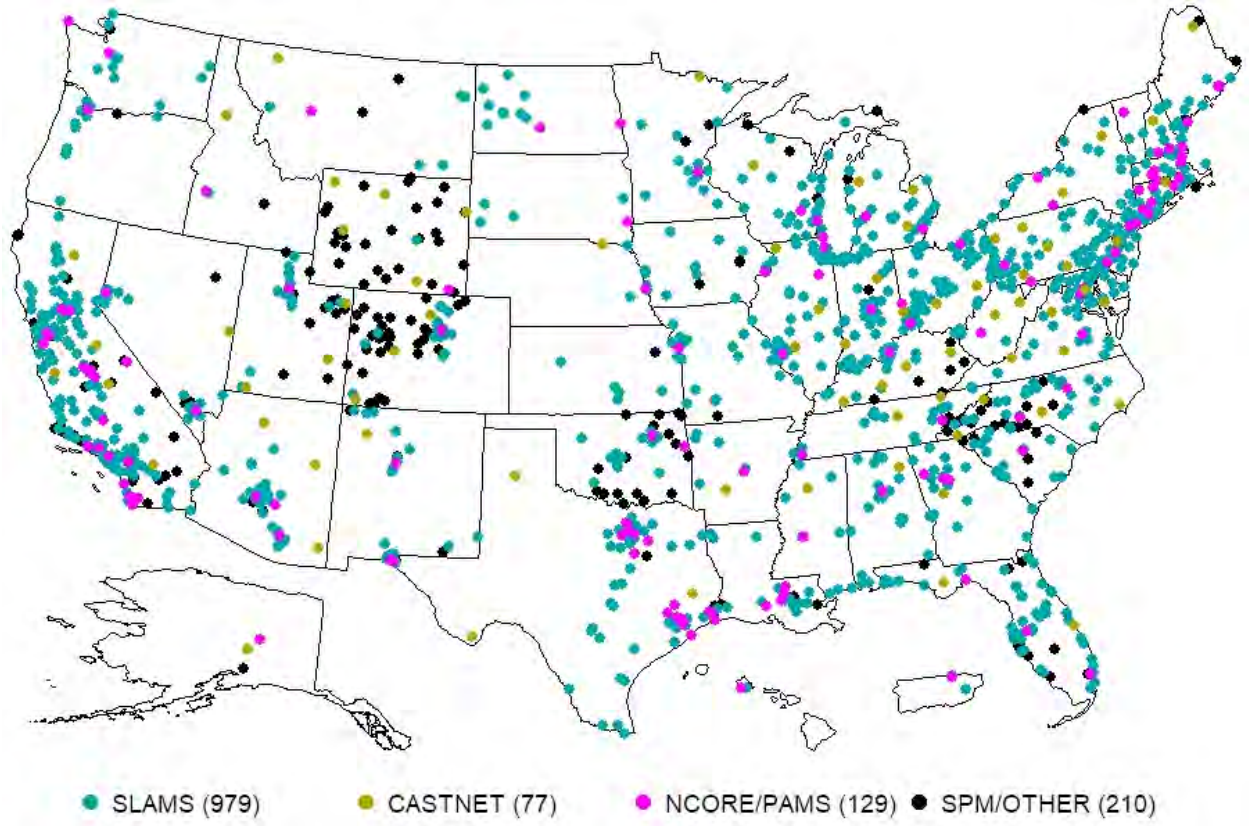


Figure 2-7. Map of U.S. ambient air O_3 monitoring sites reporting data to the EPA during the 2016-2018 period.

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

To assess whether a monitoring site or geographic area (usually a county or urban area) meets or exceeds a NAAQS, the monitoring data are analyzed consistent with the established regulatory requirements for the handling of monitoring data for the purposes of deriving a design value. A design value summarizes ambient air concentrations for an area in terms of the indicator, averaging time and form for a given standard such that its comparison to the level of the standard indicates whether the area meets or exceeds the standard. The procedures for calculating design values for the current O_3 NAAQS (established in 2015) are detailed in Appendix U to 40 CFR Part 50 and are summarized below.

Hourly average O_3 concentrations at the monitoring sites used for assessing whether an area meets or exceeds the NAAQS are required to be reported in ppm to the third decimal place, with additional digits truncated, consistent with the typical measurement precision associated with most O_3 monitoring instruments. Monitored hourly O_3 concentrations flagged by the States as having been affected by an exceptional event, having been the subject of a demonstration, and having received concurrence from the appropriate EPA Regional Office, are excluded from

design value calculations consistent with 40 CFR 50.14.[16] The hourly concentrations are used to compute moving 8-hour averages, which are stored in the first hour of each 8-hour period (e.g., the 8-hour average for the 7:00 AM to 3:00 PM period is stored in the 7:00 AM hour), and digits to the right of the third decimal place are truncated. Each 8-hour average is considered valid if 6 or more hourly concentrations are available for the 8-hour period.

Next, the daily maximum 8-hour average (MDA8) concentration for each day is identified as the highest of the 17 consecutive, valid 8-hour average concentrations beginning at 7:00 AM and ending at 11:00 PM (which includes hourly O_3 concentrations from the subsequent day). MDA8 values are considered valid if at least 13 valid 8-hour averages are available for the day, or if the MDA8 value is greater than the level of the NAAQS. Finally, the O_3 design value is calculated as the 3-year average of the annual 4th highest MDA8 value[17]. An O_3 design value less than or equal to the level of the NAAQS is considered to be valid if valid MDA8 values are available for at least 90% of the days in the O_3 monitoring season (as defined for each state and shown in Figure 2-6) on average over the 3 years, with a minimum of 75% data completeness in any individual year. Design values greater than the level of the NAAQS are always considered to be valid.

An O_3 monitoring site meets the NAAQS if it has a valid design value less than or equal to the level of the standard, and it exceeds the NAAQS if it has a design value greater than the level of the standard. A geographic area meets the NAAQS if all ambient air monitoring sites in the area have valid design values meeting the standard. Conversely, if one or more monitoring sites has a design value exceeding the standard, then the area exceeds the NAAQS.

2.4 O_3 IN AMBIENT AIR

2.4.1 Concentrations Across the U.S.

Figure 2-8 below shows a map of the O_3 design values at U.S. ambient air monitoring sites based on data from the 2016-2018 period. From the figure it is apparent that many monitoring sites have design values exceeding the current NAAQS, and that most of these sites are located in or near urban areas. The highest design values are located in California, Texas, along the shoreline of Lake Michigan, and near large urban areas in the northeastern and western U.S. There are also high design values associated with wintertime O_3 in the Uinta Basin in Utah.

[16] 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*.

[17] 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).

The lowest design values are located in the north central region of the U.S., rural parts of New England and the southeastern U.S., and along the Pacific Ocean, including Alaska and Hawaii.

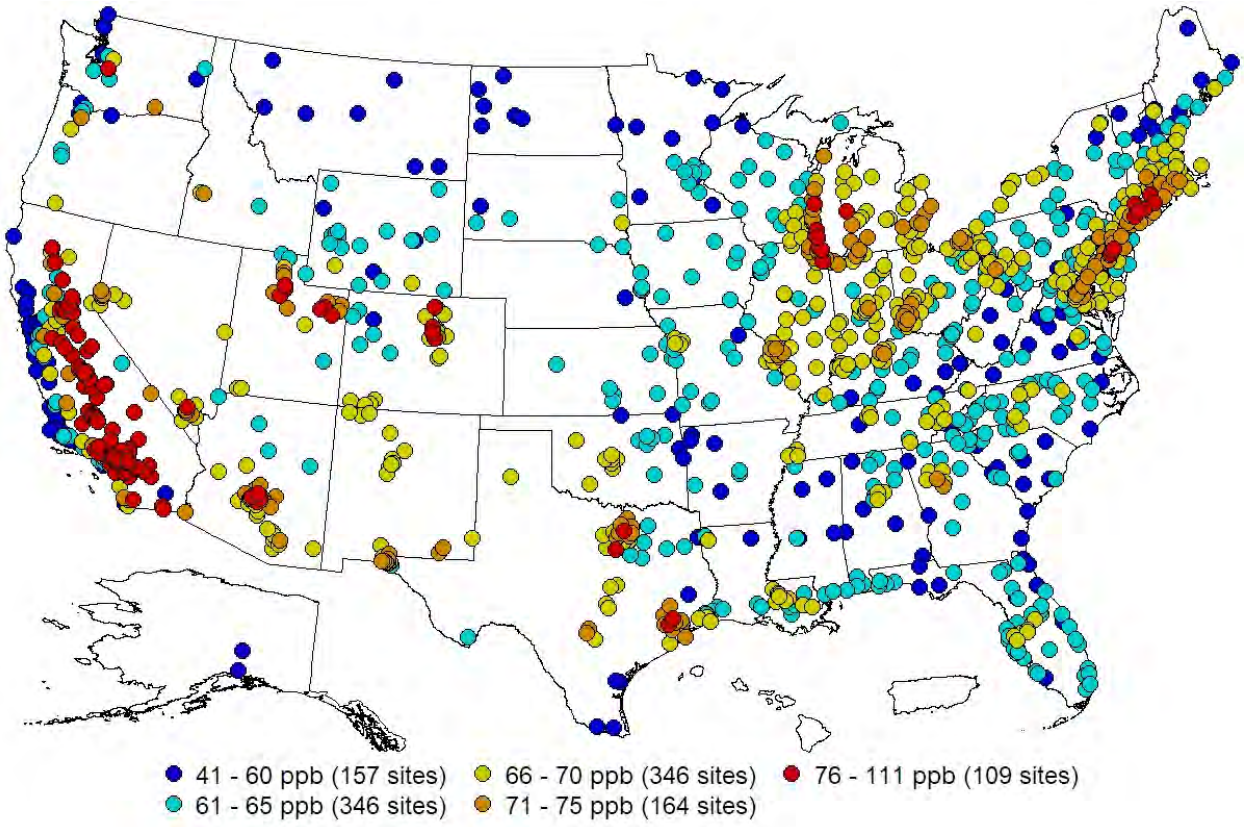


Figure 2-8. O_3 design values in ppb for the 2016-2018 period.

2.4.2 Trends in U.S. O_3 Concentrations

Figure 2-9 shows a map of the site-level trends in the O_3 design values at U.S. monitoring sites having complete data[18] from 2000-2002 through 2016-2018. The trends were computed using the Thiel-Sen estimator (Sen, 1968; Thiel, 1950), and tests for significance were computed using the Mann-Kendall test (Kendall, 1948; Mann, 1945). From this figure it is apparent that design values have decreased significantly over most of the eastern U.S. during this period. These decreases are in part due to EPA programs such as the Clean Air Interstate Rule and the Cross-State Air Pollution Rule with the goal of achieving broad, regional reductions in summertime NO_X emissions, as well as mobile emission reductions from federal motor vehicle emissions and fuel standards and local controls resulting from implementation of the existing O_3 standards. Other areas of the country have also experienced decreases in design values, most notably in California and near urban areas in the intermountain west.

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

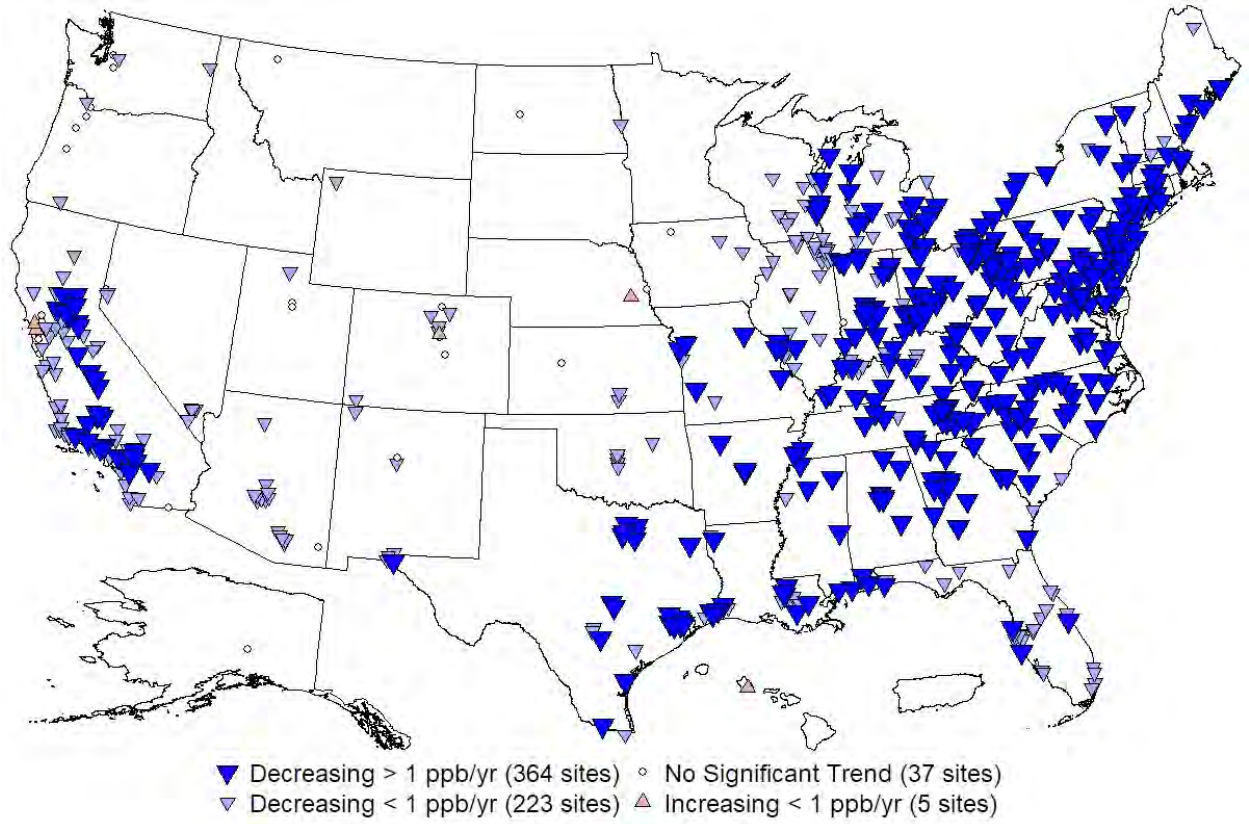
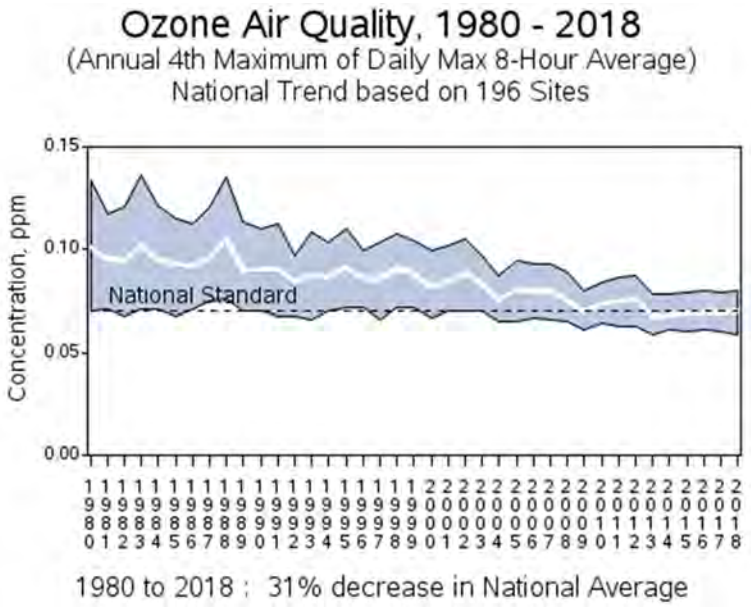


Figure 2-9. Trends in O_3 design values based on data from 2000-2002 through 2016-2018.

Figure 2-10 shows the national trend in the annual 4th highest MDA8 values based on 196 ambient air monitoring sites with complete data from 1980 to 2018. This figure shows that, on average, there has been a 31% 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 870 monitoring sites with complete data from 2000 to 2018. 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 generally 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). However, the trend in the annual 4th highest MDA8 concentrations has been relatively flat since 2013, and the design values have been relatively constant since 2015. In general, the design value metric is more stable and therefore better reflects long-term changes in O_3 than the annual 4th highest MDA8 metric.



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

Figure 2-10. National trend in annual 4th highest MDA8 values, 1980 to 2018. 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.

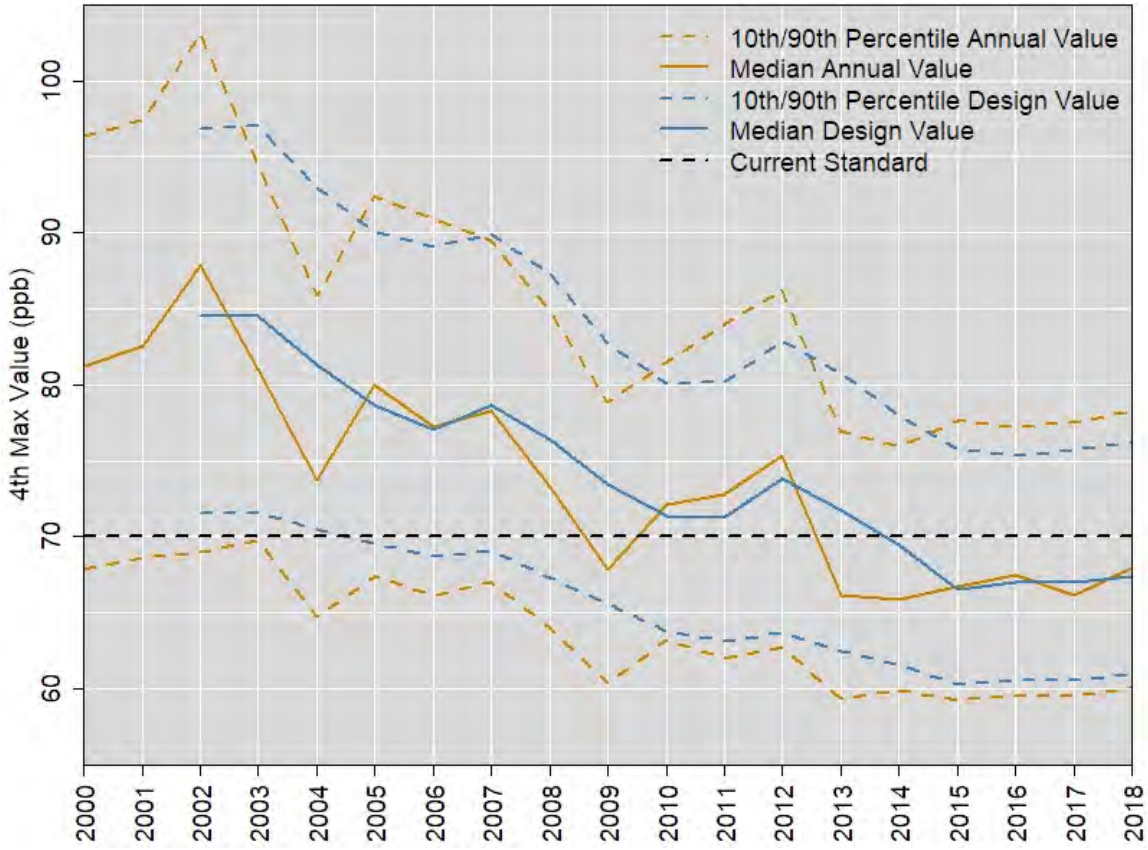


Figure 2-11. National trend in annual 4th highest MDA8 concentrations and O_3 design values in ppb, 2000 to 2018.

Figure 2-12 shows regional trends in the median annual 4th highest MDA8 values for the 9 National Oceanic and Atmospheric Administration (NOAA) climate regions[19] based on ambient air monitoring sites with complete O_3 monitoring data for 2000-2018. The five eastern U.S. regions (Central, East North Central, Northeast, Southeast, South) have all shown decreases of at least 10 ppb in median annual 4th highest MDA8 values since the early 2000's, with the Southeast region in particular showing the largest decrease of over 20 ppb. On the other hand, the median annual 4th highest MDA8 values have changed by less than 10 ppb in each of the four western U.S. regions (Northwest, Southwest, West, West North Central). The large increase in the Northwest region in 2017 and 2018 is largely due to the influence of wildfires.

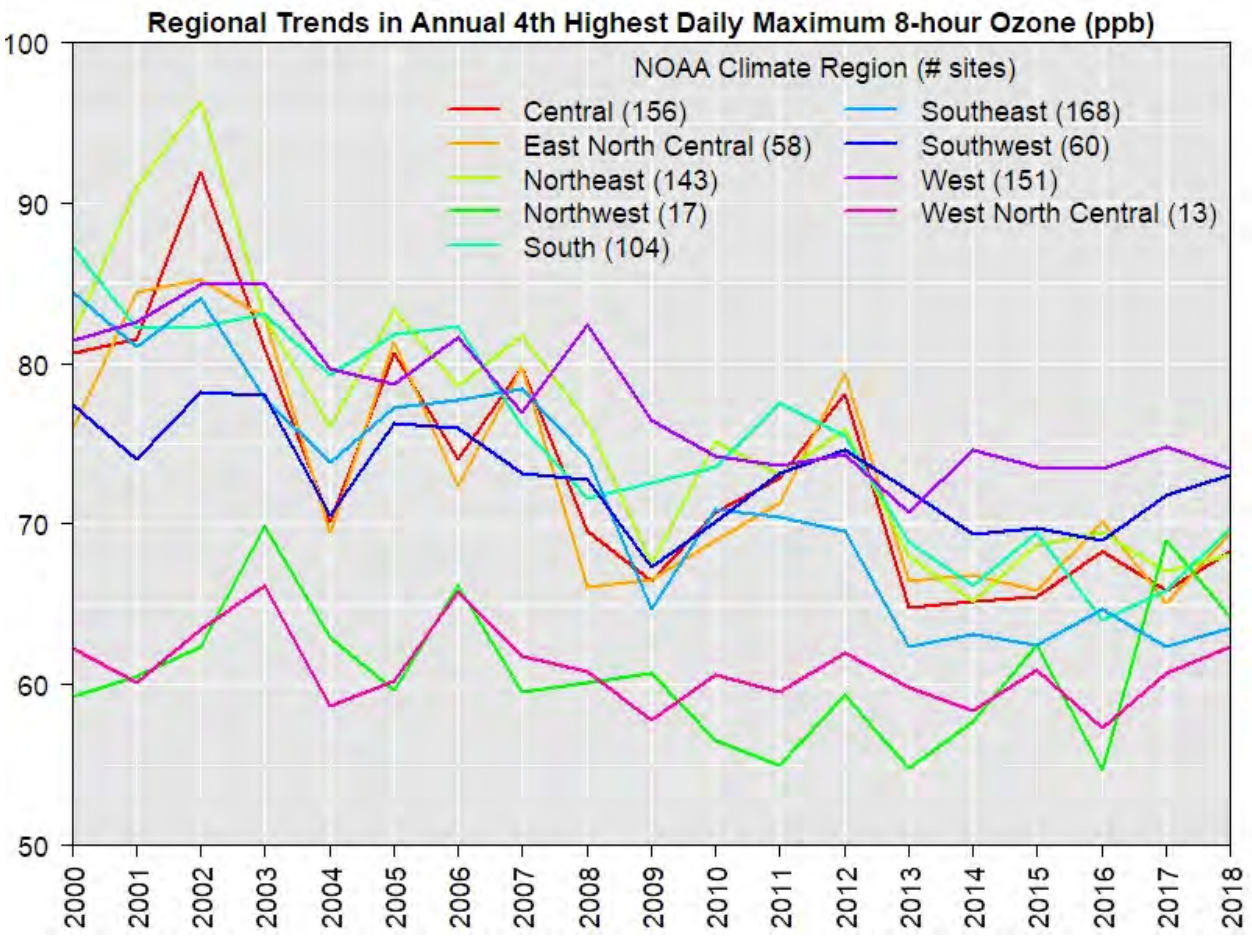


Figure 2-12. Regional trends in median annual 4th highest MDA8 concentrations, 2000 to 2018.

Trends presented in this section have focused on annual 4th high MDA8 concentrations and design values. Additional information from the published literature has examined trends in

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

MDA8 concentrations across the distribution of high and low O_3 days. Simon et al., 2015) found that, similar to results presented in this section for DVs and annual 4th high MDA8 concentrations, the 95th percentile of summertime MDA8 concentrations decreased significantly at most sites across the U.S. between 1998 and 2013. In contrast, trends over that time period for the 5th percentile, median and mean of MDA8 varied with location and time of year. Similarly, Lefohn et al. (2017) reported that between 1980 and 2014 there was a compression of the distribution of measured hourly O_3 values with extremely high and extremely low concentrations becoming less common. As a result, O_3 metrics impacted by high hourly O_3 concentrations, such as the annual 4th highest MDA8 value, decreased at most US sites across this period. Concurrently, metrics that are impacted by averaging longer time periods of hourly O_3 measurements, such as the 6-month (April-September) average of daytime (8am-7pm) O_3 concentrations, were more varied with only about half of the sites exhibiting decreases in this metric and most other sites exhibiting no trend (Lefohn et al., 2017).

2.4.3 Diurnal Patterns

Tropospheric O_3 concentrations in most locations exhibit a diurnal pattern due to the photochemical reactions that drive formation and destruction of O_3 molecules. Figure 2-13 shows boxplots of O_3 concentrations in ambient air, by hour of the day for four monitoring sites that represent diurnal patterns commonly observed in the U.S. The top panels show diurnal patterns, based on available data from 2015-2017, at urban (panel A) and downwind suburban (panel B) monitoring sites in the Los Angeles metropolitan area. Both sites generally experience their highest O_3 concentrations during the early afternoon hours, and their lowest concentrations during the early morning hours, as is typical of most urban and suburban areas in the U.S. However, higher levels of NO_X emissions near the urban site may suppress O_3 formation throughout the day and increase the O_3 titration rate at night, resulting in lower O_3 concentrations than those typically observed at the downwind site.

Rural areas generally experience lower O_3 concentrations than urban and suburban areas, with less pronounced diurnal patterns. However, elevation and transport also play a larger role in influencing concentrations in rural areas than in urban areas. The bottom panels in Figure 2-13 show diurnal patterns at low elevation (panel C) and high elevation (panel D) rural monitoring sites in New Hampshire. The low elevation site experiences O_3 concentrations that are 10-20 ppb lower, on average, than the high elevation site. The low elevation site experiences a slight diurnal pattern similar to that seen at the urban and suburban sites (generally related to photochemical O_3 formation that increases concentrations in the late morning and afternoon), while the high elevation site does not appear to experience any sort of diurnal pattern in O_3 concentrations. The lack of a diurnal pattern observed at this site is typical of high elevation rural sites throughout the

U.S., suggesting that observed O_3 concentrations are primarily driven by transport from upwind areas rather than being formed from local precursor emissions. The presence of higher peak O_3 concentrations at the high elevation site than the low elevation site at all hours of the day indicates that the high elevation site may be influenced by transport from the free troposphere to a greater extent than the low elevation site.

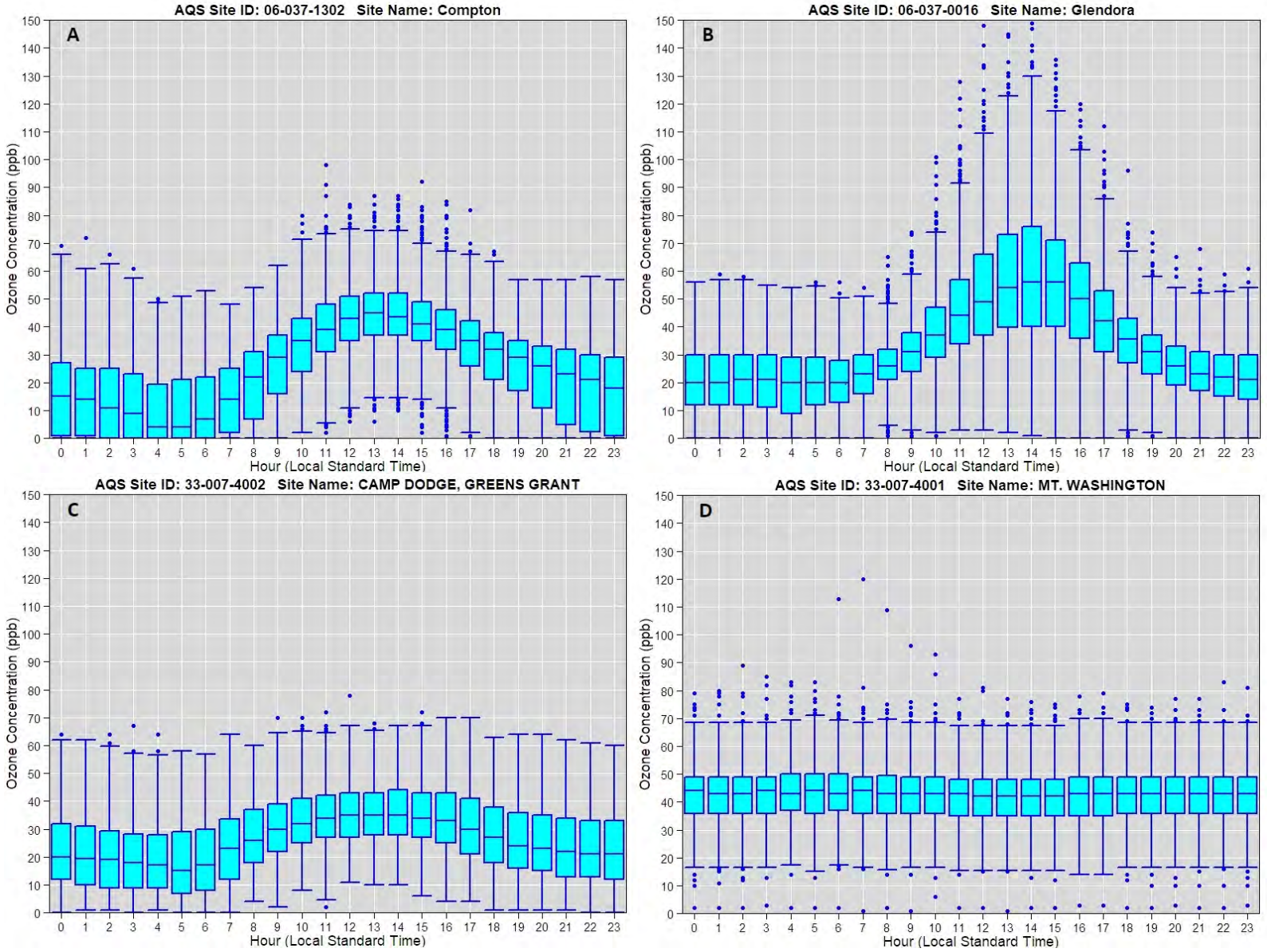


Figure 2-13. Diurnal patterns in hourly O_3 concentrations at selected monitoring sites: A) an urban site in Los Angeles; B) a downwind suburban site in Los Angeles; C) a low elevation rural site in New Hampshire; and D) a high elevation rural site in New Hampshire.

2.4.4 Seasonal Patterns

Tropospheric O_3 concentrations also tend to experience seasonal patterns due to seasonal changes in meteorological conditions and the length and intensity of daylight. High O_3 concentrations are most commonly observed on hot, sunny, and stagnant days during the spring and summer. Figure 2-14 shows boxplots of MDA8 O_3 concentrations by month of the year for four monitoring sites that represent different kinds of seasonal patterns commonly observed in the U.S. This figure is based on data from 2015-2017. Panel A shows the seasonal pattern for an urban site in Baltimore, MD, which reflects the typical seasonal pattern observed at many urban and suburban monitoring sites across the U.S. The highest O_3 concentrations are observed during May to September, when the days are the longest and solar radiation is strongest.

Panel B shows the seasonal pattern for an urban site in Baton Rouge, LA. In parts of the southeastern U.S., the highest O_3 concentrations are often observed in April and May due to the onset of warm temperatures combined with abundant emissions of biogenic VOCs at the start of the growing season. This is often followed by lower concentrations during the summer months, which is associated with high humidity levels that tend to suppress O_3 formation in the region (Camalier et al., 2007). Some areas, particularly in the states bordering the Gulf of Mexico, may experience a second peak in O_3 concentrations in September and October.

Panel C shows the seasonal pattern for a high elevation rural site in Colorado. The highest O_3 concentrations in rural areas are typically observed in the spring. This can be due to several factors, including those mentioned previously, and additionally, long-range transport from Asia is most prevalent at this time of year. Stratospheric Tropospheric Exchange (STE) events, which most often affect high elevation areas in the western U.S., are also most common during the spring.

Finally, Panel D shows the seasonal pattern for a monitoring site in Utah where high wintertime O_3 concentrations were observed. Over the past decade, high O_3 concentrations have been observed in two mountain basins in the western U.S. during the winter months (December to March). These wintertime O_3 episodes require a unique set of conditions, including a shallow inversion layer, snow cover, calm or light winds, and pervasive local NO_X and VOC emissions (in these cases, from oil and gas extraction). These conditions are relatively uncommon, and elevated wintertime O_3 levels may not occur in some years.

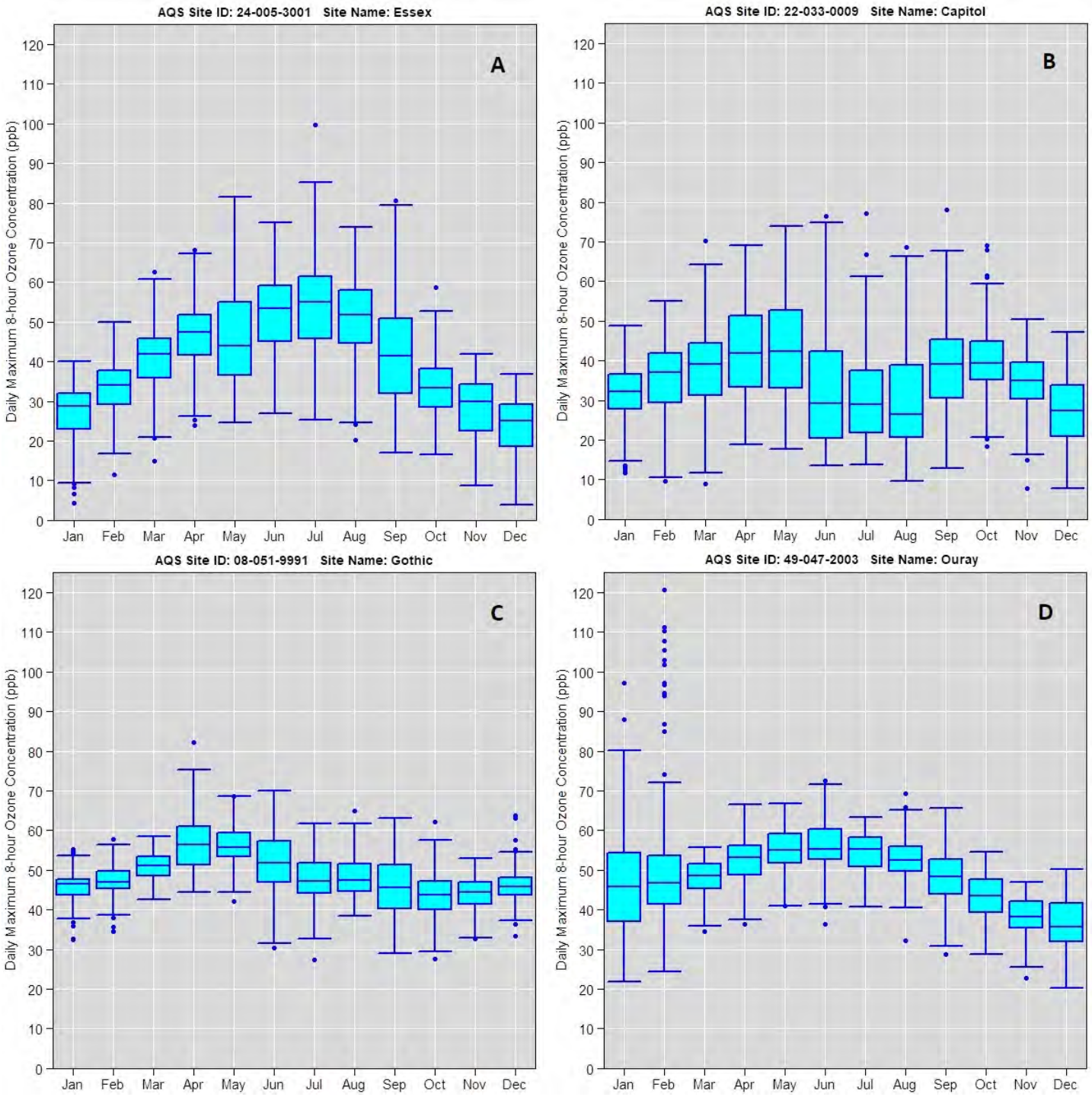


Figure 2-14. Seasonal patterns in MDA8 O_3 concentrations at selected monitoring sites (2015-2017): A) an urban site in Baltimore, MD; B) an urban site in Baton Rouge, LA; C) a rural site in Colorado; and D) a site in Utah experiencing high wintertime O_3.

2.4.5 Variation in Recent Daily Maximum 1-hour Concentrations

To provide a characterization of recent O_3 concentrations in the U.S. for periods shorter than 8 hours, this section presents recent O_3 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 O_3 standard.

Figure 2-15 shows boxplots of MDA1 values at U.S. monitoring sites based on 2016-2018 data stratified by each site's 8-hour O_3 design value. The boxes representing the 25th percentile, median, and 75th percentile MDA1 values increase slightly with higher design values. The range (min/max) of observed MDA1 values does not appear to change much, except for the presence of higher MDA1 values up to around 160 ppb for the rightmost bin which includes only sites that exceed the current standards. The boxplots show that there are only a small number 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 2016-2018 data compared to the site's 2016-2018 design value. According to the figure, a small proportion of O_3 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 standard. There are no sites that were meeting the current standards based on 2016-2018 data that had MDA1 values above 120 ppb more than twice over the same 3-year period (Appendix 2A, Table 2A-2).

Figure 2-17 shows the national trend in the annual 2nd highest MDA1 O_3 concentration, which was the metric used to track progress towards meeting the 1-hour O_3 NAAQS, originally set in 1979 and later replaced by the current 8-hour metric in 1997 (62 FR 38856, July 18, 1997).[20] The monitoring sites represented in Figure 2-17 are the 861 sites with complete data from 2000 to 2018 (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.

[20] The 1-hour O_3 standards were formally revoked in 2005 (70 FR 44470, August 3, 2005).

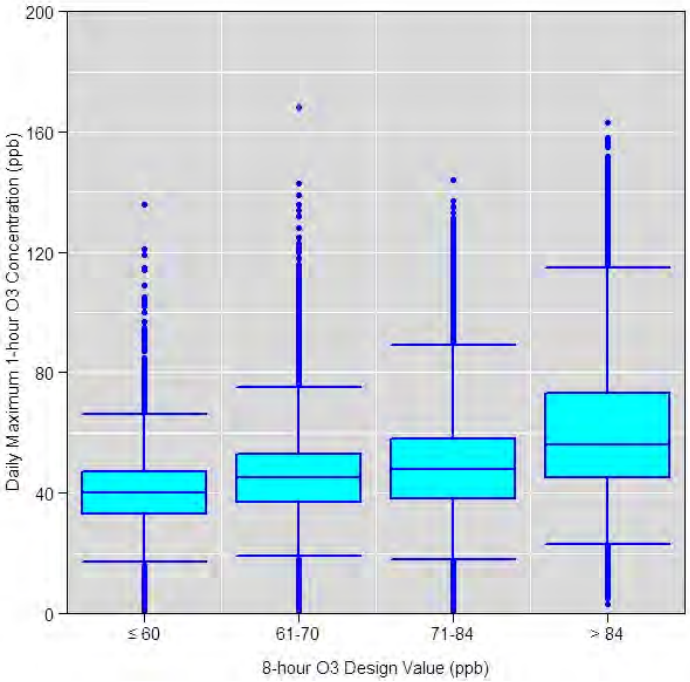


Figure 2-15. Boxplots showing the distribution of MDA1 concentrations (2016-2018), binned according to each site's 2016-2018 design value.

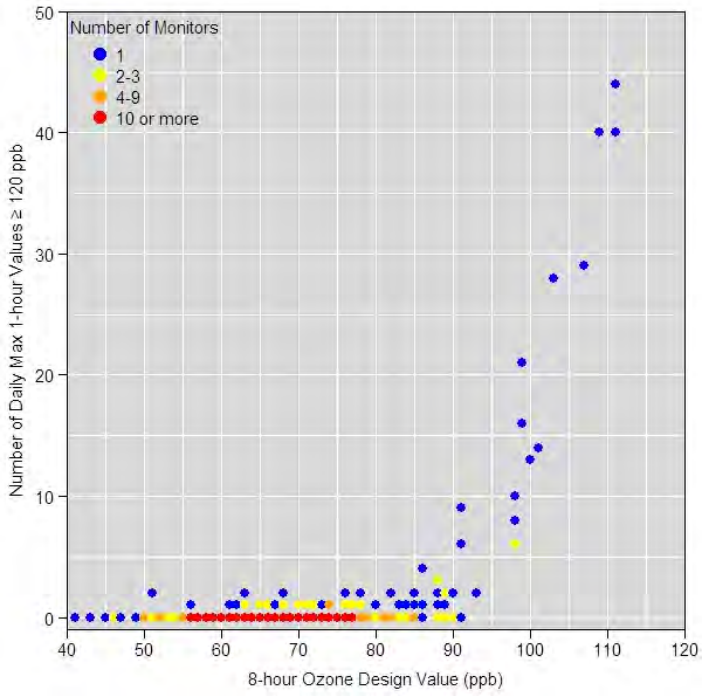


Figure 2-16. Number of days in 2016-2018 at each monitoring site with a MDA1 concentration greater than or equal to 120 ppb compared to its 8-hour design value in ppb.

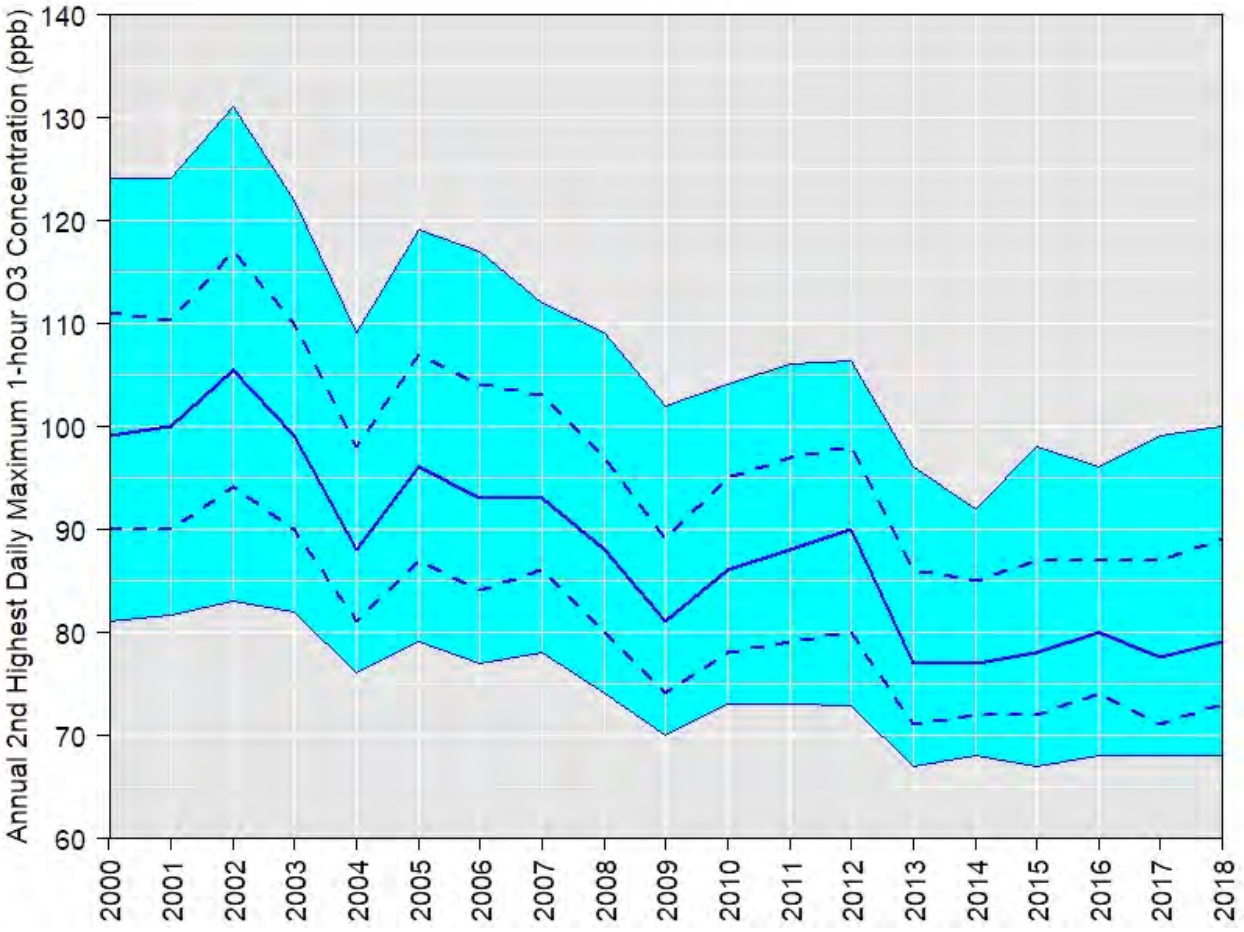


Figure 2-17. National trend in the annual 2nd highest MDA1 O_3 concentration, 2000 to 2018. The solid blue line represents the median value, dotted blue lines represent the 25th and 75th percentile values, and the light blue shaded area represents the range from the 10th to the 90th percentile values.

2.5 BACKGROUND O_3

There are a number of definitions of background O_3 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 review, as in past reviews, the EPA generally characterizes O_3 concentrations that would exist in the absence of U.S. anthropogenic emissions as U.S. background (USB). An alternative phrasing for USB is the O_3 concentrations created collectively from global natural sources and from anthropogenic sources existing outside of the U.S. Such a definition helps distinguish the O_3 that can be controlled by precursor emissions reductions within the U.S. from O_3 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 O_3 they measure,[21] photochemical grid models have been widely used to estimate the contribution of background sources to observed surface O_3 concentrations. This section summarizes results of a state-of-the-science modeling analysis that the EPA performed for this assessment to estimate the magnitude of present-day USB and its various components. Conceptually, these USB estimates represent O_3 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 O_3 concentrations that would occur in the absence of any U.S. anthropogenic O_3 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 O_3 concentration was greater than either 60 ppb or 70 ppb, and for the days on which the 4th-highest MDA8 O_3 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 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 described in section 2.5.2, including estimates specific to certain subgroups of sources. Section 2.5.4 summarizes key findings of the analyses.

2.5.1 Summary of U.S. Background O_3 Sources

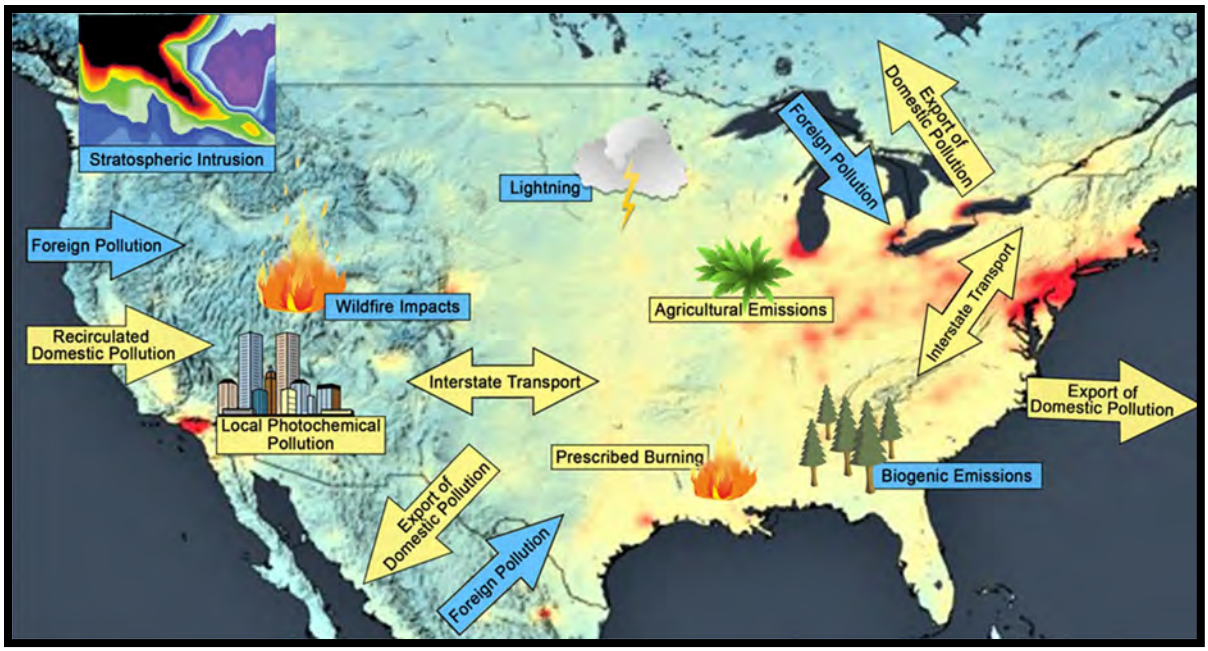
Jaffe et al. (2018) most recently reviewed the literature on sources that contribute to USB. While the term "background" may imply a low concentration well-mixed[22] environment, background sources can create well-defined plumes and/or contribute to the well-mixed environment. The USB definition, which is based on sources, includes both the well-mixed environment and more well-defined plumes. Figure 2-18a (adapted from Jaffe et al. (2018))

[21] Ozone concentrations that do not include contributions from U.S. anthropogenic emissions cannot be determined exclusively from O_3 measurements because even relatively remote monitoring sites in U.S. receive transport of U.S. anthropogenic O_3 from other locations.

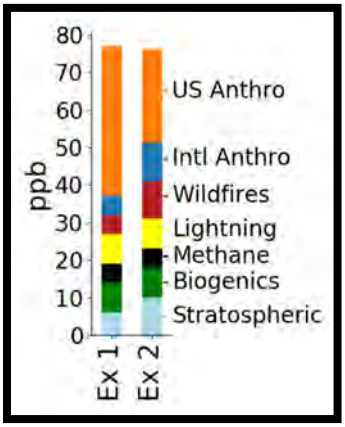
[22] We use the term "well-mixed" here to refer to conditions when the contributions from various types of sources are mixed due to chemistry or physical processes to the point where it is not possible to discern the contribution to O_3 from each individual source.

illustrates sources of USB O_3 (blue) and US anthropogenic sources of O_3 (yellow). Figure 2-18b shows two theoretical examples where background sources contribute to the total ground-level O_3. The first example (Ex 1) highlights a typical monitoring site with lower USB, and the second example (Ex 2) presents a scenario in which USB is a large contributor. Both examples oversimplify methane, which has both natural and anthropogenic and both domestic and foreign contributions. Source contributions to USB vary in space and time, and the stacked bar plot in this figure oversimplifies the complex relationship between USB and total O_3. Even so, USB sources can broadly be discussed as global natural sources (see sections 2.5.1.1 to 2.5.1.6) and international anthropogenic sources (see section 2.5.1.7). In the simplest interpretation, the natural sources are background regardless of where they occur, or which definition of background is being used (e.g., USB or natural background[23]). By contrast, anthropogenic sources are only considered as background when they are not from sources within the focus area. However, this paradigm is complicated by the fact that many sources of O_3 precursors are the result of interactions between human and natural systems (for instance forest management practices can impact both biogenic VOC emissions from trees and wildfires). In the context of USB, anthropogenic background is synonymous with O_3 originating from international anthropogenic emission sources. The relative contribution of international and natural background sources can vary dramatically from place to place and are most notably larger at locations near borders (international) or high elevation (natural). At non-border locations and many border locations, the natural background is usually the dominant background source.

[23] Natural background is the O_3 that would exist in the absence of anthropogenic emission sources.



(a)



(b)

(a) U.S. O_3 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 NO_2 columns for 2014 from the Ozone Monitoring Instrument (OMI) onboard the NASA Aura satellite (Credit: NASA Goddard's Scientific Visualization Studio/T. Schindler). NO_2 column amounts are relative with red colors showing highest values, followed by yellow then blue. We use the OMI NO_2 columns as a proxy to show local O_3 precursor emission sources. (b) The bar chart shows two theoretical examples of USB O_3 contributions combine with domestic sources to produce elevated O_3 at a specific location on any given day. Each source varies daily and there are also nonlinear interactions between USB O_3 sources and anthropogenic sources that can further add to O_3 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*

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

The natural and anthropogenic sources of background O_3 vary by location and by season. Emissions from anthropogenic sources largely occur in the same areas year after year. Natural sources of O_3 and precursors, on the other hand, vary both in magnitude and in location from day to day and year to year. As a result, certain types of natural sources may have large O_3 contributions measured at a monitor at one point in time but not at other times. The combination of varying proximity and magnitude means that natural sources can contribute to background in the form of localized plumes of elevated O_3 that contribute to O_3 at monitoring sites on an episodic basis. In the absence of locally well-defined plumes, global natural and international anthropogenic sources are constantly contributing to the well-mixed background.

USB varies by location and by season due to both the nature of sources and the loss processes. The nature of emission sources leads to seasonal and spatial patterns that will be described further below. The contribution of these sources is modulated by transport patterns that interact with deposition and chemical losses. For illustration, two emission sources of identical magnitudes may have different contributions if one emits near the surface in summer and the other emits in the free troposphere in spring. Warmer moister air in the summer at the surface enhances O_3 chemistry losses and deposition of O_3 to the surface increases losses further. In contrast, cooler, drier temperatures in the spring and free troposphere lengthen O_3 lifetimes and faster winds in the free troposphere enable longer transport. The seasonality of temperature and transport patterns gives O_3 USB a distinct seasonal cycle that results from both sinks and sources.

The sections below summarize the state of the science estimates of USB contributions. Each source type is described with respect to its seasonality as well as its local vs well-mixed contribution potential. Jaffe et al. (2018) reviewed contributions of various sources to USB O_3 from modeling studies and the references therein are used to illustrate the range of O_3 contributions from each source. The literature-based estimate ranges provide context to the estimates of USB that are reported in section 2.5.3.

2.5.1.1 Stratosphere

The only direct source of O_3 to the troposphere with appreciable contributions to O_3 concentrations is STE (other sources are indirect via precursors). STE occurs when stratospheric air, which is relatively rich in O_3, is transported across the tropopause where it enhances tropospheric concentrations. Most STE events create enhancements that do not immediately reach the surface. Instead, STE-enhanced O_3 mixes into the free troposphere where it is dispersed. In cases when the transported air reaches the surface before enough dispersion occurs, it creates a localized plume of O_3 referred to as a Stratospheric Ozone Intrusion (SOI). The total stratospheric contribution includes both the well-mixed contribution from the distant stratosphere exchanges as well as any localized SOI plume.

The total global O_3 flux from the stratosphere to the troposphere is estimated at 510±90 teragrams per year (Tg/y) compared to 4620±600 Tg/y (post-2000 literature in Table 2 in Wu et al., 2007) produced within the troposphere. The majority of the earth's surface is outside the U.S. and only STE that take place over the U.S. are likely to create a large magnitude local enhancement at a U.S. monitor.[24] A SOI that occurs outside the U.S. would likely be dispersed

[24] Recently methods have been developed for identifying and estimating SOIs that have clear localized contributions to O_3 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*.

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

Modeling and observational studies show that SOI can episodically contribute large amounts of O_3 at a subset of U.S. monitors, but stratospheric mixing more frequently contributes smaller quantities of O_3. Modeling studies focused on seasons with frequent SOI find median total stratospheric contributions to MDA8 are 10-22 ppb in the West and 3-13 ppb in the East with episodic contributions up to 40 ppb mostly in the West (Table S2, Jaffe et al., 2018). Because these studies focus on the most active season, these medians are expected to be upper bounds for the annual average. Further, SOI are most common in the spring when MDA8 O_3 concentrations above 70 ppb are less common (ISA, section 1.3.2).

2.5.1.2 Biogenic VOC

Biogenic VOCs are the quintessential "natural" source of O_3 precursors. At global scales, biogenic sources are the largest contributor to VOCs – even though local anthropogenic sources of highly reactive VOCs can be very important in some areas. VOCs are also an important source of carbon monoxide. Biogenic VOCs are emitted by various types of vegetation and emissions peak in summer which is also when O_3 production is fast and O_3 lifetimes are short.

The large abundance of biogenic VOCs leads to NO_X-limited O_3 production in most of the world. That is, concentrations of biogenic VOCs are in excess with respect to concentrations of NO_X; therefore O_3 production is controlled by the availability of NO_X. The methodologies[25] typically used by the air quality community estimate contribution based on sensitivity of O_3 production. As a result, the sensitivity-based contribution estimate of biogenic VOC sources to O_3 shows relatively small contributions considering the large amount of emissions.

Estimates of biogenic VOC contributions in the literature are generally small compared to NO_X. For example, Lapina et al. (2014) found that North American Background (NAB)[26] for W126[27] O_3 was relatively insensitive to VOC (10.8% of NAB sensitivity) compared to NO_X (79.8% of NAB sensitivity). This well-known global-scale sensitivity to NO_X would not exist if concentrations of biogenic VOCs were a broadly limiting factor. Even though background O_3 is not particularly sensitive to small changes in the biogenic VOC, natural sources of VOCs are a critical component of all background O_3 estimates.

[25] 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.

[26] North American Background is analogous to USB; but NAB is generally characterized as the O_3 concentrations that would exist in the absence of North American anthropogenic emissions.

[27] 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 O_3. 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 O_3 enhancements (McClure and Jaffe, 2018).[28]

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 O_3 contributions to seasonal mean O_3 of up to several ppb during high fire years in the Western U.S. (Jaffe et al., 2018), O_3 production from individual fires varies substantially (Akagi et al., 2013). Several studies have shown that locations near large fires can even experience suppressed O_3 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 O_3 precursor emissions from fires combined with complex in-plume dynamics and chemistry make accurately quantifying O_3 production from fires extremely difficult at both regional and local scales.[29]

New data from recent and upcoming field and aircraft campaigns[30] are expected to provide new insights that expand current understanding of contributions from fires to O_3 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 O_3 precursor source. Lightning produces NO_X from molecular nitrogen and oxygen, similar to traditional combustion processes. Because NO_X is the

[28] 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.

[29] Recently methods have been developed for identifying and estimating wild or prescribed fire contributions to O_3 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*.

[30] 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.

globally limiting precursor for O_3 production and lightning emits where there are few other sources, O_3 production is quite sensitive to this source. Over the U.S., lightning NO_X (LNO_X) emissions peak in summer with convective activity and are characterized as having high interannual variability (Murray, 2016). Allen et al. (2012) showed that the majority of LNO_X is emitted in the free troposphere (i.e, troposphere above the planetary boundary layer). Thus, LNO_X is produced in a NO_X-limited environment where any O_3 formed as a result will be efficiently transported and loss pathways are limited.

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

Jaffe et al. (2018) reviewed contributions from lightning to surface USB O_3 based on modeling studies using various flash rate yields, which shows large single day contributions to modeled MDA8 O_3 (up to 46 ppb, Murray, 2016) and smaller contributions to annual means (1-6 ppb) and seasonal means (6-10 ppb). Lapina et al. (2014) showed that, in their modeling, W126 had a 15% contribution from lightning NO_X over the U.S.[31] A 15% contribution is consistent with the annual and seasonal mean contributions to MDA8 reported by Zhang et al. (2014) and Murray (2016). Lapina et al. (2014) also noted that 40% of the lightning NO_X sensitivity comes from lightning strikes outside the U.S. The findings from these studies highlight the primary importance of lightning NO_X as a contributor to the well-mixed background concentrations (Murray, 2016).

2.5.1.5 Natural and Agricultural Soil NO_X

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

[31] 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%).

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

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

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

2.5.1.6 Post-Industrial Methane

Like other VOCs, CH_4 is a hydrocarbon that can form O_3 in the presence of NO_X and sunlight. While some atmospheric methane is emitted naturally from wetlands, wildfires, geogenic sources, and insects, significant global methane enhancements following the industrial revolution are clearly associated with increased emissions from anthropogenic fossil fuel combustion (Pachauri et al., 2015). Other human activities such as livestock cultivation, landfills and land use modification (e.g., rice paddies) also release methane. More recently, changing climate conditions have led to increased emissions from natural sources (e.g., permafrost melting) in some areas (Reay et al., 2018), although the exact magnitude of these effects on global methane concentrations, and consequently O_3 in the U.S., over longer time scales remains uncertain.

Due to its long atmospheric lifetime (~10 years), methane is well-mixed at seasonal and annual time scales. As a result, isolating contributions to atmospheric methane concentrations from individual geographic areas or specific emission sectors is very difficult (Turner et al., 2017). However, sensitivity simulations with chemical transport models can be used to assess the overall influence of global methane concentrations on regional O_3 budgets. For example, Lin et al. (2017) used the GFDL-AM3 chemistry-climate model to estimate that increasing global methane concentrations contributed ~20% to background MDA8 O_3 trends during boreal spring and summer at several western U.S. sites during the period 1988 to 2012. In general, post-industrial anthropogenic methane is estimated to contribute ~5 ppb to surface O_3 in the U.S., an estimate that primarily comes from modeling studies (Jaffe et al., 2018 and references therein).

A major limitation with existing model-based estimates of the influence of global methane on current U.S. O_3 concentrations is our limited understanding of historical methane emissions. The U.S. and the rest of the world's anthropogenic methane emissions have not been tracked quantitatively in detail until relatively recently. As a result, the pre-industrial methane concentration is relatively unconstrained. Further, post-industrial methane can be attributed to direct emissions and emissions from natural sources (e.g., permafrost). Many modeling studies, including this one, do not explicitly track methane sources and sinks, further complicating attribution in an air quality context. Therefore, the post-industrial methane contribution is difficult to quantitatively attribute. The post-industrial enhancement of methane is clearly related to emissions and human activity, which includes both foreign and domestic contribution.

2.5.1.7 International Anthropogenic Emissions

International anthropogenic emissions are the only anthropogenic contribution to USB. For the purposes of discussion, NO_X and VOCs will be discussed separately from methane (methane is covered in section 2.5.1.6). NO_X and VOC emission estimates from outside the U.S. are derived from international collaborative efforts like the Hemispheric Transport of Air Pollutants (HTAP) task force of the United Nations Economic Commission for Europe. HTAP harmonized national emission databases from individual countries with global estimates that cover areas without their own estimates. Collecting and harmonizing these emission datasets requires coordination and technical expertise, which recently occurred twice (HTAP Phase I and HTAP Phase II) and may occur again soon. Global estimates that incorporate national information are available (e.g., Community Emissions Data System and Emissions Database for Global Atmospheric Research), but do not always have as much participation from individual countries. This is particularly important because individual countries are most aware of regulations and controls that have been promulgated within their borders.

International anthropogenic sources of O_3 include emissions within the borders of other countries (e.g., onroad sources, power plants, etc.) as well as sources in international waters and air space. Sources within the borders of other countries can be easily attributed to those countries using geographical bounds based on emission source location. Some studies (e.g., Lin et al., 2014), however, have done more complex analyses to spatially attribute emissions globally based on the consumption of produced goods. For the purposes of this document, international emissions are attributed based on the emission source location. Using emission source location, maritime shipping and aircraft sources require more artificial distinctions. Typically, aircraft takeoff and landing are assigned completely to the country where it occurs. Aircraft cruising emissions are attributed based on geographic boundaries. This assumes that both inbound and outbound flights change source type (domestic/international) when they cross a border.

2.5.2 Approach for Quantifying U.S. Background Ozone

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

This assessment uses a firmly source-oriented definition of USB based on modeling. The source composition of a model estimate can be quantified using tagging techniques or by sensitivity analysis. By contrast, the source composition of measured O_3 is difficult to isolate. In most areas at most times, measured O_3 concentrations are the result of contributions from a variety of anthropogenic and non-anthropogenic sources. Measurements from locations sometimes suggested to be representative of USB often have contributions from U.S. anthropogenic sources. As a result, some researchers have filtered measurements to focus on times when US contributions are minimized (e.g., based on wind direction or other indicators). The measurement filtering approach is based on conceptual or quantitative models of source contributions as a function of wind direction or another environmental variable. After correction, the degree of contamination is minimized but not precisely known. Recently, urban measurements have been paired with simplistic statistical models to estimate background (Parrish et al., 2017). However, Jaffe et al. (2018) concluded that statistical adjustment cannot be directly interpreted as “background” – even though the estimate is useful for bounding simulated background. Due to the complications of quantifying background based on ambient air

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

This section of the PA quantifies O_3 from sources using a sensitivity approach. The multiscale system is applied to predict total O_3 and then applied multiple times to predict O_3 without U.S. anthropogenic emission sources. The difference between total O_3 and O_3 without the U.S. anthropogenic emissions is used to characterize the USB.

2.5.2.1 Methodology: USB Attribution

This assessment attributes O_3 to USB sources using one of several available techniques. Jaffe et al. (2018) reviewed the methods for identifying USB contributions. The methodologies reviewed range in complexity from simply turning off U.S. anthropogenic (or specific sources) emissions, to normalizing derivatives from instrumented models, to complex tagging techniques (e.g., CAMx OSAT, APCA, or Grewe, 2013).[32] This analysis follows the zero-out approach for simplicity of interpretation and consistency with previous EPA analyses. In urban areas, this approach will estimate higher natural and USB contributions when NO_X titration is present. The estimate, therefore, is an estimate of what concentrations could be without US anthropogenic emissions and not the fraction of observed O_3 that is USB.

This analysis is designed to specifically separately quantify $O_3$from global natural, international anthropogenic, and U.S. anthropogenic sources. The precursors that this analysis focuses on are NO_X and VOC because they have a response on timescales relevant to the NAAQS planning schedules (i.e., not methane). Table 2-1 lists simulations and the sources they exclude at the various spatial scales modeled (i.e., hemispheric – 108 km resolution, regional – 36 km resolution and regional – 12 km resolution). For international shipping and aviation, the U.S. domain is either included (ZROW) or excluded (ZUSA). These simulations form the basis for estimating the contributions of USB and its components. Given the long atmospheric lifetime and attributability to U.S. sources, methane is not separately identified nor is it perturbed in any simulations. This has the effect of attributing methane to natural processes, which are a background source.

[32] For a discussion of methods and the effect on estimates, see (Jaffe et al., 2018).

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

Simulation	Description
Performed at Hemispheric [A] and Regional [B] Scales	
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.
Performed at Hemispheric Scale only	
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.

Table 2-2 describes the calculations that are used to derive contributions. It is important to note that contributions are not strictly additive. Large NO_X sources can create non-linear conditions that decrease O_3 concentrations due to titration which is most relevant at night and in the winter. In some cases, removing a source only increases the efficiency of other sources. In that case, some anthropogenic contribution exists unless all anthropogenic sources are removed. This residual anthropogenic contribution occurs in the model for both International and U.S. sources. The results presented in this section focus on Base, USB, International, Natural contributions. Some components of International and Natural were separately analyzed. Canada/Mexico are separately quantified at both hemispheric and regional scales. The India, China, Fire, and shipping contributions are analyzed only at the hemispheric scale and are presented in Appendix 2B. The analyses in Appendix 2B support the interpretation in the discussion below.

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

2.5.2.2 Methodology: Strengths, Limitations and Uncertainties

The model was evaluated to assess the accuracy of predictions and infer possible biases in USB estimates. Evaluations included comparison to satellite retrievals, O_3 sondes[33], CASTNET monitors, and AQS monitors. Results were also qualitatively compared to the Tropospheric Ozone Assessment Report (TOAR) database, which has global O_3 observations that have been well characterized[34] but only extends through 2014. The evaluation of the hemispheric simulation that provides boundary conditions to the 36 km model simulation relies heavily upon the satellites, O_3 sondes and CASTNET monitors. Since the satellite data can be used to provide concentration estimates in areas without surface monitors, these data are particularly useful for evaluating O_3 column totals in the hemispheric modeling. The sonde data provide a means to evaluate predictions aloft which are important for understanding model performance of long-range transport. The regional evaluation analysis focuses on data measured at CASTNET and AQS monitors.[35] Evaluation using the AQS monitors provides information on how the model performs at urban/suburban O_3, which may exhibit large space/time gradients in O_3 concentration. CASTNET data are included in the evaluation of both the hemispheric and

[33] O_3 sondes are balloon-borne instruments that ascend through the atmosphere taking O_3 and meteorological measurements. For more information, see *https://www.esrl.noaa.gov/gmd/ozwv/ozsondes/*.

[34] The TOAR database includes O_3 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.

[35] 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).

regional models since monitoring sites in this network are intended to represent O_3 concentrations across broad areas of the U.S.

The evaluation using sonde data shows that the hemispheric model predictions of O_3 are generally within 20% of the corresponding measurements throughout much of the free troposphere. Near the tropopause, there is a low bias in the model that is most pronounced in the spring. The low bias at the tropopause likely suggests an underestimate of stratospheric exchange. Mean bias drops to below 20% in the middle troposphere (600-300 hPa). The low-bias in the free troposphere may stem from underestimation of spring time stratospheric contribution in some regions.

The acceptability of model performance was judged for the 2016 CMAQ O_3 performance results considering the range of performance found in recent regional O_3 model applications (NRC, 2002, Phillips et al., 2008, Simon et al., 2012, U.S. EPA, 2009, U.S. EPA, 2018d). The model performance results, as described in this document, demonstrate the predictions from the 2016 modeling platform closely replicate the corresponding observed concentrations in terms of the magnitude, temporal fluctuations, and spatial differences for 8-hour daily maximum O_3. At CASTNET sites, the model performance is similarly good, but has a distinct seasonal pattern (see Appendix 2B.3). The normalized mean bias increases from a low-bias in boreal Winter (West: -16%; East: -14%) to relatively neutral in boreal Fall (West: 0%; East: 7%). These results are consistent with the free troposphere bias seen in the comparison of model predictions to sonde data. Despite the conceptual consistency, the low-bias in winter at CASTNET sites is also influenced by local sources. For example, the Uintah Basin monitors have extremely high winter observations that are underpredicted by the model. These are most likely due to underestimation of O_3 formed from precursors emitted by local sources as well as the need for finer resolution meteorological inputs to capture cold pool conditions that characterize these events.[36]

Model predictions have historically shown poor performance for capturing the impacts from O_3 of wildfires and stratospheric intrusions. Wildfire contributions have been overpredicted by models (Baker et al., 2016, Baker et al., 2018). Model predictions of O_3 from stratospheric intrusions have ranged from underestimated to overestimated (e.g., Emery et al., 2012). Models are not expected to perform well in capturing the contributions from wildfires and stratospheric intrusions without a focused effort on properly characterizing the physical properties of individual events.

[36] The DIN431 CASTNET monitor, among others, is in the Uintah basin where wintertime O_3 can be caused by snow-cover enhanced photolysis combined with light VOC emissions from the oil and gas production. (see Ahmadov et al., 2015).

This analysis uses an emission inventory with known issues in the fire inventory. The "2016fe" inventory had double counting of some grassland fires.[37] To minimize the effects of double counting, a filter is applied to the data to remove large episodic natural influences including fires. The filter removes days where natural contributions deviate from the mean for that grid cell by whichever is higher: 20 ppb or twice the standard deviation for that grid cell. Using this approach, 0.1% of grid cell days were removed -- 71% of grid cells have no days removed and fewer than 5% have more than 1% removed. Of the days that were removed, fewer than 21% had MDA8 concentrations above 70 ppb.

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

2.5.3 Estimates of USB and Contributions to USB in 2016

Background O_3 is known to vary seasonally, spatially, and with elevation (as discussed in section 2.5.1, above). Seasonal variations are related to temporal changes in both sources and sinks. Spatial variations are related to differential transport patterns and the proximity to sources of background O_3. Elevation is important in determining USB because it relates to the proximity to the free troposphere. In addition, the seasonality and spatial relationships of USB and USA contributions are not always aligned. As a result, USB can be highest on days with lower total O_3. For these reasons, estimates of USB and USB components (i.e., Natural and International) contributions developed from the current modeling are summarized spatially, over time, and as a function of total O_3.

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

[37] 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*.

cells in the U.S. are included in this analysis.[38] Grid cells with water as the dominant land use (e.g., lake or ocean) were simply excluded from analysis to acknowledge the potential bias of total O_3 over water bodies (U.S. EPA, 2018). The USB estimates provided here are all in terms of a metric, MDA8, closely related to the form of the current O_3 standards, and do not directly apply to other metrics.

Section 2.5.3.1 characterizes the spatial variation of model-predicted MDA8 O_3 concentrations and contributions using maps of seasonal averages. Section 2.5.3.2 characterizes the time variation of the predicted MDA8 O_3 and contributions using time series of spatial averages. Section 2.5.3.3 characterizes the relationship between predicted USB components and predicted total O_3. Section 2.5.3.4 summarizes USB predictions across regions and seasons.

2.5.3.1 Spatial Characterization of O_3 Contributions

Figure 2-19 and Figure 2-20 provide seasonally aggregated maps that show the spatial distribution of total model-predicted MDA8 O_3 and contributions from natural, international, and U.S. anthropogenic sources across the U.S.

Figure 2-19 shows predicted MDA8 values for the 12 km domain averaged for spring months (March, April, and May) for total O_3 and contributions from Natural, International, and USA. Natural is a relatively large contributor to total O_3 in spring with a relatively small range of values (ratio max:min = 2). International contributes less with a larger range (ratio max:min = 3). There are spatial gradients primarily along parts of the Mexico border, and an overarching general West-East gradient. The USA contribution, even in spring, has the largest variation (ratio max:min > 20) with enhancements in some urban areas.

[38] 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.

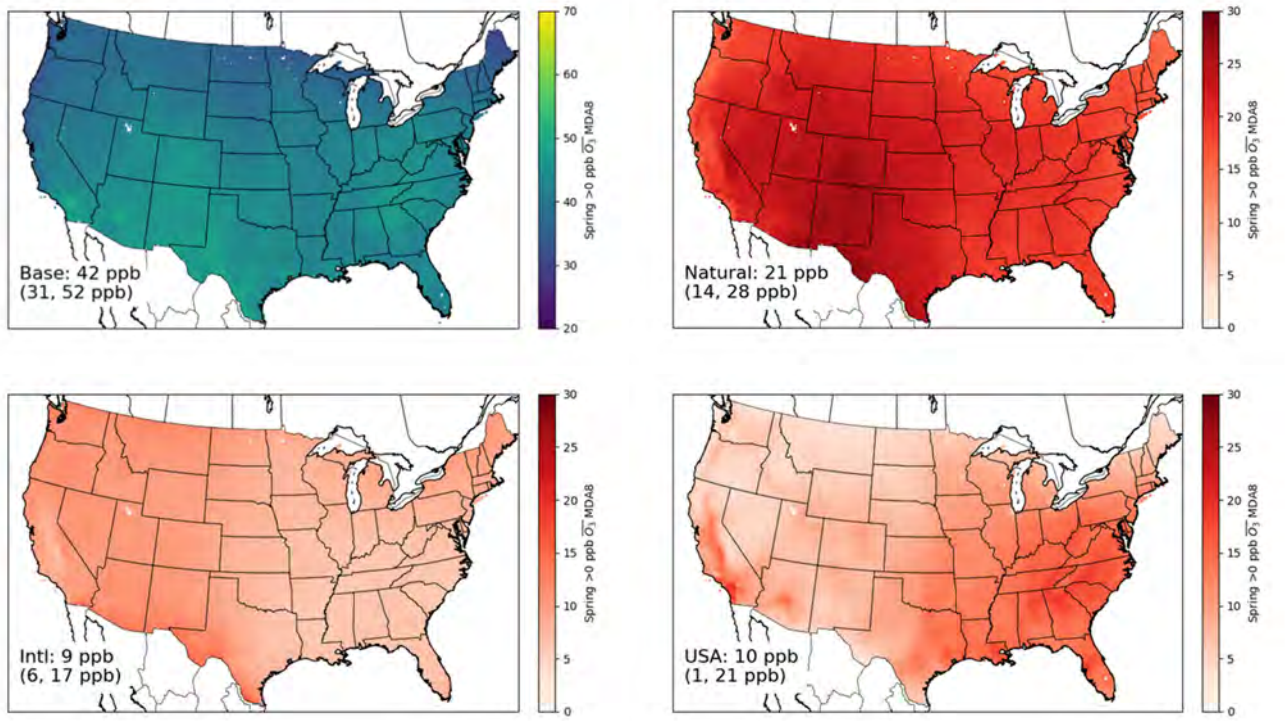


Figure 2-19. Predicted MDA8 total O_3 concentration (top left), Natural (top right), International (bottom left), and USA (bottom right) contributions in spring (March, April, May). Each panel displays the simple spatial average and range (min, max) in ppb in the lower left-hand corner of the panel.

Figure 2-20 shows the same type of information for the summer (June, July, August). The summer total concentrations are higher than spring due to increases in USA and Natural contributions. The international contribution spatial gradients have increased (reflecting shorter O_3 lifetimes), so that the maximum International contribution at the border is higher and the average contribution is lower compared to spring. Similarly, the West-East gradient of Natural, International, and USA contributions is enhanced in the summer. In addition, the USA contributions show distinct gradients in urban areas. Figure 2-20 highlights the increasingly near-border or high-elevation influence of international contribution during the summer when O_3 concentrations are most likely to violate the NAAQS.

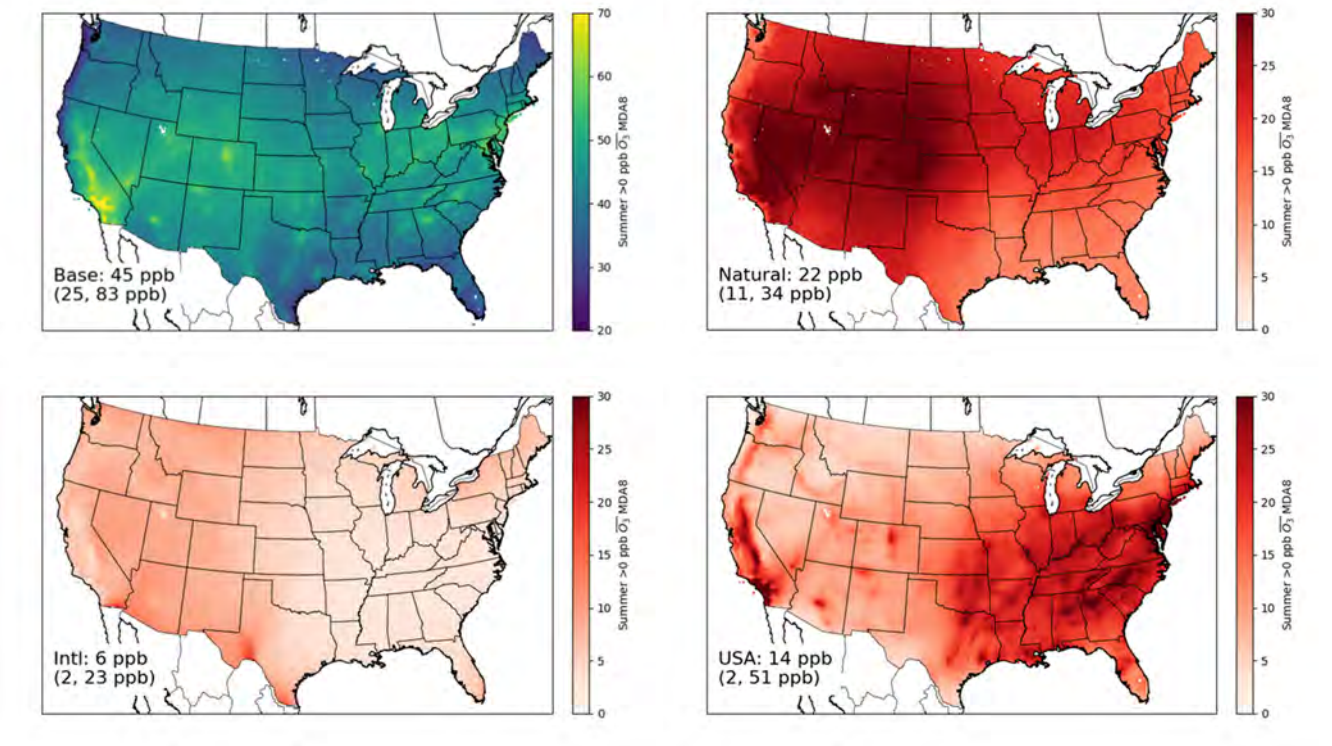


Figure 2-20. Predicted MDA8 total O_3 concentration (top left), Natural (top right), International (bottom left), and USA (bottom right) contributions in summer (June, July, Aug). Each contribution has the spatial average and range (min, max) in ppb in the lower left-hand corner of the panel.

2.5.3.2 Seasonal and Geographic Variations in Ozone Contributions

Seasonal and geographic variations are an important part of background O_3. The geographic variation helps us to understand where USB contributes appreciably to O_3 concentrations. The seasonal variation is particularly important as it determines whether high USB and MDA8 concentrations above 70 ppb are likely to occur at the same time. This section begins by characterizing the dependencies of predictions for different USB components on season and geography to define regions for further analysis. These dependencies are used to define regions for subsequent time series analysis.

Seasonal dependence: Comparing Figure 2-19 and Figure 2-20 highlights the seasonal differences in the predicted contributions from Natural, International, and USA sources. Between spring and summer, the International contribution decreases by 33%; the USA contribution increases by 40%; and the contribution from Natural sources shows a relatively small increase of 5%. The differences in contributions between the spring and summer are due to a complex relationship between O_3 production, O_3 lifetime, and therefore transport efficiency. Cooler drier conditions increase the lifetime of O_3 in winter/spring compared to summer/fall (Liu et al., 1987). As a result, winter and spring have more efficient transport of O_3 compared to summer

and fall. Summer and fall, however, have warmer weather that promotes higher local O_3 production rates. Thus, summer and fall have locally fast O_3 production and relatively inefficient transport, which combined increase the relative contribution of proximate sources.

Border dependence: In the summer, model-predicted gradients of International O_3 at the borders are most obvious. As previously discussed, summer temperatures increase O_3 production rates and decrease O_3 lifetimes. As a result, areas with locally high O_3 are evident near the border in southern California and the Big Bend and lower Rio Grande areas of Texas. These local enhancements generally occur within tens of kilometers from the border due to the short O_3 lifetime in summer as noted above.

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

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

Figure 2-21 shows that proximity to the border with Canada or Mexico is a good indicator of the role of international contributions on USB predictions. In the spring, the average international contribution can be as much as 12.4 ppb within 100 km of the border (62 miles). In the early spring, large contributions persist further from the border because of the longer O_3 lifetimes. Near the borders the contributions also have much higher variability, both from day-to-day and between locations on the border. The contribution from international sources drops notably in the summer months when O_3 concentrations are highest. The day-to-day variability is associated with the variations in wind direction, while the location variability is associated with

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

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

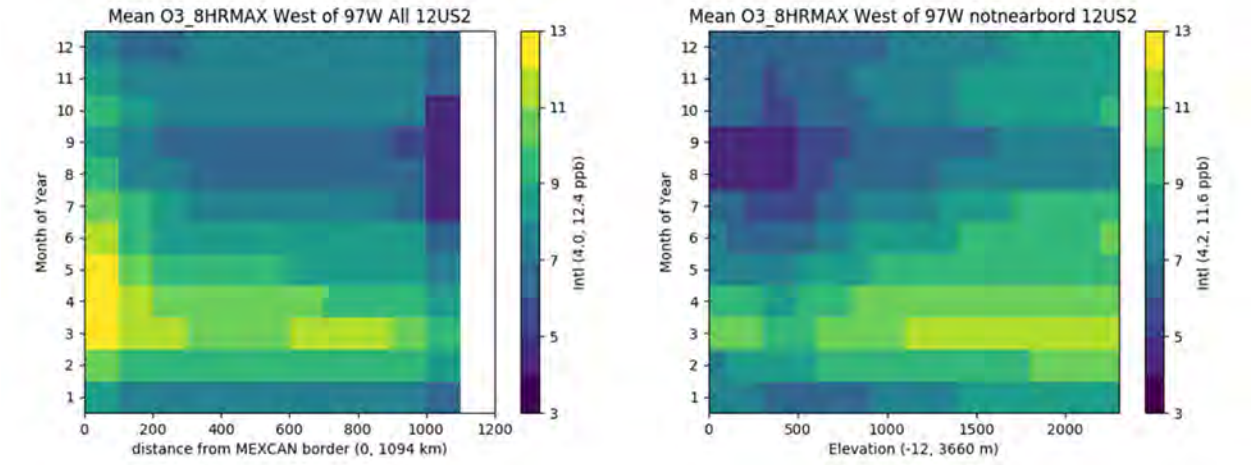


Figure 2-21. Predicted contribution of International sources as a function of distance from Mexico/Canada (left) and at "interior" locations (excluding border areas) by elevation (right).

Timeseries Analysis: The maps in Figure 2-19 and Figure 2-20 and the Hovmoller plots in Figure 2-21 highlight the impact of season and location on predicted O_3 and contributions. To further characterize the temporal variations in contributions, the contribution data are averaged over West and East regions individually using 97W as a dividing line. The coarse "all-cells" averaging of the data from individual grid cells ignores the major features of the relationship between the sources and receptors on a sub-regional basis. For example, there are more grid cells with high urban density and high anthropogenic NO_X in the East, so the USA contribution will be higher in the East. Similarly, there are more high elevation areas in the West, so transported O_3 from outside the U.S. will be higher there. Within the West, however, there are also urban areas that have both high predicted contributions from international transport and anthropogenic emissions in the U.S. An analysis using "all-cells" will highlight the general characteristics of the region. To highlight the within region variability in the West, we also include analyses that focus on urban cells at high-elevation, near borders, and elsewhere. Figure 2-22 shows regions (West

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

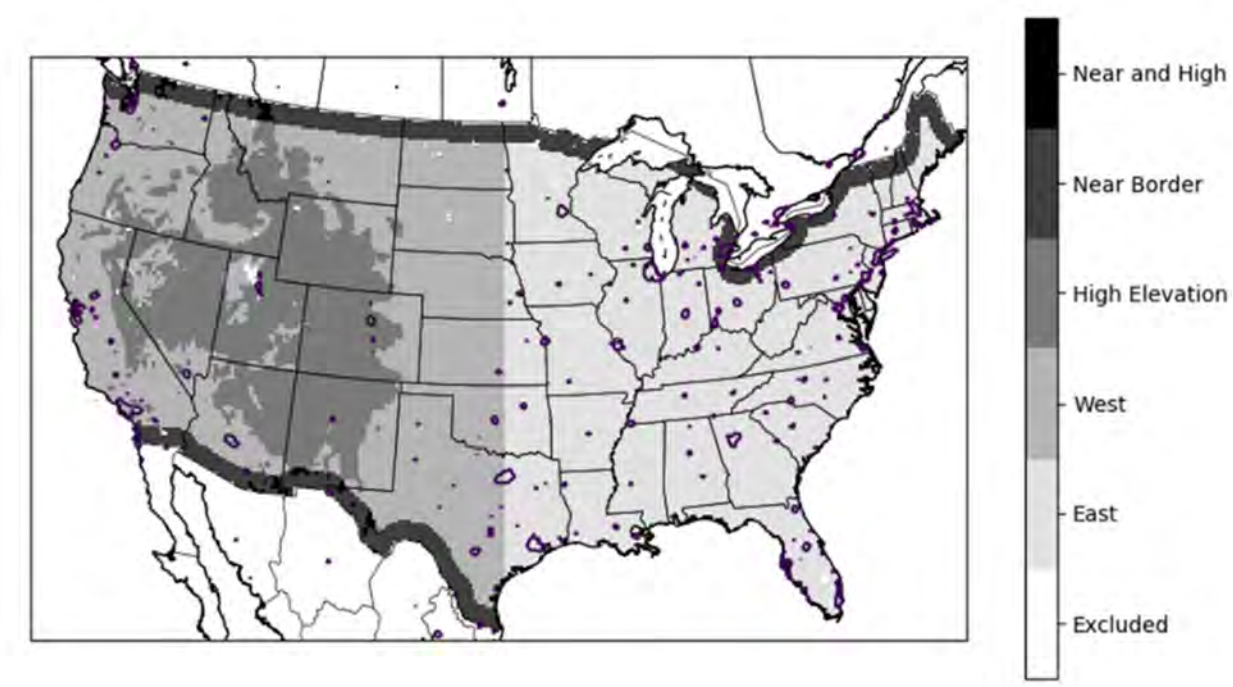


Figure 2-22. Grid cell assignments to East, West, High Elevation, Near Border, and Near and High (i.e., both High Elevation and Near Border). The purple outlines highlight grid cells with 20% or greater urban land use. Near Border areas are in both the West and East, while High Elevation areas are exclusively in the West. Areas matching colors denoted East and West, are thus the Low Elevation/Interior areas.

Figure 2-23 shows the time series of regional average ($\bar{C}$) MDA8 O_3 and O_3 contributions over the year for the West and East at "all-cells," calculated using equation 2-1.

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

where,

N_x = number of grid cells (x) included

C_x = concentration at each grid cell location (x)

The temporal pattern in the regional average clearly shows that the seasonality of MDA8 predictions for each total O_3 component varies by region. The natural contribution has a single maximum in late summer in the West, whereas, in the East there is evidence of two peaks— the largest in late Spring and a second peak in early Fall. The somewhat lower MDA8 O_3 in summer

in the East requires further analysis but may be related to the lack of lightning emissions within the regional domain. The seasonality international contribution predictions is more similar between the two regions. The international contributions in both the West and East are greatest in Spring, but the contribution in the West is larger both at its peak and its trough, compared to the East. The total international contribution and the separately analyzed long-distance components (e.g., China, India, international shipping) peak in spring when O_3 lifetimes favor long-range transport (see Appendix 2B, Figure 2B-29). However, the Canada/Mexico component of international contributions peaks in summer because of the relative proximity to the U.S. receptors. The predicted USA contribution increases in the summer for both the West and the East, but the USA contribution in the West is smaller than in the East. As mentioned previously, this "all cells" average is disproportionately rural in the West. The following analysis looks further at the different types of land in the West, including urban areas that are more representative of population centers that behave differently than the "all cells" analysis.

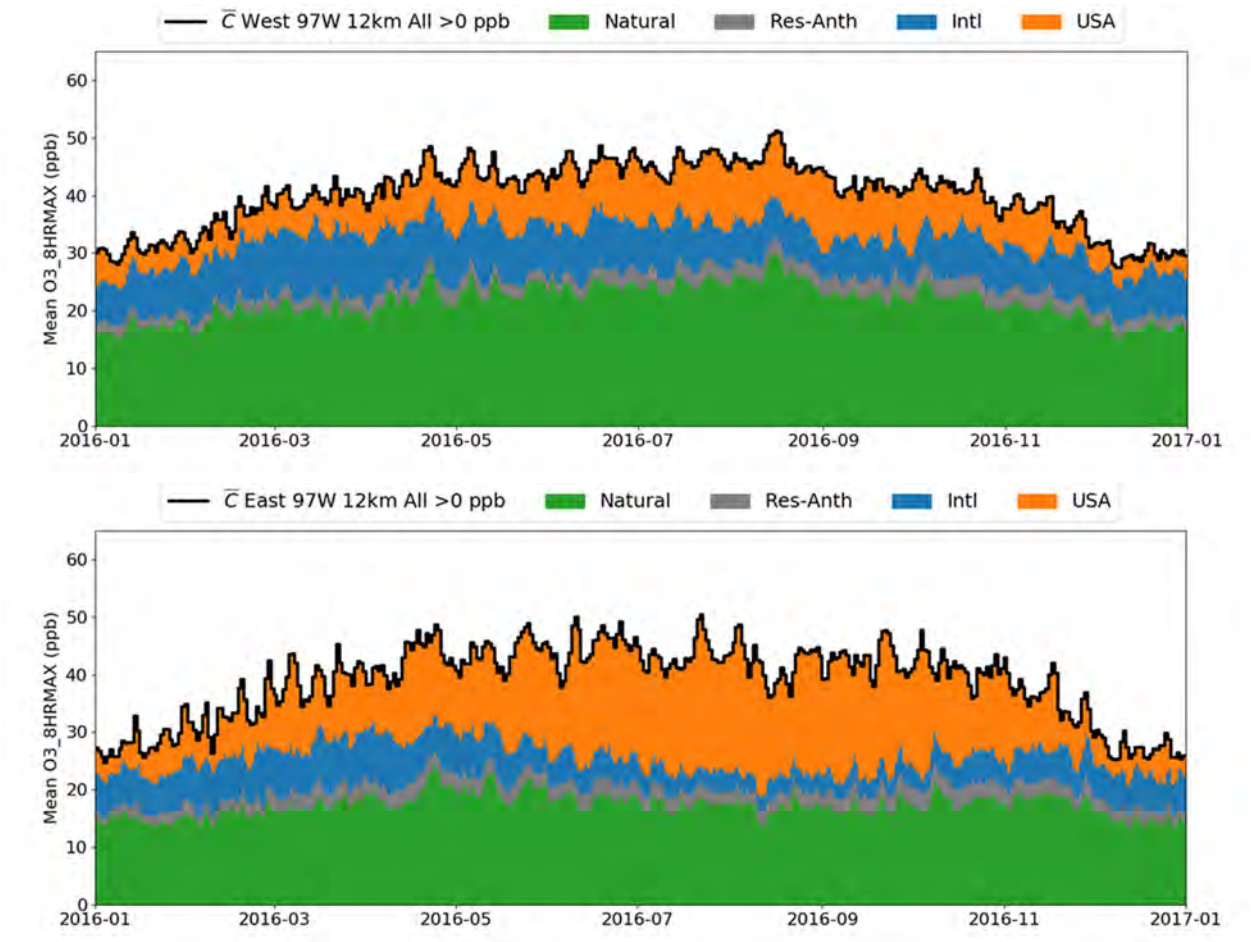


Figure 2-23. Annual time series of regional average predicted MDA8 total O_3 concentration and contributions of each source (see legend) for the West (top), and the East

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

Figure 2-24 shows the predicted contributions to total O_3 in the West split into three parts: the highest elevation areas, the near border areas, and Low/Interior areas with a weighted average focusing on urban areas. Each of these subsets is illustrated in Figure 2-22, which shows high elevation areas (exclusively in the West), near border areas (along the U.S./Mexico and U.S./Canada borders), and dense urban areas. The Low/Interior areas are neither high elevation nor near border. In each subset of cells, the purple outlines show the areas whose urban land use is highest. The effect on O_3 contributions of the relative amount of urban land use can be illustrated by computing an urban area weighted average contribution ($\overline{C^U}$), calculated using equation 2-2.

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

where,

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

The urban area weighted average gives a larger weight to data in those urban areas that have dense emission sources (e.g., mobile). The urban area weighted average shows higher contribution from USA while Natural and International are lower compared to Figure 2-23. The differences between urban-weighted and non-weighted contributions are smaller in the East (not shown) than in the West (compare Figure 2-23 top and Figure 2-24 bottom). Compared to the West, the East has a larger fraction of land use that is urban (see Figure 2-22), which explains this difference. Thus, the non-weighted regional average contributions in the East includes the effects of urban areas much more so than the West. The seasonality of International is also different between the highest elevation areas, near border areas, and urbanized areas. At low/interior and at high-elevation sites, the simulated International contribution peaks earlier in the year than at border sites. This earlier season peak is consistent with seasonality of O_3 lifetime necessary for long-range transport and a smaller contribution of long-distance sources (India, China, and Ships, see Appendix 2B, Figure 2B-30). At near-border sites, the seasonal cycle of predicted USB contributions from Canada/Mexico and from long-range transport combine to create a maximum later in the spring or early summer that is dominated by Canada/Mexico contributions (see Appendix 2B, Figure 2B-30, middle panel).

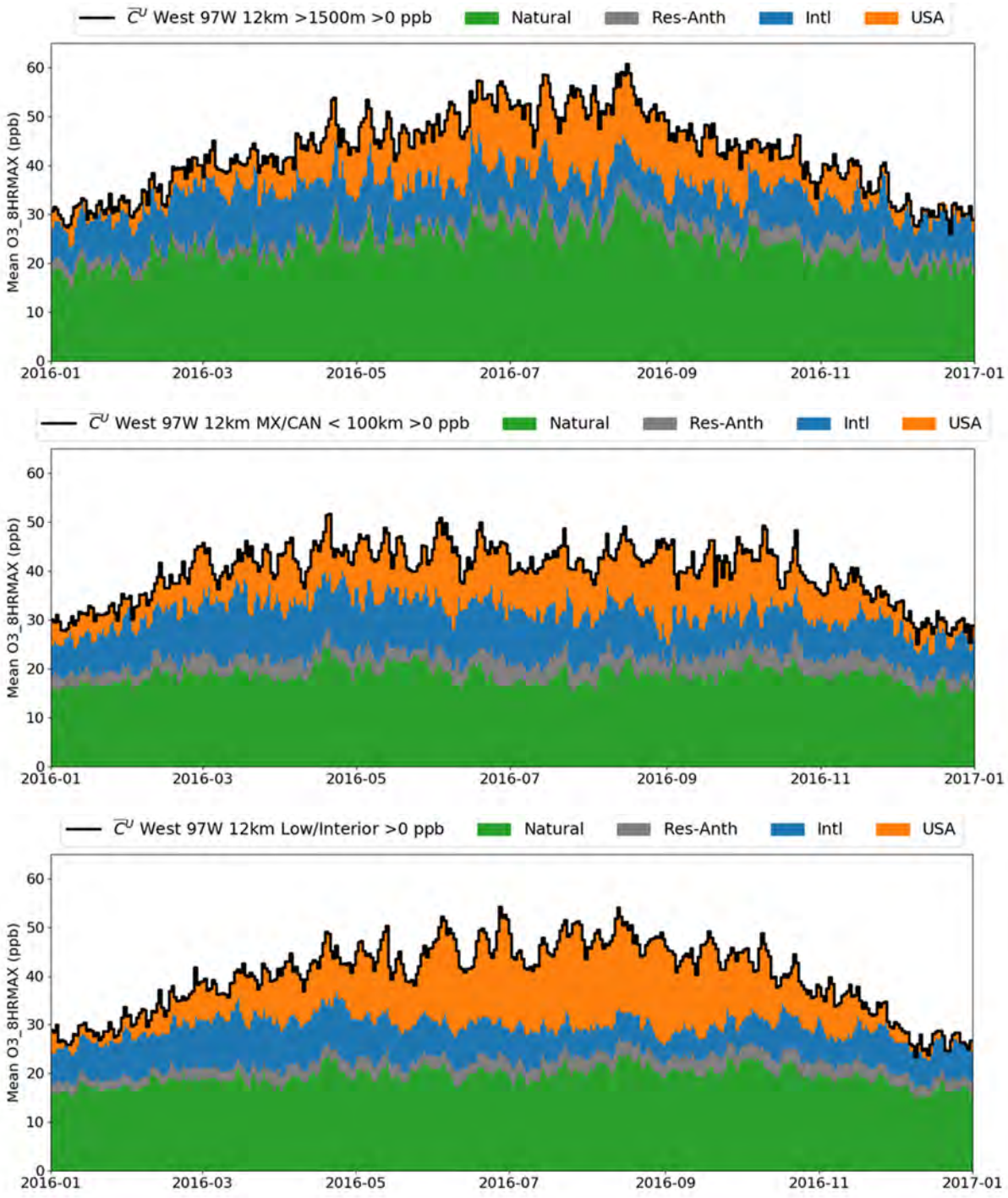


Figure 2-24. Annual time series of regional urban area-weighted average predicted MDA8 total O_3 concentration and contributions of each source (see legend) for the High-elevation West (top), near-border West (middle), and Low/Interior West (bottom). Natural is global natural sources, Intl is international anthropogenic sources, USA is U.S. anthropogenic sources, and Res-Anth is the residual 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 O_3. To illustrate the relationship, specialized scatter density plots were created to show the contributions as a function of total O_3. 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.[39] In the scatter density plots (Figure 2-25 through Figure 2-27), each pixel represents a 5 ppb O_3 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 O_3 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 O_3 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 O_3 concentrations are under 40-50 ppb. At these low concentrations, the natural contribution correlates well with total O_3 and frequently contributes half of the total O_3. 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 O_3 concentrations and is a well-known non-linearity of O_3 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 O_3, which represents enhancement by anthropogenic sources. The upper mode represents instances where natural contributions are correlated with total predicted O_3. 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 O_3 concentrations are also often overestimated by CMAQ predictions. As a result, these predictions of very high natural contributions should be interpreted

[39] When episodic natural events contribute to elevated O_3 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).

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

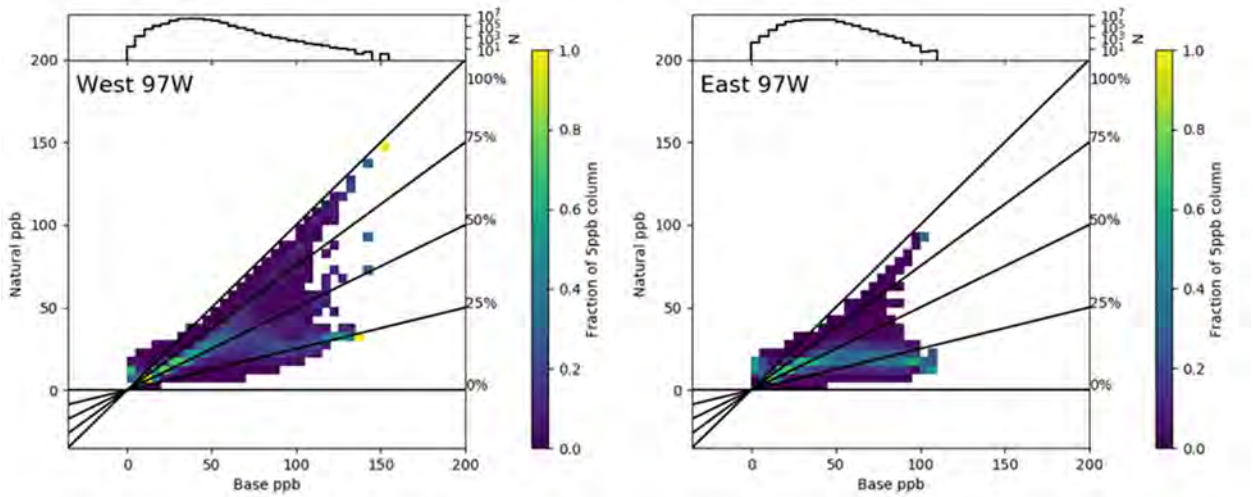


Figure 2-25. Predicted contribution of Natural as a function of predicted total (Base) MDA8 O_3 concentration in the West and East. Sloped lines show percent contribution as a quick reference. The number of cells in each column is identified using the probability density function above the plot, which is on a log scale that highlights infrequent high concentrations.

Figure 2-26 shows the predicted contribution in the West and East from international anthropogenic sources. Unlike natural contributions, there is very little correlation between international anthropogenic and total O_3. There are rare large model-predicted contributions, which are more frequent in the West than in the East and rarely contribute more than 50% total O_3 in either region. There are also negative contributions (up to -15 ppb), which arise from non-linearities in chemistry. The largest negative contribution predictions are along the Mexico border. These can either be NO_X-titration events or cases where chemistry associated with international NO_X-sources remove precursors that would otherwise enhance O_3 from U.S. sources. Negative international contributions tend to occur at relatively low total O_3 concentrations.

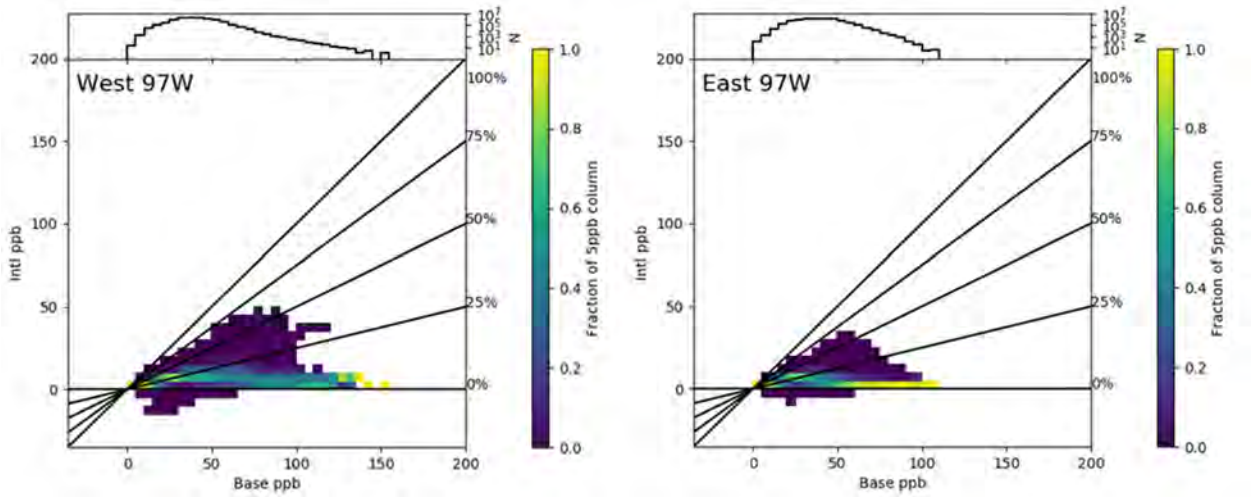


Figure 2-26. Predicted contribution of International as a function of predicted total (Base) MDA8 O_3 concentration in the West and East. Sloped lines show percent contribution as a quick reference. The number of cells in each column is identified using the probability density function above the plot, which is on a log scale that highlights infrequent high concentrations.

Figure 2-27 illustrates the relationship between predictions of U.S. anthropogenic sources and total O_3. Above 50 ppb, the predicted contribution from USA increases with total O_3 in both the West and the East. The relationship is stronger in the East, than the West, where near border contributions, fire contributions, and stratospheric exchange are smaller. Even so, the higher total O_3 in the West has a similar association of larger USA contributions at larger concentrations. This is consistent with previous findings (Henderson et al., 2012; U.S. EPA, 2014).

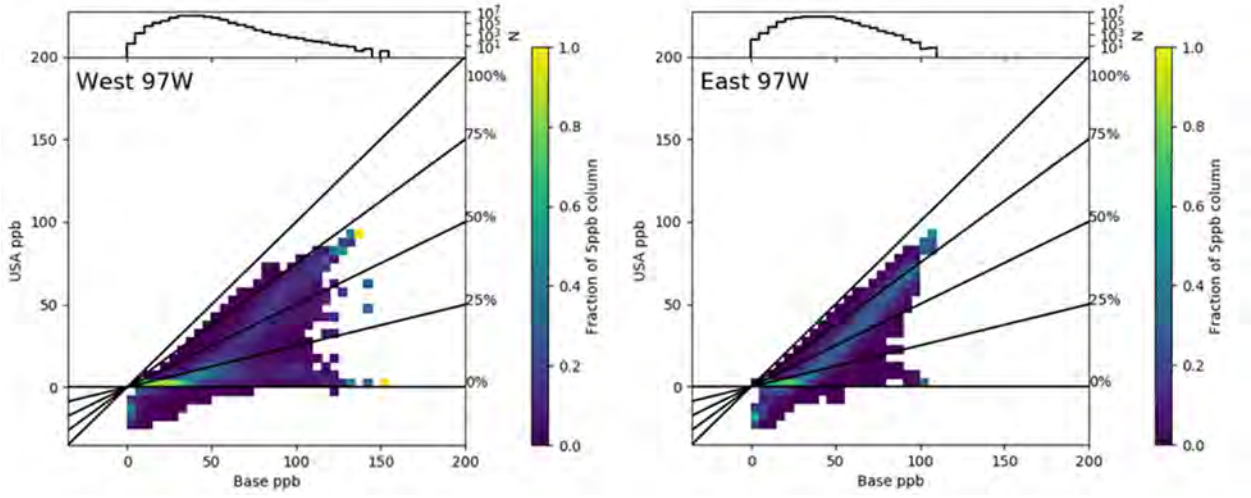


Figure 2-27. Predicted contribution of USA as a function of predicted total (Base) MDA8 O_3 concentration in the West and East. Sloped lines show percent contribution as a quick reference. The number of cells in each column is identified using the probability density function above the plot, which is on a log scale that highlights infrequent high concentrations.

Another way of looking at the contributions is to restrict the time series to grid cells where the concentration is above a threshold. Restricting to grid cells with high concentrations implicitly weights the results toward urban areas where these high concentrations occur most frequently. Figure 2-28 shows the seasonal and regional variation of USB (International Anthropogenic and Natural) and USA (anthropogenic only) sources on high O_3 days (MDA8 >70 ppb). The largest magnitude differences between sources in the East and West come from contributions predicted for Natural and USA sources. Recall that the West contains all the high-elevation areas (>1500 m) and the full length of the U.S./Mexican border. Figure 2-29 includes time series for high elevation, near Mexico border, and low-elevation interior areas separately. Compared to the East, the low/interior sites in the West have 9 ppb larger contribution from Natural and 2 ppb more from International. Compared to low/interior sites in the West, the high-elevation sites have 7 ppb larger contributions from Natural and 4 ppb more from International. For border areas, the International contribution is 13 ppb greater than in Low/Interior sites. As previously noted, there are large gradients of predicted international contributions even within the border areas, such that some locations within the 100 km of the border are predicted to receive larger international contributions while others are predicted to receive substantially smaller international contributions than noted above.

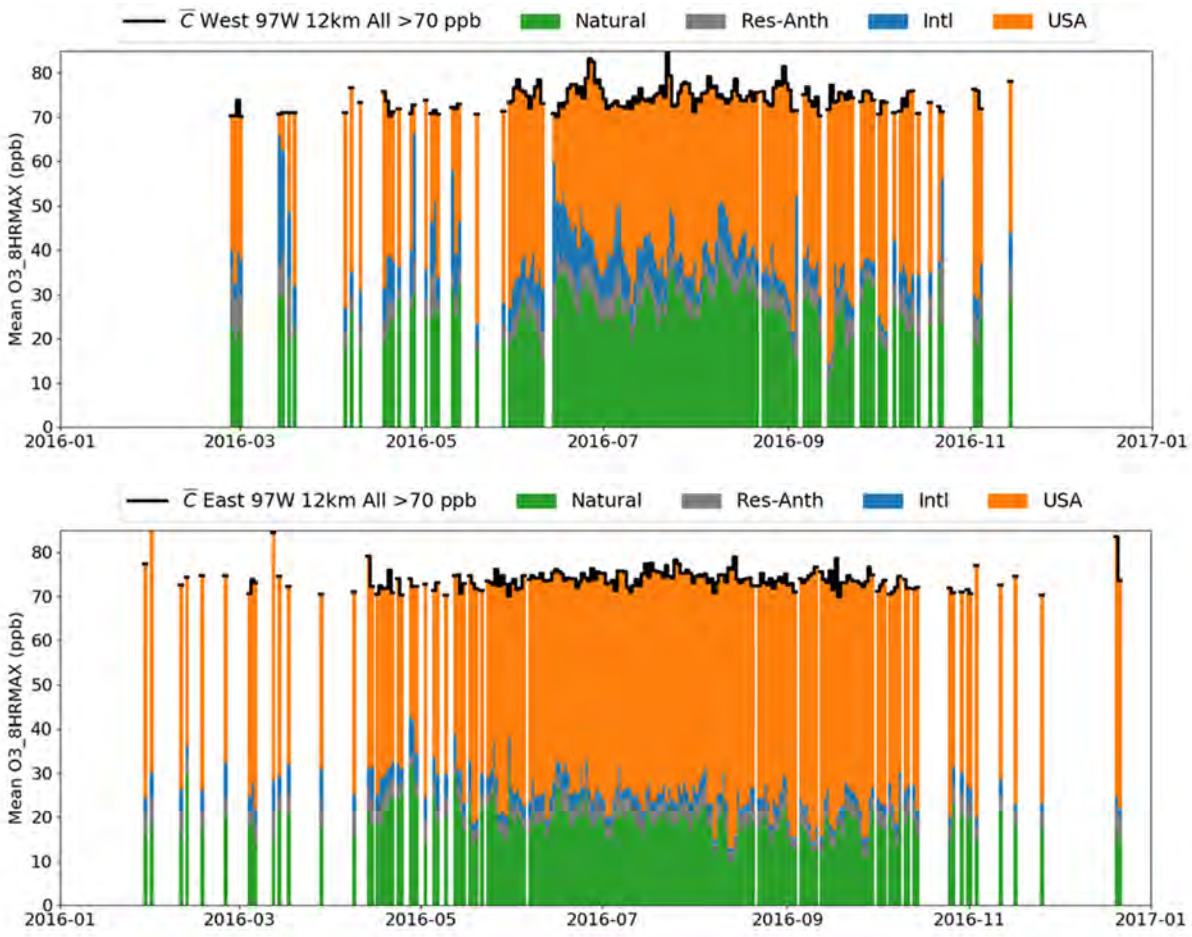


Figure 2-28. Annual time series of regional average predicted MDA8 O_3 and contributions of each source to predicted MDA8 total O_3 (see legend) in the West (top) and East (bottom) including only those grid-cell days with MDA8 greater than 70 ppb. Natural is global natural sources, Intl is international anthropogenic sources, USA is U.S. anthropogenic sources, and Res-Anth is the residual anthropogenic (see Table 2-2 for further descriptions).

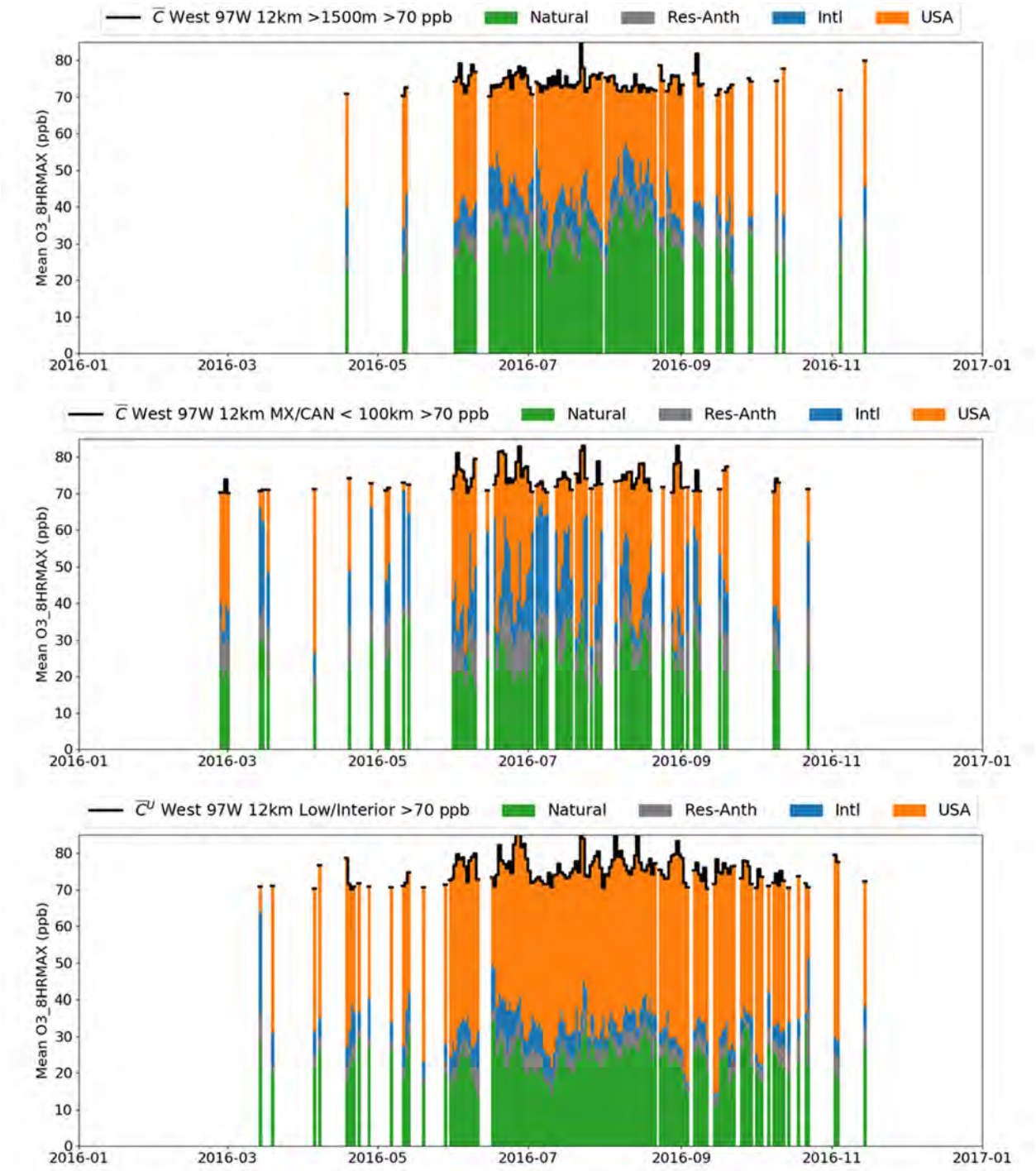


Figure 2-29. Annual time series of regional average predicted MDA8 O_3 and contributions of each source to predicted MDA8 O_3 (see legend) in the high-elevation West (top), in the near-border West (middle), and in the Low/Interior West weighted toward urban areas (bottom) including only those grid-cell days with MDA8 O_3 greater than 70 ppb. Natural is global natural sources, Intl is international anthropogenic sources, USA is U.S. anthropogenic sources, and Res-Anth is the residual anthropogenic (see Table 2-2 for further descriptions).

2.5.3.4 Predicted USB Seasonal Mean and USB on Peak O_3 Days

The analyses above describe the contributions from the components of USB to MDA8 O_3 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 O_3 concentrations (top 10 days); (2) on days predicted to have the 4th highest MDA8 total O_3 concentrations in the year; and, (3) on days when predicted MDA8 for total O_3 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 O_3 concentrations are predicted to be above 70 ppb. On the 10 highest predicted MDA8 O_3 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 O_3 concentrations above 70 ppb tend to occur in summer and fall when anthropogenic sources result in locally high increments of O_3. 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 O_3. As a

result, the areas with predicted top 10 days having MDA8 total O_3 above 70 ppb tend to have lower percentage USB contributions than the surrounding areas.

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

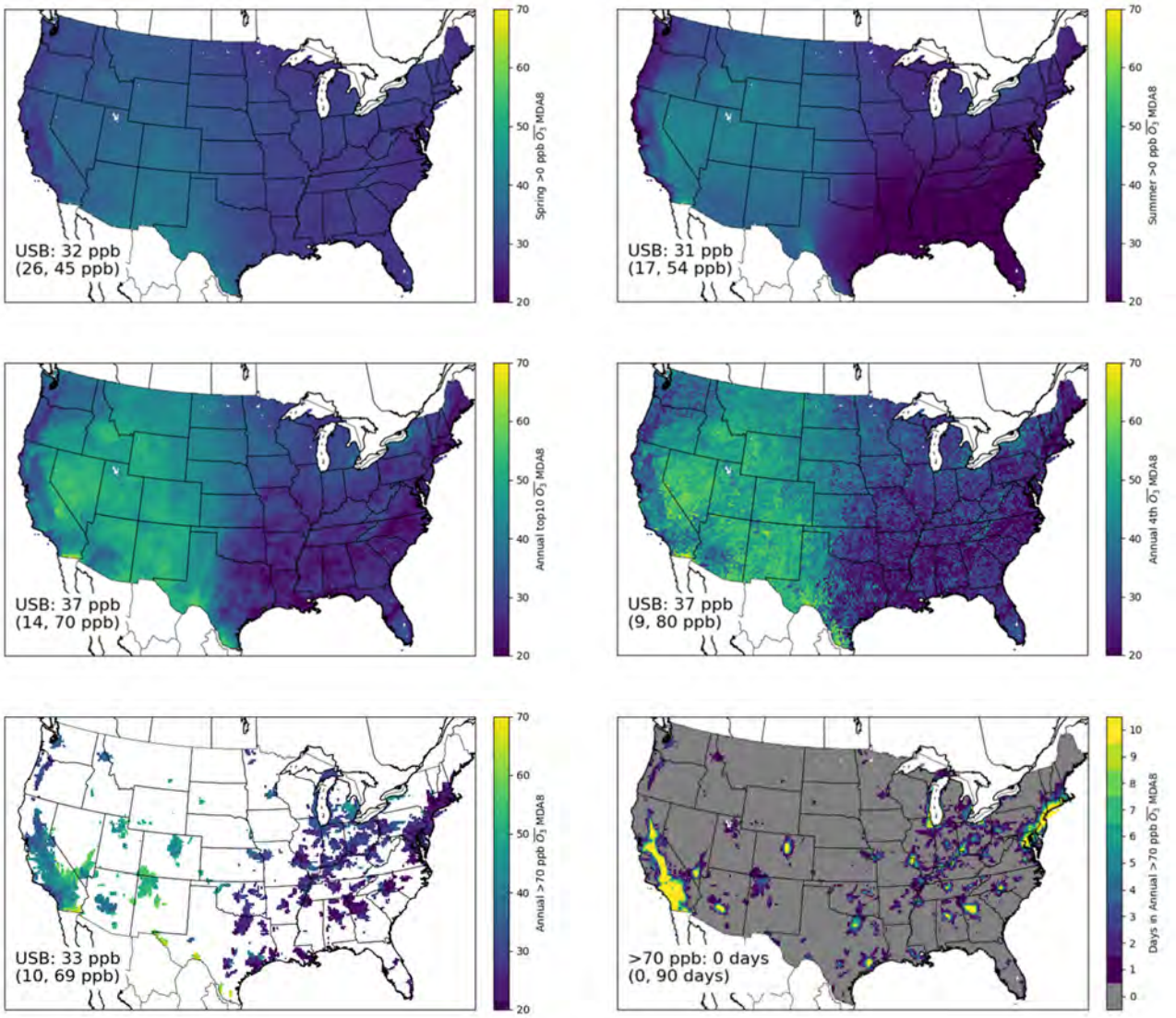


Figure 2-30. Map of predicted USB contributions by O_3 season for spring average (top left), summer average (top right), top 10 predicted total O_3 days (center left), 4th highest total O_3 simulated day (center right), and all days with total O_3 greater than 70 ppb (bottom left), along with a map of the number of days with total O_3 above 70 ppb (bottom right). Each contribution has the spatial average and range (min, max) in the lower left-hand corner of the panel.

The maps in Figure 2-30 provide a detailed spatial representation of predicted USB but may imply more precision than can be expected from a modeling system. For example, the maximum USB on predicted fourth highest day reaches 80 ppb near the Mexico border. The largest USB at nearby monitoring sites was 71 ppb.[40] The observed 4th highs at those monitors occurred in late February and early March, while the predicted 4th highs occurred in summer. After selecting the 4th highs based on the observations and applying bias correction proportionally to contributions, the new USB at these locations is 51 and 63 ppb. The USB values for any given grid cell may be biased due to local features of topography, meteorology, emissions bias, or model construct.

To complement the spatially resolved data and reduce bias associated with individual daily model predictions, we also spatially aggregate the data by NOAA climate region. The predicted USB values by climate region are provided in Table 2-3 to Table 2-6. Similar to the figures, the tables separately quantify all grid cells (Table 2-3), high elevation (>1500 m) areas (Table 2-4), near border areas (Table 2-5), and low-elevation (≤1500 m) interior areas (Table 2-6). These tables show the spatial averages of USB within each climate region for the annual average, seasonal averages, averages of days when MDA8 O_3 is greater than 60 or 70 ppb, averages of each grid cell's top 10-days, and each cell's 4th highest day. Note that top 10-day average and 4th high day for each grid cell may be from different times of the year compared to the neighboring grid cells. As a result, grid cells with highest O_3 driven by transport in the Spring are being mixed with grid cells with highest O_3 driven by local formation. Applying these averages to interpret observations must, therefore, be done in the full context of time, space, and concentration range.

[40] 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.

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

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	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.

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.

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.

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

	Mean MDA8 for Seasons or Year					Mean MDA8 of values in subset			**Annual 4th** highest MDA8
Regions[A]	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.

2.5.4 Summary of USB

Background O_3 results from a variety of sources, each of which has its own temporal pattern and spatial distribution. The location and timing of these sources impacts O_3 production, dispersion and loss and thus different background O_3 sources have unique seasonality and spatial patterns. The analysis presented here provides updated model-based estimates of magnitude, seasonality and spatial patterns of background O_3 contributions. The analysis separately characterizes the estimated magnitude and spatial/temporal patterns of MDA8 O_3 from three sources: natural, international anthropogenic, and USA anthropogenic.

The current analysis indicates that natural and USA O_3 contributions peak during the traditional O_3 season (May through September), while long-range intercontinental transport of international O_3 (i.e. contributions from China, India etc.) peaks in the spring (February through May). The contributions from Canada/Mexico at near-border locations are associated with relatively short-range transport and the seasonality peaks during May through September, similar to USA anthropogenic O_3. The influence of Canada/Mexico, however, is indicated by the model predictions to have a stronger spatial gradient in summer, so Canada/Mexico contributions are most evident near the border. Of the three categories of contributions, the USA anthropogenic is best correlated with total O_3 at concentrations above 40-50 ppb in both the West and the East

suggesting that US anthropogenic emissions are usually the driving cause of high O_3 events in the US. This is largely explained by temporal patterns of background O_3 influences in relation to typical high O_3 events. There can be exceptions to this rule that are generally associated natural contributions at high-elevation, during fires events, or at near-border sites.

This modeling analysis indicates the relationship between predicted international and USA anthropogenic contributions depend upon the international sources and the location. Long-range transport and USA anthropogenic contributions tend peak at different times of the year, so the contribution of international is often at its minimum when local sources are the driving factor for high total O_3 during the May through September O_3 season. Even in cases where O_3 formed from international anthropogenic emissions does coincide seasonally with high O_3 periods, the impact of those sources can have large spatial variation. For example, O_3 formed from anthropogenic emissions in Canada and Mexico can peak in late spring or early summer when total O_3 is high. During this time-period, there is a strong spatial variability not shown in the regional mean. As a result, specific days at specific locations may experience larger or smaller contributions from cross-border transport on an episodic basis that is not well characterized by average seasonal contributions. Another example of spatial heterogeneity is exemplified by wintertime O_3 events associated with emissions from local oil and gas production in the Intermountain West. Even though these episodes can occur as early in the year as February, international emissions may not contribute to them substantially. The conditions associated with these events result in decoupling of the local air masses from the upper atmosphere, essentially isolating air in the mountain valleys from the atmosphere above and reducing the influence of long-range transport compared to other winter and early spring days. As a result, these unique wintertime O_3 episodes may have little relative influence from international emissions despite occurring at a time of year when long-range transport from Asia is efficient. This highlights the need to perform location specific analysis rather than relying on regional averages.

In addition to seasonal patterns, the ISA highlights interannual patterns in background O_3 as well as long-term trends (ISA, section IS.2.2.1). Natural emissions and international transport are highly impacted by meteorological patterns which vary from year to year. One key ISA finding is that decreasing East Asian NO_X emissions starting around 2010, which would suggest decreasing contributions from East Asia in the future if those trends continue, and therefore decreasing spring USB.

Assessments of background O_3 in the last review reported regional variation in background O_3 (2013 ISA; 2014 PA). Consistent with those assessments, modeling presented here predicts that USB is higher in the West than in the East. In this analysis, we found that on high O_3 days (greater than 70 ppb) the West-East differences are largely associated with international contributions in near-border areas and natural contributions at high-elevation

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

The modeling performed for this assessment does not differentiate between natural sources of ozone. For this analysis we did not attempt to separately quantify the contributions from individual Natural sources (e.g., lightning, soil, fires, stratosphere) or to address exceptional events beyond basic screening to remove very large fire plumes. Literature-based emissions estimates and photochemical modeling studies can help to inform the likely contributors to natural. In the northern hemisphere, the natural NO_X sources with the largest emissions estimates are lightning (9.4 megatonN/yr), soils (5.5 megatonN/yr), and wildland fires (~2.2 megatonN/yr). Because NO_X is the limiting precursor at hemispheric scales, the emissions estimates suggest that lightning and soils are most likely the largest contributors to Natural O_3, except when impacted by specific fire episodes. As noted by Lapina et al. (2014), a large contribution from lightning may be the result of lightning strikes outside the U.S. while the contribution from soil NO_X tends to be largest from emissions within the U.S. The distant lightning source is likely to have its effect as part of the well-mixed background. The local soil NO_X emissions have a clear seasonal cycle and are known to have large local contributions. The relative effect at any specific site would require further analysis.

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

- USB has important spatial variation that is related to geography, topography, and international borders. The spatial variation is influenced by seasonal variation with long-range international transport contributions peaking in the spring while US anthropogenic contributions peak in summer.
- The West has higher predicted USB concentrations than the East, which includes higher contributions from International and Natural sources. Within the West, high-elevation and near-border areas stand out as having particularly high USB. The high-elevation areas have more International and Natural contributions than low-interior areas in the same region. The near-border areas in the West can have substantially more international contribution than other parts of the West.
- The USA contributions that drive predicted MDA8 total O_3 concentrations above 70 ppb are predicted to typically peak in summer. In this typical case, the predicted USB is overwhelmingly from Natural sources. The most notable exception to the typical case is reflected by predictions for an area near the Mexico border where the modeling indicates

that a combination of Natural and Canada/Mexico contributions can lead to predicted MDA8 USB concentrations 60-80 ppb, on specific days, which is consistent with the previous O_3 PA (Section 2.4).[41]

- Predicted international contributions, in most places, are lowest during the season with the most frequent occurrence of MDA8 concentrations above 70 ppb. Except for the near-border areas, the International contribution requires long-distance transport that is most efficient in Spring.
- Days for which MDA8 total O_3 concentrations are predicted to be above 70 ppb tend to have a substantially higher model-predicted USA (anthropogenic) contribution than other days in both the West and the East.

[41] 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 O_3 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 REVIEW OF THE PRIMARY STANDARD

This chapter presents and evaluates the policy implications of the key aspects of the currently available scientific and technical information pertaining to this review of the O_3 primary standard. In so doing, the chapter presents key aspects of the current evidence of the health effects of O_3, as documented in the ISA, with support from the prior ISA and AQCDs, and associated public health implications. It also presents key aspects of updated quantitative risk and exposure analyses conducted for this review, as detailed in the appendices associated with this chapter. Together this information provides the basis for our evaluation of the current scientific information regarding health effects of O_3 in ambient air and the potential for effects to occur under air quality conditions associated with the existing standard (or any alternatives considered), as well as the associated implications for public health. Our evaluation is framed around key policy-relevant questions derived from the IRP (IRP, section 3.1.1), and also takes into account conclusions reached in the last review. 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 review of the primary O_3 standard.

Within this chapter, background information on the current standard, including considerations in its establishment in the last review, is summarized in section 3.1. The general approach for considering the currently available information in this review, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 3.2. Key aspects of the currently available health effects evidence and associated public health implications and uncertainties are addressed in section 3.3, and the current air quality and exposure information, with associated uncertainties is addressed in section 3.4. Section 3.5 summarizes the key evidence- and air quality or exposure-based considerations identified in our evaluation, as well as the advice and recommendations received from the CASAC during its review of the draft PA and public comments received on the draft document, and also presents associated summary conclusions of this analysis. Key remaining uncertainties and areas for future research are identified in section 3.6.

3.1 BACKGROUND ON THE CURRENT STANDARD

The current primary standard was set in 2015 based on the scientific evidence and quantitative exposure and risk analyses available at that time, and on the Administrator's judgments regarding the available scientific evidence, the appropriate degree of public health protection for the revised standard, and the available exposure and risk information regarding the exposures and risk that may be allowed by such a standard (80 FR 65292, October 26, 2015).

The 2015 decision revised the level of the primary standard from 0.075 to 0.070 ppm,[1] in conjunction with retaining the then-current indicator (O_3), averaging time (eight hours), and form (annual fourth-highest daily maximum 8-hour average concentration, averaged across three consecutive years). This action provided increased protection for at-risk populations,[2] such as children and people with asthma, against an array of adverse health effects. The 2015 decision drew upon the available scientific evidence assessed in the 2013 ISA, the exposure and risk information presented and assessed in the 2014 health REA (HREA), the consideration of that evidence and information in the 2014 PA, the advice and recommendations of the CASAC, and public comments on the proposed decision (79 FR 75234, December 17, 2014).

The health effects evidence base available in the 2015 review included extensive evidence from previous reviews as well as the evidence that had emerged since the prior review had been completed in 2008. This evidence base, spanning several decades, documents the causal relationship between exposure to O_3 and a broad range of respiratory effects (2013 ISA, p. 1-14). Such effects range from small, reversible changes in pulmonary function and pulmonary inflammation (documented in controlled human exposure studies involving exposures ranging from 1 to 8 hours) to more serious health outcomes such as emergency department visits and hospital admissions, which have been associated with ambient air concentrations of O_3 in epidemiologic studies (2013 ISA, section 6.2). 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. Based on this evidence, the 2013 ISA concluded there to be a causal relationship between short-term O_3 exposures and respiratory effects, and also concluded that the relationship between longer-term exposure and respiratory effects was likely to be causal (2013 ISA, p. 1-14).[3]

[1] 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.

[2] 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 sulfur oxides as well as extrinsic, nonbiological factors, such as those related to socioeconomic status, reduced access to health care, or exposure.

[3] The 2013 ISA also concluded there likely to be a causal relationship between short-term exposure and mortality, as well as short-term exposure and cardiovascular effects, including related mortality, and that the evidence was suggestive of causal relationships between long-term O_3 exposures and total mortality, cardiovascular effects and

With regard to the short-term respiratory effects that were the primary focus of the 2015 decision, the controlled human exposure studies were recognized to provide the most certain evidence indicating the occurrence of health effects in humans following specific O_3 exposures (80 FR 65343, October 26, 2015; 2014 PA, section 3.4). These studies additionally illustrate the role of ventilation rate[4] and exposure duration in eliciting responses to O_3 exposure at the lowest studied concentrations. The exposure concentrations eliciting a given level of response in subjects at rest are higher than those eliciting a response in subjects exposed while at elevated ventilation, such as while exercising (2013 ISA, section 6.2.1.1).[5] Further, 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 2013 ISA identified several groups, including children and adults with asthma, as being at increased risk of O_3-related effects. In light of this finding with regard to children and adults with asthma, the exposure-based analyses in the HREA included these population groups (U.S. EPA, 2014, hereafter 2014 HREA, p. 3-14).

The exposure and risk information available in the 2015 review included exposure and risk estimates for air quality conditions just meeting the then-existing standard, and also for air quality conditions just meeting potential alternative standards. Estimates were derived for two exposure-based analyses, as well as for an analysis based on epidemiologic study associations. The first of the exposure-based analyses involved comparison of population exposure estimates at elevated exertion to exposure benchmark concentrations (exposures of concern).[6] These benchmark concentrations are based on exposure concentrations from controlled human

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reproductive and developmental effects, and between short-term and long-term O_3 exposure and nervous system effects (2013 ISA, section 2.5.2).

[4] Ventilation rate is a specific technical term referring to breathing rate in terms of volume of air taken into the body per unit of time. The units for ventilation rate ($\dot{V}_E$) are usually liters (L) per minute(min). Another related term is equivalent ventilation rate (EVR), which refers to $\dot{V}_E$ normalized by a person's body surface area in square meters (m^2). Accordingly, the units for EVR are generally L/min-m^2. For different activities, a person will experience different levels of exertion and different ventilation rates.

[5] In the controlled human exposure studies, the magnitude or severity of the respiratory effects induced by O_3 is influenced by ventilation rate and exposure duration, as well as 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 O_3-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 O_3-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 period (2013 ISA, section 6.2.1.1; Gong et al., 1986) after 2-hour exposure (heavy intermittent exercise) of young healthy adults (2013 ISA, section 6.2.1.1; McDonnell et al., 1983).

[6] The benchmark concentrations to which exposure concentrations experienced while at moderate or greater exertion were compared were 60, 70 and 80 ppb.

exposure studies in which lung function changes and other effects were measured in healthy, young adult volunteers exposed to O_3 while engaging in quasi-continuous moderate physical activity for a defined period (generally 6.6 hours).[7] The second exposure-based analysis provided population risk estimates of the occurrence of days with O_3-attributable lung function reductions of varying magnitudes by using the exposure-response (E-R) information in the form of E-R functions or other quantitative descriptions of biological processes.[8] In the epidemiologic study-based analysis, risk estimates were also derived from ambient air concentrations using concentration-response (C-R) functions derived from epidemiologic studies. These latter estimates were given less weight by the Administrator in her decision on the standard in light of conclusions reached in the PA and the HREA, which reflected lower confidence in these estimates (80 FR 65316-17, October 26, 2015).

The 2014 HREA developed exposure-based estimates for several population groups including all children and all adults. The type of exposure-based estimates that involved comparison of exposures to benchmarks was also derived for children with asthma and adults with asthma. The estimates of percentages of all children with exposures at or above benchmarks were virtually indistinguishable from the corresponding estimates for children with asthma.[9] When considered in terms of the number of children (rather than percentages of the child populations), the estimates for all children were much higher than those for children with asthma, with the magnitude of the differences varying based on asthma prevalence in each study area (2014 HREA, sections 5.3.2, 5.4.1.5 and section 5F-1). The estimates for percent of children experiencing an exposure at or above the benchmarks were higher than percent of adults due to the greater time that children spend outdoors and engaged in activities at elevated exertion (2014 HREA, section 5.3.2). Thus, consideration of the exposure-based results in the 2015 decision focused on the results for all children and children with asthma.

In weighing the 2013 ISA conclusions with regard to the health effects evidence and making judgments regarding the public health significance of the quantitative estimates of exposures and risks allowed by the then-existing standard and potential alternative standards considered, as well as judgments regarding margin of safety, the Administrator considered the

[7] The studies given primary focus were those for which O_3 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 O_3 exposure were compared to those after filtered air exposure.

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

[9] This reflects use of the same time-location-activity diary pool to construct each simulated individual's time-activity series, which is based on the similarities observed in the available diary data with regard to time spent outdoors and exertion levels (2014 HREA, sections 5.3.2 and 5.4.1.5).

currently available information and commonly accepted guidelines or criteria within the public health community, including statements of the American Thoracic Society (ATS), an organization of respiratory disease specialists,[10] advice from the CASAC and public comments. In so doing, she recognized that the determination of what constitutes an adequate margin of safety is expressly left to the judgment of the EPA Administrator (*Lead Industries Ass'n v. EPA*, 647 F.2d 1130, 1161-62 [D.C. Cir 1980], *cert. denied*, 449 U.S. 1042 [1980]; *Mississippi v. EPA*, 744 F.3d 1334, 1353 [D.C. Cir. 2013]). In NAAQS reviews generally, evaluations of how particular primary standards address the requirement to provide an adequate margin of safety include consideration of such factors as the nature and severity of the health effects, the size of the sensitive population(s) at risk, and the kind and degree of the uncertainties present. Consistent with past practice and long-standing judicial precedent, the Administrator took the need for an adequate margin of safety into account as an integral part of her decision-making.

In the 2015 decision, the Administrator first addressed the adequacy of protection provided by the then-existing primary standard and decided that the standard should be revised. Considerations related to that decision are summarized in section 3.1.1 below. The considerations and decisions on the revisions to the then-existing standard in order to provide the requisite protection under the Act, including an adequate margin of safety, are summarized in section 3.1.2.

3.1.1 Considerations Regarding Adequacy of the Prior Standard

The Administrator's conclusion regarding the adequacy of the primary standard that existed at the time of the last review was based on careful consideration of the available evidence, analyses and conclusions contained in the 2013 ISA, including information newly available in the review; the quantitative exposure and risk analyses in the 2014 HREA; the information, evaluations, considerations and conclusions presented in the 2014 PA; advice from the CASAC; and public comments. Key considerations informing the Administrator's decision that the then-current standard should be revised are summarized below.

The Administrator gave primary consideration to the evidence of respiratory effects from controlled human exposure studies, including those newly available in the review, and for which the exposure concentrations were at the lower end of those studied (80 FR 65343, October 26, 2015). This emphasis was consistent with CASAC comments on the strength of this evidence (Frey, 2014, p. 5). In placing weight on these studies, the Administrator took note of the variety of respiratory effects reported from the studies of healthy adults engaged in six 50-minute periods of moderate exertion within a 6.6-hour exposure to O_3 concentrations of 60 ppb and

[10] With regard to commonly accepted guidelines or criteria within the public health community, the 2014 PA considered statements issued by the ATS that had also been considered in prior reviews (ATS, 2000; ATS, 1985).

higher. The broadest range of effects (lung function decrements, respiratory symptoms, airway inflammation, airway hyperresponsiveness, and impaired lung host defense) have been studied and reported following such 6.6-hour exposures to 80 ppb O_3 or higher, and the most severe respiratory effects have been reported at these concentrations. The lowest exposure concentration in such studies for which a combination of statistically significant reduction in lung function and increase in respiratory symptoms was reported was 72 ppb,[11] while reduced lung function and increased pulmonary inflammation were reported following such exposures to O_3 concentrations as low as 60 ppb. In considering these findings, the Administrator noted that the combination of O_3-induced lung function decrements and respiratory symptoms met ATS criteria for an adverse response.[12] She additionally noted the CASAC comments on this point and also its caution that these study findings were for healthy adults and thus indicated the potential for such effects in some groups of people, such as people with asthma, at lower exposure concentrations (Frey, 2014, pp. 5-6). In light of this, the Administrator concluded that "the controlled human exposure studies indicate that adverse effects are likely to occur following exposures to O_3 concentrations below the [75 ppb] level of the [then-current] standard" (80 FR 65343, October 26, 2015).

The 2013 ISA indicated that the pattern of effects observed across the range of exposures assessed in the controlled human exposure studies, increasing with severity at higher exposures, is coherent with (i.e., reasonably related to) the health outcomes reported to be associated with ambient air concentrations in epidemiologic studies (e.g., respiratory-related hospital admissions, emergency department visits). With regard to the available epidemiologic studies, while analyses of O_3 air quality in the 2014 PA indicated that most O_3 epidemiologic studies reported health effect associations with O_3 concentrations in ambient air that violated the then-current (75 ppb) standard, the Administrator took particular note of a study that reported associations between short-term O_3 concentrations and asthma emergency department visits in children and adults in a U.S. location that would have met the then-current standard over the entire 5-year study period

[11] For the 70 ppb target exposure, Schelegle et al. (2009) reported, based on O_3 measurements during the six 50-minute exercise periods, that the mean O_3 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).

[12] The most recent statement from the ATS available at the time of the 2015 decision stated that "[i]n drawing the distinction between adverse and nonadverse reversible effects, this committee recommended that reversible loss of lung function in combination with the presence of symptoms should be considered as adverse" (ATS, 2000).

(80 FR 65344, October 26, 2015; Mar and Koenig, 2009).[13] [14] While uncertainties[15] limited the extent to which the Administrator based her conclusions on air quality in locations of multicity epidemiologic studies, she additionally noted some support from several multicity studies of morbidity or mortality in which the majority of study locations would have met the then-current standard (80 FR 65344, October 26, 2015; 2014 PA, section 3.1.4.2). Accordingly, looking across the body of epidemiologic evidence, the Administrator reached the conclusion that analyses of air quality in some study locations supported the occurrence of adverse O_3-associated effects at O_3 concentrations in ambient air that met, or are likely to have met, the then-current standard (80 FR 65344, October 26, 2016). Taken together, the Administrator concluded that the scientific evidence from controlled human exposure and epidemiologic studies called into question the adequacy of the public health protection provided by the 75 ppb standard that had been set in 2008.

In considering the exposure and risk information, the Administrator gave particular attention to the estimates of exposures of concern, focusing on the estimates for children, in 15 urban areas for air quality conditions just meeting the then-current standard. Consistent with the finding that larger percentages of children than adults were estimated to experience exposures at or above benchmarks, the Administrator focused on the results for all children and for children with asthma, noting that the results for these two groups, in terms of percent of the population group, are virtually indistinguishable (2014 HREA, sections 5.3.2, 5.4.1.5 and section 5F-1). In considering these estimates, she placed the greatest weight on estimates of two or more days with occurrences of exposures at or above the benchmarks, in light of her increased concern about the potential for adverse responses with repeated occurrences of such exposures. In particular, she noted that the types of effects shown to occur following exposures to O_3 concentrations from 60

[13] The design values in this location over the study period were at or somewhat below 75 ppb (Wells, 2012).

[14] The Administrator also took note of analyses in the 2014 PA for some single-city study locations where the then-current standard was not met during the study period (i.e., those evaluated in Silverman and Ito, 2010; Strickland et al., 2010), finding support for the association of hospital admissions and emergency department visits with short-term O_3 on subsets of days with virtually all ambient air O_3 concentrations below the level of the then-current standard. These analyses generally focused on the range of short-term concentrations for which the confidence intervals for the concentration-response relationship were tightest, finding these to be on many days with O_3 concentrations below the level of the then-current standard (80 FR 65344, October 26, 2015).

[15] Compared to the single-city epidemiologic studies the Administrator noted additional uncertainty in interpreting the relationships between short-term O_3 air quality in individual study cities and reported O_3 multicity effect estimates. This uncertainty applied specifically to interpreting air quality analyses within the context of multicity effect estimates for short-term O_3 concentrations, where effect estimates for individual study cities are not presented (as is the case for the key O_3 studies analyzed in the PA, with the exception of the study by Stieb et al. (2009) where none of the city-specific effect estimates for asthma emergency department visits were statistically significant) (80 FR 65344; October 26, 2015).

ppb to 80 ppb, such as inflammation, if occurring repeatedly as a result of repeated exposure, could potentially result in more severe effects based on the ISA conclusions regarding mode of action (80 FR 65343, 65345, October 26, 2015; 2013 ISA, section 6.2.3).[16] While generally placing the greatest weight on estimates of repeated exposures, the Administrator also considered estimates for single exposures at or above the higher benchmarks of 70 and 80 ppb (80 FR 65345, October 26, 2015). Further, while the Administrator recognized the effects documented in the controlled human exposure studies for exposures to 60 ppb to be less severe than those associated with exposures to higher O_3 concentrations, she also recognized there to be limitations and uncertainties in the evidence base with regard to unstudied population groups. As a result, she judged it appropriate for the standard, in providing an adequate margin of safety, to provide some control of exposures at or above the 60 ppb benchmark (80 FR 65345-65346, October 26, 2015).

With regard to multiple exposures, the HREA found that under conditions just meeting the then-current standard, fewer than 1% of children in the 15 study areas would be estimated to experience multiple days in a year with 8-hour exposures at or above 70 ppb while at elevated ventilation, while the percentage was as high as approximately 2% in the year and location with the highest exposure estimates (80 FR 65345 and Table 1, October 26, 2015). Although she expressed less concern with single occurrences, the Administrator noted that the then-current (75 ppb) standard could allow just over 3% of children to experience one or more days (i.e., at least one day), averaged over the years of analysis, with an 8-hour exposure at or above 70 ppb (while at moderate or greater exertion), based on the worst-case location, and up to 8% in the worst-case year and location (80 FR 65345, October 26, 2015). She additionally noted that, that in the worst-case year and location across the 15 study areas, the then-current standard could allow up to about 1% of children to experience at least one day per year with 8-hour exposures at elevated ventilation at or above 80 ppb, the highest benchmark evaluated (80 FR 65345, October 26, 2015). Additionally, while expressing less confidence in the adversity of effects observed following exposures as low as 60 ppb (particularly single exposures), the Administrator noted that the HREA found that under air quality conditions just meeting the then-current standard, approximately 3 to 8% of children in the 15 urban study areas (including approximately 3 to 8% of children with asthma), on average across the years of analysis, were estimated to experience two or more days per year with 8-hour exposures at or above 60 ppb, while at elevated ventilation (80 FR 65345, October 26, 2015).

[16] In addition to recognizing the potential for continued inflammation to evolve into other outcomes, the 2013 ISA also recognized that inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely (2013 ISA, p. 6-76; 80 FR 65331, October 26, 2015).

In considering these exposure estimates with regard to public health implications, the Administrator concluded that the exposures and risks projected to remain upon meeting the then-current (75 ppb) standard could reasonably be judged to be important from a public health perspective. In particular, this conclusion was based on her judgment that it is appropriate to set a standard that would be expected to eliminate, or almost eliminate, the occurrence of exposures, while at moderate exertion, at or above 70 and 80 ppb (80 FR 65346, October 26, 2015). In addition, given that the average percent of children estimated to experience two or more days with exposures at or above the 60 ppb benchmark approaches 10% in some urban study areas (on average across the analysis years), the Administrator concluded that the then-current standard did not incorporate an adequate margin of safety against the potentially adverse effects that could occur following repeated exposures at or above 60 ppb (80 FR 65345-46, October 26, 2015).

With regard to the HREA estimates of lung function risk in terms different magnitudes of decrements in forced expiratory volume in one second (FEV_1), the Administrator also gave greater weight to estimates of multiple occurrences than to single occurrences, while additionally noting CASAC advice regarding uses of FEV_1 decrement estimates as scientifically relevant surrogates for adverse health outcomes (Frey, 2014, p. 3). The Administrator noted that, when averaged over the years of evaluation, the then-current (75 ppb) standard was estimated to allow about 2 to 3% of children in the 15 urban study areas to experience two or more O_3-induced lung function decrements $\geq$15%, and to allow about 8 to 12% of children to experience two or more O_3-induced lung function decrements $\geq$10% (80 FR 65346, October 26, 2015). Although she recognized increased uncertainty in and placed less weight on both the HREA estimates for lung function risk and the epidemiologic-study-based risk estimates, the Administrator concluded that both types of estimates further support a conclusion that the O_3-associated health effects estimated to remain upon just meeting the then-current standard are an issue of public health importance on a broad national scale. Thus, she concluded that O_3 exposure and risk estimates, when taken together, supported a conclusion that the exposures and health risks associated with just meeting the then-current standard could reasonably be judged to be of public health significance, such that the then-current standard was not sufficiently protective and did not incorporate an adequate margin of safety.

In addition to the evidence and exposure/risk information, the Administrator also took note of CASAC advice, which included the finding that “the current NAAQS for ozone is not protective of human health” and the unanimous recommendation “that the Administrator revise

the current primary ozone standard to protect public health" (Frey, 2014, p. 5). She further noted similar CASAC advice in the prior 2008 review.[17]

In consideration of all of the above, the Administrator concluded that the then-current primary O_3 standard (with its level of 75 ppb) was not requisite to protect public health with an adequate margin of safety, and that it should be revised to provide increased public health protection. This decision was based on the Administrator's conclusions that the available evidence and exposure and risk information clearly called into question the adequacy of public health protection provided by the then-current primary standard such that it was "not appropriate, within the meaning of section 109(d)(1) of the CAA, to retain the current standard" (80 FR 65346, October 26, 2015).

3.1.2 Considerations for the Revised Standard

The following subsections focus on the individual elements - indicator, averaging time, form and level - for the new primary standard established in the 2015 review. While these sections summarize the Administrator's key considerations and conclusions for each element individually, she considered the elements collectively in evaluating the health protection afforded by the standard, consistent with past practice.

3.1.2.1 Indicator

In considering whether O_3 continued to be the most appropriate indicator for a standard meant to provide protection against photochemical oxidants in ambient air, the Administrator considered findings and assessments in the 2013 ISA and 2014 PA, as well as advice from the CASAC and public comment. The 2013 ISA specifically noted that O_3 is the only photochemical oxidant (other than nitrogen dioxide) that is routinely monitored and for which a comprehensive database exists (2013 ISA, section 3.6; 80 FR 65347, October 26, 2015). The PA additionally noted that, since the precursor emissions that lead to the formation of O_3 also generally lead to the formation of other photochemical oxidants, measures leading to reductions in population exposures to O_3 can generally be expected to lead to reductions in other photochemical oxidants. The CASAC indicated its view that O_3 is the appropriate indicator "based on its causal or likely causal associations with multiple adverse health outcomes and its representation of a class of pollutants known as photochemical oxidants" (Frey, 2014c, p. ii). Based on all of these considerations and public comments, the Administrator concluded that O_3 remained the most appropriate indicator for a standard meant to provide protection against photochemical oxidants

[17] The CASAC O_3 Panel for the 2008 review likewise recommended revision of the standard to one with a level below 75 ppb. This earlier recommendation was based entirely on the evidence and information in the record for the 2008 decision, which had been expanded in the 2015 review (Samet, 2011; Frey and Samet, 2012).

in ambient air, and she retained O_3 as the indicator for the primary standard (80 FR 65347, October 26, 2015).

3.1.2.2 Averaging time

The 8-hour averaging time for the primary O_3 standard was established in 1997 with the decision to replace the then-existing 1-hour standard with an 8-hour standard (62 FR 38856, July 18, 1997). The decision in that review was based on evidence from numerous controlled human exposure studies in healthy adults of adverse respiratory effects resulting from 6- to 8-hour exposures, as well as quantitative analyses indicating the control provided by an 8-hour averaging time of both 8-hour and 1-hour peak exposures and associated health risk (62 FR 38861, July 18, 1997; U.S. EPA, 1996). The 1997 decision was also consistent with advice from the CASAC (62 FR 38861, July 18, 1997; 61 FR 65727, December 13, 1996). The EPA reached similar conclusions in the subsequent 2008 review in which the 8-hour averaging time was retained (73 FR 16436, March 27, 2008).

In the review completed in 2015, the Administrator again considered the averaging time for the standard in light of both the strong evidence for O_3-associated respiratory effects following short-term exposures and the available evidence related to effects following longer-term exposures (80 FR 65347-50, October 26, 2015). In so doing, the Administrator noted the substantial health effects evidence from controlled human exposure studies that demonstrate that a wide range of respiratory effects (e.g., pulmonary function decrements, increases in respiratory symptoms, lung inflammation, lung permeability, decreased lung host defense, and airway hyperresponsiveness) occur in healthy adults following exposures ranging from 1 to 8 hours (80 FR 65348, October 26, 2015; 2013 ISA, section 6.2.1.1). The Administrator also noted the strength of evidence from epidemiologic studies that evaluated a wide variety of populations (e.g., including at-risk lifestages and populations, such as children and people with asthma, respectively) using a number of different short-term averaging times, including the maximum 1-hour concentration within a 24-hour period (1-hour max), the maximum 8-hour average concentration within a 24-hour period (8-hour max), and the 24-hour average (80 FR 65348, October 26, 2015; 2013 ISA, chapter 6). It was recognized that an 8-hour averaging time is similar to the exposure periods evaluated in the more recent controlled human exposure studies conducted at the lowest concentrations, and the Administrator noted that the epidemiologic evidence alone did not provide a strong basis for distinguishing between the appropriateness of 1-hour, 8-hour and 24-hour averaging times. Thus, in consideration of the then-available health effects information, the Administrator concluded that an 8-hour averaging time remained appropriate for addressing health effects associated with short-term exposures to ambient air O_3 (80 FR 65348, October 26, 2015).

In considering the evidence related to longer-term exposures, the Administrator initially considered the extent to which currently available evidence and exposure/risk information suggested that a standard with an 8-hour averaging time can provide protection against respiratory effects associated with longer-term exposures to ambient air O_3. As in previous reviews, the review completed in 2015 recognized and further evaluated changes in long-term air quality patterns in response to attaining an 8-hour standard and the reduction in potential risk of health effects associated with such patterns in areas meeting an 8-hour standard (80 FR 65348, October 26, 2015).[18] Furthermore, the Administrator observed that the CASAC agreed with the choice of an 8-hour averaging time (Frey, 2014, p. ii). Therefore, based on the then-available evidence and information discussed in detail in the 2013 ISA, 2014 HREA, and 2014 PA, along with CASAC advice and public comments, the Administrator concluded that a standard with an 8-hour averaging time could effectively limit health effects attributable to both short- and long-term O_3 exposures and that it was appropriate to retain the 8-hour averaging time (80 FR 65350, October 26, 2015).

3.1.2.3 Form

While giving foremost consideration to the adequacy of public health protection provided by the combination of all elements of the standard, including the form, the Administrator placed considerable weight on the findings from prior reviews with regard to the use of the *n*th-high metric, as described below (80 FR 65350-65352, October 26, 2015). Based on these findings and consideration of CASAC advice, the Administrator judged it appropriate to retain the fourth-high form, more specifically the annual fourth-highest daily maximum 8-hour O_3 average concentration, averaged over 3 years (80 FR 65352, October 26, 2015).

The concentration-based form was established in the 1997 review when it was recognized that such a form better reflects the continuum of health effects associated with increasing O_3 concentrations than an expected exceedance form, which had been the form of the standard prior to 1997. Unlike an expected exceedance form, a concentration-based form gives proportionally more weight to years when 8-hour O_3 concentrations are well above the level of the standard than years when 8-hour O_3 concentrations are just above the level of the standard. More weight was given to high O_3 concentrations, in light of the available health evidence that indicated a continuum of effects associated with exposures to varying concentrations of O_3, and because the extent to which public health is affected by exposure to O_3 in ambient air is related to the actual magnitude of the O_3 concentration, not just whether the concentration is above a specified level.

[18] Analyses described in detail in the 2014 HREA suggested that reductions in O_3 precursors emissions in order to meet a standard with an 8-hour averaging time, coupled with the appropriate form and level, would be expected to reduce O_3 concentrations in terms of the metrics reported in epidemiologic studies to be associated with respiratory morbidity and mortality (80 FR 65348, October 26, 2015).

With regard to a specific concentration-based form, the fourth-highest daily maximum was selected in 1997, recognizing that a less restrictive form (e.g., fifth highest) would allow a larger percentage of sites to experience O_3 peaks above the level of the standard, and would allow more days on which the level of the standard may be exceeded when the site attains the standard (62 FR 38868-38873, July 18, 1997), and there was not an basis identified for selection of a more restrictive form (62 FR 38856, July 18, 1997).

In the subsequent 2008 review, the EPA considered the potential value of a percentile-based form, recognizing that such a statistic is useful for comparing datasets of varying length because it samples approximately the same place in the distribution of air quality values, whether the dataset is several months or several years long (73 FR 16474, March 27, 2008). However, the EPA concluded that, because of the differing lengths of the monitoring season for O_3 across the U.S., a percentile-based statistic would not be effective in ensuring the same degree of public health protection across the country. Specifically, a percentile-based form would allow more days with higher air quality values (i.e., higher O_3 concentrations) in locations with longer O_3 seasons relative to locations with shorter O_3 seasons. Thus, the EPA concluded in the 2008 review that a form based on the n^{th}-highest maximum O_3 concentration would more effectively ensure that people who live in areas with different length O_3 seasons received the same degree of public health protection (73 FR 16474-75, March 27, 2008). The importance of a form that provides stability to ongoing control programs was also recognized (73 FR 16474, March 27, 2008). In the case of O_3, 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 O_3 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).

In the 2015 review, the Administrator continued to recognize the considerations supporting the decisions in 1997 and 2008, and additionally noted recent CASAC advice in which the CASAC indicated that the O_3 standard should be based on the fourth-highest, daily maximum 8-hour average value (averaged over 3 years), by stating that this form "provides health protection while allowing for atypical meteorological conditions that can lead to abnormally high ambient ozone concentrations which, in turn, provides programmatic stability" (Frey, 2014, p. 6; 80 FR 65352, October 26, 2015).

3.1.2.4 Level

The Administrator's decision to set the level of the revised primary O_3 standard at 70 ppb built upon her conclusion (summarized in section 3.1.1 above) that the overall body of scientific

evidence and exposure/risk information called into question the adequacy of the public health protection afforded by the then-current standard, particularly for at-risk populations and lifestages (80 FR 65362, October 26, 2015). In her decision on level, the Administrator placed the greatest weight on the results of controlled human exposure studies and on quantitative analyses based on information from these studies, particularly analyses of O_3 exposures of concern. The Administrator viewed the results of the lung function risk assessment, analyses of O_3 air quality in locations of epidemiologic studies, and epidemiologic-study-based quantitative health risk assessment as providing information in support of her decision to revise the then-current standard, but of less utility for selecting a particular standard level among a range of options (80 FR 65362, October 26, 2015).

In placing weight on information from controlled human exposure studies and analyses based on information from these studies, the Administrator noted that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O_3 exposures, noting in particular that the effects reported in the controlled human exposure studies are due solely to O_3 exposures, and are not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). The Administrator's emphasis on the information from the controlled human exposure studies was consistent with the CASAC's advice and interpretation of the scientific evidence (80 FR 65362, October 26, 2015; Frey, 2014). In this regard, the Administrator recognized that: (1) the largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures to 80 ppb O_3 or higher (i.e., decreased lung function, increased airway inflammation, increased respiratory symptoms, airway hyperresponsiveness, and decreased lung host defense); (2) exposures to O_3 concentrations somewhat above 70 ppb have been shown to both decrease lung function and to result in respiratory symptoms; and (3) exposures to O_3 concentrations as low as 60 ppb have been shown to decrease lung function and to increase airway inflammation (80 FR 65363, October 26, 2015).

The Administrator considered both ATS recommendations and CASAC advice to inform her judgments on the potential adversity to public health of effects reported in controlled human exposure studies (80 FR 65363, October 26, 2015). In so doing, the Administrator concluded that the evidence from controlled human exposure studies provided strong support for the conclusion that a revised standard with a level of 70 ppb is requisite to protect public health with an adequate margin of safety. This conclusion was based, in part, on the fact that such a standard level would be well below the O_3 exposure concentration documented to result in the widest range of respiratory effects (i.e., 80 ppb), and below the lowest O_3 exposure concentration shown to result in the adverse combination of lung function decrements and respiratory symptoms (80 FR 65363, October 26, 2015).

In considering the degree of protection provided by a revised primary O_3 standard, the Administrator considered the extent to which that standard would be expected to limit population exposures to the broad range of O_3 exposures shown to result in health effects (80 FR 65363, October 26, 2015). In considering the exposure estimates from the HREA, the Administrator focused on the estimates of two or more exposures of concern in order to provide a health-protective approach to take into account the potential for repeated occurrences of exposures that could result in adverse effects. In so doing, she placed the most emphasis on setting a standard that appropriately limits repeated occurrences of exposures at or above the 70 and 80 ppb benchmarks, while at elevated ventilation. She noted that a revised standard with a level of 70 ppb was estimated to eliminate the occurrence of two or more days with exposures at or above 80 ppb and to virtually eliminate the occurrence of two or more days with exposures at or above 70 ppb for all children and children with asthma, even in the worst-case year and location evaluated.[19] Given the considerable protection provided against repeated exposures of concern for all benchmarks evaluated in the HREA, the Administrator judged that a standard with a level of 70 ppb incorporated a margin of safety against the adverse O_3-induced effects shown to occur in the controlled human exposure studies (80 FR 65364, October 26, 2015).[20]

While she was less confident that adverse effects would occur following exposures to O_3 concentrations as low as 60 ppb,[21] as discussed above, the Administrator judged it to also be appropriate to consider estimates of exposures (while at moderate or greater exertion) for the 60 ppb benchmark (80 FR 65363-64, October 26, 2015). In so doing, she recognized that while CASAC advice regarding the potential adversity of effects observed in studies of 60 ppb was less definitive than for effects observed at the next higher concentration studied, the CASAC did clearly advise the EPA to consider the extent to which a revised standard is estimated to limit the effects observed in studies of 60 ppb exposures (80 FR 65364, October 26, 2015; Frey, 2014). The Administrator's consideration of exposures at or above the 60 ppb benchmark, and particularly consideration of multiple occurrences of such exposures, was primarily in the

[19] 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).

[20] 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 is inclusive of 70 ppb) to be a policy judgment (80 FR 65355, October 26, 2015; Frey, 2014).

[21] The Administrator was "notably less confident in the adversity to public health of the respiratory effects that have been observed following exposures to O_3 concentrations as low as 60 ppb," based on her consideration of the ATS recommendation on judging adversity from transient lung function decrements alone, the uncertainty in the potential for such decrements to increase the risk of other, more serious respiratory effects in a population (per ATS recommendations on population-level risk), and the less clear CASAC advice regarding potential adversity of effects at 60 ppb compared to higher concentrations studied (80 FR 65363, October 26, 2015).

context of considering the extent to which the health protection provided by a revised standard included a margin of safety against the occurrence of adverse O_3-induced effects (80 FR 65334-35, 65364, October 26, 2015). In this context, the Administrator noted that a revised standard with a level of 70 ppb was estimated to protect the vast majority of children in urban study areas (i.e., about 96% to more than 99% of children in individual areas) from experiencing two or more days with exposures at or above 60 ppb (while at moderate or greater exertion). Compared to the estimates for the then-current standard (with its level of 75 ppb), this represented a reduction of more than 60%. Given the considerable protection provided against repeated exposures of concern for all of the benchmarks evaluated, including the 60 ppb benchmark, the Administrator judged that a standard with a level of 70 ppb would incorporate a margin of safety against the adverse O_3-induced effects shown to occur following exposures (while at moderate or greater exertion) to a somewhat higher concentration. The Administrator also judged the HREA results for one or more exposures at or above 60 ppb to provide further support for her somewhat broader conclusion that "a standard with a level of 70 ppb would incorporate an adequate margin of safety against the occurrence of O_3 exposures that can result in effects that are adverse to public health" (80 FR 65364, October 26, 2015).[22]

While placing limited weight on the lung function risk estimates,[23] epidemiologic evidence[24] and quantitative estimates based on information from the epidemiologic studies, the Administrator additionally considered that information in the context of her consideration of a

[22] While the Administrator was less concerned about single occurrences of O_3 exposures of concern, especially for the 60 ppb benchmark, she judged that estimates of one or more exposures of concern can provide further insight into the margin of safety provided by a revised standard. 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).

[23] The Administrator noted important uncertainties in using lung function risk estimates as a basis for considering the occurrence of adverse effects in the population (also recognized in the prior review) that limited her reliance on these estimates to distinguish between the appropriateness of the health protection afforded by a standard level of 70 ppb versus lower levels (80 FR 65364, October 26, 2015). These uncertainties related to (1) the ATS recommendation that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" (ATS, 2000); (2) uncertainty in the extent to which a transient population-level decrease in FEV_1 would increase the risk of other, more serious respiratory effects in that population (i.e., per ATS recommendations on population-level risk); and (3) that the CASAC did not advise considering a standard that would be estimated to eliminate O_3-induced lung function decrements ≥10 or 15% (Frey, 2014); 80 FR 65364, October 26, 2015).

[24] While the Administrator concluded that analyses of air quality in single-city epidemiologic studies support a level at least as low as 70 ppb, based on a study (Mar and Koenig, 2009) reporting health effect associations in a location that met the then-current standard over the entire study period but that would have violated a revised standard with a level of 70 ppb, she further judged that they are of more limited utility for distinguishing between the appropriateness of the health protection estimated for a standard level of 70 ppb and the protection estimated for lower levels (80 FR 65364, October 26, 2015).

standard with a level of 70 ppb. For example, she judged that a standard with a level of 70 ppb would be expected to result in important reductions in the population-level risk of O_3-induced lung function decrements in children, including children with asthma (80 FR 65364, October 26, 2015). With regard to the epidemiologic evidence, the Administrator noted that a revised standard with a level of 70 ppb would provide additional public health protection, beyond that provided by the then-current standard, against the clearly adverse effects analyzed in epidemiologic studies (80 FR 65364, October 26, 2015). With regard to the epidemiology-based risk estimates, the Administrator judged that a revised standard with a level of 70 ppb will result in meaningful reductions in the mortality and respiratory morbidity risk that is associated with short- or long-term concentrations of O_3 in ambient air (80 FR 65365, October 26, 2015).

In summary, given her consideration of the evidence, exposure and risk information, advice from the CASAC, and public comments, the Administrator judged a primary standard of 70 ppb in terms of the 3-year average of annual fourth-highest daily maximum 8-hour average O_3 concentrations to be requisite to protect public health, including the health of at-risk populations, with an adequate margin of safety (80 FR 65365, October 26, 2015).

3.2 GENERAL APPROACH AND KEY ISSUES IN THIS REVIEW

As is the case for all such reviews, this review of the primary O_3 standard is most fundamentally based on using the Agency's assessment of the current scientific evidence and associated quantitative analyses to inform the Administrator's judgments regarding a primary standard that is requisite to protect public health with an adequate margin of safety. The approach for this review builds on the substantial assessments and evaluations performed over the course of the last review, taking into account the more recent scientific information and air quality data now available to inform our understanding of the key-policy relevant issues in this review.

The evaluations in the PA of the scientific assessments in the ISA augmented by the quantitative risk and exposure analyses[25] are intended to inform the Administrator's public health policy judgments and conclusions, including his decisions as to whether to retain or revise the primary O_3 standard. The PA evaluations consider the potential implications of various aspects of the scientific evidence, the exposure/risk-based information, and the associated uncertainties and limitations. In so doing, the approach for this PA involves evaluating the scientific and technical information to address a series of key policy-relevant questions using both evidence-

[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.

and exposure/risk-based considerations. Together, consideration of the full set of evidence and information available in this review will inform the answer to the following initial overarching question for the review:

- **Do the currently available scientific evidence and exposure-/risk-based information support or call into question the adequacy of the public health protection afforded by the current primary O_3 standard?**

In reflecting on this question, we will consider the available body of scientific evidence, assessed in the ISA and used as a basis for developing or interpreting exposure/risk analyses, including whether it supports or calls into question the scientific conclusions reached in the last review regarding health effects related to exposure to ambient air-related O_3. Information available in this review that may be informative to public health judgments regarding significance or adversity of key effects will also be considered. Additionally, the currently available exposure and risk information, whether newly developed in this review or predominantly developed in the past and interpreted in light of current information, will be considered, including with regard to the extent to which it may continue to support judgments made in the last review. Further, in considering this question with regard to the primary O_3 standard, as in all NAAQS reviews, we give particular attention to exposures and health risks to at-risk populations (including at-risk lifestages).[26]

Evaluation of the available scientific evidence and exposure/risk information with regard to this consideration of the current standard will focus on key policy-relevant issues by addressing a series of questions including the following:

- Is there newly available evidence that indicates the importance of photochemical oxidants other than O_3 with regard to abundance in ambient air, and potential for human exposures and health effects?
- Does the currently available scientific evidence alter our conclusions from the last review regarding the nature of health effects attributable to human exposure to O_3 from ambient air?
- Does the current evidence alter our understanding of populations that are particularly at risk from O_3 exposures?
- Does the current evidence alter our conclusions from the previous review regarding the exposure duration and concentrations associated with health effects? To what extent does the currently available scientific evidence indicate health effects attributable to exposures to O_3

[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 O_3. *Vulnerability* refers to an increased risk of O_3-related health effects due to factors such as those related to socioeconomic status, reduced access to health care or exposure.

concentrations lower than previously reported and what are important uncertainties in that evidence?

- To what extent have previously identified uncertainties in the health effects evidence been reduced or do important uncertainties remain?
- What are the nature and magnitude of O_3 exposures and associated health risks associated with air quality conditions just meeting the current standard?
- To what extent are the estimates of exposures and risks to at-risk populations associated with air quality conditions just meeting the current standard reasonably judged important from a public health perspective?
- What are the important uncertainties associated with any exposure/risk estimates?

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

- Does the currently available information call into question the identification of O_3 as the indicator for photochemical oxidants? Is support provided for considering a different indicator?
- Does the currently available information call into question the current averaging time? Is support provided for considering different averaging times for the standard?
- What does the currently available information indicate with regard to a range of levels and forms of alternative standards that may be supported and what are the uncertainties and limitations in that information?
- What do the available analyses indicate with regard to exposure and risk associated with specific alternative standards? What are the associated uncertainties? To what extent might such alternatives be expected to reduce adverse impacts attributable to O_3, and what are the uncertainties in the estimated reductions?

The approach to reaching conclusions on the current primary standard and, as appropriate, on potential alternative standards is summarized in general terms in Figure 3-1.

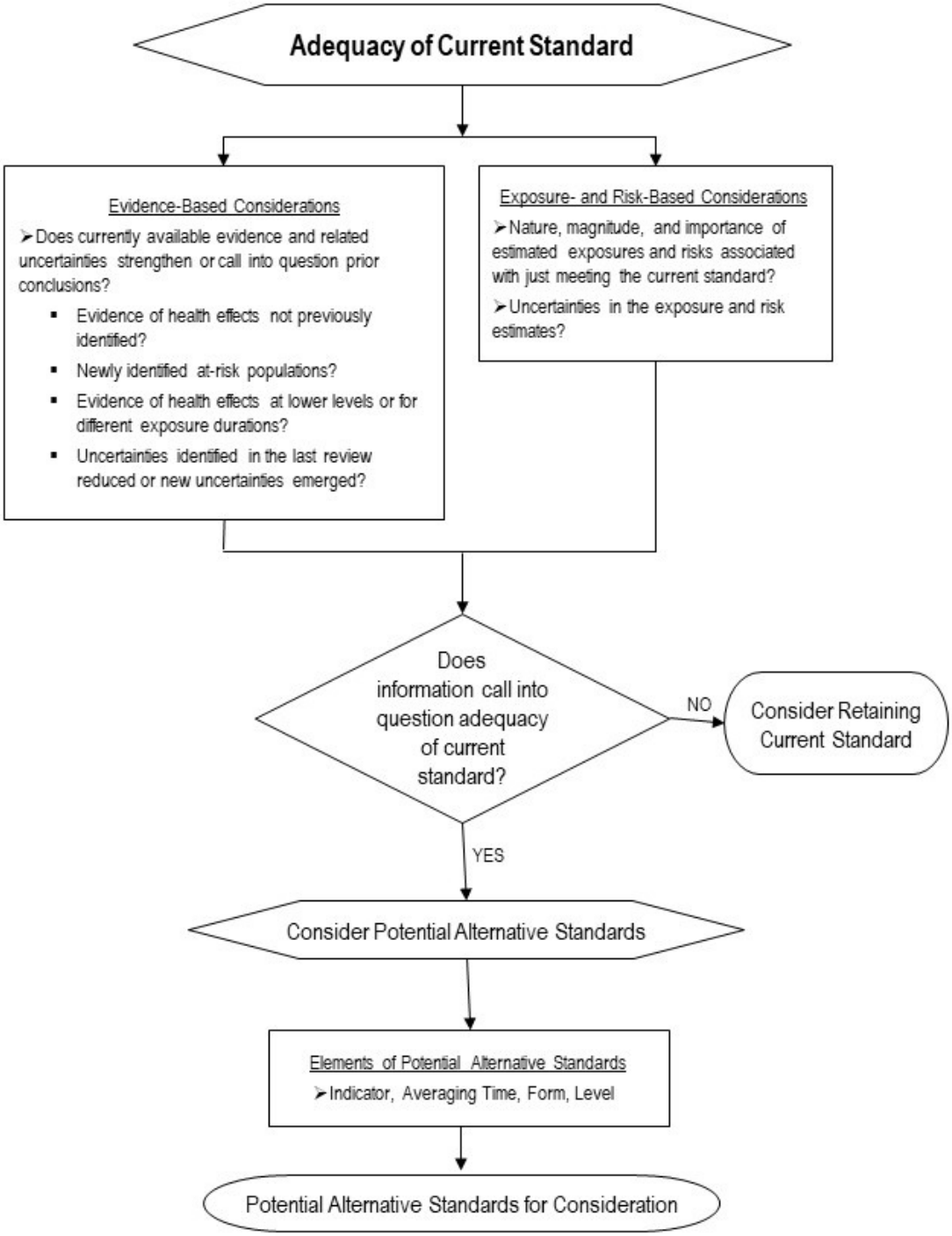

Figure 3-1. Overview of general approach for review of the primary O_3 standard.

The Agency's approach in reviewing primary standards is consistent with requirements of the provisions of the CAA related to the review of the NAAQS and with how the EPA and the

courts have historically interpreted the CAA. As discussed in section 1.1 above, these provisions require the Administrator to establish primary standards that, in the Administrator's judgment, are requisite (i.e., neither more nor less stringent than necessary) to protect public health with an adequate margin of safety. Consistent with the Agency's approach across all NAAQS reviews, the approach of the PA to informing these judgments is based on a recognition that the available health effects evidence generally reflects continuums that include ambient air exposures for which scientists generally agree that health effects are likely to occur through lower levels at which the likelihood and magnitude of response become increasingly uncertain. The CAA does not require the Administrator to establish a primary standard at a zero-risk level or at background concentration levels, but rather at a level that reduces risk sufficiently so as to protect public health, including the health of sensitive groups,[27] with an adequate margin of safety.

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

3.3 HEALTH EFFECTS EVIDENCE

3.3.1 Nature of Effects

The evidence base available in the current review includes decades of extensive evidence that clearly describes the role of O_3 in eliciting an array of respiratory effects and recent evidence suggests the potential for relationships between O_3 exposure and other effects. As was established in prior reviews, the most commonly observed effects, and those for which the evidence is strongest, are transient decrements in pulmonary function and respiratory symptoms, such as coughing and pain on deep inspiration, as a result of short-term exposures (ISA, section IS.4.3.1; 2013 ISA, p. 2-26). These effects are demonstrated in the large, long-standing evidence

[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.

base of controlled human exposure studies[28] (1978 AQCD, 1986 AQCD, 1996 AQCD, 2006 AQCD, 2013 ISA, ISA). The lung function effects are also positively associated with ambient air O_3 concentrations in epidemiologic panel studies, available in past reviews, that describe these associations for outdoor workers and children attending summer camps in the 1980s and 1990s (2013 ISA, section 6.2.1.2; ISA, Appendix 3, section 3.1.4.1.3). The epidemiologic evidence base additionally documents associations of O_3 concentrations in ambient air with more severe health outcomes, including asthma-related emergency department visits and hospital admissions (2013 ISA, section 6.2.7; ISA, Appendix 3, sections 3.1.5.1 and 3.1.5.2). Extensive animal toxicological evidence informs a detailed understanding of mechanisms underlying the respiratory effects of short-term exposures (ISA, Appendix 3, section 3.1.11), and studies in animal models also provide evidence for effects of longer-term O_3 exposure on the developing lung (ISA, Appendix 3, section 3.2.6).

- **Does the currently available scientific evidence alter our conclusions from the last review regarding the health effects attributable to exposure to O_3?**

The current evidence continues to support our prior conclusion that short-term O_3 exposure causes respiratory effects. Specifically, the full body of evidence continues to support the conclusions of a causal relationship of respiratory effects with short-term O_3 exposures and a likely causal relationship of respiratory effects with longer-term exposures (ISA, sections IS.4.3.1 and IS.4.3.2). The current evidence base, expanded by evidence newly available in this review, also indicates a likely causal relationship between short-term O_3 exposure and metabolic effects,[29] which were not evaluated as a separate category of effects in the last review when less evidence was available (ISA, section IS.4.3.3). The, newly available evidence is primarily from experimental animal research. For other types of health effects, new evidence has led to different conclusions from those reached in the prior review. Specifically, the current evidence, particularly in light of the additional controlled human exposure studies, is less consistent than

[28] The vast majority of the controlled human exposure studies (and all of the studies conducted at the lowest exposures) involved young healthy adults as study subjects. 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] 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).

what was previously available and less indicative of O_3-induced cardiovascular effects.[30] This new evidence has altered conclusions from the last review with regard to relationships between short-term O_3 exposures and cardiovascular effects and mortality, such that likely causal relationships are no longer concluded.[31] Thus, while conclusions have changed for some effects based on the new evidence, the conclusions reached in the last review on respiratory effects are supported by the current evidence, and conclusions are also newly reached for an additional category of health effects.

3.3.1.1 Respiratory Effects

As in the last review, the currently available evidence in this review supports the conclusion of a causal relationship between short-term O_3 exposure and respiratory effects (ISA, section IS.1.3.1). The strongest evidence for this comes from controlled human exposure studies, also available in the last review, demonstrating O_3-related respiratory effects in generally healthy adults.[32] Experimental studies in animals also document an array of respiratory effects resulting from short-term O_3 exposure and provide information related to underlying mechanisms (ISA, Appendix 3, section 3.1). The potential for O_3 exposure to elicit health outcomes more serious than those assessed in the experimental studies, particularly for children with asthma, continues to be indicated by the epidemiologic evidence of associations of O_3 concentrations in ambient air with increased incidence of hospital admissions and emergency department visits for an array of health outcomes, including asthma exacerbation, COPD exacerbation, respiratory infection, and combinations of respiratory diseases (ISA, Appendix 3, sections 3.1.5 and 3.1.6). The strongest such evidence is for asthma-related outcomes and specifically asthma-related outcomes for children, indicating an increased risk for people with asthma and particularly children with asthma (ISA, Appendix 3, section 3.1.5.7).

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[30] 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).

[31] The currently available 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 O_3 exposures (ISA, Table IS-1). The evidence is inadequate to infer the presence or absence of a causal relationship between long-term O_3 exposure and cancer (ISA, section IS4.3.6.6).

[32] The phrases "healthy adults" or "healthy subjects" are used to distinguish from subjects with asthma or other respiratory diseases, for which there are much fewer controlled human exposure studies. For studies of healthy subjects "the study design generally precludes inclusion of subjects with serious health conditions," such as individuals with severe respiratory diseases (2013 ISA, p. lx).

Respiratory responses observed in human subjects exposed to O_3 for periods of 8 hours or less, while intermittently exercising, include reduced lung function,[33] respiratory symptoms, increased airway responsiveness, mild bronchoconstriction (measured as a change in specific airway resistance [sRaw]), and pulmonary inflammation, with associated injury and oxidative stress (ISA, Appendix 3, section 3.1.4; 2013 ISA, sections 6.2.1 through 6.2.4). The available mechanistic evidence, discussed in greater detail in the ISA, describes pathways involving the respiratory and nervous systems by which O_3 results in pain-related respiratory symptoms and reflex inhibition of maximal inspiration (inhaling a full, deep breath), commonly quantified by decreases in forced vital capacity (FVC) and total lung capacity. This reflex inhibition of inspiration combined with mild bronchoconstriction contributes to the observed decrease in forced expiratory volume in one second (FEV_1), the most common metric used to assess O_3-related pulmonary function effects. The evidence also indicates that the additionally observed inflammatory response is correlated with mild airway obstruction, generally measured as an increase in sRaw (ISA, Appendix 3, section 3.1.3). As described in section 3.3.3 below, the prevalence and severity of respiratory effects in controlled human exposure studies, including symptoms (e.g., pain on deep inspiration, shortness of breath, and cough) increases, with increasing O_3 concentration, exposure duration, and ventilation rate of exposed subjects (ISA, Appendix 3, sections 3.1.4.1 and 3.1.4.2).

Within the evidence base from controlled human exposure studies, the majority of studies involve healthy adult subjects (generally 18 to 35 years old), although there are studies involving subjects with asthma, and a limited number of studies, generally of durations shorter than four hours, involving adolescents and adults older than 50 years. A summary of salient observations of O_3 effects on lung function, based on the controlled human exposure study evidence reviewed in the 1996 and 2006 AQCDs, and recognized in the 2013 ISA, continues to pertain to this evidence base as it exists today (ISA, Appendix 3, section 3.1.4.1.1, p. 3-11): "(1) young healthy adults exposed to ≥80 ppb O_3 develop significant reversible, transient decrements in pulmonary function and symptoms of breathing discomfort if minute ventilation ($\dot{V}_E$) or duration of exposure is increased sufficiently [i.e., as measured by FEV_1 and/or FVC]; (2) relative to young adults, children experience similar spirometric responses but lower incidence of symptoms from

[33] The measure of lung function response most commonly considered across O_3 NAAQS reviews is changes in FEV_1. In considering controlled human exposure studies, an O_3-induced change in FEV_1 is typically the difference between the decrement observed with O_3 exposure and what is generally an improvement observed with filtered air (FA) exposure. As explained in the 2013 ISA, "[n]oting that some healthy individuals experience small improvements while others have small decrements in FEV_1 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).

O_3 exposure; (3) relative to young adults, ozone-induced spirometric responses are decreased in older individuals; (4) there is a large degree of inter-subject variability in physiologic and symptomatic responses to O_3, but responses tend to be reproducible within a given individual over a period of several months; and (5) subjects exposed repeatedly to O_3 for several days experience an attenuation of spirometric and symptomatic responses on successive exposures, which is lost after about a week without exposure."

The evidence is most well established with regard to the effects, reversible with the cessation of exposure, that are associated with short-term exposures of several hours. For example, the evidence indicates a rapid recovery from O_3-induced lung function decrements (e.g., reduced FEV_1) and respiratory symptoms (2013 ISA, section 6.2.1.1). However, in some cases, such as after exposure to higher concentrations such as 300 ppb, the recovery phase may be slower and involve a longer time period (e.g., at least 24 hours [hrs]). Repeated daily exposure studies at such higher concentrations also have found FEV_1 response to be enhanced on the second day of exposure. This enhanced response is absent, however, with repeated exposure at lower concentrations, perhaps as a result of a more complete recovery or less damage to pulmonary tissues (2013 ISA, section pp. 6-13 to 6-14; Folinsbee et al., 1994).

As recognized in the last review, the persistence of other O_3-induced respiratory effects, and the potential for repeated occurrences to contribute to further effects can be an important consideration. For example, as described in the 2013 ISA, O_3-induced respiratory tract inflammation "can have several potential outcomes: (1) inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely; (2) continued acute inflammation can evolve into a chronic inflammatory state; (3) continued inflammation can alter the structure and function of other pulmonary tissue, leading to diseases such as fibrosis; (4) inflammation can alter the body's host defense response to inhaled microorganisms, particularly in potentially at-risk populations such as the very young and old; and (5) inflammation can alter the lung's response to other agents such as allergens or toxins" (2013 ISA, p. 6-76). With regard to O_3-induced increases in airway responsiveness, the controlled human exposure study evidence for healthy adults generally indicates a resolution within 18 to 24 hours after exposure (ISA, Appendix 3, section 3.1.4.3.1).

The extensive evidence base for O_3 health effects, compiled over several decades, continues to indicate respiratory responses to short exposures as the most sensitive effects of O_3. Such effects are well documented in controlled human exposure studies, most of which involve healthy adults. These studies have documented an array of respiratory effects, including reduced lung function, respiratory symptoms, increased airway responsiveness, and inflammation, in study subjects following 1- to 8-hour exposures, primarily while exercising. Such effects are of increased significance to people with asthma, particularly children, who are the age group most

likely to be outdoors at activity levels corresponding to those that have been associated with respiratory effects in the human exposure studies (as recognized below in sections 3.3.2 and 3.4). This increased significance is illustrated by the epidemiological findings of positive associations between O_3 exposure and asthma-related ED visits and hospital admissions for children with asthma. Thus, the evidence indicates O_3 exposure to increase the risk of asthma exacerbation, and associated outcomes, in children with asthma.

The 2013 ISA and past AQCDs have also concluded that the experimental animal evidence indicates the potential for O_3 exposures to increase susceptibility to infectious diseases through effects on defense mechanisms of the respiratory tract (2013 ISA, section 6.2.5). Evidence regarding respiratory infections and associated effects has been augmented by a number of epidemiologic studies reporting positive associations between short-term O_3 concentrations and emergency department visits for a variety of respiratory infection endpoints (ISA, Appendix 3, section 3.1.7.1).

Although the long-term exposure conditions that may contribute to further respiratory effects are less well understood, the conclusion based on the current evidence base remains that there is likely to be a causal relationship for such exposure conditions with respiratory effects (ISA, section IS.4.3.2). Most notably, experimental studies, including with nonhuman infant primates, have provided evidence relating O_3 exposure to allergic asthma-like effects, and epidemiologic cohort studies have reported associations of O_3 concentrations in ambient air with asthma development in children (ISA, Appendix 3, section 3.2.4.1.3 and 3.2.6). The biological plausibility of such a role for O_3 has been indicated by animal toxicological evidence on biological mechanisms (ISA, Appendix 3, sections 3.2.3 and 3.2.4.1.2). Specifically, the animal evidence, including the nonhuman primate studies of early life O_3 exposure, indicates that such exposures can cause "structural and functional changes that could potentially contribute to airway obstruction and increased airway responsiveness," which are hallmarks of asthma (ISA, Appendix 3, section 3.2.6, p. 3-113).

Overall, the respiratory effects evidence newly available in this review is generally consistent with the evidence base in the last review (ISA, Appendix 3, section 3.1.4). A few recent studies provide insights in previously unexamined areas, both with regard to human study groups and animal models for different effects, while other studies confirm and provide depth to prior findings with updated protocols and techniques (ISA, Appendix 3, sections 3.1.11 and 3.2.6). Thus, our current understanding of the respiratory effects of O_3 is similar to that in the last review.

One aspect of the evidence that has been augmented concerns pulmonary function in adults older than 50 years of age. Previously available evidence in this age group indicated smaller O_3-related decrements in middle-aged adults (35 to 60 years) than in adults 35 years of

age and younger (2006 AQCD, p. 6-23; 2013 ISA, p. 6-22; ISA, Appendix 3, section 3.1.4.1.1.2). A recent multicenter study of 55- to 70-year old subjects (average of 60 years), conducted for a 3-hour duration involving alternating 15-minute rest and exercise periods and a 120 ppb exposure concentration, reported a statistically significant O_3 FEV_1 response (ISA, Appendix 3, section 3.1.4.1.1.2; Arjomandi et al., 2018). While there is not a precisely comparable study in younger adults, the mean response for the 55- to 70-year olds, 1.2% O_3-related FEV_1 decrement, is lower than results for somewhat comparable exposures in adults aged 35 or younger, suggesting somewhat reduced responses to O_3 exposure in this older age group (ISA, Appendix 3, section 3.1.4.1.1.2; Arjomandi et al., 2018; Adams, 2000; Adams, 2006a).[34] Such a reduced response in middle-aged and older adults compared to young adults is consistent with conclusions in previous reviews (2013 ISA, section 6.2.1.1; 2006 AQCD, section 6.4).

The strongest evidence of O_3-related health effects, as was the case in the last review, continues to document the respiratory effects of O_3 (ISA, section ES.4.1). Among the newly available studies, there are several controlled human exposure studies that investigated lung function effects of higher exposure concentrations (e.g., 100 to 300 ppb) in healthy individuals younger than 35 years old, with findings generally consistent with previous studies (ISA, Appendix 3, section 3.4.1.1.2, p. 3-17). No studies are newly available of 6.6-hour controlled human exposures (with exercise) to concentrations below those previously studied.[35] The newly available animal toxicological studies augment the previously available information concerning mechanisms underlying the effects documented in experimental studies. Newly available epidemiologic studies of hospital admissions and emergency department visits for a variety of respiratory outcomes supplement the previously available evidence with additional findings of consistent associations with O_3 concentrations across a number of study locations (ISA, Appendix 3, sections 3.1.4.1.3, 3.1.5, 3.1.6.1.1, 3.1.7.1 and 3.1.8). These studies include a number that report positive associations for asthma-related outcomes, as well as a few for COPD-related outcomes. Together these studies in the current epidemiologic evidence base

[34] For the same exposure concentration of 120 ppb, Adams (2006a) observed an average 3.2%, statistically significant, O_3-related FEV_1 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-m^2 during the exercise periods (versus 15 to 17 L/min-m^2 in Arjomandi et al., 2018]). For the same concentration with a lower EVR during exercise (17 L/min-m^2), although with more exercise, Adams (2000) observed a 4%, statistically significant, O_3-related FEV_1 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).

[35] As recognized in section 3.3.1.1 above, there is 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 FEV_1 decrement in this age group of subjects (ISA, Appendix 3, section 3.1.4.1.1.2).

continue to indicate the potential for O_3 exposures to contribute to such serious health outcomes, particularly for people with asthma.

3.3.1.2 Other Effects

As was the case for the evidence available in the last review, the currently available evidence for health effects other than those on the respiratory system is more uncertain than that for respiratory effects. For some of these other categories of effects, the evidence now available has contributed to changes in conclusions reached in the last review. For example, cardiovascular effects and mortality are no longer concluded to be likely causally related to O_3 exposures based on newly available evidence in combination with the uncertainties recognized for the evidence available in the last review. Additionally, newly available evidence has also led to conclusions for another category, metabolic effects, for which formal causal determinations were previously not articulated.

The ISA finds the evidence for metabolic effects sufficient to conclude that there is likely to be a causal relationship with short-term O_3 exposures (ISA, section IS.4.3.3). The evidence of metabolic effects of O_3 comes primarily from experimental animal study findings that short-term O_3 exposure can impair glucose tolerance, increase triglyceride levels and elicit fasting hyperglycemia and increase hepatic gluconeogenesis (ISA, Appendix 5, section 5.1.8 and Table 5-3). The exposure conditions from these studies generally involve much higher O_3 concentrations than those commonly occurring in areas of the U.S. where the current standard is met. For example, the animal studies include 4-hour concentrations of 400 to 800 ppb (ISA, Appendix 5, Tables 5-8 and 5-10). In addition, an epidemiologic study available in the last review has reported positive associations of multiday average O_3 concentrations in ambient air with changes in two indicators of glucose and insulin homeostasis (ISA, Appendix 5, sections 5.1.3.1.1 and 5.1.8).

The ISA additionally concludes that the evidence is suggestive of, but not sufficient to infer, a causal relationship between long-term O_3 exposures and metabolic effects (ISA, section IS.4.3.6.2). As with metabolic effects and short-term O_3, the primary evidence is from experimental animal studies in which the exposure concentrations are appreciably higher than those commonly occurring in the U.S. For example, the animal studies include exposures over several weeks to concentrations of 250 ppb and higher (ISA, Appendix 5, section 5.2.3.1.1). The somewhat limited epidemiologic evidence related to long-term O_3 concentrations and metabolic effects includes several studies reporting increased odds of being overweight or obese or having metabolic syndrome and increased hazard ratios for diabetes incidence with increased O_3 concentrations (ISA, Appendix 5, sections 5.2.3.4.1, 5.2.5 and 5.2.9, Tables 5-12 and 5-15).

With regard to cardiovascular effects and total (nonaccidental) mortality and short-term O_3 exposures, the conclusions regarding the potential for a causal relationship have changed from what they were in the last review after integrating the previously available evidence with newly available evidence. The relationships are now characterized as suggestive of, but not sufficient to infer, a causal relationship (ISA, Appendix 4, section 4.1.17; Appendix 6, section 6.1.8). This reflects several aspects of the current evidence base: (1) a now-larger body of controlled human exposure studies providing evidence that is not consistent with a cardiovascular effect in response to short-term O_3 exposure; (2) a paucity of epidemiologic evidence indicating more severe cardiovascular morbidity endpoints,[36] that would be expected if the impaired vascular and cardiac function (observed in animal toxicological studies) was the underlying basis for cardiovascular mortality (for which epidemiologic studies have reported some positive associations with O_3); and (3) the remaining uncertainties and limitations recognized in the 2013 ISA (e.g., lack of control for potential confounding by copollutants in epidemiologic studies) that still remain. Although there exists consistent or generally consistent evidence for a limited number of O_3-induced cardiovascular endpoints in animal toxicological studies and cardiovascular mortality in epidemiologic studies, there is a general lack of coherence between these results and findings in controlled human exposure and epidemiologic studies of cardiovascular health outcomes (ISA, section IS.1.3.1). Related to this updated conclusion based on the current evidence for cardiovascular effects, the evidence for short-term O_3 and mortality is also updated (ISA, Appendix 6, section 6.1.8). While there remain consistent, positive associations between short-term O_3 and total (nonaccidental), respiratory, and cardiovascular mortality (and there are some studies reporting associations to remain after controlling for PM_{10} and NO_2), the full evidence base does not describe a continuum of effects that could lead to cardiovascular mortality.[37] Therefore, because cardiovascular mortality is the largest contributor to total mortality, the relatively limited biological plausibility and coherence within and across disciplines for cardiovascular effects (including mortality) contributes to an accompanying change in the causality determination for total mortality (ISA, section IS.4.3.5). Thus, the currently available evidence for cardiovascular effects and total mortality is concluded to be suggestive of, but not sufficient to infer, a causal relationship with short-term (as well as long-term) O_3 exposures (ISA, section IS.1.3.1).

[36] 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).

[37] Due to findings from controlled human exposure studies examining clinical endpoints (e.g., blood pressure) that do not indicate an O_3 effect and from epidemiologic studies examining cardiovascular-related hospital admissions and ED 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).

For other health effect categories, conclusions in this review are largely unchanged from those in the last review. The available evidence for reproductive effects, as well as for effects on the nervous system, is also concluded to be suggestive of, but not sufficient to infer, a causal relationship (as was the case in the last review) (ISA, section IS.4.3.6). Additionally, the evidence is inadequate to determine if a causal relationship exists between O_3 exposure and cancer (ISA, section IS.4.3.6.6).

3.3.2 Public Health Implications and At-risk Populations

The public health implications of the evidence regarding O_3-related health effects, as for other effects, are dependent on the type and severity of the effects, as well as the size of the population affected. Such factors are discussed here in the context of our consideration of the health effects evidence related to O_3 in ambient air. Additionally, we summarize the currently available information related to judgments or interpretative statements developed by public health experts, including particularly experts in respiratory health. This section also summarizes the current information on population groups at increased risk of the effects of O_3 in ambient air.

With regard to O_3 in ambient air, the potential public health impacts relate most importantly to the role of O_3 in eliciting respiratory effects, the category of effects that the ISA concludes to be causally related to O_3 exposure. Controlled human exposure studies have documented reduced lung function, respiratory symptoms, increased airway responsiveness, and inflammation, among other effects, in healthy adults exposed while at elevated ventilation, such as while exercising. Such effects, if of sufficient severity and in individuals with compromised respiratory function, such as individuals with asthma, are plausibly related to emergency department visits and hospital admissions for asthma which have been associated with ambient air concentrations of O_3 in epidemiologic studies (as summarized in section 3.3.1 above; 2013 ISA, section 6.2.7; ISA, Appendix 3, sections 3.1.5.1 and 3.1.5.2).

The clinical significance of individual responses to O_3 exposure depends on the health status of the individual, the magnitude of the changes in pulmonary function, the severity of respiratory symptoms, and the duration of the response. With regard to pulmonary function, the greater impact of larger decrements on affected individuals can be described. For example, moderate effects on pulmonary function, such as transient FEV_1 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 for most healthy individuals, while larger effects on pulmonary function (e.g., FEV_1 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).

In addition to the difference in severity or magnitude of specific effects in healthy people, it is also important to consider aspects of the same effect with regard to its impact on population groups that may have a pre-existing disease status. For example, the same reduction in FEV_1 or increase in inflammation or airway responsiveness in a healthy group and a group with asthma may increase the risk of a more severe effect in the group with asthma, such as asthma exacerbation triggered by an allergen to which they may be sensitized (Cox, 2020, Responses to Charge Questions, pp. 7-8). Duration and frequency of documented effects is also reasonably expected to influence potential adversity and interference with normal activity. In summary, consideration of differences and also the relative transience or persistence of such FEV_1 changes and respiratory symptoms, as well as pre-existing sensitivity to effects on the respiratory system, and other factors, are important to characterizing implications for public health effects of an air pollutant such as O_3 (ATS, 2000; Thurston et al., 2017).

Decisions made in past reviews of the O_3 primary standard and associated judgments regarding adversity or health significance of measurable physiological responses to air pollutants have been informed by guidance, criteria or interpretative statements developed within the public health community, including the ATS, an organization of respiratory disease specialists, as well as the CASAC. The ATS released its initial statement (titled *Guidelines as to What Constitutes an Adverse Respiratory Health Effect, with Special Reference to Epidemiologic Studies of Air Pollution*) in 1985 and updated it in 2000 (ATS, 1985; ATS, 2000). The ATS described its 2000 statement, considered in the last review of the O_3 standard, as being intended to "provide guidance to policy makers and others who interpret the scientific evidence on the health effects of air pollution for the purposes of risk management" (ATS, 2000). The ATS described the statement as not offering "strict rules or numerical criteria," but rather proposing "principles to be used in weighing the evidence and setting boundaries," and stated that "the placement of dividing lines should be a societal judgment" (ATS, 2000). Similarly, the most recent policy statement by the ATS, which once again broadens its discussion of effects, responses and biomarkers to reflect the expansion of scientific research in these areas, reiterates that concept, conveying that it does not offer "strict rules or numerical criteria, but rather proposes considerations to be weighed in setting boundaries between adverse and nonadverse health effects," providing a general framework for interpreting evidence that proposes a "set of considerations that can be applied in forming judgments" for this context (Thurston et al., 2017).

With regard to pulmonary function decrements, the earlier ATS statement concluded that "small transient changes in forced expiratory volume in 1 s[econd] (FEV_1) alone were not necessarily adverse in healthy individuals, but should be considered adverse when accompanied by symptoms" (ATS, 2000). The more recent ATS statement continues to support this conclusion and also gives weight to findings of such lung function changes in the absence of

respiratory symptoms in individuals with pre-existing compromised function, such as that resulting from asthma (Thurston et al., 2017). More specifically, the recent ATS statement expresses the view that the occurrence of "small lung function changes" in individuals with pre-existing compromised function, such as asthma, "should be considered adverse … even without accompanying respiratory symptoms" (Thurston et al., 2017). In keeping with the intent of these statements to avoid specific criteria, neither statement provides more specific descriptions of such responses, such as with regard to magnitude, duration or frequency, for consideration of such conclusions. The earlier ATS statement, in addition to emphasizing clinically relevant effects, also emphasized both the need to consider changes in "the risk profile of the exposed population," and effects on the portion of the population that may have a diminished reserve that puts its members at potentially increased risk if affected by another agent (ATS, 2000). These concepts, including the consideration of the magnitude of effects occurring in just a subset of study subjects, continue to be recognized as important in the more recent ATS statement (Thurston et al., 2017) and continue to be relevant to the evidence base for O_3.

- **Does the current evidence alter our understanding of populations that are particularly at risk from O_3 exposures? What are important uncertainties in that evidence?**

The newly available information has not altered our understanding of human populations at particular risk of health effects from O_3 exposures (ISA, section IS.4.4). For example, as recognized in prior reviews, people with asthma are the key population at risk of O_3-related effects. The respiratory effects evidence, extending decades into the past and augmented by new studies in this review, supports this conclusion (ISA, sections IS.4.3.1). For example, numerous epidemiological studies document associations with O_3 with asthma-related health outcomes (e.g., emergency department visits and hospital admissions). Such studies indicate the associations to be strongest for populations of children which is consistent with their generally greater time outdoors while at elevated exertion. Together, these considerations indicate people with asthma, including particularly children with asthma, to be at relatively greater risk of O_3-related effects than other members of the general population (ISA, sections IS.4.3.1 and IS.4.4.2, Appendix 3).[38]

With respect to people with asthma, the limited evidence from controlled human exposure studies (which are primarily in adult subjects) indicates similar magnitude of FEV_1 decrements as in people without asthma (ISA, Appendix 3, section 3.1.5.4.1). Across other respiratory effects of O_3 (e.g., increased respiratory symptoms, increased airway responsiveness

[38] Populations or lifestages can be at increased risk of an air pollutant-related health effect due to one or more of a number of 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.

and increased lung inflammation), the evidence has also found the observed responses to generally not differ due to the presence of asthma, although the evidence base is more limited with regard to study subjects with asthma (ISA, Appendix 3, section 3.1.5.7). Further, the features of asthma contribute to a risk of asthma-related responses, such as asthma exacerbation in response to asthma triggers, which may increase the risk of more severe health outcomes (ISA, section 3.1.5). For example, a particularly strong and consistent component of the epidemiologic evidence is the appreciable number of epidemiologic studies that demonstrate associations between ambient O_3 concentrations and hospital admissions and emergency department visits for asthma (ISA, section IS.4.4.3.1).[39] We additionally recognize that in these studies, the strongest associations (e.g., highest effect estimates) or associations more likely to be statistically significant are those for childhood age groups, which are recognized in section 3.4 as age groups most likely to spend time outdoors during afternoon periods (when O_3 may be highest) and at activity levels corresponding to those that have been associated with respiratory effects in the human exposure studies (ISA, Appendix 3, sections 3.1.4.1 and 3.1.4.2).[40] The epidemiologic studies of hospital admissions and emergency department visits are augmented by a large body of individual-level epidemiologic panel studies that demonstrated associations of short-term ozone concentrations with respiratory symptoms in children with asthma. Additional support comes from epidemiologic studies that observed ozone-associated increases in indicators of airway inflammation and oxidative stress in children with asthma (ISA, section IS.4.3.1). Together, this evidence continues to indicate the increased risk of population groups with asthma (ISA, Appendix 3, section 3.1.5.7).

Children, and also outdoor adult workers, are at increased risk largely due to their generally greater time spent outdoors while at elevated exertion rates (including in the summer when O_3 levels may be higher). This status makes them more likely to be exposed to O_3 in ambient air, under conditions contributing to increased dose due to greater air volumes taken into the lungs (ISA, section IS.4.4.2; 2013 ISA, section 5.2.2.7). Thus, in light of the evidence summarized in the prior paragraph, children and outdoor workers with asthma may be at

[39] In addition to asthma exacerbation, the epidemiologic evidence also includes findings of positive associations of increased O_3 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).

[40] There is limited data on activity patterns by health status. An analysis in the 2014 HREA indicated that asthma status had little to no impact on the percent of people participating in outdoor activities during afternoon hours, the amount of time spent, and whether they performed activities at elevated exertion levels (2014 HREA, section 5.4.1.5). Based on an updated evaluation of recent activity pattern data we found children, on average, spend approximately 2¼ hours of afternoon time outdoors, 80% of which is at a moderate or greater exertion level, regardless of their asthma status (see Appendix 3D, section 3D.2.5.3). Adults 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.

increased risk of more severe outcomes, such as asthma exacerbation. Further, with regard to children, there is experimental evidence from early life exposures of nonhuman primates that indicates the potential for effects in childhood (through adolescence) when human respiratory systems are under development (ISA, sections IS.4.4.2 and IS.4.4.4.1). As noted in the ISA, "these experimental studies indicate that early-life ozone exposure can cause structural and functional changes that could potentially contribute to airway obstruction and increased airway responsiveness" (ISA, p. IS-52). Overall, the evidence available in the current review continues to indicate the increased susceptibility of these population groups.

The ISA in the last review additionally identified older adults as being at greater risk of O_3-related health effects. That identification, however, was based largely on studies of short-term O_3 exposure and mortality, which are part of the larger evidence base that is now concluded to be suggestive, but not sufficient to infer a causal relationship (ISA, sections IS.4.3.5 and IS.4.4.4.2, Appendix 4, sections 4.1.16.1 and 4.1.17).[41] Other evidence available in the current review adds little to the evidence available at the time of the last review for consideration of susceptibility of older adults (ISA, section IS.4.4.2).

The ISA in the last review concluded that the information available at the time for low socioeconomic status (SES) as a factor associated with the risk of O_3-related health effects, provided suggestive evidence of potentially increased risk (2013 ISA, section 8.3.3 and p. 8-37). The 2013 ISA concluded that "[o]verall, evidence is suggestive of SES as a factor affecting risk of O_3-related health outcomes based on collective evidence from epidemiologic studies of respiratory hospital admissions but inconsistency among epidemiologic studies of mortality and reproductive outcomes," additionally stating that "[f]urther studies are needed to confirm this relationship, especially in populations within the U.S." (2013 ISA, p. 8-28). The evidence available in the current review adds little to the evidence available at the time of the last review in this area (ISA, section IS.4.4.2 and Table IS-10). The ISA in the last review additionally identified a role for dietary anti-oxidants such as vitamins C and E in influencing risk of O_3-related effects, such as inflammation, as well as a role for genetic factors to also confer either an increased or decreased risk (2013 ISA, sections 8.1 and 8.4.1). No newly available evidence has been evaluated that would inform or change these prior conclusions (ISA, section IS.4.4 and Table IS-10).

[41] As noted in the ISA, "[t]he majority of evidence for older adults being at increased risk of health effects related to ozone exposure comes from studies of short-term ozone exposure and mortality evaluated in the 2013 Ozone ISA" (ISA, p. IS-52).

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

The magnitude and characterization of a public health impact is dependent upon the size and characteristics of the populations affected, as well as the type or severity of the effects. As summarized above, a key population most at risk of health effects associated with O_3 in ambient air is people with asthma. The National Center for Health Statistics data for 2017 indicate that approximately 7.9% of the U.S. populations has asthma (Table 3-1; CDC, 2019). This is one of the principal populations that the primary O_3 NAAQS is designed to protect (80 FR 65294, October 26, 2015). Table 3-1 below considers the currently available information that helps to characterize key features of this population.

The age group for which the prevalence documented by these data is greatest is children aged five to 19, with 9.7% of children aged five to 14 and 9.4% of children aged 15-19 having asthma. In 2012 (the most recent year for which such an evaluation is available), asthma was the leading chronic illness affecting children (Bloom et al., 2013). The prevalence is greater for boys than girls (for those less than 18 years of age). Among populations of different races or ethnicities, black non-Hispanic children aged five to 14 have the highest prevalence, at 16.1%. Asthma prevalence is also increased among populations in poverty. For example, 11.7% of people living in households below the poverty level have asthma compared to 7.3%, on average, of those living above it. Populations groups with relatively greater asthma prevalence might be expected to have a relatively greater potential for O_3-related health impacts.[42]

[42] 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.

Table 3-1. National prevalence of asthma, 2017.

Characteristic [A]	Number with Current Asthma (in thousands) [B]	Percent with Current Asthma
Total	25,191	7.9
Child (Age <18)	6,182	8.4
Adult (Age 18+)	19,009	7.7
All Age Groups		
0-4 years	869	4.4
5-14 years	4,010	9.7
15-19 years	2,020	9.4
20-24 years	1,498	7.3
25-34 years	3,311	7.6
35-64 years	10,036	8.1
65+ years	3,447	7.0
Child Age Group		
0-4 years	869	4.4
5-11 years	2,548	8.8
12-17 years	2,765	11.1
12-14 years	1,462	11.8
15-17 years	1,304	10.4
Sex		
Males	10,035	6.4
Boys (Age <18)	3,569	9.5
Boys (Age 5-14)	2,165	10.3
Men (Age 18+)	6,466	5.4
Females	15,156	9.3
Girls (Age <18)	2,613	7.3
Girls (Age 5-14)	1,845	9.1
Women (Age 18+)	12,544	9.8
Race/Ethnicity		
White NH [C]	15,718	8.1
Child (Age <18)	2,918	7.7
Child (Age 5-14)	1,841	8.8
Adult (Age 18+)	12,800	8.1
Black NH	3,910	10.1
Child (Age <18)	1,231	12.6
Child (Age 5-14)	895	16.1
Adult (Age 18+)	2,679	9.2
Other NH	1,871	6.7
Child (Age <18)	617	8.2
Child (Age 5-14)	403	9.4
Adult (Age 18+)	1,254	6.1
Hispanic, all	3,692	6.4
Child (Age <18)	1,416	7.7
Child (Age 5-14)	871	8.4
Adult (Age 18+)	2,276	5.8

Characteristic [A]	Number with Current Asthma (in thousands) [B]	Percent with Current Asthma
Hispanic, Puerto Rican	695	12.8
Child (Age<18)	195*	11.3
Child (Age 5-14)	93*	10.1*
Adult (Age 18+)	499	13.5
Hispanic, Mexican/Mexican-American	1,822	5.1
Child (Age<18)	753	6.2
Child (Age 5-14)	481	7.0
Adult (Age 18+)	1,069	4.5
Federal Poverty Threshold		
Below 100% of poverty level	5,020	11.7
100% to less than 250% of poverty level	6,769	7.9
250% to less than 450% of poverty level	6,061	7.3
450% of poverty level or higher	7,342	6.8

[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
* Relative standard error of the estimate is 30% - 50%; the estimate is unreliable.
Adapted from 2017 National Health Interview Survey, Tables 3-1 (*https://www.cdc.gov/asthma/nhis/2017/table3-1.htm*) and 4-1 (*https://www.cdc.gov/asthma/nhis/2017/table4-1.htm*).

Children under the age of 18 account for 16.7% of the total U.S. population, with 6.2% of the total population being children under 5 years of age (U.S. Census Bureau, 2019). Based on a prior analysis of data from the Consolidated Human Activity Database (CHAD)[43] in the 2014 HREA, children ages 4-18 years old were found to more frequently spend time outdoors compared to other age groups (e.g., adults aged 19-34) spending more than 2 hours outdoors, particularly during the afternoon and early evening (e.g., 12:00 p.m. through 8:00 p.m.) (2014 HREA, section 5G-1.2). These results were confirmed by additional analyses of CHAD data reported in the ISA, noting greater participation in afternoon outdoor events for children ages 6-19 years old during the warm season months compared to other times of the day (ISA, Appendix 2, section 2.4.1, Table 2-1). The 2014 HREA also found that children ages 4-18 years old spent 79% of their outdoor time at moderate or greater exertion (2014 HREA, section 5G-1.4). Further analyses performed for this review using the most recent version of CHAD generated similar results, as described in section 3.4.5 below (Appendix 3D, section 3D.2.5.3 and Figure 3D-9). Each of these analyses indicate children participate more frequently and spend more afternoon

[43] The CHAD provides time series data on human activities through a database system of collected human diaries, or daily time location activity logs.

time outdoors than all other age groups while at elevated exertion, and consistently do so when considering the most important influential factors such as day-of-week and outdoor temperature. Given that afternoon time outdoors and elevated exertion were determined most important in understanding the fraction of the population that might experience O_3 exposures of concern (2014 HREA, section 5.4.2), they may be at greater risk of effects due to increased exposure to O_3 in ambient air.

About one third of workers were required to perform outdoor work in 2018 (Bureau of Labor Statistics, 2019). Jobs in construction and extraction occupations and protective service occupations required more than 90% of workers to spend at least part of their workday outdoors (Bureau of Labor Statistics, 2017). Other employment sectors, including installation, maintenance and repair occupations and building and grounds cleaning and maintenance operations, also had a high percentage of employees who spent part of their workday outdoors (Bureau of Labor Statistics, 2017). These occupations often include physically demanding tasks and involve increased ventilation rates which when combined with exposure to O_3, may increase the risk of health effects.

3.3.3 Exposure Concentrations Associated with Effects

As at the time of the last review, the EPA's conclusions regarding exposure concentrations of O_3 associated with respiratory effects reflect the extensive longstanding evidence base of controlled human exposure studies of short-term O_3 exposures of people with and without asthma.[44] These studies have documented an array of respiratory effects, including reduced lung function, respiratory symptoms, increased airway responsiveness, and inflammation, in study subjects following 1- to 8-hour exposures, primarily while exercising. The severity of observed responses, the percentage of individuals responding, and strength of statistical significance at the study group level have been found to increase with increasing exposure (ISA; 2013 ISA; 2006 AQCD). Factors influencing exposure include activity level or ventilation rate, exposure concentration, and exposure duration (ISA; 2013 ISA; 2006 AQCD). For example, evidence from studies with similar duration and exercise aspects (6.6-hour duration with six 50-minute exercise periods) demonstrates an exposure-response relationship for O_3-

[44] As recognized elsewhere, the studies are largely conducted with adult subjects.

induced reduction in lung function (Figure 3-2).[45] [46] This specific evidence was integral to the Administrator's judgments and decision in the last review (80 FR 65292, October 26, 2015).

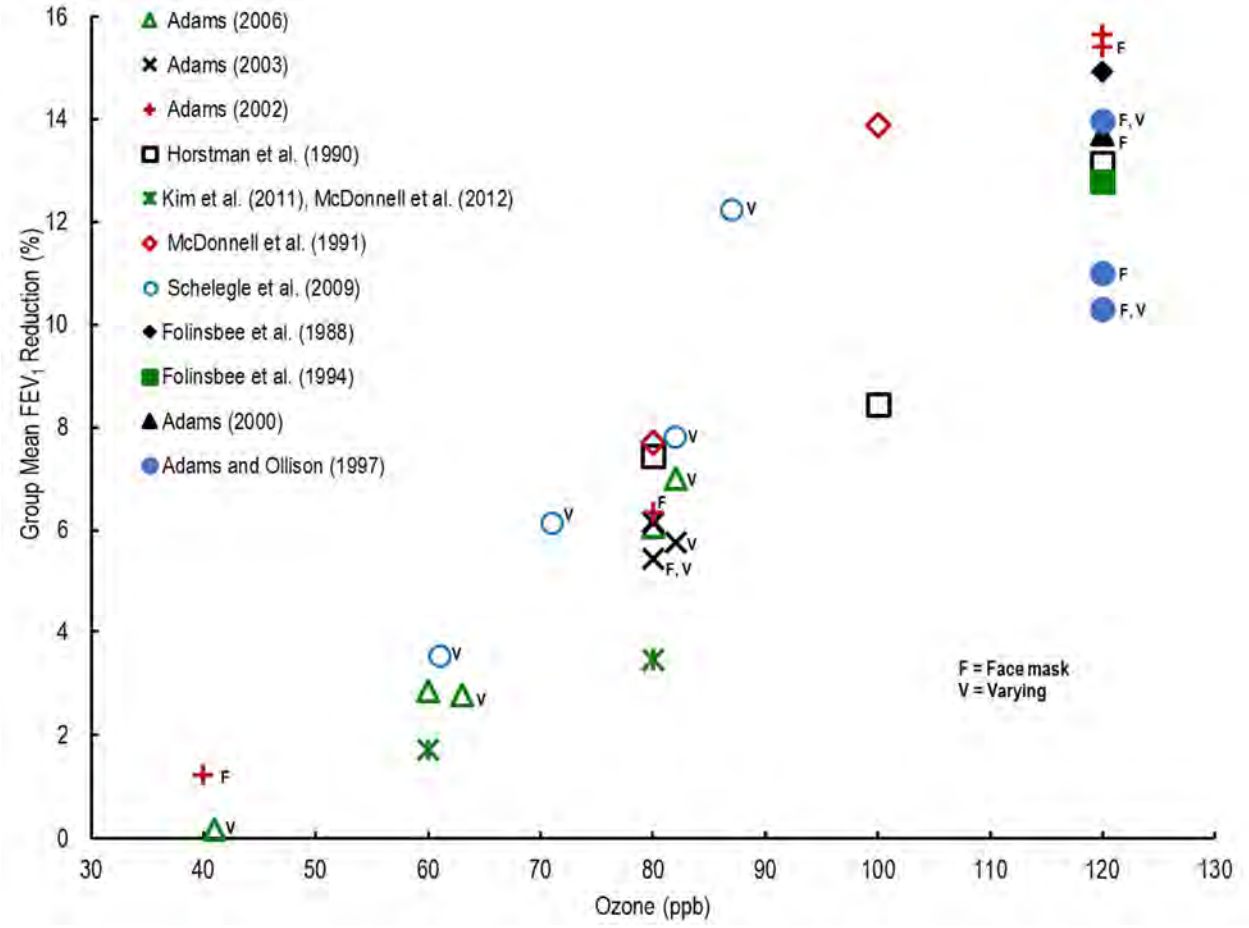

Figure 3-2. Group mean O_3-induced reduction in FEV1 from controlled human exposure studies of healthy adults exposed for 6.6 hours with quasi-continuous exercise. FEV1 values plotted reflect group mean O_3-induced percent change in FEV1, based on subtraction of the group mean filtered air percent change (post-pre exposure) from the group mean O_3 percent change in FEV1 (adapted from Appendix 3A; ISA, Appendix 3, Figure 3-1). Concentrations are the time-weighted averages of target concentrations across full 6.6-hour period in chamber studies (or across the six exposures in face mask studies).

[45] For a subset of the studies included in Figure 3-2 (those with face mask rather than chamber exposures), there is no O_3 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).

[46] The relationship also exists for size of FEV_1 decrement with alternative exposure or dose metrics, including total inhaled O_3 and intake volume averaged concentration.

- **Does the current evidence alter our conclusions from the previous review regarding the exposure duration and concentrations associated with health effects? Does the currently available scientific evidence indicate health effects attributable to exposures to O_3 concentrations lower than previously reported?**

The current evidence, including that newly available in this review, does not alter our conclusions from the last review on exposure duration and concentrations associated with O_3-related health effects. These conclusions were largely based on the body of evidence from the controlled human exposure studies. A limited number of controlled human exposure studies are newly available in the current review, with none involving lower exposure concentrations than those previously studied (e.g., Figure 3-2) or finding effects not previously reported (ISA, Appendix 3, section 3.1.4).[47]

The extensive evidence base for O_3 health effects, compiled over several decades, continues to indicate respiratory responses to short-term exposures as the most sensitive effects of O_3. As summarized in section 3.3.1.1 above, an array of respiratory effects is well documented in controlled human exposure studies of subjects exposed for 1 to 8 hours, primarily while exercising. The risk of more severe health outcomes associated with such effects is increased in people with asthma as illustrated by the epidemiological findings of positive associations between O_3 exposure and asthma-related ED visits and hospital admissions.

The magnitude of respiratory response (e.g., size of lung function reductions and magnitude of symptom scores) documented in the controlled human exposure studies is influenced by ventilation rate, exposure duration, and exposure concentration. When performing physical activities requiring elevated exertion, ventilation rate is increased, leading to greater potential for health effects due to an increased internal dose (2013 ISA, section 6.2.1.1, pp. 6-5 to 6-11). Accordingly, the exposure concentrations eliciting a given level of response after a given exposure duration is lower for subjects exposed while at elevated ventilation, such as while exercising (2013 ISA, pp. 6-5 to 6-6). For example, in studies of generally healthy young adults exposed while at rest for 2 hours, 500 ppb is the lowest concentration eliciting a statistically significant O_3-induced group mean lung function decrement, while a 1- to 2-hour exposure to 120 ppb 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, pp. 6-5 to 6-6).

The exposure conditions (e.g., duration and exercise) given primary focus in the past several reviews are those of the 6.6-hour study design, which involves six 50-minute exercise periods during which subjects maintain a moderate level of exertion to achieve a ventilation rate

[47] 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).

of approximately 20 L/min per m^2 body surface area while exercising. The 6.6 hours of exposure in these studies has generally occurred in an enclosed chamber and the study design includes three hours in each of which is a 50-minute exercise period and a 10-minute rest period, followed by a 35-minute lunch (rest) period, which is followed by three more hours of exercise and rest, as before lunch.[48] Most of these studies performed to date involve exposure maintained at a constant (unchanging) concentration for the full duration, although a subset of studies have concentrations that vary (generally in a stepwise manner) across the exposure period and are selected so as to achieve a specific target concentration as the exposure average.[49] No studies of the 6.6-hour design are newly available in this review. The previously available studies of this design document statistically significant O_3-induced reduction in lung function (FEV_1) and increased pulmonary inflammation in young healthy adults exposed to O_3 concentrations as low as 60 ppb. Statistically significant group mean changes in FEV_1, also often accompanied by statistically significant increases in respiratory symptoms, become more consistent across such studies of exposures to higher O_3 concentrations, such as 70 ppb and 80 ppb (Appendix 3A, Table 3A-1). The lowest exposures concentration for which these studies document a statistically significant increase in respiratory symptoms is somewhat above 70 ppb (Schelegle et al., 2009).[50]

In the 6.6-hour studies, the group means of O_3-induced[51] FEV_1 reductions for exposure concentrations below 80 ppb are at or below 6% (Figure 3-2, Table 3-2). For example, the group means of O_3-induced FEV_1 decrements that have been found to be statistically significantly different from the responses in filtered air are 6.1% for 70 ppb and 1.7% to 3.5% for 60 ppb (Figure 3-2, Table 3-2). The group mean O_3-induced FEV_1 decrements generally increase with increasing O_3 exposures, reflecting increases in both the number of the individuals affected and

[48] 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 O_3 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).

[49] 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 (2006), 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.

[50] 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.

[51] Consistent with the ISA and 2013 ISA, the phrase "O_3-induced" decrement or reduction in lung function or FEV_1 refers to the percent change from pre-exposure measurement of the O_3 exposure minus the percent change from pre-exposure measurement of the filtered air exposure (2013 ISA, p. 6-4).

the magnitude of the FEV_1 reduction (Figure 3-2, Table 3-2). For example, following 6.6-hour exposures to a lower concentration (40 ppb), for which decrements were not statistically significant at the group mean level, none of 60 subjects across two separate studies experienced an O_3-induced FEV_1 reduction as large as 15% or more (Table 3-2; Appendix 3D, Table 3D-19). Across the four experiments (with number of subjects ranging from 30 to 59 subjects) that have reported results for 60 ppb target exposure, the number of subjects experiencing this magnitude of FEV_1 reduction (at or above 15%) varied (zero of 30, one of 59, two of 31 and two of 30 exposed subjects). The response increases to three of 31 subjects for the study with a 70 ppb target concentration (Appendix 3D, Table 3D-19; Schelegle et al., 2009). In addition to illustrating the E-R relationship, these findings also illustrate the considerable variability in magnitude of responses observed among study subjects (Table 3-2 and Figure 3-2; ISA, Appendix 3, section 3.1.4.1.1; 2013 ISA, p. 6-13).

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]	See Appendix 3A [D]
	100 ppb	Yes	-8.5% to -13.9% [C]	
	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 [I]
	60 ppb	Yes [G]	-2.9% -2.8%	Adams, 2006b; Brown et al., 2008
		Yes	-1.7%	Kim et al., 2011
		No	-3.5%	Schelegle et al., 2009
	40 ppb	No	-1.2%	Adams, 2002
		No	-0.2%	Adams, 2006b
Increased Respiratory Symptoms	120 ppb	Yes	Increased symptom scores	See Appendix 3A
	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 [H]	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 is 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.
[B] Statistical significance based on the O_3 compared to filtered air response at the study group mean (rounded here to decimal).
[C] Ranges reflect the minimum to maximum FEV_1 decrements across multiple exposure designs and studies. Study-specific values and exposure details provided in Appendix 3A, Tables 3A-1 and 3A-2, respectively.
[D] Citations for FEV_1 and respiratory symptoms findings for exposures above 80 ppb are in Appendix 3A and not repeated here.
[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 (2006b) reported FEV_1 data for 60 ppb exposure by both constant and varying concentration designs. Subsequent analysis of the FEV_1 data from the former found the group mean O_3 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] 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).

For shorter exposure periods, ranging from one to two hours, higher exposure concentrations, ranging from 80 ppb to 400 ppb, have been studied (Appendix 3A, Table 3A-3). In these studies, some exposure protocols have included heavy intermittent or very heavy continuous exercise, which results in 2-3 times greater ventilation rate than in the prolonged (6.6- or 8-hour) exposure studies, which only incorporate moderate quasi-continuous exercise.[52] Across these shorter-duration studies, the lowest exposure concentration for which statistically significant respiratory effects were reported is 120 ppb, with the exposure combined with continuous heavy exercise. As recognized above the increased ventilation rate associated with increased exertion increases the amount of O_3 entering the lung, where depending on dose and the individual's susceptibility, it may cause respiratory effects (2013 ISA, section 6.2.1.1). Thus, for exposures involving a lower exertion level, a comparable response would not be expected to occur without a longer duration at this concentration (120 ppb), as is illustrated by the 6.6-hour study results for this concentration (Appendix 3A, Table 3A-1).

We have also considered what may be indicated by the epidemiologic studies regarding exposure concentrations associated with health effects, and particularly by such concentrations that might occur in locations when the current standard is met. In so doing, however, we recognize that these studies are generally focused on investigating the existence of a relationship between O_3 occurring in ambient air and specific health outcomes, and not on detailing the specific exposure circumstances eliciting such effects. While the evidence base of epidemiologic studies of associations between O_3 and respiratory effects and health outcomes (e.g., asthma-related hospital admission and emergency department visits), as a whole, provides strong support for the conclusions of causality, as summarized in section 3.3.1 above,[53] these studies generally do not measure personal exposures of the study population or track individuals in the population with a defined exposure to O_3 alone. Notwithstanding this, we have considered the epidemiologic studies identified in the ISA as to what they might indicate regarding O_3 exposure concentrations in this regard.

Among the epidemiologic studies finding a statistically significant positive relationship of short- or long-term O_3 concentrations with respiratory effects, there are no single-city studies conducted in the U.S. in locations with ambient air O_3 concentrations that would have met the current standard for the entire duration of the study (ISA, Appendix 3, Tables 3-13, 3-14, 3-39, 3-41, 3-42 and Appendix 6, Tables 6-5 and 6-6; PA, Appendix 3B, Tables 3B-1). There are

[52] 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).

[53] 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).

(among this large group of studies) two single city studies conducted in western Canada that include locations for which the highest-monitor design values fell just below 70 ppb (PA, Appendix 3B, Table 3B-1; Kousha and Rowe, 2014; Villeneuve et al., 2007). These studies did not, however, include analysis of correlations with other co-occurring pollutants or of the strength of the associations when accounting for effects of copollutants in copollutant models (ISA, Tables 3-14 and 3-39). Thus, the studies pose significant limitations with regard to informing conclusions regarding specific O_3 exposure concentrations and elicitation of such effects. There are also about a handful of multicity studies conducted in the U.S. or Canada[54] in which the O_3 concentrations in a subset of the study locations and for a portion of the study period appear to have met the current standard (Appendix 3B). Concentrations in other portions of the study area or study period, however, do not meet the standard, or data were not available in some cities for the earlier years of the study period when design values[55] for other cities in the study were well above 70 ppb. The extent to which reported associations with health outcomes in the resident populations in these studies are influenced by the periods of higher concentrations during times that did not meet the current standard is unknown. Additionally, with regard to multicity studies, the reported associations were based on the combined dataset from all cities, complicating interpretations regarding the contribution of concentrations in the small subset of locations that would have met the current standard compared to that from the larger number of locations that would have violated the standard (Appendix 3B, Table 3B-1 and Table 3B-2).[56] Further, given that populations in such studies may have also experienced longer-term, variable and uncharacterized exposure to O_3 (as well as to other ambient air pollutants), "disentangling the effects of short-term ozone exposure from those of long-term ozone exposure (and vice-versa) is an inherent uncertainty in the evidence base" (ISA, p. IS-87 [section IS.6.1]). While given the depth and breadth of the evidence base for O_3 respiratory effects, such uncertainties do not change our conclusions regarding the causal relationship between O_3 and respiratory effects, they affect the extent to which the two studies mentioned here (conducted in conditions that may

[54] Consistent with the evaluation of the epidemiologic evidence of associations between short-term O_3 exposure and respiratory health effects in the ISA, we focus on those studies conducted in the U.S. and Canada, and most particularly in the U.S., to provide a focus on study populations and air quality characteristics that are most relevant to circumstances in the U.S. (ISA, Appendix 3, section 3.1.2).

[55] 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.

[56] As recognized in the last review, "multicity studies do not provide a basis for considering the extent to which reported O_3 health effects associations are influenced by individual locations with ambient [air] O_3 concentrations low enough to meet the current O_3 standard versus locations with O_3 concentrations that violate this standard" (80 FR 64344, October 26, 2015).

have met the current standard) can inform our conclusions regarding the potential for O_3 concentrations allowed by the current standard to contribute to health effects.

We additionally considered the experimental animal evidence with regard to exposure conditions associated with respiratory effects. As noted in section 3.3.1 above, however, exposure concentrations in the animal studies were generally much greater than those examined in the controlled human exposure studies (and accordingly higher than concentrations commonly occurring in ambient air in areas of the U.S. where the current standard is met). This is also true for the small number of early life studies in nonhuman primates recognized in section 3.3.1.1 above that reported O_3 to contribute to allergic asthma-like effects in infant primates. The exposures eliciting the effects in these studies included multiple 5-day periods with O_3 concentrations of 500 ppb over 8-hours per day (ISA, Appendix 3, section 3.2.4.1.2).

With regard to short-term O_3 and metabolic effects, the category of effects for which the ISA concludes there to be a likely causal relationship with O_3, the evidence base is comprised primarily of experimental animal studies, as summarized in section 3.3.1.2 above (ISA, Appendix 5, section 5.1). The exposure conditions from these studies, however, generally involve much higher O_3 concentrations than those examined in the controlled human exposure studies for respiratory effects (and much higher than concentrations commonly occurring in ambient air in areas of the U.S. where the current standard is met). For example, the animal studies include 4-hour concentrations of 400 to 800 ppb (ISA, Appendix 5, Table 5-8).[57] The two epidemiologic studies reporting statistically significant positive associations of O_3 with metabolic effects (e.g., changes in glucose, insulin, metabolic clearance) are based in Taiwan and South Korea, respectively.[58] Given the potential for appreciable differences in air quality patterns between Taiwan and South Korea and the U.S., as well as differences in other factors that might affect exposure (e.g., activity patterns), those studies are of limited usefulness for informing our understanding of exposure concentrations and conditions eliciting such effects in the U.S. (ISA, Appendix 5, section 5.1).

[57] 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).

[58] Of the five epidemiologic studies discussed in the ISA that investigate associations between short-term O_3 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 O_3 concentration (ISA, Appendix 5, Tables 5-6 and 5-9). The South American study (focused on hospital admissions associated with diabetes complications) reported positive associations with 24-hr average concentrations for some subgroups, although no associations were statistically significant (ISA, Appendix 5, Table 5-9).

3.3.4 Uncertainties in the Health Effects Evidence

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

We have not identified any new uncertainties in the evidence since the last review. However, we continue to recognize important uncertainties that also existed in the last review. This array of important areas of uncertainty related to the current health evidence, including that newly available in this review, is summarized below.

Although the evidence clearly demonstrates that short-term O_3 exposures cause respiratory effects, as was the case in the last review, we continue to recognize uncertainties that remain in several aspects of our understanding of these effects. Such uncertainties include those associated with the severity and prevalence of responses to short (e.g., 6.6- to 8-hour) O_3 exposures at and below 60 ppb and responses of some population groups not well represented in the evidence base of controlled human exposure studies (e.g., children and people with asthma). There are also uncertainties concerning the potential influence of exposure history and co-exposure to other pollutants on the relationship between short-term O_3 exposure and respiratory effects. With regard to the full health effects evidence base, we also recognize as an important uncertainty the extent to which O_3 exposures are related to health effects other than respiratory effects. The following discussion touches on each of these types of uncertainty.

The majority of the available studies have generally involved healthy young adult subjects, although there are some studies involving subjects with asthma, and a limited number of studies, generally of very short durations (i.e., less than four hours), involving adolescents and adults older than 50. While there is evidence from short (6.6- to 8-hour) controlled exposure studies of healthy adult subjects to concentrations as low as 40 ppb, the only controlled human exposure study of such a duration (7.6 hours with quasi-continuous light exercise) conducted in people with asthma was for an exposure concentration of 160 ppb (Appendix 3A, Table 3A-2). Given a general lack of studies using subjects that have asthma, particularly those at exposure concentrations likely to occur under conditions meeting the current standard, uncertainties remain with regard to characterizing the response in people with asthma while at elevated ventilation to lower exposure concentrations, e.g., below 80 ppb. The extent to which the epidemiologic evidence, including that newly available, can inform this area of uncertainty also may be limited.[59] As discussed in section 3.3.2 above, given the effects of asthma on the

[59] Associations of health effects with O_3 that are reported in the epidemiologic analyses are based on air quality concentration metrics used as surrogates for the actual pattern of O_3 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.

respiratory system, exposures associated with significant respiratory responses in healthy people may pose an increased risk of more severe responses, including asthma exacerbation, in people with asthma. Such considerations remain areas of uncertainty in this review. Thus, uncertainty remains with regard to the extent to which the controlled human exposure study evidence describes the responses of the populations, such as children with asthma, that may be most at risk of O_3-related respiratory effects (e.g., through an increased likelihood of severe responses, or greatest likelihood of response).

Other areas of uncertainty concerning the potential influence of O_3 exposure history and co-exposure to other pollutants on the relationship between short-term O_3 exposures and respiratory effects also remain from the last review. As in the epidemiologic evidence in the last review, there is a limited number of studies that include copollutant analyses for a small set of pollutants (e.g., PM or NO_2). Recent studies with such analyses suggest that observed associations between O_3 concentrations and respiratory effects are independent of co-exposures to correlated pollutants or aeroallergens (ISA, sections IS.4.3.1 and IS.6.1; Appendix 3, sections 3.1.10.1 and 3.1.10.2). Despite the increased prevalence of copollutant modeling in recent epidemiologic studies, uncertainty still exists with regard to the independent effect of O_3 given the high correlations observed for some copollutants in some studies and the small fraction of all atmospheric pollutants included in these analyses (ISA, section IS.4.3.1; Appendix 2, section 2.5). We also note that neither of the two epidemiologic studies of respiratory outcomes conducting in Canadian areas that would have met the current standard included copollutant modeling (as recognized in section 3.3.3 above).

Further, although there remains uncertainty in the evidence with regard to the potential role of exposures to O_3 in eliciting health effects other than respiratory effects, the evidence has been strengthened since the last review with regard to metabolic effects. As noted in section 3.3.1.2 above, the ISA newly identifies metabolic effects as likely to be causally related to short-term O_3 exposures. The evidence supporting this relationship is limited and not without its own uncertainties. For example, as noted in section 3.3.1.2 above, the conclusion is based primarily on animal toxicological studies conducted at much higher O_3 concentrations than those common in ambient air in the U.S. A limited number of epidemiologic studies of short-term O_3 exposure and metabolic effects are available, many of which did not control for copollutants confounding; just two studies, both in Asia, report significant positive associations with changes in markers of glucose homeostasis (ISA, Appendix 5; sections 5.1.8 and 5.3). As noted in section 3.3.1.2 above, the ISA has also determined the evidence to be suggestive of, but not sufficient to infer, a causal relationship between long-term O_3 exposures and metabolic effects, and between O_3 exposures and several other categories of health effects, including effects on the cardiovascular, reproductive and nervous systems, and mortality (ISA, section IS.4.3). Additionally, the ISA

finds the evidence to be inadequate to determine if a causal relationship exists with O_3 and cancer (ISA, section IS.4.3).

In summary, a variety of uncertainties from the last review remain, including those related to the extent of effects at concentrations below those evaluated in controlled human exposure studies, and the potential for more severe impacts in individuals with asthma, including particularly children, and in other at-risk populations.

3.4 EXPOSURE AND RISK INFORMATION

Our consideration of the scientific evidence available in the current review, as at the time of the last review (summarized in section 3.1 above), is informed by results from quantitative analyses of estimated population exposure and consequent risk. Estimates from the exposure-based analyses, particularly the comparison of daily maximum exposures to benchmark concentrations, were most informative to the Administrator's decision in the last review (as summarized in section 3.1.2 above). This largely reflected the EPA conclusion that "controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O_3 exposures," and recognition that "effects reported in controlled human exposure studies are due solely to O_3 exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies)" (80 FR 65343, October 26, 2015). In the last 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).[60] Therefore, we have focused new quantitative analyses in this review on exposure-based risk analyses. This reflects the emphasis

[60] The 2015 decision notice 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 O_3 concentrations, as well as uncertainties related to the public health importance of increases in relatively low O_3 concentrations following air quality adjustment. Lower confidence was also placed in the results of the epidemiologic-based assessment of respiratory mortality risks associated with long-term O_3 exposures in consideration of several factors, as noted in section 3.1 above. Importantly in this review, the causal determinations for short-term O_3 exposure with mortality in the current ISA differ from the 2013 ISA. The current determinations for both short-term and long-term O_3 exposure (as summarized in section 3.1 above) are that the evidence is "suggestive" but not sufficient to infer causal relationships for O_3 with mortality (ISA, Table IS-1).

given to these types of analyses and the characterization of their uncertainties in the last review, along with the availability of new or updated information, models, and tools that address those uncertainties (IRP, Appendix 5A).

In the sections below, we summarize the currently available exposure and risk information for consideration in this review. In section 3.4.1, we summarize the conceptual model for the assessment, as well as key aspects of the assessment design, including the study areas, populations simulated, modeling tools, and exposure and risk metrics derived. Sections 3.4.2 and 3.4.3 summarize the assessment results. Key limitations and uncertainties associated with the assessment estimates are identified in section 3.4.4. and potential public health implications are discussed in section 3.4.5. An overarching consideration is whether the current exposure and risk information alters overall conclusions reached in the previous review regarding health risk associated with exposure to O_3 in ambient air.

3.4.1 Conceptual Model and Assessment Approach

The long-standing evidence base for O_3-related health effects is comprised of a large assemblage of controlled human exposure studies, laboratory animal research studies, and air quality epidemiologic studies. Together, these health effect studies lead to the strongly supported conclusion that O_3 exposure causes respiratory effects (as summarized in section 3.3 above). This conclusion is strongest with regard to short-term O_3 exposures, for which the ISA and science assessments in prior reviews have determined there to be a causal relationship. The ISA additionally determines the relationship between long-term exposure and respiratory effects, as well as between short-term exposures and metabolic effects to be likely causal, recognizing that associated uncertainties remain in the evidence. Given the relatively greater strength of the evidence and understanding of the relevant exposure conditions, as well as availability of appropriate data and modeling tools, we focus the exposure and risk analysis in this review on respiratory risks associated with short-term O_3 exposures.

The controlled human exposure studies document the occurrence of an array of respiratory effects in humans in a variety of short-term exposure circumstances. These studies, in combination with the laboratory animal studies, inform our understanding of the mode of action for O_3-attributable effects, including those health outcomes associated with ambient air concentrations in air quality epidemiologic studies (ISA, Appendix 3, section 3.1.3). Figure 3-3 below illustrates the conceptual model for O_3 in ambient air and respiratory effects, with a particular focus on short-term exposures and including linkages with the risk metrics assessed in the quantitative analyses performed for this review.

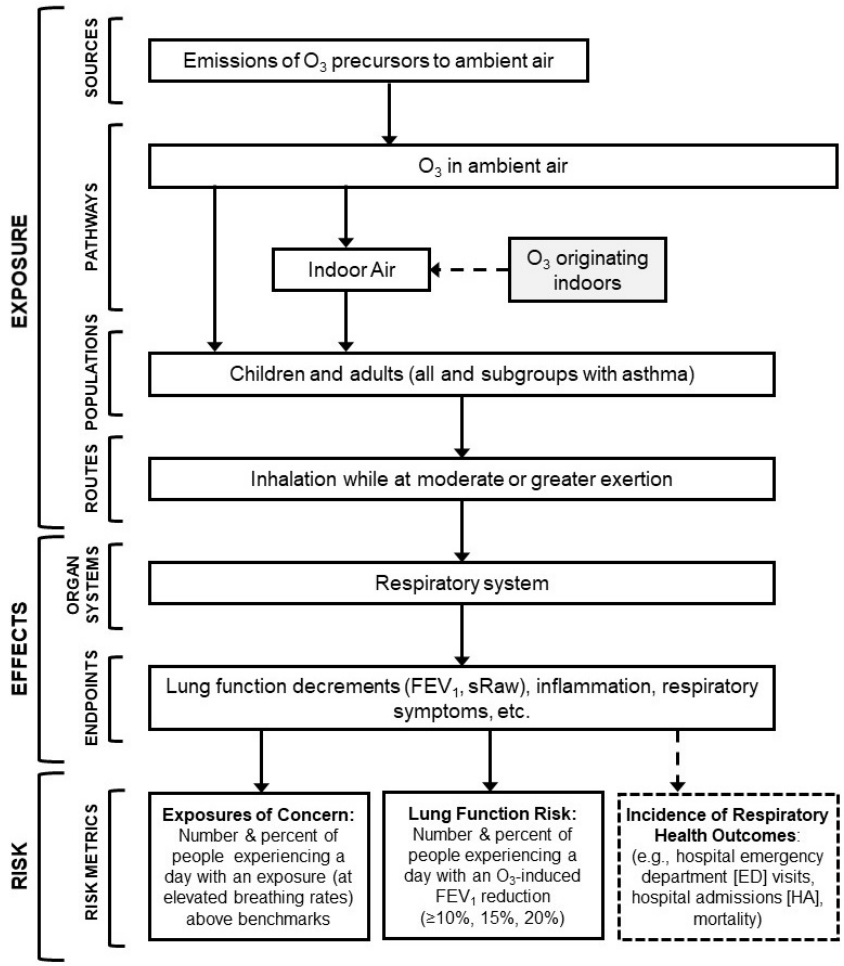

Figure 3-3. Conceptual model for exposure-based risk assessment. Solid lines indicate processes explicitly modeled in the assessment. Dashed lines indicate relationships that are not explicitly modeled.

Based on this conceptual model, as well as newly available information, an exposure-based assessment was developed for this review. In this assessment, described in detail in Appendix 3D, we have estimated O_3 exposures and resulting risk for air quality conditions of interest, most particularly air quality conditions that just meet the current primary O_3 standard. These analyses inform our understanding of the protection provided by the current primary standard from effects that the health effects evidence indicates to be elicited in some portion of exercising people exposed for several hours to elevated O_3 concentrations.

The analysis approach employed is summarized in Figure 3-4 below and described in detail in Appendices 3C and 3D. This approach incorporates the use of an array of models and data to develop population exposure and risk estimates for a set of eight urban study areas. Ambient air O_3 concentrations were estimated in each study area using an approach that relies on a combination of ambient air monitoring data, atmospheric photochemical modeling and

statistical methods (described in detail in Appendix 3C). Population exposure and risk modeling is employed to characterize exposures and related lung function risk associated with the ambient air concentration estimates(described in detail in Appendix 3D). While the lung function risk analysis focuses only on the specific O_3 effect of FEV_1 reduction, the comparison-to-benchmark approach, with its use of multiple benchmark concentrations, provides for characterization of the risk of other respiratory effects, the type and severity of which increase with increased exposure concentration.

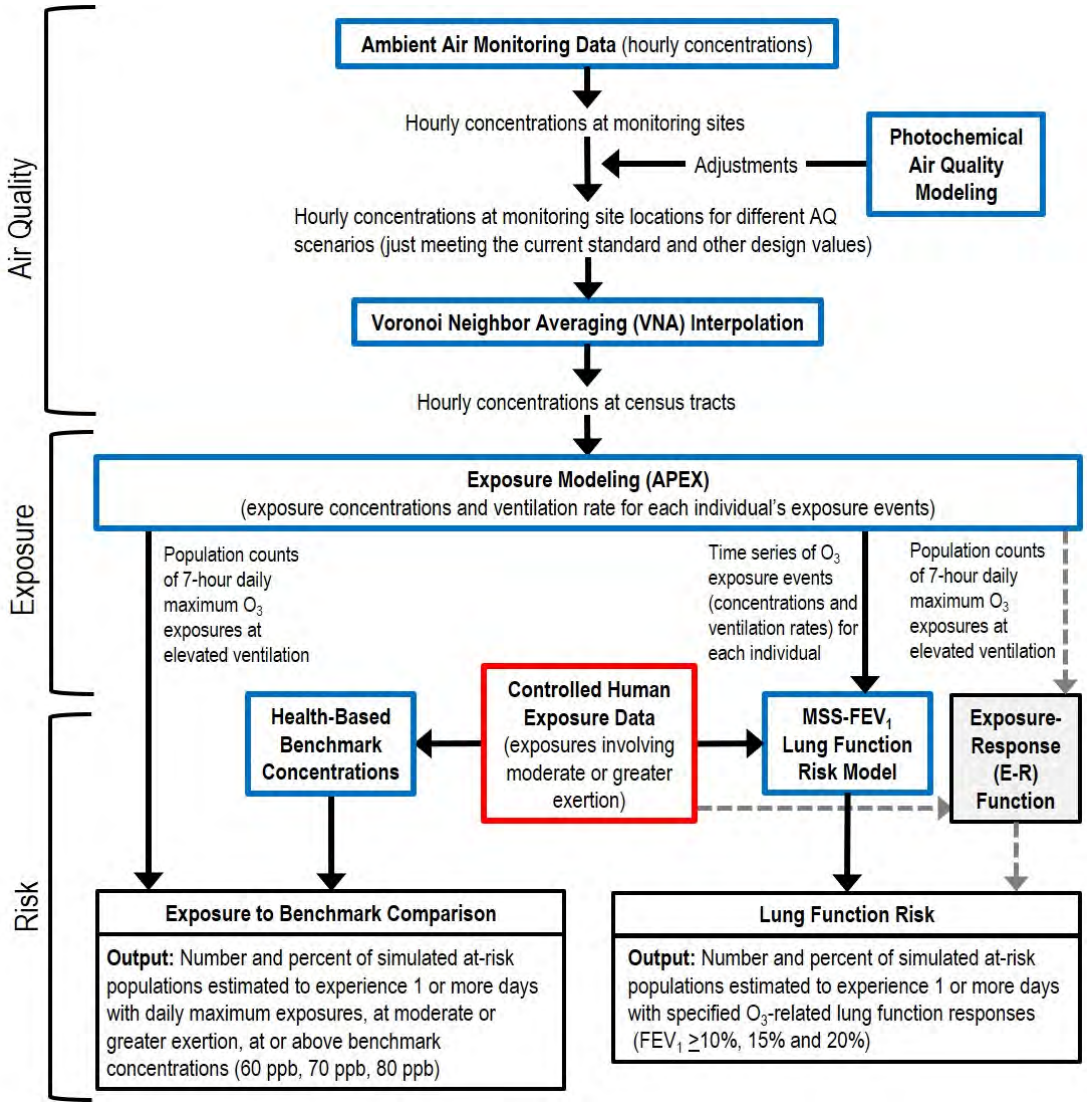

Figure 3-4. Analysis approach for exposure-based risk analyses. Dashed lines and gray box indicate the sole lung function risk approach used prior to 2014 HREA.

The analyses estimate exposure and risk for simulated populations in eight study areas in Atlanta, Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento and St. Louis. The eight study areas represent a variety of circumstances with regard to population exposure to short-term concentrations of O_3 in ambient air. The eight study areas range in total population size from

approximately two to eight million and are distributed across the U.S. in seven different NOAA climate regions: the Northeast, Southeast, Central, East North Central, South, Southwest and West (Karl and Koss, 1984). Assessment of this set of study areas and the associated exposed populations is intended to be informative to the EPA's consideration of potential exposures and risks that may be associated with the air quality conditions that meet the current primary standard.

This set of eight study areas represents a streamlined set as compared to the 15 study areas in the last review but were chosen to ensure they reflect the full range of air quality and exposure variation expected across major urban areas in the U.S. (2014 HREA, section 3.5). Accordingly, while seven of the eight study areas were also included in the 2014 HREA, the eighth study area is newly added in the current assessment to insure representation of a large city in the southwest. Additionally, the years simulated reflect more recent emissions and atmospheric conditions subsequent to data used in the 2014 HREA, and therefore represent O_3 concentrations somewhat nearer the current standard than was the case for study areas included in the HREA of the last review (Appendix 3C, Table 3C and 2014 HREA, Table 4-1). Thus, the urban study areas (e.g., combined statistical areas that include urban and suburban populations) for which the exposure and risk analyses have been conducted for this review reflect an array of air quality, meteorological, and population exposure conditions.

Consistent with the health effects evidence in this review (summarized in section 3.3 above), the focus of the assessment is on short-term exposures of individuals in the population during times when they are breathing at an elevated rate. Exposure and risk are characterized for four population groups. Two are populations of school-aged children, aged 5 to 18 years:[61] all children and children with asthma. Two are populations of adults: all adults and adults with asthma. Asthma prevalence estimates for the eight study areas ranges from 7.7 to 11.2% (Appendix 3D, section 3D.3.1). For children, the study area asthma prevalence rates range from 9.2 to 12.3% (Appendix 3D, section 3D.3.1). Spatial variation within each study area related to the population distribution of age, sex, and family income was also taken into account.[62] For children, this variation is greatest in the Detroit study area, with census tract level, age-specific

[61] 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, as well as challenges in asthma diagnoses for children younger than 5 years old.

[62] 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.

asthma prevalence estimates ranging from 6.4 to 13.2% for girls and from 7.7 to 25.5% for boys (Appendix 3D, Table 3D-3).

Ambient air O_3 concentrations were estimated in each study area for the air quality conditions of interest by adjusting ambient air monitoring data using a photochemical model-based approach and then applying a spatial interpolation technique to produce air quality surfaces with high spatial and temporal resolution (Appendix 3C).[63] The photochemical modeling outputs included both modeled O_3 concentrations and sensitivities of O_3 concentrations to changes in NO_X emissions for each hour in a single year at all ambient air monitor locations (Appendix 3C, sections 3C.4 and 3C.5). Linear regression was used with these single-year model outputs to create relationships between the sensitivities and O_3 concentrations for each hour of each of the four seasons at each monitoring location. The relationships between hourly sensitivities and hourly O_3 for each season were then used with three years of ambient air monitoring data at each location to predict hourly sensitivities for the complete 3-year record at each monitoring location. From these, we calculated hourly O_3 concentrations at each monitor location based on iteratively increasing NO_X reductions to determine the adjustments necessary for the monitor location with the highest design value in each study area to just meet the target value, e.g., 70 ppb for the current standard scenario (Appendix 3C, section 3C.5). Hourly O_3 concentrations for all census tracts comprising each study area were then derived from the model adjusted hourly concentrations at the ambient air monitor locations using the Voronoi Neighbor Averaging (VNA) spatial interpolation technique (Appendix 3C, section 3C.6). The final product was a dataset of ambient air O_3 concentration estimates with high temporal and spatial resolution (hourly concentrations in 500 to 1700 census tracts) for each of the eight study areas (Appendix 3C, section 3C.7).

The photochemical modeling approach involved use of the Comprehensive Air Quality Model with Extensions (CAMx), version 6.5, instrumented with the higher order decoupled direct method (HDDM).[64] The CAMx-HDDM was run with emissions estimates and meteorology data for calendar year 2016 to estimate the O_3 sensitivities,[65] and the linear regressions of the modeled O_3 concentrations to their respective sensitivities were applied to hourly O_3 concentrations reported at ambient air monitors for the 2015-2017 period to determine the adjustments needed for each air quality scenario (Appendix 3C, sections 3C.4 and 3C.5). We

[63] A similar approach was used to develop the air quality scenarios for the 2014 HREA.

[64] 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).

[65] Sensitivities of O_3 refer to predicted incremental changes in O_3 concentrations in response to incremental changes in precursor emissions (e.g., NO_X emissions).

maximized the spatial representation of the monitoring data by using all available monitors within each study area (between 12 and 30) in addition to those within 50 km of the study area boundaries (yielding between 5 and 31 additional monitors per area). Because we selected study areas having design values close to the level of the current standard, the levels of NO_X emissions adjustments needed to meet the air quality scenarios of interest were generally lower than those used in the 2014 HREA, thus reducing one of the important sources of uncertainty associated with these air quality estimates.

Population exposures were estimated using the EPA's Air Pollutant Exposure model (APEX) version 5, which probabilistically generates a large sample of hypothetical individuals from a population database and simulates each individual's movements through time and space to estimate their time-series of O_3 exposures occurring within indoor, outdoor, and in-vehicle microenvironments (Appendix 3D, section 3D.2).[66] The APEX model accounts for the most important factors that contribute to human exposure to O_3 from ambient air, including the temporal and spatial distributions of people and ambient air O_3 concentrations throughout a study area, the variation of ambient air-related O_3 concentrations within various microenvironments in which people conduct their daily activities, and the effects of activities involving different levels of exertion on breathing rate (or ventilation rate) for the exposed individuals of different sex, age, and body mass in the study area (Appendix 3D, section 3D.2). The APEX model generates each simulated person or profile by probabilistically selecting values for a set of profile variables, including demographic variables, health status and physical attributes (e.g., residence with air conditioning, height, weight, body surface area) and ventilation rate (Appendix 3D, section 3D.2).

The activity patterns of individuals are an important determinant of their exposure (2013 ISA, section 4.4.1). By incorporating individual activity patterns,[67] the model estimates physical exertion associated with each exposure event.[68] This aspect of the exposure modeling is critical

[66] 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 last review of the O_3 NAAQS (U.S. EPA, 2008; U.S. EPA, 2009; U.S. EPA, 2010b; U.S. EPA, 2014; U.S. EPA, 2018).

[67] 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).

[68] 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

in estimating exposure, ventilation rate, O_3 intake (dose), and health risk resulting from ambient air concentrations of O_3.[69] Because of variation in O_3 concentrations among the different microenvironments in which individuals are active, the amount of time spent in each location, as well as the exertion level of the activity performed, will influence an individual's exposure to O_3 from ambient air and potential for adverse health effects. Activity patterns vary both among and within individuals, resulting in corresponding variations in exposure across a population and over time (2013 ISA, section 4.4.1). For each exposure event, APEX tracks activity performed, ventilation rate, exposure concentration, and duration. The time-series of exposure events serve as the basis for calculating exposure and risk metrics of interest.

As in the last review, the quantitative analyses for this review uses the APEX model estimates of population exposures for simulated individuals breathing at elevated rates[70] to characterize health risk based on information from the controlled human exposure studies on the incidence of lung function decrements in study subjects who are exposed over multiple hours while intermittently or quasi-continuously exercising (Appendix 3D, section 3D.2.8). In drawing on this evidence base for this purpose, the assessment has given primary focus to the well-documented controlled human exposure studies summarized in Appendix 3A, Table 3A-1 for 6.6-hour average exposure concentrations ranging from 40 ppb to 120 ppb (Figure 3-2; ISA, Appendix 3, Figure 3-3). Health risk is characterized in two ways, producing two types of risk metrics: one involving comparison of population exposures involving elevated exertion to benchmark concentrations (that are specific to elevated exertion exposures), and the second involving estimated population occurrences of ambient air O_3-related lung function decrements (Figure 3-2). The first risk metric is based on comparison of estimated daily maximum 7-hour average exposure concentrations for individuals breathing at elevated rates to concentrations of potential concern (benchmark concentrations). The second metric (lung function risk) uses E-R information for O_3 exposures and FEV_1 decrements to estimate the portion of the simulated at-risk population expected to experience one or more days with an O_3-related FEV_1 decrement of at least 10%, 15% and 20%. Both of these metrics are used to characterize health risk associated with O_3 exposures among the simulated population during periods of elevated breathing rates. Similar risk metrics were also derived in the HREA for the last review and the associated

microenvironmental concentration and/or activity/activity level changes, a new exposure event occurs (McCurdy and Graham, 2003).

[69] Indoor sources are generally minor in comparison to O_3 from ambient air (ISA, Appendix 2, section 2.4.3) and are not accounted for by the exposure modeling in this assessment.

[70] 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).

estimates informed the Administrator's 2015 decision on the current standard (80 FR 65292, October 26, 2015).

The general approach and methodology for the exposure-based assessment used in this review is similar to that used in the last review. However, a number of updates and improvements have been implemented in this review which result in differences from the analyses in the prior review (Appendices 3C and 3D). These relate to the air quality, exposure, and risk aspects of the assessment as summarized here.

- The ambient air monitoring data used is from a more recent period (e.g., 2015-2017) during which O_3 concentrations in the eight study areas are at or near the current standard (Appendix 3C, Table 3C-1). This contrasts with the 2014 HREA use of 2006-2010 air monitoring data, that for many study areas included design values (for unadjusted concentrations) well above (e.g., by more than 10 ppb) the level of the then-existing standard (2014 HREA, section 4.3.1.1, Table 4-1). The use of more recent ambient air monitoring data in the current analysis allows for smaller adjustments to develop the air quality conditions of interest, thus contributing to generally lesser uncertainty in the concentrations estimated in each air quality scenario.
- The most recent CAMx model, with updates to the treatment of atmospheric chemistry and physics within the model, is used to derive spatially and temporally varying relationships between changes to emissions and modeled O_3 concentrations, which are then used in adjusting ambient air concentrations to just meet the air quality scenarios. Model inputs represent recent year emissions, meteorology, and international transport (e.g., 2016). The 2016-based inputs were derived using updated methods for calculating emissions, as well as updated meteorological and hemispheric photochemical models (described in more detail in Appendix 3C).
- Population exposure modeling inputs include the most recent U.S. Census demographics and commuting data (i.e., 2010), meteorological data to reflect the assessment years studied (e.g., 2015-2017), and updated estimates of asthma prevalence for all census tracts in all study areas (e.g., 2013-2017). Regarding asthma prevalence, the more recent information includes increased prevalence reported for adults and for children aged 10-17 years (Akinbami et al., 2016; CDC, 2016).[71]
- The APEX equations used to estimate of ventilation rate ($\dot{V}_E$) and resting metabolic rate have been updated such that the overall statistical model fit and predictability has been improved (U.S. EPA, 2018, Appendix H).
- The approach for deriving population exposure estimates, both for comparison to benchmark concentrations and for use in deriving lung function risk using the E-R function, has been modified to provide for a better match of the simulated population exposure estimates with the 6.6-hour duration of the controlled human exposure studies and with the study subject ventilation rates (Appendix 3D, section 3D.2.8.1). The modifications include deriving estimates for exposures of a duration and ventilation rate

[71] For more information, see *https://www.cdc.gov/nchs/products/databriefs/db239.htm*.

more closely corresponding to the duration and average ventilation rate across the 6.6-hour duration in the controlled human exposure studies (Appendix 3D, section 3D.2.8.1).[72]

- In addition to the E-R function, as updated in the 2014 HREA, an updated version of the McDonnell Stewart Smith model (MSS-FEV_1 model, McDonnell et al., 2013) is used to estimate individual-based lung function risk. Although the impact on risk estimates is unclear, the updated MSS model has been described as better accounting for intra-subject variability, yielding an improved model fit (McDonnell et al., 2013; Appendix 3D, section 3D.2.8.2.2).

The exposure-to-benchmark comparison characterizes the extent to which individuals in at-risk populations could experience O_3 exposures, while engaging in their daily activities, with the potential to elicit the effects reported in controlled human exposure studies for concentrations at or above specific benchmark concentrations. Results are characterized using three benchmark concentrations of O_3: 60, 70, and 80 ppb. These are based on the three lowest concentrations targeted in studies of 6- to 6.6-hour exposures, with quasi-continuous exercise, and that yielded different occurrences and severity of respiratory effects (section 3.3.3 above; Appendix 3A, section 3A.1; Appendix 3D, section 3D.2.8.1). The lowest benchmark, 60 ppb, represents the lowest exposure concentration for which controlled human exposure studies have reported statistically significant respiratory effects. At this concentration, there is evidence of a statistically significant decrease in lung function and increase in airway inflammation (ISA, Appendix 3, section 3.1.4.1.1; Brown et al., 2008; Adams, 2006b). Exposure to approximately 70 ppb[73] averaged over a similar time resulted in a larger group mean lung function decrement, as well as an increase in prevalence of respiratory symptoms over what was observed for 60 ppb (Figure 3-3; ISA, Appendix 3, section 3.1.4.1.1; Schelegle et al., 2009). Studies of exposures to approximately 80 ppb have reported larger lung function decrements at the study group mean than following exposures to 60 or 70 ppb, in addition to an increase in airway inflammation, increased respiratory symptoms, increased airway responsiveness, and decreased resistance to other respiratory effects (Figure 3-3 and section 3.3.3, above; ISA, Appendix 3, sections 3.1.4.1-

[72] 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-m^2 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).

[73] 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 O_3 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).

3.1.4.4). The APEX-generated exposure concentrations for comparison to these benchmark concentrations is the average of concentrations encountered by an individual while at an activity level that elicits the specified elevated ventilation rate.[74] The incidence of such exposures above the benchmark concentrations are summarized for each simulated population, study area, and air quality scenario as discussed in sections 3.4.2 and 3.4.3 below.

The lung function risk analysis provides estimates of the extent to which individuals in the populations could experience decrements in lung function. Estimates were derived for risk of experiencing a day with a lung function decrement at or above three different magnitudes, i.e., FEV_1 reductions of at least 10%, 15%, and 20%. Lung function decrement risk was estimated by two different approaches, which utilize the evidence from the 6.6-hour controlled human exposure studies in different ways.[75] One, the population-based E-R function, uses quantitative descriptions of the E-R relationships for study group incidence of the different lung function decrements based on the individual study subject observations. The second, the individual-based MSS model, uses quantitative estimations of biological processes identified as important in eliciting the different sizes of decrements at the individual level, with a factor that also provides a representation of intra- and inter-individual response variability (Appendix 3D, section 3D.2.8.2.2).

The E-R function used for estimating the risk of lung function decrements was developed from the individual study subject measurements of O_3-related FEV_1 decrements from the 6.6-hour controlled human exposure studies targeting mean exposure concentrations from 120 ppb down to 40 ppb (Appendix 3D, Table 3D-19; Appendix 3A, Figure 3A-1). The FEV_1 responses reported in these studies have been summarized in terms of percent of study subjects experiencing O_3-related decrements equal to at least 10%, 15% or 20%. Across the exposure range from 40 to 120 ppb, the percentage of exercising study subjects with asthma estimated to have at least a 10% O_3 related FEV_1 decrement increases from 0 to 7% (a statistically non-

[74] 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 (m^2). Seven-hour exposure concentrations associated with 7-hour average EVR at or above the target of 17.3 ± 1.2 L/min-m^2 (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).

[75] In so doing, the approaches also estimate responses associated with unstudied exposure circumstances and population groups in different ways.

significant response at exposures of 40 ppb) up to approximately 50 to 70% (at exposures of 120 ppb) (Appendix 3D, Section 3D.2.8.2.1, Table 3D-19). The E-R function relies on equations that describe the fraction of the population experiencing a particular size decrement as a function of the exposure concentration experienced while at the target ventilation rate. This type of risk model, which has been used in risk assessments since the 1997 O_3 NAAQS review, was updated in the last review to include the more recently available study data (Appendix 3D, section 3D.2.8.2.1). In this review, the functions (fraction of the population having of a day or more per simulation period with at least one decrement of one of the specified sizes) are applied to the APEX estimates of 7-hour average exposure concentrations concomitant with the target ventilation level estimated by APEX, with the results presented in terms of number of individuals in the simulated populations (and percent of the population) estimated to experience a day (or more) with a lung function decrement at or above 10%, 15% and 20%.

The MSS model, also used for estimating the risk of lung function decrements, was developed using the extensive database from controlled human exposure studies that has been compiled over the past several decades, and biological concepts based on that evidence (McDonnell et al., 2012; McDonnell et al., 2013). The model mathematically estimates the magnitude of FEV1 decrement as a function of inhaled O_3 dose (based on concentration & ventilation rate) over the time period of interest (Appendix 3D, section 3D.2.8.2.2). The simulation of decrements is dynamic, based on a balance between predicted development of the decrement in response to inhaled dose and predicted recovery (using a decay factor). Each occurrence of decrements of interest (e.g., at or above 10%, 15% and 20%) is tallied. This model was first applied in combination with the APEX model to generate lung function risk estimates in the last review (80 FR 65314, October 26, 2015). As noted below, the MSS model used in the current assessment has been updated since the previous review based on the most recent study by its developers (McDonnell et al., 2013). In this review, the model is applied to the APEX estimates of exposure concentration and ventilation for every exposure event experienced by a simulated individual. The model then utilizes its mathematical descriptions of dose accumulation and decay, and relationship of dose to response, to estimate the magnitude of O_3 response associated with the sequence of exposure events in each individual's day. We report the MSS model risk results using the same metrics as for the E-R function, i.e., number of individuals in the simulated populations (and percent of the population) estimated to experience a day (or more) per simulation period with a lung function decrement at or above 10%, 15% and 20%.

The comparison-to-benchmark analysis (involving comparison of 7-hour average exposure concentrations that coincide with a 7-hour average elevated ventilation rates) provides perspective on the extent to which the air quality being assessed could be associated with discrete exposures to O_3 concentrations reported to result in respiratory effects. For example,

estimates of such exposures can indicate the potential for O_3-related effects in the exposed population, including effects for which we do not have E-R functions that could be used in quantitative risk analyses (e.g., airway inflammation). The comparison-to-benchmark analysis differs from the two lung function risk analyses which estimate the population incidence of one or more days with specific lung function decrements of magnitudes of interest based on two different uses of the health effects evidence.

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

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

- **What are the nature and magnitude of O_3 exposures and associated health risks for air quality conditions just meeting the current standard? What portions of the exposed populations are estimated to experience exposures of concern or lung function decrements?**

To address these questions, we consider the estimates provided by the exposure and risk simulations for the eight urban study areas with air quality conditions adjusted to just meet the current standard (Appendix 3D, sections 3D.3.2 through 3D.3.3). In considering these estimates here and their associated limitations, uncertainties and implications in greater depth in sections 3.4.5 and 3.5 below, we particularly focus on the extent of protection provided by the standard from O_3 exposures of potential concern. As described in the prior section, the exposure and risk analyses present two types of risk estimates for the 3-year simulation in each study area: (1) the number and percent of simulated people experiencing exposures at or above the particular benchmark concentrations of interest in a year, while breathing at elevated rates; and (2) the number and percent of people estimated to experience at least one O_3-related lung function decrement (specifically, FEV_1 reductions of a magnitude at or above 10%, 15% or 20%) in a year and the number and percent of people estimated to experience multiple lung function decrements associated with O_3 exposures.

As an initial matter, we note that, as indicated by the use of an urban case study approach (summarized in section 3.4.1 above), the exposure and risk analyses are not intended to provide a comprehensive national assessment. Nor is the objective to present an exhaustive analysis of exposure and risk in the areas that currently just meet the current standard and/or of exposure and risk associated with air quality adjusted to just meet the current standard in areas that currently do not meet the standard. Rather, the analyses are intended to provide assessments of an air quality scenario just meeting the current standard for a diverse set of study areas and associated exposed populations. The purpose is to assess, based on current tools and information, the potential for exposures and risks beyond those indicated by the information available at the time

the standard was established. Accordingly, capturing an appropriate diversity in study areas and air quality conditions (that reflect the current standard scenario)[76] is important to the role of the exposure and risk analyses in informing the Administrator's conclusions on the public health protection afforded by the current standard.

Of the two types of risk metrics derived in the exposure and risk analyses, we turn first to the results for the benchmark-based risk metric with regard to the percent of the simulated populations of all children and children with asthma estimated to experience at least one day per year[77] with a daily maximum 7-hour average exposure concentration at or above the different benchmark concentrations while breathing at elevated rates under air quality conditions just meeting the current standard (Table 3-3). Estimates for adults, in terms of percentages, are lower, generally due to the lesser amount and frequency of time spent outdoors at elevated exertion (Appendix 3D, section 3D.3.2). The exception to this is for outdoor workers, who due to the requirements of their job spend more time outdoors. As information for this group, including specific durations of time spent outdoors and activity data, is limited, the group was not simulated in this assessment, although we note that a targeted analysis was performed in the 2014 HREA.[78] Given the recognition of people with asthma as an at-risk population and the relatively greater amount and frequency of time spent outdoors at elevated exertion of children, we focus here on the estimates for children, including children with asthma.

[76] A broad variety of spatial and temporal patterns of O_3 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.

[77] The three years of ambient air O_3 concentrations analyzed in the exposure assessment analyses include concentrations during the O_3 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 O_3 season for each year may vary across the study areas, for the purposes of the exposure and risk analyses, the O_3 season in each study area is considered synonymous with a year.

[78] Targeted analyses of outdoor workers in the 2014 HREA (single study area, single year) found an appreciably greater portion of this population as compared to the full population of adults estimated to experience exposures at or above benchmark concentration, and particularly to experience such exposures on multiple days (2014 HREA, section 5.4.3.2). The estimates for the outdoor worker population, for the single urban area and year simulated, were also somewhat higher than those for the child population. For a number of reasons, including the appreciable data limitations and associated uncertainties summarized in Table 3D-64 of Appendix 3D, outdoor workers are not a population that has been explicitly simulated in the current analyses. It is expected that if an approach similar to that used in the 2014 HREA were used for this assessment the distribution of exposures (single day and multiday) would be similar to that estimated in the 2014 HREA (e.g., 2014 HREA, Figure 5-14), although with slightly lower overall percentages (and based on the comparison of current estimates with estimates from the 2014 HREA) (Appendix 3D, section 3D.3.2.4).

Under air quality conditions just meeting the current standard, approximately 3% to nearly 9% of each study area's simulated children with asthma, on average across the 3-year period, are estimated to experience one or more days per year with a daily maximum 7-hour average exposure at or above 60 ppb while breathing at elevated rates (Table 3-3). This range is very similar for the populations of all children (Table 3-3). With regard to the 70 ppb benchmark, the study areas' estimates for children with asthma are as high as 0.7 percent (0.6% for all children), on average across the 3-year period, and range up to 1.0% in a single year (Table 3-3). Less than 0.1% of any area's children with asthma, on average, were estimated to experience any days per year with a daily maximum 7-hour average exposure at or above 80 ppb (Table 3-3). Looking at estimates for multiple-day occurrences, we see that no children are estimated to experience more than a single day with a daily maximum 7-hour average exposure at or above 80 ppb in any year simulated in any location (Table 3-3). For the 70 ppb benchmark, the estimate is less than 0.1% of any area's children (on average across 3-year period), both those with asthma and all children (Table 3-3, Figure 3-4). The estimates for the 60 ppb benchmark are slightly higher, with up to 3% of children estimated to experience more than a single day with a daily maximum 7-hour average exposure at or above 60 ppb, on average (and more than 4% in the highest year across all eight study area locations) (Table 3-3).

These estimates are based on analyses that, while based on conceptually similar approaches to those used in the 2014 HREA, reflect the updates and revisions to those approaches that have been implemented since that time. Taking that into consideration, the estimates for the 3-year period from the current assessment for air quality conditions simulated to just meet the current standard are of a magnitude roughly similar, although slightly lower at the upper end of the ranges, to the estimates for these same populations in the 2014 HREA. For example, for air quality conditions just meeting the standard with a level of 70 ppb, the 2014 HREA estimated 0.1 to 1.2% of children to experience at least one day with exposure at or above 70 ppb, while at elevated ventilation (Section 3D.3.2.4, Table 3D-38). There are a number of differences between the quantitative modeling and analyses performed in the current assessment and the 2014 HREA that likely contribute to the small differences in estimates between the two assessments (e.g., 2015-2017 vs. 2006-2010 distribution of ambient air concentrations, full statistical distribution of ventilation rates vs. a 5^{th} percentile point estimate, 7-hour vs. 8-hour exposure durations).

Table 3-3. Percent and number of simulated children and children with asthma estimated to experience at least one or more days per year with a daily maximum 7-hour average exposure at or above indicated concentration while breathing at an 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
Children with asthma	*- percent of simulated population* [A]					
≥ 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
- number of individuals [A]						
≥ 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
All children	*- percent of simulated population* [A]					
≥ 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
- number of individuals [A]						
≥ 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).

In framing these same exposure estimates from the perspective of estimated protection provided by the current standard, these results indicate that, in the single year with the highest concentrations across the 3-year period, 99% of the population of children with asthma would not be expected to experience such a day with an exposure at or above the 70 ppb benchmark; 99.9% would not be expected to experience such a day with exposure at or above the 80 ppb benchmark. The estimates, on average across the 3-year period, indicate that over 99.9%, 99.3% and 91.2% of the population of children with asthma would not be expected to experience a day with a daily maximum 7-hour average exposure while at elevated ventilation that is at or above 80 ppb, 70 ppb and 60 ppb, respectively (Table 3-3 above). Further, with regard to multiple days, more than approximately 97% of all children or children with asthma, on average across a 3-year period, are estimated to be protected against multiple days of exposures at or above 60 ppb.

These estimates are of a magnitude roughly consistent with the level of protection that was described in establishing the now-current standard in 2015 (as summarized in section 3.1 above).

With regard to lung function risk, the estimates for all children and for children with asthma are again roughly similar, with the higher end of the ranges for the eight study areas being just slightly higher in some cases for the children with asthma (Table 3-4). The lung function risk estimates from the MSS model are appreciably higher than those based on the E-R function (full results in Appendix 3D, section 3D.3.3). This difference relates to the fact, noted in section 3.4.1 above, that the two lung function risk approaches are based on different aspects of the controlled human exposure study evidence and differ in how they extrapolate beyond the exposure study conditions and observations. Accordingly, uncertainties associated with the two modeling approaches also differ (as discussed in section 3.4.4 below). The E-R function risk approach conforms more closely to the circumstances of the 6.6-hour controlled human exposure studies, such that the 7-hour duration and moderate or greater exertion level are necessary for nonzero risk. This approach does, however, use a continuous function which predicts responses for exposure concentrations below those studied down to zero. As a result, exposures below those studied in the controlled human exposures will result in a fraction of the population being estimated by the E-R function to experience a lung function decrement (albeit to an increasingly small degree with decreasing exposures). The MSS model, which has been developed based on a conceptualization intended to reflect a broader set of controlled human exposure studies (e.g., including studies of exposures to higher concentrations for shorter durations), does not require a 7-hour duration for estimation of a response, and lung function decrements are estimated for exertion below moderate or greater levels, as well as for exposure concentrations below those studied (Appendix 3D, section 3D.3.4.2; 2014 HREA section 6.3.3). These differences in the models, accordingly, result in differences in the extent to which they reflect the particular conditions of the available controlled human exposure studies and the frequency and magnitude of the measured responses.[79]

For example, the 6.6-hour controlled human exposure studies have reported approximately 3% of subjects exposed to an average concentration of 60 ppb and 10% of subjects exposed to 70 ppb to have at least a 15% FEV_1 decrement (Appendix 3D, Table 3D-20 and Figure 3D-11). Table 3-3 above shows that, at a maximum, approximately 11% and 1% of children with asthma are estimated in a single year to have a day with daily maximum 7-hour exposure at or above the 60 ppb and 70 ppb benchmarks, respectively, indicating that perhaps 10% (11% minus 1%) might be expected to have a day with an exposure at or above 60 ppb but

[79] 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.

less than 70 ppb. If the simulated children had the same sensitivity as the controlled human exposure study subjects, it might be expected that 0.3% (3% times 10%) of this group could have a 15% (or larger) FEV_1 decrement resulting from concentrations at or above 60 ppb and less than 70 ppb and 0.1% (10% times 1%) of this group could have a 15% (or larger) decrement resulting from concentrations at or above 70 ppb. Accordingly, this would yield an estimated lung function risk for the simulated population of 0.4% for decrements of 15% or larger. This contrasts with the estimates based on the E-R function, that are at most a 1% risk (Table 3-4), and the MSS model estimates, that are at most an 8.7% risk (Table 3-4).

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

3.4.3 Population Exposure and Risk Estimates for Additional Air Quality Scenarios

In addition to estimating population exposure and risk for O_3 concentrations simulated to occur under air quality conditions when the current standard is just met, the exposure and risk

analyses also estimated population exposure and risk in the eight study areas for two additional air quality scenarios. 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.

The results for the comparison-to-benchmarks analysis for these additional air quality scenarios are summarized in Table 3-5 below for all three benchmark concentrations. The estimates for these two additional scenarios differ markedly from the results for air quality just meeting the current standard (summarized in Table 3-3 above). For simplicity, the summary of the comparison discussed here focuses on the 70 ppb benchmark concentration, which falls just below the time-weighted exposure concentration for which there was a statistically significant lung function decrement and also a statistically significant increase in respiratory symptom score in one of the controlled human exposure studies, as noted in section 3.3.3 (ISA, Appendix 3, section 3.1.4.1.1; Schelegle et al., 2009). The pattern is similar for the other two benchmarks, although in general, the differences from the results for the current standard (presented in section 3.4.2) are somewhat greater for the higher benchmark and slightly smaller for the lower benchmark.

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

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

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

Table 3-5. Percent and number of simulated children and children with asthma estimated to experience one or more days per year with a daily maximum 7-hour average exposure at or above indicated concentration while breathing at 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
Air quality scenario for 75 ppb						
Children with asthma	*- percent of simulated population* [A]					
≥ 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
			- number of individuals [A]			
≥ 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
All children	*- percent of simulated population* [A]					
≥ 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
			- number of individuals [A]			
≥ 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
Air quality scenario for 65 ppb						
Children with asthma	*- percent of simulated population* [A]					
≥ 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
			- number of individuals [A]			
≥ 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
All children	*- percent of simulated population* [A]					
≥ 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
			- number of individuals [A]			
≥ 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.

Lung function risk estimated for children and children with asthma in air quality scenarios with design values just above and below the current standard are presented in detail in Appendix 3D, section 3D.3.3. The patterns of the estimates are, as expected, higher for the 75 ppb air quality scenario and lower for the 65 ppb scenario. For each scenario, the differences in risk estimates between the two models is similar to that which occurs with the risk estimates for air quality just meeting the current standard (as discussed in section 3.4.2 above). These estimates (for both lung function risk approaches) are less different from those for the current standard air quality scenario than are differences noted above for the comparison-to-benchmarks estimates. This is due to the greater influence on the risk results of exposures associated with the low O_3 concentrations that are less affected by air quality adjustments used to develop air concentration surfaces for which the highest-concentration location has a design value just meeting the different targets.

3.4.4 Key Uncertainties

In this section, we consider the uncertainties associated with the quantitative estimates of exposure and risk, including those recognized by the characterization of uncertainty in Appendix 3D (section 3D.3.4). This characterization is based on an approach intended to identify and compare the relative impact that important sources of uncertainty may have on the exposure and risk estimates. The approach used has been applied in REAs for past NAAQS reviews for O_3, nitrogen oxides, carbon monoxide and SO_X (U.S. EPA, 2008; U.S. EPA, 2010a; U.S. EPA, 2014; U.S. EPA, 2018). The characterization of uncertainty for the current analyses utilized a largely qualitative approach adapted from the World Health Organization (WHO) approach for characterizing uncertainty in exposure assessment (WHO, 2008) accompanied by several quantitative sensitivity analyses of key aspects of the assessment approach. This current uncertainty characterization and the supporting quantitative sensitivity analyses described in detail in Appendix 3D further build upon information generated from quantitative sensitivity analyses analysis conducted for the 2014 HREA and a previously conducted quantitative uncertainty analysis of the population-based exposure modeling performed for the O_3 NAAQS (Langstaff, 2007). The approach used varies from that of WHO (2008) in that the approach placed a greater focus on evaluating the direction and the magnitude of the uncertainty (i.e., qualitatively rating how the source of uncertainty, in the presence of alternative improved information or directly based on quantitative sensitivity analyses, may affect the estimated exposures and health risk estimates).

The exposure and risk uncertainty characterization and quantitative sensitivity analyses, presented in Appendix 3D, section 3D.3.4, involve consideration of the various types of inputs and approaches that together result in the exposure and risk estimates for the eight study areas. In

this way the limitations and uncertainties underlying these inputs and approaches and the extent of their influence on the resultant exposure/risk estimates are considered. Consistent with the WHO (2008) guidance, the overall impact of the uncertainty is scaled by considering the extent or magnitude of the impact of the uncertainty as implied by the relationship between the source of the uncertainty and the exposure and risk output. The characterization in Appendix 3D also evaluated the direction of influence, indicating how the source of uncertainty was judged, or found, to quantitatively affect the exposure and risk estimates, e.g., likely to over- or under-estimate (Appendix 3D, section 3D.3.4.1).

- **What are the important uncertainties associated with the exposure and risk estimates?**

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

An overarching and important area of uncertainty, which remains from the last review and is important to our consideration of the exposure and risk analysis results, concerns the extent to which the outcomes of the exposure and risk analysis represent important risks posed by O_3 present in ambient air under conditions meeting the current standard. As an initial matter, we recognize that this analysis addresses an array of respiratory responses documented in the controlled human exposure studies of 6.6-hour duration (e.g., lung function decrements, respiratory symptoms, increased airway responsiveness and inflammation). The comparison-to-benchmarks analysis is particularly focused on consideration of the potential for exposures that pose a risk of experiencing such effects. We note, however, the lack of evidence from controlled human exposure studies of 6.6-hour duration for people with asthma and the three benchmark concentrations. As recognized in sections 3.3.1 and 3.3.4, the controlled human exposure study evidence base for 6.6-hour studies of 60, 70 and 80 ppb does not include studies of people with asthma or children. Although uncertainties remain, the limited evidence that informs our understanding of risk to people with asthma indicates the potential for them to be at greater risk relative to other population groups under similar exposure circumstances (e.g., of asthma exacerbation), as summarized in section 3.3.4 above. Thus, the health effects documented in controlled human exposure studies of healthy adults may contribute to more severe outcomes when occurring in people with asthma. Such a conclusion is consistent with the epidemiological

study findings of positive associations of O_3 concentrations with asthma-related ED visits and hospital admissions (and the higher effect estimates from these studies), as referenced in section 3.3.1 above and presented in detail in the ISA. Further, with regard to lung function decrements, we note the lack of information on the factors contributing to increased susceptibility to O_3-induced lung function decrements among some people. Thus, there is uncertainty regarding the interpretation of the exposure and risk estimates and the extent to which they represent the populations at greatest risk of O_3-related respiratory effects.

Aspects of the analytical design that pertain to both exposure-based risk metrics include the estimation of ambient air concentrations for the assessed scenarios, as well as the main components of the exposure modeling. Key uncertainties identified include the ambient air concentrations used in developing the ambient air quality data input to the exposure model, along with the modeling approach used to adjust ambient air concentrations to meet the air quality scenarios of interest and the method used to interpolate monitor concentrations to census tracts. While we recognize the adjustment to conditions near, above, or just below the current standard as an important area of uncertainty, the approach used has taken into account the currently available information and selected study areas having design values near the level of the current standard to minimize the size of the adjustment needed to meet a given air quality scenario, along with the use of more recent data as inputs for the air quality modeling, such as more recent O_3 concentration data (2015-2017), meteorological data (2016) and emissions data (2016). Further, we consider the number of ambient monitors sited in each of the eight study areas to contribute to a reasonable representation of spatial and temporal variability in the eight study areas for the air quality conditions simulated. Among other key areas, we additionally recognize the uncertainty with regard to the simulation of study area populations (and at-risk populations) and considering appropriate physical and personal attributes. As recognized in the 2014 HREA, exposures could be underestimated for some population groups that are frequently and routinely outdoors during the summer (e.g., outdoor workers, children). In addition, longitudinal activity patterns do not exist for these and other important population groups (e.g., those having respiratory conditions other than asthma), thus limiting the extent to which the exposure model outputs reflect these groups that might routinely experience high exposure concentrations. We recognize there are important uncertainties in the approach used to estimate energy expenditure (i.e., metabolic equivalents of work or METs) which are ultimately used to estimate ventilation rates. We consider the use of longer-term average MET distributions to derive short-term estimates, along with extrapolating adult observations to children is reasonable, based on the availability of relevant data and appropriate evaluations conducted to date. We note, however, that the number of activities for which METs distributions are available has more than doubled

since the last review and the added specificity and redevelopment of these distributions is expected to more realistically estimate activity-specific energy expenditure.

With regard to the exposure and risk modeling aspects of the two risk metrics, we recognize that there are some uncertainties that apply to the estimation of lung function risk (and not related to the comparison-to-benchmarks analysis). Both of the lung function risk approaches utilized in the risk analyses incorporate some degree of extrapolation beyond the exposure circumstances that have been studied in the controlled human exposure studies. This is the case in different ways and with differing impacts for the two approaches. One way in which both approaches extrapolate beyond the exposure studies concerns estimates of lung function risk derived for exposure concentrations below those represented in the evidence base. This is in recognition of the potential for lung function decrements to be greater in unstudied at-risk population groups than is evident from the available studies. In considering these risk estimates, we recognize that the uncertainty in the response estimates likely increases with decreasing exposure concentration below those evaluated in controlled exposure studies.

The two models differ in how they extrapolate beyond the exposure study conditions. In recognition of the lack of data for some at risk groups and the potential for such groups, such as children with asthma, to experience lung function decrements at lower exposures than healthy adults, both models generate nonzero predictions for 7-hour concentrations below the 6.6-hour concentrations investigated in the controlled human exposure studies. For example, the E-R function risk approach generates nonzero predictions from the full range of potential nonzero concentrations for 7-hour average durations for which the average exertion levels meets or exceeds the target. The MSS model, which draws on evidence-based concepts of how human physiological processes respond to O_3, involves extrapolation beyond the controlled experimental conditions, with regard to exposure concentration, as well as with regard to exposure duration and ventilation rate (both magnitude and duration). The difference between the two models in the extent of extrapolation beyond the studied exposure circumstances is illustrated by differences in the percent of the risk estimates derived on days for which the highest 7-hour average concentration is below the lowest 6.6-hour exposure concentration tested (Table 3-6 and Table 3-7). For example, while 3 to 6% of the risk to children (based on single-year estimates for three study areas) of experiencing at least one day with decrements greater than 20% estimated by the E-R model is associated with exposure concentrations below 40 ppb (the lowest exposure concentration studied, and at which no decrements of this severity occurred in any study subjects), 25% to nearly 40% of MSS model estimates of decrements greater than 20% derive from exposures below 40 ppb (Table 3-6 and Table 3-7). Further, using ventilation rates lower than those used for the E-R function risk approach (which are based on the controlled human exposure study conditions) also contribute to relatively greater risks estimated by the

MSS model. Limiting the MSS model results to estimates for individuals with at least the same exertion level achieved by study subjects (≥17.3 L/min-m^2), reduces the risks of experiencing at least one lung function decrement by an amount between 24 to 42% (Appendix 3D, Table 3D-69).

The difference between the two models for risk contribution from low concentrations is smaller for risk estimates for two or more days than the estimates for one or more days. This is largely because the percent contribution to low-concentration risk for two or more decrement days predicted by the E-R approach is, by design, greater than the corresponding contribution to low-concentration risk for one or more days.[80] This also occurs because the MSS model estimates risk from a larger variety of exposure and ventilation conditions (Table 3-6 and Table 3-7). Further, many of the uncertainties previously identified as part of the 2014 HREA unique to the MSS model remain as important uncertainties in the current assessment. For example, the extrapolation of the MSS model age parameter down to age 5 (from the age range of 18- to 35-year old study subjects to which the model was fit) is an important uncertainty given that children are an at-risk population of particular interest in this assessment. Also, there is uncertainty in estimating the frequency and magnitude of lung function decrements as a result of the statistical form and parameters used for the MSS model inter- and intra-individual variability terms. Each of these, among other newly identified MSS model uncertainties, are evaluated and discussed in the current uncertainty characterization (Appendix 3D, section 3D.3.4). As a whole, the differences between the two lung function risk approaches described above and the estimates generated by these approaches indicate appreciably greater uncertainty associated with the MSS model estimates than the E-R function estimates due to the significantly greater portion of relatively low concentrations contributing to risk.

[80] 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.

Table 3-6. Percent of risk estimated for air quality just meeting the current standard in three study areas using the E-R function approach on days where the daily 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).

Table 3-7. Percent of risk estimated for air quality just meeting the current standard in three study areas using the MSS model approach on days where the daily 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).

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

In summary, among the multiple uncertainties and limitations in data and tools that affect the quantitative estimates of exposure and risk and their interpretation in the context of considering the current standard, we recognize several here as particularly important, noting that some of these uncertainties are similar to those recognized in the last review. These include

uncertainty related to estimation of the concentrations in ambient air for the current standard and the additional air quality scenarios; lung function risk approaches that rely, to varying extents, on extrapolating from controlled human exposure study conditions to lower exposure concentrations, lower ventilation rates, and shorter durations; and, characterization of risk for particular population groups that may be at greatest risk, particularly for people with asthma, particularly children. We also recognize several areas in which uncertainty has been reduced by new or updated information or methods, including more refined air quality modeling based on selection of study areas with design values near the current standard and more recent model inputs, as well as updates to several inputs to the exposure model including changes to the exposure duration to better match those in the controlled human exposure studies and an alternate approach to characterizing periods of activity while moderate or greater exertion for simulated individuals.

3.4.5 Public Health Implications

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

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

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

In considering the severity of responses associated with the exposure and risk estimates, we take note of the health effects evidence for the different benchmark concentrations and judgments made with regard to the severity of these effects in the last review. As in the last review, we recognize the greater prevalence of more severe lung function decrements among study subjects exposed to 80 ppb or higher concentrations compared to 60 or 70 ppb exposure concentrations, as well as the prevalence of other effects such as respiratory symptoms; thus, such exposures are appropriately considered to be associated with adverse respiratory effects consistent with past and recent ATS position statements. At 70 ppb, statistically significant

increases in lung function decrements (specifically reduced FEV_1) and respiratory symptoms have been reported, which has led to characterization of these exposure conditions as also being associated with adverse responses, consistent with past ATS statements as summarized in section 3.1 above (e.g., 80 FR 65343, 65345, October 26, 2015). Studies of controlled human exposures to the lowest benchmark concentration of 60 ppb have found small but statistically significant O_3-related decrements in lung function and airway inflammation.

We additionally take note of the greater significance of estimates for multiple occurrences of exposures at or above these benchmarks consistent with the evidence. This is consistent with past O_3 NAAQS reviews in which it was recognized, using the example of effects such as inflammation, that while isolated occurrences can resolve entirely, repeated occurrences from repeated exposure could potentially result in more severe effects (2013 ISA, section 6.2.3 and p. 6-76). The ascribing of greater significance to repeated occurrences of exposures of potential concern is also consistent with public health judgments in NAAQS reviews for other pollutants, such as SO_X and carbon monoxide (84 FR 9900, March 18, 2019; 76 FR 54307, August 31, 2011).

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

In light of the conclusions that people with asthma and children are at-risk populations for O_3-related health effects (summarized in section 3.3.2 above) and the exposure and risk analysis findings of higher exposures and risks for children (in terms of percent of that population), we have focused the discussion here on children, and specifically children with asthma. We recognize that the exposure and risk estimates indicate that in some areas of the U.S. where O_3 concentrations just meet the current standard, on average across the 3-year period simulated, less than 1%, and less than 0.1% of the simulated population of children with asthma might be expected to experience a single day per year with a 7-hour exposure at or above 70 ppb and 80 ppb, respectively, while breathing at an elevated rate. With regard to the lowest benchmark considered (60 ppb), the corresponding percentage is less than approximately 9%, with higher percentages in some individual years. The corresponding estimates for the air quality scenario with higher O_3 concentrations are notably higher (Table 3-5). For example, for the 75

ppb air quality scenario, 1.1% to 2.1% of children with asthma, on average across the 3-year design period, are estimated to experience at least one day with exposure concentrations at or above 70 ppb, while at moderate or greater exertion, with as many as 3.9% in a single year (Table 3-5). The estimates for the 65 ppb scenario are appreciably lower.

With regard to estimates of lung function decrements, we focus on the E-R model estimates as having less associated uncertainty, as discussed in section 3.4.4 above. The exposure and risk analysis estimates 0.2 to 0.3% of children with asthma, on average across the 3-year design period to experience one or more days with a lung function decrement at or above 20%, and 0.5 to 0.9 % to experience one or more days with a decrement at or above 15% (Table 3-4 above). In a single year, the highest estimate is 1.0% of this at-risk population expected to experience one or more days with a decrement at or above 15%. The corresponding estimate for two or more days is 0.6% (Table 3-4 above). As discussed in section 3.4.3 above, the estimates for the 75 ppb air quality scenario are notably higher, while the estimates for the 65 ppb scenario are notably lower (Table 3-5).

The size of the at-risk population (people with asthma, particularly children) in the U.S. is substantial. As summarized in section 3.3.2, nearly 8% of the total U.S. population[81]and 8.4% of U.S. children have asthma. The asthma prevalence in U.S. child populations (younger than 18 years) of different races or ethnicities ranges from 6.2% for Hispanic, Mexican or Mexican-American children to 12.6% for black non-Hispanic children (Table 3-1 above). This is well reflected in the exposure and risk analysis study areas in which the asthma prevalence ranged from 7.7% to 11.2% of the total populations and 9.2% to 12.3% of the children. In each study area, the prevalence varies among census tracts, with the highest tract having a prevalence in boys of 25.5% and a prevalence in girls of 17.1% (Appendix 3D, Table 3D-3).

The exposure and risk analyses inherently recognize that variability in human activity patterns (where people go and what they do) is key to understanding the magnitude, duration, pattern, and frequency of population exposures. For O_3 in particular, the amount and frequency of afternoon time outdoors at moderate or greater exertion is an important factor for understanding the fraction of the population that might experience O_3 exposures that have elicited respiratory effects in controlled human exposure studies (2014 HREA, section 5.4.2). In considering the available information regarding prevalence of behavior (time outdoors and exertion levels) and daily temporal pattern of O_3 concentrations, we take note of the findings of evaluations of the data in the CHAD. Based on these evaluations of human activity pattern data,

[81] 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 25,191,000 in 2017. The updated estimate from the 2018 National Health Interview Survey is 24,753,000 (CDC, 2020). For children (younger than 18 years), the 2017 estimate is approximately 6,182,000 (Table 3-1), while the estimate for 2018 is slightly lower at 5,530,131.

it appears that children and adults both, on average, spend about 2 hours of afternoon time outdoors per day, but differ substantially in their participation in these events at elevated exertion levels (rates of about 80% versus 60%, respectively) (2014 HREA, section 5.4.1.5), indicating children are more likely to experience exposures that may be of concern. This is one basis for their identification as an at-risk population for O_3-related health effects. The human activity pattern evaluations have also shown there is little to no difference in the amount or frequency of afternoon time outdoors at moderate or greater exertion for people with asthma compared with those who do not have asthma (2014 HREA, section 5.4.1.5). Further, recent CHAD analyses indicate that while 46 – 73% of people do not spend any afternoon time outdoors at moderate or greater exertion, a fraction of the population (i.e., between 5.5 – 6.8% of children) spend more than 4 hours per day outdoors at moderate or greater exertion and may have greater potential to experience exposure events of concern than adults (Appendix 3D, section 3D.2.5.3 and Figure 3D-9). It is this potential that contributes importance to consideration of the exposure and risk estimates.

In considering the public health implications of the exposure and risk estimates across the eight study areas, we note the purpose for the study areas is to illustrate exposure circumstances that may occur in areas that just meet the current standard, and not to estimate exposure and risk associated with conditions occurring in those specific locations today. To the extent that concentrations in the specific areas simulated may differ from others across the U.S., the exposure and risk estimates for these areas are informative to consideration of potential exposures and risks in areas existing across the U.S. that have air quality and population characteristics similar to the study areas assessed, and that have ambient concentrations of O_3 that just meet the current standard today or that will be reduced to do so at some period in the future. We note that numerous areas across the U.S. have air quality for O_3 that is near or above the existing standard.[82] Thus, the air quality and exposure circumstances assessed in the eight study areas are of particular importance in considering whether the currently available information calls into question the adequacy of public health protection afforded by the current standard.

The exposure and risk estimates for the study areas assessed for this review reflect differences in exposure circumstances among those areas and illustrate the exposures and risks that might be expected to occur in other areas with such circumstances under air quality

[82] Based on the most recently available data from 2016-2018, 142 counties have O_3 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*).

conditions that just meet the current standard (or the alternate conditions assessed). Thus, the exposure and risk estimates indicate the magnitude of exposure and risk that might be expected in many areas of the U.S. with O_3 concentrations at or near the current standard. Although the methodologies and data used to estimate population exposure and lung function risk in this review differ in several ways from what was used in the last review, the findings and considerations summarized here present a pattern of exposure and risk that is generally similar to that considered in the last review (as described in section 3.4.2 above), and indicate a level of protection generally consistent with that described in the 2015 decision.

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

3.5 KEY CONSIDERATIONS REGARDING THE CURRENT PRIMARY STANDARD

In considering what the currently available evidence and exposure/risk information indicate with regard to the current primary O_3 standard, the overarching question we consider is:

- **Does the currently available scientific evidence- and exposure/risk-based information support or call into question the adequacy of the protection afforded by the current primary O_3 standard?**

To assist us in interpreting the currently available scientific evidence and the results of recent quantitative exposure/risk analyses to address this question, we have focused on a series of more specific questions, as detailed in sections 3.5.1 and 3.5.2 below. In considering the scientific and technical information, we consider both the information available at the time of the last review and information newly available in this review, which have both been critically analyzed and characterized in the 2013 ISA for the last review and the ISA for the current review, respectively. In so doing, a primary consideration is whether the information newly available in this review alters our overall conclusions from the last review regarding health effects associated with photochemical oxidants, including O_3, in ambient air.

3.5.1 Evidence-based Considerations

In considering the evidence with regard to the overarching question posed above regarding the adequacy of the current standard, we address a series of more specific questions that focus on policy-relevant aspects of the evidence. These questions begin with consideration of the available evidence on health effects associated with exposure to photochemical oxidants, and particularly O_3.

- **Is there newly available evidence that indicates the importance of photochemical oxidants other than O_3 with regard to abundance in ambient air, and potential for human exposures and health effects?**

No newly available evidence has been identified in this review regarding the importance of photochemical oxidants other than O_3 with regard to abundance in ambient air, and potential for health effects.[83] As summarized in section 2.1 above, O_3 is one of a group of photochemical oxidants formed by atmospheric photochemical reactions of hydrocarbons with nitrogen oxides in the presence of sunlight, with O_3 being the only photochemical oxidant other than nitrogen dioxide that is routinely monitored in ambient air. Data for other photochemical oxidants are generally derived from a few special field studies such that national scale data for these other oxidants are scarce (ISA, Appendix 1, section 1.1; 2013 ISA, sections 3.1 and 3.6). Moreover, few studies of the health impacts of other photochemical oxidants beyond O_3 have been identified by literature searches conducted for other recent O_3 assessments (ISA, Appendix 1, section 1.1). As stated in the ISA, "the primary literature evaluating the health…effects of photochemical oxidants includes ozone almost exclusively as an indicator of photochemical oxidants" (ISA, section IS.1.1, p. IS-3). Thus, as was the case for previous reviews, the evidence base for health effects of photochemical oxidants does not indicate an importance of any other photochemical oxidants. For these reasons, discussion of photochemical oxidants in this document focuses on O_3.

- **Does the currently available scientific evidence alter our conclusions from the last review regarding the nature of health effects attributable to human exposure to O_3 from ambient air?**

The currently available evidence, including that newly available in this review, is largely consistent with the conclusion reached in the last review regarding health effects causally related to O_3 exposures, specifically respiratory effects. Specifically, as in the last review, respiratory effects are concluded to be causally related to short-term exposures to O_3. Also, as in the last

[83] Close agreement between past O_3 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 O_3 (DHEW, 1969).

review, respiratory effects are concluded to be likely causally related to longer-term O_3 exposures (ISA, section IS.1.3.1, Appendix 3). Further, while a causal determination was not made in the last review regarding metabolic effects, the ISA for this review finds there to be sufficient evidence to concluded there to likely be a causal relationship of short-term O_3 exposures and metabolic effects and finds the evidence to be suggestive of, but not sufficient to infer, such a relationship between long-term O_3 exposure and metabolic effects (ISA, section IS.1.3.1). This is based on evidence on these effects, largely from experimental animal studies, that is newly available in this review (ISA, Appendix 5). Additionally, conclusions reached in the current review differ with regard to cardiovascular effects and mortality, based on newly available evidence in combination with uncertainties in the previously available evidence that had been identified in the last review (ISA, Appendix 4, section 4.1.17 and Appendix 6, section 6.1.8). The current evidence base is concluded to be suggestive of, but not sufficient to infer, causal relationships between O_3 exposures (short- and long-term) and cardiovascular effects, mortality, reproductive and developmental effects, and nervous system effects (ISA, section IS.1.3.1). As in the last review, the strongest evidence, including with regard to characterization of relationships between O_3 exposure and occurrence and magnitude of effects, is for respiratory effects, and particularly for effects such as lung function decrements, respiratory symptoms, airway responsiveness, and respiratory inflammation.

- **Does the current evidence alter our understanding of populations that are particularly at risk from O_3 exposures?**

The current evidence does not alter our understanding of populations at risk from health effects of O_3 exposures. As in the last review, people with asthma, and particularly children, are the at-risk population groups for which the evidence is strongest. In addition to populations with asthma, groups with relatively greater exposures, particularly those who spend more time outdoors during times when ambient air concentrations of O_3 are highest and while engaged in activities that result in elevated ventilation, are recognized as at increased risk. Such groups include outdoor workers and children. Other groups for which the recent evidence is less clear include older adults, and recent evidence regarding individuals with reduced intake of certain nutrients and individuals with certain genetic variants does not provide additional information for these groups beyond the evidence available at the time of the last review (ISA, section IS.4.4).

- **Does the current evidence alter our conclusions from the previous review regarding the exposure duration and concentrations associated with health effects? To what extent does the currently available scientific evidence indicate health effects**

attributable to exposures to O_3 concentrations lower than previously reported and what are important uncertainties in that evidence?

The currently available evidence regarding O_3 exposures associated with health effects is largely similar to that available at the time of the last review and does not indicate effects attributable to exposures of shorter duration or lower concentrations than previously understood. Respiratory effects continue to be the effects for which the experimental information regarding exposure concentrations eliciting effects is well established, as summarized in section 3.3.3 above. Such information allows for characterization of potential population risk associated with O_3 in ambient air under conditions allowed by the current standard. The newly available controlled human exposure studies, as discussed in section 3.3.3 above, are conducted over shorter durations while at much higher concentrations than the key set of 6.6-hour studies that have been the focus of the last several reviews. The respiratory effects evidence includes support from a large number of epidemiologic studies. The positive associations of O_3 with respiratory health outcomes (e.g., asthma-related hospital admissions and emergency department visits) reported in these studies are coherent with findings from the controlled human exposure and experimental animal studies. All but a few of these studies, however, are conducted in areas during periods when the current standard is not met, making them less useful with regard to indication of effects of exposures allowed by the current standard.

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

Thus, the 6.6-hour controlled human exposure studies of respiratory effects remain the focus for our consideration of exposure circumstances associated with O_3 health effects. Based on these studies, the exposure concentrations investigated range from as low as approximately 40 ppb to 120 ppb. This information on concentrations that have been found to elicit effects for 6.6-hour exposures while exercising is unchanged from what was available in the last review. The lowest concentration for which lung function decrements have been found to be statistically significantly increased over responses to filtered air remains approximately 60 ppb, at which group mean decrements on the order of 2% to 4% have been reported (Table 3-2 and Figure 3-2). Respiratory symptoms were not increased with this exposure level.[84] Exposure to concentrations slightly above 70 ppb, with quasi-continuous exercise, has been reported to elicit statistically

[84] A 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 and Figure 3-2; Appendix 3A). An increase in respiratory symptoms has not been reported with this exposure level.

significant increases in both lung function decrements and respiratory symptom scores, as summarized in section 3.3.3 above. Still greater group mean and individual responses in lung function decrements and respiratory symptom scores, as well as inflammatory response and airway responsiveness, are reported for higher exposure concentrations.

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

Uncertainties identified in the health effects evidence at the time of the last review generally remain in the current evidence. These include uncertainties related to the susceptibility of population groups not studied, the potential for effects to result from exposures to concentrations below those included in controlled human exposure studies, and the potential for increased susceptibility as a result of prior exposures. We additionally recognize uncertainties associated with the epidemiologic studies (e.g., the potential for copollutant confounding and exposure measurement error). In so doing, however, we note the appreciably greater strength in the epidemiologic evidence in its support for determination of a causal relationship for respiratory effects than that related to other categories, such as metabolic effects, newly determined in this review to have a likely causal relationship with short-term O_3 exposures (as summarized in section 3.3.1 above).

3.5.2 Exposure/risk-based Considerations

Our consideration of the scientific evidence available in the current review, as at the time of the last review, is informed by results from a quantitative analysis of estimated population exposure and associated risk. The overarching consideration in this section is whether the current exposure/risk information alters our overall conclusions from the previous review regarding health risk associated with exposure to O_3 in ambient air. As in our consideration of the evidence in section 3.3.1 above, we have focused the discussion regarding the exposure/risk information around key questions to assist us in considering the exposure/risk analyses of at-risk populations living in a set of urban areas under air quality conditions simulated to just meet the existing primary O_3 standard. These questions are as follows.

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

The exposure and risk analyses conducted for this review provide exposure and risk estimates associated with air quality that might occur in an area under conditions that just meet the current standard and, in so doing, they illustrate the differences likely to occur across various locations with such air quality as a result of area-specific differences in emissions,

meteorological and population characteristics. In understanding these results, we note that the eight study areas provide a variety of circumstances with regard to population exposure to concentrations of O_3 in ambient air. These study areas reflect different combinations of different types of sources of O_3 precursor emissions, and also illustrate different patterns of exposure to O_3 concentrations in a populated area in the U.S. (Appendix 3C, section 3C.2). In this way, the eight areas provide a variety of examples of exposure patterns that can be informative to the EPA's consideration of potential exposures and risks that may be associated with air quality conditions occurring under the current O_3 standard. While the same conceptual air quality scenario is simulated in all eight study areas (i.e., conditions that just meet the existing standard), variability in emissions patterns of O_3 precursors, meteorological conditions, and population characteristics in the study areas contribute to variability in the estimated magnitude of exposure and associated risk across study areas.

In considering the exposure and risk results, we focus first on estimates for the eight study areas from the comparison-to-benchmarks analysis, the results in which we have the greatest confidence, as discussed in section 3.4.4 above. These results for urban areas with air quality that just meets the current standard indicate that as many as 0.7% of children with asthma, on average across the 3-year period, and up to 1.0% in a single year might be expected to experience, while at elevated exertion, at least one day with a 7-hour average O_3 exposure concentration at or above 70 ppb (Table 3-3). As noted earlier, this benchmark concentration reflects the finding of statistically significant O_3-related decrements and increased respiratory symptoms in a controlled human exposure study of individuals at elevated exertion. For the benchmark concentration of 80 ppb (which reflects the potential for more severe effects), a much lower percentage (0.1%) of children with asthma, on average across the 3-year period or in any single year, might be expected to experience, while at elevated exertion, at least one day with such a concentration (Table 3-3). By comparison, as many as 9% of children with asthma, on average across the 3-year period, might be expected to experience one or more days with a 7-hour average O_3 exposure concentration at or above 60 ppb (the benchmark associated with less severe effects), and just over 11% in a single year (Table 3-3). Regarding estimates for multiple days, the percent of children with asthma estimated to experience two or more days with an exposure at or above 70 ppb is less than 0.1%, on average across three years, and up to 0.1% in a single year period (Table 3-3). There are no children with asthma estimated to experience more than a single day per year with a 7-hour average O_3 concentration at or above 80 ppb (Table 3-3). With regard to the lowest benchmark concentration of 60 ppb, the percentages for more than a single day occurrence are 3%, on average across the three years, and just below 5% in a single year period (Table 3-3).

The estimates for the additional air quality scenarios differ as would be expected. For the 75 ppb air quality scenario, the percent of children with asthma that might be expected to experience at least one day with a 7-hour average O_3 exposure concentration, while at elevated exertion, at or above 70 ppb, is a factor of three or more higher than for the current standard (Table 3-5). The corresponding estimates for multiple days are a factor of four or more higher than those for air quality just meeting the current standard. By comparison, corresponding estimates for the 65 ppb scenario are approximately a third those for the current standard scenario, with a correspondingly smaller incremental difference in absolute number of children . With regard to the 80 ppb benchmark, the difference of the 75 ppb scenario from the current standard is a factor of three (for average across the 3-year period) to six (for the highest in a single year). In contrast, the estimates for the 80 ppb benchmark (which is associated with the more severe effects) in the 65 ppb air quality scenario are nearly identical to those for the current standard.

With regard to the estimates of lung function risk, as an initial matter we note the uncertainty associated with these estimates, as discussed in section 3.4.4 above. In so doing, however, we recognize the lesser uncertainty associated with estimates derived using the E-R function. Accordingly, we focus on those estimates here for air quality conditions just meeting the current standard. The E-R lung function risk analysis for the eight study areas indicates that the percent of children with asthma in an urban area that just meets the current standard that might be expected to experience one or more days with a lung function decrement of at least 15% or 20% may be as high as 0.9% or 0.3%, respectively, on average across the three years, and 1.0% or 0.4%, respectively, in a single-year period (Table 3-4). The estimates for a day with a decrement of at least 10% may be as high as 3.3%, on average across the three years, and just over 3.5% in a single-year period (Table 3-4). With regard to multiple day occurrences, the percent of children with asthma that might be expected to experience two or more days with a lung function decrement of at least 10% may be as high as 2.4%, on average across the three years, and 2.6% in a single year (Table 3-4), with much smaller percentages for larger decrements. For multiple days with a decrement of at least 15% or 20%, the percentages may be as high as 0.6% or 0.2%, respectively, on average across the three years, and 0.6% or 0.2%, respectively, in a single year period (Table 3-4).

We also consider the estimates from this assessment in light of the estimates from the 2014 HREA that were a focus of the decision on the standard in 2015. The estimates across all study areas from this assessment are generally similar to those reported in the last review across all study areas included in that HREA, particularly for the two or more occurrences and for the

80 ppb benchmark (Table 3-8).[85] In our consideration here, we focus on the full array of study areas (e.g., rather than limiting to areas common to the two assessments) given the purpose of the assessments in providing estimates across a range of study areas to inform decision making with regard to the exposures and risks that may occur across the U.S. in areas that just meet the current standard. In so doing, we note only slight differences observed for the lower benchmarks, particularly in the estimates for the highest year. For example, consideration of the percentage of children estimated to experience a day or more with an exposure at or above 70 ppb across the three air quality conditions in the two assessments indicates that differences between air quality scenarios in the current assessment remain appreciably larger than the slight differences in estimates between the two assessments for a given scenario. The factors likely contributing to the slight differences between the two assessments, such as for the lowest benchmark, include greater variation in ambient air concentrations in some of the study areas in the 2014 HREA, as well as the lesser air quality adjustments required in study areas for the current assessment due to closer proximity of conditions to meeting the current standard (70 ppb).[86] Other important differences between the two assessments are the updates made to the ventilation rates used for identifying when a simulated individual is at moderate or greater exertion and the use of 7 hours for the exposure duration. Both of these changes were made to provide closer linkages to the conditions of the controlled human exposure studies which are the basis for the benchmark concentrations. Thus, we recognize there to be reduced uncertainty associated with the current estimates. Overall, particularly in light of differences in the assessments, we conclude the current estimates to be generally similar to those which were the focus in the 2015 decision on establishing the current standard.

[85] 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 focus in section 3.4).

[86] 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.

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 (DV, ppb)	Estimated average % of simulated children with at least one day per year at or above benchmark (highest in single season)		Estimated average % of simulated children with at least two days per year at or above benchmark (highest in single season)	
	Current PA [A]	*2014 HREA* [B]	*Current PA* [A]	*2014 HREA* [B]
Benchmark Exposure Concentration of 80 ppb				
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)
Benchmark Exposure Concentration of 70 ppb				
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)
Benchmark Exposure Concentration of 60 ppb				
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".

3.5.3 CASAC Advice

In our consideration of the adequacy of the current primary O_3 standard, in addition to the evidence- and exposure and risk-based information discussed above, we have also considered the advice and recommendations of the CASAC with regard to the adequacy of the current standard, based on their review of the ISA and the earlier draft of this document, as well as comments from the public on the earlier draft of this document.

A limited number of public comments have been received in this review to date, including comments focused on the draft IRP or draft PA. Of the 11 commenters that addressed adequacy of the current primary O_3 standard, some expressed agreement with staff conclusions in the draft PA, while others expressed the view that the standard should be more restrictive based largely on advice from and considerations raised by the previous CASAC in the last review regarding adequacy of the margin of safety.

In the CASAC's review of the PA with regard to the primary standard, it agreed with the draft PA that the evidence newly available in this review does not substantially differ from that available in the 2015 review, stating that , "[t]he CASAC agrees that the evidence newly available in this review that is relevant to setting the ozone standard does not substantially differ from that of the 2015 Ozone NAAQS review" (Cox, 2020Cox, 2020, p. 12 of the Consensus Responses). Further, some CASAC members "agree with the EPA that the available evidence does not call into question the adequacy of protection provided by the current standard, and thus support retaining the current primary standard" (Cox, 2020, p. 1 of letter). Other members indicated their agreement with the previous CASAC's advice, based on review of the 2014 draft PA, that a primary standard set at 70 ppb may not be protective of public health with an adequate margin of safety (Cox, 2020, p. 1 of letter).[87]

The comments from the CASAC also included comments related to the quantitative analyses summarized in section 3.4 above. These comments have been considered in completing the analyses and associated characterizations in Appendices 3C and 3D. The CASAC additionally took note of uncertainties that remain in this review of the primary standard. Accordingly, it identified a number of additional areas for future research and data gathering that would inform the next review of the primary O_3 NAAQS (Cox, 2020, p. 14 of the Consensus Responses). These are reflected in section 3.6 below.

3.5.4 Conclusions on the Primary Standard

This section describes our conclusions for the Administrator's consideration in this review of the current primary O_3 standard. These conclusions are based on considerations described in the sections above, and in the discussion below regarding the currently available scientific evidence (as summarized in the ISA, and the ISA and AQCDs from prior reviews), and the risk and exposure information summarized above in chapter 3. Further, these conclusions have taken into account advice from the CASAC and public comment on the draft PA.

Taking into consideration the discussions responding to specific questions above in this and the prior chapter, this section addresses the following overarching policy question.

[87] In the last review, the advice from the prior CASAC included a range of recommended levels for the standard, with the CASAC concluding that "there is adequate scientific evidence to recommend a range of levels for a revised primary ozone standard from 70 ppb to 60 ppb" (Frey, 2014, p. ii). In so doing, the prior CASAC noted that "[i]n reaching its scientific judgment regarding a recommended range of levels for a revised ozone primary standard, the CASAC focused on the scientific evidence that identifies the type and extent of adverse effects on public health" and further acknowledged "that the choice of a level within the range recommended based on scientific evidence is a policy judgment under the statutory mandate of the Clean Air Act" (Frey, 2014, p. ii). The prior CASAC then described that its "*policy advice* [emphasis added] is to set the level of the standard lower than 70 ppb within a range down to 60 ppb, taking into account [the Administrator's] judgment regarding the desired margin of safety to protect public health, and taking into account that lower levels will provide incrementally greater margins of safety" (Frey, 2014, p. ii).

- **Does the currently available scientific evidence- and exposure/risk-based information support or call into question the adequacy of the protection afforded by the current primary O_3 standard?**

In considering this question, we recognize that, as is the case in NAAQS reviews in general, the extent to which the current primary O_3 standard is judged to be adequate will depend on a variety of factors, including science policy judgments and public health policy judgments to be made by the Administrator. These factors include public health policy judgments concerning the appropriate benchmark concentrations on which to place weight, as well as judgments on the public health significance of the effects that have been observed at the exposures evaluated in the health effects evidence. The factors relevant to judging the adequacy of the standards also include the interpretation of, and decisions as to the weight to place on, different aspects of the results of the exposure assessment for the eight areas studied and the associated uncertainties. Thus, we recognize that the Administrator's conclusions regarding the adequacy of the current standard will depend in part on public health policy judgments, science policy judgments regarding aspects of the evidence and exposure/risk estimates, and judgments about the degree of protection that is requisite to protect public health with an adequate margin of safety.

Our response to the overarching question above takes into consideration the discussions that address the specific policy-relevant questions in prior sections of this document (see section 3.2) and builds on the approach from the last review (summarized in section 3.1 above). We focus first on consideration of the evidence, including that newly available in this review, and the extent to which it alters key conclusions supporting the current standard. We then turn to consideration of the quantitative exposure and risk estimates developed in this review, including associated limitations and uncertainties, and the extent to which they indicate differing conclusions regarding the magnitude of risk, as well as level of protection from adverse effects, associated with the current standard. We additionally consider the key aspects of the evidence and exposure/risk estimates emphasized in establishing the now-current standard, and the associated public health policy judgments and judgments about the uncertainties inherent in the scientific evidence and quantitative analyses that are integral to decisions on the adequacy of the current primary O_3 standard.

As an initial matter, we recognize the continued support in the current evidence for O_3 as the indicator for photochemical oxidants, as recognized in section 3.5.1 above. Of the photochemical oxidants, O_3 is the only one other than nitrogen dioxide (for which there are separate NAAQS) that is routinely monitored in ambient air. Further, as stated in the ISA, "the primary literature evaluating the health and ecological effects of photochemical oxidants includes ozone almost exclusively as an indicator of photochemical oxidants" (ISA, section IS.1.1, p. IS-3). In summary, as was the case for previous reviews, the evidence base for health

effects of photochemical oxidants does not indicate an importance of any other photochemical oxidants as it includes O_3 almost exclusively as an indicator of photochemical oxidants, thus continuing to support the appropriateness of O_3 as the indicator for photochemical oxidants.

In considering the extensive evidence base for health effects of O_3, we give particular attention to the longstanding evidence of respiratory effects causally related to O_3 exposures. This array of effects, and the underlying evidence base, was an integral basis for setting the current standard. As summarized in section 3.3.1 above and addressed in detail in the ISA, the evidence base in this review does not include new evidence of respiratory effects associated with appreciably different exposure circumstances, including any that would be expected to occur under air quality conditions associated with the current standard. Thus, in considering the information available at this time, we continue to focus on exposure circumstances associated with the current standard as those of importance in this review.

Further, while the evidence base has been augmented somewhat since the time of the last review, we note that the newly available evidence does not lead to different conclusions regarding the respiratory effects of O_3 in ambient air or regarding exposure concentrations associated with those effects; nor does it identify different populations at risk of O_3-related effects. In this way, the health effects evidence available in this review is consistent with evidence available in the last review when the current standard was established. This strong evidence base continues to demonstrate a causal relationship between short-term O_3 exposures and respiratory effects, including in people with asthma. This conclusion is primarily based on evidence from controlled human exposure studies available at the time of the last review that reported lung function decrements and respiratory symptoms in people exposed to O_3 for 6.6 hours during which they engage in five hours of exercise. Support is also provided by the experimental animal and epidemiologic evidence that is coherent with the controlled exposure studies. The epidemiologic evidence, including that recently available, includes studies reporting positive associations for asthma-related hospital admissions and emergency department visits, which are strongest for children, with short-term O_3 exposures. Based collectively on this evidence, populations identified as at risk of such effects include people with asthma and children.

As in the last review, the most certain evidence of health effects in humans elicited by exposures to specific O_3 exposure concentrations is provided by controlled human exposure studies. This category of short-term studies includes an extensive evidence base of 1- to 3-hour studies, conducted with continuous or intermittent exercise and generally involving relatively

higher exposure concentrations (e.g., greater than 120 ppb).[88] Given the lack of ambient air concentrations of this magnitude in areas meeting the current standard (section 2.4.1 above), we continue to focus primarily on a second group of somewhat longer-duration studies of much lower exposure concentrations. These studies employ a 6.6-hour protocol that includes six 50-minute periods of exercise at moderate or greater exertion. There are no new such studies available in the current review. Thus, the evidence newly available in this review does not extend our understanding of the range of exposure concentrations that elicit effects in such studies beyond what was understood in the last review.

As in the last review, 60 ppb remains the lowest exposure concentration (target concentration, as average across exercise periods) at which statistically significant lung function decrements have been reported in the 6.6-hour exposure studies. Two studies have assessed exposure concentrations at the lower concentration of 40 ppb, with no statistically significant finding of O_3-related FEV_1 decrement for the group mean in either study (which is just above and well below 1% in the two studies). At 60 ppb, the group mean O_3-related decrement in FEV_1 ranges from approximately 2 to 4%, with associated individual study subject variability in decrement size. In the single study assessing the next highest exposure concentration (just above 70 ppb),[89] the group mean FEV_1 decrement (6%) was also statistically significant, as were respiratory symptom scores. At higher exposure concentrations, the incidence of both respiratory symptom scores and O_3-related lung function decrements in the study subjects is increased. Other respiratory effects, such as inflammatory response and airway resistance are also increased at higher exposures (ISA; 2013 ISA; 2006 AQCD).

In considering what may be indicated by the epidemiologic evidence with regard to exposure concentrations eliciting effects, we recognize that very few of the numerous epidemiologic studies of respiratory outcome associations with O_3 in ambient air were conducted in areas during times when the current standard was met. In fact, the vast majority of these studies were conducted in locations and during time periods that would not have met the current standard, thus making them less useful for considering the potential for O_3 concentrations allowed by the current standard to contribute to health effects. While there were about a handful

[88] Table 3A-3 in Appendix 3 summarizes controlled human exposures to O_3 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 for this review.

[89] 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 O_3 measurements during the six 50-minute exercise periods, that the mean O_3 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).

of multi-city studies in which the O_3 concentrations in a subset of the study locations and for a portion of the study period appear to have met the current standard, data were not available in some cities for the earlier years of the study period when design values for other cities were well above 70 ppb (as discussed in section 3.3.3). We recognize that the study analyses and associations reported were based on the combined dataset across the full time period (and, for multicity studies, from all cities), and the extent to which risk associated with exposures derived from the concentrations in the subset of years (and locations) that would have met the current standard compared to that from the years (and locations) that would have violated the standard influenced the study findings is not clear. There were no studies conducted in U.S. locations with ambient air O_3 concentrations that would meet the current standard for the entire duration of the study (i.e., with design values[90] at or below 70 ppb). Thus, the epidemiologic studies provide limited insight regarding exposure concentrations associated with health outcomes that might be expected under air quality conditions that meet the current standard (section 3.3.3 above).

In this review, as in the last review, we recognize some uncertainty, reflecting limitations in the evidence base, with regard to the exposure levels eliciting effects in some population groups not included in the available controlled human exposure studies, such as children and individuals with asthma, as well as the severity of the effects. Further, we note uncertainty in the extent or characterization of effects at exposure levels below those studied. In so doing, we recognize that the controlled human exposure studies, primarily conducted in healthy adults, on which the depth of our understanding of O_3-related health effects is based, provide limited, but nonetheless important information with regard to responses in people with asthma or in children. Additionally, some aspects of our understanding continue to be limited; among these aspects are the potential for effects in some people exposed to concentrations below 60 ppb. Collectively, these aspects of the evidence and associated uncertainties contribute to a recognition that for O_3, as for other pollutants, the available evidence base in a NAAQS review generally reflects a continuum, consisting of exposure levels at which scientists generally agree that health effects are likely to occur, through lower levels at which the likelihood and magnitude of the response become increasingly uncertain.

As at the time of the last review, the exposure and risk estimates developed from modeling exposures to O_3 derived from precursors emitted into ambient air are critically important to consideration of the potential for exposures and risks of concern under air quality conditions of interest, and consequently are critically important to judgments on the adequacy of public health protection provided by the current standard. In considering the public health

[90] 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.

implications of estimated occurrences of exposures to the three benchmark concentrations, we take note of guidance from the ATS, and judgments made by the EPA in considering similar effects in previous NAAQS reviews (80 FR 65343, October 26, 2015). As recognized in section 3.3.2, an additional publication by the ATS that further addresses judgments on what constitutes an adverse health effect of air pollution is newly available in this review (Thurston et al., 2017). The more recent statement expands upon the 2000 statement, that was considered in the last O_3 NAAQS review (e.g., giving increased weight to small lung function changes without accompanying respiratory symptoms when they are documented to occur in individuals with compromised lung function). We note that, in keeping with the intent of these statements to avoid specific criteria, neither statement provides more specific descriptions of respiratory responses, such as with regard to magnitude, duration or frequency, for consideration of such conclusions. The earlier ATS statement, in addition to emphasizing clinically relevant effects, also emphasized both the need to consider changes in "the risk profile of the exposed population," and effects on the portion of the population that may have a diminished reserve that puts its members at potentially increased risk if affected by another agent (ATS, 2000). These concepts, including the consideration of the magnitude of effects occurring in just a subset of study subjects, continue to be recognized as important in the more recent ATS statement (Thurston et al., 2017) and continue to be relevant to the evidence base for O_3.

As summarized in section 2.1 above, the decision in the last review considered the breadth of the O_3 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. The controlled human exposure study evidence as a whole provided context for consideration of the 2014 HREA results for the exposures of concern (i.e., the comparison-to-benchmarks analysis) (80 FR 65363, October 26, 2015).

In considering the exposure and risk analyses available in this review, we first note several ways in which these analyses differ from and improve upon those available in the last review. For example, we note the number of improvements to input data and modeling approaches summarized in section 3.4.1 above. As in prior reviews, exposure and risk are estimated from air quality scenarios designed to just meet an O_3 standard in all its elements. That is, the air quality scenarios are defined by the highest design value in the study area, which is the location with the highest 3-year average of annual fourth highest daily maximum 8-hour O_3 concentrations (e.g., equal to 70 ppb for the current standard scenario). The current risk and exposure analyses include air quality simulations based on more recent ambient air quality data that include O_3 concentrations closer to the current standard. As a result, much smaller

reductions in precursor emissions were needed in the photochemical modeling than was the case with the 2014 HREA. Further, this modeling was updated to reflect the current state of the science. Additionally, the approach for deriving population exposure estimates, both for comparison to benchmark concentrations and for use in deriving lung function risk using the E-R function approach, has been modified to provide for a better match of the simulated population exposure estimates with the 6.6-hour duration of the controlled human exposure studies and with the study subject ventilation rates. Together, these differences, as well as a variety of updates to model inputs, are believed to reduce uncertainty associated with our interpretation of the analysis results. As we consider the exposure and risk estimates, we also take note of the array of air quality and exposure circumstances represented by the eight study areas. As summarized in section 3.2.2 above, the areas fall into seven of the nine climate regions in the continental U.S. The population sizes of the associated metropolitan areas range in size from approximately 2.4 to 8 million and vary in population demographic characteristics. While there are uncertainties and limitations associated with the exposure and risk estimates, as noted in section 3.4.4 above, the factors recognized here contribute to their usefulness in informing the current review.

As at the time of the last review, people of all ages with asthma, children, and outdoor workers, are populations at increased risk of respiratory effects related to O_3 in ambient air. The size of the U.S. population with asthma is approximately 25 million. Children with asthma, which number approximately six million in the U.S., may be particularly at risk (section 3.3.2 above). While there are more adults in the U.S. with asthma than children with asthma, the exposure and risk analysis results in terms of percent of the simulated at-risk populations, indicate higher frequency of exposures of potential concern and risks for children as compared to adults. This finding relates to children's greater frequency and duration of outdoor activity, as well as their greater activity level while outdoors (section 3.4.3 above). In light of these conclusions and findings, we have focused our consideration of the exposure and risk analyses here on children.

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

While we note reduced uncertainty in several aspects of the exposure and risk analysis approach (as summarized above), we continue to recognize the relatively greater uncertainty associated with the lung function risk estimates compared to the results of the comparison-to-benchmarks analysis. Further, with regard to the lung function risk estimates, we recognize greater uncertainty with the estimates derived using the MSS model approach. Thus, we focus primarily on the estimates of exposures at or above different benchmark concentrations that represent different levels of significance of O_3-related effects, both with regard to the array of effects and severity of individual effects.

Based on all of the above, and taking into consideration related information, limitations and uncertainties, such as those recognized above, we address the extent to which the newly available information in this review supports or calls into question the adequacy of protection afforded by the current standard. In this context for the air quality scenario for the current standard, we note that across all eight study areas, which provide an array of exposure situations, less than 1% of children with asthma are estimated to experience, while breathing at an elevated rate, a daily maximum 7-hour exposure per year at or above 70 ppb, on average across the 3-year period, with a maximum of 1% for the study area with the highest estimates in the highest single year (as summarized in section 3.4.2 above). Further, the percentage for at least one day with such an exposure above 80 ppb is less than 0.1%, as an average across the 3-year period (and 0.1% or less in each of the three years simulated across the eight study areas). No simulated individuals were estimated to experience more than a single such day with an exposure at or above the 80 ppb benchmark. Although the exposure and risk analysis approaches have been updated since the last review as summarized in section 3.4.1 above, these estimates are generally similar to the comparable estimates for these benchmarks from the 2014 HREA considered at the time the current standard was set, [91] with only slight differences observed, e.g., for the lowest benchmark. We take note, however, of the differences across air quality scenarios for both sets of estimates which remain appreciably larger than the slight differences between the current and 2014 estimates. Thus, we conclude the current estimates of children and children with asthma that might be expected to experience a day with an exposure while exercising at or above the three benchmark concentrations to be generally similar to those that were a primary focus of the decision in establishing the current standard in 2015.

[91] 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 estimated to eliminate the occurrence of two or more exposures of concern to O_3 concentrations at or above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O_3 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).

We additionally consider the estimates of 7-hour exposures, at elevated ventilation, at or above 60 ppb. In so doing, we recognize the role of this consideration in the 2015 decision to be in the context of the Administrator's judgment regarding an adequate margin of safety for the new standard. We additionally recognize the greater significance of risk for multiple occurrences of days at or above this benchmark, given the associated greater potential for more lasting effects. The exposure analysis estimates indicate fewer than 1% to just over 3% of children with asthma, on average across the 3-year period to be expected to experience two or more days with an exposure at or above 60 ppb, while at elevated ventilation. This finding of about 97% to more than 99% of children protected from experiencing two or more days with exposures at or above 60 ppb while at elevated exertion is quite similar to the characterization of such estimates at the time of the 2015 decision establishing the current standard (as summarized in section 3.1.2.4 above),[92] and half that indicated by the comparable estimates for air quality just meeting the slightly higher design value of 75 ppb. In addition to this level of protection at the lower exposure level, the current information also indicates more than 99% of children with asthma, on average per year, to be protected from a day or more with an exposure at or above 70 ppb. In light of public health judgments by the EPA in prior NAAQS reviews, as well as ATS guidance, we recognize a greater concern for 7-hour exposures generally at or above 70 and 80 ppb (while at elevated exertion) than such exposures to O_3 concentrations below 70 ppb, and a greater concern for repeated (*versus* single) occurrences of such exposures at concentrations at or above 60 ppb up to 70 ppb. With this in mind, we find the exposure and risk estimates for the current review indicate that the current standard is likely to provide a high level of protection from O_3-related health effects to at-risk populations of all children and children with asthma. We additionally recognize such protection to be generally similar to what was estimated when the standard was set in the last review.

As recognized above, the protection afforded by the current standard stems from its elements collectively, including the level of 70 ppb, the averaging time of eight hours and the form of the annual fourth-highest daily maximum concentration averaged across three years. The current evidence as considered in the ISA, the current air quality information as analyzed in chapter 2 of this document, and the current risk and exposure information presented in Appendix

[92] 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 remarkedly similar at 0.6 -2.9% (Table 3-8).

3D and summarized here provide continued support to these elements, as well as to the current indicator, as discussed earlier in this section.

In summarizing the information discussed thus far, we reflect on the key aspects of the 2015 decision that established the current standard. As an initial matter, effects associated with 6.6-hour exposures in the controlled human exposure studies, that included five hours of exercise, to concentrations just above 70 ppb included both lung function decrements and respiratory symptoms, which the EPA recognized to be adverse; this judgment was based on consideration of the EPA decisions in prior NAAQS reviews and CASAC advice, as well as ATS guidance (80 FR 65343, October 26, 2015). We note that the newly available information in this review includes an additional statement from ATS on assessing adverse effects of air pollution which is generally consistent with the earlier statement (available at the time of the 2015 decision), e.g., continuing to emphasize potentially at-risk groups including specific consideration of effects in people with compromised lung function. While recognizing the differences between the current and past exposure and risk analyses, as well as uncertainties associated with such analyses, we note a rough consistency of the associated estimates when considering the array of study areas in both reviews. Overall, the newly available quantitative analyses appear to comport with the conclusions reached in the last review regarding control expected to be exerted by the current standard on exposures of concern.

We additionally recognize that conclusions regarding the adequacy of the current standard depend in part on public health policy judgments, such as those identified above, and judgments about when a standard is requisite to protect the public health, including the health of at-risk populations, allowing for an adequate margin of safety. In so doing, we take note of the long-standing health effects evidence that documents the effects of 6.6-hour O_3 exposures on people exposed while breathing at elevated rates and recognize that these such effects have been reported in a few individuals for the lowest concentration studied in exposure chambers (40 ppb). Thus, in considering the exposure analysis estimates for 7-hour exposures at and above 60 ppb, we also take note of the variability in the responses at low concentrations, including, for example, the variation in average response to a 7-hour 60 ppb exposure with exercise (group mean FEV_1 decrement of 1.7 to 3.5% change), as well as the lack of statistically significant decrements in lung functions from such exposures at concentrations below 60 ppb. Consistent with the EPA's judgments in the last review, we also recognize the greater potential for health risk from repeated (*versus* isolated single) occurrences. In so doing, we note that the exposure estimates indicate the current standard may be expected to protect more than 97% of populations of children with asthma residing in areas just meeting the current standard from experiencing more than a single day per year with an exposure at or above 60 ppb, on average over a 3-year period. We additionally note the estimates that indicate protection of more than 99.9% of

children with asthma living in such areas from experiencing any days with a 7-hour exposure while at elevated exertion of 80 ppb or higher in a 3-year period, on average. In light of ATS guidance, CASAC advice and EPA judgments and conclusions in past NAAQS reviews, these results indicate a high level of protection of key at-risk populations from O_3-related health effects that is a generally similar level of protection to what was articulated when the standard was set in 2015. Thus, the evidence and exposure/risk information, including that related to the lowest exposures studied, lead us to conclude that the combined consideration of the body of evidence and the quantitative exposure estimates including the associated uncertainties, do not call into question the adequacy of protection provided by the current standard. Rather, this information continues to provide support for the current standard, and thus supports consideration of retaining the current standard, without revision.

In reaching this conclusion, we additionally note the CASAC conclusion that the newly available evidence does not substantially differ from that available in the last review. Accordingly, some of the CASAC concluded that the currently available evidence did not support revision of the standard. Another segment of the CASAC indicated its agreement with the prior CASAC comments on the 2014 draft PA, in which the prior CASAC opined that a standard set at 70 ppb may not provide an adequate margin of safety (Cox, 2020, p. 1). In considering this advice, we take note of the complete advice from the prior CASAC which based on its focus on the scientific evidence recommended a range of levels from 70 ppb to 60 ppb and recognized that the choice of a level within this range "is a policy judgment under the statutory mandate of the Clean Air Act" (Frey, 2014, p. ii). We also note the judgments reached by the Administrator in the last review, with consideration of this CASAC advice, in setting the standard at a level of 70 ppb. (80 FR 65362, October 26, 2015).

In summary, the newly available health effects evidence, critically assessed in the ISA as part of the full body of evidence, reaffirms conclusions on the respiratory effects recognized for O_3 in the last review. Further, we observe the general consistency of the current evidence with the evidence that was available in the last review with regard to key aspects on which the current standard is based. We additionally note the quantitative exposure and risk estimates for conditions just meeting the current standard that indicate a generally similar level of protection for at-risk populations from respiratory effects, as that described in the last review for the now-current standard. We also recognize limitations and uncertainties associated with the available information, similar to those at the time of the last review. Collectively, these considerations (including those discussed above) provide the basis for the conclusion that consideration should be given to retaining the current primary standard of 0.070 ppm O_3, as the fourth-highest daily maximum 8-hour concentration averaged across three years, without revision. Accordingly, and

in light of this conclusion that it is appropriate to consider the current standard to be adequate, we have not identified any potential alternative standards for consideration in this review.

3.6 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH

In this section, we highlight key uncertainties associated with reviewing and establishing the primary O_3 standard, while additionally recognizing that research in these areas may be informative to the development of more efficient and effective control strategies. The list in this section includes key uncertainties and data gaps thus far highlighted in this review of the primary standard. A critical aspect of our consideration of the evidence and the quantitative risk/exposure estimates is our understanding of O_3 effects below the lowest concentrations studied in controlled human exposure studies, for longer exposures and for different population groups, particularly including people with asthma. Additional information in several areas would reduce uncertainty in our interpretation of the available information for purposes of risk characterization and, accordingly, reduce uncertainty in characterization of O_3-related health effects. In this section, we highlight areas for future health-related research, model development, and data collection activities to address these uncertainties and limitations in the current scientific evidence. These areas are similar to those highlighted in past reviews.

Exposure and Risk Assessment Data and Tools:

- An important aspect of risk assessment and characterization to inform decisions regarding the primary standard is our understanding of the exposure-response relationship for O_3-related health effects in at-risk populations. Additional research is needed to more comprehensively assess risk of respiratory effects in at-risk individuals exposed to O_3 in the range of 40 to 80 ppb, and lower, for 6.6 hours while engaged in moderate exertion.
- Population- or cohort-based information on human exposure and associated health effects for healthy adults and children and at-risk populations, including people with asthma, to relevant levels and durations of O_3 concentrations in ambient air, including exposure information in various microenvironments and at varying activity levels, is needed to better evaluate current and future O_3 exposure and lung function risk models. Such information across extended periods would facilitate evaluation of exposure models for the O_3 season.
- Collection of time-activity data over longer time periods, and particularly for children, is needed to reduce uncertainty in the modeled exposure distributions that form an important part of the basis for decisions regarding NAAQS for O_3 and other air pollutants. Research addressing energy expenditure and associated breathing rates in various population groups, particularly healthy children and children with asthma, in various locations, across the spectrum of physical activity, including sleep to vigorous exertion, is needed.

Health Effects Evidence Base:

- Epidemiologic studies assessing the influence of "long-term" or "short-term" O_3 exposures is complicated by a lack of knowledge regarding the exposure history of study populations. Further, existing studies generally focus on either long-term or short-term exposure separately, thereby making it difficult to assess whether a single short-term high-level exposure versus a repeated long-term low-level exposure, or a combination of both short-term high-level and repeated long-term low-level exposures, influence health outcomes of the study subjects. Epidemiologic studies that include exposure measurements across a longer-term assessment period and can simultaneously assess the impact of these various elements of exposure (i.e., magnitude, frequency, durations, and pattern) are needed.
- The extent to which the broad mix of photochemical oxidants as well as other copollutants in the ambient air (e.g., PM, NO_2, SO_2, etc.) may play a role in modifying or contributing to the observed associations between ambient air O_3 concentrations and reported health outcomes continues to be an important research question. A better understanding of the broader mixture of photochemical oxidants other than O_3 in ambient air, the associated human exposures, and of the extent to which effects of the mixture may differ from those of O_3, would be informative to future NAAQS reviews. Examine and improve analytical approaches to better understand the role of copollutants, as well as temperature, in contributing to potential confounding or effect modification in epidemiologic models.
- Most epidemiologic study designs remain subject to uncertainty due to use of fixed-site ambient air monitors serving as a surrogate for exposure measurements. Measurements made at stationary outdoor monitors have been used as independent variables for air pollution, but the accuracy with which these measurements actually reflect subjects' exposure is not yet fully understood. The degree to which discrepancies between stationary monitor measurements and actual pollutant exposures introduces error into statistical estimates of pollutant effects in epidemiologic studies needs to be investigated.
- For health endpoints reported in epidemiologic studies, such as respiratory hospital admissions, ED visits, and premature mortality, a more comprehensive characterization of the exposure circumstances (including ambient air concentrations, as well as duration of exposure and activity levels of individuals) eliciting such effects is needed
- Further research investigating additional uncertainties and factors that modify epidemiologic associations, particularly for different population groups would improve our understanding in these areas.
- The current evidence base, expanded by evidence newly available in this review, indicates a likely causal relationship between short-term O_3 exposure and metabolic effects. Further research characterizing perturbations of glucose and insulin homeostasis by O_3 in controlled human exposure studies at exertion and in animal toxicology studies at concentrations closer to the current standard are needed inform decisions regarding the primary standard. The collection of population-based information on clinical health outcomes such as metabolic syndrome, diabetes, etc., as well as intermediate indicators like insulin resistance is also needed. Such studies would provide an improved understanding of relationships between O_3 exposure and metabolic-related health outcomes.

Air Quality:

- To reduce uncertainties in photochemical modeling used in estimating O_3 concentration for different air quality scenarios, we need an improved understanding of the atmospheric chemistry that produces elevated O_3 concentrations and the extent to which key influences and precursors vary seasonally and geographically across the U.S.
- An improved understanding of relationships between variations in the form of the current standard would benefit consideration of the form in future reviews.

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4 REVIEW OF THE SECONDARY STANDARD

This chapter presents and evaluates the policy implications of the currently available scientific and technical information pertaining to this review of the O_3 secondary standard. In so doing, the chapter presents key aspects of the current evidence of the welfare effects of O_3, as documented in the ISA, with support from the prior ISA and AQCDs, and associated public welfare implications, as well as key aspects of quantitative analyses of currently available air quality and exposure-related information that is presented in appendices associated with this chapter. Together this information provides the foundation for our evaluation of the current scientific information regarding welfare effects of O_3 in ambient air and the potential for welfare effects to occur under air quality conditions associated with the current standard, 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 conclusions reached in the last review. In so doing, we additionally take note of the recent decision of the D.C. Circuit, summarized in section 4.1.2 below, remanding the secondary standard established in the last review to the EPA for further justification or reconsideration (*Murray Energy Corp. v. EPA*, 936 F.3d 597 [D.C. Cir. 2019]). 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 review of the secondary O_3 standard.

Within this chapter, background information on the current standard, including considerations in its establishment in the last review, is summarized in section 4.1. The general approach for considering the currently available information in this review, including policy-relevant questions identified to frame our policy evaluation, is summarized in section 4.2. Key aspects of the currently 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 presents associated summary 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

The current standard was set in 2015 based on the scientific and technical information available at that time, as well as the Administrator's judgments regarding the available welfare effects evidence, the appropriate degree of public welfare protection for the revised standard, and

available air quality information on seasonal cumulative exposures that may be allowed by such a standard (80 FR 65292, October 26, 2015). With the 2015 decision, the Administrator revised the level of the secondary standard for photochemical oxidants, including O_3, to 0.070 ppm, in conjunction with retaining the indicator (O_3), averaging time (8 hours) and form (fourth-highest annual daily maximum 8-hour average concentration, averaged across three years).

The welfare effects evidence base available in the 2015 review included more than fifty years of extensive research on the phytotoxic effects of O_3, conducted both in and outside of the U.S. that documents the impacts of O_3 on plants and their associated ecosystems (U.S. EPA, 1978, 1986, 1996, 2006, 2013). As was established in prior reviews, O_3 can interfere with carbon gain (photosynthesis) and allocation of carbon within the plant, making fewer carbohydrates available for plant growth, reproduction, and/or yield. For seed-bearing plants, these reproductive effects will culminate in reduced seed production or yield (U.S. EPA, 1996, pp. 5-28 and 5-29). The strongest evidence for effects from O_3 exposure on vegetation is from controlled exposure studies, which "have clearly shown that exposure to O_3 is causally linked to visible foliar injury,[1] decreased photosynthesis, changes in reproduction, and decreased growth" in many species of vegetation (2013 ISA, p. 1-15). For example, "visible foliar injury occurs only when sensitive plants are exposed to elevated O_3 concentrations in a predisposing environment," (2013 ISA, p. 9-39). Effects such as decreased photosynthesis, changes in reproduction, and decreased growth at the plant scale can also be linked to an array of effects at larger organizational (e.g., population, community, system) and spatial scales, with the evidence available in the last review indicating that "O_3 exposures can affect ecosystem productivity, crop yield, water cycling, and ecosystem community composition" (2013 ISA, p. 1-15, Chapter 9, section 9.4).

In light of this robust evidence base, the 2013 ISA concluded there to be causal relationships between O_3 and visible foliar injury, reduced vegetation growth, reduced productivity in terrestrial ecosystems, reduced yield and quality of agricultural crops and alteration of below-ground biogeochemical cycles. The 2013 ISA additionally concluded there was likely to be a causal relationship between O_3 and reduced carbon sequestration in terrestrial ecosystems, alteration of terrestrial ecosystem water cycling and alteration of terrestrial community composition (2013 ISA, p. lxviii and Table 9-19). Further, based on the then-available evidence with regard to O_3 effects on climate, the 2013 ISA also found there to be a causal relationship between changes in tropospheric O_3 concentrations and radiative forcing, found there likely to be a causal relationship between tropospheric O_3 concentrations and effects

[1] This includes visible changes to leaves or needles such as the occurrence of small dots or bleaching (2013 ISA, p. 9-38).

on climate as quantified through surface temperature response, and found the evidence to be inadequate to determine if a causal relationship exists between tropospheric O_3 concentrations and health and welfare effects related to shielding of ultraviolet radiation at wavelengths of 280 to 320 nm (UV-B shielding) (2013 ISA, section 10.5).

The 2015 decision was a public welfare policy judgment made by the Administrator, which drew upon the available scientific evidence for O_3-attributable welfare effects and on quantitative analyses of exposures and public welfare risks based on impacts to vegetation, ecosystems and their associated services, as well as judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses. The analyses utilized a cumulative, concentration-weighted exposure index for O_3, the W126 index, that gives greater weight to elevated concentrations. Use of this metric was based on conclusions in the 2013 ISA that exposure index that cumulate hourly O_3 concentrations, giving greater weight to the higher concentrations (such as the W126 index), perform well in describing exposure-response relationships documented in crop and tree seedling studies (2013 ISA, section 9.5). Included in this decision were judgments on the weight to place on the evidence of specific vegetation-related effects estimated to result across a range of cumulative seasonal concentration-weighted O_3 exposures; on the weight to give associated uncertainties, including uncertainties of predicted environmental responses (based on experimental study data); variability in occurrence of the specific effects in areas of the U.S., especially in areas of particular public welfare significance; and on the extent to which such effects in such areas may be considered adverse to public welfare.

The decision was based on a thorough review in the 2013 ISA of the scientific information on O_3-induced environmental effects. The decision also took into account: (1) assessments in the 2014 PA of the most policy-relevant information in the 2013 ISA regarding evidence of adverse effects of O_3 to vegetation and ecosystems, information on biologically-relevant exposure metrics, 2014 welfare REA (WREA) analyses of air quality, exposure, and ecological risks and associated ecosystem services, and staff analyses of relationships between levels of a W126-based exposure index[2] and potential alternative standard levels in combination with the form and averaging time of the then-current standard; (2) additional air quality analyses of the W126 index and design values based on the form and averaging time of the then-current standard; (3) CASAC advice and recommendations; and (4) public comments received during the development of these documents and on the proposal notice. In addition to reviewing the

[2] The W126 index is a cumulative seasonal metric described as the sigmoidally weighted sum of all hourly O_3 concentrations observed during a specified daily and seasonal time window, where each hourly O_3 concentration is given a weight that increases from zero to one with increasing concentration (80 FR 65373-74, October 26, 2015). Accordingly, W126 index values are in the units of ppm-hours (ppm-hrs).

most recent scientific information as required by the CAA, the 2015 rulemaking also incorporated the EPA's response to the judicial remand of the 2008 secondary O_3 standard in *Mississippi v. EPA*, 744 F.3d 1334 (D.C. Cir. 2013) and, in light of the court's decision in that case, explained the Administrator's conclusions as to the level of air quality judged to provide the requisite protection of public welfare from known or anticipated adverse effects.

Consistent with the general approach routinely employed in NAAQS reviews, the initial consideration in the last review of the secondary standard was with regard to the adequacy of protection provided by the then-existing standard. Key aspects of that consideration are summarized in section 4.1.1 below. The subsequent selection of a standard concluded by the Administrator to provide the requisite protection under the Act is summarized in section 4.1.2.

4.1.1 Considerations Regarding Adequacy of the Prior Standard

The Administrator's conclusion in the 2015 review regarding the adequacy of the secondary standard that was set in 2008 (0.075 ppm, as annual fourth-highest daily maximum 8-hour average concentration averaged over three consecutive years) gave primary consideration to the evidence of growth effects in well-studied tree species and information on cumulative seasonal O_3 exposures occurring in Class I areas[3] when the then-current standard was met (80 FR 65385-65386, October 26, 2015). In so doing, the exposure information for Class I areas was evaluated in terms of the W126 cumulative seasonal exposure index, an index recognized by the 2013 ISA as a mathematical approach "for summarizing ambient air quality information in [a] biologically meaningful form[] for O_3 vegetation effects assessment purposes" (2013 ISA, section 9.5.3, p. 9-99). The EPA focused on the W126 index for this purpose consistent with the evidence in the 2013 ISA and advice from the CASAC (80 FR 65375, October 26, 2015).

In her decision making, the Administrator considered the effects of O_3 on tree seedling growth, as suggested by the CASAC, as a surrogate or proxy for the broader array of vegetation-related effects of O_3, ranging from effects on sensitive species to broader ecosystem-level effects (80 FR 65369, 65406, October 26, 2015). The metric used for quantifying effects on tree seedling growth in the review was relative biomass loss (RBL), with the evidence base providing robust and established exposure-response (E-R) functions for seedlings of 11 tree species (80 FR 65391-92, October 26, 2015; 2014 PA, Appendix 5C).[4] The Administrator used this proxy in making her judgments on O_3 effects to the public welfare.

[3] Areas designated as Class I include all international parks, national wilderness areas which exceed 5,000 acres in size, national memorial parks which exceed 5,000 acres in size, and national parks which exceed 6,000 acres in size, provided the park or wilderness area was in existence on August 7, 1977. Other areas may also be Class I if designated as Class I consistent with the CAA.

[4] These functions for RBL estimate the reduction in a year's growth as a percentage of that expected in the absence of O_3 (2013 ISA, section 9.6.2; 2014 WREA, section 6.2).

In considering the public welfare protection provided by the then-current standard, the Administrator gave primary consideration to an analysis of cumulative seasonal exposures in or near Class I areas during periods when the then-current standard was met and the associated estimates of growth effects in tree seedlings, in terms of the O_3 attributable reductions in RBL in the median species for which E-R functions have been established (80 FR 65389-65390, October 26, 2015).[5] The Administrator noted the occurrence of exposures for which the associated median estimates of growth effects across the species with E-R functions extend above a magnitude considered to be "unacceptably high" by the CASAC.[6] This analysis estimated cumulative exposures, in terms of 3-year average W126 index values, at and elevated above 19 ppm-hrs, that occurred under the then-current standard for nearly a dozen areas, distributed across two NOAA climatic regions of the U.S (80 FR 65385-86, October 26, 2015).[7] The Administrator gave particular weight to this analysis because of its focus on exposures in Class I areas, which are lands that Congress set aside for specific uses 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. This emphasis on lands afforded special government protections, such as national parks and forests, wildlife refuges, and wilderness areas, some of which are designated Class I areas under the CAA, was consistent with a similar emphasis in the 2008 review of the standard (73 FR 16485, March 27, 2008). The Administrator additionally recognized that states, tribes and public interest groups also set aside areas that are intended to provide similar benefits to the public welfare for residents on those lands, as well as for visitors to those areas (80 FR 65390, October 26, 2015).

As noted across past reviews of O_3 secondary standards, the Administrator's judgments regarding effects that are adverse to public welfare consider the intended use of the ecological receptors, resources and ecosystems affected (80 FR 65389, October 26, 2015; 73 FR 16496, March 27, 2008). Thus, in the 2015 review, the Administrator utilized the median RBL estimate

[5] In specifically evaluating exposure levels in terms of the W126 index as to potential for impacts on vegetation, the Administrator focused on the median RBL estimate across the eleven tree species for which robust established E-R functions were available. The presentation of these E-R functions for growth effects on tree seedlings (and crops) included estimates of RBL (and relative yield loss [RYL]) at a range of W126-based exposure levels (2014 PA, Tables 5C-1 and 5C-2). The median tree species RBL or crop RYL was presented for each W126 level (2014 PA, Table 5C-3; 80 FR 65391 [Table 4], October 26, 2015). The Administrator focused on RBL as a surrogate or proxy for the broader array of vegetation-related effects of potential public welfare significance, which include effects on growth of individual sensitive species and extend to ecosystem-level effects, such as community composition in natural forests, particularly in protected public lands, as well as forest productivity (80 FR 65406, October 26, 2015).

[6] In the CASAC's consideration of RBL estimates presented in the 2014 draft PA (for the 2015 review), it characterized an estimate of 6% RBL in the median studied species as being "unacceptably high," (Frey, 2014).

[7] The NOAA climatic regions are described in section 2.4.2 above and appendices 2B and 4D.

for the studied species as a quantitative tool within a larger framework of considerations pertaining to the public welfare significance of O_3 effects. She recognized such considerations to include effects that are associated with effects on growth and that the 2013 ISA determined to be causally or likely causally related to O_3 in ambient air, yet for which there are greater uncertainties affecting estimates of impacts on public welfare. These other effects included reduced productivity in terrestrial ecosystems, reduced carbon sequestration in terrestrial ecosystems, alteration of terrestrial community composition, alteration of below-ground biogeochemical cycles, and alteration of terrestrial ecosystem water cycles. Thus, in giving attention to the CASAC's characterization of a 6% estimate for tree seedling RBL in the median studied species as "unacceptably high", the Administrator, while mindful of uncertainties with regard to the magnitude of growth impact that might be expected in the field and in mature trees, was also mindful of related, broader, ecosystem-level effects for which the available tools for quantitative estimates are more uncertain and those for which the policy foundation for consideration of public welfare impacts is less well established. As a result, the Administrator considered tree growth effects of O_3, in terms of RBL "as a surrogate for the broader array of O_3 effects at the plant and ecosystem levels" (80 FR 65389, October 26, 2015).

Based on all of these considerations, and taking into consideration CASAC advice, the Administrator concluded that the protection afforded by the then-current standard was not sufficient and that the standard needed to be revised to provide additional protection from known and anticipated adverse effects to public welfare, related to effects on sensitive vegetation and ecosystems, most particularly those occurring in Class I areas, and also in other areas set aside by states, tribes and public interest groups to provide similar benefits to the public welfare for residents on those lands, as well as for visitors to those areas. In so doing, she further noted that a revised standard would provide increased protection for other growth-related effects, including for relative yield loss (RYL) of crops, reduced carbon storage and for types of effects for which it is more difficult to determine public welfare significance, as well as for other welfare effects of O_3, such as visible foliar injury (80 FR 65390, October 26, 2015).

4.1.2 Considerations for the Revised Standard

Consistent with the approach employed for considering the adequacy of the then-current secondary standard, the approach for considering revisions that would result in a standard providing the requisite protection under the Act also focused on growth-related effects of O_3, using RBL as a surrogate for the broad array of vegetation-related effects and included judgments on the magnitude of such effects that would contribute to public welfare impacts of concern. In considering the adequacy of potential alternative standards to provide protection from such effects, the approach also focused on considering the cumulative seasonal O_3

exposures likely to occur with different alternative standards.

In light of the judicial remand of the 2008 secondary O_3 standard referenced above, the 2015 decision on selection of a revised secondary standard first considered the available evidence and quantitative analyses in the context of an approach for considering and identifying public welfare objectives for such a standard (80 FR 65403-65408, October 26, 2015). The robust and longstanding evidence of O_3 effects on vegetation and associated terrestrial ecosystems, including evidence newly available in the 2015 review, provided the foundation for the Administrator's consideration of O_3 effects, associated public welfare protection objectives, and the revisions to the standard needed to achieve those objectives. In light of the extensive evidence base in this regard, the Administrator focused on protection against adverse public welfare effects of O_3-related effects on vegetation. In so doing, she took note of effects that compromise plant function and productivity, and associated effects on ecosystems. She had particular concern about such effects in natural ecosystems, such as those in areas with protection designated by Congress for current and future generations, as well as areas similarly set aside by states, tribes and public interest groups with the intention of providing similar benefits to the public welfare. The Administrator additionally recognized that providing protection for this purpose will also provide a level of protection for other vegetation that is used by the public and potentially affected by O_3 including timber, produce grown for consumption and horticultural plants used for landscaping (80 FR 65403, October 26, 2015).

As an initial matter, the Administrator considered the use of a cumulative seasonal exposure index for purpose of assessing potential public welfare risks, and similarly, for assessing potential protection achieved against such risks on a national scale. In consideration of conclusions of the 2013 ISA and 2014 PA, as well as advice from the CASAC and public comments, the focus was on a W126 index described as a maximum 3-month, 12-hour index, defined by the 3-consecutive-month period within the O_3 season with the maximum sum of W126-weighted hourly O_3 concentrations during the period from 8:00 a.m. to 8:00 p.m. each day (80 FR 65404, October 26, 2015). While recognizing that no one definition of an exposure metric used for the assessment of protection for multiple effects at a national scale will be exactly tailored to every species or each vegetation type, ecosystem and region of the country, the Administrator judged that on balance, a W126 index derived in this way, and averaged over three years would be appropriate for such purposes (80 FR 65403, October 26, 2015). Thus, in considering revisions to the secondary standard that would specify a level of air quality to provide the necessary public welfare protection, the Administrator focused on use of a cumulative seasonal concentration-weighted exposure index (specifically the W126 index), for assessing exposure, both for making judgments with regard to the potential harm to public welfare posed by conditions allowed by various levels of air quality and for making the

associated judgments regarding the appropriate degree of protection against such potential harm (80 FR 65403, October 26, 2015).

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 an index averaged across three years for judging public welfare protection afforded by a revised secondary standard (80 FR 65404, October 26, 2015). For example, while recognizing that the scientific evidence documents the effects on vegetation resulting from individual growing season exposures of specific magnitude, including those that can affect the vegetation in subsequent years, the Administrator was also mindful of both the strengths and limitations of the evidence and of the information on which to base her judgments with regard to adversity of effects on the public welfare. In this regard, 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. While recognizing the potential for effects on vegetation associated with a single-year exposure, the Administrator concluded that use of a 3-year average metric can 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).

While the Administrator recognized the scientific information and interpretations, as well as CASAC advice, with regard to a single-year exposure index, she also took note of uncertainties associated with judging the degree of vegetation impacts for single-year effects that would be adverse to public welfare. It was noted that even in the case of annual crops, the assessment of public welfare significance of such effects is unclear due to the role of crop management and related agricultural practices. The Administrator was also mindful of the variability in ambient air O_3 concentrations from year to year, as well as year-to-year variability in environmental factors, including rainfall and other meteorological factors, that influence the occurrence and magnitude of O_3-related effects in any year, and contribute uncertainties to interpretation of the potential for harm to public welfare over the longer term (80 FR 65404, October 26, 2015).

In reaching a conclusion on the amount of public welfare protection from the presence of O_3 in ambient air that is appropriate to be afforded by a revised secondary standard, the Administrator gave particular consideration to the following: (1) the nature and degree of effects of O_3 on vegetation, including her judgments as to what constitutes an adverse effect to the public welfare; (2) the strengths and limitations of the available and relevant information; (3) comments from the public on the Administrator's proposed decision, including comments related to identification of a target level of protection; and (4) the CASAC's views regarding the

strength of the evidence and its adequacy to inform judgments on public welfare protection. The Administrator recognized that such judgments include judgments about the interpretation of the evidence and other information, such as the quantitative analyses of air quality monitoring, exposure and risk. She also recognized that such judgments should neither overstate nor understate the strengths and limitations of the evidence and information nor the appropriate inferences to be drawn as to risks to public welfare. It was also noted that the CAA does not require that a secondary standard be protective of all effects associated with a pollutant in the ambient air but rather those known or anticipated effects judged "adverse to the public welfare" (CAA section 109). She additionally recognized that the choice of the appropriate level of protection is a public welfare policy judgment entrusted to the Administrator under the CAA taking into account both the available evidence and the uncertainties (80 FR 65404-05, October 26, 2015).

With regard to the extensive evidence of welfare effects of O_3, including the established evidence base regarding O_3 and visible foliar injury, in addition to the longstanding evidence base on O_3-attributable crop RYL, the information available for tree species was judged to be more useful in informing judgments regarding the nature and severity of effects associated with different air quality conditions and associated public welfare significance. Accordingly, the Administrator gave particular attention to the effects related to native tree growth and productivity, including forest and forest community composition, recognizing the relationship of tree growth and productivity to a range of ecosystem services (80 FR 65405-06, October 26, 2015).

In so doing, the Administrator recognized that the robust evidence base documented a broad array of O_3-induced vegetation effects, among which were the occurrence of visible foliar injury and growth and/or yield loss in O_3-sensitive annual and perennial species, including crops and other commercial species, such as timber, horticultural and landscaping plants, as well as native species in unmanaged natural areas (80 FR 65405, October 26, 2015). In regard to visible foliar injury, the Administrator recognized the potential for this effect to affect the public welfare in the context of affecting value ascribed to natural forests, particularly those afforded special government protection, with the significance of O_3-induced visible foliar injury depending on the extent and severity of the injury (80 FR 65407, October 26, 2015). In so doing, however, the Administrator also took note of limitations in the available visible foliar injury information, including 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 O_3-induced vegetation effects, such as growth or related ecosystem effects, and a lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this

vegetation effect (80 FR 65407, October 26, 2015). Similarly, while O_3-related growth effects on agricultural and commodity crops had been extensively studied and robust E-R functions developed for a number of species, the Administrator found this information less useful in informing her judgments regarding an appropriate level of public welfare protection (80 FR 65405, October 26, 2015).[8]

Thus, and in light of the extensive evidence base in this regard, the Administrator focused on trees and associated ecosystems in identifying the appropriate level of protection for the secondary standard. Accordingly, the Administrator found the estimates of tree seedling growth impacts (in terms of RBL) associated with a range of W126-based index values developed from the E-R functions for 11 tree species (referenced in section 4.1.1 above) to be appropriate and useful for considering the appropriate public welfare protection objective for a revised standard (80 FR 65391-92, Table 4, October 26, 2015). The Administrator also incorporated into her considerations the broader evidence base associated with forest tree seedling biomass loss, including other less quantifiable effects of potentially greater public welfare significance. That is, in drawing on these RBL estimates, the Administrator recognized she was not simply making judgments about a specific magnitude of growth effect in seedlings that would be acceptable or unacceptable in the natural environment. Rather, though mindful of associated uncertainties, the Administrator used the RBL estimates as a surrogate or proxy for consideration of the broader array of related vegetation and ecosystem effects of potential public welfare significance that include effects on growth of individual sensitive species and extend to ecosystem-level effects, such as community composition in natural forests, particularly in protected public lands, as well as forest productivity (80 FR 65406, October 26, 2015). This broader array of vegetation-related effects included those for which public welfare implications are more significant but for which the tools for quantitative estimates were more uncertain.

In using the RBL estimates as a proxy, the Administrator recognized that the CASAC gave weight to these relationships in formulating its advice and she took particular note of the characterization by the CASAC of the 6% RBL level in the median studied species as "unacceptably high," as this comment was provided in the context of the CASAC's consideration of the significance of effects associated with a range of alternatives for the secondary standard

[8] With respect to commercial production of commodities, the Administrator noted that judgments about the extent to which O_3-related effects on commercially managed vegetation are adverse from a public welfare perspective are particularly difficult to reach, given that the extensive management of such vegetation (which, as the CASAC noted, may reduce yield variability) may also to some degree mitigate potential O_3-related effects. The management practices used on such vegetation are highly variable and are designed to achieve optimal yields, taking into consideration various environmental conditions. In addition, changes in yield of commercial crops and commercial commodities, such as timber, may affect producers and consumers differently, further complicating the question of assessing overall public welfare impacts (80 FR 65405, October 26, 2015).

(Frey, 2014, pp. iii, 13, 14; 80 FR 65406, October 26, 2015). In consideration of CASAC advice; strengths, limitations and uncertainties in the evidence; and the linkages of growth effects to larger population, community and ecosystem impacts, the Administrator considered it appropriate to focus on a standard that would generally limit cumulative exposures to those for which the median RBL estimate for seedlings of the 11 species with robust and established E-R functions would be somewhat below 6% (80 FR 65406-07, October 26, 2015).

In focusing on cumulative exposures associated with a median RBL estimate somewhat below 6%, the Administrator considered the relationships between W126-based exposure and RBL in the studied species (presented in the final PA and proposal notice), noting that the median RBL estimate was 6% for a cumulative seasonal W126 exposure index of 19 ppm-hrs (80 FR 65391-92, Table 4, October 26, 2015).[9] Given the information on median RBL at different W126 exposure levels, using a 3-year cumulative exposure index for assessing vegetation effects, the potential for single-season effects of concern, and CASAC comments on the appropriateness of a lower value for a 3-year average W126 index, the Administrator concluded it was appropriate to identify a standard that would restrict cumulative seasonal exposures to 17 ppm-hrs or lower, in terms of a 3-year W126 index, in nearly all instances (80 FR 65407, October 26, 2015). Based on such then-current information to inform consideration of vegetation effects and their potential adversity to public welfare, the Administrator additionally judged that the RBL estimates associated with marginally higher exposures in isolated, rare instances are not indicative of effects that would be adverse to the public welfare, particularly in light of variability in the array of environmental factors that can influence O_3 effects in different systems and uncertainties associated with estimates of effects associated with this magnitude of cumulative exposure in the natural environment (80 FR 65407, October 26, 2015).

The Administrator's decisions regarding the revisions to the then-current standard that would appropriately achieve these public welfare protection objectives were based on extensive air quality analyses that extended from the then most recently available data (monitoring year 2013) back more than a decade (80 FR 65408, October 26, 2015; Wells, 2015). These analyses evaluated the cumulative seasonal exposure levels in locations meeting different alternative levels for a standard of the existing form and averaging time, indicating reductions in cumulative exposures associated with air quality meeting lower levels of a standard of the existing form and averaging time. Based on these analyses, the Administrator judged that the desired level of public welfare protection could be achieved with a secondary standard having a revised level in combination with the existing form and averaging time (80 FR 65408, October 26, 2015).

[9] When stated to the first decimal place, the median RBL was 6.0% for a cumulative seasonal W126 exposure index of 19 ppm-hrs. For 18 ppm-hrs, the median RBL estimate was 5.7%, which rounds to 6%, and for 17 ppm-hrs, the median RBL estimate was 5.3%, which rounds to 5% (80 FR 65407, October 26, 2015).

The air quality analyses described the occurrences of 3-year W126 index values of various magnitudes at monitor locations where O_3 concentrations met potential alternative standards; the alternative standards were different levels for the current form and averaging time (annual fourth-highest daily maximum 8-hour average concentration, averaged over three consecutive years) (Wells, 2015). In the then-most recent period, 2011-2013, across the more than 800 monitor locations meeting the then-current standard (with a level of 75 ppb), the 3-year W126 index values were above 17 ppm-hrs in 25 sites distributed across different NOAA climatic regions, and above 19 ppm-hrs at nearly half of these sites, with some well above. In comparison, among sites meeting an alternative standard of 70 ppb, there were no occurrences of a W126 value above 17 ppm-hrs and fewer than a handful of occurrences that equaled 17 ppm-hrs.[10] For the longer time period (extending back to 2001), among the nearly 4000 instances where a monitoring site met a standard level of 70 ppb, the Administrator noted that there was only "a handful of isolated occurrences" of 3-year W126 index values above 17 ppm-hrs, "all but one of which were below 19 ppm-hrs" (80 FR 65409, October 26, 2015). The Administrator concluded that that single higher value of 19.1 ppm-hrs, observed at a monitor for the 3-year period of 2006-2008, was reasonably regarded as an extremely rare and isolated occurrence, and, as such, it was unclear whether it would recur, particularly as areas across the U.S. took further steps to reduce O_3 to meet revised primary and secondary standards. Further, based on all of the then available information, as noted above, the Administrator did not judge RBL estimates associated with marginally higher exposures in isolated, rare instances to be indicative of adverse effects to the public welfare. The Administrator concluded that a standard with a level of 70 ppb and the current form and averaging time may be expected to limit cumulative exposures, in terms of a 3-year average W126 exposure index, to values at or below 17 ppm-hrs, in nearly all instances, and accordingly, to eliminate or virtually eliminate cumulative exposures associated with a median RBL of 6% or greater (80 FR 65409, October 26, 2015). Thus, using RBL as a proxy in judging effects to public welfare, the Administrator judged that a standard with a level of 70 ppb would provide the requisite protection from adverse effects to public welfare by limiting cumulative seasonal exposures to 17 ppm-hrs or lower, in terms of a 3-year W126 index, in nearly all instances.

In summary, the Administrator judged that the revised standard would protect natural forests in Class I and other similarly protected areas against an array of adverse vegetation effects, most notably including those related to effects on growth and productivity in sensitive tree species. The Administrator additionally judged that a revised standard set at a level of 70

[10] The more than 500 monitors that would meet an alternative standard of 70 ppb during the 2011-2013 period were distributed across all nine NOAA climatic regions and 46 of the 50 states (Wells, 2015 and associated dataset in the docket [document identifier, EPA-HQ-OAR-2008-0699-4325]).

ppb would be sufficient to protect public welfare from known or anticipated adverse effects. This judgment by the Administrator appropriately recognized that the CAA does not require that standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect the public welfare from known or anticipated adverse effects. Thus, based on the conclusions drawn from the air quality analyses which demonstrated a strong, positive relationship between the 8-hour and W126 metrics and the findings that indicated the significant amount of control provided by the fourth-high metric, the evidence base of O_3 effects on vegetation and her public welfare policy judgments, as well as public comments and CASAC advice, the Administrator decided to retain the existing form and averaging time and revise the level to 0.070 ppm, judging that such a standard would provide the requisite protection to the public welfare from any known or anticipated adverse effects associated with the presence of O_3 in ambient air (80 FR 65409-10, October 26, 2015).

As noted in Chapter 1, after publication of the final rule revising the standards, a number of industry groups, environmental and public health organizations, and certain states sought judicial review in the D.C. Circuit. On August 23, 2019, the court issued an opinion concluding, in relevant part, that EPA had not provided a sufficient rationale for aspects of its decision on the 2015 secondary standard (*Murray Energy Corp. v. EPA*, 936 F.3d 597 [D.C. Cir. 2019]). Accordingly, the court remanded the secondary standard to EPA for further justification or reconsideration, particularly in relation to its decision to focus on a 3-year average for consideration of the cumulative exposure, in terms of W126, identified as providing requisite public welfare protection, and its decision to not identify a specific level of air quality related to visible foliar injury.

4.2 GENERAL APPROACH AND KEY ISSUES IN THIS REVIEW

As is the case for all such reviews, this review of the secondary standard is most fundamentally based on using the Agency's assessment of the current 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. The approach planned for this review of the secondary O_3 standard will build on the last review, including the substantial assessments and evaluations performed over the course of that review, and taking into account the more recent scientific information and air quality data now available to inform understanding of the key policy-relevant issues in the current review. As noted above, we are also considering the court's recent decision on the O_3 secondary standard, recognizing that issues raised by the court in its remand of the standard (recognized in section 4.1.2 above) will be considered over the course of this review.

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 as to whether to retain or revise this standard. 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. In so doing, 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 in this review will inform the answer to the following initial overarching question for the review:

Do the currently 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 O_3 standard?

In reflecting on this question in the remaining sections of this chapter, we consider the available 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 last review regarding welfare effects related to exposure to O_3 in ambient air. Information available in this review 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 currently available exposure and risk information, whether newly developed in this review or predominantly developed in the past and interpreted in light of current information, is considered, including with regard to the extent to which it may continue to support judgments made in the last review. Further, in considering this question with regard to the secondary O_3 standard, we give particular attention to exposures and risks for effects with the greatest potential for public welfare significance.

The approach to reaching conclusions on the current secondary O_3 standard and, as appropriate, on potential alternative standards, including consideration of policy-relevant questions that frame the current review, is illustrated in Figure 4-1.

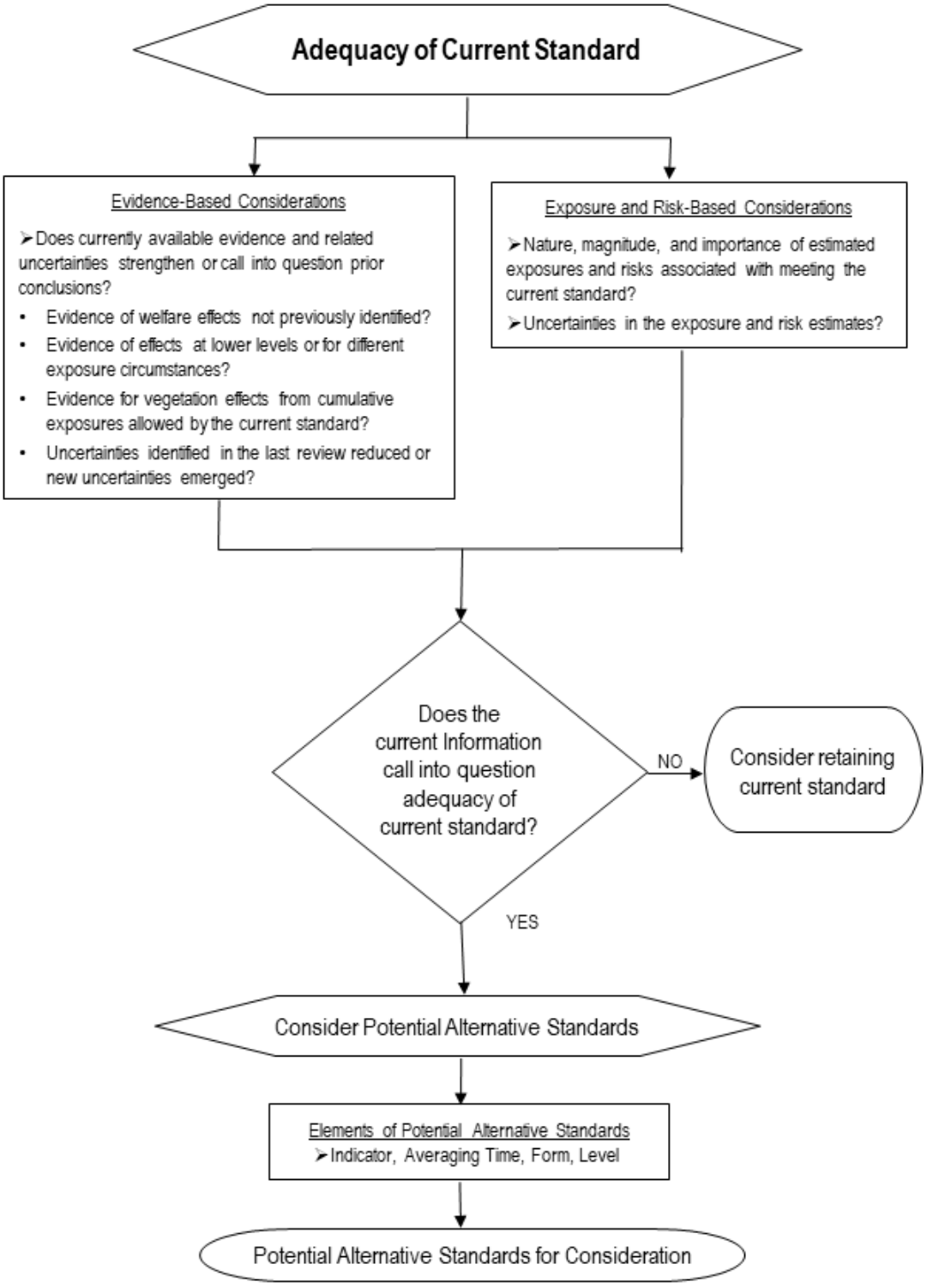

Figure 4-1. Overview of general approach for review of the secondary O_3 standard.

The Agency's approach in its review of secondary standards is consistent with the requirements of the provisions of the CAA related to the review of NAAQS and with how the EPA and the courts have historically interpreted the CAA. As discussed in section 1.2 above, these provisions require the Administrator to establish secondary standards that, in the Administrator's judgment, are requisite (i.e., neither more nor less stringent than necessary) to protect the public welfare from known or anticipated adverse effects associated with the presence of the pollutant in the ambient air. In so doing, the Administrator considers advice from the CASAC and public comment.

Consistent with the Agency's approach across all NAAQS reviews, the approach of this PA to informing the Administrator's judgments is based on a recognition that the available evidence generally reflects continuums that include ambient air exposures for which scientists generally agree that effects are likely to occur through lower levels at which the likelihood and magnitude of response become increasingly uncertain. The CAA does not require that standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect the public welfare from known or anticipated adverse effects. The Agency's decisions on the adequacy of the current secondary standard and, as appropriate, on any potential alternative standards considered in a review, are largely public welfare policy judgments made by the Administrator. The four basic elements of the NAAQS (i.e., indicator, averaging time, form, and level) are considered collectively in evaluating the protection afforded by the current standard, or any alternative standards considered. Thus, the Administrator's final decisions in such reviews draw upon the scientific information and analyses about welfare effects, environmental exposures and risks, and associated public welfare significance, as well as judgments about how to consider the range and magnitude of uncertainties that are inherent in the scientific evidence and analyses.

4.3 WELFARE EFFECTS EVIDENCE

4.3.1 Nature of Effects

The welfare effects evidence base available in the current review includes more than fifty years of extensive research on the phytotoxic effects of O_3, conducted both in and outside of the U.S., that documents the impacts of O_3 on plants and their associated ecosystems (1978 AQCD, 1986 AQCD, 1996 AQCD, 2006 AQCD, 2013 ISA, 2020 ISA). As was established in prior reviews, O_3 can interfere with carbon gain (photosynthesis) and allocation of carbon within the plant, making fewer carbohydrates available for plant growth, reproduction, and/or yield (1996 AQCD, pp. 5-28 and 5-29). For seed-bearing plants, reproductive effects can include reduced seed or fruit production or yield. The strongest evidence for effects from O_3 exposure on vegetation was recognized at the time of the last review to be from controlled exposure studies,

which "have clearly shown that exposure to O_3 is causally linked to visible foliar injury, decreased photosynthesis, changes in reproduction, and decreased growth" in many species of vegetation (2013 ISA, p. 1-15). Such effects at the plant scale can also be linked to an array of effects at larger spatial scales (and higher levels of biological organization), with the evidence available in the last review indicating that "O_3 exposures can affect ecosystem productivity, crop yield, water cycling, and ecosystem community composition" (2013 ISA, p. 1-15, Chapter 9, section 9.4). Beyond its effects on plants, the evidence in the last review also recognized O_3 in the troposphere as a major greenhouse gas (ranking behind carbon dioxide and methane in importance), with associated radiative forcing and effects on climate, with accompanying "large uncertainties in the magnitude of the radiative forcing estimate … making the impact of tropospheric O_3 on climate more uncertain than the effect of the longer-lived greenhouse gases (2013 ISA, sections 10.3.4 and 10.5.1 [p. 10-30]).

- **Does the current evidence alter conclusions from the last review regarding the nature of welfare effects attributable to O_3 in ambient air? Is there new evidence on welfare effects beyond those identified in the last review?**

The evidence newly available in this review supports, sharpens and expands somewhat on the conclusions reached in the last review (ISA, Appendices 8 and 9). Consistent with the evidence in the last review, the currently available evidence describes an array of O_3 effects on vegetation and related ecosystem effects, as well as the role of tropospheric O_3 in radiative forcing and subsequent climate-related effects. Evidence newly available in this review augments more limited previously available evidence related to insect interactions with vegetation, contributing to conclusions regarding O_3 effects on plant-insect signaling (ISA, Appendix 8, section 8.7) and on insect herbivores (ISA, Appendix 8, section 8.6), as well as for ozone effects on tree mortality (Appendix 8, section 8.4). Thus, conclusions reached in the last review are supported by the current evidence base and conclusions are also reached in a few new areas based on the now expanded evidence.

The current evidence base, including a wealth of longstanding evidence, supports the conclusion of causal relationships between O_3 and visible foliar injury, reduced vegetation growth and reduced plant reproduction,[11] as well as reduced yield and quality of agricultural crops, reduced productivity in terrestrial ecosystems, alteration of terrestrial community composition[12], and alteration of belowground biogeochemical cycles (ISA, section IS.5). Based on the current evidence base, the ISA also concluded there likely to be a causal relationship

[11] 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 O_3 with reduced vegetation growth (ISA, Table IS-12).

[12] The 2013 ISA concluded alteration of terrestrial community composition to be likely causally related to O_3 based on the then available information (ISA, Table IS-12),

between O_3 and alteration of ecosystem water cycling, reduced carbon sequestration in terrestrial ecosystems, and with increased tree mortality (ISA, section IS.5). Additional evidence newly available in this review is concluded by the ISA to support conclusions on two additional plant-related effects: the body of evidence is concluded to be sufficient to infer that there is likely to be a causal relationship between O_3 exposure and alteration of plant-insect signaling, and to infer that there is likely to be a causal relationship between O_3 exposure and altered insect herbivore growth and reproduction (ISA, Table IS-12).

As in the last review, the strongest evidence and the associated findings of causal or likely causal relationships with O_3 in ambient air, and the quantitative characterizations of relationships between O_3 exposure and occurrence and magnitude of effects are for vegetation effects. The scales of these effects range from the individual plant scale to the ecosystem scale, with potential for impacts on the public welfare (as discussed in section 4.3.2 below). The following summary addresses the identified vegetation-related effects of O_3 across these scales.

The current evidence, consistent with the decades of previously available evidence, documents and characterizes visible foliar injury in many tree, shrub, herbaceous, and crop species as an effect of exposure to O_3 (ISA, Appendix 8, section 8.2; 2013 ISA, section 9.4.2; 2006 AQCD, 1996 AQCD, 1986 AQCD, 1978 AQCD). As recognized in the last review with regard to the then-available evidence, "[r]ecent experimental evidence continues to show a consistent association between visible injury and ozone exposure" (ISA, Appendix 8, section 8.2, p. 8-13; 2013 ISA, section 9.4.2, p. 9-41). Ozone-induced visible foliar injury symptoms on certain tree and herbaceous species, such as black cherry, yellow-poplar and common milkweed, have long been considered diagnostic of exposure to elevated O_3 based on the consistent association established with experimental evidence (ISA, Appendix 8, section 8.2; 2013 ISA, p. 1-10).[13]

The currently available evidence, consistent with that in past reviews, indicates that "visible foliar injury usually occurs when sensitive plants are exposed to elevated ozone concentrations in a predisposing environment," with a major factor for such an environment being the amount of soil moisture available to the plant (ISA, Appendix 8, p. 8-23; 2013 ISA, section 9.4.2). Further, the significance of O_3 injury at the leaf and whole plant levels also depends on an array of factors that include the amount of total leaf area affected, age of plant, size, developmental stage, and degree of functional redundancy among the existing leaf area

[13] 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).

(ISA, Appendix 8, section 8.2; 2013 ISA, section 9.4.2). In this review, as in the past, such modifying factors contribute to the difficulty in quantitatively relating visible foliar injury to other vegetation effects (e.g., individual tree growth, or effects at population or ecosystem levels), such that visible foliar injury "is not always a reliable indicator of other negative effects on vegetation" (ISA, Appendix 8, section 8.2; 2013 ISA, p. 9-39).[14]

Consistent with conclusions in past reviews, the evidence, extending back several decades, continues to document the detrimental effects of O_3 on plant growth and reproduction (ISA, Appendix 8, sections 8.3 and 8.4; 2013 ISA, p. 9-42). The available studies come from a variety of different study types that cover an array of different species, effects endpoints, and exposure methods and durations. In addition to studies on scores of plant species that have found O_3 to reduce plant growth, the evidence accumulated over the past several decades documents O_3 alteration of allocation of biomass within the plant and plant reproduction (ISA, Appendix 8, sections 8.3 and 8.4; 2013 ISA, p. 1-10). The biological mechanisms underlying the effect of O_3 on plant reproduction include "both direct negative effects on reproductive tissues and indirect negative effects that result from decreased photosynthesis and other whole plant physiological changes" (ISA, section IS.5.1.2). A newly available meta-analysis of more than 100 studies published between 1968 and 2010 summarizes effects of O_3 on multiple measures of reproduction (ISA, Appendix 8, section 8.4.1).

Studies involving experimental field sites have also reported effects on measures of plant reproduction, such as effects on seeds (reduced weight, germination, and starch levels) that could lead to a negative impact on species regeneration in subsequent years, and bud size that might relate to a delay in spring leaf development (ISA, Appendix 8, section 8.4; 2013 ISA, section 9.4.3; Darbah et al., 2007, Darbah et al., 2008). A more recent laboratory study reported 6-hour daily O_3 exposures of flowering mustard plants to 100 ppb during different developmental stages to have mixed effects on reproductive metrics. While flowers exposed early *versus* later in development produced shorter fruits, the number of mature seeds per fruit was not significantly affected by flower developmental stage of exposure (ISA, Appendix 8, section 8.4.1; Black et al.,

[14] Similar to the 2013 ISA, the ISA for the current review 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.

2012). Another study assessed seed viability for a flowering plant in laboratory and field conditions, finding effects on seed viability of O_3 exposures (90 and 120 ppb) under laboratory conditions but less clear effects under more field-like conditions (ISA, Appendix 8, section 8.4.1; Landesmann et al., 2013).

With regard to agricultural crops, the current evidence base, as in the last review, is sufficient to infer a causal relationship between O_3 exposure and reduced yield and quality (ISA, section IS.5.1.2). The current evidence is augmented by new research in a number of areas, including studies on soybean, wheat and other nonsoy legumes. The new information assessed in the ISA remains consistent with the conclusions reached in the 2013 ISA (ISA, section IS.5.1.2).

The evidence base for trees includes a number of studies conducted at the Aspen free-air carbon-dioxide and ozone enrichment (FACE) experiment site in Wisconsin (that operated from 1998 through 2011) and also available in the last review (ISA, IS.5.1 and Appendix 8, section 8.1.2.1; 2013 ISA, section 9.2.4). These studies, which occurred in a field setting (more similar to natural forest stands than open-top-chamber studies), reported reduced tree growth when grown in single or three species stands within 30-m diameter rings and exposed over one or more years to elevated O_3 concentrations (hourly concentrations 1.5 times concentrations in ambient air at the site) compared to unadjusted ambient air concentrations (2013 ISA, section 9.4.3; Kubiske et al., 2006, Kubiske et al., 2007).[15]

With regard to tree mortality, the 2013 ISA did not include a determination of causality (ISA, Appendix 8, section 8.4). While the then-available evidence included studies identifying ozone as a contributor to tree mortality, which contributed to the 2013 conclusion regarding O_3 and alteration of community composition (2013 ISA, section 9.4.7.4), a separate causality determination regarding O_3 and tree mortality was not assessed (ISA, Appendix 8, section 8.4; 2013 ISA, Table 9-19). The evidence assessed in the 2013 ISA (and 2006 AQCD) was largely observational, including studies that reported declines in conifer forests for which elevated O_3 was identified as contributor but in which a variety of environmental factors may have also played a role (2013 ISA, section 9.4.7.1; 2006 AQCD, sections AX9.6.2.1, AX9.6.2.2, AX9.6.2.6, AX9.6.4.1 and AX9.6.4.2). Since the last review, three additional studies are now available (ISA, Appendix 8, Table 8-9). Two of these are analyses of field observations, one of

[15] Seasonal (90-day) W126 index values for unadjusted O_3 concentrations over six years of the Aspen FACE experiments ranged from 2 to 3 ppm-hrs, while the elevated exposure concentrations (reflecting addition of O_3 to ambient air concentrations) ranged from somewhat above 20 to somewhat above 35 ppm-hrs (ISA, Appendix 8, Figure 8-17).

which is set in the Spanish Pyrenees.[16] A second study is a large-scale empirical statistical analysis of factors potentially contributing to tree mortality in eastern and central U.S. forests during the 1971-2005 period, which reported O_3 (county-level 11-year [1996-2006] average 8 hour metric)[17] to be ninth among the 13 potential factors assessed[18] and to have a significant positive correlation with tree mortality (ISA, section IS.5.2, Appendix 8, section 8.4.3; Dietze and Moorcroft, 2011). A newly available experimental study also reported increased mortality in two of five aspen genotypes grown in mixed stands under elevated O_3 concentrations (ISA, section IS.5.1.2; Moran and Kubiske, 2013). Coupled with the plant-level evidence of phytotoxicity discussed above, as well as consideration of community composition effects, this evidence was concluded to indicate the potential for elevated O_3 concentrations to contribute to tree mortality (ISA, section IS.5.1.2 and Appendix 8, sections 8.4.3 and 8.4.4). Based on the current evidence, the ISA concludes there is likely to be a causal relationship between O_3 and increased tree mortality (ISA, Table IS-2, Appendix 8, section 8.4.4).

A variety of factors in natural environments can either mitigate or exacerbate predicted O_3-plant interactions and are recognized sources of uncertainty and variability. Such factors at the plant level include multiple genetically influenced determinants of O_3 sensitivity, changing sensitivity to O_3 across vegetative growth stages, co-occurring stressors and/or modifying environmental factors (ISA, Appendix 8, section 8.12).

Ozone-induced effects at the scale of the whole plant have the potential to translate to effects at the ecosystem scale, such as reduced productivity and carbon storage, and altered terrestrial community composition, as well as impacts on ecosystem functions, such as belowground biogeochemical cycles and ecosystem water cycling. For example, under the relevant exposure conditions, O_3-related reduced tree growth and reproduction, as well as increased mortality, could lead to reduced ecosystem productivity. Recent studies from the Aspen FACE experiment and modeling simulations indicate that O_3-related negative effects on ecosystem productivity may be temporary or may be limited in some systems (ISA, Appendix 8,

[16] The concentration gradient with altitude in the Spanish study, includes - at the highest site - annual average April-to-September O_3 concentrations for the 2004 to 2007 period that range up to 74 ppb (Diaz-de-Quijano et al., 2016), indicating O_3 concentrations likely to exceed the current U.S. secondary standard.

[17] As indicated in Figures 2-11 and 2-12, annual fourth highest daily maximum 8-hour O_3 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.

[18] 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, O_3 concentration, tree basal area, topographic moisture index, slope and topographic radiation index (Dietze and Moorcroft, 2011).

section 8.8.1). Previously available studies had reported impacts on productivity in some forest types and locations, such as ponderosa pine in southern California and other forest types in the mid-Atlantic region (2013 ISA, section 9.4.3.4). Through reductions in sensitive species growth, and related ecosystem productivity, O_3 could lead to reduced ecosystem carbon storage (ISA, IS.5.1.4; 2013 ISA, section 9.4.3). With regard to forest community composition, available studies have reported changes in tree communities composed of species with relatively greater and relatively lesser sensitivity to O_3, such as birch and aspen, respectively (ISA, section IS.5.1.8.1, Appendix 8, section 8.10; 2013 ISA, section 9.4.3; Kubiske et al., 2007). As the ISA concludes, "[t]he extent to which ozone affects terrestrial productivity will depend on more than just community composition, but other factors, which both directly influence [net primary productivity] (i.e., availability of N and water) and modify the effect of ozone on plant growth" (ISA, Appendix 8, section 8.8.1). Thus, the magnitude of O_3 impact on ecosystem productivity, as on forest composition, can vary among plant communities based on several factors, including the type of stand or community in which the sensitive species occurs (e.g., single species *versus* mixed canopy), the role or position of the species in the stand (e.g., dominant, sub-dominant, canopy, understory), and the sensitivity of co-occurring species and environmental factors (e.g., drought and other factors).

The effects of O_3 on plants and plant populations have implications for ecosystem functions. Two such functions, effects with which O_3 is concluded to be likely causally or causally related, are ecosystem water cycling and belowground biogeochemical cycles, respectively (ISA, Appendix 8, sections 8.11 and 8.9). With regard to the former, the effects of O_3 on plants (e.g., *via* stomatal control, as well as leaf and root growth and changes in wood anatomy associated with water transport) can affect ecosystem water cycling through impacts on root uptake of soil moisture and groundwater as well as transpiration through leaf stomata to the atmosphere (ISA, Appendix 8, section 8.11.1). These "impacts may in turn affect the amount of water moving through the soil, running over land or through groundwater and flowing through streams" (ISA, Appendix 8, section 8.11, p. 8-161). Evidence newly available in this review is supportive of previously available evidence in this regard (ISA, Appendix 8, section 8.11.6). The current evidence, including that newly available, indicates the extent to which the effects of O_3 on plant leaves and roots (e.g., through effects on chemical composition and biomass) can impact belowground biogeochemical cycles involving root growth, soil food web structure, soil decomposer activities, soil microbial respiration, soil carbon turnover, soil water cycling and soil nutrient cycling (ISA, Appendix 8, section 8.9).

Additional vegetation-related effects with implications beyond individual plants include the effects of O_3 on insect herbivore growth and reproduction and plant-insect signaling (ISA, Table IS-12, Appendix 8, sections 8.6 and 8.7). With regard to insect herbivore growth and

reproduction, the evidence includes multiple effects in an array of insect species, although without a consistent pattern of response for most endpoints (ISA, Appendix 8, Table 8-11). As was also the case with the studies available at the time of the last review,[19] in the newly available studies the individual-level responses are highly context- and species-specific and not all species tested showed a response (ISA, p. IS-64, Table IS-12, section IS.5.1.3 and Appendix 8, section 8.6). Evidence on plant-insect signaling that is newly available in this review comes from laboratory, greenhouse, open top chambers (OTC) and FACE experiments (ISA, section IS.5.1.3 and Appendix 8, section 8.7). The available evidence indicates a role for elevated O_3 in altering and degrading emissions of chemical signals from plants and reducing detection of volatile plant signaling compounds (VPSCs) by insects, including pollinators. Elevated O_3 concentrations degrade some VPSCs released by plants, potentially affecting ecological processes including pollination and plant defenses against herbivory. Further, the available studies report elevated O_3 conditions to be associated with plant VPSC emissions that may make a plant either more attractive or more repellant to herbivorous insects, and to predators and parasitoids that target phytophagous (plant-eating) insects (ISA, section IS.5.1.3 and Appendix 8, section 8.7).

Ozone welfare effects also extend beyond effects on vegetation and associated biota due to it being a major greenhouse gas and radiative forcing agent.[20] As in the last review, the current evidence, augmented since the 2013 ISA, continues to support a causal relationship between the global abundance of O_3 in the troposphere and radiative forcing, and a likely causal relationship between the global abundance of O_3 in the troposphere and effects on temperature, precipitation, and related climate variables[21] (ISA, section IS.5.2 and Appendix 9; Myhre et al., 2013). As was also true at the time of the last review, tropospheric O_3 has been ranked third in importance for global radiative forcing, after carbon dioxide and methane, with the radiative forcing of O_3 since pre-industrial times estimated to be about 25 to 40% of the total warming effects of anthropogenic carbon dioxide and about 75% of the effects of anthropogenic methane (ISA, Appendix 9, section 9.1.3.3). Uncertainty in the magnitude of radiative forcing estimated to be attributed to tropospheric O_3 is a contributor to the relatively greater uncertainty associated with

[19] During the last review, the 2013 ISA stated with regard to O_3 effects on insects and other wildlife that "there is no consensus on how these organisms respond to elevated O_3 (2013 ISA, section 9.4.9.4, p. 9-98).

[20] 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).

[21] 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).

climate effects of tropospheric O_3 compared to such effects of the well mixed greenhouse gases, such as carbon dioxide and methane (ISA, section IS.6.2.2).

Lastly, the evidence regarding tropospheric O_3 and UV-B shielding was evaluated in the 2013 ISA and determined to be inadequate to draw a causal conclusion (2013 ISA, section 10.5.2). The current ISA concludes there to be no new evidence since the 2013 ISA relevant to the question of UV-B shielding by tropospheric O_3 (ISA, IS.1.2.1 and Appendix 9, section 9.1.3.4).

4.3.2 Public Welfare Implications

The public welfare implications of the evidence regarding O_3 welfare effects are dependent on the type and severity of the effects, as well as the extent of the effect at a particular biological or ecological level of organization. We discuss such factors here in light of judgments and conclusions made in prior reviews regarding effects on the public welfare.

As provided in section 109(b)(2) of the CAA, the secondary standard is to "specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator … is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air." The secondary standard is not meant to protect against all known or anticipated O_3-related welfare effects, but rather those that are judged to be adverse to the public welfare, and a bright-line determination of adversity is not required in judging what is requisite (78 FR 3212, January 15, 2013; 80 FR 65376, October 26, 2015; see also 73 FR 16496, March 27, 2008). Thus, the level of protection from known or anticipated adverse effects to public welfare that is requisite for the secondary standard is a public welfare policy judgment to be made by the Administrator. In each review, the Administrator's judgment regarding the currently available information and adequacy of protection provided by the current standard is generally informed by considerations in prior reviews and associated conclusions.

- **Is there information newly available in this review relevant to consideration of the public welfare implications of O_3-related welfare effects?**

The categories of effects identified in the CAA to be included among welfare effects are quite diverse,[22] and among these categories, any single category includes many different types of effects that are of broadly varying specificity and level of resolution. For example, effects on vegetation, is a category identified in CAA section 302(h), and the ISA recognizes numerous

[22] 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)).

vegetation-related effects of O_3 at the organism, population, community and ecosystem level, as summarized in section 4.3.1 above (ISA, Appendix 8). The significance of each type of vegetation-related effect with regard to potential effects on the public welfare depends on the type and severity of effects, as well as the extent of such effects on the affected environmental entity, and on the societal use of the affected entity and the entity's significance to the public welfare. For example, a key consideration with regard to public welfare implications in prior reviews of the O_3 secondary standard was the intended use of the affected or sensitive vegetation and the significance of the vegetation to the public welfare (73 FR 16496, March 27, 2008; 80 FR 65292, October 26, 2015).

More specifically, judgments regarding public welfare significance in the last two O_3 NAAQS decisions gave particular attention to O_3 effects in areas with special federal protections, and lands set aside by states, tribes and public interest groups to provide similar benefits to the public welfare (73 FR 16496, March 27, 2008; 80 FR 65292, October 26, 2015). For example, in the decision to revise the secondary standard in the 2008 review, the Administrator took note of "a number of actions taken by Congress to establish public lands that are set aside 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). As further recognized in the 2008 notice, "[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).[23,24] Such areas include Class I areas[25] which are federally mandated to preserve certain air quality related values. Additionally, as the Administrator recognized, "States, Tribes and public interest groups also set aside areas that are intended to provide similar benefits to the public welfare, for residents on State and Tribal lands, as well as for visitors to those areas" (73 FR 16496, March 27, 2008). The Administrator took note of the "clear public interest in and value of maintaining these areas in a condition that does

[23] 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).

[24] As a second example, 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)).

[25] Areas designated as Class I include all international parks, national wilderness areas which exceed 5,000 acres in size, national memorial parks which exceed 5,000 acres in size, and national parks which exceed six thousand acres in size, provided the park or wilderness area was in existence on August 7, 1977. Other areas may also be Class I if designated as Class I consistent with the CAA.

not impair their intended use and the fact that many of these lands contain O_3-sensitive species" (73 FR 16496, March 27, 2008). Similarly, in the 2015 review, the Administrator indicated particular concern for O_3-related effects on plant function and productivity and associated ecosystem effects in natural ecosystems "such as those in areas with protection designated by Congress for current and future generations, as well as areas similarly set aside by states, tribes and public interest groups with the intention of providing similar benefits to the public welfare" (80 FR 65403, October 26, 2015).

The 2008 and 2015 decision notices recognized that the degree to which effects on vegetation in specially protected areas, such as those identified above, may be judged adverse involves considerations from the species level to the ecosystem level, such that judgments can depend on the intended use for, or service (and value) of, the affected vegetation, ecological receptors, ecosystems and resources and the significance of that use to the public welfare (73 FR 16496, March 27, 2008; 80 FR 65377, October 26, 2015). Uses or services provided by areas that have been afforded special protection can flow in part or entirely from the vegetation that grows there. For example, ecosystem services are the "benefits that people derive from functioning ecosystems" (Costanza et al., 2017; ISA, section IS.5.1).[26] Ecosystem services range from those directly related to the natural functioning of the ecosystem to ecosystem uses for human recreation or profit, such as through the production of lumber or fuel (Costanza et al., 2017). Aesthetic value and outdoor recreation depend, at least in part, on the perceived scenic beauty of the environment. Further, there have been analyses that report the American public values – in monetary as well as nonmonetary ways – the protection of forests from air pollution damage (Haefele et al., 1991). In fact, public surveys have indicated that Americans rank as very important the existence of resources, the option or availability of the resource and the ability to bequest or pass it on to future generations (Cordell et al., 2008). The spatial, temporal and social dimensions of public welfare impacts are also influenced by the type of service affected. For example, a national park can provide direct recreational services to the thousands of visitors that come each year, but also provide an indirect value to the millions who may not visit but receive satisfaction from knowing it exists and is preserved for the future (80 FR 65377, October 26, 2015).

The different types of effects on vegetation discussed in section 4.3.1 above differ with regard to aspects important to judging their public welfare significance. In the case of crop yield loss, such judgments depend on considerations related to the heavy management of agriculture in

[26] Ecosystem services analyses were one of the tools used in the last review of the secondary standards for oxides of nitrogen and sulfur to inform the decisions made with regard to adequacy of protection provided by the standards and as such, were used in conjunction with other considerations in the discussion of adversity to public welfare (77 FR 20241, April 3, 2012).

the U.S., while judgments for other categories of effects may generally relate to considerations regarding forested areas, including specifically those not managed for harvest. For example, effects on tree growth and reproduction, and also visible foliar injury, have the potential to be significant to the public welfare through impacts in Class I and other areas given special protection in their natural/existing state, although they differ in how they might be significant.

As described in section 4.3.1 above, O_3 effects on tree growth and reproduction could, depending on severity, extent and other factors, lead to effects on a larger scale including reduced productivity, altered forest and forest community (plant, insect and microbe) composition, reduced carbon storage and altered ecosystem water cycling (ISA, section IS.5.1.8.1; 2013 ISA, Figure 9-1, sections 9.4.1.1 and 9.4.1.2). For example, forest or forest community composition can be affected through O_3 effects on growth and reproductive success of sensitive species in the community, with the extent of compositional changes dependent on factors such as competitive interactions (ISA, section IS.5.1.8.1; 2013 ISA, sections 9.4.3 and 9.4.3.1). Impacts on some of these characteristics (e.g., forest or forest community composition) may be considered of greater public welfare significance when occurring in Class I or other protected areas, due to value for particular services that the public places on such areas.

Depending on the type and location of the affected ecosystem, however, a broader array of services benefitting the public can be affected in a broader array of areas well. For example, other services valued by people that can be affected by reduced tree growth, productivity and associated forest effects include aesthetic value, food, fiber, timber, other forest products, habitat, recreational opportunities, climate and water regulation, erosion control, air pollution removal, and desired fire regimes, as summarized in Figure 4-2 (ISA, section IS.5.1; 2013 ISA, sections 9.4.1.1 and 9.4.1.2). In the decisions to revise the secondary standard in the last two reviews, the Administrator recognized that by providing protection based on consideration of effects in natural ecosystems in areas afforded special protection, the revised secondary standard would also "provide a level of protection for other vegetation that is used by the public and potentially affected by O_3 including timber, produce grown for consumption and horticultural plants used for landscaping" (80 FR 65403, October 26, 2015). For example, locations potentially vulnerable to O_3-related impacts but not necessarily identified for special protection might be forested lands, both public and private, where trees are grown for timber production. Forests in urbanized areas also provide a number of services that are important to the public in those areas, such as air pollution removal, cooling, and beautification. There are also many other tree species, such as various ornamental and agricultural species (e.g., Christmas trees, fruit and nut trees), that provide ecosystem services that may be judged important to the public welfare.

Depending on its severity and spatial extent, visible foliar injury, which affects the physical appearance of the plant, also has the potential to be significant to the public welfare

through impacts in Class I and other similarly protected areas. In cases of widespread and severe injury during the growing season (particularly when sustained across multiple years, and accompanied by obvious impacts on the plant canopy), O_3-induced visible foliar injury might be expected to have the potential to impact the public welfare in scenic and/or recreational areas, particularly in areas with special protection, such as Class I areas.[27] The ecosystem services most likely to be affected by O_3-induced visible foliar injury (some of which are also recognized above for tree growth-related effects) are cultural services, including aesthetic value and outdoor recreation.

The geographic extent of protected areas that may be vulnerable to public welfare effects of O_3, such as impacts to outdoor recreation, is potentially appreciable. For example, biomonitoring surveys that were routinely administered by the U.S. Forest Service (USFS) as far back as 1994 in the eastern U.S. and 1998 in the western U.S. include many field sites at which there are plants sensitive to O_3-related visible foliar injury; there are 450 field sites across 24 states in the North East and North Central regions (Smith, 2012).[28] Since visible foliar injury is a visible indication of O_3 exposure in species sensitive to this effect, a number of such species have been established as bioindicator species, and such surveys have been used by federal land managers as tools in assessing potential air quality impacts in Class I areas (U.S. Forest Service, 2010). Additionally, the USFS has developed categories for the scoring system they use for purposes of describing and comparing injury severity at biomonitoring sites. The sites are termed biosites and the scoring system involves deriving biosite index scores that may be described with regard to one of several categories. For example, biosite index scores of zero to five are described as "little or no foliar injury," scores above five to 15 as "low" or "light to moderate" foliar injury, scores from 15 to 25 as "moderate foliar injury" and scores above 25 as "severe injury" (Campbell et al., 2007; Smith et al., 2007; Smith, 2012).[29] As noted in section 4.3.1 above, there is not an established quantitative relationship between visible foliar injury and other

[27] 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 likelihood for reduced recreational use in areas with stands of pine in which moderate to severe injury was apparent from 30 or 40 feet.

[28] This aspect of the USFS biomonitoring surveys has apparently been suspended, with the most recent surveys conducted in 2011 (USFS, 2013, USFS, 2017).

[29] Early in the USFS biomonitoring program data, there were suggestions of what might be considered "assumptions of risk" 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).

effects, such as reduced growth and productivity as visible foliar injury "is not always a reliable indicator of other negative effects" (ISA, Appendix 8, section 8.2).

Public welfare implications associated with visible foliar injury might further be considered to relate largely to effects on scenic and aesthetic values. The available information does not yet address or describe the relationships expected to exist between some level of injury severity (e.g., little, low/light, moderate or severe) and/or spatial extent affected and scenic or aesthetic values. This gap impedes consideration of the public welfare implications of different injury severities, and accordingly judgments on the potential for public welfare significance. That notwithstanding, some level of severity and widespread occurrence of visible foliar injury, particularly if occurring in specially protected areas, such as Class I areas, where the public can be expected to place value (e.g., for recreational uses), might reasonably be concluded to impact the public welfare. Thus, key considerations for public welfare significance of this endpoint in past reviews have related to qualitative consideration of the potential for such effects to affect the aesthetic value of plants in protected areas, such as Class I areas (73 FR 16490, March 27, 2008).

While, as noted above, public welfare benefits of forested lands can be particular to the type of area in which the forest occurs, some of the potential public welfare benefits associated with forest ecosystems are not location dependent. A potentially extremely valuable ecosystem service provided by forested lands is carbon sequestration or storage (ISA, section IS.5.1.4 and Appendix 8, section 8.8.3; 2013 ISA, section 2.6.2.1 and p. 9-37).[30] As noted above, the EPA has concluded that effects on this ecosystem service are likely causally related to O_3 in ambient air (ISA, Table IS-12). The importance of carbon sequestration to the public welfare relates to its role in counteracting the impact of greenhouse gases on radiative forcing and related climate effects. As summarized in section 4.3.1 above, O_3 is also a greenhouse gas and O_3 abundance in the troposphere is causally related to radiative forcing and likely causally related to subsequent effects on temperature, precipitation and related climate variables (ISA, section IS.6.2.2). Accordingly, such effects also have important public welfare implications, although their quantitative evaluation in response to O_3 concentrations in the U.S. is complicated by "[c]urrent limitations in climate modeling tools, variation across models, and the need for more comprehensive observational data on these effects" (ISA, section IS.6.2.2). The service of carbon storage is of paramount importance to the public welfare no matter in what location the trees are growing or what their intended current or future use (e.g., 2013 ISA, section 9.4.1.2). In other words, the benefit exists as long as the trees are growing, regardless of what additional functions and services it provides.

[30] 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 other plants.

With regard to agriculture-related effects, the EPA has recognized other complexities related to areas and plant species that are heavily managed to obtain a particular output (such as commodity crops or commercial timber production). For example, the EPA has recognized that the degree to which O_3 impacts on vegetation that could occur in such areas and on such species would impair the intended use at a level that might be judged adverse to the public welfare has been less clear (80 FR 65379, October 26, 2015; 73 FR 16497, March 27, 2008). While having sufficient crop yields is of high public welfare value, important commodity crops are typically heavily managed to produce optimum yields. Moreover, based on the economic theory of supply and demand, increases in crop yields would be expected to result in lower prices for affected crops and their associated goods, which would primarily benefit consumers. These competing impacts on producers and consumers complicate consideration of these effects in terms of potential adversity to the public welfare (2014 WREA, sections 5.3.2 and 5.7). When agricultural impacts or vegetation effects in other areas are contrasted with the emphasis on forest ecosystem effects in Class I and similarly protected areas, it can be seen that the Administrator has in past reviews judged the significance to the public welfare of O_3-induced effects on sensitive vegetation growing within the U.S. to differ depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located, with greater significance ascribed to areas identified for specific uses and benefits to the public welfare, such as Class I areas, than to areas for which such uses have not been established (80 FR 65292, October 26, 2015; FR 73 16496-16497, March 27, 2008).

Categories of effects newly identified as likely causally related to O_3 in ambient air, such as alteration of plant-insect signaling and insect herbivore growth and reproduction, also have potential public welfare implications. For example, given the role of plant-insect signaling in such important ecological processes as insect herbivore growth and reproduction. The potential to contribute to adverse effects to the public welfare, e.g., given the role of the plant-insect signaling process in pollination and seed dispersal, as well as natural plant defenses against predation and parasitism, particular effects on particular signaling processes can be seen to have the potential for adverse effects on the public welfare (ISA, section IS.5.1.3).However, uncertainties and limitations in the current evidence (e.g., as summarized in sections 4.3.3.3 and 4.3.4 below) preclude an assessment of the extent and magnitude of O_3 effects on these endpoints, which thus also precludes an evaluation of the potential for associated public welfare implications, particularly under exposure conditions expected to occur in areas meeting the current standard.

In summary, several considerations are recognized as important to judgments on the public welfare significance of the array of effects of different O_3 exposure conditions on

vegetation. While there are uncertainties and limitations associated with the consideration of the magnitude of key vegetation effects that might be concluded to be adverse to ecosystems and associated services, there are numerous locations where the presence of O_3-sensitive tree species may contribute to a vulnerability to impacts from O_3 on tree growth, productivity and carbon storage and their associated ecosystems and services. Exposures that may elicit effects and the significance of the effects in specific situations can vary due to differences in exposed species sensitivity, the severity and associated significance of the observed or predicted O_3-induced effect, the role that the species plays in the ecosystem, the intended use of the affected species and its associated ecosystem and services, the presence of other co-occurring predisposing or mitigating factors, and associated uncertainties and limitations.

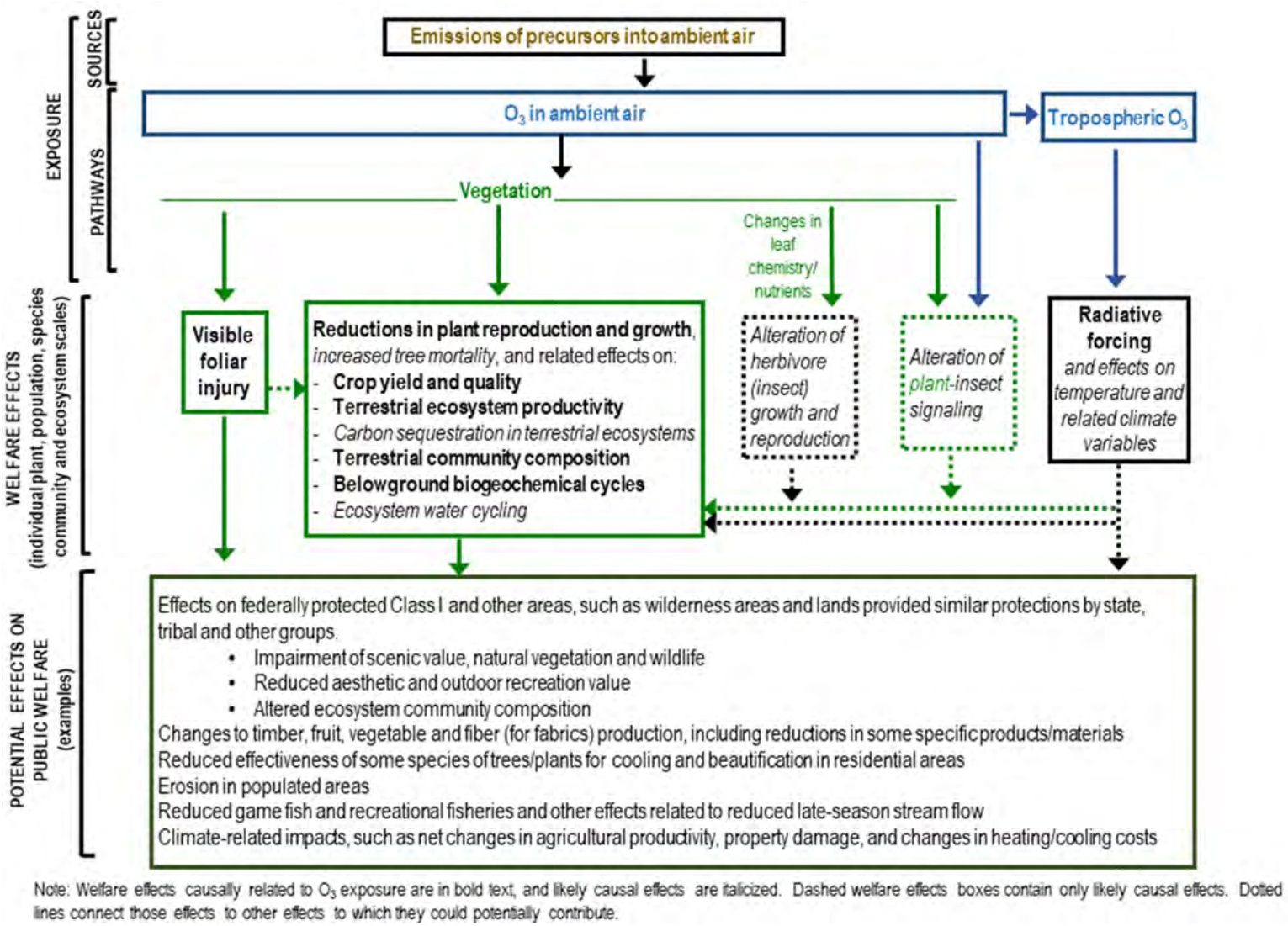

Figure 4-2. Potential effects of O_3 on the public welfare.

4.3.3 Exposures Associated with Effects

The types of effects identified in section 4.3.1 above vary widely with regard to the extent and level of detail of the available information that describes the O_3 exposure circumstances that may elicit them. The discussion in this section is organized in recognition of this. We focus first on growth and yield effects, a category of effects for which the information on exposure metric and E-R relationships is most advanced (section 4.3.3.1). Section 4.3.3.2 discusses the current information regarding exposure metrics and relationships between exposure and the occurrence and severity of visible foliar injury. The availability of such information for other categories of effects is addressed in section 4.3.3.3.

4.3.3.1 Growth-related Effects

4.3.3.1.1 Exposure Metric

The longstanding body of vegetation effects evidence includes a wealth of information on aspects of O_3 exposure that are important in influencing effects on plant growth and yield (1996 AQCD; 2006 AQCD; 2013 ISA; 2020 ISA). A variety of factors have been investigated, including "concentration, time of day, respite time, frequency of peak occurrence, plant phenology, predisposition, etc." (2013 ISA, section 9.5.2), and the importance of the duration of the exposure as well as the relatively greater importance of higher concentrations over lower concentrations have been consistently well documented (2013 ISA, section 9.5.3). Based on the associated improved understanding of the biological basis for plant response to O_3 exposure, a number of mathematical approaches have been developed for summarizing O_3 exposure for the purpose of assessing effects on vegetation, including those that cumulate exposures over some specified period while weighting higher concentrations more than lower (2013 ISA, sections 9.5.2 and 9.5.3; ISA, Appendix 8, section 8.2.2.2).

In the last several reviews, based on the then-available evidence, as well as advice from the CASAC, the EPA focused on the use of a cumulative, seasonal[31] concentration-weighted index for considering the growth-related effects evidence and in quantitative exposure analyses for purposes of reaching conclusions on the secondary standard. More specifically, the Agency used the W126-based cumulative, seasonal metric (80 FR 65404, October 26, 2015; ISA, section IS.3.2, Appendix 8, section 8.13). This metric, commonly called the W126 index, is a non-threshold approach described as the sigmoidally weighted sum of all hourly O_3 concentrations observed during a specified daily and seasonal time window, where each hourly O_3 concentration

[31] 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 O_3-sensitive vegetation, not to the seasons of the year (spring, summer, fall, winter).

is given a weight that increases from zero to one with increasing concentration (2013 ISA, p. 9-101).

Across the last several reviews of the O_3 NAAQS, several different exposure metrics have been evaluated, primarily for their ability to summarize ambient air concentrations in a way that best correlates with effects on vegetation, particularly growth-related effects. Based on extensive review of the published literature on different types of E-R metrics, including comparisons between metrics, the EPA has generally focused on cumulative, concentration-weighted indices of exposure, recognizing them as the most appropriate biologically based metrics to consider in this context (1996 AQCD; 2006 AQCD; 2013 ISA).[32] Quantifying exposure in this way has been found to improve the explanatory power of E-R models for growth and yield over using indices based only on mean and peak exposure values (2013 ISA, section 2.6.6.1, p. 2-44). The most well-studied datasets in this regard are those for 11 tree species seedlings and 10 crops referenced above and described further in section 4.3.3.2 below (e.g., Lee and Hogsett, 1996, Hogsett et al., 1997). The most detailed and well analyzed information in this regard are two datasets established two decades ago (and described in section 4.3.3.1.2 below), one for growth effects on seedlings of a set of tree species and the second for quality and yield effects for a set of crops. These datasets, which include growth and yield response information across a range of multiple seasonal cumulative exposures, were used to develop robust quantitative E-R functions for reduced growth (termed relative biomass loss or RBL) in seedlings of the tree species and E-R functions for RYL for a set of common crops (ISA, Appendix 8, section 8.13.2; 2013 ISA, section 9.6.2). The EPA's conclusions regarding exposure levels of O_3 associated with vegetation-related effects at the time of the last review were based primarily on these established E-R functions.

Along with the continuous weighted, W126 index, two other cumulative indices that have received greatest attention across the past several O_3 NAAQS reviews have been the threshold

[32] 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 O_3 exposure concentrations with regard to potential vegetation effects, particularly growth, has received strong support from CASAC in the past two reviews (Henderson, 2006; Samet, 2010; Frey, 2014).

weighted indices, AOT60[33] and SUM06.[34] Accordingly, some studies of O_3 vegetation effects have reported exposures using these metrics.

Alternative methods for characterizing O_3 exposure to predict various plant responses have, in recent years, included flux models (models that are based on the amount of O_3 that enters the leaf). However, as was the case in the last review, there remain a variety of complications, limitations and uncertainties associated with this approach. For example, "[w]hile some efforts have been made in the U.S. to calculate ozone flux into leaves and canopies, little information has been published relating these fluxes to effects on vegetation" (ISA, section IS.3.2). Further, as flux of O_3 into the plant under different conditions of O_3 in ambient air is affected by several factors including temperature, vapor pressure deficit, light, soil moisture, and plant growth stage, use of this approach to quantify the vegetation impact of O_3 would require information on these various types of factors (ISA, section IS.3.2). In addition to these data requirements, each species has different amounts of internal detoxification potential that may protect species to differing degrees. The lack of detailed species- and site-specific data required for flux modeling in the U.S. and the lack of understanding of detoxification processes continues to make this technique less viable for use in risk assessments in the U.S. (ISA, section IS.3.2).

Among the studies newly available since the last review, no new exposure indices for assessing effects on vegetation growth or other physiological process parameters have been identified. In the literature available since the 2013 ISA, the SUM06, AOTx (e.g., AOT60) and W126 exposure metrics remain the metrics that are most commonly discussed (ISA, Appendix 8, section 8.13.1). The ISA notes that "[c]umulative indices of exposure that differentially weight hourly concentrations [which would include the W126 index] have been found to be best suited to characterize vegetation exposure to ozone with regard to reductions in vegetation growth and yield" (ISA, section ES.3). Accordingly, in this review, as in the last two reviews, we use the seasonal W126-based cumulative, concentration-weighted metric for consideration of the effects evidence and quantitative exposure analyses, particularly related to growth effects (as summarized in sections 4.3.3.2 and 4.4 below).

[33] 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 O_3 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).

[34] 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.

The first step in calculating the seasonal W126 index for a specific year, as described and considered in this review, is to sum the weighted hourly O_3 concentrations in ambient air during daylight hours (defined as 8:00 a.m. to 8:00 p.m. local standard time) within each calendar month, resulting in monthly index values. The monthly W126 index values are calculated from hourly O_3 concentrations as follows.[35]

$$\boldsymbol{Monthly\ W126} = \sum_{d=1}^{N} \sum_{h=8}^{19} \frac{C_{dh}}{1+4403*\exp(-126*C_{dh})}$$

where,
N is the number of days in the month
d is the day of the month (d = 1, 2, …, N)
h is the hour of the day (h = 0, 1, …, 23)
C_{dh} is the hourly O_3 concentration observed on day *d*, hour *h*, in parts per million

The W126 index value for a specific year is the maximum sum of the monthly index values for three consecutive months within a calendar year (i.e., January to March, February to April, … October to December). Three-year average W126 index values are calculated by taking the average of seasonal W126 index values for three consecutive years (e.g., as described in Appendix 4D, section 4D.2.2).

4.3.3.1.2 Relationships Between Exposure Levels and Effects

Across the array of O_3-related welfare effects, consistent and systematically evaluated information on E-R relationships across multiple exposure levels is limited. Most prominent is the information on E-R relationships for growth effects on tree seedlings and crops,[36] which has been available for the past several reviews. The information on which these functions are based comes primarily from the U.S. EPA's National Crop Loss Assessment Network (NCLAN)[37] project for crops and the NHEERL-WED project for tree seedlings, projects implemented primarily to define E-R relationships for major agricultural crops and tree species, thus advancing understanding of responses to O_3 exposures (ISA, Appendix 8, section 8.13.2). These projects included a series of experiments that used OTCs to investigate tree seedling growth response and crop yield over a growing season under a variety of O_3 exposures and growing conditions (2013 ISA, section 9.6.2; Lee and Hogsett, 1996). These experiments have produced

[35] In situations where data are missing, an adjustment is factored into the monthly index (as described in Appendix 4D, section 4D.2.2).

[36] The E-R functions estimate O_3-related reduction in a year's tree seedling growth or crop yield as a percentage of that expected in the absence of O_3 (Appendix 4A; ISA, Appendix 8, section 8.13.2).

[37] The NCLAN program, which was undertaken in the early to mid-1980s, assessed multiple U.S. crops, locations, and O_3 exposure levels, using consistent methods, to provide the largest, most uniform database on the effects of O_3 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).

multiple studies that document O_3 effects on tree seedling growth and crop yield across multiple levels of exposure. Importantly, the information on exposure includes hourly concentrations across the season (or longer) exposure period which can then be summarized in terms of the various seasonal metrics.[38] In the initial analyses of these data, exposure was characterized in terms of several metrics, including seasonal SUM06 and W126 indices (Lee and Hogsett, 1996; 1997 Staff Paper, sections IV.D.2 and IV.D.3; 2007 Staff Paper, section 7.6;), while use of these functions in the last review focused on their implementation in terms of seasonal W126 index (2013 ISA, section 9.6; 80 FR 65391-92, October 26, 2015). This information for seedlings of the 11 tree species, in combination with air quality analyses, was a key consideration in the 2015 decision on the level for the revised secondary standard (80 FR 65292, October 26, 2015).

The 11 species for which robust and well-established E-R functions for RBL are available are black cherry, Douglas fir, loblolly pine, ponderosa pine, quaking aspen, red alder, red maple, sugar maple, tulip poplar, Virginia pine, and white pine (Figure 4-3; Appendix 4A; 2013 ISA, section 9.6).[39] While these 11 species represent only a small fraction of the total number of native tree species in the contiguous U.S., this small subset includes eastern and western species, deciduous and coniferous species, and species that grow in a variety of ecosystems and represent a range of tolerance to O_3 (Appendix 4B; 2013 ISA, section 9.6.2). The established E-R functions for most of the 11 species were derived using data from multiple studies or experiments involving a wide range of exposure and/or growing conditions. From the available data, separate E-R functions were developed for each combination of species and experiment (2013 ISA, section 9.6.1; Lee and Hogsett, 1996). From these separate species-experiment-specific E-R functions, species-specific composite E-R functions were developed (Appendix 4A). Biomass growth loss predictions using these functions were evaluated in the ISA for the last review based on a recent study for aspen (2013 ISA, section 9.6.2; ISA, Appendix 8, section 8.13.2).

[38] 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.

[39] 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 O_3 concentrations in the New York City metropolitan area, such that O_3 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 last review (80 FR 65292, October 26, 2015.)

The 11 species-specific composite median functions are described in Appendix 4A (see section 4A.1.1). For some of these species, the E-R function is based on a single study (e.g., red maple), while for other species there were as many as 11 studies available (e.g., ponderosa pine). In total, the 11 species-specific E-R functions are based on 51 tree seedling studies or experiments. A stochastic analysis performed for the 2014 WREA provides a sense of the variability and uncertainty associated with the estimated E-R relationships among and within species (Appendix 4A, section 4A.1.1, Figure 4A-13). Based on the species-specific E-R functions, the studied tree species appear to vary widely in sensitivity to reduced growth at the seedling stage (Figure 4-3). Since the initial set of studies were completed, several additional studies, focused on aspen, have been published based on the Aspen FACE experiment in a planted forest in Wisconsin; the findings were consistent with many of the OTC studies (ISA, Appendix 8, section 8.13.2).

With regard to crops, established E-R functions are available for 10 crops: barley, field corn, cotton, kidney bean, lettuce, peanut, potato, grain sorghum, soybean and winter wheat (Figure 4-4; Appendix 4A; ISA, Appendix 8, section 8.13.2). Studies available in the last review increased our confidence in the predictability of the crop E-R functions. Since then, new evidence is available for seven soybean cultivars that confirms the reliability of the soybean E-R functions developed from NCLAN data and indicates that they extend in applicability to recent cultivars (ISA, Appendix 8, section 8.13.2). In the last review, these E-R functions were used to characterize the estimated growth reduction across the studied species for a range of seasonal W126 index exposures (Appendix 4A, section 4A.1).

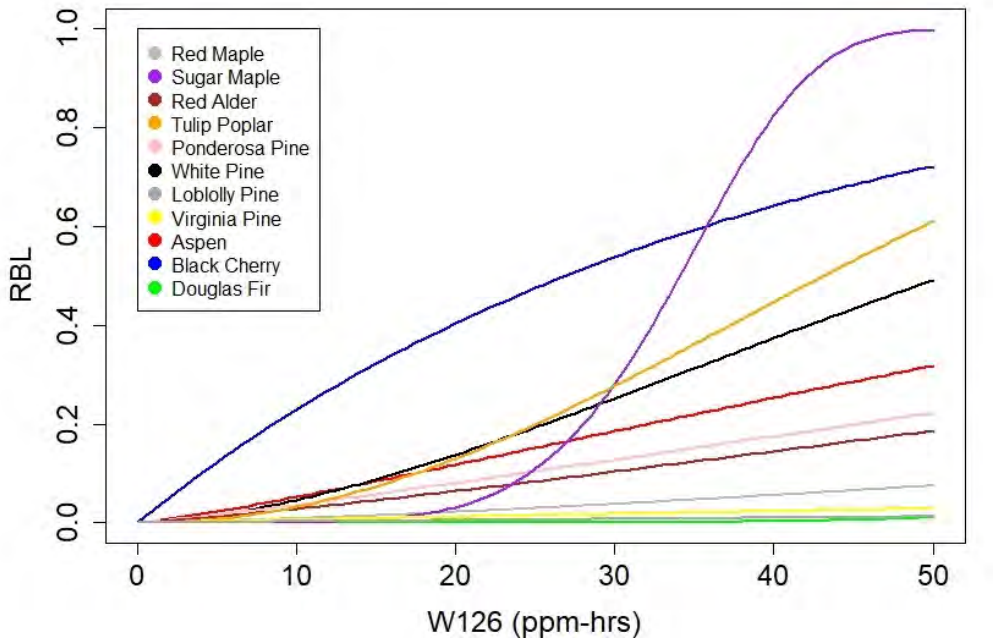

Figure 4-3. Established RBL functions for seedlings of 11 tree species.

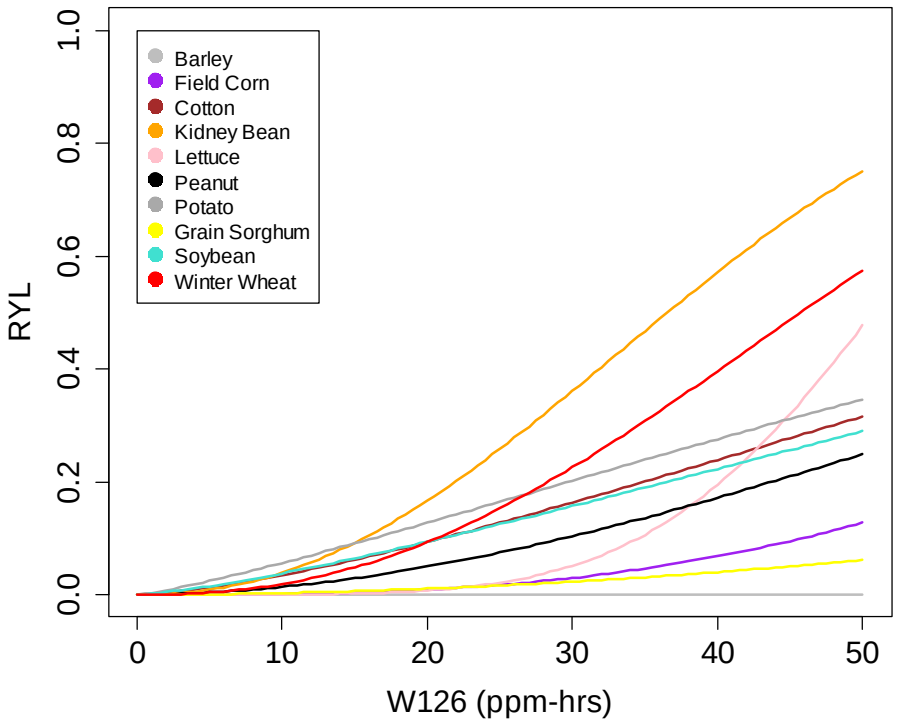

Figure 4-4. Established RYL functions for 10 crops.

Newly available studies that investigated growth effects of O_3 exposures are also consistent with the existing evidence base, and generally involved particular aspects of the effect rather than expanding the conditions under which plant species, particularly trees, have been assessed (ISA, section IS.5.1.2). The ISA notes the recent availability of a compilation of previously available studies on plant biomass response to O_3 (in terms of AOT40); the compilation reports linear regressions conducted on the associated varying datasets (ISA, Appendix 8, section 8.13.2). Little is presented in this compilation with regard to evaluation of consistent and similar O_3 exposure and biomass response measurements, and the exposure durations, which are not reported for each study, are reported to vary, with the shortest being 21 days (van Goethem et al., 2013).[40] These aspects of the publication limit its usefulness with regard to describing E-R relationships that might provide for estimation of specific impacts associated with air quality conditions meeting the current standard. As was noted in the 2013 ISA, "[i]n order to support quantitative modeling of exposure-response relationships, data should preferably include more than three levels of exposure, and some control of potential confounding

[40] The set of studies included in this compilation were described as meeting a set of criteria, such as: including O_3 only exposures in conditions described as "close to field" exposures (which were expressed as AOT40); including at least 21 days exposure above 40 ppb O_3; and, having a maximum hourly concentration that was no higher than 100 ppb (van Goethem et al., 2013).

or interacting factors should be present in order to model the relationship with sufficient accuracy" (2013 ISA, p. 9-118). The 2013 ISA further discussed the differences across available studies, recognizing that the majority of studies contrast only two (or sometimes three with the addition of a carbon filtration) O_3 exposure levels. While such studies can be important for verifying more extensive studies, they "do not provide exposure-response information that is highly relevant to reviewing air quality standards" (2013 ISA, p. 9-118).

4.3.3.2 Visible Foliar Injury

With regard to visible foliar injury, as with the evidence available in the last review, the evidence newly available in this review "continues to show a consistent association between visible injury and ozone exposure," while also recognizing the role of modifying factors such as soil moisture and time of day (ISA, section IS.5.1.1). The ISA, in concluding that the newly available information is consistent with conclusions of the 2013 ISA, also summarizes several recently available studies that continue to document that O_3 elicits visible foliar injury in many plant species, including a synthesis of previously published studies that categorizes studied species (and their associated taxonomic classifications) as to whether or not O_3-related foliar injury has been reported. Although this recent publication identifies many species in which visible foliar injury has been documented to occur in the presence of elevated O_3,[41] it does not provide quantitative information regarding specific exposure conditions or analyses of E-R relationships (ISA, Appendix 8, section 8.3). Additionally, one recent study is identified as reporting visible foliar injury in a non-native, yet established, and invasive tree species in a location with O_3 concentrations corresponding to a seasonal W126 index of 11.6 ppm-hrs (ISA, Appendix 8, sections 8.2 and 8.2.1). The annual fourth highest 8-hour daily maximum concentration for the study year and location of this study (monitoring site 42-027-9991) is 76 ppb. The design value for the 3-year design period encompassing the year and location of this study exceeds 70 ppb (monitoring site 42-027-9991 for 2011-2013 design period), indicating that the air quality associated with the exposure would not have met the current secondary standard.[42]

The evidence in the current review, as was the case in the last review, while documenting that elevated O_3 conditions in ambient air generally results in visible foliar injury in sensitive

[41] The publication identifies 245 species across 28 plant genera, many native to the U.S., in which O_3-related visible foliar injury has been reported (ISA, Appendix 8, section 8.3).

[42] 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.

species (when in a predisposing environment)[43], does not include a quantitative description of the relationship of incidence or severity of visible foliar injury in sensitive species in natural locations in the U.S. with specific metrics of O_3 exposure. Several studies of the extensive USFS field-based dataset of visible foliar injury incidence in forests across the U.S.[44] illustrate the limitations of current understanding of this relationship. For example, a study that was available in the last review presents a trend analysis of these data for sites located in 24 states of the northeast and north central U.S. for the 16-year period from 1994 through 2009 that provides some insight into the influence of changes in air quality and soil moisture on visible foliar injury and the difficulty inherent in predicting foliar injury response under different air quality and soil moisture scenarios (Smith, 2012, Smith et al., 2012; U.S. EPA, 2018; ISA, Appendix 8, section 8.2). This study, like prior analyses of such data, shows the dependence of foliar injury incidence and severity on local site conditions for soil moisture availability and O_3 exposure. For example, while the authors characterize the ambient air O_3 concentrations to be the "driving force" behind incidence of injury and its severity, they state that "site moisture conditions are also a very strong influence on the biomonitoring data" (Smith et al., 2003). In general, the USFS data analyses have found foliar injury prevalence and severity to be higher during seasons and sites that have experienced the highest O_3 than during other periods (e.g., Campbell et al., 2007; Smith, 2012).

Studies of the incidence of visible foliar injury in national forests, wildlife refuges, and similar areas have often used cumulative indices such as SUM06 to investigate variations in incidence of foliar injury (e.g., Hildebrand et al., 1996). For example, a study of six years of USFS biosite data for three western states found that the biosites with the highest O_3 exposure (SUM06 at or above 25 ppm-hrs) had the highest percentage of biosites with injury and the highest mean biosite index, with little discernable difference among the lower exposure categories; this study also identified "better linkage between air levels and visible injury" as an O_3 research need (Campbell et al., 2007). More recent studies of the complete 16 years of data in 24 northeast and north central states have suggested that a cumulative exposure index alone may not completely describe the O_3-related risk of this effect (Smith et al., 2012; Smith, 2012). For example, Smith (2012) observed there to be a declining trend in the 16-year dataset, "especially

[43] As noted in the 2013 ISA and the ISA for the current review, 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).

[44] 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).

after 2002 when peak ozone concentrations declined across the entire region" thus suggesting a role for peak concentrations.

Some studies of visible foliar injury incidence data have investigated the role of peak concentrations quantified by an O_3 exposure index that is a count of hourly concentrations (e.g., in a growing season) above a threshold 1-hour concentration of 100 ppb, N100 (e.g., Smith, 2012; Smith et al., 2012). For example, the study by Smith (2012) discussed injury patterns at biosites in 24 states in the Northeast and North Central regions in the context of the SUM06 index and N100 metrics (although not in statistical combination).[45] That study of 16 years of biomonitoring data from these sites suggested that there may be a threshold exposure needed for injury to occur, and the number of hours of elevated O_3 concentrations during the growing season (such as what is captured by a metric like N100) may be more important than cumulative exposure in determining the occurrence of foliar injury (Smith, 2012).[46] The study's authors noted this finding to be consistent with findings reported by a study of statistical analyses of seven years of visible foliar injury data from a wildlife refuge in the mid-Atlantic (Davis and Orendovici, 2006, Smith et al., 2012). The latter study investigated the fit of multiple models that included various metrics of cumulative O_3 (e.g., SUM06, SUM0, SUM08), alone and in combination with some other variables (Davis and Orendovici, 2006). Among the statistical models investigated, the model with the best fit to the visible foliar injury incidence data was found to be one that included N100 and W126 indices, as well as drought index (Davis and Orendovici, 2006).[47]

The 2013 ISA and 2006 AQCD noted the established significant role of higher or peak O_3 concentrations, as well as pattern of their occurrence, in plant responses. In identifying support with regard to foliar injury as the response, these assessments both cite studies that support the "important role that peak concentrations, as well as the pattern of occurrence, plays in plant response to O_3" (2013 ISA, p. 9-105; 2006 AQCD, p. AX9-169). For example, a study of European white birch saplings reported that peak concentrations and the duration of the exposure event were important determinants of foliar injury (2013 ISA, section 9.5.3.1; Oksanen and Holopainen, 2001). This study also evaluated tree growth, which was found to be more

[45] The current ISA, 2013 ISA and prior AQCDs have not described extensive evaluation of specific peak-concentration metrics such as the N100 that might assist in identifying the one best suited for such purposes.

[46] 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).

[47] The models evaluated included several with cumulative exposure indices alone. These included SUM60, SUM0, and SUM80. They did not include a model with W126 that did not also include N100. Across all of these models, 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).

related to cumulative exposure (2013 ISA, p. 9-105).[48] A second study that was cited by both assessments that focused on aspen, reported that "the variable peak exposures were important in causing injury, and that the different exposure treatments, although having the same SUM06, resulted in very different patterns of foliar injury (2013 ISA, p. 9-105; 2006 AQCD, p. AX9-169; Yun and Laurence, 1999). As noted in the 2006 AQCD, the cumulative exposure indices (e.g., SUM06, W126) were "originally developed and tested using only growth/yield data, not foliar injury" and "[t]his distinction is critical in comparing the efficacy of one index to another" (2006 AQCD, p. AX9-173). It is also recognized that where cumulative indices are highly correlated with the frequency or occurrence of higher hourly average concentrations, they could be good predictors of such effects (2006 AQCD, section AX9.4.4.3).

In a more recent study that is cited in the current ISA a statistical modeling analysis was performed on a subset of the years of data that were described in Smith (2012). This analysis, which involved 5,940 data records from 1997 through 2007 from the 24 northeast and north central states, tested a number of models for their ability to predict the presence of visible foliar injury (a nonzero biosite score), regardless of severity, and generally found that the type of O_3 exposure metric (e.g., SUM06 *versus* N100) made only a small difference, although the models that included both a cumulative index (SUM06) and N100 had a just slightly better fit (Wang et al., 2012). Based on their investigation of 15 different models, using differing combination of several types of potential predictors, the study authors concluded that they were not able to identify environmental conditions under which they "could reliably expect plants to be damaged" (Wang et al., 2012). This is indicative of the current state of knowledge, in which there remains a lack of established quantitative functions describing E-R relationships that would allow prediction of visible foliar injury severity and incidence under varying air quality and environmental conditions.

The available information related to O_3 exposures associated with visible foliar injury of varying severity also includes the dataset developed by the EPA in the last review from USFS biosite index (BI) scores, collected during the years 2006 through 2010 at locations in 37 states, that were combined with estimates of soil moisture[49] and estimates of seasonal cumulative O_3

[48] 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).

[49] 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).

exposure in terms of W126 index[50] (Smith and Murphy, 2015; Appendix 4C). This dataset includes more than 5,000 records of which more than 80 percent have a BI score of zero (indicating a lack of visible foliar injury).[51] While the estimated W126 index assigned to records in this dataset (described in Appendix 4C) ranges from zero to somewhat above 50 ppm-hrs, only 8% of the records have W126 index values above 15 ppm-hrs. Beyond an analysis included from the last review, the presentations in Appendix 4C are primarily descriptive (as compared to statistical analysis); this is in recognition of the limitations and uncertainties of the dataset as summarized in Appendix 4C, section 4C.5. The presentation in Appendix 4C describes the BI scores for the records in the dataset in relation to the W126 index estimate for each record, using bins of increasing W126 index values. The presentation indicates the occurrence of injury (and in severity) across the W126 index bins to be variable, and also finds the greatest incidence of records with BI scores above zero, five, or higher to occur for records with the highest W126 index values (i.e., the bin for W126 index estimates greater than 25 ppm-hrs), as seen in Figure 4-5 for records in the normal soil moisture category[52] (see also Appendix 4C, Table 4C-6).

The average BI score per W126 index bin is also variable, although for records categorized as normal soil moisture, the average BI score in the highest W126 bin is noticeably greater than for lower W126 bin scores (Figure 4-5). For example, the average BI score for the normal soil moisture category is 7.9 among records with W126 index estimates greater than 25 ppm-hrs, compared to 1.6 among records for W126 index estimates between 19 and 25 ppm-hrs. For records categorized as wet soil moisture, the sample size for the W126 bins above 13 ppm-hrs is quite small (including only 18 of the 1,189 records in that soil moisture category), precluding meaningful interpretation.[53]

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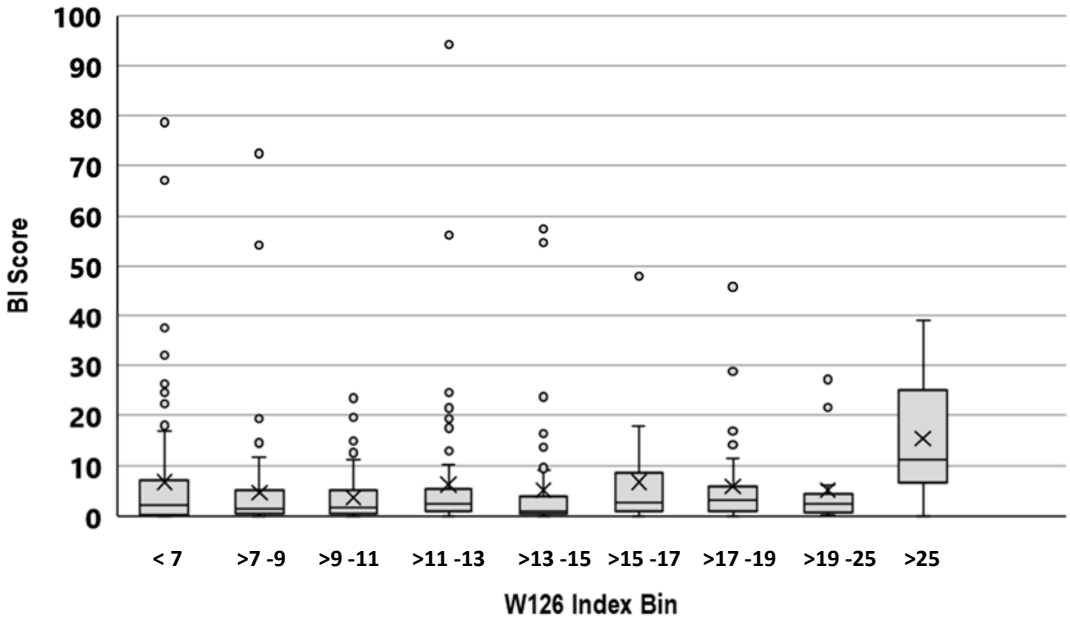
[50] The W126 index values assigned to the biosite locations are estimates developed for 12 kilometer (km) by 12 km cells in a national-scale spatial grid for each year. The grid cell estimates were derived from applying a spatial interpolation technique to annual W126 values derived from O_3 measurements at ambient air monitoring locations for the years corresponding to the biosite surveys (details in Appendix 4C, sections 4.C.2 and 4C.5).

[51] 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)..

[52] 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).

[53] The full database includes only 18 records at sites in the wet soil moisture category and a W126 index value above 13 ppm-hrs, with 9 or fewer (less than 1%) in each of the W126 bins above 13 ppm-hrs (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).

While for BI scores above zero, the data may indicate a suggestion of increased incidence among records in the W126 bins just below the highest (e.g., for the dry or normal soil moisture categories), for BI scores above 5, there is little or no difference across the W126 bins except for the highest bin, which is for W126 above 25 ppm-hrs (Appendix 4C, Table 4C-6). For example, among records in the normal soil category, the proportion of records with BI above five fluctuates between 5% and 13% across all but the highest W126 bin (>25 ppm-hrs) for which the proportion is 41% (Appendix 4C, Table 4C-6). The same pattern is observed for BI scores above 15 at sites with normal and dry soil moisture conditions, albeit with lower incidences. For example, the incidence of normal soil moisture records with BI score above 15 in the bin for W126 index values above 25 ppm-hrs was 20% but fluctuates between 1% and 4% in the bines with W126 index values at or below 25 ppm-hrs (Appendix 4C, Table 4C-6).



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 light, moderate or greater injury, to be higher at the highest W126 index values, with appreciable variability in the data for the lower bins. 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 O_3 air quality not captured by the W126 index. This appears

to be consistent with the conclusions of the studies of detailed quantitative analyses, summarized above, that the pattern is stronger at higher O_3 concentrations while uncertainty remains regarding the tools for and the appropriate metric for quantifying influence of O_3 exposures, as well as perhaps soil moisture conditions (Davis and Orendovici, 2006, Smith et al., 2012; Wang et al., 2012). Thus, the limitations recognized in the last review 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).

Dose modeling or flux models, discussed in section 4.3.3.1.1 above, have also been considered for quantifying O_3 dose that may be related to plant injury. Among the newly available evidence is a study examining relationships between short-term flux and leaf injury on cotton plants that described a sensitivity parameter that might characterize the influence on the flux-injury relationship of diel and seasonal variability in plant defenses (among other factors) and suggested additional research might provide for such a sensitivity parameter to "function well in combination with a sigmoidal weighting of flux, analogous to the W126 weighting of concentration", and perhaps an additional parameter (Grantz et al., 2013, p. 1710; ISA, Appendix 8, section 8.13.1). However, the ISA recognizes there is "much unknown" with regard to the relationship between O_3 uptake and leaf injury, and relationships with detoxification processes (ISA, Appendix 8, section 8.13.1 and p. 8-184). These uncertainties have made this technique less viable for assessments in the U.S., precluding use of a flux-based approach at this time (ISA, Appendix 8, section 8.13.1 and p. 8-184).

4.3.3.3 Other Effects

With regard to radiative forcing and subsequent climate effects associated with the global tropospheric abundance of O_3, the newly available evidence in this review does not provide more detailed quantitative information regarding O_3 concentrations at the national scale. Although tropospheric O_3, at the global scale, continues to be recognized as having a causal relationship with radiative forcing and a likely causal relationship with subsequent effects on temperature, precipitation and related climate variables, the non-uniform distribution of O_3 (spatially and temporally) makes the development of quantitative relationships between the magnitude of such effects and differing O_3 concentrations in the U.S. challenging (ISA, Appendix 9). Additionally, "the heterogeneous distribution of ozone in the troposphere complicates the direct attribution of spatial patterns of temperature change to ozone induced [radiative forcing]" and there are "ozone climate feedbacks that further alter the relationship between ozone [radiative forcing] and temperature (and other climate variables) in complex ways" (ISA, Appendix 9, section 9.3.1, p. 9-19). Thus, the ISA recognizes that "[c]urrent limitations in climate modeling tools, variation

across models, and the need for more comprehensive observational data on these effects represent sources of uncertainty in quantifying the precise magnitude of climate responses to ozone changes, particularly at regional scales" (ISA, section IS.6.2.2, p. 9-22). While these complexities impede our ability to consider specific O_3 concentrations in the U.S. with regard to specific magnitudes of impact on radiative forcing and subsequent climate effects, we note that our ability to estimate growth-related impacts of trees can also inform our consideration of the sequestration of carbon in terrestrial ecosystems, a process that can reduce tropospheric abundance of CO_2, the pollutant ranked first in importance as a greenhouse gas and radiative forcing agent.

With regard to the two newly identified categories of effects, there are multiple limitations and uncertainties regarding characterization of exposure conditions that might elicit effects and the comprehensive characterization of the effects. For example, with regard to alteration of herbivore growth and reproduction, although "there are multiple studies demonstrating ozone effects on fecundity and growth in insects that feed on ozone-exposed vegetation", "no consistent directionality of response is observed across studies and uncertainties remain in regard to different plant consumption methods across species and the exposure conditions associated with particular severities of effects " (ISA, pp. ES-18, IS-64, IS-91 and Appendix 8, section 8.6.3). Such limitations and uncertainties in the evidence base for this category of effects preclude broader characterization, as well as quantitative analysis related to air quality conditions meeting the O_3 standard. As characterized in the ISA, uncertainties remain in the evidence; these relate to the different plant consumption methods across species and the exposure conditions associated with particular responses, as well as variation in study designs and endpoints used to assess O_3 response (ISA, IS.6.2.1 and Appendix 8, section 8.6). Thus, while the evidence describes changes in nutrient content and leaf chemistry following O_3 exposure (ISA, p. IS-73), the effect of these changes on herbivores consuming the leaves is not well characterized or clear.

The evidence for a second newly identified category of effects, alteration of plant-insect signaling, draws on new research that has provided clear evidence of O_3 modification of VPSCs and behavioral responses of insects to these modified chemical signals. Most of these studies, however, have been carried out in laboratory conditions rather than in natural environments, and involve a relatively small number of plant species and plant-insect associations. While the evidence documents effects on plant production of signaling chemicals and on the atmospheric persistence of signaling chemicals, as well as on the behaviors of signal-responsive insects, it is limited with regard to characterization of mechanisms and the consequences of any modification of VPSCs by O_3 (ISA, section IS.6.2.1) Further, 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), and many of the studies involve quite short controlled exposures (hours to days) to elevated concentrations, posing limitations for our purposes of considering the potential for impacts associated with the studied effects to be elicited by air quality conditions that meet the current standard (ISA, section IS.6.2.1 and Appendix 8, section 8.7).

With regard to previously recognized categories of vegetation-related effects, other than growth and visible foliar injury, such as reduced plant reproduction, reduced productivity in terrestrial ecosystems, alteration of terrestrial community composition and alteration of below-ground biogeochemical cycles, the newly available evidence includes a variety of studies, as identified in the ISA (ISA, Appendix 8, sections 8.4, 8.8 and 8.10). Across the studies, a variety of metrics (including AOT40, 4- to 12-hour mean concentrations, and others) are used to quantify exposure over varying durations and various countries. The ISA additionally describes publications that summarize previously published studies in several ways. For example, a meta-analysis of reproduction studies categorized the reported O_3 exposures into bins of differing magnitude, grouping differing concentration metrics and exposure durations together, and performed statistical analyses to reach conclusions regarding the presence of an O_3-related effect (ISA, Appendix 8, section 8.4.1). While such studies continue to support conclusions of the ecological hazards of O_3, they do not improve capabilities for characterizing the likelihood of such effects under varying patterns of environmental O_3 conditions that occur under the current standard.

As at the time of the last review, growth impacts, most specifically as evaluated by RBL for tree seedlings and RYL for crops, remain the type of vegetation-related effects for which we have the best understanding of exposure conditions likely to elicit them. Thus, as was the case in the decision for the last review, the quantitative analyses of exposures occurring under air quality that meets the current standard (summarized in section 4.4 below) is focused primarily on the W126 index, given its established relationship with growth effects.

4.3.4 Key Uncertainties

The type of uncertainties for each category of effects tends to vary in relation to the maturity of the associated evidence base from those associated with overarching characterizations of the effects to those associated with quantification of the cause and effect relationships. For example, given the longstanding nature of the evidence for many of the vegetation effects identified in the ISA as causally or likely causally related to O_3 in ambient air, the key uncertainties and limitations in our understanding of these effects relate largely to the implications or specific aspects of the evidence, as well as to current understanding of the quantitative relationships between O_3 concentrations in the environment and the occurrence and

severity (or relative magnitude) of such effects or understanding of key influences on these relationships. For more newly identified categories of effects, the evidence may be less extensive, thus precluding consideration of such details.

- **What are important uncertainties in the evidence? To what extent have important uncertainties in the evidence identified in the last review been reduced and/or have new uncertainties been recognized?**

Among the categories of effects identified in past reviews, key uncertainties remain in the current evidence. The category of O_3 welfare effects for which current understanding of quantitative relationships is strongest is reduced plant growth. As a result, this category was the focus of the Administrator's decision-making in the last review, with RBL in tree seedlings playing the role of surrogate (or proxy) for the broader array of vegetation-related effects that range from the individual plant level to ecosystem services. Limitations in the evidence base and associated uncertainties recognized in the last review remain and include a number of uncertainties that affect characterization of the magnitude of cumulative exposure conditions eliciting growth reductions in U.S. forests.

As recognized in the last review there are uncertainties in the extent to which the 11 tree species for which there are established E-R functions encompass the range of O_3 sensitive species in the U.S., and also the extent to which they represent U.S. vegetation as a whole. These 11 species include both deciduous and coniferous trees with a wide range of sensitivities and species native to every NOAA climate region across the U.S. and in most cases are resident across multiple states and regions. In considering this issue in the last review, the CASAC stated that there is "considerable uncertainty in extrapolating from the [studied] forest tree species to all forest tree species in the U.S.," and additionally expressed 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). The CASAC also expressed the view, in the last review, that it "should not be assumed that species of unknown sensitivity are tolerant to ozone" and "[i]t is 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).

We additionally recognize important uncertainties in the extent to which the E-R functions for reduced growth in tree seedlings are also descriptive of such relationships during later lifestages, for which there is a paucity of established E-R relationships. Although such information is limited with regard to mature trees, analyses in the 2013 ISA indicated that reported growth response of young aspen over six years was similar to the reported growth response of seedlings (ISA, Appendix 8, section 8.13.2; 2013 ISA, section 9.6.3.2). Additionally, there are uncertainties with regard to the extent to which various factors in natural environments

can either mitigate or exacerbate predicted O_3-plant interactions and contribute variability in vegetation-related effects, including reduced growth. Such factors include multiple genetically influenced determinants of O_3 sensitivity, changing sensitivity to O_3 across vegetative growth stages, co-occurring stressors and/or modifying environmental factors.

Another area of uncertainty affects interpretation of the potential for harm to public welfare over multi-year periods of air quality that meet the current standard. For example, there is variability in ambient air O_3 concentrations from year to year, as well as year-to-year variability in environmental factors, including rainfall and other meteorological factors that affect plant growth and reproduction, such as through changes in soil moisture. Accordingly, these variabilities contribute uncertainties to estimates of the occurrence and magnitude of O_3-related effects in any year, and to such estimates over multi-year periods. Accordingly, limitations in our ability to estimate growth effects over tree lifetimes of year-to-year variation in O_3 concentrations, particularly those associated with conditions meeting the current standard, contribute uncertainty to estimates of cumulative growth (biomass) effects over multi-year periods in the life of individual trees and associated populations, as well as related effects in associated communities and ecosystems.

These uncertainties in estimates stem from limitations and imprecision in our tools, and variation in aspects of the underlying data. For example, the studies on which the established E-R functions for the 11 tree species are based vary in exposure duration (e.g., from periods of 82 to 140 days over a single year to periods of 180 to 555 days across two years) and in whether measurements were made immediately following exposure period or in subsequent spring. The E-R functions were derived based on the exposure duration of the experiment and, adjusted or normalized to 3-month periods based on assumptions regarding relationships between duration, cumulative exposure in terms of W126 index and plant growth response (see Lee and Hogsett, 1996, section I.3). For example, while the functions are defined as describing a seasonal response, some were derived by distributing responses observed at the end of two seasons of varying exposures equally across the two seasons (essentially applying the average to both seasons). The evidence for seasonal growth effects on trees is also somewhat limited with regard to multi-year studies (particularly longer than two years) that have reported detailed O_3 concentration data throughout the exposure. This contributes uncertainty, and accordingly a lack of precision, to an understanding of the quantitative impacts of seasonal O_3 exposure, including its year-to-year variability, on tree growth and annual biomass accumulation. This uncertainty limits our understanding of the extent to which tree biomass would be expected to appreciably differ at the end of multi-year exposures for which the overall average exposure is the same, yet for which the individual year exposures varies in different ways (e.g., as analyzed in Appendix 4D). For example, the extent of any differences in tree biomass for two multi-year scenarios with

the same 3-year average W126 index but differing single-year indices is not clear, including for exposures associated with O_3 concentrations that would meet the current standard.[54]

A study of multi-year growth effects is available for aspen (King et al., 2005). This study was assessed in the 2013 ISA and summarized in the ISA for the current review with regard the extent to which it confirmed O_3-related biomass impacts estimated using the established E-R functions for aspen (2013 ISA, section 9.6.3.2; current ISA, Appendix 8, section 8.13.2). The 2013 assessment applied the E-R functions to O_3 exposure (quantified as cumulative average seasonal W126 index) at each of six consecutive years and compared the estimated aboveground biomass to estimates based on data reported for each year by the study (2013 ISA, section 9.6.3.2). The conclusions reached were that the experimental observations are "very close" to estimates based on the established E-R function for aspen, and that "the function based on one year of growth was shown to be applicable to subsequent years" (2013 ISA, p. 9-135; ISA, Appendix 8, p. 8-186).

Further, while the tree seedling E-R relationships for 11 species are long-established, we recognize the large variation among the species regarding how much experimental evidence is available. For example, the E-R function for aspen was derived from 13 experimental studies, while the E-R functions for the red maple and Virginia pine were each derived from a single study (Appendix 4A, section 4A.2, Table 4A-6; 1996 AQCD, Table 5-28; Lee and Hogsett, 1996). Additionally, while the evidence is longstanding and robust for growth effects of O_3, there is variation across the 11 species for which we have established E-R functions with regard to the extent to which the studies include O_3 treatment levels reflecting cumulative O_3 exposures, in terms of W126 index, lower than 20 ppm-hrs. Studies for five of the eleven species include cumulative exposures likely to correspond to W126 index values below 20 ppm-hrs (Appendix 4A, Table 4A-5).[55] Further, among studies for these five species, the findings for at least one study reported statistical significance only for biomass effects observed for higher O_3 exposures (e.g., Appendix 4A, Table 4A-6, black cherry). All of the factors identified here contribute to imprecision or inexactitude in estimates based on the E-R functions.

[54] 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)..

[55] For five of the species in Table 4A-5 in Appendix 4A, SUM06 index values below 25 ppm-hrs range from 12 to 21.7. In considering these values, we note that an approach used in the 2007 Staff Paper on specific temporal patterns of O_3 concentrations concluded that a SUM06 index value of 25 ppm-hrs would be estimated to correspond to a W126 index value of approximately 21 ppm-hrs (U.S. EPA, 2007, Appendix 7B, p. 7B-2). This would imply that a SUM06 value of 21 ppm-hrs would be expected to correspond to a W126 index value below 20 ppm-hrs.

Our consideration of the magnitude of tree growth effects that might cause or contribute to adverse effects for trees, forests, forested ecosystems or the public welfare is also complicated by various uncertainties or limitations in the evidence base, including those associated with relating magnitude of tree seedling growth reduction to larger-scale forest ecosystem impacts. Further, other factors can influence the degree to which O_3-induced growth effects in a sensitive species affect forest and forest community composition and other ecosystem service flows (e.g., productivity, belowground biogeochemical cycles and terrestrial ecosystem water cycling) from forested ecosystems. These include (1) the type of stand or community in which the sensitive species is found (i.e., single species versus mixed canopy); (2) the role or position the species has in the stand (i.e., dominant, sub-dominant, canopy, understory); (3) the O_3 sensitivity of the other co-occurring species (O_3 sensitive or tolerant); and (4) environmental factors, such as soil moisture and others. The lack of such established relationships with O_3 complicates consideration of the extent to which different estimates of impacts on tree seedling growth would indicate significance to the public welfare. Further, efforts to estimate O_3 effects on carbon sequestration are handicapped by the large uncertainties involved in attempting to quantify the additional carbon uptake by plants as a result of avoided O_3-related growth reductions. Such analyses require complex modeling of biological and ecological processes with their associated sources of uncertainty.

With regard to crop yield effects, as at the time of the last review, we recognize the potential for greater uncertainty in estimating the impacts of O_3 exposure on agricultural crop production than that associated with O_3 impacts on vegetation in natural forests. This relates to uncertainty in the extent to which agricultural management methods influence potential for O_3-related effects and accordingly, the applicability of the established E-R functions for RYL in current agricultural areas.

With regard to visible foliar injury, for which longstanding evidence documents a causal role for O_3, important uncertainties and limitations fall into two categories. The first category relates to our understanding of the key aspects of O_3 concentrations - and other key variables (e.g., soil moisture) - that have a direct bearing on the severity and incidence of vegetation injury, while the second concerns the impacts on aesthetic and recreational values of various severities and incidences of injury. With regard to the former, there is a lack of detailed understanding of specific patterns of O_3 concentrations over a growing season and the key aspects of those patterns (e.g., incidence of concentrations of particular magnitude) that contribute to an increased incidence and severity of injury occurrence in the U.S. For example, “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. 8-24). Accordingly, there are no established, quantitative E-R functions that document visible foliar injury severity and incidence

under varying air quality and environmental conditions (e.g., soil moisture). As discussed in section 4.3.3.2 above, the available studies that have investigated the role of different variables, including different metrics for characterizing O_3 concentrations over a growing season, do not provide a basis for characterizing the potential for different patterns of O_3 concentrations to contribute to different incidences and severity of foliar injury in U.S. forests. The second category of uncertainties and limitations concerns the information that would support associated judgments on the public welfare significance of different patterns of and severity of foliar injury, such as the extent to which such effects in areas valued by the public for different uses may be considered adverse to public welfare. In considering this issue, we note that some level of severity to a tree stand would be obvious to the casual observer (e.g., when viewing a stand covering a hillside from a distance), and some level of severity (e.g., leaf and crown damage that appreciably affects overall plant physiology) would also be expected to affect plant growth and reproduction. The extent to which recreational values are affected by lesser levels of injury severity and incidence is not clear from the available information. Thus, limitations and uncertainties in the available information, such as those described above, complicate our ability to comprehensively estimate the potential for visible foliar injury, its severity or extent of occurrence for specific air quality conditions, and associated public welfare implications, thus affecting a precise identification of air quality conditions that might be expected to provide a specific level of protection for this effect.

During the last review, the 2013 ISA did not assess the evidence of O_3 exposure and tree mortality with regard to its support for inference of a causal relationship. Evidence available in the last several reviews included field studies of pollution gradients that concluded O_3 damage to be an important contributor to tree mortality although several confounding factors such as drought, insect outbreak and forest management were identified as potential contributors (2013 ISA, section 9.4.7.1). Since the last review, three additional studies have been identified, as summarized in section 4.3.1 above, contributing to the ISA conclusion of sufficient evidence to infer a likely causal relationship for O_3 with tree mortality (ISA, Appendix 8, section 8.4). As noted in the ISA, there is only limited evidence from experimental studies that isolate the effect of O_3 on tree mortality, with the recently available Aspen FACE study of aspen survival involving cumulative seasonal exposures above 30 ppm-hrs during the first half of the 11-year study period (ISA, Appendix 8, Tables 8-8 and 8-9). Evidence is lacking regarding exposure conditions closer to those occurring under the current standard and any contribution to tree mortality.

In the case of the two newly identified categories of effects, the key uncertainties relate to comprehensive characterization of the effects. For example, with regard to alteration of herbivore growth and reproduction, although “there are multiple studies demonstrating ozone effects on

fecundity and growth in insects that feed on ozone-exposed vegetation", "no consistent directionality of response is observed across studies and uncertainties remain in regard to different plant consumption methods across species and the exposure conditions associated with particular severities of effects " (ISA, pp. ES-18, IS-64, IS91 and Appendix 8, section 8.6.3). Such limitations and uncertainties in the evidence base for this category of effects preclude broader characterization, as well as quantitative analysis related to air quality conditions meeting the O_3 standard. As characterized in the ISA, uncertainties remain in the evidence; these relate to the different plant consumption methods across species and the exposure conditions associated with particular responses, as well as variation in study designs and endpoints used to assess O_3 response (ISA, IS.6.2.1 and Appendix 8, section 8.6). Thus, while the evidence describes changes in nutrient content and leaf chemistry following O_3 exposure, the effect of these changes on herbivores consuming the leaves is not well characterized or clear (ISA, p. IS-73).

The evidence for a second newly identified category of effects, alteration of plant-insect signaling, draws on new research that has provided clear evidence of O_3 modification of VPSCs and behavioral responses of insects to these modified chemical signals. Most of these studies, however, have been carried out in laboratory conditions rather than in natural environments, and involve a relatively small number of plant species and plant-insect associations. While the evidence documents effects on plant production of signaling chemicals and on the atmospheric persistence of signaling chemicals, as well as on the behaviors of signal-responsive insects, it is limited with regard to characterization of mechanisms and the consequences of any modification of VPSCs by O_3 (ISA, section IS.6.2.1) Further, 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), and many of the studies involve quite short controlled exposures (hours to days) to elevated concentrations, posing limitations for our purposes of considering the potential for impacts associated with the studied effects to be elicited by air quality conditions that meet the current standard (ISA, section IS.6.2.1 and Appendix 8, section 8.7).

With regard to radiative forcing and climate effects, "uncertainty in the magnitude of radiative forcing estimated to be attributed to tropospheric ozone is a contributor to the relatively greater uncertainty associated with climate effects of tropospheric ozone compared to such effects of the well mixed greenhouse gases (e.g., carbon dioxide and methane)" (ISA, section IS.6.2.2). With regard to O_3 effects on temperature, "the heterogeneous distribution of ozone in the troposphere complicates the direct attribution of spatial patterns of temperature change to ozone induced RF" and the existence of O_3 climate feedbacks "further alter the relationship between ozone RF and temperature (and other climate variables) in complex ways" (ISA, Appendix 9, section 9.3.1). Thus, various uncertainties "render the precise magnitude of the

overall effect of tropospheric ozone on climate more uncertain than that of the well-mixed GHGs" (ISA, Appendix 9, section 9.3.3). Further, "[c]urrent limitations in climate modeling tools, variation across models, and the need for more comprehensive observational data on these effects represent sources of uncertainty in quantifying the precise magnitude of climate responses to ozone changes, particularly at regional scales" (ISA, Appendix 9, section 9.3.3).

4.4 EXPOSURE AND AIR QUALITY INFORMATION

Several different exposure and risk analyses were conducted in the last review of the secondary O_3 standard, as summarized in the IRP for this review. Uncertainties associated with the results for some analyses limited their use in the Administrator's decision-making, while uncertainties regarding public welfare significance of the findings for other analyses also limited such use of those analyses. In general, decision-making in the last 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 O_3-related reduction in tree seedling growth (summarized in section 4.3.3 above). These analyses supported the consideration of the potential for O_3 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).

The air quality and exposure analyses considered in the last review were of two types: (1) W126-based cumulative exposure estimates in Class I areas during 3-year periods that met the then-current standard (80 FR 65485-86, Table 3, October 26, 2015); and, (2) analyses for all U.S. monitoring locations and time periods that met the then-current and several potential alternative standards (Wells, 2015; 80 FR 65292, October 26, 2015). In these analyses, W126 index values[56] 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). That magnitude of W126 index is 19 ppm-hrs (80 FR 65391-65392). The second set of analyses also included an evaluation of relationships between W126 index values and design values[57] based on the form and averaging time of the then-current secondary standard (Wells, 2015). As summarized in the IRP, we identified these analyses to be updated in this review in recognition

[56] 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).

[57] 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 O_3 concentrations that just met the prior standard in a specific 3-yr period.

of the relatively reduced uncertainty associated with the use of these types of analyses (compared to the national or regional-scale modeling performed in the last review) to inform a characterization of cumulative O_3 exposure (in terms of the W126 index) associated with air quality just meeting the current standard (IRP, section 5.2.2). This lesser uncertainty of these air quality monitoring-based analyses contributed to their being more informative in the last review. The sections below present findings of the updated analyses that have been performed in the current review using the now available information.

Analyses in the current review are based on the expanded set of air monitoring data now available,[58] which includes 1,557 monitoring sites with sufficient data for derivation of design values (Appendix 4D, section 4D.2.2). The current analyses are described in detail in Appendix 4D. As in the last review, we have analyzed the data both for the most recent periods, and also across the full historical period back to 2000, which is now expanded from that available in the last review[59]. We have performed analyses for all sites in the U.S., as well as for the subset of sites in or near Class I areas. The most recent data analyzed are those for the design value period from 2016 to 2018. For all monitoring sites with valid design values for this period, Figure 4-6 presents the average seasonal W126 index for the recent 3-year period (2016-2018) and also denotes whether each site meets the current standard.

[58] In addition to being expanded with regard to data for more recent time periods than were available during the last review, the current dataset also includes a small amount of newly available older data for some monitoring sites that are now available in the AQS.

[59] In the last review, the dataset analyzed included data from 2000 through 2013 (Wells, 2015).

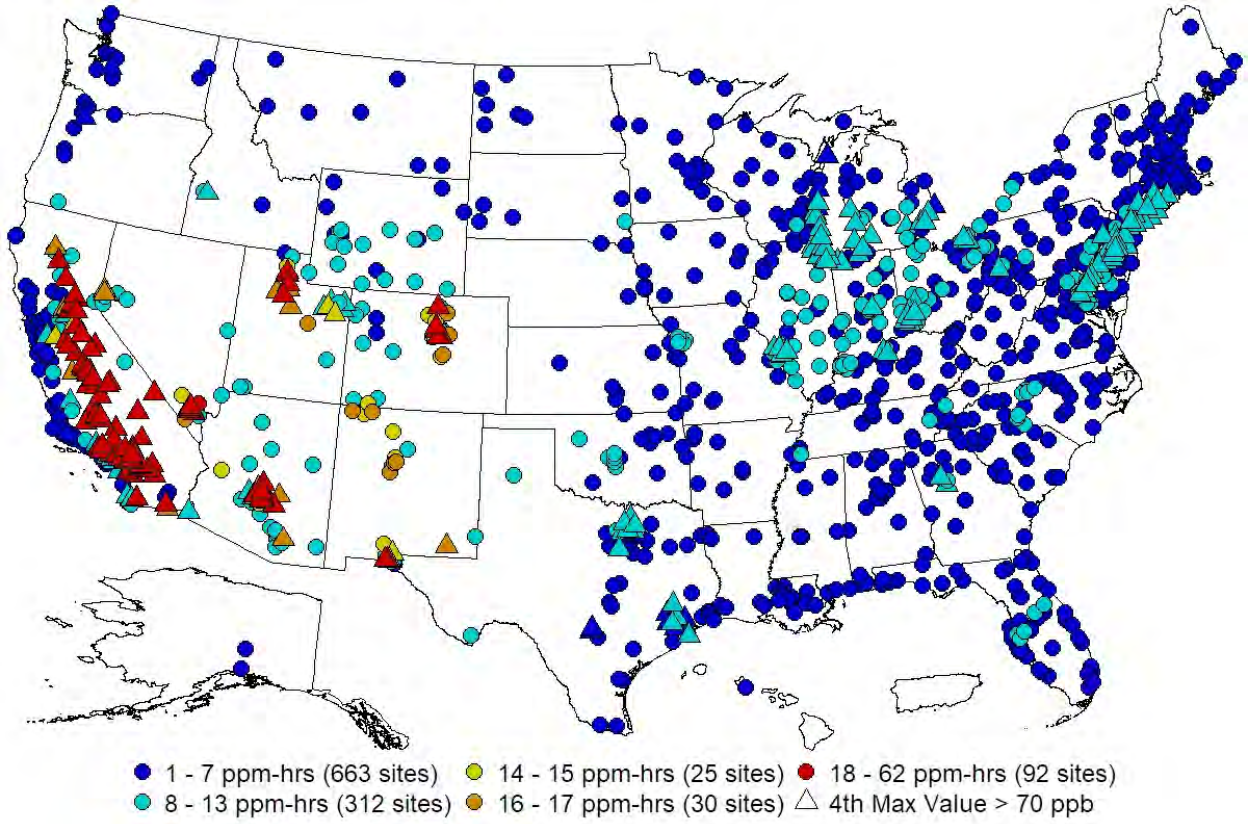


Figure 4-6. W126 index at monitoring sites with valid design values (2016-2018 average).

4.4.1 Influence of Form and Averaging Time of Current Standard on W126 Index

In revising the standard in 2015 to the now-current standard, the Administrator concluded that, with revision of the standard level, the existing form and averaging time provided the control needed to achieve the cumulative seasonal exposure circumstances identified for the secondary standard (80 FR 65408, October 26, 2015). The focus on cumulative seasonal exposure primarily reflects the evidence on E-R relationships for plant growth. The 2015 conclusion was based on the air quality data analyzed at that time (80 FR 65408, October 26, 2015). Analyses in the current review of the now expanded set of air monitoring data, which includes 1,557 monitoring sites with sufficient data for derivation of design values (Appendix 4D, section 4D.2.2), document similar findings as from the analysis of data from 2000-2013 described in the last review. The current analyses, which span 19 years and 17 3-year periods, are described in detail in Appendix 4D.

One aspect of these analyses documents the positive nonlinear relationship that is observed between cumulative seasonal exposure, quantified using the W126 index, and design values, based on the form and averaging time of the current standard. This is shown for both the average W126 index across the 3-year design value period (Figure 4-7, left) and for annual index values within the period (Figure 4-7, right). From both of these presentations, it is clear that cumulative seasonal exposures, assessed in terms of W126 index, are lower at monitoring sites with lower design values. This is seen both for design values above the level of the current standard (70 ppb), where the slope is steeper (due to the sigmoidal weighting of higher concentrations by the W126 index function), as well as for lower design values that meet the current standard (Figure 4-7; Appendix 4D, Figure 4D-4).

These presentations also indicate some regional differences. For example, as shown in Figure 4-6 for the 2016-2018 period, sites meeting the current standard in the regions outside of the West and Southwest regions, all W126 index values are at or below 13 ppm-hrs (for either W126 metric). Ozone concentrations, and W126 index values, are generally higher in the West and Southwest regions (Figure 4-6). However, the positive relationship between the W126 index and the design value is evident in all regions (Figure 4-7).

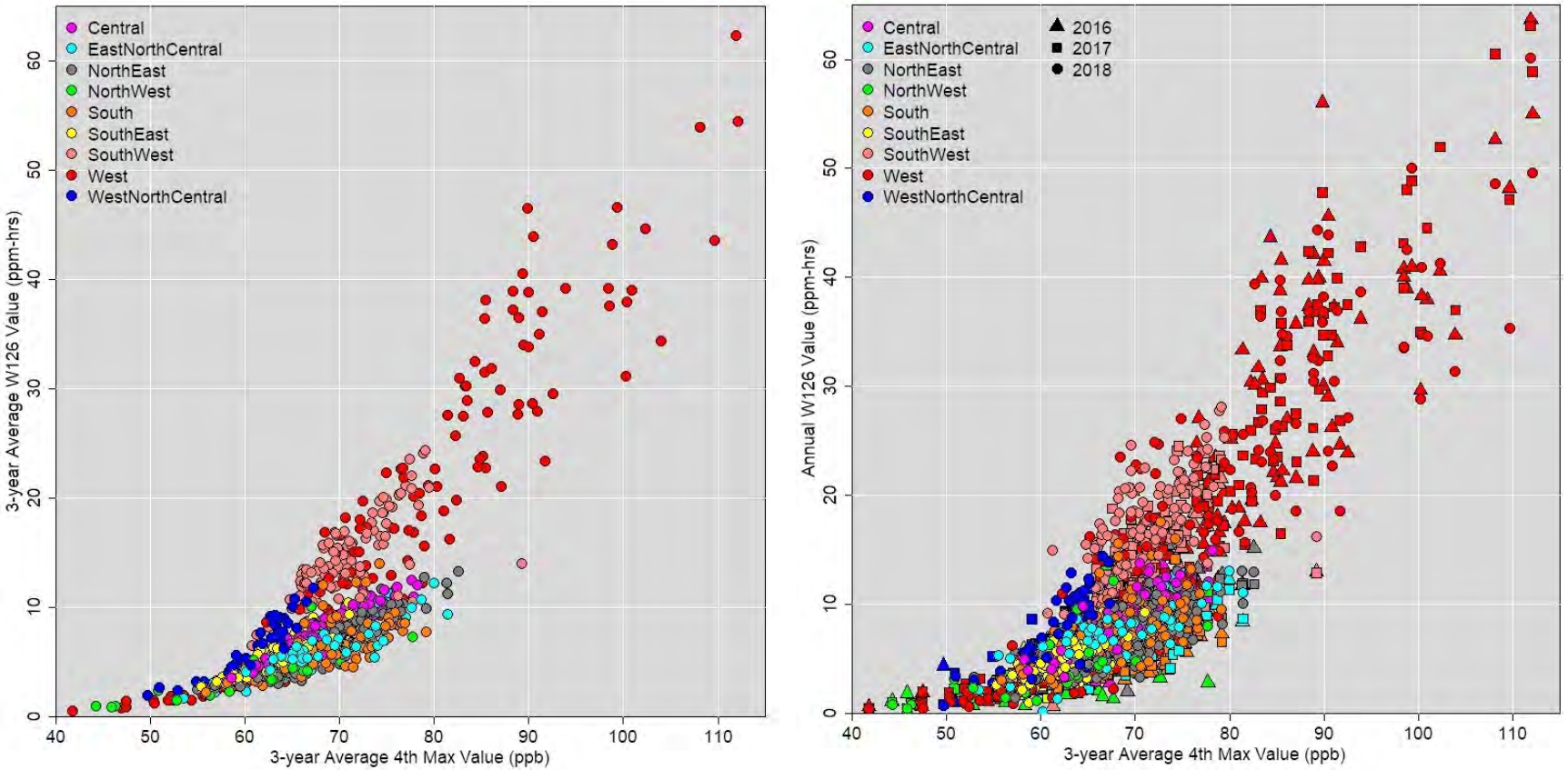


Figure 4-7. Relationship between the W126 index and design values for the current standard (2016-2018). The W126 index is analyzed in terms of averages across the 3-year design value period (left) and annual values (right).

An additional analysis assesses the relationship between long-term changes in design value and long-term changes in the W126 index. This type of analysis, which was also performed in the last review with the then-available data, is presented in detail in Appendix 4D (section 4D.3.2.2). The current analysis focuses on the relationship between changes (at each monitoring site) in the 3-year design value (termed "4th max" in Appendix 4D and Figures 4-7 and 4-8) across the 16 design value periods from 2000-2002 to 2016-2018 and changes in the W126 index over the same period.[60] This analysis, performed using either the 3-year average W126 index or annual values, shows there to be a positive, linear relationship between the changes in the W126 index and the changes in the design value at monitoring sites across the U.S. (Figure 4-8). This means that a change in the design value at a monitoring site was generally accompanied by a similar change in the W126 index. Nationally, the W126 index (in terms of 3-year average) decreased by approximately 0.62 ppm-hrs per ppb decrease in design value over the full period from 2000 to 2018. This relationship varies across the NOAA climate regions, with the greatest change in the W126 index per unit change in design value observed in the Southwest and West regions. Thus, the regions which had the highest W126 index values at site meeting the current standard (Figure 4-6) also showed the greatest improvement in the W126 index per unit decrease in their design values over the past 19 years (Appendix 4D, Table 4D-11and Figure 4D-12). This indicates that going forward as design values are reduced in areas that are presently not meeting the current standard, the W126 index in those areas would also be expected to decline (Appendix 4D, section 4D.3.2.3 and 4D.4).

The overall trend showing reductions in the W126 concurrent with the design value metric for the current standard is positive whether the W126 index is expressed in terms of the average across the 3-year design value period or the annual value (Appendix 4D, section 4D.3.2.3). This similarity is consistent with the relationship between the W126 index and the design value metric for the current standard summarized above, which shows a strong positive relationship between those metrics (Figure 4-7, Appendix 4D, section 4D.3.1.2).

[60] 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., $W126_1$, $W126_2$, $W126_3$, $W126_4$), the trend would be the median of the different per-year changes observed in the six possible pairs of values ([$W126_4$- $W126_3$]/1, [$W126_3$- $W126_2$]/1, [$W126_2$- $W126_1$]/1, [$W126_4$- $W126_2$]/2, [$W126_3$- $W126_1$]/2, [$W126_4$- $W126_1$]/3).

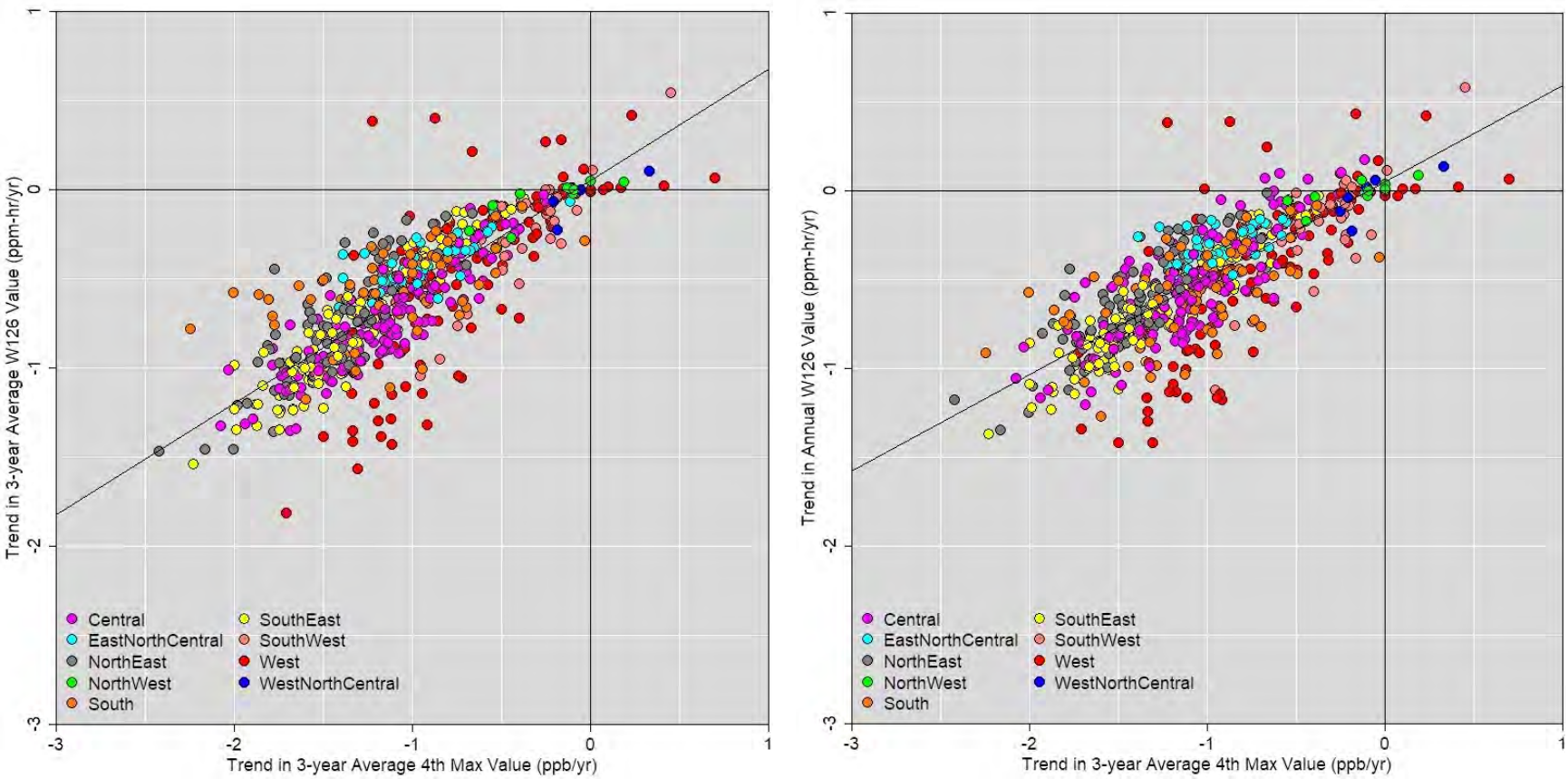


Figure 4-8. Relationship between trends in the W126 index and trends in design values across a 19-year period (2000-2018) at U.S. monitoring sites. W126 is analyzed in terms of averages across 3-year design value periods (left) and annual values (right).

In considering quantitative analyses concerning the control of the current form and averaging time on vegetation exposures of potential concern, we additionally take note of the evidence discussed in section 4.3.3.2 above regarding the potential for days with particularly high O_3 concentrations to play a contributing role in visible foliar injury. While the occurrence and severity of visible foliar injury indicates some relationship with cumulative concentration-weighted indices such as SUM06 and W126, the evidence also indicates a contributing role for occurrences of peak concentrations. We note that the current standard's form and averaging time, by their very definition, limit such occurrences. For example, the peak 8-hour average concentrations are lower at sites with lower design values, as illustrated by the declining trends in annual fourth highest MDA8 concentrations that accompany the declining trend in design values described in chapter 2 (e.g., Figure 2-11). Additionally, with regard to hourly concentrations, analyses summarized in Appendix 2A document decreasing frequency of elevated 1-hour concentrations (e.g., concentrations at or above 100 ppb) with decreasing design values (Appendix 2A, Tables 2A-2 through 2A-4). For example, in the most recent design value period (2016-2018) across all sites with adequate data to derive design values, the mean number of observations per site at or above 100 ppb was well below one (0.19) for sites that meet the current standard, compared to well above one (8.09) for sites not meeting the current (Appendix 2A, Table 2A-2).

In summary, monitoring sites with lower O_3 concentrations as measured by the design value metric (based on the current form and averaging time of the secondary standard) have lower cumulative seasonal exposures, as quantified by the W126 index (as well as lower short-term peak concentrations). As the form and averaging time of the secondary standard have not changed since 1997, the analyses performed have been able to assess the amount of control exerted by these aspects of the standard, in combination with reductions in the level (i.e., from 80 ppb in 1997 to 75 ppb in 2008 to 70 ppb in 2015) on cumulative seasonal exposures in terms of W126 index (and on the magnitude of short-term peak concentrations). The analyses have found that the reductions in design value, presumably associated with implementation of the revised standards, have been accompanied by reductions in cumulative seasonal exposures in terms of W126 index, as well as reductions in short-term peak concentrations.

4.4.2 Environmental Exposures in Terms of W126 Index

To inform the Administrator's exposure/risk-based considerations in the current review, we have developed updates to the air quality analyses of O_3 concentrations and W126 index values that were developed in the last review. Given the evidence indicating the W126 index to be strongly related to growth effects and its use in the E-R functions for tree seedling RBL, exposure is quantified using the W126 metric (Figure 4-9). In light of the importance placed on

Class I areas in past secondary standard reviews and the greater public welfare significance of O_3 related impacts in such areas, as discussed in section 4.3.2 above, a separate evaluation is conducted on cumulative O_3 exposure at monitoring sites in or near Class I areas[61], in addition to that at all monitoring sites nationwide. The potential for impacts of interest is assessed through considering the magnitude of estimated exposure in light of current information and in comparison to levels given particular focus in the 2015 decision on the current standard (80 FR 65292; October 26, 2015).[62]

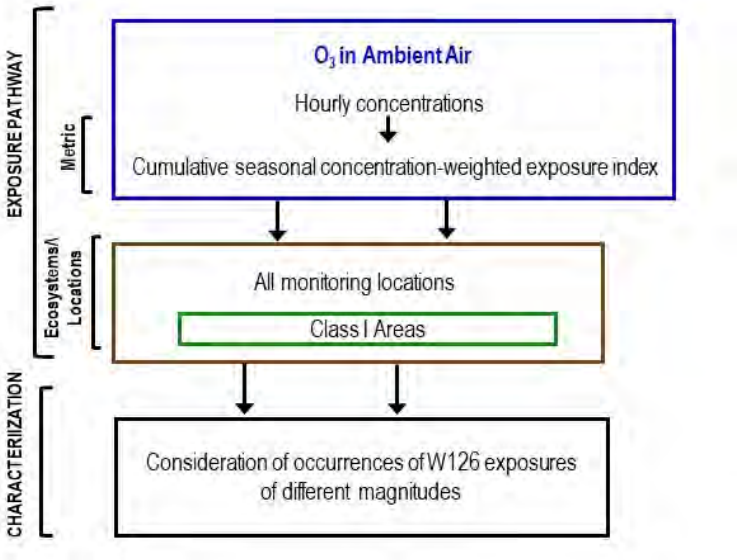


Figure 4-9. Analytical approach for characterizing vegetation exposure.

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 (2016 to 2018) 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.[63] Across the seventeen 3-year periods from 2000-2002 to 2016-2018, 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 1119 in 2015-2017. As specific monitoring sites differed somewhat across the

[61] Included are monitors sited within Class I areas or the closest monitoring site within 15 km of the area boundary.

[62] 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).

[63] Data adequacy requirements and methods for these calculations are described in Appendix 4D, section 4D.2.

19 years, there were 1,557 sites with sufficient data for calculation of valid design values and W126 index values for at least one 3-year period between 2000 and 2018, and 543 sites had such data for all seventeen 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 4D, section 4D.3.1.2). This evaluation was performed for all monitoring sites in the most recent 3-year period, 2016 to 2018. This analysis indicates the extent to which single-year values within the 3-year period deviate from the average for the period. Across the full set of sites, regardless of W126 index magnitude (or whether or not the current standard is met), single-year W126 index values differ no more than 12 or 13 ppm-hrs from the average for the 3-year period (Appendix 4D, Figure 4D-6). Focusing on the approximately 850 sites (Appendix 4D, Table 4D-1) meeting the current standard (design value at or below 70 ppb), over 99% of single-year W126 values in this subset differ from the 3-year average by no more than 5 ppm-hrs, and 87% by no more than 2 ppm-hrs (Appendix 4D, Figure 4D-7).

The following discussion is framed by a key policy-relevant question based on those identified in the IRP. The question considers all areas nationally, with particular focus on air quality data for Class I areas.

- **What are the nature and magnitude of vegetation exposures associated with conditions meeting the current standard at sites across the U.S., particularly in specially protected areas, such as Class I areas, and what do they indicate regarding the potential for O_3-related vegetation impacts?**

To further address this question, we considered both recent air quality (2016-2018) and air quality since 2000. These air quality analyses of cumulative seasonal exposures associated with conditions meeting the current standard nationally provide conclusions generally similar to those based on the data available at the time of the last review when the current standard was set, when the most recent data were available for 2011 to 2013 (Wells, 2015). Cumulative exposures vary across the U.S, with the highest W126 index values for sites that met the current standard being located exclusively in the Southwest and West climate regions (Figure 4-6, Appendix 4D, Table 4D-1). In all other NOAA climate regions, W126 index values at sites meeting the current standard are generally at or below 13 ppm-hrs (Figure 4-6 and Appendix 4D, Figure 4D-2). In the Southwest and West, W126 index values at all sites meeting the current standard are at or below 17 ppm-hrs on virtually all occasions in the most recent 3-year period and across all of the

seventeen 3-year periods in the full dataset evaluated.[64] That is, among sites meeting the current standard in the most recent period of 2016 to 2018, there are none with a W126 index, based on the 3-year average, above 19 ppm-hrs, and just one with such a value above 17 ppm-hrs (Table 4-1). Additionally, the historical dataset includes no occurrences of a 3-year average W126 index above 19 ppm-hrs at sites meeting the current standard, and just a small number of occurrences (limited to eight, all but one from a period prior to 2011) of a W126 index above 17 ppm-hrs, with the highest just equaling 19 ppm-hrs (Table 4-1; Appendix 4D, section 4D.3.2.1).

Given the recognition of more significant public welfare implications of effects in protected areas, such as Class I areas (as discussed in section 4.3.2 above), we give particular attention to Class I areas (Appendix 4D, section 4D.3.2.3). In so doing, we consider the updated air quality analysis presented in Appendix 4D for 65 Class I areas. The findings for these sites, which are distributed across all nine NOAA climate regions in the contiguous U.S., as well as Alaska and Hawaii, mirror all U.S. sites. Among the Class I area sites meeting the current standard (i.e., having a design value at or below 70 ppb) in the most recent period of 2016 to 2018, there are none with a W126 index (as average over design value period) above 17 ppm-hrs (Table 4-1). The historical dataset includes just seven occurrences (all dating from the 2000-2010 period) of a Class I area site meeting the current standard and having a 3-year average W126 index above 17 ppm-hrs, and no such occurrences above 19 ppm-hrs (Table 4-1).

The W126 exposures at sites with design values above 70 ppb range up to approximately 60 ppm-hrs (Table 4-1, Appendix 4D, Table 4D-17). Among all sites across the U.S. that do not meet the current standard in the 2016 to 2018 period, more than a quarter have average W126 index values above 19 ppm-hrs and a third exceed 17 ppm-hrs (Table 4-1).[65] A similar situation exists for Class I area sites (Table 4-1). Thus, as was the case in the last review, the currently available quantitative information continues to indicate appreciable control of seasonal W126 index-based cumulative exposure at all sites with air quality meeting the current standard.

[64] On 99.9 percent of occasions across all sites with valid design values at or below 70 ppb during the 2000 to 2018 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).

[65] 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.

Table 4-1. Distribution of 3-yr average seasonal W126 index for sites in Class I areas and 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)							
2016-2018	47	0	0	47	849	0	1	848
All from 2000 to 2018	498	0	7	491	8,292	0	8	8,284
	At sites that exceed the current standard (design value above 70 ppb)							
2016-2018	11	8	9	2	273	78	91	182
All from 2000 to 2018	362	159	197	165	10,695	2,317	3,174	7,521

[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.

As discussed in section 4.3.3 above, the evidence currently available leads us to similar conclusions regarding exposure levels associated with effects as in the last review. Based largely on this evidence in combination with the use of RBL as a surrogate or proxy for all vegetation-related effects, the value of 17 ppm-hrs was the average W126 index (over three years) was generally identified as a target level for protection in the 2015 decision (80 FR 65393; October 26, 2015). As summarized above, the information available in this review continues to indicate that average cumulative seasonal exposure levels at virtually all sites and 3-year periods with air quality meeting the current standard fall at or below the level of 17 ppm-hrs that, as summarized in section 4.1 above, was identified when the current standard was established (80 FR 65393; October 26, 2015). Additionally, the full dataset indicates that at sites meeting the current standard, annual W126 index values were less than or equal to 19 ppm-hrs well over 99% of the time (Appendix 4D, section 4D.3.2.1). Additionally, the average W126 index in Class I areas that meet the current standard for the most recent 3-year period is below 17 ppm-hrs in all areas for which there is a monitor in or nearby (Appendix 4D, Table 4D-16). Further, with the exception of seven values that occurred prior to 2011, cumulative seasonal exposures in all Class I areas during periods that met the current standard were no higher than 17 ppm-hrs. This contrasts with the occurrence of much higher seasonal W126 index values in sites when the current standard was not met. For example, out of the 11 Class I area sites with design values above 70 ppb during the most recent period, eight had had a W126 index (based on 3-year average) above 19 ppm-hrs (ranging up to 47 ppm-hrs) and nine sites had a W126 index above 17 ppm-hrs (Table 4-1; Appendix 4D, Table 4D-17).

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 O_3 monitoring sites nationwide. While there are inherent limitations in any air monitoring network, the monitors for O_3 are distributed across the U.S., covering all NOAA regions and all states (e.g., Figure 4-6).

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, and some geographical areas are more densely covered than others, which may have sparse or no data. For example, only about 40% of all Federal Class I Areas have or have had O_3 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, North East, 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 1 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 currently available information. Thus, it may or may not reflect conditions far out into the future as air quality and patterns of O_3 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 findings from these analyses in the current review are largely consistent with those from analyses of the data available in the last review. Further, we note the findings of the analysis in Appendix 4D of how changes in O_3 patterns in the past have affected the relationship between W126 index and the averaging time and form of the current standard, as represented by design values (Appendix 4D, section 4D.3.2.3). This analysis finds a positive, linear relationship between trends in design values and trends in the W126 index (both in terms of single-year W126 index and averages over 3-year design value period), as was also the case for similar analyses conducted for the data available at the time of the last review (Wells, 2015). While this relationship varies across NOAA regions, the regions showing the greatest potential for exceeding W126 index values of interest (e.g., with 3-year average values

above 17 and/or 19 ppm-hrs) also showed the greatest improvement in the W126 index per unit decrease in design value over the historical period assessed (Appendix 4D, section 4D.3.2.3). Thus, the available data and this analysis appear to indicate that as design values are reduced to meet the current standard in areas that presently do not, W126 values in those areas would also be expected to decline (Appendix 4D, section 4D.4).

4.5 KEY CONSIDERATIONS REGARDING THE CURRENT SECONDARY STANDARD

In considering what the currently available evidence and exposure/risk information indicate with regard to the current secondary O_3 standard, the overarching question we address is:

- **Does the currently available scientific evidence and air quality and exposure analyses support or call into question the adequacy of the protection afforded by the current secondary O_3 standard?**

To assist us in interpreting the currently available scientific evidence and the results of recent quantitative analyses to address this question, we have focused on a series of more specific questions. In considering the scientific and technical information, we consider both the information available at the time of the last review and information newly available since then which has been critically analyzed and characterized in the current ISA, the 2013 ISA and prior AQCDs. In so doing, an important consideration is whether the information newly available in this review alters the EPA's overall conclusions from the last review regarding welfare effects associated with photochemical oxidants, including O_3, in ambient air. We also consider the currently available quantitative information regarding environmental exposures, characterized by the pertinent metric, likely to occur in areas of the U.S. where the standard is met. Additionally, we consider the significance of these exposures with regard to the potential for O_3-related vegetation effects, their potential severity and any associated public welfare implications.

4.5.1 Evidence and Exposure/Risk-based Considerations

In considering first the currently available evidence with regard to the overarching question posed above regarding the protection provided by the current standard from welfare effects, we address a series of more specific questions that focus on policy-relevant aspects of the evidence. These questions relate to three main areas of consideration: (1) the available evidence on welfare effects associated with exposure to photochemical oxidants, and particularly O_3 (section 4.5.1.2); (2) the risk management framework or approach for reaching conclusions on the adequacy of protection provided by the secondary standard (section 4.5.1.2); and, (3)

findings from the air quality and exposure analyses pertaining to public welfare protection under the current standard (section 4.5.1.3).

4.5.1.1 Welfare Effects Evidence

- **Is there newly available evidence that indicates the importance of photochemical oxidants other than O_3 with regard to abundance in ambient air, and potential for welfare effects?**

No newly available evidence has been identified in this review regarding the importance of photochemical oxidants other than O_3 with regard to abundance in ambient air, and potential for welfare effects.[66] As summarized in section 2.1 above, O_3 is one of a group of photochemical oxidants formed by atmospheric photochemical reactions of hydrocarbons with nitrogen oxides in the presence of sunlight, with O_3 being the only photochemical oxidant other than nitrogen dioxide that is routinely monitored in ambient air (ISA, Appendix 1, section 1.1).[67] Data for other photochemical oxidants are generally derived from a few special field studies; such that national scale data for these other oxidants are scarce (ISA, Appendix 1, section 1.1; 2013 ISA, sections 3.1 and 3.6). Moreover, few studies of the welfare effects of other photochemical oxidants beyond O_3 have been identified by literature searches conducted for the 2013 ISA and prior AQCDs (ISA; Appendix 1, section 1.1). As stated in the current ISA, "the primary literature evaluating the health and ecological effects of photochemical oxidants includes ozone almost exclusively as an indicator of photochemical oxidants" (ISA, section IS.1.1). Thus, as was the case for previous reviews, the evidence base for welfare effects of photochemical oxidants does not indicate an importance of any other photochemical oxidants. For these reasons, discussion of photochemical oxidants in this document focuses on O_3.

- **Does the current evidence alter conclusions from the last review regarding the nature of welfare effects attributable to O_3 in ambient air?**

The current evidence, including that newly available in this review, supports, sharpens and expands somewhat on the conclusions reached in the last review (ISA, sections IS.1.3.2 and IS.5 and Appendices 8 and 9). A wealth of scientific evidence, spanning more than six decades, demonstrates effects on vegetation and ecosystems of O_3 in ambient air (ISA, section IS.6.2.1; 2013 ISA, 2006 AQCD, 1997 AQCD, 1986 AQCD; U.S. DHEW, 1970). Accordingly, consistent with the evidence in the last review, the currently available evidence describes an array of O_3 effects on vegetation and related ecosystem effects. The evidence also describes climate effects

[66] Close agreement between past ozone measurements and the photochemical oxidant measurements upon which the early NAAQS (for photochemical oxidants including O_3) was based indicated the very minor contribution of other oxidant species in comparison to O_3 (U.S. DHEW, 1970).

[67] Consideration of welfare effects associated with nitrogen oxides in ambient air is addressed in the review of the secondary NAAQS for oxides of nitrogen and oxides of sulfur (U.S. EPA, 2018).

of tropospheric O_3, through a role in radiative forcing and subsequent effects on temperature, precipitation and related climate variables. Evidence newly available in this review strengthens previous conclusions, provides further mechanistic insights and augments current understanding of varying effects of O_3 among species, communities and ecosystems (ISA, section IS.6.2.1). The current evidence, including a wealth of longstanding evidence, supports conclusions reached in the last review of causal relationships between O_3 and visible foliar injury, reduced yield and quality of agricultural crops, reduced vegetation growth and plant reproduction,[68] reduced productivity in terrestrial ecosystems, and alteration of belowground biogeochemical cycles. The current evidence, including a wealth of longstanding evidence, also supports conclusions reached in the last review of likely causal relationships between O_3 and reduced carbon sequestration in terrestrial systems, and alteration of terrestrial ecosystem water cycling (ISA, section IS.I.3.2). Additionally, as in the last review, the current ISA determines there to be a causal relationship between tropospheric O_3 and radiative forcing and a likely causal relationship between tropospheric O_3 and temperature, precipitation and related climate variables (ISA, section IS.1.3.3). Further, the current evidence has led to an updated conclusion on the relationship of O_3 with alteration of terrestrial community composition to causal (ISA, sections IS.I.3.2). Lastly, the current ISA concludes the current evidence sufficient to infer likely causal relationships of O_3 with three additional categories of effects (ISA, sections IS.I.3.2). While previous recognition of O_3 as a contributor to tree mortality in a number of field studies was a factor in the 2013 conclusion regarding composition composition, it has been separately assessed in this review. Additionally, evidence newly available in this review on two additional plant-related effects augments more limited previously available evidence related to insect interactions with vegetation, contributing to additional conclusions that the body of evidence is sufficient to infer likely causal relationships between O_3 and alterations of plant-insect signaling and insect herbivore growth and reproduction (ISA, Appendix 8, sections 8.6 and 8.7).[69]

As in the last review, the strongest evidence and the associated findings of causal or likely causal relationships with O_3 in ambient air, and quantitative characterizations of relationships between O_3 exposure and occurrence and magnitude of effects are for vegetation-related effects, and particularly those identified in the last review. The evidence base for the newly identified category of increased tree mortality includes previously available evidence

[68] As noted in section 4.3.1 above, the ISA in this review includes a causality determination specific to reduced plant reproduction, while this category of effects was considered in combination with reduced plant growth in the last review (ISA, Table IS.13).

[69] As in the last review, the 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).

largely comprised of field observations from locations and periods of O_3 concentrations higher than are common today and three more recently available publications assessing O_3 exposures not expected under conditions meeting the current standard. Among the three more recent publications, one assessed survival of aspen clones across an 11-year period under O_3 exposures that included single-year seasonal W126 index values ranging above 30 ppm-hrs during the first four years, and the other two were analyses based on field observations during periods when O_3 concentrations were such that they would not be expected to meet the current standard, as summarized in section 4.3.1 above (ISA, Appendix 8, section 8.4.3).

The information available regarding the newly identified categories of plant-insect signaling and insect herbivore growth and reproduction does not provide for a clear understanding of the specific environmental effects that may occur in the natural environment under specific exposure conditions (as discussed in sections 4.3.1, 4.3.3.2 and 4.3.4 above). For example, while the evidence base for effects on herbivore growth and reproduction is expanded in this review, "there is no clear trend in the directionality of response for most metrics," such that some show an increased effect and some show reductions (ISA, p. IS-64; section IS.5.1.3 and section 8.6). More specifically "no consistent directionality of response is observed across the literature, and uncertainties remain in regard to different plant consumption methods across species and the exposure conditions associated with particular severities of effects" (ISA, p. IS-91). Additionally, while the current evidence base documents effects of O_3 on some plant VPSCs (e.g., changing the floral scent composition and reducing dispersion), and indicates reduced pollinator attraction, decreased plant host detection and altered plant-host preference in some insect species in the presence of elevated O_3 concentrations, characterization of such effects is still "an emerging area of research with information available on a relatively small number of insect species and plant-insect associations," and with gaps remaining in the consequences of modification of signaling compounds by O_3 in natural environments (ISA, p. IS-91 and section IS.6.2.1). Accordingly, the focus in this review is on other vegetation effects described above, rather than these two newly identified categories.

With regard to tropospheric O_3 and effects on climate, we recognize the strength of the conclusion that tropospheric O_3 is a greenhouse gas at the global scale, with associated effects on climate (ISA, section 9.1.3.3). Accordingly, as indicated by the ISA causal determinations, O_3 abundance in the troposphere contributes to radiative forcing and likely also to subsequent climate effects. There is appreciable uncertainty, however, associated with understanding quantitative relationships involving regional O_3 concentrations near the earth's surface and climate effects of tropospheric O_3 on a global scale As recognized in the ISA (and summarized in sections 4.3.3.3 and 4.3.4 above), there are limitations in our modeling tools and associated uncertainties in interpretations related to capabilities for quantitatively estimating effects of

regional-scale lower tropospheric O_3 concentrations on climate. Thus, while additional characterizations of tropospheric O_3 and climate have been completed since the last review, uncertainties and limitations in the evidence that were also recognized in the last review remain. As summarized in sections 4.3.3.3 and 4.3.4 above, these affect our ability to make a quantitative characterization of the potential magnitude of climate response to changes in O_3 concentrations in ambient air, particularly at regional (*vs* global) scales, and thus our ability to assess the impact of changes in ambient air O_3 concentrations in regions of the U.S. on global radiative forcing or temperature, precipitation and related climate variables. Consequently, the current evidence in this area is not informative to our consideration of the adequacy of public welfare protection of the current standard.

- **To what extent does the available evidence provide E-R information (e.g., quantitative E-R relationships) for O_3-related effects that can inform judgments on the likelihood of occurrence of such effects in areas with air quality that meets the current standard? Does the currently available evidence provide new or altered such information since the last review?**

In considering what the currently available information indicates with regard to exposures associated with welfare effects and particularly in the context of what is indicated for exposures associated with air quality conditions that meet the current standard, we focus particularly on the availability of quantitatively characterized E-R relationships for key effects. While the ISA describes additional studies of welfare effects associated with O_3 exposures since the last review, the established E-R functions for tree seedling growth and crop yield that have been available in the last several reviews continue to be the most robust descriptions of E-R relationships for welfare effects. These well-established E-R functions for seedling growth reduction in 11 tree species and yield loss in 10 crop species are based on response information across multiple levels of cumulative seasonal exposure (estimated from extensive records of hourly O_3 concentrations across the exposure periods). Studies of some of the same species, conducted since the E-R function derivation, provide supporting information for these functions (ISA, Appendix 8, section 8.13.2; 2013 ISA, sections 9.6.3.1 and 9.6.3.2). The E-R functions provide for estimation of growth-related effects for a range of cumulative seasonal exposures.

The evidence newly available in this review does not include new studies that assessed reductions in tree growth or crop yield responses across multiple O_3 exposures and for which sufficient data are available for analyses of the shape of the E-R relationship across the range of cumulative exposure levels (e.g., in terms of W126 index) relevant to conditions associated with the current standard. For example, among the newly available studies are several that summarize

previously available studies or draw from them, such as for linear regression analyses.[70] However, as discussed in sections 4.3.3.2 above, these do not provide robust E-R functions or cumulative seasonal exposure levels associated with important vegetation effects that define the associated exposure circumstances in a consistent manner, limiting their usefulness for our purposes here with regard to considering the potential for occurrence of welfare effects in air quality conditions that meet the current standard. Thus, robust E-R functions are not available for growth or yield effects on any additional tree species or crops in this review.

Based on these established E-R functions for tree seedling growth reductions in 11 species, the tree seedling RBL for the median tree species is 5.3% for a W126 index of 17 ppm-hrs, rising to 5.7% for 18 ppm-hrs, 6.0% for 19 ppm-hrs and 6.4% for 20 ppm-hrs. Below 17 ppm-hrs, the median estimates include 4.9% for 16 ppm-hrs, 4.5% for 15 ppm-hrs, 4.2% for 14 ppm-hrs and 3.8% for 13 ppm-hrs (Appendix4A, Table 4A-5). These RBL estimates are unchanged from what was indicated by the evidence in the last review. As summarized in section 4.1 above, the RBL estimates were used in the 2015 decision as a surrogate or proxy for the broader array of vegetation-related effects.

With regard to visible foliar injury, as in the last review, we lack established E-R relationships that would quantitatively describe relationships between visible foliar injury (occurrence and incidence, as well as, injury severity) and O_3 exposure, as well as factors influential in those relationships, such as soil moisture conditions. As discussed in section 4.3.3.2 above, the currently available evidence continues to include both experimental studies that document foliar injury in specific plants in response to O_3 exposures, and quantitative analyses of the relationship between environmental O_3 exposures and occurrence of foliar injury. The analyses involving environmental conditions, while often using cumulative exposure metrics to quantify O_3 exposures additionally have reported there to also be a role for a metric that quantifies the incidence of "high" O_3 days (2013 ISA, p. 9-10; Smith, 2012; Wang et al., 2012). However, such analyses have not established specific air quality metrics and associated quantitative functions for describing the influence of ambient air O_3 on incidence and severity of visible foliar injury.

[70] For example, among the newly available publications cited in the ISA is a publication on tree and grassland species that compiles EC_{10} values (estimated concentration at which 10% lower biomass [compared to zero O_3] is predicted) derived using linear regression of previously published data on plant growth response and O_3 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 O_3 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).

Multiple studies have involved quantitative analysis of data collected as part of the USFS biosite biomonitoring program (e.g., Smith, 2012). These analyses continue to indicate the limitations in capabilities for predicting the exposure circumstances under which visible foliar injury would be expected to occur, as well as the circumstances contributing to increased injury severity. As noted in section 4.3.3.2 above, expanded summaries of the dataset compiled in the 2015 review from several years of USFS biosite records does not clearly and consistently describe the shape of a relationship between incidence of foliar injury or severity (based on individual site scores) and W126 index estimates. Overall, however, the dataset indicates that the proportion of records having different levels of severity score is generally highest in the group of records for sites with the highest W126 index (e.g., greater than 25 ppm-hrs for the normal and dry soil moisture categories). Thus, the currently available evidence indicates increased occurrence and severity at the highest category of exposures in the dataset (above 25 ppm-hrs in terms of a W126 index), but does not provide for identification of air quality conditions, in terms of O_3 concentrations associated with the relatively lower environmental exposures most common in the USFS dataset that would correspond to a specific magnitude of injury incidence or severity scores across locations.

Thus, based on considering the available information for the array of O_3 welfare effects, we again recognize the E-R relationships available in the last review for purposes of considering O_3 exposure levels associated with growth-related impacts to be the most robust E-R information available. The currently available evidence for growth-related effects, including that newly available in this review, does not indicate the occurrence of growth-related responses attributable to cumulative O_3 exposures lower than was established at the time of the last review. With regard to visible foliar injury, the available information continues to be limited with regard to estimating occurrence and severity across a range of air quality conditions, providing for only limited and somewhat qualitative conclusions related to potential occurrence and/or severity under different air quality conditions. The quantitative information for other effects is still more limited, as recognized in sections 4.3.3 and 4.3.4 above. Thus, the newly available evidence does not appreciably address key limitations or uncertainties needed to expand capabilities for estimating welfare impacts that might be expected as a result of differing patterns of O_3 concentrations in the U.S.

- **Does the current evidence continue to support a cumulative, seasonal exposure index, such as the W126 function, as a biologically relevant and appropriate metric for assessment of vegetation-related effects of O_3 in ambient air?**

As in the last review, the currently available evidence continues to support a cumulative, seasonal exposure index as a biologically relevant and appropriate metric for assessment of the evidence of exposure/risk information for vegetation, most particularly for growth-related

effects. The most commonly used such metrics are the SUM06, AOT40 (or AOT60) and W126 indices (ISA, section IS.3.2).[71] The evidence for growth-related effects continues to support important roles for cumulative exposure and for weighting higher concentrations over lower concentrations. Thus, among the various such indices considered in the literature, the cumulative, concentration-weighted metric, defined by the W126 function, continues to be best supported for purposes of relating O_3 air quality to growth-related effects. Accordingly, in our consideration of the potential for vegetation-related effects to occur under air quality conditions associated with the current standard, we continue to focus on the W126 index as the appropriate metric. In so doing, we also recognize, as recognized in the past, that this metric may not well describe the key circumstances of O_3 exposure for occurrences of other effects, particularly, visible foliar injury. As discussed in section 4.3.3.2 above, the evidence indicates an important role for peak concentrations (e.g., N100) in influencing the occurrence and severity of visible foliar injury.

4.5.1.2 General Approach for Considering Public Welfare Protection

The general approach and risk management framework applied in 2015 for making judgements and reaching conclusions regarding the adequacy of public welfare protection provided by the newly established secondary standard is summarized in section 4.1 above. In light of the current evidence and air quality information, we discuss here key considerations in judging public welfare protection provided by the O_3 secondary standard. In so doing, we address a series of questions.

- **Does the newly available information continue to support the use of tree seedling RBL as a proxy for the broad array of vegetation-related effects?**

As summarized in section 4.3 above, the currently available evidence is largely consistent with that available in the last review and does not call into question conceptual relationships between plant growth impacts and the broader array of vegetation effects. Rather, the ISA for the current review describes (or relies on) such relationships in considering causality determinations for ecosystem-scale effects such as altered terrestrial community composition and reduced productivity, as well as reduced carbon sequestration, in terrestrial ecosystems (ISA, Appendix 8, sections 8.8 and 8.10). Thus, the current evidence continues to support the use of tree seedling RBL as a proxy for the broad array of vegetation-related effects, most particularly those conceptually related to growth.

[71] While the evidence includes some studies reporting O_3-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.

In the last review, the Administrator recognized the view that an appropriate consideration of RBL was as a surrogate for an array of adverse welfare effects. Based on consideration of ecosystem services and potential for impacts to the public, as well as conceptual relationships between vegetation growth effects and ecosystem-scale effects, biomass loss could be appropriately described as a scientifically valid surrogate of a variety of adverse effects to public welfare (Frey, 2014, pp. iii, 9-10).[72] In light of this, and in consideration of the broader evidence base and public welfare implications, including associated strengths, limitations and uncertainties, the Administrator focused on RBL, not simply in making judgments specific to a magnitude of growth effect in seedlings that would be acceptable or unacceptable in the natural environment, but as a surrogate or proxy for consideration of the broader array of vegetation-related effects of potential public welfare significance, that included effects on growth of individual sensitive species and extended to ecosystem-level effects, such as community composition in natural forests, particularly in protected public lands (80 FR 65406, October 26, 2015). The information available in this review does not call into question this approach, indicating there to be continued support for the use of tree seedling RBL as a proxy for the broad array of vegetation-related effects, most particularly those conceptually related to growth.

Beyond tree seedling growth, on which RBL is specifically based, two other vegetation effect categories with extensive evidence bases are crop yield and visible foliar injury. In setting the current standard in 2015, as summarized in section 4.1 above, the Administrator considered what those evidence bases indicated regarding the need for additional protection specifically for those effects, judging that the available information, with associated limitations and uncertainties, did not provide support for such a conclusion. With regard to crop yield, she recognized the significant role of agricultural management practices in agricultural productivity, as well as market variability, concluding that, in describing her public welfare protection objectives, additional attention to this endpoint was not necessary. The rough similarities in estimated W126 levels of median crops and tree species are also noteworthy. In the 2015 decision, the Administrator concluded that a standard set based on public welfare protection

[72] The CASAC letter on the second draft PA in that review stated the following (Frey, 2014, p. 9-10):

> For example, CASAC concurs that trees are important from a public welfare perspective because they provide valued services to humans, including aesthetic value, food, fiber, timber, other forest products, habitat, recreational opportunities, climate regulation, erosion control, air pollution removal, and hydrologic and fire regime stabilization. Damage effects to trees that are adverse to public welfare occur in such locations as national parks, national refuges, and other protected areas, as well as to timber for commercial use. The CASAC concurs that biomass loss in trees is a relevant surrogate for damage to tree growth that affects ecosystem services such as habitat provision for wildlife, carbon storage, provision of food and fiber, and pollution removal. Biomass loss may also have indirect process-related effects such as on nutrient and hydrologic cycles. Therefore, biomass loss is a scientifically valid surrogate of a variety of adverse effects to public welfare.

objectives described in terms of cumulative exposures and relationships with tree seedling RBL was an appropriate means to, and would, provide appropriate protection for the array of vegetation-related effects. In summary, the information available in the current review does not call into question such conclusions and continues to be supportive of the use of tree seedling RBL as a proxy for the broad array of vegetation-related effects. Such effects and key considerations with regard to protection afforded by the current standard are addressed in section 4.5.1.3 below.

- **To what extent does the available information alter our understanding of an appropriate magnitude of RBL, in its role as a surrogate or proxy, reasonably expected to be of public welfare significance?**

In considering the RBL estimate on which to focus in its role as a surrogate or proxy for the full array of vegetation effects in the last review, the Administrator endeavored to identify a secondary standard that would limit 3-year average O_3 exposures somewhat below W126 index values associated with a 6% RBL median estimate from the established species-specific E-R functions.[73] This led to identification of a seasonal W126 index value of 17 ppm-hrs that the Administrator concluded appropriate as a target at or below which the new standard would generally restrict cumulative seasonal exposures (80 FR 65407, October 26, 2015). In identifying this exposure level as a target, the Administrator, recognizing limitations and uncertainties in the evidence and variability in biota and ecosystems in the natural environment, additionally judged that RBL estimates associated with isolated rare instances of marginally higher cumulative exposures (in terms of a 3-year average W126 index), e.g., those that round to 19 ppm-hrs (which corresponds to 6% RBL as median of 11 established E-R functions), were not indicative of adverse effects to the public welfare (80 FR 65409, October 26, 2015).

The information newly available in this review does not differ from that available in the last review with regard to a magnitude of RBL in the median species appropriately considered a reference for judgments concerning potential vegetation-related impacts to the public welfare. The currently available evidence continues to indicate conceptual relationships between reduced growth and the broader array of vegetation-related effects. Quantitative representations of such relationships have been used to study potential impacts of tree growth effects on such larger-scale effects as community composition and productivity with the results indicating the array of complexities involved (e.g., ISA, Appendix 8, section 8.8.4). Given their purpose in exploring complex ecological relationships and their responses to environmental variables, as well as limitations of the information available for such work, these analyses commonly utilize

[73] The CASAC in the last review stated that 6% RBL in seedlings for the median tree species (a metric it described as a valid surrogate for consideration of broader public welfare impacts) was "unacceptably high" in the context of protecting against "current and anticipated welfare effects of ozone" (Frey, 2014, p. iii).

somewhat general representations. This work indicates how established the existence of such relationships is, while also identifying complexities inherent in quantitative aspects of such relationships and interpretation of estimated responses. Thus, the currently available evidence is little changed from the last review with regard to informing identification of an RBL reference point reflecting ecosystem-scale effects with public welfare impacts elicited through such linkages.

- **What does the information available in the current review indicate with regard to support for use of a 3-year average seasonal W126 index as the cumulative exposure metric (associated with a target value of 17 ppm-hrs) for describing the public welfare protection objectives for the secondary standard?**

In setting the current standard, as described in section 4.1 above, the Administrator focused on control of seasonal cumulative exposures in terms of a 3-year average W126 index. The evaluations in the PA for that review recognized there to be limited information to discern differences in the level of protection afforded for cumulative growth-related effects by a standard focused on a single-year W126 index as compared to a 3-year W126 index (80 FR 65390). Accordingly, the identification of the 3-year average for considering the seasonal W126 index recognized that there was year-to-year variability not just in O_3 concentrations, but also in environmental factors, including rainfall and other meteorological factors, that influence the occurrence and magnitude of O_3-related effects in any year (e.g., through changes in soil moisture), contributing uncertainties to projections of the potential for harm to public welfare (80 FR 65404 October 26, 2015). Based on this recognition, as well as other considerations, the Administrator expressed greater confidence in judgments related to projections of public welfare impacts based on seasonal W126 index estimated by a 3-year average and accordingly, relied on that metric.

Among the factors referenced in the Administrator's 2015 decision to focus on a 3-year average W126 in assessing potential impacts on vegetation and protection from O_3 in ambient air, were consideration of the strengths and limitations of the evidence, and of the information on which to base her judgments with regard to adversity of effects on the public welfare. With regard to the current evidence we first consider the evidence and information underlying the E-R functions and the extent to which it is specific to a single seasonal exposure, e.g., as compared to an average across multiple seasons. In so doing, we also take note of aspects of the evidence that reflect variability in organism response under different experimental conditions and the extent to which this variability is represented in the available data, which might indicate an appropriateness of assessing environmental conditions using a mean across seasons in recognition of the existence of such year-to-year variability in conditions and responses. An additional aspect of the information underlying the E-R functions that may be relevant to

consider is the extent to which the exposure conditions represented include those associated with O_3 concentrations that meet the current standard, and the extent to which tree seedling growth responses to such conditions may have been found to not be significantly different from responses to the control (e.g., zero O_3) conditions. The extent to which E-R predictions are extrapolated beyond the tested exposure conditions also contributes to uncertainty which may argue for a less precise interpretation, such as an average across multiple seasons.

As an initial matter, we note that while the tree seedling E-R functions for the 11 species have been derived in terms of a seasonal (single-year) W126 index, the experiments from which they were derived vary in duration from periods of 82 to 140 days over a single year to periods of 180 to 555 days across two years (Appendix 4A, Table 4A-5; Lee and Hogsett, 1996). In order to produce E-R functions for 3-month periods from the experiments of variable durations adjustments were made based on assumptions regarding relationships between duration, cumulative exposure in terms of W126 index, and plant growth response (Lee and Hogsett, 1996). Specifically, the E-R functions were derived based on the exposure duration of the experiment and adjusted or normalized to 3-month periods based on assumptions regarding relationships between duration, cumulative exposure in terms of the W126 index and plant growth response (see Lee and Hogsett, 1996, section I.3). For example, while the functions are defined as describing a seasonal response, some were derived by distributing responses observed at the end of two seasons of varying exposures equally across the two seasons (essentially applying the average to both seasons). Thus, the growth response data for some of the 11 species with established E-R functions were collected over time periods longer than a single year's growing season and those functions are reflecting the average E-R relationship across the longer period. Consequently, they cannot provide precise estimates of response from a single year's exposure (e.g., *vs* averages over a longer period which may span multiple growing seasons).

Additionally, the number of experiments available for each species for which E-R functions have been established also varies. For example, there are 14 experimental studies for aspen (seven for which the E-R function for wild aspen has been derived and seven supporting a function for aspen clones) and 11 for ponderosa pine (Appendix 4A, Table 4A-5). The number of studies available for the other species, however, is much lower, e.g., only two or three for the three species generally exhibiting greater sensitivity than aspen and ponderosa pine (based on the available data) (Appendix 4A, section 4A-2, Table 4A-5; 1996 AQCD, Table 5-28; Lee and Hogsett, 1996). The E-R functions for both the aspen and ponderosa pine experiments illustrate appreciable variability in response across experiments (Appendix 4A, Figure 4A-10). Reasons for this variability may relate to a number of factors, including variability in seasonal response related to variability in non-O_3 related environmental influences on growth, such as rainfall, temperature and other meteorological variables, as well as biological variability across individual

seedlings, in addition to potentially variability in the pattern of O_3 concentrations contributing to similar cumulative exposures.

Regarding the extent or strength of the database underlying the E-R functions for cumulative exposure levels of interest in the current review, we note that the data appear to be more extensive for relatively higher (e.g., at/above a SUM06 of 30 ppm-hrs) vs lower seasonal exposures (Appendix 4A, Table 4A-6). Additionally, there are differences across the experimental studies in the extent to which they include O_3 exposure levels, in terms of W126 index, that commonly occur under air quality conditions that meet the current standard. For example, the studies appear to be somewhat limited for W126 index values below 20 ppm-hrs. As recognized in section 4.3.4 above, studies for five of the 11 species appear to have included exposure treatments likely to correspond to W126 index values at or below 20 ppm-hrs (Appendix 4A, Table 4A-5).[74] We additionally note that for at least one of these species, black cherry, the growth impacts for the lower exposure were not statistically significantly different from those for the next higher cumulative exposure (Appendix 4A, Table 4A-6).

There is also limited evidence that allows for specific evaluation of the predictability of growth impacts from single-year versus multiple-year average exposure estimates. Such evidence includes multi-year studies reporting results for each year of the study, which are the most informative to the question of plant annual and cumulative responses to individual years (high and low) over multiple-year periods. As summarized in section 4.3.4 above, the evidence is quite limited with regard to studies of O_3 effects that report seasonal observations across multi-year periods and that also include detailed hourly O_3 concentration records (to allow for derivation of exposure index values). One such study, which tracked exposures across six years, is available for aspen (King et al., 2005; 2013 ISA, section 9.6.3.2; ISA, Appendix 8, section 8.13.2). This study was used in a presentation of the 2013 ISA that compared the observed growth response to that predicted from the E-R function for aspen. Specifically, the observed aboveground biomass (and RBL) after each of the six growing seasons was compared to estimates derived from the aspen E-R function based on the cumulative multiple-year average seasonal W126 index values for each year[75] (2013 ISA, section 9.6.3.2). The conclusions reached

[74] For five of the species in Table 4A-5 in Appendix 4A, SUM06 index values below 25 ppm-hrs range from 12 to 21.7. In considering these values, we note that an approach used in the 2007 Staff Paper on specific temporal patterns of O_3 concentrations concluded that a SUM06 index value of 25 ppm-hrs would be estimated to correspond to a W126 index value of approximately 21 ppm-hrs (U.S. EPA, 2007, Appendix 7B, p. 7B-2). Accordingly, we conclude that a SUM06 value of 21 ppm-hrs would be expected to correspond to a W126 index value below 20 ppm-hrs.

[75] Although not emphasized or explained in detail in the 2013 ISA, the W126 estimates used to generate the predicted growth response were cumulative average. To clarify, the cumulative average W126 for year 1 is simply the W126 index for that year (e.g., based on highest 3 months). For year 2, it is the average of the year 1

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 O_3 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). Further analyses using observations from the multi-year study analyzed in the ISA (King et al., 2005) are presented in Appendix 4A. These analyses suggest that estimation of aboveground aspen biomass over a multi-year period using the established E-R function for aspen with a constant single-year W126 index, e.g., of 17 ppm-hrs, or with varying annual W126 index values (10, 17 and 24 ppm-hrs) for which the 3-year average is 17 ppm-hrs may yield similar total biomass estimates after multiple years (Appendix 4A, section 4A.3).

Thus, while the E-R functions are based on strong evidence of seasonal and cumulative seasonal O_3 exposure reducing tree growth, and while they provide for quantitative characterization of the extent of such effects across O_3 exposure levels of appreciable magnitude, there is uncertainty associated with the resulting RBL predictions. Further, the current evidence does not indicate single-year seasonal exposure in combination with the established E-R functions to be a better predictor of RBL than a seasonal exposure based on a multi-year average, or vice versa (Appendix 4A, section 4A.3.1). Rather, there is uncertainty, implying an imprecision or inexactitude, in the resulting predictions. In light of this, the current evidence does not support concluding there to be an appreciable difference in the effect of three years of exposure held at 17 ppm-hrs compared to a 3-year exposure that averaged 17 ppm-hrs yet varies by 5 to 10 ppm (e.g., 7 ppm-hrs) from that in any of the three years. All of the factors identified here, the currently available evidence and recognized limitations, variability and uncertainties, contribute uncertainty and resulting imprecision or inexactitude to RBL estimates of single-year seasonal W126 index values. Thus, the available information indicates no lesser support for use of an average seasonal W126 index derived from multiple years (with their representation of variability in environmental factors), such as for a 3-year period, for estimating median RBL using the established E-R functions, than for a single-year index.

- **What does the currently available information indicate for considering potential public welfare protection from O_3-related visible foliar injury?**

In establishing the current secondary standard in 2015 and its underlying public welfare protection objectives, as summarized in section 4.1, above, the Administrator focused primarily on RBL in tree seedlings as a proxy or surrogate for the full array of vegetation related effects of O_3 in ambient air, from sensitive species to broader ecosystem-level effects. In so doing, she also

seasonal W126 and year 2 seasonal W126, and so on. So that for year 6 it is the average of each of the six year's seasonal W126 index values.

concluded there to be support for establishing a strengthened standard provided by the then-available information regarding visible foliar injury, taking note that the available analyses of USFS biosite data, which indicated declines in BI scores with reductions in cumulative W126 from well above 20 ppm-hrs to lower levels (80 FR 65407-65408, October 26, 2015). She also concluded, however, that, due to associated uncertainties and complexities, the evidence was not conducive to use for identifying a quantitative public welfare protection objective focused specifically visible foliar injury. In reaching this conclusion, she specifically recognized significant challenges in judging the specific extent and severity at which such effects should be considered adverse to public welfare, in light of the variability in the occurrence of visible foliar injury and the lack of clear quantitative relationships (including robust exposure-response functions) that would allow prediction of visible foliar injury severity and incidence under varying air quality and environmental conditions, as well as the lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this vegetation effect (80 FR 65407, October 26, 2015).

As an initial matter, we note that, as recognized in the last review, some level of visible foliar injury can impact public welfare and thus might reasonably be judged adverse to public welfare.[76] As summarized in section 4.3.2 above, depending on its spatial extent and severity, there are many locations in which visible foliar injury can adversely affect the public welfare. For example, significant, readily perceivable and widespread injury in national parks and wilderness areas can adversely impact the perceived scenic beauty of these areas, impacting the aesthetic experience for both outdoor enthusiasts and the occasional park visitor. Such considerations have also been recognized by the Agency in past reviews, in which decisions to revise the O_3 secondary standard emphasized protection of Class I areas, which are areas such as national wilderness areas and national parks given special protections by the Congress (e.g., 73 FR 16496, March 27, 2008, "the Administrator concludes it is appropriate to revise the

[76] As stated in the 2015 decision notice: "both tree growth-related effects and visible foliar injury have the potential to be significant to the public welfare" (80 FR 65377, October 26, 2015); "O_3-induced visible foliar injury also has the potential to be significant to the public welfare through impacts in Class I and other similarly protected areas" (80 FR 65378, October 26, 2015); "[d]epending on the extent and severity, O_3-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 last review also stated that visible foliar injury "can impact public welfare" (Frey, 2014, p. 10).

secondary standard, in part, to provide increased protection against O_3-caused impairment to such protected vegetation and ecosystems").[77]

Further, as discussed in section 4.3.3.2 above, a study identified in the 2013 ISA that focused on visible foliar injury in west coast states observed that both percentage of USFS biosites with injury and average biosite index were higher for sites with average cumulative O_3 concentrations above 25 ppm-hrs in terms of SUM06 as compared to groups of sites with lower average cumulative exposure concentrations, with little difference apparent between the two lower exposure groups (80 FR 65395, October 26, 2015; Smith and Murphy, 2015; Campbell et al., 2007, Figures 27 and 28 and p. 30).[78] Similarly, a county-scale analysis of USFS biosite data in the 2007 Staff Paper (from earlier years than those analyzed in the 2015 review) indicated a somewhat smaller incidence of nonzero BI biosites in counties with O_3 exposures below a SUM06 metric of 15 ppm-hrs (or a fourth-high metric of 74 ppb) as compared to larger groups that also included sites with SUM06 values up to 25 ppm-hrs (or fourth-high metric up to 84 ppb) (U.S. EPA 2007, pp. 7-63 to 7-64; 80 FR 65395, October 26, 2015). This indication that the averaging time and form of the current standard, which emphasize peak concentrations through a short (8-hour) averaging time and a rare-occurrence form (annual fourth highest daily maximum), exert some control on the incidence of sites with visible foliar injury has a conceptual similarity to the finding of the most recent and extensive USFS data analysis that reductions in peak 1-hour concentrations have influenced the declining trend in visible foliar injury since 2002 (Smith, 2012).

Additional characterization of USFS biosite data in the current review is based on a more complete and further description of a dataset developed in the last review by combining USFS biosite records with W126 index estimates and a categorization of soil moisture (Appendix 4C). While recognizing limitations in the dataset[79] and considering the records for the normal or dry

[77] 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 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 2008 decision notice 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).

[78] 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).

[79] 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

soil moisture categories, for which there is somewhat better representation of W126 index levels above 13 ppm-hrs,[80] we note that the percentage of USFS records recording visible foliar injury (of any severity level) presents no clear trend across W126 index estimates below 17 ppm-hrs. Among records in the normal soil category, BI scores are noticeably increased in the highest W126 index bin (above 25 ppm-hrs) compared to the others. The percentages of records in the greater than 25 ppm-hrs bin that have BI scores above 15 ("moderate" and "severe" injury) and above 5 ("light," "moderate" and "severe" injury) are more than three times greater than percentages for these score levels in any of the lower W126 bins. Additionally, the average BI of 7.9 in the greater-then-25-ppm-hrs bin is more than three times the next highest bin average. The average BI in the next two lower W126 bins (which vary inversely with W126 index) are just slightly higher than average BIs for the rest of the bins, and the average BI for all bins at or below 25 ppm-hrs are well below 5. Among records in the dry soil moisture category, the two highest W126 bins (which together include the W126 index estimates above 19 ppm-hrs) exhibit the highest percentages of records with BI above 15 or above 5. The average score for all dry soil moisture records in each W126 index bin is highest for W126 above 25 ppm and somewhat higher for W126 above 17 ppm-hrs. Yet all three of those averages are below 5, indicating little or no injury.

Thus, the strongest conclusions that can be reached from the USFS dataset described in Appendix 4C are that the incidence/prevalence of sites with more severe injury (e.g., BI score above 15 or 5) is also lower at sites with W126 index values below 25 ppm-hrs than at sites with higher index values and that clear trends in such incidence/prevalence related to increasing W126 index levels are not evident across the lower W126 estimates. When scores characterized as "little injury" by the USFS classification scheme are included (i.e., when considering all scores above zero for the normal soil moisture category), there is a slight suggestion of increased incidence of records for the W126 index bins above 19 or 17 ppm-hrs, although for of the bins at or below 25 ppm-hrs, the incidence is less than 5%. As discussed in section 4.3.3.2 above, variability in the data across sites, and uncertainty with regard to the role of peak O_3 concentrations as an influence on occurrence of visible foliar injury separate from cumulative W126 index, lead to the conclusion that the currently available information does not support precise conclusions as to the severity and extent of such injury associated with the lower values of W126 index most common at USFS sites during the time of the dataset (2006-2010). Notwithstanding this, records categorized as normal soil moisture indicate there to be an

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).

[80] 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).

appreciable difference in severity of injury between records with W126 index estimates above 25 ppm-hrs and those with estimates at or below 25 ppm-hrs (e.g., Appendix 4C, Figures 4C-5 and 4C-6 and Table 4C-5). The records categorized as dry soil moisture do not indicate such a clear pattern. The records categorized as wet soil moisture are much too limited (and variability) for W126 index estimates above 13 ppm-hrs to support a conclusion (Appendix 4C). Thus, we conclude, based primarily on the records categorized as having normal soil moisture, that under conditions that maintain W126 index values below 25 ppm-hrs a reduced severity (average BI score below 5) and incidence of visible foliar injury, as quantified by biosite index scores, would be expected.

As discussed in section 4.3.3.2 above, consistent relationships of injury extent and severity with vegetation exposure circumstances have not been developed. The current evidence indicates a role for cumulative seasonal concentration-weighted metrics such as SUM06 and W126 indices, while also indicating an importance of the occurrence of particularly high concentrations (e.g., hours above a concentration such as 100 ppb). Thus, in making judgements regarding air quality conditions of concern with regard to impacts associated with incidence and severity of visible foliar injury, it is appropriate to consider both cumulative concentration-weighted seasonal exposures and the occurrence of peak concentrations. In this context, it is appropriate to recognize the control of peak concentrations exerted by the form and averaging time of the current standard. For example, as noted in chapter 2, daily maximum 1-hour, as well as 8-hour average O_3 concentrations have declined over the past 15 years, a period in which there have been two revisions of the level of the secondary standard, each providing greater stringency, while retaining the same averaging time and form as the current standard (e.g., Figures 2-8, 2-9 and 2-14).

Further, we note that judgments related to the extent of public welfare impacts of visible foliar injury depend on the severity and extent of the injury, as well as the location where the effects occur and the associated intended use. As noted in section 4.3.2 above, aesthetic value and outdoor recreation depend, at least in part, on the perceived scenic beauty of the environment. Accordingly, depending on its spatial extent and severity, visible foliar injury in national parks and wilderness areas can adversely impact the aesthetic experience for both outdoor enthusiasts and the occasional park visitor. Beyond the limitations associated with the evidence for descriptive quantitative relationships for O_3 concentrations and visible foliar injury, we further face a paucity of information clearly relating differing severity and prevalence of injury to conditions in natural areas that would reasonably be concluded to impact public use and enjoyment in a way that might suggest adversity to the public welfare. The available information does not yet address or describe the relationships expected to exist between some level of severity and/or extent of location affected and scenic or aesthetic values (e.g., reflective of visitor

enjoyment and likelihood of frequenting such areas). However, it might reasonably be expected that in cases of widespread and relatively more severe injury during the growing season (particularly when sustained across multiple years, and accompanied by obvious impacts on the plant canopy), O_3-induced visible foliar injury could adversely impact the public welfare in scenic and/or recreational areas, particularly in parks and other areas with special protection, such as Class I areas. In summary, the available evidence does not include characterization of USFS biosite scores with regard to public perception and potential impacts on public enjoyment. Nor does it address this in combination with information on whether air quality conditions in sites with scores of a particular severity level do or do not meet the current standard.

In consideration of all of the above, we recognize the appreciable limitations of the current information touched on above with regard to providing a foundation for judgments on public welfare protection objectives specific to visible foliar injury. In light of such limitations and in light of the above discussion, we then consider what the available information indicates with regard to potential for adverse effects to the public welfare related to visible foliar injury under air quality conditions allowed by the current standard, which was established with a focus on protecting against RBL as a surrogate/proxy for the broad array of vegetation-related effects. As recognized in section 4.3.1 above, while the evidence continues to show a consistent association between the occurrence of visible injury and ozone, "visible foliar injury is not always a reliable indicator of other negative effects on vegetation" (ISA, Appendix 8, section 8.2). Based on the USFS biosite data, the conditions associated with visible foliar injury in locations with sensitive species appear to relate to peak concentration as well as sustained exposure to higher concentrations over the growing season, such that cumulative exposure metrics may not well or completely describe or predict the occurrence and severity of injury.

- **What does the currently available information indicate for considering potential public welfare protection from O_3-related climate effects?**

In considering the currently available information for the effects of the global abundance of O_3 in the troposphere on radiative forcing, and temperature, precipitation and related climate variables, we note as an initial matter that, as summarized in section 4.3.3 above, there are limitations and uncertainties in the associated evidence bases with regard to assessing potential for occurrence of climate-related effects as a result of varying O_3 concentrations in ambient air of locations in the U.S. The current evidence is limited with regard to support for such quantitative analyses that might inform considerations related to the current standard. For example, as stated in the ISA, "[c]urrent limitations in climate modeling tools, variation across models, and the need for more comprehensive observational data on these effects represent sources of uncertainty in quantifying the precise magnitude of climate responses to ozone changes, particularly at regional scales" (ISA, section 9.3.1). These are "in addition to the key sources of uncertainty in

quantifying ozone RF changes, such as emissions over the time period of interest and baseline ozone concentrations during preindustrial times" (ISA, section IS.9.3.1). Together such uncertainties limit development of quantitative estimates of climate-related effects in response to earth surface O_3 concentrations at the regional scale, such as in the U.S. While these complexities inhibit our ability to consider tropospheric O_3 effects, such as radiative forcing, we note that our consideration of O_3 growth-related impacts on trees inherently encompasses consideration of the potential for O_3 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 O_3-related effects on growth would be expected to also limit reductions in carbon sequestration, a process that can reduce the tropospheric abundance of CO_2, the greenhouse gas ranked highest in importance (section 4.3.3.3 above; ISA, section 9.1.1).

4.5.1.3 Public Welfare Implications of Air Quality under the Current Standard

Our consideration of the scientific evidence available in the current review, as at the time of the last review, is informed by results from a quantitative analysis of estimated exposure and associated risk. An overarching consideration is whether the current exposure/risk and air quality information calls into question the adequacy of protection provided by the now-current standard. As in our consideration of the evidence above, we have organized the discussion regarding the information related to exposures and potential risks around a key question to assist us in considering the quantitative analyses of air quality at U.S. locations nationwide, particularly including those in Class I areas. In so doing, we consider first analyses particular to cumulative O_3 exposures, in terms of the W126 index, given its established relationship with growth-related effects and specifically RBL as the identified proxy or surrogate for the full array of such effects.

To understand the cumulative O_3 exposures likely occurring under the current standard, we consider the air quality analyses summarized in section 4.4 above and presented in detail in Appendix 4D. These air quality analyses of monitoring data at sites across the U.S., including sites in Class I areas, document seasonal cumulative concentration-weighted exposures occurring when the current standard is met. In so doing, they indicate that, as described in section 4.4.2 above, with very few exceptions (one in the most recent 3-year period), the seasonal W126 index at sites nationwide (including those in Class I areas), as assessed by the 3-year average, are at or below 17 ppm-hrs when the current standard is met. Further, such exposures are generally well below 17 ppm-hrs across most of the U.S. The overall pattern for single-year seasonal W126 index values at monitors meeting the current standard in the recent period is generally similar, with about a dozen of the 849 sites nationwide having a single-year W126 index above 19 ppm-

hrs (and under two dozen above 17 ppm-hrs).[81] The frequency of higher single-year W126 index values during periods when the current standard is met is much lower for the Class I area monitors. During the most recent three years, when the average seasonal W126 index is at or below 17 ppm-hrs in all Class I areas meeting the current standard, there were just three single-year W126 index values above 17 ppm-hrs and none above 19 ppm-hrs (Appendix 4D, Table 4D-16).[82]

Combining this information regarding likely W126-based exposure levels with the established E-R functions for 11 tree seedling species indicates that based on monitoring data for locations meeting the current standard during the most recent design period, the median species RBL for tree seedlings is at or below 5.3% based on the 3-year average W126, with very few exceptions, with the highest estimates occurring in areas not near or within Class I areas. Looking at the data over a longer time period (2000-2018) confirms this general pattern for the bulk of the data, with some infrequent higher occurrences, and virtually all RBL estimates below 6%.[83] Further, given the variability and uncertainty associated with the data underlying the E-R functions (as discussed in section 4.5.1.2 above), the few higher single-year occurrences are reasonably considered to be of less significance than 3-year average values.

With regard to visible foliar injury, as discussed in section 4.3.3.2 above, the evidence is somewhat limited and unclear with regard to the metric and quantitative approach that well describes a relationship between incidence or severity of injury in U.S. forests across a broad range of air quality conditions. As indicated in the Appendix 4C presentation of the dataset developed from USFS biosite records, W126 index estimates and categorizations of soil moisture, while most of the records (more than 95%) in the dataset are for W126 exposure index estimates below 17 ppm-hrs - and there is appreciable variability in incidence of records with nonzero BI scores, and more importantly with records above 5 or 15 (scores associated with injury considered greater than “a little” by the USFS scheme), the increased incidence of such scores appears most consistently with higher W126 estimates. The incidence is greatest in the bin for the highest estimated exposures, i.e., W126 index above 25 ppm-hrs, which are not seen to

[81] These highest W126 index values occur in the South West and West regions in which there are nearly 150 monitor locations meeting the current standard (Figure 4-6; Appendix 4D, Figure 4D-5, Table 4D-1).

[82] Across the full 19-year dataset for Class I area monitors meeting the current standard (58 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).

[83] Although potential for effects on crop yield was not given particular emphasis in the last 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%.

occur in Class I area monitoring sites that meet the current standard (Appendix 4C, section 4C.3). Further, as discussed in section 4.3.3.2 above, the evidence indicates a role for occurrences of higher concentrations, such as above 100 ppb, the frequency of which has declined in U.S. monitoring sites over the past 15 years. The analyses of hourly concentrations in section 2A.2 of Appendix 2A demonstrates the substantial control of peak 1-hour concentrations exerted by the current standard. For example, in three different datasets of monitoring data since 2000, the average number of observations at or above 100 ppb per site-design value period is well below one for when sites were meeting the current standard, and well above at sites when not meeting the current standard (Appendix 2A, Tables 2A-2, 2A-3 and 2A-4). Thus, although the current information does not establish a metric or combination of metrics that well describes the relationship between occurrence and severity of visible foliar injury across a broad range of O_3 concentration patterns from those more common in the past to those in areas recently meeting today's standard, the current evidence and currently available air quality information indicates that the exposure conditions occurring at sites with air quality meeting the current standard are not those that might reasonably be concluded to elicit the occurrence of significant foliar injury (with regard to severity and extent).

- **Are such exposures (in terms of W126 index) that occur in areas that meet the current standard indicative of welfare effects reasonably judged important from a public welfare perspective? What are important associated uncertainties?**

Given the findings summarized in section 4.4 above regarding W126 index values in areas where the current standard is met, we reflect on the potential public welfare significance of vegetation-related effects that may be associated with such exposures. This consideration is important to informing the Administrator's judgment on the secondary standard, which is not meant to protect against all known or anticipated O_3-related welfare effects, but rather those that are judged to be adverse to the public welfare (as noted in section 4.3.2 above). Accordingly, for the purposes of informing that judgment, we consider here the exposures indicated to occur under conditions that meet the current standard, the associated potential for effects and the potential public welfare implications.

As an initial matter, we recognize the increased significance to the public welfare of effects in areas that have been accorded special protection, such as Class I areas, while noting some general similarities of the exposure estimates in Class I areas for periods when the current standard was met to such estimates at monitoring sites in other areas, as documented in the larger air quality data analysis. Across both datasets, and extending back 19 years, the cumulative exposure estimates, averaged over the design value period, for these air quality conditions were virtually all at or below 17 ppm-hrs, with most of the W126 index values below 13 ppm-hrs (Appendix 4D, Table 4D-9), corresponding to median RBL estimates of 3.8% or less (based on

the established tree seedling E-R relationships detailed in Appendix 4A). We additionally note that single-year W126 index values in Class I areas over the 19-year dataset evaluated were generally at or below 19 ppm-hrs, particularly in the more recent years (Appendix 4D, section 4D.3.2.3).

Regarding the effects associated with the exposures commonly occurring, we consider first the categories of effects for which the quantitative information related to exposure and associated effects is most well developed. As in the last review, these are effects on plant growth. Based on the median of RBL estimates derived from the established E-R functions for 11 tree species seedlings, W126 index values at or below 17 ppm-hrs correspond to median species tree seedling RBL estimates at or below 5.3% (Appendix 4A, Table 4A-5). Judgments in the last review (in the context of the framework considered in section 4.5.1.2 above) concluded isolated rare occurrences of exposures for which median RBL estimates might be at or just above 6% to not be indicative of conditions adverse to the public welfare, particularly considering the variability in the array of environmental factors that can influence O_3 effects in different systems, and the uncertainties associated with estimates of effects in the natural environment.

In the last review, the Administrator focused on cumulative exposure estimates derived as the average W126 index over the 3-year design value period, concluding variations of single-year W126 index from the average to be of little significance. This focus generally reflected the judgment that estimates based on the average adequately, and appropriately reflected the precision of current understanding of O_3-related growth reductions, given the various limitations and uncertainties in such predictions. Additional analyses have been explored in the current review to further examine this issue, as summarized in section 4.5.1.2 above. The current air quality data indicates single-year W126 index values generally to vary by less than 5 ppm-hrs from the 3-year average when the 3-year average is below 20 ppm-hrs (which is the case for locations meeting the current standard). With such variation, year-to-year differences in tree growth responding to each year's seasonal exposure from estimated response based on the 3-year average of those seasonal exposures would, given the offsetting impacts of seasonal exposures above and below the average, reasonably be expected to generally be small over tree lifetimes. Additionally, we have also further considered the experimental data underlying the E-R functions for estimating RBL, particularly those pertaining to cumulative exposures on the order of 17 ppm-hrs and informing estimates of multiyear impacts. We note limitations in the evidence base in these regards, as discussed further in section 4.5.1.2 above, that contribute to imprecision or inexactitude to estimates of growth impacts associated with multi-year exposures in this range. Further, the information newly available in the current review does not appreciably address these limitations and uncertainties to improve the certainty or precision in RBL estimates for such exposures.

With regard to visible foliar injury, as noted in section 4.3.3.2 above, the dataset based on USFS biomonitoring data that was developed in the last review (see Appendix 4C) does not provide for a clear predictive relationship between O_3, in terms of W126 index, and incidence of injury or magnitude of injury score. It additionally indicates variability in the incidence of nonzero injury scores (or of scores of relatively greater severity), particularly for records with generally lower W126 index estimates (e.g., below 19 ppm-hrs). Further, the sample size for sites with wet soil moisture conditions is quite limited for sites with W126 index above 13 ppm-hrs, limiting conclusions for those situations. Further, as discussed in sections 4.3.3.2 and 4.5.1.2 above, a quantitative description of the relationship between O_3 concentrations and visible foliar injury extent or incidence, as well as severity, that would support estimation of injury under varying air quality and environmental conditions (e.g., moisture), most particularly for locations that meet the current standard is not yet established in the evidence.

In light of the potential role of peak O_3 concentrations (e.g., hourly concentrations at or above 100 ppb) as an influence on visible foliar injury occurrence and severity, it is of interest to take note of analyses in Chapter 2 and Appendix 2A. These indicate that the magnitude of daily maximum 1-hour concentrations has declined appreciably since 2000. For example, the median annual 2nd highest MDA1 concentration across U.S. monitoring sites has declined from 100 to 80 ppb (Figure 2-17 above). The analysis in Appendix 2A of three recent design value periods (covering 2014 through 2018) and three periods more than ten years prior (covering 2000 through 2004) show that for all sites with DVs that would meet the current standard during these periods, there is less than one observation per site, on average, at or above 100 ppb. There are roughly 40 times more such observations per site, on average, for sites with DVs that would not meet the current standard (Appendix 2A, section 2A.2). These data indicate that the current standard provides appreciable control of peak 1-hour concentrations, and thus, to the extent that such peak concentrations play a role in the occurrence and severity of visible foliar injury, the current standard also provides appreciable control of these.

Additionally, as discussed in section 4.3.2 above, the public welfare implications associated with visible foliar injury (when considered as an effect separate from effects on plant physiology) relate largely to effects on scenic and aesthetic values. The available information does not yet address or describe the relationships expected to exist for some level of visible foliar injury severity (below that at which broader physiological effects on plant growth and survival might also be expected) and/or extent of location or site injury (e.g., BI) scores with values held by the public and associated impacts on public uses of the locations.[84] As discussed in section

[84] 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).

4.3.2 above, this gap limits our ability to identify air quality conditions that might be expected to provide a specific level of protection from public welfare effects of this endpoint (e.g., separate from effects that might relate to plant growth and reproduction under conditions where foliar injury may also be severe). Additionally, as recognized in the sections above, 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). Thus, key considerations of this endpoint in past reviews have related to qualitative consideration of potential impacts related to the plant's aesthetic value in protected forested areas and the somewhat general, nonspecific judgment that a more restrictive standard is likely to provide increased protection. Nevertheless, while minor spotting on a few leaves of a plant may easily be concluded to be of little public welfare significance, it is reasonable to conclude that cases of widespread and relatively severe injury during the growing season (particularly when sustained across multiple years, and accompanied by obvious impacts on the plant canopy) would likely impact the public welfare in scenic and/or recreational areas, particularly in areas with special protection, such as Class I areas.

The currently available evidence, as discussed above, as well as in sections 4.3.3.2 and 4.5.1.2 (with consideration of presentations in Appendix 4C and air quality analyses in Appendices 2A and 4D) do not indicate that a situation of widespread and relatively severe visible foliar injury is likely associated with air quality that meets the current standard. While the USFS biosite dataset includes appreciable variability in biosite index scores, including the occurrence of scores above 15 in records with W126 index estimates in the lowest bin, it does not demonstrate a clear trend in biosite index score across the lower W126 index bins. That notwithstanding, as noted in section 4.5.1.2 above, records with W126 index estimates below 25 ppm-hrs appear to have BI scores below 5, on average, with fewer than 10% of records having a BI score above 15 or 5. The latter compares to some 20 to 40% of records with higher W126 index estimates having scores above 5 (in the USFS dataset presented in Appendix 4C, see Table 4C-6). In this context, we note that the current air quality analyses indicate that virtually all 3-year average, and single-year, W126 index values at locations meeting the current standard are at or below 25 ppm-hr. Further, the average number of observations of 1-hour concentrations at or above 100 ppb per site and design value period are well below one during periods when the current standard is met. Thus, while the current evidence is limited for the purposes of identifying public welfare protection objectives related to visible foliar injury in terms of specific air quality metrics, the current information indicates that the occurrence of injury categorized as more severe than "little" by the USFS categorization (i.e., a BI scores above 5 or above 15) would be expected to be infrequent in areas that meet the current standard. Based on the USFS

dataset presentations as well as the air quality analyses of W126 index values and frequency of 1-hour observations at or above 100 ppb, the prevalence of injury scores categorized as severe, which, depending on spatial extent, might reasonably be concluded to have potential to be adverse to the public welfare would be expected to be appreciably more likely for air quality conditions unlikely to meet the current standard.

With regard to other vegetation-related effects, including those at the ecosystem scale, such as alteration in community composition or reduced productivity in terrestrial ecosystems, as recognized in section 4.5.1.1, the available evidence is not clear with regard to the risk of such impacts (and their magnitude or severity) associated with the environmental O_3 exposures estimated to occur under air quality conditions meeting the current standard (e.g., W126 index at or below 17 ppm-hrs). In considering effects on crop yield, the air quality analyses at monitoring locations that meet the current standard indicate estimates of RYL for such conditions to be at and below 5.1%, based on the median estimate derived from the established E-R functions for 10 crops (Appendix 4A, Table 4A-5). We additionally recognize there to be complexities involved in interpreting the significance of such small estimates in light of the factors also recognized in the last review. These included the extensive management of crops in agricultural areas that may to some degree mitigate potential O_3-related effects, as well as the use of variable management practices to achieve optimal yields, while taking into consideration various environmental conditions. We also recognize, as was recognized in the last review, that changes in yield of commercial crops and commercial commodities may affect producers and consumers differently, further complicating the question of assessing overall public welfare impacts for such RYL estimates (80 FR 65405, October 26, 2015).

4.5.2 CASAC advice

In our consideration of the adequacy of the current secondary O_3 standard, in addition to the evidence- and air quality/exposure/risk-based information discussed above, we have also considered the advice and recommendations of the CASAC, based on their review of the ISA and the earlier draft of this PA, as well as comments from the public on the earlier draft of this PA. A limited number of public comments have been received in this review to date, including comments focused on the draft IRP or draft PA. Of the commenters that addressed adequacy of the current secondary O_3 standard, most expressed agreement with staff conclusions in the draft PA, while some expressed the view that the standard should be revised to a W126-based form based on advice from the previous CASAC in the last review. The CASAC provided its advice regarding the current secondary standard in the context of its review of the draft PA (Cox, 2020). In so doing, the CASAC concurred with the PA conclusions, stating 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). The CASAC additionally stated that it "commends the EPA for the thorough discussion and rationale for the secondary standard" while also providing comments particular to the consideration of climate and growth-related effects (Cox, 2020, pp. 2, 22).

With regard to O_3 effects on climate, the CASAC recommended quantitative uncertainty and variability analyses, with associated discussion (Cox, 2020, pp. 2, 22).[85] With regard to growth-related effects and consideration of the evidence in quantitative exposure analyses, it stated that the W126 index, "appears reasonable and scientifically sound" "particularly related to growth effects" (Cox, 2020, p 16). Additionally, with regard to the Administrator in the last review expressing greater confidence in judgements related to public welfare impacts based on seasonal W126 Index estimated by a three-year average and accordingly relying on that metric the CASAC expressed the view that it "appears of reasonable thought and scientifically sound" (Cox, 2020, p. 19). Further, the CASAC stated that "RBL appears to be appropriately considered as a surrogate for an array of adverse welfare effects and based on consideration of ecosystem services and potential for impact to the public as well as conceptual relationships between vegetation growth effects and ecosystem scale effects" and that it agrees "that biomass loss, as reported in RBL, is a scientifically-sound surrogate of a variety of adverse effects that could be exerted to public welfare," concurring that this approach is not called into question by the current evidence which continues to support "the use of tree seedling RBL as a proxy for the broader array of vegetation related effects, most particularly those related to growth that could be impacted by ozone" (Cox, 2020, p 21). The CASAC additionally concurred that the strategy of a secondary standard that limits 3-yr average W126 index values somewhat below those associated with a 6% RBL in the median species is "still scientifically reasonable" and that, accordingly, a W126 index target value of 17 ppm-hrs for generally restricting cumulative exposures "is still effective in particularly protecting the public welfare in light of vegetation impacts from ozone" (Cox, 2020, p 21).

With regard to the court's remand of the 2015 secondary standard to the EPA for further justification or reconsideration ("particularly in relation to its decision to focus on a 3-year average for consideration of the cumulative exposure, in terms of W126, identified as providing requisite public welfare protection, and its decision to not identify a specific level of air quality related to visible foliar injury"), while the CASAC stated that it was not clear whether the draft

[85] As recognized in the ISA, [c] and "[c]urrent limitations in climate modeling tools, variation across models, and the need for more comprehensive observational data on these effects represent sources of uncertainty in quantifying the precise magnitude of climate responses to ozone changes, particularly at regional scales" (ISA, section IS.6.2.2, Appendix 9, section 9.3.3, p. 9-22). As noted in section 4.3.3.3 above, these complexities impede our ability to consider specific O3 concentrations in the U.S. with regard to specific magnitudes of impact on radiative forcing and subsequent climate effects.

PA had fully addressed this concern (Cox, 2020, p. 21), it described there to be a solid scientific foundation for the current secondary standard and also commented on areas related to the remand.. With regard to the focus on the 3-year index, in addition to the comments summarized above, the CASAC concluded that the EPA Administrator's focus on 3-year average and her judgments in doing so "appears of reasonable thought and scientifically sound" (Cox, 2020, p 19). Further, while recognizing the existence of established E-R functions that relate cumulative seasonal exposure of varying magnitudes to various incremental reductions in expected tree seedling growth (in terms of RBL) and in expected crop yield, the CASAC letter also noted that while decades of research also recognizes visible foliar injury as an effect of O_3, "uncertainties continue to hamper efforts to quantitatively characterize the relationship of its occurrence and relative severity with ozone exposures" (Cox, 2020, p 20). In summary, the CASAC 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" (Cox, 2020, p. 22).

4.5.3 Conclusions

This section describes conclusions for the Administrator's consideration in this review of the current secondary O_3 standard. These conclusions are based on consideration of the assessment and integrative synthesis of the evidence (as summarized in the ISA, and the 2013 ISA and AQCDs from prior reviews), and the information on quantitative exposure and air quality analyses summarized above, as well as CASAC advice and public comment on the draft PA. Taking into consideration the discussions above in this chapter, this section addresses the following overarching policy question.

- **Does the currently available scientific evidence and air quality and exposure analyses support or call into question the adequacy of the protection afforded by the current secondary O_3 standard?**

In considering this question, we first recognize what the CAA specifies with regard to protection to be provided by the secondary standard. Under section 109(b)(2) of the CAA, a secondary standard must "specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air." Accordingly, as noted in section 4.3.2 above, the secondary standard is meant to protect against O_3-related welfare effects that are judged to be adverse to the public welfare (78 FR 8312, January 15, 2013; see also 73 FR 16496, March 27, 2008). Thus, our consideration of the currently available information regarding welfare effects of O_3 is in this context, while recognizing that the level of protection from known or anticipated adverse effects

to public welfare that is requisite for the secondary standard is a public welfare policy judgment to be made by the Administrator.

As is the case in NAAQS reviews in general, the extent to which the protection provided by the current secondary O_3 standard is judged to be adequate will depend on a variety of factors, including science policy judgments and public welfare policy judgments. These factors include public welfare policy judgments concerning the appropriate benchmarks on which to place weight, as well as judgments on the public welfare significance of the effects that have been observed at the exposures evaluated in the welfare effects evidence. The factors relevant to judging the adequacy of the standard also include the interpretation of, and decisions as to the weight to place on, different aspects of the quantitative analyses of air quality and cumulative O_3 exposure and any associated uncertainties. Thus, we recognize that the Administrator's conclusions regarding the adequacy of the current standard will depend in part on public welfare policy judgments, science policy judgments regarding aspects of the evidence and exposure/risk estimates, as well as judgments about the level of public welfare protection that is requisite under the Clean Air Act.

Our response to the overarching question above takes into consideration the discussions that address the specific policy-relevant questions in prior sections of this document and the approach described in section 4.2 that builds on the approach from the last review, with some further attention to the issues highlighted in the court's remand. We focus first on consideration of the evidence, including that newly available in this review, and the extent to which it alters key conclusions supporting the current standard. We then turn to consideration of the quantitative analyses, including associated limitations and uncertainties, and the extent to which they indicate differing conclusions regarding level of protection indicated to be provided by the current standard from adverse effects. We additionally consider the key aspects of the evidence and air quality/exposure information emphasized in establishing the now-current standard, and the associated public welfare policy judgments and judgments about inherent uncertainties that are integral to decisions on the adequacy of the current secondary O_3 standard.

In considering the currently available evidence, we recognize the longstanding evidence base of the vegetation-related effects of O_3, augmented in some aspects since the last review. Consistent with the evidence in the last review, the currently available evidence describes an array of O_3 effects on vegetation and related ecosystem effects, as well as the role of tropospheric O_3 in radiative forcing and subsequent effects on temperature, precipitation and related climate variables. The current evidence base, including the wealth of longstanding evidence, supports the conclusion of causal relationships between O_3 and visible foliar injury, reduced plant growth and reproduction, as well as reduced yield and quality of agricultural crops, reduced productivity in terrestrial ecosystems, alteration of terrestrial community composition,

and alteration of belowground biogeochemical cycles (ISA, section IS.5). This current evidence base also supports likely causal relationships for O_3 with alteration of terrestrial ecosystem water cycling and reduced carbon sequestration in terrestrial ecosystems, and also with increased tree mortality (ISA, section IS.5). Evidence available in this review also supports Agency conclusions on two additional plant-related effects: the body of evidence is determined to be sufficient to infer a likely causal relationship between O_3 exposure and alteration of plant-insect signaling, and to infer a likely causal relationship between O_3 exposure and altered insect herbivore growth and reproduction (ISA, section IS.5).

We additionally recognize that uncertainties in categories of effects newly identified in this review, such as alteration of plant-insect signaling and insect herbivore growth and reproduction, limit our consideration of the protection that might be provided by the current standard against these effects. Depending on a number of factors, such effects may have a potential for adverse effects to the public welfare, e.g., given the role of plant-insect signaling in such important ecological processes as pollination and seed dispersal, as well as, natural plant defenses against predation and parasitism (as discussed in section 4.3.2 above). Uncertainties in the current evidence, however, preclude a full understanding of such effects, the air quality conditions that might elicit them, the potential for impacts in a natural ecosystem and, consequently, the potential for such impacts under air quality conditions associated with meeting the current standard. As one example of such uncertainties, although there are multiple statistically significant measures of O_3 effects on insect herbivore growth and reproductive endpoints, there is no clear trend in the directionality of response for most endpoints studied. Additionally, the characterization of effects on plant VPSCs in natural ecosystems is still an emerging area of research that includes knowledge gaps with regard to the role of O_3, including an understanding of the air quality conditions and O_3 concentrations that would be expected to cause effects in the natural environment, and the magnitude or severity of such effects.

As was the case in the last review, a category of effects for which the evidence supports quantitative description of relationships between air quality conditions and response is plant growth or yield. The evidence base continues to indicate growth-related effects as sensitive welfare effects, with the potential for ecosystem-scale ramifications. For this category of effects, there are established E-R functions that relate cumulative seasonal exposure of varying magnitudes to various incremental reductions in expected tree seedling growth (in terms of RBL) and in expected crop yield (in terms of RYL). Many decades of research also recognize visible foliar injury as an effect of O_3, although uncertainties continue to hamper efforts to quantitatively characterize the relationship of its occurrence and relative severity with O_3 exposures. The evidence for these categories of O_3 effects is discussed further below.

Before focusing further on the key vegetation-related effects identified above, we first recognize the strong evidence documenting tropospheric O_3 as a greenhouse gas causally related to radiative forcing, and likely causally related to subsequent effects on variables such as temperature and precipitation. In so doing, however, we take note of the limitations and uncertainties in the evidence base that affect characterization of the extent of any relationships between O_3 concentrations in ambient air in the U.S. and climate-related effects. Accordingly, we recognize, as was recognized at the time of the last review, the lack of important quantitative tools with which to consider such effects in this context (as summarized in sections 4.3.3.3 and 4.3.4 above).[86] Notwithstanding consideration of these effects, a focus in this review, as in the last, 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.

Turning next to consideration of visible foliar injury, the available information has been examined and analyzed as to what it indicates and supports with regard to adequacy of protection provided by the current standard (e.g., as discussed in section 4.5.1 above). Visible foliar injury is an effect for which an association with O_3 in ambient air is well documented, and the public welfare significance of visible foliar injury of vegetation in areas not closely managed for harvest, particularly specially protected natural areas, has generally been considered in the context of potential effects on aesthetic and recreational values, such as the aesthetic value of scenic vistas in protected natural areas such as national parks and wilderness areas (e.g., 73 FR 16496, March 27, 2008). Accordingly, depending on its severity and spatial extent, as well as the location(s) and the associated intended use, its effects on the physical appearance of the plant have the potential to be significant to the public welfare. For example, cases of widespread and relatively severe injury during the growing season (particularly when sustained across multiple years and accompanied by obvious impacts on the plant canopy) might reasonably be expected to have the potential to adversely impact the public welfare in scenic and/or recreational areas, particularly in areas with special protection, such as Class I areas. Thus, we consider the currently available information with regard to the potential for such an occurrence with air quality conditions that meet the current standard. In so doing, we recognize that important uncertainties remain in the understanding of the O_3 exposure conditions that will elicit visible

[86] With regard to radiative forcing and effects on temperature, precipitation, and related climate variables, while additional characterizations have been completed since the last review, uncertainties and limitations in the evidence that were also recognized in the last review remain. As summarized in sections 4.3.3.3 and 4.3.4 above, these affect our ability to make a quantitative characterization of the magnitude of climate response to changes in O_3 concentrations in ambient air at regional (vs global) scales.

foliar injury (and its severity), and particularly in light of the other environmental variables that influence its occurrence. For example, as discussed in section 4.5.1.2 above, while analyses of USFS data for foliar injury often consider O_3 concentrations in terms of a cumulative exposure metric, multiple studies also have indicated a role for an additional metric related to the occurrence of days with relatively high concentrations (e.g., number of days with a 1-hour concentration at or above 100 ppb), although, there has not yet been extensive work done to confirm the specific peak concentration that would be appropriate for such a metric. With regard to an implied importance of peak or elevated short-term (e.g., 1-hour) concentrations, the dataset of BI scores at USFS biosites (sites with O_3-sensitive vegetation assessed for visible foliar injury) analyzed in Appendix 4C indicates variability in incidence of BI scores indicative of moderate or greater severity injury across the bins for W126 index values most common in areas where the standard is met (Appendix 4C, section 4.C.3; Appendix 4D, Figures 4D-3 and 4D-4, and section 4D.3.2.3). The incidence of nonzero scores, and of relatively higher scores[87] appears to markedly increase only with W126 index values above 25 ppm-hrs, a magnitude not seen to occur at monitoring locations (including in or near Class I areas) where the current standard is met (Appendix 4C, section 4C.3; Appendix 4D, section 4D.3.2.3).

Publications related to the evidence base for the USFS biosite monitoring program document reductions in the incidence of the higher BI scores over the 16-year period of the program (1994 through 2010), especially after 2002, leading to researcher conclusions of a "declining risk of probable impact" on the monitored forests over this period (e.g., Smith, 2012). These reductions parallel the O_3 concentration trend information nationwide that show clear reductions in cumulative seasonal exposures, as well as in peak O_3 concentrations such as the annual fourth highest daily maximum 8-hour concentration, from 2000 through 2018 (Appendix 4D, Figure 4D-9 and Figure 2-11 above). The foliar injury reductions also parallel reductions in the occurrence of 1-hour concentrations above 100 ppb (Appendix 2A, Tables 2A-2 to 2A-4). Thus, the extensive air quality evidence of trends across the past nearly 20 years indicate reductions in peak concentrations that some studies have suggested to be influential in the severity of visible foliar injury, as discussed in section 4.5.1 above.

Further, we note the paucity of established approaches for interpreting specific levels of severity and extent of foliar injury in protected forests with regard to impacts on public welfare effects, e.g., related to recreational services.[88] As discussed in sections 4.3.2 and 4.5.1 above, injury to whole stands of trees of a severity apparent to the casual observer (e.g., when viewed as

[87] In the USFS categorization, scores from zero to just below 5 are described as "little or no foliar injury."

[88] 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).

a whole from a distance) would reasonably be expected to affect recreational values. Current information, however, particularly in locations meeting the current standard or with W126 index estimates likely to occur under the current standard does not indicate a significant extent and degree of injury (e.g., based on BI scores analyzed in Appendix 4C) or specific impacts on recreational or related services for areas, such as wilderness areas or national parks. Thus, the evidence does not appear to suggest public welfare significance for BI scores reported at sites likely to meet the current standard.

We additionally take note of the recognition by the CASAC that "uncertainties continue to hamper efforts to quantitatively characterize the relationship of [visible foliar injury] occurrence and relative severity with ozone exposures" (Cox, 2020, p. 20 of the Response to Charge), and of the CASAC advice, which concurred with the draft PA preliminary conclusion "that the available evidence does not reasonably call into question the adequacy of the current secondary ozone standard" (Cox, 2020, p. 1). Based on all of the above considerations it appears reasonable to conclude that the current evidence and quantitative exposure information for visible foliar injury does not call into question the adequacy of protection provided by the current standard.

Uncertainties additionally affect our understanding of the extent to which RYL estimates on the order of 5% (or less), based on the set of 10 established E-R functions, would be expected to be of public welfare significance, given the extensive management of such crops, and other factors summarized in sections 4.3.2 and 4.5.1.3 above. Further, we recognize uncertainties in the details and quantitative aspects of relationships between plant-level effects such as growth and reproduction, and ecosystem impacts, the occurrence of which are influenced by many other ecosystem characteristics and processes. These examples illustrate the role of public welfare policy judgments, both with regard to the extent of protection that is requisite and concerning the weighing of uncertainties and limitations of the underlying evidence base and associated quantitative analyses. Such judgments will inform the Administrator's decision in the current review, as they did in the setting of the current standard in 2015, as summarized in section 4.1.2 above.

We recognize that public welfare policy judgments play an important role in each review of a secondary standard, just as public health policy judgments have important roles in primary standard reviews. One type of public welfare policy judgment focuses on how to consider the nature and magnitude of the array of uncertainties that are inherent in the scientific evidence and analyses. These judgments are traditionally made with a recognition that current understanding of the relationships between the presence of a pollutant in ambient air and associated welfare effects is based on a broad body of information encompassing not only more established aspects of the evidence but also aspects in which there may be substantial uncertainty. This may be true

even of the most robust aspect of the evidence base. In the case of the secondary O_3 standard review, as an example, we recognize increased uncertainty, and associated imprecision, at lower cumulative exposures in application of the established and well-founded E-R functions, and in the current understanding of aspects of relationships of such estimated effects with larger-scale impacts, such as those on populations, communities and ecosystems, as summarized in sections 4.5.1.3 and 4.3.4 above.

The category of effects for which the evidence is most certain with regard to quantitative functions describing relationships between O_3 in ambient air and response continues to be reduced plant growth or yield. The evidence base includes established E-R functions for seedlings of 11 tree species that relate cumulative seasonal exposure of varying magnitudes to various incremental reductions in expected tree seedling growth (in terms of RBL) and in expected crop yield. These functions are well established and have been recognized across multiple O_3 NAAQS reviews. Uncertainties related to use of the RBL estimates include the limited information regarding the extent to which they reflect growth impacts in mature trees, and the fact that the 11 species represent a very small portion of the tree species across the U.S. While recognizing these and other uncertainties, RBL estimates based on the median of the 11 species were used as a surrogate in the last review for comparable information on other species and lifestages, as well as a proxy or surrogate for other vegetation-related effects, including larger-scale effects. Use of this approach continues to appear to be a reasonable judgment in this review. More specifically, the currently available information continues to support (and does not call into question) the use of RBL as a useful and evidence-based approach for consideration of the extent of protection from the broad array of vegetation-related effects associated with O_3 in ambient air. The currently available evidence, while somewhat expanded since the last review does not indicate an alternative metric for such a use; nor is an alternative approach evident. As noted in section 4.5.2 above the CASAC concurred that this approach is not called into question by the current evidence which continues to support "the use of tree seedling RBL as a proxy for the broader array of vegetation related effects" (Cox, 2020x, p. 21).

In considering the use of RBL and recognizing the role of the established E-R functions for 11 species of tree seedlings, and the median across the 11 species, we note that assessment of cumulative exposure in the 2015 review focused on use of the 3-year average seasonal W126 index with these functions. Thus, the discussion in the sections above (e.g., section 4.5.1.2) of the information available in the current review summarized and expanded on the technical issues that had informed the EPA's focus on the average over the 3-year design period in the last review.

Summarizing key points of that discussion here, we take note of the uncertainties associated with RBL estimates derived from the E-R functions. For example, as discussed in

section 4.3.4 above, while the E-R functions for the 11 species have been derived in terms of a seasonal (single-year) W126 index, the experiments from which they were derived vary in duration from periods of 82 to 140 over a single year days to periods of 180 to 555 days across two years. Thus, the "adjustment" to a single season carries uncertainties and contributes some imprecision to the resulting function and estimates derived using it. Additionally, we note that the exposure levels represented in the data underlying the E-R functions are somewhat limited at the lower cumulative exposure levels, such as those most commonly associated with the current standard (as characterized in Appendix 4D). Further, we recognize the variability that is associated with tree growth in the natural environment (e.g., related to variability in plant, soil, meteorological and other factors). In similar manner, we note the variability associated with plant responses to O_3 exposures in the natural environment. For example, we note the comparisons performed in the 2013 ISA and current ISA of RBL estimates based on either cumulative average multi-year W126 index or single-year W126 with estimates derived from measurements from a multi-year O_3 exposure study (as summarized in Appendix 4A, section 4.A.3.1). These presentations illustrate the variability inherent in the magnitude of growth impacts of O_3 and in the quantitative relationship of O_3 exposure and RBL, while also providing general agreement of predictions (based on either metric) with observations. Further, an illustrative example in Appendix 4A also provides quantitative estimates of potential differences in growth impacts of O_3 exposure controlled in terms of a 3-year average, such that the single-year values may vary while meeting the value specified for the average, compared to exposure controlled to such a value annually. While simplistic in nature, this example illustrates that based on the magnitude of variation documented for annual W126 index values occurring under the current standard, the magnitude of any differences in tree biomass between single-year and multi-year average approaches to controlling cumulative exposure would be expected to be quite small (Appendix 4A, section 4A.3). All of the factors identified here, the currently available evidence and recognized limitations, variability and uncertainties, contribute uncertainty and resulting imprecision or inexactitude to RBL estimates of single-year seasonal W126 index values, thus providing support for use of an average seasonal W126 index derived from multiple years (with their representation of variability in environmental factors), such as for a 3-year period, for estimating median RBL using the established E-R functions. Additionally, we take note of the CASAC advice in this review which affirmed the EPA's focus on a 3-year average W126, concluding that the EPA Administrator's focus on a 3-year average and her greater confidence in judgments related to public welfare impacts in doing so "appears of reasonable thought and scientifically sound" (Cox, 2020x, p. 19).

In considering tree growth effects, we also take note of the public welfare policy judgments inherent in the Administrator's decision in establishing the current standard in 2015.

Those judgments include her adoption of the median tree seedling RBL estimate for the studied species as a surrogate for the broad array of vegetation related effects that extend to the ecosystem scale, and her identification of cumulative seasonal exposures (in terms of the average W126 index across the 3-year design period for the standard) associated with a median RBL somewhat below 6% as an appropriate focus for considering target levels of protection for the 2015 standard. The newly available information in this review does not appear to call into question such judgements, indicating them to continue to appear reasonable in this review, and the current CASAC has agreed that this strategy "is still scientifically reasonable" (Cox 2020, p. 21).

Reviews of secondary NAAQS also require judgments on the extent to which particular welfare effects (e.g., with regard to type, magnitude/severity or extent) are important from a public welfare perspective. In the case of O_3, such a judgment includes consideration of the public welfare significance of small magnitude estimates of RBL and associated unquantified potential for larger-scale related effects. In establishing the current standard in 2015 with a focus on RBL as a proxy or surrogate for the broad array of vegetation effects, the Administrator took note of the 2014 CASAC characterization of 6% RBL (in seedlings of median tree species). As described in section 4.1 above, the rationale provided by the CASAC with this characterization was primarily conceptual and qualitative, rather than quantitative. The conceptual characterization recognized linkages between effects at the plant scale and broader ecosystem impacts, with the CASAC recommending that the Administrator consider RBL as a surrogate or proxy for the broader impacts that could be elicited by O_3. In the 2015 decision, the Administrator took note of this CASAC advice regarding use of RBL as a proxy and set the standard with an "underlying objective of a revised secondary standard that would limit cumulative exposures in nearly all instances to those for which the median RBL estimate would be somewhat lower than 6%" (80 FR 65407, October 26, 2015). While noting the CASAC view regarding 6% RBL in describing this objective, the Administrator did not additionally find that a cumulative seasonal exposure, for which such a magnitude of median species RBL was estimated, represented conditions that were adverse to the public welfare. Rather the 2015 decision 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). In the comments from the current CASAC in the context of its review of the draft PA, it expressed the view that the strategy described by the prior Administrator for the secondary standard established in 2015 with its W126 index target of 17 ppm-hrs (in terms of a 3-year average), at or below which the 2015 standard was expected to generally restrict cumulative seasonal exposure, is "still effective in

particularly protecting the public welfare in light of vegetation impacts form ozone" (Cox, 2020, p. 21).

The current evidence base and available information (qualitative and quantitative), as in the last review, continue to support consideration of the potential for O_3-related vegetation impacts in terms of the RBL estimates from established E-R functions as a quantitative tool within a larger framework of considerations pertaining to the public welfare significance of O_3 effects. Such consideration would include effects that are associated with effects on vegetation, and particularly those that conceptually relate to growth, and that are causally or likely causally related to O_3 in ambient air, yet for which there are greater uncertainties affecting estimates of impacts on public welfare. This approach to weighing the available information in reaching judgments regarding the secondary standard additionally takes into account uncertainties regarding the magnitude of growth impact that might be expected in mature trees, and of related, broader, ecosystem-level effects for which the available tools for quantitative estimates are more uncertain and those for which the policy foundation for consideration of public welfare impacts is less well established. (80 FR 65389, October 26, 2015).

In considering the quantitative analyses available in this review, we note the findings from the analysis of recent air quality at sites across the U.S., including in or near 65 Class I areas, and also analyses of historical air quality. Findings from the analysis of the air quality data from the most recent period and from the larger analysis of historical air quality data extending back to 2000 are consistent with the air quality analysis findings that were part of the basis for the current standard. That is, in virtually all design value periods and all locations at which the current standard was met (more than 99.9% of the observations), the 3-year average W126 metric was at or below 17 ppm-hrs, the target identified by the Administrator in establishing the current standard and in all such design value periods and locations the W126 metric was at or below 19 ppm-hrs as was also the case for the earlier and smaller dataset (80 FR 65404-65410, October 26, 2015). Additionally, across the full 19-year dataset for 56 Class I areas with monitors meeting the current standard during at least one or as many as seventeen 3-year periods since 2000, there are no more than 15 occurrences of a single-year W126 index above 19 ppm-hrs, with relatively fewer occurrences during the more recent part of the historical period, 2010 to 2018 (Appendix 4D, section 4D.3.2.3). Based on considerations summarized in section 4.5.1 above, the currently available information, including such infrequent single-year deviations of this magnitude above the average, could reasonably be judged not to pose meaningful risks of public welfare impacts to Class I areas.

In summary, the new information available is consistent with that available in the last review for the principal effects for which the evidence is strongest (e.g., growth, reproduction, and related larger-scale effects, as well as, visible foliar injury) and for key aspects of the

decision in that review. As discussed above, the currently available information does not provide established quantitative relationships and tools for estimating incidence and severity of visible foliar injury in protected areas across the U.S. or provide information linking extent and severity of injury to aesthetic values that might be useful for considering public welfare implications. Further, the currently available evidence for forested locations across the U.S., such as studies of USFS biosites, does not indicate widespread incidence of significant visible foliar injury. Additionally, the evidence regarding RBL and air quality in areas meeting the current standard does not appear to call into question the adequacy of protection. For other vegetation-related effects that the ISA newly concludes likely to be causally related to O_3, the new information does not provide us an indication of the extent to which such effects might be anticipated to occur in areas that meet the current standard of a significance reasonably judged significant to public welfare. Thus, we do not find the current information for these newly identified categories to call into question the adequacy of the current standard. Similarly, the current information regarding O_3 contribution to radiative forcing or effects on temperature, precipitation and related climate variables is not strengthened from that available in the last review, including with regard to uncertainties that limit quantitative evaluations. In recognizing similarities with the information based on which the current standard was set in 2015, we additionally note that, as in the last review, the Administrator's decision on the adequacy of public welfare protection afforded by the secondary O_3 standard from identified O_3-related effects and their potential to present adverse effects to the public welfare will be based in part on public welfare policy judgments regarding uncertainties and limitations in the available information.

We additionally note the advice from the CASAC in this review. With regard to the adequacy of the current secondary standard, it stated 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). The CASAC additionally stated that it "commends the EPA for the thorough discussion and rationale for the secondary standard." (Cox, 2020, p. 2).

Based on all of the above considerations, we conclude that the currently available evidence and quantitative exposure/risk information does not call into question the adequacy of the current secondary standard such that it is appropriate to consider retaining the current standard without revision. In so doing, we also recognize that, as is the case in NAAQS reviews in general, the extent to which the Administrator judges the current secondary O_3 standard to be adequate will depend on a variety of factors, including science policy judgments and public welfare policy judgments.

4.6 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH

In this section, we highlight key uncertainties associated with reviewing and establishing the secondary O_3 standard and additionally recognize that research in these areas may additionally be informative to the development of more efficient and effective control strategies. The list in this section includes key uncertainties and data gaps thus far highlighted in this review of the secondary standard. Additional information in several areas would reduce uncertainty in our interpretation of the available information and, accordingly, reduce uncertainty in our characterization of O_3-related welfare effects. For example, the items listed below generally include uncertainties associated with the extrapolation to plant species and environments outside of specific experimental or field study conditions and the assessment of ecosystem-scale impacts, such as structure and function. Additional E-R studies in different species or for responses other than reduced growth over multiple exposure conditions over growing seasons, that include details on exposure circumstances (e.g., hourly concentrations throughout the exposure), and exposure history, etc. would improve on and potentially expand characterizations of the potential for and magnitude of the identified vegetation effects under different seasonal exposures. Accordingly, in this section, we highlight areas for future welfare effects research, model development, and data collection activities to address these uncertainties and limitations in the current scientific evidence. These areas are similar to those highlighted in past reviews.

- While national visible foliar injury surveys have provided an extensive dataset on the incidence of such effects at sites across the country that experienced differing cumulative seasonal O_3 exposures and soil moisture conditions, there remain uncertainties in the current understanding of the relationship between seasonal O_3 exposures (and other influential factors, such as relative soil moisture) and the incidence and relative severity of visible foliar injury. Research to better characterize the relationship between O_3, soil moisture and foliar injury and specifically a quantifiable relationship between these (and any other influential) factors. Additionally, research would assist in interpreting connections between O_3-related foliar injury and other physiological effects and ecosystem services. For example, research is needed on the extent and severity of visible foliar injury that might impact ecosystem services (e.g., tourism), and the extent of impact it might have.
- Additional controlled exposure studies of effects, such as biomass impacts, that include multiple exposure levels within the lower range of exposures associated with ambient air quality conditions common today, extend over multiple years, and include the collection of detailed O3 concentration data over the exposure would reduce uncertainty in estimates of effects across multiple-year periods and at the O_3 exposures common today.
- Evidence newly available since the last review includes studies on insect-plant interactions that have established some statistically significant effects, but the evidence is still limited with regard to discerning a pattern of responses in growth, reproduction, or mortality, and a directionality of responses for most effects. More research is needed to investigate the degree of response and directionalities of these relationships, and to

investigate potential effects on pollination. The evidence is also limited with regard to the species represented (i.e., currently confined to three insect orders).

- Some evidence provides for linkages of effects on tree seedlings with larger trees and similarities in results between exposure techniques. Uncertainties remain in this area as well as uncertainties in extrapolating from O_3 effects on young trees (e.g., seedlings through a few years of age) to mature trees and from trees grown in the open versus those within the forest canopy.
- Uncertainties that remain in extrapolating individual plant response spatially or to higher levels of biological organization, including ecosystems, could be informed by research that explores and better quantifies the nature of the relationship between O_3, plant response and multiple biotic and abiotic stressors, including those associated with the affected ecosystem services (e.g., hydrology, productivity, carbon sequestration).
- Other uncertainties are associated with estimates of the effects of O_3 on the ecosystem processes of water, carbon, and nutrient cycling, particularly at the stand and community levels. These below- and above-ground processes include interactions of roots with the soil or microorganisms, effects of O_3 on structural or functional components of soil food webs and potential impacts on plant species diversity, changes in the water use of sensitive trees, and if the sensitive tree species is dominant, potential changes to the hydrologic cycle at the watershed and landscape level. Research on competitive interactions under different O_3 exposures and any associated impacts on biodiversity or genetic diversity would improve current understanding.
- Uncertainties related to characterizing the potential public welfare significance of O_3-induced effects and impacts to associated ecosystem services could also be informed by research. Research relating effects such as those on plant reproduction and propagation to effects on production of non-timber forest products, and research to characterize public preferences including valuation related to non-use and recreation for foliar injury, could 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 PA 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 O_3 concentration data from the EPA's Air Quality System (AQS) database (retrieved on August 14, 2019) for the years 2000 to 2018 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:
    - 8-hr periods which have at least six hourly concentrations[1]; and
    - 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:
    - 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
    - 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.

[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.

Table 2A-1. Summary of criteria describing the sites for which 8-hour metrics are 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	2016-2018	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)
Figure 2-9, DVs	2000-2018	
Figure 2-10, Trends	1980-2018	Annual fourth highest MDA8 values are based on all sites with at least 75% annual data completeness for at least 30 of the 39 years, with no more than two consecutive years having less than 75% complete data (n = 196 sites)
Figure 2-11, Trends	2000-2018	Annual fourth highest MDA8 values are based on all sites with at least 75% annual data completeness for at least 15 of the 19 years, with no more than two consecutive years having less than 75% complete data (n = 870 sites) Design values are presented for sites with valid DVs for at least 13 of the 17 3-year periods, with no more than two consecutive periods having invalid DVs (n = 629 sites)
Figure 2-12, Trends	2000-2018	
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

2A.2 ANALYSES OF 1-HOUR CONCENTRATIONS

Figure 2-15 of Chapter 2 presents hourly concentrations available in AQS (at the time of the data query on August 14, 2019) from any site with such data during the 2016-2018 period. The daily maximum 1-hr (MDA1) values presented in section 2.4.5 and (summary statistics shown in Table 2A-2 below) were calculated according to Appendix H to 40 CFR Part 50 for all sites with valid 2016-2018 design values for the current 8-hour standards. Generally, MDA1 values are derived (as the maximum 1-hr concentration during a day) for days for which at least 18 1-hr concentrations are available in AQS or for which a 1-hr concentration greater than 0.12 ppm has been reported in AQS. For this most recent design value period, the mean number of observations per site at or above 100 parts per billion (ppb) was well below one (0.19) for sites meeting the current standards compared to well above one (8.09) for sites not meeting the current standards.

Table 2A-2. Summary statistics for MDA1 concentrations at sites with differing design values for 2016-2018.

Statistic	Design Value (ppb)			
	41-60	61-70	71-84	85-111
Number of observations (obs)	137,443	635,822	226,876	44,059
Number of sites	149	695	241	42
25th percentile concentration (ppb)	33	37	38	45
Median concentration (ppb)	40	45	48	56
Mean concentration (ppb)	40.1	45.0	49.0	59.9
75th percentile concentration (ppb)	47	53	58	73
95th percentile concentration (ppb)	58	65	76	96
99th percentile concentration (ppb)	66	75	89	115
# of obs (# of sites) ≥ 240 ppb	1 (1)	0 (0)	0 (0)	0 (0)
# of obs (# of sites) ≥ 200 ppb	1 (1)	1 (1)	0 (0)	0 (0)
# of obs (# of sites) ≥ 160 ppb	1 (1)	2 (2)	0 (0)	1 (1)
# of obs (# of sites) ≥ 120 ppb	3 (2)	17 (11)	26 (23)	316 (27)
# of obs (# of sites) ≥ 100 ppb	14 (9)	149 (94)	561 (165)	1,729 (42)
Mean # of obs ≥ 100 ppb per site[A]	0.09	0.21	2.33	41.17

[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 0.19 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 8.09 obs ≥ 100 ppb per site.

The figures and tables presented below contain additional analyses based on the MDA1 concentrations for years 2000-2004 and 2014-2018. Figure 2A-1 compares the distribution of MDA1 concentrations for each 8-hour design value bin between the earlier (2000-2004; red boxes) and latter (2014-2018; blue boxes) periods. The comparison shows a slight upward shift in the mid-range concentrations for the highest (≥ 85 ppb) and lowest (≤ 60 ppb) DV bins, while the two middle bins show little change. The range between the 1st and 99th percentiles as represented by the whiskers shrinks slightly between the earlier and latter periods in all four bins. Finally, the very highest concentrations (shown as dots above the top whisker) are reduced in the two highest DV bins. This is also reflected in Table 2A-3 and Table 2A-4, which show summary statistics similar to Table 2A-2 for the 2000-2004 and 2014-2018 periods, respectively. These tables show, as might be expected, that sites with higher design values have a larger number of days with MDA1 values at or above 100 ppb than sites with lower design values. This statistic is nearly 40 times higher in both periods for sites not meeting the current standards compared to sites meeting the current standards. Across the three design value periods in 2014 to 2018, sites not meeting the current standards have on average nearly 20 observations at or above 100 ppb per 3-year period, while the average for sites meeting the current standards is less than 0.5.

Figure 2A-2 and Figure 2A-3 show maps of the average number of days where the MDA1 concentrations were greater than or equal to 100 ppb (also known as the N100 metric) for the 2000-2004 and 2014-2018 periods, respectively. These maps show that nearly all sites in the

U.S. have seen a large reduction in the number of days with high MDA1 concentrations since the beginning of the century. This is also reflected in the final rows of Table 2A-3 and Table 2A-4, which indicate a decrease of 84% in the total number MDA1 values greater than or equal to 100 ppb between 2000-2004 and 2014-2018.

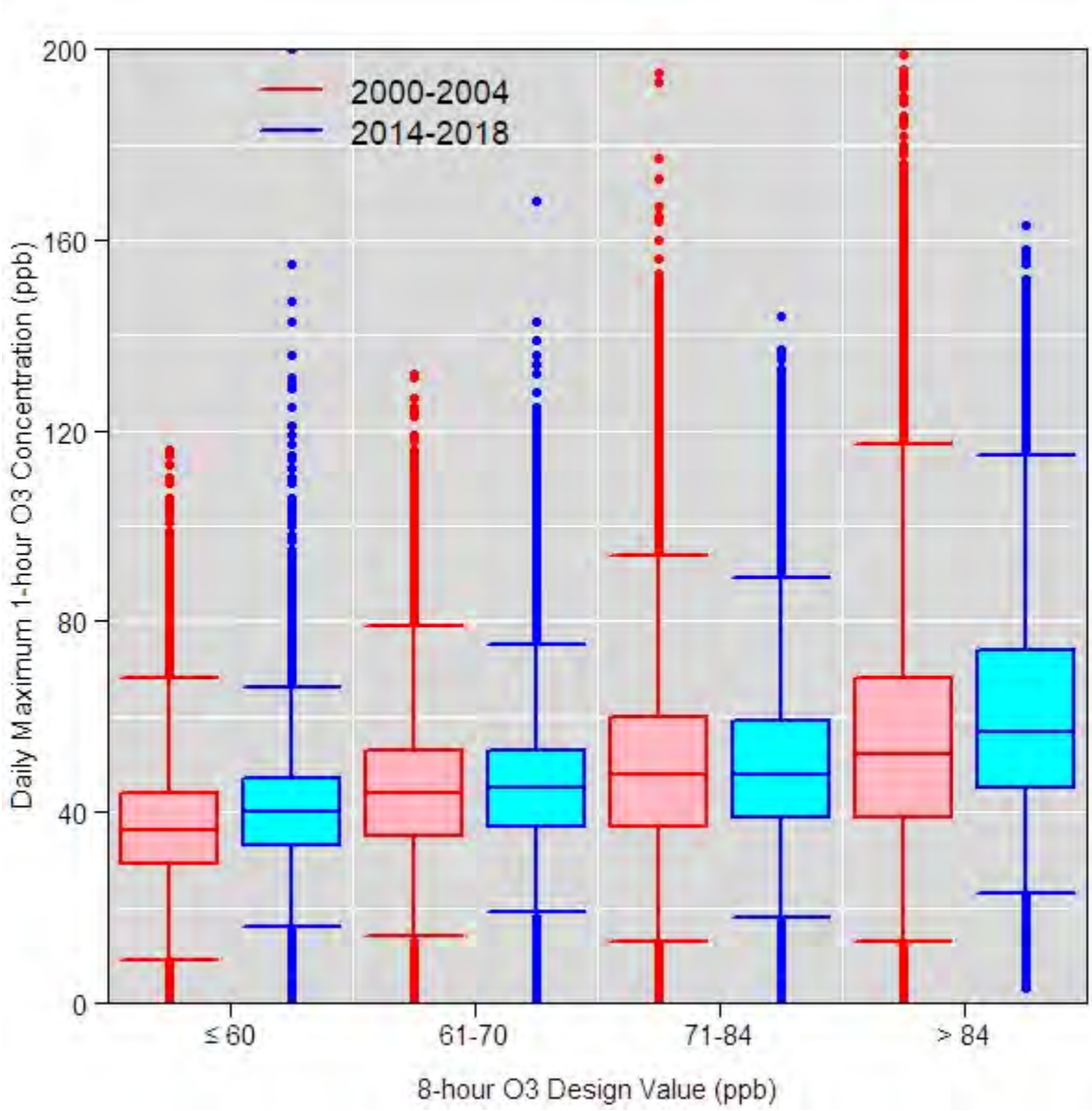

Figure 2A-1. Boxplots comparing the distribution of MDA1 concentrations for 2000-2004 (red) to the distribution of MDA1 concentrations for 2014-2018 (blue), binned by the 8-hour design value at each monitoring site. The boxes represent the 25th, 50th and 75th percentiles and the whiskers represent the 1st and 99th percentiles. Outlier values are represented by circles.

Table 2A-3. Summary statistics for MDA1 concentrations at differing design values for 2000-2004.

Statistic	Design Value (ppb)			
	39-60	61-70	71-84	85-131
Number of observations (obs)	115,061	286,026	1,410,915	964,577
Number of design values (DVs)[A]	127	313	1,660	1,229
25th percentile concentration (ppb)	29	35	37	39
Median concentration (ppb)	36	44	48	52
Mean concentration (ppb)	36.4	44.4	49.4	54.9
75th percentile concentration (ppb)	44	53	60	68
95th percentile concentration (ppb)	56	68	79	95
99th percentile concentration (ppb)	68	79	94	117
# 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)	5 (5)
# of obs (# of DVs[A]) ≥ 160 ppb	0 (0)	0 (0)	15 (9)	270 (97)
# of obs (# of DVs[A]) ≥ 120 ppb	0 (0)	8 (6)	720 (373)	7,967 (977)
# of obs (# of DVs[A]) ≥ 100 ppb	24 (15)	162 (77)	7,838 (1,360)	34,587 (1,229)
Mean # of obs ≥ 100 ppb per site[B]	0.19	0.52	4.72	28.14

[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 0.42 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.

Table 2A-4. Summary statistics for MDA1 concentrations at differing design values for 2014-2018.

Statistic	Design Value (ppb)			
	27-60	61-70	71-84	85-112
Number of observations (obs)	473,542	1,903,711	620,637	114,656
Number of design values (DVs)[A]	523	2,120	667	110
25th percentile concentration (ppb)	33	37	39	45
Median concentration (ppb)	40	45	48	57
Mean concentration (ppb)	40.3	45.2	49.4	60.2
75th percentile concentration (ppb)	47	53	59	74
95th percentile concentration (ppb)	58	66	76	97
99th percentile concentration (ppb)	66	75	89	115
# of obs (# of DVs[A]) ≥ 240 ppb	1 (1)	2 (2)	0 (0)	0 (0)
# of obs (# of DVs[A]) ≥ 200 ppb	3 (3)	5 (5)	0 (0)	0 (0)
# of obs (# of DVs[A]) ≥ 160 ppb	4 (4)	6 (6)	0 (0)	3 (3)
# of obs (# of DVs[A]) ≥ 120 ppb	18 (13)	40 (29)	88 (61)	788 (78)
# of obs (# of DVs[A]) ≥ 100 ppb	47 (31)	404 (242)	1,540 (445)	4,822 (110)
Mean # of obs ≥ 100 ppb per site[B]	0.09	0.19	2.31	43.84

[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 0.17 obs ≥ 100 ppb per site, and for the two highest bins combined (i.e., sites with a design value > 70 ppb), the mean is 8.19 obs ≥ 100 ppb per site.

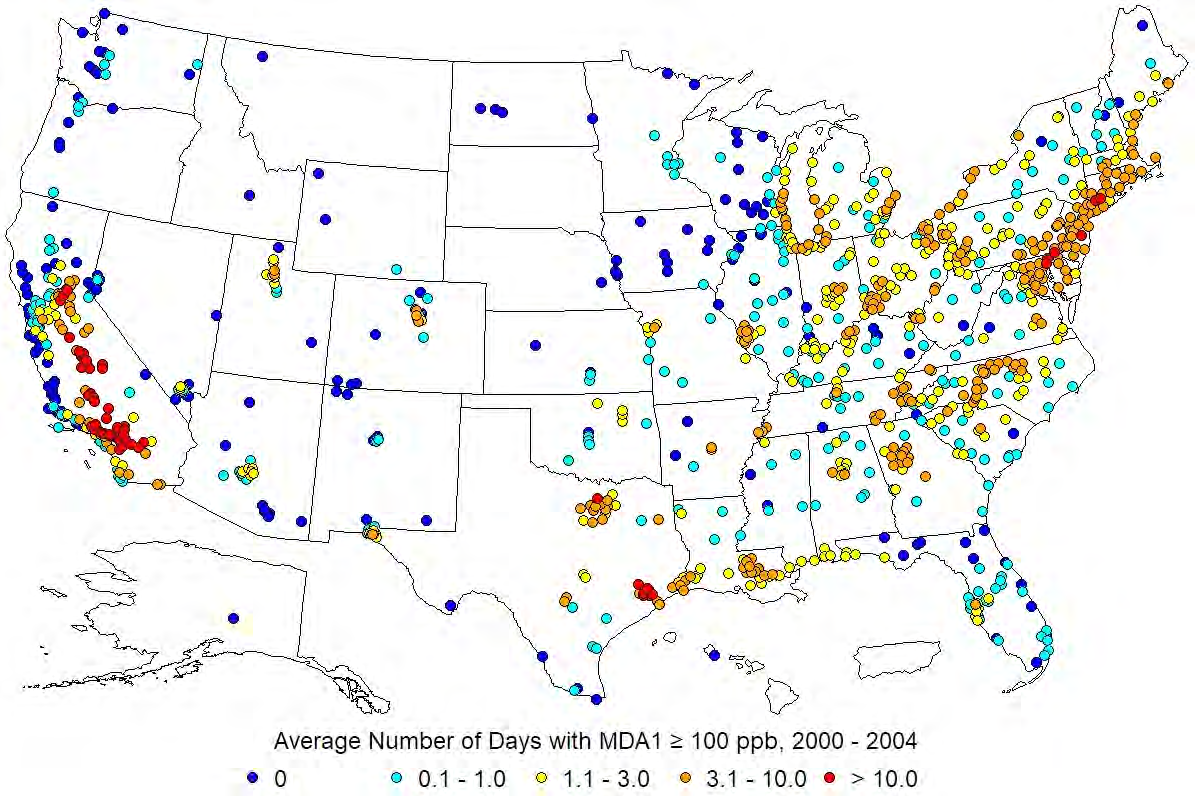

Figure 2A-2. Map showing the average number of days with MDA1 ≥ 100 ppb, 2000-2004.

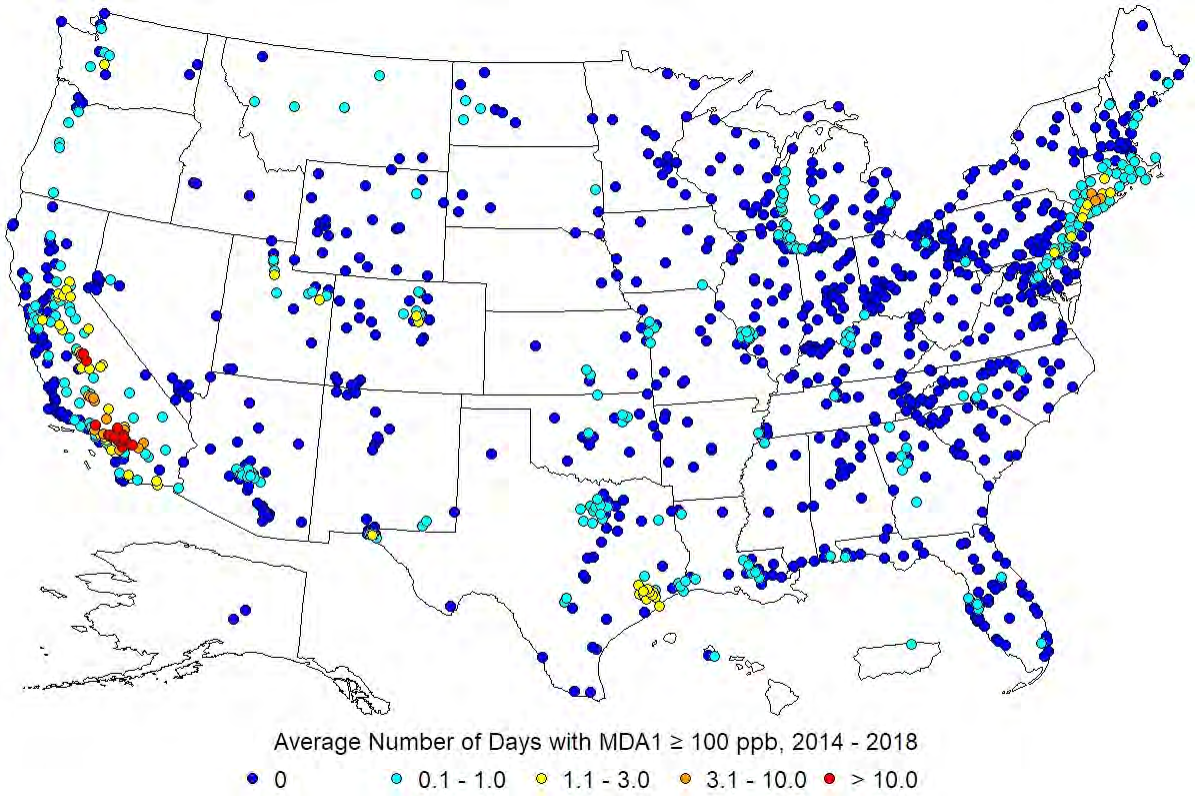

Figure 2A-3. Map showing the average number of days with MDA1 ≥ 100 ppb, 2014-2018.

Figure 2A-4 below shows the number of days in 2016-2018 with an MDA1 concentration at or above 100 ppb and 8-hour design values (similar to Figure 2-16), for all sites with a 2016-2018 design value less than 98 ppb. All sites meeting the current standard had six or fewer (i.e., two or fewer per year) MDA1 values at or above 100 ppb, and all but three sites meeting the current standard had three or fewer (i.e., one or fewer per year) MDA1 values at or above 100 ppb.

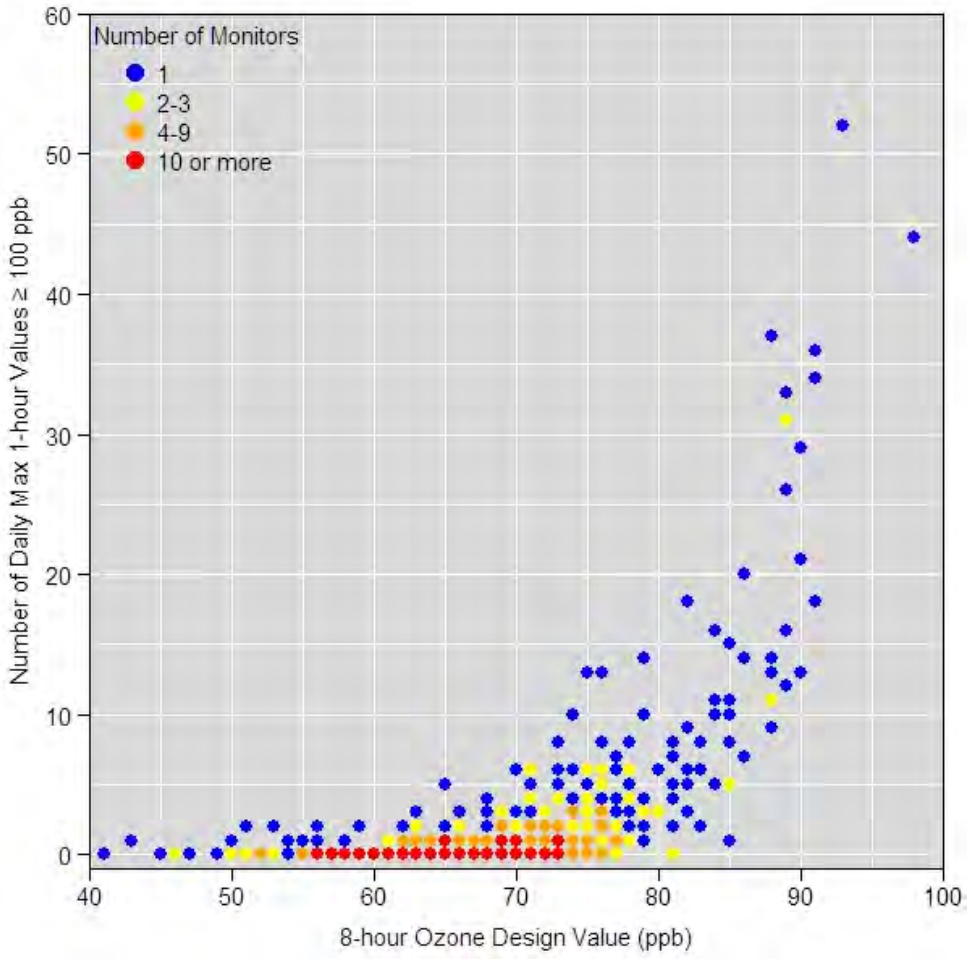

Figure 2A-4. Number of days in 2016-2018 at each monitoring site with a MDA1 concentration greater than or equal to 100 ppb and an 8-hour design value less than 98 ppb. Sites with higher design values had more days, up to a maximum of 164 (at a site in southern CA).

APPENDIX 2B

ADDITIONAL DETAILS ON BACKGROUND OZONE MODELING AND ANALYSIS

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This appendix for the background ozone (O_3) modeling and analysis includes a description of the methodology for photochemical modeling, an evaluation of the modeling, and a more detailed analysis of the predicted contributions from international anthropogenic emissions. The methodology section includes a description of the modeling platform and emissions. The evaluation section includes comparisons against surface, sondes and satellite measurements. The international component analysis separately estimates O_3 impacts from China, India, Canada/Mexico, and global shipping at the hemispheric scale.

2B.1 PHOTOCHEMICAL MODELING METHODOLOGY

2B.1.1 Modeling Platform Overview

A multiscale modeling system is applied at both hemispheric and regional scales with consistent methodologies for emissions inputs, meteorological inputs, model chemistry, and photochemical models. Consistency across spatial scales reduces the number of assumptions that have to be made in integrating predictions from the global and the regional modeling. However, methodological consistency does not address sources of uncertainty associated with individual inputs used by the modeling system.

The modeling system uses one emission model, one meteorological model, and one chemical transport model. The meteorological model is the Weather Research and Forecasting model (WRF v3.8). The emissions model is the Sparse Matrix Operating Kernel for Emissions (SMOKE v4.5). The chemical transport model is the Community Multiscale Air Quality model (CMAQ) version 5.2.1 with the Carbon Bond mechanism (CB6r3) and the non-volatile aerosol option (AE6). Each of these models is applied at hemispheric and regional scales. The regional meteorology components of the modeling system are described in more detail in section 3C.4.1.4 of Appendix 3C, while emissions inputs are summarized here.

The models identified above are configured differently for the hemispheric and regional scales as appropriate for the intended purpose. The hemispheric scale model uses a polar stereographic projection at 108 kilometer (km) resolution to completely and continuously cover the Northern Hemisphere. At the regional scale, the model employs a Lambert conic conformal projection at 36 km resolution to cover North America and at 12 km resolution to cover the lower 48 contiguous states. The hemispheric scale allows for long-range free tropospheric transport with 44 layers between the surface and 50 hPa (~20 km asl). The 36 km and 12 km regional modeling has 35 vertical layers between the surface and 50 hPa. The hemispheric modeling system was initiated on May 1, 2015 and run continuously through December 31, 2016. The regional model was initialized using the hemispheric result on December 21, 2015 and run continuously through December 31, 2016.

2B.1.2 Emissions Overview

The emissions inventories are summarized here and more information is available in the Emissions Technical Support Documents (U.S. EPA, 2019a, U.S. EPA, 2019b) and in Appendix 3C. The emissions model inputs are discussed separately for natural and anthropogenic emissions. The stratospheric fluxes (section 2.5.1.1 of main document) are not discussed here because, although they are a source of ozone, they are not emissions. The regional inventories over North America are based on the Inventory Collaborative 2016 emissions modeling platform (*http://views.cira.colostate.edu/wiki/wiki/9169*), which was developed through the summer of 2019. Three versions of the 2016 inventory developed: "alpha" (also known as the 2016v7.1 platform) – which consisted of data closely related to the 2014 National Emissions Inventory (NEI) version 2 and 2016-specific data for some sectors; "beta" (also known as the 2016v7.2 platform) – which incorporated data from state and local agencies and adjustments to better represent the year 2016; and "version 1" (also known as the 2016v7.3 platform) – which has the completed representation of 2016 and some elements from the 2017 NEI. For any regional inventories, this analysis used the 2016 "alpha release" (specifically the modeling case abbreviated 2016fe) that is publicly available from *https://www.epa.gov/air-emissions-modeling/2016-alpha-platform*. Any changes in the 2016 "beta" or "version 1" platforms are not included in this modeling and therefore are not captured in the subsequent analysis.

2B.1.2.1 Natural Emission Inventory

The natural emission inventory databases cover all the sources discussed in section 2.5.1 except the International Anthropogenics. The databases that are available depend upon the scale. At the global scale, lightning NO_X emissions are based on monthly climatological data; biogenic VOC emissions have hourly and day-specific (MEGAN v2.1, Guenther et al., 2012) temporal scales; soil NO_X also has hourly and day-specific temporal scales (Berkeley Dalhousie Soil NO_X Parameterization, as implemented by Hudman et al., 2012); and fire emissions are based on day-specific data (FINN v1.5, Wiedinmyer et al., 2011). Over our regional domain, regional inventories supersede the biogenic VOCs, soil NO_X, and fire emissions using estimates consistent with the 2016 collaborative emissions modeling platform (*https://www.epa.gov/air-emissions-modeling/2016-alpha-platform*). The regional biogenic VOCs and soil NO_X are derived from the Biogenic Emission Inventory System (BEIS v3.61). Of the natural inventories, only fires are expected to change significantly in future versions of the 2016 emissions platform. The biogenic VOC and NO_X changes will be minor due to small changes to the land use data input to BEIS3.

Emissions of NO_X are of particular importance to this study and the natural inventory is summarized here. The total natural NO_X emissions[3] in this platform is 56 megatons NO_X (reported as equivalent NO_2 mass) which is approximately 15.5 TgN. The contributors in order of magnitude are lightning (55%), soil (33%), and wildfires (12%). Lightning is treated as a climatological monthly mean contribution, while soils and wildfires are day-specific. It is important to note that outside North America, prescribed fires are not identified distinctly from wildfires. Therefore, all wildland fires outside North America are treated as natural. Though not directly comparable, the lightning and soil magnitudes are consistent with the ranges reported by (Lamarque et al., 2012). Consistent with previous regional modeling platforms, the lightning emissions are not included in the emissions inputs to the regional modeling platform. At the regional scale, the representation of lightning as a monthly mean rate would add lightning on days where it may not have occurred. At the hemispheric scale, omitting lightning would remove an important contribution to the well-mixed background O_3.

2B.1.2.2 Anthropogenic Emission Inventory

Anthropogenic emissions inputs include both domestic and international sources. The domestic inventory includes a high-level of detail that is consistent with previous EPA emissions platforms such as those used to model the year 2011 (*https://www.epa.gov/air-emissions-modeling/2011-version-6-air-emissions-modeling-platforms*). For the hemispheric emissions modeling platform, there are over thirty anthropogenic sector of emission files. The traditional regional platform covers North America including the U.S. sectors, Canadian sectors, and Mexican sectors. In addition to the typical regional platform sectors, there are nine sectors based on the Hemispheric Transport of Air Pollution Version 2 (EDGAR-HTAPv2) inventory and 15 sectors that represent emissions in China which together comprise the anthropogenic emissions outside of North America. The international emission inventories are synthesized from the EDGAR-HTAP v2 harmonized emission inventory and country specific databases where updates were likely to be influential. Previous assessments like HTAP (2010, Phase 1) and HTAP (Phase 2) have shown that the anthropogenic portion of USB is most sensitive to emissions in Mexico, Canada, and China. For Mexico and Canada, the hemispheric platform relies on the same country-specific databases as the regional platform. For China, as mentioned above, the hemispheric platform uses a new country specific database. The sources are detailed further below.

The EDGAR-HTAP v2 inventories were projected to represent the year 2014. Projection factors were calculated from the Community Emissions Data System (CEDS) inventory at a

[3] 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.

country-sector level. This allowed our inventory to evolve without the risks associated with transitioning to a new inventory system. Especially because EDGAR-HTAP v2 is superseded for critical counties, this was the optimal approach. Details of scaling factor development are described in Section 2.1.5 of the 2016v7.1 Hemispheric Modeling Platform Technical Support Document (U.S. EPA, 2019a).

Emissions estimates over Mexico are a combination of emissions supplied by the Mexican government and emissions developed by the EPA. For the 2016 platform, emissions for point, nonpoint, and nonroad sources were developed based on projections of Secretariat of Environment and Natural Resources (SEMARNAT)-supplied data for the year 2008. For the onroad mobile sources, the EPA developed year-specific inventories for 2014 and 2017 by applying the MOVES-Mexico model and interpolating to the year 2016. More details are available in the 2016v7.1 emissions platform TSD (U.S. EPA, 2019b).

Emissions for Canada were supplied by Canadian agencies and reprocessed by the EPA for the domains and model years used in this analysis. Environment and Climate Change Canada (ECCC) supplied data for four broad inventory sectors (point, on-road mobile, fugitive dust, and area and non-road mobile sources, the latter including commercial marine vessels). The ECCC emissions were interpolated to 2016 based on inventories from the years 2013 and 2025.

The China emission inventory was developed at Tsinghua University (THU) and documented in Zhao et al., 2018 (see supplement). This inventory was extensively compared to the EDGAR-HTAP v2 and EDGAR v4.3 inventories before use. The largest differences for NO_X in 2016 occurred in individual emissions sectors rather than inventory totals. The SO_2 emissions were more different than NO_X emissions between the two inventories because the THU inventory applies controls to the metal industry that have been adopted by China. The difference between emissions, primarily NOx emissions, causes small decrease in the spring time surface O_3 over the U.S. compared to using EDGAR-HTAP v2. Comparisons of this update are summarized by Henderson et al.(2019).

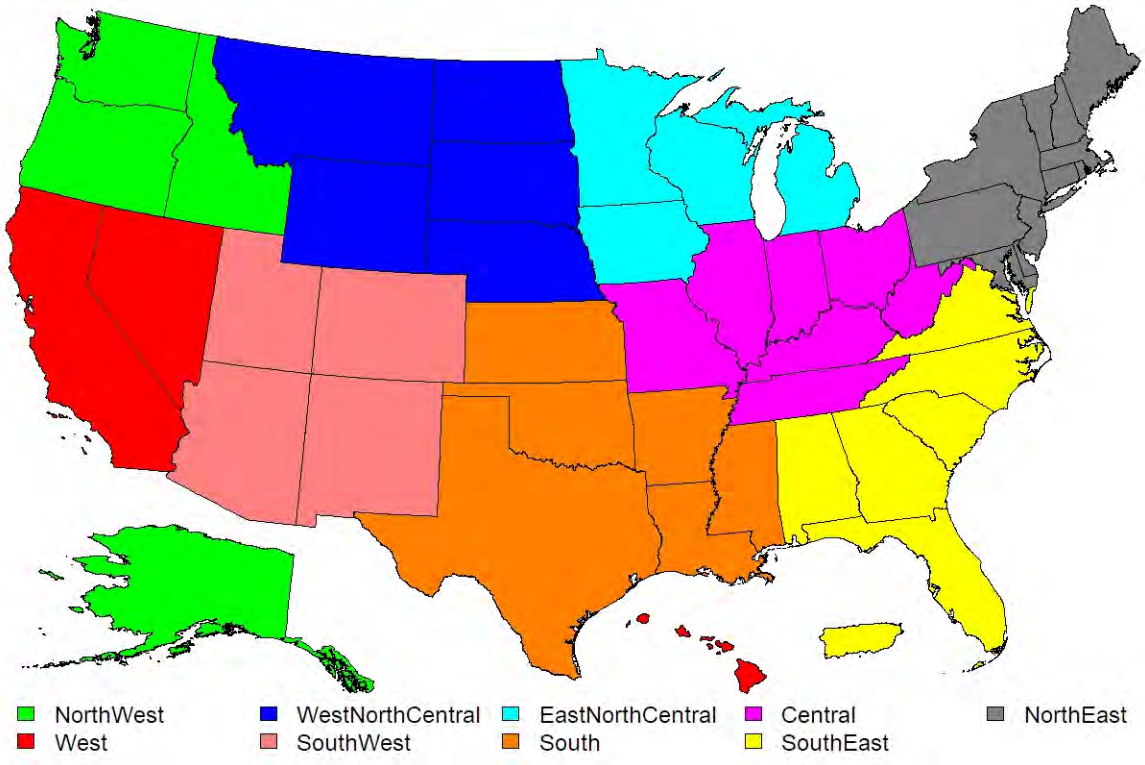
Emissions for the United States representing the year 2016 were developed using the 2014 National Emissions Inventory version 2 (2014NEIv2) as the starting point, although emissions for some data categories were updated to better represent the year 2016. The point source emission inventories for the platform are partially updated to represent 2016. Because 2016 is not a year for which a full NEI is compiled, states are only required to submit emissions for their larger point sources. For units without 2016-specific emissions, the emissions were carried forward from the 2014 NEIv2. For electric generating units, 2016-specific Continuous Emissions Monitoring System (CEMS) data are used where the data can be matched to units in the NEI. Point and nonpoint oil and gas emissions were projected from 2014 to 2016 using factors based on historic production levels.

Other sectors are briefly summarized here and the reader is directed to the TSD for more details (U.S. EPA, 2019a). Agricultural and wildland (including prescribed) fire emissions were developed for the year 2016 using methods similar to those used to develop the 2014 NEI, except that the input data relied on nationally-available data sets and did not benefit from state-submitted data as are used for NEI year emissions. The assignment of wildland fires to wild or prescribed is a complex process that is documented in the regional platform emissions TSD (U.S. EPA, 2019b). Most area source sectors for this platform use unadjusted 2014 NEIv2 emissions estimates except for commercial marine vehicles (CMV), fertilizer emissions, oil and gas emissions, and onroad and nonroad mobile source emissions. For CMV, SO_2 emissions were updated to reflect new rules for the North American Emission Control Area (regulation 13.6.1 and appendix VII of MARPOL Annex VI) on sulfur emissions that took effect in the year 2015. For fertilizer ammonia emissions, a 2016-specific emissions inventory is used in this platform, while animal ammonia emissions were the same as those in 2014 NEIv2. Onroad and nonroad emissions were developed based on MOVES2014a outputs for the year 2016, and the activity data used to compute the onroad emissions were projected from 2014 to 2016 based on distinct state-specific factors for urban and rural roads. Emissions from 2014 NEIv2 were used directly for residential wood combustion, fugitive dust, and other nonpoint sources, although meteorological-based adjustments for dust sources and temporal allocation for residential wood and agricultural ammonia sources were based on 2016 meteorology. Additional details on the development of the U.S., Canada, and Mexico emissions are provided in the 2016v7.1 (U. S. EPA, 2019b).

2B.2 EVALUATION

An operational model performance evaluation for O_3 was conducted for the 2016fe simulation (as referred to in Section 2.5.2.2) using monitoring data, ozone sonde data, and satellite data in order to estimate the ability of the CMAQv5.2.1 modeling system to replicate the 2016 base year O_3 concentrations for the 12 km continental U.S. domain and the 108 km Northern Hemispheric domain. The purpose of this evaluation is to examine the ability of the 2016 air quality modeling platform to represent the magnitude and spatial and temporal variability of measured (i.e., observed) O_3 concentrations within the modeling domain. The model evaluation for O_3 focuses on comparisons of model-predicted 8-hour daily maximum concentrations (MDA8) to the corresponding concentrations from monitoring data (for 2016) collected at monitoring sites in the AQS. The evaluation divided these data into two datasets, one limited to only CASTNET sites (described in section 2.3.1), and the second comprised of all other sites. We refer to this second dataset as “AQS.”

Included in the evaluation are statistical measures of model performance based upon model-predicted versus observed MDA8 O_3 concentrations that were paired in space and time. Statistics were generated for each of the nine National Oceanic and Atmospheric Administration (NOAA) climate regions of the 12-km U.S. modeling domain (Figure 2B-1). The regions include the Northeast, Central, EastNorthCentral, Southeast, South, Southwest, WestNorthCentral, Northwest and West as were originally identified in Karl and Koss (1984). Note that most monitoring sites in the West region are located in California, therefore statistics for the West will be mostly representative of California O_3 model performance.



Source: *http://www.ncdc.noaa.gov/monitoring-references/maps/us-climate-regions.php#references*

Figure 2B-1. NOAA U.S. climate regions.

For MDA8 O_3, model performance statistics were calculated for each climate region by season and for the May through September O_3 season of 2016. Seasons were defined as: winter (December-January-February), spring (March-April-May), summer (June-July-August), and fall (September-October-November). Observational data were excluded from the analysis and model evaluations for sites that did not meet a 75% completeness criterion.[4] In addition to the performance statistics, several graphical presentations of model performance were prepared for MDA8 O_3 concentrations. These graphical presentations include:

[4] 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) density scatter plots of observations obtained from the AQS system excluding CASTNET (hereafter AQS) and predicted MDA8 O_3 concentrations for May through September;

(2) regional maps that show the mean bias and error as well as normalized mean bias and error calculated for MDA8 ≥ 60 ppb for May through September at individual AQS and CASTNET monitoring sites;

(3) tile plots that show normalized mean bias (%) and mean bias (ppb) of MDA8 and MDA8 ≥ 60 ppb by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites;

(4) O_3 sonde evaluations comparing vertically resolved ozone model predictions to ozone sondes measurements from the World Ozone and Ultraviolet Data Centre (*woudc.org*).

(5) satellite evaluation comparing simulated tropospheric vertical column densities of O_3, nitrogen dioxide, and formaldehyde to OMI retrievals.

The Atmospheric Model Evaluation Tool (AMET) was used to calculate the model performance statistics used in this evaluation (Gilliam et al., 2005). For this evaluation of the O_3 predictions in the 2016fe CMAQ modeling platform, we have selected the mean bias, mean error, normalized mean bias, and normalized mean error to characterize model performance, statistics which are consistent with the recommendations in Simon et al. (2012) and the photochemical modeling guidance (U.S. EPA, 2018).

Mean bias (MB) is used as average of the difference (predicted – observed) divided by the total number of replicates (*n*). Mean bias is defined as:

MB = $\frac{1}{n}\sum_1^n(P - O)$, where P = predicted and O = observed concentrations for every site and day included in the evaluation.

Mean error (ME) calculates the absolute value of the difference (predicted - observed) divided by the total number of replicates (*n*). Mean error is defined as:

ME = $\frac{1}{n}\sum_1^n |P - O|$

Normalized mean bias (NMB) is used as a normalization to facilitate a range of concentration magnitudes. This statistic averages the difference (predicted - observed) over the sum of observed values. NMB is a useful model performance indicator because it avoids overinflating the observed range of values, especially at low concentrations. Normalized mean bias is defined as:

$$NMB = \frac{\sum_{1}^{n}(P - O)}{\sum_{1}^{n}(O)} * 100$$, where P = predicted concentrations and O = observed

Normalized mean error (NME) is also similar to NMB, where the performance statistic is used as a normalization of the mean error. NME calculates the absolute value of the difference (model - observed) over the sum of observed values. Normalized mean error is defined as

$$NME = \frac{\sum_{1}^{n}|P - O|}{\sum_{1}^{n}(O)} * 100$$

As described in more detail below, the model performance statistics indicate that the MDA8 O_3 concentrations predicted by the 2016 CMAQ modeling platform closely reflect the corresponding monitoring data-based MDA8 O_3 concentrations in space and time in each region of the U.S. modeling domain. The acceptability of model performance was judged for the 2016 CMAQ O_3 performance results considering the range of performance found in recent regional O_3 model applications (NRC, 2002; Phillips et al., 2008; Simon et al., 2012; U.S. EPA, 2009; U.S. EPA, 2018). These other modeling studies represent a wide range of modeling analyses that cover various models, model configurations, domains, years and/or episodes, chemical mechanisms, and aerosol modules. Overall, the 2016 CMAQ O_3 model performance results are within the range found in other recent peer-reviewed and regulatory applications. The model performance results, as described in this document, demonstrate the predictions from the 2016 modeling platform closely replicate the corresponding observed concentrations in terms of the magnitude, temporal fluctuations, and spatial differences for 8-hour daily maximum O_3.

The model performance bias and error statistics for MDA8 O_3 predictions in each of the nine NOAA climate regions and each season are provided in Table 2B-1. As noted above, seasons were defined as: winter (December-January-February), spring (March-April-May), summer (June-July-August), and fall (September-October-November). As indicated by the statistics in Table 2-7, mean bias and error for 8-hour daily maximum O_3 are relatively low in each subregion, not only in the summer when concentrations are highest, but also during other times of the year. Generally, MB for MDA8 $O_3 \geq 60$ ppb is less than $\pm$ 10 ppb. Generally, MDA8 O_3 at the AQS sites in the summer and fall is over predicted except in the Southwest, with the greatest over-prediction in the EastNorthCentral and WestNorthCentral. Likewise, MDA8 O_3 at the CASTNET sites in the summer and fall is typically over predicted except in the West, Southwest and WestNorthCentral where the bias shows an under-prediction. In the winter and spring,

MDA8 O_3 is under predicted at AQS and CASTNET sites in all the climate regions (with NMBs less than approximately $\pm$ 25 percent in each subregion).

Figure 2B-2 and Figure 2B-3 are tile plots that summarize to provide an overview of model performance by region and by season. Figure 2B-2 shows NMB (%) and MB (ppb) of MDA8 by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites. Likewise, Figure 2B-3 shows the NMB (%) and MB (ppb) of MDA8 $\geq$ 60 ppb by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites. Figure 2B-2 shows that for the majority of the nine climate regions throughout each year the NMB is within ±10 percent. There is greater over-prediction (<20%) during the fall in the South, EastNorthCentral (*aka* Upper Midwest), and Central (*aka* Ohio Valley) regions and during the summer in the South, Southeast and Central (*aka* Ohio Valley) regions. However, there is greater under-prediction (up to 30 percent) during the winter in the Northwest, Southwest, WestNorthCentral (*aka* NRockiesPlains), EastNorthCentral (*aka* Upper Midwest), Central (*aka* Ohio Valley), and Northeast regions as well during the spring in the Northwest.

The density scatterplots in Figure 2B-4 to Figure 2B-12 provide a qualitative comparison of model-predicted and observed MDA8 O_3 concentrations for each climate region by season. In these plots the intensity of the colors indicates the density of individual observed/predicted paired values. The greatest number of individual paired values is denoted by locations in the plot denoted in warmer colors. The plots indicate that the predictions correspond closely to the observations in that a large number of observed/predicted paired values lie along or close to the 1:1 line shown on each plot. The model is more likely to over-predict the observed values at low and mid-range concentrations generally < 60 ppb in each of the regions. There are some relatively infrequent very large over predictions at high concentrations. Preliminary review of these biases finds that some are related to fire impacts.

Spatial plots of the MB, ME, NMB and NME for individual monitors are shown in Figure 2B-13 through Figure 2B-16, respectively. The statistics shown in these two figures were calculated over the May through September period, using data pairs on days with observed 8-hr O_3 of greater than or equal to 60 ppb. Model bias at individual sites during the O_3 season is similar to that seen on a sub-regional basis for the summer. Figure 2B-13 shows the mean bias for 8-hr daily maximum O_3 greater than 60 ppb is under predicted overall, but generally within ±10 ppb across the AQS and CASTNET sites. The greatest exceptions are most evident at certain near-coastal sites where, on average, the model over predicts MDA8 observed $O_3 \geq 60$ ppb. Likewise, the information in Figure 2B-15 indicates that the normalized mean bias for days with observed 8-hr daily maximum O_3 greater than 60 ppb is within ± 10% at the vast majority of monitoring sites across the U.S. domain. Model error, as seen from Figure 2B-14 and Figure 2B-16, is generally 2 to 10 ppb and 20 percent or less at most of the sites across the U.S. modeling

domain. Somewhat greater error is evident at sites in several areas most notably in the West, WestNorthCentral, Northeast, EastNorthCentral, Southeast, and along portions of the Gulf Coast and Great Lakes coastlines.

Sonde evaluations are shown for the 108 km Northern Hemisphere domain in Figure 2B-18 through Figure 2B-21. The sondes used in this analysis and their release frequencies are shown in Figure 2B-17. Figure 2B-18 shows that the annual mean prediction is generally within 20% of the measured sonde data, except for near the tropopause. Figure 2B-19 shows that the performance of all sites is generally not as good in the spring (March, April, May) than in the summer (June, July, August). The seasonal performance of each monitor is shown in Figure 2B-20 for spring and Figure 2B-21 for summer. By comparison, Figure 2B-20 shows that low biases extend deeper into the troposphere in spring than in summer. The structure of the bias seems to suggest a stratospheric causal mechanism because the bias is near the tropopause.

Satellite evaluations in this analysis include tropospheric vertical columns of O_3, nitrogen dioxide (an ozone precursor as described in chapter 2), and formaldehyde (a VOC reaction product which is an indicator of VOCs and total reactivity of the atmosphere). At this time, only formaldehyde comparison includes the application of the scattering weights and air mass factor to the model, which are often used to create an averaging kernel. Similar processing for O_3 and NO_2 was not available at the time this appendix was completed. Satellite evaluations focus exclusively on the 108 km results over the Northern Hemisphere.

Simulated O_3 tropospheric vertical column densities are compared to the O_3 product described and evaluated by Huang et al. (2017). Figure 2B-22 and Figure 2B-23 compares the model to the retrieved column data without application of the averaging kernel. Omitting the averaging kernel introduces some error into the comparison (Huang et al., 2017; see Figure 9 for details). Even so, the comparison shows reasonable performance within the mid-latitudes. There is a notable low bias in January mid-latitudes and near the north pole in April. In addition, high biases are consistently seen near the corners of the domain in January and April. This cause of this high-bias pattern will require further analysis. Within the mid latitudes, the model is performing well with notable low biases in January and scattered high biases in Asia in July. Given the limitations of the comparison, the performance is quite good.

Simulated nitrogen dioxide (NO_2) vertical columns are compared is the OMNO2d (Krotkov et al., 2017, as processed by Lok Lamsal called OMNO2D_HR). Similar to O_3, the averaging kernel is not being applied for NO_2. Figure 2B-24 and Figure 2B-25 show larger relative biases for NO_2 than O_3, particularly in low NO_2 regions like over the oceans. Best performance was over land during July. Model comparisons to NO_2 have commonly shown biases and research in the broader community continues to resolve this issue.

Formaldehyde retrieval comparisons are shown in Figure 2B-26 and Figure 2B-27 using the OMHCHO files, but using the recommended product described by González Abad et al. (2015). The formaldehyde retrievals show a seasonal cycle in the evaluation with a low bias for the northern-most retrievals in January and October. During April there are high biases that seem to migrate northward by July. Though we note this bias feature, the main result is reasonable spatial consistency between the satellite product and the model results. Future work should explore this evaluation further.

Table 2B-1. Summary of 12km resolution CONUS CMAQ 2016 model performance statistics for MDA8 O_3 by NOAA climate region, by season and monitoring 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

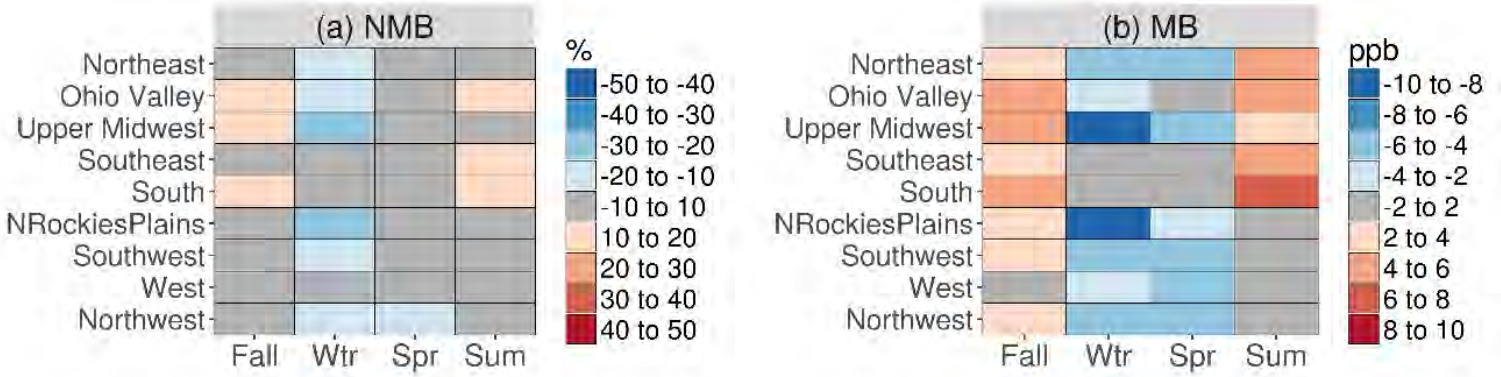


Figure 2B-2. (a) Normalized Mean Bias (%) and (b) Mean Bias (ppb) of maximum daily average 8-hr ozone (MDA8) by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites. In the text, alternative names are used: Ohio Valley is Central, Upper Midewest is EastNorthCentral, and NRockiesPlains is NorthWestCentral.

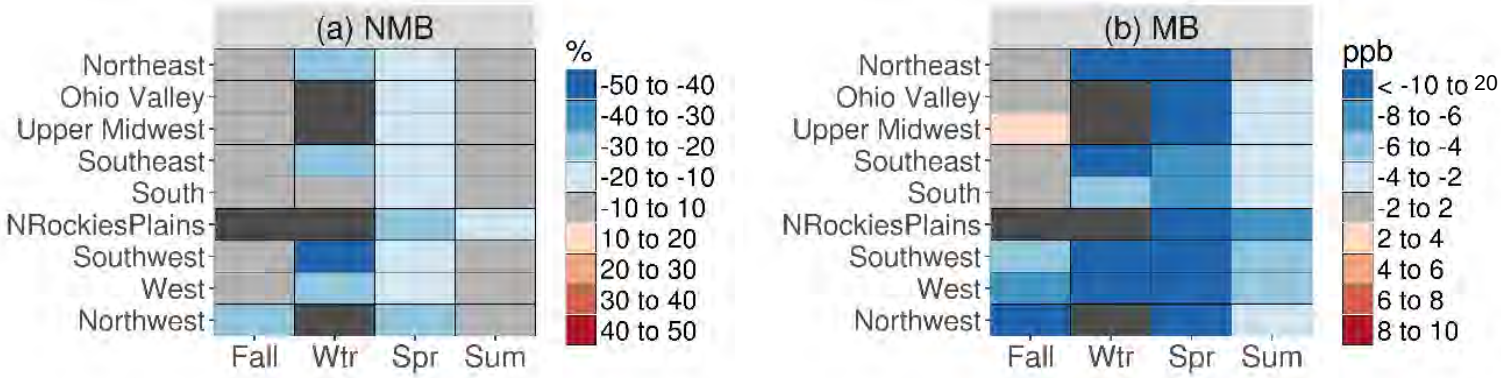


Figure 2B-3. NMB (a) and MB (b) of MDA8 O_3 greater than or equal to 60 ppb from the 12km resolution CONUS simulation by NOAA climate region (y-axis) and by season (x-axis) at AQS monitoring sites. Dark grey cells indicate missing values (i.e., no monitored days with MDA8 >= 60 ppb in that region). In the text, alternative names are used: Ohio Valley is Central, Upper Midewest is EastNorthCentral, and NRockiesPlains is NorthWestCentral.

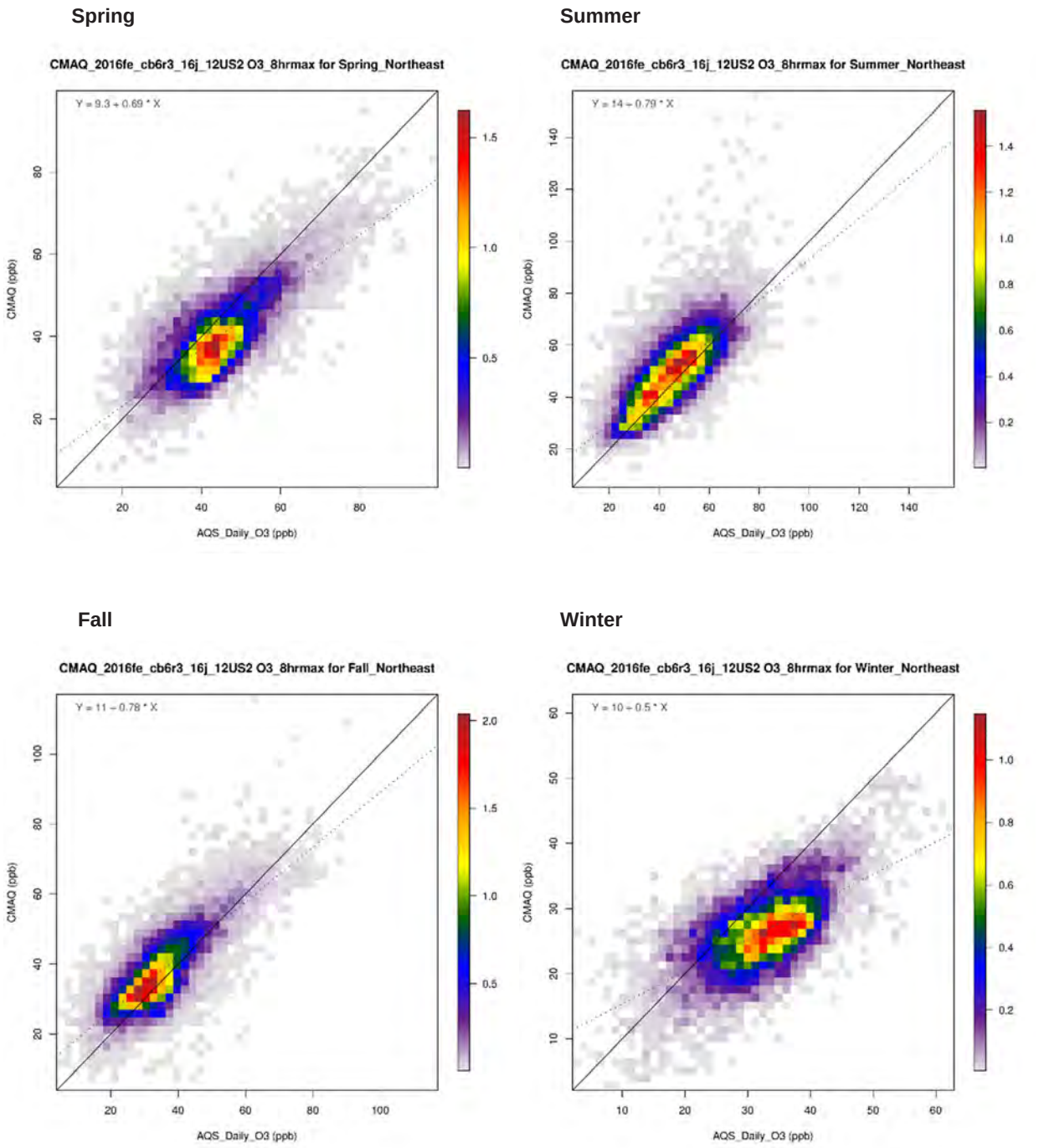


Figure 2B-4. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the Northeast region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

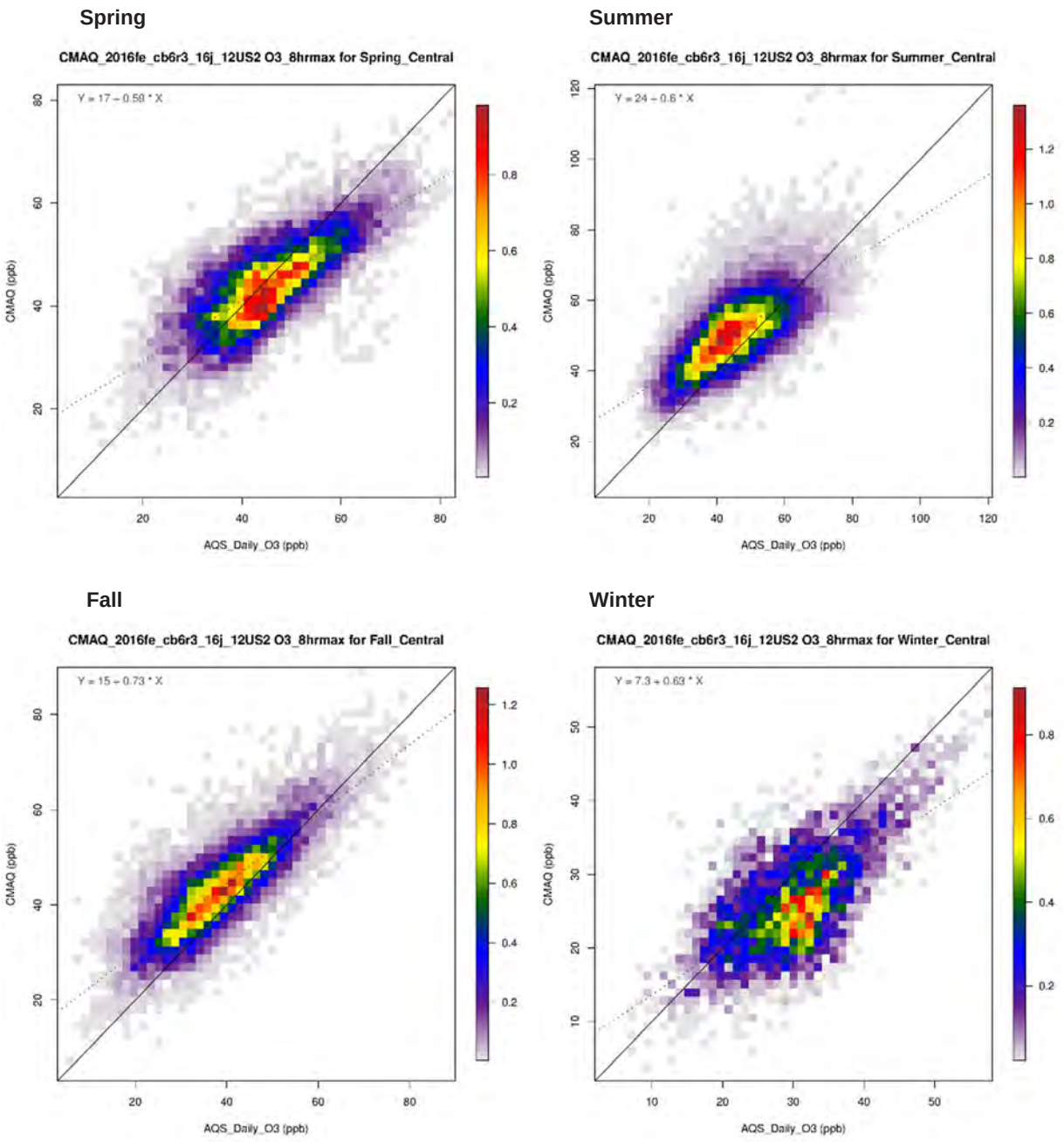


Figure 2B-5. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the Central region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

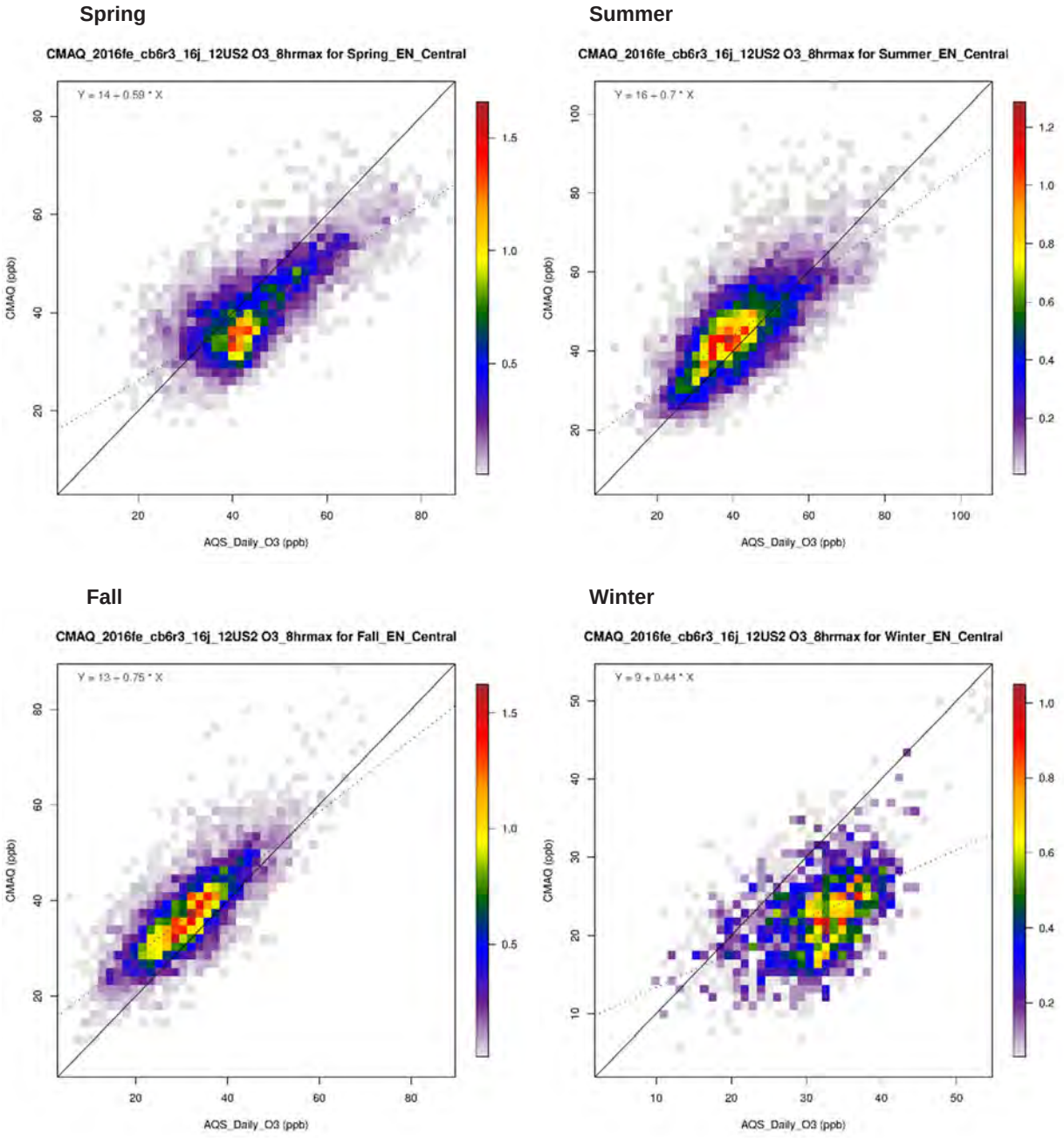


Figure 2B-6. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the EastNorthCentral region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

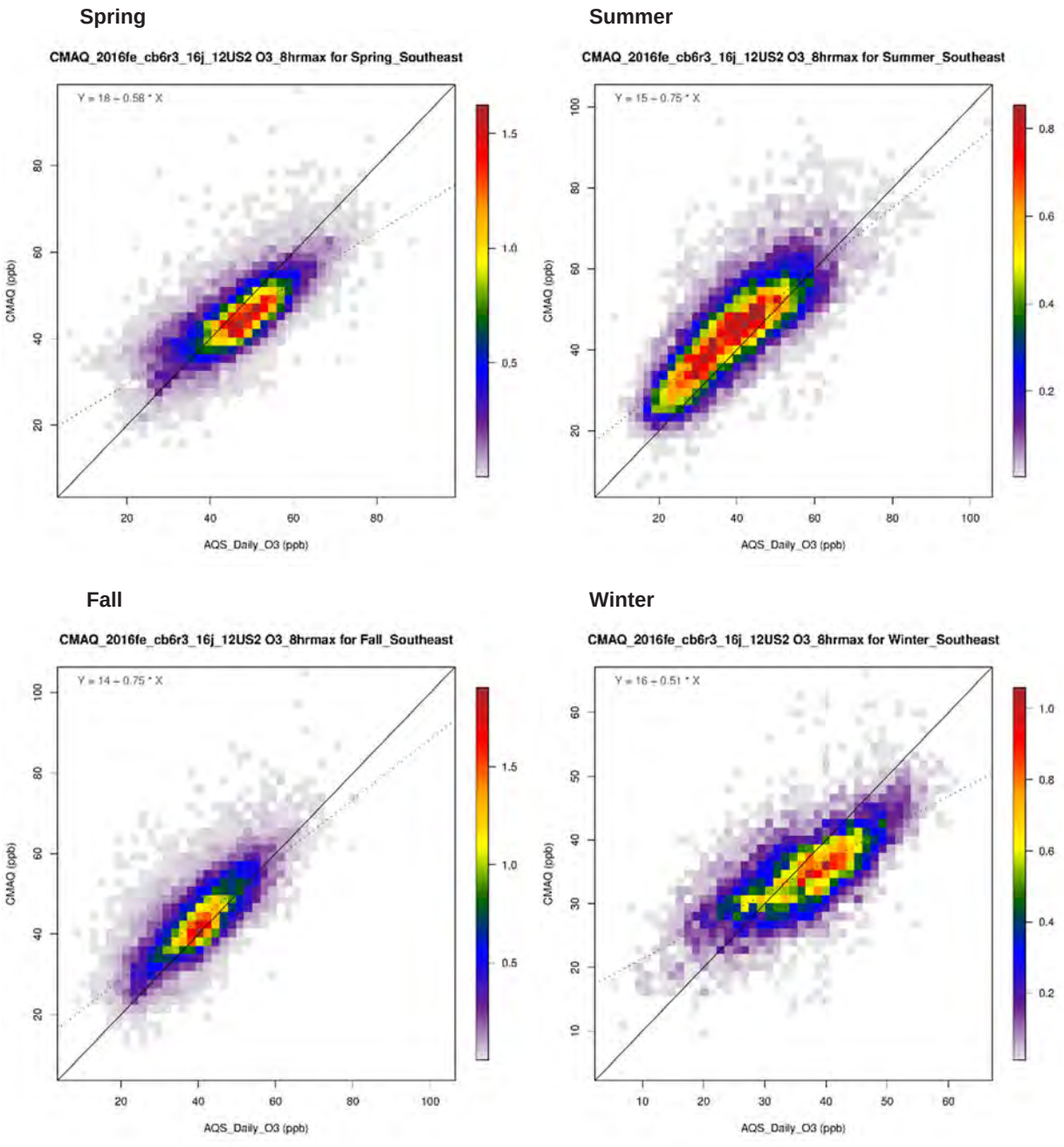


Figure 2B-7. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the Southeast region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

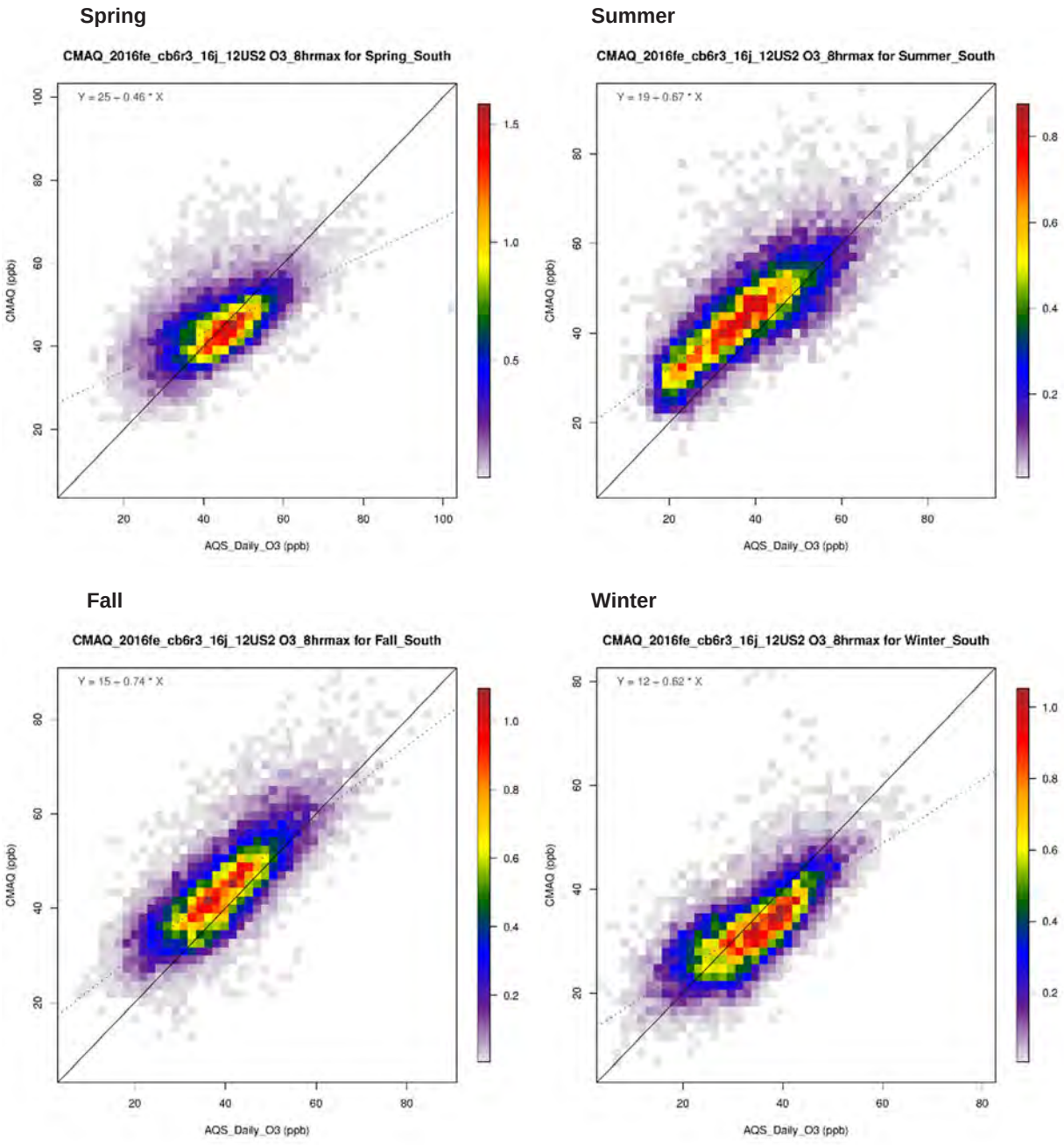


Figure 2B-8. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the South region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

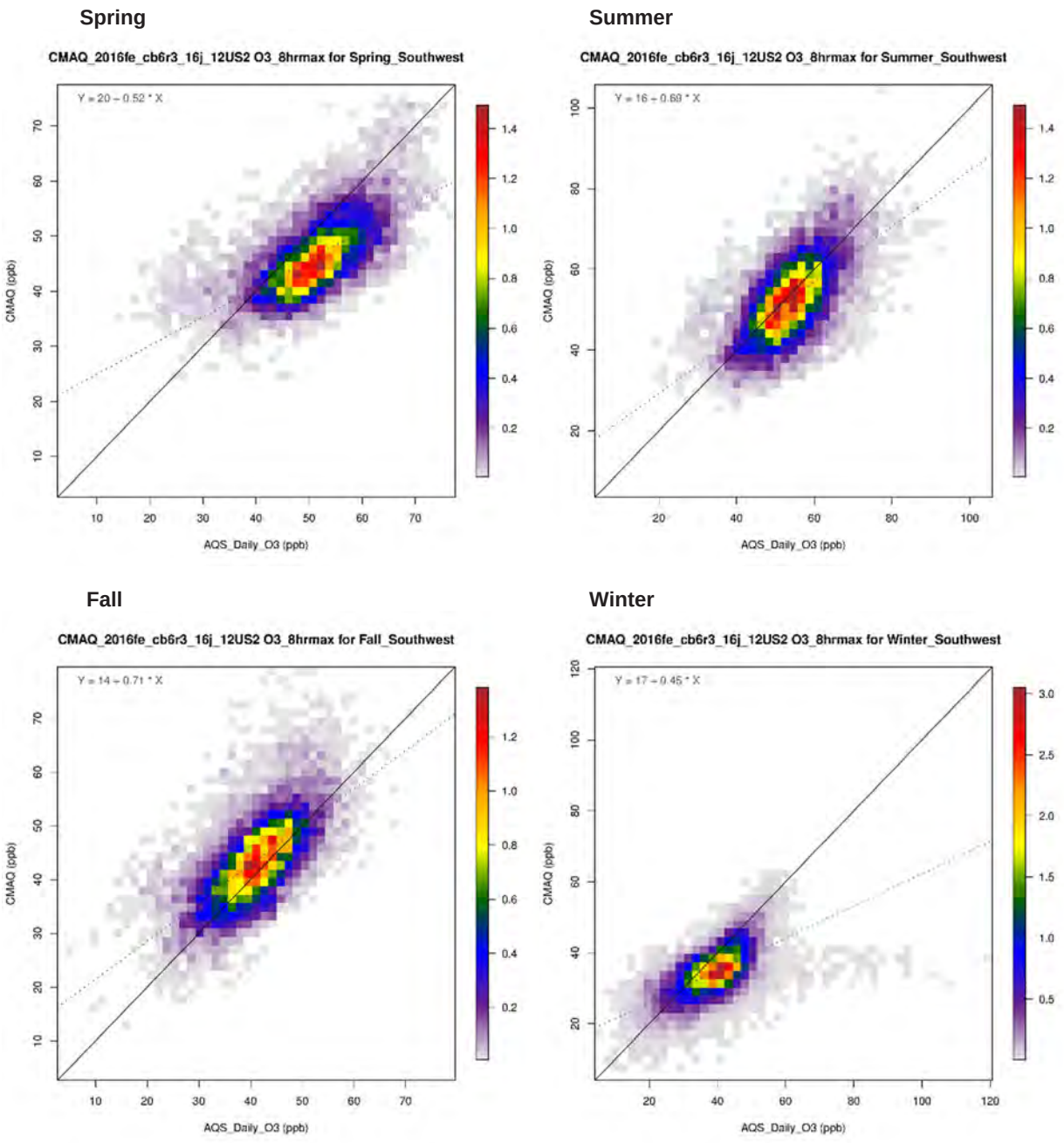


Figure 2B-9. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the Southwest region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

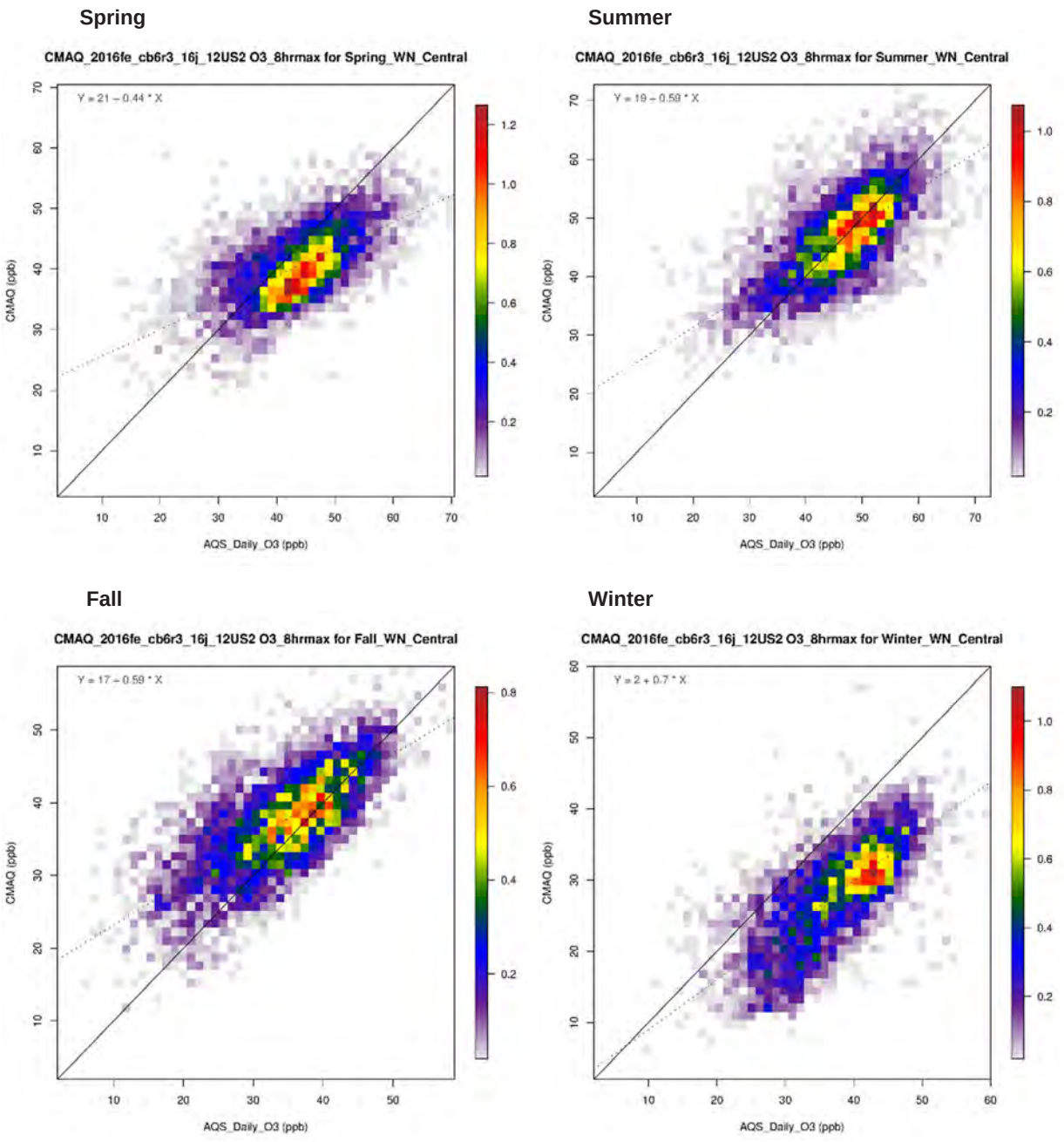


Figure 2B-10. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the WestNorthCentral region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

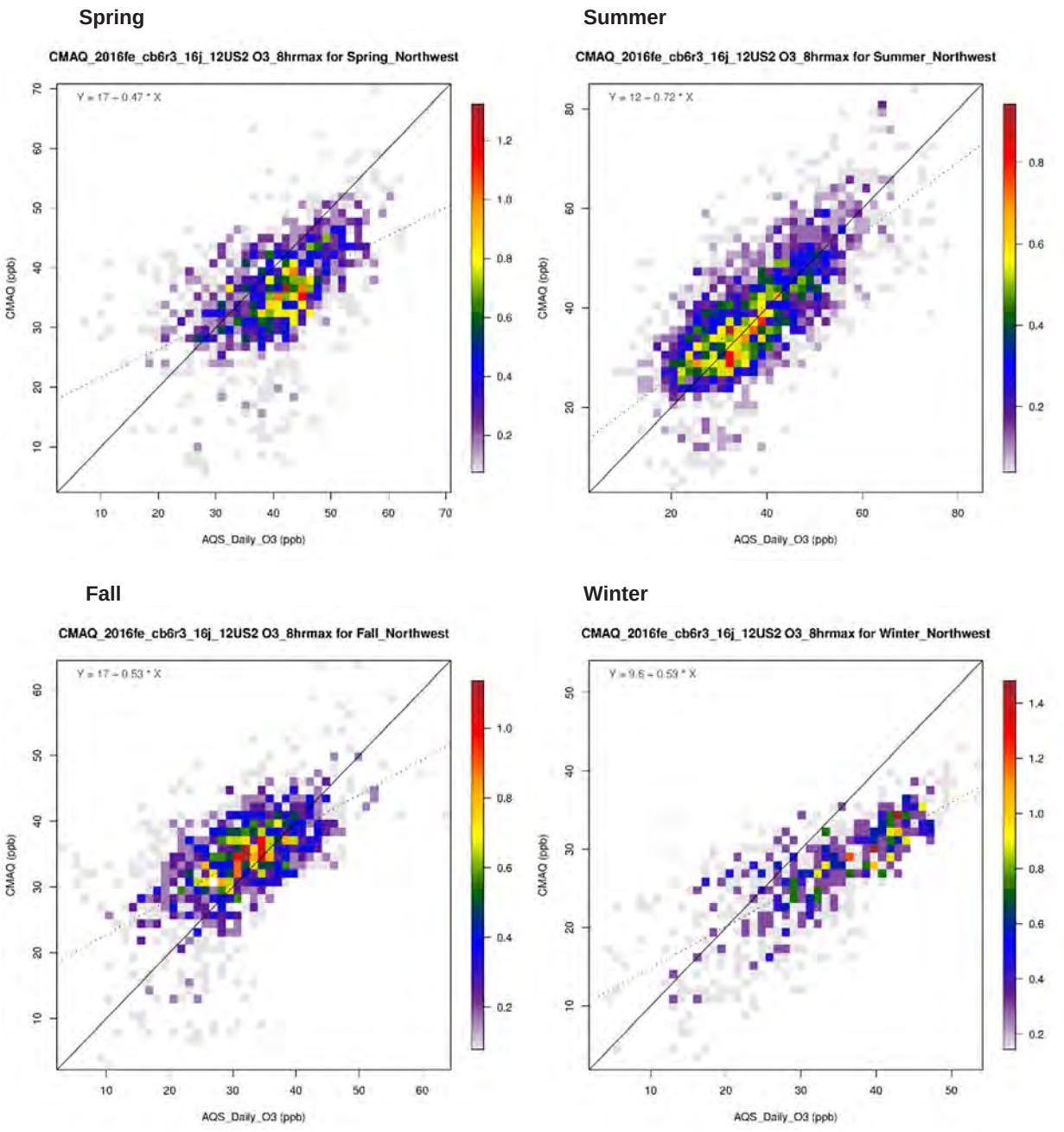


Figure 2B-11. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the Northwest region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

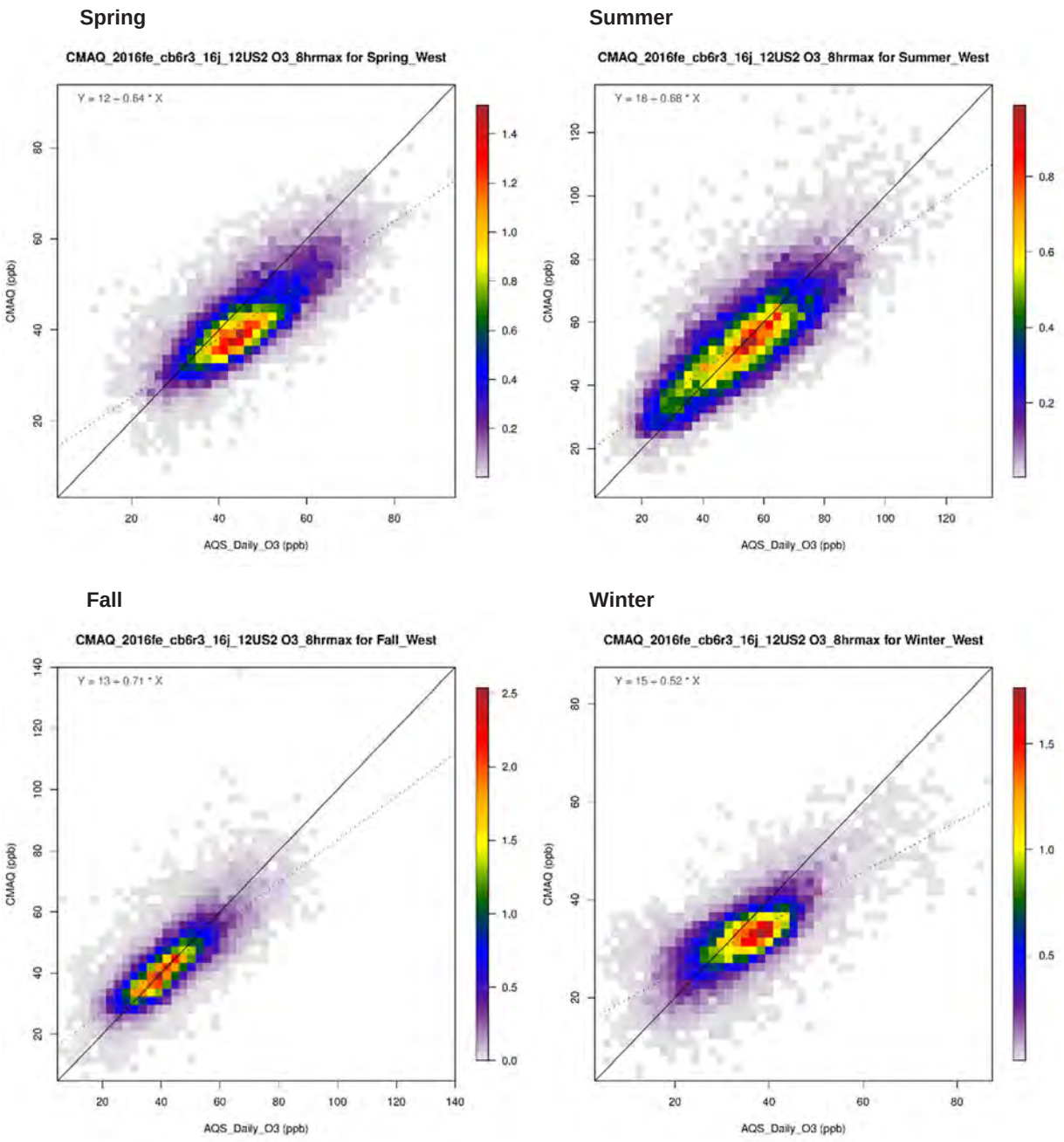


Figure 2B-12. Density scatter plots of observed versus predicted MDA8 O_3 from the 12km resolution CONUS simulation for the West region by season. Each plot has a separate scale that is shared for the x and y axes. The dashed line represents the best fit linear regression line.

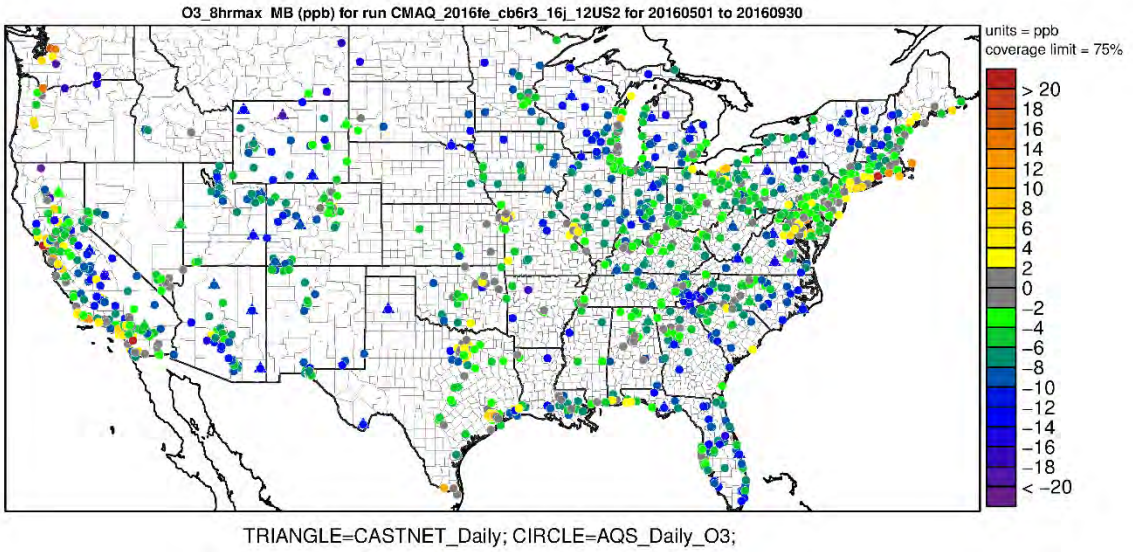


Figure 2B-13. Mean Bias (ppb) from the 12km resolution CONUS simulation of MDA8 O_3 greater than or equal to 60 ppb over the period May through September 2016 at AQS and CASTNET monitoring sites in the continental U.S. modeling domain.

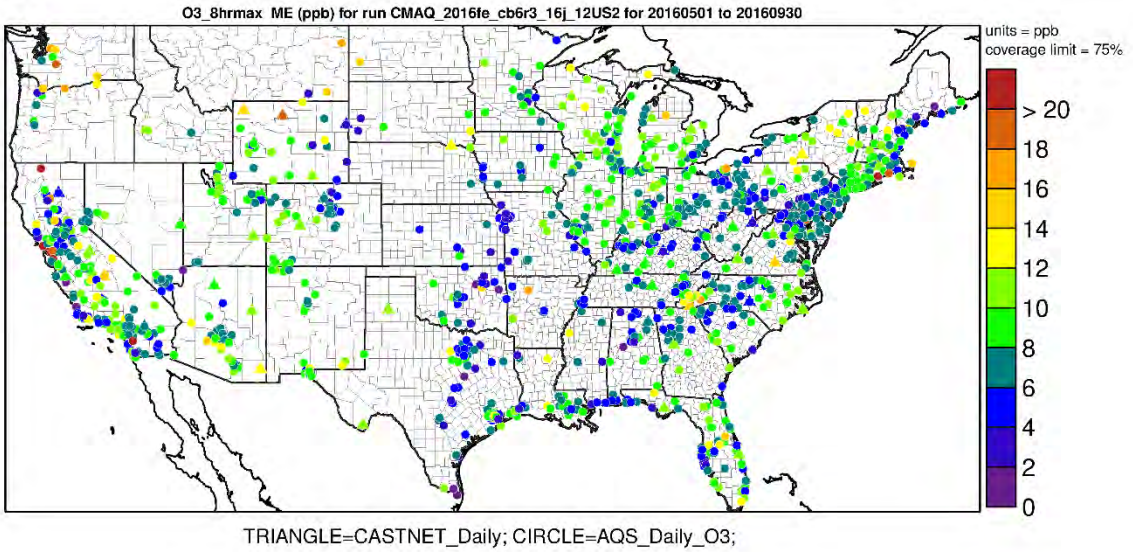


Figure 2B-14. Mean Error (ppb) from the 12km resolution CONUS simulation of MDA8 O_3 greater than or equal to 60 ppb over the period May through September 2016 at AQS and CASTNET monitoring sites in the continental U.S. modeling domain.

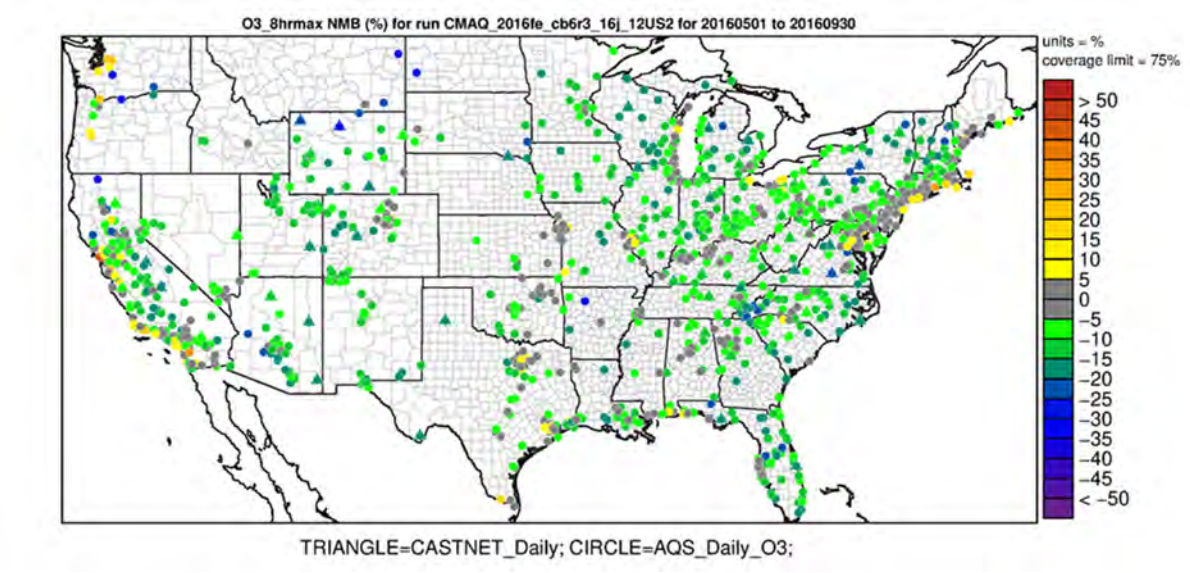


Figure 2B-15. NMB (%) from the 12km resolution CONUS simulation of MDA8 O_3 greater than or equal to 60 ppb over the period May through September 2016 at AQS and CASTNET monitoring sites in the continental U.S. modeling domain.

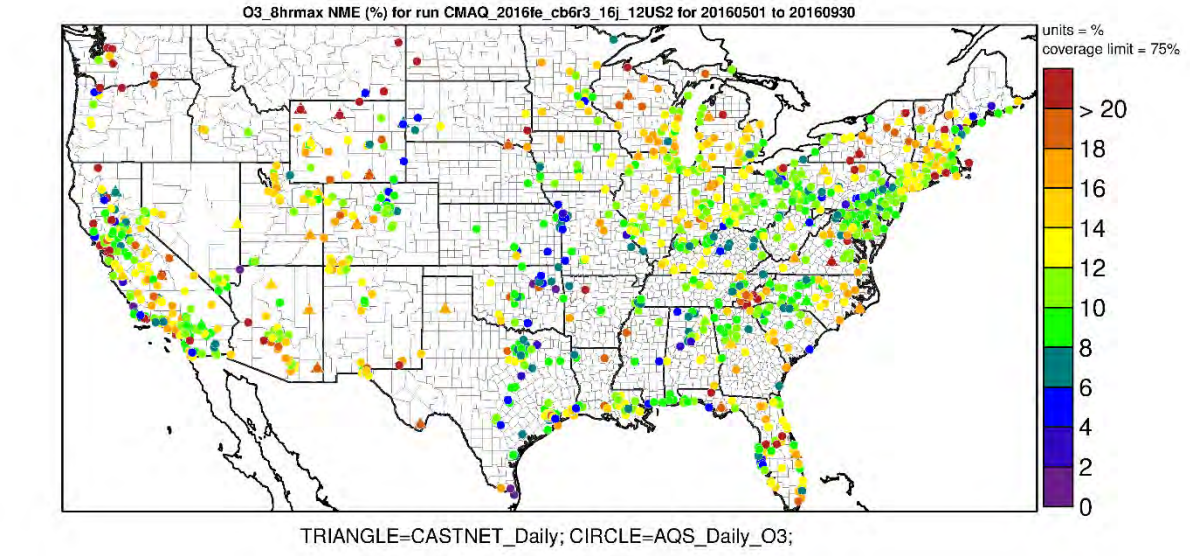


Figure 2B-16. NME (%) from the 12km resolution CONUS simulation of MDA8 O_3 greater than or equal to 60 ppb over the period May through September 2016 at AQS and CASTNET monitoring sites in the continental U.S. modeling domain.

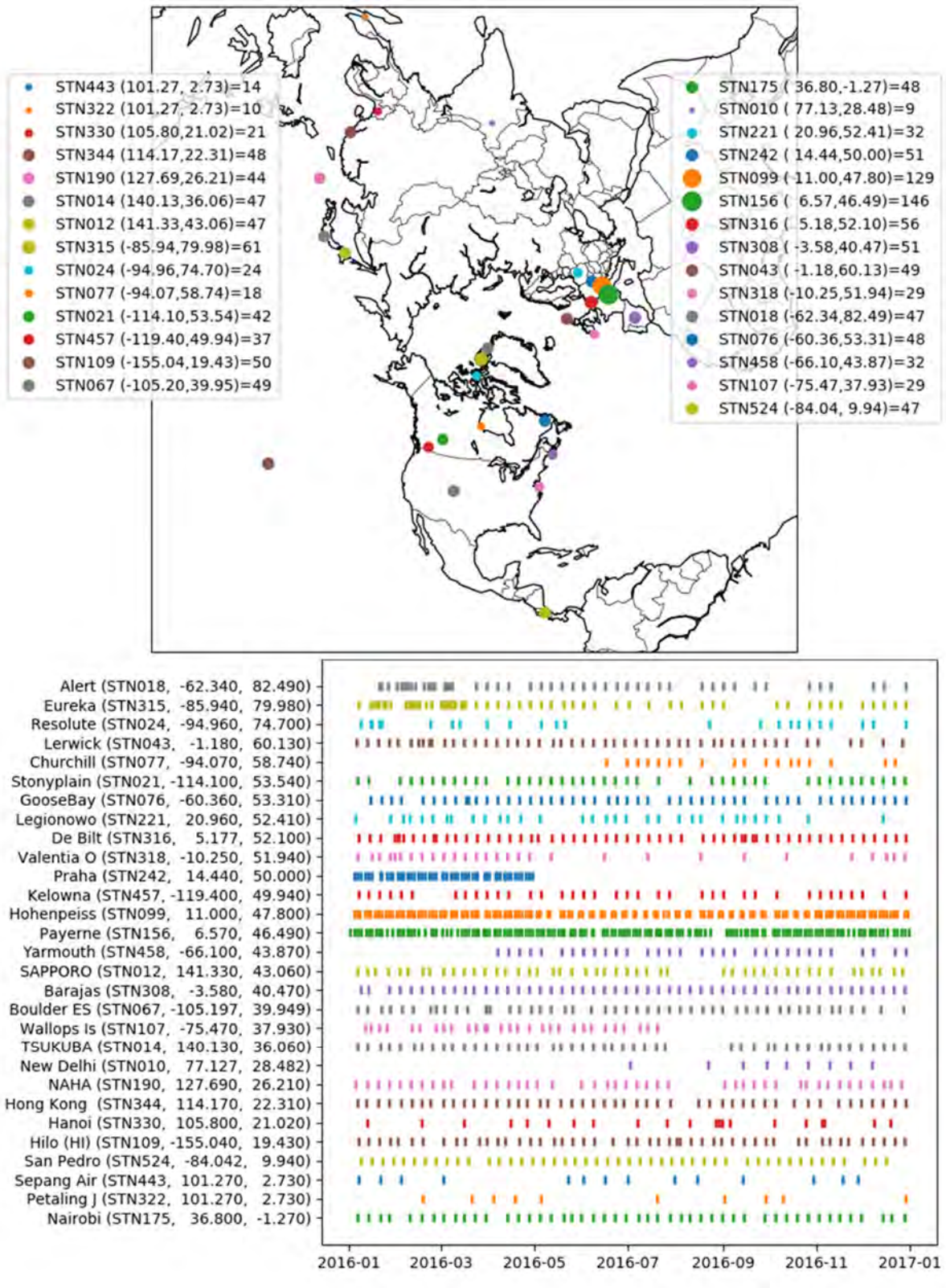


Figure 2B-17. WOUDC sonde locations and sampling frequency used in evaluation of hemispheric model simulation.

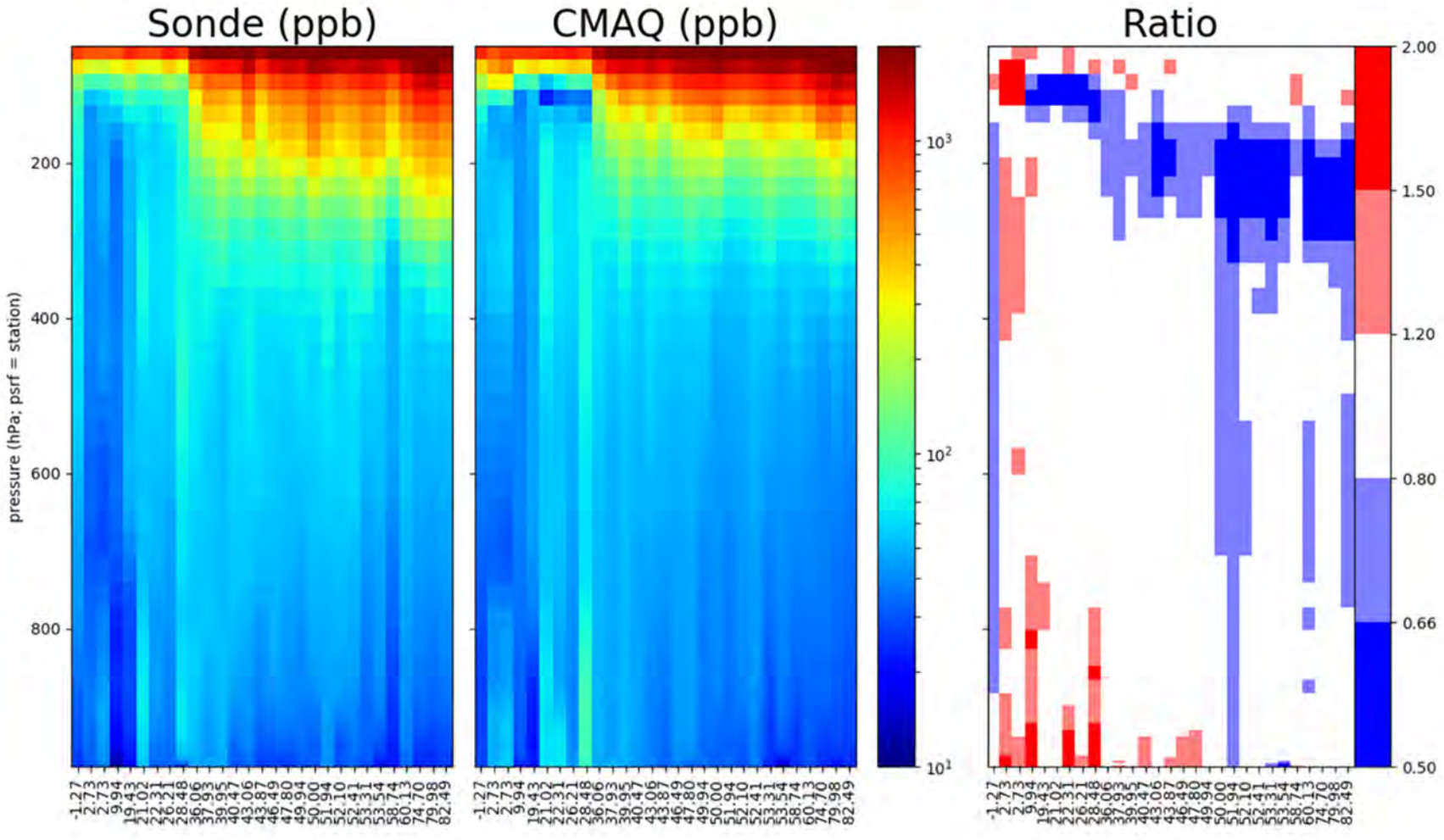


Figure 2B-18. WOUDC sonde releases averaged by release location over 2016; observations (left), predictions from the hemispheric CMAQ simulation (middle), ratio (right). Observations are ordered with increasing latitude (South to North).

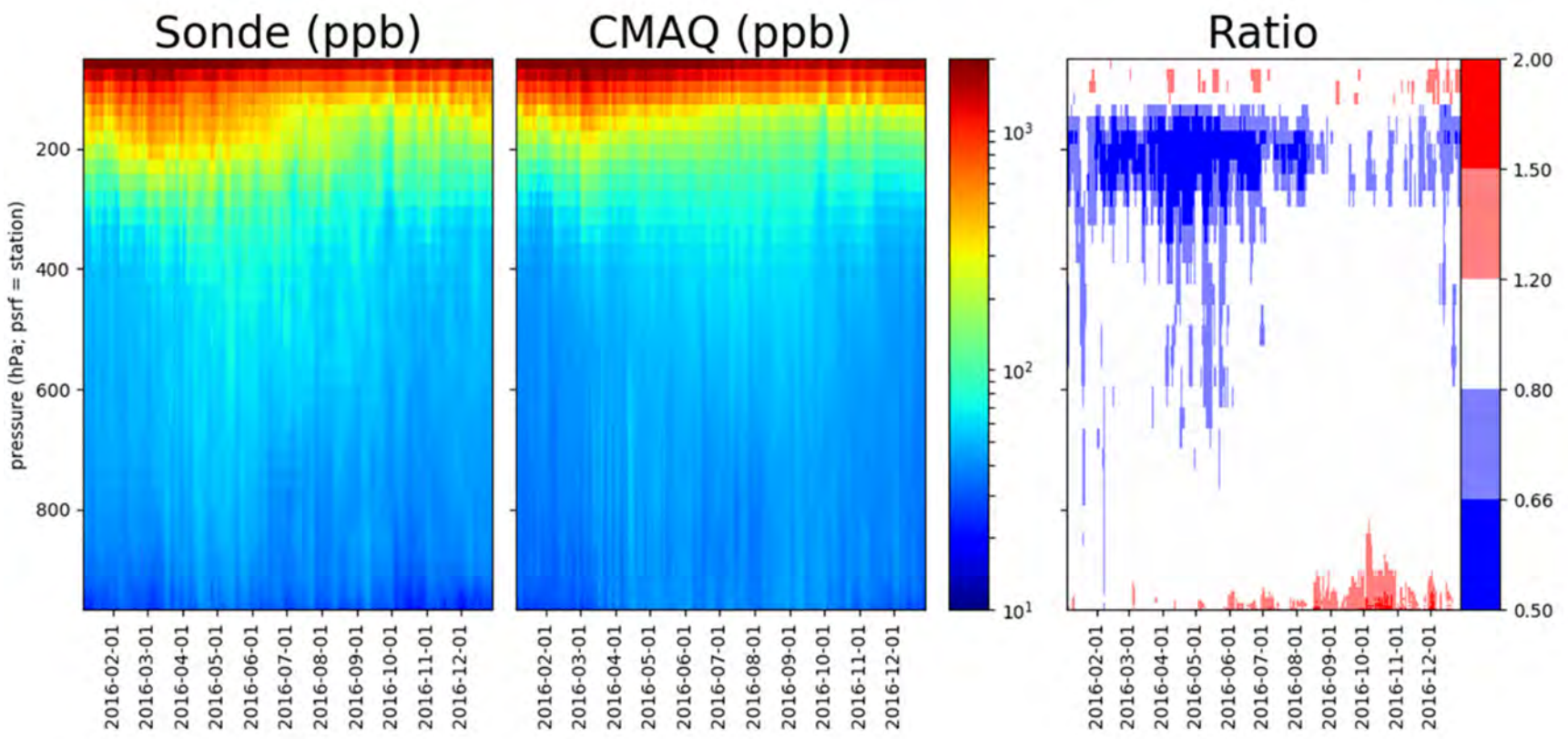


Figure 2B-19. WOUDC sonde releases averaged by day with a 20-point moving average; observations (left), predictions from the hemispheric CMAQ simulation (middle), ratio (right).

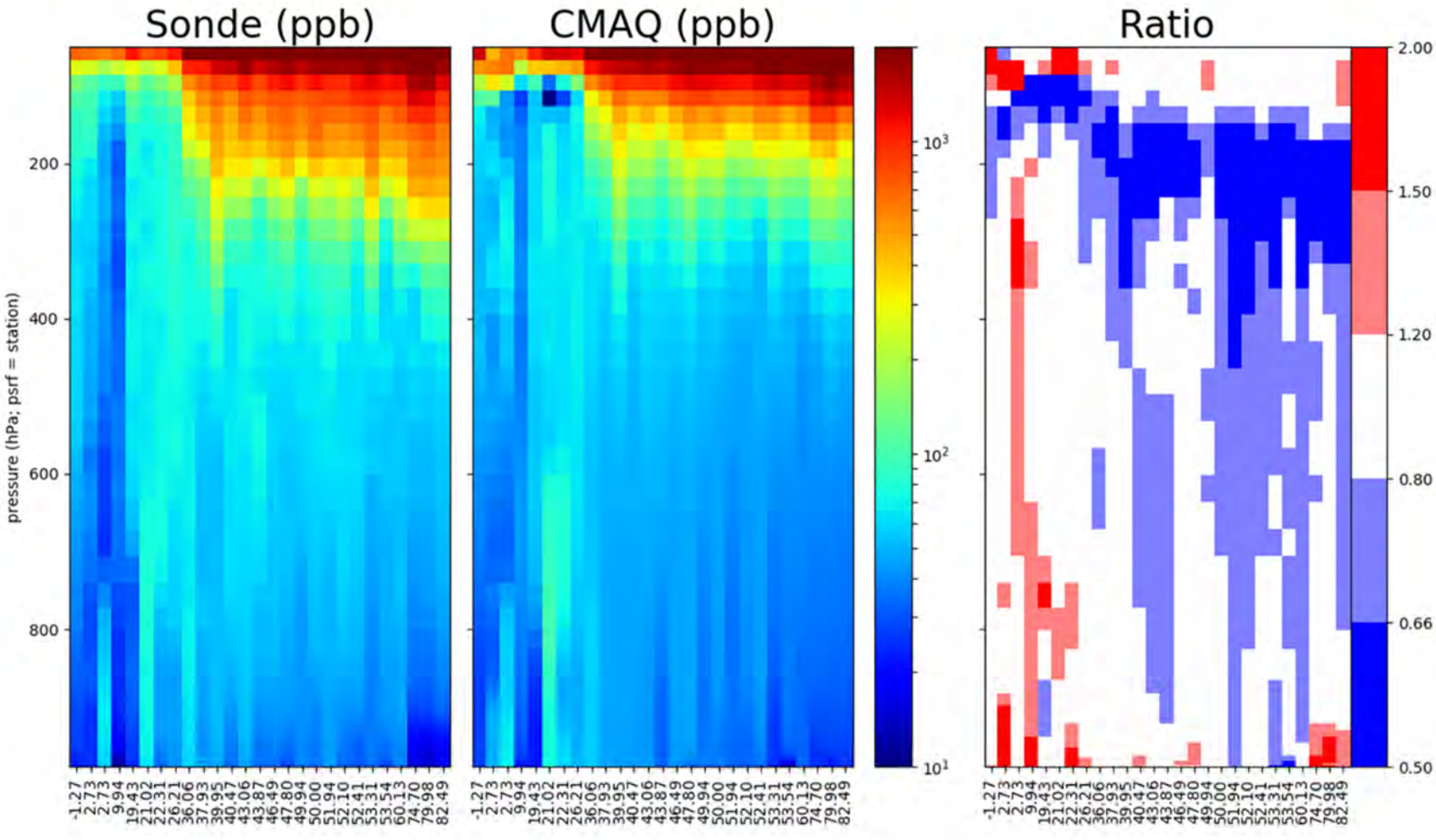


Figure 2B-20. WOUDC sonde releases averaged by release location over March, April, May in 2016; observations (left), predictions from the hemispheric CMAQ simulation (middle), ratio (right).

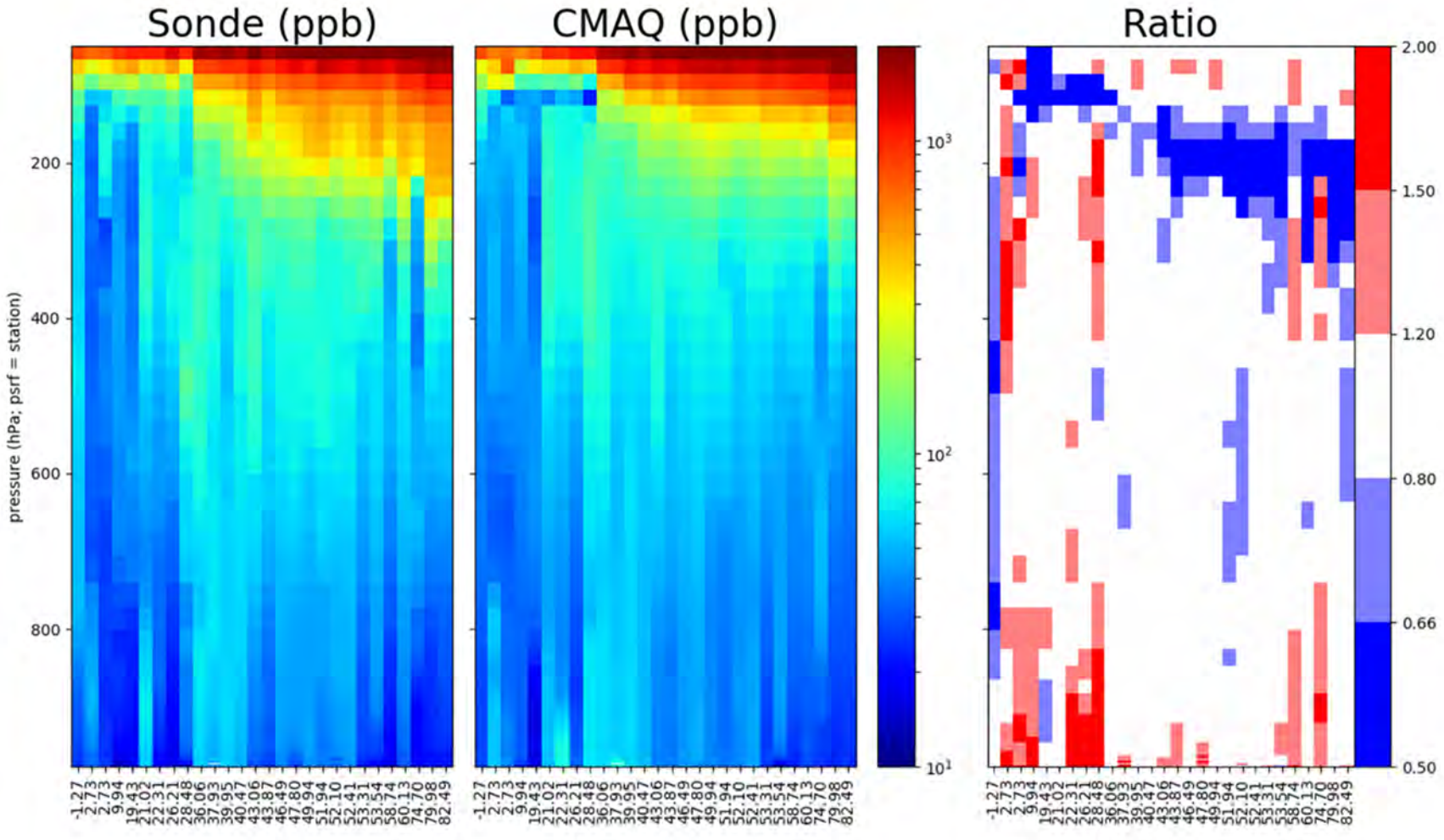


Figure 2B-21. WOUDC sonde releases averaged by release location over June, July, August in 2016; observations (left), predictions from the hemispheric CMAQ simulation (middle), ratio (right).

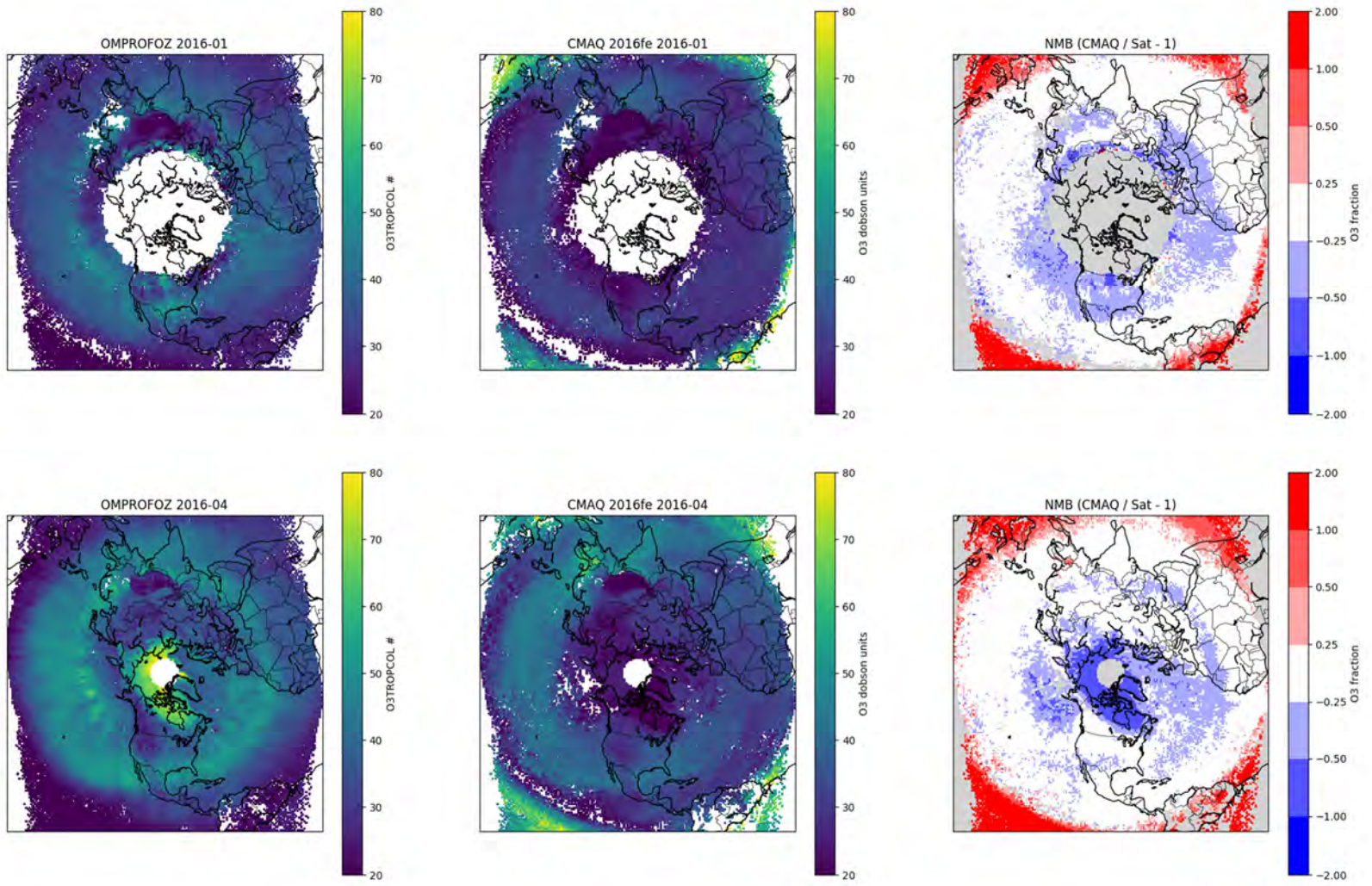


Figure 2B-22. OMI O_3 (OMPROFOZ v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios (right) of vertical column densities for January (top) and April (bottom).

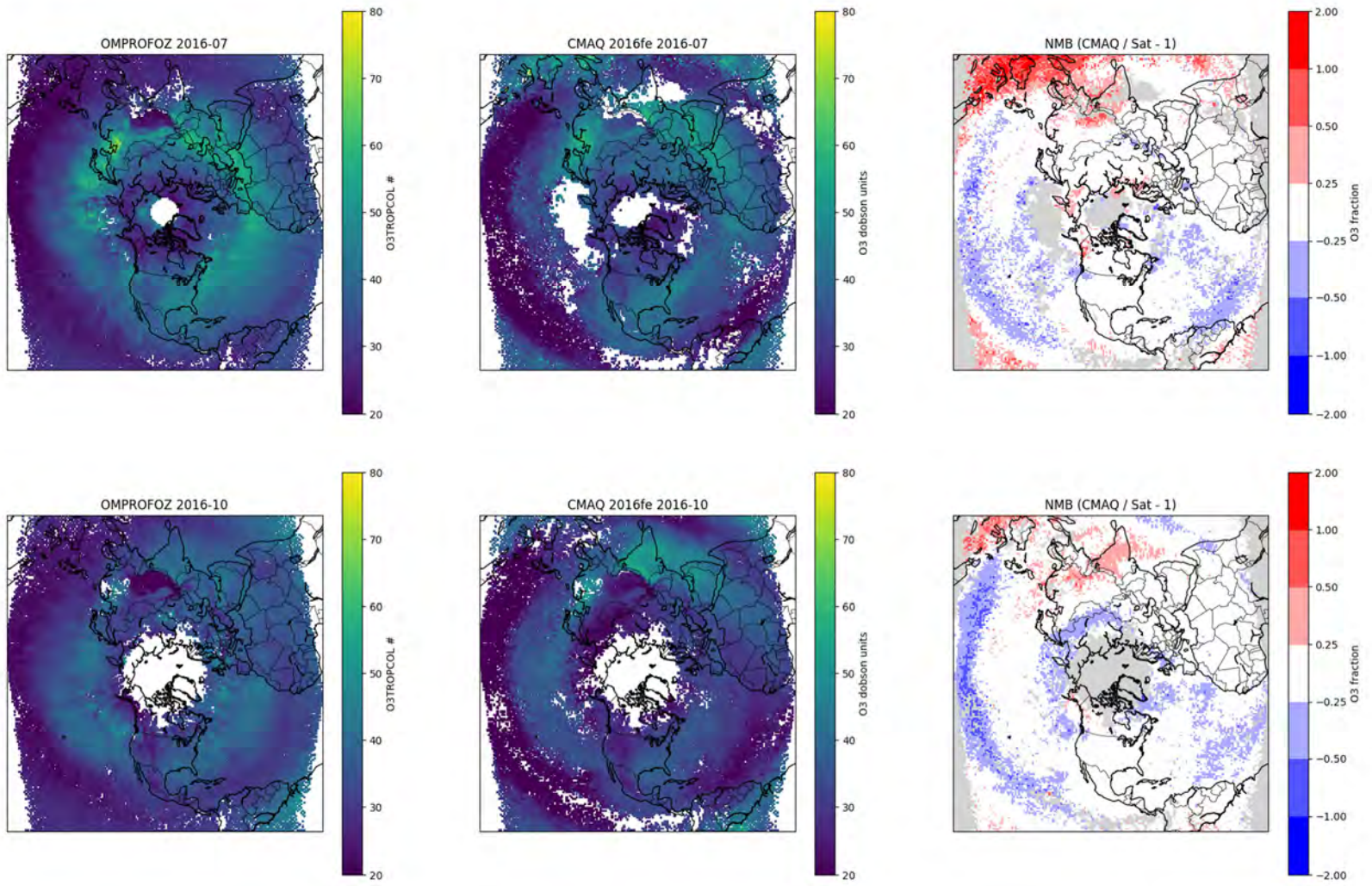


Figure 2B-23. OMI O_3 (OMPROFOZ v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios (right) of vertical column densities for July (top), and October (bottom).

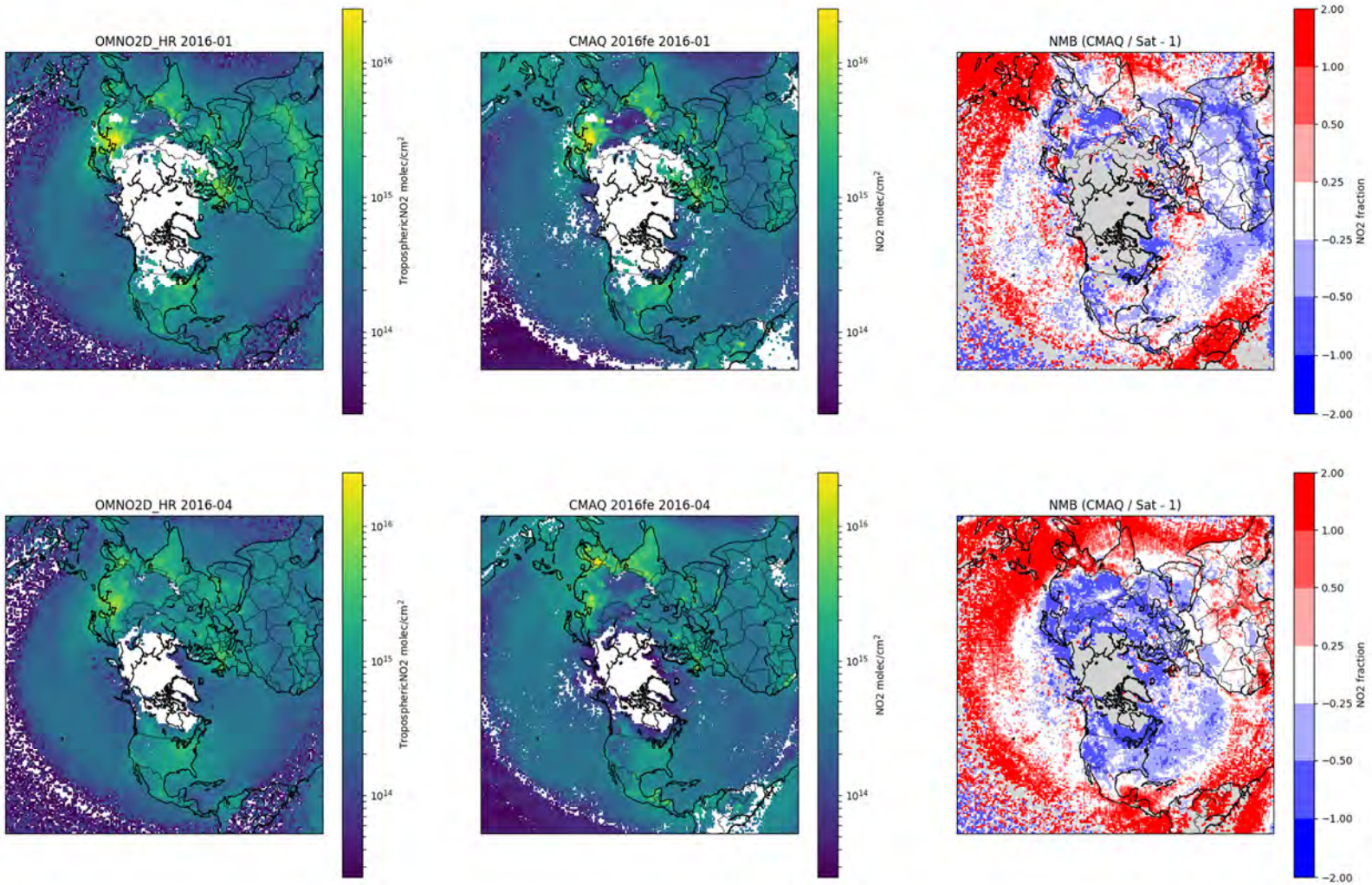


Figure 2B-24. OMI Nitrogen Dioxide (OMNO2D_HR v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios (right) of vertical column densities for January (top) and April (bottom).

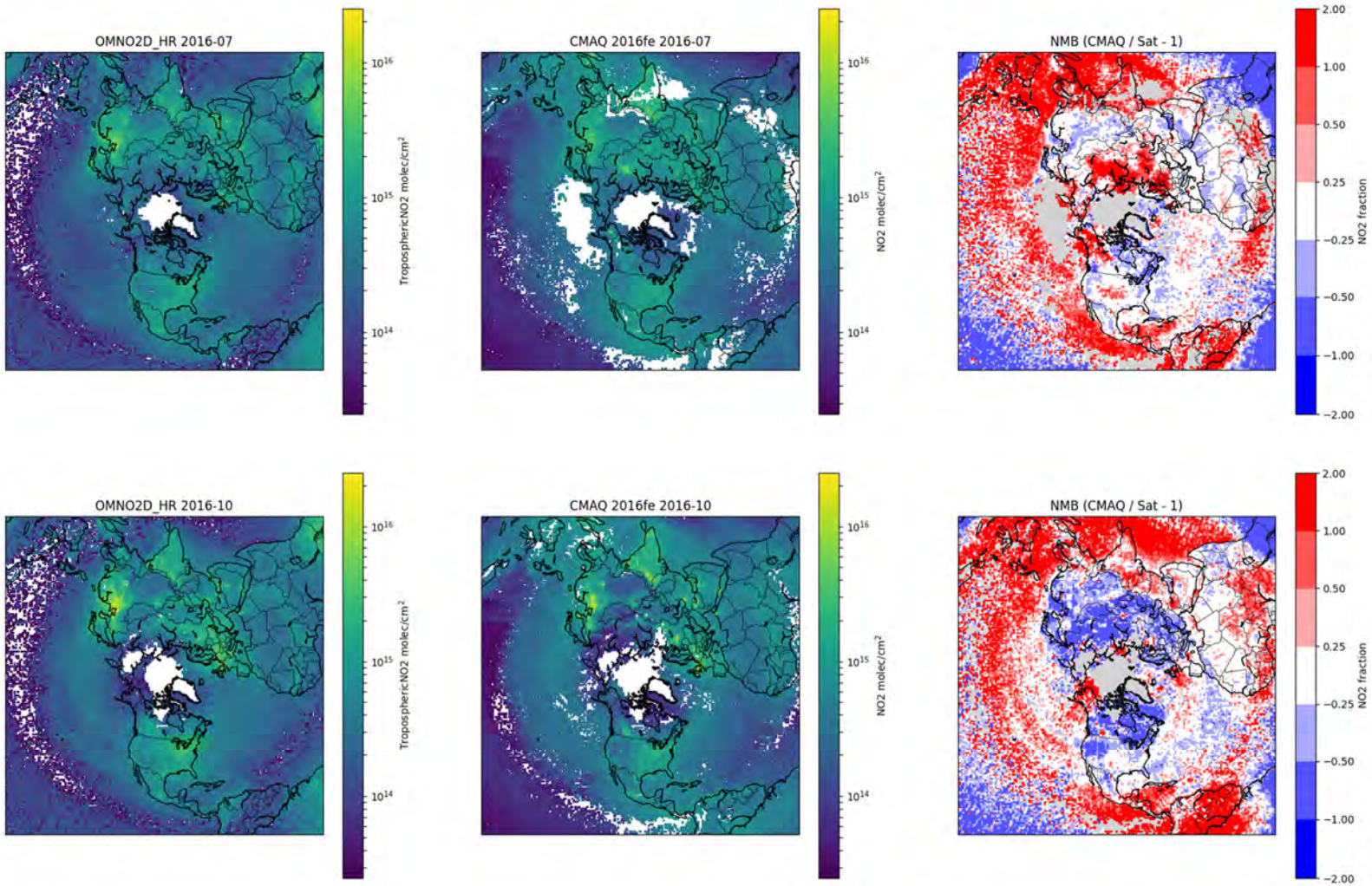


Figure 2B-25. OMI Nitrogen Dioxide (OMNO2D_HR v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios (right) of vertical column densities for July (top) and and October (bottom).

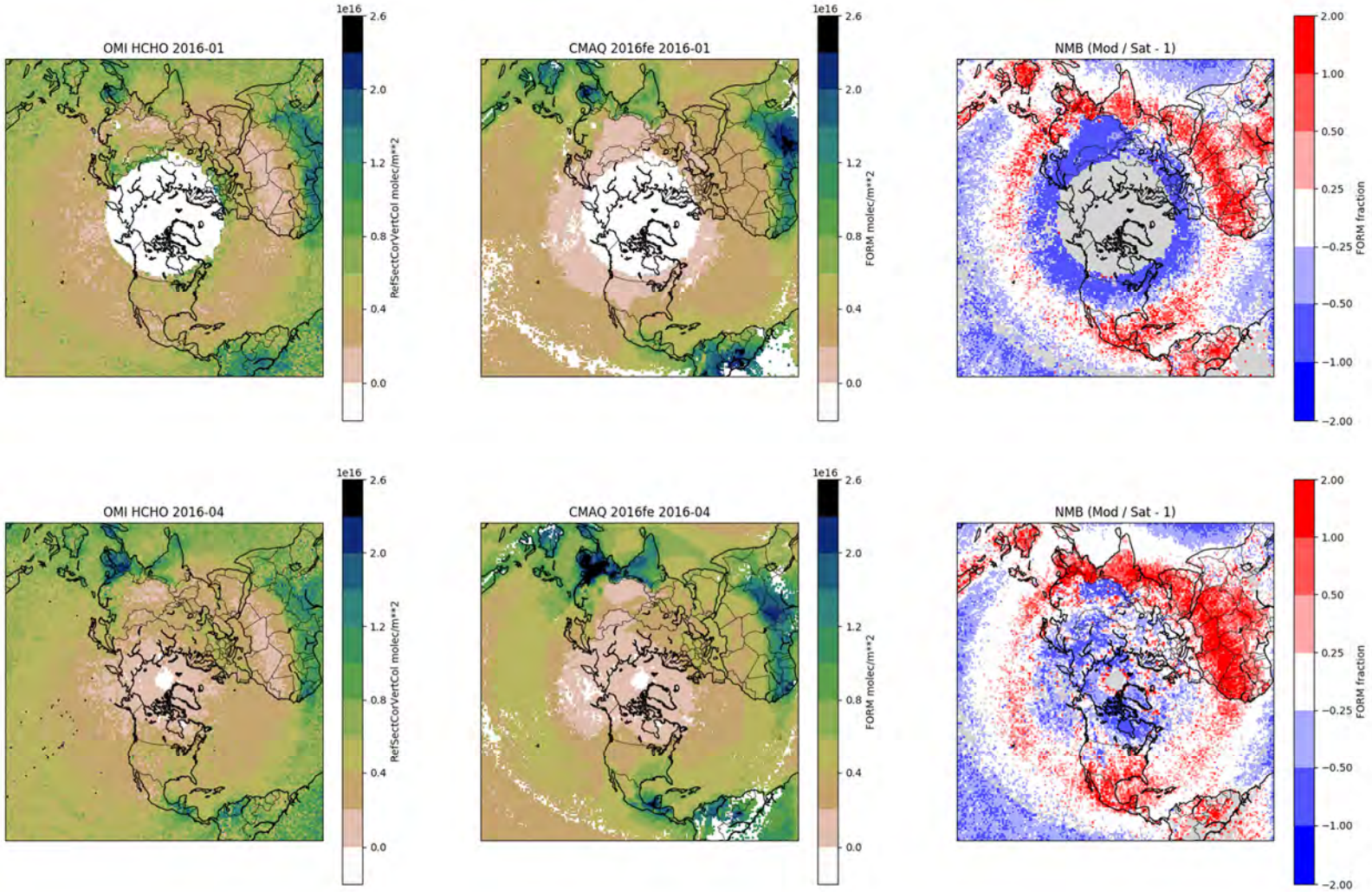


Figure 2B-26. OMI Formaldehyde (OMHCHO v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios (right) of vertical column densities for January (top) and April (bottom).

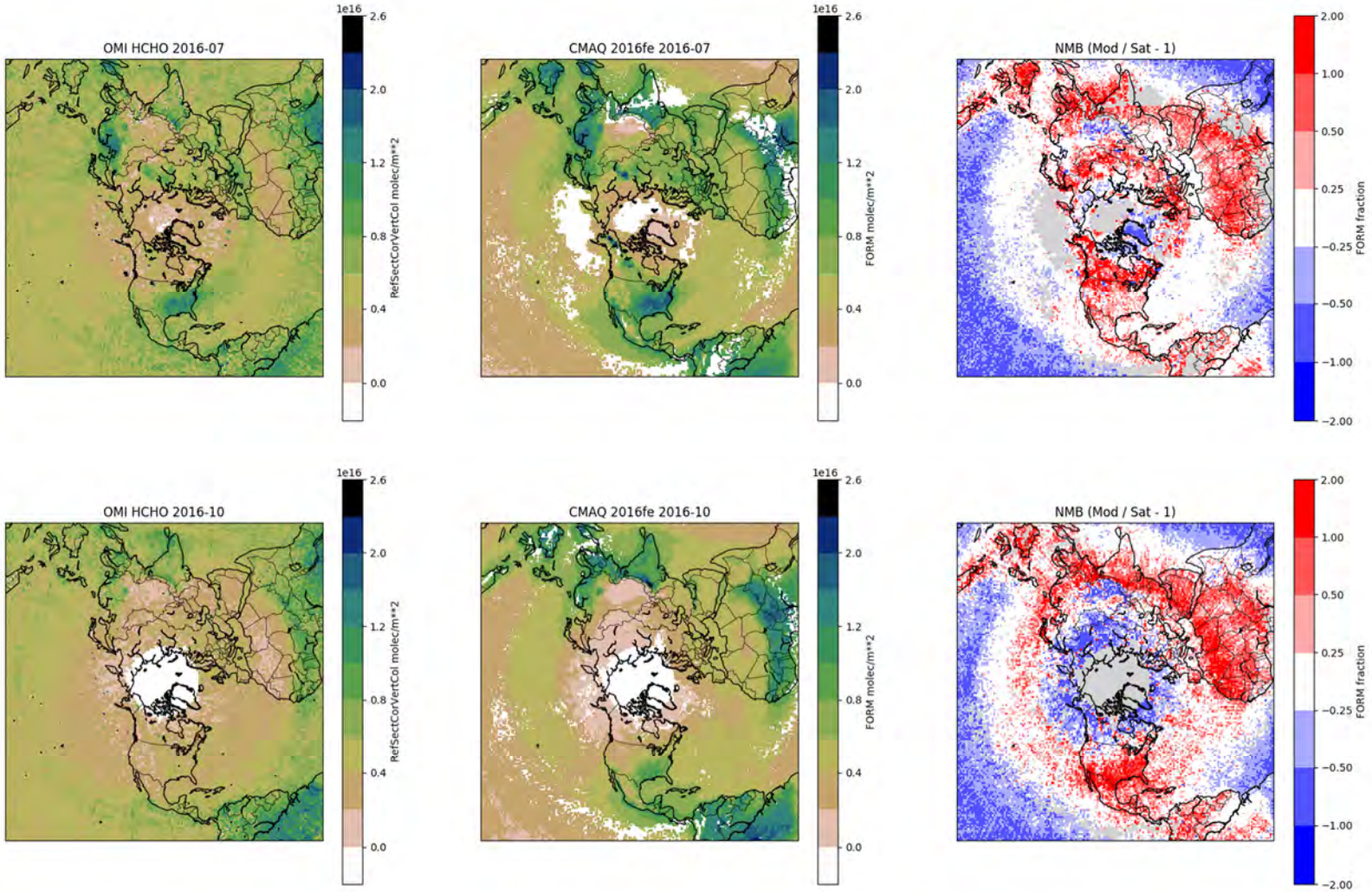


Figure 2B-27. OMI Formaldehyde (OMHCHO v003, left) compared to simulated (hemispheric CMAQ simulation, center), and ratios (right) of vertical column densities for July (top), and October (bottom).

2B.3 INTERNATIONAL CONTRIBUTIONS

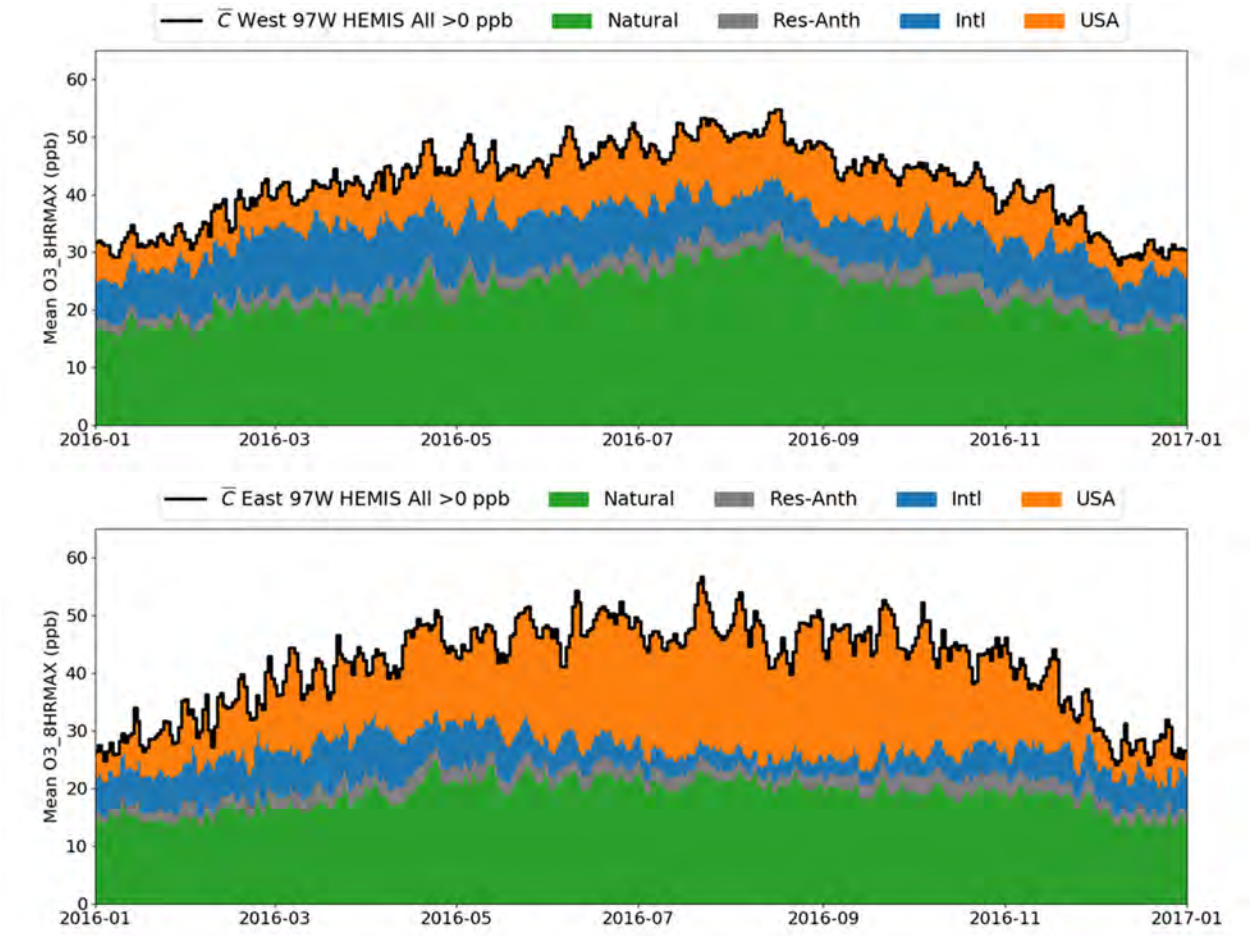
This section characterizes the components of predicted international anthropogenic contributions to local O_3 concentrations and the sensitivities to model resolution. The main characterization of predicted O_3 contributions focused on results, based on simulations at a 12 km grid cell resolution, that separated Natural, International, and USA contributions to O_3. 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 O_3 (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 O_3 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

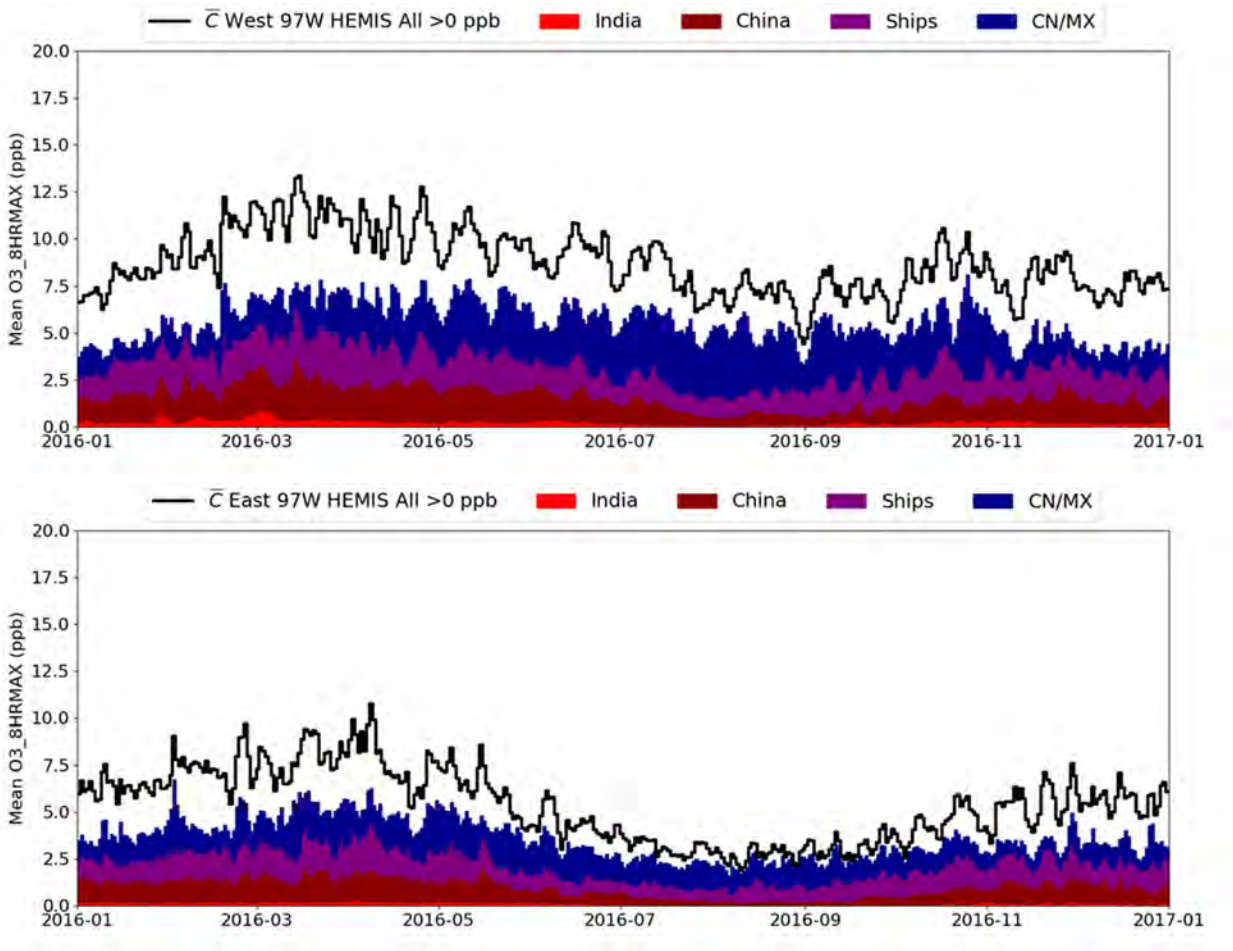
Canada/Mexico are more important will have a later peak of International than those influenced by the long-range components (e.g., India, China). The 108 km results cannot resolve the border well and will likely not fully capture the "near-border" effect.

Figure 2B-30 demonstrates the effect of International contribution on seasonality. Figure 2B-30 shows the West broken out into high-elevation, near-border, and Low/Interior sites. The near-border areas have a larger Canada/Mexico component. The combination of long-range sources and Canada/Mexico create a peak International contribution at near-border sites that is one to two months later than at high-elevation or Low/Interior sites. Note that "near-border" sites are not well resolved by the 108 km simulations.



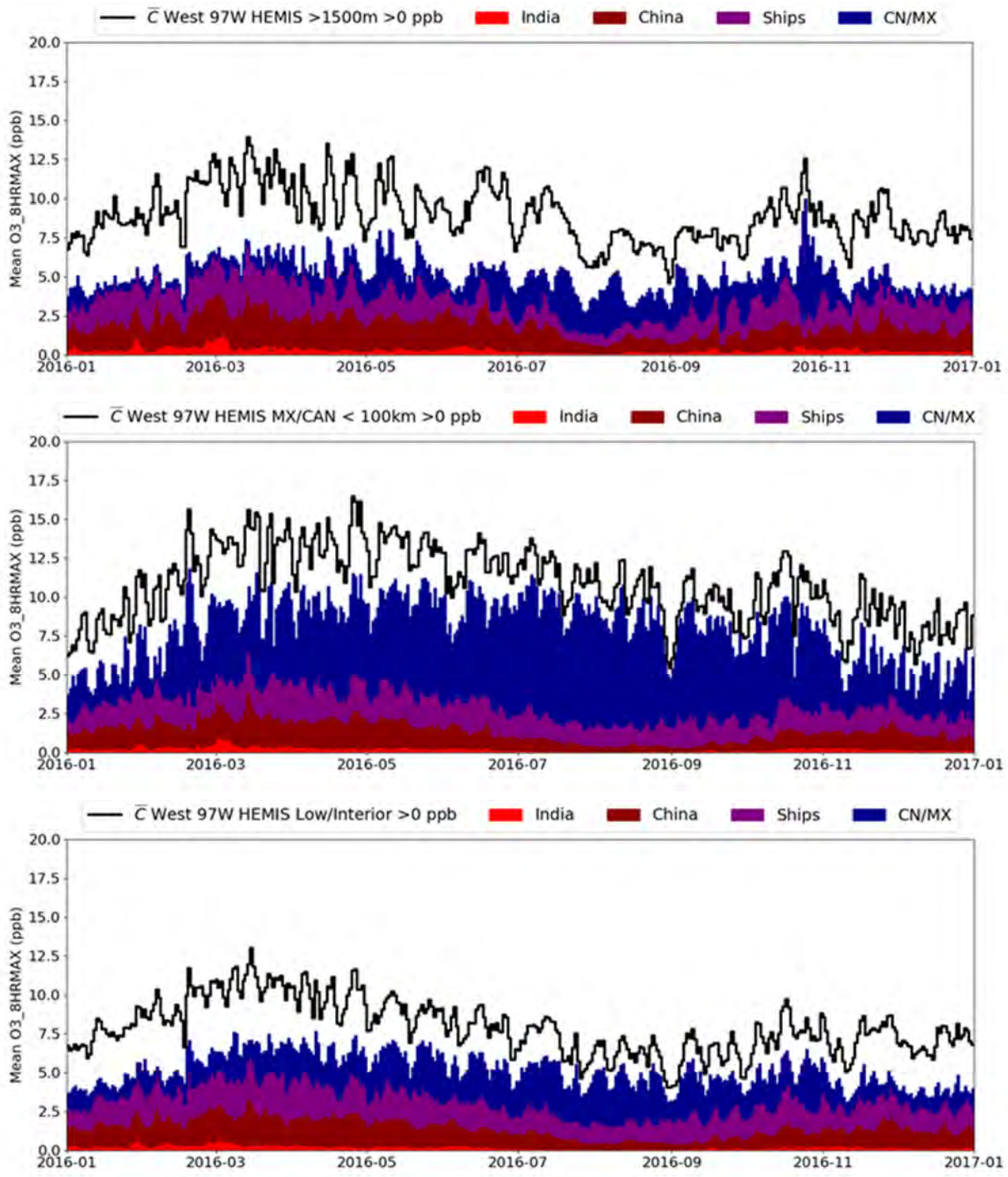
Average across all grid cells derived as $\overline{C} = \frac{1}{N_x}\sum_x C_x$

Figure 2B-28. Total predicted MDA8 O_3 and contributions (see legend) over time in the West (top), and all East (bottom) averaged over all grid cells and days in the U.S.



Average across all grid cells derived as $\overline{\boldsymbol{C}} = \frac{\mathbf{1}}{\boldsymbol{N_x}} \sum_{\boldsymbol{x}} \boldsymbol{C_x}$

Figure 2B-29. International contribution (black line) to predicted MDA8 O_3 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.



Average across all grid cells derived as $\overline{C} = \frac{1}{N_x}\sum_x C_x$

Figure 2B-30. International contribution (black line) to predicted MDA8 O_3 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 O_3 exposures during exercise. In these studies, the magnitude or severity of the respiratory effects induced by O_3 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 O_3 induced change in forced expiratory volume in one second (FEV_1) in 6.6 to 8-hour controlled human exposure studies (involving quasi-continuous or intermittent exercise). The FEV_1 values presented are derived by subtracting the percent changes in mean FEV_1 in response to filtered air exposure with exercise from the corresponding percent changes in FEV_1 in response to O_3 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 O_3. 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 O_3 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 O_3 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 m^2. Values reflect the study mean EVR across the six exercise periods except for R11, as described below.

Table 3A-1. Cross-study comparison of mean O_3-induced FEV_1 decrements in 6.6 to 8-hour controlled human exposure studies (that include periods of exercise).

Exposure Design[C]		Ref[D]	EVR[E] (L/min -m^2)	**ΔFEV$_1$**[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 O_3-induced percent change in FEV_1 at the group mean level, based on subtraction of the filtered air percent change (post-pre exposure) from the O_3 % change in FEV_1. For studies R1, R2, R4, R5 and R9, ΔFEV_1 values were calculated from individual subject data provided by author. ΔFEV_1 values for R3, R6, R12 were calculated from individual subject data in publication; R7, R8, R10, R11, R13 and R14 ΔFEV_1 values were derived from group mean response provided in publication. Statistically significant findings are indicated by asterisk (*). A lack of statistical testing is indicted by (^). Unless indicated otherwise, all studies were in healthy adults.

[B] In addition to ΔFEV_1, 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 O_3 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-m^2).
[15-23] indicates hourly ventilation rate varied from 15-23 L/min-m^2; 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-m^2; 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-m^2; 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 O_3 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.

Table 3A-2. Study-specific details of O_3 exposure protocols for 6.6 to- 8-hour controlled human exposure studies (that include periods of exercise).

Ref[A]	EVR[B] during exercise (L/min-m²)	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 *red=O_3 exposure, black = no exposure (i.e., no facemask) bold =exercise periods, non-bold=rest periods*						
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	$10^{9\text{-}12}$, $11^{7\text{-}11}$, $18^{\#}$, $20^{15\text{-}23}$	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)	Hazucha 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.

$^{9\text{-}12}$ indicates the study protocol varied the hourly ventilation rate from 9-12 L/min-m² and value reflects the average mean EVR across the 6-hr experimental period which includes 50-min of exercise and 10 min of rest.

$^{7\text{-}11}$ indicates the study protocol varied the hourly ventilation rate from 7-11 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.

[#] The study protocol describes the target exercise EVR as 20 L/min-m^2 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-m^2.

[15-23] indicates the study varied the hourly ventilation rate from 15-23 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.

[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 O_3 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 O_3 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 O_3 concentration measurements taken during the 6 exercise periods, the average O_3 concentrations for the four protocols are 0.063 ppm, 0.072 ppm, 0.081 ppm and 0.088 ppm, while the 6.6-hourTWA concentrations are 0.063 ppm, 0.073 ppm, 0.083 ppm and 0.088 ppm

Table 3A-3. Summary of controlled human exposures to O_3 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 wildypes 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	12M and 10F; (mean 30 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 (19-28 yrs)	**PF:** No significant change at any ventilation rate	Folinsbee et al., 1978; 1996 AQCD, p. 7-9
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
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, Table AX6-7

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	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, 7-10
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, $\dot{V}_E$=65 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. 7-164, 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. 7-164, Table 7-1
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] Al	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 Al subjects exposed for 4 days. **IF:** Significant increase in sputum eosinophils in As[A] and Al 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, 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

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]		
0.15	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
0.15	3 hr IE (4×15 min, $\dot{V}_E$=26 L/min) and	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, Table AX6-3
0.16	1 hr CE (mean $\dot{V}_E$ =57 L/min)	H[At]	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. 7-10, 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[At]	10M (19-29 yrs)	**PF:** ↓ FVC* and ↓ FEV1* 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)	Al	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. 7-164, 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. 7-164, 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 (threshhold 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[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 SY: ↑ respiratory symptoms*	Adams and Schelegle, 1983; 1996 AQCD, Table 7-1
0.20	1 hr CE ($\dot{V}_E$=89 L/min)	H[Ath]	15M and 2F (19-30 yrs)	**PF:** ↓ V_{Emax}*, ↓ VO_{2max}*, ↓ V_{Tmax}*, ↓ work load*, ↓ ride time*, ↓ FVC*, and ↓ FEV_1* compared with FA AR: > 20% increase in histamine responsiveness in nine subjects SY: ↑ respiratory symptoms*	Gong et al., 1986; 1996 AQCD, Tables 7-1, 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]		
0.20	1hr CE (mean $\dot{V}_E$=60 L/min); 2 exposures × 24 hr apart	H^{NS}	15M (mean 25 yrs)	**PF:** Consecutive days of exposure produced similar ↓ FVC* and ↓ FEV_1* on each day compared to FA SY: Consecutive days of exposure produced similar ↑ respiratory symptoms*	Brookes et al., 1989; 2006 AQCD. Table AX6-9
0.20	2 hr IE (4×15 min, 2 × 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 (4×15 min, $\dot{V}_E$=20 L/min)	As^A	4M and 5F (21-42 yrs)	**PF:** ↓ FEV_1* but not FVC AR: No change in sRaw **IF**: 6 hr postexposure ↑ 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 (4×15 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 (4×15 min, EVR=20 L/min-m^2)	H^{NS}	8M and 5F (20-31 yrs)	**PF:** ↓FVC*, ↓ FEV_1*, and ↓ 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 (4×15 min, EVR=20 L/min-m^2)	H	10M and 12F (mean 24 yrs)	**PF:** ↓ 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 (4×15 min, EVR=20 L/min-m^2) chronic inhaled corticosteroid	As	8M and 5F (mean 33 yrs)	**PF:** ↓FEV_1* and ↓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
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

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	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, p. 7-52, 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
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

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]		
0.24	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. 7-164, 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-$PGF_{2\alpha}$* 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
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 (4×15 min, EVR=14 L/min-m^2)	H	15M and 3F (mean 43yrs)	**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, Table 3-9

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	3 hr IE (4×15 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 (4×15 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, AX6-11
0.25	3 hr IE (4×15 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 (20-48 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, Table AX6-13
0.25	3 hr IE (4×15 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, Table 3-9
0.25	3 hr IE (4×15 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, Table 3-4
0.25	3 hr IE (4×15 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, Table 3-4
0.25	3 hr IE (4×15 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, Table AX6-3
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

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) 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 ($\dot{V}_E$=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, $\dot{V}_E$, 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 ($\dot{V}_E$ =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 ($\dot{V}_E$ =60 L/min) or 2hr IE ($\dot{V}_E$ =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
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

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)	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 (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; 1996 AQCD p. 7-9
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. 7-164, 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. 7-164, 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
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 ($\dot{V}_E$=80L/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

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	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: Signifciant 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 ($\dot{V}_E$=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
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

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 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 $PGF_{2\alpha}$; 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 myeleperoxidase increased; possible relationship of IL-8 and PMN levels.	Fahy et al., 1995; 2006 AQCD, Table AX6-12
0.40	2 hr IE (4×15 min, EVR=18 L/min-m^2) Postexposure, H^{WR} treated with naxloxone 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 that 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 FEV1 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

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=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
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] Al[NAs] As[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

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, $\dot{V}_E$=53-55 L/min)	H^{NAs} As^M	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 As^M 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
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

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	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. 7-164, Table 7-1
0.40	2.5 hr IE (4×15 min at $\dot{V}_E$=65 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. 7-164 Table 7-1
0.40	2 Hr IE (4×15 min, 2 × resting $\dot{V}_E$) 2 Hr IE (4×15 min, 2 × resting $\dot{V}_E$) × 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 histmine responsiveness was not different from sham exposures	Dimeo et al., 1981; 2006 AQCD, Table AX6-11
0.40	IE (2×15 min, $\dot{V}_E$=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, 6-12
0.40	3 hr/day (2 hr resting followed by 1 hr CE at 4-5 resting $\dot{V}_E$) 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
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-m2)	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[G]

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]		
0.10	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 responsivness 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]
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
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

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	2hr × 2 days during and out of grass pollen season	Al	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*, ↓ FVC* but no change in MVV	Folinsbee et al., 1978; 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*, ↓ FEF_{25}*, 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 for this review are in blue font.

[A] Reported target mean O_3 concentrations

[W] Focused on O_3 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^{At}), nonsmokers (H^{NS}), nonasthmatics (H^{NAs}), nonasthmatics with allergies (Al^{NAs}), asthmatics (As), mild asthmatics (As^{M}), SO_2-sensitive asthmatics (As^{SO2}), asthmatics with allergies (As^{A}), subjects with allergies (Al), smokers (S), healthy subjects with the GSTM1 genotype (H^{GSTM+}) or null for the GSTM1 genotype (H^{GSTM-}), healthy subjects that have a weak O_3 response (H^{WR}) or have a strong O_3 response (H^{SR}), healthy weight subjects (H^{Wt}) 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 O_3-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 O_3 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 O_3 exposure to subjects at rest was referenced in 1996 AQCD, Table 7-1.

[J] Subtracted from FA, the group mean decrement in FEV_1 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; FEF_{25}, (formerly designated as $V_{25\%VC}$) instantaneous forced expiratory flow after 25% of forced vital capacity; FEF_{25-75}, forced expiratory flow over the middle half of forced vital capacity; FEF_{50}, (formerly designated as $V_{50\%VC}$) instantaneous forced expiratory flow after 50% of forced vital capacity; FEV_1, 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; LTB_4, leukotriene; MMP9, metallopeptidase 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; PGE_2, prostaglandin E_2 a mediator of inflammation; PGE2, bronchodilatory prostaglandin; PGF2α, prostaglandin 2 alpha; PMN, polymorphonuclear neutrophils; ROS, reactive oxygen species; sGaw, specific airway conductance; 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, radiolabled 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-$PGF_{2\alpha}$, prostaglandin 2 alpha; ^{99m}Tc-DTPA, technetium 99m-labelled diethylenetriamine penta-acetic acid used aerosol ventilation studies

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APPENDIX 3B

AIR QUALITY INFORMATION FOR LOCATIONS OF EPIDEMIOLOGIC STUDIES OF RESPIRATORY EFFECTS

This appendix provides summary information about the O_3 concentrations in locations and time periods of epidemiologic studies of associations between O_3 in ambient air and respiratory health outcomes. Included here are studies conducted in the U.S. and Canada that found associations between O_3 exposure and respiratory health effects such as emergency department visits and hospital admissions, including studies that are newly available in this review, as well as those that were available at the time of the last review, and that are identified in the ISA. Information for studies identified in the ISA[1] as short-term are summarized in Table 3B-1 and a subset of studies identified as long-term are summarized in Table 3B-2.

Air quality information for U.S.-based studies was obtained from the EPA's Air Quality System (AQS) database.[2] For Canada-based studies, air quality information was obtained from the National Air Pollutant Surveillance (NAPS) program.[3] In Table 3B-1 and Table 3B-2, design values (DVs)[4] are presented as a range across all locations and time periods in the study.[5] Detailed information about designs values for individual study locations and time periods are 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 O_3 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.

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 O3 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 O3 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 O3 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	Winquist 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	Winquist 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 O3 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 O3 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.

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	Weichenthal 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 O_3 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 Pollutant Surveillance (NAPS) program for Canadian studies.
[C] Data for monitors were obtained for 1977-2000. Daily maximum 1-hour O_3 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 O_3 concentration for each monitor. The warm-season O_3 concentrations for the time period 1977-2000 were computed for each year to form a single annual time series of O_3 measurements for 96 metropolitan areas.
[D] Inverse distance weighted monthly average O_3 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 O_3 concentrations was generated for the months of May-October for years 2002-2009 using an air pollution-specific interpolation technique to generate a 21 km^2 grid value. The interpolation method incorporates modeled O_3 from the Canadian Hemispheric Regional Ozone and NO_X (CHRONOS) air quality forecast model with observations from Canada and the U.S.

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. O_3: 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. O_3: 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. O_3: 2002-2009

Air quality data are not described for this study as it relied on O_3 concentrations for the years 2002–2009 as surrogates for study population annual O_3 concentrations during the 1984 to 2011 period (Cakmak, 2017).

Crouse et al., 2015 (3019335) - Long-Term Ozone and Respiratory Mortality

Nationwide, Canada. O_3: 2002-2009

Air quality data are not described for this study as it relied on O_3 concentrations for the years 2002–2009 as surrogates for study population annual O_3 concentrations during the 1984 to 2006 period (Crouse, 2015).

Darrow et al., 2011 (202800) - ED Visits for Aggregate Respiratory Diseases

20-county Atlanta area, GA, U.S. O_3: 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.											

Darrow et al., 2014 (2526768) - ED Visit - Respiratory Infection

20-county Atlanta area, GA, U.S. O_3: 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

Eckel et al., 2016 (3426159) - Long-Term Ozone and Respiratory Mortality

California, U.S. O_3: 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

Garcia et al., 2019 (5119704) - Asthma Incidence
Nine communities in Southern California, U.S. O_3: 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

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

Gleason et al., 2014 (2369662) - ED Visits for Asthma

New Jersey (statewide), U.S. O_3: April-September, 2004-2007

State	dv.2004.2006	dv.2005.2007
New Jersey	93	92

Goodman et al., 2017a (3859548) - Hospital Admissions for Asthma,

New York City (20-mi radius from center), NY, U.S. O_3: 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

Goodman et al., 2017b (4169406) - Hospital Admissions for Asthma

Houston, Dallas, and Austin, TX metro areas, U.S. O_3: 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

Ito et al., 2007 (156594) - Emergency Department Visits for Asthma

New York City, NY. O_3: 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.			

Jerrett et al., 2009 (194160) - Long-Term Ozone and Respiratory Mortality
Nationwide, U.S. O_3: 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.								

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								

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.									

Jerrett et al., 2013 (2094363) - Long-Term Ozone and Respiratory Mortality

California, U.S. O_3: 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

Katsouyanni et al., 2009 (199899) - Short-Term Ozone and Respiratory Mortality

Nationwide, U.S. O_3: 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.									

Klemm et al., 2011 (1011160) - Short-Term Ozone and Respiratory Mortality

Atlanta (Fulton, DeKalb, Gwinnet & Cobb counties), GA, U.S. O_3: 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.2004.2006	dv.2005.2007
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	121	107	99	91	93	90	91	95

Kousha and Rowe, 2014 (2443421) - ED Visit - Respiratory Infection

Edmonton, Canada. O_3: 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

Kousha and Castner, 2016 (3160295) - ED Visit - Respiratory Infection

Windsor, Canada. O_3: 2004-2010

City	dv.2004.2006	dv.2005.2007	dv.2006.2008	dv.2007.2009	dv.2008.2010
Windsor	80	87	84	80	73

Malig et al., 2016 (3285875) - ED Visits for Asthma, ED Visits Aggregate Respiratory Diseases, ED Visit for Respiratory Infection

California (statewide), U.S. O_3: 2005-2008

State	dv.2005.2007	dv.2006.2008
California	122	119

Nishimura et al., 2013 (1632336)

Four U.S. cities (Chicago, Houston, New York, San Francisco) and Puerto Rico.

This is a case control study with study participants, aged 8-21 years, identified during 2006-2011. Associations examined for annual average O_3 concentration (1-h max; 8-h max, per ISA), averaged across first three years of life. Median birth year was 1996.

O'Lenick et al., 2017 (3421578) - ED Visits for Asthma

20-county Atlanta metro area, GA, U.S. O_3: 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

O'Lenick et al., 2017 (3859553) - ED Visits Aggregate Respiratory Diseases

20-co Atlanta, GA; 12-co Dallas, TX, and 16-co St. Louis, MO, U.S. O_3: 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

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. O_3: 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. O_3: 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. O_3: 1999-2002

City	Census Area Name	dv.1999.2001	dv.2000.2002
Atlanta, GA	Atlanta-Sandy Springs-Roswell, GA	107	99

Sarnat et al., 2015 (2772940) - ED Visits for Asthma

St. Louis metro area, MO (8 MO counties, 8 IL counties), U.S. O_3: 2001-2003

City	Census Area Name	dv.2001.2003	dv.2002.2004
St. Louis	St. Louis-St. Charles-Farmington, MO-IL	92	92

Sheffield et al., 2015 (3025138) - ED Visits for Asthma

New York City (all boroughs), NY, U.S. O_3: 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

Shmool et al., 2016 (3288326) - ED Visits for Asthma

New York City, NY, U.S. O_3: 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

Silverman and Ito, 2010 (386252) HA for Asthma

New York, NY. O_3: 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.							

Stieb et al., 2009 (195858) - ED Visits for Asthma

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

Strickland et al., 2014 (2519636) - ED Visits for Asthma

20-county Atlanta area, GA, U.S. O_3: 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

Szyszkowicz et al., 2018 (4245266) - ED Visits for Asthma, [ED Visit - Respiratory Infection]

Multicity (9), Canada. O_3: 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).						

Tolbert et al., 2007 (90316) - ED Visits for Aggregate Respiratory Diseases

Atlanta, GA. O_3: 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.											

Tétreault et al., 2016 (3073711) – Asthma Incidence

Quebec Province, Canada. O_3: 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

Turner et al., 2016 (3060878) - Long-Term Ozone and Respiratory Mortality

Nationwide, U.S. O_3: 2002-2004

Air quality data are not described for this study as it relied on estimated O_3 concentrations for the years 2002–2004 as surrogates for study population O_3 concentrations during the 1982 to 2004 period (Turner et al., 2016).

Vanos et al., 2014 (2231512) - Short-Term Ozone and Respiratory Mortality

10 Canadian cities, Canada. O_3: 1981 - 1999. The table below does not include design values prior to 1988 as data are not readily 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

Villeneuve et al., 2007 (195859) - ED Visits for Asthma

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

Weichenthal et al., 2017 (4165121) - Long-Term Ozone and Respiratory Mortality

Nationwide, Canada. O_3: 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

Winquist et al., 2012 (1668375) - Hospital Admissions for Asthma, ED Visits for Asthma, Hospital Admissions for Aggregate Respiratory, ED Visits for Aggregate Respiratory Diseases, ED Visits for Respiratory Infection

St. Louis, MO (8 MO and 8 IL counties, 269 zip codes), U.S. O_3: 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

Winquist et al., 2014 (2347402) - ED Visits for Asthma

Atlanta metro area, GA, U.S. O_3: 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

Xiao et al., 2016 (3455927) - ED Visits for Asthma, ED Visit - Respiratory Infection

Georgia (statewide), U.S. O_3: 2002-2008

State	dv.2002.2004	dv.2003.2005	dv.2004.2006	dv.2005.2007	dv.2006.2008
Georgia	93	93	91	95	95

Zanobetti and Schwartz, 2008 (101596) - Short-Term Ozone and Respiratory Mortality

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.

Zu et al., 2017 (3859551) - Hospital Admissions for Asthma

6 Texas City Metro areas (Austin, Dallas, El Paso, Ft Worth, Houston, San Antonio), U.S. *(pooled, not individually assessed)*

O_3: 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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3C.1 OVERVIEW

This appendix describes the development of the ozone (O_3) air quality estimates used in the population exposure and risk modeling described in Appendix 3D. Figure 3C-1 below shows a flowchart of the various data sources, processes and outputs involved in generating these ambient O_3 concentration surfaces. This approach was used for eight urban study areas, which are described further in section 3C.2.

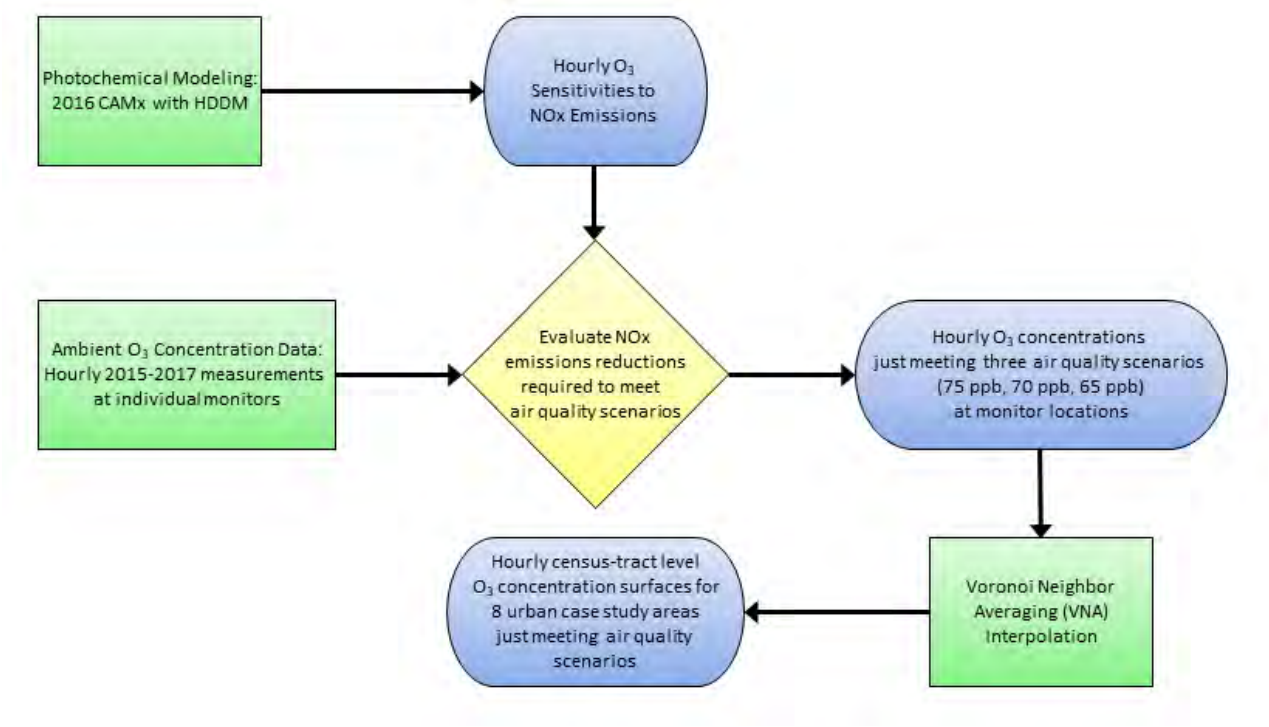


Figure 3C-1. Flowchart showing inputs, processes and outputs of the approach to generate ambient air concentration estimates for use in the exposure and risk modeling.

Generation of the O_3 concentration surfaces for the exposure and risk modeling relied on a combination of recent monitoring data and a model-based adjustment. Ambient hourly O_3 monitoring data for years 2015 through 2017 in each of the eight urban study areas was adjusted using a model-based adjustment approach to create three different air quality scenarios. These scenarios included conditions that just meet the current O_3 standard (design value of 70 ppb), as well as conditions that just meet two alternative air quality scenarios having design values of 75 ppb and 65 ppb. Section 3C.3 provides additional information on the monitoring data. Section 3C.4 describes the air quality modeling that was used to perform the adjustments, as well as results from the model evaluation that was performed to assess the accuracy of the modeled concentrations. Section 3C.5 describes the model-based adjustment approach and its application to the ambient air quality data to create the three air quality scenarios.

The final step in preparing the air quality input data for the exposure and risk modeling is to interpolate the adjusted air quality data from the ambient air monitoring site locations to each census tract in the eight urban study areas using Voronoi Neighbor Averaging (VNA), which is described in section 3C.6. Finally, section 3C.7 provides various results from the model-based adjustment procedure and the final air quality dataset used as inputs to the Air Pollutants Exposure Model (APEX). The APEX model and its application to air quality in the eight urban study areas is described in Appendix 3D.

The draft PA, with a draft version of this appendix was provided to the CASAC for its review and to the public for public comment, as summarized in section 1.4 of this PA. In consideration of the CASAC and public comments, this appendix incorporates a number of additions and clarifications, including the following:

- Cites section in Appendix 3D for description of study area selection (section 3C.2);
- Summarizes differences in emissions between 2014 NEI and 2016 Platform used for modeling in this assessment (section 3C.4.1.5);
- Adds clarifications regarding the model evaluation tables and figures presented in section 3C.4.2 (Figure 3C-12 to Figure 3C-47; Table 3C-5 to Table 3C-17);
- Provides rationale for choosing nitrogen oxides (NO_X) reductions only instead of the combined NO_X and volatile organic compounds (VOC) reductions which were used in the previous review (section 3C.5.2.2.3); and
- Adds a reference to a cross-validation analysis conducted in the last review, which supports the use of the VNA technique for generating the air quality spatial fields (section 3C.6).

3C.2 URBAN STUDY AREAS

Eight urban study areas were chosen for analysis based on several criteria, including geographic distribution, population, current air quality levels, availability of exposure model inputs, air quality model performance, and ambient air monitoring network coverage. The selection criteria and any other considerations in study area selection are described in Appendix 3D, section 3D.2.1, of this PA. The eight urban study areas selected were: Atlanta, GA; Boston, MA; Dallas, TX; Detroit, MI; Philadelphia, PA; Phoenix, AZ; Sacramento, CA; and St. Louis, MO. Figure 3C-2 shows a map of these eight study areas and 0 provides summary information for each area. The spatial extent of each study area was determined using the Combined Statistical Area (CSA), with the exception of the Phoenix study area, which is not in a CSA. In that case, the Core Based Statistical Area (CBSA) was used as the area boundary. [1]

[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*.

Figure 3C-2. Map showing the location of the eight urban study areas.

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 0 is the CBSA name.

3C.3 AMBIENT AIR OZONE MONITORING DATA

Hourly O_3 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 values[2] 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 O_3 concentration. A monitoring site meets the current standard if its design value is less than or equal to 70 ppb.

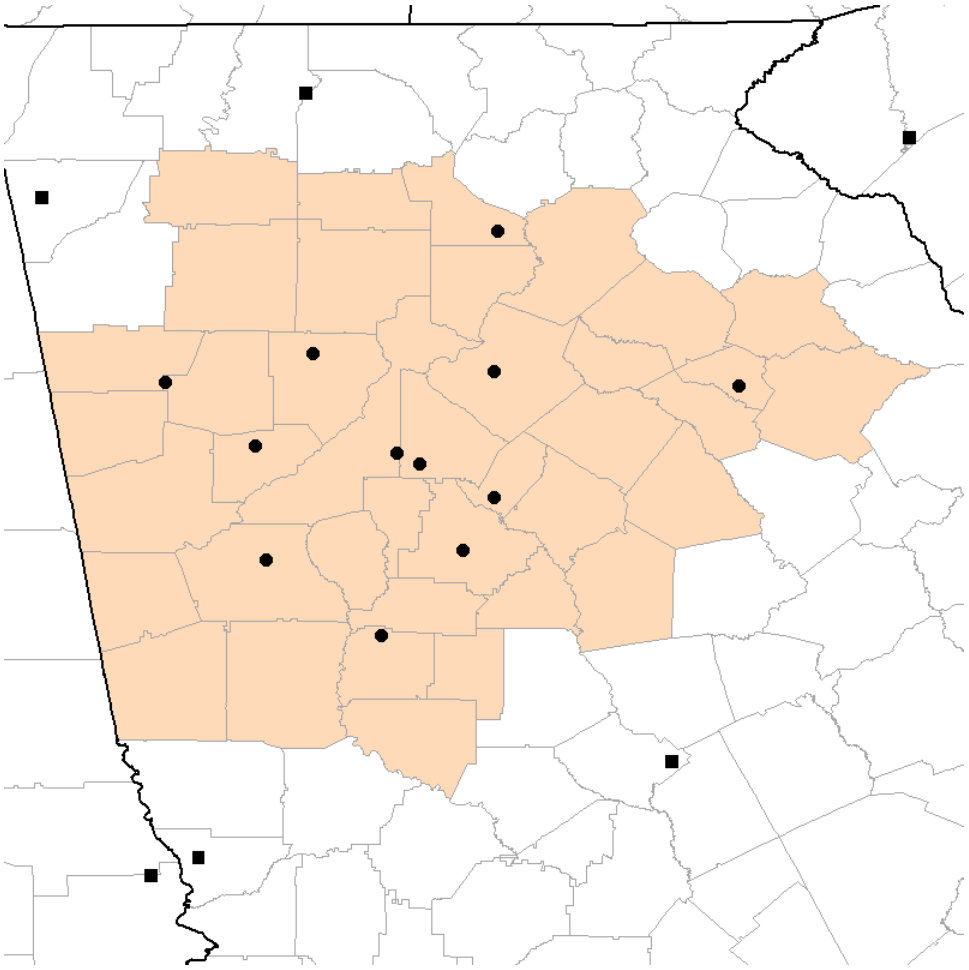


Figure 3C-3. Map of the Atlanta study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

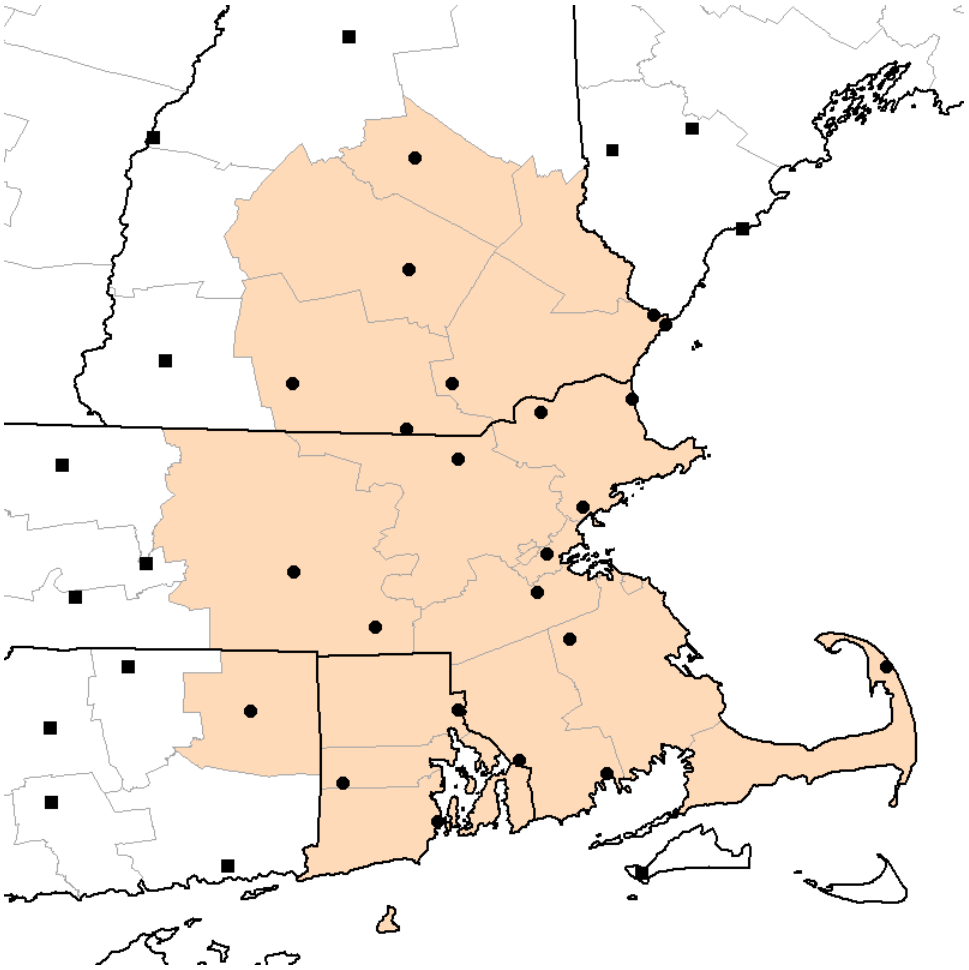


Figure 3C-4. Map of the Boston study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

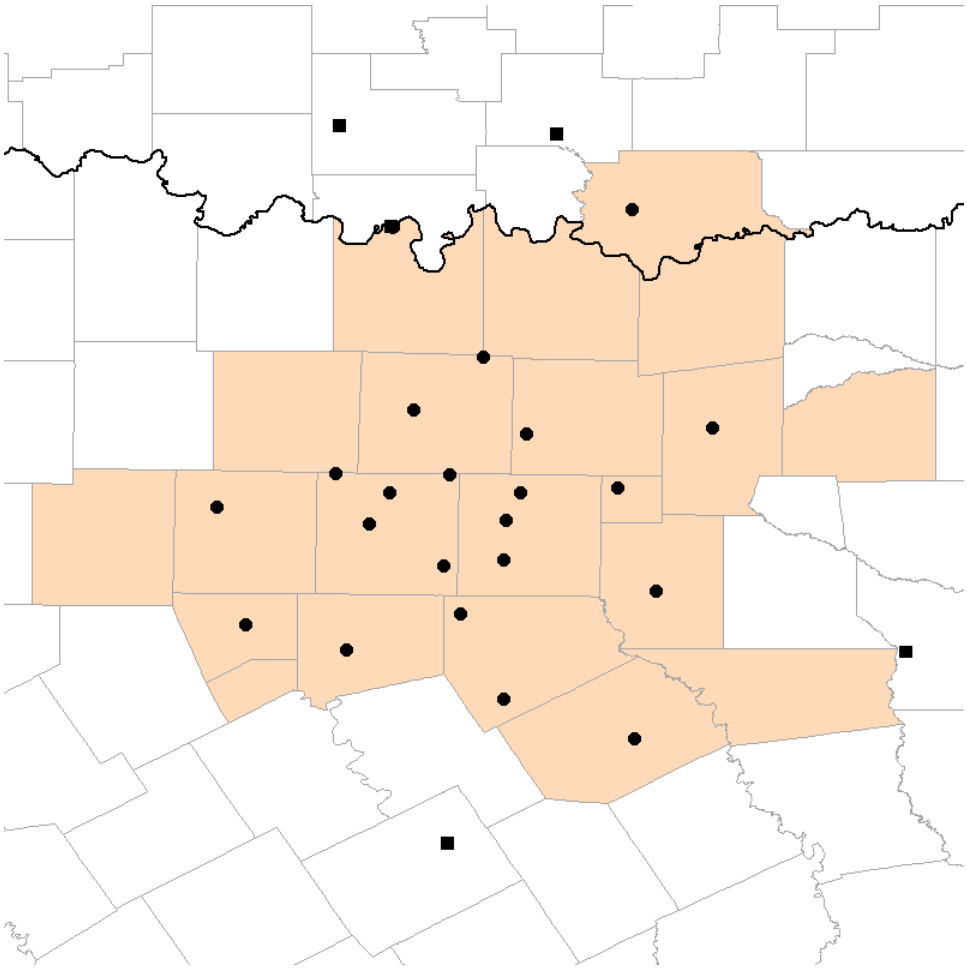


Figure 3C-5. Map of the Dallas study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

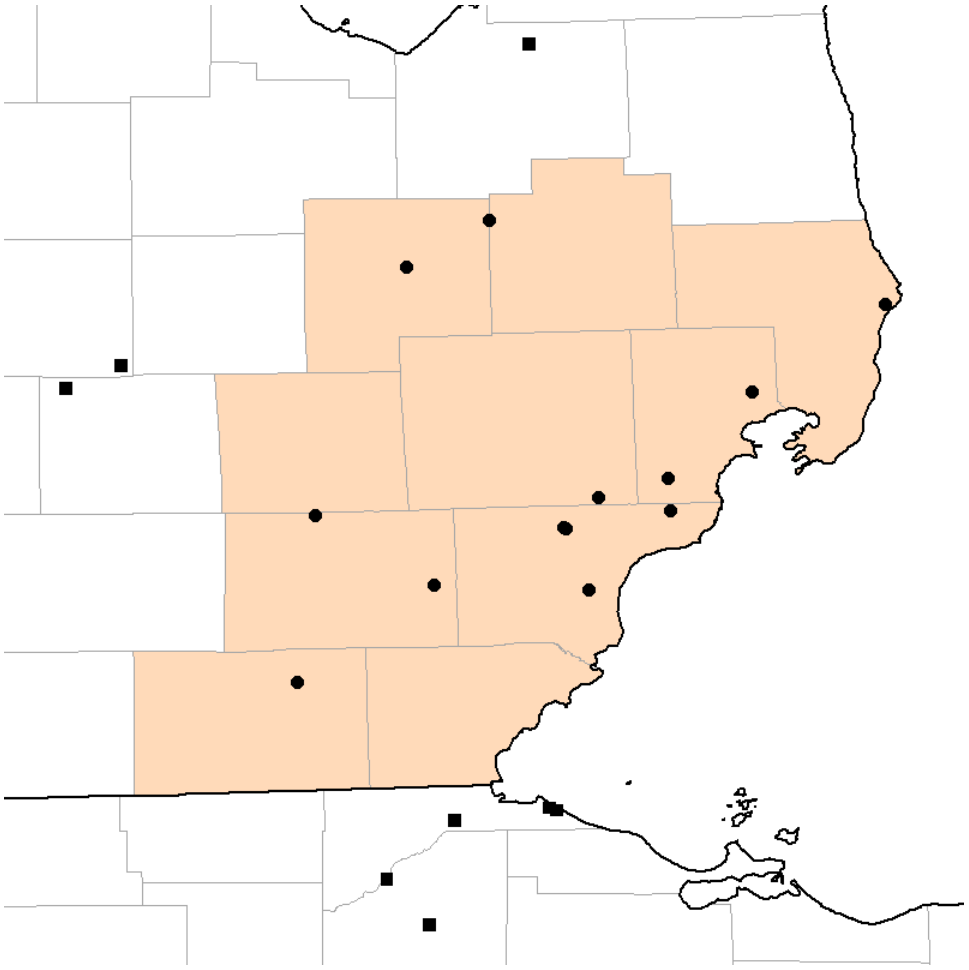


Figure 3C-6. Map of the Detroit study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

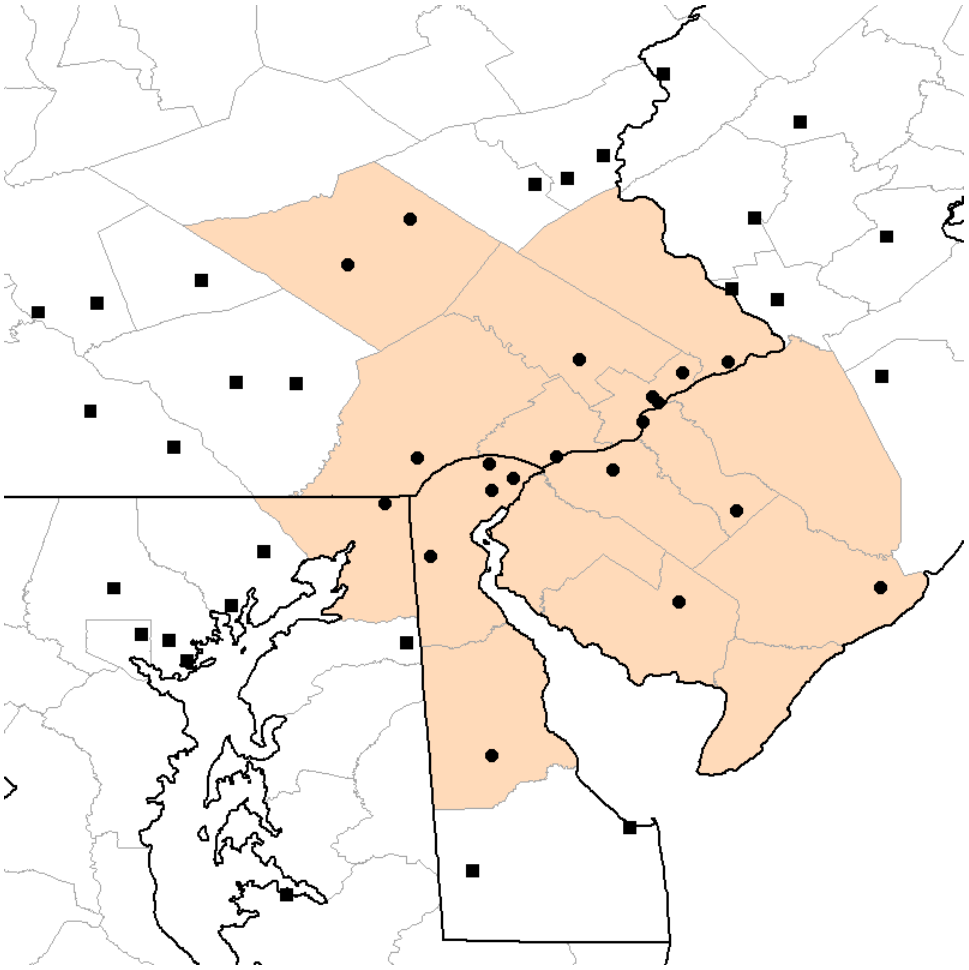


Figure 3C-7. Map of the Philadelphia study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

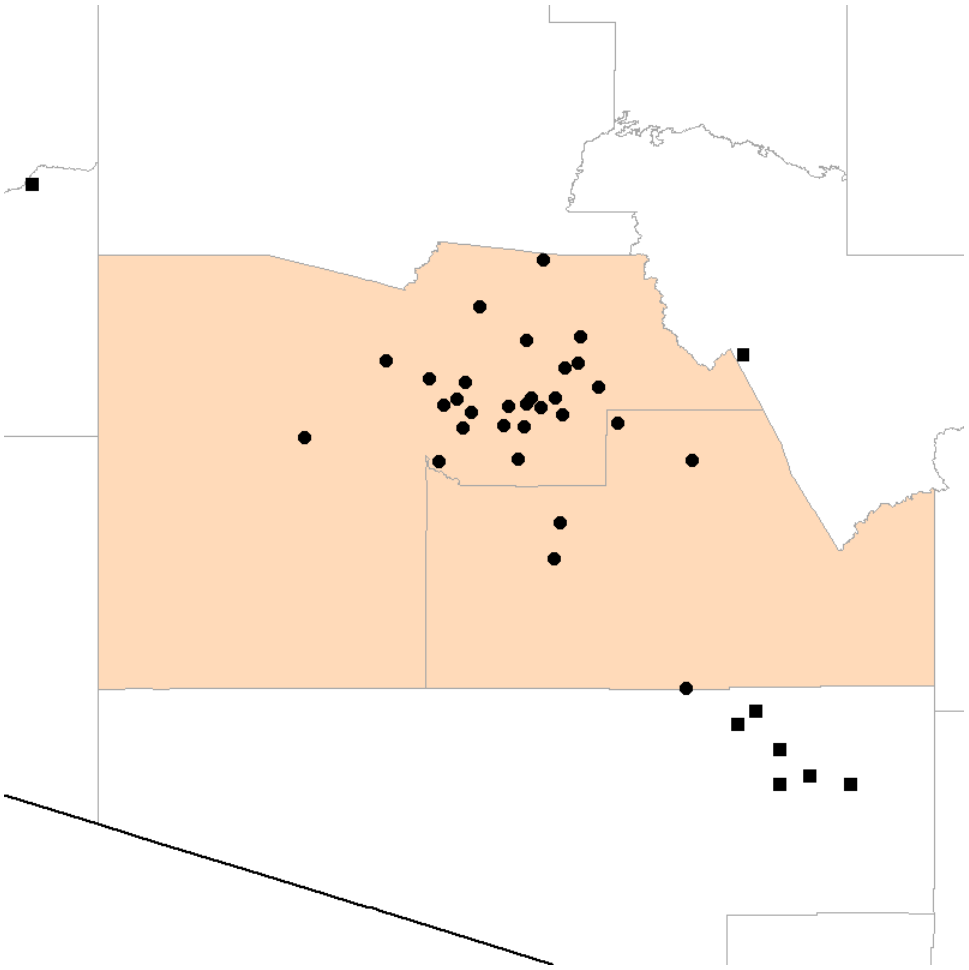


Figure 3C-8. Map of the Phoenix study area. Counties in the CBSA are shaded, monitoring sites in the CBSA are denoted by black circles, and buffer sites are denoted by black squares.

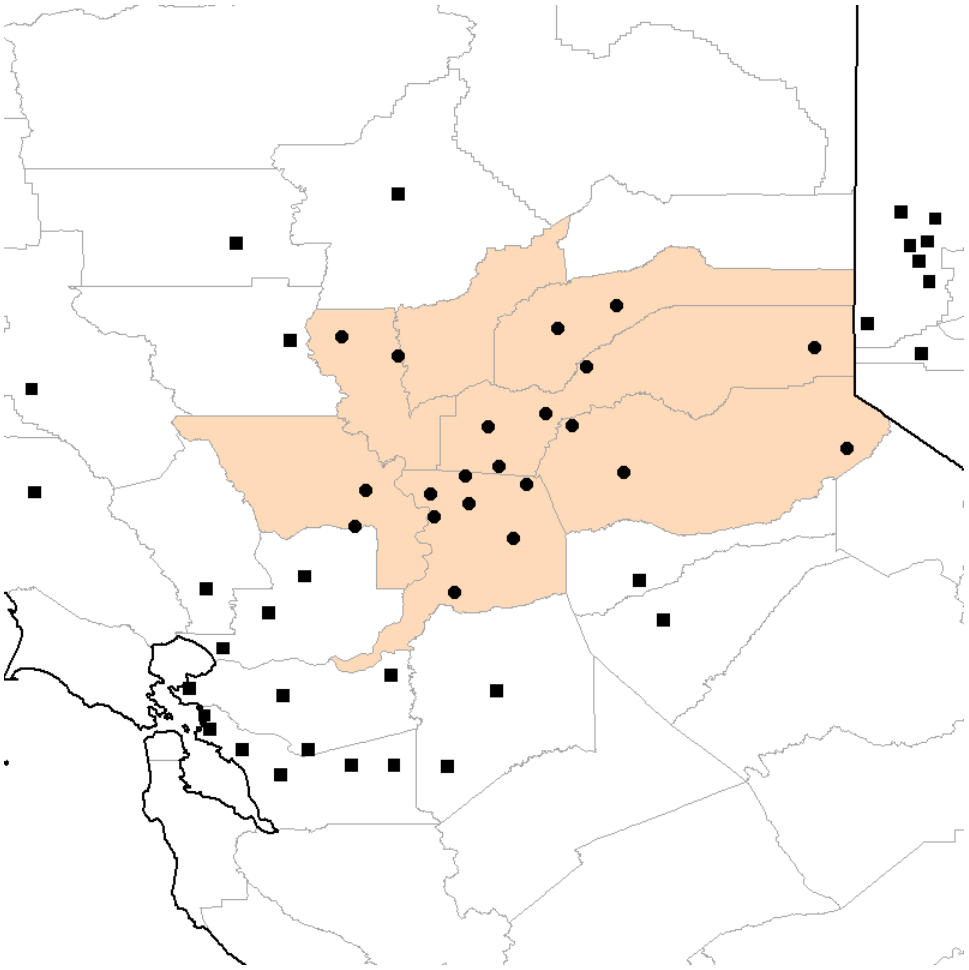


Figure 3C-9. Map of the Sacramento study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

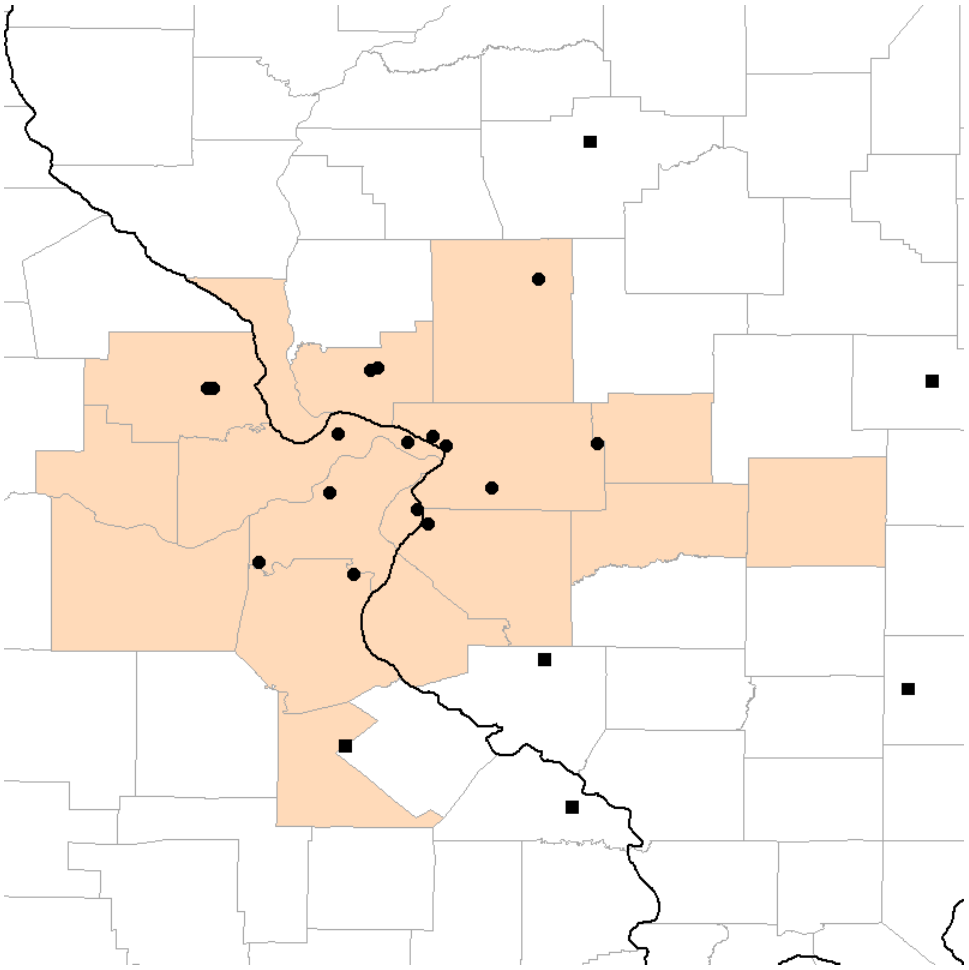


Figure 3C-10. Map of the St. Louis study area. Counties in the CSA are shaded, monitoring sites in the CSA are denoted by black circles, and buffer sites are denoted by black squares.

It is worth noting that for an area to show compliance with the current O_3 standard, all monitors within the urban area must have design values less than or equal to 70 ppb. According to Appendix U to 40 CFR Part 50, air quality monitors must also meet certain data completeness requirements to show compliance with the standard. However, any design value based on 3 years of monitoring data that exceeds the standard is not in compliance, regardless of data completeness. Therefore, when performing the air quality adjustments to create the three air quality scenarios, all monitors in each urban study area with data reported for each of the 3 years were included, regardless of data completeness.

Finally, per Appendix U to 40 CFR Part 50, data not meeting the ambient air monitoring requirements in 40 CFR Part 58, data reported using methods other than Federal Reference or Equivalent Methods, and data concurred by the appropriate EPA Regional Office as having been affected by an exceptional event were excluded from design value calculations. However, once the emissions changes required to determine compliance with the various air quality scenarios

had been determined, these values were included in the final adjustment and spatial interpolation. In practice, fewer than 10,000 hourly concentrations out of more than 3 million (~0.3%) were excluded from design value calculations in this manner.

3C.4 AIR QUALITY MODELING DATA

3C.4.1 Comprehensive Air Quality Model with Extensions (CAMx)

3C.4.1.1 Model Set-up and Simulation

The Comprehensive Air Quality Model with Extensions (CAMx) was used as the modeling tool for this assessment. CAMx is a peer-reviewed model that simulates the formation and fate of photochemical oxidants, aerosol concentrations, acid deposition, and air toxics, over multiple scales for given input sets of meteorological conditions and emissions. CAMx is used frequently for a range of scientific and regulatory applications related to the analysis of air quality in the U.S. The Higher Order Direct Decoupled Method (HDDM) was implemented in CAMx to estimate the model sensitivities to emissions changes as described in section 3C.5 of this appendix. The CAMx-HDDM configuration tracks gas-phase species concentrations through all modeled processes. However, HDDM implemented in CAMx does not track the effects of aerosol and cloud processing on calculated O_3 sensitivities. Differences in predicted O_3 concentrations between the CAMx-HDDM configuration described here and a standard CAMx v6.5 simulation with full treatment of aerosol-O_3 interactions did not influence O_3 predictions in the urban study areas examined in this assessment. CAMx v6.5[3] was run using the carbon bond version 6 (CB06r4) gas-phase chemical mechanism (Yarwood et al., 2010; Gery et al., 1989) and the AERO6 aerosol module which includes ISORROPIA for gas-particle partitioning of inorganic species (Nenes et al., 1998) and secondary organic aerosol treatment as described in Carlton et al. (2010).

3C.4.1.2 Model Domain

For this analysis, all CAMx runs were performed for a domain that covers the 48 contiguous states including portions of southern Canada and Northern Mexico with a 12 x 12 km resolution (Figure 3C-11). The CAMx simulations were performed with 35 vertical layers with a top layer at about 17,600 meters, or 50 millibars (mb). Table 3C-2 and Table 3C-3 provide some basic geographic information regarding the CAMx domain and vertical layer structure, respectively. Results from the lowest layer of the model were used for analyses to support the risk and exposure analyses described in Appendix 3D.

[3] For more information, see: *http://www.camx.com/files/camxusersguide_v6-50.pdf.*

Figure 3C-11. Map of the CAMx modeling domain.

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

3C.4.1.3 Model Time Period

The CAMx/HDDM modeling was performed for January 1 - December 31 of 2016. The simulations included a 10-day spin-up period[4] from December 22-31, 2015. The spin-up days were not considered in the analysis for the HDDM results.

3C.4.1.4 Model Inputs: Meteorology

CAMx model simulations require inputs of meteorological fields, emissions, and initial and boundary conditions. The gridded meteorological data for the entire year of 2016 at the 12 km continental U.S. scale domain were derived from version 3.8 of the Weather Research and Forecasting Model (WRF), Advanced Research WRF (ARW) core (Skamarock et al., 2008). The WRF Model is a mesoscale numerical weather prediction system developed for both operational forecasting and atmospheric research applications.[5] The 2016 WRF simulation included the physics options of the Pleim-Xiu land surface model (LSM), Asymmetric Convective Model version 2 planetary boundary layer (PBL) scheme, Morrison double moment microphysics, Kain-Fritsch cumulus parameterization scheme and the RRTMG long-wave radiation (LWR) scheme (Gilliam and Pleim, 2009). Additionally, lightning data assimilation was utilized to suppress (force) deep convection where lightning was absent (present) in observational data. This method is described by Heath et al. (2016) and was employed to help improve precipitation estimates generated by the WRF model.

The WRF and CAMx simulations used the same map projection, a lambert conformal projection centered at (-97, 40) with true latitudes at 33 and 45 degrees north. The WRF and CAMx simulations utilized 35 vertical layers with a surface layer of approximately 19 meters. Table 3C-3 shows the vertical layer structure used in WRF to generate the CAMx meteorological inputs.

The WRF meteorological outputs were processed to create model-ready inputs for CAMx using the wrfcamx version 4.3 meteorological pre-processor (Ramboll Environ, 2014). The specific meteorological inputs to CAMx include: horizontal wind components (i.e., speed and direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each grid cell in 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*

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

A detailed meteorological model performance evaluation was conducted for the 2016 WRF simulations (U.S. EPA, 2017). The analysis included statistical evaluation of temperature, wind speed, and water vapor mixing ratios against observational data from airports, as well as evaluations of monthly precipitation compared to the Parameter-elevation Relationships on Independent Slopes Model (PRISM) and shortwave radiation compared to data from the Surface

Radiation Budget Measurement Network (SURFRAD) and the Solar Radiation Network (SOLRAD).

3C.4.1.5 Model Inputs: Emissions

The emissions data used are based on the alpha version of the Inventory Collaborative 2016 emissions modeling platform.[6] The modeling case used is abbreviated "2016fe" and is publicly available.[7]

Emissions were processed to photochemical model inputs with the SMOKE modeling system version 4.5 (Houyoux et al., 2000). For this analysis, emissions from wildfires and prescribed burns were based on year 2016 nationally available fire datasets. Electric generating unit (EGU) emissions are temporally allocated to hourly values based on patterns derived from year 2016 Continuous Emissions Monitoring System (CEMS) data. In addition, U.S. emissions are included from other point sources, area sources, agricultural sources (ammonia only), anthropogenic fugitive dust sources, nonroad mobile sources, onroad mobile sources, and biogenic sources. Emissions for onroad mobile sources were created using the EPA's MOVES 2014a model,[8] except that California emissions were adjusted to match the county total emissions obtained directly from the California Air Resources Board. Biogenic emissions were estimated using the Biogenic Emissions Inventory System version 3.61 (BEISv3.61) (Pouliot and Bash, 2015). Other North American emissions from areas outside the U.S. are based on a 2013 Canadian inventory scaled to 2015, and projections of the 2008 Mexican inventory to the year 2016 along with the scaling of MOVES-Mexico emissions to year 2016 (ERG, 2017). The construction of the emissions is described in more detail in the technical support document Preparation of Emissions Inventories for the Version 7.1 2016 Regional Emissions Modeling Platform (U.S. EPA, 2019). Emissions totals within the United States are summarized in Table 3C-4 for CO, NH_3, NO_X, PM_{10}, $PM_{2.5}$, SO_2, and VOC. Anthropogenic NO_X emissions in the 2016 platform are about 19% lower than those reported in the 2014 NEI due to both improved inventory development methods and updates to specific components (e.g., cleaner vehicles entering the onroad mobile fleet or EGUs transitioning from coal to natural gas).

[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*

Table 3C-4. Summary of U.S. emissions totals by sector for the 12km CONUS domain (in 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

3C.4.1.6 Model Inputs: Boundary and Initial Conditions

Initial and lateral boundary concentrations for the 12 km US2 domain are provided by the hemispheric version of the Community Multi-scale Air Quality model (H-CMAQ) v5.2.1. H-CMAQ was run for 2016 with a horizontal grid resolution of 108 km and 44 vertical layers up to 50 hPa. The H-CMAQ predictions were used to provide one-way dynamic boundary conditions at one-hour intervals. An operational evaluation against sonde and satellite observations showed

that the 2016 H-CMAQ simulation reasonably captured general patterns of O_3 transport within the northern Hemisphere that are relevant for the 12US2 domain (Henderson et al., 2018).

3C.4.2 Evaluation of Modeled Ozone Concentrations

In this section we present the results of an evaluation of the CAMx configuration used to produce the air quality results described in Chapter 3. Specifically, we summarize the ability of the CAMx model to reproduce the corresponding 2016 measured O_3 concentrations. This operational evaluation shows that in general for most regions and seasons, the CAMx model predictions for 2016 generally reproduce patterns of observed O_3. The notable exception to this is a persistent underestimate in winter across almost all regions, particularly at higher latitude sites.

In the following sections we present general model performance statistics and plots for five regions of the U.S. We compare model predictions of maximum daily 8-hr average (MDA8) O_3 concentrations to measurements reported in EPA's AQS. We note that these comparisons are based on MDA8 values calculated across all available modeled CAMx values and all observed (AQS) concentrations, and that these comparisons include buffer sites. Model performance could be different for comparisons without buffer sites, or using the modeled CAMx MDA8 values only when the corresponding observed MDA8 values are available.

The model statistics presented here include mean bias, mean error, normalized mean bias, and normalized mean error as calculated below, where *n* represents the total number of observations:

Mean Bias:	$(\sum modeled - observed)/n$
Mean Error:	$(\sum \lvert modeled - observed \rvert)/n$
Normalized Mean Bias:	$(\sum modeled - observed)/(\sum observed)$
Normalized Mean Error	$(\sum \lvert modeled - observed \rvert)/(\sum observed)$

Our analysis focuses on regional model evaluation statistics from five US regions as well as evaluations of the eight urban study areas included in the exposure and risk analysis – Atlanta, Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento, and St. Louis.[9,10] Statistics for CAMx model performance in these regions and urban study areas are shown by season in Table 3C-5 through Table 3C-17 for observed days with MDA8 O_3 values $\geq$ 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.

with MDA8 O_3 < 60 ppb, and for all observed days. For each of the five regions listed above, spatial plots are provided for each season showing Normalized Mean Bias (NMB) for MDA8 O_3 at individual sites. Summary NMB ranges are included at the bottom of each map showing the min and max values for the season/region across all sites, as well as the 25th, 50th, and 75th percentile values. Time series plots are provided for MDA8 O_3 in each urban study area for the period from January-December 2016. Hourly time series plots are also provided for one month in each season (January, April, July, October).[11]

3C.4.2.1 Operational Evaluation in the Northeastern U.S.

Table 3C-5 shows that in the Northeast Region, model mean bias was generally less than 7 ppb and normalized mean bias was less than 15% in most cases. Errors were largest in the winter, with underestimates also extending to the spring. Spatial maps of normalized mean bias are shown in Figure 3C-12 through Figure 3C-15. During the O_3 season performance was best on high O_3 days, particularly in the summer and fall. Two of the eight urban study areas evaluated were in the Northeast: Boston and Philadelphia.

Model performance at the Boston study area monitoring sites (Table 3C-6) was similar to that of the Northeast Region. The time series plots show that the model reasonably reproduces the measured day-to-day variability in MDA8 O_3 concentrations (Figure 3C-16). The underestimate in winter-spring observed in the Northeast region statistics is particularly pronounced in Boston, likely due to its relatively northerly location where seasonal daylight and temperature changes are more exaggerated. Variability of hourly daytime and nighttime O_3 concentrations is generally well modeled in all seasons, again noting the persistent underestimate in January/April. Model characterization of hourly variability is particularly good in July, although peak daytime O_3 is slightly overestimated. Nighttime O_3 is also consistently overestimated in July/October (Figure 3C-17).[12]

Bulk model performance statistics for Philadelphia (Table 3C-7) are again similar to those for the Northeast as a whole, with more moderate performance compared to Boston during both winter (not as poor) and summer/fall (not as good). The spring underestimate present in the Boston comparisons is much smaller for Philadelphia (Figure 3C-18, Figure 3C-19), again suggesting that the winter-spring underestimate is more pronounced at more northerly sites. Philadelphia also exhibits the nighttime overestimates in the July/October hourly comparisons 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.

Table 3C-5. CAMx model performance at monitoring sites in the Northeastern U.S. Statistics shown are mean bias (MB), normalized mean bias (NMB), mean 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

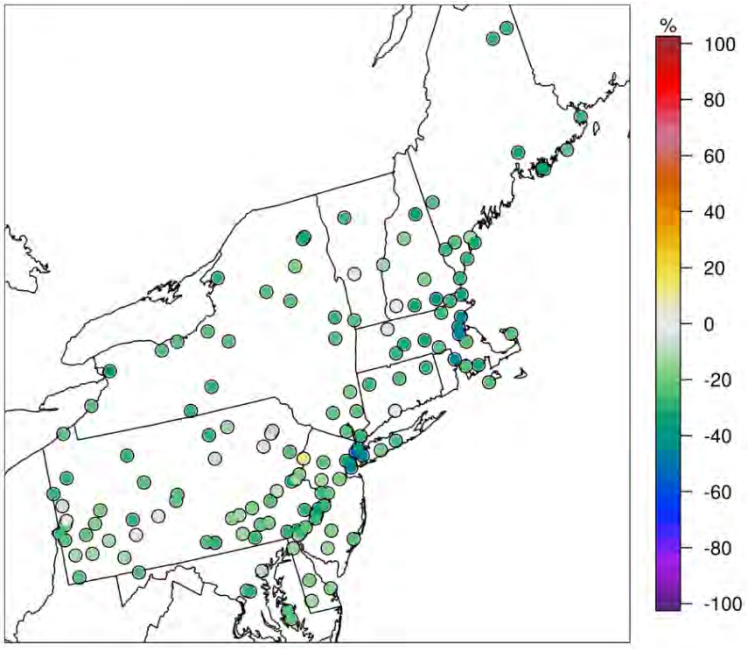
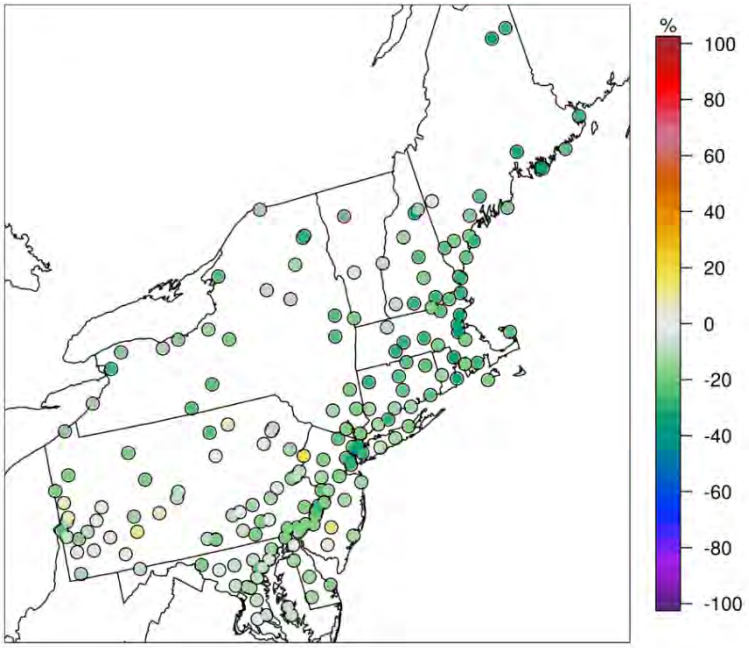
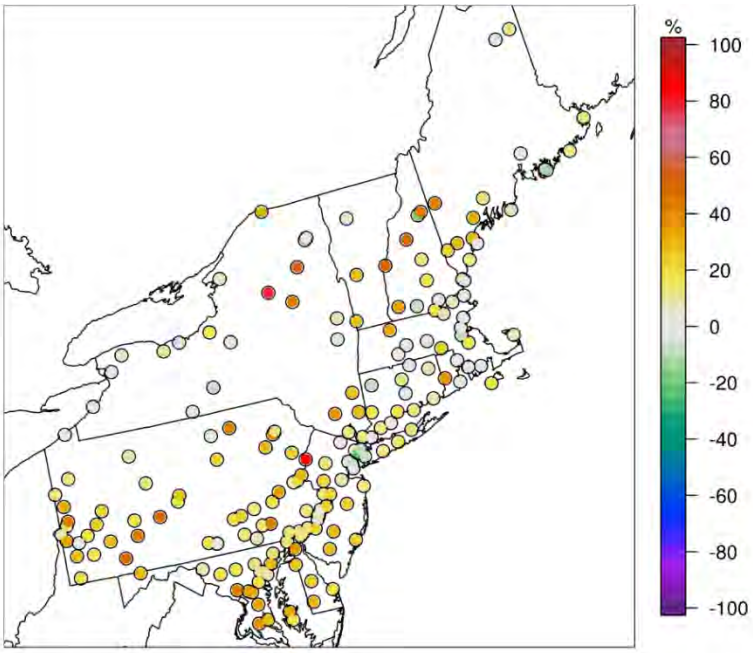


Figure 3C-12. Normalized mean bias for MDA8 O_3 in the Northeastern U.S., winter 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-43, -20, -14, -7.1, 23]

Figure 3C-13. Normalized mean bias for MDA8 O_3 in the Northeastern U.S., spring 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-21, 7.8, 17, 28, 85]

Figure 3C-14. Normalized mean bias for MDA8 O_3 in the Northeastern U.S., summer 2016.

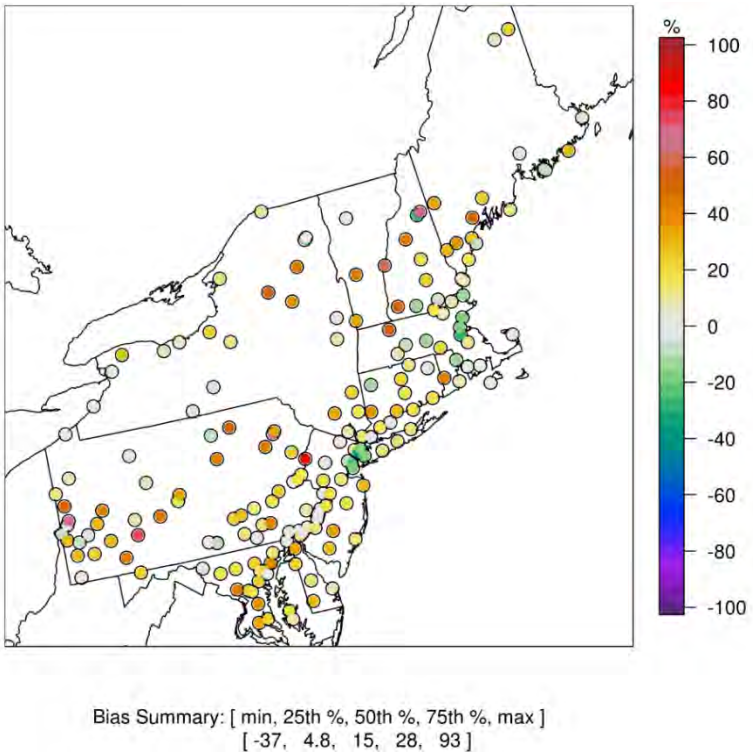


Figure 3C-15. Normalized mean bias for MDA8 O_3 in the Northeastern U.S., fall 2016.

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

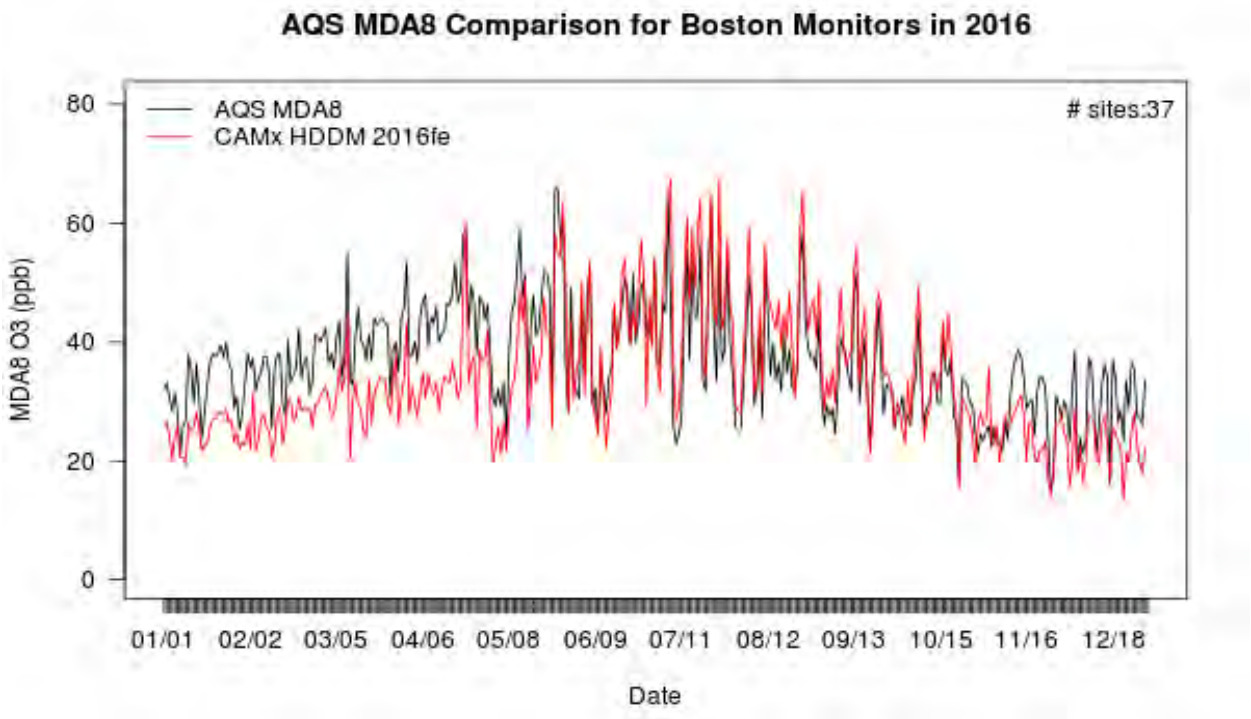


Figure 3C-16. Time series of monitored (black) and modeled (red) MDA8 O_3 at Boston monitoring sites in 2016.

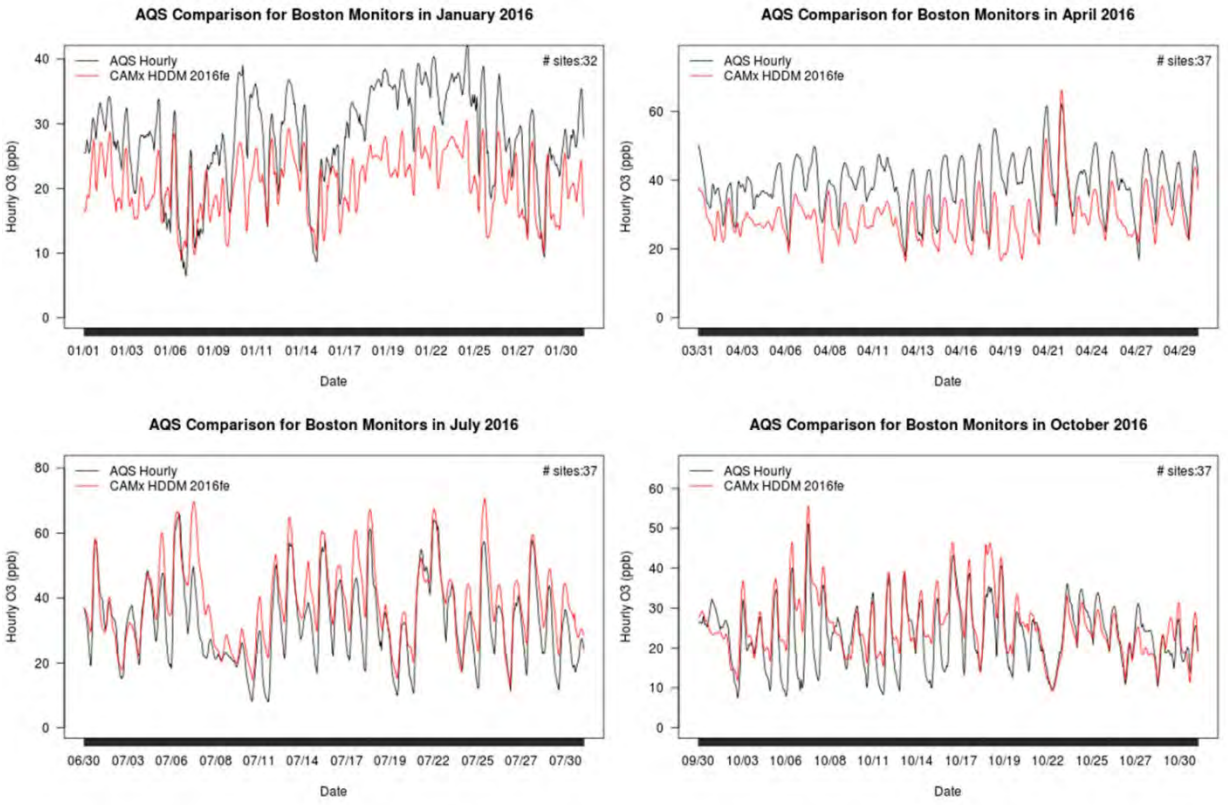


Figure 3C-17. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at Boston monitoring sites in January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

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

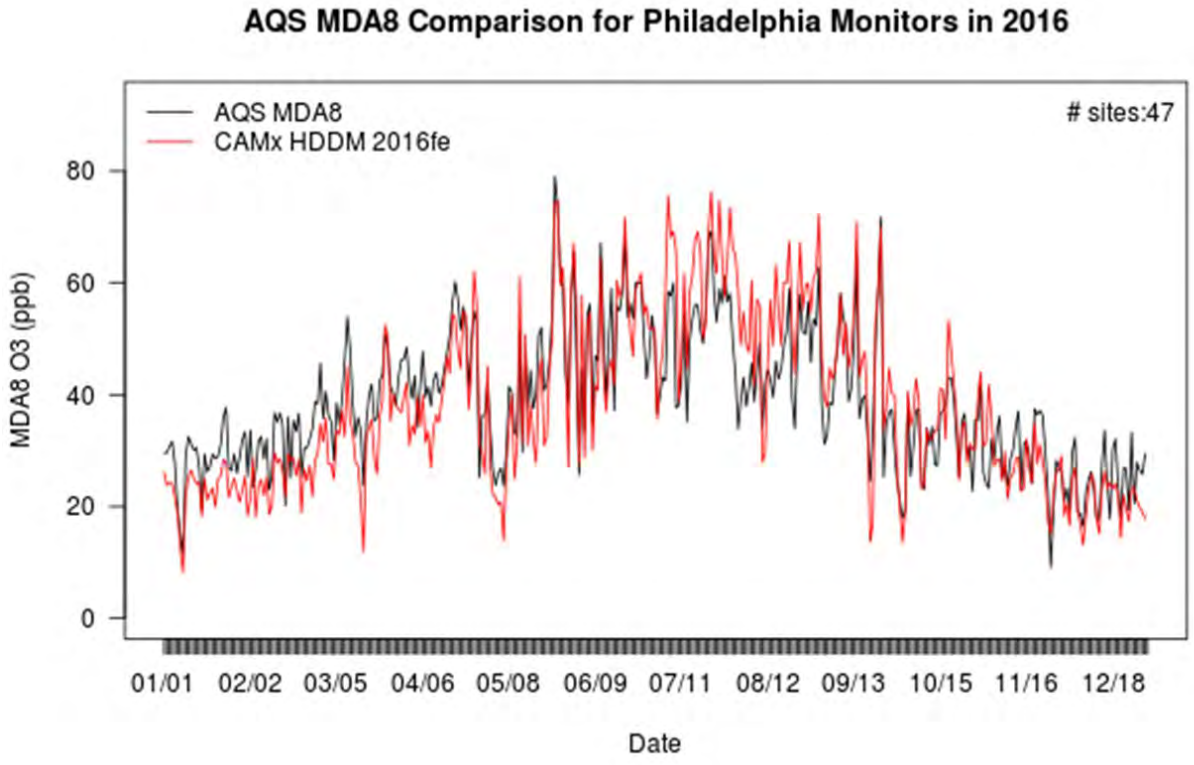


Figure 3C-18. Time series of monitored (black) and modeled (red) MDA8 O_3 at Philadelphia monitoring sites in 2016.

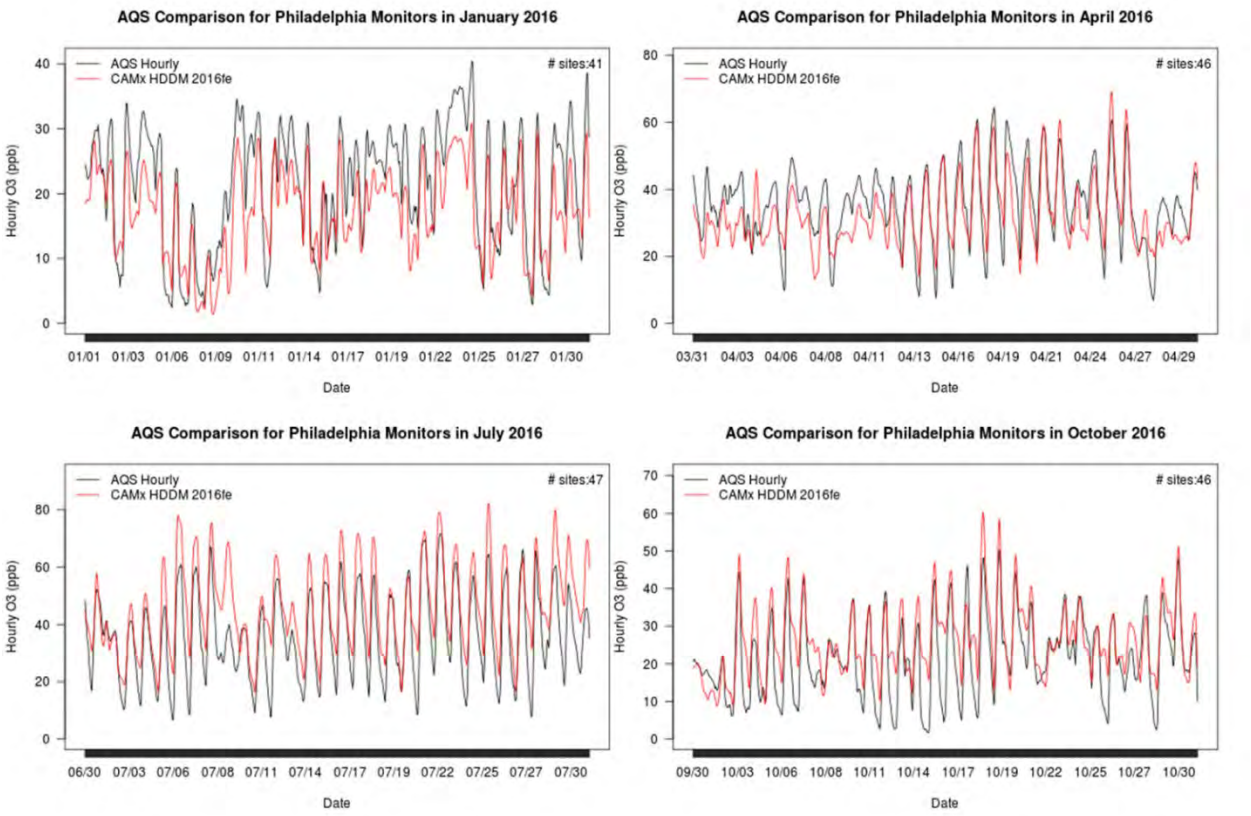


Figure 3C-19. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at Philadelphia monitoring sites for January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

3C.4.2.2 Operational Evaluation in the Southeastern U.S.

In the Southeast region, mean bias for MDA8 O_3 was generally less than ~5 ppb at most sites in all seasons, as indicated in Table 3C-8. The exception is winter, where there were only four days with measured MDA8 > 60 ppb and all were largely underpredicted. Spatial maps of normalized mean bias are shown in Figure 3C-20 through Figure 3C-23. Performance was best in the spring (slightly underestimated) and on high O_3 days in the summer/fall. Atlanta was the only one of the eight urban study areas located in the Southeast region.

Mean bias and normalized mean bias at Atlanta sites for the spring, summer, and fall months were typical of performance throughout the Southeast region, with much better performance in winter. The MDA8 O_3 time series (Figure 3C-24) shows that the model reasonably represents the variability occurring on high and low O_3 concentration days. The hourly time series plots (Figure 3C-25) also show reasonable model performance during daytime hours but some persistent overestimates of both nighttime and peak daytime O_3 occur, especially in July.

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

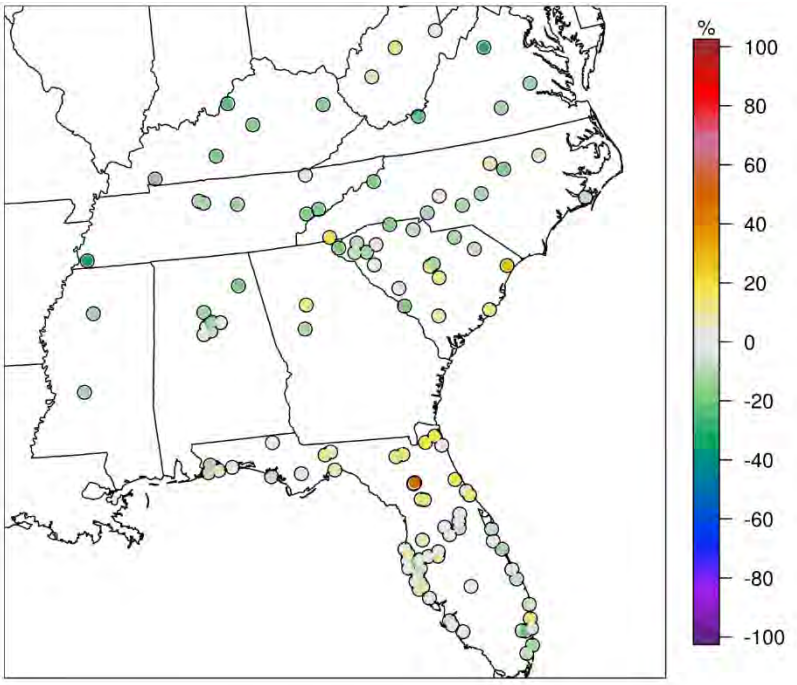
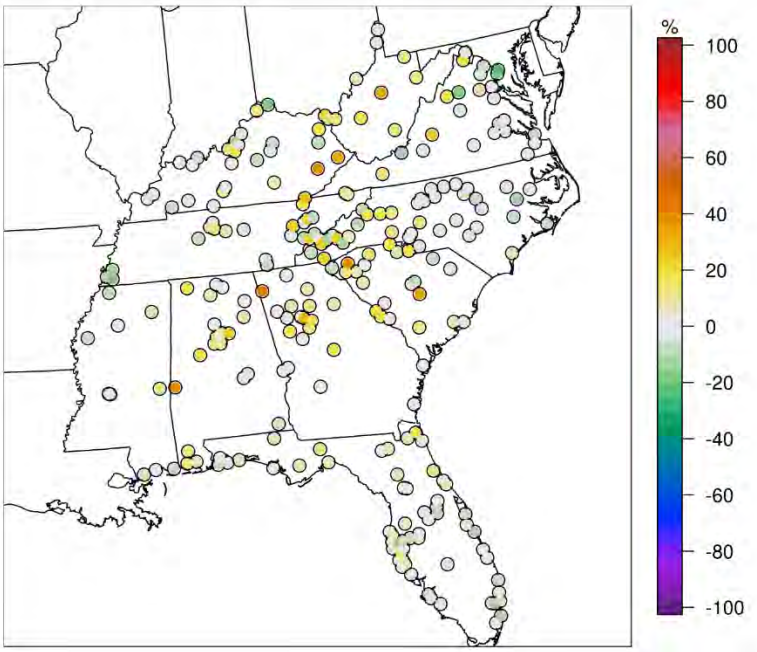
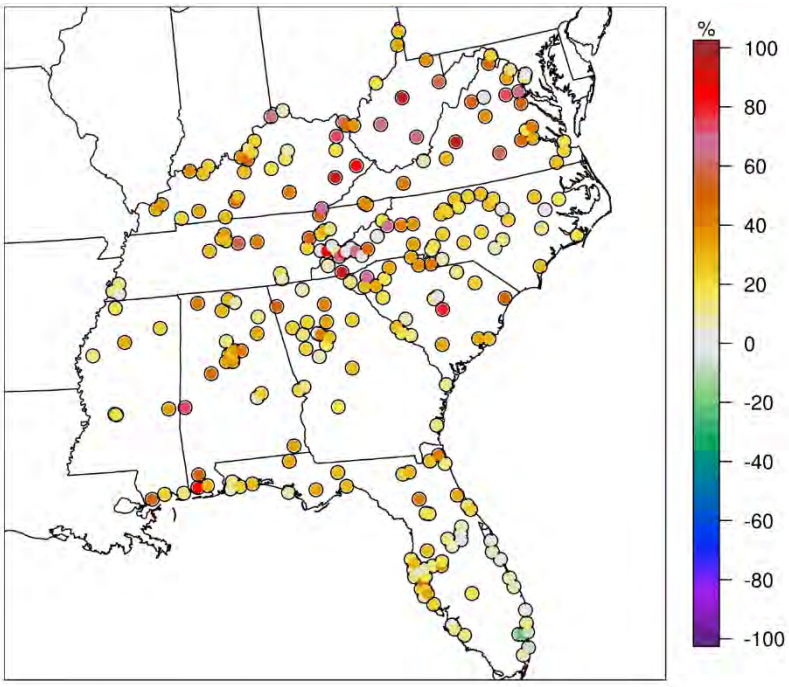


Figure 3C-20. Normalized mean bias for MDA8 O_3 in the Southeastern U.S., winter 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-18, -1.4, 5.1, 13, 44]

Figure 3C-21. Normalized mean bias for MDA8 O_3 in the Southeastern U.S., spring 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-22, 16, 27, 39, 130]

Figure 3C-22. Normalized mean bias for MDA8 O_3 in the Southeastern U.S., summer 2016.

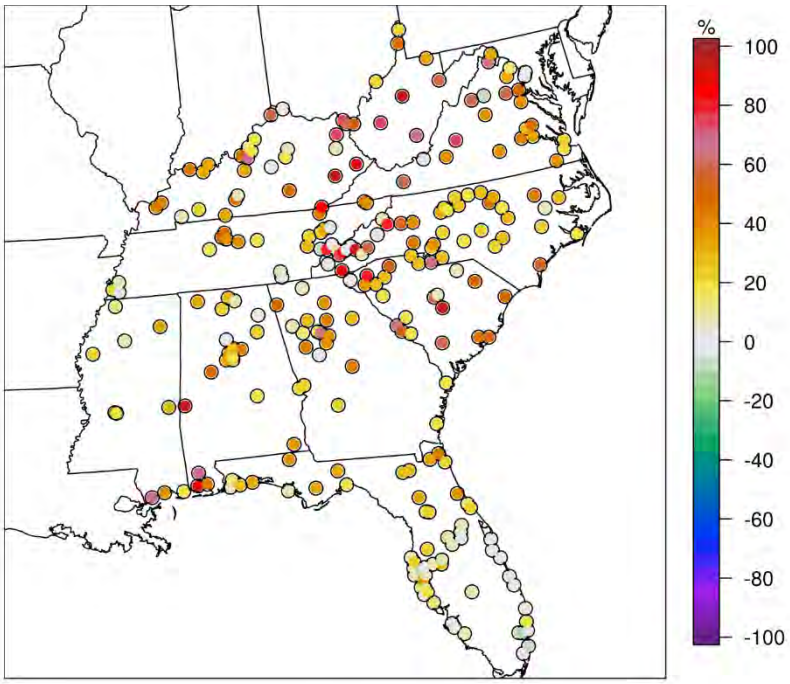


Figure 3C-23. Normalized mean bias for MDA8 O_3 in the Southeastern U.S., fall 2016.

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

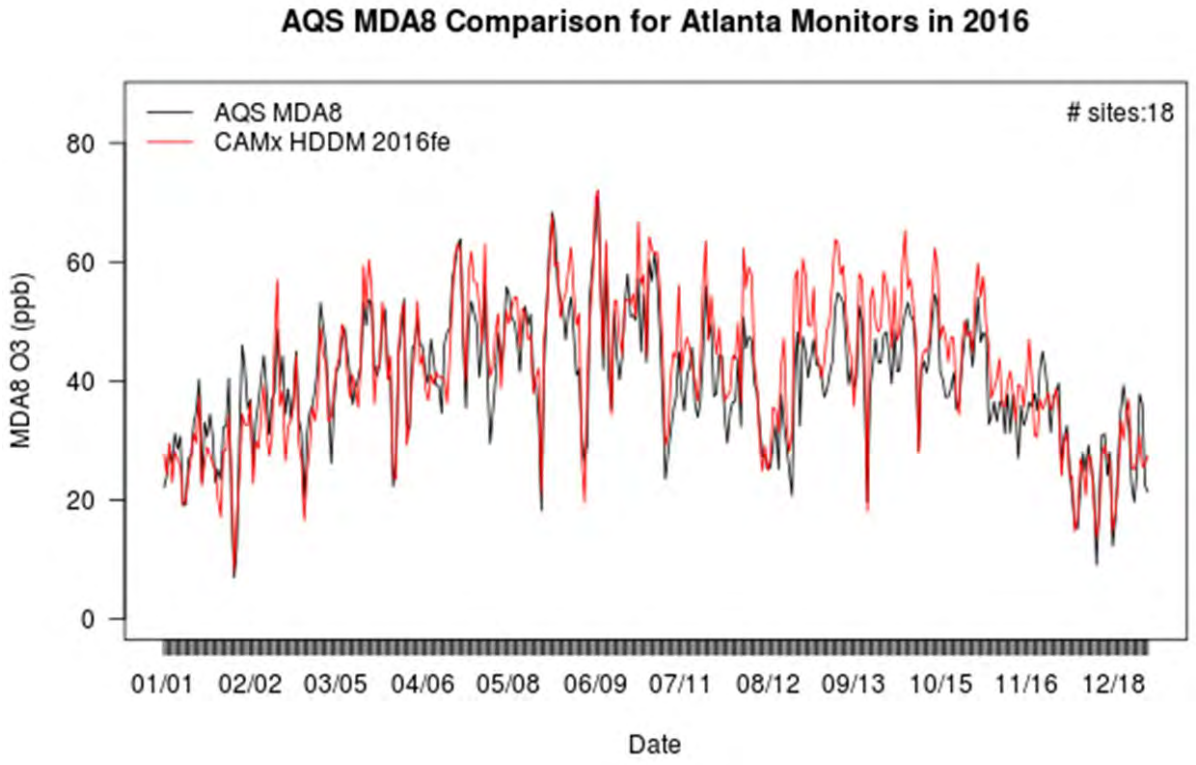


Figure 3C-24. Time series of monitored (black) and modeled (red) MDA8 O_3 at Atlanta monitoring sites in 2016.

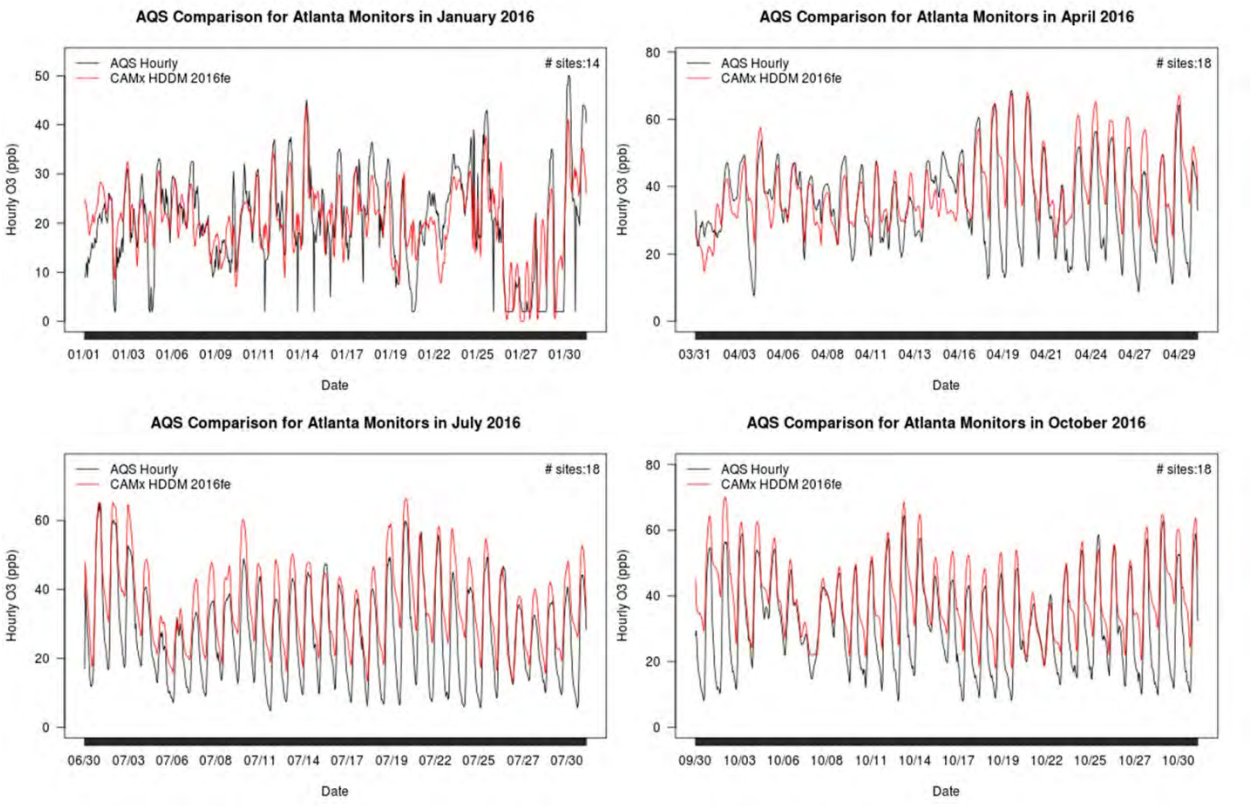


Figure 3C-25. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at Atlanta monitoring sites in January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

3C.4.2.3 Operational Evaluation in the Midwest U.S.

Mean bias for MDA8 O_3 in the Midwest region was around 6 ppb or less at most sites for all seasons (Table 3C-10), except for high O_3 days in spring. Normalized mean bias for MDA8 O_3 was less than 15%, except in the winter when it was somewhat higher (~20%). Normalized mean error was lowest on high O_3 days in spring, summer, and fall, even though bias performance was not notably better during these times. No distinct spatial patterns are apparent from the maps of normalized mean bias (Figure 3C-26 through Figure 3C-29). Detroit was the only one of the eight urban study areas located in the Midwest.

Detroit performance statistics for MDA8 O_3 were similar to those from the rest of the Midwest. However, under-estimates on high O_3 days were more pronounced in Detroit than in the rest of the region. The time series shows that the model accurately estimates both day and nighttime hourly O_3 in Detroit in April and July and generally captures the variations in MDA8 O_3 throughout the year, although the persistent under-estimate in winter-spring is evident (Figure 3C-30, Figure 3C-31).

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

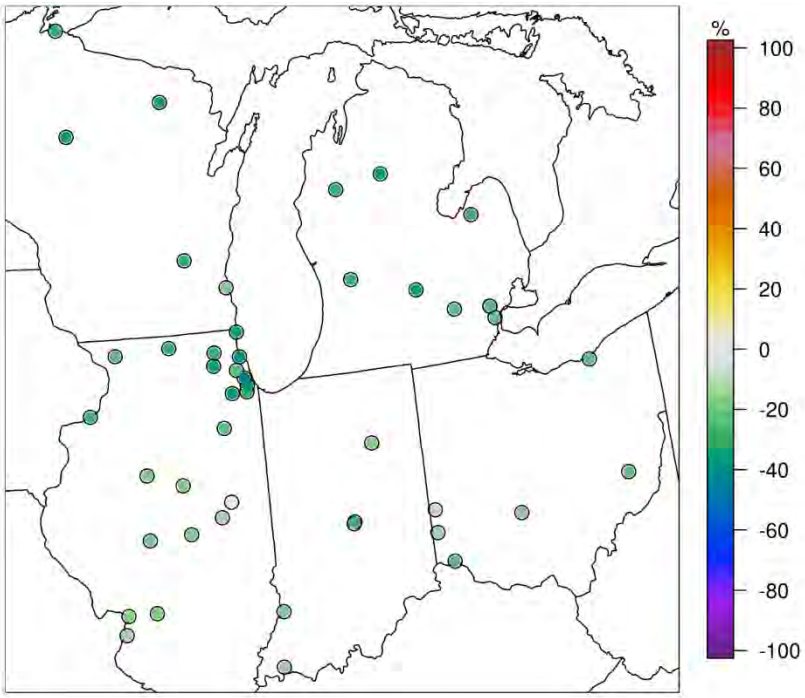
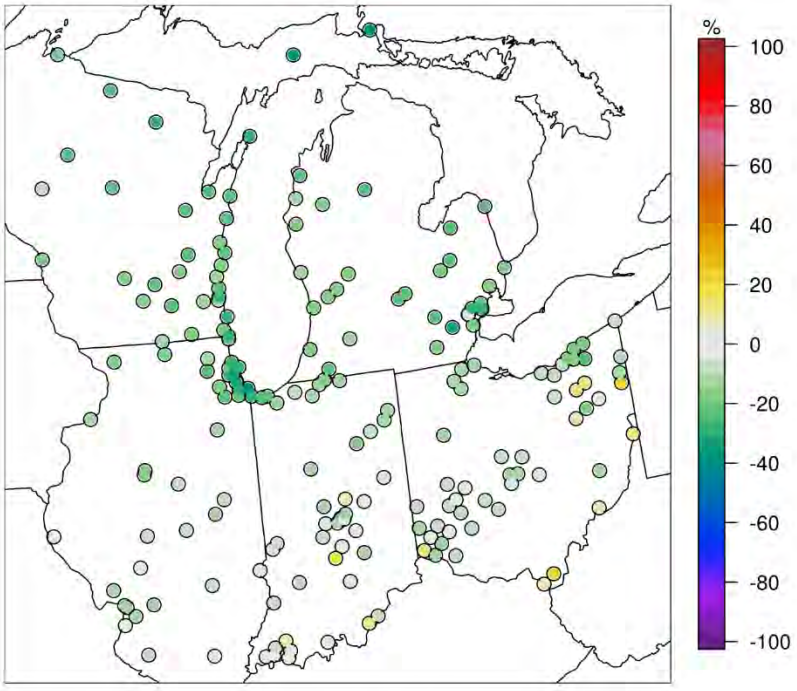
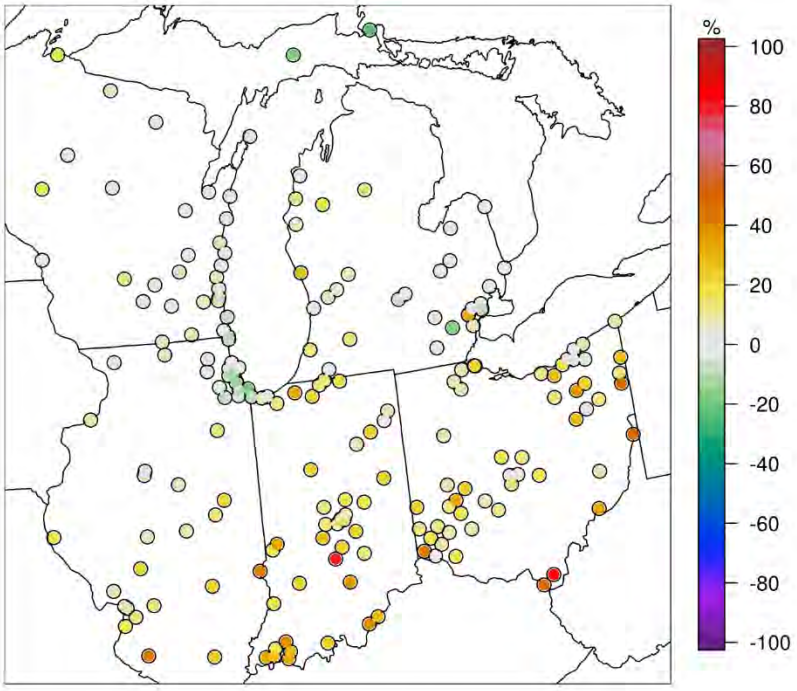


Figure 3C-26. Normalized mean bias for MDA8 O_3 in the Midwest U.S., winter 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-38, -18, -11, -4, 27]

Figure 3C-27. Normalized mean bias for MDA8 O_3 in the Midwest U.S., spring 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-20, 4.4, 11, 22, 87]

Figure 3C-28. Normalized mean bias for MDA8 O_3 in the Midwest U.S., summer 2016.

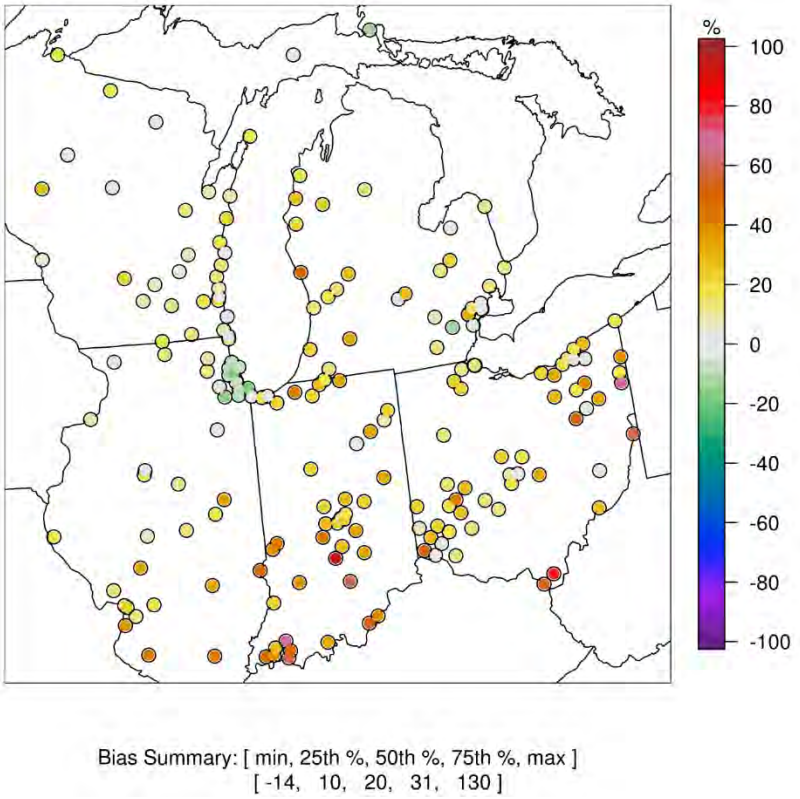


Figure 3C-29. Normalized mean bias for MDA8 O_3 in the Midwest U.S., fall 2016.

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

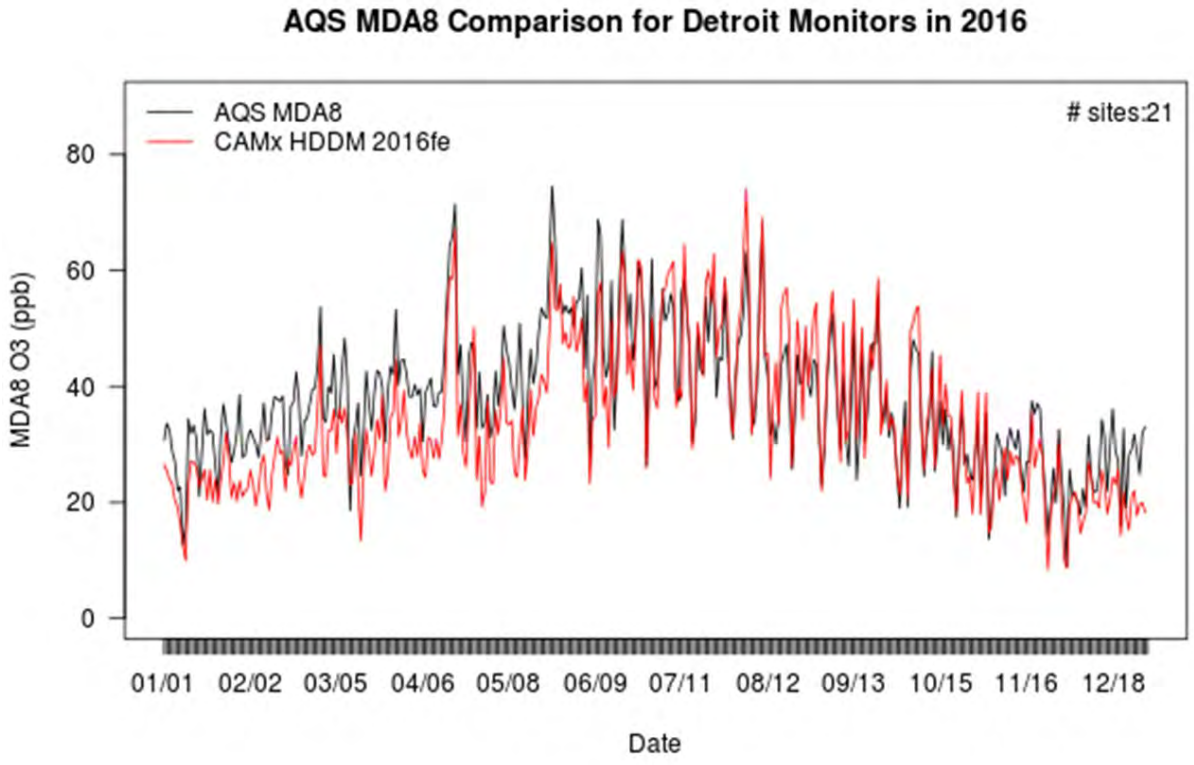


Figure 3C-30. Time series of monitored (black) and modeled (red) MDA8 O_3 at Detroit monitoring sites in 2016.

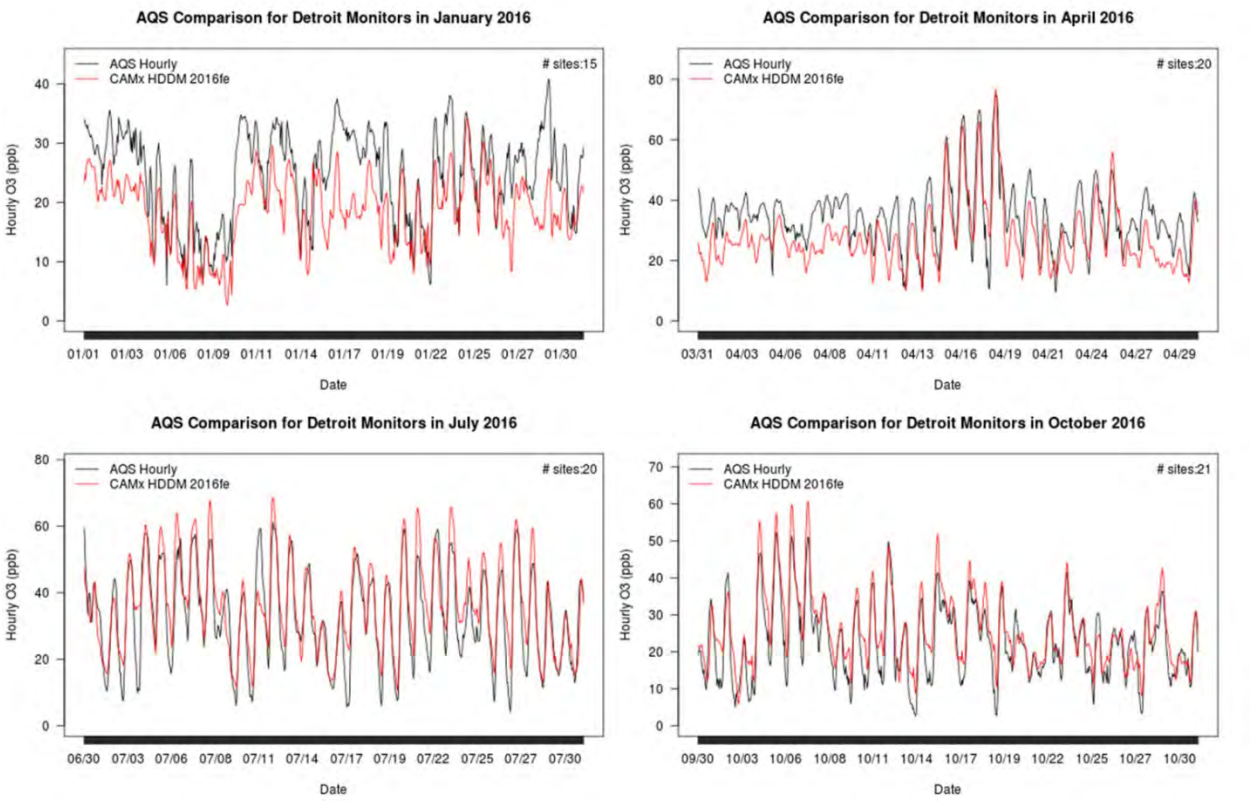


Figure 3C-31. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at Detroit monitoring sites in January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

3C.4.2.4 Operational Evaluation in the Central U.S.

Mean bias for MDA8 O_3 concentrations in the Central U.S. is within 4 ppb, except for high days in winter (-6 ppb) and spring (-7 ppb) (Table 3C-12). Normalized mean error is within 15%, except for days < 60 ppb in winter and summer (~18%). Spatial maps of normalized mean bias are shown in Figure 3C-32 through Figure 3C-35. Overall performance is best on lower O_3 days in spring and high O_3 days in summer and fall. St. Louis and Dallas were the only two of the eight study areas which are located in the Central U.S. region.

St. Louis mean bias for MDA8 was within 5 ppb for all days and seasons. A north-south gradient in NMB is apparent during both the winter and spring seasons in the maps shown in Figure 3C-32 and Figure 3C-33, with larger underestimates visible at higher latitude/more northerly monitors. Overall performance for St. Louis was best on high O_3 days in summer. The MDA8 time series shows reasonable agreement between CAMx and the monitor data for most of the year (Figure 3C-36), with underestimates in January and overestimates in July also apparent in the hourly time series (Figure 3C-37).

Performance statistics for MDA8 O_3 in Dallas were better than those for the broader region, with mean bias less than 5 ppb and normalized mean error just at or below 15% for all days and seasons. The MDA8 and hourly time series also show excellent model performance, with slightly underestimated peak day time O_3 in January (Figure 3C-38, Figure 3C-39). Overestimates of night-time O_3 in April and October, although these overpredictions are less pronounced in Dallas compared to many of the other urban study areas examined in the assessment.

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

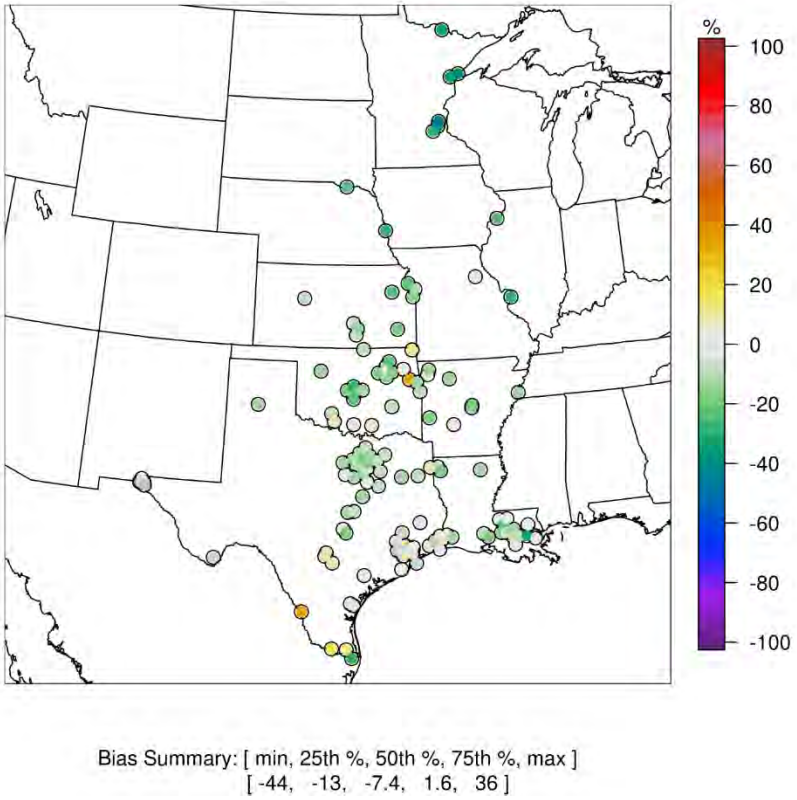
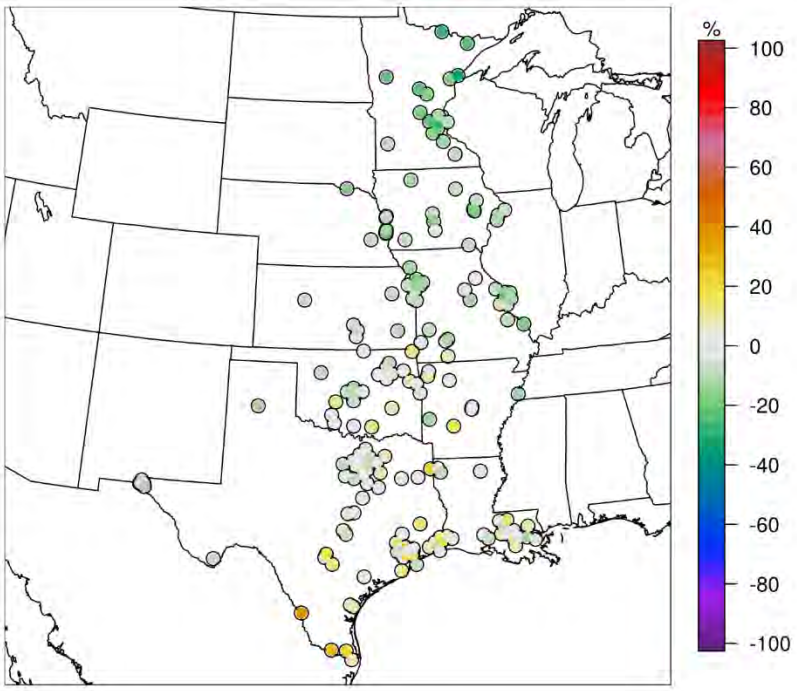
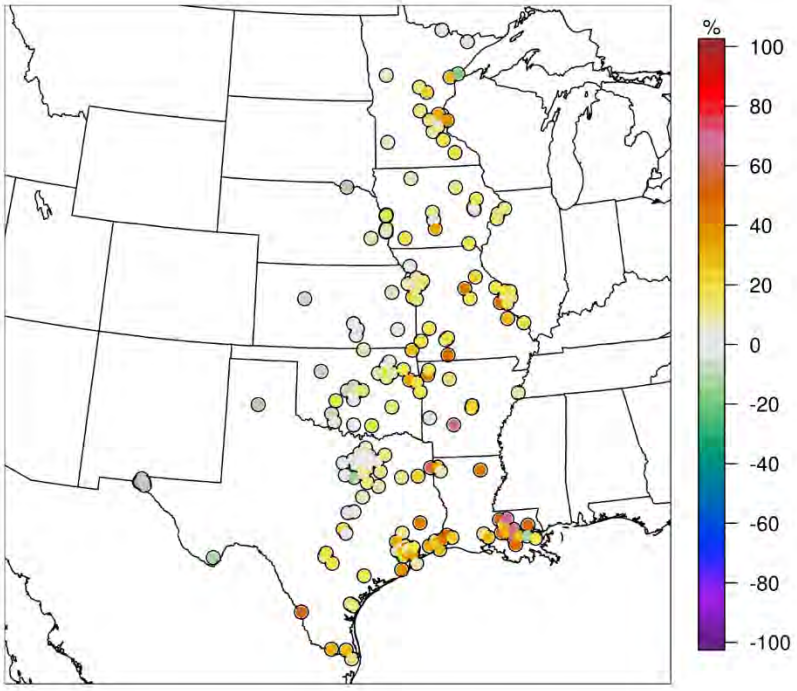


Figure 3C-32. Normalized mean bias for MDA8 O_3 in the Central U.S., winter 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-28, -7.6, -0.93, 6.6, 41]

Figure 3C-33. Normalized mean bias for MDA8 O_3 in the Central U.S., spring 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-21, 6.9, 16, 24, 72]

Figure 3C-34. Normalized mean bias for MDA8 O_3 in the Central U.S., summer 2016.

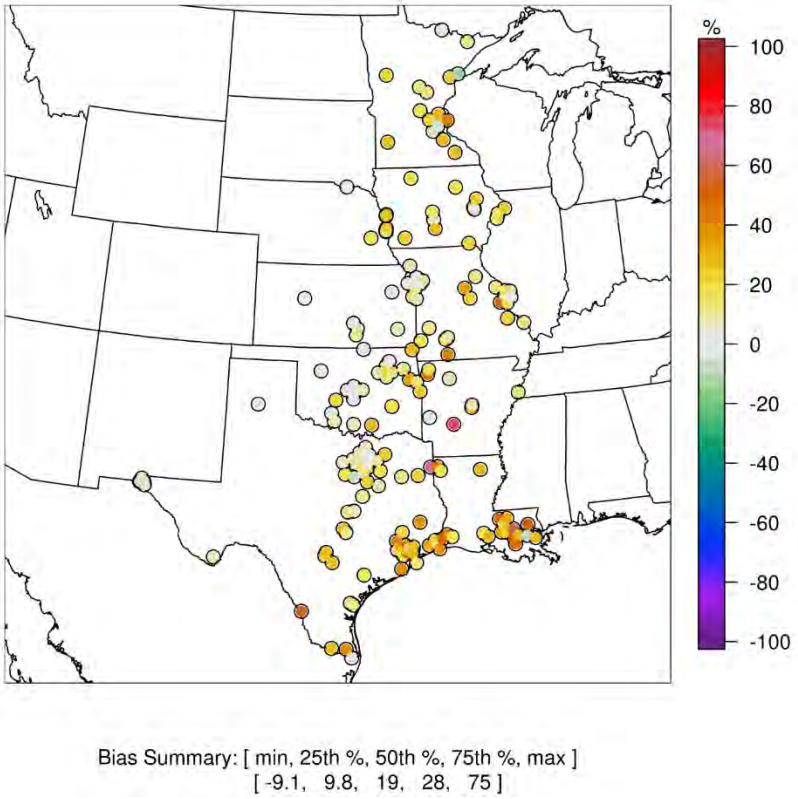


Figure 3C-35. Normalized mean bias for MDA8 O_3 in the Central U.S., fall 2016.

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

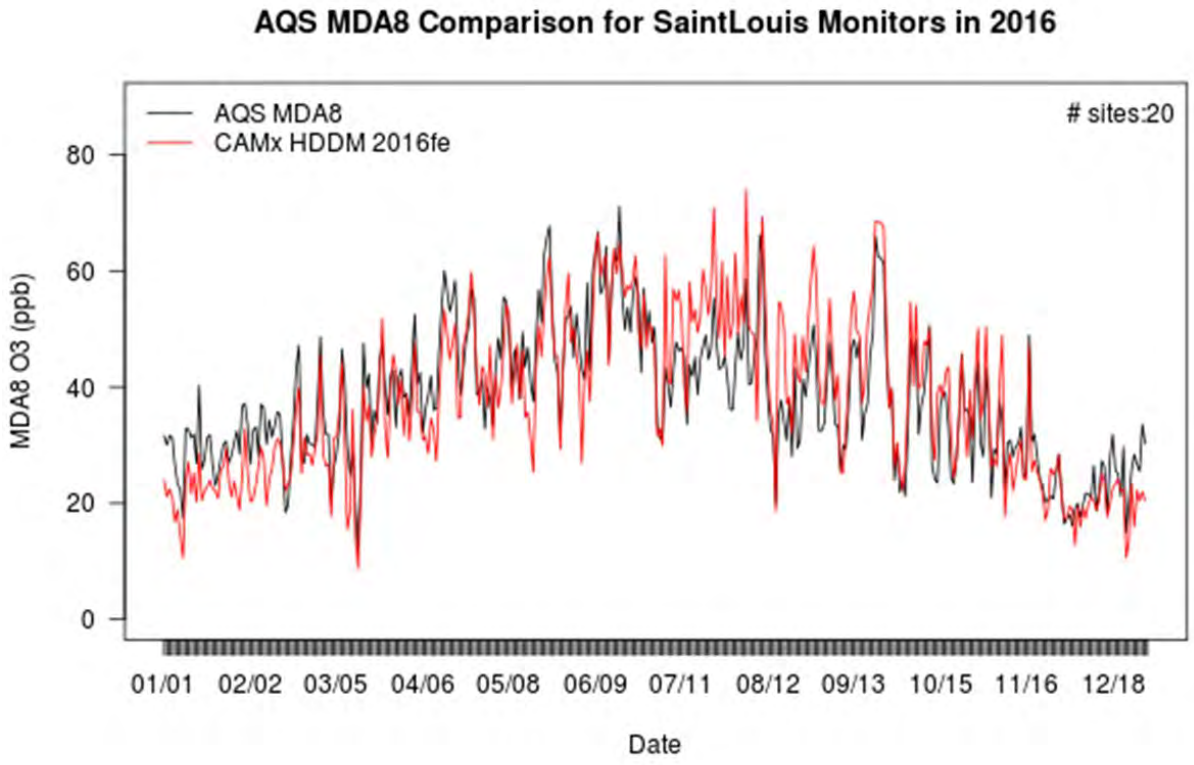


Figure 3C-36. Time series of monitored (black) and modeled (red) MDA8 O_3 at St. Louis monitoring sites in 2016.

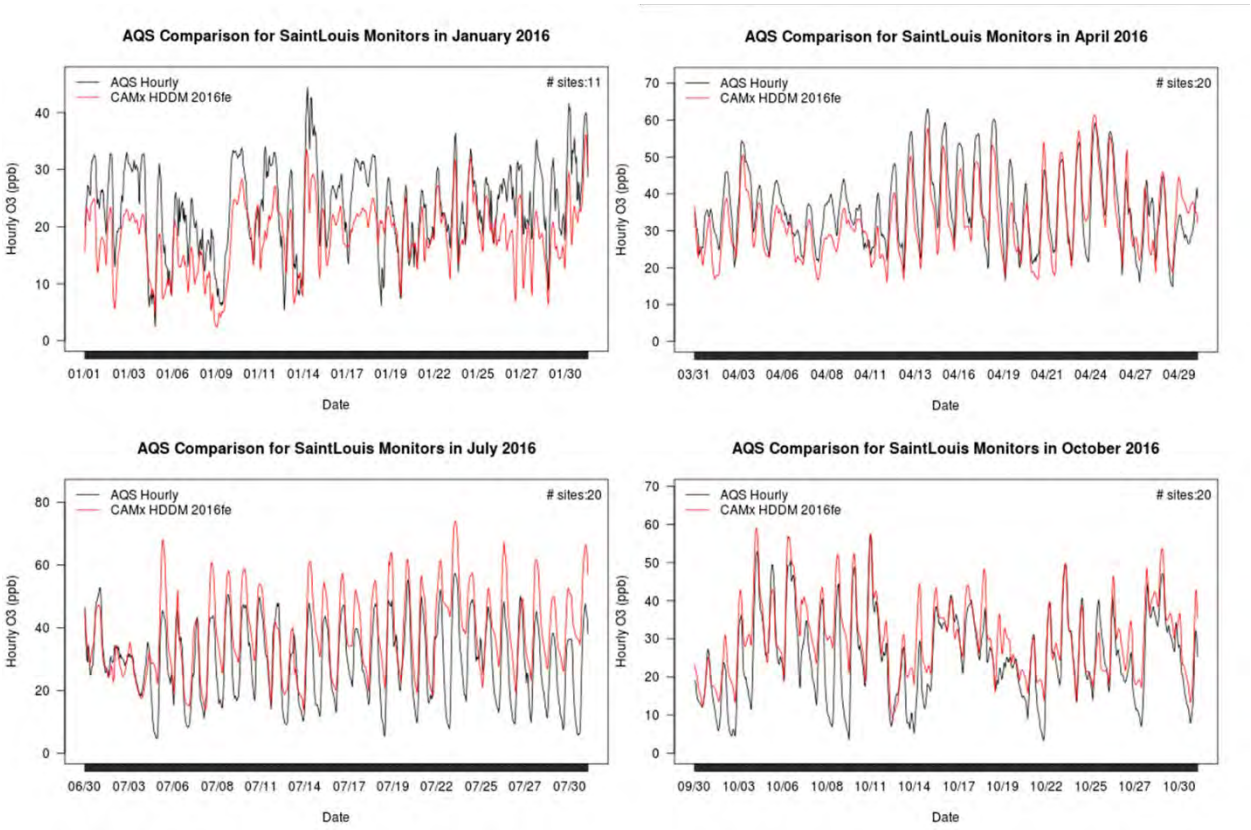


Figure 3C-37. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at St. Louis monitoring sites in January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

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

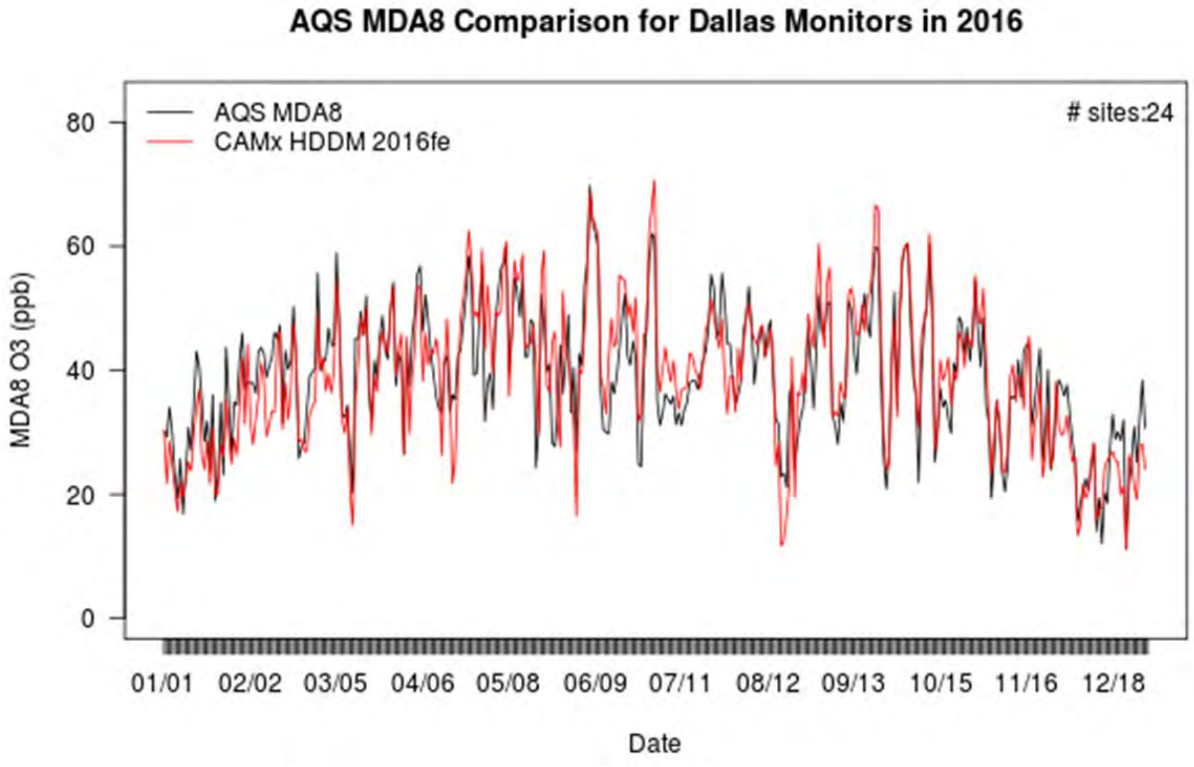


Figure 3C-38. Time series of monitored (black) and modeled (red) MDA8 O_3 at Dallas monitoring sites in 2016.

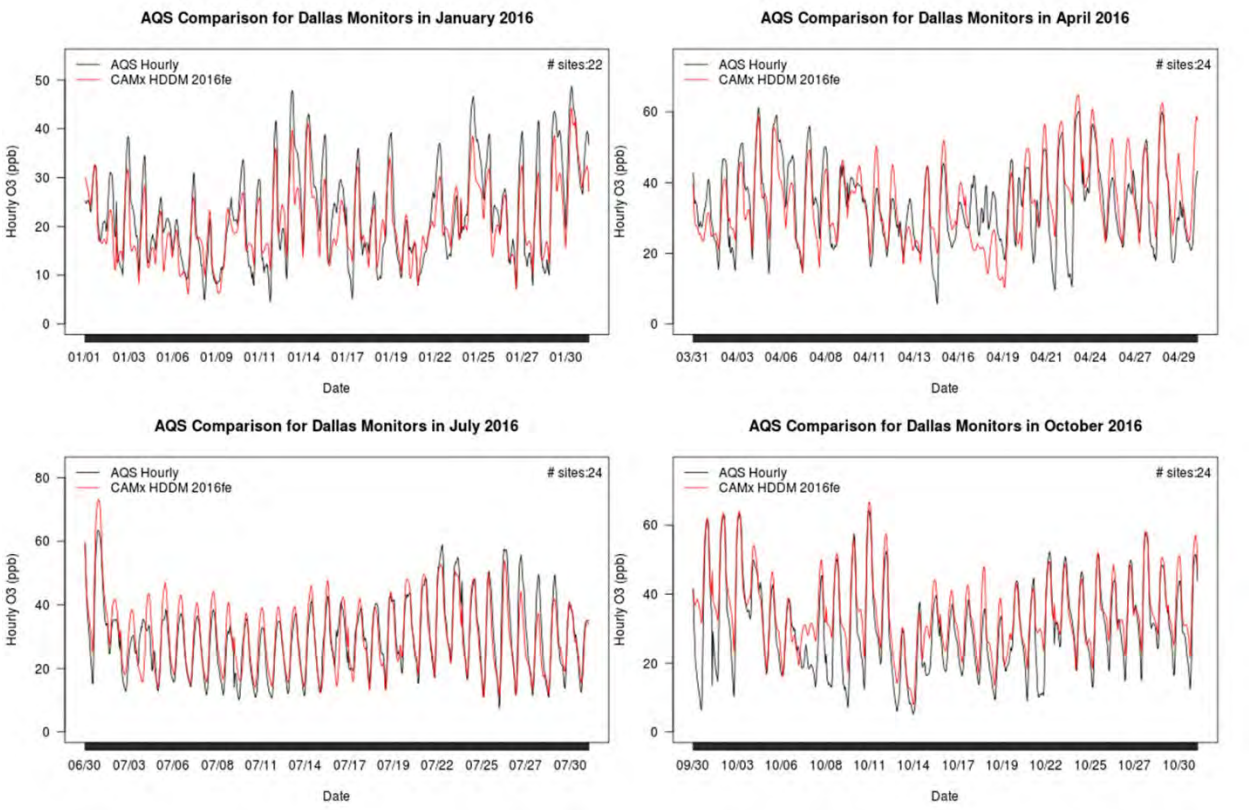


Figure 3C-39. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at Dallas monitoring sites in January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

3C.4.2.5 Operational Evaluation in the Western U.S.

Model statistics for MDA8 O_3 in the Western U.S. are best on low O_3 days in summer and fall (Table 3C-15). High wintertime observations were substantially underestimated by the model with an average MB of -26 but likely for different reasons. The high days in Riverside California are probably due to traditionally understood O_3 formation that occurs on warm sunny days. The high O_3 concentrations in Wyoming are an example of wintertime O_3 formation that occurs during cold pool meteorology events which have substantial snow cover and extreme temperature inversions and are still an active area of research. Some spatial patterns in normalized mean bias are apparent in the winter and in the summer (Figure 3C-40 through Figure 3C-43), with overestimates on the West Coast and underestimates in the Intermountain West. Two urban study areas are located in the Western U.S. and are evaluated in this section: Sacramento and Phoenix.

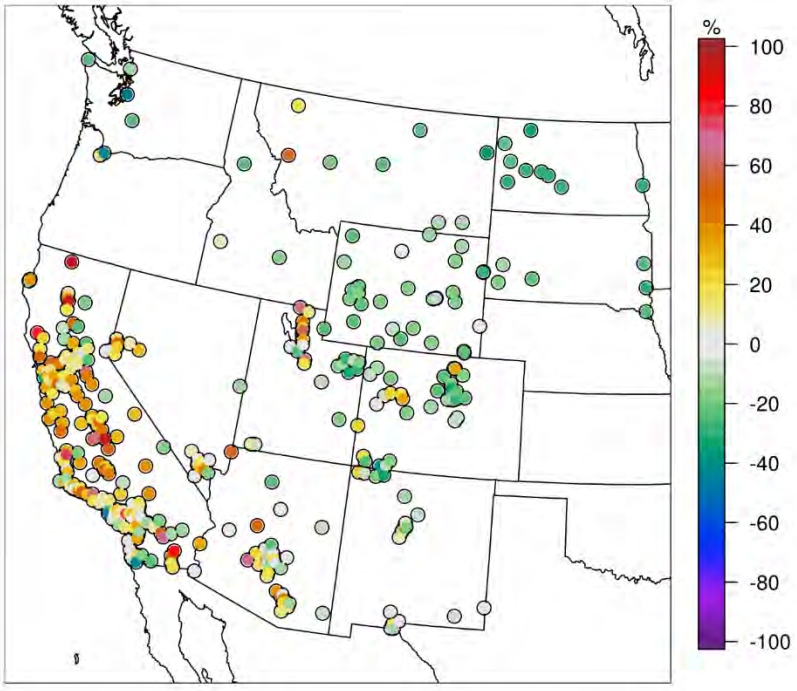
The model performance for MDA8 O_3 values in the Sacramento study area was best on lower O_3 days in summer and fall (Figure 3C-44). In Sacramento there were no days during the

winter with measured MDA8 O_3 > 60 ppb. Normalized mean error is at or below 15% for all seasons except winter. Hourly time series show good agreement in Sacramento, except for winter when the model does not capture very much of the day to day variability in O_3 concentrations Figure 3C-45).

While normalized mean error was at or less than 15% in Phoenix on all days in all seasons, the MDA8 time series shows frequent underestimates in winter-spring as well as overestimates in summer-fall (Figure 3C-46). The hourly time series also show that though the model captures some of the overnight O_3 patterns in Phoenix, night time O_3 is significantly overestimated, particularly in January and October (Figure 3C-47).

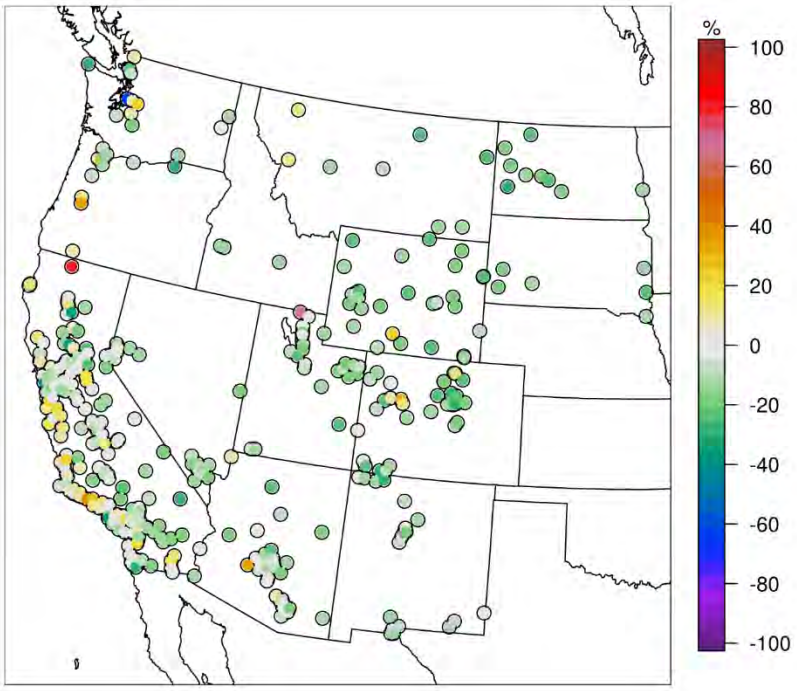
Table 3C-15. CAMx model performance at monitoring sites in the Western U.S.

		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



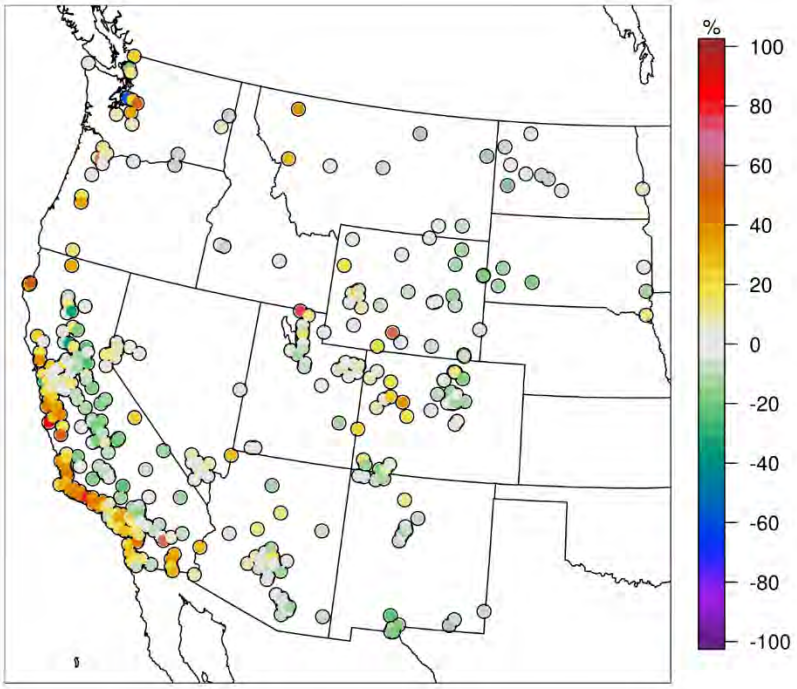
Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-47, -12, 8.3, 27, 110]

Figure 3C-40. Normalized mean bias for MDA8 O_3 in the Western U.S., winter 2016.



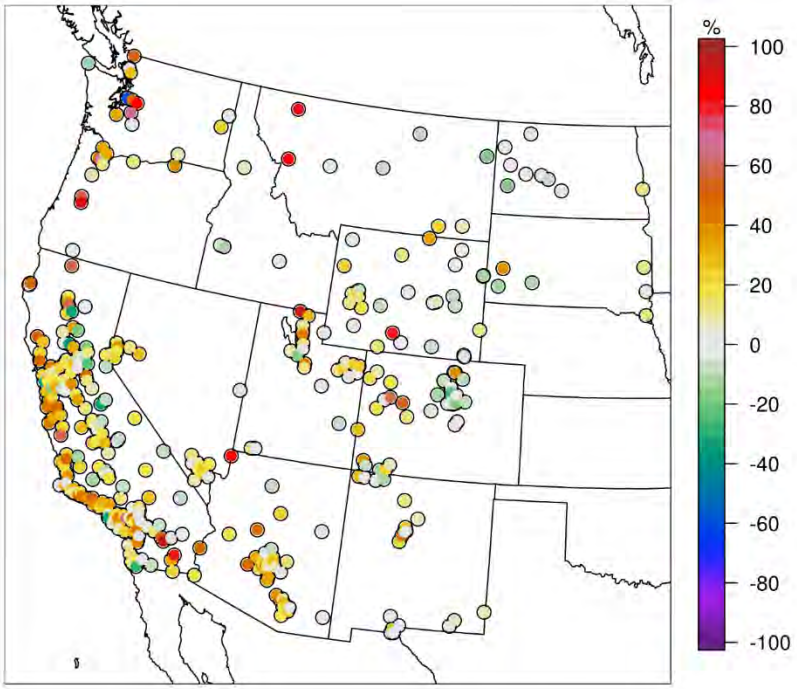
Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-60, -12, -5.7, 3.1, 82]

Figure 3C-41. Normalized mean bias for MDA8 O_3 in the Western U.S., spring 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-55, -5.2, 3.2, 16, 90]

Figure 3C-42. Normalized mean bias for MDA8 O_3 in the Western U.S., summer 2016.



Bias Summary: [min, 25th %, 50th %, 75th %, max]
[-57, 1.5, 16, 30, 120]

Figure 3C-43. Normalized mean bias for MDA8 O_3 in the Western U.S., fall 2016.

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

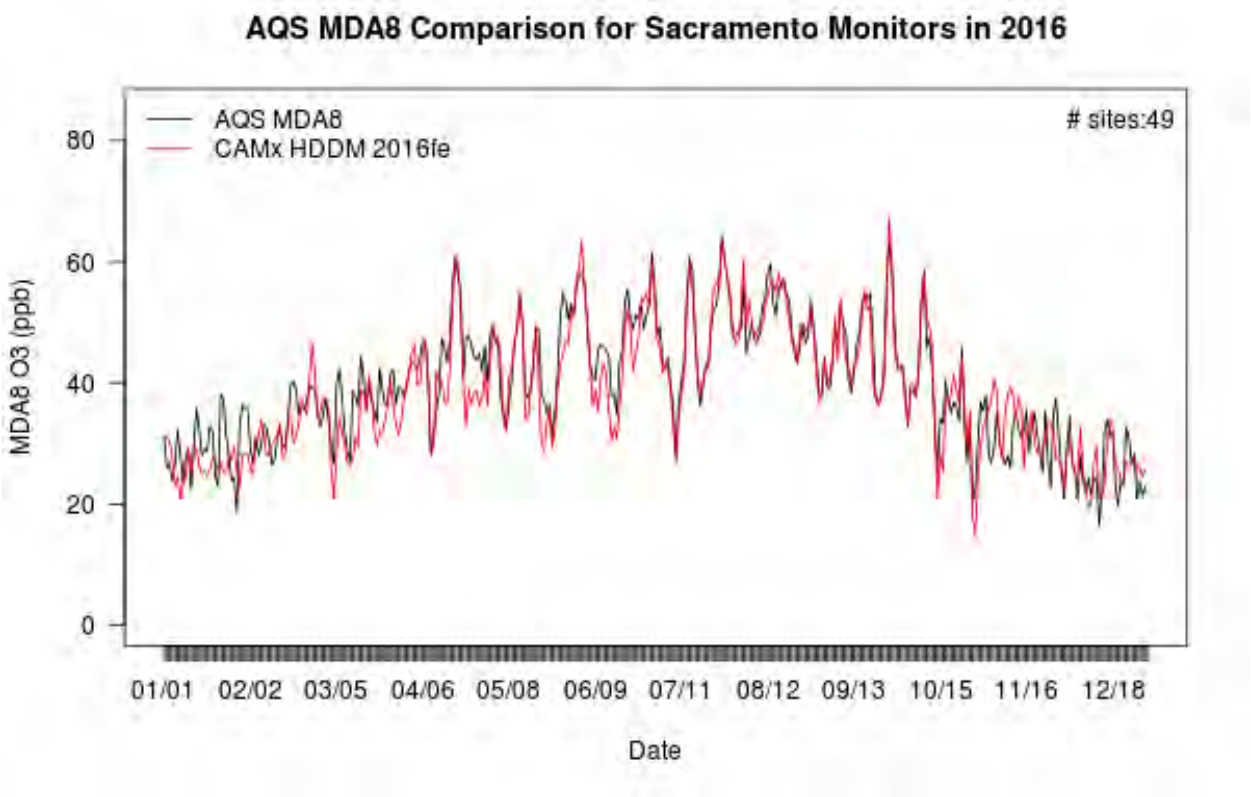


Figure 3C-44. Time series of monitored (black) and modeled (red) MDA8 O_3 at Sacramento monitoring sites in 2016.

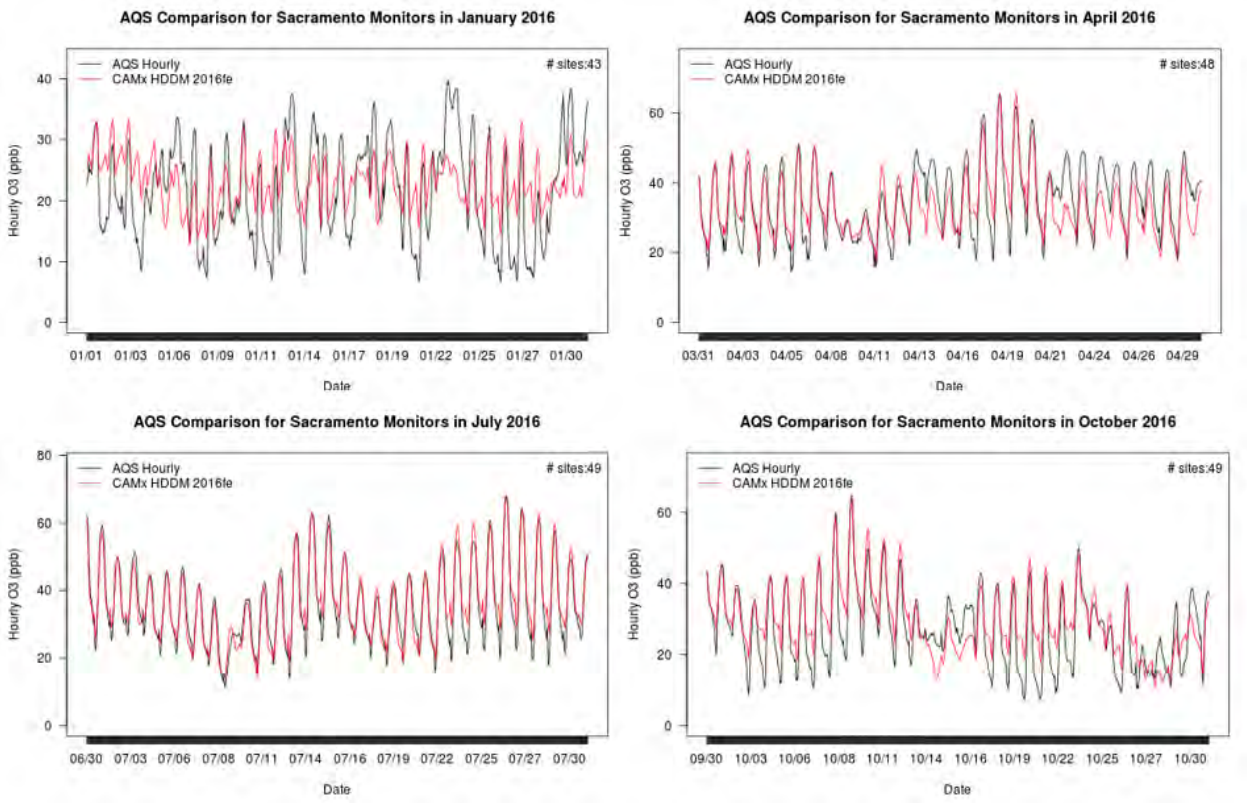


Figure 3C-45. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at Sacramento monitoring sites in January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

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

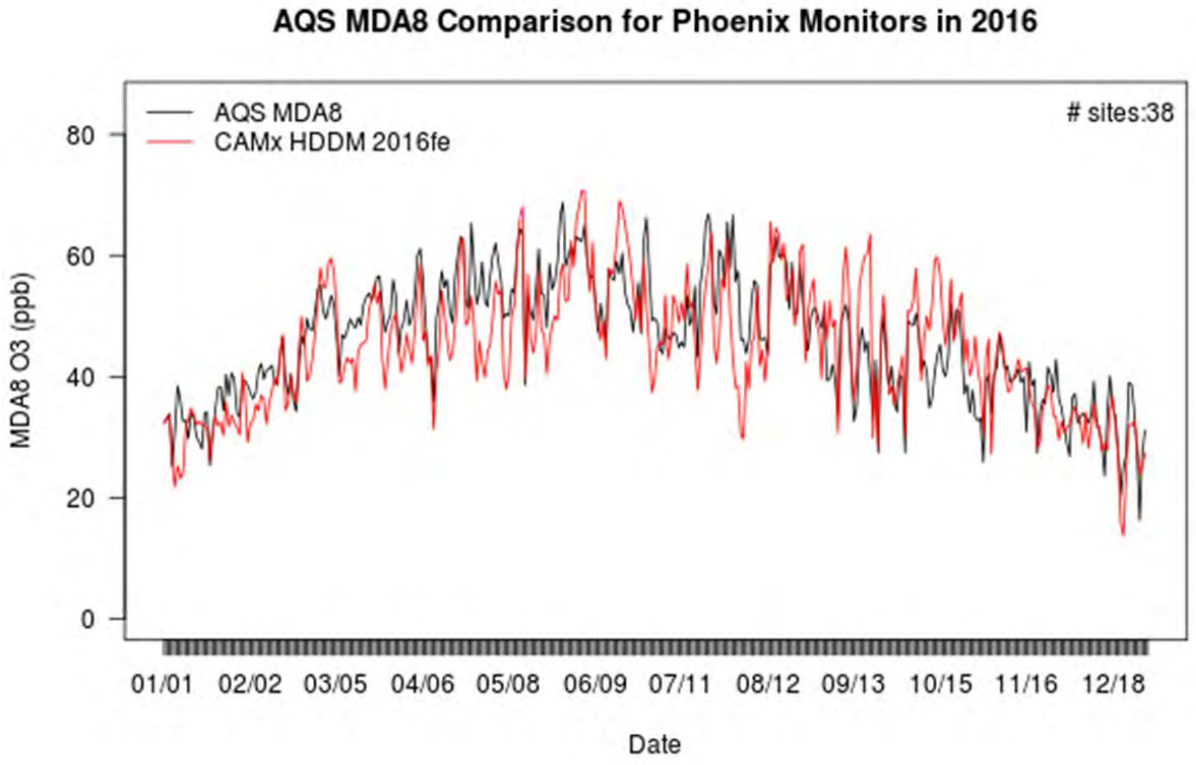


Figure 3C-46. Time series of monitored (black) and modeled (red) MDA8 O_3 at Phoenix monitoring sites in 2016.

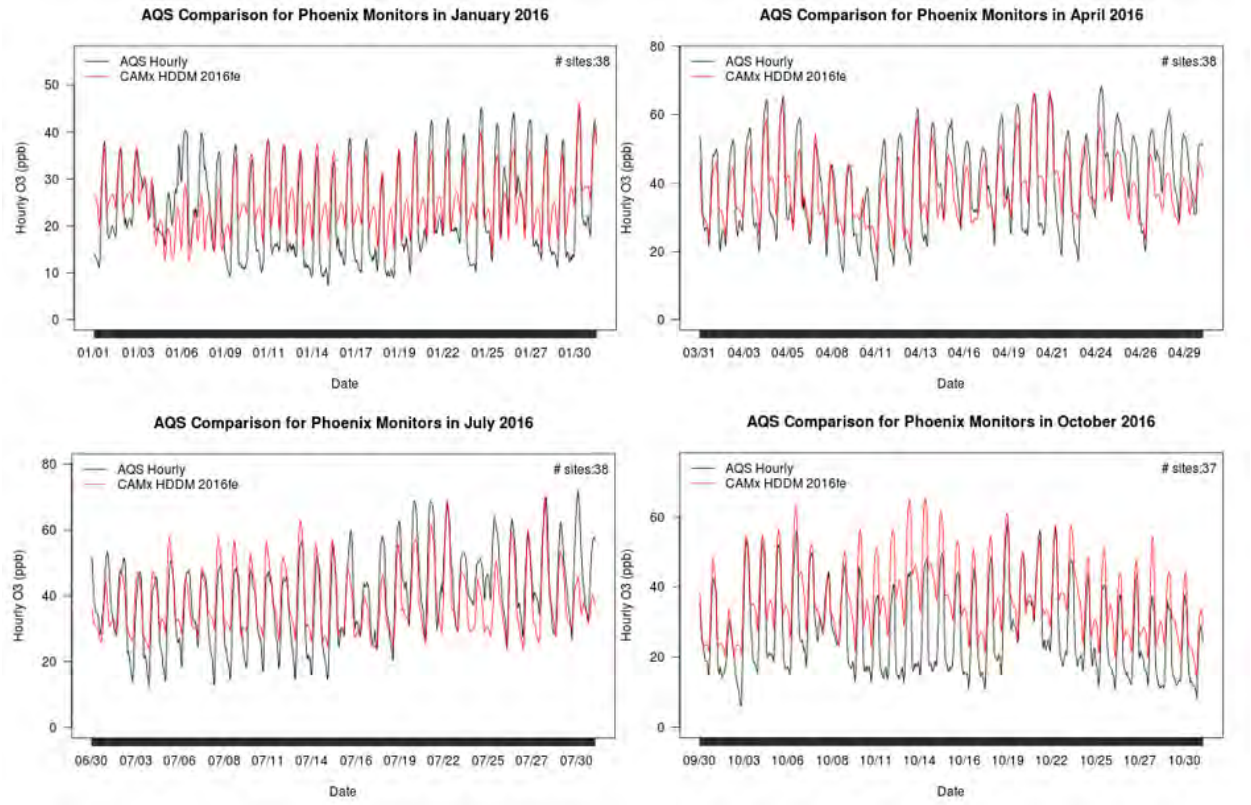


Figure 3C-47. Time series of monitored (black) and modeled (red) hourly O_3 concentrations at Phoenix monitoring sites in January (top left), April (top right), July (bottom left), and October (bottom right) 2016.

3C.5 AIR QUALITY ADJUSTMENT TO MEET CURRENT AND ALTERNATIVE AIR QUALITY SCENARIOS

3C.5.1 Overview of the Higher Order Direct Decoupled Method (HDDM)

In this section we present a model-based O_3 adjustment methodology that allows for adjustments to observed hourly O_3 concentrations to reflect the expected impacts of changes in NO_X emissions. This methodology uses the CAMx model, described above in section 3C.4, instrumented with the Higher order Decoupled Direct Method (HDDM) - a tool that generates modeled sensitivities of O_3 to emissions changes. The outputs of the HDDM are used to estimate the distribution of O_3 concentrations associated with just meeting three air quality scenarios (O_3 monitor design values of 75 ppb, 70 ppb, and 65 ppb) within multiple urban study areas. The HDDM sensitivities are applied to ambient air measurements of O_3 to estimate how O_3 concentrations would respond to changes in U.S. anthropogenic emissions. This approach, based on Simon et al. (2013), was applied previously for the 2015 O_3 NAAQS review.

The CAMx photochemical modeling incorporates emissions from non-anthropogenic sources and anthropogenic emissions from sources in the U.S and in the portions of Canada and Mexico within the regional modeling domain. Pollution from sources in other locations within and outside of North America is included as transport into the boundary of the modeling domain.

3C.5.1.1 Capabilities

Chemical transport models, such as CAMx, simulates physical and chemical processes in the atmosphere to predict 3-dimensional (3-D) gridded pollutant concentrations. These models account for the impacts of emissions, transport, chemistry, and deposition on spatially and temporally varying pollutant concentrations. Required model inputs include time-varying emissions and meteorology fields, time varying concentrations of pollutants at the boundaries of the model domain (i.e. boundary conditions), and a characterization of the 3-D field of chemical concentrations to initialize the model (i.e. initial conditions).

Beyond modeling the ambient air concentrations of O_3, chemical transport models can be used to estimate the response of ambient air O_3 concentrations to changes in emissions. One technique to simulate the response of O_3 to emissions changes, the brute force method, requires the modeler to explicitly model this response by directly altering the emissions inputs in the model simulation. This technique provides an estimate of the O_3 concentration at the altered emission level, but often does not provide accurate information regarding the response of O_3 to other levels of emissions since the chemistry for O_3 formation is nonlinear. Therefore, when using only the brute force method, a new model simulation would need to be performed for every emissions scenario under consideration.

Other analytical techniques have been developed to estimate the O_3 response to emission perturbations without performing multiple simulations. One such method is termed the Decoupled Direct Method (DDM) (Dunker, 1984). DDM, solves for sensitivity coefficients which are defined as the partial derivative of the atmospheric diffusion equations that underly the model calculations, Equations (3C-1) and (3C-2).

$$s_{ij}(t) = \frac{\partial C_i(t)}{\partial p_j}$$

Equation (3C-1)

$$S_{ij}(t) = \tilde{P}_j \frac{\partial C_i(t)}{\partial p_j} = \tilde{P}_j \frac{\partial C_i(t)}{\partial (\epsilon_j \tilde{P}_j)} = \frac{\partial C_i(t)}{\partial \epsilon_j}$$

Equation (3C-2)

Here, $S_{ij}(t)$, the sensitivity, gives the change in model concentration, C_i, (for instance O_3 concentration) with an incremental change in any input parameter, p_j (in this case emissions). Equation (3C-2) allows us to normalize the sensitivity coefficient, $S_{ij}(t)$, so that it shows response in relative terms for the input rather than in absolute units. Therefore, $\tilde{P}_j$ (x,t) is the normalized input and ε_j is a scaling variable (Yang et al., 1997). In general terms, the sensitivity coefficient tells us how a model output (O_3 concentration) will change if a model input (emissions of NO_X or VOC) is perturbed. This first order sensitivity coefficient, $S_{ij}(t)$ is quite suitable for small perturbations, but gives a linear response which is unlikely to represent the results of large perturbations in very nonlinear chemical environments. Second (and third) order derivatives can be calculated to give higher order sensitivity coefficients (Hakami et al., 2003). Higher order sensitivity coefficients give the curvature and inflection points for the response curve and can capture the nonlinearities in the response of O_3 to emissions changes. Using Higher order DDM (HDDM) allows for the sensitivities to be more appropriately applied over larger emissions perturbations. Hakami et al. (2003) report that for an application in California, HDDM gave reasonable approximations of O_3 changes compared to that generated using brute force emissions reductions of up to 50% using the first three terms of the Taylor series expansion, Equation (3C-3).

$$C(+\Delta\epsilon) = C(0) + \Delta\epsilon S(0) + \frac{\Delta\epsilon^2}{2} S^2(0) + \ \ldots + \frac{\Delta\epsilon^n}{n!} S^n(0) + R_{n+1}$$

Equation (3C-3)

Here $\Delta\epsilon$ represents the relative change in emissions (for instance $\Delta\epsilon$ = -0.2 would be equivalent to reducing emissions by 20%), $S^n(0)$ is the n^{th} order sensitivity coefficient, C(0) is the concentration under baseline conditions (no perturbation in emissions) and R_{n+1} is a remainder term.

A variant of DDM called DDM-3D has been implemented into several chemical transport models, including CAMx, for both O_3 and particulate matter (PM) predictions (Cohan et al., 2005, Hakami et al., 2003, Napelenok et al., 2011, Dunker, 1984,Yang et al., 1997, Koo et al., 2007, Zhang et al., 2012). These implementations allow the modeler to define the parameters for which first and higher order sensitivities will be calculated. For instance, the sensitivity can be calculated for emissions from a specific source type, for emissions in a specific geographic region, and for emissions of a single O_3 precursor or for multiple O_3 precursors. In addition, sensitivities can be calculated to boundary conditions, initial conditions, and various other model inputs. Sensitivities to different sets of parameters can be calculated in a single model simulation

but computation time increases as the number of sensitivities increases. Outputs from an HDDM simulation consist of time varying 3-D fields of first and second order sensitivities.

3C.5.1.2 Limitations

For the purposes of the O_3 NAAQS analysis, an HDDM-based approach is well-suited given its ability to 1) capture the non-linearity of O_3 response to emissions changes, 2) characterize different O_3 responses at different locations (downtown urban versus downwind suburban) and at different times of day, allowing us to incorporate temporal and spatial variations in response into the O_3 adjustment methodology, and 3) explicitly account for physical and chemical processes influencing predicted sensitivities such as background O_3 sources. However, in addition to the many potential benefits of using HDDM to understand and characterize O_3 response to emissions changes, there are several limitations.

First, HDDM encompasses all of the uncertainties of the base photochemical model formulation and inputs. So, uncertainties in how the physical and chemical processes are treated in the model and in the model inputs propagate to the HDDM results. Also, HDDM can capture response to larger emissions perturbations than DDM but it is still most accurate for small perturbations. The larger the relative change in emissions, the less likely that the HDDM sensitivities will properly capture the change in O_3 that would be predicted by using brute force emission reductions. Several studies have reported reasonable performance of HDDM for O_3 up to 50% emissions perturbations (Hakami et al., 2004, Hakami et al., 2003, Cohan et al., 2005), but the magnitude of perturbation over which HDDM will give accurate estimates will depend on the specific modeling episode, size of the model domain, emissions and meteorological inputs, and the size of the emissions source to which the sensitivity is being calculated. In this work, we applied sensitivities derived from model simulations done under varying NO_X levels (see section 3C.5.2.2) and found that using this technique we were able to replicate O_3 concentrations estimated using brute force emission reductions with HDDM sensitivities for up to 90% NO_X emissions reductions with a mean bias of less than 3 ppb and a mean error of less than 4 ppb.

3C.5.2 Using CAMx/HDDM to Adjust Monitored Ozone Concentrations

3C.5.2.1 Conceptual Framework

This section outlines the methodology in which we apply CAMx/HDDM to estimate hourly O_3 concentrations that might result from just meeting three air quality scenarios (75 ppb, 70 ppb, and 65 ppb). These methods closely follow those documented in Simon et al. (2013) and the risk and exposure assessment performed in the 2015 O_3 NAAQS review (U.S. EPA, 2014). As part of the methodology, photochemical modeling results are not used in an absolute sense,

but instead are applied to modulate ambient air measurements, thus tying estimated O_3 distributions to measured values. The basic steps are outlined below and in Figure 3C-48.

Step 1: Run CAMx simulation with HDDM to determine hourly O_3 sensitivities to NO_X emissions changes for the grid cells containing monitoring sites in an urban study area.
Step 2: For each monitoring site, season, and hour of the day use linear regression to relate first order sensitivities of NO_X (S_{NOx}) to modeled O_3 and second order sensitivities of NO_X (S^2_{NOx}) to the first order sensitivities.
Step 3: For each measured hourly O_3 value, calculate the first and second order sensitivities based on monitoring site-, season-, and hour-specific functions calculated in Step 2.
Step 4: Adjust measured hourly 2015-2017 O_3 concentrations for incrementally increasing levels of emissions reductions using assigned sensitivities, then recalculate 2015-2017 design values until all monitors in the urban study area just meet the levels of the air quality scenario.

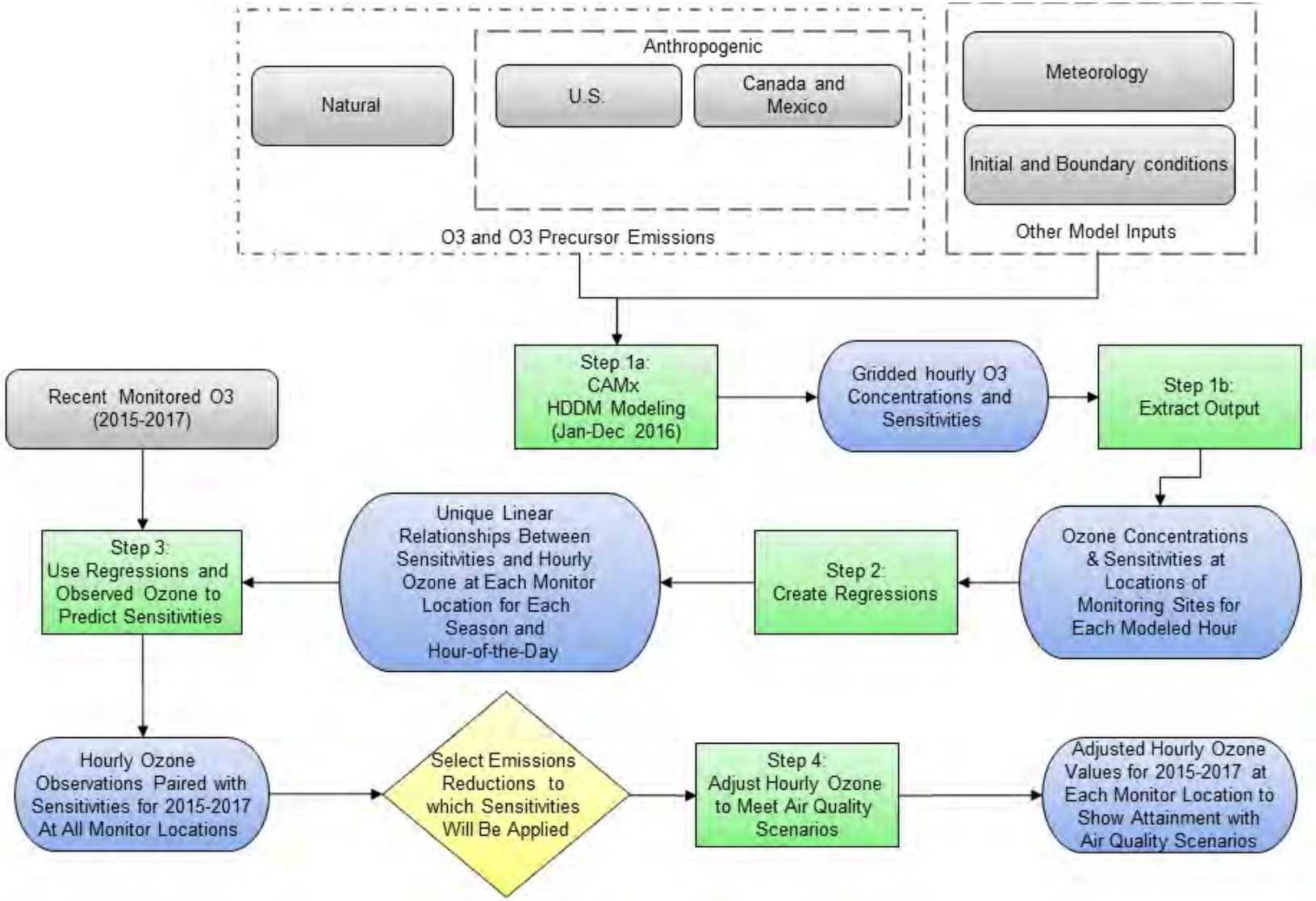


Figure 3C-48. Flow diagram demonstrating HDDM model-based O_3 adjustment approach.

3C.5.2.2 Application to Measured O_3 Concentrations in Urban Study Areas

The model-based adjustment approach described above was applied to the eight urban study areas (Atlanta, Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento, and St. Louis) for an air quality scenario adjusted to just meet the current standard of 70 ppb and two alternative air quality scenarios having design values of 75 ppb and 65 ppb. The analysis used CAMx photochemical modeling for January-December of 2016 and ambient air data for the years 2015-2017. When running CAMx with HDDM, additional information is required to designate model inputs for calculating sensitivities. In this analysis, HDDM was set up to calculate the sensitivity of O_3 concentrations to U.S. anthropogenic NO_X emissions.[13]

U.S. anthropogenic emissions were defined as all emissions in the following sectors: commercial marine, rail, residential wood combustion, agricultural fires, onroad mobile, offroad mobile, EGU point sources, oil and natural gas point, non-EGU point, non-point oil and gas, and non-point area. These anthropogenic sectors account for 10.5 million of the total CONUS-wide 11.8 million tons per year of NO_X emissions in 2016 (the remaining 1.3 million tons are from biogenics and wildland fires, which included prescribed burns). Sensitivities were not calculated for biogenic, wildland fire, Canadian, or Mexican emissions. In addition, sensitivities were not calculated for any emissions originating from outside the domain (i.e., entering through the use of boundary concentrations).

3C.5.2.2.1 Multi-step Application of HDDM Sensitivities

As discussed in section 3C.5.1.2 of this appendix, HDDM has been reported to reasonably replicate brute force emissions reductions up to a 50% change in emissions. For this analysis, it was desirable to have confidence that the HDDM sensitivities could replicate the entire range of emissions reductions. Evaluations of the HDDM estimated O_3 concentrations compared to that estimated from brute force emissions reduction model runs confirm that the HDDM estimates of O_3 response to NO_X reductions are fairly comparable for a 50% change. However, O_3 concentrations estimated from the HDDM sensitivities and the brute force method begin to diverge in comparisons under larger emissions changes (90%). Consequently, two additional CAMx/HDDM runs were performed under different levels of NO_X emissions reductions in order to characterize O_3 sensitivities to NO_X reductions over a larger range of 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.

NO_X emissions reduced by 50%. A second additional simulation was performed with a 90% NO_X reduction. Emissions of other species were not modified from the base case in these two additional simulations. These additional HDDM simulations provide O_3 sensitivities to NO_X under chemical regimes with lower NO_X emissions. The sensitivities are used in a multistep adjustment approach, as described in the following sections.

Figure 3C-49 provides a conceptual picture of the multistep adjustment procedure using first-order sensitivities. Sensitivities from the base run are used to adjust O_3 concentrations for NO_X emissions reductions up to X%. Additional emission reductions beyond X% use sensitivities from the 50% NO_X cut run until reductions exceed (X+Y)%. Finally, sensitivities from the 90% NO_X emissions reduction run are applied for any emission reductions beyond (X+Y)%. In order to more closely approximate the non-linear O_3 response to any level of emissions reductions, 2nd order terms are added to the multistep approximation method in Equations (3C-4) through (3C-7). P represents the percentage NO_X cut for which the ΔO_3 values are being calculated, S and S^2 are the first and second order O_3 sensitivities to U.S. NO_X emissions, and X and Y are described above.

$$\Delta O_3 = -a \times S_{NOx_{base}} + \frac{a^2}{2} \times S^2_{NOx_{base}} - b \times S_{NOx_{50}\%cut} + \frac{b^2}{2} \times S^2_{NOx_{50}\%cut} - c \times S_{NOx_{90}\%cut} + \frac{c^2}{2} \times S^2_{NOx_{90}\%cut}$$

Equation (3C-4)

$$a = \begin{cases} \frac{P}{100} & for\ P \leq X \\ \frac{X}{100} & for\ P > X \end{cases}$$

Equation (3C-5)

$$b = \begin{cases} 0 & for\ P \leq X \\ \frac{2\times(P-X)}{100} & for\ X < P \leq (X+Y) \\ \frac{2\times Y}{100} & for\ P > (X+Y) \end{cases}$$

Equation (3C-6)

$$c = \begin{cases} 0 & for\ P \leq (X+Y) \\ \frac{10\times(P-(X+Y))}{100} & for\ 100 \geq P > (X+Y) \end{cases}$$

Equation (3C-7)

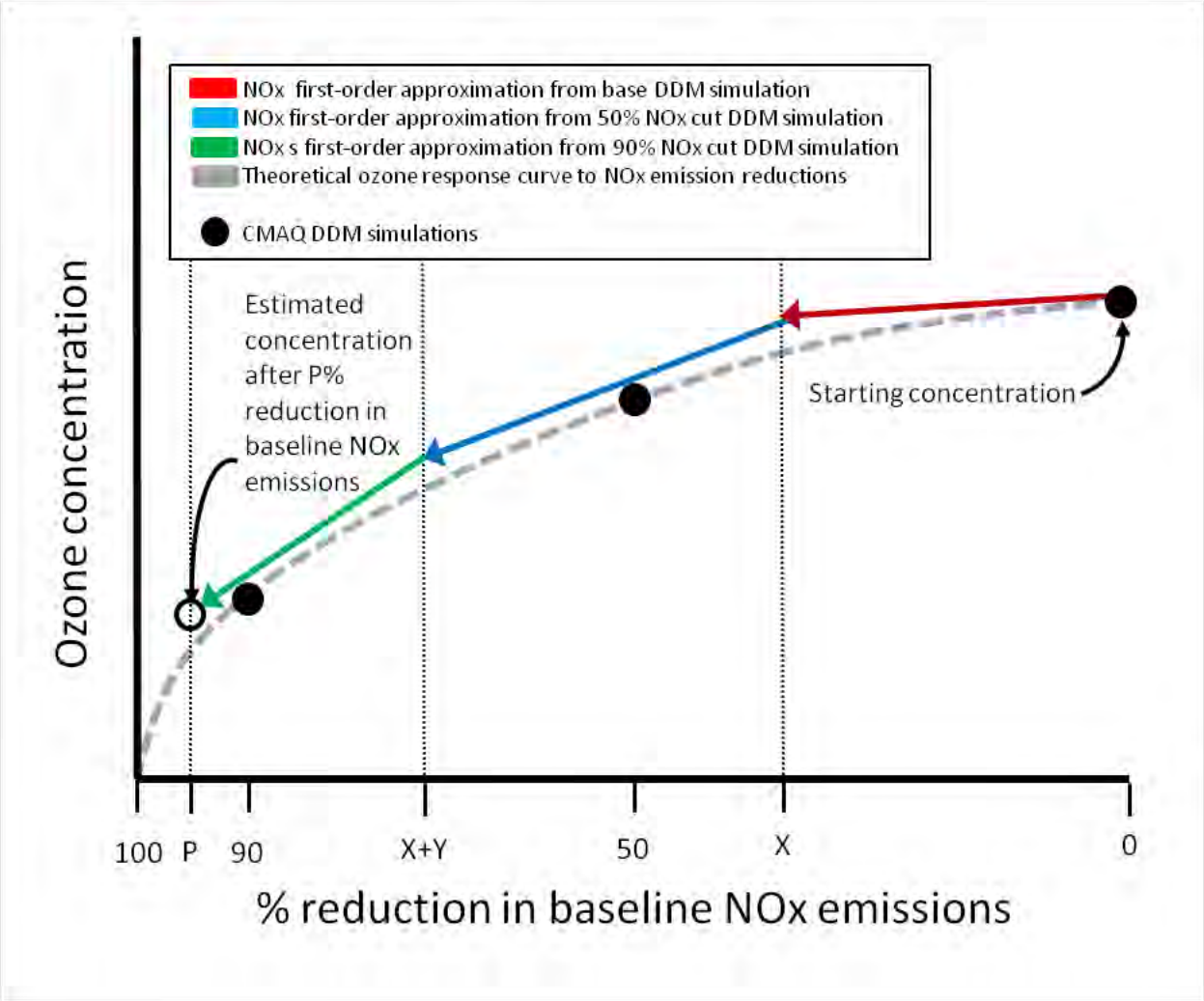


Figure 3C-49. Conceptual picture of 3-step application of HDDM sensitivities.

The ideal value for equation transition points, X and Y, are determined by minimizing the least square mean error between the adjusted concentrations using the multistep approach and modeled concentrations from brute force NO_X emissions reduction runs. We first determined the value of X which gave the lowest error compared to brute forces estimates at 50% NO_X emissions reductions. Then holding X constant, we determined the value of Y which gave the lowest error compared to brute force method O_3 concentration estimates using 90% NO_X emissions reductions. This process was performed independently for each of the eight urban study areas in this analysis.

Error in HDDM estimates of hourly O_3 is defined here as the difference between HDDM estimated O_3 and O_3 estimated using the brute force method. Based on equations (3C-4) through (3C-7), this can be calculated from Equations (3C-8) and (3C-9) for 50% NO_X emissions reductions:

$$\varepsilon = \Delta Ozone_{HDDM,50} - \Delta Ozone_{BF,50}$$

Equation (3C-8)

$$\varepsilon = \frac{-X}{100} \times S_{NOx\_base} + \frac{X^2}{2 \times 100^2} \times S^2_{NOx\_base} - \frac{2(50-X)}{100} \times S_{NOx_{50}\%cut} + \frac{\left(2 \times (50-X)\right)^2}{2 \times 100^2} \times S^2_{NOx_{50}\%cut} - \Delta Ozone_{BF,50}$$

Equation (3C-9)

Equation (3C-10) can be rearranged to appear in the form: $AX^2 + BX + C$:

$$\varepsilon = \left(\frac{S^2_{NOx_{base}}}{2 \times 100^2} + \frac{4 \times S^2_{NOx_{50}\%cut}}{2 \times 100^2}\right)X^2 + \left(\frac{-S_{NOx_{base}}}{100} + \frac{2 \times S_{NOx_{50}\%cut}}{100} - \frac{400 \times S^2_{NOx_{50}\%cut}}{2 \times 100^2}\right)X + \left(-S_{NOx_{50}\%cut} + \frac{S^2_{NOx_{50}\%cut}}{2} - \Delta Ozone_{BF,50}\right)$$

Equation (3C-10)

$$A = \left(\frac{S^2_{NOx\_base}}{2 \times 100^2} + \frac{4 \times S^2_{NOx_{50}\%cut}}{2 \times 100^2}\right)$$

Equation (3C-11)

$$B = \left(\frac{-S_{NOx\_base}}{100} + \frac{2 \times S_{NOx_{50}\%cut}}{100} - \frac{400 \times S^2_{NOx_{50}\%cut}}{2 \times 100^2}\right)$$

Equation (3C-12)

$$C = \left(-S_{NOx_{50}\%cut} + \frac{S^2_{NOx_{50}\%cut}}{2} - \Delta Ozone_{BF,50}\right)$$

Equation (3C-13)

Next, the error is squared, summed over all points (error can be calculated for each hourly O_3 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)).

$$\varepsilon^2 = A^2X^4 + 2ABX^3 + (2AC + B^2)X^2 + 2BCX + C^2$$

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).

$$\varepsilon = \frac{-X}{100} \times S_{NOx_{base}} + \frac{X^2}{2 \times 100^2} \times S^2_{NOx_{base}} - \frac{2Y}{100} \times S_{NOx_{50}\%cut} + \frac{2^2Y^2}{2 \times 100^2} \times S^2_{NOx_{50}\%cut} - \frac{10\left(90 - (X+Y)\right)}{100} \times S_{NOx_{90}\%cut} + \frac{\left(10^2(90-(X+Y))\right)^2}{2\times 100^2} \times S^2_{NOx_{90}\%cut} - \Delta Ozone_{BF,90}$$

Equation (3C-17)

$$\varepsilon^2 = A^2Y^4 + 2ABY^3 + (2AC + B^2)Y^2 + 2BCY + C^2$$

Equation (3C-18)

$$A = \left(\frac{4\times S^2_{NOx_{50}\%cut}}{2\times 100^2} + \frac{100\times S^2_{NO_{90}\%cut}}{2\times 100^2}\right)$$

Equation (3C-19)

$$B = \left(\frac{-2\times S_{NOx_{50}\%cut}}{100} + \frac{10\times S_{NO_{90}\%cut}}{100} - \frac{200\times(90-X)S^2_{NOx_{90}\%cut}}{2\times 100^2}\right)$$

Equation (3C-20)

$$C = \left(\frac{-X}{100} S_{NOx\_base} + \frac{X^2}{2 \times 100^2} S^2_{NOx\_base} - \frac{10 \times (90 - X)}{100} S_{NOx_{90}\%cut} + \frac{100 \times (90 - X)^2}{2 \times 100^2} S^2_{NOx_{90}\%cut} - \Delta Ozone_{BF,90}\right)$$

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)

$$(\sum \varepsilon^2)' = (4\sum A^2)Y^3 + (3\sum 2AB)Y^2 + (2\sum 2AC + B^2)Y + (\sum 2BC) = 0$$

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 O_3 estimates from brute force with hourly O_3 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 O_3 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

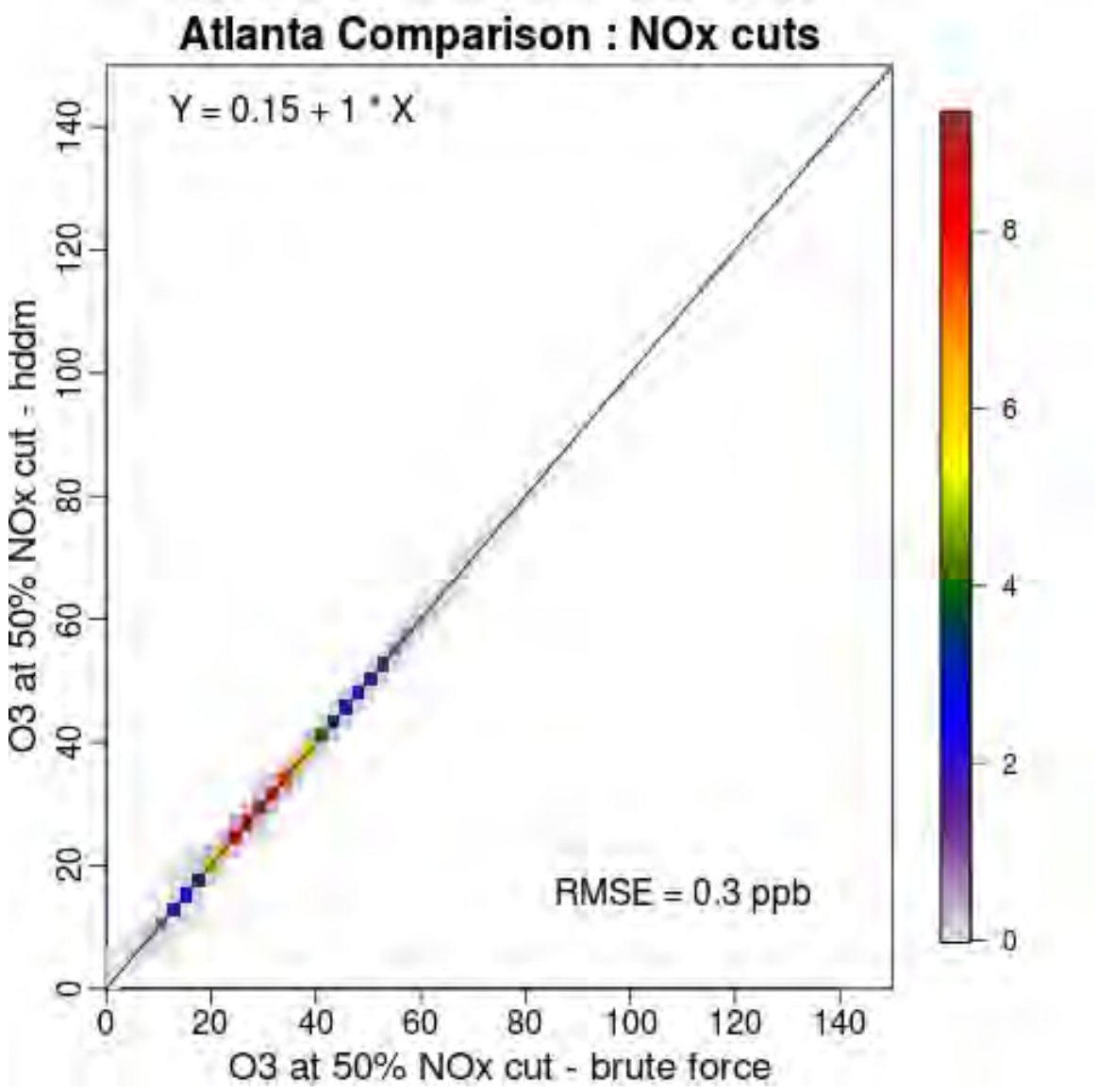


Figure 3C-50. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in Atlanta.

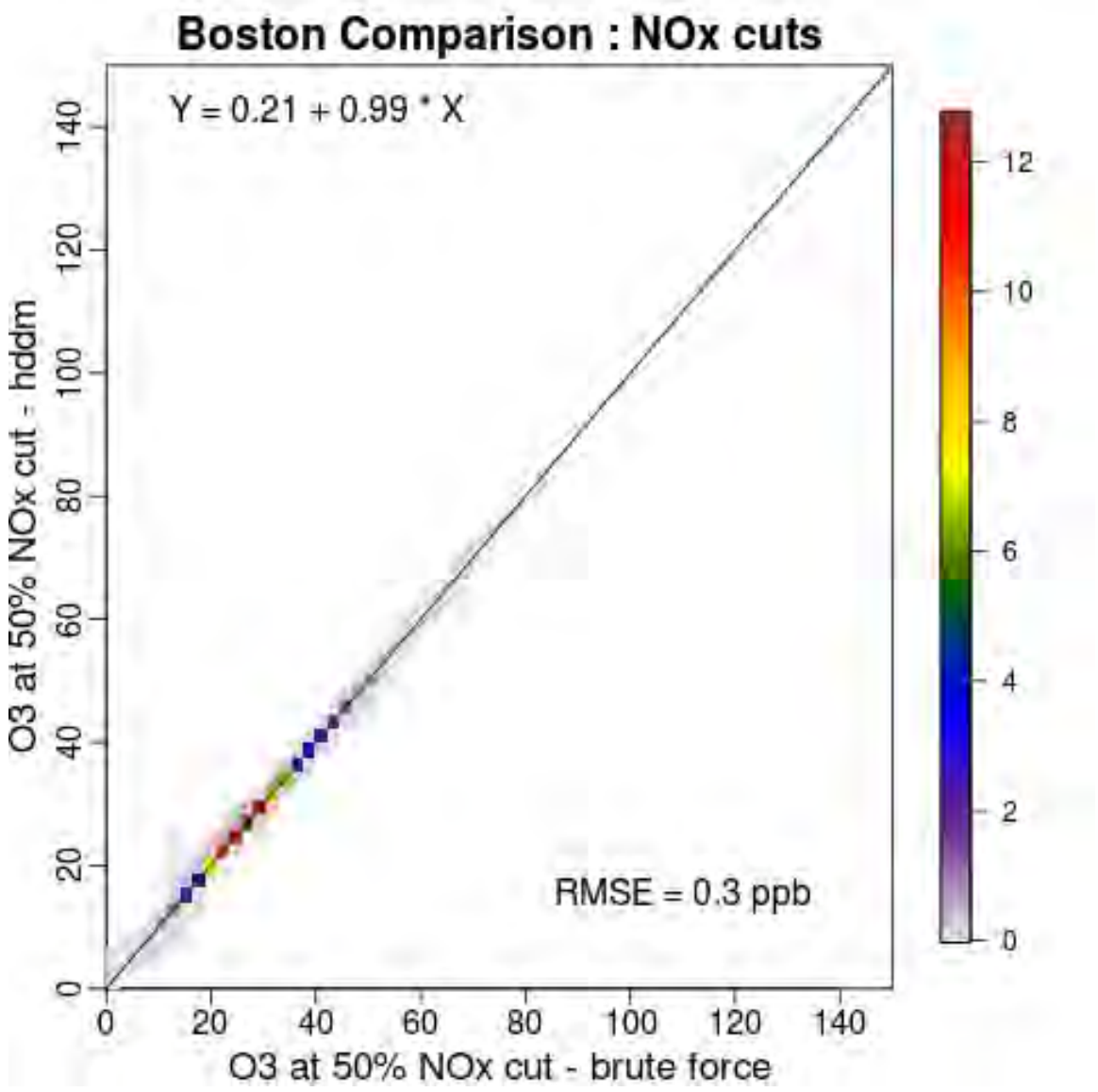


Figure 3C-51. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in Boston.

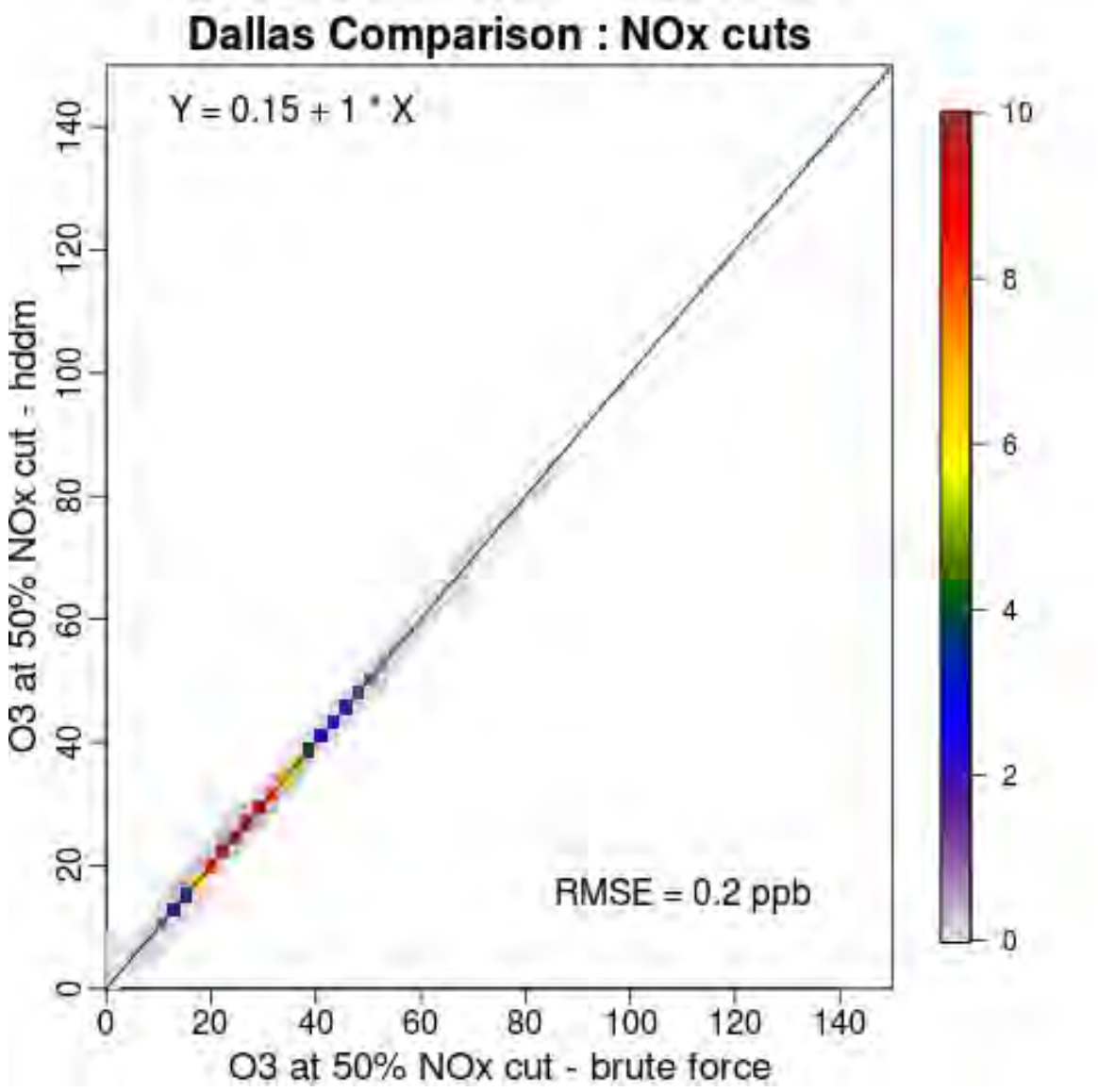


Figure 3C-52. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in Dallas.

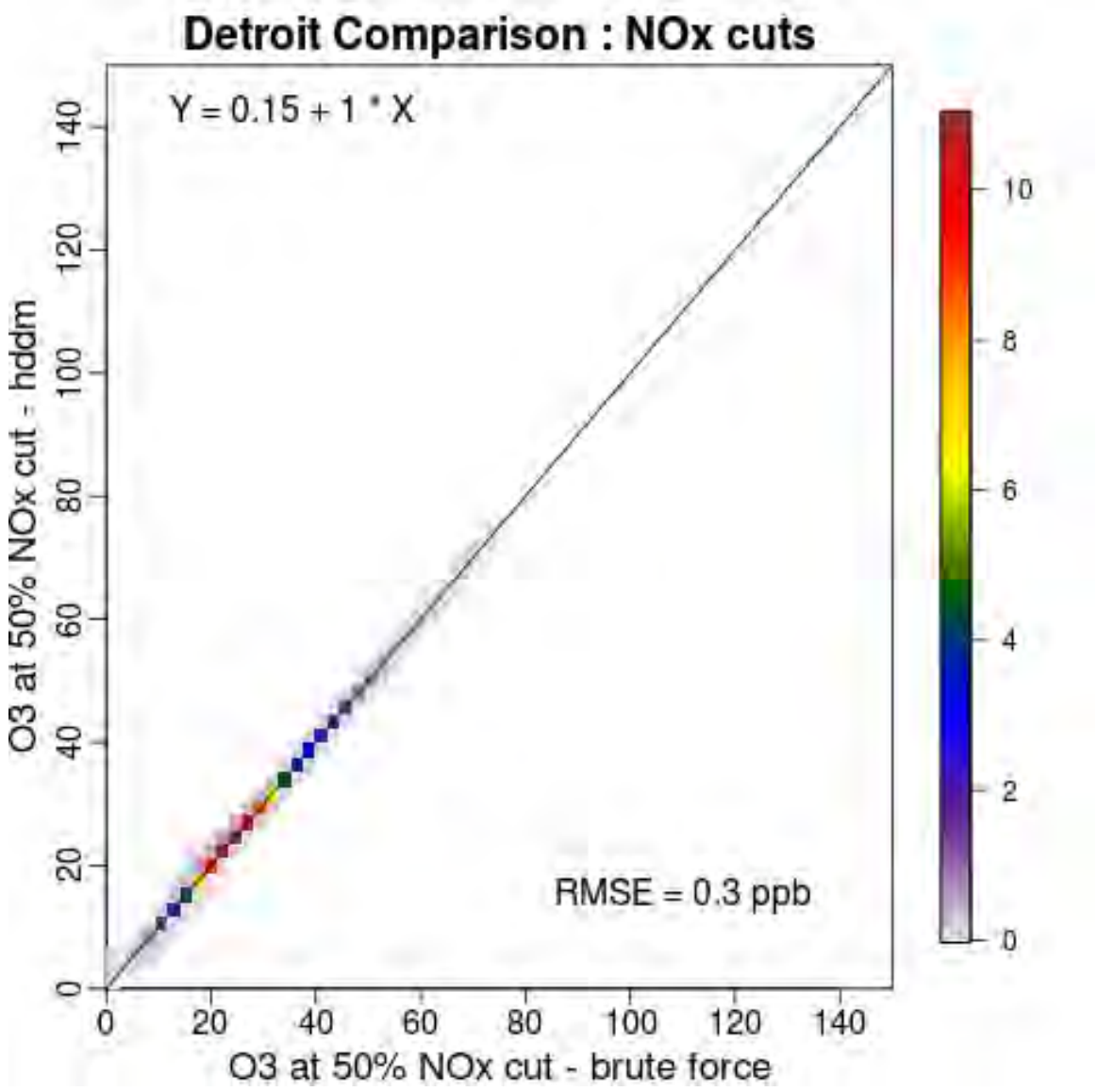


Figure 3C-53. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in Detroit.

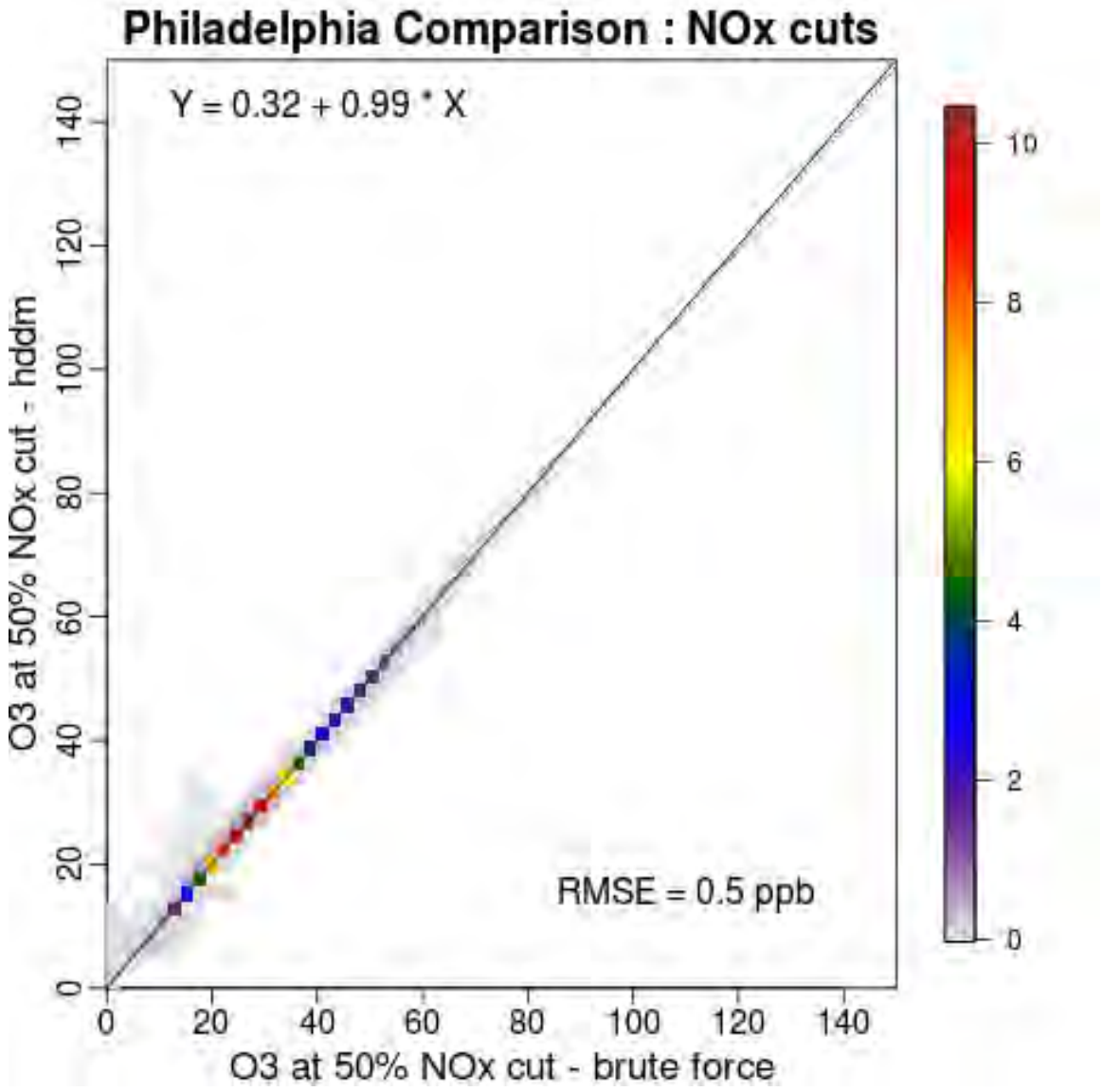


Figure 3C-54. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in Philadelphia.

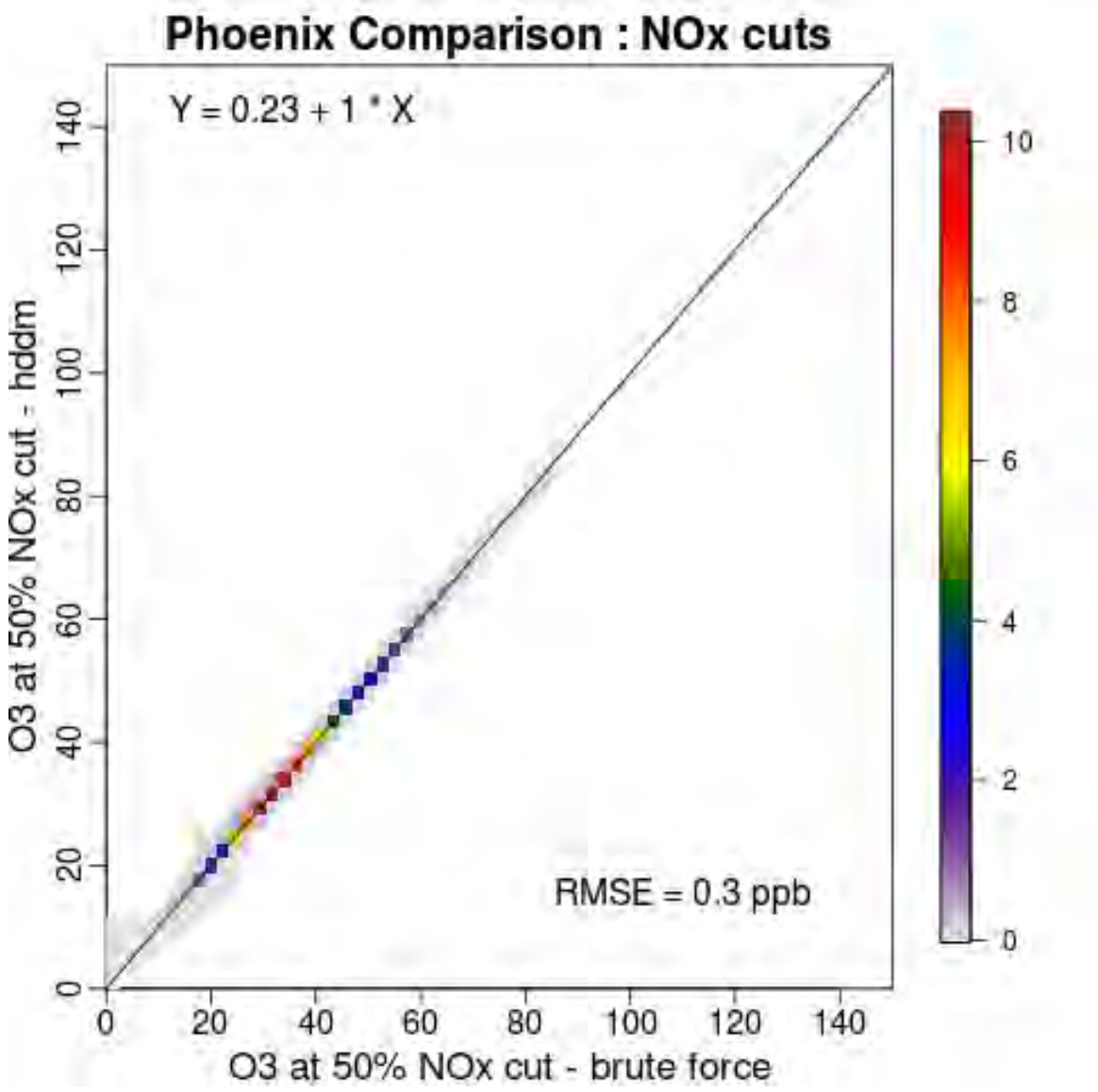


Figure 3C-55. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in Phoenix.

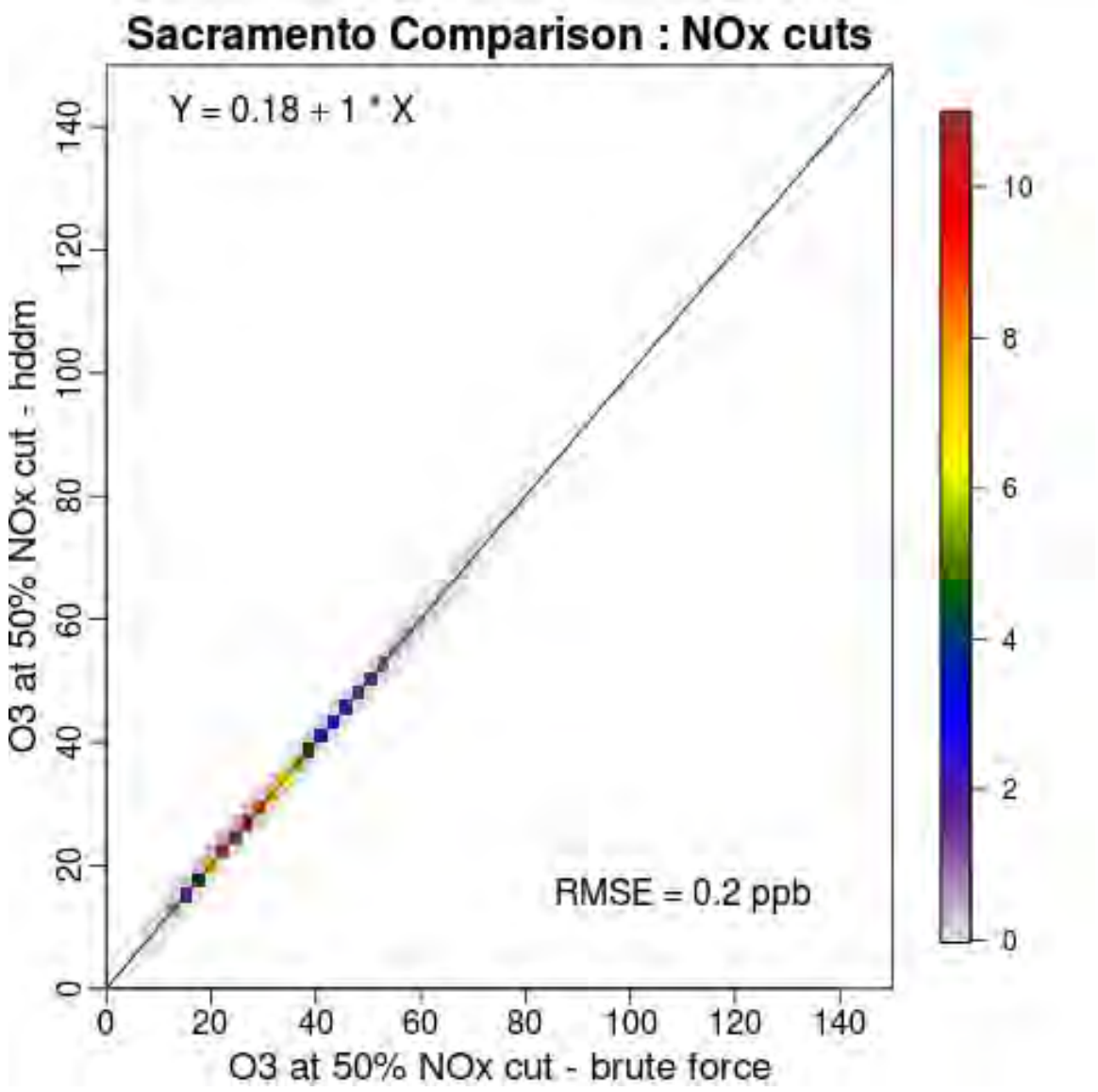


Figure 3C-56. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in Sacramento.

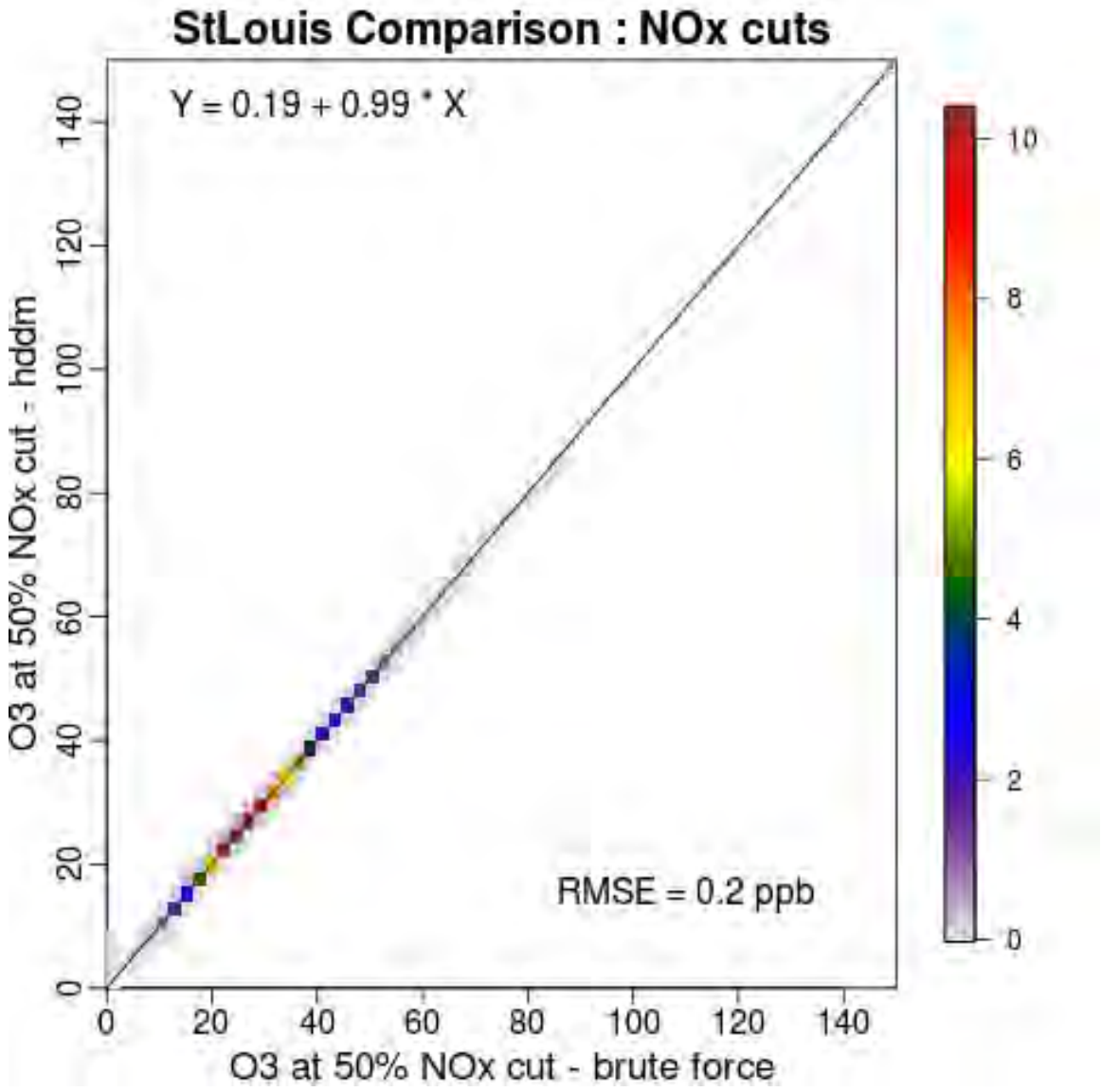


Figure 3C-57. Comparison of brute force and 3-step HDDM O_3 estimates for 50% NO_X cut conditions in St. Louis.

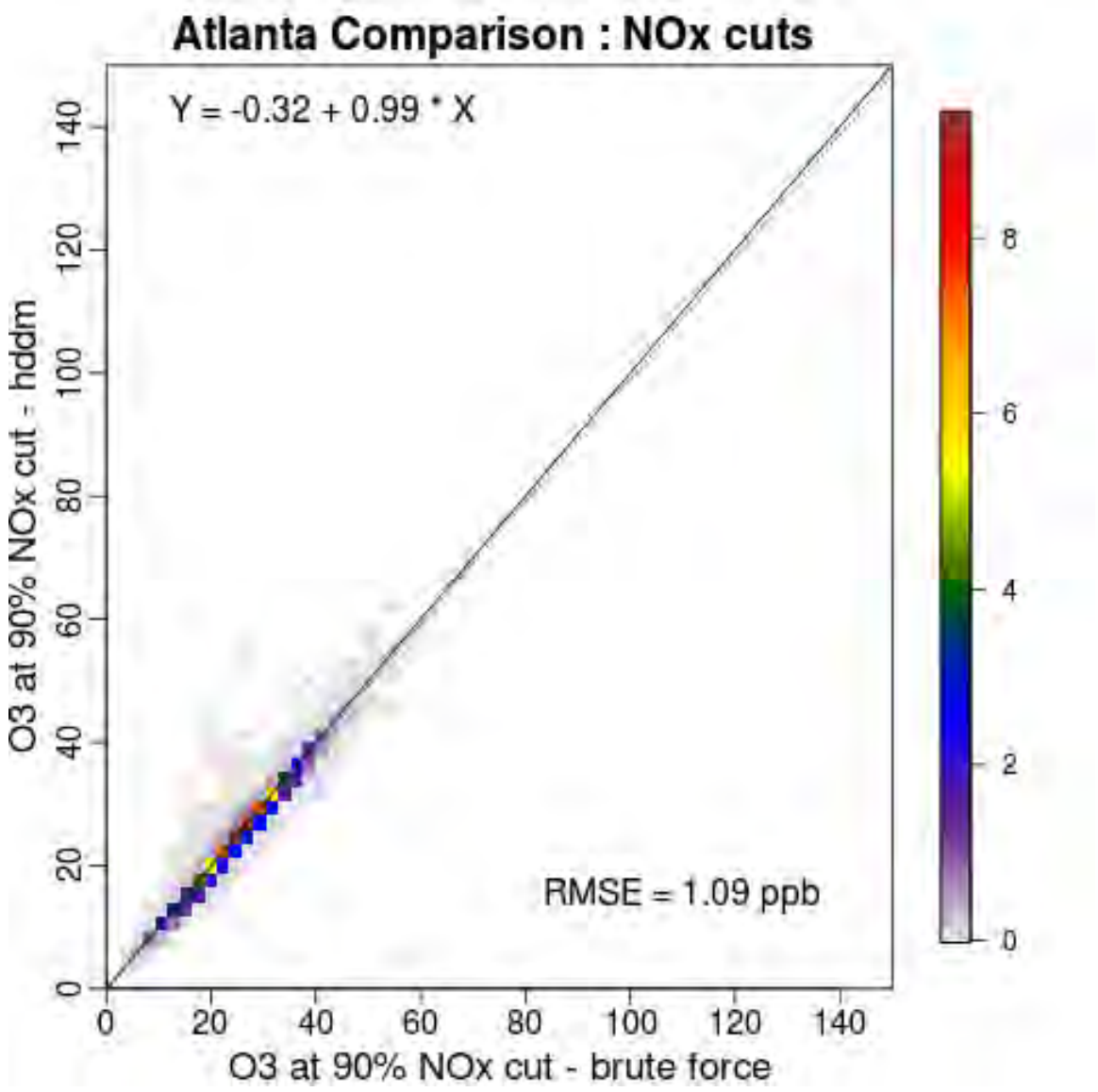


Figure 3C-58. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in Atlanta.

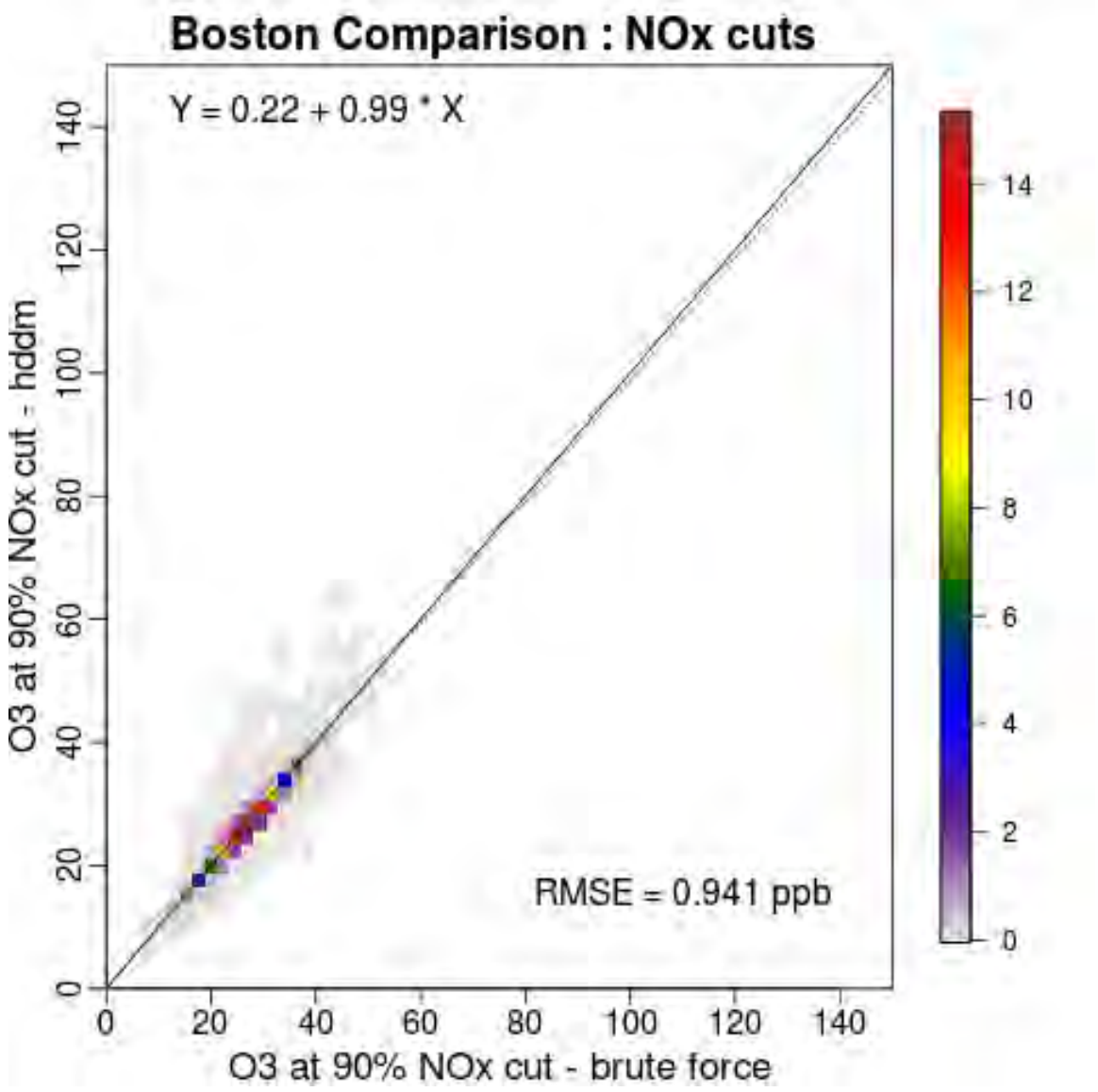


Figure 3C-59. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in Boston.

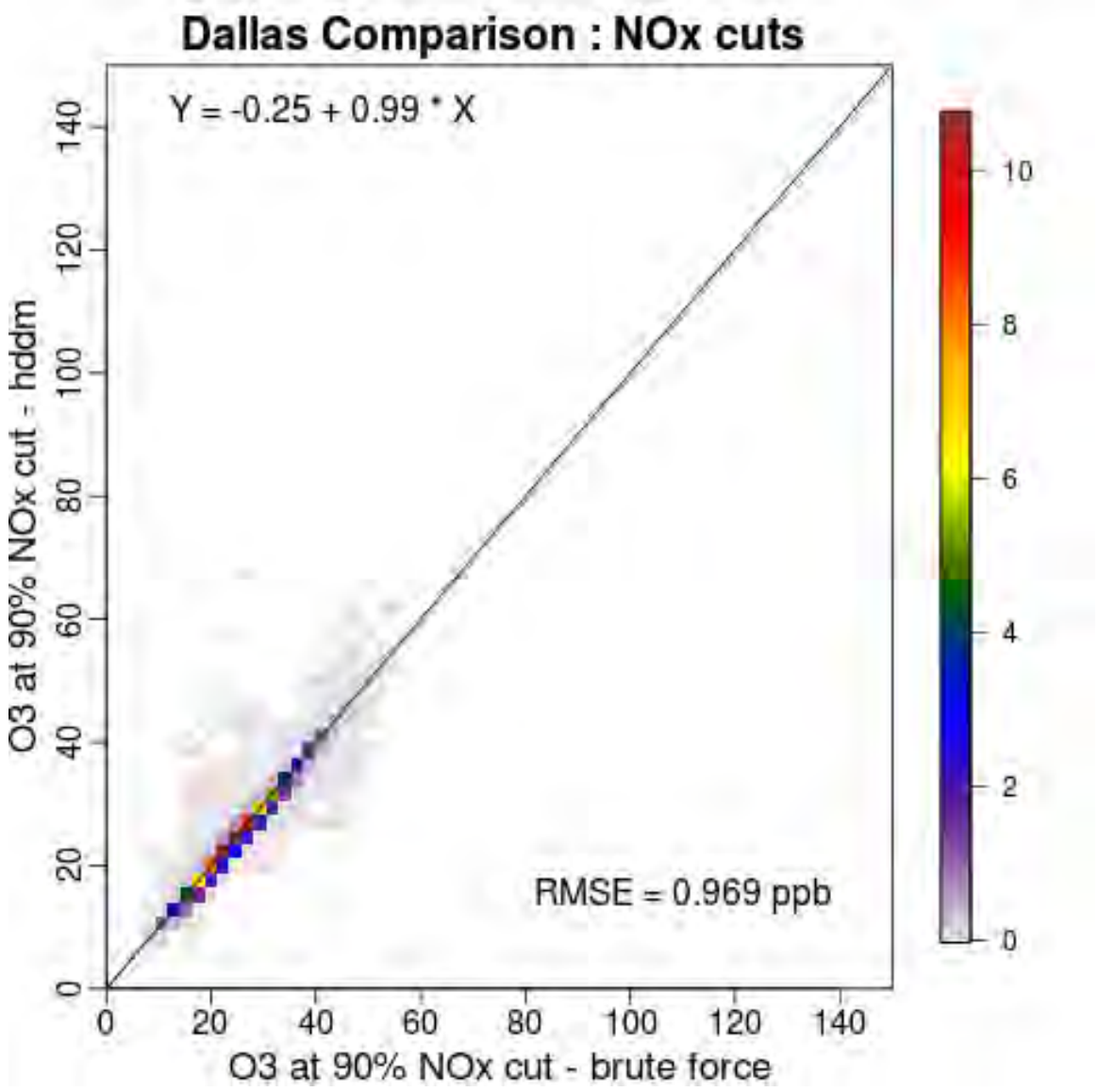


Figure 3C-60. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in Dallas.

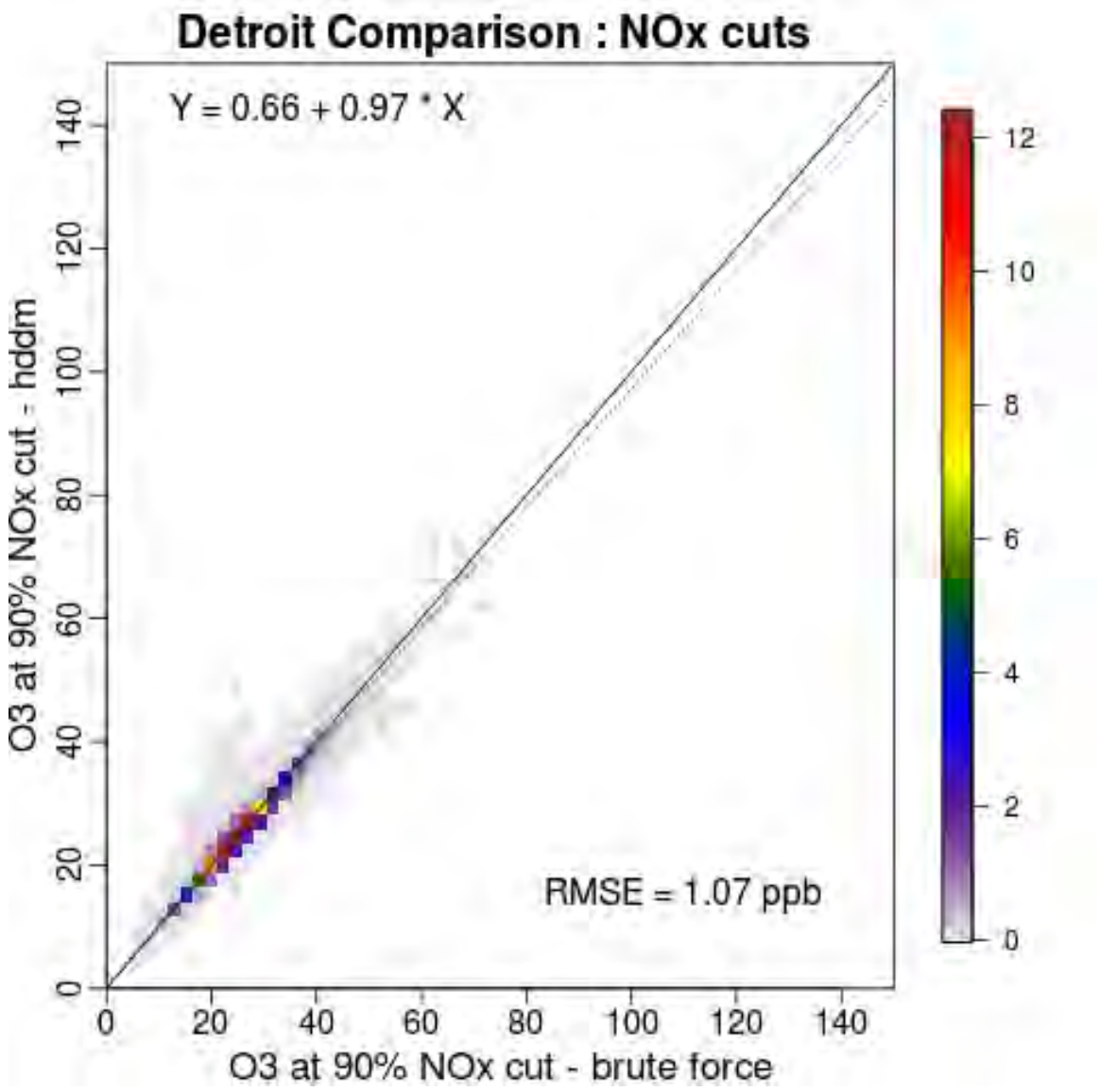


Figure 3C-61. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in Detroit.

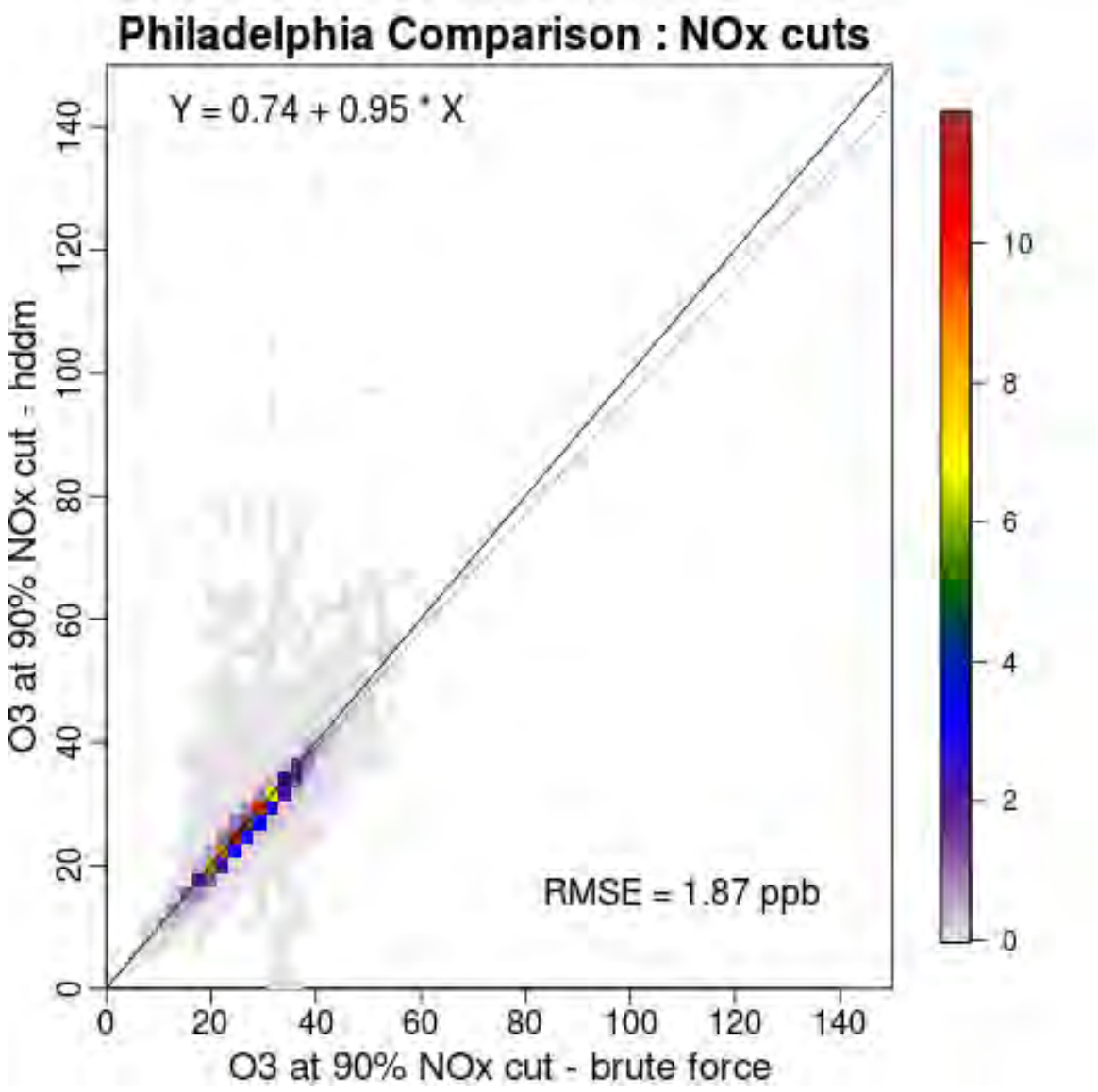


Figure 3C-62. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in Philadelphia.

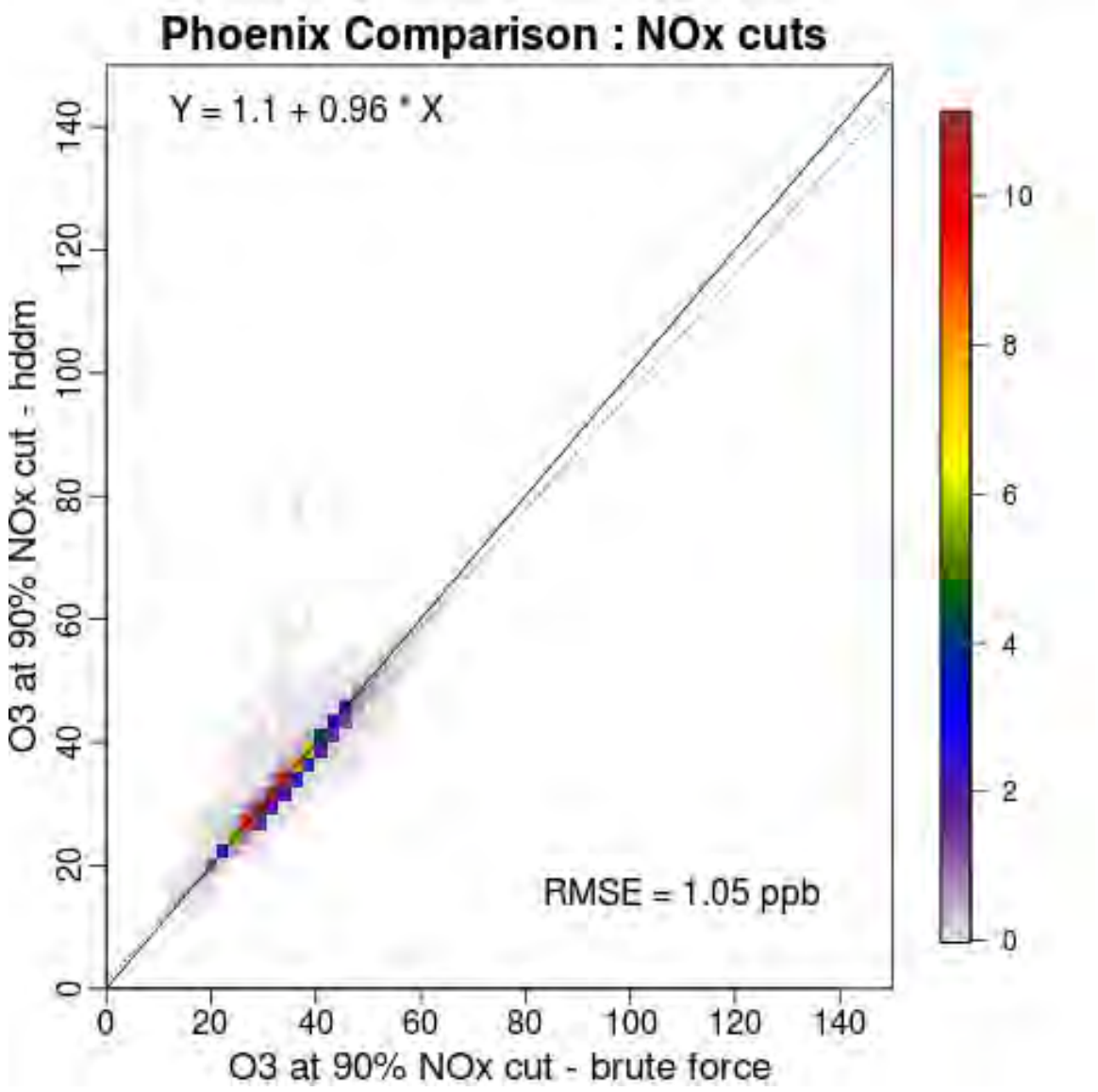


Figure 3C-63. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in Phoenix.

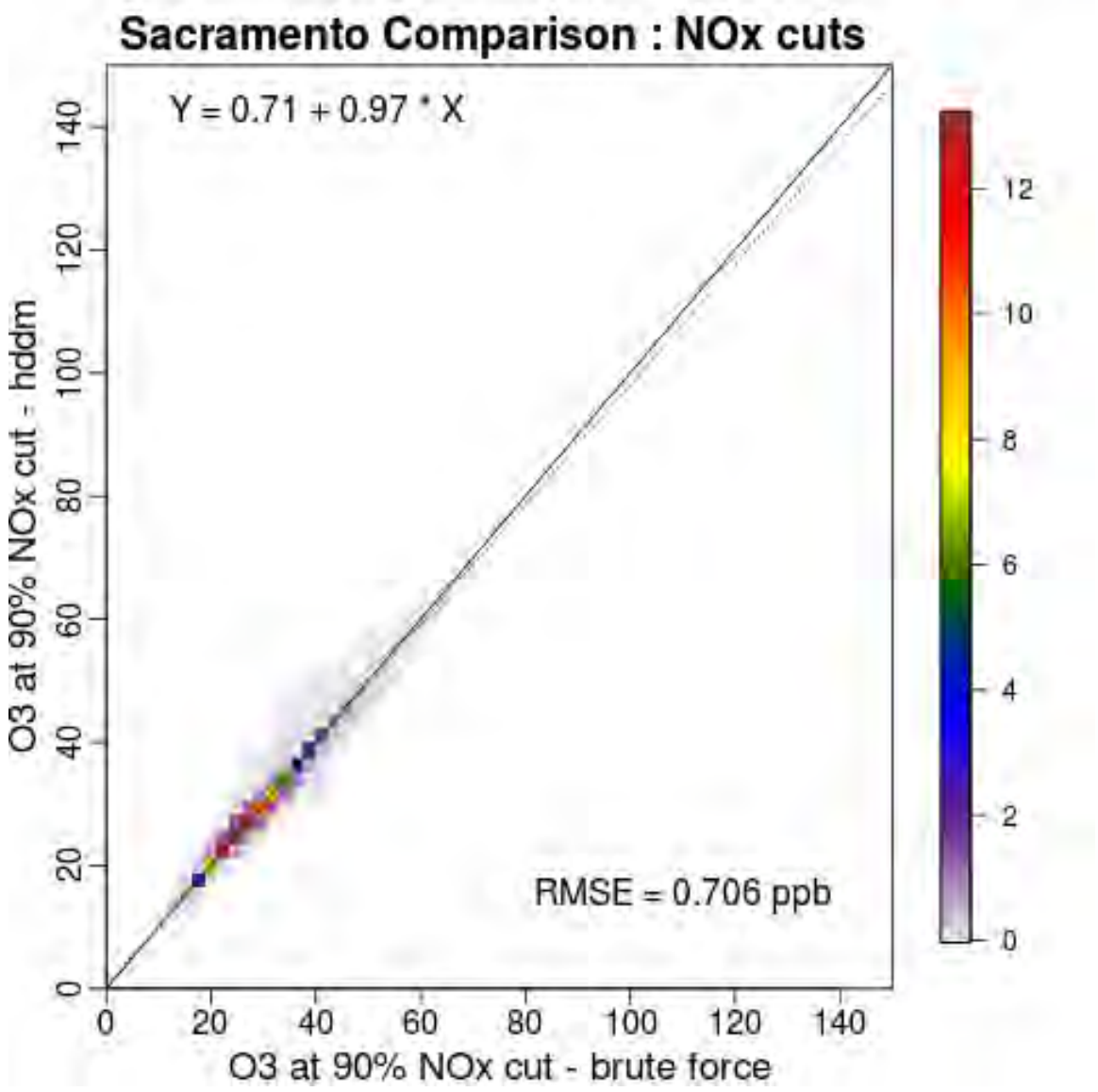


Figure 3C-64. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in Sacramento.

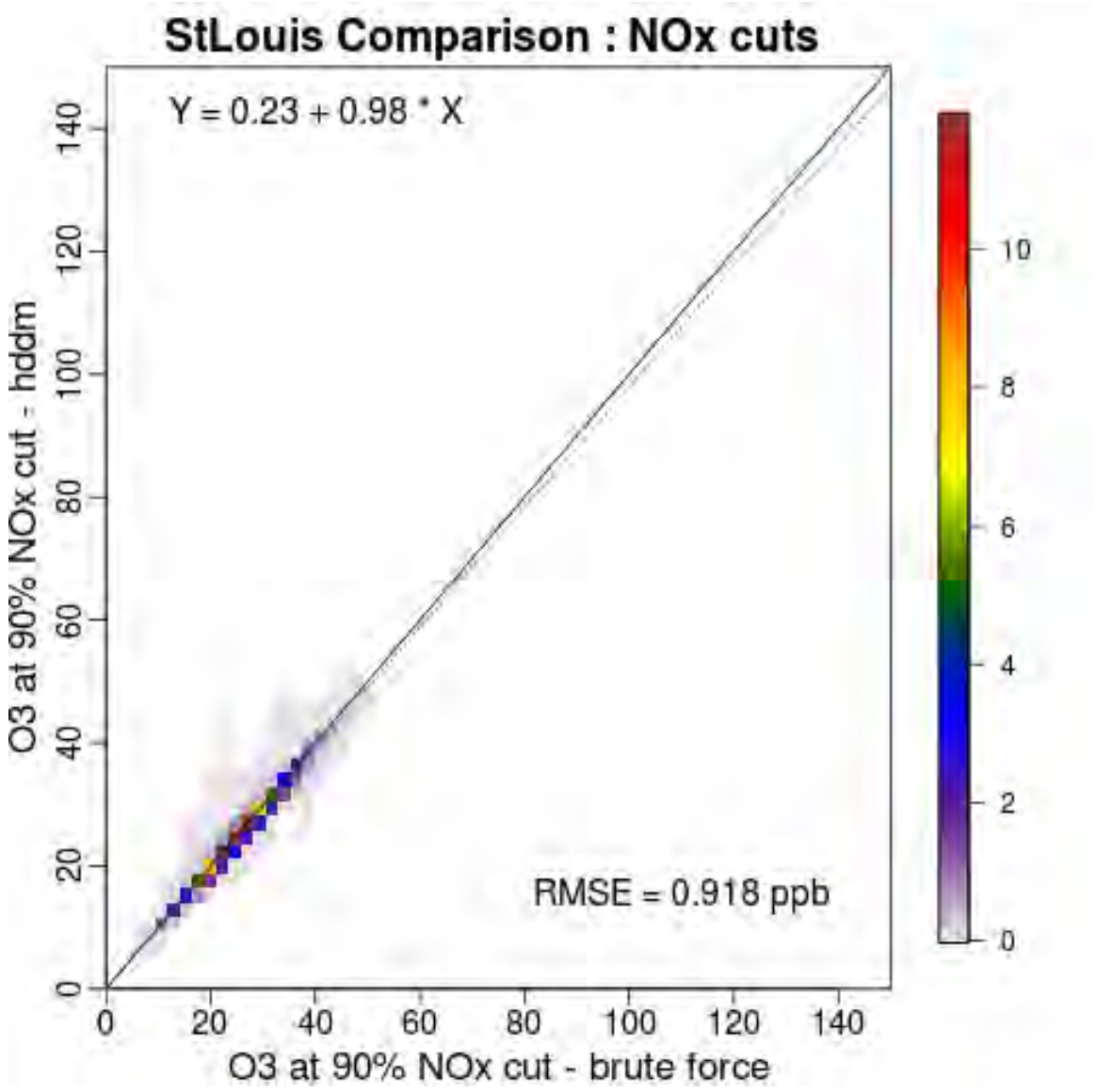


Figure 3C-65. Comparison of brute force and 3-step HDDM O_3 estimates for 90% NO_X cut conditions in St. Louis.

3C.5.2.2.2 Relationships between HDDM Sensitivities and Modeled O_3 Concentrations

First and second order hourly O_3 sensitivities to NO_X emissions reductions were extracted from the HDDM simulation for model grid cells that contained the O_3 monitors in the eight urban study areas. Extracted data included modeled sensitivities at monitor locations for all modeled hours in 2016. These sensitivities cannot be applied directly to observed values for two reasons: 1) high modeled O_3 days/hours do not always occur concurrently with high observed O_3 days/hours and 2) the modeling time period includes only 2016 but the time period we are

analyzing in this assessment includes three full years of ambient air data, 2015-2017. As to the first point, photochemical models are generally used in a relative sense for purposes of projecting design values. In this manner, model predictions are "anchored" to ambient air measurements. In general, the average response on high modeled days is used for this purpose. This allows for more confidence in calculated results when "less than ideal model performance [occurs] on individual days" (U.S. EPA, 2007). Similarly, for this analysis we believe it is appropriate to account for the fact the model does not always perfectly agree with measurements and that sensitivities from a low O_3 modeled day would not be appropriate to apply to a high O_3 measured day (and vice-versa) even if they occur on the same calendar day. For this reason, a method was developed to generalize the modeled site-, season-, and hour-specific sensitivities so that they could be applied to ambient air data during 2015-2017.[14]

Simon et al. (2013) describe how first order sensitivities are generally well correlated with hourly modeled O_3 concentrations and second order sensitivities are well correlated to first order sensitivities. Based on their analysis, we create a separate linear regression for S_{NOx} as a function of hourly O_3 (i.e. $S_{NOx} = m \times O_3 + b$) for every site, season[15], and hour-of-the day examined in this analysis. For instance, for summer 8-am hours at Detroit monitor site ID 260990009, S_{NOx} and O_3 values from all 8-am hours in June-August 2016 are used to fit this relationship. Similarly, S^2_{NOx} was calculated as a function of S_{NOx}.

Comparisons between brute force and HDDM O_3 estimates shown in Figure 3C-50 through Figure 3C-65 demonstrate that for the vast majority of data points, HDDM replicates brute force with minimal errors. These figures show a small number of instances, particularly for Philadelphia, in which HDDM predicts very high hourly O_3 (> 100 ppb) while the brute force emissions simulations for the 90% reduction show much lower O_3 (< 40 ppb). In these isolated cases, base modeled O_3 is low due to NO_X titration and increases occur with reductions of NO_X. The HDDM sensitivities for these few points appear to be too high to be applied over large (>50%) emissions changes because of strongly nonlinear chemistry. However these extreme cases are not relevant for this analysis, since the largest emissions reduction required for Philadelphia was 53% to meet the air quality scenario for 65 ppb (Table 3C-19). The two urban study areas requiring emission cuts larger than ~50%, Phoenix and Sacramento, both show much better agreement between the 90% brute force and HDDM predictions (Figure 3C-63 and Figure 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.

For the 50% and 90% emissions reduction CAMx/HDDM simulation, regressions were performed for first order NO_X sensitivities with modeled O_3 from the base HDDM simulation. The regression technique was performed for the first and second order NO_X sensitivities from the base run and the 50% emissions reduction and 90% emissions reduction simulations. The sensitivities from the emissions reduction runs were fitted to hourly O_3 concentrations in the base simulation. Simon et al. (2013) found that correlation coefficients using for sensitivities from NO_X reduction simulations to base case O_3 concentrations were similar to those with O_3 concentrations from the NO_X reduction runs.

3C.5.2.2.3 Application of Sensitivity Regressions to Ambient Air Data

To apply the HDDM adjustments to observed data, sensitivities must be determined for each hour from 2015-2017 at each site based on the linear relationship from the modeled data and the observed O_3 concentration. The linear regression model also allows us to quantify the standard error of each predicted sensitivity value at each hour and site.

Observed hourly O_3 from 2015-2017 at each monitor location was adjusted by applying incrementally increasing emissions reductions using equations (3C-4) through (3C-8) and recalculating MDA8 values for incrementally increasing emissions reductions until an emissions level is reach for which all monitors in an urban study area achieved design values at the level of the air quality scenario being evaluated (design values of 75, 70, or 65 ppb). Therefore, all monitors within an urban study area were treated as responding to the same percentage reduction in NO_X emissions.

The precursor reductions used to estimate spatial and temporal patterns of O_3 concentrations for the three air quality scenarios were NO_X-only reductions. We focused on NO_X-only reductions in light of several key findings from analyses for the 2014 HREA that explored the use of both NO_X and VOC reductions versus NO_X-only scenarios (2014 HREA, Appendix 4D). There were several key findings from that comparison. First, in most of the urban study areas, the NO_X /VOC scenario did not affect O_3 response at the monitor having the highest design value in such a way to reduce the total required emissions cuts. Further, evidence in the literature has shown that locations in the U.S. have gotten more NO_X-limited since 2007 (the year modeled in the 2014 HREA) (Jin et al., 2017, Laughner and Cohen, 2019) and thus VOC reductions would be expected to have less impact on resulting O_3 concentrations in our scenarios for the 2016 modeling used here than they had in the previous analysis. Finally, the two areas (Denver and Chicago) in which VOC emissions had the most impact in the 2014 HREA were not included in the current analysis. For these reasons, NO_X-only reductions were determined to be

the most appropriate scenarios for this analysis. The final emissions reductions that were applied in each urban study area are given in Table 3C-19 below.[16]

Table 3C-19. Percent emissions changes used for each urban study area to just meet each 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%

The 2014 HREA included a thorough analysis of the standard error associated with the predicted O_3 concentrations produced using the HDDM adjustment approach. This analysis found that while the error in predicted values varied by site and air quality scenario being evaluated, the magnitudes were small (<1.5 ppb in most cases). We did not repeat such an analysis here given the small magnitude of the standard errors found in this previous assessment.

3C.6 INTERPOLATION OF ADJUSTED AIR QUALITY USING VORONOI NEIGHBOR AVERAGING

The APEX exposure model uses spatial fields of ambient air quality concentrations at variable spatial scales (e.g., 500 m regular grid, census tract centroid) as inputs, but requires that there be no missing values. The final air quality data used as inputs to the APEX model were the hourly O_3 concentrations at monitoring sites adjusted using CAMx/HDDM, then interpolated to each census tract centroid in the eight urban study areas using the Voronoi Neighbor Averaging (VNA; Gold et al., 1997; Chen et al., 2004) technique described below. A cross-validation analysis supporting the use of the VNA technique for the creation of hourly O_3 spatial fields was conducted in the previous 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.

The following paragraphs provide a numerical example of VNA used to estimate an O_3 concentration value for census tract "E" in Figure 3C-66 below.

The first step in the VNA technique is to identify the set of nearest monitors for each census tract. The left-hand panel of Figure 3C-66 presents a numerical example with nine census tracts (squares) and seven monitoring sites (stars), with the focus on identifying the set of nearest neighboring sites to census tract "E" in the center of the panel. The Delaunay triangulation algorithm identifies the set of nearest neighboring monitors by drawing a set of polygons called the "Voronoi diagram" around the census tract "E" centroid and each of the monitoring sites. Voronoi diagrams have the special property that each edge of each of the polygons are the same distance from the two closest points, as shown in the right-hand panel of Figure 3C-66.

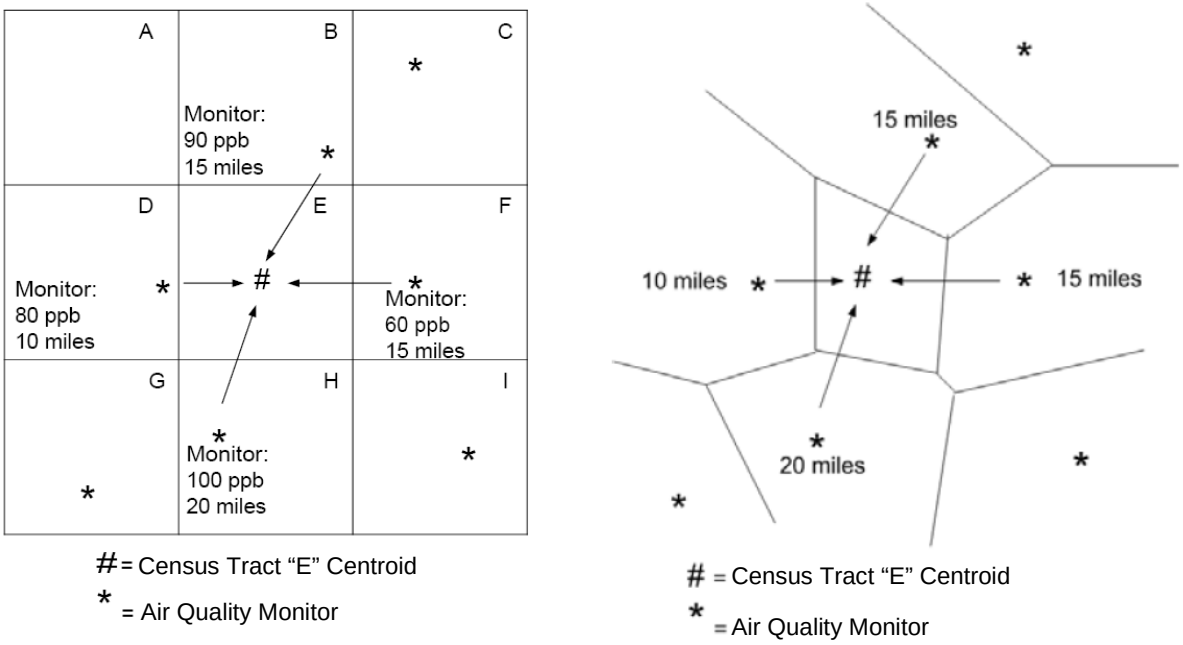


Figure 3C-66. Numerical example of the Voronoi Neighbor Averaging (VNA) technique.

The VNA technique then chooses the monitoring sites whose polygons share a boundary with the census tract "E" centroid. These monitors are the "Voronoi neighbors", which are used to estimate the concentration value for census tract "E". The VNA estimate of the concentration value in census tract "E" is the inverse distance squared weighted average of the four monitored concentrations. The further the monitor is from the center of census tract "E", the smaller the weight. For example, the weight for the monitor in census tract "D" 10 miles from the census tract "E" centroid is calculated as follows:

$$\frac{1/10^2}{1/10^2 + 1/15^2 + 1/15^2 + 1/20^2} = 0.4675$$

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:

$$VNA(E) = 0.4675 * 80 + 0.2078 * 90 + 0.2078 * 60 + 0.1169 * 100 = 80.3\ ppb$$

Equation (3C-25)

The adjusted hourly O_3 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 O_3 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 O_3 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

75 ppb, 70 ppb and 65 ppb, the location of the highest monitor shifted slightly west to monitor 421010024 (east of downtown Philadelphia).

The pattern for Phoenix was unique among the eight urban study areas. One monitor (040139997) was consistently high in the observations and for all adjusted levels. However, two other monitors were equally as high in the observations (040132005; 040131003 – also high at 75 ppb) but responded more strongly to the applied NO_X reductions. While monitors 040132005 and 040131003 are slightly removed from downtown Phoenix (near Pinnacle Peak to the northeast and Mesa to the southeast, respectively), monitor 040139997 is closer the center of the Phoenix metropolitan area. This location is likely near higher concentrations of urban NO_X sources, making this monitor slightly less responsive to the NO_X emissions adjustments.

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.

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.

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.

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.

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.

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.

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.

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.

3C.7.2 Distribution of Hourly O_3 Concentrations

Figure 3C-67 through Figure 3C-74 display diurnal boxplots of hourly O_3 concentrations for 2015-2017 at monitor locations in each urban study area. For each hour of the day, the rectangular box represents the 25th and 75th percentiles of the distribution, with a solid line representing the median of the distribution through the center. Each box has "whiskers" which extend up to 1.5 times the interquartile range (i.e., the 75th percentile minus the 25th percentile) from the box, and dots which represent outlier values. Black boxplots represent observed hourly O_3 concentrations, while blue boxplots represent hourly O_3 concentrations adjusted to meet the current standard of 70 ppb. Red boxplots represent hourly O_3 concentrations adjusted for the 75 ppb[17] scenario, and green boxplots represent hourly O_3 concentrations adjusted for the 65 ppb scenario.

The boxplots include the observed O_3 concentrations as well as the concentrations adjusted to just meet the current standard and the two alternative air quality scenarios. Note that these plots include data from all sites in the study area, and thus the plots provide the overall distribution of O_3 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 O_3 concentrations for that scenario.

show similar patterns in most of the urban study areas. O_3 concentrations during daytime hours decrease from observed values (black) to values adjusted to meet the current standard of 70 ppb (blue) and decrease further under the alternative scenario of 65 ppb (green). These daytime decreases are mainly seen on high O_3 days represented by outlier dots extending above the box and whiskers. Some study areas had observed 2015-2017 design values already meeting the alternative scenario of 75 ppb, therefore some plots show increases in O_3 concentrations while other study areas show decreases in O_3 concentrations for the 75 ppb scenario.

In some urban study areas O_3 concentrations on the mid-range days, represented by the 25^{th} – 75^{th} percentile boxes, remained fairly constant (e.g. Boston) while in other urban study areas O_3 on mid-range days decreased (e.g. Atlanta). Although daytime O_3 decreased, concentrations during morning rush-hour period generally increase. These increases are associated with VOC-limited and NO_X titration conditions near NO_X sources during rush-hour periods. Reducing NO_X under these conditions results in less O_3 titration and thus increases O_3 concentrations. Nighttime increases in O_3 as a results of NO_X reductions are often seen to a lesser extent than morning rush-hour period increases. Collectively these features generally lead to a flattening of the diurnal O_3 pattern with smaller differences between daytime and nighttime concentrations as NO_X emissions are reduced. Urban study areas that required more substantial NO_X reductions for the 65 ppb scenario generally had more pronounced patterns of decreases in daytime O_3 and increases in nighttime O_3 leading to a flatter diurnal O_3 pattern (e.g., Sacramento in Figure 3C-73).

Figure 3C-75 through Figure 3C-82 display the same information as Figure 3C-67 through Figure 3C-74 but for monthly rather than diurnal distributions. Similar to the diurnal plots, the seasonal distributions become flatter when adjusted to meet the 70 ppb and 65 ppb scenarios, especially on the highest O_3 days. This is due to more O_3 decreases during summer months and more O_3 increases in winter months. The O_3 increases in the winter are consistent with the understanding that solar insolation rates are lower in the winter reducing total photochemical activity and shifting the net effect of NO_X emissions on O_3 which can both create O_3 through photochemical pathways and destroy O_3 through titration. In addition, the decreases on the highest O_3 days and increases on the lowest O_3 days show a visible compression of the O_3 distribution in these plots, similar to what was seen in the diurnal plots.

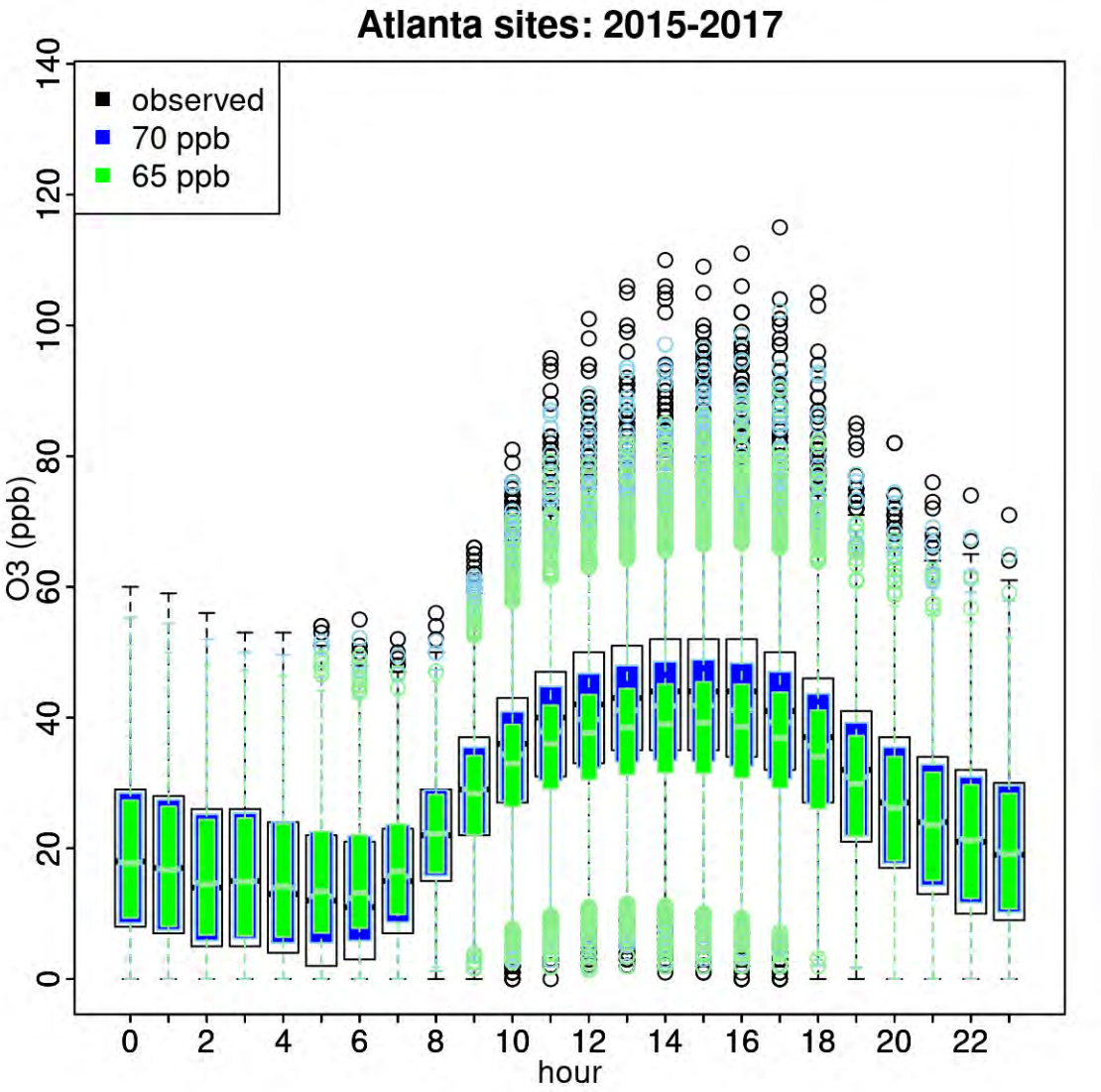


Figure 3C-67. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the Atlanta study area. Note: Observed concentrations in this area have a design value of 75 ppb.

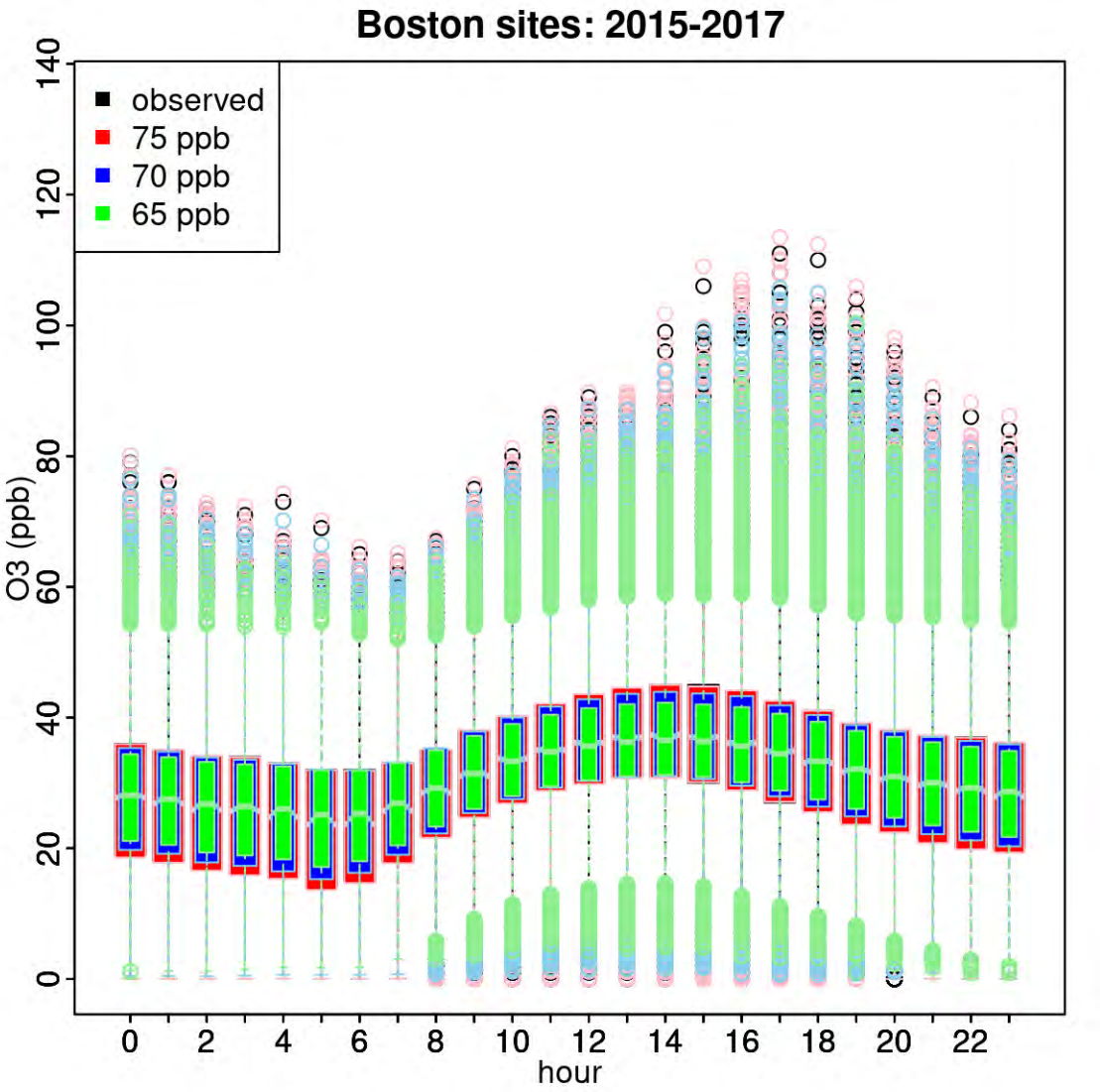


Figure 3C-68. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the Boston study area.

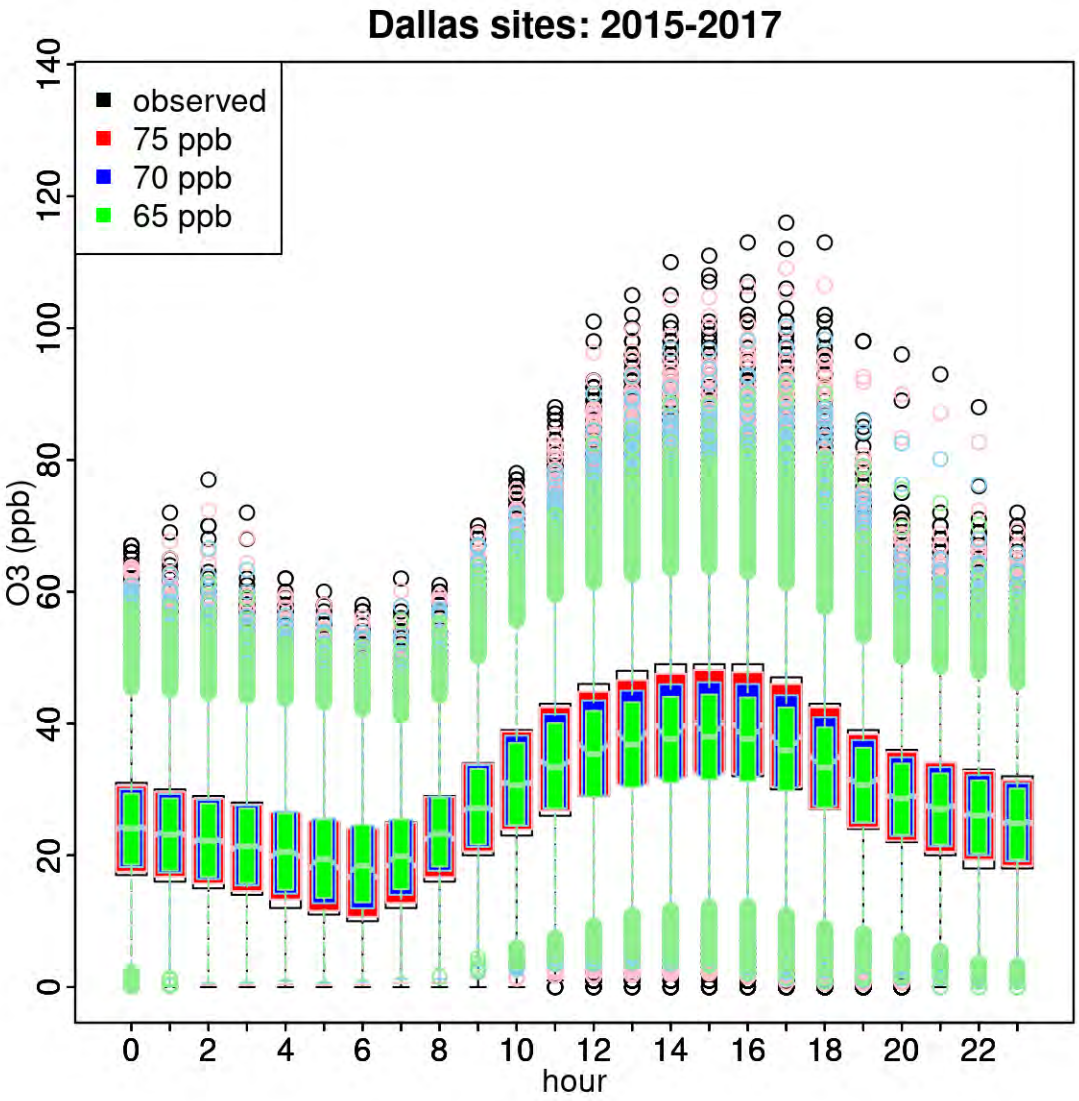


Figure 3C-69. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the Dallas study area.

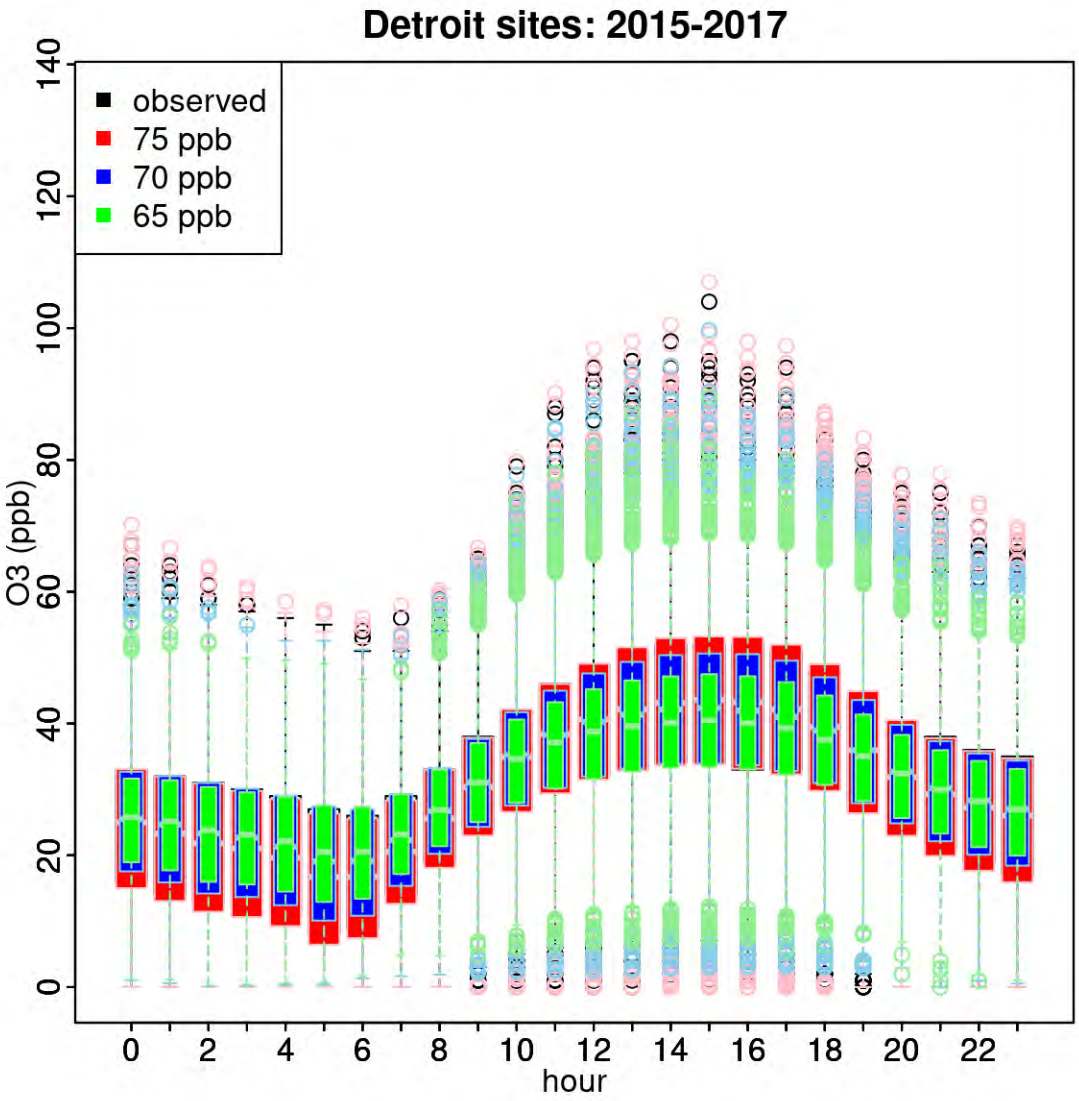


Figure 3C-70. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the Detroit study area.

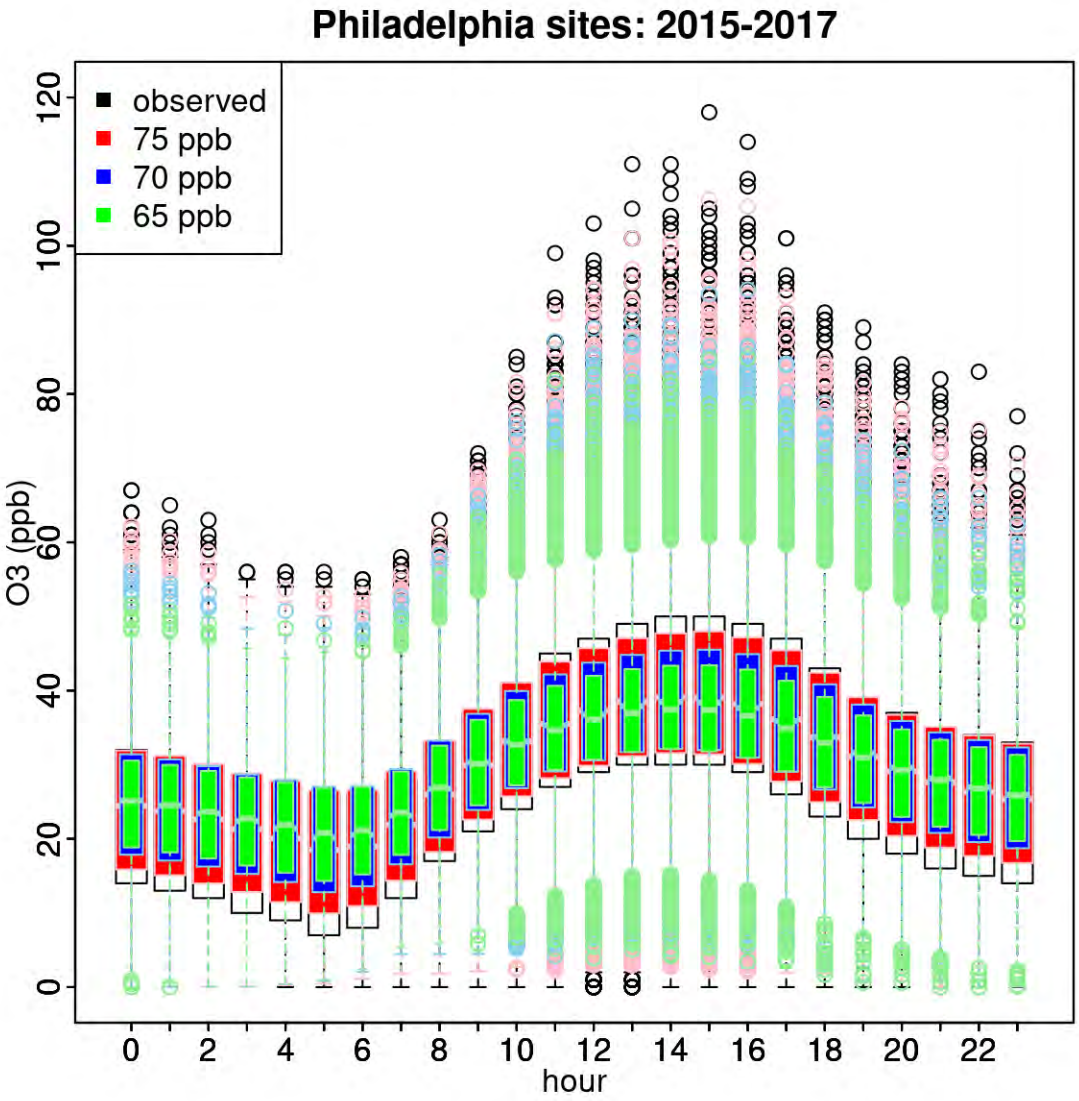


Figure 3C-71. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the Philadelphia study area.

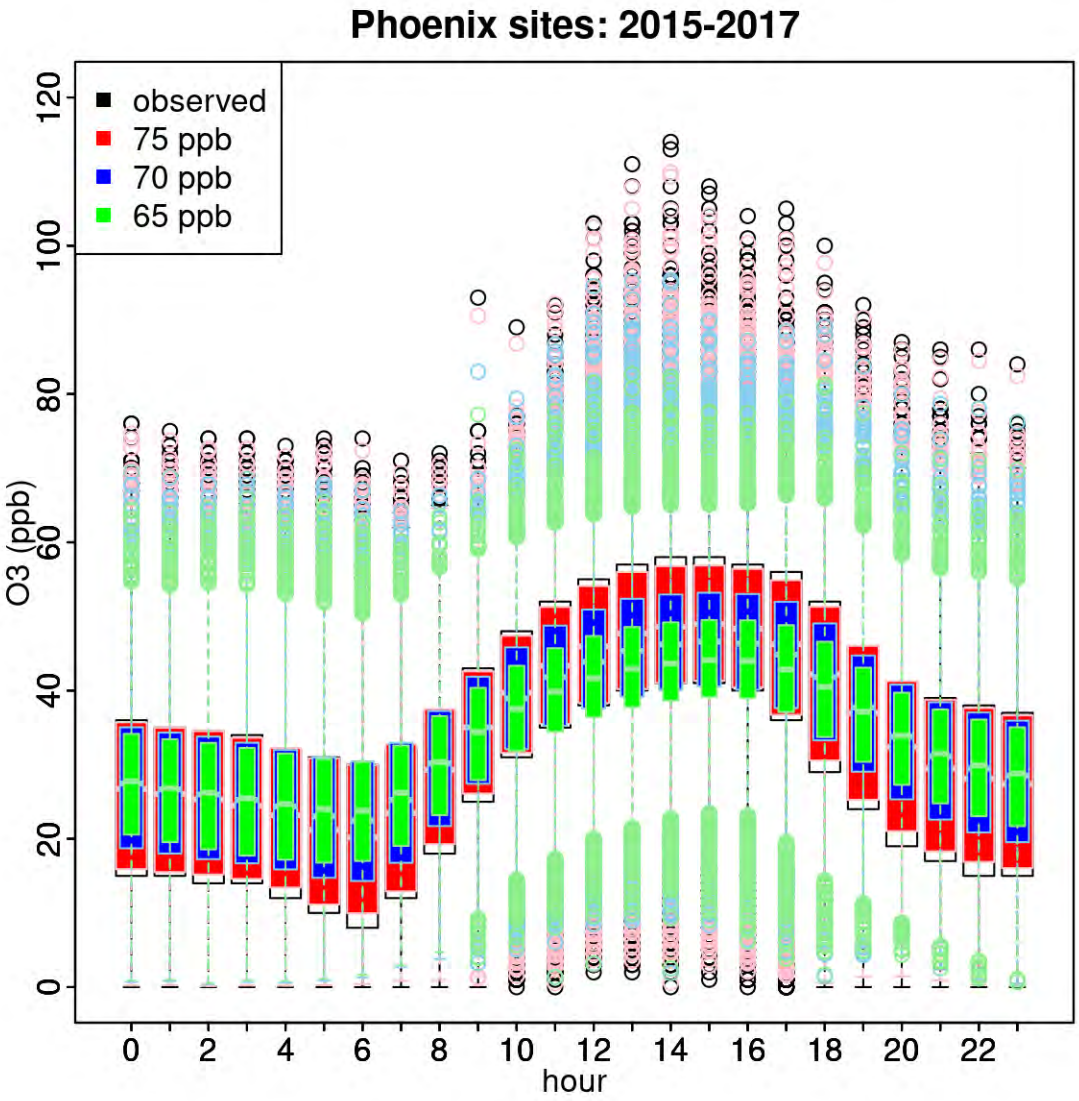


Figure 3C-72. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the Phoenix study area.

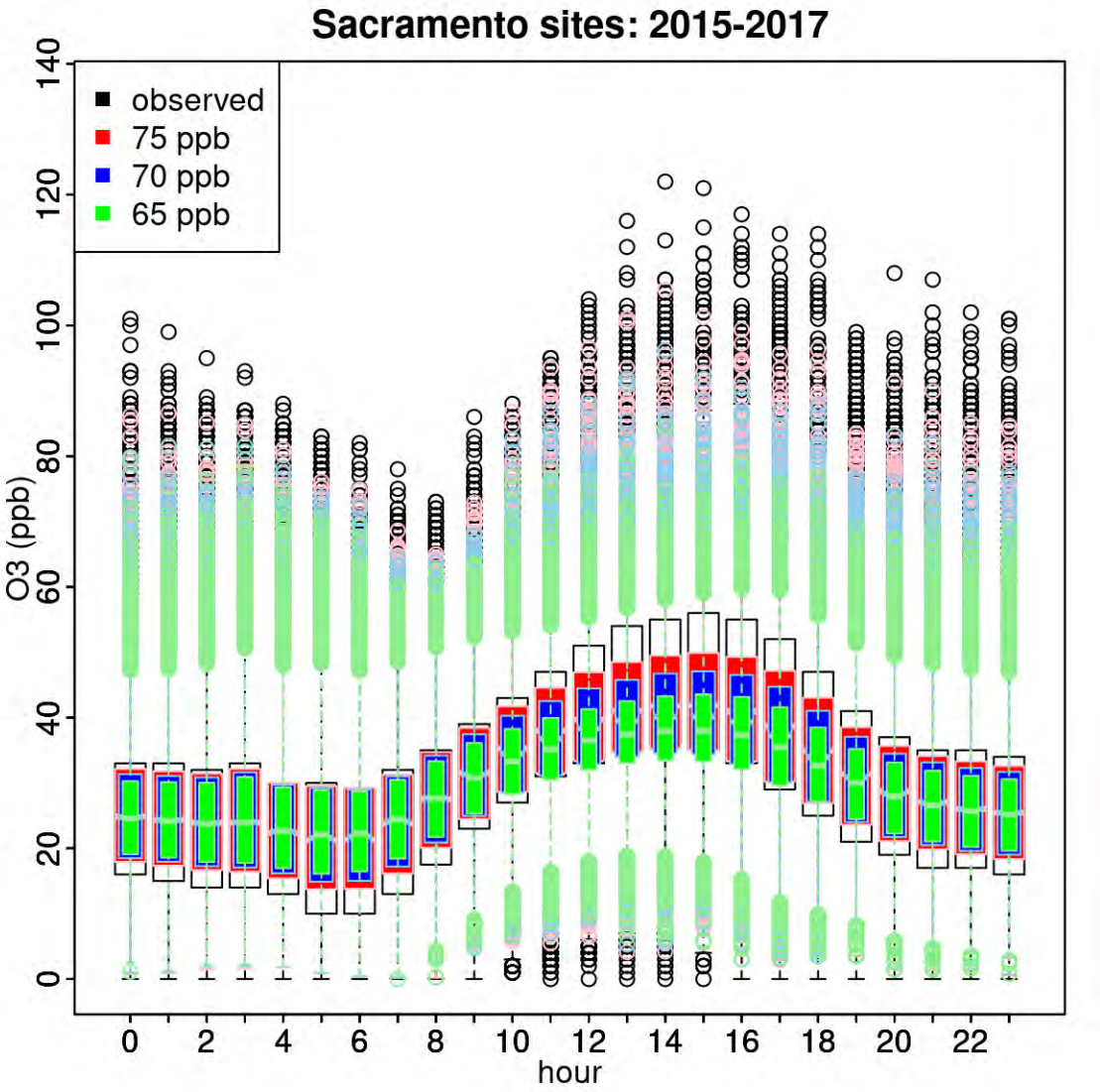


Figure 3C-73. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the Sacramento study area.

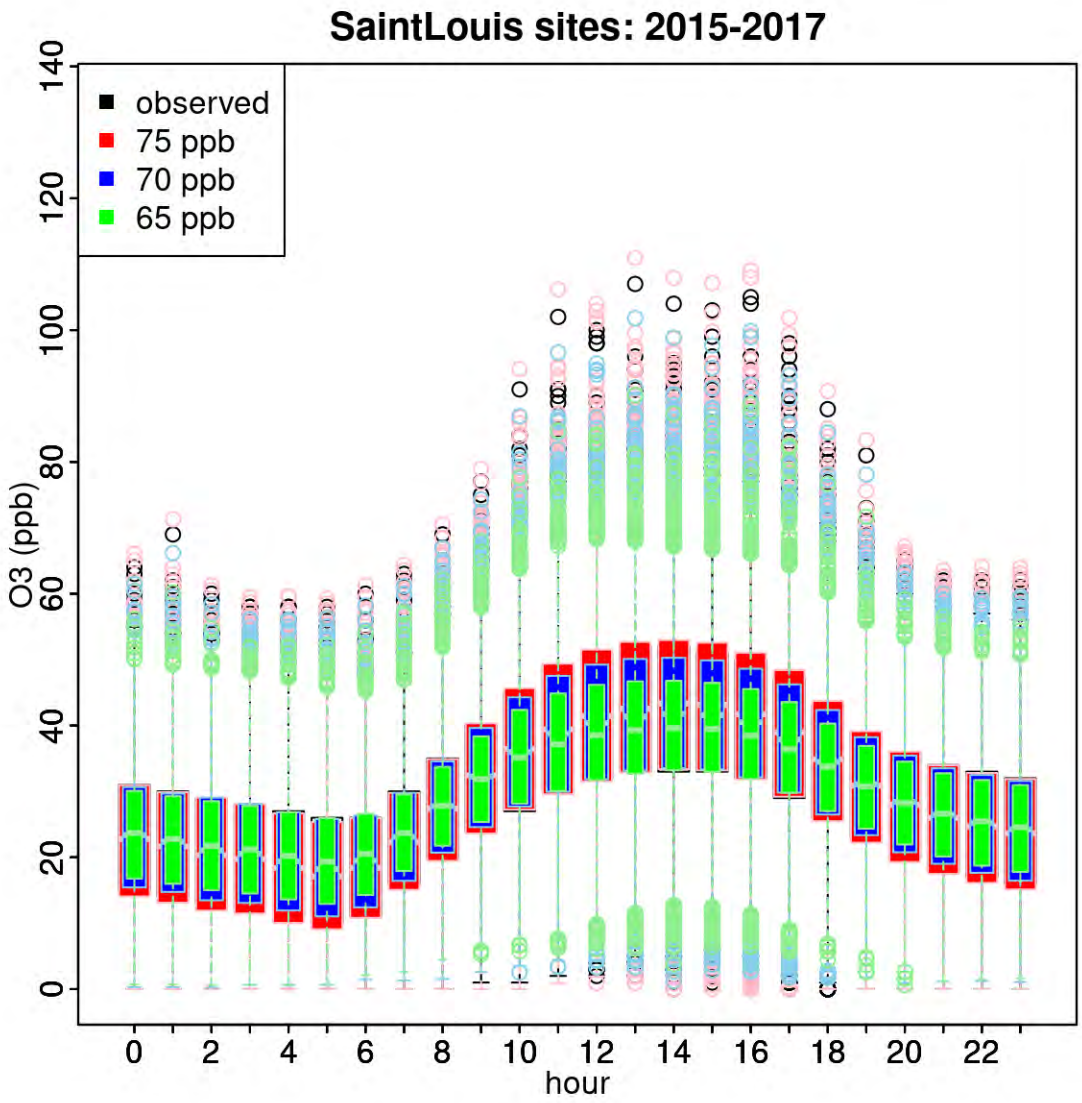


Figure 3C-74. Diurnal distribution of hourly O_3 concentrations at monitoring sites in the St. Louis study area.

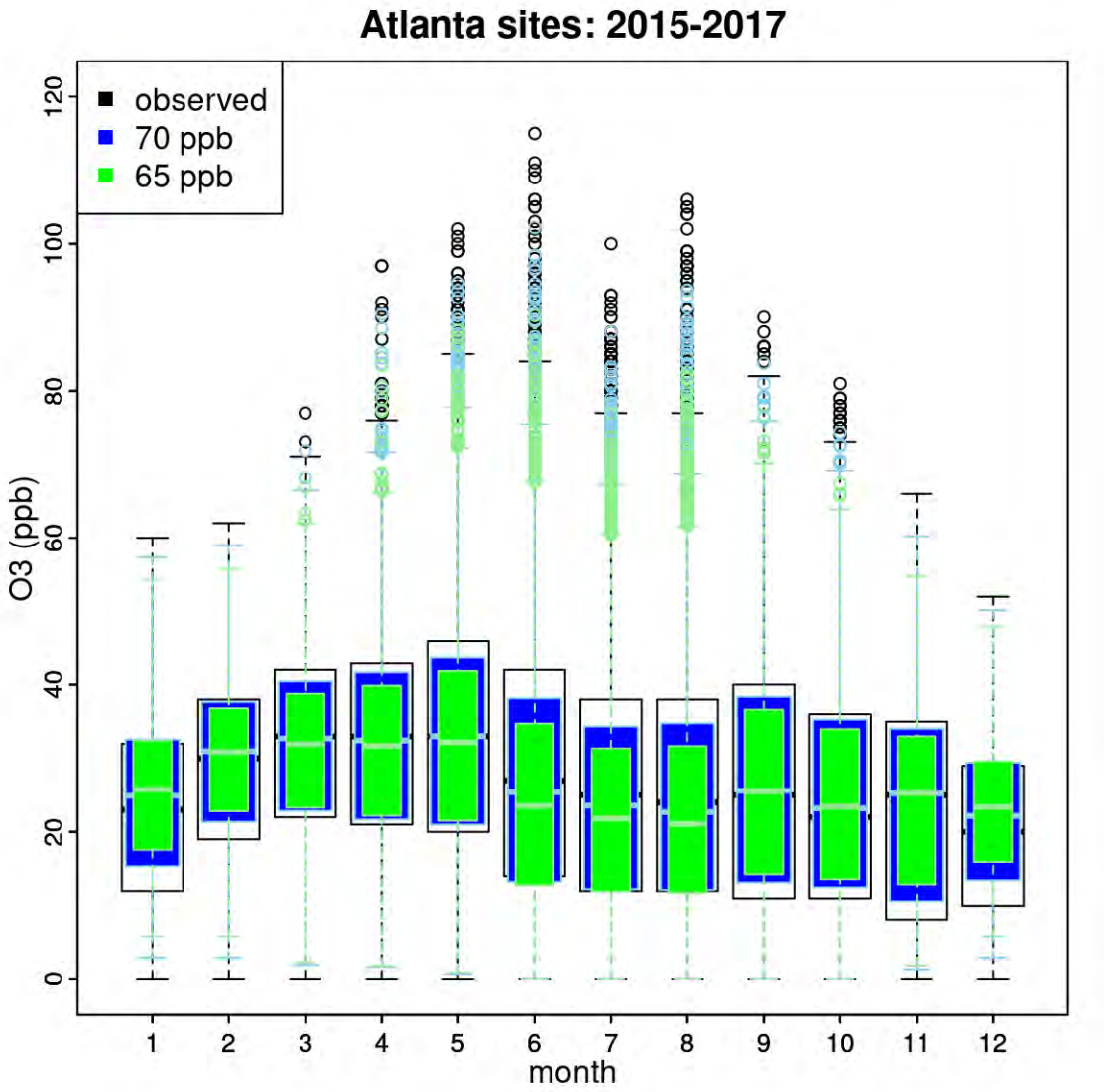


Figure 3C-75. Monthly distribution of hourly O_3 concentrations at monitoring sites in the Atlanta study area. Note: Observed concentrations in this area have a design value of 75 ppb.

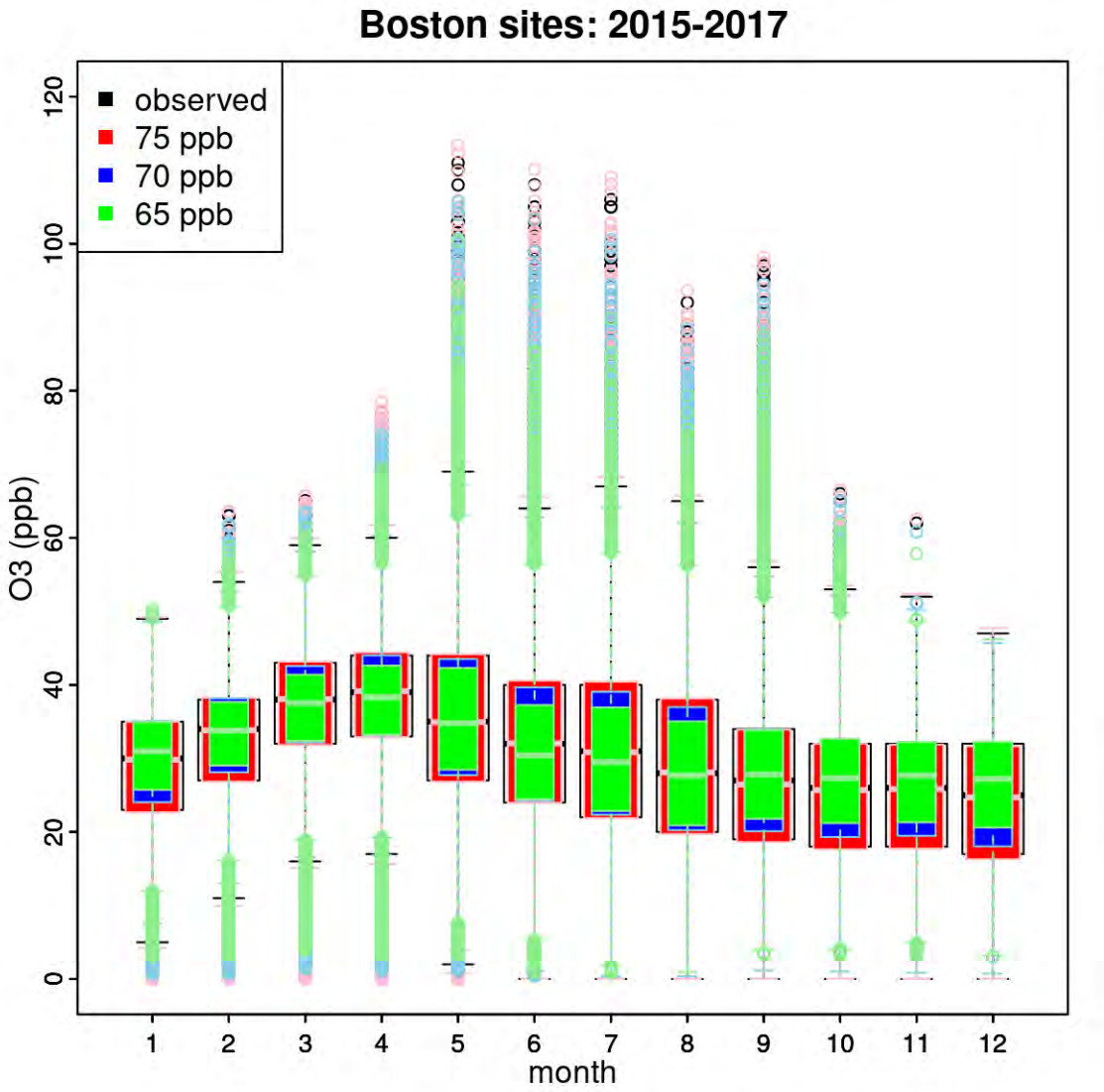


Figure 3C-76. Monthly distribution of hourly O_3 concentrations at monitoring sites in the Boston study area.

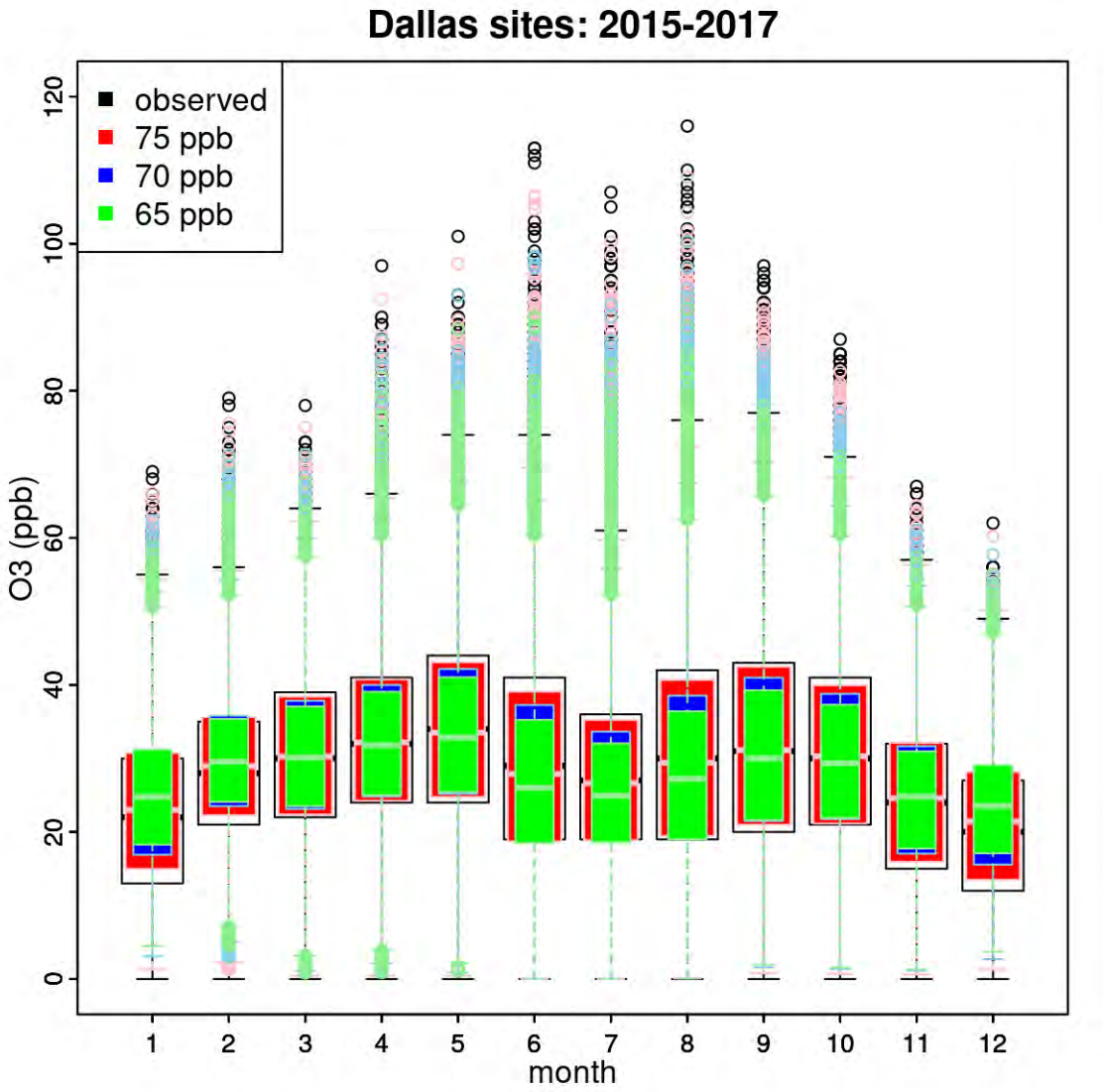


Figure 3C-77. Monthly distribution of hourly O_3 concentrations at monitoring sites in the Dallas study area.

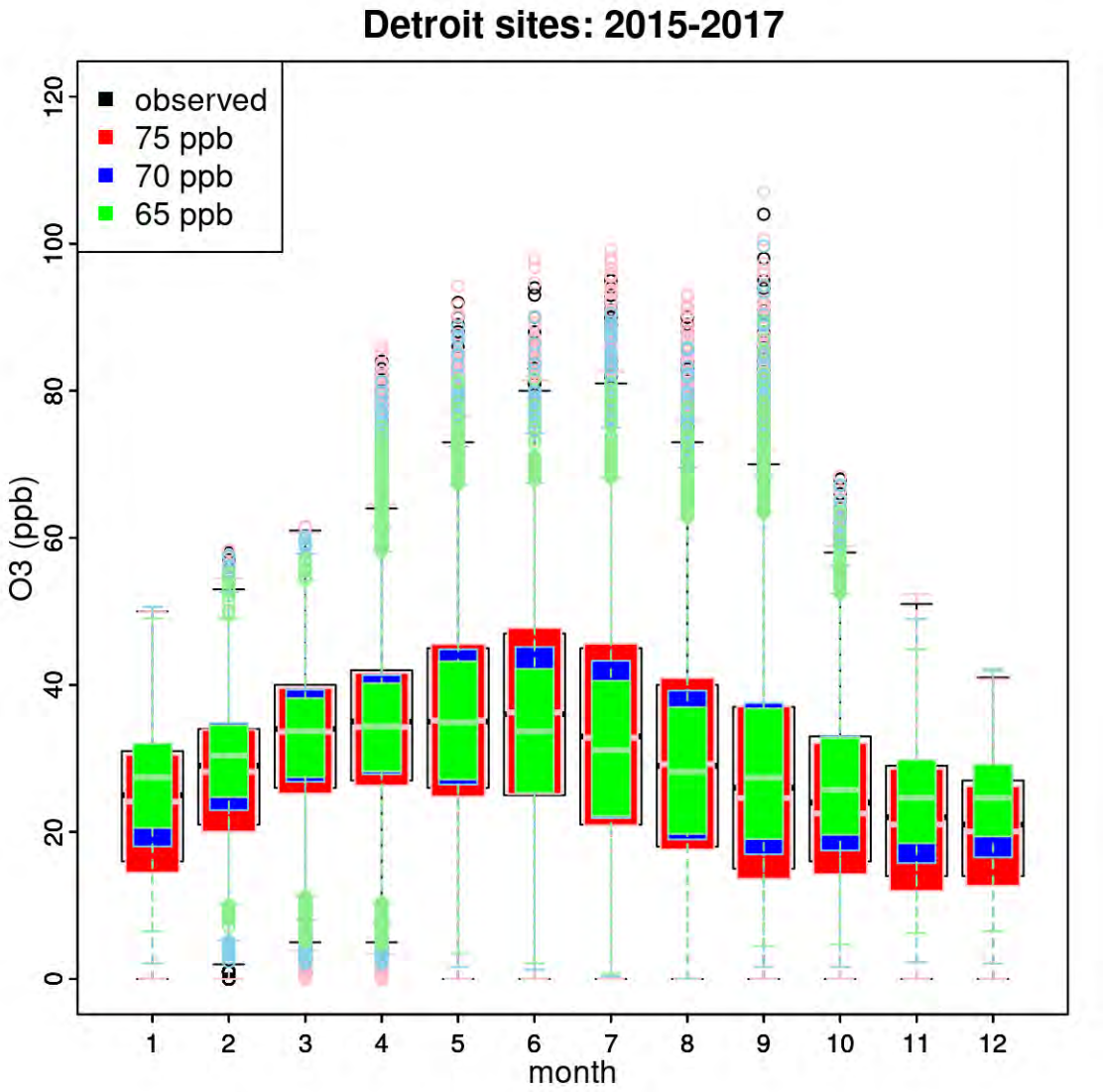


Figure 3C-78. Monthly distribution of hourly O_3 concentrations at monitoring sites in the Detroit study area.

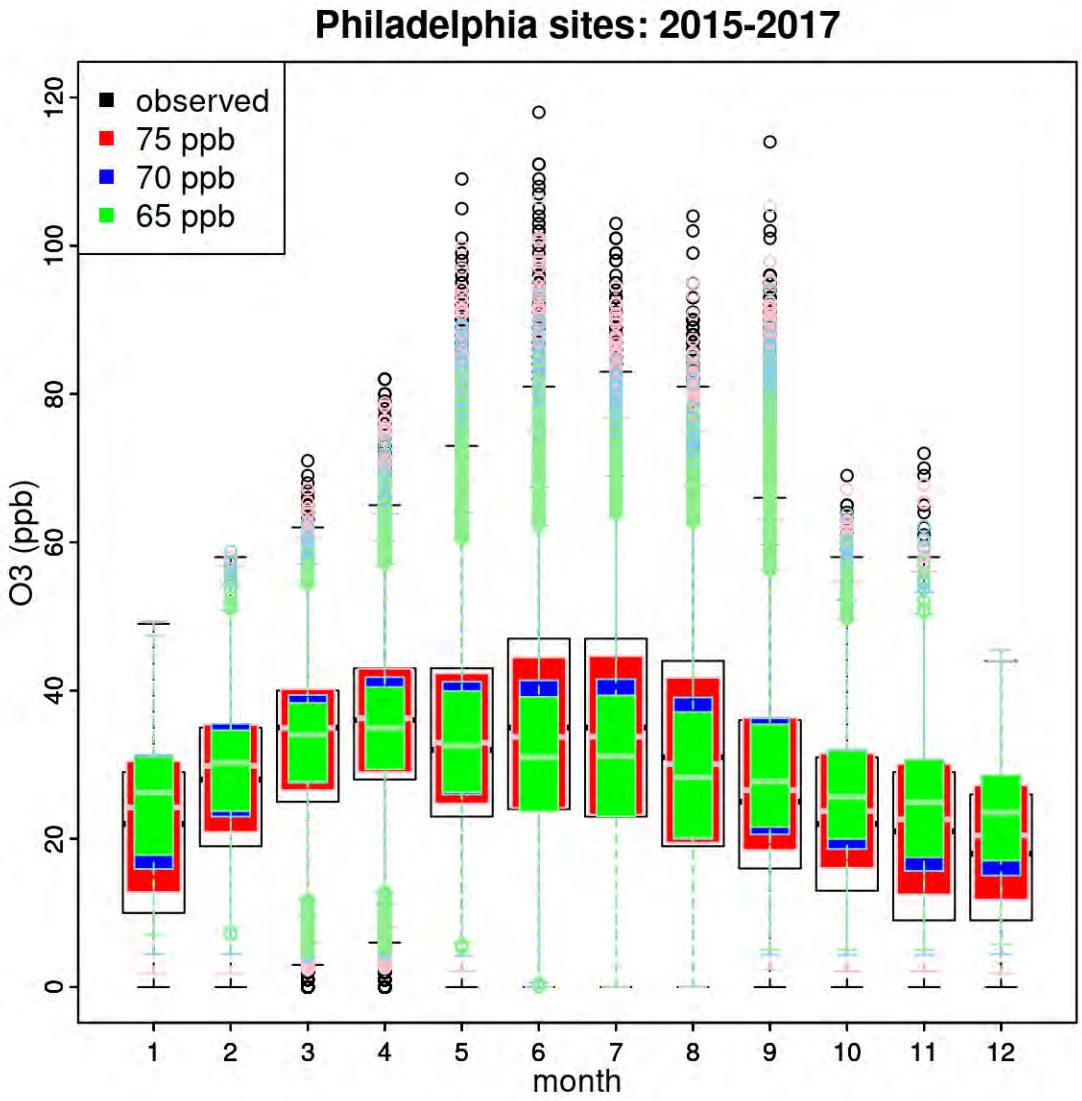


Figure 3C-79. Monthly distribution of hourly O_3 concentrations at monitoring sites in the Philadelphia study area.

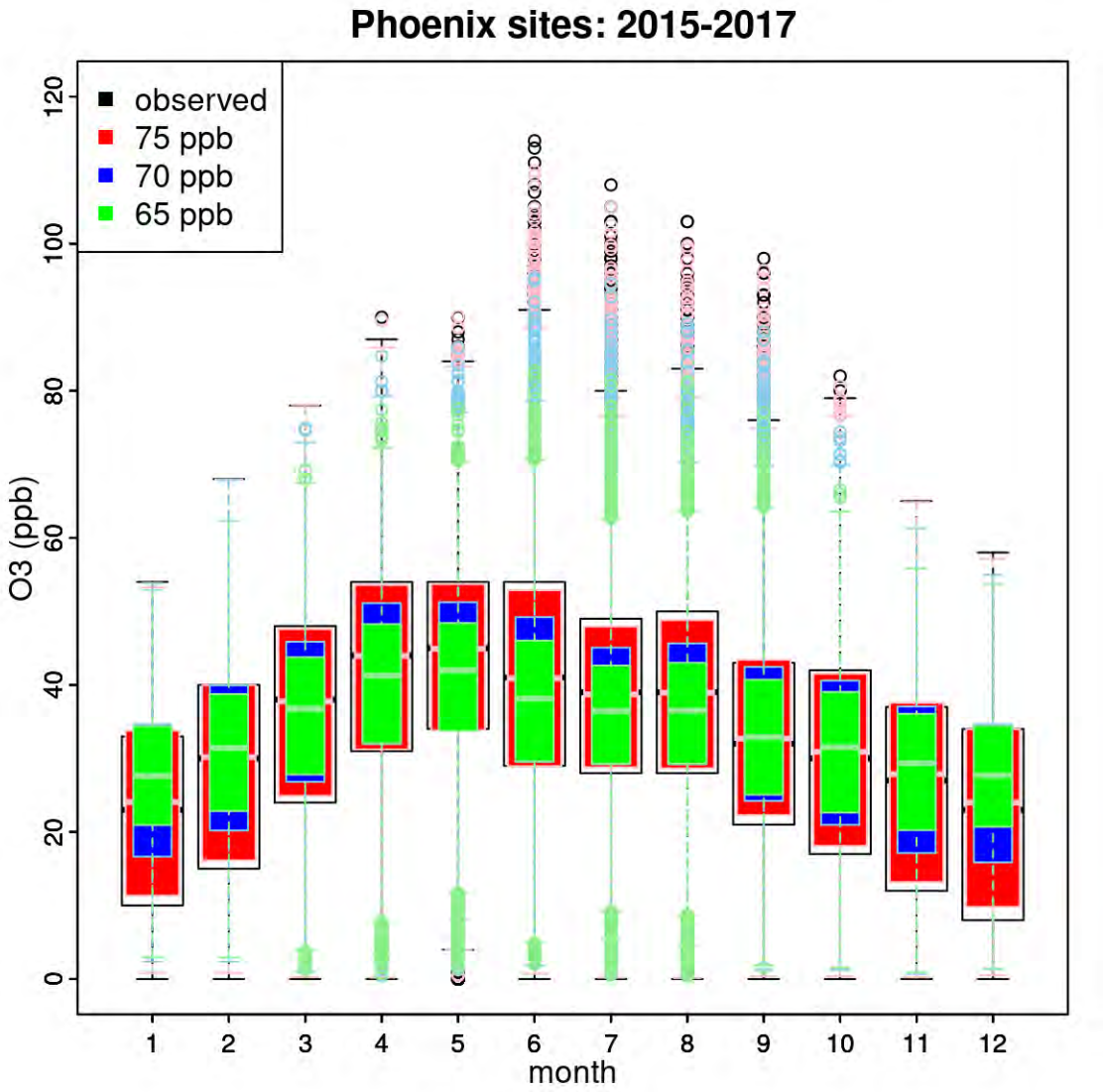


Figure 3C-80. Monthly distribution of hourly O_3 concentrations at monitoring sites in the Phoenix study area.

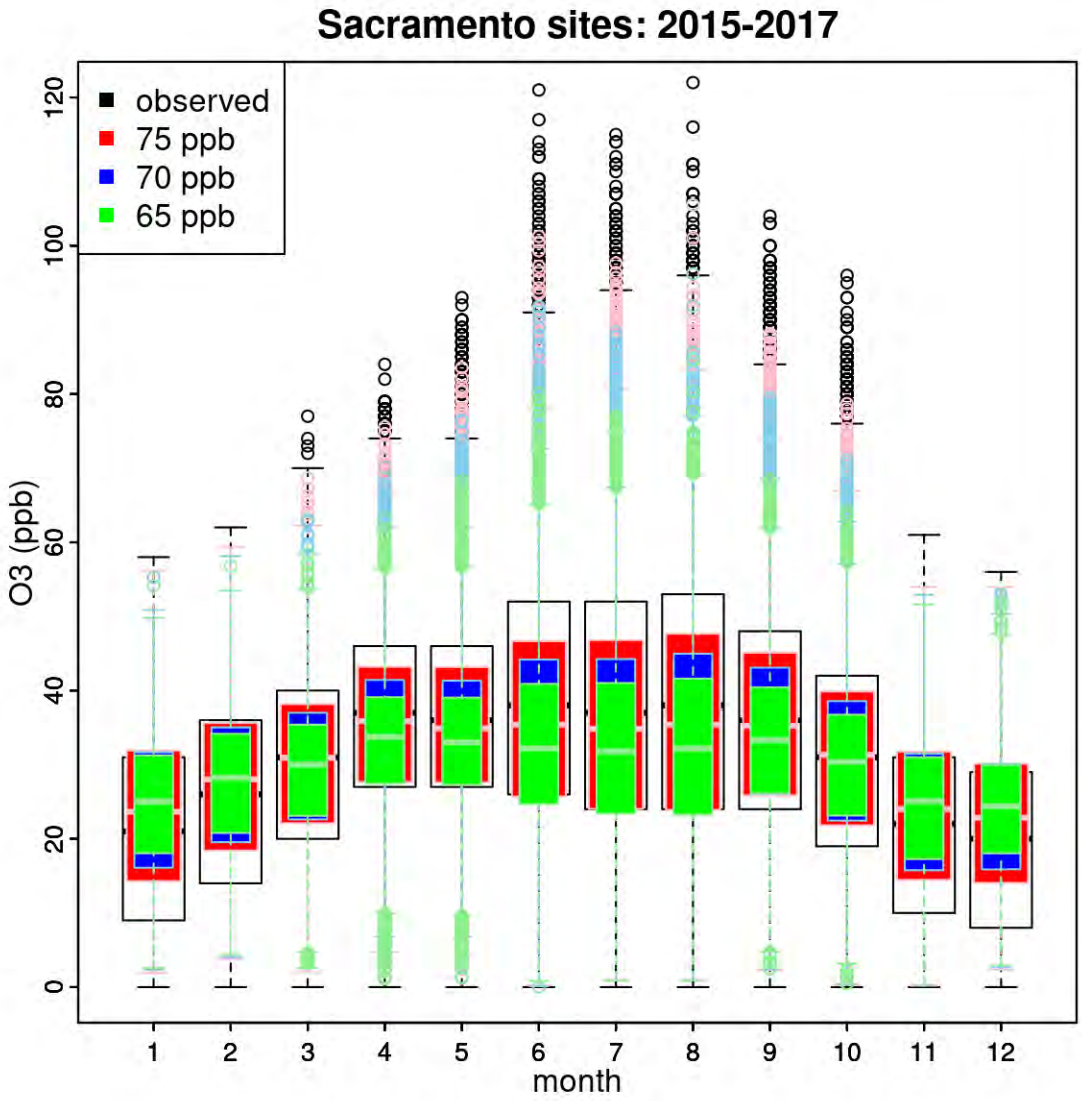


Figure 3C-81. Monthly distribution of hourly O_3 concentrations at monitoring sites in the Sacramento study area.

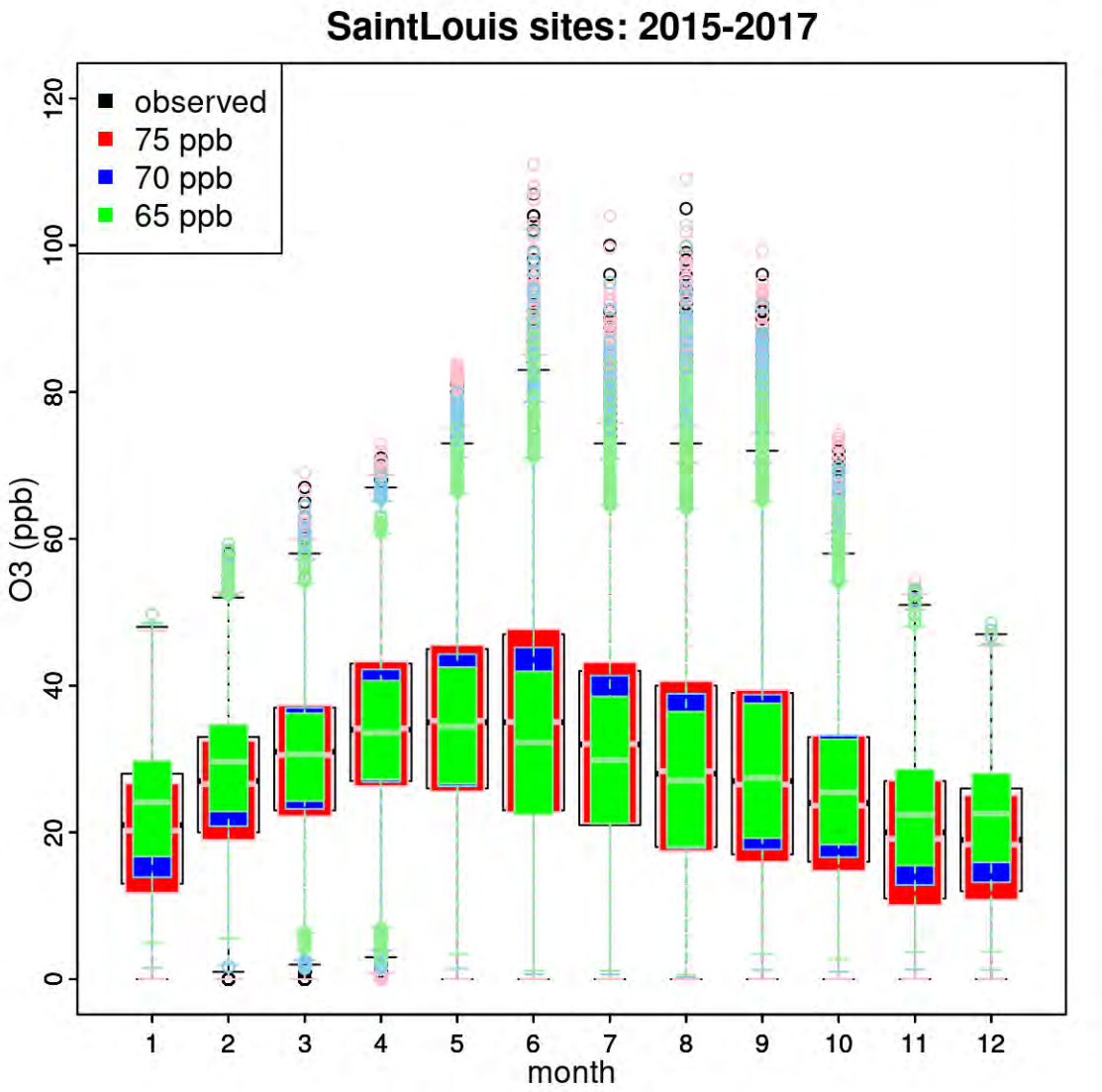


Figure 3C-82. Monthly distribution of hourly O_3 concentrations at monitoring sites in the St. Louis study area.

3C.7.3 Air Quality Inputs for the Exposure and Risk Analyses

The air quality inputs for the exposure and risk analyses discussed in chapter 3 of this PA include spatial surfaces of hourly O_3 concentrations estimated for each census tract in the eight urban study areas using the VNA technique described in section 3C.6. In this section, we present three types of figures which summarize the data from the hourly VNA surfaces for observed air

quality, and air quality adjusted to meet the current standard of 70 ppb, and air quality adjusted to meet alternative scenarios of 75 ppb[18] and 65 ppb.

The first set of figures (Figure 3C-83 through Figure 3C-90) shows density scatter plots of the change in MDA8 O_3 concentrations versus the observed concentrations based on the hourly VNA estimates in each study area. In each of these figures, the left-hand panel shows the observed MDA8 values (x-axis) versus the change in those values that occur when air quality is adjusted for the 75 ppb scenario (y-axis). The middle panel shows the MDA8 values for air quality adjusted to meet the 75 ppb scenario (x-axis) versus the additional change in those values that occur when air quality is adjusted to meet the current standard of 70 ppb (y-axis). Finally, the right-hand panels show the corresponding changes from the current standard to the 65 ppb scenario. Within each panel, the x and y values are rounded to the nearest integer and colored to show the relative frequency of each 1 x 1 ppb square within the plot region. Values falling outside of the plot region were set to the nearest value within the plot region, and frequencies above the range in the color bar were set to the highest value within the color bar.

The second set of figures (Figure 3C-91 through Figure 3C-106) provides maps of the adjusted design values (3-year average of the annual 4th highest MDA8 values) and May-September average MDA8 values based on the ambient air data and the hourly VNA surfaces, as well as difference maps showing the changes between these surfaces. For the difference maps, the panels on the left show the changes in these values that occur when air quality is adjusted for the 75 ppb scenario, the panels in the middle show the additional changes in these values that occur when air quality is further adjusted to meet the current standard of 70 ppb, and the right-hand panels show the additional changes that occur then air quality is further adjusted for the 65 ppb scenario. Within each panel, squares show values based on observed data at ambient air monitoring sites while circles show values based on VNA estimates at census tract centroids. While each panel shows both monitors in the study area for each selected urban study area as well as some additional monitors located outside of the study area, only the monitors located within the study area were used when determining the emissions reductions necessary to meet the various standards.

The third set of figures (Figure 3C-107 through Figure 3C-114) shows changes in design values (3-year average of the annual 4th highest MDA8 values) and May-September average MDA8 values in the eight urban case study areas versus population and population density. The total population and population density information for each census tract were obtained from the 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.

population stratified by the change in design value or seasonal average. The bars are also color-coded by population density bin. Values falling outside of the plot region set to the nearest values within the plot region.

In general, the density scatter plots show that the HDDM adjustment procedure predicts increases in MDA8 O_3 at low ambient air concentrations and decreases in MDA8 O_3 at high concentrations (Figure 3C-83 through Figure 3C-90). The vast majority of the increases in MDA8 O_3 occur at ambient air concentrations below 50 ppb. The relationship between the starting concentrations and the changes in these values based on the HDDM adjustments is fairly linear with strong negative correlation in all eight urban study areas.[19] In some study areas, such as Philadelphia and Detroit, there is a bimodal pattern near the center of the distribution, which may be indicative of differing behavior near the urban population center versus the surrounding suburban areas.

The maps reveal consistent spatial patterns of O_3 changes across the urban study areas. The design values generally decreased when air quality was adjusted to meet the current standard of 70 ppb[20] and continued to decrease when air quality was further adjusted for the 65 ppb scenario (Figure 3C-91 through Figure 3C-106). The design values tend to decrease more quickly in suburban and rural areas than in the urban population centers. The May-September "seasonal" average MDA8 values also followed this trend to some extent, although the behavior in the urban population centers varied slightly amongst the urban study areas (Figure 3C-107 through Figure 3C-114). In summary, these figures show that using the CAMx/HDDM adjustment methodology, peak O_3 concentrations are reduced in urban study areas with large 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.

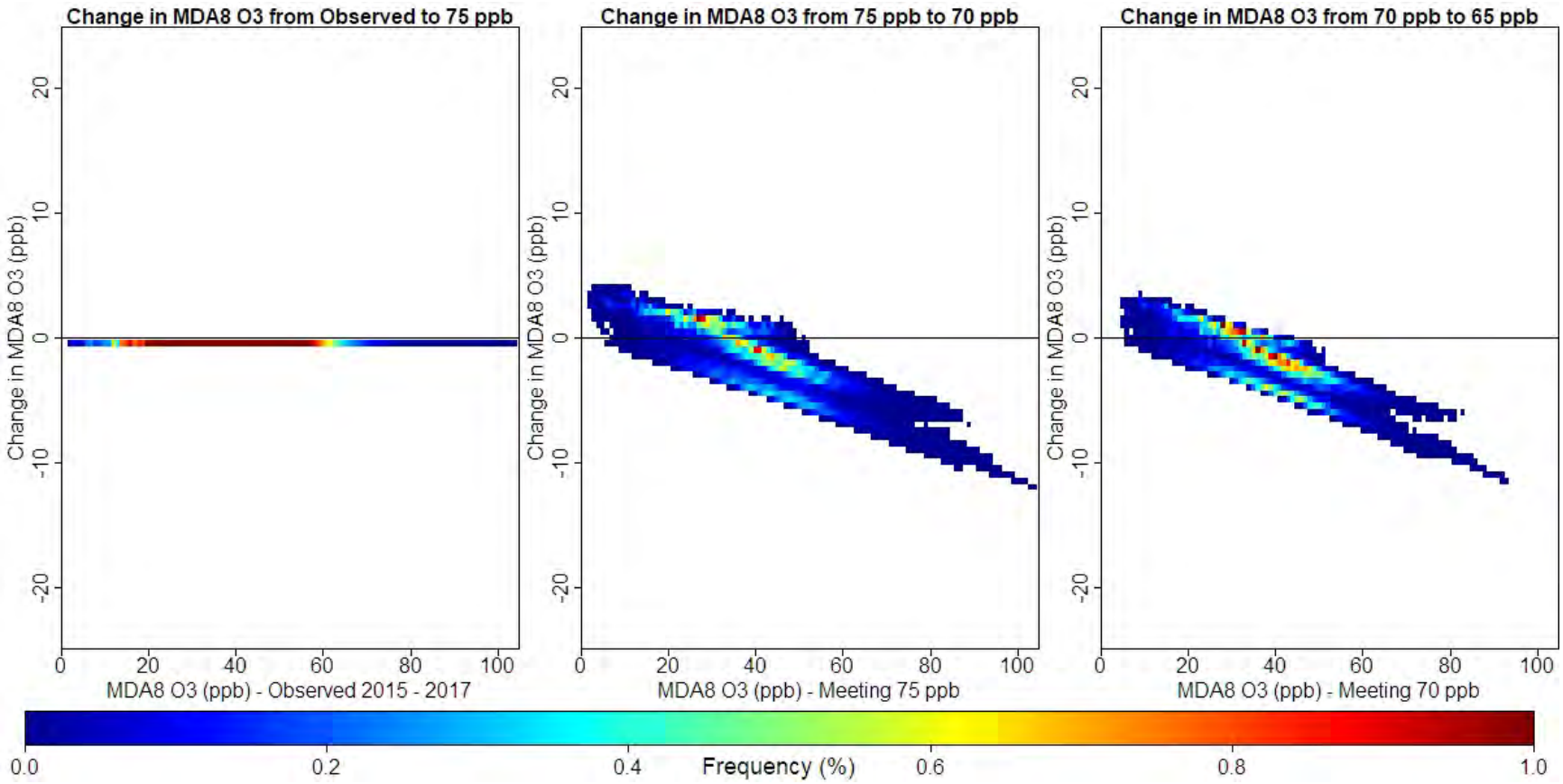


Figure 3C-83. Changes in MDA8 O_3 based on HDDM adjustments in the Atlanta study area.

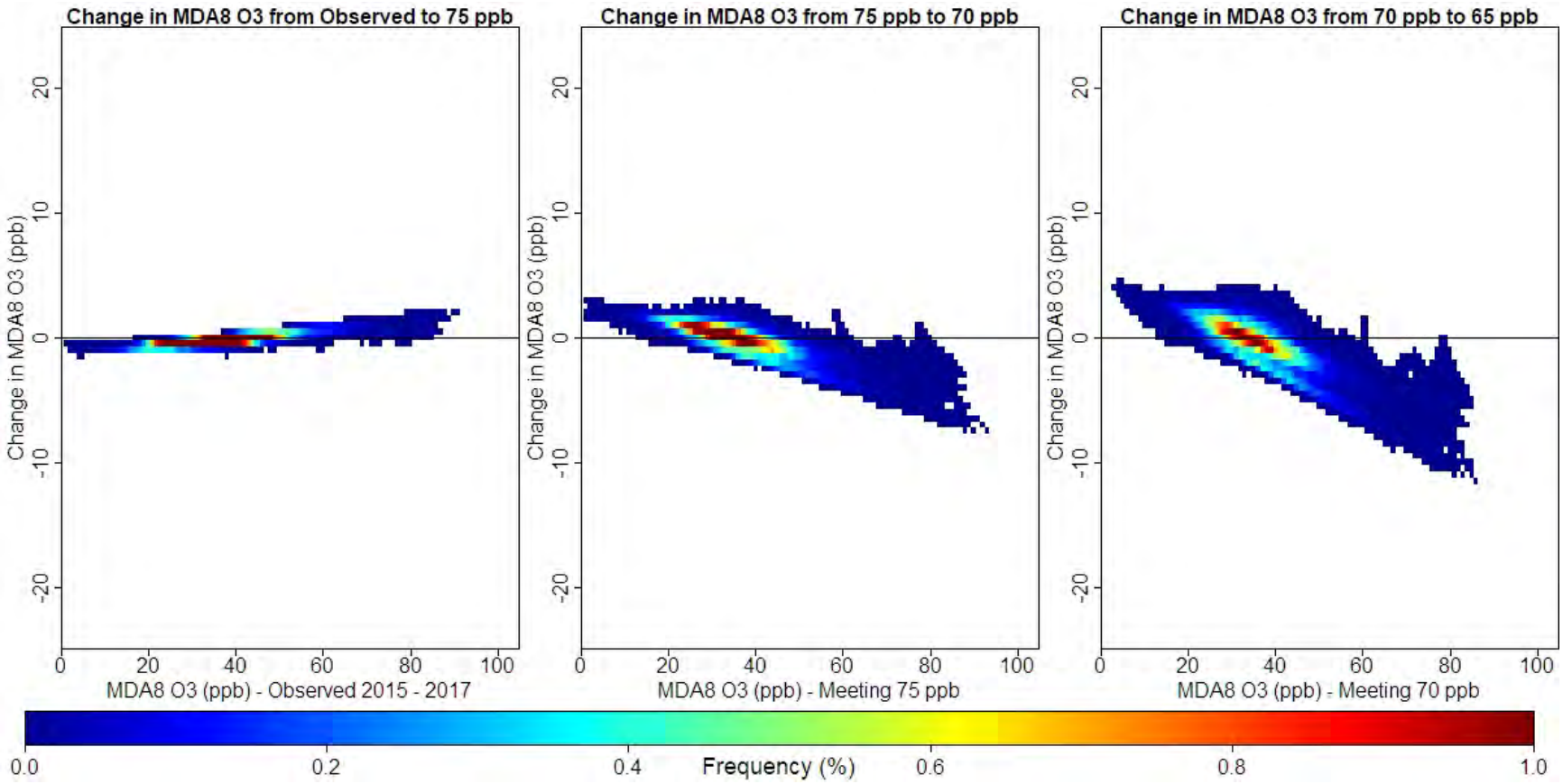


Figure 3C-84. Changes in MDA8 O_3 based on HDDM adjustments in the Boston study area.

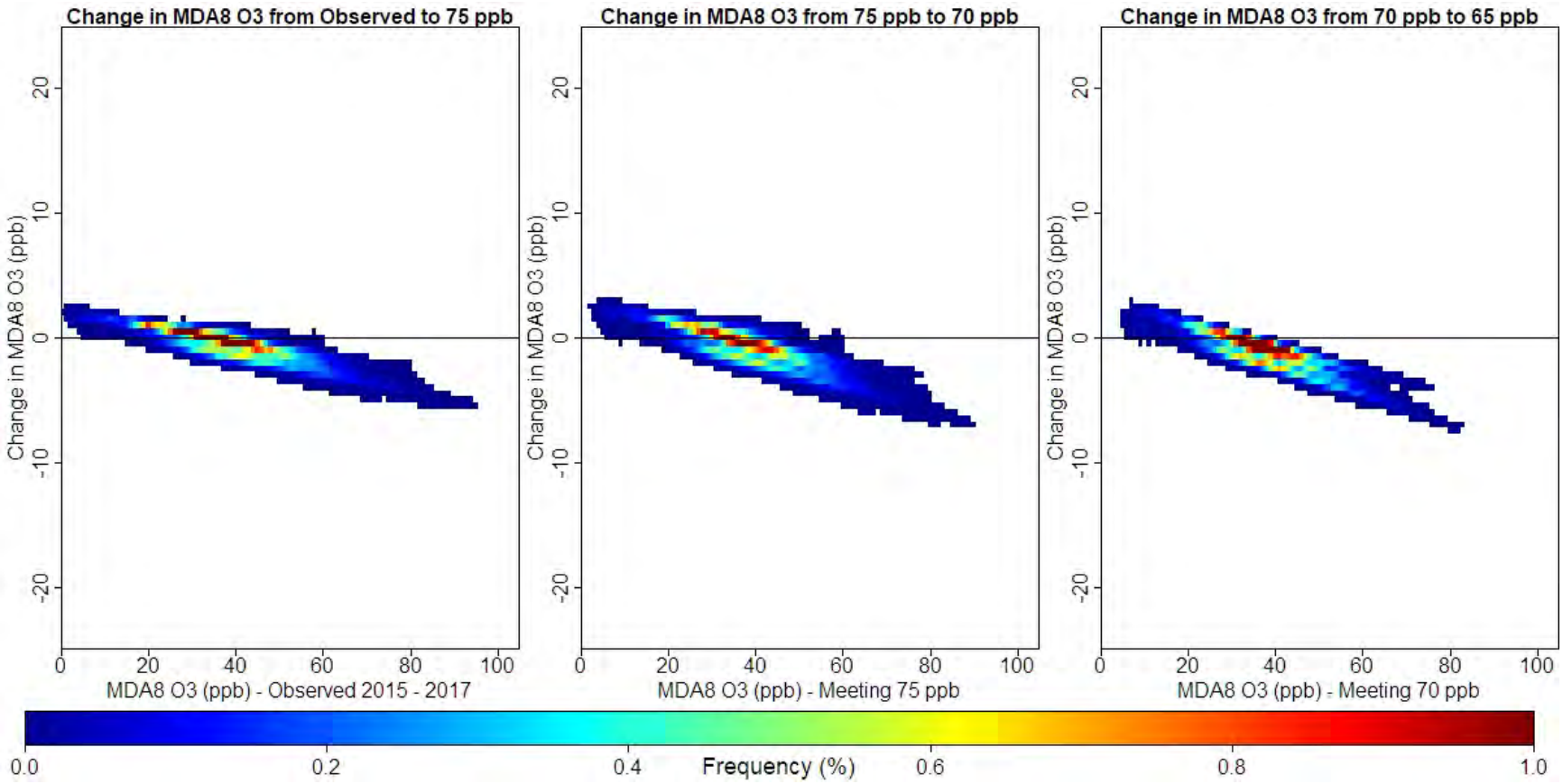


Figure 3C-85. Changes in MDA8 O_3 based on HDDM adjustments in the Dallas study area.

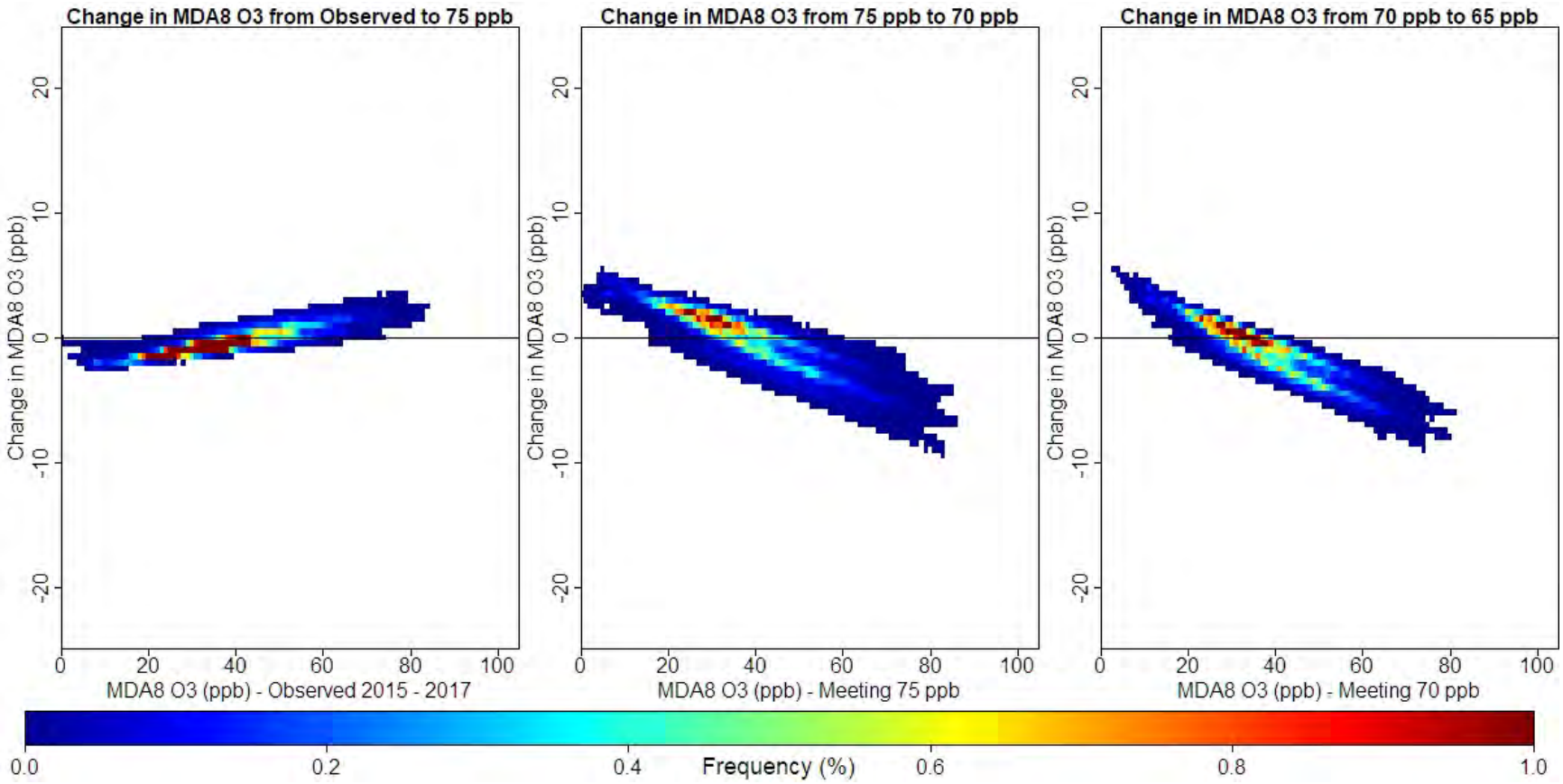


Figure 3C-86. Changes in MDA8 O_3 based on HDDM adjustments in the Detroit study area.

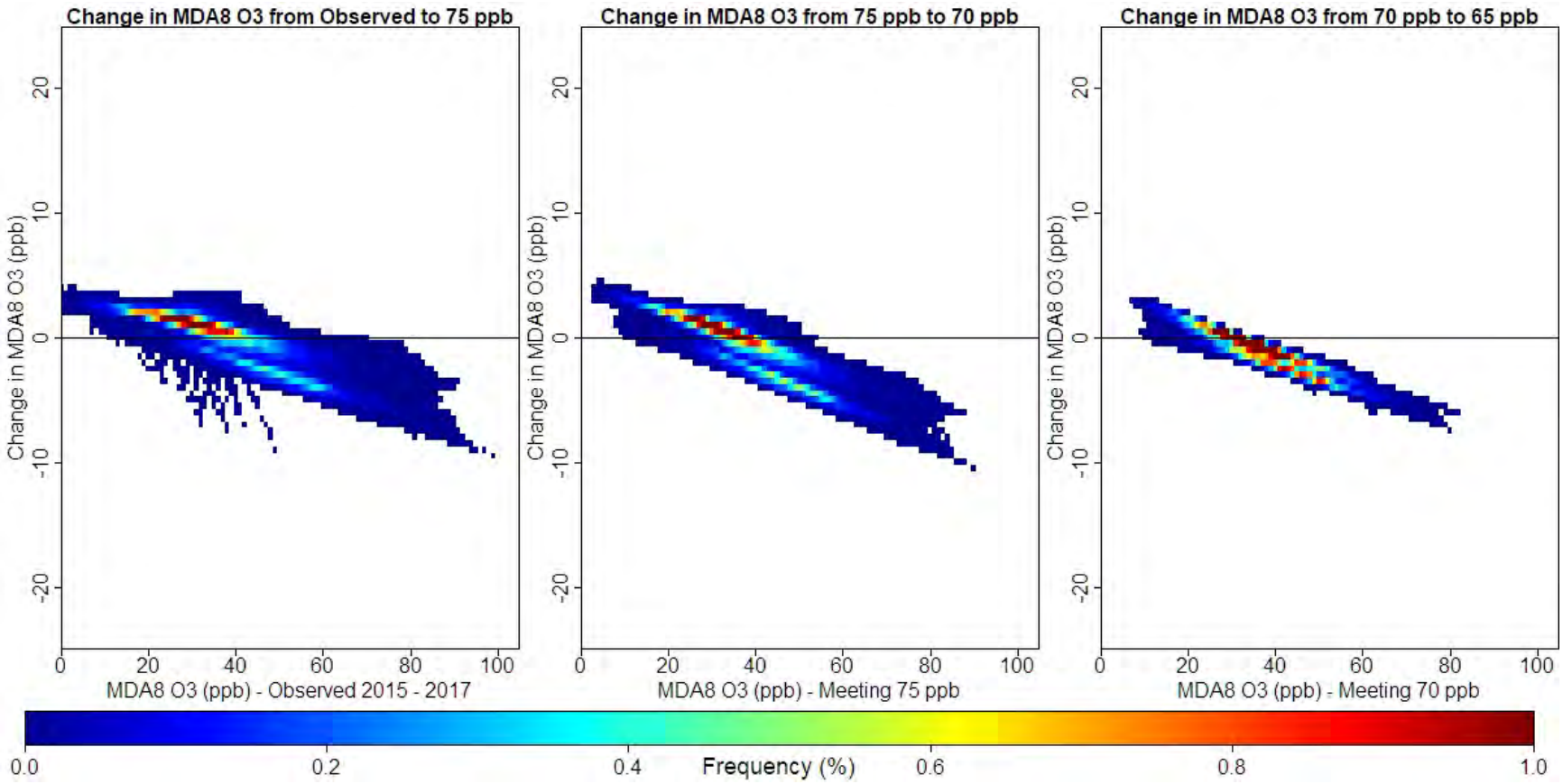


Figure 3C-87. Changes in MDA8 O_3 based on HDDM adjustments in the Philadelphia study area.

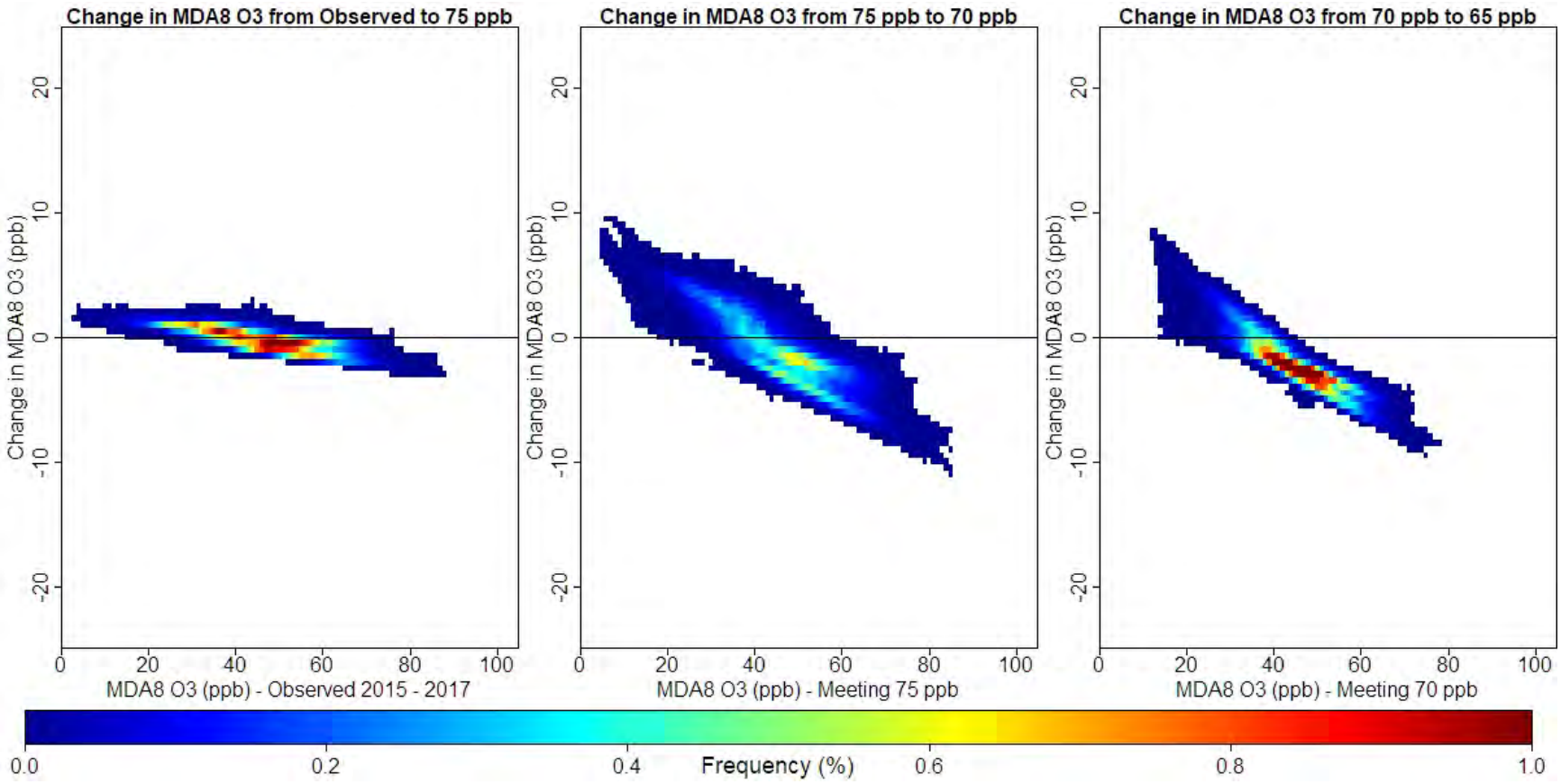


Figure 3C-88. Changes in MDA8 O_3 based on HDDM adjustments in the Phoenix study area.

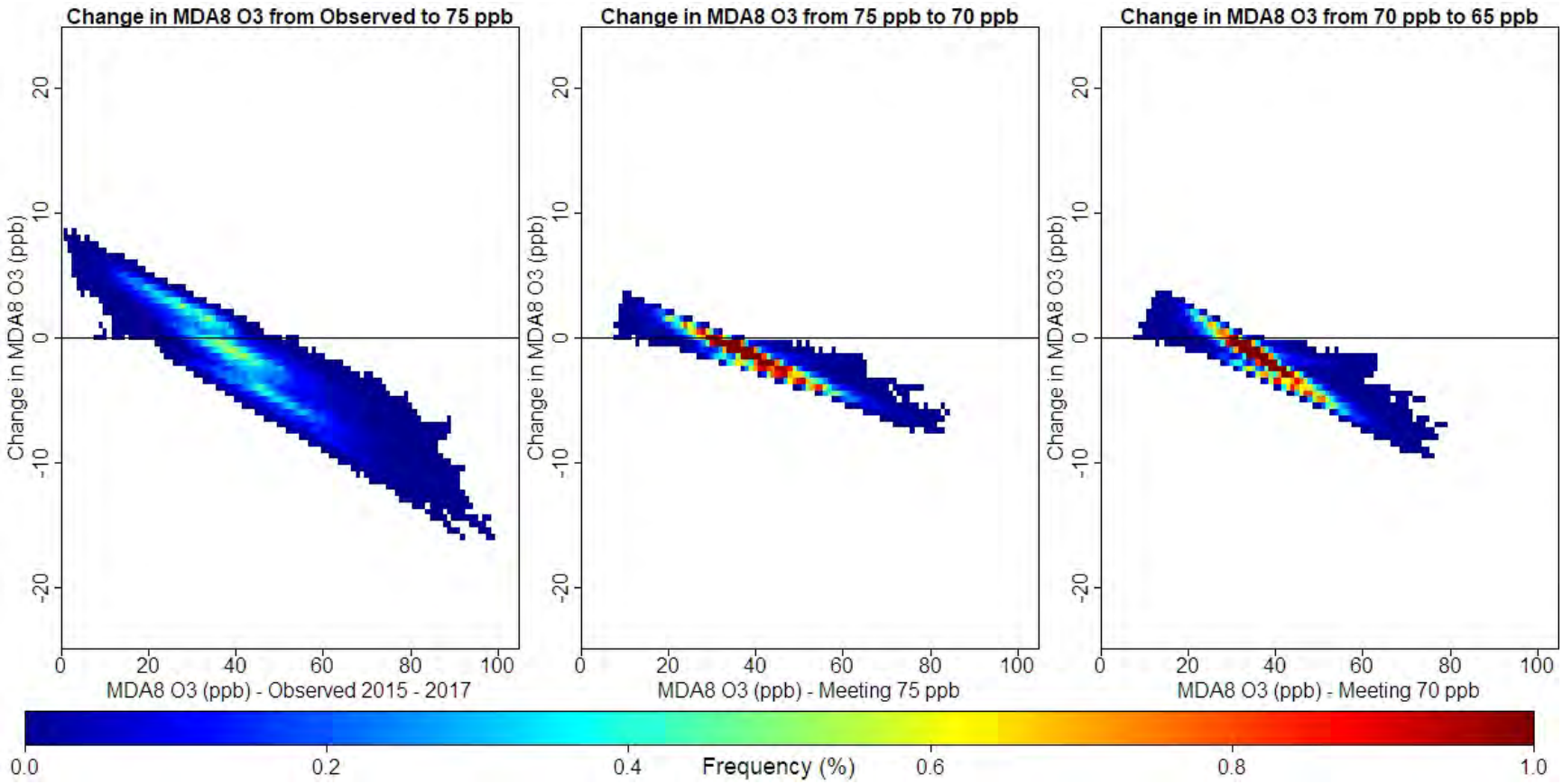


Figure 3C-89. Changes in MDA8 O_3 based on HDDM adjustments in the Sacramento study area.

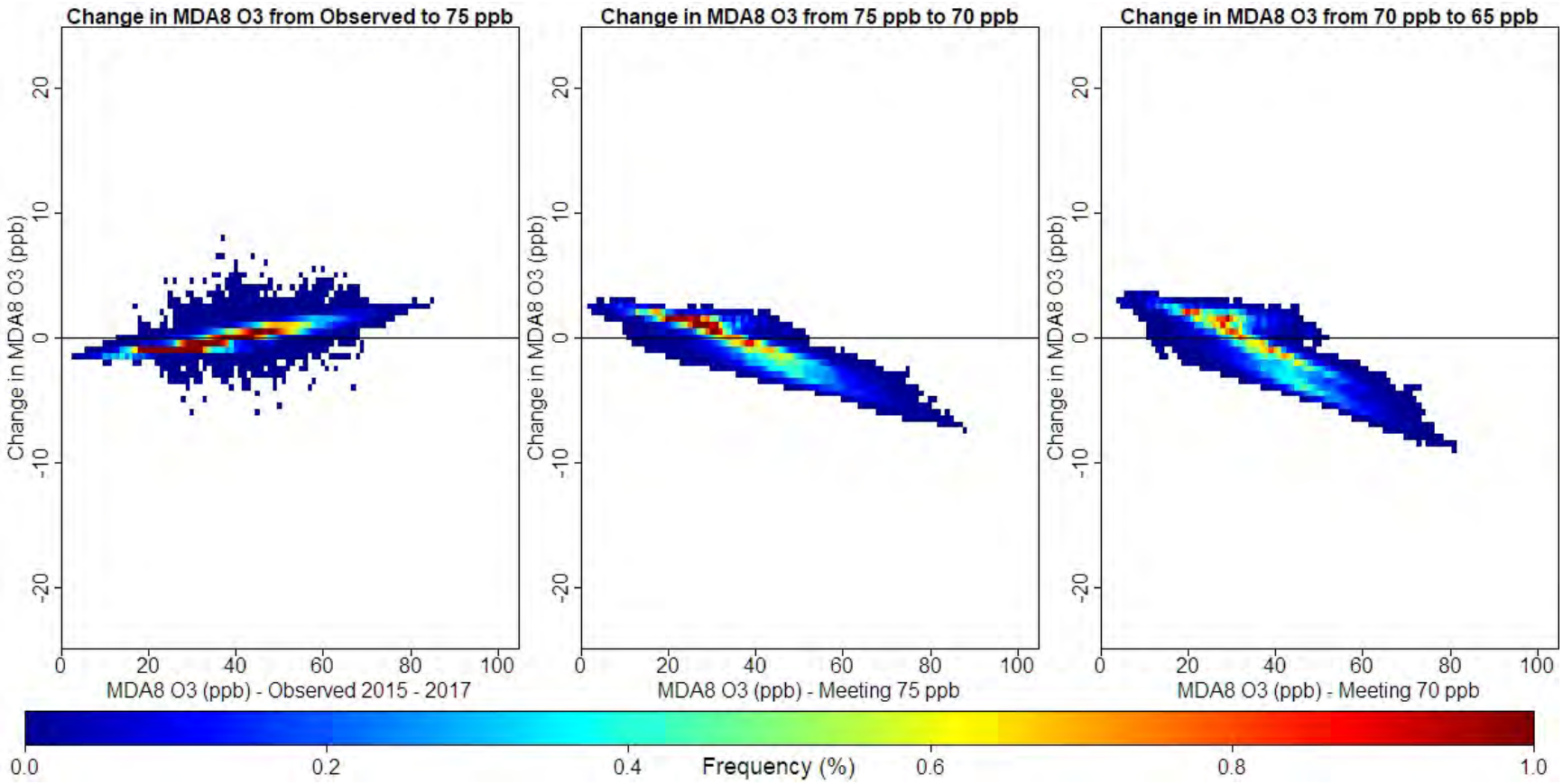


Figure 3C-90. Changes in MDA8 O_3 based on HDDM adjustments in the St. Louis study area.

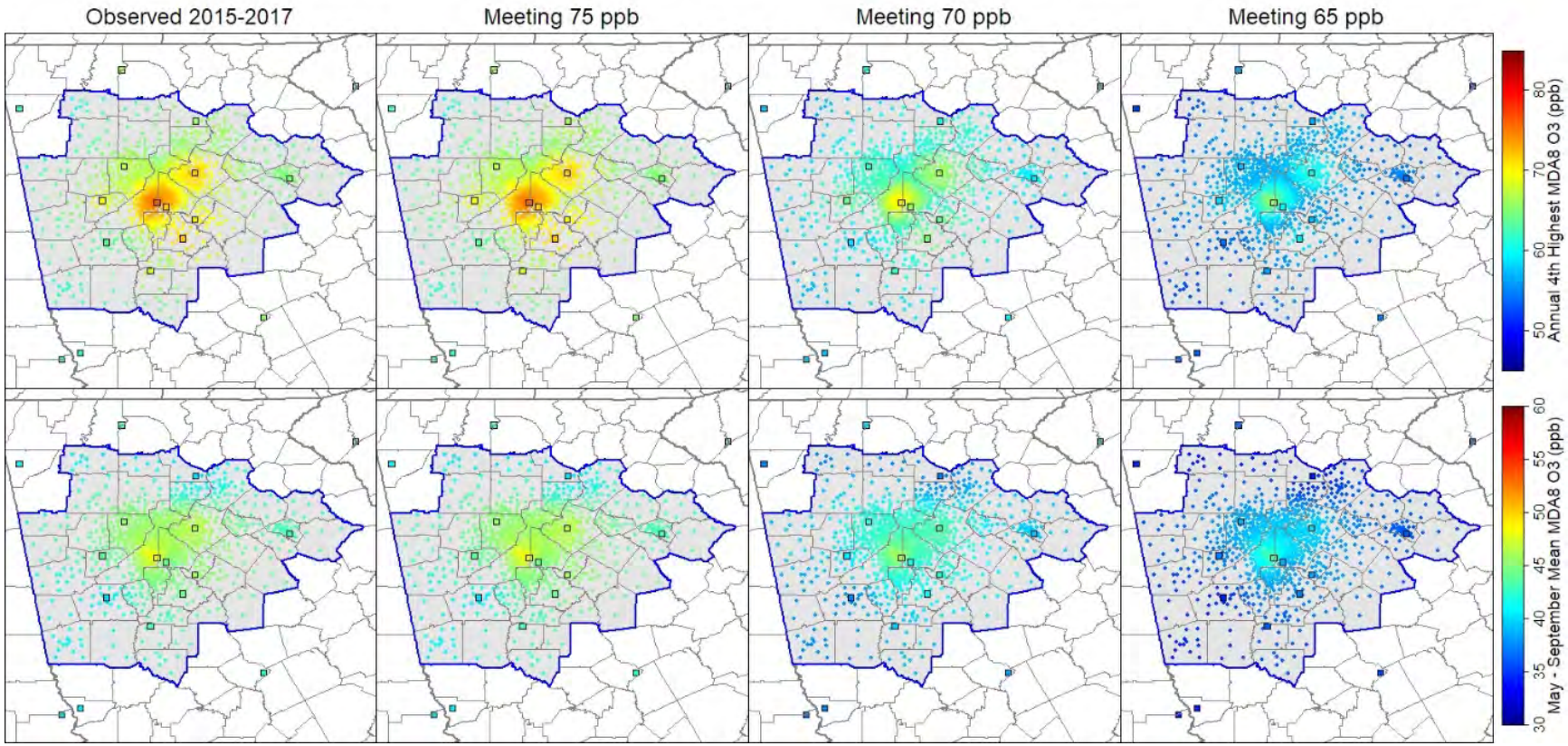


Figure 3C-91. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Atlanta study area.

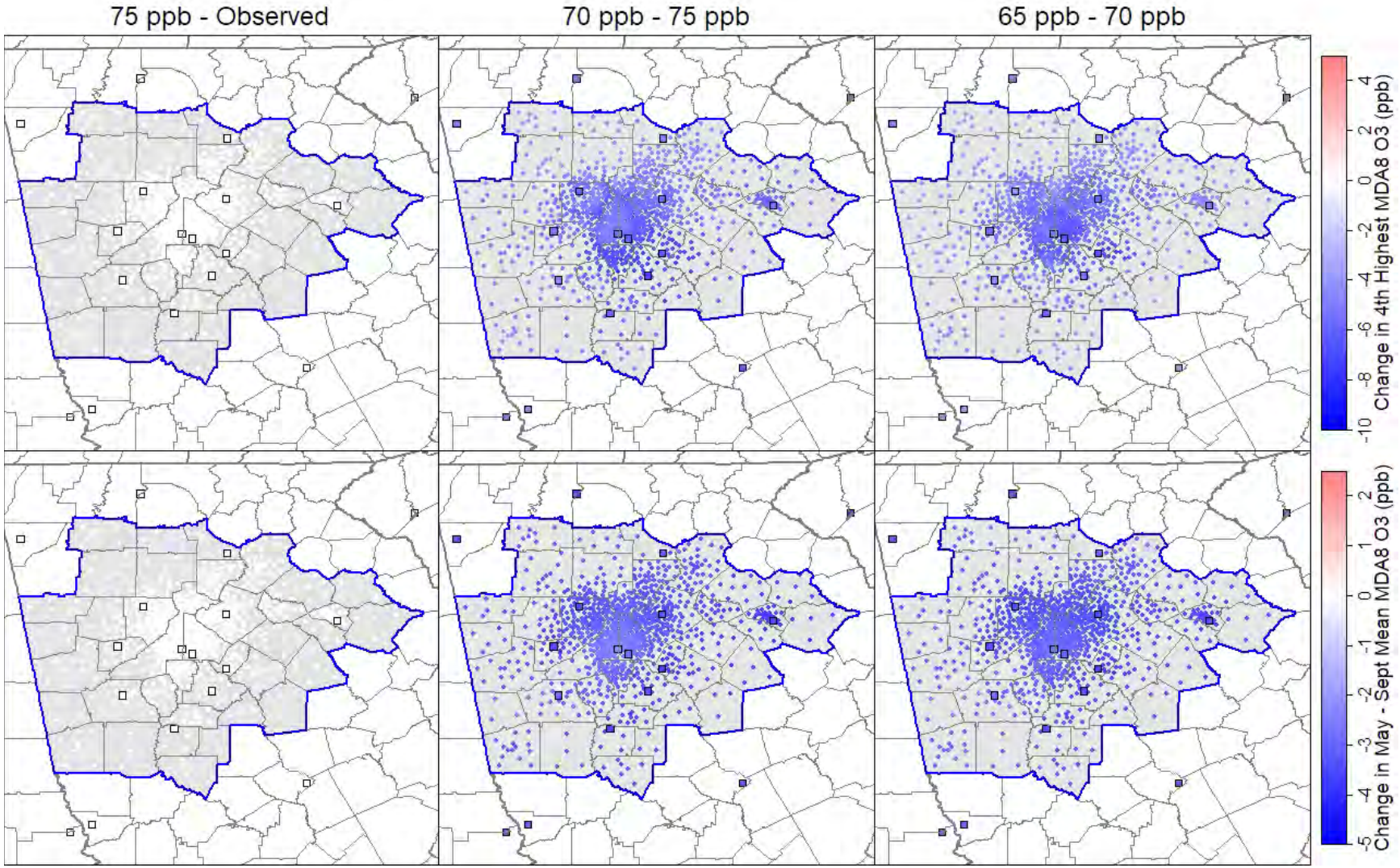


Figure 3C-92. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Atlanta study area.

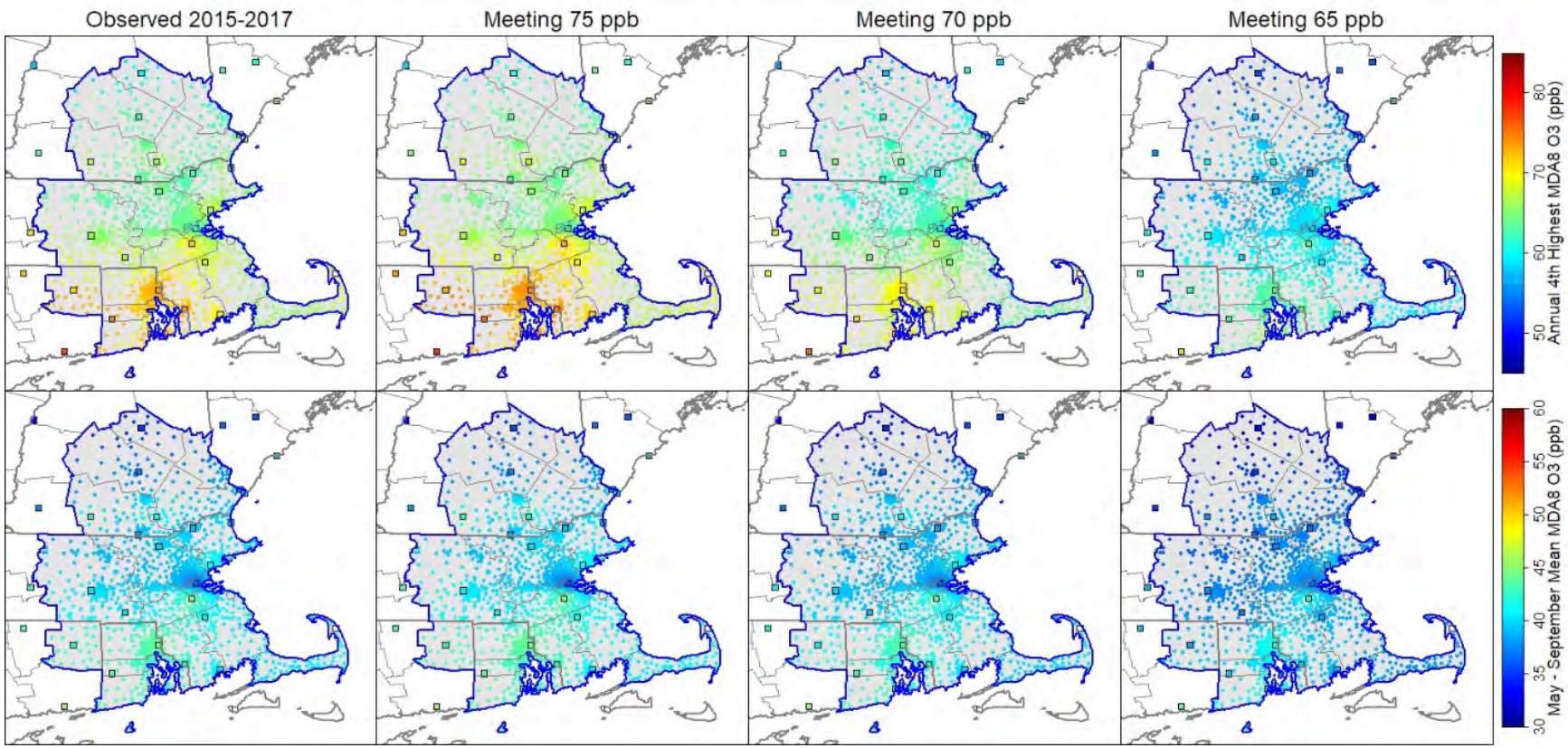


Figure 3C-93. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Boston study area.

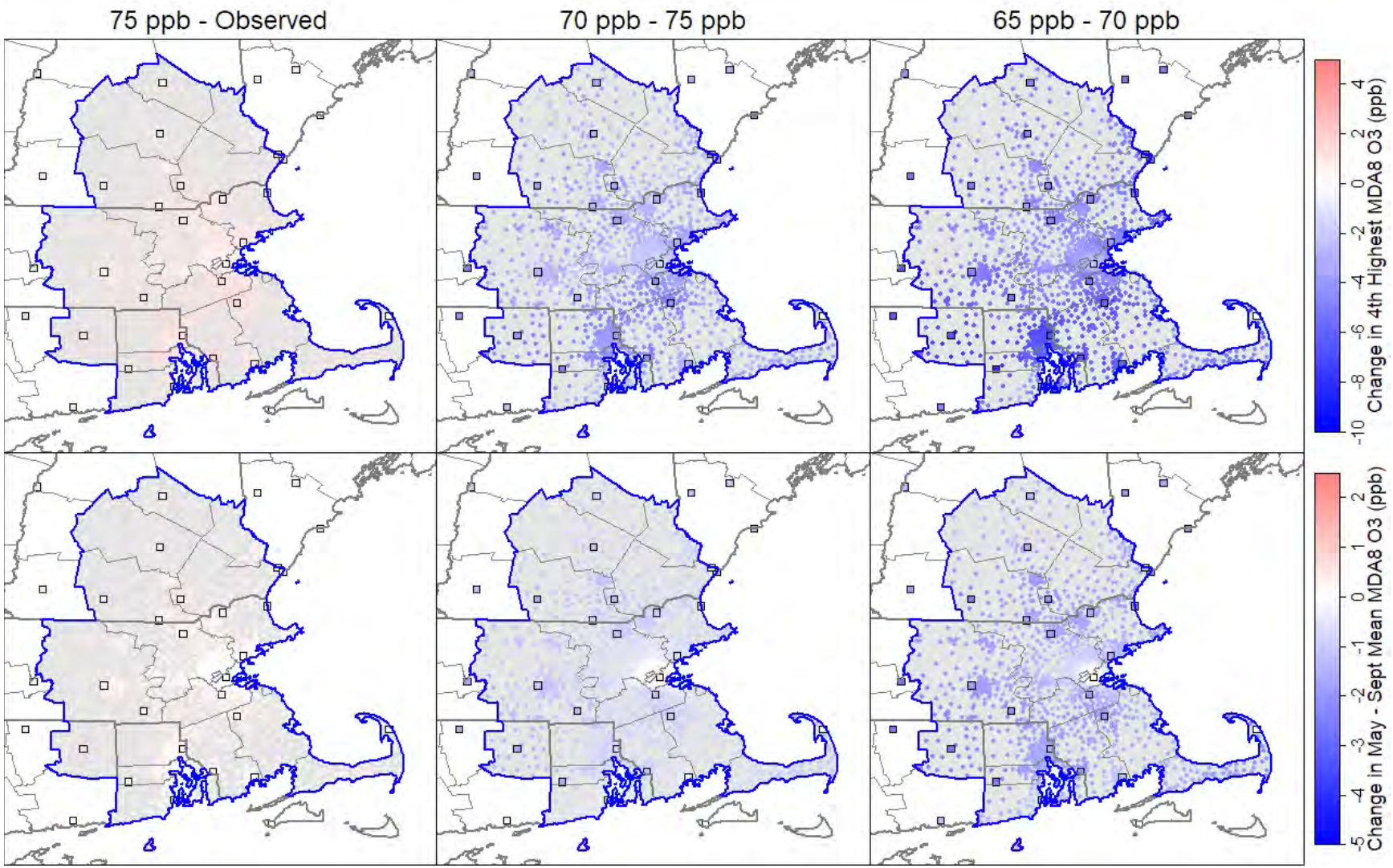


Figure 3C-94. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Boston study area.

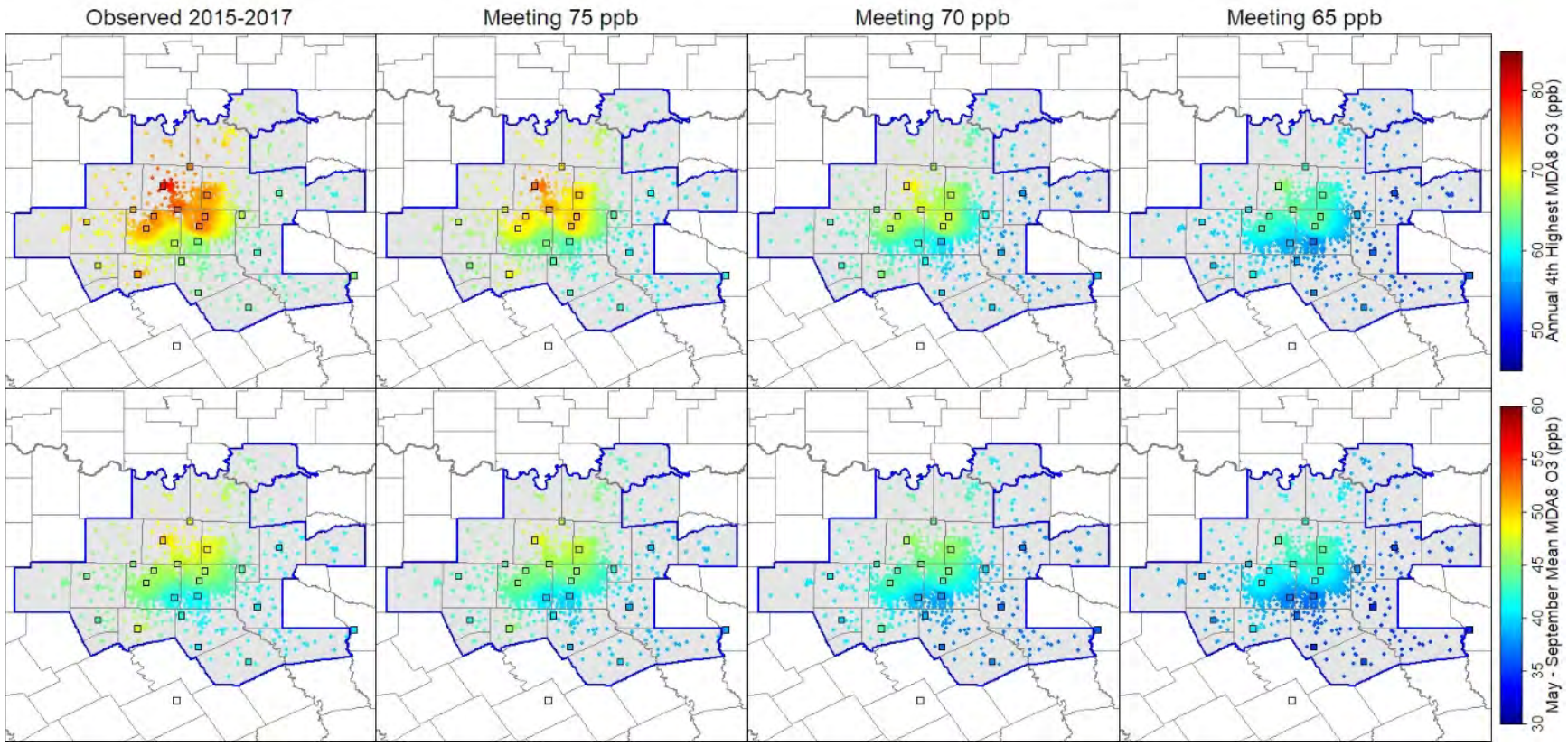


Figure 3C-95. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Dallas study area.

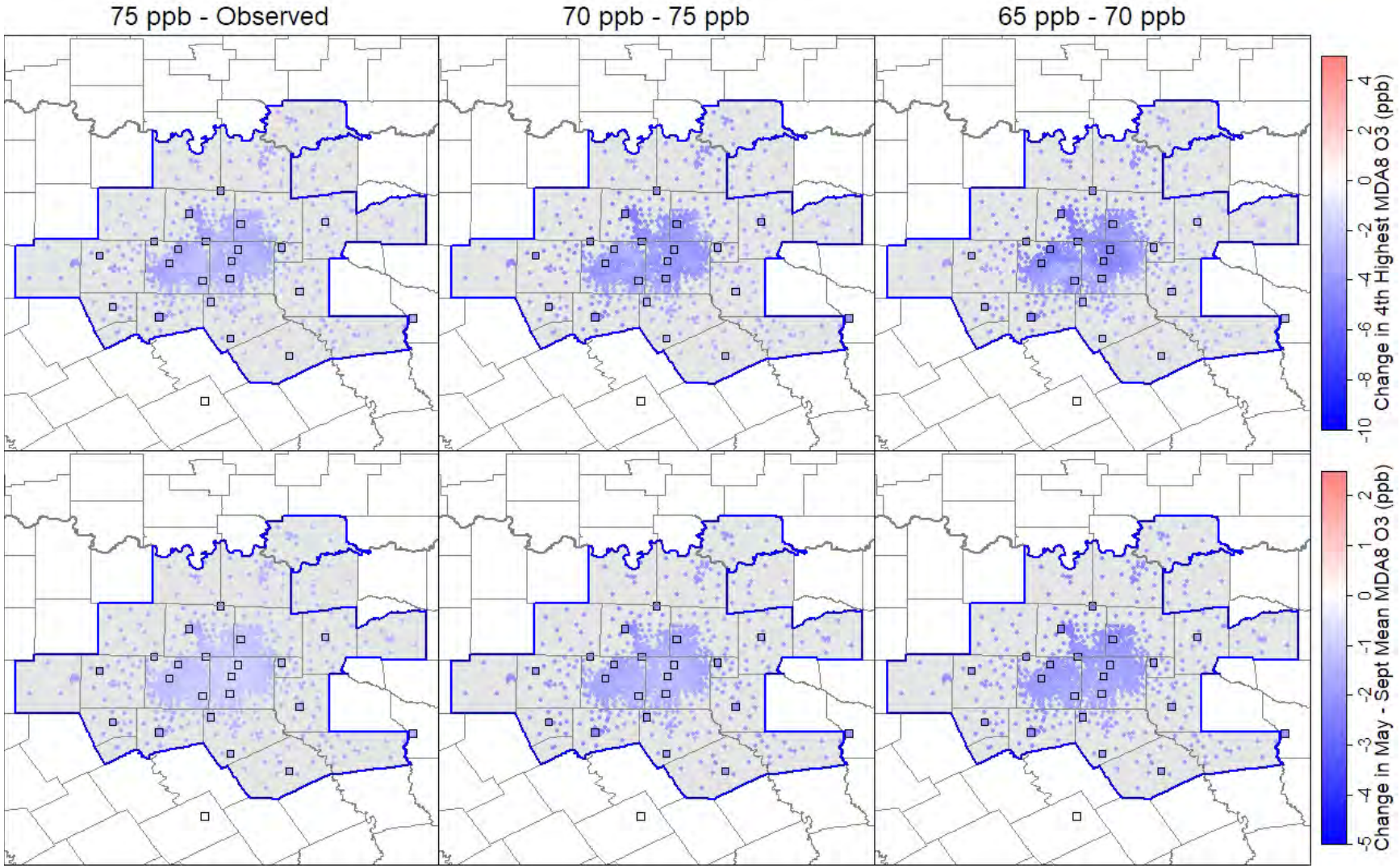


Figure 3C-96. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Dallas study area.

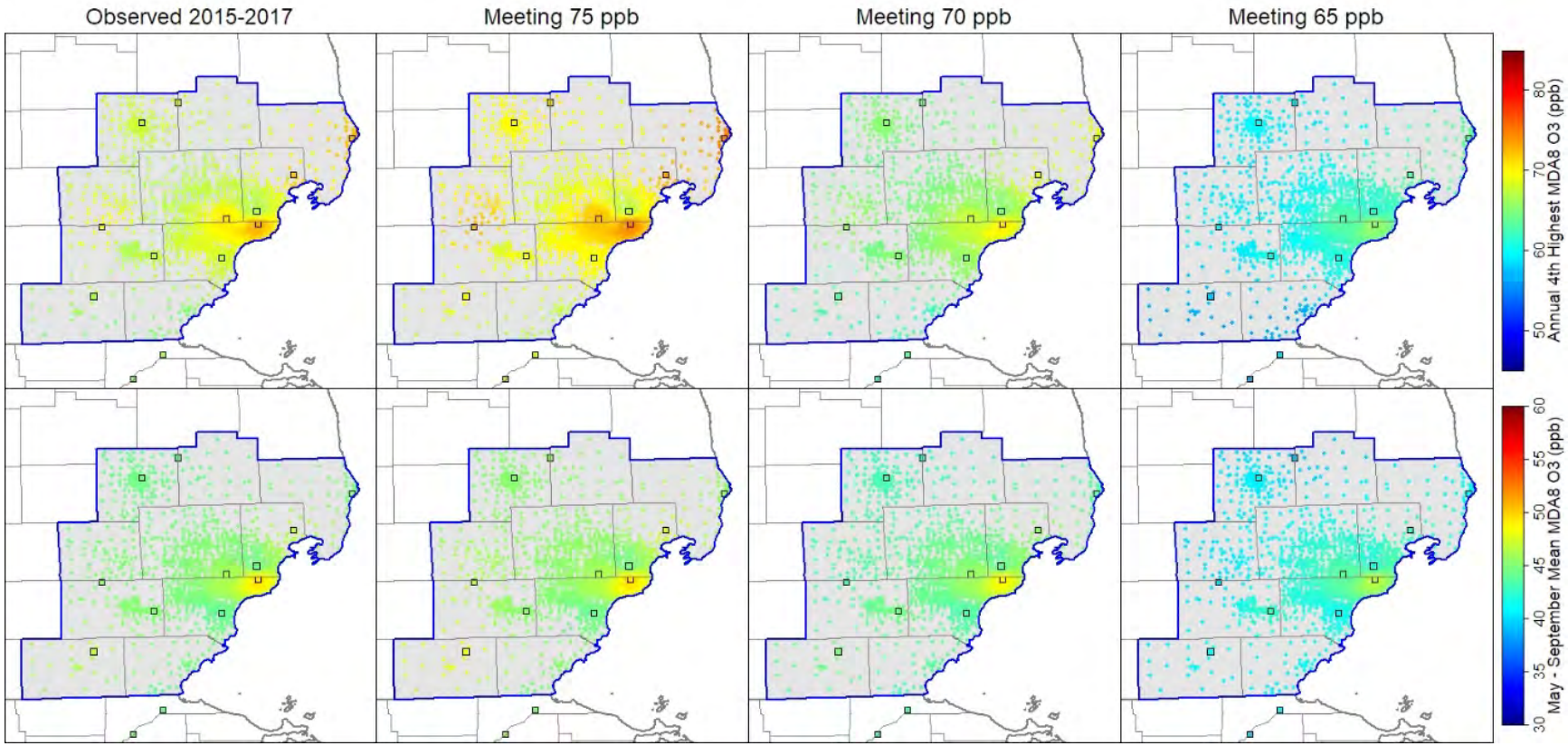


Figure 3C-97. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Detroit study area.

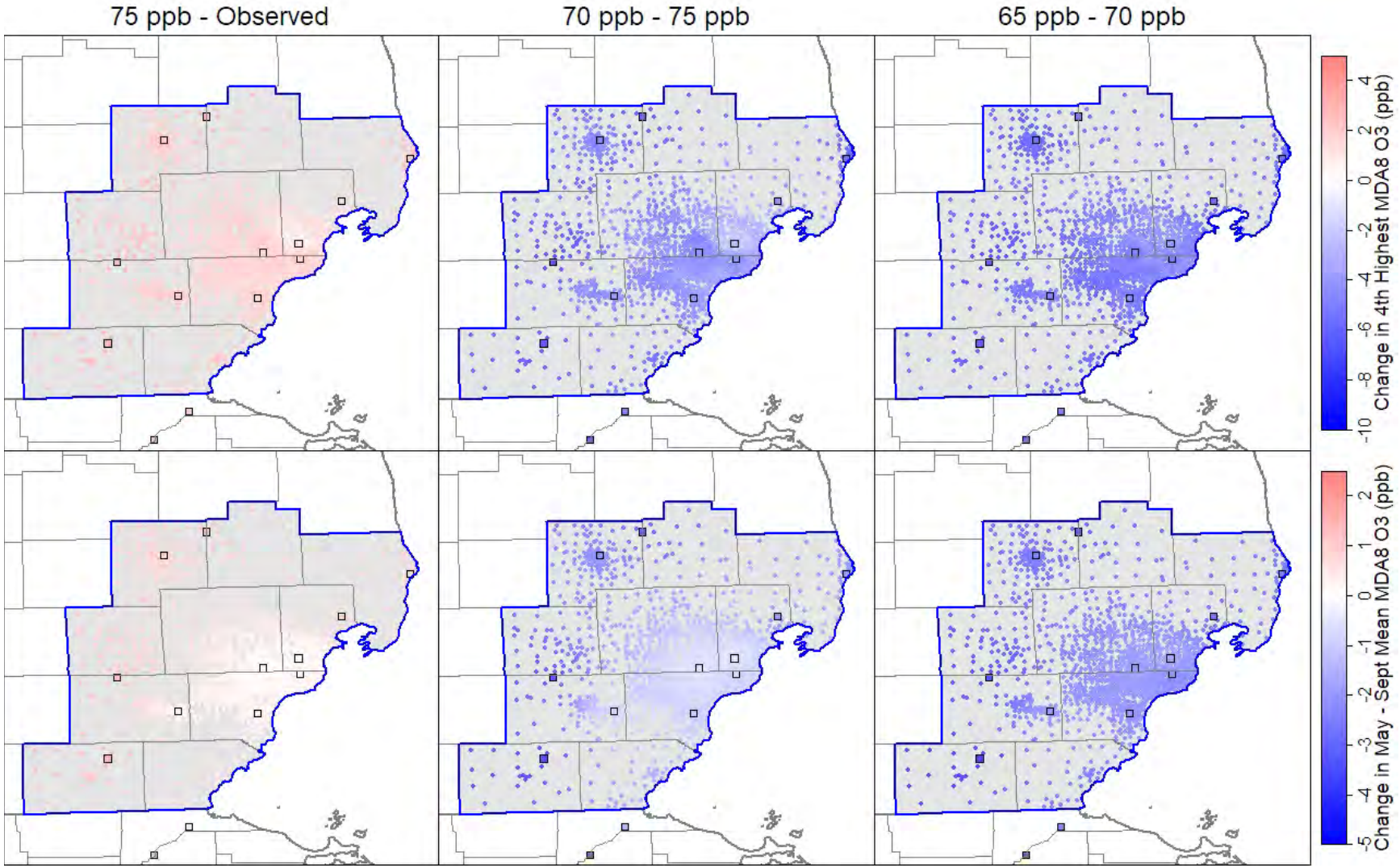


Figure 3C-98. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Detroit study area.

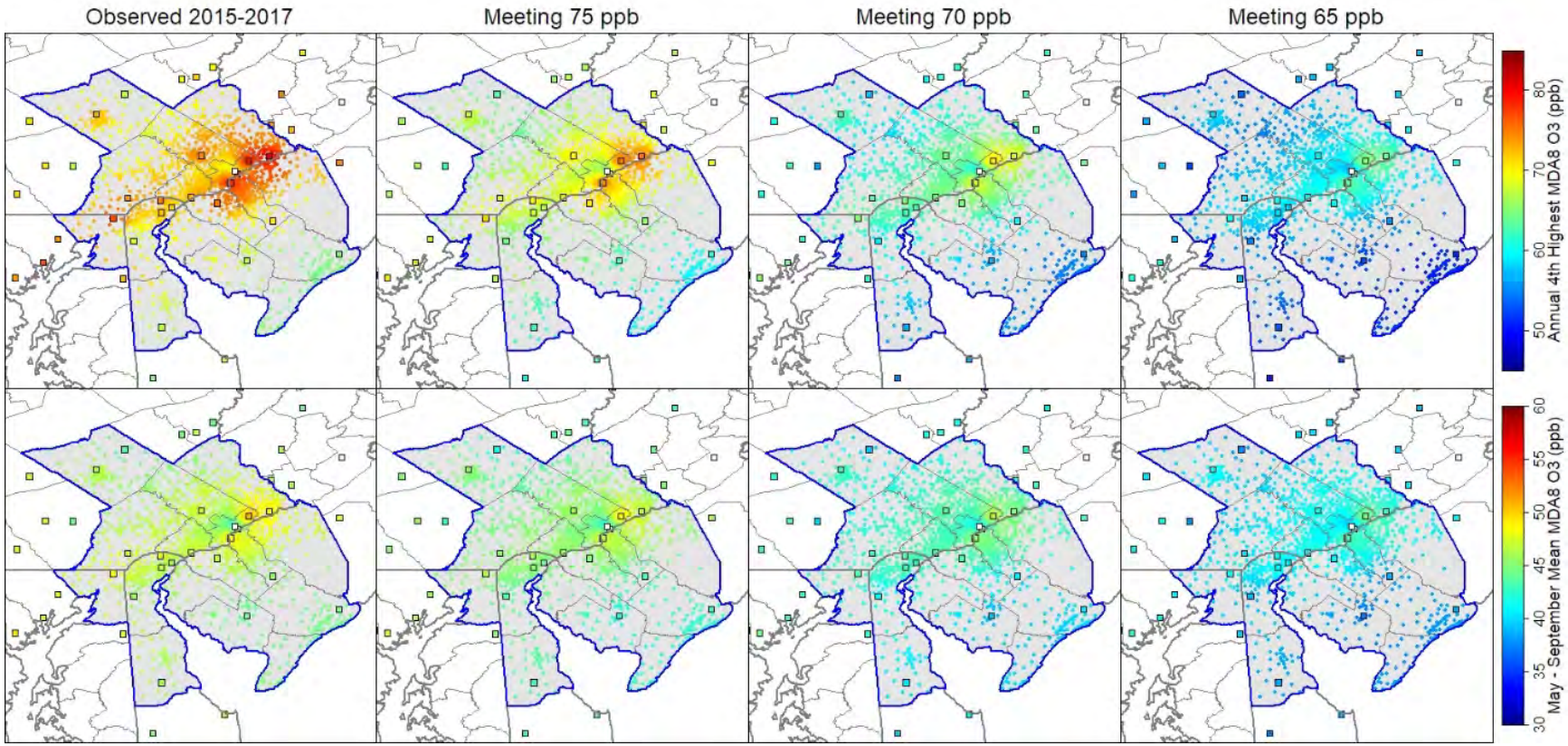


Figure 3C-99. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Philadelphia study area.

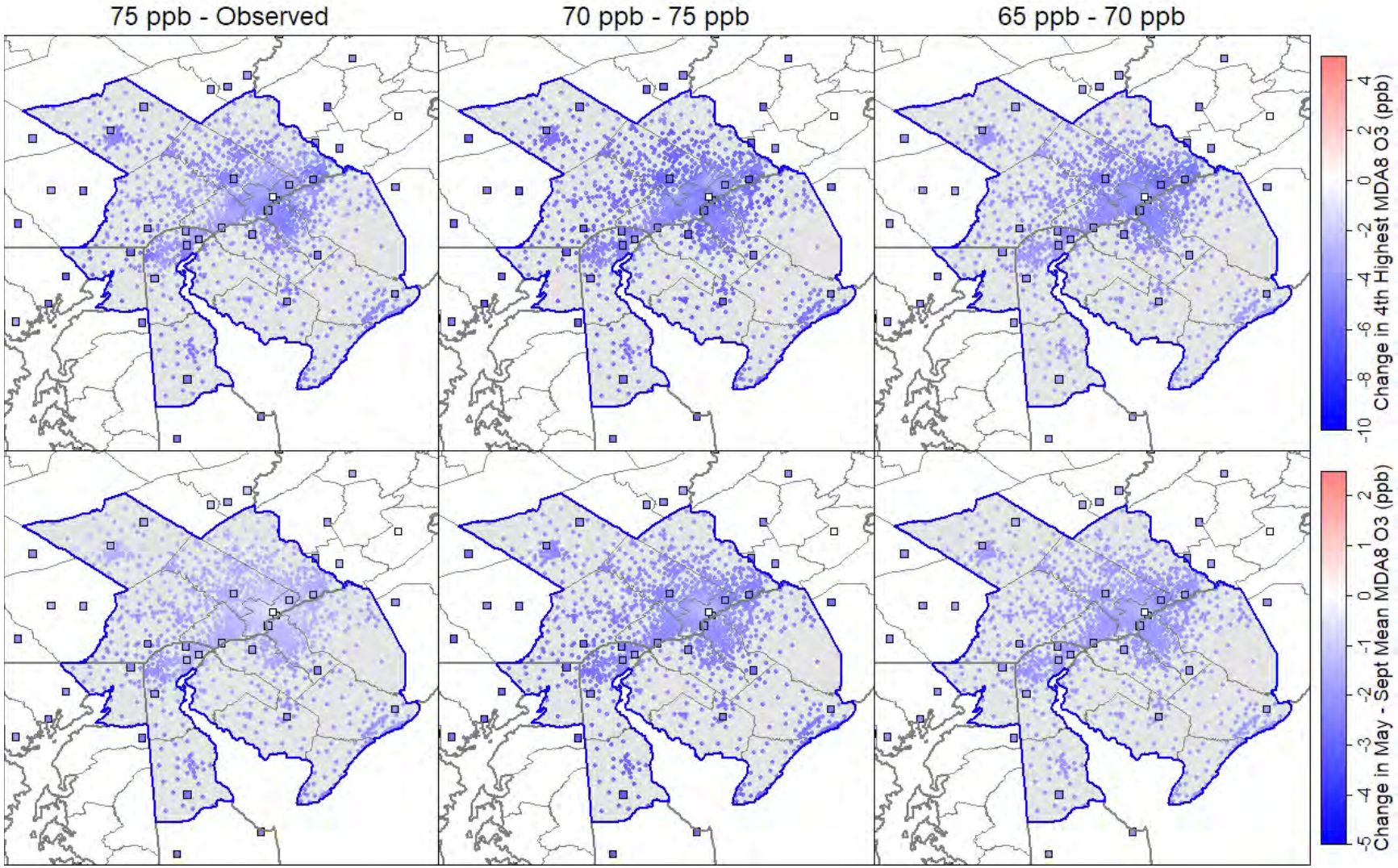


Figure 3C-100. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Philadelphia study area.

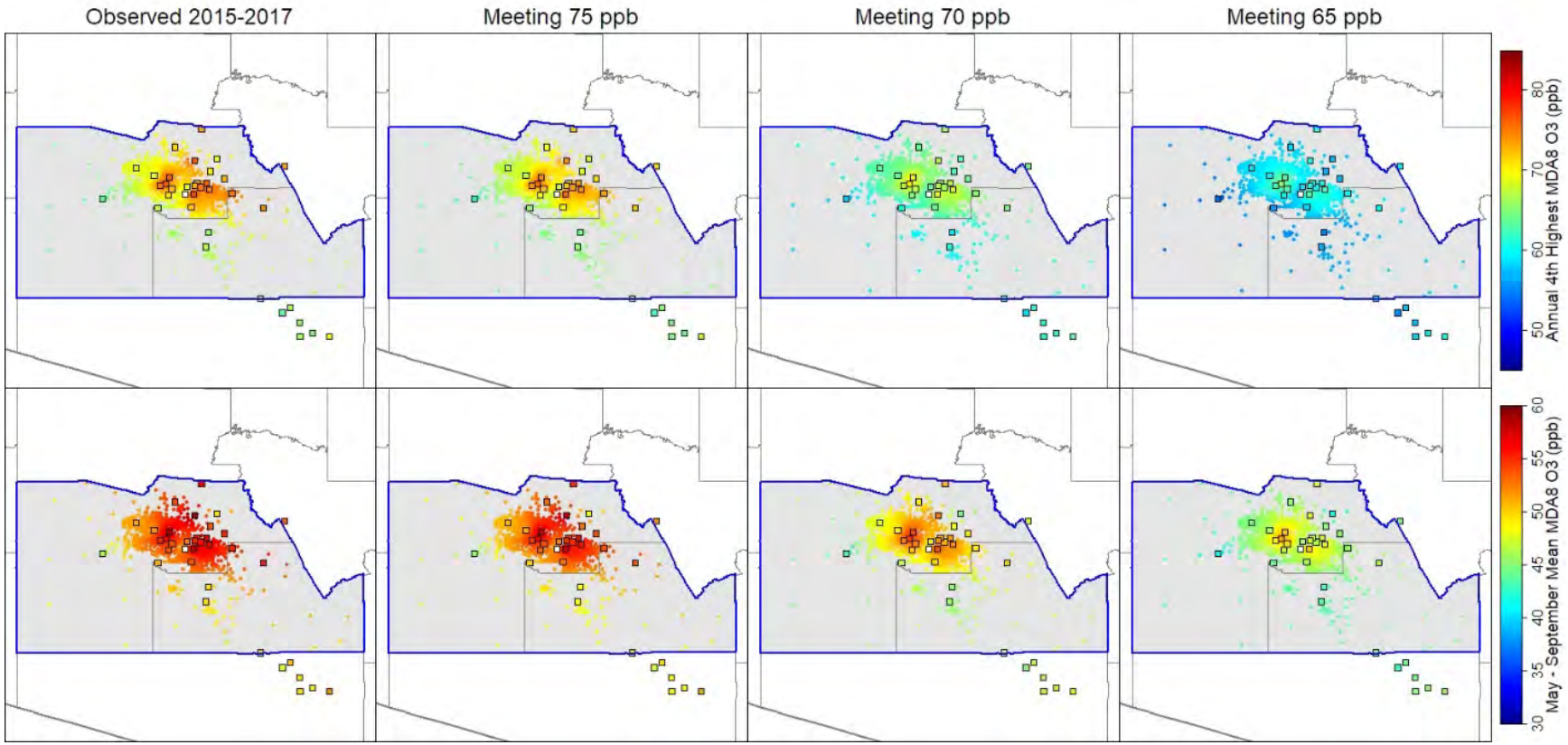


Figure 3C-101. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Phoenix study area.

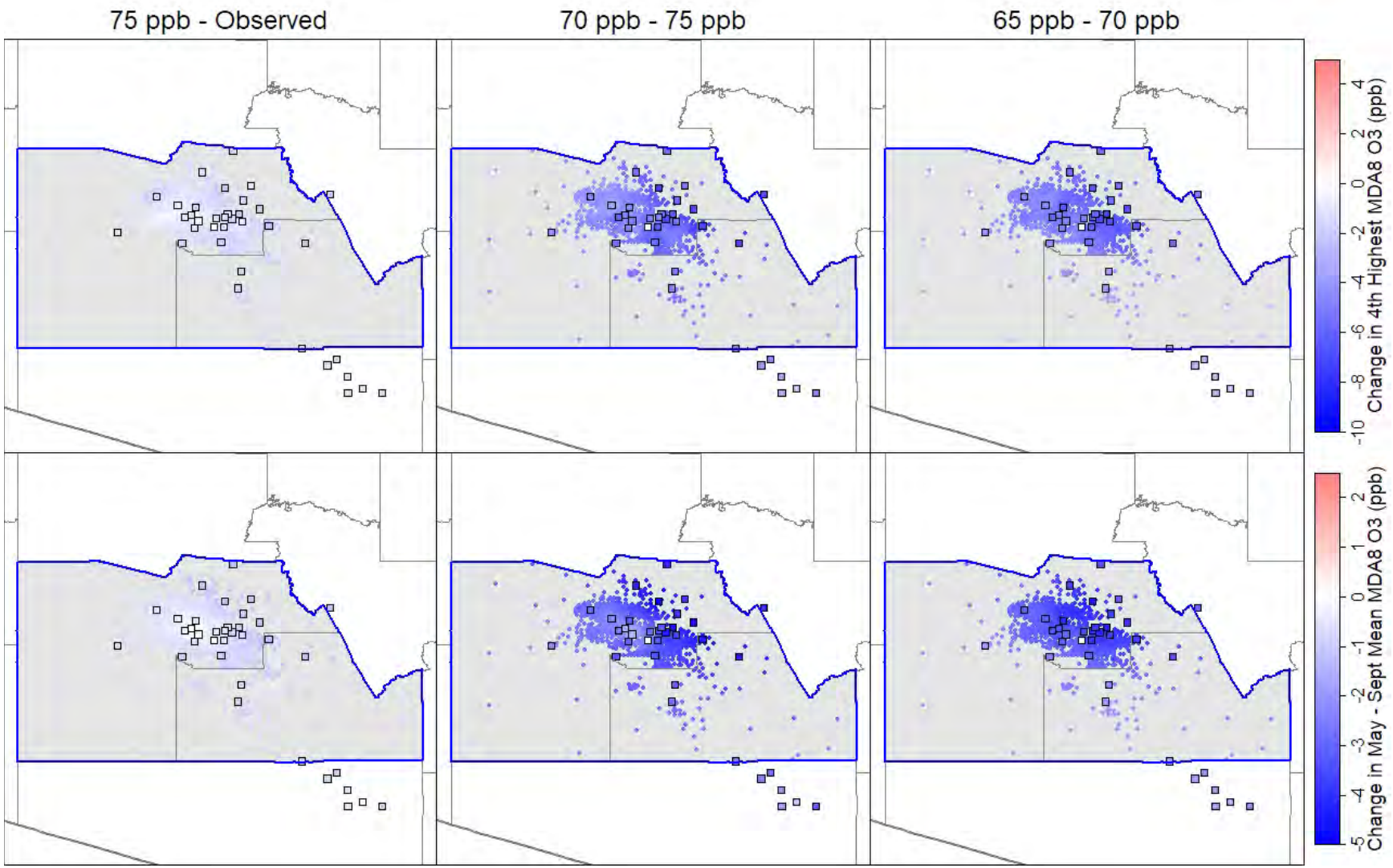


Figure 3C-102. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Phoenix study area.

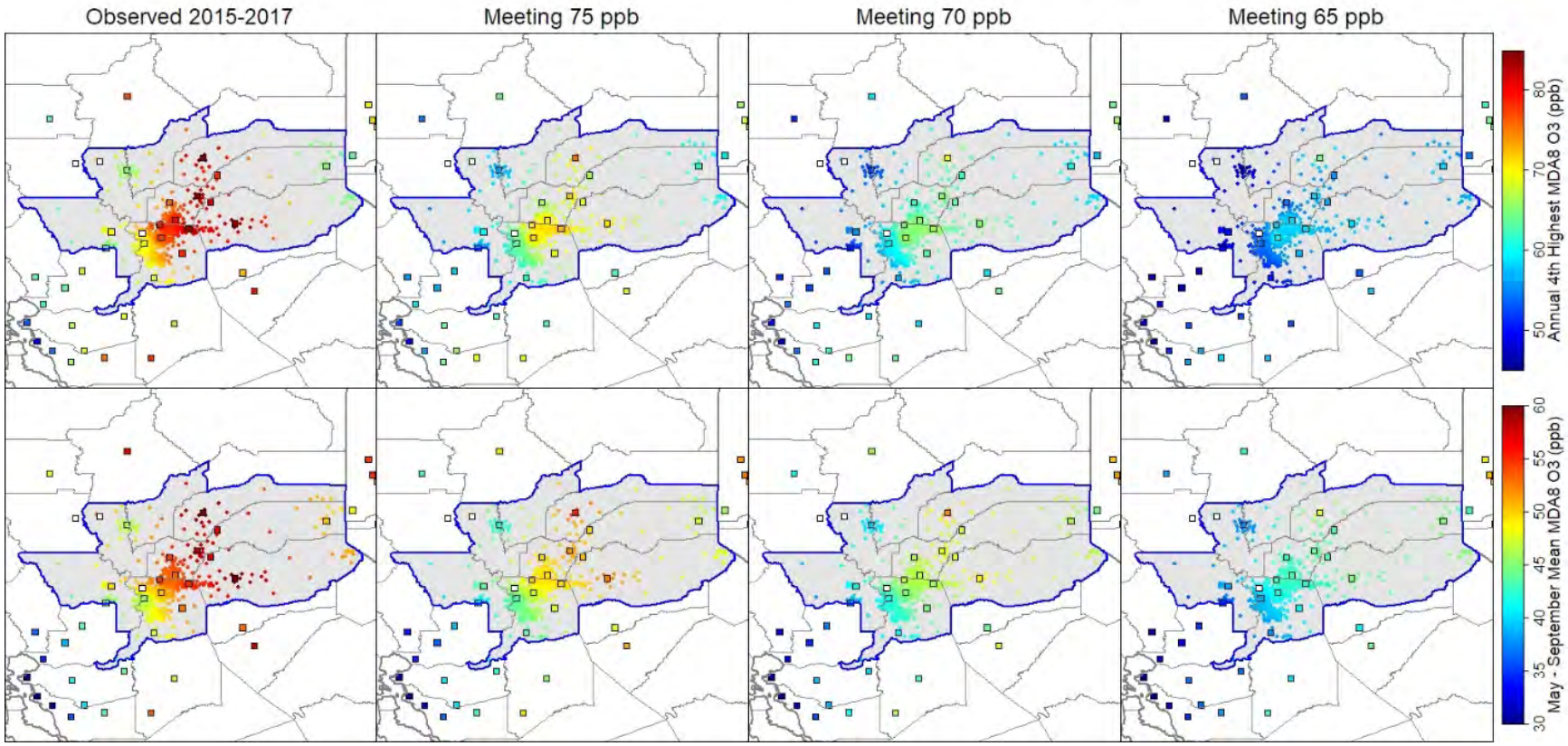


Figure 3C-103. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Sacramento study area.

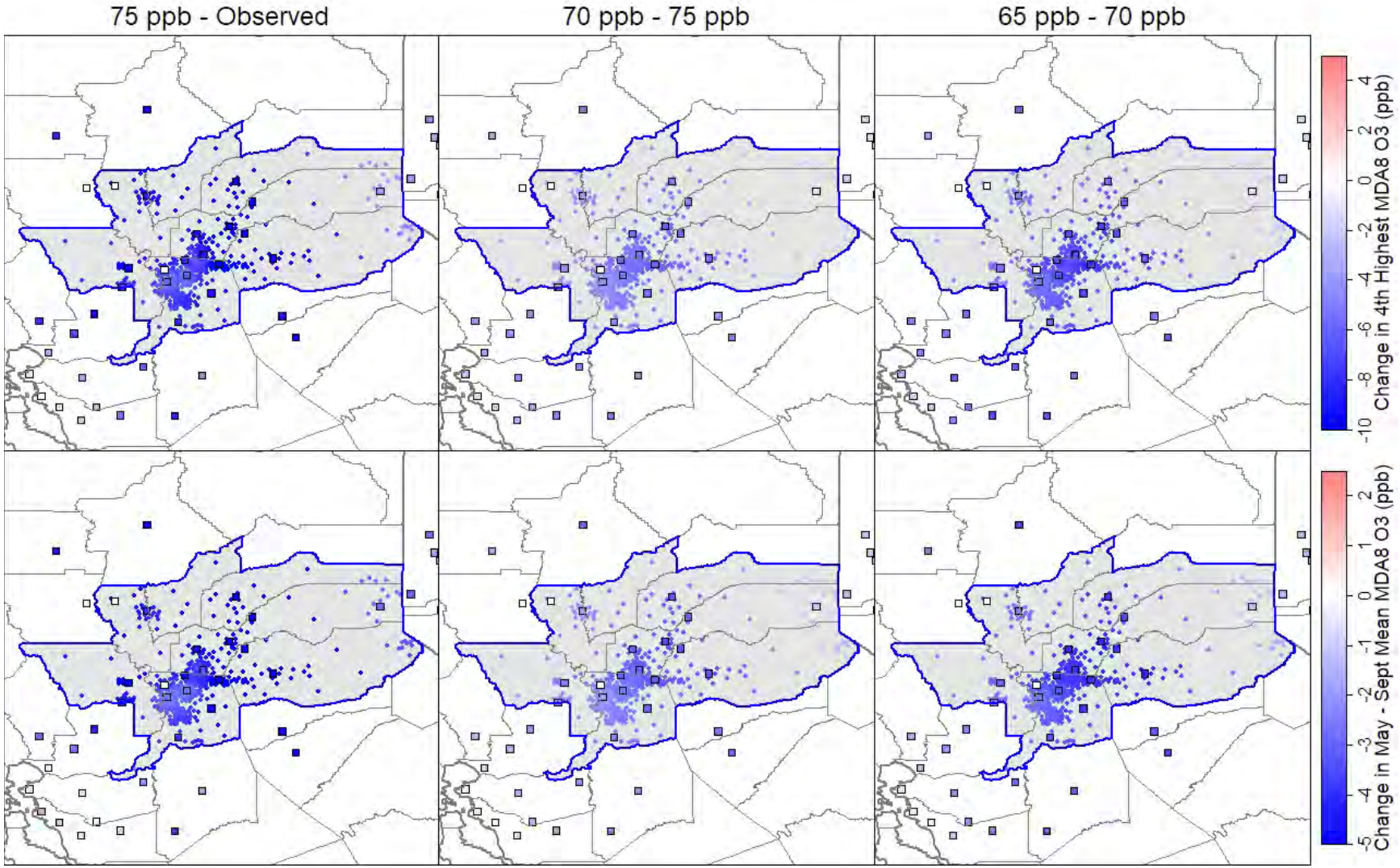


Figure 3C-104. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the Sacramento study area.

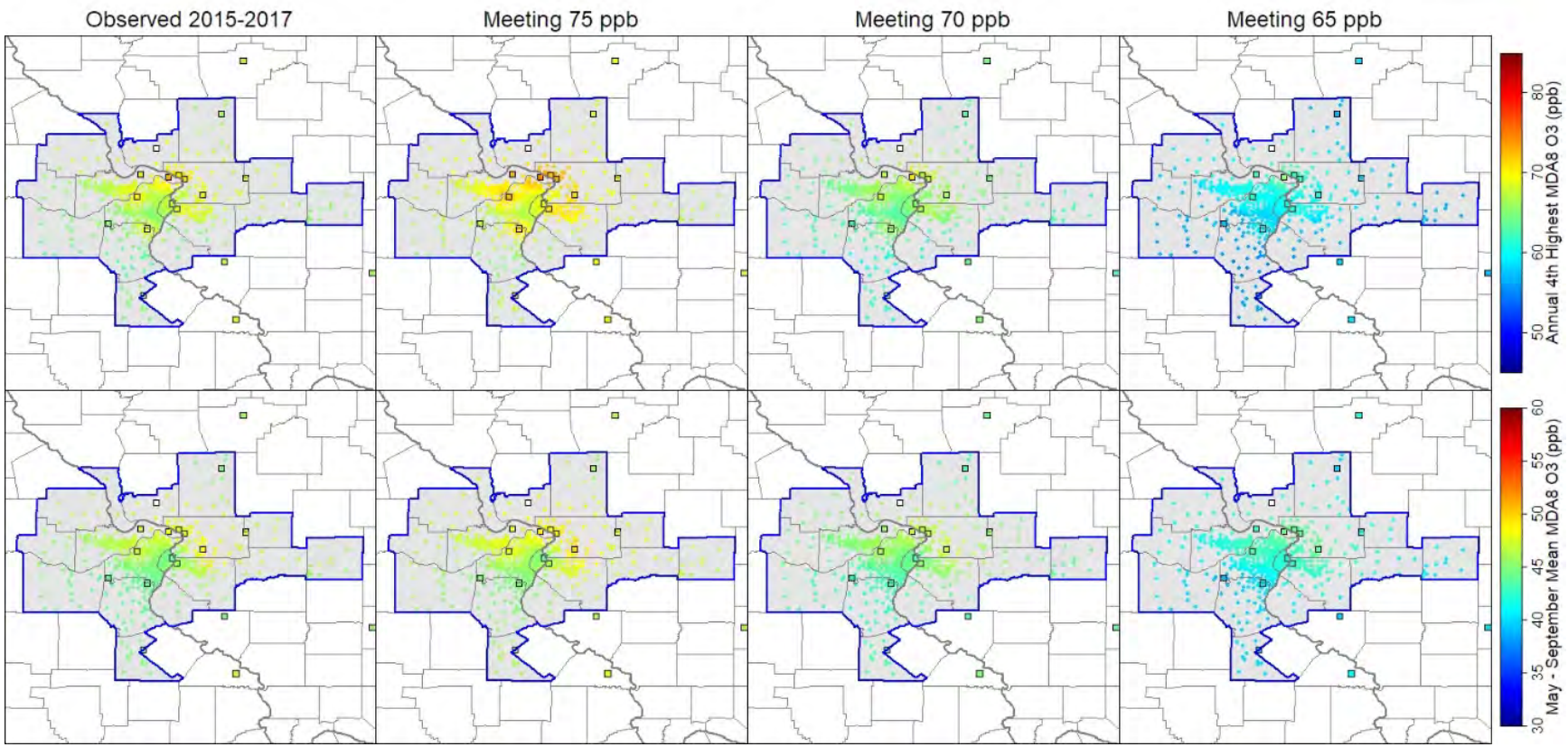


Figure 3C-105. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the St. Louis study area.

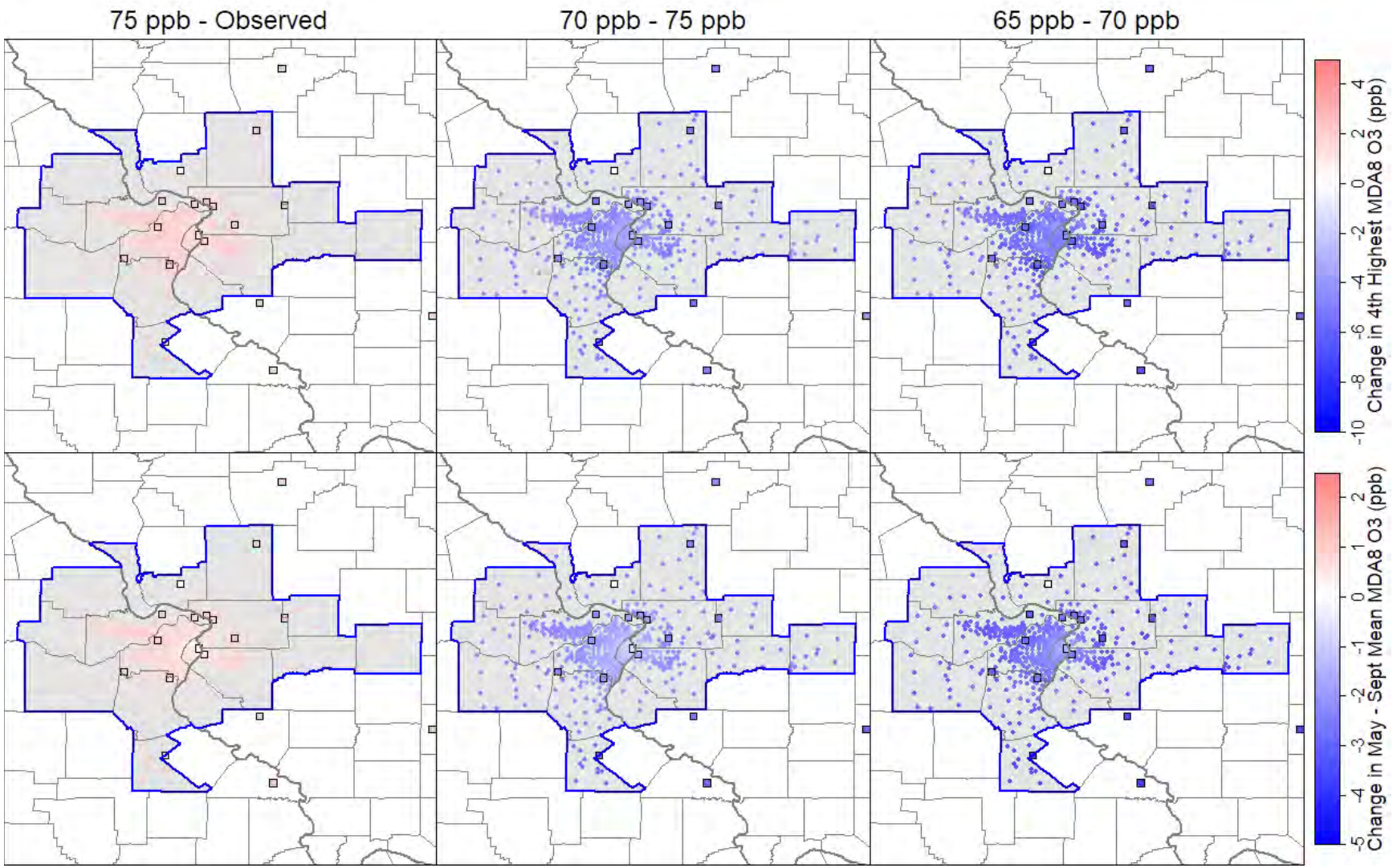


Figure 3C-106. Changes in annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 based on HDDM adjustments in the St. Louis study area.

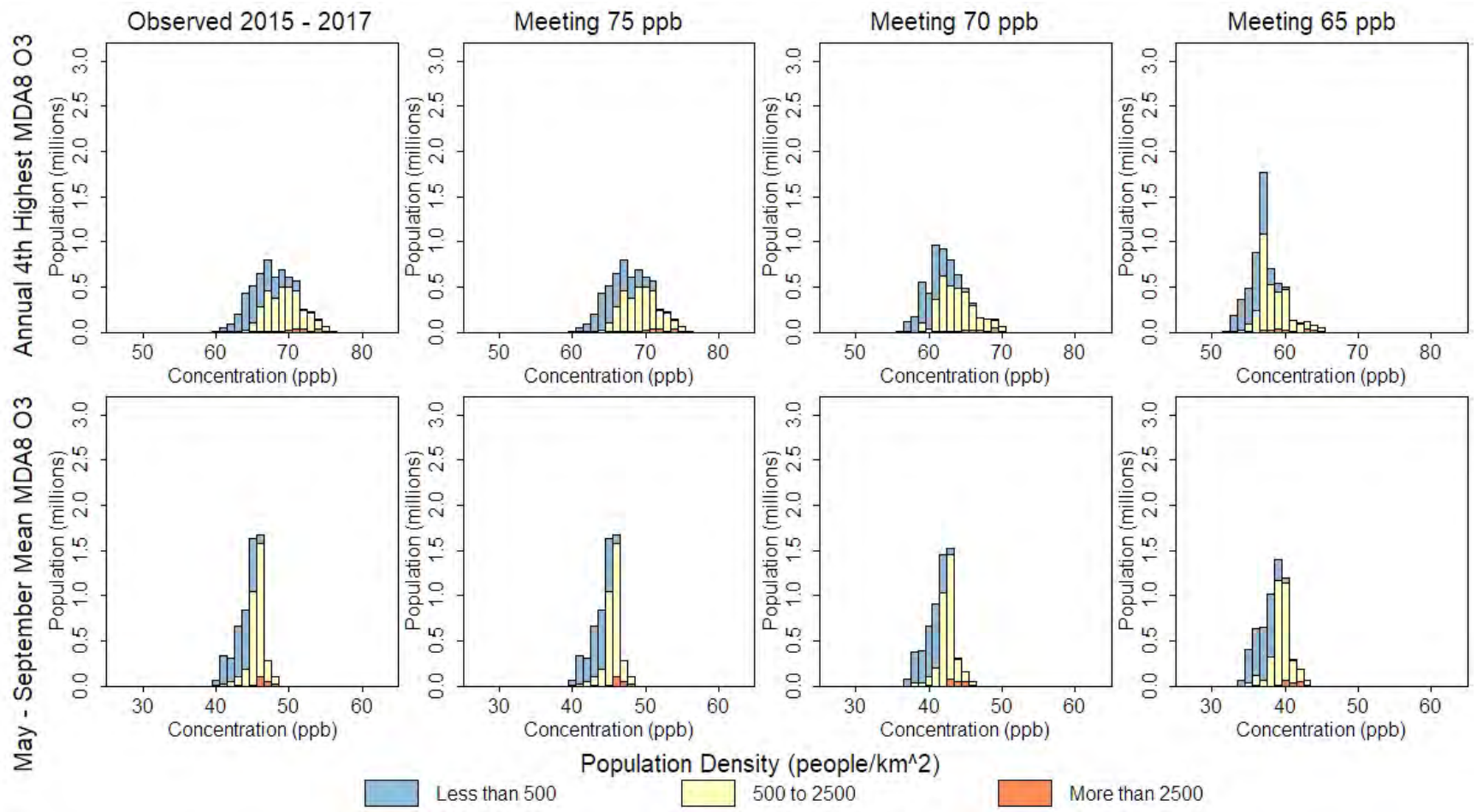


Figure 3C-107. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the Atlanta study area.

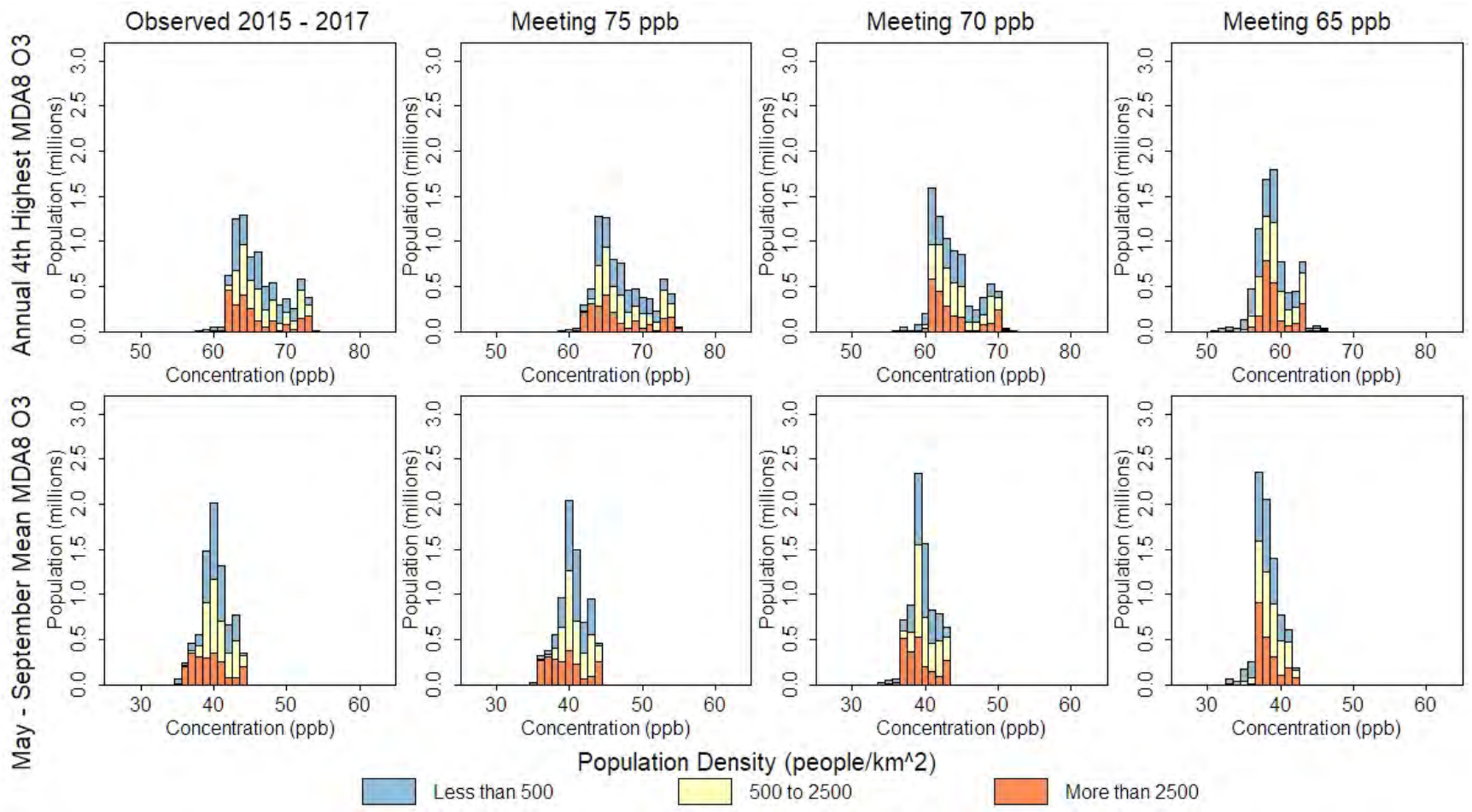


Figure 3C-108. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the Boston study area.

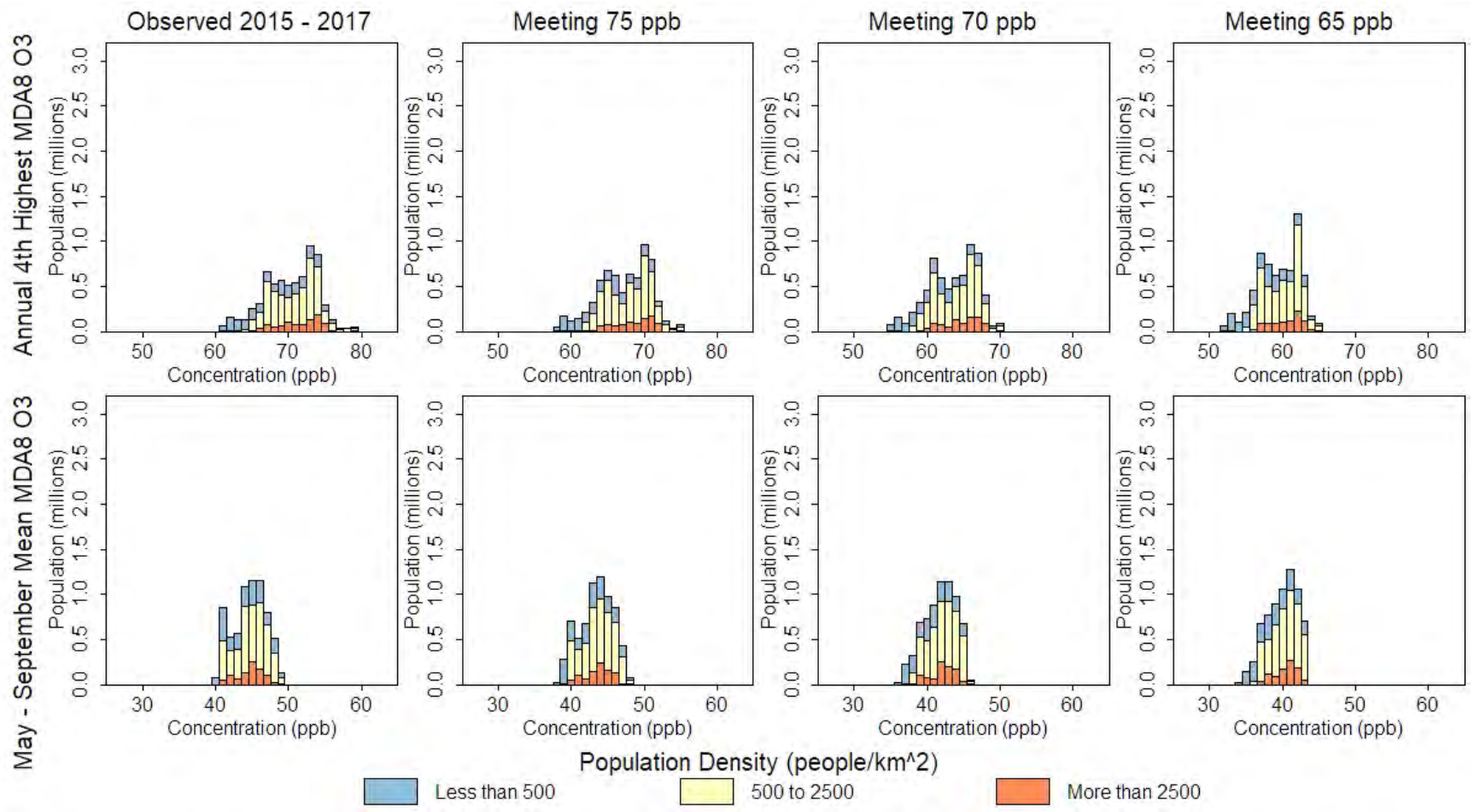


Figure 3C-109. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the Dallas study area.

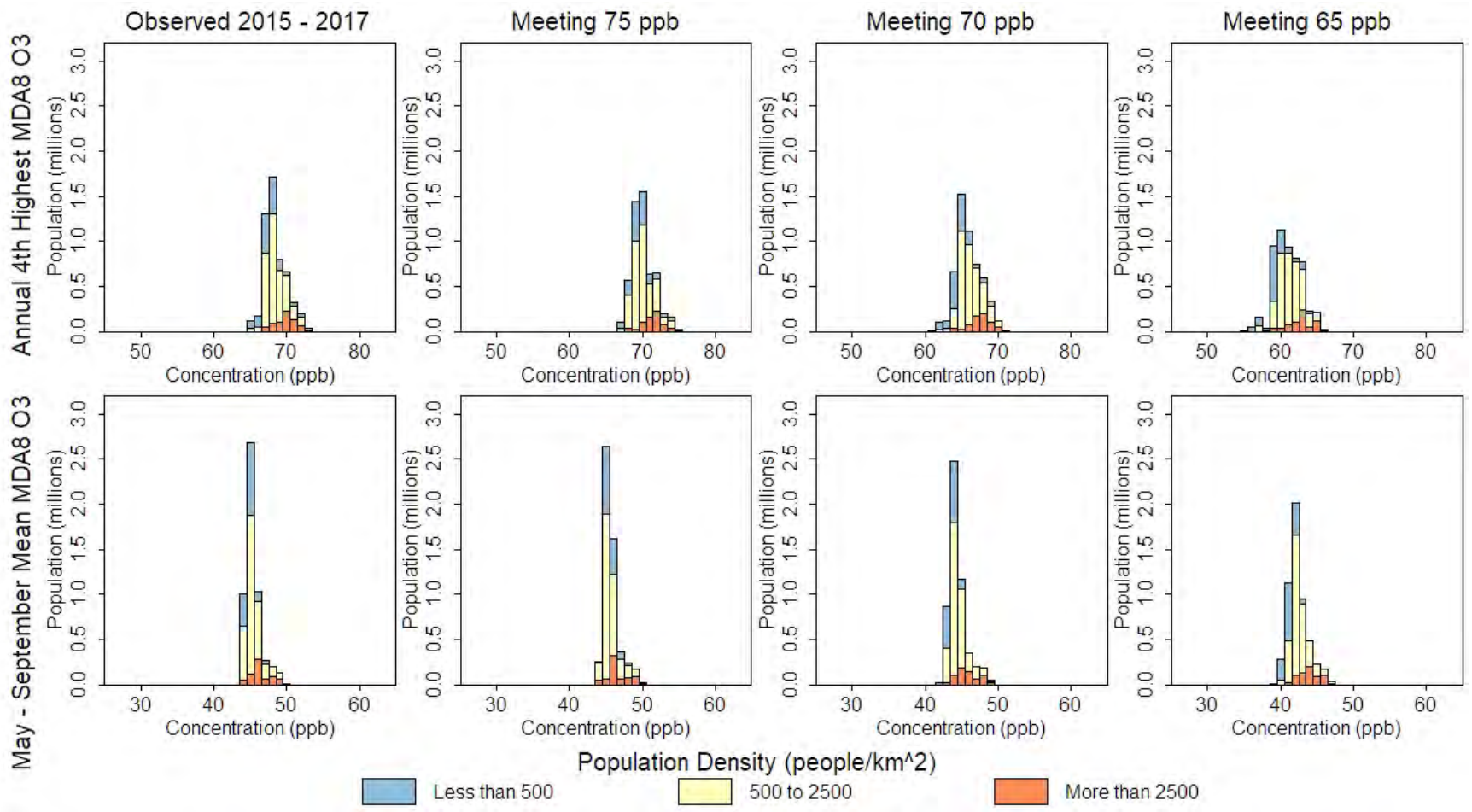


Figure 3C-110. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the Detroit study area.

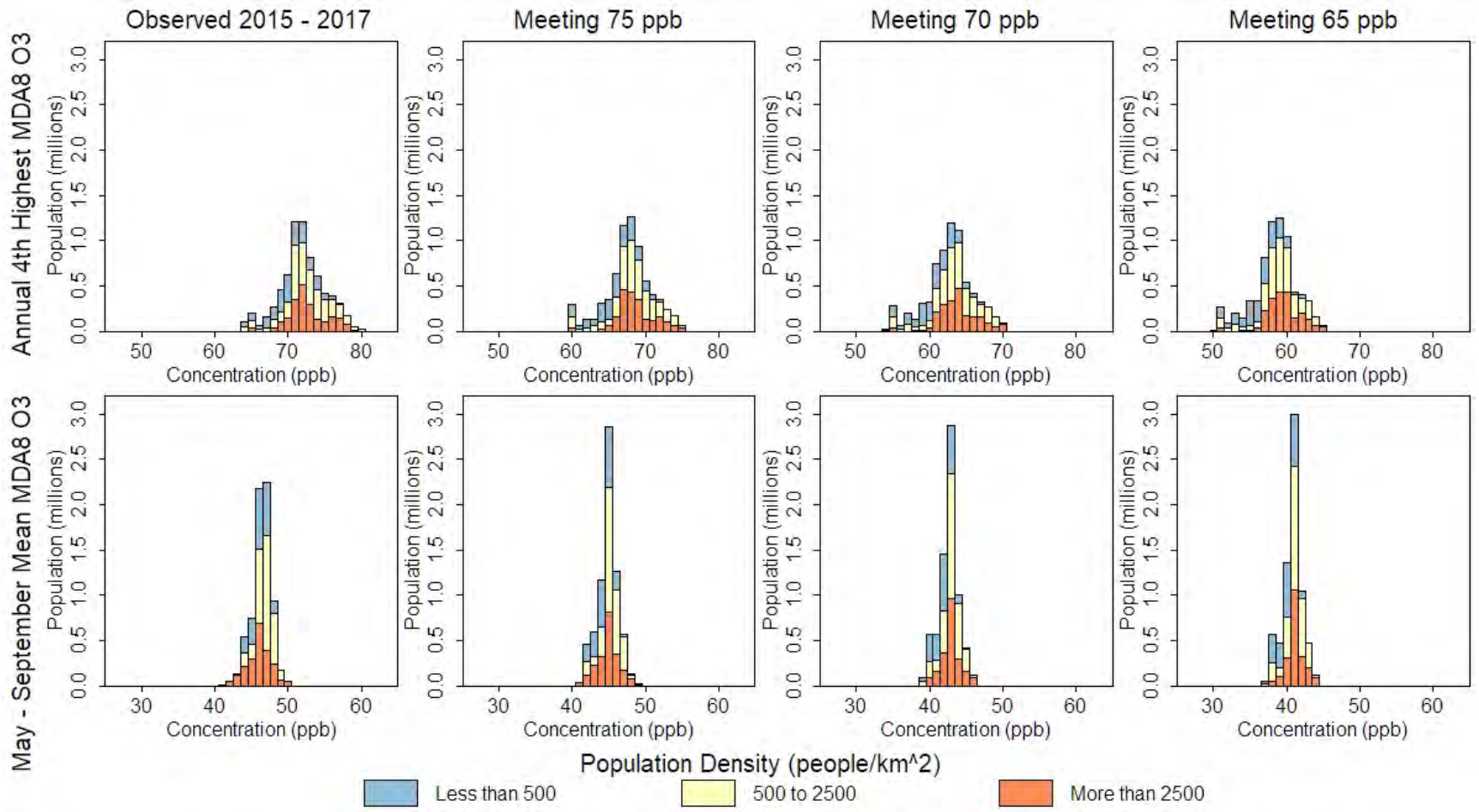


Figure 3C-111. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the Philadelphia study area.

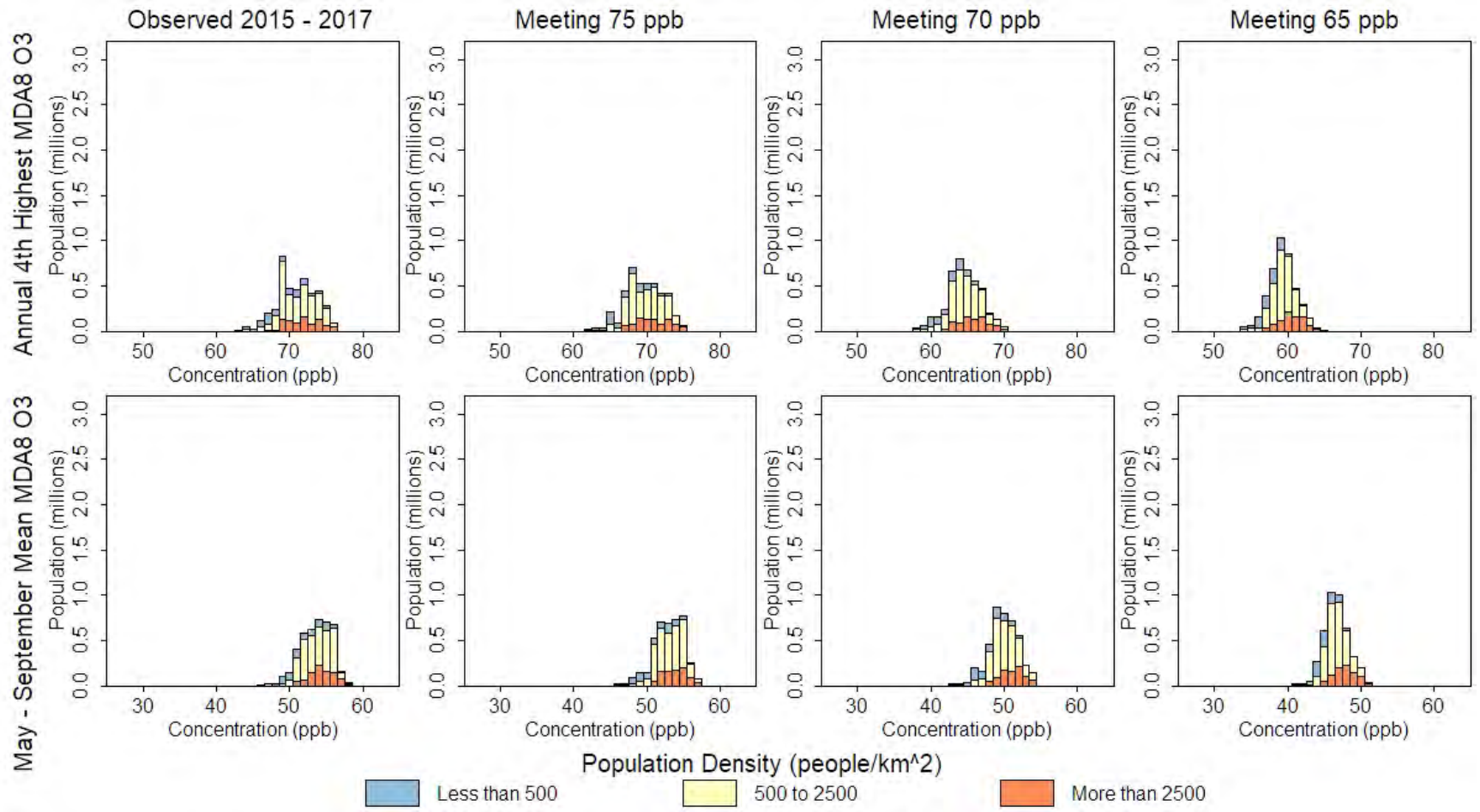


Figure 3C-112. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the Phoenix study area.

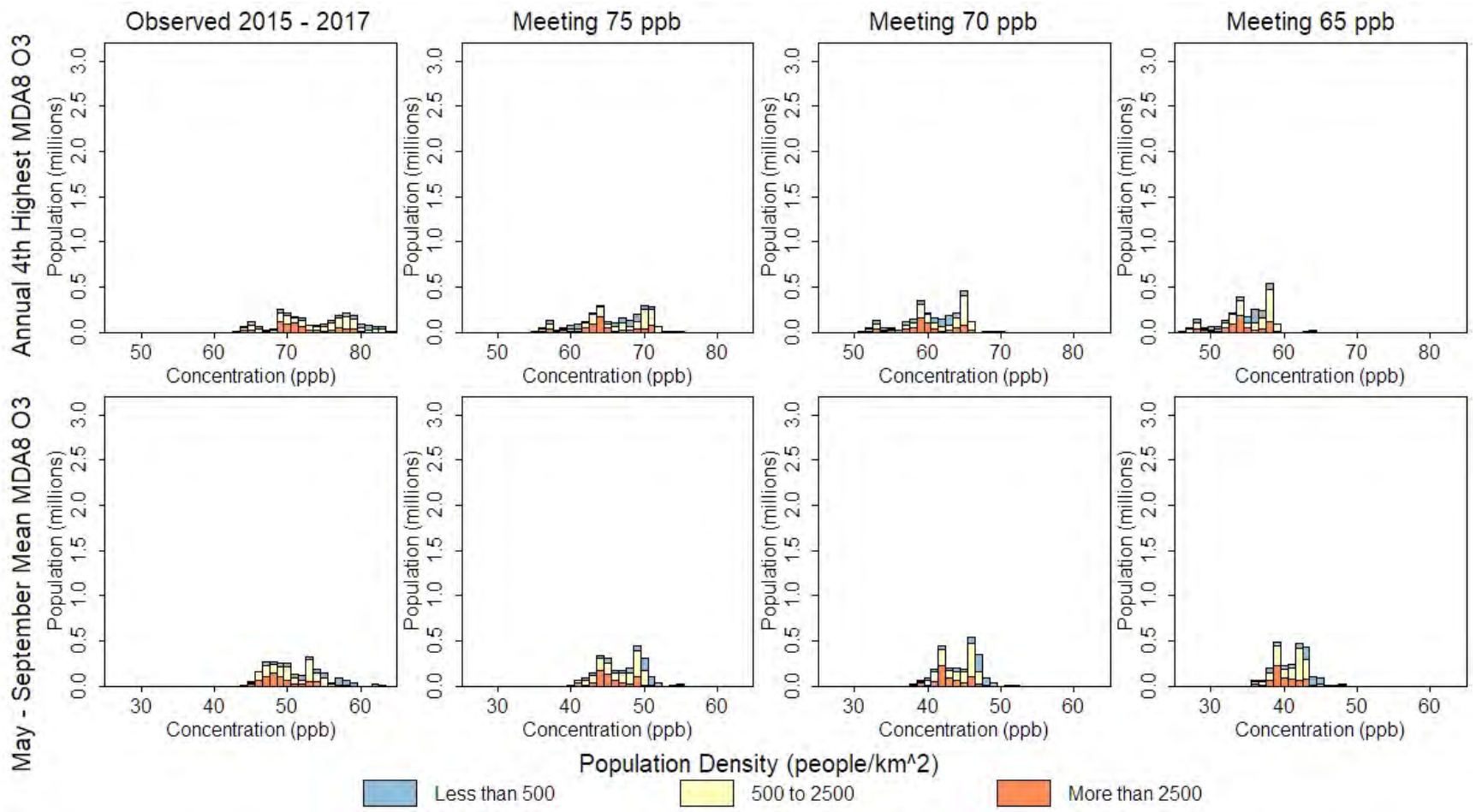


Figure 3C-113. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the Sacramento study area.

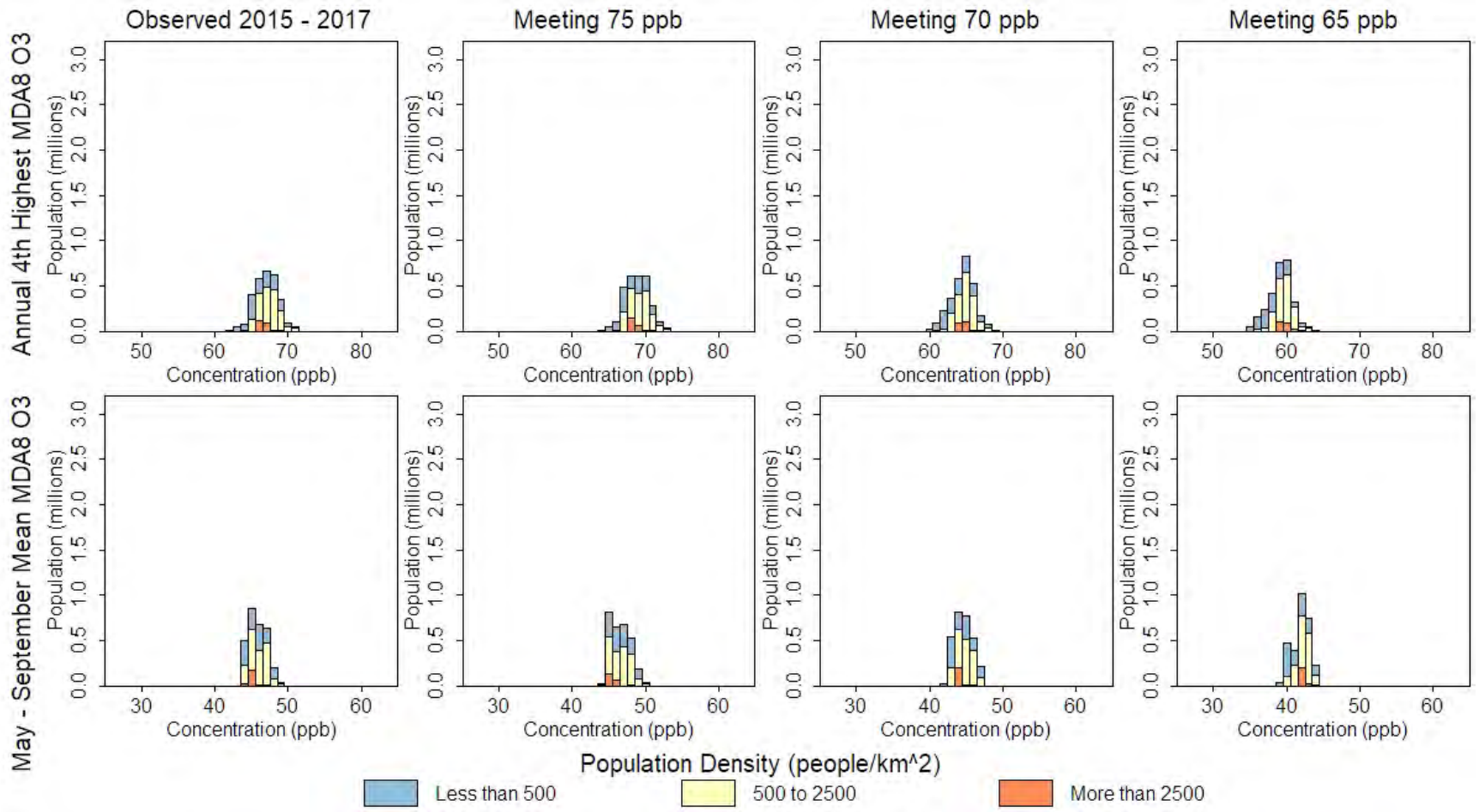


Figure 3C-114. Annual 4th highest MDA8 O_3 and May-September mean MDA8 O_3 by population based on HDDM adjustments in the St. Louis study area.

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APPENDIX 3D

EXPOSURE AND RISK ANALYSIS FOR THE OZONE NAAQS REVIEW

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3D.1 INTRODUCTION

This appendix to the O_3 Policy Assessment (PA) summarizes the quantitative exposure and risk analysis performed for the current O_3 NAAQS review. The analysis builds upon the methodology and lessons learned from the human exposure and risk analyses conducted in the prior O_3 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 O_3 Integrated Science Assessment (ISA; U.S. EPA, 2020), which builds on the 2013 ISA (U.S. EPA, 2013).

For the current O_3 NAAQS review, 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 O_3 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 O_3 concentrations across the study area such that the location with the highest design value[2] 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 of this PA and described in the ISA (ISA, section IS.6.l).

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

[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 O_3 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.

ambient air O_3-related lung function decrements (PA, Figure 3-3). The first risk metric is based on comparison of estimated daily maximum 7-hour (7-hr) average exposures for individuals breathing at elevated rates to concentrations of potential concern (benchmark concentrations),[5] and the second uses exposure-response (E-R) information for study subjects experiencing FEV_1 decrements (specifically O_3-related decrement of 10% or more) to estimate the portion of the simulated at-risk population expected to experience one or more days with an O_3-related FEV_1 decrement of at least 10%, 15% and 20%.

A description of the exposure and risk modeling performed, including a summary of (1) the ways in which scientific and public review of the current analysis occurred, and (2) the 2014 HREA and important updates in modeling tools and approaches that contributed to planning and completion of the analyses presented in this document is provided in sections 3D.1.1 through 3D.1.4. The detailed description of the modeling tools, algorithms, input data and output metrics, along with an assessment of how variability is addressed in the analysis is provided in section 3D.2. Finally, the exposure and risk results, including a characterization of uncertainties, are found in section 3D.3.

3D.1.1 Planning and Scientific/Public Review of the Current Analysis

As described in section 1.4 of the PA, a consultation with the Clean Air Scientific Advisory Committee (CASAC) was held in November 2018 on the draft IRP to receive their input and comments from the public were also solicited on the draft IRP. Both comments from the CASAC and the public were considered in shaping the analysis plans, which were summarized in the final IRP.

The draft PA, with a draft version of this appendix was provided to the CASAC for its review and to the public for public comment, as summarized in section 1.4 of this (final) PA. In consideration of comments from the CASAC (Cox, 2020) and the public a number of additional analyses and presentations have been added. In consideration of CASAC recommendations and public comments, this document includes presentations reflecting further analyses, investigations and/or clarifications of the available data with regard to a number of areas.

- Analyses of data on outdoor activity by different population groups including those identified as at risk in this review (e.g., children with asthma and older adults) during times of day when O_3 may be elevated (section 3D.2.5.3);
- Estimates for the comparison-to-benchmarks analysis additionally summarized in light of the estimates from the last review (section 3D.3.2.4);

[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.

- Evaluation of risk characterization uncertainty related to its representation of population groups having health conditions other than asthma, of older adults, and of outdoor workers (section 3D.3.4.1);
- Evaluation of uncertainty in estimates for people with asthma that may be associated with method for identifying individuals with asthma (section 3D.3.4.1);
- Evaluation of uncertainty with the E-R function and risk estimates (section 3D.3.4.1);
- Analyses investigating the sensitivity of the MSS model outputs to the value assigned the individual variability parameter, and to low-level ventilation rates, as well as overall model uncertainty in the MSS model (section 3D.3.4.1).

3D.1.2 Overview

Estimates of human exposure to O_3 can provide meaningful answers to policy-relevant questions regarding exposures of concern and resulting risk estimates. This is particularly true when the important elements of O_3 exposure, i.e., the frequency, magnitude, duration, and pattern, are accounted for and when the exposures are estimated using policy-relevant ambient air quality scenarios, i.e., ambient air conditions that either just meet the current O_3 standard or other air quality scenarios. Further, the policy-relevance of these estimated O_3 exposures can be extended when they are linked with adverse health outcome data obtained from controlled human exposure studies to quantitatively estimate health risk. As a result, via the quantitative relationships that exist between ambient air concentrations, exposures, and health effects, one can estimate the impact varying air quality conditions have on public health.

Exposure to O_3 can be directly estimated by monitoring the concentration of O_3 in a person's breathing zone (close to the nose/mouth) using a personal exposure monitor. Studies employing this measurement approach have been reviewed in the current and 2013 O_3 ISAs and in past O_3 Air Quality Criteria Documents (AQCDs; U.S. EPA, 1986, 1996, U.S. EPA, 2006). Personal exposure measurements from these studies can be useful in describing a general range of exposure concentrations (among other reported measurement data) and in identifying factors that may influence varying exposure levels. However, these measurement studies of personal exposure to O_3 are largely limited by the disparity between measurement sample durations and durations of interest, and in appropriately capturing variability in population exposure occurring over large geographic areas, particularly when considering both O_3 concentrations in ambient air (e.g., spatial variability) and population (e.g., age, sex) attributes that greatly influence exposure.

Because of these limitations in personal exposure measurement data, more commonly human exposure is estimated using sophisticated models that better account for physical (e.g., meteorology) or personal (e.g., age) attributes that may strongly influence variability in exposures. These exposure models can combine information on ambient air O_3 concentrations in various microenvironments, e.g., near roads, in schools, etc., with information on activity

patterns for individuals sampled from the general population or specific subpopulations, e.g., children with asthma. When integrating these varied data (among many others such as population demographics and disease prevalence) and understanding the key factors affecting exposure, exposure models can be more informative than the limited information given by measurement data alone.

Ozone exposure is highly dependent on the ambient air concentrations in an urban area, which vary spatially and temporally. An exposure model can reasonably estimate exposures for any perceivable at-risk population (e.g., people with asthma living in a large urban area) and considering any number of defined hypothetical air quality conditions (e.g., those in which concentrations just meet a particular air quality standard) provided underlying data exist to generate such estimates. Further, exposure models that account for variability in human physiology can also realistically estimate pollutant intake dose by using activity-specific ventilation rates. Each of these important features of O_3 exposure cannot realistically be measured for a study group or population of interest over wide ranging temporal and spatial scales, particularly when considering time, cost, and other constraints, and serve as the justification for using a modeling approach to estimate exposure and health risks.

3D.1.3 2014 Ozone Exposure and Risk Assessment

The 2014 HREA included two types of risk analyses. The first type of risk analysis, exposure-based risk, used health effect information obtained from controlled human exposure studies (summarized in the IRP, section 5.1.1.1). The second type, epidemiologic-based risk, used concentration-response functions derived from epidemiologic studies (IRP, section 5.1.1.2). Because we used only the exposure-based risk analysis approach for this review (see section 3D.1.4 below; IRP, 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.

For the 2014 HREA, two exposure-based risk analyses[8] were performed in a set of 15 urban study areas[9] and for five different air quality scenarios: unadjusted ambient air O_3 conditions, air quality adjusted to just meet the then-existing standard (75 ppb, annual 4th highest daily maximum 8-hr average concentration, averaged over a 3-year period), and air quality adjusted to just meet potential alternative O_3 standards having the same form and averaging times, with levels of 70, 65 and 60 ppb.[10] The scenarios were based on air quality from two 3-year periods: 2006-2008 and 2008-2010. The first exposure-based risk analysis involved comparison of population exposures, while at elevated exertion, to benchmark concentrations. The exposure-to-benchmark comparison characterizes the extent to which individuals in at-risk populations could experience exposures of concern (i.e., average exposure concentrations at or above specific benchmarks while at moderate or greater exertion levels) while engaging in their daily activities in study areas with air quality adjusted to just meet the then-existing standard and other O_3 air quality conditions. Results were characterized using three benchmark concentrations (60, 70, and 80 ppb O_3), exposures to which in controlled human exposure studies yielded different occurrences and severity of respiratory effects in the human subjects (2014 HREA, section 5.2.8). The second exposure-based risk analysis involves estimated population occurrences of ambient air O_3-related lung function decrements. The lung function risk analysis provides estimates of the extent to which populations in such areas could experience decrements in lung function. Based on the range of health effects considered clinically relevant and the potential for varied responses in healthy individuals versus people with asthma, the lung function risk analysis reported estimates for risk of lung function decrement at or above three different magnitudes, i.e., forced expiratory volume in one second (FEV_1) reductions of at least 10%, 15%, and 20% (2014 HREA, section 6.2.1).

Key observations and insights from the O_3 exposure-to-benchmark comparison and lung function risks, in addition to important caveats and limitations, were addressed in Section II.B of 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.

the 2014 HREA, and most particularly the exposure to benchmarks analysis were important considerations in the 2015 decision on revisions to the primary O_3 standard (80 FR 65362-65365, October 26, 2015).

3D.1.4 Current Analysis

As described in the IRP (section 5.1.2.2), the quantitative analyses for this review focus on the comparison to benchmark exposure-based risk analysis approach, based on the controlled human exposure studies. In part, this is because substantial updates to data, information, models, and tools are available, ensuring that the new exposure and risk estimates are both improved and appropriately targeted. Additionally, estimates from the exposure-based analyses, particularly the comparison of daily maximum exposures to benchmark concentrations, were most informative to the Administrator's decision in the last review (IRP, section 3.1.2). This largely reflected the EPA conclusion that "controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O_3 exposures," and recognition that "effects reported in controlled human exposure studies are due solely to O_3 exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies)" (80 FR 65343, October 26, 2015). In the last 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).

3D.1.4.1 Aspects updated since 2014

A number of aspects of the exposure-based risk analyses were updated since the 2014 HREA. The updates were based on important uncertainties characterized in the last review and having newly available data, information, models, and tools that could provide risk estimates in which we have greater confidence that was the case for the risks estimated in the last review, as summarized in Appendix 5A of the IRP. These updates include:

- Air quality
  - More recent (2015-2017) ambient air monitoring data from US EPA's Air Quality System (AQS) having unadjusted concentrations at or near the current standard (section 3D.2.3.2);
  - Updated photochemical model (CAMx version 6.5)[11] to adjust ambient air concentrations to just meet the air quality scenarios to be assessed (section 3D.2.3.3).
- 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/*.

- More recent (2010) U.S. Census demographics and commuting data (section 3D.2.2.1);
- More recent (2013-2017) asthma prevalence for census tracts in all study areas (section 3D.2.2.2);
- Updated equations to estimate resting metabolic rate (RMR) (section 3D.2.2.3.2) and associated ventilation rate ($\dot{V}_E$) (section 3D.2.2.3.3);
- Improved matching of controlled human exposure study duration (6.6-hr) and target ventilation rate to that estimated for simulated individuals (7-hr duration, distribution accounting for resting ventilation) and used for benchmark comparisons and population-based E-R lung function risk (section 3D.2.2.3.3 and 3D.2.8.1);
- More recent (2015-2017) meteorological data to reflect the assessment years (section 3D.2.4)
- Increased number of diary-days and added new activity descriptions to activity pattern data base (section 3D.2.5.1);
- Most recent MSS-FEV_1 model (McDonnell et al., 2013) to estimate individual lung function risk (section 3D.2.8.2.2);
- New evaluations of important uncertainties (section 3D.3.4.2): form of E-R function, E-R function risk confidence intervals, low exposure concentration contribution to lung function risk, influence of ventilation rate on lung function risk, influence of variability parameter settings in MSS-FEV_1 model.

3D.2 POPULATION EXPOSURE AND RISK APPROACH

This section describes the data, information, models, and tools used to characterize exposure and health risk associated with O_3 in ambient air for three air quality scenarios. As summarized above in section 3D.1.4, the overall analysis approach is based on linking the health effects information observed in controlled human exposure studies to estimated population-based exposures that reflect our current understanding of concentrations of O_3 in the ambient air.

Population exposures and risks were estimated using the EPA's Air Pollution Exposure Model (APEX), version 5. APEX is a multipollutant, population-based, stochastic, microenvironmental model that can be used to estimate human exposure via inhalation for criteria and toxic air pollutants. APEX is designed to estimate human exposure to these pollutants at the local, urban, and consolidated metropolitan level. In this analysis, we used APEX to estimate exposure and risk in eight study areas, the details of which are provided in the following subsections. Additional information not provided here regarding all of APEX modules, algorithms, and modeling options can be found in the APEX User's Guide (U.S. EPA, 2019a; U.S. EPA, 2019b).

Briefly, APEX calculates the exposure time-series for a user-specified duration and number of individuals. Collectively and by design, these simulated individuals are intended to be

a representative random sample of the population in the chosen study area. To this end, demographic data from the decennial census are used so that appropriate model sampling probabilities can be derived considering personal attributes such as age and sex and used to properly weigh the distribution of individuals in any given geographical area. For the exposure and risk analyses performed here, the core demographic geographical units for estimating exposure are census tracts. For each simulated person, the following general steps are performed:

- Select personal attribute variables and choose values to characterize the simulated individual (e.g., age, sex, body weight, disease status);
- Construct an activity event sequence (a minute-by-minute time-series) by selecting a sequence of appropriate daily activity diaries for the simulated individual (using demographic and other influential variables);
- Calculate the pollutant concentrations in the microenvironments (MEs) that simulated individuals visit;
- Calculate the simulated individual's exposure, and simultaneously, their breathing rate for each exposure event and summarize for the selected exposure metric.

A simulated individual's complete time-series of exposures (i.e., *exposure profile*), representing intra-individual variability in exposures, is combined with the exposure profiles for all simulated individuals in each study area and summarized to generate the population distribution of exposures, representing inter-individual variability in exposures. As described above regarding air quality and in the sections that follow describing APEX model inputs and approaches to estimating exposure, the overarching goal of the exposure and risk analysis is to account for the most significant factors contributing to inhalation exposure and risk, i.e., the temporal and spatial distribution of people and pollutant concentrations throughout the study area and among the microenvironments. The population distributions of exposures are then combined with the health effects information to characterize associated risk via two types of metrics: a comparison to benchmark concentrations and lung function risk. The details of the model input data and general approaches used for estimating exposure and risk are described in the sections that follow.

3D.2.1 Urban Study Areas

To identify a list of urban areas for the current analysis, we first considered the list of 15 urban study areas evaluated in the 2014 HREA, which represented a range of geographic areas, encompassing variability in air quality, climate, and population demographics. We also considered other candidate study areas (e.g., Phoenix). As was done for the 2014 HREA, we developed criteria to select urban study areas for the current exposure and risk analysis. Those criteria are as follows:

- Have at least 10 ambient air monitors having complete year data for the 2015-2017 period;
- Combined statistical area (CSA)/metropolitan statistical area (MSA) ambient air monitor design values are between 60-80 ppb, thus having minimal adjustment needed to just meet the current 8-hr O_3 NAAQS;
- CSA/MSA population between 2 to 10 million;
- Anticipated reasonable air quality model performance[12]; and
- Reasonable geographic distribution across continental U.S.

Based on these selection criteria, we chose the eight study areas listed in Table 3D-1 (and shown in Figure 3D-1) to develop our population exposure estimates. Included also are the nine other study areas considered but not selected for the current exposure and risk analysis. We recognize the Sacramento study area does not meet the design value criterion (i.e., 86 ppb is outside the range of values considered), however we relaxed this criterion to include a study area in the Pacific/West region of the U.S and because exposure and risk was evaluated in the 2014 HREA (as opposed to using Los Angeles which was also evaluated in the 2014 HREA but has a 2015-17 design value of 112 ppb).

We broadly defined the study areas using geographic coordinates to center the overall exposure modeling domain for the APEX modeling (Table 3D-2). A wide city radius (i.e., 30 km) along with standard political/statistical county aggregations (e.g., whether in a CSA/MSA) were then used to identify the specific counties that comprise each study area. As a result, 131 counties containing 9,725 census tracts were used to define the air quality domain in the eight study areas.[13] As done for prior exposure-based assessments, ambient air O_3 concentrations were estimated to census tracts to capture spatial heterogeneity that may exist within each study area (PA, 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 O_3 concentrations is summarized in section 3D.2.3 below and is described fully in the Appendix 3C of this PA.

Figure 3D-1. Locations of the eight study areas selected for the current O_3 exposure and risk analysis.

Table 3D-1. Criteria used to identify and select urban study areas for inclusion in the O_3 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 O_3 issues (Denver), low monitor density (Salt Lake City), monitor issues (New York), and high DVs (Los Angeles).

Table 3D-2. General description of ambient air quality domains for the eight study areas.

CSA/MSA			Coordinates		Counties [A] (n)	Tracts (n)
Name	ID#	Abbrev.	Longitude (degrees)	Latitude (degrees)		
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 (PA, Appendix 3C, section 3C.2).

3D.2.2 Simulated Populations

APEX stochastically generates a user-specified number of simulated people to represent the population in the study area. The number of simulated individuals can vary and is dependent on the size of the population to be represented. For the current analysis, the number of simulated individuals was set at 60,000 for each of the children and adult study groups (which includes people with asthma for both of these study groups) to represent population residing within each study area (i.e., between 2 and 10 million). Each simulated person is represented by a *personal profile*. The personal profile includes specific attributes such as an age, a home tract, a work tract (or is not employed), housing characteristics, physiological parameters, and so on. The profile does not correspond to any particular individual that resides in the study area, but rather represents a simulated person. Accordingly, while a single profile does not, in isolation, provide information about the study population, a distribution of profiles represents a random sample drawn from the study area population. As such, the statistical properties of the distribution of simulated profiles are meant to reflect statistical properties of the population in the study area.

APEX generates population-based exposures using several population databases. Based on the geographic boundaries defining the study areas and the study groups of interest, APEX simulates representative individuals using appropriate geographic, demographic, and health status information provided by existing population-based surveys. For the current exposure and risk analysis, population input data sets are organized by U.S. census tracts.

Several updates were made to the APEX model inputs and algorithms for use in simulating the populations of interest in this exposure and risk analysis and are described in the following sections: population demographic data that are based on the 2010 census (section 3D.2.2.1), asthma prevalence rates based on the 2013-2017 National Health Interview Survey (NHIS) that vary by age, sex and geographic location (section 3D.2.2.2), and data and equations used to approximate personal attributes such as body weight, resting metabolic rate, and breathing rate (section 3D.2.2.3).

3D.2.2.1 Demographics

As briefly described in section 3D.2.1 (and more fully in section 3D.2.3 below and the PA, Appendix 3C), ambient air concentrations were modeled to census tracts in each study area to capture spatial heterogeneity in ambient air O_3 concentrations. Population data were generated using the same spatial scale to also account for variability in population demographics. Tract-level population counts were obtained from the 2010 Census of Population and Housing Summary File 1.[14] Summary File 1 contains what the Census program calls "the 100-percent data," which is the compiled information from the questions asked of all (100% of) people and housing units in the U.S. Three national-based APEX input files[15] are used for the current exposure and risk analysis as follows.

- *Population_sectors_US_2010.txt*: census tract identifiers (IDs), latitudes and longitudes in degrees.
- *Population_female_All_2010.txt*: census tract IDs, tract-level population counts for females, stratified by 23 age groups.[16]
- *Population_male_All_2010.txt*: census tract IDs, tract-level population counts for males, stratified by the same 23 age groups as done for females.

3D.2.2.2 Asthma Prevalence

The four population study groups included in this exposure assessment are adults (19 to 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/sf1.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.

asthma, based on their identification as an at-risk population (PA, section 3.3.2; ISA, section IS.4.4.2). To best approximate the number (and percent) of individuals comprising the latter two population groups in each study area, we considered several influential variables that could affect asthma prevalence. It is widely recognized that there are significant differences in asthma prevalence based on age, sex, U.S. region, and family income level, among other factors.[19] There is spatial heterogeneity in family income level across census geographic areas (and also across age groups)[20] and spatial variability in local scale ambient air concentrations of O_3 (e.g., PA, Appendix 3C, Figures 3C-91 through 3C-106). Thus, we accounted for these particular attributes of this study group and their spatial distribution across each of the study areas to better estimate the variability in population-based O_3 exposures and risks for these at-risk population groups.

With regard to asthma prevalence, the data are used to identify if a simulated individual residing within a modeled census geographic area has asthma. The data are not used for selection of any other personal attribute nor in the selection of activity pattern data. Thus, our primary objective with these data was to generate census tract-level prevalence that reflect variability in asthma prevalence contributed by several known influential attributes (i.e., age, sex, family income level, geographic location). Two data sets were identified and linked together to estimate asthma prevalence used for this exposure and risk analysis: asthma prevalence and population data.

First, asthma prevalence data were obtained from the 2013-2017 National Health Interview Survey (NHIS) and are stratified by NHIS defined regions (Midwest, Northeast, South, and West), age, and sex.[21] These asthma prevalence data are particularly useful given that age is expressed as a continuous variable, a feature not found in other asthma prevalence data that are available (e.g., state or county level data). We explored variables that were available in the NHIS data set that contributed to variability in asthma prevalence and that could be used to extrapolate the asthma prevalence to a finer geographic scale than the NHIS-provided four regions. The linking variable had to be common with variables available in the population demographic data. Based on this criterion, we selected family income level to poverty thresholds (i.e., whether the family income was considered at/below or above a factor of 1.5 of the U.S. Census estimate of poverty level for the given year) and used that as an additional variable to stratify the NHIS asthma prevalence.

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[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*.

Then, we obtained population data from the 2017 Census American Community Survey (ACS) to estimate family income level to poverty thresholds at the census tract level and stratified by several ages and age groups.[22] By combining the NHIS and U.S. Census population data sets, we developed census tract level asthma prevalence for children (by age in years) and adults (by age groups), also stratified by sex (male, female) that were weighted by the individual census tract population and family income level proportions. Finally, we adjusted the census tract-level asthma prevalence data based on individual state-level prevalence data from the 2013-2016 Behavioral Risk Factor Surveillance System (BRFSS).[23] This was done because overall, the asthma prevalence data reported from BRFSS were consistently higher than that derived from the NHIS data, particularly when considering adults, and thus resulted in an upward adjustment to the initially derived NHIS census tract level data set. A detailed description of how the NHIS, U.S. Census, and BRFSS data were processed and combined to create the data set used for input to APEX is provided in Attachment 1. The national-based APEX input file is used for the current exposure and risk analysis as follows:

- *asthma_prev_1317_tract_053119_adjusted.txt*: census tract IDs, tract-level asthma prevalence (in fractional form) stratified by sex, 18 single year ages (for ages <18),[24] and 7 age groups (for ages > 17).

The asthma prevalence varies for the different ages and sexes of children and adults[25] that reside in each census tract of each study area. We evaluated the spatial distribution of the asthma prevalence using the tracts that comprise the air quality domain in each study area. We first separated the estimates for children from those for adults and calculated the distribution of asthma prevalence for the tracts, stratified by sex (Table 3D-3). These summary statistics represent the range of age- and sex-specific probabilities for the census tracts comprising each study area that are used by APEX to estimate the number of individuals that have asthma.

[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).

Table 3D-3. Descriptive statistics for children and adult asthma prevalence, using all census tracts within eight consolidated statistical areas (CSAs) in the APEX 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	95th percentile	99th 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.

In general and consistent with broadly defined national asthma prevalence (e.g., Table 3-1 of the PA), male children have higher rates than female children[26] and adult females have higher rates than adult males.[27] The overall asthma prevalence for children was similar to that estimated for adults, largely the result of having a greater BRFSS adjustment applied to adult females compared to that applied to children of either sex.[28] As described above, and by design (i.e., in using age, sex, and family income variables) there is wide ranging spatial variability in the estimated asthma prevalence. For instance, the Boston, Detroit, and Philadelphia study areas have some of the highest asthma prevalence for boys and adult women considering most of the descriptive statistics, with rates of 25% or higher in one or more census tracts for a given year of age (Table 3D-3). In contrast, the Dallas study area exhibits some of the lowest asthma prevalence (and low variability) for any of the four age/sex groups compared to the other study areas.

There are other personal attributes shown to influence asthma prevalence, such as race, ethnicity, obesity, smoking, health insurance, and activity level (e.g., Zahran and Bailey, 2013). The set of variables chosen to stratify asthma prevalence for use in this exposure and risk analysis (i.e., age, sex, and family income level) was based on maximizing the potential range in asthma prevalence variability, maximizing the number of survey respondents comprising a representative subset study group, and having the ability to link the set of attributes to variables within the Census population demographic data sets. Many of the additional influential factors identified here are not available in the census population data and/or have limited representation in the asthma prevalence data (e.g., the survey participant does/does not have health insurance, or they did/did not provide a response to a question regarding their body weight). Race is perhaps the only attribute common to both the prevalence and population data sets that could be an important influential factor and was not directly used to calculate asthma prevalence. However, the use of race in calculating asthma prevalence, either alone or in combination with family income level, would further stratify the NHIS analytical data set and appreciably reduce the number of individuals of specific age, sex, race, and family income level, potentially reducing 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).

stratification. Because family income level already strongly influences asthma prevalence across all races and stratifies the NHIS data into only two subgroups (i.e., above or below the poverty threshold) in comparison to the larger number of subgroups a race variable might yield, family income was chosen as the next most important variable beyond age and sex to rely on for weighting the spatial distribution of asthma prevalence.

3D.2.2.3 Personal Attributes

In addition to using the above demographic information to construct the simulated individuals, each modeled person is assigned anthropometric and physiological attributes by APEX. All of these variables are treated probabilistically, accounting for interdependencies where possible, and reflecting variability in the population. It is not the intention of this document to provide detailed description of all the model inputs in each of the files and the data used in their derivation, and where additional details exist, appropriate reference materials are provided. We describe further a few APEX model inputs that have been recently updated and that are available for use in this exposure and risk analysis. These are new statistical distributions for estimating body weight, equations for estimating resting metabolic rate, and equations for estimating activity-specific ventilation rate. Each of these data and algorithms are important, particularly the ventilation rate (section 3D.2.2.3.3), because the health response observed in the controlled human exposure studies is concomitant with elevated breathing rate. Brief descriptions of the data used to develop these generalized (i.e., non-O_3 specific) input files are provided in the sections below. For additional detail, see U.S. EPA (2018) Appendices G and H, and the data within the APEX input files.

3D.2.2.3.1 Body Weight and Surface Area

Anthropometric attributes utilized by APEX in various assessments for estimating exposures or doses can include height, body weight (BW), and body surface area (BSA). Two key personal attributes determined for each individual in this assessment are BW and BSA, both of which are used in the calculation of a number of other variables associated with estimating exposures (e.g., ventilation rate).

Regarding the estimation of body weight, a new APEX input file was recently generated using 2009-2014 National Health and Nutrition Examination Survey (NHANES) data.[29] Briefly, body weight and height data for surveyed individuals were obtained and stratified by sex and single years for ages 0 – 79; all ages above 80 were combined as a single age group. Statistical 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.

log-likelihood statistic. Body weight was found to best fit a lognormal distribution; height was found to best fit a normal distribution. Because height and body weight are not independent, the joint distributions of height and logarithm of body weight were fit assuming a bivariate normal distribution. Then, parameters defining the joint distributions[30] were smoothed using a natural cubic spline to have them represent continuous functions of age rather than vary discontinuously. In addition, having the smoothed parameters could be used to extrapolate information obtained from the single age year distributions (ages 0 – 79) to approximate statistical distributions of body weight for ages ≥80. To do so, a linear function was fit to ages 70 and above to extrapolate the parameter values (and hence the statistical distributions of body weight) up to age 100.

These body weight distributions are randomly sampled by APEX to estimate an age and sex-specific body weight for each simulated individual. Comparison of the new distributions to the body weight distributions previously used by APEX and developed from the 1999-2004 NHIS indicate, for both sexes and across all ages, simulated body weight is about two percent greater using the updated distributions. This difference is expected given the consistent trend of increasing body weight that has occurred in the U.S. population over the past few decades.

Age- and sex-specific body surface area, a variable used in conjunction with breathing rate to approximate moderate or greater exertion (section 3D.2.2.3.3) is estimated for each simulated individual (Equation 3D-1) and is based on an equation provided in Burmaster (1998):

$$BSA = e^{-2.2781} \times BW^{0.6821} \qquad \text{Equation 3D-1}$$

One standard APEX input file is used for the current O_3 exposure and risk analysis:

- *Physiology051619_Ufixed.txt*: Provides parameters for estimating body weight (log BW, standard deviation of BW, lower and upper bounds of BW, by single age years 0-100 and by two sexes) and regression coefficients used in estimating BSA for all sexes and ages.

3D.2.2.3.2 Energy Expenditure and Oxygen Consumption Rates

Energy expended by different individuals engaged in different activities can have an important role in pollutant-specific exposure and/or dose. For example, energy expenditure is related to ventilation rate, which is an important variable in estimating exposure and risk given that the O_3-induced lung function response has been documented to occur under conditions of elevated ventilation (PA, section 3.3.1.1). In addition, because we are also interested in exposures that occur over relatively short durations (i.e., < 8 hours), estimating activity-specific ventilation rate ($\dot{V}_E$) has always been an important motivation behind the development of the algorithm used by APEX. The fundamental basis for $\dot{V}_E$ algorithm is founded in energy expenditure which, for our modeling purposes here, can be related to an individual's resting

[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.

metabolic rate (RMR) or the energy expended while an individual is at complete rest, along with the energy expended while an individual performs activities involving greater exertion, termed here as metabolic equivalents of work (METs) (McCurdy, 2000). The approaches used by APEX for estimating RMR and METs are described below, beginning first with the update to the equations used for estimating a simulated individual's RMR.

Since the 2014 HREA,[31] we have reviewed recent RMR literature and other published sources containing individual data and have compiled the associated individual RMR measurements, along with associated influential attributes such as age, sex, and body weight, where available. Data from these individual studies were then combined with RMR data reported in the Oxford-Brookes database (Henry, 2005; IOM, 2005) and screened for duplicate entries. In addition, observations missing values for RMR, BW, age, or sex were deleted, resulting in a dataset containing 16,254 observations (9,377 males and 6,877 females). Using this new RMR dataset and having a goal of updating the previous RMR equations and reducing discontinuities in RMR between age groups, new equations were developed.

Details regarding the data, the derivation, and performance evaluation of the new equation that APEX uses to estimate RMR are provided in U.S. EPA (2018), Appendix H. Briefly, the equations follow the general format of a multiple linear regression (MLR) model, using age and body weight as independent variables to estimate each simulated individual's RMR, along with a residual error term (ε).[32] It is known that RMR and BW, as well as RMR and age, are not exactly linearly related; the algorithms developed here use BW (in kg), age (in years), and the natural logarithms of BW and (age+1)[33] as follows in Equation 3D-2, with their parameter estimates provided in Table 3D-4.

$$RMR = \beta_0 + \beta_1 \mathrm{BW} + \beta_2 \log(\mathrm{BW}) + \beta_3 Age + \beta_3 \log(Age) + \varepsilon_i \qquad \text{Equation 3D-2}$$

When comparing observed versus predicted values, the new RMR equations have a bias of less than 0.5%, compared to the previously used APEX equations which had a bias of between 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.

previous equations have been reduced when using these updated equations in APEX. One standard APEX input file is used for the O_3 exposure and risk analysis:

- *Physiology051619_Ufixed.txt*: Regression coefficients used to estimate RMR (kcal day^{-1}) for two sexes and six age groups.

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								

Following the estimation of an age- and sex-specific RMR for simulated individuals, the next variable used for estimating ventilation rate involved an approximation of the energy expended for activities an individual performs throughout their day. As mentioned above, activity-specific energy expenditure is highly variable and can be estimated using metabolic equivalents of work (METs), or the ratios of the rate of energy consumption for non-rest activities to the resting metabolic rate of energy consumption, as follows in Equation 3D-3:

$$EE = MET \times RMR \qquad \text{Equation 3D-3}$$

where,

EE = Energy expenditure (kcal/minute)
MET = Metabolic equivalent of work (unitless)
RMR = Resting metabolic rate (kcal/minute)

Statistical distributions of METs were developed for simulated activities using the physical-activity compendium (Ainsworth et al., 2011; hereafter "the compendium"). The compendium contains a point value for the MET associated with each of several hundred different activities. Activity-specific MET distributions were developed by cross-walking the

activities described in the compendium with the descriptions of activities in the activity pattern data base used by APEX (section 3D.2.5). The shape of the statistical distribution (e.g., normal, lognormal, triangular, point) for each activity was assigned based on the number of corresponding activities in the compendium and goodness-of-fit statistics. When simulating individuals, APEX randomly samples from the activity-specific METs distributions to obtain values for every activity performed. Two standard APEX input files are used for the current O_3 exposure and risk analysis:

- *MET_distributions_092915.txt*: MET distribution number, statistical form, distribution parameters, lower and upper bounds, activity description
- *MET_mapping_071018.txt:* activity codes, age group (where applicable), occupation group, MET distribution number, and activity description used to link of MET distributions to activities performed

The rate of oxygen consumption ($\dot{V}O_2$, Liters min^{-1}) for each activity is then calculated from the energy expended (kcal min^{-1}) using an energy conversion factor (ECF, Liters O_2 $kcal^{-1}$) as follows in Equation 3D-4:

$$\dot{V}O_2 = EE \times ECF \qquad \text{Equation 3D-4}$$

The value of the ECF is randomly selected from a uniform distribution for each person, U[0.20, 0.21] (Johnson, 2002, adapted from Esmail et al., 1995). One standard APEX input file is used for the current O_3 exposure and risk analysis:

- *Physiology051619_Ufixed.txt*: Parameters of the uniform distribution representing the ECF used for all ages and both sexes.

3D.2.2.3.3 Ventilation Rate

Human activities are variable over time, with a wide range of activities possible within only a single hour of the day. The type of activity an individual performs, such as sleeping or jogging (as well as individual-specific factors such as age, weight, RMR) will influence their ventilation rate. APEX estimates minute-by-minute ventilation rates that account for the expected variability in the activities performed by simulated individuals. Ventilation rate is important in this assessment because the lung function responses associated with short-term O_3 exposures coincide with moderate or greater exertion (2013 ISA, Table 6-1). In our exposure modeling approach, APEX generates the complete time-series of activity-specific ventilation rates and the corresponding time-series of estimated O_3 exposures and is directly used for the individual-based lung function risk (section 3D.2.8.2.2). APEX can then aggregate both the ventilation rate and exposure concentration for the duration of interest (e.g., 7-hr average), and they can be used for the benchmark comparison (section 3D.2.8.1) and estimating the population-based lung function risk (section 3D.2.8.2.1). Thus, the model provides O_3 exposure

estimates for the simulated individuals that pertain to specific target levels for both ventilation rate and exposure concentration. The approach to estimating activity-specific energy expenditure and associated ventilation rate involves several algorithms and physiological variables, with details found in the APEX User's Guide (U.S. EPA, 2019a, U.S. EPA, 2019b).

Using the existing measurement $\dot{V}_E$ dataset from Graham and McCurdy (2009), new $\dot{V}_E$ algorithms were developed for predicting activity specific $\dot{V}_E$ in the individuals simulated by APEX (Appendix H of U.S. EPA (2018)). The new $\dot{V}_E$ algorithms do not directly employ previously used variables to stratify the data (age groups, sex) and explain variability (age, body weight, height) in ventilation rate, effectively simplifying and reducing the number of equations. The new algorithms utilize a new variable, the maximum volume of oxygen consumed ($\dot{V}O_2m$) as an input.[34] Body weight, height, and sex – as well as fitness level (which is often represented by $\dot{V}O_2m$) - influence oxygen consumption for a particular activity. However, variability for each of these influential variables are already captured in the algorithm used to estimate each simulated individual's RMR, and subsequently, the estimation of their activity-specific $\dot{V}O_2$.[35] 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 which are estimated by APEX.

Details for the derivation of and performance evaluation of the new equation that APEX uses to estimate ventilation rate are provided in U.S. EPA (2018) Appendix H. Briefly, the $\dot{V}_E$ dataset contains 6,636 observations, with 4,565 males and 2,071 females. Similar to the earlier ventilation equation by Graham and McCurdy (2009), a mixed-effects regression (MER) model was fit because the MER separates residuals into within-person (e_w) and between-person (e_b) effects, known as intrapersonal and interpersonal effects, respectively.[37] It was found that the actual values of $\dot{V}O_2$ and $\dot{V}O_2m$ are less relevant than the fraction of maximum capacity, 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 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 *inter*personal variability, which is sampled once per person. $N(0, e_w)$ is an *intra*personal residual with standard deviation of $e_w = 0.07852$, which is sampled daily due to natural *intra*personal fluctuations in $\dot{V}_E$ that occur daily.

(using PROC TRANSREG; SAS, 2017) was used to determine the most appropriate variable transformation, indicating a power of 4 to 5 be used when only the log transformed $\dot{V}O_2$ was used as the independent variable and described in Equation 3D-5.

$$\dot{V}_E = e^{(3.300 + 0.8128 \times \ln(\dot{V}O_2) + 0.5126 \times (\dot{V}O_2 \div \dot{V}O_2m)^4 + N(0,e_b) + N(0,e_w))} \quad \text{Equation 3D-5}$$

In comparing the statistical fit of the new equation with the equations used by APEX previously to estimate ventilation rate, the resulting coefficient of determination (R^2 values) for the new equation ($R^2 = 0.94$) indicates an improved fit compared to that of the previous equations ($R^2 = 0.89$ - 0.92). Further, because the data were not stratified by age groups (or any other groupings), there are no discontinuities in predictions made across age boundaries as was observed when employing the previous equations. Information used in estimating ventilation rate is found in the following APEX two input files:

- *Physiology051619_Ufixed.txt*: parameters describing statistical distributions of normalized maximum oxygen consumption rate ($N\dot{V}O_2m$) for two sexes by single age years (0-100) (see, Isaacs and Smith, 2005).
- *Ventilation_062117.txt*: minimum and maximum age ranges, regression coefficients, between and within error terms used to estimate individual activity-specific ventilation.

To use this information to estimate health risks for children, the ventilation rates observed for the adult controlled human exposure study subjects need to be converted into rates that best reflect the different physiology of children. Consistent with prior REAs (U.S. EPA, 2009, 2014, 2018; Whitfield et al., 1996) we used an equivalent ventilation rate (EVR, L/min-m^2), which is essentially an allometrically normalized ventilation rate (Equation 3D-6), to estimate instances when any simulated individual reaches a ventilation rate relatively as high as that of the study subjects (i.e., termed here as moderate or greater exertion).

$$EVR = \frac{\dot{V}_E}{BSA} \quad \text{Equation 3D-6}$$

Before discussing the value used to determine whether a simulated individual is at moderate or greater exertion, a brief description of the controlled human exposure study protocol is warranted. Most of the controlled human exposure studies evaluating O_3 health effects of interest for our exposure benchmark analysis (e.g., Adams, 2006; Folinsbee et al., 1988) were conducted over a 6.6-hr exposure period, thus, the most relevant exposures and associated breathing rates for the exposure benchmark comparisons would be those occurring on average over a 6.6-hr period (not an 8-hr period as was used in previous REAs). The typical protocol for the 6.6-hr controlled human exposure studies employed a mixture of exercise and rest periods varied across the duration of the study, with an expectation that the study subject achieves, on

average, a target EVR of 20 L/min-m^2 (i.e., a ventilation rate of ~35 L/min in females and ~40 L/min in males) while exercising using a treadmill or cycle ergometer (e.g., Schelegle et al., 2009). Most researchers collected the ventilation data during periods of exertion and therefore reported the exercise-only conditions (e.g., Horstman et al., 1990; Folinsbee et al., 1988).

More specifically, during the 6.6-hr study experiments, 5 hours were used for exercise (i.e., six 50-minute (min) periods on a treadmill or cycle ergometer), with the remaining 1.6 hours comprised of a series of 10-min rest periods occurring immediately after the exercise along with a 35-min lunch break before the fourth exercise period. As a result of these rest/lunch periods, the study subject's actual ventilation rates (and hence EVRs) are expected to be less than the target/observed exercise levels reported in the controlled human exposure studies. Note, the simulated individuals used to estimate exposure and risk perform numerous activities throughout the day, each having varied durations and exertion levels (e.g., jogging, sleeping, eating). As such, when time-averaging across a simulated exposure period of interest, the period likely would contain ventilation rates of varying duration and intensity. For this review, to better match the ventilation information obtained from the controlled human exposure studies with that of the simulated individuals, we accounted for the impact from the rest/lunch time ventilation rate along with that attained during exercise to estimate an appropriate EVR for the study subjects.

Attachment 2 provides details regarding the data and approach used to estimate the EVR, an APEX model variable used to identify when a simulated individual is at moderate or greater exertion. Briefly, the controlled human exposure study data set available used to calculate EVR was comprised of 177 study subjects, each evaluated for 2 or more exposure levels (i.e., totaling 485 experiments), and having multiple measurements for each exercise period, yielding 4,024 individual EVR data points. Of these six studies providing raw data,[38] only Schelegle et al. (2009) mentioned resting $\dot{V}_E$ (and hence a resting EVR), with an average value for males and females estimated as 7.61 and 8.05 L/min-m^2, respectively and based on regression equations provided by Aitken et al. (1986). We calculated total (exercise and rest) EVR for each person across the 6.6-hr study period as a weighted average based on the observed EVR for the 5 hours of exercise and the estimated EVR for 1.6 hours of rest/lunch. Descriptive statistics were calculated and indicated the person-level EVR data were normally distributed, having a mean value of 17.32 (L/min-m^2) and a standard deviation of 1.25 (L/min-m^2). To reflect variability across simulated individuals, an EVR is probabilistically selected from this distribution once per person and used for the duration of their simulation period. This new approach for assigning a 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).

based on the distribution of ventilation rates achieved by all controlled human exposure study subjects, more appropriately reflects the EVR variability expected to exist in the simulated population compared to the approach used in the last two reviews (e.g., U.S. EPA, 2007a; U.S. EPA, 2007b; 2014 HREA) that assigned a single lower bound EVR value to all individuals.[39]

For practical and tractable modeling reasons, this individual-level EVR threshold is applied to APEX simulated individuals using a 7-hr averaging time (representing the 6.6-hr period rounded to whole numbers) in order to better represent the exposure study design than the previously used 8-hr average. Then, once a simulated individual is identified as having surpassed their personal 7-hr average EVR threshold in a given day, the level of their simultaneously occurring 7-hr average O_3 exposure is recorded by APEX. Retained for each simulated individual are the daily maximum 7-hr average exposure concentration(s) that occurred while at moderate or greater exertion over the assessment period.

3D.2.3 Ambient Air Concentrations

Ambient air concentrations serve as a fundamental input used by APEX to estimate exposure. There are two important attributes of ambient air concentrations to consider when estimating population exposure and risk using APEX: spatial and temporal variability. This is because there can be significant spatial and temporal heterogeneity in O_3 concentrations across each of the study areas and there is substantial flexibility by APEX in handling ambient air concentrations at varying scales, both temporally (e.g., hourly, daily) and spatially (e.g. 500-meter grid, census tract).

For this exposure and risk analysis (as done for the last review), we were interested in having hourly O_3 concentrations at the census tract level. Having these temporally and spatially resolved ambient air concentrations in each study area allows for better utilization of APEX temporal and spatial capabilities in estimating exposure and risk (e.g., the population data described in section 3D.2.2 are at a census tract level). Because APEX simulates where individuals are located and what they are doing at specific times of the day, more realistic exposure estimates are obtained in simulating the contact of individuals with these temporally and spatially diverse concentrations.

Ambient air monitors for O_3 capture the temporal scale of interest (i.e., hourly) and can provide general information regarding O_3 levels across an urban area. However, given their

[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-m^2 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-m^2 (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-m^2.

limited spatial representativeness, i.e., tens of monitors extending across areas >10,000 km^2, the monitors may not fully inform concentration variability that may exist at a finer spatial scale. In addition, of interest in this review are concentrations that represent a specific air quality scenario (e.g., ambient air quality that just meets the current standard). In general, due to varying levels of precursor emissions and meteorological conditions, most monitored 3-year periods do not have O_3 concentrations that just meet a specific air quality scenario of interest. Therefore, due to these two realities, modeling methods are used to achieve the desired temporal and spatial scale along with estimating ambient air O_3 concentrations that represent a specific air quality scenario.

The sections that follow briefly summarize the data and approaches used to estimate the air quality concentrations used by APEX. A detailed description on the air quality data collection, processing, adjustment, and evaluation is provided in Appendix 3C. First, section 3D.2.3.1 below provides information for the overall bounding of the modeling domains. The identification of ambient air monitoring data used as a foundation for representing fine-scale temporal and broad-scale spatial concentration variability is provided in section 3D.2.3.2. The approach used to adjust concentrations to just meet air quality scenarios of interest is described in section 3D.2.3.3. And finally, Section 3D.2.3.4 describes the technique used to interpolate the concentrations from the monitor locations to the desired spatial scale (i.e., census tracts). It is these estimated hourly census tract O_3 concentrations representing air quality scenarios that serve as the basic ambient air concentrations from which each simulated individual's microenvironmental concentrations and exposures are estimated (sections 3D.2.6 and 3D.2.7, respectively). Multiple unique APEX input files are used for the current exposure and risk analyses, one for each year and study area, and in the following two formats:

- *concsCSA[number]S[air quality scenario]Y[year].txt*: Tract IDs, hourly concentrations (ppm), calendar date, by study area and year
- *districtsCSA[number]Y[year].txt*: Tract IDs, latitude, longitude, begin and end date

3D.2.3.1 Spatial and Temporal Boundaries of Modeling Domains

APEX has several options to select air quality data to use for estimating exposure and risk. For this exposure and risk analysis, we used the list of counties that comprise each CSA/MSA and their geographic boundaries to define the broad spatial characteristics of each study area (Table 3D-5). As a result, simulated individuals residing within these counties would be part of the exposure modeling domain and any ambient air concentrations estimated within these counties would be used by APEX. Figure 3D-2 to Figure 3D-5 depict the spatial extent of the exposure and risk modeling domain in each study area, along with a visualization of tract-level population density and location of meteorological stations (see section 3D.2.4). The air

radius for APEX, a variable used to define the modeling domain, was set at 30 km to include all air quality receptors (i.e., census tracts) within each county to model exposures and risks.

For each study area, three years of recent air quality were selected to estimate exposures. The exposure periods are the O_3 seasons[40] for which routine hourly O_3 monitoring data were available, and defined by 40 CFR part 58, Appendix D, Table D-3. These periods are designed to reasonably capture variability in ambient air O_3 concentrations and meteorology and include the high concentration events occurring in each area. Having this range of air quality data across multiple years allows us to realistically estimate a range of exposures, rather than using a single year of air quality. The number of O_3 monitors in operation did not vary from year to year, thus, the overall spatial representation of each study area by the ambient air monitors (and that using the statistically interpolated data) remained constant for each year over the simulation period.

Table 3D-5. List of states, counties, and O_3 seasons that define the air quality and 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 (PA Appendix 3C, section 3C.2).
[B] These are the regulatorily required monitoring seasons (see section 2.3.1 of the main PA).

[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 O_3 season is considered to be synonymous with a year and exposure results are reported on a per year basis.

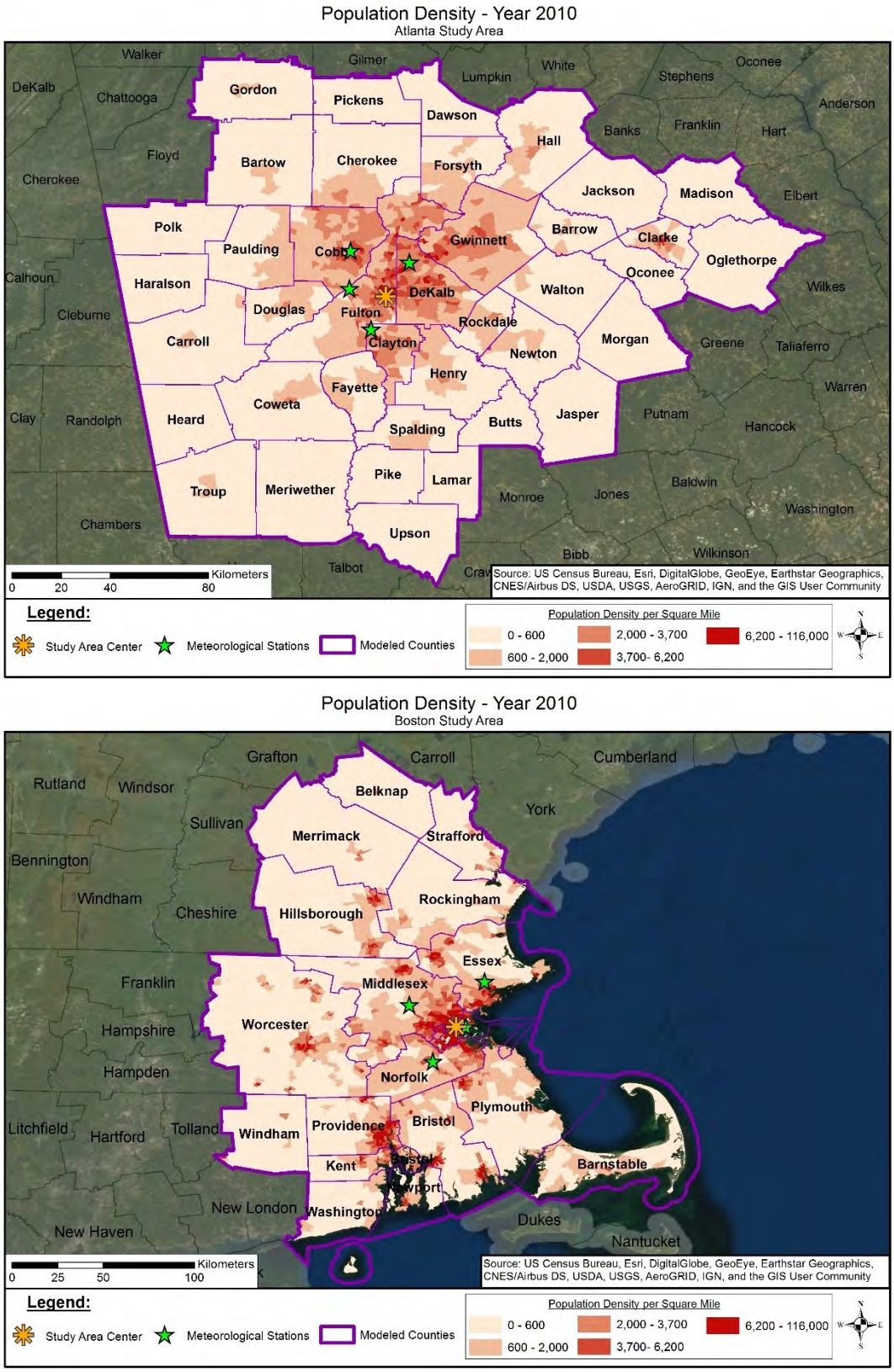

Figure 3D-2. County boundaries, census tract population densities, and meteorological stations in the Atlanta (top) and Boston (bottom) study areas.

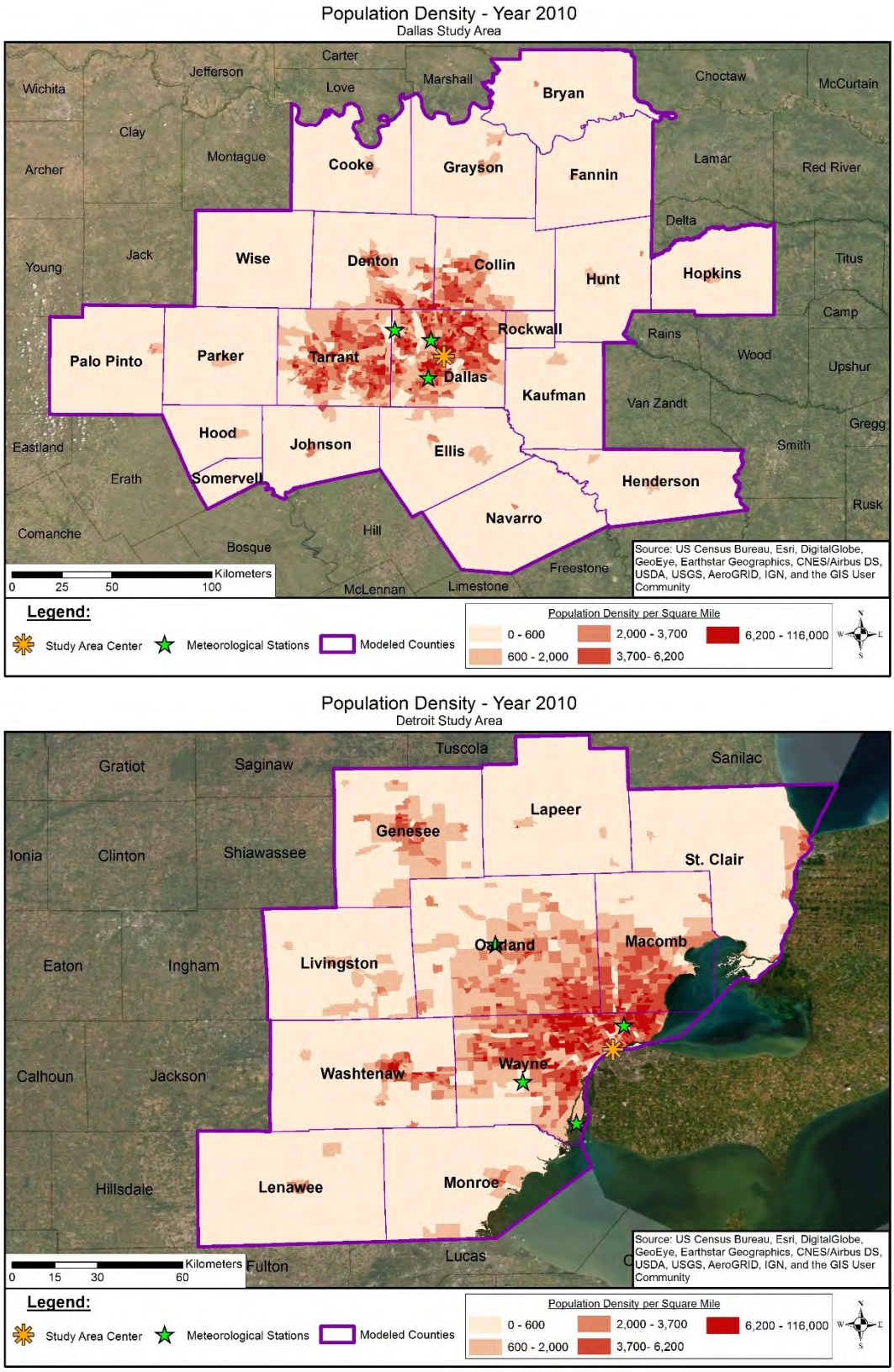

Figure 3D-3. County boundaries, census tract population densities, and meteorological stations in the Dallas (top) and Detroit (bottom) study areas.

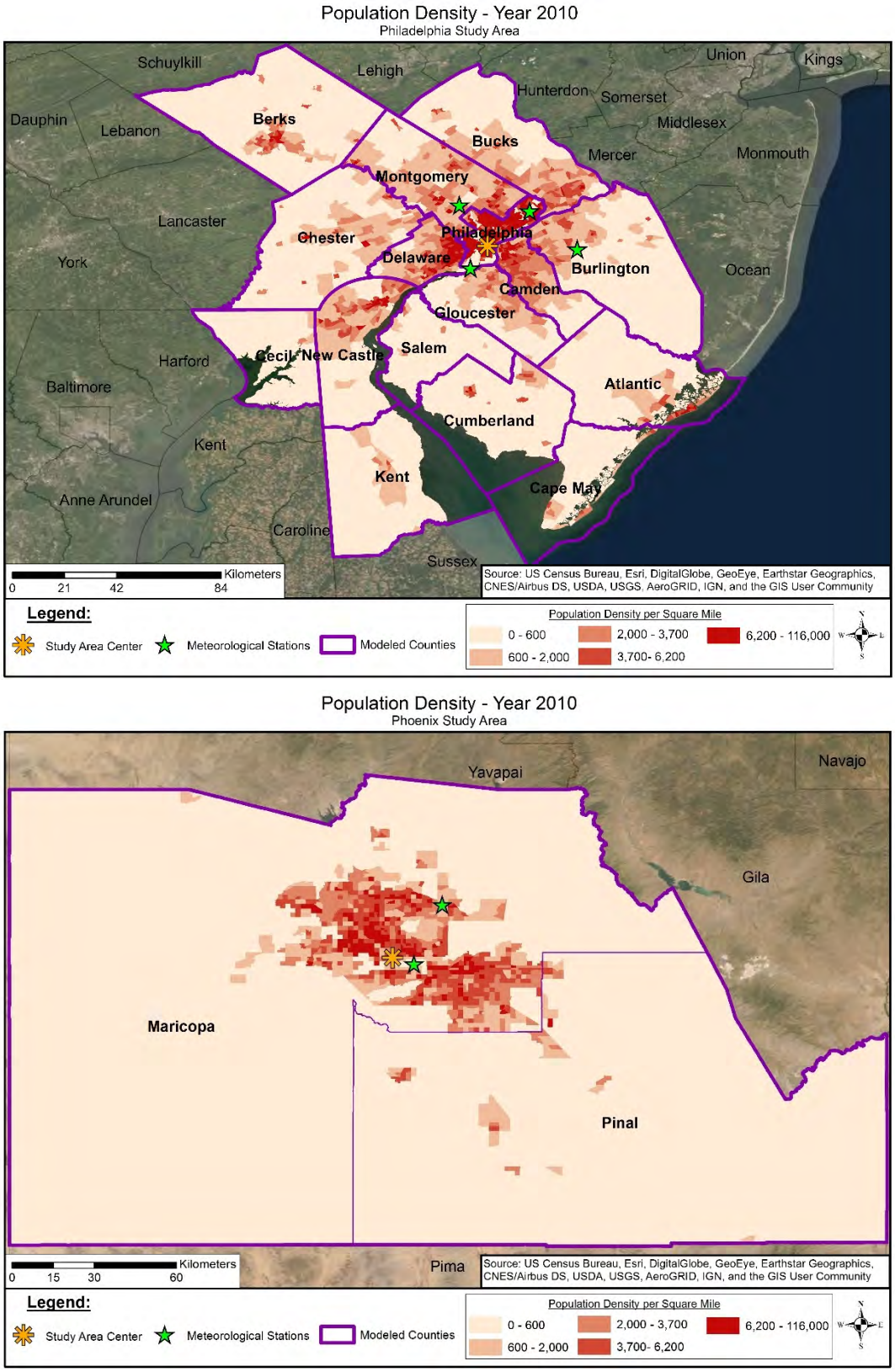

Figure 3D-4. County boundaries, census tract population densities, and meteorological stations in the Philadelphia (top) and Phoenix (bottom) study areas.

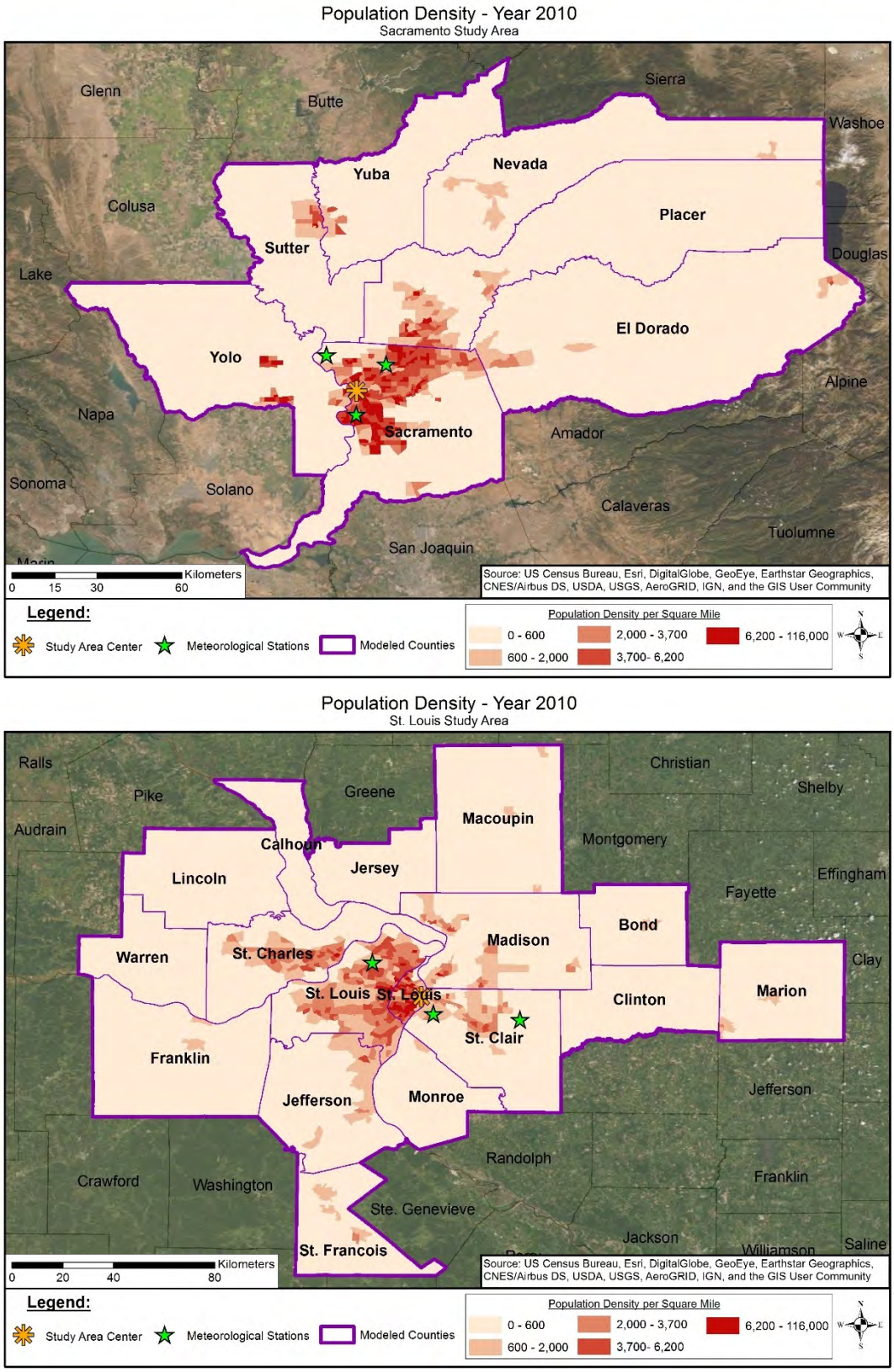

Figure 3D-5. County boundaries, census tract population densities, and meteorological stations in the Sacramento (top) and St. Louis (bottom) study areas.

3D.2.3.2 Ambient Air Monitoring Data

We used hourly O_3 concentrations from ambient air monitors in each study area for the 2015-2017 period to develop the air quality surface used for estimating exposure and risk (Table 3D-6; details in PA, Appendix 3C, section 3C.3).[41] Design values for monitors in each study area were used to determine the direction and magnitude of adjustments needed to just meet the current standard and the other two air quality scenarios (section 3D.2.3.3). The two other air quality scenarios are O_3 concentrations for which the highest design value in the area is just above or just below the current standard level: 75 ppb and 65 ppb. Ambient air monitors outside each study area, but within 50 km, were also used to improve spatial interpolation of air quality near the edges of the study areas (section 3D.2.3.4). All available ambient air O_3 monitor data were used to develop the adjusted air quality surfaces, however design values were not calculated for monitors having incomplete data.

[41] Briefly, hourly O_3 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).

Table 3D-6. List of ambient air monitor IDs, range of O_3 design values, and number of 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, *132230003*, 132319991, 132470001	63 – 75 (12)
Boston	CT: 090159991 MA: *250010002*, **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: *400130380* 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**, *261630093*, *261630094*	66 – 73 (13)
Philadelphia	DE: 100010002, 100031007, 100031010, 100031013, 100032004 MD: 240150003 NJ: 340010006, 340070002, 340071001, 340110007, 340150002 PA: 420110006, 420110011, *421010004*, **420170012**, 420290100, 420450002, 420910013, **421010024**, 421010048	64 – 80 (20)
Phoenix	AZ: 040130019, 040131003, 040131004, 040131010, 040132001, 040132005, 040133002, 040133003, 040134003, 040134004, *040134005*, 040134008, 040134010, 040134011, *040135100*, 040137003, 040137020, 040137021, 040137022, 040137024, 040139508, 040139702, 040139704, 040139706, **040139997**, 040213001, 040213003, 040213007, 040217001, 040218001	63 – 76 (30)
Sacramento	CA: 060170010, *060170012*, 060170020, **060570005**, *060570007*, 060610003, 060610004, 060610006, 060611004, 060612002, 060670002, 060670006, 060670010, 060670011, 060670012, *060670014*, 060675003, 061010003, *061010004*, 061130004, 061131003	63 – 86 (21)
St. Louis	IL: *170830117*, *170831001*, 171170002, 171190008, 171191009, 171193007, 171199991, 171630010 MO: 290990019, *291130003*, *291130004*, **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 PA, Appendix 3C, Tables 3C-20 to 3C-27. *Italic font* indicates monitor did not meet completeness criteria to calculate a design value.

3D.2.3.3 Model Adjusted Concentrations at Monitor Locations to Represent Air Quality Scenarios

Details of the approach used to develop the three air quality scenarios (design values of 70, 65 and 75 ppb) are provided in the PA, Appendix 3C, sections 3C.4 and 3C.5. Briefly, the ambient air concentrations described above in section 3D.2.3.2 were adjusted to just meet the current standard (70 ppb, annual 4th highest daily maximum 8-hr average concentration,

averaged over a 3-year period) and two other air quality scenarios (75 and 65 ppb, annual 4th highest daily maximum 8-hr average concentration, averaged over a 3-year period)[42] using a model-based O_3 methodology that adjusts the observed hourly O_3 concentrations to reflect the expected spatially and temporally varying impacts of changes in NO_X emissions. The methodology is similar to that used for the 2014 HREA and employs a photochemical air quality model combined with a tool that calculates modeled sensitivities of O_3 to precursor emission changes.

For the current analysis, the Comprehensive Air Quality Model with Extensions (CAMx)[43] served as the chemical transport model,[44] with 2016 selected as the base year for determining the adjustments needed for the 2015-2017 ambient air monitoring data. Model inputs include meteorological data,[45] emissions,[46] and initial and boundary conditions.[47] The evaluation of modeled versus observed O_3 concentrations for 2016 indicated CAMx generally reproduced the observed spatial and temporal patterns, with the exception of concentration underestimates occurring in winter across almost all regions (PA, Appendix 3C, section 3C.4.2).

The CAMx model was instrumented with the Higher order Decoupled Direct Method (HDDM) to calculate modeled nonlinear sensitivities of O_3 to emission changes (PA, Appendix 3C, section 3C.5). The photochemical modeling outputs included both modeled O_3 concentrations and sensitivities of O_3 concentrations to changes in NO_X emissions for each hour in a single year at all ambient air monitor locations (Appendix 3C, sections 3C.4 and 3C.5). Linear regression was used with these single-year 2106 model outputs to create relationships between the sensitivities and O_3 concentrations for each hour of each of the four seasons at each monitoring location. The relationships between hourly sensitivities and hourly O_3 for each season were then used with three years of ambient air monitoring data at each location to predict

[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.

hourly sensitivities for the complete 3-year record at each monitoring location. From these, we calculated hourly O_3 concentrations at each monitor location based on iteratively increasing NO_X reductions to determine the adjustments necessary for the monitor location with the highest design value in each study area to just meet the target value, e.g., 70 ppb for the current standard scenario (Appendix 3C, section 3C.5). For the 75 ppb air quality scenario, we note that three areas required an increase in NO_X emissions as their highest O_3 design values were below 75 ppb. For the other five study areas and that same air quality scenario and for all study areas with the other two air quality scenarios (i.e., 65 and 70 ppb), emission reductions were required (Table 3D-7).

Table 3D-7. Range of the percent NO_X emission changes needed to adjust air quality in the 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 PA, Appendix 3C, Table 3C-19.	

3D.2.3.4 Interpolation of Adjusted Monitor Concentrations to the Census Tracts Comprising Each Study Area

As described above, model-based relationships between O_3 and NO_X emissions were used to adjust hourly O_3 concentrations at the ambient air monitor locations (section 3D.2.3.2) to represent conditions in which the study area just meets the selected air quality scenario (section 3D.2.3.3). Simulated O_3 concentrations were then needed at a finer spatial scale than that given by the monitor sites to better represent the spatial heterogeneity in O_3 concentrations across locations frequented by the simulated population (and during the times frequented) across the study area. To accomplish this in each of the eight study areas, the adjusted hourly O_3 concentrations at monitoring sites were interpolated to census tract centroids using the Voronoi Neighbor Averaging (VNA; PA, Appendix 3C, section 3C.6). Nearby monitoring concentrations, for each hour, inform the estimation of O_3 for a given census tract using inverse distance weighting. In so doing, both spatial and temporal gaps in the desired air quality surface are filled simultaneously, resulting in a final dataset of ambient air O_3 concentration estimates with high temporal and spatial resolution (hourly concentrations in 500 to 1700 census tracts) for each of 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 O_3 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).

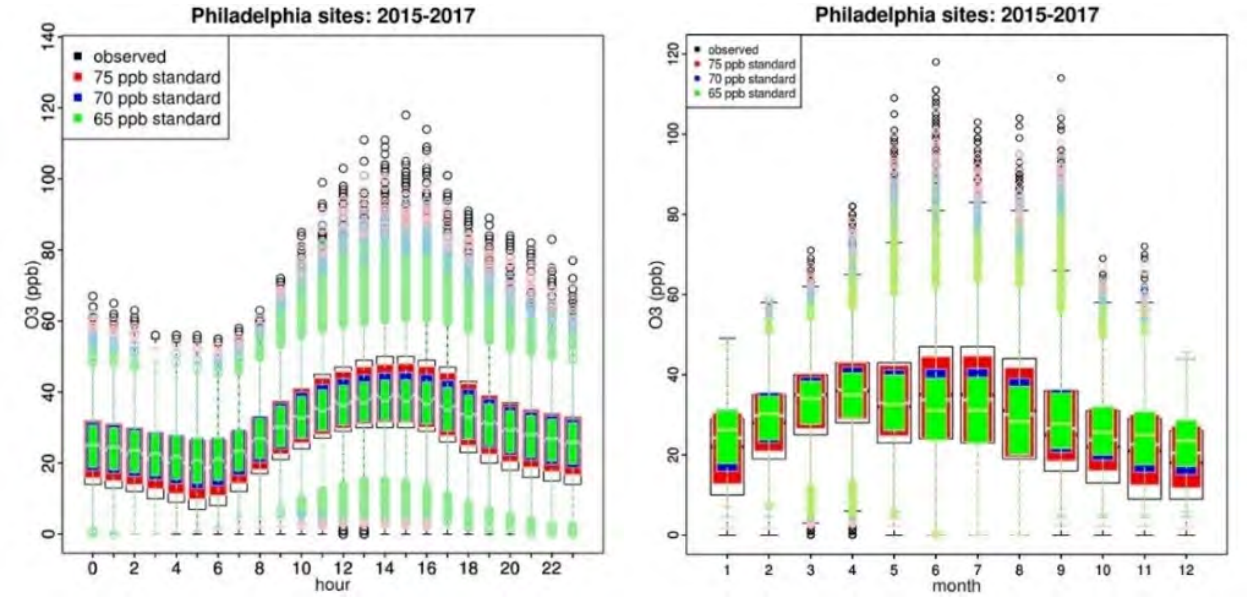

Figure 3D-6. Hourly O_3 distributions by hour-of-day (left panel) and month (right panel) at ambient air monitoring sites in Philadelphia for observed air quality (black), air quality adjusted to meet the current standard (70 ppb, blue) and two other design values (75 ppb, red; and 65 ppb, green). From PA, 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 O_3 concentrations during the during late morning/afternoon hours and during spring/summer months. In addition, the upper end of the O_3 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 O_3 hours-of-the day and represented by the data points that extend beyond the whiskers of the boxplots. Further, the

overall pattern flattens when decreasing the level of the O_3 standard, considering both the diurnal and monthly distributions. Regarding the diurnal pattern, O_3 increases during early morning hours are associated with VOC-limited and NO_X titration conditions near NO_X sources during rush-hour periods. Lower O_3 concentrations in the winter months result from lower solar insolation rates and a reduction in total photochemical activity. See PA, Appendix 3C (Section 3C.7.2 and Figures 3C-67 through 3C-82) for details for temporal characteristics of all eight study areas.

We also evaluated the hourly O_3 concentrations by considering the overall shape of the concentration distribution using the census-tract resolution interpolated data. Even though both the temporal and spatial attributes may be conflated in such a presentation, a histogram can be useful in illustrating important features of the distribution (e.g., skewness, kurtosis, upper percentile tails) that may be influential in estimated exposures and risks. For example, Figure 3D-7 illustrates the overall shape[48] of the hourly concentration distribution in each of the eight study areas for the air quality scenario just meeting the current standard. The distribution for all study areas are skewed to the right, generally representing a lognormal form.

There are notable differences across the collection of study areas. For example, the distributions for Boston, Dallas, Philadelphia, and Sacramento are slender (i.e., leptokurtic), showing much higher peaks around the mean value, relative to the other four study areas, Atlanta, Detroit, Phoenix, and St. Louis which exhibit relatively flatter (i.e., platykurtic) distributions, and the latter three of which, show an increased frequency of upper percentile concentrations. Phoenix, in particular, exhibits the greatest right-most shift in the hourly O_3 concentration distribution and would reflect other areas of the U.S. having a similar distribution of ambient air O_3 concentrations. Also, there are only limited instances of hourly O_3 concentrations >70 ppb in all study areas for the air quality scenario just meeting the current standard (Figure 3D-7). This is consistent with recent (unadjusted) ambient air monitoring data, whereas hourly O_3 concentrations are rarely at or above 100 ppb when design values are ≤70 ppb (i.e., <0.02% frequency; see Appendix 2A, Table 2A-4). This is important to note because these distinct features of the O_3 concentration distribution, along with the spatial and temporal intersection of concentrations with population demographics and activity patterns, play an important role in contributing to variation in the estimated population exposures and risks 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 O_3 concentrations relative to the occurrence of low O_3 concentrations).

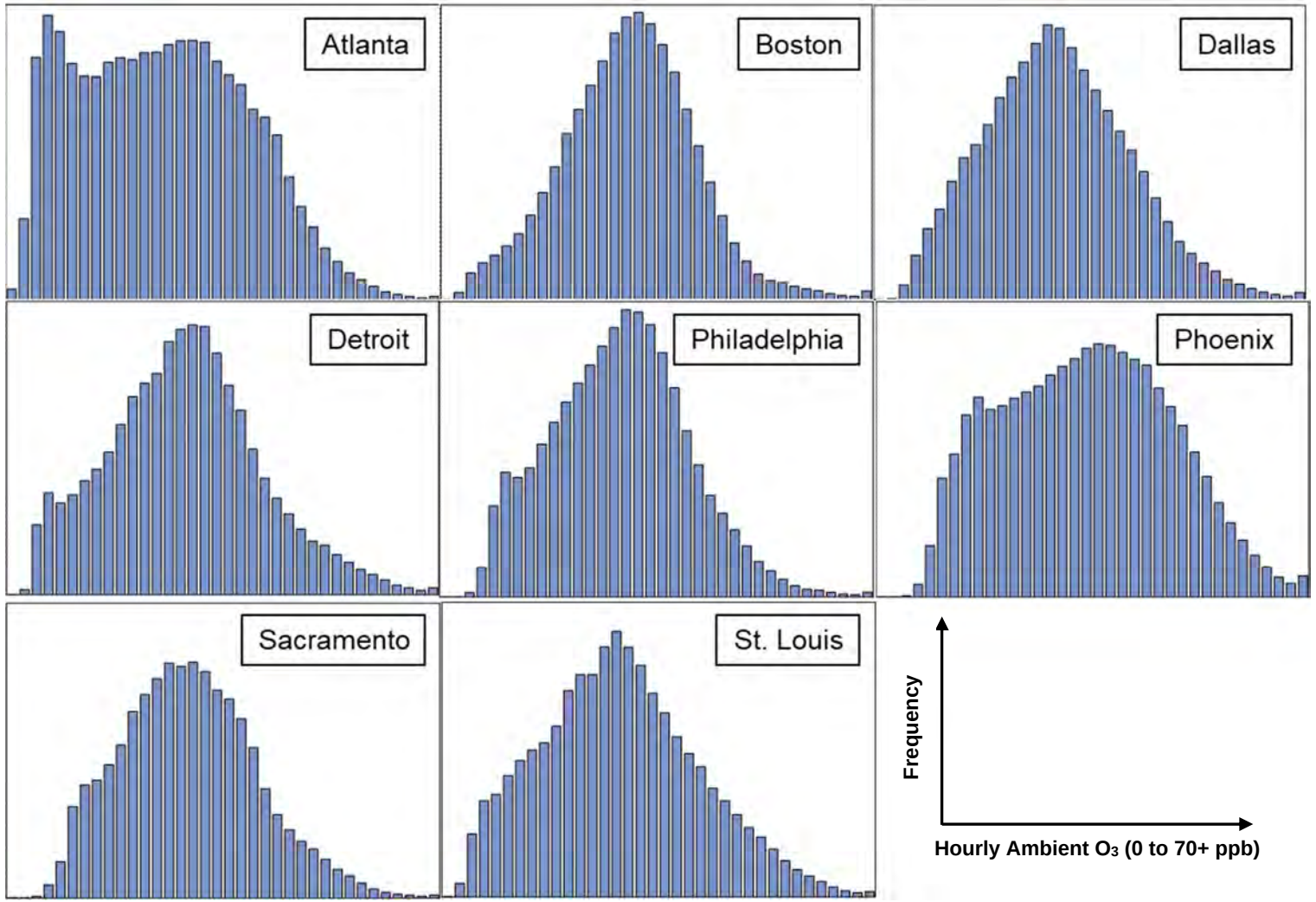

Figure 3D-7. Histograms of hourly O_3 concentrations (ppb, x-axis) for the air quality scenario just meeting the current O_3 standard in the eight study areas. The x-axis midpoint concentrations range from 0 to 70 ppb, in 2 ppb increments (rightmost, maximum histogram bar for all study areas represents the frequency of all hourly concentrations >70 ppb).

Regarding spatial variability, Figure 3D-8 displays census tract design values for each of the three air quality scenarios in Philadelphia. A decline in the highest ambient air O_3 concentrations is predicted across the study area when considering air quality scenarios at lower design values.

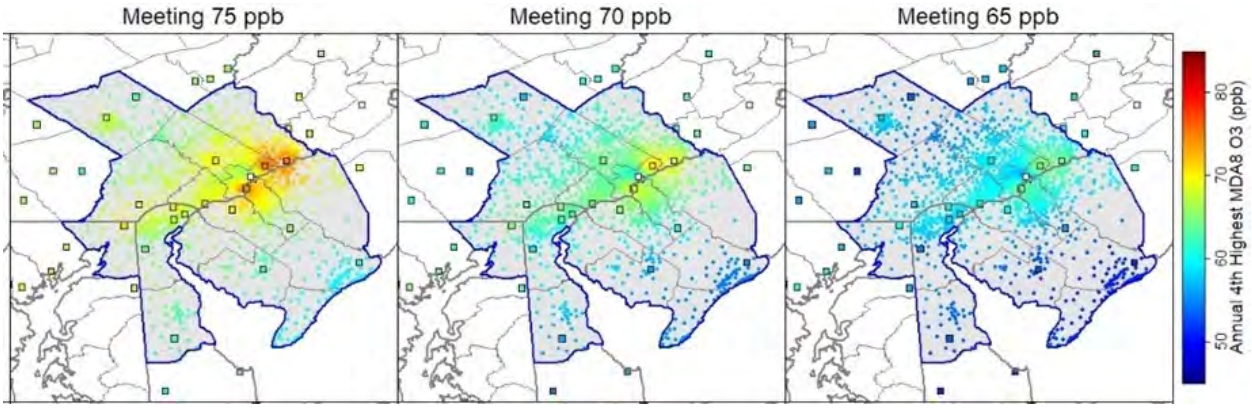

Figure 3D-8. Calculated design values for census tracts in the Philadelphia study area, derived from a VNA interpolation of CAM_X/HDDM adjusted O_3 concentrations. Figure modified from PA, Appendix 3C, Figure 3C-99.

3D.2.4 Meteorological Data

Temperature data are used by APEX in selecting human activity data and in estimating air exchange rates (AERs) for indoor residential microenvironments (MEs). When developing profiles, APEX uses temperature data from the closest weather station to each Census tract. Hourly surface temperature measurements were obtained from the National Oceanic and Atmospheric Administration (NOAA) Integrated Surface Hourly (ISH) data files.[49] The weather stations used for each study area are given in Table 3D-8, along with general locations provided in Figure 3D-2 to Figure 3D-5.

In general, the occurrence of missing temperature data was limited to a few hours per year. Missing hourly temperature data were estimated by the following procedure. Where there were consecutive strings of missing values (data gaps) of 9 or fewer hours, missing values were estimated by linear interpolation between the observed values at the ends of the gap. Remaining missing values at a meteorological station were estimated by fitting linear regression models for each hour of the day, with each of the other monitors, and choosing the model which maximizes R^2, for each hour of the day, subject to the constraints that R^2 be greater than 0.40 and the number of regression data values (days) is at least 100. If there no suitable regression models to 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/*

interpolation between the valid values at the ends of the gap. Any remaining missing values were replaced with the value at the closest station for that hour. Because there were limited instances of missing data, there were negligible differences between the statistically filled and the original temperature data with missing values.

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	*na*	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

Multiple unique APEX input files are used for the current exposure and risk analyses, one for each year and study area, and in the following two formats:

- *METdataCSA[number]Y[year].txt*: meteorological station IDs, hour of day, hourly temperature (°F) for each meteorological station, by study area and year
- *METlocsCSA[number]Y[year].txt*: meteorological station IDs, latitudes and longitudes, start and stop dates of temperature data

3D.2.5 Construction of Human Activity Pattern Sequences

Exposure models use human activity pattern data to estimate exposure to pollutants. Different human activities, such as outdoor exercise, indoor reading, or driving a motor vehicle can lead to different pollutant exposures, intakes and doses. This may be due to differences in the pollutant concentration in the varied locations where different activities are performed as well as to differences in the energy expended in performing the activities (because energy expended influences inhalation and thus may influence pollutant intake). To model exposures to ambient air pollutants, it is critical to have information on the locations where people spend time and the activities performed in such locations. The following subsections describe the activity pattern data, population commuting data, and the approaches used to simulate where individuals might be and what they might be doing.

After the basic demographic variables are identified by APEX for a simulated individual in the study area, values for the other variables are selected as well as the development of the activity patterns that account for the places the simulated individual visits and the activities they perform. The following subsections describe the population data we used in the assessment to assign key features of the simulated individuals, and approaches used to simulate the basic physiological functions important to the exposure estimates for this exposure and risk analysis.

3D.2.5.1 Consolidated Human Activity Database

The Consolidated Human Activity Database (CHAD) provides time series data on human activities through a database system of collected human diaries, or daily time location activity logs (U.S. EPA, 2019c). The purpose of CHAD is to provide a basis for conducting multi-route, multi-media exposure assessments (McCurdy, 2000). The data contained within CHAD come from multiple surveys with variable, study-specific structure (e.g., real time minute-by-minute recording of diary events versus a recall method using time-block-averaging). Common to all of the peer-reviewed studies, individuals provided information on their locations visited and activities performed for each surveyed day. Personal attribute data for the surveyed individuals, such as age and sex, are included in CHAD and are used as variables to link to the population data. The latest version of CHAD contains data for nearly 180,000 individual diary days. Most of the CHAD data are from studies conducted since 2000, several of which are newly included or

updated since the 2014 HREA.[50] Table 3D-9 provides the survey study information including the geographic coverage, year, and the number of diaries available for use by APEX.[51]

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".

Three standard APEX input files are used for the current exposure and risk analyses to create the activity pattern profiles for all simulated individuals.

- *CHADEvents_060419A.txt:* CHAD ID, clock hour (hhmm), duration of event (minutes), CHAD activity code, and CHAD location code, serving as a daily sequence of locations visited, activities performed, and their duration
- *CHADQuest_060419A.txt*: CHAD ID, day-of-week, sex, race, employment status, age, maximum daily temperature, average temperature, occupation, missing time (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

provides the number of people in the labor force, which were stratified by sex/age/tract, considering both civilian workers and workers in the Armed Forces. The data were stratified by sex and age group and were processed so that each sex-age group combination is given an employment probability fraction (ranging from 0 to 1) within each census tract. Children under 16 years of age were assumed to be unemployed. One national-based APEX input file is used for the current exposure and risk analyses as follows:

- *Employment_US_2010.txt*: census tract IDs, employment probabilities (in fractional form), stratified by 13 age groups.[53]

3D.2.5.3 Assignment of Activity Pattern Data to Individuals

Once APEX identifies the basic personal attributes of a simulated individual (section 3D.2.2) and daily air temperatures (section 3D.2.4), activity pattern data obtained from CHAD (section 3D.2.5.1) are then selected based on age, sex, temperature category, and day of the week. These attributes are considered first-order attributes in selecting CHAD diaries when modeling human exposures (Graham and McCurdy, 2004). The particular locations people visit, amount of time spent there, and frequency of these visits can also be influenced by local weather conditions. When considering seasonal temperature ranges (i.e., cold/not cold during cool months; hot/not hot during warm months), (Graham and McCurdy, 2004) found daily maximum temperature (DMT) influences time spent outdoors. Participation rate and amount of time outdoors was found lower on cold DMT days compared to the other three temperature categories, while the participation rate on hot days was less than that on not hot days. Because of these findings, we use a similar DMT range (<55, 55-83, ≥84 °F) to select activity pattern data that best match each study area's meteorological data for every day of the simulated individual's exposure profile. This information for the selecting of activity pattern data is found in the following APEX input file, varying by study area and simulation year:

- *Functions_O3_CSA[number]_040219.txt*: probabilities and interval definitions associated with a few input variables. For activity diary selection - day of week intervals (weekend or weekday) by three temperature ranges.

While there may be other important attributes that may influence activity patterns (e.g., 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.

that may be of interest in modeling an individual's activities to the CHAD data. This is largely because CHAD is a compilation of data collected from numerous individual activity pattern studies conducted over several decades, many of which had a unique survey design. As a result, there is a varying amount of missing personal attribute data for the surveyed individuals in CHAD. For instance, there are only a limited number of CHAD diaries with survey-requested health information (e.g., the health status of respondents). Specifically regarding whether or not a survey participant had asthma, very few of the available diaries have either a 'yes' or 'no' response to this health condition. When considering the 177,210 diary days used by APEX, there are only 4,935 diary days from individuals having asthma (of which 3,133 are children ages 5-18),[54] representing a small fraction of the CHAD data. On its own, having approximately 5,000 diaries may appear to be a large number of diaries, however, following a grouping of the diaries by their first-order attributes when developing simulated profiles (e.g., age, sex, day-of-week, etc., daily temperature), would likely result in fewer than 100 diaries available for simulating a single day for a particular individual. Accordingly, the selection of diaries to use for APEX-simulated individuals does not consider health status (i.e., any diary is used, regardless of whether the individual indicated they did or did not have asthma, or that information was unknown).

This restriction in the number of diaries from individuals having asthma is not considered to be a significant limitation for estimating exposures for simulated individuals with asthma. In general, modeling people with asthma similarly to healthy individuals (i.e., using the same time-location-activity profiles) is supported by the activity analyses reported by van Gent et al. (2007) and Santuz et al. (1997). Other researchers, for example, Ford et al. (2003), have shown significantly lower leisure time activity levels in asthmatics when compared with individuals who have never had asthma. Based on these inconsistent findings, we evaluated this issue in the 2014 HREA and, using the available activity pattern data in the CHAD database, we compared participation in afternoon outdoor activities at elevated exertion levels among people having asthma, people not having asthma, and unknown health status (2014 HREA, Appendix G, section 5G-1.4). The 2014 HREA analysis indicated health status had little to no impact on the participation in afternoon activities at elevated exertion levels. A similar analysis was repeated here to include the diary data currently used by APEX, not just those that would be included in the simulations for the 2014 HREA (i.e. ~50,000 diaries).

Of interest in this current risk and exposure analysis are instances when individuals experience their highest O_3 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.

when individuals spend time outdoors, particularly during the afternoon hours (2014 HREA, Appendix 5G section 5G-2). To prepare the APEX activity dataset for analysis here, afternoon hours were characterized as the time between 12 PM and 8 PM and only those persons that spent some time outdoors were retained. As is done by APEX in simulating individuals, level of exertion was estimated by sampling from the specific METs distributions assigned for each person's activity performed. Then, we identified activities having a METs value of greater than three as instances where a person was at moderate or greater exertion levels (U.S. DHHS, 1999). Afternoon outdoor time was then stratified by exertion level, summed for two study groups of interest (i.e., children and adults), and presented in percent form within Table 3D-10.

Regarding the diaries for children of interest for these exposure and risk analyses (ages 5-18), about 13% are from an individual having asthma, 48% are from those who do not have asthma, and the remaining portion of children's diaries have unknown health status. About 1% of CHAD diaries for adults are from individuals with asthma and about 11% are from those who do not have asthma. Far fewer children's diaries are from persons whose asthma status is unknown (40%) compared to adults (88%), and the proportions are smaller still in terms of the total available person-days. On average, about 42% of all children having known asthma status spent some afternoon time outdoors, and the percent is actually higher for children with asthma (48.4%) than for children not having asthma (40.5%). About half of the adults whose asthma status was known spent afternoon time outdoors with a participation rate generally similar for adults having asthma and adults not having asthma. Participation in outdoor events for children having unknown asthma status varied little from that of persons with known asthma status. Contrary to this, there were fewer adults with unknown asthma status that participated in outdoor events (29%) when compared to those having known asthma status.

The amount of afternoon time spent outdoors by the persons that did so varied little across the two study groups and two asthma classifications. Children, on average, spend approximately 2¼ hours of afternoon time outdoors, 80% of which is at a moderate or greater exertion level, regardless of their asthma status. For children whose asthma status is unknown, slightly more afternoon time is spent outdoors (about 150 minutes) but the percent of afternoon time at moderate or greater exertion levels is slightly lower (about 69%). As seen with children, adults spend approximately 2¼ hours of afternoon time outdoors regardless of their asthma status. However, the percent of afternoon time at moderate or greater exertion levels for adults (about 55%) is lower than that observed for children.

Based on this updated analysis and additional comparisons of CHAD diary days with literature reported values of outdoor time participation at varying activity levels (see 2014 HREA), there are strong similarities in outdoor time, outdoor event participation, and activity levels achieved among the two study groups and with those reported in independent studies of

people with asthma. Thus, we conclude the use of any CHAD diary, regardless of known/unknown asthma status, is reasonable for purposes of simulating people with asthma in this exposure and risk analysis.

Table 3D-10. Comparison of time spent outdoors and exertion level by asthma status for children and adult diaries used by APEX.

	CHAD: Children (5 to 18) [A]			CHAD: Adults (>18) [B]		
Has Asthma?	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.

We also evaluated how temperature influences the amount of afternoon time spent outdoors while at moderate or greater exertion by children (5-18 years) and adults (19-90 years). This differs from analyses in Graham and McCurdy (2004) in which all outdoor time at any exertion level was evaluated and the number of diary days available in CHAD was much less at that time (~23,000 diary days). Also, in this current analysis, each CHAD/APEX diary day was grouped by both DMT (<55, 55-83, or ≥84 °F) and day-type (weekday or weekend). Total available diary days for each of these groups is provided in Table 3D-11. Then, afternoon time outdoors (12:00 PM to 8:00 PM) was summed and place into one of five hourly groupings (0, 0-≤½, ½-≤2, 2-≤4, and >4 hours per day) and the percent of diary days in each group was calculated, the results of which are provided in Figure 3D-9 for children and adults.

Overall, the greatest proportion of diary days would be characterized as not having any afternoon time spent outdoors at moderate or greater exertion (46 - 76%), with adults consistently having a greater frequency of not spending afternoon time outdoors than children (Figure 3D-9). Afternoon time outdoors at moderate or greater exertion for both children and adults is less likely to occur on cold days (DMT <55 °F), with progressively increased frequency of outdoor time with increasing temperatures for both day-types. Children are more frequently spending afternoon time outdoors at elevated exertion levels, particularly when considering the largest duration assessed (e.g., for durations of time outdoors ≥2 hours, the percent of child diary days is greater than adults by a factor of 1.3 to 2.7).

Table 3D-11. Number of diary days in CHAD for children and adults, grouped by 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.				

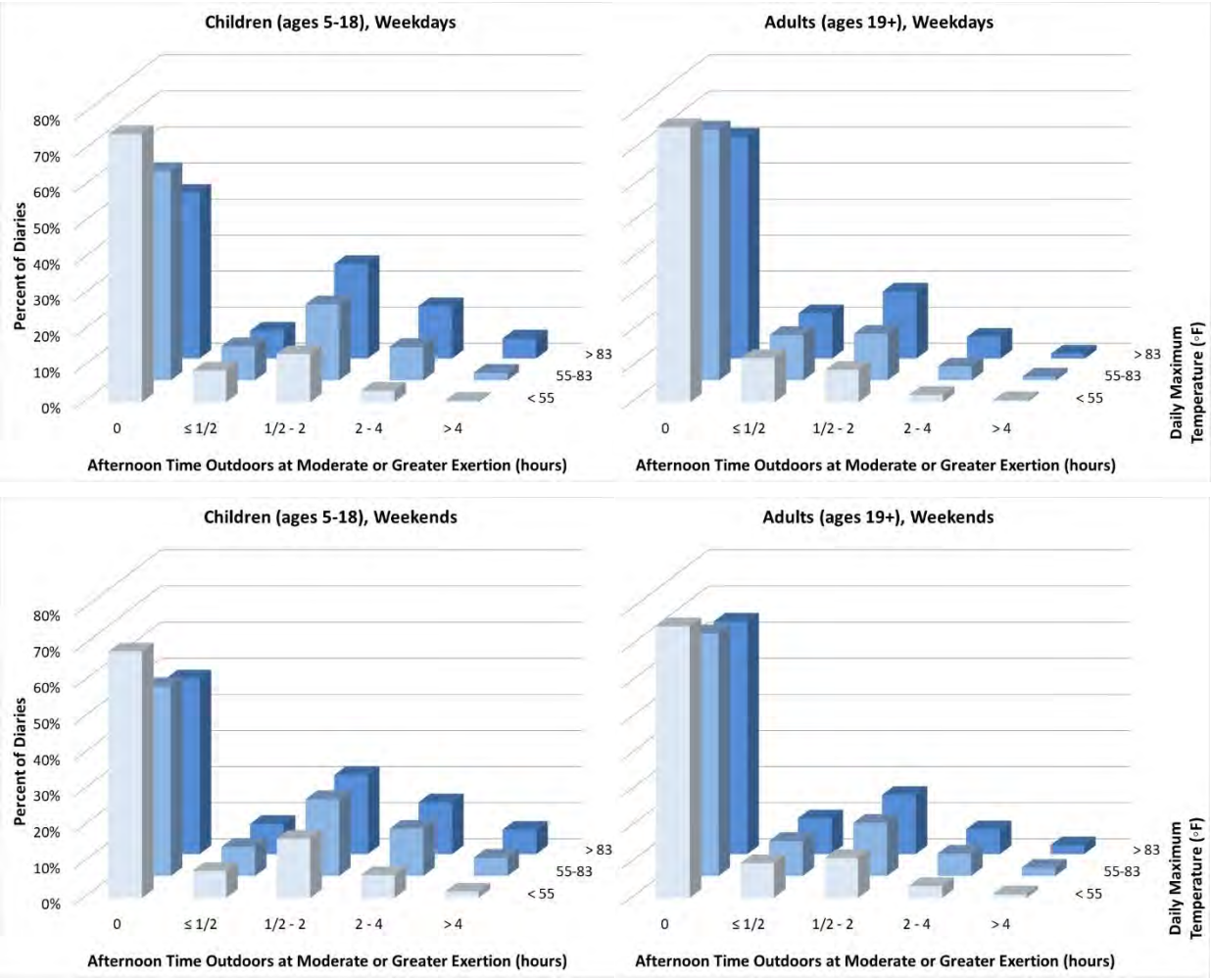

Figure 3D-9. Percent of children (5-18 years) and adults (19-90 years) having afternoon time outdoors while at moderate or greater exertion, categorized by daily maximum temperature (°F) and time (hours/day) groups.

3D.2.5.4 Method for Longitudinal Activity Pattern Sequence

In order to estimate population exposure over a full year, a year-long activity sequence needed to be created for each simulated individual based on CHAD, which is largely a cross-sectional activity database of 24-hr records. On average, the typical surveyed subject provided in CHAD has about two days of diary data. For this reason, the construction of a season-long activity sequence for each individual requires some combination of repeating the same data from one subject and using data from multiple subjects. The best approach would reasonably account for the day-to-day and week-to-week repetition of activities common to individuals and recognizing even these diary sequences are not entirely correlated, while maintaining realistic variability among individuals comprising each study group.

APEX provides three methods of assembling composite diaries: a basic method, a diversity and autocorrelation (D&A) method, and a Markov-chain clustering (MCC) approach. We have selected the diversity and autocorrelation (D&A) method for this assessment based on our consideration of the assessment objectives, an evaluation of differences in results produced by the three methods, and consideration of flexibility provided by each approach with regard to specifying key variable values, as discussed below. First a brief description of each method is provided below.

The basic method involves randomly selecting an activity diary for the simulated individual from a user-defined diary pool (e.g., age, sex). While the method is adequate for estimating a mean short-term exposure for a population as a whole, it is less useful for estimating how often individuals in a population may experience peak O_3 exposures over a year.

The D&A method is a complex algorithm for assembling longitudinal diaries that attempts to realistically simulate day-to-day (within-person correlations) and between-person variation in activity patterns (and thus their exposures to the extent they are influenced by spatial and temporal variability in ambient air and microenvironmental O_3 concentrations). This method was designed to capture the tendency of individuals to repeat activities, based on reproducing realistic variation in a key diary variable, which is a user selected function of diary variables. The method targets two statistics: a population diversity statistic (*D*) and a within-person autocorrelation statistic (*A*). The *D* statistic reflects the relative importance of within and between-person variance in the key variable. The *A* statistic quantifies the lag-one (day-to-day) key variable autocorrelation. Values of *D* and *A* for the key variable are selected by the model user and set in the APEX parameters file, and the method algorithm constructs longitudinal diaries that preserve these parameters. Further details regarding this methodology can be found in Glen et al. (2008).

The Markov-chain clustering (MCC) approach is similarly complex in attempting to recreate realistic patterns of day-to-day variability. First, cluster analysis is employed to divide

the daily activity pattern records into three groups based on time spent in, for example, five microenvironments: indoor-residence, other indoors, outdoor-near roads, other outdoors, and inside vehicles. For each simulated individual, a single time-activity record is randomly selected from each cluster. Then the Markov process determines the probability of a given time-activity pattern occurring on a given day based on the time-activity pattern of the previous day and cluster-to-cluster transition probabilities (and are estimated from the available multi-day time-activity records), thus constructing a long-term sequence for a simulated individual. Details regarding the MCC method and supporting evaluations are provided in U.S. EPA (U.S. EPA, 2019a, U.S. EPA, 2019b).

Che et al. (2014) performed an evaluation of the impact of the three APEX methods on $PM_{2.5}$ exposure estimates. As expected, little difference was observed across the methods with regard to estimates of the mean exposures of simulated individuals. Differences were observed, however, in the number of multiday exposures exceeding a selected benchmark concentration. With regard to the number of simulated individuals experiencing 3 or more days above benchmark concentrations, the MCC method estimates were approximately 12-14% greater than either the random or D&A methods. For the number of persons experiencing at least one exposure of concern, however, the MCC method estimates were approximately 4% lower than those of the other two methods. For additional context, we note that, using all methods, there is an order of magnitude difference in the number of persons exposed at least once versus three or more times, indicating that, overall, the occurrence of simulated multiday exposures are rare events regardless of method selection.

Che et al. (2014) concludes that while the MCC method produces a higher number of multiday exposures, there remains a question whether the MCC method has greater accuracy relative to the other two methods. We note this conclusion applies to both the estimations of single day and multiday exposures, as there is an inverse relationship between the two when simulating exposures using APEX and a finite set of activity pattern data. Thus, the MCC method produces a smaller number of single day exposures above benchmarks relative to the other two methods, estimations also subject to a degree of uncertainty.

In the absence of having a robust data set (e.g., multiday/week diary data from a random population) to better evaluate the accuracy of any of the methods, we considered selection of the longitudinal approach for this assessment from a practical perspective, guided by a balancing of the single day and multiday exposures that can be estimated by each method. In so doing, we selected the D&A approach, recognizing that the D&A method allows for flexibility in the selection of the key influential variable and its setting values, and also the ability to directly observe the impact of changes to these values on model outputs.

The key variable selected for this exposure and risk analysis is the amount of time an individual spends each day outdoors, as that is the most important determinants of exposure to high levels of O_3 (2014 HREA, Appendix 5G, section 5G-2). In their evaluation, Che et al. (2014) varied the values of *D* and *A* for this variable to determine the impact to estimated exposures. Compared to their base level simulation (i.e., *D*=0.19 and *A*=0.22), increasing both D and A by 100% increased the number of persons having at least three exposures above the selected benchmark by about 4%, while also reducing the percent of persons experiencing at least one day above benchmarks by less than 1% (Che et al., 2014). In recognizing uncertainty in the parameterization of *D* and *A* (i.e., based on Xue et al., 2004) a limited field study of a small subset of the population, children 7-12) and that the Che et al., 2014 base level simulation D&A values produced a lower estimate of repeated exposures compared with the MCC method, we have used values of 0.5 for D and 0.2 for A for all ages to potentially increase representation of multiday exposures without significantly reducing the percent of the population experiencing at least one day at or above benchmark concentrations.

3D.2.6 Microenvironmental Concentrations

In APEX, exposure of simulated individuals occurs in microenvironments (MEs) rather than assuming people are exposed continuously and consistently to ambient air. To best estimate personal exposures, it is important to maintain the spatial and temporal sequence of MEs people inhabit and to appropriately represent the time series of concentrations that occur within them. Two methods are available in APEX for calculating pollutant concentrations within MEs: a mass balance model and a transfer factor approach. In both approaches, ME concentrations depend on the ambient (outdoor) air O_3 concentrations and ambient air temperatures, as well as statistical distributions to parameterize the variables used by each approach. Further, the statistical distributions of some of the key variables depend on values of other variables in the model. For example, the distribution of air exchange rates inside an individual's residence depends on the type of heating and air conditioning present, which are also probabilistic inputs to the model. The value of a variable can be set as a constant for the entire simulation (e.g., house volume remains identical throughout the exposure period), or APEX can sample a new value hourly, daily, or seasonally from user-specified statistical distributions. APEX also allows the user to specify diurnal, weekly, or seasonal patterns for certain ME parameters. Details regarding the two methods can be found in (U.S. EPA, 2019a, U.S. EPA, 2019b) and are briefly described below.

The mass balance method, used for the indoor MEs, assumes that an enclosed microenvironment (e.g., a room within a home) is a single well-mixed volume in which the air concentration is approximately spatially uniform (Figure 3D-10). The concentration of an air pollutant in such a microenvironment is estimated using (1) inflow of air into the

microenvironment, (2) outflow of air from the microenvironment, (3) removal of a pollutant from the microenvironment due to deposition, filtration, and chemical degradation, and (4) emissions from sources of a pollutant inside the microenvironment (not used for this exposure and risk analysis). Considering the microenvironment as a well-mixed fixed volume of air, the mass balance equation for a pollutant in the microenvironment can be written in terms of concentration as follows in Equation 3D-7:

$$\frac{dC(t)}{dt} = \dot{C}_{in} - \dot{C}_{out} - \dot{C}_{removal} \qquad \text{Equation 3D-7}$$

where,

$C(t)$ = Concentration in the microenvironment at time t

$\dot{C}_{in}$ = Rate of change in $C(t)$ due to air entering the microenvironment

$\dot{C}_{out}$ = Rate of change in $C(t)$ due to air leaving the microenvironment

$\dot{C}_{removal}$ = Rate of change in $C(t)$ due to all internal removal processes

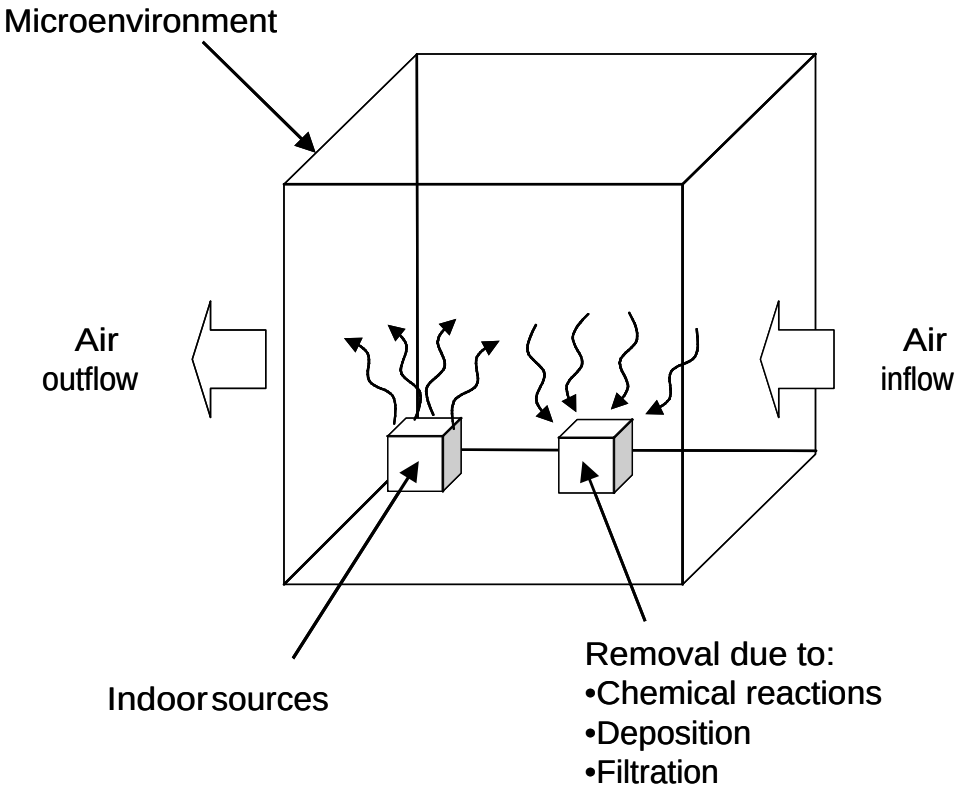

Figure 3D-10. Illustration of the mass balance model used by APEX to estimate concentrations within indoor microenvironments.

The factors model (used for the outdoor and inside vehicle MEs) is simpler than the mass balance model. In this method, the value of the ME concentration is not dependent on the ME concentration during the previous time step. Rather, this model uses Equation 3D-8 to calculate the concentration in an ME from the ambient air quality data:

$$C_{mean} = C_{ambient} \times f_{proximity} \times f_{pollutant} \quad \text{Equation 3D-8}$$

where,

C_{mean} = Mean concentration over the time step in a microenvironment (ppm)
$C_{ambient}$ = The concentration in the ambient (outdoor) air (ppm)
$f_{proximity}$ = Proximity factor (unitless)
$f_{pollutant}$ = fraction of ambient air pollutant entering microenvironment (unitless)

Based on findings from the 2014 HREA, we have specified seven MEs to simulate in this assessment, largely based on two factors: the expectation of a particular ME leading to exposures of interest and the availability of factors needed to reasonably model the ME. The 2014 HREA indicated that high (≥50 ppb) 8-hr daily maximum O_3 exposures occurred while individuals spent much larger amounts of afternoon time outdoors compared with those experiencing low (< 50 ppb) exposure levels (2014 HREA, Appendix 5G, Figure 5G-5). Given that finding and the objective for the exposure assessment (i.e., understanding how often and where maximum O_3 exposures occur), we recognized the added efficiency of minimizing the number of MEs compared to that done in the 2014 HREA (i.e., 28 microenvironments), particularly reducing the number of lower-exposure indoor MEs that were parameterized and included at that time.

Accordingly, we aggregated the number of MEs to seven and estimate exposures of ambient air origin that occur within a core group of indoor, outdoor, and inside vehicle MEs. Four indoor MEs (indoor-residence, indoor-restaurant, indoor-school, and indoor-other[55]) were modeled based on having specific air exchange rate data available for each (section 3D.2.6.1). All outdoor locations were assumed to have O_3 concentrations equivalent to ambient air, however there were two MEs used to do so, distinguished by whether or not they occurred near roads. The outdoor near road ME was modeled separately due to the expected decrease in concentrations occurring in that ME relative to that of ambient air concentrations. And finally, an inside-vehicle ME was modeled based on the expectation that it would lead to some instances of relatively lower exposures compared with ambient air concentrations. Table 3D-12 lists the 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.

each. The variables and their associated parameters used to calculate ME concentrations are summarized in subsequent sections below.

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.

The seven microenvironments were mapped to the 115 CHAD locations[56] because using such a large number of MEs would go well beyond the practical scale needed for the exposure and risk analyses. Note that the ambient air concentration used in calculating ME concentration for each exposure event varies temporally and spatially. For example, commuters (i.e., employed individuals who do not work at home) are assigned to either their home tract or work tract concentration, depending on whether the population probabilities and commuting data base produce either a home or work event. Additionally, depending on the particular ME (i.e., other than home or work), the mapping of CHAD locations to the seven MEs also uses an identifier that designates the relative location in the air quality surface from which the ambient air concentration (used to calculate the ME concentration) is selected. For this assessment, such locations would include the Census tract for a simulated individual's home (H), work (W), near work (NW), near home (NH), last (L, either NH or NW), other (O, average of all), or unknown (U, last ME determined) location. Specific designations are provided in the APEX ME mapping file, with selection based on known factors and professional judgement. For example, when an individual is in their home, the ambient air concentration in the home tract is used to calculate their ME concentration. When the individual is at work, the tract the individual commuted to is used to calculate their ME concentration. Travel inside vehicles used the ambient air concentration data from the tract used to calculate the prior ME concentration. Most other MEs (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.

Status attribute variables are also important in estimating ME concentrations, and can include, but are not limited to, housing type, whether the house has air conditioning, and whether the car has air conditioning. Because outdoor MEs are expected to contribute the most to an individuals' highest O_3 exposure (and potential health risk) and the status attribute variables pertain to indoor MEs, the setting of these particular variables will have limited impact to the exposure and risk results generated here. In this assessment, a number of temperature ranges are used in selecting the particular distribution for estimating air exchange rates (AERs). Maximum daily temperature is also used in diary selection to best match the study area meteorological data for the simulated individual (Graham and McCurdy, 2004) and air conditioning use.

Multiple APEX input files (the first and third in the list below), of the same general format, are used for estimating ME concentrations in each study area. A single APEX ME mapping file is used for all study areas. These ME input files contain the parameter settings for all variables described in the subsections that follow.

- *ME_descriptions_O3_7MEs_CSA[number].txt*: defines ME calculation method, conditional variables used (e.g., temperature categories – see functions file), distribution type, distribution parameters (mean, standard deviation, minimum, maximum) for AERs, decay rates, proximity factors, and PE fractions used to estimate O_3 in 7 MEs.
- *Microenvironment_mappings_07_MEs.txt*: maps 115 CHAD locations to the 7 APEX MEs and assigns the tract-level ambient air concentrations to use for each location. Contains CHAD location code, CHAD description, APEX ME number, and ambient air concentration location identifier
- *Functions_O3_CSA[number]_040219.txt:* variables used for selecting AER - air conditioning (A/C) prevalence (home has A/C, does not have A/C) by five temperature ranges for air exchange rate (<50, 50-67, 68-76, 77-85, or >85 °F). (see section 3D.2.6.1)

3D.2.6.1 Indoor Microenvironments

As described above, all four indoor MEs (indoor-residential, indoor-restaurant, indoor-school, and indoor-other) were modeled using a mass balance model. The three variables used to calculate ME concentrations, air exchange rates (section 3D.2.6.1.1), air conditioning prevalence (section 3D.2.6.1.2), and ozone removal rate (section 3D.2.6.1.3) are described below.

3D.2.6.1.1 Air Exchange Rates

Distributions of air exchange rates (AERs, hr^{-1}) for the indoor residential ME were developed using data from several studies. The analysis of these data and the development of most of the distributions used in the modeling were originally described in detail in the 2007 exposure analysis (U.S. EPA (2007a), Appendix A) and updated in the 2014 HREA (see Appendix 5E). Briefly, AER distributions for the residential microenvironments depend on the type of air conditioning (A/C) and on the outdoor temperature, among other variables for which we do not have sufficient data to estimate. AER distributions were found vary greatly across

cities, A/C types, and temperatures, so that the selected AER distributions for the modeled cities should also depend on these attributes. For example, the mean AER for residences with A/C ranges from 0.38 in Research Triangle Park, NC at temperatures > 25 ºC upwards to 1.244 in New York, NY considering the same temperature range (2014 HREA, Appendix 5E). For each combination of A/C type, city, and temperature with a minimum of 11 AER values, exponential, lognormal, normal, and Weibull distributions were fit to the AER values and compared. Generally, the lognormal distribution was the best-fitting of the four distributions, and so, for consistency, the fitted lognormal distributions are used for all the cases.

There were a number of limitations in generating study-area specific AER stratified by temperature and A/C type. For example, AER data and derived distributions were available only for selected cities, and yet the summary statistics and comparisons demonstrate that the AER distributions depend upon the city as well as the temperature range and A/C type. As a result, city-specific AER distributions were used where possible; otherwise staff selected AER data from a similar city. Another important limitation of the analysis was that distributions were not able to be fitted to all of the temperature ranges due to limited number of available measurement data in these ranges. A description of how these limitations were addressed can be found in the 2014 HREA, Appendix 5E. The AER distributions used for the exposure modeling are given in Table 3D-13 (Residences with A/C) and Table 3D-14 (Residences without A/C).

Table 3D-13. Air exchange rates (AER, hr^{-1}) for indoor residential microenvironments with 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	

Table 3D-14. Air exchange rates (AER, hr^{-1}) for indoor residential microenvironments 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	

The AER distribution (hr^{-1}) used for indoor restaurants in all study areas is a fitted lognormal distribution, having a geometric mean = 3.712, geometric standard deviation = 1.855 and bounded by the lower and upper values of the sample data set {1.46, 9.07}. This distribution was developed using data from Bennett et al. (2012) who measured AER in restaurants (details on derivation provided in the 2014 HREA, Appendix 5E). The AER distribution (hr^{-1}) used for

indoor schools in all study areas is a fitted Weibull distribution,[57] having a threshold (τ) = 0, shape (C) = 1.26, and scale (σ) = 1.75, bounded by a lower and upper range {0, 10}. This distribution was developed from Lagus Applied Technology, 1995, Shendell et al., 2004, and Turk et al., 1989 who measured AER in schools (raw data provided in Table 3D-15).

Table 3D-15. Individual air exchange rate data (hr^{-1}) obtained from three studies used to 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				

The AER distribution (hr^{-1}) used for indoor other in all study areas is a fitted lognormal distribution, having a geometric mean = 0.949, geometric standard deviation = 1.857 and bounded by the lower and upper values of the sample data set {0.30, 4.02}. This distribution was developed using data from Bennett et al. (2012) who measured AER in non-residential buildings (details on derivation provided in the 2014 HREA, Appendix 5E).

3D.2.6.1.2 Air Conditioning Prevalence

The selection of an AER distribution for the indoor residence ME is conditioned on the presence or absence of A/C. We assigned this housing attribute to indoor residential 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

A/C prevalence data were assigned to our study areas where the AHS data best matched our exposure simulation years and or study area. In all study areas and for each year, housing units containing either central or 3 or more room AC were summed, followed by the calculation of the A/C prevalence. If multiple years were available, these data were averaged to generate the final A/C prevalence (unitless) for each study area (Table 3D-16). For the other three indoor MEs (indoor-restaurant, indoor-school, and indoor-other) mechanical ventilation was assumed to be present in all buildings (i.e., A/C prevalence = 1.0).

Table 3D-16. A/C prevalence from US Census American Housing Survey (AHS) data by 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

3D.2.6.1.3 Ozone Decay and Deposition Rates

As done for the 2014 HREA, a distribution for combined O_3 decay and deposition rates was obtained from the analysis of measurements from a study by Lee et al. (1999). This study measured decay rates in the living rooms of 43 residences in Southern California. Measurements of decay rates in a second room were made in 24 of these residences. The 67 decay rates range 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).

yielding a geometric mean of 2.51 hr^{-1} and a geometric standard deviation of 1.53 hr^{-1}. These values are constrained to lie between 0.95 and 8.05 hr^{-1}. This combined O_3 decay and deposition rate distribution was used for all four indoor microenvironments.

3D.2.6.2 Outdoor Microenvironments

As mentioned above, the two outdoor MEs (outdoor-general and outdoor-near road) used the factors approach to estimate ME concentrations. The factors approach uses two variables in combination with ambient air O_3 concentrations: a proximity factor and a factor expressing the fraction of a pollutant entering (PE factor) an ME, and these are discussed below.

Proximity factors are used to adjust ambient air O_3 concentrations, based on the ME location relative to that of the ambient air concentration. For the outdoor-general ME, there is no adjustment used (proximity = 1.0); it is assumed that wherever an individual is outdoors, the individual experiences the ambient air O_3 concentrations for the tract they are present in at that time (e.g., at home, at work, or nearby census tract). For the outdoor-near road ME, a proximity factor is used, recognizing that ambient air concentrations measured away from roadways tend to increase with distance. As done for the 2014 HREA, we employed the distribution for local roads (i.e., a normal distribution {0.755, 0.203}, bounded by 0.422 and 1.0) derived from the Cincinnati Ozone Study (American Petroleum Institute, 1997, Appendix B; Johnson et al., 1995), based on the assumption that most of the outdoors-near-road ozone exposures will occur proximal to local roads (see Table 3D-17 and details below in section 3D.2.6.3).

PE factors are used to adjust for the percent of a pollutant entering a ME. PE factors for the outdoor-general and outdoor-near road MEs, because they are effectively aligned with the ambient air O_3 concentrations, are set equivalent to 1.

3D.2.6.3 Inside-Vehicle Microenvironments

As done for the 2014 HREA, for the in-vehicle ME, proximity and PE factor distributions were obtained from the Cincinnati Ozone Study (American Petroleum Institute, 1997, Appendix B; Johnson et al., 1995). This field study was conducted in the greater Cincinnati metropolitan area in August and September 1994. Vehicle tests were conducted according to an experimental design specifying the vehicle type, road type, vehicle speed, and ventilation mode. Vehicle types were defined by the three study vehicles: a minivan, a full-size car, and a compact car. Road types were interstate highways (interstate), principal urban arterial roads (urban), and local roads (local). Nominal vehicle speeds (typically met over 1-min intervals within 5 mph) were at 35 mph, 45 mph, or 55 mph. Ozone concentrations were measured inside the vehicle, outside the vehicle, and at six fixed-site monitors in the Cincinnati area. Table 3D-17 lists the parameters of the normal distributions developed for proximity and PE factors (both are unitless) for in-vehicle microenvironments used in this exposure and risk analysis.

A daily conditional variable was used to select the three proximity factor distributions to use in estimating the inside-vehicle ME concentrations. The 2015-2017 Vehicle Miles of Travel (VMT) data available from the U.S. Department of Transportation (DOT) were used to generate these daily conditional variables.[59] For local and interstate road types, the VMT for the same DOT categories were used. For urban roads, the VMT for all other DOT road types were summed (i.e., other freeways/expressways, other principal arterial, minor arterial, and collector). Table 3D-18 summarizes the conditional variables used for each study area to select for the proximity factor distribution used to estimate inside-vehicle ME concentrations.

Table 3D-17. Parameter values for distributions of penetration and proximity factors used 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.

Table 3D-18. VMT (2015-2017) derived conditional probabilities for interstate, urban, and local roads used to select inside-vehicle proximity factor distributions in each 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 O_3 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 last 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 FEV_1)[60] along with supporting health evidence from O_3-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 O_3, all but a few of which were available in the last 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 complete six 50-min

[60] There are other respiratory responses resulting from O_3 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.

periods of exercise followed by 10-min rest periods (along with a 35-min lunch/rest period) (ISA, Appendix 3, section 3.1.4.1.1).

For lung function risk, we estimate risk of an O_3-related decrement at or above 10%, 15% and 20%. These sizes of decrements have been used in the risk assessments for the past three reviews, i.e., those completed in 2015, 2008 and 1997 (2014 HREA; U.S. EPA, 2007a, U.S. EPA, 2007b; Whitfield et al., 1996). In the last review, the CASAC concurred with the EPA's use in the 2014 HREA of estimated FEV_1 decrements of ≥15% as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, and an FEV_1 decrement of ≥10% as a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease (Frey, 2014, p. 3).

3D.2.8.1 Comparison to Benchmark Concentrations

For the comparison of simulated exposures to benchmark concentrations that reflect observations from the 6.6-hr controlled human exposure studies, APEX estimates the daily maximum 7-hr average O_3 exposure[61] for every simulated individual, stratified by exertion level at the time of exposure. This indicator was selected based on controlled human exposure studies where reported adverse health responses were associated with exposure to O_3 and while the study subject was exercising.[62] A 7-hr average exposure concentration is more appropriate than using an 8-hr average (as was done for the prior REAs) because it aligns more closely to the 6.6-hr durations of the controlled human exposure studies on which the benchmark concentrations are based.[63] The 7-hr average exposure concentrations experienced by simulated individuals while at moderate or greater exertion (EVR ≥17.32 ± 1.25 L/min-m^2 body surface area; see above section 3D.2.2.3.3) are then compared to the benchmark concentrations.

Benchmark concentrations used in this assessment include O_3 exposure concentrations of 60, 70 and 80 ppb; the same benchmarks used for the 2014 HREA (based on there being no new 6.6-hr controlled human exposure studies that might inform consideration of alternatives). Estimating the occurrence of ambient air-related 7-hr average O_3 exposures at and above these

[61] Only the maximum 7-hr average O_3 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 O_3, 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 O_3 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 O_3 concentrations are now evaluated with a more appropriate exposure and risk metric (i.e., a 7-hr average exposure benchmark).

benchmark levels is intended to provide perspective on the potential for public health impacts of O_3-related health effects observed in human clinical and toxicological studies, but for which available data do not support development of E-R functions, precluding their evaluation in quantitative risk assessments (e.g., lung inflammation, increased airway responsiveness, and decreased resistance to infection), as well as lung function decrements which are currently evaluated in quantitative risk assessments. The 80 ppb benchmark concentration represents an exposure where multiple controlled human exposure studies (of the 6.6-hr, with exercise design) demonstrate a range of O_3-related respiratory effects including lung inflammation and airway responsiveness, as well as respiratory symptoms, in healthy adults. The 70 ppb benchmark concentration reflects a study that found statistically significant decrements in lung function as well as increased respiratory symptoms. The 60 ppb benchmark level represents the lowest exposure level at which statistically significant decrements in lung function, but not respiratory symptoms, have been observed in studies of healthy individuals (see Table 3-2 of PA).[64] This is summarized in Table 3D-19 below. Further details on the body of evidence supporting the selection of these benchmark levels is described in the ISA, Appendix 3 and summarized in the PA, section 3.3 and Appendix 3A.

APEX then calculates two general types of exposure estimates for the population of interest: the estimated number of people exposed to a specified O_3 concentration level and, the number of days per year that they are so exposed, while at moderate or greater exertion. The former highlights the number of individuals exposed *one or more* times per year (i.e., at least once) at or above a selected benchmark level. The latter is expressed as *multiday* exposures, that is, the number of times per year each simulated individual experiences a daily maximum exposure at or above a benchmark. These same exposure results are also used in estimating population-based lung function risk (section 3D.2.8.2.1).

[64] Prolonged exposure to 40 ppb O_3 results in a small decrease in group mean FEV_1 that is not statistically different from responses following exposure to filtered air (Adams, 2002; Adams, 2006).

Table 3D-19. Responses reported in 6.6-hr controlled human exposure studies at a given 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 of the PA for 6.6-hr exposure protocol with exercise EVR of 20 L/min/m^2 (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 FEV_1 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).

3D.2.8.2 Lung Function Risk

We used two approaches to estimate health risk. As done for the lung function risk assessments conducted during the prior O_3 NAAQS reviews, the first approach used a Bayesian Markov Chain Monte Carlo technique to develop probabilistic population-based Exposure-Response (E-R) functions. These population-based E-R functions were then combined with the APEX estimated population distribution of 7-hr maximum exposures for people at or above moderate exertion (EVR $\geq 17.32 \pm 1.25$ L/min-m^2 body surface area) to estimate the number of people expected to experience lung function decrements. The second approach is based on the McDonnell-Stewart-Smith (MSS) FEV_1 model (McDonnell et al., 2013). The MSS model uses the time-series of O_3 exposure and corresponding ventilation rates for each APEX simulated

individual to estimate their personal time-series of FEV_1 reductions, selecting the daily maximum reduction for each person. As done for the exposure benchmark analysis, APEX calculates, for the population of interest, the estimated number of simulated individuals expected to experience an FEV_1 response at or above a selected level and the number of days per year that may occur per person. A key difference between these approaches is that the population-based E-R method directly approximates a population distribution of FEV_1 reductions while the MSS model estimates FEV_1 reductions at the individual level (which are then aggregated to a population level). Each of these approaches is discussed in detail below.

3D.2.8.2.1 Population-based E-R function

For developing the population-based E-R function, we used the exact same E-R function as used for the 2014 HREA given CASAC advice on the approach used for the prior O_3 review (Henderson, 2006) and that there were no new controlled human exposure study data to justify the generating of a new E-R function for this current analysis. Briefly, data from several controlled human exposure studies that evaluated 6.6-hr exposures at moderate exertion were combined and used to estimate E-R functions. Considering the above discussion and as done in the 2014 HREA, we separated the controlled human exposure study data into three lung function decrement categories. The mid- to upper-end of the range of moderate levels of functional responses and higher (i.e., FEV_1 decrements ≥15% and ≥20%) are included to generally represent potentially adverse lung function decrements in active healthy adults, while for people with asthma or lung disease, a focus on moderate functional responses (FEV_1 decrements down to 10%) may be appropriate (Table 3D-20 and Figure 3D-11). The controlled human exposure study data in this table were first corrected on an individual basis for study effects in clean filtered air to remove any systemic bias that might be present in the data attributable to the effects of the experimental procedures and extraneous responses (e.g., exercise, diurnal variability, etc.) (2013 ISA, pp. 6-4 and 6-5). This is done by subtracting the FEV_1 decrement in filtered air from the FEV_1 decrement (at the same time point) during exposure to O_3. An example of this calculation is given in the 2014 HREA, Appendix 6D.

Table 3D-20. Summary of controlled human exposure study data stratified by concentration level and lung function decrements, corrected for individual 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 O_3					
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 O_3					
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 O_3					
Schelegle et al. (2009)	Variable levels (exercise avg= 0.070 ppm)	31	6	3	2
0.080 ppm O_3					
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 O_3					
Schelegle et al. (2009)	Variable levels (exercise avg=0.087 ppm)	31	14	10	7
0.100 ppm O_3					
F-H-M [1]	Square-wave	32	13	11	6
0.120 ppm O_3					
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).

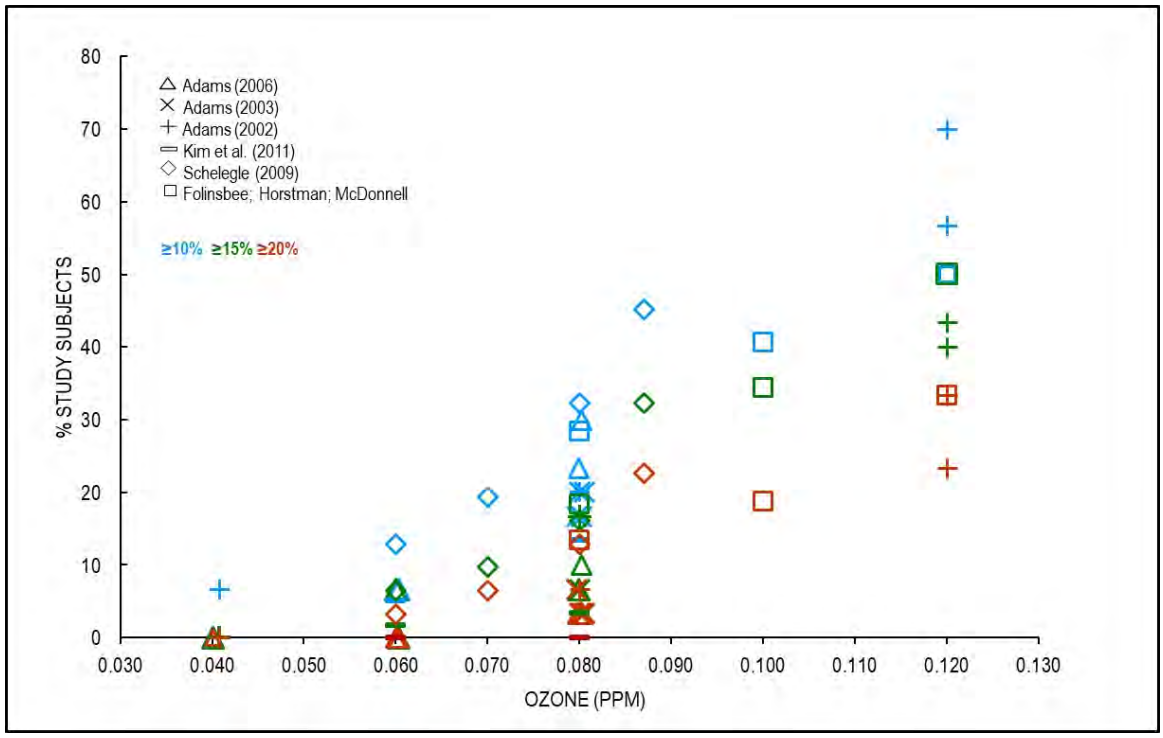

Figure 3D-11. Controlled human exposure data for FEV_1 responses in individual study subjects.

A Bayesian Markov Chain Monte Carlo (BMCMC) approach (Lunn et al., 2012) developed as part of an earlier O_3 exposure and risk analysis (U.S. EPA, 2007a, U.S. EPA, 2007b, section 3.1.2) was modified for the 2014 HREA and used to generate the population-based E-R functions using the updated controlled human exposure study data (Abt, 2013).[65] Briefly, we considered both linear and logistic functional forms in estimating the E-R function and chose a 90 percent logistic/10 percent piecewise-linear split using a BMCMC approach. For each of the three measures of lung function decrement, we first assumed a 90 percent probability that the E-R function has the following 3-parameter logistic form indicated by Equation 3D-9:[66]

$$y(x;\alpha,\beta,\gamma) = \frac{\alpha\, e^{\gamma}(1-e^{\beta x})}{(1+e^{\gamma})(1+e^{\beta x+\gamma})} \qquad \text{Equation 3D-9}$$

[65] In some of the controlled human exposure studies, subjects were exposed to a given O_3 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.

[66] 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.

where x denotes the O_3 concentration (in ppm) to which the individual is exposed, y denotes the corresponding response (decrement in $FEV_1 \geq 10\%$, $\geq 15\%$ or $\geq 20\%$), and α, β, and γ are the three parameters whose values are estimated.

We then assumed a 10 percent probability that the E-R function has the following 2-piece linear with threshold (hockey stick) form[67] indicated by Equation 3D-10:

$$y(x;\alpha,\beta) = \begin{cases} \alpha + \beta x, & for\ \alpha + \beta x > 0 \\ 0, & for\ \alpha + \beta x < 0 \end{cases} \qquad \text{Equation 3D-10}$$

The selection of the 90 percent logistic/10 percent piecewise-linear split was based largely on the results of sensitivity analyses in the 2007 O_3 risk assessment combined with CASAC advice on the model form (U.S. EPA, 2007b),[68] and from model fit determined in the 2014 HREA.[69] Therefore, as done for the 2014 HREA, we are using only the 90/10 E-R function in the current analysis to estimate risk. Further, because there were no newly available controlled human exposure study data for 6.6-hr duration exposures since the 2014 HREA, we used the exact same 90/10 E-R function derived at that time, the overall approach of which is briefly described below.

To generate the E-R functions, prior distributions needed to be specified to estimate the posterior distribution for each of the unknown parameters (Box and Tiao, 1973). For the logistic functional form, we assumed lognormal priors and used Max likelihood estimates (MLE) of the means and variances for the 3 parameters. For the linear functional form, we assumed normal priors using ordinary least square (OLS) estimates for the means and variances for the parameters.

For each of the two functional forms (logistic and linear), we derived the posterior distributions using the binomial likelihood function and prior distributions for each of the unknown parameters. Specifically, we used three Markov chains (each chain corresponds to a set

[67] 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.

[68] The 1997 risk assessment used a linear form consistent with the advice from the CASAC O_3 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 O_3 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).

[69] 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.

of initial parameter values) and for each chain we used 4,000 iterations as the "burn-in" period[70] followed by 96,000 iterations for the estimation. Each iteration corresponds to a set of estimates for the parameters of the (logistic or linear) exposure-response function. We then examined the outputs using the options WinBUGS provides to check convergence and auto-correlation (e.g., trace plot, auto correlation). Finally, we combined 8,100 sets of values from the logistic model runs (the last 2,700 iterations from each chain) with 900 sets of values from the linear model runs (the last 300 iterations from each chain) to obtain a single combined distribution for each predicted value, reflecting the 90 percent/10 percent assumptions stated above (WinBUGS v 1.4.3; Lunn et al., 2012).

We selected the median (50th percentile) E-R function from the 9,000 sets of functions to estimate the risk for changes in FEV_1 $\geq$10%, $\geq$15%, and $\geq$20% (Figure 3D-12). The original E-R data to which the curves were fit are also provided in the figure, along with the derived E-R function data used to combine with the daily maximum 7-hr exposures for the simulated population, while at moderate exertion (section 3D.2.8.1). The population at-risk is estimated by multiplying the expected response rate by the number of people exposed in the relevant population (and stratified by 7-hr average exposures, in 0.01 ppm increments), as shown in Equation 3D-11:

$$R_k = \sum_{j=1}^{N} P_j x(RR_k \mid e_j) \qquad \text{Equation 3D-11}$$

where:

e_j = (the midpoint of) the j^{th} interval of personal exposure to O_3

P_j = fraction of the population with O_3 exposures of e_j ppm

$RR_k \mid e_j$ = k^{th} response rate at O_3 exposure concentration e_j

N = number of intervals (categories) of O_3 personal exposure concentration.

The number of 0.01 ppm intervals was maximally set to 16 (Figure 3D-12), however, given the adjusted air quality scenarios, the midpoint values used in the risk calculation typically ranged from 0.05 to 0.095 ppm. Conventional rounding was applied to the sum of the calculated risk value.[71]

[70] 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.

[71] 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

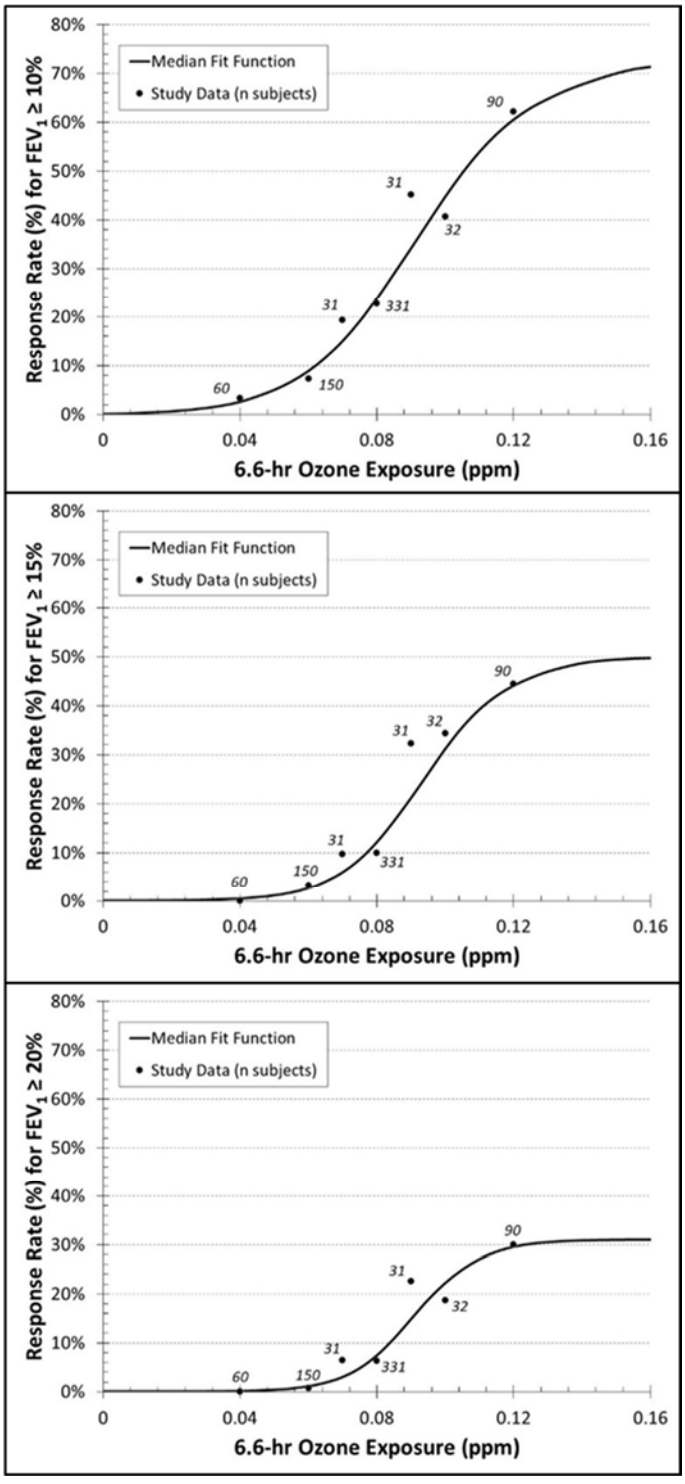

Figure 3D-12. Median value of Bayesian fit population-based E-R function data (left panel) and illustrative curves (right panel) for FEV_1 decrements ≥10% (top panel), ≥15 (middle panel), ≥20% (bottom panel). Drawn from the 2014 HREA, Table 6A-1 with processing and model development described by Abt (2013).

From a practical perspective, the population-based E-R function risk approach takes into account that there is a fraction of the population that could experience a lung function decrement at any daily maximum 7-hr average exposure level (i.e., from the minimum to the maximum, including the level of the exposure benchmarks), having a low probability of decrements resulting from low exposures and higher probability at the highest exposures. That said, the approach allows for decrements to occur at exposures below those tested/observed in the controlled human exposure studies, albeit a small population fraction (e.g., see the response frequency for exposures below 60 ppb in Figure 3D-12), recognizing there is potential for variability in the degree of sensitivity between the controlled human exposure study subjects and the simulated population. Note also that because there is a strict limit on attaining a particular ventilation rate for the simulated individuals (i.e., 7-hr average exposures for individuals must simultaneously occur at moderate or greater exertion, section 3D.2.2.3.3), there may be some potential to underestimate lung function responses if they were to occur at the higher end of the exposure distribution (i.e., where exposures are >60 ppb) that coincide with breathing rates just below those specified by the moderate or greater exertion requirement.

3D.2.8.2.2 The McDonnell-Stewart-Smith (MSS) Model

The McDonnell-Stewart-Smith (MSS) model, a statistical model to estimate FEV_1 responses for individuals associated with short-term exposures to O_3, was developed using controlled human exposure data[72] from studies using varying exposure durations and varying exertion levels and breathing rates (McDonnell et al., 2007). Following the development of the model by McDonnell et al., 2007), Schelegle et al. (2009) found a delay in response when modeling FEV_1 decrements as a function of accumulated dose and estimated a threshold associated with the delay. McDonnell et al. (2012) refit a 2010 version of the model that included a body mass index (BMI) variable (McDonnell et al., 2010), adding data from eight additional studies[73] and incorporating a threshold parameter into the model, which allows for modeling a delay in response until accumulated dose (i.e., accounting for decreases over time according to first order reaction kinetics) reaches a threshold value. The threshold is not a concentration threshold and does not preclude responses at low concentration exposures.

The MSS model was first used for estimating lung function risk in the 2014 HREA and was based on the revised version of the model available at that time (McDonnell et al., 2012). Another version of the MSS model has become available since the last review, which differs

[72] Data were from 15 controlled human exposure studies that included 531 volunteers (ages 18 to 35), exposed to O_3 on a total of 864 occasions (McDonnell et al., 2007).

[73] Data from these eight additional studies included 201 individuals.

from the prior model in that it assumes that the intra-subject variance term (Var(ε)) increases with the response (McDonnell et al., 2013).[74] Therefore, with a fixed ventilation rate, Var(ε) in this most recent version of the MSS model will be larger for higher exposure concentrations and smaller for lower exposure concentrations. The most recent version of the MSS model is the model described here and is the model used in this risk analysis.

The lung function model is conceptually a two-compartment model (Figure 3D-13). The accumulated amount O_3 (exposure concentration × ventilation rate, used to represent dose) is modeled in the first compartment and modified by an exponential decay factor to yield an intermediate quantity **X**. The response (FEV_1 reduction) of the individual to **X** is modeled in the second compartment as a sigmoid-shaped function of the net accumulated dose. A threshold parameter imposes the constraint that there is no response while the value of **X** is below the threshold value.

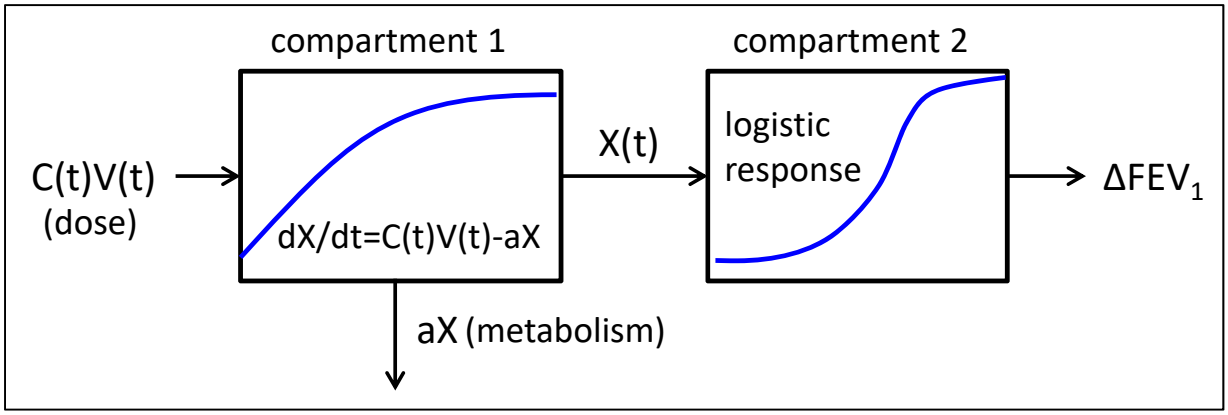

Figure 3D-13. Conceptual representation of the two-compartment model used by the MSS model. *C* is exposure concentration, *V* is ventilation rate, *t* is time, *X* is an intermediate quantity, *a* is a decay constant. Adapted from Figure 1 in McDonnell et al. (1999).

X is given by the solution of the differential Equation 3D-12:

$$\frac{d\boldsymbol{X}}{dt} = C(t)V(t)^{\beta_6} - \beta_5 \boldsymbol{X}(t) \qquad \text{Equation 3D-12}$$

X(t) increases with "normalized dose" ($C \cdot V^{\beta 6}$) over time for an individual and allows for removal of "normalized dose" with a half-life of $1/\beta_5$ through the 2nd term in Equation 3D-12.

[74] 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 FEV_1 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).

The response function **M** is described in Equation 3D-13:

$$\boldsymbol{M}_{ijk} = (\beta_1 + \beta_2 A_{ik} + \beta_8 B_{ik}) \left\{ \frac{1}{1+\beta_4 e^{-\beta_3 T_{ijk}}} - \frac{1}{1+\beta_4} \right\} \qquad \text{Equation 3D-13}$$

where,

$$\boldsymbol{T}_{ijk} = max\{0, \boldsymbol{X}_{ijk} - \beta_9\} \qquad \text{Equation 3D-14}$$

β_9 is a threshold parameter which allows **X** to increase up to the threshold before the median response is allowed to exceed zero. By construction, when **X** = 0, then **M**=0. Because β_3 and β_4 are positive, when **X** > 0 then **M** > 0. Because **X** is never negative, neither is **M**. This model calculates the percent FEV_1 decrement due to O_3 exposure (compartment 2) as:

$$\%\Delta FEV1_{ijk} = e^{U_i} M_{ijk} + \varepsilon_{ijk} \qquad \text{Equation 3D-15}$$

$$Var(\varepsilon_{ijk}) = \nu_1 + \nu_2 e^{U_i} M_{ijk} \qquad \text{Equation 3D-16}$$

Note that a positive value of *%ΔFEV1* means a decrease in effective lung volume or a decrement in lung function. The above variance structure also allows for negative *%ΔFEV1* values or an increase in lung volume, i.e., an improvement in lung function. The indices *i,j,k* in Equations 3D-12 to 3D-16 refer to the i^{th} subject at the j^{th} time for the k^{th} exposure protocol for that subject, while the variables are defined as:

t = time (minutes)

t_0 = time at the start of the event

t_1 = time at the end of the event

C(t) = O_3 exposure (ppm) at time *t* during the event

$V_E(t)$ = expired minute volume (L min^{-1}) at time *t*

BSA = body surface area (m^2),

V(t) = $V_E(t)/BSA$ (L/min-m^2) at time *t*

A_{ik} = age (years) of the i^{th} subject in the k^{th} exposure protocol minus 23.8, the mean age of the subjects

B_{ik} = the body mass index (BMI, kg/m^2) of the i^{th} subject in the k^{th} exposure protocol minus 23.1, the mean BMI of the subjects

U_i = subject-level zero-mean Gaussian random effect error/variability term (between-individual variability not otherwise captured by the model)

ε_{ijk} = Gaussian error/variability term, which includes measurement error and within-individual variability not otherwise captured by the model

ν_1 , ν_2 = constants used to parameterize the variance of ε_{ijk}. ν_1 captures the intra-individual noise in FEV_1 that is not due to ozone exposure, while ν_2 captures the remaining intra-individual variability in FEV_1.

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 βs 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	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:

$$X(t_1) = X(t_0)e^{-\beta_5(t_1-t_0)} + \frac{C(t_1)}{\beta_5}V(t_1)^{\beta_6}\left(1 - e^{-\beta_5(t_1-t_0)}\right) \qquad \text{Equation 3D-17}$$

Note that $C(t_1)$ and $V(t_1)$ denote the (constant) values of *C(t)* and *V(t)* during the event[75] 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 var(*U*) and var(ε), constrained to be within ±2 standard deviations from the means (when sampled values fall outside of this range, they are discarded and resampled).

[75] 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.

The values of U_i are chosen once for each individual and remain constant for individuals throughout the simulation. Values for ε_{ijk} are sampled daily for each individual.

We are using this model to estimate lung function decrements for people ages 5 and older. However, this model was developed using only data from individuals aged 18 to 35 and the age adjustment term [$\beta_1 + \beta_2$ ($Age_{ik} - 23.8$)] in the numerator of Equation 3D-13 is not appropriate for all ages.[76] Clinical studies data for children which could be used to fit the model for children are not available at this time. In the absence of data, we are extending the model to ages 5 to 18 by holding the age term constant at the age 18 level. Since the response increases as age decreases in the range 18 to 35, this trend may extend into ages of children, in which case the responses of children could be underestimated. However, the slope of the age term in the MSS model is estimated based on data for ages 18 to 35 and does not capture differences in age trend within this range; in particular, we do not know at what age the response peaks, which could be above or below age 18. The evidence from clinical studies indicates that the responsiveness of children to O_3 is about the same as for young adults (ISA, Appendix 3, section 3.1.4.1.1) This suggests that the age term for children should not be higher than the age term for young adults (2014 HREA).[77]

Because the responses to O_3 continuously declines from age 18 to 55 and for ages >55 the response is generally considered minimal,[78] here we assume the MSS model age term for ages 35 to 55 linearly decreases to zero and set it to zero for ages >55.[79] To extend the age term to ages outside the range of ages the MSS model is based on (ages 18-35), we re-parameterized the age term in the numerator of Equation 3D-13 by [$\beta_1 + \beta_2(\alpha_1$ Age + $\alpha_2)$], for different ranges of ages (α_1 and α_2 depend on age), requiring that these terms match at each boundary to form a piecewise linear continuous function of age. As a result, the values of α_1 and α_2 for four age ranges are provided in Table 3D-22.

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[76] 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.

[77] See 2014 HREA Chapter 6 (sections 6.4.2 and 6.5.3) and Appendices 6D and 6E for details.

[78] There is a recent 3-hr controlled human exposure study (EVR = 15-17 L/min-m^2 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 FEV_1 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.

[79] "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)

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.				

As described above for the population-based E-R function risk approach (section 3D.2.8.2.1), the individual-based MSS model risk approach also allows for decrements to occur at exposures below those tested/observed in the controlled human exposure studies, however, for this approach there is not a strict limit on the ventilation *per se*. Indeed, FEV_1 decrements are more likely to occur with high breathing rates (and concomitant with high exposures), but it is not necessary that an individual's 7-hr average EVR reach their particular threshold (EVR $\geq 17.32 \pm 1.25$ L/min-m^2) for an individual to experience an adverse response as is used for both the exposure benchmarks and the E-R function risk approach. The time-series of exposures, breathing rate, and FEV_1 will vary with each diary event, with FEV_1 non-linearly dependent on exposure levels/breathing rate from both the prior and current exposure/breathing events. That said in doing so, the MSS approach could overstate risk when including instances where both the exposures and ventilation rates are less than that tested/observed in the controlled human exposure studies.

3D.2.9 Assessing Variability/Co-Variability and Characterizing Uncertainty

An important issue associated with any population exposure and risk assessment is the assessment of variability and characterization of uncertainty. Variability refers to the inherent heterogeneity in a population or variable of interest (e.g., residential air exchange rates). The degree of variability cannot be reduced through further research, only better characterized with additional measurement. Uncertainty refers to the lack of knowledge regarding the values of model input variables (i.e., parameter uncertainty), the physical systems or relationships used (i.e., use of input variables to estimate exposure or risk or model uncertainty), and in specifying the scenario that is consistent with purpose of the assessment (i.e., scenario uncertainty). Uncertainty is, ideally, reduced to the maximum extent possible through improved measurement of key parameters and iterative model refinement.

Section 3D.2.9.1 summarizes how variability and co-variability are addressed in the current exposure and risk analysis and is based on the above described input data and model algorithms used. Section 3D.2.9.2 summarizes the overall approach used for the uncertainty

characterization. The outcome of the updated uncertainty characterization, which builds upon the important uncertainties identified in the IRP (Appendix 5A) and addressed in this current exposure and risk analyses, is discussed below in section 3D.3.4.

3D.2.9.1 Variability and Co-variability Assessment

The goal in addressing variability in this exposure and risk analysis is to ensure that the estimates of exposure and risk reflect the variability of O_3 concentrations in ambient air, population characteristics, associated O_3 exposures, physiological characteristics of simulated individuals, and potential health risk across the study areas and for the simulated at-risk populations. The details regarding many of the variability distributions used as model inputs are described above, while details regarding the variability addressed within its algorithms and processes are found in the APEX User Guides (U.S. EPA, 2019a, U.S. EPA, 2019b).

APEX is designed to account for variability in the model input data, including the physiological variables that are important inputs to determining exertion levels and associated ventilation rates. APEX simulates individuals and then calculates O_3 exposure and lung function risk for each of these simulated individuals. This collection of probabilistically sampled individuals represents the variability of the target population, and by accounting for several types of variability, including demographic, physiological, and human behavior, APEX is able to represent much of the variability in the exposure and risk estimates. For example, variability may arise from differences in the population residing within census tracts (e.g., age distribution) and the activities that may affect population exposure to O_3 (e.g., time spent outdoors, performing moderate or greater exertion level activities outdoors). The range of exposure and associated risk estimates are intended to reflect such sources of variability, although we note that the range of values obtained reflects the input parameters, algorithms, and modeling system used, and may not necessarily reflect the complete range of the true exposure or risk values.

We note also that correlations and non-linear relationships between variables input to the model can result in the model producing inaccurate results if the inherent relationships between these variables are not preserved. APEX is designed to account for co-variability, or linear and nonlinear correlation among the model inputs, provided that enough is known about these relationships to specify them. This is accomplished by providing inputs that enable the correlation to be modeled explicitly within APEX. For example, there is a non-linear relationship between the outdoor temperature and air exchange rate in homes. One factor that contributes to this non-linear relationship is that windows tend to be closed more often when temperatures are at either low or high extremes than when temperatures are moderate. This relationship is explicitly modeled in APEX by specifying different probability distributions of air exchange rates for different ambient air temperatures. Note that where possible, we identified and

incorporated the observed variability in input data sets rather than employing standard default assumptions and/or using point estimates to describe model inputs. In any event, APEX models variability and co-variability in two ways:

- **Stochastically**. The user provides APEX with probability distributions characterizing the variability of many input parameters. These are treated stochastically in the model and the estimated exposure distributions reflect this variability. For example, the rate of O_3 decay in houses can depend on a number of factors which we are not able to explicitly model at this time, due to a lack of data. However, we can specify a distribution of removal rates that reflects observed variations in O_3 decay. APEX randomly samples from this distribution to obtain values that are used in the mass balance model. Further, co-variability can be modeled stochastically through the use of conditional distributions. If two or more parameters are related, conditional distributions that depend on the values of the related parameters are input to APEX. For example, the distribution of air exchange rates (AERs) in a house depends on the outdoor temperature and whether or not air conditioning (A/C) is in use. In this case, a set of AER distributions is provided to APEX for different ranges of temperatures and A/C use, and the selection of the distribution in APEX is driven by the temperature and A/C status at that time.
- **Explicitly**. For some variables used in modeling exposure, APEX models variability and co-variability explicitly and not stochastically. For example, the complete series of hourly ambient air O_3 concentrations and hourly temperatures are used in the exposure and risk calculations. These are input to the model continuously in the time period modeled at different spatial locations, and in this way the variability and co-variability of hourly O_3 concentrations and hourly temperatures are modeled explicitly.

Important sources of the variability and co-variability accounted for by APEX and used for this exposure and risk analysis are provided in Table 3D-23 and Table 3D-24, respectively.

Table 3D-23. Summary of how variability was incorporated into the exposure and risk 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 (Burmaster, 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.

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.

3D.2.9.2 Approach for Uncertainty Characterization

While it may be possible to capture a range of exposure or risk values by accounting for variability inherent to influential factors, the true exposure or risk for any given individual within a study area may be unknown, although it can be estimated. To characterize health risks, exposure and risk assessors commonly use an iterative process of gathering data, developing models, estimating exposures and risks, evaluating results for correctness and identifying areas for potential improvement, given the goals of the assessment, scale and complexity of the assessment performed, and limitations of the input data available. However, important

uncertainties often remain in any one of the data sets, tools, and approaches used and emphasis is then placed on characterizing the nature of that uncertainty and its impact on exposure and risk estimates.

The overall approach used for this exposure and risk generally follows that described by WHO (2008) but varies in that a greater focus has been placed on evaluating the direction and the magnitude of the uncertainty. This refers to qualitatively rating how the source of uncertainty, in the presence of alternative information, may affect the estimated exposures and health risk results. Following the identification of key uncertainties, we subjectively scale the overall impact of the identified uncertainty by considering the relationship between the source of uncertainty and the exposure concentrations (e.g., low, medium, or high potential impact). Also to the extent possible, we include an assessment of the direction of influence, indicating how the source of uncertainty may be affecting exposure or risk estimates (e.g., the uncertainty could lead to over-estimates, under-estimates, or both directions). Further, and consistent with the WHO (2008) guidance, we discuss the uncertainty in the knowledge-base (e.g., the accuracy of the data used, recognition of data gaps) and, where possible, particular assessment design decisions (e.g., selection of particular model forms). The output of the uncertainty characterization is a summary that describes, for each identified source of uncertainty, the magnitude of the impact and the direction of influence the uncertainty may have on the exposure and risk results.

We further recognize that uncertainties associated with APEX exposure modeling have been previously characterized in the REAs for nitrogen dioxide (NO_2), carbon monoxide (CO) and sulfur dioxide (SO_2) conducted for recent primary NAAQS reviews, along with other pollutant-specific issues (U.S. EPA, 2008, 2010, 2014, 2018), all complementary to quantitative uncertainty characterizations conducted for the 2007 O_3 exposure assessment by Langstaff (2007). Conclusions drawn from each of these characterizations are also considered here in light of new information, data, tools, and approaches used in this exposure and risk analysis.

3D.3 POPULATION EXPOSURE AND RISK RESULTS

Exposure and risk results are presented here for simulated populations residing in the eight study areas – Atlanta, Boston, Dallas, Detroit, Philadelphia, Phoenix, Sacramento, and St. Louis – for a three-year air quality scenario in which air quality conditions just meet the current primary 8-hr O_3 standard (70 ppb, annual 4th highest daily maximum 8-hr average concentration, averaged across 3-years) and two other air quality scenarios (i.e., design values of 75 and 65 ppb). Hourly concentrations of O_3 in ambient air for the three hypothetical air quality scenarios are estimated at census tracts in each study area as described in section 3D.2 above. Population exposure and risk associated with these concentrations is estimated using the APEX model simulations (section 3D.2) and is briefly described with the following.

APEX uses the hourly air quality surface in each study area, along with U.S. census tract population demographics, to estimate the number of days per year each simulated individual in a particular study area experiences a daily maximum 7-hr average O_3 exposure at or above benchmark levels of 60, 70, and 80 ppb (section 3D.2.8.1). These short-term exposures were evaluated for children (5-18 years old), adults (>18 years old), and those with asthma within each of these two study groups when the exposure corresponded with moderate or greater exertion (i.e., the individual's EVR $\geq 17.32 \pm 1.25$ L/minute-m^2).

Then, individuals expected to experience a lung function decrement (i.e., reduction in FEV_1 ≥10%, ≥15%, ≥20%) were estimated using two approaches. The first approach linked the population-based daily maximum 7-hr exposures while at moderate or greater exertion with an exposure-response function derived from controlled human exposure study data (section 3D.2.8.2.1). The second lung function risk approach, considered an individual-based approach here, used the McDonnell-Stewart-Smith (MSS) FEV_1 model (McDonnell et al., 2013) (section 3D.2.8.2.2). The MSS uses the time-series of O_3 exposure and corresponding ventilation rates for each APEX simulated individual to estimate their personal time-series of FEV_1 reductions, selecting the daily maximum reduction for each person. The number of individuals estimated to experience decrements are then aggregated to the population level. Again, of interest for both of these lung function risk approaches is the number of days per year each simulated individual in a particular study area experiences a lung function decrement.

Study area characteristics and the composition of the simulated population are provided in section 3D.3.1. Exposure results are presented in a series of tables that allow for simultaneous comparison of the exposure and risk metrics across the eight study areas and three simulation years. Two types of results are provided for each study area: the percent (and number) of the simulated population exposed at or above selected benchmarks, stratified by the number of occurrences (i.e., days) in a year (section 3D.3.2) and the percent (and number) of the simulated population experiencing a reduction in FEV_1 ≥10%, ≥15%, ≥20%, also stratified by the number of days in a year (section 3D.3.3). Tables summarizing all of the exposure and risk results for each study area are provided in Attachment 4.

3D.3.1 Characteristics of the Simulated Population and Study Areas

The eight study areas differ in population, geographic size, and demographic features (Table 3D-25). In each of the eight study areas, APEX simulated O_3 exposures and risks for 60,000 individuals,[80] the demographic features of which were based on the information

[80] 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.

associated with the hundreds to thousands of census tracts within each area (as described in section 3D.2.1 above).

Asthma prevalence in each modeling domain was estimated based on the 2013-2017 NHIS asthma prevalence data and the demographic characteristics for each study area (e.g., age, sex and family income) using the methodology summarized in section 3D.2.2.2. Accordingly, the percent of the simulated populations with asthma within the exposure modeling domain varied by study area (Table 3D-25). The Dallas, Phoenix, and Sacramento study areas had the lowest percent of children with asthma (9.2 to 9.6%), while Atlanta and Boston had the highest percent of children with asthma (11.8 to 12.3%). The Dallas study area had the lowest percent of adults with asthma (7.2%), while Boston and Detroit had the highest percent of adults with asthma (both 10.9%). The statistics presented here are the aggregate of the study area as a whole, within which asthma prevalence varied widely as the modeling approach fully accounted for the variation in asthma prevalence across census tracts with demographic factors such as family income to poverty ratios, age, and sex (and as described in section 3D.2.2.2).[81] Nationally, asthma prevalence is 7.9%; for children it is 8.4% and for adults it is 7.7% (Chapter 3, Table 3-1). The asthma prevalence for children, adults, and the total population estimated for each of the eight study areas are all greater than that of the national asthma prevalence, except for adults in Dallas which has a slightly lower asthma prevalence. This suggests that overall, the at-risk population simulated in the eight study areas could represent at-risk populations in other U.S. urban areas that have a similarly above average asthma prevalence.

[81] 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.

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.

3D.3.2 Exposures at or above Benchmark Concentrations

The exposure to benchmark comparisons are presented in a series of tables focusing on the benchmark levels (i.e., people experiencing daily maximum 7-hr average O_3 exposures ≥60, 70, and 80 ppb while at moderate or greater exertion). The full range of ambient air O_3 concentrations for a 3-year O_3 season (2015-2017) were used by APEX, providing a range of estimated exposures. Adjusted air quality surfaces used to represent three air quality scenarios were developed using 2015-2017 design values modeled sensitivities to changes in precursor emissions (section 3D.2.3.3), and then interpolated to census tract centroids (section 3D.2.3.4).

Exposures were estimated for four study groups of interest (i.e., school-age children (5-18), school-age children with asthma, adults (19-90), and adults with asthma).

In this exposure and risk analysis, we are primarily interested in O_3 exposures associated with the ambient air quality adjusted to just meet the current standard (70 ppb, annual 4th highest daily maximum 8-hr average concentration, averaged over a 3-year period). Provided are the percent and number of people in each study group estimated to experience 7-hr exposures at or above the benchmarks, while at moderate or greater exertion (section 3D.3.2.1). For each exposure metric and study group, the occurrence of single-day (at least one day per year) and multi-day (at least 2, 4, or 6 days per year) exposures are presented. Exposure results for the two other adjusted air quality scenarios (the 75 ppb and 65 ppb scenarios) are presented in sections 3D.3.2.2 and 3D.3.2.3, respectively. These two sections present only the percent of each study group estimated to experience exposures at or above benchmarks while at moderate or greater exertion, for single-day and multiday exposures during a year, and not also the number of simulated individuals in each study group. The complete exposure results associated with all simulated years, air quality scenarios, the four study groups, and eight study areas are found in Attachment 4.

In general, and for all air quality scenarios, the percent of children estimated to experience exposures at or above any of the benchmarks is consistently higher than that estimated for adults. This is expected because children spend a greater amount of time outdoors, and at a greater frequency, while at moderate or greater exertion when compared to adults (2014 HREA, sections 5.4.1 and 5.4.2). Estimated exposures for healthy people are similar to people with asthma when considered on a percent of population basis. This is because similar diary data are used to simulate the activity patterns of each study group, justified by evaluations that indicated similarities in time spent outdoors, participation rate, and exertion level for people with asthma when compared to healthy individuals (section 3D.2.5.3). When considering the estimated exposures in terms of population counts, while children comprise about 20% of the simulated population (Table 3D-25), the number of children experiencing exposures at or above the benchmarks is greater than that of adults. Again, this a direct result of the differences in time spent outdoors performing activities at elevated exertion. And finally, Detroit, Phoenix, and St. Louis have a higher percent of individuals at or above benchmark levels relative to the other study areas, likely influenced by their having an hourly O_3 concentration distribution shape that, overall, is more skewed to the right and/or has heavy tails at the uppermost percentiles (Figure 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 ≤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 O_3 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 O_3 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.

Multiday exposures are limited when considering air quality adjusted to just meet the current standard. For example, there are no children estimated to experience at least two days with 7-hr O_3 exposures at or above the 80-ppb benchmark and ≤0.1% at or above the 70-ppb benchmark (Table 3D-28 and Table 3D-29). When considering the worst air quality year, <5% of children (and ≤0.4% of adults) are estimated to experience at least two days with 7-hr O_3 exposures at or above the 60-ppb benchmark. There are no people estimated to experience at least four days with 7-hr O_3 exposures at or above the 70-ppb benchmark except in one study area (Table 3D-30 and Table 3D-31), and ≤0.5% experience at least six days with 7-hr O_3 exposures at or above the 60-ppb benchmark (Attachment 4).

Table 3D-26. Percent of people estimated to experience at least one exposure at or above benchmarks while at moderate or greater exertion, for air quality adjusted to 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".

Table 3D-27. Number of people estimated to experience at least one exposure at or above benchmarks while at moderate or greater exertion, for air quality adjusted to 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).

Table 3D-28. Percent of people estimated to experience at least two exposures at or above benchmarks while at moderate or greater exertion, for air quality adjusted to 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".

Table 3D-29. Number of people estimated to experience at least two exposures at or above benchmarks while at moderate or greater exertion, for air quality adjusted to 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).

Table 3D-30. Percent of people estimated to experience at least four exposures at or above benchmarks while at moderate or greater exertion, for air quality adjusted to 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".

Table 3D-31. Number of people estimated to experience at least four exposures at or above benchmarks while at moderate or greater exertion, for air quality adjusted to 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).

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 O_3 exposures at or above each of the benchmarks. For example, estimated exposures to O_3 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 O_3 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 O_3 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 O_3 exposures at or above the 60-ppb benchmark for the worst air quality year (Attachment 4).

Table 3D-32. Percent of people estimated to experience at least one exposure 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	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".

Table 3D-33. Percent of people estimated to experience at least two 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	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".

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".

3D.3.2.3 Additional Air Quality Scenario: 65 ppb

With increasing stringency (i.e., lowering) of the design value used to represent the air quality scenario, there is a reduction in the percent and number of simulated individuals experiencing 7-hr exposures at or above the benchmarks. Under the 65 ppb air quality scenario, in 6 of the 8 study areas, there are no people estimated to experience at least one benchmark at or above the 80-ppb benchmark (Table 3D-35). Exposures at or above the 70-ppb benchmark are also limited, with at most 0.2% of children (and 0.3% of children with asthma) estimated experience one such exposure during the worst air quality year. On average, between 0.4 to 2.3% of children (and 0.5 to 2.5% of children with asthma) and between 0.1 to 0.4% of adults (and <0.1 to 0.3% of adults with asthma) are estimated to experience at least one 7-hr O_3 exposure at or above the 60-ppb benchmark, while during the worst air quality year, upwards to 3.7% of children (and 4.3% of children with asthma) would experience such an exposure.

Multiday exposures at or above the 70-ppb benchmark are nearly eliminated under the 65 ppb air quality scenario, with only three study areas having at most, <0.1% of children (and no children with asthma) estimated to experience 7-hr exposures at or above that benchmark for at least two days (Table 3D-36). When considering the worst air quality year, ≤0.5% of children (and 0.6% of children with asthma) and ≤0.1% of adults (and similarly for adults with asthma) are estimated to experience at least two days with 7-hr O_3 exposures at or above the 60-ppb benchmark. There are no people in any of the study areas estimated to experience at least four days with 7-hr O_3 exposures at or above the 70-ppb benchmark (Table 3D-37), and there no simulated individuals estimated to experience at least six days with 7-hr O_3 exposures at or above the 60-ppb benchmark in all but two study areas (Attachment 4).

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".

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".

Table 3D-37. Percent of people estimated to experience at least four 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	<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".

3D.3.2.4 Comparison with 2014 HREA Exposure Results

We compared the exposure results for the current exposure and risk analysis with those generated for the 2014 HREA. Table 3D-38 presents the percent of children experiencing at least one exposure at or above the three benchmarks for the two assessments and Table 3D-39 presents the similar comparison for two or more exposures. Results are presented for all study areas, and for the seven study areas common to both assessments. In general, the comparison indicates similarity between the two assessments, particularly for the highest benchmark and when focusing on the summary for all areas in each assessment. Such a focus is appropriate given the purpose of the assessments in providing estimates across a range of study areas to inform decision making with regard to the exposures and risks that may occur across the U.S. in areas that just meet the current standard. For the lower benchmarks and particularly in comparing for the seven areas common to both assessments, the current assessment estimates are slightly lower than the 2014 HREA results, most notably for the highest single year, likely reflecting the greater variation in ambient air concentrations in some study areas in the 2014 HREA. This is supported by recent analyses that show changes to the distribution of ambient air O_3 concentrations over time occur primarily as reductions to the highest and lowest concentrations (Downey et al., 2015; Simon et al., 2012).

In addition to generally lower baseline O_3 concentrations and lower variability in the concentrations in the three air quality scenarios for the current assessment compared to 2014 HREA, there were also two important differences in the exposure modeling approach. The first is the use, in the current assessment, of an EVR distribution (17.32 ± 1.25 L/minute-m^2) to indicate when a simulated individual is at moderate or greater exertion (section 3D.2.2.3.3) rather than using a lower value for all simulated individuals (13 L/minute-m^2; 5^{th} percentile). The current approach would be expected to result in far fewer individuals reaching the exertion level concomitant with the exposure level of interest, thus reducing the percent of the population at or above benchmarks. The second difference is the focus on 7-hr average exposures (compared to the benchmarks) in this assessment rather than 8-hr averages. With this change, it would be expected that there would be more simulated individuals at or above a given benchmark concentration. While these two changes to the exposure modeling approach compete in their overall influence on the exposure results, it would be expected that the change to using the EVR distribution would have a greater impact.

As suggested above, the difference between the two assessments in the highest year estimates is likely a function of the baseline ambient air concentrations in the study areas. As a reminder, the 2014 HREA used air quality scenarios developed from 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). In the current

exposure analysis, we selected study areas that had 2015-2017 design values close to the current standard, requiring less of an adjustment for the current standard (70 ppb) air quality scenario.

Table 3D-38. Comparison of current assessment to 2014 HREA for percent of children estimated to experience at least one exposure at or above benchmarks while at 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]
Benchmark Exposure Concentration of 80 ppb				
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)
Benchmark Exposure Concentration of 70 ppb				
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)
Benchmark Exposure Concentration of 60 ppb				
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".

Table 3D-39. Comparison of current assessment to 2014 HREA for percent of children estimated to experience at least two exposure at or above benchmarks while 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]
Benchmark Exposure Concentration of 80 ppb				
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)
Benchmark Exposure Concentration of 70 ppb				
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)
Benchmark Exposure Concentration of 60 ppb				
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".

3D.3.3 Lung Function Risk

As described above, lung function risk was estimated using two approaches. The first, a population-based risk approach (i.e., using E-R functions, section 3D.2.8.2.1), combined the population distribution of daily maximum 7-hr exposures occurring while at moderate or greater exertion with continuous E-R functions derived from the controlled human exposure study data (Table 3D-20 and Figure 3D-12). Note that the E-R function risk approach uses the full distribution of daily maximum 7-hr exposures, from the minimum to the maximum exposures (i.e., not simply including the upper level exposures or benchmarks). It is, however, necessary that the daily maximum exposure did occur at a 7-hr EVR $\geq 17.32 \pm 1.25$ L/min-m^2. The results for the population-based (E-R function) risk approach, represented as percent (or counts) of the population estimated to experience lung function decrements (i.e., ≥10%, ≥15%, and ≥20%

reduction in FEV_1) is provided in section 3D.3.3.1. A similar format to that provided for the benchmark results above is followed, focusing largely on the air quality scenario for just meeting the current standard and presenting the percent (and counts) of the population estimated to experience lung function decrements while at elevated exertion.

The second risk approach, an individual-based risk approach (i.e., the MSS model, section 3D.2.8.2.2), calculates the decrements in lung function continuously for each simulated person using their unique time-series of O_3 exposures, simultaneously occurring breathing rates, and personal attributes (e.g., age, body mass). Note that when using the MSS model risk approach, the estimated reduction in FEV_1 considers the prior and current exposures/breathing rates and has no hard restriction on either the exposure or exertion level. As such, lung function decrements could also occur at exposures and/or breathing rates below that observed in the controlled human exposure studies. The results for the individual-based (MSS model) risk approach are found in section 3D.3.3.2. The complete results for both of the risk approaches can be found in Attachment 4.

3D.3.3.1 Population-based (E-R Function) Risk Approach

As was observed with the exposure benchmarks and considering any of the air quality scenarios, a smaller percent (and number) of adults are estimated to experience lung function decrements when compared to children (Table 3D-40 to Table 3D-51). Again, this is driven largely by the difference in time spent outdoors at elevated exertion. Even though there is limited variability across the eight study areas, Detroit, Phoenix, and St. Louis generally exhibited higher risk estimates relative to the other study areas for instances where risk estimates were above 1% (e.g., where FEV_1 reductions $\geq$10%). This is expected given the observation made above regarding the results for the exposure to benchmark comparison and its relationship with the overall distribution of O_3 concentrations in ambient air (Figure 3D-7).

In general, when comparing E-R function risk estimates to the benchmark results, the attenuation of the percent estimated to experience lung function decrements is at a lesser rate than that observed for the percent of the population at or above the benchmark levels, with increasing stringency of the design values, and when considering the number of times per year either might occur. For example, while as much as 0.9% of children (and 1.0% of children with asthma) are estimated to experience at least one FEV_1 reduction $\geq$15% while at elevated exertion with air quality just meeting the current standard (Table 3D-40), on average between 0.2 to 0.4% of children (and similarly for children with asthma) in all 8 study areas are estimated to experience at least four such decrements (Table 3D-44) when considering the same air quality scenario. For comparison, while as much as 0.9% of children (and 1.0% of children with asthma) are estimated to experience at least one exposure at or above the 70 ppb benchmark while at

elevated exertion for air quality just meeting the current standard (Table 3D-26), there are no children (and similarly for children with asthma) estimated to experience at least four such exposures in all but one study area (Table 3D-30) when considering the same air quality scenario. This relative decreased rate of change observed for the E-R function risk results is likely a function of the broader range (and low level) of exposures used in the calculation compared to that represented by the exposure benchmarks.

The risks of lung function decrements in the 75 ppb air quality scenario, which allows higher O_3 concentrations, are of course greater (Table 3D-46 through Table 3D-48) than those for air quality adjusted to just meet the current standard (Table 3D-40 through Table 3D-45), differing by at most a few tenths of a percentage point for both the 15% and 20% reduction in FEV_1. A similar pattern is exhibited when comparing the lung function results for the current standard to those for the 65 ppb air quality scenario (Table 3D-49 through Table 3D-51). A few tenths of a percentage point lower risks are estimated for the lower design value scenario compared to those estimated for the current standard.

Table 3D-40. Percent of people estimated to experience at least one lung function decrement 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	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".

Table 3D-41. Number of people estimated to experience at least one lung function decrement 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	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).

Table 3D-42. Percent of people estimated to experience at least two 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	1.4	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".

Table 3D-43. Number of people estimated to experience at least two 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	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).

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".

Table 3D-45. Number 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	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).

Table 3D-46. Percent of people estimated to experience at least one lung function decrement at or above the indicated level, for the 75 ppb air quality scenario, 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".

Table 3D-47. Percent of people estimated to experience at least two lung function decrements at or above the indicated level, for the 75 ppb air quality scenario, 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".

Table 3D-48. Percent of people estimated to experience at least four lung function decrements at or above the indicated level, for the 75 ppb air quality scenario, 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
	St. Louis	1.3	1.1	1.4	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
	St. Louis	1.3	1.1	1.4	0.3	0.2	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
	St. Louis	0.2	0.2	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".

Table 3D-49. Percent of people estimated to experience at least one lung function decrement at or above the indicated level, for the 65 ppb air quality scenario, 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".

Table 3D-50. Percent of people estimated to experience at least two lung function decrements at or above the indicated level, for the 65 ppb air quality scenario, 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".

Table 3D-51. Percent of people estimated to experience at least four lung function decrements at or above the indicated level, for the 65 ppb air quality scenario, 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".

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.

Table 3D-52. Percent of people estimated to experience at least one lung function decrement at or above the indicated level, for air quality adjusted to just meet 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".

Table 3D-53. Number of people estimated to experience at least one lung function decrement at or above the indicated level, for air quality adjusted to just meet 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).

Table 3D-54. Percent of people estimated to experience at least two lung function decrements at or above the indicated level, for air quality adjusted to just meet 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	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".

Table 3D-55. Number of people estimated to experience at least two lung function decrements at or above the indicated level, for air quality adjusted to just meet 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	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).

Table 3D-56. 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 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	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".

Table 3D-57. Number 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 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	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).

Table 3D-58. Percent of people estimated to experience at least one lung function decrement at or above the indicated level, for the 75 ppb air quality scenario, 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".

Table 3D-59. Percent of people estimated to experience at least two lung function decrements at or above the indicated level, for the 75 ppb air quality scenario, 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".

Table 3D-60. Percent of people estimated to experience at least four lung function decrements at or above the indicated level, for the 75 ppb air quality scenario, 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".

Table 3D-61. Percent of people estimated to experience at least one lung function decrement at or above the indicated level, for the 65 ppb air quality scenario, 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".

Table 3D-62. Percent of people estimated to experience at least two lung function decrements at or above the indicated level, for the 65 ppb air quality scenario, 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".

Table 3D-63. Percent of people estimated to experience at least four lung function decrements at or above the indicated level, for the 65 ppb air quality scenario, 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".

3D.3.4 Uncertainty Characterization

While it may be possible to estimate a range of O_3 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 O_3 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 O_3 exposure.

3D.3.4.1 Summary of the Uncertainty Characterization

The REAs for the previous reviews of the O_3, NO_2, SO_2, 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

over-estimate ("*over*"), under-estimate ("*under*"), or have an *unknown* impact to exposure/risk estimates. A summary of the key findings of the prior uncertainty characterizations that are most relevant to the current O_3 exposure and risk analysis are also provided in Table 3D-64.

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 $\mu g/m^3$ 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 (PA, 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 (PA, 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 3rd 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	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).	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	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 ≥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.	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)[82] 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

[82] For example, regarding the development of an E-R function describing lung function decrements associated with exposure to SO_2 for the risk assessment performed in the 2012 review of the SO_2 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).

forms in a 90/10 percent proportion, respectively, using a Bayesian Markov Chain Monte Carlo (BMCMC) modeling approach (section 3D.2.8.2.1). The selection of this model was based on advice received from the CASAC review in the 2008 O_3 NAAQS review (Henderson, 2006) and evaluation of the curve fit statistics for this function (U.S. EPA, 2007a; 2014 HREA).

Of practical importance for this assessment is how the response curve is extrapolated from the lowest observed exposure to zero exposure/response. For general context, in comparing a probit to a logit link in a logistic regression, the probit link would yield a relatively lower response at the lowest level exposures. The two-piece linear model used in part for developing the current E-R function resembles a hockey stick, with the paddle representing a zero response for the lowest exposures, and the handle representing the increased response that coincides with increasing exposures, beginning at the junction between the paddle and the stick.[83] Based on this statistical form, when combining the logit linked logistic model with a hockey stick type model (as done for this assessment), it was assumed the 90/10 percent proportion logit/linear E-R form would have a response curve shape for low-level exposures similar to that using a probit link. To better evaluate these E-R functions, we fit the controlled human exposure study data (Figure 3D-12) using a probit link and compared that with the 90/10 logit/linear curve.

As an example, Figure 3D-14 illustrates the E-R functions fit from these two approaches, using the ≥15% lung function decrement observations. Plotted for the probit approach is the curve derived from the best estimate of the model coefficients, along with 95% confidence intervals derived from the model coefficient variability. For the 90/10 logit/linear approach, the median (50^{th} percentile) function is plotted, along with 95% confidence intervals derived from the 2.5^{th} and 97.5^{th} percentile E-R functions obtained from the 9,000 BMCMC model iterations. As expected, the probit curve is very similar to the 90/10 logit/linear curve, albeit with the former having a response just below the latter for the lower exposures. The opposite occurs for exposures above 55 ppb; for those higher exposures, a relatively greater response is indicated using the 90/10 logit/linear E-R function. Based on there being little difference between the two curves and only slight off-setting of the response at different exposure levels, it is likely that applying a probit fit for the E-R function to the population distribution of daily maximum 7-hr exposures would result in little to no difference from the risk estimates derived with the 90/10 logit/linear E-R function.[84]

[83] 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.

[84] 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

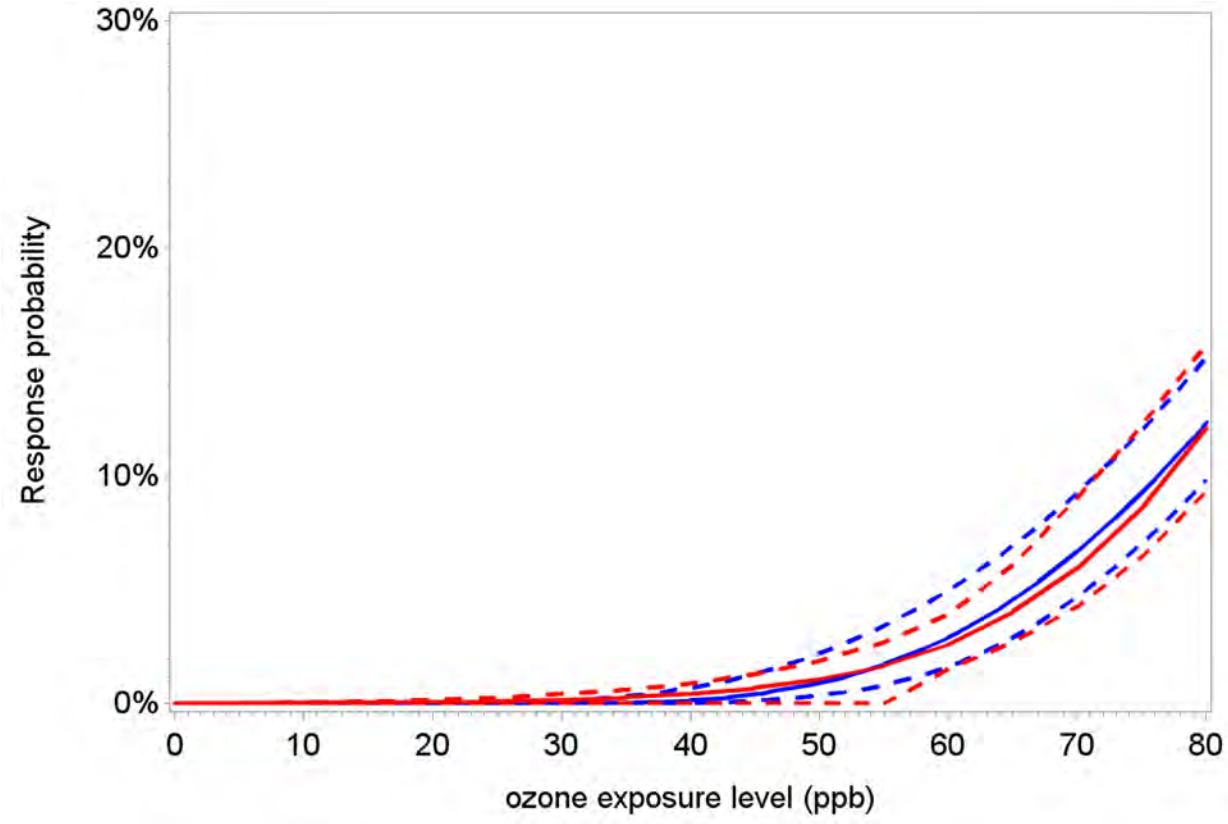

Figure 3D-14. Comparison of a probit function curve (blue line) with the Bayesian logistic/linear function curve (red) in estimating the probability of lung function decrements ≥15% (based on data in Table 3D-20). Confidence intervals for the probit model reflect variability in the regression model coefficients.

3D.3.4.2.2 Confidence Intervals for the Population-based E-R Function and Effect on Lung Function Risk Estimates

To estimate lung function risk using the population-based E-R function approach, the results of which are presented in the main body of this document (section 3D.3.3.1), we selected the median (50^{th} percentile) E-R function originally developed as part of the 2014 HREA. This E-R function was derived from Bayesian Markov Chain Monte Carlo (BMCMC) approach that iteratively combined logistic and linear E-R functions fit to the controlled human exposure study data in Table 3D-20 (section 3D.2.8.2.1). The selection of the median E-R function to estimate 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.

individuals that have a similar response frequency as that of the general collection of controlled human exposure study test subjects.

Because there were two or more studies reporting observed responses at most of the exposure levels and the BMCMC approach generates numerous E-R functions, statistical uncertainty in the E-R function can be used to approximate lower and upper bounds to the lung function risk estimates. To evaluate such bounds here, a 95% confidence interval for lung function risk was estimated by combining the population distribution of daily maximum 7-hr exposures (occurring while at moderate or greater exertion) for simulated children in each study area with the 2.5^{th} and 97.5^{th} percentile population-based E-R functions (2014 HREA, Appendix 6A, Table 6A-1). Lung function risk estimates based on these lower and upper percentile functions and those based on the median function (for air quality just meeting the existing standard) are presented, in terms of the minimum and maximum year results, in Table 3D-65 for each of the three lung function decrements (i.e., FEV_1 decrements ≥10, 15, and 20%). The estimates for the median E-R function are drawn from Table 3D-40. The estimates for the best and worst air quality years yield the minimum and maximum estimates for each of the three functions providing a range for estimates based on each of the particular E-R functions.

The range of values for the estimated risk generated by each of the E-R functions as a result of using different air quality years (i.e., the distance in estimated risk between the minimum and maximum values) is small, on the order of a few tenths of a percentage point, with the smallest range of values associated with the largest lung function decrement (Table 3D-65). In general, the range of the overall 95% confidence interval (i.e., the distance in estimated risk between the 2.5^{th} and 97.5^{th} values) is also small when considering percentage points. For example, the lower bound percent of children estimated to experience at least one lung function decrement ≥10% for the Atlanta study area is about 1.5% and the upper bound value is about 4.0% (median E-R function value ~ 2.4%). With increasing magnitude of the decrement, the range of percentage points becomes smaller (e.g., a ≥20% decrement has a range of about 0.6 percentage points for Atlanta). In terms of relative magnitude, one might consider this range large (i.e., a factor of 6 or more), but because there are so few children estimated to experience these large lung function decrements, this interpretation of the confidence interval would be inappropriate. Further, it would be unreasonable to simply assume that use of the lower and upper bounds of the E-R functions would appropriately estimate lower and upper bounds of risk without additional context regarding the controlled human exposure study data, the interpretation of the bounds on the E-R function, and how these might relate to statistical uncertainty in the risk estimates.

Table 3D-65. Percent of children estimated to experience at least one lung function decrement 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.

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.

As a reminder, while the controlled human exposure study subjects are volunteers (and assumed to be selected at random), it is important to note there are important fundamental biases in their collective composition: none of the individuals have known preexisting health conditions

(e.g., cardiovascular disease, asthma) and all of the subjects are required to be physically fit enough to meet a study's exercise target levels. Clearly, not every member of the simulated population has these attributes, but the risk approach does select for when simulated individuals are at moderate or greater exertion while exposed, as was done for the controlled human exposure study subjects. Therefore, representation of potentially sensitive individuals (i.e., those with pre-existing health conditions) in the study data and thus, in the derived E-R functions, is absent.

In addition, use of this type of statistical approach to estimate lower and upper bounds of lung function risk does not suggest that the range of functions could be equally applied to the simulated population as a whole (e.g., that the entire population could have a risk as low as *X* or as high as *Y*, based on the 2.5^{th} and 97.5^{th} percentile functions selected, respectively) nor does the range of E-R functions likely represent individuals that are least sensitive, or more importantly (given the NAAQS review context for these analyses), those most sensitive to O_3 exposure. The variability in the observed response in study subjects at given O_3 exposures can be due to many factors (e.g., uncertainties in exposure conditions, response/concentration measurements, study subject sensitivity for healthy individuals, number of subjects per study, etc.). When used in such an analysis here, one might suggest the lower and upper bounds account for some of these uncertainties, however, they would be bounded by their collective representativeness and actual weighting of these uncertainties present in the study observations. In its application, it would be assumed that the distribution of the features of the study subjects are similarly reflected in the simulated population, which as described above, is not entirely the case.

Further, the range of functions used to represent lower and upper bounds is derived from a distribution of functions. If there were a perfect matching of the study subject attributes with those of the simulated population, the risks estimated using either end of the 95% interval for the E-R function would certainly have a much smaller likelihood and better apply to a smaller proportion of the population, than those estimated from using the median E-R function. That said, even in the absence of perfectly matching the attributes of the controlled human exposure study subjects with those of the simulated population, the median E-R function may be most appropriate to estimate lung function risk for the simulated population as a whole. Still, the median E-R function may be underestimating the number/percent of individuals experiencing decrements to the extent that the general population includes individuals that would experience greater decrements than experienced by individuals represented in the controlled human exposure studies. Further, as recognized in Chapter 3 of the PA, similarly sized decrements in individuals with compromised respiratory function or in individuals with asthma 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., O_3 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 O_3 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%).

Table 3D-66. Estimated lung function risk contribution resulting from selected 7-hr average O_3 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%

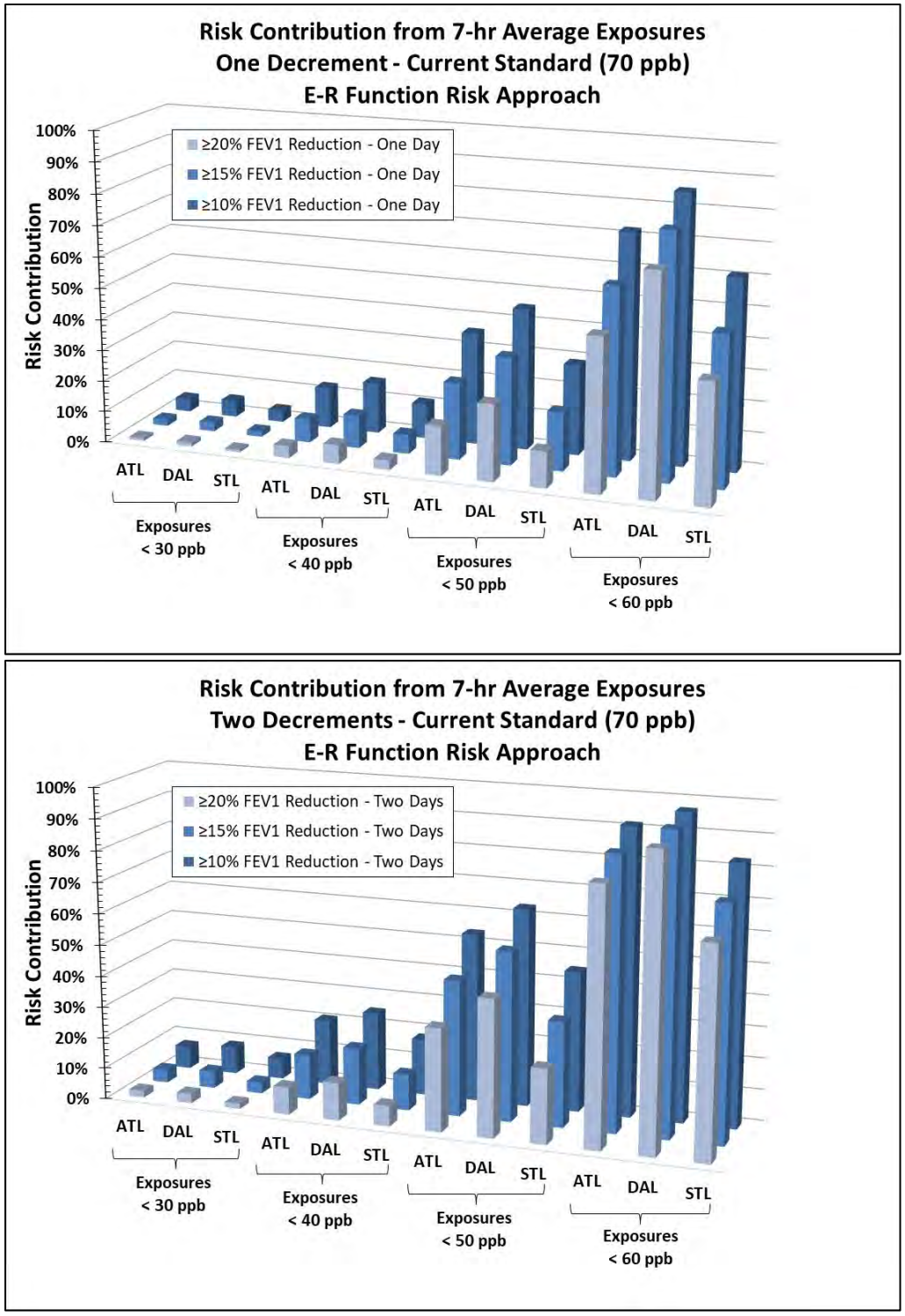

Figure 3D-15. Estimated lung function risk contribution resulting from selected 7-hr average O_3 exposures in children, using the E-R function risk approach and air quality adjusted to just meet the current standard, for one decrement (top panel) and two decrements (bottom panel), 2016.

As was done with the E-R function results, we evaluated the influence exposure level has on risks estimated using the MSS model. New APEX simulations were performed to estimate the continuous hourly time-series of O_3 exposures and FEV_1 decrements. All simulation conditions remained the same as done for the main body risk results except that for this evaluation, a single year of air quality (2016) was used and fewer children were simulated to maintain a tractable analysis (10,000 rather than the 60,000 done for the main body results). Note, there is little difference in risks estimated when varying the total number of simulated children (Table 3D-67). Because the risk estimated using the MSS model is calculated from a cumulative time-series of O_3 exposures (and EVR, along with contributions from other variables used by the MSS model), we calculated the 7-hr average O_3 exposure occurring just prior to the FEV_1 decrements to allow for reasonable comparison with the above E-R function risk contribution results.

Table 3D-68 and Figure 3D-16 present the risk contribution resulting from selected 7-hr average O_3 exposures that occur prior to a lung function decrement of interest, estimated using the MSS model. While the general pattern in the risk contributions across the air quality scenarios, study areas, and decrements are similar to that described above using the E-R function approach, there are noteworthy differences between the two risk approaches. First, there is less variability in the risk contribution values across the study areas and decrements when using the MSS model risk approach. For example, the overall coefficient of variation (COV; standard deviation/mean) ranges from 1 to 31% (mean 11%) across study areas when evaluating the MSS model risk contributions, while the COV ranges from 6 to 49% (mean 26%) for the same evaluation using the E-R function. Second, the MSS model consistently calculates a greater percent of lung function decrements that result from low O_3 exposures (Table 3D-68) relative to that estimated when using the E-R function (Table 3D-66). While the majority of risk (84 to 98%, mean 91%) using the E-R function risk approach was attributed to 7-hr average exposures ≥40 ppb, when using the MSS model, between 33 to 75% (mean 54%) of risk is attributed to 7-hr average exposures ≥40 ppb when considering the three air quality scenarios and all three decrements. Based on this evaluation, the MSS model more frequently predicts responses to occur at lower O_3 exposures than does the E-R function approach.

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 ≥10%	FEV_1 ≥15%	FEV_1 ≥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%

Table 3D-68. Estimated lung function risk contribution resulting from selected 7-hr average O_3 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%

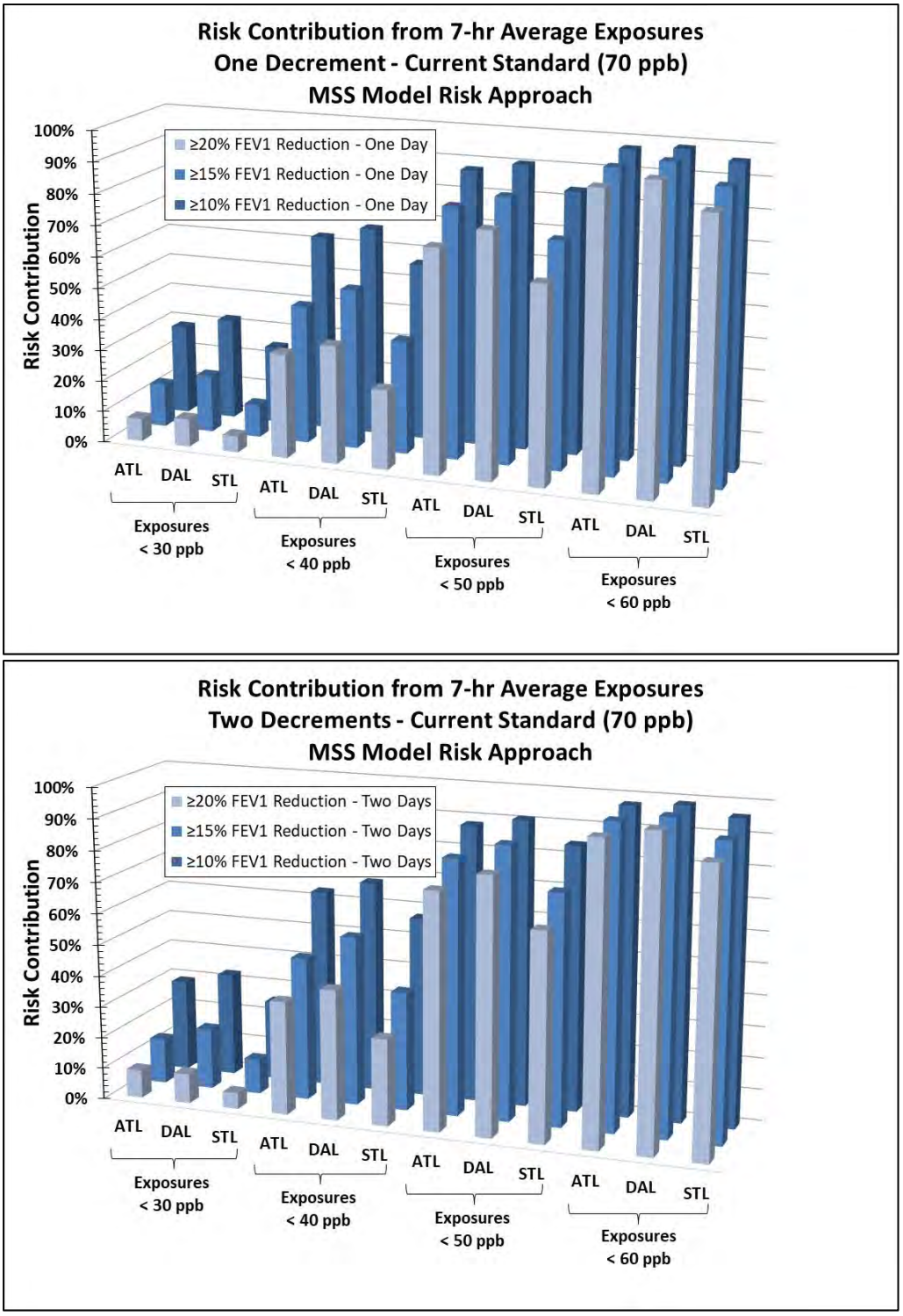

Figure 3D-16. Lung function risk contribution resulting from selected 7-hr average O_3 exposures in children, using the MSS model risk approach and air quality adjusted to just meet the current standard, for one decrement (top panel) and two decrements (bottom panel), 2016.

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 ≥17.32 ± 1.25 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).

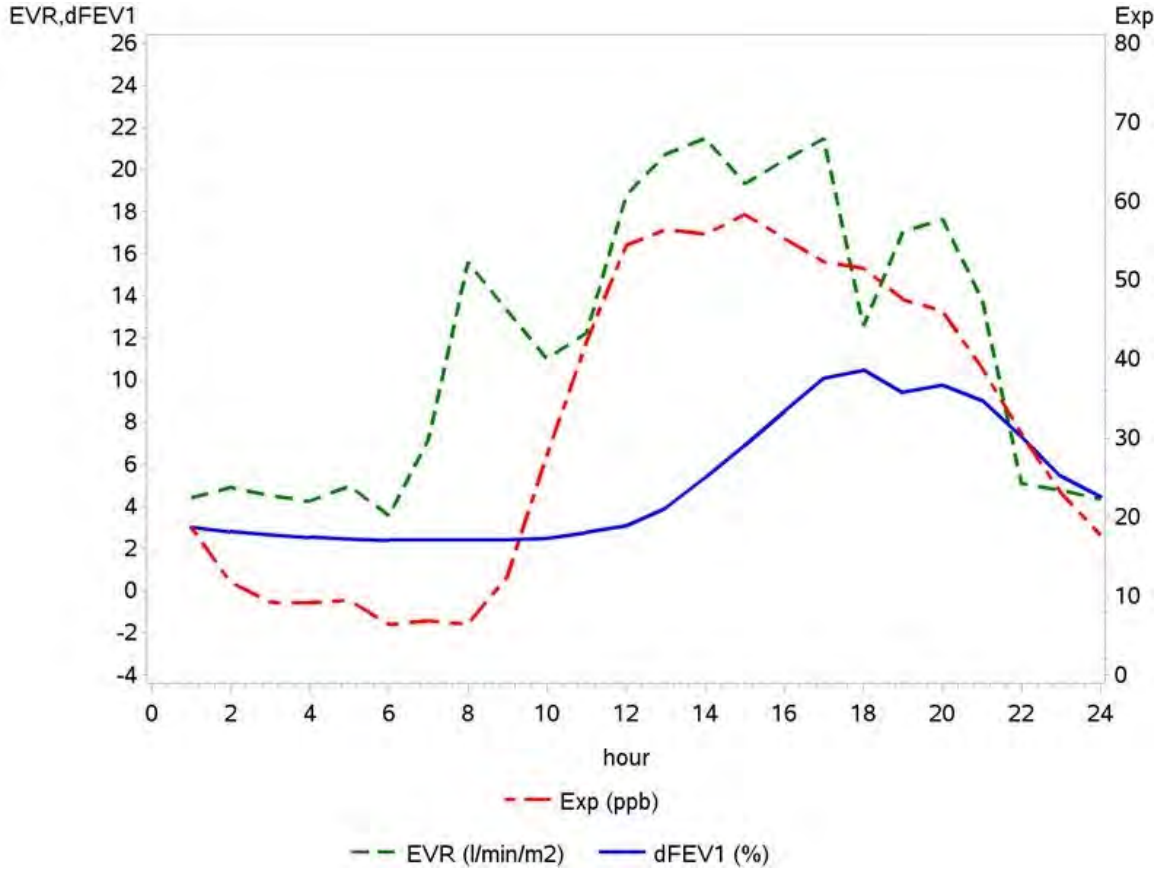

Figure 3D-17. Example time-series of O_3 exposures, EVR, and FEV_1 reductions estimated using MSS model for a simulated child in the Atlanta study area, based on a day in a year (2016) of the current standard air quality scenario.

When considering the influence of EVR in isolation, we can discern how, in many instances, the MSS model risk estimates are greater than those estimated using the E-R function approach when both use a generally similar O_3 exposure profile (i.e., any level, though using different averaging times). Recall, that the E-R function risk is only estimated for those attaining moderate or greater exertion levels EVR ≥17.32 ± 1.25 L/min-m^2. While there is likely a minimum EVR in the MSS model, considering both the level and duration, that would lead to lung function decrements, that minimum is not explicitly defined as it is in the E-R function risk approach.

Note, the E-R method is a fairly direct translation of the controlled human exposure study data to exposure-dependent response probabilities, particularly considering the strict adherence to exertion level needed for a response. As described above (section 3D.3.4.2.2), there is low statistical uncertainty associated with the risk estimates. We already know that relatively lower ventilation rates substantially influence MSS model risk estimates based on analyses described in the 2014 HREA (Chapter 6, Tables 6-9 and 6-10). In that assessment, when restricting the MSS

results to when an 8-hr EVR of at least 13 L/min-m^2 was not achieved by simulated individuals (at that time, the threshold for moderate exertion threshold), about 40 to 50% fewer simulated individuals were estimated to experience a lung function decrement, a result better aligned with the E-R function risk results.

As a second evaluation of the influence of EVR, a similar evaluation of the degree to which low-level EVRs influence MSS risk estimates was performed here. We limited the evaluation to a single year (2016) of air quality adjusted to just meet the current standard in the three study areas and using the same simulation of 10,000 children described above for generating the hourly data for the MSS model lung function risks. We identified the days when children were exercising at moderate or greater exertion, i.e., 7-hr average EVR ≥17.3 L/min-m^2 and calculated the percent of children experiencing one or more lung function decrements of interest (i.e., ≥10%, ≥15%, and ≥20%). Results for each the main body MSS model approach and the MSS model restricted to children at moderate or greater exertion are presented, along with results using the E-R function risk approach (Table 3D-69).

The pattern of risk estimates was consistent across the three study areas. Using the Atlanta study area results as an example, the E-R function risk approach predicts the percent of children experiencing one or more FEV_1 decrements ≥10% to be 2.5%, while the main body MSS model risk approach predicts 14.6% of children experience the same decrement (Table 3D-69). When using the MSS model and restricting the risk results to children at moderate or greater exertion, 8.5% of children experiencing one or more FEV_1 decrements ≥10%. Even with this adjustment for moderate or greater exertion, this indicates an uncertainty in the MSS model estimates such that the MSS model is potentially overpredicting risks for children by about a factor of three or more, particularly when considering the larger lung function decrements.

Note that the MSS model used an age-term that extends information developed for 18-year olds to estimate lung function risks in the simulated children (ages 5 to 18). The age term at age 18 is at a maximum value and progressively decreases in value (and hence risk) through age 35 adults (the age range of study subjects in the controlled human exposure studies). Therefore, use of this extrapolation might also contribute to some of the noted differences in the two risk approaches because this approach uses the maximum possible observed value. However, the 2013 ISA indicates children's responses to O_3 exposure are similar to those for young adults (2013 ISA, section 8.3.1.1), which lends credence to use of the age-term extrapolation in the MSS model and, overall, supports the application of E-R risk approach for children.

Table 3D-69. Percent of children experiencing one or more FEV_1 decrements ≥10, 15, 20%, 2016 air quality adjusted to just meet the current standard, considering influence of moderate or greater exertion level in the MSS model and E-R 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)).[85] 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

[85] 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 $\nu_1 + \nu_2 \times (e^{Ui} \times M_{ijk})$ (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."

values for *U* would likely yield high lung function decrements, particularly for instances of high O_3 exposures that occur simultaneously with high ventilation rates over a few to several hours. Note that when comparing the variance of *U* in the 2012 MSS model versus the 2013 MSS model, its standard error is greater (0.917 versus 1.123) in the most recent model.

For this evaluation, we used the same APEX simulation (as described in the prior section) of 10,000 children (and 2016 air quality), which output the hourly time series of O_3 exposure, EVR, and FEV_1 decrements for each simulated individual. We screened the output data for simulated individuals having experienced each of the three FEV_1 decrements of interest (i.e., ≥10%, ≥15%, and ≥20%) and occurring on separate days. We recognize there are a limited number of children experiencing lung function decrements on multiple days per year (e.g., Table 3D-57), particularly when considering the highest lung function decrement, but we were interested in controlling for the influence personal variables might have on the magnitude of each of the decrements. We identified a few simulated individual children having multiple decrements at each level of interest, and first visually compared how variation in the value assigned the *U* variable appeared to influence the magnitude of FEV_1 reduction for the subset of these simulated children that had similar time-series of O_3 exposure and ventilation rate.

As an example, Figure 3D-18 illustrates the estimated hourly time-series of O_3 exposure, EVR, and FEV_1 decrement for two simulated children (top and bottom panels) that differ in the value they were assigned for the *U* variable (both runs used the 2016 year for the current standard air quality scenario for the Atlanta study area). In both cases, the O_3 exposure and EVR are well correlated for each child prior to the occurrence of a lung function decrement, consistent with the controlled human exposure study data. With increasing magnitude of the FEV_1 decrement (Figure 3D-18, from left to right panels) there is also progressively higher exposures and breathing rates, each occurring as peak events that continue over a few to several hours just prior to eliciting the indicated FEV_1 decrement of interest. In general, for each of the three magnitudes of FEV_1 decrement, the time-series of O_3 exposures appears similar for the two simulated children – a consistently high exposure maintained across multiple hours for all of the instances where a lung function decrement occurred, with the highest decrement achieved when exposures were also highest. There is however a recognizable difference in the EVR time-series for the two simulated children. For the first child, with the lower value for the *U* variable (top panels, Figure 3D-18), the peak of the EVR time-series is broader, that is, longer in duration, than it is for the peak EVR for the second child (bottom panels, Figure 3D-18). The peak EVR for the second child (that has the higher value for the *U* variable) is similar in magnitude to that for the first child, but it does not persist over as long a duration. The figure illustrates this difference for three magnitudes of decrement (10%, 15% and 20%) in vertical pairs of panels from left to right, with the pairs of upper and lower panels differing only by the value of

parameter U. Specifically, the lower panel child achieves the same decrement as the upper panel child but while having a lower average EVR for the event.

The first simulated child (upper panel) has a *U* value of 0.963, which falls within one standard deviation of the distribution of *U* (i.e., *U* has a standard error of 1.123, Table 3D-21). The second (lower panel) simulated child has a *U* value of 1.78, within the *U* variable parameterization (i.e., within 2 standard deviations), but is nearly twice that of the first child. Specifically, while the second child has a lower overall "normalized dose" (i.e., $C \times V^{\beta 6}$ in Equation 3D-12) over a similar exposure duration as the first child, the similar risk result is likely a result of the second child being assigned a higher value for *U*. This higher value of *U* yielded lung function decrements for the second child similar in magnitude to that predicted for the first simulated child even though the second child had relatively lower doses than the first child for each of the three days.

The second variable, ν_1, a constant, is used on the calculation of the intra-individual variance term ε (Equations 3D-16). In evaluating the MSS model parameters used for this assessment, McDonnell et al. (2013) notes the estimate of ν_1 is consistent with intra-subject FEV_1 variability observed in the forced air trials and below threshold O_3 exposures. The variable ν_1 could be interpreted to represent a separate, non-ozone related contribution to response variability in the study observations. This suggests the use of non-zero values for ν_1, as is provided by McDonnell et al. (2013) in MSS model applications (and as was done for the current risk analysis), could lead to a greater number of simulated individuals at or above the lung function decrements (in particular the lowest decrement) and a greater portion of that risk would be attributed to relatively lower exposure levels and ventilation rates, when compared to simulation results having ν_1 set as zero.

We evaluated the influence that the value of ν_1 has on risk estimates. A new APEX simulation was required for this evaluation. All model settings were the same as was done for generating the main assessment results reported in section 3D.3.3.2, except for varying the value of ν_1 (the MSS model default ν_1 value is 9.112, a new simulation had ν_1 set as zero) Again, both simulations were performed for 10,000 children in three study areas (Atlanta, Dallas, and St. Louis) for a simulated year using 2016 air quality adjusted just meet the current standard. Results for this evaluation are presented in Table 3D-70.

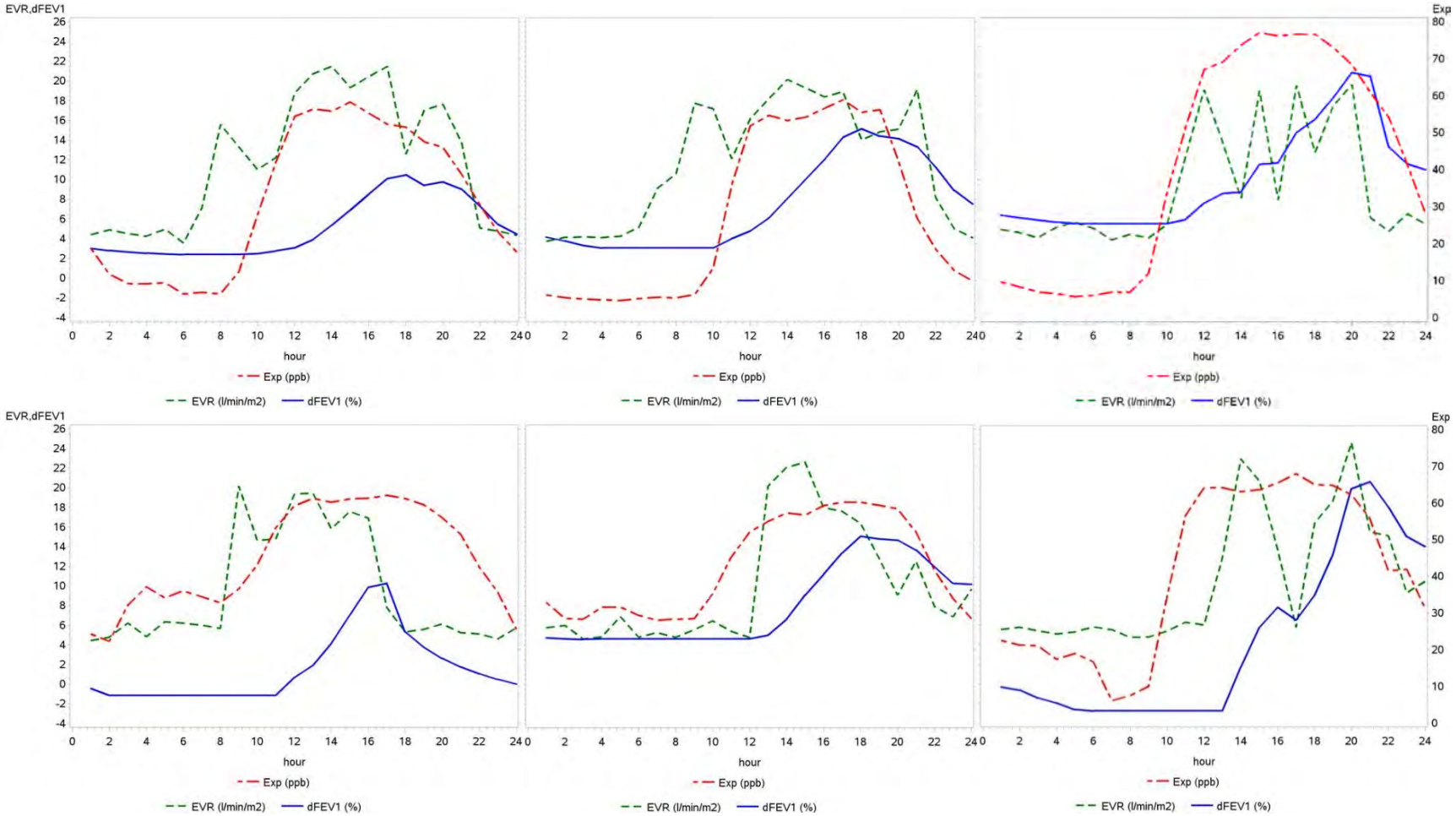

Figure 3D-18. Time-series of O_3 exposures, EVR, and FEV_1 reductions of 10% (left panel), 15% (middle panel), and 20% (right panel) estimated using MSS model for two simulated children (interpersonal variability parameter *U* = 0.963, top panel; *U* = 1.78, bottom panel) in the Atlanta study area on three days in a year (2016) of the current air quality scenario.

For each value of v_1, there were small differences in estimated risk across the three study areas. However, setting the v_1 to zero (compared to the value reported by McDonnell et al., 2013) resulted in a decrease in the percent of children experiencing lung function decrements of ≥10, ≥15, and ≥20% of about 35, 22, and 20% (regardless of study area). This reduction in risk is similar in magnitude to that resulting from excluding the contribution from low-level exposures (section 3D.3.4.2.3) and not using ventilation rates below moderate or greater exertion (section 3D.3.4.2.4) when estimating lung function decrements using the MSS model.

Table 3D-70. Percent of children experiencing one or more FEV_1 decrements ≥10, 15, 20%, 2016 air quality adjusted to just meet the current standard, considering the setting of variability parameter, v_1, in the MSS model.

Study Area	MSS Model Parameter Setting [A]	Decrement (FEV_1 Reduction)		
		≥10%	≥15%	≥20%
Atlanta	v_1 = 9.112 (default)	15%	5.1%	2.1%
	v_1 = 0	9.7%	3.9%	1.7%
Dallas	v_1 = 9.112 (default)	13%	4.1%	1.7%
	v_1 = 0	7.9%	3.2%	1.3%
St. Louis	v_1 = 9.112 (default)	16%	5.8%	2.5%
	v_1 = 0	11%	4.6%	2.1%

[A] See Table 3D-21 and Equation 3D-16.

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 prevelance 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 O_3 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 SO_2 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 O_3 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 O_3 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 fours 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:

$$Beta = logit\ \{prob\ (asthma)\} = log\ \{prob\ (asthma) / [1 – prob\ (asthma)]\}.$$

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:

Linear in age	*Beta = $\alpha + \beta \times age$, where α and β vary with strata*
Quadratic in age	*Beta = $\alpha + \beta \times age + \gamma \times age^2$ where α β and γ vary with strata*
Cubic in age	*Beta = $\alpha + \beta \times age + \gamma \times age^2 + \delta \times age^3$ where α β, γ, and δ vary with the strata*
f(age)	*Beta = arbitrary function of age, with different functions for different strata*

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, sexr, 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 analyses[9] 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(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,

$$Prob\ (asthma) = exp(\ beta) / (1 + exp(beta)\ ),$$

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:

$$Std\ Error\ \{Prob\ (asthma)\} = Std\ Error\ (beta) \times exp(-\ beta) / (1 + exp(beta)\ )^2,$$

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), startified 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:

$$Weight = \{1 - [\ |age - A| / q\ ]^3\}, \text{ where } |\ age - A| <= q.$$

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(beta) / (1 + exp(beta)). In addition to the tri-weight variable, each beta value was assigned a weight of $1 / [\text{std error (beta)}]^2$, to account for their uncertainties.

In this application of LOESS, weights of $1 / [\text{std error (beta)}]^2$ 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 / estimated σ 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.

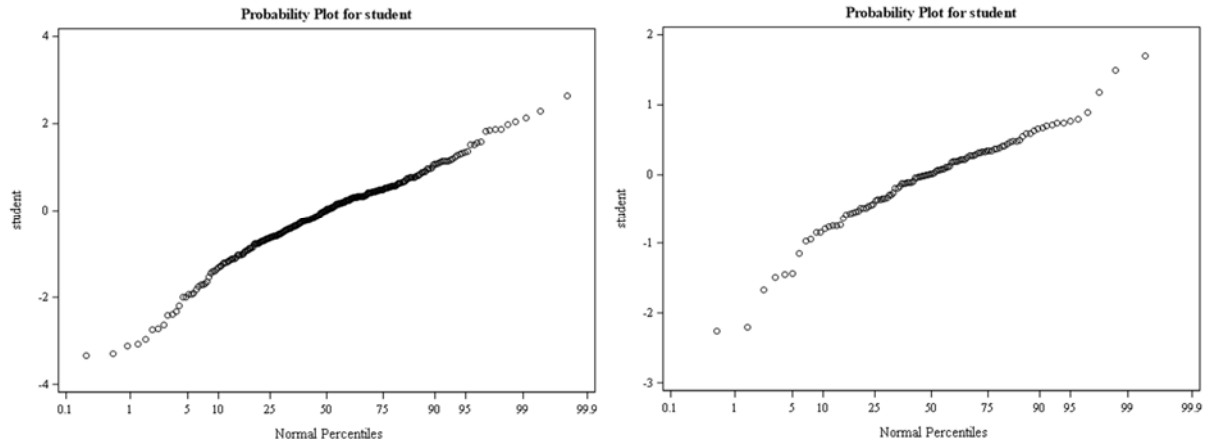

Figure 1. Normal probability plot of studentized residuals generated using logistic model, 'STILL' prevalence data, with smoothing set to 0.7 and 0.9 for children (left) and adults (right), respectively.

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.

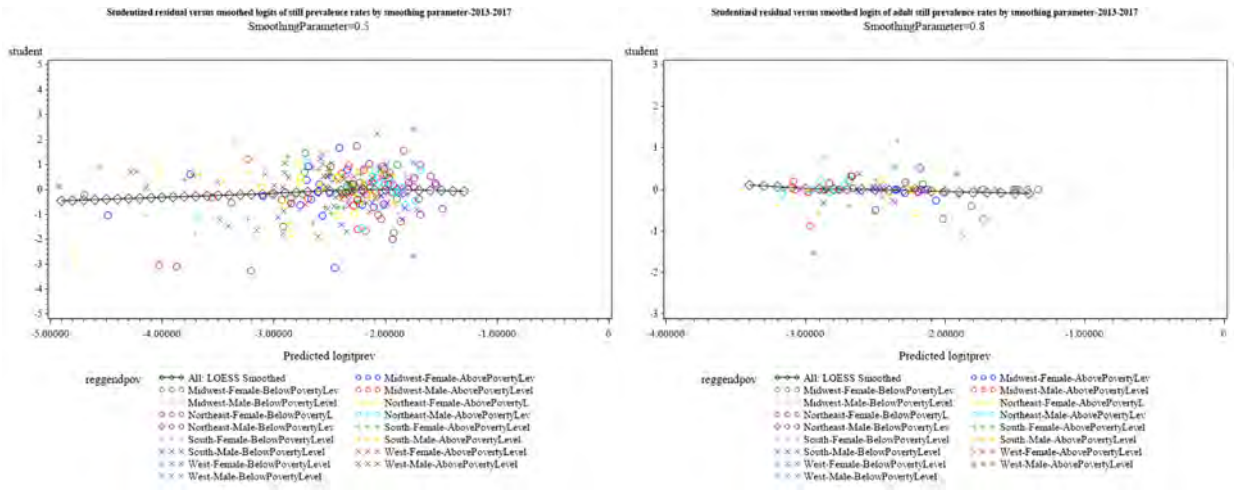

Figure 2. Studentized residuals versus model predicted betas generated using a logistic model and the 'STILL' prevalence data, smoothing set to 0.5 and 0.8 for children (left) and adults (right), respectively.

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 peformed 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 fle 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-weighed 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 aggreement 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:

$$State\ Adjustment\ Factor = (NHIS\_Census_{regional\ prevalence} - BRFSS_{state\ prevalence})/BRFSS_{state\ prevalence}$$

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
Coloroado	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: prevelance 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 O_3 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	95th percentile	99th 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 particpants 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. Chidren's responses to four questions regarding their asthma status, 2017 NHIS.

Diganosed 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.

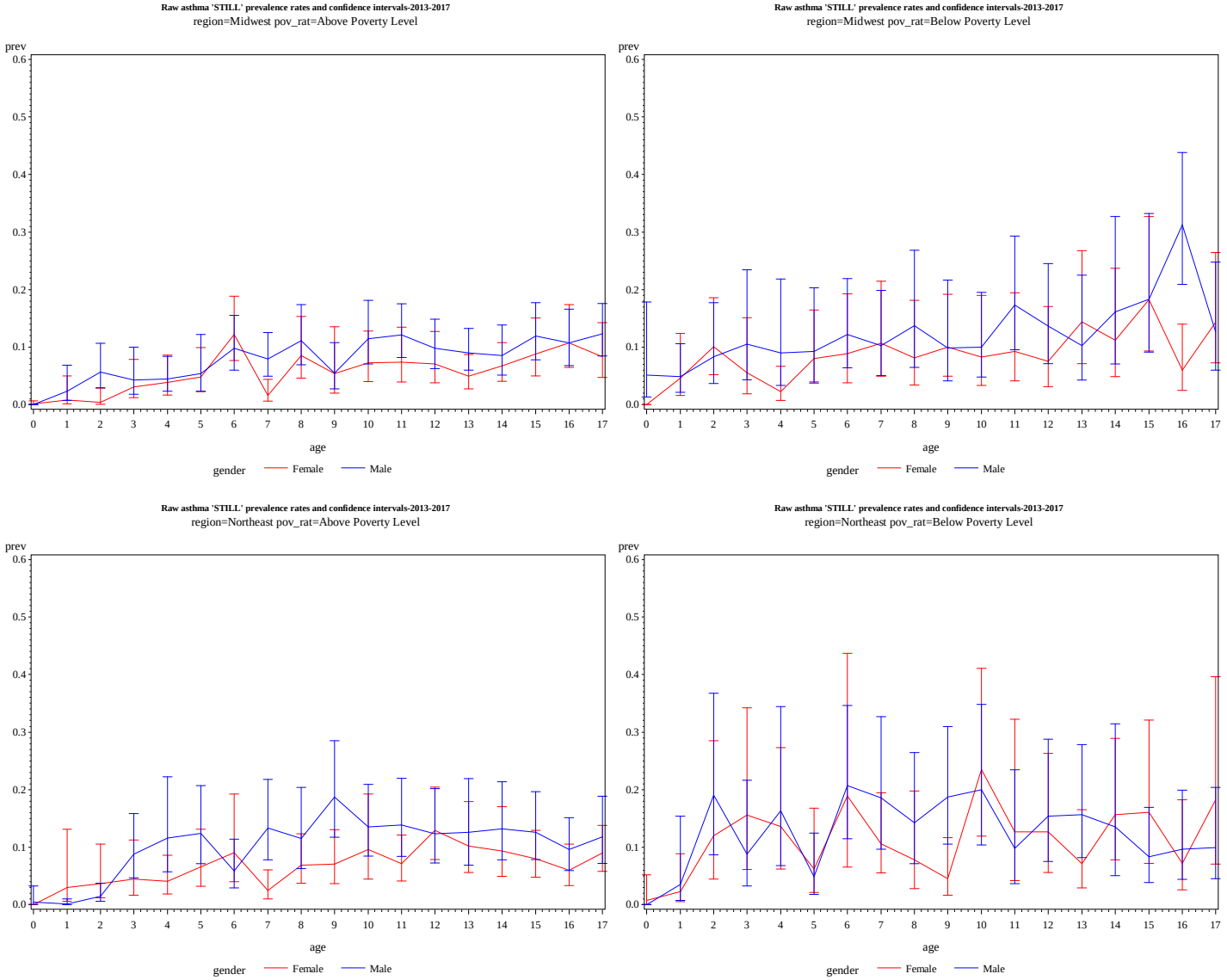


Figure S-2. Non-smoothed asthma prevalence for children that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.

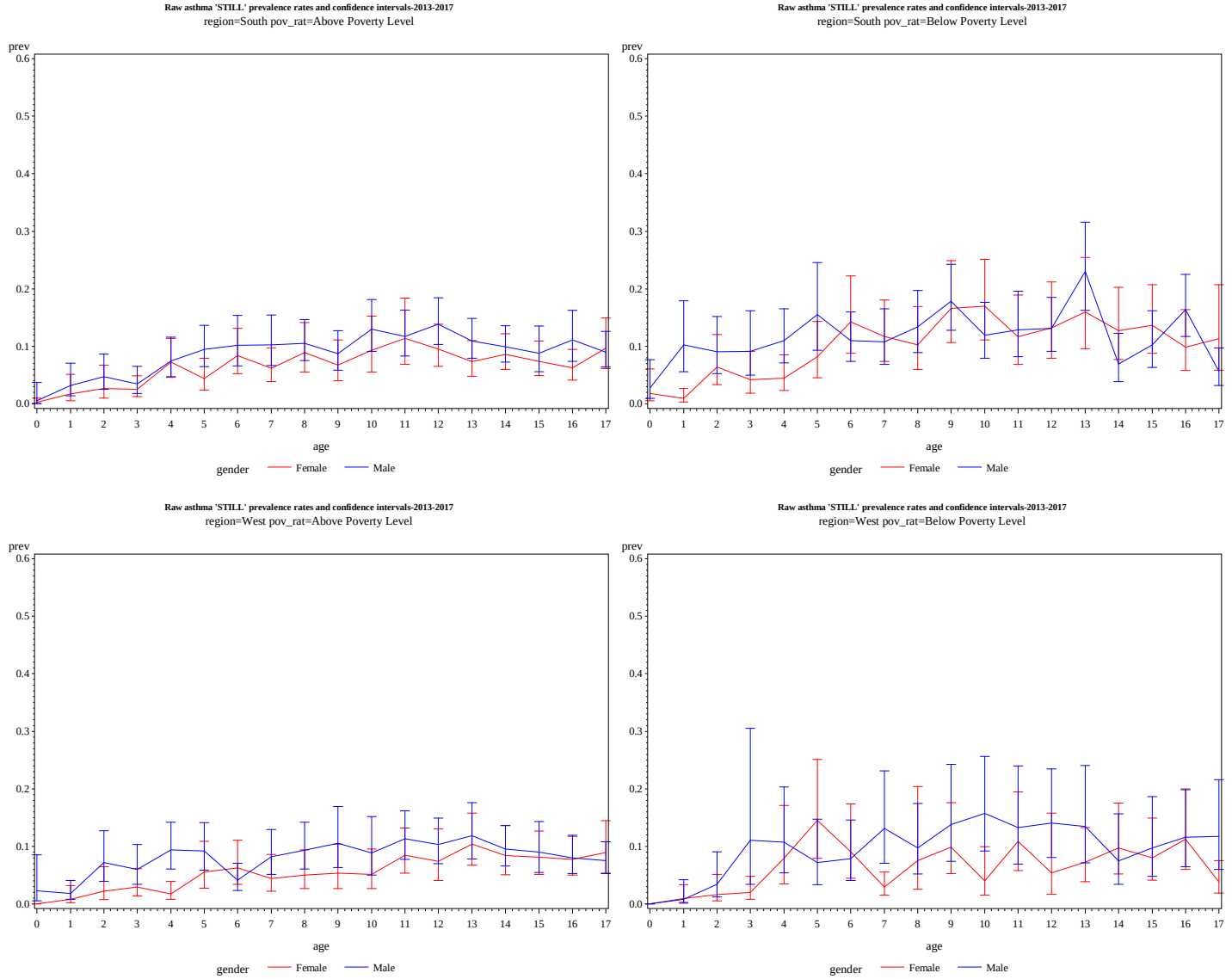


Figure S-3. Non-smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for Midwest (top panels) and Northeast (bottom panels) regions.

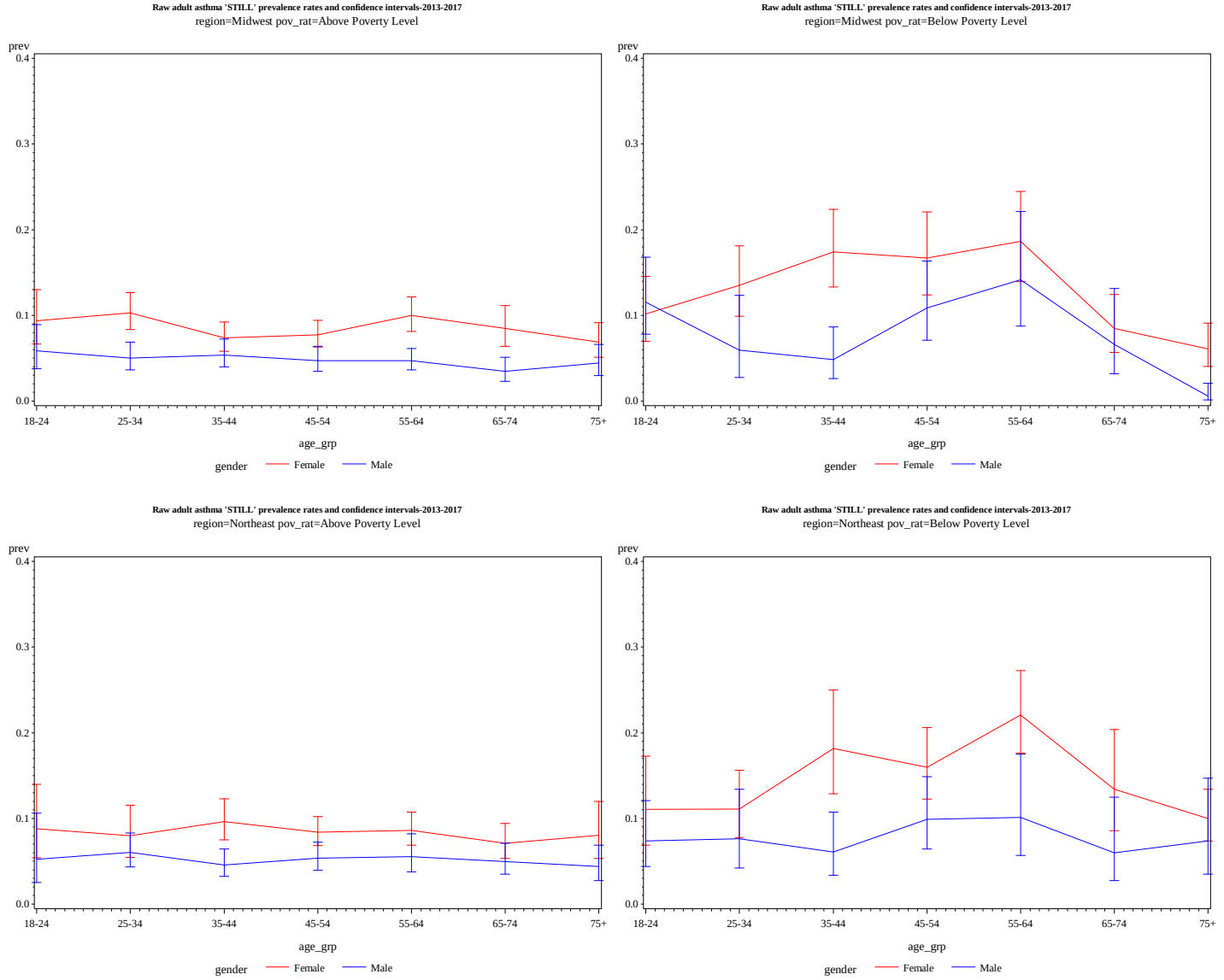
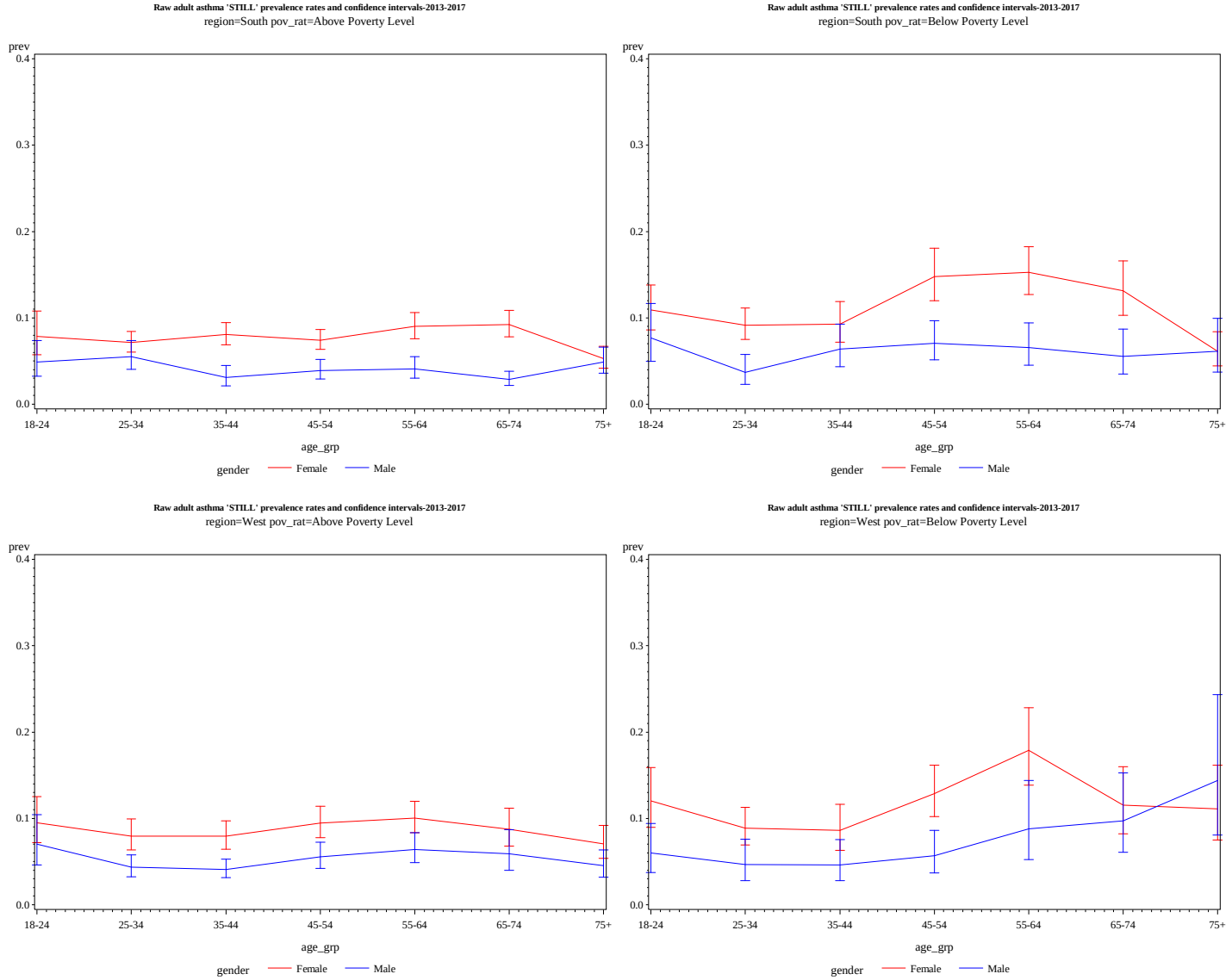


Figure S-4. Non-smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.



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.

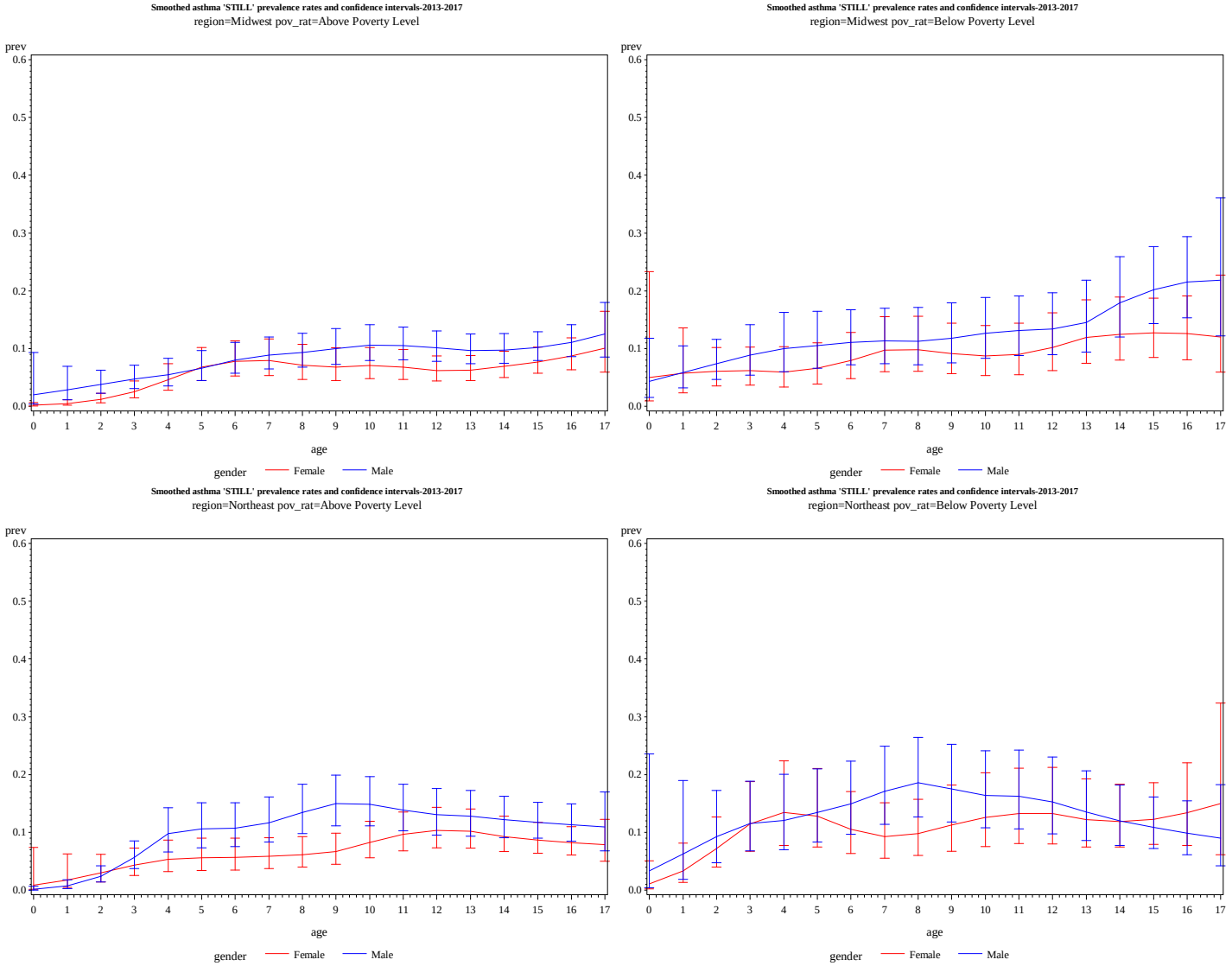


Figure S-6. Smoothed asthma prevalence for children that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.

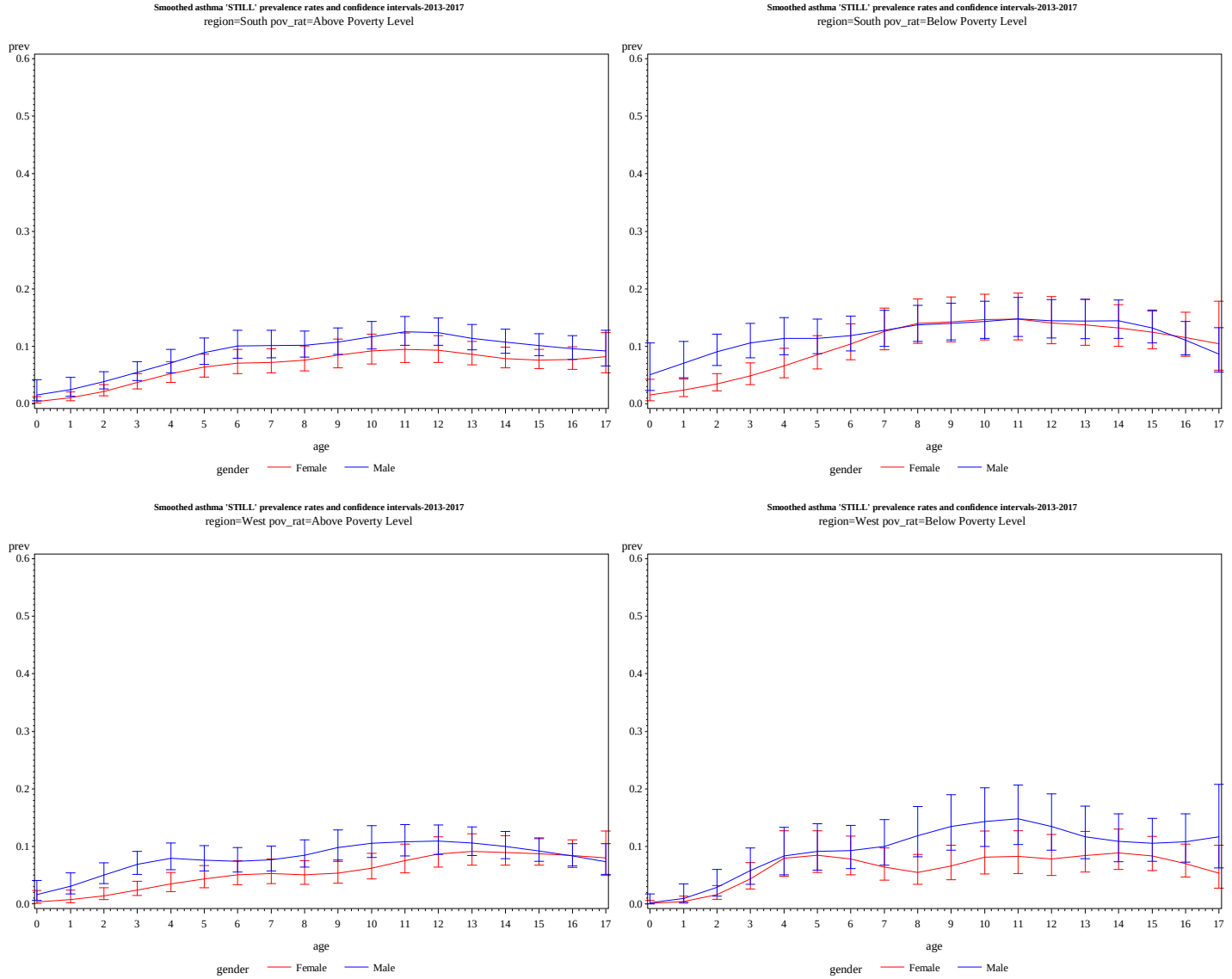


Figure S-7. Smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for Midwest (top panels) and Northeast (bottom panels) regions.

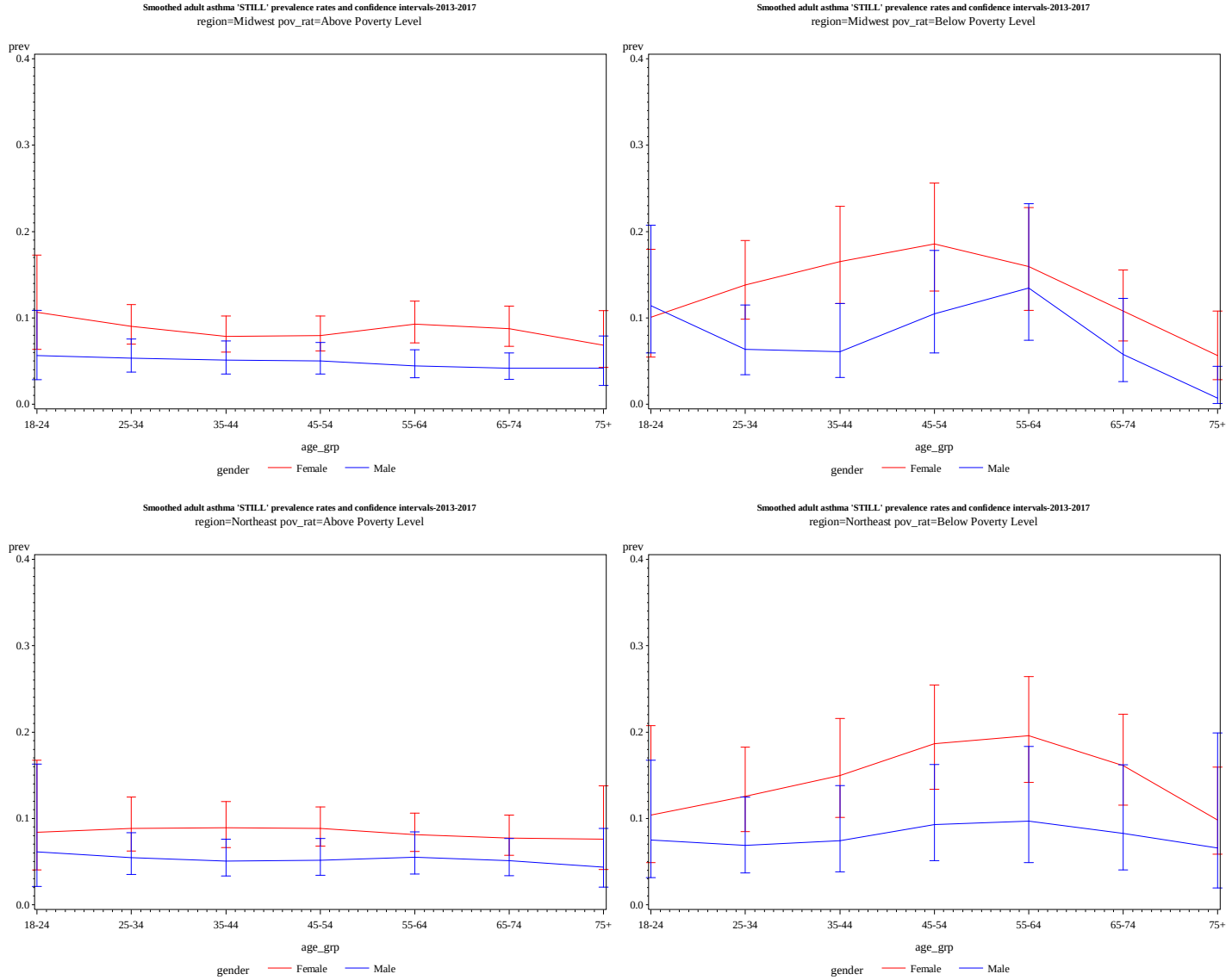
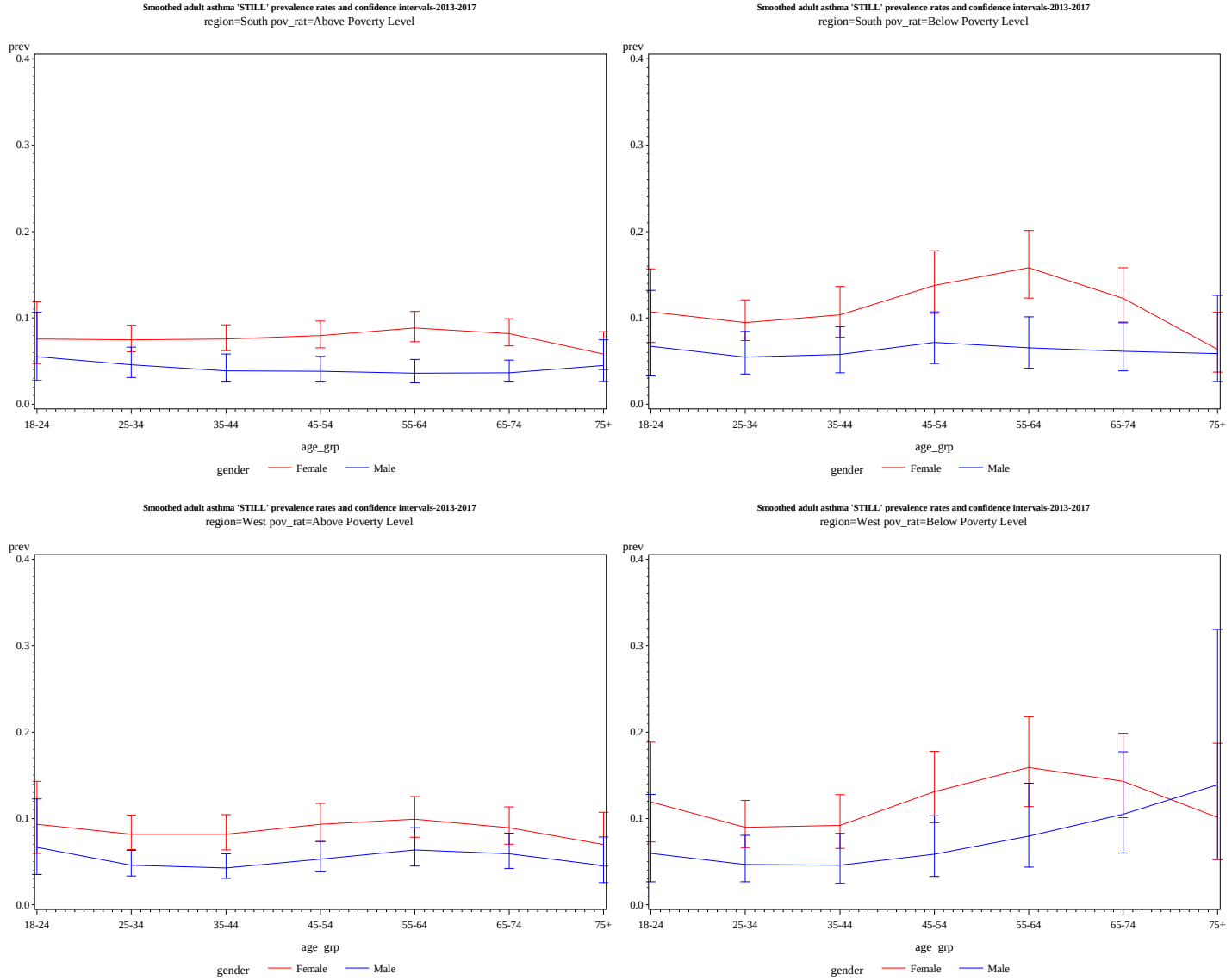


Figure S-8. Smoothed asthma prevalence for adults that still have asthma. Above (left panels) and below poverty level (right panels) for South (top panels) and West (bottom panels) regions.



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

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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-m^2, 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-m^2 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-m^2.

- A ***ModEVR8*** value of 13 L/min-m^2 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-m^2 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-m^2.

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-m^2 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; O_3 = 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-m^2 typically used in regulatory runs of APEX was based on the range of 13–27 L/min-m^2 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-m^2 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-m^2)			
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-m^2)			
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-m^2 = 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-m^2 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 $V_e = 7.61 \times BSA$, and for college-age females, this was $V_e = 8.05 \times BSA$. These resting EVR values, 7.61 and 8.05 L/min-m^2 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-m^2 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-m^2 = 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: ■ 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: ■ 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. ■ 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-m^2 = 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)

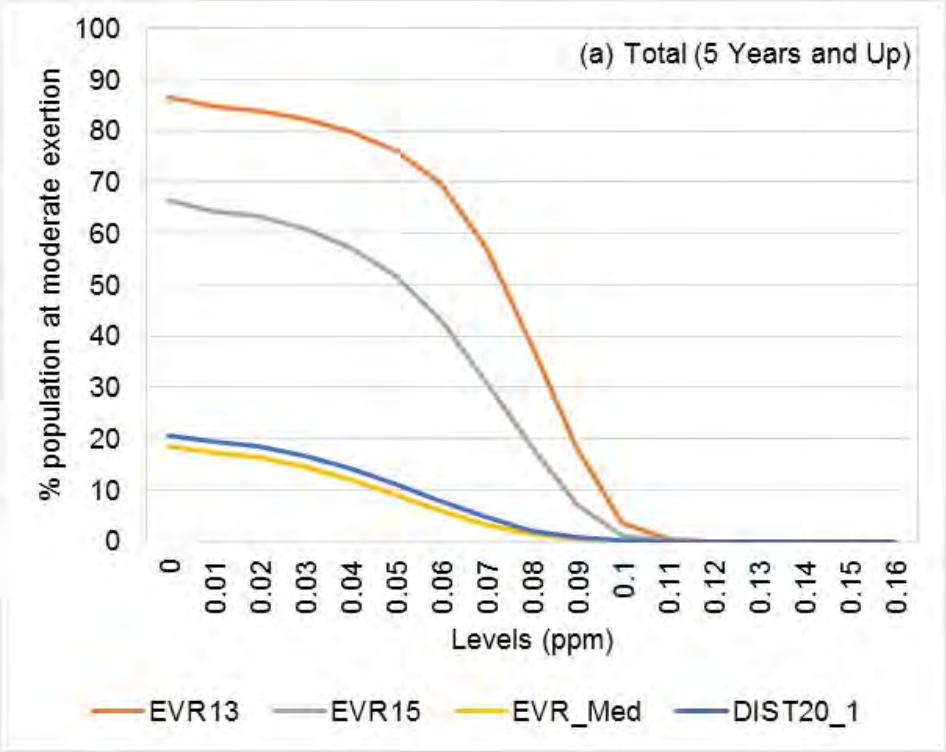
	Run Name (see Table 4)			
Level (ppm)	EVR13	EVR15	EVR_Med	DIST20_1
0	86.7	66.3	18.5	20.8
0.01	84.8	64.3	17.2	19.4
0.02	83.9	63.3	16.3	18.5
0.03	82.2	61.0	14.4	16.8
0.04	79.8	57.3	12.1	14.3
0.05	76.3	51.7	9.1	11.2
0.06	69.8	43.2	6.0	7.9
0.07	57.3	31.2	3.4	4.8
0.08	38.3	18.4	1.5	2.1
0.09	18.3	7.3	0.46	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

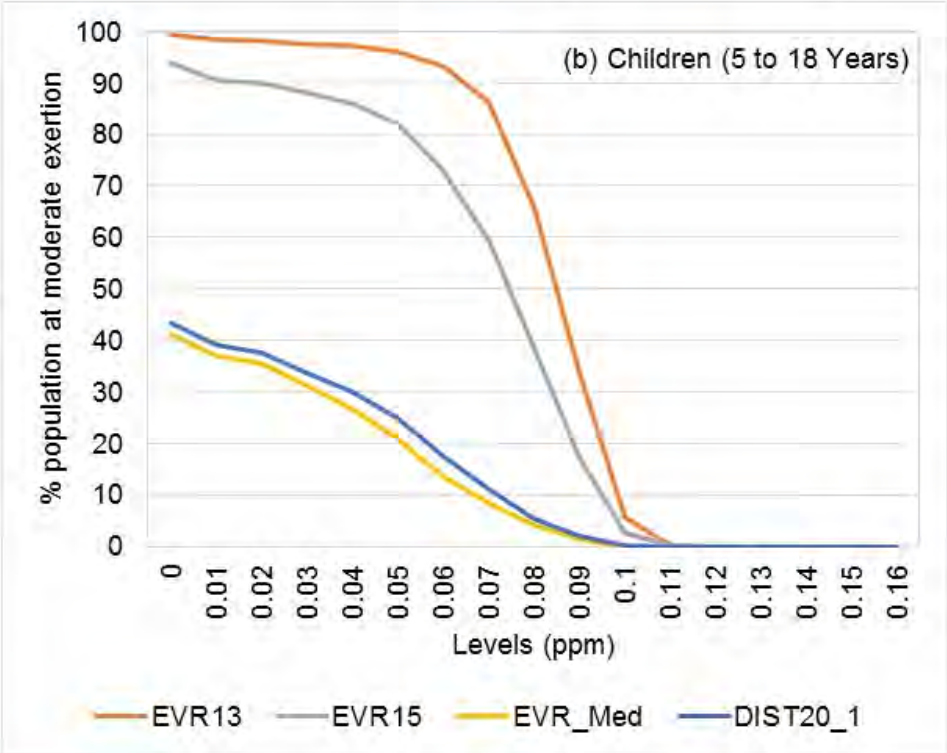
Notes: ppm = parts per million.
Shading indicates relative magnitude of values (reds and oranges are higher values; yellows and greens are lower values).

Table 6. Percent of Modeled Child Profiles (Ages 5 to 18 Years) Reaching Moderate Exertion

	Run Name (see Table 4)			
Level (ppm)	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)

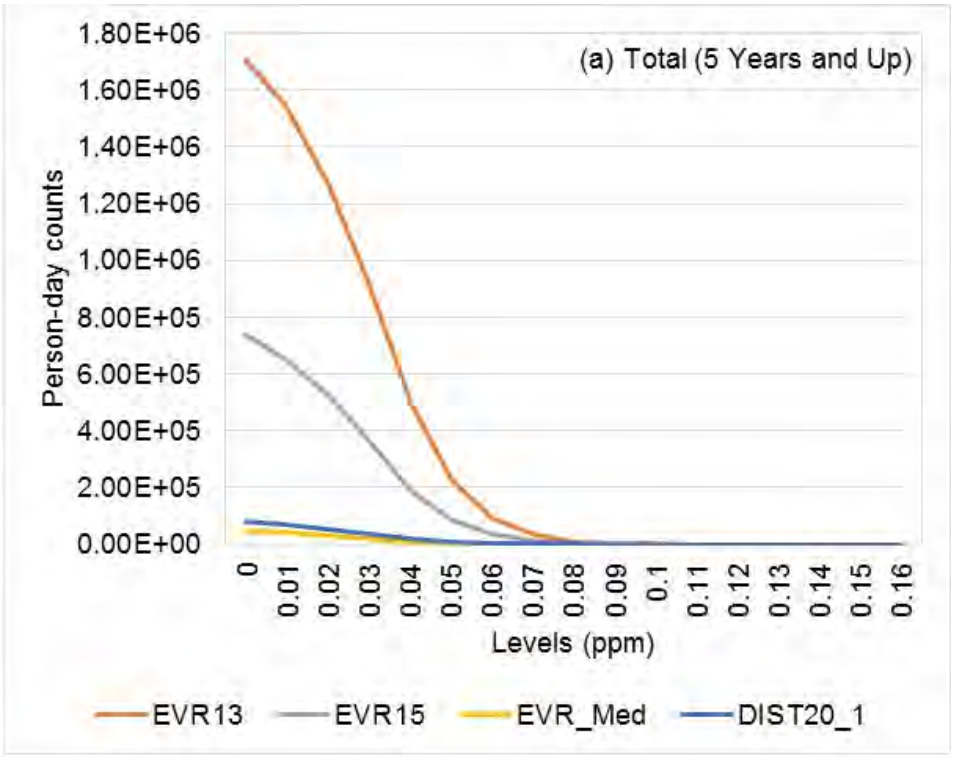
	Run Name (see Table 4)			
Level (ppm)	EVR13	EVR15	EVR_Med	DIST20_1
0	1.7E+06	7.4E+05	4.9E+04	8.1E+04
0.01	1.5E+06	6.5E+05	4.1E+04	6.9E+04
0.02	1.3E+06	5.3E+05	3.2E+04	5.4E+04
0.03	9.1E+05	3.6E+05	2.2E+04	3.6E+04
0.04	5.0E+05	1.9E+05	1.1E+04	1.8E+04
0.05	2.3E+05	8.6E+04	4.8E+03	8.0E+03
0.06	9.3E+04	3.4E+04	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

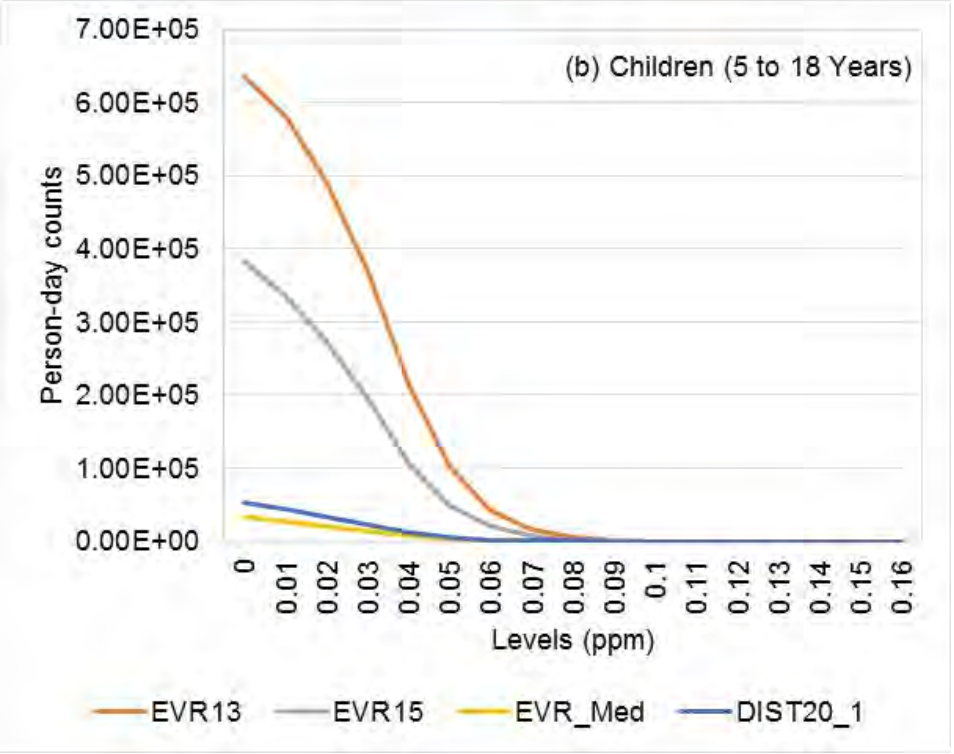
Notes: ppm = parts per million.
Shading indicates relative magnitude of values (reds and oranges are higher values; yellows and greens are lower values).

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; ºF) 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

			How County Locations Were Determined (showing counts of diary-days)		
	Count	Percent of All 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 polygons[1] 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/carto-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 24–50º N and 126–66º W (for contiguous U.S.), 51–72º N and 179.999–129º W (for Alaska; we did not use any stations in the far-western Aleutian Islands), and 18.5–22.5º N and 160.5–154.5º 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.

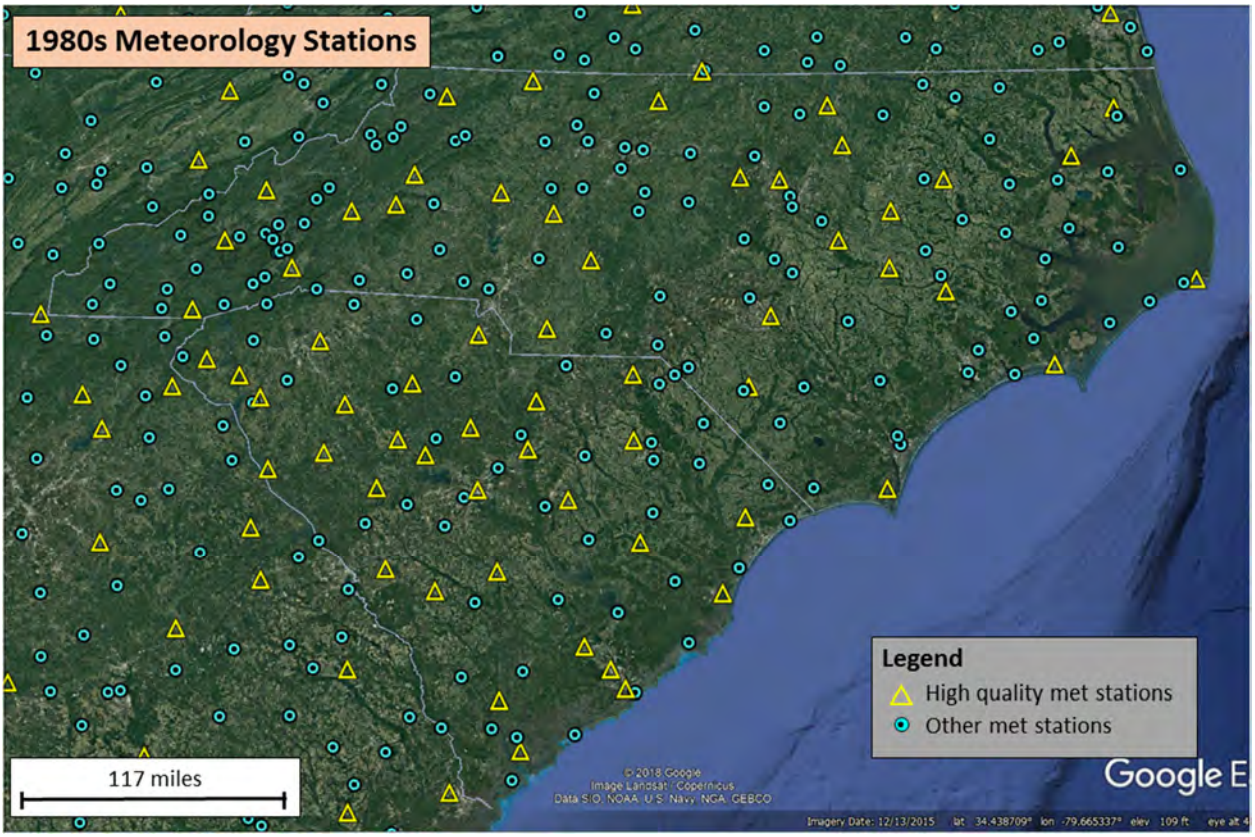

Figure 2-1. GHCND Meteorological Stations from the 1980s Meeting Selection Criteria, in the North and South Carolina Region

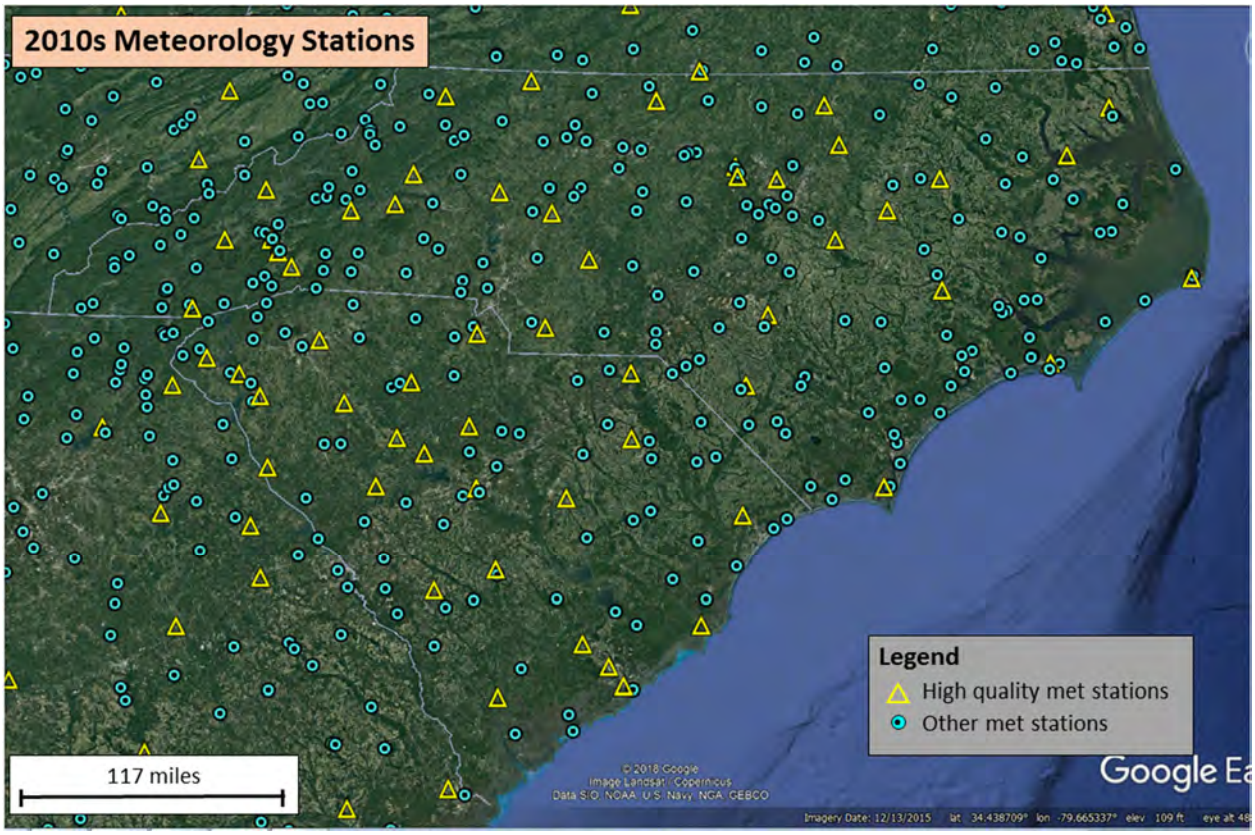

Figure 2-2. GHCND Meteorological Stations from the 2010s Meeting Selection Criteria, in the North and South Carolina Region

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 (≤ 5°), 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

	BLS		DEA		EPA		RTP		SEA		SUP	
Location Category	**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.

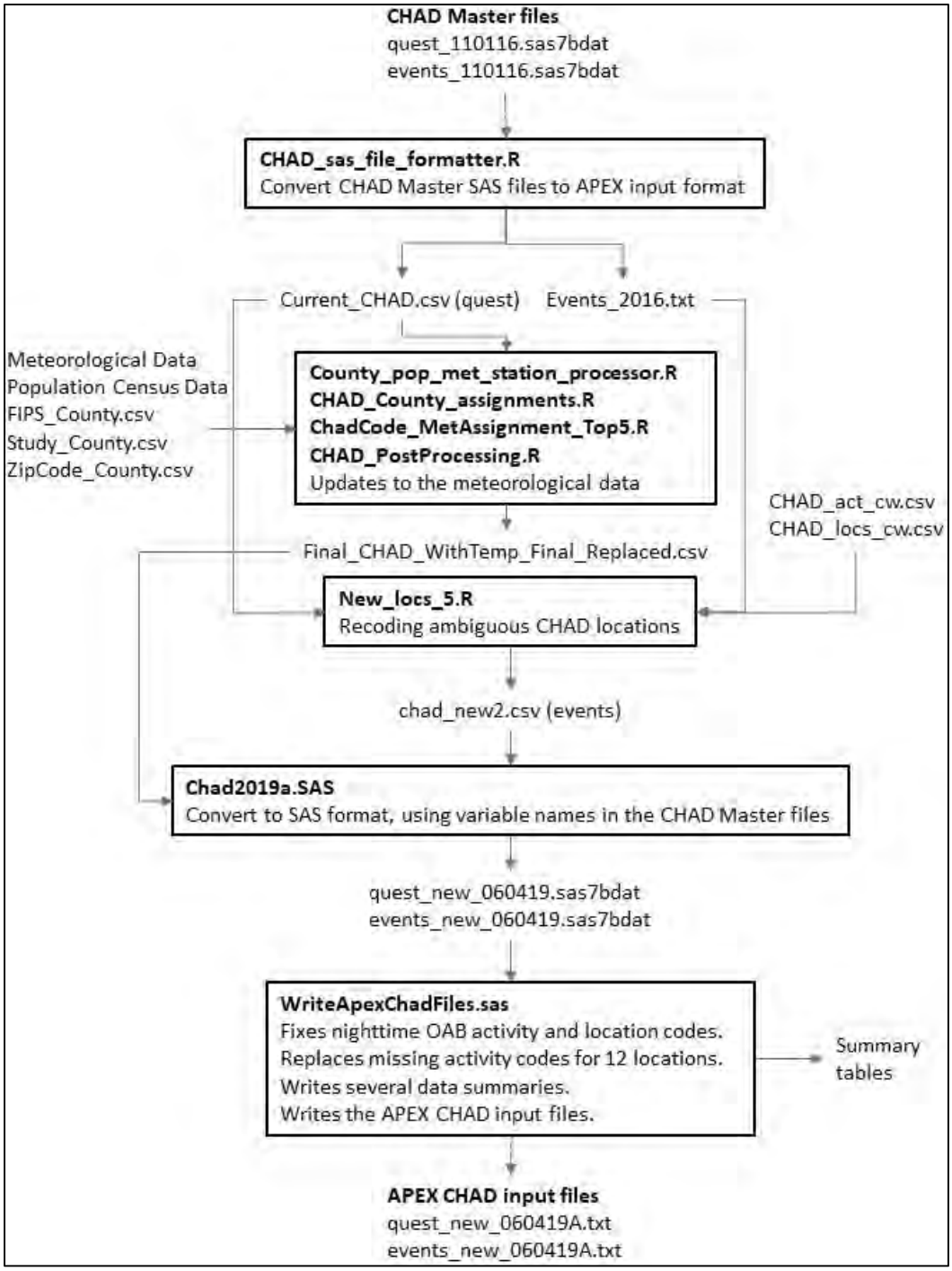

Figure 4-1. Files and Processing Programs Used in this Task

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					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
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					
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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					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
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					
					≥ 1 Day	≥ 2 Days	≥ 3 Days	≥ 4 Days	≥ 5 Days	≥ 6 Days
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

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	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					
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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

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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

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	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

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	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

Study Group	Study Area	AQ Scenario	Year	FEV_1 (percent)	Number of People at or above FEV_1 Decrement					
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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

Study Group	Study Area	AQ Scenario	Year	FEV_1 (percent)	Number of People at or above FEV_1 Decrement					
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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

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	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

APPENDIX 4A

EXPOSURE-RESPONSE FUNCTIONS FOR 11 TREE SPECIES AND TEN CROPS

TABLE OF CONTENTS

4A.1 BACKGROUND

Air quality criteria documents (AQCDs) for prior ozone (O_3) reviews have assessed and characterized the results of a series of studies on the growth effects of a range of seasonal O_3 exposure levels. These studies included research conducted by the National Crop Loss Assessment Network (NCLAN) on commercial crop species and by the EPA's National Health and Environmental Effects Laboratory Western Ecology Division (NHEERL/WED) on seedlings of 11 tree species[1]. These studies included documentation of hourly concentrations across the full exposures, and multiple exposure scenarios per experiment, which has resulted in their being the focus of work to characterize exposure-response (E-R) relationships for growth impacts on crops and tree species.

A subsequent set of publications analyzed the experimental study results to define a quantitative model that would well describe the E-R relationships of seasonal O_3 exposure and first impaired tree seedling growth and crop yield[2]. Those studies, which used several different metrics to quantify exposure (e.g., SUM06, W126), concluded that a three-parameter Weibull model form provides the most appropriate model for the response of absolute yield and growth to O_3 exposure because of the interpretability of its parameters, its flexibility (given the small number of parameters), and its tractability for estimation (2013 ISA, section 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

where:

Y = total yield or biomass;

W126 = O_3 exposure (e.g., 3-month sum of daily cumulative W126 from 8am to 8pm); and,

η and β are species-specific variables

With removal of the intercept term, α, the model estimates relative yield or biomass without any further reparameterization. In order to compare E-R functions and associated estimated 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 current 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).

absolute values of the response may vary greatly, the model is reformulated in terms of relative annual yield (or biomass) or relative yield (or biomass) loss (yield loss=[1-relative yield]). The resultant 2-parameter model of relative yield was presented in the 1996 and 2006 AQCDs and 2013 and current ISA as basis for deriving common models for multiple species, multiple genotypes within species and multiple experimental locations (2013 ISA, section 9.6.2; 2020 ISA, Appendix 8, section 8.13.2). The models presented in the AQCDs were in terms of SUM06 over a 3-month season; those models were updated for 12-hour W126 over a 3-month season in the 2013 ISA (2013 ISA, section 9.6.2). The 2-parameter model structure, for relative biomass loss (RBL) or relative yield loss (RYL) as a function of W126 is described in equation 4A-2.

$$RBL = 1 - \exp[-(W126/\eta)\beta] \qquad \text{Equation 4A-2}$$

Based on this model structure, functions for estimating RBL from seasonal W126 index, parameterized for each of eleven tree species, are presented and discussed in section 4A.1.1 below, and RYL functions for the 10 crop species are presented in section 4A.1.2.

4A.1.1 Tree Species Seedling E-R Functions

The RBL functions for each of 11 tree species were derived as median composite functions from response estimates based on functions derived for each study or experiment for which data were collected for that species (Lee and Hogsett, 1996, Tables 12 and 13). The eleven species-specific (composite median) functions, based on Lee and Hogsett (1996)[3] are presented in Table 4A-1.

Table 4A-1. RBL functions for tree species.

Species	RBL Function	η (ppm)	β
Red Maple (*Acer rubrum*)	$1 - \exp[-(W126/\eta)^{\beta}]$	318.12	1.3756
Sugar Maple (*Acer saccharum*)		36.35	5.7785
Red Alder (*Alnus rubra*)		179.06	1.2377
Tulip Poplar (*Liriodendron tulipifera*)		51.38	2.0889
Ponderosa Pine (*Pinus ponderosa*)		159.63	1.1900
Eastern White Pine (*Pinus strobus*)		63.23	1.6582
Loblolly Pine (*Pinus taeda*)		1,021.63	0.9954
Virginia Pine (*Pinus virginiana*)		1,714.64	1.0000
Quaking Aspen (*Populus tremuloides*), wild		109.81	1.2198
Black Cherry (*Prunus serotina*)		38.92	0.9921
Douglas Fir (Pseudotsuga menzeiesii)		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.

[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).

Figure 4A-1 presents species-specific E-R functions for the tree seedlings. The figures illustrate how the values of the two parameters affect the shape of the resulting curves. The value of η in the RBL function affects the point of the curve where the slope appreciably changes, and β affects the steepness of the curve. The response functions with smaller values of β (e.g., Virginia Pine) or with η values that are above the range shown for of W126 index values (e.g., functions for ponderosa pine and red alder) exhibit smaller slopes that have less change across this W126 range. These functions describe a more constant rate of change in RBL over the range of O_3 exposure shown (e.g., up to 30 ppm-hrs). In contrast, the response functions with larger β values (e.g., the function for Sugar Maple) exhibit a threshold-like behavior, with large changes in RBL over a small range of W126 index values and relatively small changes at other index values. In these cases, the "threshold" is determined by the η parameter of the model.

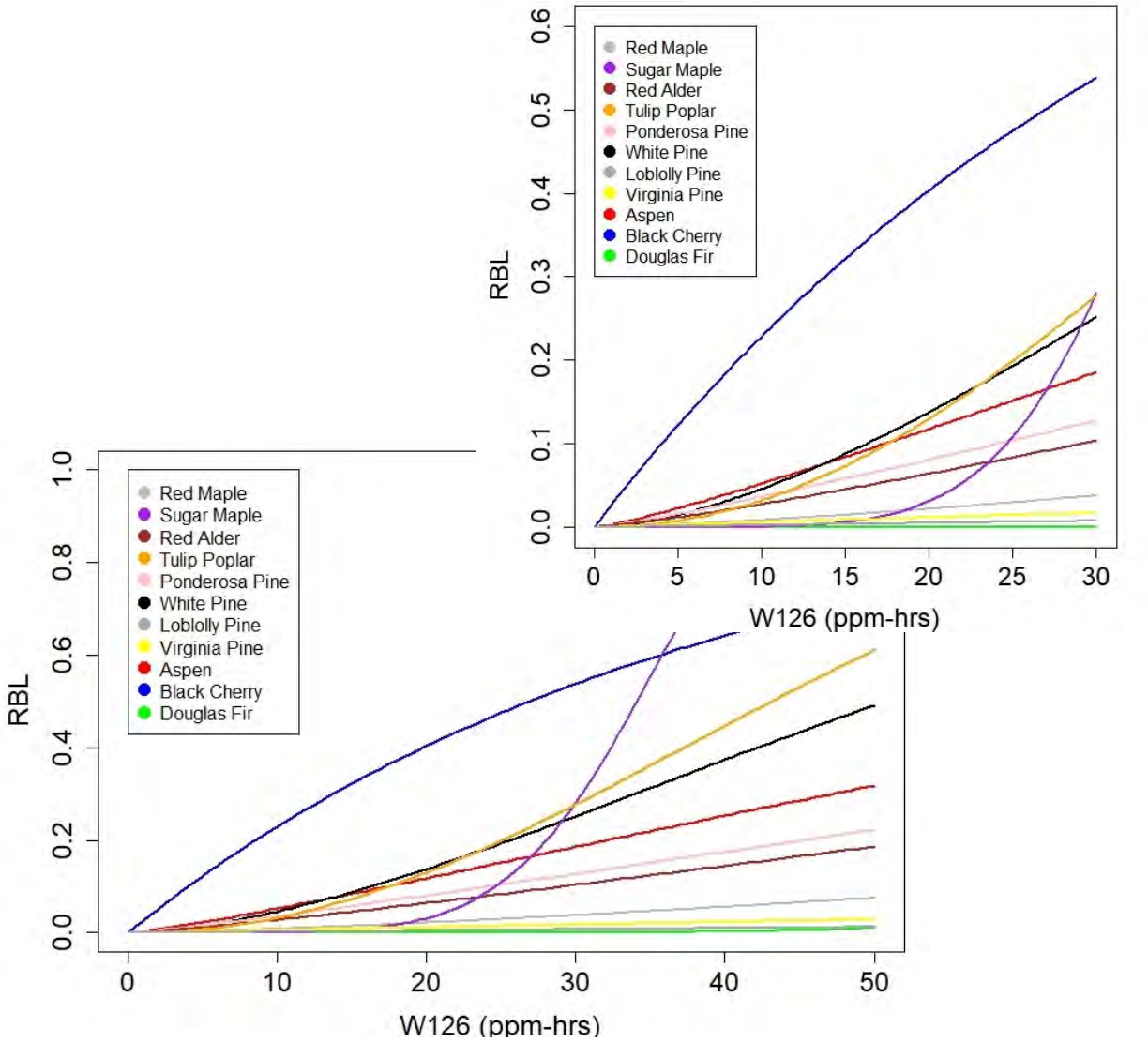

Figure 4A-1. RBL functions for seedlings of 11 tree species.

The shape of curves presented in Figure 4A-1 also illustrate how sensitive the RBL value is to changes in O_3. Two species, Loblolly Pine (dark grey line) and Virginia Pine (yellow line) have E-R functions that approach linearity within the W126 range represented on the x-axis, meaning that a 1 percent change in W126 produces an equal change in RBL. Black Cherry (blue line) has an E-R function with exhibits a declining slope with increasing W126, with the appearance of leveling off (Figure 4A-1), which produces a smaller change in RBL relative to the change in W126. The functions for the remaining species produce somewhat similar percent changes in RBL relative to changes in W126.

As mentioned above, the species-specific functions were derived from median estimates based on the functions from the individual experiments for each species. Figure 4A-2 through Figure 4A-12 present the species-specific functions along with the functions derived from the experiments available for that species.[4] These figures provide a sense of the across-experiment variability for each species, where such information is available.

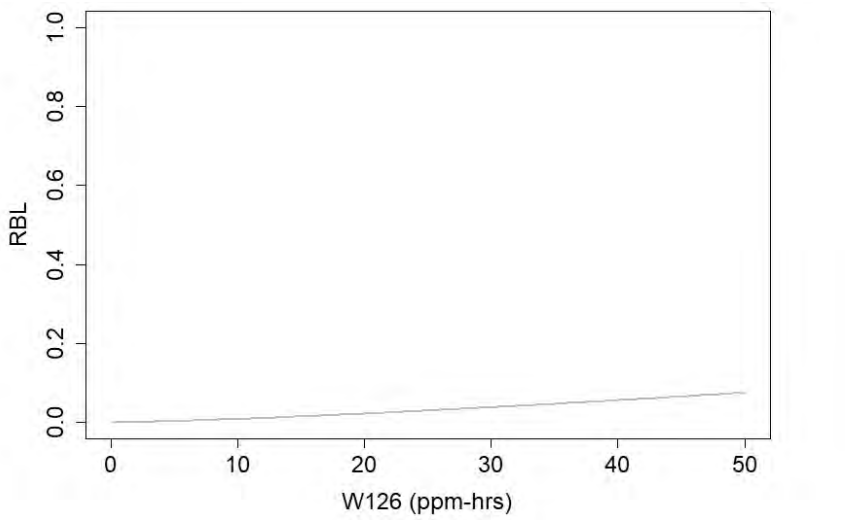

Figure 4A-2. RBL functions for Red Maple (*Acer rubrum*).

[4] For aspen, the dark (red) line shown in Figure 4A-1 is the median composite for wild (vs clonal genotype) studies.

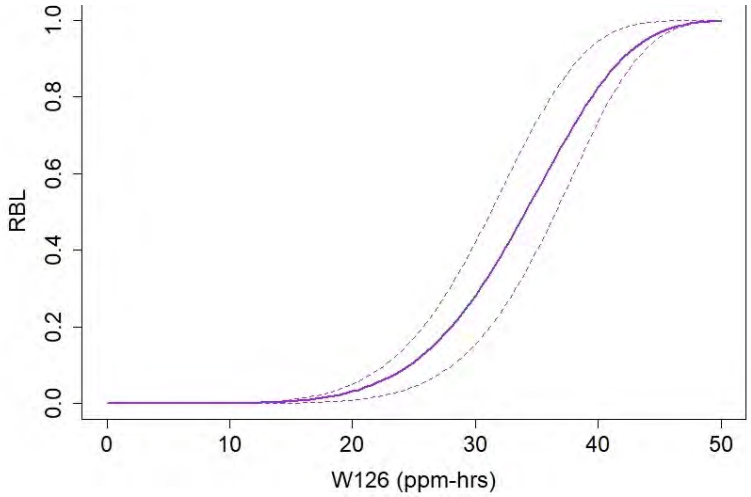

Figure 4A-3. RBL functions for Sugar Maple (*Acer saccharum*).

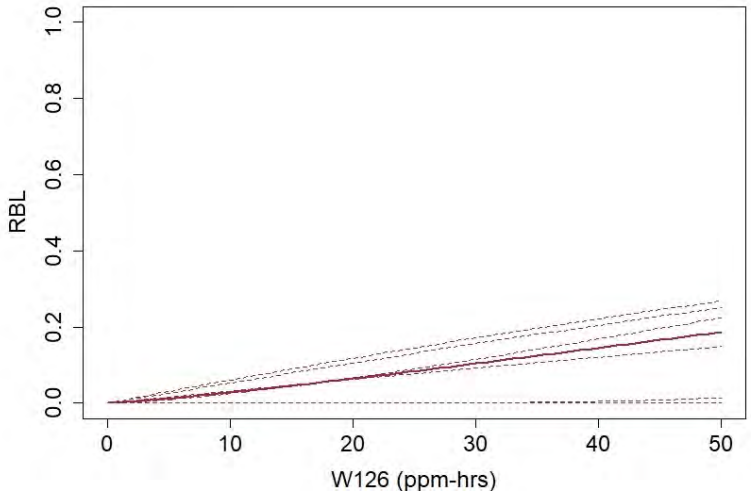

Figure 4A-4. RBL functions for Red Alder (*Alnus rubra*).

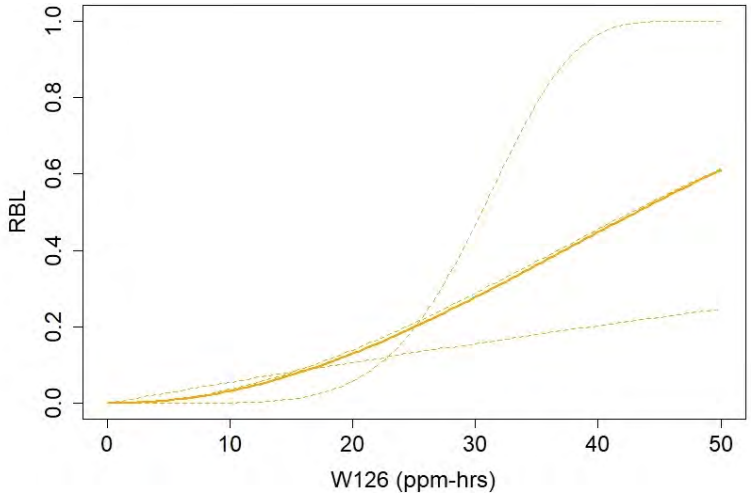

Figure 4A-5. RBL functions for Tulip Poplar (*Liriodendron tulipifera*).

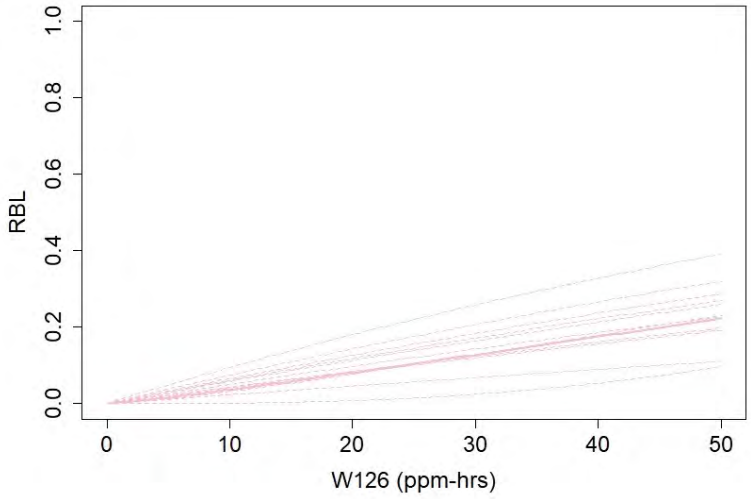

Figure 4A-6. RBL functions for Ponderosa Pine (*Pinus ponderosa*).

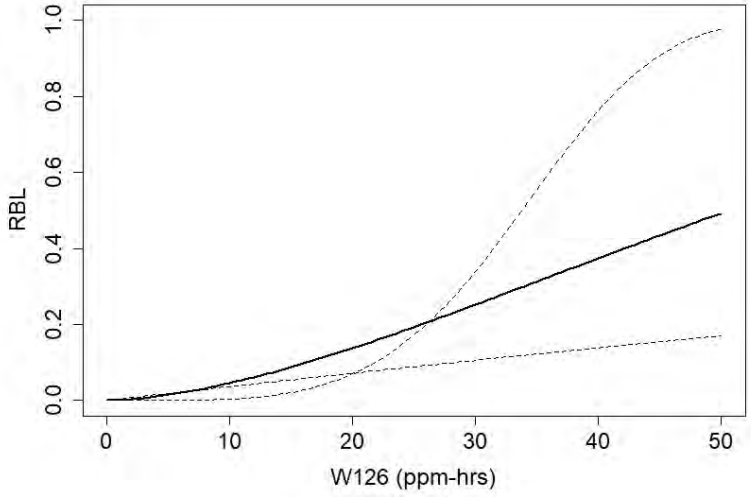

Figure 4A-7. RBL functions for White Pine (*Pinus strobus*).

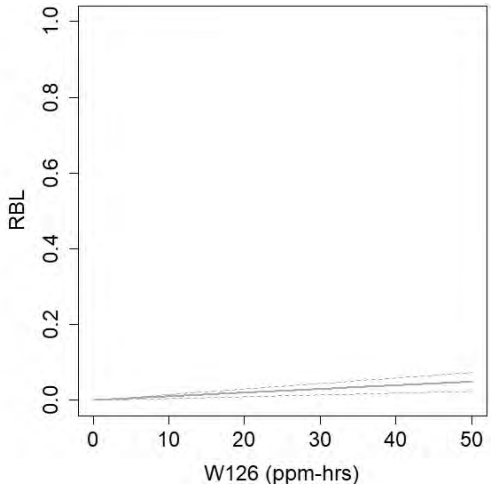

Figure 4A-8. RBL functions for Loblolly Pine (*Pinus taeda*).

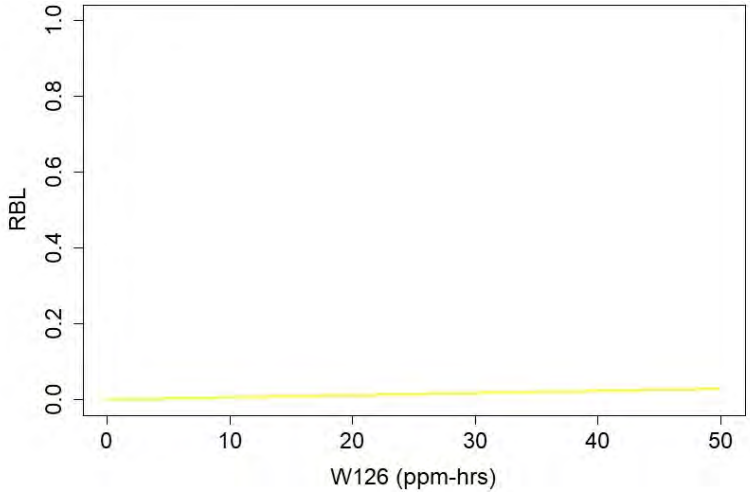

Figure 4A-9. RBL functions for Virginia Pine (*Pinus virginiana*).

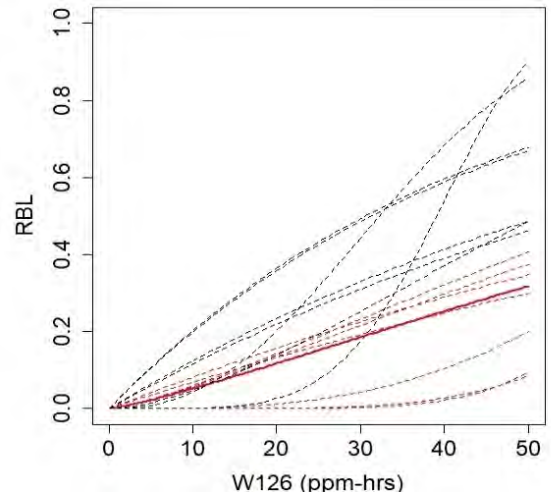

Figure 4A-10. RBL functions for Aspen (*Populus tremuloides*). Red lines = wild, black=clone.

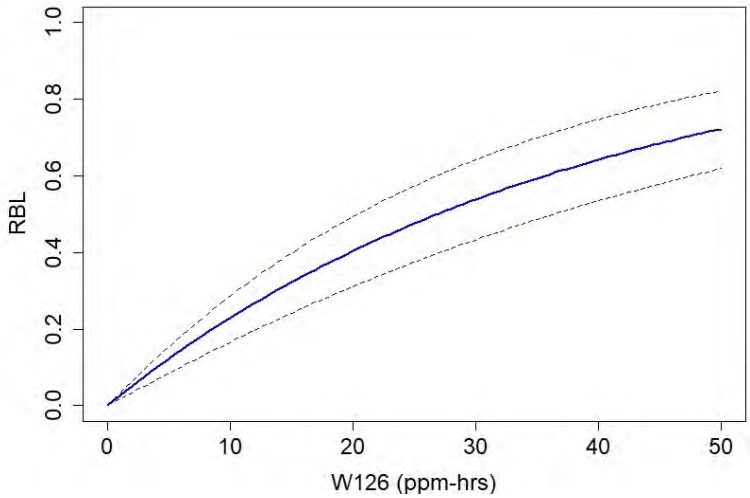

Figure 4A-11. RBL functions for Black Cherry (*Prunus serotina*).

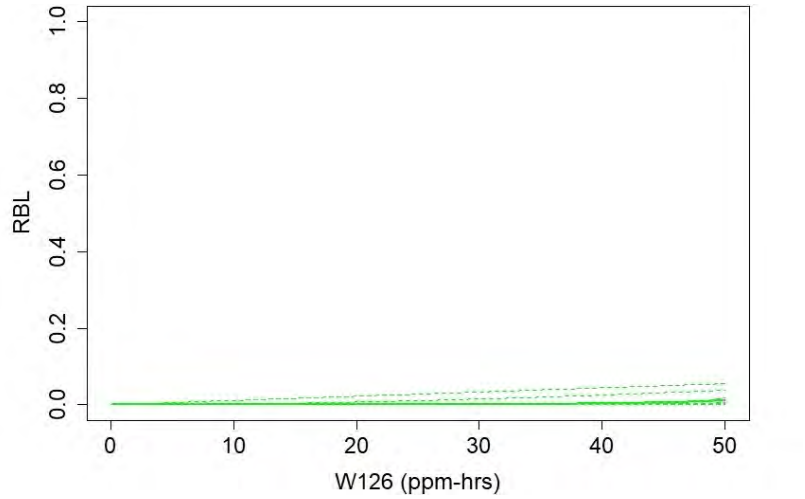

Figure 4A-12. RBL functions for Douglas Fir (*Pseudotsuga menzeiesii*).

In the 2015 review, consideration of the E-R functions for the seedlings of 11 tree species focused on the median estimate across the 11 species-specific functions. Recognizing the extent to which experimental variation contributes to uncertainty in the species-specific E-R functions, a stochastic analysis was performed in the quantitative exposure/risk assessment for the 2015 review as an approach to investigating the impact of uncertainty and variability in the E-R function dataset; an update of this analysis is presented in Figure 4A-13. This figure illustrates different approaches to estimating a median E-R function from the functions from the individual experiments. In this figure, each grey curve is the median across 11 species-specific functions where the species-specific functions are represented by a random draw from the experiment-specific functions available for each species.[5] The red points are the median across the random draws at that W126 value and the whiskers extend to the 75th and 25th percentiles of those draws. For reference, the green line is the median across the 11 species-specific functions, and the red 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 ang 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 to26 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

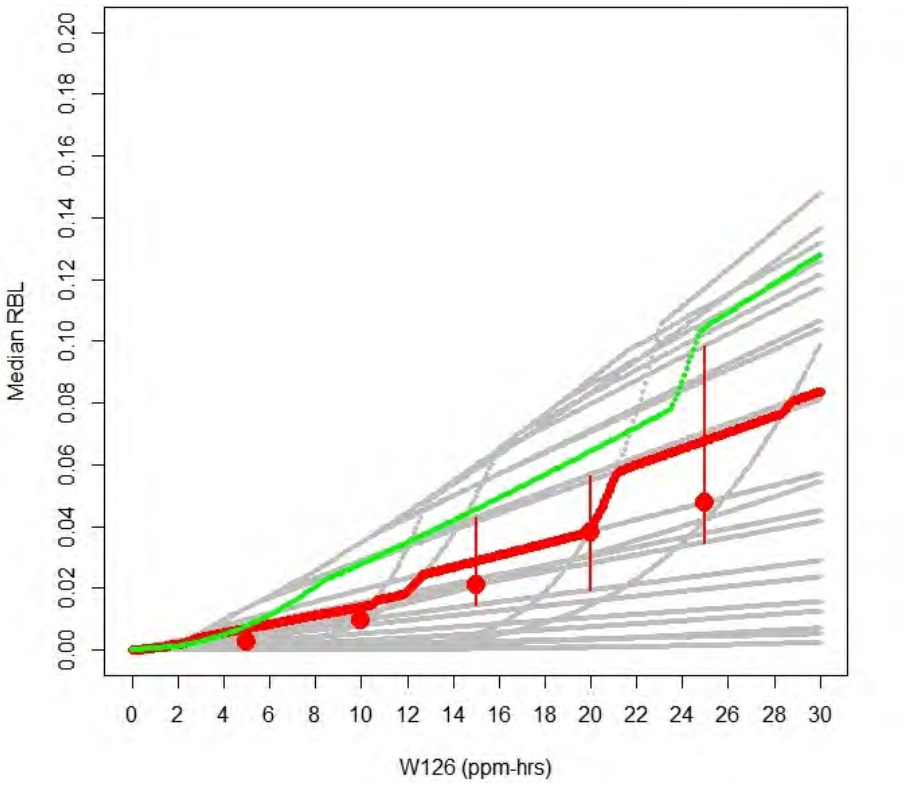

Figure 4A-13. Stochastic analyses of median E-R function across 11 species.

4A.1.2 Crop Species E-R Functions

The RYL functions for the 10 crop species are presented in Table 4A-2, and Figure 4A-14 presents the functions graphically.

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.

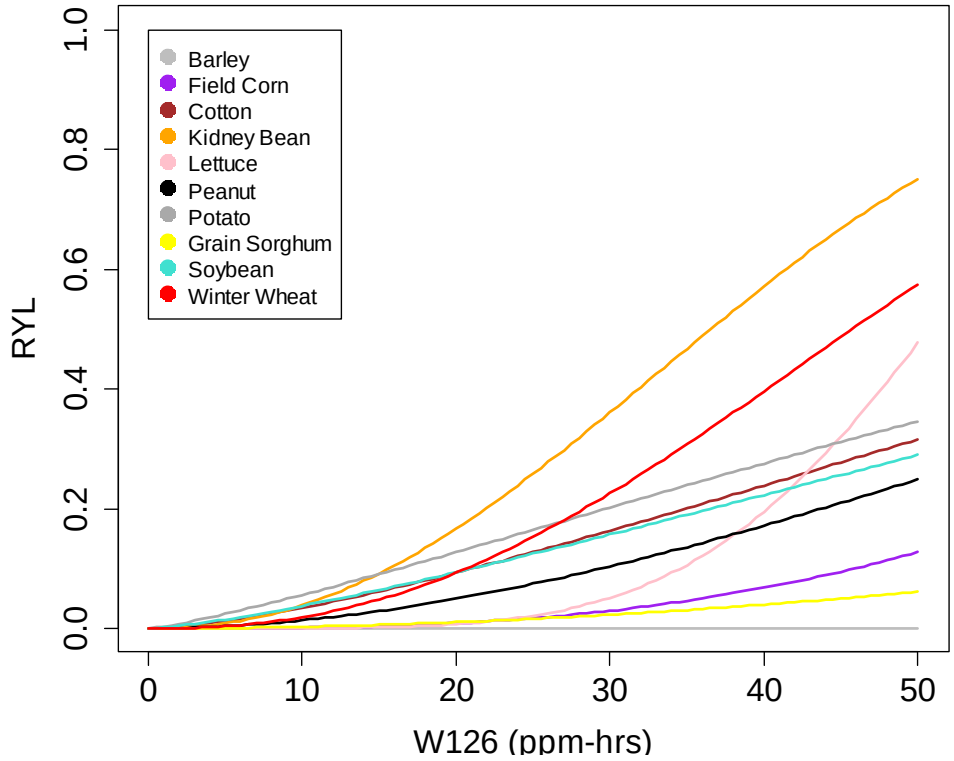

Figure 4A-14. RYL functions for crop species.

4A.1.3 Summary Tables for Tree Species and Crops

Table 4A-3 and Table 4A-4 below provide estimates of the relative loss for tree biomass and crop yield, respectively, at various W126 index values using the composite E-R functions for each species for each integer W126 index value between 7 ppm-hrs and 30 ppm-hrs. The cross-species median of the species-specific composite functions is calculated for all 11 tree species. These tables also provide estimates of the number of species for trees and crops respectively that would be below various reference values (e.g., 2% RBL for trees) at various W126 index values. Table 4A-5 summarizes the median values for each integer W126 index value between 7 ppm-hrs and 23 ppm-hrs.

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

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

Table 4A-5. Tree seedling RBL and CYL estimated for seasonal W126 O_3 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]	≤ 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: 6/10 species >5, <10% loss: 4/10 species
14 ppm-hrs	Median species w. 4.2% 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: 6/10 species >5,<10% loss: 4/10 species
13 ppm-hrs	Median species w. 3.8% loss [B]	≤ 2% loss: 5/11 species <5% loss: 7/11 species <10% loss: 10/11 species >20% loss: 1/11 species	Median species w.≤5% loss [D]	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species
12 ppm-hrs	Median species w. 3.5% loss [B]	≤ 2% loss: 5/11 species ≤ 5% loss: 8/11 species ≤10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss [D]	≤ 5% loss: 8/10 species >5,<10% loss: 2/10 species
11 ppm-hrs	Median species w. 3.1% loss [B]	≤ 2% loss: 5/11 species ≤5% loss: 8/11 species ≤10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss [D]	≤ 5% loss: 9/10 species >5, <10% loss: 1/10 species
10 ppm-hrs	Median species w. 2.8% loss [B]	≤ 2% loss: 5/11 species ≤ 5% loss: 9/11 species <10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss [D]	≤ 5% loss: 9/10 species >5,<10% loss: 1/10 species
9 ppm-hrs	Median species w. 2.4% loss [B]	≤ 2% loss: 5/11 species ≤ 5% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss [D]	≤ 5% loss: all species
8 ppm-hrs	Median species w. 2.0% loss [B]	≤ 2% loss: 5/11 species ≤ 5% loss: 10/11 species >15% loss: 1/11 species	Median species w. ≤5% loss [D]	≤ 5% loss: all species
7 ppm-hrs	Median species w. ≤2% loss [B]	≤ 2% loss: 7/11 species ≤5% loss: 10/11 species >15% loss: 1/11 species	Median species w. ≤5% loss [D]	≤ 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.

4A.2 TREE SEEDLING RBL STUDIES

Table 4A-6 below lists the 51 tree seedling exposure cases for which E-R functions were derived. Eleven tree species are represented by the 51 cases. The exposures in terms of SUM06 for the cases are presented as available.[7] As described in section 4A.1 above, species-specific (composite) functions were derived for each species, and Table 4A-5 above presents median RBL estimates from the 11 species-specific functions.

[7] Based on the approach that the EPA used in the 2007 Staff Paper, a SUM06 index value of 25 ppm-hrs is estimated to correspond to a W126 index of approximately 21 ppm-hrs (U.S. EPA, 2007, Appendix 7B, p. 7B-2).

Table 4A-6. Individual tree seedling experimental cases for which E-R functions were derived in Lee and Hogsett (1996).

Study ID[A]	Species	Site	year	Duration (days)[B]	Harvest [C]	SUM06 (ppm-hr)[D]	Study/Source and notes
Per 1996 AQCD, Table 5-28; Lee and Hogsett, 1996, Table 12						*Per Hogsett et al 1997, Table 2*	
1	Aspen - wild	OR	1989	84	1		
1	Aspen - wild	OR	1989	84	2		
2	Aspen - wild	OR	1991	118	1		
2	Aspen - wild	OR	1991	118	2		
3	Aspen - wild	OR	1990	112	1	0.2, 16.1, 72.1, 102.8	Hogsett (unpublished) cited in Hogsett et al., 1997 Hogsett et al., 1995
3	Aspen - wild	OR	1990	112	2		
4	Aspen - 216	MI	1990	82	1		May be described in Karnosky et al., 1996, who reported statistically significant total biomass loss for clones 259 (at the 2 highest exposure treatments) and clone 271 (at the highest treatment).
4	Aspen 253	MI	1990	82	1		
4	Aspen 259	MI	1990	82	1		
4	Aspen - 271	MI	1990	82	1		
5	Aspen – 216	MI	1991	98	1		Karnosky et al., 1996, who reported statistically significant total biomass loss at the highest exposure concentrations.
5	Aspen – 259	MI	1991	98	1	0.0, 11.5, 24.5, 32.4, 40.3, 60.5	Karnosky et al (1995, in press) cited in Hogsett et al., 1995 Hogsett et al., 1997 corresponds to Karnosky et al., 1996, who reported statistically significant total biomass loss averaged across all clones at the highest exposure treatment.
5	Aspen – 271	MI	1991	98	1		
6	Aspen-wild	MI	1991	98	1		
7	Douglas Fir	OR	1989-90	113	1		
7	Douglas Fir	OR	1989-90	113	2		
7	Douglas Fir	OR	1989-90	234	3	0.1, 33.4, 147.2, 207.2, 261.5	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997
7	Douglas Fir	OR	1989-90	234	4		
8	Douglas Fir	OR	1991-92	118	1		
8	Douglas Fir	OR	1991-92	118	2		
8	Douglas Fir	OR	1991-92	230	3	0.1, 30.4, 60.6, 143.0, 202.9	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997
9	Ponderosa Pine	OR	1989	111	1		
9	Ponderosa Pine	OR	1989	111	2		
10	Ponderosa Pine	OR	1989-90	113	1		May be described in Andersen et al., 1997 Seedlings exposed to O_3 for two growing seasons were statistically significant smaller than CF-exposed seedlings when measured in the dormant condition (SUM00 greater than 253). Total biomass was reduced 58% at the highest exposure.
10	Ponderosa Pine	OR	1989-90	113	2		
10	Ponderosa Pine	OR	1989-90	234	3		
10	Ponderosa Pine	OR	1989-90	234	4		
11	Ponderosa Pine	OR	1991-92	118	1		Lee and Hogsett, 1999, who statistically significant biomass loss at the 2 highest exposures (12-hr W126 greater than 59)
11	Ponderosa Pine	OR	1991-92	118	2		

Study ID[A]	Species	Site	year	Duration (days)[B]	Harvest [C]	SUM06 (ppm-hr)[D]	Study/Source and notes
Per 1996 AQCD, Table 5-28; Lee and Hogsett, 1996, Table 12						*Per Hogsett et al 1997, Table 2*	
11	Ponderosa Pine	OR	1991-92	230	3	0.1, 30.4, 60.6, 143.0, 202.9	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997
12	Ponderosa Pine	OR	1992	140	1		May be described in Andersen and Scagel, 1997 Statistically significant reduction in the biomass of all plant components after two seasons O_3 exposure (1992+1993); reductions were greater with nutrient restriction+O_3
13	Ponderosa Pine	OR	1991	84	1		
14	Red Alder	OR	1990	121	1		
15	Red Alder	OR	1989	113	1		
15	Red Alder	OR	1989	113	2		
16	Red Alder	OR	1991	118	1		
16	Red Alder	OR	1991	118	2	0.0, 16.0, 31.8, 73.4, 103.6	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997
17	Red Alder	OR	1992	112	1	0.1, 14.5, 29.1, 70.1, 99.9	Hogsett (unpublished) cited in Hogsett et al., 1995 Hogsett et al., 1997
18	Black Cherry	SMNP[F]	1989	76	1	0.0, 1.9, 17.1, 40.6	Neufeld et al., 1995 cited in Hogsett et al., 1995 Hogsett et al., 1997 Also described in Neufeld and Renfro, 1993. Statistically significant reduction in highest treatment group
19	Black Cherry	SMNP	1992	140	1	0.0, 0.0, 0.8, 18.1, 50.2	Neufeld, personal comm cited in Hogsett et al., 1995 Hogsett et al., 1997 Described in Neufeld et al., 1995 Neufeld and Renfro, 1993 Statistically significant reduction in highest treatment
20	Red Maple	SMNP	1988	55	1	9.2, 12, 47, 125.4	Neufeld (pers comm) cited in Hogsett et al., 1995 Hogsett et al., 1997
21	Tulip Poplar	SMNP	1990-91	75	1	0.1, 2.1, 0.2, 0.9, 16.6, 38.8	SUM06 exposures provided by Henry Lee email (8/16/2019)
21	Tulip Poplar	SMNP	1990-91	184	3	0.1, 0.5, 1.4, 34.5, 88.7	Neufeld (pers comm) cited in Hogsett et al., 1995 Hogsett et al., 1997
22	Tulip Poplar	SMNP	1992	81	1		
23	Loblolly GAKR 15-23	AL	1988-89	555	3	4.9, 58.5, 301.5, 507.0	Qiu et al., 1992and Lefohn et al., 1992 cited by Hogsett et al., 1995 Hogsett et al., 1997, who reported GARK 15-23 was more tolerant to O_3 with no significant biomass losses over the entire exp period, while the more sensitive GARK 15-23 clone had statistically significant decreases in foliar biomass and root square area at the highest exposure treatment.
23	Loblolly GAKR 15-91	AL	1988-89	555	3		
24	Sugar Maple	MI	1990-91	83	1		

Study ID[A]	Species	Site	year	Duration (days)[B]	Harvest[C]	SUM06 (ppm-hr)[D]	Study/Source and notes
Per 1996 AQCD, Table 5-28; Lee and Hogsett, 1996, Table 12						*Per Hogsett et al 1997, Table 2*	
24	Sugar Maple	MI	1990-91	180	3	0.0, 25.2, 27.8, 49.8, 67.6, 94.4	Karnosky (pers. comm.) cited by Hogsett et al., 1995 Hogsett et al., 1997 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	1990-91	83	1		
25	E. White Pine	MI	1990-91	180	3	0.0, 25.2, 27.7, 49.8, 64.2, 94.4	Karnosky (pers. comm.) cited by Hogsett et al., 1995 Hogsett et al., 1997 May be described in Isebrands et al., 2000 pg 170 which reported no statistically significant difference in height, stem, root or current year needle biomass in response to O_3
26	Virginia Pine	SMNP	1992	98	1	0.0, 0.0, 1.9, 21.7, 51.6	Neufeld (pers. comm.) cited in Hogsett et al., 1995 Hogsett et al., 1997 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)

A Study ID as in Lee and Hogsett (1996), Table 12.
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 of exposure for Harvest 3.
C 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.
D First SUM06 treatment value corresponds to charcoal-filtered exposure (Hogsett et al., 1997 Table 2).
E Based on an approach used in the 2007 Staff Paper (and the associated temporal patterns of O_3 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 (U.S. EPA, 2007, Appendix 7B, p. 7B-2).
F SMNP = Smoky Mountains National Park.

4A.3 ANALYSIS OF MULTIPLE-YEAR RBL

The sections below consider two illustrative analyses of the influence of seasonal O_3 exposures across multiple years.

4A.3.1 Comparison of Predicted and Observed O_3 Growth Impacts

The 2013 and 2020 ISAs present comparisons of aspen stand growth observations from an Aspen FACE multiyear O_3 exposure study and predictions derived through the application of a median composite E-R function for wild aspen and aspen clones[8] to seasonal W126 index values (2013 ISA, section 9.6.3.2; 2020 ISA, Appendix 8, Figure 8-17). The Aspen FACE study monitored growth of aspen stands annually from 1997 through 2003 (King et al., 2005).[9] Growth was monitored for stands grown in ambient air and under elevated O_3 conditions. The elevated O_3 treatment involved increasing hourly concentrations by approximately 1.5 times over the O_3 concentrations occurring in ambient air at the site (King et al., 2005). Hourly O_3 measurements were obtained from the authors (for both the "ambient" and "elevated" treatments) and used to calculate seasonal W126 index for use in the comparisons presented in the 2013 and 2020 ISAs (cumulative seasonal average in 2013 ISA and single-year average in 2020 ISA). The values for "observed" above ground total biomass for the aspen stands were derived from measurements obtained from the authors and allometric equations (2013 ISA, section 9.6.3.2; King et al., 2005 and associated Corrigendum).[10]

The 2013 ISA presents a comparison of observed biomass to predicted biomass based on application of the E-R function to W126 in terms of cumulative (multiyear) seasonal average.[11] Based on the results of this analysis (which are presented in the Tables 9-14 and 9-15, and Figure 9-20 of the 2013 ISA), the 2013 ISA concludes "the agreement between predictions … and observations was very close" and "the function based on one year of growth was shown to be applicable to subsequent years." (p 9-135). The 2020 ISA also presents such a comparison, but this one represents the W126 index in terms of single-year seasonal index (2020 ISA, Appendix

[8] 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).

[9] 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 O- 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.

[10] The publication by King et al., (2005) reports on measurements for the years 1997 through 2003.

[11] 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.

8, Figure 8-17). These presentations illustrate the variability inherent in the magnitude of growth impacts of O_3 and in the quantitative relationship of O_3 exposure and RBL (2013 ISA, Figure 9-20; 2020 ISA, Appendix 8, Figure 8-17).

4A.3.2 Comparison of Estimated Impacts of Constant and Annually Varying Seasonal Exposure

The presentation here considers potential differences in aspen growth of O_3 exposures expressed as a constant annual W126 index value compared to exposures expressed as the same W126 index value in terms of a 3-year average such that the annual values vary while meeting the 3-year average limit (Figure 4A-15), with the variation reflecting what is shown to be common at U.S. monitoring locations (e.g., Appendix 4D, section 4D.3.1.2). This analysis is not intensive or elaborate; rather, it is intended to provide an illustration of concepts associated with application of the E-R functions described in section 4A.1 using data from a study with aspen of the effects of a six-year exposure on accumulating biomass (King et al., 2005), which is also utilized in a different type of analysis in the 2013 ISA, that is summarized in the ISA and also in section 4A.3.1 above (2020 ISA, Appendix 8, Figure 8-17; 2013 ISA, section 9.6.3.2).

Description of Analysis: The analysis presented here is intended to simply illustrate the application of the tree seedling E-R function for aspen over a multi-year period using two types of air quality scenarios: (1) one in which the O_3 concentrations are limited such that each year's W126 is no higher than 17 ppm-hrs, and (2) a second in which the O_3 concentrations are allowed to vary each year as long as the 3-year average is no higher than 17 ppm-hrs. More specifically, the two scenarios are (1) repeated years of W126=17 ppm-hrs and (2) repeated 3-year cycles of the same varying W126 (e.g., 10, 17 and 24 ppm-hrs). This analysis was intended to inform consideration of potential magnitude of an over or under estimation of growth reduction when the target W126 value was calculated from a 3-year average or for each individual year. In this analysis, above-ground tree biomass is estimated for each year through a six-year period.[12]

The example for this analysis uses aspen, beginning with a seedling, and utilizes data on growth rates (annual biomass increases) for the control treatment[13] in a study by King et al.,

[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] Yearly growth– specifically above-ground biomass production- for the baseline situation is based on data presented in Table 3 of King et al (2005) for the treatment labeled "control." This treatment reflects ambient air concentrations of O_3 at the Aspen FACE location (King et al., 2005). These "control" conditions included single-year seasonal W126 index values estimated in the 2020 ISA to range from approximately 3 to 9 ppm-hrs across the 6-year period (2020 ISA, Appendix 8, Figure 8-17), and in the 2013 ISA to be approximately 3 ppm-hrs as the cumulative multiyear seasonal average across the 6-year period (2013 ISA, Tables 9-14 to 9-15; King et al., 2005).

2005). The O_3 growth effect is estimated by applying the established E-R function for aspen. In our analysis, above ground biomass loss[14] was calculated using the estimated growth rate (yearly biomass production) and the relative biomass loss (RBL) for the pertinent W126 value based on the aspen E-R function (Table 4A-8). This biomass loss was calculated for the 3-year average W126 of 17 ppm and for each of the three individual year values of 10, 17 and 24 ppm (Table 4A-7). King et al., 2005) provided initial biomass for the control and yearly biomass production for each of the subsequent six years of study. In this analysis, the growth rate information derived from King were applied over six years of growth (using the yearly growth rates from the study, g/m^2/year for the stand). The above ground biomass of the aspen stand in each year was compared across the two exposure scenarios (Figure 4A-15; Table 14-7). The difference between the two scenarios in total above ground biomass for the stand varied from year to year. After the first year, this difference in the year's total above ground biomass (not to be confused with annual growth in biomass, to which RBL is applied) was always less than 2%.

Summary of Analysis Limitations, Assumptions and Uncertainties: Given the limited availability of controlled tree exposure data for individual years/seasons in a multi-year exposure, as well as the simply conceptual or illustrative nature of the analysis, there are assumption, limitations and uncertainties inherent in the analysis.

- This analysis assumes that baseline growth rate, to which the O_3 effect in terms of RBL is applied, is unaffected by the O_3 exposure in the prior year. Although the potential for "carryover" of an effect of particularly high exposure years into subsequent years (e.g., through a reduction in carbohydrate storage) has been discussed in assessments for past reviews (e.g., 2006 AQCD, section AX9.2.8), the specific exposure levels that might trigger significant "carryover" effects are not established. Studies cited in those discussions include exposure levels much higher than conditions associated with meeting the current standard (e.g., 80 ppb O_3 over a full growing season, 2006 AQCD, section AX9.2.8).
- Additionally, while the availability of multi-year experimental data that can be examined with regard to this issue for the range of exposures investigated here is limited, a multi-year study available in the last review (King et al., 2005) did not appear to indicate such impacts (2013 ISA section 9.6.3.2). The multi-year experimental dataset from King et al (2005) was assessed in the 2013 ISA and is also discussed in the ISA for this review with regard to growth effects and correspondence of E-R function predictions with study observations (2020 ISA, Appendix 8, section 8.13.2 and Figure 8-17; 2013 ISA, section 9.6.3.2, Table 9-15, Figure 9-20). The analysis in the 2013 ISA, which focused on the six years for which the aspen study reported data, compared observed reductions in growth for each year of a 6-year period to those predicted by applying the established E-R function for Aspen to cumulative multi-year average W126 index values (2013 ISA,

[14] Above-ground growth (foliage and wood) is used consistent with 2013 and 2020 ISA analyses described in section 4A.3.1 above).

section 9.6.3.2).[15] 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), indicating that the approach employed in the analysis presented here -for the initial six years- may be reasonable for the circumstances examined here. In the ISA for this review, 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 just slightly less tight fit in the later year, 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 generally reliably predict the yearly findings from the six years of exposures of the Aspen FACE experiment.

- Variables other than O_3 that can affect growth in a given year (e.g., precipitation, temperature, community competition) are not represented in the current analysis other than the extent to which they affect the baseline growth rate provided by the "control" from the aspen study by King et al., 2005).
- Additionally, this analysis is based on aspen, and the specific pattern of differences between the two scenarios might be expected to vary for species with different biomass growth rates (and E-R functions). However, datasets of tree growth across multiple-year periods such as that available for aspen in the study by King et al., 2005) are not prevalent.

[15] 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.

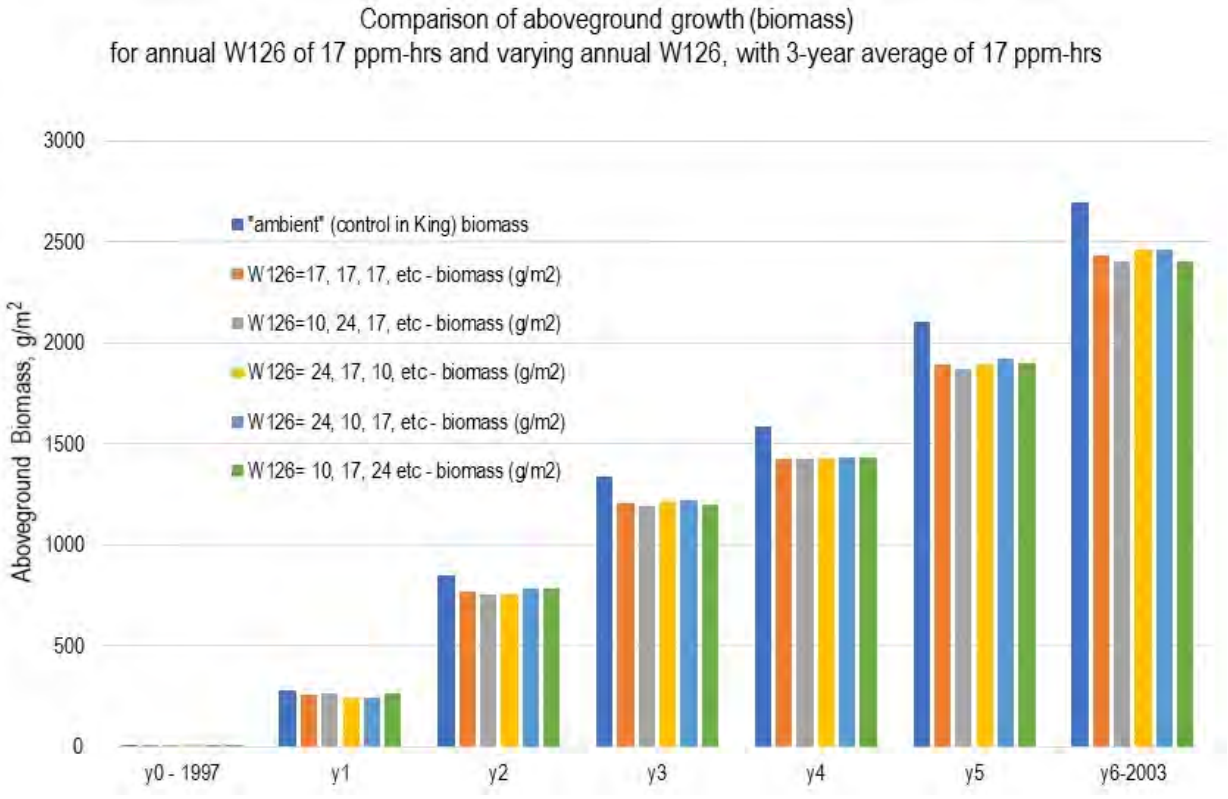

Figure 4A-15. Estimated aboveground biomass of aspen with different patterns of annual seasonal W126 index.

Table 4A-7. Comparison of total aspen above ground biomass estimated for different patterns of varying annual exposures and constant exposure equal to 3-year average (17 ppm-hrs).

	"ambient" (control in King) 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, 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	% difference in total tree biomass of W126= 10, 17, 24 vs 17
y0 - 1997	9.1		9.1	9.1	9.1	9.1	9.1				
y1	280.2	2979.1%	253.6	266.1	240.9	240.9	266.1	4.9%	-5.0%	-5.0%	4.9%
y2	849.5	203.2%	767.1	752.9	754.4	780.6	779.6	-1.9%	-1.7%	1.8%	1.6%
y3	1335.5	57.2%	1205.5	1191.2	1215.1	1219.0	1195.1	-1.2%	0.8%	1.1%	-0.9%
y4	1581.1	18.4%	1427.0	1424.1	1425.1	1428.9	1428.0	-0.2%	-0.1%	0.1%	0.1%
y5	2099.4	32.8%	1894.6	1867.2	1892.6	1920.3	1895.5	-1.4%	-0.1%	1.4%	0.0%
y6-2003	2695.2	28.4%	2432.0	2404.6	2457.4	2457.7	2404.9	-1.1%	1.0%	1.1%	-1.1%

Table 4A-8. Aboveground growth calculations for subset of scenarios.

	"ambient" (control in King) annual WOOD growth that year	"ambient" (control in King) annual FOLIAGE growth that year	"ambient" (control in King) annual growth that year (uses yr-yr delta for foliage)	"ambient" (control in King) aboveground biomass	Growth - % increase	O_3 W126 (ppm-hrs)	O_3-impact (RBL)	W126=17 biomass (g/ m^2)	O3 W126 (ppm-hrs) - low->hi->ave	O_3-impact (RBL)	W126=10, 24, 17, etc - biomass (g/m^2)	% diff from constant 3-yr ave
y0 (1997)	7.6	1.5		9.1				9.1			9.1	
y1	226.1	46.5	271.1	280.2	2979.12%	17.0	0.098	253.6	10.0	0.052	266.1	
y2	476.6	139.2	569.3	849.5	203.18%	17.0	0.098	767.1	24.0	0.145	752.9	
y3	390.5	234.7	486	1335.5	57.21%	17.0	0.098	1205.5	17.0	0.098	1191.2	-1.2%
y4	209	271.3	245.6	1581.1	18.39%	17.0	0.098	1427.0	10.0	0.052	1424.1	-0.2%
y5	434.5	355.1	518.3	2099.4	32.78%	17.0	0.098	1894.6	24.0	0.145	1867.2	-1.4%
y6 (2003)	500.1	450.8	595.8	2695.2	28.38%	17.0	0.098	2432.0	17.0	0.098	2404.6	-1.1%

Attachment 1 to Appendix A

Derivation of Composite Median Equations (parameterized models) in Lee and Hogsett (1996)

The following describes the methodology used to produce the sets of parameters in Tables 2, 12, and 13 of Lee and Hogsett (1996), which have been used in some form in AQCDs and ISAs since 1996. "Regression", "parameter estimation", "model estimation" and "model fitting" all refer to the same statistical procedure of using nonlinear ordinary least square regression to obtain values for model parameters from a dataset.

1) Tables 12, 13, and 2 in Lee and Hogsett (1996) primarily summarize parameter values estimated through regression from 51 controlled exposure studies of tree seedlings conducted by NHEERL/WED. In those studies, 11 species of trees were exposed to a set of ozone concentrations for durations varying from 55 to 234 days or up to 555 days (in the case of one species).

2) The model fitted to the data from each of the 51 individual studies (in Table 12) is a three-parameter Weibull model with the following parameterization: *Predicted Biomass = A exp(-[exposure/B]^c).*[16] When removing the intercept *A*, this model gives biomass relative to no exposure, and the resulting two-parameter equations all have the same 0-1 range of relative biomass response and can thus be compared or aggregated across studies with different ranges of absolute biomass. *Predicted Relative Biomass = exp(-[exposure/B]^c),* and *Predicted Relative Biomass Loss= 1- exp(-[exposure/B]^c).* When estimating each set of three parameters for each separate study, the ozone exposure was quantified using the 12-hour daytime W126 index, summed over the duration of each study.

   a. Table 12 gives parameter values for 51 models, one per study, that reflect W126 over each study duration. This table also presents the W126 index estimated for a 92-day duration for RBLs of 10% and 20%.

   b. Table 13 presents parameter values for the 51 models given in Table 12, as well as parameter values for composite models for the 11 tree species included in those 51 studies, two sets of values per species (one for the median and the second for the 75th percentile). This table also presents W126 index estimates for RBLs of 10%, 20% and 30%. These three estimates for the composite models are estimates for a 92-day duration.

   c. Table 2 presents values for composite models for all experiments for all species aggregated.

3) The median composite models, one per species (table 13) are derived as follows. For each of the studies for a given species, the predicted relative biomass loss is first generated at six values of exposure: 10, 20, 30, 40, 50, and 60 ppm-hr, using the study-specific two-parameter equation. This is done in a way to obtain six values of exposure for 12-hour daytime exposures summed over 92 days. [17] All but the median of the relative biomass loss estimates

[17] 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

at each value of exposure are then discarded, and the two-parameter model for relative biomass loss is fitted to the remaining six median points. For example, Ponderosa Pine was the subject of 11 studies; 11 sets of parameters were estimated through regression (see item 2 above); 66 values of predicted relative biomass loss were then computed, 11 at each of the six exposure values. All but the median of those 11 relative biomass loss estimates were discarded at each of the six levels of exposure, and the two-parameter model fitted to the six remaining points, giving the Ponderosa Pine median composite equation for a 92-day exposure.

4) The all-species, median composite models (table 2) were estimated using the same aggregation method, but applied to all 51 studies at once. The 51 equations in Table 12 were used to compute 306 values of relative biomass loss, six values each for the 51 sets of *B* and *C* parameters, with those six values of exposure generated in a way to obtain 12-hour daytime exposures summed over 92 days. The two-parameter model was then fitted to the 75th percentile and the median as in item 3 above. Table 2 also includes the results of the same method using other exposure indices besides the 12-hour W126 index.

5) For every equation in the tables, values of exposure at which some given percent loss is expected relative to no exposure, or any other exposure, can be back-calculated using: $Exposure = B*(-ln(1-predicted\ relative\ biomass\ loss))^{1/C}$. Some of those expected values of exposure are presented for various loss percentages in tables 2, 12, and 13 for the all-species median composite model, the 51 studies, and the 11 species-level median composite, respectively. In the case of single-study calculations in Table 12, the value of exposure for a given loss percentage was first calculated based on the respective duration of each study, then simply prorated. For example, the duration of Study 1 Harvest 1 in table 12 was 84 days and the exposure at which 10% loss is expected over that duration is 13.71 ppm-hr. The prorated exposure value for a 10% loss over 92 days is calculated as 13.71*92/84 = 15.01 ppm-hr. Median models for species in Table 13 or for all species in Table 2 were parameterized with 92-day durations and the exposure values for the various loss percentages did not therefore require prorating.

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.

Attachment 2 to Appendix A

As reference, the following table presents calculations for a simple case of multi-year exposure, application of E-R function estimates, and tree biomass accumulating over that period.

Example calculations for simple case of multi-year exposure application of E-R function estimates.

CONCEPTUAL EXAMPLE (RBL =fraction reduction from control tree growth that year)

Growth rate (fraction of biomass at end of prior year)		control	O3 RBL	O3 tree		Growth rate (fraction of biomass at end of prior year)		control	O3 RBL	O3 tree	
0.3	Y0	1		1		0.3	Y0	1		1	
	Y1	1.3	0.138	1.259			Y1	1.3	0.118	1.265	
	Y2	1.69	0.118	1.603			Y2	1.69	0.118	1.609	
	Y3	2.197	0.098	2.060			Y3	2.197	0.118	2.056	
				0.115	<- RBL over full period					0.118	<- RBL over full period
0.2	Y0	1		1		0.2	Y0	1		1	
	Y1	1.2	0.138	1.172			Y1	1.2	0.118	1.176	
	Y2	1.44	0.118	1.384			Y2	1.44	0.118	1.388	
	Y3	1.728	0.098	1.644			Y3	1.728	0.118	1.642	
				0.116	<- RBL over full period					0.118	<- RBL over full period
0.5	Y0	1		1		0.5	Y0	1		1	
	Y1	1.5	0.138	1.431			Y1	1.5	0.118	1.441	
	Y2	2.25	0.118	2.093			Y2	2.25	0.118	2.103	
	Y3	3.375	0.098	3.107			Y3	3.375	0.118	3.095	
				0.113	<- RBL over full period					0.118	<- RBL over full period
0.1	Y0	1		1		0.1	Y0	1		1	
	Y1	1.1	0.138	1.086			Y1	1.1	0.118	1.088	
	Y2	1.21	0.118	1.183			Y2	1.21	0.118	1.185	
	Y3	1.331	0.098	1.292			Y3	1.331	0.118	1.292	
				0.117	<- RBL over full period					0.118	<- RBL over full period

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APPENDIX 4B

U.S. DISTRIBUTION OF 11 TREE SPECIES

4B.1. DESCRIPTION

This appendix presents maps of the distribution across the U.S. of 11 tree species for which there are established exposure-response (E-R) functions, as described in Appendix 4A. Historical ranges were based on Little (1971, 1976, 1977, and 1978) and basal area of each species was taken from Wilson et. al (2013) raster data to show present range and estimated density. Basal area is computed at the stand level as the sum of the basal area values for each individual tree (in sq. ft.), which is summed across all of the basal area per tree in the hectare. The map construction consists of tree species abundance, distribution, and basal area at a 250-meter (m) pixel size for the contiguous United States (Wilson 2013).

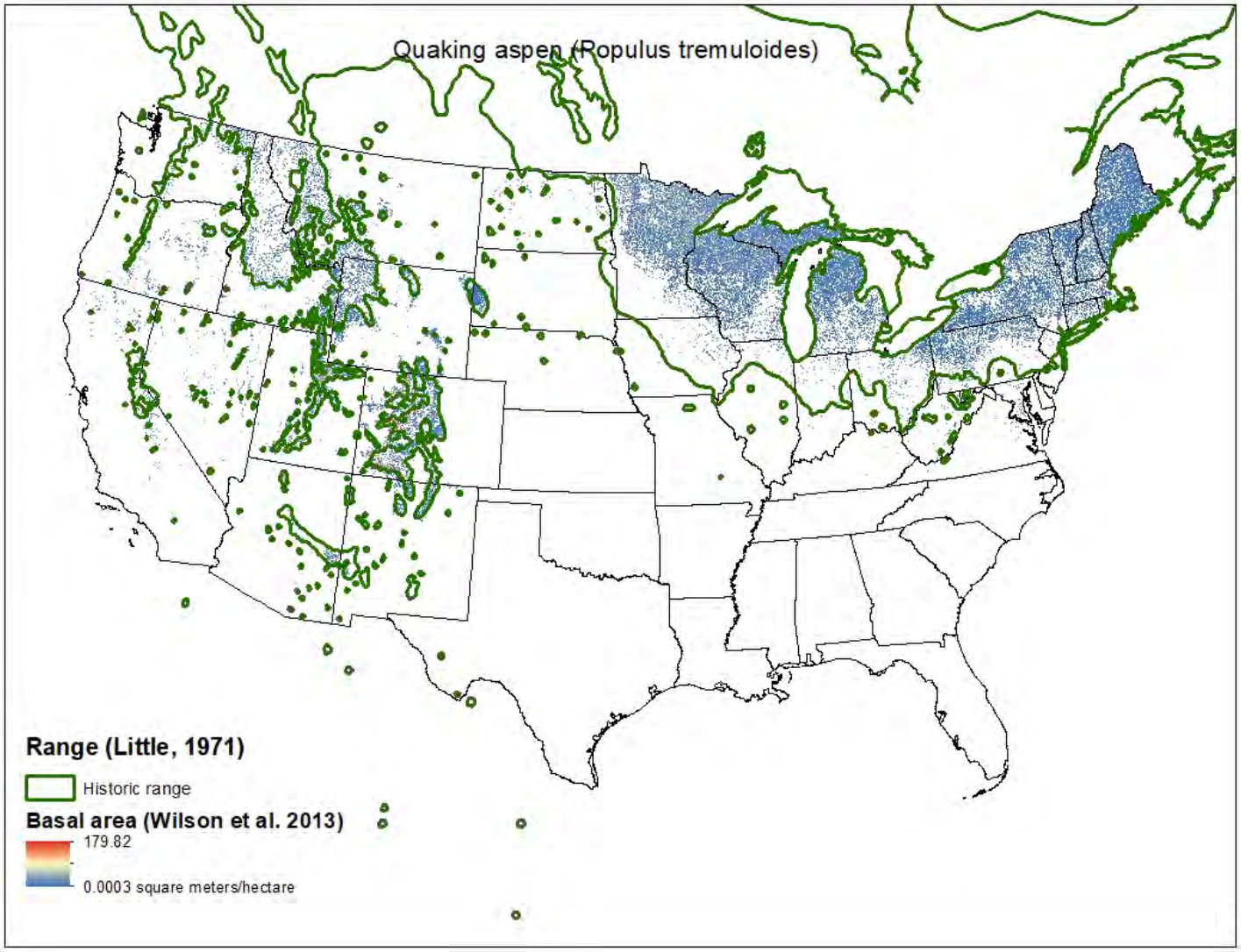

Table 4B-1. Distribution of quaking aspen (*Populus tremuloides*) in the continental U.S.

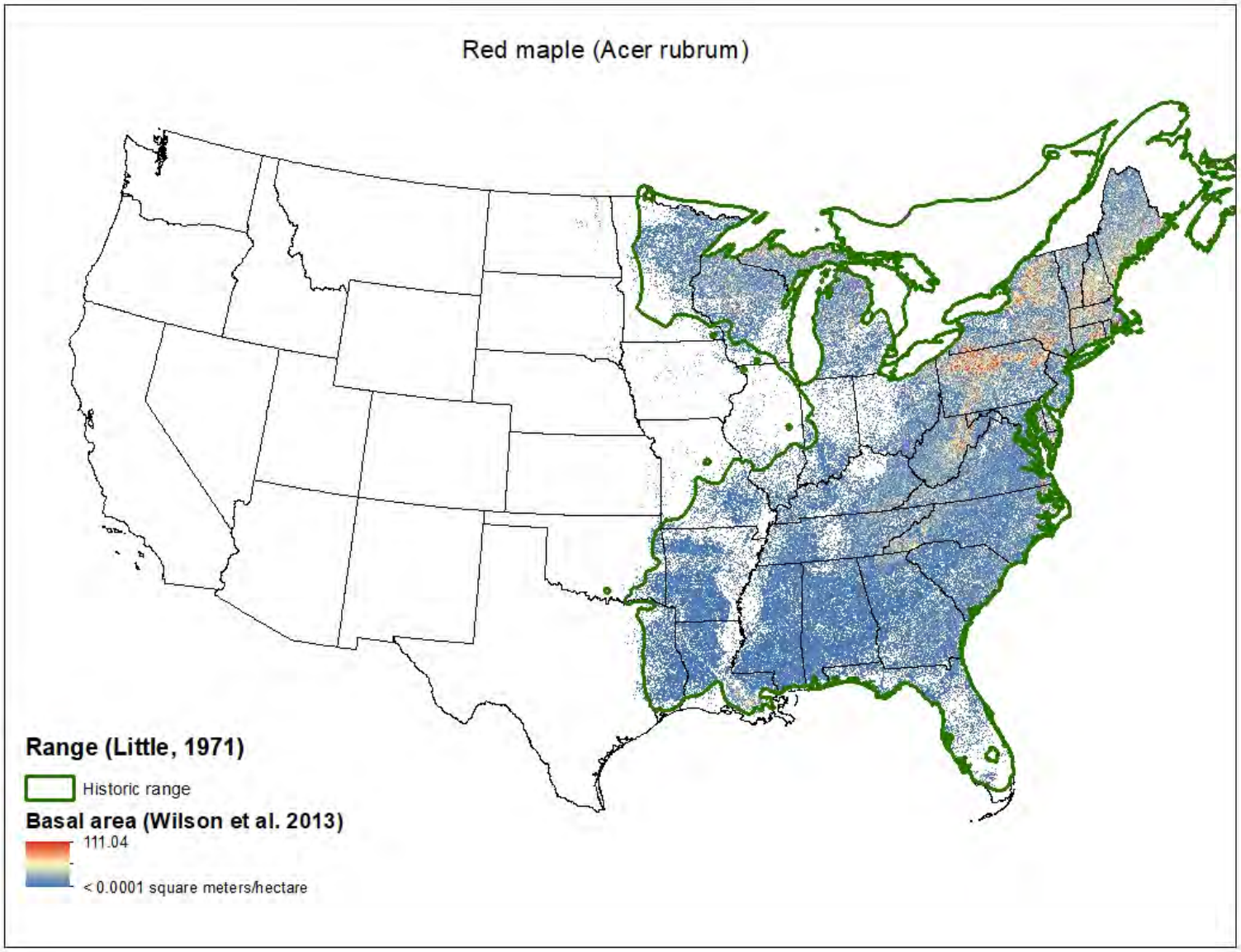

Table 4B-2. Distribution of red maple (*Acer rubrum*) in the continental U.S.

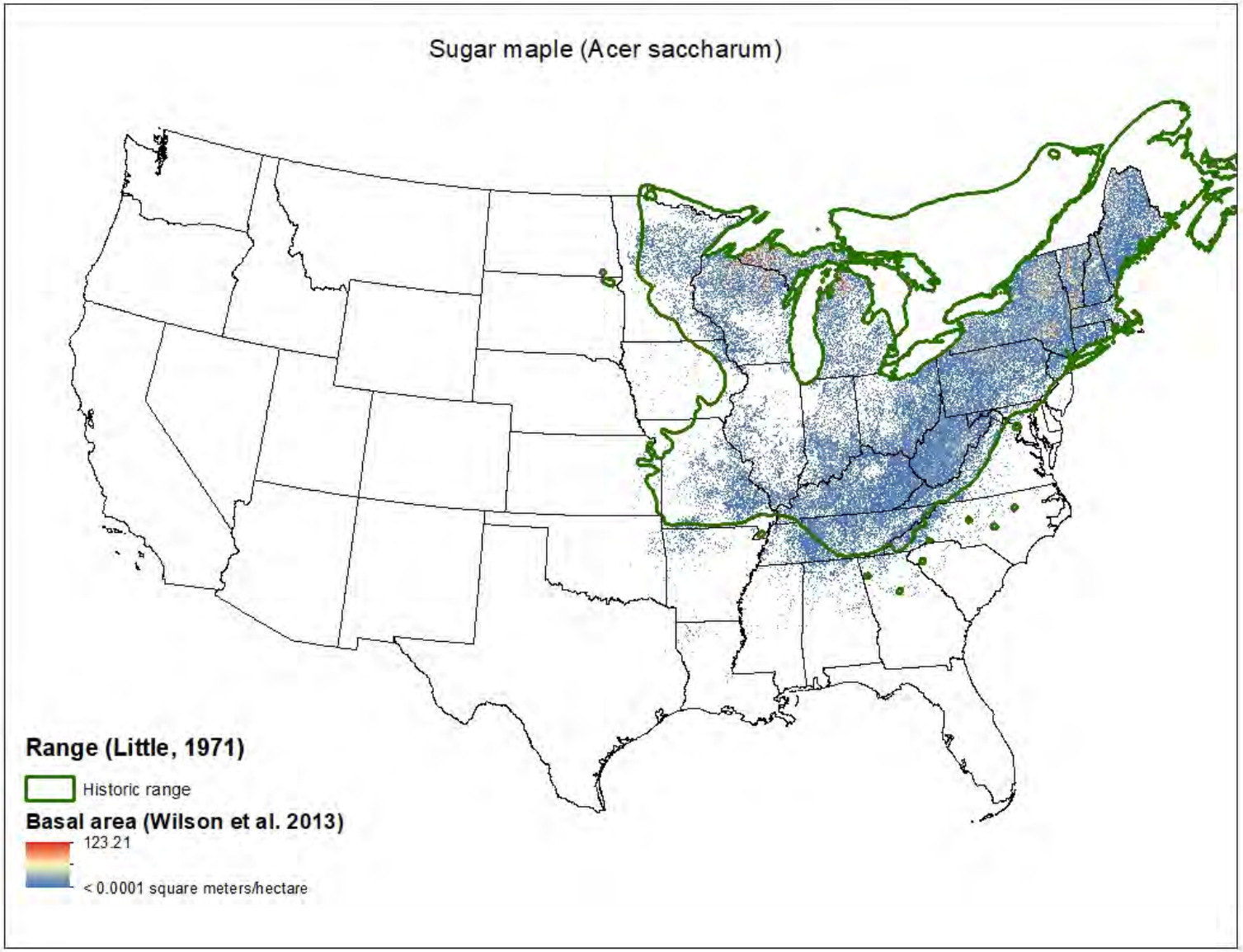

Table 4B-3. Distribution of sugar maple (*Acer saccharum*) in the continental U.S.

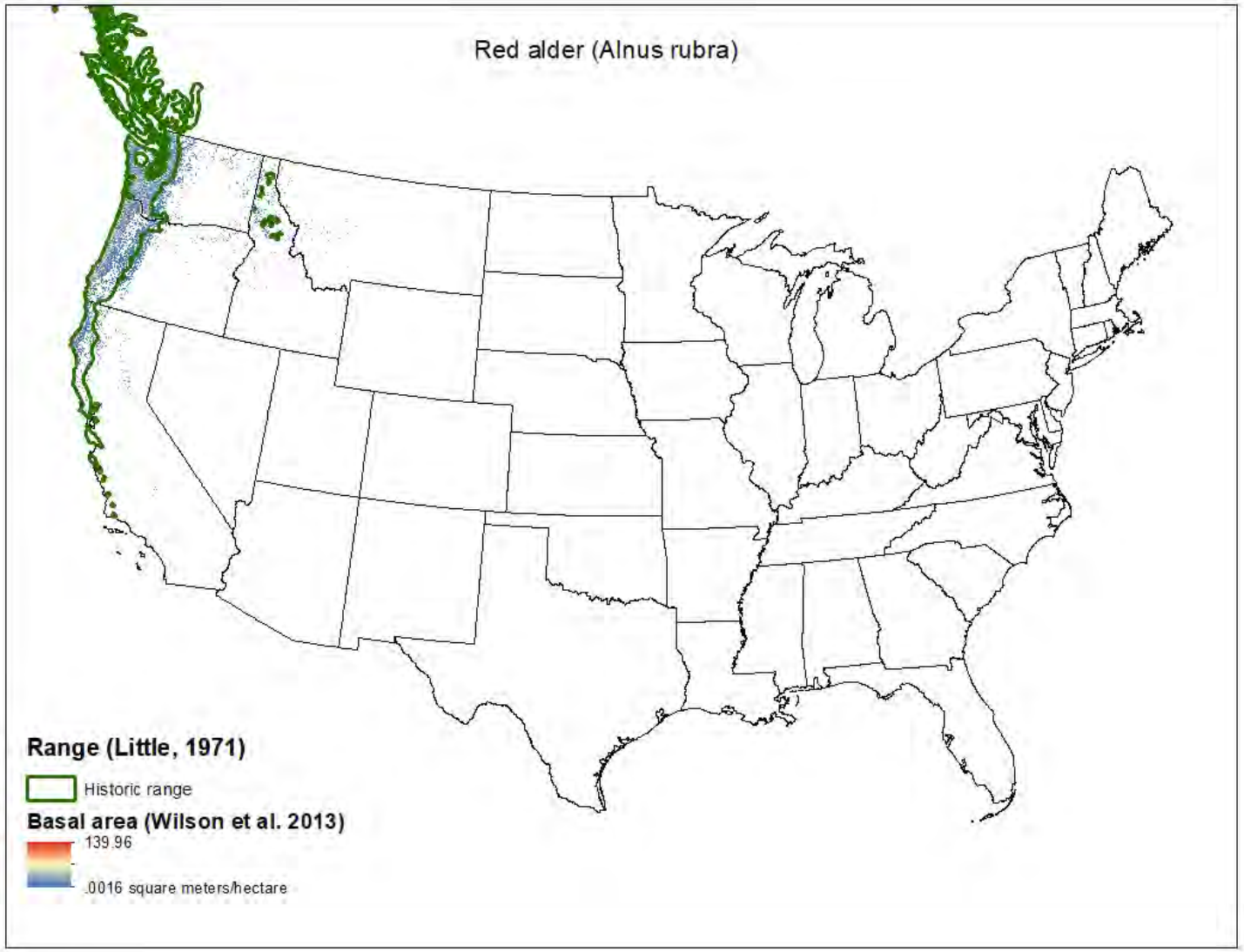

Table 4B-4. Distribution of red alder (*Alnus rubra*) in the continental U.S.

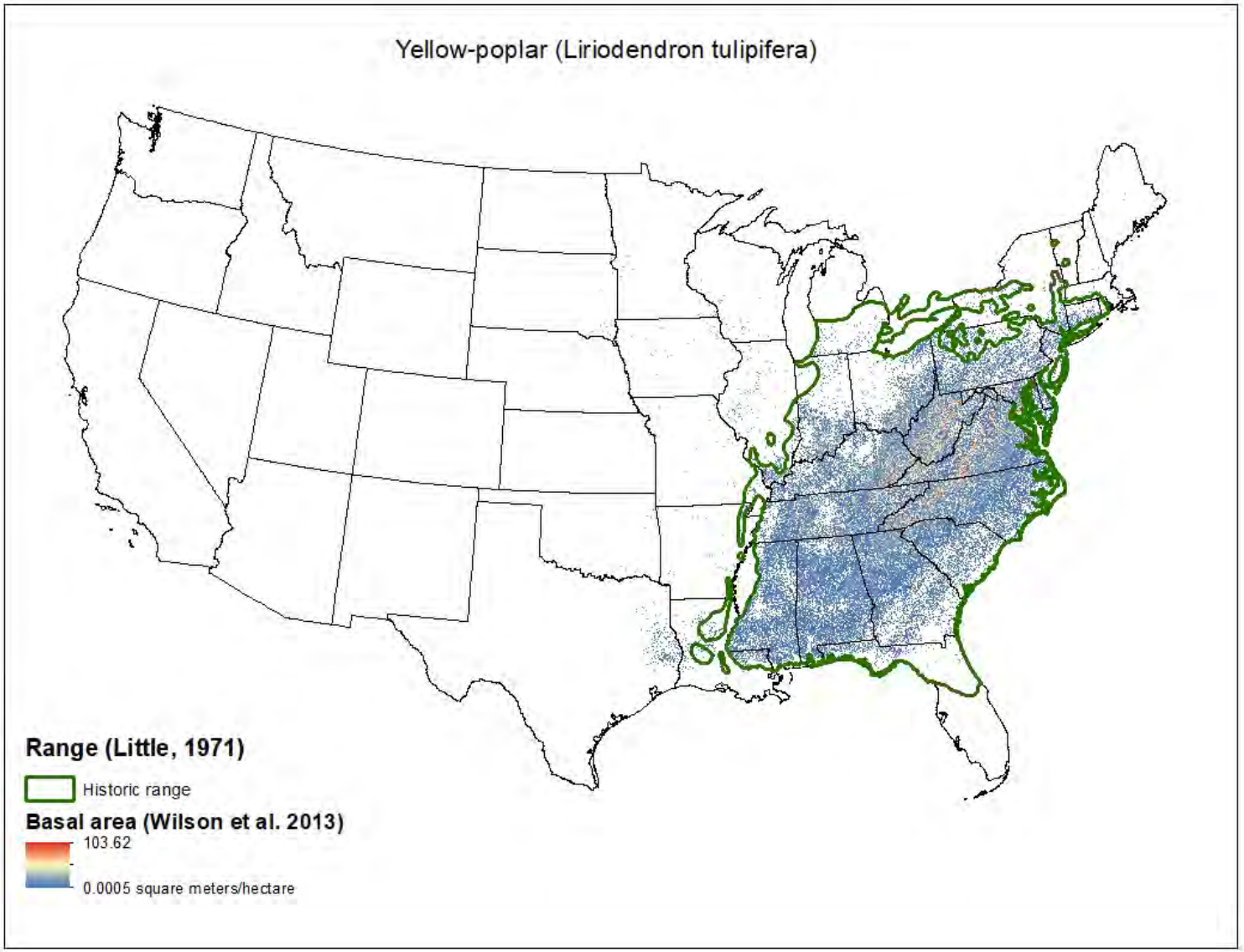

Table 4B-5. Distribution of tulip poplar (*Liriodendrun tulipifera*) in the continental U.S.

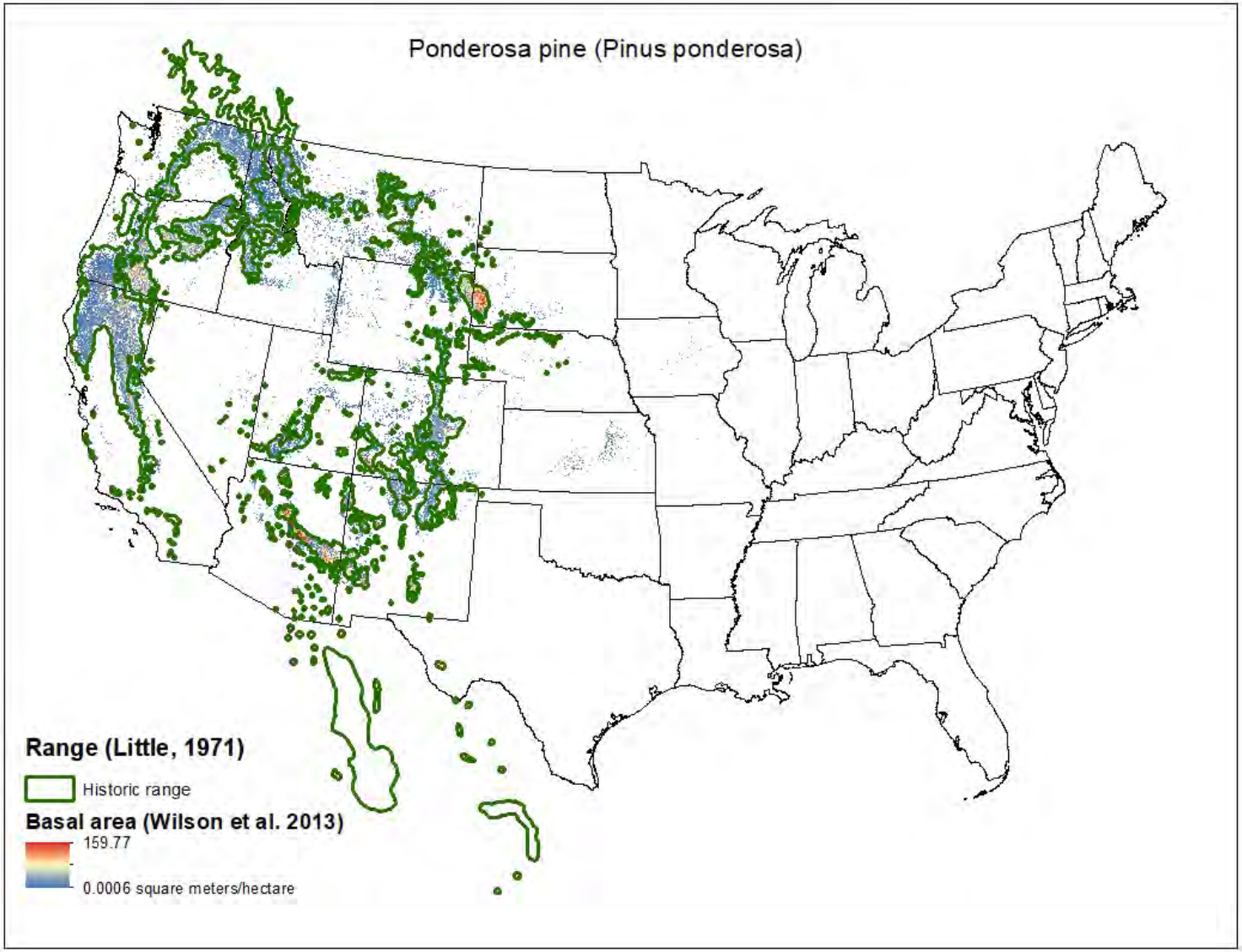

Table 4B-6. Distribution of ponderosa pine (*Pinus ponderosa*) in the continental U.S.

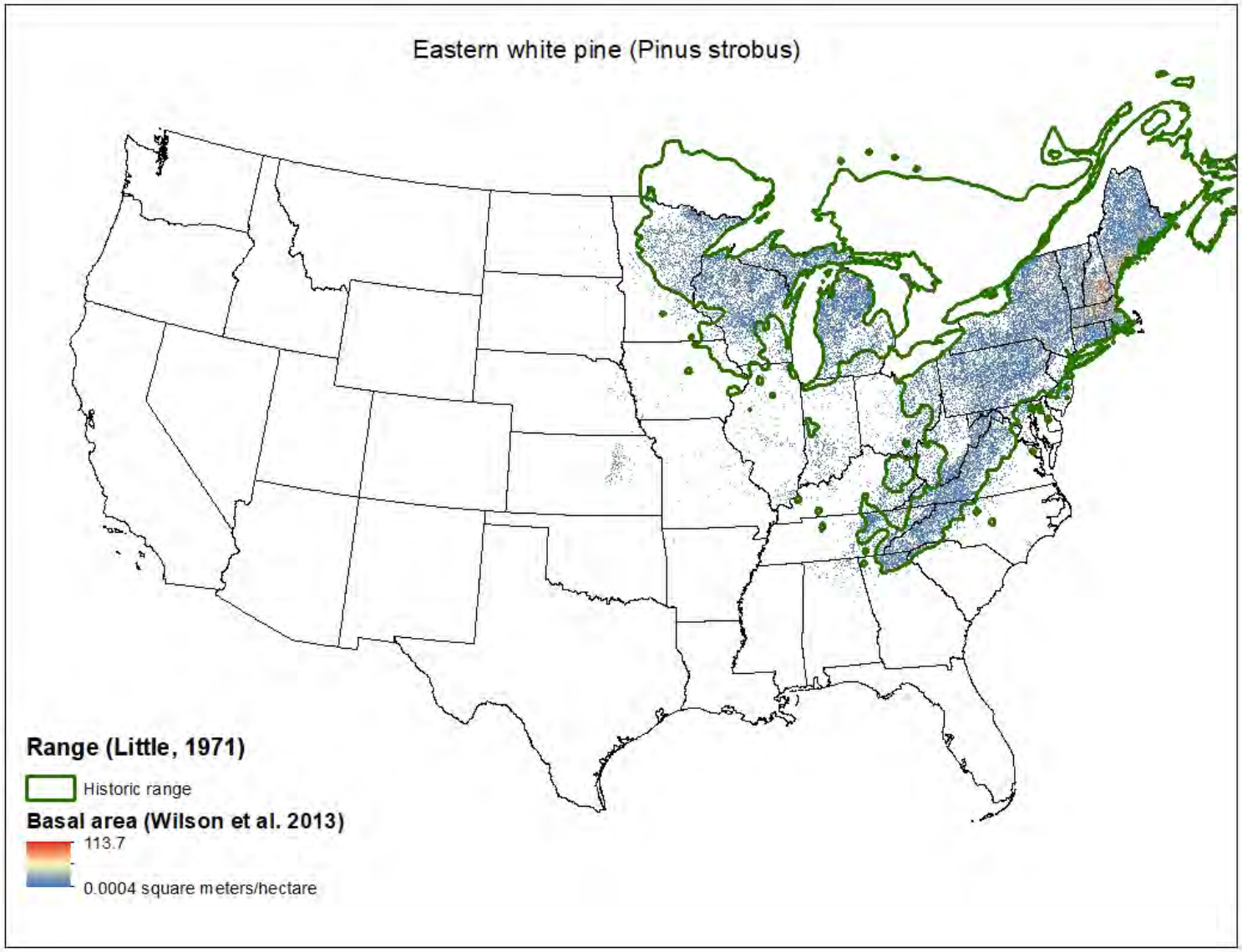

Table 4B-7. Distribution of eastern white pine (*Pinus strobus*) in the continental U.S.

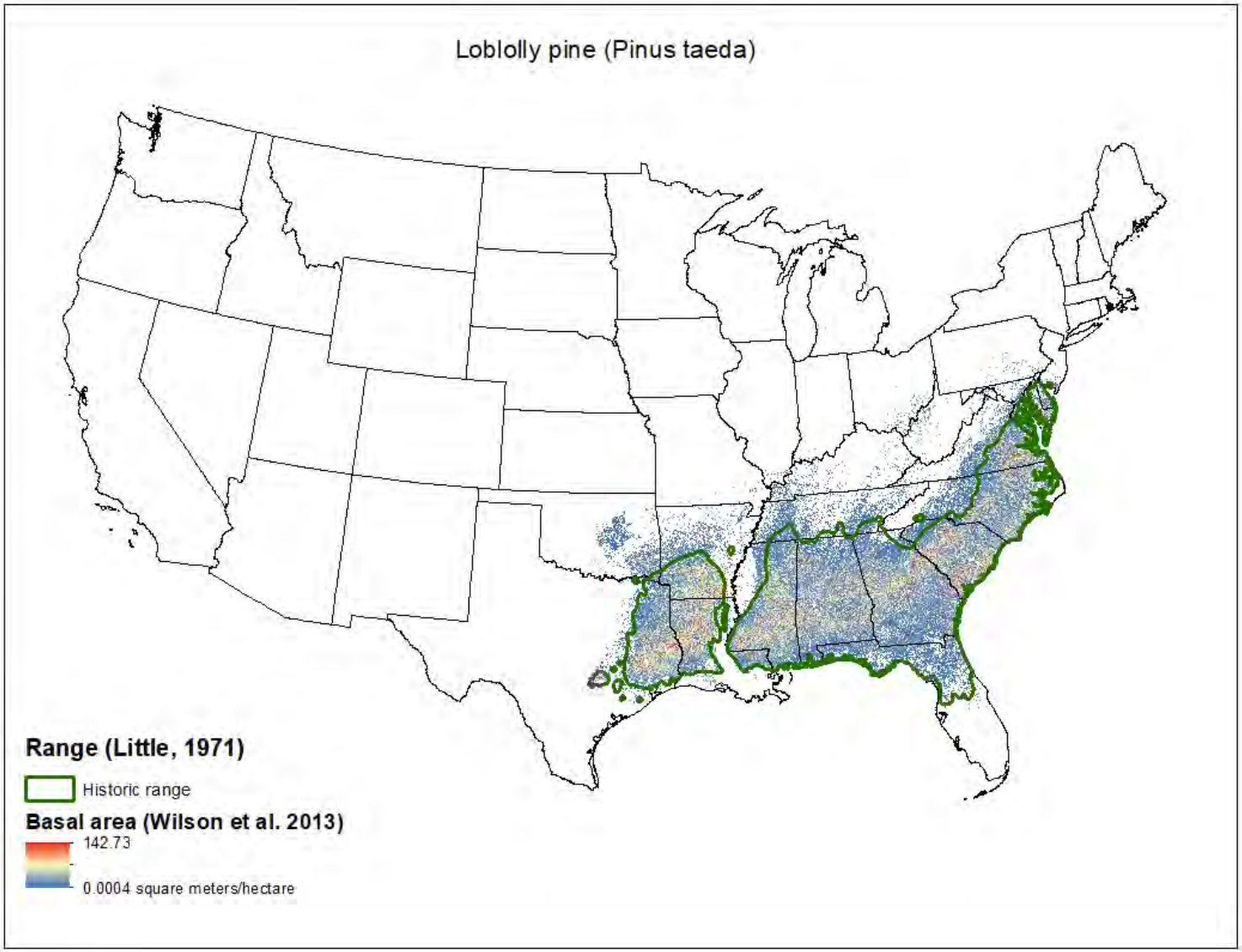

Table 4B-8. Distribution of loblolly pine (*Pinus taeda*) in the continental U.S.

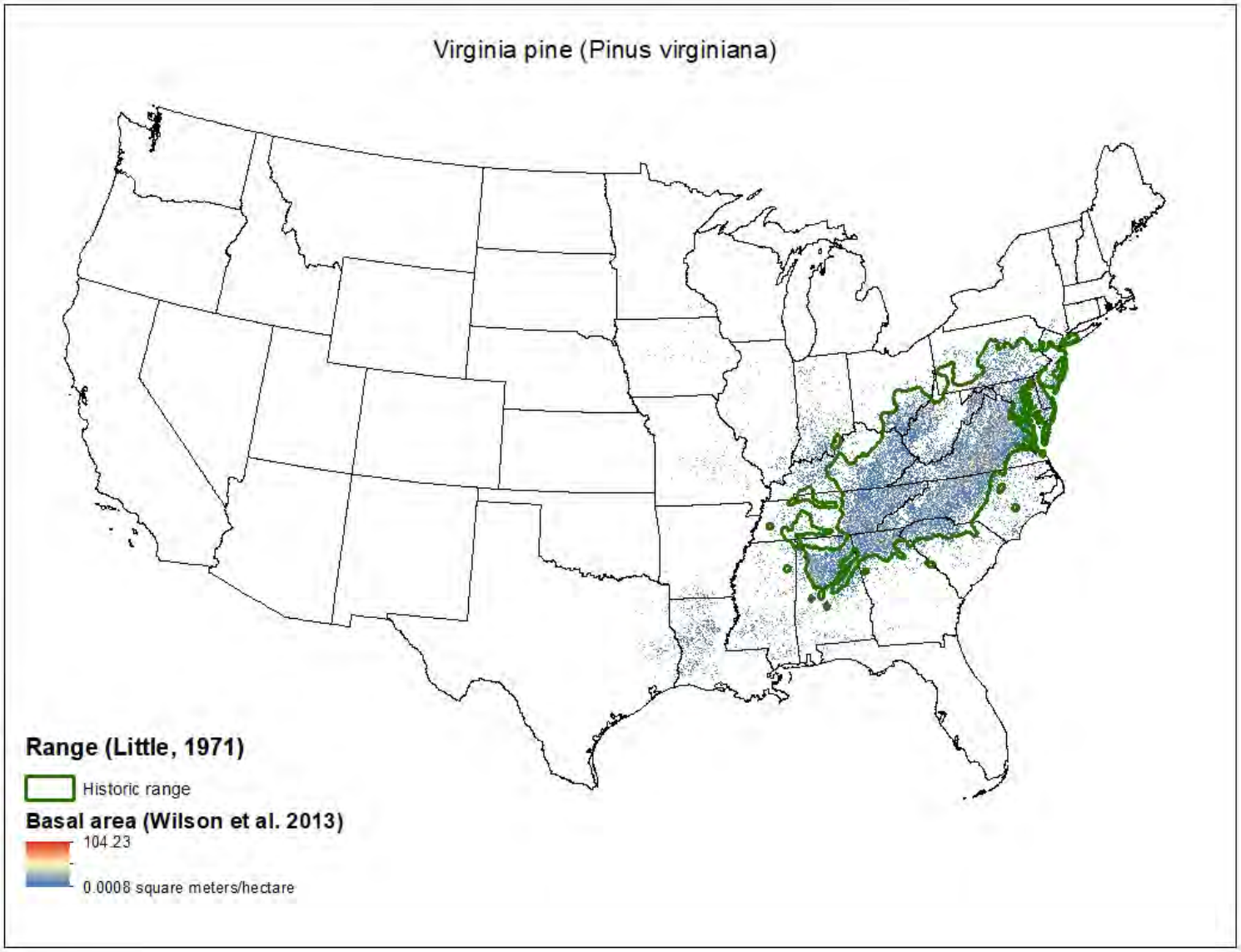

Table 4B-9. Distribution of Virginia pine (*Pinus virginiana*) in the continental U.S.

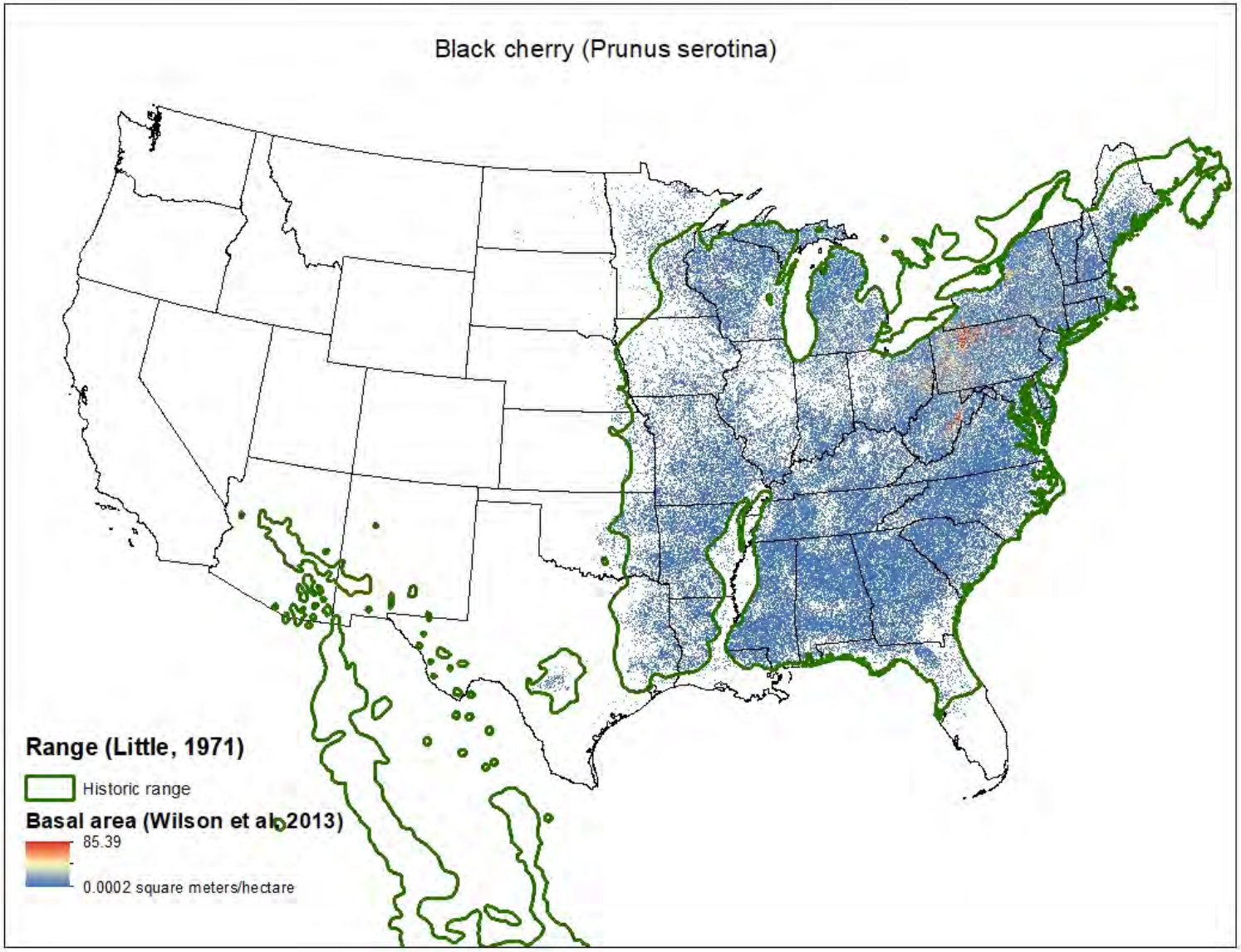

Table 4B-10. Distribution of black cherry (*Prunus serotina*) in the continental U.S.

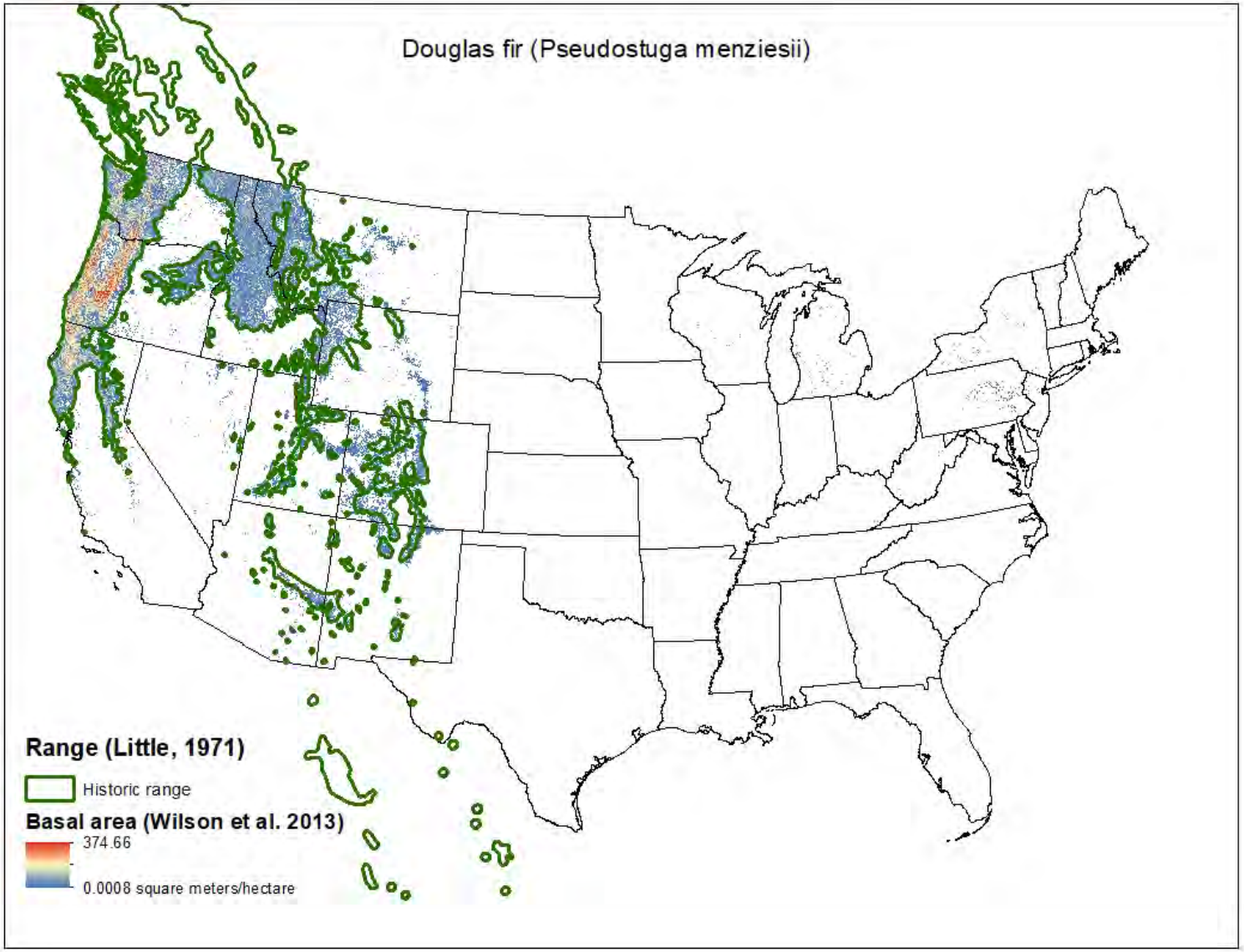

Table 4B-11. Distribution of Douglas fir (*Pseudotsuga menziesii*) in the continental U.S.

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APPENDIX 4C

VISIBLE FOLIAR INJURY SCORES AT U.S. FOREST SERVICE BIOSITES (2006-2010)

TABLE OF CONTENTS

4C.1 INTRODUCTION

It has long been recognized that elevated ozone (O_3) can cause visible foliar injury in some plants (ISA, Appendix 8, section 8.2). As discussed in the current and past ISAs as well as past Air Quality Criteria Documents, the severity and extent of visible foliar injury can vary with a variety of environmental variables (e.g., climatic variables as well as pollutant exposure) as well as variation in genetic factors within the same plant population (ISA, Appendix 8, section 8.2). Visible foliar injury "occurs only when sensitive plants are exposed to elevated O_3 concentrations in a predisposing environment," and "a major modifying factor is the amount of soil moisture available to a plant during the year when assessed" (U.S. EPA, 2013 [2013 ISA], p. 9-39).

In recognition of the long-standing evidence regarding O_3 and visible foliar injury in susceptible species, the U.S. Forest Service (USFS) and U.S. Park Service have used plant species with this susceptibility in their biomonitoring programs. A number of publications have focused on findings from biomonitoring surveys in the USFS-Forest Health Monitoring (FHM) and Forest Inventory and Analyses (FIA) programs. From the mid 1990s through 2010, this survey work included collecting information on the presence of visible foliar injury at the biomonitoring sites (biosites). Data on visible foliar injury incidence and severity data were collected each year at biosites in forested areas at states across the U.S. and summarized in terms of a biosite index (BI). The BI is a measure of the severity of O_3-induced visible foliar injury observed at each biosite.

Data from the multi-year USFS survey were used in analyses developed in the 2015 O_3 NAAQS review (80 FR 65292, October 26, 2015). These analyses utilized a dataset that had been developed by merging biosite data collected as part of the USFS FHM/FIA Network during the years 2006 through 2010, with NOAA soil moisture index values (as a surrogate for soil moisture measurements) and W126 estimates of seasonal O_3 exposure for those sites based on ambient air monitoring data for those 5 years (Smith and Murphy, 2015; U.S. EPA, 2014 [2014 WREA]) The resultant combined dataset included a BI score, soil moisture index value and a W126 index estimate each for 5,284 records at locations in 37 states for 1 or more of the years in the 5-year period from 2006-2010. This appendix brings forward key presentations developed from the combined dataset for the 2015 O_3 review and also includes additional presentations of key aspects of the dataset and the variables represented within it.

4C.2 DATASET PREPARATION

The combined dataset was developed from three datasets: (1) the national-scale FIA/FHM dataset of BI scores, (2) the NOAA's National Climatic Data Center national dataset of monthly drought indices and (3) national surfaces of estimated seasonal W126 index

developed by the EPA for the WREA in the last O_3 NAAQS review and further analyzed in a subsequent technical memo (Smith and Murphy, 2015). These individual datasets and how they were used to create the combined dataset, are described below.

Biosite Index: The USFS O_3 biomonitoring program has developed a national-scale data set focused on visible foliar injury and that includes BI scores at biosites in U.S. forests (Smith, 2012). The field methods, sampling procedures, and analytical techniques are consistent across biosites and years. The BI is calculated from species-specific scores based on a combination of the proportion of leaves affected on individual bioindicator plants and the severity of symptoms on injured foliage using an established scale (Horsfall and Cowling, 1978; Smith, 2012). Each site is sampled until 30 plants of at least two species have been evaluated (Smith et al., 2007). The site BI is the average score for each species averaged across all species on the biosite multiplied by 1,000 (Smith, 2012). The BI score ranges from zero to greater than 25, with a score of zero indicating no presence of foliar injury symptoms and scores increasingly greater than zero indicating increasingly greater severity of symptoms (Smith, 2012). Categories that have been used in publications include little or very light injury (BI greater than 0 up to 5), light injury (BI greater than 5 up to 15), moderate (BI greater than 15 up to 25) and heavy/severe (BI above 25) (Smith, 2012; Coulston et al., 2003).

The biosite data (BI scores) were obtained from the USFS for the years 2006 to 2010. While including most states in the contiguous U.S., the data obtained did not include records for most of the western states (Montana, Idaho, Wyoming, Nevada, Utah, Colorado, Arizona, New Mexico, Oklahoma, and portions of Texas) because biosite data were not available for those states during the 2006-2010 period (Smith et al., 2012).

Soil Moisture Index: The NOAA Palmer Z drought index is a monthly moisture anomaly index that is derived from measurements such as precipitation and temperature. This index represents the difference between monthly soil moisture and long-term average soil moisture (Palmer, 1965). The Palmer Z index is derived each month for each of 344 climate region divisions within the contiguous U.S. by the National Climatic Data Center (NCDC).[1] The index values typically range from -4 to +4, with positive values representing more wetness than normal and negative values representing more dryness than normal. For the combined dataset, index values for April through August in the years 2006-2010 were obtained from the NCDC website (NOAA, 2012). These monthly values were then averaged to create a single growing season 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

NOAA's Palmer Z drought index, with index values less than -1.25 identified as "dry", values greater than or equal to 1 identified as "wet", and index values between -1.25 and 1 identified as "normal." Values beyond the range from -2.75 to +3.5 could be interpreted as extreme drought and extremely moist, respectively (NCDC, 2012c). The NCDC climate divisions with Palmer Z data are shown in Figure 4C-1.

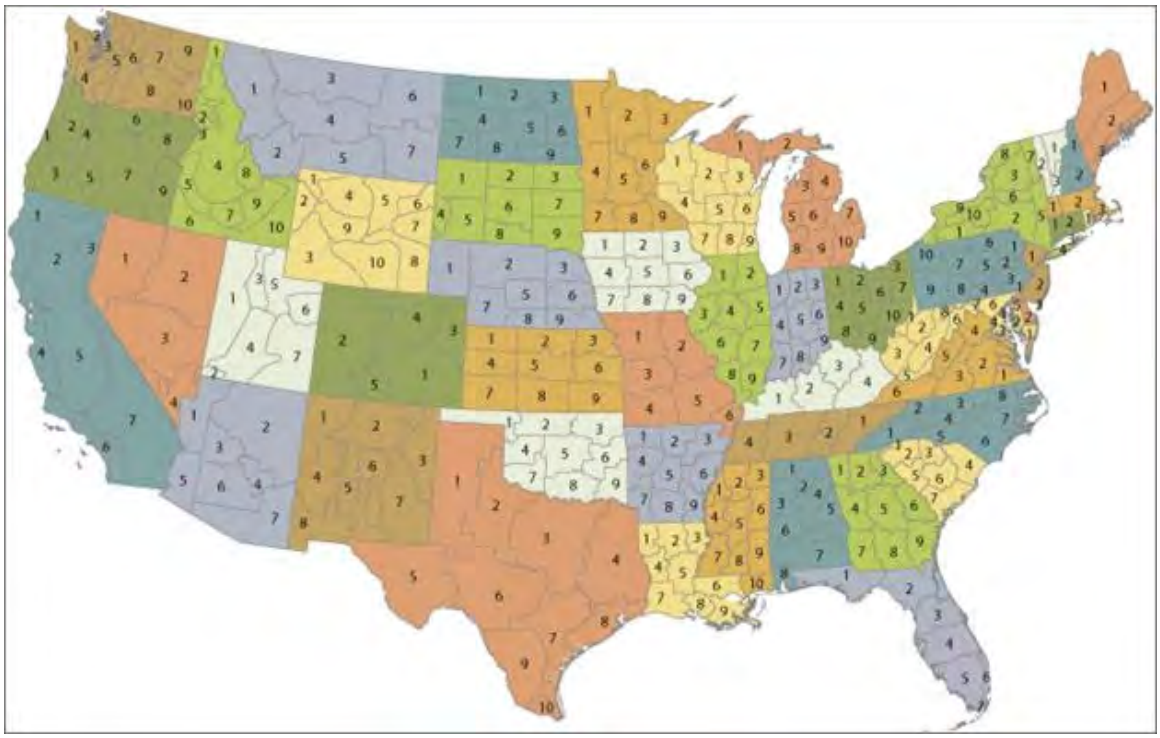


Figure 4C-1. Climate divisions for which there are Palmer Z soil moisture index values.

W126 Index Estimates: Estimates of seasonal W126 exposure index for the years 2006 through 2010 were developed for 12 kilometer (km) by 12 km grid cells in a national-scale spatial surface. The estimates at this scale were derived from applying a spatial interpolation technique to annual W126 values derived from O_3 measurements at ambient air monitoring locations. Specifically, the Voronoi Neighbor Averaging (VNA) spatial interpolation technique was applied to the monitor-location W126 index values to derive an W126 index estimates for each grid cell (U.S. EPA, 2014, Appendix 4A).[2]

Combined Dataset: To create the dataset that relates the grid cells with W126 index estimates to grid cells with BI scores, the EPA provided a file with the national-scale surface of grid cells (a "shape" file) to USFS staff, who assigned the BI scores (with sampling year specified) to grid cells for all but three states. Having this step performed by the USFS ensured

[2] The 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).

that the precise and accurate geographic coordinates for each biosite were used in this step, which allowed the most accurate matching of Palmer Z and W126 index values as possible with these datasets.[3] For three states (California, Oregon, and Washington) the EPA downloaded biosite indices from the public website and assigned them to the grid cells in which the biosite was located based on the publicly available geographic coordinates.[4] The EPA overlaid the Palmer Z dataset for each year on the national surface of W126 index estimates for that year to assign a Palmer Z index to each grid cell in each year's national surface. The completed dataset (Smith and Murphy, 2015, Appendix) includes the following variables: identifier, year, W126 index, BI score, Palmer Z index, state and soil moisture category (dry, wet, normal)[5].

4C.3 DATASET CHARACTERISTICS

The dataset for the analyses included 5,284 biosite records distributed across the 37 different states and the five years from 2006 – 2010 (Smith and Murphy, 2015, Appendix). Figure 4C-2, reprinted from 2014 WREA, indicates the distribution of sites across the continental U.S. Table 4C-2 summarizes the biosite index values for each year. The "Damage" categories used follow the USFS risk categories with the exception of including a separate category for a biosite index of zero (Smith, 2008, 2012). The zero category was defined and used as a measure of the presence or absence of any level of visible foliar injury. Across all of the sites, over 81 percent of the observations recorded no foliar injury. This percentage was similar across all of the years, with a low value of 78 percent and a high value of 85 percent. Across the 5,284 records in the dataset, only 998 had BI scores greater than zero.

[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."

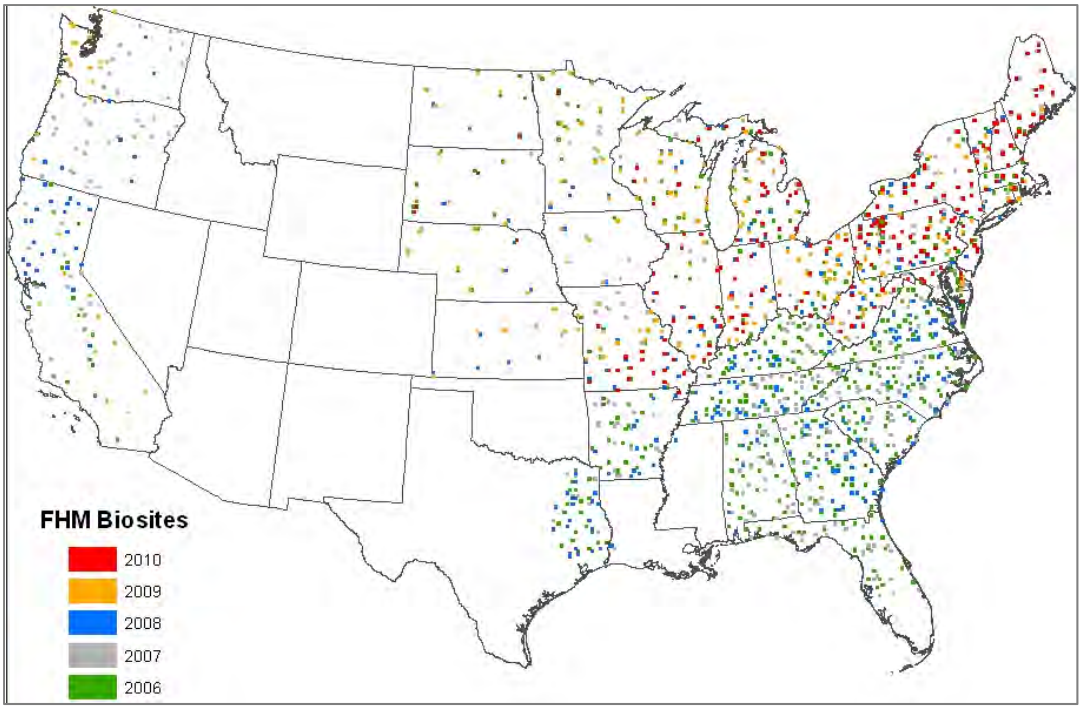


Figure 4C-2. USFS biomonitoring sites for visible foliar injury ("Biosites").

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

4C.4 RELATIONSHIPS OF BIOSITE INDEX SCORES WITH W126 ESTIMATES AND SOIL MOISTURE CATEGORIES

4C.4.1 Relationships Examined in Full Dataset

Scatterplots of the full dataset show no clear relationship between O_3 and biosite index (Figure 4C-3), as well as no clear relationship between O_3 and the Palmer Z drought index, measured as an average value of the months from April to August (Figure 4C-4). The lack of a clear relationship is partly due to the high number of observations with no foliar injury (see Table 4C-1 above and also the distribution of records by soil moisture category and W126 summarized in section 4C.4.2 below) and may also reflect, in part, differing spatial resolutions of

the O_3 exposure surface, NCDC climate divisions, and the biosites. To investigate the strength of any relationship in light of the high percentage of zero values, a censored regression was conducted using a threshold of zero (i.e., including only the non-zero observations). The results of the regression (Table 4C-2) are consistent with the evaluation of the evidence in the ISA (and prior ISA and AQCDs), indicating a significant relationship between foliar injury and both O_3 and moisture (as measured by Palmer Z), and also a significant interaction between O_3 and moisture. The censored regression does not provide a "goodness of fit" statistic as easily interpreted as the r-squared value associated with a standard regression, so the results are more difficult to interpret. Thus, while higher O_3 corresponds to higher BI score, the parameters describing such a relationship in predictive quantitative terms are unresolved.

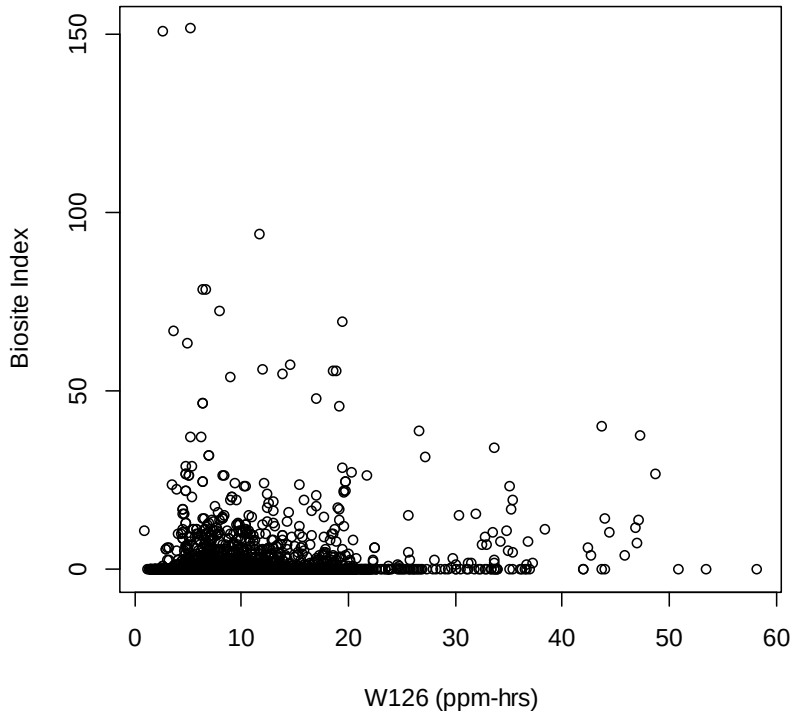


Figure 4C-3. Scatter plot of biosite index score *versus* W126 index (ppm-hrs).

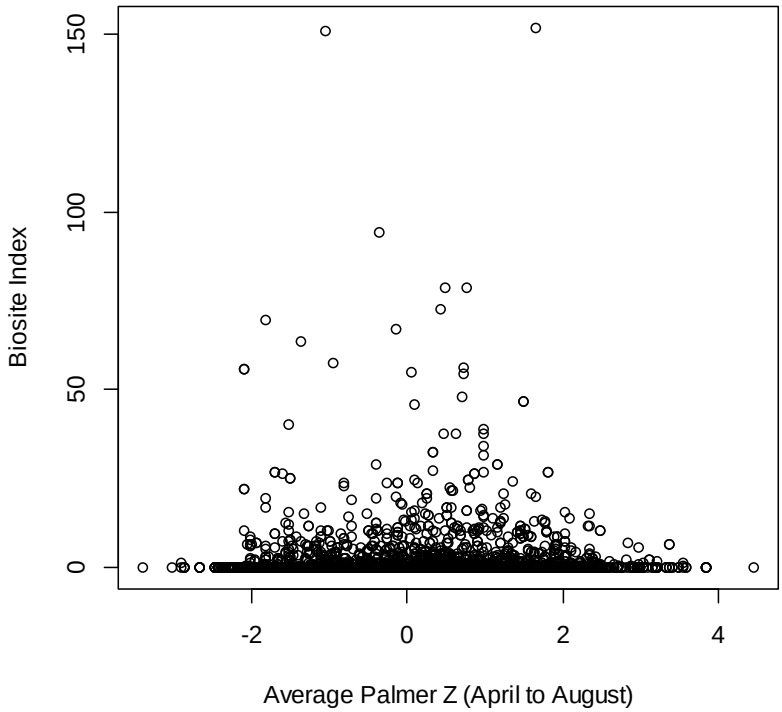


Figure 4C-4. Scatter plot of biosite index score *versus* Palmer Z (April to August).

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

An exploration (in the 2014 WREA) of the use of regression coefficients to calculate estimated biosite index values did not accurately predict the observed values, likely due in part to the large number of non-injury observations. It is also of note that the W126 index bin with the largest percentage of records of each category of BI score (e.g., all, zero, above zero, above 5, above 15) is that for the lowest W126 index values (0).

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
Cumulative Percentage of Records (percent of records in bin plus all bins to its left)								
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%

4C.4.2 Examination of Relationships in Dataset Stratified by Soil Moisture Category

The following tables and figures describe the data in this dataset with a focus on consideration of potential trends with W126 index for the different soil moisture categories. The W126 index estimates were rounded to integer values for consistency with Appendix 4D analyses and associated clarity in binning of the values.[6] Additionally, consistent with USFS publications (e.g., Campbell et al., 2007), the BI scores[7] are rounded to one decimal place. Table 4C-4 presents the counts of records in total and stratified by soil moisture category and W126 index bin. Table 4C-5 presents average BI scores by soil moisture category and W126 bin, and Table 4C-6 presents the fraction of records with BI scores of differing severity levels (corresponding to the USFS severity scheme), in the full dataset and also in the subsets by soil moisture category.

The distribution of records across W126 bins are presented in Table 4C-4 and Figure 4C-5, and the distribution of scores per bin is presented in Figure 4C-6 through Figure 4C-11. These figures show that even the lowest W126 index bin (for estimates below 7 ppm-hrs) includes scores well above 5, and several above 15. Further, zero scores comprise more than half the dry 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.

seen by the median lines merged with the zero line in Figure 4C-6 and Figure 4C-8. This is also the case for all but the two highest bins for the wet soil moisture category records, which, however, contain just a total of 9 records, limiting the extent to which they provide a basis for interpretation of patterns across W126 bins. The wet soil moisture records have quite limited sample sizes for the higher W126 index bins, e.g., the number of samples in bins for W126 index estimates above 13 ppm-hrs represent no more than 1 percent of the total number of wet soil moisture records (Figure 4C-10).

Focusing on the distribution of scores for records in the normal soil moisture category, it can be seen that scores are noticeably increased in the highest W126 bin, index estimates greater than 25 ppm-hrs, over those for the lower bins (Figure 4C-6 and Figure 4C-7). This is also for the average BI scores per bin, where the highest W126 bin (>25 ppm-hrs) has an average BI appreciably higher than the others (Table 4C-5). The average BI in this highest bin is 7.9 *versus* averages of 1.6 (for W126 >19 to 25 ppm-hrs) and 2.3 (for W126 >17 to 19 ppm-hrs) in the next lower bins and varying from 0.8 to 1.2 in all the others. Among the records with nonzero scores, the highest average BI is also in the highest W126 index bin (>25 ppm-hrs); in this case the BI is approximately 15, more than double the next highest average BI scores for any of the other W126 index bins (for which no other trend is exhibited). The incidence of records with BI scores categorized by the USFS as “moderate” or “severe” injury (BI score above 15) is also greatest in the bin for the highest W126 index estimates (> 25 ppm-hrs), with 20% of those records in this bin having such a BI score compared to only 2 to 4% of the records in each of the lower bins. A similar pattern holds for the records with BI scores above 5, while there is much more variability across the bins for records with any nonzero score (Table 4C-6).

With regard to the dry soil moisture category, there is a suggestion of an increased incidence of the highest severity scores in the highest two W126 bins. For example, the proportion of dry soil moisture category records with BI scores categorized by the USFS as “moderate” or “severe” injury (BI score above 15), is 7 and 8% in the bin for the two highest W126 index estimates (>19 to 25 and > 25 ppm-hrs, respectively), compared to 0 to 3% in each of the lower bins. It is noteworthy, however, that the percentages of 7 and 8% reflect no more than 4 or 5 individual records with this severity score.

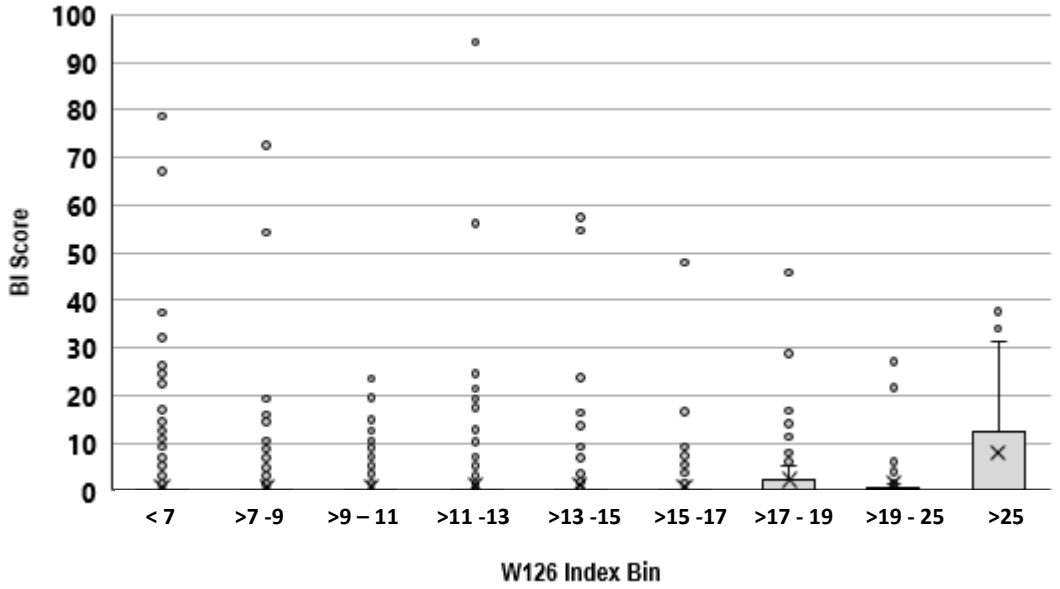
As noted above, sample size for the wet soil moisture category is particularly limited for the W126 index bins above 13 ppm-hrs. In the lower W126 bins, the proportion of such records with BI scores above 15 varies from 1 to 2%. For BI scores above 5 or above 0, there is a suggestion of an increased incidence in the relatively higher *versus* lower W126 index bins, although it is not known if the significant reduction in sample size that also occurs in comparing across these bins (see Table 4C-4) and associated variability is playing a role (Table 4C-6).

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

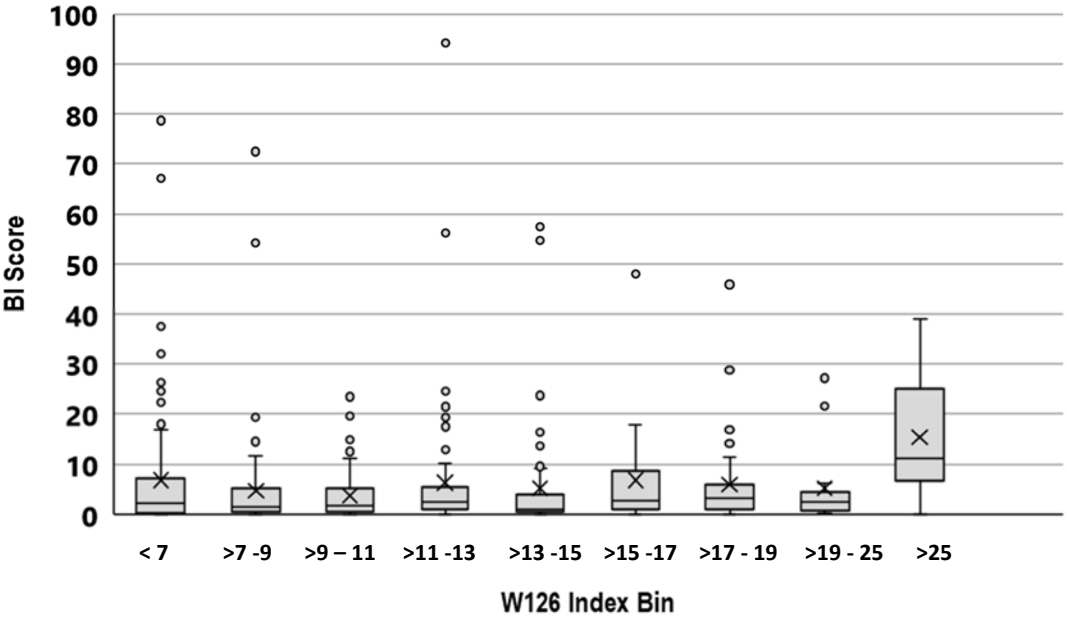
[A] As noted in the beginning of section 4C.1.2, this count reflects the omission of two outlier values.
[B] Sample size for this W126 bin is below 1% of all samples assigned this soil moisture category.

Figure 4C-5. Distribution of biosite records by W126 bin and soil moisture type.



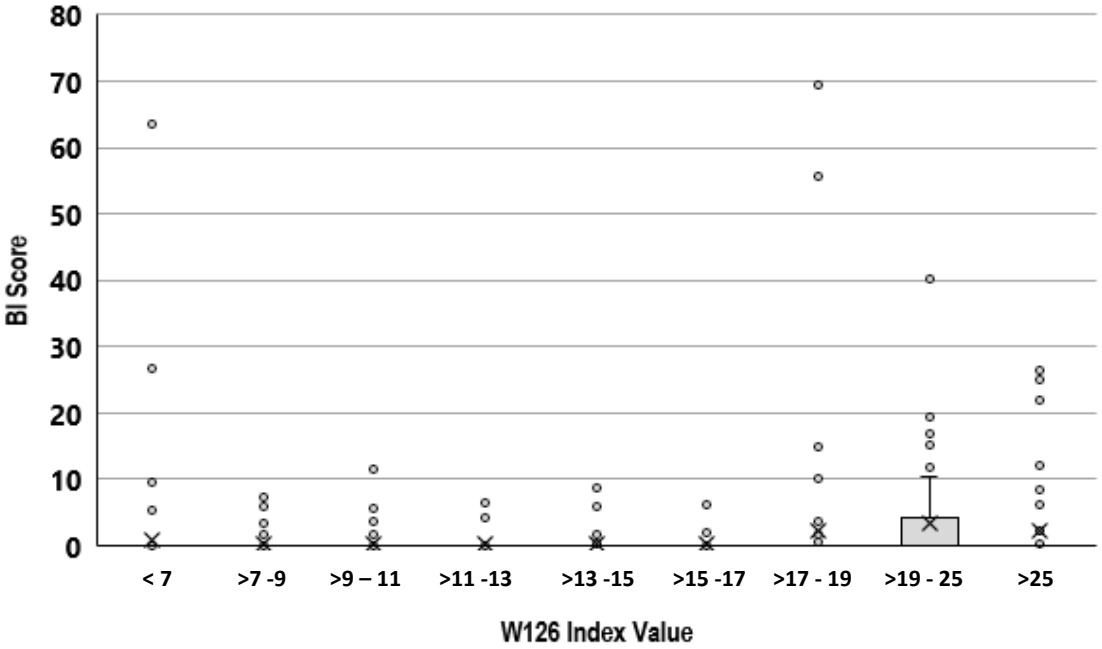
Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

Figure 4C-6. Distribution of BI scores (including zeros) at USFS biosites (normal soil moisture) grouped by W126 index values.



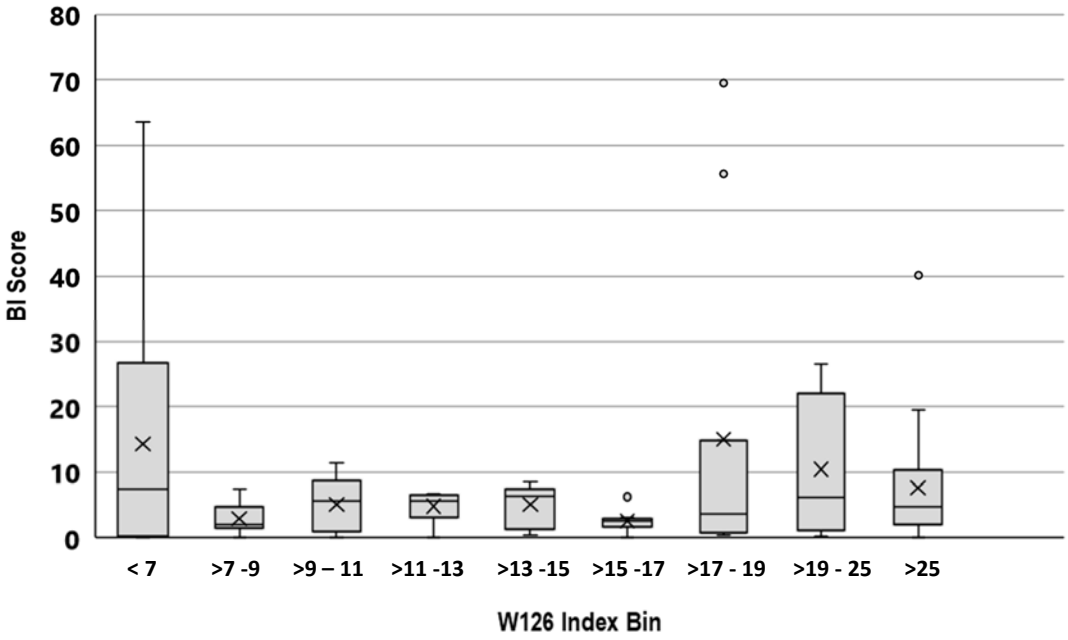
Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

Figure 4C-7. Distribution of nonzero BI scores at USFS biosites (normal soil moisture) grouped by W126 index values.



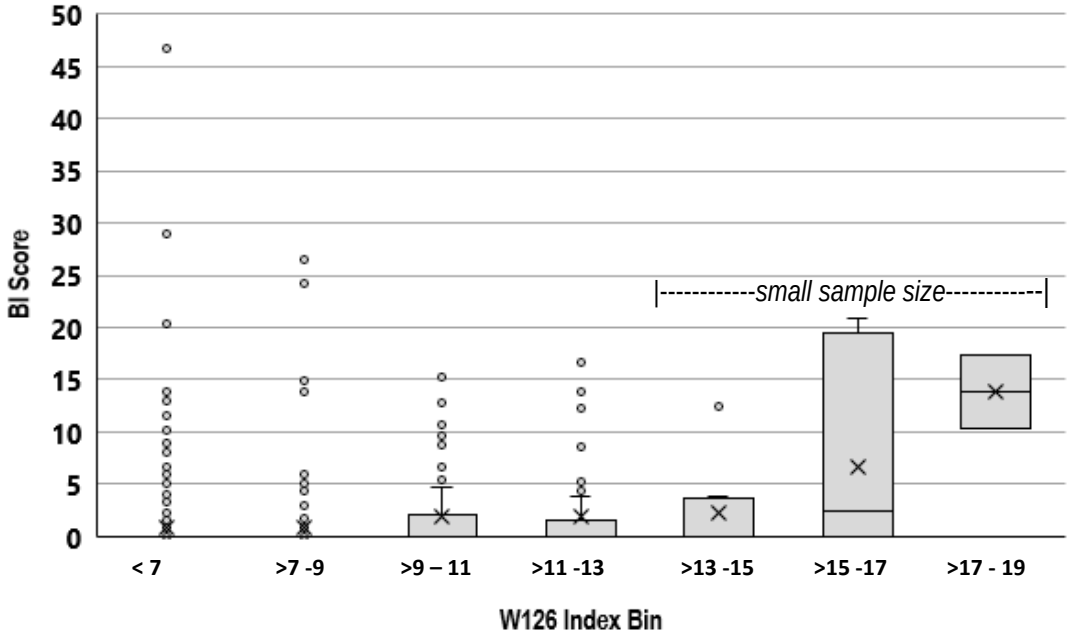
Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus 1.5 times the interquartile range (75th minus 25th percentile). Circles show scores higher than that.

Figure 4C-8. Distribution of BI scores (including zeros) at USFS biosites (dry soil moisture) grouped by W126 index values.



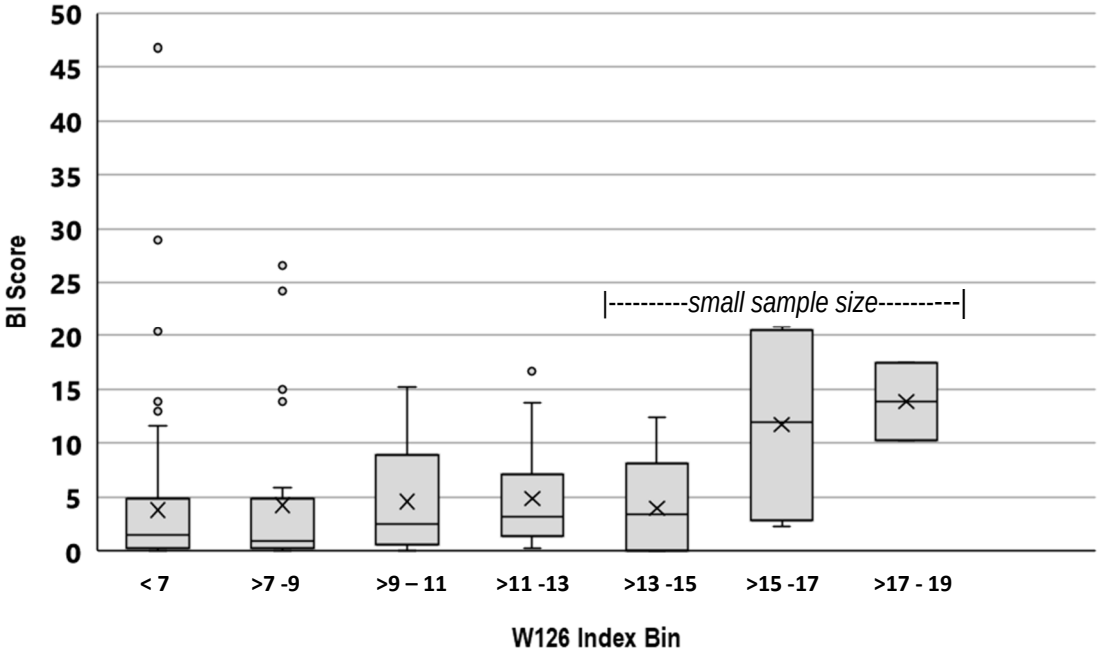
Key: The boxes denote the 25th, 50th and 75th percentiles, the x's the mean and the whiskers denote the value equal to the 75th percentile plus (or 25th percentile minus) 1.5 times the interquartile range (75th minus 25th percentile). Circles show still higher scores

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.

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).

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.

The observations of visible foliar injury for the highest W126 bin compared to the others is generally consistent with the evidence regarding visible foliar injury as an indicator of O_3 exposure (e.g., ISA, Appendix 8, section 8.2; 2013 ISA, section 9.4.2; U.S. EPA, 2006 [2006 AQCD], p. AX9-22). The evidence indicates a generally greater extent and severity of visible foliar injury with higher O_3 exposure levels and an influence for soil moisture conditions (ISA, Appendix 8, Section 8.2). Further, consistent with this evidence, the censored regression of the USFS dataset described in section 4C.1.1 above found a significant relationship between visible foliar injury and both O_3 and moisture, as measured by Palmer Z.

A study cited in the current and 2013 ISAs, which analyzed trends in the incidence and severity of foliar injury, observed a declining trend in the incidence of foliar injury as peak O_3 concentrations declined (2013 ISA, p. 9-40; Smith, 2012). Another study, also available in the last review, that focused on O_3-induced visible foliar injury in west coast forests observed that both percentage of biosites with injury and average BI were higher for sites with average cumulative O_3 concentrations above 25 ppm-hrs in terms of SUM06[8] as compared to groups of biosites with lower average cumulative exposure concentrations, with much less clear differences between the two lower exposure groups (Campbell et al., 2007, Figures 27 and 28 and p. 30). A similar finding was reported in the 2007 Staff Paper which reported on an analysis that showed a smaller percentage of biosites with injury among the group of biosites with O_3 exposures aot or below a SUM06 metric of 15 ppm-hrs or a 4th high metric of 74 ppb as compared to larger groups that also included biosites with SUM06 values up to 25 ppm-hrs or 4th high metric up to 84 ppb, respectively (U.S. EPA, 2007 [2007 Staff Paper], pp. 7-63 to 7-64).

The observations described here have a general consistency with the extensive evidence base on foliar injury, which indicates that visible foliar injury prevalence and severity are generally higher at higher (compared to lower) O_3 concentrations. As the FIA/FHM biosites vary in the type of vegetation and species that are present and the vegetation types and species vary in sensitivity, BI scores would be expected to differ even between two biosites identical in all environmental characteristics when there are different species present. Therefore, limitations in the biosite dataset can affect patterns and relationships observed in the BI scores. Additionally, various environmental and genetic factors influence the exposure-response relationship, with the most well understood being soil moisture conditions (ISA, Appendix 8, Section 8.2). Our understanding of specific aspects of these influences on the relationship between O_3 exposures, the most appropriate exposure metrics, and the occurrence or severity of visible foliar injury is, however, still incomplete.

[8] Based on an approach used in the 2007 Staff Paper (and the associated temporal patterns of O_3 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).

4C.5 LIMITATIONS AND UNCERTAINTIES

The purpose of the analyses and presentations summarized above was to investigate the potential relationship between BI scores at USFS biosites and O_3 in terms of the seasonal W126 index. The lack of a clear relationship (across W126 bins below 25 ppm-hrs) in the presentations above may relate to inherent limitations and uncertainties in the different aspects of the dataset. The limitations and uncertainties associated with aspects of the dataset developed for the 2014 WREA, and further investigated above, are presented here. In summarizing these below, they are grouped into four areas: 1) biosite scores, 2) soil moisture categorization, 3) W126 index estimates, and 4) combining of datasets.

Biosite data: Site selection, availability, and species presence also contribute to uncertainty within the dataset and analysis. Data are lacking from many western states including Montana, Idaho, Wyoming, Nevada, Utah, Colorado, Arizona, New Mexico, Oklahoma, and portions of Texas. Furthermore, in certain states (California, Washington, and Oregon) exact locations of sampled sites were not available, and these sites were assigned to the grid based on publicly available geographic coordinates, increasing the level of uncertainty. Because the grid sizes are relatively small, limiting the geographic skew of estimated location (7 km in any direction), it is likely that these locations were at least assigned to adjacent grid cells. While the extent of such differences and magnitude of any effect on the resultant dataset are unknown, it may have relatively small difference and low magnitude of influence on the dataset (2014 WREA, p. 7-60).

Soil moisture categories: The use of the Palmer Z soil moisture index contributes uncertainty of unknown directionality and magnitude. Short-term estimates of soil moisture can be highly variable from month to month within a single year. Using averages contributes to a potential temporal mismatch between soil moisture and injury. Soil moisture is also substantially spatially variable, and the soil moisture data can be hundreds of miles wide in climate regions. There is much diversity within regions, and some vegetation, such as that along riverbanks, may experience sufficient soil moisture during periods of drought to exhibit foliar injury. All of these factors contribute uncertainty to this categorization (2014 WREA, p. 7-61).

W126 index estimates: Ambient air quality measurements have some inherent uncertainties (considered low [2014 WREA, p. 4-39]) associated with them. These uncertainties relate to monitoring network design, O_3 monitoring seasons, monitor malfunctions, wildlife and wildfire/smoke impacts, and interpolations of missing data. There is likely somewhat greater uncertainty associated with the assignment of W126 index estimates to all biosites due to the need for interpolating between monitor sites to estimate concentrations in unmonitored areas

(2014 WREA, sections 4A.2.1).[9] Accordingly, there is relatively greater uncertainty associated with sites at some distance from monitoring sites and lesser uncertainty in densely monitored areas (2014 WREA, p. 4-40). Unfortunately, which sites are which is unknown.

Combining datasets: Uncertainty is associated with the combination of data types of different spatial resolution. For example, the biosite scores are available at a much finer spatial resolution than the W126 index estimates, which represent a much small spatial area than that represented by the soil moisture categorization. Yet, as recognized above, soil moisture may vary on much finer scales. To avoid losing resolution of the finest-scale dataset (the biosite scores), the finest spatial resolution available was used (e.g., rather than averaging the BI scores across the grids for which W126 index was estimated or across the climate regions for which the soil moisture scores area available), although this approach contributes its own uncertainty.

There is also uncertainty in the combination step associated with the differing temporal scales or time-of-year represented by the three types of data.

Overall, we recognize a number of limitations and uncertainties that may be affecting our ability to identify a relationship between O_3, as quantified by seasonal W126 index, and visible foliar injury at USFS locations (based on BI scores), particularly at sites with W126 index estimates at or below 25 ppm-hrs.

4C.6 SUMMARY AND KEY OBSERVATIONS

The following are key observations concerning the dataset presented in this appendix, which includes the subset of USFS biosite data for the years 2006 through 2010, and for which limitations and uncertainties are recognized in section 4C.5 above.

Full Dataset:

- The combined dataset includes more than 5,000 records, each of which documents a biotic index scores, soil moisture index value and W126 index estimate, for USFS biosites in 37 states in one or more years from 2006 to 2010.
- The majority of the records are for W126 index estimates at or below 9 ppm-hrs, with fewer than 10% of records assigned W126 index estimates above 15 ppm-hrs.
- The BI scores (in all soil moisture categories) are quite variable, with at least half the scores in nearly all bins being zero, and even the bin for the lowest W126 index estimates (below 7 ppm-hrs) having at least one scores above 5 and 15.
- With regard to soil moisture conditions most of the dataset (61% of all records) are for 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).

records for wet soil moisture conditions than dry, with 23% of all records categorized as wet soil moisture conditions and 16% as dry soil moisture.

Records in Wet Soil Moisture Category:

- The wet soil moisture records are concentrated in the two lower W126 index bins which contain nearly 90% of all records for this soil moisture category.
  - Accordingly, interpretations of patterns across W126 bins for this soil moisture category are limited by small sample size across the bins. For example, the number of records in each of the W126 bins above 13 ppm-hrs (ranging from zero to 9) comprise less than 1% of the records in this soil moisture category.

Records in Normal Soil Moisture Category:

- Among records in the normal soil moisture category, BI scores are noticeably increased in the highest W126 index bin (index estimates above 25 ppm-hrs), averaging 7.9.
  - The percentages of records in this W126 bin with scores above 15 or above 5 are more than three times greater than percentages for these score magnitudes in any of the lower W126 index bins.
  - The average BI score for records in the highest W126 bin is also appreciably greater than scores for records in the other bins. The average scores in the next two highest W126 bins are 1.6 and 2.3, respectively, which are only slightly higher than average scores for the rest of the bins, which vary from 0.8 to 1.2 without a clear relationship to estimated W126 index.
  - Among the records in this category with nonzero scores, the highest average BI is also in the highest W126 index bin (>25 ppm-hrs); in this case the BI is approximately 15, more than double the next highest average scores across the other W126 index bins (for which no other trend is exhibited). The proportion of records with any injury is also highest in the highest W126 index bin; it is also slightly increased in the next lower W126 bins compared to the rest (the bins at or below 17 ppm-hrs) across which there is little evident pattern.

Records in Dry Soil Moisture Category:

- Dry soil moisture category records in the two highest W126 bins (>19 and > 25 ppm-hrs) exhibit the greatest percentages of records with BI above 15 and above 5. For scores above 15, the percentages are 7 and 8% compared to 0 to 3 % in the other bins, and for scores above 5, the scores are 11 and 20% compared to 1 to 5% in the other bins.

In summary, the observations described here are generally consistent with the extensive evidence base on foliar injury and O_3, which indicates that foliar injury prevalence and severity are generally higher at higher (compared to lower) O_3 concentrations. The presentations here of USFS data do not indicate clear trends in BI across the full range of W126 index estimates. Rather, they indicate increased BI for the highest estimates, with the increase in both incidence of higher scores and in average score being most clear for W126 index estimates above 25 ppm-hrs, with a suggestion of slight increase for some records with W126 index estimates above 17 or

19 ppm-hrs (dry soil moisture category). Variability as well as sample size limitations contribute to the lack of more precise conclusions. Additionally, as indicated in the evidence summarized in the ISA and prior scientific assessments, various environmental and genetic factors influence the exposure-response relationship. Our understanding of specific aspects of these influences on the relationship between O_3 exposures, the most appropriate exposure metrics, and the occurrence or severity of visible foliar injury is, however, still incomplete.

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APPENDIX 4D

ANALYSIS OF THE W126 O_3 EXPOSURE INDEX AT U.S. AMBIENT AIR MONITORING SITES

Table of Contents

4D.1 OVERVIEW

This appendix presents various analyses of ambient air monitoring data for ozone (O_3) concentrations in the U.S. relating to the W126-based cumulative exposure index. These analyses focus on the annual maximum 3-month sum of daytime hourly weighted O_3 concentrations, averaged over 3 consecutive years, hereafter referred to as the "W126 metric," calculated as described in section 2 below. These analyses examine spatial and temporal patterns in the W126 metric using monitoring data from 2000 to 2018 and make various comparisons between the W126 metric and design values for the current O_3 standard (the annual 4th highest daily maximum 8-hour O_3 concentration, averaged over 3 consecutive years; hereafter referred to as the "4th max metric"). Additional analyses assess the relative variability between the W126 metric and its constituent annual index values and the magnitude of W126 index values at monitoring sites in or near federally protected ecosystems known as Class I areas. These analyses are largely parallel to analyses that were completed for the last review of the O_3 NAAQS (79 FR 75331, December 17, 2014; 80 FR 65385, October 26, 2015; U.S. EPA, 2014a, Wells, 2014, Wells, 2015).

4D.2 DATA HANDLING

4D.2.1 Data Retrieval and Preparation

Hourly O_3 concentration data were retrieved from the EPA's Air Quality System (AQS, *https://www.epa.gov/aqs*) database for 1,981 ambient air monitoring sites which operated between 2000 and 2018. These data were used to calculate W126 and 4th max metric values for each 3-year period from 2000-2002 to 2016-2018. Before calculating these metrics, some initial processing was done on the hourly data. First, data collected using monitoring methods other than federal reference or equivalent methods, and data collected at monitoring sites not meeting EPA's quality assurance or other criteria in 40 CFR part 58 were removed from the analysis. Second, data collected by multiple monitoring instruments operating at the same location were combined according to Appendix U to 40 CFR Part 50. Finally, data were combined across 95 pairs of monitoring sites approved for such combination by the EPA Regional Offices. The final hourly O_3 concentration dataset contained 1,788 monitoring sites.

4D.2.2 Derivation of the 4th Max and W126 Metrics

The 4th max metric values were calculated according to the data handling procedures in Appendix U to 40 CFR part 50. First, moving 8-hour averages were calculated from the hourly O_3 concentration data for each site. For each 8-hour period, an 8-hour average value was calculated if there were at least 6 hourly O_3 concentrations available. Each 8-hour average was stored in the first hour of the period (e.g., the 8-hour average from 12:00 PM to 8:00 PM is

stored in the 12:00 PM hour). Daily maximum 8-hour average values were found using the 8-hour periods beginning from 7:00 AM to 11:00 PM each day. These daily maximum values were used if at least 13 of the 17 possible 8-hour averages were available, or if the daily maximum value was greater than 70 parts per billion (ppb). Finally, the annual 4th highest daily maximum value was found for each year, then averaged across each consecutive 3-year period to obtain the final set of 4th max metric values in units of ppb. Any decimal digits in these values were truncated for applications requiring direct comparison to a 4th max level (e.g., Table 4D-2), otherwise, all decimal digits were retained. The 4th max metric values were considered valid if daily maximum values were available for at least 90% of the days in the O_3 monitoring season (defined in Appendix D to 40 CFR part 58) on average across the three years, with a minimum of 75% of the days in the O_3 monitoring season in any calendar year. In addition, 4th max metric values were considered valid if they were greater than the 4th max levels to which they were being compared.

The W126 metric values were calculated using the hourly O_3 concentration data in parts per million (80 FR 65374, October 26, 2015). For daytime hours (defined as the 12-hour period from 8:00 AM to 8:00 PM Local Standard Time each day), the hourly concentration values at each O_3 monitoring site were weighted using the following equation:

$$\text{Weighted } O_3 = O_3 / (1 + 4403*\exp(-126 * O_3)).$$

These weighted values were summed over each calendar month, then adjusted for missing data (e.g.; if 80% of the daytime hourly concentrations were available, the sum would be multiplied by 1/0.8 = 1.25) to obtain the monthly W126 index values. Monthly W126 index values were not calculated for months where fewer than 75% of the possible daytime hourly concentrations were available. Next, moving 3-month sums were calculated from the monthly index values, and the highest of these 3-month sums was determined to be the annual W126 index. Three-month periods spanning multiple years (e.g., November to January, December to February) were not considered in these calculations. The annual W126 index values were averaged across each consecutive 3-year period to obtain the final W126 metric values, with units in parts per million-hours (ppm-hrs). The W126 metric values were rounded to the nearest unit ppm-hr for applications requiring direct comparison to a W126 level (e.g., Table 4D-3), otherwise, all decimal digits were retained. For consistency with the 4^{th} max metric calculations, the W126 metric values were considered valid if hourly O_3 concentration values were available for at least 90% of the daytime hours during the O_3 monitoring season on average across the three years, with a minimum of 75% of the daytime hours during the O_3 monitoring season in any calendar year. Also for consistency with the 4^{th} max metric calculations, the W126 metric

values were considered valid if they were greater than the W126 levels to which they were being compared.

In the final dataset, 1,557 of the 1,788 O_3 monitoring sites had sufficient data to calculate valid 4th max and W126 metric values for at least one 3-year period between 2000-2002 and 2016-2018. The number of sites with valid 4th max and W126 metric values ranged from a low of 992 in 2000-2002 to a high of 1,119 in 2015-2017, and 543 sites had valid 4th max and W126 metric values for all seventeen 3-year periods.

4D.2.3 Derivation of Temporal Trends

Site-level trends for the W126 metric and annual W126 index values were computed in a similar manner to the site-level trends for the 4th max metric presented in Chapter 2. Specifically, for the annual W126 index, a site must have at least 75% annual data completeness for at least 15 of the 19 years, with no more than two consecutive years having less than 75% data completeness in order to be included in the analysis. For the W126 metric, a site must have a valid W126 metric value (according to the data completeness criteria presented in the previous section) in at least 13 of the 17 3-year periods, and no more than two consecutive 3-year periods that do not havevalid W126 metric values. There were 852 sites meeting these criteria for the annual W126 index and 713 sites meeting these criteria for the W126 metric. The national median, 10th percentile, and 90th percentile values of these site-level trends are presented in Figure 4D-9.

Other analyses presented in Section 4D.3.2.2 use trends in the 4th max and W126 metrics as well as the annual W126 index calculated with non-parametric regression methods. These trends were computed using the Theil-Sen estimator (Sen, 1968; Theil, 1950), a type of regression method that chooses the median slope among all lines crossing through each possible pair of sample points[1]. These trends are reported in units of ppb/yr for the 4th max metric or ppm-hr/yr for the W126 metric and annual W126 index. The data completeness criteria described in the previous paragraph were also applied to site for which these trends were calculated.[2] Statistical tests for significance of the Theil-Sen estimator were computed using the non-parametric Mann-Kendall test (Kendall, 1948; Mann; 1945).

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[1] For example, if applying this method to a dataset with W126 metric values for four consecutive years (e.g., $W126_1$, $W126_2$, $W126_3$, and $W126_4$), the trend would be the median of the per-year changes observed in the six possible pairs of values (e.g., the median of $[W126_4\text{-}W126_3]/1$, $[W126_3\text{-}W126_2]/1$, $[W126_2\text{-}W126_1]/1$, $[W126_4\text{-}W126_2]/2$, $[W126_3\text{-}W126_1]/2$, and $[W126_4\text{-}W126_1]/3$).

[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 13 of the 17 3-year periods, and no more than two consecutive periods that do not have valid 4th max metric values. There were 629 sites meeting these criteria, and all of these sites also met the data completeness criteria for the W126 metric and the annual W126 index.

4D.2.4 Identification of O_3 Monitoring Sites in Federal Class I Areas

The Clean Air Act (section 162) designated certain federally areas as Class I areas. These areas are federally mandated to preserve certain air quality values. Class I designation allows the least amount of deterioration of existing air quality. Areas designated as Class I include all international parks, national wilderness areas which exceed 5,000 acres in size, national memorial parks which exceed 5,000 acres in size, and national parks which exceed 6,000 acres in size, provided the park or wilderness area was in existence on August 7, 1977. There are 158 such areas (e.g., 44 FR 69122, November 30, 1979). Other areas may, and have been, subsequently designated as Class I consistent with the CAA (section 162). As of July 2019, six Class II areas on Tribal lands have been re-designated as Class I.[3]

To identify which O_3 monitoring sites represented air quality in federal Class I areas, shapefiles (i.e., files that specify area boundaries) for all 158 mandated federal Class I areas[4] were downloaded from EPA's Environmental Dataset Gateway (EDG; *https://edg.epa.gov/*) and augmented with the six tribal areas redesignated as Class I. These boundaries were matched to the 1,788 O_3 monitoring sites in the hourly O_3 concentration dataset described in section 4D.2.1. Since Class I areas include federally designated wilderness areas in which permanent structures such as air monitoring trailers are prohibited, if there was no monitor located within the area boundary, the matching was expanded to include the nearest monitoring site within 15 km of the boundary. For each Class I area and 3-year period, if a 4th max or W126 metric value was not available for the nearest monitor, the values from the next nearest monitor within 15 km were used, where applicable. In addition, if a Class I area had multiple monitors inside the boundary, we used the monitor with the highest 4th max metric value in each 3-year period. These monitors were extracted from the 4th max and W126 dataset described in section 4D.2.2, yielding a final Class I areas dataset with a total of 860 records that had valid 4th max and W126 metric values at 79 O_3 monitoring sites representing 65 Class I areas (out of 164 total Class I areas).

4D.2.5 Assignment of Monitoring Sites to NOAA Climate Regions

In order to examine regional differences, many of the further analyses were stratified into the nine NOAA climate regions (Karl and Koss, 1984), which are shown in Figure 4D-1. Since the NOAA climate regions only cover the contiguous U.S., Alaska was added to the Northwest region, Hawaii was added to the West region, and Puerto Rico was added to the Southeast region.

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[3] The Class I areas on Tribal lands as of December 2018 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."

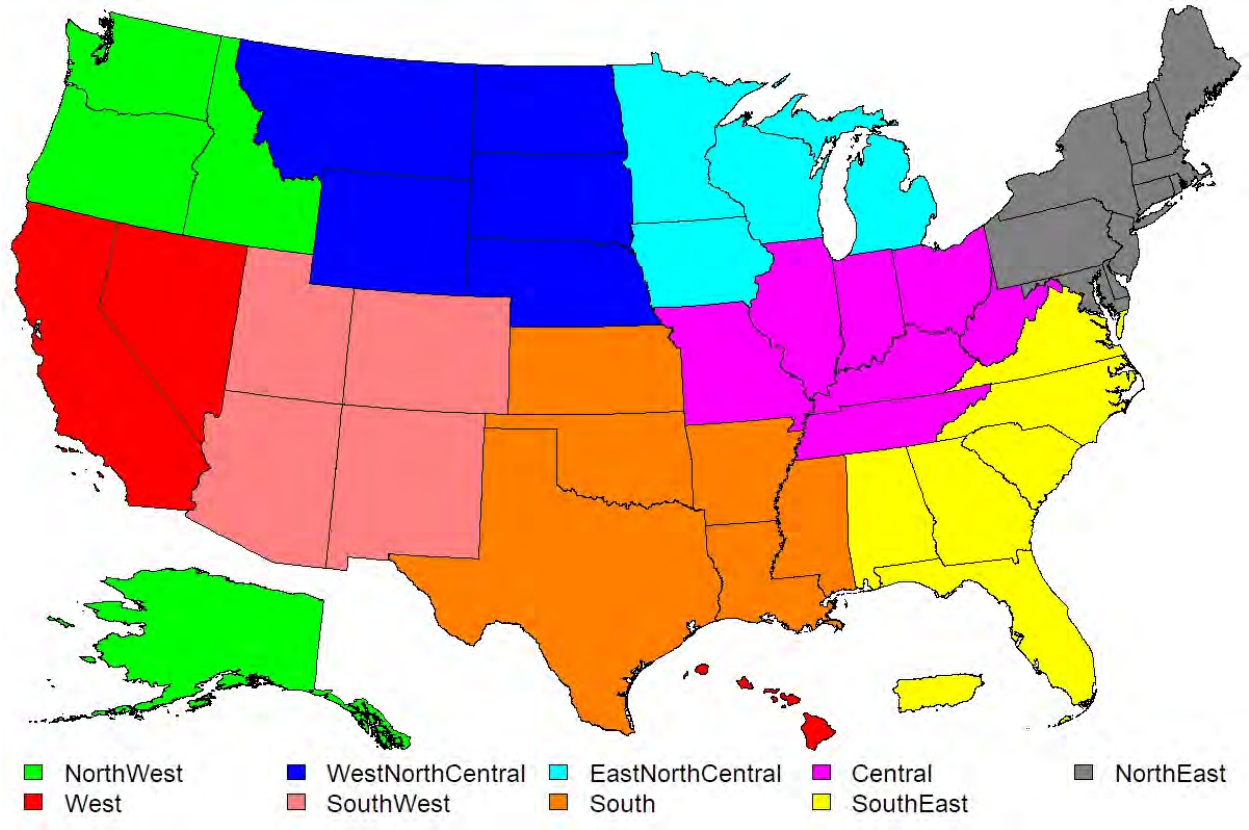

Figure 4D-1. Map of the nine NOAA climate regions.

4D.3 RESULTS

4D.3.1 National Analysis Using Recent Air Quality Data

This section presents various results based on the 4th max and W126 metrics for the 2016-2018 period. Figure 4D-2 shows a map of the observed W126 metric values based on 2016-2018 data. From this figure, it is apparent that W126 metric values are generally at or below 13 ppm-hrs in the eastern and northwestern U.S. In the U.S. as a whole, about 60% of all monitoring sites recorded W126 metric values at or below 7 ppm-hrs, and over 90% of all monitoring sites recorded W126 metric values at or below 17 ppm-hrs. The highest W126 metric values occur in the southwestern U.S. where there are numerous monitoring sites with W126 metric values above 17 ppm-hrs, however, only one of these sites (near Las Vegas, NV), with a W126 metric value of 18 ppm-hrs, meets the current standard. Table 4D-1 shows the number of sites in each NOAA climate region that have a valid 2016-2018 design value meeting the current standard and the number of sites in each region that have a 2016-2018 design value not meeting the current standard.

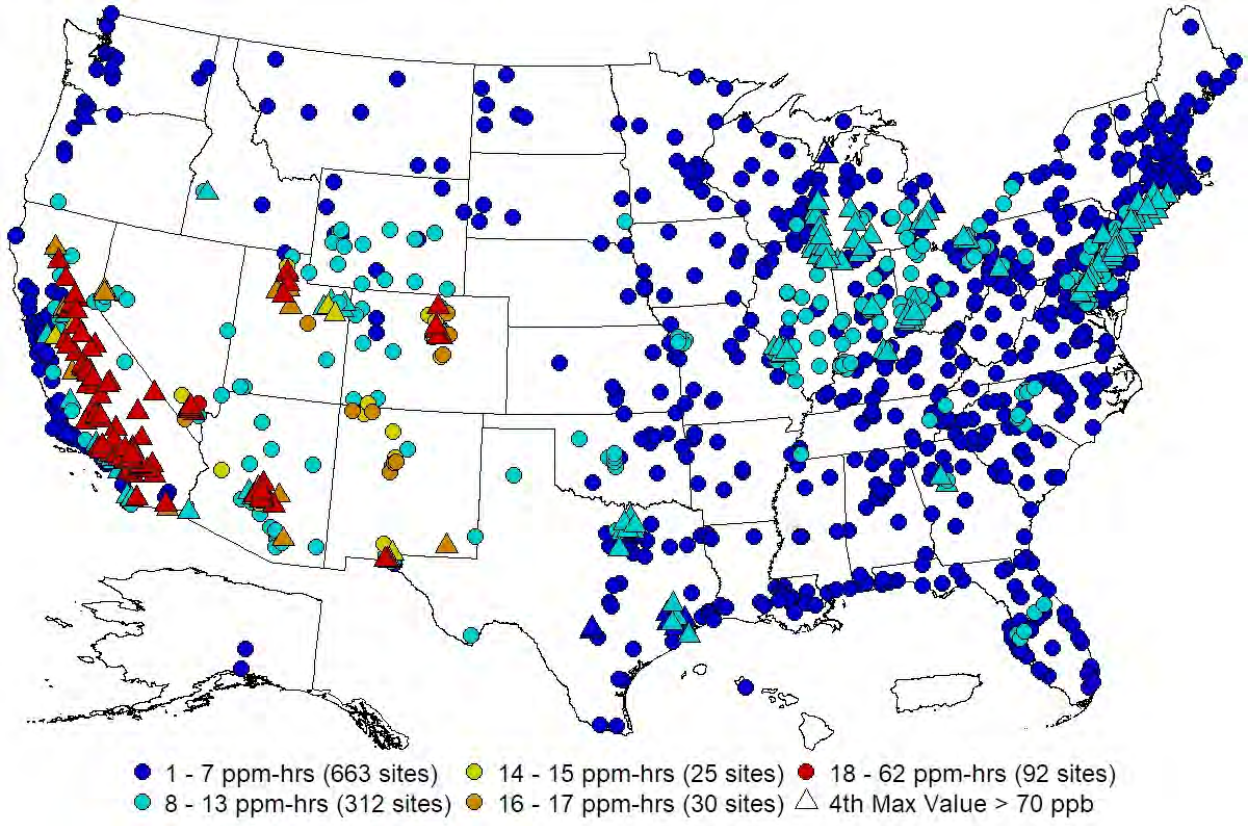

Figure 4D-2. Map of W126 metric values at U.S. O_3 monitoring sites based on 2016-2018 data. Circles indicate monitoring sites with 4th max metric values less than or equal to 70 ppb, while triangles indicate monitoring sites with 4th max metric values greater than 70 ppb.

Table 4D-1. Number of O_3 monitoring sites with valid 2016-2018 design values in each NOAA climate region

NOAA Climate Region	Total # of Sites	# of Sites with Design Value ≤ 70 ppb	# of Sites with Design Value > 70 ppb
Central	202	169	33
EastNorthCentral	86	66	20
NorthEast	179	130	49
NorthWest	26	22	4
South	132	105	27
SouthEast	166	164	2
SouthWest	109	65	44
West	176	82	94
WestNorthCentral	46	46	0
National	1122	849	273

4D.3.1.1 Comparison of the 4th Max and W126 Metrics

The following analyses make several comparisons between the 4th max and W126 metric values based on 2016-2018 data. Table 4D-2 shows the number of sites with 4th max metric values greater than each 4th max level, and the number of sites with 4th max metric values less than or equal to each 4th max level. Table 4D-3 shows the number of sites with W126 metric values greater than each W126 level, and the number of sites with W126 metric values less than or equal to each W126 level.

The 4th max and W126 metric values were also compared to each combination of 4th max and W126 levels based on 2016-2018 data. Table 4D-4 shows the number of sites with 4th max metric values greater than each 4th max level, and W126 metric values less than or equal to each W126 level (e.g., 147 sites had 4th max metric values greater than 70 ppb and W126 metric values less than or equal to 13 ppm-hrs). Table 4D-5 shows the number of sites with 4th max metric values less than or equal to each 4th max level, and W126 metric values greater than each W126 level (e.g., 27 sites with a 4th max metric value at or below 70 ppb had a W126 metric value greater than 13 ppm-hrs). Finally, Table 4D-6 shows the number of sites with 4th max metric values greater than each 4th max level, and W126 metric values greater than each W126 level.

Table 4D-2. Number of sites with 4th max metric values greater than various 4th max levels based on 2016-2018 data.

4th Max Level (ppb)	75	70	65
# of Sites > Level	109	273	632
# of Sites ≤ Level	1,006	849	503
Total # of Sites[A]	1,115	1,122	1,135

[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.

Table 4D-3. Number of sites with W126 metric values greater than various W126 levels based on 2016-2018 data.

W126 Level (ppm-hrs)	19	17	15	13	11	9	7
# of Sites > Level	78	92	122	147	204	283	471
# of Sites ≤ Level	1,040	1,027	998	975	923	844	663
Total # of Sites[A]	1,118	1,119	1,120	1,122	1,127	1,127	1,134

[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.

Table 4D-4. Number of sites with 4th max metric values greater than various 4th max levels and W126 metric values less than or equal to various W126 levels based on 2016-2018 data.

# Sites > 4th Max Level AND ≤ W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4th Max Level (ppb)	75	33	30	26	23	16	7	1
	70	185	173	155	147	127	77	26
	65	533	520	491	468	420	350	186

Table 4D-5. Number of sites with 4th max metric values less than or equal to various 4th max levels and W126 metric values greater than various W126 levels based on 2016-2018 data.

# Sites ≤ 4th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4th Max Level (ppb)	75	6	15	40	60	105	175	351
	70	0	1	12	27	59	88	219
	65	0	0	0	0	4	13	30

Table 4D-6. Number of sites with 4th max metric values greater than various 4th max levels and W126 metric values greater than various W126 levels based on 2016-2018 data.

# Sites > 4th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4th Max Level (ppb)	75	72	76	81	84	92	101	107
	70	78	91	110	119	142	192	245
	65	78	92	122	147	200	270	438

According to Table 4D-2, 10% of U.S. O_3 monitoring sites had 2016-2018 4th max metric values greater than 75 ppb, 24% of sites had 4th max metric values greater than 70 ppb, and 56% of sites had 4th max metric values greater than 65 ppb. According to Table 4D-3, 8% of U.S. O_3 monitoring sites had 2016-2018 W126 metric values greater than 17 ppm-hrs, 13% of sites had W126 metric values greater than 13 ppm-hrs, and 41% of sites had W126 metric values greater than 7 ppm-hrs. According to Table 4D-5, there were no monitoring sites with a 4th max metric value less than or equal to 70 ppb and a W126 metric value greater than 19 ppm-hrs, only one monitoring site with a 4th max less than or equal to 70 ppb and a W126 greater than 17 ppm-hrs.

4D.3.1.2 Relationships Between Metrics and the Annual W126 Index

Figure 4D-3 shows a scatter plot comparing the 4th max (x-axis) and W126 (y-axis) metric values based on 2016-2018 data, with points colored by NOAA climate region. This figure indicates that there is a strong, positive, non-linear relationship between the 4th max and W126 metrics. The amount of variability in the relationship between the 4th max and W126 metrics appears to increase as the metric values themselves increase. The relationship between the 4th max and W126 metrics also appears to vary across regions. In particular, the Southwest and West regions (i.e., the southwestern U.S.) appear to have higher W126 metric values relative to their respective 4th max metric values than the rest of the U.S.

Figure 4D-4 shows the same information as Figure 4D-3, but only for monitoring sites meeting the current standard. This figure shows that all monitoring sites meeting the current standard have W126 metric values of 18 ppm-hrs or less, and all sites outside the Southwest and West climate regions have W126 metric values of 12 ppm-hrs or less.

Finally, Figure 4D-5 shows a scatter plot comparing the 4th max metric values (x-axis) to the annual W126 index values (y-axis) based on 2016-2018 data, with points colored by NOAA climate region. This figure shows that the annual W126 index values have a similar positive, non-linear relationship with the 4th max metric values as the W126 metric values. As might be expected, there is generally more variability in the relationship between the annual W126 index values and the 4th max metric values than between the W126 metric values and the 4th max metric values.

Figure 4D-6 shows a scatter plot of the deviations in the 2016, 2017, and 2018 annual W126 index values (y-axis) from the 2016-2018 average W126 metric values (x-axis). This figure shows that the magnitude of the annual W126 index deviations from the 3-year average tend to increase as the W126 metric value increases. About 55% of the annual W126 index values are within +/- 1 ppm-hr of the 3-year average value, about 81% are within +/- 2 ppm-hrs of the 3-year average value, and about 98% are within +/- 5 ppm-hrs of the 3-year average value. Figure 4D-7 also presents the deviations in the 2016, 2017, and 2018 annual W126 index values from their respective 2016-2018 averages, but with the 2016-2018 average 4th max metric values plotted on the x-axis instead of the 2016-2018 average W126 metric values. From this figure it can be seen that lower 4th max metric values generally correspond to smaller inter-annual variation within W126 metric values, especially for sites meeting the current standard.

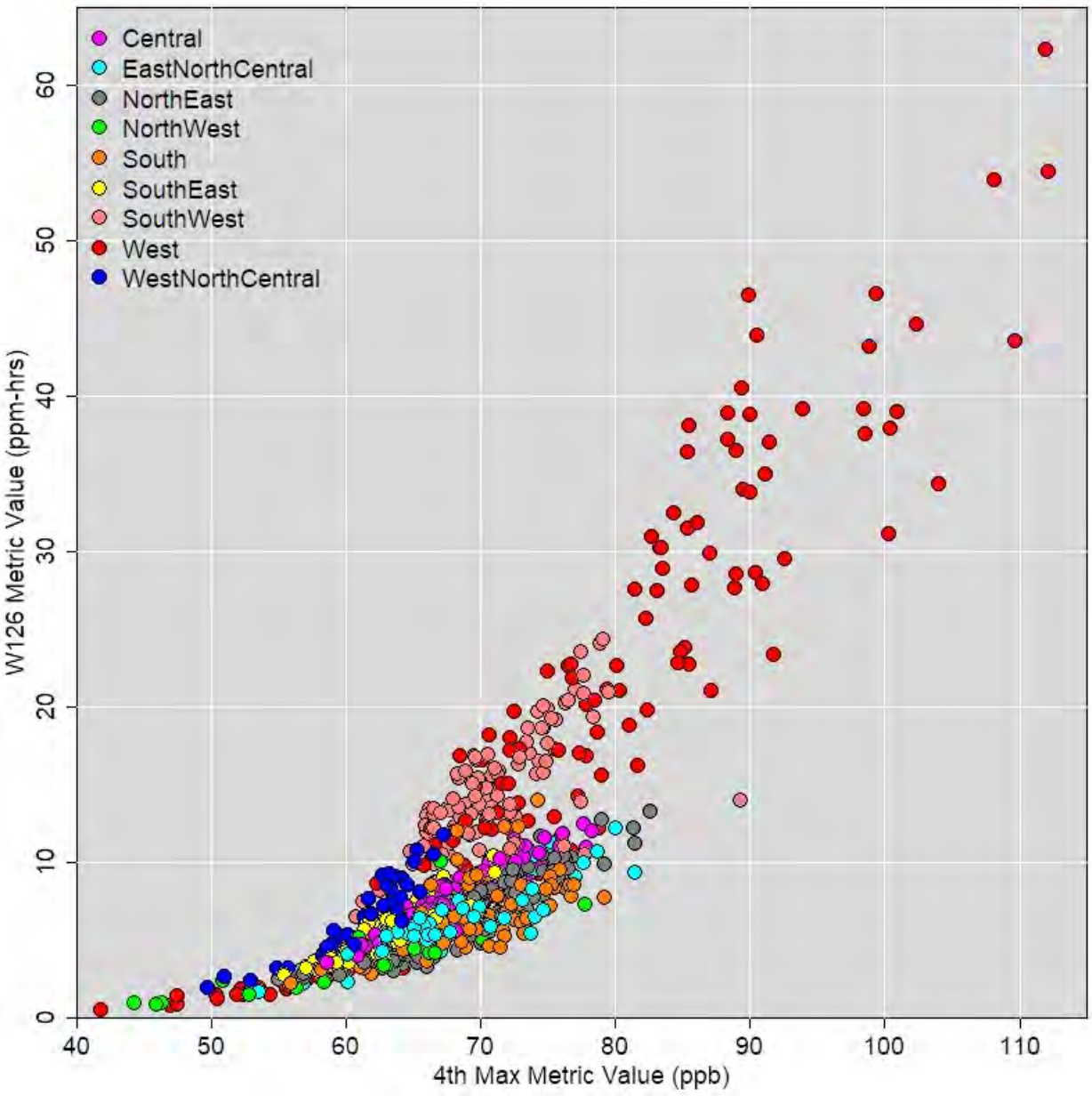

Figure 4D-3. Scatter plot of W126 metric values versus 4th max metric values (design values) based on 2016-2018 monitoring data.

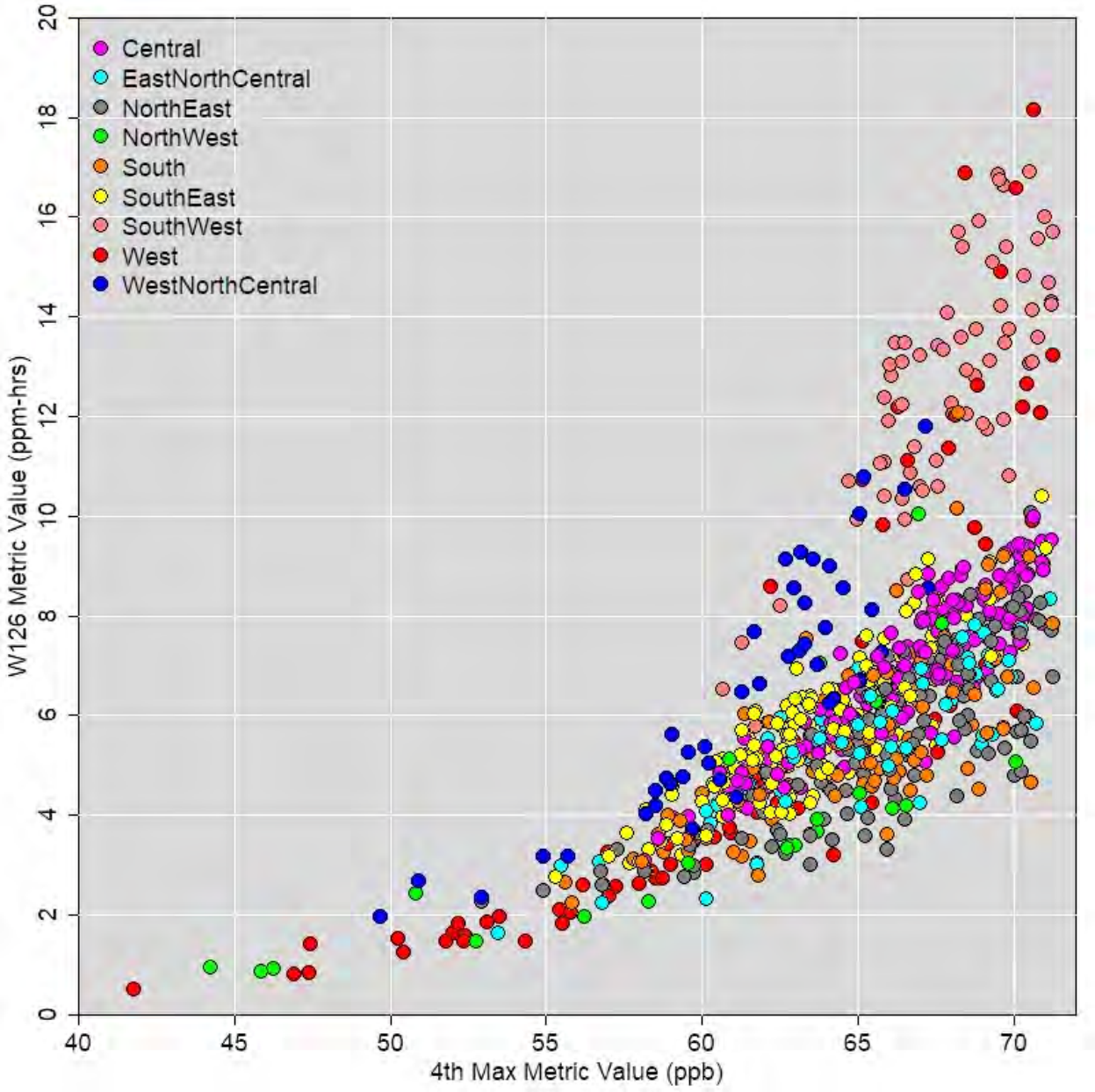

Figure 4D-4. Scatter plot of W126 metric values versus 4th max metric values (design values) at monitoring sites meeting the current standard based on 2016-2018 monitoring data.

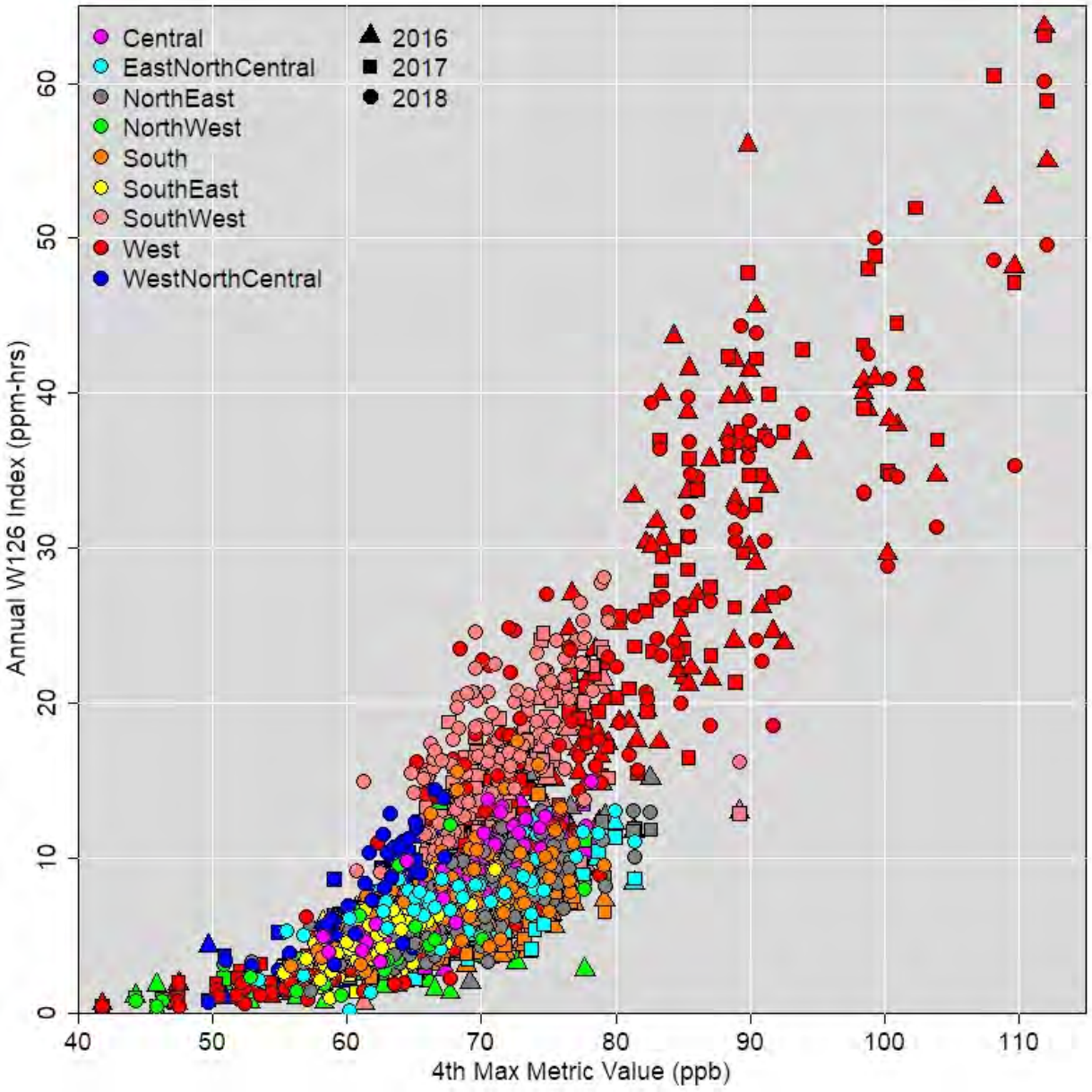

Figure 4D-5. Scatter plot of annual W126 index values versus 4th max metric values (design values) based on 2016-2018 monitoring data.

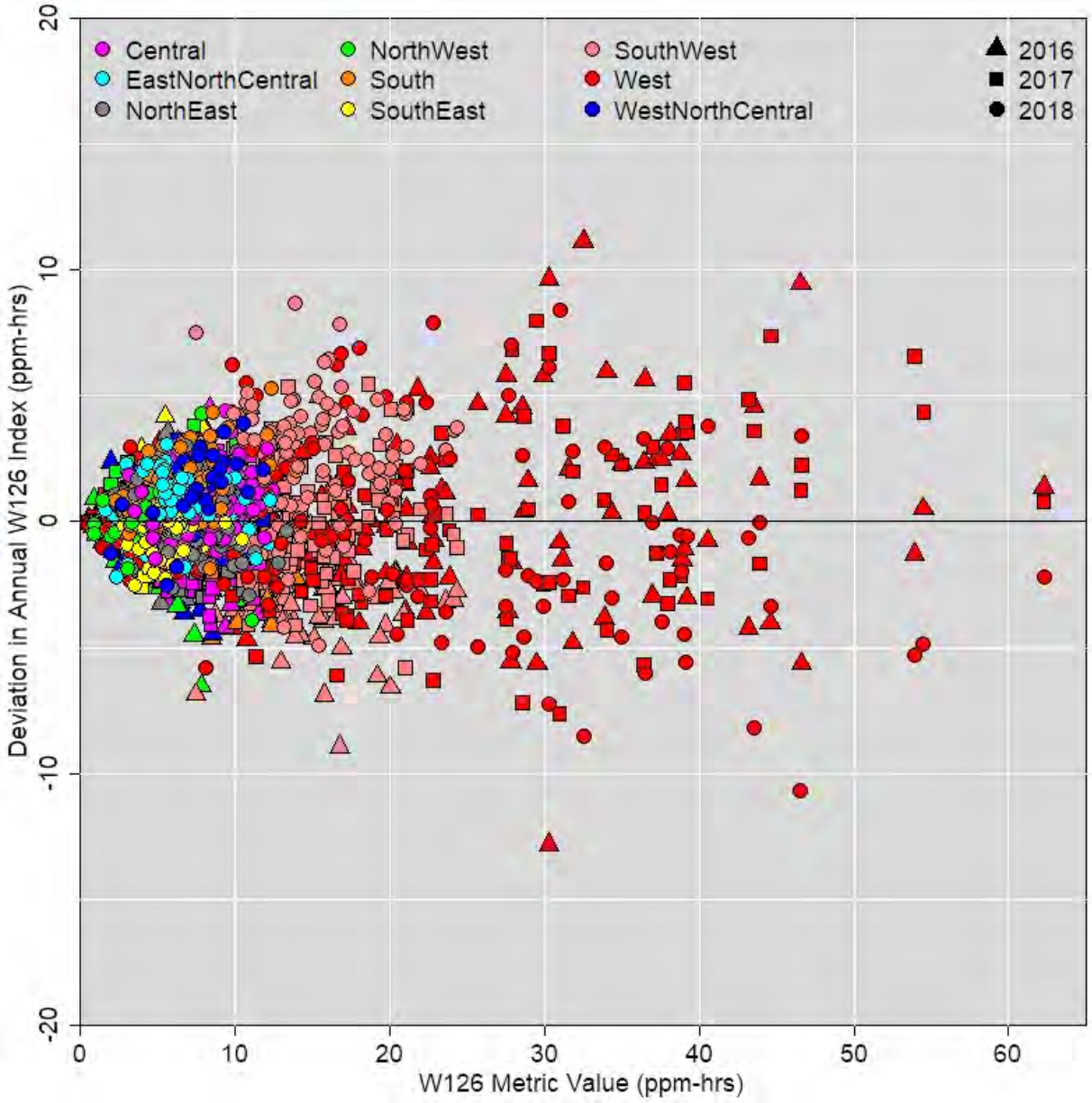

Figure 4D-6. Deviation in annual W126 index values from their respective 3-year averages for all U.S. monitoring sites in 2016-2018.

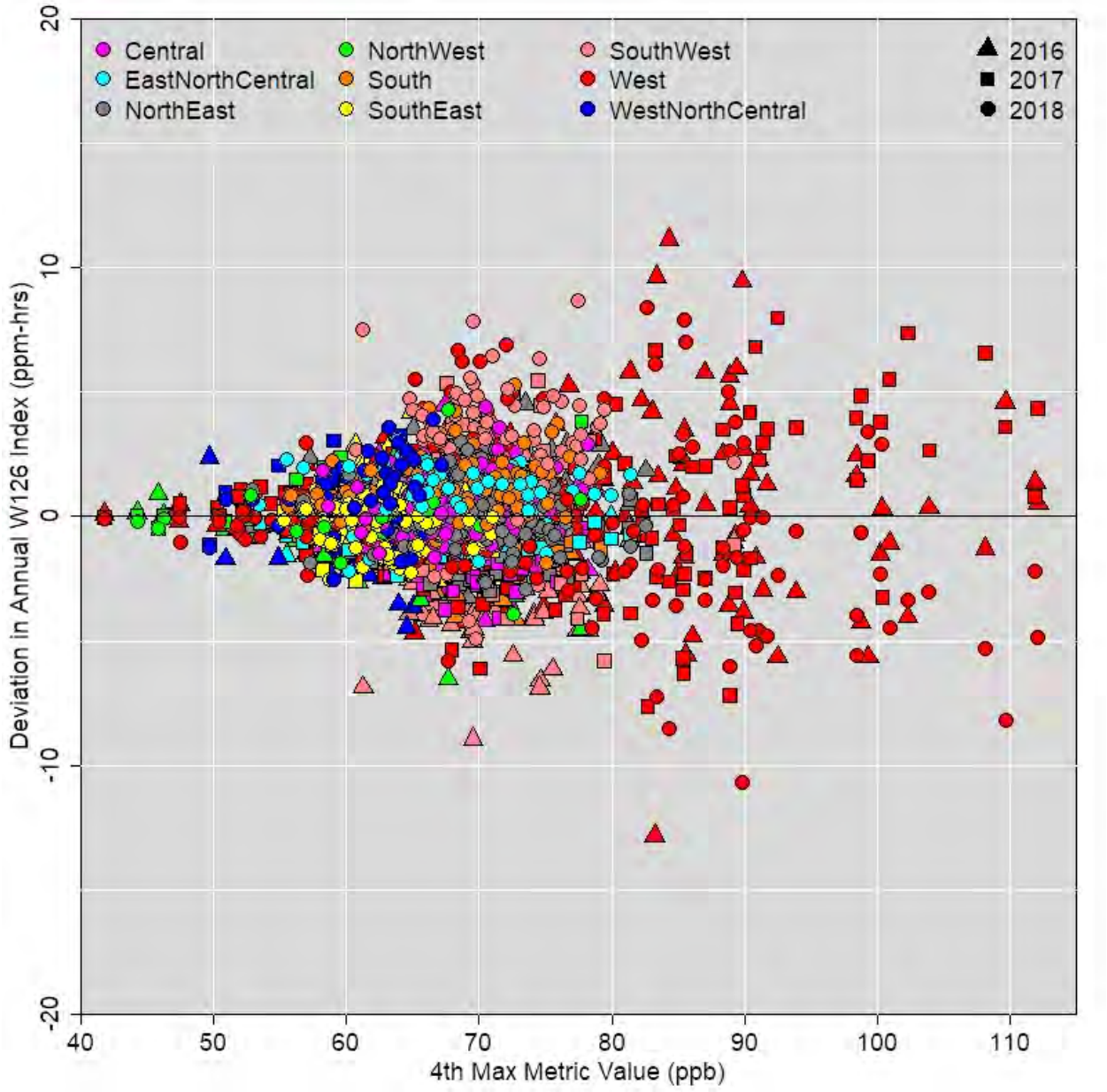

Figure 4D-7. Deviation in annual W126 index values from their respective 3-year averages (y-axis) compared to their 4th max metric values (x-axis) in 2016-2018.

4D.3.2 National Analysis Using Historical Air Quality Data

This section presents various results based on the 4th max and W126 metrics for the full 19-year period spanning years 2000 to 2018. Comparisons similar to those shown in section 4D.3.1 are shown in section 4D.3.2.1, trends in W126 are shown in section 4D.3.2.2, and several comparisons of the trends in the 4th max and W126 metrics are shown in section 4D.3.2.3.

4D.3.2.1 Comparison of the 4th Max and W126 Metrics

Table 4D-7 to Table 4D-11 present similar information to Table 4D-2 to Table 4D-6, respectively, except that the values shown in each cell contain the number of occurrences summed over all 17 consecutive 3-year periods (2000-2002 to 2016-2018) instead of just the 2016-2018 period. For example, Table 4D-10 shows that over all 17 consecutive 3-year periods, there were 243 occurrences where sites had 4th max metric values less than or equal to 70 ppb and W126 metric values greater than 13 ppm-hrs. In general, the relative magnitudes of the numbers shown in Table 4D-7 to Table 4D-11 compare well to their respective counterparts in Table 4D-2 to Table 4D-6. According to Table 4D-10, there have been no occurrences over the entire 19-year period where a site has had a 4th max metric value less than or equal to 70 ppb and a W126 metric value greater than 19 ppm-hrs.[5]

Figure 4D-8 shows the distribution of annual W126 index values observed at sites during 3-year periods with different 4th max metric values. These distributions are illustrated by box-and-whisker plots with boxes showing the 25th, 50th, and 75th percentile of the annual W126 index values occurring with 4th max metric values within each bin, whiskers extending to the 1st and 99th percentiles of the annual W126 index values, and points occurring outside the 1st and 99th percentiles represented by dots. This figure shows that for the bin with the highest 4th max metric values meeting the current standard, 66-70 ppb, the 99th percentile of the annual W126 index values was about 19 ppm-hours, or in other words, for sites meeting the current standard, annual W126 index values were less than or equal to 19 ppm-hrs well over 99% of the time.

[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.

Table 4D-7. Total number of 4th max metric values greater than various 4th max levels based on all 17 consecutive 3-year periods (2000-2002 to 2016-2018).

4th Max Level (ppb)	75	70	65
Values > Level	6,671	10,695	14,942
Values ≤ Level	12,060	8,292	4,396
Total # of Values[A]	18,731	18,987	19,338

[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.

Table 4D-8. Total number of W126 metric values greater than various W126 levels based on all 17 consecutive 3-year periods (2000-2002 to 2016-2018).

W126 Level (ppm-hrs)	19	17	15	13	11	9	7
Values > Level	2,317	3,186	4,397	6,027	7,974	10,347	12,952
Values ≤ Level	16,244	15,414	14,244	12,683	10,840	8,586	6,153
Total # of Values[A]	18,561	18,600	18,641	18,710	18,824	18,933	19,105

[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.

Table 4D-9. Total number of 4th max metric values greater than various 4th max levels and W126 metric values less than or equal to various W126 levels based on all 17 consecutive 3-year periods (2000-2002 to 2016-2018).

Values > 4th Max Level AND ≤ W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4th Max Level (ppb)	75	4,227	3,554	2,680	1,679	832	267	40
	70	7,920	7,099	5,980	4,609	3,062	1,414	375
	65	11,822	10,992	9,822	8,261	6,433	4,292	2,079

Table 4D-10. Total number of 4th max metric values less than or equal to various 4th max levels and W126 metric values greater than various W126 levels based on all 17 consecutive 3-year periods (2000-2002 to 2016-2018).

Values ≤ 4th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4th Max Level (ppb)	75	84	236	530	1,085	2,076	3,748	5,947
	70	0	8	58	243	536	1,135	2,529
	65	0	0	0	0	15	126	343

Table 4D-11. Total number of 4th max metric values greater than various 4th max levels and W126 metric values greater than various W126 levels based on all 17 consecutive 3-year periods (2000-2002 to 2016-2018).

Values > 4th Max Level AND > W126 Level		W126 Level (ppm-hrs)						
		19	17	15	13	11	9	7
4th Max Level (ppb)	75	2,223	2,923	3,820	4,855	5,747	6,341	6,583
	70	2,317	3,174	4,328	5,747	7,369	9,088	10,193
	65	2,317	3,186	4,397	6,027	7,957	10,201	12,556

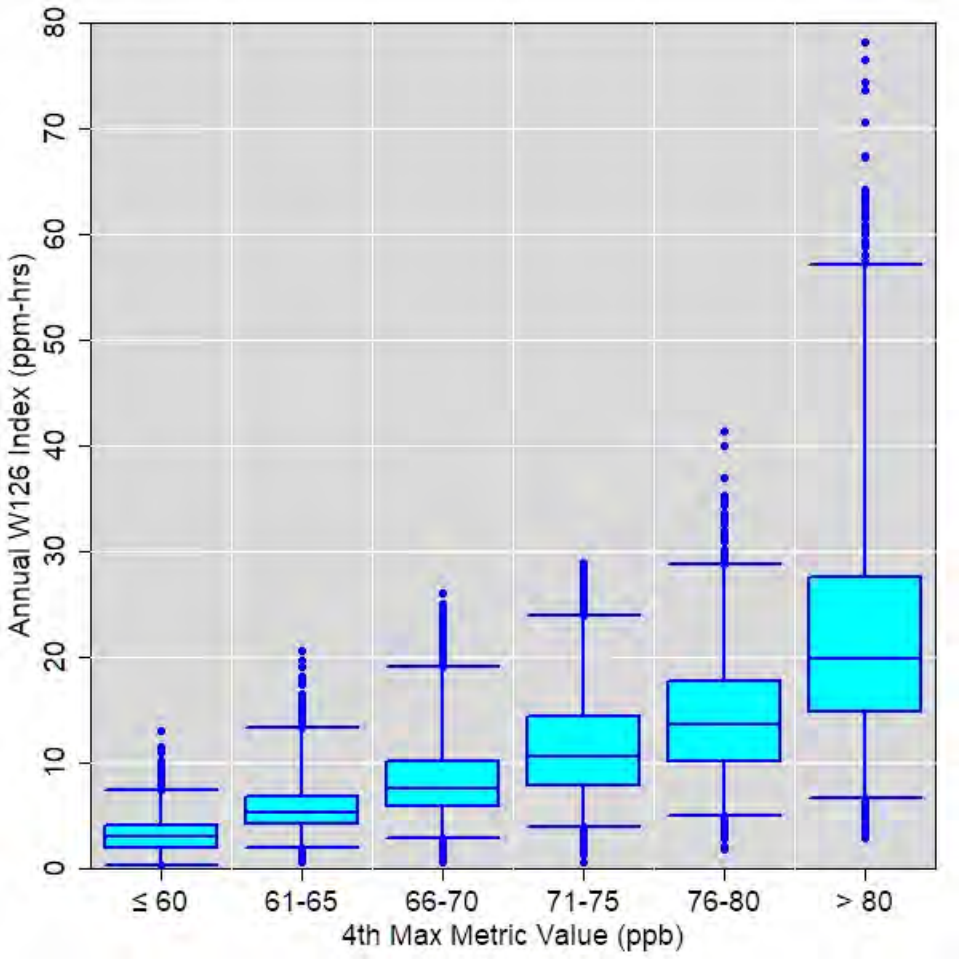

Figure 4D-8. Annual W126 index values in ppm-hrs binned by 4th max metric values based on monitoring data for years 2000-2018. Boxes show 25th, 50th, and 75th percentiles, whiskers extend to the 1st and 99th percentiles, and points below the 1st percentile or above the 99th percentile are represented by dots.

4D.3.2.2 Trends in W126 Metric

Figure 4D-9 below shows national trends in both the annual W126 index and the 3-year W126 metric based on the monitoring sites reporting data for the full period. Most notably, the

figure shows decreasing trends in W126 metric values, with the median value decreasing by about 60% from 2002 to 2018. The annual W126 index shows considerable year-to-year variability, with the median value sometimes increasing or decreasing by up to a factor of two from one year to the next, while the 3-year average is less impacted by this inter-annual variability, resulting in a smoother trend line.

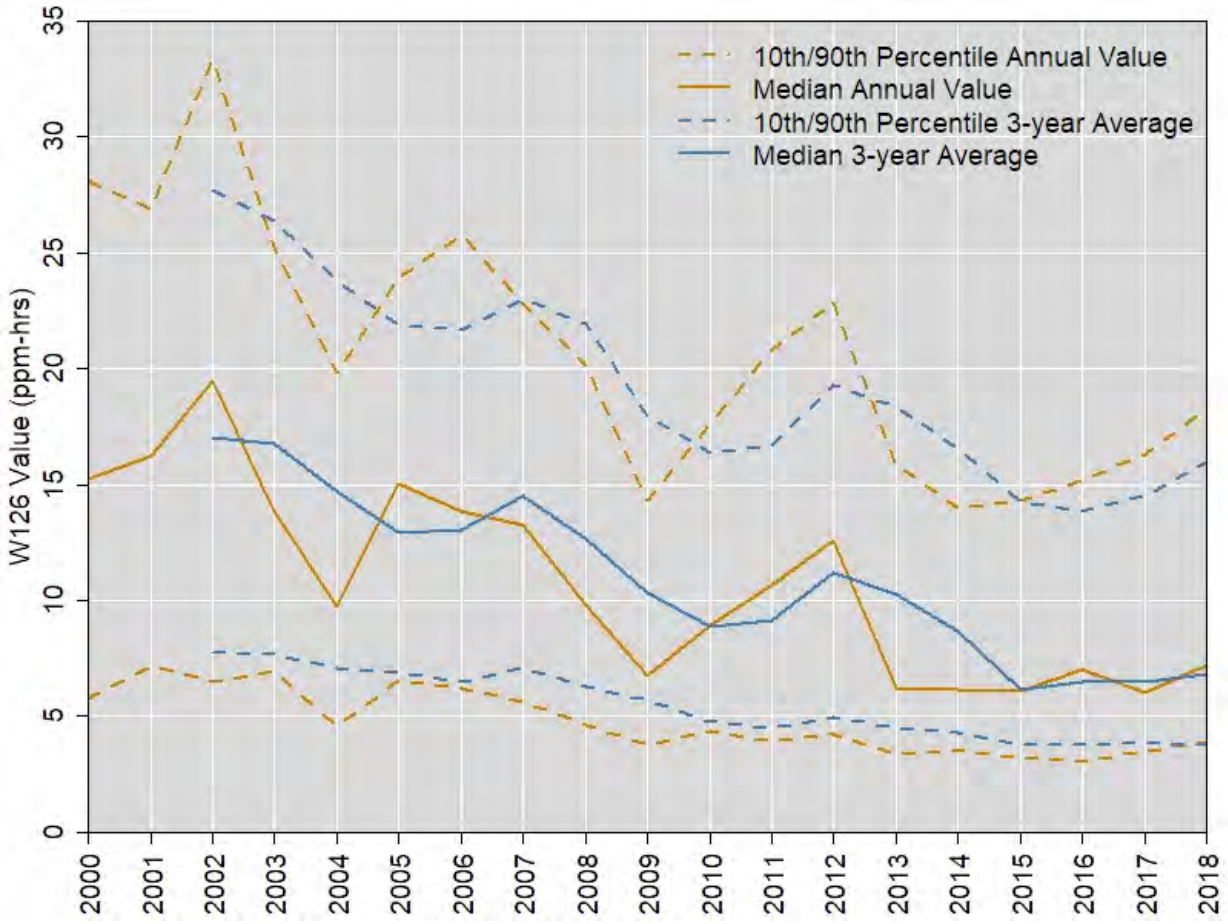

Figure 4D-9. National trends in annual W126 index values (2000-2018) and W126 metric values (2002-2018).

Figure 4D-10 shows a map of the site-level trends in the W126 metric values from 2000-2002 to 2016-2018. According to Figure 4D-10, nearly 90% of U.S. monitoring sites experienced significant decreases in W126 over this period, especially in the eastern U.S. and California where many O_3 monitoring sites saw decreases of 1 ppm-hr/yr or more. Many locations in the western U.S. experienced little or no change over this period. Only seven monitors in disparate locations showed significant increasing trends in the W126 metric during the 2002-2018 period.

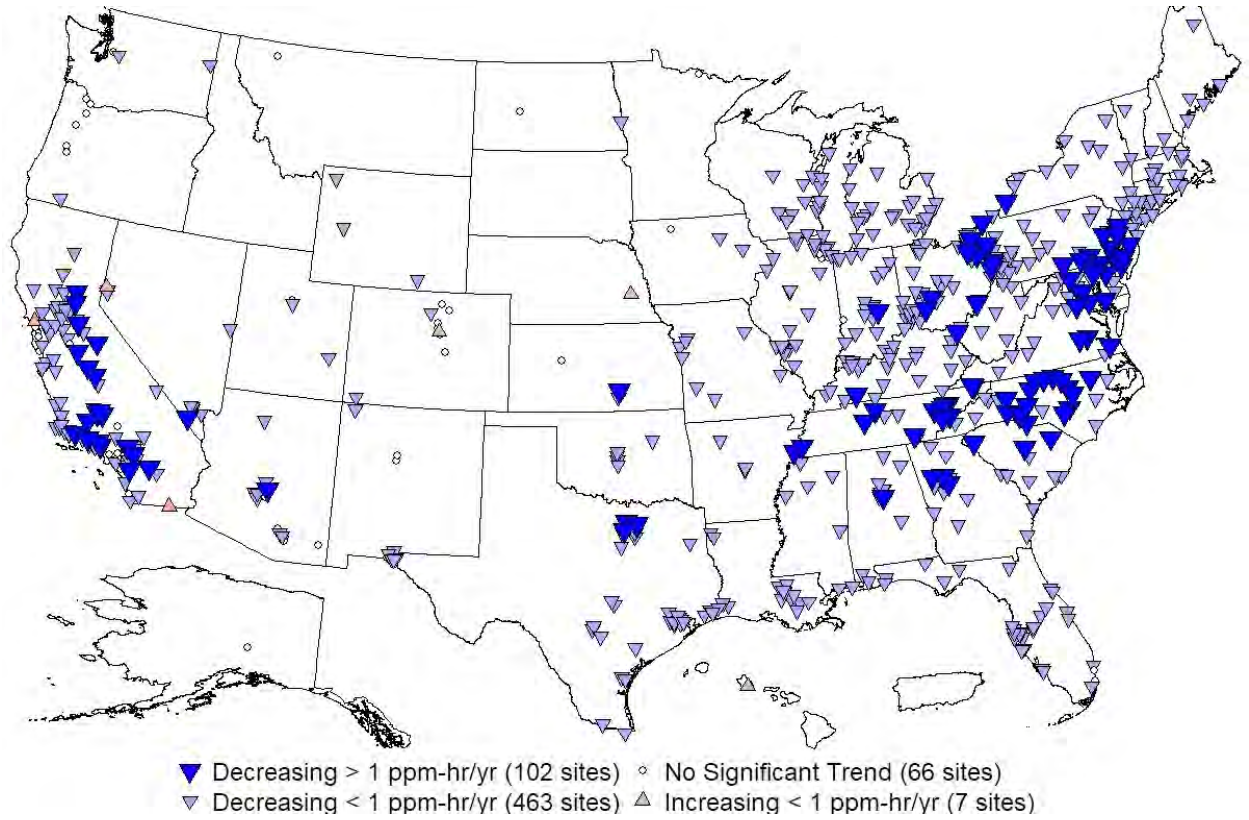

Figure 4D-10. Map of trends in W126 metric values at U.S. O_3 monitoring sites from 2000-2002 to 2016-2018.

4D.3.2.3 Comparison of Trends in the 4th Max and W126 Metrics

Figure 4D-11 shows a scatter plot comparing the trends in the 4th max metric values (x-axis, ppb/yr) to the trends in the W126 metric values (y-axis, ppm-hr/yr). These trends are calculated using the Thiel-Sen estimator as in Figure 4D-10. The relationship between the trends in the two metrics was linear and positive (Pearson correlation coefficient R = 0.82), meaning a decrease in the 4th max metric is usually accompanied by a decrease in the W126 metric. The slope of the regression line shown in Table 4D-12 indicates that, on average, there was a change of approximately 0.62 ppm-hr in the W126 metric values per unit ppb change in the 4th max metric values.

Figure 4D-12 shows scatter plots comparing the trends in the 4th max metric values (x-axis, ppb/yr) to the trends in the W126 metric values (y-axis, ppm-hr/yr) in each NOAA climate region and the associated regression lines fit using the sites within each region. Table 4D-12 provides some summary statistics based on the regional trends comparisons. Figure 4D-12 and Table 4D-12 show that the positive, linear relationship between the trends in the 4th max metric values and the trends in the W126 metric values persists within each region, with Pearson correlation coefficients ranging from 0.53 to 0.94. The regression lines shown in Figure 4D-12

with slopes listed in Table 4D-12 indicate that the Southwest region, which had the greatest potential for sites having higher W126 metric values relative to their 4th max metric values, also exhibited the greatest response in the W126 metric values per unit change in the 4th max metric values. In Figure 4D-11 and Figure 4D-13 (as well as the West region panels in Figure 4D-12 and Figure 4D-14), there appear to be three sites in the West region with an increasing trend in the W126 metric (slope > 0) and a decreasing trend in the 4th max metric (slope < -0.5). These three sites are all located downwind of Los Angeles, CA and generally have 4th max metric values of 100 ppb or greater, along with W126 metric values in the 30-50 ppm-hr range.

Figure 4D-13, Figure 4D-14 and Table 4D-13 present information similar to that shown in Figure 4D-11, Figure 4D-12 and Table 4D-12, respectively, except that trends in annual W126 index values are presented instead of W126 metric values. The figures show that the same general pattern occurs when comparing annual W126 index values to the 4th max metric values as was seen for the W126 metric values. There is slightly more variability in the relationship, as can be seen from the slight increase in scatter in the figures and the slightly lower correlation values shown in Table 4D-12 as compared to Table 4D-11.

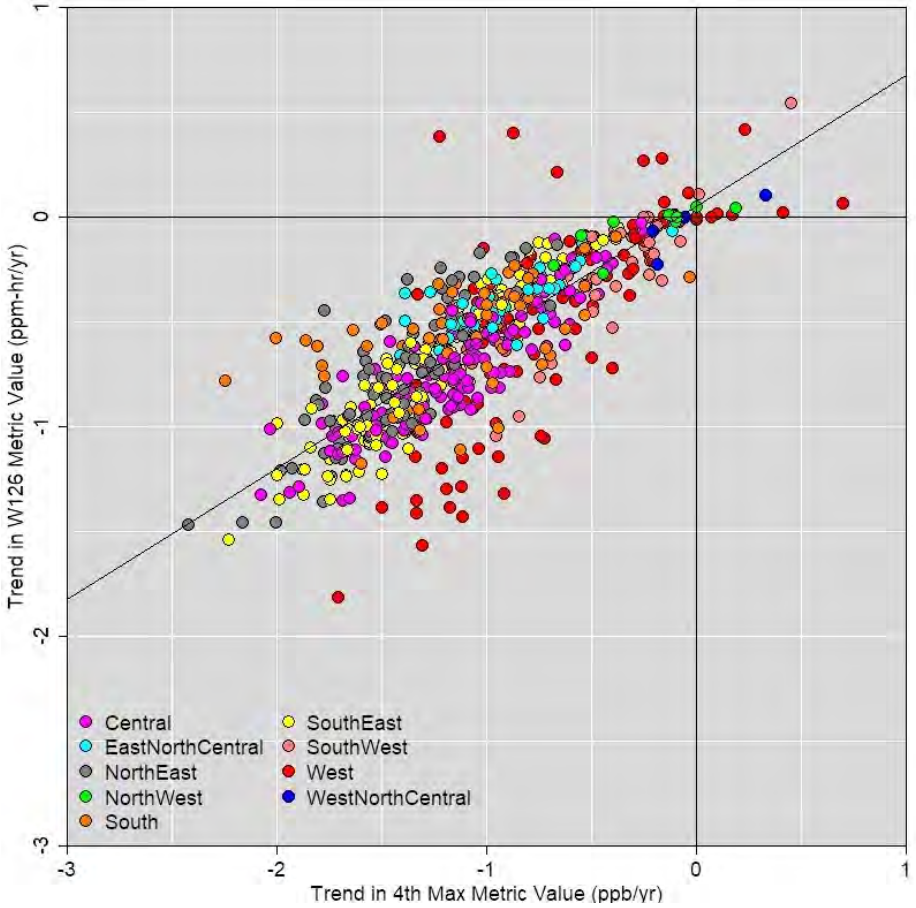

Figure 4D-11. Scatter plot comparing trends in 4th max metric values (x-axis) to trends in W126 metric values (y-axis).

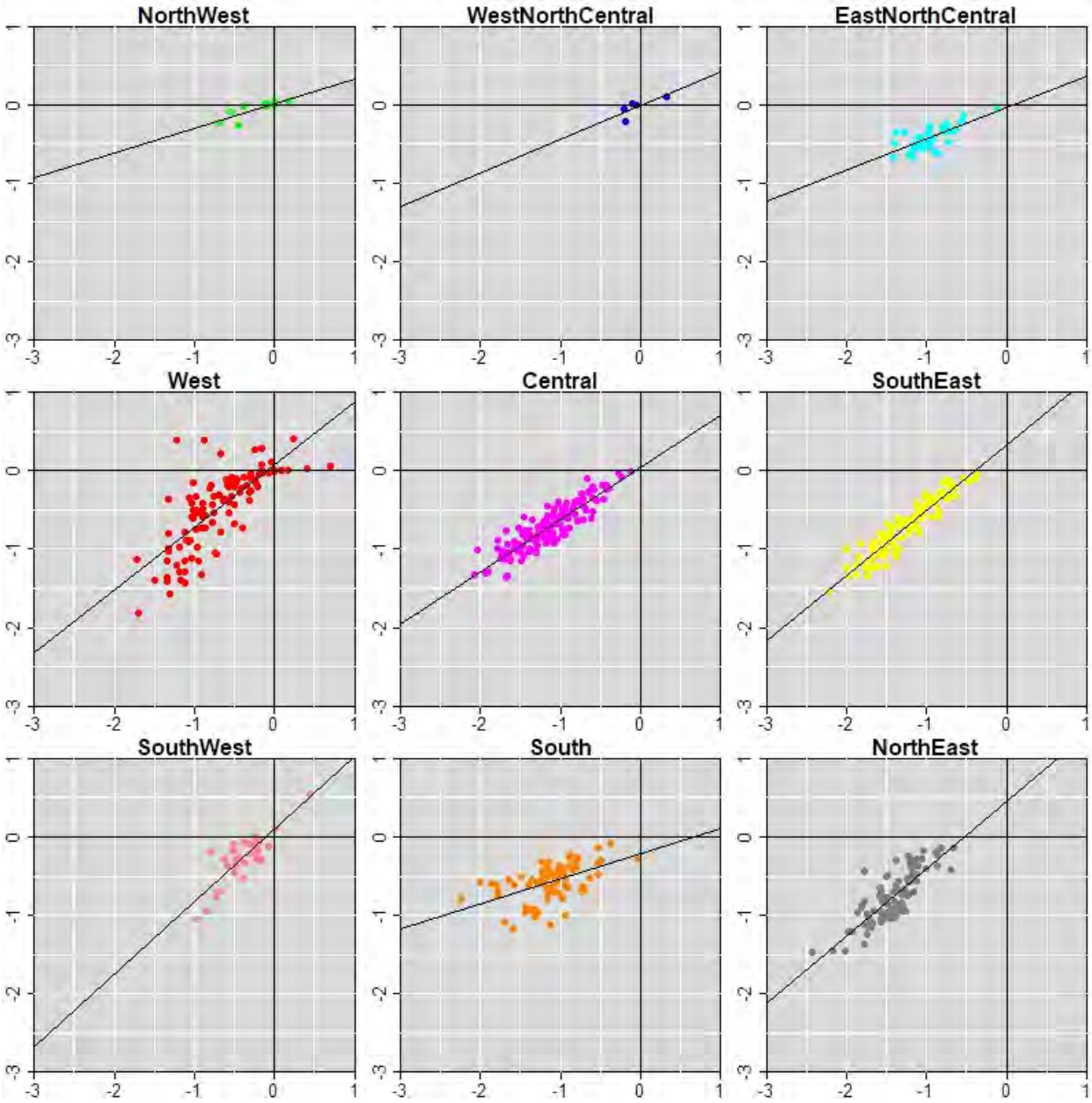

Figure 4D-12. Scatter plots comparing the trends in 4th max metric values (x-axis, ppb) and W126 metric values (y-axis, ppm-hrs) based on O_3 monitoring sites within each of the nine NOAA climate regions.

Table 4D-12. Summary statistics based on regional comparisons of trends in 4th max metric 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	152	-1.16	-0.73	0.66	0.89
East North Central	43	-0.95	-0.41	0.40	0.73
Northeast	98	-1.41	-0.76	0.86	0.83
Northwest	11	-0.20	-0.06	0.32	0.81
South	73	-1.11	-0.57	0.32	0.53
Southeast	105	-1.29	-0.73	0.83	0.94
Southwest	36	-0.37	-0.24	0.93	0.83
West	105	-0.70	-0.49	0.80	0.75
West North Central	6	-0.08	-0.18	0.43	0.83
National	629	-1.05	-0.61	0.62	0.82

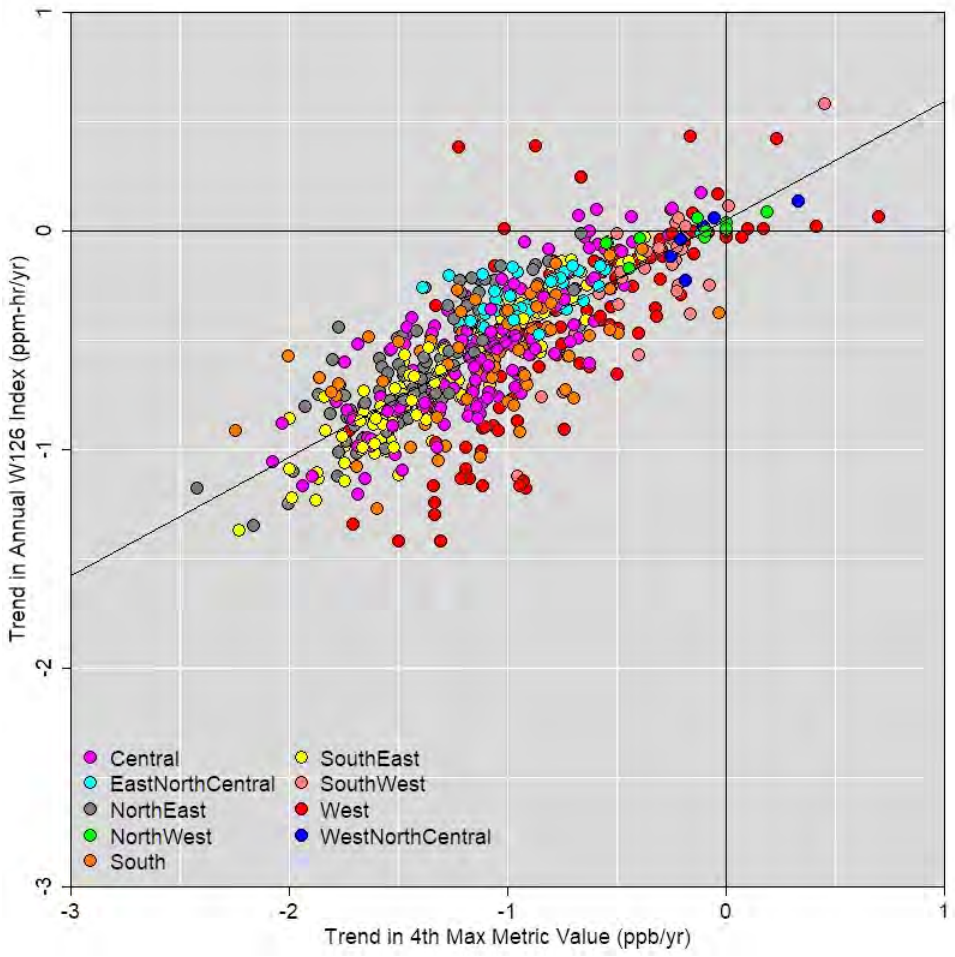

Figure 4D-13. Scatter plot comparing trends in 4th max metric values (x-axis) and trends in annual W126 index values (y-axis).

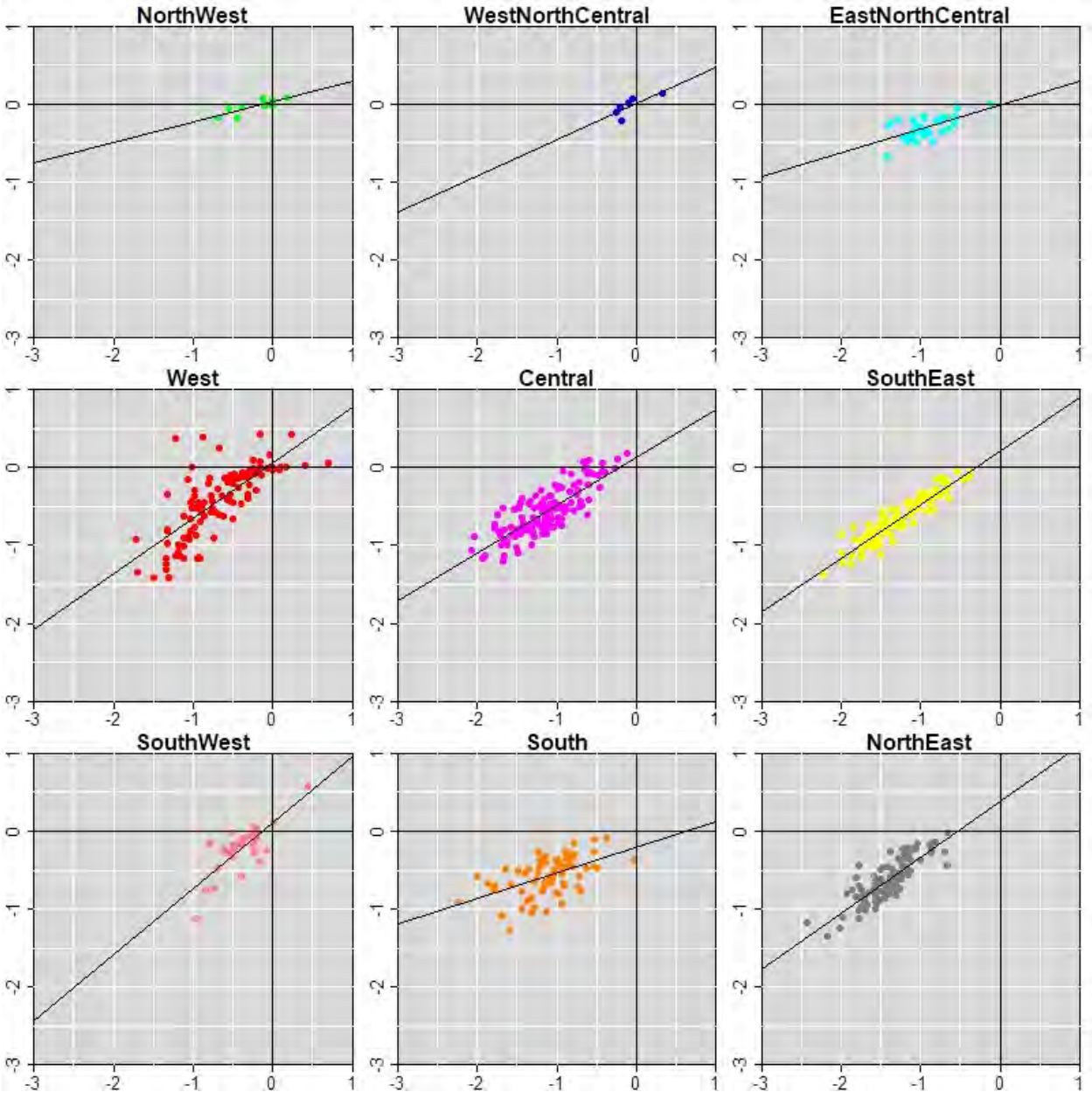

Figure 4D-14. Scatter plots comparing trends in 4th max metric values (x-axis, ppb) to trends in annual W126 index values (y-axis, ppm-hrs) based on O_3 monitoring sites within each of the nine NOAA climate regions.

Table 4D-13. Summary statistics based on regional comparisons of trends in 4th max metric values and trends in annual W126 index values.

NOAA Climate Region	Number of O_3 Sites	Mean Trend in 4th Max Metric Value (ppb/yr)	Mean Trend in Annual W126 Index Value (ppm-hr/yr)	Regression Slope	Pearson Correlation Coefficient
Central	152	-1.16	-0.59	0.61	0.80
East North Central	43	-0.95	-0.29	0.30	0.63
Northeast	98	-1.41	-0.62	0.72	0.83
Northwest	11	-0.20	-0.06	0.26	0.84
South	73	-1.11	-0.57	0.33	0.53
Southeast	105	-1.29	-0.64	0.69	0.93
Southwest	36	-0.37	-0.22	0.85	0.78
West	105	-0.70	-0.45	0.71	0.74
West North Central	6	-0.08	-0.11	0.46	0.77
National	629	-1.05	-0.52	0.54	0.79

4D.3.2.4 W126 Metric Values in Federal Class I Areas

Table 4D-14 below lists the 65 federal Class I areas for which we have monitoring data available for at least one 3-year period within the 2000-2018 period from a monitor located either within the area boundaries or within 15 km of the boundary. This summary table indicates the number of three-year periods for which the two metrics are available, the number of periods where 4th max metric values were at or below 70 ppb and the range of the W126 metric values during those periods. In total, the table is summarizing the 860 combinations of Class I area and 3-year period of which 498 have a 4th max metric value at or below 70 ppb and 362 have a 4th max metric value above 70 ppb. In the most recent period (2016-2018), of the 58 areas for which we have monitors, 47 sites have 4th max metric values at or below 70 ppb.

Table 4D-15 lists the Class I areas with the highest W126 metric values when the 4th max metric value is at or below 70 ppb. Among areas with a 4th max metric value at or below 70 ppb during any of the 3-year periods from 2000 to 2018, five areas (all located in the Southwest region) had one or more W126 metric values above 17 ppm-hrs, with the highest W126 metric values equal to 19 ppm-hrs and the highest annual W126 index values equal to 23 ppm-hrs, when rounded. All seven instances where a Class I area observed a 4th max metric value at or below 70 ppb and a W126 metric value above 17 ppm-hrs occurred prior to 2011. This contrasts with the much higher values observed in Class I areas when the current standard is not met (Table 4D-17). In the 2016-2018 period, the W126 metric values range up to 47 ppm-hrs at sites in Class I areas when the standard is not met, with higher values in the historical Class I dataset.

Figure 4D-15 shows the distribution of annual W126 index values in Class I areas during 3-year periods with different 4th max metric values. The full distribution of annual W126 index values, including the minimum and maximums, increase with increasing 4th max metric values.

For example, the 99th percentile increases from about 20 ppm-hrs or lower to higher than 25 ppm-hrs for 4th max metric values at and below 70 ppb compared to 4th max metric values above 70 ppb. As indicated by Table 4D-15, the 3-year periods with the highest W126 metric values occurring for 4th max metric values at or below 70 ppb occurred in the earlier years of the dataset (2000-2010).

Table 4D-16 summarizes the occurrence of relatively higher annual W126 index values in Class I areas during 3-year periods when the 4th max metric value is at or below 70 ppb. This figure summarizes the W126 metric (i.e., 3-year average of annual W126 index values), as well as maximum annual W126 index values in each 3-year period meeting the current standard. For all instances of an area and 3-year period with a maximum annual W126 index value above 19 ppm-hrs, Figure 4D-16 illustrates the variation in among the annual W126 index values and the extent to which they differ from the 3-year average.

Finally, Table 4D-17 further documents the ranges of W126 metric values occurring during periods when the 4th max metric value was above 70 ppb, indicating the extent to which the current standard appears to be controlling the W126 metric.

Table 4D-14. W126 metric values in Class I areas with 4th max metric values at or below 70 ppb (2000-2018).

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 4th max ≤ 70 ppb	Range of W126 Metric Values when 4th max ≤ 70 ppb
Central (7, 4)	Kentucky	Mammoth Cave National Park RM, VP	17	7	5-10
	Tennessee	Great Smoky Mountains National Park[3] SM, YP, LP, VP, RM, BC, WP	17	6	7-10
	West Virginia	Otter Creek Wilderness VP, YP, RM, SM, BC, LP, WP	16	12	5-8
EastNorthCentral (6, 3)	Michigan	Seney Wilderness Area* QA, RM, SM, BC, WP	15	7	4-6
	Minnesota	Boundary Waters Canoe Area Wilderness Area* SM, QA, WP	6	6	2-4
		Voyageurs National Park QA, RM, WP	13	13	2-6
NorthEast (6, 4)	Maine	Acadia National Park RM, QA, SM, WP	17	6	4-5
	New Hampshire	Great Gulf Wilderness Area* WP	14	14	3-8
	New Jersey	Brigantine Wilderness Area* BC	16	5	6-8
NorthWest (29, 4)	Idaho	Craters of the Moon Wilderness Area* DF, QA	11	11	6-13
	Washington	Alpine Lakes Wilderness* DF, PP	17	15	2-6
		Mount Rainer National Park, DF	15	14	2-6
		North Cascades National Park* PP, DF, RA	3	3	1-2
		Olympic National Park DF, RA	7	7	1-2
	Alaska	Denali National Park QA (Formerly Mt. McKinley Nat Pk)	17	17	2-4
South (6, 4)	Arkansas	Caney Creek Wilderness Area*	12	6	4-7
		Upper Buffalo Wilderness Area* SM	17	11	3-8
	Texas	Big Bend National Park QA, DF, PP	16	15	7-13
SouthEast (16, 6)	Alabama	Sipsey Wilderness* WP, RM, SM, YP, LP, VP	6	1	11
	Florida	St. Marks Wilderness Area*	15	10	4-11
	Georgia	Cohutta Wilderness Area* WP, VP, YP	17	12	5-6
	North Carolina	Great Smoky Mountains National Park* SM, YP, LP, VP, RM, BC, WP	See Tennessee for monitor with highest design value (4th max)		
		Linville Gorge Wilderness Area* VP, WP, RM, YP	17	13	5-11
		Shining Rock Wilderness Area*	15	8	6-10
	South Carolina	Cape Romain Wilderness*	17	10	3-9
	Virginia	James River Face Wilderness* WP	17	13	3-8
		Shenandoah National Park WP, VP, QA, BC, RM, SM, YP	17	6	6-11
SouthWest (38. 4)	Arizona	Chiricahua National Monument DF, PP	17	9	13-17
		Grand Canyon National Park DF, PP, QA	17	9	11-19
		Mazatzal Wilderness Area DF, PP	17	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 4th max ≤ 70 ppb	Range of W126 Metric Values when 4th max ≤ 70 ppb
		Petrified Forest National Park	9	9	11-17
		Saguaro Wilderness Area* [2] DF, PP	17	5	13-15
		Superstition Wilderness Area* PP	17	0	-
		Yavapai Reservation* QA, PP, DF	2	2	14-15
	Colorado	Maroon Bells-Snowmass Wilderness. Area* QA, DF	12	12	11-19
		Mesa Verde National Park* PP, DF	17	15	12-18
		Rocky Mountain National Park* DF, PP, QA	17	4	13-15
		Weminuche Wilderness Area* DF, PP	8	3	13-18
	New Mexico	San Pedro Parks Wilderness* PP, DF	3	3	10-18
	Utah	Canyonlands National Park PP, DF	16	11	10-17
		Zion National Park* DF, PP, QA	11	6	11-18
West (32, 3)	California	Agua Tibia Wilderness* DF	4	0	-
		Cucamonga Wilderness Area* DF, PP	17	0	-
		Desolation Wilderness Area* PP	8		8-13
		Joshua Tree Wilderness Area*	17	0	-
		Kaiser Wilderness Area*	1	0	-
		Lassen Volcanic National Park DF, PP	17	13	7-14
		Pinnacles Wilderness Area*	17	8	8-10
		San Gabriel Wilderness Area* DF, PP	17	0	-
		San Gorgonio Wilderness Area* PP, QA	13	0	-
		San Jacinto Wilderness Area* PP	17	0	-
		San Rafael Wilderness Area*	17	9	5-9
		Sequoia National Park PP, QA, DF	17	0	-
		Ventana Wilderness Area*	17	17	2-4
		Yosemite National Park DF, PP, QA	17	0	-
	Hawaii	Hawaii Volcanoes National Park	2	2	0
WestNorthCentral (26, 4)	Montana	Gates of the Mountain Wilderness Area*	6	6	3-5
		Glacier National Park QA, PP, DF	17	17	2-3
		Northern Cheyenne Reservation*	7	7	3-5
	North Dakota	Lostwood Wilderness*	13	13	4-5
		Theodore Roosevelt National Park[2] PP	17	17	5-7
	South Dakota	Badlands Wilderness*	11	11	3-12
		Wind Cave National Park PP	10	10	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 4th max ≤ 70 ppb	Range of W126 Metric Values when 4th max ≤ 70 ppb
	Wyoming	Bridger Wilderness*	13	12	9-16
		Grand Teton National Park DF, QA	5	5	5-8
		Yellowstone National Park DF, QA	17	17	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: QA=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.

Table 4D-15. Highest W126 metric values occurring in Class I areas when the 4th max metric value is at or below 70 ppb (2000-2018).

Class I Area	State/County	4th 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.

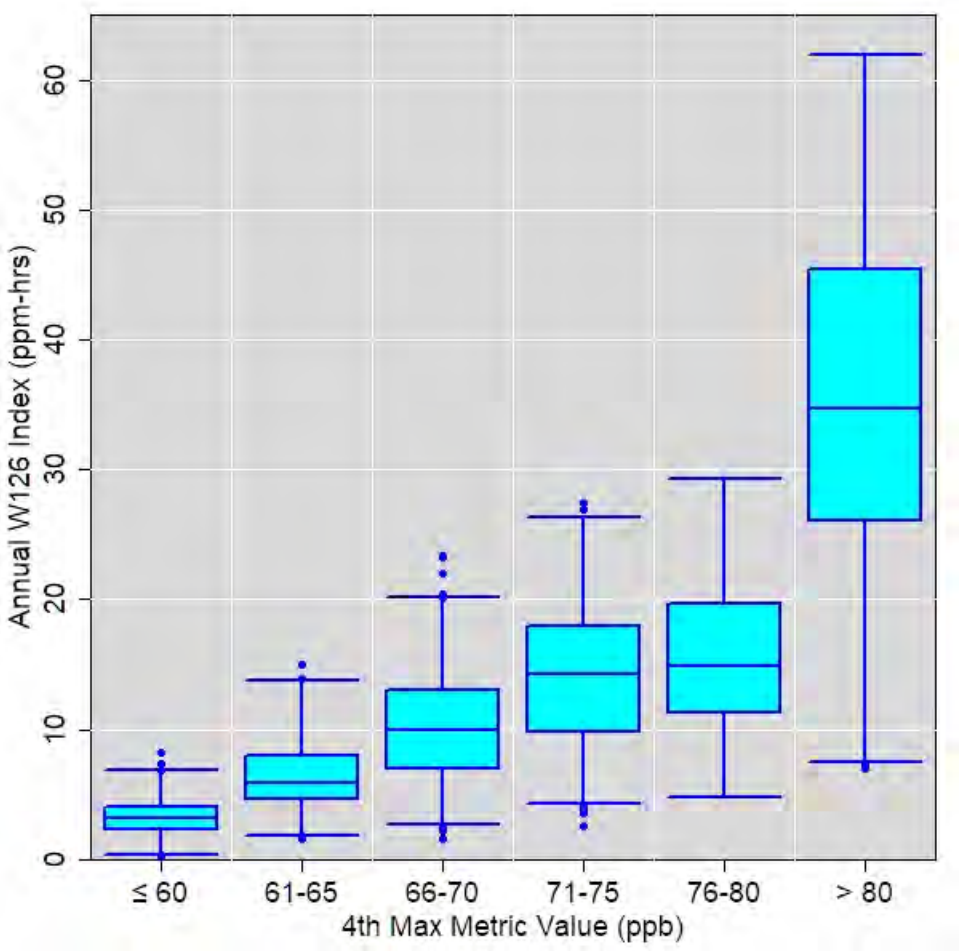

Figure 4D-15. Range of annual W126 index values in ppm-hrs observed at monitoring sites in Class I areas based on 2000-2018 monitoring data. Values are binned according to 4th max metric values in ppb. Boxes show 25th, 50th, and 75th percentiles, whiskers extend to 1st and 99th percentiles, and points below the 1st percentile or above the 99th percentile are represented by dots.

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
2016-2018	58 (58)	0	0	47 (47)	0	3 (3) [A]	44 (44)
2000-2018	860 (58)	0	7 (5) [A]	492 (56)	15 (10) [B]	39 (19) [C]	460 (56)

[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.

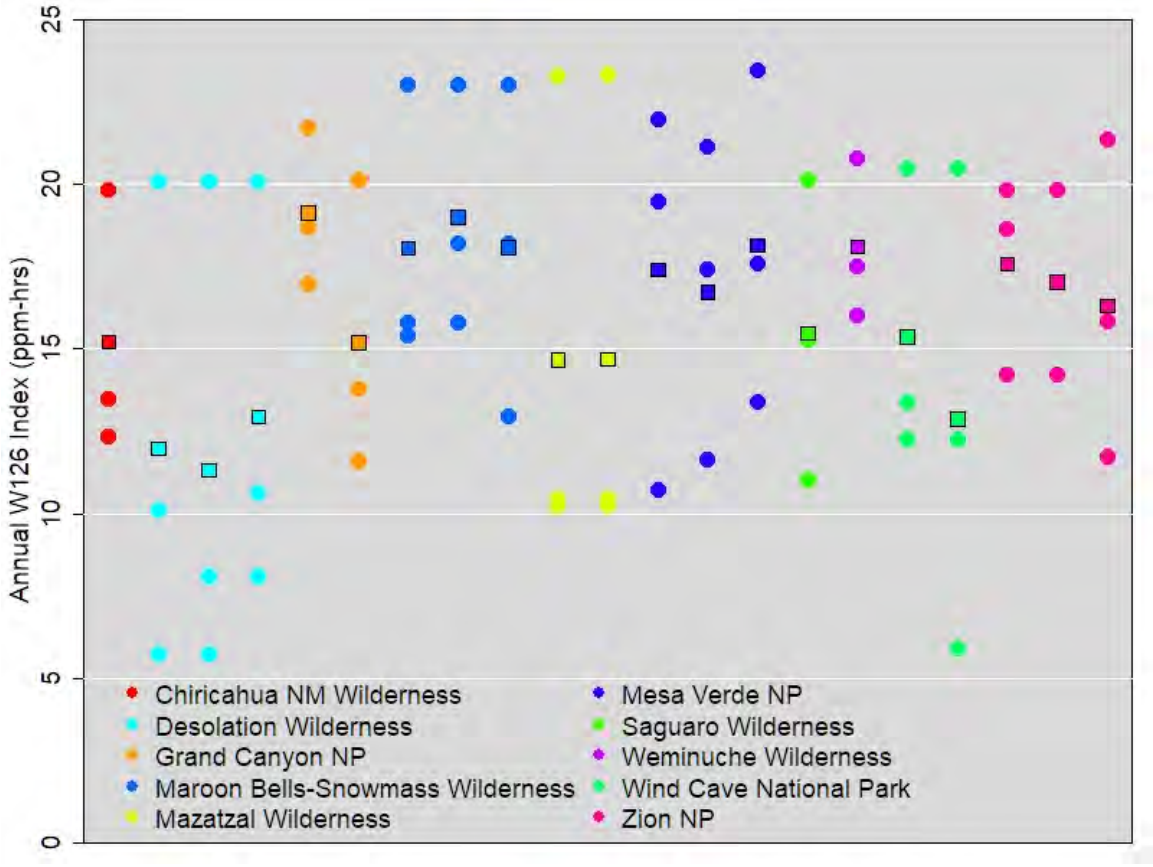

Figure 4D-16. Range of annual W126 index values observed in each 3-year period where a site in a Class I area had a design value meeting the current standard and had at least one annual W126 index value greater than 19 ppm-hrs. Dots show annual W126 index values and squares show the W126 metric value.

Table 4D-17. W126 values in Class I areas with 4th max metric values above 70 ppb (2000-2018).

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)
	2016-2018											
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	-	-	-	1	21-	19-26	3	16-19	12-21	0	-	-
West	8	21-47	17-56	8	21-47	17-56	8	21-47	17-56	0	-	-
WestNorthCentral	0	-	-	0	-	-	0	-	-	0	-	-
	2000-2018											
Central	1	20-31	9-37	2	18-31	9-37	2	16-31	9-37	3	9-15	6-22
EastNorthCentral	0	-	-	0	-	-	0	-	-	1	6-8	4-11
NorthEast	1	20	18-24	1	20	18-24	1	17-20	9-24	2	5-14	4-18
NorthWest	0	-	-	0	-	-	0	-	-	2	6-7	4-10
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	10-39	10	18-33	10-39	10	16-37	10-34	5	11-15	7-24
West	12	20-61	16-74	13	18-61	12-74	13	16-61	12-74	4	10-15	8-20
WestNorthCentral	0	-	-	0	-	-	1	17	14-19	0	-	-

4D.4 KEY LIMITATIONS AND UNCERTAINTIES

This section summarizes key limitations and uncertainties associated with aspects of the datasets analyzed in the preceding sections. The first section summarizes key limitations and uncertainties associated with complete dataset monitoring sites in all U.S. locations (urban and rural), which focus on patterns and relationships across all monitoring sites. The second section concentrates on the Class I area sites. Overall, we recognize that while the datasets analyzed are quite extensive (e.g., more than 1,100 sites covering all 50 states in the most recent 3-year period), there are limitations and uncertainties associated with the spatial representation of O_3 monitoring sites in rural areas and Class I areas, specifically.

Analyses of data for all U.S. monitoring sites: Given that there has been a longstanding emphasis on urban areas in the EPA's monitoring regulations, urban areas are generally well represented in the U.S. dataset, with the effect being that the current dataset is more representative of locations where people live than of complete spatial coverage for all areas in the U.S., (i.e., the current dataset is more population weighted than geographically weighted). As O_3 precursor sources are also generally more associated with urban areas, one impact of this may be a greater representation of relatively higher concentration sites. One method that has been suggested to create a more geographically representative dataset is the use of photochemical air quality modeling to estimate concentrations. However, this approach has been found to present its own uncertainties with regard to estimating annual W126 index values (U.S. EPA, 2014b, Appendix 4A), making it less useful for the current analyses.

Dataset for Class I monitoring sites: A limitation of this dataset is that it includes sites in only 65 of the 164 Class I areas in the U.S. The representation of states containing Class I areas is somewhat greater, with monitoring sites in Class I areas in 29 of the 36 states that have such an area. All nine NOAA climate regions are represented. As can be seen from Figure 4D-2, sites outside of Class I areas in the states not represented (LA, MO, NV, OK, OR, VT, WI) have W126 metric values at or below 13 ppm-hrs during the recent 3-year period (2016-2018). Across the states represented in the dataset, the fraction of a given state's Class I areas included in the dataset generally ranges from about a third to 100%. An exception to that is New Mexico, for which a monitoring site is in or near only one of the nine Class I areas in the state. This contrasts with neighboring Arizona, also in the Southwest region and for which more than half the Class I areas are represented in the dataset.

4D.5 SUMMARY

The preceding sections present analyses based on 19 years of O_3 concentration data reported at monitoring sites across the U.S. These analyses, intended to inform the review of the

current O_3 secondary standard, investigate spatial and temporal patterns in the W126 metric using monitoring data from 2000 to 2018 and the extent of relationships between the W126 metric, annual W126 index values and design values for the current secondary O_3 standard (i.e., the 4th max metric). Further analyses of O_3 concentrations in or near federally protected ecosystems known as Class I areas focus on examining the levels and distributions of levels of the W126 metric and the annual W126 index occurring in such areas when the current secondary standard is met and also when the current secondary standard is not met.

The analyses based on recent (2016-2018) data showed that about one quarter of U.S. sites had 4th max metric values greater than the current standard level of 70 ppb. By contrast, only about 1 in 12 U.S. sites had W126 metric values greater than 17 ppm-hrs, and about 1 in 8 U.S. sites had W126 metric values greater than 13 ppm-hrs. There were O_3 monitors exceeding the current standard level of 70 ppb in 8 of 9 climate regions, while two regions, the West and Southwest, had O_3 monitors with W126 metric values exceeding 13 ppm-hrs.

When examining the 4th max and W126 metrics in combination, the 2016-2018 data showed that there were many U.S. O_3 sites with 4th max metric values exceeding the current standard that had W126 metric values less than or equal to 17 ppm-hrs (173) and 13 ppm-hrs (147). By contrast, there were relatively few sites meeting the current standard that had W126 metric values greater than 13 ppm-hrs (27); and there was a single site that had a W126 metric value above 17 ppm-hrs. The 27 sites that met the current standard and had W126 metric values greater than 13 ppm-hrs were located exclusively in the Southwest and West climate regions, whereas the 147 sites that exceeded the current standard and had W126 metric values less than or equal to 13 ppm-hrs had a much broader geographic distribution.

Among O_3 monitoring sites in Federal Class I areas, few areas since 2000 have had 4th max metric values meeting the current standard and W126 metric values above 17 ppm-hrs, the most recent of which occurred during the 2012-2014 period. These instances are all in or near Class I areas in the Southwest region, with the highest (19 ppm-hrs) occurring during the 2006-2008 period.

The analysis of inter-annual variability shows that the distribution of annual W126 index deviations from their respective 3-year averages generally increase with increasing W126 metric values. For sites with W126 metric values below 20 ppm-hrs (e.g., focusing on W126 metric values that have occurred with 4th max metric values at or below 70 ppb), the annual deviation was generally within 5 ppm-hrs. Additionally, well over 99% of 4th max metric values meeting the current standard were associated with annual W126 index values of less than or equal to 19 ppm-hrs.

The trends analysis showed that both W126 metric values and annual W126 index values have generally decreased since 2000, with U.S. median W126 metric values decreasing by over

60%, from nearly 17 ppm-hrs in 2002 to less than 7 ppm-hrs in 2018. A substantial number of U.S. sites have experienced decreases of over 10 ppm-hrs in the past decade, particularly in the eastern U.S.

The analysis comparing trends in the 4th max metric values and to trends in the W126 metric values based on data from 2000-2018 showed that there was a positive, linear relationship between the per-year changes in the 4th max and W126 metrics. Nationally, the W126 metric values decreased by approximately 0.6 ppm-hr per unit ppb decrease in the 4th max metric values. This relationship varied across the NOAA climate regions. The Southwest and West regions which showed the greatest potential for exceeding only the W126 levels of interest also showed the greatest improvement in the W126 metric values per unit decrease in 4th max metric values. This analysis indicates that W126 metric values in those areas not meeting the current standard would be expected to decline as the 4th max metric values are reduced to meet the current standard, consistent with the relationship shown in Figure 4D-11.

4D.6 REFERENCES

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APPENDIX 4E

OZONE WELFARE EFFECTS AND RELATED ECOSYSTEM SERVICES AND PUBLIC WELFARE ASPECTS

Table 4E-1. Ecosystem services and aspects of public welfare potentially affected by the different types of O_3 welfare effects.

O_3 Effect[A]	Aspect of Public Welfare Potentially Affected (Examples)[B]	Ecosystem Services [C]
Visible foliar injury	• 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 Reduced plant reproduction Reduced yield and quality of agricultural crops Reduced productivity in terrestrial ecosystems	• 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 carbon sequestration in terrestrial systems	• Regulation/control of climatological features and meteorological phenomena • Changes in pollution removal in urban areas	Regulating Supporting
Increased tree mortality	• 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
Alteration of terrestrial community composition	• 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	• 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
Alteration of ecosystem water cycling	• Water quality and resource management • Regulation of hydraulic flow	Provisioning Regulating Supporting
Altered of herbivore growth and reproduction	• Food sources, habitat, and protection for native fauna	Supporting Regulating
Alteration of plant insect signaling	• Plant-pollinator interactions • Timber and agricultural plant resistance to insect pest damage	Supporting Provisioning
Radiative forcing and *related climate effects*	• 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 O_3 (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

Table 4E-2. Ecosystem services and specific uses of the 11 tree species with robust E-R functions for reduced growth.

Tree Species	O_3 Effect	Role in Ecosystems and Public Uses
Black Cherry *Prunus serotina*	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 song birds, game birds, and mammals
Eastern White Pine *Pinus strobus*	Biomass loss	Commercial timber, furniture, woodworking, and Christmas trees; Medicinal uses as expectorant and antiseptic; Food and habitat for song birds and mammals; Used to stabilize strip mine soils
Quaking Aspen *Populus tremuloides*	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 *Liriodendron tulipifera*	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 *Pinus ponderosa*	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 *Alnus rubra*	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^ *Acer rubrum*	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 *Pinus virginiana*	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 *Acer saccharum*	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 *Pseudotsuga menziesii*	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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