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

Second External Review Draft

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

Second External Review Draft

U.S. Environmental Protection Agency

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EXECUTIVE SUMMARY

This second draft Policy Assessment (PA) has been prepared by staff in the Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards (OAQPS) as part of the Agency's ongoing review of the primary (health-based) and secondary (welfare-based) national ambient air quality standards (NAAQS) for ozone (O_3). It presents analyses and preliminary staff conclusions regarding the policy implications of the key scientific and technical information that informs this review. Preliminary staff conclusions are presented regarding the adequacy of the current standards and, as appropriate, potential alternative standards appropriate for consideration in this review. Staff analyses in this second draft PA are based on the scientific and technical information, as well as uncertainties and limitations related to this information, assessed in other EPA documents, including the scientific assessment presented in the *Integrated Science Assessment for Ozone*, the second draft *Health Risk and Exposure Assessment for Ozone* and the second draft *Welfare Risk and Exposure Assessment for Ozone*. The final PA is intended to "bridge the gap" between the relevant scientific evidence and technical information and the judgments required of the EPA Administrator in determining whether to retain or revise the current standards. Development of the PA is also intended to facilitate advice and recommendations on the standards 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).

The overarching questions in this review, as in all NAAQS reviews, regard the support provided by the currently available scientific evidence and exposure/risk-based information for the adequacy of the current standards and the extent to which the scientific evidence and technical information provides support for concluding that consideration of alternative standards may be appropriate. Comments and recommendations from CASAC and public comments based on review of this draft PA will inform final staff conclusions and the presentation of information in the final PA.

Health Effects and Review of the Primary Standard

The longstanding and comprehensive evidence base, stronger today than in the last review, documents the effects of O_3 in ambient air on health. In particular, O_3 affects the respiratory system, posing greatest hazard to those with respiratory disease and those with highest exposures, including children with asthma. The evidence indicates that higher exposures and repeated occurrence of exposures lead to more severe effects, including increased susceptibility to other respiratory stressors, and that higher exposures lead to greater prevalence of effects among the exposed population. Based on the staff evaluation presented in this draft

1   document, staff preliminarily concludes that the currently available evidence and exposure and

2   risk information call into question the adequacy of the current primary standard and that

3   consideration should be given to revising the standard to provide increased public health

4   protection. With regard to potential alternative standards, staff concludes it is appropriate to

5   consider standards with the same indicator, averaging time and form as the current standard with

6   alternative levels within the range from 70 ppb to 60 ppb.

7   In drawing these preliminary conclusions, staff additionally notes that the final decision

8   on the adequacy of the current standard and consideration of potential alternative standards is

9   largely a public health policy judgment to be made by the Administrator, drawing upon the

10   scientific information as well as judgments about how to consider the range and magnitude of

11   uncertainties that are inherent in the scientific evidence and technical analyses.

Welfare Effects and Review of the Secondary Standard

13   The longstanding evidence base, strengthened since the last review, documents the

14   welfare-related effects of O_3 in ambient air. In particular, O_3 affects vegetation and poses risk of

15   related effects on terrestrial ecosystems. Based on the staff evaluation presented in this draft

16   document, staff preliminarily concludes that the currently available evidence and exposure and

17   risk information call into question the adequacy of the current secondary standard and that

18   consideration should be given to revising the standard to provide increased public welfare

19   protection. In considering the level of protection achieved by potential alternative standards,

20   staff preliminarily concludes it is appropriate for the Administrator to judge O_3 welfare impacts

21   using the W126-based cumulative seasonal index, defined as an index of the sum of weighted

22   hourly concentrations, cumulated over 12 hours per day (8 am to 8 pm) during the consecutive

23   three-month period within the O_3 season with the maximum index value. With regard to

24   potential alternative standards, staff preliminarily concludes it is appropriate to consider

25   standards in terms of the W126-based cumulative seasonal metric with a form averaged across

26   three consecutive years and levels extending somewhat above 15 ppm-hrs (e.g., to 17 ppm-hrs)

27   down to 7 ppm-hrs.

28   In drawing these preliminary conclusions, staff additionally notes that the final decision

29   on the adequacy of the current standard and consideration of potential alternative standards is

30   largely a public welfare policy judgment to be made by the Administrator, drawing upon the

31   scientific information as well as judgments about how to consider the range and magnitude of

32   uncertainties that are inherent in the scientific evidence and technical analyses.

1 INTRODUCTION

1.1 PURPOSE

The U.S. Environmental Protection Agency (EPA) is presently conducting a review of the primary (health-based) and secondary (welfare-based) national ambient air quality standards (NAAQS) for ozone (O_3). The overall plan for this review was presented in the *Integrated Review Plan for the O_3 National Ambient Air Quality Standards* (IRP, U.S. EPA, 2011a). The IRP also identified key policy-relevant issues to be addressed in this review and discussed the key documents that generally inform NAAQS reviews, including an Integrated Science Assessment (ISA), Risk and Exposure Assessments (REAs), and a Policy Assessment (PA). The PA is prepared by the staff in EPA's Office of Air Quality Planning and Standards (OAQPS). It presents a staff evaluation of the policy implications of the key scientific and technical information in the ISA and REAs for EPA's consideration.[1] The PA provides a transparent evaluation, and staff conclusions, regarding policy considerations related to reaching judgments about the adequacy of the current standards, and if revision is considered, what revisions may be appropriate to consider.

When final, the PA is intended to help "bridge the gap" between the Agency's scientific assessments presented in the ISA and REAs, and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS.[2] In evaluating the adequacy of the current standard and whether it is appropriate to consider potential alternative standards, the PA focuses on information that is most pertinent to evaluating the basic elements of the NAAQS: indicator,[3] averaging time, form,[4] and level. These elements, which together serve to define each standard, must be considered collectively in evaluating the health and welfare protection afforded by the O_3 standards. The PA integrates and interprets the information from the ISA and REAs to frame policy options for consideration by the Administrator. In so doing, the PA recognizes that the selection of a specific approach to reaching final decisions on the primary and secondary O_3 standards will reflect the judgments of the Administrator.

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[1] The terms "staff" and "we" through this document refer to personnel in the EPA's Office of Air Quality Planning and Standards (OAQPS).

[2] American Farm Bureau Federation v. EPA, 559 F. 3d 512, 521 (D.C. Cir. 2009); Natural Resources Defense Council v. EPA, 902 F. 2d 962, 967-68, 970 (D.C. Cir. 1990).

[3] The "indicator" of a standard defines the chemical species or mixture that is to be measured in determining whether an area attains the standard. The indicator for photochemical oxidants is ozone.

[4] 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 current 8-hour O_3 NAAQS is the 3-year average of the annual fourth-highest daily maximum 8-hour average.

1   The development of the PA is also intended to facilitate advice to the Agency and
2 recommendations to the Administrator from an independent scientific review committee, the
3 Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act. As
4 discussed below in section 1.2.1, the CASAC is to advise not only on the Agency's assessment
5 of the relevant scientific information, but also on the adequacy of the existing standards, and to
6 make recommendations as to any revisions of the standards that may be appropriate. The EPA
7 facilitates CASAC advice and recommendations, as well as public input and comment, by
8 requesting CASAC review and public comment on one or more drafts of the PA.
9   The decision whether to prepare one or more drafts of the PA is influenced by
10 preliminary staff conclusions and associated CASAC advice and public comment, among other
11 factors. Typically, as in this review, staff prepares a second draft PA where the available
12 information calls into question the adequacy of the current standard and analyses of potential
13 alternative standards are developed taking into consideration CASAC advice and public
14 comment. In such cases, a second draft PA includes preliminary staff conclusions regarding
15 potential alternative standards and undergoes CASAC review and public comment prior to
16 preparation of the final PA.[5]
17   In this second draft of the PA for the review of the O_3 NAAQS, we consider the scientific
18 and technical information available in this review as assessed in the *Integrated Science*
19 *Assessment for O_3 and Related Photochemical Oxidants* (ISA, U.S. EPA, 2013), prepared by
20 EPA's National Center for Environmental Assessment (NCEA), and the second drafts of the
21 quantitative human exposure and health risk assessment and welfare risk assessment documents
22 (HREA, U.S. EPA, 2014a; WREA, U.S. EPA, 2014b). The evaluation and preliminary staff
23 conclusions presented in this second draft PA have been informed by comments and advice
24 received from CASAC in their review of the first draft PA and of the other draft Agency
25 documents prepared for this NAAQS review. Review and comments on this second draft PA
26 from CASAC and the public will inform the final evaluation and staff conclusions in the final
27 PA.
28   Beyond informing the EPA Administrator and facilitating the advice and
29 recommendations of CASAC and the public, the PA is also intended to be a useful reference to
30 all parties interested in the NAAQS review. In these roles, it is intended to serve as a single
31 source of the most policy-relevant information that informs the Agency's review of the NAAQS,
32 and it is written to be understandable to a broad audience.

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[5] When such analyses are not undertaken, a second draft PA may not be warranted.

1   **1.2 BACKGROUND**

2   **1.2.1   Legislative Requirements**

3   Two sections of the Clean Air Act (CAA) govern the establishment and revision of the

4   NAAQS. Section 108 (42 U.S.C. section 7408) directs the Administrator to identify and list

5   certain air pollutants and then to issue air quality criteria for those pollutants. The Administrator

6   is to list those air pollutants that in her "judgment, cause or contribute to air pollution which may

7   reasonably be anticipated to endanger public health or welfare;" "the presence of which in the

8   ambient air results from numerous or diverse mobile or stationary sources;" and "for which . . .

9   [the Administrator] plans to issue air quality criteria…." Air quality criteria are intended to

10   "accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all

11   identifiable effects on public health or welfare which may be expected from the presence of [a]

12   pollutant in the ambient air . . ." 42 U.S.C. § 7408(b). Section 109 (42 U.S.C. 7409) directs the

13   Administrator to propose and promulgate "primary" and "secondary" NAAQS for pollutants for

14   which air quality criteria are issued. Section 109(b)(1) defines a primary standard as one "the

15   attainment and maintenance of which in the judgment of the Administrator, based on such

16   criteria and allowing an adequate margin of safety, are requisite to protect the public health." [6]

17   A secondary standard, as defined in section 109(b)(2), must "specify a level of air quality the

18   attainment and maintenance of which, in the judgment of the Administrator, based on such

19   criteria, is requisite to protect the public welfare from any known or anticipated adverse effects

20   associated with the presence of [the] pollutant in the ambient air." [7]

21   The requirement that primary standards provide an adequate margin of safety was

22   intended to address uncertainties associated with inconclusive scientific and technical

23   information available at the time of standard setting. It was also intended to provide a reasonable

24   degree of protection against hazards that research has not yet identified. See Lead Industries

25   Association v. EPA, 647 F.2d 1130, 1154 (D.C. Cir 1980); American Petroleum Institute v.

26   Costle, 665 F.2d 1176, 1186 (D.C. Cir. 1981); American Farm Bureau Federation v. EPA, 559 F.

27   3d 512, 533 (D.C. Cir. 2009); Association of Battery Recyclers v. EPA, 604 F. 3d 613, 617-18

28   (D.C. Cir. 2010). Both kinds of uncertainties are components of the risk associated with pollution

29   at levels below those at which human health effects can be said to occur with reasonable

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[6] 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).

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

1   scientific certainty. Thus, in selecting primary standards that provide an adequate margin of

2   safety, the Administrator is seeking not only to prevent pollution levels that have been

3   demonstrated to be harmful but also to prevent lower pollutant levels that may pose an

4   unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The

5   CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at

6   background concentration levels, see Lead Industries v. EPA, 647 F.2d at 1156 n.51; State of

7   Mississippi v. EPA, 723 F. 3d 246, 255, 262-63 (D.C. Cir. 2013), but rather at a level that

8   reduces risk sufficiently so as to protect public health with an adequate margin of safety.

9   In addressing the requirement for an adequate margin of safety, the EPA considers such

10   factors as the nature and severity of the health effects, the size of sensitive population(s)[8] at risk,

11   and the kind and degree of the uncertainties that must be addressed. The selection of any

12   particular approach for providing an adequate margin of safety is a policy choice left specifically

13   to the Administrator's judgment. See Lead Industries Association v. EPA, 647 F.2d at 1161-62;

14   State of Mississippi, 723 F. 3d at 265.

15   In setting primary and secondary standards that are "requisite" to protect public health

16   and welfare, respectively, as provided in section 109(b), EPA's task is to establish standards that

17   are neither more nor less stringent than necessary for these purposes. In so doing, the EPA may

18   not consider the costs of implementing the standards. See generally, Whitman v. American

19   Trucking Associations, 531 U.S. 457, 465-472, 475-76 (2001). Likewise, "[a]ttainability and

20   technological feasibility are not relevant considerations in the promulgation of national ambient

21   air quality standards." American Petroleum Institute v. Costle, 665 F. 2d at 1185.

22   Section 109(d)(1) requires that "not later than December 31, 1980, and at 5-year intervals

23   thereafter, the Administrator shall complete a thorough review of the criteria published under

24   section 108 and the national ambient air quality standards . . . and shall make such revisions in

25   such criteria and standards and promulgate such new standards as may be appropriate . . . ."

26   Section 109(d)(2) requires that an independent scientific review committee "shall complete a

27   review of the criteria . . . and the national primary and secondary ambient air quality standards . .

28   . and shall recommend to the Administrator any new . . . standards and revisions of existing

29   criteria and standards as may be appropriate . . . ." Since the early 1980's, the Clean Air

30   Scientific Advisory Committee (CASAC) has performed this independent review function. [9]

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[8] As used here and similarly throughout this document, the term population refers to persons having a quality or characteristic in common, including a specific pre-existing illness or a specific age or life stage.

[9] Lists of CASAC members and of members of the CASAC Ozone Review Panel are available at: http://yosemite.epa.gov/sab/sabpeople.nsf/WebCommitteesSubCommittees/Ozone%20Review%20Panel.

1   **1.2.2   History of O₃ NAAQS Reviews**

2   Table 1-1 summarizes the O3 NAAQS that the EPA has promulgated to date. In each

3 review, the EPA set the secondary standard at a level identical to the primary standard. These

4 reviews are briefly described below.

5   **Table 1-1.   Summary of primary and secondary O₃ NAAQS promulgated during the**
6   **period from 1971 to 2008.**

Final Rule	Indicator	Averaging Time	Level (ppm)	Form
1971 (36 FR 8186)	Total photochemical oxidants	1 hour	0.08	Not to be exceeded more than one hour per year
1979 (44 FR 8202)	O₃	1 hour	0.12	Attainment is defined when the expected number of days per calendar year, with maximum hourly average concentration greater than 0.12 ppm, is equal to or less than 1
1993 (58 FR 13008)	The EPA decided that revisions to the standards were not warranted at the time.			
1997 (62 FR 38856)	O₃	8 hours	0.08	Annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years
2008 (73 FR 16483)	O₃	8 hours	0.075	Form of the standards remained unchanged relative to the 1997 standard

7

8   The EPA first established primary and secondary NAAQS for photochemical oxidants in

9 1971 (36 FR 8186, April 30, 1971). The EPA set both primary and secondary standards at a level

10 of 0.08 parts per million (ppm), 1-hr average, total photochemical oxidants, not to be exceeded

11 more than one hour per year. The EPA based the standards on scientific information contained in

12 the 1970 *Air Quality Criteria for Photochemical Oxidants* (U.S. DHEW, 1970). We initiated the

13 first periodic review of the NAAQS for photochemical oxidants in 1977. Based on the 1978 *Air*

14 *Quality Criteria for Ozone and Other Photochemical Oxidants* (U.S. EPA, 1978), the EPA

15 published proposed revisions to the original NAAQS in 1978 (43 FR 16962) and final revisions

16 in 1979 (44 FR 8202). At that time, the EPA revised the level of the primary and secondary

17 standards from 0.08 to 0.12 ppm and changed the indicator from photochemical oxidants to O₃,

18 and the form of the standards from a deterministic to a statistical form. This statistical form

1 defined attainment of the standards as occurring when the expected number of days per calendar

2 year with maximum hourly average concentration greater than 0.12 ppm equaled one or less.

3   Following the final decision in the 1979 review, the City of Houston challenged the

4 Administrator's decision arguing that the standard was arbitrary and capricious because natural

5 O_3 concentrations and other physical phenomena in the Houston area made the standard

6 unattainable in that area. The U.S. Court of Appeals for the District of Columbia circuit (D.C.

7 Circuit) rejected this argument, stating (as noted above) that attainability and technological

8 feasibility are not relevant considerations in the promulgation of the NAAQS. The court also

9 noted that the EPA need not tailor the NAAQS to fit each region or locale, pointing out that

10 Congress was aware of the difficulty in meeting standards in some locations and had addressed

11 this difficulty through various compliance related provisions in the Act. See API v. Costle, 665

12 F.2d 1176, 1184-6 (D.C. Cir. 1981). In 1982, we announced plans to revise the 1978 Air Quality

13 Criteria document (47 FR 11561), and in 1983, we initiated the second periodic review of the O_3

14 NAAQS (48 FR 38009). We subsequently published the 1986 *Air Quality Criteria for Ozone*

15 *and Other Photochemical Oxidants* (U.S. EPA, 1986) and the 1989 Staff Paper (U.S. EPA,

16 1989). Following publication of the 1986 Air Quality Criteria Document (AQCD), a number of

17 scientific abstracts and articles were published that appeared to be of sufficient importance

18 concerning potential health and welfare effects of O_3 to warrant preparation of a Supplement

19 (U.S. EPA, 1992). On August 10, 1992, under the terms of a court order, the EPA published a

20 proposed decision to retain the existing primary and secondary standards. (57 FR 35542). The

21 notice explained that the proposed decision would complete EPA's review of information on

22 health and welfare effects of O_3 assembled over a 7-year period and contained in the 1986

23 AQCD and its 1992 Supplement. The proposal also announced EPA's intention to proceed as

24 rapidly as possible with the next review of the air quality criteria and standards for O_3 in light of

25 emerging evidence of health effects related to 6- to 8-hour O_3 exposures. On March 9, 1993, the

26 EPA concluded the review by affirming its proposed decision to retain the existing primary and

27 secondary standards. (58 FR 13008).

28   In August 1992, we announced plans to initiate the third periodic review of the air quality

29 criteria and O_3 NAAQS (57 FR 35542). In December 1996, the EPA proposed to replace the then

30 existing 1-hour primary and secondary standards with 8-hour average O_3 standards set at a level

31 of 0.08 ppm (equivalent to 0.084 ppm using standard rounding conventions) (61 FR 65716). The

32 EPA also proposed to establish a new distinct secondary standard using a biologically-based

33 cumulative, seasonal form. The EPA completed this review on July 18, 1997 (62 FR 38856) by

34 setting the primary standard at a level of 0.08 ppm, based on the annual fourth-highest daily

35 maximum 8-hr average concentration, averaged over three years, and setting the secondary

36 standard identical to the revised primary standard. In reaching this decision, the EPA identified

1   several reasons supporting its decision to reject a potential alternate standard set at 0.07 ppm.

2   Most importantly, the EPA pointed out the scientific uncertainty at lower concentrations and

3   placed significant weight on the fact that no CASAC panel member supported a standard level

4   set lower than 0.08 ppm (62 FR 38868). In addition to noting the uncertainties in the health

5   evidence for exposure concentrations below 0.08 ppm and the advice of CASAC, the EPA noted

6   that a standard set at a level of 0.07 ppm would be closer to peak background concentrations that

7   infrequently occur in some areas due to nonanthropogenic sources of O_3 precursors (62 FR

8   38856, 38868; July 18, 1997).

9   On May 14, 1999, in response to challenges by industry and others to EPA's 1997

10 decision, the U.S. Court of Appeals for the District of Columbia Circuit remanded the O_3

11 NAAQS to the EPA, finding that section 109 of the Act, as interpreted by the EPA, effected an

12 unconstitutional delegation of legislative authority. American Trucking Assoc. vs. EPA, 175

13 F.3d 1027, 1034-1040(D.C. Cir. 1999) ("ATA I"). In addition, the court directed that, in

14 responding to the remand, the EPA should consider the potential beneficial health effects of O_3

15 pollution in shielding the public from the effects of solar ultraviolet (UV) radiation, as well as

16 adverse health effects. Id. At 1051-53. In 1999, the EPA petitioned for rehearing *en banc* on

17 several issues related to that decision. The court granted the request for rehearing in part and

18 denied it in part, but declined to review its ruling with regard to the potential beneficial effects of

19 O_3 pollution. 195 F3d 4, 10 (D.C Cir., 1999) ("ATA II"). On January 27, 2000, the EPA

20 petitioned the U.S. Supreme Court for certiorari on the constitutional issue (and two other

21 issues), but did not request review of the ruling regarding the potential beneficial health effects

22 of O_3. On February 27, 2001, the U.S. Supreme Court unanimously reversed the judgment of the

23 D.C. Circuit on the constitutional issue. Whitman v. American Trucking Assoc., 531 U. S. 457,

24 472-74 (2001) (holding that section 109 of the CAA does not delegate legislative power to the

25 EPA in contravention of the Constitution). The Court remanded the case to the D.C. Circuit to

26 consider challenges to the O_3 NAAQS that had not been addressed by that court's earlier

27 decisions. On March 26, 2002, the D.C. Circuit issued its final decision on remand, finding the

28 1997 O_3 NAAQS to be "neither arbitrary nor capricious," and so denying the remaining petitions

29 for review. American Trucking Associations, Inc. v EPA, 283 F.3d 355, 379 (D.C Cir.,

30 2002)("ATA III").

31   Specifically, in ATA III, the D.C. Circuit upheld EPA's decision on the 1997 O_3 standard

32 as the product of reasoned decision-making. The Court made clear that the most important

33 support for EPA's decision was the health evidence and the concerns it raised about setting a

34 standard level below 0.08 ppm. ("the record is replete with references to studies demonstrating

35 the inadequacies of the old one-hour standard", as well as extensive information supporting the

36 change to an 8-hour averaging time). 283 F 3d at 378. The Court also pointed to the significant

1 weight that the EPA properly placed on the advice it received from CASAC. Id. at 379. The

2 court further noted that "although relative proximity to peak background ozone concentrations

3 did not, in itself, necessitate a level of 0.08, EPA could consider that factor when choosing

4 among the three alternative levels." Id.

5 Independently of the litigation, the EPA also responded to the Court's remand to

6 consider the potential beneficial health effects of O_3 pollution in shielding the public from effects

7 of solar (ultraviolet or UV-B) radiation. The EPA provisionally determined that that the

8 information linking changes in patterns of ground-level O_3 concentrations to changes in relevant

9 patterns of exposures to ultraviolet (UV-B) radiation of concern to public health was too

10 uncertain, at that time, to warrant any relaxation in 1997 O_3 NAAQS. The EPA also expressed

11 the view that any plausible changes in UV-B radiation exposures from changes in patterns of

12 ground-level O_3 concentrations would likely be very small from a public health perspective.   In

13 view of these findings, the EPA proposed to leave the 1997 8-hour NAAQS unchanged (66 FR

14 57268, Nov. 14, 2001). After considering public comment on the proposed decision, the EPA

15 published its final response to this remand on January 6, 2003, re-affirming the 8-hour O_3

16 NAAQS set in 1997 (68 FR 614).

17 The EPA initiated the fourth periodic review of the air quality criteria and O_3 standards in

18 September 2000 with a call for information (65 FR 57810). The schedule for completion of that

19 review was ultimately governed by a consent decree resolving a lawsuit filed in March 2003 by

20 plaintiffs representing national environmental and public health organizations, who maintained

21 that EPA was in breach of a mandatory legal duty to complete review of the O_3 NAAQS within a

22 statutorily-mandated deadline. On July 11, 2007, the EPA proposed to revise the level of the

23 primary standard within a range of 0.075 to 0.070 ppm. (72 FR 37818). Documents supporting

24 this proposed decision included the *Air Quality Criteria for Ozone and Other Photochemical*

25 *Oxidants* (U.S. EPA, 2006) and the Staff Paper (U.S EPA, 2007a) and related technical support

26 documents. The EPA also proposed two options for revising the secondary standard: (1) replace

27 the current standard with a cumulative, seasonal standard, expressed as an index of the annual

28 sum of weighted hourly concentrations cumulated over 12 daylight hours during the consecutive

29 3-month period within the O_3 season with the maximum index value, set at a level within the

30 range of 7 to 21 ppm-hrs, and (2) set the secondary standard identical to the proposed primary

31 standard. The EPA completed the review with publication of a final decision on March 27, 2008

32 (73 FR 16436). In that final rule, the EPA revised the NAAQS by lowering the level of the 8-

33 hour primary O_3 standard from 0.08 ppm to 0.075 ppm, not otherwise revising the primary

34 standard, and adopting a secondary standard identical to the revised primary standard. In May

35 2008, state, public health, environmental, and industry petitioners filed suit challenging EPA's

36 final decision on the 2008 O_3 standards. On September 16, 2009, the EPA announced its

1   intention to reconsider the 2008 O_3 standards, and initiated a rulemaking to do so. At EPA's

2   request, the Court held the consolidated cases in abeyance pending EPA's reconsideration of the

3   2008 decision.

4   On January 19, 2010 (75 FR 2938), the EPA issued a notice of proposed rulemaking to

5   reconsider the 2008 final decision. In that notice, the EPA proposed that further revisions of the

6   primary and secondary standards were necessary to provide a requisite level of protection to

7   public health and welfare. The EPA proposed to decrease the level of the 2008 8-hour primary

8   standard from 0.075 ppm to a level within the range of 0.060 to 0.070 ppm, and to change the

9   secondary standard to a new cumulative, seasonal standard expressed as an annual index of the

10 sum of weighted hourly concentrations, cumulated over 12 hours per day (8 am to 8 pm), during

11 the consecutive 3-month period within the O_3 season, with a maximum index value set at a level

12 within the range of 7 to 15 ppm-hours. The Agency also solicited CASAC review of the

13 proposed rule on January 25, 2010 and solicited additional CASAC advice on January 26, 2011.

14 After considering comments from CASAC and the public, the EPA prepared a draft final rule,

15 which was submitted for interagency review pursuant to Executive Order 12866. On September

16 2, 2011, consistent with the direction of the President, the Administrator of the Office of

17 Information and Regulatory Affairs ("OIRA"), Office of Management and Budget ("OMB"),

18 returned the draft final rule to the EPA for further consideration. In view of this return and the

19 timing of the Agency's ongoing periodic review of the O_3 NAAQS required under Clean Air Act

20 section 109 (as announced on September 29, 2008), the EPA decided to coordinate further

21 proceedings on its voluntary rulemaking on reconsideration with that ongoing periodic review,

22 by deferring the completion of its voluntary rulemaking on reconsideration until it completes its

23 statutorily-required periodic review.

24   In light of EPA's decision to consolidate the reconsideration with the current review, the

25 Court proceeded with the litigation on the 2008 final decision. On July 23, 2013, the D.C. Circuit

26 Court of Appeals upheld EPA's 2008 primary O_3 standard, but remanded the 2008 secondary

27 standard to the EPA. State of Mississippi v. EPA. 723 F. 3d 246. With respect to the primary

28 standard, the court first held that the EPA reasonably determined that the existing standard was

29 not requisite to protect public health with an adequate margin of safety, and consequently

30 required revision. Specifically, the court noted that there were "numerous epidemiological

31 studies linking health effects to exposure to ozone levels below 0.08 ppm and clinical human

32 exposure studies finding a causal relationship between health effects and exposure to ozone

33 levels at and below 0.08 ppm". Id. at 257. The court also specifically endorsed the weight of

34 evidence approach utilized by EPA in its deliberations. Id. at 256.

35   The court went on to reject arguments that EPA should have adopted a more stringent

36 primary standard. Dismissing arguments that a single clinical study (properly interpreted by

1 EPA) to show effects at 0.06 ppm necessitated a standard level lower than that selected, the court
2 noted that this was a single, limited study. Id. at 262. With respect to the epidemiologic evidence,
3 the court accepted EPA's argument that there could be legitimate uncertainty that a causal
4 relationship between O_3 and 8-hour exposures less than 0.075 ppm exists, so that associations at
5 lower levels reported in epidemiologic studies did not necessitate a more stringent standard. Id.
6 at 264-65.[10]

7 The court also rejected arguments that an 8-hour primary standard of 0.075 ppm failed to
8 provide an adequate margin of safety, noting that margin of safety considerations involved policy
9 judgments by the agency, and that by setting a standard "appreciably below" the level of the
10 current standard (0.08 ppm), the agency had made a reasonable policy choice . Id. Finally, the
11 court rejected arguments that EPA's decision was inconsistent with CASAC's scientific
12 recommendations because CASAC had been insufficiently clear in its recommendations whether
13 it was providing scientific or policy recommendations, and EPA had reasonably addressed
14 CASAC's policy recommendations. Id. at 269-70.

15 With respect to the secondary standard, the court held that because EPA had failed to
16 identify a level of air quality requisite to protect public welfare, EPA's comparison between the
17 primary and secondary standards for determining if requisite protection for public welfare was
18 afforded by the primary standard was inherently arbitrary. The court thus rejected EPA's
19 determination that the revised 8-hour primary standard afforded requisite protection of public
20 welfare, and remanded the standard to EPA. Id. at 272-73.

21 **1.2.3   Current O_3 NAAQS Review**

22 On September 29, 2008, the EPA announced the initiation of a new periodic review of
23 the air quality criteria for O_3 and related photochemical oxidants and issued a call for
24 information in the Federal Register (73 FR 56581, Sept. 29, 2008). A wide range of external
25 experts, as well as EPA staff, representing a variety of areas of expertise (e.g., epidemiology,
26 human and animal toxicology, statistics, risk/exposure analysis, atmospheric science, ecology,
27 biology, plant science, ecosystem services) participated in a workshop. This workshop was held
28 on October 28-29, 2008 in Research Triangle Park, NC. The workshop provided an opportunity
29 for a public discussion of the key policy-relevant issues around which the EPA would structure
30 this O_3 NAAQS review and the most meaningful new science that would be available to inform
31 our understanding of these issues.

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[10] The court cautioned, however, that "perhaps more [clinical] studies like the Adams studies will yet reveal that the 0.060 ppm level produces significant adverse decrements that simply cannot be attributed to normal variation in lung function", and further cautioned that "agencies may not merely recite the terms 'substantial uncertainty' as a justification for their actions'". Id. at 262, 269 (internal citations omitted).

1   Based in part on the workshop discussions, the EPA developed a draft IRP outlining the
2   schedule, process, and key policy-relevant questions that would guide the evaluation of the air
3   quality criteria for O_3 and the review of the primary and secondary O_3 NAAQS. A draft of the
4   integrated review plan was released for public review and comment in September 2009. This IRP
5   was the subject of a consultation with the CASAC on November 13, 2009 (74 FR 54562;
6   October 22, 2009).[11] We considered comments received from that consultation and from the
7   public in finalizing the plan and in beginning the review of the air quality criteria. The EPA's
8   overall plan and schedule for this review is presented in the *Integrated Review Plan for the*
9   *Ozone National Ambient Air Quality Standards*.[12]
10   As part of the process of preparing the O_3 ISA, NCEA hosted a peer review workshop in
11   October 29-30, 2008 (73 FR 56581, September 29, 2008) on preliminary drafts of key ISA
12   chapters. The CASAC and the public reviewed the first external review draft ISA (U.S. EPA,
13   2011b; 76 FR 10893, February 28, 2011) at a meeting held in May 19-20, 2011 (76 FR 23809;
14   April 28, 2011). Based on CASAC and public comments, NCEA prepared a second draft ISA
15   (U.S. EPA, 2011c; 76 FR 60820, September 30, 2011). CASAC and the public reviewed this
16   draft at a January 9-10, 2012 (76 FR 236, December 8, 2011) meeting. Based on CASAC and
17   public comments, NCEA prepared a third draft ISA (U.S. EPA 2012a; 77 FR 36534; June 19,
18   2012), which was reviewed at a CASAC meeting in September 2012. The final ISA was released
19   in February 2013.
20   The EPA presented its plans for conducting the Risk and Exposure Assessments (REAs)
21   that build on the scientific evidence presented in the ISA, in two planning documents titled
22   *Ozone National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and*
23   *Exposure Assessment* and *Ozone National Ambient Air Quality Standards: Scope and Methods*
24   *Plan for Welfare Risk and Exposure Assessment* (henceforth, Scope and Methods Plans).[13]
25   These planning documents outlined the scope and approaches that staff planned to use in
26   conducting quantitative assessments, as well as, key issues that would be addressed as part of the
27   assessments. We released these documents for public comment in April 2011, and consulted with
28   CASAC on May 19-20, 2011 (76 FR 23809; April 28, 2011). In designing and conducting the
29   initial health risk and welfare risk assessments, we considered CASAC comments (Samet 2011)
30   on the Scope and Methods Plans and also considered public comments. In May 2012, we issued

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[11] See http://yosemite.epa.gov/sab/sabproduct.nsf/WebProjectsbyTopicCASAC!OpenView for more information on
CASAC activities related to the current O_3 NAAQS review.

[12] EPA 452/R-11-006; April 2011; Available:
http://www.epa.gov/ttn/naaqs/standards/ozone/data/2011_04_OzoneIRP.pdf

[13] EPA-452/P-11-001 and -002; April 2011; Available:
http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_pd.html

1   a memo titled, *Updates to Information Presented in the Scope and Methods Plans for the Ozone*
2   *NAAQS Health and Welfare Risk and Exposure Assessments*, that described changes to elements
3   of the scope and methods plans and provided a brief explanation of each change and the reason
4   for it.
5   In July 2012, EPA made the first drafts of the Health and Welfare REAs available for
6   CASAC review and public comment (77 FR 42495, July 19, 3023). The first draft PA was made
7   available for CASAC review and public comment in August 2012. These documents were
8   reviewed by CASAC O_3 Panel at a public meeting in September 2012. The second draft REAs
9   and PA have been prepared in consideration of CASAC (Frey and Samet, 2012a, 2012b) and
10   public comment and will be reviewed by the CASAC O_3 Panel at a public meeting in March
11   2014.

12   **1.3 GENERAL APPROACH FOR REVIEW OF THE STANDARDS**
13   As described in section 1.1 above, the final PA will present a transparent evaluation and
14   staff conclusions regarding policy considerations related to reaching judgments about the
15   adequacy of the current standards and what, if any, revisions may be appropriate to consider.
16   Preliminary staff considerations and conclusions in this document are based on the available
17   body of scientific evidence assessed in the ISA (U.S. EPA, 2013), exposure and risk analyses
18   presented in the 2nd draft REAs (U.S. EPA, 2014a, b), advice and recommendations from
19   CASAC on the first draft PA and other draft EPA documents in this review, as well as on public
20   comments. When final, this evaluation and associated conclusions on the range of policy options
21   that, in staff's view, could be supported by the available scientific evidence and exposure/risk
22   information will inform the Administrator's decisions as to whether the existing primary and/or
23   secondary O_3 standards should be revised and, if so, what revised standard or standards is/are
24   appropriate.
25   Staff's considerations and conclusions related to the current and alternative primary and
26   secondary O_3 standards are framed by a series of key policy-relevant questions, expanding upon
27   those presented in the IRP at the outset of this review (U.S. EPA, 2011a). Answers to these
28   questions in the final PA will inform the Administrator's decisions as to whether, and if so how,
29   to revise the current O_3 standards. The first overarching question is as follows.

30   • **Do the currently available scientific evidence and exposure/risk information, as**
31   **reflected in the ISA and REAs, support or call into question the adequacy of the**
32   **protection afforded by the current O_3 standards?**

33   If the answer to this question, which is informed by staff's consideration of more specific
34   questions related to the primary and secondary standards, suggests that revision of the current

1 standards may be appropriate, then staff further considers the currently available evidence and

2 information with regard to the following question.

3   • **What range of potential alternative standards is appropriate to consider based on**
4   **the scientific evidence, air quality analyses, and exposure/risk-based**
5   **information?**

6 The general approaches for consideration of these overarching questions in review of the primary

7 and secondary standards are described separately in sections 1.3.1 and 1.3.2 below.

8   **1.3.1   Approach for the Primary Standard**

9   Staff's approach in this review of the current primary O_3 standard takes into

10 consideration the approaches used in previous O_3 NAAQS reviews. The past and current

11 approaches described below are both based, fundamentally, on using EPA's assessment of the

12 current scientific evidence and associated quantitative analyses to inform the Administrator's

13 judgment regarding a primary standard for O_3 that is "requisite" (i.e., neither more nor less

14 stringent than necessary) to protect public health with an adequate margin of safety.

15   In reaching conclusions on options for the Administrator's consideration, we note that the

16 final decision to retain or revise the current primary O_3 standard is a public health policy

17 judgment to be made by the Administrator. This final decision by the Administrator will draw

18 upon the available scientific evidence for O_3-attributable health effects, and on analyses of

19 population exposures and health risks, including judgments about the appropriate weight to

20 assign the range of uncertainties inherent in the evidence and analyses. Our general approach to

21 informing these judgments, discussed more fully below, recognizes that the available health

22 effects evidence reflects a continuum from relatively higher O_3 concentrations, at which

23 scientists generally agree that health effects are likely to occur, through lower concentrations, at

24 which the likelihood and magnitude of a response become increasingly uncertain. Therefore, in

25 developing conclusions in this second draft PA, we are mindful that the Administrator's ultimate

26 judgments on the primary standard will most appropriately reflect an interpretation of the

27 available scientific evidence and exposure/risk information that neither overstates nor understates

28 the strengths and limitations of that evidence and information. This approach is consistent with

29 the requirements of sections 108 and 109 of the Act, as well as with how the EPA and the courts

30 have historically interpreted the Act.

31   Section 1.3.1.1 below provides an overview of the general approach taken in the last

32 review of the primary O_3 NAAQS (i.e., the 2008 review), and a summary of the rationale for the

33 decision on the level of the standard in that review (73 FR 16436). Section 1.3.1.2 presents our

34 approach in the current review, including our approach to considering the health evidence and

1   exposure/risk information, and considerations regarding ambient O_3 concentrations attributable
2   to background sources.

1.3.1.1 Approach Used in the Last Review

4   In the 2008 review of the O_3 NAAQS, the Administrator relied upon consideration of the
5   available scientific evidence and exposure/risk information, the advice and recommendations of
6   CASAC, and comments from the public. Based on this, he revised the level of the 8-hour
7   primary O_3 standard from 0.08 ppm[14] to 0.075 ppm (75 ppb[15]). In reaching a decision to revise
8   the 1997 8-hour primary O_3 standard, the Administrator noted that much new evidence had
9   become available since the 1997 review. He noted that this body of scientific evidence was very
10   robust and provided consistent and coherent evidence of an array of O_3-related respiratory
11   morbidity effects, and possibly cardiovascular-related morbidity, as well as total nonaccidental
12   and cardiorespiratory mortality. The Administrator specifically observed that (1) the evidence of
13   a range of respiratory-related morbidity effects had been considerably strengthened; (2) newly
14   available evidence from controlled human exposure and epidemiologic studies identified people
15   with asthma as an important susceptible population for which estimates of respiratory effects in
16   the general population likely underestimate the magnitude or importance of these effects; (3)
17   newly available evidence about mechanisms of toxicity more completely explained the
18   biological plausibility of O_3-induced respiratory effects and was beginning to suggest
19   mechanisms that may link O_3 exposure to cardiovascular effects; and (4) there was relatively
20   strong evidence for associations between short-term O_3 concentrations and total nonaccidental
21   and cardiopulmonary mortality. The Administrator believed that this very robust body of
22   evidence enhanced our understanding of O_3- related effects and provided increased confidence
23   that various respiratory morbidity effects and other effects marked by indicators of respiratory
24   morbidity are causally related to O_3 exposures, and that the evidence was highly suggestive that
25   O_3 exposures during the warm O_3 season contribute to premature mortality.[16]
26   The Administrator also noted important new health evidence reporting a broad array of
27   adverse effects following short-term exposures to O_3 concentrations below the level of the 1997
28   standard, and concerns for such or related effects in at-risk populations,[17] including people with

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[14] Due to rounding convention, the 1997 standard level of 0.08 ppm corresponded to 0.084 ppm (84 ppb).

[15] The level of the O_3 standard is specified as 0.075 ppm rather than 75 ppb. However, in this draft PA we refer to ppb, which is most often used in the scientific literature and in the ISA, in order to avoid the confusion that could result from switching units when discussing the evidence in relation to the standard level.

[16] 73 FR 16470-16471 (March 27, 2008)

[17] Here, as in the ISA, the term "at-risk population" is used to encompass populations or lifestages that have a greater likelihood of experiencing health effects related to exposure to an air pollutant due to a variety of factors; other terms used in the literature include susceptible, vulnerable, and sensitive. These factors may be intrinsic, such

1 asthma or other lung diseases, older adults with increased susceptibility, and those who are likely

2 to be vulnerable as a result of spending a lot of time outdoors engaged in physical activity (e.g.,

3 especially active children and outdoor workers).

4   He specifically noted new scientific evidence, which built upon existing evidence,

5 demonstrating O_3-induced lung function effects and respiratory symptoms in some healthy

6 individuals following exposures down to 80 ppb. He also noted very limited new evidence

7 demonstrating such effects at exposure concentrations well below 80 ppb. In addition, the

8 Administrator noted (1) epidemiologic evidence of statistically significant associations with O_3-

9 related health effects in areas that likely would have met the then-current standard; (2)

10 epidemiologic studies conducted in areas that likely would have violated the existing standard

11 but which nonetheless reported statistically significant associations that generally extended down

12 to ambient O_3 concentrations below the level of that standard; (3) the few studies that had

13 reported statistically significant associations with respiratory morbidity outcomes and mortality

14 in subsets of data that included only days with ambient O_3 concentrations below the level of the

15 existing standard; and (4) controlled human exposure studies, together with animal toxicological

16 studies, that provided considerable support for the biological plausibility of the respiratory

17 morbidity associations observed in the epidemiologic studies. Based on the available evidence,

18 the Administrator agreed with the CASAC and the majority of public commenters that the

19 existing standard was not requisite to protect public health with an adequate margin of safety (FR

20 73 16471).

21   Beyond this focus on the available health evidence, the Administrator also considered

22 estimates of O_3 exposures and health risks based on analyses where air quality was adjusted to

23 simulate just meeting the existing and potential alternative standards. For the various air quality

24 simulations, he specifically considered the pattern of estimated reductions in O_3 exposures across

25 health benchmark concentrations of 80, 70, and 60 ppb. The 80 ppb benchmark reflected an

26 exposure concentration for which there was strong evidence for respiratory effects in healthy

27 people, including airway inflammation, respiratory symptoms, airway hyperresponsiveness, and

28 impaired lung host defense (U.S. EPA, 2007, section 4.7). The 60 ppb benchmark reflected an

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as genetic factors, lifestage, or the presence of preexisting diseases, or they may be extrinsic, such as socioeconomic status (SES), activity pattern and exercise level, or increased pollutant exposures (U.S. EPA 2013, p. lxx, 8-1, 8-2). The courts and the Act's legislative history refer to these at-risk subpopulations as "susceptible" or "sensitive" populations. See, e.g., American Lung Ass'n v. EPA, 134 F. 3d 388, 389 (D.C. Cir. 1998) ("NAAQS must protect not only average health individuals, but also 'sensitive citizens' – children, for example, or people with asthma, emphysema, or other conditions rendering them particularly vulnerable to air pollution" (quoting S. Rep. No. 91-1196 at 10).

1 exposure concentration for which the Administrator judged the evidence of such effects to be
2 very limited (73 FR 16471).
3   The Administrator took note of the magnitudes of estimated health risks for a range of
4 health effects, including moderate and large lung function decrements, respiratory symptoms,
5 respiratory-related hospital admissions, and nonaccidental and cardiorespiratory mortality. He
6 recognized that these quantitative risk estimates for a limited number of specific health effects
7 were indicative of a much broader array of O_3-related effects, including various indicators of
8 morbidity in at-risk populations that we could not analyze in the risk assessment (e.g., school
9 absences, increased medication use, emergency department visits). The Administrator concluded
10 that quantitative exposure and risk estimates, as well as the broader array of O_3-related health
11 endpoints that could not be quantified, provided additional support for the evidence-based
12 conclusion that the existing standard needed to be revised (73 FR 16472).
13   Based on the above considerations, and consistent with CASAC's unanimous conclusion
14 that there was no scientific justification for retaining the existing standard, the Administrator
15 concluded that the primary O_3 standard set in 1997 was not sufficient and thus not requisite to
16 protect public health with an adequate margin of safety. He further concluded that revision of
17 this standard was needed to provide increased public health protection (73 FR 16472).
18   Throughout the 2008 review, CASAC supported a standard level in the range of 60 to 70
19 ppb (without change to the form, indicator, or averaging time). In a letter to the Administrator on
20 the second draft Staff Paper, CASAC unanimously recommended "that the current primary
21 ozone standard be revised and that the level that should be considered for the revised standard be
22 from 0.060 to 0.070 ppm'' (60 to 70 ppb) (Henderson, 2006, p. 5). This recommendation, based
23 in part on the placement of more weight on the evidence for effects following exposures to 60
24 ppb O_3, followed from the CASAC's more general recommendation that the 1997 standard
25 needed to be made substantially more protective of human health, particularly for at-risk
26 populations. In a subsequent letter sent specifically to offer advice to aid the Administrator and
27 Agency staff in developing the 2007 O_3 proposal, CASAC reiterated that Panel members ''were
28 unanimous in recommending that the level of the current primary ozone standard should be
29 lowered from 0.08 ppm to no greater than 0.070 ppm'' (Henderson, 2007, p. 2).[18]
30   After considering CASACs comments, the Administrator judged that the appropriate
31 balance to draw, based on the entire body of evidence and information available in the 2008

[18] The D.C. Circuit, in its review of the 2008 primary standard, stated that it was unclear whether CASAC's advice reflected issues of pure science or issues of science and policy. That is, the court was unable to determine whether CASAC's conclusion in its 2007 letter that the standard be set no higher than 70 ppb "was based on its scientific judgment that adverse effects would occur at that level or instead based on its more qualitative judgment that the range it proposed would be more appropriately protective of human health with an adequate margin of safety." *Mississippi.* 723 F. 3d at 269.

1　review, was a standard set at a level of 75 ppb (and leaving all other elements of the NAAQS

2　unchanged). In making this decision, the Administrator placed primary emphasis on the body of

3　available scientific evidence, while viewing the results of exposure and risk assessments as

4　providing supporting information. Specifically, the Administrator judged that a standard set at

5　75 ppb would be appreciably below 80 ppb, the level in controlled human exposure studies at

6　which adverse effects had been demonstrated at the time, and would provide a significant

7　increase in protection compared to the then-current standard. Based on results of the exposure

8　assessment, he also noted that exposures to O_3 concentrations at and above a benchmark level of

9　80 ppb would be essentially eliminated with a standard level of 75 ppb, and that exposures at and

10　above a 70 ppb benchmark level would be substantially reduced or eliminated for the vast

11　majority of people in at-risk groups. In addition, the Administrator concluded that the body of

12　evidence did not support setting a lower standard level, specifically judging that the available

13　evidence for effects following exposures to O_3 concentrations of 60 ppb was "too limited to

14　support a primary focus at this level" (75 FR 2938). With respect to the epidemiologic evidence,

15　the Administrator stated that a standard set at a level lower than 75 ppb "would only result in

16　significant further public health protection if, in fact, there is a continuum of health risks in areas

17　with 8-hour average O_3 concentrations that are well below the concentrations observed in the key

18　controlled human exposure studies and if the reported associations observed in the

19　epidemiological studies are, in fact, causally related to O_3 at those lower levels" (73 FR 16483).

20　　　In making his final decision about the level of the primary O_3 standard, the Administrator

21　noted that the level of 75 ppb was above the range recommended by CASAC (i.e., 70 to 60 ppb).

22　He concluded that "CASAC's recommendation appeared to be a mixture of scientific and policy

23　considerations" (75 FR 2992). The Administrator reached a different policy judgment than the

24　CASAC Panel, placing less weight than CASAC on the available controlled human exposure

25　studies reporting effects following exposures to 60 ppb O_3 and less weight on the results from

26　exposure and risk assessments, particularly on estimates of exposures to O_3 concentrations at or

27　above 60 ppb (73 FR 16482-3).

28　　**1.3.1.2　Approach for the Current Review**

29　　　To identify the range of options appropriate for the Administrator to consider in the

30　current review, we apply an approach that builds upon the general approach used in the last

31　review (and in the 2010 reconsideration proposal) and that reflects the broader body of scientific

32　evidence, updated exposure/risk information, and advances in O_3 air quality modeling now

33　available. As summarized above, the Administrator's decisions in the prior review were based on

34　an integration of information on health effects associated with exposure to O_3, judgments on the

1 adversity and public health significance of key health effects, and expert and policy judgments as
2 to when the standard is requisite to protect public health with an adequate margin of safety.
3   Staff's preliminary conclusions on the primary O_3 standard reflect our consideration of
4 the available scientific evidence, exposure/risk information, and air quality modeling
5 information, within the context of the overarching questions related to: (1) the adequacy of the
6 current primary O_3 standard to protect against effects associated with both short- and long-term
7 exposures and (2) potential alternative standards, if any, that are appropriate to consider in this
8 review. In addressing these broad questions, we organize the discussions in chapters 3 and 4 of
9 this document around a series of more specific questions reflecting different aspects of each
10 overarching question. When evaluating the health protection afforded by the current or potential
11 alternative standards, we take into account the four basic elements of the NAAQS: the indicator,
12 averaging time, form, and level.
13   Figure 1-1 below provides an overview of our approach in this review. We believe that
14 the general approach summarized in this section, and outlined in Figure 1-1, provides a
15 comprehensive basis to help inform the judgments required of the Administrator in reaching
16 decisions about the current and potential alternative primary O_3 standards. In the subsections
17 below, we describe our general approaches to considering the scientific evidence (evidence-
18 based considerations) and to considering the human exposure- and health risk information
19 (exposure- and risk-based considerations). We also recognize considerations related to ambient
20 O_3 attributable to background sources.

Adequacy of Current 8-Hour Primary O$_3$ Standard

Evidence-Based Considerations
➢ISA weight-of-evidence conclusions for health effects and at-risk populations
➢Controlled human exposure and animal toxicology studies: Nature, magnitude, likely adversity of effects; consistency across studies?
➢Epidemiologic studies: Statistical precision; confounding; link effects to O$_3$ air quality in specific locations?
    ➢Health effect associations reported in locations meeting current standard?
    ➢Confidence in concentration-response relationships over distributions of ambient concentrations, including concentrations below level of current standard?
➢Uncertainties in evidence for O$_3$-attriubutable effects across distributions of exposure concentrations and ambient concentrations

Exposure-/Risk-Based Considerations
➢Nature, magnitude, and importance of estimated exposures and risks associated with current O$_3$ standard
    ➢Focus on at-risk populations
➢Uncertainties in the exposure and risk estimates

Does information call into question adequacy of current 8-hour Primary O$_3$ standard?

NO → **Consider retaining current 8-hour O$_3$ standard**

↓YES

Consider Potential Alternative Standards

Indicator
➢Support for retaining O$_3$?

Averaging Time
➢Support for 8-hour only?
➢Support for longer-term averaging time?

Form
➢Support for retaining annual 4th highest?

Level
➢Evidence-based considerations : Consider controlled human exposure studies and epidemiologic studies, including uncertainties
➢Exposure- and risk-based considerations : Consider exposure and risk reductions for alternative O$_3$ standards, exposures and risks estimated to remain upon meeting alternatives, and uncertainties and limitations in exposure/risk estimates

Identify range of potential alternative standards for consideration

1

2   **Figure 1-1. Overview of approach to reviewing the primary standard.**

1.3.1.2.1 Consideration of the Scientific Evidence

Our approach in this review draws upon an integrative synthesis of the entire body of available scientific evidence for O_3-related health effects, including the evidence newly available in the current review and the evidence from previous reviews, as presented in the ISA (U.S. EPA, 2013). Our approach to considering the scientific evidence is based fundamentally on using information from controlled human exposure and epidemiologic studies, supplemented by information from animal toxicology studies. Such evidence informs our consideration of the health endpoints and at-risk populations on which to focus the current review, and our consideration of the O_3 concentrations at which various health effects can occur.

Since the 2008 review of the O_3 NAAQS, the Agency has developed formal frameworks for characterizing the strength of the scientific evidence with regard to health effects associated with exposures to O_3 in ambient air and factors that may increase risk in some populations or lifestages (U.S. EPA, 2013, Preamble; Chapter 8). These frameworks provide the basis for robust, consistent, and transparent processes for evaluating the scientific evidence, including uncertainties in the evidence, and for drawing weight-of-evidence conclusions on air pollution-related health effects and at-risk populations.

With regard to characterization of health effects, the ISA uses a five-level hierarchy to classify the overall weight-of-evidence into one of the following categories: causal relationship, likely to be a causal relationship, suggestive of a causal relationship, inadequate to infer a causal relationship, and not likely to be a causal relationship (U.S. EPA, 2013, Preamble Table II). In this PA, we place the greatest weight on the evidence for health effects that have been judged in the ISA to be caused by, or likely to be caused by, O_3 exposures. Our consideration of the available evidence for such effects is presented below in Chapter 3 (consideration of the adequacy of the current standard) and in Chapter 4 (consideration of potential alternative standards).

As discussed below, we further consider the evidence base assessed in the ISA with regard to the types and levels of exposure at which health effects are indicated. This further consideration of the evidence, which directly informs EPA's conclusions regarding the adequacy of current or potential alternative standards in providing requisite public health protection, differs from consideration of the evidence in the ISA with regard to overarching determinations of causality. Therefore, studies that inform determinations of causality may or may not be concluded to be informative with regard to the adequacy of the current or potential alternative standards.

As with health endpoints, the ISA's characterization of the weight-of-evidence for potential at-risk populations is based on the evaluation and synthesis of evidence from across scientific disciplines. The ISA characterizes the evidence for a number of "factors" that have the

1 potential to place populations at increased risk for O_3-related effects. The categories considered
2 in evaluating the evidence for these potential at-risk factors are "adequate evidence," "suggestive
3 evidence," "inadequate evidence," and "evidence of no effect." These categories are discussed
4 in more detail in the ISA (U.S. EPA, 2013, chapter 8, Table 8-1). In this draft PA, we focus our
5 consideration of potential at-risk populations on those factors for which the ISA judges there is
6 "adequate" evidence (U.S. EPA, 2013, Table 8-5). At-risk populations are discussed in more
7 detail in section 3.2.1, below.
8   Using the available scientific evidence to inform conclusions on the adequacy of the
9 current primary O_3 standard, and on potential alternative standards appropriate for consideration,
10 is complicated by the recognition that a population-level threshold in exposure or ambient O_3
11 concentrations has not been identified, below which it can be concluded with confidence that O_3-
12 attributable effects do not occur in exposed populations (U.S. EPA, 2013, section 2.5.4.4). In the
13 absence of a discernible threshold, our general approach to considering the available O_3 health
14 evidence involves characterizing our confidence in the extent to which O_3-attributable effects
15 occur, and the extent to which such effects are adverse, over the ranges of O_3 exposure
16 concentrations evaluated in controlled human exposure studies and over the distributions of
17 ambient O_3 concentrations in locations where epidemiologic studies have been conducted. As
18 noted above, we recognize that the available health effects evidence reflects a continuum from
19 relatively high O_3 concentrations, at which scientists generally agree that adverse health effects
20 are likely to occur, through lower concentrations, at which the likelihood and magnitude of a
21 response become increasingly uncertain. Aspects of our approach particular to evidence from
22 controlled human exposure and epidemiologic studies, respectively, are discussed below.
23   ***Controlled Human Exposure Studies***
24   Controlled human exposure studies provide direct evidence of relationships between
25 pollutant exposures and human health effects (U.S. EPA, 2013, p.lx). Such studies are
26 particularly useful in defining the specific conditions under which pollutant exposures can result
27 in health impacts, including the exposure concentrations, durations, and ventilation rates under
28 which effects can occur. As discussed in the ISA, controlled human exposure studies provide
29 clear and compelling evidence for an array of human health effects that are directly attributable
30 to acute exposures to O_3 *per se* (i.e., as opposed to O_3 and other photochemical oxidants, for
31 which O_3 is an indicator, or other co-occurring pollutants) (U.S. EPA, 2013, Chapter 6).
32 Together with animal toxicological studies, which can provide information about more serious
33 health outcomes as well as the effects of long-term exposures and mode of action, controlled
34 human exposure studies also help to provide biological plausibility for health effects observed in
35 epidemiologic studies.

1   In this draft PA, we consider the evidence from controlled human exposure studies in two
2   ways. First, we consider the extent to which controlled human exposure studies provide evidence
3   for health effects following exposures to different O_3 concentrations, down to the lowest-
4   observed-effects levels in those studies. Second, we use such studies to inform our evaluation of
5   the extent to which we have confidence in health effect associations reported in epidemiologic
6   studies down through lower ambient O_3 concentrations, where the likelihood and magnitude of
7   O_3-attributable effects become increasingly uncertain.

8   We consider the range of O_3 exposure concentrations evaluated in controlled human
9   exposure studies, including concentrations near or below the level of the current standard. We
10   consider both group mean responses, which provide insight into the extent to which observed
11   changes are due to O_3 exposures rather than to chance alone, and inter-individual variability in
12   responses, which provides insight into the fraction of the population that might be affected by
13   such O_3 exposures (U.S. EPA, 2013, section 6.2.1.1). When considering the relative weight to
14   place on various controlled human exposure studies, we consider the exposure conditions
15   evaluated (e.g., exercising versus resting, exposure duration); the nature, magnitude, and likely
16   adversity of effects over the range of reported O_3 exposure concentrations; the statistical
17   precision of reported effects; and the consistency of results across studies for a given health
18   endpoint and exposure concentration. In addition, because controlled human exposure studies
19   typically involve healthy individuals and do not evaluate the most sensitive individuals in the
20   population (U.S. EPA, 2013, Preamble p. lx), when considering the implications of these studies
21   for our evaluation of the current and potential alternative standards, we also consider the extent
22   to which reported effects are likely to reflect the magnitude and/or severity of effects in at-risk
23   groups.

24   *Epidemiologic Studies*
25   We also consider epidemiologic studies of short- and long-term O_3 concentrations in
26   ambient air. Epidemiologic studies provide information on associations between variability in
27   ambient O_3 concentrations and variability in various health outcomes, including lung function
28   decrements, respiratory symptoms, school absences, hospital admissions, emergency department
29   visits, and premature mortality (U.S. EPA, 2013, Chapters 6 and 7). Epidemiologic studies can
30   inform our understanding of the effects in the study population (which may include at-risk
31   groups) of real-world exposures to the range of O_3 concentrations in ambient air.

32   Available studies have generally not indicated a discernible population threshold, below
33   which O_3 is no longer associated with health effects (U.S. EPA, section 2.5.4.4). However, the
34   currently available epidemiologic evidence indicates decreased confidence in reported
35   concentration-response relationships for O_3 concentrations at the lower ends of ambient
36   distributions (U.S. EPA, section 2.5.4.4). Therefore, our general approach to considering the

1 results of epidemiologic studies within the context of the current and potential alternative
2 standards focuses on characterizing the range of ambient O_3 concentrations over which we have
3 the most confidence in O_3-associated health effects, and the concentrations below which our
4 confidence in such health effect associations becomes appreciably lower. In doing so, we
5 consider the statistical precision of O_3 health effect associations reported in study locations with
6 various ambient O_3 concentrations; confidence intervals around concentration-response functions
7 reported over distributions of ambient O_3 (where available); and the extent to which the
8 biological plausibility of associations at various ambient O_3 concentrations is supported by
9 evidence from controlled human exposure and/or animal toxicological studies.
10   We consider both multi-city and single-city studies assessed in the ISA, each of which
11 have strengths and limitations. Multi-city studies evaluate large populations and provide greater
12 statistical power than single-city studies. Multi-city studies also reflect O_3-associated health
13 impacts across a range of diverse locations, providing spatial coverage for different regions
14 across the country and reflecting differences in exposure-related factors that could impact O_3
15 risks. In addition, compared to single-city studies, multi-city studies are not prone to publication
16 bias and they afford the possibility of generalizing to the broader national population (U.S. EPA,
17 2004, p. 8-30). In contrast, while single-city studies are more limited than multicity studies in
18 terms of statistical power and geographic coverage, conclusions regarding the extent to which air
19 quality met the current or potential alternative standards in the cities for which associations have
20 been reported can be made with greater certainty for single-city studies (compared to multicity
21 studies reporting only multicity effect estimates) because the associations are reported for city-
22 specific analyses (U.S. EPA, 2011d, section 2.3.4.1).[19] In some cases, single-city studies can
23 also provide evidence for locations or population-specific characteristics not reflected in
24 multicity studies (U.S. EPA, 2013, section 6.2.7.1). Therefore, when considering available
25 epidemiologic studies we evaluate both multi-city and single-city studies, recognizing the
26 strengths and limitations of each.
27   In placing emphasis on specific epidemiologic studies, we focus on studies conducted in
28 the U.S. and Canada. Such studies reflect air quality and exposure patterns that are likely more
29 typical of the U.S. population than the air quality and exposure patterns reflected in studies
30 conducted outside the U.S. and Canada.[20] We also focus on studies reporting associations with
31 effects judged in the ISA to be robust to confounding by other factors, including co-occurring air
32 pollutants.

[19] Though in some cases multicity studies present single-city effect estimates in addition to multi-city estimates.

[20] All studies, including other international studies inform the causal determinations in the ISA.

1   **1.3.1.2.2   Consideration of Exposure and Risk Estimates**

2   To put judgments about O_3-related health effects into a broader public health context, we

3   consider exposure and risk estimates from the second draft HREA, which develops and applies

4   models to estimate human exposures to O_3 and O_3-related health risks in urban case study areas

5   across the United States (U.S. EPA, 2014). The second draft HREA estimates exposures of

6   concern, based on interpreting quantitative exposure estimates within the context of controlled

7   human exposure study results; lung function risks, based on applying exposure-response

8   relationships from controlled human exposure studies to quantitative estimates of exposures; and

9   epidemiologic-based risk estimates, based on applying concentration-response relationships

10   drawn from epidemiologic studies to adjusted air quality. Each of these types of assessments is

11   discussed briefly below.

12   As in the 2008 review, the second draft HREA estimates exposures at or above

13   benchmark concentrations of 60, 70, and 80 ppb, reflecting exposure concentrations of concern

14   based on the available health evidence.[21] Estimates of exposures at or above discrete benchmark

15   concentrations provide perspective on the public health risks of O_3-related health effects that

16   have been demonstrated in controlled human exposure and toxicological studies but that, because

17   of a lack of exposure-response information from those studies, cannot be assessed using a

18   quantitative risk assessment. Though this analysis is conducted using discrete benchmark

19   concentrations, health-relevant exposures are more appropriately viewed as a continuum with

20   greater confidence and less uncertainty about the existence of health effects at higher O_3

21   exposure concentrations and less confidence and greater uncertainty at lower exposure

22   concentrations. We recognize that there is no sharp breakpoint within the exposure-response

23   relationship for exposure concentrations at and above 80 ppb down to 60 ppb.

24   The second draft HREA also generates quantitative estimates of O_3 health risks for air

25   quality adjusted from recent conditions to those just meeting the current and potential alternative

26   standards. As noted above, one approach to estimating O_3 health risks is to combine modeled

27   exposure estimates with exposure-response relationships derived from controlled human

28   exposure studies of O_3-induced health effects. The second draft HREA uses this approach to

29   estimate the occurrence of O_3-induced lung function decrements in simulated at-risk populations.

30   The available exposure-response information does not support this approach for other endpoints

31   evaluated in controlled human exposure studies (U.S. EPA, 2014a, section 2.3).

32   Another approach to estimating O_3-associated health risks is to apply concentration-

33   response relationships derived from short- and/or long-term epidemiologic studies to air quality

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[21] For example, see 75 FR 2945-2946 (January 19, 2010) and 73 FR 16441-16442 (March 27, 2008) discussing "exposures of concern".

1   adjusted to just meet current and potential alternative standards. The concentration-response

2   relationships drawn from epidemiologic studies are based on population exposure surrogates,

3   such as 8-hour concentrations averaged across monitors and over more than one day

4   (incorporation of lag) (U.S. EPA, 2013, Chapter 6). The second draft HREA presents

5   epidemiologic-based risk estimates for O_3-associated mortality, hospital admissions, emergency

6   department visits, and respiratory symptoms (U.S. EPA, 2014a, section 2.3). These estimates are

7   derived from the full distribution of ambient O_3 concentrations estimated for the study

8   locations.[22] In addition, the second draft HREA estimates mortality risks attributable to various

9   portions of those distributions (U.S. EPA, 2014a). In this second draft PA we consider risk

10   estimates based on the full distributions of ambient O_3 concentrations, and estimates of the risk

11   associated with various portions of those ambient distributions. In doing so, we take note of the

12   ISA conclusions regarding confidence in linear concentration-response relationships over

13   distributions of ambient concentrations, and of the extent to which health effect associations at

14   various ambient O_3 concentrations are supported by the evidence from experimental studies for

15   effects following specific O_3 exposures.

16   **1.3.1.2.3   Considerations Regarding Ambient O_3 Concentration Estimates**
17   **Attributable to Background Sources**

18   As noted above, our approach in this review utilizes recent advances in modeling

19   techniques to estimate the contributions of U.S. anthropogenic, international anthropogenic, and

20   natural sources to ambient O_3 (discussed in detail in Chapter 2 of this draft PA). Such model

21   estimates can provide insights into the extent to which different types of background emissions

22   sources contribute to total ambient O_3 concentrations. Consideration of this issue in the current

23   review is informed by the approaches taken in previous reviews, as well as by court decisions.

24   In 1979, the EPA set a 1-hour O_3 standard with a level of 0.12 ppm. Following the final

25   decision in that review, the City of Houston argued that the standard was arbitrary and capricious

26   because natural O_3 concentrations and other physical phenomena in the Houston area made the

27   standard unattainable in that area. The D.C. Circuit rejected this argument, stating that

28   attainability and technological feasibility are not relevant considerations in the promulgation of

29   the NAAQS. The Court also noted that the EPA need not tailor the NAAQS to fit each region or

30   locale, pointing out that Congress was aware of the difficulty in meeting standards in some

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[22] In previous reviews, including the 2008 review and reconsideration, such risks were separately estimated for O_3 concentrations characterized as above policy-relevant background concentrations. Policy-relevant background concentrations were defined as the distribution of ozone concentrations attributable to sources other than anthropogenic emissions of ozone precursor emissions (e.g., VOC, CO, NOx) in the U.S., Canada, and Mexico. The decision to estimate total risk across the full range of O_3 concentrations reflects current OAQPS views and consideration of advice from CASAC (Frey and Samet, 2012b).

1   locations and had addressed this difficulty through various compliance related provisions in the

2   Act. See API v. Costle, 665 F.2d 1176, 1184-6 (D.C. Cir. 1981).

3   More recently, in the 1997 review of the O_3 NAAQS, the Administrator set an 8-hour

4   standard with a level of 0.08 ppm (84 ppb). In reaching this decision, the EPA identified several

5   reasons supporting its decision to reject a more stringent standard of 0.07 ppm. Most

6   importantly, the EPA pointed out the scientific uncertainty at lower concentrations and placed

7   significant weight on the fact that no CASAC panel member supported a standard level set lower

8   than 0.08 ppm (62 FR 38868). In addition to noting the uncertainties in the health evidence for

9   exposure concentrations below 0.08 ppm and the advice of CASAC, the EPA noted that a

10 standard set at a level of 0.07 ppm would be closer to peak background concentrations that

11 infrequently occur in some areas due to nonanthropogenic sources of O_3 precursors (62 FR

12 38856, 38868; July 18, 1997).

13   In subsequent litigation, the D.C. Circuit upheld the EPA's decision as the product of

14 reasoned decision-making. The Court made clear that the most important support for the EPA's

15 decision was the health evidence and the concerns it raised about setting a standard level below

16 0.08 ppm. The Court also pointed to the significant weight that the EPA properly placed on the

17 advice it received from CASAC. Finally (as noted in section 1.2.2 above), the Court noted that

18 the EPA could also consider relative proximity to peak natural background O_3 when evaluating

19 alternative standards. See ATA III, 283 F.3d at 379 (D.C. Cir. 2002).

20   These cases provide a framework for considering the contributions of U.S.

21 anthropogenic, international anthropogenic, and natural sources, within the context of

22 considering the health evidence and CASAC advice, when evaluating various potential

23 alternative standards.

24 **1.3.2   Approach for the Secondary Standard**

25   Staff's approach in this review of the current secondary standard takes into consideration

26 aspects of the approaches used in past O_3 NAAQS reviews. The past and current approaches,

27 generally described below, are both based fundamentally on using EPA's assessment of the

28 current scientific evidence and associated quantitative analyses to inform the Administrator's

29 judgment regarding a secondary standard for O_3 that is requisite (i.e., neither more nor less

30 stringent than necessary) to protect public welfare.

31   In reaching conclusions on options for the Administrator's consideration, we note that the

32 final decision to retain or revise the current secondary O_3 standard is a public welfare policy

33 judgment to be made by the Administrator. This final decision will draw upon the available

34 scientific evidence for O_3-attributable welfare effects and on analyses of vegetation and

35 ecosystem exposures and public welfare risks based on impacts to vegetation, ecosystems and

1 their associated services, including judgments about the appropriate weight to place on the range

2 of uncertainties inherent in the evidence and analyses. In determining the requisite level of

3 protection for crops and trees, the Administrator will need to weigh the importance of the

4 predicted risks of these effects in the overall context of public welfare protection, along with a

5 determination as to the appropriate weight to place on the associated uncertainties and limitations

6 of this information. Our general approach to informing these judgments, discussed more fully

7 below, recognizes that the available welfare effects evidence reflects a continuum from relatively

8 high O_3 concentrations at which scientists generally agree that welfare effects are likely to occur,

9 through lower concentrations at which the likelihood and magnitude of a response become

10 increasingly uncertain. Therefore, in developing conclusions in this second draft PA, we are

11 mindful that the Administrator's ultimate judgments on the secondary standard will most

12 appropriately reflect an interpretation of the available scientific evidence and exposure/risk

13 information that neither overstates nor understates the strengths and limitations of that evidence

14 and information.

15   Section 1.3.2.1 below provides an overview of the general approach taken in the last

16 review of the secondary standard for O_3 (i.e., the 2008 review), and a summary of the rationale

17 for the decision on the standard in that review (73 FR 16436). Section 1.3.2.2 presents our

18 approach in the current review, including our approach to considering the vegetation effects

19 evidence and exposure/risk information, and considerations regarding ambient O_3 concentrations

20 attributable to background sources.

21 **1.3.2.1   Approach Used in the Last Review**

22   In the 2008 review of the secondary NAAQS for O_3, the Administrator relied upon

23 consideration of the available scientific evidence and exposure/risk information, information

24 regarding biologically-relevant exposure indices, air quality information regarding the degree of

25 overlap between different exposure index forms, the advice and recommendations of CASAC,

26 considerations regarding adversity, and comments from the public. Based on all of this, he

27 revised the level of the secondary O_3 standard from 0.08 ppm[23] to 0.075 ppm (75 ppb[24]).

28   In reaching a decision to revise the 1997 8-hour secondary standard, the Administrator

29 found, after carefully considering the public comments, that the fundamental scientific

30 conclusions on the effects of O_3 on vegetation and sensitive ecosystems reached in the 2006

31 Criteria Document and 2007 Staff Paper, as discussed in section IV.A of the final rule remained

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[23] Due to rounding convention, the 1997 standard level of 0.08 ppm corresponded to 0.084 ppm (84 ppb).

[24] The level of the O_3 standard is specified as 0.075 ppm rather than 75 ppb. However, in this draft PA we refer to ppb, which is most often used in the scientific literature and in the ISA, n order to avoid the confusion that could result from switching units when discussing the evidence in relation to the standard level.

1   valid (73 FR 16496). He further recognized that several additional lines of evidence had
2   progressed sufficiently since the 1997 review to provide a more complete and coherent picture of
3   the scope of O_3-related vegetation risks (i.e., visible foliar injury, tree biomass loss, crop yield
4   loss, and others), especially those faced by sensitive seedling, sapling and mature growth stage
5   tree species growing in field settings, and their associated forested ecosystems. This new
6   research reflected an increased emphasis on field-based exposure methods (e.g., free-air, ambient
7   gradient and biomonitoring surveys) (73 FR 16490) in addition to the more traditional controlled
8   open-top chamber (OTC) studies (73 FR 16485), and began to address one of the key data gaps
9   cited by the Administrator in the 1997 review (73 FR 16486). Specifically, by providing
10 additional evidence that O_3-induced crop yield loss and tree seedling biomass loss effects
11 observed in chambers also occurs in the field, this new research qualitatively increased support
12 for, and confidence in, the continued use of OTC-derived crop and tree seedling concentration-
13 response (C-R) functions developed in the National Crop Loss Assessment Network (NCLAN)
14 and National Health and Environmental Effects Research Laboratory – Western Ecology
15 Division (NHEERL-WED) studies, respectively, to predict O_3-induced impacts on crops and tree
16 seedlings in the field (72 FR 37886). All of these areas were considered together, along with
17 associated uncertainties, in an integrated weight-of-evidence approach (73 FR 16490).
18   Beyond the available vegetation effects evidence, the Administrator also considered
19 estimates of O_3 exposures and risks when air quality was adjusted to simulate just meeting the
20 existing and potential alternative standards. On the basis of these assessments, the Administrator
21 concluded that O_3 exposures that would be expected to remain after meeting the existing
22 standard would be sufficient to cause visible foliar injury and seedling and mature tree biomass
23 loss in O_3-sensitive vegetation (73 FR 16496) and would still allow O_3-related yield loss to occur
24 in some commodity crop species and fruit and vegetable species grown in the U.S. (73 FR
25 16489). Other O_3-induced effects described in the literature, including an impaired ability of
26 many sensitive species and genotypes within species to adapt to or withstand other
27 environmental stresses, such as freezing temperatures, pest infestations and/or disease, and to
28 compete for available resources, would also be anticipated to occur. In the long run, the result of
29 these impairments (e.g., loss in vigor) could lead to premature plant death in O_3 sensitive species.
30 Though effects on other ecosystem components had only been examined in isolated cases, the
31 Administrator noted effects such as those described above could have significant implications for
32 plant community and associated species biodiversity and the structure and function of whole
33 ecosystems (73 FR 16496).
34   Although the Administrator concluded that the then-current standard was not sufficient to
35 protect against the known and anticipated effects described above, he also recognized that the
36 secondary standard is not meant to protect against all known observed or anticipated O_3-related

1   effects, but only those that can reasonably be judged to be adverse to the public welfare. The

2   Administrator recognized that the degree to which such effects should be considered to be

3   adverse depended on the intended use of the vegetation and its significance to the public welfare

4   (73 FR 16496). In this regard, he took note of a number of actions taken by Congress to establish

5   public lands that are set aside for specific uses that are intended to provide benefits to the public

6   welfare, including lands that are to be protected so as to conserve the scenic value and the natural

7   vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment of

8   future generations. Based on these considerations, and taking into consideration the advice and

9   recommendations of CASAC, the Administrator concluded that the protection afforded by the

10   existing standard was not sufficient, and that the standard needed to be revised to provide

11   additional protection from known and anticipated adverse effects on sensitive natural vegetation

12   and ecosystems (73 FR 16497).

13   Given his judgment on the need to revise, the Administrator then considered what

14   revisions to the standard were requisite to protect public welfare. Regarding the form of the

15   standard, the Administrator took note that at the conclusion of the 1997 review, the biological

16   basis for a cumulative, seasonal form was not in dispute[25] and that the 2006 Criteria Document

17   also concluded that O_3 exposure indices that cumulate differentially-weighted hourly

18   concentrations are the best candidates for relating exposure to plant growth responses (EPA,

19   2006) (61 FR 65716; 73 FR 16486). The CASAC, in its letter to the Administrator following its

20   review of the second draft Staff Paper, stated that "there is a clear need for a secondary standard

21   which is distinctly different from the primary standard in averaging time, level and form" and

22   that "the CASAC unanimously agrees that it is not appropriate to try to protect vegetation from

23   the substantial, known or anticipated, direct and/or indirect, adverse effects of ambient ozone by

24   continuing to promulgate identical primary and secondary standards for ozone" (Henderson,

25   October 24, 2006, pp. 5-7). Although many possible cumulative, seasonal concentration-

26   weighted exposure metrics exist, the Staff Paper and the CASAC Panel concluded that the

27   W126[26] form is the most biologically-relevant cumulative, seasonal form appropriate to consider

28   in the context of the secondary standard review (73 FR 16486-87).[27]

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[25] In the 1997 review, a different cumulative metric (SUM06) was proposed. Metric selection in both 1997 and 2008 was based on both science and policy considerations.

[26] W126 is a cumulative exposure index that is biologically based. The W126 index focuses on the higher hourly average concentrations, while retaining the mid-and lower-level values. It is defined as the sum of sigmoidally weighted hourly O_3 concentrations over a specified period, where the daily sigmoidal weighting function is defined as: $1 - \exp[-(W126/\eta)^\beta]$

[27] In a subsequent letter offering unsolicited advice to the Administrator and Agency staff on development of the proposed rulemaking, the CASAC reiterated that Panel members ''were unanimous in supporting the recommendation in the Final Ozone Staff Paper that protection of managed agricultural crops and natural terrestrial ecosystems requires a secondary Ozone NAAQS that is substantially different from the primary ozone standard in

1   Although agreeing with the Criteria Document, Staff Paper and CASAC conclusions that
2   a cumulative exposure index that differentially weights O_3 concentrations could represent a
3   reasonable policy choice for a seasonal secondary standard to protect against the effects of O_3 on
4   vegetation and that the most appropriate cumulative, concentration-weighted form to consider
5   was the sigmoidally weighted W126 form (73 FR 16498), the Administrator also took note of the
6   1997 decision to make the revised secondary standard identical to a revised primary standard
7   after similar considerations (73 FR 16498). In considering the rationale for the 1997 decision, the
8   Administrator observed that it was based in part on an analysis that compared the degree of
9   overlap in county-level air quality measured in terms of alternative standard forms (62 FR
10   38876). Recognizing that significant uncertainty remained in 1997 regarding conclusions drawn
11   from such analyses, the Administrator also considered the results of a similar analysis of recent
12   monitoring data undertaken in the 2007 Staff Paper to assess the degree of overlap expected
13   between the existing standard (4th high, daily maximum 8-hour concentration averaged over
14   three years) and potential alternative standards based on W126 cumulative seasonal forms.
15   The Administrator noted that this analysis showed significant overlap between the 8-hour
16   secondary standard and selected levels of W126 standard forms, with the degree of overlap
17   between these potential alternative standards depending greatly on the W126 level selected and
18   the distribution of hourly O_3 concentrations within the annual and/or 3-year average period.
19   From this analysis, the Administrator recognized that a secondary standard set identical to a
20   revised primary standard would provide a significant degree of additional protection for
21   vegetation as compared to that provided by the existing secondary standard. In further
22   considering the significant uncertainties in the available body of evidence and in the exposure
23   and risk analyses, and the difficulty in determining at what point various types of vegetation
24   effects become adverse for sensitive vegetation and ecosystems, the Administrator focused his
25   consideration on a level for an alternative W126 standard (with an annual form) at the upper end
26   of the proposed range (i.e., 21 ppm-hours). The Staff Paper analysis showed that at a W126 level
27   of 21 ppm-hours, there would be essentially no counties with air quality expected both to exceed
28   such an alternative W126 standard and to meet the revised 8-hour primary standard—that is,
29   based on this analysis of counties with ambient O_3 monitors, a W126-based level of 21 ppm-
30   hours would be unlikely to provide additional protection in any areas beyond that likely to be
31   provided by the revised 2008 primary standard (73 FR 16499/500).

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averaging time, level and form"...and "[t]he recommended metric for the secondary ozone standard is the
(sigmoidally-weighted) W126 index, accumulated over at least the 12 'daylight' hours and over at least the three
maximum ozone months of the summer 'growing season" (Henderson, March 26, 2007, p.3).

1   The Administrator also considered the Staff Paper finding that the degree of overlap

2   between counties (with areas of concern for vegetation) expected to meet an 8-hour level for the

3   form of the existing standard and potential alternative levels of a W126-based standard was

4   inconsistent across years analyzed. This variation depended greatly on levels selected for a

5   W126-based standard and a 3-year average 4th high daily maximum 8-hour standard,

6   respectively, and the distribution of hourly O_3 concentrations within the annual and/or 3-year

7   average period. From this, the Staff Paper recognized the need for caution in evaluating the

8   likely vegetation impacts associated with a given level of air quality expressed in terms of the

9   existing 8-hour average standard in the absence of parallel W126 information. In considering

10   these findings, the Administrator "recognize[d] that the general lack of rural monitoring data

11   made uncertain the degree to which the revised 8-hour standard or an alternative W126 standard

12   would be protective, and that there was the potential for not providing the appropriate degree of

13   protection for vegetation in areas with air quality distributions that resulted in a high cumulative,

14   seasonal exposure but did not result in high 8-hour average exposures" (73 FR 16500). With

15   regard to the 8-hour standard, he also noted that "[w]hile this potential for under-protection was

16   clear, the number and size of areas [then] at issue and the degree of risk [was] hard to determine.

17   However, such a standard would also tend to avoid the potential for providing more protection

18   than is necessary, a risk that would have arisen from moving to a new form for the secondary

19   standard despite the significant uncertainty in determining the degree of risk for any exposure

20   level and the appropriate level of protection, as well as uncertainty in predicting exposure and

21   risk patterns" (73 FR 16500).

22   Thus, although the Administrator agreed with the views and recommendations of

23   CASAC that a cumulative, seasonal standard was the most biologically relevant way to relate

24   exposure to plant growth response, he also recognized that there remained significant

25   uncertainties in determining or quantifying the degree of risk attributable to varying levels of O_3

26   exposure, the degree of protection that any specific cumulative, seasonal standard would

27   produce, and the associated potential for error in determining the secondary standard that would

28   provide a requisite degree of protection—i.e., sufficient but not more than what is necessary.

29   Given these significant uncertainties, the Administrator concluded that establishing a new

30   secondary standard with a cumulative, seasonal form, at that time, would have resulted in

31   uncertain benefits beyond those afforded by the revised primary standard, and therefore, might

32   have been more than necessary to provide the requisite degree of protection (73 FR 16500).

33   Based on his consideration of these issues (73 FR 16497), the Administrator judged that the

34   appropriate balance to be drawn was to set a secondary standard identical in every way to the

35   revised 8-hour primary standard of 0.075 ppm. The Administrator believed that such a standard

36   would be sufficient to protect public welfare from known or anticipated adverse effects, and did

1   not believe that an alternative cumulative, seasonal standard was needed to provide this degree of

2   protection (73 FR 16500).

3   As noted above, on July 23, 2013 the D.C. Circuit found this approach to be contrary to

4   law because EPA had failed to identify a level of air quality requisite to protect public welfare

5   and, therefore, EPA's comparison between the primary and secondary standards for determining

6   if requisite protection for public welfare was afforded by the primary standard was inherently

7   arbitrary. The court remanded the secondary standard to EPA for further consideration. 723 F. 3d

8   at 270-74.

9   **1.3.2.2   Approach for the Current Review**

10   To identify the range of options appropriate for the Administrator to consider in the

11   current review, we apply an approach that builds upon the general approach used in the 2008

12   review(and in the 2010 reconsideration proposal), and that reflects the broader body of scientific

13   evidence, updated exposure/risk information, and advances in O_3 air quality modeling now

14   available. As summarized above, the Administrator's decisions in the prior review were based on

15   an integration of information on welfare effects associated with exposure to O_3, judgments on the

16   adversity and public welfare significance of key effects, and, expert and policy judgments as to

17   when the standard is requisite to protect public welfare. These considerations were informed by

18   air quality and related analyses, quantitative exposure and risk assessments, and qualitative

19   assessment of impacts that could not be quantified. In performing the evaluation in this

20   document, we are additionally mindful of the recent remand of the secondary standard by the

21   D.C. Circuit.

22   Our approach in this review of the secondary O_3 standard reflects our consideration of the

23   available scientific evidence, information on biologically-relevant exposure indices,

24   exposure/risk information, and air quality modeling information, within the context of

25   overarching questions related to: (1) the adequacy of the current secondary O_3 standard to protect

26   against effects associated with cumulative, seasonal exposures and (2) potential alternative

27   standards, if any, that are appropriate to consider in this review. In addressing these broad

28   questions, we have organized the discussions in chapters 5 and 6 of this document around a

29   series of more specific questions reflecting different aspects of each overarching question. When

30   evaluating the welfare protection afforded by the current or potential alternative standards, we

31   take into account the four basic elements of the NAAQS: the indicator, averaging time, form,

32   and level.

33   Figure 1-2 below provides an overview of our approach in this review. We believe that

34   the general approach summarized in this section, and outlined in Figure 1-2, provides a

35   comprehensive basis to help inform the judgments required of the Administrator in reaching

1   decisions about the current and potential alternative secondary O_3 standards. In the subsections

2   below, we summarize our general approaches to considering the scientific evidence (evidence-

3   based considerations) and to considering the exposure and risk information (exposure- and risk-

4   based considerations). We also recognize considerations related to ambient O_3 attributable to

5   background sources.

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┌─────────────────────────────────────────────────────────┐
│         Adequacy of Current 8-Hour Secondary O₃ Standard  │
└─────────────────────────────────────────────────────────┘
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Evidence-Based Considerations
- ISA weight-of-evidence conclusions
- Evidence for vegetation effects from cumulative exposures allowed by the current standard (OTC)?
- Evidence for vegetation effects in locations that would likely have met the current standard (field-based)?

Exposure-/Risk-Based Considerations
- Nature, magnitude, and importance of estimated exposures and risks associated with current O₃ standard
- Risks attributable to U.S. anthropogenic, international, and natural sources
- Uncertainties in the exposure and risk estimates?

Does information call into question the adequacy and appropriateness of current 8-hour secondary O₃ standard?

NO → Consider retaining current 8-hour O₃ standard

YES

Consider Potential Alternative Standards

Indicator
- Support for retaining O₃?

Form
- Support for cumulative, seasonal form?
- Support for concentration-weighted form?
- Support for single vs. 3-year period?

Averaging Time
- Support for 12 hour diurnal period?
- Support for 3-month seasonal period?

Level
- Evidence-based considerations
  - O₃ exposure concentrations from OTC and field-based studies
  - Uncertainties in the evidence
- Exposure and risk considerations
  - What is the nature, magnitude, and importance of estimated exposures and risks associated with potential alternative O₃ standards?
  - Uncertainties in the exposure/ risk assessment?

Identify range of potential alternative standards for consideration

1

Figure 1-2.   Overview of approach to reviewing the secondary standard.

2

1.3.2.2.1   Consideration of the Scientific Evidence

Our approach in this review draws upon an integrative synthesis of the entire body of available scientific evidence for O_3-related welfare effects, including the evidence newly available in the current review and the evidence from previous review, as presented in the ISA (U.S. EPA, 2013). Our approach to considering the scientific evidence for effects on vegetation is based fundamentally on using information from controlled chamber studies and field-based studies. Such evidence informs our consideration of welfare endpoints and at-risk species and ecosystems on which to focus the current review, and our consideration of the ambient O_3 conditions under which various welfare effects can occur.

As in each NAAQS review, we consider the entire body of evidence for the subject criteria pollutant. With regard to identification of the welfare effects that could be caused by a pollutant, we look to controlled exposure studies using chamber or free air methodologies and field-based observational, survey and gradient studies. Evaluating all of the evidence together, the ISA makes a determination with regard to the strength of the evidence for a causal relationship between the air pollutant and specific welfare effects. These determinations inform our identification of welfare effects for which the NAAQS may provide protection.

Since the 2008 review of the O_3 NAAQS, the Agency has developed a formal framework for characterizing the strength of the scientific evidence with regard to a causal relationship between ambient O_3 and welfare effects (U.S. EPA, 2013, Preamble; Chapter 9). This framework provides the basis for a robust, consistent, and transparent process for evaluating the scientific evidence, including uncertainties in the evidence, and for drawing weight-of-evidence conclusions regarding air pollution-related welfare effects. In so doing, the ISA uses a five-level hierarchy, classifying the overall weight of evidence into one of the following categories: causal relationship, likely to be a causal relationship, suggestive of a causal relationship, inadequate to infer a causal relationship, and not likely to be a causal relationship (U.S. EPA, 2013, Preamble Table II). In our approach here, we place the greatest weight on the evidence for welfare effects that have been judged in the ISA to be caused by, or likely caused by, O_3 exposures. Our consideration of the available evidence for such effects is presented below in Chapter 5 (consideration of the adequacy of the current standard) and in Chapter 6 (consideration of potential alternative standards).

We further consider the evidence base, as assessed in the ISA, with regard to the types and levels of exposure at which welfare effects are indicated. This further consideration of the evidence base, which directly informs EPA's conclusions regarding the adequacy of current or potential alternative standards in providing requisite public welfare protection, differs from consideration of the evidenc in the ISA with regard to overarching determinations of causality.

1   Studies that have informed determinations of causality may or may not be concluded to be

2   informative with regard to the adequacy of the current or potential alternative standards.

3   Our approach in this review, as in past reviews, included recognition that the available

4   evidence has not provided identification of a threshold in exposure or ambient O_3 concentrations

5   below which it can be concluded with confidence that O_3-attributable vegetation effects do not

6   occur across the broad range of O_3-sensitive plant species growing within the U.S. This is due in

7   part to the fact that research shows that there is variability in sensitivity between and within

8   species and that numerous factors, i.e. chemical, physical, biological, and genetic, can influence

9   the direction and magnitude of the studied effect (U.S. EPA, 2013, section 9.4.8). In the absence

10   of a discernible threshold, our general approach to considering the available O_3 welfare evidence

11   involves characterizing our confidence in conclusions regarding O_3-attributable vegetation

12   effects over the ranges of cumulative seasonal O_3 exposure values evaluated in chamber studies

13   and in field studies in areas where O_3-sensitive vegetation are known to occur, as well as

14   characterizing the extent to which these effects can be considered adverse. In addition, because

15   O_3 can indirectly affect other ecosystem components (such as soils, water, and wildlife, and their

16   associated goods and services, through its effects on vegetation) our approach also considers

17   those indirect effects for which the ISA concludes, based on multiple lines of evidence, including

18   mechanistic and physiological processes, to have a causal or likely to be a causal relationship.

19   With respect to ecosystem services for which we may have only limited or qualitative

20   information regarding an association with O_3 exposures, our approach is to consider their policy-

21   relevance in the context of section 109(b)(2) of the CAA which specifies that secondary

22   standards provide requisite protection of "public welfare from any … known or anticipated

23   adverse effects associated with the presence of [the] pollutant in the ambient air". As noted

24   above, our approach recognizes that the effects evidence reflects a general continuum from

25   higher O_3 concentrations, at which scientists generally agree that adverse vegetation and

26   ecosystem effects are likely to occur, through lower concentrations, at which the likelihood and

27   magnitude of a response becomes increasingly more uncertain.

28   In this review, the evidence base includes quantitative information across a broad array of

29   vegetation effects (e.g., growth impairment during seedlings, saplings and mature tree growth

30   stages, visible foliar injury, and yield loss in annual crops) and across a diverse set of exposure

31   methods from laboratory and field studies. These methods include the more traditional OTC

32   studies, as well as field-based exposure studies. While we consider the full breadth of

33   information available, we place greater weight on U.S. studies due to the often species-, site-,

34   and climate-specific nature of O_3-related vegetation responses. We especially weight those

35   studies that include O_3 exposures that fall within the range of those likely to occur in the ambient

36   air. Further, our approach in the context of the quantitative exposure and risk assessments

1   (discussed below), places greatest emphasis on studies that have evaluated plant response over

2   multiple exposure levels and developed exposure-response relationships that allow the prediction

3   (estimation) of plant responses over the range of potential alternative standards being assessed.

4   In considering the evidence, we recognize differences across different study types in what

5   information they provide. For example, because conditions can be controlled in laboratory

6   studies, responses in such studies may be less variable and smaller differences may be easier to

7   detect. However, the control conditions may limit the range of responses or incompletely reflect

8   pollutant bioavailability, so they may not reflect responses that would occur in the natural

9   environment. Alternatively, field data can provide important information for assessments of

10 multiple stressors or where site-specific factors significantly influence exposure. They are also

11 often useful for analyses of larger geographic scales and higher levels of biological organization.

12 However, because most field study conditions can not be controlled, variability is expected to be

13 higher and differences harder to detect. The presence of confounding factors can also make it

14 difficult to attribute observed effects to specific stressors.

15   In considering information from across multiple lines of evidence, our approach is to first

16 integrate the evidence from both controlled and field-based studies and assess the coherence and

17 consistency across the available evidence for each effect. We then consider the extent to which

18 these identified effects should be considered adverse to the public welfare, relying largely on the

19 paradigm used in the 2008 review and 2010 proposed reconsideration (e.g., 75 FR 3006). This

20 paradigm recognizes that the significance to the public welfare of O_3-induced effects on sensitive

21 vegetation growing within the U.S. can vary depending on the nature of the effect, the intended

22 use of the sensitive plants or ecosystems, and the types of environments in which the sensitive

23 vegetation and ecosystems are located. Accordingly, any given O_3-related effect on vegetation

24 and ecosystems (e.g., biomass loss, crop yield loss, foliar injury) may be judged to have a

25 different degree of impact on the public welfare depending, for example, on whether that effect

26 occurs in a Class I area, a city park, or commercial cropland. Our approach takes this variation in

27 the significance of O_3-related vegetation effects into account in evaluating the currently available

28 evidence with regard to the extent to which it calls into question the adequacy of the current

29 standard and, as appropriate, indicates potential alternative standards that would be appropriate

30 for the Administrator to consider. In the 2010 proposed reconsideration, the Administrator

31 proposed to place the highest priority and significance on vegetation and ecosystem effects to

32 sensitive species that are known to or are likely to occur in federally protected areas such as

33 national parks and other Class I areas, or on lands set aside by States, Tribes and public interest

34 groups to provide similar benefits to the public welfare (75 FR 3023/24). Our approach in this

35 review considers whether newly available information would suggest any evolution to this

36 paradigm, in particular in the context of considering associated ecosystem services.

1   Finally, our approach continues to give great weight to the scientific evidence available in
2   this and previous reviews indicating the relevance of cumulative, seasonal, concentration-
3   weighted exposures in inducing vegetation effects. Therefore, we continue to express exposures
4   in terms of the W126 index, and continue to consider the important policy implications regarding
5   selection of an appropriate exposure index for vegetation. Our approach also places primary
6   emphasis on studies that evaluated plant response to exposures that were or can be described
7   using such an index. The policy-relevant discussions in chapters 5 and 6 focus on vegetation
8   effects evidence and exposure/risk information that can be associated with cumulative, seasonal
9   peak-weighted exposures, where possible. Discussions pertaining to the adequacy of the current
10   secondary standard will consider what cumulative seasonal exposures would be allowed under
11   air quality that would just meet the current standard.

12   **1.3.2.2.2   Consideration of Exposure and Risk Estimates and Air Quality Analyses**

13   To put judgments about O_3-related vegetation and ecosystem effects and services into a
14   broader public welfare context, we consider national scale exposure and risk assessments
15   described in the second draft WREA (U.S. EPA, 2014b). We particularly focused on the WREA
16   quantitative risks related to three types of vegetation effects: foliar injury, biomass loss, and crop
17   yield loss. These risks were assessed in a range of WREA analyses variously involving recent O_3
18   monitoring data and/or national-scale model-adjusted air quality scenarios for the current
19   secondary standard and, in some analyses, for a cumulative, seasonal W126 form at one or more
20   levels (15, 11 and 7 ppm-hours). Our consideration of these WREA results provide insight into
21   the extent to which the current or potential alternative standards would be expected to maintain
22   distributions of cumulative, seasonal O3 exposures below those associated with adverse
23   vegetation effects.
24   With regard to quantitative O_3 risks related to welfare effects and ecosystem services for
25   foliar injury, we consider two main analyses in the WREA: a screening-level assessment of 214
26   National Parks and a case study focused on three National Parks. In the screening-level
27   assessment, O_3 concentrations in national parks are assessed using criteria developed from a U.S.
28   Forest Service nationwide dataset on foliar injury, ambient O_3 concentrations (in terms of W126
29   index) and soil moisture (which can influence susceptibility of vegetation to foliar injury).
30   Additionally, we consider a case study for Class I areas (Great Smoky Mountain National Park,
31   Rocky Mountain National Park, and Sequoia/Kings Canyon National Park). We consider results
32   from this case study for three metrics: 1) percent of vegetation cover affected by foliar injury; 2)
33   percent of trails affected by foliar injury; 3) estimates of species specific biomass loss within the
34   case study area. We also consider qualitative analyses on ecosystem services effects for this
35   endpoint. For example, the second draft WREA uses GIS mapping to illustrate where effects

1   may be occurring and relates those areas to national scale statistics for recreational use and data
2   on hiking trails, campgrounds and other park amenities that intersect with potentially affected
3   areas. These are used to identify impacts on ecosystem services associated with recreation in
4   national parks. We additionally consider analyses relating elevated O_3 concentrations to
5   increased vulnerability to fire risk regimes, insect attacks and impacts on hydrological cycles.
6   With regard to risks related to biomass and crop yield loss, we consider WREA results
7   based on exposure-response functions for tree and crop species that predict the growth or yield
8   response of each species, based on the exposure patterns estimated within its growing region. To
9   compare exposure-response across species, genotypes or experiments for which absolute
10   response values may vary greatly, the second draft WREA instead uses estimates of relative
11   biomass loss for trees or yield loss for crops. The WREA develops such estimates nationally and
12   separately for more than 100 federally designated Class I areas. Additionally, we consider
13   WREA-developed estimates of associated impacts on the agriculture and forestry sectors
14   quantifying how O_3 exposure to vegetation is estimated to affect the provision of timber and
15   crops and carbon sequestration. We consider estimates for impacts related to tree biomass loss on
16   ecosystem services such as pollution removal, carbon storage and sequestration in five urban
17   case study areas. We consider biomass and crop yield loss estimates in light of advice from
18   CASAC, as discussed in sections 5.3 and 5.4 below.
19   In considering the amount of weight to place on the estimates of exposures and risks at or
20   above specific W126 values described in the second draft WREA, our approach: 1) evaluates the
21   weight of the scientific evidence concerning vegetation effects associated with those O_3
22   exposures; 2) considers the importance, from a public welfare perspective, of the O_3-induced
23   effects on sensitive vegetation and associated ecosystem services that are known or anticipated to
24   occur as a result of exposures at selected W126 values; and, 3) recognizes that predictions of
25   effects associated with any given O_3 exposure may be mitigated or exacerbated by actual
26   conditions in the field (i.e., co-occurring modifying environmental and genetic factors). When
27   considering analyses in the second draft WREA that involve discrete exposure levels or varying
28   levels of severity of effects, our approach recognizes that welfare-relevant exposures are more
29   appropriately viewed as a continuum with greater confidence and less uncertainty about the
30   existence of welfare effects at higher O_3 exposure concentrations and less confidence and greater
31   uncertainty as one considers lower exposure concentrations. We recognize that there is no sharp
32   breakpoint within the continuum ranging from concentrations at and above the level of the
33   current secondary standard down to the lowest cumulative, seasonal W126 value assessed. In
34   considering these results in this second draft PA, we consider both concerns about the potential
35   for welfare effects and their severity with the increasing uncertainty associated and our
36   understanding of the likelihood of such effects following exposures to lower O_3 concentrations.

1.3.2.2.3 Considerations Regarding Ambient O_3 Concentration Estimates Attributable to Background Sources

As noted above, our approach in this review utilizes recent advances in modeling techniques to estimate the contributions of U.S. anthropogenic, international anthropogenic, and natural sources to ambient O_3 (discussed in detail in Chapter 2 of this document). Such model estimates can provide insights into the extent to which different types of emissions sources contribute to total ambient O_3 concentrations. Our consideration of this issue in the current review is informed by the approaches taken in previous reviews, and by court decisions on subsequent litigation, as discussed in section 1.3.1.2.3 above.

Further, in the 1996 proposal, O_3 background concentrations were one of the factors the Administrator considered in selecting the SUM06 index as a form for an alternative secondary standard. This and other cumulative exposure indices under consideration were judged to be equally capable at estimating exposures relevant to vegetation, given the lack of evidence for a discernible threshold for vegetation effects in general (U.S. EPA 1996, p. 225), which might have provided a scientific basis for selecting among different cumulative exposure indices. At that time, the SUM06 metric was selected over the W126 metric because it focused on the policy-relevant (above background) portion of the total cumulative seasonal exposures reaching plants (62 FR 38856). At the conclusion of that review, the Administrator ultimately chose to set the secondary standard identical to the primary standard, including using the 8-hour average instead of a cumulative seasonal form (62 FR 38868). In the 2008 review, staff analyses concluded that the W126 index was more biologically-relevant based on the available science; staff additionally noted, based on then-available estimates of background, that this form was also not likely to be significantly impacted by background concentrations given the very low weight assigned to lower O_3 concentrations by the W126 index (2007 SP, 7-22; 72 FR 37893).

1.3.3 Organization of this Document

Chapter 2 of this second draft PA provides an overview of the O_3 ambient monitoring network and O_3 air quality, including estimates of O_3 concentrations attributable to background sources. The remaining chapters are organized into two main parts. Chapters 3 and 4 focus on the review of the primary O_3 NAAQS while chapters 5 and 6 focus on the review of the secondary O_3 NAAQS. Staff's considerations and conclusions related to the current primary and secondary standards are discussed in chapters 3 and 5, respectively. Staff's considerations and conclusions related to potential alternative primary and secondary standards are discussed in chapters 4 and 6, respectively. Key uncertainties in the review and areas for future research and data collection are additionally identified in chapters 4 and 6 for the two types of standards.

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2   O$_3$ MONITORING AND AIR QUALITY

This section provides overviews of ambient O$_3$ monitoring in the U.S. (section 2.1); O$_3$ precursor emissions and atmospheric chemistry (section 2.2); ambient O$_3$ concentrations (section 2.3); and available evidence and information related to background O$_3$ (section 2.4). These issues are also discussed in detail in chapter 3 of the ISA (US EPA, 2013).

2.1 O$_3$ MONITORING

2.1.1   O$_3$ Monitoring Network

To monitor compliance with the NAAQS, state and local environmental agencies operate O$_3$ monitoring sites at various locations, depending on the population of the area and typical peak O$_3$ concentrations[1]. In 2010, there were over 1,300 state, local, and tribal O$_3$ monitors reporting concentrations to EPA. In areas for which O$_3$ monitors are required, at least one site must be designed to record the maximum concentration for that particular metropolitan area. Since O$_3$ concentrations are usually significantly lower in the colder months of the year, O$_3$ is required to be monitored only during the O$_3$ monitoring season, which varies by state.[2]

Figure 2-1 shows the locations of the U.S. ambient O$_3$ monitoring sites reporting data to EPA at any time during the 2006-2010 period. The gray dots which make up over 80% of the O$_3$ monitoring network are "State and Local Monitoring Stations" (SLAMS) monitors, which are operated by state and local governments to meet regulatory requirements and provide air quality information to public health agencies. Thus, the SLAMS monitoring sites are largely focused on urban areas. The blue dots highlight two important subsets of monitoring sites within the SLAMS network: the "National Core" (NCore) multi-pollutant monitoring network and the "Photochemical Assessment Monitoring Stations" (PAMS) network.

While the existing U.S. O$_3$ monitoring network has a largely urban focus, to address ecosystem impacts of O$_3$ such as biomass loss and foliar injury, it is equally important to focus on O$_3$ monitoring in rural areas. The green dots in Figure 2-1 represent the Clean Air Status and Trends Network (CASTNET) monitors which are located in rural areas. There were about 80 CASTNET sites operating in 2010, with sites in the eastern U.S. being operated by EPA and sites in the western U.S. being operated by the National Park Service (NPS). Finally, the black dots represent "Special Purpose Monitoring Stations" (SPMS), which include about 20 rural monitors as part of the "Portable O$_3$ Monitoring System" (POMS) network operated by the NPS.

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[1] The minimum O3 monitoring network requirements for urban areas are listed in Table D-2 of Appendix D to 40 CFR Part 58.

[2] The required O$_3$ monitoring seasons for each state are listed in Table D-3 of Appendix D to 40 CFR Part 58.

1   Between the CASTNET, NCore, and POMS networks, there were about 120 rural O_3 monitoring

2   sites operating in the U.S. in 2010.

3

4
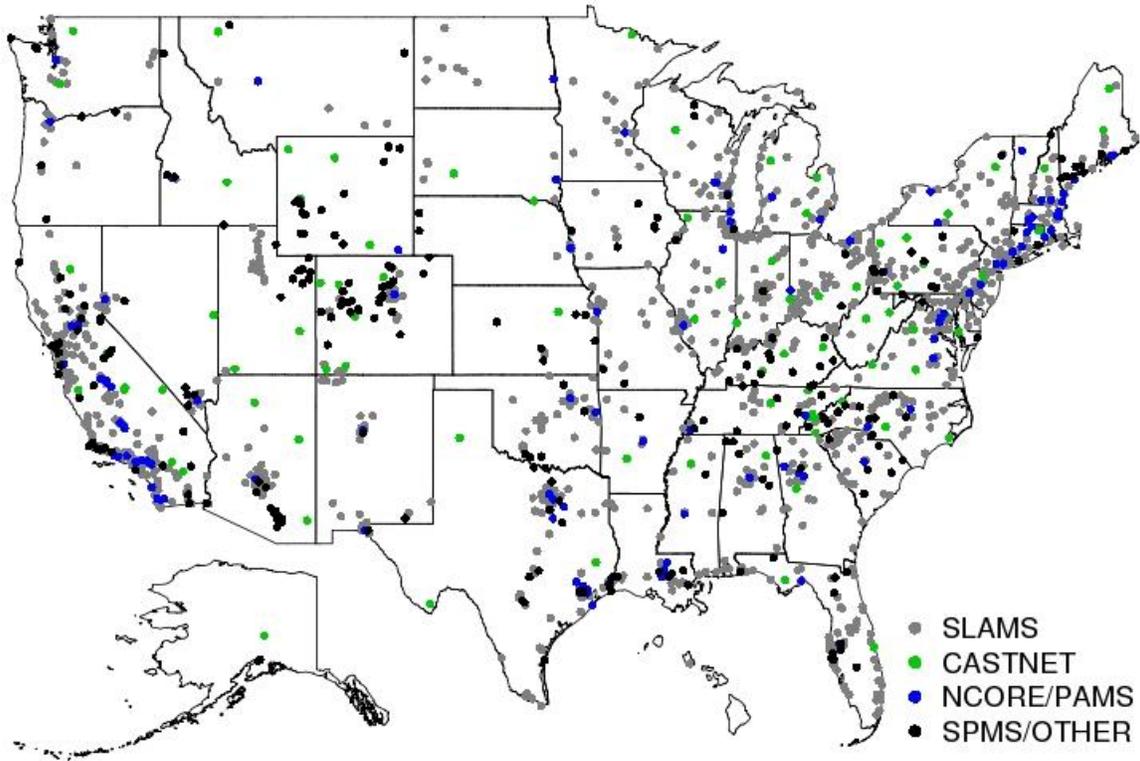
**Figure 2-1.   Map of U.S. ambient O3 monitoring sites reporting data to EPA during the
                2006-2010 period.**

2.1.2   Recent O_3 Monitoring Data and Trends

8   To determine whether or not the O_3 NAAQS has been met at an ambient monitoring site,

9   a statistic commonly referred to as a "design value" must be calculated based on 3 consecutive

10   years of data collected from that site. The form of the existing O_3 NAAQS design value statistic

11   is the 3-year average of the annual 4th highest daily maximum 8-hour O_3 concentration in parts

12   per billion (ppb), with decimal digits truncated. The existing primary and secondary O_3 NAAQS

13   are met at an ambient monitoring site when the design value is less than or equal to 75 ppb.[3] In

14   counties or other geographic areas with multiple monitors, the area-wide design value is defined

15   as the design value at the highest individual monitoring site, and the area is said to have met the

16   NAAQS if all monitors in the area are meeting the NAAQS.

17   Figure 2-2 shows the trend in the annual 4th highest daily maximum 8-hour O_3

18   concentrations in ppb based on 933 "trends" sites with complete data records over the 2000 to

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[3]For more details on the data handling procedures used to calculate design values for the existing O_3 NAAQS, see
40 CFR Part 50, Appendix P.

1   2012 period. The center line in this figure represents the median value across the trends sites,
2   while the dashed lines represent the 25th and 75th percentiles, and the bottom and top lines
3   represent the 10th and 90th percentiles. Figures 2-3 and 2-4 show maps of the O_3 design values
4   (ppb) at all U.S. monitoring sites for the 2009-2011 and 2010-2012 periods, respectively. The
5   trend shows that the annual 4th highest daily maximum values decreased for the vast majority of
6   monitoring sites in the U.S. between 2000 and 2009. The decreasing trend is especially sharp
7   from 2002 to 2004, when EPA implemented the "NOx SIP Call", a program designed to reduce
8   summertime emissions of NOx in the eastern U.S.

9   The trends also show a modest increase in the 4th highest daily maximum values from
10   2009 to 2012. This is reflected in the design value maps, which show an increase in the number
11   of monitors violating the existing O_3 standard in 2010-2012 relative to 2009-2011. Meteorology
12   played an important role in these trends. O_3 concentrations tend to be higher on days with hot
13   and stagnant conditions and lower on days with cool or wet conditions. According to the
14   National Oceanic and Atmospheric Administration's National Climactic Data Center (NOAA-
15   NCDC), the summer of 2009 was cooler and wetter than average over most of the eastern U.S.,
16   while conversely, the summers, of 2010, 2011, and 2012 were all much warmer than average. In
17   particular, the central and eastern U.S. experienced a 2-week period of record-breaking heat in
18   late June and early July of 2012, which contributed to hundreds of violations of the existing O_3
19   standard. In contrast, the most recent climatological information available from NOAA-NCDC
20   (http://www.ncdc.noaa.gov/sotc/) shows that the summer of 2013 was cooler and wetter than
21   average for much of the U.S. Thus, EPA does not expect the recent increasing trend in the 4th
22   highest daily maximum O_3 concentrations to continue in 2013.
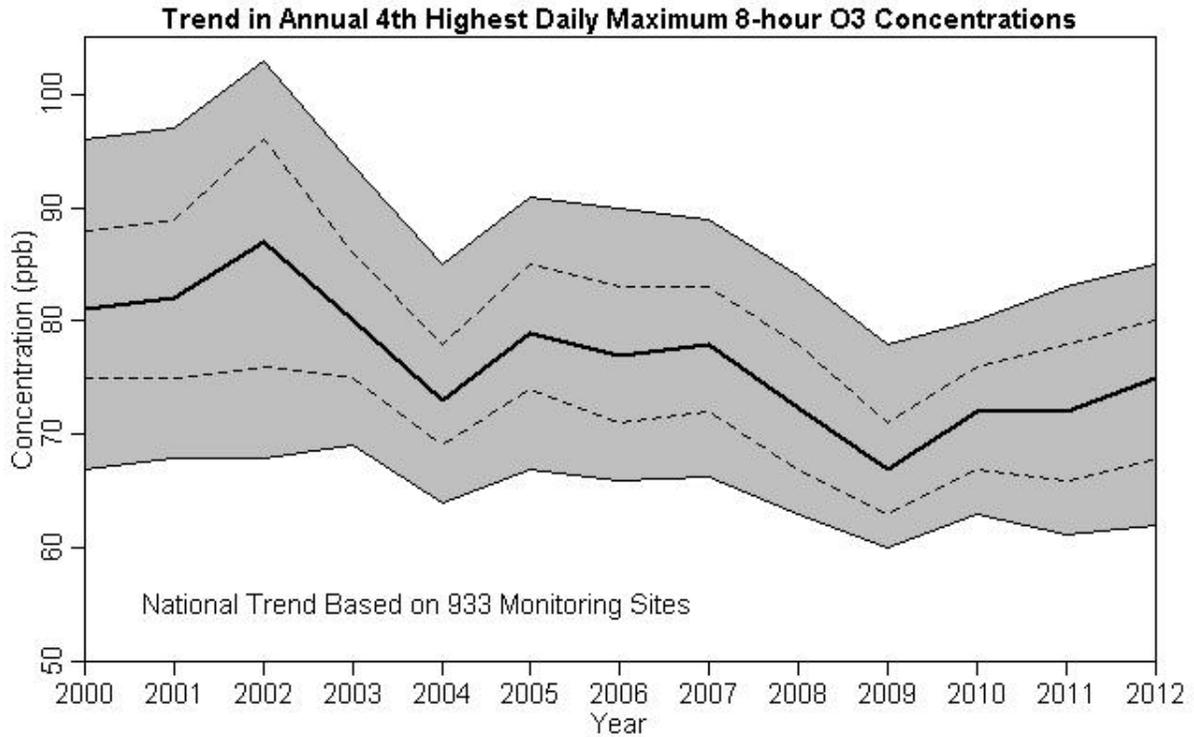
Figure 2-2. Trend in U.S. annual 4th highest daily maximum 8-hour O_3 concentrations in ppb, 2000 to 2012.

1

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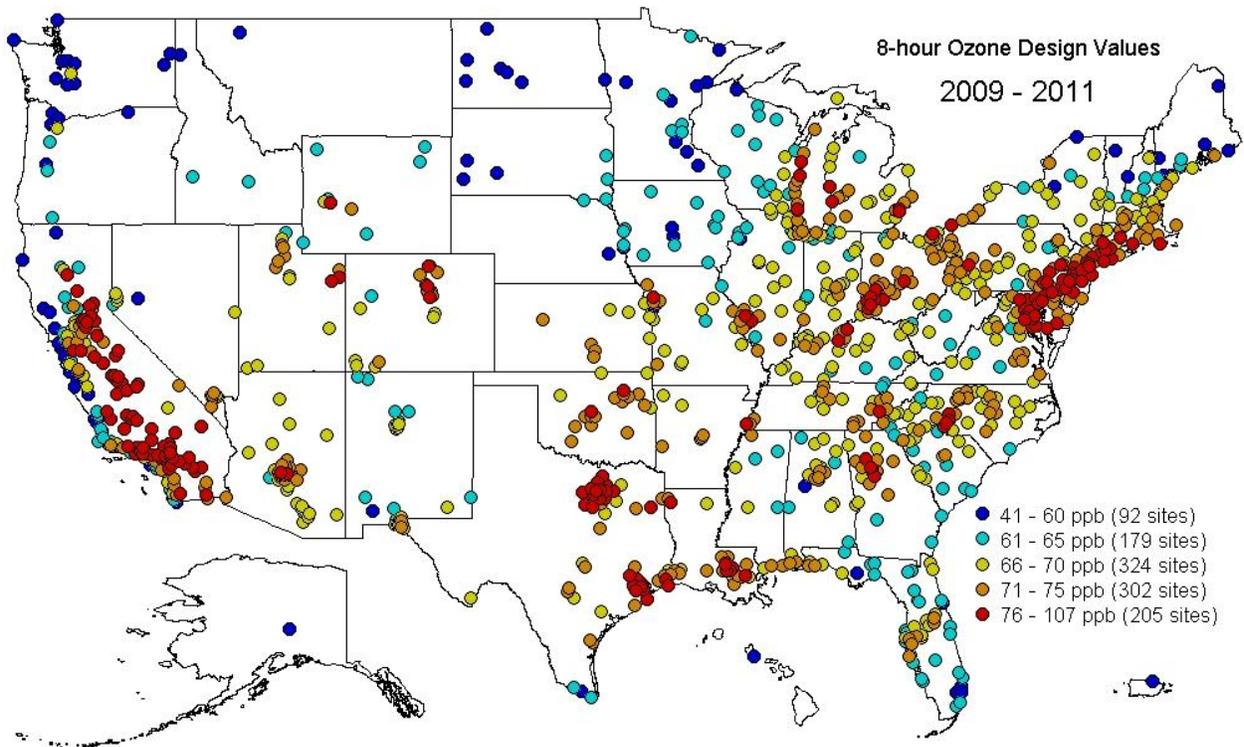
Figure 2-3. Map of 8-hour O_3 design values in ppb for the 2009-2011 period.

5

8-hour Ozone Design Values
2010 - 2012

- 43 - 60 ppb (87 sites)
- 61 - 65 ppb (130 sites)
- 66 - 70 ppb (237 sites)
- 71 - 75 ppb (309 sites)
- 76 - 106 ppb (363 sites)

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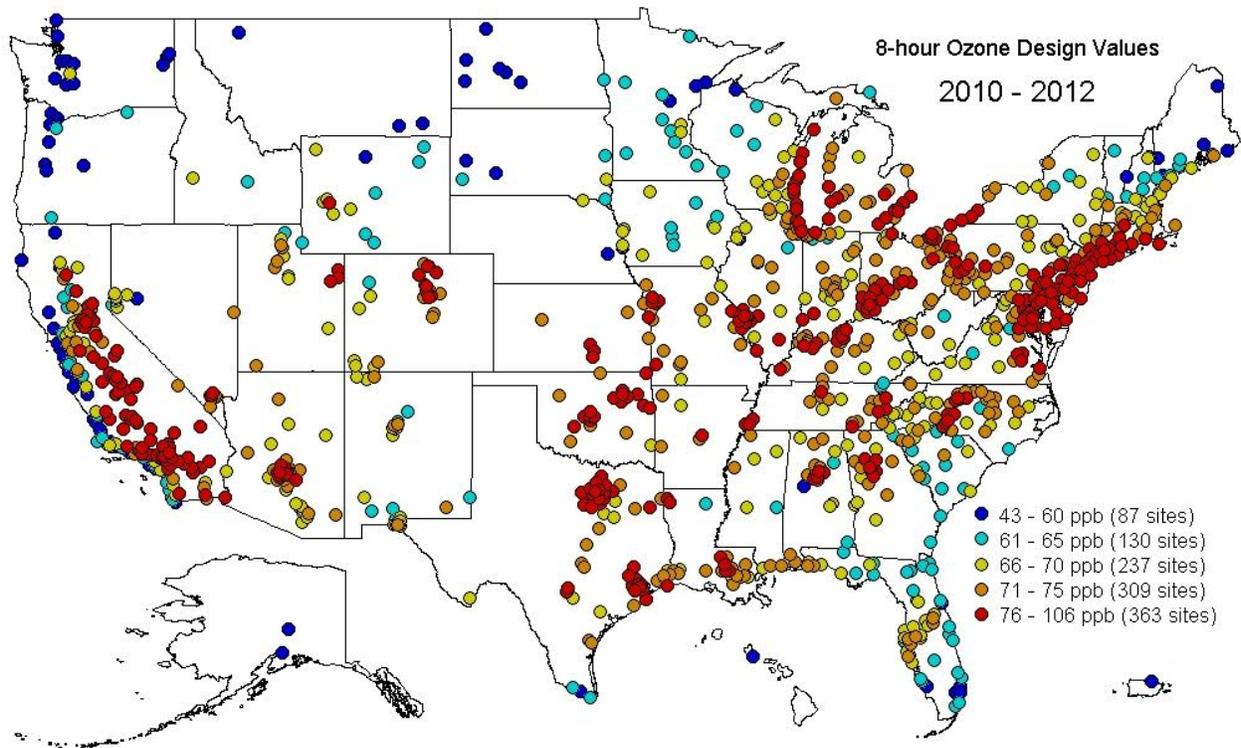
Figure 2-4.   Map of 8-hour O$_3$ design values in ppb for the 2010-2012 period.

In addition, EPA focused our analyses of welfare and ecosystem effects on a W126 O$_3$ exposure metric in this review. The W126 metric[4] is a seasonal aggregate of daytime (8:00 AM to 8:00 PM) hourly O$_3$ concentrations, designed to measure the cumulative effects of O$_3$ exposure on vulnerable plant and tree species, with units in parts per million-hours (ppm-hrs). The W126 metric uses a logistic weighting function to place less emphasis on exposure to low hourly O$_3$ concentrations and more emphasis on exposure to high hourly O$_3$ concentrations (Lefohn et al, 1988).

Figure 2-5 shows the trend in annual W126 concentrations in ppm-hrs based on 933 "trends" sites with complete data records over the 2000 to 2012 period. Figures 2-6 and 2-7 show maps of the 3-year average annual W126 concentrations in ppm-hrs at all U.S. monitoring sites for the 2009-2011 and 2010-2012 periods, respectively. The general patterns seen in these figures are similar to those seen in the design value metric for the existing standard.

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[4] Details on the procedure used to calculate the W126 metric are provided in Chapter 4 of the welfare Risk and Exposure Assessment.
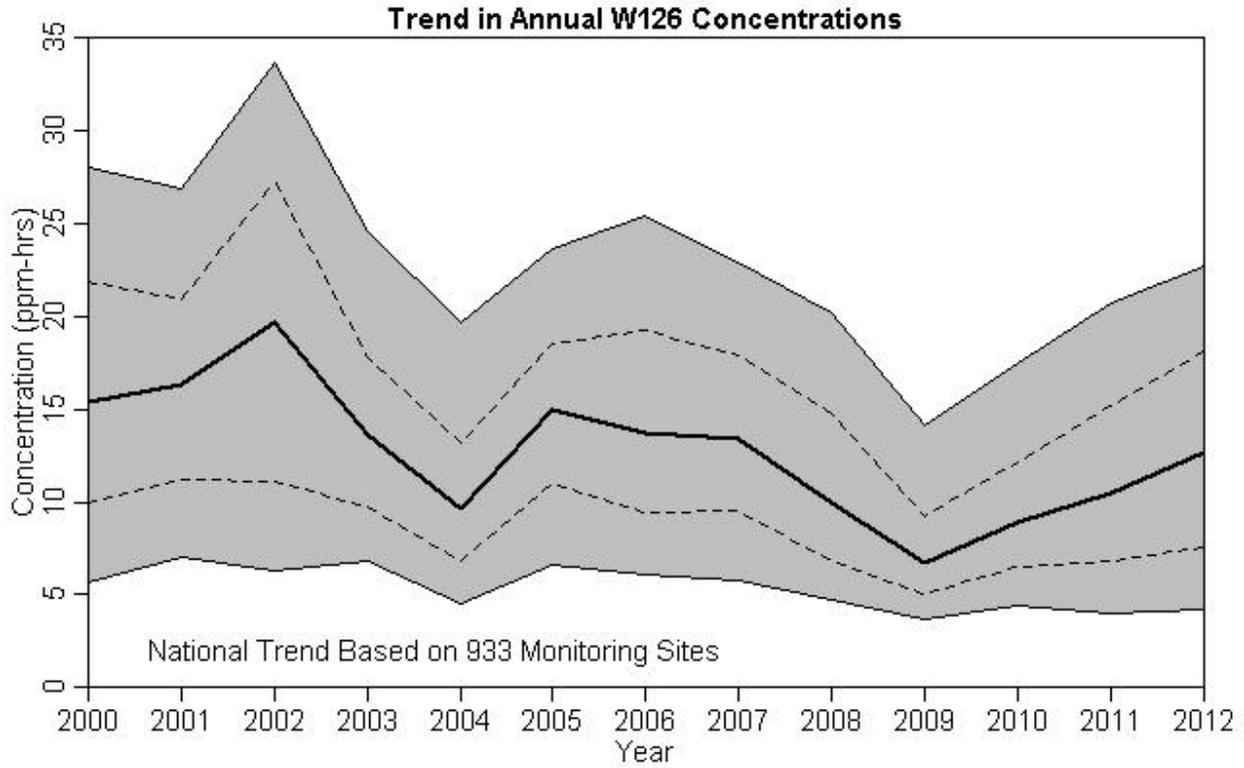
Figure 2-5.   Trend in U.S. annual W126 concentrations in ppm-hrs, 2000 to 2012.
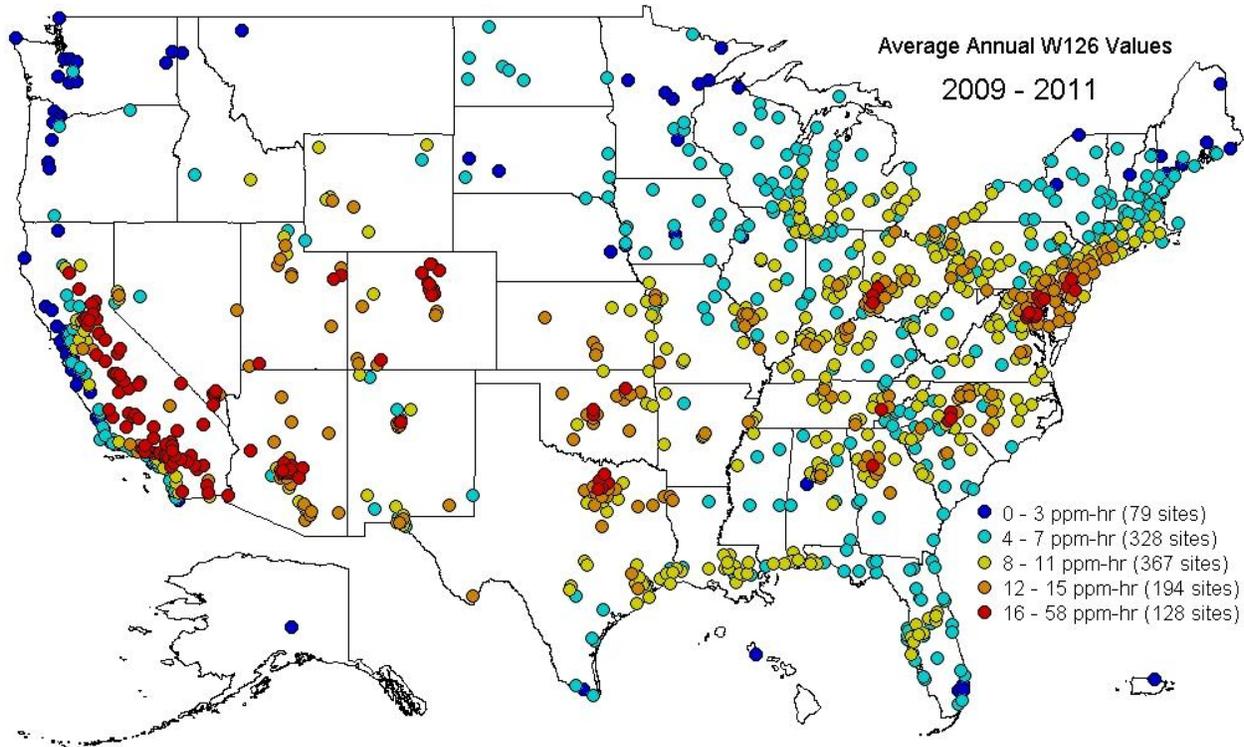
Figure 2-6.   Map of 2009-2011 average annual W126 values in ppm-hrs.

1
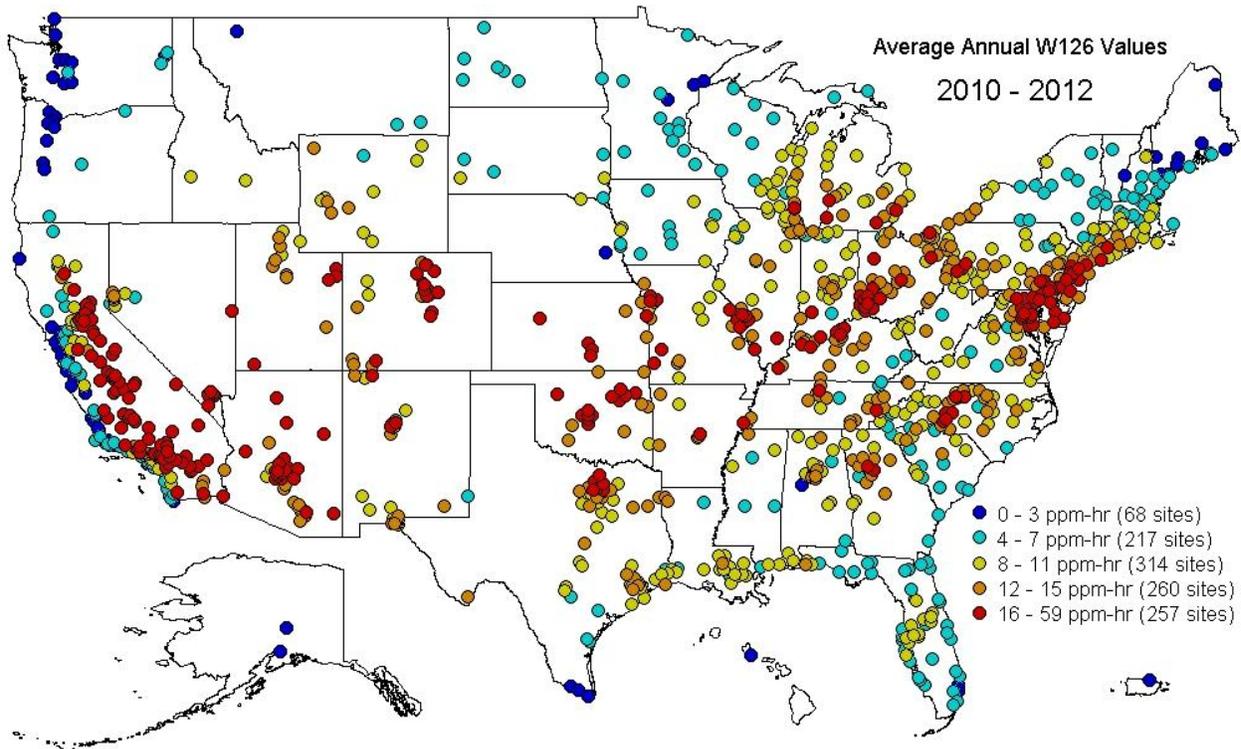
Figure 2-7.   Map of 2010-2012 average annual W126 values in ppm-hrs.

2.2 EMISSIONS AND ATMOSPHERIC CHEMISTRY

O_3 is formed by photochemical reactions of precursor gases and is not directly emitted from specific sources. In the stratosphere, O_3 occurs naturally and provides protection against harmful solar ultraviolet radiation. In the troposphere, near ground level, O_3 forms through atmospheric reactions involving two main classes of precursor pollutants: non-methane volatile organic compounds (NMVOCs) and nitrogen oxides (NO_X). Carbon monoxide (CO) and methane (CH_4) are also important for O_3 formation over longer time periods (US EPA, 2013, section 3.2.2).

Emissions of O_3 precursor compounds can be divided into anthropogenic and natural source categories, with natural sources further divided into biogenic emissions (from vegetation, microbes, and animals) and abiotic emissions (from biomass burning, lightning, and geogenic sources). Anthropogenic sources, including mobile sources and power plants, account for the majority of NOx and CO emissions. Anthropogenic sources are also important for NMVOC emissions, though in some locations and at certain times of the year (e.g., southern states during summer) the majority of NMVOC emissions come from vegetation (US EPA, 2013, section 3.2.1). 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

1　natural sources during the preindustrial era. Thus, emissions from plants, animals, and wildfires

2　could be considered either natural or anthropogenic, depending on whether emissions result from

3　agricultural practices, forest management practices, lightning strikes, or other types of events

4　(US EPA, 2013, sections 3.2 and 3.7.1).

5　　　Rather than varying directly with emissions of its precursors, O_3 changes in a nonlinear

6　fashion with the concentrations of its precursors. NO_x emissions lead to both the formation and

7　destruction of O_3, depending on the local quantities of NO_x, NMVOC, radicals, and sunlight. In

8　areas dominated by fresh emissions of NO_x, these radicals are removed, which lowers the O_3

9　formation rate. In addition, the scavenging of O_3 by reaction with NO is called "titration" and is

10　often found in downtown metropolitan areas, especially near busy streets and roads, and in

11　power plant plumes. This short-lived titration results in localized areas in which O_3

12　concentrations are suppressed compared to surrounding areas, but which contain NO_2 that

13　contributes to O_3 formation later and further downwind. Consequently, O_3 response to

14　reductions in NO_x emissions is complex and may include O_3 decreases at some times and

15　locations and increases of O_3 at other times and locations. In areas with low NO_x to VOC ratios,

16　such as those found in remote continental areas and rural and suburban areas downwind of urban

17　centers, O_3 production typically varies directly with NO_x concentrations (e.g. increases with

18　increasing NO_x emissions).

19　　　At some times and in some locations, reductions in O_3 precursors may also yield

20　reductions in ambient air pollutants other than O_3. For example, given that NO_x emissions

21　contribute to ambient NO_2 (i.e., both because NO_2 is a component of NO_x emissions and because

22　NO can convert rapidly to NO_2), reductions in directly emitted NO_x will also result in reductions

23　in ambient NO_2. In addition, NO_x and VOCs can contribute to secondary formation of $PM_{2.5}$

24　constituents. NO_x can act as both a direct precursor to NH_4NO_3, and can affect the formation of

25　other $PM_{2.5}$ constituents because it adds to the oxidative capacity of the atmosphere[5]. The effects

26　of reducing NO_x emissions on ambient $PM_{2.5}$ concentrations can vary in time and space, with the

27　largest reductions in ambient $PM_{2.5}$ likely occurring at times when and in locations where

28　concentrations of NH_4NO_3 are highest. This is usually during the cooler times of the year (e.g.

29　April-November) and in some areas of California, Salt Lake City, The Great Lakes States, and

30　the Northeast corridor between Baltimore and New York City (Carlton et al, 2010)[6].

31　　　The formation of O_3 from precursor emissions is also affected by the intensity and

32　spectral distribution of sunlight and atmospheric mixing. Major episodes of high ground-level

33　O_3 concentrations in the eastern United States are associated with slow-moving high pressure

---

[5] Across North America, approximately 7% of summertime $PM_{2.5}$ mass is estimated to result from anthropogenic NO_x emissions and up to 0.5 ug/m^3 of secondary organic aerosol is estimated to form from NOx emissions (Carlton et al, 2010).

[6] In these locations, NH_4NO_3 contributes more than 30% to average $PM_{2.5}$ concentrations.

1   systems. High pressure systems during the warmer seasons are associated with the sinking of

2   air, resulting in warm, generally cloudless skies, with light winds. The sinking of air results in

3   the development of stable conditions near the surface which inhibit or reduce the vertical mixing

4   of O_3 precursors. The combination of inhibited vertical mixing and light winds minimizes the

5   dispersal of pollutants emitted in urban areas, allowing their concentrations to build up. In

6   addition, in some parts of the United States (e.g., in Los Angeles), mountain barriers limit mixing

7   and result in a higher frequency and duration of days with high O_3 concentrations.

8   Photochemical activity involving precursors is enhanced during warmer seasons because of the

9   availability of sunlight and higher temperatures (US EPA, 2013, section 3.2).

10   O_3 concentrations in a region are affected both by local formation and by transport of O_3

11   and its precursors from upwind areas. O_3 transport occurs on many spatial scales including local

12   transport between cities, regional transport over large regions of the U.S. and international/long-

13   range transport. In addition, O_3 can be transferred into the troposphere from the stratosphere,

14   which is rich in O_3, through stratosphere-troposphere exchange (STE). These inversions or

15   "folds" usually occur behind cold fronts, bringing stratospheric air with them and typically affect

16   O_3 concentrations in high elevation areas (e.g. > 1500 m) more than areas at low elevations (U.S.

17   EPA, 2012, section 3.4.1.1). The role of long-range transport of ozone and other elements of

18   ozone background is discussed in more detail in Section 2.4,

19   **2.3 AIR QUALITY CONCENTRATIONS**

20   Because O_3 is a secondary pollutant formed in the atmosphere from precursor emissions,

21   concentrations are generally more regionally homogeneous than concentrations of primary

22   pollutants emitted directly from stationary and mobile sources (US EPA, 2013, section 3.6.2.1).

23   However, variation in local emissions characteristics, meteorological conditions, and topography

24   can result in daily and seasonal temporal variability in ambient O_3 concentrations, as well as

25   local and national-scale spatial variability.

26   Temporal variation in ambient O_3 concentrations results largely from daily and seasonal

27   patterns in sunlight, precursor emissions, atmospheric stability, wind direction, and temperature

28   (US EPA, 2013, section 3.7.5). On average, ambient O_3 concentrations follow well-recognized

29   daily and seasonal patterns, particularly in urban areas. Specifically, daily maximum O_3

30   concentrations in urban areas tend to occur in mid-afternoon, with more pronounced peaks in the

31   warm months of the O_3 season than in the colder months (US EPA, 2013, Figures 3-54, 3-156 to

32   3-157). Rural sites also followed this general pattern, though it is less pronounced in colder

33   months (US EPA, 2013, Figure 3-55). With regard to day-to-day variability, median maximum

34   daily average 8-hour (MDA8) O_3 concentrations in U.S. cities in 2007-2009 were approximately

1   47 ppb, with typical ranges between 35 to 60 ppb and the highest MDA8 concentrations above
2   100 ppb in several U.S. cities (as noted further below).

3   In addition to temporal variability, there is considerable spatial variability in ambient O_3
4   concentrations within cities and across different cities in the United States. With regard to
5   spatial variability within a city, local emissions characteristics, geography, and topography can
6   have important impacts. For example, as noted above, fresh NO emissions from motor vehicles
7   titrate O_3 present in the urban background air, resulting in an O_3 gradient around roadways with
8   O_3 concentrations increasing as distance from the road increases (US EPA, 2013, section
9   3.6.2.1). In comparing urban areas, the ISA notes that measured O_3 concentrations are relatively
10   uniform and well-correlated across some cities (e.g., Atlanta) while they are more variable in
11   others (e.g., Los Angeles) (US EPA, 2013, section 3.6.2.1 and Figures 3-28 to 3-36).

12   With regard to spatial variability across cities, when the ISA evaluated the distributions
13   of 8-hour O_3 concentrations for the years 2007 to 2009 in 20 cities, the highest concentrations
14   were reported in Los Angeles, with high concentrations also reported in several eastern and
15   southern cities. The maximum recorded MDA8 was 137 ppb in Los Angeles, and was near or
16   above 120 ppb in Atlanta, Baltimore, Dallas, New York City, Philadelphia, and St. Louis (US
17   EPA, 2013, Table 3-10). The pattern was similar for the 98th percentile of the distribution of
18   MDA8 concentrations[7], with Los Angeles recording the highest 98th percentile concentration (91
19   ppb) and many eastern and southern cities reporting 98th percentile concentrations near or above
20   75 ppb. In contrast, somewhat lower 98th percentile O_3 concentrations were recorded in cities in
21   the western United States outside of California (US EPA, 2013, Table 3-10).

22   Although rural monitoring sites tend to be less directly affected by anthropogenic
23   pollution sources than urban sites, rural sites can be affected by transport of O_3 or O_3 precursors
24   from upwind urban areas and by local anthropogenic sources such as motor vehicles, power
25   generation, biomass combustion, or oil and gas operations (US EPA, 2013, section 3.6.2.2). In
26   addition, O_3 tends to persist longer in rural than in urban areas due to lower rates of chemical
27   scavenging in non-urban environments. At higher elevations, increased O_3 concentrations can
28   also result from stratospheric intrusions (US EPA, 2013, sections 3.4, 3.6.2.2). As a result, O_3
29   concentrations measured in some rural sites can be higher than those measured in nearby urban
30   areas (US EPA, 2013, section 3.6.2.2), and the ISA concludes that cumulative exposures for
31   humans and vegetation in rural areas can be substantial, often higher than cumulative exposures
32   in urban areas (US EPA, 2013, section 3.7.5).

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[7] Table 3-10 in the ISA analyzes the warm season. Therefore, the 98th percentile values would be an approximation
of the 4th highest value.

2.4 BACKGROUND O$_3$

Generically, background O$_3$ can originate from natural sources of O$_3$ and O$_3$ precursors, as well as from manmade international emissions of O$_3$ precursors. Natural sources of O$_3$ precursor emissions such as wildfires, lightning, and vegetation can lead to O$_3$ formation by chemical reactions with other natural sources. Another important natural component of background is O$_3$ that is naturally formed in the stratosphere through interactions of ultraviolet light with molecular oxygen. Stratospheric O$_3$ can mix down to the surface at high concentrations in discrete events called intrusions, especially at higher-altitude locations. The manmade portion of the background includes any O$_3$ formed due to anthropogenic sources of O$_3$ precursors emitted far away from the local area (e.g., international emissions). Finally, both biogenic and international anthropogenic emissions of methane, which can be chemically converted to O$_3$ over relatively long time scales, can also contribute to global background O$_3$ levels.

As indicated in the first draft policy assessment (US EPA, 2012, sections 1.3.4 and 3), EPA has updated several aspects of our methodology for estimating the change in health risk and exposure that would result from a revision to the O$_3$ NAAQS. First, risk estimates are now based on total O$_3$ concentrations, as opposed to previous reviews which only considered risk above background levels. Second, EPA is now using air quality models to estimate the spatial patterns of O$_3$ that would result from attaining various levels of the NAAQS, as opposed to a quadratic rollback approach that required the estimation of a background "floor" beyond which the rollback would not take place. Both of these revisions have had the indirect effect of reducing the need for estimates of background O$_3$ levels as part of the O$_3$ risk and exposure assessment (REA). Regardless, EPA expects that a well-founded understanding of the fractional contribution of background sources and processes to surface O$_3$ levels will be valuable towards informing policy decisions about the O$_3$ NAAQS. Accordingly, in this section, we briefly summarize existing results on background O$_3$ from the ISA (US EPA, 2013, section 3.4) as supplemented by additional EPA modeling recently conducted for a 2007 base year. The summary will focus on national estimates of: 1) seasonal mean background O$_3$ concentrations for three specific definitions of background O$_3$, 2) the relative proportion of background O$_3$ to total O$_3$ for the same three definitions from a seasonal mean perspective, 3) the distributions of background O$_3$ within a seasonal mean, 4) the fractional background O$_3$ in the 12 REA urban case study areas, 5) the relative proportion of background O$_3$ concentrations to total ozone from a W126 perspective, and 6) the relative roles of different components of background O$_3$.

The definition of background O$_3$ can vary depending upon context, but it generally refers to O$_3$ that is formed by sources or processes that cannot be influenced by actions within the jurisdiction of concern. In the first draft policy assessment document (US EPA, 2012), EPA

1   presented three specific definitions of background O_3: natural background, North American

2   background, and United States background. Natural background (NB) was the narrowest

3   definition of background, and it was defined as the O_3 that would exist in the absence of any

4   manmade O_3 precursor emissions. The other two previously established definitions of

5   background presume that the U.S. has little influence over anthropogenic emissions outside our

6   continental or domestic borders. North American background (NAB) is defined as that O_3 that

7   would exist in the absence of any manmade O_3 precursor emissions from North America. U.S.

8   background (USB) is defined as that O_3 that would exist in the absence of any manmade

9   emissions inside the United States. Each of these three definitions of background O_3 requires

10   photochemical modeling simulations to estimate what the residual O_3 concentrations would be

11   were the various anthropogenic emissions to be removed. Previous modeling studies have

12   estimated what background levels would be in the absence of certain sets of emissions by simply

13   assessing the remaining O_3 in a simulation in which certain emissions were removed (Zhang et

14   al. (2011), Emery et al. (2012)). This basic approach is often referred to as "zero-out" modeling

15   or "emissions perturbation" modeling.

16   While the zero-out approach has traditionally been used to estimate background O_3

17   levels, the methodology has some acknowledged limitations. First, from a policy perspective,

18   the hypothetical and unrealizable zero manmade emissions scenarios have limited application.

19   Secondly, the assumption that background O_3 is what is left after specific emissions have been

20   removed within the model simulation can be misleading in locations where O_3 chemistry is

21   highly non-linear. Depending upon the local composition of O_3 precursors, NOx emissions

22   reductions can either increase or decrease O_3 levels in the immediate vicinity of those reductions.

23   For those specific urban areas in which NOx titration of O_3 can be significant, zero-out modeling

24   can result in inflated estimates of background O_3 when these NOx emissions are completely and

25   unrealistically removed. Paradoxically, in certain times and locations in a zero-out scenario

26   there can be more background O_3 than actual O_3 within the model. A separate modeling

27   technique circumvents these limitations by apportioning the total O_3 within the model to its

28   contributing source terms. This basic approach, referred to as "source apportionment" modeling,

29   has been described and evaluated in the peer-reviewed literature (Dunker et al., 2002; Kemball-

30   Cook et al., 2009). While source apportionment modeling has not been previously used in the

31   context of estimating background ozone levels as part of an ozone NAAQS review, it has

32   frequently been used in other regulatory settings to estimate the "contribution" to ozone of

33   certain sets of emissions (EPA 2005, EPA 2011). The source apportionment technique provides

34   a means of estimating the contributions of user-identified source categories to ozone formation in

35   a single model simulation. This is achieved by using multiple tracer species to track the fate of

36   ozone precursor emissions (VOC and NOx) and the ozone formation caused by these emissions.

1   The methodology is designed so that all ozone and precursor concentrations are attributed to the
2   selected source categories at all times without perturbing the inherent chemistry. EPA recently
3   completed updated zero-out and source apportionment modeling for a 2007 base year to
4   supplement our characterization of background O_3 over the U.S. Prior to using model
5   simulations to estimate background O_3 levels over the U.S., EPA confirmed that the modeling
6   was able to reproduce historical O_3 levels and that there was limited correlation between model
7   errors and the background estimates. The key findings from the updated modeling are described
8   below; a more detailed description of the modeling is provided in Appendix A.

9   **2.4.1   Seasonal Mean Background O3 in the U.S.**

10   The ISA (US EPA 2013, section 3.4) previously established that background
11   concentrations vary spatially and temporally and that simulated mean background concentrations
12   are highest at high-elevation sites within the western U.S. Background levels typically are
13   greatest over the U.S. in the spring and early summer. Figure 2-8 displays the spatial patterns of
14   seasonal mean[8] natural background O_3 as estimated by a 2007 zero-out scenario. This figure
15   shows the average daily maximum 8-hour O_3 concentration (MDA8) that would exist in the
16   absence of any anthropogenic O_3 precursor emissions at monitor locations. Seasonal mean NB
17   levels range from approximately 15-35 ppb with the highest values at higher-elevation sites in
18   the western U.S. The median value over these locations is 24.2 ppb, and more than 50 percent of
19   the locations have natural background levels of 20-25 ppb. The highest modeled estimate of
20   seasonal average, natural background, MDA8 O_3 is 34.3 ppb at the high-elevation CASTNET
21   site (Gothic) in Gunnison County, CO. Natural background levels are higher at these high-
22   elevation locations primarily because natural stratospheric O_3 impacts and international transport
23   impacts increase with altitude (where O_3 lifetimes are longer).

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[8] The recent EPA modeling focused on the period from April through October. Seasonal means are computed over
those seven months.

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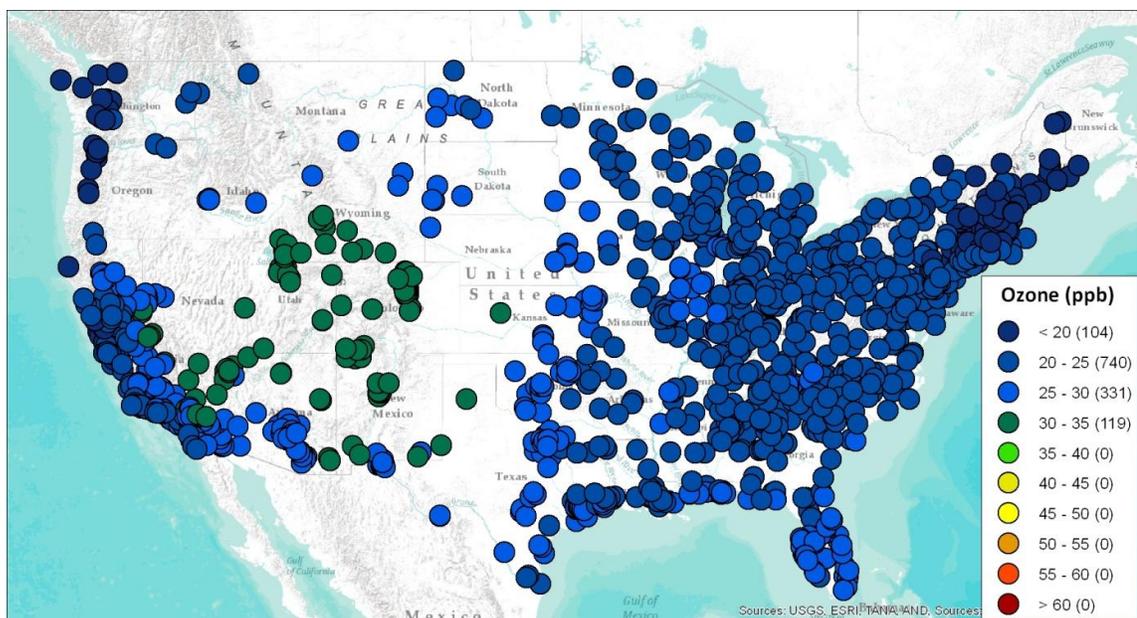
Figure 2-8.   Map of 2007 CMAQ-estimated seasonal mean natural background O$_3$ levels from zero-out modeling.

4   Figures 2-9 and 2-10 show the same information for the NAB and USB scenarios. In

5   these model runs, all anthropogenic O$_3$ precursor emissions were removed from the U.S.,

6   Canada, and Mexico portions of the modeling domain (NAB scenario) and then only from the

7   U.S. (USB scenario). The figures show that there is not a large difference between the NAB and

8   USB scenarios. Seasonal mean NAB and USB O$_3$ levels range from 25-50 ppb, with the most

9   frequent values estimated in the 30-35 ppb bin. The median seasonal mean background levels

10 are 31.5 and 32.7 ppb (NAB and USB, respectively). Again, the highest levels of seasonal mean

11 background are predicted over the intermountain western U.S. Locations with NAB and USB

12 concentrations greater than 40 ppb are confined to Colorado, Nevada, Utah, Wyoming, northern

13 Arizona, eastern California, and parts of New Mexico. The 2007 EPA modeling suggests that

14 seasonal mean USB concentrations are on average 1-3 ppb higher than NAB background. These

15 results were similar to those reported by Wang et al. (2009). From a seasonal mean perspective,

16 background levels are typically well-below the NAAQS thresholds.

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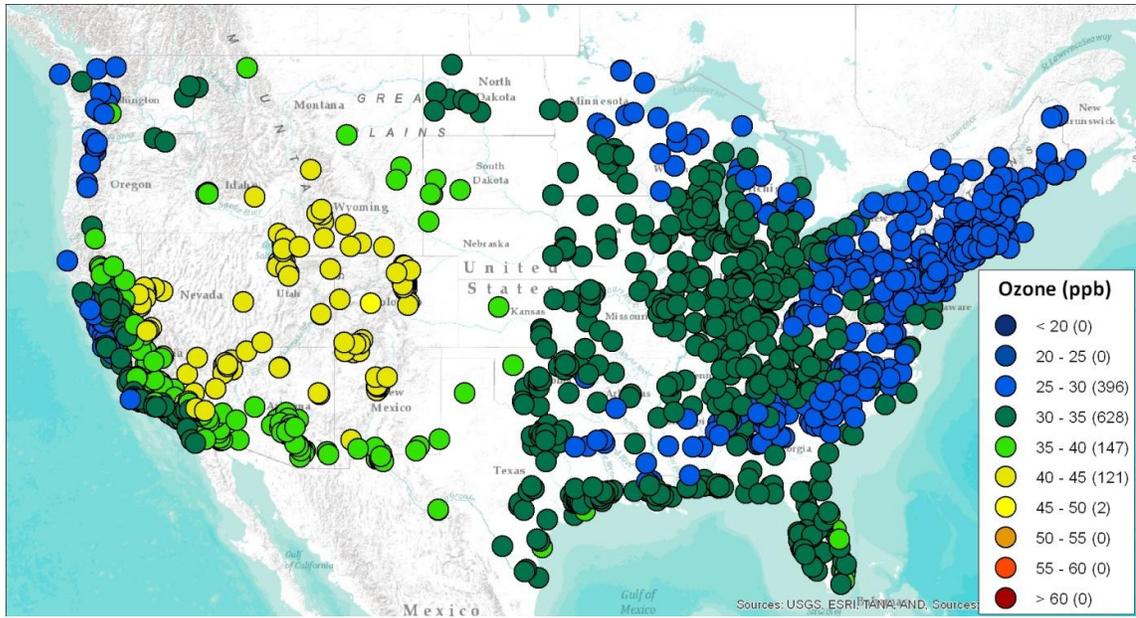
Figure 2-9.   Map of 2007 CMAQ-estimated seasonal mean North American background O$_3$ levels from zero-out modeling.

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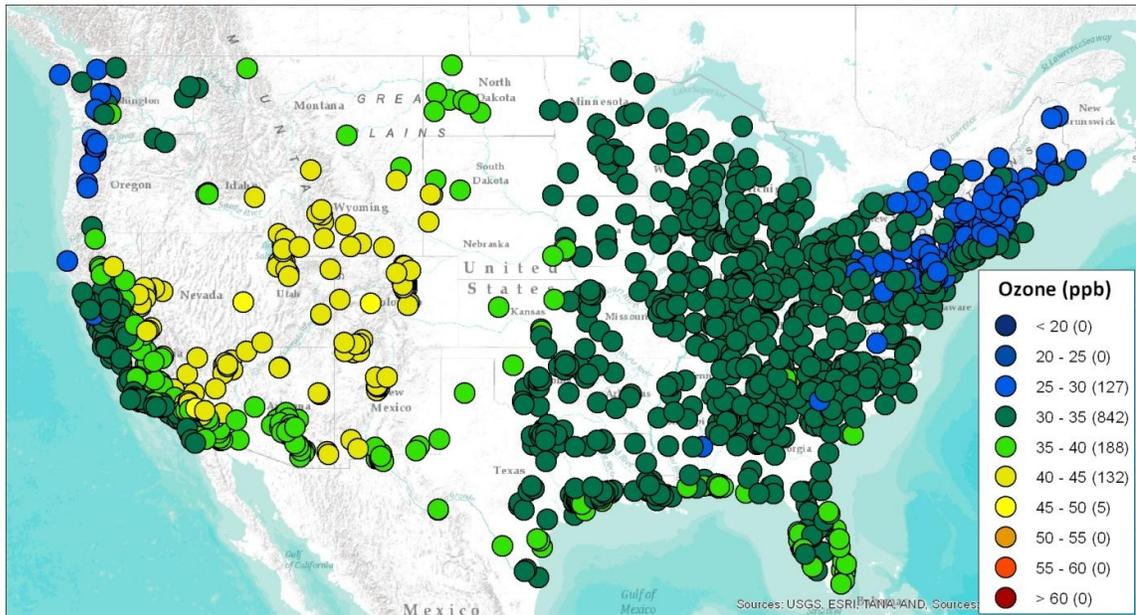
Figure 2-10.   Map of 2007 CMAQ-estimated seasonal mean United States background O$_3$ levels from zero-out modeling.

7   **2.4.2   Seasonal Mean Background O3 in the U.S. as a Proportion of Total O3**

8   Another informative way to assess the importance of background as part of seasonal

9   mean O$_3$ levels across the U.S. is to consider the fractional contribution of NB, NAB, and USB

10   to total modeled O$_3$ at each monitoring location. Considering the proportional role of

2-15

1   background allows for an initial assessment of the relative importance of background and non-
2   background sources. Figures 2-11 and 2-12 show the percent contribution of U.S. anthropogenic
3   sources to total O_3 using the metric of the seasonal mean MDA8 O_3 concentrations as estimated
4   by both the zero-out and source apportionment modeling methodologies. Recall that the terms
5   NB, NAB, and USB are explicitly linked to the zero-out modeling approach. For comparison
6   sake, in Figure 2-12 we are extending the definition of USB to also include the source
7   apportionment model estimates of the O_3 that is *attributable to sources other than U.S.*
8   *anthropogenic emissions*. To preserve the original definition of USB, this second term will be
9   hereafter referred to as "apportionment-based USB". As noted earlier, the advantage of the
10   source apportionment modeling is that all of the modeled O_3 is attributed to various source terms
11   and thus this approach is not affected by the confounding occurrences of background O_3 values
12   exceeding the base O_3 values as can happen in the zero-out modeling (i.e., background
13   proportions > 100%). Consequently, one would expect the fractional background levels to be
14   lower in the source apportionment methodology as a result of removing this artifact.
15   When averaged over all sites, O_3 from sources other than U.S. anthropogenic emissions
16   is estimated to comprise 66 (zero-out) and 59 (source apportionment) percent of the total
17   seasonal O_3 mean. The spatial patterns of apportionment-based USB are similar across the two
18   modeling exercises. Background O_3 is a relatively larger percentage (e.g., 70-80%) of the total
19   seasonal mean O_3 in locations within the intermountain western U.S. and along the U.S. border.
20   In locations where O_3 levels are generally higher, like California and the eastern U.S. the
21   seasonal mean background fractions are relatively smaller (e.g., 40-60%). The additional 2007
22   modeling confirms that background ozone, while generally not approaching levels of the ozone
23   standard, can comprise a considerable fraction of total seasonal mean ozone across the U.S.

24   **2.4.3   Daily Distributions of Background O_3 within the Seasonal Mean**

25   As a first-order understanding, it is valuable to be able to characterize seasonal mean
26   levels of background O_3. However, it is well established that background levels can vary
27   substantially from day-to-day. From an implementation perspective, the values of background
28   O_3 on possible exceedance days are a more meaningful consideration. The first draft policy
29   assessment (US EPA, 2012) considered this issue in detail, via summaries of the existing 2006
30   zero-out modeling (Henderson et al., 2012), and concluded that "results suggest that background
31   concentrations on the days with the highest total O_3 concentrations are not dramatically higher
32   than typical seasonal average background concentrations." Based on this finding, EPA
33   determined that "anthropogenic sources within the U.S. are largely responsible for 4th highest 8-
34   hour daily maximum O_3 concentrations." The recent EPA modeling using a 2007 base year and
35   the two distinct modeling methodologies supports this finding from the previous 2006-based

1 modeling analyses. That is, the highest modeled O_3 site-days tend to have background O_3 levels

2 similar to mid-range O_3 days. Figure 2-13 and 2-14 show the distribution of daily MDA8

3 apportionment-based USB levels (absolute magnitudes and relative fractions, respectively) from

4 the CAMx source apportionment simulation[9]. Again, the 2007 modeling shows that the days

5 with highest O_3 levels have similar distributions (i.e., means, inter-quartile ranges) of

6 background levels as days with lower values down to approximately 40 ppb. As a result, the

7 proportion of total O_3 that has background origins is smaller on high O_3 days (e.g., days > 70

8 ppb) than the more common lower O_3 days that drive seasonal means. This helps put the results

9 from section 2.4.2 into better context. For example, for site-days in which base O_3 is between

10 70-75 ppb, the source apportionment modeling estimates that approximately 37 percent of those

11 O_3 levels originate from sources other than U.S. anthropogenic emissions (i.e., apportionment-

12 based USB). Figure 2-14 also indicates that there are cases in which the model predicts much

13 larger background proportions, as shown by the upper outliers in the figure. These infrequent

14 episodes usually occur in relation to a specific event, and occur more often in specific

15 geographical locations, such as at high elevations or wildfire prone areas during the local dry

16 season.

17   It should be noted here that EPA has policies for treatment of air quality monitoring data

18 affected by these types of events. EPA's exceptional events policy allows exclusion of certain

19 air quality monitoring data from regulatory determinations if a State adequately demonstrates

20 that an exceptional event has caused the exceedance or violation of a NAAQS. In addition,

21 Section 179B of the CAA also provides for treatment of air quality data from international

22 transport when an exceedance or violation of a NAAQS would not have occurred but for

23 emissions emanating from outside of the United States. From an overarching perspective, the

24 Clean Air Act requires the NAAQS to be set at a level requisite to protect public health and

25 welfare. Case law makes it clear that attainability and technical feasibility are not relevant

26 considerations in the setting of a NAAQS. In previous reviews, EPA has assessed the proximity

27 of ozone concentrations to peak background levels only as a secondary consideration between

28 potential threshold levels where health and welfare was determined to have been protected.

29

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[9] Similar plots from the zero-out modeling for natural background, North American background, and U.S. background are provided in Appendix A.

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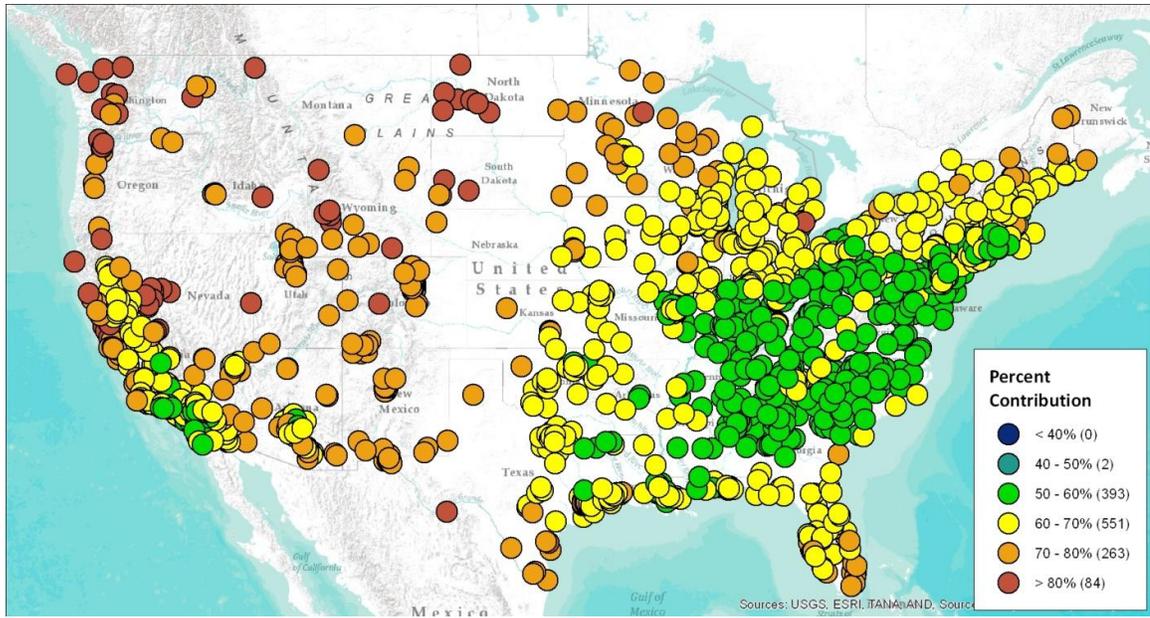
Figure 2-11. Map of U.S. background percent contribution to seasonal mean O$_3$ based on 2007 CMAQ zero-out modeling.

4

5
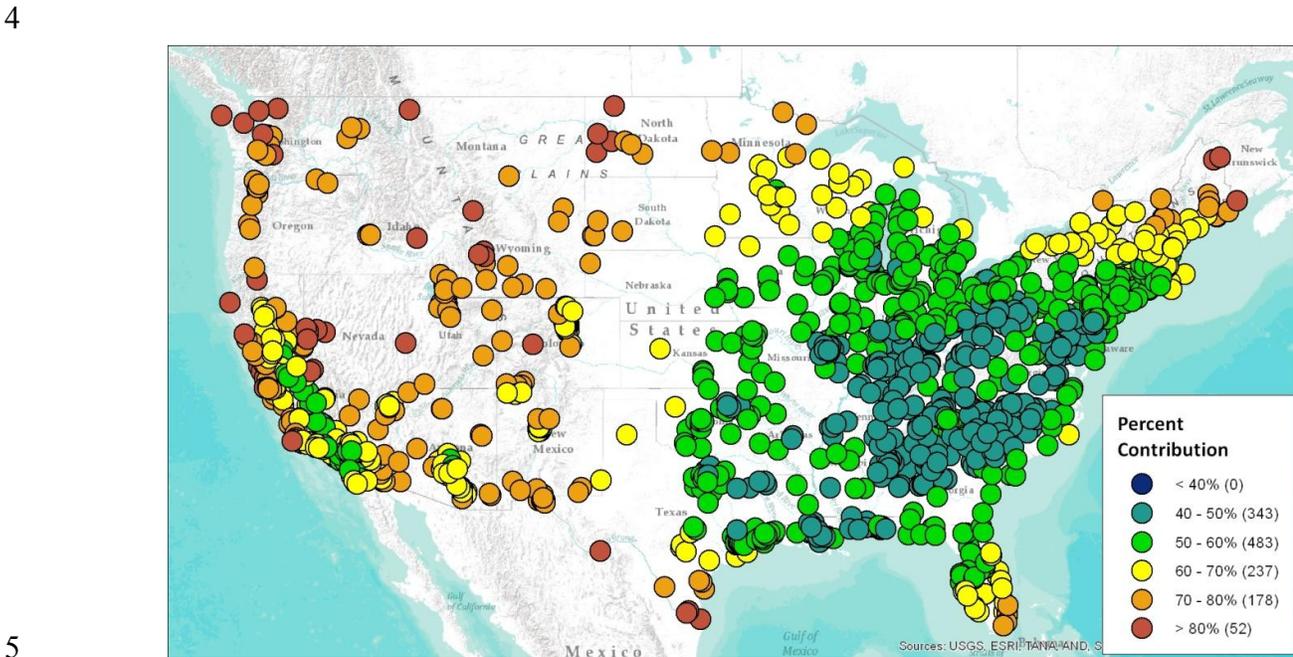
Figure 2-12. Map of apportionment-based U.S. background percent contribution to seasonal mean O$_3$ based on 2007 CAMx source apportionment modeling.

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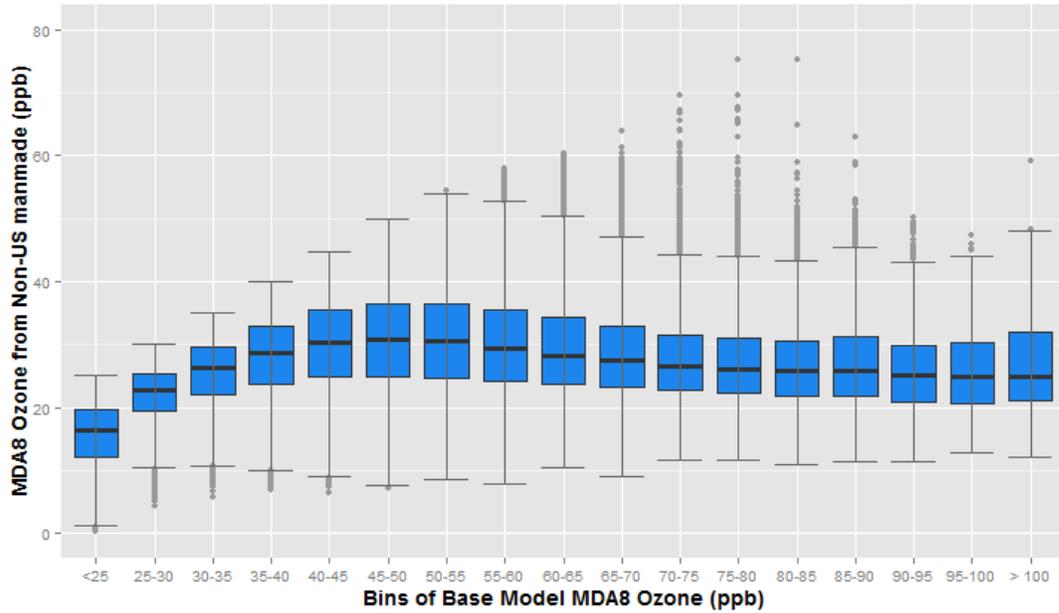
Figure 2-13. Distributions of absolute estimates of apportionment-based U.S. Background (all site-days), binned by modeled MDA8 from the 2007 source apportionment simulation.

5

6
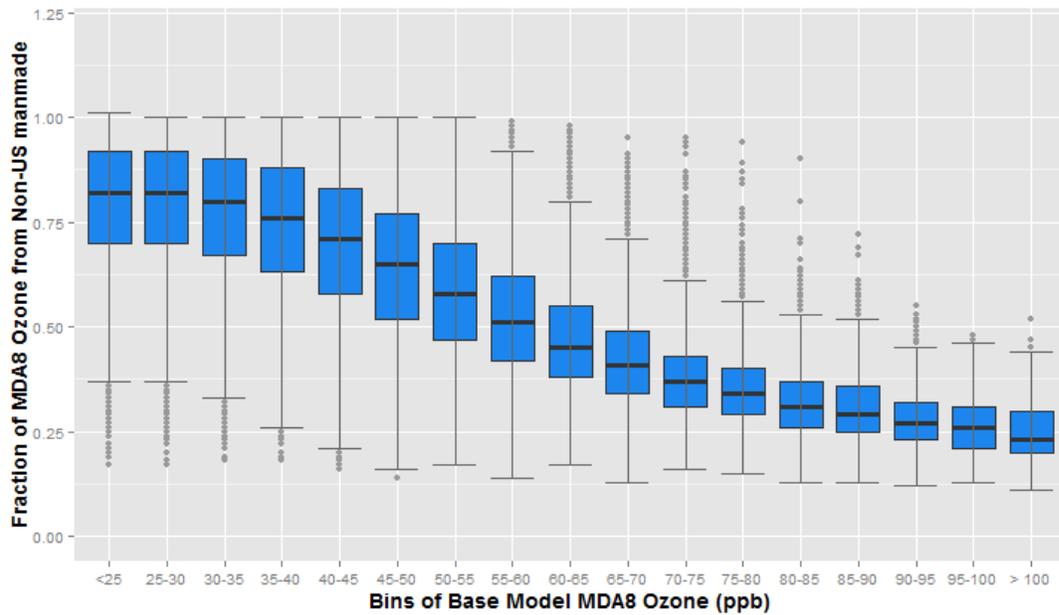
Figure 2-14. Distributions of the relative proportion of apportionment-based U.S. Background to total O₃ (all site-days), binned by modeled MDA8 from the 2007 source apportionment simulation.

1

2.4.4 Proportion of Background O_3 in 12 Urban Case Study Areas

As noted in the introduction, the current approach to estimating changes in risk across various possible levels of the O_3 NAAQS no longer requires a quantitative estimate of background O_3 levels. However, EPA expects to be asked the question: "how much of the total risk is due to background sources." This section of the policy assessment presents estimates of the overall fraction of O_3 that is estimated to result from background sources or processes in each of the 12 urban case study areas considered in the epidemiological-based risk assessment of the REA (US EPA 2014, Chapter 7). The results are based on the recent EPA 2007 source apportionment modeling. Table 2-1 summarizes the estimated fractional contributions of sources other than U.S. anthropogenic emissions (i.e., apportionment-based USB) to total seasonal mean MDA8 O_3 in each of the 12 urban case study areas. The table shows that the fractional contributions from sources other than anthropogenic emissions within the U.S. can range from 43 to 66 percent across these 12 urban areas. These fractions are consistent with the national ratios summarized in section 2.4.2, although the fractions of background are generally smaller at urban sites than at rural sites.

As shown in section 2.4.3, the fractional contributions from background are smaller on days with high modeled O_3 (i.e., days that may exceed the level of the NAAQS). Table 2-2 provides the fractional contributions from these apportionment-based USB sources, only considering days in which base model MDA8 O_3 was greater than 60 ppb. As expected, the fractional background contributions are smaller, ranging from 31 to 55 percent.

Rather than taking the fractions of the seasonal means (as in Table 2-1), Table 2-3 displays the mean and median daily MDA8 background fractions. These metrics may be more appropriate for application to health studies. The fractional contributions to backgrounds calculated via this approach are very similar to the Table 2-1 calculations. Although EPA expects the source apportionment results to provide a more realistic estimate of fractional background values, for completeness, we also provide USB fractions based on the zero-out modeling for the 12 cities (see Table 2-4). The results are similar to the source apportionment findings (Table 2-1), though the zero-out technique provides slightly higher background proportions, as expected. It should be noted that all fractional contributions are based on recent conditions from 2007. These fractional contributions would be expected to change as anthropogenic emissions and O_3 levels are lowered. Based on the source apportionment modeling for these 12 areas, there is evidence that background levels comprise a non-negligible fraction of the total ozone observed within these locations. However, for site-days in which model MDA8 ozone exceeds 60 ppb, ozone formed from U.S. anthropogenic emissions comprise

1   an even larger fraction of the total ozone in 11 of the 12 areas. The major metropolitan areas in

2   the eastern U.S. (e.g., Atlanta, New York City, Philadelphia) are less influenced by background

3   sources than a higher-elevation, western U.S., location like Denver. Even in Denver, though,

4   U.S. anthropogenic emissions have a large influence on total ozone (45 percent).

5   **Table 2-1.   Seasonal mean MDA8 O3 (ppb), seasonal mean apportionment-based USB**
6   **contribution (ppb), and fractional apportionment-based USB contribution to**
7   **total O3 (all site-days) in the 12 REA urban case study areas (%).**

All days, CAMx	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	59.3	54.4	43.0	48.9	47.3	39.1	48.5	51.1	45.4	48.7	46.4	49.8
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.3	25.9	26.2	25.7	31.3	23.3	27.0	29.1	24.5	24.2	29.7	24.3
Fractional contribution from background	0.43	0.48	0.61	0.52	0.66	0.60	0.56	0.57	0.54	0.50	0.64	0.49

8

9   **Table 2-2.   Seasonal mean MDA8 O_3 (ppb), seasonal mean apportionment-based USB**
10   **contribution (ppb), and fractional apportionment-based USB contribution to**
11   **total O_3 (site-days > 60 ppb) in the 12 REA urban study areas (%).**

Only days w/ base MDA8 > 60 ppb	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	74.0	75.3	70.7	72.0	67.5	68.9	70.3	74.4	74.1	74.0	68.3	70.0
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.4	23.7	24.4	25.4	37.3	24.4	28.0	31.9	23.5	22.9	32.1	25.4
Fractional contribution from background	0.34	0.31	0.35	0.35	0.55	0.35	0.40	0.43	0.32	0.31	0.47	0.36

12

13   **Table 2-3.   Fractional contribution of apportionment-based USB in the 12 REA urban**
14   **study areas (%), using the means and medians of daily MDA8 fractions**
15   **(instead of fractions of seasonal means).**

	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Mean of daily MDA8 background fractions	0.46	0.53	0.68	0.58	0.69	0.64	0.59	0.61	0.61	0.56	0.67	0.52
Median of daily MDA8 background fractions	0.43	0.51	0.73	0.54	0.69	0.66	0.59	0.60	0.63	0.54	0.66	0.49

16

1 **Table 2-4.   Seasonal mean MDA8 O$_3$ (ppb), seasonal mean USB contribution (ppb), and**
2 **fractional USB contribution to total O$_3$ (all site-days) in the 12 REA urban case**
3 **study areas (%).**

All days, CMAQ	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	58.6	55.6	45.2	51.8	57.1	43.5	49.4	54.8	47.7	50.5	51.9	52.6
Model MDA8 seasonal mean from USB emissions	30.0	29.9	28.5	31.6	42.2	31.7	33.0	33.3	29.1	29.4	34.4	32.0
Fractional contribution from background	0.51	0.54	0.63	0.61	0.74	0.73	0.67	0.61	0.61	0.58	0.66	0.61

5 ### 2.4.5   Influence of Background O$_3$ on W126 levels

6   EPA also conducted a limited assessment of the impacts of background O$_3$ sources on the

7 W126 metric. The W126 metric (LeFohn et al., 1988) is a cumulative peak-weighted index

8 designed to estimate longer-term effects of daytime ozone levels on sensitive vegetation and

9 ecosystems. EPA used the 2007 zero-out modeling to assess NB, NAB, and USB influences at

10 four sample locations: Atlanta GA, Denver CO, Farmington NM, and Riverside CA. As shown

11 in Figure 2-7, each of the four analyses locations had relatively high observed values of W126 in

12 2010-2012.

13   As discussed above, in previous EPA reviews of the O$_3$ NAAQS, the influence of

14 background ozone was estimated according to a counterfactual assumption. Background O$_3$ was

15 defined as the ozone that would exist in the absence of a particular set of emissions (e.g., NAB is

16 the ozone that would exist if there were no anthropogenic emissions in North America). In the

17 current review, EPA is supplementing the counterfactual assessment with analyses that estimate

18 the fraction of the existing ozone that is due to background sources. This has important

19 ramifications for assessing the influence of background on W126 concentrations, because of the

20 non-linear weighting function used in the metric, which emphasizes high ozone hours (e.g.,

21 periods in which ozone is greater than ~60 ppb). As an example, consider a sample site in the

22 intermountain western U.S. region with very high modeled estimates of U.S background (e.g.,

23 seasonal mean of 45 ppb with some days as high as 65 ppb). Even at this high background

24 location, when the W126 calculation is made for the USB simulation, the resultant annual W126

25 (USB) values are quite low, on the order of 3 ppm-hrs. Sites in the domain with lower U.S.

26 background levels have even smaller USB W126 values, on the order of the 1 ppm-hrs, which is

27 consistent with values mentioned in past reviews (USEPA, 2007). Using the counterfactual

28 scenarios, background ozone has a relatively small impact on W126 levels across the U.S.

29   However, because of the non-linear weighting function used in the W126 calculation, the

30 sum of the W126 from the USB scenario and the W126 resulting from US anthropogenic sources

31 will not equal the total W126. In most cases, the sum of those two components will be

32 substantially less than total W126. As a result, EPA believes it is more informative to estimate

1 the fractional influence of background ozone to W126 levels. Using a methodology that is

2 described in more detail in Appendix A, EPA considered the fractional influence of background

3 ozone on annual W126 levels in four locations. The fractional influence methodology utilizes

4 the 2007 zero-out modeling but places higher weights on background fractions on days that are

5 going to contribute most substantially to the yearly W126 value. Figure 2-15 shows the results.

6 Based on the fractional influence methodology, natural background sources are estimated to

7 contribute 29-50% of the total modeled W126 with the highest relative influence in the

8 intermountain western U.S. (e.g., Farmington NM) and the lowest relative influence in the

9 eastern U.S. (e.g., Atlanta). U.S. background is estimated to contribute 37-65% of the total

10 modeled W126. The proportional impacts of background are slightly less for the W126 metric

11 than for seasonal mean MDA8 (discussed in section 2.4.2), because of the sigmoidal weighting

12 function that places more emphasis on higher ozone days when background fractions are

13 generally lower.

14   The key conclusion from this cursory analysis is that background ozone may comprise a

15 non-negligible portion of current W126 levels across the U.S. These fractional influences are

16 greatest in the intermountain western U.S. and are slightly smaller than the seasonal mean

17 MDA8 metric. In the counterfactual cases, when non-background sources are completely

18 removed, the remaining W126 levels are low (< 3 ppm-hrs).
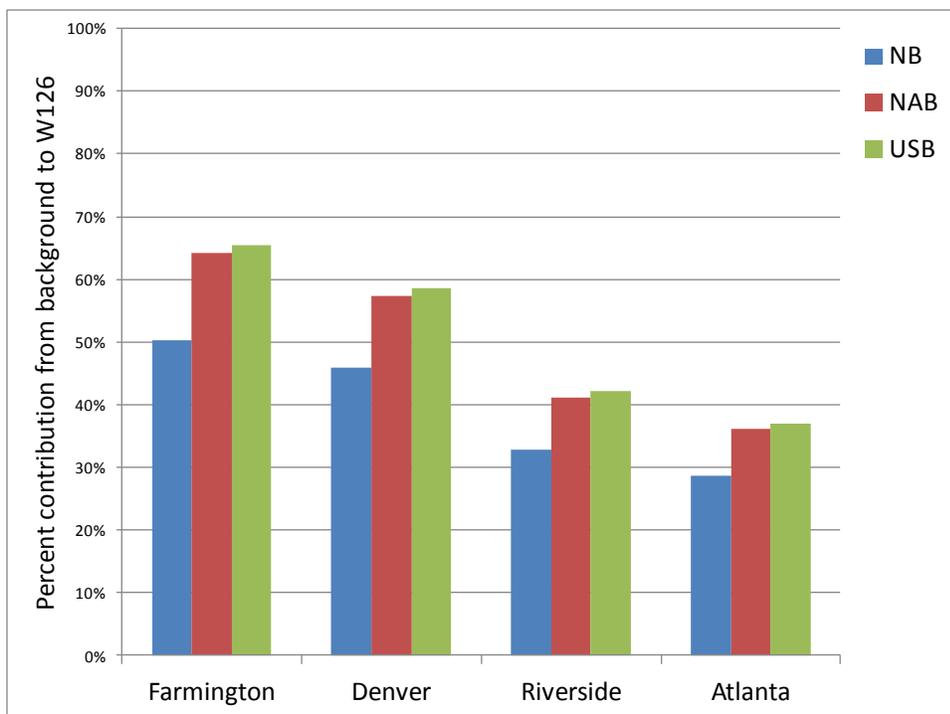
19

20 **Figure 2-15. Fractional influence of background sources to W126 levels in four sample**
21   **locations. Model estimates based on 2007 CMAQ zero-out modeling.**

1   **2.4.6   Estimated Magnitude of Individual Components of Background O_3**

2   While local and regional controls of manmade O_3 precursor emissions are still expected

3   to be the most effective mechanism for reducing local O_3 levels, an understanding of the relative

4   contributions of various background elements can be instructive in determining ways to mitigate

5   the impact of background. This section will utilize the supplemental 2007 air quality modeling

6   estimates to consider the relative importance of specific elements of background O_3. Several

7   background elements were isolated in either the zero-out or source apportionment modeling.

8   Appendix A provides more detail on these analyses. In conjunction with the previous analyses

9   summarized in the ISA, some broad characterizations of the individual components of

10   background O_3 can be developed.

11   The recent 2007 EPA modeling confirms the importance of methane emissions in

12   contributing to background O_3. Methane has an atmospheric lifetime of about a decade and can

13   be an important contributor to ozone on longer time scales. Anthropogenic methane emission

14   sources include agriculture, coal mines, landfills, and natural gas and oil systems. The difference

15   between the NAB and NB zero-out scenarios provides an estimate of contributions from

16   international anthropogenic emissions and anthropogenic methane, which is modeled by

17   reducing model concentrations from present-day values to pre-industrial levels. The ISA (US

18   EPA, 2013, section 3.4) estimated that roughly half of the difference between the NB and NAB

19   scenarios resulted from the removal of anthropogenic methane emissions and that the other half

20   resulted from international anthropogenic emissions of shorter-lived O_3 precursors (e.g., NOx

21   and nmVOC). Figure 2-16 shows the difference in seasonal mean MDA8 O_3 levels between the

22   NB and NAB scenarios. North American seasonal mean background is 6-15 ppb higher than

23   comparable natural background levels. The most frequent increment is an 8-10 ppb increase

24   when the methane is increased and the non-North American emissions are re-added. It is not

25   possible via the EPA 2007 modeling to parse out what fraction of this change is due to emissions

26   outside of North America, as opposed to the fraction due to anthropogenic methane emissions,

27   but the modeling suggests that both of these terms have the potential to contribute in an

28   important way to average background levels in the U.S.

29   The difference between the NAB and USB scenarios is easier to interpret as it only

30   involves one change, the inclusion of anthropogenic emissions from the in-domain portion of

31   Canada and Mexico. These emissions (not shown here, but depicted in Appendix A) contribute

32   less than 2 ppb to the seasonal mean MDA8 O_3 levels over most of the U.S. There are 70 sites,

33   near an international border, where the modeling estimates Canadian/Mexican seasonal average

34   impacts of 2-4 ppb. Peak single-day MDA8 impacts from these specific international emissions

35   sources can approach 25 ppb (e.g., San Diego, Buffalo NY).

36

Ozone (ppb)
- < 1 (0)
- 1 - 2 (0)
- 2 - 3 (0)
- 3 - 4 (0)
- 4 - 6 (0)
- 6 - 8 (141)
- 8 - 10 (821)
- 10 - 12 (267)
- 12 - 15 (65)
- > 15 (0)
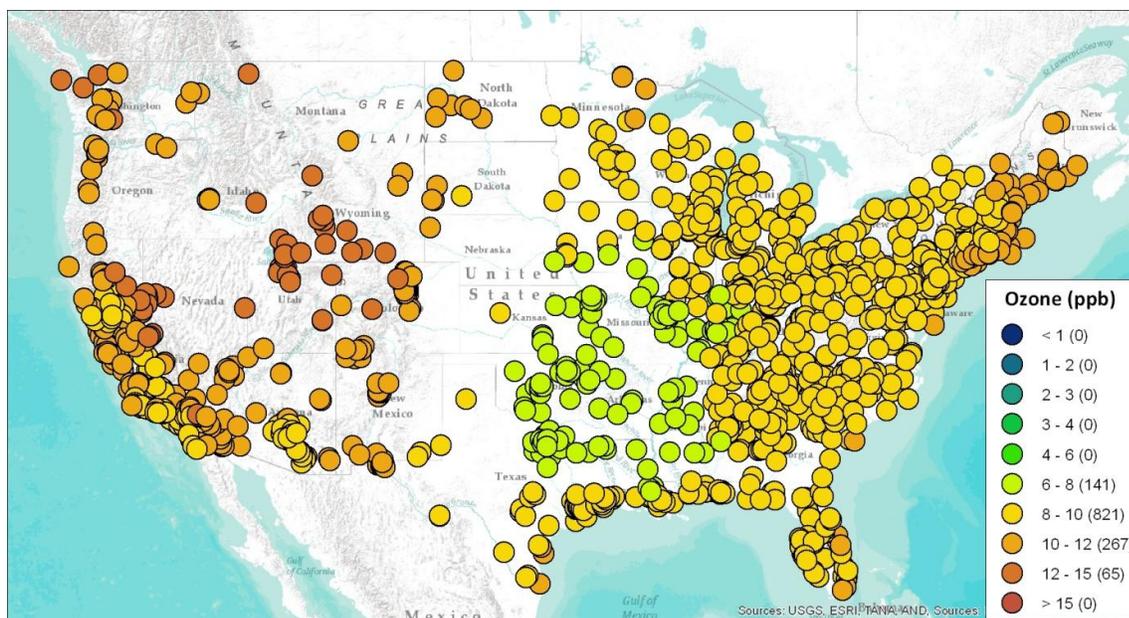
Figure 2-16. Differences in seasonal mean O$_3$ between the NAB and NB scenarios.

Eleven separate source categories were tracked in the source apportionment modeling, including five boundary condition terms (East, South, West, North, and top) and six emissions sectors within the domain. The contributions of each of these terms is provided in the Appendix and summarized below. At most locations, the five model boundary terms contributed an aggregate 40-60 percent of the total seasonal mean MDA8 O$_3$ across the U.S. The highest proportional impacts from the boundary conditions are along the coastlines and the intermountain West. The O$_3$ entering the model domain via the boundary conditions can have a variety of origins including: a) natural sources of O$_3$ and precursors emanating from outside the domain, b) anthropogenic sources of O$_3$ precursors emanating from outside the domain, and c) some fraction of U.S. emissions (natural and anthropogenic) which exit the model domain but get re-imported into the domain via synoptic-scale recirculation. Accordingly, it is not possible to relate the boundary condition contribution to any specific background element. The single largest sector that was tracked in the source apportionment modeling was U.S. anthropogenic emissions. Figure 2-17 shows the contributions from this sector to seasonal mean MDA8 O$_3$ levels. Over most of the U.S. this term contributes 40-60 percent to the total seasonal mean O$_3$. As discussed in section 2.4.3, these contributions are even higher when only high O$_3$ days are considered. International shipping emissions, as well as fires and other biogenic emissions also contribute in a non-negligible way to background O$_3$ over the U.S. The key finding from this analysis is that air quality planning efforts to reduce anthropogenic methane emissions and international NOx/nmVOC emissions (e.g., migrating from Asia, Canada, and Mexico; and from

1   commercial shipping) have the potential to lower background levels and ease eventual attainment
2   of the NAAQS.
3
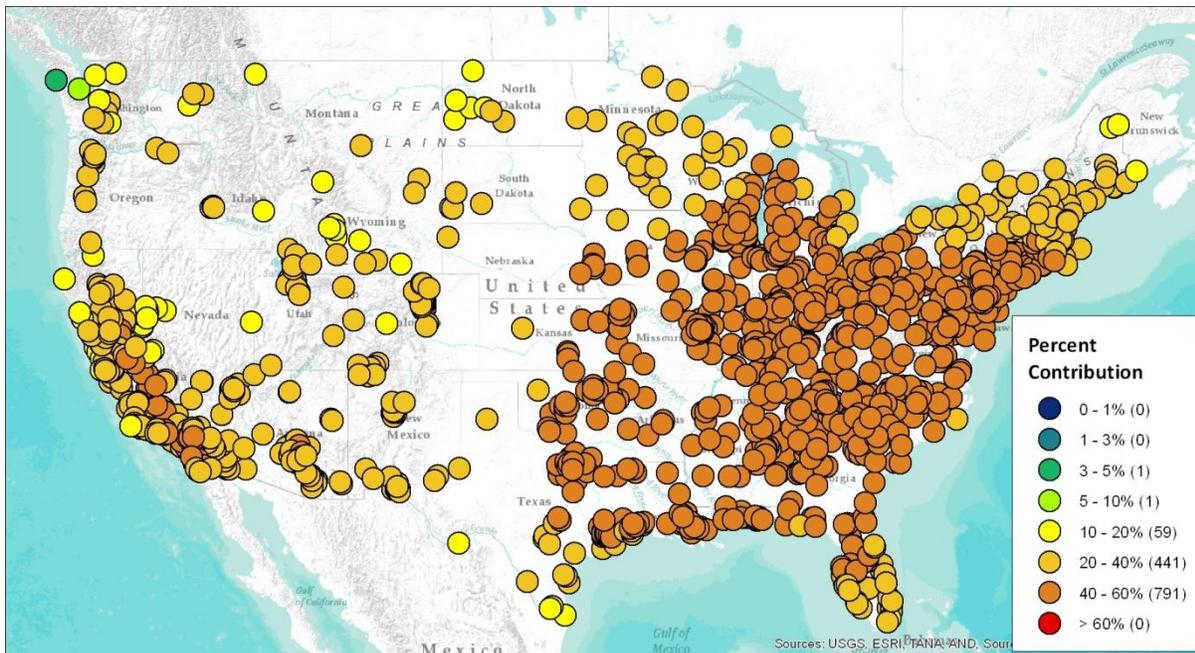
4

5   **Figure 2-17. Percent contribution of U.S. anthropogenic emissions to total seasonal mean**
6   **MDA8 O$_3$ levels, based on 2007 source apportionment modeling.**

7

8   ### 2.4.7   Summary

9   For a variety of reasons, it is challenging to present a comprehensive summary of all the
10   components and implications of background O$_3$. In many forums the term "background" is used
11   generically and the lack of specificity can lead to confusion as to what sources are being
12   considered. Additionally, it is well established that the impacts of background sources can vary
13   greatly over space and time which makes it difficult to present a simple summary of background
14   O$_3$ levels. Further, background O$_3$ can be generated by a variety of processes, each of which can
15   lead to differential patterns in space and time, and which often have different regulatory
16   ramifications. Finally, background O$_3$ is difficult to measure and thus, typically requires air
17   quality modeling which has inherent uncertainties and potential errors and biases.
18   That said, EPA believes the following concise and four-stage summary of the
19   implications of background O$_3$ on the NAAQS review is appropriate, as based on previous
20   modeling exercises and the more recent EPA analyses summarized herein. First, background O$_3$
21   exists and can comprise a considerable fraction of total seasonal mean O$_3$ and W126 across the

1   U.S. Air quality models can estimate the fractional contribution of backgroud sources to total O_3

2   in an individual area. The largest absolute values of background (NB, NAB, USB, or

3   apportionment-based USB) are modeled to occur at locations in the intermountain western U.S.

4   and are maximized in the spring and early summer seasons. Second, the modeling indicates that

5   U.S. anthropogenic emission sources are the dominant contributor to the majority of modeled O_3

6   exceedances of the NAAQS. Higher O_3 days generally have smaller fractional contributions

7   from background. This finding indicates that the relative importance of background O_3 would

8   increase were O_3 concentrations to decrease with a lower level of the O_3 NAAQS. Third, while

9   the majority of modeled O_3 exceedances have local and regional emissions as their primary

10   cause, there can be events where O_3 levels approach or exceed 60-75 ppb due to background

11   sources. These events are relatively infrequent and EPA has policies that could allow for the

12   exclusion of air quality monitoring data affected by these types of events from design value

13   calculations. Fourth and finally, the Clean Air Act requires the NAAQS to be set at a level

14   requisite to protect public health and welfare. Proximity to background levels could be an

15   additional consideration, but only where it would support a decision based on the health evidence

16   and analyses.

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3   Adequacy of the Current Primary Standard

This chapter presents staff's considerations and conclusions regarding the adequacy of the current primary O_3 NAAQS. In doing so, we pose the following overarching question:

> **Does the currently available scientific evidence and exposure/risk information, as reflected in the ISA and HREA, support or call into question the adequacy of the current O_3 standard?**

In addressing this overarching question, we pose a series of more specific questions, as discussed in sections 3.1 through 3.4 below. Section 3.1 presents our consideration of the available scientific evidence (i.e., evidence-based considerations) about the health effects associated with short- and long-term O_3 exposures. Section 3.2 presents our consideration of available estimates of O_3 exposures and health risks (exposure- and risk-based considerations). Section 3.3 discusses the advice and recommendations that we have received from the CASAC on the first draft O_3 PA, and on documents from previous reviews of the O_3 NAAQS. Section 3.4 revisits the overarching question of this section, and presents staff's conclusions regarding the adequacy of the current primary O_3 NAAQS.

3.1   EVIDENCE-BASED CONSIDERATIONS

This section presents our consideration of the available scientific evidence with regard to the adequacy of the current O_3 standard. Our approach, as summarized in section 1.3.1 above, is based on the full body of evidence in this review. We use information from the full evidence base to characterize our confidence in the extent to which O_3-attributable effects occur, and the extent to which such effects are adverse, over the ranges of O_3 exposure concentrations evaluated in controlled human exposure studies and over the distributions of ambient O_3 concentrations in locations where epidemiologic studies have been conducted. In doing so, we recognize that the available health effects evidence reflects a continuum from relatively high O_3 concentrations, at which scientists generally agree that adverse health effects are likely to occur, through lower concentrations, at which the likelihood and magnitude of a response become increasingly uncertain.

1   Section 3.1.1 summarizes a mode of action framework for understanding the effects of

2   both short- and long-term O_3 exposures, based on Chapter 5 of the ISA (U.S. EPA, 2013).

3   Section 3.1.2 presents our consideration of the evidence for health effects attributable to short-

4   term and long-term O_3 exposures. Section 3.1.3 discusses the adversity of the effects. Section

5   3.1.4 presents our consideration of evidence with regard to concentrations associated with health

6   effects and section 3.1.5 presents our consideration of the public health implications of exposures

7   to O_3, including the adversity of effects and evidence for at-risk populations and lifestages.[1]

3.1.1 Modes of Action

9   Our consideration of the evidence of effects attributable to short-and long-term exposures

10   and the factors that increase risk in populations and lifestages builds upon evidence about the

11   modes of action by which O_3 exerts effects (U.S. EPA, 2013; section 5.3). Mode of action refers

12   to a sequence of key events and processes that result in a given toxic effect; elucidation of

13   mechanisms provides a more detailed understanding of these key events and processes. The

14   purpose of this section is to describe the key events and pathways that contribute to health effects

15   resulting from both short-term and long-term exposures to O_3. The extensive research carried out

16   over several decades in humans and animals has yielded numerous studies on mechanisms by

17   which O_3 exerts its effects. It is well-understood that secondary oxidation products, which form

18   as a result of O_3 exposure, initiate numerous responses at the cellular, tissue and whole organ

19   level of the respiratory system. These responses include the activation of neural reflexes,

20   initiation of inflammation, alteration of barrier epithelial function, sensitization of bronchial

21   smooth muscle, modification of lung host defenses, and airways remodeling, as discussed below.

22   These key events have the potential to affect other organ systems such as the cardiovascular

23   system. It has been proposed that secondary oxidation products, which are bioactive and

24   cytotoxic in the respiratory system, are also responsible for systemic effects. Recent studies in

25   animal models show that inhalation of O_3 results in systematic oxidative stress.

26   Figure 3.1 below, adapted from Figure 5-8 of the ISA (ISA, Section 5.3.10, U.S. EPA,

27   2013), shows key events in the toxicity pathway of O_3 that are described in more detail in

28   Appendix 3-A. The initial key event in the toxicity pathway of O_3 is the formation of secondary

---

[1]The term "at-risk populations" includes the factor lifestages, specifically childhood and older adulthood, that are
experienced by most people over the course of a lifetime, unlike other factors associated with at-risk populations.

1   oxidation products in the respiratory tract (ISA, section 5-3, U.S. EPA, 2013). This mainly

2   involves direct reactions with components of the extracellular lining fluid (ELF). Although the

3   ELF has inherent capacity to quench (based on individual antioxidant capacity), this attenuative

4   capacity can be overwhelmed, especially with exposure to elevated concentrations of O_3[2].

5   The resulting secondary oxidation products transmit signals to the epithelium, pain receptive

6   nerve fibers and, if present, immune cells (i.e., eosinophils, dendritic cells and mast cells)

7   involved in allergic responses. Thus, the effects of O_3 are mediated by components of ELF and

8   by the multiple cell types found in the respiratory tract. Further, oxidative stress[3] is an implicit

9   part of this initial key event.

Mode of Action/Possible Pathways

Ozone + Respiratory Tract

Formation of secondary oxidation products

| Activation of neural reflexes | Initiation of inflammation | Alteration of epithelial barrier function | Sensitization of bronchial smooth muscle | Modification of innate and adaptive immunity | Airways remodeling |

Systemic inflammation and oxidative/nitrosative stress

Extrapulmonary Effects

Decrements in pulmonary function
Pulmonary inflammation/oxidative stress
Increases in airways permeability
Airways hyperresponsiveness
Exacerbation/induction of asthma
Decreased host defenses
Epithelial metaplasia and fibrotic changes
Altered lung development

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[2] The ELF is a complex mixture of lipids (fats), proteins, and antioxidants that serve as the first barrier and target for inhaled O_3. The quenching ability of antioxidant substances present in the ELF appear in most cases to limit interaction of O_3 with underlying tissues and to prevent penetration of O_3 deeper into the lung. However, as the ELF thickness decreases and becomes ultra thin in the alveolar region, it may be possible for direct interaction of O_3 with the underlying epithelial cells to occur. The formation of secondary oxidation products is likely related to the concentration of antioxidants present and the quenching ability of the lining fluid.

[3] Oxidative stress reflects an imbalance between the systemic manifestation of reactive oxygen species, such as superoxides, and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage.

Figure 3-1. Modes of action/possible pathways underlying the health effects resulting from inhalation exposure to O_3. (Adapted from U.S. EPA, 2013, Figure 5-8)

Figure 3-1 illustrates pathways identified in the ISA by which the array of key events identified may lead to health effects associated with inhalation exposure to O_3. For example, the activation of neural reflexes, which may be triggered by secondary oxidative products, lead to lung function decrements and may play a role in extrapulmonary effects such as slow resting heart rate or bradycardia. Secondary oxidation products have also been implicated in the initiation of inflammation and inflammation further contributes to O_3-mediated oxidative stress. Alteration of epithelial barrier function may play a role in allergic sensitization and in enhanced sensitization of bronchial smooth muscle, resulting in airways hyperresponsiveness; genetic susceptibility has been associated with this pathway. In addition to genetic factors, pre-existing conditions and diseases, nutritional status, lifestage and co-exposures may affect multiple key events in Figure 3-1 and contribute to altered risk of O_3-induced effects (U.S. EPA, 2013, section 5.4). Evidence also indicates that exposure to O_3 modifies innate and adaptive immunity; such effects can result in both short- and longer-term consequences related to the exacerbation and/or induction of asthma and to alterations in host defense. Another event, airways remodeling, has been demonstrated following chronic and/or intermittent exposure to O_3 in animal models. Additionally, there is evidence that O_3 exposure results in systemic inflammation and vascular oxidative/nitrosative stress, which may lead to downstream systemic responses (U.S. EPA, 2013, section 2.4).

Experimental evidence for such O_3-induced changes contributes to our understanding of the biological plausibility of adverse O_3-related health effects, including a range of respiratory effects as well as effects outside the respiratory system (e.g., cardiovascular effects) (U.S. EPA, 2013, Chapters 6 and 7). The range of respiratory effects that could be mediated by the secondary oxidation products formed following reactions with O_3 include decrements in pulmonary function; pulmonary inflammation and injury; increased airway permeability; airway hyperresponsiveness; decreased lung host defense, exacerbation and/or induction of asthma; and alterations in pulmonary structure and/or development (Figure 3-1, above). These effects are logically linked to the types of adverse O_3-attributable effects evaluated and observed in epidemiologic studies, including respiratory symptoms, respiratory hospital admissions and emergency department visits, and premature mortality (U.S. EPA, 2013, Chapters 6 and 7). Moreover, it has been proposed that some of these key events, including O_3-mediated systemic oxidative stress and activation of neural reflexes, are linked to the extrapulmonary effects of O_3 that have been noted for decades (U.S. EPA, 2013, section 5.3.2 and 5.3.8). Further, interindividual variability in the various key events (e.g., due to genetic variants or diet affecting

1   antioxidant defenses) illustrated in Figure 3-1 may contribute to differences in susceptibility to

2   O_3 health effects (ISA, section 5.4).

3   **3.1.2   Nature of Effects**

4   •   **To what extent does the currently available scientific evidence alter or strengthen**
5   **our conclusions from the last review regarding health effects attributable to O_3**
6   **exposure in ambient air? Are previously identified uncertainties reduced or do**
7   **important uncertainties remain?**

8   The health effects of ozone are described in detail in the assessment of the evidence

9   available in this review which is largely consistent with conclusions of past CDs. In some

10   categories of health effects, there is newly available evidence regarding some aspects of the

11   effects described in the last review or that strengthens our conclusions regarding aspects of O_3

12   toxicity on a particular physiological system (U.S. EPA, 2013, Table 1-1). A sizeable number of

13   studies on O_3 health effects are newly available in this review and are critically assessed in the

14   ISA as part of the full body of evidence. Based on this assessment, the ISA determined that a

15   causal relationship[4] exists between short-term exposure to O_3 in ambient air[5] and effects on the

16   respiratory system and that a likely to be causal relationship[6] exists between long-term exposure

17   to O3 in ambient air and respiratory effects (U.S. EPA 2013, pp. 1-6 to 1-7). As stated in the

18   ISA, "[c]ollectively, a very large amount of evidence spanning several decades supports a

19   relationship between exposure to O_3 and a broad range of respiratory effects" (ISA, p. 1-6).

20   Additionally, the ISA determined likely to be causal relationships exist between short-term

21   exposures to O_3 in ambient air and both total mortality and cardiovascular effects, based on

22   expanded evidence bases in the current review (U.S. EPA, 2013, pp. 1-7 to 1-8). In the ISA, EPA

23   additionally determined that the currently available evidence for additional endpoints is

24   suggestive of causal relationships between short-term (central nervous system effects) and long-

25   term exposure (cardiovascular effects, central nervous system effects and total mortality) to

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[4] Since the last O3 NAAQS review, the ISAs which have replaced CDs in documenting each review of the scientific evidence (or air quality criteria) employ a systematic framework for weighing the evidence and describing associated conclusions with regard to causality, using established descriptors, as summarized in section 1.3.1 above (U.S. EPA, 2013, Preamble).

[5] In determining that a causal relationship exists for O_3 with specific health effects, EPA has concluded that "[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures" (ISA, p. lxiv).

[6] In determining a likely to be a causal relationship exists for O_3 with specific health effects, EPA has concluded that "[e]vidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain" (ISA, p. lxiv).

1   ambient O_3. Consistent with emphasis in past reviews on O_3 health effects for which the evidence

2   is strongest, we place the greatest emphasis on studies of health effects that have been judged in

3   the ISA to be caused by, or likely to be caused by, O_3 exposures (U.S. EPA, 2013, section 2.5.2).

4   This section presents our consideration of the evidence for health effects attributable to

5   O_3 exposures, including respiratory morbidity and mortality effects attributable to short- and

6   long-term exposures, and cardiovascular system effects (including mortality) and total mortality

7   attributable to short-term exposures. We focus particularly on considering the extent to which the

8   scientific evidence available in the current review has been strengthened since the last review,

9   and the extent to which important uncertainties and limitations in the evidence from the last

10   review have been addressed. In section 3.1.2.2, we then consider the extent to which the

11   available evidence indicates health effects may be attributable to ambient O_3 concentrations

12   likely to be allowed by the current O_3 NAAQS. In this section, we address the following specific

13   question for each category of health effects considering the evidence available in the 2008

14   review of the standard as well as evidence that has become available since then. The ISA

15   summarizes the longstanding body of evidence for O_3 respiratory effects as follows (U.S. EPA,

16   2013, p. 1-5).

17   *The clearest evidence for health effects associated with exposure to O_3 is provided*
18   *by studies of respiratory effects. Collectively, a very large amount of evidence*
19   *spanning several decades supports a relationship between exposure to O3 and a*
20   *broad range of respiratory effects (see Section 6.2.9 and Section 7.2.8). The*
21   *majority of this evidence is derived from studies investigating short-term*
22   *exposures (i.e., hours to weeks) to O_3, although animal toxicological studies and*
23   *recent epidemiologic evidence demonstrate that long-term exposure (i.e., months*
24   *to years) may also harm the respiratory system.*

25   The extensive body of evidence supporting a causal relationship between short-term O_3

26   exposures and respiratory effects is discussed in detail in Chapter 6 of the ISA (U.S. EPA, 2013),

27   while evidence for respiratory effects associated with long-term or repeated O_3 exposures are

28   discussed in chapter 7 of that document (U.S., EPA, 2013).

29   **3.1.2.1   Respiratory Effects – Short-term Exposures**

30   •   **To what extent does the currently available scientific evidence, including related**
31   **uncertainties, strengthen or alter our understanding from the last review of respiratory**
32   **effects attributable to short-term O_3 exposures?**

1   The 2006 O_3 AQCD concluded that there was clear, consistent evidence of a causal

2   relationship between short-term O_3 exposure and respiratory effects (U.S. EPA, 2006b). This

3   conclusion was substantiated by evidence from controlled human exposure and toxicological

4   studies indicating a range of respiratory effects in response to short-term O_3 exposures, including

5   pulmonary function decrements and increases in respiratory symptoms, lung inflammation, lung

6   permeability, and airway hyperresponsiveness. Toxicological studies provided additional

7   evidence for O_3-induced impairment of host defenses. Combined, these findings from

8   experimental studies provided support for epidemiologic evidence, in which short-term increases

9   in ambient O_3 concentration were consistently associated with decreases in lung function in

10   populations with increased outdoor exposures, especially children with asthma and healthy

11   children; increases in respiratory symptoms and asthma medication use in children with asthma;

12   and increases in respiratory-related hospital admissions and asthma-related ED visits (U.S. EPA,

13   2013, pp. 6-1 to 6-2).

14   As discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.9), studies evaluated since

15   the completion of the 2006 O_3 AQCD support and expand upon the strong body of evidence that,

16   in the last review, indicated a causal relationship between short-term O_3 exposures and

17   respiratory health effects. Recent controlled human exposure studies conducted in young, healthy

18   adults with moderate exertion have reported FEV_1 decrements and pulmonary inflammation

19   following prolonged exposures to O_3 concentrations as low as 60 ppb, and respiratory symptoms

20   following exposures to concentrations as low as 70 ppb. Epidemiologic studies provide evidence

21   that increases in ambient O_3 exposures can result in lung function decrements, increases in

22   respiratory symptoms, and pulmonary inflammation in children with asthma; increases in

23   respiratory-related hospital admissions and emergency department visits; and increases in

24   respiratory mortality. Some of these studies report such associations even for O_3 concentrations

25   at the low end of the distribution of daily concentrations. Recent epidemiologic studies report

26   that associations with respiratory morbidity and mortality are stronger during the warm/summer

27   months and remain robust after adjustment for copollutants. Recent toxicological studies

28   reporting O_3-induced inflammation, airway hyperresponsiveness, and impaired lung host defense

29   continue to support the biological plausibility and modes of action for the O_3-induced respiratory

30   effects observed in the controlled human exposure and epidemiologic studies. Further support is

31   provided by recent studies that found O_3-associated increases in indicators of airway

1   inflammation and oxidative stress in children with asthma (U.S. EPA, 2013, section 6.2.9).

2   Together, epidemiologic and experimental studies support a continuum of respiratory effects

3   associated with O_3 exposure that can result in respiratory-related emergency department visits,

4   hospital admissions, and/or mortality (U.S. EPA, 2013, section 6.2.9).

5   Across respiratory endpoints, evidence indicates antioxidant capacity may modify the

6   risk of respiratory morbidity associated with O_3 exposure (U.S. EPA, 2013, section 6.2.9, p. 6-

7   161). The potentially elevated risk of populations with diminished antioxidant capacity and the

8   reduced risk of populations with sufficient antioxidant capacity identified in epidemiologic

9   studies are strongly supported by similar findings from controlled human exposure studies and

10   by evidence that characterizes O_3-induced decreases in intracellular antioxidant levels as a mode

11   of action for downstream effects.

12   We describe key aspects of this evidence below with regard to lung function decrements;

13   pulmonary inflammation, injury, and oxidative stress; airway hyperresponsiveness; respiratory

14   symptoms and medication use; lung host defense; allergic and asthma-related responses; hospital

15   admissions and emergency department visits; and respiratory mortality.

16   **Lung Function Decrements**

17   In the 2008 review, a large number of controlled human exposure studies[7] reported O_3-

18   induced lung function decrements in young, healthy adults engaged in intermittent, moderate

19   exertion following 6.6 hour exposures to O_3 concentrations at or above 80 ppb. Although two

20   studies also reported effects following exposures to lower concentrations, an important

21   uncertainty in the last review was the extent to which exposures to O_3 concentrations below 80

22   ppb result in lung function decrements. In addition, in the last review epidemiologic panel

23   studies had reported O_3-associated lung function decrements in a variety of different populations

24   (e.g., children, outdoor workers) likely to experience increased exposures. In the current review,

25   additional controlled human exposure studies are available that have evaluated exposures to O_3

26   concentrations of 60 or 70 ppb. The available evidence from controlled human exposure and

27   panel studies is assessed in detail in the ISA (U.S. EPA, section 6.2.1) and is summarized below.

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[7] The controlled human exposure studies discussed in this PA utilize only healthy adult subjects. In the absence of
controlled human exposure data for children, HREA estimates of lung function decrements are based on the
assumption that children exhibit the same lung function responses following O_3 exposures as healthy 18 year olds
(U.S. EPA, 2014, section 6.2.4 and 6.5). Thus, the conclusions about the occurrence of lung function decrements
that follow generally apply to children as well as adults.

1   Controlled exposures to O_3 concentrations that can be found in the ambient air can result

2   in a number of lung function effects, including decreased inspiratory capacity; mild

3   bronchoconstriction; and rapid, shallow breathing patterns during exercise. Reflex inhibition of

4   inspiration results in a decrease in forced vital capacity (FVC) and total lung capacity (TLC) and,

5   in combination with mild bronchoconstriction, contributes to a decrease in the forced expiratory

6   volume in 1 second (FEV_1) (U.S. EPA, 2013, section 6.2.1.1). Accumulating evidence indicates

7   that such effects are mediated by activation of sensory nerves, resulting in the involuntary

8   truncation of inspiration and a mild increase in airway obstruction due to bronchoconstriction

9   (U.S. EPA, 2013, section 5.3.10).

10   Data from controlled human exposure studies indicate that increasing the duration of O_3

11   exposures and increasing ventilation rates decreases the O_3 exposure concentrations required to

12   impair lung function. Ozone exposure concentrations well above those typically found in

13   ambient air are required to impair lung function in healthy resting adults, while exposure to O_3

14   concentrations at or below those in the ambient air have been reported to impair lung function in

15   healthy adults exposed for longer durations while undergoing intermittent, moderate exertion

16   (U.S. EPA, 2012a, section 6.2.1.1). With repeated O_3 exposures over several days, FEV_1

17   responses become attenuated in both healthy adults and adults with mild asthma, though this

18   attenuation of response is lost after about a week without exposure (U.S. EPA, 2013, section

19   6.2.1.1; page 6-27).

20   When considering controlled human exposures studies of O_3-induced lung function

21   decrements we evaluate both group mean changes in lung function and the interindividual

22   variability in the magnitude of responses. To the extent studies report statistically significant

23   decrements in mean lung function following O_3 exposures after controlling for other factors, we

24   have more confidence that measured decrements are due to the O_3 exposure itself, rather than

25   chance alone. As discussed below, group mean changes in lung function are often small,

26   especially following exposures to relatively low O_3 concentrations (e.g., 60 ppb). However, even

27   when group mean decrements in lung function are small, some individuals could experience

28   decrements that are "clinically meaningful" (Pellegrino et al., 2005; ATS, 1991; EPA, 2010)

29   with respect to criteria for spirometric testing, and/or that could be considered "adverse" with

30   respect to public health policy decisions. See section 3.1.3 below.

1   At the time of the last review, a number of controlled human exposure studies had

2 reported lung function decrements in young, healthy adults following prolonged (6.6-hour)

3 exposures while at moderate exertion to O_3 concentrations at and above 80 ppb. In addition,

4 there were two controlled human exposure studies by Adams (2002, 2006) that examined lung

5 function effects following exposures to O_3 concentrations of 60 ppb. The EPA's analysis of the

6 data from the Adams (2006) study reported a small but statistically significant O_3-induced

7 decrement in group mean FEV_1 following exposures of young, healthy adults, while at moderate

8 exertion, to 60 ppb O_3, when compared with filtered air controls (Brown, 2007). [8] Further

9 examination of the post-exposure FEV_1 data, and mean data for other time points and other

10 concentrations, indicated that the temporal pattern of the response to 60 ppb O_3 was generally

11 consistent with the temporal patterns of responses to higher O_3 concentrations in this and other

12 studies. (75 FR 2950, January 19, 2010). This suggested a pattern of response following

13 exposures to 60 ppb O_3 that was consistent with a dose-response relationship, rather than random

14 variability. See also *State of Mississippi v. EPA*, 723 F. 3d at 259 (upholding EPA's

15 interpretation of the Adams studies).

16   Figure 6-1 in the ISA summarizes the currently available evidence from multiple

17 controlled human exposure studies evaluating group mean changes in FEV_1 following prolonged

18 O_3 exposures (i.e., 6.6 hours) in young, healthy adults engaged in moderate levels of physical

19 activity (U.S. EPA, 2012, section 6.2.1.1). With regard to the group mean changes reported in

20 these studies, the ISA specifically notes the following (U.S. EPA, 2012a, section 6.2.1.1, Figure

21 6-1):

22   1. Prolonged exposure to 40 ppb O_3 results in a small decrease in group mean FEV_1 that is
23   not statistically different from responses following exposure to filtered air (Adams, 2002;
24   Adams, 2006).

25   2. Prolonged exposure to an average O_3 concentration of 60 ppb results in group mean FEV_1
26   decrements ranging from 1.8% to 3.6% (Adams 2002; Adams, 2006;[9] Schelegle et al.,
27   2009; Kim et al., 2011). Based on data from multiple studies, the weighted average group
28   mean decrement was 2.7%. In some analyses, these group mean decrements in lung

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[8] Adams (2006a) did not find effects on FEV1 at 60 ppb to be statistically significant. In an analysis of the Adams (2006a) data, even after removal of potential outliers, Brown et al. (2008) found the average effect on FEV1 at 60 ppb to be small, but highly statistically significant (p < 0.002) using several common statistical tests.

[9] Adams (2006); (2002) both provide data for an additional group of 30 healthy subjects that were exposed via facemask to 60 ppb (square-wave) O_3 for 6.6 hours with moderate exercise (   ) = 23 L/min per m^2 BSA). These subjects are described on page 133 of Adams (2006) and pages 747 and 761 of Adams (2002). The FEV_1 decrement may be somewhat increased due to a target (   ) of 23 L/min per m^2 BSA relative to other studies with which it is listed having the target (   ) of 20 L/min per m^2 BSA. The facemask exposure is not expected to affect the FEV_1 responses relative to a chamber exposure.

1   function were statistically significant (Brown et al., 2008; Kim et al., 2011), while in
2   other analyses they were not (Adams, 2006; Schelegle et al., 2009).[10]

3.   Prolonged exposure to an average O_3 concentration of 70 ppb results in a statistically
4   significant group mean decrement in FEV_1 of about 6% (Schelegle et al., 2009).

4.   Prolonged square-wave exposure to average O_3 concentrations of 80 ppb, 100 ppb, or 120
6   ppb O_3 results in statistically significant group mean decrements in FEV_1 ranging from 6
7   to 8%, 8 to 14%, and 13 to 16%, respectively (Folinsbee et al., 1988; Horstman et al.,
8   1990; McDonnell et al., 1991; Adams, 2002; Adams, 2003; Adams, 2006).

9   As illustrated in Figure 6-1 of the ISA, there is a smooth dose-response curve without
10   evidence of a threshold for exposures between 40 and 120 ppb O_3 (U.S. EPA, 2012a, Figure 6-
11   1). When these data are taken together, the ISA concludes that "mean FEV_1 is clearly decreased
12   by 6.6-h exposures to 60 ppb O_3 and higher concentrations in [healthy, young adult] subjects
13   performing moderate exercise" (U.S. EPA, 2012a, p. 6-9).

14   With respect to interindividual variability in lung function, in an individual with
15   relatively "normal" lung function, with recognition of the technical and biological variability in
16   measurements, within-day changes in FEV_1 of $\geq 5\%$ are clinically meaningful (Pellegrino et al.,
17   2005; ATS, 1991). The ISA (U.S. EPA, 2013, section 6.1.) focuses on individuals with >10%
18   decrements in FEV_1 for two reasons. A 10% FEV_1 decrement is accepted by the American
19   Thoracic Society (ATS) as an abnormal response and a reasonable criterion for assessing
20   exercise-induced bronchoconstriction (Dryden et al., 2010; ATS, 2000a). (U.S. EPA, 2013,
21   section 6.2.1.1). Also, some individuals in the Schelegle et al. (2009) study experienced 5-10%
22   FEV_1 decrements following exposure to filtered air.

23   In previous NAAQS reviews, the EPA has made judgments regarding the potential
24   implications for individuals experiencing FEV_1 decrements of varying degrees of severity.[11] For
25   people with lung disease, the EPA judged that moderate functional decrements (e.g., FEV_1
26   decrements ≥ 10 percent but < 20 percent, lasting up to 24 hours) would likely interfere with

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[10]Adams (2006) did not find effects on FEV_1 at 60 ppb to be statistically significant. 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 hour exposure period and found the average effect on FEV_1 at 60 ppb to be small, but highly statistically significant using several common statistical tests, even after removal of potential outliers.

[11]Such judgments have been made for decrements in FEV_1 as well as for increased airway responsiveness and symptomatic responses (e.g., cough, chest pain, wheeze). Ranges of pulmonary responses and their associated potential impacts are presented in Tables 3-2 and 3-3 of the 2007 Staff Paper (U.S. EPA, 2007).

1   normal activity for many individuals, and would likely result in more frequent use of medication

2   (75 FR 2973, January 19, 2010). In previous reviews CASAC has endorsed these conclusions. In

3   the context of standard setting, in the last review O_3 review CASAC indicated that it is

4   appropriate to focus on the lower end of the range of moderate functional responses (e.g., FEV_1

5   decrements ≥ 10 percent) when estimating potentially adverse lung function decrements in

6   people with lung disease, especially children with asthma (Henderson, 2006c; transcript of

7   CASAC meeting, day 8/24/06, page 149). More specifically, CASAC stated that "[a] 10%

8   decrement in FEV_1 can lead to respiratory symptoms, especially in individuals with pre-existing

9   pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease

10   have decreased ventilatory reserve (i.e., decreased baseline FEV_1) such that a ≥ 10% decrement

11   could lead to moderate to severe respiratory symptoms" (Samet, 2011). Therefore, in considering

12   interindividual variability in O_3-induced lung function decrements in the current review, we also

13   focus on the extent to which individuals were reported to experience FEV_1 decrements of 10% or

14   greater.

15   New studies (Schelegle et al., 2009; Kim et al., 2011) add to the previously available

16   evidence for interindividual variability in the responses of healthy adults following exposures to

17   O_3. Following prolonged exposures to 80 ppb O_3 while at moderate exertion, the proportion of

18   healthy adults experiencing FEV_1 decrements greater than 10% was 17% by Adams (2006a),

19   26% by McDonnell (1996), and 29% by Schelegle et al. (2009). Following exposures to 60 ppb

20   O_3, that proportion was 20% by Adams (2002), 3% by Adams (2006a), 16% by Schelegle et al.

21   (2009), and 5% by Kim et al. (2011). Based on these studies, the weighted average proportion of

22   young, healthy adults with >10% FEV_1 decrements is 25% following exposure to 80 ppb O_3 and

23   10% following exposure to 60 ppb O_3 (U.S. EPA, 2013, page 6-19).[12] The ISA notes that

24   responses within an individual tend to be reproducible over a period of several months,

25   indicating that interindividual differences reflect differences in intrinsic responsiveness. Given

26   this, the ISA concludes that "a considerable fraction" of healthy individuals experience clinically

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[12]The ISA also notes that by considering responses uncorrected for filtered air exposures, during which lung function typically improves (which would increase the size of the change, pre-and post-exposure), 10% is an underestimate of the proportion of healthy individuals that are likely to experience clinically meaningful changes in lung function following exposure for 6.6 hours to 60 ppb O_3 during intermittent moderate exertion (U.S. EPA, 2012, section 6.2.1.1).

1   meaningful decrements in lung function when exposed for 6.6 hours to 60 ppb O_3 during quasi

2   continuous, moderate exertion (U.S. EPA, 2013, section 6.2.1.1, p. 6-20).

3   As discussed above (Figure 3-1) and in the ISA (U.S EPA, 2013, Section 5.3.2),

4   secondary oxidation products formed following O_3 exposures can activate neural reflexes leading

5   to decreased lung function. Two new quantitative models, discussed in section 6.2.1.1 of the ISA

6   (U.S. EPA, 2013, p. 6-15), make use of the concept of oxidant stress to estimate the occurrence

7   of lung function decrements following exposures to relatively low O_3 concentrations (McDonnell

8   et al., 2012; Schelegle et al., 2012). These models reflect the protective effect of antioxidants in

9   the ELF at lower ambient O_3 concentrations, and include a threshold related to an integrated dose

10   rate.

11   McDonnell et al. (2012) and Schelegle et al. (2012) developed models using data on O_3

12   exposure concentrations, ventilation rates, duration of exposures, and lung function responses

13   from a number of controlled human exposure studies. The McDonnell et al. (2012) and Schelegle

14   et al. (2012) studies analyzed large datasets to fit compartmental models that included the

15   concept of a dose of onset in lung function response or a response threshold based upon the

16   inhaled O_3 dose. The first compartment in the McDonnell et al. (2012) model considers the level

17   of oxidant stress in response to O_3 exposure to increase over time as a function of dose rate

18   (C×) and decrease by clearance or metabolism over time. In the second compartment of the

19   McDonnell model, once oxidant stress reaches a threshold level the decrement in FEV_1 increases

20   as a sigmoid-shaped function. In the Schelegle et al. (2012) model, a first compartment acts as a

21   reservoir in which oxidant stress builds up until the dose of onset, at which time it spills over into

22   a second compartment. The second compartment is identical to the first compartment in

23   McDonnell et al. (2012) model. The oxidant levels in the second compartment were multiplied

24   by a responsiveness coefficient to predict FEV_1 responses for the Schelegle et al. (2012) model.

25   The McDonnell et al. (2012) model was fit to a large dataset consisting of the FEV_1

26   responses of 741 young, healthy adults (18-35 years of age) from 23 individual controlled

27   exposure studies. Concentrations across individual studies ranged from 40 ppb to 400 ppb,

28   activity level ranged from rest to heavy exercise, duration of exposure was from 2 to 7.6 hours.

29   The extension of the McDonnell et al. (2012) model to children and older adults is discussed in

30   section 6.2.4 of U.S. EPA (2014). Schelegle et al. (2012) also analyzed a large dataset with

31   substantial overlap to that used by McDonnell et al. (2012). The Schelegle et al. (2012) model

32   was fit to the FEV_1 responses of 220 young healthy adults (taken from a dataset of 704

33   individuals) from 21 individual controlled exposure studies. The resulting empirical models can

34   estimate the frequency distribution of individual responses for any exposure scenario as well as

1   summary measures of the distribution such as the mean or median response and the proportions
2   of individuals with FEV_1 decrements > 10%, 15%, and 20%.

3   The predictions of the McDonnell and Schelegle models are consistent with the observed
4   results from the individual studies of O_3-induced FEV_1 decrements. Specifically, the model
5   developed by McDonnell et al. (2012) predicts that 9% of healthy exercising adults would
6   experience FEV_1 decrements greater than 10% following 6.6 hour exposure to 60 ppb O_3, and
7   that 22% would experience such decrements following exposure to 80 ppb O_3 (U.S. EPA, 2013,
8   p. 6-18 and Figure 6-3). The model developed by Schelegle et al. (2012) predicts that, for a
9   prolonged (6.6 hours) O_3 exposure with moderate, quasi continuous exercise, the average dose of
10 onset for FEV_1 decrement would be reached following 4 to 5 hours of exposure to 60 ppb, and
11 following 3 to 4 hours of exposure to 80 ppb. However, 14% of the individuals had a dose of
12 onset that was less than 40% of the average. Those individuals would reach their dose of onset
13 following 1 to 2 hours of exposure to 50 to 80 ppb O_3 (U.S. EPA, 2013, p. 6-16), which is
14 consistent with the threshold FEV_1 responses reported by McDonnell et al. (2012).

15   Epidemiologic studies[13] have consistently linked short-term increases in ambient O_3
16 concentrations with lung function decrements in diverse populations and lifestages, including
17 children attending summer camps, adults exercising or working outdoors, and groups with pre-
18 existing respiratory diseases such as asthmatic children (U.S. EPA, 2013, section 6.2.1.2). Some
19 of these studies reported ozone-associated lung function decrements accompanied by respiratory
20 symptoms[14] in asthmatic children (Just et al., 2002; Mortimer et al., 2002; Ross et al., 2002;
21 Gielen et al., 1997; Romieu et al., 1997; Thurston et al., 1997; Romieu et al., 1996). In contrast,
22 studies of children in the general population have reported similar O_3-associated lung function
23 decrements but without accompanying respiratory symptoms (Ward et al., 2002; Gold et al.,
24 1999; Linn et al., 1996) (U.S. EPA, 2013, section 6.2.1.2).

25   Several panel studies reported that associations with lung function decrements persisted
26 at relatively low ambient O_3 concentrations. For outdoor recreation or exercise, associations were
27 reported in analyses restricted to 1-hour average O_3 concentrations less than 80 ppb (Spektor et
28 al., 1988a; Spektor et al., 1988b), 60 ppb (Brunekreef et al., 1994; Spektor et al., 1988a), and
29 50 ppb (Brunekreef et al., 1994). Among outdoor workers, Brauer et al. (1996) found a robust
30 association using daily 1-hour max O_3 concentrations less than 40 ppb. Ulmer et al. (1997) found
31 a robust association in schoolchildren using 30-minute maximum O_3 concentrations less than
32 60 ppb. For 8-hour average O_3 concentrations, associations with lung function decrements in

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[13] Unless otherwise specified, the epidemiologic studies discussed in this PA evaluate only adults.

[14] Reversible loss of lung function in combination with the presence of symptoms meets the ATS definition of adversity (ATS, 2000).

1   children with asthma were found to persist at concentrations less than 80 ppb in a U.S. multicity

2   study (Mortimer et al., 2002) and less than 51 ppb in a study conducted in the Netherlands

3   (Gielen et al., 1997).

4   Studies investigating the effects of short-term exposure to O_3 provided information on

5   potential confounding by copollutants such as $PM_{2.5}$, PM_{10}, NO_2, or SO_2. These studies varied in

6   how they evaluated confounding. Some studies of subjects exercising outdoors indicated that

7   ambient concentrations of copollutants such as NO_2, SO_2, or acid aerosol were low, and thus not

8   likely to confound associations observed for O_3 (Hoppe et al., 2003; Brunekreef et al., 1994;

9   Hoek et al., 1993). In other studies of children with increased outdoor exposures, O_3 was

10 consistently associated with decreases in lung function, whereas other pollutants such as $PM_{2.5}$,

11 sulfate, and acid aerosol individually showed variable associations across studies (Thurston et

12 al., 1997; Castillejos et al., 1995; Berry et al., 1991; Avol et al., 1990; Spektor et al., 1988a).

13 Studies that conducted copollutant modeling generally found O_3-associated lung function

14 decrements to be robust (i.e., most copollutant-adjusted effect estimates fell within the 95% CI of

15 the single-pollutant effect estimates) (U.S. EPA, 2013, Figure 6-10 and Table 6-14). Most O_3

16 effect estimates for lung function were robust to adjustment for temperature, humidity, and

17 copollutants such as $PM_{2.5}$, PM_{10}, NO_2, or SO_2. Although examined in only a few epidemiologic

18 studies, O_3 also remained associated with decreases in lung function with adjustment for pollen

19 or acid aerosols (U.S. EPA, 2013, section 6.2.1.2).

20   Several epidemiologic studies demonstrated the protective effects of vitamin E and

21 vitamin C supplementation, and increased dietary antioxidant intake, on O_3-induced lung

22 function decrements (Romieu et al., 2002) (U.S. EPA, 2013, Figure 6-7 and Table 6-8). These

23 results provide support for the new, quantitative models (McDonnell et al., 2012; Schelegle et

24 al., 2012), discussed above, which make use of the concept of oxidant stress to estimate the

25 occurrence of lung function decrements following exposures to relatively low O_3 concentrations.

26   In conclusion, new information from controlled human exposure studies considerably

27 strengthens the evidence and reduces the uncertainties, relative to the evidence that was available

28 at the time of the 2008 review, regarding the presence and magnitude of lung function

29 decrements in healthy adults following prolonged exposures to O_3 concentrations below 80 ppb.

30 As discussed in Section 6.2.1.1 in the ISA (EPA, 2013, p. 6-12), there is information available

31 from four separate studies that evaluated exposures to 60 ppb O_3 (Kim et al., 2011; Schelegle et

32 al., 2009; Adams 2002; 2006). Although not consistently statistically significant, group mean

33 FEV_1 decrements following exposures to 60 ppb O_3 are consistent among studies. Moreover, as

34 is illustrated in Figure 6-1 of the ISA (U.S. EPA, 2013), the group mean FEV_1 responses at

35 60 ppb fall on a smooth intake dose-response curve for exposures between 40 and 120 ppb O_3.

36 These studies also indicate that, on average, 10% of young, healthy adults experience clinically

1   meaningful decrements in lung function when exposed for 6.6 hours to 60 ppb O_3 during

2   intermittent, moderate exertion. One recent study has also reported statistically significant

3   decrements following exposures to 70 ppb O_3 (Schelegle et al., 2009). Predictions from newly

4   developed quantitative models, based on the concept that O_3-induced oxidation results in lung

5   function decrements, are consistent with these experimental results. Additionally, as discussed in

6   more detail in section 3.1.4 below, epidemiologic studies continue to provide evidence of lung

7   function decrements in people who are active outdoors, including people engaged in outdoor

8   recreation or exercise, children, and outdoor workers, at low ambient O_3 concentrations. While

9   few new epidemiologic studies of O_3-associated lung function decrements are available in this

10   review, previously available studies have reported associations with decrements, including at

11   relatively low ambient O_3 concentrations.

12   **Pulmonary Inflammation, Injury, and Oxidative Stress**

13   Ozone exposures result in increased respiratory tract inflammation and epithelial

14   permeability. Inflammation is a host response to injury, and the induction of inflammation is

15   evidence that injury has occurred. Oxidative stress has been shown to play a key role in initiating

16   and sustaining O_3-induced inflammation. Secondary oxidation products formed as a result of

17   reactions between O_3 and components of the ELF can increase the expression of molecules (i.e.,

18   cytokines, chemokines, and adhesion molecules) that can enhance airway epithelium

19   permeability (U.S. EPA, 2013, Sections 5.3.3 and 5.3.4). As discussed in detail in the ISA (U.S.

20   EPA, 2013, section 6.2.3), O_3 exposures can initiate an acute inflammatory response throughout

21   the respiratory tract that has been reported to persist for at least 18-24 hours after exposure.

22   Inflammation induced by exposure of humans to O_3 can have several potential outcomes:

23   (1) inflammation induced by a single exposure (or several exposures over the course of a

24   summer) can resolve entirely; (2) continued acute inflammation can evolve into a chronic

25   inflammatory state; (3) continued inflammation can alter the structure and function of other

26   pulmonary tissue, leading to diseases such as asthma; (4) inflammation can alter the body's host

27   defense response to inhaled microorganisms, particularly in potentially at-risk populations or

28   lifestages such as the very young and old; and (5) inflammation can alter the lung's response to

29   other agents such as allergens or toxins (U.S. EPA, 2013, Section 6.2.3). Thus, lung injury and

30   the resulting inflammation provide a mechanism by which O_3 may cause other more serious

31   morbidity effects (e.g., asthma exacerbations).

1   In the last review, controlled human exposure studies reported O_3-induced airway

2   inflammation following exposures at or above 80 ppb. In the current review, the link between O_3

3   exposures and airway inflammation and injury has been evaluated in additional controlled human

4   exposure studies, as well as in recent epidemiologic studies. Controlled human exposure studies

5   have generally been conducted in young, healthy adults or in adults with asthma using lavage

6   (proximal airway and bronchoalveolar), bronchial biopsy, and more recently, induced sputum.

7   These studies have evaluated one or more indicators of inflammation, including neutrophil[15]

8   (PMN) influx, markers of eosinophilic inflammation, increased permeability of the respiratory

9   epithelium, and/or prevalence of proinflammatory molecules (U.S. EPA, 2013, section 6.2.3.1).

10   Epidemiologic studies have generally evaluated associations between ambient O_3 and markers of

11   inflammation and/or oxidative stress, which plays a key role in initiating and sustaining

12   inflammation (U.S. EPA, 2013, section 6.2.3.2).

13   There is an extensive body of evidence from controlled human exposure studies

14   indicating that short-term exposures to O_3 can cause pulmonary inflammation. Previously

15   available evidence indicated that O_3 causes an inflammatory response in the lungs (U.S. EPA,

16   1996a). A single acute exposure (1-4 hours) of humans to moderate concentrations of O_3 (200-

17   600 ppb) while exercising at moderate to heavy intensities resulted in a number of cellular and

18   biochemical changes in the lung, including inflammation characterized by increased numbers of

19   PMNs, increased permeability of the epithelial lining of the respiratory tract, cell damage, and

20   production of proinflammatory molecules (i.e., cytokines and prostaglandins, U.S. EPA, 2006b).

21   A meta-analysis of 21 controlled human exposure studies (Mudway and Kelly, 2004) using

22   varied experimental protocols (80-600 ppb O_3 exposures; 1-6.6 hours exposure duration; light to

23   heavy exercise; bronchoscopy at 0-24 hours post-O_3 exposure) reported that PMN influx in

24   healthy subjects is linearly associated with total O_3 dose. Animal toxicological studies also

25   provided evidence for increases in inflammation and permeability in rabbits at levels as low as

26   100 ppb O_3 (Section 2.5.3.1, ISA, U.S. EPA, 2013).

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[15] Referred to as either neutrophils or polymorphonuclear neutrophils (or PMNs), these are the most abundant type of white blood cells in mammals. PMNs are recruited to the site of injury following trauma and are the hallmark of acute inflammation. The presence of PMNs in the lung has long been accepted as a hallmark of inflammation and is an important indicator that O_3 causes inflammation in the lungs. Neutrophilic inflammation of tissues indicates activation of the innate immune system and requires a complex series of events, that then are normally followed by processes that clear the evidence of acute inflammation.

1   Several studies, including one published since the last review (Alexis et al., 2010), have

2 reported O_3-induced increases in PMN influx and permeability following exposures at or above

3 80 ppb (Alexis et al., 2010; Peden et al., 1997; Devlin et al., 1991), and eosinophilic

4 inflammation following exposures at or above 160 ppb (Scannell et al., 1996; Peden et al., 1997;

5 Hiltermann et al., 1999; Vagaggini et al., 2002). In addition, one recent controlled human

6 exposure study has reported O_3-induced PMN influx following exposures of healthy adults to O_3

7 concentrations of 60 ppb (Kim et al., 2011), the lowest concentration at which inflammatory

8 responses have been evaluated in human studies.

9   As with FEV_1 responses to O_3, inflammatory responses to O_3 are generally reproducible

10 within individuals, with some individuals experiencing more severe O_3-induced airway

11 inflammation than indicated by group averages (Holz et al., 2005; Holz et al., 1999). Unlike O_3-

12 induced decrements in lung function, which are attenuated following repeated exposures over

13 several days (U.S. EPA, 2013, section 6.2.1.1), some markers of O_3-induced inflammation and

14 tissue damage remain elevated during repeated exposures, indicating ongoing damage to the

15 respiratory system (U.S. EPA, 2013, section 6.2.3.1).

16   Most controlled human exposure studies have reported that asthmatics experience larger

17 O_3-induced inflammatory responses than non-asthmatics. Specifically, asthmatics exposed to

18 200 ppb O_3 for 4-6 hours with exercise show significantly more neutrophils in bronchoalveolar

19 lavage fluid (BALF) than similarly exposed healthy individuals (Scannell et al., 1996; Basha et

20 al., 1994). Bosson et al. (2003) reported significantly greater expression of a variety of pro-

21 inflammatory cytokines in asthmatics, compared to healthy subjects, following exposure to

22 200 ppb O_3 for 2 hours. In addition, research available in the last review, combined with a recent

23 study newly available in this review, indicates that pretreatment of asthmatics with

24 corticosteroids can prevent the O_3-induced inflammatory response in induced sputum, though

25 pretreatment did not prevent FEV_1 decrements (Vagaggini et al., 2001; 2007). In contrast,

26 Stenfors et al. (2002) did not detect a difference in the O_3-induced increases in neutrophil

27 numbers between 15 subjects with mild asthma and 15 healthy subjects by bronchial wash at the

28 6 hours postexposure time point, although the neutrophil increase in the asthmatic group was on

29 top of an elevated baseline.

30   In people with allergic airway disease, including people with rhinitis and asthma,

31 evidence available in the last review indicated that proinflammatory mediators also cause

1 accumulation of eosinophils in the airways (Bascom et al., 1990; Jorres et al., 1996; Peden et
2 al.,1995 and 1997; Frampton et al., 1997; Michelson et al., 1999; Hiltermann et al., 1999; Holz et
3 al., 2002; Vagaggini et al., 2002). The eosinophil, which increases inflammation and allergic
4 responses, is the cell most frequently associated with exacerbations of asthma (75 CFR 2969,
5 January 19, 2010).

6 Studies reporting inflammatory responses and markers of lung injury have clearly
7 demonstrated that there is important variation in the responses of exposed subjects (75 FR 2953,
8 January 19, 2010). Some individuals also appear to be intrinsically more susceptible to increased
9 inflammatory responses (Holz et al., 2005). In healthy adults exposed to each 80 and 100 ppb O_3,
10 Devlin et al. (1991) observed group average increases in neutrophilic inflammation of 2.1- and
11 3.8-fold, respectively. However, there was a 20-fold range in inflammatory responses between
12 individuals at both concentrations. Relative to an earlier, similar study conducted at 400 ppb
13 (Koren et al., 1989), Devlin et al. (1991) noted that although some of the study population
14 showed little or no increase in inflammatory and cellular injury indicators analyzed after
15 exposures to lower levels of O_3 (i.e., 80 and 100 ppb), others had changes that were as large as
16 those seen when subjects were exposed to 400 ppb O_3. The data suggest that as a whole the
17 healthy population, on average, may have small inflammatory responses to near-ambient levels
18 of O_3, though there may be a significant subpopulation that is very sensitive to low levels of O_3.
19 Devlin et al. (1991) expressed the view that ''susceptible subpopulations such as the very young,
20 elderly, and people with pulmonary impairment or disease may be even more affected.''

21 A number of studies report that O_3 exposures increase epithelial cell permeability.
22 Increased BALF protein, suggesting O_3-induced changes in epithelial permeability, has been
23 reported at 1 hour and 18 hours postexposure (Devlin et al., 1997; Balmes et al., 1996). A meta-
24 analysis of results from 21 publications (Mudway and Kelly, 2004a) for varied experimental
25 protocols (80-600 ppb O_3; 1-6.6 hours duration; light to heavy exercise; bronchoscopy at 0-24
26 hours post-O_3 exposure; healthy subjects), showed that increased BALF protein is associated
27 with total inhaled O_3 dose (i.e., the product of O_3 concentration, exposure duration, and   ). As
28 noted in the 2009 PM ISA (U.S. EPA, 2009), it has been postulated that changes in permeability
29 associated with acute inflammation may provide increased access of inhaled antigens, particles,
30 and other inhaled substances deposited on lung surfaces to the smooth muscle, interstitial cells,
31 immune cells underlying the epithelium, and the blood (U.S. EPA, 2013, sections 5.3.4, 5.3.5).

1   Animal toxicology studies have provided some support for this hypothesis (Adamson and

2   Prieditis, 1995; Chen et al 2006), though these studies did not specifically evaluate O_3 exposures

3   (U.S. EPA, 2009). Because of this potentially increased access, it has been postulated that

4   increases in epithelial permeability following O_3 exposure might lead to increases in airway

5   responsiveness to specific and nonspecific agents. In a recent study, Que et al. (2011)

6   investigated this hypothesis in healthy young adults (83M, 55 F) exposed to 220 ppb O_3 for 2.25

7   hours (alternating 15 min periods of rest and brisk treadmill walking). As has been observed for

8   FEV_1 responses, within-individual changes in permeability were correlated between sequential

9   O_3 exposures, indicating intrinsic differences among individuals in susceptibility to epithelial

10   damage following O_3 exposures. However, increases in epithelial permeability at 1 day post-O_3

11   exposure were not correlated with with changes in airway responsiveness assessed 1 day post-O_3

12   exposure. The authors concluded that changes in epithelial permeability is relatively constant

13   over time in young healthy adults, although changes in permeability and AHR appear to be

14   mediated by different physiologic pathways.

15   The limited epidemiologic evidence reviewed in the 2006 O_3 AQCD (U.S. EPA, 2006)

16   demonstrated an association between short-term increases in ambient O_3 concentrations and

17   airways inflammation in children (1-hour max O_3 of approximately 100 ppb). In the 2006 O_3

18   AQCD (U.S. EPA, 2006), there was limited evidence for increases in nasal lavage levels of

19   inflammatory cell counts and molecules released by inflammatory cells (i.e., eosinophilic

20   cationic protein, and myeloperoxidases). Since 2006, as a result of the development of less

21   invasive methods, there has been a large increase in the number of studies assessing ambient O_3-

22   associated changes in airway inflammation and oxidative stress, the types of biological samples

23   collected (e.g., lower airway), and the types of indicators. Most of these recent studies have

24   evaluated biomarkers of inflammation or oxidative stress in exhaled breath, nasal lavage fluid, or

25   induced sputum (U.S. EPA, 2013, section 6.2.3.2). These recent studies form a larger database to

26   establish coherence with findings from controlled human exposure and animal studies that have

27   measured the same or related biological markers. Additionally, results from these studies provide

28   further biological plausibility for the associations observed between ambient O_3 concentrations

29   and respiratory symptoms and asthma exacerbations.

30   A number of epidemiologic studies provide evidence that short-term increases in ambient

31   O_3 exposure increase pulmonary inflammation and oxidative stress in children, including those

1   with asthma (Sienra-Monge et al., 2004; Barraza-Villarreal et al., 2008; Romieu et al., 2008;

2   Berhane et al., 2011). Multiple studies examined and found increases in exhaled NO (eNO)[16]

3   (Berhane et al., 2011; Khatri et al., 2009; Barraza-Villarreal et al., 2008). In some studies of

4   subjects with asthma, increases in ambient O_3 concentration at the same lag were associated with

5   both increases in pulmonary inflammation and respiratory symptoms (Khatri et al., 2009;

6   Barraza-Villarreal et al., 2008). Although more limited in number, epidemiologic studies also

7   found associations with cytokines such as IL-6 or IL-8 (Barraza-Villarreal et al., 2008; Sienra-

8   Monge et al., 2004), eosinophils (Khatri et al., 2009), antioxidants (Sienra-Monge et al., 2004),

9   and indicators of oxidative stress (Romieu et al., 2008) (ISA, Section 6.2.3.2, U.S. EPA, 2013).

10   Because associations with inflammation were attenuated with higher antioxidant intake the study

11   by Sienra-Monge et al. (2004) provides additional evidence that inhaled O_3 is likely to be an

12   important source of reactive oxygen species in airways and/or may increase pulmonary

13   inflammation via oxidative stress-mediated mechanisms among all age groups. Limitations in

14   some recent studies have contributed to inconsistent results in adults (U.S. EPA, 2013, section

15   6.2.3.2).

16   Exposure to ambient O_3 on multiple days can result in larger increases in pulmonary

17   inflammation and oxidative stress, as discussed in section 6.2.3.2 of the ISA (U.S. EPA, 2013).

18   In studies that examined multiple O_3 lags, multiday averages of 8-hour maximum or

19   8-hour average concentrations were associated with larger increases in pulmonary inflammation

20   and oxidative stress (Berhane et al., 2011; Delfino et al., 2010a; Sienra-Monge et al., 2004),

21   consistent with controlled human exposure (U.S. EPA, 2013, section 6.2.3.1) and animal studies

22   (U.S. EPA, 2013, section 6.2.3.3) reporting that some markers of pulmonary inflammation

23   remain elevated with O_3 exposures repeated over multiple days. Evidence from animal

24   toxicological studies also clearly indicates that O_3 exposures result in damage and inflammation

25   in the lung (ISA, Section 5.3, U.S. EPA, 2013). In the few studies that evaluated the potential for

26   confounding, O_3 effect estimates were not confounded by temperature or humidity, and were

27   robust to adjustment for $PM_{2.5}$ or PM_{10} (Barraza-Villarreal et al., 2008; Romieu et al., 2008;

28   Sienra-Monge et al., 2004).

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[16] Exhaled NO has been shown to be a useful biomarker for airway inflammation in large population-based studies
(Linn et al., 2009) (ISA, U.S. EPA, 2013, Section 7.2.4).

1   In conclusion, a relatively small number of controlled human exposure studies evaluating

2   O_3-induced airway inflammation have become available since the last review. For purposes of

3   reviewing the current O_3 NAAQS, the most important of these recent studies reported a

4   statistically significant increase in airway inflammation in healthy adults at moderate exertion

5   following exposures to 60 ppb O_3, the lowest concentration that has been evaluated for

6   inflammation. In addition, a number of recent epidemiologic studies report O_3-associated

7   increases in markers of pulmonary inflammation, particularly in children. Thus, recent studies

8   continue to support the evidence for airway inflammation and injury that was available in

9   previous reviews, with new evidence for such effects following exposures to lower

10   concentrations than had been evaluated previously.

11   **Airway Hyperresponsiveness**

12   Airway hyperresponsiveness (AHR) refers to a condition in which the conducting

13   airways undergo enhanced bronchoconstriction in response to a variety of stimuli. Airway

14   hyperresponsiveness is an important consequence of exposure to ambient O_3 because its presence

15   reflects a change in airway smooth muscle reactivity, and indicates that the airways are

16   predisposed to narrowing upon inhalation of a variety of ambient stimuli including specific

17   triggers (i.e., allergens) and nonspecific triggers (e.g., SO_2, and cold air). People with asthma are

18   generally more sensitive to bronchoconstricting agents than those without asthma, and the use of

19   an airway challenge to inhaled bronchoconstricting agents is a diagnostic test in asthma.

20   Standards for airway responsiveness testing have been developed for the clinical laboratory

21   (ATS, 2000a), although variation in the methodology for administering the bronchoconstricting

22   agent may affect the results (Cockcroft et al., 2005). There is a wide range of airway

23   responsiveness in people without asthma, and responsiveness is influenced by a number of

24   factors, including cigarette smoke, pollutant exposures, respiratory infections, occupational

25   exposures, and respiratory irritants. Dietary antioxidants have been reported to attenuate O_3-

26   induced bronchial hyperresponsiveness in people with asthma (Trenga et al., 2001).

27   Evidence for airway hyperresponsiveness following O_3 exposures is derived primarily

28   from controlled human exposure and toxicological studies (U.S. EPA, 2013, section 6.2.2).

29   Airway responsiveness is often quantified by measuring changes in pulmonary function

30   following the inhalation of an aerosolized allergen or a nonspecific bronchoconstricting agent

1   (e.g., methacholine), or following exposure to a bronchoconstricting stimulus such as cold air. In

2   the last review, controlled human exposure studies of mostly adults ($\geq$ 18 years of age) had

3   shown that exposures to O_3 concentrations at or above 80 ppb increase airway responsiveness, as

4   indicated by a reduction in the concentration of specific (e.g., ragweed) and non-specific (e.g.,

5   methacholine) agents required to produce a given reduction in lung function (e.g., as measured

6   by FEV_1 or specific airway resistance) (U.S. EPA, 2013, section 6.2.2.1). This O_3-induced AHR

7   has been reported to be dose-dependent (Horstman et al., 1990). Animal toxicology studies have

8   reported O_3-induced airway hyperresponsiveness in a number of species, with some rat strains

9   exhibiting hyperresponsiveness following 4-hour exposures to O_3 concentrations as low as 50

10   ppb (Depuydt et al., 1999). Since the last review, there have been relatively few new controlled

11   human exposure and animal toxicology studies of O_3 and airway hyperresponsiveness, and no

12   new studies have evaluated exposures to O_3 concentrations at or below 80 ppb (U.S. EPA, 2013,

13   section 6.2.2.1)

14   Airway hyperresponsiveness is linked with the accumulation and/or activation of

15   eosinophils in the airways of asthmatics, which is followed by production of mucus and a late-

16   phase asthmatic response (75 FR 2970, January 19, 2010). In a study of 16 intermittent

17   asthmatics, Hiltermann et al. (1999) found that there was a significant inverse correlation

18   between the O_3-induced change in the percentage of eosinophils in induced sputum and the

19   concentration of methacholine causing a 20% decrease in FEV_1. Hiltermann et al. (1999)

20   concluded that the results point to the role of eosinophils in O_3-induced airway

21   hyperresponsiveness. Increases in O_3-induced nonspecific airway responsiveness incidence and

22   duration could have important clinical implications for children and adults with asthma, such as

23   exacerbations of their disease.

24   Airway hyperresponsiveness after O_3 exposure appears to resolve more slowly than

25   changes in FEV_1 or respiratory symptoms (Folinsbee and Hazucha, 2000). Studies suggest that

26   O_3-induced AHR usually resolves 18 to 24 hours after exposure, but may persist in some

27   individuals for longer periods (Folinsbee and Hazucha, 1989). Furthermore, in studies of

28   repeated exposure to O_3, changes in AHR tend to be somewhat less susceptible to attenuation

29   with consecutive exposures than changes in FEV_1 (Gong et al., 1997a; Folinsbee et al., 1994;

30   Kulle et al., 1982; Dimeo et al., 1981) (U.S. EPA, 2013, section 6.2.2). In animal studies a 3-day

31   continuous exposure resulted in attenuation of O_3-induced airway hyperresponsiveness (Johnston

1   et al., 2005) while repeated exposures for 2 hours per day over 10 days did not (Chhabra et al.,

2   2010), suggesting that attenuation could be lost when repeated exposures are interspersed with

3   periods of rest (U.S. EPA, 2013, section 6.2.2.2).

4   Increases in airway responsiveness do not appear to be strongly associated with

5   decrements in lung function or increases in symptoms (Aris et al., 1995). Recently, Que et al.

6   (2011) assessed methacholine responsiveness in healthy young adults (83M, 55 F) one day after

7   exposure to 220 ppb O_3 and filtered air for 2.25 hours (alternating 15 minute periods of rest and

8   brisk treadmill walking). Increases in airways responsiveness at 1 day post-O_3 exposure were not

9   correlated with FEV_1 responses immediately following the O_3 exposure or with changes in

10 epithelial permeability assessed 1-day post-O_3 exposure. This indicates that airway hyper-

11 responsiveness also appears to be mediated by a differing physiologic pathway.

12   As mentioned above, in addition to human subjects a number of species, including

13 nonhuman primates, dogs, cats, rabbits, and rodents, have been used to examine the effect of O_3

14 exposure on airway hyperresponsiveness (see Table 6-14, (U.S. EPA, 1996n) of the 1996 O_3

15 AQCD and Annex Table AX5-12 on page AX5-36 (U.S. EPA, 2006h) of the 2006 O_3 AQCD). A

16 body of animal toxicology studies, including some recent studies conducted since the last review,

17 provides support for the O_3-induced AHR reported in humans (U.S. EPA, 2013, section 6.2.2.2).

18 Although most of these studies evaluated O_3 concentrations above those typically found in

19 ambient air in cities in the United States (i.e., most studies evaluated O_3 concentrations of 100

20 ppb or greater), one study reported that a very low exposure concentration (50 ppb for 4 hours)

21 induced AHR in some rat strains (Depuydt et al., 1999). Additional recent rodent studies

22 reported O_3-induced AHR following exposures to O_3 concentrations from 100 to 500 ppb

23 (Johnston et al., 2005; Chhabra et al., 2010; Larsen et al., 2010). In characterizing the relevance

24 of these exposure concentrations, the ISA noted that a study using radiolabeled O_3 suggests that

25 even very high O_3 exposure concentrations in rodents could be equivalent to much lower

26 exposure concentrations in humans. Specifically, a 2000 ppb (2 ppm) O_3 exposure concentration

27 in resting rats was reported to be roughly equivalent to a 400 ppb exposure concentration in

28 exercising humans (Hatch et al., 1994). Given this relationship, the ISA noted that animal data

29 obtained in resting conditions could underestimate the risk of effects for humans (U.S. EPA,

30 2013, section 2.4, p. 2-14).

1   The 2006 AQCD (U.S. EPA, 2006, p. 6-34) concluded that spirometric responses to O_3

2   are independent of inflammatory responses and markers of epithelial injury (Balmes et al., 1996;

3   Blomberg et al., 1999; Hazucha et al., 1996; Torres et al., 1997). Significant inflammatory

4   responses to O_3 exposures that did not elicit significant spirometric responses have been reported

5   (Holz et al., 2005; McBride et al., 1994). A recent study (Que et al., 2011) indicates that airway

6   hyper-responsiveness also appears to be mediated by a differing physiologic pathway. These

7   results from controlled human exposure studies indicate that sub-populations of healthy study

8   subjects consistently experience larger than average lung function decrements, greater than

9   average inflammatory responses and pulmonary injury as expressed by increased epithelial

10   permeability, and greater than average airway responsiveness, and that these effects are mediated

11   by apparently different physiologic pathways. Except for lung function decrements, we do not

12   have the concentration- or exposure-response function information about the other, potentially

13   more sensitive,[17] clinical endpoints (i.e., inflammation, increased epithelial permeability, airway

14   hyperresponsiveness) that would allow us to quantitatively estimate the size of the population

15   affected and the magnitude of their responses. Moreover, some uncertainties about the exact

16   physiological pathways underlying these endpoints prevents us from knowing whether the

17   exaggerated responses are distributed in sub-populations evenly across the population, or may be

18   clustered with more than one type of exaggerated response in particular sub-populations, or both.

19   In summary, a strong body of controlled human exposure and animal toxicological

20   studies, most of which were available in the last review of the O_3 NAAQS, report O_3-induced

21   airway hyperresponsiveness after either acute or repeated exposures (U.S. EPA, 2013, section

22   6.2.2.2). People with asthma often exhibit increased airway responsiveness at baseline relative to

23   healthy controls, and they can experience further increases in responsiveness following

24   exposures to O_3. Studies reporting increased airway responsiveness after O_3 exposure contribute

25   to a plausible link between ambient O_3 exposures and increased respiratory symptoms in

26   asthmatics, and increased hospital admissions and emergency department visits for asthma (U.S.

27   EPA, 2013, section 6.2.2.2).

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[17] CASAC noted that "…[W]hile measures of FEV1 are quantitative and readily obtainable in humans, they are not the only measures — and perhaps not the most sensitive measures — of the adverse health effects induced by ozone exposure." (Henderson, 2006).

Respiratory Symptoms and Medication Use

1 Because respiratory symptoms are associated with adverse outcomes such as limitations

2 in activity, and are the primary reason for people with asthma to use quick relief medication and

3 seek medical care, studies evaluating the link between O_3 exposures and such symptoms allow a

4 more direct characterization of the clinical and public health significance of ambient O_3 exposure

5 than measures of lung function decrements and pulmonary inflammation. Controlled human

6 exposure and toxicological studies have described modes of action through which short-term O_3

7 exposures may increase respiratory symptoms by demonstrating O_3-induced airway

8 hyperresponsiveness (U.S. EPA, 2013, section 6.2.2) and pulmonary inflammation (U.S. EPA,

9 2013, section 6.2.3).

10

11 The link between subjective respiratory symptoms and O_3 exposures has been evaluated

12 in both controlled human exposure and epidemiologic studies, and the link with medication use

13 has been evaluated in epidemiologic studies. In the last review, several controlled human

14 exposure studies reported respiratory symptoms following exposures to O_3 concentrations at or

15 above 80 ppb. In addition, one study reported such symptoms following exposures to 60 ppb O_3,

16 though the increase was not statistically different from filtered air controls. Epidemiologic

17 studies reported associations between ambient O_3 and respiratory symptoms and medication use

18 in a variety of locations and populations, including asthmatic children living in U.S. cities. In the

19 current review, additional controlled human exposure studies have evaluated respiratory

20 symptoms following exposures to O_3 concentrations below 80 ppb and recent epidemiologic

21 studies have evaluated associations with respiratory symptoms and medication use (U.S. EPA,

22 2013, sections 6.2.1, 6.2.4).

23 In controlled human exposure studies available in the last review as well as newly

24 available studies, statistically significant increases in respiratory symptoms have been

25 consistently reported in healthy volunteers engaged in intermittent, moderate exertion following

26 6.6 hour exposures to average O_3 concentrations at or above 80 ppb (Adams, 2003; Adams,

27 2006; Schelegle et al., 2009). Such symptoms have been reported to increase with increasing O_3

28 exposure concentrations, duration of exposure, and activity level (McDonnell et al., 1999b). For

29 example, in a study available during the last review, Adams (2006) reported an increase in

30 respiratory symptoms in healthy adults during a 6.6 hour exposure protocol with an average O_3

1 exposure concentration of 60 ppb. This increase was significantly different from initial

2 respiratory symptoms, but not from filtered air controls. Two recent controlled human exposure

3 studies that have become available since the last review did not report statistically significant

4 increases in respiratory symptoms following exposures of healthy adults to 60 ppb O_3 (Schelegle

5 et al., 2009; Kim et al., 2011). A recent study by Schelegle et al. (2009) did report a statistically

6 significant increase in respiratory symptoms in healthy adults following 6.6 hour exposures to an

7 average O_3 concentration of 70 ppb. The findings for O_3-induced respiratory symptoms in

8 controlled human exposure studies, and the evidence integrated across disciplines describing

9 underlying modes of action, provide biological plausibility for epidemiologic associations

10 observed between short-term increases in ambient O_3 concentration and increases in respiratory

11 symptoms (U.S. EPA, 2013, section 6.2.4).

12 In epidemiologic studies, respiratory symptom data typically are collected by having

13 subjects (or their parents) record symptoms and medication use in a diary without direct

14 supervision by study staff. Several limitations of symptom reports are well recognized, as

15 described in the ISA (U.S. EPA, 2013, section 6.2.4). Nonetheless, symptom diaries remain a

16 convenient tool to collect individual-level data from a large number of subjects and allow

17 modeling of associations between daily changes in O_3 concentration and daily changes in

18 respiratory morbidity over multiple weeks or months. Importantly, many of the limitations in

19 these studies are sources of random measurement error that can bias effect estimates to the null

20 or increase the uncertainty around effect estimates (U.S. EPA, 2013, Section 6.2.4). Because

21 respiratory symptoms are associated with limitations in activity and daily function and are the

22 primary reason for using medication and seeking medical care, the evidence is directly coherent

23 with the associations consistently observed between increases in ambient O_3 concentration and

24 increases in asthma emergency department visits, discussed below (U.S. EPA, 2013, Section

25 6.2.4).

26 Most epidemiologic studies of O_3 and respiratory symptoms and medication use have

27 been conducted in children and/or adults with asthma, with fewer studies, and less consistent

28 results, in non-asthmatic populations (U.S. EPA, 2013, section 6.2.4). The 2006 AQCD (U.S.

29 EPA, 2006b, U.S. EPA, 2013, section 6.2.4) concluded that the collective body of epidemiologic

30 evidence indicated that short-term increases in ambient O_3 concentrations are associated with

31 increases in respiratory symptoms in children with asthma. A large body of single-city and

1 single-region studies of asthmatic children provides consistent evidence for associations between

2 short-term increases in ambient O_3 concentrations and increased respiratory symptoms and

3 asthma medication use in children with asthma (U.S. EPA, 2013, Figure 6-12, Table 6-20).

4   Methodological differences among studies make comparisons across recent multicity

5 studies of respiratory symptoms difficult. Because of fewer person-days of data (Schildcrout et

6 al., 2006) or examination of 19-day averages of ambient O_3 concentrations (O'Connor et al.,

7 2008), the ISA did not give greater weight to results from recent multicity studies than results

8 from single-city studies (U.S. EPA, 2013, section 6.2.4.5). While evidence from the few

9 available U.S. multicity studies is less consistent (O'Connor et al., 2008; Schildcrout et al., 2006;

10 Mortimer et al., 2002), the overall body of epidemiologic evidence with respect to the

11 association betweeen exposure to O_3 and respiratory symptoms in asthmatic children remains

12 compelling (U.S. EPA, 2013, section 6.2.4.1). Findings from a small body of studies indicate that

13 O_3 is also associated with increased respiratory symptoms in adults with asthma (Khatri et al.,

14 2009; Feo Brito et al., 2007; Ross et al., 2002) (U.S. EPA, 2013, section 6.2.4.2).

15   Available evidence indicates that O_3-associated increases in respiratory symptoms are not

16 confounded by temperature, pollen, or copollutants (primarily PM) (U.S. EPA, 2013, section

17 6.2.4.5; Table 6-25; Romieu et al., 1996; Romieu et al., 1997; Thurston et al., 1997; Gent et al.,

18 2003). However, identifying the independent effects of O_3 in some studies was complicated due

19 to the high correlations observed between O_3 and PM or different lags and averaging times

20 examined for copollutants. Nonetheless, the ISA noted that the robustness of associations in

21 some studies of individuals with asthma, combined with findings from controlled human

22 exposure studies for the direct effects of O_3 exposure, provide substantial evidence supporting

23 the independent effects of short-term ambient O_3 exposure on respiratory symptoms (U.S. EPA,

24 2013, section 6.2.4.5).

25   Epidemiologic studies of medication use have reported associations with

26 1-hour maximum O_3 concentrations and with multiday average O_3 concentrations (Romieu et al.,

27 2006; Just et al., 2002). Some studies reported O_3 associations for both respiratory symptoms and

28 asthma medication use (Escamilla-Nuñez et al., 2008; Romieu et al., 2006; Schildcrout et al.,

29 2006; Jalaludin et al., 2004; Romieu et al., 1997; Thurston et al., 1997) while others reported

30 associations for either respiratory symptoms or medication use (Romieu et al., 1996; Rabinovitch

31 et al., 2004; Just et al., 2002; Ostro et al., 2001).

32   In summary, both controlled human exposure and epidemiologic studies have reported

33 respiratory symptoms attributable to short-term O_3 exposures. In the last review, the majority of

34 the evidence from controlled human exposure studies in young, healthy adults was for symptoms

1   following exposures to O_3 concentrations at or above 80 ppb. Although studies that have become
2   available since the last review have not reported respiratory symptoms in young, healthy adults
3   following exposures with moderate exertion to 60 ppb, one recent study has reported increased
4   symptoms in young, healthy adults while at moderate exertion following exposures to O_3
5   concentrations as low as 70 ppb. As was concluded in the 2006 O_3 AQCD (U.S. EPA, 2006b,
6   1996a), the collective body of epidemiologic evidence indicates that short-term increases in
7   ambient O_3 concentration are associated with increases in respiratory symptoms in children with
8   asthma (U.S. EPA, 2013, section 6.2.4). Recent studies of respiratory symptoms and medication
9   use, primarily in asthmatic children, add to this evidence. In a smaller body of studies, increases
10   in ambient O_3 concentration were associated with increases in respiratory symptoms in adults
11   with asthma.

12   **Lung Host Defense**

13   The mammalian respiratory tract has a number of closely integrated defense mechanisms
14   that, when functioning normally, provide protection from the potential health effects of
15   exposures to a wide variety of inhaled particles and microbes. These defense mechanisms
16   include mucociliary clearance, alveolobronchiolar transport mechanism, alveolar macrophages[18],
17   and adaptive immunity[19] (U.S. EPA, 2013, section 6.2.5). The previous O_3 AQCD (U.S. EPA,
18   2006) concluded that animal toxicological studies provided evidence that acute exposure to O_3
19   concentrations as low as 100 to 500 ppb can increase susceptibility to infectious diseases due to
20   modulation of these lung host defenses. This conclusion was based in large part on animal
21   studies of alveolar macrophage functioning and mucociliary clearance (U.S. EPA, 2013, section
22   6.2.5).

23   With regard to alveolar macrophage functioning, the previous O_3 AQCD (U.S. EPA,
24   2006) concluded that short periods of O_3 exposure can cause a reduction in the number of free
25   alveolar macrophages available for pulmonary defense, and that these alveolar macrophages are
26   more fragile, less able to engulf particles (i.e., phagocytic), and exhibit decreased lysosomal[20]
27   enzyme activities (U.S. EPA, 2013, section 6.2.5). These conclusions were based largely on

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[18] Phagocytic white blood cells within the alveoli of the lungs that ingest inhaled particles.

[19] The adaptive immune system, is also known as the acquired immune system. Acquired immunity creates immunological memory after an initial response to a specific pathogen, leading to an enhanced response to subsequent encounters with that same pathogen.

[20] Lysosomes are cellular organelles that contain acid hydrolase enzymes that break down waste materials and cellular debris.

1   studies conducted in animals exposed for several hours up to several weeks to O_3 concentrations

2   from 100 to 250 ppb (Hurst et al., 1970; Driscoll et al., 1987; Cohen et al., 2002). Consistent

3   with the animal evidence, a controlled human exposure study available in the last review had

4   reported decrements in the ability of alveolar macrophages to phagocytize yeast following

5   exposures of healthy volunteers to O_3 concentrations of 80 and 100 ppb for 6.6-hour during

6   moderate exercise (Devlin et al., 1991). Integrating the animal study results with human

7   exposure evidence available in the 1996 Criteria Document, the 2006 Criteria Document

8   concluded that available evidence indicates that short-term O_3 exposures have the potential to

9   impair host defenses in humans, primarily by interfering with alveolar macrophage function. Any

10   impairment in alveolar macrophage function may lead to decreased clearance of microorganisms

11   or nonviable particles. Compromised alveolar macrophage functions in asthmatics may increase

12   their susceptibility to other O_3 effects, the effects of particles, and respiratory infections (EPA,

13   2006a, p. 8–26).

14   With regard to mucociliary clearance, in the last review a number of studies conducted in

15   different animal species had reported morphological damage to the cells of the tracheobronchial

16   tree from acute and sub-chronic exposure to O_3 concentrations at or above 200 ppb. The cilia

17   were either completely absent or had become noticeably shorter or blunt. In general, functional

18   studies of mucociliary transport had observed a delay in particle clearance soon after acute

19   exposure, with decreased clearance more evident at higher doses (1 ppm) (U.S. EPA, 1986; U.S.

20   EPA, 2013, section 6.2.5.1).

21   Alveolobronchiolar transport mechanisms refers to the transport of particles deposited in

22   the deep lung (alveoli) which may be removed either up through the respiratory tract (bronchi)

23   by alveolobronchiolar transport or through the lymphatic system. The pivotal mechanism of

24   alveolobronchiolar transport involves the movement of alveolar macrophages with ingested

25   particles to the bottom of the conducting airways. These airways are lined with ciliated epithelial

26   cells and cells that produce mucous, which surrounds the macrophages. The ciliated epithelial

27   cells move the mucous packets up the resiratory tract, hence the term "mucociliary escalator."

28   Although some studies show reduced tracheobronchial clearance after O_3 exposure, alveolar

29   clearance of deposited material is accelerated, presumably due to macrophage influx, which in

30   itself can be damaging.

1   With regard to adaptive immunity, a limited number of epidemiologic studies have

2   examined associations between O_3 exposure and hospital admissions or ED visits for respiratory

3   infection, pneumonia, or influenza. Results have been mixed, and in some cases conflicting (U.S.

4   EPA, 2013, Sections 6.2.7.2 and 6.2.7.3). With the exception of influenza, it is difficult to

5   ascertain whether cases of respiratory infection or pneumonia are of viral or bacterial etiology.

6   A recent study that examined the association between O_3 exposure and respiratory hospital

7   admissions in response to an increase in influenza intensity did observe an increase in respiratory

8   hospital admissions (Wong et al., 2009), but information from toxicological studies of O_3 and

9   viral infections is ambiguous.

10   In summary, relatively few studies conducted since the last review have evaluated the

11   effects of O_3 exposures on lung host defense. When the available evidence is taken as a whole,

12   the ISA concludes that acute O_3 exposures impair the host defense capability of animals,

13   primarily by depressing alveolar macrophage function and perhaps also by decreasing

14   mucociliary clearance of inhaled particles and microorganisms. Coupled with limited evidence

15   from controlled human exposure studies, this suggests that humans exposed to O_3 could be

16   predisposed to bacterial infections in the lower respiratory tract (EPA, 2013, section 6.2.5.5).

17   The seriousness of such infections may depend on how quickly bacteria develop virulence

18   factors and how rapidly PMNs are mobilized to compensate for the deficit in alveolar

19   macrophage function.

20   **Allergic and Asthma-Related Responses**

21   Effects resulting from combined exposures to O_3 and allergens have been studied in a

22   variety of animal species, generally as models of experimental asthma. Pulmonary function and

23   AHR in animal models of asthma are discussed in detail in Section 6.2.1.3 and Section 6.2.2.2,

24   respectively, in the ISA (U.S. EPA, 2013). Studies of allergic and asthma-related responses are

25   discussed in detail in sections 5.3.6 and 6.2.6 of the ISA (U.S. EPA, 2013).

26   Evidence available in the last review indicates that O_3 exposure skews immune responses

27   toward an allergic phenotype. For example, Gershwin et al. (1981) reported that O_3 (800 and 500

28   ppb for 4 days) exposure caused a 34-fold increase in the number of IgE (allergic antibody)-

29   containing cells in the lungs of mice. In general, the number of IgE-containing cells correlated

30   positively with levels of anaphylactic sensitivity. In humans, allergic rhinoconjunctivitis

1   symptoms are associated with increases in ambient O_3 concentrations (Riediker et al., 2001).

2   Controlled human exposure studies have observed O_3-induced changes indicating allergic

3   skewing. Airway eosinophils, which are white blood cells that participate in allergic disease and

4   inflammation, were observed to increase in volunteers with atopy[21] and mild asthma (Peden et

5   al., 1997). In a more recent study, expression of IL-5, a cytokine involved in eosinophil

6   recruitment and activation, was increased in subjects with atopy but not in healthy subjects

7   (Hernandez et al., 2010). Epidemiologic studies describe associations between eosinophils in

8   both short- (U.S. EPA, 2013, Section 6.2.3.2) and long-term (U.S. EPA, 2013, Section 7.2.5) O_3

9   exposure, as do chronic exposure studies in non-human primates. Collectively, findings from

10   these studies suggest that O_3 can induce or enhance certain components of allergic inflammation

11   in individuals with allergy or allergic asthma.

12   Evidence available in the last review indicates that ozone may also increase AHR to

13   specific allergen triggers (75 FR 2970, January 19, 2010). Two studies (Jörres et al., 1996; Holz

14   et al., 2002) observed increased airway responsiveness to O_3 exposure with bronchial allergen

15   challenge in subjects with preexisting allergic airway disease. Ozone-induced exacerbation of

16   airway responsiveness persists longer and attenuates more slowly than O_3-induced lung function

17   decrements and respiratory symptom responses and can have important clinical implications for

18   asthmatics.

19   Animal toxicology studies indicate that O_3 enhances inflammatory and allergic responses

20   to allergen challenge in sensitized animals. In addition to exacerbating existing allergic

21   responses, toxicology studies indicate that O_3 can also act as an adjuvant to produce sensitization

22   in the respiratory tract. Along with its pro-allergic effects (inducing or enhancing certain

23   components of allergic inflammation in individuals with allergy or allergic asthma), O_3 could

24   also make airborne allergens more allergenic. When combined with NO_2, O_3 has been shown to

25   enhance nitration of common protein allergens, which may increase their allergenicity Franze et

26   al. (2005).

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[21] Atopy is a predisposition toward developing certain allergic hypersensitivity reactions. A person with atopy typically presents with one or more of the following: eczema (atopic dermatitis), allergic rhinitis (hay fever), allergic conjunctivitis, or allergic asthma.

1   **Hospital Admissions and Emergency Department Visits**

2   The 2006 O_3 AQCD evaluated numerous studies of respiratory-related emergency

3   department visits and hospital admissions. These were primarily time-series studies conducted in

4   the U.S., Canada, Europe, South America, Australia, and Asia. Based on such studies, the 2006

5   O_3 AQCD concluded that "the overall evidence supports a causal relationship between acute

6   ambient O_3 exposures and increased respiratory morbidity resulting in increased ED visits and

7   [hospital admissions] during the warm season[22]" (U.S. EPA, 2006). This conclusion was

8   "strongly supported by the human clinical, animal toxicologic[al], and epidemiologic evidence

9   for [O_3-induced] lung function decrements, increased respiratory symptoms, airway

10   inflammation, and airway hyperreactivity" (U.S. EPA, 2006).

11   The results of recent studies largely support the conclusions of the 2006 O_3AQCD (U.S.

12   EPA, 2013, section 6.2.7). Since the completion of the 2006 O_3 AQCD, relatively fewer studies

13   conducted in the U.S., Canada, and Europe have evaluated associations between short-term O_3

14   concentrations and respiratory hospital admissions and emergency department visits, with a

15   growing number of studies conducted in Asia. This epidemiologic evidence is summarized in

16   Appendix 3-B and discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.7).

17   In considering this body of evidence, the ISA focused primarily on multicity studies

18   because they examine associations with respiratory-related hospital admissions and emergency

19   department visits over large geographic areas using consistent statistical methodologies (U.S.

20   EPA, 2013, section 6.2.7.1). The ISA also focused on single-city studies that encompassed a

21   large number of hospital admissions or emergency department visits, included long study-

22   durations, were conducted in locations not represented by the larger studies, or examined

23   population-specific characteristics that were not evaluated in the larger studies (U.S. EPA, 2013,

24   section 6.2.7.1). When examining the association between short-term O_3 exposure and

25   respiratory health effects that require medical attention, the ISA distinguishes between hospital

26   admissions and emergency department visits because it is likely that a small percentage of

27   respiratory emergency department visits will be admitted to the hospital, therefore respiratory

28   emergency department visits may represent potentially less serious, but more common outcomes

29   (U.S. EPA, 2013, section 6.2.7.1).

30   The results of studies evaluated in this review largely support the conclusions of the 2006

31   AQCD. Several recent multicity studies (e.g., Cakmak et al., 2006; Dales et al., 2006) and a

32   multi-continent study (Katsouyanni et al., 2009) report associations between short-term O_3

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[22]Epidemiologic associations for O_3 are more robust during the warm season than during cooler months (e.g.,
smaller measurement error, less potential confounding by copollutants). Rationale for focusing on warm season
epidemiologic studies for O_3 can be found at 72 FR 37838-37840.

1   concentrations and increased respiratory-related hospital admissions and emergency department

2   visits. These multicity studies are supported by consistent results from single-city studies using

3   different exposure assignment approaches (i.e., average of multiple monitors, single monitor,

4   population-weighted average) and averaging times (i.e., 1-hour max and 8-hour max) (U.S. EPA,

5   2013, sections 6.2.7.1 to 6.2.7.5). Recent studies also report associations with hospital

6   admissions and emergency department visits for asthma (Strickland et al., 2010; Stieb et al.,

7   2009) and COPD (Stieb et al., 2009; Medina-Ramon et al., 2006), with more limited evidence for

8   pneumonia (Medina-Ramon et al., 2006; Zanobetti and Schwartz, 2006). In seasonal analyses

9   (Figure 3-2 below; U.S. EPA, 2013, Figure 6-19, Table 6-28), stronger associations were

10   reported in the warm season or summer months (red circles) compared to the cold season (blue

11   circles), particularly for asthma (Strickland et al., 2010; Ito et al., 2007b) and COPD (Medina-

12   Ramon et al., 2006). The available evidence indicates that children are at greatest risk for

13   O_3-induced respiratory effects (Silverman and Ito, 2010; Strickland et al., 2010; Mar and Koenig,

14   2009; Villeneuve et al., 2007; Dales et al., 2006).

15   Although the collective evidence across studies indicates a mostly consistent positive

16   association between O_3 exposure and respiratory-related hospital admissions and ED visits, the

17   magnitude of these associations may be underestimated due to behavioral modification in

18   response to air quality forecasts (U.S. EPA, 2013, Section 4.6.6).

Figure 3-2. Percent increase in respiratory-related hospital admission and emergency department visits in studies that presented all-year and/or seasonal results.

Note: Effect estimates are for a 20 ppb increase in 24-h; 30 ppb increase in 8-h max; and 40 ppb increase in 1-h max O_3 concentrations. HA=hospital admission; ED=emergency department. Black=All-year analysis; Red=Summer only analysis; Blue=Winter only analysis.

[a] Wheeze used as indicator of lower respiratory disease.

[b] APHENA-Canada results standardized to approximate IQR of 5.1 ppb for 1-h max O_3 concentrations.

[c] Study included 8 cities; but of those 8, only 4 had O_3 data.

[d] non-ICU effect estimates.

[e] The study did not specify the lag day of the summer season estimate.

1

2
3

1   Studies examining the potential confounding effects of copollutants have reported that O_3

2   effect estimates remained relatively robust upon the inclusion of PM and gaseous pollutants in

3   two-pollutant models (U.S. 2013, Figure 6-20, Table 6-29). Additional studies that conducted

4   copollutant analyses, but did not present quantitative results, also support these conclusions

5   (Strickland et al., 2010; Tolbert et al., 2007; Medina-Ramon et al., 2006) (U.S. 2013, section

6   6.2.7.5).

7   In the last review, studies had not evaluated the concentration-response relationship

8   between short-term O_3 exposure and respiratory-related hospital admissions and emergency

9   department visits. A preliminary examination of this relationship in studies that have become

10   available since the last review found no evidence of a deviation from linearity when examining

11   the association between short-term O_3 exposure and asthma hospital admissions (U.S. EPA,

12   2013, page 6-157, and Silverman and Ito, 2010). In addition, an examination of the

13   concentration-response relationship for O_3 exposure and pediatric asthma emergency department

14   visits found no evidence of a threshold at O_3 concentrations as low as 30 ppb (for 8-hour daily

15   maximum concentrations) (Strickland et al., 2010). However, in both studies there is uncertainty

16   in the shape of the concentration-response curve at the lower end of the distribution of O_3

17   concentrations due to the low density of data in this range (U.S. 2013, page 6-157).

18   **Respiratory Mortality**

19   The controlled human exposure, epidemiologic, and toxicological studies discussed in

20   section 6.2 of the ISA (U.S. EPA, 1013a, section 6.2) provide strong evidence for respiratory

21   morbidity effects, including ED visits and hospital admissions, in response to short-term O_3

22   exposures. Moreover, evidence from experimental studies indicates multiple potential pathways

23   of respiratory effects from short-term O_3 exposures, which support the continuum of respiratory

24   effects that could potentially result in respiratory-related mortality in adults (U.S. EPA, 1013a,

25   section 6.2.8). The 2006 O_3 AQCD found inconsistent evidence for associations between short-

26   term O_3 concentrations and respiratory mortality (U.S. EPA, 2006). Although some studies

27   reported a strong positive association between O_3 and respiratory mortality, additional studies

28   reported small associations or no associations. New epidemiologic evidence for respiratory

29   mortality is discussed in detail in section 6.2.8 of the ISA (U.S. EPA, 2013). The majority of

30   recent multicity studies have reported positive associations between short-term O_3 exposures and

31   respiratory mortality, particularly during the summer months (U.S. EPA, 2013, Figure 6-36).

32   Specifically, recent multicity studies from the U.S. (Zanobetti and Schwartz, 2008b),

33   Europe (Samoli et al., 2009), Italy (Stafoggia et al., 2010), and Asia (Wong et al., 2010), as well

34   as a multi-continent study (Katsouyanni et al., 2009), reported associations between short-term

1 O_3 concentrations and respiratory mortality (U.S. EPA, 2013, Figure 6-37, page 6-259). With

2 respect to respiratory mortality, summer-only analyses were consistently positive and most were

3 statistically significant. All-year analyses had more mixed results, but most were positive.

4   Of the studies evaluated, only the studies by Katsouyanni et al. (2009) and by Stafoggia

5 et al. (2010) analyzed the potential for copollutant confounding of the O_3-respiratory mortality

6 relationship. Based on the results of these analyses, the ISA concluded that O_3 respiratory

7 mortality risk estimates appear to be moderately to substantially sensitive (e.g., increased or

8 attenuated) to inclusion of PM_{10}. However, in the APHENA study, the mostly every-6th-day

9 sampling schedule for PM_{10} in the Canadian and U.S. datasets greatly reduced their sample size

10 and limits the interpretation of these results (U.S. EPA, 2013, section 6.2.8).

11   In summary, recent epidemiologic studies support and reinforce the epidemiologic

12 evidence for O_3-associated respiratory hospital admissions and emergency department visits

13 from the last review. In addition, the evidence for associations with respiratory mortality has

14 been strengthened considerably since the last review, with the addition of several large multicity

15 studies. The plausibility of the associations reported in these studies is supported by the

16 experimental evidence for respiratory effects.

17   **3.1.2.2 Respiratory Effects – Long-term Exposures**

18 • **To what extent does the currently available scientific evidence, including related**
19 **uncertainties, strengthen or alter our understanding from the last review of**
20 **respiratory effects attributable to long-term O_3 exposures?**

21   As recognized in section 3.1.2.1, "the clearest evidence for health effects associated with

22 exposure to O_3 is provided by studies of respiratory effects" (U.S. EPA, 2013, section 1, p. 1-6).

23 Collectively, there is a vast amount of evidence spanning several decades that supports a causal

24 association between exposure to O_3 and a continuum of respiratory effects (U.S. EPA, 2013,

25 section 2.5). While the majority of this evidence is derived from studies investigating short-term

26 exposures, evidence from animal toxicological studies and recent epidemiologic evidence

27 indicate that long-term exposures (i.e., months to years) may also be detrimental to the

28 respiratory system. Across this evidence, particularly the epidemiologic evidence, the exposures

29 of focus vary, often involving repeated short concentrations extending over a long period, rather

30 than a continuous long-term exposure period.

31   In the 2006 O_3 AQCD, evidence was examined for relationships between long-term O_3

32 exposure and effects on respiratory health outcomes including declines in lung function,

33 increases in inflammation, and development of asthma in children and adults. Animal toxicology

34 data provided a clearer picture indicating that long-term O_3 exposure may have lasting effects.

1   Chronic[23] exposure studies in animals have reported biochemical and morphological changes
2   suggestive of irreversible long-term O_3 impacts on the lung. In contrast to supportive evidence
3   from chronic animal studies, the epidemiologic studies on longer-term (annual) lung function
4   declines, inflammation, and new asthma development remained inconclusive.

5   Several epidemiologic studies collectively indicated that O_3 exposure averaged over
6   several summer months was associated with smaller increases in lung function growth in
7   children. For longer averaging periods (annual), the analysis in the Children's Health Study
8   (CHS) reported by Gauderman et al. (2004) provided little evidence that such long-term
9   exposure to ambient O_3 was associated with significant deficits in the growth rate of lung
10   function in children. Limited epidemiologic research examined the relationship between long-
11   term O_3 exposures and inflammation. Cross-sectional studies detected no associations between
12   long-term O_3 exposures and asthma prevalence, asthma-related symptoms or allergy to common
13   aeroallergens in children. However, longitudinal studies provided evidence that long-term O_3
14   exposure influences the risk of asthma development in children and adults.

15   The currently available body of evidence supporting a relationship between long-term O_3
16   exposures and adverse respiratory health effects that is likely to be causal is discussed in detail in
17   the ISA (EPA 2013, section 7.2). New evidence reports interactions between genetic variants and
18   long-term O_3 exposure affect the occurrence of new-onset asthma in multi-community, U.S.
19   cohort studies where protection by specific oxidant gene variants was restricted to children living
20   in low O_3 communities. A new line of evidence reports a positive concentration-response
21   relationship between first asthma hospitalization and long-term O_3 exposure. Related studies
22   report coherent relationships between asthma severity and control, and respiratory symptoms
23   among asthmatics and long-term O_3 exposure. There is also limited evidence for an association
24   between long-term exposure to ambient O_3 concentrations and respiratory mortality. These
25   studies are summarized briefly below for new-onset asthma and asthma prevalence, asthma
26   hospital admissions and other morbidity effects, pulmonary structure and function, and
27   respiratory mortality.

28   Currently available scientific evidence of the adverse health effects attributable to long-
29   term O_3 exposures, even considering related uncertainties, is much stronger than the body of
30   evidence available at the time of the 2008 review of the O_3 standard. The 2006 O_3 AQCD (U.S.
31   EPA 2006) concluded that epidemiologic studies provided no evidence of associations between
32   long-term (annual) O_3 exposures and asthma-related symptoms, asthma prevalence, or allergy to

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[23] Unless otherwise specified, the term "chronic" generally refers to an annual exposure duration for epidemiologic studies and a duration of greater than 10% of the lifespan of the animal in toxicological studies.

1 common allergens after controlling for covariates. It found limited evidence for a relationship
2 between long-term exposures to ambient O_3 and deficits in the growth rate of lung-function in
3 children, pulmonary inflammation and other endpoints. Episodic exposures were also known to
4 cause more severe pulmonary morphological changes than continuous exposure.

5   The evidence base available in this review includes additional epidemiologic studies
6 using a variety of designs and analysis methods evaluating the relationship between long-term O_3
7 exposures and measures of respiratory morbidity and mortality effects conducted by different
8 research groups in different locations. The ISA (U.S. EPA, 2013, p. 7-33), in Table 7-2 presents
9 selected key new longitudinal and cross-sectional studies of respiratory health effects and
10 associated O_3 concentrations. The positive results from various designs and locations support a
11 relationship between long-term exposure to ambient O_3 and respiratory health effects and
12 mortality.

13   In this review, the evidence of effects associated with long-term exposures strengthens
14 the relationship between O_3 exposure and health effects defined as adverse by the American
15 Thoracic Society (ATS), a definition that has been used in previous reviews of the O_3 standard.
16 As discussed in more detail in section 3.1.3 below, the ATS (1985) defined adverse as
17 "medically significant physiologic or pathologic changes generally evidenced by one or more of
18 the following: (1) interference with the normal activity of the affected person or persons, (2)
19 episodic respiratory illness, (3) incapacitating illness, (4) permanent respiratory injury, and/or (5)
20 progressive respiratory dysfunction." As discussed below, in this review there is now credible
21 evidence of respiratory health effects associated with long-term O_3 exposures that would fall in
22 to each of these five categories that define adversity.

23   From a policy perspective, the recent epidemiologic studies from the CHS of long-term
24 O_3 exposures that shed light on the interaction between genetic variability, O_3 exposures, and
25 health effects in children are important, not only because they help clarify previous findings, but
26 also because the effects evaluated, such as new-onset asthma, are clearly adverse. The ISA (U.S.
27 EPA, 2013, p. 7-12) notes that the collective evidence from CHS provides an important
28 demonstration of gene-environment interactions. It further notes that in the complex
29 gene-environment setting a modifying effect might not be reflected in an exposure main effect
30 and that the simultaneous occurrence of main effect and interaction effect can occur. Moreover,
31 the study of gene-environment interactions elucidates disease mechanisms in humans by using
32 information on susceptibility genes to focus on the biological pathways that are most relevant to
33 that disease (Hunter, 2005).

34   In the CHS cohort of children in 12 Southern California communities, long-term
35 exposure to O_3 concentrations was not associated with increased risk of developing asthma

1	(McConnell et al., 2010); however, greater outdoor exercise was associated with development of
2	asthma in children living in communities with higher ambient O_3 concentrations (McConnell et
3	al., 2002). Recent CHS studies examined interactions among genetic variants, long-term O_3
4	exposure, and new onset asthma in children. These prospective cohort studies are
5	methodologically rigorous epidemiologic studies, and evidence indicates gene-O_3 interactions.
6	These studies have provided data supporting decreased risk of certain genetic variants on new
7	onset asthma (e.g., HMOX-1, ARG) that is limited to children either in low (Islam et al., 2008)
8	or high (Salam et al., 2009) O_3 communities. Gene-environment interaction also was
9	demonstrated with findings that greater outdoor exercise increased risk of asthma in GSTP1
10	Ile/Ile children living in high O_3 communities (Islam et al., 2009). Biological plausibility for
11	these gene-O_3 environment interactions is provided by evidence that these enzymes have
12	antioxidant and/or anti-inflammatory activity and participate in well recognized modes of action
13	in asthma pathogenesis. As O_3 is a source of oxidants in the airways, oxidative stress serves as
14	the link among O_3 exposure, enzyme activity, and asthma. Cross-sectional studies by Akinbami
15	et al. (2010) and Hwang et al. (2005) provide further evidence relating O_3 exposures with asthma
16	prevalence.

17	Studies using a cross-sectional design provide support for a relationship between long-
18	term O_3 exposure and adverse health effects in asthmatics, including: bronchitic symptoms
19	(related to TNF-308 genotype in asthmatic children) (Lee et al., 2009b); asthma severity (Rage et
20	al., 2009b) and asthma control (Jacquemin et al., 2012) in an adult cohort; respiratory-related
21	school absences (related to CAT and MPO variant genes) (Wenten et al., 2009); asthma ED
22	visits in adults (Meng et al., 2010); and, asthma hospital admissions in adults and children (Lin et
23	al., 2008; Meng et al., 2010; Moore et al., 2008). Several studies, shown in Table 7-3 (ISA, U.S.
24	EPA, 2013, p. 7-35), provide results adjusted for potential confounders presenting results for
25	both O_3 and PM (in single and multipollutant models) as well as other pollutants where PM
26	effects were not provided. As shown in this table, O_3 associations were generally robust to
27	adjustment by potential confounding by PM.

28	Information from toxicological studies in nonhuman primates indicates that long term
29	exposure to O_3 during gestation or development can result in irreversible morphological changes
30	in the lung, which in turn can influence the function of the respiratory tract. This nonhuman
31	primate evidence of an O_3-induced change in airway responsiveness supports the biologic
32	plausibility of long term exposure to O_3 contributing to effects of asthma in children. However,
33	results from epidemiologic studies examining long-term O_3 exposure and pulmonary function
34	effects are inconclusive with some new studies relating effects at higher exposure levels.

1   The ISA (U.S. EPA, 2013, p. 7-31) concludes that there is limited evidence for an

2   association between long-term exposure to ambient O_3 concentrations and respiratory mortality

3   in adults (Jerrett et al., 2009). This effect was robust to the inclusion of $PM_{2.5}$ and insensitive to a

4   number of different model specifications. Moreover, there is evidence that long-term exposure to

5   O_3 is associated with mortality among individuals that had previously experienced an emergency

6   hospital admission due to COPD (Zanobetti and Schwartz, 2011).

7   In conclusion, since the last review, the body of evidence about the effects of long-term

8   O_3 exposure has been considerably strengthened. The scientific evidence available for this

9   review, including related uncertainties, provides an overall strong body of evidence of adverse

10   health effects attributable to long-term O_3 exposures. These include a coherent range of asthma

11   morbidity effects such as new-onset asthma, asthma prevalence, symptoms, school absences, ED

12   visits and hospital admissions. There is also new evidence of respiratory mortality associated

13   with long-term O_3 exposure. Further discussion of key studies is below.

14   **New-onset Asthma and Asthma Prevalence**

15   Asthma is a heterogeneous disease with a high degree of temporal variability. The on-set,

16   progression, and symptoms can vary within an individual's lifetime, and the course of asthma

17   may vary markedly in young children, older children, adolescents, and adults. In the previous

18   review, longitudinal cohort studies that examined associations between long-term O_3 exposures

19   and the onset of asthma in adults and children indicated a direct effect of long-term O_3 exposures

20   on asthma risk in adults (McDonnell et al., 1999a, 15-year follow-up; Greer et al., 1993, 10-year

21   follow-up) and effect modification by O_3 in children (McConnell et al., 2002). Since that review,

22   important new evidence has become available about the association between long-term

23   exposures to O_3 and new-onset asthma that has increased our understanding of the gene-

24   environment interaction and the mechanisms and biological pathways most relevant to assessing

25   O_3-related effects.

26   In children, the relationship between long-term O_3 exposure and new-onset asthma has

27   been extensively studied in the CHS; a long-term study that was initiated in the early1990's

28   which has evaluated effects in several cohorts of children. The CHS was initially designed to

29   examine whether long-term exposure to ambient pollution was related to chronic respiratory

30   outcomes in children in 12 communities in southern California. In the CHS, new-onset asthma

31   was classified as having no prior history of asthma at study entry with subsequent report of

32   physician-diagnosed asthma at follow-up, with the date of onset assigned to be the midpoint of

33   the interval between the interview date when asthma diagnosis was first reported and the

34   previous interview date. The results of one study (McConnell et al., 2002) available in the

1 previous review indicated that within high O_3 communities, asthma risk was 3.3 times greater for
2 children who played three or more outdoor sports as compared with children who played no
3 sports.

4   For this review, as discussed in section 7.2.1.1 of the ISA (U.S. EPA 2013), recent
5 studies from the CHS provide evidence for gene-environment interactions in effects on new-
6 onset asthma by indicating that the lower risks associated with specific genetic variants are found
7 in children who live in lower O_3 communities. These studies indicate that the risk for new-onset
8 asthma is related in part to genetic susceptibility, as well as behavioral factors and environmental
9 exposure. The onset of a chronic disease, such as asthma, is partially the result of a sequence of
10 biochemical reactions involving exposures to various environmental agents metabolized by
11 enzymes related to a number of different genes. Oxidative stress has been proposed to underlie
12 the mechanistic hypotheses related to O_3 exposure. Genetic variants may impact disease risk
13 directly, or modify disease risk by affecting internal dose of pollutants and other environmental
14 agents and/or their reaction products, or by altering cellular and molecular modes of action.
15 Understanding the relation between genetic polymorphisms and environmental exposure can
16 help identify high-risk subgroups in the population and provide better insight into pathway
17 mechanisms for these complex diseases.

18   The CHS analyses (Islam et al., 2008; Islam et al. 2009; Salam et al., 2009) have found
19 that asthma risk is related to interactions between O_3 and variants in genes for enzymes such as
20 heme-oxygenase (HO-1), arginases (ARG1 and 2), and glutathione S transferase P1 (GSTP1).
21 Biological plausibility for these findings is provided by evidence that these enzymes have
22 antioxidant and/or anti-inflammatory activity and participate in well-recognized modes of action
23 in asthma pathogenesis. Further, several lines of evidence demonstrate that secondary oxidation
24 products of O_3 initiate the key modes of action that mediate downstream health effects (ISA,
25 Section 5.3, U.S. EPA, 2013). For example, HO-1 responds rapidly to oxidants, has anti-
26 inflammatory and anti-oxidant effects, relaxes airway smooth muscle, and is induced in the
27 airways during asthma. Gene-environment interactions are discussed in detail in Section 5.4.2.1
28 in the ISA (U.S. EPA, 2013).

29 **Asthma Hospital Admissions**

30   In the 2006 AQCD, studies on O_3-related hospital discharges and emergency department
31 (ED) visits for asthma and respiratory disease mainly looked at short-term (daily) metrics. The
32 short-term O_3 studies presented in section 6.2.7.5 of the ISA (U.S. EPA 2013) and discussed
33 above in section 3.1.2.1 continue to indicate that there is evidence for increases in both hospital
34 admissions and ED visits in children and adults related to all respiratory outcomes, including

1   asthma, with stronger associations in the warm months. New studies, discussed in section 7.2.2

2   of the ISA (U.S. EPA, 2013) also evaluated long-term O_3 exposure metrics, providing a new line

3   of evidence that suggests a positive exposure-response relationship between the first hospital

4   admission for asthma and long-term O_3 exposure, although the ISA cautions in attributing the

5   associations in that study to long-term exposures since there is potential for short-term exposures

6   to contribute to the observed associations.

7   Evidence associating long-term O_3 exposure to first asthma hospital admission in a

8   positive concentration-response relationship is provided in a retrospective cohort study (Lin et

9   al., 2008b). This study investigated the association between chronic exposure to O_3 and

10 childhood asthma admissions by following a birth cohort of more than 1.2 million babies born in

11 New York State (1995-1999) to first asthma admission or until 31 December 2000. Three annual

12 indicators (all 8-hour maximum from 10:00 a.m. to 6:00 p.m.) were used to define chronic O_3

13 exposure: (1) mean concentration during the follow-up period (41.06 ppb); (2) mean

14 concentration during the O_3 season (50.62 ppb); and (3) proportion of follow-up days with O_3

15 levels >70 ppb. The effects of co-pollutants were controlled, and interaction terms were used to

16 assess potential effect modifications. A positive association between chronic exposure to O_3 and

17 childhood asthma hospital admissions was observed, indicating that children exposed to high O_3

18 levels over time are more likely to develop asthma severe enough to be admitted to the hospital.

19 The various factors were examined and differences were found for younger children (1-2 years),

20 poor neighborhoods, Medicaid/self-paid births, geographic region and others. As shown in the

21 ISA, Figure 7-3 (EPA 2013, p. 7-16), positive concentration-response relationships were

22 observed. Asthma admissions were significantly associated with increased O_3 levels for all

23 chronic exposure indicators.

24   In considering the relationship between long-term pollutant exposures and chronic

25 disease heath endpoints, where chronic pathologies are found with acute expression of chronic

26 disease, Künzli (2012) hypothesizes that if the associations of pollution with events are much

27 larger in the long-term studies, it provides some indirect evidence that air pollution increases the

28 pool of subjects with chronic disease, and that more acute events are to be expected to be seen

29 for higher exposures. The results of Lin et al (2008) for first asthma hospital admission,

30 presented in Figure 7-3 (EPA 2013, p. 7-16), show effects estimates that are larger than those

31 reported in a study of childhood asthma hospital admission in New York state (Silverman and

32 Ito, 2010), discussed in section 3.1.2.1 and 3.1.2.2 above. The ISA (U.S. EPA, 2013, p. 7-16)

33 notes that this provides some support for the hypothesis that O_3 exposure may not only have

34 triggered the events but also increased the pool of asthmatic children, but cautions in attributing

1 the associations in Lin et al. (2008) study to long-term exposures since there is potential for
2 short-term exposures to contribute to the observed associations.

3 **Pulmonary structure and function**

4   In the 2006 O_3 AQCD, few epidemiologic studies had investigated the effect of chronic
5 O_3 exposure on pulmonary function. The strongest evidence was for medium-term effects of
6 extended O_3 exposures over several summer months on lung function (FEV_1) in children, i.e.,
7 reduced lung function growth being associated with higher ambient O_3 levels. Short-term O_3
8 exposure studies presented in ISA (EPA, 2013, Section 6.2.1.2), and above in section 3.1.2.1,
9 provide a cumulative body of epidemiologic evidence that strongly supports associations
10 between ambient O_3 exposure and decrements in lung function among children. A recent study
11 (Rojas-Martinez et al., 2007) of long-term exposure to O_3, described in section 7.2.3.1 of the ISA
12 (U.S. EPA, 2013, p. 7-19), observed a relationship with pulmonary function declines in school-
13 aged children where O_3 and other pollutant levels were higher (90 ppb at high end of the range)
14 than those in the CHS. Two studies of adult cohorts provide mixed results where long-term
15 exposures were at the high end of the range.

16   Long-term studies in animals allow for greater insight into the potential effects of
17 prolonged exposure to O_3 that may not be easily measured in humans, such as structural changes
18 in the respiratory tract. Despite uncertainties, epidemiologic studies observing associations of O_3
19 exposure with functional changes in humans can attain biological plausibility in conjunction with
20 long-term toxicological studies, particularly O_3-inhalation studies performed in non-human
21 primates whose respiratory systems most closely resembles that of the human. An important
22 series of studies, discussed in section 7.2.3.2 of the ISA (U.S. EPA, 2013), have used nonhuman
23 primates to examine the effect of O_3 alone, or in combination with an inhaled allergen, house
24 dust mite antigen (HDMA), on morphology and lung function. These animals exhibit the
25 hallmarks of allergic asthma defined for humans, including: a positive skin test for HDMA with
26 elevated levels of IgE in serum and IgE-positive cells within the tracheobronchial airway walls;
27 impaired airflow which is reversible by treatment with aerosolized albuterol; increased
28 abundance of immune cells, especially eosinophils, in airway exudates and bronchial lavage; and
29 development of nonspecific airway responsiveness (NHLBI, 2007). These studies and others
30 have demonstrated changes in pulmonary function and airway morphology in adult and infant
31 nonhuman primates repeatedly exposed to environmentally relevant concentrations of O_3 (ISA,
32 section 7.2.3.2, 2013).

33   The initial observations in adult nonhuman primates have been expanded in a series of
34 experiments using infant rhesus monkeys repeatedly exposed to 0.5 ppm O_3 starting at 1 month

1   of age (Plopper et al., 2007). The purpose of these studies was to determine if a cyclic regimen of
2   O_3 inhalation would amplify the allergic responses and structural remodeling associated with
3   allergic sensitization and inhalation in the infant rhesus monkey. After several episodic
4   exposures of infant monkeys to O_3, they observed a significant increase in the baseline airway
5   resistance, which was accompanied by a small increase in airway responsiveness to inhaled
6   histamine (Schelegle et al., 2003), although neither measurement was statistically different from
7   filtered air control values. Exposure of animals to inhaled house dust mite antigen alone also
8   produced small but not statistically significant changes in baseline airway resistance and airway
9   responsiveness, whereas the combined exposure to both (O_3 + antigen) produced statistically
10   significant and greater than additive changes in both functional measurements. This nonhuman
11   primate evidence of an O_3-induced change in airway resistance and responsiveness provides
12   biological plausibility of long-term exposure, or repeated short-term exposures, to O_3
13   contributing to the effects of asthma in children.

14   To understand which conducting airways and inflammatory mechanisms are involved in
15   O_3-induced airway hyperresponsiveness in the infant rhesus monkey, results of a follow-up study
16   (Joad et al., 2006) suggest that effect of O_3 on airway responsiveness occurs predominantly in
17   the smaller bronchioles, where dosimetric models indicate the dose would be higher.
18   The functional changes in the conducting airways were accompanied by a number of cellular and
19   morphological changes, including a significant 4-fold increase in eosinophils. Thus, these studies
20   demonstrate both functional and cellular changes in the lung of infant monkeys after cyclic
21   exposure to 0.5 ppm O_3, providing relevant information to understanding the potentially
22   damaging effects of ambient O_3 exposure on the respiratory tract of children.

23   In addition, significant structural changes in the respiratory tract development, during
24   which conducting airways increase in diameter and length, have been observed in infant rhesus
25   monkeys after cyclic exposure to O_3 (Fanucchi et al., 2006). Observed changes included more
26   proximal first alveolar outpocketing, decreases in the diameter and length of the terminal and
27   respiratory bronchioles, increases in mucus-producing goblet cell mass, alterations in smooth
28   muscle orientation in the respiratory bronchioles, epithelial nerve fiber distribution, and
29   basement membrane zone morphometry. The latter effects are noteworthy because of their
30   potential contribution to airway obstruction and airway hyperresponsiveness which are central
31   features of asthma. A number of studies in both non-human primates and rodents demonstrate
32   that O_3 exposure can increase collagen synthesis and deposition, including fibrotic-like changes
33   in the lung (ISA, section 7.2.3.2, U.S. EPA, 2013).

34   Collectively, evidence from animal studies strongly suggests that chronic O_3 exposure is
35   capable of damaging the distal airways and proximal alveoli, resulting in lung tissue remodeling

1 and leading to apparent irreversible changes. Potentially, persistent inflammation and interstitial

2 remodeling play an important role in the progression and development of chronic lung disease.

3 Further discussion of the modes of action that lead to O_3-induced morphological changes can be

4 found in Section 5.3.7 of the ISA (U.S. EPA, 2013). Discussion of mechanisms involved in

5 lifestage susceptibility and developmental effects can be found in Section 5.4.2.4 of the ISA

6 (U.S. EPA, 2013). The findings reported in chronic animal studies offer insight into potential

7 biological mechanisms for the suggested association between seasonal O_3 exposure and reduced

8 lung function development in children as observed in epidemiologic studies (see Section 7.2.3.1).

9 **Respiratory Mortality**

10 A limited number of epidemiologic studies have assessed the relationship between long-

11 term exposure to O_3 and mortality in adults. The 2006 O_3 AQCD concluded that an insufficient

12 amount of evidence existed "to suggest a causal relationship between chronic O_3 exposure and

13 increased risk for mortality in humans" (U.S. EPA, 2006b). Though total and cardio-pulmonary

14 mortality were considered in these studies, respiratory mortality was not specifically considered.

15 In the most recent follow-up analysis of the ACS cohort (Jerrett et al., 2009), cardiopulmonary

16 deaths were separately subdivided into respiratory and cardiovascular deaths, rather than

17 combined as in the Pope et al. (2002) work. Increased O_3 exposure was associated with the risk

18 of death from respiratory causes, and this effect was robust to the inclusion of $PM_{2.5}$. The

19 association between increased O_3 concentrations and increased risk of death from respiratory

20 causes was insensitive to the use of different models and to adjustment for several ecologic

21 variables considered individually. Additionally, a recent multi-city time series study (Zanobetti

22 and Schwartz, 2011), which followed (from 1985 to 2006) four cohorts of Medicare enrollees

23 with chronic conditions that might predispose to O_3-related effects, observed an association

24 between long-term (warm season) exposure to O_3 and elevated risk of mortality in the cohort that

25 had previously experienced an emergency hospital admission due to COPD. A key limitation of

26 this study is the inability to control for $PM_{2.5}$, because data were not available in these cities until

27 1999.

28 **3.1.2.3 Total Mortality – Short-term Exposures**

29 - **To what extent does the currently available scientific evidence, including related**
30   **uncertainties, strengthen or alter our understanding from the last review of**
31   **mortality attributable to short-term O_3 exposures?**

32 The 2006 O_3 AQCD concluded that the overall body of evidence was highly suggestive

33 that short-term exposure to O_3 directly or indirectly contributes to nonaccidental and

34 cardiopulmonary-related mortality in adults, but additional research was needed to more fully

1 establish underlying mechanisms by which such effects occur (U.S. EPA, 2013, p. 2-18). In

2 building on the 2006 evidence, the ISA states the following (U.S. EPA, 2013, p. 6-261).

3 *The evaluation of new multicity studies that examined the association between*
4 *short-term O_3 exposures and mortality found evidence that supports the*
5 *conclusions of the 2006 AQCD. These new studies reported consistent positive*
6 *associations between short-term O_3 exposure and all-cause (non-accidental)*
7 *mortality, with associations persisting or increasing in magnitude during the*
8 *warm season, and provide additional support for associations between O_3*
9 *exposure and cardiovascular and respiratory mortality*

10 The 2006 O_3 AQCD reviewed a large number of time-series studies of associations

11 between short-term O_3 exposures and total mortality including single- and multicity studies, and

12 meta-analyses. In the large U.S. multicity studies that examined all-year data, effect estimates

13 corresponding to single-day lags ranged from a 0.5-1% increase in all-cause (nonaccidental) total

14 mortality per a 20 ppb (24-hour), 30 ppb (8-hour maximum), or 40 ppb (1-hour maximum)

15 increase in ambient O_3 (U.S. EPA, 2013, section 6.6.2). Available studies reported some

16 evidence for heterogeneity in O_3 mortality risk estimates across cities and across studies. Studies

17 that conducted seasonal analyses reported larger O_3 mortality risk estimates during the warm

18 season. Overall, the 2006 O_3 AQCD identified robust associations between various measures of

19 daily ambient O_3 concentrations and all-cause mortality, which could not be readily explained by

20 confounding due to time, weather, or copollutants. With regard to cause-specific mortality,

21 consistent positive associations were reported between short-term O_3 exposure and

22 cardiovascular mortality, with less consistent evidence for associations with respiratory

23 mortality. The majority of the evidence for associations between O_3 and cause-specific mortality

24 were from single-city studies, which had small daily mortality counts and subsequently limited

25 statistical power to detect associations. The 2006 O_3 AQCD concluded that "the overall body of

26 evidence is highly suggestive that O_3 directly or indirectly contributes to non-accidental and

27 cardiopulmonary-related mortality" (U.S. EPA, 2012a, section 6.6.1).

28 Recent studies have strengthened the body of evidence that supports the association

29 between short-term O_3 concentrations and mortality in adults. This evidence includes a number

30 of studies reporting associations with non-accidental as well as cause-specific mortality. Multi-

31 continent and multicity studies have consistently reported positive and statistically significant

32 associations between short-term O_3 concentrations and all-cause mortality, with evidence for

33 larger mortality risk estimates during the warm or summer months (Figure 3-3 below, reprinted

34 from the ISA) (U.S. EPA, 2013, Figure 6-27; Table 6-42). Similarly, evaluations of cause-

1   specific mortality have reported consistently positive associations with O_3, particularly in
2   analyses restricted to the warm season (U.S. EPA, 2013, Figure 6-37; Table 6-53).[24]

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[24]Respiratory mortality is discussed in more detail above.

1

2

Study	Location	Lag
Gryparis et al. (2004; 57276)	APHEA2 (23 cities)	0-1
Bell et al. (2007; 93256)	98 U.S. communities	0-1
Schwartz (2005; 57333)	14 U.S. cities	0
Bell and Dominici (2008; 193828)	98 U.S. communities	0-6
Bell et al. (2004; 94417)a	95 U.S. communities	0-6
Levy et al. (2005; 74347)a	U.S. and Non-U.S.	
Katsouyanni et al. (2009; 199899)	APHENA-Europe	DL(0-2)
Bell et al. (2005; 74345)a	U.S. and Non-U.S.	
Ito et al. (2005; 74346)a	U.S. and Non-U.S.	
Wong et al. (2010; 732535)	PAPA (4 cities)	0-1
Katsouyanni et al. (2009; 199899)	APHENA-U.S.	DL(0-2)
Cakmak et al. (2011, 699135)	7 Chilean cities	DL(0-6)
Katsouyanni et al. (2009; 199899)	APHENA-Canada	DL(0-2)
Katsouyanni et al. (2009; 199899)b	APHENA-Canada	DL(0-2)

All-Year

Study	Location	Lag
Samoli et al. (2009; 195855)	21 European cities	0-1
Bell et al. (2004; 94417)a	95 U.S. communities	0-6
Schwartz (2005; 57333)	14 U.S. cities	0
Zanobetti and Schwartz (2008; 195755)	48 U.S. cities	0
Zanobetti and Schwartz (2008; 101596)	48 U.S. cities	0-3
Franklin and Schwartz (2008; 156448)	18 U.S. communities	0
Gryparis et al. (2004; 57276)	APHEA2 (21 cities)	0-1
Medina-Ramon and Schwartz (2008)	48 U.S. cities	0-2
Katsouyanni et al. (2009; 199899)	APHENA-Europe	DL(0-2)
Bell et al. (2005; 74345)a	U.S. and Non-U.S.	
Katsouyanni et al. (2009; 199899)	APHENA-Canada	DL(0-2)
Katsouyanni et al. (2009; 199899)b	APHENA-Canada	DL(0-2)
Levy et al. (2005; 74347)a	U.S. and Non-U.S.	
Ito et al. (2005; 74346)a	U.S. and Non-U.S.	
Katsouyanni et al. (2009; 199899)	APHENA-U.S.	DL(0-2)
Stafoggia et al. (2010; 625034)	10 Italian cities	DL(0-5)

Summary

% Increase
-1   1   3   5   7   9   11

4

Figure 3-3.   Summary of mortality risk estimates for short-term O$_3$ and all-cause (nonaccidental) mortality.[25]

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[25]Reprinted from the ISA (U.S. EPA, 2013, Figure 6-27).

1   In assessing the evidence for O_3-related mortality, the 2006 AQCD also noted that
2   multiple uncertainties remained regarding the relationship between short-term O_3 concentrations
3   and mortality, including the extent of residual confounding by co-pollutants; characterization of
4   the factors that modify the O_3-mortality association; the appropriate lag structure for identifying
5   O_3-mortality effects; and the shape of the O_3-mortality concentration-response function and
6   whether a threshold exists. Many of the studies, published since the last review, have attempted
7   to address one or more of these uncertainties. The ISA (U.S. EPA, 2013; Section 6.6.2) discusses
8   the extent to which recent studies have evaluated these uncertainties in the relationship between
9   O_3 and mortality.

10   In particular, recent studies have evaluated different statistical approaches to examine the
11 shape of the O_3-mortality concentration-response relationship and to evaluate whether a
12 threshold exists for O_3-related mortality. In an analysis of the NMMAPS data, Bell et al. (2006)
13 evaluated the potential for a threshold in the O_3-mortality relationship. The authors reported
14 positive and statistically significant associations with mortality in a variety of restricted analyses,
15 including analyses restricted to days with 24-hour area-wide average O_3 concentrations below
16 60, 55, 50, 45, 40, 35, and 30 ppb. In these restricted analyses O_3 effect estimates were of similar
17 magnitude, were statistically significant, and had similar statistical precision. In analyses
18 restricted to days with 24-hour average O_3 concentrations below 25 ppb, the O_3 effect estimate
19 was similar in magnitude to the effect estimates resulting from analyses with the higher cutoffs,
20 but had somewhat lower statistical precision, with the estimate approaching statistical
21 significance (i.e., based on observation of Figure 2 in Bell et al., 2006). In analyses restricted to
22 days with lower 24-hour average O_3 concentrations (i.e., below 20 and 15 ppb), effect estimates
23 were similar in magnitude to analyses with higher cutoffs, but with notably less statistical
24 precision, and were not statistically significant (i.e., confidence intervals included no O_3-
25 associated mortality based on observation of Figure 2 in Bell et al., 2006). Ozone was no longer
26 positively associated with mortality when the analysis was restricted to days with 24-hour O_3
27 concentrations below 10 ppb. Given the relatively small number of days included in these
28 restricted analyses, especially for cut points of 20 ppb and below,[26] statistical uncertainty is
29 increased.

30   Bell et al. (2006) also evaluated the shape of the concentration-response relationship
31 between O_3 and mortality. Although the results of this analysis suggested the lack of threshold in
32 the O_3-mortality relationship, the ISA noted that it is difficult to interpret such a curve because:
33 (1) there is uncertainty around the shape of the concentration-response curve at 24-hour average

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[26]For example, Bell et al. (2006) reported that for analyses restricted to 24-hour O_3 concentrations at or below 20 ppb, 73% of days were excluded on average across the 98 communities.

1   O_3 concentrations generally below 20 ppb and (2) the concentration-response curve does not take

2   into consideration the heterogeneity in O_3-mortality risk estimates across cities (U.S. EPA, 2013,

3   section 6.6.2.3).

4   Several additional studies have used the NMMAPS dataset to evaluate the concentration-

5   response relationship between short-term O_3 concentrations and mortality. For example, using

6   the same data as Bell et al. (2006), Smith et al. (2009) conducted a subset analysis, but instead of

7   restricting the analysis to days with O_3 concentrations below a cutoff the authors only included

8   days *above* a defined cutoff. The results of this analysis were consistent with those reported by

9   Bell et al. (2006). Specifically, the authors reported consistent positive associations for all cutoff

10 concentrations up to concentrations where the total number of days available were so limited that

11 the variability around the central estimate was increased (U.S. EPA, 2013, section 6.6.2.3). In

12 addition, using NMMAPS data for 1987-1994 for Chicago, Pittsburgh, and El Paso, Xia and

13 Tong (2006) reported evidence for a threshold around a 24-hour average O_3 concentration of

14 25 ppb, though the threshold values estimated in the analysis were sometimes in the range of

15 where data density was low (U.S. EPA, 2013, section 6.6.2.3). Stylianou and Nicolich (2009)

16 examined the existence of thresholds following an approach similar to Xia and Tong (2006)

17 using data from NMMAPS for nine major U.S. cities (i.e., Baltimore, Chicago, Dallas/Fort

18 Worth, Los Angeles, Miami, New York, Philadelphia, Pittsburgh, and Seattle) for the years

19 1987-2000. The authors reported that the estimated O_3-mortality risks varied across the nine

20 cities, with the models exhibiting apparent thresholds in the 10-45 ppb range for O_3 (24-hour

21 average). However, given the city-to-city variation in risk estimates, combining the city-specific

22 estimates into an overall estimate complicated the interpretation of the results. Additional studies

23 in Europe, Canada, and Asia did not report the existence of a threshold (Katsouyanni et al.,

24 2009), with inconsistent and/or inconclusive results across cities, or a non-linear relationship in

25 the O_3-mortality concentration-response curve (Wong et al., 2010).

26 ### 3.1.2.4 Cardiovascular effects – Short-term Exposure

27 • **To what extent does the currently available scientific evidence, including related**
28 **uncertainties, strengthen or alter our understanding from the last review of**
29 **cardiovascular effects attributable to short-term O_3 exposures?**

30   A relatively small number of studies have examined the potential effect of short-term O_3

31 exposure on the cardiovascular system. The 2006 O_3 AQCD (U.S. EPA, 2006, p.8-77) concluded

32 that "O_3 directly and/or indirectly contributes to cardiovascular-related morbidity" but added that

33 the body of evidence was limited. This conclusion was based on a controlled human exposure

34 study that included hypertensive adult males; a few epidemiologic studies of physiologic effects,

1   heart rate variability, arrhythmias, myocardial infarctions, and hospital admissions; and

2   toxicological studies of heart rate, heart rhythm, and blood pressure.

3   More recently, the body of scientific evidence available that has examined the effect of

4   O_3 on the cardiovascular system has expanded. There is an emerging body of animal

5   toxicological evidence demonstrating that short-term exposure to O_3 can lead to autonomic

6   nervous system alterations (in heart rate and/or heart rate variability) and suggesting that

7   proinflammatory signals may mediate cardiovascular effects. Interactions of O_3 with respiratory

8   tract components result in secondary oxidation product formation and subsequent production of

9   inflammatory mediators, which have the potential to penetrate the epithelial barrier and to initiate

10   toxic effects systemically. In addition, animal toxicological studies of long-term exposure to O_3

11   provide evidence of enhanced atherosclerosis and ischemia/reperfusion (I/R) injury,

12   corresponding with development of a systemic oxidative, proinflammatory environment. Recent

13   experimental and epidemiologic studies have investigated O_3-related cardiovascular events and

14   are summarized in Section 6.3 of the ISA (U.S. EPA, 2013, Section 6.3). Overall, the ISA

15   summarized the evidence in this review as follows (U.S. EPA, 2013, p. 6-211).

16   *In conclusion, animal toxicological studies demonstrate O_3-induced*
17   *cardiovascular effects, and support the strong body of epidemiologic evidence*
18   *indicating O_3-induced cardiovascular mortality. Animal toxicological and*
19   *controlled human exposure studies provide evidence for biologically plausible*
20   *mechanisms underlying these O_3-induced cardiovascular effects. However, a lack*
21   *of coherence with epidemiologic studies of cardiovascular morbidity remains an*
22   *important uncertainty.*

23   Animal toxicological studies support that short-term O_3 exposure can lead to

24   cardiovascular morbidity. Animal studies provide evidence for both increased and decreased

25   heart rate (HR), however it is uncertain if O_3-induced reductions in HR are relevant to humans.

26   Animal studies also provide evidence for increased heart rate variability (HRV), arrhythmias,

27   vascular disease and injury following short-term O_3 exposure. In addition, a series of studies

28   highlight the role of genetic variability and age in the induction of effects and attenuation of

29   responses to O_3 exposure.

30   Biologically plausible mechanisms have been described for the cardiovascular effects

31   observed in animal exposure studies (U.S. EPA, 2013, Section 5.3.8). Evidence that

32   parasympathetic pathways may underlie cardiac effects is described in more detail in Section

33   5.3.2 of the ISA (U.S. EPA, 2013). Recent studies suggest that O_3 exposure may disrupt the

34   endothelin system that constricts blood vessels and increase blood pressure, which can result in

35   an increase in HR, HRV; and disrupt the $NO^{\cdot}$ system and the production of atrial natriuretic

36   factor (ANF), vasodilators that reduce blood pressure. Additionally, O_3 may increase oxidative

37   stress and vascular inflammation promoting the progression of atherosclerosis and leading to

1 increased susceptibility to I/R injury. As O_3 reacts quickly with the ELF and does not translocate
2 to the heart and large vessels, studies suggest that the cardiovascular effects exhibited could be
3 caused by secondary oxidation products resulting from O_3 exposure. However, direct evidence of
4 translocation of O_3 reaction products to the cardiovascular system has not been demonstrated *in*
5 *vivo*. Alternatively, extrapulmonary release of diffusible mediators (such as cytokines or
6 endothelins) may initiate or propagate inflammatory responses throughout the body leading to
7 the cardiovascular effects reported in toxicology studies. Ozone reacts within the lung to induce
8 pulmonary inflammation and the influx and activation of inflammatory cells, resulting in a
9 cascading proinflammatory state, and may lead to the extrapulmonary release of diffusible
10 mediators that could result in cardiovascular injury.

11   Controlled human exposures studies discussed in previous AQCDs have not
12 demonstrated any consistent extrapulmonary effects. In this review, evidence from controlled
13 human exposure studies suggests cardiovascular effects in response to short-term O_3 exposure
14 (see ISA, U.S. EPA, 2013, Section 6.3.1) and provides some coherence with evidence from
15 animal toxicology studies. Controlled human exposure studies also support the animal
16 toxicological studies by demonstrating O_3-induced effects on blood biomarkers of systemic
17 inflammation and oxidative stress, as well as changes in biomarkers that can indicate a
18 prothrombogenic response to O_3. Increases and decreases in high frequency HRV have been
19 reported following relatively low (120 ppb during rest) and high (300 ppb with exercise) O_3
20 exposures, respectively. These changes in cardiac function observed in animal and human studies
21 provide preliminary evidence for O_3-induced modulation of the autonomic nervous system
22 through the activation of neural reflexes in the lung (see ISA,U.S. EPA 2013, Section 5.3.2).

23   Overall, the ISA concludes that the available body of epidemiologic evidence examining
24 the relationship between short-term exposures to O_3 concentrations and cardiovascular morbidity
25 is inconsistent (U.S. EPA, 2013, Section 6.3.2.9). Across studies, different definitions, (i.e., ICD-
26 9 diagnostic codes) were used for both all-cause and cause-specific cardiovascular morbidity
27 (ISA, U.S. EPA, 2013, see Tables 6-35 to 6-39), which may contribute to inconsistency in
28 results. However, within diagnostic categories, no consistent pattern of association was found
29 with O_3. Generally, the epidemiologic studies used nearest air monitors to assess O_3
30 concentrations, with a few exceptions that used modeling or personal exposure monitors.
31 The inconsistencies in the associations observed between short-term O_3 and cardiovascular
32 disease (CVD) morbidities are unlikely to be explained by the different exposure assignment
33 methods used (see Section 4.6, ISA, U.S. EPA 2013). The wide variety of biomarkers considered
34 and the lack of consistency among definitions used for specific cardiovascular disease endpoints
35 (e.g., arrhythmias, HRV) make comparisons across studies difficult.

1   Despite the inconsistent evidence for an association between O_3 concentration and CVD

2   morbidity, mortality studies indicate a consistent positive association between short-term O_3

3   exposure and cardiovascular mortality in multicity studies and in a multicontinent study. When

4   examining mortality due to cardiovascular disease, epidemiologic studies consistently observe

5   positive associations with short-term exposure to O_3. Additionally, there is some evidence for an

6   association between long-term exposure to O_3 and mortality, although the association between

7   long-term ambient O_3 concentrations and cardiovascular mortality can be confounded by other

8   pollutants as evident by a study of cardiovascular mortality that reported no association after

9   adjustment for $PM_{2.5}$ concentrations. The ISA (U.S. EPA 2013, section 6.3.4) states that taken

10   together, the overall body of evidence across the animal and human studies is sufficient to

11   conclude that there is likely to be a causal reationship between relevant short-term exposures to

12   O3 and cardiovascular system effects.

13   **3.1.3   Adversity of Effects**

14   In this section we address the following question:

15   •   **To what extent does the currently available scientific evidence expand our**
16   **understanding of the adversity of O_3-related health effects?**

17   In making judgments as to when various O_3-related effects become regarded as adverse

18   to the health of individuals, in previous NAAQS reviews staff has relied upon the guidelines

19   published by the American Thoracic Society (ATS) and the advice of CASAC. In 2000, the ATS

20   published an official statement on "What Constitutes an Adverse Health Effect of Air

21   Pollution?" (ATS, 2000), which updated and built upon its earlier guidance (ATS, 1985). The

22   earlier guidance defined adverse respiratory health effects as "medically significant physiologic

23   changes generally evidenced by one or more of the following: (1) interference with the normal

24   activity of the affected person or persons, (2) episodic respiratory illness, (3) incapacitating

25   illness, (4) permanent respiratory injury, and/or (5) progressive respiratory dysfunction", while

26   recognizing that perceptions of "medical significance" and "normal activity" may differ among

27   physicians, lung physiologists and experimental subjects (ATS, 1985). The 2000 ATS guidance

28   builds upon and expands the 1985 definition of adversity in several ways. The guidance

29   concludes that transient, reversible loss of lung function in combination with respiratory

30   symptoms should be considered adverse. There is also a more specific consideration of

31   population risk (ATS, 2000). Exposure to air pollution that increases the risk of an adverse effect

32   to the entire population is adverse, even though it may not increase the risk of any individual to

33   an unacceptable level. For example, a population of asthmatics could have a distribution of lung

34   function such that no individual has a level associated with significant impairment. Exposure to

35   air pollution could shift the distribution to lower levels that still do not bring any individual to a

1   level that is associated with clinically relevant effects. However, this would be considered to be

2   adverse because individuals within the population would have diminished reserve function, and

3   therefore would be at increased risk to further environmental insult (U.S. EPA, 2013, p. lxxi; and

4   SO_2 NAAQS review, 75 FR at 35526/2, June 22, 2010).

5   The ATS also concluded that elevations of biomarkers such as cell types, cytokines and

6   reactive oxygen species may signal risk for ongoing injury and more serious effects or may

7   simply represent transient responses, illustrating the lack of clear boundaries that separate

8   adverse from nonadverse events. More subtle health outcomes also may be connected

9   mechanistically to health effects that are clearly adverse, so that small changes in physiological

10   measures may not appear clearly adverse when considered alone, but may be part of a coherent

11   and biologically plausible chain of related health outcomes that include responses that are clearly

12   adverse, such as mortality (section 3.1.2.1, above).

13   In this review, the new evidence provides further support for relationships between O_3

14   exposures and a spectrum of health effects, including effects that meet the ATS criteria for being

15   adverse (ATS, 1985 and 2000). The ISA judgment that there is a causal relationship between

16   short-term O_3 exposure and a full range of respiratory effects, including respiratory morbidity

17   (e.g., lung function decrements, respiratory symptoms, inflammation, hospital admissions, and

18   emergency department visits) and mortality, provides support for concluding that short-term O_3

19   exposure is associated with adverse effects (U.S. EPA 2013, section 2.5.2). Overall, including

20   new evidence of cardiovascular system effects, the evidence supporting an association between

21   short-term O_3 exposures and total (non-accidental, cardiopulmonary) respiratory mortality is

22   stronger in this review (U.S. EPA 2013, section 2.5.2). And the judgment of likely causal

23   associations between long-term measures of O_3 exposure and respiratory effects such as new-

24   onset asthma, prevalence of asthma, asthma symptoms and control, and asthma hospital

25   admissions provides support for concluding that long-term O_3 exposure is associated with

26   adverse effects ranging from episodic respiratory illness to permanent respiratory injury or

27   progressive respiratory decline (U.S. EPA 2013, section 7.2.8).

28   This review provides additional evidence of O_3-attributable effects that are clearly

29   adverse, including premature mortality. Application of the ATS guidelines to the least serious

30   category of effects related to ambient O_3 exposures, which are also the most numerous and

31   therefore are also important from a public health perspective, involves judgments about which

32   medical experts on CASAC panels and public commenters have in the past expressed diverse

33   views. To help frame such judgments, EPA staff defined gradations of individual functional

34   responses (e.g., decrements in FEV_1 and airway responsiveness) and symptomatic responses

35   (e.g., cough, chest pain, wheeze), together with judgments as to the potential impact on

36   individuals experiencing varying degrees of severity of these responses. These gradations were

1   used in the 1997 O_3 NAAQS review and slightly revised in the 2008 review (U.S. EPA, 1996, p.

2   59; 2007, p.3-72; 72 FR 37849, July 11, 2007). These gradations and impacts are summarized in

3   Tables 3-2 and 3-3 in the 2007 O_3 Staff Paper (U.S. EPA, 2007, pp.3-74 to 3-75).

4   For active healthy people, including children, moderate levels of functional responses

5   (e.g., FEV_1 decrements of $\geq 10\%$ but $< 20\%$, lasting 4 to 24 hours) and/or moderate symptomatic

6   responses (e.g., frequent spontaneous cough, marked discomfort on exercise or deep breath,

7   lasting 4 to 24 hours) would likely interfere with normal activity for relatively few sensitive

8   individuals (U.S. EPA, 2007, p.3-72; 72 FR 37849, July 11, 2007); whereas large functional

9   responses (e.g., FEV_1 decrements $\geq 20\%$, lasting longer than 24 hours) and/or severe

10   symptomatic responses (e.g., persistent uncontrollable cough, severe discomfort on exercise or

11   deep breath, lasting longer than 24 hours) would likely interfere with normal activities for many

12   sensitive individuals (U.S. EPA, 2007, p.3-72; 72 FR 37849, July 11, 2007) and therefore would

13   be considered adverse under ATS guidelines. For the purpose of estimating potentially adverse

14   lung function decrements in active healthy people in the 2008 O_3 NAAQS review, the CASAC

15   panel for that review indicated that a focus on the mid to upper end of the range of moderate

16   levels of functional responses is most appropriate (e.g., FEV_1 decrements $\geq 15\%$ but $< 20\%$)

17   (Henderson, 2006; 2007 Staff Paper, p. 3-76). However, for children and adults with lung

18   disease, even moderate functional (e.g., FEV_1 decrements $\geq 10\%$ but $< 20\%$, lasting up to 24

19   hours) or symptomatic responses (e.g., frequent spontaneous cough, marked discomfort on

20   exercise or with deep breath, wheeze accompanied by shortness of breath, lasting up to 24 hours)

21   would likely interfere with normal activity for many individuals, and would likely result in

22   additional and more frequent use of medication (U.S. EPA, 2007, p.3-72; 72 FR 37849, July 11,

23   2007). For people with lung disease, large functional responses (e.g., FEV_1 decrements $\geq 20\%$,

24   lasting longer than 24 hours) and/or severe symptomatic responses (e.g., persistent

25   uncontrollable cough, severe discomfort on exercise or deep breath, persistent wheeze

26   accompanied by shortness of breath, lasting longer than 24 hours) would likely interfere with

27   normal activity for most individuals and would increase the likelihood that these individuals

28   would seek medical treatment (U.S. EPA, 2007, p.3-72; 72 FR 37849, July 11, 2007). In the last

29   O_3 NAAQS review, for the purpose of estimating potentially adverse lung function decrements

30   in people with lung disease the CASAC panel indicated that a focus on the lower end of the

31   range of moderate levels of functional responses is most appropriate (e.g., FEV_1 decrements

32    $\geq 10\%$) (Henderson, 2006; 2007 Staff Paper, p. 3-76). In addition, in the reconsideration of the

33   2008 final decision, CASAC stated that "[a] 10% decrement in FEV1 can lead to respiratory

34   symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For

35   example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve

1   (i.e., decreased baseline FEV1) such that a ≥10% decrement could lead to moderate to severe

2   respiratory symptoms" (Samet, 2011) (section 3.1.2.1, above).

3   In judging the extent to which these impacts represent effects that should be regarded as

4   adverse to the health status of individuals, in previous NAAQS reviews we also considered

5   whether effects were experienced repeatedly during the course of a year or only on a single

6   occasion (Staff Paper, U.S. EPA, 2007). Although some experts would judge single occurrences

7   of moderate responses to be a ''nuisance,'' especially for healthy individuals, a more general

8   consensus view of the adversity of such moderate responses emerges as the frequency of

9   occurrence increases. Thus it has been judged that repeated occurrences of moderate responses,

10 even in otherwise healthy individuals, may be considered to be adverse since they could well set

11 the stage for more serious illness (61 FR 65723). The CASAC panel in the 1997 NAAQS review

12 expressed a consensus view that these "criteria for the determination of an adverse physiological

13 response were reasonable" (Wolff, 1995). In the review completed in 2008, estimates of repeated

14 occurrences continued to be an important public health policy factor in judging the adversity of

15 moderate lung function decrements in healthy and asthmatic people (72 FR 37850, July 11,

16 2007).

17   Evidence new to this review indicates that 6.6-hour exposures to 60 ppb O_3 during

18 moderate exertion can result in pulmonary inflammation in healthy adults. As discussed in

19 section 3.1.2 above, the initiation of inflammation can be considered as evidence that injury has

20 occurred. Inflammation induced by a single O_3 exposure can resolve entirely, but continued

21 acute inflammation can evolve into a chronic inflammatory state (ISA, U.S. EPA, 2013, p. 6-76),

22 which is clearly adverse. Therefore, like moderate lung function decrements, whether

23 inflammation is experienced repeatedly during the course of a year or only on a single occasion

24 is judged by staff to be reasonable criteria for determining adverse inflammatory effects

25 attributable to O_3 exposures at 60 ppb.

26   Responses measured in controlled human exposure studies indicate that the range of

27 effects elicited in humans exposed to ambient O_3 concentrations include: decreased inspiratory

28 capacity; mild bronchoconstriction; rapid, shallow breathing pattern during exercise; and

29 symptoms of cough and pain on deep inspiration (EPA, 2013, section 6.2.1.1). Some young,

30 healthy adults exposed to O_3 concentrations ≥ 60 ppb, while engaged in 6.6 hours of intermittent

31 moderate exertion, develop statistically significant reversible, transient decrements in lung

32 function, symptoms of breathing discomfort, and inflammation if minute ventilation or duration

33 of exposure is increased sufficiently (EPA, 2013, section 6.2.1.1). Among healthy subjects there

34 is considerable interindividual variability in the magnitude of the FEV_1 responses, but averaged

35 across studies at 60 ppb (EPA, 2013, pp. 6-17 to 6-18), 10% of healthy subjects had >10% FEV_1

36 decrements. The combination of lung function decrements and respiratory symptoms, which has

1 been considered adverse in previous reviews, has been demonstrated in healthy adults following

2 prolonged (6.6 hour) exposures, while at intermittent moderate exertion, to 70 ppb. For these

3 types of effects, information from controlled human exposure studies, which provides an

4 indication of the magnitude and thus adversity of effects at different O_3 concentrations,

5 combined with estimates of occurrences in the population from the HREA, provide information

6 about their importance from a policy perspective.

7 ### 3.1.4 Ozone Concentrations Associated With Health Effects

8 In evaluating O_3 exposure concentrations reported to result in health effects, within the

9 context of the adequacy of the current standard, we first consider the following specific question:

10 - **To what extent does the currently available scientific evidence indicate morbidity**
11 **and/or mortality attributable to exposures to O_3 concentrations lower than**
12 **previously reported or that would meet the current standard?**

13 In addressing this question, we characterize the extent to which O_3-attributable effects have been

14 reported over the ranges of O_3 exposure concentrations evaluated in controlled human exposure

15 studies and over the distributions of ambient O_3 concentrations in locations where epidemiologic

16 studies have been conducted.

17 ### 3.1.4.1 Concentrations in Controlled Human Exposure Studies and in Epidemiologic
18 Panel Studies

19 In considering what the currently available evidence indicates with regard to effects

20 associated with exposure concentrations lower than those identified in the last review, or that

21 could meet the current standard, we first consider the evidence from controlled human exposure

22 studies and epidemiologic panel studies. This evidence is assessed in section 6.2 of the ISA and

23 is summarized in section 3.1.2 above. Controlled human exposure studies have generally been

24 conducted with young, healthy adults, and have evaluated exposure durations less than 8 hours.

25 Epidemiologic panel studies have evaluated a wider range of study populations, including

26 children, and have generally evaluated associations with O_3 concentrations averaged over several

27 hours (U.S. EPA, 2013, section 6.2.1.2).[27]

28 As summarized above (section 3.1.2.1), and as discussed in detail in the ISA (U.S. EPA,

29 2013, section 6.2), a large number of controlled human exposure studies have reported lung

30 function decrements, respiratory symptoms, airway inflammation, airway hyperresponsiveness,

31 and/or impaired lung host defense in young, healthy adults engaged in moderate, intermittent

---

[27]In this section we focus on panel studies that used on-site monitoring, and that are highlighted in the ISA for the extent to which monitored ambient O_3 concentrations reflect exposure concentrations in their study populations (U.S. EPA, 2013, section 6.2.1.2).

1   exertion, following 6.6-hour O_3 exposures. These studies have consistently reported such effects

2   following exposures to O_3 concentrations of 80 ppb or greater. Available studies have also

3   evaluated some of these effects (i.e., lung function decrements, respiratory symptoms, airway

4   inflammation) following exposures to O_3 concentrations below 75 ppb. Table 3-1 highlights the

5   group mean results of individual controlled human exposure studies that have evaluated

6   exposures of healthy adults to O_3 concentrations below 75 ppb.

7   **Table 3-1.   Group mean results of controlled human exposure studies that have evaluated**
8   **exposures to ozone concentrations below 75 ppb in young, healthy adults.**

Endpoint	O_3 Exposure Concentration	Study	Statistically Significant O_3-Induced Effect[28]
FEV$_1$ decrements	70 ppb	Schelegle et al., 2009	yes
	60 ppb	Kim et al., 2011	yes
		Schelegle et al., 2009	no
		Adams, 2006	yes[29]
		Adams, 2002	no
	40 ppb	Adams, 2006	no
		Adams, 2002	no
Respiratory Symptoms	70 ppb	Schelegle et al., 2009	yes
	60 ppb	Kim et al., 2011	no
		Schelegle et al., 2009	no
		Adams, 2006	no[30]
	40 ppb	Adams, 2006	no
		Adams, 2002	no
Airway Inflammation (neutrophil influx)	60 ppb	Kim et al., 2011	yes

9

10   In further evaluating O_3-induced FEV$_1$ decrements following exposures to O_3

11   concentrations below 75 ppb, the ISA also combined the individual data from multiple studies of

12   healthy adults exposed for 6.6 hours to 60 ppb O_3 (Kim et al., 2011; Brown et al., 2008; Adams,

13   2006a, 2002, 1998). Based on these data, the ISA reports that 10% of exposed subjects

14   experienced FEV$_1$ decrements of 10% or more (i.e., abnormal and large enough to be potentially

---

[28]Based on study population means.

[29] In an analysis of the Adams (2006) data for square-wave chamber exposures, even after removal of potential outliers, Brown et al. (2008) reported the average effect on FEV1 at 60 ppb to be statistically significant (p < 0.002) using several common statistical tests (U.S. EPA, 2013, section 6.2.1.1) (section 3.1.2.1, above).

[30]Adams (2006) reported increased respiratory symptoms during a 6.6 hour exposure protocol with an average O_3 exposure concentration of 60 ppb. The increase in symptoms was reported to be statistically different from initial respiratory symptoms, though not statistically different from filtered air controls.

1 adverse for people with pulmonary disease, based on past CASAC advice (section 3.1.3,

2 above))[31] (U.S. EPA, 2013, section 6.2.1.1). Consistent with these findings, recently developed

3 empirical models predict that the onset of O_3-induced FEV_1 decrements in healthy adults occurs

4 following exposures to 60 ppb O_3 for 4 to 5 hours while at moderate, intermittent exertion

5 (Schelegle et al., 2012), and that 9% of healthy adults exposed to 60 ppb O_3 for 6.6 hours would

6 experience FEV_1 decrements greater than or equal to 10% (McDonnell et al., 2012) (U.S. EPA,

7 2013, section 6.2.1.1). When the evidence for O_3-induced lung function decrements was taken

8 together, the ISA concluded that (1) "mean FEV_1 is clearly decreased by 6.6-h exposures to 60

9 ppb O_3 and higher concentrations in subjects performing moderate exercise" (U.S. EPA, 2013, p.

10 6-9) and (2) although group mean decrements following exposures to 60 ppb O_3 are biologically

11 small, "a considerable fraction of exposed individuals experience clinically meaningful

12 decrements in lung function" (U.S. EPA, 2013, p. 6-20).

13   In considering the specific question above, we note that controlled human exposure

14 studies have reported decreased lung function, increased airway inflammation, and increased

15 respiratory symptoms in healthy adults following exposures to O_3 concentrations below 75 ppb.

16 Such impairments in respiratory function have the potential to be adverse, based on ATS

17 guidelines for adversity and based on previous advice from CASAC (section 3.1.3, above). In

18 addition, if they become serious enough, these respiratory effects could lead to the types of

19 clearly adverse effects commonly reported in O_3 epidemiologic studies (e.g., respiratory

20 emergency department visits, hospital admissions). Therefore, following exposures to O_3

21 concentrations lower than 75 ppb, controlled human exposure studies have reported respiratory

22 effects that could be adverse in some individuals, particularly if experienced by members of at-

23 risk populations (e.g., asthmatics, children).[32]

24   In further considering effects following exposures to O_3 concentrations below 75 ppb, we

25 also note that the ISA highlights some epidemiologic panel studies for the extent to which

26 monitored ambient O_3 concentrations reflect exposure concentrations in their study populations

27 (U.S. EPA, 2013, section 6.2.1.2). Specifically, Table 3-2 below includes O_3 panel studies that

28 have evaluated associations with lung function decrements for O_3 concentrations at or below 75

29 ppb, and that measured O_3 concentrations with monitors located in the areas where study

30 subjects were active (e.g., on site at summer camps or in locations where exercise took place)

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[31]CASAC has previously stated that "[a] 10% decrement in FEV1 can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV1) such that a ≥10% decrement could lead to moderate to severe respiratory symptoms" (Samet, 2011) (section 3.1.3, above).

[32]These effects were reported in healthy individuals. It is thus a reasonable inference that the effects would be greater in magnitude and potential severity for at-risk groups. See *National Environmental Development Ass'n Clean Air Project v. EPA*, 686 F. 3d 803, 811 (D.C. Cir. (2012) (making this point).

1　(U.S. EPA, 2013, section 6.2.1.2 and Table 6-6). Epidemiologic panel studies have evaluated a
2　wider range of populations and lifestages than controlled human exposure studies of O_3
3　concentrations below 75 ppb (e.g., including children).

4　**Table 3-2.** **Panel studies of lung function decrements with analyses restricted to O_3**
5　**concentrations below 75 ppb.**

Study	Population	O_3 Concentrations	Statistically Significant Association with Lung Function Decrements
Spektor et al. (1988)	Children at summer camp	Restricted to 1-hour concentrations below 60 ppb	Yes
Chan and Wu (2005)	Mail carriers	Maximum 8-hour average was 65 ppb	Yes
Korrick et al. (1998)	Adult hikers	2- to 12-hour average from 40 to 74 ppb during hikes	Yes
Brauer et al. (1996)	Farm workers	Restricted to 1-hour maximum below 40 ppb	Yes
		Restricted to 1-hour maximum below 30 ppb	No
Brunekreef et al. (1994)	Exercising adults	Restricted to 10-minute to 2.4-hour averages below 61 ppb	No
		Restricted to 10-minute to 2.4-hour averages below 51 ppb	No
		Restricted to 10-minute to 2.4-hour averages below 41 ppb	No

6　Although these studies report health effect associations for different averaging times, and it is not
7　clear the extent to which specific O_3 exposure conditions (i.e., concentrations, durations of
8　exposure, degrees of activity) were responsible for eliciting reported decrements, they are
9　consistent with the findings of the controlled human exposure studies discussed above.
10　Specifically, the epidemiologic panel studies in Table 3-2 indicate O_3-associated lung function
11　decrements when on-site monitored concentrations (ranging from minutes to hours) were below
12　75 ppb, with the evidence becoming less consistent at lower O_3 concentrations.

13　**3.1.4.2　Concentrations in Epidemiologic Studies – Short-term Metrics**

14　　　We next consider distributions of ambient O_3 concentrations in locations where
15　epidemiologic studies have evaluated O_3-associated hospital admissions, emergency department
16　visits, and/or mortality. When considering epidemiologic studies within the context of the current
17　standard, we emphasize those studies conducted in the U.S. and Canada. Such studies reflect air
18　quality and exposure patterns that are likely more typical of the U.S. population than the air
19　quality and exposure patterns reflected in studies conducted outside the U.S. and Canada (section

1   1.3.1.2, above).[33] We also emphasize studies reporting associations with effects judged in the

2   ISA to be robust to confounding by other factors, including co-occurring air pollutants. In

3   addition to these factors, we consider the statistical precision of study results, the extent to which

4   studies report associations in at-risk populations, and the extent to which the biological

5   plausibility of associations at various ambient O_3 concentrations is supported by controlled

6   human exposure and/or animal toxicological studies. These considerations help inform the range

7   of ambient O_3 concentrations over which we have the most confidence in O_3-associated health

8   effects, and the range of concentrations over which our confidence in such associations is

9   appreciably lower. We place particular emphasis on characterizing those portions of distributions

10   of ambient O_3 concentrations likely to meet the current standard.

11   In our consideration of these issues, we first address the following question:

12   •   **To what extent have U.S. and Canadian epidemiologic studies reported**
13   **associations with mortality or morbidity in locations that would have met the**
14   **current O_3 standard during the study period?**

15   Addressing this question can provide important insights into the extent to which O_3-health effect

16   associations are present for distributions of ambient O_3 concentrations that would be allowed by

17   the current standard. To the extent O_3 health effect associations are reported in study areas that

18   would have met the current standard, we have greater confidence that the current standard could

19   allow the clearly adverse O_3-associated effects indicated by those studies (e.g., mortality,

20   hospital admissions, emergency department visits).[34]

21   We note that epidemiologic studies evaluate statistical associations between variation in

22   the incidence of health outcomes and variation in ambient O_3 concentrations. In many of the O_3

23   epidemiologic studies assessed in the ISA, ambient concentrations are averaged across multiple

24   monitors within study areas, and in some cases over multiple days. These averages are used as

25   surrogates for the spatial and temporal patterns of O_3 exposures in study populations. In this

26   second draft PA, we refer to these averaged concentrations as "area-wide" O_3 concentrations.

27   We recognize that these epidemiologic studies do not identify the O_3 exposures that

28   population members have experienced, and do not identify the exposures that may be eliciting

29   the observed health outcomes. Thus, in considering epidemiologic studies of mortality and

30   morbidity, we are not drawing conclusions regarding single short-duration O_3 concentrations in

31   ambient air that, alone, are eliciting the reported health outcomes. Rather, our focus in this

32   section is to consider what these studies convey regarding the extent to which health effects may

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[33]Nonetheless, we recognize the importance of all studies, including international studies, in the ISA's assessment of the weight of the evidence that informs causality determinations.

[34]See *ATA III*, 283 F.3d at 370 (EPA justified in revising NAAQS when health effect associations are observed at levels allowed by the NAAQS).

1   be occurring (i.e., as indicated by associations) under air quality conditions meeting the current

2   standard.

3   In order to facilitate consideration of the question above, we have identified U.S. and

4   Canadian studies of respiratory hospital admissions, respiratory emergency department visits,[35]

5   and mortality (total, respiratory, cardiovascular) from the ISA (studies identified from U.S. EPA,

6   2013, Table 6-28, section 6.2.8, and Table 6-42) (Appendix 3-D). For each monitor in the areas

7   evaluated by these studies, we have identified the 3-year averages of the annual 4th highest daily

8   maximum 8-hour O_3 concentrations (Appendix 3-D). [36] To provide perspective on whether study

9   cities would have met or violated the current O_3 NAAQS during the study period, these O_3

10   concentrations were compared to the level of the current standard. Based on this approach, a

11   study city was judged to have met the current standard during the study period if all of the 3-year

12   averages of annual 4th highest 8-hour O_3 concentrations in that area were below 75 ppb.

13   Based on these analyses, the large majority of epidemiologic study areas evaluated would

14   have violated the current standard during study periods (Appendix 3-D). Table 3-3 below

15   highlights the subset of U.S. and Canadian studies reporting O_3 health effect associations in

16   locations that would have met the current standard during study periods. This includes a U.S.

17   single-city study that would have met the current standard over the entire study period (Mar and

18   Koenig, 2009) and four Canadian multicity studies for which the majority of study cities would

19   have met the current standard over the entire study periods (Cakmak et al., 2006; Dales et al.,

20   2006; Katsouyanni et al., 2009; Stieb et al., 2009).[37]

21

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[35]Given the inconsistency in results across cardiovascular morbidity studies (U.S. EPA, 2013, section 6.3.2.9), our consideration of the morbidity evidence in this section focuses on studies of respiratory hospital admissions and emergency department visits.

[36]These concentrations are referred to as "design values." A design value is a statistic that is calculated at individual monitors and based on 3 consecutive years of data collected from that site. In the case of O_3, the design value for a monitor is based on the 3-year average of the annual 4th highest daily maximum 8-hour O_3 concentration in parts per billion (ppb). For U.S. study areas, we used EPA's Air Quality System (AQS) (http://www.epa.gov/ttn/airs/airsaqs/) to identify design values. For Canadian study areas, we used publically available air quality data from the Environment Canada National Air Pollution Surveillance Network (http://www.etc-cte.ec.gc.ca/napsdata/main.aspx). We followed the data handling protocols for calculating design values as detailed in 40 CFR Part 50, Appendix P.

[37]In addition, a study by Vedal et al. (2003) was included in the 2006 CD (U.S. EPA, 2006). This study reported positive and statistically significant associations with mortality in Vancouver during a time period when the study area would have met the current standard (U.S. EPA, 2007). This study was not highlighted in the ISA in the current review (U.S. EPA, 2013).

Table 3-3. **U.S. and Canadian epidemiologic studies reporting O_3 health effect associations in locations that would have met the current standard during study periods.**

Authors	Study Results	Cities	Number of cities meeting the current standard over entire study period
Cakmak et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	10 Canadian cities	7
Dales et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	11 Canadian cities	7
Katsouyanni et al. (2009)	Positive and statistically significant associations with respiratory hospital admissions	12 Canadian cities	10
Katsouyanni et al. (2009)	Positive and statistically significant associations with all-cause and cardiovascular mortality[38]	12 Canadian cities	8
Mar and Koenig (2009)	Positive and statistically significant associations with asthma emergency department visits in children (< 18 years) and adults (> 18 years)	Seattle	1
Stieb et al. (2009)	Positive and statistically significant association with asthma emergency department visits	7 Canadian cities	5

As illustrated in Table 3-3, one U.S. single-city study highlighted in the ISA has reported health effect associations with asthma emergency department visits in a location that would have met the current standard over the entire study period. In addition, four Canadian multicity studies reported associations with mortality or morbidity when the majority of study locations would have met the current standard over the entire study periods. While there is uncertainty in ascribing the multicity effect estimates reported in these Canadian studies entirely to ambient concentrations that would have met the current standard (i.e., given that some study locations would have violated the current standard over at least part of the study period), the information in Table 3-3 suggests that reported multicity effect estimates are largely influenced by locations meeting the current standard. Together, these U.S. and Canadian epidemiologic studies suggest a relatively high degree of confidence in the presence of associations with mortality and morbidity for ambient O_3 concentrations meeting the current standard.

We next consider the extent to which additional epidemiologic studies of mortality or morbidity (i.e., those conducted in locations that violated the current standard) can also inform our consideration of adequacy of the current standard. In doing so, we note that health effect

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[38]Katsouyanni et al. (2009) report a positive and statistically significant association with cardiovascular mortality for people aged 75 years or older.

1   associations reported in epidemiologic studies are influenced by the full distributions of ambient
2   O_3 concentrations, including concentrations below the level of the current standard. We focus on
3   studies that have explicitly characterized such O_3 health effect associations, including confidence
4   in those associations, for various portions of distributions of ambient O_3 concentrations. In doing
5   so, we consider the following question:

6   - **To what extent do analyses from epidemiologic studies indicate confidence in health**
7   **effect associations over distributions of ambient O_3 concentrations, including at**
8   **concentrations lower than previously identified or below the current standard?**

9   We first focus on those studies that have reported confidence intervals around
10 concentration-response functions over distributions of ambient O_3 concentrations. Confidence
11 intervals around concentration-response functions can provide insights into the range of ambient
12 concentrations over which the study indicates the most confidence in the reported health effect
13 associations (i.e., where confidence intervals are narrowest), and into the range of ambient
14 concentrations below which the study indicates that uncertainty in the nature of such associations
15 becomes notably greater (i.e., where confidence intervals become markedly wider). The
16 concentrations below which confidence intervals become markedly wider in such analyses are
17 intrinsically related to data density, and do not necessarily indicate the absence of an association.
18 The ISA identifies several epidemiologic studies that have reported confidence intervals
19 around concentration-response functions in U.S. cities. The ISA concludes that studies generally
20 indicate a linear concentration-response relationship "across the range of 8-h max and 24 h avg
21 O3 concentrations most commonly observed in the U.S. during the O3 season" and that "there is
22 less certainty in the shape of the C-R curve at the lower end of the distribution of O_3
23 concentrations" (U.S. EPA, 2013, pp. 2-32 to 2-34). In characterizing the O_3 concentrations
24 below which such certainty decreases, the ISA discusses area-wide O_3 concentrations as low as
25 20 ppb and as high as 40 ppb (U.S. EPA, 2013, section 2.5.4.4).
26 Consistent with these conclusions, the range of ambient concentrations over which the
27 evidence indicates the most certainty in concentration-response relationships can vary across
28 studies. Such variation is likely due at least in part to differences in the O_3 metrics evaluated and
29 differences in the distributions of ambient concentrations and health events. Thus, although
30 consideration of confidence intervals around concentration-response functions can provide
31 valuable insights into the ranges of ambient concentrations over which studies indicate the most
32 confidence in reported health effect associations, there are limitations in the extent to which
33 these analyses can be generalized across O_3 metrics, study locations, study populations, and
34 health endpoints.

1   The ISA emphasizes two U.S. single-city studies that have reported confidence intervals
2   around concentration-response functions (Silverman and Ito, 2010; Strickland et al., 2010).
3   These studies, and their associated O_3 air quality, are discussed below.
4   Silverman and Ito (2010) evaluated associations between 2-day rolling average O_3
5   concentrations[39] and asthma hospital admissions in New York City from 1999 to 2006 (a time
6   period when the study area would have violated the current standard, Appendix 3-D). As part of
7   their analysis, the authors evaluated the shape of the concentration-response relationship for O_3
8   using a co-pollutant model that included $PM_{2.5}$ (reprinted in Figure 3-4, below). Based on their
9   analyses, Silverman and Ito (2010) concluded a linear relationship between O_3 and hospital
10 admissions is a reasonable approximation of the concentration-response function throughout
11 much of the range of ambient O_3 concentrations. Based on visual inspection of Figure 3-4 below
12 (Figure 3 from published study), we note that confidence in the reported concentration-response
13 relationship is highest for area-wide average O_3 concentrations around 40 ppb (i.e., near the
14 reported median of 41 ppb), and decreases notably for concentrations at and below about 20 ppb.
15

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[39] 2-day rolling averages of 8-hour daily maximum O_3 concentrations were calculated throughout the study period, averaged across study monitors.
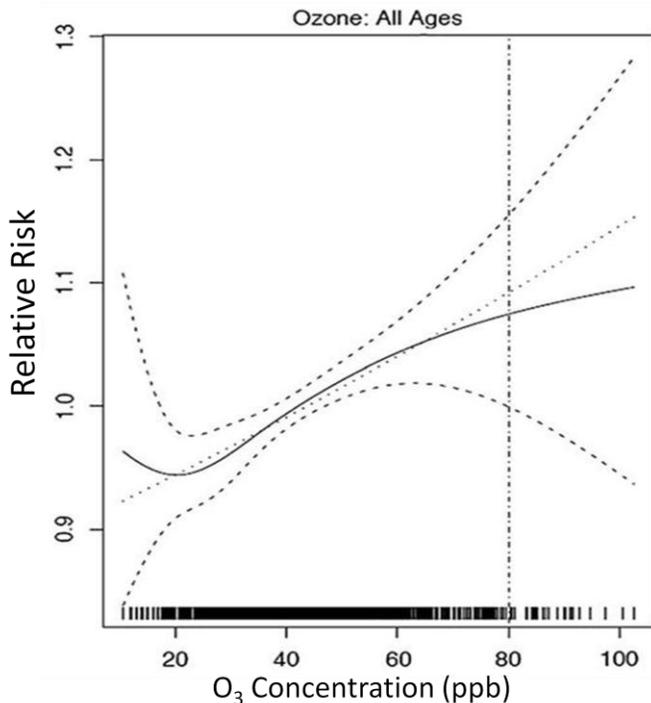
Figure 3-4. **Concentration-response function for asthma hospital admissions over the distribution of area-wide averaged O$_3$ concentrations (adapted from Silverman and Ito, 2010).[40]**

In considering the concentration-response function presented by Silverman and Ito (2010) within the context of the adequacy of the current standard, we evaluate the extent to which the O$_3$ concentrations contributing to various portions of the function would likely have been allowed by the current standard. In doing so, we recognize that true design values cannot be identified for the subsets of air quality data contributing to various portions of the concentration-response function.[41] Therefore, to use this analysis to inform our consideration of the adequacy of the current standard we evaluate the extent to which the concentration-response function indicates a relatively high degree of confidence in the reported health effect association on days when all monitored 8-hour O$_3$ concentrations were below 75 ppb (Table 3-4, below). This approach can provide insight into the extent to which the reported O$_3$ health effect association is present when all monitored O$_3$ concentrations are below the level of the current standard.

Based on the information in Table 3-4 below, when 2-day averaged O$_3$ concentrations ranged from 36 to 45 ppb (i.e., around the median, where confidence intervals are narrowest), there was 1 day (out of 236) with at least one monitor recording an 8-hour daily maximum O$_3$

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[40]This figure was also reprinted in the ISA (U.S. EPA, 2013; Figure 6-16).

[41]As discussed above, O$_3$ design values are calculated using all data available from a monitor.

1 concentration above the level of the current standard (approximately 0.4% of days). When 2-day

2 averaged O_3 concentrations ranged from 26 to 45 ppb (i.e., extending to concentrations below the

3 median, but still above the concentrations where confidence intervals widen notably), there were

4 4 days (out of 816) with at least one monitor recording an 8-hour daily maximum O_3

5 concentration above the level of the current standard (approximately 0.5% of days). Thus, on

6 over 99% of the days when area-wide "averaged" O_3 concentrations were between 26 and 45

7 ppb, the highest 8-hour daily maximum O_3 concentrations were below 75 ppb. For comparison,

8 the annual 4th highest 8-hour daily maximum O_3 concentration generally corresponds to the 98th

9 or 99th percentile of the seasonal distribution, depending on the length of the O_3 season.

10 **Table 3-4.** **Distributions of daily 8-hour maximum ozone concentrations from highest**
11 **monitors over range of 2-day moving averages from composite monitors (for**
12 **study area evaluated by Silverman and Ito, 2010)**

8-hr max from highest monitor during those 2 days	2-day moving average across monitors (ppb)								
	11 to 20 (62 days)	21 to 25 (92 days)	26 to 30 (178 days)	31 to 35 (206 days)	36 to 40 (236 days)	41 to 45 (196 days)	46 to 50 (153 days)	51 to 55 (111 days)	56 to 60 (71 days)
Min	16	27	30	36	41	45	52	58	62
5th	20	28	34	39	44	49	56	61	67
25th	26	31	37	43	47	53	61	67	72
50th	29	35	42	46	51	59	64	71	76
75th	35	38	46	50	55	64	69	78	85
95th	39	50	54	60	63	74	80	85	96
98th	41	54	60	70	68	82	85	93	99
99th	42	55	67	72	71	87	87	94	103
Max	42	59	80	75	79	100	97	96	108
Days > 75 ppb	0	0	1	0	1	2	9	15	20

14 In a separate study, Strickland et al. (2010) evaluated associations between 3-day rolling

15 average O_3 concentrations[42] and asthma hospital admissions in Atlanta during the warm season

16 from 1994 to 2004 (a time period when the study area would have violated the current standard,

17 Appendix 3-D). As part of this analysis, Strickland et al. (2010) evaluated the concentration-

18 response relationship for O_3 and pediatric asthma emergency department visits. The authors

19 reported the shape of the concentration-response function to be approximately linear with no

20 evidence of a threshold when 3-day averaged 8-hour daily maximum O_3 concentrations were

21 approximately 30 to 80 ppb (Figure 3-5 below and U.S. EPA, 2013, Figure 6-18). Figure 3-5

22 below illustrates that the confidence intervals around the concentration-response function are

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[42] Three-day rolling averages of population-weighted 8-hour daily maximum O_3 concentrations were calculated throughout the study period (Strickland et al., 2010).

1 narrowest around the study mean (i.e., 55 ppb), and that these confidence intervals do not widen
2 notably for "averaged" O_3 concentrations as low as about 30 ppb.

3
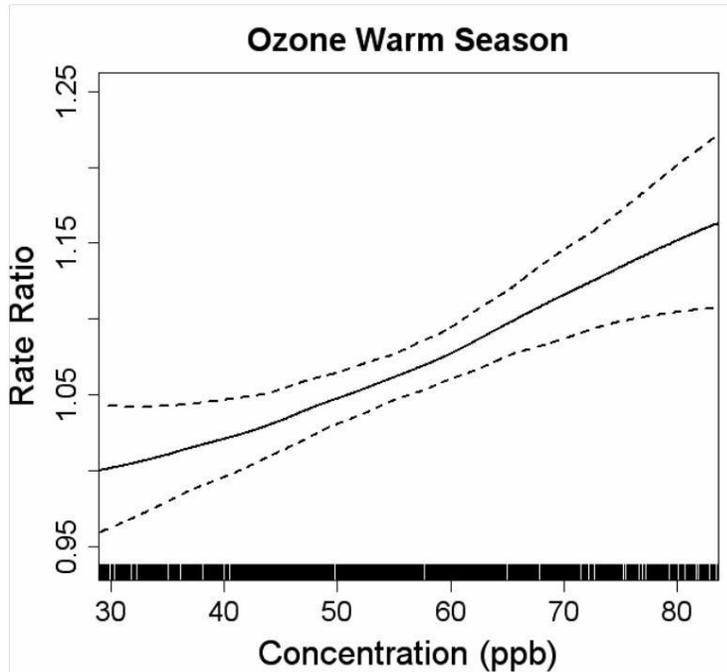
Figure 3-5. Concentration-response function for pediatric asthma emergency department visits over the distribution of averaged, population-weighted 8-hour O_3 concentrations (reprinted from Strickland et al., 2010).[43]

7 Similar to the study by Silverman and Ito (2010), we consider the extent to which the
8 reported concentration-response function indicates a relatively high degree of confidence in
9 health effect associations on days when all monitored 8-hour O_3 concentrations are below 75 ppb
10 (Tabe 3-5, below).[44] In considering the information presented in Table 3-5, we first note that
11 when 3-day averaged O_3 concentrations were in the range of the mean (i.e., 51 to 60 ppb), there
12 were 77 days (out of 516; 14.9%) with at least one monitor recording an 8-hour daily maximum
13 O_3 concentration above the level of the current standard. In contrast, during the 519 days when
14 averaged O_3 concentrations were in the lower portion of the distribution where study authors
15 indicate relatively high confidence in the reported concentration-response relationship (i.e.,
16 between 31 and 45 ppb), there were 4 days with at least one monitor in the study area measuring
17 an 8-hour daily maximum O_3 concentration greater than 75 ppb (approximately 0.8% of days).

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[43] This figure was also reprinted in the ISA (U.S. EPA, 2013; Figure 6-18).

[44] The study by Strickland et al. (2010) used five monitors. For our evaluation of highest 8-hour daily maximum concentrations (i.e., from the individual monitor recording the highest such concentration), we obtained information from the four of these study area monitors that report data to AQS (Appendix 3-D).

1 Thus, on over 99% of the days when "averaged" O_3 concentrations were between 31 and 45 ppb,

2 all monitors measured 8-hour daily maximum O_3 concentrations below 75 ppb.

3 **Table 3-5.   Distribution of daily 8-hour maximum ozone concentrations from highest**
4 **monitors over range of 3-day moving averages of population-weighted**
5 **concentrations (for study area evaluated by Strickland et al., 2010)**

8-hr max from highest monitor during	3-day moving average across monitors (ppb)										
	26-30 (75 days)	31-35 (144 days)	36-40 (165 days)	41-45 (210 days)	46-50 (235 days)	51-55 (244 days)	56-60 (272 days)	61-65 (234 days)	66-70 (169 days)	71 to 75 (124 days)	76 to 80 (106 days)
Min	27	24	33	30	36	38	49	52	57	67	69
5th	29	33	36	45	48	54	60	64	69	75	85
25th	36	40	45	51	56	63	68	73	79	85	95
50th	38	44	50	58	62	68	73	78	86	92	101
75th	44	49	54	62	70	74	81	85	96	101	108
95th	53	56	70	74	81	84	95	95	104	109	120
98th	59	59	75	84	86	93	100	98	107	110	124
99th	66	61	78	89	88	95	103	102	108	112	124
Max	70	64	83	99	97	107	106	122	129	120	138
Days > 75	0	0	2	2	10	24	53	80	89	87	87

6

7 In summary, analyses of air quality data from the study locations evaluated by Silverman

8 and Ito (2010) and Strickland et al. (2010) indicate a relatively high degree of confidence in

9 reported statistical associations with respiratory health outcomes on days when all monitors

10 recorded 8-hour average O_3 concentrations of 75 ppb or below. Though these analyses do not

11 identify true design values, the presence of O_3-associated respiratory effects on such days

12 provides insight into the types of health effects that could occur in locations that meet the current

13 standard.

14 There are several important uncertainties that are specifically related to our analyses of

15 distributions of O_3 air quality in the study locations evaluated by Silverman and Ito (2010) and

16 Strickland et al. (2010). Although these studies report health effect associations with two-day

17 (Silverman and Ito) and three-day (Strickland) averages of daily O_3 concentrations, it is possible

18 that the respiratory morbidity effects reported in these studies were also at least partly

19 attributable to the days immediately preceding these two- and three-day periods. In support of

20 this possibility, Strickland et al. reported positive and statistically significant associations with

21 emergency department visits for multiple lag periods, including lag periods exceeding three days.

22 Our analysis of highest monitored concentrations focuses on two- and three- day periods, as used

23 in the published study to generate concentration-response functions. This could have important

24 implications for our interpretation of the reported concentration-response functions if a 2-day

25 period with no monitors measuring 8-hour concentrations at or above 75 ppb is immediately

26 preceded by one or more days with monitors that do exceed 75 ppb. Although we do not know

27 the extent to which O_3 concentrations on a larger number of days could have contributed to

1   reported health effect associations, we note this as a potentially important uncertainty in our

2   consideration of concentration-response functions within the context of the current standard.

3   In addition, an important uncertainty that applies to epidemiologic studies in general is

4   the extent to which reported health effects are caused by exposures to O_3 itself, as opposed to

5   other factors such as co-occurring pollutants or other pollutant mixtures. Although both of the

6   studies evaluated above reported health effect associations in co-pollutant models, this

7   uncertainty becomes an increasingly important consideration as health effect associations are

8   evaluated at lower ambient O_3 concentrations (i.e., resulting in lower exposure concentrations).

9   One approach to considering the potential importance of this uncertainty in

10   epidemiologic studies is to evaluate the extent to which there is coherence with the results of

11   experimental studies (i.e., in which the study design dictates that exposures to O_3 itself are

12   responsible for reported effects). Therefore, in further considering uncertainties associated with

13   the above air quality analyses for the study areas evaluated by Silverman and Ito (2010) and

14   Strickland et al. (2010), we evaluate the following question:

15   •   **To what extent is there coherence between evidence from controlled human**
16   **exposure studies and epidemiologic studies supporting the occurrence of O_3-**
17   **attributable respiratory effects when 8-hour daily maximum ambient O_3**
18   **concentrations are at or below 75 ppb?**

19   As summarized above and as discussed in the ISA (U.S. EPA, 2013, section 6.2),

20   controlled human exposure studies demonstrate the occurrence of respiratory effects in an

21   appreciable percentage of healthy adults following single short-term exposures to O_3

22   concentrations as low as 60 ppb. In addition, as O_3 exposure concentrations exceed 60 ppb: 1)

23   effects in healthy adults become larger and more serious; 2) a broader range of effects are

24   observed in a greater percentage of exposed individuals; and 3) effects are reported more

25   consistently across studies. Thus, as the potential increases for exposures to O_3 concentrations

26   approaching or exceeding 60 ppb, our confidence increases that reported respiratory health

27   effects could be caused by exposures to the ambient O_3 concentrations present in study locations.

28   In considering coherence with results of controlled human exposure studies in this way, it

29   is important to note the relative lack of experimental data for exposure concentrations below 60

30   ppb. It is possible that lower exposure concentrations can result in respiratory effects serious

31   enough to lead to hospital admissions or emergency department visits, particularly in at-risk

32   populations such as children and asthmatics. Thus, although we consider coherence between

33   epidemiologic and controlled human exposure study results, we also acknowledge that an O_3

34   exposure concentration of 60 ppb does not constitute a bright line below which we are confident

35   that effects no longer occur, particularly for at-risk groups.

1   As discussed above, for the study by Silverman and Ito (2010), 26 to 45 ppb represents

2 the lower end of the range of "averaged" daily maximum 8-hour concentrations over which the

3 study indicates a relatively high degree of confidence in the statistical association with

4 respiratory hospital admissions (and for which virtually all monitored concentrations were 75

5 ppb or below). As averaged concentrations increase from 26 to 45 ppb, the number of days with

6 maximum monitored concentrations exceeding 60 ppb increases dramatically (Table 3-4,

7 above).[45] For example, of the 178 days with area-wide average daily maximum 8-hour

8 concentrations from 26 to 30 ppb, only about 2% had any monitors recording ambient

9 concentrations of 60 ppb or greater. In contrast, of the 196 days with area-wide average

10 concentrations from 41 to 45 ppb, about half had at least one monitor recording an ambient

11 concentration near or above 60 ppb, with monitors on some days measuring concentrations

12 greater than 80 ppb. Thus, as averaged concentrations approach 45 ppb there is an increasing

13 likelihood that at least some portion of the study population could have been exposed to O_3

14 concentrations approaching or exceeding those reported in controlled human exposure studies to

15 cause respiratory effects in healthy adults.

16   For the study by Strickland et al (2010), "averaged" concentrations from 30 to 45 ppb

17 represent the lower end of the range of concentrations over which the study indicates a relatively

18 high degree of confidence in the statistical association with respiratory emergency department

19 visits (and for which virtually all monitored concentrations were 75 ppb or below). Similar to the

20 study area evaluated by Silverman and Ito, as 8-hour area-wide average O_3 concentrations

21 approach 45 ppb, maximum monitored concentrations exceed 60 ppb more frequently. On most

22 days contributing to averaged O_3 concentrations from 41 to 45 ppb, maximum monitor

23 concentrations were near or above 60 ppb. On a small number of these days, maximum

24 monitored concentrations were greater than 80 ppb. Therefore, similar to the study by Silverman

25 and Ito (2010), at least some portion of the study population on these days are likely to have been

26 exposed to O_3 concentrations exceeding those reported in controlled human exposure studies to

27 cause respiratory effects in some healthy adults.

28   Thus, consideration of distributions of individual monitored concentrations, in

29 conjunction with the results of O_3 controlled human exposure studies, supports the role of

30 ambient O_3 concentrations in eliciting the reported respiratory hospital admissions and

31 emergency department visits. Specifically, these analyses support the occurrence of O_3-

32 attributable hospital admissions and emergency department visits on days when virtually all

33 monitors measure 8-hour ambient concentrations below 75 ppb.

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[45]Though, as noted above, the epidemiologic studies by Silverman and Ito (2010) and Strickland et al. (2010) do not provide information on the extent to which reported health effects result from exposures to any specific O_3 concentrations.

1   In further evaluating O_3 concentration-response relationships within the context of the

2 adequacy of the current standard, we note that some epidemiologic studies report health effect

3 associations for air quality distributions restricted to ambient pollutant concentrations below one

4 or more predetermined cut points. Such "cut point" analyses can provide information on the

5 magnitude and statistical precision of effect estimates for defined distributions of ambient

6 concentrations, which may in some cases include distributions that would meet the current

7 standard. Therefore, we next consider the following question:

8   • **To what extent do cut-point analyses from epidemiologic studies report health effect**
9   **associations at ambient O_3 concentrations lower than previously identified or that**
10   **would likely meet the current standard?**

11   By considering the magnitude and statistical significance of effect estimates for restricted

12 air quality distributions, cut-point analyses can provide insight into the extent to which health

13 effect associations are driven by ambient concentrations above the cut point, versus

14 concentrations below the cut point. For studies that evaluate multiple cut points, these analyses

15 can provide insights into the magnitude and statistical precision of health effect associations for

16 different portions of the distribution of ambient concentrations, including insights into the

17 ambient concentrations below which uncertainty in reported associations becomes notably

18 greater. As with analyses of concentration-response functions, discussed above, the cut points

19 below which confidence intervals become notably wider depend in large part on data density.[46]

20   In the U.S. multicity study by Bell et al. (2006), study authors used the NMMAPS data

21 set to evaluate associations between 2-day rolling average O_3 concentrations[47] and total (non-

22 accidental) mortality in 98 U.S. cities from 1987 to 2000. Based on the full distributions of

23 ambient O_3 concentrations in study cities, the large majority of the NMMAPS cities would have

24 violated the current standard during the study period (Appendix 3-D). However, Bell et al.

25 (2006) also reported health effect associations in a series of cut-point analyses, with effect

26 estimates based only on the subsets of days contributing to "averaged" O_3 concentrations below

27 cut points ranging from 5 to 60 ppb (see Figure 2 in Bell et al., 2006). The lowest cut-point for

28 which the association between O_3 and mortality was reported to be statistically significant was

29 30 ppb (based on visual inspection of Figure 2 in published study). As with the studies by

30 Silverman and Ito (2010) and Strickland et al. (2010), we consider what these cut point analyses

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[46]As such, these analyses provide insight into the ambient concentrations below which the available air quality information becomes too sparse to support conclusions about the nature of concentration-response relationships, with a high degree of confidence.

[47]Two-day rolling averages of 24-hour average O_3 concentrations were calculated throughout the study period. This calculation was done across study monitors in study cities with multiple monitors.

1 indicate with regard to the potential for health effect associations to extend to ambient O_3

2 concentrations likely to be allowed by the current O_3 NAAQS.

3   We attempted to recreate the subsets of air quality data used in the cut point analyses

4 presented by Bell et al. (2006). In doing so, we applied the criteria described in the published

5 study to generate air quality subsets corresponding to those defined by the cut points evaluated

6 by study authors.[48] From the days with averaged O_3 concentrations below each cut point, we

7 identified 3-year averages of annual 4th highest 8-hour daily maximum O_3 concentrations in each

8 study area. We then compared these 4th highest O_3 concentrations to the level of the current

9 standard in order to provide insight into the extent to which the air quality distributions included

10 in various cut point analyses would likely have met the current standard.

11   We particularly focus on the lowest cut-point for which the association between O_3 and

12 mortality was reported to be statistically significant (i.e., 30 ppb, as noted above). Based on the

13 O_3 air quality concentrations that met the criteria for inclusion in the 30 ppb cut point analysis,

14 95% of study areas had 3-year averages of annual 4th highest 8-hour daily maximum O_3

15 concentration at or below 75 ppb over the entire study period. For the 35 ppb cut point, which

16 also resulted in a statistically significant association with mortality, 68% of study areas had 3-

17 year averages of annual 4th highest 8-hour daily maximum O_3 concentration at or below 75 ppb.

18 This suggests that the large majority of air quality distributions that provided the basis for

19 positive and statistically significant associations with mortality (i.e., for the 30 and 35 ppb cut

20 points) would likely have met the current O_3 standard. For higher cut points, all of which also

21 resulted in statistically significant associations with mortality, the majority of study cities had 3-

22 year averages of annual 4th highest 8-hour daily maximum concentrations greater than 75 ppb.

23

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[48]We were unable to obtain the air quality data used to generate the cut-point analyses in the study published by Bell et al. (2006). Therefore, we generated 2-day averages of 24-hour O_3 concentrations in study locations using the air quality data available in AQS, combined with the published description of study area definitions. In doing so, we did not recreate the trimmed means used by Bell. As discussed below, this represents an important uncertainty in our analysis.

Table 3-6. **Number of study cities with 4th highest 8-hour daily maximum concentrations greater than 75 ppb, for various cut-point analyses presented in Bell et al. (2006)**

	Cut-point for 2-day moving average across monitors and cities (24-hour average)								
	25	30	35	40	45	50	55	60	All
Number (%) of Cities with 4th highest >75 (any 3-yr period; 1987-2000)	0 (0%)	5 (5%)	31 (32%)	70 (71%)	86 (88%)	88 (90%)	92 (94%)	92 (94%)	92 (94%)

In addition to the uncertainties noted above for our analysis of the single-city studies by Silverman and Ito (2010) and Strickland et al. (2010) (e.g., attributing effects specifically to air quality included in various subsets), an important uncertainty related to this analysis is that we were unable to obtain the air quality data used to generate the cut-point analyses in the study published by Bell et al. (2006). Therefore, as noted above, we generated 2-day averages of 24-hour O_3 concentrations in study locations using the air quality data available in AQS, combined with the published description of study area definitions. In doing so, we did not recreate the trimmed means used by Bell. An important uncertainty in this approach is the extent to which we were able to appropriately recreate the cut-point analyses in the published study.

The ISA also notes important uncertainties inherent in multicity studies that evaluate the potential for thresholds to exist, as was done in the study by Bell et al. (2006). Specifically, the ISA highlights the regional heterogeneity in O_3 health effect associations as a factor that could obscure the presence of thresholds, should they exist, in multicity studies (U.S. EPA, 2013, sections 2.5.4.4 and 2.5.4.5). The ISA notes that community characteristics (e.g., activity patterns, housing type, age distribution, prevalence of air conditioning) could be important contributors to reported regional heterogeneity (U.S. EPA, 2013, section 2.5.4.5). Given this heterogeneity, the ISA concludes that "a national or combined analysis may not be appropriate to identify whether a threshold exists in the O_3-mortality C-R relationship" (U.S. EPA, 2013, p. 2-33). This represents an important source of uncertainty when characterizing our confidence in reported concentration-response relationships over distributions of ambient O_3 concentrations, based on multicity studies. This uncertainty becomes increasingly important when interpreting concentration-response relationships at lower ambient O_3 concentrations, particularly those concentrations corresponding to portions of distributions where data density decreases notably.

3.1.4.3 Concentrations in Epidemiologic Studies – "Long-term" Metrics

We next consider the extent to which epidemiologic studies employing longer-term ambient O_3 concentration metrics inform our understanding of the air quality conditions

1 associated with O_3-attributable health effects, and specifically inform consideration of the extent
2 to which such effects could occur under air quality conditions meeting the current standard.
3 Unlike for the studies of short-term O_3 discussed above, the available U.S. and Canadian
4 epidemiologic studies evaluating long-term ambient O_3 concentration metrics have not been
5 conducted in locations likely to have met the current 8-hour O_3 standard during the study period
6 (Appendix 3-D). Therefore, although these studies contribute to our understanding of health
7 effects associated with long-term or repeated exposures to ambient O_3 (as summarized in section
8 3.1.2 above), consideration of study area design values does not inform our consideration of the
9 extent to which those health effects may be occurring in locations that met the current standard.
10 In further considering epidemiologic studies of long-term O_3 concentrations, we also
11 evaluate the extent to which concentration-response functions have been reported over
12 distributions of ambient concentrations, and what those functions can tell us about health effect
13 associations with O_3 concentrations likely to be allowed by the current standard. Specifically, we
14 consider the following question:

15 • **To what extent do confidence intervals around concentration-response functions**
16 **indicate O_3-associated health outcomes at ambient concentrations meeting the**
17 **current O_3 standard?**

18 The ISA identifies a single epidemiologic study reporting confidence intervals around a
19 concentration-response function for "long-term" O_3 concentrations and respiratory mortality
20 (Jerrett et al., 2009; U.S. EPA, 2013, sections 7.2.7, 7.2.8 and 7.7). Jerrett et al. (2009) reported
21 that when seasonal averages of 1-hour daily maximum O_3 concentrations[49] ranged from 33 to
22 104 ppb, there was no statistical deviation from a linear concentration-response relationship
23 between O_3 and respiratory mortality across 96 U.S. cities (U.S. EPA, 2013, section 7.7).
24 However, the authors reported "limited evidence" for an effect threshold at an O_3 concentration
25 of 56 ppb (p=0.06). Visual inspection of this concentration-response function (Figure 3-6)
26 confirms the possibility of an inflection point just below 60 ppb, which is close to the median
27 concentration across cities (i.e., 57 ppb).

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[49]Jerrett et al. (2009) evaluated the April to September averages of 1-hour daily maximum O_3 concentrations across 96 U.S. metropolitan areas from 1977- 2000. In urban areas with multiple monitors, April to September 1-hour daily maximum concentrations from each individual monitor were averaged. This step was repeated for each year in the study period. Finally, each yearly averaged O_3 concentrations was then averaged again to yield the single averaged 1-hour daily maximum O_3 concentration depicted on the x axis of Figure 3-6 below.
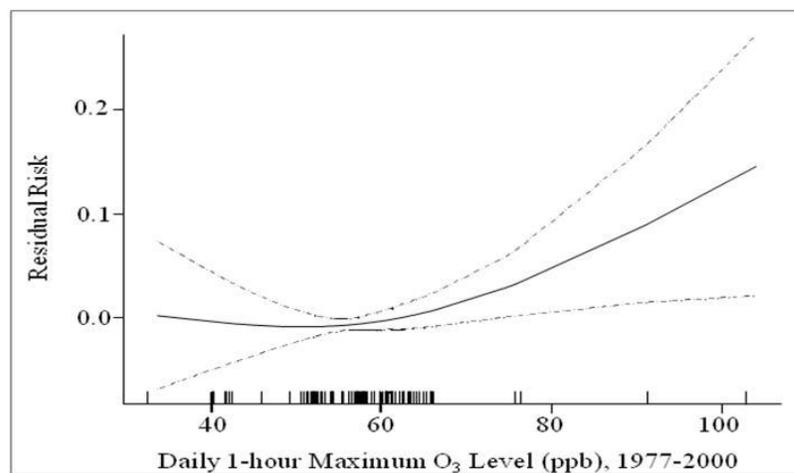
Figure 3-6. Exposure-Response relationship between risk of death from respiratory causes and ambient O_3 concentration study metric (Jerrett et al., 2009).

We consider the extent to which this concentration-response function indicates confidence in the reported health effect association at various ambient O_3 concentrations. In doing so, we note the following: (1) most of the study cities had O_3 concentrations above 53.1 ppb (i.e., the upper bound of the first quartile), accounting for approximately 72% of the respiratory deaths in the cohort (Table 2 in Jerrett et al. 2009); (2) confidence intervals widen notably for O_3 concentrations in the first quartile (based on visual inspection of Figure 3-6); and (3) study authors noted limited evidence for a threshold at 56 ppb.[50] In considering this information, we conclude that the analysis reported by Jerrett indicates the greatest confidence in the linear concentration-response function for "long-term" O_3 concentrations in the upper three quartiles (i.e., above about 53 ppb).

Based on information in the published study (Figure 1 in Jerrett et al., 2009), we identified 72 of the 96 study cities as having ambient O_3 concentrations in the highest three quartiles (Appendix 3-D). As noted above, these 72 cities account for approximately 72% of the respiratory deaths in the cohort (Table 2 in Jerrett et al. 2009). Of these 72 cities, 71 had 3-year averages of annual 4th highest 8-hour daily maximum O_3 concentrations above 75 ppb (Appendix 3-D). Thus, the current 8-hour NAAQS would have been violated during the study period in virtually all of the study cities that contribute to the range of long-term O_3 concentrations over which we have the greatest confidence in the reported relationship with respiratory mortality. Thus, while the study by Jerrett et al. (2009) contributes to our understanding of health effects associated with ambient O_3 (as summarized in section 3.1.2 above), it is less informative

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[50]The ISA does not reach conclusions regarding the potential for a threshold in the association between "long-term" O_3 concentrations and respiratory mortality.

1 regarding the extent to which those health effects may be occurring under air quality conditions
2 allowed by the current standard.

3.1.5 Public Health Implications

4 In this section, we address the public health implications of O_3 exposures with respect to
5 the factors that put populations at increased risk from exposures (section 3.1.5.1), the size of at-
6 risk populations (section 3.1.5.2), and the potential effects of averting behavior on reducing O_3
7 exposures and associated health effects (section 3.1.5.3). Providing appropriate public health
8 protection requires consideration of the factors that put populations at greater risk from O_3
9 exposure. In order to estimate potential overall for public health impacts, it is important to
10 consider not only the adversity of the health effects, but also the populations at greater risk and
11 potential behaviors that may reduce exposure.

3.1.5.1 At-Risk Populations

13 In this section we address the following question:

14 • **To what extent does the currently available scientific evidence expand our**
15 **understanding of at-risk populations?**

16 The currently available evidence expands our understanding of populations that were
17 identified to be at greater risk of O_3-related health effects at the time of the last review (i.e.,
18 people who are active outdoors, people with lung disease, children and older adults and people
19 with increased responsiveness to O_3) and supports the identification of additional factors that
20 may lead to increased risk (U.S. EPA 2006, section 3.6.2; U.S. EPA, 2013, chapter 8).
21 Populations and lifestages may be at greater risk for O_3-related health effects due to factors
22 contribute to their susceptibility and/or vulnerability to ozone. The definitions of susceptibility
23 and vulnerability have been found to vary across studies, but in most instances "susceptibility"
24 refers to biological or intrinsic factors (e.g., lifestage, sex, preexisting disease/conditions) while
25 "vulnerability" refers to non-biological or extrinsic factors (e.g., socioeconomic status [SES])
26 (U.S. EPA, 2013, p. 8-1; U.S. EPA, 2010c, 2009d). In some cases, the terms "at-risk" and
27 "sensitive" have been used to encompass these concepts more generally. In the ISA and this PA,
28 "at-risk" is the all-encompassing term used for groups with specific factors that increase their
29 risk of O_3-related health effects. Further discussion of at-risk populations can be found in
30 Appendix 3C below.

31 There are multiple avenues by which groups may experience increased risk for O_3-
32 induced health effects. A population or lifestage[51] may exhibit greater effects than other

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[51] Lifestages, which in this case includes childhood and older adulthood, are experienced by most people over the
course of a lifetime, unlike other factors associated with at-risk populations.

1   populations or lifestages exposed to the same concentration or dose, or they may be at greater
2   risk due to increased exposure to an air pollutant (e.g., time spent outdoors). A group with
3   intrinsically increased risk would have some factor(s) that increases risk through a biological
4   mechanism and, in general, would have a steeper concentration-risk relationship, compared to
5   those not in the group. Factors that are often considered intrinsic include asthma, genetic
6   background, and lifestage. A group of people could also have extrinsically increased risk, which
7   would be through an external, non-biological factor, including, for example, socioeconomic
8   status (SES) and diet. Some groups are at risk of increased internal dose at a given exposure
9   concentration, for example, because of breathing patterns. This category would include people
10 who work or exercise outdoors. Finally, there are those who might be placed at increased risk for
11 experiencing greater exposures by being exposed at higher concentrations. This would include,
12 for example, groups of people with greater exposure to ambient O_3 due to less availability or use
13 of home air conditioners such that they are more likely to be in locations with open windows on
14 high ozone days. Some groups may be at increased risk of O_3-related health effects through a
15 combination of factors. For example, children tend to spend more time outdoors when O_3 levels
16 are high, and at higher levels of activity than adults, which leads to increased exposure and dose,
17 and they also have biological, or intrinsic, risk factors (e.g., their lungs are still developing)
18 (U.S. EPA, 2013, Chapter 8). An at-risk population or lifestage is more likely to experience
19 adverse health effects related to O_3 exposures and/or, develop more severe effects from exposure
20 than the general population.

21   Based on the currently available evidence, the at-risk populations for O_3- related health
22 effects are based on factors that include: asthma, lifestages (children and older adults), genetic
23 variability, dietary factors, and working outdoors (U.S. EPA, 2013, section 8.5, Table 8-6). This
24 conclusion is supported by consistency in findings across studies and evidence of coherence in
25 results from different scientific disciplines. The current evidence is suggestive of a potential for
26 three other factors to influence risk of O_3-related health effects. The evidence suggests that
27 women may be at greater risk than men, groups with low SES or living in neighborhoods with
28 low SES may be at greater risk than other socioeconomic groups, and obesity may be a potential
29 risk factor. Further studies are needed, however, on these factors. Overall, the factors most
30 strongly supported as contributing to increased risk of O_3-related effects are related to asthma,
31 lifestage (children and older adults), genetic variability, dietary factors, and working outdoors
32 (U.S. EPA, 2013; chapter 8).

33   In summary, the evidence available in this review supports the identification of the
34 following populations and lifestages as having increased risk for O_3-related health effects, based
35 on consistency in findings across studies and evidence of coherence in results from different

1 scientific disciplines (U.S. EPA, 2013, section 8.5): individuals with certain genotypes,

2 individuals with asthma, younger and older age groups, individuals with reduced intake of

3 certain nutrients, and outdoor workers. Multiple genetic variants have been observed in

4 epidemiologic and controlled human exposure studies to affect the risk of O_3-related respiratory

5 outcomes and support is provided by toxicological studies of genetic factors (U.S. EPA, 2013,

6 section 8.1). Asthma has been recognized in past reviews and continues to be well established in

7 this review as a risk factor for O_3-related health effects based on multiple lines of evidence,

8 included controlled human exposure and toxicological studies in animal models, as well as some

9 evidence from epidemiologic studies. This evidence supports our understanding of the

10 biological (intrinsic) factors that put individuals with asthma at greater risk than other groups

11 (U.S. EPA, 2013, section 8.2.2). As noted above, some extrinsic (exposure-related) and intrinsic

12 factors contribute to the identification of children as an at-risk lifestage. Children have higher

13 exposure and dose due to increased time spent outdoors and ventilation rate, their lungs are still

14 developing, and they are more likely than adults to have asthma (U.S. EPA, 2013, section

15 8.3.1.1). Older adults may also withstand greater O_3 exposure and not seek relief as quickly as

16 younger adults. Multiple epidemiologic, controlled human exposure and toxicological studies

17 reported that diets deficient in vitamins E and C are associated with risk of O_3-related health

18 effects for all lifestages. Previous studies have shown that increased exposure to O_3 due to

19 outdoor work leads to increased risk of O_3-related health effects and it is clear that outdoor

20 workers have higher exposures, and possibly greater internal doses, of O_3, which may lead to

21 increased risk of O_3-related health effects (U.S. EPA, 2013, section 8.5).

22   In some cases, it is difficult to determine a factor that results in increased risk of effects.

23 For example, previous assessments have included controlled human exposure studies in which

24 some healthy individuals demonstrate greater O_3-related health effects compared to other healthy

25 individuals. Intersubject variability has been observed for lung function decrements,

26 symptomatic responses, pulmonary inflammation, AHR, and altered epithelial permeability in

27 healthy adults exposed to O_3 and these results tend to be reproducible within a given individual

28 over a period of several months indicating differences in the intrinsic responsiveness. In many

29 cases the reasons for the variability is not clear. This may be because one or some of the factors

30 described above have not been evaluated in studies, or it may be that additional, unidentified

31 factors influence individual responses to O_3 (U.S. EPA, 2013, section 8.5).

32   As discussed in chapter 8 of the ISA, and further in Appendix 3C below, the challenges

33 and limitations in evaluating the factors that can increase risk for experiencing O_3-related health

34 effects may contribute to a lack of information about the factors that may increase risk from O_3

35 exposures. This lack of information may contribute to conclusions that evidence for some

1   factors, such as sex, SES, and obesity provided "suggestive" evidence of increased risk, or that
2   for a number of factors the evidence was inadequate to draw conclusions about potential increase
3   in risk of effects. Overall, the factors most strongly supported as contributing to increased risk of
4   populations for experience O_3-related effects were related to asthma, lifestage (children and older
5   adults), genetic variability, dietary factors, and working outdoors.

6   **3.1.5.2 Size of At-Risk Populations and Lifestages in the United States**

7   One consideration in the assessment of potential public health impacts is the size of
8   various population groups for which there is adequate evidence of increased risk for health
9   effects associated with O_3-related air pollution exposure. The factors for which the ISA judged
10   the evidence to be "adequate" with respect to contributing to increased risk of O_3-related effects
11   among various populations and lifestages included: asthma; childhood and older adulthood; diets
12   lower in vitamins C and E; certain genetic variants and, working outdoors (EPA, 2013, section
13   8.5).
14   With regard to asthma, Table 3-12 below summarizes information on the prevalence of
15   current asthma by age in the U.S. adult population in 2010 (Schiller et al. 2012; children - Bloom
16   et al., 2011). Individuals with current asthma constitute a fairly large proportion of the
17   population, including more than 25 million people. Asthma prevalence tends to be higher in
18   children than adults.
19   Within the U.S., approximately 8.2% of adults have reported currently having asthma
20   (Schiller et al., 2012) and 9.5% of children have reported currently having asthma (Bloom et al.,
21   2011). Table 3-12 below provides more detailed information on prevalence of asthma by age in
22   the U.S.
23

Table 3-7.   Prevalence of asthma by age in the U.S.

Age (years)	N (in thousands)	Percent
0-4	1,285	6.0
5-11	3,020	10.5
12-17	2,672	10.9
18-44	8,902	8.1
45-64	6,704	8.4
65-74	1,849	8.7
75+	1,279	7.4
Asthma prevalence is reported for "still has asthma"		
Source: Statistics for adults: Schiller et al. (2012); Statistics for children: Bloom et al. (2011)		

With regard to lifestages, based on U.S. census data from 2010 (Howden and Meyer, 2011), about 74 million people, or 24% of the U.S. population, are under 18 years of age and more than 40 million people, or about 13% of the U.S. population, are 65 years of age or older. Hence, a large proportion of the U.S. population, more than 33%, is included in age groups that are considered likely to be at increased risk for health effects from ambient O_3 exposure.

With regard to dietary factors, no statistics are available to estimate the size of an at-risk population based on nutritional status.

With regard to outdoor workers, in 2010 approximately 11.7% of the total number of people (143 million people) employed, or about 16.8 million people, worked outdoors one or more day per week (based on worker surveys).[52] Of these approximately 7.4% of the workforce, or about 7.8 million people, worked outdoors three or more days per week.

The health statistics data illustrate what is known as the "pyramid" of effects. At the top of the pyramid, there are approximately 2.5 million deaths from all causes per year in the U.S. population, with about 250 thousand respiratory-related deaths (CDC-WONDER, 2008). For respiratory health diseases, there are nearly 3.3 million hospital discharges per year (HCUP, 2007), 8.7 million respiratory ED visits (HCUP, 2007), 112 million ambulatory care visits

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[52] The O*NET program is the nation's primary source of occupational information. Central to the project is the O*NET database, containing information on hundreds of standardized and occupation-specific descriptors. The database, which is available to the public at no cost, is continually updated by surveying a broad range of workers from each occupation. http://www.onetcenter.org/overview.html
http://www.onetonline.org/find/descriptor/browse/Work_Context/4.C.2/

1   (Woodwell and Cherry, 2004), and an estimated 700 million restricted activity days per year due
2   to respiratory conditions (Adams et al., 1999). Combining small risk estimates with relatively
3   large baseline levels of health outcomes can result in quite large public health impacts. Thus,
4   even a small percentage reduction in O_3 health impacts on cardiopulmonary diseases would
5   reflect a large number of avoided cases.

6   ### 3.1.5.3   Averting Behavior

7   The activity pattern of individuals is an important determinant of their exposure (ISA,
8   U.S. EPA, 2013, section 4.4.1). Variation in O_3 concentrations among various
9   microenvironments means that the amount of time spent in each location, as well as the level of
10 activity, will influence an individual's exposure to ambient O_3. Activity patterns vary both
11 among and within individuals, resulting in corresponding variations in exposure across a
12 population and over time. Individuals can reduce their exposure to O_3 by altering their behaviors,
13 such as by staying indoors, being active outdoors when air quality is better, and by reducing their
14 activity levels or reducing the time being active outdoors on high-O_3 days (U.S. EPA, 2013,
15 section 4.4.2). The evidence in this topic area, while not addressed in the 2006 AQCD, is
16 evaluated in the ISA for this review.

17   The widely reported Air Quality Index (AQI) conveys advice to the public, and
18 particularly at-risk populations, on reducing exposure on days when ambient levels of common
19 air pollutants are elevated (www.airnow.gov). The AQI describes the potential for health effects
20 from O_3 (and other individual pollutants) in six color-coded categories of air-quality, ranging
21 from Good (green), Moderate (yellow), Unhealthy for Sensitive Groups (orange), Unhealthy
22 (red), and Very Unhealthy (purple), and Hazardous (maroon). Levels in the unhealthy ranges
23 (i.e., Unhealthy for Sensitive Groups and above) come with recommendations about reducing
24 exposure. Forecasted and actual AQI values for O_3 are reported to the public during the O_3
25 season. The AQI advisories explicitly state that children, older adults, people with lung disease,
26 and people who are active outdoors, may be at greater risk from exposure to O_3. People are
27 advised to reduce exposure depending on the predicted O_3 levels and the likelihood of risk. This
28 advice includes being active outdoors when air quality is better, and reducing activity levels or
29 reducing the time being active outdoors on high-O_3 days. Staying indoors to reduce exposure is
30 not recommended until air quality reaches the Very Unhealthy or Hazardous categories.
31   Evidence of individual averting behaviors in response to AQI advisories has been found
32 in several studies, including activity pattern and epidemiologic studies, especially for the at-risk
33 populations, such as children, older adults, and people with asthma, who are targeted by the
34 advisories. Such effects are less pronounced in the general population, possibly due to the
35 opportunity cost of behavior modification. Epidemiologic evidence from a study (Neidell and

1 Kinney, 2010) conducted in the 1990's in Los Angeles, CA reports increased asthma hospital

2 admissions among children and older adults when O3 alert days (1-hour max O3 concentration

3 >200 ppb) were excluded from the analysis of daily hospital admissions and O3 concentrations

4 (presumably thereby eliminating averting behavior based on high O3 forecasts). The lower rate

5 of admissions observed when alert days were included in the analysis suggests that estimates of

6 health effects based on concentration-response functions that do not account for averting

7 behavior may be biased towards the null (U.S. EPA, 2013, section 4.4.2).

8 **3.2   AIR QUALITY-, EXPOSURE-, AND RISK-BASED CONSIDERATIONS**

9 In order to inform judgments about the public health impacts of O_3-related health

10 effects, the second draft HREA has developed and applied models to estimate human exposures

11 to O_3 and O_3-associated health risks across the United States, with a specific focus on urban case

12 study areas (U.S. EPA, 2014).[53] The second draft HREA uses photochemical modeling to adjust

13 air quality from the 2006-2010 O_3 seasons to just meet the current and alternative standards for

14 the 2006-2008 and 2008-2010 periods.[54] In this section, staff considers estimates of short-term

15 O_3 exposures and estimates of health risks associated with short- and long-term O_3 exposures, for

16 air quality adjusted to just meet the current O_3 standard. In section 3.2.1, we consider the

17 implications for exposure and risk estimates of the approach used in the second draft HREA to

18 adjust air quality. Sections 3.2.2 and 3.2.3 discuss our exposure-based and risk-based

19 considerations, respectively. In these sections we specifically consider the following question:

20 • **What are the nature and magnitude of O_3 exposures and health risks remaining**
21 **upon adjusting recent air quality to just meet the current O_3 standard, and what are**
22 **the important uncertainties associated with those exposure and risk estimates?**

23 **3.2.1   Consideration of the Adjusted Air Quality Used in Exposure and Risk**
24 **Assessments**

25 In the first draft HREA for this review, as in the last review, the EPA relied upon

26 quadratic rollback to adjust hourly O_3 concentrations in urban case study areas to just meet the

27 current O_3 standard (U.S. EPA, 2012b). Although the quadratic rollback method reproduces

28 historical patterns of air quality changes better than some alternative methods, it relies on

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[53] The 15 urban case study areas analyzed for exposures are Atlanta, Baltimore, Boston, Chicago, Cleveland, Dallas, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC. Morbidity and mortality risk estimates are presented for these same areas, with the exception of Chicago, Dallas, and Washington, DC. The second draft HREA also presents a national scale mortality risk assessment for unadjusted (recent) air quality. This national-scale assessment, which focuses on existing air quality conditions and does not estimate the health risks associated with just meeting the current or alternative standards, can provide perspective on the relationship between national-scale O_3 public health impacts and impacts estimated in specific urban areas.

[54] Three-year periods are used recognizing that the current standard is the average across three years of the annual fourth-highest daily maximum 8-hour average concentration.

1   statistical relationships without explicitly accounting for atmospheric chemistry and precursor

2   emissions (U.S. EPA, 2014, Chapter 4). An important drawback of the quadratic rollback

3   approach, recognized in the first draft HREA (U.S. EPA, 2012b), is that it forces all monitors in

4   an assessment area to exhibit the same response when air quality is adjusted. It does not allow for

5   the spatial or temporal heterogeneity in responses that result from the non-linear atmospheric

6   chemistry that influences ambient O_3 concentrations (U.S. EPA, 2014, Chapter 4). Because

7   quadratic rollback does not account for physical and chemical atmospheric processes, or the

8   sources of emissions precursors that lead to O_3 formation, a backstop or "floor" must be used

9   when applying quadratic rollback to just meet current or alternative standards to ensure that

10   estimated O_3 is not reduced in a manner inconsistent with O_3 chemistry, such as to reduce

11   concentrations below that associated with background sources (U.S. EPA, 2014, Chapter 4).

12   Consistent with recommendations from the National Research Council of the National

13   Academies (NRC, 2008), the second draft HREA uses a photochemical model to estimate

14   sensitivities of O_3 to changes in precursor emissions, in order to estimate ambient O_3

15   concentrations that would just meet the current and alternative standards (U.S. EPA, 2014,

16   Chapter 4).[55] For the urban case study areas evaluated in the second draft HREA, this model-

17   based adjustment approach was set up to estimate hourly O_3 concentrations at each monitor

18   location when modeled U.S. anthropogenic precursor emissions (i.e., NO_X, VOC)[56] were reduced

19   to estimate air quality that just meets the current and alternative O_3 standards.[57]

20   As discussed in Chapter 4 of the second draft HREA (U.S. EPA, 2014), this approach

21   models the physical and chemical atmospheric processes that influence ambient O_3

22   concentrations. Compared to the quadratic rollback approach, it provides more realistic estimates

23   of the spatial and temporal responses of O_3 to reductions in precursor emissions. These improved

24   estimates avoid many of the limitations inherent in the quadratic rollback method, including the

25   requirement that all monitors in an assessment area exhibit the same response upon model

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[55]The second draft HREA uses the CMAQ photochemical model instrumented with the higher order direct decoupled method (HDDM) to estimate ozone concentrations that would occur with the achievement of the current and alternative O_3 standards (U.S. EPA, 2014, Chapter 4).

[56]Exposure and risk analyses for most urban case study areas focus on reducing NO_X emissions alone (NO_X emissions were reduced by about 40 to 85% for the current standard, and up to 95% for alternatives). In most of the urban case study areas, the addition of modeled reductions in VOC emissions did not alter the reductions in NO_X emissions required to simulate the current or alternative standards. However, in Chicago and Denver, the addition of reductions in VOC emissions allowed for smaller NO_X emissions reductions to simulate the current and alternative standards. Therefore, exposure and risk analyses for Chicago and Denver focus on reductions in emissions of both NO_X and VOC (U.S. EPA, 2014, section 4.3.3.1).

[57]Although this chapter focuses on the current standard, our overarching considerations regarding model-adjusted air quality also apply to alternative standards simulated in the second draft HREA. Alternative standards are discussed in chapter 4 of this second draft PA.

1   adjustment to the current and/or alternative standards. Because model-adjusted air quality

2   scenarios are based on reducing only U.S. anthropogenic emissions, this approach also does not

3   require the specification of background concentrations as a rollback "floor" (U.S. EPA, 2014,

4   section 4.3.3).

5   The use of this model-based air quality adjustment approach in the second draft HREA

6   has important implications for the patterns of ambient O_3 concentrations estimated in urban case

7   study areas. Specifically, in locations and time periods when NO_X is predominantly contributing

8   to O_3 formation (e.g., downwind of important NO_X sources, where the highest O_3 concentrations

9   often occur), model-based adjustment to the current and alternative standards decreases

10 estimated ambient O_3 concentrations compared to recent monitored concentrations (U.S. EPA,

11 2014, section 4.3.3.2). In contrast, in locations and time periods when NO_X is predominantly

12 contributing to O_3 titration (e.g., in urban centers with high concentrations of NO_X emissions,

13 where ambient O_3 concentrations are often suppressed and thus relatively low[58]), model-based

14 adjustment increases ambient O_3 concentrations compared to recent measured levels (U.S. EPA,

15 2014, section 4.3.3.2) (Chapter 2, above).

16   Within urban case study areas, the overall impacts of model-based air quality adjustment

17 are to reduce relatively high ambient O_3 concentrations (i.e., concentrations at the upper ends of

18 ambient distributions) and to increase relatively low O_3 concentrations (i.e., concentrations at the

19 lower ends of ambient distributions) (U.S. EPA, 2014, section 4.3.3.2, Figures 4-8 to 4-11).

20 Seasonal means of daily concentrations generally exhibit only modest changes upon model

21 adjustment, reflecting the seasonal balance between daily decreases and increases in ambient

22 concentrations (U.S. EPA, 2014, Figures 4-10 and 4-11). The resulting compression in

23 distributions of ambient O_3 concentrations is evident in all of the urban case study areas

24 evaluated, though the degree of compression varies considerably across areas (U.S. EPA, 2014,

25 Figures 4-10 and 4-11).

26   Adjusted patterns of O_3 air quality have important implications for exposure and risk

27 estimates in urban case study areas. Estimates influenced largely by the upper ends of the

28 distribution of ambient concentrations (i.e., exposures of concern and lung function risk

29 estimates, as discussed in sections 3.2.2 and 3.2.3.1 below) will decrease with model-adjustment

30 to the current and alternative standards. In contrast, seasonal risk estimates influenced by the full

31 distribution of ambient O_3 concentrations (i.e., epidemiology-based risk estimates, as discussed

32 in section 3.2.3.2 below) will either increase or decrease in response to model adjustment,

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[58]Titration is also prominent during time periods when photochemistry is limited, such as at night and on cool, cloudy days.

1　depending on the balance between the daily decreases in high O_3 concentrations and increases in

2　low O_3 concentrations.[59]

3　　　We further consider the implications of the spatial and temporal patterns of adjusted air

4　quality within the context of exposure (section 3.2.2) and risk (section 3.2.3) estimates for O_3

5　concentrations adjusted to just meet the current standard. As discussed below (section 3.2.3.2),

6　these altered patterns are particularly important to consider when interpreting epidemiology-

7　based risk estimates.

8　　**3.2.2　Exposure-Based Considerations**

9　　　The exposure assessment presented in the second draft HREA (U.S. EPA, 2014) provides

10　estimates of the number of people exposed to various concentrations of ambient O_3, while at

11　specified exertion levels. The second draft HREA estimates exposures in 15 urban case study

12　areas for school-age children (ages 5 to 18), asthmatic school-age children, asthmatic adults, and

13　older adults, reflecting the strong evidence indicating that these populations are potentially at

14　increased risk for O_3-attributable effects (EPA, 2013, Chapter 8; section 3.1.2, above). An

15　important purpose of these exposure estimates is to provide perspective on the extent to which

16　air quality adjusted to just meet the current O_3 NAAQS could be associated with exposures to O_3

17　concentrations reported to result in respiratory effects.[60] Estimates of such "exposures of

18　concern" provide perspective on the potential public health impacts of O_3-related effects,

19　including for effects that cannot currently be evaluated in a quantitative risk assessment (e.g.,

20　airway inflammation).

21　　　In the absence of large scale exposure studies that encompass the general population, as

22　well as at-risk populations, modeling is the preferred approach to estimating exposures to O_3.

23　Additionally, the use of exposure modeling facilitates the estimation of exposures resulting from

24　ambient air concentrations differing from those in exposure studies (e.g., concentrations just

25　meeting the current standard). In the second draft HREA, population exposures to ambient O_3

26　concentrations are estimated using the current version of the Air Pollutants Exposure (APEX)

27　model. The APEX model simulates the movement of individuals through time and space and

28　estimates their exposures to a given pollutant in indoor, outdoor, and in-vehicle

29　microenvironments (U.S. EPA, 2014, section 5.1.3). APEX takes into account the most

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[59]In addition, because epidemiology-based risk estimates use "area-wide" average O_3 concentrations, calculated by averaging concentrations across multiple monitors in urban case study areas (section 3.2.3.2 below), risk estimates on a given day depend on the daily balance between increasing and decreasing O_3 concentrations at individual monitors.

[60]In addition, the range of modeled personal exposures to ambient O_3 provide an essential input to the portion of the health risk assessment based on exposure-response functions (for lung function decrements) from controlled human exposure studies. The health risk assessment based on exposure-response information is discussed in section 3.2.3, below.

1   significant factors that contribute to total human exposure to ambient O_3, including the temporal

2   and spatial distributions of people and O_3 concentrations throughout an urban area, the variation

3   of O_3 concentrations within various microenvironments, and the effects of exertion on breathing

4   rate in exposed individuals (U.S. EPA, 2014, section 5.1.3). To the extent spatial and/or temporal

5   patterns of ambient O_3 concentrations are altered upon model adjustment, as discussed above,

6   exposure estimates reflect population exposures to those altered patterns.

7   The second draft HREA estimates 8-hour exposures at or above benchmark

8   concentrations of 60, 70, and 80 ppb for individuals engaged in moderate or greater exertion.

9   Benchmarks reflect exposure concentrations at which O_3-induced respiratory effects are known

10   to occur in some healthy adults engaged in moderate, intermittent exertion, based on evidence

11   from controlled human exposure studies (section 3.1.2.1 above and U.S. EPA, 2013, section 6.2).

12   The amount of weight to place on the estimates of exposures at or above specific benchmark

13   concentrations depends in part on the weight of the scientific evidence concerning health effects

14   associated with O_3 exposures at that concentration. It also depends on judgments about the

15   importance, from a public health perspective, of the health effects that are known or can

16   reasonably be inferred to occur as a result of exposures at benchmark concentrations (sections

17   3.1.3, 3.1.5 above).

18   As discussed in more detail above (section 3.1.2.1), the health evidence that supports

19   evaluating exposures of concern at or above benchmark concentrations of 60, 70, and 80 ppb

20   comes from a large body of controlled human exposure studies reporting a variety of respiratory

21   effects in healthy adults. The lowest O_3 exposure concentration for which controlled human

22   exposure studies have reported respiratory effects in healthy adults is 60 ppb, with more

23   evidence supporting this benchmark concentration in the current review than in the last review.

24   In healthy adults, exposures to 60 ppb O_3 have been reported to decrease lung function and to

25   increase airway inflammation. Exposures of healthy adults to 70 ppb O_3 have been reported to

26   result in larger lung function decrements, compared to 60 ppb, as well as in increased respiratory

27   symptoms. Exposures of healthy adults to 80 ppb O_3 have been reported to result in larger lung

28   function decrements than following exposures to 60 or 70 ppb, increased airway inflammation,

29   increased respiratory symptoms, increased airways responsiveness, and decreased lung host

30   defense (section 3.1.2.1, above). As discussed above (section 3.1.3), respiratory effects reported

31   following exposures to O_3 concentrations of 60, 70, or 80 ppb meet ATS criteria for adverse

32   effects, result in effects judged important by CASAC in past reviews, and/or could contribute to

33   the clearly adverse effects reported in epidemiologic studies evaluating broader populations.

34   Compared to the healthy individuals included in the studies that provided the basis for the

35   benchmarks, at-risk populations (e.g., asthmatics, children) are more likely to experience larger

36   and/or more serious effects (e.g., U.S. EPA 2013, p. 6-21).

1   In considering estimates of O_3 exposures of concern at or above benchmarks of 60, 70,

2   and 80 ppb, within the context of the adequacy of the current standard, we first address the

3   following specific question:

4   •   **What are the nature and magnitude of the short-term O_3 exposures of concern**

5   **remaining upon adjustment of air quality to just meet the current O_3 standard?**

6   In addressing this question, we focus on modeled exposures for school-age children (ages

7   5-18) and asthmatic school-age children, two of the at-risk populations identified in the ISA

8   (section 3.1.5 above). The percentages of children estimated to experience exposures of concern

9   are considerably larger than the percentages estimated for adult populations (i.e., approximately

10   3-fold larger across cities) (U.S. EPA, 2014, Figures 5-5, 5-6, 5-7). The larger exposure

11   estimates for children are due primarily to the larger percentage of children estimated to spend an

12   extended period of time being physically active outdoors (U.S. EPA, 2014, section 5.4.1, Figure

13   5-16).

14   Key results for children are summarized below for air quality adjusted to simulate just

15   meeting the current O_3 NAAQS (Figures 3-7 to 3-10),[61] and we note that estimates for all

16   children and asthmatic children are virtually indistinguishable (U.S. EPA, 2014, section 5.3.2).

17   The estimates presented in Figures 3-7 to 3-10 below reflect consistent reductions in estimated

18   exposures of concern across urban case study areas, relative to recent (i.e., unadjusted) air quality

19   (U.S. EPA, 2014, Appendix to Chapter 5). When averaged over the years evaluated in the

20   HREA, reductions of up to about 70% were estimated, compared to recent air quality. These

21   reductions in estimated exposures of concern, relative to unadjusted air quality, reflect the

22   consistent reductions in the highest ambient O_3 concentrations upon model adjustment to just

23   meet the current standard (section 3.2.1 above; U.S. EPA, 2014, Chapter 4).

24   Although exposure estimates differ between children and adults, the patterns of results

25   across the cities and years are similar among all of the populations evaluated (U.S. EPA, 2014,

26   Figures 5-12 to 5-15). Therefore, while we highlight estimates in children, we also note that the

27   patterns of exposures estimated for children represent the patterns estimated for adult asthmatics

28   and older adults.

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[61]Figures 3-7 and 3-8 present estimates of one or more exposures of concern. Figures 3-9 and 3-10 present estimates of two or more exposures of concern.

1
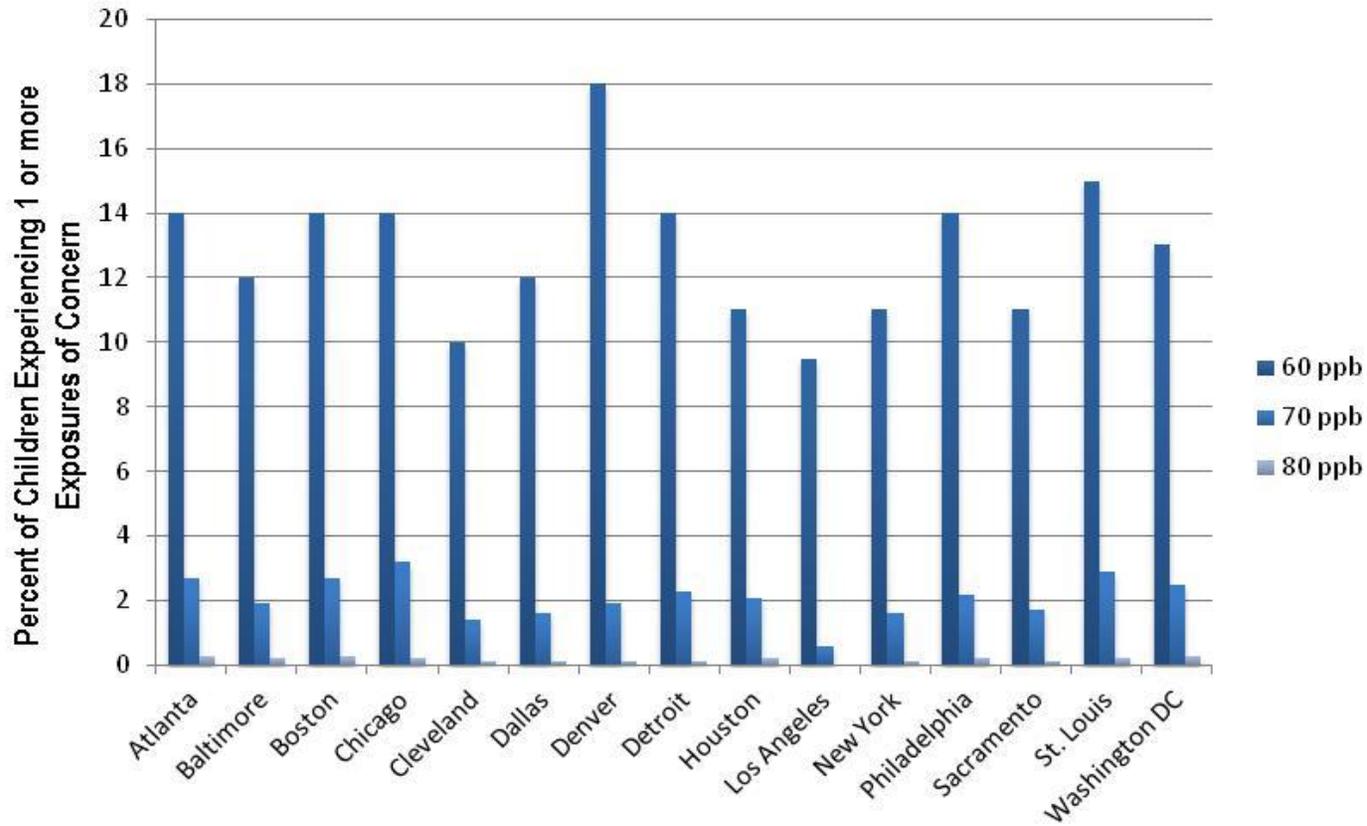
Figure 3-7. Percent of children estimated to experience one or more exposures of concern at or above 60, 70, 80 ppb with air quality adjusted to just meet the current standard (averaged over 2006 to 2010).

4

1

2
3

4

5 **Figure 3-8. Percent of children estimated to experience one or more exposures of concern at or above 60, 70, 80 ppb with air**
6 **quality adjusted to just meet the current standard (worst-case year, 2006 to 2010).**

7
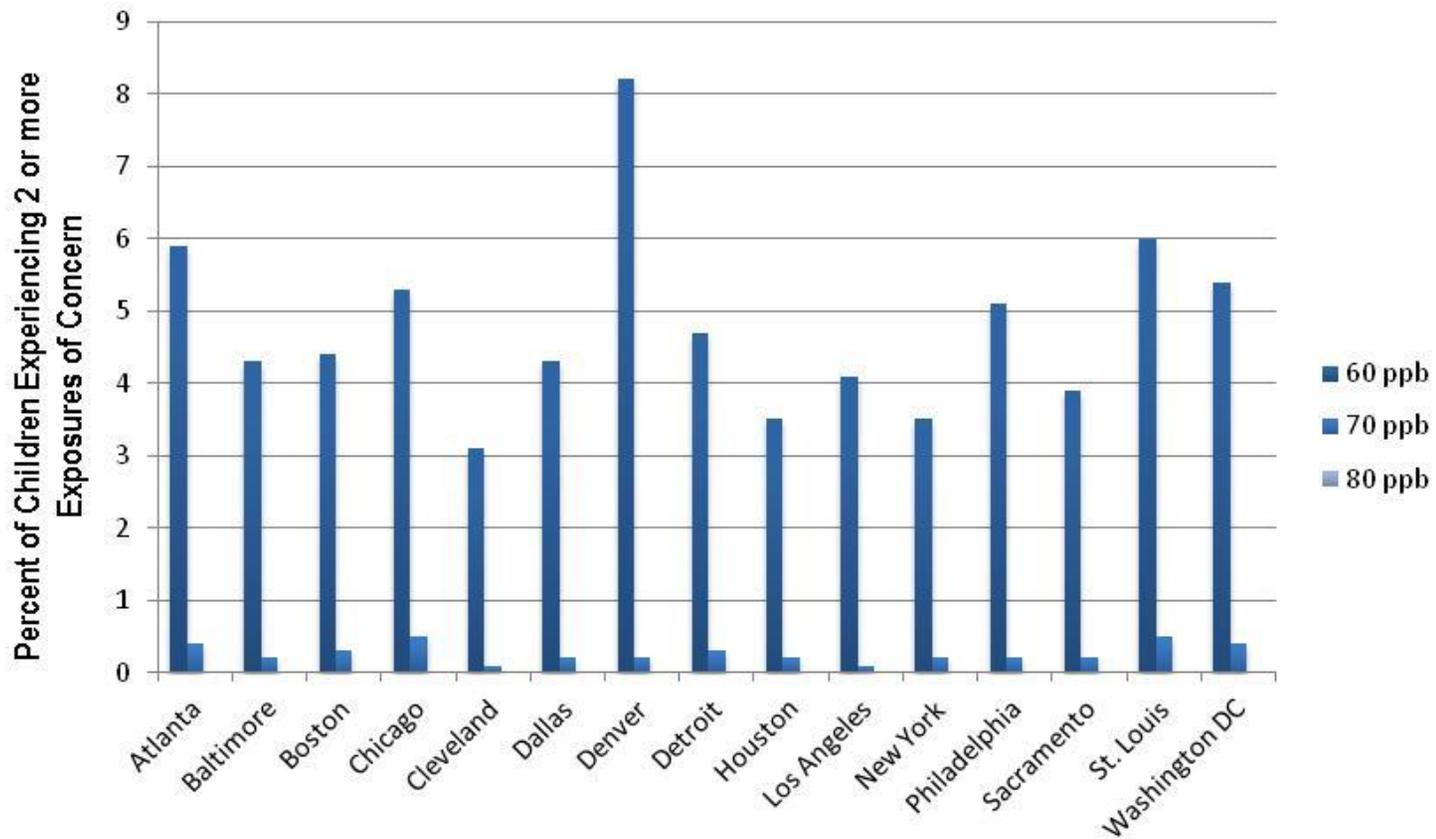
1

2 **Figure 3-9. Percent of children estimated to experience two or more exposures of concern at or above 60, 70, 80 ppb with air**
3   **quality adjusted to just meet the current standard (Averaged over 2006 to 2010)**

4

1
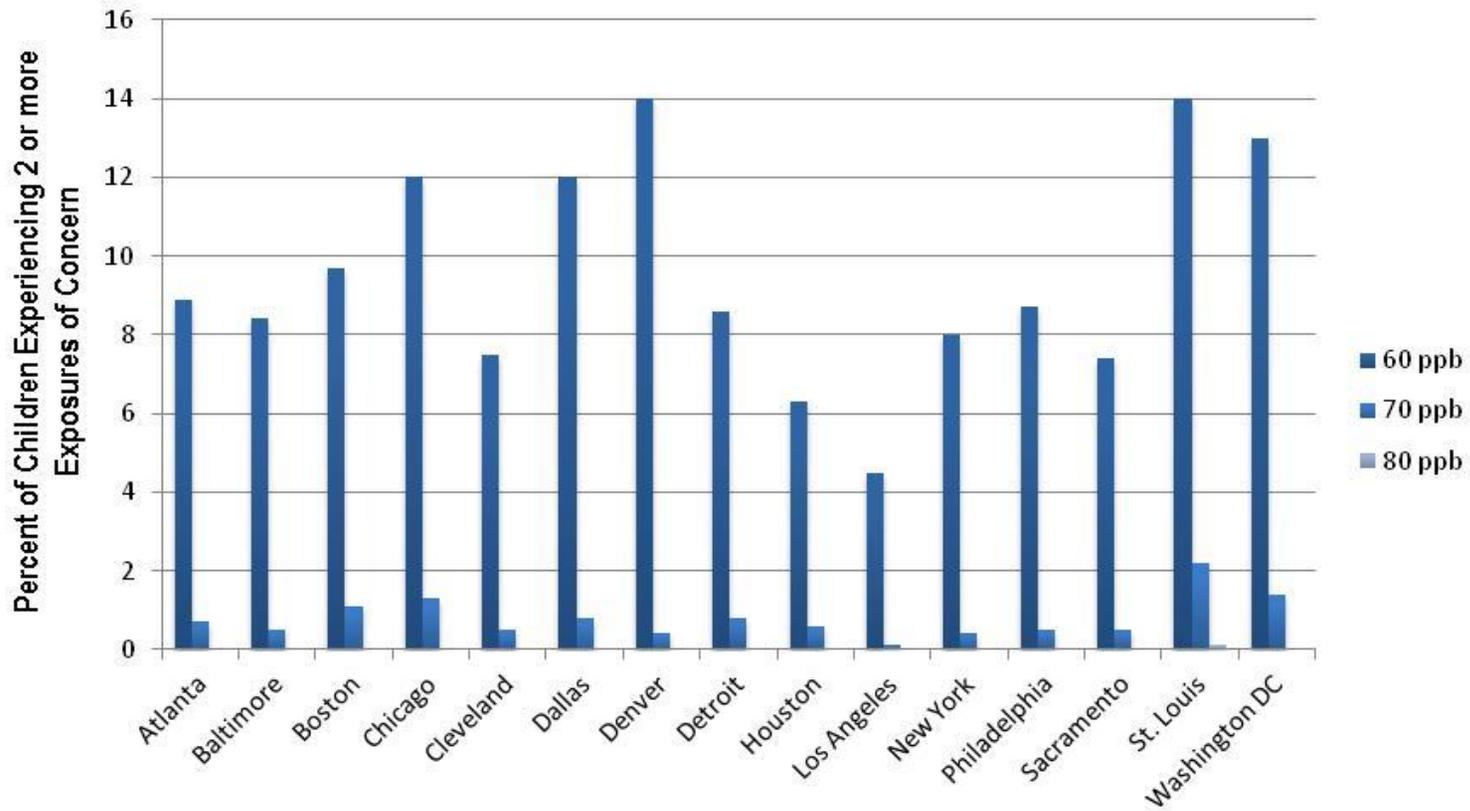
2

3   **Figure 3-10. Percent of children estimated to experience two or more exposures of concern at or above 60, 70, 80 ppb with air**
4   **quality adjusted to just meet the current standard (Worst-Case Year, 2006 to 2010)**

5

1   Based on Figures 3-7 to 3-10 and the associated details described in the second draft

2   HREA (U.S. EPA 2014, Chapter 5), we take note of the following with regard to exposures that

3   are estimated to be allowed by the current standard:

4   1. For exposures of concern at or above 60 ppb:

        a. On average over the years 2006 to 2010, the current standard is estimated to allow
6   approximately 10 to 18% of children in urban case study areas to experience one or
7   more exposures of concern at or above 60 ppb. Summing across urban case study
8   areas, these percentages correspond to almost 2.5 million children experiencing
9   approximately 4 million exposures of concern at or above 60 ppb during a single O_3
10   season. Of these children, almost 250,000 are asthmatics.

12   b. On average over the years 2006 to 2010, the current standard is estimated to allow
13   approximately 3 to 8% of children in urban case study areas to experience two or
14   more exposures of concern to O_3 concentrations at or above 60 ppb. Summing across
15   the urban case study areas, these percentages correspond to almost 900,000 children
16   (including almost 90,000 asthmatic children) estimated to experience at least two O_3
17   exposure concentrations at or above 60 ppb during a single O_3 season.

19   c. In the worst-case years (i.e., those with the largest exposure estimates), the current
20   standard is estimated to allow approximately 10 to 25% of children to experience one
21   or more exposures of concern at or above 60 ppb, and approximately 4 to 14% to
22   experience two or more exposures of concern at or above 60 ppb.

24   2. For exposures of concern at or above 70 ppb:

25   a. On average over the years 2006 to 2010, the current standard is estimated to allow up
26   to approximately 3% of children in urban case study areas to experience one or more
27   exposures of concern at or above 70 ppb. Summing across urban case study areas,
28   almost 400,000 children (including almost 40,000 asthmatic children) are estimated to
29   experience O_3 exposure concentrations at or above 70 ppb during a single O_3 season.

31   b. On average over the years 2006 to 2010, the current standard is estimated to allow
32   less than 1% of children in urban case study areas to experience two or more
33   exposures of concern to O_3 concentrations at or above 70 ppb.

35   c. In the worst-case years, the current standard is estimated to allow approximately 1 to
36   8% of children to experience one or more exposures of concern at or above 70 ppb,
37   and up to approximately 2% to experience two or more exposures of concern, at or
38   above 70 ppb.

40   3. For exposures of concern at or above 80 ppb: The current standard is estimated to allow
41   about 1% or fewer children in urban case study areas to experience exposures of concern at
42   or above 80 ppb, even in years with the highest exposure estimates.

1   In further evaluating estimated exposures of concern from the 2nd draft HREA, we next
2   consider the following question:

3   • **What are the important sources of uncertainty associated with exposure estimates?**

4   Due to variability in responsiveness, only a subset of individuals who experience
5   exposures at or above a benchmark concentration can be expected to experience health effects.
6   Given the lack of sufficient exposure-response information for most of the health effects that
7   informed benchmark concentrations, estimates of the number of people likely to experience
8   exposures at or above benchmark concentrations generally cannot be translated into quantitative
9   estimates of the number of people likely to experience specific health effects.[62] We view health-
10   relevant exposures as a continuum with greater confidence and less uncertainty about the
11   existence of health effects at higher O_3 exposure concentrations, and less confidence and greater
12   uncertainty as one considers lower exposure concentrations. This view draws from the overall
13   body of available health evidence, which indicates that as exposure concentrations increase the
14   incidence, magnitude, and severity of effects increases.
15   Though we have less confidence in the likelihood of adverse health effects as O_3
16   exposure concentrations decrease, we also note that the controlled human exposure studies that
17   provided the basis for health benchmark concentrations have not evaluated at-risk populations.
18   Compared to the healthy individuals included in controlled human exposure studies, members of
19   at-risk populations (e.g., asthmatics, children) could be more likely to experience adverse effects,
20   could experience larger and/or more serious effects, and/or could experience effects following
21   exposures to lower O_3 concentrations. In considering estimated exposures of concern within the
22   context of drawing conclusions on the adequacy of the current standard (section 3.4, below), we
23   balance concerns about the potential for adverse health effects, including effects in at-risk
24   populations, with our increasing uncertainty regarding the likelihood of such effects following
25   exposures to lower O_3 concentrations.
26   Uncertainties associated with the APEX exposure modeling also have the potential to be
27   important in our consideration of the adequacy of the current standard. For example, the HREA
28   concludes that exposures of concern could be underestimated for some individuals who are
29   frequently and routinely active outdoors during the warm season (U.S. EPA, section 5.5.2). This
30   could include outdoor workers and children who are frequently active outdoors. The HREA
31   specifically notes that long-term diary profiles (i.e., monthly, annual) do not exist for such
32   populations, limiting the extent to which APEX outputs reflect people who follow similar daily
33   routines resulting in high exposures, over extended periods of time. Thus, exposure estimates

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[62]The exception to this is lung function decrements, as discussed below (section 3.2.3.1).

1 generated from the general pool of available diary profiles likely do not reflect the most highly
2 exposed individuals in the population.

3   In order to evaluate the potential implications of this uncertainty for exposure estimates,
4 the second draft HREA reports the results of limited sensitivity analyses using subsets of diaries
5 specifically selected to reflect groups spending a larger proportion of time being active outdoors
6 during the O_3 season. When diaries were selected to mimic exposures that could be experienced
7 by outdoor workers, the percent of modeled individuals estimated to experience exposures of
8 concern increased compared to other adult populations evaluated. The percent of outdoor
9 workers estimated to experience exposures of concern were generally similar to the percentages
10 estimated for children (i.e., using the full database of diary profiles) in the worst-case cities and
11 years (i.e., cities and years with the highest exposure estimates) (U.S. EPA, 2014, Figure 5-11).
12 In addition, when diaries were restricted to children who did not report any time spent inside a
13 school or performing paid work (i.e., to mimic children spending particularly large portions of
14 their time outdoors during the summer), the number experiencing exposures of concern increased
15 by approximately 30% (U.S. EPA, 2014, section 5.3.3). Though these sensitivity analyses are
16 limited to single urban case study areas, and though there is uncertainty associated with diary
17 selection approaches to mimic highly exposed populations, they suggest the possibility that some
18 portions of the population could experience more frequent exposures of concern than indicated
19 by estimates based on the full database of activity diary profiles.

20   In further considering activity diaries, the HREA also notes that growing evidence
21 indicates that people can change their behavior in response to high O_3 concentrations, reducing
22 the time spent being active outdoors (U.S. EPA, 2014, section 5.4.3). Commonly termed
23 "averting behaviors," these altered activity patterns could reduce personal exposure
24 concentrations. Therefore, the second draft HREA also performed limited sensitivity analyses to
25 evaluate the potential implications of averting behavior for estimated exposures of concern.
26 These analyses suggest that averting behavior could reduce the percentages of children estimated
27 to experience exposures of concern at or above the 60 or 70 ppb benchmark concentrations by
28 approximately 10 to 30%, with larger reductions possible for the 80 ppb benchmark (U.S. EPA,
29 2014, Figure 5-12). As discussed above for other sensitivity analyses, these analyses are limited
30 to a single urban case study area and are subject to uncertainties associated with assumptions
31 about the prevalence and duration of averting behaviors. However, the results suggest that
32 exposures of concern could be overestimated, particularly in children (Neidell et al., 2009; U.S.
33 EPA, 2013, Figures 4-7 and 4-8), if the possibility for averting behavior is not incorporated into
34 estimates.

1   **3.2.3   Risk-Based Considerations**

2   For some health endpoints, there is sufficient scientific evidence and information
3   available to support the development of quantitative estimates of O_3-related health risks. In the
4   last review of the O_3 NAAQS, the quantitative health risk assessment estimated O_3-related lung
5   function decrements, respiratory symptoms, respiratory-related hospital admissions, and non-
6   accidental and cardiorespiratory-related mortality (U.S. EPA, 2007). In those analyses, both
7   controlled human exposure and epidemiologic studies were used for the quantitative assessment
8   of O_3-related human health risks.

9   In the current review, for short-term O_3 concentrations the second draft HREA estimates
10   lung function decrements; respiratory symptoms in asthmatics; hospital admissions and
11   emergency department visits for respiratory causes; and all-cause mortality (U.S. EPA, 2014).
12   For "long-term" O_3 concentrations, the second draft HREA estimates respiratory mortality (U.S.
13   EPA, 2014).[63] Estimates of O_3-induced lung function decrements are based on exposure
14   modeling, as noted above, combined with exposure-response relationships from controlled
15   human exposure studies (U.S. EPA, 2014, Chapter 6). Estimates of O_3-associated respiratory
16   symptoms; hospital admissions and emergency department visits; and mortality are based on
17   concentration-response relationships from epidemiologic studies (U.S. EPA, 2014, Chapter 7).
18   As with the exposure assessment discussed above, O_3-associated health risks are estimated for
19   recent air quality and for ambient concentrations model-adjusted to just meet the current 8-hour
20   O_3 NAAQS, based on 2006-2010 air quality and adjusted precursor emissions.

21   Section 3.2.3.1 below discusses risk results for O_3-induced lung function decrements
22   following short-term exposures, based on exposure modeling results and exposure-response
23   relationships from controlled human exposure studies. Section 3.2.3.2 discusses epidemiology-
24   based risk estimates, with a focus on all-cause mortality (short-term O_3 concentrations);
25   respiratory-related morbidity outcomes (short-term O_3 concentrations); and respiratory mortality
26   (long-term O_3 concentrations).

27   **3.2.3.1 Risk of Lung Function Decrements**

28   In the last review, EPA conducted a health risk assessment that produced risk estimates
29   for the number and percent of school-aged children, asthmatic school-aged children, and the
30   general population experiencing lung function decrements associated with O_3 exposures for 12
31   urban areas. These estimates were based on exposure-response relationships developed from

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[63] Risk estimates for "long-term" concentrations are based on the concentration-response relationship identified in the study by Jerrett et al. (2009). As discussed above, study authors used April to September averages of 1-hour daily maximum O_3 concentrations as surrogates for "long-term" exposures.

1   analysis of data from several controlled human exposure studies, combined with exposure

2   estimates developed for children and adults (U.S. EPA, 2007a).

3   In the current review, the second draft HREA estimates risks of lung function decrements

4   in school-aged children (ages 5 to 18), asthmatic school-aged children, and the general adult

5   population for 15 urban case study areas.[64] The results presented in the second draft HREA are

6   based on an updated dose-threshold model that estimates FEV_1 responses for individuals

7   following short-term exposures to O_3 (McDonnell, Stewart, and Smith, 2010), reflecting

8   methodological improvements since the last review (U.S. EPA, 2014, section 6.2.4). The impact

9   of the dose threshold is that O_3-induced FEV_1 decrements result primarily from exposures on

10   days with ambient O_3 concentrations above about 40 ppb (U.S. EPA, 2013, Chapter 6).

11   As discussed above (section 3.1.3), for effects such as lung function decrements, which

12   are transient and reversible, aspects such as the likelihood that these effects would occur

13   repeatedly and would interfere with normal activities are important to consider in making

14   judgments about adversity to individuals. As stated in the 2006 Criteria Document (Table 8-3,

15   p.8-68), for people with lung disease even moderate functional responses (e.g., FEV_1 decrements

16   ≥ 10% but < 20%) would likely interfere with normal activities for many individuals, and would

17   likely result in more frequent medication use. Moreover, as noted above, in a recent letter to the

18   Administrator, the CASAC O_3 Panel stated that "[c]linically relevant effects are decrements >

19   10%, a decrease in lung function considered clinically relevant by the American Thoracic

20   Society" (Samet, 2011, p.2). The CASAC O_3 Panel also stated that:

21   [A] 10% decrement in FEV_1 can lead to respiratory symptoms, especially in
22   individuals with pre-existing pulmonary or cardiac disease. For example, people
23   with chronic obstructive pulmonary disease have decreased ventilatory reserve
24   (i.e., decreased baseline FEV_1) such that a ≥ 10% decrement could lead to
25   moderate to severe respiratory symptoms (Samet, 2011, p.7).

26   In judging the extent to which moderate lung function decrements represent effects that

27   should be regarded as adverse to the health status of individuals, in previous NAAQS reviews we

28   have also considered the extent to which decrements were experienced repeatedly during the

29   course of a year (Staff Paper, U.S. EPA, 2007). Although some experts would judge single

30   occurrences of moderate responses to be a ''nuisance,'' especially for healthy individuals, a

31   more general consensus view of the adversity of such moderate responses emerges as the

32   frequency of occurrence increases. Thus in the past EPA has judged that repeated occurrences of

33   moderate responses, even in otherwise healthy individuals, may be considered to be adverse

---

[64]As noted for the exposure assessment above, the 15 cities assessed are Atlanta, Baltimore, Boston, Chicago,
Cleveland, Dallas, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and
Washington, DC.

1   since they could well set the stage for more serious illness (61 FR 65723). The CASAC panel in
2   the 1997 NAAQS review expressed a consensus view that these "criteria for the determination of
3   an adverse physiological response were reasonable" (Wolff, 1995). In the review completed in
4   2008, estimates of repeated occurrences continued to be an important public health policy factor
5   in judging the adversity of moderate lung function decrements in healthy and asthmatic
6   populations (72 FR 37850, July 11, 2007).

7   The second draft HREA estimates risks of moderate to large lung function decrements,
8   defined as FEV_1 decrements $\geq$ 10%, $\geq$ 15%, or $\geq$ 20%. In evaluating these lung function risk
9   estimates within the context of considering the adequacy of the current O_3 standard, we first
10   consider the following specific question:

11   • **What are the nature and magnitude of lung function risks remaining upon just meeting**
12   **the current O_3 standard?**

13   In considering risks of O_3-induced FEV_1 decrements, we focus on the percent of children
14   estimated to experience decrements $\geq$ 10, 15, and 20%, noting that the percentage of asthmatic
15   children estimated to experience such decrements is virtually the same as the percentage
16   estimated for all children. Compared to children, only a very small percentage of adults were
17   estimated to experience O_3-induced FEV_1 decrements (U.S. EPA, 2014, Appendix 6B). As for
18   exposures of concern (see above), the patterns of results across urban case study areas and over
19   the years evaluated are similar in children and adults (U.S. EPA, 2014, Chapter 6). Therefore,
20   while we highlight estimates in children, we note that these results are also representative of the
21   patterns estimated for adult populations.

22   Key results for children are summarized below for air quality adjusted to just meet the
23   current O_3 NAAQS (Figures 3-11 to 3-14).[65] The estimates presented in Figures 3-11 to 3-14
24   below reflect consistent reductions across urban case study areas in the percent of children
25   estimated to experience O_3-induced lung function decrements, relative to recent (i.e., unadjusted)
26   air quality (U.S. EPA, 2014, Appendix to Chapter 6). When averaged over the years evaluated
27   in the HREA, reductions of up to about 40% were estimated compared to recent air quality.
28   These reductions reflect the consistent decreases in relatively high ambient O_3 concentrations
29   upon adjustment to just meet the current standard (section 3.2.1 above; U.S. EPA, 2014, Chapter
30   4).[66]

---

[65]Figures 3-11 and 3-12 present estimates of one or more decrements. Figures 3-13 and 3-14 present estimates of
two or more decrements.
[66]As noted above, the impact of the dose threshold is that O_3-induced FEV_1 decrements result primarily from days
with ambient O_3 concentrations above about 40 ppb (U.S. EPA, 2013, Chapter 6).

1
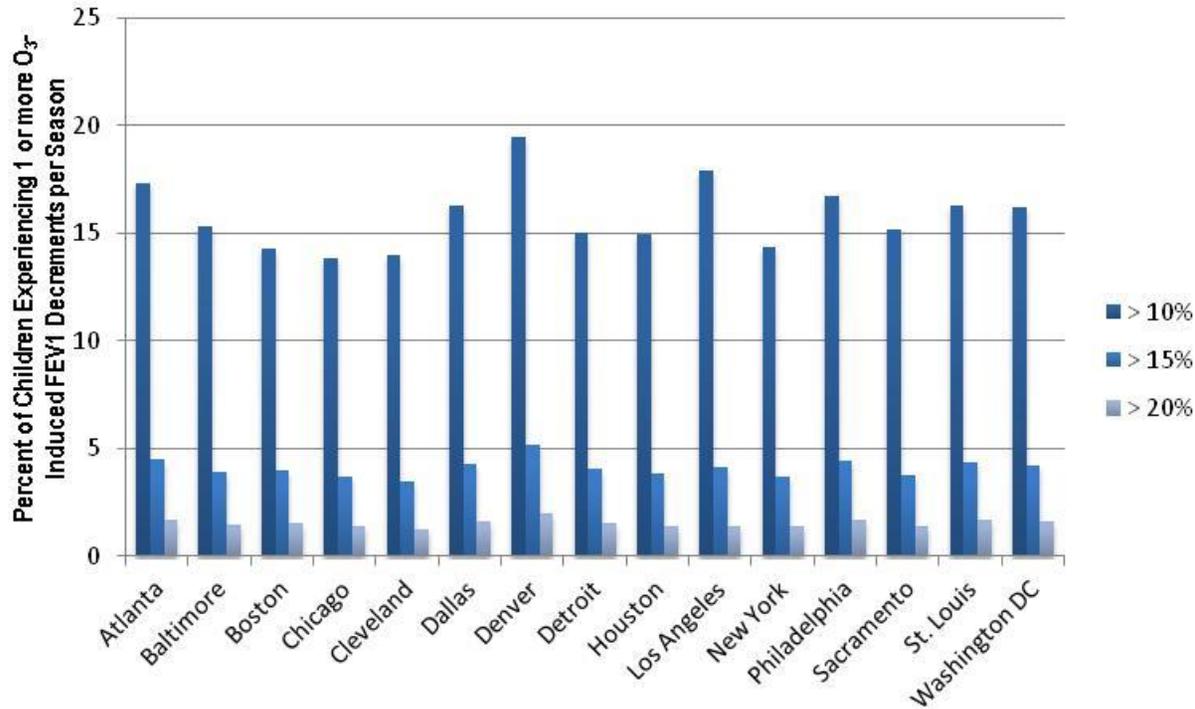
Figure 3-11. Percent of school-aged children (5-18 yrs) estimated to experience one or more days with FEV_1 decrements $\geq$ 10, 15, or 20% with air quality adjusted to just meet the current standard (Averaged over 2006 to 2010)

4

1

2
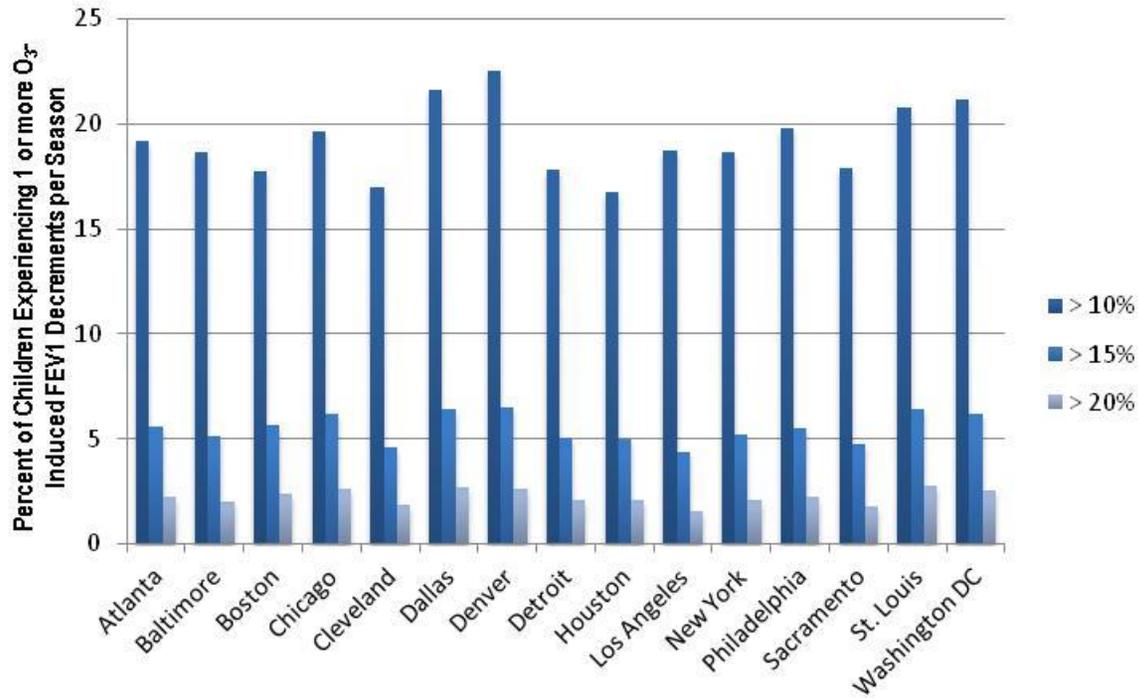
Figure 3-12. Percent of school-aged children (5-18 yrs) estimated to experience one or more days with FEV_1 decrements $\geq$ 10, 15, or 20% with air quality adjusted to just meet the current standard (Worst-Case Year from 2006 to 2010)

5

1
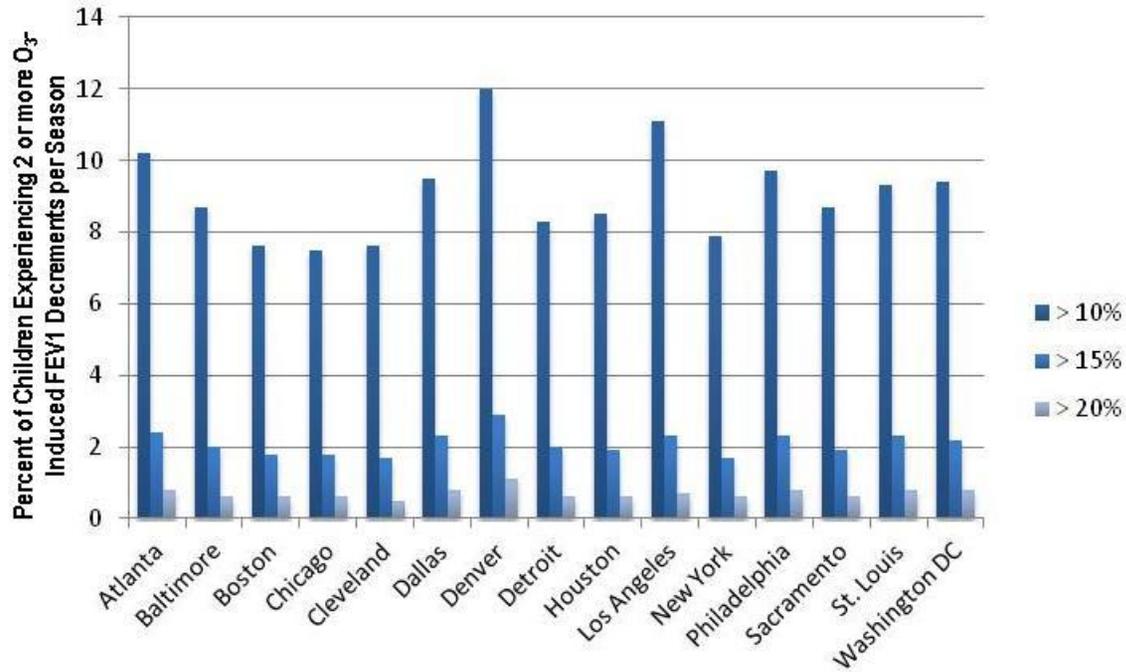
Figure 3-13. Percent of school-aged children (5-18 yrs) estimated to experience two or more days with FEV_1 decrements $\geq$ 10,
15, or 20% with air quality adjusted to just meet the current standard (Averaged over 2006 to 2010)

4

1
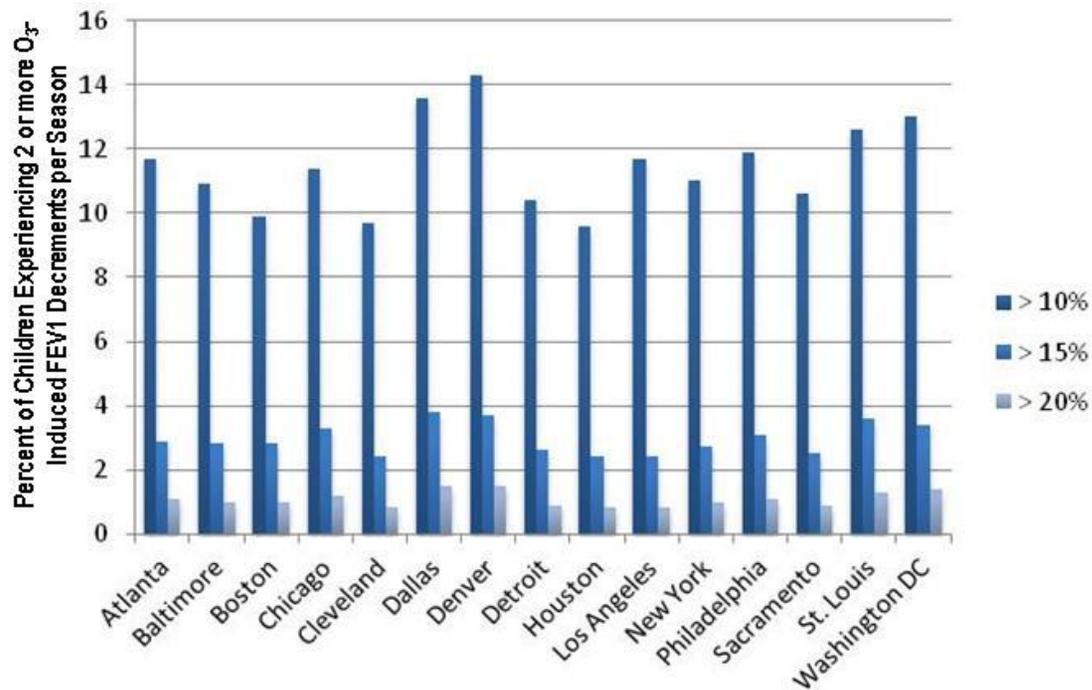
Figure 3-14. Percent of school-aged children (aged 5-18 yrs) estimated to experience two or more days with FEV$_1$ decrements $\geq$ 10, 15, or 20% with air quality adjusted to just meet the current standard (Worst-Case Year from 2006 to 2010)

1   Based on Figures 3-11 to 3-14 and the associated details described in the second draft
2 HREA (U.S. EPA 2014, Chapter 6), we take note of the following with regard to lung function
3 decrements estimated to be allowed by the current standard:

4   1. For FEV_1 decrements $\geq$ 10%:
5   a. On average over the years 2006 to 2010, the current standard is estimated to allow
6   approximately 14 to 19% of children in urban case study areas to experience one or
7   more lung function decrements $\geq$ 10%. Summing across urban case study areas, this
8   corresponds to approximately 3 million children experiencing 15 million O_3-induced
9   lung function decrements $\geq$ 10% during a single O_3 season. Of these children, about
10   300,000 are asthmatics.
11

12   b. On average over the years 2006 to 2010, the current standard is estimated to allow
13   approximately 7 to 12% of children in urban case study areas to experience two or
14   more O_3-induced lung function decrements $\geq$ 10%. Summing across the urban case
15   study areas, this corresponds to almost 2 million children (including almost 200,000
16   asthmatic children) estimated to experience two or more O_3-induced lung function
17   decrements greater than 10% during a single O_3 season.
18

19   c. In the worst-case years, the current standard is estimated to allow approximately 17 to
20   23% of children in urban case study areas to experience one or more lung function
21   decrements $\geq$ 10%, and approximately 10 to 14% to experience two or more O_3-
22   induced lung function decrements $\geq$ 10%.
23

24   2. For FEV_1 decrements $\geq$ 15%:
25   a. On average over the years 2006 to 2010, the current standard is estimated to allow
26   approximately 3 to 5% of children in urban case study areas to experience one or
27   more lung function decrements $\geq$ 15%. Summing across urban case study areas, this
28   corresponds to approximately 800,000 children (including approximately 80,000
29   asthmatic children) estimated to experience at least one O_3-induced lung function
30   decrement $\geq$ 15% during a single O_3 season.
31

32   b. On average over the years 2006 to 2010, the current standard is estimated to allow
33   approximately 2 to 3% of children in urban case study areas to experience two or
34   more O_3-induced lung function decrements $\geq$ 15%.
35

36   c. In the worst-case years, the current standard is estimated to allow approximately 4 to
37   6% of children in urban case study areas to experience one or more lung function
38   decrements $\geq$ 15%, and approximately 2 to 4% to experience two or more O_3-induced
39   lung function decrements $\geq$ 15%.
40

41   3. For FEV_1 decrements $\geq$ 20%:
42   a. On average over the years 2006 to 2010, the current standard is estimated to allow
43   approximately 1 to 2% of children in urban case study areas to experience one or
44   more lung function decrements $\geq$ 20%. Summing across urban case study areas, this
45   corresponds to approximately 300,000 children (including approximately 30,000

asthmatic children) estimated to experience at least one O_3-induced lung function decrement $\geq$ 20% during a single O_3 season.

b. On average over the years 2006 to 2010, the current standard is estimated to allow less than 1% of children in urban case study areas to experience two or more O_3-induced lung function decrements $\geq$ 20%.

c. In the worst-case years, the current standard is estimated to allow approximately 2 to 3% of children to experience one or more lung function decrements $\geq$ 20%, and less than 2% to experience two or more O_3-induced lung function decrements $\geq$ 20%.

In further considering estimated lung function risks from the 2nd draft HREA, we next consider the following question:

- **What are the important sources of uncertainty associated with lung function risk estimates?**

In addition to the uncertainties noted above for exposure estimates, the HREA identifies several key uncertainties associated with estimates of O_3-induced lung function decrements. An uncertainty with particular potential to impact our consideration of risk estimates in this Policy Assessment stems from the lack of exposure-response information in children. In the absence of controlled human exposure data for children, risk estimates are based on the assumption that children exhibit the same lung function response following O_3 exposures as healthy 18 year olds (i.e., the youngest age for which controlled human exposure data is available) (U.S. EPA, 2014, section 6.2.4 and 6.5). This assumption was justified in part by the findings of McDonnell et al. (1985), who reported that children 8-11 year old experienced FEV_1 responses similar to those observed in adults 18-35 years old. In addition, as discussed in the ISA (U.S. EPA, 2013, section 6.2.1), summer camp studies of school-aged children reported O_3-induced lung function decrements similar in magnitude to those observed in controlled human exposure studies using adults. In extending the risk model to children, the second draft HREA fixes the age term in the model at its highest value, the value for age 18. This approach could result in either over- or underestimates of O_3-induced lung function decrements in children, depending on how children compare to the adults used in controlled human exposure studies (U.S. EPA, 2014, section 6.5).

A related source of uncertainty is that the risk assessment estimates O_3-induced decrements in asthmatics using the exposure-response relationship developed from data collected from healthy individuals. Although the evidence has been mixed (U.S. EPA, 2013, section 6.2.1.1), several studies have reported larger O_3-induced lung function decrements in asthmatics than in non-asthmatics (Kreit et al., 1989; Horstman et al., 1995; Jorres et al., 1996; Alexis et al., 2000). To the extent asthmatics experience larger O_3-induced lung function decrements than the healthy adults used to develop exposure-response relationships, the second draft HREA could

1   underestimate the impacts of O_3 exposures on lung function in asthmatics, including asthmatic

2   children. The HREA notes that the magnitude this uncertainty might have on risk estimates

3   remains unknown at this time (U.S. EPA, 2014, Chapter 6).

3.2.3.2   Estimated Health Risks Associated with Short- or Long-Term O_3 Exposures, Based on Epidemiologic Studies

6   Risk estimates based on epidemiologic studies can provide perspective on the most

7   serious O_3-associated public health outcomes (e.g., mortality, hospital admissions, emergency

8   department visits) in populations that include at-risk groups. The second draft HREA estimates

9   O_3-associated risks in 12 urban case study areas[67] using concentration-response relationships

10   drawn from epidemiologic studies. These concentration-response relationships are based on

11   "area-wide" average O_3 concentrations.[68] The HREA estimates risks for the years 2007 and 2009

12   in order to provide estimates of risk for a year with generally higher O_3 concentrations (2007)

13   and a year with generally lower O_3 concentrations (2009) (U.S. EPA, 2014, section 7.2).

14   In the last review, epidemiologic-based risks were estimated for O_3 concentrations above

15   mean "policy-relevant background concentrations." As discussed above (Chapter 2), policy-

16   relevant background (PRB) concentrations were defined as the distribution of ozone

17   concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made)

18   emissions of ozone precursor emissions (e.g., VOC, CO, NOx) in the U.S., Canada, and Mexico.

19   This approach provided a focus on O_3 concentrations "that can be controlled by U.S. regulations

20   (or through international agreements with neighboring countries)" (U.S. EPA, 2007, pp. 2-48 to

21   2-54).

22   As in the last review, we recognize that ambient O_3 concentrations, and therefore O_3-

23   associated health risks, result from precursor emissions from various types of sources. Based on

24   the air quality modeling discussed above in chapter 2, approximately 30 to 60% of average

25   daytime O_3 during the warm season (i.e., 8-hour daily maximum concentrations averaged from

26   April to October) is attributable to precursor emissions from U.S. anthropogenic sources (section

27   2.4.4). This suggests that, for recent air quality (i.e., not adjusted to meet the current or

28   alternative standards), approximately 30 to 60% of total O_3-associated health risk in the urban

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[67] The 12 urban areas evaluated are Atlanta, Baltimore, Boston, Cleveland, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, and St. Louis.

[68] In the epidemiologic studies that provide the health basis for HREA risk assessments, concentration-response relationships are based on daytime O_3 concentrations, averaged across multiple monitors within study areas. These daily averages are used as surrogates for the spatial and temporal patterns of exposures in study populations. Consistent with this approach, the HREA epidemiologic-based risk estimates also utilize daytime O_3 concentrations, averaged across monitors, as surrogates for population exposures. In this second draft PA, we refer to these averaged concentrations as "area-wide" O_3 concentrations. Area-wide concentrations are discussed in more detail in section 3.1.4, above.

1 case study areas is attributable to O_3 from U.S. anthropogenic emissions. The remainder is

2 attributable to precursor emissions from international anthropogenic sources and natural sources.

3 Because the second draft HREA characterizes health risks from all O_3, regardless of source, risk

4 estimates reflect emissions from U.S. anthropogenic, international anthropogenic, and natural

5 sources. Given that HREA risk estimates for adjusted air quality are based on decreasing U.S.

6 anthropogenic precursor emissions, the contributions of U.S. anthropogenic emissions to the risk

7 estimates for the current standard would generally be smaller than the 30 to 60% indicated for

8 recent air quality.

9   In evaluating epidemiology-based risk estimates within the context of the adequacy of the

10 current standard, we first consider the following question:

11 • **What are the nature and magnitude of the O_3-associated mortality and morbidity risks**
12   **remaining upon adjustment of air quality to just meet the current O_3 standard?**

13   In addressing this question, as an initial matter we note that the area-wide average O_3

14 concentrations associated with health effects in epidemiologic studies, and used to estimate

15 mortality and morbidity risks in the HREA, are surrogates for the ambient O_3 exposures expected

16 to have elicited the reported health outcomes (also discussed in section 3.1.4.2, above). The area-

17 wide average concentrations present in epidemiologic study locations represent the spatial and

18 temporal patterns of O_3 exposures (magnitudes, frequencies, durations of exposures) experienced

19 by study populations. Differences in the patterns of O_3 exposures would be expected to result in

20 differences in the health outcome response. Thus, in considering the quantitative risk estimates

21 below we are mindful of uncertainties related to the differences between the spatial and temporal

22 patterns of O_3 that existed in the epidemiologic study areas, which contributed to the health

23 outcomes reported in these studies, and the altered patterns associated with adjusted air quality

24 that just meets the current standard (section 3.2.1, above). Among the three main types of

25 exposure/risk analyses generated in the HREA, these altered spatial/temporal patterns have the

26 greatest potential to introduce uncertainty into risk estimates based on epidemiology study

27 concentration-response relationships (see 2nd question below for further discussion).

28   We also note that the second draft HREA estimates mortality and morbidity risks

29 associated with just meeting the current standard by applying concentration-response

30 relationships from epidemiologic studies to the entire distributions of model-adjusted "area-

31 wide" average O_3 concentrations present in urban case study areas (U.S. EPA, 2014, Chapter 7).

32 Implicit in this approach to estimating risks is the assumption that concentration-response

33 relationships are linear over those distributions. Therefore, as noted in section 3.2.1, when air

34 quality is adjusted to just meet the current standard, risk estimates are influenced by the

35 decreases in area-wide O_3 concentrations at the upper ends of warm season distributions and the

1   increases in area-wide O_3 concentrations at the lower ends of those distributions (U.S. EPA,

2   2014, section 4.3.3.2, Figures 4-8 to 4-11).[69] When the decreases and increases are of the same

3   magnitude, they result in the same degree of change in estimated risks, though opposite in

4   direction. Therefore, seasonal estimates of O_3-associated mortality and morbidity risks either

5   increase or decrease in response to air quality adjustment, depending on the seasonal balance

6   between the modeled daily decreases in high area-wide O_3 concentrations and increases in low

7   area-wide O_3 concentrations. One consequence is that the estimated impacts on mortality and

8   morbidity risks of adjusting air quality to just meet the current standard are more modest, and

9   less directionally consistent across urban case study areas, than on either exposures of concern or

10   O_3-induced lung function decrements.

11   In the remainder of this section, we consider estimates of total (non-accidental) mortality

12   and respiratory morbidity associated with short-term O_3 concentrations, and respiratory mortality

13   associated with "long-term" O_3 concentrations.

Total Mortality – Short-Term O_3

15   Risk estimates for total mortality are based on concentration-response relationships

16   described by Smith et al. (2009). To generate risk estimates, the second draft HREA uses "area-

17   wide" averages of 8-hour daily maximum O_3 concentrations over the full monitoring periods in

18   urban case study areas. These monitoring periods vary across areas, in some cases including

19   more of the cooler months that are often characterized by relatively low daytime O_3

20   concentrations.[70] When air quality was adjusted to the current standard in the 2007 model year

21   (the year with generally "higher" O_3-associated risks), 10 of 12 urban case study areas exhibited

22   either small decreases or virtually no change in estimates of O_3-associated total mortality (U.S.

23   EPA, 2014, Appendix to Chapter 7).[71] Small increases in mortality were estimated in two of the

24   urban case study areas (Houston, Los Angeles) (U.S. EPA, 2014, Appendix to Chapter 7).

25   Figure 3-15 below presents estimates of O_3-associated all-cause mortality in urban case

26   study areas for 2007 and 2009, with air quality adjusted to just meet the current O_3 standard. The

27   second draft HREA estimates that upon just meeting the current standard, O_3 could be associated

28   with from 0.8 to 4.1% of all-cause mortality across the urban case study areas. This corresponds

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[69]On a given day, area-wide O_3 concentrations and estimated risks decrease when the sum of the changes at monitors with decreasing O_3 are larger than the sum of the changes at monitors with increasing O_3. Area-wide O_3 concentrations and estimated risks increase when the opposite occurs.

[70]Decreases in relatively higher ambient O_3 concentrations are more prominent during the warmest months, when daytime concentrations tend to be highest. In most urban case study areas, increases in relatively low daytime concentrations have greater influence on risk estimates during cooler months, when O_3 concentrations tend to be lower overall (U.S. EPA, 2014, compare Figures 4-8 and 4-9).

[71]Decreases were smaller in the 2009 model-adjusted year (i.e., the year with generally lower O_3 concentrations).

1   to approximately 80 to 2,800 O_3-associated deaths per season in individual urban case study

2   areas, and approximately 8,000 to 9,000 O_3-associated deaths per season summed over the 12

3   urban case study areas (U.S. EPA, 2014, Tables 7-7 and 7-8).

4
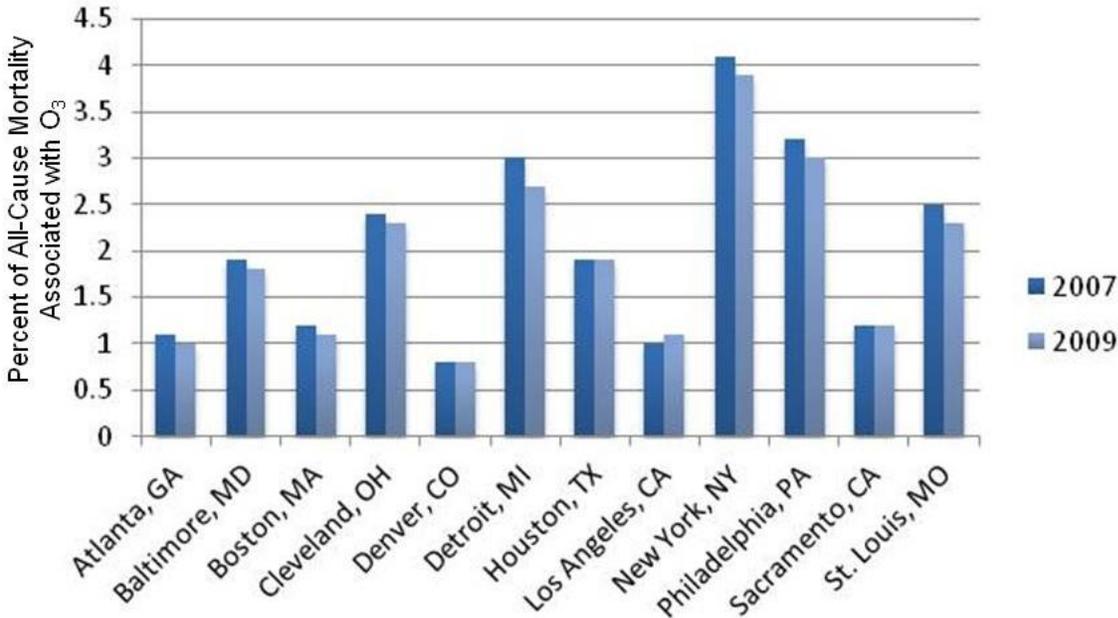
Figure 3-15. Percent of all-cause mortality associated with O_3 for air quality adjusted to
just meet the current standard.

7   In considering the risk estimates presented in Figure 3-15, which are based on applying

8   linear concentration-response relationships to the full distributions of daily 8-hour "area-wide"

9   O_3 concentrations, we note the ISA conclusion that there is less certainty in the shape of

10   concentration-response functions for area-wide O_3 concentrations at the lower ends of warm

11   season distributions (i.e., below about 20 to 40 ppb depending on the O_3 metric, health endpoint,

12   and study population) (U.S. EPA, 2013, section 2.5.4.4). We also recognize that for the range of

13   health endpoints evaluated, controlled human exposure and animal toxicological studies provide

14   greater certainty in the increased incidence, magnitude, and severity of effects at higher exposure

15   concentrations (discussed in sections 3.1.2.2 and 3.1.4.2, above).[72] Thus, in addition to

16   considering estimates of total O_3-associated risks, we also consider the extent to which risks are

17   associated with days with higher, versus lower, area-wide O_3 concentrations.

18   Figure 3-16 presents risk estimates, summed across urban case study areas, for days with

19   area-wide concentrations at or above 20, 40, and 60 ppb. Daytime O_3 concentrations in the upper

---

[72]As discussed in section 3.1.4.2, as ambient concentrations increase the potential for exposures to higher O_3 concentrations also increases. Thus with increasing ambient concentrations, controlled human exposure and animal toxicological studies provide greater certainty in the increased incidence, magnitude, and severity of O_3-attributable effects.

1   portion of the distribution of area-wide concentrations (e.g., at or above 40 or 60 ppb) are

2   estimated to be associated with hundreds to thousands of deaths per year in urban case study

3   areas.[73]
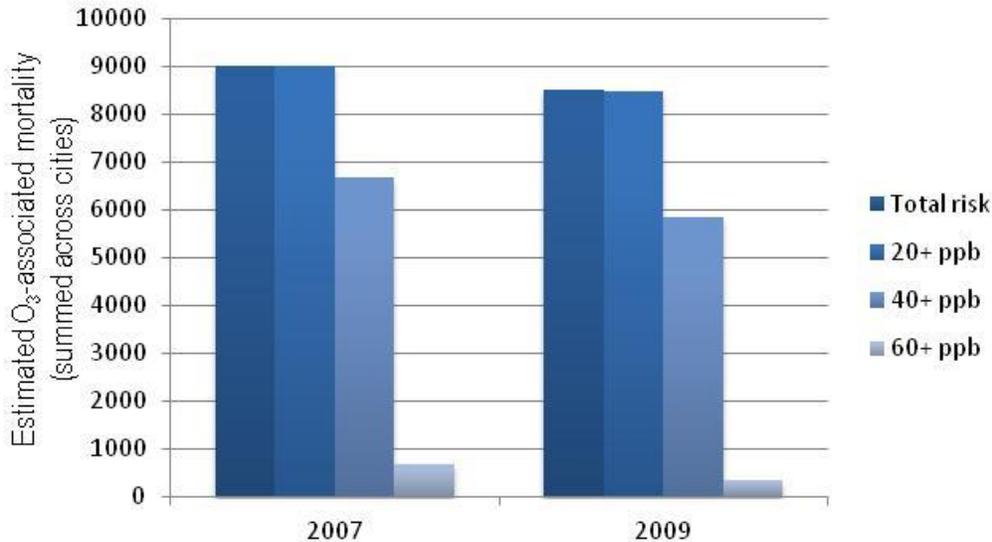
4

5   **Figure 3-16. Estimated O$_3$-associated mortality attributable to days above various area-**
6   **wide average O$_3$ concentrations, with air quality adjusted to just meet current**
7   **standard (2007 Model Adjustment Year).**

8   **Respiratory Mortality – "Long-Term" O$_3$**

9   The second draft HREA estimates the risk of respiratory mortality associated with long-

10   term O$_3$ exposures, based on the study by Jerrett et al. (2009) (U.S. EPA, 2014, Chapter 7). To

11   generate risk estimates, the second draft HREA uses "area-wide" averages of 1-hour daily

12   maximum O$_3$ concentrations during the warm season (April to September). When air quality was

13   adjusted to just meet the current standard 11 of the 12 urban case study areas exhibited modest

14   decreases in estimated O$_3$-associated respiratory mortality (i.e., compared to recent, unadjusted

15   air quality). Risk estimates remained virtually unchanged in the remaining urban case study area

16   (i.e., Los Angeles). Risk estimates for air quality adjusted to just meet the current standard are

17   presented below in Figure 3-17.[74]

18

---

[73]The relatively small proportion of O$_3$-associated deaths attributable to days with area-wide concentrations of 60 ppb or greater reflects the relatively small proportion of days with such elevated area-wide concentrations.

[74]The second draft HREA does not characterize distributions of respiratory mortality risks over distributions of ambient O$_3$ concentrations. Therefore, in considering respiratory mortality risks we evaluate only estimates of total risk.
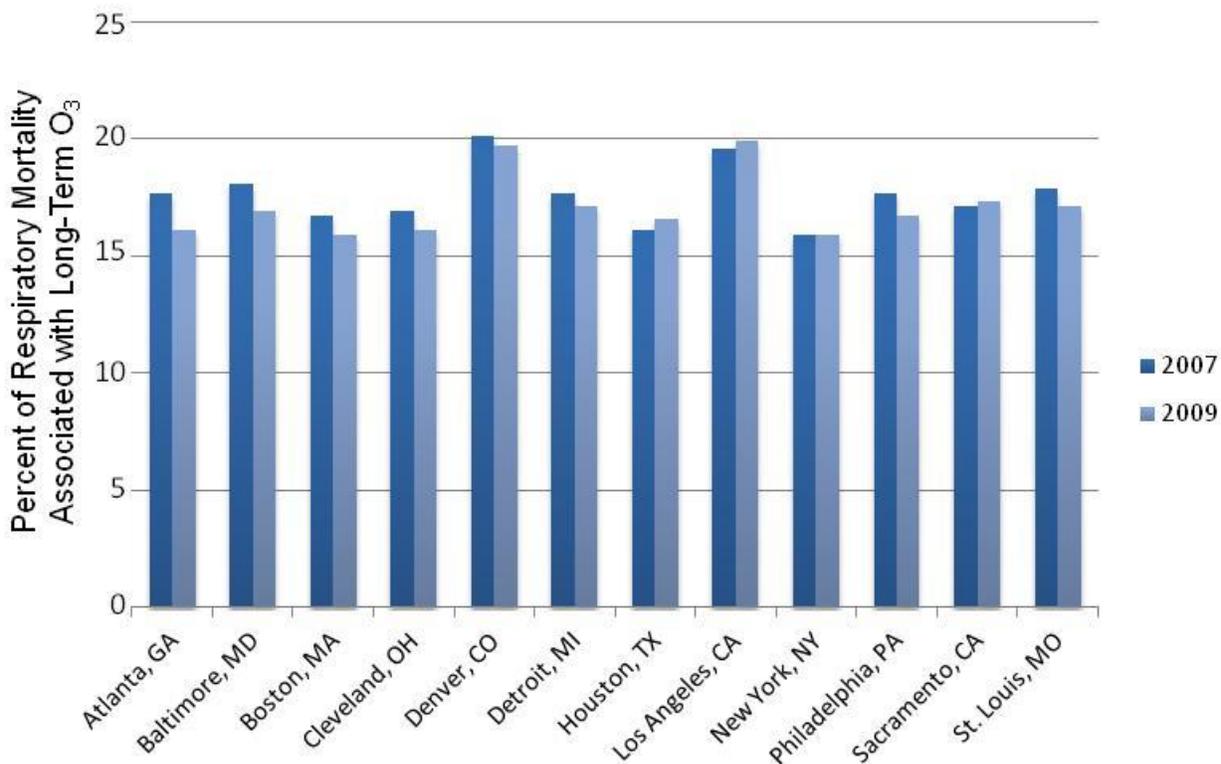
Figure 3-17. Percent of baseline respiratory mortality estimated to be associated with long-term O_3.

Across urban case study areas, O_3 is estimated to be associated with approximately 16 to 20% of respiratory mortality during the warm season. This corresponds to approximately 500 to 2,800 O_3-associated deaths per season across areas, and a total of approximately 12,000 O_3-associated deaths in all 12 urban case study areas.

Hospital Admissions, Emergency Department Visits, and Asthma Exacerbations

Risk estimates for respiratory-related hospital admissions, emergency department visits, and asthma exacerbations associated with air quality adjusted to just meet the current standard are based on several studies, as presented in Table 7-2 of the second draft HREA (U.S. EPA, 2014).[75] Estimates indicate that O_3-associated respiratory-related hospital admissions account for approximately 2 to 3% of total respiratory-related admissions in urban case study locations. Depending on the city, this corresponds to 10's to 100's of O_3-associated hospital admissions per season. Estimates indicate that O_3-associated respiratory-related emergency department visits account for approximately 3 to 20% of total respiratory-related emergency department visits in

---

[75]As with respiratory mortality above, the second draft HREA does not characterize distributions of respiratory morbidity risks over distributions of ambient O_3 concentrations. Therefore, in considering respiratory morbidity risks we evaluate estimates of total risk.

1   Atlanta (approximately 4,000 to 8,000 visits per season), and that O_3-associated asthma

2   exacerbations account for approximately 15 to 30% of total exacerbations in Boston (45,000 to

3   130,000 exacerbations per season). Full estimates are presented in Tables 7-9 to 7-11 in the

4   second draft HREA (U.S. EPA, 2014).

5   Based on Figures 3-15 to 3-17 above, and the more detailed information presented in

6   Chapter 7 of the second draft HREA (U.S. EPA, 2014), we note the following key observations:

7   1. In focusing on total risk, the current standard is estimated to allow thousands of O_3-
8   associated deaths per year in the urban case study areas. These estimates are based on
9   concentration-response functions from epidemiologic studies that used either 8-hour daily
10   O_3 concentrations (total mortality associated with short-term O_3) or seasonal averages of
11   1-hour daily O_3 concentrations (respiratory mortality associated with long-term O_3).
12
13   2. In focusing on the risks associated with the upper portions of distributions of ambient
14   concentrations, the current standard is estimated to allow hundreds to thousands of O_3-
15   associated deaths per year in the urban case study areas. These estimates are based on
16   concentration-response functions from an epidemiologic study that evaluated associations
17   between 8-hour daily O_3 concentrations and total mortality.
18
19   3. In urban case study areas, the current standard is estimated to allow tens to thousands of
20   O_3-associated morbidity events per year. Distributions of O_3-associated morbidity over
21   distributions of ambient O_3 concentrations would likely be similar to mortality, though
22   the second draft HREA did not analyze such distributions for morbidity endpoints.

23   In further considering estimated O_3-associated mortality and morbidity risks from the 2nd

24   draft HREA, we next consider the following question:

25   • **What are the important sources of uncertainty associated with mortality and morbidity**
26   **risk estimates?**

27   Upon adjusting air quality to the current standard, O_3-associated mortality and morbidity

28   risks generally decrease in locations and time periods with relatively high ambient O_3

29   concentrations and increase in locations and time periods with relatively low concentrations.

30   Therefore, an important consideration for epidemiology-based risk estimates is the extent to

31   which seasonal risk estimates in urban case study areas represent the U.S. as a whole, in terms of

32   the O_3 response to decreasing precursor emissions. To address this, the second draft HREA

33   conducted national air quality analyses evaluating the response of ambient O_3 concentrations to

34   reductions in NO_X emissions. Those analyses indicate that the 12 urban case study areas may not

35   represent the response of O_3 in other populated areas of the U.S., including suburban areas,

36   smaller urban areas, and rural areas, and that the majority of the U.S. population lives in

37   locations where reducing NO_X emissions would be expected to result in decreases in warm

1   season averages of daily maximum 8-hour ambient O_3 concentrations. One implication of this is

2   that HREA estimates for the urban case study areas are likely to understate the average reduction

3   in O_3-assocaiated mortality and morbidity risk that would be experienced across the population

4   upon reducing NO_X emissions (U.S. EPA, 2014, Chapter 8).

5   Section 7.4 of the second draft HREA also highlights some additional uncertainties

6   associated with epidemiologic-based risk estimates (U.S. EPA, 2014). This section of the HREA

7   identifies and discusses sources of uncertainty and presents a qualitative evaluation of key

8   parameters that can introduce uncertainty into risk estimates (U.S. EPA, 2014, Table 7-4). For

9   several of these parameters the HREA also presents quantitative sensitivity analyses (U.S. EPA,

10 2014, sections 7.4.2 and 7.5.3). Of the uncertainties discussed in Chapter 7 of the HREA, those

11 related to the application of concentration-response functions from epidemiologic studies can

12 have particularly important implications for our consideration of epidemiology-based risk

13 estimates in this second draft PA.

14   As noted above, an important uncertainty is the shape of concentration-response

15 functions at low ambient O_3 concentrations (U.S. EPA, 2014, Table 7-4). Consistent with the

16 ISA conclusion that there is no discernible population threshold in O_3-associated health effects,

17 the second draft HREA estimates epidemiology-based mortality and morbidity risks for entire

18 distributions of ambient O_3 concentrations, with the assumption that concentration-response

19 relationships remain linear over those distributions. In addition, in recognition of the ISA

20 conclusion that certainty in the shape of O_3 concentration-response functions decreases at low

21 ambient concentrations, the second draft HREA also estimates distributions of total mortality

22 incidence for various portions of the distribution of ambient O_3 concentrations. In this second

23 draft PA, we consider both types of risk estimates while recognizing that we have greater

24 certainty in the increased incidence and severity of O_3-attributable effects at higher ambient O_3

25 concentrations (which drive higher exposure concentrations, section 3.2.2 above), as compared

26 to lower concentrations.

27   The second draft HREA also notes important uncertainties associated with using a

28 concentration-response relationship developed for a particular population in a particular location

29 to estimate health risks in different populations and locations (U.S. EPA, 2014, Table 7-4). As

30 discussed above, concentration-response relationships derived from epidemiologic studies reflect

31 the spatial and temporal patterns of population exposures during the study. The second draft

32 HREA applies concentration-response relationships from epidemiologic studies to adjusted air

33 quality in study areas that are different from, and often larger in spatial extent than, the areas

34 used to generate the relationships. This approach ensures the inclusion of the actual non-

35 attainment monitors that often determine the magnitude of emissions reductions for the air

36 quality adjustments throughout the urban case study areas. This approach also allows the HREA

1 to estimate patterns of health risks more broadly across a larger area, including a broader range
2 of air quality concentrations and a larger population. The second draft HREA notes that it is not
3 possible to quantify the impacts of this uncertainty on risk estimates in most urban case study
4 locations, though the HREA notes that mortality effect estimates for different portions of the
5 New York City CBSA-based assessment area vary by a factor of almost 10 (U.S. EPA, 2014,
6 section 7.5.3).

7   An additional, related uncertainty is that associated with applying concentration-response
8 functions from epidemiologic studies to adjusted air quality. Concentration-response functions
9 from the O_3 epidemiologic studies used in the HREA are based on associations between day to
10 day variation in "area-wide" O_3 concentrations (i.e., averaged across multiple monitors) and
11 variation in health effects. Epidemiologic studies use these area-wide O_3 concentrations, which
12 reflect the particular spatial and temporal patterns of ambient O_3 present in study locations, as
13 surrogates for the pattern of O_3 exposures experienced by study populations. To the extent
14 adjusting O_3 concentrations to just meet the current standard results in important alterations in
15 the spatial and/or temporal patterns of ambient O_3, there is uncertainty in the appropriateness of
16 applying concentration-response functions from epidemiologic studies to estimate health risks
17 associated with adjusted O_3 air quality.[76] Although the impact of this uncertainty on risk
18 estimates cannot be quantified (U.S. EPA, 2014, Table 7-4), it has the potential to become more
19 important as model adjustment results in larger changes in spatial and temporal patterns of
20 ambient O_3 concentrations across urban case study areas.

21   There is also uncertainty related specifically to the public health importance of the
22 increases in relatively low O_3 concentrations following air quality adjustment. This uncertainty
23 relates to the fact that risk estimates are equally influenced by decreasing high concentrations
24 and increasing low concentrations, when the increases and decreases are of equal magnitude.
25 Even on days with increases in relatively low area-wide average concentrations, resulting in
26 increases in estimated risks, some portions of the urban case study areas could experience

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[76]As discussed above (section 3.2.1), decreasing modeled NO_X emissions to just meet the current standard can dramatically alter the spatial and temporal patterns of ambient O_3 concentrations across urban case study areas. Specifically, the relatively high O_3 concentrations that often occur downwind of important NO_X sources (e.g., outside urban centers) generally decrease, while the relatively low O_3 concentrations near important NO_X sources (e.g., in urban centers) generally increase (U.S. EPA, 2014, section 4.3.3.2). In addition, decreases or increases in ambient O_3 could occur more broadly across areas in some instances, depending in part on meteorological conditions. Such decreases in high O_3 concentrations and increases in low concentrations can result in compression of the spatial distributions of ambient O_3 used to calculate area-wide average concentrations. Decreases and increases can also result in compression of the temporal distributions of the area-wide O_3 concentrations used to estimate mortality and morbidity risks over a season, such that area-wide concentrations decrease on "high"-O_3 days and increase on "low"-O_3 days. As indicated in the second draft HREA (U.S. EPA, 2014, Figures 4-10 and 4-11), this compression of seasonal distributions of O_3 concentrations is evident in all of the urban case study areas evaluated, though the degree of compression varies considerably across areas. The most dramatic compression occurs in Los Angeles (U.S. EPA, 2014, Figures 4-10 and 4-11).

1 decreases in high O_3 concentrations. To the extent O_3-attributable effects are more strongly

2 supported for higher ambient concentrations, likely resulting in higher exposure concentrations

3 for some portions of study areas, the impacts on risk estimates of increasing low O_3

4 concentrations reflect an important source of uncertainty.

5   Finally, we note the second draft HREA does not quantify any reductions in risk that

6 could be associated with reductions in the ambient concentrations of pollutants other than O_3,

7 resulting from control of NO_X. For example as discussed in chapter 2 of this second draft PA,

8 NO_x emissions contribute to ambient NO_2, and NO_x and VOCs can contribute to secondary

9 formation of $PM_{2.5}$ constituents, including ammonium sulfate (NH_4SO_4), ammonium nitrate

10 (NH_4NO_3), and organic carbon (OC). Therefore, at some times and in some locations, control

11 strategies that would reduce NO_X emissions (i.e., to meet an O_3 standard) could reduce ambient

12 concentrations of NO_2 and $PM_{2.5}$, resulting in health benefits beyond those directly associated

13 with reducing ambient O_3 concentrations.[77]

14 **3.3   CASAC ADVICE**

15   Following the 2008 decision to revise the primary O_3 standard by setting the level at

16 0.075 ppm (75 ppb), CASAC strongly questioned whether the standard met the requirements of

17 the CAA, further described below. In September 2009, EPA announced its intention to

18 reconsider the 2008 standards, issuing a notice of proposed rulemaking in January 2010 (FR 75

19 2938). Soon after, EPA solicited CASAC review of that proposed rule and in January 2011

20 solicited additional advice. This proposal was based on the scientific and technical record from

21 the 2008 rulemaking, including public comments and CASAC advice and recommendations. As

22 further described in section 1.2.2 above, EPA in the fall of 2011 did not revise the standard as

23 part of the reconsideration process but decided to coordinate further proceedings on the

24 reconsideration rulemaking with this ongoing periodic review. Accordingly, in this section we

25 describe CASAC's advice related to the 2008 final decision and the subsequent reconsideration,

26 as well as its advice on the NAAQS review that was initiated in September 2008.

27   In April 2008, the members of the CASAC Ozone Review Panel sent a letter to EPA

28 stating "[I]n our most-recent letters to you on this subject—dated October 2006 and March

29 2007—the CASAC unanimously recommended selection of an 8-hour average Ozone NAAQS

30 within the range of 0.060 to 0.070 parts per million [60 to 70 ppb] for the primary (human

31 health-based) Ozone NAAQS" (Henderson, 2008). The letter continued:

32   *The CASAC now wishes to convey, by means of this letter, its additional,*

33   *unsolicited advice with regard to the primary and secondary Ozone NAAQS. In*

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[77]We expect little focus by states on controlling NO_X for purposes of controlling $PM_{2.5}$ given the more efficient
control of $PM_{2.5}$ through reduction of SO_2 and direct $PM_{2.5}$ emissions in most locations.

doing so, the participating members of the CASAC Ozone Review Panel are
unanimous in strongly urging you or your successor as EPA Administrator to
ensure that these recommendations be considered during the next review cycle for
the Ozone NAAQS that will begin next year ... numerous medical organizations
and public health groups have also expressed their support of these CASAC
recommendations' ... [The CASAC did] not endorse the new primary ozone
standard as being sufficiently protective of public health. The CASAC—as the
Agency's statutorily-established science advisory committee for advising you on
the national ambient air quality standards—unanimously recommended
decreasing the primary standard to within the range of 0.060–0.070 ppm [60 to
70 ppb]. It is the Committee's consensus scientific opinion that your decision to
set the primary ozone standard above this range fails to satisfy the explicit
stipulations of the Clean Air Act that you ensure an adequate margin of safety for
all individuals, including sensitive populations.

In response to EPA's solicitation of their advice on the Agency's proposed rulemaking as part of the reconsideration, CASAC conveyed support (Samet, 2010).

CASAC fully supports EPA's proposed range of 0.060 – 0.070 parts per million
(ppm) for the 8-hour primary ozone standard. CASAC considers this range to be
justified by the scientific evidence as presented in the Air Quality Criteria for
Ozone and Related Photochemical Oxidants (March 2006) and Review of the
National Ambient Air Quality Standards for Ozone: Policy Assessment of
Scientific and Technical Information, OAQPS Staff Paper (July 2007). As stated
in our letters of October 24, 2006, March 26, 2007 and April 7, 2008 to former
Administrator Stephen L. Johnson, CASAC unanimously recommended selection
of an 8-hour average ozone NAAQS within the range proposed by EPA (0.060 to
0.070 ppm). In proposing this range, EPA has recognized the large body of data
and risk analyses demonstrating that retention of the current standard would
leave large numbers of individuals at risk for respiratory effects and/or other
significant health impacts including asthma exacerbations, emergency room
visits, hospital admissions and mortality.

In response to EPA's request for additional advice on the reconsideration in 2011, CASAC reaffirmed their conclusion that "the evidence from controlled human and epidemiological studies strongly supports the selection of a new primary ozone standard within the 60 – 70 ppb range for an 8-hour averaging time" (Samet, 2011). As requested by EPA, CASAC's advice and recommendations were based on the scientific and technical record from the 2008 rulemaking. In considering the record for the 2008 rulemaking, CASAC stated the following to summarize the basis for their conclusions (Samet, 2011, pp. ii to iii).

- *The evidence available on dose-response for effects of ozone shows*
  associations extending to levels within the range of concentrations
  currently experienced in the United States.
- *There is scientific certainty that 6.6-hour exposures with exercise of*
  young, healthy, non-smoking adult volunteers to concentrations ≥ 80 ppb
  cause clinically relevant decrements of lung function.

- *Some healthy individuals have been shown to have clinically relevant responses, even at 60 ppb.*
- *Since the majority of clinical studies involve young, healthy adult populations, less is known about health effects in such potentially ozone sensitive populations as the elderly, children and those with cardiopulmonary disease. For these susceptible groups, decrements in lung function may be greater than in healthy volunteers and are likely to have a greater clinical significance.*
- *Children and adults with asthma are at increased risk of acute exacerbations on or shortly after days when elevated ozone concentrations occur, even when exposures do not exceed the NAAQS concentration of 75 ppb.*
- *Large segments of the population fall into what EPA terms a "sensitive population group," i.e., those at increased risk because they are more intrinsically susceptible (children, the elderly, and individuals with chronic lung disease) and those who are more vulnerable due to increased exposure because they work outside or live in areas that are more polluted than the mean levels in their communities.*

With respect to evidence from epidemiologic studies, CASAC stated "[W]hile epidemiological studies are inherently more uncertain as exposures and risk estimates decrease (due to the greater potential for biases to dominate small effect estimates), specific evidence in the literature does not suggest that our confidence on the specific attribution of the estimated effects of ozone on health outcomes differs over the proposed range of 60-70 ppb." (Samet, 2011, p.10).

In advice offered so far in the current review, which is considering an updated scientific and technical record since the 2008 rulemaking, CASAC has not yet conveyed their view on the adequacy of the current standard. In the first draft PA for the current review, staff reached the preliminary conclusion that the currently available evidence supports revising the standard to afford greater public health protection and that it does not support retention of the current standard (USEPA, 2012xx). Staff also concluded that the available evidence provides support for conducting further exposure and risk analyses of alternative standard levels in the range of 60 to 70 ppb (USEPA, 2012xx). CASAC commented the draft PA provided "a strong scientific rationale for consideration of ozone levels (8 hour averages of 60 ppb to 70 ppb)" (Frey and Samet, 2012).

3.4 PRELIMINARY STAFF CONCLUSIONS ON ADEQUACY OF PRIMARY STANDARD

This section presents staff's preliminary conclusions for the Administrator to consider in deciding whether it is appropriate to revise the existing primary O_3 standard. Our conclusions are based on consideration of the assessment and integrative synthesis of the evidence presented in

1   the ISA, including consideration of air quality distributions in locations of selected

2   epidemiologic studies; exposure and risk analyses in the second draft HREA; and the comments

3   and advice of CASAC and public comment on earlier drafts of this document, and on the ISA

4   and HREA.

5   As an initial matter, staff concludes that reducing precursor emissions to achieve O_3

6   concentrations that meet the current standard will provide important improvements in public

7   health protection. This initial conclusion is based on (1) the strong body of scientific evidence

8   indicating a wide range of adverse health outcomes attributable to exposures to O_3

9   concentrations found in the ambient air and (2) estimates indicating decreased O_3 exposures and

10   health risks upon meeting the current standard, compared to recent air quality.

11   Strong support for this initial conclusion is provided by controlled human exposure

12   studies of respiratory effects, and by quantitative estimates of exposures of concern and lung

13   function decrements based on the information in these studies. Analyses in the second draft

14   HREA estimate that the percentages of at-risk populations experiencing exposures of concern or

15   abnormal and potentially adverse lung function decrements are substantially lower for air quality

16   that just meets the current O_3 standard than for recent air quality.

17   Some support for this initial conclusion is also provided by estimates of O_3-associated

18   mortality and morbidity based on application of concentration-response relationships from

19   epidemiologic studies to adjusted air quality. These estimates are more variable than estimates of

20   O_3 exposures and O_3-induced lung function risks, and are associated with uncertainties that

21   complicate their interpretation. However, epidemiology-based risk estimates for short- and long-

22   term O_3 concentrations, in combination with the HREA's national analysis of O_3 responsiveness

23   to reductions in NO_X emissions and the larger body of health effects evidence, lead us to

24   conclude that O_3-associated mortality and morbidity would be expected to decrease following

25   reductions in O_3 precursor emissions to meet the current O_3 standard.

26   We next revisit the overarching policy question for this chapter, taking into consideration

27   the responses to specific questions focused on the adequacy of the current primary O_3 standard

28   discussed above.

29   • **Does the currently available scientific evidence and exposure/risk information, as**
30   **reflected in the ISA and HREA, support or call into question the adequacy of the**
31   **protection afforded by the current primary O_3 standard?**

32   In considering the available evidence and information, staff concludes that the O_3-

33   attributable health effects estimated to be allowed by air quality that meets the current primary

34   standard for O_3 can reasonably be judged important from a public health perspective. Thus, we

35   conclude that the available health evidence and exposure/risk information call into question the

1 adequacy of the public health protection provided by the current standard. We further conclude

2 that it is appropriate in this review to consider alternative standards that would increase public

3 health protection, compared to the current standard, and that it is not appropriate to consider

4 alternative standards with levels higher than the current standard, which would decrease public

5 health protection (see chapter 4). The basis for these conclusions is discussed below.

6   Studies evaluated since the completion of the 2006 O_3 AQCD support and expand upon

7 the strong body of evidence that, in the last review, indicated a causal relationship between short-

8 term O_3 exposures and respiratory health effects. Together, experimental and epidemiologic

9 studies support conclusions regarding a continuum of O_3 respiratory effects ranging from small

10 reversible changes in pulmonary function to more serious effects that can result in respiratory-

11 related emergency department visits, hospital admissions, and/or mortality. Recent animal

12 toxicological studies support descriptions of modes of action for these respiratory effects and

13 augment support for biological plausibility for the role of O_3 in reported effects. With regard to

14 mode of action, evidence indicates that antioxidant capacity may modify the risk of respiratory

15 morbidity associated with O_3 exposure. In addition, based on the consistency of findings across

16 studies and evidence for the coherence of results from different scientific disciplines, strong

17 evidence indicates that certain populations are at increased risk of O_3-related effects. These

18 include populations identified in previous reviews (i.e., people with asthma, children, older

19 adults, outdoor workers) and populations identified since the last review (i.e., people with certain

20 genotypes related to anti-oxidant and/or anti-inflammatory status; people with reduced intake of

21 certain nutrients, such as Vitamins C and E).

22   Evidence for adverse respiratory health effects attributable to "long-term" or repeated

23 daily O_3 exposures is much stronger than in previous reviews, and the ISA concludes there is

24 likely to be a causal relationship between such O_3 exposures and adverse respiratory health

25 effects. Uncertainties related to the extrapolation of data generated by rodent toxicology studies

26 to the understanding of health effects in humans have been reduced by studies in non-human

27 primates and by recent epidemiologic studies. The evidence available in this review includes new

28 epidemiologic studies using a variety of designs and analysis methods, conducted by different

29 research groups in different locations, evaluating the relationships between long-term O_3

30 exposures and measures of respiratory morbidity and mortality. New evidence supports

31 associations between long-term or repeated O_3 exposures and the development of asthma, with

32 several studies reporting interactions between genetic variants and such O_3 exposures. Studies

33 also report associations between long-term or repeated O_3 exposure and asthma prevalence,

34 asthma severity and control, respiratory symptoms among asthmatics, and respiratory mortality.

35   In considering the specific exposure concentrations reported to elicit respiratory effects,

36 we note that recent evidence includes controlled human exposure studies reporting lung function

1   decrements and pulmonary inflammation in healthy adults engaged in intermittent, moderate

2   exertion following 6.6 hour exposures to O_3 concentrations as low as 60 ppb, and lung function

3   decrements and respiratory symptoms following exposures to concentrations as low as 70 ppb.

4   Compared to the evidence available in the last review, these studies have strengthened support

5   for the occurrence of abnormal and potentially adverse respiratory effects following short-term

6   exposures to O_3 concentrations below 80 ppb.[78] It is reasonable to judge exposures to such O_3

7   concentrations to be potentially important from a public health perspective given the following:

8   1. The respiratory effects reported following exposures to O_3 concentrations of 60 and 70
9   ppb, while at moderate exertion, can reasonably be judged adverse based on ATS criteria
10   and past advice from CASAC.
11

12   2. The controlled human exposure studies reporting these respiratory effects were conducted
13   in healthy adults, while at-risk groups (e.g., asthmatics) could experience larger and/or
14   more serious effects.
15

16   3. These respiratory effects are coherent with the serious health outcomes that have been
17   reported in epidemiologic studies (e.g., respiratory-related hospital admissions,
18   emergency department visits, and mortality).

19   Exposure estimates from the second draft HREA for urban case study areas indicate that,

20   in areas just meeting the current O_3 standard, approximately 10 to 20% of children would

21   experience one or more exposures of concern to O_3 concentrations of 60 ppb or above. In the

22   case study areas evaluated in the HREA, this corresponds to over 2 million children experiencing

23   approximately 4 million such exposures, including over 200,000 asthmatic children. Nationally,

24   far more children would be expected to experience such exposures of concern in areas where the

25   current standard is just met. On average over the years evaluated in the HREA, approximately 3

26   to 8% of children are estimated to experience two or more exposures of concern to O_3

27   concentrations of 60 ppb or greater. For the worst-case years (i.e., years with air quality patterns

28   resulting in the highest exposure estimates), approximately 10 to 25% of children could

29   experience one or more exposures of concern at or above 60 ppb, and up to 14% could

30   experience two or more.

31   Although the current standard more effectively limits exposures of concern at or above

32   higher O_3 concentrations (i.e., 70, 80 ppb), up to about 8% of children are estimated to

33   experience exposures of concern at/above 70 ppb in the worst-case city and year (i.e., city and

34   year with the largest estimates). In the worst-case city and year, about 2% of children were

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[78] Cf. *Misisssippi*. 723 F. 3d at 262 ("Perhaps more studies like the Adams studies will yet reveal that the 0.060 ppm level produces significant adverse decrements that simply cannot be attributed to normal variation in lung function.")

1 estimated to experience two or more exposures of concern to O_3 concentrations of 70 ppb or

2 greater.

3   Though we focus on children in these analyses of O_3 exposures, we also recognize that

4 exposures to 8-hour average O_3 concentrations at or above 60, 70, or 80 ppb could be of concern

5 for some adult populations. As discussed above, the patterns of exposure estimates over years

6 and across cities are similar in adult asthmatics, older adults, and children, though smaller

7 percentages of adult populations are estimated to experience exposures of concern. Thus, the

8 results for children are one part of a broader range of potentially at-risk populations that also

9 includes asthmatic adults and older adults.

10   Consistent with estimates of exposures of concern, the second draft HREA also estimates

11 that under air quality conditions just meeting the current O_3 NAAQS, hundreds of thousands of

12 asthmatic children would be expected to experience O_3-induced lung function decrements that

13 are large enough to be potentially adverse in people with lung disease. On average over the years

14 evaluated in the HREA, the current standard is estimated to allow about 14% to 19% of children

15 in urban case study areas, including asthmatic children, to experience one or more O_3-induced

16 lung function decrements $\geq$ 10%. This corresponds to about 300,000 asthmatic children.

17 Nationally, far more children would be expected to experience such O_3-induced lung function

18 decrements. About 8% to 12% of children are estimated to experience two or more decrements $\geq$

19 10%, on average. In the worst-case years, approximately 17% to 22% of children in the urban

20 case study areas are estimated to experience one or more decrements $\geq$ 10% and about 10% to

21 14% are estimated to experience two or more such decrements. As with exposures of concern,

22 the current standard more effectively limits larger O_3-induced lung function decrements (i.e., $\geq$

23 15%, 20%). However, up to about 7% of children are estimated to experience one or more O_3-

24 induced decrements $\geq$ 15% in the worst-case city and year analyzed in the HREA (and as high as

25 about 4% for two or more decrements).

26   Recent epidemiologic studies also provide support, beyond that available in the last

27 review, for associations between short-term O_3 exposures and a wide range of adverse

28 respiratory outcomes (including respiratory-related hospital admissions, emergency department

29 visits, and mortality) and with total mortality. Associations with morbidity and mortality are

30 stronger during the warm or summer months, and remain robust after adjustment for co-

31 pollutants. In one U.S. and several Canadian studies, associations with respiratory morbidity or

32 mortality were reported in locations that would have met the current O_3 standard. Even in some

33 study locations where the current standard was not met, considering reported concentration-

34 response functions or cut-point analyses in the context of available air quality data indicate the

35 existence of O_3-health effect associations on the subsets of days with ambient O_3 concentrations

36 below 75 ppb. Taken together, these studies and associated air quality data indicate a relatively

1   high degree of confidence in the occurrence of O_3-associated hospital admissions, emergency

2   department visits, and mortality at ambient concentrations that meet the current standard.

3   The HREA epidemiology-based risk estimates in 12 urban cases study areas indicate

4   thousands of O_3-associated hospital admissions, emergency department visits, and deaths per

5   year for air quality conditions associated with meeting the current standard. Based on area-wide

6   O_3 concentrations from the upper portions of seasonal distributions, a focus that we judge

7   appropriate given the greater certainty in O_3-attributable effects at higher concentrations,

8   hundreds to thousands of O_3-associated deaths per year are estimated for air quality associated

9   with the current standard in urban case study areas, indicating the potential for substantial public

10   health risk. As recognized above in section 3.2.3.2, we note greater uncertainty in O_3-attributable

11   effects at lower concentrations, which are subject to increases upon air quality adjustment.

12   Although there are additional uncertainties in quantifying risks by applying concentration-

13   response functions from epidemiology studies to adjusted O_3 air quality, the general magnitude

14   of risk estimates suggests the potential for a substantial number of O_3-associated deaths and

15   adverse respiratory events nationally when the current standard is met.

16   In addition to the evidence and exposure/risk information discussed above, we also take

17   note of the CASAC advice provided to the EPA Administrator on the proposed reconsideration

18   of the 2008 decision establishing the current standard and the advice of the CASAC O_3 Panel

19   thus far in the current review. In commenting on the proposed reconsideration, the prior CASAC

20   O_3 Panel emphatically recommended revision of the standard to one with a lower level based

21   entirely on the evidence and information in the record for the 2008 standard, which has been

22   substantially strengthened in the current review (Samet, 2011; Samet, 2012). Based on review of

23   the first draft PA in the current review, the current CASAC O_3 Panel also described the draft PA

24   as providing strong scientific rationale for consideration of lower standard levels (Frey and

25   Samet, 2012).

26   In consideration of all of the above, staff reaches the preliminary conclusion that the

27   available evidence and exposure and risk information clearly calls into question the adequacy of

28   public health protection provided by the current primary standard. This evidence and information

29   provides strong support for the occurrence of a range of adverse respiratory effects, and

30   mortality, under air quality conditions that would meet the current standard. Based on the

31   analyses in the second draft HREA, we conclude that the exposures and risks projected to remain

32   upon meeting the current standard are indicative of risks that can reasonably be judged to be

33   important from a public health perspective. Thus, staff concludes that the evidence and

34   information provides strong support for giving consideration to revising the current primary

35   standard in order to provide increased public health protection against an array of adverse health

36   effects that range from decreased lung function and respiratory symptoms to more serious

1   indicators of morbidity (e.g., including emergency department visits and hospital admissions),

2   and mortality. We further conclude that it is not appropriate to consider alternative standards

3   with levels higher than the current standard, which would decrease public health protection. In

4   consideration of all of the above, staff draws the preliminary conclusion that it is appropriate for

5   the Administrator to consider revision of the current primary O_3 standard to provide increased

6   public health protection.

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4 CONSIDERATION OF ALTERNATIVE PRIMARY STANDARDS

Having reached the conclusion that the currently available scientific evidence and exposure/risk information calls into question the adequacy of the current O_3 standard, we next consider the following overarching question:

- **What is the range of potential alternative standards that are supported by the currently available scientific evidence and exposure/risk information, as reflected in the ISA and HREA respectively?**

To address this overarching question, in the sections below we evaluate a series of more specific questions related to the major elements of the NAAQS: indicator (section 4.1), averaging time (section 4.2), form (section 4.3), and level (section 4.4). In addressing these questions, we consider the currently available scientific evidence and exposure/risk information, including the evidence and information available at the time of the last review and that newly available in the current review, as assessed in the ISA and the second draft HREA. In so doing, we note that the final decision by the Administrator in this review will consider these elements collectively in evaluating the health protection afforded by the primary standard.[1]

4.1 INDICATOR

In the last review, EPA focused on O_3 as the most appropriate indicator for a standard meant to provide protection against ambient photochemical oxidants. In this review, while the complex atmospheric chemistry in which O_3 plays a key role has been highlighted, no alternatives to O_3 have been advanced as being a more appropriate indicator for ambient photochemical oxidants. More specifically, the ISA noted that O_3 is the only photochemical oxidant (other than NO_2) that is routinely monitored and for which a comprehensive database exists (ISA section 3.6). Data for other photochemical oxidants (e.g., PAN, H_2O_2, etc.) typically have been obtained only as part of special field studies. Consequently, no data on nationwide patterns of occurrence are available for these other oxidants; nor are extensive data available on the relationships of concentrations and patterns of these oxidants to those of O_3 (ISA section 3.6).

We further note that meeting an O_3 standard can be expected to provide some degree of protection against potential health effects that may be independently associated with other photochemical oxidants, even though such effects are not discernible from currently available studies indexed by O_3 alone. That is, since the precursor emissions that lead to the formation of O_3 generally also lead to the formation of other photochemical oxidants, measures leading to

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[1] We also take note of the 1997 review (discussed in section 1.3.1.2.3), in which O_3 background concentrations were an additional consideration in selecting a standard. Background O_3 is discussed in more detail in chapter 2 of this second draft PA.

1   reductions in population exposures to O_3 can generally be expected to lead to reductions in

2   population exposures to other photochemical oxidants. Taken together, we conclude that O_3

3   remains the most appropriate indicator for a standard meant to provide protection against

4   photochemical oxidants.[2]

5   4.2   AVERAGING TIME

6   The EPA established the current 8-hour averaging time[3] for the primary O_3 NAAQS in

7   1997 (62 FR 38856). The decision on averaging time in that review was based on numerous

8   controlled human exposure and epidemiologic studies reporting associations between 6 to 8 hour

9   O_3 concentrations and adverse respiratory effects (62 FR 38861). It was also noted that a

10   standard with a max 8-hour averaging time is likely to provide substantial protection against

11   respiratory effects associated with 1-hour peak O_3 concentrations. Similar conclusions were

12   reached in the last O_3 NAAQS review and thus, the 8-hour averaging time was retained in 2008.

13   In the current review, we first consider the following question related to averaging time:

14   • **To what extent does the available evidence continue to support the**
15   **appropriateness of a standard with an 8-hour averaging time?**

16   In reaching conclusions related to this question, staff considers causality judgments from the

17   ISA, as well as results from the specific controlled human exposure and epidemiologic studies

18   that informed those judgments. These considerations are described below in more detail.

19   As an initial consideration with respect to the most appropriate averaging time for the O_3

20   NAAQS, we note that the strongest evidence for O_3-associated health effects is for respiratory

21   effects following short-term exposures. More specifically, the ISA concludes that evidence

22   relating short-term O_3 exposures to respiratory effects is "sufficient to infer a causal

23   relationship." The ISA also judges that short-term exposures to O_3 are "likely to cause" both

24   cardiovascular effects and mortality (U.S. EPA, 2013, section 2.5.2). Therefore, as in past

25   reviews, the strength of the available scientific evidence provides strong support for a standard

26   that protects the public health against short-term exposures to O_3.

27   In first considering the level of support available for specific short-term averaging times,

28   we note the evidence available from controlled human exposure studies. As discussed in more

29   detail in chapter 3 of this second draft PA, substantial health effects evidence from controlled

30   human exposure studies demonstrates that a wide range of respiratory effects (e.g., pulmonary

31   function decrements, increases in respiratory symptoms, lung inflammation, lung permeability,

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[2]The D.C. Circuit upheld the use of O_3 as the indicator for photochemical oxidants based on these same considerations. *American Petroleum Inst. v. Costle*, 665 F. 2d 1176, 1186 (D.C. Cir. 1981).

[3]This 8-hour averaging time reflects daily max 8-hour average O_3 concentrations.

1   decreased lung host defense, and airway hyperresponsiveness) occur in healthy adults following
2   6.6 hour exposures to O_3 (EPA 2013, section 6.2.1.1). Compared to shorter exposure durations
3   (e.g., 1-hour), studies evaluating 6.6 hour exposures in healthy adults have reported respiratory
4   effects at lower O_3 exposure concentrations and at more moderate levels of exertion.

5   We also note the strength of evidence from epidemiologic studies that have evaluated a
6   wide variety of populations (e.g., including at-risk lifestages and populations, such as children
7   and people with asthma, respectively). A number of different averaging times are used in O_3
8   epidemiologic studies, with the most common being the max 1-hour concentration within a 24-
9   hour period (1-hour max), the max 8-hour average concentration within a 24-hour period (8-hr
10   max), and the 24-hour average. These studies are discussed in chapter 3 of this second draft PA,
11   and are assessed in detail in chapter 6 of the ISA (U.S. EPA, 2013). Limited evidence from time-
12   series and panel epidemiologic studies comparing risk estimates across averaging times does not
13   indicate that one exposure metric is more consistently or strongly associated with respiratory
14   health effects or mortality, though the ISA notes some evidence for "smaller O_3 risk estimates
15   when using a 24-hour average exposure metric" (EPA 2013, section 2.5.4.2; p. 2-31). For single-
16   and multi-day average O_3 concentrations, lung function decrements were associated with 1-hour
17   max, 8-hour max, and 24-hour average ambient O_3 concentrations, with no strong difference in
18   the consistency or magnitude of association among the averaging times (EPA 2013, p. 6-71).
19   Similarly, in studies of short-term exposure to O_3 and mortality, Smith et al. (2009) and Darrow
20   et al. (2011) have reported high correlations between risk estimates calculated using 24-hour
21   average, 8-hour max, and 1-hour max averaging times (EPA 2013, p. 6-253). Thus, the
22   epidemiologic evidence alone does not provide a strong basis for distinguishing between the
23   appropriateness of 1-hour, 8-hour, and 24-hour averaging times.

24   Considering the health information discussed above, we conclude that an 8-hour
25   averaging time remains appropriate for addressing health effects associated with short-term
26   exposures to ambient O_3. An 8-hour averaging time is similar to the exposure periods evaluated
27   in controlled human exposure studies, including recent studies that provide evidence for
28   respiratory effects following exposures to O_3 concentrations below the level of the current
29   standard. In addition, epidemiologic studies provide evidence for health effect associations with
30   8-hour O_3 concentrations, as well as with 1-hour and 24-hour concentrations. As in previous
31   reviews, we note that a standard with an 8-hour averaging time (combined with an appropriate
32   standard form and level) would also be expected to provide substantial protection against health
33   effects attributable to 1-hour and 24-hour exposures (e.g., 62 FR 38861, July 18, 1997).

34   The ISA also concludes that long-term O_3 exposures are "likely to cause" respiratory
35   effects (US EPA, 2013, chapter 7). Thus, in this review we also consider the extent to which
36   currently available evidence and exposure/risk information suggests that a standard with an 8-

1   hour averaging time can provide protection against respiratory effects associated with longer
2   term exposures to ambient O_3. In doing so, staff considers the following question:

3   - **To what extent does the available evidence and exposure/risk information indicate**
4   **that a standard with the current 8-hour averaging time could provide protection**
5   **against long-term exposures to ambient O_3?**

6   In considering this issue in the last review of the O_3 NAAQS, staff noted that "because long-term
7   air quality patterns would be improved in areas coming into attainment with an 8-hr standard, the
8   potential risk of health effects associated with long-term exposures would be reduced in any area
9   meeting an 8-hr standard" (U.S. EPA, 2007, p. 6-57).
10   In the current review, we further evaluate this issue, with a focus on the "long-term" O_3
11   metrics reported to be associated with mortality or morbidity in recent epidemiologic studies. As
12   discussed in section 3.1.3, much of the recent evidence for such associations is based on studies
13   that defined long-term O_3 in terms of seasonal averages of daily max concentrations (e.g.,
14   seasonal averages of 1-hour or 8-hour daily max concentrations).
15   As an initial consideration, we note the risk results from the second draft HREA for
16   respiratory mortality associated with long-term O_3 concentrations. As discussed in section
17   3.2.3.2, HREA analyses indicate that as air quality is adjusted to just meet the current 8-hour
18   standard, most urban case study areas are estimated to experience reductions in respiratory
19   mortality associated with long-term O_3 concentrations based on the seasonal averages of 1-hour
20   daily max O_3 concentrations evaluated in the study by Jerrett et al. (2009) (U.S. EPA, 2014,
21   chapter 7). As air quality is adjusted to meet lower potential alternative standard levels, for
22   standards based on 3-year averages of the annual fourth-highest daily max 8-hour O_3
23   concentrations, respiratory mortality risks are estimated to be reduced further in urban case study
24   areas (section 4.4.2.3, below). This analysis indicates that an O_3 standard with an 8-hour
25   averaging time, when coupled with an appropriate form and level, can reduce respiratory
26   mortality reported to be associated with "long-term" O_3 concentrations.
27   In further considering the study by Jerrett et al. (2009), we compare long-term O_3
28   concentrations following model adjustment in urban case study areas (i.e., adjusted to meet the
29   current and potential alternative 8-hour standards) to the concentrations present in study cities
30   that provided the basis for the positive and statistically significant association with respiratory
31   mortality. As indicated below (Table 4-3), this comparison suggests that a standard with an 8-
32   hour averaging time can decrease seasonal averages of 1-hour daily max O_3 concentrations, and
33   can maintain those O_3 concentrations below the seasonal average where we have the most
34   confidence in the reported concentration-response relationship with respiratory mortality (see
35   section 4.4.1 for further discussion).

1   The second draft HREA also conducted analyses evaluating the impacts of reducing

2 regional NO_X emissions on the seasonal averages of 8-hour daily max O_3 concentrations.[4]

3 Seasonal averages of 8-hour daily max O_3 concentrations reflect long-term metrics that have

4 been reported to be associated with respiratory morbidity effects in several recent O_3

5 epidemiologic studies (e.g., Islam et al., 2008; Lin et al., 2008; Salam et al., 2009). The HREA

6 analyses indicate that the large majority of the U.S. population lives in locations where reducing

7 NO_X emissions would be expected to result in decreases in seasonal averages of daily max 8-

8 hour ambient O_3 concentrations (U.S. EPA, 2014, chapter 8). Thus, consistent with the

9 respiratory mortality risk estimates noted above, this analysis suggests that reductions in O_3

10 precursor emissions in order to meet a standard with an 8-hour averaging time would also be

11 expected to reduce the types of long-term O_3 concentrations that have been reported in recent

12 epidemiologic studies to be associated with respiratory morbidity.

13   Taken together, we conclude that a standard with an 8-hour averaging time, coupled with

14 the current 4th high form and an appropriate level, would be expected to provide appropriate

15 protection against the long-term O_3 concentrations that have been reported to be associated with

16 respiratory morbidity and mortality. This issue is considered further, within the context of

17 specific potential alternative standard levels, in section 4.4 below.

18 **4.3   FORM**

19   The "form" of a standard defines the air quality statistic that is to be compared to the

20 level of the standard in determining whether an area attains the standard. The foremost

21 consideration in selecting a form for potential alternative primary standards is the adequacy of

22 the public health protection provided by the combination of the form and the other elements of

23 the standard. As such, in reaching staff conclusions regarding the appropriate form(s) to consider

24 for a potential alternative primary O_3 standard, we consider the following question:

25   • **To what extent do the available evidence and/or information continue to support**
26   **the appropriateness of a standard with a form defined by the 3-year average of**
27   **annual 4th-highest 8-hour daily max O_3 concentrations?**

28   The EPA established the current form of the primary O_3 NAAQS in 1997 (62 FR 38856).

29 Prior to that time, the standard had a "1-expected-exceedance" form.[5] An advantage of the

30 current concentration-based form recognized in the 1997 review is that such a form better

31 reflects the continuum of health effects associated with increasing ambient O_3 concentrations.

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[4]Analyses are based on regional NO_X reductions, which are effective in bringing down peak ambient O_3 concentrations, but can have variable impacts on seasonal mean concentrations.

[5]For a standard with a 1-expected-exceedance form to be met at an air quality monitoring site, the fourth-highest air quality value in 3 years, given adjustments for missing data, must be less than or equal to the level of the standard.

1 Unlike an expected exceedance form, a concentration-based form gives proportionally more
2 weight to years when 8-hour O_3 concentrations are well above the level of the standard than to
3 years when 8-hour O_3 concentrations are just above the level of the standard. It was judged
4 appropriate to give more weight to higher O_3 concentrations, given that available health evidence
5 indicated a continuum of effects associated with exposures to varying concentrations of O_3, and
6 given that the extent to which public health is affected by exposure to ambient O_3 is related to the
7 actual magnitude of the O_3 concentration, not just whether the concentration is above a specified
8 level.
9   During the 1997 review, EPA considered a range of alternative "concentration-based"
10 forms, including the second-, third-, fourth- and fifth-highest daily max 8-hour concentrations in
11 an O_3 season. The fourth-highest daily max was selected, recognizing that a less restrictive form
12 (e.g., fifth highest) would allow a relatively large percentage of sites to experience O_3 peaks well
13 above the level of the standard, and would allow more days on which the level of the standard
14 may be exceeded when attaining the standard (62 FR 38856). Consideration was also given to
15 setting a standard with a form that would provide a margin of safety against possible but
16 uncertain chronic effects, and would provide greater stability to ongoing control programs.[6] A
17 more restrictive form was not selected, recognizing that the differences in the degree of
18 protection afforded by the alternatives were not well enough understood to use any such
19 differences as a basis for choosing the most restrictive forms (62 FR 38856).
20   In the 2008 review, EPA additionally considered the potential value of a percentile-based
21 form. In doing so, EPA recognized that such a statistic is useful for comparing datasets of
22 varying length because it samples approximately the same place in the distribution of air quality
23 values, whether the dataset is several months or several years long. However, EPA concluded
24 that a percentile-based statistic would not be effective in ensuring the same degree of public
25 health protection across the country. Specifically, a percentile-based form would allow more
26 days with higher air quality values in locations with longer O_3 seasons relative to places with
27 shorter O_3 seasons.
28   Thus, in the 2008 review EPA concluded that a form based on the nth-highest max O_3
29 concentration would more effectively ensure that people who live in areas with different length
30 O_3 seasons receive the same degree of public health protection. Based on analyses for forms
31 specified in terms of an nth-highest concentration (n ranged from 3 to 5), advice from CASAC,

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[6] See American Trucking Assn's v. EPA, 283 F. 3d 355, 374-75 (D.C. Cir. 2002) (less stable implementation programs may be less effective, and therefore EPA can consider programmatic stability in determining the form of a NAAQS).

1 and public comment,[7] the Administrator concluded that a 4th-highest daily max should be

2 retained (73 FR 16465). In reaching this decision, the Administrator recognized that "the

3 adequacy of the public health protection provided by the combination of the level and form is a

4 foremost consideration" (73 FR 16475).

5   The Administrator also recognized that it is important to have a form that provides

6 stability with regard to implementation of the standard. In the case of O_3, for example, he noted

7 the importance of a form insulated from the impacts of the meteorological events that are

8 conducive to O_3 formation. Such events could have the effect of reducing public health

9 protection, to the extent they result in frequent shifts between meeting and violating the standard

10 due to meteorological conditions. The Administrator noted that such frequent shifting could

11 disrupt an area's ongoing implementation plans and associated control programs (73 FR 16474).

12 In its notice of proposed rulemaking to reconsider the 2008 standard, the EPA did not propose to

13 reconsider the form of the standard.

14   In the current review, we consider the extent to which newly available information

15 provides support for consideration of alternative forms. In so doing, we take note of the

16 conclusions of prior reviews summarized above. We recognize the value of an nth-high statistic

17 over that of an expected exceedance or percentile-based form in the case of the O_3 standard, for

18 the reasons summarized above. We additionally take note of the importance of stability in

19 implementation to achieving the level of protection specified by the NAAQS. Specifically, we

20 note that to the extent that areas engaged in implementing the O_3 NAAQS frequently shift from

21 meeting to violating the standard, it is possible that ongoing implementation plans and associated

22 control programs could be disrupted, thereby reducing public health protection.

23   In light of this, while giving foremost consideration to the adequacy of public health

24 protection provided by the combination of all elements of the standard, including the form, we

25 consider particularly findings from prior reviews with regard to the use of the nth-high metric.

26 As noted above, the 4th-highest daily max was selected in 1997 in recognition of the public

27 health protection provided by this form, when coupled with an appropriate averaging time and

28 level, and recognizing that such a form can provide stability for implementation programs. The

29 currently available evidence and information does not call into question these conclusions from

30 previous reviews. Therefore, we conclude that it would be appropriate to retain the current 4th-

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[7] In the 2008 review, one group of commenters expressed the view that the standard was not adequate and supported a more health-protective form (e.g., a second- or third-highest daily max form). Another group of commenters expressed the view that the standard was adequate and did not provide any views on alternative forms that would be appropriate should the Administrator consider revisions to the standard. The Administrator considered the protection afforded by the combination of level and form in revising the standard in 2008 to 75 ppb, as a 3-year average of the annual fourth-highest daily max 8-hour concentrations (73 FR 16475).

1   highest daily max form for an O_3 standard with an 8-hour averaging time and a revised level, as

2   discussed below.

3   **4.4   LEVEL**

4   In considering potential alternative standards levels to provide greater protection than that

5   afforded by the current standard against O_3-related adverse health effects, we address the

6   following overarching question.

7   • **For an O_3 standard defined in terms of the current indicator, averaging time, and**
8   **form, what alternative levels are appropriate to consider in order to provide**
9   **adequate public health protection against short- and long- term exposures to O_3**
10   **in ambient air?**

11   In considering this question, we take into account the experimental and epidemiologic evidence

12   as presented in the ISA, as well as the uncertainties and limitations associated with this evidence

13   (section 4.4.1). In addition, we consider the quantitative estimates of exposure and risk provided

14   by the HREA, as well as the uncertainties and limitations associated with these risk estimates

15   (section 4.4.2).

16   **4.4.1   Evidence-based Considerations**

17   In this section, we consider the available evidence from controlled human exposure and

18   epidemiologic studies, including the uncertainties and limitations associated with that evidence,

19   within the context of potential alternative standard levels. We consider both the exposure

20   concentrations at which controlled human exposure studies provide evidence for health effects,

21   and the ambient O_3 concentrations present in locations where epidemiologic studies have

22   reported health effect associations (see also section 3.1).

23   *Controlled human exposure studies*

24   We consider the following question related to controlled human exposure studies:

25   • **To what extent does the available evidence from controlled human exposure**
26   **studies provide support for consideration of potential alternative standard levels**
27   **lower than 75 ppb?**

28   To inform our conclusions regarding this question, we consider the lowest O_3 concentrations at

29   which various effects have been evaluated and statistically significant effects reported. We also

30   consider the potential for reported effects to be adverse, including in at-risk populations.

31   As discussed in section 3.1.2.1, in healthy adults group mean O_3-induced lung function

32   decrements exhibit a smooth dose-response relationship without evidence of a threshold from 40

33   to 120 ppb O_3 (US EPA, 2013, Figure 6-1). The lowest O_3 exposure concentration for which

34   statistically significant decrements have been reported is 60 ppb (Brown, 2006; Kim et al., 2011).

1 The ISA concludes that mean FEV_1 is clearly decreased by 6.6-hour exposures to O_3

2 concentrations of 60 ppb and higher in young, healthy adults during moderate exertion (US EPA,

3 2013, p. 6-9). As discussed in section 3.1.3, such a decrease in mean lung function meets the

4 ATS criteria for an adverse response given that a downward shift in the distribution of FEV_1

5 would result in diminished reserve function, and therefore would increase risk from further

6 environmental insult. In addition, the ISA notes that following exposures to 60 ppb O_3 10% of

7 healthy individuals experience FEV_1 decrements > 10% (U.S. EPA, 2013, page 6-19). A 10%

8 decrement in FEV_1 is accepted by ATS as an abnormal response, and based on advice received

9 from CASAC in previous reviews, such decrements could be adverse in people with lung disease

10 (section 3.1.3).

11   As discussed in section 3.1.2.1, one recent controlled human exposure study has reported

12 O_3-induced pulmonary inflammation (PMN influx to the ELF) following exposures of young,

13 healthy adults to O_3 concentrations of 60 ppb (Kim et al., 2011), the lowest concentration at

14 which inflammatory responses have been evaluated in human studies. Induction of pulmonary

15 inflammation is evidence that injury has occurred. The possibility of chronic effects due to

16 repeated inflammatory events has been evaluated in animal studies. Repeated events of acute

17 inflammation can have several potentially adverse outcomes including: induction of a chronic

18 inflammatory state; altered pulmonary structure and function, leading to diseases such as asthma;

19 altered lung host defense response to inhaled microorganisms, particularly in potentially at-risk

20 populations such as the very young and old; and, altered lung response to other agents such as

21 allergens or toxins (U.S. EPA, 2013, Section 6.2.3). Thus, lung injury and the resulting

22 inflammation, particularly if experienced repeatedly, provide a mechanism by which O_3 may

23 cause other more serious respiratory effects (e.g., asthma exacerbations) and possibly

24 extrapulmonary effects.

25   With respect to respiratory symptoms, a recent study by Schelegle et al. (2009) reported a

26 statistically significant increase in respiratory symptoms in young, healthy adults following 6.6

27 hour exposures to an average O_3 concentration of 70 ppb. This study also reported a statistically

28 significant decrease in FEV_1 following such exposures. As discussed in section 3.1.3, the

29 occurrence of both lung function decrements and respiratory symptoms meets criteria established

30 by the ATS defining an "adverse" respiratory response. Although some studies have reported

31 that respiratory symptoms develop during exposures at 60 ppb, the increases in symptoms in

32 these studies have not reached statistical significance by the end of the 6.6 hr exposures (Adams

33 2006; Schelegle et al., 2009).[8]

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[8]Adams (2006) reported an increase in respiratory symptoms in healthy adults during a 6.6 hour exposure protocol
with an average O_3 exposure concentration of 60 ppb. This increase was significantly different from initial
respiratory symptoms, but not from filtered air controls.

1   Based on the results discussed above and in section 3.1.2.1, we conclude that controlled
2   human exposure studies provide evidence of potentially adverse lung function decrements and
3   airway inflammation in healthy individuals following exposures to 60 ppb O_3, and evidence of
4   respiratory symptoms combined with lung function decrements (an "adverse" response based on
5   ATS criteria) following exposures to 70 ppb. In reaching these conclusions, we recognize that
6   most studies have not evaluated exposure concentrations below 60 ppb, and that 60 ppb does not
7   necessarily reflect an exposure concentration below which effects no longer occur. Specifically,
8   given the occurrence of airway inflammation following exposures to 60 ppb and higher, it may
9   be reasonable to expect that inflammation would also occur following exposures to O_3
10   concentrations somewhat below 60 ppb. Although some studies show that respiratory symptoms
11   develop during exposures at 60 ppb, they have not reached statistical significance by the end of
12   the 6.6 hr exposures (Adams 2006; Schelegle et al. 2009). Thus, respiratory symptoms combined
13   with lung function decrements are likely to occur to some degree in healthy individuals with 6.6-
14   hr exposures to concentrations below 70 ppb. Further, we note that these controlled human
15   exposure studies were conducted in healthy adults and that people with asthma, including
16   asthmatic children, are likely to be more sensitive to O_3-induced respiratory effects. Therefore,
17   these exposure concentrations are more likely to cause adverse respiratory effects in children and
18   adults with asthma, and more generally in people with respiratory disease.
19   In further considering effects following exposures to O_3 concentrations below 75 ppb, in
20   section 3.1.4.1 we discuss panel studies highlighted in the ISA for the extent to which monitored
21   ambient O_3 concentrations reflect exposure concentrations in their study populations (U.S. EPA,
22   2013, section 6.2.1.2). These panel studies used on-site monitoring to evaluate O_3-attributable
23   lung function decrements in individuals engaged in outdoor recreation, exercise, or work. Table
24   3-2 includes O_3 panel studies that report analyses of O_3-attributable lung function decrements for
25   O_3 concentrations at or below 75 ppb, and that measure O_3 concentrations with monitors located
26   in the areas where study subjects were active (e.g., on site at summer camps or in locations
27   where exercise took place). Consistent with the results of controlled human exposure studies
28   discussed above, these panel studies report associations with lung function decrements for
29   subjects exposed to on-site monitored O_3 concentrations below 75 ppb. Associations in panel
30   studies have been reported for a wider range of populations than has been evaluated in controlled
31   human exposure studies, including children.
32   In considering controlled human exposure studies of other O_3-induced effects, we note
33   that airway hyper-responsiveness and impaired lung host defense capabilities have been reported
34   in healthy adults engaged in moderate exertion following exposures to O_3 concentrations as low
35   as 80 ppb, the lowest concentration evaluated for these effects. As discussed in section 3.1.2.1,
36   these physiological effects have been linked to aggravation of asthma and increased

1   susceptibility to respiratory infection, potentially leading to increased medication use, increased

2   school and work absences, increased visits to doctors' offices and emergency departments, and

3   increased hospital admissions. These are all indicators of adverse O_3-related morbidity effects,

4   which are consistent with, and provide plausibility for, the adverse morbidity effects and

5   mortality effects observed in epidemiologic studies.

6   In revisiting the question above, we conclude that the available controlled human

7   exposure evidence supports an upper end of the range of potential alternative standard levels for

8   consideration no higher than 70 ppb. For 6.6 hour exposures at 70 ppb, lung function decrements

9   and respiratory symptoms, a combination of effects that meet ATS criteria for an adverse

10 response (as discussed in section 3.1.3), have been demonstrated in healthy adults in controlled

11 human exposure studies.[9] In addition, potentially adverse respiratory effects, including lung

12 function decrements and airway inflammation, have been demonstrated following 6.6 hour

13 exposures to O_3 concentrations below 70 ppb (i.e., at 60 ppb, as discussed below). A level of 70

14 ppb would also be below the lowest-observed-effects level for effects such as airway hyper-

15 responsiveness and impaired host-defense capabilities in healthy adults while at prolonged

16 moderate exertion. As discussed in section 3.1.2.1 of this second draft PA, such physiological

17 effects have been linked to aggravation of asthma and increased susceptibility to respiratory

18 infection, potentially leading to increased medication use, increased school and work absences,

19 increased visits to doctors' offices and emergency departments, and increased hospital

20 admissions.

21   Based on the above considerations, we also conclude that the evidence from controlled

22 human exposure studies supports setting the lower end to the range of alternative O_3 standards at

23 60 ppb. Potentially adverse lung function decrements and pulmonary inflammation have been

24 demonstrated to occur at in healthy adults at 60 ppb. This is a short-term exposure concentration

25 that may be reasonably concluded to elicit adverse effects in at-risk groups. Pulmonary

26 inflammation, particularly if experienced repeatedly, provides a mechanism by which O_3 may

27 cause other more serious respiratory morbidity effects (e.g., asthma exacerbations) and possibly

28 extrapulmonary effects.

29 *Epidemiologic evidence*

30   We also consider what the information from epidemiologic studies indicates with regard

31 to potential alternative standard levels appropriate for consideration. Based on the information in

32 section 3.1.4.2 of this second draft PA (see Table 3-3), we first note that several epidemiologic

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[9]Although some studies report that respiratory symptoms develop during exposures to 60 ppb O_3, these effects have not reached statistical significance by the end of the 6.6 hour exposures (Adams, 2006; Schelegle et al., 2009). Thus respiratory symptoms, in combination with lung function decrements, are likely to occur to some degree in healthy individuals following exposures to O_3 concentrations somewhat below 70 ppb.

1   studies have reported positive and statistically significant associations with hospital admissions,

2   emergency department visits, and/or mortality in study areas where ambient O_3 concentrations

3   would have met the current standard (i.e., with its level of 75 ppb). This includes Canadian

4   multicity studies in which the majority of study cities would have met the current standard over

5   entire study periods (Cakmak et al., 2006; Dales et al., 2006; Katsouyanni et al., 2009; Stieb et

6   al., 2009), and a U.S. single-city study conducted in a location likely to have met the current

7   standard over the entire study period (Mar and Koenig, 2009).

8   In further evaluating these studies, and building upon our conclusions based on controlled

9   human exposures studies, as discussed above, we consider the following question related to the

10   epidemiologic evidence:

11   - **To what extent have U.S. and Canadian epidemiologic studies reported associations**
12   **with mortality or morbidity in locations likely to have met potential alternative O_3**
13   **standards with levels from 70 to 60 ppb?**

14   Addressing this question can provide important insights into the extent to which O_3-

15   health effect associations are present for distributions of ambient O_3 concentrations that would be

16   allowed by various potential alternative standards. To the extent O_3 health effect associations are

17   reported in study areas that would have met potential alternative standards, we have greater

18   confidence that exposures to ambient O_3 concentrations allowed by such alternatives could result

19   in the types of clearly adverse effects evaluated in these studies.[10] Therefore, our focus in this

20   section is to consider what these studies convey regarding the extent to which health effects may

21   be occurring (i.e., as indicated by associations) under air quality conditions allowed by potential

22   alternative standards. Specifically, we consider the numbers of study locations likely to have met

23   potential alternative standards with levels of 70, 65, and 60 ppb during study periods (Table 4-1).

24

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[10]See *ATA III*, 283 F.3d at 370 (EPA justified in revising NAAQS when health effect associations are observed at
levels allowed by the NAAQS).

Table 4-1. Numbers of epidemiologic study locations likely to have met potential alternative standards with levels of 70, 65, and 60 ppb

Study	Result	Cities	Number of study cities meeting potential alternative standards during entire study period		
			70 ppb	65 ppb	60 ppb
Cakmak et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	10 Canadian cities	7	6	2
Dales et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	11 Canadian cities	5	4	0
Katsouyanni et al. (2009)	Positive and statistically significant associations with respiratory hospital admissions	12 Canadian cities	9	9	5
Katsouyanni et al. (2009)	Positive and statistically significant associations with total and cardiovascular mortality	12 Canadian cities	7	5	1
Mar and Koenig (2009)	Positive and statistically significant associations with asthma emergency department visits	Single city: Seattle	0	0	0
Stieb et al. (2009)	Positive and statistically significant association with respiratory emergency department visits	7 Canadian cities	5	4	3

No U.S. or Canadian studies reported positive and statistically significant health effect associations when all study locations would have met a standard with a level from 70 to 60 ppb over the entire study period. However, for the studies by Cakmak et al. (2006), Katsouyanni et al. (2009), and Stieb et al. (2009), the majority of study locations would likely have met a standard with a level of either 70 or 65 ppb (Cakmak et al., 2006; Katsouyanni et al., 2009; Stieb et al., 2009). In contrast, the majority of locations in these studies would likely have violated a standard with a level of 60 ppb. While there is uncertainty in ascribing the multicity effect estimates reported in these Canadian studies to ambient concentrations that would have met standards with levels of 70 or 65 ppb (i.e., given that some study locations would have violated such standards over at least part of the study period), reported multicity effect estimates are largely influenced by locations meeting these potential alternative standards.

As with our consideration of the current standard (section 3.1.4.2), we next consider the extent to which epidemiologic studies have characterized O_3 health effect associations, including confidence in those associations, for various portions of distributions of ambient O_3 concentrations. In considering such analyses within the context of potential alternative standards,

1   we focus on the extent to which epidemiologic studies report health effect associations for air

2   quality distributions restricted to ambient pollutant concentrations below one or more

3   predetermined cut-points. As discussed in section 3.1.4.2, such "cut-point" analyses can provide

4   information on the magnitude and statistical precision of effect estimates for defined

5   distributions of ambient concentrations, which may in some cases include distributions that

6   would be allowed by potential alternative standards. Specifically, we consider the following

7   question:

8   - **To what extent do cut-point analyses from epidemiologic studies report health effect**
9   **associations at ambient O_3 concentrations that are likely to be allowed by potential**
10   **alternative standards with levels from 70 to 60 ppb?**

11   As with our consideration of the current standard in section 3.1.4.2 of this second draft

12   PA, we evaluate the cut-point analyses presented in the U.S. multicity study by Bell et al. (2006).

13   These cut-point analyses can provide insights into the magnitude and statistical precision of

14   health effect associations for different portions of the distribution of ambient concentrations,

15   including insights into the ambient concentrations below which uncertainty in reported

16   associations becomes notably greater. Our analysis of air quality data associated with the cut-

17   points evaluated by Bell et al., and uncertainties associated with that analysis, is described

18   elsewhere in this document (section 3.1.4.2). In this section, we consider what these cut-point

19   analyses indicate with regard to the potential for health effect associations to extend to ambient

20   O_3 concentrations likely to be allowed by a revised O_3 NAAQS with a level below 75 ppb.

21   We particularly focus on the lowest cut-point for which the association between O_3 and

22   mortality was reported to be statistically significant (i.e., 30 ppb, as discussed in section 3.1.4.2).

23   Based on the O_3 air quality concentrations that met the criteria for inclusion in the 30 ppb cut-

24   point analysis, 84% of study areas had 3-year averages of annual 4th highest 8-hour daily max O_3

25   concentrations at or below 70 ppb over the entire study period (Table 4-2). In addition, 64% of

26   study areas had 3-year averages of annual 4th highest 8-hour daily max O_3 concentrations at or

27   below 65 ppb (Table 4-2). In contrast, the majority of study areas had 4th highest concentrations

28   above 60 ppb. While there are uncertainties in interpreting these cut-point analyses within the

29   context of potential alternative standard levels, they suggest that the majority of the air quality

30   distributions that provided the basis for a positive and statistically significant association with

31   mortality would have been allowed by a standard with a level of 70 or 65 ppb, but would have

32   violated a standard with a level of 60 ppb. For higher cut-points, all of which also resulted in

33   statistically significant associations with mortality, the majority of study cities had 3-year

34   averages of annual 4th highest 8-hour daily max concentrations greater than 70 ppb.

35

Table 4-2. **Number of study cities with 3-year averages of 4th highest 8-hour daily max concentrations greater than 70, 65, or 60 ppb, for various cut-point analyses presented in Bell et al. (2006)**

	Cut-point for 2-day moving average across monitors and cities (24-h avg)								
	25	30	35	40	45	50	55	60	All
Number (%) of Cities with 4th highest >70 (any 3-yr period; 1987-2000)	0 (0%)	16 (16%)	55 (56%)	82 (84%)	89 (91%)	92 (94%)	94 (96%)	95 (97%)	95 (97%)
Number (%) of Cities with 4th highest >65 (any 3-yr period; 1987-2000)	3 (3%)	35 (36%)	77 (79%)	89 (91%)	94 (96%)	95 (97%)	95 (97%)	95 (97%)	95 (97%)
Number (%) of Cities with 4th highest >60 (any 3-yr period; 1987-2000)	16 (16%)	61 (62%)	86 (88%)	94 (96%)	95 (97%)	96 (8%)	96 (8%)	96 (8%)	96 (8%)

In considering the implications of these analyses for potential alternative standard levels, we also note the important uncertainties described in section 3.1.4. Several of these uncertainties become increasingly important as health effect associations are evaluated for lower ambient O_3 concentrations, such as when considering associations reported at the lower ends of the distributions of ambient O_3. These include uncertainties that could obscure presence of potential thresholds, affecting our characterization of confidence in O_3 health effect associations over distributions of ambient concentrations; uncertainty in the extent to which the relatively low ambient O_3 concentrations present in some study areas cause or contribute to reported effects; and uncertainty in the extent to which we were able to identify the air quality data associated with health effects in some published analyses (particularly for the subset analyses by Bell et al., 2006) (section 3.1.4.2).

We next consider the extent to which epidemiologic studies employing longer-term ambient O_3 concentration metrics can inform our consideration of potential alternative standard levels. In doing so, we consider the following question:

- **To what extent does the available evidence indicate that a standard with a level from 70 to 60 ppb, combined with the current 8-hour averaging time and 4th high form, could provide protection from long-term exposures to ambient O_3 concentrations for which there is evidence of health effects?**

We first note that, as discussed in section 3.1.4.3 of this second draft PA, virtually all of the study cities that provided the basis for the positive and statistically significant association

1 between long-term O_3 and respiratory mortality (Jerrett et al., 2009) would have violated the

2 current standard, and therefore potential alternative standards with lower levels. Thus, as with

3 our consideration of the current standard in section 3.1.4.3, while the study by Jerrett et al.

4 (2009) contributes to our understanding of health effects associated with ambient O_3

5 (summarized in section 3.1.2), it is less informative regarding the extent to which those health

6 effects may be occurring under air quality conditions that would meet potential alternative

7 standards.

8   To further evaluate this issue, we use the adjusted air quality in urban case study areas, as

9 described in the second draft HREA, to consider the extent to which just meeting alternative O_3

10 standards with levels of 70, 65, and 60 ppb could maintain long-term O_3 concentrations below

11 those in the cities that provided the basis for the positive and statistically significant association

12 with respiratory mortality reported by Jerrett et al. (2009).[11] Upon adjustment of air quality in

13 U.S. urban case study areas to meet the current and potential alternative 8-hour standards,

14 seasonal average 1-hour daily max concentrations were calculated and compared to the

15 concentrations in study cities.

16   As discussed in section 3.1.4.3, Jerrett et al. (2009) reported that when seasonal averages

17 of 1-hour daily max O_3 concentrations[12] ranged from 33 to 104 ppb, there was no statistical

18 deviation from a linear concentration-response relationship between O_3 and respiratory mortality

19 across 96 U.S. cities (U.S. EPA, 2013, section 7.7). However, as discussed in section 3.1.4.3, we

20 have the greatest confidence in the reported linear concentration-response function for "long-

21 term" O_3 concentrations above the first quartile (i.e., 53.2 ppb), given the notable widening in

22 confidence intervals for lower concentrations (based on visual inspection of Figure 3-6 in section

23 3.1.4.3); the limited evidence noted by study authors for a threshold at 56 ppb;[13] and the fact that

24 most study cities contributing to the linear function had O_3 concentrations in the highest three

25 quartiles (accounting for approximately 72% of the respiratory deaths in the cohort, based on

26 Table 2 in the published study).

27   Given the above, we note the extent to which long-term O_3 concentrations (i.e., seasonal

28 average of 1-hour daily max) in urban case study areas are estimated to be at or below 53 ppb

[11]Air quality in U.S. urban case study areas was adjusted to just meet the current 8-hour standard at 75 ppb, as well as potential potential alternative 8-hour standards at 70 ppb, 65 ppb, and 60 ppb, as described in the second draft HREA (chapter 4). After a given adjustment, seasonal average 1-hour daily max concentrations were calculated.

[12]Jerrett et al. (2009) evaluated the April to September averages of 1-hour daily max O_3 concentrations across 96 U.S. metropolitan areas from 1977- 2000. In urban areas with multiple monitors, April to September 1-hour daily max concentrations from each individual monitor were averaged. This step was repeated for each year in the study period. Finally, each yearly averaged O_3 concentrations was then averaged again to yield the single averaged 1-hour daily max O_3 concentration depicted on the x-axis of Figure 3-6 below.

[13]The ISA does not reach conclusions regarding the potential for a threshold in the association between "long-term" O_3 concentrations and respiratory mortality.

1   following model adjustment to meet potential alternative standards with levels of 70, 65, and 60

2   ppb. To the extent air quality adjustment to just meet potential alternative short-term standards

3   results in long-term concentrations near or below 53 ppb, we have greater confidence in the

4   degree to which those short-term standards could protect against the health effects associated

5   with longer term O_3 exposures. Though there is uncertainty associated with these comparisons

6   (e.g., due to uncertainty in the potential for a threshold to exist; uncertainty in the long-term

7   concentration below which confidence intervals widen notably, based on visual inspection of

8   concentration-response function in the published study; and the limited number of urban case

9   study areas for which adjusted air quality is available), this analysis can provide insight into the

10   extent to which various alternative short-term standards would be expected to maintain long-term

11   O_3 concentrations below those where we have the most confidence in the reported concentration-

12   response relationship with respiratory mortality.

13   Table 4-3 indicates that when considering recent (i.e., unadjusted) air quality, 2 of 12

14   urban case study areas had seasonal average 1-hour daily max O_3 concentrations at or below 53

15   ppb in all of the years examined. When air quality was adjusted to just meet the current 8-hour

16   standard (75 ppb in Table 4-3), 6 of 12 urban case study areas had seasonal average 1-hour daily

17   max O_3 concentrations at or below 53 ppb in all of the years examined. When air quality is

18   further adjusted to just meet potential alternative standards with lower levels, seasonal averages

19   of 1-hour daily max O_3 concentrations are estimated to be at or below 53 ppb in 9 of 12 urban

20   case study areas (70 ppb level), 10 of 12 urban case study areas (65 ppb level), and 11 of 11

21   urban case study areas (60 ppb level).[14] Though as noted above there are important uncertainties

22   associated with interpreting these comparisons, they suggest that in many locations across the

23   U.S. a standard with an 8-hour averaging time, when combined with the current 4th high form

24   and an appropriate standard level, would be expected to maintain seasonal averages of 1-hour

25   daily max O_3 concentrations below those where analyses indicate the most confidence in the

26   concentration-response relationship with respiratory mortality reported by Jerrett et al. (2009).

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[14]As described in the second draft HREA, a standard level of 60 ppb was not evaluated in New York City (U.S. EPA, 2014, chapter 4).

1
2
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Table 4-3. Seasonal averages of 1-hour daily max O_3 concentrations in U.S. urban case study areas for recent air quality and air quality adjusted to just meet the current and potential alternative standards.

	Air Quality Adjusted to:	2006 (Adj Yrs 2006-2008)	2007 (Adj Yrs 2006-2008)	2008 (Adj Yrs 2008-2010)	2009 (Adj Yrs 2008-2010)	2010 (Adj Yrs 2008-2010)
Atlanta	Recent	65	63	57	50	56
	75	53	52	53	47	52
	70	50	49	49	44	49
	65	47	46	46	42	46
	60	45	44	44	40	44
Baltimore	Recent	60	59	57	52	60
	75	54	54	53	49	55
	70	52	51	51	48	53
	65	49	49	48	46	50
	60	46	46	46	44	48
Boston	Recent	49	50	46	45	49
	75	48	49	49	45	48
	70	46	47	48	44	48
	65	44	45	46	43	46
	60	43	43	44	41	44
Cleveland	Recent	51	52	53	49	54
	75	49	50	51	47	51
	70	47	48	48	45	48
	65	45	45	45	43	45
	60	41	41	41	40	42
Denver	Recent	63	63	63	58	60
	75	62	61	63	58	60
	70	60	59	62	58	58
	65	58	58	59	56	55
	60	53	53	53	51	50
Detroit	Recent	50	54	51	48	52
	75	50	52	NA	NA	NA
	70	48	50	51	49	52
	65	47	49	49	47	50
	60	45	46	46	45	47
Houston	Recent	53	48	47	47	46
	75	48	46	47	48	46
	70	47	45	46	47	46
	65	46	44	45	46	45
	60	45	43	43	44	44
Los Angeles	Recent	65	61	64	62	57
	75	58	59	60	60	58
	70	55	56	57	58	56
	65	52	53	54	54	53
	60	50	51	52	52	50
New York City	Recent	53	54	55	48	55
	75	47	47	51	47	51
	70	44	45	48	45	48
	65	36	36	39	38	39
	60	NA	NA	NA	NA	NA
Philadelphia	Recent	56	59	57	51	58
	75	51	52	54	49	54
	70	49	50	51	47	52
	65	47	48	49	45	49
	60	45	46	47	43	47
Sacramento	Recent	66	59	65	61	55
	75	55	50	54	51	48
	70	52	48	51	49	46
	65	50	46	49	47	44
	60	47	44	46	44	42
Saint Louis	Recent	58	58	52	51	55
	75	53	53	51	50	54
	70	50	51	50	48	52
	65	47	48	48	46	49
	60	44	45	45	43	46

4

1   Based on the above analyses, we conclude that the available epidemiologic evidence is
2 consistent with the available evidence from controlled human exposure studies in providing
3 support for consideration of a standard level in the range of 70 to 60 ppb. Compared to the
4 current standard, a standard level from within this range would expected to be more effective at
5 maintaining short-term and long-term ambient O_3 concentrations below those where the evidence
6 indicates O_3-associated mortality and/or morbidity.

7   In reaching overall staff conclusions about an appropriate range of standard levels for
8 consideration, we further evaluate the results of the exposure and risk assessments that are based
9 on modeling changes in the entire distribution of ambient O_3 concentrations to simulate just
10 meeting potential alternative standards. These results are discussed below in section 4.4.2.

11 **4.4.2   Air Quality-, Exposure-, and Risk-Based Considerations**

12   Beyond considering the available evidence, we also consider the extent to which specific
13 potential alternative standard levels, in conjunction with the current averaging time and form (3-
14 year average of annual 4th highest 8-hour daily max), could reduce estimated O_3 exposures and
15 health risks. In the first draft PA (U.S. EPA, 2012b), we concluded that the available evidence
16 supports conducting further exposure and risk analyses of potential alternative standard levels in
17 the range of 70 down to 60 ppb. Based on these conclusions, the second draft HREA evaluates
18 exposures and risks estimated to be associated with potential alternative standard levels from the
19 upper (70 ppb), middle (65 ppb), and lower (60 ppb) portions of this range. In considering these
20 analyses in this second draft PA, we consider the following question:

21   • **To what extent does the available exposure and risk information provide support**
22   **for considering potential alternative standard levels from 70 to 60 ppb, when**
23   **combined with the current 8-hour averaging time and 4th high form?**

24 In considering exposure and risk analyses, we emphasize the nature and magnitude of the O_3
25 exposures and health risks estimated to remain upon just meeting each alternative standard level,
26 and the changes in exposures and risks estimated for each alternative level when compared to the
27 current standard. Section 4.4.2.1 below discusses our exposure-based considerations. Sections
28 4.4.2.2 and 4.4.2.3 discuss our consideration of estimates of lung function risks and estimates of
29 epidemiology-based mortality/morbidity risks, respectively.

30 **4.4.2.1   Exposure-Based Considerations**

31   As discussed in more detail in section 3.2.2 of this second draft PA, the exposure
32 assessment presented in the second draft HREA (U.S. EPA, 2014) provides estimates of the
33 number and percent of people exposed to O_3 concentrations at or above benchmark
34 concentrations of 60, 70, and 80 ppb, while at moderate or greater exertion. Estimates of such

1 "exposures of concern" provide perspective on the potential public health impacts of O_3-related

2 effects, including for effects that cannot currently be evaluated in a quantitative risk assessment.

3 The approach taken in the second draft HREA to estimating exposures of concern, and the key

4 uncertainties associated with exposure estimates, are summarized in section 3.2.2 for air quality

5 adjusted to just meet the current standard and are discussed in more detail in chapter 5 of the

6 second draft HREA (U.S. EPA, 2014). As discussed in section 3.2.2, when evaluating potential

7 alternative standard levels we focus on modeled exposures for school-age children (ages 5-18),

8 noting that percentages of asthmatic school-age children estimated to experience exposures of

9 concern are virtually indistinguishable from those for all children, and that patterns of exposure

10 in children represent a broader range of at-risk populations, which includes adult asthmatics and

11 older adults.

12   In this section, we consider the following question:

13   • **To what extent are potential alternative standards with revised levels estimated to**
14   **reduce the occurrence of O_3 exposures of concern, compared to the current**
15   **standard, and what are the nature and magnitude of the exposures remaining**
16   **for each alternative standard level evaluated?**

17 Key results related to this question are summarized below (Figures 4-1 to 4-4). Figures 4-1 and

18 4-2 present estimates of one or more exposures of concern, and Figures 4-3 and 4-4 present

19 estimates of two or more exposures of concern.

1 **Figure 4-1.** **Percent of children estimated to experience one or more exposures of concern at or above 60, 70, or 80 ppb for air**
2 **quality adjusted to just meet the current and potential alternative standards (averaged over 2006 to 2010)**

3
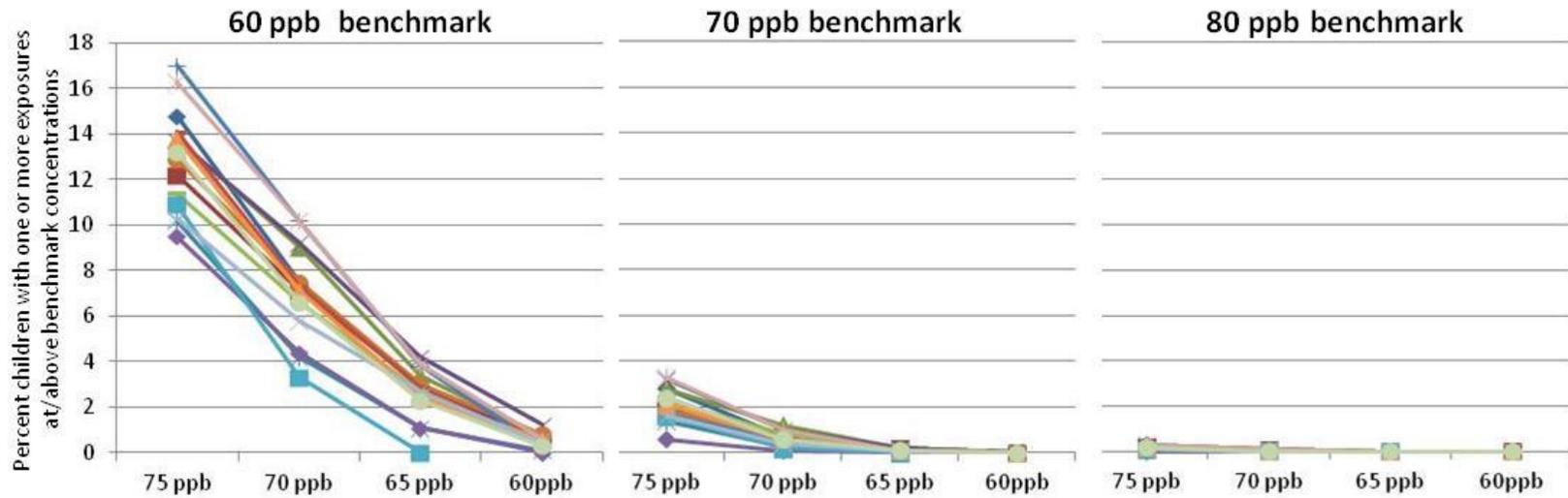
4

1   **Figure 4-2. Percent of children estimated to experience one or more exposures of concern at or above 60, 70, or 80 ppb for air**
2   **quality adjusted to just meet the current and potential alternative standards (worst-case year from 2006 to**
3   **2010[15])**
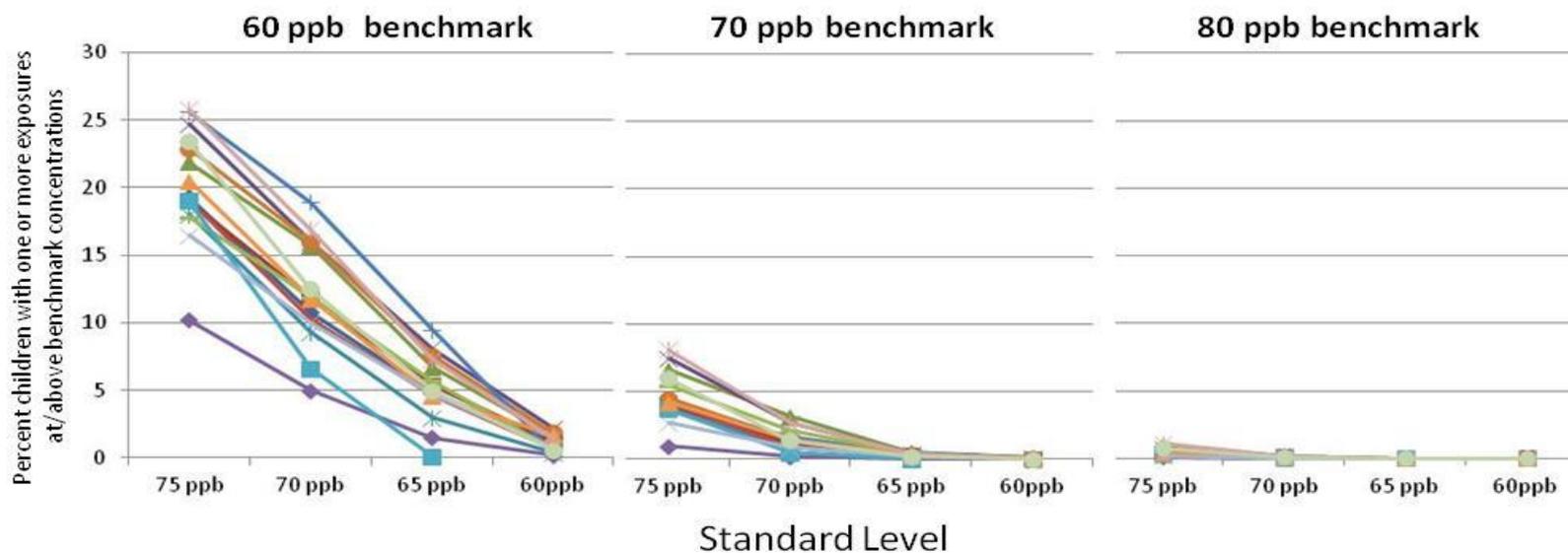
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5

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[15]"Worst-case" year refers to the year in each urban case study area with the largest percentage of children estimated to experience exposures of concern.

1   **Figure 4-3.** **Percent of children estimated to experience two or more exposures of concern at or above 60, 70, or 80 ppb for air**
2   **quality adjusted to just meet the current and potential alternative standards (averaged over 2006 to 2010)**

3
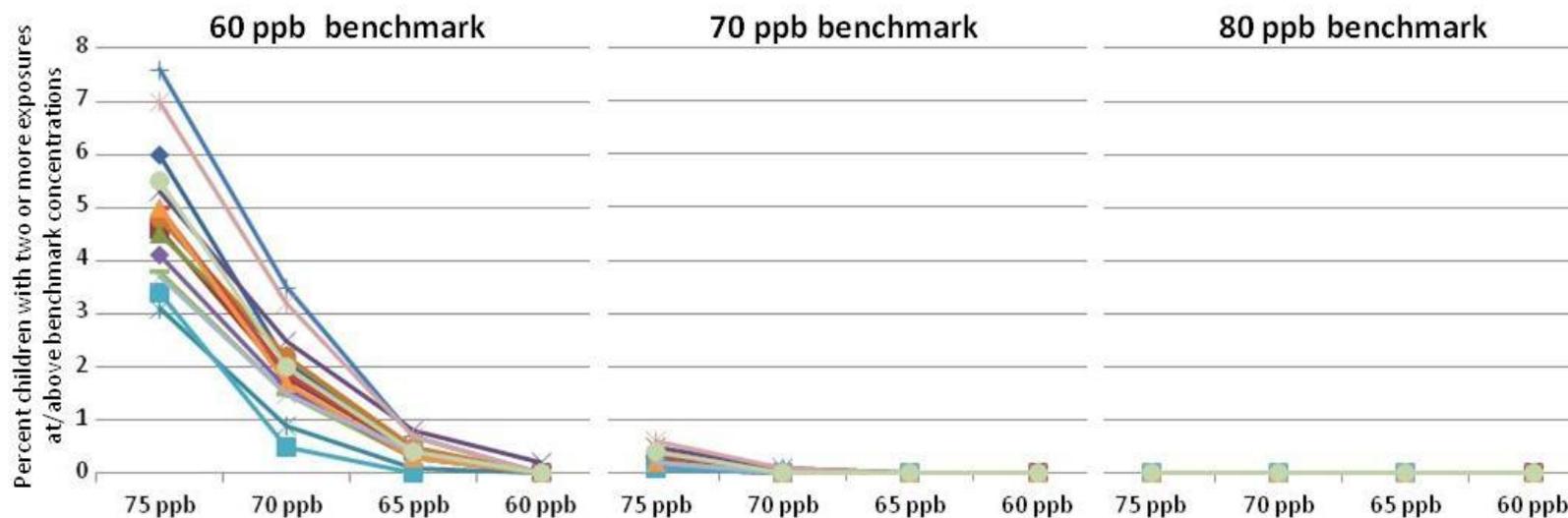
Figure 4-4. Percent of children estimated to experience two or more exposures of concern at or above 60, 70, or 80 ppb for air quality adjusted to just meet the current and potential alternative standards (worst-case year from 2006 to 2010)
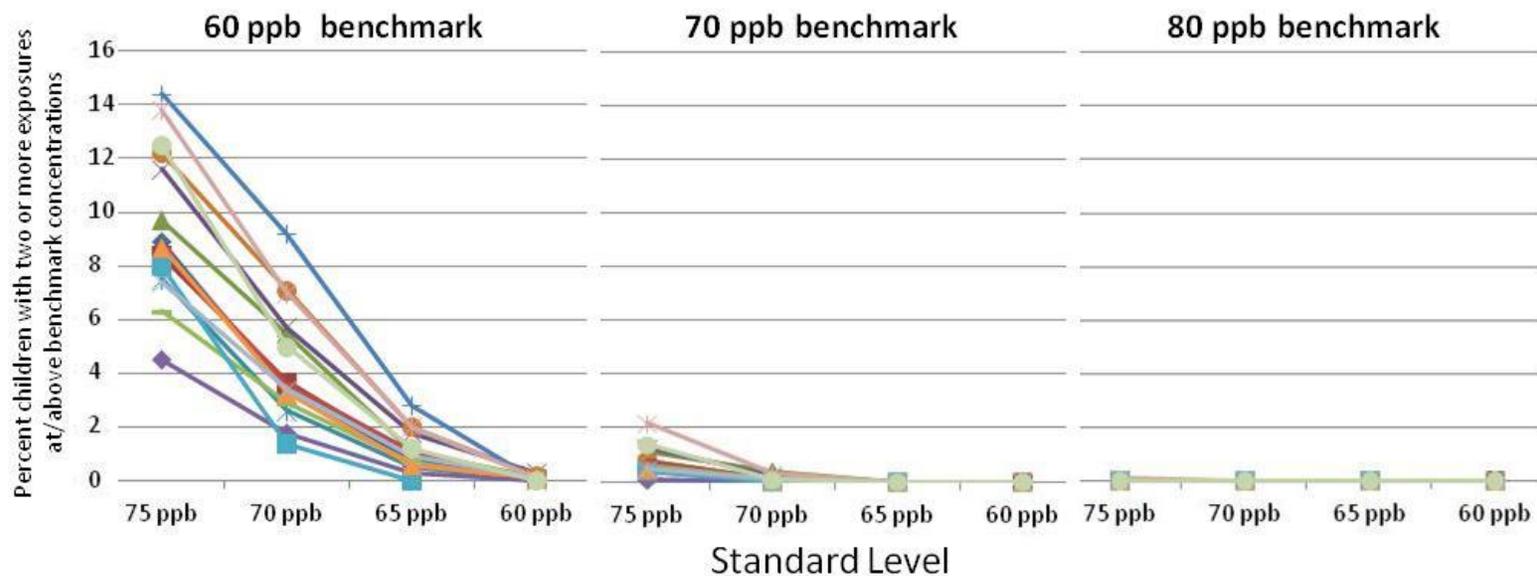
1   As illustrated above in Figures 4-1 to 4-4, adjusting air quality to just meet progressively

2 lower potential alternative standard levels reduces estimated exposures of concern consistently

3 across urban case study areas. These results reflect the consistent reductions in the highest

4 ambient O_3 concentrations upon model adjustment, as summarized in section 3.2.1 and as

5 discussed in more detail in the second draft HREA (U.S. EPA, 2014, chapter 4). Based on

6 Figures 4-1 to 4-4 and the associated details described in the second draft HREA (U.S. EPA

7 2014, chapter 5), we take note of the following with regard to exposures of concern for specific

8 potential alternative standard levels:

9   1. For a standard level of 70 ppb:

10   a. On average over the years 2006 to 2010, a standard with a level of 70 ppb is
11   estimated to allow approximately 3 to 11% of children in urban case study areas to
12   experience one or more exposures of concern at or above 60 ppb (approximately 30 to
13   70% reduction, relative to current standard). Summing across urban case study areas,
14   these percentages correspond to over 1 million children experiencing over 1.5 million
15   exposures of concern at or above 60 ppb during a single O_3 season. Of these children,
16   over 100,000 are asthmatics.

17

18   b. On average over the years 2006 to 2010, a standard with a level of 70 ppb is
19   estimated to allow approximately 0.5 to 3.5% of children in urban case study areas to
20   experience two or more exposures of concern at or above 60 ppb (approximately 50
21   to 85% reduction, relative to current standard).

22

23   c. In the worst-case years (i.e., those with the largest exposure estimates), a standard
24   with a level of 70 ppb is estimated to allow approximately 5 to 19% of children in
25   urban case study areas to experience one or more exposures of concern at or above 60
26   ppb, and approximately 2 to 9% to experience two or more.

27

28   d. On average over the years 2006 to 2010, a standard with a level of 70 ppb is
29   estimated to allow approximately 1% or less of children to experience one or more
30   exposures of concern at or above 70 ppb (approximately 55 to 90% reduction, relative
31   to current standard), and far less than 1% to experience two or more such exposures
32   (approximately 65 to 100% reduction, relative to current standard).

33

34   e. In the worst-case years, approximately 3% or less of children are estimated to
35   experience one or more exposures of concern at or above 70 ppb, and less than 1%
36   are estimated to experience two or more such exposures.

37

38   f. A standard with a level of 70 ppb is estimated to allow less than 1% of children to
39   experience one or more exposures of concern at or above 80 ppb, even in the worst-
40   case years. No children are estimated to experience two or more such exposures.

41

42   2. For a standard level of 65 ppb:

a. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow approximately 4% or less of children in urban case study areas to experience one or more exposures of concern at or above 60 ppb (approximately 70 to 100% reduction, relative to current standard). Summing across urban case study areas, these percentages correspond to almost 500,000 children experiencing approximately 500,000 exposures of concern at or above 60 ppb during a single O_3 season. Of these children, almost 50,000 are asthmatics.

b. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow less than 1% of children to experience two or more exposures of concern at or above 60 ppb (approximately 85 to 100% reduction, relative to current standard).

c. In the worst-case years, a standard with a level of 65 ppb is estimated to allow approximately 9% or less of children to experience one or more exposures of concern at or above 60 ppb, and approximately 3% or less to experience two or more such exposures.

d. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow less than 1% of children to experience one or more exposures of concern at or above 70 ppb (approximately 90 to 100% reduction, relative to current standard), and no children to experience two or more such exposures (100% reduction, relative to current standard). Even in the worst-case years, a level of 65 ppb is estimated to allow less than 1% of children to experience exposures of concern at or above 70 ppb.

e. A standard with a level of 65 ppb is estimated to allow virtually no children to experience exposures of concern at or above 80 ppb, even in the worst-case years.

3. For a standard level of 60 ppb:
a. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to allow approximately 1% or less of children to experience one or more exposures of concern at or above 60 ppb (approximately 90 to 100% reduction, relative to current standard), and virtually no children to experience multiple such exposures.

b. In the worst-case years, a standard with a level of 60 ppb is estimated to allow approximately 2% or less of children to experience one or more exposures of concern at or above 60 ppb, and virtually no children to experience multiple such exposures.

c. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to eliminate exposures of concern at or above 70 ppb (100% reduction, relative to current standard) or 80 ppb. Even in years with the highest exposure estimates, virtually no children are estimated to experience such exposures.

1   In further considering these exposure estimates, we take note of the associated

2   uncertainties, as discussed in more detail in section 3.2.2 of this second draft PA. These include

3   (1) individual variability in responsiveness to O_3 exposures; (2) potential to underestimate

4   exposures in most highly exposed populations; and (3) potential to overestimate exposures in

5   populations who alter behavior in response to high O_3 days (i.e., spend less time being active

6   outdoors).

4.4.2.2   Risk-Based Considerations: Lung Function

8   As discussed above in more detail in section 3.2.3.1 of this second draft PA, the

9   assessment of lung function risks presented in the second draft HREA (U.S. EPA, 2014)

10   provides estimates of the number and percent of people experiencing O_3-induced lung function

11   decrements greater than or equal to 10, 15, and 20%. In the last review, CASAC advised EPA to

12   focus on decrements of 10% or greater when considering people with pre-existing lung disease

13   (Samet, 2011).

14   Lung function risk estimates are based on an updated dose-threshold model that estimates

15   FEV_1 responses for individuals following short-term exposures to O_3 (McDonnell, Stewart, and

16   Smith, 2010), reflecting methodological improvements since the last review (U.S. EPA, 2014,

17   section 6.2.4). The approach taken in the second draft HREA to estimating O_3-induced lung

18   function decrements, and the key uncertainties associated with these estimates, are summarized

19   in section 3.2.3.1 for air quality adjusted to just meet the current standard and are discussed in

20   more detail in chapter 6 of the HREA (U.S. EPA, 2014).

21   As discussed in section 3.2.3.1, in evaluating potential alternative standard levels we

22   focus on modeled exposures for school-age children, with an emphasis on asthmatic children. As

23   with exposures of concern, the percentages of all school age children and asthmatic school age

24   children estimated to experience particular O_3-induced lung function decrements are virtually

25   indistinguishable.

26   In this section, we consider the following question:

27   • **To what extent are potential alternative standards with revised levels estimated to**
28   **decrease the occurrence of O_3-induced lung function decrements, compared to**
29   **the current standard, and what are the nature and magnitude of the decrements**
30   **remaining for each alternative standard level evaluated?**

31   Key results related to this question are summarized below (Figures 4-5 to 4-8). Figures 4-5 and

32   4-6 present estimates of one or more O_3-induced lung function decrements, and Figures 4-7 and

33   4-8 present estimates of two or more decrements.

1 **Figure 4-5. Percent of children estimated to experience one or more O$_3$-induced lung function decrements greater than 10, 15,**
2 **or 20% for air quality adjusted to just meet the current and potential alternative standards (averaged over 2006**
3 **to 2010)**
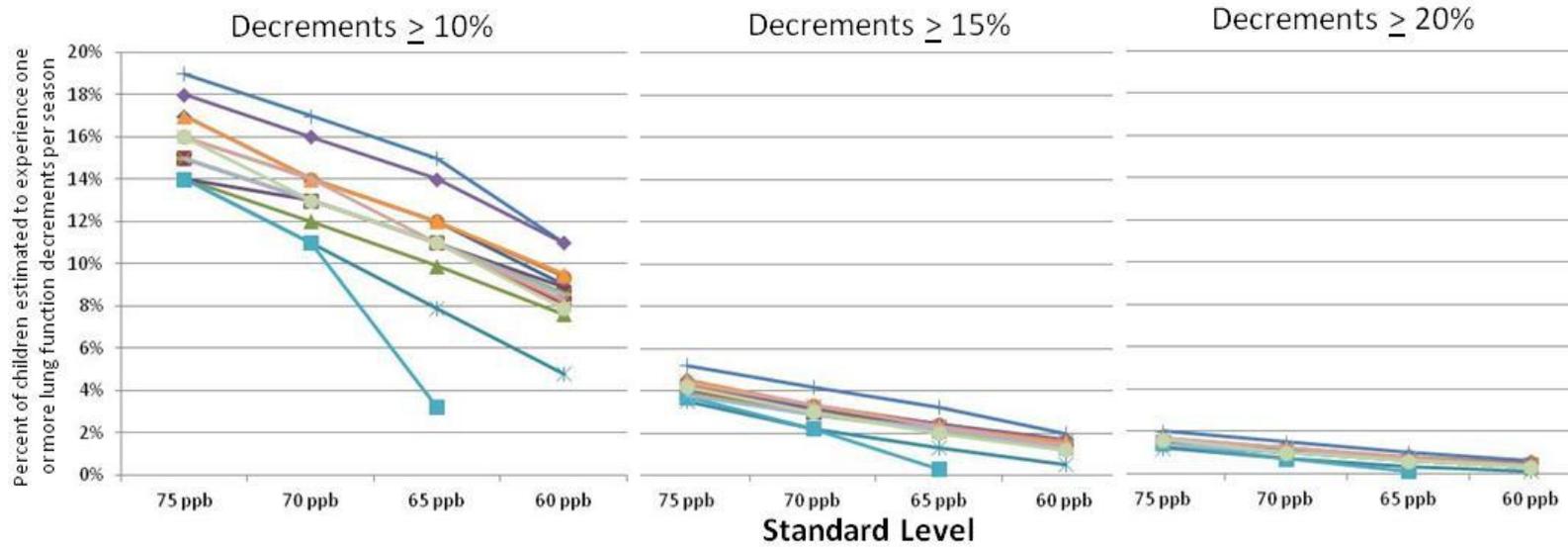
4

Atlanta
Baltimore
Boston
Chicago
Cleveland
Dallas
Denver
Detroit
Houston
Los Angeles
New York
Philadelphia
Sacramento
St Louis
Washington

5

1　**Figure 4-6.**　**Percent of children estimated to experience one or more O_3-induced lung function decrements greater than 10, 15,**
2　　　　　　　**or 20% for air quality adjusted to just meet the current and potential alternative standards (worst-case year from**
3　　　　　　　**2006 to 2010)**
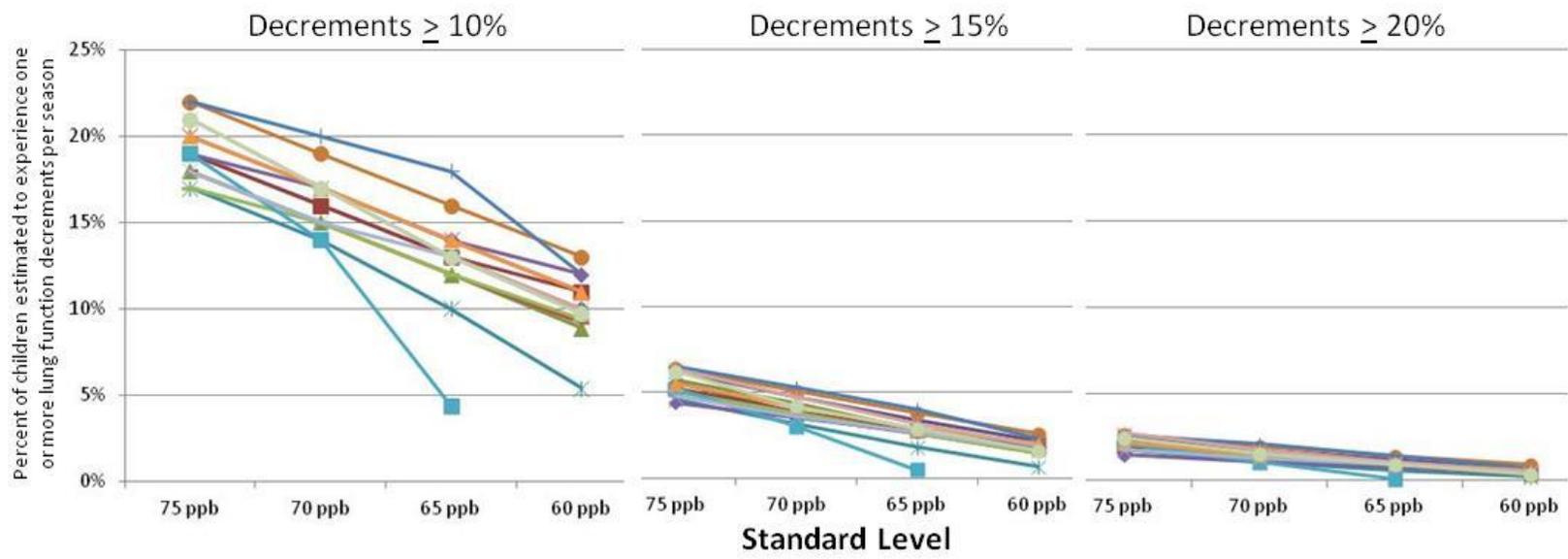
4

1　Figure 4-7.　**Percent of children estimated to experience two or more O$_3$-induced lung function decrements greater than 10,**
2　　　　　　　**15, or 20% for air quality adjusted to just meet the current and potential alternative standards (averaged over**
3　　　　　　　**2006 to 2010)**
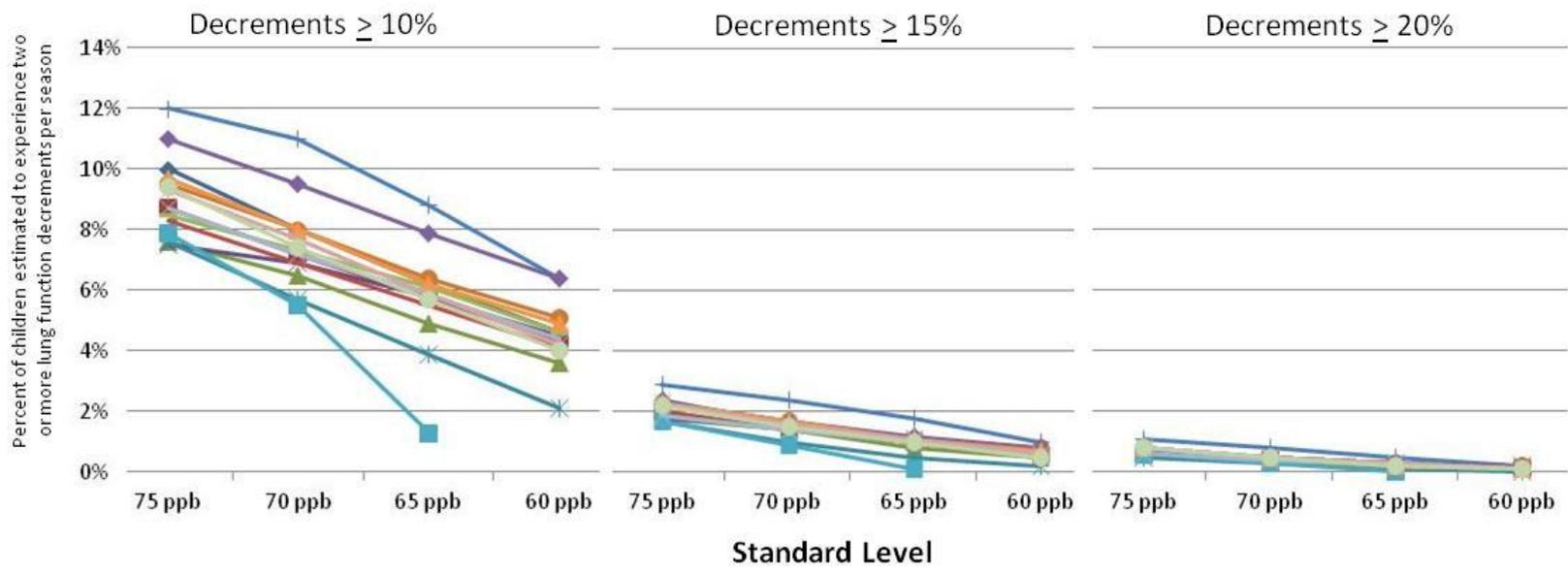
4

Legend:
- Atlanta
- Baltimore
- Boston
- Chicago
- Cleveland
- Dallas
- Denver
- Detroit
- Houston
- Los Angeles
- New York
- Philadelphia
- Sacramento
- St Louis
- Washington

5

1 **Figure 4-8.** **Percent of children estimated to experience two or more O$_3$-induced lung function decrements greater than 10,**
2 **15, or 20% for air quality adjusted to just meet the current and potential alternative standards (worst-case year**
3 **from 2006 to 2010)**
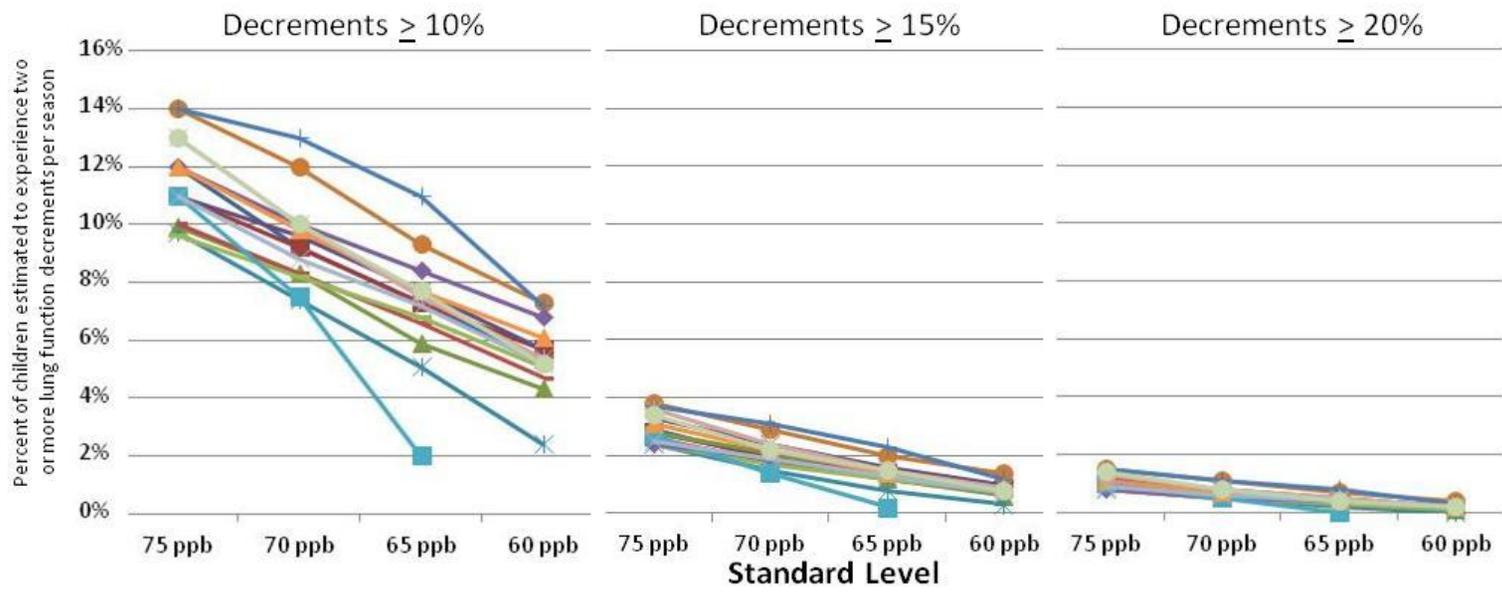
4

Legend:
- Atlanta
- Baltimore
- Boston
- Chicago
- Cleveland
- Dallas
- Denver
- Detroit
- Houston
- Los Angeles
- New York
- Philadelphia
- Sacramento
- St Louis
- Washington

5

1   As illustrated above in Figures 4-5 to 4-8, adjusting air quality to just meet progressively

2   lower potential alternative standard levels consistently reduces the percent of children estimated

3   to experience potentially adverse lung function decrements. These results reflect the consistent

4   reductions in the highest ambient O_3 concentrations upon model adjustment (section 3.2.1; U.S.

5   EPA, 2014, chapter 4).[16] Based on Figures 4-5 to 4-8 and the associated details described in the

6   second draft HREA (U.S. EPA 2014, chapter 6), we take note of the following with regard to

7   specific potential alternative standard levels:

8   1. For a standard level of 70 ppb:

9   a. On average over the years 2006 to 2010, a standard with a level of 70 ppb is
10   estimated to allow approximately 11 to 17% of children in urban case study areas,
11   including asthmatic children, to experience one or more O_3-induced lung function
12   decrements $\geq 10\%$ (approximately 6 to 27% reduction, relative to current
13   standard) per season. Summing across case study areas, these percentages
14   correspond to approximately 260,000 asthmatic children experiencing
15   approximately 1 million total occurrences of O_3-induced lung function
16   decrements greater than or equal to 10%.

18   b. On average over the years 2006 to 2010, a standard with a level of 70 ppb is
19   estimated to allow approximately 6 to 11% of children, including asthmatic
20   children, to experience two or more O_3-induced lung function decrements $\geq 10\%$
21   (approximately 8 to 30% reduction, relative to current standard).

23   c. In the worst-case years, a standard with a level of 70 ppb is estimated to allow
24   approximately 14 to 20% of children, including asthmatic children, to experience
25   one or more O_3-induced lung function decrements $\geq 10\%$, and approximately 7 to
26   13% to experience two or more such decrements.

28   d. On average over the years 2006 to 2010, a standard with a level of 70 ppb is
29   estimated to allow approximately 2 to 4% of children, including asthmatic
30   children, to experience one or more O_3-induced lung function decrements $\geq 15\%$,
31   and approximately 1 to 2.5% of children to experience two or more such O_3-
32   induced decrements. In the worst-case years, approximately 3 to 5% of children
33   are estimated to experience one or more O_3-induced lung function decrements
34           $\geq 15\%$, and approximately 1 to 3% are estimated to experience two or more such
35   decrements.

37   e. A standard with a level of 70 ppb is estimated to allow 2% or fewer children to
38   experience any O_3-induced lung function decrements $\geq 20\%$, even in the worst-
39   case years. Approximately 1% or fewer children are estimated to experience two

[16]The impact of the dose threshold in the lung function risk model is that O_3-induced FEV1 decrements result
primarily from exposures to O_3 concentrations above about 40 ppb (US EPA, 2013, chapter 6).

1   or more O_3-induced lung function decrements $\geq$ 20%, even in the worst-case
2   years.
3

4 2. For a standard level of 65 ppb:
5   a. On average over the years 2006 to 2010, a standard with a level of 65 ppb is
6   estimated to allow approximately 3 to 15% of children, including asthmatic
7   children, to experience one or more O_3-induced lung function decrements $\geq$ 10%
8   (approximately 20 to 77% reduction, relative to current standard). Summing
9   across urban case study areas, these percentages correspond to approximately
10   190,000 asthmatic children experiencing almost 750,000 total occurrences of O_3-
11   induced lung function decrements $\geq$ 10%.
12

13   b. On average over the years 2006 to 2010, a standard with a level of 65 ppb is
14   estimated to allow approximately 1 to 9% of children, including asthmatic
15   children, to experience two or more O_3-induced lung function decrements $\geq$ 10%
16   (approximately 20 to 80% reduction, relative to current standard).
17

18   c. In the worst-case years, a standard with a level of 65 ppb is estimated to allow
19   approximately 4 to 18% of children to experience one or more O_3-induced lung
20   function decrements $\geq$ 10%, and approximately 2 to 11% to experience two or
21   more such decrements.
22

23   d. On average over the years 2006 to 2010, a standard with a level of 65 ppb is
24   estimated to allow approximately 3% or less of children to experience one or
25   more O_3-induced lung function decrements $\geq$ 15%, and approximately 2% or less
26   of children to experience two or more such O_3-induced decrements. In the worst-
27   case years, approximately 4% or less of children are estimated to experience one
28   or more O_3-induced lung function decrements $\geq$ 15%, and up to approximately
29   2.5% are estimated to experience two or more such decrements.
30

31   e. A standard with a level of 65 ppb is estimated to allow less than 1.5% of children
32   to experience any O_3-induced lung function decrements $\geq$ 20%, even in the worst-
33   case years. A standard with a level of 65 ppb is estimated to allow less than 1% of
34   children to experience two or more O_3-induced lung function decrements $\geq$ 20%,
35   even in the worst-case years.
36

37 3. For a standard level of 60 ppb:
38   a. On average over the years 2006 to 2010, a standard with a level of 60 ppb is
39   estimated to allow approximately 5 to 11% of children, including asthmatic
40   children, to experience one or more O_3-induced lung function decrements $\geq$ 10%
41   (approximately 35 to 77% reduction, relative to current standard). Summing
42   across urban case study areas, these percentages correspond to approximately
43   140,000 asthmatic children experiencing approximately 500,000 total occurrences
44   of O_3-induced lung function decrements $\geq$ 10%.
45

b. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to allow approximately 2 to 6% of children to experience two or more O_3-induced lung function decrements $\geq$ 10% (approximately 40 to 70% reduction, relative to current standard).

c. In the worst-case years, a standard with a level of 60 ppb is estimated to allow approximately 5 to 13% of children to experience one or more O_3-induced lung function decrements $\geq$ 10%, and approximately 2 to 7% to experience two or more such decrements.

d. A standard with a level of 60 ppb is estimated to allow less than about 3% of children to experience any O_3-induced lung function decrements $\geq$ 15% and less than 1% to experience decrements greater than 20%, even in years with the highest exposure estimates. A standard with a level of 60 ppb is estimated to allow less than 1.5% of children to experience two or more O_3-induced lung function decrements $\geq$ 15% and less than 0.5% to experience two or more decrements $\geq$ 20%, even in years with the highest exposure estimates.

In further considering these exposure estimates, we take note of the associated uncertainties, as discussed in more detail in section 3.2.2 of this second draft PA. In addition to the uncertainties in exposure estimates noted above, these include the relative lack of exposure-response information for key at-risk populations (i.e., children and asthmatics), since most controlled human exposures studies are conducted in healthy adults.

4.4.2.3 Risk-Based Considerations: Epidemiology-Based Mortality and Morbidity

The epidemiology-based risk assessments presented in the second draft HREA (U.S. EPA, 2014, chapter 7) provide estimates of total mortality, respiratory hospital admissions and emergency department visits, and asthma exacerbations associated with short-term O_3 concentrations. The HREA also presents estimates of respiratory mortality associated with long-term[17] concentrations. In evaluating these risk estimates, we consider the following question:

- **To what extent are potential alternative standards with revised levels estimated to decrease O_3 health risks, compared to the current standard, and what are the nature and magnitude of the health risks remaining for each alternative standard level evaluated?**

As discussed in more detail in section 3.2.3.2 of this second draft PA, in considering this question we are mindful that the model-based approach used to adjust air quality in the second

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[17]Estimates of respiratory mortality associated with long-term O_3 concentrations are based on the study by Jerrett et al. (2009). Consistent with the O_3 metric used in the study, risk estimates are based on seasonal averages of 1-hour daily max O_3 concentrations.

1   draft HREA has important implications for risk estimates developed by applying concentration-

2   response relationships from epidemiologic studies (section 3.2.1). In particular, we note the

3   uncertainty associated with using such concentration-response relationships to estimate risks for

4   model-adjusted air quality with spatial and temporal patterns of ambient O_3 that are different

5   from those present in the epidemiologic study locations. In addition, given the use of linear

6   concentration-response relationships, risk estimates are equally influenced by decreasing high O_3

7   concentrations and increasing low O_3 concentrations following model adjustment, when the

8   increases and decreases are of equal magnitude. These and other uncertainties associated with

9   risk estimates are discussed in section 3.2.3.2.

10   Key results from the second draft HREA (U.S. EPA, 2014, chapter 7) are summarized

11   below for estimates of total mortality associated with short-term O_3 concentrations (Figures 4-9

12   and 4-10), respiratory hospital admissions associated with short-term O_3 concentrations (Figure

13   4-11), and respiratory mortality associated with long-term O_3 concentrations (Figure 4-12). The

14   other morbidity effects evaluated in the second draft HREA (i.e., respiratory emergency

15   department visits and asthma symptoms associated with short-term concentrations) exhibit

16   patterns across standard levels that are similar to those reported for total mortality and respiratory

17   hospital admissions (U.S. EPA, 2014, chapter 7).

18   As discussed in section 3.2.3.2, for total mortality associated with short-term O_3

19   concentrations we consider estimates of risk based on the full distributions of area-wide O_3

20   concentrations (Figure 4-9) and estimates of risk associated with various portions of those

21   distributions (Figure 4-10).[18] In doing so, we recognize the reduced certainty in a linear

22   concentration-response relationship at the lower ends of air quality distributions, and the greater

23   certainty in increased incidence and severity of effects at higher exposure concentrations

24   (discussed in more detail in section 3.2.3.2).[19]

---

[18]The second draft HREA does not present distributions of risk over distributions of area-wide concentrations for other epidemiology-based risk endpoints (U.S. EPA, 2014, chapter 7).

[19]As discussed in section 3.1.2.2, as ambient concentrations increase the potential for exposures to higher O_3 concentrations also increases. Thus with increasing ambient concentrations, controlled human exposure and animal toxicological studies provide greater certainty in the increased incidence, magnitude, and severity of O_3-attributable effects.

Figure 4-9. Estimates of Total Mortality Associated with Short-Term O$_3$ Concentrations in Urban Case Study Areas (Air Quality Adjusted to Current and Potential alternative standard levels) – Total Risk

1　**Figure 4-10. Estimates of Total Mortality Attributable to Days with 8-Hour Area-Wide O_3 Concentrations at or above 20, 40,**
2　**or 60 ppb, Summed Across Urban Case Study Areas (Air Quality Adjusted to Current and Potential alternative**
3　**standard levels)**
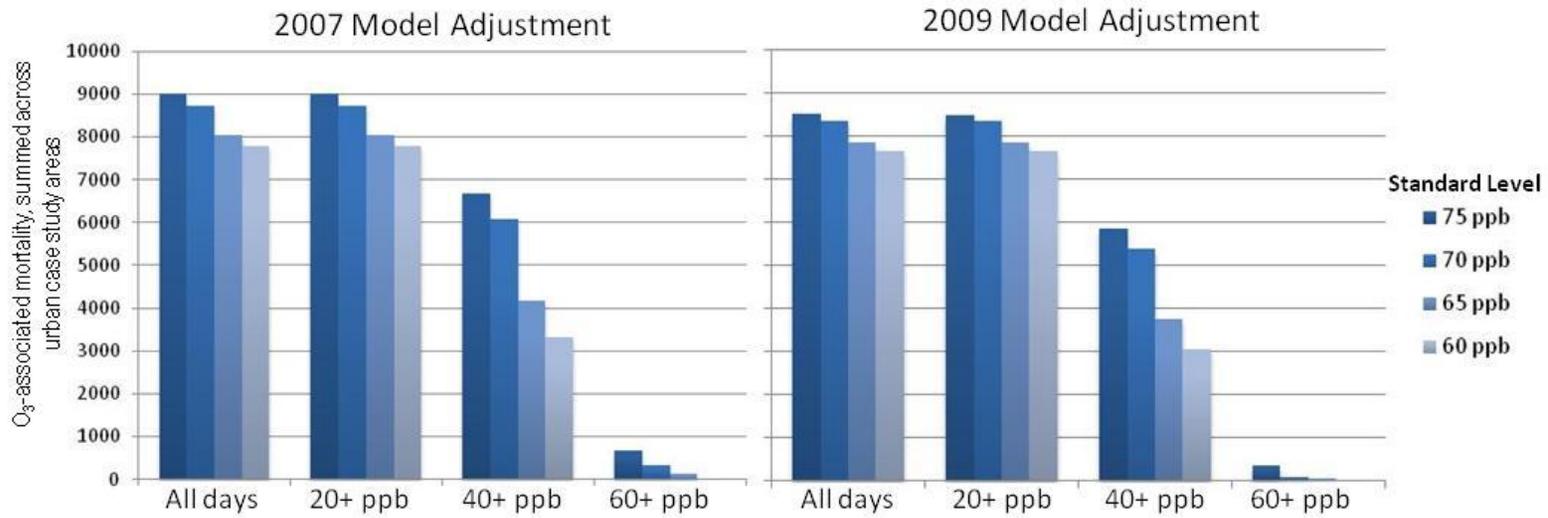
4
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6

1   **Figure 4-11. Estimates of Respiratory Hospital Admissions Associated with Short-Term O$_3$ Concentrations in Urban Case**
2   **Study Areas (Air Quality Adjusted to Current and Potential alternative standard levels) – Total Risk**
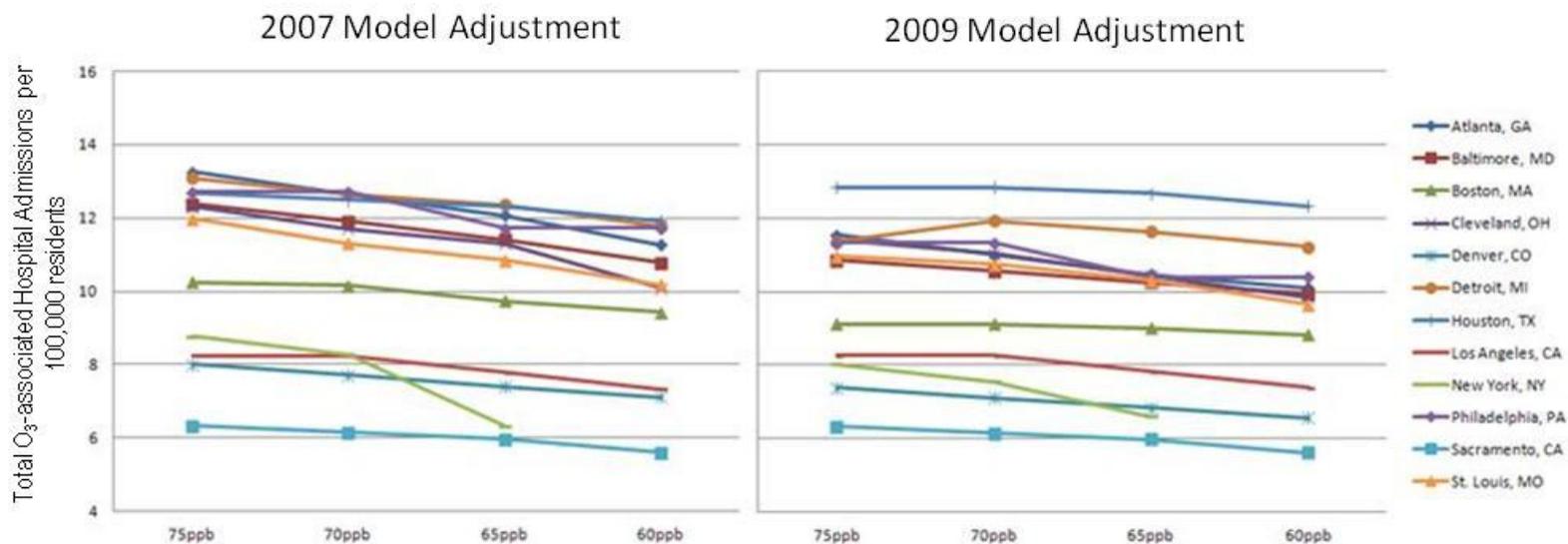
3
4

1   **Figure 4-12. Estimates of Respiratory Mortality Associated with long-term O$_3$ Concentrations in Urban Case Study Areas (Air**
2   **Quality Adjusted to Current and Potential alternative standard levels) – Total Risk**
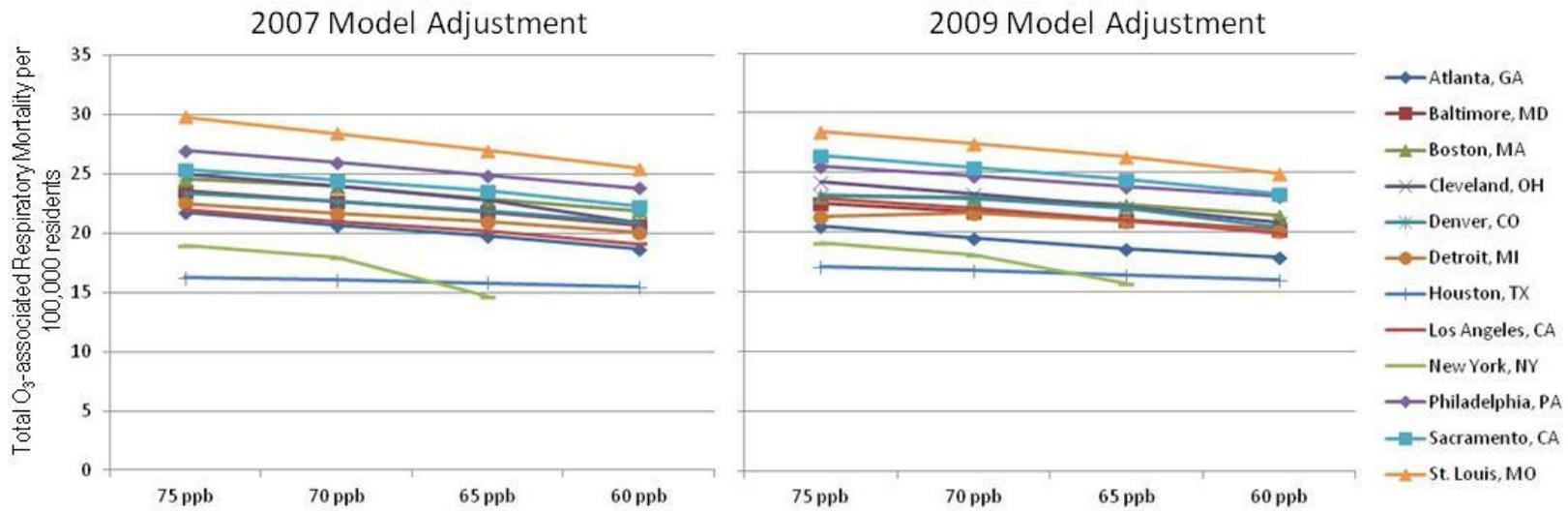
3

1   Based on Figures 4-9 to 4-12 and the associated details described in the second draft
2   HREA (U.S. EPA 2014, chapter 7), we take note of the following for a standard level of 70 ppb:

3   1. Total mortality associated with short-term O_3 concentrations:
4   a. A standard level of 70 ppb results in modest changes in total risk, compared to the
5   current standard. Across urban case study areas, risks are estimated to decrease by
6   up to approximately 5%. These risk reductions are estimated most consistently for
7   the model year with generally higher O_3-associated risks (2007). In the year with
8   generally lower risks (2009), a standard level of 70 ppb results in either no change
9   or small reductions in estimated risks in most urban case study areas. In one area
10   (Detroit) for the 2009 model year, O_3-associated mortality is estimated to increase
11   by approximately 4%, compared to the current standard (see section 3.2.3.2 for
12   further discussion of increased risk estimates following model adjustment[20]).
13
14   b. When summed across urban case study areas, a standard level of 70 ppb is
15   estimated to reduce O_3-associated total mortality by approximately 4% (2007
16   model year) and 2% (2009 model year), compared to the current standard. For
17   days with area-wide concentrations at or above 40 ppb, a standard level of 70 ppb
18   is estimated to reduce O_3-associated total mortality by approximately 9% (2007
19   model year) and 8% (2009 model year). For days with area-wide concentrations at
20   or above 60 ppb, a standard level of 70 ppb is estimated to reduce O_3-associated
21   total mortality by approximately 50% (2007 model year) and 70% (2009 model
22   year).[21]
23
24   2. Respiratory hospital admissions associated with short-term O_3 concentrations: Compared to
25   the current standard, changes in total risk estimated for a standard level of 70 ppb are similar
26   to the changes in total risks estimated for total mortality (U.S. EPA, 2014, chapter 7).
27
28   3. Respiratory mortality associated with long-term O_3 concentrations: A standard level of 70
29   ppb reduces total risk, compared to the current standard. Across urban case study areas, risks
30   are estimated to decrease by up to approximately 8%. These risk reductions are estimated
31   most consistently for the model year with generally higher O_3-associated risks (2007). In the
32   year with generally lower O_3 concentrations (2009), a standard level of 70 ppb results in
33   either no change or small reductions in estimated risks in most urban case study areas. In one
34   area (Detroit) for the 2009 model year, O_3-associated mortality is estimated to increase by
35   approximately 1%, compared to the current standard.

---

[20]As discussed in more detail above (section 3.2.3.2), because of the influence of the entire distribution of ambient O_3 concentrations on total risk estimates, the impacts of adjusting air quality to just meet potential alternative standards are more modest, and are less directionally consistent across urban case study areas, than observed for exposures of concern or O_3-induced lung function decrements.

[21]These results reflect the fact that increases in area-wide O_3 concentrations upon model adjustment occur primarily at relatively low concentrations (i.e., at or below area-wide concentrations of approximately 45 ppb) (U.S. EPA, 2014, section 4.3.3.2 and appendix 7B).

1   Based on Figures 4-9 to 4-12 and the associated details described in the second draft
2   HREA (U.S. EPA 2014, chapter 7), we take note of the following for a standard level of 65 ppb:

3   1. Total mortality associated with short-term O_3 concentrations:
4   a. A standard level of 65 ppb results in small changes in total risk, compared to the
5   current standard. Across most urban case study areas, risks are estimated to
6   decrease by up to approximately 9%. In one area (New York City), risks are
7   estimated to decrease by up to approximately 22%.[22] These risk reductions are
8   estimated most consistently for the model year with generally higher O_3-
9   associated risks (2007). In the year with generally lower risks (2009), a standard
10   level of 65 ppb results in smaller reductions in estimated risks in most urban case
11   study areas. In one area (Detroit) for the 2009 model year, O_3-associated mortality
12   is estimated to increase by approximately 1% compared to the current standard.
13
14   b. When summed across urban case study areas, a standard level of 65 ppb is
15   estimated to reduce O_3-associated total mortality by approximately 11% (2007
16   model year) and 8% (2009 model year), compared to the current standard. For
17   days with area-wide concentrations at or above 40 ppb, a standard level of 65 ppb
18   is estimated to reduce O_3-associated total mortality by almost 40% (2007 and
19   2009 model years). For days with area-wide concentrations at or above 60 ppb, a
20   standard level of 65 ppb is estimated to reduce O_3-associated total mortality by
21   over 80% (2007 and 2009 model years).
22
23 2. Respiratory hospital admissions associated with short-term O_3 concentrations: Compared to
24   the current standard, changes in total risk estimated for a standard level of 65 ppb are similar
25   to the changes in total risk estimated for total mortality (U.S. EPA, 2014, chapter 7).
26
27 3. Respiratory mortality associated with long-term O_3 concentrations: A standard level of 65
28   ppb reduces total risk, compared to the current standard. Across most urban case study areas,
29   risks are estimated to decrease by up to approximately 10%. In one area (New York City),
30   risks are estimated to decrease by up to approximately 22%. Risk reductions are estimated
31   across all urban case study areas and in both model years evaluated, with larger reductions
32   estimated for 2007 (i.e., the model year with generally higher O_3-associated risks).

33   Based on Figures 4-9 to 4-12 and the associated details described in the second draft
34 HREA (U.S. EPA 2014, chapter 7), we take note of the following for a standard level of 60 ppb:

35 1. Total mortality associated with short-term O_3 concentrations:
36   a. A standard level of 60 ppb is estimated to reduce total risk, compared to the
37   current standard, in all urban case study areas. Across urban case study areas,

---

[22]As discussed in the second draft HREA (U.S. EPA, 2014, section 4.5), the New York and Los Angeles urban case study areas required the largest reductions in NO_X in order to meet the existing and potential alternative standards. The HDDM-based O_3 estimates become more uncertain for larger changes in precursor emissions, and the HREA notes less overall confidence in results for New York and Los Angeles.

1   risks are estimated to decrease by up to approximately 15%. Estimated risk
2   reductions are larger for the model year with generally higher O_3-associated risks
3   (2007).
4
5   b. When summed across urban case study areas, a standard level of 60 ppb is
6   estimated to reduce O_3-associated total mortality by approximately 14% (2007
7   model year) and 10% (2009 model year), compared to the current standard. For
8   days with area-wide concentrations at or above 40 ppb, a standard level of 60 ppb
9   is estimated to reduce O_3-associated total mortality by approximately 50% (2007
10   and 2009 model years). For days with area-wide concentrations at or above 60
11   ppb, a standard level of 60 ppb is estimated to reduce O_3-associated mortality by
12   over 95% (2007 and 2009 model years).
13
14 2. Respiratory hospital admissions associated with short-term O_3 concentrations: Compared to
15   the current standard, changes in total risk estimated for a standard level of 60 ppb are similar
16   to the changes in total risk estimated for total mortality (U.S. EPA, 2014, chapter 7).
17
18 3. Respiratory mortality associated with long-term O_3 concentrations: A standard level of 60
19   ppb reduces total risk, compared to the current standard. Across urban case study areas, risks
20   are estimated to decrease by up to approximately 17%. Risk reductions are estimated across
21   all urban case study areas and in both model years evaluated, with larger reductions
22   estimated for 2007 (i.e., the model year with generally higher O_3-associated risks).

23   In further considering these risk estimates, we take note of the associated uncertainties, as
24 discussed in more detail in section 3.2.3.2 of this second draft PA. These include (1) the national
25 representativeness of urban case study areas in terms of the O_3 response to reductions in NO_X
26 emissions; (2) the shape of the concentration-response function at lower ambient concentrations;
27 (3) the use of concentration-response relationships developed for particular populations in
28 particular locations to estimate health risks in different populations and locations; (4) the
29 applications of concentration-response relationships to model-adjusted air quality, given the
30 altered spatial/temporal patterns of ambient O_3 and the potential for increases in relatively low
31 O_3 concentrations to increase risk estimates; and (5) the possibility for reductions in risk
32 associated with reductions in PM and/or NO_2 resulting from control of NO_X.

33 **4.5   CASAC ADVICE**
34   In the fall of 2011, rather than revising the O_3 NAAQS as part of the reconsideration
35 process, EPA coordinated further proceedings on the reconsideration rulemaking with the current
36 ongoing periodic review. Accordingly, in this section we are briefly describing CASAC advice
37 from the reconsideration of the 2008 final decision as well as CASAC advice received during the
38 current review as it pertains to potential alternative standards.

1   Consistent with their advice in 2008, CASAC reiterated during the reconsideration its

2   support for an 8-hour primary O_3 standard with a level ranging from 60 to 70 ppb, combined

3   with the current form. Specifically, in response to EPA's solicitation of their advice during the

4   reconsideration, the CASAC letter (Samet 2010) to the Administrator stated:

5   *CASAC fully supports EPA's proposed range of 0.060 – 0.070 parts per million*

6   *(ppm) for the 8-hour primary ozone standard. CASAC considers this range to be*

7   *justified by the scientific evidence as presented in the Air Quality Criteria for*

8   *Ozone and Related Photochemical Oxidants (March 2006) and Review of the*

9   *National Ambient Air Quality Standards for Ozone: Policy Assessment of*

10   *Scientific and Technical Information, OAQPS Staff Paper (July 2007).*

11

12   Similarly, in response to EPA's request for additional advice on the reconsideration in

13   2011, CASAC reaffirmed their conclusion that "the evidence from controlled human and

14   epidemiological studies strongly supports the selection of a new primary ozone standard within

15   the 60 – 70 ppb range for an 8-hour averaging time" (Samet, 2011). CASAC further concluded

16   that this range "would provide little margin of safety at its upper end" (Samet, 2011, p. 2).

17   In the first draft PA, staff concluded that the available evidence provides support for

18   conducting further exposure and risk analyses of potential alternative standard levels in the range

19   of 60 to 70 ppb (USEPA, 2012b). In response, CASAC noted that the draft PA provided "a

20   strong scientific rationale for consideration of ozone levels (8 hour averages) of 60 ppb to 70

21   ppb" (Frey and Samet, 2012).

22   **4.6   PRELIMINARY STAFF CONCLUSIONS ON ALTERNATIVE PRIMARY**
23   **STANDARDS FOR CONSIDERATION**

24   Staff's consideration of alternative primary O_3 standards builds upon our conclusion,

25   discussed in section 3.4, that the overall body of evidence and exposure/risk information calls

26   into question the adequacy of public health protection afforded by the current standard,

27   particularly for at-risk populations. In section 3.4, we further conclude that it is appropriate in

28   this review to consider alternative standards that would increase public health protection,

29   compared to the current standard, and that it is not appropriate to consider alternative standards

30   with levels higher than the current standard, which would decrease public health protection.

31   As an initial matter, for the reasons discussed in section 4.1 above, we conclude it is

32   appropriate to continue using O_3 as the indicator for the standard that protects against exposures

33   to ambient O_3 and other photochemical oxidants. For the reasons discussed in sections 4.2 and

34   4.3 above, we also conclude that it is appropriate for the Administrator to consider retaining the

35   current averaging time and form. In the remainder of this section, we present our more focused

36   discussion on the range of alternative levels that, in our judgment, it is appropriate for the

37   Administrator to consider.

1   For a standard that is defined in terms of the current indicator, averaging time, and form,
2   we reach the conclusion that, depending on the public health policy judgments made by the
3   Administrator, the evidence and information available in this review supports consideration of
4   alternative levels from 70 down to 60 ppb (section 4.4, above).[23] Compared to the current
5   standard, a revised standard with a level from 70 to 60 ppb would be expected to increase public
6   health protection against both short- and long-term O_3 exposures, including for members of at-
7   risk populations. The scientific evidence and exposure/risk estimates that could support revised
8   standards with levels from the upper, middle, and lower portions of this range are summarized
9   below, with a specific focus on levels of 70 ppb, 65 ppb, and 60 ppb. Key exposure/risk
10   information is summarized in Tables 4-4 and 4-5, and Figure 4-13.
11

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[23]As discussed in sections 3.1.2, 3.2, and 3.4 of this second draft PA, we further conclude that it would not be
appropriate to consider a standard level higher than 75 ppb, which would decrease public health protection
compared to the current standard.

1 **Table 4-4.** **Summary of Estimated Exposures of Concern for Potential alternative**
2 **standard levels of 70, 65, and 60 ppb in Urban Case Study Areas[24]**

Benchmark Level	Alternative Standard Level (ppb)	Average % Children Exposed[25]	Number of Children (5 to 18 years) [Number of Asthmatic Children][26]	Average % Reduction from Current Standard	% Children - Worst Year and Worst Area
One or more exposures of concern per season					
≥ 70 ppb	70	0.1-1.2	95,000 [10,000]	73	3.2
	65	0-0.2	16,000 [1,600]	95	0.5
	60	0	0 [0]	100	0.1
≥ 60 ppb	70	3.3-10.2	1,177,000 [122,000]	46	18.9
	65	0-4.2	392,000 [40,000]	80	9.5
	60	0-1.2	69,000 [7,000]	96	2.2
Two or more exposures of concern per season					
≥ 70 ppb	70	0-0.1	3,000 [360]	95	0.4
	65	0	0 [0]	100	0
	60	0	0 [0]	100	0
≥ 60 ppb	70	0.5-3.5	319,000 [33,000]	61	9.2
	65	0-0.8	65,000 [7,000]	92	2.8
	60	0-0.2	3,800 [400]	100	0.3

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4

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[24]As illustrated above in Figures 4-1 to 4-4, all alternative standard level s evaluated in the HREA were effective at limiting exposures of concern at or above 80 ppb. Therefore, Table 4-4 focuses on exposures of concern at or above the 70 and 60 ppb benchmark concentrations.

[25]Estimates for each urban case study area were averaged for the years evaluated in the second draft HREA (2006 to 2010). Ranges reflect the ranges across urban case study areas.

[26]Numbers of children exposed in each urban case study area were averaged over the years 2006 to 2010. These averages were then summed across urban case study areas. Numbers are rounded to nearest thousand unless otherwise indicated.

**Table 4-5.   Summary of Estimated Lung Function Decrements for Potential alternative
standard levels of 70, 65, and 60 ppb in Urban Case Study Areas**

Lung Function Decrement	Alternative Standard Level	Average % Children[27]	Number of Children (5 to 18 years) [Number of Asthmatic Children][28]	Average % Reduction from Current Standard	% Children Worst Year and Area
One or more decrements per season					
≥ 10%	70	11-17	2,547,000 [263,000]	15	20
	65	3-15	1,931,000 [195,000]	31	18
	60	5-11	1,392,000 [139,000]	45	13
≥ 15%	70	2-4	564,000 [58,000]	26	5
	65	0-3	355,000 [36,000]	50	4
	60	1-2	224,000 [22,000]	67	3
≥ 20%	70	1-2	189,000 [20,000]	32	2.1
	65	0-1	107,000 [11,000]	59	1.4
	60	0-1	57,000 [6,000]	77	0.7
Two or more decrements per season					
≥ 10%	70	5.5-11	1,418,000 [146,000]	17	13
	65	1.3-8.8	1,020,000 [102,000]	37	11
	60	2.1-6.4	743,000 [74,000]	51	7.3
≥ 15%	70	0.9-2.4	274,000 [28,000]	29	3.1
	65	0.1-1.8	169,000 [17,000]	54	2.3
	60	0.2-1.0	100,000 [10,000]	71	1.4
≥ 20%	70	0.3-0.8	84,000 [9,000]	34	1.1
	65	0-0.5	40,000 [4,000]	66	0.8
	60	0-0.2	22,000 [2,000]	83	0.4

[27]Estimates in each urban case study area were averaged for the years evaluated in the second draft HREA (2006 to 2010). Ranges reflect the ranges across urban case study areas.

[28]Numbers of children estimated to experience decrements in each study urban case study area were averaged over 2006 to 2010. These averages were then summed across urban case study areas. Numbers are rounded to nearest thousand unless otherwise indicated.

1   **Figure 4-13. Estimates of Total Mortality Attributable to Days with 8-Hour Area-Wide O$_3$**
2   **Concentrations at or above 20, 40, or 60 ppb - Risks Summed Across Urban**
3   **Case Study Areas and Expressed Relative to 75 ppb**
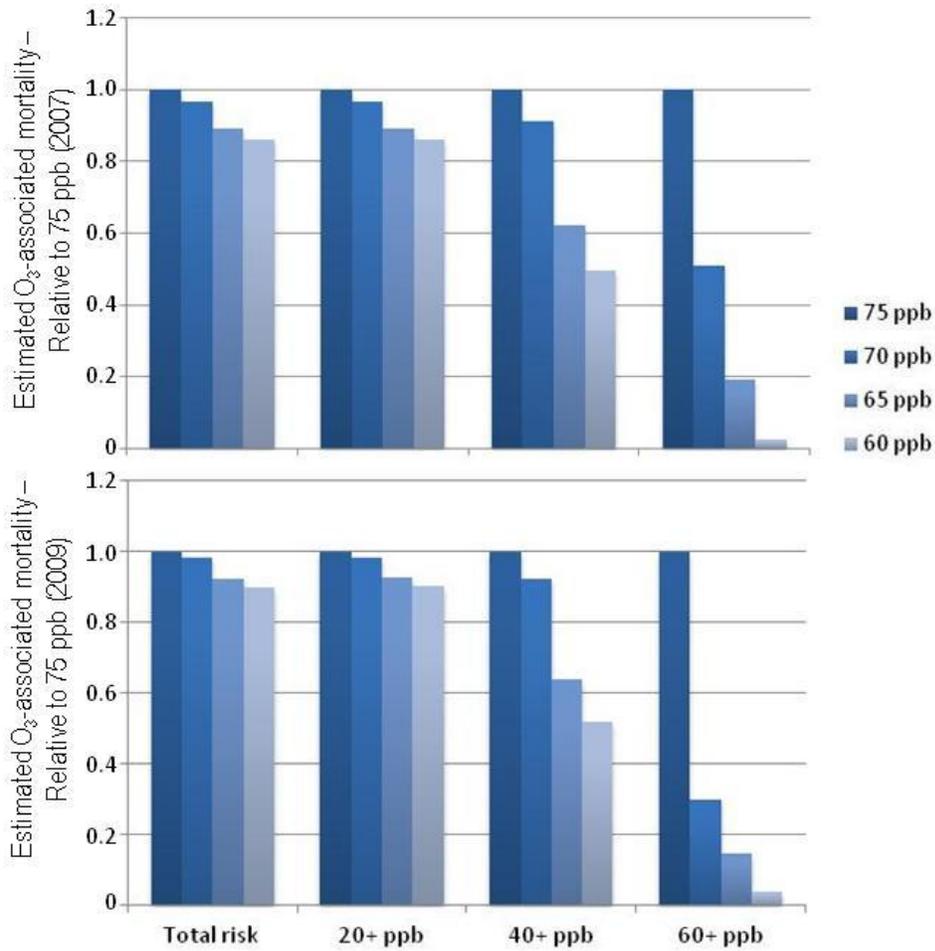
4
5

1   As an initial observation, we note that controlled human exposure studies provide the
2   most certain evidence indicating the occurrence of health effects in humans following exposures
3   to specific O_3 concentrations. As discussed above and in section 3.1.2.1, controlled human
4   exposure studies have reported a variety of respiratory effects in healthy adults following
5   exposures to O_3 concentrations of 60, 70, or 80 ppb, and higher. The largest respiratory effects,
6   and the broadest range of effects, have been reported following exposures to 80 ppb O_3 or higher,
7   in part because most exposure studies have been conducted at these higher concentrations.
8   Exposures to O_3 concentrations of 80 ppb or higher have been reported to decrease lung function,
9   increase airway inflammation, increase respiratory symptoms, result in airway
10   hyperresponsiveness, and decrease lung host defenses in healthy adults. Most of these effects
11   have also been reported in healthy adults following exposures to lower O_3 concentrations.[29]
12   Exposures to O_3 concentrations of 70 ppb have been reported to decrease lung function and
13   increase respiratory symptoms, a combination that meets the ATS criteria for an "adverse"
14   response (section 3.1.3). Exposures to O_3 concentrations of 60 ppb have been demonstrated to
15   decrease lung function, with decrements in some individuals large enough to be judged an
16   abnormal response by ATS, and which could be adverse in individuals with lung disease.
17   Exposures to O_3 concentrations of 60 ppb have also been reported in one study to increase
18   airway inflammation, which provides a mechanism by which O_3 may cause other more serious
19   respiratory effects (e.g., asthma exacerbations).

20   Compared to the current standard, the second draft HREA estimates that a revised
21   standard with a level of 70 ppb would reduce exposures of concern to O_3 concentrations of 60,
22   70, and 80 ppb, with such a standard level estimated to be most effective at limiting exposures at
23   or above the higher health benchmark concentrations. On average over the years 2006 to 2010, a
24   standard with a level of 70 ppb is estimated to allow only up to about 1% of children (i.e., ages 5
25   to 18) to experience exposures of concern at or above 70 ppb (73% reduction, compared to
26   current standard), and far less than 1% to experience two or more such exposures (95%
27   reduction, compared to current standard). In the worst-case location and year (i.e., location and
28   year with the largest exposure estimate), about 3% of children are estimated to experience one or
29   more exposures of concern at or above 70 ppb, and less than 1% are estimated to experience two
30   or more. A standard with a level of 70 ppb is estimated to allow far less than 1% of children to
31   experience exposures of concern at or above the 80 ppb benchmark concentration, even in the
32   worst-case year (Table 4-4).

---

[29]Though airway hyperresponsiveness and lung host defense have not been evaluated following exposures to O_3 concentrations below 80 ppb. The extent to which these respiratory effects occur following lower exposure concentrations is not clear from the available evidence, though we have no basis for concluding that an exposure concentration of 80 ppb reflects an effects threshold.

1   A standard with a level of 70 ppb is estimated to allow about 3 to 10% of children to

2   experience one or more exposures of concern at or above 60 ppb in a single O_3 season.

3   Compared to the current standard, this reflects about a 46% reduction, on average across urban

4   case study areas. A standard with a level of 70 ppb is estimated to allow about 1% to 4% of

5   children to experience two or more exposures of concern at or above 60 ppb. In the worst-case

6   location and year, a standard set at 70 ppb is estimated to allow about 19% of children to

7   experience one or more exposures of concern at or above 60 ppb, and 9% to experience two or

8   more such exposures (Table 4-4).

9   Compared to the current standard, the second draft HREA estimates that a revised

10   standard with a level of 70 ppb would also reduce O_3-induced lung function decrements in

11   children. A level of 70 ppb is estimated to be most effective at limiting the occurrences of

12   moderate and large lung function decrements (i.e., FEV_1 decrements $\geq 15\%$ and $\geq 20\%$,

13   respectively). On average over the years 2006 to 2010, a standard with a level of 70 ppb is

14   estimated to allow about 2 to 4% of children to experience one or more moderate O_3-induced

15   lung function decrements (i.e., $\geq 15\%$), which would be of concern for healthy people, and about

16   1 to 2.5% of children to experience two or more such decrements (approximately 30% reduction,

17   compared to the current standard). In the worst-case location and year, up to 5% of children are

18   estimated to experience one or more O_3-induced lung function decrements $\geq 15\%$, and up to 3%

19   are estimated to experience two or more such decrements. A standard set at 70 ppb is estimated

20   to allow about 2% or fewer children to experience large O_3-induced lung function decrements

21   (i.e., $\geq 20\%$), and to allow about 1% or fewer children to experience two or more such

22   decrements, even in the years and locations with the largest exposure estimates (Table 4-5).

23   On average over the years 2006 to 2010, a standard set at 70 ppb is estimated to allow

24   about 11 to 17% of children to experience one or more moderate O_3-induced lung function

25   decrements (i.e., $\geq 10\%$), which could be adverse for people with lung disease. This reflects an

26   average reduction of about 15%, compared to current standard. A standard with a level of 70 ppb

27   is also estimated to allow about 6 to 11% of children to experience two or more such decrements

28   (17% reduction, compared to current standard). In the worst-case location and year, a standard

29   set at 70 ppb is estimated to allow about 20% of children, including asthmatic children, to

30   experience one or more O_3-induced lung function decrements $\geq 10\%$, and 13% to experience two

31   or more such decrements (Table 4-5).

32   With regard to our analyses of epidemiologic studies we note that, compared to the

33   current standard, a revised standard with a level of 70 ppb would be more effective in

34   maintaining short-term ambient O_3 concentrations below those present in locations that provided

35   the basis for positive and statistically significant health effect associations. In particular, the

36   study by Mar and Koenig (2009) reported positive and statistically significant associations with

1   respiratory emergency department visits in Seattle, a location that met the current standard over

2   the entire study period but that would have violated a revised standard with a level of 70 ppb. In

3   addition, most of the Canadian study cities evaluated by Dales et al. (2006), who reported

4   positive and statistically significant associations with respiratory hospital admissions, would

5   have met the current standard over the entire study period but would have violated a revised

6   standard with a level of 70 ppb over at least part of the study period (Table 4-1). Finally,

7   compared to the current standard, fewer of the study cities evaluated by Katsouyanni et al.

8   (2009) (for mortality and hospital admissions) and Bell et al. (for the 30 ppb cut-point) would

9   have met a revised standard with a level of 70 ppb (Tables 4-1, 4-2).[30]

10   With regard to long-term O_3 concentrations, air quality analyses indicate that in 9 out of

11   the 12 urban case study areas a revised standard with a level of 70 ppb would be expected to

12   maintain long-term O_3 concentrations below those where the study by Jerrett et al. (2009)

13   indicates the most confidence in the reported association with respiratory mortality. This is

14   compared to 6 out of 12 areas for the current standard.

15   In further considering the potential implications of epidemiology studies for potential

16   alternative standard levels, we note that the emphasis given to estimates of O_3-associated

17   mortality or morbidity risks will depend in large part on the extent to which key uncertainties in

18   these estimates are emphasized (e.g., uncertainties in applying concentration-response

19   relationships from epidemiologic studies to adjusted air quality with different spatial/temporal

20   distributions of ambient O_3). To the extent emphasis is placed on estimates of O_3-associated

21   mortality and morbidity risks, it could be judged appropriate to focus on estimates of total risk

22   (i.e., based on the full distributions of ambient O_3 concentrations) and/or on estimates of risk

23   associated with O_3 concentrations in the upper portions of ambient distributions.

24   A focus on total risks could be judged appropriate to the extent greater emphasis is placed

25   on the possibility that concentration-response relationships remain linear over the entire

26   distributions of ambient O_3 concentrations, and thus to the extent greater emphasis is placed on

27   the potential for mortality and/or morbidity to be affected by changes in relatively low O_3

28   concentrations (as discussed above and in section 3.2.3.2). When summed across urban case

29   study areas, a standard with a level of 70 ppb is estimated to reduce total mortality associated

30   with short-term O_3 exposures by about 2 to 3%, compared to the current standard (with

31   reductions up to about 5% for individual urban case study areas).[31] Based on a national modeling

32   analysis, the majority of the U.S. population would be expected to experience modest reductions

33   in such risks upon reducing precursor emissions. A standard with a level of 70 ppb is estimated

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[30]Though in the analyses presented in both of these studies, the majority of cities evaluated would have met a standard with a level of 70 ppb over the entire study periods.

[31]Similar changes are estimated for respiratory morbidity associated with short-term O_3 concentrations.

1   to reduce respiratory mortality associated with long-term O_3 concentrations by up to about 8% in

2   urban case study areas. For both short- and long- term metrics, risk reductions are larger, and are

3   estimated more consistently across urban case study areas, in a year with relatively higher O_3

4   concentrations.

5   A focus on risks associated with O_3 concentrations in the upper portions of ambient

6   distributions may reasonably be judged appropriate in light of the overall evidence indicating

7   increasing magnitude, severity, and incidence of O_3-attributable effects as exposure

8   concentrations increase, as well as the greater uncertainty associated with the shapes of

9   concentration-response curves for O_3 concentrations in the lower portions of ambient

10 distributions (section 3.2.3.2 and 3.4). There is no single ambient concentration below which

11 uncertainty in O_3-attributable effects increases notably in all locations, for all health endpoints,

12 and in all populations. Therefore, we consider the distribution of mortality associated with

13 various portions of the distribution of area-wide O_3 concentrations (Figure 4-13, above).

14   For days with area-wide concentrations at or above 20 ppb, a standard with a level of 70

15 ppb is estimated to reduce mortality associated with short-term O_3 exposures by about 2 to 3%

16 compared to the current standard, when O_3-associated deaths are summed across urban case

17 study areas. For days with area-wide concentrations at or above 40 ppb, a standard with a level

18 of 70 ppb is estimated to reduce mortality associated with short-term O_3 exposures by about 8 to

19 9% compared to the current standard. For days with area-wide concentrations at or above 60 ppb,

20 a standard with a level of 70 ppb is estimated to reduce O_3-associated mortality by about 50% to

21 70% (Figure 4-13).[32]

22   Based on all of the above considerations, we conclude that a standard with a level of 70

23 ppb would be expected to provide additional incremental protection over that provided by the

24 current O_3 standard. A level of 70 ppb could be supported to the extent more emphasis is placed

25 on the public health importance of higher O_3 exposure concentrations (e.g., $\geq$ 70, 80 ppb), larger

26 O_3-induced lung function decrements (e.g., $\geq$ 15, 20%), and estimates of multiple occurrences of

27 exposures of concern and O_3-induced lung function decrements. In addition, a revised standard

28 with a level of 70 ppb would be expected to be more effective than the current standard at

29 maintaining short- and long-term ambient O_3 concentrations below those where we have the

30 most confidence in associations with mortality and morbidity. Overall across the U.S., such a

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[32]Fewer than 10% of total O_3-associated deaths are attributable to days with such high area-wide concentrations, due to the relatively small number of days with area-wide concentrations of 60 ppb or above.

1 standard would also be expected to reduce risks of O_3-associated mortality and morbidity,

2 particularly the portions of the risk associated with relatively high ambient concentrations.[33]

3   Next, we consider a standard with a level of 65 ppb. A level of 65 ppb is well-below the

4 lowest O_3 exposure concentration (i.e., 80 ppb) that has been reported to elicit a wide range of

5 effects, including: lung function decrements, airway inflammation, respiratory symptoms, airway

6 hyperresponsiveness, and decreased lung host defense in healthy adults, as noted above. A

7 standard level of 65 ppb is also somewhat below the lowest exposure concentration at which the

8 combined occurrence of respiratory symptoms and lung function decrements has been reported

9 (i.e., 70 ppb), a combination judged adverse by the ATS (section 3.1.3). A level of 65 ppb is

10 above the lowest exposure concentration demonstrated to result in lung function decrements

11 large enough to be judged an abnormal response by ATS, and that could be adverse in

12 individuals with lung disease. A level of 65 ppb is also above the lowest exposure concentration

13 at which pulmonary inflammation has been reported (i.e., 60 ppb).

14   Compared to the current standard and a revised standard with a level of 70 ppb, the

15 second draft HREA estimates that a standard with a level of 65 ppb would reduce exposures of

16 concern to the range of O_3 benchmark concentrations analyzed (i.e., 60, 70, and 80 ppb). The

17 HREA estimates that meeting a standard with a level of 65 ppb would eliminate exposures of

18 concern at or above 80 ppb in urban case study areas. Such a standard is estimated to allow far

19 less than 1% of children to experience one or more exposures of concern at or above the 70 ppb

20 benchmark level, even in the years and locations with the largest exposure estimates, and is

21 estimated to eliminate the occurrence of two or more exposures at or above 70 ppb (Table 4-4).

22   In addition, on average over the years 2006 to 2010, a standard with a level of 65 ppb is

23 estimated to allow between 0 and about 4% of children in urban case study areas to experience

24 exposures of concern at or above 60 ppb. This reflects an 80% reduction (on average across

25 areas), relative to the current standard. A standard with a level of 65 ppb is estimated to allow

26 less than 1% of children to experience two or more exposures of concern at or above 60 ppb (>

27 90% reduction, compared to current standard). In the worst-case location and year, about 10% of

28 children are estimated to experience one or more exposures of concern at or above 60 ppb, with

29 about 3% estimated to experience two or more such exposures (Table 4-4).

30   Compared to the current standard and a revised standard with a level of 70 ppb, the

31 second draft HREA estimates that a standard with a level of 65 ppb would also reduce the

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[33]In reaching this conclusion we recognize that, as discussed in detail in chapter 3 (sections 3.2.1 and 3.2.3.2), reducing NO_X emissions to meet alternative O_3 standards could result in increases in relatively low ambient concentrations. When we consider the epidemiologic-based estimates in light of all of the health effects evidence, and the considerations discussed more fully in section 3.2.3.2 above, we have greater certainty in the health benefits of reducing high ozone concentrations, and appreciable uncertainty regarding estimates of risk at lower concentrations. Accordingly, we judge that the range of levels discussed here is appropriate.

1 occurrence of O_3-induced lung function decrements. A level of 65 ppb is estimated to allow
2 about 4% or less of children to experience O_3-induced lung function decrements $\geq$ 15% (50%
3 reduction, compared to current standard), even considering the worst-case location and year.
4 Such a standard is estimated to allow about 2% or less of children to experience two or more
5 such decrements. A standard set at 65 ppb is estimated to allow about 1% or less of children to
6 experience large O_3-induced lung function decrements (i.e., $\geq$ 20%), even in the worst-case year
7 and location (Table 4-5).
8   On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to
9 allow about 3 to 15% of children to experience one or more moderate O_3-induced lung function
10 decrements (i.e., $\geq$ 10%), which could be adverse for people with lung disease. This reflects an
11 average reduction of about 30%, relative to current standard. A standard with a level of 65 ppb is
12 also estimated to allow about 1 to 9% of children to experience two or more such decrements
13 (37% reduction, compared to current standard). In the worst-case location and year, a standard
14 set at 65 ppb is estimated to allow up to about 18% of children to experience one or more O_3-
15 induced lung function decrements $\geq$ 10%, and up to 11% to experience two or more such
16 decrements (Table 4-5).
17   With regard to O_3 epidemiologic studies we note that, compared to the current standard
18 and a standard with a level of 70 ppb, a revised standard with a level of 65 ppb would be more
19 effective in maintaining short-term ambient O_3 concentrations below those present in locations
20 that provided the basis for positive and statistically significant health effect associations. In
21 addition to the studies by Mar and Koenig (2009) and Dales et al. (2006) (discussed above for a
22 level of 70 ppb), a revised standard with a level of 65 ppb would not allow the ambient O_3
23 concentrations that provided the basis for mortality associations in most of the Canadian study
24 cities evaluated by Katsouyanni et al. (2009) (Table 4-1).[34] In addition, fewer of the study cities
25 evaluated by Bell et al. (for the 30 ppb cut-point) would have met a revised standard with a level
26 of 65 ppb (Table 4-2).[35]
27   With regard to long-term O_3 concentrations, air quality analyses indicate that in 10 out of
28 the 12 urban case study areas a revised standard with a level of 65 ppb would be expected to
29 maintain long-term O_3 concentrations below those where the study by Jerrett et al. (2009)
30 indicates the most confidence in the reported association with respiratory mortality. This is
31 compared to 6 out of 12 areas for the current standard and 9 out of 12 for a standard with a level
32 of 70 ppb (Table 4-3).

[34]As discussed above, most of the study cities evaluated by Katsouyanni et al. (2009) for mortality would have met the current standard and a revised standard with a level of 70 ppb.

[35]Though the majority of cities evaluated, based on the 30 ppb cut point analysis, would have met a standard with a level of 65 ppb over the entire study period (Table 4-2).

1   Compared to the current standard and a revised standard with a level of 70 ppb, the
2   second draft HREA estimates that a revised standard with a level of 65 ppb would generally
3   reduce O_3-associated mortality and morbidity risks based on estimates that use the full
4   distributions of ambient O_3 concentrations. When summed across urban case study areas, a
5   standard with a level of 65 ppb is estimated to reduce total mortality associated with short-term
6   O_3 exposures by about 10%, compared to the current standard (Figure 4-13). Risks of respiratory
7   mortality associated with long-term exposures were reduced by up to about 10%, compared to
8   the current standard. As with a level of 70 ppb discussed above, reductions are estimated to be
9   larger and more consistent across urban case study areas for the year with relatively higher
10 ambient O_3 concentrations.
11   Similar to a standard with a level of 70 ppb, risk estimates indicate that a standard with a
12 level of 65 ppb more effectively reduces mortality associated with the upper portions of
13 distributions of ambient O_3 concentrations. For days with area-wide concentrations at or above
14 40 ppb, a standard level of 65 ppb is estimated to reduce O_3-associated total mortality by about
15 40%, when summed across cities. For days with area-wide concentrations at or above 60 ppb, a
16 standard level of 65 ppb is estimated to reduce O_3-associated total mortality by more than 80%
17 (Figure 4-13).
18   In summary, the rationale to support a level of 65 ppb would emphasize the potential
19 public health significance of exposures of concern to 60 and 70 ppb O_3, as well as 80 ppb, and of
20 O_3-attributable health effects that could occur across the range of ambient O_3 concentrations.
21 Compared to a level of 70 ppb, a level of 65 ppb would place more emphasis on evidence from
22 controlled human exposure studies showing lung function decrements and pulmonary
23 inflammation in healthy adults at 60 ppb O_3. A standard with a level of 65 ppb would also place
24 more emphasis on the potential for mortality and morbidity to be caused by the relatively low
25 ambient O_3 concentrations present in locations that would have met a standard with a level of 70
26 ppb, and on the potential for further reductions in health risks, beyond those estimated for a level
27 of 70 ppb. In addition, compared to a standard with a level of 70 ppb, a level of 65 ppb would
28 place relatively less emphasis on the uncertainties associated with the evidence for O_3-
29 attributable health effects at lower exposure and ambient concentrations, and less emphasis on
30 uncertainties in the public health significance of O_3 exposure and risk estimates for lower
31 ambient concentrations.
32   We next consider a standard with a level of 60 ppb. A level of 60 ppb is well-below the
33 lowest O_3 exposure concentration that has been reported to elicit a wide range of potentially
34 adverse respiratory effects in healthy adults (i.e., 80 ppb), as noted above. A level of 60 ppb is
35 also well-below the concentration where the combined occurrence of respiratory symptoms and
36 lung function decrements was observed, a combination judged adverse by the ATS (discussed in

1   section 3.1.3). A level of 60 ppb corresponds to the lowest exposure concentration demonstrated
2   to result in lung function decrements large enough to be judged an abnormal response by ATS,
3   and that could be adverse in individuals with lung disease. A level of 60 ppb also corresponds to
4   the lowest exposure concentration at which pulmonary inflammation has been reported.
5   Based on the HREA analyses of O_3 exposures of concern, a standard with a level of 60
6   ppb is estimated to eliminate exposures of concern at or above the 70 and 80 ppb benchmark
7   concentrations and to be more effective than higher standard levels at limiting exposures of
8   concern at or above 60 ppb. On average over the years 2006 to 2010, a standard with a level of
9   60 ppb is estimated to allow between 0 and about 1% of children in urban case study areas to
10 experience exposures of concern at or above 60 ppb. This reflects a 96% reduction (on average
11 across areas), compared to the current standard. A standard with a level of 60 ppb is estimated to
12 allow virtually no children to experience two or more exposures of concern at or above 60 ppb.
13 In the location and year with the highest exposure estimate, about 2% of children are estimated
14 to experience exposures of concern at or above 60 ppb, with far less than 1% estimated to
15 experience two or more such exposures (Table 4-4).
16   Based on the HREA analyses of O_3-induced lung function decrements, a standard with a
17 level of 60 ppb would be expected to be more effective than a level of 70 or 65 ppb at limiting
18 the occurrence of O_3-induced lung function decrements. A standard with a level of 60 ppb is
19 estimated to allow about 2% or less of children to experience one or more O_3-induced lung
20 function decrements $\geq 15\%$ (almost 70% reduction, compared to current standard), and about 1%
21 or less to experience two or more such decrements (3% in the location and year with the largest
22 estimates). A standard set at 60 ppb is estimated to allow about 1% or less of children to
23 experience large O_3-induced lung function decrements (i.e., $\geq 20\%$), even in the worst-case
24 locations and year (Table 4-5).
25   On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to
26 allow about 5 to 11% of children, including asthmatic children, to experience one or more O_3-
27 induced lung function decrements $\geq 10\%$. This reflects an average reduction of about 45%,
28 compared to current standard. A standard with a level of 60 ppb is also estimated to allow about
29 2 to 6% of children to experience two or more such decrements (51% reduction, compared to
30 current standard). In the worst-case location and year, a standard set at 60 ppb is estimated to
31 allow up to about 13% of children to experience one or more O_3-induced lung function
32 decrements $\geq 10\%$, and 7% to experience two or more such decrements (Table 4-5).
33   A revised standard with a level of 60 ppb would be more effective than a level of 70 or
34 65 ppb at maintaining short-term ambient O_3 concentrations below those present in U.S. and
35 Canadian epidemiologic study locations during study periods. Specifically, in all of the U.S. and
36 Canadian epidemiologic studies evaluated, the majority of study cities had ambient O_3

1 concentrations that would likely have violated a standard with a level of 60 ppb. This includes
2 the studies noted above, for which the majority of study cities would have met the current
3 standard or a revised standard with a level of either 70 or 65 ppb (Tables 4-1 and 4-2).
4   With regard to long-term O_3 concentrations, air quality analyses indicate that in all of the
5 urban case study areas evaluated a revised standard with a level of 60 ppb would be expected to
6 maintain long-term O_3 concentrations below those where the study by Jerrett et al. (2009)
7 indicates the most confidence in the reported association with respiratory mortality. This is
8 compared to 6 out of 12 areas for the current standard, 9 out of 12 for a standard with a level of
9 70 ppb, and 10 out of 12 for a standard with a level of 65 ppb (Table 4-3).
10   A standard with a level of 60 ppb would also be estimated to reduce epidemiology-based
11 mortality and morbidity risks, compared to the current standard and potential alternative
12 standards with levels of 70 or 65 ppb. Across urban case study areas, a standard set at 60 ppb is
13 estimated to reduce total mortality associated with short-term O_3 concentrations by up to 15%,
14 and respiratory mortality associated with long-term O_3 concentrations by up to 17%. Estimated
15 risk reductions are larger for the model year with generally higher O_3-associated risks (2007).
16 When summed across urban case study areas, a standard set at 60 ppb is estimated to reduce total
17 O_3-associated mortality by about 10% (2009) and 14% (2007), compared to the current standard
18 (Figure 4-13).
19   As for standard levels of 70 and 65 ppb, risk estimates indicate that a standard with a
20 level of 60 ppb more effectively reduces mortality associated with the upper portions of
21 distributions of ambient O_3 concentrations. For days with area-wide concentrations at or above
22 40 ppb, a standard set at 60 ppb is estimated to reduce O_3-associated total mortality by
23 approximately 50%. For days with area-wide concentrations at or above 60 ppb, a standard level
24 of 60 ppb is estimated to reduce O_3-associated total mortality by over 95% (Figure 4-13).
25   Overall we note that, compared to a standard with a level of 70 or 65 ppb, a level of 60
26 ppb would place relatively more emphasis on the potential public health significance of the O_3
27 exposures and O_3-attributable health effects that could occur at lower ambient concentrations. A
28 standard with a level of 60 ppb would reflect placing a relatively large emphasis on the evidence
29 from controlled human exposure studies reporting lung function decrements and pulmonary
30 inflammation in some healthy adults following exposures to 60 ppb O_3, and relatively little
31 emphasis on the uncertainties associated with the public health significance of these effects. A
32 standard with a level of 60 ppb would also emphasize the importance of the small number of
33 studies that reported health effect associations in locations that would have met a revised
34 standard with a level of 65 ppb (Table 4-1), and on the potential for further reductions in health
35 risks, beyond those estimated for a level of 65 ppb. In addition, compared to a standard with a
36 level of 65 ppb, a level of 60 ppb would place relatively little emphasis on the uncertainties

1   associated with the evidence for O_3-attributable health effects at lower exposure and ambient

2   concentrations, and little emphasis on uncertainties in the public health significance of O_3

3   exposure and risk estimates for lower ambient concentrations.

4   Based on our consideration of the available scientific evidence and exposure/risk

5   information, we also conclude that standard levels below 60 ppb are not appropriate for

6   consideration in this review. In reaching this conclusion, we take particular note of the following:

7   • The HREA estimates that meeting a standard with a level from 70 to 60 ppb would
8   effectively reduce exposures of concern and lung function decrements. In particular, a
9   level from the low end of this range could virtually eliminate exposures of concern, even
10   for the lowest health benchmark concentration supported by the available controlled
11   human exposure evidence (i.e., 60 ppb). To the extent lower exposure concentrations may
12   result in adverse respiratory effects in some individuals, a standard level from 70 to 60
13   ppb (particularly at or near 60 ppb) would be expected to also reduce exposures to O_3
14   concentrations somewhat below 60 ppb.

16 • A revised standard with a level from 70 to 60 ppb would be more effective than the
17   current standard at maintaining short- and long-term ambient O_3 concentrations below
18   those in locations that provided the basis for positive and statistically significant health
19   effect associations in epidemiologic studies. In particular, in all of the U.S. and Canadian
20   epidemiologic studies evaluated, the majority of study cities had ambient O_3
21   concentrations that would likely have violated a standard with a level of 60 ppb.

23 • To the extent emphasis is placed on epidemiology-based mortality and/or morbidity risk
24   estimates, meeting a standard with a level from 70 to 60 ppb would generally reduce such
25   risks across the majority of the U.S. This is particularly the case for risks associated with
26   ambient O_3 concentrations from the upper portions of seasonal distributions. Given that
27   uncertainty in quantitative estimates of O_3-associated mortality and morbidity increases
28   with the magnitude of NO_X reductions required to simulate potential alternative
29   standards, risk estimates associated with standard levels below 60 ppb would be
30   associated with increasing uncertainty.

31   Given the above, we conclude that, compared to standard levels from 70 to 60 ppb, the

32 extent to which standard levels below 60 ppb could result in further public health improvements

33 becomes notably less certain. Therefore, we conclude that it is not appropriate to consider

34 standard levels below 60 ppb in this review.

35 **4.7   KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND**
36 **DATA COLLECTION**

37   It is important to highlight the uncertainties associated with establishing standards for O_3

38 during and after completion of the NAAQS review process. Research needs go beyond what is

39 necessary to understand health effects, population exposures, and risks of exposure for purposes

1   of setting standards. Research can also support the development of more efficient and effective

2   control strategies. In this section, we highlight areas for future health-related research, model

3   development, and data collection activities to address these uncertainties and limitations in the

4   current body of scientific evidence.

5   As has been presented and discussed in the ISA, particularly chapters 4 through 7, the

6   scientific body of evidence informing our understanding of health effects associated with long-

7   and short-term exposures to O_3 has been broadened and strengthened since the O_3 NAAQS

8   review completed in 2008. Still, we have concluded that O_3 health research needs and priorities

9   have not changed substantially since the 2007 O_3 Staff Paper (EPA 2007). Key uncertainties and

10   research needs that continue to be high priority for future reviews of the health-based standards

11   are identified below:

12   (1) An important aspect of risk characterization and decision making for air quality

13   standard levels for the O_3 NAAQS is the characterization of the shape of exposure-response

14   functions for O_3, including the identification of potential population threshold levels. Recent

15   controlled human exposure studies of measurable lung function effects provide evidence for a

16   smooth dose-response curve without evidence of a threshold for exposures between 40 and 120

17   ppb O_3 (US EPA, 2013, Figure 6-1). Considering the importance of estimating health risks in the

18   range below 80 ppb O_3, additional research is needed to evaluate responses in healthy and

19   especially asthmatic individuals in the range of 40 to 70 ppb for 6-8 hr exposures while engaged

20   in moderate exertion.

21   (2) Similarly, for health endpoints reported in epidemiologic studies such as hospital

22   admissions, ED visits, and premature mortality, an important aspect of characterizing risk is the

23   shape of concentration-response functions for O_3, including identification of potential population

24   threshold levels. Most of the recent studies and analyses continue to show no evidence for a clear

25   threshold in the relationships between O_3 concentrations commonly observed in the U.S. during

26   the O_3 season and these health endpoints, though evidence indicates less certainty in the shape of

27   the concentration-response curve at the lower end of the distribution of O_3 concentrations.

28   However, there continues to be heterogeneity in the O_3-mortality relationship across cities (or

29   regions), including effect modifiers that are also expected to vary regionally, which are sources

30   of uncertainty. Additionally, whether or not exposure errors, misclassification of exposure, or

31   potential impacts of other copollutants may be obscuring potential population thresholds is still

32   unknown.

33   (3) The extent to which the broad mix of photochemical oxidants and more generally

34   other copollutants in the ambient air (e.g., PM, NO_2, SO_2, etc.) may play a role in modifying or

35   contributing to the observed associations between ambient O_3 and various morbidity effects and

36   mortality continues to be an important research question. Ozone has long been known as an

1　indicator of health effects of the entire photochemical oxidant mix in the ambient air and has

2　served as a surrogate for control purposes. A better understanding of sources of the broader

3　pollutant mix, of human exposures, and of how other pollutants may modify or contribute to the

4　health effects of O_3 in the ambient air, and vice versa, is needed to better inform future NAAQS

5　reviews.

6　　　(4) As epidemiologic research has continued to be an important factor in assessing the

7　public health impacts of O_3, methodological issues in epidemiologic studies have received

8　greater visibility and scrutiny. There remains a need to further examine alternative modeling

9　specifications and control of time-varying factors, and to better understand the role of

10　copollutants in the ambient air. Additionally, there remains uncertainty around the role of

11　temperature as a potential confounder or effect modifier in epidemiologic models.

12　　　(5) Recent animal toxicological evidence, combined with limited evidence from

13　controlled human exposure studies of cardiovascular morbidity and epidemiologic studies of

14　cardiovascular mortality, have provided evidence of both direct and indirect effects on the

15　cardiovascular system. However, additional work will need to examine biologically plausible

16　mechanisms of cardiovascular effects, expand upon preliminary evidence from controlled human

17　exposure studies, address inconsistencies observed in epidemiologic studies of cardiovascular

18　morbidity, and determine the extent to which O_3 is directly implicated or works together with

19　other pollutants in causing adverse cardiovascular effects in both at-risk and the general

20　populations.

21　　　(6) Most epidemiologic studies of short-term exposure effects have employed time-series

22　or case-crossover study designs and have been conducted in large populations. These study

23　designs remain subject to uncertainty due to use of ambient fixed-site data serving as a surrogate

24　for ambient exposures, and to the difficulty of determining the impact of any single pollutant

25　among the mix of pollutants in the ambient air. Measurements made at stationary outdoor

26　monitors have been used as independent variables for air pollution, but the accuracy with which

27　these measurements actually reflect subjects' exposure is not yet fully understood. Also,

28　additional research is needed to improve the characterization of the degree to which discrepancy

29　between stationary monitor measurements and actual pollutant exposures introduces error into

30　statistical estimates of pollutant effects in epidemiologic studies.

31　　　(7) Recent studies of "long-term" O_3 often evaluate associations with daily maximum

32　concentrations, averaged over the O_3 season. Research is needed to better understand the extent

33　to which health effects associated with such long-term metrics are attributable to long-term

34　average concentrations versus the repeated occurrence of daily maximum concentrations.

35　　　(8) Improved understanding of human exposures to ambient O_3 and to related

36　copollutants is an important research need. Population-based information on human exposure for

1   healthy adults and children and at-risk populations, including people with asthma, to ambient O_3

2   concentrations, including exposure information in various microenvironments, is needed to better

3   evaluate current and future O_3 exposure models. Such information is needed for sufficient

4   periods to facilitate evaluation of exposure models throughout the O_3 season.

5   (9) Information is needed to improve inputs to current and future population-based O_3

6   exposure and health risk assessment models. Collection of time-activity data over longer time

7   periods is needed to reduce uncertainty in the modeled exposure distributions that form an

8   important part of the basis for decisions regarding NAAQS for O_3 and other air pollutants.

9   Research addressing energy expenditure and associated breathing rates in various population

10 groups, particularly healthy children and children with asthma, in various locations, across the

11 spectrum of physical activity, including sleep to vigorous exertion, is needed.

12   (10) An important consideration in the O_3 NAAQS review is the characterization of

13 background levels. There still remain substantial uncertainties in the characterization of 8-hr

14 daily max O_3 background concentrations. Further research to improve the evaluation of the

15 global and regional models which have been used to characterize estimates of background levels

16 would improve understanding of the role of non-U.S. anthropogenic emissions on O_3 levels over

17 the U.S.

4.8   SUMMARY OF PRELIMINARY STAFF CONCLUSIONS ON PRIMARY STANDARD

18 **4.8   SUMMARY OF PRELIMINARY STAFF CONCLUSIONS ON PRIMARY**
19   **STANDARD**

20   In this section, we summarize our preliminary conclusions regarding the primary O_3

21 standard. Staff conclusions in the final PA will be informed by our consideration of the available

22 scientific evidence as assessed in the ISA, air quality/exposure/risk information assessed in the

23 final HREA, recommendations received from CASAC based on their review of this second draft

24 document, and comments received from members of the public.

25   As an initial matter, as discussed in section 3.4, staff concludes that reducing precursor

26 emissions to achieve O_3 concentrations that meet the current standard will provide important

27 improvements in public health protection. This initial conclusion is based on (1) the strong body

28 of scientific evidence indicating a wide range of adverse health outcomes attributable to

29 exposures to O_3 concentrations found in the ambient air and (2) estimates indicating decreased

30 O_3 exposures and health risks upon meeting the current standard, compared to recent air quality.

31 Strong support for this conclusion is provided by the available health evidence and HREA

32 estimates of O_3 exposures of concern and O_3-induced lung function risks. Some support for this

33 conclusion is also provided by HREA estimates of O_3-associated mortality and morbidity.

34   In considering the available evidence and information, staff further concludes that the O_3-

35 attributable health effects estimated to be allowed by air quality that meets the current primary

1   standard for O_3 can reasonably be judged important from a public health perspective. Thus, we

2   conclude that the available health evidence and exposure/risk information call into question the

3   adequacy of the public health protection provided by the current standard. This conclusion is

4   based on consideration of the scientific evidence assessed in the ISA, including controlled human

5   exposure and epidemiologic studies, as well as animal toxicology studies; the air quality,

6   exposure, and risk analyses presented in the second draft HREA for air quality that just meets the

7   current standard; and advice received from CASAC in their review of the first draft PA and in

8   previous reviews.

9   In reaching the above conclusion regarding the current standard, we also reach

10 preliminary conclusions for the Administrator's consideration in making decisions on the

11 elements of a potential alternative primary O_3 standard, as summarized below. We recognize that

12 selecting from among potential alternative standards will necessarily reflect consideration of

13 qualitative and quantitative uncertainties inherent in the relevant evidence and in the assumptions

14 of the quantitative exposure and risk assessments. Any such standard should protect public health

15 against health effects associated with exposure to O_3, alone or in combination with related

16 photochemical oxidants, taking into account both evidence-based and exposure- and risk-based

17 considerations, and the nature and degree of uncertainties in such information. In reaching

18 conclusions about these ranges of potential alternative standards for consideration, we are

19 mindful that the Act requires primary standards that, in the judgment of the Administrator, are

20 requisite to protect public health with an adequate margin of safety. The primary standards are to

21 be neither more nor less stringent than necessary. Thus, the Act does not require that primary

22 NAAQS be set at zero-risk levels, but rather at levels that reduce risk sufficiently to protect

23 public health with an adequate margin of safety.

24   The degree of public health protection provided by the standard is due to the collective

25 impact of the elements of the standard, including the indicator, averaging time, level, and form.

26 Staff's preliminary conclusions on each of these elements are summarized below.

27 (1)   It is appropriate to continue to use O_3 as the indicator for a standard that is intended to

28   address effects associated with exposure to O_3, alone or in combination with related

29   photochemical oxidants. Based on the available information staff preliminarily concludes

30   that there is no basis for considering any alternative indicator at this time. Meeting an O_3

31   standard can be expected to provide some degree of protection against potential health

32   effects that may be independently associated with other photochemical oxidants, even

33   though such effects are not discernible from currently available studies indexed by O_3

34   alone. Staff notes that control of ambient O_3 levels is generally understood to provide the

35   best means of controlling photochemical oxidants in general, and thus of protecting

1   against effects that may be associated with individual species and/or the broader mix of
2   photochemical oxidants, independent of effects specifically related to O_3.

3

4   (2)   It is appropriate to continue to use an 8-hour averaging time for the primary O_3 standard.

5   (a)   Staff preliminarily concludes that an 8-hour averaging time remains appropriate
6   for addressing health effects associated with short-term exposures to ambient O_3.
7   An 8-hour averaging time is similar to the exposure periods evaluated in
8   controlled human exposure studies, including recent studies that provide evidence
9   for respiratory effects following exposures to O_3 concentrations below the level of
10   the current standard. In addition, epidemiologic studies provide evidence for
11   health effect associations with 8-hour O_3 concentrations, as well as with 1-hour
12   and 24-hour concentrations. A standard with an 8-hour averaging time (combined
13   with an appropriate standard form and level) would also be expected to provide
14   substantial protection against health effects attributable to 1- and 24-hour
15   exposures.

16

17   (b)   Staff also preliminarily concludes that a standard with an 8-hour averaging time
18   can provide protection against respiratory effects associated with longer term O_3
19   exposures. Analyses in the HREA show that as air quality is adjusted to just meet
20   the current 8-hour standard, most study areas are estimated to experience
21   reductions in respiratory mortality associated with long-term O_3 concentrations,
22   indicating that an O_3 standard with an 8-hour averaging time can reduce
23   respiratory mortality reported to be associated with long-term O_3 concentrations.
24   Moreover, the large majority of the U.S. population lives in locations where
25   reducing NO_X emissions would be expected to result in modest decreases in warm
26   season averages of daily 8-hour ambient O_3 concentrations. This suggests that
27   reductions in precursor emissions in order to meet a standard with an 8-hour
28   averaging time would also be expected to reduce the types of long-term O_3
29   concentrations that have been associated with respiratory morbidity in
30   epidemiologic studies.

31

32   In addition, an analysis in the PA of whether just meeting the current or
33   alternative O_3 standards, with 8-hour averaging times, would be expected to
34   maintain long-term O_3 concentrations (i.e., seasonal average of 1-hour daily max)
35   below those present in most of the cities that provided the basis for a positive and
36   statistically significant association with respiratory mortality (Jerrett et al., 2009).

1   This suggests that a standard with an 8-hour averaging time can maintain seasonal
2   averages of 1-hour daily max O_3 concentrations below those that provided the
3   basis for the association with respiratory mortality (and below the concentration at
4   which study authors noted limited evidence of an effects threshold). Taken
5   together, these analyses suggest that a standard with an 8-hour averaging time,
6   coupled with the current 4th-highest form and an appropriate level, could provide
7   appropriate protection against the long-term O_3 concentrations reported to be
8   associated with respiratory morbidity and mortality.

9

10 (3)   It is appropriate to revise the level of the standard since the evidence and information
11   from the exposure and risk assessments in this review provide strong support for
12   consideration of an O_3 standard with a level that would provide increased health
13   protection for at-risk groups, including people with asthma, especially children; the
14   lifestages of children and older adults; people with certain genetic variants; people with
15   reduced intake of certain nutrients; and outdoor workers against an array of adverse
16   health effects. These health effects range from decreased lung function, pulmonary
17   inflammation, and respiratory symptoms to serious indicators of respiratory morbidity
18   including ED visits and hospital admissions for respiratory causes, respiratory and all-
19   cause, non-accidental mortality. We also conclude that exposures of concern and health
20   risks projected to remain upon meeting the current standard, based on the exposure and
21   risk assessments, are indicative of risks to these populations and lifestages that can
22   reasonably be judged to be important from a public health perspective. This reinforces
23   our conclusion that consideration should be given to revising the level of the standard so
24   as to provide increased public health protection.

25

26   It is appropriate to consider a standard level within the range of 70 ppb to 60 ppb,
27   reflecting our judgment that a standard set within this range could provide an appropriate
28   degree of public health protection and would result in important improvements in
29   protecting the health of at-risk populations and lifestages. Standard levels within this
30   range that were considered in staff analyses of air quality, exposure, and risk include 70,
31   65 and 60 ppb, representative of levels within the upper, middle, and lower parts of this
32   range, respectively. Further, it would not be appropriate to consider increasing the level
33   of the current standard, thereby decreasing public health protection.

34

35 (4)   It is appropriate to continue to use the 4th-highest daily max form of the standard. Staff
36   notes that the 4th-highest daily max was selected in 1997 in recognition of the public

1   health protection provided by this form, when coupled with an appropriate averaging

2   time and level, combined with its stability for implementation programs. The currently

3   available evidence and exposure/risk information does not call into question these

4   conclusions from previous reviews. Therefore, we reach the preliminary conclusion that

5   it is appropriate to consider retaining the current 4th-highest daily max form for an O_3

6   standard with an 8-hour averaging time and a revised level, as discussed above.

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5 ADEQUACY OF THE CURRENT SECONDARY STANDARD

This chapter presents staff's considerations and preliminary conclusions regarding the adequacy of the current secondary O_3 NAAQS. In doing so, we pose the following overarching question:

- **Does the currently available scientific evidence- and exposure/risk-based information, as reflected in the ISA and WREA, support or call into question the adequacy and/or appropriateness of the protection afforded by the current secondary O_3 standard?**

In addressing this overarching question, we pose a series of more specific questions, as discussed in sections 5.1 through 5.5 below. We consider the nature of O_3-induced effects, including the nature of the exposures that drive the biological and ecological response and related biologically-relevant exposure metrics (section 5.1); the scientific evidence and exposure/risk information, including that of associated ecosystem services, regarding (a) tree growth, productivity and carbon storage (section 5.2), (b) crop yield loss (section 5.3), (c) visible foliar injury (section 5.4), and (d) other welfare effects (section 5.5). Section 5.6 describes advice and recommendations received from CASAC.   In section 5.7, we revisit the overarching question of this chapter and present preliminary staff conclusions on the adequacy of the current secondary standard.

5.1 NATURE OF EFFECTS AND BIOLOGICALLY-RELEVANT EXPOSURE METRIC

- **Does the current evidence alter our conclusions from the previous review regarding the nature of O_3-induced welfare effects?**

The current body of O_3 welfare effects evidence confirms the conclusions reached in the last review on the nature of O_3-induced welfare effects and is summarized in the ISA as follows (U.S. EPA, 2013, p. 1-8):

> *The welfare effects of O_3 can be observed across spatial scales, starting at the subcellular and cellular level, then the whole plant and finally, ecosystem-level processes. Ozone effects at small spatial scales, such as the leaf of an individual plant, can result in effects along a continuum of larger spatial scales. These effects include altered rates of leaf gas exchange, growth, and reproduction at the individual plant level, and can result in broad changes in ecosystems, such as productivity, carbon storage, water cycling, nutrient cycling, and community composition.*

1   This body of evidence has been expanding since the phytotoxic effects of O_3 were first

2   identified by Richards, et al in 1958 who showed that "O_3 was a constituent of smog that caused

3   foliar injury on grapes in California" (Flagler, 1998). In the half century that has followed, a

4   large number of studies have been conducted both in and outside of the U.S. to examine the

5   impacts of O_3 on plants and their associated ecosystems. Taken together, these studies

6   demonstrate that O_3 -induced effects that occur at the subcellular and cellular levels, at sufficient

7   magnitudes propagate up to produce larger scale effects that affect the whole organism. In

8   addition, the studies assessed in this review have further increased our understanding of the

9   molecular, biochemical and physiological mechanisms that explain how plants are affected by

10   O_3, in the absence of other stressors, particularly in the area of genomics (U.S. EPA, 2013,

11   Chapter 9, section 9.3). Based on its assessment of this extensive body of science, the ISA

12   determined that a causal relationship exists between exposure to O_3 in ambient air and visible

13   foliar injury effects on vegetation, reduced vegetation growth, reduced productivity in terrestrial

14   ecosystems, reduced yield and quality of agricultural crops and alteration of below-ground

15   biogeochemical cycles (U.S. EPA 2013, Table 1-2). Additionally, the ISA determined that a

16   likely to be causal relationship exists between exposures to O_3 in ambient air and reduced carbon

17   sequestration in terrestrial ecosystems, alteration of terrestrial ecosystem water cycling and

18   alteration of terrestrial community composition (U.S. EPA, 2013, Table 1-2).

19   From this set of effects that the ISA has concluded to be causally or likely causally

20   related to O_3 in ambient air, we focus primarily on three categories of effects (i.e., visible foliar

21   injury; impacts on tree growth, productivity and carbon storage; and crop yield loss). As

22   recognized in the ISA, controlled exposure studies, which are the best method for isolating or

23   characterizing the role of O_3 in inducing the observed plant effects, "have clearly shown that

24   exposure to O_3 is causally linked to visible foliar injury, decreased photosynthesis, changes in

25   reproduction, and decreased growth" in vegetative species (U.S. EPA 2013, p. 1-15). These plant

26   level effects are also linked to a cascade of other ecosystem level effects. For example, studies at

27   larger spatial scales support the controlled exposure study results and indicate that "ambient O_3

28   exposures can effect ecosystem productivity, crop yield, water cycling, and ecosystem

29   community composition" (U.S. EPA 2013, p. 1-15). Thus, O_3 effects on vegetation may have

30   implications at the individual, species, population and whole ecosystem level, including

31   associated ecosystem services. [1]

---

[1] Ecosystem services are the benefits that people obtain from ecosystems and have been stated to include "provisioning services such as food and water; regulating services such as regulation of floods, drought, land degradation, and disease; supporting services such as soil formation and nutrient cycling; and cultural services such as recreational, spiritual, religious, and other nonmaterial benefits" according to the Millennium Ecosystem Assessment (UNEP, 2003; U.S. EPA, 2013, p. lxxii).

1   Our consideration of O_3 welfare effects and the significance or weight to place on a given
2   study or exposure/risk assessment result, is informed by the understanding, based on the entire
3   body of vegetation effects science, that a variety of other factors can either mitigate or exacerbate
4   the O_3-plant interactions and are recognized sources of uncertainty and variability. These
5   include: 1) multiple genetically-influenced determinants of O_3 sensitivity; 2) changing sensitivity
6   across growth stages; 3) co-occurring stressors and/or modifying environmental factors; 4) a
7   paucity of information on most of the 43,000 U.S. plant species (U.S. EPA 2013, section 9.4.8;
8   U.S. EPA 2006; U.S. EPA, 2007, section 7.4.2).

9   - **Does the current evidence continue to support a cumulative, seasonal exposure**
10   **index as a biological-relevant and appropriate metric for assessment of the**
11   **evidence and exposure/risk information?**

12   In this review, the ISA assessment of the full body of currently available evidence stated
13   the following regarding biological indices (U.S. EPA, 2013, p. 2-44):

14   *The main conclusions from the 1996 and 2006 O_3 AQCDs regarding indices*
15   *based on ambient exposure remain valid. These key conclusions can be restated*
16   *as follows:*

17   - *ozone effects in plants are cumulative;*

18   - *higher O_3 concentrations appear to be more important than lower*
19   *concentrations in eliciting a response;*

20   - *plant sensitivity to O_3 varies with time of day and plant development*
21   *stage;*

22   - *quantifying exposure with indices that cumulate hourly O_3 concentrations*
23   *and preferentially weight the higher concentrations improves the*
24   *explanatory power of exposure/response models for growth and yield,*
25   *over using indices based on mean and peak exposure values.*

26   Thus, the current evidence, as in other recent reviews, continues to support a cumulative,
27   seasonal exposure index as a biologically-relevant and appropriate metric for assessment of the
28   evidence and exposure/risk information. To this point, the available body of evidence provides a
29   wealth of information on the aspects of O_3 exposure that are most important in influencing plant
30   response. While a variety of "factors with known or suspected bearing on the exposure-response
31   relationship, including concentration, time of day, respite time, frequency of peak occurrence,
32   plant phenology, predisposition, etc.," have been identified (U.S. EPA, 2013, section 9.5.2), the
33   importance of the duration of the exposure and the relatively greater importance of higher
34   concentrations over lower in determining plant response to O_3 have been well documented (U.S.
35   EPA, 2013, section 9.5.3). Much of this work was completed by the mid-1990s, and was

1 summarized in the 1996 Criteria Document (U.S. EPA, 1996), while the additional newer work

2 is assessed in the subsequent 2006 Criteria Document and 2013 ISA.

3   In conjunction with this research on plant response to O_3 exposures, others have

4 developed "mathematical approaches for summarizing ambient air quality information in

5 biologically meaningful forms for O_3 vegetation effects assessment purposes …" (U.S. EPA,

6 2013, section 9.5.3). A large set of exposure indices have been developed that use a variety of

7 functions to weight factors that have been shown to influence vegetation exposure-response

8 relationships (U.S. EPA, 2013, section 9.5.2). As discussed in the ISA, several indices have

9 been developed to attempt to incorporate some of the biological, environmental, and exposure

10 factors that influence the magnitude of the biological response and contribute to observed

11 variability (U.S. EPA, 2013, section 9.5.2). As with any summary statistic, these exposure

12 indices retain information on some, but not all, characteristics of the original observations.

13   Flux models have been identified in recent years to take into account more of the factors

14 that influence the response and contribute to observed variability (U.S. EPA, 2013, section

15 9.5.4). We note that "some researchers have claimed that using flux models can be used to better

16 predict vegetation responses to O_3 than exposure-based approaches…" because flux models

17 estimate the ambient O_3 concentration that actually enters the leaf (i.e., flux or deposition) (U.S.

18 EPA, 2013, p. 9-114). However, "[f]lux calculations are data intensive and must be carefully

19 implemented" (U.S. EPA, 2013, p. 9-114). Further, "[t]his uptake-based approach to quantify

20 the vegetation impact of O_3 requires inclusion of those factors that control the diurnal and

21 seasonal O_3 flux to vegetation (e.g., climate patterns, species and/or vegetation-type factors and

22 site-specific factors)" (U.S. EPA, 2013, p. 9-114). Each species has different amounts of internal

23 detoxification potential that may protect species to differing degrees. This balance between O_3

24 flux and detoxification processes has been termed the "effective flux".   Accordingly, the

25 "models have to distinguish between stomatal and non-stomatal components of O_3 deposition to

26 adequately estimate actual concentration reaching the target tissue of a plant to elicit a response"

27 and "[d]etermining this O_3 uptake via canopy and stomatal conductance relies on models to

28 predict flux and ultimately the 'effective' flux" (U.S. EPA, 2013, pp. 9-114).   The lack of

29 detailed species- and site-specific data required for flux modeling in the U.S. and the lack of

30 understanding of detoxification processes have made this technique less viable for use in

31 vulnerability and risk assessments at the national scale in the U.S. (U.S. EPA, 2013, section

32 9.5.4).

33   Thus, in the last two reviews of the O_3 secondary standard, completed in 1997 and 2008,

34 the EPA concluded that the risk to vegetation comes primarily from cumulative exposures to O_3

35 over a season or seasons and, in both reviews, the EPA proposed, as one alternative, a secondary

36 standard set in terms of such a form (61 FR 65716, 72 FR 37818). Although in both reviews the

1   secondary standard was revised to be identical to a revised primary standard (with an 8-hour
2   averaging time), the Administrator, in both cases, also concluded, consistent with CASAC
3   advice, that a cumulative, seasonal index was the most biologically relevant way to relate
4   exposure to plant growth response (62 FR 38856, 73 FR 16436). Most recently, in the 2010
5   proposed reconsideration of the 2008 decision, the EPA again proposed to conclude that O_3
6   exposure indices that cumulate differentially weighted hourly concentrations are the best
7   candidates for relating exposure to plant growth responses and proposed to set the secondary
8   standard only in terms of one such form, the W126 (75 FR 2938).
9   Based on the long-established conclusions and long-standing supporting evidence
10 described above, we continue to focus on the aspects of ambient O_3 exposures that have
11 biological relevance and the biologically-relevant exposure indices or metrics that have been
12 designed in light of this consideration, i.e., cumulative seasonal indices. Since the review
13 completed in 1997, which was the first to focus on cumulative indices, attention has been given
14 primarily to two different cumulative index forms: SUM06 and W126. The SUM06 index is a
15 threshold-based approach described as the sum of all hourly O_3 concentrations greater or equal to
16 0.06 ppm observed during a specified daily and seasonal time window (U.S. EPA, 2013, section
17 9.5.2). The W126 index is a non-threshold approach described as the sigmoidally weighted sum
18 of all hourly O_3 concentrations observed during a specified daily and seasonal time window,
19 where each hourly O_3 concentration is given a weight that increases from 0 to 1 with increasing
20 concentration (Lefohn et al, 1988; Lefohn and Runeckles, 1987; U.S. EPA, 2013, section 9.5.2).
21 The EPA used the W126 index to consider welfare effects in the last review, as well as the 2010
22 proposed reconsideration of the 2008 decision; this approach received support from CASAC, as
23 discussed in section 5.6 below. Consistent with the ISA conclusions regarding the
24 appropriateness of considering cumulative exposure indices for O_3 effects of concern based on
25 the evidence available in this review, we again conclude that the current evidence continues to
26 support a cumulative, seasonal exposure index as a biologically-relevant and appropriate metric
27 for assessment of the evidence and exposure/risk information, and in particular, the W126
28 cumulative, seasonal metric (U.S. EPA, 2013, p. 2-44 and section 9.5.2).

29 • **Within what paradigm may it be appropriate to consider the potential adversity**
30   **of public welfare effects of O_3?**

31   The Clean Air Act requires that a secondary standard be protective against those known
32 or anticipated O_3 effects that are "adverse" to the public welfare, not all identifiable O_3-induced
33 effects. Unlike the use of the terms adverse, injury or damage in the scientific literature, in the
34 NAAQS policy context, these terms have been interpreted in a particular way. Specifically, O_3-
35 induced "injury" to vegetation has been defined as encompassing all plant reactions, including

1   reversible changes or changes in plant metabolism (e.g., altered photosynthetic rate), altered

2   plant quality or reduced growth that does not impair the intended use or value of the plant. In

3   contrast, "damage" has been defined to include only those injury effects that reach sufficient

4   magnitude as to also reduce or impair the intended use or value of the plant to the public and thus

5   potentially become adverse to the public welfare. Examples of vegetation effects that have been

6   classified as damage include reductions in aesthetic values (e.g., foliar injury in ornamental

7   species) as well as losses in terms of weight, number, or size of harvestable plant parts. Biomass

8   loss in tree species can also be considered damage or adverse to the public welfare if it includes

9   slower growth in species harvested for timber or other fiber uses. In the context of evaluating

10   effects on single plants or species grown in monocultures such as managed forests, this construct

11   continues to remain useful (73 FR 16492/96).

12   However, given the increasing scientific literature linking O_3 effects on plants or species

13   to effects at the community or ecosystem level, a more expansive construct or paradigm of what

14   constitutes O_3 "damage" beyond that of the individual or species level is appropriate. A number

15   of broader paradigms have been discussed in the literature (72 FR 37890; Hogsett et al., 1997;

16   Young and Sanzone, 2002). In the 2008 review, the Administrator expressed support for relying

17   on a definition of "adverse" discussed in section IV.A.3 of the proposal (62 FR 37889-37890)

18   that embeds "the concept of 'intended use' of the ecological receptors and resources that are

19   affected, and applies that concept beyond the species level to the ecosystem level" (73 FR

20   16496). For example, in the 2008 rulemaking notice, the Administrator took note of "a number

21   of actions taken by Congress to establish public lands that are set aside for specific uses that are

22   intended to provide benefits to the public welfare, including lands that are to be protected so as to

23   conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave

24   them unimpaired for the enjoyment of future generations" (73 FR 16496).

25   Since the 2008 O_3 review, our approach to assessing adversity to the public welfare in

26   NAAQS reviews has continued to evolve. In particular, we consider the concept of ecosystem

27   services in a broader paradigm. An extensive look at the range of services than can be provided

28   by ecosystems is described in the Millennium Ecosystem Assessment (MEA, 2005). Ecosystem

29   services can be generally defined as the benefits that individuals and organizations obtain from

30   ecosystems. The EPA has previously defined ecological goods and services for the purposes of

31   benefits assessment as the ''outputs of ecological functions or processes that directly or

32   indirectly contribute to social welfare or have the potential to do so in the future. Some outputs

33   may be bought and sold, but most are not marketed'' and has relied on this definition in

34   regulatory impact analyses for previous NAAQS reviews (U.S. EPA, 2006b). In the review of

35   the secondary NAAQS for oxides of nitrogen and sulfur, EPA recognized that changes in

36   ecosystem services may be used to aid in characterizing a known or anticipated adverse effect to

1   public welfare and that an evaluation of adversity to the public welfare might consider the
2   likelihood, type, magnitude, and spatial scale of the effect, as well as the potential for recovery
3   and any uncertainties relating to these conditions (77 FR 20232). In the context of this review,
4   ecosystem services are being evaluated and assessed in the REA as one way to characterize the
5   possible public welfare benefits received from ecosystem resources and how those services
6   might be expected to change under air quality scenarios representing the current and potential
7   alternative secondary standards (U.S. EPA, 2014, chapter 5). Thus, in considering the evidence
8   and exposure risk information associated with the welfare endpoints identified below, in the
9   context of consideration of adequacy of the current standard in this chapter and potential
10   alternative standards in chapter 6, we consider also how they fit within this paradigm.

5.2   FOREST TREE GROWTH, PRODUCTIVITY AND CARBON STORAGE

12   This section considers the current evidence and exposure/risk information to inform
13   consideration of the adequacy of the protection provided by the current standard from known and
14   anticipated adverse welfare effects of O_3 related to growth, productivity, and carbon storage of
15   forest trees, and other associated effects. Trees are important from a public welfare perspective
16   because they provide many valued services to humans. In addition to the aesthetic value
17   discussed below in 5.4, these include: food, fiber, timber, other forest products, habitat,
18   recreational opportunities, climate regulation, erosion control, air pollution removal, hydrologic
19   and fire regime stabilization (U.S. EPA, 2014, section 6.1, Figure 6-1, section 6.4, Table 6-12).
20   This section includes a discussion of the policy-relevant evidence and weight-of-evidence
21   conclusions discussed in the ISA (section 5.2.1) and the exposure/risk results, including
22   associated ecosystem services (section 5.2.2) described in the second draft WREA. Important
23   uncertainties and limitations in the available information are discussed throughout the sections.
24   These discussions highlight the information we consider relevant to answering the overarching
25   question and associated policy-relevant questions included in this section.

5.2.1   Evidence-based Considerations

27   - **To what extent has scientific information become available that alters or**
28   **substantiates our prior conclusions of O_3-related effects on forest tree growth,**
29   **productivity and carbon storage and of factors that influence associations between**
30   **O_3 concentrations and these effects?**

31   Research published since the 2006 AQCD substantiates prior conclusions regarding O_3-
32   related effects on forest tree growth, productivity and carbon storage. Information supporting
33   these previous conclusions comes from a variety of different types of studies and which cover an
34   array of different species, endpoints and exposure methods.   One subset of studies focused on

1   underlying mechanisms as they relate to growth, productivity and carbon storage including:
2   reduced carbon dioxide uptake due to stomatal closure (U.S. EPA 2013, section 9.3.2.1); the
3   upregulation of genes associated with plant defense, signaling, hormone synthesis and secondary
4   metabolism (U.S. EPA 2013, section 9.3.3.2); the down regulation of genes related to
5   photosynthesis and general metabolism (U.S. EPA 2013, section 9.3.3.2); loss of carbon
6   assimilation capacity due to declines in the quantity and activity of key proteins and enzymes
7   (U.S. EPA, 2013, section 9.3.5.1); and negative impacts on the efficiency of the photosynthetic
8   light reactions (U.S. EPA, 2013, section 9.3.5.1). These new studies "have increased knowledge
9   of the molecular, biochemical and cellular mechanisms occurring in plants in response to O_3",
10   adding "to the understanding of the basic biology of how plants are affected by oxidative
11   stress…" (U.S. EPA, 2013, p. 9-11).
12   The recent studies cover a variety of exposure methods, species, and settings. In
13   particular, a recent meta-analysis indicates a relationship between O_3 concentrations in the
14   northern hemisphere and effects with the potential to affect growth (i.e., stomatal conductance
15   and photosynthesis) (U.S. EPA, 2013, section 9.4.3.1; Wittig et al., 2007). A second meta-
16   analysis, which quantitatively compiled peer-reviewed studies from the past 40 years, found that
17   ambient concentrations reported in those studies significantly decreased annual total biomass
18   growth (7%) across the studies (U.S. EPA, 2013, section 9.4.3.1). The ISA states that these two
19   meta-analyses demonstrate the coherence of O_3 effects on plant photosynthesis and growth
20   across numerous studies and species using a variety of experimental techniques", and including a
21   recent study, that "recent meta-analyses have generally indicated that O_3 reduced carbon
22   allocation to roots (Wittig et al., 2009; Grantz et al., 2006)" (U.S. EPA, 2013, pp. 9-45 to 9-46).
23   Recent field-based studies also have added to the evidence base. For example, a study
24   conducted in forest stands in the southern Appalachian Mountains found that "the cumulative
25   effects of ambient levels of O_3 decreased seasonal stem growth by 30-50% for most tree species
26   in a high O_3 year in comparison to a low O_3 year (McLaughlin et al., 2007a). The authors also
27   reported that high ambient O_3 concentrations can increase whole-tree water use and in turn
28   reduce late-season streamflow (McLaughlin et al., 2007b)" (U.S. EPA, 2013, p. 9-43).
29   Additionally, a recent study has provided concentration-response information for tree seedlings
30   of an additional species beyond the 11 previously studied (Gregg, et al., 2003). This study on
31   cottonwood expands the dataset of studied species such that the 12 species for which C-R
32   functions are available in this review include deciduous, coniferous, eastern, western, sensitive
33   and tolerant species (U.S. EPA 2013, section 9.6.2; U.S. EPA, 2014, section 6.2, Figure 6-2,
34   Table 6-1).
35   The "previous O_3 AQCDs concluded that there is strong evidence that exposures to O_3
36   decreases photosynthesis and growth in numerous plant species" and that "[s]tudies published

1   since the 2008 review support those conclusions" (U.S. EPA, 2013, p. 9-42). The previously

2   available strong evidence included the development of robust concentration-response (C-R)

3   functions for tree seedling biomass loss in 11 species under the National Health and

4   Environmental Effects Research Laboratory-Western Ecology Division (NHEERL-WED)

5   program. This series of experiments used open-top-chambers (OTC) to study seedling growth

6   response under a variety of O_3 exposures (ranging from near background to well above current

7   ambient concentrations) and growing conditions (U.S. EPA 2013, section 9.6.2, Lee and Hogsett,

8   1996).

9   We additionally recognize that, because trees are long lived, in addition to the effects of

10   O_3 exposures over the annual growing season, trees and other perennials can also cumulate

11   effects across multiple years. For example, growth affected by a reduction in carbohydrate

12   storage in one year may result in the limitation of growth in the following year, so that effects

13   "carry over" from one year to another (U.S. EPA, 2013, section 9.4.8; Andersen, et al., 1997). In

14   past reviews, such carry-over effects have been documented in the growth of some tree seedlings

15   and in roots. A number of recent studies based on the FACE exposure method in a planted forest

16   at the Aspen FACE site in Wisconsin have augmented and supported the earlier information.

17   These studies observed tree growth responses over seven years beyond the seedling growth stage

18   and growing in field settings more similar to natural forest stands than OTC studies. In addition

19   to affecting tree heights, diameters, and main stem volumes in the aspen community, elevated O_3

20   was reported to change intra- and inter-species competition (Kubiske et al., 2006; Kubiske et al.,

21   2007). For example, O_3 treatments increased the rate of conversion from a mixed aspen-birch

22   community to a birch dominated community, potentially changing intra- and inter-species

23   competition.

24   The EPA comparison of biomass results from the first seven years of the recent study by

25   Kubiske et al (2007) to that predicted using the C-R function established from the earlier OTC

26   experiments indicated close agreement (U.S. EPA 2013, Section 9.6.3). Accordingly, the ISA

27   concludes that "[o]verall, the studies at the Aspen FACE experiment were consistent with many

28   of the open-top chamber (OTC) studies that were the foundation of previous O_3 NAAQS

29   reviews" and that "[t]hese {recent} results strengthen the understanding of O_3 effects on forests

30   and demonstrate the relevance of the knowledge gained from trees grown in OTC studies" (U.S.

31   EPA 2013, p. 2-38). In addition to growth effects, these scientists also found that elevated O_3

32   decreased birch seed weight, germination, and bud starch levels as well as aspen bud size. The

33   effects on birch seeds could lead to a negative impact on species regeneration, while the bud

34   effects may have been related to the observed delay in spring leaf development and have the

35   potential to alter carbon metabolism of overwintering buds. These latter effects likely have

36   implications for the subsequent growing season (i.e. carry-over effects) in the following year,

1   including effects on forest biomass, buds and seeds that carry into subsequent years (U.S. EPA,
2   2013, section 9.4).

3   The recent studies, in combination with the entire body of evidence, form the basis for
4   the ISA determinations that: 1) there is a causal relationship between O_3 exposures and reduced
5   vegetative growth; 2) there is a causal relationship between O_3 exposures and biomass
6   accumulation, including altered carbon allocation to below ground tissues, rates of leaf and root
7   production, and turnover and decomposition that can alter below-ground biogeochemical cycles;
8   and 3) there is likely to be a causal relationship between O_3 exposure and a reduction in carbon
9   sequestration in terrestrial ecosystems (U.S. EPA, 2013, Table 2-2). Therefore, the current
10   evidence base substantiates prior conclusions of O_3-related effects on tree growth, productivity
11   and carbon storage. The results from some of these studies are discussed more fully under
12   different questions below.

13   • **To what extent have important uncertainties in the evidence identified in the last**
14   **review been reduced and/or new uncertainties emerged?**

15   As stated above, the ISA concludes that the new evidence confirms, strengthens or
16   expands our understanding of O_3 effects on plants. Much of this new evidence is focused on the
17   molecular and genetic level, providing very important new mechanistic information that in some
18   cases enhances our understanding of the complexity of the O_3–plant response. This information
19   has, in general, reduced overall uncertainties at the subcellular and cellular scales. However,
20   because these studies were primarily conducted using artificial exposure conditions and model
21   plants, uncertainties remain regarding the extent to which these plant responses reflect those in
22   other plant species and exposure conditions (U.S. EPA, 2013, section 9.3.6). With regard to O_3
23   impacts at the whole plant, species, and ecosystem scales, recent information has informed our
24   understanding of associated uncertainties in a variety of ways.
25   Importantly, one key uncertainty has been significantly reduced. This relates to the C-R
26   functions we have used in previous reviews to estimate tree seedling biomass loss for different
27   O_3 exposure conditions (U.S. EPA, section 9.6). As these functions were derived from OTC
28   experiments, an associated uncertainty has been with regard to the extent to which they reflected
29   concentration-response relationships for field conditions. In the current review, EPA staff have
30   conducted an analysis comparing OTC data with field-based data for one crop and one tree
31   species (U.S. EPA, 2013, section 9.6.3.2). One comparison was done using soybean OTC data
32   from the National Crop Loss Assessment Network (NCLAN)[2] and field-based data (Soy FACE),

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[2] The NCLAN program was conducted from 1980 to 1987 at five different locations across the US. At each site, open top chambers were used to expose plants to O_3 treatments that represented the range of

1   as discussed in section 5.3 below. The second was done using aspen seedling OTC data from the

2   NHEERL-WED and field-based data (Aspen FACE). The result of the aspen analysis, similar to

3   that for soybean, showed very close agreement between the predictions based on NHEERL-

4   WED data and Aspen FACE observations, even when comparing the results of experiments that

5   used different exposure methodologies, different genotypes, locations, and durations. Based on

6   this analysis, the ISA additionally stated that "the [C-R] function based on one year of growth

7   was applicable to subsequent years" (of the six-year dataset) (U.S. EPA, 2013, section 9.6.3.2).

8   This information reduces the uncertainties associated with potential impacts of other

9   experimental factors on the O_3-plant response. Other studies, such as the meta-analyses

10   discussed in the ISA and below, also demonstrate the coherence of O_3 growth effects across

11   numerous studies and species that used a variety of experimental techniques. While recognizing

12   that uncertainties may remain for some individual species for which the database is relatively

13   less robust (such as the more recent information on cottonwood), taken together, this information

14   substantially reduces uncertainties associated with use of the tree seedling OTC-derived C-R

15   functions to predict the response of trees beyond the seedling stage in field settings. Thus, in the

16   current review, the ISA and WREA have continued to use these functions to estimate tree growth

17   response for different cumulative O_3 exposures (U.S. EPA, 2013, section 9.6.2; U.S. EPA, 2014,

18   section 6.2).

19   Other uncertainties associated with studying or modeling O_3 impacts on trees, including

20   those identified in the last review, still remain, due in part to a lack of additional research but

21   also due to the growth characteristics of trees which present a unique set of experimental

22   challenges. For example, while trees are long-lived, with life spans which range from decades to

23   centuries, most studies are designed to take place within an annual or 2-3 year timeframe, which

24   represents only a small fraction of the lifetime of a tree. Further, trees grow very large, making it

25   difficult to use controlled exposure environments beyond the seedling or sapling growth stage.

26   Additionally, sensitivity to O_3 varies over the life of the tree so that different growth stages of the

27   tree may be more or less sensitive and this variation in growth-stage sensitivity is species-

28   specific. Thus, while some limited information exists regarding tree sensitivity beyond the

29   seedling growth stage (e.g. aspen, cottonwood) and in some species for both seedling and mature

30   trees within a species (e.g., red oak), it remains uncertain to what degree effects observed on

31   trees during one growth stage (e.g., seedling) can be extrapolated to trees at other growth stages.

32   An analysis in the WREA comparing seedling to adult tree biomass loss, discussed in 5.2.2

concentrations that occur in different areas of the world. The NCLAN focused on the most important U.S
agricultural crops (Heagle et al., 1989; http://www.ars.usda.gov/Main/docs.htm?docid=12462).

1 below, informs our consideration of this remaining uncertainty (U.S. EPA, 2014, section

2 6.2.1.1).

3   Lastly, we recognize that exposures in one year have the potential to cause effects not

4 observed until a subsequent year (termed "carry over" effects). While recent studies provide

5 evidence of some carry-over effects and the potentially serious implications they could have for

6 associated ecosystems and services, the true extent of this effect is unclear because most studies

7 on the effects of O_3 on growth do not measure or take into account the possibility of carry-over

8 effects in subsequent years. Additionally, uncertainties remain regarding the extent of

9 compounding of growth effects across multiple years for different air quality conditions. These

10 uncertainties affect our characterization of such impacts, particularly the quantitative aspects, for

11 differing exposure scenarios. Therefore our characterization of subsequent growing season

12 effects is uncertain, affecting our ability to fully describe impacts of observed annual biomass

13 losses, particularly in quantitative terms. In section 5.2.2 below, the potential variation of

14 growth effect compounding across multiple years is discussed further drawing on the WREA

15 evaluation of this (U.S. EPA, 2014, section 6.2.1.4). Further discussion of these tree-related

16 uncertainties that are relevant in the context of informing our understanding of the quantitative

17 and qualitative exposure and risk results are discussed in the appropriate sections below.

18 • **What are the ecosystem services potentially affected by O_3 effects on tree growth,**
19 **productivity and carbon storage and to what extent are they important from a**
20 **public welfare perspective?**

21   A variety of ecosystem services are potentially affected by O_3 impacts on tree growth,

22 productivity and carbon storage. The ISA identifies as causal the relationship of O_3 and reduced

23 productivity in terrestrial ecosystems and alteration of below ground biogeochemical cycles. It

24 further identifies as likely to be a causal relationship O_3impacts on reduced carbon sequestration

25 in terrestrial ecosystems; alteration of terrestrial ecosystem water cycling; alteration of terrestrial

26 community composition (U.S. EPA, 2013, Table 9-19).   These effects are important to the

27 public welfare, and in particular include those associated with national parks, national refuges

28 and other protected areas ranging to the harvesting of timber for commercial uses. The

29 ecosystem services most directly affected by biomass loss include: (1) habitat provision for

30 wildlife, (2) carbon storage, (3) provision of food and fiber, and (4) pollution removal (see also

31 U.S. EPA, 2014, Figure 6-1).[3] Less direct impacts can occur on process-related effects such as

32 nutrient and hydrologic cycles.

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[3] The impacts of O_3 in ambient air on some of these services are characterized in the WREA, as described
in section 5.2.2 below.

1   With regard to the extent of O_3 impact on carbon transport or allocation to roots,

2   information from a few recent individual studies have shown mixed results, including negative,

3   non-significant, and positive effects on root biomass and root:shoot ratio. However, as assessed

4   in the ISA, "[g]enerally, there is clear evidence that O_3 reduces carbon transport or allocation to

5   roots", although (U.S. EPA, 2013, p. 9-44).

6   • **To what extent does the available evidence indicate the occurrence of O_3-related**
7   **effects on forest growth, productivity and carbon storage attributable to**
8   **cumulative exposures at lower ambient O_3 concentrations than previously**
9   **established or to exposures that might be expected to occur under the current**
10   **standard?**

11   The evidence available in this review indicates that O_3-induced effects on tree growth,

12 productivity and carbon storage occur as a result of cumulative exposure concentrations similar

13 to those identified in the previous review and that these effects can occur at exposures associated

14 with air quality conditions that might be expected to meet the current standard. We first consider

15 the evidence on O_3 effects on growth, particularly for tree seedlings, and that supports the C-R

16 functions describing the relationship between cumulative O_3 exposure and reduction in growth

17 (relative biomass loss). As described above, this evidence base currently includes functions for

18 12 species.[4] Figure 5-1, below (based on species-specific composite functions described in the

19 ISA and WREA), illustrates the appreciable variation in sensitivity across individual species

20 (U.S. EPA 2013, section 9.6.2; U.S. EPA, 2014, section 6.2, Table 6-1 and Figure 6-2). For

21 example, in seven or the 12 species, the W126 index value for which 2% seedling biomass loss is

22 estimated is below 8 ppm-hours and in the other five species, the W126 value for which 2%

23 biomass loss is estimated is above 18 ppm-hours.   Within the group of seven more sensitive

24 species, the most sensitive are cottonwood and black cherry (U.S. EPA 2014, section 6.2).

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[4] Among the 12 species, in addition to the functions for 11 species studied in the FACE research, is the field study by Gregg et al (2003) for cottonwood. As noted in discussion of uncertainties above, there is only the single study available for this species which focused on a gradient of O3 exposure extending from the New York metropolitan area which may contribute uncertainty to the quantitative characterization of C-R.
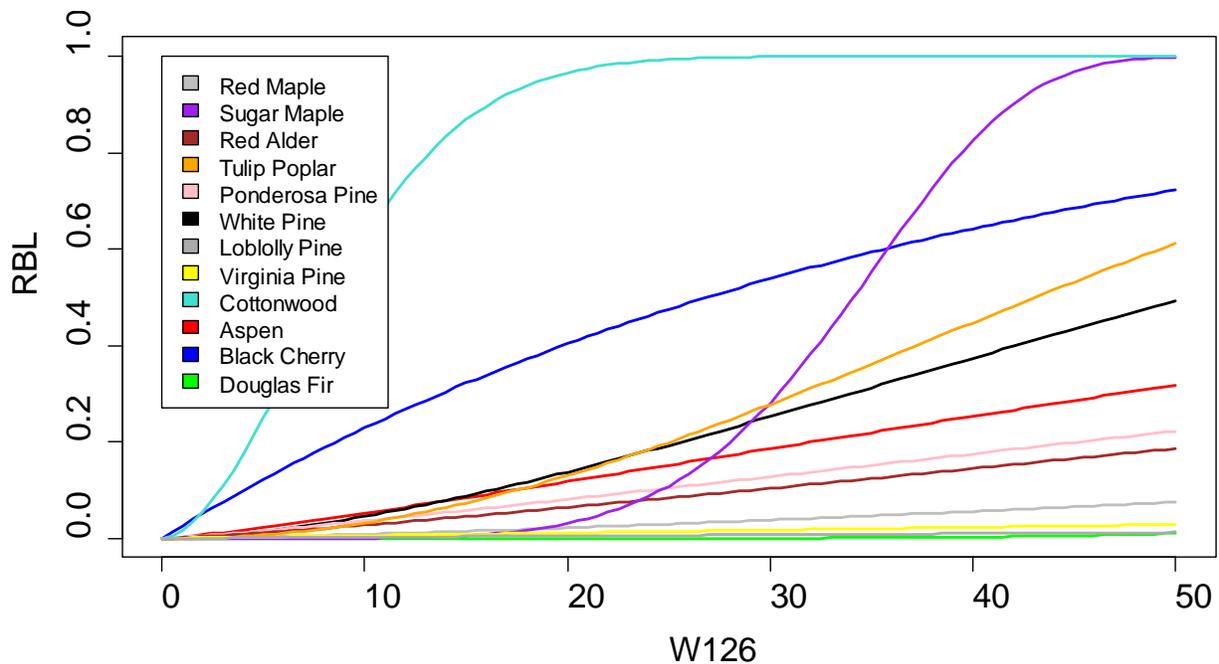
Figure 5-1. Relative biomass loss in seedlings for 12 studied species in response to seasonal ozone concentrations in terms of seasonal W126 index values (U.S. EPA 2014, Figure 6-2).

In considering the evidence for the two most sensitive species, we recognize the more limited quantitative information for cottonwood. The C-R function for this species is based on the study by Gregg et al (2003) which described O_3 effects on growth on eastern cottonwood (*Populus deltoides*) saplings along an O_3 gradient between urban New York City and downwind rural areas for the years 1992, 1993 and 1994. While the authors additionally studied cottonwood response in a companion controlled OTC experiment, the C-R function described here is based on the field data and reflects the single study (U.S. EPA, 2013, sections 9.2.5 and 9.6.3). Given these factors, as noted in consideration of uncertainties above, we recognize there may be additional uncertainty in this function.

Using the 11 concentration-response (C-R) functions for tree seedlings from the FACE research, and with the addition of the cottonwood C-R function, Figure 5-2 below presents three approaches for characterizing the median response function (U.S. EPA, 2014, section 6.2.1.2 and Figure 6-4). The first approach used a composite function developed from the C-R functions for all 52 tree seedling studies (across the 12 species) combined (red line). The second approach used only the median C-R function, when available, for each of the 12 tree species included (green line)[5]. The third approach used a stochastic sampling method to randomly select a C-R

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[5] For some species, only one study was conducted so that C-R function was used.

1   function for each species from the studies conducted for that species. For some species only one
2   study was available (e.g., Red Maple), and for other species there were as many as 11 studies
3   available (Ponderosa Pine). The process was repeated 1,000 times (grey lines), and the median
4   value was plotted for biomass loss values of 1% to 7%, and 10% (the bar associated with each
5   median point denotes the 25th and 75th percentile values. This third approach provides an
6   illustration of the effect of within-species variability on estimates of the median species. The
7   median W126 values are similar, when using all of the studies or just the composite C-R function
8   for each species; however, the median value is higher when within-species variability is included
9   (U.S. EPA, 2014, section 6.2.1.2). Among these three approaches, the median seasonal W126
10   index value for which a two percent biomass loss is estimated in seedlings for the studied species
11   ranges between approximately 7 and 14 ppm-hrs.
12
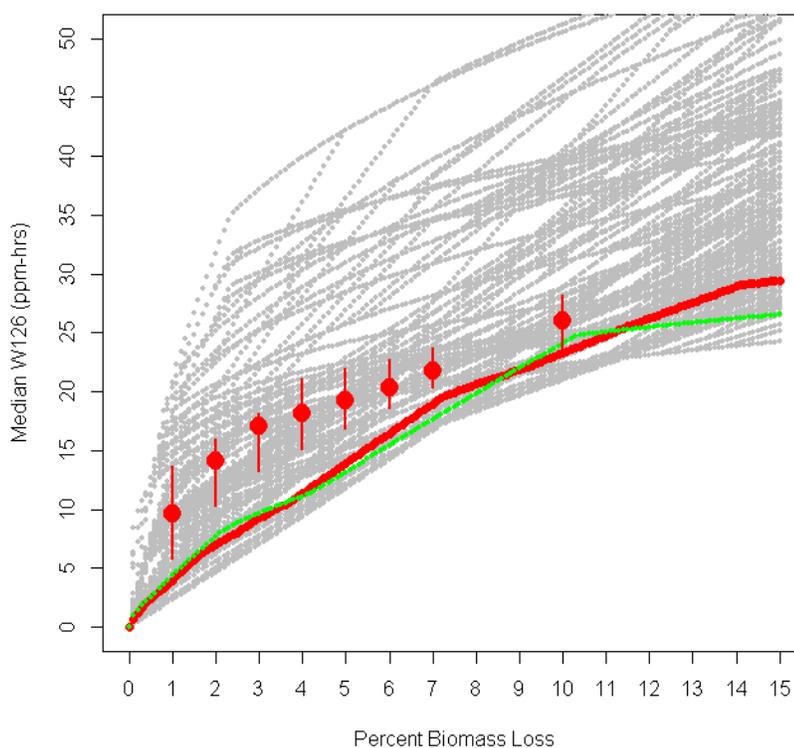
13   **Figure 5-2.   Relationship of tree seedling percent biomass loss with seasonal W126 index.**
14   **(From U.S. EPA 2014, Figure 6-4)**

15   We further consider several recent studies that have provided important evidence of
16   growth effects occurring in the field at ambient exposure concentrations, including some
17   exposure concentrations which are at or below the level of the current standard. For example,
18   the study by McLaughlin et al., (2007a, b) investigated the effects of ambient O$_3$ on tree growth
19   (measured by change in stem circumference) in the field at 3 forest sites (Cade's Cove, Look

1   Rock and Oak Ridge) in the southern Appalachian Mountains for three years (2001, 2002, 2003)

2   (U.S. EPA, 2013, section 9.4.5). Compared to the growth observed in 2001 (base year), the

3   authors reported that ambient O_3 exposures, decreased seasonal stem growth by 30-50% for most

4   tree species in a high O_3 year (2002), relative to the 2001 baseline. As shown in Table 5-6 below

5   (modified from Table 6-4 in U.S. EPA, 2014), the seasonal W126 index values ranged from

6   approximately 9 to 20 ppm-hours in the low O_3 year (2003) and from approximately 18 to 40

7   ppm-hours in the high O_3 year (2002), across the three sites.   When these exposure

8   concentrations for the three years are expressed in terms of the 4th highest daily maximum 8-hour

9   average concentration, the values range from 71 to 90 ppb during the low year (2003) to 82 –

10   102 ppb during the high year (2002). The 3-year average of these 4th highest daily maximums[6]

11   for the period 2001-2003 are 76 ppb, 93 ppb, and 87 ppb for Cade's Cove, Look Rock, and Oak

12   Ridge, respectively. Thus, while two of the years at Cade's Cove had annual values at or below

13   75 ppb, the concentrations at all sites for the 2001- 2003 period appear to exceed the current

14   standard. Only one species, tulip poplar, has data for all three years and was present at all three

15   sites (U.S. EPA, 2014, Table 6-4). This subset of data is what is shown in table 5-1 below.

16   **Table 5-1. Ozone concentrations associated with effects on tulip poplar in southern**
17   **Appalachian Mountains (2001-2003).**

Ambient O_3 concentrations for McLaughlin et al., 2007[A]									
	Cade's Cove			Look Rock			Oak Ridge		
	Max4 high (ppm)	W126 (ppm-hrs)	Percent change in circumference[B]	Max4 high (ppm)	W126 (ppm-hrs)	Percent change in circumference[B]	Max4 high (ppm)	W126 (ppm-hrs)	Percent change in circumference[B]
2001	75	15	baseline	86	23	baseline	85	20	baseline
2002	82	18	-62%	102	40	-26%	99	32	-49.6%
2003	71	9	N/A	90	20	-38%	78	11	7.5%
2001-2003	76	N/A		93	28		87	21	

[A]Air quality values obtained from http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm. W126 rounded to nearest whole ppm-hr. 2002 and 2003 study results measured as % change in tulip poplar stem circumference compared to 2001 baseline.
[B] Study results for percent change in circumference of Tulip Poplar from WREA Table 6-4.

18

19   Based on the table above, appreciable growth reductions occurred in tulip poplar at all

20   three sites during a high O_3 year (2002), though it spanned a rather large range of -26% to -62%.

---

[6] This is the form of the current standard. Thus, the current standard is met when the 3-year average of the 4th highest daily maximum 8-hour concentrations is at or below 75 ppb.

1 The results of this study are hard to interpret given the different exposure base lines, magnitude

2 of changes in subsequent year exposures, and other undocumented site-specific factors. The

3 study findings do show, however, that higher O_3 concentrations (above the current standard)

4 significantly reduced growth compared to that in a lower O_3 year.

5   With regard to evidence of effects associated with exposures that may be allowed by the

6 current standard, we additionally consider the study by Gregg et al (2003) referenced above that

7 documented O_3-induced growth loss on eastern cottonwood (*Populus deltoides*) saplings along

8 an O_3 gradient between urban New York City and downwind rural areas for the years 1992, 1993

9 and 1994 (U.S. EPA, 2013, section 9.2.5). Fourth highest daily max 8-hour average

10 concentrations during the experimental period (July 7 – Sept 20) ranged from 0.056 ppm to

11 0.098 ppm along the gradient (data in 2008 O_3 docket) while W126 index values ranged from 1.9

12 to 12 ppm-hrs. At some sites, the highest daily maximum 8-hour average ambient concentrations

13 (0.056 ppm, 0.072 ppm, and 0.073 ppm) were below the level of the current standard, indicating

14 that the standard would likely have been met. Figure 5-3 below shows the growth suppression

15 response of the cottonwood saplings to the range of O_3 exposures occurring along the gradient.
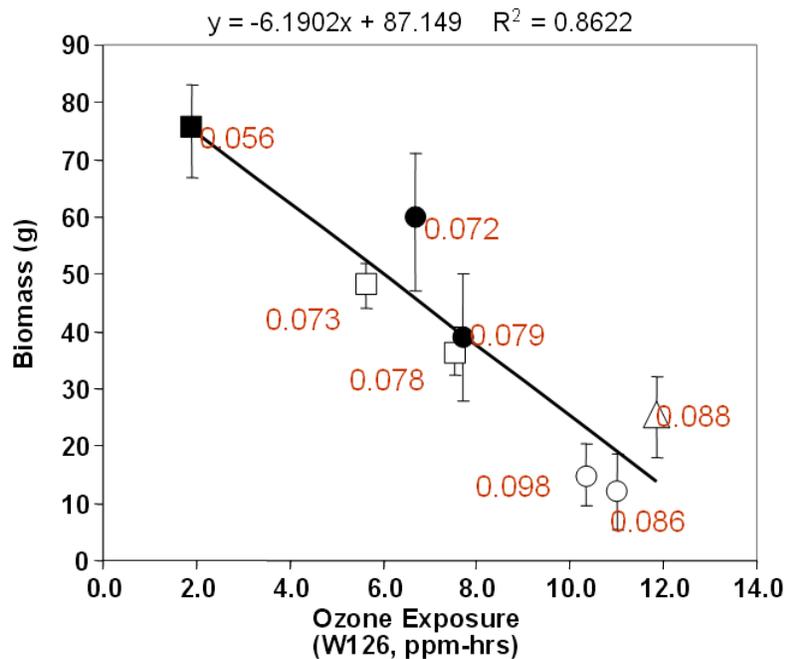
16

17 **Figure 5-3. Association of O_3 with cottonwood biomass in gradient downwind of New**
18   **York City (Gregg et al., 2003).** Symbol Key: Squares (1992); Circles (1993);
19   Triangles (1994); Shaded (urban); open (rural). Figure modified from Gregg et al.,
20   2003 and U.S. EPA, 2007 Figure 7-17 to add 8-hour average concentrations (red
21   font).

1   We additionally recognize some limitations in the extent to which some studies that look
2   at O_3-induced effects on tree growth, productivity and carbon storage can inform consideration
3   of effects that might occur under air quality conditions that meet the current standard. In some
4   cases this is because studies were designed to look only at what is happening under ambient
5   conditions and/or above ambient conditions and the ambient conditions often did not meet the
6   standard. In addition, because tree growth is known to be affected by cumulative exposures,
7   studies do not commonly provide exposure concentration data pertaining to daily 8-hour
8   averages. Unless hourly O_3 concentration data are provided, translation from one index to
9   another is not usually feasible. Thus, to consider effects that might occur under conditions that
10   meet the current standard we have traditionally relied on monitored hourly O_3 air quality data to
11   calculate both types of exposure metrics (i.e. cumulative and 8-hour average) used in
12   combination with the robust tree seedling C-R functions developed in OTC studies (as described
13   above) to predict the magnitude of the growth effects associated with exposure concentrations at
14   or below that of the current standard. These tree seedling C-R functions are also used in the
15   WREA estimates of effects on tree growth that might be expected to remain under air quality
16   conditions that just meet the current secondary standard (discussed in section 5.2.2 below).

17   - **To what extent does currently available evidence suggest locations where the**
18   **vulnerability of sensitive species, ecosystems and/or their associated services to**
19   **O_3-related effects on forest tree growth, productivity and carbon storage would**
20   **have special significance to the public welfare?**

21   Areas with special significance to the public welfare include federally designated Class I,
22   non-Class I national parks, and other areas set aside to provide similar public welfare benefits.
23   Therefore Table 5-2 provides some examples of Class I sites where the current secondary
24   standard is met but W126 index values fall above 15 ppm-hours. At W126 index values above
25   15 ppm-hours, relative biomass loss in seedlings might be expected to range from less than 1%
26   to more than 30% across the 12 species for which C-R functions have been developed (as
27   described above). Relative biomass loss above 5% might be expected in seedlings of six of the
28   12 species and above 2% in seedlings (but less than 5%) in a seventh, indicating that if present in
29   these specially protected areas, meeting the current standard may not prevent W126 index values
30   that may cause these levels of growth effect.

31

32

5-18

1 **Table 5-2. Examples of counties containing Class I areas where recent air quality might be**
2 **expected to meet the current standard and where 3-yr W126 index values are**
3 **above 15 ppm-hrs.**

Monitor ID	Years	3-yr 8-hr max	3-yr W126 (annual values)	State/Co.	Class I Area
806710041	2008-2010	71	15 (16, 11, 18)	Colorado/ La Plata	Weminuche WA[a]
806710041	2009-2011	74	17 (11, 18, 21)	Colorado/ La Plata	Weminuche WA
806710041	2010-2012	73	19 (18, 21, 18)	Colorado/ La Plata	Weminuche WA
80830101	2006-2008	71	18 (24, 18, 14)	Colorado/ Montezuma	Mesa Verde NP[b]
80830101	2007-2009	69	16 (18, 14, 15)	Colorado/ Montezuma	Mesa Verde NP
4005800	2006-2008	70	19 (22,19, 22)	Arizona/ Coconino	Grand Canyon NP
4005800	2007-2009	68	15 (19, 22, 11)	Arizona/ Coconino	Grand Canyon NP
4005800	2010-2012	72	18 (15, 18, 20)	Arizona/ Coconino	Grand Canyon NP
49053013	2006-2008	71	21 (24, 19, 20)	Utah/ Washington	Zion NP
49053013	2007-2009	70	18 (19, 20, 15)	Utah/ Washington	Zion NP
49053013	2008-2010	70	18 (20, 15, 21)	Utah/ Washington	Zion NP
49053013	2009-2011	70	18 (15, 21,18)	Utah/ Washington	Zion NP
49053013	2010-2012	73	20 (21, 18, 22)	Utah/ Washington	Zion NP
49037010	2006-2008	71	18 (19, 18, 18)	Utah/ San Juan	Canyonlands NP
49037010	2007-2009	70	16 (18, 18, 13)	Utah/ San Juan	Canyonlands NP
49037010	2010-2012	69	15 (14, 14, 17)	Utah/ San Juan	Canyonlands NP

Based on data from http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdata.htm
 a = Wilderness Area; b= National Park

4

1   ## 5.2.2   Exposure/Risk-based Considerations

2   The WREA presents a number of quantitative analyses of cumulative exposure and risk

3   related to tree growth, productivity and carbon storage for air quality scenarios intended to

4   inform our consideration of exposure and risk for considerations associated with the current

5   standard (see Table 5-3 below, U.S. EPA, 2014, chapter 6).

6   **Table 5-3. Exposure, risk and ecosystem services analyses related to tree growth,**
7   **productivity and carbon storage.**

	Species Level Effects	Ecosystem Level Effects	Ecosystem Services
WREA estimates[A]		Percent of total geographic area[B] with annual relative biomass loss above 2% Number of assessed Class I areas with annual relative biomass loss above 2%	• Economic surplus to timber producers and consumers (WREA, Table 6-11) • Carbon storage, nationally (WREA, Table 6-18) • Carbon storage, in 5 urban areas (WREA, Table 6-20) • Air pollutant removal in 5 urban areas (WREA, Table 6-21)
[A] See WREA chapter 6. [B] The total geographic area includes the contiguous U.S..			

8

9   The relevant quantitative exposure and risk analyses for tree biomass loss, productivity

10   and carbon storage include:

11   1) Species-specific and composite biomass loss estimates.

12   2) National-scale assessments for: a) basal area weighted relative biomass loss for tree

13   seedlings; b) timber production; c) carbon sequestration.

14   3) Case study-scale assessments for: a) carbon sequestration; b) air pollution removal.

15   • **For what air quality scenarios were exposures and risks estimated? What**
16   **approaches were used to estimate W126 exposures for those conditions? What**
17   **are associated limitations and uncertainties?**

18   In the analyses related to O_3 effects on tree growth, productivity and carbon storage,

19   quantitative estimates were developed for five air quality scenarios by the methodology

20   summarized in Table 5-4 below. In general, this methodology involved two steps. The first is

21   derivation of the average W126 value (across the three years) at each monitor location. This

22   value is based on unadjusted data for recent conditions and model-adjusted concentrations for the

1   four other scenarios. The development of model adjusted concentrations was done for each of 9

2   regions independently (see U.S. EPA, 2014, section 4.3.4.1). In the second step, national-scale

3   spatial surfaces (W126 values for each model grid cell) were created using the monitor-location

4   values and the Voronoi Neighbor Averaging (VNA) spatial interpolation technique (details on

5   the VNA technique are presented in U.S. EPA, 2014 Appendix 4A).

6   **Table 5-4. Summary of methodology by which national surface of average W126 index**
7   **values was derived for each air quality scenario.**

| Scenario | Development of W126 values for Each Air Quality Scenario | |
	Monitor-location-specific calculations and any model adjustment	Derivation of national surface of average W126 index values
Recent Conditions (2006-2008)	An annual W126 index value is calculated for each year at each monitor location, using the highest 3-month period. A location-specific 3-year W126 was calculated by averaging annual W126 index values from 3 consecutive years which may have used different 3-month periods..	The VNA method is applied to the monitor-location average W126 values to create a national distribution of W126 values within model grid-cells for each scenario.
Current Standard	2006-2008 hourly O_3 concentrations at each monitor location are model-adjusted[A] to create a three year record of O_3 concentrations that just meets the current standard (see WREA, section 4.3.4). A seasonal W126 index value is calculated for each year at each monitor location using the same 3-month period for each year (which is the highest as a 3-yr average and is highest in at least one of the years). A location-specific average is derived from these three index values.	
Average W126 Index of 15 ppm-hrs	Hourly O_3 concentrations at each monitor location, within each modeling region, are model-adjusted to create a record for which the highest location-specific average index value in the region (the controlling location) just meets the scenario target index value.	
Average W126 Index of 11 ppm-hrs		
Average W126 Index of 7 ppm-hrs	A seasonal W126 index value is calculated for each year (of 2006-2008 period) at each monitor location, using the same 3-month period for each year (which is the highest in at least one of the years). A location-specific average is derived from these three index values.	

[A] The model-based adjustment approach is based on regional emission reduction scenarios at monitor sites followed by spatial interpolation for broader spatial coverage. See WREA, chapters 4 and 7, and Appendices 4A and 7A).

1

2   During the recent conditions period (2006 through 2008), the average W126 index values
3   (across the three-year recent conditions period) at the monitor locations ranged from below 10
4   ppm-hrs to 48.6 ppm-hrs (U.S. EPA 2014, Figure 4-4 and Table 4-1). Across the nine modeling
5   regions, the maximum average W126 index values ranged from 48.6 ppm-hrs in the west region
6   down to 6.6 ppm-hrs in the northwest region.   After model-adjustment of the 2006-2008 data to
7   just meet the current standard in each region, the region-specific maximum values range from
8   18.9 ppm-hrs in the west region to 2.6 ppm-hrs in the northeast region (U.S. EPA, 2014, Table 4-
9   1). After application of the VNA technique to the current standard scenario monitor location
10   values, the average W126 values was below 15 ppm-hrs across the national surface with the
11   exception of a very small area of the southwest region (near Phoenix) where the average W126
12   values was near or just above 15 ppm-hrs. A lowering of the highest values occurred with
13   application of the interpolation method as a result of estimating W126 values at a 12x12 km^2 grid
14   resolution rather that at the exact location of a monitor. This indicates one uncertainty associated
15   with this aspect of the approach to estimating W126 values for the model-adjusted air quality just
16   meeting the current standard.
17   The WREA also recognizes other sources of uncertainty for the W126 estimates for each
18   air quality scenario and qualitatively characterizes the magnitude of uncertainty and potential for
19   directional bias. These sources of uncertainty are described in more detail in the WREA Chapter
20   4 and summarized below. Because the W126 estimates generated in the air quality analyses are
21   inputs to the vegetation risk analyses for biomass loss and foliar injury, any uncertainties in the
22   air quality analyses are propagated into the those analyses (U.S. EPA 2014, section 8.5).
23   An important large uncertainty in the analyses is the assumed response of the W126
24   concentrations to emissions reductions needed to meet the existing standard (U.S. EPA, 2014,
25   section 8.5.1). We note that any approach to characterizing O_3 air quality over broad geographic
26   areas based on concentrations at monitor locations will convey inherent uncertainty. The model-
27   based adjustments, based on U.S.-wide emissions reductions in oxides of nitrogen (NOx), do not
28   represent air quality distributions from an optimized control scenario that just meets the current
29   standard (or target W126 values for other scenarios), but characterize one potential distribution
30   of air quality across a region when all monitor locations meet the standard (U.S. EPA 2014,

1   section 4.3.4.2).[7] An additional uncertainty comes from the creation of a national W126 surface

2   using the VNA technique to interpolate recent air quality measurements of O_3. In general, spatial

3   interpolation techniques perform better in areas where the O_3 monitoring network is denser.

4   Therefore, the W126 estimated in the rural areas in the West, Northwest, Southwest, and West

5   North Central with few or no monitors (Figure 2-1) are more uncertain than those estimated for

6   areas with more dense monitoring[8]. Additionally, the surface is created from the three-year

7   average at the monitor locations, rather than creating a surface for each year and then averaging

8   across years at each grid cell; the potential impact of this on the resultant estimates is considered

9   in the WREA (U.S. EPA, 2014, Appendix 4A).

10 - **What are the nature and magnitude of exposure- and risk-related estimates for**
11 **tree growth, productivity, and carbon storage under recent conditions or**
12 **conditions remaining upon meeting the current standard? To what extent are**
13 **these exposures and risks important from a public welfare perspective?**

14   The WREA used the C-R functions for 12 species described above with information on

15 the distribution of those species across the U.S., and average W126 exposure estimates to

16 estimate relative biomass loss for each of the studied species for each national air quality

17 scenario (U.S. EPA, 2014, section 6.2.1.3 and Appendix 6A). For example, the estimates of

18 relative biomass loss of Ponderosa Pine for air quality model-adjusted to just meet the current

19 standard are illustrated in Figure 5-4 below. While relative biomass loss below 2% is estimated

20 for most areas where this species is found, estimates in some areas of the southwest fall in a bin

21 defined as 2.01-4% biomass loss (U.S. EPA 2014, Figure 6-7 and Appendix 6A).

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[7] Because our analyses used U.S.-wide NOx emissions reductions to simulate just meeting the existing standard independently in each region, there are broad regional reductions in O_3 even in meeting standards in urban areas when targeting a few high-ozone urban monitors for reductions. However, the assumption of broad regional or national NOx reductions are not unreasonable given current EPA regulations such as the Clean Air Interstate Rule (CAIR), which requires NOx cuts across the Eastern U.S. to reduce regional ozone transport, and the multitude of onroad and offroad mobile source rules that will lead to reduction in NOx from these sources across the country in future years.

Relative Biomass Loss

- 0.09% - 1%
- 1.01% - 2%
- 2.01% - 4%
- 4.01% - 7%
- 7.01% - 10%
- 10.01% - 15%
- 15.01% - 20%
- 20.01% - 20%

1
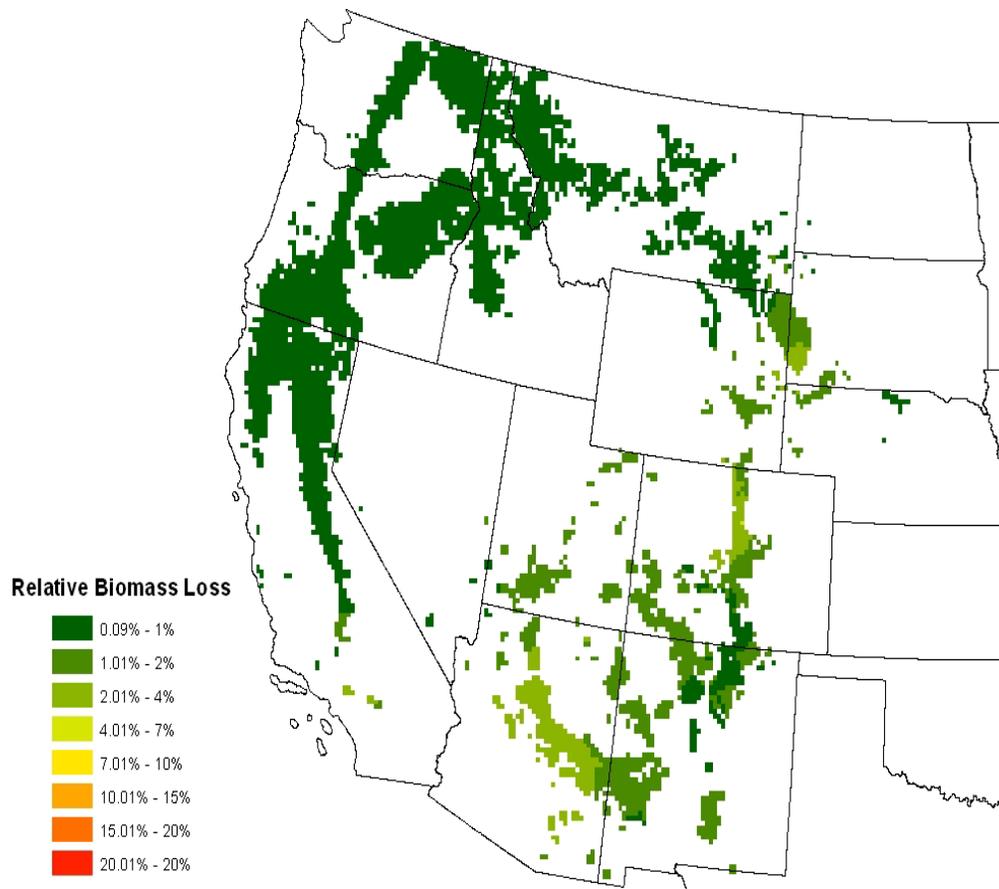
Figure 5-4. Relative biomass loss of Ponderosa Pine for air quality model-adjusted to just meet the current standard (U.S. EPA 2014, Figure 6-7).

The WREA also developed national-scale estimates of O_3 biomass ecosystem-level impacts considering the 12 studied species together (U.S. EPA 2014, section 6.8, Table 6-22). This was done using the species-specific biomass loss C-R functions, information on prevalence of the studied species across the U.S., and a weighting approach based on proportion of the basal area that each species contributes. The RBL values for multiple tree species were weighted by their basal area and combined into a weighted RBL value. The weighted relative biomass loss (wRBL) is intended to inform our understanding of the potential magnitude of the ecological effect that could occur in some ecosystems. Specifically, the more basal area that is affected in a given ecosystem, the larger the potential ecological effect. A wRBL value for each grid cell is generated by weighting the RBL value for each studied tree species found within that grid cell by the proportion of basal area it contributes to the total basal area of all tree species within the grid cell, and then summing those individual wRBLs. The percent of total basal area that exceeds a 2% weighted relative biomass loss in the recent conditions scenario is 10.1% (U.S. EPA 2014, Table 6-24). Based on the average W126 values estimated for the air quality scenario just

1　meeting the current standard across the contiguous U.S., the WREA estimates 0.8 % of the total

2　geographic area to have a wRBL above 2% (U.S. EPA 2014, Table 6-24).

3　　　　We also consider WREA estimates (quantitative and qualitative) of effects on several

4　ecosystem services. First, impacts on growth related to O_3 concentrations in federally-designated

5　Class I areas were derived from an average weighted RBL value (discussed above) for 145 of the

6　155 Class I areas (U.S. EPA 2014, section 6.8.1). Given established objectives for Class I areas

7　(e.g., to maintain in perpetuity), effects in Class I areas may be considered to have the potential

8　to adversely affect the intended use of the ecosystem, i.e., to maintain in pristine natural

9　conditions for future generations.   For the recent conditions scenario, this analysis estimates

10　wRBL values above 2% in 13 of the 145 assessed Class I areas. In comparison, the analysis

11　estimates wRBL values above 2% in only 2 areas based on average W126 values estimated for

12　the current standard scenario (U.S. EPA 2013, Table 6-26).

13　　　　The WREA also presents national-scale estimates of the effects of biomass loss on timber

14　production and agricultural harvesting, as well as on carbon sequestration. The WREA used the

15　O_3 C-R functions for tree seedlings to calculate relative biomass loss. Because the forestry and

16　agriculture sectors are related, and trade-offs occur between the sectors, the WREA also

17　calculated the resulting market-based welfare effects of O_3 exposure in the forestry and

18　agriculture sectors. In the analyses for commercial timber production, based on the average

19　W126 values estimated for the air quality scenario just meeting the existing standard, relative

20　biomass losses (RBL) estimates were below one percent in all regions except the Southwest,

21　Southeast, Central, and South regions (U.S. EPA, 2014, section 6.3, Table 6-8) (see U.S. EPA,

22　2014, Table 6-14 for clarification on region names)). Relative biomass losses remain above one

23　percent for the average W126 scenarios for 15 and 11 ppm-hrs in parts of the Southeast, Central,

24　and South regions, and for the 7 ppm-hr scenario in the Southeast and South regions (U.S. EPA,

25　2014, section 6.3, Table 6-8).

26　　　　In addition to estimating changes in forestry and agricultural yields, the WREA presents

27　estimated changes in consumer and producer/farmer surplus associated with the change in yields.

28　Changes in biomass affect individual tree species, but the overall effect on forest ecosystem

29　productivity depends on the composition of forest stands and the relative sensitivity of trees

30　within those stands. Economic welfare impacts resulting from just meeting the existing and

31　alternative standards were largely similar between the forestry and agricultural sectors --

32　consumer surplus, or consumer gains, generally increased in both sectors because higher

33　productivity under lower O_3 concentrations increased total yields and reduced market prices.

34　Comparisons are not straightforward to interpret due to market dynamics. For example, because

35　demand for most forestry and agricultural commodities is not highly responsive to changes in

1   price, there were more examples for which producer surplus, or producer gains declines. In
2   some cases, lower prices reduce producer gains more than can be offset by higher yields.[9] In
3   general, the WREA estimates benefits for agricultural yield of approximately 1 billion dollars
4   from differences in average W126 index values representing recent conditions from those
5   representing model-adjusted air quality for the current standard (U.S. EPA 2014, section 6.3).
6   The national-scale analysis of carbon dioxide (CO_2) sequestration estimates substantially more
7   storage under the current standard compared to recent conditions (U.S. EPA 2014, section 6.6.1,
8   Table 6-18, Appendix 6B). In considering the significance of the potential climate and
9   ecosystem service impact, we also take note of the large uncertainties associated with this
10 analysis (see U.S. EPA 2014, Table 6-27).

11   We additionally consider the WREA estimates of tree growth and ecosystem services
12 provided by urban trees over a 25-year period, for five urban areas based on case-study scale
13 analyses that quantified the effects of biomass loss on carbon sequestration and pollution
14 removal (U.S. EPA 2014, sections 6.6.2 and 6.7).[10] The urban areas included in this analysis
15 represent diverse geography in the Northeast, Southeast, and Central regions, although do not
16 include an urban area in the western part of the U.S. Estimates of the effects of O_3-related
17 biomass loss on carbon sequestration indicate the potential for an increase of somewhat more
18 than a million metric tons of CO_2 equivalents for average W126 values associated with meeting
19 for the current standard scenario as compared to recent conditions. Somewhat smaller increases
20 are estimated for the three W126 scenarios in comparison to the current standard scenario (U.S.
21 EPA 2014, section 6.6.2 and Appendix 6D).

22   In addition to the quantitative assessments discussed above, qualitative assessments for
23 some ecosystem services, such as commercial non-timber forest products and recreation (U.S.
24 EPA, 2014, section 6.4), aesthetic and non-use values (U.S. EPA, 2014, section 6.4), increased
25 susceptibility to insect attack and fire damage (U.S. EPA, 2014, sections 5.3 and 5.4,
26 respectively), were also conducted. Other ecological effects that are causally or likely causally
27 associated with O_3 exposure such as terrestrial productivity, water cycle, biogeochemical cycle,
28 and community composition (U.S. EPA 2013, Table 9-19) were not directly addressed in the
29 WREA due to a lack of sufficient quantitative information.

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[9] The relative biomass loss estimates were also used with the Forest and Agricultural Sector Optimization Model with Greenhouse Gases (FASOMGHG). FASOMGHG is a national-scale model that provides a complete representation of the U.S. forest and agricultural sectors' impacts of meeting alternative standards. FASOMGHG simulates the allocation of land over time to competing activities in both the forest and agricultural sectors. FASOMGHG results include multi-period, multi-commodity results over 60 to 100 years in 5-year time intervals when running the combined forest-agriculture version of the model. See Chapter 6, Section 6.3 of the WREA for a discussion of economic welfare and consumer and producer surplus.

[10] The model, iTree, a peer-reviewed suite of software tools provided by USFS was used in this analysis.

1   There is substantial heterogeneity in plant responses to O_3, both within species, between
2   species, and across regions of the U.S. The O_3-sensitive tree species are different in the eastern
3   and western U.S. -- the eastern U.S. has far more species. Ozone exposure and risk is somewhat
4   easier to assess in the eastern U.S. because of the availability of more data and the greater
5   number of species to analyze. In addition, there are more O_3 monitors in the eastern U.S. but
6   fewer national parks (U.S. EPA, 2014, chapter 8).

7   - **What are the uncertainties associated with both quantitative and qualitative**
8   **information?**

9   Several key limitations and uncertainties, which may have a large impact on both overall
10 confidence and confidence in individual analyses, are discussed here. Key uncertainties
11 associated with the assessment of impacts on ecosystem services at the national and case-study
12 scales, as well as across species, U.S. geographic regions and future years include those
13 associated with the following seedling C-R functions, as well as interpolated and model-adjusted
14 O_3 concentrations used to estimate W126 exposures in the REA air quality scenarios. The
15 uncertainties in the W126 exposure estimates are discussed above at the beginning of section
16 5.2.2.
17   With regard to the seedling C-R functions, the description of Figure 5-2 above provides
18   some characterization of the variability of individual study results and the impact of that on
19   estimates of W126 index values that might elicit different percentages of biomass loss in tree
20   seedlings (U.S. EPA, 2014, section 6.2.1.2). Even though the evidence shows that there are
21   additional species adversely affected by O_3-related biomass loss, the WREA only has C-R
22   functions available to quantify this loss for 12 tree species and 10 crop species. This absence of
23   information only allows a partial characterization of the O_3-related biomass loss impacts in trees
24   and crops associated with recent O_3 index values and with just meeting the existing and
25   potential alternative secondary standards. In addition, there are uncertainties inherent in these C-
26   R functions, including the extrapolation of relative biomass loss rates from tree seedlings to
27   adult trees and information regarding within-species variability. The overall confidence in the
28   C-R function varies by species based on the number of studies available for that species. Some
29   species have low within-species variability (e.g., many agricultural crops) and high
30   seedling/adult comparability (e.g., Aspen), while other species do not (e.g., Black Cherry). The
31   uncertainties in the C-R functions for biomass loss and in the air quality analyses are propagated
32   into the analysis of the impact of biomass loss on ecosystem services, including provisioning
33   and regulating services (U.S. EPA, 2014, Table 6-27). The WREA characterizes the direction
34   of potential influence of C-R function uncertainty as unknown, yet its magnitude as high,

1   concluding that further studies are needed to determine how accurately the assessed species
2   reflect the larger suite of O_3-sensitive tree species in the U.S. (U.S. EPA, 2014, Table 6-27).
3   Another uncertainty associated with interpretation of the WREA biomass loss-related
4   estimates concerns the potential for underestimation of compounding of growth effects across
5   multiple years of varying concentrations. Though tree biomass loss impacts were estimated
6   using air quality scenarios of 3-year average W126 values, the WREA also conducted an
7   analysis to compare the impact of using a variable compounding rate based on yearly variations
8   in W126 exposures to that of using a W126 index value averaged across three years. The WREA
9   compared the compounded values for two examples. In both examples, one species (Tulip Polar
10   and Ponderosa Pine) and one climate region where that species occurred (Southeast and
11   Southwest regions) were chosen and air quality values associated with just meeting the existing
12   standard of 75 ppb were used. Within each region the WREA analysis used both the W126 value
13   at each monitor in the region for each year and the three-year average W126 value using the
14   method described in Chapter 4. The results show that the use of the three-year average W126
15   index value may underestimate RBL values slightly, but the approach does not account for
16   moisture levels or other environmental factors that could affect biomass loss (U.S. EPA, 2014,
17   section 6.2.1.4 and Figure 6-13). In considering these results, we note that in both regions and in
18   all three years, the three-year average W126 value is sometimes above and sometimes below the
19   individual year W126 index value.

20   In the national-scale analyses of timber production, agricultural harvesting, and carbon
21   sequestration, the WREA used the FASOMGHG model, which includes functions for carbon
22   sequestration, assumptions regarding proxy species, and non-W126 C-R functions for three
23   crops. However, FASOMGHG does not include agriculture and forestry on public lands,
24   changes in exports due to O_3 into international trade projections, or forest adaptation. Despite
25   the inherent limitations and uncertainties, the WREA concludes that the FASOMGHG model
26   reflects reasonable and appropriate assumptions for a national-scale assessment of changes in
27   the agricultural and forestry sectors due to changes in vegetation biomass associated with O_3
28   exposure (U.S. EPA, 2014, sections 6.3, 6.5, 6.6, and 8.5.2).

29   In the case study analyses of five urban areas, the WREA used the iTree model, which
30   includes an urban tree inventory for each area and species-specific pollution removal and carbon
31   sequestration functions. However, iTree does not account for the potential additional VOC
32   emissions from tree growth, which could contribute to O_3 formation. Despite the inherent
33   limitations and uncertainties, the WREA concludes that the iTree model reflects reasonable and

1   appropriate assumptions for a case study assessment of pollution removal and carbon

2   sequestration for changes in biomass associated with O_3 exposure (U.S. EPA, 2014, sections

3   6.6.2, 6.7, and 8.5.2).

4   The overall effect of the combined set of uncertainties on confidence in the interpretation

5   of the results of the analyses is difficult to quantify. Due to differences in available information,

6   the degree to which each analysis was able to incorporate quantitative assessments of uncertainty

7   differed.

5.3   CROP YIELD LOSS

9   This section considers the current evidence and exposure/risk information to inform

10   consideration of the adequacy of the protection provided by the current standard from known and

11   anticipated adverse welfare effects of O_3 related to crop yield and other associated effects. Crops

12   are important from a public welfare perspective because they provide food and fiber services to

13   humans. This section includes a discussion of the policy-relevant science and weight-of-

14   evidence conclusions discussed in the ISA (section 5.3.1) and the exposure/risk results (section

15   5.3.2) described in the second draft WREA. Important uncertainties and limitations in the

16   available information are discussed throughout the sections. These discussions highlight the

17   information we consider relevant to answering the overarching question and associated policy-

18   relevant questions included in this section.

5.3.1   Evidence-based Considerations

20   Ozone can interfere with carbon gain (photosynthesis) and allocation of carbon. As a

21   result of decreased carbohydrate availability, fewer carbohydrates are available for plant growth,

22   reproduction, and/or yield. For seed-bearing plants, these reproductive effects will culminate in

23   reduced seed production or yield. The detrimental effect of O_3 on crop production has been

24   recognized since the 1960s, and current O_3 concentrations in many areas across the U.S. are high

25   enough to cause yield loss in a variety of agricultural crops including, but not limited to,

26   soybeans, wheat, potatoes, watermelons, beans, turnips, onions, lettuces, and tomatoes.

27   Increases in O_3 concentration may further decrease yield in these sensitive crops while also

28   causing yield losses in less sensitive crops (U.S. EPA 2013, section 9.4.4). The ISA concluded

29   that the evidence is sufficient to determine that there is a causal relationship between O_3

30   exposure and reduced yield and quality of agricultural crops (U.S. EPA 2013, Table 2-2).

1 • **To what extent has scientific information become available that alters or**
2 **substantiates our prior conclusions regarding O₃-related crop yield loss and of**
3 **factors that influence associations between O₃ levels and crop yield loss?**

4 In general, the vast majority of the new scientific information has substantiated our prior
5 conclusions regarding O_3 crop yield loss. On the whole, this evidence supports previous
6 conclusions that exposure to O_3 decreases growth and yield of crops. The ISA describes average
7 yield loss reported across a number of meta-analytic studies have been published recently for
8 soybean wheat, rice, semi-natural vegetation, potato, bean and barley (U.S. EPA 2013, section
9 9.4.4.1). Meta-analysis allows for the objective development of a quantitative consensus of the
10 effects of a treatment across a wide body of literature. Further, several new exposure studies
11 continue to show decreasing yield and biomass in a variety of crops with increased O_3 exposure
12 (U.S. EPA 2013, section 9.4.4.1, Table 9-17). Research has linked increasing O_3 concentration
13 to decreased photosynthetic rates and accelerated aging (U.S. EPA 2013, section 9.4.4) in leaves,
14 which are related to yield. Recent research has highlighted the effects of O_3 on crop quality.
15 Increasing O_3 concentration decreases nutritive quality of grasses, decreases macro- and micro-
16 nutrient concentrations in fruits and vegetable crops (U.S. EPA 2013, section 9.4.4). The
17 findings of these studies did not change our understanding of O_3-related crop loss and little
18 information has emerged on factors that influence associations between O_3 levels and crop yield
19 loss.

20 • **To what extent have important uncertainties identified in the last review been**
21 **reduced and/or new uncertainties emerged?**

22 Important uncertainties have been reduced regarding the confidence placed in using crop
23 exposure-response functions, especially for soybean. In general, the ISA reports consistent
24 results across exposure techniques and across crop varieties.
25 Two important uncertainties have been reduced regarding the C-R functions for yield
26 effects of O_3 in crop species, especially for soybean. First, in the last several reviews, the extent
27 to which C-R functions developed in OTC predicted plant responses in the field and under
28 different exposure conditions was not clear. In this review, staff from the EPA's ORD/NCEA
29 performed an analysis comparing OTC data with field-based data for one crop and one tree
30 species (U.S. EPA, 2013, section 9.6.3.2). The crop comparison was done using soybean OTC
31 data from NCLAN and field-based data from Soy FACE. The NCLAN studies were undertaken
32 in the early to mid 1980's and provide the largest, most uniform database on the effects of O_3 on
33 agricultural crop yields (U.S. EPA 1996; U.S. EPA 2006; U.S. EPA 2013, sections 9.2, 9.4, and

1   9.6). [11] The Soy FACE experiment was a chamberless field-based exposure study in Illinois that
2   was conducted from 2001 – 2009 (U.S. EPA 2013, section 9.2.4). Yield loss in soybean from O_3
3   exposure at the Soy FACE field experiment was reliably predicted by soybean C-R functions
4   developed in NCLAN (U.S. EPA, 2013, Section 9.6). This analysis supports the robustness and
5   use of the C-R functions developed in NCLAN to predict relative yield loss to O_3 exposure.
6   A second area of uncertainty that was reduced is that regarding the application of the
7   NCLAN C-R functions, developed in the 1980s, to more recent cultivars currently growing in the
8   field. However, recent studies continue to find yield loss levels in crop species studied
9   previously under NCLAN that reflect the earlier findings. There has been little new evidence
10   that crops are becoming more tolerant of O_3 (U.S. EPA, 2006a; U.S. EPA 2013). This is
11   especially evident in the research on soybean. In a meta-analysis of 53 studies, Morgan et al.
12   (2003) found consistent deleterious effects of O_3 exposures on soybean from studies published
13   between 1973 and 2001. Further, Betzelberger et al. (2010) has recently utilized the SoyFACE
14   facility to compare the impact of elevated O_3 concentrations across 10 soybean cultivars to
15   investigate intraspecific variability of the O_3 response. The C-R functions derived for these 10
16   current cultivars were similar to the response functions derived from the NCLAN studies
17   conducted in the 1980s (Heagle, 1989), suggesting there has not been any selection for increased
18   tolerance to O_3 in more recent cultivars. The 2013 ISA reported comparisons between yield
19   predictions based on data from cultivars used in NCLAN studies, and yield data for modern
20   cultivars from SoyFACE (U.S. EPA, 2013, section 9.6.3). They confirm that the average
21   response of soybean yield to O_3 exposure has not changed in current cultivars. Thus, staff
22   concludes that at least for soybean, uncertainties associated with use of the NCLAN generated C-
23   R functions to estimate biomass loss in recent cultivars has been reduced.

24   - **To what extent does the available evidence indicate the occurrence of O_3-related
25   effects on crop yield loss attributable to cumulative exposures at lower ambient O_3
26   concentrations than previously established or to exposures at or below the level of
27   the current standard?**

28   Very little evidence has emerged to indicate a lower level cumulative exposures that can
29   affect crop yield (levels of concern), that have been based on C-R functions from OTC

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[11] The NCLAN protocol was designed to produce crop exposure-response data representative of the areas
in the U.S. where the crops were typically grown. In total, 15 species (e.g., corn, soybean, winter wheat, tobacco,
sorghum, cotton, barley, peanuts, dry beans, potato, lettuce, turnip, and hay [alfalfa, clover, and fescue]), accounting
for greater than 85 percent of U.S. agricultural acreage planted at that time, were studied. Of these 15 species, 13
species including 38 different cultivars were combined in 54 cases representing unique combinations of cultivars,
sites, water regimes, and exposure conditions. Crops were grown under typical farm conditions and exposed in
open-top chambers to ambient O_3, sub-ambient O_3, and above ambient O_3.

1 experiments. As described above, the new evidence has strengthened the basis for using the

2 information from the C-R functions.

3 Where the current evidence on crop yield loss is not in terms of parts per billion

4 concentrations over a specific exposure period such as eight hours, assessing whether O_3

5 concentrations associated with meeting the current standard would allow crop yield effects is

6 more complex. Where feasible, we have attempted to characterize the O_3 exposures associated

7 with crop yield loss in terms of seasonal W126 index and we have separately considered the

8 extent to which such index values might be expected to occur in agricultural locations that meet

9 the current standard. For example, Sedgwick and Sumner counties in Kansas met the level of the

10 3-year 8-hr standard of 75 ppb in 2011. However, the W126 for 2011 in those counties was 19

11 ppm-hours and would be predicted to result in a 9% yield loss for soybean grown in those

12 counties.

13 • **To what extent does currently available evidence suggest locations where the**
14   **vulnerability of sensitive species, ecosystems and/or their associated services to**
15   **O_3-related crop yield loss would have special significance to the public welfare?**

16 During the previous NAAQS reviews, there were very few studies that estimated O_3

17 impacts on crop yields at large geographical scales (i.e., regional, national or global). Recent

18 modeling studies of the historical iimpact of O_3 concentrations found that increased O_3 generally

19 reduced crop yield, but the impacts varied across regions and crop species (U.S. EPA, 2013,

20 Section 9.4.4.1). The largest O_3-induced crop yield losses were estimated to occur in high-

21 production areas exposed to elevated O_3 concentrations, such as the Midwest and the Mississippi

22 Valley regions of the United States. Among crop species, the estimated yield loss for wheat and

23 soybean were higher than rice and maize. Additionally, satellite and ground-based O_3

24 measurements have been used to assess yield loss caused by O_3 over the continuous tri-state area

25 of Illinois, Iowa, and Wisconsin. The results indicate that O_3 concentrations during the assessed

26 period reduced soybean yield, which correlates well with the previous results from FACE- and

27 OTC-type experiments (U.S. EPA 2013, section 9.4.4.1).

28 Thus, the recent scientific literature continues to support the conclusions of the 1996 and

29 2006 Criteria Documents and 2013 ISA that ambient O_3 concentrations can reduce the yield of

30 major commodity crops in the U.S. and support the use of crop C-R functions based on OTC

31 experiments. Agricultural areas that would be likely to have the most significance to the public

32 welfare would be those high production areas for sensitive crops that also are exposed to high O_3

33 concentrations, such as areas in the Midwest and Mississippi Valley regions.

1   **5.3.2   Exposure/Risk-based Considerations**

2   Two main analyses are conducted in the second draft WREA to estimate O_3 impacts

3   related to crop yield. Annual yield losses are estimated for 10 commodity crops and these

4   estimates are then additionally used to estimate O_3 impacts on producer and consumer economic

5   surpluses (Table 5-5 below; U.S. EPA, 2014, sections 6.2, 6.5).

6   **Table 5-5. Exposure, risk and ecosystem services analyses related to crop yield.**

	Crop-level impact[A]	Agri-Ecosystem Services[B]
REA estimates	Annual Relative Yield Loss Corn, Cotton, Potato, Sorghum, Soybean, Winter Wheat	Economic surplus to crop producers and consumers
[A]See section 6.2 WREA. [3]See section 6.5 WREA.		

7

8   •   **For what air quality scenarios were exposures and risks estimated? What**
9   **approaches were used to estimate W126 exposures for those conditions? What**
10   **are associated limitations and uncertainties?**

11   The WREA crop analyses described here were performed for five air quality scenarios by

12   the methodology summarized in Table 5-4 above. In general, this methodology involved two

13   steps. The first is derivation of the average W126 value (across the three years) at each monitor

14   location. This value is based on unadjusted data for recent conditions and model-adjusted

15   concentrations for the 4 other scenarios. The development of model adjusted concentrations was

16   done for each of 9 regions independently (see U.S. EPA, 2014, section 4.3.4.1). In the second

17   step, national-scale spatial surfaces (W126 values for each model grid cell) were created using

18   the monitor-location values and the Voronoi Neighbor Averaging (VNA) spatial interpolation

19   technique (details on the VNA technique are presented in U.S. EPA, 2014 Appendix 4A). The

20   results of model adjustments on estimated average W126 values and grid cell estimates produced

21   from the VNA interpolation approach is summarized in section 5.2.2 above. A lowering of the

22   highest values occurred with application of the interpolation method as a result of estimating

23   W126 values at a 12x12 km^2 grid resolution rather that at the exact location of a monitor. This

24   indicates one uncertainty associated with this aspect of the approach to estimating W126 values

25   for the model-adjusted air quality just meeting the current standard. Other areas of uncertainty

26   associated with the model adjustment and VNA interpolation approach are briefly summarized in

27   section 5.2.2 above and described in more detail in the WREA (U.S. EPA, 2014, chapter 4 and

28   Appendix 4A).

- **What is the nature and magnitude of the cumulative exposure- and risk-related estimates for crop yield loss associated with remaining upon simulating just meeting the current O$_3$ standard? What are the uncertainties associated this information?**

The WREA presents estimates of crop yield loss for the five air quality scenarios described above using C-R functions for commodity crops across the country (U.S. EPA, 2014, section 6.5). The largest reduction in O$_3$ exposure-related crop yield loss occurs when moving from the recent conditions scenario to that for just meeting the current standard (U.S. EPA, 2014, section 6.5). In the analyses for agricultural harvest, the largest estimates of yield changes also occur when comparing the recent conditions scenario to that for the current standard. Under recent conditions, the West, Southwest, and Northeast regions generally have the highest yield losses. For the three average W126 scenarios, relative yield losses for winter wheat[12] are less than one percent. For soybeans, yield losses for these scenarios range from just above 1 percent to below one percent (U.S. EPA 2014, section 6.5).

The WREA estimates of O$_3$-attributable percent yield loss based on average W126 values estimated for just meeting the current standard are relatively small (0.0 – 2.72%, U.S. EPA 2014, section 6.5, Appendix 6B). In considering these estimates, we recognize the significant uncertainties associated with several aspects of the analyses. Because the W126 estimates generated in the air quality analyses are inputs to the vegetation risk analyses for biomass loss, crop loss and foliar injury, any uncertainties in the air quality analyses are propagated into the those analyses (U.S. EPA 2014, Table 6-27, section 8.5).

- **To what extent are the exposures and risks remaining upon simulating just meeting the current O$_3$ standard important from a public welfare perspective?**

From a public welfare prospective, the O$_3$ attributable risks to crops estimated for conditions that just meet the current standard are small. As discussed in the WREA and summarized above, there are multiple areas of uncertainty associated with these estimates, including those associated with the model-based adjustment methodology as well as those associated with projection of yield loss at the estimated O$_3$ concentrations (U.S. EPA, 2014, Table 6-27, section 8.5).

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[12] Among the major crops, because winter wheat and soybeans are more sensitive to ambient O$_3$ levels than other crops we include these crops for this discussion.

- **What are the ecosystem services potentially affected by O₃-related crop yield loss and to what extent are they important from a public welfare perspective? To what degree can the magnitude of the O3 effect on these services be qualitatively or quantitatively characterized?**

The WREA presents national-scale estimates of the effects of biomass loss on timber production and agricultural harvesting, as well as on carbon sequestration (U.S. EPA 2014, section 6.5). Because the forestry and agriculture sectors are related, and trade-offs occur between the sectors, the WREA also calculated the resulting market-based welfare effects of O₃ exposure in the forestry and agriculture sectors. Overall effect on agricultural yields and producer and consumer surplus depends on the (1) ability of producers/farmers to substitute other crops that are less O₃ sensitive, and (2) responsiveness, or elasticity, of demand and supply (U.S. EPA, 2014, sections 6.5, 8.2.1.3). Estimated O₃-attributable economic welfare impacts on agricultural sectors associated with air quality conditions model-adjusted to just meet the existing and potential alternative W126 standard levels were largely similar between the forestry and agricultural sectors. Estimates of consumer surplus, or consumer gains, were generally higher under those conditions (compared to recent conditions) in both sectors because higher productivity under lower O₃ concentrations increased total yields and reduced market prices (U.S. EPA 2014, Table 6-16). Because demand for most forestry and agricultural commodities is not highly responsive to changes in price, there were more examples for which producer surplus, or producer gains, decline. For agricultural welfare, annualized combined consumer and producer surplus gains were estimated to be $2.6 trillion for model adjustment to meet the current standard. Combined gains were essentially unchanged in comparisons of the current standard scenario to the average W126 scenario for 15 ppm-hrs, but gains increased by $21 million for the W126 scenario for 11 ppm-hrs and $231 million for the W126 scenario for 7 ppm-hrs. In some cases, lower prices reduce producer gains more than can be offset by higher yields (U.S. EPA, 2014, Table 6-17).

The WREA discusses multiple areas of uncertainty associated with these estimates (also summarized above), including those associated with the model-based adjustment methodology as well as those associated with projection of yield loss at the estimated O₃ concentrations (U.S. EPA, 2014, Table 6-27, section 8.5).

5.4 VISIBLE FOLIAR INJURY

Visible foliar injury resulting from exposure to O₃ has been well characterized and documented over several decades of research on many tree, shrub, herbaceous, and crop species (U.S. EPA, 2013, 2006, 1996, 1984, 1978). The significance of O₃ injury at the leaf and whole plant levels depends on an array of factors and there is difficulty in relating visible foliar injury

1 symptoms to other vegetation effects such as individual tree growth, or effects at population or
2 ecosystem characteristics (U.S. EPA, 2013, p. 9-39). Visible foliar injury by itself, however, can
3 impact the public welfare through damaging or impairing the intended use of the affected entity
4 or the service it provides. For example, ways by which O_3-induced visible foliar injury may
5 impact the public welfare thus include: 1) visible damage to ornamental species or leafy crops
6 (spinach, lettuce, tobacco) that affects the economic value, yield, or usability of that plant (U.S.
7 EPA 2007, section 7.4.1; Abt Associates, Inc. 1995); 2) visible damage to plants with special
8 cultural significance (e.g., those used in tribal practices); 3) visible damage to species occurring
9 in natural settings valued for their scenic beauty and/or recreational appeal, including in areas
10 specially designated for more protection (i.e., federal Class I areas) (73 FR 16490). Given
11 limitations in the available information pertaining to the first two categories, [13] the discussions of
12 the evidence and exposure/risk information in sections 5.4.1 and 5.4.2 below focus primarily on
13 what is known about visible foliar injury that has been shown to occur in natural settings valued
14 for their scenic beauty and/or recreational appeal.

15   At the time of the last review, the following was known:

16   1) Ozone causes diagnostic visible injury symptoms on studied bioindicator species.

17   2) Soil moisture is a major confounding effect that can decrease the incidence and
18   severity of visible foliar injury under dry conditions and visa versa.

19   3) The most extensive dataset regarding visible foliar injury incidence across the U.S.
20   was that collected by the U.S. Forest Service (USFS) Forest Health Monitoring/Forest
21   Inventory and Analysis (FHM/FIA) Program.

22   4) Staff analyses of county level air quality data and USFS biomonitoring data showed
23   that for each year within a four year period (2001 – 2004) the percentage of counties
24   having a biosite with visible foliar injury ranged between 11-30% at an 8-hour average
25   annual level of 0.074 ppm (U.S. EPA, 2007, section 7.6.3.2).

26   In the remainder of this section, we consider how the currently available evidence and
27 exposure/risk information informs our understanding of the relationship that exists between
28 visible foliar injury and exposures to O_3 in ambient air and consideration of the adequacy of
29 protection provided by the current standard. The policy-relevant evidence and weight-of-
30 evidence conclusions drawn from the ISA are discussed in section 5.4.1, and the exposure/risk
31 and associated ecosystem services estimates from the second draft WREA, are discussed in
32 section 5.4.2. Important uncertainties and limitations in each type of available information are
33 also discussed in these two sections.

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[13] Qualitative information regarding potential cultural impacts of O_3-induced visible foliar injury is noted in section 5.5 and Appendix 5A).

1   **5.4.1   Evidence-based Considerations**

2   • **To what extent has scientific information become available that alters or**
3   **substantiates our previous conclusions of O$_3$-related visible foliar injury and of**
4   **factors that influence associations between O$_3$ exposures or concentrations and**
5   **visible foliar injury?**

6   Recent research continues to build and substantiate the previous conclusions and findings

7   drawn from several decades of research on many tree, shrub, herbaceous, and crop species (U.S.

8   EPA, 2013, 2006, 1996, 1984, 1978) that O$_3$-induced visible foliar injury symptoms are well

9   characterized and considered diagnostic on certain bioindicator plant species. Diagnostic usage

10   for these plants has been verified experimentally in exposure-response studies, using exposure

11   methodologies such as continuous stirred tank reactors (CSTRs), open-top chambers (OTCs),

12   and free-air fumigation (FACE). Although there remains a lack of robust exposure-response

13   functions that would allow prediction of visible foliar injury severity and incidence under

14   varying air quality and environmental conditions, experimental and observational evidence has

15   clearly established a consistent association of the presence of visible injury symptoms with O$_3$

16   exposure, with greater exposure often resulting in greater and more prevalent injury (U.S. EPA

17   2013, section 9.4.2). This new research includes: 1) controlled exposure studies conducted to

18   test and verify the O$_3$ sensitivity and response of potential new bioindicator plant species; 2)

19   multi-year field surveys in several National Wildlife Refuges documenting the presence of foliar

20   injury in valued areas; 3) ongoing data collection and assessment by the USDA Forest Service

21   FHM/FIA program, including multi-year trend analysis (U.S. EPA 2013, section 9.4.2). These

22   recent studies, in combination with the entire body of available evidence, thus form the basis for

23   the ISA determinations of a causal relationship between ambient O$_3$ exposure and the occurrence

24   of O$_3$-induced visible foliar injury on sensitive vegetation across the U.S. (U.S. EPA 2013, p. 9-

25   42).

26   With regard to evidence from controlled exposure studies, a recent study of 28 plant

27   species confirmed prior findings of O$_3$ causing predictable diagnostic visible foliar injury

28   symptoms on some species of plants. This study selected 28 plant species, most of which grow

29   naturally throughout the northeast and midwest US, including in national parks and wilderness

30   areas, that were suspected of being O$_3$ sensitive, and exposed them to four different O$_3$

31   concentrations (30, 60, 90, and 120 ppb) in continuously stirred tank reactor (CSTR) chambers

32   (Kline et al., 2008). Two experiments were conducted in each year of the study (2003 and 2004).

33   Plants were exposed to O$_3$ for 7 hours a day, five days a week over the course of each

34   experiment. Specifically, in 2003, the first experiment lasted from July 14 to August 21and

35   included 29 days of O$_3$ exposure and the second from September 9 to 30 and included 16

36   exposure days. In 2004, the first experiment was conducted from July 13 to August 10 with 21

1   O$_3$ exposure days and the second from August 27 to September 24, including 21 days of O$_3$

2   exposure. Though the exposures were cumulative over the course of the study, exposures were

3   reported only in terms of the target exposure concentration for each experiment. The study

4   reported O$_3$-induced responses in 12, 20, 28 and 28 of the 28 tested species at the 30, 60, 90 and

5   120 ppb exposure concentrations[14], respectively. Based on their findings, the authors suggest

6   that American sycamore, aromatic sumac, bee-balm, buttonbush, common milkweed, European

7   dwarf elderberry, New England aster, snowberry and swamp milkweed would make the most

8   useful bioindicator species. Some of these species are native to U.S. locations designated of

9   national significance (discussed further below). The staff additionally concludes that given that

10   the exposure protocol was designed to create a continuous exposure level, not a fluctuating one,

11   this study shows that O$_3$-induced foliar injury can occur from 7-hour exposures repeated over

12   multiple days at O$_3$ concentrations that are below the 75 ppb level of the current standard.[15]

13   While this type of controlled study provides clear evidence of cause and effect, it also has

14   limitations. The authors, recognizing this cautioned that "extrapolation of these CSTR results to

15   the field must be done carefully, since CSTR/greenhouse conditions … are not representative of

16   natural environmental conditions" (Kline et al., 2008).

17   A string of recently published multi-year field studies provide a complimentary line of

18   field-based evidence by documenting the incidence of visible foliar injury symptoms on a variety

19   of O$_3$-sensitive species over multiple years and across a range of cumulative, seasonal exposure

20   values in several eastern and midwestern Class I national wildlife refuges (NWRs) (U.S. EPA

21   2013, section 9.4.2.1; Davis and Orendovinci 2006; Davis 2007a, b; Davis 2009). Some of these

22   studies also included information regarding soil moisture stress using the Palmer Drought

23   Severity Index (PDSI). While environmental conditions and species varied across the four

24   NWRs, visible foliar injury was documented to a greater or lesser degree at each site. As

25   discussed further below, visible foliar injury incidence in these types of areas has greater

26   significance to the public welfare.

27   • **To what extent have important uncertainties identified in the last review been**
28   **reduced and/or new uncertainties emerged?**

29   The studies mentioned above also provide additional information regarding an important

30   uncertainty indentified in the previous review, i.e., the role of soil moisture in influencing visible

---

[14] Two of the target exposure levels, 30 and 60 ppb, fall below the level of the current standard (75 ppb). The mean exposure concentrations achieved in the CTSRs for the 30 ppb target level for each year and study were 27.9, 26.3, 27.1, and 29.3 ppb and for the 60 ppb target level were 56.6, 55.8, 57.9, and 59.0 ppb, for 2003 study 1, 2003 study 2, 2004 study 1, and 2004 study 2, respectively.

[15] The current standard is met when the 3-year average of the 4th highest daily maximum 8-hour average concentrations is at or below 75 ppb.

1   foliar injury response (U.S. EPA 2013, section 9.4.2). These studies confirm that adequate soil
2   moisture creates an environment conducive to greater visible foliar injury in the presence of O_3
3   than drier conditions. As stated in the ISA, "[a] major modifying factor for O_3-induced visible
4   foliar injury is the amount of soil moisture available to a plant during the year that the visible
5   foliar injury is being assessed … because lack of soil moisture generally decreases stomatal
6   conductance of plants and, therefore, limits the amount of O_3 entering the leaf that can cause
7   injury" (U.S. EPA, 2013, p. 9-39). As a result, "many studies have shown that dry periods in
8   local areas tend to decrease the incidence and severity of O_3-induced visible foliar injury;
9   therefore, the incidence of visible foliar injury is not always higher in years and areas with higher
10 O_3, especially with co-occurring drought (Smith, 2012; Smith et al., 2003)" (U.S. EPA, 2013, p.
11 9-39). This "…partial 'protection' against the effects of O_3 afforded by drought has been
12 observed in field experiments (Low et al., 2006) and modeled in computer simulations
13 (Broadmeadow and Jackson, 2000)" (U.S. EPA, 2013, p. 9-87). In considering the extent of any
14 protective role of drought conditions, however, the ISA also notes that other studies have shown
15 that "drought may exacerbate the effects of O_3 on plants (Pollastrini et al., 2010; Grulke et al.,
16 2003)" and that "[t]here is also some evidence that O_3 can predispose plants to drought stress
17 (Maier-Maercker, 1998)". Accordingly, the ISA concludes that "the nature of the response is
18 largely species-specific and will depend to some extent upon the sequence in which the stressors
19 occur" (U.S. EPA, 2013, p. 9-87). Such uncertainties associated with describing the potential for
20 foliar injury and its severity or extent of occurrence for any given air quality scenario due to
21 confounding by soil moisture levels make it difficult to identify an appropriate degree of annual
22 protection (as well as ambient O_3 exposure conditions that might be expected to provide that
23 protection).

24   • **To what extent does the available evidence indicate the occurrence of O_3-related**
25   **visible foliar injury attributable to cumulative exposures at lower ambient O_3**
26   **concentrations than previously established or to exposures at or below the level of**
27   **the current standard?**

28   Recently available evidence confirms that available in previous reviews that visible foliar
29 injury can occur when sensitive plants are exposed to elevated O_3 concentrations in a
30 predisposing environment (i.e. adequate soil moisture (U.S. EPA, 2013, section 9.4.2). Recent
31 evidence also continues to indicate the occurrence of visible foliar injury at cumulative ambient
32 O_3 exposures previously established. Since the 2006 O_3 AQCD, results from several multi-year
33 field surveys and experimental screenings of O_3-induced visible foliar injury on vegetation also
34 show that visible foliar injury can occur under conditions where the annual 8-hour average O_3
35 concentrations are at or below the level of the current standard, as discussed here. Limited

1   information exists regarding the incidence of visible foliar injury occurring in areas that have

2   design values that meet the current 3-year average 8-hour standard.

3   To facilitate comparison with other studies reporting foliar injury response to W126

4   cumulative exposures, we obtained air quality data from EPA's AQS database for monitors in

5   each study location and calculated the 12-hr W126 values and obtained the maximum 4th high 8-

6   hour average values for a subset of the most recent years included in each study (Table 5-6). As

7   the shaded rows in Table 5-6 below show, in the years 2002/2003 and 2004 in the Cape Romain

8   National Wildlife Refuge in SC, and the Seney National Wildlife Refuge in Michigan,

9   respectively, the 4th highest daily maximum 8-hour average O_3 concentrations were at or below

10   the level of the current standard. We additionally note that the Cape Romain site met the current

11   standard of 75 in every 3-year period during the study and has consistently met the standard from

12   2001 to 2012.[16] Under these air quality conditions, three species exhibited O_3-induced stipple

13   (winged sumac, Chinese tallow tree, and wild grape). In 2002, 32% of the examined wild grape

14   plants, 20% of the winged sumac plants, and 4.6% of the Chinese tallow tree plants, respectively,

15   were symptomatic (Davis 2009). At the same time, the 12 hour W126 value was 20 ppm-hrs. In

16   2003, when air quality was somewhat improved, foliar injury declined, with only 13.3% of wild

17   grape showing ozone stipple at a maximum 4th high 8-hr of 74 ppb and a W126 of 11 ppm-hr.

18   The PSDI values were 0.27 and 2.45 in 2002 and 2003, respectively. These values show that

19   2003 was a wetter year than 2002, though 2002 would have been considered within the normal

20   soil moisture range.

21   At the Seney NWR site, by comparison, the annual W126 level was similar in 2004 to

22   that at Cape Romain in 2003, and the annual 8-hour average level was below that of the current

23   standard, though the 3-year average design values were above that of the current standard for that

24   year. Not surprisingly, given the lower O_3 air quality in 2004, the Seney study reported injury

25   ranging from about 2% on common milkweed to about 6% on spreading dogbane. Though this

26   study does not provide the PDSI values, the authors provided some discussion of a possible

27   relationship stating that "the incidence of ozone injury on spreading dogbane, but not other

28   species, was weakly, but not significantly, related to the drought index (PDSI)….However this

29   relationship was too weak to be used for predictive purposes." The authors then conclude that

30   "[n]evertheless, the threshold SUM06 ozone level needed to induce stipple on sensitive plants

31   within the Seney refuge is likely 5000 ppb-hrs under the environmental conditions of these

32   surveys." On the basis of the above, the staff concludes that these studies confirm that visible

33   foliar injury has been shown to occur in the field at W126 index values ranging down to 10 ppm-

[16]Design values (concentrations in the form of the standard) for this monitoring site during this period are presented in the file available at: http://www.epa.gov/airtrends/values.html

1   hrs and provide limited evidence that such foliar injury can occur in areas with special public

2   welfare significance during periods that meet the current standard.

3   **Table 5-6.   Visible foliar injury incidence in four National Wildlife Refuges.**

Name/ Site #/ Ref.[A]	Year[B]	4th highest daily maximum 8-hr average	12 hr. W126	% Plants with visible injury
Cape Romain NWR, SC/ 450190046 (Davis, 2009)	2002	0.075 ppm	20 ppm-hr	5 - 32
	2003	0.074 ppm	11 ppm-hr	3-13
Moosehorn NWR, ME/ 230090102 (Davis, 2007a)	2002	0.1 ppm	24 ppm-hr	0 - 17
	2003	0.083 ppm	22 ppm-hr	0 - 13
	2004	0.082 ppm	14 ppm-hr	3 - 10
Seney NWR, MI/ 261530001 (Davis, 2007b)	2002	0.083 ppm	11 ppm-hr	0 - 13
	2003	0.076 ppm	15 ppm-hr	1 - 6
	2004	0.074 ppm	10 ppm-hr	2 - 6
NWR, NJ/ 340010005/ (Davis and Orendovici, 2006)	2001	0.095 ppm	39 ppm-hr	0 – 45
	2002	0.092 ppm	53 ppm-hr	0 – 4
	2003	0.085 ppm	36 ppm-hr	0 - 4

[A]Studies (cited above) reported exposures in terms of SUM06 form. EPA staff, using AQS data for the same monitors, calculated exposures in terms of the current 8-hr and W126 forms : http://www.epa.gov/ttn/airs/airsaqs/
[B]Only recent years with available W126 data were included in Table.

4

5   By far the most extensive field-based dataset of visible foliar injury incidence is that

6   obtained by of the USDA Forest Service FHM/FIA biomonitoring network program. A trend

7   analysis of data from the sites located in the Northeast and North Central U.S. for the 16 year

8   period (1994-2009) (Smith, 2012) provides additional evidence of foliar injury occurrence in the

9   field as well as some insight into the influence of changes in air quality and soil moisture on

10   visible foliar injury and the difficulty inherent in predicting foliar injury response under different

11   air quality/soil moisture scenarios (Smith, 2012; U.S. EPA 2013, section 9.2.4.1). In this study

12   ambient exposures were expressed in terms of the SUM06 cumulative index coupled with a

13   measure of the number of peak hourly concentrations above 100 ppb (N100). Soil moisture

14   conditions were generated using both the Palmer Drought Severity Index (PSDI) and the plant

15   moisture availability index (MI). Foliar injury was expressed in terms of the biosite index (BI)[17].

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[17] Biosite index (BI) is the average score (proportion of leaves with injury "amount" x mean severity of symptoms on injured leaves "severity") for each species averaged across all species on the biosite multiplied by 1,000.

1   The authors observed that over this 16-year period, "injury indices have fluctuated annually in
2   response to seasonal ozone concentrations and site moisture conditions. Sites with and without
3   injury occur at all ozone exposures but when ambient concentrations are relatively low, the
4   percentage of uninjured sites is much greater than the percentage of injured sites; and regardless
5   of ozone exposure, when drought conditions prevail, the percentage of uninjured sites is much
6   greater than the percentage of injured sites" (Smith, 2012). The authors further note that while
7   "both site moisture and ozone exposure play a role in foliar injury expression … the interplay
8   among these three factors is unique for each year and possibly each site. Extreme moisture
9   deficits decrease foliar injury, … [and] … [i]n no year do high ozone exposures override the
10   controlling effect of site moisture, although at the other end of the scale, injury severity is
11   minimized under conditions of low ozone exposure regardless of site moisture conditions. This
12   implies a necessary threshold of ozone exposure for injury to occur…." "In a similar analysis,
13   Rose and Coulston (2009) reported a high percentage of biosites with injury across the Southern
14   region in 2003, a year when SUM06 values >10 ppm-h were widespread at the same time that
15   the land area was in moisture surplus or balance." Thus, Rose and Coulston (2009) also "found
16   evidence that it is the co-occurrence of sufficient moisture and elevated ozone that determine
17   whether injury occurs to bioindicator plants, not ozone exposure alone." Regarding the role of
18   peak ozone concentrations (>100 ppb O_3), Smith (2012) reported that over the 16-year period
19   concentrations above 100 ppb have declined, and that this "... may account for the observed
20   decrease in the severity of ozone-induced foliar injury to ozone sensitive bioindicator plants in
21   eastern forests." They also note that "[t]here is no compelling evidence, however, that moderate
22   ozone concentrations, as reflected in seasonal mean SUM06 data, are on the decline…." "This
23   may explain why injury continues to be detected on many of the same sites every year…" The
24   authors thus conclude that, "[a]lthough it is reasonable to remain concerned about long-term
25   impacts of ozone pollution on our forest ecosystems, the findings of this biomonitoring survey
26   point to a declining risk of probable impact on eastern forests over the 16-year period from 1994
27   to 2009."
28   In a similar assessment of the USDA Forest Service FHM/FIA data in the West, six years
29   (2000 to 2005) of biomonitoring data for O_3 injury were evaluated for the three coastal states of
30   California, Oregon and Washington (Campbell et al., 2007; U.S. EPA 2013, section 9.4.2.1).
31   Campbell et al., 2007 found that "…ozone injury occurs frequently (25 to 37 percent of sampled
32   biosites) in California forested ecosystems demonstrating that ozone is present at phytotoxic
33   levels." This study concluded that, "in California, an estimated 1.3 million acres of forest land
34   and 596 million cubic feet of wood are at moderate to high risk to impacts from ozone.
35   However, [m]ore years of data are needed to discern any trends." Though this study does not
36   discuss the role of soil moisture in describing the results, the criteria used to select the

1　biomonitoring sites include one that considers soil conditions. The best sites are identified as

2　those with low drought potential and good fertility.   Thus, given the relatively high O_3

3　concentrations that occur in California and the likelihood that many of the biomonitoring sites

4　occur in areas that have sufficient soil moisture, the high percentage of sampled biosites with

5　foliar injury is not unexpected.[18]

6　　　These recent studies continue to provide evidence of O_3-induced foliar injury occurring in

7　many areas across the U.S. and augment our understanding of O_3-related visible foliar injury and

8　of factors that influence associations between O_3 exposures or concentrations and visible foliar

9　injury such as soil moisture.

10　　• **To what extent does currently available evidence suggest locations where the**
11　　**vulnerability of sensitive species, ecosystems and/or their associated services to**
12　　**O_3-related visible foliar injury would have special significance to the public**
13　　**welfare?**

14　　　As mentioned above, federally designated Class I areas are afforded stringent protections

15　under the 1977 amendments to the Clean Air Act (Act). The Act gives federal land managers of

16　Class I areas "the responsibility to protect all air quality related values (AQRVs)…from

17　deterioration…. In order to determine if deterioration is occurring, baseline AQRVs must be

18　established" (Davis, 2009). Because of this need and the significance of these areas, studies

19　often focus on these sites. For example, a study by Kohut (2007) was undertaken to assess the

20　risks of O_3-induced visible foliar injury on O_3 bioindicators (i.e., O_3-sensitive vegetation) in 244

21　parks managed by the NPS. Kohut (2007) estimated O_3 exposure using hourly O_3 monitoring

22　data collected at 35 parks from 1995 to 1999, estimated O_3 exposure at 209 additional parks

23　using kriging, a spatial interpolation technique, and qualitatively assessed risk. Kohut (2007)

24　applied a subjective evaluation based on three criteria: (1) the frequency of exceedance of foliar

25　injury thresholds[19,20] using several O_3 exposure metrics (i.e., SUM06, W126 and N100), (2) the

26　extent that low soil moisture constrains O_3 uptake during periods of high exposure, and (3) the

27　presence of O_3 sensitive species within each park. Based on these criteria, Kohut (2007)

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[18] Staff additionally notes that a large proportion of O_3 monitoring sites in California did not meet the current standard during the study period (see: http://www.epa.gov/airtrends/values.html).

[19] Kohut (2007) uses the term "foliar injury thresholds". In the WREA foliar injury analyses, we use the term "benchmarks" or "benchmark criteria" to avoid implying that foliar injury could not occur below these levels.

[20] Consistent with advice from CASAC (Frey and Samet, 2012a), the WREA modified the approach used by Kohut (2007) to apply the W126 metric. The WREA analysis, described in section 5.2.2 below developed and considered these different W126 benchmarks for foliar injury after further investigation into the benchmarks applied in Kohut (2007), which were derived from biomass loss rather than visible foliar injury. Kohut cited a threshold of 5.9 ppm-hrs for highly sensitive species from Lefohn (1997), which was based on the lowest W126 estimate corresponding to a 10% growth loss for black cherry. For soil moisture, Kohut (2007) qualitatively assessed whether there appeared to be an inverse relationship between soil moisture and high O_3 exposure.

1   concluded that the risk of visible foliar injury was high in 65 parks (27 percent), moderate in 46

2   parks (19 percent), and low in 131 parks (54 percent). Thus, while this study suggests that there

3   may be a reason for concern in as much as 46% of the parks, there were a number of important

4   limitations associated with this study (described in footnotes 8 and 9 below) that weakened this

5   conclusion. Given the importance of this kind of assessment, the WREA used Kohut (2007) as

6   the conceptual basis for the subsequent WREA national-scale and screening-level analyses,

7   though numerous modifications were made to the approach to make it applicable to the context

8   of this O_3 NAAQS review (see section 5.4.2 below).

9   In addition, as described above, several recently published studies (U.S. EPA 2013,

10   section 9.4.2.1; Davis and Orendovici 2006; Davis 2007a, b; Davis 2009, Kohut 2007) were

11   conducted in federally protected areas including federally designated Class I areas and national

12   parks. These studies confirm that visible foliar injury has been observed in these areas under

13   annual air quality conditions with ambient concentrations at or below the level of the current

14   standard and at W126 index values within the CASAC range recommended in past reviews.

15   This evidence continues to suggest that O_3-sensitive species and their associated ecosystems and

16   services continue to remain vulnerable to visible foliar injury incidence in areas that have been

17   afforded special protection by Congress and that have special significance to the public welfare.

18   **5.4.2   Exposure-/Risk-based Considerations**

19   The WREA presents a number of analyses considering air quality conditions associated

20   with increased prevalence of visible foliar injury and potential associated welfare impacts (see

21   Table 5-7 below, U.S. EPA, 2014, Chapter 7). An initial analysis included the development of

22   benchmark criteria reflecting different prevalences of visible foliar injury or of the occurrence of

23   elevated injury in conjunction with different W126 exposures and in some cases, soil moisture

24   conditions. These criteria were then used in a national scale screening level assessment to

25   characterize potential risk of foliar injury incidence under 2006-2010 conditions in 214 national

26   parks. The last analysis was a case study assessment on three national parks, which also

27   provides limited characterization of the associated ecosystem services. Despite the limitations

28   and uncertainties associated with these analyses, and recognizing that the air quality conditions

29   in most cases (prior to any model adjustment) did not meet the current standard, staff believes

30   that they help inform our understanding of the relationship between soil moisture and foliar

31   injury incidence, as well as provide limited support for our conclusion of a risk of visible foliar

32   injury incidence under air quality conditions likely to meet the current standard in areas of

33   special significance to the public welfare.

Table 5-7. Exposure, risk and ecosystem services analyses related to visible foliar injury.

	Ecosystem Level Effects	Ecosystem Services
WREA estimates	Proportion of USFS biosites with foliar injury incidence, degree of injury, and soil moisture at various W126-based benchmarks Percent of 214 national parks exceeding criteria based on USFS biosite dataset analysis during years considered	Case study of 3 national parks characterized impacts using available visitor and use data, including monetary data for activities and visitor expenditures: • Utilized Willingness-to-Pay studies for scenic impairment; • Assessed the overall cover of sensitive species; • Compared sensitive species cover to trails and overlooks; and, • Estimated percent of park area with O_3 concentrations above different W126 index values averaged over three consecutive years.
B The screening-level assessment of 214 national parks additionally included observations based on the model-based adjustments to just meet the current standard and targets for the three W126 scenarios (discussed below) but did not conduct a full analysis using these data.		

2

3   • **For what air quality scenarios were exposures and risks estimated? What**
4   **approaches were used to estimate W126 exposures for those conditions? What**
5   **are associated limitations and uncertainties?**

6   Three types of foliar injury analyses were performed in the WREA and are considered

7   below. They include an analysis using U.S. Forest Service biosite data, a National Park

8   screening-level assessment and a National Park case study (focused on three parks). The

9   analysis of U.S. Forest Service (USFS) biosite data was done using O_3 concentrations estimated

10   for a national-scale surface of concentrations (at a 12 km grid cell resolution in contiguous U.S.)

11   using interpolation methodology applied to concentrations at O_3 monitor locations (U.S. EPA,

12   2014, section 4.3.2, Appendix 4A). The analysis of USFS data used surfaces for each year from

13   2006 through 2010 (U.S. EPA, 2014, Appendix 4A, section 4.2). In both the National Park

14   screening-level assessment and the case study analysis, observations related to air quality were

15   made for five air quality scenarios by the methodology summarized in Table 5-4 above.[21]

16   The W126 index values in the individual years from 2006 to 2010 ranged from less than

17   5 ppm-hrs to 25 ppm-hrs. Concentration estimates varied appreciably across the five years with

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[21] In general, this methodology involved two steps. The first is derivation of the average W126 value (across the three years) at each monitor location. This value is based on unadjusted data for recent conditions and model-adjusted concentrations for the 4 other scenarios. The development of model adjusted concentrations was done for each of 9 regions independently (see U.S. EPA, 2014, section 4.3.4.1). In the second step, national-scale spatial surfaces (W126 values for each model grid cell) were created using the monitor-location values and the Voronoi Neighbor Averaging (VNA) spatial interpolation technique (details on the VNA technique are presented in U.S. EPA, 2014 Appendix 4A).

1   the median index values across grid cells ranging from a low of 5.5 ppm-hrs in 2009 to 11 ppm-
2   hrs in 2006 (U.S. EPA, 2014, Appendix 4A, section 4.2). During the recent conditions period
3   (2006 through 2008), the average W126 index values (across the three-year recent conditions
4   period) at the monitor locations ranged from below 10 ppm-hrs to 48.6 ppm-hrs (U.S. EPA 2014,
5   Figure 4-4 and Table 4-1). After model-adjustment of the 2006-2008 data to just meet the
6   current standard in each region, and subsequent application of the VNA technique to the current
7   standard scenario monitor location values, the average W126 values were below 15 ppm-hrs
8   across the national surface with the exception of a very small area of the southwest region (near
9   Phoenix) where the average W126 values was near or just above 15 ppm-hrs. A lowering of the
10   highest values occurred with application of the interpolation method as a result of estimating
11   W126 values at a 12x12 km^2 grid resolution rather that at the exact location of a monitor. This
12   indicates one uncertainty associated with this aspect of the approach to estimating W126 values
13   for the model-adjusted air quality just meeting the current standard. Other uncertainties are
14   summarized in section 5.2.2 above.

15   • **What are the nature and magnitude of the cumulative exposure- and risk-related**
16   **estimates for visible foliar injury under recent conditions or conditions meeting**
17   **the current O$_3$ standard?**

18   As an initial matter, we consider the analysis of the biomonitoring site data from the
19   USDA Forest Service FHM/FIA Network, described in section 7.2 of the WREA.[22] Using this
20   dataset and associated data for soil moisture conditions during the sample years along with
21   ambient air O$_3$ concentrations based on monitoring data from 2006 to 2010 and spatial
22   interpolation methodology (as described above), the proportion of sites with any or elevated
23   foliar injury are observed to increase with increasing annual W126 index values up to specific
24   values after which there is little change in proportion of affected sites with higher W126 values
25   (see Figure 5-5 below; U.S. EPA, 2014, section 7.2). The proportion of sites metric is derived by
26   first ordering the data (across sites and sample years) by W126 index value estimated for that site
27   and year. Then for each W126 index value the proportion of sites exceeding the selected biosite
28   index value for all observations at or below that W126 index value is calculated. The WREA
29   repeated this using a biosite index value greater than zero, indicating presence of any foliar
30   injury, and an index value >5, corresponding to a USFS cutoff for "low" or more injury (USFS,
31   2011).
32   When looking only at presence or absence of foliar injury ("any injury") with the
33   exception of 2008, the proportion of sites across all W126 index values with foliar injury exceeds

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[22] Data were not available for several western states (Montana, Idaho, Wyoming, Nevada, Utah, Colorado, Arizona, New Mexico, Oklahoma, and portions of Texas).

1   15 percent; in 2006, it exceeds 20 percent, while in 2008 the proportion of sites with foliar injury

2   across all W126 index values was just below 15 percent (U.S.EPA, 2014, section 7.2.3, Figure 7-

3   8). When categorized by moisture levels, the data demonstrate a distinct pattern. In general, the

4   WREA concludes that the results of these foliar injury analyses demonstrate a similar pattern –

5   the proportion of sites showing the presence of any foliar injury (biosite>0) or at least little foliar

6   injury (biosite>5) increases from zero to about 20% and 6% respectively (Figure 5-5 below).

7   This increase occurs with increasing W126 index values up to approximately10 ppm-hrs for any

8   foliar injury (biosite index >0), with little change in proportion of sites with any injury at higher

9   W126 values. The data for sites during normal moisture years are very similar to the dataset as a

10   whole, with an overall proportion of close to 18 percent for presence of any foliar injury, and

11   close to 6 percent for sites exceeding a biosite index of 5. Among the sites with relatively wet

12   season (average Palmer Z => 1), the highest proportion of sites observed is much higher for both

13   index categories of injury and the relationship with annual W126 index value is much steeper.

14   Much lower proportions of sites are reached for both injury categories at sites with relatively dry

15   seasons (average Palmer Z < -1.24), potentially indicating that drought may provide some

16   protection from foliar injury as discussed in the ISA (U.S. EPA, 2014, section 7.2.3, Figures 7-10

17   and 7-11). This information provides insight into the relationship between soil moisture and

18   foliar injury and the issue of whether drought provides protection from foliar injury. For both

19   categories of injury, there is relatively little change in the proportion of sites beyond a W126 of

20   20 ppm-hrs. There are two important observations that can be made from these analyses: (1) the

21   proportion of sites exhibiting foliar injury rises rapidly at increasing W126 index values below

22   10 ppm-hrs, and (2) there is relatively little change in the proportions above W126 index values

23   of 20 ppm-hrs.

Biosite Index > 0

1

Biosite Index > 5
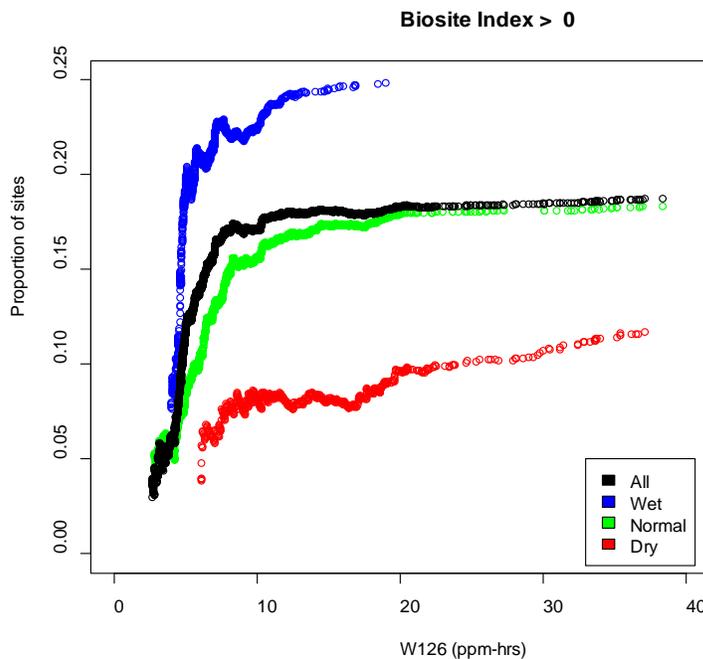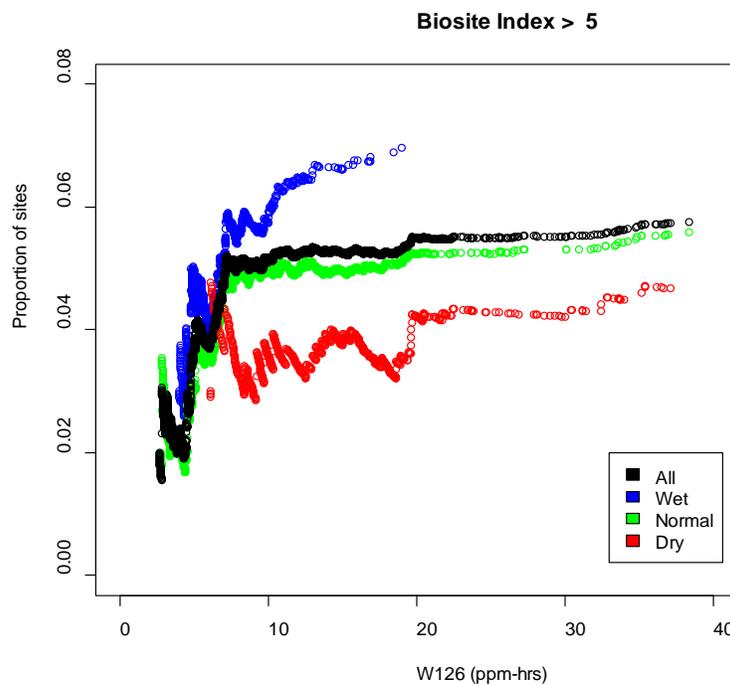
2

3 **Figure 5-5.   Cumulative proportion of sites with a) any foliar injury or b) elevated injury**
4 **present, by moisture category (U.S. EPA 2014, Figures 7-10 and 7-11).**

5   We additionally consider the WREA screening-level assessment in 214 parks in the
6 contiguous U.S. that employed benchmark criteria developed from the above analysis (Table 5-

1   8).[23],[24] For example, annual O_3 concentrations corresponding to a W126 seasonal index value of

2   10.46 ppm-hrs represents the O_3 exposure concentration above which a consistent percentage of

3   all biosites (17.7 percent) showed any injury, without consideration of soil moisture. The W126

4   benchmarks across the six scenarios range from 3.05 ppm-hrs (five percent of biosites, normal

5   moisture, any injury) up to 46.87 ppm-hrs (five percent of biosites, dry, elevated injury). For the

6   scenario of 10% biosites with injury, W126 values were approximately 4, 6, and 25 ppm-hours

7   for wet, normal and dry years, respectively. The national-scale screening-level assessment

8   includes 42 parks with O_3 monitors and 214 parks with O_3 exposure estimated from the

9   interpolated O_3 surface for individual years from 2006 to 2010 (U.S. EPA, 2014, Appendix 7A).

10   These data were combined with lists from the NPS of the parks containing O_3 sensitive

11   vegetation species (NPS, 2003, 2006). Based on NPS lists, 95 percent of the parks in this

12   assessment contain at least one O_3-sensitive species. This analysis for recent air quality

13   conditions, estimates that 58 percent of parks exceeded the benchmark criteria corresponding to

14   the base scenario (W126>10.46 ppm-hrs, 17.7 percent of biosites, all moisture categories, any

15   injury) for at least three years in the period from 2006 to 2010, and 34 percent of parks would

16   exceed the benchmark criteria for the elevated injury scenario (five percent of biosites, multiple

17   moisture categories, elevated foliar injury) for at least three years (U.S. EPA, 2014, section

18   7.3.2).[25] Based on model-adjustments to meet the current standard, none of the 214 parks have

19   average W126 index values that would exceed the annual benchmark criteria for the base

20   scenario (W126 >10.46 ppm-hrs).

21

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[23] The parks assessed here include lands managed by the NPS in the continental U.S., which includes National Parks, Monuments, Seashores, Scenic Rivers, Historic Parks, Battlefields, Reservations, Recreation Areas, Memorials, Parkways, Military Parks, Preserves, and Scenic Trails.

[24] The WREA applied different foliar injury benchmarks in this assessment after further investigation into the benchmarks applied in Kohut (2007), which were derived from biomass loss rather than visible foliar injury. Kohut cited a threshold of 5.9 ppm-hrs for highly sensitive species from Lefohn (1997), which was based on the lowest W126 estimate corresponding to a 10% growth loss for black cherry. For soil moisture, Kohut (2007) qualitatively assessed whether there appeared to be an inverse relationship between soil moisture and high O_3 exposure.

[25] The lack of national surfaces of O_3 concentrations for each of three years in each scenario (as described earlier and summarized in Table 5-3) precluded derivation of similar estimates (of parks with W126 index values above any of the benchmark criteriaafter model-adjustment of air quality to just meet the current standard, although we note that the W126 averaged across three years in all 214 parks was below 10.46 ppm-hrs (the individual year benchmark criteria for the base scenario) (U.S. EPA, 2014, section 7.3.2).

Table 5-8. Benchmark criteria for O$_3$ exposure and relative soil moisture used in screening-level assessment of parks (from U.S. EPA 2014, Table 7-5).

Description		Normal (-1.25<Palmer Z<1)	Wet (Palmer Z>1)	Dry (Palmer Z<-1)
Any injury, without considering soil moisture conditions (Base Scenario)	17.7% of all biosites with any foliar injury (consistent proportion of all biosites showed injury in years with W126 index value above this value)	W126>10.46 (soil moisture not considered)		
Any injury, considering soil moisture conditions	5% of biosites showing any foliar injury, by relative moisture category	W126>3.05	W126>3.76	W126>6.16
	10% of biosites showing any foliar injury, by relative moisture category	W126>5.94	W126>4.42	W126>24.61
	15% of biosites showing any foliar injury, by relative moisture category	W126>8.18	W126>4.69	N/A
	20% of biosites showing any foliar injury, by relative moisture category	N/A	W126>5.65	N/A
5% of biosites, Injury ≥ 5	5% of biosites showing foliar injury equal or greater than 5 on the biosite injury index (e.g., 5% of leaf shows injury in 10% of the leaves), by relative moisture category	W126>12.23	W126>7.02	W126>46.87

Lastly, we consider the WREA case study analysis which focused on characterizing the ecosystem services potentially associated with visible foliar injury in three specific national parks (case study assessment). The parks included were Great Smoky Mountains National Park (GRSM), Rocky Mountain National Park (ROMO), and Sequoia/Kings National Parks (SEKI). For each park, the potential impact of O$_3$-related foliar injury on recreation (cultural services) was considered in light of information on visitation patterns, recreational activities and visitor expenditures. For example, visitor spending in 2011 exceeded $800 million, $170 million and $97 million dollars in GRSM, ROMO and SEKI, respectively. This assessment also included percent cover of species sensitive to foliar injury and focused on the overlap between recreation areas within the park and elevated W126 concentrations. Ozone concentrations in GRSM have been among the highest in the eastern U.S. In the recent conditions scenario, the grid cells representing 44 percent of GRSM had three year average W126 value above 15 ppm-hrs. After adjustments to just meet the current standard, no grid cell had a three-year average W126 value above 7 ppm-hrs. In the recent conditions scenario for ROMO, three-year average W126 values for all grid cells were above15 ppm-hrs. In the current standard scenario, values for 59 percent

1   of the park were below 7 ppm-hrs. For SEKI, three-year average W126 values for all grid cells
2   were above 15 ppm-hrs in the recent conditions scenario, but dropped below 7 ppm-hrs for the
3   current standard scenario (U.S. EPA, 2014, section 7.4).
4   In summary, these analyses indicate that O_3 concentrations in U.S. national parks in
5   recent years correspond to W126 index values at which some foliar injury may occur, with
6   variation associated with relative soil moisture conditions. None of the 214 parks assessed are
7   estimated to exceed the annual benchmark criteria for the base scenario (W126 >10.46 ppm-hrs)
8   after adjusting air quality to meet the current standard. Although model-adjusted scenarios to
9   just meet the current standard indicate substantial reductions in three-year average W126 index
10   values estimated by the VNA approach, some individual year values may range higher. The case
11   study analysis of three parks indicates the potential for appreciable ecosystem services impact
12   associated with foliar injury, although it might be expected that such impact would relate more to
13   severe and/or widespread foliar injury occurrences. While these analysis indicate the potential
14   for foliar injury to occur under conditions that meet the current standard, the extent of foliar
15   injury that might be expected under such conditions is unclear from these analyses.

16   • **To what extent are the exposures and risks remaining upon simulating just**
17   **meeting the current O_3 standard important from a public welfare perspective?**

18   The screening level assessment, as described above, indicates that risk of visible foliar
19   injury is likely to be lower in some national parks after simulating just meeting the current
20   standard. Based on the national-scale analysis, visible foliar injury would likely continue to
21   occur at lower O_3 exposures, including some sensitive species growing in areas (e.g., National
22   Parks and other Class I areas) that may provide important cultural ecosystem services to the
23   public. Staff notes that such occurrences might reasonably be considered to have some
24   importance from a public welfare perspective, as discussed in section 5.1 above.

25   • **What are the ecosystem services potentially affected by visible foliar injury, to**
26   **what degree can the magnitude of these effects be qualitatively or quantitatively**
27   **characterized, and to what extent are they important from a public welfare**
28   **perspective?**

29   The ecosystem services most likely to be affected by O_3-induced foliar injury are cultural
30   services, including aesthetic value and outdoor recreation. Aesthetic value and outdoor
31   recreation depend on the perceived scenic beauty of the environment. Many outdoor recreation
32   activities directly depend on the scenic value of the area, in particular scenic viewing, wildlife-
33   watching, hiking, and camping. These activities and services are of significant importance to
34   public welfare as they are enjoyed by millions of Americans every year and generate millions of
35   dollars in economic value (U.S. EPA, 2014, Chapter 5, Chapter 7). These aesthetic values are at

1 risk of impairment because of O_3-induced damage directly due to foliar injury. Other ecosystem

2 services that have also been found to be associated with O_3-sensitive plants include those that fall

3 under the categories of provisioning. For example, several tribes have indicated that many of the

4 known confirmed O_3-sensitive species (including bioindicator species) are culturally significant

5 (see Appendix 5A). Although data are not available to explicitly quantify these negative effects

6 on ecosystem services, several qualitative analyses conducted in the WREA are summarized

7 below.

8   To assess the effects of visible foliar injury on recreation, the WREA reviewed the

9 National Survey on Recreation and the Environment (NSRE), as well as the 2006 National

10 Survey of Fishing, Hunting, and Wildlife-Associated Recreation (FHWAR) and a 2006 analysis

11 done for the Outdoor Industry Foundation (OIF). According to the NSRE, some of the most

12 popular outdoor activities are walking, including day hiking and backpacking; camping; bird

13 watching; wildlife watching; and nature viewing. Participant satisfaction with these activities

14 can depend on the quality of the natural scenery, which can be adversely affected by O_3-related

15 visible foliar injury. According to the FHWAR and the OIF reports, the total expenditures across

16 wildlife watching activities, trail-based activities, and camp-based activities are approximately

17 $230 billion dollars annually. While the WREA could not quantify the magnitude of the impacts

18 of O_3 damage to the scenic beauty and outdoor recreation, the existing losses associated with

19 current O_3-related foliar injury are reflected in reduced outdoor recreation expenditures (U.S.

20 EPA, 2014, section 7.1).

21   The WREA also assessed O_3 impacts on cultural ecosystem services related to foliar

22 injury at three national parks – Great Smoky Mountains National Park, Rocky Mountain

23 National Park, and Sequoia/Kings National Parks - by considering information on visitation

24 patterns, recreational activities and visitor expenditures. The analysis included percent cover of

25 species sensitive to foliar injury and focused on the overlap between recreation areas within the

26 park and elevated W126 concentrations. All three of these park units are in areas that are known

27 to have high ambient O_3 concentrations, have vegetation maps, and have species that are

28 considered O_3 sensitive. Using GIS, the NPS vegetation maps were compared to the national O_3

29 surface to illustrate where foliar injury may be occurring, particularly with respect to park

30 amenities such as trails (U.S. EPA, 2014, section 7.4).

31   Great Smoky Mountains National Park is prized, in part, for its rich species diversity.

32 The large mix of species includes 37 O_3-sensitive species and many areas contain several

33 sensitive species. With 3.8 million hikers using the trails every year and those hikers willing to

34 pay (WTP) over $266 million for that activity, even a small benefit of reducing O_3 damage in the

35 park could result in a significant economic value. Ozone concentrations in Great Smoky

36 Mountains National Park have been among the highest in the eastern U.S. – at times twice as

1   high as neighboring cities such as Atlanta (U.S. EPA, 2014, p. 7-52).   Unlike Great Smoky

2   Mountains National Park, sensitive species cover in Rocky Mountain National Park is driven by

3   a few O_3-sensitive species (7 species) and most notably by Quaking Aspen. This is significant in

4   that many of the visitors to Rocky Mountain National Park visit specifically to see this tree in its

5   fall foliage. Given 1.5 million hikers in Rocky Mountain National Park and their $70 million

6   WTP for the hiking experience, even a small improvement in the scenic value could be

7   economically significant (U.S. EPA, 2014, section 7.4.2, p. 7-56). Sequoia/Kings National Parks

8   is home to 12 identified sensitive species. Again, although the EPA is not able to quantify the

9   impact of this scenic damage on hiker satisfaction for hikers in Sequoia/Kings National Parks

10 and their $26 million WTP for the experience, even a small improvement in the scenic value

11 could be economically significant ((U.S. EPA, 2014, section 7.4.3, p. 7-63).

12 • **What are the uncertainties associated with this information and what is the level**
13 **of confidence associated with those estimates?**

14   Uncertainties associated with these analyses are discussed in the WREA, sections 7.3.2

15 and 8.5.3, and in WREA Table 7.23. As discussed in the WREA (section 8.5.3), evaluating soil

16 moisture is more subjective than evaluating O_3 exposure because of its high spatial and temporal

17 variability within the O_3 season, and there is considerable subjectivity in the categorization of

18 relative drought. The WREA generally concludes that the spatial and temporal resolution for the

19 soil moisture data is likely to underestimate the potential of foliar injury that could occur in some

20 areas. In addition, there is lack of a clear threshold for drought below which visible foliar injury

21 would not occur. In general, low soil moisture reduces the potential for foliar injury, but injury

22 could still occur, and the degree of drought necessary to reduce potential injury is not clear. Due

23 to the absence of biosite injury data in the Southwest region and limited biosite data in the West

24 and West North Central regions, the benchmarks applied may not be applicable to these regions.

25   There are also important uncertainties in the estimated O_3 concentrations for the different

26 air quality scenarios evaluated (U.S. EPA, 2014, section 8.5), as discussed earlier in this section.

27 In general, this interpolation method under-predicts higher 12-hr W126 exposures. Due to the

28 important influence of higher exposures in determining risks to plants, the potential for the VNA

29 interpolation approach to under-predict higher W126 exposures could result in an under-

30 estimation of risks to vegetation in some areas.   The WREA applied the benchmarks from the

31 national-scale analysis to a screening-level analysis of 214 national parks and case studies of

32 three national parks. Therefore, uncertainties in the foliar injury benchmarks and in the air

33 quality analyses are propagated into the national park analyses. The uncertainties associated

34 with air quality assessments include those resulting from use of an unevenly distributed

35 monitoring network with fewer monitors in rural and western sites to drive a VNA interpolation

1   and the use of model-adjusted air quality to generate alternative air quality scenarios including

2   just meeting the current standard (these uncertainties are described in more detail above).

3   Additional uncertainties are associated with the national park analyses. Specifically,

4   survey estimates of participation rates, visitor spending/economic impacts, and willingness-to-

5   pay are inherently uncertain. These surveys potentially double-count impacts based on the

6   allocation of expenditures across activities but also potentially exclude other activities with

7   economic value. Each survey uses different survey methods, so it is not appropriate to generalize

8   across the surveys. In general, the national level surveys apply standard approaches, which

9   minimize potential bias. Other sources of uncertainty are associated with the mapping, including

10   park boundaries, vegetation species cover, and park amenities, such as scenic overlooks and

11   trails. In general, the WREA concludes that there is high confidence in the park mapping (U.S.

12   EPA, 2014, Table 7-23)..

13   **5.5   OTHER WELFARE EFFECTS**

14   In addition to the welfare effects discussed in the previous sections, there is evidence of

15   other O_3 effects, such as those related to climate impacts that we consider here. In this section,

16   the WREA national-scale analyses of the effects of insect damage to forests related to elevated

17   O_3 exposures are considered in section 5.5.1, and a case study-scale characterization of the effect

18   community composition changes on forest susceptibility and fire regulation in California is

19   considered in section 5.5.2. As above, these sections, where possible, consider the WREA

20   information regarding risk remaining under model adjusted conditions just meeting the current

21   standard and associated uncertainties (U.S. EPA 2014, section 8.5). Chapters 5, 6, and 7 of the

22   WREA also qualitatively assessed additional ecosystem services, including regulating services

23   such as hydrologic cycle and pollination; provisioning services such as commercial non-timber

24   forest products; and cultural services with aesthetic and non-use values. The information

25   associated with these latter effects is insufficient to inform the target protection of the standard.

26   The effects of O_3 on climate are also considered in section 5.5.3 below, drawing primarily on the

27   evidence presented in the ISA (U.S. EPA 2013, chapter 10).

28   **5.5.1   Forest Susceptibility to Insect Infestation**

29   Ozone in ambient air can contribute to increased susceptibility of some forests to

30   infestation by some chewing insects, including the southern pine beetle and western bark beetle

31   (U.S. EPA 2013, chapter 9; U.S. EPA 2014, sections 5.3.3 and 5.4). These infestations can cause

32   economically significant damage to tree stands and the associated timber production. The

33   WREA described the potential impacts of this effect on timber markets (U.S. EPA 2014, section

34   5.4). In the short-term, the immediate increase in timber supply that results from the additional

1   harvesting of damaged timber depresses prices for timber and benefits consumers. In the longer-
2   term, the decrease in timber available for harvest raises timber prices, potentially benefitting
3   producers. The United States Forest Service (USFS) reports timber producers have incurred
4   losses of about $1.4 billion (2010$), and wood-using firms have gained about $966 million, due
5   to beetle outbreaks between 1977 to 2004. It is not possible to attribute a portion of these
6   impacts resulting from the effect of O_3 on trees' susceptibility to insect attack; however, the
7   losses are embedded in the estimates cited.

8   To provide some quantitative estimates related to insect infestation-related risks, the
9   WREA reported the estimates of 3-year average W126 values in areas estimated to be at risk of
10 greater than 25% timber loss (high loss) due to pine beetle infestation. This was done for all six
11 WREA air quality scenarios. For example, for the recent conditions scenario, approximately 57
12 percent of the at-risk area has W126 estimates above 15 ppm-hrs, with the percentage dropping
13 to approximately five percent in the current standard scenario (U.S. EPA 2014, section 5.4).

14 **5.5.2   Fire Regulation**

15   Evidence indicates that fire regime regulation may also be negatively affected by O_3
16 exposure (U.S. EPA 2013, chapter 9; U.S. EPA 2014, section 5.3.3). For example, Grulke et al.
17 (2008) reported various lines of evidence indicating that O_3 exposure may contribute to southern
18 California forest susceptibility to wildfires by increasing leaf turnover rates and litter, increasing
19 fuel loads on the forest floor. According to the National Interagency Fire Center, in the U.S. in
20 2010 over 3 million acres burned in wildland fires and an additional 2 million acres were burned
21 in prescribed fires. From 2004 to 2008, Southern California alone experienced, on average, over
22 4,000 fires per year burning, on average, over 400,000 acres per fire. The California Department
23 of Forestry and Fire Protection (CAL FIRE) estimated that losses to homes due to wildfire were
24 over $250 million in 2007 (CAL FIRE, 2008). In 2008, CAL FIRE's costs for fire suppression
25 activities were nearly $300 million (CAL FIRE, 2008).

26   The WREA developed maps that overlay the mixed conifer forest area of California with
27 areas of moderate or high fire risk defined by CAL FIRE and with recent W126 concentrations
28 and surfaces adjusted to just meet existing and alternative standards. The highest fire risk and
29 highest O_3 concentrations overlap with each other, as well as with significant portions of mixed
30 conifer forest. In the recent concentrations scenario, over 97 percent of mixed conifer forest area
31 has average W126 values over 7 ppm-hrs with a moderate to severe fire risk, and 74 percent has
32 average W126 values over 15 ppm-hrs with a moderate to severe fire risk. The scenario for air
33 quality adjusted to just meet the current standard, almost all of the mixed conifer forest area with
34 a moderate to high fire risk shows a reduction in O_3 to below a W126 index value of 7 ppm-hrs
35 (average across three years of scenario). In the scenario for an average W126 index value of 15

1   ppm-hrs, all but 0.18 percent of the area has average index values below 7 ppm-hrs, and for the

2   W126 scenarios of 11 and 7 ppm-hrs, all of the moderate to high fire threat area has estimated

3   average W126 values below 7 ppm-hrs (U.S. EPA 2014, section 5.3.3, figure 5-3).

5.5.3   Ozone Effects on Climate

5   Tropospheric O_3 is a major greenhouse gas, third in importance after carbon dioxide

6   (CO_2) and methane (CH_4). While the developed world has successfully reduced emissions of O_3

7   precursors in recent decades, many developing countries have experienced large increases in

8   precursor emissions and these trends are expected to continue, at least in the near term (U.S.

9   EPA 2013, section 10.3.6.2). Projections of radiative forcing due to changing O_3 over the 21st

10   century show wide variation, due in large part to the uncertainty of future emissions of source

11   gases (U.S. EPA 2013, section 10.3.6.2). In the near-term (2000-2030), projections of O_3

12   radiative forcing range from near zero to +0.3 W/m^2, depending on the emissions scenario (U.S.

13   EPA 2013, section 10.3.6.2; Stevenson et al., 2006). Reduction of tropospheric O_3

14   concentrations could therefore provide an important means to slow climate change in addition to

15   the added benefit of improving surface air quality (U.S. EPA, 2013, section 10.5).

16   It is clear that increases in tropospheric O_3 lead to warming. However the precursors of

17   O_3 also have competing effects on the greenhouse gas CH_4, complicating emissions reduction

18   strategies. A decrease in CO or VOC emissions would enhance OH concentrations, shortening

19   the lifetime of CH_4, while a decrease in NO_X emissions could depress OH concentrations in

20   certain regions and lengthen the CH_4 lifetime (U.S. EPA, 2013, section 10.5).

21   Abatement of CH_4 emissions would likely provide the most straightforward means to

22   address O_3-related climate change since CH_4 is itself an important precursor of background O_3

23   (West et al., 2007; West et al., 2006; Fiore et al., 2002). A reduction of CH_4 emissions would

24   also improve air quality in its own right. A set of global abatement measures identified by West

25   and Fiore (2005) could reduce CH_4 emissions by 10% at a cost savings, decrease background O_3

26   by about 1 ppb in the Northern Hemisphere summer, and lead to a global net cooling of 0.12

27   W/m^2. West et al. (2007) explored further the benefits of CH_4 abatement, finding that a 20%

28   reduction in global CH_4 emissions would lead to greater cooling per unit reduction in surface O_3,

29   compared to 20% reductions in VOCs or CO (U.S. EPA, 2013, section 10.5).

30   Important uncertainties remain regarding the effect of tropospheric O_3 on future climate

31   change. To address these uncertainties, further research is needed to: (1) improve knowledge of

32   the natural atmosphere; (2) interpret observed trends of O_3 in the free troposphere and remote

33   regions; (3) improve understanding of the CH_4 budget, especially emissions from wetlands and

34   agricultural sources, (4) understand the relationship between regional O_3 radiative forcing and

1 regional climate change; and (5) determine the optimal mix of emissions reductions that would

2 act to limit future climate change (U.S. EPA, 2013, section 10.5).

3   The IPCC has estimated the effect of the tropospheric O_3 change since preindustrial times

4 on climate has been estimated to be about 25-40% of the anthropogenic CO_2 effect and about

5 75% of the anthropogenic CH_4 effect. There are large uncertainties in the radiative forcing

6 estimate attributed to tropospheric O_3, making the effect of tropospheric O_3 on climate more

7 uncertain than the effect of the long-lived greenhouse gases (U.S. EPA, 2013, section 10.5).

8   Radiative forcing does not take into account the climate feedbacks that could amplify or

9 dampen the actual surface temperature response. Quantifying the change in surface temperature

10 requires a complex climate simulation in which all important feedbacks and interactions are

11 accounted for. As these processes are not well understood or easily modeled, the surface

12 temperature response to a given radiative forcing is highly uncertain and can vary greatly among

13 models and from region to region within the same model (U.S. EPA, 2013, section 10.5).

14   **5.5.4 Additional Effects**

15   Recent information available since the last review considers the effects of O_3 on chemical

16 signaling in insect and wildlife interactions. Specifically, studies on O_3 effects on pollination and

17 seed dispersal, defenses against herbivory and predator-prey interactions all consider the ability

18 of O_3 to alter the chemical signature of VOCs emitted during these pheromone-mediated events.

19 The effects of O_3 on chemical signaling between plants, herbivores and pollinators as well as

20 interactions between multiple trophic levels is an emerging area of study that may result in

21 further elucidation of O_3 effects at the species, community and ecosystem-level (U.S. EPA, 2013,

22 p. 9-98).

23   **5.6 CASAC ADVICE**

24   Following the 2008 decision to revise the secondary standard by setting it identical to the

25 revised primary standard, CASAC conveyed additional advice to the Administrator regarding

26 that decision. Shortly after that, several petitioners filed suit challenging the decision and in

27 September 2009, the EPA announced its intention to reconsider the 2008 standards, issuing a

28 notice of proposed rulemaking in January 2010 (FR 75 2938). Soon after, the EPA solicited

29 CASAC review of that proposed rule and in January 2011 solicited additional advice. This

30 proposal was based on the scientific and technical record from the 2008 rulemaking, including

31 public comments and CASAC advice and recommendations. As further described in section

32 1.2.2 above, the EPA in the fall of 2011 did not promulgate final rulemaking in that process but

33 decided to coordinate further proceedings on the reconsideration rulemaking with this ongoing

1 periodic review. Accordingly, we are describing CASAC advice related to the 2008 final

2 decision and the subsequent reconsideration in this section, as well as advice on the NAAQS

3 review that was initiated in September 2008.

4   In April 2008, the members of the CASAC Ozone Review Panel sent a letter to EPA

5 stating that "[i]n our most-recent letters to you on this subject - dated October 2006 and March

6 2007 - … the Committee recommended an alternative secondary standard of cumulative form

7 that is substantially different from the primary Ozone NAAQS in averaging time, level and form

8 — specifically, the W126 index within the range of 7 to 15 ppm-hours, accumulated over at least

9 the 12 'daylight' hours and the three maximum ozone months of the summer growing season "

10 (Henderson, 2008). The letter continued:

11   *The CASAC now wishes to convey, by means of this letter, its additional,*
12   *unsolicited advice with regard to the primary and secondary Ozone NAAQS. In*
13   *doing so, the participating members of the CASAC Ozone Review Panel are*
14   *unanimous in strongly urging you or your successor as EPA Administrator to*
15   *ensure that these recommendations be considered during the next review cycle for*
16   *the Ozone NAAQS that will begin next year ... The CASAC was also greatly*
17   *disappointed that you failed to change the form of the secondary standard to*
18   *make it different from the primary standard. As stated in the preamble to the*
19   *Final Rule, even in the previous 1996 ozone review, "there was general*
20   *agreement between the EPA staff, CASAC, and the Administrator, ... that a*
21   *cumulative, seasonal form was more biologically relevant than the previous 1-*
22   *hour and new 8-hour average forms (61 FR 65716)" for the secondary*
23   *standard......Unfortunately, this scientifically-sound approach of using a*
24   *cumulative exposure index for welfare effects was not adopted...*

25   In response to the EPA's solicitation of their advice on the Agency's proposed

26 rulemaking as part of the reconsideration, CASAC conveyed their support as follows (Samet,

27 2010).

28   *CASAC also supports EPA's secondary ozone standard as proposed: a new*
29   *cumulative, seasonal standard expressed as an annual index of the sum of*
30   *weighted hourly concentrations (i.e., the W126 form), cumulated over 12 hours*
31   *per day (8am to 8pm) during the consecutive 3-month period within the ozone*
32   *season with the maximum index value, set as a level within the range of 7 to [1]5*
33   *ppm-hours. This W126 metric can be supported as an appropriate option for*
34   *relating ozone exposure to vegetation responses, such as visible foliar injury and*
35   *reductions in plant growth. We found the Agency's reasoning, as stated in the*
36   *Federal Register notice of January 19, 2010, to be supported by the extensive*
37   *scientific evidence considered in the last review cycle. In choosing the W126 form*
38   *for the secondary standard, the Agency acknowledges the distinction between the*
39   *effects of acute exposures to ozone on human health and the effects of chronic*
40   *ozone exposures on welfare, namely that vegetation effects are more dependent on*

1   *the cumulative exposure to, and uptake of, ozone over the course of the entire*
2   *growing season (defined to be a minimum of at least three months).*

3   In advice offered so far in the current review, which is considering an updated scientific
4   and technical record since the 2008 rulemaking, CASAC indicated that a conclusion that the
5   current standard is inadequate to protect vegetation and ecosystems is "warranted" although the
6   foundation needs to be broadened beyond analysis focused on Class I areas and trees to include
7   "effects on sensitive crops, trees in regions outside of Class I areas, and additional ecosystem
8   impacts" (Frey and Samet, 2012b, p. 2). The Panel additionally endorsed the first draft PA
9   discussions and conclusions on biologically relevant exposure metrics, stating that "the focus on
10   the W126 form is appropriate" and that "there is a strong justification made for using a
11   cumulative and weighted exposure standard for welfare effects (i.e. the W126)…" (Frey and
12   Samet, 2012b, p. 2).

13   **5.7   PRELIMINARY STAFF CONCLUSIONS ON ADEQUACY OF**
14   **   SECONDARY STANDARD**

15   This section presents preliminary staff conclusions for the Administrator to consider in
16   deciding whether the existing secondary O_3 standard is adequate and whether it should be
17   retained or revised. Our conclusions are based on consideration of the assessment and integrative
18   synthesis of information presented in the ISA, as well as our analyses of air quality distributions;
19   analyses in the second draft WREA; and the comments and advice of CASAC and public
20   comment on an earlier draft of this document and the ISA and WREA, as discussed above.
21   Taking into consideration the responses to specific questions discussed above, we revisit the
22   overarching policy question for this chapter:

23   • **Does the currently available scientific evidence and exposure/risk information, as**
24   **reflected in the ISA and REA, support or call into question the adequacy and/or**
25   **appropriateness of the protection afforded by the current secondary O_3 standard?**

26   There is a longstanding evidence base on the phytotoxic effects of O_3. Since first
27   identified by Richards, et al in 1958, numerous studies have been conducted on the effects of O_3
28   on plants and their associated ecosystems. Taken together, these studies demonstrate that O_3 -
29   induced effects that occur at the subcellular and cellular levels, at sufficient magnitudes
30   propagate up to produce larger scale effects that affect the whole organism. In particular in this
31   review, many of the recent studies have focused on and further increased our understanding of
32   the molecular, biochemical and physiological mechanisms that explain how plants are affected
33   by O_3, in the absence of other stressors, particularly in the area of genomics (U.S. EPA, 2013,
34   Chapter 9, section 9.3).

1   Based on its assessment of this extensive body of science, the ISA determined that a
2 causal relationship exists between exposure to O_3 in ambient air and visible foliar injury effects
3 on vegetation, reduced vegetation growth, reduced productivity in terrestrial ecosystems, reduced
4 yield and quality of agricultural crops and alteration of below-ground biogeochemical cycles
5 (U.S. EPA 2013, Table 1-2). Additionally, the ISA determined that a likely to be causal
6 relationship exists between exposures to O_3 in ambient air and reduced carbon sequestration in
7 terrestrial ecosystems, alteration of terrestrial ecosystem water cycling and alteration of
8 terrestrial community composition (U.S. EPA, 2013, Table 1-2).

9   Recent studies also continue to provide evidence that adverse vegetation effects are
10 attributable to cumulative seasonal O_3 exposures. On the basis of the entire body of evidence in
11 this regard, the ISA concludes that "quantifying exposure with indices that cumulate hourly O_3
12 concentrations and preferentially weight the higher concentrations improves the explanatory
13 power of exposure/response models for growth and yield, over using indices based on mean and
14 peak exposure values" (U.S. EPA, 2013, p. 2-44). Thus, as in other recent reviews, the evidence
15 continues to provide a strong basis for concluding that it is appropriate to judge impacts of O_3 on
16 vegetation, related effects and services, and the level of public welfare protection achieved, using
17 a cumulative, seasonal exposure metric, such as the W126-based metric. In addition, CASAC
18 has consistently since the 1997 review expressed support for the use of such a metric, including
19 most recently in its letter on the first draft PA (Frey and Samet, 2012b, p. 2). Thus, based on the
20 long-established conclusions and long-standing supporting evidence described above, we
21 continue to conclude that the most appropriate and biologically-relevant way to relate O_3
22 exposure to plant growth, and to determine what would be adequate protection for public welfare
23 effects attributable to the presence of O_3 in the ambient air, is to express exposures in terms of a
24 cumulative seasonal form, and in particular the W126 metric. Accordingly, in considering the
25 current evidence and exposure/risk information with regard to the adequacy of public welfare
26 protection it affords, staff has considered both the evidence of vegetation and welfare impacts in
27 areas of the U.S. likely to have met the current standard, as well as air quality information
28 regarding W126 index values (and evidence of effects associated with those values) in areas
29 likely to have met the current standard.

30   We further take note of CASAC advice in the last review, where they have recognized
31 that the current secondary standard is of an averaging time and form pertaining more to O_3
32 effects on human health than to the effects of O_3 exposure on welfare. In so doing, they
33 emphasized the dependency of vegetation effects on the cumulative exposure to and uptake of O_3
34 over the course of the entire growing season (Henderson, 2006). Accordingly, we recognize

1 potential uncertainty in the level of protection provided by the current standard from vegetation
2 related effects, due to its averaging time and form.

3 Based on the considerations described in the sections above and summarized below, we
4 preliminarily conclude that the currently available evidence and exposure/risk information calls
5 into question the adequacy of public welfare protection provided by the current standard and
6 provides support for considering potential alternative standards to achieve greater public welfare
7 protection, especially for sensitive vegetation and ecosystems in federally protected Class I and
8 similar areas.

9 As an initial matter, we note that the CAA does not require that a secondary standard be
10 protective of all effects associated with a pollutant in the ambient air, but only those considered
11 adverse to the public welfare (as described in section 1.3.2 above). In judging whether particular
12 known or anticipated effects should be considered adverse, the Administrator considers a number
13 of factors, including the intended use of the affected entity(s) and the location(s) in which those
14 effects occur or are predicted to occur. In this regard, effects that have been observed in
15 federally protected Class I areas which are afforded special protections under the CAA may have
16 greater significance to the public welfare. In this context, staff has given particular consideration
17 to recent study findings of welfare-related effects in such areas, in combination with information
18 on air quality during the study periods to inform our conclusions regarding the nature and scope
19 of welfare effects in such protected areas, including areas where the current standard is met or
20 likely to have been met.

21 Regarding visible foliar injury, new research includes: 1) controlled exposure studies; 2)
22 multi-year field surveys; 3) US FS FIA biomonitoring program surveys and assessments. In
23 addition to supporting the ISA causal determination, these studies also address some
24 uncertainties indentified in the previous review (i.e., influence of soil moisture on visible injury
25 development) and further show that visible foliar injury can occur under conditions where 8-hour
26 average O_3 concentrations are or would be expected to be below the level of the current standard
27 (e.g., Kline et al., 2008, as discussed in section 5.4.1 above). Incidence of visible foliar injury
28 symptoms on O_3-sensitive vegetation has also been documented in the field in federally
29 protected areas that have met the current standard. As one example, in Cape Romain National
30 Wildlife Refuge in SC, where air quality data indicate O_3 concentrations have met the current
31 standard over the period from 2001-2012, recent study findings indicate that three species have
32 exhibited O_3-induced visible foliar injury (winged sumac, Chinese tallow tree, and wild grape).
33 In at least one of the studied locations, up to 32 % of the wild grape plants examined showed
34 foliar injury symptoms during years where 8-hour O_3 concentrations were similar to the level of
35 the current standard (U.S. EPA 2013, section 9.4.2.1). Importantly, these O_3-induced vegetation

1 effects were found in Class I areas which have identified O_3-impacts to vegetation as an air
2 quality related value that should be protected commiserate with its congressionally recognized
3 public welfare significance (75 FR 3024). A staff air quality assessment identified Class I areas
4 with recent air quality that met the current standard but was above a W126 index value of 15
5 ppm-hrs (Table 5-2 above). Given evidence of the potential occurrence of visible foliar injury at
6 W126 index values of this magnitude, staff concludes that air quality levels that are at or below
7 the level of the current standard may allow levels of visible foliar injury incidence to occur in
8 areas of special significance to the public welfare.

9   Results from the exposure and risk assessments also indicate visible foliar injury at sites
10 with air quality likely to meet the current standard. In the national-scale analysis of the presence
11 or absence of foliar injury across the years 2006-2010 in U.S. National Forest sites with annual
12 W126 index values of from 7 to 15 ppm-hrs, the percent of sites indicating the presence of foliar
13 injury generally ranges from about 5 to 20 percent, varying with soil moisture conditions for the
14 year. Across all sites, regardless of the year's soil moisture conditions, these analyses find an
15 increase in the proportion of sites showing the presence of foliar injury or elevated foliar injury
16 with increasing W126 index values up to W126 values of approximately 10 ppm-hrs (see U.S.
17 EPA 2014, Figure 7-10). Using this relationship, a National Park screening assessment (214
18 parks) found that 58% of the parks analyzed exceeded a W126 of 10.46 ppm-hrs, which is
19 associated with 17.7% injury prevalence. Data indicate large reductions in injury prevalence
20 with incrementally lower W126 index values. Just meeting the current standard would bring all
21 parks under 10.46 ppm-hrs. However, as noted above, there are important uncertainties
22 associated with the model-adjusted air quality used in these assessments (U.S. EPA, 2014, Table
23 7-23, section 8.5.2) that we note in considering what the adjustment results convey with regard to
24 W126 conditions associated with just meeting the current standard.

25   We also note that visible foliar injury is associated with important cultural and
26 recreational ecosystem services to the public, such as scenic viewing, wildlife-watching, hiking,
27 and camping, that are of significance to the public welfare and enjoyed by millions of Americans
28 every year, generating millions of dollars in economic value (U.S. EPA 2014, section 7.1). In
29 addition, several tribes have indicated that many of the known confirmed O_3-sensitive species
30 (including bioindicator species) are culturally significant (see Appendix 5A). These ecosystem
31 services are at risk of impairment because of O_3-induced damage directly due to foliar injury,
32 though data is not available to explicitly quantify these negative effects. Thus, while it is unclear
33 to what extent O_3-induced visible foliar injury impacts these services, we conclude it is
34 appropriate to consider the potential for these effects to contribute to adverse effects on public

1   welfare in considering the adequacy and appropriateness of the protection afforded by the current
2   standard.

3   Tree biomass loss is another O_3-induced effect that is important from a public welfare
4   perspective, both alone and in conjunction with associated ecosystem-level impacts. This effect
5   occurs in both Class I and non-Class I areas and its importance comes from whether this effect
6   reduces the intended use of the plant or its associated ecosystems in a way that is relevant to the
7   public welfare. Recent studies confirm and extend the evidence of O_3-related effects on tree
8   growth, productivity and carbon storage. Analysis of existing data conducted by the EPA staff
9   and discussed in the ISA has substantially reduced the uncertainty associated with using OTC C-
10 R functions to predict tree growth effects in the field, as described in section 5.2.1 above (U.S.
11 EPA, 2013, section 9.6.3.2). Based on the 12 individual tree seedling C-R functions, seven of
12 the 12 species show 2% seedling biomass loss at a W126 index value below 8 ppm-hrs and in the
13 other five species at a W126 value above 18 ppm-hrs. Within the group of seven more sensitive
14 species, the most sensitive are cottonwood and black cherry (U.S. EPA 2014, section 6.2).
15 Based on the median composite C-R functions, the W126 index values associated with a 2%
16 biomass loss range from approximately 7 to 14 ppm-hrs (U.S. EPA, 2014, Figure 6-4, section
17 6.2.1.2). Recent studies have provided additional evidence on tree biomass or growth effects
18 associated with multiple year exposures in the field, including the potential for compounding and
19 carry-over effects. These effects could lead to a negative impact on species regeneration and
20 have implications for the subsequent growing season in the following year (U.S. EPA, 2013,
21 section 9.4), as recognized in recent advice from CASAC (Frey and Samet, 2012b).
22 Accordingly, in considering WREA biomass loss estimates, we also consider the WREA
23 evaluation of a potential for underestimation of such effects (U.S. EPA, 2014, section 6.2.1.4).
24 While it is not possible at this time to identify the extent or magnitude of such effects in the field
25 under exposure levels that may be associated with the current standard, their occurrence, when of
26 a magnitude of concern, on federally protected lands or any associated impacts on ecosystem
27 services such as habitat quality, hydrologic regimes, carbon storage or air pollution removal in
28 areas important to the public might reasonably be concluded to be significant to public welfare.
29 We additionally take note of the WREA findings with regard to urban areas where tree CO_2
30 sequestration and air pollutant removal are important public welfare services.
31   With respect to crops, the detrimental effect of O_3 on crop production has been
32 recognized since the 1960s, and recent O_3 concentrations in many areas across the U.S. are high
33 enough that they might be expected to cause yield loss in a variety of agricultural crops
34 including, but not limited to, soybeans, wheat, potatoes, watermelons, beans, turnips, onions,
35 lettuces, and tomatoes (U.S. EPA, 2013, section 9.4.4). In general, the vast majority of the new
36 scientific information confirms prior conclusions that exposure to O_3 can decrease growth and

1   yield of crops. Recent research has highlighted the effects of O_3 on crop quality. Increasing O_3

2   concentration decreases nutritive quality of grasses, decreases macro- and micro-nutrient

3   concentrations in fruits and vegetable crops (U.S. EPA 2013, section 9.4.4). Recent studies

4   continue to find yield loss levels in crop species studied previously under NCLAN that reflect

5   the earlier findings. There has been little published evidence that crops are becoming more

6   tolerant of O_3 (U.S. EPA, 2006a; U.S. EPA 2013). This is especially evident in the research on

7   soybean. The 2013 ISA reported comparisons between yield predictions based on data from

8   cultivars used in NCLAN studies, and yield data for modern cultivars from SoyFACE (U.S.

9   EPA, 2013, section 9.6.3). They confirm that the average response of soybean yield to O_3

10   exposure has not changed in current cultivars. In addition, satellite and ground-based O_3

11   measurements have been used to assess yield loss caused by O_3 over the continuous tri-state area

12   of Illinois, Iowa, and Wisconsin. The results showed that O_3 concentrations reduced soybean

13   yield, which correlates well with the previous results from FACE- and OTC-type experiments

14   (U.S. EPA, 2013, section 9.4.4.1). Thus, the recently available evidence, as assessed in the ISA,

15   continues to support the conclusions of the 1996 and 2006 Criteria Documents that ambient O_3

16   concentrations can reduce the yield of major commodity crops in the U.S.

17   The currently available evidence, as assessed in the ISA, continues to support the use of

18   C-R functions based on OTC experiments. Further, important uncertainties have been reduced

19   regarding the exposure-response functions for crop yield loss, especially for soybean. In general,

20   the ISA reports consistent results across exposure techniques and across crop varieties (U.S. EPA

21   2013, section 9.6.3.2). Based on the crop C-R functions, the W126 index values associated with

22   a five percent yield loss range from approximately 12 to 17 ppm-hrs. The lower end of the range

23   would be more protective of soybeans which are the second-most planted field crop in the U.S.

24   (http://www.ers.usda.gov/topics/crops/soybeans-oil-crops/background.aspx). Staff analyses of

25   recent monitoring data (2009-2011) indicate that O_3 concentrations in multiple agricultural areas

26   in the U.S. that meet the current standard correspond to W126 index levels above 12 ppm-hrs.

27   The information for other welfare effects, including those with causal or likely causal

28   relationships with O_3 (i.e., alteration of ecosystem water cycling, changes in climate), is limited

29   with regard to our ability to consider potential impacts of air quality conditions associated with

30   the current standard, although the WREA provides some perspective on this issue with regard to

31   susceptibility to insect attack and fire regime change. We note, however, the importance of these

32   effects categories to the public welfare.

33   Given all of the above, staff reaches the preliminary conclusion that the available

34   evidence and exposure and risk information calls into question the adequacy of public welfare

35   protection provided by the current standard, and provides strong support to giving consideration

1 to revising the current secondary standard to provide increased public welfare protection. More
2 specifically, staff preliminarily concludes that it is appropriate for the Administrator to consider
3 revision of the current secondary O_3 standard to increase protection against O_3-attributable tree
4 biomass loss, crop yield loss, and visible foliar injury, and particularly for those effects
5 associated with cumulative, seasonal exposures that occur in specially protected natural areas. In
6 reaching conclusions on options for the Administrator's consideration, we note that the final
7 decision to retain or revise the current secondary O_3 standard is a public welfare policy judgment
8 to be made by the Administrator as to what standards would be requisite (i.e., neither more nor
9 less stringent than necessary) to protect the public welfare from any known or anticipated
10 adverse welfare effects. This final decision will draw upon the available scientific evidence for
11 O_3-attributable welfare effects and on analyses of vegetation and ecosystem exposures and public
12 welfare risks based on impacts to vegetation, ecosystems and their associated services, including
13 judgments about the appropriate weight to place on the range of uncertainties inherent in the
14 evidence and analyses. In determining the requisite level of protection for crops and trees, the
15 Administrator will need to weigh the importance of the predicted risks of these effects in the
16 overall context of public welfare protection, along with a determination as to the appropriate
17 weight to place on the associated uncertainties and limitations of this information.
18   As noted in section 1.3.2 above, our general approach to informing these judgments,
19 recognizes that the available welfare effects evidence reflects a continuum consisting of higher
20 O_3 concentrations at which scientists generally agree that welfare effects are likely to occur,
21 through lower concentrations at which the likelihood and magnitude of a response become
22 increasingly uncertain. Therefore, in presenting conclusions here, we are mindful that the
23 Administrator's ultimate judgment on the adequacy of protection afforded by the current
24 standard will most appropriately reflect an interpretation of the available scientific evidence and
25 exposure/risk information that neither overstates nor understates the strengths and limitations of
26 that evidence and information.

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6 CONSIDERATION OF ALTERNATIVE SECONDARY STANDARDS

To the extent that the information available in this review suggests that revision of the current secondary standard is appropriate to consider, as discussed in chapter 5, staff has evaluated the available body of evidence, and exposure, risk and air quality information with regard to the support for consideration of alternative standards, as articulated by the following overarching question:

- **What alternative secondary standards are supported by the currently available scientific evidence, exposure/risk information and air quality analyses?**

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 in sections 6.1, 6.2 and 6.3 below. We consider both the scientific and technical information available at the time of the last review and information newly available since the last review which has been critically analyzed and characterized in the ISA. Specifically, we consider the currently available scientific evidence and technical information in the context of decisions regarding the basic elements of the NAAQS: indicator, (section 6.1); averaging time and form (section 6.2); and level (section 6.3). CASAC advice on potential alternative standards is described in section 6.4 and preliminary staff conclusions on potential alternative standards are discussed in section 6.5. Section 6.6 summarizes preliminary staff conclusions on adequacy of the current standard and potential alternative standards appropriate to consider. Key uncertainties in this review and areas in which future research and data collection would better inform the next review are identified in section 6.7.

6.1 INDICATOR

With regard to the indicator for potential alternative secondary standards, we consider the following question.

- **Does the information available in this review continue to support O_3 as the indicator for ambient air photochemical oxidants?**

In the last review of the air quality for O_3 and other photochemical oxidants and of the O_3 standard, as in other prior reviews, the EPA focused on a standard for O_3 as the most appropriate surrogate for ambient photochemical oxidants. Ozone is a long-established surrogate for ambient photochemical oxidants, among which it is by far the most widely studied with regard to effects on welfare and specifically on vegetation. The information available in this review adds to our understanding of the atmospheric chemistry for photochemical oxidants and O_3 in

1 particular (as described in the ISA, sections 3.2 and 3.6, and summarized in section 2.2 in this

2 document). The 1996 Staff Paper noted that the database on vegetation effects is generally

3 considered to raise concern at levels found in the ambient air for O_3 and, therefore, control of

4 ambient O_3 levels has previously been concluded to provide the best means of controlling other

5 photochemical oxidants of potential welfare concern (U.S. EPA, 1996b, p. 277). In the current

6 review, while the complex atmospheric chemistry in which O_3 plays a key role has been

7 highlighted, no alternatives to O_3 have been advanced as being a more appropriate surrogate for

8 ambient photochemical oxidants. Ozone continues to be the only photochemical oxidant other

9 than nitrogen dioxide that is routinely monitored and for which a comprehensive database exists

10 (U.S. EPA, 2013, section 3.6). Thus, staff concludes that O_3 remains the appropriate pollutant

11 indicator for use in a secondary NAAQS that provides protection for public welfare from

12 exposure to all photochemical oxidants.

13 **6.2 FORM AND AVERAGING TIME**

14   In considering potential forms and averaging times alternative to that of the current

15 secondary standard (i.e. 4th high daily maximum 8-hour average, averaged over 3 years) we

16 address several specific questions.

17 • **To what extent does the currently available information provide support for**
18   **considering forms different from that of the current secondary standard?**

19   In characterizing the current evidence, the ISA states that "[n]o recent information is

20 available since 2006 that alters the basic conclusions put forth in the 2006 and 1996 O_3 AQCDs"

21 with regard to biologically relevant exposure indices (U.S. EPA, 2013, section 2.6.6.1). Based

22 on the current state of knowledge and the best available data assessed in this review, the ISA

23 therefore concludes that exposure indices that cumulate and differentially weight the higher

24 hourly average concentrations over a season and also include the mid-level values continue to

25 offer the most defensible approach for use in developing response functions and in defining

26 indices for vegetation protection (U.S. EPA, 2013, section 2.6.6.1). In particular, the available

27 body of evidence provides a wealth of information, compiled over several decades, on the

28 aspects of O_3 exposure that are most important in influencing plant response. As discussed in the

29 ISA, the importance of the duration of the exposure and the relatively greater importance of

30 higher concentrations (over lower concentrations) in determining plant response to O_3 have been

31 well documented (U.S. EPA, 2013, section 9.5.3). Building on this research, other work has

32 been focused on developing "mathematical approaches for summarizing ambient air quality

33 information in biologically meaningful forms for O_3 vegetation effects assessment purposes …"

34 (U.S. EPA, 2013, section 9.5.3), including those known as cumulative, concentration weighted

1 forms (i.e. SUM06, W126). Much of this work was completed by the mid-1990s, and was

2 summarized in the 1996 Criteria Document (CD) (U.S. EPA, 1996a, section 5.5).

3   On the basis of this longstanding and extensive evidence demonstrating that the risk to

4 vegetation comes from cumulative seasonal exposures, the EPA in the 1997 and 2008 reviews, as

5 well as in the 2010 proposed rulemaking to reconsider the 2008 decision, recognized the

6 importance of cumulative, seasonal exposures as a primary determinant of plant responses to O_3

7 in ambient air (61 FR 65741-42; 62 FR 38878; 72 FR 37893, 37896, 37900, 37904; 73 FR

8 16488-90, 16493-94; 75 FR 3000, 3010, 3012). For example, in the 1996 notice of proposed

9 rulemaking, the Administrator recognized that the scientific evidence supported the conclusion

10 that "a cumulative seasonal exposure index is more biologically relevant than a single event or

11 mean index" (61 FR 65742). In the 2008 review, CASAC recognized that an important

12 difference between the effects of short-term exposures to O_3 on human health and the effects of

13 O_3 exposures on welfare is that "vegetation effects are more dependent on the cumulative

14 exposure to, and uptake of, O_3 over the course of the entire growing season" (Henderson, 2006).

15 In that review, the CASAC O_3 Panel members were unanimous in supporting the final Staff

16 Paper recommendation that "protection of managed agricultural crops and natural terrestrial

17 ecosystems requires a secondary Ozone NAAQS that is substantially different from the primary

18 ozone standard in averaging time, level and form" (Henderson, 2007). Accordingly, in both the

19 1997 and 2008 reviews as well as the 2010 reconsideration, the Administrator proposed a

20 secondary standard with a cumulative seasonal form as an appropriate policy option (61 FR

21 65742-44; 72 FR 37899-905; 75 FR 3012-3027).

22   Over the past two decades, several indices have been considered with regard to their

23 representation of O_3 exposure in terms best describing its effects on vegetation. The 1996 CD

24 and 1996 Staff Paper evaluated different types of forms to determine which performed the best in

25 describing the available data on plant response to O_3 exposures. These documents noted that a

26 number of forms (e.g., the one event, mean and unweighted cumulative SUM00) are unable to

27 reliably predict plant response because they either ignore the role of duration or ignore the

28 disproportionate impact of higher concentrations by weighting all concentrations equally (U.S.

29 EPA, 1996b, p. 224). Other forms that were considered include multicomponent forms.

30 Because these forms take into account many other relevant factors (e.g., plant growth stage,

31 predisposition from earlier exposures), they consistently predict plant response best of all the

32 different exposure forms. However, due to being species-specific and highly complex, they were

33 not considered suitable for more general application in the context of standard setting (U.S. EPA,

34 1996b, pp. 224-225). On the other hand, it was also found that concentration-weighted forms

35 that take into account the role of duration and concentration perform almost as well as the

36 multicomponent forms. These include several threshold forms (e.g., SUM06, AOT60) and

1   sigmoidally weighted cumulative indices (e.g., W126) (U.S. EPA, 1996a, pp. 5-84 to 5-136; U.S.
2   EPA, 1996b, pp. 223-227). Given that these cumulative concentration-weighted forms all were
3   able to similarly predict plant response on the datasets for which they were evaluated (i.e.
4   NCLAN), it was not possible to distinguish between them on this basis. Partly as a result,
5   CASAC deliberations in 1995 did not produce a consensus on which form would be best suited
6   for a secondary NAAQS. As discussed further in 6.3 below, a workshop held in January of 1996
7   provided a consensus recommendation on the SUM06 form as appropriate for use in secondary
8   standards, while also recognizing that a W126 form could also be appropriate (Heck and
9   Cowling, 1997). Subsequent to this, the final 1996 Staff Paper and 1996 proposal notice both
10   identified the SUM06 form as appropriate to consider and propose, respectively (U.S. EPA,
11   1996b, p. 285, 61 FR 65716). In selecting the SUM06 form, it was noted that while it imposed a
12   threshold even while evidence was lacking for a discernible threshold for O_3-related vegetation
13   effects, it had the benefit of not including concentrations considered to be from background
14   sources, which was an important feature (U.S. EPA, 1996b, pp. 223-227).
15   In the subsequent review, the form of the standard was revisited in light of continued
16   evidence that there remained a lack of discernible threshold for vegetation effects in general, and
17   newer estimates of O_3 concentrations associated with background sources that were lower than in
18   the previous review so that their inclusion was less of a concern. On these bases, the 2007 Staff
19   Paper recommended consideration of the W126 index[1] as the basis for the form of a distinct
20   secondary standard (U.S. EPA, 2006, pp. 9-11 to 9-15 and pp. AX9-159 to AX9-187; U.S. EPA,
21   2007, pp. 7-15/16). The EPA then proposed two options for the secondary standard, one of
22   which was to adopt a cumulative, seasonal standard based on the W126 index, while the other
23   option was a secondary standard identical to the proposed revised primary standard (72 FR
24   37818). The CASAC Panel in that review expressed preference for the W126 index (Henderson,
25   2006). In EPA's proposed reconsideration of the 2008 decision, the Agency proposed only the
26   option of a cumulative, seasonal standard based on the W126 function (75 FR 2938).
27   In this review, we conclude that specific features associated with the W126 index still
28   make it the most appropriate cumulative concentration-weighted form for use in the context of
29   the secondary O_3 NAAQS review. In particular, the W126 index does not apply an arbitrary
30   exposure threshold below which concentrations are not included. Given the acknowledged range
31   in vegetation sensitivity and the continued lack of evidence of an exposure threshold for effects
32   above a W126 index of zero, such a feature is desirable. In addition, we conclude the W126

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[1] The W126 is a non-threshold approach described as the sigmoidally weighted sum of all hourly O_3
concentrations observed during a specified diurnal and seasonal exposure period, where each hourly O_3
concentration is given a weight that increases from 0 to 1 with increasing concentration (Lefohn et al, 1988; Lefohn
and Runeckles, 1987; U.S. EPA, 2013, section 9.5.2).

1   sigmoidal weighting function provides the best way to weight concentrations associated with

2   background sources. Thus, we conclude that the W126 form is best matched to the evidence

3   associated with vegetation effects, as well as addressing the policy-relevant issue of how to

4   weight exposures associated with background sources.

5   - **To what extent does the currently available information provide support for**
6   **consideration of a cumulative seasonal form derived as a sum of weighted O$_3$**
7   **concentrations over daylight hours (8:00 am to 8:00 pm) for the 3 month period**
8   **having the highest sum within the O$_3$ season?**

9   For a standard with a cumulative seasonal form, including one defined in terms of the

10   W126 exposure index, the NAAQS element of "averaging time" is appropriately considered in

11   terms of exposure periods – diurnal and seasonal -- over which the index would be summed in

12   any given year. In the EPA's consideration of such a standard in both the 1997 and 2008

13   reviews, the diurnal period of interest was identified to be the daylight hours, which were defined

14   for these purposes as spanning the 12-hour period from 8 am to 8 pm during the growing season.

15   The seasonal period of interest was identified as the consecutive 3 month period with the

16   maximum W126 index value that occurs within the monitored O$_3$ season. Since many species of

17   vegetation have growing seasons longer than 3-months in some locations within the U.S., this

18   seasonal period is viewed as the minimum duration that can appropriately serve as a surrogate

19   for the range of growing seasons associated with U.S. vegetation (72 FR 37883; U.S. 2013,

20   section 9.5.3, p. 9-112/13. As discussed below, the evidence available in this review continues

21   to provide support for use of these periods in considering a cumulative seasonal secondary

22   standard (U.S. EPA, 2013, section 9.5.3).

23   For the majority of plants, the diurnal conditions of maximum O$_3$ uptake occur mainly

24   during the daytime hours (U.S. EPA, 2013, section 9.5.3.2). This is because, in general, (1)

25   plants have the highest stomatal conductance during the daytime; (2) atmospheric turbulent

26   mixing is greatest during the day in many areas; (3) the high temperature and high light

27   conditions that typically promote the formation of tropospheric O$_3$ also promote physiological

28   activity in vegetation (U.S. EPA, 2013, section 9.5.3.2). In consideration of such a cumulative

29   metric in the prior review, the EPA has identified the 12-hour period from 8 am to 8 pm as

30   appropriately capturing the diurnal window with most relevance to the photosynthetic process

31   (72 FR 37900; 75 FR 3013). In so doing, the EPA recognized, as did CASAC that in some parts

32   of the country this period may not include all daytime hours or exposures of importance to

33   vegetation, thus potentially underestimating the impact of O$_3$ at those sites (72 FR 37900-01; 75

34   FR 3013-14; Henderson, 2007, p. 3, pp. C-22-23).

35   In addition to daytime uptake, a number of studies have also reported O$_3$ uptake at night

36   in some species (U.S. EPA, 2013, section 9.5.3.2). Typically the rate of stomatal conductance at

1   night is much lower than during the day. Several field studies have attempted to quantify night-
2   time O_3 uptake with a variety of methods. Across the studies discussed in the ISA, nocturnal
3   conductance ranged from negligible to 25% of daytime values (U.S. EPA, 2013, section 9.5.3.2).
4   In some studies the percent of nocturnal uptake varied by season and drought conditions.
5   However, many of these studies did not link the night-time flux to measured effects on plants,
6   making it difficult to know whether the impacts on the plant from nocturnal exposures are
7   greater or less than those from similar daytime exposures, and whether or not they should be
8   considered as separate impacts or as additive or synergistic with impacts from the preceding
9   daytime exposure. In addition to the uncertainties associated with understanding the plant
10   response to nocturnal uptake, there are also uncertainties associated with the extent of its
11   occurrence. For significant nocturnal stomatal flux and O_3 effects to occur, a susceptible plant
12   with nocturnal stomatal conductance and low defenses must be growing in an area with relatively
13   high night-time O_3 concentrations (often high elevation sites) and appreciable nocturnal
14   atmospheric turbulence. It is unclear how many areas there are in the U.S. where these conditions
15   occur. It may be possible that these conditions exist in mountainous areas of southern California,
16   front-range of Colorado and the Great Smoky Mountains of North Carolina and Tennessee.
17   More information is needed in locations with high night-time O_3 to assess the local O_3 patterns,
18   micrometeorology and responses of potentially vulnerable plant species (U.S. EPA, 2013,
19   section 9.5.3.2).
20   In consideration of such uncertainties regarding the importance and extent of nocturnal
21   exposures associated with plant uptake, and whether and how they might be incorporated into a
22   national index, staff continues to focus on the 12-hour daytime exposure period of 8:00 am to
23   8:00 pm, consistent with CASAC advice in the last review (Henderson, 2007, p. 3). In so doing,
24   we recognize the variability in daylight hours across the country and across the O_3 seasons, such
25   that this period will not always include all the daylight hours in all areas. Thus, a focus on this
26   set period for all locations has the potential to underestimate the impact of O_3 exposures in some
27   areas, although, given that available monitoring data indicates that the daily increase in O_3
28   concentrations generally does not begin until after 8 am (U.S. EPA, 2013, section 3.6.3.2), as
29   well as our additional focus on the 3-month period with the highest index (as discussed below),
30   the extent of such underestimation is unclear. We note, however, the variability in daylight
31   hours nationwide in areas to which a W126-based secondary standard would apply as well as in
32   areas where studies have been conducted that form the basis for consideration of such a standard
33   is an important consideration in identifying the appropriate elements for a W126-based
34   secondary standard.
35   With regard to identification of the seasonal period over which to cumulate exposures,
36   EPA notes that a plant is vulnerable to O_3 pollution as long as it has foliage and is

1   physiologically active (U.S. EPA, 2013, section 9.5.3, p. 9-112), i.e. during its growing season.
2   The length of vegetative growing seasons varies depending on the type or species of vegetation
3   and where it grows. For example, as discussed in the ISA, annual crops are typically grown for
4   periods of two to three months while perennial species may be photosynthetically active longer,
5   and up to 12 months each year for some species (U.S. EPA 2013, section 9.5.3, p. 9-112). In
6   general, the period of maximum physiological activity and thus, potential O_3 uptake for
7   vegetation coincides with some or all of the intra-annual period defined as the O_3 season, which
8   varies on a state-by-state basis (U.S. EPA, 2013, Figure 3-24, p. 3-83). This is because the high
9   temperature and high light conditions that typically promote the formation of tropospheric O_3
10 also promote physiological activity in vegetation (U.S. EPA, 2013, section 9.5.3, p. 9-112).

11   The exposure periods used in studies of O_3 effects on vegetation reflect this
12 understanding, with crop studies typically using shorter seasonal exposure periods, while studies
13 of longer lived trees and other perennial vegetation often extend for the entire annual growing
14 season or in some cases over multiple growing seasons. Specifically, the ISA notes that "[m]ost
15 of the crop studies done through NCLAN had exposures less than three months with an average
16 of 77 days. Open-top chamber studies of tree seedlings, compiled by the EPA, had an average
17 exposure of just over three months or 99 days. In more recent FACE experiments, SoyFACE
18 exposed soybeans for an average of approximately 120 days per year and the Aspen FACE
19 experiment exposed trees to an average of approximately 145 days per year of elevated O_3,
20 which included the entire growing season at those particular sites. Further, the U.S. Forest
21 Service and federal land managers have typically used the 6 months from April through
22 September as the accumulation period (U.S, EPA, 2013, section 9.5.3.2, p. 9-112). However,
23 despite the possibility that plants may be exposed to ambient O_3 longer than 3 months in some
24 locations, the ISA notes that "[t]he exposure period in the vast majority of O_3 exposure studies
25 conducted in the U.S. has been much shorter than 6 months…" and "there is generally a lack of
26 exposure experiments conducted for longer than 3 months" (U.S. EPA, 2013, section 9.5.3.2, p.
27 9-112)

28   As a result, analyses of effects in terms of the W126 exposure index have typically
29 defined the index in terms of a 3-month exposure period or at least in terms of periods shorter
30 than 6 months (e.g. SoyFACE, AspenFACE) (U.S, EPA, 2013, p. 112). In addition, the O_3
31 season within which O_3 monitoring is required is shorter than 6 months in many areas in the
32 country, yet longer than 3 months in all locations (U.S. EPA, 2013, section 3.6.2, p. 3-83). Thus,
33 in the last review, the EPA proposed use of the consecutive 3-month period within the O_3 season
34 having the maximum W126 index value. A 3-month exposure period was also supported by
35 CASAC in advice provided both during the last review and on the 2010 proposed reconsideration
36 (Henderson, 2006; Samet, 2010). In revisiting this issue in the current review, the EPA

1   conducted a new analysis to further inform the consideration of the most appropriate seasonal
2   accumulation period (U.S. EPA, 2013, section 9.5.3). This analysis calculated and compared the
3   3- and 6-month maximum W126 values for over 1,200 AQS and CASTNET EPA monitoring
4   sites for the years 2008-2009. The two accumulation periods were found to have highly
5   correlated metrics (U.S. EPA, 2013, Figure 9-13). The two accumulation periods were centered
6   on the yearly maximum for each monitoring site, and it is possible that this correlation would be
7   weaker if the two periods were not temporally aligned (U.S. EPA, 2013, section 9.5.3). The
8   analysis indicates that in the U.S., W126 cumulated over 3 months and W126 cumulated over 6
9   months are proxies of one another, as long as the period in which daily W126 is accumulated
10 corresponds to the seasonal maximum. Therefore, it is expected that either statistic will predict
11 vegetation response equally well.

12 • **Are there other aspects of the form that are appropriate to consider that**
13   **contribute to welfare protection provided by a cumulative seasonal standard?**

14   In considering a secondary standard in terms of the W126 exposure index with daylight
15 12-hour averaging time and maximum consecutive 3-month cumulative exposure period, we find
16 it appropriate to evaluate the protection that might be afforded by a form limited to a single year
17 or one that is based on evaluation of W126 index values across multiple years. In so doing, we
18 are mindful that the protection provided by the secondary standard derives from the combination
19 of all elements of the standard. Accordingly, the discussion below explores the information
20 relevant to consider *in conjunction with* identification of standard level, as well as whether there
21 is a lack of support in the current information for either single or multi-year options.
22   In considering an annual form of a standard, we particularly take note of O_3-induced
23 vegetation effects that can occur as a result of a single year's exposure. These include visible
24 foliar injury symptoms, growth reduction in annual species, and crop yield loss in annual crops.
25 The following discussion considers these effects, in the context of their potential public welfare
26 significance, with regard to the extent to which a W126-based standard with an annual form or
27 one based on evaluation across multiple years may be able to provide appropriate protection.
28   Visible foliar injury that occurs on public lands that have been afforded special
29 protections (e.g., Class I areas) is an annual effect with potential public welfare significance,
30 particularly when the injury is of some severity. However, we recognize the complexity
31 associated with defining O_3 exposure conditions that might consistently provide appropriate
32 protection from this endpoint. As summarized in section 5.4.2 and described more completely in
33 the WREA, over 80% of USFS biosites show no foliar injury in a wide variety of tree species
34 under air quality conditions (2006-2010) that include a broad range of W126 index values (U.S.
35 EPA 2014, section 7.2). The WREA USFS biosite analysis additionally suggested a reduction in
36 prevalence of sites with visible foliar injury with W126 index values below approximately 10

1   ppm-hrs. However, the full body of evidence indicates wide variability in this endpoint, such

2   that although evidence shows visible foliar injury can occur under very low cumulative O_3

3   concentrations, "…the degree and extent of visible foliar injury development varies from year to

4   year and site to site…, even among co-members of a population exposed to similar O_3 levels, due

5   to the influence of co-occurring environmental and genetic factors" (U.S. EPA 2013, section

6   9.4.2, p. 9-38). In particular, drought conditions which can frequently occur with high seasonal

7   O_3 concentrations can potentially offer some protection so that the visible foliar injury that might

8   be expected based only on O_3 exposure conditions does not develop. Accordingly, staff

9   recognizes the challenges associated with characterizing the role of relative soil moisture, and its

10   appreciable spatial and temporal variability. For example, the WREA analysis of the

11   relationship between foliar injury at USFS biosites and soil moisture data found foliar injury to

12   be less prevalent in years when sites were drier (U.S. EPA, 2014, section 7.2 and Figure 7-10).

13   The uncertainties associated with describing the potential for the occurrence of foliar injury and

14   its severity or extent of occurrence for any given set of O_3 exposure conditions, limit our ability

15   to identify annual ambient O_3 exposure conditions that might be expected to provide an

16   appropriate degree of protection. Thus, the evidence for this endpoint does not appear to provide

17   support for the necessity of consideration of a single year form.

18   Another annual effect with the potential for public welfare significance is that of reduced

19   yields in annual commodity crops. It has been well documented that certain important

20   commodity crops are sensitive to O_3 and that achieving the optimum level of crop yields is of

21   special importance to the public welfare. Although the impact of this effect can be monetized,

22   determining what the optimum yields are is less straightforward. This is because optimizing the

23   public welfare associated with annual crop production is not necessarily the same as maximizing

24   the annual yield but instead by achieving the maximum levels of producer and consumer

25   surpluses. These latter objectives depend less on the magnitude of the annual O_3 exposure and

26   more on the management strategies employed by the agricultural community during that year,

27   strategic planting choices that are made from year to year, and environmental factors (e.g.,

28   drought) which can in large part offset these concerns (U.S. EPA, 2014, Chapter 6, section 6.5).

29   Thus, since the optimum annual yield is not necessarily equivalent to the greatest public welfare

30   value, it is difficult to know what degree of protection from O_3 would be requisite in any given

31   year. Thus, as in the 2008 review, we do not believe that a public welfare benefit of optimizing

32   crop yields by protection of commodity crops from annual O_3 impacts is necessarily achievable

33   through the secondary NAAQS (73 FR 16492, 16497). Further, depending on the level of

34   protection being considered for other vegetation effects (i.e. seedling biomass loss), crops may

35   indirectly receive protection. Thus, similar to the annual foliar injury effect, the uncertainties

36   associated with identifying at what point annual yield loss in commodity crops becomes adverse

1   to the public welfare make it difficult to identify an appropriate degree of protection, particularly

2   when considering the effect of a single year.

3   In contrast to impacts on annual species that accrue in the single growing season in which

4   the O_3 exposures occur, annual effects in perennial species can be "carried over" into the

5   subsequent year where they affect growth and reproduction (U.S. EPA, 2013, pp. 9-43 to 9-44

6   and p. 9-86). In addition, when these effects occur over multiple years due to elevated O_3

7   exposures across several years, they are compounded, increasing the potential for effects at the

8   ecosystem level and associated ecosystem services that may be of significance to the public

9   welfare. For example, in perennial species, O_3-induced reduction in photosynthesis decreases

10   the amount of carbohydrate produced by and available to the plant for growth and storage. The

11   plant often responds to decreases in carbohydrate production by reallocating the remaining

12   carbohydrate to above ground growth at the expense of storage in and growth of the roots (U.S.

13   EPA, 2013, section 9.4.3.1). When fewer carbohydrates are available in the roots, less energy is

14   available for root-related functions such as water and nutrient acquisition. By inhibiting the

15   amount of carbohydrate transfer to the roots, O_3 also can disrupt the symbiotic relationship that

16   exists between some plants and mycorrhizal fungi in the soil. Mycorrhizal fungi have been

17   shown to improve the uptake of nutrients, protect host plant roots against pathogens, and produce

18   plant growth hormones (U.S. EPA, 2007, 7.3.3.2). Thus, such effects of elevated O_3 in a given

19   year may affect the plant's resistance in a subsequent year, with multiple such years having the

20   potential for larger impacts. However, where there is variation across years such that lower O_3

21   years with lesser impact occur between elevated O_3 years, it may be that the longer-term impacts

22   are tempered.

23   Effects of elevated O_3 years on perennial plants, when they occur over several years, may

24   also contribute to effects on ecosystem services, i.e. alteration of below-ground biogeochemical

25   cycles, and alteration of both above- and below- ground terrestrial community composition and

26   terrestrial ecosystem water cycling (U.S. EPA, 2013, Table 9-19). Ozone has also been shown to

27   affect plant reproduction in numerous ways (U.S. EPA, 2007, 7.3.3.3; U.S. EPA, 2013, 9.4.3.1).

28   These effects, when they occur at sufficient magnitude for a single species, may result in

29   impaired recruitment and loss of the species from the stand or community. This has the potential

30   to change the community composition and biodiversity. If these effects occur in multiple plant

31   species and/or over multiple years, they can result in a reduction in the productivity and carbon

32   sequestration of terrestrial ecosystems. Such ecosystem-related effects and others discussed in

33   the ISA may be considered to reflect impacts of critical O_3 exposures over the longer term. We

34   additionally note that as compared to intermittent (or single year) critical O_3 exposures, multiple

35   years of such exposures might be expected to result in larger impacts on forested areas, i.e.

1   increased susceptibility to other stressors such as insect pests, disease, co-occurring pollutants

2   and harsh weather, due to the compounding or carry-over effects on tree growth.

3   Given the above, we find it reasonable to conclude that the public welfare significance of

4   the effects that can occur as a result of multiple-year O_3 exposures are greater than those

5   associated with a single year. Thus, to the extent that the Administrator's focus for public

6   welfare protection to be afforded by the secondary O_3 standard is on long-term effects that occur

7   in sensitive tree species in natural forested ecosystems including in federally protected areas such

8   as Class I areas or on lands set aside by States, Tribes and public interest groups to provide

9   similar benefits to the public welfare, a standard with a form that evaluates the cumulative

10   seasonal index across multiple years (in combination with an appropriate level) might be

11   considered to provide an appropriate match to the nature of O_3-related effects on vegetation most

12   at risk of being adverse to the public welfare and thus, upon which the secondary O_3 standard is

13   focused.

14   Although cumulative, seasonal exposure values of interest for vegetation effects have

15   been expressed in terms of a single season (e.g., W126 index values for a season's exposure of

16   tree seedlings), we recognize that it is also appropriate to consider a form that is evaluated over a

17   multiple-year period, such as three years (U.S. EPA, 2007; 72 FR 37901; 75 FR 3021). Such an

18   aspect to the form specifies the number of years for which monitoring data are considered in

19   judging attainment with a standard. For example, under a standard with a single year form, a

20   monitor may be judged to meet the standard based on a single year of data, while under a

21   standard with a form requiring evaluation over a multi-year period, a monitor is not judged to

22   have met the standard until a complete multi-year record is available. For metrics with

23   substantial year-to-year variability, a multi-year evaluation period can provide stability in air

24   quality management programs, thus facilitating achievement of the protection intended by a

25   standard, while a standard with an annual form has the potential to contribute to challenges to

26   implementation of air pollution control programs in areas identified as not meeting the standard

27   that, through variability in the metric, meet the standard level in other years.

28   For a W126-based potential standard, the multi-year form identified for consideration in

29   the last review was the average cumulative seasonal metric over three consecutive years (75 FR

30   3027). Such a multi-year form remains appropriate to consider to provide stability to an

31   alternative secondary standard, just as the multi-year form provides for the current standard

32   (average over three years of annual fourth-highest daily maximum 8-hour average O_3

33   concentrations).[2] In considering the issue of stability in the context of such a form, we first note

34   the inter-annual variability of seasonal W126 index, which is not unexpected given the logistic

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[2] See *ATA III*, 283 F. 3d at 374-75 (recognizing programmatic stability as a legitimate consideration in the NAAQS standard-setting process).

1   weighting function and also inter-annual variability in meteorological conditions which
2   contribute to O_3 formation (see Appendix 2C). The staff analysis in Appendix 2C describes the
3   variability in annual W126 values in relation to variability in the 3-year average, which indicates
4   that a standard based on an annual W126 index would be expected to have a lower degree of
5   year-to-year stability relative to a standard based on a form that averages seasonal indices across
6   three consecutive years. A more stable standard can be expected to contribute to greater public
7   welfare protection by limiting year-to-year disruptions in ongoing control programs that would
8   occur if an area was frequently shifting in and out of attainment due to extreme year-to-year
9   variations in meteorological conditions. 283 F. 3d at 374. Thus, we recognize the public welfare
10   benefit of having a standard of a form with more year-to-year stability.
11   In considering the relative value of a multi-year form for an alternative secondary
12   standard in affording the desired protection, we also take note of the influence such a form might
13   be expected to have on air quality conditions and the associated public welfare protection. For
14   example a W126-based standard with an annual form would have a level defining the maximum
15   cumulative seasonal index. Where air quality is attained that reliably meets such a standard, the
16   cumulative seasonal index values would be at or lower than the level of the standard in all years
17   and, given the significant inter-annual variability in seasonal W126 values (described above),
18   would be appreciably below the standard level in many years. Alternatively, for a standard with
19   a form that averages the cumulative seasonal index values across three consecutive years, the
20   seasonal index would be above the level in some years, but would have to be below it in others
21   within the same 3-year period, restricting there to no more than two years out of three to have
22   indices above the level, and depending on magnitude of each year's index, potentially no more
23   than one.
24   In our consideration of this aspect of form for a cumulative seasonal secondary standard,
25   we additionally take note of advice from CASAC on this topic in prior reviews. For example, in
26   comments provided on the final Staff Paper in 2007, CASAC expressed the view that "[i]f multi-
27   year averaging is employed to increase the stability of the secondary standard, the level of the
28   standard should be revised downward to assure that the desired threshold is not exceeded in
29   individual years" (Henderson, 2007). Accordingly, in considering all elements for a revised
30   standard, including level, as well as form, we note the importance of considering the potential for
31   single-year O_3 exposures that would result in adverse effects to public welfare. As noted above,
32   a standard with a form that averages across three years can also control year-to-year variability
33   and individual year concentrations. In so noting, we recognize the availability of air quality
34   analyses that can inform our consideration of the likely extent of such control (e.g., Appendix
35   2C). Additionally, we note the potential that, depending on type of effects and the magnitude
36   which may be judged adverse, low O_3 years within each three-year period might play a

1   countervailing role with regard to the extent of O_3-attributable impacts that might pose public

2   welfare significance. The WREA analyses on the potential for biomass loss estimates based on a

3   3-year average to underestimate the cumulative impact on growth inform this point. Those

4   limited analyses indicate the potential for underestimation, although of relatively small

5   magnitude (U.S. EPA, 2014, section 6.2.1.4). We further recognize the role of public policy

6   judgments in drawing conclusions regarding adversity of effects to public welfare.

7   In light of the relationships described above, the appropriate level and form combination

8   will depend on the Administrator's objectives for the target level of protection. In articulating

9   these objectives it will be appropriate to evaluate the nature of the O_3 induced effects and their

10   significance or importance to the public welfare, as well as the role that year-to-year exposure

11   variability can play in public welfare impacts. For example, to the extent that the

12   Administrator's priority for public welfare protection is against long-term effects in natural

13   forested ecosystems, and given uncertainty with regard to the extent to which effects from

14   single-year exposures may be judged adverse, articulation of a target level of protection in terms

15   of a longer-term condition, such as through a form that averages the index across multiple years,

16   may be appropriate.

17   •   **What does the available information indicate with regard to protection of welfare**
18   **from cumulative O_3 exposures that might be afforded by alternative secondary**
19   **standards based on the form of the current standard (a 3-year average of 4th high**
20   **8-hour average concentrations)?**

21   In staff consideration of the primary standard in chapter 4, staff preliminarily concludes it

22   is appropriate to consider alternative primary standards of the same form and averaging time as

23   the current primary standard. Thus, although the discussion in this chapter, with regard to the

24   secondary standard, indicates the appropriateness of considering an alternative secondary

25   standard with a cumulative seasonal form, we also recognize that, to the extent that the

26   Administrator may find it more effective to control air quality using the same form for both the

27   primary and secondary standards, it may be practical to consider the extent to which a standard

28   in the form of the primary standard might be expected to also reduce and provide protection from

29   cumulative seasonal exposures of concern. For example, if a clear and robust relationship was

30   found to exist between 8-hour daily peak O_3 concentrations and cumulative, seasonal exposures,

31   the averaging time and form of the current standard might be concluded to have the potential to

32   be effective as a surrogate. Addressing this point, the ISA describes the results of a recent focus

33   study that examined the diel variability in O_3 concentrations in six rural areas between 2007 and

34   2009 (U.S. EPA, 2013, pp. 3-131 to 3-133). The ISA reported that "[t]here was considerable

35   variability in the diel patterns observed in the six rural focus areas" with the three mountainous

36   eastern sites exhibiting a "generally flat profile with little hourly variability in the median

1   concentration and the upper percentiles", while the three western rural areas demonstrated a

2   "clear diel pattern to the hourly O_3 data with a peak in concentration in the afternoon similar to

3   those seen in the urban areas", which was especially obvious at the San Bernardino National

4   Forest site, 90 km east of Los Angeles at an elevation of 1,384 meters (U.S. EPA, 2013, p. 3-

5   132). Thus, while the western sites that are influenced by upwind urban plumes may have

6   increased cumulative seasonal values coincident with increased daily 8-hour peak O_3

7   concentrations, this analysis indicates that, in sites without such an urban influence (the eastern

8   sites in this analysis), such a relationship does not occur (U.S. EPA, 2013, section 3.6.3.2).

9   Thus, the lack of such a relationship indicates that in some locations, O_3 air quality patterns can

10 lead to elevated cumulative, seasonal O_3 exposures without the occurrence of elevated daily

11 maximum 8-hour average O_3 concentrations (U.S. EPA, 2013, section 3.6.3.2). Further staff

12 notes that the prevalence and geographic extent of such locations is unclear, since as in the last

13 review, there continue to be relatively fewer monitors in the West, including in high elevation

14 remote sites. In considering the findings of this analysis, we additionally recognize, however,

15 that in general, the cumulative seasonal values for the eastern rural sites, where cumulative

16 seasonal O_3 concentrations appear to be relatively less related to daily maximum 8-hour

17 concentrations, are lower than those of the western, urban-influenced sites.

18   In addition to the focus study described in the ISA (U.S. EPA, 2013, section 3.6.3.2), we

19 considered analyses of air quality monitoring data and air quality modeling analyses. Chapter 2

20 of this document characterizes recent monitoring data on O_3 air quality in rural areas. While

21 approximately 80% of the O_3 monitoring network is urban focused, about 120 rural monitors are

22 divided among CASTNET, NCore, and POMs sites (Chapter 2, pp. 2-2 to 2-3, Figure 2.1).

23 Specifically, as stated in chapter 2 "[a]lthough rural monitoring sites tend to be less directly

24 affected by anthropogenic pollution sources than urban sites, rural sites can be affected by

25 transport of O_3 or O_3 precursors from upwind urban areas and by local anthropogenic sources

26 such as motor vehicles, power generation, biomass combustion, or oil and gas operations" (U.S.

27 EPA, 2013, section 3.6.2.2). In addition, O_3 tends to persist longer in rural than in urban areas

28 due to lower rates of chemical scavenging in non-urban environments. At higher elevations,

29 increased O_3 concentrations can also result from stratospheric intrusions (U.S. EPA, 2013,

30 sections 3.4, 3.6.2.2). As a result, O_3 concentrations measured in some rural sites can be higher

31 than those measured in nearby urban areas (U.S. EPA, 2013, section 3.6.2.2) and the ISA

32 concludes that "cumulative exposures for humans and vegetation in rural areas can be

33 substantial, and often higher than cumulative exposures in urban areas" (U.S. EPA, 2013, p. 3-

34 120). These known differences between urban and rural sites suggest that there is the potential

35 for an inconsistent relationship between 8-hour daily peak O_3 concentrations and cumulative,

1   seasonal exposures in those areas where protection of O_3-sensitive vegetation may be most
2   needed.

3   In addition, as was done in both the 1997 and 2008 reviews, staff has analyzed
4   relationships between O_3 levels in terms of the current averaging time and form and a W126
5   cumulative form, based on recent air quality data. One analysis describes the W126 index values
6   and current standard design values at each monitor for two periods: 2001-2003 and 2009-2011
7   (e.g., Appendix 2B, Figures 2B-2 and 2B-3). This shows that between the two periods, during
8   which broad scale O_3 precursor emission reductions occurred, O_3 concentrations in terms of both
9   metrics were reduced. There is a fairly strong, positive degree of correlation between the two
10   metrics (Appendix 2B).[3] Focusing only on the latter dataset (2009-2011), it can be seen that at
11   monitors just meeting the current standard (3-year average fourth-highest daily maximum 8-hour
12   average concentration equal to 0.075 ppm), W126 values (in this case 3-year averages) varied
13   from less than 9 ppm-hrs to approximately 20 ppm-hrs (Appendix 2B, Figure 2B-3b). At sites
14   with a 3-year average fourth-highest daily maximum 8-hour average concentration at or below a
15   potential alternative primary standard level of 70 ppb, 3-year W126 index values range from 5 to
16   18 ppm-hrs, with very few above 15 ppm-hrs. An alternative presentation of such data for recent
17   3-year periods back to 2006 – 2008 indicate that among the counties with O_3 concentrations that
18   met the current standard, the number with 3-year W126 index values above 15 ppm-hrs ranges
19   from fewer than 10 to 24 (Appendix 2B, Figure 2B-9). In general during this longer period,
20   W126 index values above 15 ppm-hrs and meeting the current standard were pre-dominantly in
21   Southwest region. As the first analysis in Appendix 2B (for the 2001-2003 and 2009-2011
22   periods) indicates, monitors in the West and Southwest tend to have higher W126 values relative
23   to their design values than do monitors in other regions. This pattern is noteworthy because the
24   Southwest region has a less dense monitoring network than regions in the Eastern US (see Figure
25   2-1), so that the extent to which this pattern occurs throughout these regions is uncertain.
26   Although single-year W126 index values were not separately analyzed in this analysis of the
27   monitor data, it indicates appreciable variation in cumulative, seasonal O_3 concentrations among
28   monitor locations meeting different levels of a standard of the current form.

29   Analyses of the WREA air quality scenarios indicate the potential for O_3 precursor
30   emission reductions achieving O_3 concentrations that just meet different 8-hour standards to
31   produce a significant reduction in 3-year W126 index values. For example, for the current
32   standard scenario, nearly all model-adjusted monitors are at or below an estimated 3-year
33   average W126 value of 15 ppm-hrs (as summarized in section 5.2.2 above and described in U.S.

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[3] Appendix 2B additionally observes that the program implemented for reducing precursor emissions, especially NOx, appears to have been an effective strategy for lowering both design values and W126 values.

1   EPA, 2014, Table 4-1). Those monitors above 15 ppm-hrs would be limited to large urban areas

2   in the southwestern U.S. (i.e., Phoenix, Los Angeles and Denver). When meeting a 4th high 8-

3   hour average scenario of 70 ppb, averaged across 3 years, nearly all monitors in the U.S. would

4   meet a 3-year W126 of 11 ppm-hrs, though some monitors in the southwest would remain

5   between 11 and 15 ppm-hrs. At 65 ppb, all locations are at or below 11 ppm-hrs. Thus, similar

6   to the monitoring analysis, the modeling analysis generally indicates reductions in W126 levels

7   with reduced O_3 concentrations in terms of the current standard averaging time and form. This

8   suggests that depending on the level for a standard of the current averaging time and form, a

9   degree of welfare protection may be afforded. The extent to which such protection provides

10   adequate public welfare protection additionally depends on the level of protection identified by

11   the Administrator for the public welfare in terms of the W126 index. In so noting, however, we

12   recognize the importance of also considering uncertainties in both the model-adjustment analyses

13   and those based on monitoring data. These uncertainties, including those related to monitor

14   coverage, the extent to which recent data can be expected to describe future relationships, and

15   modeling approaches, among others, should be kept in mind when assessing the strength of this

16   apparent relationship.

17   **6.3 LEVEL**

18   In considering potential levels for alternative secondary standards, we have taken into

19   account scientific evidence characterized in the ISA and exposure and risk estimates for different

20   air quality scenarios analyzed in the WREA, as well as the uncertainties and limitations in this

21   information. We consider this information together with regard to support for alternative

22   standards that might be appropriate to consider to provide the requisite protection from adverse

23   effects to public welfare. Drawing from section 6.2 above, our discussion here is primarily in

24   terms of the W126 sigmoidally-weighted metric, cumulated over the 12 daylight hour period

25   (8:00 am to 8:00 pm) for the consecutive three month period with the maximum index value

26   within the growing season. In addition to considering the information in the context of a single

27   growing season, we also consider it in the context of a form for this W126 metric averaged

28   across 3 consecutive growing seasons for reasons discussed in section 6.2 above.

29   In the discussion below, we turn first to consideration of the currently available scientific

30   evidence as assessed and characterized in the ISA. We then consider the WREA findings with

31   regard to vegetation, ecosystem and ecosystem services effects or risks estimated for different air

32   quality scenarios. We additionally take note of important uncertainties and limitations in the

33   evidence and exposure/risk analyses. Lastly, we take note of judgments to be made by the

34   Administrator in drawing conclusions regarding effects and risks that represent adverse

35   environmental effects to public welfare. In so doing, we identify key considerations with regard

1   to the currently available evidence, exposure/risk information and associated uncertainties in

2   identifying levels that may be appropriate to consider for a cumulative seasonal secondary

3   standard. Such levels are described in section 6.5 below which describes preliminary staff

4   conclusions regarding alternative secondary standards appropriate to consider in this review.

5   • **What does the currently available evidence indicate with regard to the range of**
6   **W126-based index values that may provide protection from vegetation effects of**
7   **O_3?**

8   In considering this question in terms of a cumulative seasonal W126-based index, we

9   consider first quantitative evidence for O_3 exposure effects on plant growth, productivity and

10   related endpoints. In so doing, we draw on the C-R functions derived from the evidence on tree

11   seedlings and crops assessed in the ISA which are further described in the WREA and

12   summarized in sections 5.2.1 and 5.3.1 (U.S. EPA 2013, section 9.6; U.S. EPA, 2014, section

13   6.2). It is important to note that these functions are used to provide estimates of growth and yield

14   reduction in tree seedlings and crops that might be expected to result from exposure over a single

15   growing season to various O_3 concentrations expressed in terms of a W126 index (Figure 5-1

16   above).

17   Table 6-1 below presents estimates of relative yield and biomass loss for the studied

18   species of crops and tree seedlings, respectively, for a growing season exposure to a number of

19   W126 levels. In this table, we have included observations related to 2% relative biomass loss in

20   tree seedlings and 5% relative crop yield loss. These values are consistent with advice from

21   CASAC thus far in this review (Frey and Samet, 2012) on factors on which to base consideration

22   of levels for a secondary standard (see section 6.4 below), and with values given focus in the

23   1996 consensus-building workshop, described below. The observations in Table 6-1 for each

24   W126 index value include relative biomass or crop yield loss estimates for the median across the

25   studied species, as well as the proportion of species with estimates below 2%, 5% or 10%, as

26   examples of benchmarks that may be of interest in different contexts.

27   Staff recognizes Table 6-1 as a useful way to consider the magnitude of biomass and crop

28   yield loss estimates across the 12 and 10 studied species, respectively, for the purposes of

29   informing consideration of potential levels for alternative secondary standards based on the

30   W126 index. The different index levels can be considered with regard to the magnitude of

31   median species response and the proportion of species with responses expected to be below the

32   various benchmarks. For example, at an O_3 exposure equivalent to a W126 index value of 7

33   ppm-hrs, the median tree seedling response across the 12 studied species is projected to be near

34   or below 2% reduction in growth compared to that for the control plants exposed to W126 index

35   values of zero (Table 6-1; U.S. EPA, 2014, section 6.2, Appendix 6A). Seven of the 12 species

36   are projected to have smaller than 2% reduction in growth, while three other species are

1   projected to have a reduction between 2 and 5%, for a total of 10 of 12 species projected to have

2   less than 5% reduction in growth, at a W126 index value of 7 ppm-hrs. The remaining two

3   species, black cherry and cottonwood, are projected to have reductions just over 15% and 40%,

4   respectively, at 7 ppm-hrs and above (Table 6-1). The median growth reduction response

5   projected for a W126 index value of 15 ppm-hrs is approximately 5%, with responses less than

6   2% projected for five of the twelve studied species and less than 5% for six, while less than 10%

7   reduction in growth is projected for ten of the twelve species. Somewhat similar estimates are

8   projected for a W126 index value of 17 ppm-hrs, with the median growth reduction response

9   projected to be 6%, and also with five of the twelve species less than 2%, nine less than 10% and

10   a tenth just over 10% (Table 6-1). Crop yield loss estimates are also described in Table 6-1.

11

1 **Table 6-1. Seedling and crop growth or yield reductions estimated for O$_3$ exposure over a**
2 **season.**

W126 value for exposure period	Tree seedling growth reduction (biomass loss)[A]		Crop yield loss[A]
21 ppm-hrs	[B]Median species w. 7.7 % loss (varying lower) 3/12 species w. $\leq$ ~2%[C] loss; 5/12 w. $\leq$ 5% loss 7/12 species w. <10% loss; 10/12 <15% loss		Median species[D] = 7.7 % loss 4/10 species w. $\leq$ 5% loss 3/10 species w. >5,<10% loss 3/10 species w. >10,<20% loss[E]
19 ppm-hrs	[B]Median species w. 6.8 % loss (varying lower) 3/12 species w. $\leq$ ~2% loss; 5/12 w. <5% loss 7/12 w. <10% loss; 10/12 w. <15% loss	Note: Most sensitive species are cottonwood (relative biomass loss estimates $\geq$ 40% for W126$\geq$7) and black cherry (relative biomass loss estimates $\geq$15% for W126 $\geq$ 7).	Median species[D] = 6.4 % loss 5/10 species w. $\leq$ 5% loss 3/10 species w. >5, <10% loss 2/10 species w. >10,<20% loss
17 ppm-hrs	[B]Median species w. 6.0 % loss (varying lower) 5/12 species w. $\leq$ ~2% loss; 5/12 w. <5% loss 9/12 w. <10% loss; 10/12 w. <15% loss		Median species[D] = 5.1 % loss 5/10 species w. $\leq$ 5% loss 3/10 species w. >5, <10% loss 2/10 species w. >10,<20% loss
15 ppm-hrs	[B]Median species w. 5.2 % loss (varying lower) 5/12 species w. $\leq$ ~2% loss; 6/12 w. <5% loss 10/12 species w. <10% loss		Median species[D] $\leq$5% loss 6/10 species w. $\leq$ 5% loss 4/10 species w. >5, <10% loss [F]
13 ppm-hrs	[B]Median species w. 4.4% loss (varying lower) 5/12 species w. $\leq$ ~2% loss; 7/12 w. <5% loss 10/12 species w.<10% loss		Median species[D] $\leq$5% loss 7/10 species w. $\leq$ 5% loss 3/10 species w. >5, <10% loss
11 ppm-hrs	[B]Median species w. 3.5% loss (varying lower) 5/12 species w. $\leq$ ~2% loss; 8/12 w. <5% loss 10/12 species w. <10% loss		Median species[D] $\leq$5% loss 9/10 species w. $\leq$ 5% loss 1/10 species w. >5, <10% loss [g]
9 ppm-hrs	[B]Median species w. 2.5% loss (varying lower) 5/12 species w. $\leq$ ~2% loss; 10/12 w. <5% loss		Median species[D] $\leq$5% loss All species $\leq$ 5% loss
7 ppm-hrs	[B]Median species w. $\leq$2% loss (varying lower) 7/12 species w. $\leq$ ~2% loss; 10/12 w. <5% loss		Median species[D] $\leq$5% loss All species $\leq$ 5% loss

[A] Estimates here are based on the C-R functions described in WREA, section 6.2 and discussed in section 5.2.1
[B] This median value is the composite median from WREA, Figure 6-4 (also discussed in section 5.2.1). The note in parentheses refers to the median of the stochastic sampling estimate shown in WREA Figure 6-4 (and Figure 5-2 in this document).
[C] In making comparisons, biomass loss estimates described as $\leq$ 2 refer to values $\leq$ 2.04.
[D] This median is the composite median from WREA, Figure 6-5 (also discussed in section 5.2.1).
[E] Kidney bean , potato, and winter wheat >15%.
[F] Cotton, kidney bean, potato, & soybean >5%.
[G] Kidney bean at 5.7% loss.

3

4   With respect to W126 index values associated with different degrees of protection for
5 visible foliar injury, we first recognize the value of this endpoint in its long-standing use as a
6 well established bioindicator of O$_3$ exposure, as described in the ISA (U.S. EPA 2013, section
7 9.4.2).

1   In addition to the role of visible foliar injury as an indicator, we note that the aesthetic
2   aspects of visible foliar injury itself have the potential to be important to public welfare (as
3   described in section 5.4 above). Analyses such as those presented in the WREA, and
4   summarized in section 5.4.2 above, provide approaches by which foliar injury observations
5   under existing air quality may be analyzed. However, there is very little information on which to
6   base judgments of the prevalence and/or severity of injury that might be considered adverse. We
7   further recognize that there is substantial variability such that "the degree and extent of visible
8   foliar injury development varies from year to year and site to site … even among co-members of
9   a population exposed to similar O_3 levels, due to the influence of co-occurring environmental
10   and genetic factors" (U.S. EPA, 2013, p. 9-38). Thus, based on consideration of the evidence,
11   staff recognizes the lack of a consistent or generally predictable relationship between particular
12   W126 exposures and visible foliar injury incidence, given the influence of and variability in
13   relative soil moisture and other environmental factors. We additionally note uncertainty in what
14   can be concluded from foliar injury in relation to plant health, productivity and ecological
15   function as "it is not presently possible to determine, with consistency across species and
16   environments, what degree of injury at the leaf level has significance to the vigor of the whole
17   plant" (U.S. EPA, 2013, p. 9-39). Thus, on the basis of the evidence, we are not able to identify
18   a range of appropriate W126 index values.
19   In further considering the available information pertaining to the question above, we
20   additionally recognize conclusions that have been drawn by expert committees with regard to
21   these endpoints (i.e., tree seedling growth, crop yields and visible foliar injury). For example, in
22   their review of staff documents during the O_3 NAAQS review completed in 1997 review, the
23   CASAC O_3 panel members expressed a wide range of opinions on aspects of the evidence
24   important to consider in judging the adequacy of the O_3 secondary standard and in considering
25   the form and level that would be appropriate for a secondary O_3 standard (Wolff, 1996).
26   Subsequent to CASAC meetings in 1995 on this topic, a consensus-building workshop sponsored
27   by the Southern Oxidant Study group was held on the topic of the O_3 secondary standard in
28   January 1996 (Heck and Cowling, 1997). This workshop was attended by 16 scientists with
29   backgrounds in agricultural, managed forest, natural systems, and air quality, all of whom were
30   leaders in their fields and whose research formed the basis of much of the research examined in
31   the 1996 Criteria Document. These scientists expressed their judgments on what standard
32   level(s) would provide vegetation with protection from O_3-related adverse effects that would be
33   adequate, in their view.[4,5] As the 1997 workshop publication indicates, the scientists at the 1996

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[4] At the time of the workshop, the secondary O_3 standard being reviewed by EPA was a 1-hour average of
0.08 ppm (identical to the primary standard at that time). In 1997, EPA concluded the review by revising both
standards to a longer averaging time of 8 hours with a level of 0.08 ppm (62 FR 38856).

1 workshop also reached consensus views regarding the types of exposures that were important in
2 eliciting plant response and the types of metrics that were best at predicting these responses
3 (Heck and Cowling, 1997). Before coming to agreement on daily and seasonal durations and
4 forms pertinent to a distinct secondary standard, the participants discussed and identified
5 endpoints to consider for natural, forest and agricultural ecosystems.[6] With regard to form of the
6 standard, participants concurred with either the SUM06 or W126 metrics, with consensus finally
7 reached for SUM06, with some qualification regarding implications for a threshold. The
8 participants identified the ranges they felt should be considered for each of three endpoints.
9 Overall, the SUM06 values ranged from 8 to 20 ppm-hr (corresponding to W126 values ranging
10 from 5 to 17 ppm-hr, based on EPA analysis focused on conditions in NCLAN studies).[7] This
11 overall range reflected ranges for each of the three endpoints, with the following considerations
12 (Heck and Cowling, 1997).

13   – Crops (yield reductions): SUM06 of 15-20 ppm-hrs (13-17 ppm-hrs, W126). This
14   range was recognized to generally consider <10% yield loss in more than 75% of
15   species.

16   – Trees (growth effects): SUM06 of 10-16 ppm-hrs (7 to 14 ppm-hrs, W126). This
17   range was recognized to generally consider 1-2% per year growth reduction; in so
18   doing,the group identified a need to consider the potential for year-to-year
19   compounding of impacts in long-lived perennial species.

20   – Visible Foliar Injury: SUM06 of 8 to 12 ppm-hrs (5 to 9 ppm-hrs, W126).

21   Since the publication of 1996 workshop report and conclusion of the 1997 NAAQS
22 review, the evidence base has continued to expand as described in the 2006 CD and 2013 ISA
23 (U.S. EPA, 2006; U.S. EPA, 2013). With regard to tree growth effects and crop yield reductions,
24 results of additional studies conducted in the field have confirmed the tree seedling biomass loss
25 and crop yield loss concentration-response relationships derived from earlier studies that used
26 open top chambers (U.S. EPA 2013, section 9.6). Further, similar data, although from an

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[5] The workshop publication describes the primary objective for the workshop as having been to assemble knowledgeable scientists to develop a group consensus on "various critical components associated with a possible revised secondary ozone standard" (Heck and Cowling, 1997).

[6] For natural ecosystems, they focused on foliar injury as an indicator. For forest ecosystems, concluded the data did not support selection of an indicator of effects on forest structure or function. As a result, they identified two indicators pertinent to the systems: growth effects on seedlings from species of natural forest stands (1-2% per year reduction), and growth effects on seedlings and saplings from tree plantations (1-2% per year reduction). For agricultural systems, the participants focused on protection against crop yield reductions, with their acknowledgment of high uncertainties at 5% leading them to a crop yield endpoint of 10% yield reduction (Heck and Cowling, 1997).

[7] During the last review, W126 index values corresponding to the SUM06 values cited in the report were estimated using the NCLAN crop loss data, a key dataset considered by workshop participants (see Appendix 7B of 2007 Staff Paper; Appendix 6A of this document).

1   ambient gradient study (outside New York City) rather than a controlled experiment, are

2   available for an additional, apparently quite sensitive species, the cottonwood (Gregg, 2003).

3   In the 2008 review, CASAC provided comments related to a cumulative seasonal

4   secondary standard in the context of their comments on the draft and final Staff Papers and on

5   the final decision (Henderson, 2006; Henderson, 2007; Henderson, 2008). In all instances, they

6   conveyed support for establishment of a distinct secondary standard with a cumulative seasonal

7   form. While the EPA, in the 2007 Staff Paper and 2007 notice of proposed rulemaking,

8   recognized a broader range of W126 values as appropriate for consideration with regard to a

9   distinct secondary standard, the CASAC Panel focused on a range they described as

10 approximately equivalent to that identified by the 1996 workshop participants (Henderson, 2007,

11 pp. 3, C-27).[8] In the CASAC Panel 2006-2007 advice on levels for such a standard, their

12 suggestion was a focus on levels for a W126 index approximately equivalent to a SUM06 range

13 of 10 to 20 ppm-hr (Henderson, 2006, 2007, 2008), which they estimated in 2007 to be a range

14 from 7 (or 7.5) to 15 ppm-hrs. Based on their consideration of the information available in that

15 review (e.g., with regard to potential magnitude of effects across multiple years), the CASAC

16 Panel further advised that "[i]f multi-year averaging is employed to increase the stability of the

17 secondary standard, the level of the standard should be revised downward to assure that the

18 desired threshold is not exceeded in individual years" (Henderson, 2007, p. 3). The CASAC

19 advice provided on the 2010 proposed reconsideration and thus far in this review is summarized

20 in section 6.4 below.

21   In considering the evidence briefly summarized above in the context of levels for a

22 W126-based standard, we recognize that given the different types of O_3-induced effects, genetic

23 variability within and between species, and environmental modifiers of effects that also

24 contribute to variability, it is not feasible to identify a range of cumulative seasonal exposures

25 from the vegetation effects evidence which would provide a consistent degree of protection for

26 all species. In so doing, we note the importance of considering this evidence in several

27 dimensions that pertain to judgments required by the Administrator regarding public welfare

28 significance. For example, we take note of the usefulness of considering the cumulative seasonal

29 exposure at which the median species response or the majority of the species' responses are

30 expected to be below minimal response benchmarks of interest and at which only a very few

31 species' responses are expected to exceed more substantial response benchmarks. Before

32 articulating such considerations with regard to specific benchmarks and index values, we first

33 consider the WREA findings in the context of the following question.

---

[8] Appendix C of the March 26, 2007 CASAC letter used a 2001 ambient concentration dataset and other factors, rather than study data considered in the 1996 workshop, in estimating an "equivalency" between the two indices.

1   • **What are the nature and magnitude of risks to vegetation estimated for the**
2   **average W126 index scenarios evaluated in the WREA, and what is the**
3   **magnitude of risk reduction from risks estimated for air quality conditions**
4   **estimated for the current standard?**

5   In considering the WREA estimates here we take note of uncertainties in the extent to

6   which the results for each modeled air quality scenario represent cumulative seasonal O_3

7   exposures that would be expected to occur across the three years represented in each scenario. In

8   general, each scenario is represented by a dataset of 3-year average W126 index values across

9   the national modeling area. Thus, the results estimated for the various analyses performed do not

10   reflect any year-to-year variability that would be expected in single year results. Rather, they

11   reflect average estimates for the three year period modeled. Such estimates do not account for

12   the potential for compounding of effects in perennial species that has been discussed previously.

13   Limited analyses in the WREA describe the potential for the WREA estimates to underestimate

14   cumulative biomass-related effects in perennial species (as noted in sections 6.2 and 5.2.2 above

15   and described in detail in U.S. EPA, 2014, chapter 6, 6.2.1.4). This potential for underestimation

16   is recognized in the context of the uncertainties associated with other aspects of the different

17   analyses in section 6.9 of the WREA (e.g., U.S. EPA, 2014, Table 6-27). We additionally note

18   that the limited WREA compounding analyses do not take into account other variables which can

19   affect the magnitude of these effects in the field.

20   In considering the question posed above, we focus particularly on WREA estimates

21   related to O_3 effects on plant biomass and associated ecosystem services effects. In so doing, we

22   note the relationships among effects on individual plants to other ecosystem components and

23   functions, such as carbon sequestration and air pollutant removal (U.S. EPA 2013, section

24   9.4.3.4; U.S. EPA, 2014, sections 6.6 and 6.7), as well as market responses to changes in timber

25   and agricultural production (U.S. EPA, 2014, sections 6.3 and 6.5). We additionally recognize

26   other biomass-related responses, such as non-timber forest products, and other O_3-attributable

27   ecosystem responses for which we have primarily qualitative characterizations of impacts (U.S.

28   EPA, 2014, chapter 5).

29   We turn first to the WREA estimates for a range of effects related to biomass loss, which

30   are based on application of the C-R functions for seedlings of 12 tree species described in the

31   ISA (U.S. EPA, 2013, section 9.6.2) and the WREA (U.S. EPA, 2014, section 6.2). First we

32   note (as considered above) the range of responses for the individual species for which C-R

33   functions have been developed. These twelve species vary appreciably in sensitivity of growth

34   reduction (in terms of relative biomass loss, or RBL) in response to O_3 exposure. For example,

35   2% seedling biomass loss is estimated to occur following exposure represented by W126 index

36   values below 10 ppm-hours for seven of the studied species and at or above approximately 20

1   ppm-hours in the other five studied species (Figure 5-1 above). The WREA presents three

2   approaches for characterizing a median response across the species (Figure 5-2; U.S. EPA 2014,

3   section 6.2.1.2). Across the three approaches, the median seasonal W126 index value for which

4   a 2% biomass loss is estimated in seedlings for the studied species ranges between approximately

5   7 and 14 ppm-hrs (see description in section 5.2.1 above).

6   We additionally consider the WREA estimates of overall ecosystem-level effects from

7   biomass loss considering the 12 studied species together (U.S. EPA 2014, section 6.8). The

8   WREA analysis used the species-specific biomass loss C-R functions, information on prevalence

9   of the studied species across the U.S., and a weighting approach based on proportion of the basal

10   area within each grid cell that each species contributes. A weighted RBL value for each grid cell

11   is generated by weighting the RBL value for each studied tree species found within that grid cell

12   by the proportion of basal area it contributes to the total basal area of tree species within the grid

13   cell, and then summing those individual weighted RBLs. Based on the average W126 values

14   estimated for the air quality scenario just meeting the current standard across the contiguous

15   U.S., the WREA estimates 0.8 percent of the total geographic area to have a weighted relative

16   biomass loss above 2% (Table 6-2 below; U.S. EPA 2014, Table 6-24). In the W126 air quality

17   scenarios for 15, 11, and 7 ppm-hrs (average across three years), the percent of total area having

18   weighted relative biomass loss greater than two percent was 0.7 percent, 0.5 percent and 0.2

19   percent, respectively (Table 6-2 below; U.S. EPA 2014, Table 6-25). In considering these

20   estimates, we note that the values for percentages of basal area include many grid cells in which

21   none of the 12 studied species are found. Thus, since only 64 percent of grid cells contained one

22   or more of the 12 species, these values are likely to be low and illustrate the potential

23   uncertainties associated with the limited number studied tree species for which C-R functions

24   exist (U.S. EPA, 2014, section 6.8).

25

1 **Table 6-2. Percent of assessed geographic area exceeding 2% weighted relative biomass**
2 **loss in WREA air quality scenarios.**

	----------------Air Quality Scenarios ---------------------------			
		W126 index scenarios[B]		
	Conditions just meeting the current standard[A]	15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
Percent of total area with wRBL>2%	0.8 %	0.7 %	0.5 %	0.2 %

[A] This analysis uses air quality values that are estimated per model grid cell using the W126 value assigned to the grid cell based on application of the VNA method to the monitor-location W126 values that are the average at that location across the 3 years of W126 values for the model-adjusted dataset that just meets the current standard (fourth-highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb).

[B] The national distribution of W126 values within model grid-cells for each scenario reflects model adjustment of 2006-2008 O_3 concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA, 2014, section 4.3.4.1 and Appendix 4A).

3
4 We next consider the wRBL estimates from the WREA analysis of 145 (of the 155)
5 federally-designated Class I areas for which there was sufficient information regarding O_3-
6 sensitive species (U.S. EPA, 2014, section 6.8.1, Table 6-26, Appendix 6E). These 145 parks
7 had at least one O_3-sensitive tree species for which a C-R function for RBL was available. Using
8 the C-R functions for the species found within each park, the WREA calculated an average
9 wRBL value for each park for the 3-year average W126 index values estimated in those locations
10 for the current standard and three W126 air quality scenarios. Under conditions model-adjusted
11 to just meet the current standard, the average wRBL in 2 of the 145 parks is estimated to be
12 above 2%, as presented in Table 6-3 below. We compare this estimate to those for the W126
13 scenarios. For the W126 scenarios of 15 and 11 ppm-hrs, the estimated weighted RBL is greater
14 than 2% in two of the 145 parks, while it is great than 2% in only 1 park for the 7 ppm-hrs
15 scenario.
16
17

1 **Table 6-3. Number of Class I areas (of 145 assessed) with weighted relative biomass loss**
2 **greater than 2%.**

	Air Quality Scenarios			
	Conditions just meeting the current standard[A]	3-Year Average W126 index scenarios[B]		
		15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
Number of Class I areas with wRBL>2%	2	2	2	1

[A] The wRBL is estimated per model grid cell (in which there are any of the 12 studied species) from W126 value assigned to the grid cell based on application of the VNA method to the monitor-location W126 values that are the average at that location across the 3 years of W126 values for the model-adjusted dataset that just meets the current standard (fourth-highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb).

[B] The national distribution of W126 values within model grid-cells for each scenario reflects model adjustment of 2006-2008 O_3 concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA, 2014, section 4.3.4.1 and Appendix 4A).

3
4   With respect to crops, based on the C-R functions described in the ISA and additionally
5 analyzed in the WREA, a 5% median relative yield loss (RYL) for all studied crop cases occurs
6 within the range of W126 index values from 12-17 ppm-hrs (U.S. EPA 2014, Figure 6-5). The
7 species-specific C-R functions project less than 5% yield loss for 6 out of 10 species for O_3
8 exposure equivalent to a seasonal W126 index value of 15 ppm-hrs and for all of the 10 studied
9 species after an exposure equivalent to a seasonal W126 index of 7 ppm-hrs (Table 6-1 above,
10 U.S. EPA 2014, section 6.2 and Appendix 6A). For soybeans, less than 5% yield loss was
11 estimated for the W126 index value of 12 ppm-hrs (U.S. EPA 2014, Figure 6-3). The WREA
12 estimates of crop yield loss for the modeled air quality scenarios are summarized in Table 6-4
13 below (details are provided in U.S. EPA 2014, section 6.5.1 and Appendix 6B). For the recent
14 air quality conditions scenario, the means for all crops were less than 5% loss across all states.
15 Crop yield loss estimates for all states were also less than 5% in the air quality scenario
16 representing conditions just meeting the current standard (U.S. EPA, 2014, section 6.5.1 and
17 Appendix 6B).
18

Table 6-4. Estimated mean yield loss (and range across states) due to O$_3$ exposure for two important crops.

Crop	Recent Conditions (2006-2008)	Conditions just meeting the current standard[A]	Average W126 index scenarios[B]		
			15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
Corn	<5% [C] (0.01-0.88)	<5% (0.0-0.01)	<5% (0.0-0.01)	<5% (0.0 – 0.0)	<5% (0.0 – 0.0)
Soybean	<5% (0.69-8.30)	<5% (0.01 – 1.39)	<5% (0.01 – 1.13)	<5% (0.01 – 0.75)	<5% (0.01 – 0.59)

[A] The crop yield loss is estimated per grid cell (and per FASOMGHG region) from W126 value assigned to the cell based on application of the VNA method to the monitor-location W126 values that are the average at that location across the 3 years of W126 values for the model-adjusted dataset that just meets the current standard (fourth-highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb).

[B] The national distribution of W126 values within grid cells for each scenario reflects model adjustment of 2006-2008 O$_3$ concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA 2014 section 4.3.4.1 and Appendix 4A).

[C] Mean yield loss is the mean across modeling units. The range presented in parentheses below the mean represents the minimum and maximum estimates across modeling units (U.S. EPA 2014, Appendix 6B).

As explained above, however, comparisons of the WREA's air quality scenarios for the national-scale estimates of timber production and consumer and producer surpluses are not straightforward to interpret due to market dynamics. Estimates for the recent conditions and current standard scenarios are compared to the three W126 scenarios. In general, substantially greater economic surpluses (approximately 51 billion in terms of 2010 dollars) are estimated from the comparison of the recent conditions (2006-2008) scenario to the current standard scenario. The vast majority of these economic surpluses are estimated for agricultural production. Differences of the average W126 scenarios from the current standard scenario are much smaller (U.S. EPA 2014, Appendix 6B).

Because increases in timber production represent increased tree growth and concurrent carbon sequestration, we also consider WREA estimates of the potential increase in carbon storage that potentially could occur for different air quality scenarios (U.S. EPA 2014, section 6.6.1). Comparisons of the W126 scenarios to the current standard scenario with regard to carbon sequestration estimates do not indicate an appreciable difference for the W126 scenario of 15 ppm-hrs. The majority of the enhanced carbon sequestration potential in the forest biomass increases over time, for alternatives, is predicted to occur for the W126 scenarios of 11 and 7 ppm-hrs. Over 30 years, the current standard scenario projection is 89,184 million metric tons of CO$_2$ equivalents (MMtCO$_2$e). The WREA estimates additional sequestration potential of 13, 593

1   and 1,600 MMtCO$_2$e, for the W126 scenarios of 15, 11 and 7 ppm-hrs, respectively, as compared

2   to the current standard baseline (U.S. EPA 2014, Table 6-18)[9]. We also take note of the

3   relatively smaller estimates for carbon sequestration associated with improved crop yields (over

4   30 years) in the agricultural sector, which indicate little difference among the different W126

5   scenarios.

6   We additionally consider the WREA estimates for five urban areas of how reduced

7   growth of O$_3$-sensitive trees in urban forests may affect the ecosystem services of air pollutant

8   removal and carbon sequestration (U.S. EPA, 2014, sections 6.6.2 and 6.7 and Appendix 6D).

9   With regard to air pollutant removal, the WREA estimated metric tons of carbon monoxide,

10   nitrogen dioxide, ozone and sulfur dioxide removed under the modeled air quality scenarios. In

11   considering these estimates we note the general assumptions made to estimate order of

12   magnitude effects of O$_3$ removal by trees on O$_3$ concentrations in the five urban areas and the

13   associated uncertainties (U.S. EPA 2014, sections 6.7 and 6.9 and Appendix 6D). Estimates for

14   all five areas indicate increased pollutant removal for air quality model-adjusted from recent

15   conditions to just meet the current standard, with much smaller difference where they exist

16   between the current standard and three W126 scenarios (Table 6-5 below). With respect to

17   carbon sequestration, relative to the scenario representing just meeting the current standard,

18   again the largest difference is generally observed with the existing conditions scenario (Table 6-

19   5). This is because of the largest difference in W126 estimates occurring between these two

20   scenarios. In addition to the small differences in W126 index values among the current standard

21   and W126 scenarios for these five areas, which contribute to the similarities among scenarios, we

22   also note that as only 2 or 3 tree species were able to be assessed in each city, these results may

23   underestimate the overall impacts nationally, although other areas of uncertainty (recognized

24   below) may tend to contribute to the opposite potential (U.S. EPA 2014, Table 6-27).

25

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[9] 1 million metric tons of carbon dioxide equivalents (MMtCO$_2$e) is equivalent to 208,000 passenger vehicles or the electricity to run 138,000 homes for 1 year as calculated by the EPA Greenhouse Gas Equivalencies Calculator (updated September 2013and available at http://www.epa.gov/cleanenergy/energy-resources/calculator.html).

Table 6-5. Estimated effect of O$_3$-sensitive tree growth-related impacts on the ecosystem services of air pollutant removal and carbon sequestration in five urban case study areas.

Case Study Area	\-\-\- Air Quality Scenarios \-\-\-				
	Recent Conditions (2006-2008)	Conditions just meeting the current standard[A]	Average W126 index scenarios[B]		
			15 ppm-hrs	15 ppm-hrs	15 ppm-hrs
	Air Pollutant Removal (metric tons, CO, NO$_2$, O$_3$, SO$_2$)				
Atlanta	33,000	35,800	35,800	36,000	36,300
Baltimore	8,500	9,200	9,200	9,200	9,200
Chicago	355,000	359,000	359,000	361,000	365,000
Syracuse	1,500	1,700	1,700	1,700	1,700
Tennessee urban	474,000	511,000	511,000	516,000	522,000
	Million Metric Tons of CO$_2$ Equivalents, Carbon Storage (cumulative over 25 years)				
Atlanta	1.2	1.32	1.32	1.32	1.34
Baltimore	0.5	0.57	0.57	0.57	0.57
Chicago	16.9	17.05	17.05	17.10	17.21
Syracuse	0.14	0.17	0.17	0.17	0.17
Tennessee urban	18.0	19.67	19.67	19.89	20.16

[A] Results are derived from estimates per model grid cell (in which there are any of the 12 studied species) from W126 value assigned to the grid cell based on application of the VNA method to the monitor-location W126 values that are the average at that location across the 3 years of W126 values for the model-adjusted dataset that just meets the current standard (fourth-highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb).

[B] The national distribution of W126 values within model grid-cells for each scenario reflects model adjustment of 2006-2008 O$_3$ concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA, 2014, section 4.3.4.1 and Appendix 4A).

With regard to foliar injury, we take note of the WREA analyses of the nationwide dataset (2006- 2010) for U.S. Forest Service biosites described in section 5.4.2 above, including the presentation indicating that the proportion of biosites with injury varies with soil moisture conditions and O$_3$ W126 index values, and that the proportion of biosites with injury severity greater than 5 also varied with soil moisture (U.S. EPA 2014, Chapter 7, Figure 7-11). The evidence of O$_3$-attributable visible foliar injury incidence occurring in USFS biosites shows that the proportion of biosites showing foliar injury incidence increases steeply with W126 index values up to approximately10 ppm-hrs. At W126 index levels greater than approximately 10 ppm-hrs the proportion of sites showing foliar injury incidence is relatively constant.

In reflecting across the range of W126 estimates identified in various WREA analyses, we first note the substantial benefits and reductions in biomass-related risks estimated for air

1 quality adjusted to just meet the current standard. Additional incremental benefits or risk

2 reduction, generally of relatively smaller magnitude, is estimated across the W126 scenarios. In

3 considering this information discussed above in the context of identifying levels appropriate to

4 consider for a W126-based standard, we first take note of associated uncertainties in the context

5 of the following question.

6   • **What are important uncertainties and limitations in the evidence and**
7   **exposure/risk analyses?**

8   In considering the evidence and exposure/risk information summarized above and the

9 weight to place on this information, we are mindful of the uncertainties and limitations

10 associated with several key aspects of this information. We first consider the uncertainties

11 associated with the evidence underlying the tree seedling and crop C-R functions, given the

12 importance of these functions for many of the ecosystem service analyses described in the

13 WREA. Several key uncertainties associated with this information are listed here.

14   • Uncertainty regarding the extent to which the subset of studied tree and crop species
15   encompass the total number of O_3 sensitive species in the nation and to what extent it
16   is representative of U.S. vegetation as a whole, given that information is available for
17   only a small fraction of the number of total species of trees and crops grown in the
18   U.S. (U.S. EPA, 2013, section 9.6, U.S. EPA, 2014, Table 6-27).

19   • Uncertainties regarding intra-species variability due to the different numbers of studies
20   that exist for different species so that the weight of evidence is not the same for each
21   species. Those species with more than one study show variability in response and C-R
22   functions. The potential variability in less well studied species is therefore unknown
23   (U.S. EPA, 2013, pp. 9-123/125, U.S. EPA, 2014, section 6.2.1.2, and Table 6-27).

24   • Uncertainty regarding the extent to which tree seedling C-R functions can be used to
25   represent mature trees since seedling sensitivity has been shown in some cases to not
26   reflect mature tree O_3 sensitivity in the same species (U.S. EPA, 2013, section 9.6,
27   U.S. EPA, 2014, section 6.2.1.1 and Tables 6-5 and 6-27).

28   • Uncertainty in the relationship of O_3 effects on tree seedlings (e.g., relative biomass
29   loss) in one or a few growing seasons to effects that might be expected to accrue over
30   the life of the trees extending into adulthood (U.S. EPA, 2013, pp. 9-52/53, U.S.
31   EPA, 2014, section 6.2.1.4 and Table 6-27).

32   • Uncertainties associated with estimating the national scale ecosystem-level impacts
33   using weighted relative biomass loss (U.S. EPA, 2014, section 6.8, and Table 6-27)

34   • Uncertainties associated with potential biomass loss in federally designated Class I
35   areas (U.S. EPA, 2014, section 6.8. and Table 6-27)

36   Turning to consideration of the air quality conditions estimated for the various air quality

37 scenarios, we take note of the following uncertainties associated particularly with estimates of O_3

38 exposures in rural areas nationally. These are described more completely in chapter 4 of the

1   WREA (see for example, U.S. EPA, 2014, section 4.4) and summarized in chapter 8 of the REA

2   (U.S. EPA, 2014, section 8.5).

3   • Uncertainties in O_3 exposures due to a lack of rural monitors, especially in the western
4   U.S. and at high elevation sites.

5   • Uncertainties associated with the method (VNA) used to interpolate monitor values to
6   estimate W126 index values in locations without monitors.

7   • Uncertainties in model-adjusted estimates of O_3 concentrations associated with
8   meeting the current standard and potential alternative W126-based standards.

9   Numerous ecosystem services assessments were described in the WREA. These

10 assessments relied heavily on models, which also relied on the inputs of the seedling and crop C-

11 R functions and model-adjusted air quality estimates. Thus, including the uncertainties from the

12 first two categories discussed above, additional uncertainties associated with the ecosystem

13 services models include the following.

14 • Uncertainties associated with use of the iTree model to estimate pollution removal and
15   carbon storage in 5 urban area case studies, including uncertainties in the base
16   inventory of city trees, the functions used for air pollution removal and carbon storage
17   (U.S. EPA, 2014, sections 6.6.2, 6.7, and Table 6-27).

18 • Uncertainties associated with use of the FASOMGHG model for national timber and
19   crop production, including use of median C-R functions for crops in FASOM and
20   crop proxy and forest type assumptions to fill in where there was insufficient data
21   (U.S. EPA, 2014, sections 6.3, 6.5, 6.6.1, and Table 6-27).

22 • Uncertainties associated with use of the FASOMGHG model to estimate national scale
23   carbon sequestration, including those associated with the functions for carbon
24   sequestration (U.S. EPA, 2014, sections 6.2.1.1, 6.6.1, and Table 6-27).

25 In addition to biomass loss and crop yield loss, the WREA estimates the incidence and/or

26 severity of O_3-induced visible foliar injury, both at the national and national park scales.

27 Numerous uncertainties are associated with these assessments and include the following.

28 • Uncertainties associated with our understanding of the number and sensitivity of O_3
29   sensitive species (U.S. EPA, 2014, sections 7.2.1, 7.5 and Table 7-22).

30 • Uncertainties associated with spatial assignment of foliar injury biosite data to 12x12
31   km grids (U.S. EPA, 2014, sections 7.2.1, 7.5 and Table 7-22).

32 • Uncertainties associated with availability of biosite sampling data in some locations in
33   the western U.S. (U.S. EPA, 2014, sections 7.2.1, 7.5 and Table 7-22).

34 • Uncertainties associated with soil moisture threshold for foliar injury (U.S. EPA,
35   2014, sections 7.2.2, 7.2.3, 7.5 and Table 7-22).

- Uncertainties associated with spatial resolution of soil moisture data, time period for soil moisture data, drought categories and the combination of soil moisture and biosite data (U.S. EPA, 2014, sections 7.3.3.2, 7.5 and Table 7-22).

- Uncertainties associated with O_3 exposure data of vegetation and recreational areas within parks (U.S. EPA, 2014, sections 7.4, 7.5 and Table 7-22).

- Uncertainties associated with surveys of recreational activities (U.S. EPA, 2014, sections 7.1.1.2, 7.5 and Table 7-22).

Additionally, there is uncertainty associated with the extent to which the endpoints and associated risk estimates considered above represent effects reasonably judged adverse in the context of public welfare. All of these uncertainties are important to considerations below in the context of target levels of protection with regard to weight to be placed on various lines of evidence and assessment results.

- **What considerations may be important to the Administrator's judgments on the public welfare significance of O_3 associated vegetation effects that may be expected under air quality conditions associated with different levels for a seasonal cumulative standard?**

Our consideration of this question is intended to provide a public welfare context for consideration of the evidence and exposure/risk information discussed above, which includes the nature and magnitude of observed and predicted effects at various levels of cumulative seasonal exposures. We also note the importance of considering information in an integrated manner, rather than focusing only on results from any one analysis. For example, we find it appropriate, in considering the evidence with regard to seedling growth reduction (or biomass loss), to consider the WREA estimates of affected area based on tree basal area together with estimates of individual species responses based simply on the evidence-based C-R functions, and in light of other potential impacts summarized above. In so doing in section 6.5 below, we take into account considerations relevant to public welfare policy judgments required of the Administrator, such as those described here.

As recognized in sections 1.3.2 and 5.1, the Clean Air Act specifies that secondary standards protect against known or anticipated adverse effects to public welfare. In the Administrator's judgment as to the standards that would be requisite (i.e., neither more nor less stringent than necessary) to protect the public welfare under the Act, she will consider a number of factors including 1) what can be considered to constitute an adverse effect to the public welfare; 2) the nature and magnitude of the effects and the risks that remain after meeting the level of the current standard; and, 3) what is necessary to achieve the requisite (no more and no less) degree of protection. In the 2008 decision by which the current standard was established, the Administrator considered these factors in judging the previously-existing standard to not provide the requisite public welfare protection. At that time the Administrator found that the

1 exposure- and risk-based analyses available in that review indicated that adverse effects to
2 vegetation would be predicted to occur under air quality conditions associated with just meeting
3 the then-current standard. The effects identified were "visible foliar injury and seedling and
4 mature tree biomass loss in O_3-sensitive vegetation" (73 FR 16496). In so noting, the
5 Administrator indicated that he believed that "the degree to which such effects should be
6 considered to be adverse depends on the intended use of the vegetation and its significance to
7 public welfare" (73 FR 16496). With regard to consideration of intended use, the Administrator
8 took note of the specific uses of public lands set aside by Congress and intended to provide
9 benefits to the public welfare, "including lands that are to be protected so as to conserve the
10 scenic value and the natural vegetation and wildlife within such areas, and to leave them
11 unimpaired for the enjoyment for future generations" such as Class I areas (73 FR 16496). The
12 Administrator also recognized areas set aside by States, Tribes and public interest groups with
13 the intent "to provide similar benefits to the public welfare, for residents on State and Tribal
14 lands, as well as for visitors to those areas" (73 FR 16496).[10]
15   In the Administrator's judgments in the 2008 review, he did not identify specific criteria
16 or benchmarks or an overall level of protection from adverse environmental effects to public
17 welfare judged to be requisite under the Act.[11] As noted above, the scientists at the 1996
18 workshop identified ranges of cumulative seasonal index values (e.g., in terms of SUM06 or
19 W126) in the context of considering a degree of protection for vegetation effects defined in terms
20 of relative yield loss in crops and relative biomass loss in tree seedlings. In considering this
21 information in the context of a secondary standard, judgments are required by the Administrator
22 with regard to the degree that these or other benchmarks and other effects should be judged
23 adverse to the public welfare. In considering levels for a W126-based secondary standard that
24 may be appropriate to consider, we recognize that the statute requires that a secondary standard
25 be protective against only those known or anticipated O_3 effects that are "adverse" to the public
26 welfare, not all identifiable O_3-induced effects. Thus, we recognize both the importance of
27 scientific consensus statements that have been made regarding vegetation-related endpoints and
28 O_3 exposure levels that might protect against such key endpoints and the importance of placing
29 such conclusions in the context of consideration of the public welfare more broadly.

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[10] In considering areas that have not been afforded such special protection, ranging from vegetation used for residential or commercial ornamental purposes, such as land use categories that are heavily managed for commercial production of commodities such as agricultural crops, timer and ornamental vegetation, the Administrator indicated his expectation that protection of sensitive natural vegetation and ecosystems might be expected to also provide some degree of additional protection for heavily managed commercial vegetation (73 FR 16496).

[11] In remanding the 2008 decision on the secondary standard back to EPA, the Court indicated this omission, as described in section 1.2.2 above.

1   As discussed in section 5.1 and recognized by the EPA in prior reviews, staff recognizes
2   the importance of a more expansive construct or paradigm that addresses what constitutes
3   adverse effects of O_3 to public welfare. In so doing, we also recognize several aspects or
4   dimensions of vegetation effects for consideration within this paradigm. These include the
5   likelihood, type, magnitude, and spatial scale of the effect, as well as the potential for recovery
6   and any uncertainties relating to these conditions (77 FR 20231). As in the last review, we also
7   continue to recognize that the public welfare significance of O_3-induced effects on sensitive
8   vegetation growing within the U.S. can vary, depending on the nature of the effect, the intended
9   use of the sensitive plants or ecosystems, and the types of environments in which the sensitive
10   vegetation and ecosystems are located. Any given O_3-related effect on vegetation and
11   ecosystems (e.g., biomass loss, foliar injury), therefore, may be judged to have a different degree
12   of impact on the public welfare depending, for example, on whether that effect occurs in a Class
13   I area, a city park, or in commercial cropland. In the 2008 review, the Administrator judged it
14   appropriate that this variation in the significance of O_3-related vegetation effects should be taken
15   into consideration in judging the level of ambient O_3 that is requisite to protect the public welfare
16   from any known or anticipated adverse effects (73 FR 16496). For example, in considering
17   visible foliar injury and seedling and mature tree biomass loss in O_3-sensitive vegetation
18   expected under alternative air quality scenarios, the Administrator noted that "the degree to
19   which such effects should be considered to be adverse depends on the intended use of the
20   vegetation and its significance to the public welfare" (73 FR 16496). Further, the rulemaking
21   notice stated that "[i]n considering what constitutes a vegetation effect that is adverse from a
22   public welfare perspective, the Administrator believes it is appropriate to continue to rely on the
23   definition of 'adverse,' … that imbeds the concept of ''intended use'' of the ecological receptors
24   and resources that are affected, and applies that concept beyond the species level to the
25   ecosystem level." The notice went on to state that "[i]n so doing, the Administrator has taken
26   note of a number of actions taken by Congress to establish public lands that are set aside for
27   specific uses that are intended to provide benefits to the public welfare, including lands that are
28   to be protected so as to conserve the scenic value and the natural vegetation and wildlife within
29   such areas, and to leave them unimpaired for the enjoyment of future generations" (73 FR
30   16496). Such public lands that are protected areas of national interest include national parks and
31   forests, wildlife refuges, and wilderness areas.
32   We also consider effects on ecosystem services in considering adversity to public
33   welfare. For example, the WREA has evaluated the economic value of ecosystem services
34   affected by O_3 and how those services might be expected to change under different air quality
35   scenarios representing the current and potential alternative standards (U.S. EPA, 2014, chapter
36   6).

1   Thus, we recognize several important considerations in evaluating levels of protection
2   and levels for a cumulative seasonal W126-based standard including: the extent of areas
3   expected to be affected nationwide and the magnitude of those effects; the extent of effects in
4   areas of national significance; the extent to which these impacts might be judged significant from
5   a public welfare perspective and associated uncertainties in the information. Accordingly, we
6   recognize that the range of alternative standard levels that may be appropriate to consider differs
7   based on the weight placed on different aspects of the evidence and on different aspects of the
8   quantitative exposure/risk information, and the associated uncertainties, as well as on public
9   welfare policy decisions regarding the public welfare significance of the effects considered and
10   the approaches for considering benchmarks for growth or biomass loss and other vegetation
11   effects of O_3. As described in chapter 1, our objective is to identify the range of policy options
12   supported by the current evidence- and exposure/risk-based information and with consideration
13   of the role of the Administrator's public welfare judgments. In so doing, we recognize support
14   for consideration of a broad range of W126 index values, which we discuss in section 6.5, with
15   recognition of the different judgments that might provide support for different parts of such a
16   range.

17   **6.4 CASAC ADVICE**

18   In our consideration of potential alternative standards, in addition to the evidence-based,
19   risk/exposure-based, and air quality information discussed above, we also consider the advice
20   and recommendations of CASAC in EPA's proposed 2010 reconsideration of the 2008 decision,
21   as well as comments received thus far in the current review, in the context of its review of the
22   ISA, and earlier drafts of this document and the WREA. We have additionally considered public
23   comments received to date, some of which have suggested a lack of new information for support
24   of a distinct secondary standard and others that urge the consideration of a secondary standard
25   with a cumulative seasonal form using the W126 metric and a level within the range of 7 to 15
26   ppm-hrs.[12]
27   In response to the EPA's solicitation of CASAC's advice on the Agency's proposed
28   rulemaking as part of the reconsideration,[13] CASAC conveyed their support for a secondary
29   standard distinct from the primary standard, noting that "vegetation effects are more dependent

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[12] Public comment received thus far in this review are in the docket EPA–HQ–OAR-2008-0699, accessible at www.regulations.gov.
    [13] The reconsideration proposal included a proposed new cumulative, seasonal secondary standard, expressed as an index of the annual sum of weighted hourly concentrations (the W126 index), cumulated over 12 hours per day during the consecutive 3-month period within the O_3 season with the maximum index value, averaged over three years, set within a range of 7 to 15 ppm-hour (75 FR 3027).

1 on the cumulative exposure to, and uptake of, ozone over the course of the entire growing

2 season" (Samet, 2010).

3   *CASAC also supports EPA's secondary ozone standard as proposed: a new*
4   *cumulative, seasonal standard expressed as an annual index of the sum of*
5   *weighted hourly concentrations (i.e., the W126 form), cumulated over 12 hours*
6   *per day (8am to 8pm) during the consecutive 3-month period within the ozone*
7   *season with the maximum index value, set as a level within the range of 7 to [1]5*
8   *ppm-hours. This W126 metric can be supported as an appropriate option for*
9   *relating ozone exposure to vegetation responses, such as visible foliar injury and*
10   *reductions in plant growth. We found the Agency's reasoning ... to be supported*
11   *by the extensive scientific evidence considered in the last review cycle.*

12   In advice offered so far in the current review, which considers an updated scientific and

13 technical record since the 2008 rulemaking, the CASAC stated that "the focus [of the first draft

14 PA] on the W126 form is appropriate" (Frey and Samet, 2012, p. 2). They further commented

15 with regard to the support provided in the first draft PA for the consideration of such a form for a

16 secondary standard (Frey and Samet, 2012, p. 2).

17   *There is a strong justification made for using a cumulative and weighted exposure*
18   *standard for welfare effects (i.e. the W126), and for the utility of using a 3-month*
19   *daylight exposure metric. Averaging across years is not recommended because a*
20   *single high exposure year could have lasting effects because of the perennial*
21   *nature of many plants and the lag times associated with propagating effects*
22   *through ecosystem trophic levels. Averaging would obscure such critical impacts*
23   *and lead to inadequate protection against welfare effects.*

24 Additionally, in advice regarding consideration of levels, their advice indicated that "[o]ptions

25 for levels [of a secondary standard] should be based on factors including predicted 5% loss of

26 crop yield and predicted 1-2% loss for trees" (Frey and Samet, 2012, p. 2).

27   **6.5 PRELIMINARY STAFF CONCLUSIONS ON ALTERNATIVE STANDARD**

28   Staff's consideration of alternative secondary O_3 standards builds on our conclusion from

29 section 5.7 above that the body of evidence, in combination with the results of the WREA, calls

30 into question the adequacy of the current secondary standard and provides support for

31 consideration of alternative standards. In sections 6.1 to 6.3 above, we consider how the

32 currently available scientific evidence and exposure/risk information inform decisions regarding

33 the basic elements of the NAAQS: indicator (6.1), form and averaging time (6.2), and level (6.3).

34 In so doing, we consider both the information available at the time of the last review and

35 information newly available since the last review which has been critically analyzed and

36 characterized in the 2013 ISA. As an initial matter, with regard to the indicator, we conclude

37 that based on the available science it is still appropriate to continue to use measurements of O_3 in

1   accordance with federal reference methods as the indicator to address effects associated with

2   exposure to ambient O_3 alone or in combination with related photochemical oxidants.

3   In considering alternative standards, staff has considered the available body of evidence

4   as comprehensively assessed in the ISA, the risk and exposure information presented in the

5   second draft REA and CASAC advice and public comment thus far in this review with regard to

6   support for consideration of options that are different from the current standard, as articulated by

7   the following overarching question

8   • **To what extent does the currently available scientific evidence- and exposure/risk**
9   **based information, as reflected in the ISA and WREA, support consideration of**
10   **alternatives to the current O_3 standard to provide increased protection from**
11   **ambient O_3 exposures?**

12   In considering potential forms alternative to that of the current standard, we note that the

13 form for the current secondary standard is the 4th highest daily maximum 8-hour average,

14 averaged over three years. As discussed in chapter 5 and section 6.2 above, the longstanding

15 evidence regarding the fundamental aspects of O_3 exposure that are directly responsible for

16 inducing vegetation response indicates that plant response to O_3 is driven by the cumulative

17 exposure to O_3 during the growing season, rather than by a single event (U.S. EPA, 2013, section

18 2.6.6.1). This cumulative exposure depends on both the total duration of the exposure (from

19 repeated O_3 episodes) and the concentrations of those exposures (higher concentrations having a

20 disproportionate impact over lower concentrations). On the basis of this longstanding and

21 extensive evidence, the ISA concludes that exposure indices that cumulate and differentially

22 weight the higher hourly average concentrations over a season and also include the mid-level

23 values offer the most defensible approach for use in developing response functions and in

24 defining indices for vegetation protection (U.S. EPA, 2013, section 2.6.6.1).

25   CASAC advice in the 2008 review and on the 2010 proposed reconsideration has

26 additionally recognized that the nature of the exposures relevant to vegetation response is well

27 described by a cumulative seasonal form and has supported the use of such a form for a

28 secondary O_3 standard (Henderson, 2006; Samet, 2010). The current CASAC O_3 Panel has

29 expressed similar views, stating "[t]here is a strong justification made for using a cumulative and

30 weighted exposure standard for welfare effects (i.e. the W126)…" (Frey and Samet, 2012, p. 2).

31 We also note that on the basis of the evidence and exposure/risk information available in the two

32 previous reviews, and in consideration of CASAC advice, the Administrator has recognized the

33 importance of protecting vegetation from cumulative, seasonal exposures and proposed such a

34 form as an appropriate reasonable policy option (61 FR 65741-44; 62 FR 37899-905; 75 FR

35 3012-3027).

1   Thus, in considering alternative forms of the standard we conclude that it is reasonable
2   and appropriate to consider a cumulative, concentration-weighted form to provide protection
3   against cumulative, seasonal exposures to O_3 that are known or anticipated to harm sensitive
4   vegetation or ecosystems.   Such a form is specifically designed to directly measure the kind of
5   O_3 exposures that can cause harm to vegetation and would have a distinct advantage over the
6   form of the current standard in characterizing air quality conditions potentially of concern for
7   vegetation and demonstrating that the desired degree of protection against those conditions was
8   being achieved.
9   In considering the appropriate index for a cumulative seasonal form, we recognize that a
10   number of different cumulative concentration weighted indices have been developed and have
11   been evaluated in the scientific literature and in past NAAQS reviews in terms of their ability to
12   predict vegetation response and their usefulness in the NAAQS context (U.S. EPA, 2006, pp. 9-
13   11 to 9-15 and pp. AX9-159 to AX9-187; U.S. EPA, 2007, pp. 7-15/16). While these various
14   forms have different strengths and limitations, as noted in the ISA (U.S. EPA, 2013, section 9.5),
15   the W126 index[14] has some important aspects not shared by other non-sigmoidally weighted
16   cumulative indices. For example, given the lack of discernible threshold for vegetation effects in
17   general, we recognize the fact that the W126 metric does not have a cut-off in its weighting
18   scheme (down to about 30 ppb below which the weighting factor is effectively zero), such that it
19   includes consideration of potentially damaging lower O_3 concentrations. Additionally, the W126
20   metric also adds increasing weight to hourly concentrations from about 40 ppb to about 100 ppb,
21   an important feature because "as hourly concentrations become higher, they become increasingly
22   likely to overwhelm plant defenses and are known to be more detrimental to vegetation" (U.S.
23   EPA, 2013, p. 9-104). We additionally take note of CASAC advice in the 2008 review and on
24   the 2010 proposed reconsideration recommending the use of the W126 index for a cumulative
25   seasonal form for a secondary O_3 standard (Henderson, 2006; Samet, 2010). Similarly, the
26   current CASAC O_3 Panel has indicated that a focus on a W126 form is appropriate (Frey and
27   Samet, 2012) Therefore, on the basis of the strength of the evidence and advice from CASAC,
28   we conclude that the W126 index is the most appropriate cumulative seasonal form to consider
29   in the context of the secondary O_3 NAAQS review.
30   We next turn to the exposure periods – diurnal and seasonal – over which the W126
31   index would be summed in any given year. As discussed in section 6.2 above, the currently
32   available information continues to provide support for a definition of the diurnal period of

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[14] The W126 is a non-threshold approach described as the sigmoidally weighted sum of all hourly O_3 concentrations observed during a specified diurnal and seasonal exposure period, where each hourly O_3 concentration is given a weight that increases from 0 to 1 with increasing concentration (Lefohn et al, 1988; Lefohn and Runeckles, 1987; U.S. EPA, 2013, section 9.5.2).

1   interest as the 12-hour period from 8 am to 8 pm (U.S. EPA, 2013, section 9.5.3). In prior

2   reviews, the EPA has identified the 12-hour period from 8 am to 8 pm as appropriately capturing

3   the diurnal window with most relevance to the photosynthetic process (72 FR 37900; 75 FR

4   3013) and CASAC has generally supported the 12 hour daylight period (Henderson, 2006, 2007).

5   In light of the continued support in the evidence base, and no evidence on this issue differing

6   from that in previous reviews, we again conclude that it is appropriate to use the 12-hour period

7   from 8 am to 8 pm to cumulate daily O_3 exposures. On this basis, we conclude that the 12-hour

8   diurnal window (8:00 am to 8:00 pm) represents the portion of the diurnal exposure period that is

9   most relevant to predicting or inducing plant effects related to photosynthesis and growth and

10   thus is an appropriate diurnal period to use in conjunction with a W126 cumulative metric.

11   With regard to a seasonal period of interest, the current evidence base continues to

12   provide support for a seasonal period with a minimum duration of three months (U.S. EPA,

13   2013, section 9.5.3). Included in the currently available evidence is a new analysis that

14   compared 3- and 6-month maximum W126 values for over 1,200 AQS and CASTNET EPA

15   monitoring sites for the years 2008-2009 that found that the two accumulation periods were

16   highly correlated (U.S. EPA, 2013, section 9.5.3, Figure 9-13). Thus, although we recognize that

17   the selection of a single seasonal time period over which to cumulate O_3 exposures for a national

18   standard necessarily represents a balance of factors, given the significant variability in growth

19   patterns and lengths of growing season among vegetative species growing within the U.S., we

20   conclude it is appropriate to identify the seasonal W126 index value as that derived from the

21   consecutive 3-month period within the O_3 season with the highest W126 index value. We note

22   that such a 3-month exposure period was also supported by CASAC in advice provided during

23   the last review and on the 2010 proposed reconsideration (Henderson, 2006; Samet, 2010).

24   With regard to form, we additionally consider the period of time over which a cumulative

25   seasonal W126-based standard should be evaluated. In so doing, we have considered the support

26   for both a single year standard and one with a form averaged over three years (section 6.2). We

27   recognize that there are a number of O_3-induced effects that have the potential for public welfare

28   significance within the annual timeframe (i.e. reduced crop yields and visible foliar injury).

29   However, as noted in section 6.2 above, there are uncertainties associated with these effects that

30   make it difficult to determine the degree of annual protection needed to protect the public

31   welfare from any known or anticipated adverse effects. On the other hand, annual effects in

32   perennial species can be "carried over" into the subsequent year where they affect growth and

33   reproduction (U.S. EPA, 2013, pp. 9-43 to 9-44 and p. 9-86). When these annual effects occur

34   over multiple years due to elevated O_3 exposures across several years, they have the further

35   potential to be compounded, increasing the potential for effects at the population and ecosystem

36   level, including effects on associated ecosystem services that may be of greater significance to

1   the public welfare. These impacted services can include alteration of below-ground

2   biogeochemical cycles, and alteration of both above- and below- ground terrestrial community

3   composition and terrestrial ecosystem water cycling (U.S. EPA, 2013, Table 9-19) and

4   reductions in productivity and carbon sequestration in terrestrial ecosystems. We additionally

5   note that multiple years of critical O_3 exposures might be expected to result in larger impacts on

6   forested areas, i.e. increased susceptibility to other stressors such as insect pests, disease, co-

7   occurring pollutants and harsh weather, than intermittent occurrences of such exposures due to

8   the compounding or carry-over effects on tree growth.

9   Given the above, we conclude that the public welfare significance of the effects that can

10   occur as a result of multiple year O_3 exposures are greater than those associated with a single

11   year. Thus, to the extent that the Administrator's priority for public welfare protection to be

12   afforded by the secondary O_3 standard is on long-term effects that occur in sensitive tree species

13   in natural forested ecosystems including federally protected areas such as Class I areas or on

14   lands set aside by States, Tribes and public interest groups to provide similar benefits to the

15   public welfare, a standard with a form that evaluates the cumulative seasonal index across

16   multiple years might be considered to provide an appropriate match to the nature of O_3-related

17   effects on vegetation upon which the secondary O_3 standard is focused. In considering such

18   forms, we focus on one that averages the W126 index values across three years, as discussed in

19   section 6.2 above.

20   We take note, however, of comments from CASAC on this matter, in particular their

21   comment in the current review that "…[a]veraging across years is not recommended because a

22   single high exposure year could have lasting effects because of the perennial nature of many

23   plants and the lag times associated with propagating effects through ecosystem trophic levels.

24   Averaging would obscure such critical impacts and lead to inadequate protection against welfare

25   effects" (Frey and Samet, 2012, p. 2). We recognize that annual effects on perennials can

26   propagate into subsequent years, and thus first consider the available analyses of year-to-year

27   variability in W126 index values. For example, based on an analysis of the inter-annual

28   variability of seasonal W126 index values (using 2008-2010 data from the AQS database), the

29   W126 index values can vary significantly from year to year (see Appendix 2C). This is not

30   unexpected given the logistic weighting function and also inter-annual variability in

31   meteorological conditions which contribute to O_3 formation (see Appendix 2C). As a result,

32   areas that meet a three-year average standard form for which there is substantial inter-annual

33   variability of seasonal W126 index, are likely to have some years that are below the level of the

34   standard and others that are at or above. Given this fact, several important implications should

35   be noted. First, in regard to implications for potential long-term compounding of vegetation or

36   ecosystem effects, it would be expected that annual impacts in years with cumulative air quality

1 below that of the three year average would allow species to do better than the target level of

2 protection expected to be achieved on average across the three year period. Thus, staff note the

3 importance, when considering an appropriate level for a form that averages W126 index values

4 across three years, of considering the extent to which the cumulative effect of different average

5 W126 exposures across the three-year period would be judged adverse.

6   Additionally, in regard to implications for standard stability, a standard based on an

7 annual W126 index would be expected to have a lower degree of year-to-year stability relative to

8 a standard based on a form that averages seasonal indices across three consecutive years, given

9 the potential for large year-to-year variability in annual W126 values in areas across the country.

10 Thus, a three-year evaluation period can contribute to greater public welfare protection by

11 limiting year-to-year disruptions in ongoing control programs that would occur if an area was

12 frequently shifting in and out of attainment due to extreme year-to-year variations in

13 meteorological conditions. This greater stability in air quality management programs thus

14 facilitates achievement of the protection intended by a standard.

15   Thus, to the extent that the Administrator puts greater weight on protecting those effects

16 associated with multi-year exposures and given the described public welfare benefit of having a

17 standard of a form with more year-to-year stability, we conclude that it is appropriate to consider

18 a secondary standard form that averages the seasonal W126 index values across three

19 consecutive years to achieve the appropriate target level of protection for longer-term effects,

20 including compounding, and to achieve greater stability in air quality management programs, and

21 thus, public welfare protection, than might result from an annual standard.

22   Turning to consideration of an appropriate range of levels for a W126 based standard, we

23 first note that the available O_3-related vegetation effects evidence reflects a continuum from

24 relatively higher O_3 concentrations, at which scientists generally agree that vegetation effects are

25 likely to occur, through lower concentrations at which the likelihood and magnitude of a

26 response become increasingly uncertain. Further, we recognize the different types of O_3-induced

27 effects and genetic variability within and between species which contribute variability to

28 observed responses across species. In light of this, we recognize the role of the Administrator's

29 judgments regarding the adversity of the known and anticipated effects to the public welfare.

30 Thus, the EPA has developed a paradigm to assist the Administrator in putting the available

31 science and exposure/risk information into the context of public welfare (as discussed in section

32 5.1 above). This paradigm has evolved over the course of the O_3 NAAQS reviews and has also

33 been informed by similar constructs developed for other secondary NAAQS reviews. Most

34 recently, this paradigm has expanded to include consideration of adversity in terms of effects on

35 the ecosystem services associated with identified O_3-induced effects (see discussion in 6.3

36 above).

1   In considering the range of W126 index values most recently cited by CASAC, which
2   generally correspond to conclusions from the 1996 workshop, we note that the tree seedling
3   biomass loss percentages were per-year percentages, with consideration of longer-term impacts.
4   Staff is mindful of this in light of our consideration of a potential alternative standard with a
5   multi-year form. We additionally take note of the workshop objectives to identify values that
6   might provide protection of vegetative components of natural ecosystems (recognizing foliar
7   injury as the indicator), protection of some aspect of the integrity of forest ecosystems (using
8   growth effects on seedlings as surrogate) and protection against crop yield reductions (while
9   acknowledging uncertainties). In considering the range that derives, at least in part, from this
10   workshop, we additionally recognize the need for our purposes with regard to a secondary O_3
11   standard, to consider the public welfare significance of identified effects, as discussed in section
12   6.3 above. We find that this need to consider public welfare significance may lead to
13   identification of a somewhat different range of W126 index values as appropriate to consider for
14   levels for a W126-based standard.[15]

15   In considering potential levels for an alternative standard based on the W126 metric, we
16   find it useful to consider the observations of biomass loss and crop yield loss in Table 6-1 above.
17   In so doing, we take note of the different index value estimates with regard to number of studied
18   species below different response benchmarks, as well as the median response. For example, we
19   note that the tree seedling relative biomass loss estimates for 15-17 ppm-hrs include five of the
20   twelve studied species below 2%, five to six species below 5% and nine to ten species below
21   10%, as well as a median species response of 5 to 6%. At these index values, the median crop
22   yield loss estimate across studied crops is just at or below 5% (and eight of ten are below 10%).
23   At the lower end of the index values in Table 6-1, the tree seedling estimates for 7 ppm-hrs
24   include ten species below 5%, seven below 2% and a median response just at or below 2%. We
25   additionally consider the WREA estimates which indicate 143 or 144 of the 145 assessed Class I
26   areas with tree seedling weighted relative biomass loss estimates below 2% for air quality
27   scenarios representing W126 values of 15 and below. Such estimates are of particular relevance
28   to judgments required of the Administrator regarding effects of public welfare importance.
29   Further we note other WREA estimates indicating potential benefits for effects related to public
30   welfare, such as carbon sequestration and pollutant removal. In so doing, however, we also take
31   note of the appreciable uncertainty in these quantitative estimates, and the policy judgements
32   required of the Administrator with regard to consideration of such uncertainties.
33   On the basis of all the considerations described above, including the evidence and
34   exposure/risk analyses, and advice from CASAC, we conclude that an appropriate range of

[15] We additionally recognize variability in derivation of W126 estimates generally equivalent to SUM06 estimates from the 1996 workshop, as noted in section 6.3 above.

1 W126 index values for the Administrator to consider in identifying a target degree of public

2 welfare protection, extends from 7 ppm-hrs to 15 ppm-hrs or somewhat higher (as further

3 described below). In so doing, we primarily consider the evidence- and exposure/risk-based

4 information for cumulative seasonal O_3 exposures represented by W126 index values (including

5 those represented by the WREA average W126 scenarios) associated with biomass loss in

6 studied tree species, both in and outside areas that have been afforded special protections. We

7 additionally recognize foliar injury as an important O_3 effect which, depending somewhat on

8 severity and spatial extent, may reasonably be concluded to be of public welfare significance

9 when occurring in nationally protected areas. However, we additionally take note of the

10 appreciable variability in this endpoint, as summarized in chapter 5 and section 6.3 above, which

11 poses challenges to giving it primary emphasis in identifying potential alternative standard

12 levels. Similarly, we give less emphasis to consideration of crop yield loss in our consideration

13 of potential standard levels here and in section 6.3 above, noting the median estimates of

14 approximately 5% or lower for W126 index levels at and below 17 ppm-hrs. We also note the

15 range of factors affecting annual crop yields, including those related to the role of management

16 strategies as recognized in sections 5.3 and 6.2 above which complicate the identification of a

17 degree of impact that can be considered adverse to the public welfare. On the other hand, tree

18 biomass loss can be an indicator of more significant ecosystem-wide effects which might

19 reasonably be concluded to be significant to public welfare. For example, when it occurs over

20 multiple years at a sufficient magnitude, it is linked to an array of effects on other ecosystem-

21 level processes, such as nutrient and water cycles, changes in above and below ground

22 communities, carbon storage and air pollution removal (U.S. EPA, 2014, Figure 5-1), that have

23 the potential to be adverse to the public welfare.

24 In focusing on trees and their associated ecosystem services, we first note that the studied

25 tree species vary widely in their sensitivity to O_3-induced relative biomass loss. For example,

26 2% seedling biomass loss is estimated to occur with cumulative seasonal O_3 exposure in terms of

27 W126 index values below 10 ppm-hrs for seven of the studied species and at or above

28 approximately 20 ppm-hrs in the other five studied species (Figure 5-1 above). The median

29 W126 index value (across studied species) for which a 2% biomass loss is estimated ranges

30 between approximately 7 and 14 ppm-hrs, among the three approaches presented in the WREA

31 (see description in section 5.2.1). In considering the potential magnitude of the ecosystem

32 impact of tree species, we focus on the WREA estimates of weighted relative biomass loss for

33 the W126 air quality scenarios (U.S. EPA, 2014, section 6.8). For the current standard and the

34 three W126 scenarios, these estimates indicate weighted relative biomass loss less than or equal

35 to 2% in 143-144 of 145 assessed nationally protected Class I areas. To the extent that emphasis

36 is given to such estimates for nationally protected Class I areas and for appreciable percentages

1   of forested areas nationwide, a W126 index value extending up to 15 ppm (or perhaps somewhat

2   higher) may be appropriate to consider.

3   In considering the evidence-based information regarding tree seedling growth effects and

4   specifically biomass loss, we recognize an array of W126 index values that may be appropriate

5   to consider depending on the weight placed on different policy-related objectives with regard to

6   proportions of the 12 studied tree species at or below different growth response benchmarks and

7   on associated uncertainties. For example, conclusions may be reached regarding the higher index

8   values (e.g., up to 17 ppm-hrs) to the extent weight is placed on W126 index values for which

9   tree seedling C-R functions project less than 2% biomass loss in approximately half of the

10 studied species and less than 10% biomass loss in the large majority of studied species and on

11 index values for which weighted RBL is below 2% in nearly all assessed Class I areas, and to the

12 extent that estimates associated with the remaining species are judged too variable and/or

13 uncertain. For index values of 15 or 17 ppm-hrs, the species-specific composite C-R functions

14 indicate relative biomass loss less than 2% in at least five of the 12 studied species and less than

15 10% in at least nine or ten, in addition to an estimated median across studied species of

16 approximately 5-6% . Additionally, the WREA estimates for all three W126 scenarios include

17 weighted RBL below 2% in 143 to 144 of 145 assessed Class I areas.   Alternatively, to the

18 extent weight is given to median biomass across tree species of no more than 2% based on the

19 evidence-based C-R functions, and to the potential, while uncertain, for appreciable gains in

20 carbon sequestration, a focus on the lower end of the range (e.g., down to 7 ppm-hr) may be

21 appropriate.

22   Thus, in staff's view, the evidence- and exposure/risk-based information relevant to tree

23 biomass loss and the associated ecosystem services important to the public welfare support

24 consideration of a W126-based secondary standard with index values within the assessed range

25 of 7-15 ppm-hrs or somewhat higher (e.g., 17 ppm-hrs). We consider such a range for a

26 potential alternative cumulative seasonal W126-based standard, averaged over three years. In so

27 doing, we take note of CASAC's advice regarding the importance of considering the lasting or

28 carry-over effects that can derive from single year exposures of perennial plants, and recognize

29 the importance of considering the available evidence and exposure/risk based information related

30 to such effects, as well as associated uncertainties. We additionally recognize uncertainty

31 associated with any characterization of a relationship between the level of protection afforded for

32 cumulative growth-related effects by potential alternative W126-based standards of single year

33 or three-year average form. Lastly, we are mindful of the public welfare judgments required of

34 the Administrator with regard to the public welfare significance of identified effects and the

35 requisite level of protection, as well as the appropriate weight to assign the range of uncertainties

36 inherent in the evidence and analyses.

1   In also noting the potential ecosystem services benefits related to tree biomass loss for

2   potential alternative W126-based standards in light of the qualitative and semi-quantitative

3   information in the WREA regarding the types and potential magnitude of impacts on associated

4   services, we recognize, in particular that impacts on climate can reasonably be concluded

5   significant from a public welfare perspective and CO_2 sequestration has been identified as a

6   potentially important tool for managing anthropogenic impacts on climate. However, we

7   additionally take note of significant uncertainties and limitations associated with WREA

8   estimates related to carbon sequestration. Thus, in selecting a target level of protection for forest

9   trees and their associated ecosystem services, the Administrator will need to exercise judgments

10   regarding the appropriate weight to place on the potential for benefits to the public welfare with

11   respect to ecosystem services of carbon storage and urban air pollution removal associated with

12   tree growth, as well as the large uncertainties associated with this information.

13   We further recognize that public welfare considerations taken into account by the

14   Administrator may have the potential to affect the target protection judged requisite by the

15   Administrator and the associated range of W126 index values. For example, to the extent the

16   Administrator chooses to put more weight on effects associated with longer-term conditions, it

17   may be appropriate to evaluate the significance of these longer term effects to the public welfare,

18   as well as the role that year-to-year exposure variability can play in realizing the potential public

19   welfare impacts. In so doing, the Administrator may put weight on a range of percentages of

20   biomass loss (either greater or less than 2%) with an objective to achieve substantial protection

21   of a large proportion of the studied species. Thus, in considering a range of 3-year average

22   W126 index values appropriate to provide the target protection, we recognize that the

23   Administrator may consider a range somewhat beyond the staff identified range.

24   In considering the lower end of the staff-identified range, the Administrator would need

25   to put more weight on the uncertainties that suggest the analyses may be underestimating the

26   public welfare impacts associated with different cumulative exposure levels. These uncertainties

27   can include the relatively small number of O_3 sensitive species for which we have robust C-R

28   functions included in the analyses, O_3-sensitive species for which we have no C-R function, the

29   lack of information regarding the relationship of O_3 effects on tree seedlings (e.g., relative

30   biomass loss) that occur in one or a few growing seasons to longer-term effects that might be

31   expected to accrue over the life of the trees extending into adulthood, the paucity of ambient air

32   monitoring data in some areas (e.g., the west) that leads to less than complete national coverage,

33   recognition that co-occurring stressors may in some cases exacerbate predicted O_3-induced

34   effects, and the limited number of WREA urban area case studies, uncertainties associated with

35   the model-adjusted air quality exposure surfaces,and the inability to quantify some potentially

1   important associated ecosystem services and incremental impacts (i.e. for insect damage, fire

2   regimes).

3   In considering a range somewhat above that of the staff-identified range, the

4   Administrator would need to put more weight on the uncertainties suggesting the potential for

5   overestimating the beneficial public welfare impacts that might be achieved at 3-year average

6   levels within the staff-identified range. These uncertainties include the lack of information

7   (particularly quantitative) on the relationship of tree seedling O_3 effects (e.g., relative biomass

8   loss) that occur in one or a few growing seasons to longer-term effects that might be expected to

9   accrue over the life of the trees extending into adulthood, recognition that co-occurring stressors

10 or environmental factors (e.g., drought) may in some cases mitigate predicted O_3-induced effects,

11 the variability in number of C-R functions available for each of the 12 studied tree species, and

12 information concerning the extent to different endpoints and effects might be considered adverse

13 to public welfare . Given these and potentially other uncertainties, the Administrator may choose

14 to select a higher range of levels judged to have less potential to provide overprotection.

15   Lastly, we also conclude that, to the extent the Administrator finds it useful to consider

16 the public welfare protection that might be afforded by a revised primary standard, this is

17 appropriately judged through the use of a cumulative seasonal W126-based exposure metric.

18 Such a use could inform a judgment of whether the primary standard would be expected to

19 achieve the level of public welfare protection concluded to be requisite under the Act in terms of

20 a metric considered appropriate to judging impacts on public welfare. See Mississippi. 723 F. 3d

21 at 272-73. The staff further concludes that the drawing of conclusions with regard to the public

22 welfare protection afforded by such a standard, as well as identification of the requisite level of

23 protection for such a standard, should entail consideration of the air quality conditions likely to

24 be achieved in terms of the cumulative seasonal W126-based metric described above. In such a

25 consideration, such as through the review of overlap analyses discussed in section 6.2 above,

26 staff further concludes it is important to take into account associated uncertainties, including

27 those associated with the limited monitor coverage in many rural areas, including those in the

28 west and southwest and at high elevation sites.

29 **6.6 SUMMARY OF PRELIMINARY CONCLUSIONS ON THE SECONDARY**
30 **STANDARD**

31   Staff preliminary conclusions are informed by our consideration of the available

32 scientific evidence as assessed in the ISA, the air quality/exposure/risk information in the second

33 draft WREA, advice from CASAC thus far in this review and in prior reviews, and public

34 comment thus far in this review. Staff conclusions in the final PA will be further informed by

35 comments from CASAC and the public on this draft document and by the final WREA.

1   Staff preliminary conclusions on policy options that are appropriate for the
2 Administrator's consideration in making decisions on the secondary standards for O_3, together
3 with supporting conclusions from sections 5.7 and 6.5 above, are briefly summarized below. In
4 reaching conclusions on alternative standards to provide requisite protection for public welfare
5 effects associated with ambient O_3 exposures, staff has considered these standards in terms of the
6 basic elements of the NAAQS: indicator, form, averaging time, and level. In drawing these
7 conclusions, we are mindful that the Act requires secondary standards to be set so that, in the
8 Administrator's judgment, they are requisite to protect public welfare from known or anticipated
9 adverse environmental effects, such that the standards are to be neither more nor less stringent
10 than necessary. Thus, the Act does not require that NAAQS be set at zero-risk levels, but rather
11 at levels that reduce risk sufficiently to protect public welfare from adverse effects.

12   (1) Staff preliminarily concludes, based on the combined consideration of the body of
13   evidence and the results from the quantitative exposure/risk assessment, that the
14   available evidence and exposure/risk information call into question the adequacy
15   of the public welfare protection provided by the current standard and it is
16   appropriate to consider revising the standard to provide greater public welfare
17   protection.

18   (2) In considering an appropriate target level of protection for a revised standard,
19   staff additionally preliminarily concludes that it is appropriate to judge O_3 public
20   welfare impacts using the cumulative seasonal W126-based metric.

21   a. To the extent the Administrator finds it useful to consider the extent of
22   public welfare protection that might be afforded by a revised primary
23   standard, staff preliminarily concludes that public welfare protection is
24   appropriately judged through the use of the cumulative seasonal W126-
25   based metric.

26   (3) With regard to indicator, staff preliminarily concludes that it is appropriate to
27   continue to use O_3 as the indicator for a standard that is intended to address
28   welfare effects associated with exposure to O_3, alone or in combination with
29   related photochemical oxidants. Based on the available information, staff
30   preliminarily concludes that there is no basis for considering an alternative
31   indicator at this time.

32   (4) With regard to averaging time and form, staff preliminarily concludes that it is
33   appropriate to consider a revised secondary standard in terms of the cumulative,
34   seasonal, concentration-weighted form, the W126 index. With regard to

1   definition of the W126 index for this purpose, staff makes the additional
2   preliminary conclusions.

3   a. It is appropriate to consider the consecutive 3-month period within the O_3
4   season with the maximum index value as the seasonal period over which
5   to cumulate hourly O_3 exposures. Staff notes that the maximum 3-month
6   period generally coincides with maximum biological activity for most
7   vegetation, making the 3-month duration a suitable surrogate for longer
8   growing seasons.

9   b. It is appropriate to cumulate daily exposures for the 12-hr period from 8
10   am to 8 pm, generally representing the daylight period during the 3-month
11   period identified above.

12   c. It is appropriate to consider a form that averages W126 index values
13   across three consecutive years. Staff concludes it is appropriate to
14   consider this form in conjunction with appropriate levels in order to
15   provide the desired degree of public welfare protection from O_3 effects
16   across multiple years.

17   With regard to level for a standard as described above, we preliminarily conclude that it
18   is appropriate to give consideration to a range of levels from somewhat above 15 ppm-hrs to 7
19   ppm-hrs, expressed in terms of the W126 index. Staff additionally notes that, consideration of
20   the support provided by the information available in this review will depend on public welfare
21   policy judgments by the Administrator regarding the protection of public welfare. This range
22   reflects staff judgment that a standard set within this range could provide an appropriate degree
23   of public welfare protection.

24   **6.7 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND**
25   **DATA COLLECTION**

26   Staff believes it is important to highlight key uncertainties associated with establishing
27   secondary standards for O_3. Such key uncertainties and recommendations for welfare-related
28   research, model development, and data gathering are outlined below. In some cases, research in
29   these areas can go beyond aiding standard setting to aiding in the development of more efficient
30   and effective control strategies. We note, however, that a full set of research recommendations
31   to meet standards implementation and strategy development needs is beyond the scope of this
32   discussion. Based on items highlighted in chapter 9 of the ISA and chapter 5 and 6 herein,we
33   have identified the following key uncertainties, research questions and data gaps that have been
34   highlighted in this review of the welfare-based secondary standard. The first set of key

1 uncertainties and research recommendations discussed below is that associated with the
2 extrapolation to plant species and environments outside of specific experimental or field study
3 conditions. The second set of key uncertainties and research recommendations pertain to our
4 ability to assess the impact of O_3 on other welfare effects categories such as climate, ecosystem
5 components such as wildlife, and whole ecosystem structure and function. Third, we identify
6 research areas related to the development of approaches, tools, or methodologies useful in
7 characterizing the relationship between O_3 and public welfare in a policy context. These three
8 areas are described below.

9   There have been five decades of research regarding O_3 effects on plants and much
10 information has been compiled in previous reviews. One of the most important research results
11 for the review of the secondary O_3 standard is C-R relationships for plant species. This review
12 uses C-R functions from 22 crop and tree species. However, there are tens of thousands of plant
13 species in the U.S. (USDA, NRCS. 2014[16]) and 66 plant species have been identified as O_3
14 sensitive on National Park Service and US Fish and Wildlife Service lands[17]. Studies using large
15 numbers of native plant species across regions where those species are indigenous, might be
16 expected to reduce uncertainties associated with extrapolating plant response for a given level of
17 O_3 using composite response functions across differing regions and climates. Research on
18 additional species might additionally improve our understanding of the full range of response of
19 plant species to O_3. Studies focused on fruits and vegetables might assist in reducing
20 uncertainties associated with O_3 effects on agriculture. Particular focus is suggested on
21 organically grown vegetables that may receive less intensive management than conventionally
22 grown crops. Recent studies indicate that watermelons may be particularly sensitive to O_3
23 exposure (U.S. EPA, 2013, section 9.4.4.1) and older studies indicate grapes, honeydew melon,
24 lemons and oranges may also be O_3 sensitive (Abt Associates Inc., 1995).

25   National visible foliar injury surveys can indicate how widespread O_3 effects may be
26 within the US. However, there remain uncertainties about the nature of the effects indicated by
27 the observed foliar injury. These include uncertainty associated with estimating the risk to
28 vegetation of differing amounts of O_3-induced visible foliar injury over the plant's leaf area and
29 the relationship between relative soil moisture and the incidence and severity of foliar injury in
30 sensitive species, as well as the extent to which visible foliar injury impacts ecosystem services
31 (e.g., tourism).

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[16] USDA, NRCS. 2014. The PLANTS Database (http://plants.usda.gov, 3 January 2014). National Plant Data Team, Greensboro, NC 27401-4901 USA.

[17] See http://www2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf

1   Some new information has emerged linking effects on tree seedlings with larger trees and
2   similarities in results between exposure techniques (U.S. EPA 2013, section 9.6). Uncertainties
3   remain in this area and in relationships between effects on individual plants and ecosystem
4   effects. There are also uncertainties in extrapolating from O_3 effects on juvenile to mature trees
5   and from trees grown in the open versus those in a closed forest canopy in a competitive
6   environment. Uncertainties in extrapolating individual plant response spatially or to higher
7   levels of biological organization, including ecosystems, could be informed by research that
8   explores and better quantifies the nature of the relationship between O_3, plant response and
9   multiple biotic and abiotic stressors, including those associated with climate change. Because
10   these uncertainties are multiple and significant due to the complex interactions involved, new
11   research will likely require a combination of manipulative experiments with model ecosystems,
12   community and ecosystem studies along natural O_3 gradients, and extensive modeling efforts to
13   project landscape-level, regional, national and international impacts of O_3.
14   Uncertainties associated with projections of the effects of O_3 on the ecosystem processes
15   of water, carbon, and nutrient cycling, particularly at the stand and community levels might be
16   addressed through research on the effects on below ground ecosystem processes in response to
17   O_3 exposure alone and in combination with other stressors. These below-ground processes
18   include interactions of roots with the soil or microorganisms, effects of O_3 on structural or
19   functional components of soil food webs and potential impacts on plant species diversity,
20   changes in the water use of sensitive trees, and if the sensitive tree species is dominant, potential
21   changes to the hydrologic cycle at the watershed and landscape level. Research on competitive
22   interactions under different O_3 exposures might improve our understanding of how O_3 may
23   affects biodiversity or genetic diversity. Such research could be strengthened by modern
24   molecular methods to quantify impacts on diversity.
25   Important interactions with biotic and abiotic stressors have been identified in this
26   review. More tools and research would improve our understanding of relationships between O_3
27   exposure and stressors such as insect infestations, plant diseases, drought and potential stressors
28   from climate change. It is also important to understand how such interactions may affect
29   ecosystem services such as CO_2 sequestration; food and fiber production; wildlife habitat and
30   water resources.
31   One of the most important uncertainties in this review is the characterization of air
32   quality in rural areas where there is limited monitoring. More comprehensive monitoring in
33   these areas would reduce uncertainties associated with O_3 exposures in many rural areas. Areas
34   of particular uncertainty include protected natural areas in the western U.S, including those at
35   high elevation, as well as those downwind of recently expanded oil and gas development areas.
36   Uncertainties associated with quantifying exposure in areas with and without monitors might be

1 addressed through additional work on interpolation methods and air quality models that are

2 tailored to estimating cumulative seasonal exposures, as well as on more refined spatial grids and

3 complex terrain.

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APPENDICES

1 **APPENDIX 2A**

2 **SUPPLEMENTAL AIR QUALITY MODELING ANALYSES OF**
3 **BACKGROUND OZONE**
4

5 **Table of Contents**

20

2A-2

150 **1.**   **Introduction**

151   One of the aspects of ozone that is unusual relative to the other pollutants with National
152   Ambient Air Quality Standards (NAAQS) is that, periodically, in some locations, an appreciable fraction of
153   the observed ozone results from sources or processes other than local and regional anthropogenic
154   emissions of ozone precursors (Fiore *et al*., 2002). Any ozone formed by processes other than the
155   chemical conversion of local or regional ozone precursor emissions, such as nitrogen oxides (NOx) or
156   volatile organic emissions (VOC), is generically referred to as "background" ozone. As part of this review
157   of the ozone NAAQS, EPA completed an extensive review of the known aspects of background ozone
158   and summarized the findings in the Integrated Science Assessment (ISA) in March 2013 (USEPA, 2013).
159   The purpose of this appendix is to present the results from supplemental air quality modeling analyses
160   related to background ozone that were completed by EPA subsequent to the ISA. While these updated
161   analyses use a recent base year (2007) and consider an alternative modeling methodology which can
162   better account for non-linear ozone chemistry in some conditions, the results are largely consistent with
163   previous determinations about the magnitude of background ozone contributions across the U.S.

164   Away from the surface, ozone can have an atmospheric lifetime on the order of weeks. As a
165   result, background ozone can be transported long distances at heights above the boundary layer and,
166   when meteorological conditions are favorable, be available to mix down to the surface and add to the
167   total ozone loading from non-background sources. Generically, background ozone can originate from
168   natural sources of ozone and ozone precursors, as well as from far upwind manmade emissions of ozone
169   precursors.   Natural sources of ozone precursor emissions such as wildfires, lightning, and vegetation
170   can lead to ozone formation by chemical reactions with other natural sources[1]. Another important
171   natural component of background is ozone that is naturally formed in the stratosphere through
172   interactions of UV light with atomic oxygen (O_2). Stratospheric ozone can periodically mix down to the
173   surface at high concentrations, especially at higher altitude locations. The manmade portion of the
174   background includes any ozone formed due to anthropogenic sources of ozone precursors emitted far
175   away from the local area (e.g., international emissions). Finally, both biogenic and international
176   anthropogenic emissions of methane, which can be chemically converted to ozone over relatively long
177   time scales, can also contribute to global background ozone levels.

178   The precise definition of background ozone can vary depending upon context, but it generally
179   refers to ozone that is formed by sources or processes that cannot be influenced by actions within the
180   jurisdiction of concern. In the first draft policy assessment document (EPA, 2012), EPA presented three
181   specific definitions of background ozone: natural background, North American background, and U.S.
182   background. Natural background (NB) was the narrowest definition of background and it was defined as
183   the ozone that would exist in the absence of any manmade ozone precursor emissions. The other two
184   previously-established definitions of background presume that the U.S. has little influence over
185   anthropogenic emissions outside our continental or domestic borders. North American background
186   (NAB) is defined as that ozone that would exist in the absence of any manmade ozone precursor

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[1] Ozone formed through reactions between natural emissions and local anthropogenic emissions (e.g., biogenic
VOC with man-made NOx) is generally not considered to be background ozone.

187   emissions inside of North America. U.S. background (USB) is defined as that ozone that would exist in
188   the absence of any manmade emissions inside the United States. It is important to note that **each of**
189   **these three definitions of background ozone requires photochemical modeling simulations** to estimate
190   what the residual ozone concentrations would be were the various anthropogenic emissions to be
191   removed.

192   As noted in the first draft policy assessment, EPA has revised several aspects of our
193   methodology for estimating the change in health risk and exposure that would result from a revision to
194   the ozone NAAQS. First, risk estimates are now based on total ozone concentrations as opposed
195   previous reviews which only considered risk above background levels. Second, EPA is now using air
196   quality models to estimate the spatial patterns of ozone that would result from attaining various levels
197   of the NAAQS, as opposed to simplistic rollback techniques that required the estimation of a background
198   ozone "floor" beyond which the rollback would not take place. Both of these revisions have had the
199   indirect effect of obviating the need for estimating background ozone levels as part of the ozone risk
200   and exposure assessment (REA). Regardless, EPA expects that a well-founded understanding of the
201   fractional contribution of background sources and processes to surface ozone levels will be valuable
202   towards informing policy decisions about the NAAQS. Section 2 of this document will describe the
203   supplemental air quality modeling simulations that have recently been completed by EPA to bolster our
204   understanding of background ozone. Section 3 will present the results from the updated analyses and
205   provide estimates of average background ozone levels, and how they can vary in time and space across
206   the U.S. Based on the same modeling, Section 4 will consider the entire spectrum of variable
207   background ozone levels with special emphasis on areas and times in which background can approach or
208   exceed the level of the NAAQS. Section 5 will utilize the supplemental air quality modeling estimates to
209   determine the relative importance of specific components of background ozone. Section 6 will present
210   estimates of the overall fraction of ozone that is estimated to result from background sources or
211   processes in each of the 12 urban case study areas in the epidemiology study based analyses in Chapter
212   7 of the Risk and Exposure Assessment (REA) (EPA, 2014) based on the updated modeling. Finally,
213   Section 7 will conclude with a limited analysis of how background ozone levels impact longer-term
214   ozone metrics that may be important from a welfare perspective (i.e., W126).

215   **2.   Description of modeling methodologies**

216   As noted above, air quality models are typically used to estimate background ozone as it is quite
217   difficult to measure directly. Without special monitoring, it is impossible to determine how much of the
218   ozone measured by a monitor originated from sources that are considered background. Even the most
219   remote monitors within the U.S. can periodically be affected by U.S. anthropogenic emissions. Previous
220   modeling studies have estimated what background levels would be in the absence of certain sets of
221   emissions by simply comparing the ozone differences between a base model simulation and a control
222   simulation in which emissions were removed. This basic approach is often referred to as "zero out"
223   modeling or "emissions perturbation" modeling. Examples of zero out modeling include the three major
224   studies summarized in the ISA (Zhang et al., 2011; Emery et al., 2012, Lin et al., 2013). It is important to
225   note that the specific concepts of NB, NAB, and USB are all explicitly tied to zero-out modeling, as those
226   definitions are based on estimating what remains *in the absence of* specific sets of man-made emissions.

227   EPA has conducted and will describe updated air quality modeling for a 2007 base year that employs a
228   regional air quality model nested within a coarser-scale global chemical transport model to estimate NB,
229   NAB, and USB levels when the respective manmade emissions are zeroed. This modeling is described in
230   detail in section 2a.

231   While the zero-out approach has traditionally been used to estimate background ozone levels,
232   the methodology has some acknowledged limitations. First, from a policy perspective, the purely
233   hypothetical and ultimately unrealizable zero manmade emissions scenarios have limited application in
234   this regard. Secondly, the assumption that background ozone is what is left after specific emissions have
235   been removed within the model simulation can be misleading in locations where ozone chemistry is
236   highly non-linear. Depending upon the local composition of ozone precursors, NOx emissions
237   reductions can either increase or decrease ozone levels in the immediate vicinity of those reductions.
238   For those specific urban areas in which NOx titration of ozone can be significant, zero-out modeling can
239   result in inflated estimates of background ozone when these NOx emissions are completely and
240   unrealistically removed. Paradoxically, in certain times and locations in a zero-out scenario there can be
241   more background ozone than actual ozone within the model (EPA, 2014).

242   A separate modeling technique attempts to circumvent these limitations by apportioning the
243   total ozone within the model to its contributing source terms. This basic approach is referred to as
244   "source apportionment" modeling. While source apportionment modeling has not been previously used
245   in the context of estimating background ozone levels as part of an ozone NAAQS review, it has
246   frequently been used in other regulatory settings to estimate the "contribution" to ozone of certain sets
247   of emissions (EPA 2005, EPA 2011). The source apportionment technique provides a means of
248   estimating the contributions of user-identified source categories to ozone formation in a single model
249   simulation. This is achieved by using multiple tracer species to track the fate of ozone precursor
250   emissions (VOC and NOx) and the ozone formation caused by these emissions. The methodology is
251   designed so that all ozone and precursor concentrations are attributed to the selected source categories
252   at all times without perturbing the inherent chemistry. The zero out modeling attempts to determine
253   what ozone be in the absence of background sources. The source apportionment modeling attempts to
254   determine how much of the modeled ozone has resulted from background sources. EPA has conducted
255   and will describe new source apportionment modeling that employs a regional air quality model nested
256   within a coarser-scale global chemical transport model to assess the contributions of boundary
257   conditions and other potential background sources (e.g., wildfires, biogenic emissions, and
258   Canadian/Mexican emissions). This modeling is described in detail in section 2b.

259   a. 2007 GEOS-Chem/CMAQ zero-out modeling:

260   In order to provide estimates of the overall fraction of ozone that is estimated to result from
261   background sources in each of the 12 REA urban study areas, EPA conducted new modeling that utilized
262   the same model base year (2007) as was used in the ozone modeling that inform the risk and exposure
263   analyses (EPA, 2014, Appendix 4b). The EPA modeling used a model configuration similar to that of
264   Emery (2012), in that it nested a regional-scale (12 km) air quality model inside a global air quality model

265   simulation with a much coarser horizontal grid resolution (2.0 by 2.5 degrees). Figure 1a shows a map of
266   the model domain.

267   The global scale simulation utilized the GEOS-Chem model, version v8-03-02, except for the
268   chemistry package which was from version v8-02-01. The emissions estimates used in the 2007 base
269   year modeling were aggregated from a variety of sources, starting with the global Emissions Database
270   for Global Atmospheric Research (EDGAR) emission inventory. These initial estimates were then
271   improved by utilizing various area-specific inventories, such as the 2005 National Emissions Inventory
272   (NEI) for the U.S. portions of the domain, and available inventories for Asia, Canada, Europe, and
273   Mexico. In addition to the anthropogenic estimates, emissions were specified for a variety of
274   background sources including: lightning NO, soil NOx, wildfires, and biogenic VOC emissions. The
275   wildfire data is from the Global Fire Emissions Database (GFED). The biogenic VOC estimates were
276   simulated by the Model of Emissions of Gases and Aerosols from Nature (MEGAN) version 2.1. The
277   meteorological data is based on the Goddard Earth Observing System Model, Version 5 (GEOS-5)
278   analysis fields. More information on the global simulation is available within Henderson *et al.* (2013).
279   This reference also provides a broad evaluation of the ability of this specific GEOS-Chem configuration to
280   provide accurate lateral boundary conditions of ozone to finer-scale regional simulations. Using satellite
281   retrievals from the Tropospheric Emissions Spectrometer (TES), Henderson *et al.* (2013) concluded that
282   the GEOS-Chem ozone prediction biases and errors are generally within TES uncertainty estimates. For
283   instance, for the ozone season month of August, model predictions are within plus or minus 20 percent
284   of the satellite estimates between nearly 80 percent of the time, with slightly better performance along
285   the southern boundary.

286   The lateral boundary conditions from the global model were then used as inputs for a 12 km
287   horizontal resolution, CMAQ version 4.7.1, model simulation. Four scenarios were modeled: 1) a 2007
288   base case simulation which was the basis of the air quality modeling performed for the 2nd draft ozone
289   REA and is described in more detail in Appendix 4b of EPA (2014), 2) a natural background run with
290   anthropogenic ozone precursor emissions[2] removed in both the global and regional models, 3) a North
291   American background run with anthropogenic ozone precursor emissions removed across North
292   America (global and regional model simulations), and 4) a U.S. background run with anthropogenic
293   ozone precursor emissions were removed over the U.S (global and regional model simulations).
294   Detailed analyses of EPA's 2007 zero out modeling results are provided in sections 3 through 6 of this
295   appendix.

296   An operational model performance evaluation was completed for surface ozone in the 2007
297   base simulation as described separately (EPA 2014, Appendix 4b).   For the purposes of this analysis, EPA
298   assessed the model ability to reproduce measured daily maximum 8-hour (MDA8) ozone values and
299   seasonal mean MDA8 ozone concentrations for the period April to October 2007. As noted earlier, the
300   base year modeling in this analysis used climatological monthly-average wildfire emissions which are not

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[2] In the global model all ozone precursor species were removed (i.e., VOC, NOx, CO), except for methane which
was reset to pre-industrial levels to reflect natural contributions. In the regional modeling, the methane levels
were left unchanged.

301   intended to capture discrete events from specific fires that occurred in 2007, so perfect correlation
302   between observations and model predictions should not be expected. Figure 1b provides a density
303   scatterplot of the observed and predicted daily 8-hour ozone peaks paired in space and time for the
304   2007 CMAQ base. As can be seen, the majority of pairs line up along the 1:1 line. There is a tendency
305   for the model to overestimate site-days with low 8-hour ozone peaks, and underestimate the site-days
306   with higher peak ozone values. Modeled 8-hour ozone peak concentrations exhibited relatively small
307   bias and error compared to the observations. The average bias in MDA8 ozone estimates was 3.5 ppb.
308   Figure 1c depicts the spatial bias patterns in MDA8 ozone at all sites that measured valid ozone data for
309   at least 100 days during the April-October period. CMAQ overestimations are greatest along the Gulf
310   Coast region, along the Atlantic coastline, and over the central U.S. The majority of underestimated
311   seasonal mean MDA8 occurs within southern California. The model performance for the 2007 base
312   simulation is equivalent or better than typical state-of-the-science photochemical model performance
313   recently reported in the literature (Simon et al, 2012).

314   Certainly some remote monitoring locations are more affected by background sources than
315   other locations in the network. However, this and numerous other analyses have shown that even the
316   most remote ozone monitoring locations in the U.S. are periodically affected by U.S. manmade
317   emissions. In this analysis we carefully assess model performance to ensure that model error does not
318   influence the characterization of background ozone. As noted in the recent ISA (EPA, 2013), there is
319   greater confidence in the ability of the model to predict mean contributions from background sources
320   rather than individual events. Beyond the statistical analyses summarized in the previous paragraph and
321   in appendix 4b of the 2nd draft ozone REA (EPA, 2014), it is valuable to attempt to diagnose the model
322   ability to account for background ozone within the simulation. EPA assessed whether any correlation
323   existed between daily model biases and daily background ozone estimates. Figure 1d shows that at
324   high-elevation sites (i.e., sites more than 1km above sea level) the highest estimates of natural
325   background ozone tend occur on days with greatest overestimation. Conversely, the site-days with the
326   lowest natural background estimates tend to occur when the model underestimates the observed daily
327   peaks at these sites. This relationship between background estimates and simulation bias appears to be
328   constrained to the mountainous portion of the Western U.S. Figure 1d also shows that estimates of
329   natural background ozone greater than 60 ppb are associated with large over-predictions. However,
330   based on the relatively low model bias and the general lack of correlation between daily bias values and
331   background estimates, EPA believes that these model estimates can be used to help characterize
332   background ozone levels over the U.S.   Although the highest background estimates should be
333   considered with caution.

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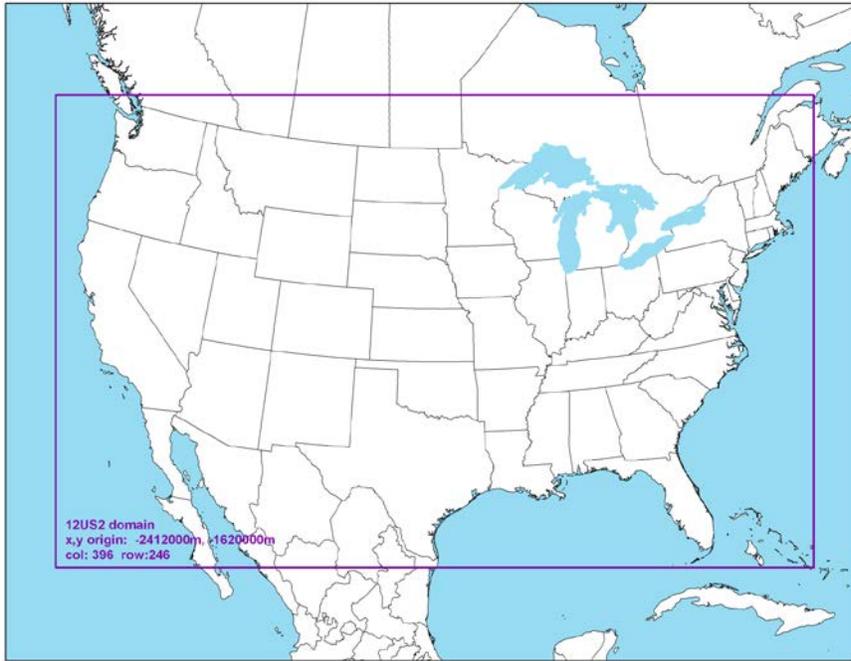
Figure 1a. Modeling domain used in 2007 CMAQ and CAMx modeling.

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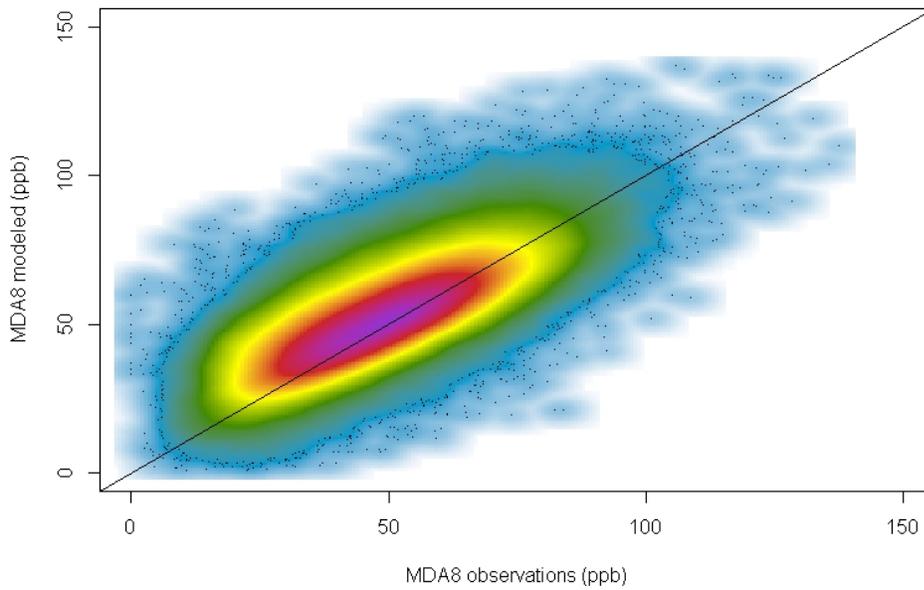
Figure 1b. Density scatterplot comparing CMAQ base daily peak 8-hour ozone predictions against observed 8-hour ozone peaks paired in space and time for all sites during April-October 2007.

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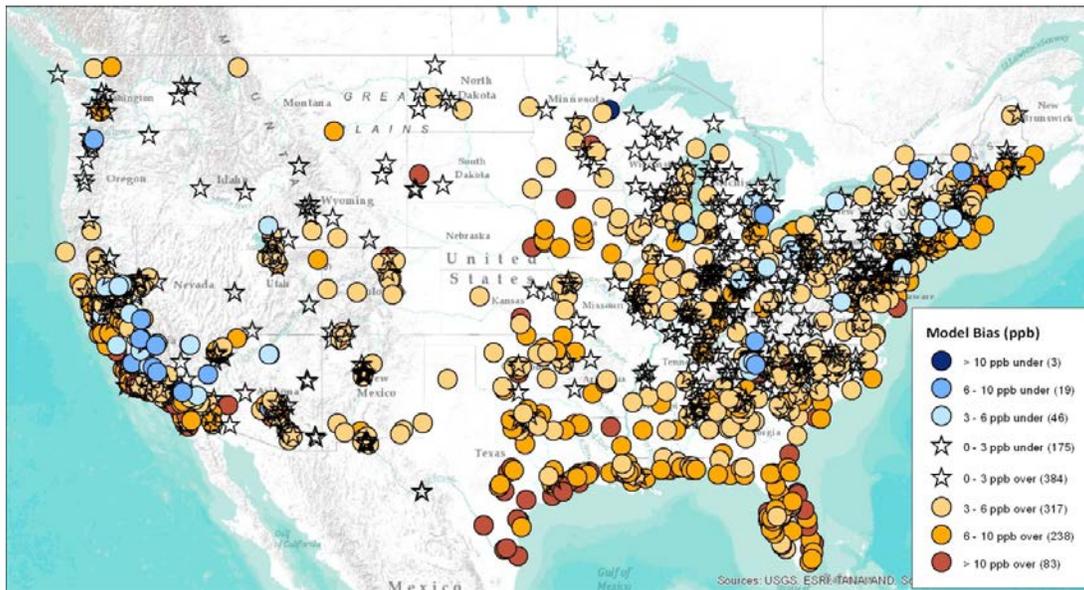
Figure 1c. Bias in seasonal mean (April-October) maximum daily 8-hour ozone predictions in the 2007 CMAQ base simulation.

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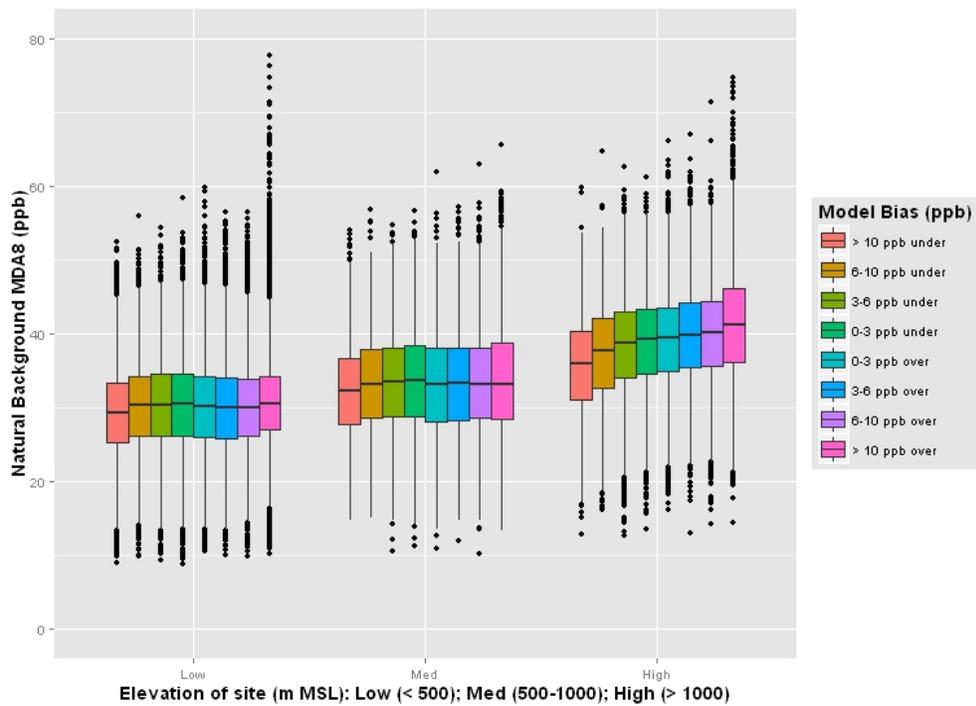
Figure 1d. Relationship between CMAQ estimations of MDA8 natural background ozone and daily model biases.

2A-11

349   b. 2007 GEOS-Chem/CAMx source apportionment modeling:

350   The same global modeling described above was used to assign lateral boundary conditions to
351   the regional-scale (12 km) CAMx v5.0 source apportionment simulations. Wherever possible, the
352   emissions and meteorological inputs in the CAMx modeling were chosen to mimic the 2007 base CMAQ
353   simulation described earlier. Figure 1a shows a map of the model domain.

354   As with the CMAQ base case, a limited operational model performance evaluation was also
355   completed for surface ozone in the 2007 base simulation. For the purposes of this analysis, EPA
356   assessed the model ability to reproduce measured daily maximum 8-hour (MDA8) ozone values and
357   seasonal mean MDA8 ozone concentrations for the period April to October 2007. Figure 2a provides a
358   density scatterplot of the observed and predicted daily 8-hour ozone peaks paired in space and time for
359   the 2007 CAMx base simulation. As can be seen, the majority of pairs line up along the 1:1 line. Again,
360   there is a tendency for the model to overestimate site-days with low 8-hour ozone peaks and
361   underestimate the site-days with higher peak ozone values. Modeled 8-hour ozone peak concentrations
362   exhibited relatively small bias and error compared to the observations. The average bias in MDA8 ozone
363   estimates was 3.5 ppb. Figure 2b depicts the spatial bias patterns in MDA8 ozone at all sites that
364   measured valid ozone data for at least 100 days during the April-October period. CAMx overestimations
365   are greatest along the Gulf Coast region, along the Atlantic and Pacific coastlines, and within the
366   southeastern U.S. The majority of underestimated seasonal mean MDA8 occurs in California away from
367   the coastline.

368   The apportionment tools in CAMx utilized here to estimate the contribution of background
369   sources are well-established and have previously been peer-reviewed (UNC, 2009). EPA used the
370   Anthropogenic Precursor Culpability Assessment (APCA) tool in this analysis. The APCA tool attributes
371   ozone production to manmade sources whenever ozone is determined to result from a combination of
372   anthropogenic and biogenic emissions (Environ, 2011). The APCA methodology defines natural ozone as
373   the production resulting from the interaction of biogenic VOC with biogenic NOx emissions. Eleven
374   separate source categories were tracked in the source apportionment analysis, including five boundary
375   condition terms and six in-domain sectors:

376   • Boundary condition terms:
377   o Northern edge
378   o Eastern edge
379   o Southern edge
380   o Western edge
381   o Top boundary
382
383   • In-domain sectors:
384   o U.S. anthropogenic emissions
385   o Point sources located within the Gulf of Mexico
386   o Category 3 marine vessels outside State boundaries
387   o Climatologically-averaged wildfire emissions

2A-12

388   o   Biogenic emissions

389   o   Canada/Mexico emissions (only those sources within the domain)

390   It should be noted that the source apportionment modeling conducted here does not allow for
391   replication of natural background because of the construct of boundary conditions. The boundary
392   conditions for our applications can include ozone and/or ozone precursors that were originally
393   generated by natural sources, as well as ozone produced from far upstream anthropogenic emissions
394   (e.g., Asia). It is not possible to disentangle these two terms. Instead, the source apportionment
395   modeling is primarily used to help estimate background into the U.S., which is assumed to be the
396   contributions from nine of the modeled sectors; that is, everything except U.S. anthropogenic emissions
397   and point sources located within the Gulf of Mexico.

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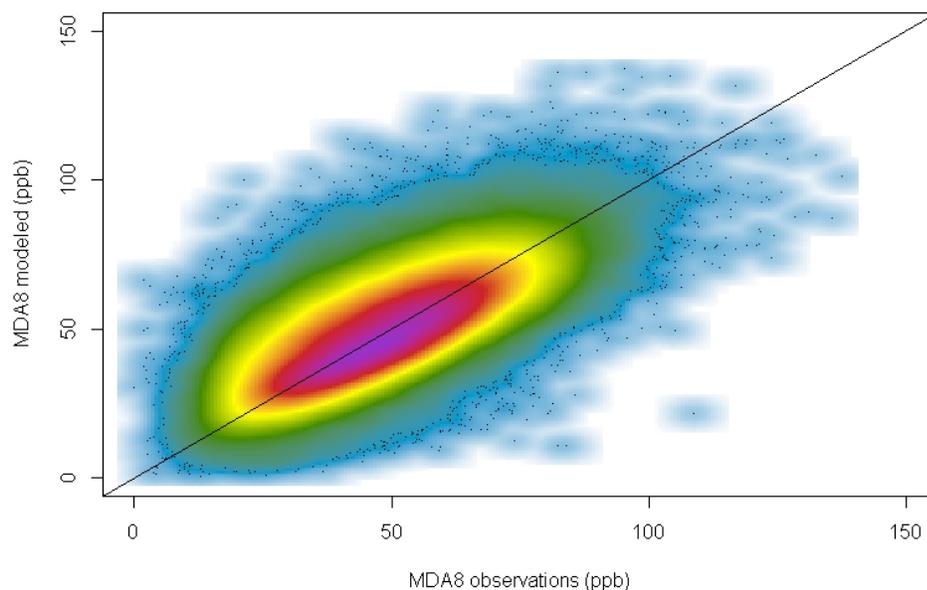
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400   **Figure 2a. Density scatterplot comparing CAMx base daily peak 8-hour ozone predictions against**
401   **observed 8-hour ozone peaks paired in space and time for all sites during April-October 2007.**

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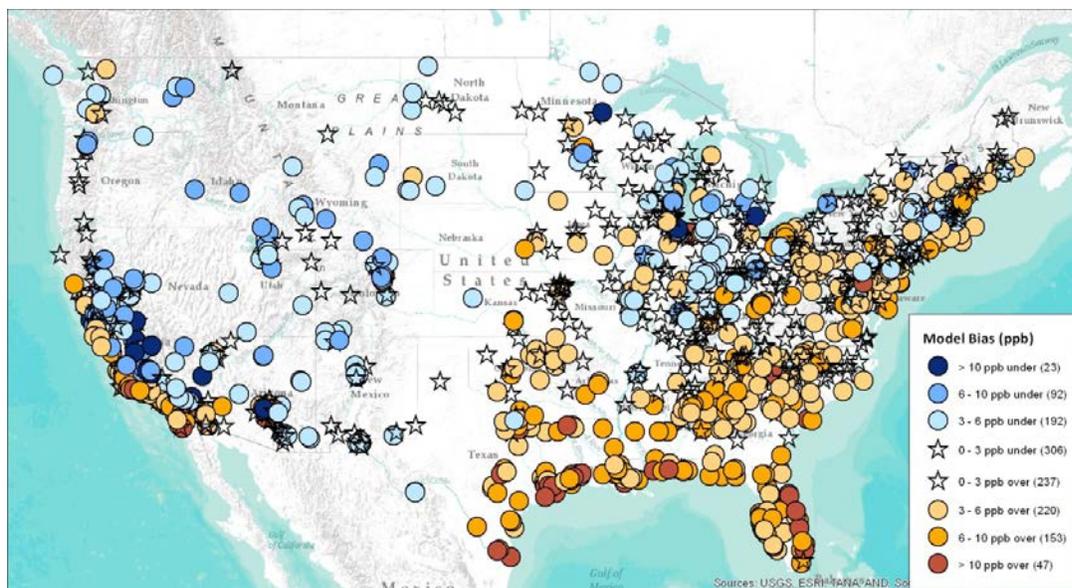
Figure 2b. Bias in seasonal mean (April-October) maximum daily 8-hour ozone predictions in the 2007 CAMx base simulation.

3.   Estimates of seasonal-average background ozone levels

This section of the appendix provides estimates of seasonal average background ozone levels over the U.S. As noted in the introduction and as discussed in detail in the ISA, background ozone values can vary significantly in space and time. There can be atypical episodes of higher background ozone concentrations amidst the routine days that drive seasonal average background. The highest background episodic concentrations are typically associated with stratospheric intrusions or wildfires. These background "events" can be difficult to model as they require event-specific model inputs. The primary goal of the EPA modeling was to estimate the seasonal average background concentrations between April and October 2007. Previous analyses have shown that this is the period in which average background levels are highest (Zhang et al., 2011). This section of the appendix focuses on seasonal mean levels of background. (Section 4 will consider the upper range of possible background ozone.)

The analysis focus on the maximum daily 8-hour ozone average in ppb. This metric is referred to as MDA8. This section will first present model estimates of seasonal mean ozone levels in the base simulation. This will be followed by estimates of NB, NAB, and USB from the CMAQ zero out modeling. After discussing the magnitudes of background levels, the section switches to a consideration of the relative percentage of background to total ozone across the U.S. This portion of the text will utilize both the CMAQ zero out and CAMx source apportionment modeling.

Figure 3a displays the 2007 base case, CMAQ model-predicted, seasonal mean (April-October) MDA8 ozone concentrations in grid cells with active monitoring locations over the U.S. The model results are shown at the monitoring site level as opposed to in the default gridded format to foster subsequent site-level estimates of background magnitudes. Each grid cell containing an Air Quality System (AQS) ozone monitor that was collecting valid data in 2007 was identified and the model

428   background estimates were extracted for those grid cells and displayed accordingly. The base
429   predictions are provided for context to allow easier interpretation of the following plots which isolate
430   specific background levels. As can be seen, most of the U.S. experiences seasonal mean MDA8 ozone
431   levels greater than 50 ppb in the base case simulation. The median value over the 1,294 monitoring
432   locations is 52.5 ppb.

433   Figure 3b provides an estimate of what seasonal-average MDA8 would be in a natural
434   background scenario, using the 2007 EPA zero out modeling. Again, in this GEOS-Chem/CMAQ
435   simulation, all anthropogenic ozone precursor emissions were removed from both the global and
436   regional simulations, and methane levels were adjusted to pre-industrial levels in the global simulation.
437   As shown, natural background ozone levels range from approximately 15-35 ppb with the highest values
438   occurring over the higher-elevation sites in the western U.S. The median value over these locations is
439   24.2 ppb, and more than 50 percent of the sites have natural background levels of 20-25 ppb. The
440   highest modeled estimate of seasonal average, natural background, MDA8 ozone is 34.3 ppb at the
441   high-elevation CASTNET site (Gothic) in Gunnison County, CO.

442   Figures 3c and 3d show the same information for the North American and U.S. background
443   scenarios. In these model runs, all anthropogenic ozone precursor emissions were removed from the
444   U.S., Canada, and Mexico (NAB scenario) and then only the U.S. (USB scenario). The figures show that
445   there is not a large difference between the NAB and USB scenarios. Seasonal mean MDA8 NAB and USB
446   ozone levels range from 25-50 ppb, with the most frequent values estimated in the 30-35 ppb bin. The
447   median seasonal mean background levels are 31.5 and 32.7 ppb (NAB and USB, respectively). Again, the
448   highest levels of background are predicted over the intermountain western U.S. Locations with NAB and
449   USB concentrations greater than 40 ppb are confined to Colorado, Nevada, Utah, Wyoming, northern
450   Arizona, eastern California, and parts of New Mexico. Similar to NB, the highest NAB and USB levels
451   were modeled to occur at the Gothic CO site (46.7/47.7). This remote rural site is located 2,926 meters
452   (9,600 feet) above mean sea level and should not be considered representative of background ozone at
453   lower-altitude, more-populated regions. The high USB and NAB values along the Gulf Coast are most
454   likely due to model biases.

455   Absolute model estimates of various background definitions are useful, but they can be
456   influenced by any local biases and errors in the modeling. A separate way to look at the role of
457   background in seasonal mean ozone levels is to consider the fractional contribution of NB, NAB, and USB
458   to total ozone at each location. Considering the proportional role of background allows for an
459   informative comparison between the two modeling approaches without having to account for the
460   differences in base case biases and errors. Figures 4a, 4b, and 4c show the estimated fractional
461   contribution of NB, NAB, and USB to total seasonal average MDA8 ozone levels at the monitoring
462   locations from the CMAQ zero out modeling. The modeling estimates that approximately 35-80 percent
463   of the seasonal average MDA8 ozone at monitoring locations is due to natural background sources. A
464   majority sites have NB fractions between 40 and 60 percent. The mean natural background proportion
465   over all sites is 47 percent. That is, when all global anthropogenic emissions are removed and global
466   methane levels in GEOS-Chem are restored to pre-industrial levels, seasonal average MDA8 levels are
467   reduced by approximately half. The fractional proportions of NAB and USB are very similar. In both

468   cases, most sites have background fractions that range from 50 to 80 percent. The mean NAB fraction
469   (to seasonal mean MDA8) is 63 percent. The mean USB fraction is 66 percent.

470   As noted in the introduction, the advantage of the source apportionment modeling is that all of
471   the modeled ozone is attributed to various source terms and thus this approach is not affected by the
472   confounding occurrences of background ozone values exceeding the base ozone values as can happen in
473   the zero out modeling (i.e., background proportions > 100%). Consequently, one would expect the
474   fractional background levels to be lower in the source apportionment methodology as a result of
475   removing this artifact. It is also important to remember that the terms NB, NAB, and USB are explicitly
476   linked to the zero out modeling approach. (USB is the ozone that would exist in the absence of U.S.
477   anthropogenic emissions.) In contrast, the source apportionment modeling performed here provides
478   estimates the amount of MDA8 ozone that is attributable to U.S. anthropogenic emissions relative to
479   total base model ozone. Figure 4d shows the relative contribution from **sources other than U.S.**
480   **anthropogenic emissions** to total seasonal mean MDA8 ozone based on the 2007 source apportionment
481   modeling. The fractional contribution fields between CMAQ zero out USB estimates and CAMx source
482   apportionment estimates of source other than U.S. anthropogenic emissions are quite similar. The
483   spatial patterns in Figures 4c and 4d are consistent, with the highest fractional contributions from
484   sources other than U.S. anthropogenic emissions occurring along U.S. borders and over the
485   intermountain western States. The source apportionment modeling estimates that approximately 40-
486   80% of the seasonal average MDA8 ozone at monitoring locations is due to sources other than
487   manmade ozone precursor emissions from the U.S. A majority of sites have non-U.S. fractions between
488   40 and 70 percent. The mean proportion attributable to international and natural sources over all sites
489   is 59 percent. Despite the differences in the methodologies this is very similar to the mean USB
490   estimate of 66 percent from the zero out modeling.

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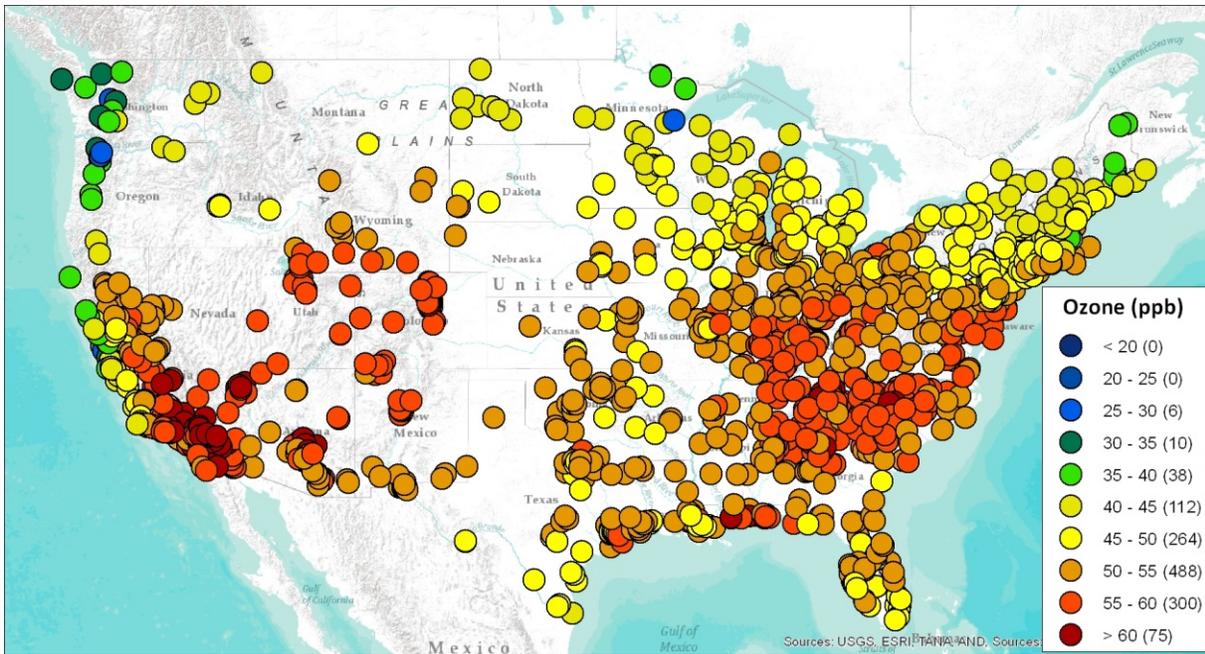
Figure 3a. April-October average MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ base simulation.

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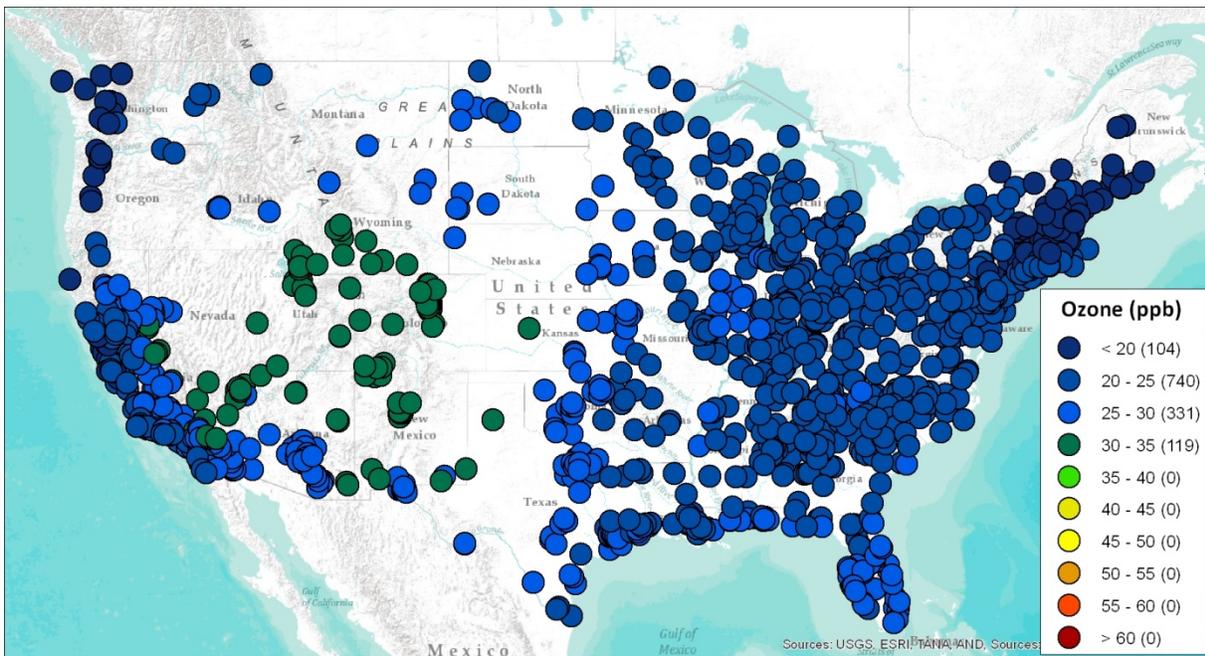
Figure 3b. April-October average *natural background* MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ zero out simulation.

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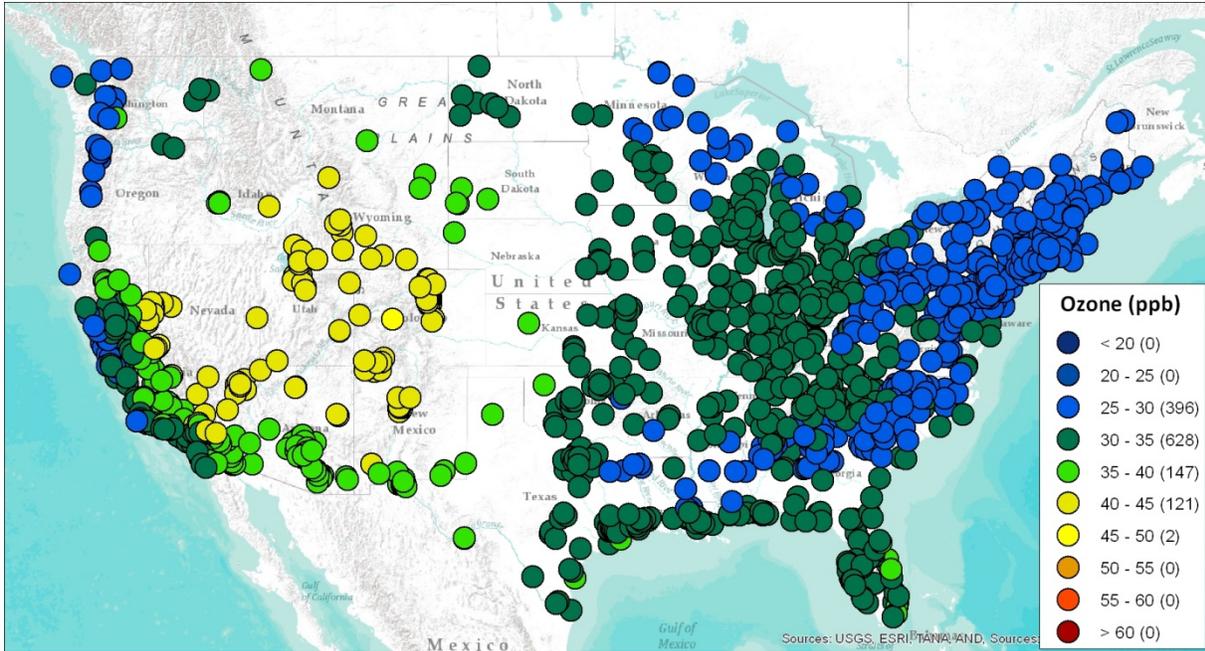
Figure 3c. April-October average *North American background* MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ zero out simulation.

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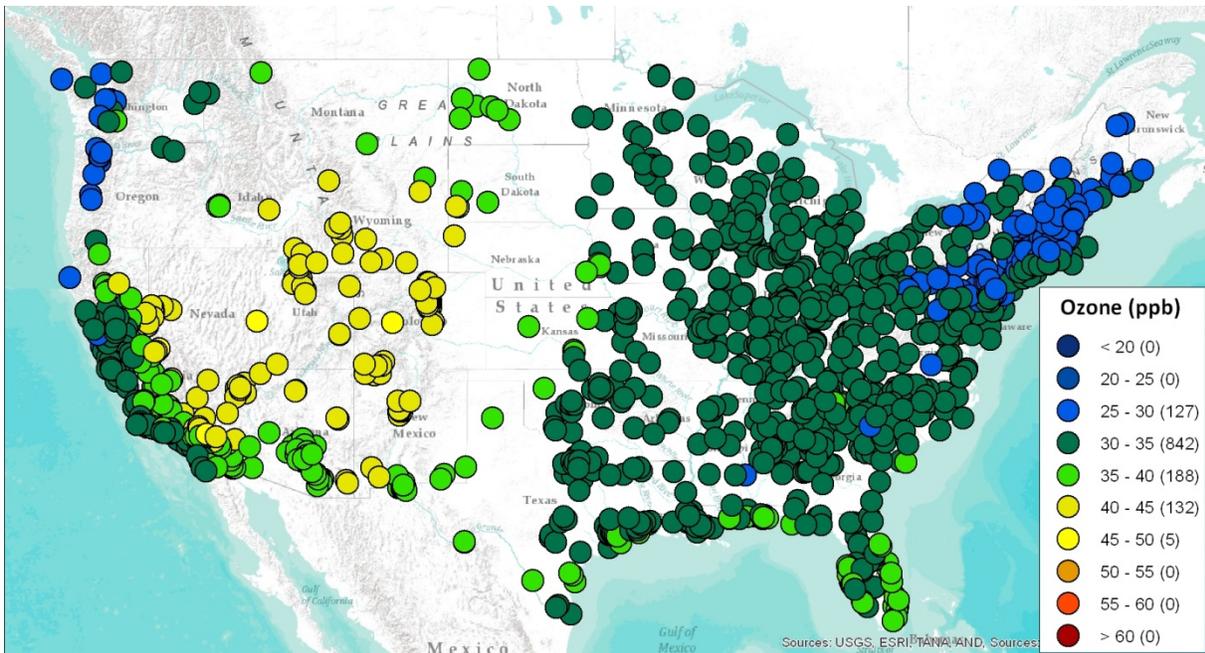
Figure 3d. April-October average *United States background* MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ zero out simulation.
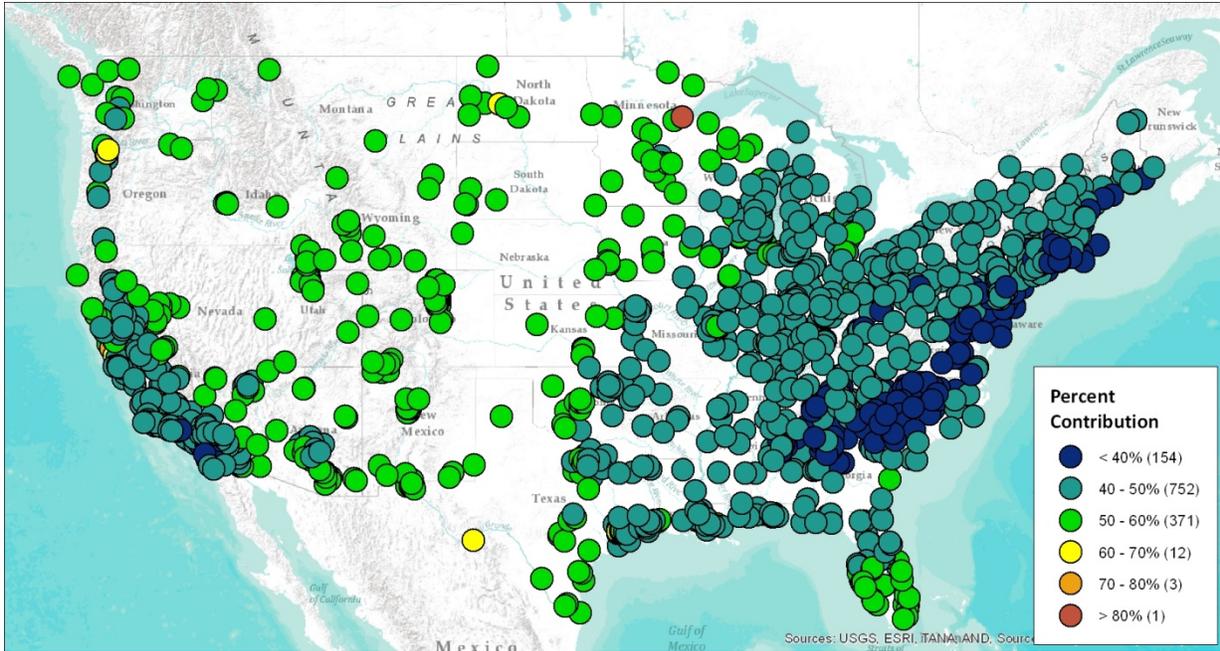
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511   **Figure 4a. Ratio of *natural background* to total April-October average MDA8 ozone at monitoring**
512   **locations across the U.S. as estimated based on 2007 CMAQ simulations.**

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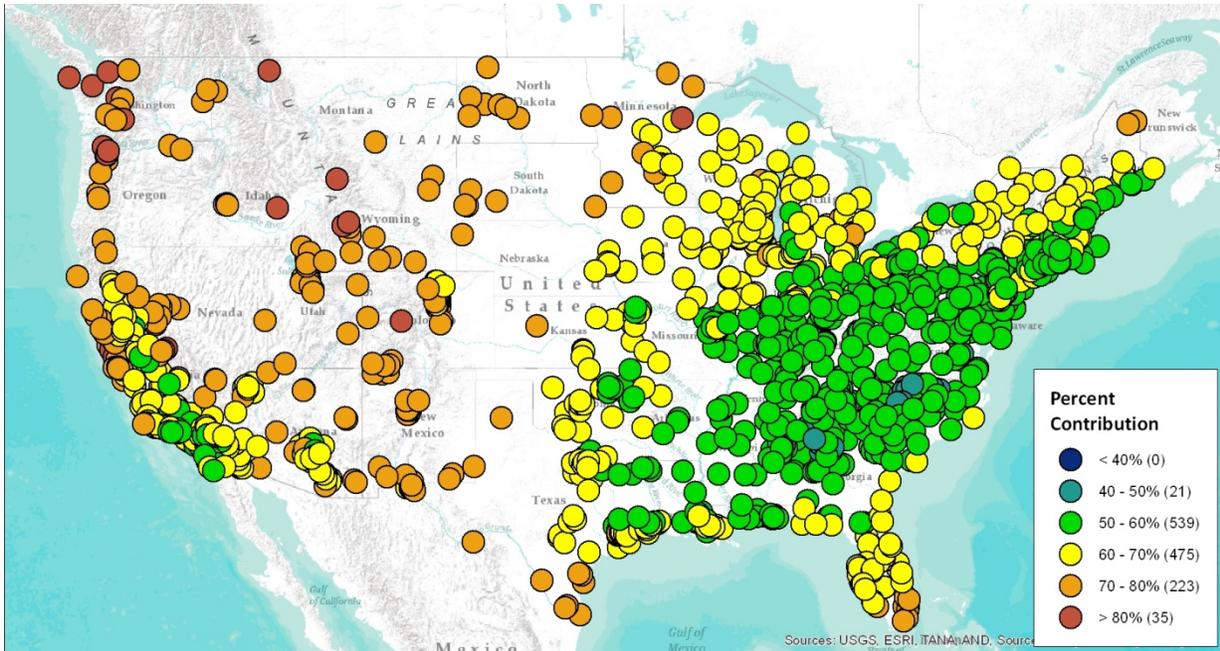
515

516   **Figure 4b. Ratio of *N. American background* to total April-October average MDA8 ozone at**
517   **monitoring locations across the U.S. as estimated based on 2007 CMAQ simulations.**

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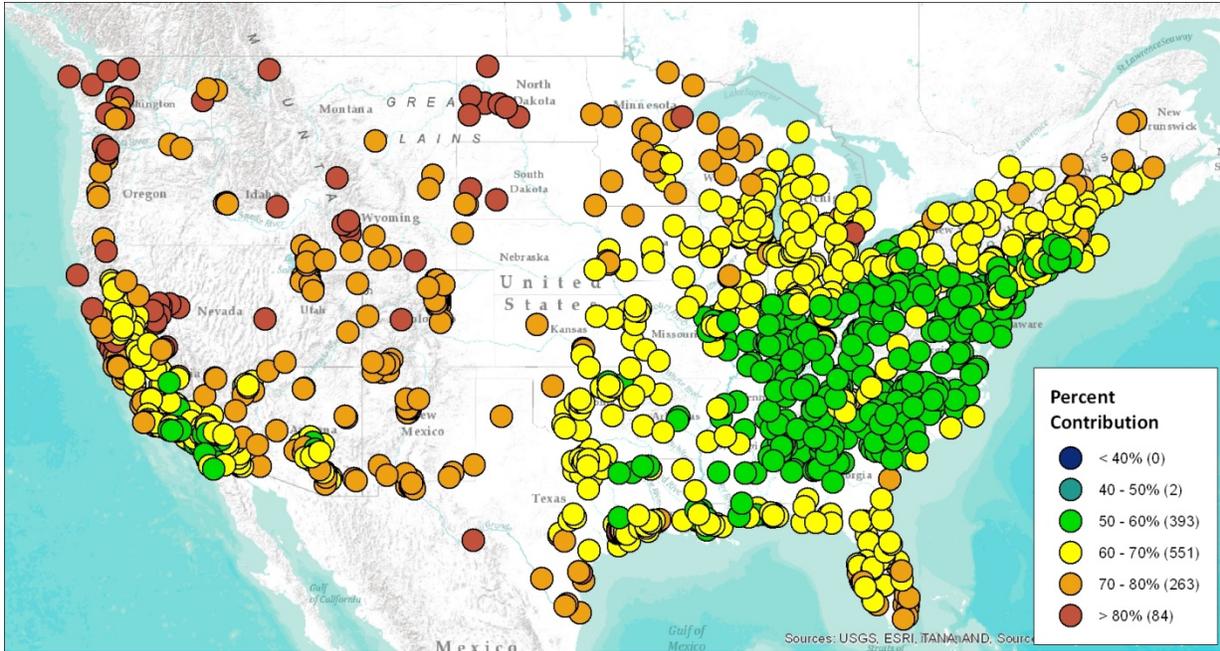
Figure 4c. Ratio of *U.S. background* to total April-October average MDA8 ozone at monitoring locations across the U.S. as estimated based on 2007 CMAQ simulations.

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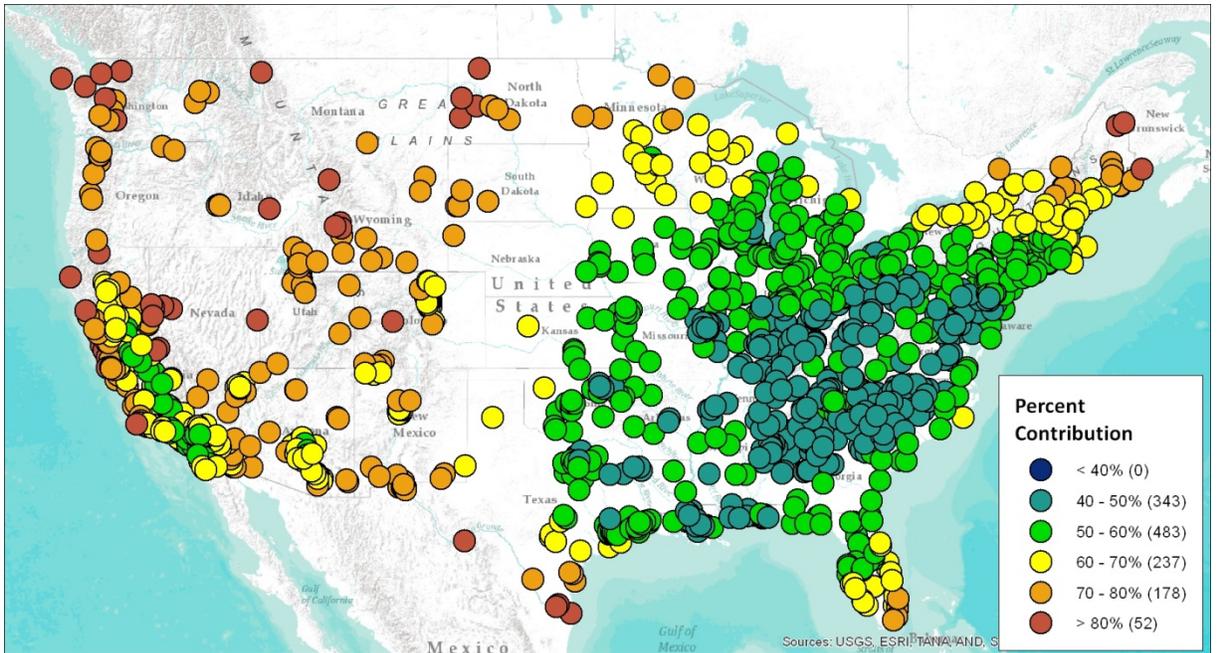
Figure 4d. Ratio of *sources other than U.S. anthropogenic emissions* to total April-October average MDA8 ozone at monitoring locations across the U.S. as estimated by a 2007 CAMx source apportionment simulation.

526 **4.   Distributions of background ozone levels**

527   As a first-order understanding, it is valuable to be able to characterize seasonal mean levels of
528 background ozone. However, it is well established that background levels can vary substantially from
529 day-to-day. From an implementation perspective, the values of background ozone on possible
530 exceedance days is a more meaningful distinction. The first draft policy assessment (EPA, 2012)
531 considered this issue in detail, via summaries of the existing 2006 zero out modeling (Henderson et al.,
532 2012), and concluded that "results suggest that background concentrations on the days with the highest
533 total ozone concentrations are not dramatically higher than typical seasonal average background
534 concentrations." Based on this finding, the 1st draft policy assessment determined that "anthropogenic
535 sources within the U.S. are largely responsible for 4th highest 8-hour daily maximum ozone
536 concentrations." This portion of the appendix will consider the entire spectrum of variable background
537 ozone levels with special emphasis on days in which base model ozone concentrations approach or
538 exceed the level of the NAAQS.

539   The 2007 modeling agrees with the finding from the previous 2006-based modeling analyses
540 that the highest modeled ozone site-days tend to have background ozone levels similar to mid-range
541 ozone days. Figures 5a-5c show the distribution of April-October MDA8 background levels (NB, NAB,
542 and USB, respectively) from the CMAQ zero out runs. As noted in section 2, zeroing out emissions can
543 remove the effects of local NOx titration and result in modeled background values that are higher than
544 the base model ozone. The "box and whisker" plots shown in these figures display four key features of
545 the distributions:

546   a. the median concentration (black horizontal line) per bin,
547
548   b. the inter-quartile range (blue colored box) which represents the 25th-75th percentile range in
549   values within the distribution,
550
551   c. the "whiskers" (dark gray vertical lines with top and bottom whiskers) which represent the
552   range of values within 1.5 times the inter-quartile range, and
553
554   d. the "outliers" (gray points) which are any values outside the whiskers.

555   As can be seen in Figure 5a, natural background values do not vary greatly as a function of the
556 base modeled ozone. Recall that the seasonal average natural background MDA8 ozone values were
557 modeled to range from 15-35 ppb across the U.S. with a median value of 24 ppb. The highest values
558 were at the high-elevation sites in the western U.S. Based on the distributional analysis, the 75th
559 percentile values are on the order of 30 ppb. Natural background levels exceeding 40-45 ppb are
560 considered to be statistical outliers, due to their infrequency. Figure 5b shows the same type of
561 distributions but for NAB instead of NB. NAB values are generally 6-12 ppb higher than their NB
562 counterparts, due to the affect of higher global methane values and the influence of anthropogenic
563 emissions from Asia. It was previously reported (in section 3) that the median seasonal average NAB
564 MDA8 values were 31.5 ppb. Based on the distributions, it can be seen that 75th percentile values are

565   approximately 40 ppb; it is rare for NAB MDA8 values to exceed 50-55 ppb. NAB values are constant in
566   magnitude once the base ozone exceeds 50 ppb indicating that the higher base ozone values are driven
567   by non-NAB sources (i.e., North American emissions). Finally in Figure 5c, the USB MDA8 distributions
568   by base model MDA8 are shown. The results are similar to NAB.

569   Figure 5d shows the results from the source apportionment modeling of non-U.S. anthropogenic
570   source contributions to MDA8 ozone (i.e., the nine source apportionment categories other than U.S.
571   anthropogenic emissions and Gulf of Mexico point sources). This non-counterfactual approach is
572   expected to give a better indication of background levels at low concentrations. At low levels, almost all
573   of the ozone is determined to be from background origins. The CAMx modeling shows that
574   contributions from non-U.S. anthropogenic emissions peak when base ozone ranges from 45-55 ppb and
575   then drop off slightly at higher base MDA8 values. The source apportionment modeling of non-U.S.
576   impacts (similar to USB) indicates slightly lower background levels than the zero out modeling. The 75th
577   percentile values are generally less than 35 ppb, compared to 40 ppb in the zero out modeling. It is rare
578   to have background impacts greater than 55ppb. Interestingly, when base model MDA8 ozone exceeds
579   70 ppb, it is rare to have background impacts greater than 45 ppb in the CAMx source apportionment
580   modeling.

581   Figures 6a-6d show the equivalent plots as 5a-5d, but use background fractions (background
582   MDA8 / base MDA8) as the dependent variable instead of the absolute background concentrations.
583   These plots show the same effect; that is, the proportional relative contribution of background sources
584   and processes decreases as peak ozone increases. For natural background (Figure 6a), the median
585   fractions drop from 50% background for values between 45-50 ppb to only 35% background for base
586   MDA8 values between 70-75 ppb. For NAB and USB (Figures 6b and 6c), the median fractions drop from
587   70% background for values between 45-50 ppb to only 45% background for base MDA8 values between
588   70-75 ppb. The source apportionment modeling (Figure 6d) estimates less of a proportional role of non-
589   U.S. anthropogenic emissions. In that modeling, the median fractions drop from 65% background for
590   values between 45-50 ppb to only 35% background for base MDA8 values between 70-75 ppb. A key
591   observation, as noted in the first draft policy assessment document, is that the relative importance of
592   background decreases on days most likely to violate the NAAQS. An additional policy-relevant finding
593   from the distributional analyses is that the relative role of background sources would be increased if the
594   level of the NAAQS were lowered. At 60 ppb, the modeling suggests that the median fractional
595   contribution from background is 45-55 percent, but there can be cases where background comprises 80-
596   90 percent of the total ozone.

597   Many of the cases when background ozone is estimated to contribute in large proportions to
598   relatively high ozone days may be eligible for consideration as exceptional events, but again, this
599   modeling is not designed to resolve specific events that occurred in 2007. While there is greater
600   confidence in the model's ability to predict mean contributions from background sources than from
601   individual events, it is also useful to briefly consider the upper end of the background ozone

602   distributions. Figure 7 shows the 95th percentile[3] USB estimates from the zero out modeling. The 95th

603   percentile MDA8 USB ozone levels range from 35-60 ppb, with the most frequent values residing in the

604   35-40 and 40-45 ppb bins. The median 95th percentile background USB ozone level is 42.0 ppb. As with

605   the seasonal mean MDA8 USB, the highest levels of high background days (i.e., 95th percentile days) are

606   observed over the intermountain western U.S. At these locations, 95th percentile USB levels can exceed

607   50 ppb. Background values at the 95th percentile end of the distribution are 4-12 ppb higher than the

608   mean background values at the same locations.

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[3] During the April-October period, there were 214 days of modeling results. Thus, the 95th percentile values
represent approximately the 10th highest days from the distribution.

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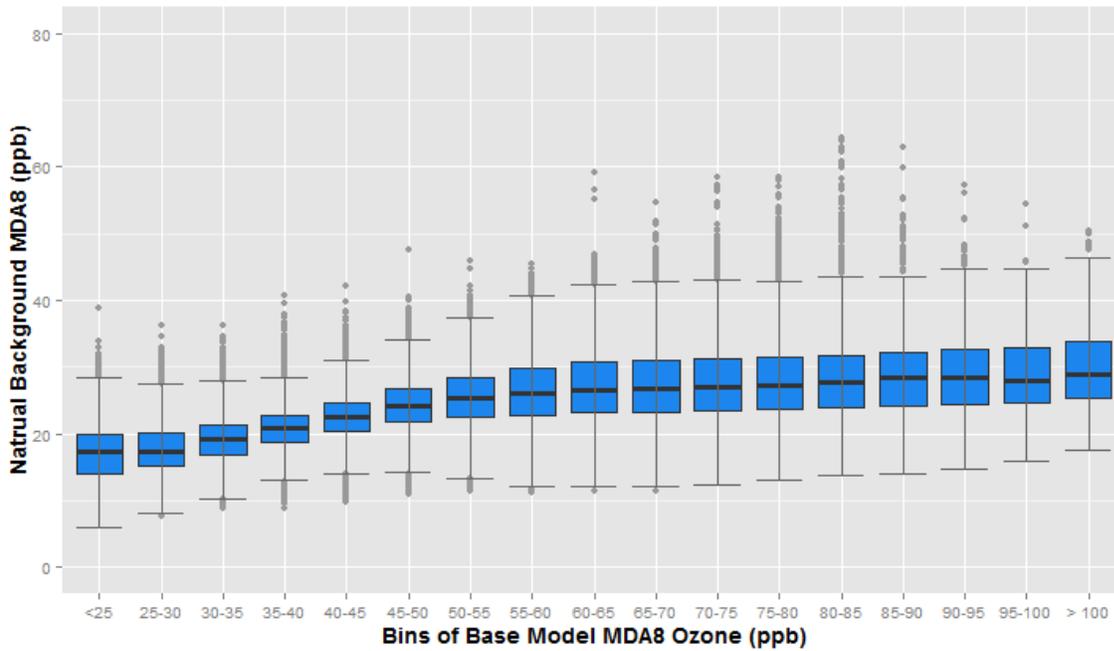
611

612 **Figure 5a. Distribution of *natural background* MDA8 ozone (ppb) at monitoring locations across the**
613 **U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.**
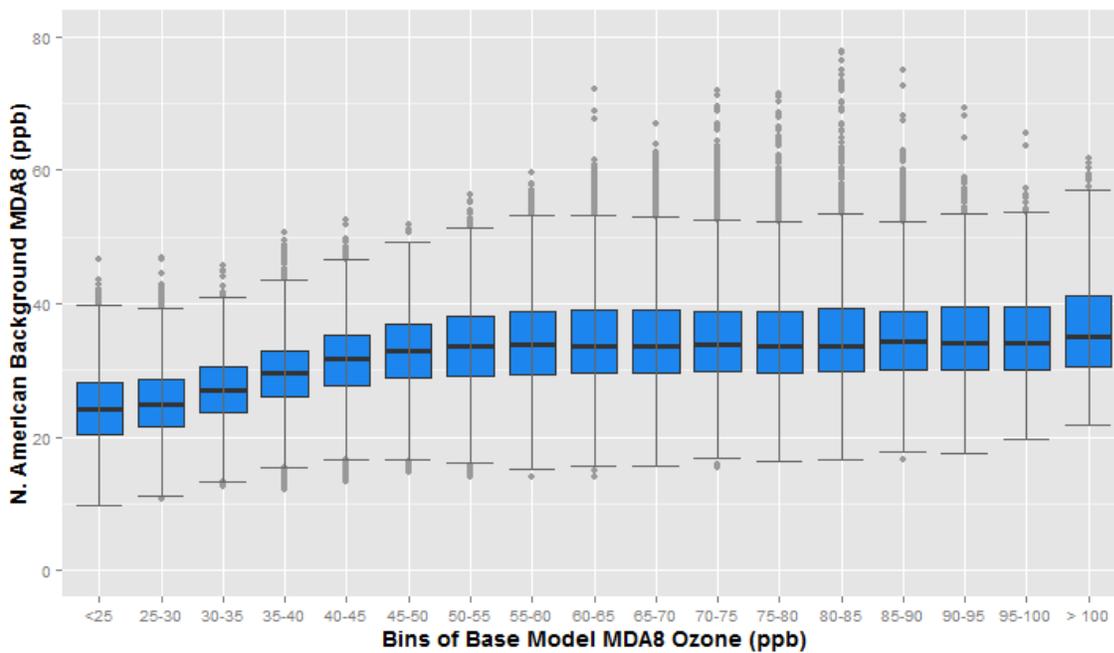
614

615 **Figure 5b. Distribution of *N. American background* MDA8 ozone (ppb) at monitoring locations across**
616 **the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.**

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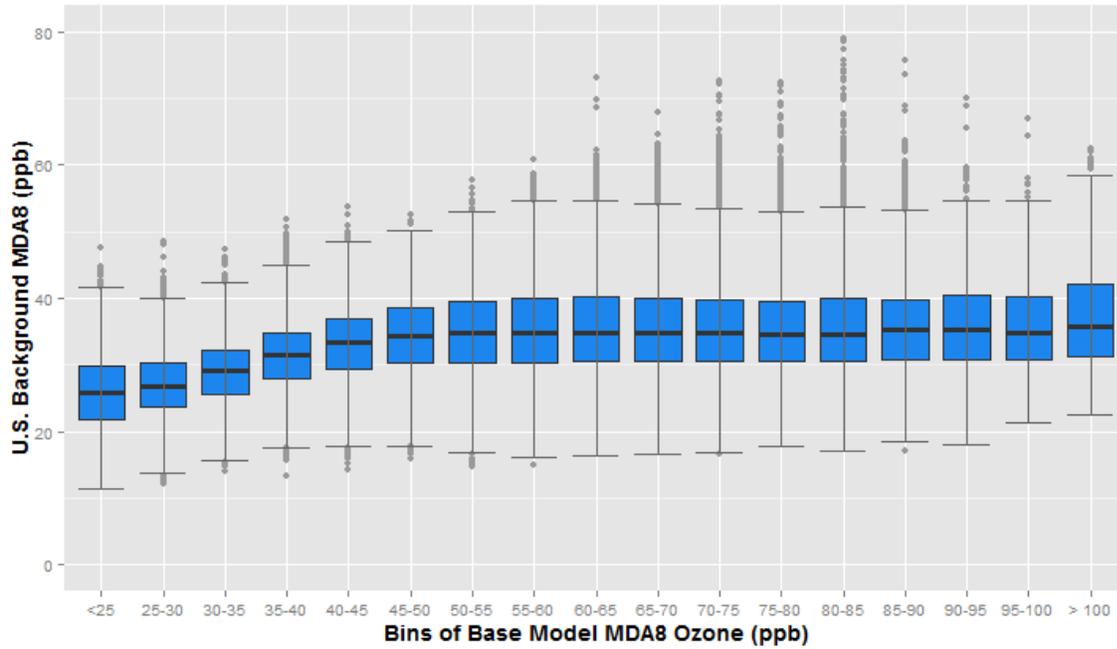
Figure 5c. Distribution of *U.S. background* MDA8 ozone (ppb) at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

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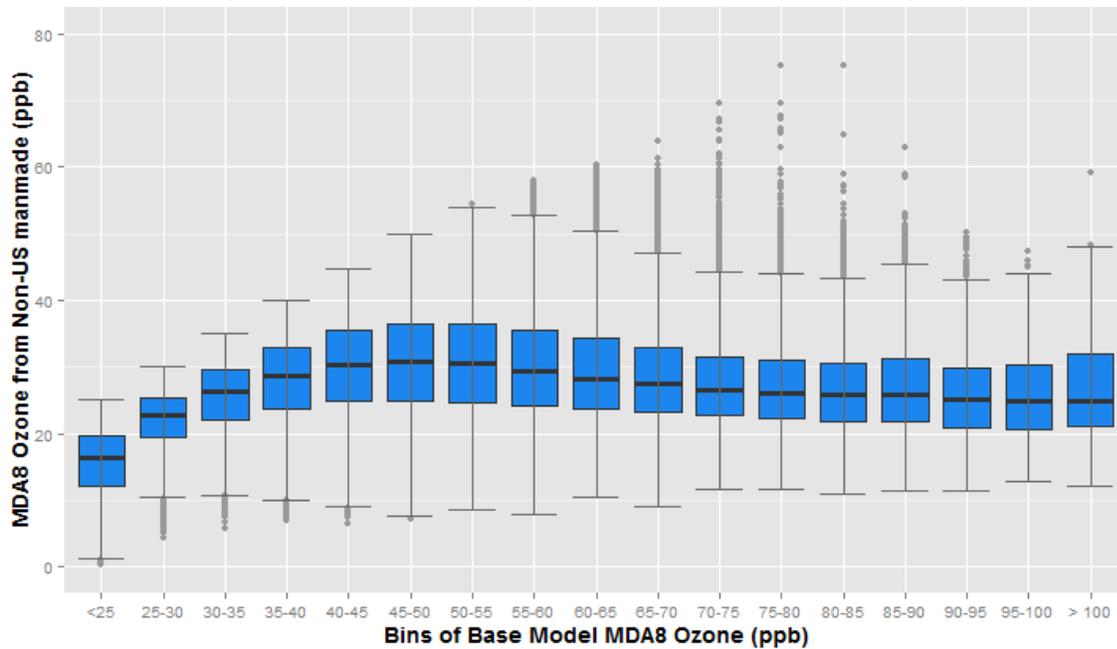
Figure 5d. Distribution of MDA8 ozone contributions from non-U.S. manmade sources (ppb) at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CAMx simulations.

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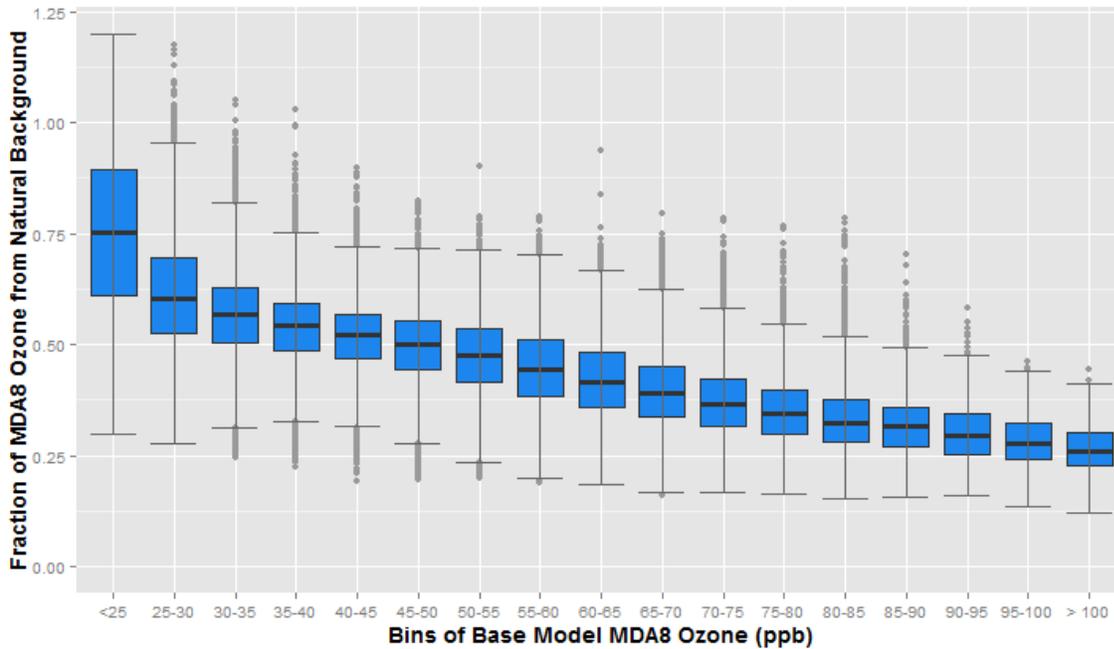
Figure 6a. Distribution of *natural background* MDA8 ozone fractions at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

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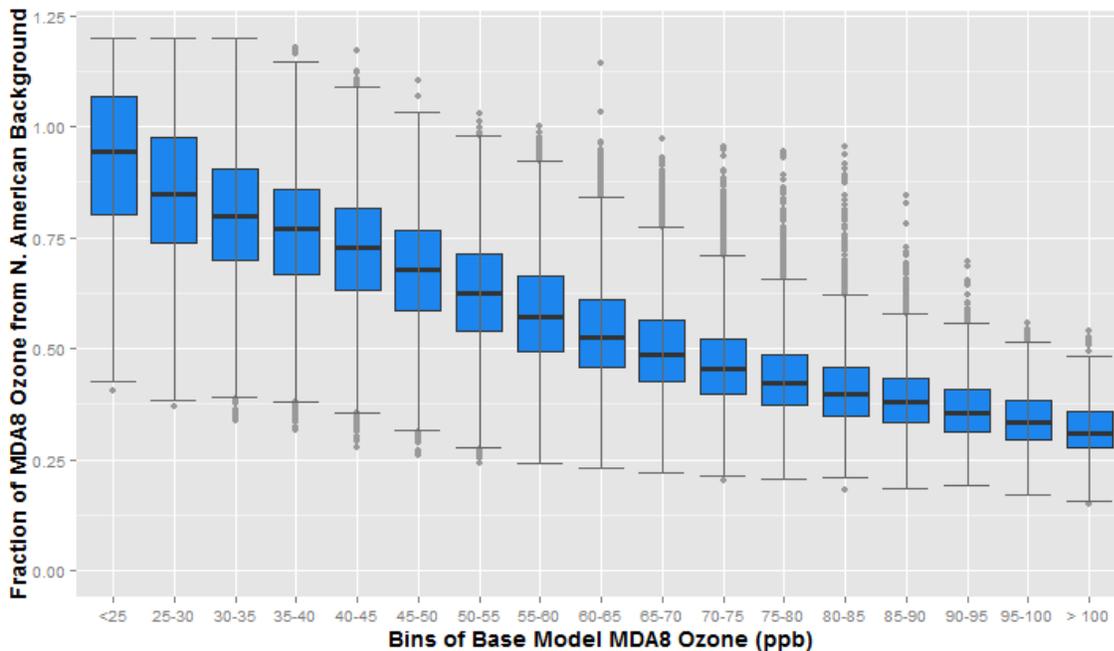
Figure 6b. Distribution of *N. American background* MDA8 ozone fractions at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

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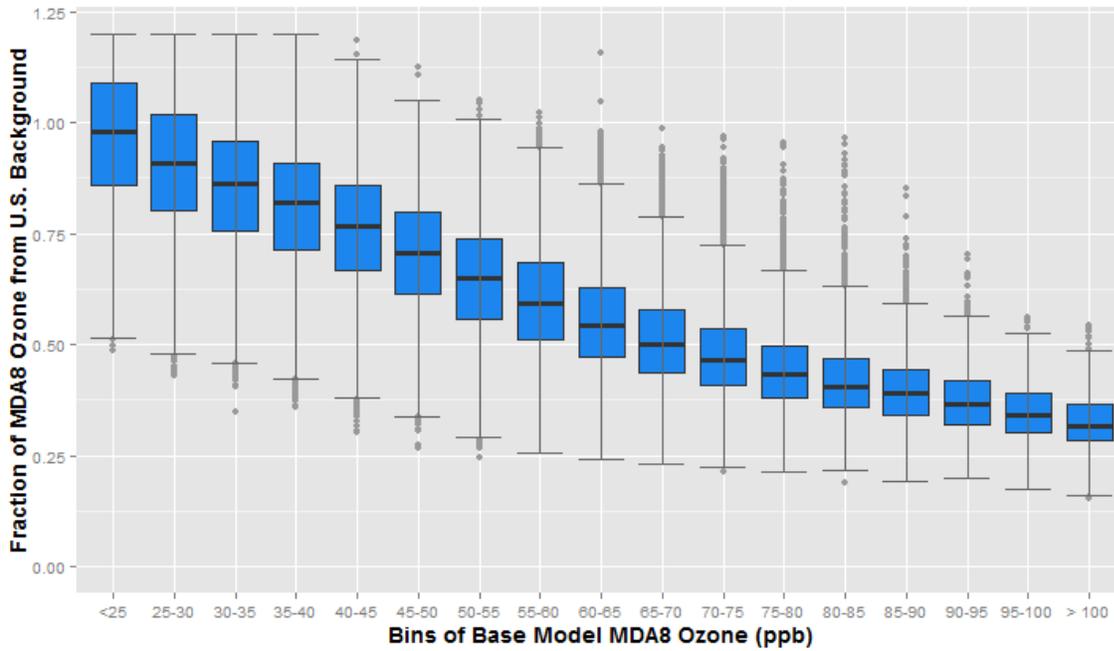
634

635 **Figure 6c. Distribution of *U.S. background* MDA8 ozone fractions at monitoring locations across the**

636 **U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.**
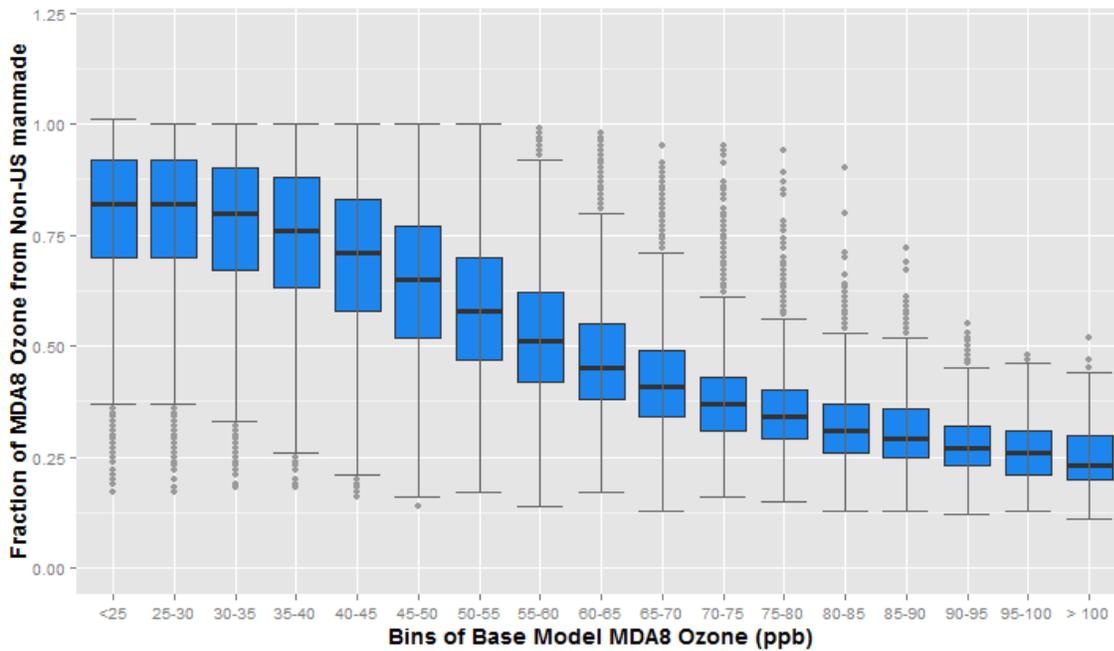
637

638 **Figure 6d. Distribution of MDA8 ozone fractions *from non-U.S. anthropogenic sources* at monitoring**

639 **locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by the 2007**

640 **CAMx simulation.**

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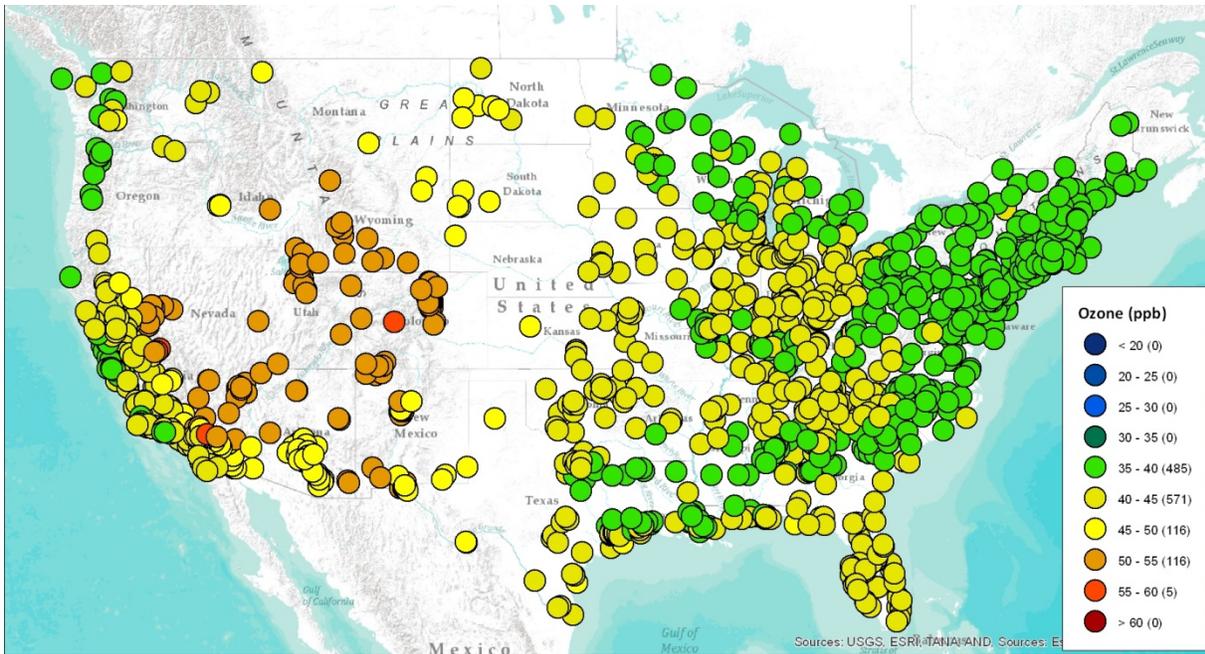
Figure 7. April-October 95th percentile *United States background* MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ base simulation.

647 **5.   Contribution of various processes and sources to total background ozone**

648   This section will utilize the supplemental 2007 air quality modeling estimates to determine the
649 relative importance of specific elements of background ozone. Comparing the differences between the
650 three zero out scenarios can provide some information about the role of certain sets of emissions.
651 Figure 8a compares the NAB (zero out North American manmade emissions) and USB (zero out U.S.
652 manmade emissions) scenarios. The difference between these two runs is the inclusion of
653 anthropogenic emissions within the Canada and Mexico portions of the modeling domain. These
654 emissions contribute less than 2 ppb to the seasonal mean MDA8 ozone levels over most of the U.S.
655 There are 70 sites, near an international border, where the modeling estimates Canadian/Mexican
656 seasonal average impacts of 2-4 ppb. While not shown, the modeled peak single day impacts from
657 these specific international emissions sources can approach 25 ppb (e.g., San Diego, Buffalo NY). Figure
658 8b compares the NB (zero out all manmade emissions and reset GEOS-Chem methane values to pre-
659 industrial levels) to the NAB. The difference between these two runs is the inclusion of global methane
660 emissions related to recent human activity as well as anthropogenic emissions outside of North America.
661 These emissions are estimated to contribute 6-15 ppb to seasonal mean ozone levels over the U.S. The
662 most frequent bin is the 8-10 ppb increase. It is not possible via these runs to parse out what fraction of
663 this change is due to international emissions as opposed to methane emissions, but the ISA summarized
664 existing modeling (Zhang *et al.*, 2011) that suggested that the rise in methane from pre-industrial levels
665 to present-day levels led to increases in seasonal average ozone levels of 4-5 ppb. The greatest impacts
666 from these sources occurs over the western U.S., where international emissions would be expected to
667 have the largest impacts.

668   Figures 9a-9g show the fractional contribution to total seasonal mean MDA8 values of
669 individual source sectors that were tracked in the CAMx source apportionment modeling. Figure 9a
670 shows the impact from the regional model boundary conditions. The ozone entering the model domain
671 via the boundary conditions could have a variety of origins including: a) natural sources of ozone and
672 ozone precursors (including methane) emanating from outside the domain, b) anthropogenic sources of
673 ozone precursors (including methane) from international emitters, and c) some fraction of U.S.
674 emissions (natural and anthropogenic) which are exported and then re-imported into the domain via
675 synoptic-scale recirculation. Thus, one should not presume that the boundary condition contribution is
676 directly tied to any particular background definition. At most locations, boundary conditions
677 contributed 40-60 percent of the total MDA8 seasonal mean at sites across the U.S. The highest
678 proportional impacts from the boundary conditions (the top boundary contributes negligibly) are along
679 the coastlines and the intermountain West.

680   Figure 9b shows the source apportionment contribution (to seasonal mean MDA8) from the
681 most significant sector that was tracked: U.S. anthropogenic ozone precursor emissions. Again the most
682 common outcome at an individual site was that 40-60% of the seasonal mean ozone values originated
683 from U.S. anthropogenic emissions. The locations with smaller fractional contributions (e.g., 10-20
684 percent) from U.S. sources are generally located in places where ozone values are typically low such as
685 the Pacific Northwest. Figures 9c-9g display the fractional contributions from the other five in-domain
686 sectors listed in section 2. The impacts from these sectors are briefly summarized below:

687   • Biogenic emissions:
688   o Most frequent bin: 3-5 percent
689   o Highest site-specific contribution: 10-20 percent
690   o Region with greatest impacts: Great Plains states where soil NOx emissions are large
691   • Climatologically-average fire emissions:
692   o Most frequent bin: 0-1 percent
693   o Highest site-specific contribution: 3-5 percent
694   o Region with greatest impacts: California, Kansas/Oklahoma region
695   • Within-domain Canadian/Mexican manmade emissions:
696   o Most frequent bin: 0-1 percent
697   o Highest site-specific contribution: 10-20 percent
698   o Region with greatest impacts: Sites along international borders (NY, VT, CA, AZ, TX)
699   • Category 3 marine vessels outside U.S. territorial waters:
700   o Most frequent bin: 0-1 percent
701   o Highest site-specific contribution: 10-20 percent
702   o Region with greatest impacts: Coastal sites (especially southern CA)
703   • Gulf of Mexico point sources[4]:
704   o Most frequent bin: 0-1 percent
705   o Highest site-specific contribution: 1-3 percent
706   o Region with greatest impacts: Sites in southeast TX and southern LA
707
708
709
710

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[4] This sector was also included as part of U.S. anthropogenic source impacts in Figure 9b, but is broken out separately in Figure 9g.

711
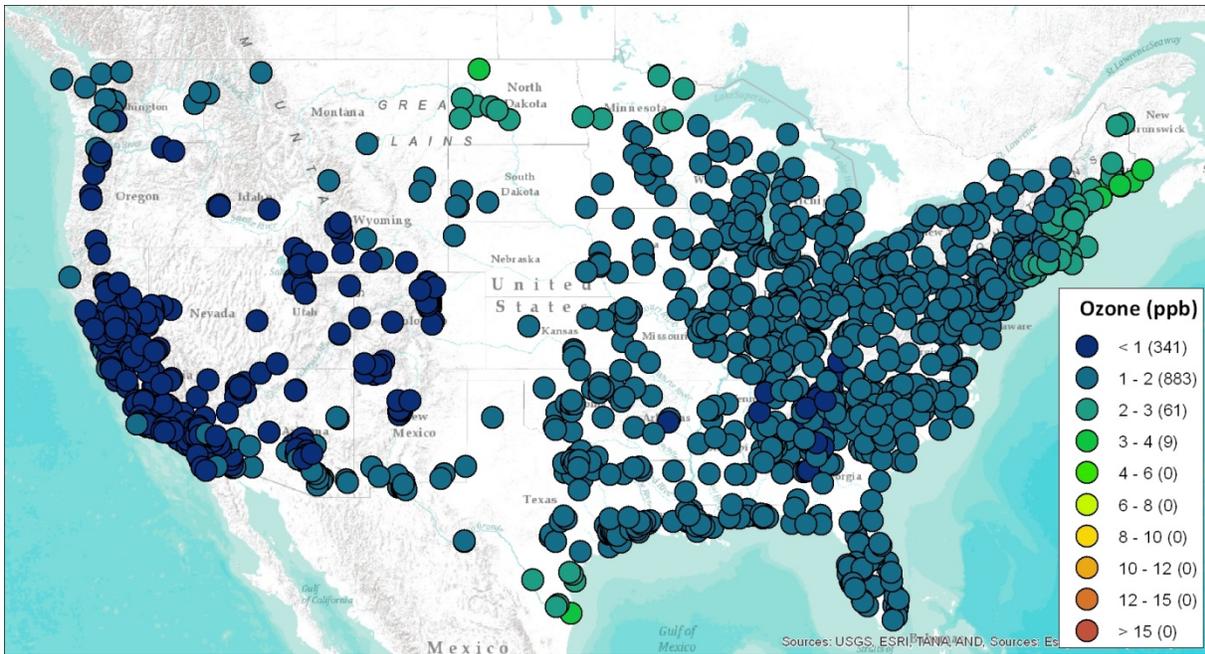
Figure 8a. Difference in April-October average MDA8 ozone (ppb) at monitoring locations across the U.S. between the USB scenario and the NAB scenario. The difference between these two runs isolates the impact of within-the-domain anthropogenic emissions from Canada and Mexico.

715
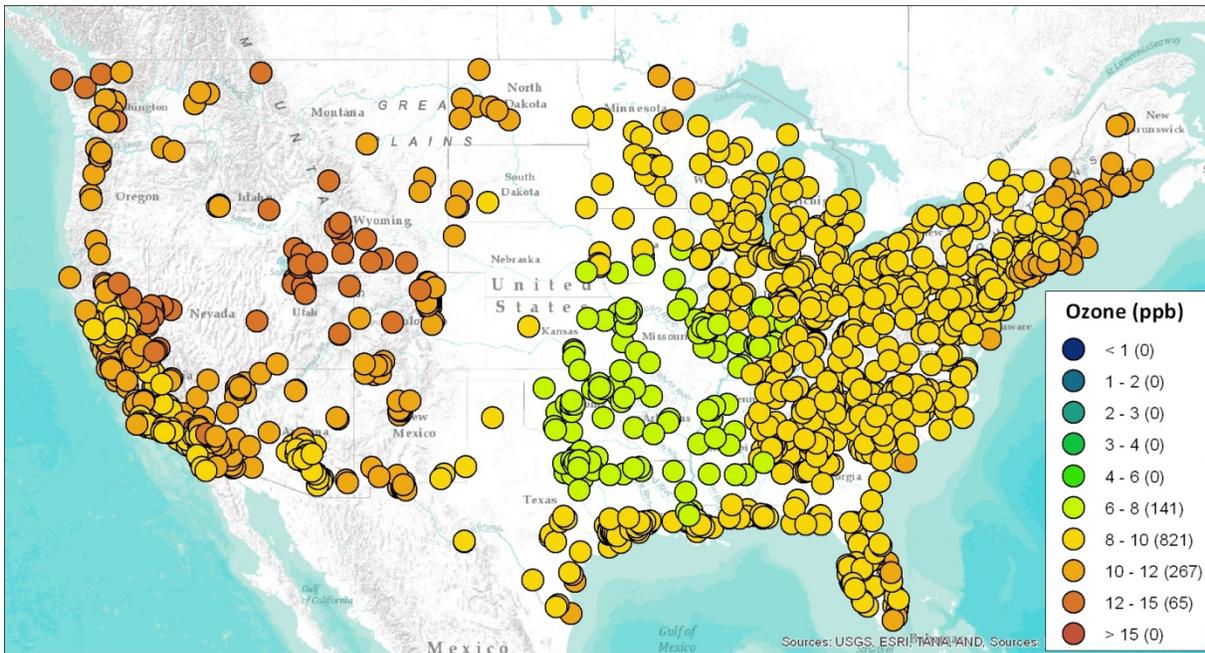
Figure 8b. Difference in April-October average MDA8 ozone (ppb) at monitoring locations across the U.S. between the NAB scenario and the NB scenario. The difference between these two runs isolates the impact of the rise in global methane emissions from the pre-industrial and anthropogenic emissions from outside North America.
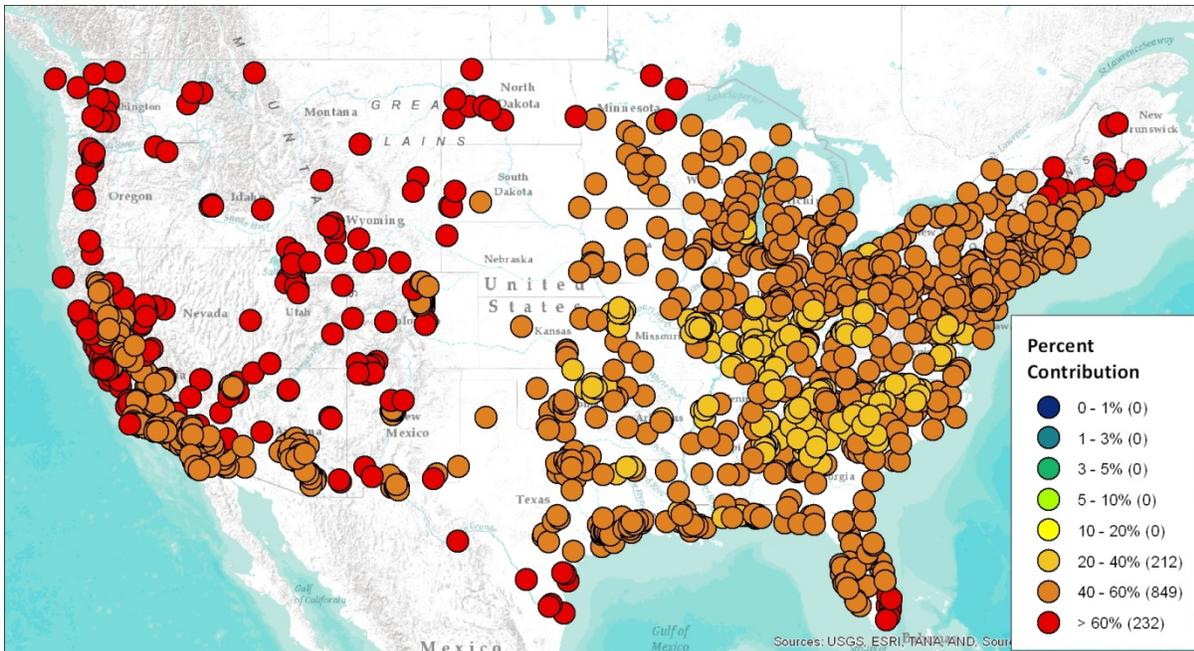
720

721 **Figure 9a. Percentage of April-October average MDA8 ozone that is apportioned to *boundary***

722 ***conditions* as estimated at monitoring locations by a 2007 CAMx simulation.**

723

724
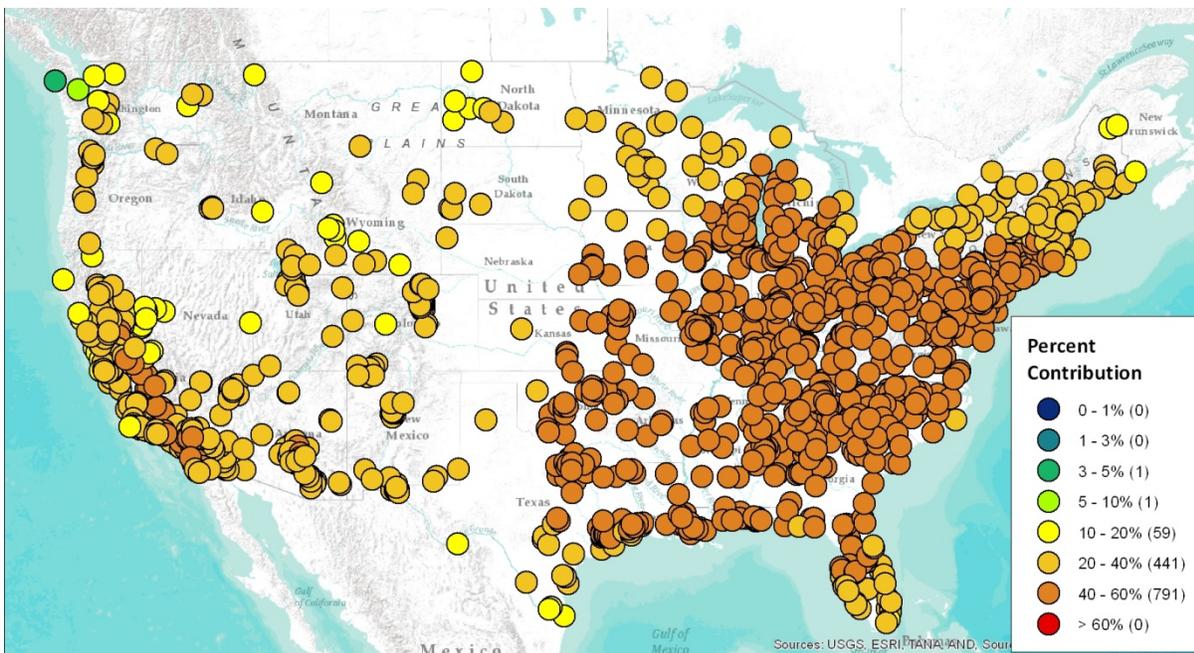
725

726 **Figure 9b. Percentage of April-October average MDA8 ozone that is apportioned to *U.S.***

727 ***anthropogenic sources* as estimated at monitoring locations by a 2007 CAMx simulation.**

728
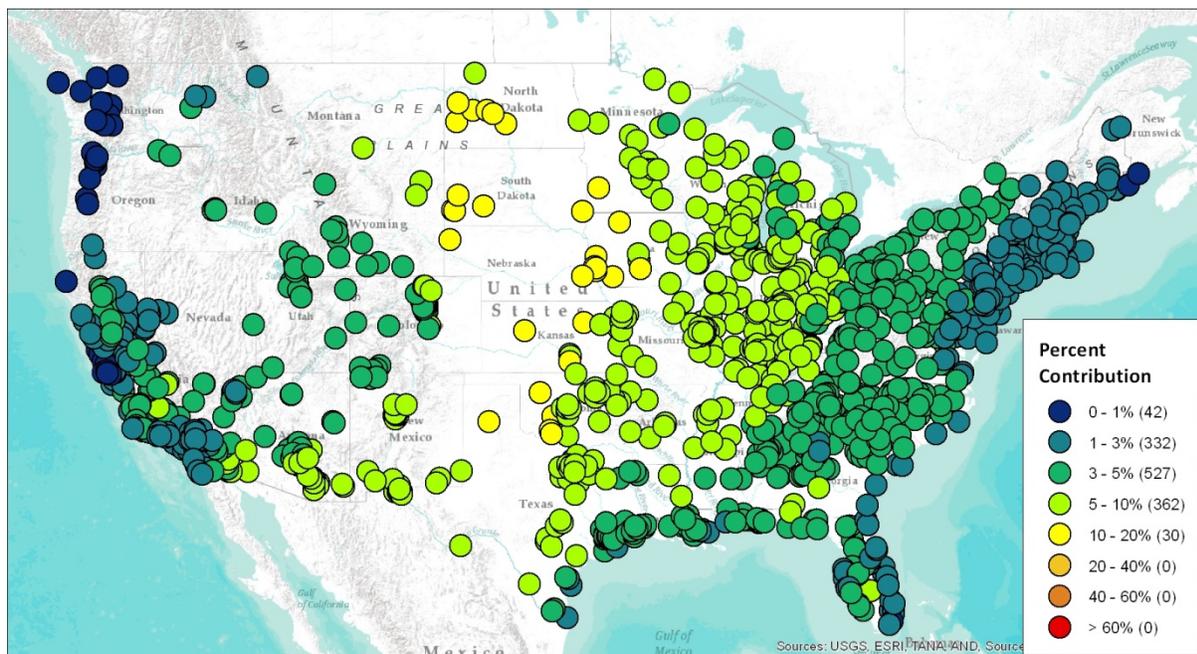
729
730   **Figure 9c. Percentage of April-October average MDA8 ozone that is apportioned to *purely biogenic***
731   ***emissions* as estimated at monitoring locations by a 2007 CAMx simulation.**

732
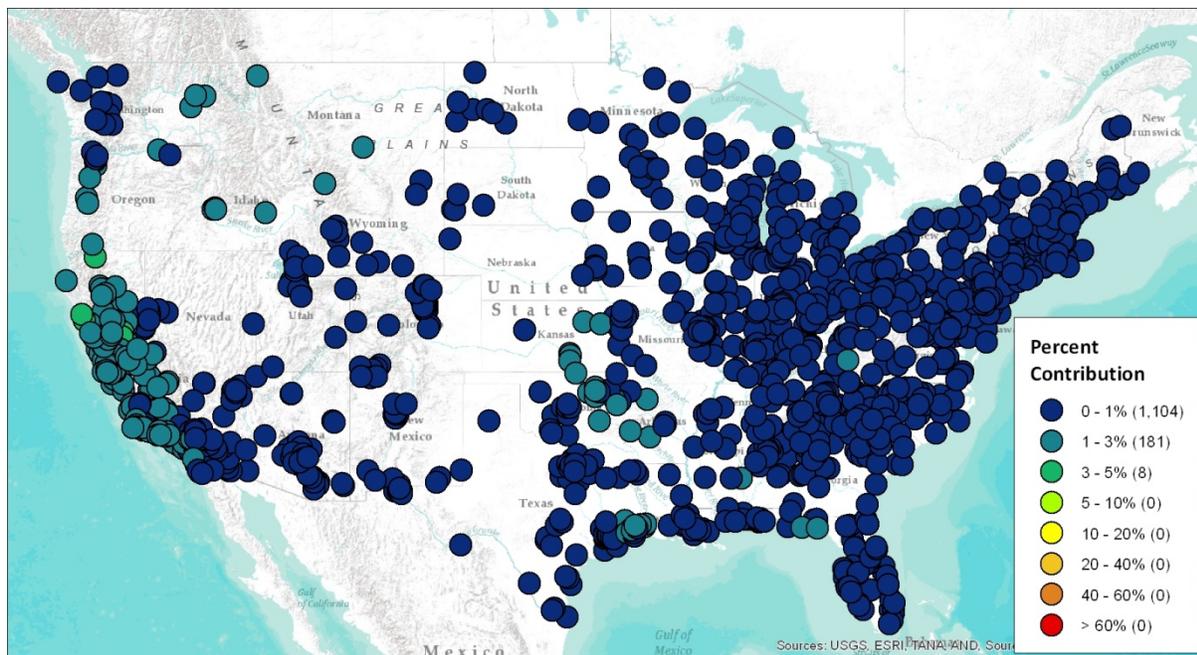
733

734   **Figure 9d. Percentage of April-October average MDA8 ozone that is apportioned to *climatological fire***
735   ***emissions* as estimated at monitoring locations by a 2007 CAMx simulation.**
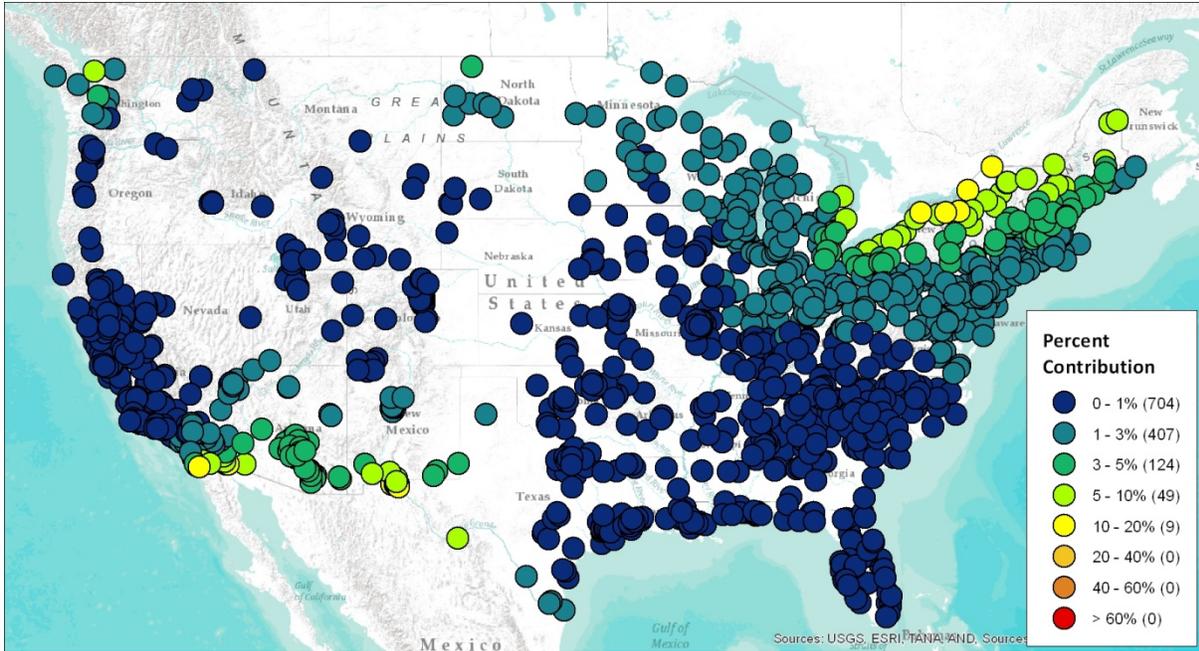
736
737   **Figure 9e. Percentage of April-October average MDA8 ozone that is apportioned to *anthropogenic***
738   ***emissions from in-domain Canadian and Mexican sources* as estimated at monitoring locations by a**
739   **2007 CAMx simulation.**

740
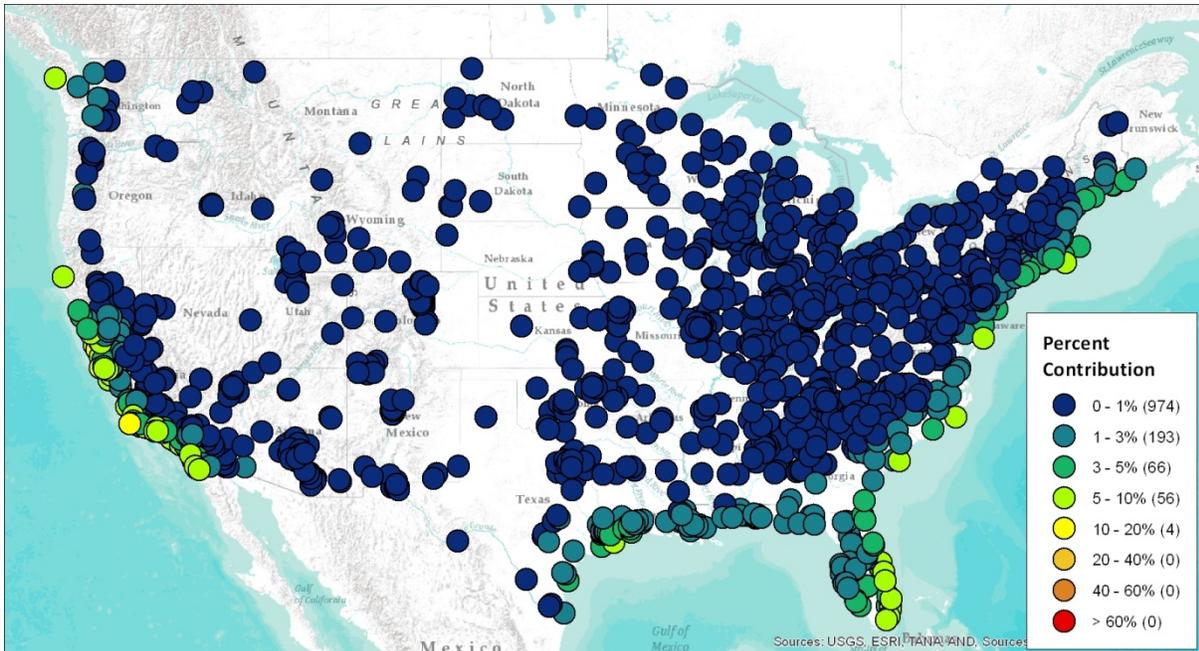
741
742   **Figure 9f. Percentage of April-October average MDA8 ozone that is apportioned to *Category 3 marine***
743   ***vessel emissions beyond U.S. territorial waters* as estimated at monitoring locations by a 2007 CAMx**
744   **simulation.**

745

746
747 **Figure 9g. Percentage of April-October average MDA8 ozone that is apportioned to *Gulf of Mexico***
748 ***point sources* as estimated at monitoring locations by a 2007 CAMx simulation.**

749

750 **6.   Estimates of the fractional background contribution to total ozone in 12 specific areas**

751   This penultimate section of the appendix presents estimates of the overall fraction of ozone that
752 is estimated to result from background sources or processes based on the updated modeling in each of
753 the 12 urban case study areas in the epidemiology study based analyses in Chapter 7 of the Risk and
754 Exposure Assessment (REA). Tables 1a-1c summarize the CAMx-estimated fractional contributions of
755 sources other than U.S. anthropogenic emissions to total ozone in each of the 12 areas. Table 1a shows
756 that the fractional contributions from sources other than anthropogenic emissions within the U.S. to
757 seasonal mean MDA8 levels can range from 43 to 66 percent across these 12 urban areas. These
758 fractions are consistent with the national ratios summarized in section 3, although the urban fractions of
759 background tend to be smaller than at rural sites. As shown in section 4, the fractional contributions
760 from background are smaller on days with high modeled ozone (i.e., days that may exceed the level of
761 the NAAQS). Table 1b provides the fractional contributions from these non-U.S. sources, only
762 considering days in which base model MDA8 ozone was greater than 60 ppb. As expected, the fractional
763 background contributions are less and range from 31 to 55 percent. Rather than taking the fractions of
764 the seasonal means (as in Table 1a), Table 1c displays the mean and median daily MDA8 background
765 fractions. These metrics may be more appropriate for application to health studies, but as can be seen
766 the fractional contribution to backgrounds calculated via this approach are very similar to the Table 1a
767 calculations. For completeness sake, although EPA expects the source apportionment results to provide
768 a more realistic estimate of fractional background values, for completeness, we also provide USB
769 fractions based on zero out modeling for the 12 cities (see Table 1d). The results are similar to the
770 source apportionment findings (compare against Table 1a), but the zero out technique provides slightly
771 higher background proportions.

772

773
All days, CAMx	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	59.3	54.4	43.0	48.9	47.3	39.1	48.5	51.1	45.4	48.7	46.4	49.8
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.3	25.9	26.2	25.7	31.3	23.3	27.0	29.1	24.5	24.2	29.7	24.3
Fractional contribution from background	0.43	0.48	0.61	0.52	0.66	0.60	0.56	0.57	0.54	0.50	0.64	0.49

774 **Table 1a. April-October average MDA8 ozone, average MDA8 ozone from sources other than U.S.**
775 **manmade emissions, and the fractional contribution of these background sources in the 12 REA urban**
776 **study areas, as estimated by a 2007 CAMx simulation.**

Only days w/ base MDA8 > 60 ppb	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	74.0	75.3	70.7	72.0	67.5	68.9	70.3	74.4	74.1	74.0	68.3	70.0
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.4	23.7	24.4	25.4	37.3	24.4	28.0	31.9	23.5	22.9	32.1	25.4
Fractional contribution from background	0.34	0.31	0.35	0.35	0.55	0.35	0.40	0.43	0.32	0.31	0.47	0.36

Table 1b. Average MDA8 ozone, average MDA8 ozone from sources other than U.S. manmade emissions, and the fractional contribution of these background sources in the 12 REA areas, as estimated by a 2007 CAMx simulation using site-days in which base MDA8 ozone exceeded 60 ppb.

	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Mean of daily MDA8 background fractions	0.46	0.53	0.68	0.58	0.69	0.64	0.59	0.61	0.61	0.56	0.67	0.52
Median of daily MDA8 background fractions	0.43	0.51	0.73	0.54	0.69	0.66	0.59	0.60	0.63	0.54	0.66	0.49

Table 1c. Fractional contribution of non-U.S. manmade emissions sources in the 12 REA urban study areas, as estimated by a 2007 CAMx simulation using means and medians of daily MDA8 fractions.

All days, CMAQ	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	58.6	55.6	45.2	51.8	57.1	43.5	49.4	54.8	47.7	50.5	51.9	52.6
Model MDA8 seasonal mean from USB emissions	30.0	29.9	28.5	31.6	42.2	31.7	33.0	33.3	29.1	29.4	34.4	32.0
Fractional contribution from background	0.51	0.54	0.63	0.61	0.74	0.73	0.67	0.61	0.61	0.58	0.66	0.61

Table 1d. April-October average MDA8 ozone, average MDA8 ozone from USB, and the fractional contribution of these background sources in the 12 REA urban study areas, as estimated by two separate 2007 CMAQ simulations.

7.   Background ozone and W126

792

793   As discussed in section 5 of the second draft policy assessment, EPA is considering the adequacy
794   of the current secondary standard to protect against welfare effects. One metric that has been
795   considered previously as a potential cumulative seasonal index is the W126 metric. The W126 index is a
796   sigmoidally weighted sum of all hourly O_3 concentrations observed during a specified daily and seasonal
797   time window, where each hourly O_3 concentration is given a weight that increases from 0 to 1 with
798   increasing concentration (Lefohn et al, 1988). The weights are defined such that values of 0.060 ppm
799   get a weight of ~0.3; 0.070 ppm values get a weight of ~0.6; and 0.085 ppm values get a weight of ~0.9.
800   The remainder of this section uses the 2007 zero out modeling to conduct a limited assessment of the
801   role of background ozone on W126 levels over the U.S.

802   The analysis of background influence on W126 is not as detailed as the analyses related to
803   seasonal mean MDA8 ozone. Instead of considering impacts at every monitoring location, EPA assessed
804   NB, NAB, and USB influences at four sample locations: Atlanta GA, Denver CO, Farmington NM, and
805   Riverside CA. Each of these four locations had relatively high observed values of W126 in 2010-2012.
806   Atlanta is an urban area in the Eastern U.S. with high primary ozone design values but relatively low
807   levels of seasonal background ozone. Riverside and Denver also have high primary ozone design values
808   but are in the Western U.S. where background ozone levels are generally higher. Farmington NM was
809   chosen as a site that has relatively lower primary ozone design values along with its relatively high W126
810   levels. The varying characteristics of each of these locations perhaps allows broader national
811   extrapolation of the 4-site results.

812   In previous EPA reviews of the O_3 NAAQS, the influence of background ozone was estimated
813   according to a counterfactual (i.e., how much ozone would exist in the absence of certain sets of
814   emissions). In the current review, EPA is supplementing the counterfactual assessment with analyses
815   that estimate the fraction of the existing ozone that is due to background sources. This has important
816   ramifications for assessing the influence of background on W126 concentrations, because of the non-
817   linear weighting function used in the metric which emphasizes high ozone hours (e.g., periods in which
818   ozone is greater than ~60 ppb). As an example, consider a sample site in the intermountain western
819   U.S. region with very high modeled estimates of U.S background (e.g., seasonal mean USB of 45 ppb
820   with some days as high as 65 ppb). Even at this high background location, the calculated annual W126
821   values in the USB scenario are quite low, on the order of 3 ppm-hrs. Most sites in the domain where
822   background levels are lower than the location cited above will have even smaller background W126
823   estimates, on the order of 1 ppm-hrs, which is consistent with values mentioned in past reviews (USEPA,
824   2007). Using the counterfactual scenarios, background ozone has a relatively small impact on W126
825   levels across the U.S.

826   However, because of the non-linear weighting function used in the W126 calculation, the sum of
827   the W126 from the USB scenario and the W126 resulting from US anthropogenic sources will not equal
828   the total W126. In most cases, the sum of those two components will be substantially less than total
829   W126. As a result, EPA believes it is more informative to estimate the fractional contribution of
830   background ozone to W126 levels. The 5-step methodology for assessing the fractional influence of

831 background ozone to annual W126 levels in the four locations is described below. The fractional
832 influence methodology essentially places higher weights on background fractions on days that are going
833 to contribute most substantially to the yearly W126 value.

834 • Step 1a: Calculate the MDA8 ozone values from the base and the three zero out
835   modeling scenarios at each grid cell containing a site in an area.
836 • Step 1b: Calculate the W126 daily index for the base model scenario.
837 • Step 2: For each site, find the three months with highest summed W126 daily indices.
838 • Step 3: Normalize the daily MDA8 values in the base, NB, NAB, and USB scenarios by the
839   corresponding W126 daily index from the base scenario.
840 • Step 4: Calculate the average W126-weighted MDA8 values over the three month
841   period for each of the four scenarios (base, NB, NAB, USB).
842 • Step 5: Calculate the NB/Base, NAB/Base, and USB/Base ratios based on step 4 outputs.
843   These values represent an estimate of the fractional influence of background ozone on
844   modeled W126 levels.

845   Figure 7a shows the estimated fractional influence of the three background definitions on W126
846 levels in Atlanta, Denver, Farmington, and Riverside. Based on this limited assessment, natural
847 background sources are estimated to contribute 29-50% of the total modeled W126 with the highest
848 relative influence in the intermountain western U.S. (e.g., Farmington NM) and the lowest relative
849 influence in the eastern U.S. (e.g., Atlanta). U.S. background is estimated to contribute 37-65% of the
850 total modeled W126. Figure 7b compares the relative influence of background on W126 versus seasonal
851 mean MDA8 ozone. The proportional impacts of background are slightly less for the W126 metric than
852 for seasonal mean MDA8 (discussed in section 2.4.2), because of the weighting function that places
853 more emphasis on higher ozone days when background fractions are generally lower.

854   There are several caveats associated with this analysis. First, only the zero out modeling was
855 used to assess the fractional influence of background sources on W126. The source apportionment
856 approach estimated slightly smaller relative contributions for seasonal mean MDA8 levels, so from that
857 perspective the zero out estimates could represent the high end of background influence on W126.
858 Additionally, the methodology used for this analysis relies on daily MDA8 values as a surrogate (the data
859 were readily available) for the 8a-8p time period relevant to the W126 metric. The key conclusion from
860 this cursory analysis is that background ozone may comprise a non-negligible portion of current W126
861 levels across the U.S. This fractional influence is greatest in the intermountain western U.S. and are
862 slightly smaller than the seasonal mean MDA8 metric. In the counterfactual cases, when non
863 background sources are completely removed, the remaining W126 levels are low (< 3 ppm-hrs).

864
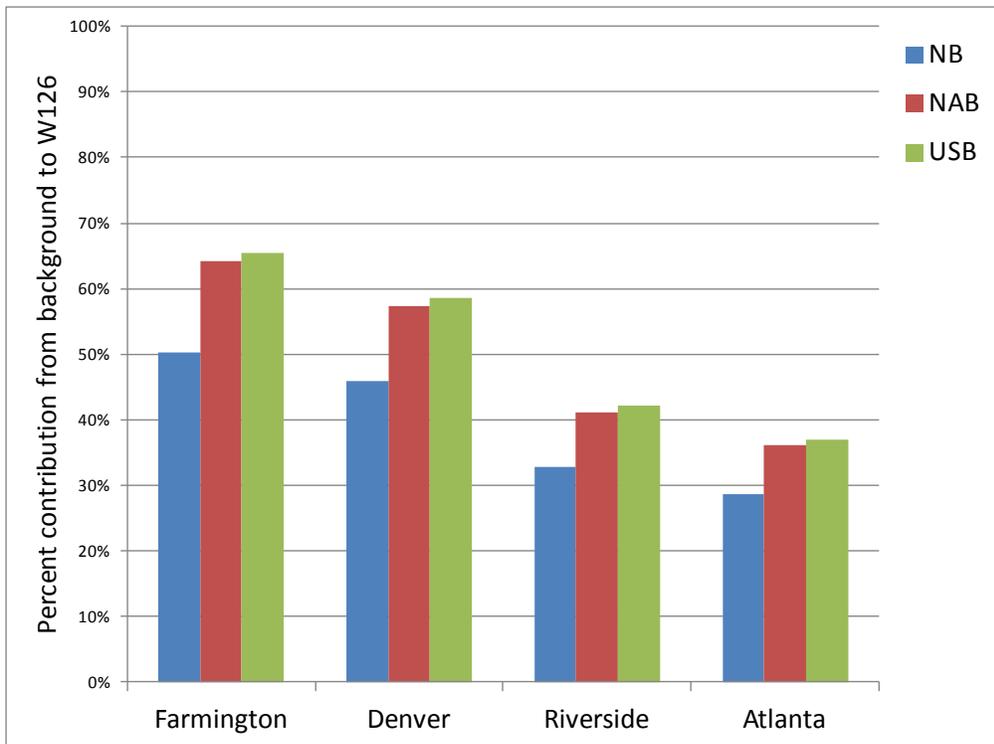
Figure 7a. Fractional contribution of background sources to W126 levels in four sample locations.
Model estimates based on 2007 CMAQ zero out modeling.

867
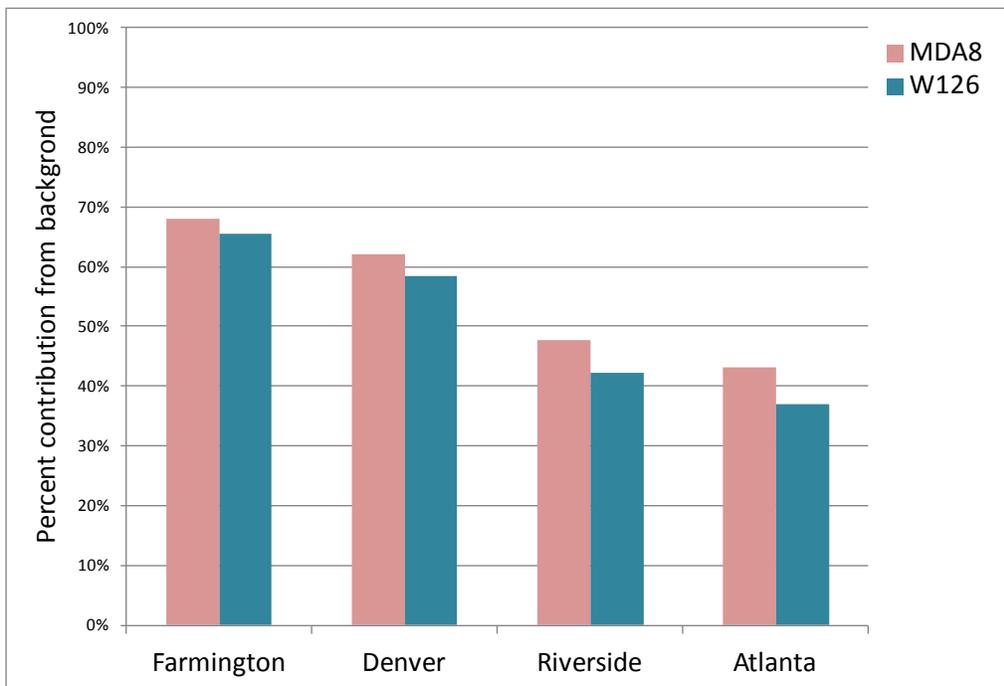
Figure 7b. Fractional contribution of U.S. background to seasonal mean MDA8 ozone and W126 levels
in four sample locations. Model estimates based on 2007 CMAQ zero out modeling.

870 **8.   Summary**

871   The precise definition of background ozone can vary depending upon context, but it generally
872 refers to ozone that is formed by sources or processes that cannot be influenced by local control
873 measures. Background ozone can originate from natural sources of ozone and ozone precursors, as well
874 as from upwind manmade emissions of ozone precursors. In order to help further characterize
875 background ozone levels over the U.S., EPA has completed additional air quality modeling analyses
876 subsequent to the 1st-draft policy assessment. As shown above, the results are largely consistent with
877 previous determinations about the magnitude of background ozone contributions across the U.S.

878   For a variety of reasons, it is challenging to present a comprehensive summary of all the
879 components and implications of background ozone. In many forums the term "background" is used
880 generically and the lack of specificity can lead to confusion as to what sources are being considered.
881 Additionally, it is well established that the impacts of background sources can vary greatly over space
882 and time which makes it difficult to present a simple summary of background ozone levels. Further,
883 background ozone can be generated by a variety of processes, each of which can lead to differential
884 patterns in space and time, and which often have different regulatory ramifications. Finally, background
885 ozone is difficult to measure and thus, typically requires air quality modeling which has inherent
886 uncertainties and potential errors and biases. Even with all of these complexities in mind, EPA believes
887 the following concise and step-wise summary of background ozone is appropriate as based on previous
888 modeling exercises and the more recent EPA analyses summarized herein.

889   •   The most fundamental definition of background is "natural background" (NB). NB ozone is that
890   which is produced by processes other than manmade emissions. Examples of sources of natural
891   background include: stratospheric ozone intrusions, wildfire emissions, and biogenic emissions
892   from vegetation and soils. To date, NB ozone has been estimated to be that ozone that would
893   exist in the absence of anthropogenic ozone precursor emissions worldwide. Modeling analyses
894   have shown that NB levels can vary in time and space. As shown in Section 3, April-October
895   average NB levels range from approximately 15-35 ppb with the highest values in the spring and
896   at higher-elevation sites.
897
898   •   More expansive definitions of background include North American background (NAB) and U.S.
899   background (USB). These definitions represent the ozone that originates from sources and
900   processes other than North American or U.S. anthropogenic sources. Sources of NAB and USB
901   include all the same sources of natural background, plus manmade ozone precursors emitted
902   outside the North America or the U.S. Modeling analyses have shown that NAB and USB
903   background levels can vary in time and space. As discussed in Section 3, seasonal mean NAB
904   and USB background levels range from approximately 25-45 ppb with the highest values in the
905   spring and at higher-elevation sites. USB levels are slightly higher than NAB, usually by less than
906   2 ppb.
907

908 • Estimates of seasonal mean background ozone levels are valuable in terms of a first-order
909 characterization, however because levels can vary significantly from day-to-day, it is also
910 instructive to consider the distribution of daily model estimates of background ozone over a
911 season. Typically, model background is slightly higher in the April-June period than in the later
912 portion of the ozone season (July-October) (EPA, 2012). More importantly, the modeling shows
913 that the days with highest ozone levels, on average, have similar background levels to days with
914 lower values. As a result, the proportion of total ozone that has background origins is smaller
915 on high ozone days (e.g., days > 70 ppb) than the more common lower ozone days that drive
916 seasonal means. Section 4 provides information about the distribution of background ozone
917 fractions. Based on the source apportionment modeling, it is shown that U.S. anthropogenic
918 emissions typically comprise the majority of the total ozone on site-days with base modeled
919 ozone MDA8 values greater than 60 ppb.
920
921 • While it is important to recognize that most high ozone days (i.e., potential exceedance days)
922 are estimated to be driven predominantly by non-background emissions, the recent EPA
923 modeling also shows times and locations in which background contributions are estimated to
924 approach 60-80 ppb. As described in Sections 4 and 6 of this document, these occurrences are
925 relatively infrequent. While the modeling was not expressly developed to capture these types
926 of events, ambient observations have also shown relatively rare events where background
927 ozone sources (wildfires, stratospheric intrusions) have overwhelmingly contributed to an
928 ozone exceedance. From a policy perspective, these background events must be viewed in the
929 context of their relative infrequency and the existing mechanisms within the Clean Air Act (e.g.,
930 exceptional event policy, 179B international determinations) that help ensure States are not
931 required to control for events that are inherently outside their ability to influence. While
932 background ozone levels can approach and periodically exceed the NAAQS at some locations,
933 these conditions are not a constraining factor in the selection of a NAAQS. The Clean Air Act
934 requires the NAAQS to be set at a level requisite to protect public health and welfare. Case law
935 makes it clear that attainability and technical feasibility are not relevant considerations. In
936 previous reviews, EPA assessed the proximity of potential levels to peak background levels as a
937 *secondary consideration* between levels where health and welfare was protected.
938
939 • Section 5 shows that the contributions to background are multi-dimensional. Daily peak 8-hour
940 ozone values over the U.S. are a function of local and regional anthropogenic emissions,
941 anthropogenic emissions from outside the U.S. (including shipping emissions), natural and
942 anthropogenic methane emissions, wildfire emissions, and purely natural sources. While local
943 and regional controls are still considered to be the most effective at reducing local ozone levels,
944 any measures to reduce the international contributions or methane-induced background will
945 also be valuable.
946
947 • In previous ozone NAAQS reviews, EPA estimated risk from exposure only to ozone
948 concentrations above background. In the first drafts of the REA and PA for the current ozone

949   review, EPA estimated risk from exposure to total measured ozone concentrations, which
950   include those concentrations from background sources. EPA will continue to provide estimates
951   of risk from exposure to total ozone, consistent with CASAC advice, in the second draft policy
952   assessment. The recent EPA modeling was completed to assist in determining, in a limited
953   sense, the risk attributable to background ozone. The fractional values of background
954   contributions in the 12 REA study areas (43-66 percent) could be used as first order
955   approximations of the risk due to ozone background.
956

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APPENDIX 2B

MONITORING DATA ANALYSIS OF RELATIONSHIPS
BETWEEN CURRENT STANDARD AND W126 METRIC

4   Presented here are monitoring data analyses evaluating relationship between ozone (O_3)

5 concentrations in the averaging time and form of the current secondary standard (3-year average

6 of the annual 4th highest daily maximum 8-hour concentrations, in parts per billion), and a three-

7 year W126 metric (3-year average of the annual maximum 3-month sum of weighted daytime

8 concentrations, in parts per million-hours). We also consider the responsiveness of these two

9 metrics to historical changes in air quality related to ozone precursor emissions.

10   For this analysis, we chose to examine monitoring data from a base period (2001-2003)

11 as well as a recent period (2009-2011). The base period was chosen to represent air quality

12 conditions before the implementation of the 1997 national ambient air quality standard

13 (NAAQS) for O_3 (0.08 ppm). In 2004, EPA designated 113 areas as nonattainment for the 1997

14 standard, which required many areas to begin precursor emissions control programs for the first

15 time. At about the same time, EPA began implementation of the NOx Budget Trading Program

16 under the NOx State Implementation Plan, also known as the "NOx SIP Call[1]," which required

17 summertime reductions in NOx emissions from power plants and other large sources throughout

18 the Eastern U.S. These programs were successful in reducing peak O3 concentrations, especially

19 in the Eastern U.S., and as a result only 8 of the original 113 nonattainment areas were still

20 violating the 1997 O3 NAAQS during the 2009-2011 period.

21   Hourly O3 concentration data were retrieved from EPA's Air Quality System (AQS)

22 database[2] for both periods, and used to calculate design values for the current standard as well as

23 3-year average W126 values for both periods. The procedures for calculating design values for

24 the current standard from hourly O_3 concentration data are described in 40 CFR Part 50,

25 Appendix P, and the procedures for calculating the 3-year average W126 values are described in

26 section 4.3.1. of the 2nd draft Welfare Risk and Exposure Assessment (WREA). There were 838

27 monitoring sites with sufficient data to calculate these values for both periods. In order to

28 identify regional patterns in the relationships, these sites were grouped into the nine NOAA

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[1] http://www.epa.gov/airmarkets/progsregs/nox/sip.html

[2] EPA's Air Quality System (AQS) database is a national repository for many types of air quality and related monitoring data. AQS contains monitoring data for the six criteria pollutants dating back to the 1970's, as well as more recent additions such as PM2.5 speciation, air toxics, and meteorology data. At present, AQS receives hourly O3 monitoring data collected from nearly 1,400 monitors operated by over 100 state, local, and tribal air quality monitoring agencies.

29   climate regions (Karl and Koss, 1984) used in the WREA. Figure 2B-1 presents a map of these

30   regions, which are color-coded to match the scatter plots in the subsequent figures.

31   Figures 2B-2a, 2B-2b, 2B-3a and 2B-3b show scatter plots of the design values for the

32   current standard (x-axis) versus 3-year average W126 values (y-axis) for the base period and

33   recent period, respectively. Most monitors in the U.S. both exceeded the current standard of 75

34   ppb and a three-year average W126 value of 15 ppm-hrs during the base period. During the

35   recent period, both the design values and 3-year average W126 values were much lower, and

36   there also appears to be less scatter between the two metrics. In both periods, the highest design

37   values and W126 values occurred in the West region which includes California. Finally, it is

38   worth noting that monitors in the Southwest and West regions tend to have higher W126 values

39   relative to their design values than in other regions.

40   Figure 2B-4 shows a scatter plot of the design values for the current standard for the base

41   period (x-axis) versus for the recent period (y-axis), while Figure 2B-5 shows this same

42   relationship based on the 3-year average W126 values. The relationship between the two periods

43   appears to be fairly linear for both metrics, indicating that larger decreases in these metrics

44   tended to occur at monitors with higher base values. Figures 2B-6 and 2B-7 show design values

45   for the current standard and 3-year average W126 values, respectively, compared to the unit

46   changes in those values between the base period and recent period. Figures 2B-6 and 2B-7 show

47   the difference between each point and the one-to-one lines in Figures 2B-4 and 2B-5,

48   respectively. In particular, these figures highlight that there were some monitors where design

49   values for the current standard and/or W126 values increased. However, those monitors also

50   tended to have lower base values, and were mostly located outside of areas subject to emissions

51   controls under the 1997 standard.

52   Finally, Figure 2B-8 compares the unit change in design values (in ppb; x-axis) to the

53   unit change in 3-year average W126 values (in ppm-hrs; y-axis). This figure shows that in most

54   locations, the current standard metric and the W126 metric exhibit similar responses to changes

55   in precursor emissions. In particular, the NOx SIP Call, which was implemented in the states

56   east of the Mississippi River, was effective at reducing both design values and W126 values at

57   nearly all monitors in the Eastern U.S. The relationship was much more variable in the

58   remaining regions, where emissions control programs were mostly local and limited to areas

59   which were violating the NAAQS.

60   Based on this analysis of ambient monitoring data, we can make the following general

61   conclusions about the relationship between the design value metric for the current O_3 standard

62   and the 3-year average W126 metric:

63   1. There is a fairly strong, positive degree of correlation between the two metrics.

64   2. Monitors in the West and Southwest regions tend to have higher W126 values relative to
65   their design values than in other regions.

66   3. Reducing precursor emissions, especially NOx, is an effective strategy for lowering both
67   design values and W126 values. In particular, regional control programs such as the NOx
68   SIP call are effective at reducing both metrics over a broad area.
69

70   In addition, Figure 2B-9 examines the number of counties with 8-hour design values

71 meeting the current standard and 3-year average W126 index values greater than 15 ppm-hrs.

72 Most of these counties were located in the Southwest region of the country. There were no

73 counties in any of the studied 3-year periods that had design values less than or equal to 65 ppb

74 and 3-year average W126 index values greater than 15 ppm-hrs.

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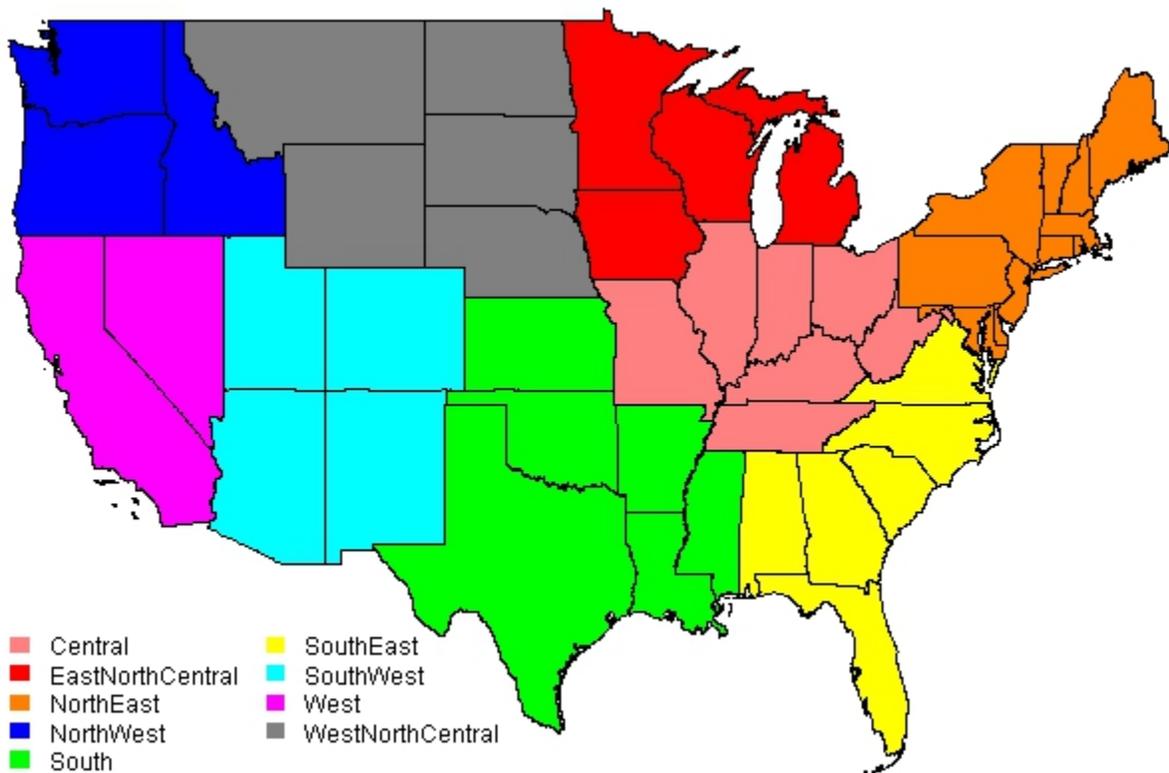
76

77 **Figure 2B-1. Map of the 9 NOAA climate regions (Karl and Koss, 1984), color coded to**
78 **match the subsequent scatter plots.**

79
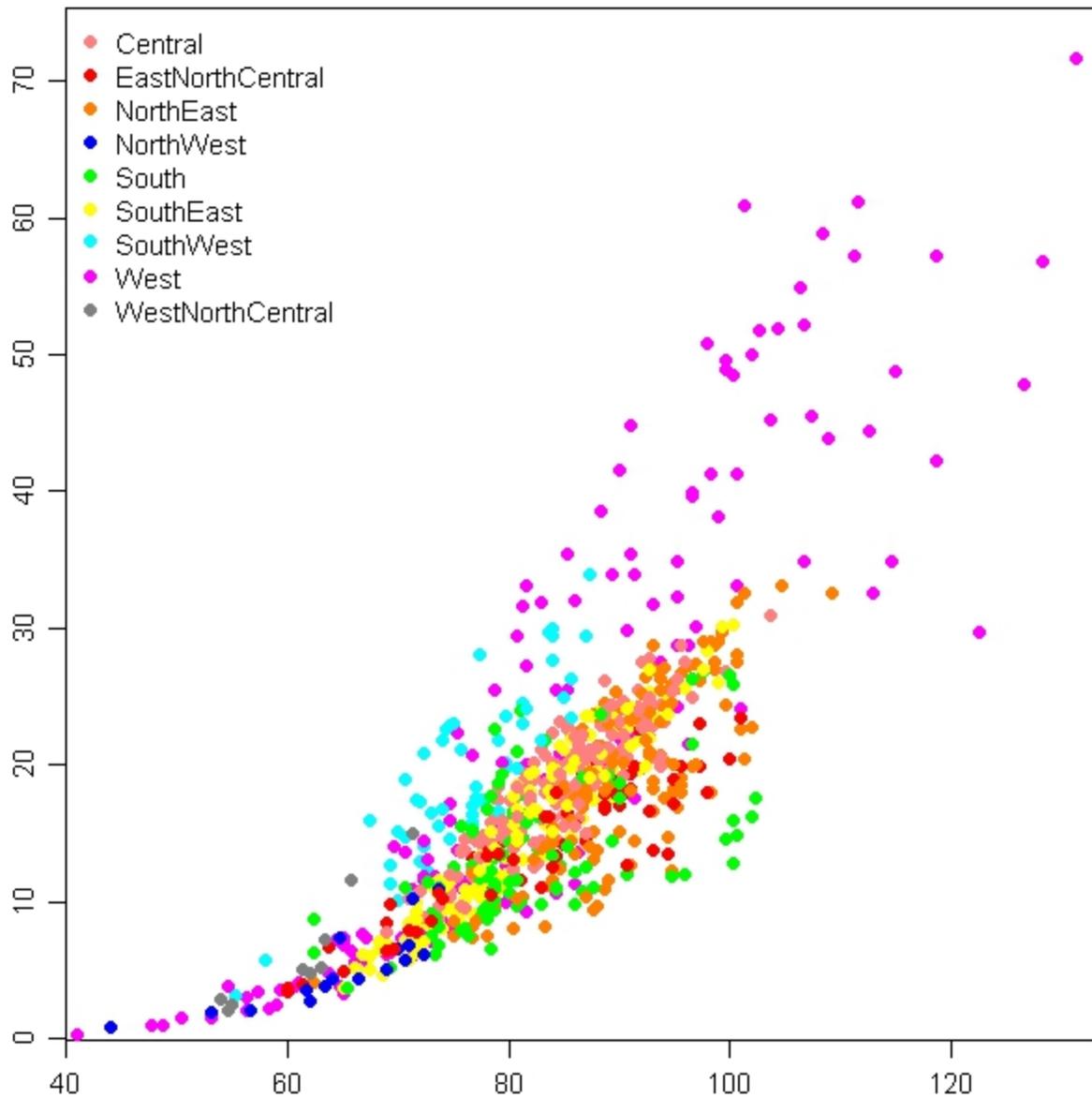
Figure 2B-2a. Design values for the current O$_3$ standard in ppb (x-axis) versus 3-year average W126 values in ppm-hrs (y-axis) based on ambient monitoring data for 2001-2003.

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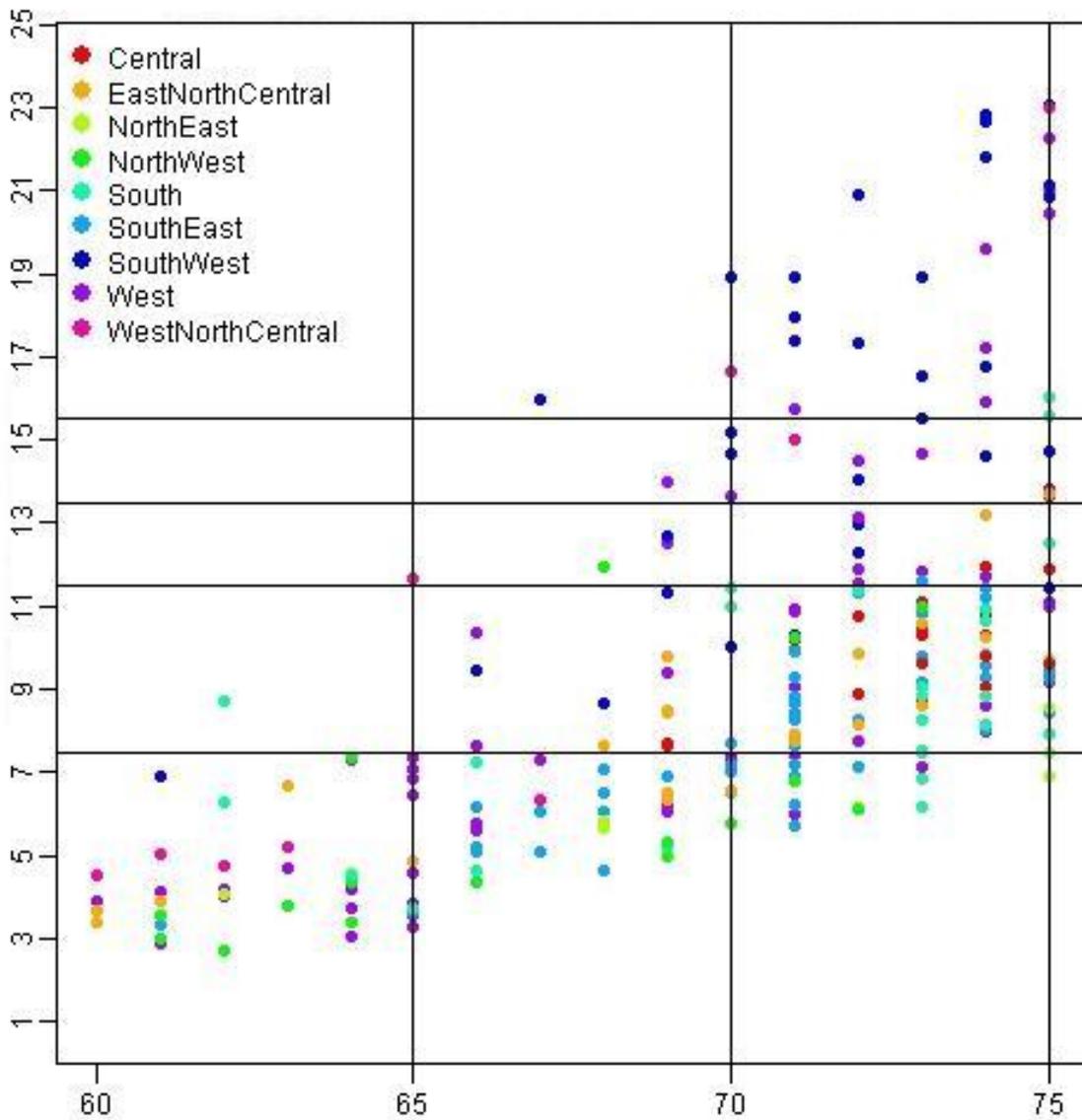
84
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86   **Figure 2B-2b. Design values for the current O$_3$ standard in ppb (x-axis) versus 3-year**
87   **average W126 values in ppm-hrs (y-axis) based on ambient monitoring data**
88   **for 2001-2003 with a focus on monitors with 2001-2003 design values below 75**
89   **ppb.**
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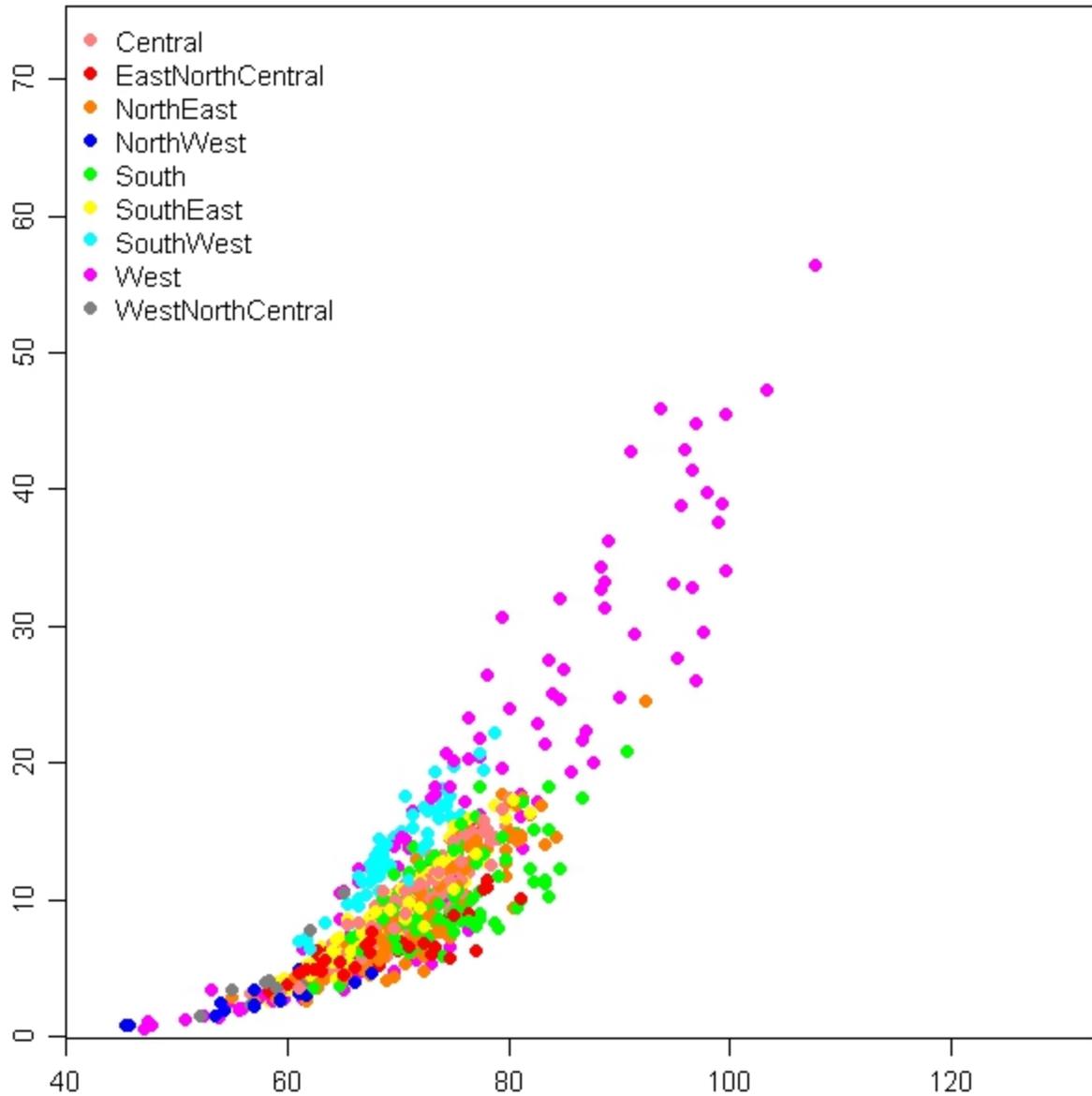
Figure 2B-3a. Design values for the current O$_3$ standard in ppb (x-axis) versus 3-year average W126 values in ppm-hrs (y-axis) based on ambient monitoring data for 2009-2011.
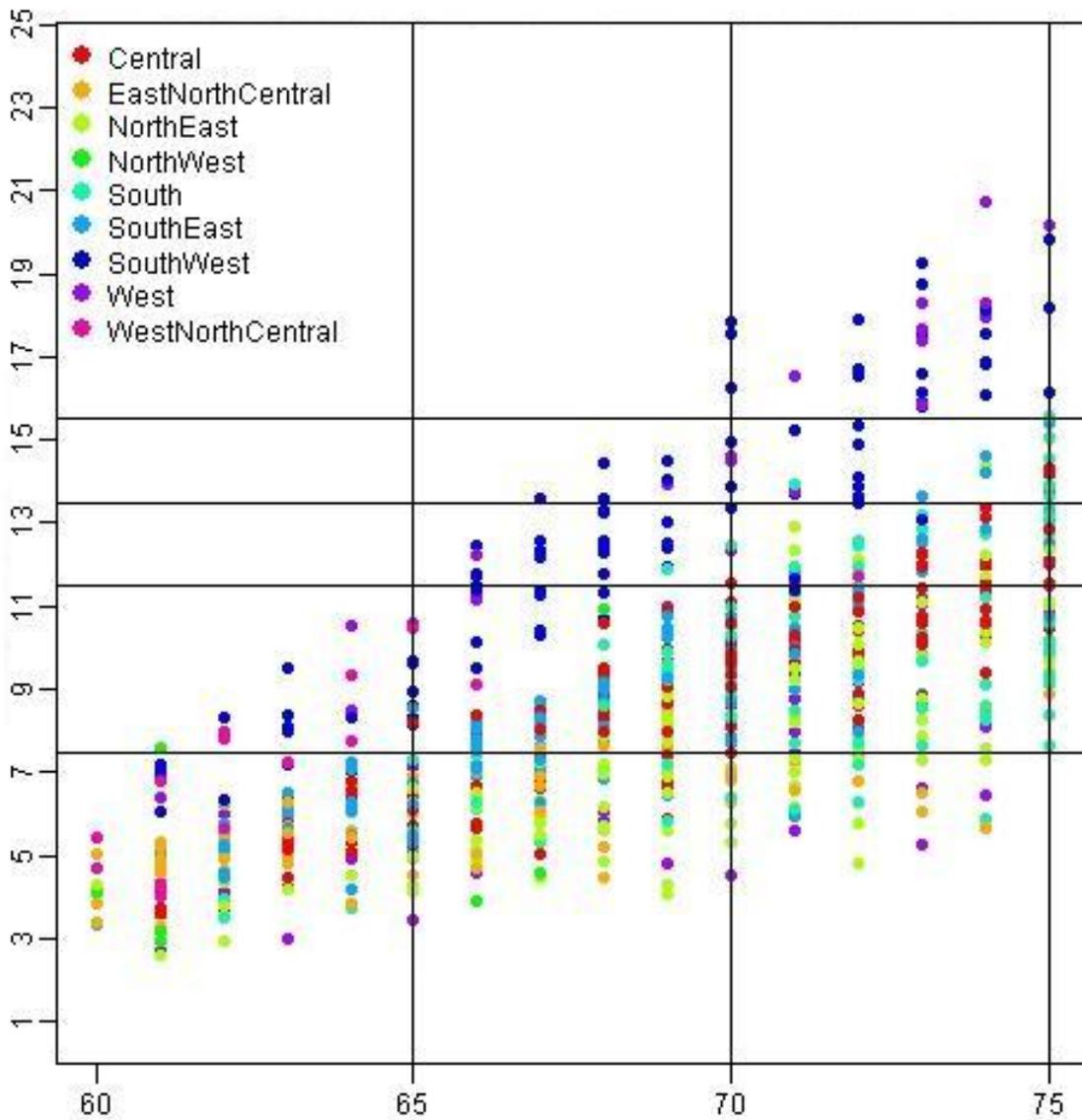
Figure 2B-3b. Design values for the current O$_3$ standard in ppb (x-axis) versus 3-year average W126 values in ppm-hrs (y-axis) based on ambient monitoring data for 2009-2011 with a focus on monitors with 2009-2011 design values below 75 ppb.

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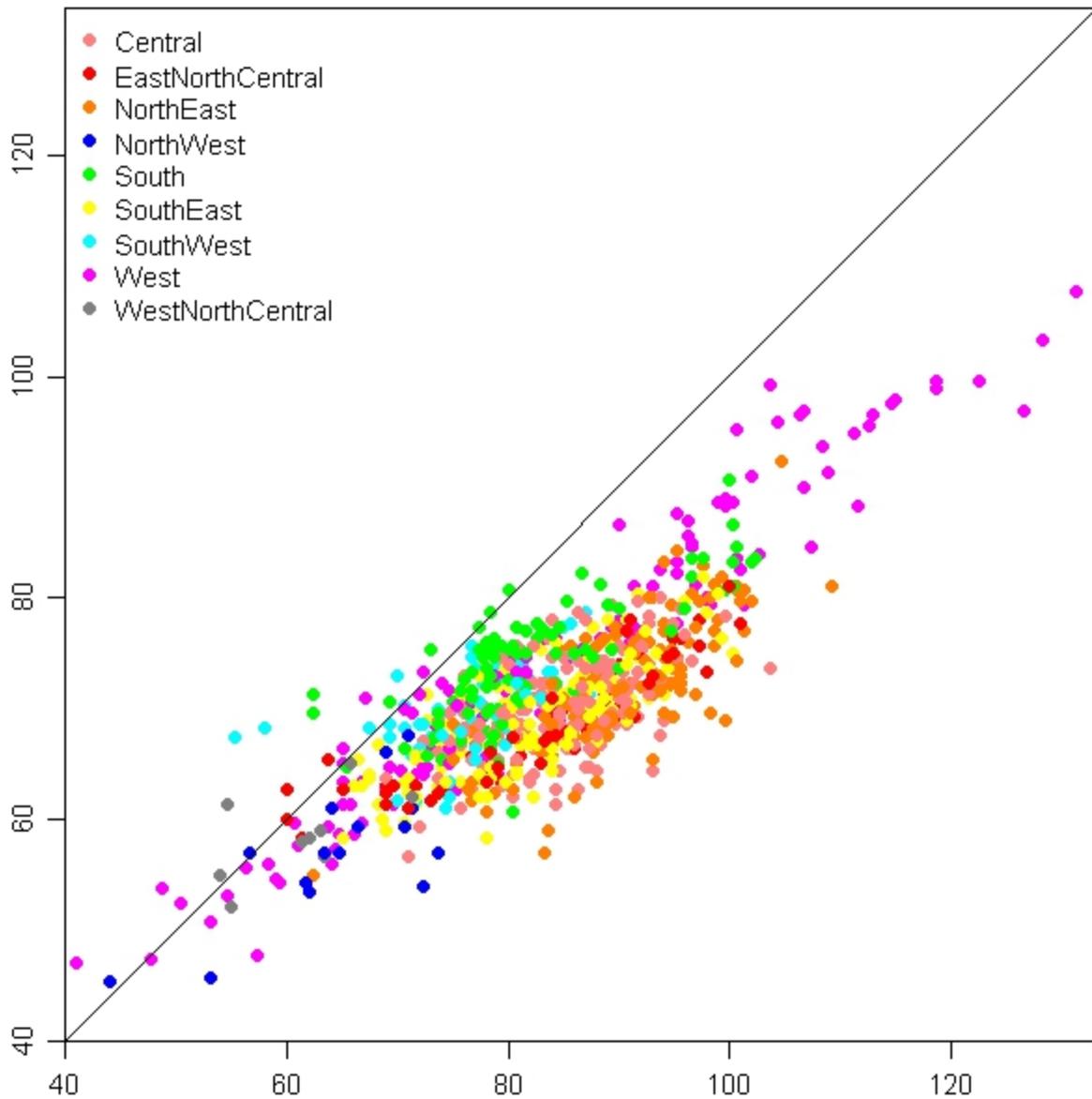
Figure 2B-4. Design values for the current O$_3$ standard in ppb based on ambient monitoring data for 2001-2003 (x-axis) versus 2009-2011 (y-axis).

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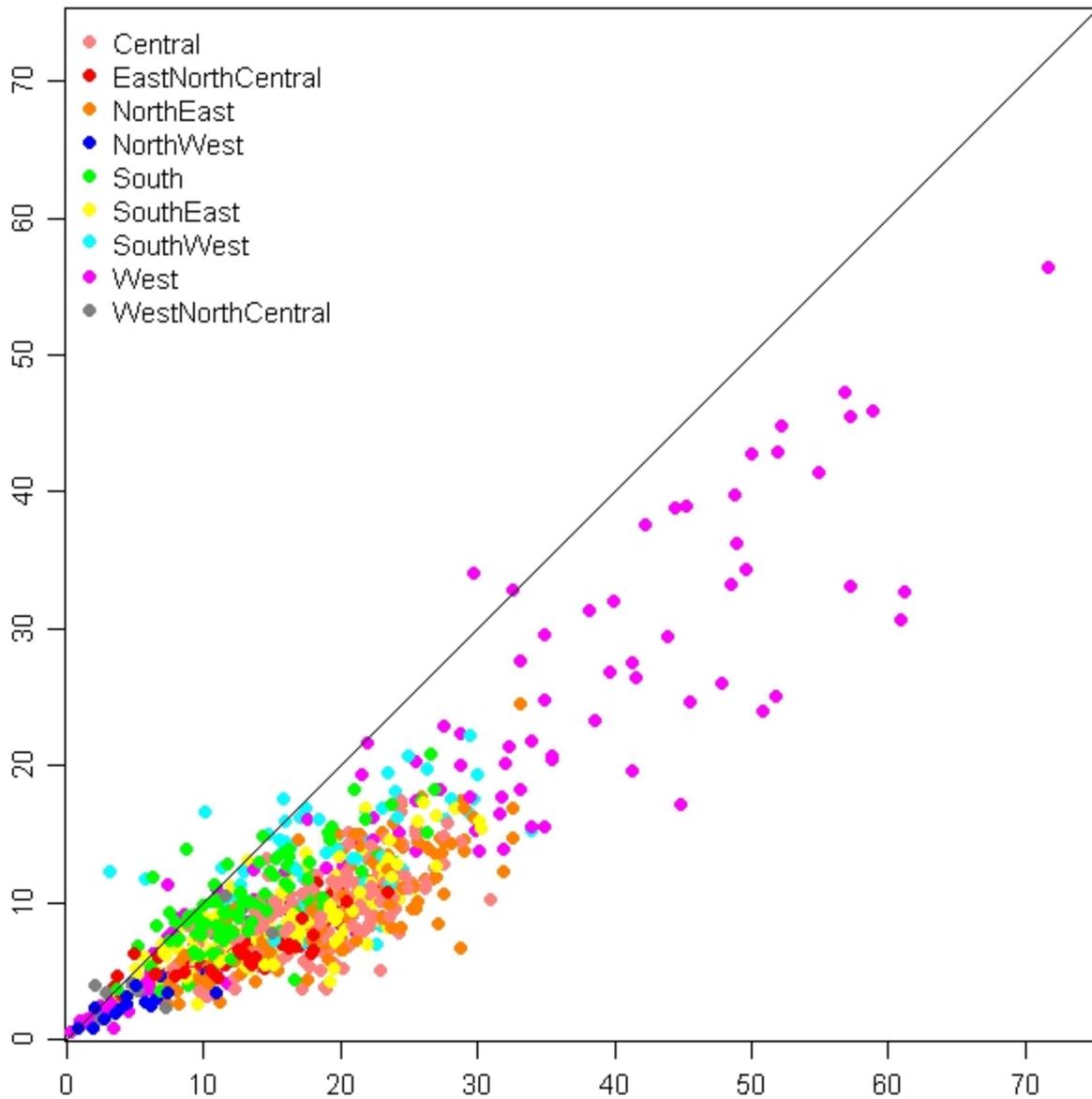
Figure 2B-5. Three-year average W126 values in ppm-hrs based on ambient monitoring data for 2001-2003 (x-axis) versus 2009-2011 (y-axis).

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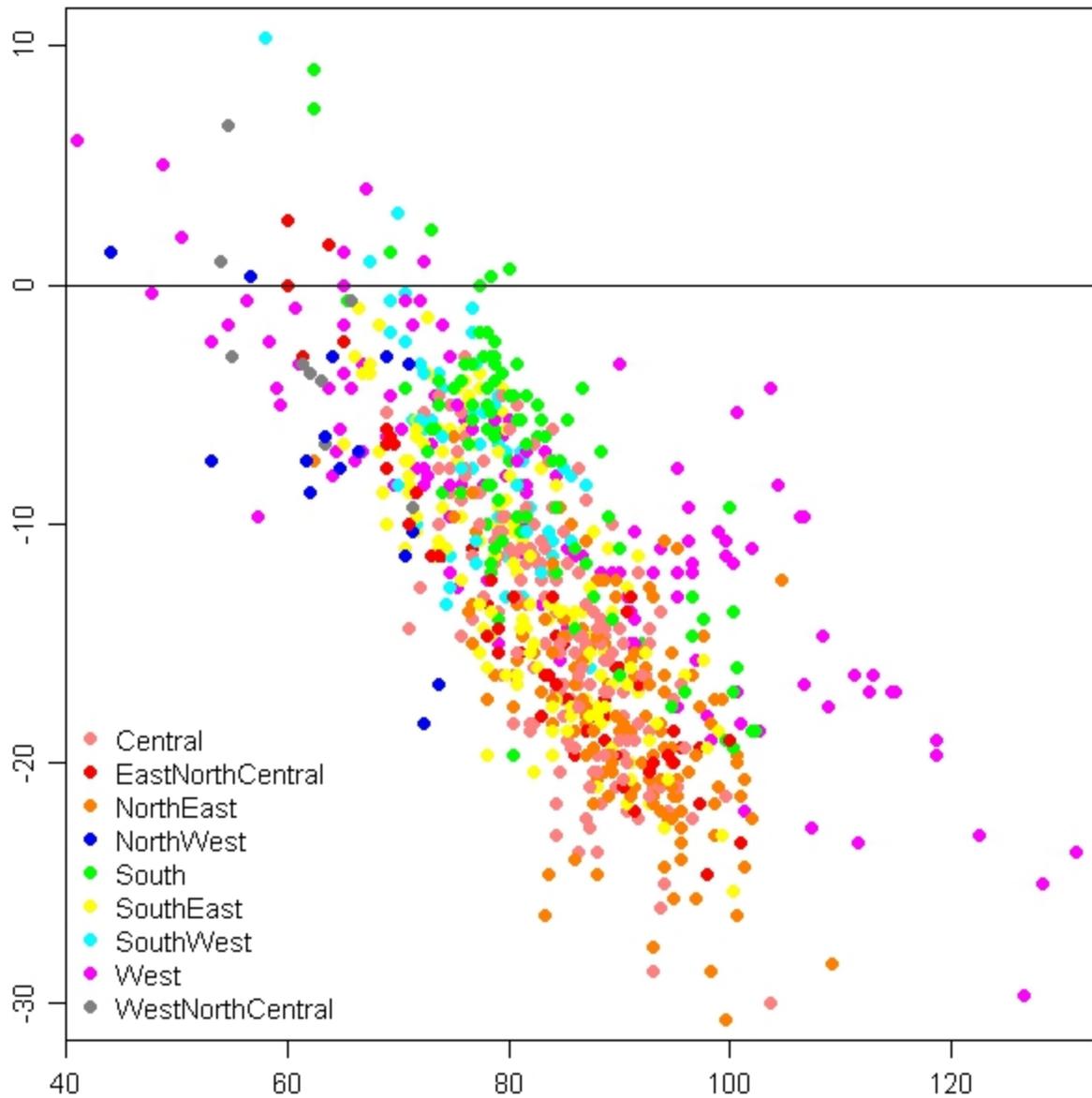
Figure 2B-6. Design values for the current O$_3$ standard in ppb based on ambient monitoring data for 2001-2003 (x-axis) versus unit (ppb) change in design values from 2001-2003 to 2009-2011 (y-axis).

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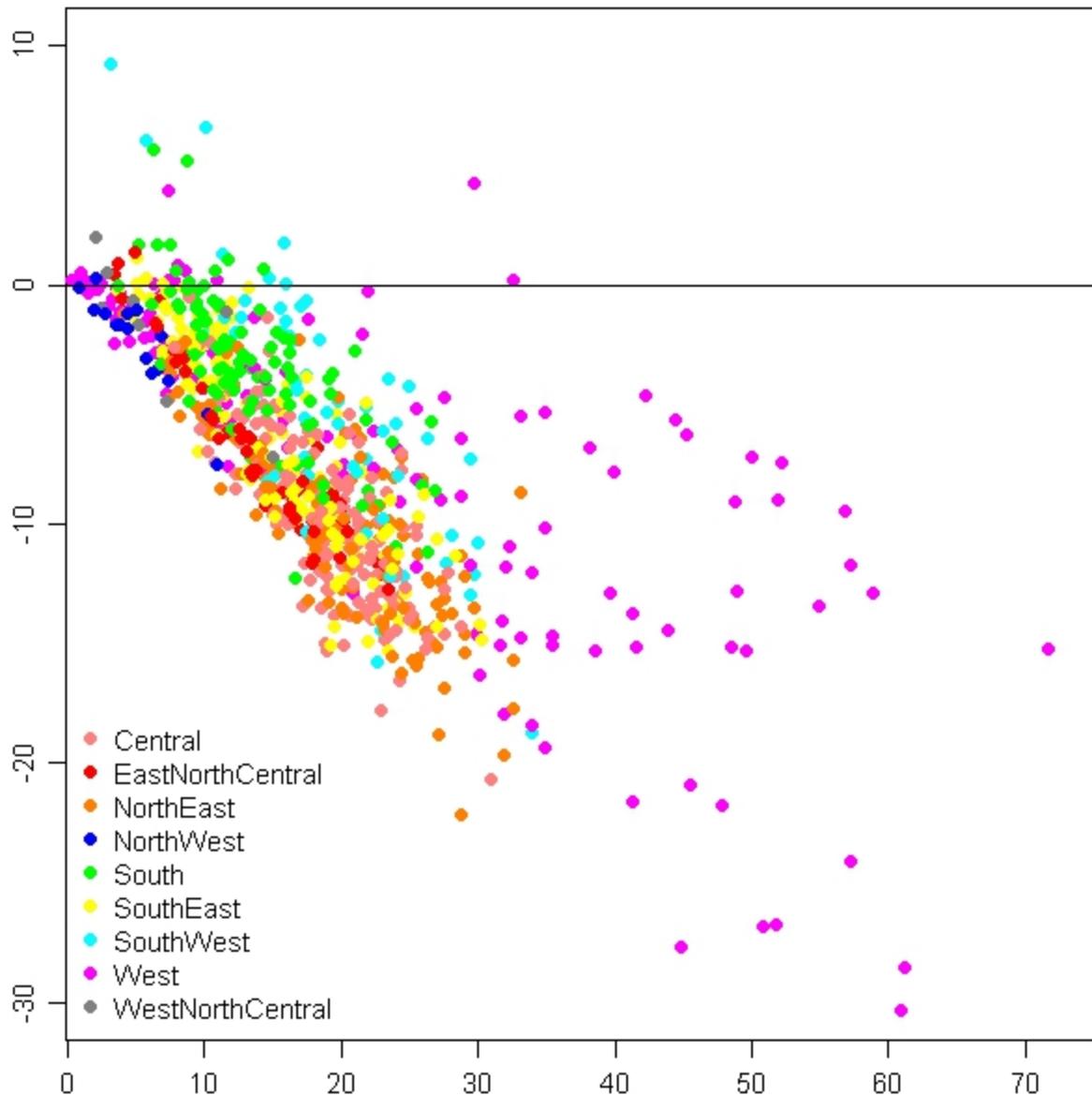
Figure 2B-7. Three-year average W126 values in ppm-hrs based on ambient monitoring data for 2001-2003 (x-axis) versus unit (ppm-hr) change in 3-year average W126 values from 2001-2003 to 2009-2011 (y-axis).

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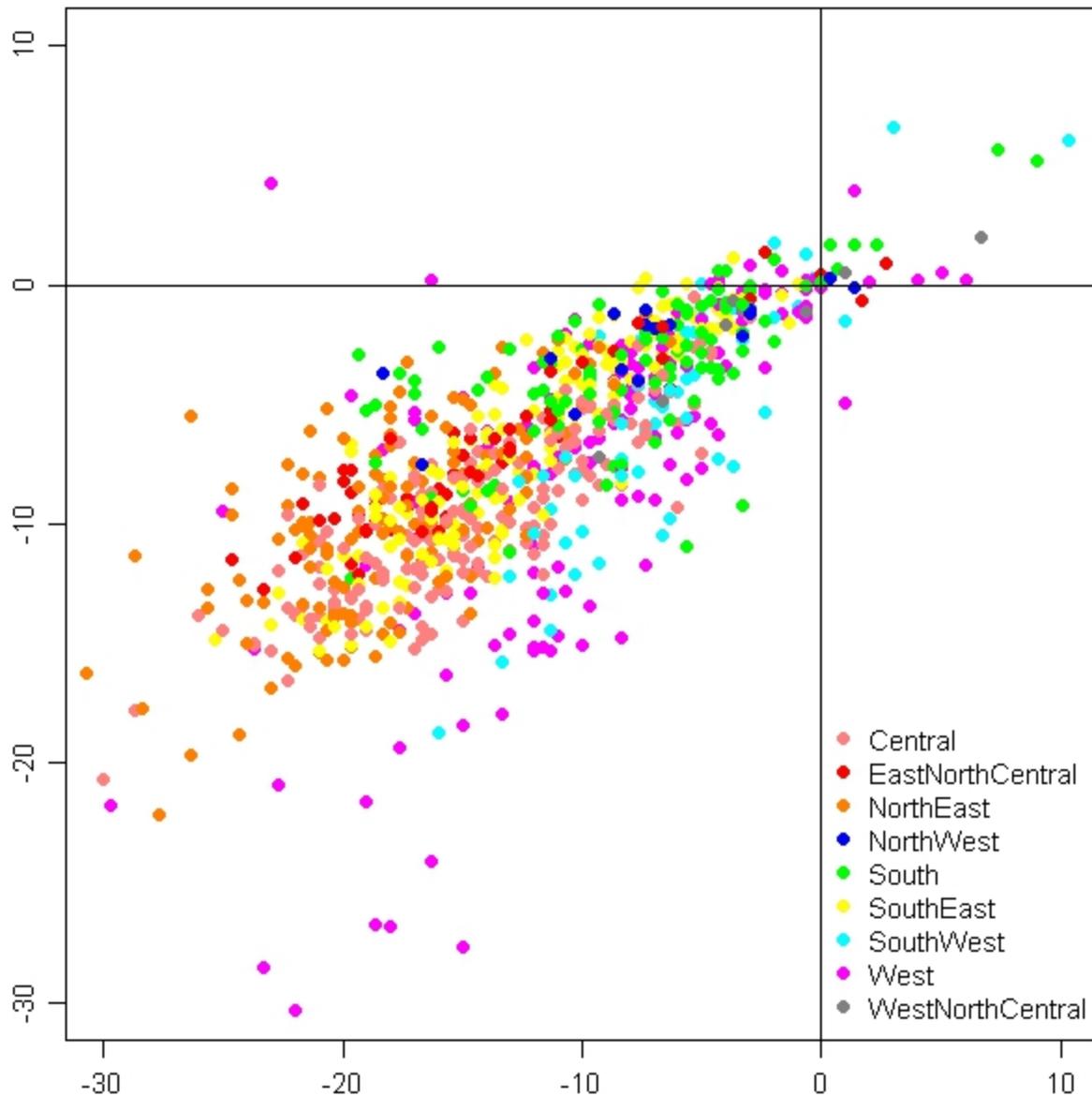
Figure 2B-8. Unit (ppb) change in design values for the current O$_3$ standard from 2001-2003 to 2009-2011 (x-axis) versus unit (ppm-hr) change in 3-year average W126 values from 2001-2003 to 2009-2011 (y-axis).

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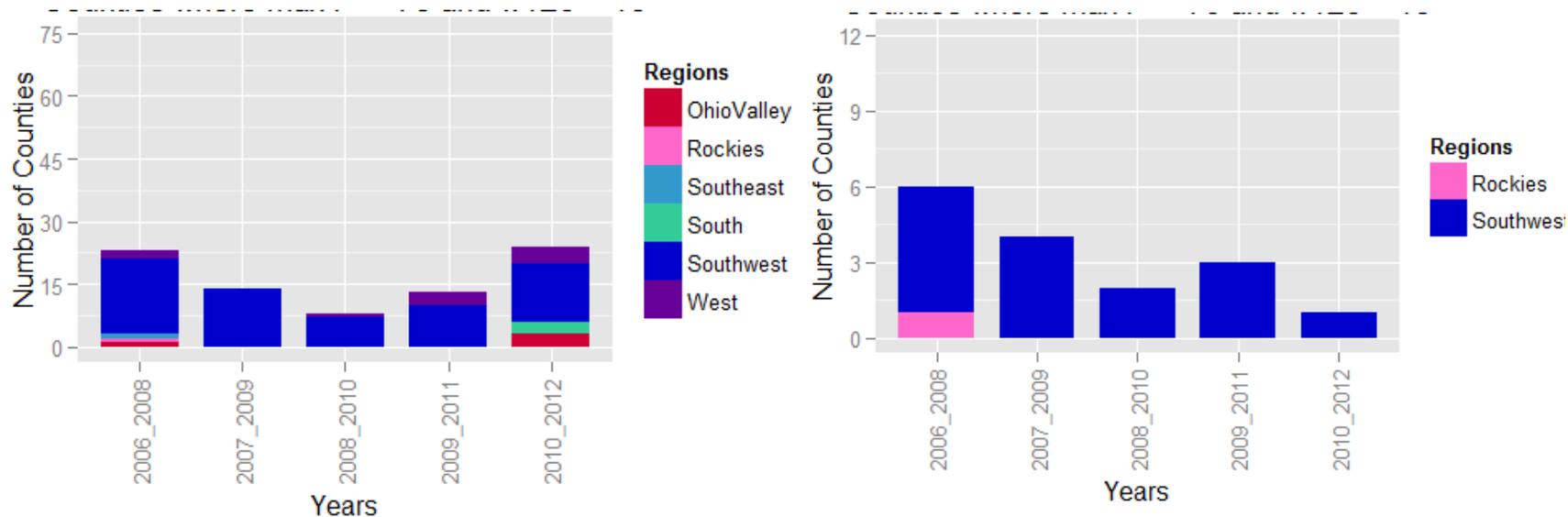
Figure 2B-9. Number of counties where the 8-hour design value is meeting the current standard and 3-year average W126 index value is greater than 15 ppm-hrs (left), and number of counties where the 8-hour design value is less than or equal to 70 ppb and 3-year average W126 index value is greater than15 ppm-hrs (right)[3].

131

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[3] No counties in any of the studied 3-year periods were at or below a 3-year average of 4th highest daily maximum 8-hour averages of 65 ppb and also above a 3-year W126 index value of 15 ppm-hrs.

1 **APPENDIX 2C**

2 **INTER-ANNUAL VARIABILITY IN W126 INDEX VALUES:**
3 **COMPARING ANNUAL AND 3-YEAR AVERAGE METRICS (2008-2010)**

4 **2C.1 OVERVIEW**

5   This appendix describes an analysis comparing values for a single-year or annual W126
6 metric to a W126 metric averaged over three consecutive years. The purpose of this analysis is
7 to compare values based on a 3-year average of annual W126 indices to values based on a single
8 annual W126 index. The deviations of the annual W126 index values in 2008, 2009, and 2010
9 from the 2008-2010 average W126 index values are presented.

10 **2C.2 GENERAL DATA PROCESSING**

11   The air quality data for this analysis originated from EPA's Air Quality System (AQS)
12 data base, the official repository of ambient air measurements. The data used in this analysis
13 consisted of W126 index values calculated from hourly ozone concentrations measured at 1082
14 ozone monitors nationwide. Ozone monitors must have submitted data to AQS for at least 75%
15 days in their required ozone monitoring season in 2008, 2009, and 2010 to be included in the
16 analysis.

17 **2C.3 RESULTS & CONCLUSION**

18   The figure below shows a scatter plot of the deviations in the annual W126 index from
19 the 3-year average by monitor. The solid curves represent the average deviation in a moving
20 window along the x-axis for each year. From this figure, it is apparent that the highest annual
21 W126 index value occurred in 2008 for most monitoring locations, the lowest annual W126
22 index value occurred in 2009 for most monitoring locations, and the 2010 W126 index value was
23 generally somewhere in between. It is also apparent that the inter-annual variability in the W126
24 index increases along with the 3-year average. For monitors with 3-year average W126 values
25 near 15 ppm-hrs, the average deviation was +3.5 ppm-hrs in 2008 and -3.8 ppm-hrs in 2009.
26 This represents a 1-year swing of -7.3 ppm-hrs.
27   The model-based air quality adjustments in the 2$^{\text{nd}}$ draft of the O$_3$ Welfare REA show
28 that reducing NOx emissions is effective for reducing 3-year average W126 levels. In Appendix
29 2B, the analyses based on ambient monitoring data also show that large-scale reductions in NOx
30 emissions are associated with lower W126 levels. Finally, the data analysis presented in this
31 appendix shows that the inter-annual variability in the annual W126 index tends to decrease with
32 decreasing W126 levels. Thus, it is expected that reductions in NOx emissions will not only

33   result in lower 3-year average W126 levels, but also result in less inter-annual variability

34   associated with annual W126 levels.

35   The W126 index is based on a logistic weighting function that increases the weights

36   assigned to hourly ozone concentrations very rapidly. Hourly ozone concentrations of 50 parts

37   per billion are given a weight of about 10% while concentrations of 80 parts per billion are given

38   a weight of nearly 90%. The annual W126 index is calculated as a 3-month sum of weighted

39   ozone concentrations during daylight hours, which amounts to a sum of roughly 1100 weighted

40   hourly concentrations. Thus, even a modest change in the average daily ozone level may have a

41   significant impact upon the annual W126 index. Since ozone formation is heavily influenced by

42   meteorology, the inter-annual variability in meteorological conditions tends to cause a large

43   inter-annual variability in the W126 index.

44   In conclusion, this evaluation indicates the extent to which a form for the secondary

45   ozone standard that averages the annual W126 index values over three consecutive years might

46   be expected to account for the annual variability in this index since the 3-year period would be

47   expected to include year(s) below as well as above the 3-year average.

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Annual W126 Index vs. 3-Year Average W126 Index
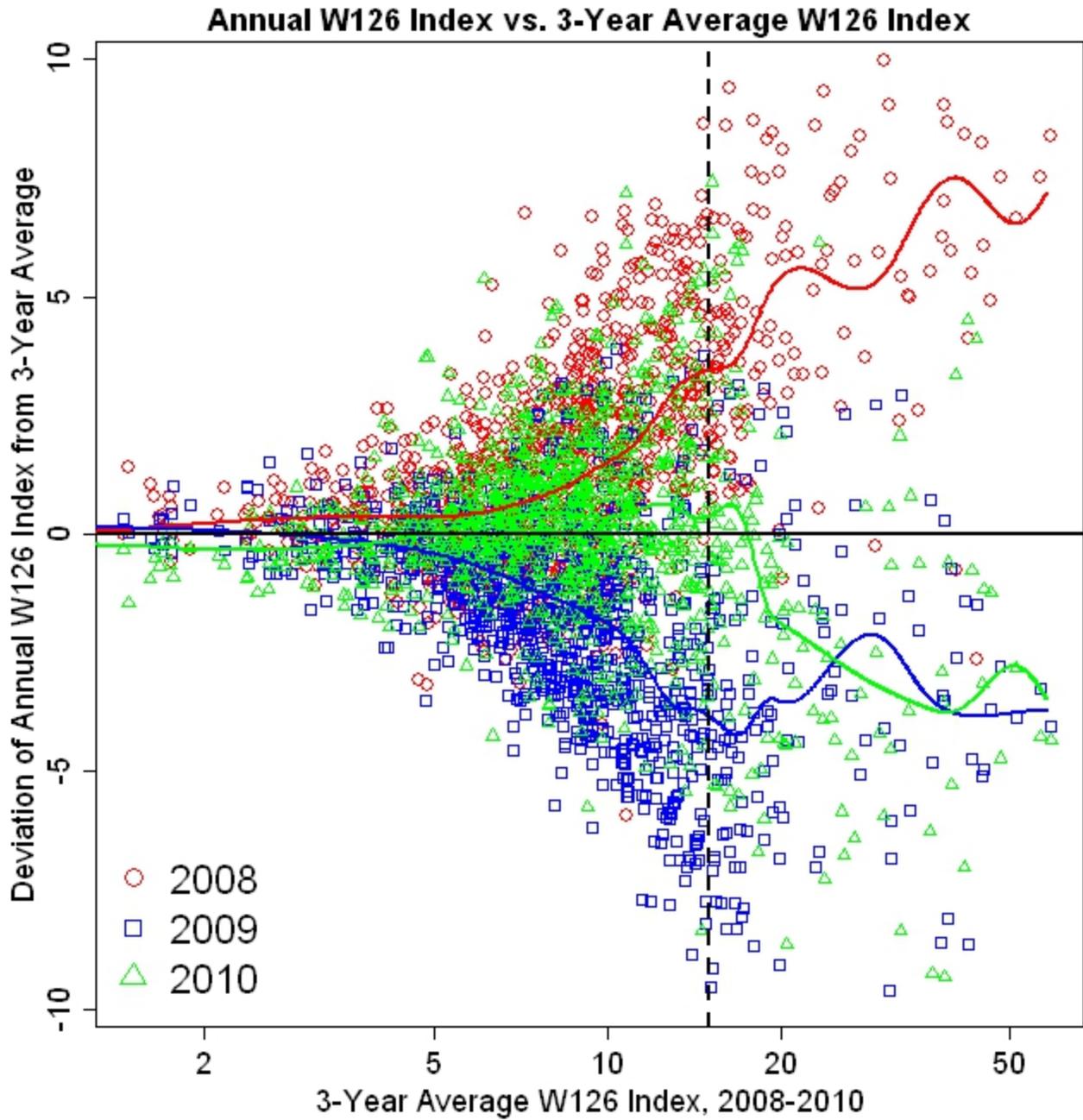
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51   **Figure 2C-1. Deviation of the annual W126 index values in 2008, 2009, and 2010 (y-axis)**
52   **from the 3-year average W126 index value (x-axis).**

MODES OF ACTION SUMMARY

1

2

3   The initial key event in the toxicity pathway of O_3 is the formation of secondary

4   oxidation products in the respiratory tract (ISA, section 5-3, U.S. EPA, 2013). This mainly

5   involves direct reactions with components of the extracellular lining fluid (ELF)[1]. The resulting

6   secondary oxidation products transmit signals to the epithelium, pain receptive nerve fibers and,

7   if present, immune cells (i.e., eosiniphils, dendritic cells and mast cells). Thus, the effects of O_3

8   are mediated by components of ELF and by the multiple cell types found in the respiratory tract.

9   Further, oxidative stress[2] is an implicit part of this initial key event.

10   Another key event in the toxicity pathway of O_3 is the activation of neural reflexes which

11   lead to lung function decrements. Evidence is accumulating that secondary oxidation products

12   are responsible for this effect. Different receptors on bronchial sensory nerves (i.e., C-fibers)

13   have been shown to mediate separate effects of O_3 on pulmonary function. For example, pain

14   (i.e., nociceptive) sensory nerves are involved in the involuntary truncation of inspiration which

15   results in decreases in FVC, FEV_1, tidal volume and pain upon deep inspiration. Ozone exposure

16   also results in activation of vagal sensory nerves and a mild increase in airway obstruction

17   measured as increased sRaw. Activation of neural reflexes also results in extrapulmonary effects

18   such as slow resting heart rate (i.e., bradycardia).

19   Initiation of inflammation is also a key event in the toxicity pathway of O_3. Secondary

20   oxidation products, as well as cell signaling molecules (i.e., chemokines and cytokines) from

21   airway epithelial cells and white blood cells (i.e., macrophages), have been implicated in the

22   initiation of inflammation. Airways neutrophilia has been demonstrated in bronchoalveolar

23   lavage fluid (BALF), mucosal biopsy and induced sputum samples. Influx of other cells (i.e.

24   mast cells, monocytes and macrophages) also occur. Inflammation further contributes to

25   O_3-mediated oxidative stress. It should be noted that inflammation, as measured by airways

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[1] The ELF is a complex mixture of lipids (fats), proteins, and antioxidants that serve as the first barrier and target for inhaled O3. The antioxidant substances present in the ELF appear in most cases to limit interaction of O3 with underlying tissues and to prevent penetration of O3 deeper into the lung. However, as the ELF thickness decreases and becomes ultra thin in the alveolar region, it may be possible for direct interaction of O3 with the underlying epithelial cells to occur. The formation of secondary oxidation products is likely related to the concentration of antioxidants present and the quenching ability of the ELF.

[2] Oxidative stress reflects an imbalance between the systemic manifestation of reactive oxygen species, such as superoxide, and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage.

1   neutrophilia, is not correlated with decrements in pulmonary function as measured by

2   spirometry.

3   A fourth key event in the toxicity pathway of O_3 is alteration of epithelial barrier

4   function. Increased permeability[3] occurs as a result of damage to tight junctions between

5   epithelial cells subsequent to O_3-induced injury and inflammation. It may play a role in allergic

6   sensitization and in airway hyperresponsiveness (AHR). Genetic susceptibility has been

7   associated with this pathway.

8   A fifth key event in the toxicity pathway of O_3 is the sensitization of bronchial smooth

9   muscle. Airway hyperresponsiveness (AHR), or increased bronchial reactivity, can be both a

10   rapidly occurring and a persistent response. The mechanisms responsible for early and later AHR

11   are not well-understood. Tachykinins, peptides that can excite neurons and cause smooth muscle

12   contraction, and the secondary oxidation products of O_3 have been proposed as mediators of the

13   early response and inflammation-derived products have been proposed as mediators of the later

14   response. Other chemical signaling molecules (i.e., cytokines and chemokines) have been

15   implicated in the AHR response to O_3 in animal models. Antioxidants may confer protection.

16   A sixth key event in the toxicity pathway of O_3 is the modification of innate/adaptive

17   immunity. While the majority of evidence for this key event comes from animal studies, there

18   are several studies suggesting that this pathway may also be relevant in humans. Ozone exposure

19   of human subjects resulted in recruitment of activated innate immune cells to the airways.

20   Animal studies further linked O_3-mediated activation of the innate immune system to the

21   development of nonspecific AHR, demonstrated an interaction between allergen and O_3 in the

22   induction of nonspecific AHR, and found that O_3 acted as an adjuvant for allergic sensitization

23   through the activation of both innate and adaptive immunity. These studies provide evidence that

24   O_3 can alter host immunologic response and lead to immune system dysfunction. These

25   mechanisms may underlie the exacerbation and induction of asthma, as well as decreases in lung

26   host defense.

27   Another key event in the toxicity pathway of O_3 is airways remodeling. Persistent

28   inflammation and injury, which are observed in animal models of chronic and intermittent

29   exposure to O_3, are associated with morphologic changes such as mucous cell metaplasia[4] of

---

[3] Cells in epithelium are very densely packed together, leaving very little intercellular space. All epithelial cells rest on a basement membrane, a thin sheet of fibers that acts as scaffolding on which epithelium can grow and regenerate after injuries. Epithelial tissue is innervated but avascular; it must be nourished by substances diffusing from the blood vessels in the underlying tissue. Injury to epithelial cells, such as caused by oxidative stress, can cause the epithelium to become more permeable to substances in the underlying vasculature.

[4] Metaplasia is the reversible replacement of one differentiated cell type with another mature differentiated cell type. The change from one type of cell to another may generally be a part of normal maturation process or

1   nasal epithelium, bronchiolar metaplasia of alveolar ducts and fibrotic changes in small airways

2   (see Section 7.2.3 of the ISA, U.S. EPA 2013). Mechanisms responsible for these responses are

3   not well-understood. However, a recent study in mice demonstrated a key role for a signaling

4   pathway in the deposition of collagen in the airway wall following chronic intermittent exposure

5   to O_3. Chronic intermittent exposure to O_3 has also been shown to result in effects on the

6   developing lung and immune system.

7   Systemic inflammation and vascular oxidative/nitrosative stress are also key events in the

8   toxicity pathway of O_3. Extrapulmonary effects of O_3 occur in numerous organ systems,

9   including the cardiovascular, central nervous, reproductive, and hepatic systems (see

10   Sections 6.3 to 6.5 and Sections 7.3 to 7.5 of the ISA, U.S. EPA, 2013). It has been proposed that

11   lipid oxidation products resulting from reaction of O_3 with lipids and/or cellular membranes in

12   the ELF are responsible for systemic responses; however, it is not known whether they gain

13   access to the circulation. Alternatively, release of diffusible mediators from the lung into the

14   circulation may initiate or propagate inflammatory responses in the circulation or other organ

15   systems.

16   Responses to O_3 exposure are variable within the population. Studies have shown a large

17   range of pulmonary function responses to O_3 among healthy young adults. Since individual

18   responses were relatively consistent across time in some of these studies, it was thought that

19   responsiveness reflected an intrinsic characteristic of the subject (Mudway and Kelly, 2000).

20   Other responses to O_3 have also been characterized by a large degree of inter-individual

21   variability. For example, inter-individual variability in the neutrophilic response has been noted

22   in human subjects. Two studies demonstrated a 3- to 20-fold difference in airways neutrophilia,

23   under different exposure conditions (Schelegle et al., 1991 and Devlin et al., 1991, respectively).

24   Reproducibility of intraindividual responses to O_3 exposures in human subjects, measured as

25   sputum neutrophilia, was demonstrated by Holz et al (1999). While the basis for the observed

26   inter-individual variability in responsiveness to O_3 is not clear, both dosimetric and mechanistic

27   factors are likely to contribute. These are discussed in sections 5.4.1 and 5.4.2 in the ISA (U.S.

28   EPA, 2013). Section 5.4.2 of the ISA discusses studies that provide evidence for the mechanisms

29   that may underlie the variability in responsiveness seen among individuals. Certain functional

30   genetic polymorphisms, pre-existing conditions or diseases, nutritional status, lifestages, and co-

31   exposures contribute to altered risk of O_3-induced effects.

32

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caused by some sort of abnormal stimulus. In simplistic terms, it is as if the original cells are not robust enough to
withstand the new environment, and so they change into another type more suited to the new environment. If the
stimulus that caused metaplasia is removed or ceases, tissues return to their normal pattern of differentiation.

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RECENT STUDIES OF RESPIRATORY-RELATED EMERGENCY DEPARTMENT VISITS AND HOSPITAL ADMISSIONS

Hospital Admissions for All Respiratory Causes

The APHENA study (APHENA is for Air Pollution and Health: A European and North American Approach) analyzed air pollution and health outcome data from existing Canadian, European, and U.S. multi-city studies and examined the influence of varying model specification to control for season and weather (Katsouyanni et al., 2009). The U.S.-based portion of the APHENA study utilized the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) cohort which, for the Katsouyanni et al. (2009) analysis, comprised respiratory hospital admissions among individuals 65 years of age and older from 14 US cities with O_3 data from 1985-1994 (7 cities had summer only O_3 data). For the year round analysis, Katsouyanni et al. (2009) reported consistently positive, and statistically significant in models with 8 degrees of freedom per year (U.S. EPA, 2013, section 6.2.7.2), associations between 1-hour O_3 concentrations and respiratory hospital admissions across the datasets from the U.S., Canada, and Europe (U.S. EPA 2013, Figure 6-15).[5] In co-pollutant models adjusting for PM_{10}, O_3 effect estimates remained positive, though effect estimates were somewhat attenuated in the U.S. and European datasets, possibly due to the PM sampling schedule (U.S. EPA 2013, Figure 6-15). Effect estimates for the warm season were larger than for the year-round analysis in the Canadian dataset, but generally similar in magnitude to the year-round analysis in the U.S. and European datasets.

Several additional multicity studies examined respiratory disease hospital admissions in Canada and Europe. Cakmak et al. (2006) reported a statistically significant increase in respiratory hospital admissions in 10 Canadian cities (4.4% increase per 20 ppb increase in 24-hour average O_3, 95% CI: 2.2, 6.5%). In analyses of potential effect modifiers of the O_3-respiratory hospital admission relationship, individuals with an education level less than the 9th grade were found to be at greater risk. Dales et al. (2006) reported a 5.4% (95% CI: 2.9, 8.0%) increase in neonatal respiratory hospital admissions for a 20 ppb increase in 24-hour average O_3

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[5]The study by Katsouyanni et al. (2009) evaluated different statistical models. Although the investigators did not identify the model they deemed to be the most appropriate for comparing the results across study locations, they did specify that "overall effect estimates (i.e., estimates pooled over several cities) tended to stabilize at high degrees of freedom" (Katsouyanni et al., 2009). In discussing of the results of this study, the ISA focused on models with 8 degrees of freedom per year (US EPA, 2012a, section 6.2.7.2).

1 concentrations in 11 Canadian cities from 1986 to 2000. In contrast, Biggeri et al. (2005) did not

2 detect an association between short-term O_3 exposure and respiratory hospital admissions in four

3 Italian cities from 1990 to 1999.

4   In addition to the large multi-city studies discussed above, several smaller-scale studies

5 have also reported associations with total respiratory hospital admissions. Specifically, Lin et al.

6 (2008) reported a positive association between O_3 and pediatric (i.e., <18 years) respiratory

7 admissions in an analysis of 11 geographic regions in New York state from 1991 to 2001, though

8 results were not presented quantitatively. In co-pollutant models with PM_{10}, the authors reported

9 that region-specific O_3 associations with respiratory hospital admissions remained relatively

10 robust.

11 **Cause-Specific Hospital Admissions**

12   With regard to cause-specific respiratory outcomes, the limited evidence available in the

13 last review indicated that the strongest findings were for ambient O_3 associated asthma and

14 chronic obstructive pulmonary disease (COPD) respiratory hospital admissions (U.S. EPA 2013,

15 6.2.7.2). Since the last review, a few additional studies have investigated cause-specific

16 respiratory admissions (i.e., COPD, asthma, pneumonia) in relation to O_3 exposure (Medina-

17 Ramon et al, 2006; Yang et al., 2005; Zanobetti and Schwartz, 2006; Silverman and Ito, 2010).

18   Medina-Ramon et al. (2006) examined the association between short-term ambient O_3

19 concentrations and Medicare hospital admissions for COPD among individuals ≥ 65 years of age

20 for COPD in 35 cities in the U.S. for the years 1986-1999. The authors reported an increase in

21 COPD admissions for lag 0-1 day in the warm season for a 30 ppb increase in 8-h max O_3

22 concentrations. The authors found no evidence for such associations in cool season or in year

23 round analyses. In a co-pollutant model with PM_{10}, the association between O_3 and COPD

24 hospital admissions remained robust. In Vancouver from 1994-1998, a location with low ambient

25 O_3 concentrations (U.S. EPA, 2013, Table 6-26), Yang et al. (2005) reported a statistically non-

26 significant increase in COPD admissions per 20 ppb increase in 24-hour average O_3

27 concentrations. In two-pollutant models with every-day data for NO_2, SO_2, CO, and PM_{10}, O_3

28 risk estimates remained robust, though not statistically significant (U.S. EPA, 2013, Figure 6-20;

29 Table 6-29). In addition, Wong et al. (2009) reported increased O_3-associated COPD admissions

30 during periods of increased influenza activity in Hong Kong.

31   The ISA assessed a study that evaluated asthma-related hospital admissions in New York

32 City (U.S. EPA, 2013, section 6.2.7.2) (Silverman and Ito, 2010). This study examined the

33 association of 8-hour max O_3 concentrations with severe acute asthma admissions (i.e., those

34 admitted to the Intensive Care Unit [ICU]) during the warm season in the years 1999 through

1   2006 (Silverman and Ito, 2010)). The investigators reported positive associations between O_3 and

2   ICU asthma admissions for the 6- to 18-year age group for a 30 ppb increase in max 8-hour

3   average O_3 concentrations, but little evidence of associations for the other age groups examined

4   (<6 years, 19-49, 50+, and all ages). However, positive associations were observed for each

5   age-stratified group and all ages for non-ICU asthma admissions, but again the strongest

6   association was reported for the 6- to 18-years age group. In two-pollutant models, O_3 effect

7   estimates for both non-ICU and ICU hospital admissions remained robust to adjustment for

8    $PM_{2.5}$. In an additional analysis, using a smooth function, the authors examined whether the

9   shape of the concentration-response curve for O_3 and asthma hospital admissions (i.e., both

10   general and ICU for all ages) is linear. When comparing the curve to a linear fit line, the authors

11   found that the linear fit was a reasonable approximation of the concentration-response

12   relationship between O_3 and asthma hospital admissions, but the limited data density at relatively

13   low O_3 concentrations contributes to uncertainty in the shape of the concentration-response

14   relationship at the low end of the distribution of O_3 concentrations (U.S. EPA, 2013, Figure 6-

15   16).

16   In contrast to COPD and asthma, the evidence for pneumonia-related admissions was less

17   consistent. Medina-Ramon et al. (2006) examined the association between short-term ambient O_3

18   concentrations and Medicare hospital admissions among individuals $\geq$ 65 years of age for

19   pneumonia. The authors reported an increase in pneumonia hospital admissions in the warm

20   season for a 30 ppb increase in 8-hour max O_3 concentrations, with no evidence of an association

21   in the cool season or year round. In two-pollutant models restricted to days for which PM_{10} data

22   was available, the association between O_3 exposure and pneumonia hospital admissions

23   remained robust. In contrast, Zanobetti and Schwartz (2006) reported a decrease in pneumonia

24   admissions for a 20 ppb increase in 24-hour average O_3 concentrations in Boston for the average

25   of lags 0 and 1 day.

26   The magnitude of associations with respiratory-related hospital admissions may be

27   underestimated due to behavioral modification in response to forecasted air quality (U.S. EPA,

28   2013, section 4.6.6). Recent studies (Neidell and Kinney, 2010; Neidell, 2009) conducted in

29   Southern California demonstrates that controlling for avoidance behavior increases O_3 effect

30   estimates for respiratory hospital admissions, specifically for children and older adults. This

31   study shows that on days where no public alert warning of high O_3 concentrations was issued,

32   there was an increase in asthma hospital admissions. Although only one study has examined

33   averting behavior and this study is limited to the outcome of asthma hospital admissions in one

34   location and time period (i.e., Los Angeles, CA for the years 1989-1997), it does provide

35   preliminary evidence indicating that some epidemiologic studies may underestimate associations

1   between O_3 and health effects by not accounting for behavioral modification when public health
2   alerts are issued.

Emergency Department Visits for All Respiratory Causes

4   A large single-city study conducted in Atlanta by Tolbert et al. (2007), and subsequently
5   reanalyzed by Darrow et al. (2011) using different air quality data and evaluating associations
6   with different metrics, provides evidence for associations between short-term exposures to
7   ambient O_3 concentrations and respiratory emergency department visits. Tolbert et al. (2007)
8   reported an increase in respiratory emergency department visits for a 30 ppb increase in 8-hour
9   max O_3 concentrations during the warm season. In copollutant models with CO, NO_2, and PM_{10},
10   limited to days in which data for all pollutants were available, associations between O_3 and
11   respiratory emergency department visits remained positive, but were attenuated. Darrow et al.
12   (2011) reported the strongest associations with respiratory emergency department visits for 8-
13   hour daily max, 1-hour daily max, and day-time O_3 exposure metrics (all associations positive
14   and statistically significant), while positive, but statistically non-significant, associations were
15   reported with 24-hour average and commuting period exposure metrics. In addition, a negative
16   association was observed when using the night-time exposure metric (U.S. EPA, 2013, Figure 6-
17   17). The results of Darrow et al. (2011) suggest that averaging over nighttime hours may lead to
18   smaller O_3 effect estimates for respiratory emergency department visits due to dilution of
19   relevant O_3 concentrations (i.e., the higher concentrations that occur during the daytime); and
20   potential negative confounding by other pollutants (e.g., CO, NO_2) during the nighttime hours
21   (U.S. EPA, 2013, section 6.2.7.3)

Cause-Specific Emergency Department Visits

23   In evaluating asthma emergency department visits in an all-year analysis, a Canadian
24   multi-city study (Stieb et al., 2009) reported that 24-hour O_3 concentrations were positively
25   associated with emergency department visits for asthma at lag 1 and lag 2. Though the authors
26   did not present seasonal analyses, they stated that no associations were observed with emergency
27   department visits in the winter season, suggesting that the positive associations reported in the
28   all-year analysis were due to the warm season (Stieb et al., 2009). In addition to asthma, the
29   authors reported that O_3 was positively associated with COPD emergency department visits in
30   all-year analyses, but that associations with COPD visits were statistically significant only for the
31   warm season (i.e., April-September).

1   Several single-city studies have also provided evidence for positive associations between

2   asthma emergency department visits and ambient O_3 concentrations. Ito et al. (2007) reported

3   positive and statistically significant associations with asthma emergency department visits in

4   New York City during the warm season, and an inverse association in the cool season, for a 30

5   ppb increase in 8-hour max O_3 concentrations. In two-pollutant models with $PM_{2.5}$, NO_2, SO_2,

6   and CO, the authors found that O_3 risk estimates were not substantially changed during the warm

7   season (U.S. EPA, 2013, Figure 6-20; Table 6-29).

8   Strickland et al. (2010) examined the association between O_3 exposure and pediatric

9   asthma emergency department visits (ages 5-17 years) in Atlanta using air quality data over the

10   same years as Darrow et al. (2011) and Tolbert et al. (2007), but using population-weighting to

11   combine daily pollutant concentrations across monitors. Strickland et al. (2010) reported an

12   increase in emergency department visits for a 30 ppb increase in 8-hour max O_3 concentrations

13   in an all-year analysis. In seasonal analyses, stronger associations were observed during the

14   warm season (i.e., May-October) than the cold season. In co-pollutant analyses that included CO,

15   NO_2, $PM_{2.5}$ elemental carbon, or $PM_{2.5}$ sulfate, Strickland et al. (2010) reported that O_3 risk

16   estimates were not substantially changed. The authors also examined the concentration-response

17   relationship between O_3 exposure and pediatric asthma emergency department visits and

18   reported that positive associations with O_3 persist at 8-hour ambient O_3 concentrations (3-day

19   average of 8-hour daily max concentrations) at least as low as 30 ppb.

20   In a single-city study conducted in Seattle, WA, Mar and Koenig (2009) examined the

21   association between O_3 exposure and asthma emergency department visits for children (< 18)

22   and adults (≥ 18). For children, positive and statistically significant associations were reported

23   across multiple lags, with the strongest associations observed at lag 0 and lag 3. Ozone was also

24   found to be positively associated with asthma emergency department visits for adults at all lags,

25   except at lag 0. The slightly different lag times for children and adults suggest that children may

26   be more immediately responsive to O_3 exposures than adults (Mar and Koenig, 2009).

27   In addition to the U.S. single-city studies discussed above, a single-city study conducted

28   in Alberta, Canada (Villeneuve et al., 2007) provides support for the findings from Stieb et al.

29   (2009), but also attempts to identify those lifestages at greatest risk for O_3-associated asthma

30   emergency department visits. Villeneuve et al. reported an increase in asthma emergency

31   department visits in an all-year analysis across all ages with associations being stronger during

the warmer months. When stratified by age, the strongest associations were observed in the
warm season for individuals 5-14 and 15-44. These associations were not found to be
confounded by the inclusion of aeroallergens in age-specific models.

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APPENDIX 3C

AT-RISK POPULATIONS

3 **People with Specific Genetic Variants**

4   Overall, for variants in multiple genes there is adequate evidence for involvement in
5 populations being more at-risk than others to the effects of O_3 exposure on health (U.S. EPA,
6 2013, section 8.1). Controlled human exposure and epidemiologic studies have reported evidence
7 of O_3-related increases in respiratory symptoms or decreases in lung function with variants
8 including GSTM1, GSTP1, HMOX1, and NQO1. NQO1 deficient mice were found to be
9 resistant to O_3-induced AHR and inflammation, providing biological plausibility for results of
10 studies in humans. Additionally, studies of rodents have identified a number of other genes that
11 may affect O_3-related health outcomes, including genes related to innate immune signaling and
12 pro- and anti-inflammatory genes, which have not been investigated in human studies.

13 **People with Asthma**

14   Previous O_3 AQCDs identified individuals with asthma as a population at increased risk
15 of O_3-related health effects. Multiple new epidemiologic studies included in the ISA have
16 evaluated the potential for increased risk of O_3-related health effects in people with asthma,
17 including: lung function; symptoms; medication use; airway hyperresponsiveness (AHR); and
18 airway inflammation (also measured as exhaled nitric oxide fraction, or FeNO). A study of
19 lifeguards in Texas reported decreased lung function with short-term O_3 exposure among both
20 individuals with and without asthma, however, the decrease was greater among those with
21 asthma (Thaller et al., 2008). A Mexican study of children ages 6-14 detected an association
22 between short-term O_3 exposure and wheeze, cough, and bronchodilator use among asthmatics
23 but not non-asthmatics, although this may have been the result of a small non-asthmatic
24 population (Escamilla-Nuñez et al., 2008). A study of modification by AHR (an obligate
25 condition among asthmatics) reported greater short-term O_3-associated decreases in lung
26 function in elderly individuals with AHR, especially among those who were obese (Alexeeff et
27 al., 2007). With respect to airway inflammation, in one study, a positive association was reported
28 for airway inflammation among asthmatic children following short-term O_3 exposure, but the
29 observed association was similar in magnitude to that of non-asthmatics (Barraza-Villarreal et
30 al., 2008). Similarly, another study of children in California reported an association between O_3
31 concentration and FeNO that persisted both among children with and without asthma as well as
32 those with and without respiratory allergy (Berhane et al., 2011). Finally, Khatri et al. (2009)
33 found no association between short-term O_3 exposure and altered lung function for either

1   asthmatic or non-asthmatic adults, but did note a decrease in lung function among individuals

2   with allergies.

3   New evidence for difference in effects among asthmatics has been observed in studies

4   that examined the association between O_3 exposure and altered lung function by asthma

5   medication use. A study of children with asthma living in Detroit reported a greater association

6   between short-term O_3 and lung function for corticosteroid users compared with

7   noncorticosteroid users (Lewis et al., 2005). Conversely, another study found decreased lung

8   function among noncorticosteroid users compared to users, although in this study, a large

9   proportion of non-users were considered to be persistent asthmatics (Hernández-Cadena et al.,

10   2009). Lung function was not related to short-term O_3 exposure among corticosteroid users and

11   non-users in a study taking place during the winter months in Canada (Liu et al., 2009).

12   Additionally, a study of airway inflammation reported a counterintuitive inverse association with

13   O_3 of similar magnitude for all groups of corticosteroid users and non-users (Qian et al., 2009).

14   Controlled human exposure studies that have examined the effects of O_3 on adults with

15   asthma and healthy controls are limited. Based on studies reviewed in the 1996 and 2006 O_3

16   AQCDs, subjects with asthma appeared to be more sensitive to acute effects of O_3 in terms of

17   FEV_1 and inflammatory responses than healthy non-asthmatic subjects. For instance, Horstman

18   et al. (1995) observed that mild-to-moderate asthmatics, on average, experienced double the

19   O_3-induced FEV_1 decrement of healthy subjects (19% versus 10%, respectively, p = 0.04).

20   Moreover, a statistically significant positive correlation between FEV_1 responses to O_3 exposure

21   and baseline lung function was observed in individuals with asthma, i.e., responses increased

22   with severity of disease. Minimal evidence exists suggesting that individuals with asthma have

23   smaller O_3-induced FEV_1 decrements than healthy subjects (3% versus 8%, respectively)

24   (Mudway et al., 2001). However, the asthmatics in that study also tended to be older than the

25   healthy subjects, which could partially explain their lesser response since FEV_1 responses to O_3

26   exposure diminish with age. Individuals with asthma also had significantly more neutrophils in

27   the BALF (18 hours postexposure) than similarly exposed healthy individuals (Peden et al.,

28   1997; Scannell et al., 1996; Basha et al., 1994). Furthermore, a study examining the effects of O_3

29   on individuals with atopic asthma and healthy controls reported that greater numbers of

30   neutrophils, higher levels of cytokines and hyaluronan, and greater expression of macrophage

31   cell-surface markers were observed in induced sputum of atopic asthmatics compared with

32   healthy controls (Hernandez et al., 2010). Differences in O_3-induced epithelial cytokine

33   expression were noted in bronchial biopsy samples from asthmatics and healthy controls (Bosson

34   et al., 2003). Cell-surface marker and cytokine expression results, and the presence of

35   hyaluronan, are consistent with O_3 having greater effects on innate and adaptive immunity in

36   these asthmatic individuals. In addition, studies have demonstrated that O_3 exposure leads to

1   increased bronchial reactivity to inhaled allergens in mild allergic asthmatics (Kehrl et al., 1999;

2   Jorres et al., 1996) and to the influx of eosinophils in individuals with pre-existing allergic

3   disease (Vagaggini et al., 2002; Peden et al., 1995). Taken together, these results point to several

4   mechanistic pathways which could account for the enhanced sensitivity to O_3 in subjects with

5   asthma (see Section 5.4.2.2 in the ISA).

6   Toxicological studies provide additional evidence of the biological basis for the greater

7   effects of O_3 among those with asthma or AHR (U.S. EPA, 2013, section 8.2.2). In animal

8   toxicological studies, an asthmatic phenotype is modeled by allergic sensitization of the

9   respiratory tract. Many of the studies that provide evidence that O_3 exposure is an inducer of

10 AHR and remodeling utilize these types of animal models. For example, a series of experiments

11 in infant rhesus monkeys have shown these effects, but only in monkeys sensitized to house dust

12 mite allergen. Similarly, adverse changes in pulmonary function were demonstrated in mice

13 exposed to O_3; enhanced inflammatory responses were in rats exposed to O_3, but only in animals

14 sensitized to allergen. In general, it is the combined effects of O_3 and allergic sensitization which

15 result in measurable effects on pulmonary function. In a pulmonary fibrosis model, exposure O_3

16 for 5 days increased pulmonary inflammation and fibrosis, along with the frequency of

17 bronchopneumonia in rats. Thus, short-term exposure to O_3 may enhance damage in a previously

18 injured lung (U.S. EPA, 2013, section 8.2.2).

19 In the 2006 O_3 AQCD, the potential for individuals with asthma to have greater risk of

20 O_3-related health effects was supported by a number of controlled human exposure studies,

21 evidence from toxicological studies, and a limited number of epidemiologic studies. In section

22 8.2.2, the ISA reports that in the recent epidemiologic literature some, but not all, studies report

23 greater risk of health effects among individuals with asthma. Studies examining effect measure

24 modification of the relationship between short-term O_3 exposure and altered lung function by

25 corticosteroid use provided limited evidence of O_3-related health effects. However, recent studies

26 of behavioral responses have found that studies do not take into account individual behavioral

27 adaptations to forecasted air pollution levels (such as avoidance and reduced time outdoors),

28 which may underestimate the observed associations in studies that examined the effect of O_3

29 exposure on respiratory health (Neidell and Kinney, 2010). This could explain some

30 inconsistency observed among recent epidemiologic studies. The evidence from controlled

31 human exposure studies provides support for increased detriments in FEV_1 and greater

32 inflammatory responses to O_3 in individuals with asthma than in healthy individuals without a

33 history of asthma. The collective evidence for increased risk of O_3-related health effects among

34 individuals with asthma from controlled human exposure studies is supported by recent

35 toxicological studies which provide biological plausibility for heightened risk of asthmatics to

1 respiratory effects due to O_3 exposure. Overall, the ISA finds there is adequate evidence for

2 asthmatics to be an at-risk population.

3 **Children**

4   Children are considered to be at greater risk from O_3 exposure because their respiratory

5 systems undergo lung growth until about 18-20 years of age and are therefore thought to be

6 intrinsically more at risk for O_3-induced damage (U.S. EPA, 2006b). It is generally recognized

7 that children spend more time outdoors than adults, and therefore would be expected to have

8 higher exposure to O_3 than adults. The ventilation rates also vary between children and adults,

9 particularly during moderate/heavy activity. Children aged 11 years and older and adults have

10 higher absolute ventilation rates than children aged 1-11 years. However, children have higher

11 ventilation rates relative to their lung volumes, which tends to increase dose normalized to lung

12 surface area. Exercise intensity has a substantial effect on ventilation rate, with high intensity

13 activities resulting in nearly double the ventilation rate during moderate activity among children

14 and those adults less than 31 years of age. For more information on time spent outdoors and

15 ventilation rate differences by age group, see Section 4.4.1 in the ISA (U.S. EPA, 2013).

16   The 1996 O_3 AQCD reported clinical evidence that children, adolescents, and young

17 adults (<18 years of age) appear, on average, to have nearly equivalent spirometric responses to

18 O_3 exposure, but have greater responses than middle-aged and older adults (U.S. EPA, 1996a).

19 Symptomatic responses (e.g., cough, shortness of breath, pain on deep inspiration) to O_3

20 exposure, however, appear to increase with age until early adulthood and then gradually decrease

21 with increasing age (U.S. EPA, 1996). Complete lung growth and development is not achieved

22 until 18-20 years of age in women and the early 20s for men; pulmonary function is at its

23 maximum during this time as well.

24   Recent epidemiologic studies have examined different age groups and their risk to

25 O_3-related respiratory hospital admissions and emergency department (ED) visits. Evidence for

26 greater risk in children was reported in several studies. A study in Cyprus of short-term O_3

27 concentrations and respiratory hospital admissions (HA) detected possible effect measure

28 modification by age with a larger association among individuals < 15 years of age compared

29 with those > 15 years of age; the effect was apparent only with a 2-day lag (Middleton et al.,

30 2008). Similarly, a Canadian study of asthma-ED visits reported the strongest O_3-related

31 associations among 5- to 14-year olds compared to the other age groups (ages examined 0-75+)

32 (Villeneuve et al., 2007). Greater O_3-associated risk in asthma-related ED visits were also

33 reported among children (<15 years) as compared to adults (15 to 64 years) in a study from

34 Finland (Halonen et al., 2009). A study of New York City hospital admissions demonstrated an

35 increase in the association between O_3 exposure and asthma-related hospital admissions for 6- to

1   18-year olds compared to those < 6 years old and those > 18 years old (Silverman and Ito, 2010).
2   When examining long-term O_3 exposure and asthma HA among children, associations were
3   determined to be larger among children 1 to 2 years old compared to children 2 to 6 years old
4   (Lin et al., 2008). A few studies reported positive associations among both children and adults
5   and no modification of the effect by age.
6   The evidence reported in epidemiologic studies is supported by recent toxicological
7   studies which observed O_3-induced health effects in immature animals. Early life exposures of
8   multiple species of laboratory animals, including infant monkeys, resulted in changes in
9   conducting airways at the cellular, functional, ultra-structural, and morphological levels. The
10   studies conducted on infant monkeys are most relevant for assessing effects in children. Carey et
11   al. (2007) conducted a study of O_3 exposure in infant rhesus macaques, whose respiratory tract
12   closely resemble that of humans. Monkeys were exposed either acutely or in episodes designed
13   to mimic human exposure. All monkeys acutely exposed to O_3 had moderate to marked
14   necrotizing rhinitis, with focal regions of epithelial exfoliation, numerous infiltrating neutrophils,
15   and some eosinophils. The distribution, character, and severity of lesions in episodically exposed
16   infant monkeys were similar to that of acutely exposed animals. Neither exposure protocol for
17   the infant monkeys produced mucous cell metaplasia proximal to the lesions, an adaptation
18   observed in adult monkeys exposed in another study (Harkema et al., 1987). Functional and
19   cellular changes in conducting airways were common manifestations of exposure to O_3 among
20   both the adult and infant monkeys (Plopper et al., 2007). In addition, the lung structure of the
21   conducting airways in the infant monkeys was significantly stunted by O_3 and this aberrant
22   development was persistent 6 months postexposure (Fanucchi et al., 2006).
23   Age may also affect the inflammatory response to O_3 exposure. Toxicological studies
24   reported that the difference in effects among younger lifestage test animals may be due to
25   age-related changes in antioxidants levels and sensitivity to oxidative stress. Further discussion
26   of these studies may be found in section 8.3.1.1 of the ISA (U.S. EPA, 2013, p. 8-18).
27   The previous and recent human clinical and toxicological studies reported evidence of
28   increased risk from O_3 exposure for younger ages, which provides coherence and biological
29   plausibility for the findings from epidemiologic studies. Although there was some inconsistency,
30   generally, the epidemiologic studies reported positive associations among both children and
31   adults or just among children. The interpretation of these studies is limited by the lack of
32   consistency in comparison age groups and outcomes examined. However, overall, the
33   epidemiologic, controlled human exposure, and toxicological studies provide adequate evidence
34   that children are potentially at increased risk of O_3-related health effects.

1 **Older Adults**

2   The ISA notes that older adults are at greater risk of health effects associated with O_3

3   exposure through a variety of intrinsic pathways (U.S. EPA, 2013, section 8.3.1.2). In addition,

4   older adults may differ in their exposure and internal dose. Older adults were outdoors for a

5   slightly longer proportion of the day than adults aged 18-64 years. Older adults also have

6   somewhat lower ventilation rates than adults aged 31 - less than 61 years. For more information

7   on time spent outdoors and ventilation rate differences by age group, see Section 4.4 in the ISA

8   (U.S. EPA, 2013). The gradual decline in physiological processes that occur with aging may lead

9   to increased risk of O_3-related health effects (U.S. EPA, 2006a). Respiratory symptom responses

10 to O_3 exposure appears to increase with age until early adulthood and then gradually decrease

11 with increasing age (U.S. EPA, 1996a); lung function responses to O3 exposure also decline

12 from early adulthood (U.S. EPA, 1996a). The reductions of these responses with age may put

13 older adults at increased risk for continued O_3 exposure. In addition, older adults, in general,

14 have a higher prevalence of preexisting diseases compared to younger age groups and this may

15 also lead to increased risk of O_3-related health effects (U.S. EPA, 2013, section 8.3.1.2). With

16 the number of older Americans increasing in upcoming years (estimated to increase from 12.4%

17 of the U.S. population to 19.7% between 2000 to 2030, which is approximately 35 million and

18 71.5 million individuals, respectively) this group represents a large population potentially at risk

19 of O_3-related health effects (SSDAN CensusScope, 2010a; U.S. Census Bureau, 2010).

20   The majority of recent studies reported greater effects of short-term O_3 exposure and

21 mortality among older adults, which is consistent with the findings of the 2006 O_3 AQCD. A

22 study (Medina-Ramón and Schwartz, 2008) conducted in 48 cities across the U.S. reported larger

23 effects among adults ≥65 years old compared to those < 65 years; further investigation of this

24 study population revealed a trend of O_3-related mortality risk that gets larger with increasing age

25 starting at age (Zanobetti and Schwartz, 2008). Another study conducted in 7 urban centers in

26 Chile reported similar results, with greater effects in adults ≥65 years old (Cakmak et al., 2007).

27 More recently, a study conducted in the same area reported similar associations between O_3

28 exposure and mortality in adults aged < 64 years old and 65 to 74 years old, but the risk was

29 increased among older age groups (Cakmak et al., 2011). A study performed in China reported

30 greater effects in populations ≥45 years old (compared to 5 to 44 year olds), with statistically

31 significant effects present only among those ≥65 years old (Kan et al., 2008). An Italian study

32 reported higher risk of all-cause mortality associated with increased O_3 concentrations among

33 individuals ≥85 year old as compared to those 35 to 84 years old (Stafoggia et al., 2010). The Air

34 Pollution and Health: A European and North American Approach (APHENA) project examined

35 the association between O_3 exposure and mortality for those <75 and ≥ 75 years of age. In

1   Canada, the associations for all-cause and cardiovascular mortality were greater among those
2   ≥75 years old. In the U.S., the association for all-cause mortality was slightly greater for those
3   <75 years of age compared to those ≥75 years old in summer-only analyses. No consistent
4   pattern was observed for CVD mortality. In Europe, slightly larger associations for all-cause
5   mortality were observed in those <75 years old in all-year and summer-only analyses. Larger
6   associations were reported among those <75years for CVD mortality in all-year analyses, but the
7   reverse was true for summer-only analyses (Katsouyanni et al., 2009).

8   With respect to epidemiologic studies of O_3 exposure and hospital admissions, a positive
9   association was reported between short-term O_3 exposure and respiratory hospital admissions for
10   adults ≥65 years old but not for those adults aged 15 to 64 years (Halonen et al., 2009). In the
11   same study, no association was observed between O_3 concentration and respiratory mortality
12   among those ≥65 years old or those 15 to 64 years old. No modification by age (40 to 64 year
13   olds versus >64 year olds) was observed in a study from Brazil examining O_3 levels and COPD
14   ED visits.

15   Although some outcomes reported mixed findings regarding an increase in risk for older
16   adults, recent epidemiologic studies report consistent positive associations between short-term
17   O_3 exposure and mortality in older adults. The evidence from mortality studies is consistent with
18   the results reported in the 2006 O_3 AQCD and is supported by toxicological studies providing
19   biological plausibility for increased risk of effects in older adults. Also, older adults may be
20   experiencing increased exposure compared to younger adults. Overall, the ISA (U.S. EPA 2013)
21   concludes adequate evidence is available indicating that older adults are at increased risk of
22   O_3-related health effects.

23   **People with Diets Lower in Vitamins C and E**

24   Diet was not examined as a factor potentially affecting risk in previous O_3 AQCDs, but
25   recent studies have examined modification of the association between O_3 and health effects by
26   dietary factors. Because O_3 mediates some of its toxic effects through oxidative stress, the
27   antioxidant status of an individual is an important factor that may contribute to increased risk of
28   O_3-related health effects. Supplementation with vitamins C and E has been investigated in a
29   number of studies as a means of inhibiting O_3-mediated damage.

30   Two epidemiologic studies have examined effect measure modification by diet and found
31   evidence that certain dietary components are related to the effect O_3 has on respiratory outcomes.
32   In one recent study the effects of fruit/vegetable intake and Mediterranean diet were examined.
33   Increases in these food patterns, which have been noted for their high vitamins C and E and
34   omega-3 fatty acid content, were positively related to lung function in asthmatic children living

1   in Mexico City, and modified by O_3 exposure (Romieu et al., 2009). Another study examined
2   supplementation of the diets of asthmatic children in Mexico with vitamins C and E (Sienra-
3   Monge et al., 2004). Associations were detected between short-term O_3 exposure and nasal
4   airway inflammation among children in the placebo group but not in those receiving the
5   supplementation.

6   The epidemiologic evidence is supported by controlled human exposure studies,
7   discussed in section 8.4.1 of the ISA (U.S. EPA 2013), that have shown that the first line of
8   defense against oxidative stress is antioxidants-rich extracellular lining fluid (ELF) which
9   scavenge free radicals and limit lipid peroxidation. Exposure to O_3 depletes antioxidant levels in
10 nasal ELF probably due to scrubbing of O_3; however, the concentration and the activity of
11 antioxidant enzymes either in ELF or plasma do not appear to be related to O_3 responsiveness.
12 Controlled studies of dietary antioxidant supplementation have demonstrated some protective
13 effects of α-tocopherol (a form of vitamin E) and ascorbate (vitamin C) on spirometric measures
14 of lung function after O_3 exposure but not on the intensity of subjective symptoms and
15 inflammatory responses. Dietary antioxidants have also afforded partial protection to asthmatics
16 by attenuating postexposure bronchial hyperresponsiveness. Toxicological studies discussed in
17 section 8.4.1 of the ISA (U.S. EPA 2013) provide evidence of biological plausibility to the
18 epidemiologic and controlled human exposure studies.

19   There is adequate evidence that individuals with diets lower in vitamins C and E are at
20 risk for O_3-related health effects. The evidence from epidemiologic studies is supported by
21 controlled human exposure and toxicological studies.

22 **Outdoor Workers**

23   Studies included in the 2006 O_3 AQCD reported that individuals who participate in
24 outdoor activities or work outside to be a population at increased risk based on consistently
25 reported associations between O_3 exposure and respiratory health outcomes in these groups (U.S.
26 EPA, 2006b). Outdoor workers are exposed to ambient O_3 concentrations for a greater period of
27 time than individuals who spend their days indoors. As discussed in Section 4.7 of the ISA (U.S.
28 EPA, 2013) outdoor workers sampled during the work shift had a higher ratio of personal
29 exposure to fixed-site monitor concentrations than health clinic workers who spent most of their
30 time indoors. Additionally, an increase in dose to the lower airways is possible during outdoor
31 exercise due to both increases in the amount of air breathed (i.e., minute ventilation) and a shift
32 from nasal to oronasal breathing. The association between FEV_1 responses to O_3 exposure and
33 minute ventilation is discussed more fully in Section 6.2.3.1 of the 2006 O_3 AQCD.

1   Previous studies have shown that increased exposure to O_3 due to outdoor work leads to

2   increased risk of O_3-related health effects, specifically decrements in lung function (U.S. EPA,

3   2006b). The strong evidence from the 2006 O_3 AQCD which demonstrated increased exposure,

4   dose, and ultimately risk of O_3-related health effects in this population supports the conclusion

5   that there is adequate evidence to indicate that increased exposure to O_3 through outdoor work

6   increases the risk of O_3-related health effects.

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APPENDIX 3-D: AMBIENT O_3 CONCENTRATIONS IN LOCATIONS OF HEALTH STUDIES

Annual 4th highest daily maximum O_3 concentrations for all U.S. monitors operating during the 1975 – 2010 period were retrieved from EPA's AQS database. These data were used to calculate O_3 design values for the 2008 8-hour O_3 NAAQS of 0.075 parts per million (ppm) according to 40 CFR part 50, Appendix P. Design values were calculated for each O_3 monitor and each 3-year period between 1975-1977 and 2008-2010 whenever sufficient data were available.

Ozone Design Values in Study Locations

Ozone monitors were matched to 200 health study locations on a case-by-case basis, using the following guidelines:

1) Areas defined by a Metropolitan Statistical Area (MSA) were matched with O_3 monitors by incorporating all of the monitors located in within the MSA boundaries.

2) Areas not represented by a MSA were matched to monitors by incorporating all of the monitors in the county central to location of the health study area.

3) In some cases, EPA staff made judgment calls. For example, EPA staff matched the Los Angeles, CA study area to the Los Angeles-Long Beach-Santa Ana, CA MSA defined by Los Angeles County, CA and Orange County, CA, while the Long Beach, CA study area was matched to Los Angeles County, CA and the Santa Ana, CA study area was matched to Orange County, CA.

In some cases, EPA staff matched two or more study areas to the same county or MSA. In other cases, a study area was matched to a MSA and another study area was matched to a county within the same MSA. For each 3-year period, the area design value was determined by the monitor reporting the highest design value in the county or MSA.   This has two implications for the design values:

1) Design values are sensitive to changes in the monitoring network. The addition or discontinuation of O_3 monitors in an area may cause increases or decreases in the design value trend.

2) Only valid design values are reported. According to 40 CFR Part 50, Appendix P, design values greater than the level of the NAAQS (0.075 ppm) are always valid, while design values less than or equal to 0.075 ppm must have 75% annual data completeness in order to be valid. This may cause anomalies in the design value trend. For example, a monitor may report a valid design value based on as few as 12 days of data, or a monitor with less than 75% annual data completeness may have valid design values in some 3-year periods and invalid design values in others.

36 We have identified design values for the U.S. O$_3$ epidemiologic studies identified in Sections
37 3.1.4.2 and 3.1.4.3 of the second draft Policy Assessment (see Tables 3D-1 and 3D-2,
38 respectively). For each study, design values were identified for the cities evaluated and for the
39 years over which the study was conducted. These design values are reported in tables A-1 to A-
40 22 of the Wells et al, 2012 memo "Analysis of Recent U.S. Ozone Air Quality Data to Support
41 the O$_3$ NAAQS Review and Quadratic Rollback Simulations to Support the First Draft of the
42 Risk and Exposure Assessment".

43 **Table 3D-1   Number of Study Cities from Epidemiologic Studies Using Short-Term O$_3$**
44 **Metrics with 3-Year Averages of Annual 4th Highest Daily Maximum 8-hour**
45 **O$_3$ Concentrations > 75, 70, 65, or 60 ppb[1]**

Study	Number of Cities	Study Period	Number (Percent) of Cities >75	Number (Percent) of Cities >70	Number (Percent) of Cities >65	Number (Percent) of Cities >60
Bell et al. 2004[2]	95	1987-2000	89 (94%)	91 (96%)	93 (98%)	94 (99%)
Bell et al. 2006, 2007, 2008	98	1987-2000	92 (94%)	94 (96%)	96 (98%)	97 (99%)
Cakmak al. 2006	10	1993-2000	3 (30%)	3 (30%)	4 (40%)	8 (80%)
Dales et al. 2006	11	1986-2000	4 (36%)	6 (55%)	7 (64%)	10 (91%)
Franklin & Schwartz, 2008	18	2000-2005	17 (94%)	18 (100%)	18 (100%)	18 (100%)
Katsouyanni et al. 2009[3]	89[4]	1987-1996	83 (93%)	84 (94%)	86 (97%)	88 (99%)
Katsouyanni et al. 2009[5]	14	1987-1996	12 (86%)	12 (86%)	14 (100%)	14 (100%)
Katsouyanni et al. 2009[6]	12	1987-1996	4 (33%)	6 (50%)	7 (58%)	11 (92%)
Medina-Ramon et al. 2006	26[7]	1986-1999	24 (92%)	24 (92%)	26 (100%)	26 (100%)
Schwartz, 2005	14	1986-1993	13 (93%)	13 (93%)	14 (100%)	14 (100%)
Stieb et al. 2009	7	1992-2003	2 (29%)	2 (29%)	3 (43%)	4 (57%)
Zanobetti & Schwartz, 2008a,b	48	1989-2000	44 (92%)	45 (94%)	47 (98%)	47 (98%)

[1] For U.S. study areas, we used EPA's Air Quality System (AQS) (http://www.epa.gov/ttn/airs/airsaqs/) to identify 8-hour O$_3$ concentrations. For Canadian study areas, we used publically available air quality data from the Environment Canada National Air Pollution Surveillance Network (http://www.etc-cte.ec.gc.ca/napsdata/main/aspx). We followed the data handling protocols for calculating design values as detailed in 40 CFR Part 50, Appendix P.

[2] We also evaluated the bayes-adjusted effect estimates for individual cities presented by Bell et al. (2004). None of the cities for which individual city effect estimates were statistically significant would have met the current standard over the study period.

[3] U.S. cities; examining mortality

[4] Study authors included 90 cities in their analyses; air quality data that met completeness criteria described above were available for 89 cities

[5] U.S. cities; examining morbidity

[6] Canadian cities; examining mortality and morbidity

[7] Study authors included 27 cities in their analyses; air quality data that met completeness criteria described above were available for 26 cities

Table 3D-2   Number of Study Cities from Epidemiologic Studies Using Long-Term O$_3$ Metrics with 3-Year Averages of Annual 4th Highest Daily Maximum 8-hour O$_3$ Concentrations > 75, 70, 65, or 60 ppb

Study	Number of Cities	Study Period	Number (Percent) of Cities with Maximum conc >75	Number (Percent) of Cities with Maximum conc >70	Number (Percent) of Cities with Maximum conc >65	Number (Percent) of Cities with Maximum conc >60
Islam et al. 2008, 2009	11[8]	1994-2003	11 (100%)	11 (100%)	11 (100%)	11 (100%)
Jerrett et al. 2009	94[9]	1977-2000	91 (97%)	92 (98%)	93 (99%)	94 (100%)
Lin et al. 2008	26[10]	1991-2001	24 (92%)	24 (92%)	26 (100%)	26 (100%)
Meng et al. 2010	7	1997-2002	7 (100%)	7 (100%)	7 (100%)	7 (100%)
Moore et al. 2008	8	1980-2000	8 (100%)	8 (100%)	8 (100%)	8 (100%)
Salam et al. 2009	11[11]	1992-2005	12 (100%)	12 (100%)	12 (100%)	12 (100%)
Zanobetti & Schwartz 2011	105	1985-2006	100 (95%)	104 (99%)	104 (99%)	104 (99%)

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[8] Study authors included 12 cities in their analyses, air quality data that met completeness criteria described above were available for 11 cities

[9] Study authors included 96 cities in their analyses, air quality data that met completeness criteria described above were available for 94 cities

[10] Study authors included 27 cities in their analyses, air quality data that met completeness criteria described above were available for 26 cities

[11] Study authors included 12 cities in their analyses, air quality data that met completeness criteria described above were available for 11 cities

Relationship between average 24-hour and highest 8-hour O$_3$ concentrations for cities analyzed by Bell et al. (2006)

Bell et al. (2006) reported associations between mortality and 24-hour average O$_3$ concentrations (i.e., averaged across monitors in cities with multiple monitors) in a multi-city study of 98 U.S. cities. Positive associations persisted in a series of analyses that restricted O$_3$ concentrations to those below various cut points (cut points ranged from 5 to 60 ppb in 5 ppb increments). To facilitate consideration of these cut point analyses for the second draft of the O$_3$ Policy Assessment, so as to match the form and averaging time of the existing primary standard, we evaluated the relationship between 24-hour average O$_3$ concentrations, averaged across monitors in cities with multiple monitors, and the highest 8-hour daily maximum O$_3$ concentrations among the individual monitors in each city.

EPA staff retrieved daily 24-hour average and 8-hour maximum O$_3$ concentrations reported to EPA by monitors in the 98 study areas defined in Bell et al. (2006) during the 1987-2012 period from EPA's Air Quality System (AQS) database. Next, EPA staff obtained the study area boundaries from the published study (Bell et al., 2006) and used them to determine which O$_3$ monitoring sites were associated with each study area. The 24-hour average O$_3$ concentrations were averaged spatially across all available monitors within each study area on each day where monitoring data were collected. Next, days where the area-wide 24-hour average concentration (i.e., averaged spatially across monitors in areas with multiple monitors) was greater than 60 ppb were removed from the data. Based on the data remaining (i.e., with 24-hour average concentrations of 60 ppb or below), the annual 4th highest 8-hour daily maximum concentrations were identified for each study area and for each year from 1987-2012 (Table 3D-3). This process was repeated by further removing days with area-wide 24-hour average concentrations greater than 55 ppb, 50 ppb, etc., down to 5 ppb, and re-calculating the same statistics after each removal. The resulting dataset consisted of the annual 4th highest 8-hour daily maximum concentrations for all study areas.

78 **Table 3D-3   Number of Study Cities with 4th Highest 8-hour Daily Maximum**
79 **Concentrations Greater Than the Level of the Current Standard and**
80 **Potential Alternative Standards For Various Cut-Point Analyses Presented**
81 **in Bell et al. (2006)[12]**

	Cut-point for 2-day moving average across monitors and cities (24-h avg)									
	20	25	30	35	40	45	50	55	60	All
Number (%) of Cities with 4th highest >75 (any year; 1987-2000)	0 (0%)	0 (0%)	12 (12%)	52 (53%)	77 (79%)	88 (90%)	93 (95%)	94 (96%)	94 (96%)	94 (96%)
Number (%) of Cities with 4th highest >70 (any year; 1987-2000)	0 (0%)	3 (3%)	31 (32%)	77 (79%)	86 (88%)	93 (95%)	94 (96%)	94 (96%)	95 (97%)	95 (97%)
Number (%) of Cities with 4th highest >65 (any year; 1987-2000)	0 (0%)	10 (10%)	58 (59%)	84 (86%)	93 (95%)	94 (96%)	94 (96%)	94 (96%)	94 (96%)	94 (96%)
Number (%) of Cities with 4th highest >60 (any year; 1987-2000)	1 (1%)	36 (37%)	74 (76%)	93 (95%)	96 (8%)	97 (99%)	97 (99%)	97 (99%)	97 (99%)	97 (99%)

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[12] Study authors included 98 cities in their analyses, air quality data only available for 95

84   **Relationship between average and highest 8-hour daily maximum O_3 concentrations for**
85   **New York City, as analyzed by Silverman and Ito (2010)**

86   EPA staff retrieved daily maximum 8-hour O_3 concentrations for the 13 monitors in the
87   New York City area used in the Silverman and Ito (2010) study for April-August of 1999-2006
88   from the AQS database. Next, EPA staff spatially averaged these concentrations across monitors
89   for each day during this period, and then paired them with the highest 8-hour daily maximum
90   value reported across the 13 monitors on each day.

91   Next, the range of observed average daily maximum 8-hour concentrations was broken
92   into 5 ppb increments. The number of days where the area-wide average daily maximum 8-hour
93   concentration fell within the increment and the number of days where one or more monitored 8-
94   hour daily maximum values were greater than 75, 70, 65 and 60 ppb were recorded for each 5
95   ppb increment. These numbers are summarized in Table 3D-4.

96   **Table 3D-4   Summary statistics for Observed O_3 Concentrations in the New York City**
97   **Area, April – August 1999 – 2006**

	2-day moving average across monitors (ppb)								
	11 to 20 (62 days)	21 to 25 (92 days)	26 to 30 (178 days)	31 to 35 (206 days)	36 to 40 (236 days)	41 to 45 (196 days)	46 to 50 (153 days)	51 to 55 (111 days)	56 to 60 (71 days)
Days > 75 ppb	0	0	1	0	1	2	9	15	20
Days > 70 ppb	0	0	1	4	1	12	17	23	30
Days > 65 ppb	0	0	1	6	5	18	37	42	45
Days > 60 ppb	0	0	2	7	12	39	67	61	53

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Relationship between average and highest 8-hour daily maximum O$_3$ concentrations for Atlanta, as analyzed by Strickland et al. (2010)

For our assessment of the Strickland et al. (2010) study, based in the Atlanta metropolitan area, we retrieved 8-hour daily maximum concentration data for 4 of the 5 monitors used in the study during the study period (May-October, 1993-2004) from the AQS database. The 5th monitor was a part of the Southeastern Aerosol Research and Characterization (SEARCH) network, which does not report data to EPA. EPA staff calculated the area-wide average of the 8-hour daily maximum concentrations for each day, and compared to population-weighted average concentrations obtained from the author. The correlation between the arithmetic average values and the population-weighted average values was very high (R = 0.985), thus EPA staff deemed the arithmetic average to be a suitable surrogate for the population-weighted average used in the study. Finally, 3-day moving averages were calculated from the daily area-wide average values (matching the air quality metric used in the study), and paired with the highest monitored 8-hour daily maximum value occurring during each 3-day period.

Next, the range of observed average daily maximum 8-hour concentrations was broken into 5 ppb increments. The number of days where the area-wide average daily maximum 8-hour concentration fell within the increment and the number of days where one or more monitored 8-hour daily maximum values were greater than 75, 70, 65 and 60 ppb were recorded for each 5 ppb increment. These numbers are summarized in Table 3D-5.

Table 3D-5   Summary statistics for Observed O$_3$ Concentrations in the Atlanta Area, April – August 1999 – 2006

	3-day moving average across monitors (ppb)										
	26-30 (75 days)	31-35 (144 days)	36-40 (165 days)	41-45 (210 days)	46-50 (235 days)	51-55 (244 days)	56-60 (272 days)	61-65 (234 days)	66-70 (169 days)	71 to 75 (124 days)	76 to 80 (106 days)
Days > 75	0	0	2	2	10	24	53	80	89	87	87
Days > 70	0	0	6	6	20	49	81	111	107	96	95
Days > 65	1	0	8	19	38	75	118	147	133	106	100
Days > 60	1	2	15	33	68	115	152	173	147	116	102

123   **Relationship between annual and highest 1-hour daily maximum O_3 concentrations for 12**
124   **study areas, as analyzed by Jerrett et al. (2009)**

125   The Jerrett et al. (2009) study used a long-term metric based on seasonal averages of 1-
126   hour daily maximum O_3 concentrations to evaluate associations between respiratory mortality
127   and long-term or repeated exposures to O_3. Authors divided study cities into quartiles based on
128   these seasonal averages of 1-hour daily O_3 concentrations. Using AQS, we identified the 3-year
129   averages of annual 4th highest daily maximum 8-hour O_3 concentrations in study cities during the
130   study period. Table 3D-6 presents the means and maximums of these concentrations over the
131   study period.

132   In addition, for the 12 urban case study areas included in the epidemiology-based risk
133   assessment of the 2nd draft of the Health REA we identified the seasonal averages of 1-hour daily
134   maximum concentrations (i.e., the O_3 metric evaluated by Jerrett et al., 2009) for air quality
135   adjusted to the current and alternative standards. Specifically, for adjusted air quality "quarterly"
136   averages of 1-hour concentrations for April-June and July-August were calculated for each area
137   and year. The quarterly values were considered to be valid if valid daily maximum 1-hour
138   values were available for at least 75% of the days in the quarter. The two quarterly values were
139   then averaged, as was done by Jerrett et al. (2009) to generate the long-term metric used in the
140   study. This process was repeated for the various model-based adjustment scenarios in each of
141   the 12 study areas. Summary statistics based on this seasonal average of daily O_3 concentrations
142   are presented in Table 3D-7 for recent air quality and for air quality adjusted to just meet the
143   current and alternative standards.

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Table 3D-6   Three-Year Averages of Annual 4th Highest Daily Maximum 8-hour O$_3$ Concentrations in 94[13] Study Areas Examined in Jerrett et al. (2009)

	City	Mean over study period	Max over study period
Cities in the lowest quartile of average exposure[14]	Charleston, WV	81	99
	Chicago, IL	103	114
	Colorado Springs, CO	62	66
	Corpus Christi, TX	82	89
	Detroit, MI	95	103
	Flint, MI	83	91
	Ft. Lauderdale, FL	74	79
	Kansas City, MO	87	97
	Lansing, MI	81	90
	Madison, WI	82	102
	Minneapolis, MN	74	80
	New Orleans, LA	86	99
	Orlando, FL	79	82
	Portland, OR	81	91
	Providence, RI	110	124
	Salinas, CA	68	74
	San Antonio, TX	85	92
	San Francisco, CA	88	96
	San Jose, CA	91	103
	Seattle, WA	78	88
	Tacoma, WA	78	88
	Vallejo, CA	74	82
	Wichita, KS	75	81
Cities in the highest three quartiles of average exposure[15]	Charleston, SC	79	90
	Charlotte, NC	97	112
	Chattanooga, TN	90	97
	Cincinnati, OH	101	119
	Cleveland, OH	98	108
	Columbia, SC	85	109
	Columbus, OH	93	103
	Dallas/Ft Worth, TX	106	118
	Dayton, OH	95	122
	Denver, CO	83	91

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[13] Jerrett et al. (2009) examined 96 MSAs; this analysis included the 94 cities that met data completeness criteria described above, after linking monitors to MSAs (see lines 10-28, above).
[14] Based on visual inspection of Figure 1 in Jerrett et al. (2009)
[15] Based on visual inspection of Figure 1 in Jerrett et al. (2009)

	El Paso, TX	85	96
	Evansville, IN	93	100
	Fresno, CA	112	123
	Gary, IN	91	105
	Greely, CO	69	75
	Greensboro, NC	89	100
	Greenville, SC	86	94
	Harrisburg, PA	94	103
	Houston, TX	121	140
	Huntington, WV	94	103
	Indianapolis, IN	93	103
	Jackson, MS	79	98
	Jacksonville, FL	81	87
	Jersey City, NJ	106	118
	Johnstown, PA	90	107
	Kenosha, WI	101	114
	Knoxville, TN	91	97
	Lancaster, PA	94	101
	Las Vegas, NV	80	85
	Lexington, KY	88	99
	Little Rock, AR	86	107
	Los Angeles, CA	193	248
	Memphis, TN	94	103
	Milwaukee, WI	103	117
	Nashville, TN	94	106
	Nassau, NY	NA[16]	NA
	New Haven, CT	116	136
	New York City, NY	118	129
	Newark, NJ	90	105
	Norfolk, VA	91	101
	Oklahoma City, OK	86	93
	Philadelphia, PA	117	136
	Phoenix, AZ	86	96
	Pittsburgh, PA	101	123
	Portland, ME	106	117
	Portsmouth, NH	92	104
	Racine, WI	102	124
	Raleigh, NC	90	104
	Reading, PA	99	114
	Richmond, VA	94	104
	Riverside, CA	196	245
	Roanoke, VA	83	95

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[16] Air quality data did not meet completeness criteria described above

	Rochester, NY	89	99
	Sacramento, CA	110	118
	San Diego, CA	121	141
	Shreveport, LA	83	88
	South Bend, IN	90	102
	Springfield, MA	102	115
	St Louis, MO	105	122
	Steubenville, OH	82	99
	Syracuse, NY	85	96
	Tampa, FL	85	91
	Toledo, OH	93	108
	Trenton, NJ	112	124
	Tucson, AZ	76	82
	Ventura, CA	118	132
	Washington, DC	105	116
	Wilmington, DE	103	116
	Worcester, MA	92	102
	York, PA	95	107
	Youngstown, OH	93	103

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Table 3D-7   **Long-Term O₃ Concentrations in 12 Urban Case Study Areas (Using the O₃ Metric Evaluated by Jerrett et al., 2009) for Recent Air Quality and Air Quality Adjusted to Meet Standard Levels of 75, 70, 65, and 60 ppb**

	Air Quality Adjusted to:	2006 (Adj Yrs 2006-2008)	2007 (Adj Yrs 2006-2008)	2008 (Adj Yrs 2008-2010)	2009 (Adj Yrs 2008-2010)	2010 (Adj Yrs 2008-2010)
Atlanta	Recent	65	63	57	50	56
	75	53	52	53	47	52
	70	50	49	49	44	49
	65	47	46	46	42	46
	60	45	44	44	40	44
Baltimore	Recent	60	59	57	52	60
	75	54	54	53	49	55
	70	52	51	51	48	53
	65	49	49	48	46	50
	60	46	46	46	44	48
Boston	Recent	49	50	46	45	49
	75	48	49	49	45	48
	70	46	47	48	44	48
	65	44	45	46	43	46
	60	43	43	44	41	44
Cleveland	Recent	51	52	53	49	54
	75	49	50	51	47	51
	70	47	48	48	45	48
	65	45	45	45	43	45
	60	41	41	41	40	42
Denver	Recent	63	63	63	58	60
	75	62	61	63	58	60
	70	60	59	62	58	58
	65	58	58	59	56	55
	60	53	53	53	51	50
Detroit	Recent	50	54	51	48	52
	75	50	52	N/A	N/A	N/A
	70	48	50	51	49	52
	65	47	49	49	47	50
	60	45	46	46	45	47
Houston	Recent	53	48	47	47	46
	75	48	46	47	48	46
	70	47	45	46	47	46
	65	46	44	45	46	45
	60	45	43	43	44	44
Los Angeles	Recent	65	61	64	62	57
	75	58	59	60	60	58
	70	55	56	57	58	56
	65	52	53	54	54	53
	60	N/A	N/A	N/A	N/A	N/A
New York City	Recent	53	54	55	48	55
	75	47	47	51	47	51
	70	N/A	N/A	N/A	N/A	N/A
	65	N/A	N/A	N/A	N/A	N/A
	60	N/A	N/A	N/A	N/A	N/A
Philadelphia	Recent	56	59	57	51	58
	75	51	52	54	49	54
	70	49	50	51	47	52
	65	47	48	49	45	49
	60	45	46	47	43	47
Sacramento	Recent	66	59	65	61	55
	75	55	50	54	51	48
	70	52	48	51	49	46
	65	50	46	49	47	44
	60	47	44	46	44	42
Saint Louis	Recent	58	58	52	51	55
	75	53	53	51	50	54
	70	50	51	50	48	52
	65	47	48	48	46	49
	60	44	45	45	43	46

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Appendix 5A

Ozone-Sensitive Plant Species[A] Used by Some Tribes*		
(Based on Feedback from 3 Tribes)		
Common Name (other common names)	**Scientific Name**	**Confirmed bioindicator species**
Red alder (Oregon alder, Western alder)	*Alnus rubra*	Y
Speckled alder (Tag alder, Gray alder, Hoary alder)	*Alnus rugosa (Alnus incana)*	Y
Groundnut (Wild bean, American potato bean)	*Apios americana*	Y
Spreading Dogbane (Common dogbane)	*Apocynum androsamifolium*	Y
Common milkweed	*Asclepias syriaca*	Y
New England Aster	*Aster novae-angliae Symphyotrichum novae-angliae*	
Green ash	*Fraxinus pennsylvanica*	
Twinberry	*Lonicera involucrate*	Y
Bee-balm	*Monarda didyma*	
Virginia creeper	*Parthenocissus quinquefolia*	Y
Jack pine	*Pinus banksiana*	Y
Lodgepole pine	*Pinus contorta*	
White pine	*Pinus strobus*	
Black poplar (Balsam poplar)	*Populus balsamifera trichocarpa*	
Quaking aspen (Trembling aspen)	*Populus tremuloides*	Y
Black cherry	*Prunus serotina*	Y
Choke cherry	*Prunus virginiana*	
Douglas fir	*Pseudotsuga menziesii*	
Allegheny blackberry (Common blackberry)	*Rubus allegheniensis*	Y
Thimbleberry	*Rubus parviflorus*	Y
Cutleaf coneflower (Coneflower, Golden glow)	*Rudbeckia laciniata*	Y
Pussy willow	*Salix discolor*	
Shinning willow	*Salix lucida*	
American elder (White elder)	*Sambucus canadensis*	Y
Red elderberry	*Sambucus racemosa*	Y
Sassafras	*Sassafras albidum*	
Goldenrod	*Solidago altissima*	
Huckleberry	*Vaccinium membranaceum*	Y
Wild grape	*Vitis spp.*	
European wine grape	*Vitis vinifera*	Y
[A]Species included in this list are identified in one or more of the following sources: **1)SP 2007 (www.2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf)** **2) NPS O$_3$ Bioindicators 2006 (www.nature.nps.gov/air/Pubs/bioindicators/index.cfm)** **3) Kline et al., 2008; 4) Davis, 2007/ 2009; 5) Flagler, et al., eds., 1998** **6) USDA FS FHM/FIA: Ozone Bioindicator Sampling and Estimation (www.nrs.fs.fed.us/fia/topics/ozone/pubs/pdfs/ozone%20estimation%20document.pdf) and Ozone Injury in West Coast Forests: 6 Years of Monitoring (2007).**		

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APPENDIX 6A

Calculation of Approximate Equivalent 12-hr SUM06 and 12-hr W126

SOURCE: 2007 Staff Paper, Appendix 7B (U.S. EPA, 2007).

Despite various metrics reported in the vegetation effects literature, there is no standard method for calculating equivalent levels between metrics. The maximum 3-month 12-hr SUM06 of 25 ppm-hr secondary standard that was proposed in the last review (62 FR 38877) was based on a yield loss prevention of approximately 10% in 50% of crop cases studied in the National Crop Loss Analysis Network (NCLAN) experiments. For consistency, staff judged it appropriate to use the NCLAN experiments to derive equivalents between the 12-hr SUM06 and W126. For example, below are the 12-hr SUM06 and W126 NCLAN equations to protect 50% of crop cases from a specified percent yield loss (Lee and Hogsett 1996):

Metric	Weibull Equation
12-hr SUM06	Predicted Relative Yield Loss = 1- exp(-[SUM06/87.42]^1.82)
12-hr W126	Predicted Relative Yield Loss = 1- exp(-[W126/96.05]^1.48)

In the first equation, solving for a SUM06 of 25 ppm-hr equals a predicted relative yield loss of 10%. Solving the second equation for a 10% yield loss equals a W126 of 21 ppmhr. Thus, staff considers a 12-hr SUM06 of 25 ppm-hr and a 12-hr W126 of 21 ppm-hr approximately equivalent.

REFERENCES

Lee, E. H.; Hogsett, W. E. (1996) Methodology for calculating inputs for ozone secondary standard benefits analysis: part II. Report prepared for Office of Air Quality Planning and Standards, Air Quality Strategies and Standards Division, U.S. Environmental Protection Agency, Research Triangle Park, N.C., March.

U.S. EPA (U.S. Environmental Protection Agency). (2007). Review of the national ambient air quality standards for ozone: Policy assessment of scientific and technical information: OAQPS staff paper [EPA Report]. (EPA/452/R-07/003). Research Triangle Park, NC. http://www.epa.gov/ttn/naaqs/standards/ozone/data/2007_01_ozone_staff_paper