

ENVIRONMENTAL PROTECTION AGENCY**40 CFR Parts 50 and 58**

[EPA-HQ-OAR-2005-0172; FRL-9102-1]

RIN 2060-AP98

National Ambient Air Quality Standards for Ozone**AGENCY:** Environmental Protection Agency (EPA).**ACTION:** Proposed rule.

SUMMARY: Based on its reconsideration of the primary and secondary national ambient air quality standards (NAAQS) for ozone (O₃) set in March 2008, EPA proposes to set different primary and secondary standards than those set in 2008 to provide requisite protection of public health and welfare, respectively. With regard to the primary standard for O₃, EPA proposes that the level of the 8-hour primary standard, which was set at 0.075 ppm in the 2008 final rule, should instead be set at a lower level within the range of 0.060 to 0.070 parts per million (ppm), to provide increased protection for children and other “at risk” populations against an array of O₃-related adverse health effects that range from decreased lung function and increased respiratory symptoms to serious indicators of respiratory morbidity including emergency department visits and hospital admissions for respiratory causes, and possibly cardiovascular-related morbidity as well as total non-accidental and cardiopulmonary mortality. With regard to the secondary standard for O₃, EPA proposes that the secondary O₃ standard, which was set identical to the revised primary standard in the 2008 final rule, should instead be a new cumulative, seasonal standard expressed as an annual index of the sum of weighted hourly concentrations, cumulated over 12 hours per day (8 am to 8 pm) during the consecutive 3-month period within the O₃ season with the maximum index value, set at a level within the range of 7 to 15 ppm-hours, to provide increased protection against O₃-related adverse impacts on vegetation and forested ecosystems.

DATES: Written comments on this proposed rule must be received by March 22, 2010.

Public Hearings: Three public hearings are scheduled for this proposed rule. Two of the public hearings will be held on February 2, 2010 in Arlington, Virginia, and Houston, Texas. The third public hearing will be held on February 4, 2010 in Sacramento, California.

ADDRESSES: Submit your comments, identified by Docket ID No. EPA-HQ-OAR-2005-0172, by one of the following methods:

- <http://www.regulations.gov>: Follow the on-line instructions for submitting comments.

- *E-mail:* a-and-r-Docket@epa.gov.

- *Fax:* 202-566-9744.

- *Mail:* Docket No. EPA-HQ-OAR-2005-0172, Environmental Protection Agency, Mail code 6102T, 1200 Pennsylvania Ave., NW., Washington, DC 20460. Please include a total of two copies.

- *Hand Delivery:* Docket No. EPA-HQ-OAR-2005-0172, Environmental Protection Agency, EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC. Such deliveries are only accepted during the Docket’s normal hours of operation, and special arrangements should be made for deliveries of boxed information.

Public Hearings: Three public hearings are scheduled for this proposed rule. Two of the public hearings will be held on February 2, 2010 in Arlington, Virginia and Houston, Texas. The third public hearing will be held on February 4, 2010 in Sacramento, California. The hearings will be held at the following locations:

Arlington, Virginia—February 2, 2010

Hyatt Regency Crystal City @ Reagan National Airport, Washington Room (located on the Ballroom Level), 2799 Jefferson Davis Highway, Arlington, Virginia 22202, Telephone: 703-418-1234.

Houston, Texas—February 2, 2010

Hilton Houston Hobby Airport, Moody Ballroom (located on the ground floor), 8181 Airport Boulevard, Houston, Texas 77061, Telephone: 713-645-3000.

Sacramento, California—February 4, 2010

Four Points by Sheraton Sacramento International Airport, Natomas Ballroom, 4900 Duckhorn Drive, Sacramento, California 95834, Telephone: 916-263-9000.

See the **SUPPLEMENTARY INFORMATION** under “Public Hearings” for further information.

Instructions: Direct your comments to Docket ID No. EPA-HQ-OAR-2005-0172. The EPA’s policy is that all comments received will be included in the public docket without change and may be made available online at www.regulations.gov, including any personal information provided, unless the comment includes information claimed to be Confidential Business

Information (CBI) or other information whose disclosure is restricted by statute. Do not submit information that you consider to be CBI or otherwise protected through www.regulations.gov or e-mail. The www.regulations.gov Web site is an “anonymous access” system, which means EPA will not know your identity or contact information unless you provide it in the body of your comment. If you send an e-mail comment directly to EPA without going through www.regulations.gov, your e-mail address will be automatically captured and included as part of the comment that is placed in the public docket and made available on the Internet. If you submit an electronic comment, EPA recommends that you include your name and other contact information in the body of your comment and with any disk or CD-ROM you submit. If EPA cannot read your comment due to technical difficulties and cannot contact you for clarification, EPA may not be able to consider your comment. Electronic files should avoid the use of special characters, any form of encryption, and be free of any defects or viruses.

Docket: All documents in the docket are listed in the www.regulations.gov index. Although listed in the index, some information is not publicly available, e.g., CBI or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, will be publicly available only in hard copy. Publicly available docket materials are available either electronically in www.regulations.gov or in hard copy at the Air and Radiation Docket and Information Center, EPA/DC, EPA West, Room 3334, 1301 Constitution Ave., NW., Washington, DC. The Public Reading Room is open from 8:30 a.m. to 4:30 p.m., Monday through Friday, excluding legal holidays. The telephone number for the Public Reading Room is (202) 566-1744 and the telephone number for the Air and Radiation Docket and Information Center is (202) 566-1742.

FOR FURTHER INFORMATION CONTACT: Ms. Susan Lyon Stone, Health and Environmental Impacts Division, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Mail Code C504-06, Research Triangle Park, NC 27711; telephone: 919-541-1146; fax: 919-541-0237; e-mail: stone.susan@epa.gov.

SUPPLEMENTARY INFORMATION:

General Information

What Should I Consider as I Prepare My Comments for EPA?

1. *Submitting CBI.* Do not submit this information to EPA through <http://www.regulations.gov> or e-mail. Clearly mark the part or all of the information that you claim to be CBI. For CBI information in a disk or CD-ROM that you mail to EPA, mark the outside of the disk or CD-ROM as CBI and then identify electronically within the disk or CD-ROM the specific information that is claimed as CBI. In addition to one complete version of the comment that includes information claimed as CBI, a copy of the comment that does not contain the information claimed as CBI must be submitted for inclusion in the public docket. Information so marked will not be disclosed except in accordance with procedures set forth in 40 CFR part 2.

2. *Tips for Preparing Your Comments.* When submitting comments, remember to:

- Identify the rulemaking by docket number and other identifying information (subject heading, **Federal Register** date and page number).
- Follow directions—The Agency may ask you to respond to specific questions or organize comments by referencing a Code of Federal Regulations (CFR) part or section number.
- Explain why you agree or disagree, suggest alternatives, and substitute language for your requested changes.
- Describe any assumptions and provide any technical information and/or data that you used.
- Provide specific examples to illustrate your concerns, and suggest alternatives.
- Explain your views as clearly as possible, avoiding the use of profanity or personal threats.
- Make sure to submit your comments by the comment period deadline identified.

Availability of Related Information

A number of documents relevant to this rulemaking are available on EPA web sites. The Air Quality Criteria for Ozone and Related Photochemical Oxidants (2006 Criteria Document) (two volumes, EPA/and EPA/, date) is available on EPA's National Center for Environmental Assessment Web site. To obtain this document, go to <http://www.epa.gov/ncea>, and click on Ozone in the Quick Finder section. This will open a page with a link to the March 2006 Air Quality Criteria Document. The 2007 Staff Paper, human exposure and health risk assessments, vegetation

exposure and impact assessment, and other related technical documents are available on EPA's Office of Air Quality Planning and Standards (OAQPS) Technology Transfer Network (TTN) web site. The updated final 2007 Staff Paper is available at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html and the exposure and risk assessments and other related technical documents are available at http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html. The Response to Significant Comments Document is available at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_rc.html. These and other related documents are also available for inspection and copying in the EPA docket identified above.

Public Hearings

The public hearings on February 2, 2010 and February 4, 2010 will provide interested parties the opportunity to present data, views, or arguments concerning the proposed rule. The EPA may ask clarifying questions during the oral presentations, but will not respond to the presentations at that time. Written statements and supporting information submitted during the comment period will be considered with the same weight as any oral comments and supporting information presented at the public hearing. Written comments must be received by the last day of the comment period, as specified in this proposed rulemaking.

The public hearings will begin at 9:30 a.m. and continue until 7:30 p.m. (local time) or later, if necessary, depending on the number of speakers wishing to participate. The EPA will make every effort to accommodate all speakers that arrive and register before 7:30 p.m. A lunch break is scheduled from 12:30 p.m. until 2 p.m.

If you would like to present oral testimony at the hearings, please notify Ms. Tricia Crabtree (C504-02), U.S. EPA, Research Triangle Park, NC 27711. The preferred method for registering is by e-mail (crabtree.tricia@epa.gov). Ms. Crabtree may be reached by telephone at (919) 541-5688. She will arrange a general time slot for you to speak. The EPA will make every effort to follow the schedule as closely as possible on the day of the hearing.

Oral testimony will be limited to five (5) minutes for each commenter to address the proposal. We will not be providing equipment for commenters to show overhead slides or make computerized slide presentations unless we receive special requests in advance. Commenters should notify Ms. Crabtree if they will need specific audiovisual

(AV) equipment. Commenters should also notify Ms. Crabtree if they need specific translation services for non-English speaking commenters. The EPA encourages commenters to provide written versions of their oral testimonies either electronically on computer disk, CD-ROM, or in paper copy.

The hearing schedules, including lists of speakers, will be posted on EPA's Web site for the proposal at http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_fr.html prior to the hearing. Verbatim transcripts of the hearings and written statements will be included in the rulemaking docket.

Children's Environmental Health

Consideration of children's environmental health plays a central role in the reconsideration of the 2008 final decision on the O₃ NAAQS and EPA's decision to propose to set the 8-hour primary O₃ standard at a level within the range of 0.060 to 0.070 ppm. Technical information that pertains to children, including the evaluation of scientific evidence, policy considerations, and exposure and risk assessments, is discussed in all of the documents listed above in the section on the availability of related information. These documents include: the Air Quality Criteria for Ozone and Other Related Photochemical Oxidants; the 2007 Staff Paper; exposure and risk assessments and other related documents; and the Response to Significant Comments. All of these documents are available on the Web, as described above, and are in the public docket for this rulemaking. The public is invited to submit comments or identify peer-reviewed studies and data that assess effects of early life exposure to O₃.

Table of Contents

The following topics are discussed in this preamble:

- I. Background
 - A. Legislative Requirements
 - B. Related Control Requirements
 - C. Review of Air Quality Criteria and Standards for O₃
 - D. Reconsideration of the 2008 O₃ NAAQS Final Rule
 1. Decision to Initiate a Rulemaking to Reconsider
 2. Ongoing Litigation
- II. Rationale for Proposed Decision on the Level of the Primary Standard
 - A. Health Effects Information
 1. Overview of Mechanisms
 2. Nature of Effects
 3. Interpretation and Integration of Health Evidence
 4. O₃-Related Impacts on Public Health
 - B. Human Exposure and Health Risk Assessments

1. Exposure Analyses
2. Quantitative Health Risk Assessment
- C. Reconsideration of the Level of the Primary Standard
 1. Evidence and Exposure/Risk-Based Considerations
 2. CASAC Views Prior to 2008 Decision
 3. Basis for 2008 Decision on the Primary Standard
 4. CASAC Advice Following 2008 Decision
 5. Administrator's Proposed Conclusions
- D. Proposed Decision on the Level of the Primary Standard
- III. Communication of Public Health Information
- IV. Rationale for Proposed Decision on the Secondary Standard
 - A. Vegetation Effects Information
 1. Mechanisms
 2. Nature of Effects
 3. Adversity of Effects
 - B. Biologically Relevant Exposure Indices
 - C. Vegetation Exposure and Impact Assessment
 1. Exposure Characterization
 2. Assessment of Risks to Vegetation
 - D. Reconsideration of Secondary Standard
 1. Considerations Regarding 2007 Proposed Cumulative Seasonal Standard
 2. Considerations Regarding 2007 Proposed 8-Hour Standard
 3. Basis for 2008 Decision on the Secondary Standard
 4. CASAC Views Following 2008 Decision
 5. Administrator's Proposed Conclusions
 - E. Proposed Decision on the Secondary O₃ Standard
- V. Revision of Appendix P—Interpretation of the NAAQS for O₃ and Proposed Revisions to the Exceptional Events Rule
 - A. Background
 - B. Interpretation of the Secondary O₃ Standard
 - C. Clarifications Related to the Primary Standard
 - D. Revisions to Exceptions From Standard Data Completeness Requirements for the Primary Standard
 - E. Elimination of the Requirement for 90 Percent Completeness of Daily Data Across Three Years
 - F. Administrator Discretion To Use Incomplete Data
 - G. Truncation Versus Rounding
 - H. Data Selection
 - I. Exceptional Events Information Submission Schedule
- VI. Ambient Monitoring Related to Proposed O₃ Standards
 - A. Background
 - B. Urban Monitoring Requirements
 - C. Non-Urban Monitoring Requirements
 - D. Revisions to the Length of the Required O₃ Monitoring Season
- VII. Implementation of Proposed O₃ Standards
 - A. Designations
 - B. State Implementation Plans
 - C. Trans-boundary Emissions
- VIII. Statutory and Executive Order Reviews
 - A. Executive Order 12866: Regulatory Planning and Review
 - B. Paperwork Reduction Act
 - C. Regulatory Flexibility Act
 - D. Unfunded Mandates Reform Act
 - E. Executive Order 13132: Federalism

- F. Executive Order 13175: Consultation and Coordination With Indian Tribal Governments
- G. Executive Order 13045: Protection of Children From Environmental Health and Safety Risks
- H. Executive Order 13211: Actions That Significantly Affect Energy Supply, Distribution or Use
- I. National Technology Transfer and Advancement Act
- J. Executive Order 12898: Federal Actions To Address Environmental Justice in Minority Populations and Low-Income Populations

References

I. Background

The proposed decisions presented in this notice are based on a reconsideration of the 2008 O₃ NAAQS final rule (73 FR 16436, March 27, 2008), which revised the level of the 8-hour primary O₃ standard to 0.075 ppm and revised the secondary O₃ standard by making it identical to the revised primary standard. This reconsideration is based on the scientific and technical information and analyses on which the March 2008 O₃ NAAQS rulemaking was based. Therefore, much of the information included in this notice is drawn directly from information included in the 2007 proposed rule (72 FR 37818, July 11, 2007) and the 2008 final rule (73 FR 16436).

A. Legislative Requirements

Two sections of the Clean Air Act (CAA) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. 7408) directs the Administrator to identify and list “air pollutants” that in her “judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare” and satisfy two other criteria, including “whose presence * * * in the ambient air results from numerous or diverse mobile or stationary sources” and to issue air quality criteria for those that are listed. Air quality criteria are intended to “accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air. * * *

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

health.”¹ A secondary standard, as defined in section 109(b)(2), must “specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air.”²

The requirement that primary standards include an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. *Lead Industries Association v. EPA*, 647 F.2d 1130, 1154 (DC Cir 1980), *cert. denied*, 449 U.S. 1042 (1980); *American Petroleum Institute v. Costle*, 665 F.2d 1176, 1186 (DC Cir. 1981), *cert. denied*, 455 U.S. 1034 (1982). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that include an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, see *Lead Industries Association v. EPA*, 647 F.2d at 1156 n. 51, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

In addressing the requirement for an adequate margin of safety, EPA considers such factors as the nature and severity of the health effects involved, the size of the population(s) at risk, and the kind and degree of the uncertainties

¹ 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)].

² 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.”

that must be addressed. The selection of any particular approach to providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. *Lead Industries Association v. EPA*, 647 F.2d at 1161–62; *Whitman v. American Trucking Associations*, 531 U.S. 457, 495 (2001).

In setting standards that are “requisite” to protect public health and welfare, as provided in section 109(b), EPA’s task is to establish standards that are neither more nor less stringent than necessary for these purposes. *Whitman v. American Trucking Associations*, 531 U.S. 457, 473. In establishing “requisite” primary and secondary standards, EPA may not consider the costs of implementing the standards. *Id.* at 471.

Section 109(d)(1) of the CAA requires that “not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards * * * and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate. * * *” Section 109(d)(2) requires that an independent scientific review committee “shall complete a review of the criteria * * * and the national primary and secondary ambient air quality standards * * * and shall recommend to the Administrator any new * * * standards and revisions of existing criteria and standards as may be appropriate. * * *” This independent review function is performed by the Clean Air Scientific Advisory Committee (CASAC) of EPA’s Science Advisory Board.

B. Related Control Requirements

States have primary responsibility for ensuring attainment and maintenance of ambient air quality standards once EPA has established them. Under section 110 of the Act (42 U.S.C. 7410) and related provisions, States are to submit, for EPA approval, State implementation plans (SIPs) that provide for the attainment and maintenance of such standards through control programs directed to emission sources.

The majority of man-made nitrogen oxides (NO_x) and volatile organic compounds (VOC) emissions that contribute to O₃ formation in the United States come from three types of sources: Mobile sources, industrial processes (which include consumer and commercial products), and the electric

power industry.³ Mobile sources and the electric power industry were responsible for 78 percent of annual NO_x emissions in 2004. That same year, 99 percent of man-made VOC emissions came from industrial processes (including solvents) and mobile sources. Emissions from natural sources, such as trees, may also comprise a significant portion of total VOC emissions in certain regions of the country, especially during the O₃ season, which are considered natural background emissions.

The EPA has developed new emissions standards for many types of stationary sources and for nearly every class of mobile sources in the last decade to reduce O₃ by decreasing emissions of NO_x and VOC. These programs complement State and local efforts to improve O₃ air quality and meet the 0.084 ppm 8-hour national standards. Under title II of the CAA, 42 U.S.C. 7521–7574, EPA has established new emissions standards for nearly every type of automobile, truck, bus, motorcycle, earth mover, and aircraft engine, and for the fuels used to power these engines. EPA also established new standards for the smaller engines used in small watercraft, lawn and garden equipment. In March 2008, EPA promulgated new standards for locomotive and marine diesel engines and in August 2009, proposed to control emissions from ocean-going vessels.

Benefits from engine standards increase modestly each year as older, more-polluting vehicles and engines are replaced with newer, cleaner models. In time, these programs will yield substantial emission reductions. Benefits from fuel programs generally begin as soon as a new fuel is available.

The reduction of VOC emissions from industrial processes has been achieved either directly or indirectly through implementation of control technology standards, including maximum achievable control technology, reasonably available control technology, and best available control technology standards; or are anticipated due to proposed or upcoming proposals based on generally available control technology or best available controls under provisions related to consumer and commercial products. These standards have resulted in VOC emission reductions of almost a million tons per year accumulated starting in 1997 from a variety of sources including combustion sources, coating categories, and chemical manufacturing. EPA has

also finalized emission standards and fuel requirements for new stationary engines. In the area of consumer and commercial products, EPA has finalized new national VOC emission standards for aerosol coatings and is working toward amending existing rules to establish new nationwide VOC content limits for household and institutional consumer products and architectural and industrial maintenance coatings. The aerosol coatings rule took effect in July 2009; the compliance date for both the amended consumer product rule and architectural coatings rule is anticipated to be January 2011. These actions are expected to yield significant new VOC reductions—about 200,000 tons per year. Additionally, in ozone nonattainment areas, we anticipate reductions of an additional 25,000 tons per year as States adopt rules this year implementing control techniques recommendations issued in 2008 for 4 additional categories of consumer and commercial products, typically surface coatings and adhesives used in industrial manufacturing operations. These emission reductions primarily result from solvent controls and typically occur where and when the solvent is used, such as during manufacturing processes.

The power industry is one of the largest emitters of NO_x in the United States. Power industry emission sources include large electric generating units (EGU) and some large industrial boilers and turbines. The EPA’s landmark Clean Air Interstate Rule (CAIR), issued on March 10, 2005, was designed to permanently cap power industry emissions of NO_x in the eastern United States. The first phase of the cap was to begin in 2009, and a lower second phase cap was to begin in 2015. The EPA had projected that by 2015, the CAIR and other programs would reduce NO_x emissions during the O₃ season by about 50 percent and annual NO_x emissions by about 60 percent from 2003 levels in the Eastern U.S. However, on July 11, 2008 and December 23, 2008, the U.S. Court of Appeals for the DC Circuit issued decisions on petitions for review of the CAIR. In its July 11 opinion, the court found CAIR unlawful and decided to vacate CAIR and its associated Federal implementation plans (FIPs) in their entirety. On December 23, the court granted EPA’s petition for rehearing to the extent that it remanded without vacatur for EPA to conduct further proceedings consistent with the Court’s prior opinion. Under this decision, CAIR will remain in place only until replaced by EPA with a rule that is consistent with the Court’s July

³ See EPA report, *Evaluating Ozone Control Programs in the Eastern United States: Focus on the NO_x Budget Trading Program*, 2004.

11 opinion. The EPA recognizes the need in our CAIR replacement effort to address the reconsidered ozone standard, and we are currently assessing our options for the best way to accomplish this. It should also be noted that new electric generating units (EGUs) are also subject to NO_x limits under New Source Performance Standards (NSPS) under CAA section 111, as well as either nonattainment new source review or prevention of significant deterioration requirements.

With respect to agricultural sources, the U.S. Department of Agriculture (USDA) has approved conservation systems and activities that reduce agricultural emissions of NO_x and VOC. Current practices that may reduce emissions of NO_x and VOC include engine replacement programs, diesel retrofit programs, manipulation of pesticide applications including timing of applications, and animal feeding operations waste management techniques. The EPA recognizes that USDA has been working with the agricultural community to develop conservation systems and activities to control emissions of O₃ precursors.

These conservation activities are voluntarily adopted through the use of incentives provided to the agricultural producer. In cases where the States need these measures to attain the standard, the measures could be adopted. The EPA will continue to work with USDA on these activities with efforts to identify and/or improve the control efficiencies, prioritize the adoption of these conservation systems and activities, and ensure that appropriate criteria are used for identifying the most effective application of conservation systems and activities.

The EPA will work together with USDA and with States to identify appropriate measures to meet the primary and secondary standards, including site-specific conservation systems and activities. Based on prior experience identifying conservation measures and practices to meet the PM NAAQS requirements, the EPA will use a similar process to identify measures that could meet the O₃ requirements. The EPA anticipates that certain USDA-approved conservation systems and activities that reduce agricultural emissions of NO_x and VOC may be able to satisfy the requirements for applicable sources to implement reasonably available control measures for purposes of attaining the primary and secondary O₃ NAAQS.

C. Review of Air Quality Criteria and Standards for O₃

In 1971, EPA first established primary and secondary NAAQS for photochemical oxidants (36 FR 8186). Both primary and secondary standards were set at a level of 0.08 parts per million (ppm), 1-hr average, total photochemical oxidants, not to be exceeded more than one hr per year. In 1977, EPA announced the first periodic review of the air quality criteria in accordance with section 109(d)(1) of the Act. The EPA published a final decision in 1979 (44 FR 8202). Both primary and secondary standard levels were revised from 0.08 to 0.12 ppm. The indicator was revised from photochemical oxidants to O₃, and the form of the standards was revised from a deterministic to a statistical form, which defined attainment of the standards as occurring 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 one. In 1983, EPA announced that the second periodic review of the primary and secondary standards for O₃ had been initiated. Following review and publication of air quality criteria and a supplement, EPA published a proposed decision (57 FR 35542) in August 1992 that announced EPA's intention to proceed as rapidly as possible with the next review of the air quality criteria and standards for O₃ in light of emerging evidence of health effects related to 6- to 8-hr O₃ exposures. In March 1993, EPA concluded the review by deciding that revisions to the standards were not warranted at that time (58 FR 13008).

In August 1992 (57 FR 35542), EPA announced plans to initiate the third periodic review of the air quality criteria and O₃ NAAQS. On the basis of the scientific evidence contained in the 1996 CD (U.S. EPA 1996a) and the 1996 Staff Paper (U.S. EPA, 1996b), and related technical support documents, linking exposures to ambient O₃ to adverse health and welfare effects at levels allowed by the then existing standards, EPA proposed to revise the primary and secondary O₃ standards in December 1996 (61 FR 65716). The EPA proposed to replace the then existing 1-hour primary and secondary standards with 8-hour average O₃ standards set at a level of 0.08 ppm (equivalent to 0.084 ppm using standard rounding conventions). The EPA also proposed, in the alternative, to establish a new distinct secondary standard using a biologically based cumulative seasonal form. The EPA completed the review in July 1997 (62 FR 38856) by setting the

primary standard at a level of 0.08 ppm, based on the annual fourth-highest daily maximum 8-hr average concentration, averaged over three years, and setting the secondary standard identical to the revised primary standard.

The EPA initiated the most recent periodic review of the air quality criteria and standards for O₃ in September 2000 with a call for information (65 FR 57810; September 26, 2000) for the development of a revised Air Quality Criteria Document for O₃ and Other Photochemical Oxidants (henceforth the "2006 Criteria Document"). A project work plan (EPA, 2002) for the preparation of the Criteria Document was released in November 2002 for CASAC and public review. The EPA held a series of workshops in mid-2003 on several draft chapters of the Criteria Document to obtain broad input from the relevant scientific communities. These workshops helped to inform the preparation of the first draft Criteria Document (EPA, 2005a), which was released for CASAC and public review on January 31, 2005; a CASAC meeting was held on May 4–5, 2005 to review the first draft Criteria Document. A second draft Criteria Document (EPA, 2005b) was released for CASAC and public review on August 31, 2005, and was discussed along with a first draft Staff Paper (EPA, 2005c) at a CASAC meeting held on December 6–8, 2005. In a February 16, 2006 letter to the Administrator, CASAC provided comments on the second draft Criteria Document (Henderson, 2006a), and the final 2006 Criteria Document (EPA, 2006a) was released on March 21, 2006. In a June 8, 2006 letter to the Administrator (Henderson, 2006b), CASAC provided additional advice to the Agency concerning chapter 8 of the final 2006 Criteria Document (Integrative Synthesis) to help inform the second draft Staff Paper.

A second draft Staff Paper (EPA, 2006b) was released on July 17, 2006 and reviewed by CASAC on August 24–25, 2006. In an October 24, 2006 letter to the Administrator, CASAC provided advice and recommendations to the Agency concerning the second draft Staff Paper (Henderson, 2006c). A final 2007 Staff Paper (EPA, 2007a) was released on January 31, 2007. In a March 26, 2007 letter (Henderson, 2007), CASAC offered additional advice to the Administrator with regard to recommendations and revisions to the primary and secondary O₃ NAAQS.

The schedule for completion of the 2008 rulemaking was governed by a consent decree resolving a lawsuit filed in March 2003 by a group of plaintiffs representing national environmental

and public health organizations, alleging that EPA had failed to complete the review within the period provided by statute.⁴ The modified consent decree that governed the 2008 rulemaking, entered by the court on December 16, 2004, provided that EPA sign for publication notices of proposed and final rulemaking concerning its review of the O₃ NAAQS no later than March 28, 2007 and December 19, 2007, respectively. That consent decree was further modified in October 2006 to change these proposed and final rulemaking dates to no later than May 30, 2007 and February 20, 2008, respectively. These dates for signing the publication notices of proposed and final rulemaking were further extended to no later than June 20, 2007 and March 12, 2008, respectively. The proposed decision was signed on June 20, 2007 and published in the **Federal Register** on July 11, 2007 (72 FR 37818).

Public hearings on the proposed decision were held on Thursday, August 30, 2007 in Philadelphia, PA and Los Angeles, CA. On Wednesday, September 5, 2007, hearings were held in Atlanta, GA, Chicago, IL, and Houston, TX. A large number of comments were received from various commenters on the 2007 proposed revisions to the O₃ NAAQS. A comprehensive summary of all significant comments, along with EPA's responses (henceforth "Response to Comments"), can be found in the docket for the 2008 rulemaking, which is also the docket for this reconsideration rulemaking.

The EPA's final decision on the O₃ NAAQS was published in the **Federal Register** on March 27, 2008 (73 FR 16436). In the 2008 rulemaking, EPA revised the level of the 8-hour primary standard for O₃ to 0.075 parts per million (ppm), expressed to three decimal places. With regard to the secondary standard for O₃, EPA revised the 8-hour standard by making it identical to the revised primary standard. The EPA also made conforming changes to the Air Quality Index (AQI) for O₃, setting an AQI value of 100 equal to 0.075 ppm, 8-hour average, and making proportional changes to the AQI values of 50, 150 and 200.

D. Reconsideration of the 2008 O₃ NAAQS Final Rule

Consistent with a directive of the new Administration regarding the review of new and pending regulations (Emanuel memorandum, 74 FR 4435; January 26, 2009), the Administrator reviewed a

number of actions that were taken in the last year by the previous Administration. The 2008 final rule was included in this review in recognition of the central role that the NAAQS play in enabling EPA to fulfill its mission to protect the nation's public health and welfare. In her review, the Administrator was mindful of the need for judgments concerning the NAAQS to be based on a strong scientific foundation which is developed through a transparent and credible NAAQS review process, consistent with the core values highlighted in President Obama's memorandum on scientific integrity (March 9, 2009).

1. Decision To Initiate a Rulemaking To Reconsider

In her review of the 2008 final rule, several aspects of the final rule related to the primary and secondary standards stood out to the Administrator. As an initial matter, the Administrator noted that the 2008 final rule concluded that the 1997 primary and secondary O₃ standards were not adequate to protect public health and public welfare, and that revisions were necessary to provide increased protection. With respect to revision of the primary standard, the Administrator noted that the revised level established in the 2008 final rule was above the range that had been unanimously recommended by CASAC.⁵ She also noted that EPA received comments from a large number of commenters from the medical and public health communities, including EPA's Children's Health Protection Advisory Committee, all of which endorsed levels within CASAC's recommended range.

With respect to revision of the secondary O₃ standard, the Administrator noted that the 2008 final rule differed substantially from CASAC's recommendations that EPA adopt a new secondary O₃ standard based on a cumulative, seasonal measure of exposure. The 2008 final rule revised the secondary standard to be identical to the revised primary standard, which is based on an 8-hour daily maximum measure of exposure. She also noted that EPA received comments from a number of commenters representing environmental interests, all of which endorsed CASAC's recommendation for a new cumulative, seasonal secondary standard.⁶

⁵ The level of the 8-hour primary ozone standard was set at 0.075 ppm, while CASAC unanimously recommended a range between 0.060 and 0.070 ppm.

⁶ The Administrator also noted the exchange that had occurred between EPA and the Office of

Subsequent to issuance of the 2008 final rule, in April 2008, CASAC took the unusual step of sending EPA a letter expressing strong, unanimous disagreement with EPA's decisions on both the primary and secondary standards (Henderson, 2008). The CASAC explained that it did not endorse the revised primary O₃ standard as being sufficiently protective of public health because it failed to satisfy the explicit stipulation of the Act to provide an adequate margin of safety. The CASAC also expressed the view that failing to revise the secondary standard to a cumulative, seasonal form was not supported by the available science. In addition to CASAC's letter, the Administrator noted a recent adverse ruling issued by the U.S. Court of Appeals for the District of Columbia Circuit on another NAAQS decision. In February 2009, the DC Circuit remanded the Agency's decisions on the primary annual and secondary standards for fine particles (PM_{2.5}). In so doing, the Court found that EPA had not adequately explained the basis for its decisions, including why CASAC's recommendations for a more health-protective primary annual standard and for secondary standards different from the primary standards were not accepted. *American Farm Bureau v. EPA*, 559 F.3d. 512 (DC Cir. 2009).

Based on her review of the information described above, the Administrator is initiating a rulemaking to reconsider parts of the 2008 final rule. Specifically, the Administrator is reconsidering the level of the primary standard to ensure that it is sufficiently protective of public health, as discussed in section II below, and is reconsidering all aspects of the secondary standard to ensure that it appropriately reflects the available science and is sufficiently protective of public welfare, as discussed in section IV below. Based on her review, the Administrator has serious cause for concern regarding whether the revisions to the primary and secondary O₃ standards adopted in the 2008 final rule satisfy the requirements of the CAA, in light of the body of scientific evidence before the Agency. In addition, the importance of the O₃ NAAQS to public health and welfare weigh heavily in favor of reconsidering parts of the 2008 final rule as soon as possible, based on the scientific and technical information upon which the 2008 final rule was based.

Management and Budget (OMB) with regard to the final decision on the secondary standard, as discussed in the 2008 final rule (73 FR 16497).

⁴ *American Lung Association v. Whitman* (No. 1:03CV00778, D.DC 2003).

Also, EPA conducted a provisional assessment of “new” scientific papers (EPA, 2009) of scientific literature evaluating health and ecological effects of O₃ exposure published since the close of the 2006 Criteria Document upon which the 2008 O₃ NAAQS were based. The Administrator notes that the provisional assessment of “new” science found that such studies did not materially change the conclusions in the 2006 Criteria Document. This provisional assessment is supportive of the Administrator’s decision to reconsider parts of the 2008 final rule at this time, based on the scientific and technical information available for the 2008 final rule, as compared to foregoing such reconsideration and taking appropriate action in the future as part of the next periodic review of the air quality criteria and NAAQS, which will include such scientific and technical information.

The reconsideration of parts of the 2008 final rule discussed in this notice is based on the scientific and technical record from the 2008 rulemaking, including public comments and CASAC advice and recommendations. The information that was assessed during the 2008 rulemaking includes information in the 2006 Criteria Document (EPA, 2006a), the 2007 Policy Assessment of Scientific and Technical Information, referred to as the 2007 Staff Paper (EPA, 2007b), and related technical support documents including the 2007 REAs (U.S. EPA, 2007c; Abt Associates, 2007a,b). Scientific and technical information developed since the 2006 Criteria Document will be considered in the next periodic review, instead of this reconsideration rulemaking, allowing the new information to receive careful and comprehensive review by CASAC and the public before it is used as a basis in a rulemaking that determines whether to revise the NAAQS.

2. Ongoing Litigation

In May 2008, following publication of the 2008 final rule, numerous groups, including state, public health, environmental, and industry petitioners, challenged EPA’s decisions in federal court. The challenges were consolidated as *State of Mississippi, et al. v. EPA* (No. 08–1200, DC Cir. 2008). On March 10, 2009, EPA filed an unopposed motion requesting that the Court vacate the briefing schedule and hold the consolidated cases in abeyance. The Agency stated its desire to allow time for appropriate officials from the new Administration to review the O₃ standards to determine whether they should be maintained, modified or

otherwise reconsidered. The EPA further requested that it be directed to notify the Court and all the parties of any actions it has taken or intends to take, if any, within 180 days of the Court vacating the briefing schedule. On March 19, 2009, the Court granted EPA’s motion. Pursuant to the Court’s order, on September 16, 2009 EPA notified the Court and the parties of its decision to initiate a rulemaking to reconsider the primary and secondary O₃ standards set in March 2008 to ensure they satisfy the requirements of the CAA.⁷ In its notice to the Court, EPA stated that this notice of proposed rulemaking would be signed by December 21, 2009, and that the final rule will be signed by August 31, 2010.

II. Rationale for Proposed Decision on the Level of the Primary Standard

As an initial matter, the Administrator notes that the 2008 final rule concluded that the 1997 primary O₃ standard was “not sufficient and thus not requisite to protect public health with an adequate margin of safety, and that revision is needed to provide increased public health protection” (73 FR 16472). The Administrator is not reconsidering this aspect of the 2008 decision, which is based on the reasons discussed in section II.B of the 2008 final rule (73 FR 16443–16472). The Administrator also notes that the 2008 final rule concluded that it was appropriate to retain the O₃ indicator, the 8-hour averaging time, and form of the primary O₃ standard (specified as the annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years), while concluding that revision of the standard level was appropriate.⁸ The Administrator is not reconsidering these aspects of the 2008 decision, which are based on the reasons discussed in sections II.C.1–3 of the 2008 final rule, which address the indicator, averaging time, and form, respectively, of the primary O₃ standard (73 FR 16472–16475). For these reasons, the Administrator is not reopening the 2008

decision with regard to the need to revise the 1997 primary O₃ standard nor with regard to the indicator, averaging time, and form of the 2008 primary O₃ standard. Thus, the information that follows in this section specifically focuses on a reconsideration of level of the primary O₃ standard.

This section presents the rationale for the Administrator’s proposed decision that the O₃ primary standard, which was set at a level of 0.075 ppm in the 2008 final rule, should instead be set at a lower level within the range from 0.060 to 0.070 ppm. As discussed more fully below, the rationale for the proposed range of standard levels is based on a thorough review of the latest scientific information on human health effects associated with the presence of O₃ in the ambient air presented in the 2006 Criteria Document. This rationale also takes into account: (1) Staff assessments of the most policy-relevant information in the 2006 Criteria Document and staff analyses of air quality, human exposure, and health risks, presented in the 2007 Staff Paper, upon which staff recommendations for revisions to the primary O₃ standard in the 2008 rulemaking were based; (2) CASAC advice and recommendations, as reflected in discussions of drafts of the 2006 Criteria Document and 2007 Staff Paper at public meetings, in separate written comments, and in CASAC’s letters to the Administrator both before and after the 2008 rulemaking; and (3) public comments received during the development of these documents, either in connection with CASAC meetings or separately, and on the 2007 proposed rule.

In developing this rationale, the Administrator recognizes that the CAA requires her to reach a public health policy judgment as to what standard would be requisite to protect public health with an adequate margin of safety, based on scientific evidence and technical assessments that have inherent uncertainties and limitations. This judgment requires making reasoned decisions as to what weight to place on various types of evidence and assessments, and on the related uncertainties and limitations. Thus, in selecting standard levels to propose, and subsequently in selecting a final level, the Administrator is seeking not only to prevent O₃ levels that have been demonstrated to be harmful but also to prevent lower O₃ levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree.

In this proposed rule, EPA has drawn upon an integrative synthesis of the entire body of evidence, published

⁷ The EPA also separately announced that it will move quickly to implement any new standards that might result from this reconsideration. To reduce the workload for states during the interim period of reconsideration, the Agency intends to propose to defer compliance with the CAA requirement to designate areas as attainment or nonattainment. EPA will work with states, local governments and tribes to ensure that air quality is protected during that time.

⁸ The use of O₃ as the indicator for photochemical oxidants was adopted in the 1979 final rule and retained in subsequent rulemaking. An 8-hour averaging time and a form based on the annual fourth-highest daily maximum 8-hour concentration, averaged over 3 years, were adopted in the 1997 final rule and retained in the 2008 rulemaking.

through early 2006, on human health effects associated with the presence of O₃ in the ambient air. As discussed below in section II.A, this body of evidence addresses a broad range of health endpoints associated with exposure to ambient levels of O₃ (EPA, 2006a, chapter 8), and includes over one hundred epidemiologic studies conducted in the U.S., Canada, and many countries around the world.⁹ In reconsidering this evidence, EPA focuses on those health endpoints that have been demonstrated to be caused by exposure to O₃, or for which the 2006 Criteria Document judges associations with O₃ to be causal, likely causal, or for which the evidence is highly suggestive that O₃ contributes to the reported effects. This rationale also draws upon the results of quantitative exposure and risk assessments, discussed below in section II.B. Section II.C focuses on the considerations upon which the Administrator's proposed conclusions on the level of the primary standard are based. Policy-relevant evidence-based and exposure/risk-based considerations are discussed, and the rationale for the 2008 final rulemaking on the primary standard and CASAC advice, given both prior to the development of the 2007 proposed rule and following the 2008 final rule, are summarized. Finally, the Administrator's proposed conclusions on the level of the primary standard are presented. Section II.D summarizes the proposed decision on the level of the primary O₃ standard and the solicitation of public comments.

Judgments made in the 2006 Criteria Document and 2007 Staff Paper about the extent to which relationships between various health endpoints and short-term exposures to ambient O₃ are likely causal have been informed by several factors. As discussed below in section II.A, these factors include the nature of the evidence (*i.e.*, controlled human exposure, epidemiological, and/or toxicological studies) and the weight of evidence, which takes into account such considerations as biological plausibility, coherence of evidence, strength of association, and consistency of evidence.

In assessing the health effects data base for O₃, it is clear that human studies provide the most directly applicable information for determining causality because they are not limited

⁹ In its assessment of the epidemiological evidence judged to be most relevant to making decisions on the level of the O₃ primary standard, EPA has placed greater weight on U.S. and Canadian epidemiologic studies, since studies conducted in other countries may well reflect different demographic and air pollution characteristics.

by the uncertainties of dosimetry differences and species sensitivity differences, which would need to be addressed in extrapolating animal toxicology data to human health effects. Controlled human exposure studies provide data with the highest level of confidence since they provide human health effects data under closely monitored conditions and can provide exposure-response relationships. Epidemiological data provide evidence of associations between ambient O₃ levels and more serious acute and chronic health effects (*e.g.*, hospital admissions and mortality) that cannot be assessed in controlled human exposure studies. For these studies the degree of uncertainty introduced by potentially confounding variables (*e.g.*, other pollutants, temperature) and other factors affects the level of confidence that the health effects being investigated are attributable to O₃ exposures, alone and in combination with other copollutants.

In using a weight of evidence approach to inform judgments about the degree of confidence that various health effects are likely to be caused by exposure to O₃, confidence increases as the number of studies consistently reporting a particular health endpoint grows and as other factors, such as biological plausibility and strength, consistency, and coherence of evidence, increase. Conclusions regarding biological plausibility, consistency, and coherence of evidence of O₃-related health effects are drawn from the integration of epidemiological studies with mechanistic information from controlled human exposure studies and animal toxicological studies. As discussed below, this type of mechanistic linkage has been firmly established for several respiratory endpoints (*e.g.*, lung function decrements, lung inflammation) but remains far more equivocal for cardiovascular endpoints (*e.g.*, cardiovascular-related hospital admissions). For epidemiological studies, strength of association refers to the magnitude of the association and its statistical strength, which includes assessment of both effects estimate size and precision. In general, when associations yield large relative risk estimates, it is less likely that the association could be completely accounted for by a potential confounder or some other bias. Consistency refers to the persistent finding of an association between exposure and outcome in multiple studies of adequate power in different persons, places, circumstances and times. For example, the magnitude

of effect estimates is relatively consistent across recent studies showing association between short-term, but not long-term, O₃ exposure and mortality.

Based on the information discussed below in sections II.A.1–II.A.3, judgments concerning the extent to which relationships between various health endpoints and ambient O₃ exposures are likely causal are summarized below in section II.A.3.c. These judgments reflect the nature of the evidence and the overall weight of the evidence, and are taken into consideration in the quantitative exposure and risk assessments, discussed below in section II.B.

To put judgments about health effects that have been demonstrated to be caused by exposure to O₃, or for which the 2006 Criteria Document judges associations with O₃ to be causal, likely causal, or for which the evidence is highly suggestive that O₃ contributes to the reported effects into a broader public health context, EPA has drawn upon the results of the quantitative exposure and risk assessments. These assessments provide estimates of the likelihood that individuals in particular population groups that are at risk for various O₃-related physiological health effects would experience “exposures of concern” and specific health endpoints under varying air quality scenarios (*i.e.*, just meeting various standards¹⁰), as well as characterizations of the kind and degree of uncertainties inherent in such estimates.

In the 2008 final rulemaking and in this reconsideration, the term “exposures of concern” is defined as personal exposures while at moderate or greater exertion to 8-hour average ambient O₃ levels at and above specific benchmark levels which represent exposure levels at which O₃-related health effects are known or can reasonably be inferred to occur in some individuals, as discussed below in section II.B.1.¹¹ The EPA emphasizes

¹⁰ The exposure assessment done as part of the 2008 final rulemaking considered several air quality scenarios, including just meeting what was then the current standard set at a level of 0.084 ppm, as well as just meeting alternative standards at levels of 0.080, 0.074, 0.070, and 0.064 ppm.

¹¹ Exposures of concern were also considered in the 1997 review of the O₃ NAAQS, and were judged by EPA to be an important indicator of the public health impacts of those O₃-related effects for which information was too limited to develop quantitative estimates of risk but which had been observed in humans at and above the benchmark level of 0.08 ppm for 6- to 8-hour exposures * * * including increased nonspecific bronchial responsiveness (for example, aggravation of asthma), decreased pulmonary defense mechanisms (suggestive of increased susceptibility to respiratory infection), and indicators of pulmonary inflammation (related

Continued

that although the analysis of “exposures of concern” was conducted using three discrete benchmark levels (*i.e.*, 0.080, 0.070, and 0.060 ppm), the concept is more appropriately viewed as a continuum with greater confidence and less uncertainty about the existence of health effects at the upper end and less confidence and greater uncertainty as one considers increasingly lower O₃ exposure levels. The EPA recognizes that there is no sharp breakpoint within the continuum ranging from at and above 0.080 ppm down to 0.060 ppm. In considering the concept of exposures of concern, it is important to balance concerns about the potential for health effects and their severity with the increasing uncertainty associated with our understanding of the likelihood of such effects at lower O₃ levels.

Within the context of this continuum, estimates of exposures of concern at discrete benchmark levels provide some perspective on the public health impacts of O₃-related health effects that have been demonstrated in controlled human exposure and toxicological studies but cannot be evaluated in quantitative risk assessments, such as lung inflammation, increased airway responsiveness, and changes in host defenses. They also help in understanding the extent to which such impacts have the potential to be reduced by meeting various standards. These O₃-related physiological effects are plausibly linked to the increased morbidity seen in epidemiological studies (*e.g.*, as indicated by increased medication use in asthmatics, school absences in all children, and emergency department visits and hospital admissions in people with lung disease). Estimates of the number of people likely to experience exposures of concern cannot be directly translated into quantitative estimates of the number of people likely to experience specific health effects, since sufficient information to draw such comparisons is not available—if such information were available, these health outcomes would have been included in the quantitative risk assessment. Due to individual variability in responsiveness, only a subset of individuals who have exposures at and above a specific benchmark level can be expected to experience such adverse health effects, and susceptible subpopulations such as those with asthma are expected to be affected more by such exposures than healthy individuals. The amount of weight to place on the estimates of exposures of concern at any of these

benchmark levels depends in part on the weight of the scientific evidence concerning health effects associated with O₃ exposures at and above that benchmark level. It also depends on judgments about the importance from a public health perspective of the health effects that are known or can reasonably be inferred to occur as a result of exposures at and above the benchmark level. Such public health policy judgments are embodied in the NAAQS standard setting criteria (*i.e.*, standards that, in the judgment of the Administrator, are requisite to protect public health with an adequate margin of safety).

As discussed below in section II.B.2, the quantitative health risk assessment conducted as part of the 2008 final rulemaking includes estimates of risks of lung function decrements in asthmatic and all school age children, respiratory symptoms in asthmatic children, respiratory-related hospital admissions, and non-accidental and cardiorespiratory-related mortality associated with recent ambient O₃ levels, as well as risk reductions and remaining risks associated with just meeting the then current 0.084 ppm standard and various alternative O₃ standards in a number of example urban areas. There are two parts to this risk assessment: one part is based on combining information from controlled human exposure studies with modeled population exposure, and the other part is based on combining information from community epidemiological studies with either monitored or adjusted ambient concentrations levels. This assessment provides estimates of the potential magnitude of O₃-related health effects, as well as a characterization of the uncertainties and variability inherent in such estimates. This assessment also provides insights into the distribution of risks and patterns of risk reductions associated with meeting alternative O₃ standards.

As discussed below, a substantial amount of new research conducted since the 1997 review of the O₃ NAAQS was available to inform the 2008 final rulemaking, with important new information coming from epidemiologic studies as well as from controlled human exposure, toxicological, and dosimetric studies. The research studies newly available in the 2008 final rulemaking that were evaluated in the 2006 Criteria Document and the exposure and risk assessments presented in the 2007 Staff Paper have undergone intensive scrutiny through multiple layers of peer review and many opportunities for public review and comment. While important

uncertainties remain in the qualitative and quantitative characterizations of health effects attributable to exposure to ambient O₃, and while different interpretations of these uncertainties can result in different public health policy judgments, the review of this information has been extensive and deliberate. In the judgment of the Administrator, this intensive evaluation of the scientific evidence provides an adequate basis for this reconsideration of the 2008 final rulemaking.

A. Health Effects Information

This section outlines key information contained in the 2006 Criteria Document (chapters 4–8) and in the 2007 Staff Paper (chapter 3) on known or potential effects on public health which may be expected from the presence of O₃ in ambient air. The information highlighted here summarizes: (1) New information available on potential mechanisms for health effects associated with exposure to O₃; (2) the nature of effects that have been associated directly with exposure to O₃ and indirectly with the presence of O₃ in ambient air; (3) an integrative interpretation of the evidence, focusing on the biological plausibility and coherence of the evidence; and (4) considerations in characterizing the public health impact of O₃, including the identification of “at risk” populations.

The decision in the 1997 review focused primarily on evidence from short-term (*e.g.*, 1 to 3 hours) and prolonged (6 to 8 hours) controlled-exposure studies reporting lung function decrements, respiratory symptoms, and respiratory inflammation in humans, as well as epidemiology studies reporting excess hospital admissions and emergency department (ED) visits for respiratory causes. The 2006 Criteria Document prepared for the 2008 rulemaking emphasized the large number of epidemiological studies published since the last review with these and additional health endpoints, including the effects of acute (short-term and prolonged) and chronic exposures to O₃ on lung function decrements and enhanced respiratory symptoms in asthmatic individuals, school absences, and premature mortality. It also emphasized important new information from toxicology, dosimetry, and controlled human exposure studies. Highlights of the evidence include:

(1) Two new controlled human-exposure studies are now available that examine respiratory effects associated with prolonged O₃ exposures at levels below 0.080 ppm, which was the lowest

to potential aggravation of chronic bronchitis or long-term damage to the lungs). (62 FR 38868)

exposure level that had been examined in the 1997 review.

(2) Numerous controlled human-exposure studies have examined indicators of O₃-induced inflammatory response in both the upper respiratory tract (URT) and lower respiratory tract (LRT), and increased airway responsiveness to allergens in subjects with allergic asthma and allergic rhinitis exposed to O₃, while other studies have examined changes in host defense capability following O₃ exposure of healthy young adults.

(3) Animal toxicology studies provide new information regarding mechanisms of action, increased susceptibility to respiratory infection, and the biological plausibility of acute effects and chronic, irreversible respiratory damage.

(4) Numerous acute exposure epidemiological studies published during the past decade offer added evidence of ambient O₃-related lung function decrements and respiratory symptoms in physically active healthy subjects and greater responses in asthmatic subjects, as well as evidence on new health endpoints, such as the relationships between ambient O₃ concentrations and asthma medication use and school absenteeism, and between ambient O₃ and cardiac-related physiological endpoints.

(5) Several additional studies have been published over the last decade examining the temporal associations between O₃ exposures and emergency department visits for asthma and other respiratory diseases and respiratory-related hospital admissions.

(6) A large number of newly available epidemiological studies have examined the effects of acute exposure to PM and O₃ on mortality, notably including large multicity studies that provide much more robust and credible information than was available in the 1997 review, as well as recent meta-analyses that have evaluated potential sources of heterogeneity in O₃-mortality associations.

1. Overview of Mechanisms

Evidence on possible mechanisms by which exposure to O₃ may result in acute and chronic health effects is discussed in chapters 5 and 6 of the 2006 Criteria Document.¹² Evidence from dosimetry, toxicological, and

¹² While most of the available evidence addresses mechanisms for O₃, O₃ clearly serves as an indicator for the total photochemical oxidant mixture found in the ambient air. Some effects may be caused by one or more components in the overall pollutant mix, either separately or in combination with O₃. However, O₃ clearly dominates these other oxidants with their concentrations only being a few percent of the O₃ concentration.

human exposure studies has contributed to an understanding of the mechanisms that help to explain the biological plausibility and coherence of evidence for O₃-induced respiratory health effects reported in epidemiological studies. More detailed information about the physiological mechanisms related to the respiratory effects of short- and long-term exposure to O₃ can be found in section II.A.3.b.i and II.A.3.b.iii, respectively. In the past, however, little information was available to help explain potential biological mechanisms which linked O₃ exposure to premature mortality or cardiovascular effects. As discussed more fully in section II.A.3.b.ii below, since the 1997 review an emerging body of animal toxicology and controlled human exposure evidence is beginning to suggest mechanisms that may mediate acute O₃ cardiovascular effects. While much is known about mechanisms that play a role in O₃-related respiratory effects, additional research is needed to more clearly understand the role that O₃ may have in contributing to cardiovascular effects.

With regard to the mechanisms related to short-term respiratory effects, scientific evidence discussed in the 2006 Criteria Document (section 5.2) indicates that reactions of O₃ with lipids and antioxidants in the epithelial lining fluid and the epithelial cell membranes of the lung can be the initial step in mediating deleterious health effects of O₃. This initial step activates a cascade of events that lead to oxidative stress, injury, inflammation, airway epithelial damage and increased alveolar permeability to vascular fluids. Inflammation can be accompanied by increased airway responsiveness, which is an increased bronchoconstrictive response to airway irritants and allergens. Continued respiratory inflammation also can alter the ability of the body to respond to infectious agents, allergens and toxins. Acute inflammatory responses to O₃ in some healthy people are well documented, and precursors to lung injury are observed within 3 hours after exposure in humans. Repeated respiratory inflammation can lead to a chronic inflammatory state with altered lung structure and lung function and may lead to chronic respiratory diseases such as fibrosis and emphysema (EPA, 2006a, section 8.6.2). The severity of symptoms and magnitude of response to acute exposures depend on inhaled dose, as well as on individual susceptibility to O₃, as discussed below. At the same O₃ dose, individuals who are more susceptible to O₃ will have a larger

response than those who are less susceptible; among individuals with similar susceptibility, those who receive a larger dose will have a larger response to O₃.

The inhaled dose is the product of O₃ concentration (C), minute ventilation or ventilation rate, and duration of exposure (T), or (C × ventilation rate × T). A large body of data regarding the interdependent effect of these components of inhaled dose on pulmonary responses was assessed in the 1986 and 1996 O₃ Criteria Documents. In an attempt to describe O₃ dose-response characteristics, acute responses were modeled as a function of total inhaled O₃ dose, which was generally found to be a better predictor of response than O₃ concentration, ventilation rate, or duration of exposure, alone, or as a combination of any two of these factors (EPA 2006a, section 6.2). Predicted O₃-induced decrements in lung function have been shown to be a function of exposure concentration, duration and exercise level for healthy, young adults (McDonnell *et al.*, 1997). A meta-analysis of 21 studies (Mudway and Kelly, 2004) showed that markers of inflammation and increased cellular permeability in healthy subjects are associated with total O₃ dose.

The 2006 Criteria Document summarizes information on potentially susceptible and vulnerable groups in section 8.7. As described there, the term *susceptibility* refers to innate (*e.g.*, genetic or developmental) or acquired (*e.g.*, personal risk factors, age) factors that make individuals more likely to experience effects with exposure to pollutants. A number of population groups and lifestages have been identified as potentially susceptible to health effects as a result of O₃ exposure, including people with existing lung diseases, including asthma, children and older adults, and people who have larger than normal lung function responses that may be due to genetic susceptibility. In addition, some population groups and lifestages have been identified as having increased *vulnerability* to O₃-related effects due to increased likelihood of exposure while at elevated ventilation rates, including healthy children and adults who are active outdoors, for example, outdoor workers, and joggers. Taken together, the susceptible and vulnerable groups are more commonly referred to as “at-risk” groups,¹³ as discussed more fully below in section II.A.4.b.

¹³ In previous Staff Papers and Federal Register notices announcing proposed and final decisions on the O₃ and other NAAQS, EPA has used the phrase

Based on a substantial body of new evidence from animal, controlled human exposure and epidemiological studies, the 2006 Criteria Document concludes that people with asthma and other preexisting pulmonary diseases are likely to be among those at increased risk from O₃ exposure. Altered physiological, morphological and biochemical states typical of respiratory diseases like asthma, COPD and chronic bronchitis may render people sensitive to additional oxidative burden induced by O₃ exposure (EPA 2006a, section 8.7). Children and adults with asthma are the group that has been studied most extensively. Evidence from controlled human exposure studies indicates that asthmatics may exhibit larger lung function decrements in response to O₃ exposure than healthy controls. As discussed more fully in section II.A.4.b.ii below, asthmatics present a differential response profile for cellular, molecular, and biochemical parameters (EPA, 2006a, section 8.7.1) that are altered in response to acute O₃ exposure. They can have larger inflammatory responses, as manifested by larger increases in markers of inflammation such as white blood cells (e.g., PMNs) or inflammatory cytokines. Asthmatics, and people with allergic rhinitis, are more likely to mount an allergic-type response upon exposure to O₃, as manifested by increases in white blood cells associated with allergy (i.e., eosinophils) and related molecules, which increase inflammation in the airways. The increased inflammatory and allergic responses also may be associated with the larger late-phase responses that asthmatics can experience, which can include increased bronchoconstrictor responses to irritant substances or allergens and additional inflammation. In addition to the experimental evidence of lung function decrements, respiratory symptoms, and other respiratory effects in asthmatic populations, two large U.S. epidemiological studies as well as several smaller U.S. and international studies, have reported fairly robust associations between ambient O₃ concentrations and measures of lung function and daily symptoms (e.g., chest tightness, wheeze, shortness of breath) in children with moderate to severe asthma and between O₃ and increased asthma medication use (EPA, 2007a, chapter 6). These responses in

asthmatics and others with lung disease provide biological plausibility for the more serious respiratory morbidity effects observed in epidemiological studies, such as emergency department visits and hospital admissions.

Children with and without asthma were found to be particularly susceptible to O₃ effects on lung function and generally have greater lung function responses than older people. The American Academy of Pediatrics (2004) notes that children and infants are among the population groups most susceptible to many air pollutants, including O₃. This is in part because their lungs are still developing. For example, eighty percent of alveoli are formed after birth, and changes in lung development continue through adolescence (Dietert *et al.*, 2000). Moreover, children have high minute ventilation rates and relatively high levels of physical activity which also increases their O₃ dose (Plunkett *et al.*, 1992). Thus, children are at-risk due to both their susceptibility and vulnerability.

Looking more broadly at age-related differences in susceptibility, several mortality studies have investigated age-related differences in O₃ effects (EPA, 2006a, section 7.6.7.2), primarily in the older adult population. Among the studies that observed positive associations between O₃ and mortality, a comparison of all age or younger age (65 years of age) O₃-mortality effect estimates to that of the elderly population (>65 years) indicates that, in general, the elderly population is more susceptible to O₃ mortality effects. There is supporting evidence of age-related differences in susceptibility to O₃ lung function effects. The 2006 Criteria Document (section 7.6.7.2) concludes that the elderly population (>65 years of age) appear to be at greater risk of O₃-related mortality and hospitalizations compared to all ages or younger populations, and children (<18 years of age) experience other potentially adverse respiratory health outcomes with increased O₃ exposure.

Controlled human exposure studies have also indicated a high degree of interindividual variability in some of the pulmonary physiological parameters, such as lung function decrements. The variable effects in individuals have been found to be reproducible, in other words, a person who has a large lung function response after exposure to O₃ will likely have about the same response if exposed again to the same dose of O₃ (EPA 2006a, section 6.1). In controlled human exposure studies, group mean responses are not representative of this segment of

the population that has much larger than average responses to O₃. Recent studies, discussed in section II.A.4.b.iv below, reported a role for genetic polymorphism (i.e., the occurrence together in the same population of more than one allele or genetic marker at the same locus with the least frequent allele or marker occurring more frequently than can be accounted for by mutation alone) in observed differences in antioxidant enzymes and genes involved in inflammation to modulate pulmonary function and inflammatory responses to O₃ exposure. These observations suggest a potential role for these markers in the innate susceptibility to O₃, however, the validity of these markers and their relevance in the context of prediction to population studies needs additional experimentation.

Controlled human exposure studies that provide information about mechanisms of the initial response to O₃ (e.g., lung function decrements, inflammation, and injury to the lung) also inform the selection of appropriate lag times to analyze in epidemiological studies through elucidation of the time course of these responses (EPA 2006a, section 8.4.3). Based on the results of these studies, it would be reasonable to expect that lung function decrements could be detected epidemiologically within lags of 0 (same day) or 1 to 2 days following O₃ exposure, given the rapid onset of lung function changes and their persistence for 24 to 48 hours among more responsive human subjects in controlled human exposure studies. Other responses take longer to develop and can persist for longer periods of time. For example, although asthmatic individuals may begin to experience symptoms soon after O₃ exposure, it may take anywhere from 1 to 3 days after exposure for these subjects to seek medical attention as a result of increased airway responsiveness or inflammation that may persist for 2 to 3 days. This may be reflected by epidemiologic observations of significantly increased risk for asthma-related emergency department visits or hospital admissions with 1- to 3-day lags, or, perhaps, enhanced distributed lag risks (combined across 3 days) for such morbidity indicators. Analogously, one might project increased mortality within 0- to 3-day lags as a possible consequence of O₃-induced increases in clotting agents arising from the cascade of events, starting with cell injury described above, occurring within 12 to 24 hours of O₃ exposure. The time course for many of these initial responses to O₃ is highly variable.

“sensitive population groups” to include both population groups that are at increased risk because they are more intrinsically susceptible and population groups that are more vulnerable due to an increased potential for exposure. In this notice, we use the phrase, “at risk” populations to include both types of population groups.

Moreover these observations pertain only to the initial response to O₃. Consequent responses can follow. For example, Jörres *et al.*, (1996) found that in subjects with asthma and allergic rhinitis, a maximum percent fall in FEV₁ of 27.9% and 7.8%, respectively, occurred 3 days after O₃ exposure when they were challenged with of the highest common dose of allergen.

2. Nature of Effects

The 2006 Criteria Document provides new evidence that notably enhances our understanding of short-term and prolonged exposure effects, including effects on lung function, symptoms, and inflammatory effects reported in controlled exposure studies. These studies support and extend the findings of the previous Criteria Document. There is also a significant body of new epidemiological evidence of associations between short-term and prolonged exposure to O₃ and effects such as premature mortality, hospital admissions and emergency department visits for respiratory (*e.g.*, asthma) causes. Key epidemiological and controlled human exposure studies are summarized below and discussed in chapter 3 of the 2007 Staff Paper, which is based on scientific evidence critically reviewed in chapters 5, 6, and 7 of the 2006 Criteria Document, as well as the Criteria Document's integration of scientific evidence contained in chapter 8.¹⁴ Conclusions drawn about O₃-related health effects are based upon the full body of evidence from controlled human exposure, epidemiological and toxicological data contained in the 2006 Criteria Document.

a. Morbidity

This section summarizes scientific information on the effects of inhalation of O₃, including public health effects of short-term, prolonged, and long-term exposures on respiratory morbidity and cardiovascular system effects, as discussed in chapters 6, 7 and 8 of the 2006 Criteria Document and chapter 3 of the 2007 Staff Paper. This section also summarizes the uncertainty about the potential indirect effects on public health associated with changes due to increases in UV-B radiation exposure, such as UV-B radiation-related skin cancers, that may be associated with reductions in ambient levels of ground-level O₃, as discussed in chapter 10 of the 2006 Criteria Document and chapter 3 of the 2007 Staff Paper.

i. Effects on the Respiratory System From Short-term and Prolonged O₃ Exposures

Controlled human exposure studies have shown that O₃ induces a variety of health effects, including: Lung function decrements, respiratory symptoms, increased airway responsiveness, respiratory inflammation and permeability, increased susceptibility to respiratory infection, and acute morphological effects. Epidemiology studies have reported associations between O₃ exposures (*i.e.*, 1-hour, 8-hour and 24-hour) and a wide range of respiratory-related health effects including: pulmonary function decrements; respiratory symptoms; increased asthma medication use; increased school absences; increased emergency department visits and hospital admissions.

(a) Pulmonary Function Decrements, Respiratory Symptoms, and Asthma Medication Use

(i) Results From Controlled Human Exposure Studies

A large number of studies published prior to 1996 that investigated short-term O₃ exposure health effects on the respiratory system from short-term O₃ exposures were reviewed in the 1986 and 1996 Criteria Documents (EPA, 1986, 1996a). In the 1997 review, 0.50 ppm was the lowest O₃ concentration at which statistically significant reductions in forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV₁) were reported in sedentary subjects. During exercise, spirometric (lung function) and symptomatic responses were observed at much lower O₃ exposures. When minute ventilation was considerably increased by continuous exercise (CE) during O₃ exposures lasting 2 hour or less at ≥ 0.12 ppm, healthy subjects generally experienced decreases in FEV₁, FVC, and other measures of lung function; increases in specific airway resistance (sRaw), breathing frequency, and airway responsiveness; and symptoms such as cough, pain on deep inspiration, shortness of breath, throat irritation, and wheezing. When exposures were increased to 4- to 8-hours in duration, statistically significant lung function and symptom responses were reported at O₃ concentrations as low as 0.08 ppm and at lower minute ventilation (*i.e.*, moderate rather than high level exercise) than the shorter duration studies.

The most important observations drawn from studies reviewed in the 1996 Criteria Document were that: (1)

Young healthy adults exposed to O₃ concentrations ≥ 0.080 ppm develop significant, reversible, transient decrements in pulmonary function if minute ventilation or duration of exposure is increased sufficiently; (2) children experience similar lung function responses but report lesser symptoms from O₃ exposure relative to young adults; (3) O₃-induced lung function responses are decreased in the elderly relative to young adults; (4) there is a large degree of intersubject variability in physiological and symptomatic responses to O₃ but responses tend to be reproducible within a given individual over a period of several months; (5) subjects exposed repeatedly to O₃ for several days show an attenuation of response upon successive exposures, but this attenuation is lost after about a week without exposure; and (6) acute O₃ exposure initiates an inflammatory response which may persist for at least 18 to 24 hours post exposure.

The development of these respiratory effects is time-dependent during both exposure and recovery periods, with great overlap for development and disappearance of the effects. In healthy human subjects exposed to typical ambient O₃ levels near 0.120 ppm, lung function responses largely resolve within 4 to 6 hours postexposure, but cellular effects persist for about 24 hours. In these healthy subjects, small residual lung function effects are almost completely gone within 24 hours, while in hyperresponsive subjects, recovery can take as much as 48 hour to return to baseline. The majority of these responses are attenuated after repeated consecutive exposures, but such attenuation to O₃ is lost one week postexposure.

Since 1996, there have been a number of studies published investigating lung function and symptomatic responses that generally support the observations previously drawn. Recent studies for acute exposures of 1 to 2 hours and 6 to 8 hours in duration are compiled in the 2007 Staff Paper (Appendix 3C). As summarized in more detail in the 2007 Staff Paper (section 3.3.1.1), among the more important of the recent studies that examined changes in FEV₁ in large numbers of subjects over a range of 1–2 hours at exposure levels of 0.080 to 0.40 ppm were studies by McDonnell *et al.* (1997) and Ultman *et al.* (2004). These studies observed considerable intersubject variability in FEV₁ decrements, which was consistent with findings in the 1996 Criteria Document.

For prolonged exposures (4 to 8 hours) in the range of 0.080 to 0.160 ppm O₃ using moderate intermittent

¹⁴ Health effects discussions are also drawn from the more detailed information and tables presented in the Criteria Document's annexes.

exercise and typically using square-wave exposure patterns (*i.e.*, a constant exposure level during time of exposure), several pre- and post-1996 studies (Folinsbee *et al.*, 1988,1994; Horstman *et al.*, 1990; Adams, 2002, 2003a, 2006) have reported statistically significant lung function responses and increased symptoms in healthy adults with increasing duration of exposure, O₃ concentration, and minute ventilation. Studies that employed triangular exposure patterns (*i.e.*, integrated exposures that begin at a low level, rise to a peak, and return to a low level during the exposure) (Hazucha *et al.*, 1992; Adams 2003a, 2006) suggest that the triangular exposure pattern can potentially lead to greater FEV₁ decrements and respiratory symptoms than square-wave exposures (when the overall O₃ doses are equal). These results suggest that peak exposures, reflective of the pattern of ambient O₃ concentrations in some locations, are important in terms of O₃ health effects.

McDonnell (1996) used data from a series of studies to investigate the frequency distributions of FEV₁ decrements following 6.6 hour exposures and found statistically significant, but relatively small, group mean decreases in average FEV₁ responses (between 5 and 10 percent) at 0.080 ppm O₃.¹⁵ Notably, about 26 percent of the 60 exposed subjects had lung function decrements > 10 percent, including about 8 percent of the subjects that experienced large decrements (> 20 percent) (EPA, 2007b, Figure 3–1A). These results (which were not corrected for exercise in filtered air responses) demonstrate that while average responses may be relatively small at the 0.080 ppm exposure level, some individuals experience more severe effects that may be clinically significant. Similar results at the 0.080 ppm exposure level (for 6.6 hours during intermittent exercise) were seen in more recent studies of 30 healthy young adults by Adams (2002, 2006).¹⁶ In Adams (2006), relatively small but statistically significant lung function decrements and respiratory symptom responses were found (for both square-wave and triangular exposure patterns), with 17 percent of the subjects (5 of 30) experiencing ≥ 10 percent FEV₁

decrements (comparing pre- and post-exposures) when the results were not corrected for the effects of exercise alone in filtered air (EPA, 2007b, Figure 3–1B) and with 23 percent of subjects (7 of 30) experiencing such effects when the results were corrected (EPA, 2007b, p. 3–6).¹⁷

These studies by Adams (2002, 2006) were notable in that they were the only controlled exposure human studies available at the time of the 2008 rulemaking that examined respiratory effects associated with prolonged O₃ exposures at levels below 0.080 ppm, which was the lowest exposure level that had been examined in the 1997 review. The Adams (2006) study investigated a range of exposure levels (0.000, 0.040, 0.060, and 0.080 ppm O₃) using square-wave and triangular exposure patterns. The study was designed to examine hour-by-hour changes in pulmonary function (FEV₁) and respiratory symptom responses (total subjective symptoms (TSS) and pain on deep inspiration (PDI)) between these various exposure protocols at six different time points within the exposure periods to investigate the effects of different patterns of exposure. At the 0.060 ppm exposure level, the author reported no statistically significant differences for FEV₁ decrements nor for most respiratory symptoms responses. Statistically significant responses were reported only for TSS for the triangular exposure pattern toward the end of the exposure period, with the PDI responses being noted as following a closely similar pattern (Adams, 2006, p. 131–132). EPA's reanalysis of the data from the Adams (2006) study addressed the more fundamental question of whether there were statistically significant differences in responses before and after the 6.6 hour exposure period (Brown, 2007), and used a standard statistical method appropriate for a simple before-and-after comparison. The statistical method used by EPA had been used previously by other researchers to address this same question. EPA's reanalysis of the data from the Adams (2006) study, comparing FEV₁ responses pre- and post-exposure at the 0.060 ppm exposure level, found small group mean differences from responses to filtered air that were statistically significant (Brown, 2007).¹⁸

Further examination of the post-exposure FEV₁ data and mean data at other time points and concentrations also suggest a pattern of response at 0.06 ppm that is consistent with a dose-response relationship rather than random variability. For example, the response at 5.6 hours was similar to that of the post-exposure 6.6 hour response and appeared to also differ from the FA response. At the 0.08 ppm level, the subjects in this study did not appear to be more responsive to O₃ than subjects in previous studies, as the observed response was similar to that of previous studies (Adams, 2003a,b; Horstman *et al.*, 1990; McDonnell *et al.*, 1991). Although of much smaller magnitude, the temporal pattern of the 0.06 ppm response was generally consistent with the temporal patterns of response to higher concentrations of O₃ in this and other studies. These findings are not unexpected because the previously observed group mean FEV₁ responses to 0.08 ppm were in the range of 6–9% suggesting that exposure to lower concentrations of O₃ would result in smaller, but real group mean FEV₁ decrements, *i.e.*, the responses to 0.060 ppm O₃ are consistent with the presence of a smooth exposure-response curve with responses that do not end abruptly below 0.080 ppm.

Moreover, the Adams studies (2002, 2006) also report a small percentage of subjects experiencing moderate lung function decrements (≥ 10 percent) at the 0.060 ppm exposure level. Based on study data (Adams, 2006) provided by the author, 7 percent of the subjects (2 of 30 subjects) experienced notable FEV₁ decrements (≥ 10 percent) with the square wave exposure pattern at the 0.060 ppm exposure level (comparing pre- and post-exposures) when the results were corrected for the effects of exercise alone in filtered air (EPA, 2007b, p. 3–6). Furthermore, in a prior publication (Adams, 2002), the author stated that, “some sensitive subjects experience notable effects at 0.06 ppm,” based on the observation that 20% of subjects exposed to 0.06 ppm O₃ (in a face mask exposure study) had greater than a 10% decrement in FEV₁ even though the group mean response was not statistically different from the filtered air response. The effects described by Adams (2002), along with

exposure level in his study (Adams, 2006a) does not demonstrate a significant mean effect by ordinarily acceptable statistical analysis, but is rather in somewhat of a gray area, both in terms of a biologically meaningful response and a statistically significant response (Adams, 2007). The EPA responded to these comments in the 2008 final rule (73 FR 16455) and in the Response to Comments (EPA, 2008, pp. 26–28).

¹⁵This study and other studies (Folinsbee *et al.*, 1988; Horstman *et al.*, 1990; and McDonnell *et al.*, 1991), conducted in EPA's human studies research facility in Chapel Hill, NC, measured ozone concentrations to within +/- 5 percent or +/- 0.004 ppm at the 0.080 ppm exposure level.

¹⁶These studies, conducted at a facility at the University of California, in Davis, CA, reported O₃ concentrations to be accurate within +/- 0.003 ppm over the range of concentrations included in these studies.

¹⁷These distributional results presented in the Criteria Document and Staff Paper for the Adams (2006) study are based on data for square-wave exposures to 0.080 ppm that were not included in the publication but were obtained from the author.

¹⁸Dr. Adams submitted comments on EPA's reanalysis in which he concluded that the FEV₁ response in healthy young adults at the 0.060 ppm

the reanalysis of the Adams (2006) data as described above, demonstrate considerable inter-individual variability in responses of healthy adults at the 0.060 ppm level with some individuals experiencing greater than 10% decrements in FEV₁. The observation of statistically significant small group mean lung function decrements in healthy adults at 0.060 ppm O₃ lowers the lowest-observed-effects level found in controlled human exposure studies for lung function decrements and respiratory symptoms.

Of potentially greater concern is the magnitude of the lung function decrements in the small group of healthy subjects who had the largest responses (*i.e.*, FEV₁ decrements \geq 10%). This is a concern because for active healthy people, moderate levels of functional responses (*e.g.*, FEV₁ decrements of \geq 10% but $<$ 20%) and/or moderate symptomatic responses would likely interfere with normal activity for relatively few responsive individuals. However, for people with lung disease, even moderate functional or symptomatic responses would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication (see section II.A.4 below).

(ii) Results of Epidemiological and Field Studies

A relatively large number of field studies investigating the effects of ambient O₃ concentrations, in combination with other air pollutants, on lung function decrements and respiratory symptoms has been published over the last decade that support the major findings of the 1996 Criteria Document that lung function changes, as measured by decrements in FEV₁ or peak expiratory flow (PEF), and respiratory symptoms in healthy adults and asthmatic children are closely correlated to ambient O₃ concentrations. Pre-1996 field studies focused primarily on children attending summer camps and found O₃-related impacts on measures of lung function, but not respiratory symptoms, in healthy children. The newer studies have expanded to evaluate O₃-related effects on outdoor workers, athletes, the elderly, hikers, school children, and asthmatics. Collectively, these studies confirm and extend clinical observations that prolonged (*i.e.*, 6–8 hour) exposure periods, combined with elevated levels of exertion or exercise, increase the dose of O₃ to the lungs at a given ambient exposure level and result in larger lung function effects. The results of one large study of hikers (Korrick *et al.*, 1998), which reported

outcome measures stratified by several factors (*e.g.*, gender, age, smoking status, presence of asthma) within a population capable of more than normal exertion, provide useful insight. In this study, lung function was measured before and after hiking, and individual O₃ exposures were estimated by averaging hourly O₃ concentrations from ambient monitors located at the base and summit. The mean 8-hour average O₃ concentration was 0.040 ppm (8-hour average concentration range of 0.021 ppm to 0.074 ppm O₃). Decreased lung function was associated with O₃ exposure, with the greatest effect estimates reported for the subgroup that reported having asthma or wheezing, and for those who hiked for longer periods of time.

Asthma panel studies conducted both in the U.S. and in other countries have reported that decrements in PEF are associated with routine O₃ exposures among asthmatic and healthy people. One large U.S. multicity study, the National Cooperative Inner City Asthma Study or NCICAS, (Mortimer *et al.*, 2002) examined O₃-related changes in PEF in 846 asthmatic children from 8 urban areas and reported that the incidence of \geq 10 percent decrements in morning PEF are associated with increases in 8-hour average O₃ for a 5-day cumulative lag, suggesting that O₃ exposure may be associated with clinically significant changes in PEF in asthmatic children; however, no associations were reported with evening PEF. The mean 8-hour average O₃ was 0.048 ppm across the 8 cities. Excluding days when 8-hour average O₃ was greater than 0.080 ppm (less than 5 percent of days), the associations with morning PEF remained statistically significant. Mortimer *et al.* (2002) discussed potential biological mechanisms for delayed effects on pulmonary function in asthma, which included increased nonspecific airway responsiveness secondary to airway inflammation due to O₃ exposure. Two other panel studies (Romieu *et al.*, 1996, 1997) carried out simultaneously in northern and southwestern Mexico City with mildly asthmatic school children reported statistically significant O₃-related reductions in PEF, with variations in effect depending on lag time and time of day. Mean 1-hour maximum O₃ concentrations in these locations ranged from 0.190 ppm in northern Mexico City to 0.196 ppm in southwestern Mexico City. While several studies report statistically significant associations between O₃ exposure and reduced PEF in asthmatics, other studies did not,

possibly due to low levels of O₃ exposure. EPA concludes that these studies collectively indicate that O₃ may be associated with short-term declines in lung function in asthmatic individuals and that the Mortimer *et al.* (2002) study showed statistically significant effects at concentrations in the range below 0.080 ppm O₃.

Most of the panel studies which have investigated associations between O₃ exposure and respiratory symptoms or increased use of asthma medication are focused on asthmatic children. Two large U.S. studies (Mortimer *et al.*, 2002; Gent *et al.*, 2003) have reported associations between ambient O₃ concentrations and daily symptoms/asthma medication use, even after adjustment for copollutants. Results were more mixed, meaning that a greater proportion of studies were not both positive and statistically significant, across smaller U.S. and international studies that focused on these health endpoints.

The NCICAS reported morning symptoms in 846 asthmatic children from 8 U.S. urban areas to be most strongly associated with a cumulative 1- to 4-day lag of O₃ concentrations (Mortimer *et al.*, 2002). The NCICAS used standard protocols that included instructing caretakers of the subjects to record symptoms (including cough, chest tightness, and wheeze) in the daily diary by observing or asking the child. While these associations were not statistically significant in several cities, when the individual data are pooled from all eight cities, statistically significant effects were observed for the incidence of symptoms. The authors also reported that the odds ratios remained essentially the same and statistically significant for the incidence of morning symptoms when days with 8-hour O₃ concentrations above 0.080 ppm were excluded. These days represented less than 5 percent of days in the study.

Gent and colleagues (2003) followed 271 asthmatic children under age 12 and living in southern New England for 6 months (April through September) using a daily symptom diary. They found that mean 1-hour max O₃ and 8-hour max O₃ concentrations were 0.0586 ppm and 0.0513 ppm, respectively. The data were analyzed for two separate groups of subjects, those who used maintenance asthma medications during the follow-up period and those who did not. The need for regular medication was considered to be a proxy for more severe asthma. Not taking any medication on a regular basis and not needing to use a bronchodilator would suggest the

presence of very mild asthma. Statistically significant effects of 1-day lag O₃ were observed on a variety of respiratory symptoms only in the medication user group. Both daily 1-hour max and 8-hour max O₃ concentrations were similarly related to symptoms such as chest tightness and shortness of breath. Effects of O₃, but not PM_{2.5}, remained significant and even increased in magnitude in two-pollutant models. Some of the associations were noted at 1-hour max O₃ levels below 0.060 ppm. In contrast, no effects were observed among asthmatics not using maintenance medication. In terms of person-days of follow-up, this is one of the larger studies currently available that address symptom outcomes in relation to O₃ and provides supportive evidence for effects of O₃ independent of PM_{2.5}. Study limitations include the post-hoc nature of the population stratification by medication use. Also, the study did not account for all of the important meteorological factors that might influence these results, such as relative humidity or dew point.

The multicity study by Mortimer *et al.* (2002), which examined an asthmatic population representative of the United States, and several single-city studies indicate a robust association of O₃ concentrations with respiratory symptoms and increased medication use in asthmatics. While there are a number of well-conducted, albeit relatively smaller, U.S. studies which showed only limited or a lack of evidence for symptom increases associated with O₃ exposure, these studies had less statistical power and/or were conducted in areas with relatively low 1-hour maximum average O₃ levels, in the range of 0.03 to 0.09 ppm. The 2006 Criteria Document concludes that the asthma panel studies, as a group, and the NCICAS in particular, indicate a positive association between ambient concentrations and respiratory symptoms and increased medication use in asthmatics. The evidence has continued to expand since 1996 and now is considered to be much stronger than in the 1997 review of the O₃ primary standard.

School absenteeism is another potential surrogate for the health implications of O₃ exposure in children. The association between school absenteeism and ambient O₃ concentrations was assessed in two relatively large field studies. The first study, Chen *et al.* (2000), examined total daily school absenteeism in about 28,000 elementary school students in Nevada over a 2-year period (after adjusting for PM₁₀ and CO

concentrations) and found that ambient O₃ concentrations with a distributed lag of 14 days were statistically significantly associated with an increased rate of school absences. The second study, Gilliland *et al.* (2001), studied O₃-related absences among about 2,000 4th grade students in 12 southern California communities and found statistically significant associations between 8-hour average O₃ concentrations (with a distributed lag out to 30 days) and all absence categories, and particularly for respiratory causes. Neither PM₁₀ nor NO₂ were associated with any respiratory or nonrespiratory illness-related absences in single pollutant models. The 2006 Criteria Document concludes that these studies of school absences suggest that ambient O₃ concentrations, accumulated over two to four weeks, may be associated with school absenteeism, and particularly illness-related absences, but further replication is needed before firm conclusions can be reached regarding the effect of O₃ on school absences. In addition, more research is needed to help shed light on the implications of variation in the duration of the lag structures (*i.e.*, 1 day, 5 days, 14 days, and 30 days) found both across studies and within data sets by health endpoint and exposure metric.

(b) Increased Airway Responsiveness

As discussed in more detail in the 2006 Criteria Document (section 6.8) and the 2007 Staff Paper (section 3.3.1.1.2), increased airway responsiveness, also known as airway hyperresponsiveness (AHR) or bronchial hyperreactivity, refers to a condition in which the propensity for the airways to bronchoconstrict due to a variety of stimuli (*e.g.*, exposure to cold air, allergens, or exercise) becomes augmented. This condition is typically quantified by measuring the decrement in pulmonary function after inhalation exposure to specific (*e.g.*, antigen, allergen) or nonspecific (*e.g.*, methacholine, histamine) bronchoconstrictor stimuli. Exposure to O₃ causes an increase in airway responsiveness as indicated by a reduction in the concentration of stimuli required to produce a given reduction in FEV₁ or increase in airway obstruction. Increased airway responsiveness is an important consequence of exposure to O₃ because its presence means that the airways are predisposed to narrowing on exposure to various stimuli, such as specific allergens, cold air or SO₂. Statistically significant and clinically relevant decreases in pulmonary function have

been observed in early phase allergen response in subjects with allergic rhinitis after consecutive (4-day) 3-hour exposures to 0.125 ppm O₃ (Holz *et al.*, 2002). Similar increased airway responsiveness in asthmatics to house dust mite antigen 16 to 18 hours after exposure to a single dose of O₃ (0.160 ppm for 7.6 hours) was observed. These observations, based on O₃ exposures to levels much higher than the 0.084 ppm standard level suggest that O₃ exposure may be a clinically important factor that can exacerbate the response to ambient bronchoconstrictor substances in individuals with preexisting allergic asthma or rhinitis. Further, O₃ may have an immediate impact on the lung function of asthmatics as well as contribute to effects that persist for longer periods.

Kreit *et al.* (1989) found that O₃ can induce increased airway responsiveness in asthmatic subjects to O₃, who typically have increased airway responsiveness at baseline. A subsequent study (Jörres *et al.*, 1996) suggested an increase in specific (*i.e.*, allergen-induced) airway reactivity in subjects with allergic asthma, and to a lesser extent in subjects with allergic rhinitis after short-term exposure to higher O₃ levels; other studies reported similar results. According to one study (Folinsbee and Hazucha, 2000), changes in airway responsiveness after O₃ exposure resolve more slowly than changes in FEV₁ or respiratory symptoms. Other studies of repeated exposure to O₃ suggest that changes in airway responsiveness tend to be somewhat less affected by attenuation with consecutive exposures than changes in FEV₁ (EPA, 2006a, section 6.8).

The 2006 Criteria Document (section 6.8) concludes that O₃ exposure is linked with increased airway responsiveness. Both human and animal studies indicate that increased airway responsiveness is not mechanistically associated with inflammation, and does not appear to be strongly associated with initial decrements in lung function or increases in symptoms. As a result of increased airway responsiveness induced by O₃ exposure, human airways may be more susceptible to a variety of stimuli, including antigens, chemicals, and particles. Because asthmatic subjects typically have increased airway responsiveness at baseline, enhanced bronchial response to antigens in asthmatics raises potential public health concerns as they could lead to increased morbidity (*e.g.*, medication usage, school absences, emergency room visits, hospital admissions) or to more persistent alterations in airway

responsiveness (EPA 2006a, p. 8–21). As such, increased airway responsiveness after O₃ exposure represents a plausible link between O₃ exposure and increased hospital admissions.

(c) Respiratory Inflammation and Increased Permeability

Based on evidence from the 1997 review, acute inflammatory responses in the lung have been observed subsequent to 6.6 hour O₃ exposures to the lowest tested level—0.080 ppm—in healthy adults engaged in moderately high exercise (section 6.9 of the 2006 Criteria Document and section 3.3.1.3 of the 2007 Staff Paper). Some of these prior studies suggest that inflammatory responses may be detected in some individuals following O₃ exposures in the absence of O₃-induced pulmonary decrements in those subjects. These studies also demonstrate that short-term exposures to O₃ also can cause increased permeability in the lungs of humans and experimental animals. Inflammatory responses and epithelial permeability have been seen to be independent of spirometric responses. Not only are the newer lung inflammation and increased cellular permeability findings discussed in the 2006 Criteria Document (section 8.4.2) consistent with the 1997 review, but they provide better characterization of the physiological mechanisms by which O₃ causes these effects.

Lung inflammation and increased permeability, which are distinct events controlled by different mechanisms, are two commonly observed effects of O₃ exposure observed in all of the species studied. Increased cellular permeability is a disruption of the lung barrier that leads to leakage of serum proteins, influx of polymorphonuclear leukocytes (neutrophils or PMNs), release of bioactive mediators, and movement of compounds from the airspaces into the blood.

A number of controlled human exposure studies have analyzed bronchoalveolar lavage (BAL) and nasal lavage (NL)¹⁹ fluids and cells for markers of inflammation and lung damage (EPA, 2006a, Annex AX6). Increased lung inflammation is demonstrated by the presence of neutrophils found in BAL fluid in the lungs, which has long been accepted as a hallmark of inflammation. It is apparent, however, that inflammation

within airway tissues may persist beyond the point that inflammatory cells are found in the BAL fluid. Soluble mediators of inflammation, such as cytokines and arachidonic acid metabolites have been measured in the BAL fluid of humans exposed to O₃. In addition to their role in inflammation, many of these compounds have bronchoconstrictive properties and may be involved in increased airway responsiveness following O₃ exposure. An in vitro study of epithelial cells from nonatopic and atopic asthmatics exposed to 0.010 to 0.100 ppm O₃ showed significantly increased permeability compared to cells from normal persons. This indicates a potentially inherent susceptibility of cells from asthmatic individuals for O₃-induced permeability.

In the 1996 Criteria Document, assessment of controlled human exposure studies indicated that a single, acute (1 to 4 hours) O₃ exposure (≥ 0.080 to 0.100 ppm) of subjects engaged in moderate to heavy exercise could induce a number of cellular and biochemical changes suggestive of pulmonary inflammation and lung permeability (EPA, 2006a, p. 8–22). These changes persisted for at least 18 hours. Markers from BAL fluid following both 2-hour and 4-hour O₃ exposures repeated up to 5 days indicate that there is ongoing cellular damage irrespective of attenuation of some cellular inflammatory responses of the airways, pulmonary function, and symptom scores (EPA, 2006a, p. 8–22). Acute airway inflammation was shown in Devlin *et al.* (1990) to occur among adults exposed to 0.080 ppm O₃ for 6.6 hours with exercise. McBride *et al.* (1994) reported that asthmatic subjects were more sensitive than non-asthmatics to upper airway inflammation for O₃ exposures that did not affect pulmonary function (EPA, 2006a, p. 6–33). However, the public health significance of these changes is not entirely clear.

The studies reporting inflammatory responses and markers of lung injury have clearly demonstrated that there is significant variation in response of subjects exposed, especially to 6.6 hours O₃ exposures at 0.080 and 0.100 ppm. To provide some perspective on the public health impact for these effects, the 2007 Staff Paper (section 3.3.1.1.3) notes that one study (Devlin *et al.*, 1991) showed that roughly 10 to 50 percent of the 18 young healthy adult subjects experienced notable increases (*i.e.*, ≥ 2 fold increase) in most of the inflammatory and cellular injury indicators analyzed, associated with 6.6-hour exposures at 0.080 ppm. Similar,

although in some cases higher, fractions of the population of 10 healthy adults tested saw > 2 fold increases associated with 6.6-hour exposures to 0.100 ppm. The authors of this study expressed the view that “susceptible subpopulations such as the very young, elderly, and people with pulmonary impairment or disease may be even more affected” (Devlin *et al.*, 1991).

Since 1996, a substantial number of human exposure studies have been published which have provided important new information on lung inflammation and epithelial permeability. Mudway and Kelly (2004) examined O₃-induced inflammatory responses and epithelial permeability with a meta-analysis of 21 controlled human exposure studies and showed that an influx in neutrophils and protein in healthy subjects is associated with total O₃ dose (product of O₃ concentration, exposure duration, and minute ventilation) (EPA, 2006a, p. 6–34). Results of the analysis suggest that the time course for inflammatory responses (including recruitment of neutrophils and other soluble mediators) is not clearly established, but there is evidence that attenuation profiles for many of these parameters are different (EPA, 2006a, p. 8–22).

The 2006 Criteria Document (chapter 8) concludes that interaction of O₃ with lipid constituents of epithelial lining fluid (ELF) and cell membranes and the induction of oxidative stress is implicated in injury and inflammation. Alterations in the expression of cytokines, chemokines, and adhesion molecules, indicative of an ongoing oxidative stress response, as well as injury repair and regeneration processes, have been reported in animal toxicology and human in vitro studies evaluating biochemical mediators implicated in injury and inflammation. While antioxidants in ELF confer some protection, O₃ reactivity is not eliminated at environmentally relevant exposures (2006 Criteria Document, p. 8–24). Further, antioxidant reactivity with O₃ is both species-specific and dose-dependent.

(d) Increased Susceptibility to Respiratory Infection

As discussed in more detail in the 2006 Criteria Document (sections 5.2.2, 6.9.6, and 8.4.2), short-term exposures to O₃ have been shown to impair physiological defense capabilities in experimental animals by depressing alveolar macrophage (AM) functions and by altering the mucociliary clearance of inhaled particles and microbes resulting in increased susceptibility to respiratory infection.

¹⁹Graham and Koren (1990) compared inflammatory mediators present in NL and BAL fluids of humans exposed to 0.4 ppm O₃ for 2 hours and found similar increases in PMNs in both fluids, suggesting a qualitative correlation between inflammatory changes in the lower airways (BAL) and upper respiratory tract (NL).

Short-term O₃ exposures also interfere with the clearance process by accelerating clearance for low doses and slowing clearance for high doses. Animal toxicological studies have reported that acute O₃ exposures suppress alveolar phagocytosis and immune system functions. Impairment of host defenses and subsequent increased susceptibility to bacterial lung infection in laboratory animals has been induced by short-term exposures to O₃ levels as low as 0.080 ppm.

A single controlled human exposure study reviewed in the 1996 Criteria Document (p. 8–26) reported that exposure to 0.080 to 0.100 ppm O₃ for 6.6 hours (with moderate exercise) induced decrements in the ability of AMs to phagocytose microorganisms. Integrating the recent animal study results with human exposure evidence available in the 1996 Criteria Document, the 2006 Criteria Document concludes that available evidence indicates that short-term O₃ exposures have the potential to impair host defenses in humans, primarily by interfering with AM function. Any impairment in AM function may lead to decreased clearance of microorganisms or nonviable particles. Compromised AM functions in asthmatics may increase their susceptibility to other O₃ effects, the effects of particles, and respiratory infections (EPA, 2006a, p. 8–26).

(e) Morphological Effects

The 1996 Criteria Document found that short-term O₃ exposures cause similar alterations in lung morphology in all laboratory animal species studied, including primates. As discussed in the 2007 Staff Paper (section 3.3.1.1.5), cells in the centriacinar region (CAR) of the lung (the segment between the last conducting airway and the gas exchange region) have been recognized as a primary target of O₃-induced damage (epithelial cell necrosis and remodeling of respiratory bronchioles), possibly because epithelium in this region receives the greatest dose of O₃ delivered to the lower respiratory tract. Following chronic O₃ exposure, structural changes have been observed in the CAR, the region typically affected in most chronic airway diseases of the human lung (EPA, 2006a, p. 8–24).

Ciliated cells in the nasal cavity and airways, as well as Type I cells in the gas-exchange region, are also identified as targets. While short-term O₃ exposures can cause epithelial cell proliferation and fibrotic changes in the CAR, these changes appear to be transient with recovery occurring after exposure, depending on species and O₃ dose. The potential impacts of repeated

short-term and chronic morphological effects of O₃ exposure are discussed below in the section on effects from long-term exposures. Long-term or prolonged exposure has been found to cause chronic lesions similar to early lesions found in individuals with respiratory bronchiolitis, which have the potential to progress to fibrotic lung disease (2006 Criteria Document, p. 8–25).

Recent studies continue to show that short-term and sub-chronic exposures to O₃ cause similar alterations in lung structure in a variety of experimental animal species. For example, a series of new studies that used infant rhesus monkeys and simulated seasonal ambient exposure (0.5 ppm 8 hours/day for 5 days, every 14 days for 11 episodes) reported remodeling in the distal airways; abnormalities in tracheal basement membrane; eosinophil accumulation in conducting airways; and decrements in airway innervation (2006 Criteria Document, p. 8–25). Based on evidence from animal toxicological studies, short-term and sub-chronic exposures to O₃ can cause morphological changes in the respiratory systems, particularly in the CAR, of a number of laboratory animal species (EPA, 2006a, section 5.2.4).

(f) Emergency Department Visits/Hospital Admissions for Respiratory Causes

Increased summertime emergency department visits and hospital admissions for respiratory causes have been associated with ambient exposures to O₃. As discussed in section 3.3.1.1.6 of the 2007 Staff Paper, numerous studies conducted in various locations in the U.S. and Canada consistently have shown a relationship between ambient O₃ levels and increased incidence of emergency department visits and hospital admissions for respiratory causes, even after controlling for modifying factors, such as weather and copollutants. Such associations between elevated ambient O₃ during summer months and increased hospital admissions have a plausible biological basis in the human and animal evidence of functional, symptomatic, and physiologic effects discussed above and in the increased susceptibility to respiratory infections observed in laboratory animals.

In the 1997 review of the O₃ NAAQS, the Criteria Document evaluated emergency department visits and hospital admissions as possible outcomes following exposure to O₃ (EPA, 2006a, section 7.3). The evidence was limited for emergency department visits, but results of several studies

generally indicated that short-term exposures to O₃ were associated with respiratory emergency department visits. The strongest and most consistent evidence, at both lower levels (*i.e.*, below 0.120 ppm 1-hour max O₃) and at higher levels (above 0.120 ppm 1-hour max O₃), was found in the group of studies which investigated summertime²⁰ daily hospital admissions for respiratory causes in different eastern North American cities. These studies consistently demonstrated that ambient O₃ levels were associated with increased hospital admissions and accounted for about one to three excess respiratory hospital admissions per million persons with each 0.100 ppm increase in 1-hour max O₃, after adjustment for possible confounding effects of temperature and copollutants. Overall, the 1996 Criteria Document concluded that there was strong evidence that ambient O₃ exposures can cause significant exacerbations of preexisting respiratory disease in the general public. Excess respiratory-related hospital admissions associated with O₃ exposures for the New York City area (based on Thurston *et al.*, 1992) were included in the quantitative risk assessment in the 1997 review and are included in the current assessment along with estimates for respiratory-related hospital admissions in Cleveland, Detroit, and Los Angeles based on more recent studies (2007 Staff Paper, chapter 5). Significant uncertainties and the difficulty of obtaining reliable baseline incidence numbers resulted in emergency department visits not being used in the quantitative risk assessment in either the 1997 or the 2008 O₃ NAAQS review.

In the past decade, a number of studies have examined the temporal pattern associations between O₃ exposures and emergency department visits for respiratory causes (EPA, 2006a, section 7.3.2). These studies are summarized in the 2006 Criteria Document (chapter 7 Annex) and some are shown in Figure 1 (in section II.A.3). Respiratory causes for emergency department visits include asthma, bronchitis, emphysema, pneumonia, and other upper and lower respiratory infections, such as influenza, but asthma visits typically dominate the daily incidence counts. Most studies report positive associations with O₃. Among studies with adequate controls for seasonal patterns, many reported at least one significant positive association involving O₃.

²⁰ Discussion of the reasons for focusing on warm season studies is found in the section 2.A.3.a below.

In reviewing evidence for associations between emergency department visits for asthma and short-term O₃ exposures, the 2006 Criteria Document (Figure 7–8, p. 7–68) notes that in general, O₃ effect estimates from summer only analyses tended to be positive and larger compared to results from cool season or all year analyses. Several of the studies reported significant associations between O₃ concentrations and emergency department visits for respiratory causes, in particular asthma. However, inconsistencies were observed which were at least partially attributable to differences in model specifications and analysis approach among various studies. For example, ambient O₃ concentrations, length of the study period, and statistical methods used to control confounding by seasonal patterns and copollutants appear to affect the observed O₃ effect on emergency department visits.

Hospital admissions studies focus specifically on unscheduled admissions because unscheduled hospital admissions occur in response to unanticipated disease exacerbations and are more likely than scheduled admissions to be affected by variations in environmental factors, such as daily O₃ levels. Results of a fairly large number of these studies published during the past decade are summarized in 2006 Criteria Document (chapter 7 Annex), and results of U.S. and Canadian studies are shown in Figure 1 below (in section II.A.3). As a group, these hospital admissions studies tend to be larger geographically and temporally than the emergency department visit studies and provide results that are generally more consistent. The strongest associations of respiratory hospital admissions with O₃ concentrations were observed using short lag periods, in particular for a 0-day lag (same day exposure) and a 1-day lag (previous day exposure). Most studies in the United States and Canada indicated positive, statistically significant associations between ambient O₃ concentrations and respiratory hospital admissions in the warm season. However, not all studies found a statistically significant relationship with O₃, possibly because of very low ambient O₃ levels. Analyses for confounding using multipollutant regression models suggest that copollutants generally do not confound the association between O₃ and respiratory hospitalizations. Ozone effect estimates were robust to PM adjustment in all-year and warm-season only data.

Overall, the 2006 Criteria Document concludes that positive and robust

associations were found between ambient O₃ concentrations and various respiratory disease hospitalization outcomes, when focusing particularly on results of warm-season analyses. Recent studies also generally indicate a positive association between O₃ concentrations and emergency department visits for asthma during the warm season (EPA, 2006a, p. 7–175). These positive and robust associations are supported by the controlled human exposure, animal toxicological, and epidemiological evidence for lung function decrements, increased respiratory symptoms, airway inflammation, and increased airway responsiveness. Taken together, the overall evidence supports a causal relationship between acute ambient O₃ exposures and increased respiratory morbidity outcomes resulting in increased emergency department visits and hospitalizations during the warm season (EPA, 2006a, p. 8–77).

ii. Effects on the Respiratory System of Long-Term O₃ Exposures

The 1996 Criteria Document concluded that there was insufficient evidence from the limited number of studies to determine whether long-term O₃ exposures resulted in chronic health effects at ambient levels observed in the U.S. However, the aggregate evidence suggested that O₃ exposure, along with other environmental factors, could be responsible for health effects in exposed populations. Animal toxicological studies carried out in the 1980's and 1990's demonstrated that long-term exposures can result in a variety of morphological effects, including permanent changes in the small airways of the lungs, including remodeling of the distal airways and CAR and deposition of collagen, possibly representing fibrotic changes. These changes result from the damage and repair processes that occur with repeated exposure. Fibrotic changes were also found to persist after months of exposure providing a potential pathophysiological basis for changes in airway function observed in children in some recent epidemiological studies. It appears that variable seasonal ambient patterns of exposure may be of greater concern than continuous daily exposures.

Several studies published since 1996 have investigated lung function changes over seasonal time periods (EPA, 2006a, section 7.5.3). The 2006 Criteria Document (p. 7–114) summarizes these studies which collectively indicate that seasonal O₃ exposure is associated with smaller growth-related increases in lung function in children than they would

have experienced living in areas with lower O₃ levels. There is some limited evidence that seasonal O₃ also may affect lung function growth in young adults, although the uncertainty about the role of copollutants makes it difficult to attribute the effects to O₃ alone.

Lung capacity grows during childhood and adolescence as body size increases, reaches a maximum during the twenties, and then begins to decline steadily and progressively with age. Long-term exposure to air pollution has long been thought to contribute to slower growth in lung capacity, diminished maximally attained capacity, and/or more rapid decline in lung capacity with age (EPA, 2006a, section 7.5.4). Toxicological findings evaluated in the 1996 Criteria Document demonstrated that repeated daily exposure of rats to an episodic profile of O₃ caused small, but significant, decrements in growth-related lung function that were consistent with early indicators of focal fibrogenesis in the proximal alveolar region, without overt fibrosis. Because O₃ at sufficient concentrations is a strong respiratory irritant and has been shown to cause inflammation and restructuring of the respiratory airways, it is plausible that long-term O₃ exposures might have a negative impact on baseline lung function, particularly during childhood when these exposures might be associated with long-term risks.

Several epidemiological studies published since 1996 have examined the relationship between lung function development and long-term O₃ exposure. The most extensive and robust study of respiratory effects in relation to long-term air pollution exposures among children in the U.S. is the Children's Health Study carried out in 12 communities of southern California starting in 1993. One analysis (Peters *et al.*, 1999a) examined the relationship between long-term O₃ exposures and self-reports of respiratory symptoms and asthma in a cross sectional analysis and found a limited relationship between outcomes of current asthma, bronchitis, cough and wheeze and a 0.040 ppm increase in 1-hour max O₃ (EPA, 2006a, p. 7–115). Another analysis (Peters *et al.*, 1999b) examined the relationship between lung function at baseline and levels of air pollution in the community. They reported evidence that annual mean O₃ levels were associated with decreases in FVC, FEV₁, PEF and forced expiratory flow (FEF_{25–75}) (the latter two being statistically significant) among females but not males. In a separate analysis (Gauderman *et al.*, 2000) of 4th, 7th, and

10th grade students, a longitudinal analysis of lung function development over four years found no association with O₃ exposure. The Children's Health Study enrolled a second cohort of more than 1500 fourth graders in 1996 (Gauderman *et al.*, 2002). While the strongest associations with negative lung function growth were observed with acid vapors in this cohort, children from communities with higher 4-year average O₃ levels also experienced smaller increases in various lung function parameters. The strongest relationship with O₃ was with PEF. Specifically, children from the least-polluted community had a small but statistically significant increase in PEF as compared to those from the most-polluted communities. In two-pollutant models, only 8-hour average O₃ and NO₂ were significant joint predictors of FEV₁ and maximal midexpiratory flow (MMEF). Although results from the second cohort of children are supportive of a weak association, the definitive 8-year follow-up analysis of the first cohort (Gauderman *et al.*, 2004a) provides little evidence that long-term exposure to ambient O₃ at current levels is associated with significant deficits in the growth rate of lung function in children. Avol *et al.* (2001) examined children who had moved away from participating communities in southern California to other states with improved air quality. They found that a negative, but not statistically significant, association was observed between O₃ and lung function parameters. Collectively, the results of these reports from the children's health cohorts provide little evidence to support an impact of long-term O₃ exposures on lung function development.

Evidence for a significant relationship between long-term O₃ exposures and decrements in maximally attained lung function was reported in a nationwide study of first year Yale students (Kinney *et al.*, 1998; Galizia and Kinney, 1999) (EPA, 2006a, p. 7–120). Males had much larger effect estimates than females, which might reflect higher outdoor activity levels and correspondingly higher O₃ exposures during childhood. A similar study of college freshmen at University of California at Berkeley also reported significant effects of long-term O₃ exposures on lung function (Künzli *et al.*, 1997; Tager *et al.*, 1998). In a comparison of students whose city of origin was either Los Angeles or San Francisco, long-term O₃ exposures were associated with significant changes in mid- and end-expiratory flow measures, which could be considered early

indicators for pathologic changes that might progress to COPD.

There have been a few studies that investigated associations between long-term O₃ exposures and the onset of new cases of asthma (EPA, 2006a, section 7.5.6). The Adventist Health and Smog (AHSMOG) study cohort of about 4,000 was drawn from nonsmoking, non-Hispanic white adult Seventh Day Adventists living in California (Greer *et al.*, 1993; McDonnell *et al.*, 1999). During the ten-year follow-up in 1987, a statistically significant increased relative risk of asthma development was observed in males, compared to a nonsignificant relative risk in females (Greer *et al.*, 1993). In the 15-year follow-up in 1992, it was reported that for males, there was a statistically significant increased relative risk of developing asthma associated with 8-hour average O₃ exposures, but there was no evidence of an association in females. Consistency of results in the two studies with different follow-up times provides supportive evidence of the potential for an association between long-term O₃ exposure and asthma incidence in adult males; however, representativeness of this cohort to the general U.S. population may be limited (EPA, 2006a, p. 7–125).

In a similar study (McConnell *et al.*, 2002) of incident asthma among children (ages 9 to 16 at enrollment), annual surveys of 3,535 children initially without asthma were used to identify new-onset asthma cases as part of the Children's Health Study. Six high-O₃ and six low-O₃ communities were identified where the children resided. There were 265 children who reported new-onset asthma during the follow-up period. Although asthma risk was no higher for all residents of the six high-O₃ communities versus the six low-O₃ communities, asthma risk was 3.3 times greater for children who played three or more sports as compared with children who played no sports within the high-O₃ communities. This association was absent in the communities with lower O₃ concentrations. No other pollutants were found to be associated with new-onset asthma (EPA, 2006a, p. 7–125). Playing sports may result in extended outdoor activity and exposure occurring during periods when O₃ levels are higher. It should be noted, however, that the results of the Children's Health Study were based on a small number of new-onset asthma cases among children who played three or more sports. Future replication of these findings in other cohorts would help determine whether a causal interpretation is appropriate.

In animal toxicology studies, the progression of morphological effects reported during and after a chronic exposure in the range of 0.50 to 1.00 ppm O₃ (well above current ambient levels) is complex, with inflammation peaking over the first few days of exposure, then dropping, then plateauing, and finally, largely disappearing (EPA, 2006a, section 5.2.4.4). By contrast, fibrotic changes in the tissue increase very slowly over months of exposure, and, after exposure ceases, the changes sometimes persist or increase. Epithelial hyperplasia peaks soon after the inflammatory response but is usually maintained in both the nose and lungs with continuous exposure; it also does not return to pre-exposure levels after the end of exposure. Patterns of exposure in this same concentration range determine effects, with 18 months of daily exposure, causing less morphologic damage than exposures on alternating months. This is important as environmental O₃ exposure is typically seasonal. Long-term studies by Plopper and colleagues (Evans *et al.*, 2003; Schelegle *et al.*, 2003; Chen *et al.*, 2003; Plopper and Fanucchi, 2000) investigated infant rhesus monkeys exposed to simulated, seasonal O₃ and demonstrated: (1) Remodeling in the distal airways, (2) abnormalities in tracheal basement membrane; (3) eosinophil accumulation in conducting airways; and (4) decrements in airway innervation (EPA, 2006a, p. 5–45). These findings provide additional information regarding possible injury-repair processes occurring with long-term O₃ exposures suggesting that these processes are only partially reversible and may progress following cessation of O₃ exposure. Further, these processes may lead to nonreversible structural damage to lung tissue; however, there is still too much uncertainty to characterize the significance of these findings to human exposure profiles and effect levels (EPA, 2006a, p. 8–25).

In summary, in the past decade, important new longitudinal studies have examined the effect of chronic O₃ exposure on respiratory health outcomes. Limited evidence from recent long-term morbidity studies have suggested in some cases that chronic exposure to O₃ may be associated with seasonal declines in lung function or reduced lung function development, increases in inflammation, and development of asthma in children and adults. Seasonal decrements or smaller increases in lung function measures have been reported in several studies; however, the extent to which these

changes are transient remains uncertain. While there is supportive evidence from animal studies involving effects from chronic exposures, large uncertainties still remain as to whether current ambient levels and exposure patterns might cause these same effects in human populations. The 2006 Criteria Document concludes that epidemiological studies of new asthma development and longer-term lung function declines remain inconclusive at present (EPA, 2006a, p. 7–134).

iii. Effects on the Cardiovascular System of O₃ Exposure

At the time of the 1997 review, the possibility of O₃-induced cardiovascular effects was largely unrecognized. Since then, a very limited body of evidence from animal, controlled human exposure, and epidemiologic studies has emerged that provides evidence for some potential plausible mechanisms for how O₃ exposures might exert cardiovascular system effects, however further research is needed to substantiate these potential mechanisms. Possible mechanisms may involve O₃-induced secretions of vasoconstrictive substances and/or effects on neuronal reflexes that may result in increased arterial blood pressure and/or altered electrophysiologic control of heart rate or rhythm. Some animal toxicology studies have shown O₃-induced decreases in heart rate, mean arterial pressure, and core temperature. One controlled human exposure study that evaluated effects of O₃ exposure on cardiovascular health outcomes found no significant O₃-induced differences in ECG or blood pressure in healthy or hypertensive subjects but did observe a significant O₃-induced increase the alveolar-to-arterial PO₂ gradient and heart rate in both groups resulting in an overall increase in myocardial work and impairment in pulmonary gas exchange (Gong *et al.*, 1998). In another controlled human exposure study, inhalation of a mixture of PM_{2.5} and O₃ by healthy subjects increased brachial artery vasoconstriction and reactivity (Brook *et al.*, 2002).

The evidence from a few animal studies also includes potential direct effects such as O₃-induced release from lung epithelial cells of platelet activating factor (PAF) that may contribute to blood clot formation that would have the potential to increase the risk of serious cardiovascular outcomes (*e.g.*, heart attack, stroke, mortality). Also, interactions of O₃ with surfactant components in epithelial lining fluid of the lung may result in production of oxysterols and reactive oxygen species

that may exhibit PAF-like activity contributing to clotting and also may exert cytotoxic effects on lung and heart muscle cells.

Epidemiological panel and field studies that examined associations between O₃ and various cardiac physiologic endpoints have yielded limited evidence suggestive of a potential association between acute O₃ exposure and altered heart rate variability (HRV), ventricular arrhythmias, and incidence of heart attacks (myocardial infarction or MI). A number of epidemiological studies have also reported associations between short-term exposures and hospitalization for cardiovascular diseases. As shown in Figure 7–13 of the 2006 Criteria Document, many of the studies reported negative or inconsistent associations. Some other studies, especially those that examined the relationship when O₃ exposures were higher, have found robust positive associations between O₃ and cardiovascular hospital admissions (EPA, 2006a, p. 7–82). For example, one study reported a positive association between O₃ and cardiovascular hospital admissions in Toronto, Canada in a summer-only analysis (Burnett *et al.*, 1997b). The results were robust to adjustment for various PM indices, whereas the PM effects diminished when adjusted for gaseous pollutants. Other studies stratified their analysis by temperature (*i.e.*, by warm days versus cool days). Several analyses using warm season days consistently produced positive associations.

The epidemiologic evidence for cardiovascular morbidity is much weaker than for respiratory morbidity, with only one of several U.S. and Canadian studies showing statistically significant positive associations of cardiovascular hospitalizations with warm-season O₃ concentrations. Most of the available European and Australian studies, all of which conducted all-year O₃ analyses, did not find an association between short-term O₃ concentrations and cardiovascular hospitalizations. Overall, the currently available evidence is inconclusive regarding an association between cardiovascular hospital admissions and ambient O₃ exposure (EPA, 2006a, p. 7–83).

In summary, based on the evidence from animal toxicology, controlled human exposure, and epidemiological studies, from the 2006 Criteria Document (p. 8–77) concludes that this generally limited body of evidence is suggestive that O₃ can directly and/or indirectly contribute to cardiovascular-related morbidity, but that much needs to be done to more fully integrate links

between ambient O₃ exposures and adverse cardiovascular outcomes.

b. Mortality

i. Mortality and Short-term O₃ Exposure

The 1996 Criteria Document concluded that an association between daily mortality and O₃ concentration for areas with high O₃ levels (*e.g.*, Los Angeles) was suggested. However, due to a very limited number of studies available at that time, there was insufficient evidence to conclude that the observed association was likely causal.

The 2006 Criteria Document included results from numerous epidemiological analyses of the relationship between O₃ and mortality. Additional single city analyses have also been conducted since 1996, however, the most pivotal studies in EPA's (and CASAC's) finding of increased support for the relationship between premature mortality and O₃ is in part related to differences in study design—limiting analyses to warm seasons, better control for copollutants, particularly PM, and use of multicity designs (both time series and meta-analytic designs). Key findings are available from multicity time-series studies that report associations between O₃ and mortality. These studies include analyses using data from 90 U.S. cities in the National Mortality, Morbidity and Air Pollution (NMMAPS) study (Dominici *et al.*, 2003) and from 95 U.S. communities in an extension to the NMMAPS analyses (Bell *et al.*, 2004).

The original 90-city NMMAPS analysis, with data from 1987 to 1994, was primarily focused on investigating effects of PM₁₀ on mortality. A significant association was reported between mortality and 24-hour average O₃ concentrations in analyses using all available data as well as in the warm season only analyses (Dominici *et al.*, 2003). The estimate using all available data was about half that for the summer-only data at a lag of 1-day. The extended NMMAPS analysis included data from 95 U.S. cities and included an additional 6 years of data, from 1987–2000 (Bell *et al.*, 2004). Significant associations were reported between O₃ and mortality in analyses using all available data. The effect estimate for increased mortality was approximately 0.5 percent per 0.020 ppm change in 24-hour average O₃ measured on the same day, and approximately 1.04 percent per 0.020 ppm change in 24-hour average O₃ in a 7-day distributed lag model (EPA, 2006a, p. 7–88). In analyses using only data from the warm season, the results were not significantly different from the full-year results. The authors also report

that O₃-mortality associations were robust to adjustment for PM (EPA, 2006a, p. 7–100). Using a subset of the NMMAPS data set, Huang *et al.* (2005) focused on associations between cardiopulmonary mortality and O₃ exposure (24-hour average) during the summer season only. The authors report an approximate 1.47 percent increase per 0.020 ppm change in O₃ concentration measured on the same day and an approximate 2.52 percent increase per 0.020 ppm change in O₃ concentration using a 7-day distributed lag model. These findings suggest that the effect of O₃ on mortality is immediate but also persists for several days.

As discussed below in section II.A.3.a, confounding by weather, especially temperature, is complicated by the fact that higher temperatures are associated with the increased photochemical activities that are important for O₃ formation. Using a case-crossover study design, Schwartz (2005) assessed associations between daily maximum concentrations and mortality, matching case and control periods by temperature, and using data only from the warm season. The reported effect estimate of approximately 0.92 percent change in mortality per 0.040 ppm O₃ (1-hour maximum) was similar to time-series analysis results with adjustment for temperature (approximately 0.76 percent per 0.040 ppm O₃), suggesting that associations between O₃ and mortality were robust to the different adjustment methods for temperature.

An initial publication from APHEA, a European multicity study, reported statistically significant associations between daily maximum O₃ concentrations and mortality in four cities in a full year analysis (Toulomi *et al.*, 1997). An extended analysis was done using data from 23 cities throughout Europe (Gryparis *et al.*, 2004). In this report, a positive but not statistically significant association was found between mortality and 1-hour daily maximum O₃ in a full year analysis. Gryparis *et al.* (2004) noted that there was a considerable seasonal difference in the O₃ effect on mortality; thus, the small effect for the all-year data might be attributable to inadequate adjustment for confounding by seasonality. Focusing on analyses using summer measurements, the authors report statistically significant associations with total mortality, cardiovascular mortality and respiratory mortality (EPA, 2006a, p. 7–93, 7–99).

Numerous single-city analyses have also reported associations between mortality and short-term O₃ exposure,

especially for those analyses using warm season data. As shown in Figure 7–21 of the 2006 Criteria Document, the results of recent publications show a pattern of positive, often statistically significant associations between short-term O₃ exposure and mortality during the warm season. In considering results from year-round analyses, there remains a pattern of positive results but the findings are less consistent. In most single-city analyses, effect estimates were not substantially changed with adjustment for PM (EPA, 2006a, Figure 7–22).

In addition, several meta-analyses have been conducted on the relationship between O₃ and mortality. As described in section 7.4.4 of the 2006 Criteria Document, these analyses reported fairly consistent and positive combined effect estimates ranging from approximately 1.5 to 2.5 percent increase in mortality for a standardized change in O₃ (EPA, 2006a, Figure 7–20). Three recent meta-analyses evaluated potential sources of heterogeneity in O₃-mortality associations (Bell *et al.*, 2005; Ito *et al.*, 2005; Levy *et al.*, 2005). The 2006 Criteria Document (p. 7–96) observes common findings across all three analyses, in that all reported that effect estimates were larger in warm season analyses, reanalysis of results using default convergence criteria in generalized additive models (GAM) did not change the effect estimates, and there was no strong evidence of confounding by PM. Bell *et al.* (2005) and Ito *et al.* (2005) both provided suggestive evidence of publication bias, but O₃-mortality associations remained after accounting for that potential bias. The 2006 Criteria Document concludes that the “positive O₃ effects estimates, along with the sensitivity analyses in these three meta-analyses, provide evidence of a robust association between ambient O₃ and mortality” (EPA, 2006a, p. 7–97).

Most of the single-pollutant model estimates from single-city studies range from 0.5 to 5 percent excess deaths per standardized increments. Corresponding summary estimates in large U.S. multicity studies ranged between 0.5 to 1 percent with some studies noting heterogeneity across cities and studies (EPA, 2006a, p. 7–110).

Finally, from those studies that included assessment of associations with specific causes of death, it appears that effect estimates for associations with cardiovascular mortality are larger than those for total mortality. The meta-analysis by Bell *et al.* (2005) observed a slightly larger effect estimate for cardiovascular mortality compared to mortality from all causes. The effect

estimate for respiratory mortality was approximately one-half that of cardiovascular mortality in the meta-analysis. However, other studies have observed larger effect estimates for respiratory mortality compared to cardiovascular mortality. The apparent inconsistency regarding the effect size of O₃-related respiratory mortality may be due to reduced statistical power in this subcategory of mortality (EPA, 2006a, p. 7–108).

In summary, many single- and multicity studies observed positive associations of ambient O₃ concentrations with total nonaccidental and cardiopulmonary mortality. The 2006 Criteria Document finds that the results from U.S. multicity time-series studies provide the strongest evidence to date for O₃ effects on acute mortality. Recent meta-analyses also indicate positive risk estimates that are unlikely to be confounded by PM; however, future work is needed to better understand the influence of model specifications on the risk coefficient (EPA, 2006a, p. 7–175). A meta-analysis that examined specific causes of mortality found that the cardiovascular mortality risk estimates were higher than those for total mortality. For cardiovascular mortality, the 2006 Criteria Document (Figure 7–25, p. 7–106) suggests that effect estimates are consistently positive and more likely to be larger and statistically significant in warm season analyses. The findings regarding the effect size for respiratory mortality have been less consistent, possibly because of lower statistical power in this subcategory of mortality. The 2006 Criteria Document (p. 8–78) concludes that these findings are highly suggestive that short-term O₃ exposure directly or indirectly contribute to non-accidental and cardiopulmonary-related mortality, but additional research is needed to more fully establish underlying mechanisms by which such effects occur.²¹

²¹ In commenting on the Criteria Document, the CASAC Ozone Panel raised questions about the implications of these time-series results in a policy context, emphasizing that “* * * while the time-series study design is a powerful tool to detect very small effects that could not be detected using other designs, it is also a blunt tool” (Henderson, 2006b). They note that “* * * not only is the interpretation of these associations complicated by the fact that the day-to-day variation in concentrations of these pollutants is, to a varying degree, determined by meteorology, the pollutants are often part of a large and highly correlated mix of pollutants, only a very few of which are measured” (Henderson, 2006b). Even with these uncertainties, the CASAC Ozone Panel, in its review of the Staff Paper, found “* * * premature total non-accidental and cardiorespiratory mortality for inclusion in the quantitative risk assessment to be appropriate.” (Henderson, 2006b)

ii. Mortality and Long-Term O₃ Exposure

Little evidence was available in the 1997 review on the potential for associations between mortality and long-term exposure to O₃. In the Harvard Six City prospective cohort analysis, the authors report that mortality was not associated with long-term exposure to O₃ (Dockery *et al.*, 1993). The authors note that the range of O₃ concentrations across the six cities was small, which may have limited the power of the study to detect associations between mortality and O₃ levels (EPA, 2006a, p. 7–127).

As discussed in section 7.5.8 of the 2006 Criteria Document, in this review there are results available from three prospective cohort studies: the American Cancer Society (ACS) study (Pope *et al.*, 2002), the Adventist Health and Smog (AHSMOG) study (Beeson *et al.*, 1998; Abbey *et al.*, 1999), and the U.S. Veterans Cohort study (Lipfert *et al.*, 2000, 2003). In addition, a major reanalysis report includes evaluation of data from the Harvard Six City cohort study (Krewski *et al.*, 2000).²² This reanalysis also includes additional evaluation of data from the initial ACS cohort study report that had only reported results of associations between mortality and long-term exposure to fine particles and sulfates (Pope *et al.*, 1995). This reanalysis was discussed in the 2007 Staff Paper (section 3.3.2.2) but not in the 2006 Criteria Document.

In this reanalysis of data from the previous Harvard Six City prospective cohort study, the investigators replicated and validated the findings of the original studies, and the report included additional quantitative results beyond those available in the original report (Krewski *et al.*, 2000). In the reanalysis of data from the Harvard Six Cities study, the effect estimate for the association between long-term O₃ concentrations and mortality was negative and nearly statistically significant (relative risk = 0.87, 95 percent CI: 0.76, 1.00).

The ACS study is based on health data from a large prospective cohort of approximately 500,000 adults and air quality data from about 150 U.S. cities. The initial report (Pope *et al.*, 1995) focused on associations with fine particles and sulfates, for which significant associations had been reported in the earlier Harvard Six Cities study (Dockery *et al.*, 1993). As part of the major reanalysis of these

data, results for associations with other air pollutants were also reported, and the authors report that no significant associations were found between O₃ and all-cause mortality. However, a significant association was reported for cardiopulmonary mortality in the warm season (Krewski *et al.*, 2000). The ACS II study (Pope *et al.*, 2002) reported results of associations with an extended data base; the mortality records for the cohort had been updated to include 16 years of follow-up (compared with 8 years in the first report) and more recent air quality data were included in the analyses. Similar to the earlier reanalysis, a marginally significant association was observed between long-term exposure to O₃ and cardiopulmonary mortality in the warm season. No other associations with mortality were observed in both the full-year and warm season analyses.

The Adventist Health and Smog (AHSMOG) cohort includes about 6,000 adults living in California. In two studies from this cohort, a significant association has been reported between long-term O₃ exposure and increased risk of lung cancer mortality among males only (Beeson *et al.*, 1998; Abbey *et al.*, 1999). No significant associations were reported between long-term O₃ exposure and mortality from all causes or cardiopulmonary causes. Due to the small numbers of lung cancer deaths (12 for males, 18 for females) and the precision of the effect estimate (*i.e.*, the wide confidence intervals), the 2006 Criteria Document (p. 7–130) discussed concerns about the plausibility of the reported association with lung cancer.

The U.S. Veterans Cohort study (Lipfert *et al.*, 2000, 2003) of approximately 50,000 middle-aged males diagnosed with hypertension, reported some positive associations between mortality and peak O₃ exposures (95th percentile level for several years of data). The study included numerous analyses using subsets of exposure and mortality follow-up periods which spanned the years 1960 to 1996. In the results of analyses using deaths and O₃ exposure estimates concurrently across the study period, there were positive, statistically significant associations between peak O₃ and mortality (EPA, 2006a, p. 7–129).

Overall, the 2006 Criteria Document (p. 7–130) concludes that consistent associations have not been reported between long-term O₃ exposure and all-cause, cardiopulmonary or lung cancer mortality.

c. Role of Ground-Level O₃ in Solar Radiation-Related Human Health Effects

Beyond the direct health effects attributable to inhalation exposure to O₃ in the ambient air discussed above, the 2006 Criteria Document also assesses potential indirect effects related to the presence of O₃ in the ambient air by considering the role of ground-level O₃ in mediating human health effects that may be directly attributable to exposure to solar ultraviolet radiation (UV-B). The 2006 Criteria Document (chapter 10) focuses this assessment on three key factors, including those factors that govern (1) UV-B radiation flux at the earth's surface, (2) human exposure to UV-B radiation, and (3) human health effects due to UV-B radiation. In so doing, the 2006 Criteria Document provides a thorough analysis of the current understanding of the relationship between reducing ground-level O₃ concentrations and the potential impact these reductions might have on increasing UV-B surface fluxes and indirectly contributing to UV-B related health effects.

There are many factors that influence UV-B radiation penetration to the earth's surface, including latitude, altitude, cloud cover, surface albedo, PM concentration and composition, and gas phase pollution. Of these, only latitude and altitude can be defined with small uncertainty in any effort to assess the changes in UV-B flux that may be attributable to any changes in tropospheric O₃ as a result of any revision to the O₃ NAAQS. Such an assessment of UV-B related health effects would also need to take into account human habits, such as outdoor activities (including age- and occupation-related exposure patterns), dress and skin care to adequately estimate UV-B exposure levels. However, little is known about the impact of these factors on individual exposure to UV-B.

Moreover, detailed information does not exist regarding other factors that are relevant to assessing changes in disease incidence, including: Type (*e.g.*, peak or cumulative) and time period (*e.g.*, childhood, lifetime, current) of exposures related to various adverse health outcomes (*e.g.*, damage to the skin, including skin cancer; damage to the eye, such as cataracts; and immune system suppression); wavelength dependency of biological responses; and interindividual variability in UV-B resistance to such health outcomes. Beyond these well recognized adverse health effects associated with various wavelengths of UV radiation, the 2006 Criteria Document (section 10.2.3.6) also

²² This reanalysis report and the original prospective cohort study findings are discussed in more detail in section 8.2.3 of the *Air Quality Criteria for Particulate Matter* (EPA, 2004).

discusses protective effects of UV-B radiation. Recent reports indicate the necessity of UV-B in producing vitamin D. Vitamin D deficiency can cause metabolic bone disease among children and adults, and may also increase the risk of many common chronic diseases (e.g., type I diabetes and rheumatoid arthritis) as well as the risk of various types of cancers. Thus, the 2006 Criteria Document concludes that any assessment that attempts to quantify the consequences of increased UV-B exposure on humans due to reduced ground-level O₃ must include consideration of both negative and positive effects. However, as with other impacts of UV-B on human health, this beneficial effect of UV-B radiation has not been studied in sufficient detail to allow for a credible health benefits or risk assessment. In conclusion, the effect of changes in surface-level O₃ concentrations on UV-B-induced health outcomes cannot yet be critically assessed within reasonable uncertainty (2006 Criteria Document, p. 10–36).

The Agency last considered indirect effects of O₃ in the ambient air in its 2003 final response to a remand of the Agency's 1997 decision to revise the O₃ NAAQS. In so doing, based on the available information in the 1997 review, EPA determined that the information linking (a) changes in patterns of ground-level O₃ concentrations likely to occur as a result of programs implemented to attain the 1997 O₃ NAAQS to (b) changes in relevant exposures to UV-B radiation of concern to public health was too uncertain at that time to warrant any relaxation in the level of public health protection previously determined to be requisite to protect against the demonstrated direct adverse respiratory effects of exposure to O₃ in the ambient air (68 FR 614). At that time, the more recent information on protective effects of UV-B radiation was not available, such that only adverse UV-B-related effects could be considered. Taking into consideration the more recent information available for the 2008 review, the 2006 Criteria Document and 2007 Staff Paper conclude that the effect of changes in ground-level O₃ concentrations, likely to occur as a result of revising the O₃ NAAQS, on UV-B-induced health outcomes, including whether these changes would ultimately result in increased or decreased incidence of UV-B-related diseases, cannot yet be critically assessed.

3. Interpretation and Integration of Health Evidence

As discussed below, in assessing the health evidence, the 2006 Criteria Document integrates findings from experimental (e.g., toxicological, dosimetric and controlled human exposure) and epidemiological studies, to make judgments about the extent to which causal inferences can be made about observed associations between health endpoints and exposure to O₃. In evaluating the evidence from epidemiological studies, the EPA focuses on well-recognized criteria, including: The strength of reported associations, including the magnitude and precision of reported effect estimates and their statistical significance; the robustness of reported associations, or stability in the effect estimates after considering factors such as alternative models and model specification, potential confounding by co-pollutants, and issues related to the consequences of exposure measurement error; potential aggregation bias in pooling data; and the consistency of the effects associations as observed by looking across results of multiple- and single-city studies conducted by different investigators in different places and times. Consideration is also given to evaluating concentration-response relationships observed in epidemiological studies to inform judgments about the potential for threshold levels for O₃-related effects. Integrating more broadly across epidemiological and experimental evidence, the 2006 Criteria Document also focuses on the coherence and plausibility of observed O₃-related health effects to reach judgments about the extent to which causal inferences can be made about observed associations between health endpoints and exposure to O₃ in the ambient air.

a. Assessment of Evidence From Epidemiological Studies

Key elements of the evaluation of epidemiological studies are briefly summarized below.

(1) The strength of associations most directly refers to the magnitude of the reported relative risk estimates. Taking a broader view, the 2006 Criteria Document draws upon the criteria summarized in a recent report from the U.S. Surgeon General, which define strength of an association as “the magnitude of the association and its statistical strength” which includes assessment of both effect estimate size and precision, which is related to the statistical power of the study (CDC, 2004). In general, when associations are

strong in terms of yielding large relative risk estimates, it is less likely that the association could be completely accounted for by a potential confounder or some other source of bias, whereas with associations that yield small relative risk estimates it is especially important to consider potential confounding and other factors in assessing causality. Effect estimates between O₃ and some of the health outcomes are generally small in size and could thus be characterized as weak. For example, effect estimates for associations with mortality generally range from 0.5 to 5 percent increases per 0.040 ppm increase in 1-hour maximum O₃ or equivalent, whereas associations for hospitalization range up to 50 percent increases per standardized O₃ increment. However, the 2006 Criteria Document notes that there are large multicity studies that find small associations between short-term O₃ exposure and mortality or morbidity and have done so with great precision due to the statistical power of the studies (p. 8–40). That is, the power of the studies allows the authors to reliably distinguish even weak relationships from the null hypothesis with statistical confidence.

(2) In evaluating the robustness of associations, the 2006 Criteria Document (sections 7.1.3 and 8.4.4.3) and 2007 Staff Paper (section 3.4.2) have primarily considered the impact of exposure error, potential confounding by copollutants, and alternative models and model specifications.

In time-series and panel studies, the temporal (e.g., daily or hourly) changes in ambient O₃ concentrations measured at centrally-located ambient monitoring stations are generally used to represent a community's exposure to ambient O₃. In prospective cohort or cross-sectional studies, air quality data averaged over a period of months to years are used as indicators of a community's long-term exposure to ambient O₃ and other pollutants. In both types of analyses, exposure error is an important consideration, as actual exposures to individuals in the population will vary across the community.

Ozone concentrations measured at central ambient monitoring sites may explain, at least partially, the variance in individual exposures to ambient O₃; however, this relationship is influenced by various factors related to building ventilation practices and personal behaviors. Further, the pattern of exposure misclassification error and the influence of confounders may differ across the outcomes of interest as well as in susceptible populations. As discussed in the 2006 Criteria Document

(section 3.9), only a limited number of studies have examined the relationship between ambient O₃ concentrations and personal exposures to ambient O₃. One of the strongest predictors of the relationship between ambient concentrations and personal exposures appears to be time spent outdoors. The strongest relationships were observed in outdoor workers (Brauer and Brook, 1995, 1997; O'Neill *et al.*, 2004). Statistically significant correlations between ambient concentrations and personal exposures were also observed for children, who likely spend more time outdoors in the warm season (Linn *et al.*, 1996; Xu *et al.*, 2005). There is some concern about the extent to which ambient concentrations are representative of personal O₃ exposures of another particularly susceptible group of individuals, the debilitated elderly, since those who suffer from chronic cardiovascular or respiratory conditions may tend to protect themselves more than healthy individuals from environmental threats by reducing their exposure to both O₃ and its confounders, such as high temperature and PM. Studies by Sarnat *et al.* (2001, 2005) that included this susceptible group reported mixed results for associations between ambient O₃ concentrations and personal exposures to O₃. Collectively, these studies observed that the daily averaged personal O₃ exposures tend to be well correlated with ambient O₃ concentrations despite the substantial variability that existed among the personal measurements. These studies provide supportive evidence that ambient O₃ concentrations from central monitors may serve as valid surrogate measures for mean personal exposures experienced by the population, which is of most relevance for time-series studies. A better understanding of the relationship between ambient concentrations and personal exposures, as well as of the other factors that affect relationship will improve the interpretation of concentration-population health response associations observed.

The 2006 Criteria Document (section 7.1.3.1) also discusses the potential influence of exposure error on epidemiologic study results. Zeger *et al.* (2000) outlined the components to exposure measurement error, finding that ambient exposure can be assumed to be the product of the ambient concentration and an attenuation factor (*i.e.*, building filter) and that panel studies and time-series studies that use ambient concentrations instead of personal exposure measurements will

estimate a health risk that is attenuated by that factor. Navidi *et al.* (1999) used data from a children's cohort study to compare effect estimates from a simulated "true" exposure level to results of analyses from O₃ exposures determined by several methods, finding that O₃ exposures based on the use of ambient monitoring data overestimate the individual's O₃ exposure and thus generally result in O₃ effect estimates that are biased downward (EPA, 2006a, p. 7–8). Similarly, in a reanalysis of a study by Burnett *et al.* (1994) on the acute respiratory effects of ambient air pollution, Zidek *et al.* (1998) reported that accounting for measurement error, as well as making a few additional changes to the analysis, resulted in qualitatively similar conclusions, but the effects estimates were considerably larger in magnitude (EPA, 2006a, p. 7–8). A simulation study by Sheppard *et al.* (2005) also considered attenuation of the risk based on personal behavior, their microenvironment, and the qualities of the pollutant in time-series studies. Of particular interest is their finding that risk estimates were not further attenuated in time-series studies even when the correlations between personal exposures and ambient concentrations were weak. In addition to overestimation of exposure and the resulting underestimation of effects, the use of ambient O₃ concentrations may obscure the presence of thresholds in epidemiologic studies (EPA, 2006a, p. 7–9).

As discussed in the 2006 Criteria Document (section 3.9), using ambient concentrations to determine exposure generally overestimates true personal O₃ exposures by approximately 2- to 4-fold in available studies, resulting in attenuated risk estimates. The implication is that the effects being estimated occur at fairly low exposures and the potency of O₃ is greater than these effects estimates indicate. As very few studies evaluating O₃ health effects with personal O₃ exposure measurements exist in the literature, effect estimates determined from ambient O₃ concentrations must be evaluated and used with caution to assess the health risks of O₃. In the absence of available data on personal O₃ exposure, the use of routinely monitored ambient O₃ concentrations as a surrogate for personal exposures is not generally expected to change the principal conclusions from O₃ epidemiologic studies. Therefore, population health risk estimates derived using ambient O₃ levels from currently available observational studies, with appropriate caveats about personal

exposure considerations, remain useful. The 2006 Criteria Document recommends caution in the quantitative use of effect estimates calculated using ambient O₃ concentrations as they may lead to underestimation of the potency of O₃. However, the 2007 Staff Paper observes that the use of these risk estimates for comparing relative risk reductions between alternative ambient O₃ standards considered in the risk assessment (discussed below in section II.B.2) is less likely to suffer from this concern.

Confounding occurs when a health effect that is caused by one risk factor is attributed to another variable that is correlated with the causal risk factor; epidemiological analyses attempt to adjust or control for potential confounders. Copollutants (*e.g.*, PM, CO, SO₂ and NO₂) can meet the criteria for potential confounding in O₃-health associations if they are potential risk factors for the health effect under study and are correlated with O₃. Effect modifiers include variables that may influence the health response to the pollutant exposure (*e.g.*, co-pollutants, individual susceptibility, smoking or age). Both are important considerations for evaluating effects in a mixture of pollutants, but for confounding, the emphasis is on controlling or adjusting for potential confounders in estimating the effects of one pollutant, while the emphasis for effect modification is on identifying and assessing the effects for different modifiers.

The 2006 Criteria Document (p. 7–148) observes that O₃ is generally not highly correlated with other criteria pollutants (*e.g.*, PM₁₀, CO, SO₂ and NO₂), but may be more highly correlated with secondary fine particles, especially during the summer months, and that the degree of correlation between O₃ and other pollutants may vary across seasons. For example, positive associations are observed between O₃ and pollutants such as fine particles during the warmer months, but negative correlations may be observed during the cooler months (EPA, 2006a, p. 7–17). Thus, the 2006 Criteria Document (section 7.6.4) pays particular attention to the results of season-specific analyses and studies that assess effects of PM in potential confounding of O₃-health relationships. The 2006 Criteria Document also discussed the limitations of commonly used multipollutant models that include the difficulty in interpreting results where the copollutants are highly colinear, or where correlations between pollutants change by season (EPA, 2006a, p. 7–150). This is particularly the situation where O₃ and a copollutant, such as

sulfates, are formed under the same atmospheric condition; in such cases multipollutant models would produce unstable and possibly misleading results (EPA, 2006a, p. 7–152).

For mortality, the results from numerous multicity and single-city studies indicate that O₃-mortality associations do not appear to be substantially changed in multipollutant models including PM₁₀ or PM_{2.5} (EPA, 2006a, p. 7–101; Figure 7–22). Focusing on results of warm season analyses, effect estimates for O₃-mortality associations are fairly robust to adjustment for PM in multipollutant models (EPA, 2006a, p. 7–102; Figure 7–23). The 2006 Criteria Document concludes that in the few multipollutant analyses conducted for these endpoints, copollutants generally do not confound the relationship between O₃ and respiratory hospitalization (EPA, 2006a, p. 7–79 to 7–80; Figure 7–12). Multipollutant models were not used as commonly in studies of relationships between respiratory symptoms or lung function with O₃, but the 2006 Criteria Document reports that results of available analyses indicate that such associations generally were robust to adjustment for PM_{2.5} (p. 7–154). For example, in a large multicity study of asthmatic children (Mortimer *et al.*, 2002), the O₃ effect was attenuated, but there was still a positive association; in Gent *et al.* (2003), effects of O₃, but not PM_{2.5}, remained statistically significant and even increased in magnitude in two-pollutant models (EPA, 2006a, p. 7–53). Considering this body of studies, the 2006 Criteria Document (p. 7–154) concludes: “Multipollutant regression analyses indicated that O₃ risk estimates, in general, were not sensitive to the inclusion of copollutants, including PM_{2.5} and sulfate. These results suggest that the effects of O₃ on respiratory health outcomes appear to be robust and independent of the effects of other copollutants.”

The 2006 Criteria Document (p. 7–14) observes that another challenge of time-series epidemiological analysis is assessing the relationship between O₃ and health outcomes while avoiding bias due to confounding by other time-varying factors, particularly seasonal trends and weather variables. These variables are of particular interest because O₃ concentrations have a well-characterized seasonal pattern and are also highly correlated with changes in temperature, such that it can be difficult to distinguish whether effects are associated with O₃ or with seasonal or weather variables in statistical analyses.

The 2006 Criteria Document (section 7.1.3.4) discusses statistical modeling

approaches that have been used to adjust for time-varying factors, highlighting a series of analyses that were done in a Health Effects Institute-funded reanalysis of numerous time-series studies. While the focus of these reanalyses was on associations with PM, a number of investigators also examined the sensitivity of O₃ coefficients to the extent of adjustment for temporal trends and weather factors. In addition, several recent studies, including U.S. multicity studies (Bell *et al.*, 2005; Huang *et al.*, 2005; Schwartz *et al.*, 2005) and a meta-analysis study (Ito *et al.*, 2005), evaluated the effect of model specification on O₃-mortality associations. As discussed in the 2006 Criteria Document (section 7.6.3.1), these studies generally report that associations reported with O₃ are not substantially changed with alternative modeling strategies for adjusting for temporal trends and meteorologic effects. In the meta-analysis by Ito *et al.* (2005), a separate multicity analysis was presented that found that alternative adjustments for weather resulted in up to 2-fold difference in the O₃ effect estimate. Significant confounding can occur when strong seasonal cycles are present, suggesting that season-specific results are more generally robust than year-round results in such cases. A number of epidemiological studies have conducted season-specific analyses, and have generally reported stronger and more precise effect estimates for O₃ associations in the warm season than in analyses conducted in the cool seasons or over the full year.

(3) Consistency refers to the persistent finding of an association between exposure and outcome in multiple studies of adequate power in different persons, places, circumstances and times (CDC, 2004). In considering results from multicity studies and single-city studies in different areas, the 2006 Criteria Document (p. 8–41) observes general consistency in effects of short-term O₃ exposure on mortality, respiratory hospitalization and other respiratory health outcomes. The variations in effects that are observed may be attributable to differences in relative personal exposure to O₃, as well as varying concentrations and composition of copollutants present in different regions. Thus, the 2006 Criteria Document (p. 8–41) concludes that “consideration of consistency or heterogeneity of effects is appropriately understood as an evaluation of the similarity or general concordance of results, rather than an expectation of finding quantitative results with a very narrow range.”

(4) The 2007 Staff Paper recognizes that it is likely that there are biological thresholds for different health effects in individuals or groups of individuals with similar innate characteristics and health status. For O₃ exposure, individual thresholds would presumably vary substantially from person to person due to individual differences in genetic susceptibility, pre-existing disease conditions and possibly individual risk factors such as diet or exercise levels (and could even vary from one time to another for a given person). Thus, it would be difficult to detect a distinct threshold at the population level below which no individual would experience a given effect, especially if some members of a population are unusually sensitive even down to very low concentrations (EPA, 2004, p. 9–43, 9–44).

Some studies have tested associations between O₃ and health outcomes after removal of days with higher O₃ levels from the data set; such analyses do not necessarily indicate the presence or absence of a threshold, but provide some information on whether the relationship is found using only lower-concentration data. For example, using data from 95 U.S. cities, Bell *et al.* (2004) found that the effect estimate for an association between short-term O₃ exposure and mortality was little changed when days exceeding 0.060 ppm (24-hour average) were excluded in the analysis. Using data from 8 U.S. cities, Mortimer and colleagues (2002) also reported that associations between O₃ and both lung function and respiratory symptoms remained statistically significant and of the same or greater magnitude in effect size when concentrations greater than 0.080 ppm (8-hour average) were excluded (EPA, 2006a, p. 7–46). Several single-city studies also report similar findings of associations that remain or are increased in magnitude and statistical significance when data at the upper end of the concentration range are removed (EPA, 2006a, section 7.6.5).

Other time-series epidemiological studies have used statistical modeling approaches to evaluate whether thresholds exist in associations between short-term O₃ exposure and mortality. As discussed in section 7.6.5 of the 2006 Criteria Document, one European multicity study included evaluation of the shape of the concentration-response curve, and observed no deviation from a linear function across the range of O₃ measurements from the study (Gryparis *et al.*, 2004; EPA, 2006a p. 7–154). Several single-city studies also observed a monotonic increase in associations between O₃ and morbidity that suggest

that no population threshold exists (EPA, 2006a, p. 7–159).

On the other hand, a study in Korea used several different modeling approaches and reported that a threshold model provided the best fit for the data. The results suggested a potential threshold level of about 0.045 ppm (1-hour maximum concentration; < 0.035 ppm, 8-hour average) for an association between mortality and short-term O₃ exposure during the summer months (Kim *et al.*, 2004; EPA, 2006a, p. 8–43). The authors reported larger effect estimates for the association for data above the potential threshold level, suggesting that an O₃-mortality association might be underestimated in the non-threshold model. A threshold analysis recently reported by Bell *et al.* (2006) for 98 U.S. communities, including the same 95 communities in Bell *et al.* (2004), indicated that if a population threshold existed for mortality, it would likely fall below a 24-hour average O₃ concentration of 0.015 ppm (< 0.025 ppm, 8-hour average). In addition, Burnett and colleagues (1997a,b) plotted the relationships between air pollutant concentrations and both respiratory and cardiovascular hospitalization, and it appears in these results that the associations with O₃ are found in the concentration range above about 0.030 ppm (1-hour maximum; < 0.025 ppm, 8-hour average). Vedal and colleagues (2003) reported a significant association between O₃ and mortality in British Columbia where O₃ concentrations were quite low (mean 1-hour maximum concentration of 0.0273 ppm). The authors did not specifically test for threshold levels, but the fact that the association was found in an area with such low O₃ concentrations suggests that any potential threshold level would be quite low in this data set.

In summary, the 2006 Criteria Document finds that, taken together, the available evidence from controlled human exposure and epidemiological studies suggests that no clear conclusion can now be reached with regard to possible threshold levels for O₃-related effects (EPA, 2006a, p. 8–44). Thus, the available epidemiological evidence neither supports nor refutes the existence of thresholds at the population level for effects such as increased hospital admissions and premature mortality. There are limitations in epidemiological studies that make discerning thresholds in populations difficult, including low data density in the lower concentration ranges, the possible influence of exposure measurement error, and interindividual differences in

susceptibility to O₃-related effects in populations. There is the possibility that thresholds for individuals may exist in reported associations at fairly low levels within the range of air quality observed in the studies but not be detectable as population thresholds in epidemiological analyses.

b. Biological Plausibility and Coherence of Evidence

The body of epidemiological studies discussed in the 2007 Staff Paper emphasizes the role of O₃ in association with a variety of adverse respiratory and cardiovascular effects. While recognizing a variety of plausible mechanisms, there exists a general consensus suggesting that O₃, could either directly or through initiation, interfere with basic cellular oxidation processes responsible for inflammation, reduced antioxidant capacity, atherosclerosis and other effects. Reasoning that O₃ influences cellular chemistry through basic oxidative properties (as opposed to a unique chemical interaction), other reactive oxidizing species (ROS) in the atmosphere acting either independently or in combination with O₃ may also contribute to a number of adverse respiratory and cardiovascular health effects. Consequently, the role of O₃ should be considered more broadly as O₃ behaves as a generator of numerous oxidative species in the atmosphere.

In considering the biological plausibility of reported O₃-related effects, the 2007 Staff Paper (section 3.4.6) considers this broader question of health effects of pollutant mixtures containing O₃. The potential for O₃-related enhancements of PM formation, particle uptake, and exacerbation of PM-induced cardiovascular effects underscores the importance of considering contributions of O₃ interactions with other often co-occurring air pollutants to health effects due to O₃-containing pollutant mixes. The 2007 Staff Paper summarizes some examples of important pollutant mixture effects from studies that evaluate interactions of O₃ with other co-occurring pollutants, as discussed in chapters 4, 5, and 6 of the 2006 Criteria Document.

All of the types of interactive effects of O₃ with other co-occurring gaseous and nongaseous viable and nonviable PM components of ambient air mixes noted above argue that O₃ acts not only alone but that O₃ also is a surrogate indicator for air pollution mixes which may enhance the risk of adverse effects due to O₃ acting in combination with other pollutants. Viewed from this perspective, those epidemiologic

findings of morbidity and mortality associations, with ambient O₃ concentrations extending to quite low levels in many cases, become more understandable and plausible.

The 2006 Criteria Document integrates epidemiological studies with mechanistic information from controlled human exposure studies and animal toxicological studies to draw conclusions regarding the coherence of evidence and biological plausibility of O₃-related health effects to reach judgments about the causal nature of observed associations. As summarized below, coherence and biological plausibility is discussed for each of the following types of O₃-related effects: Short-term effects on the respiratory system, effects on the cardiovascular system, effects related to long-term O₃ exposure, and short-term mortality-related health endpoints.

i. Coherence and Plausibility of Short-Term Effects on the Respiratory System

Acute respiratory morbidity effects that have been associated with short-term exposure to O₃ include such health endpoints as decrements in lung function, increased respiratory symptoms, increased airway responsiveness, airway inflammation, increased permeability related to epithelial injury, immune system effects, emergency department visits for respiratory diseases, and hospitalization due to respiratory illness.

Recent epidemiological studies have supported evidence available in the previous O₃ NAAQS review on associations between ambient O₃ exposure and decline in lung function for children. The 2006 Criteria Document (p. 8–34) concludes that exposure to ambient O₃ has a significant effect on lung function and is associated with increased respiratory symptoms and medication use, particularly in asthmatics. Short-term exposure to O₃ has also been associated with more severe morbidity endpoints, such as emergency department visits and hospital admissions for respiratory cases, including specific respiratory illness (*e.g.*, asthma) (EPA, 2006a, sections 7.3.2 and 7.3.3). In addition, a few epidemiological studies have reported positive associations between short-term O₃ exposure and respiratory mortality, though the associations are not generally statistically significant (EPA, 2006a, p. 7–108).

Considering the evidence from epidemiological studies, the results described above provide evidence for coherence in O₃-related effects on the respiratory system. Effect estimates from U.S. and Canadian studies are shown in

Figure 1, where it can be seen that mostly positive associations have been reported with respiratory effects ranging from respiratory symptoms, such as cough or wheeze, to hospitalization for various respiratory diseases, and there is suggestive evidence for associations with respiratory mortality. Many of the reported associations are statistically significant, particularly in the warm season. In Figure 1, the central effect estimate is indicated by a square for

each result, with the vertical bar representing the 95 percent confidence interval around the estimate. In the discussions that follow, an individual study result is considered to be statistically significant if the 95 percent confidence interval does not include zero.²³ Positive effect estimates indicate

²³ Results for studies of respiratory symptoms are presented as odds ratios; an odds ratio of 1.0 is equivalent to no effect, and thus is presented as equivalent to the zero effect estimate line.

increases in the health outcome with O₃ exposure. In considering these results as a whole, it is important to consider not only whether statistical significance at the 95 percent confidence level is reported in individual studies but also the general pattern of results, focusing in particular on studies with greater statistical power that report relatively more precise results.

BILLING CODE 6560-50-P

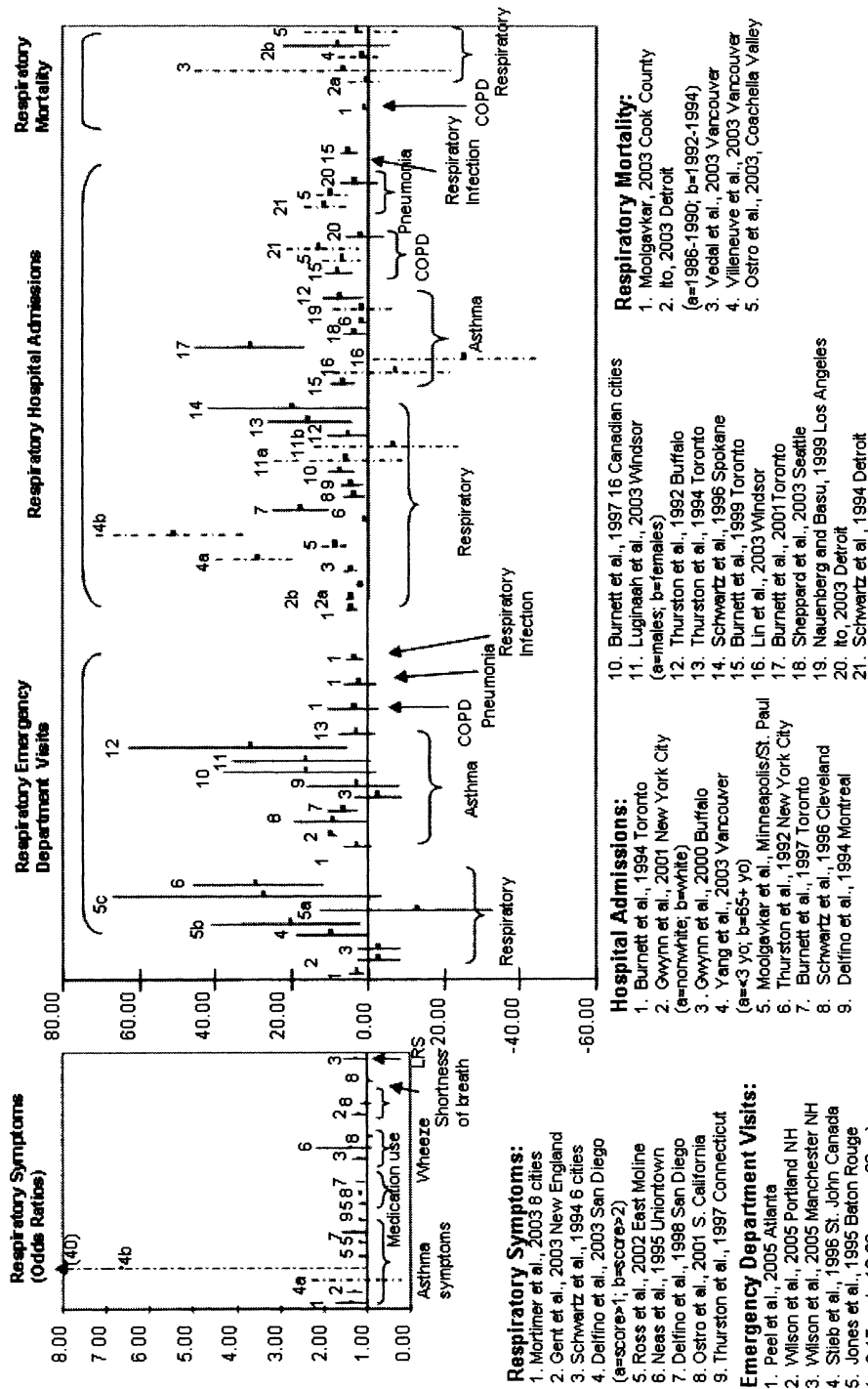


Figure 1. Effect estimates (with 95% confidence intervals) for associations between short-term ozone exposure and respiratory health outcomes.
 Effect estimates expressed as odds ratios for associations with respiratory symptoms and % increase for other outcomes, per standardized increments: 20 ppb for 24-hr O₃, 30 ppb for 8-hr O₃, and 40 ppb for 1-hr O₃, presented in order of decreasing statistical power from left to right in each category. Dotted line (blue) indicates all year analyses; solid line (red) indicates warm season results. LRS=lower respiratory symptoms; COPD=chronic obstructive pulmonary disease

BILLING CODE 6560-50-C

Considering also evidence from toxicological, controlled human exposure, and field studies, the 2006 Criteria Document (section 8.6) discusses biological plausibility and coherence of evidence for acute O₃-induced respiratory health effects. Inhalation of O₃ for several hours while subjects are physically active can elicit both acute adverse pathophysiological changes and subjective respiratory tract symptoms (EPA, 2006a, section 8.4.2).

Acute pulmonary responses observed in healthy humans exposed to O₃ at ambient concentrations include: decreased inspiratory capacity; mild bronchoconstriction; rapid, shallow breathing during exercise; subjective symptoms of tracheobronchial airway irritation, including cough and pain on deep inspiration; decreases in measures of lung function; and increased airway resistance. The severity of symptoms and magnitude of response depends on inhaled dose, individual O₃ sensitivity,

and the degree of attenuation or enhancement of response resulting from previous O₃ exposures. Lung function studies of several animal species acutely exposed to relatively low O₃ levels from a toxicological perspective (*i.e.*, 0.25 to 0.4 ppm) show responses similar to those observed in humans, including increased breathing frequency, increased tidal volume, increased resistance, and decreased FVC. Alterations in breathing pattern return to normal within hours of exposure, and

attenuation in functional responses following repeated O₃ exposures is similar to those observed in humans.

Physiological and biochemical alterations investigated in controlled human exposure and animal toxicology studies tend to support certain hypotheses of underlying pathological mechanisms which lead to the development of respiratory-related effects reported in epidemiology studies (e.g., increased hospitalization and medication use). Some of these are: (a) Decrements in lung function, (b) bronchoconstriction, (c) increased airway responsiveness, (d) airway inflammation, (e) epithelial injury, (f) immune system activation, (g) host defense impairment, and (h) sensitivity of individuals, which depends on at least a person's age, disease status, genetic susceptibility, and the degree of attenuation present due to prior exposures. The time sequence, magnitude, and overlap of these complex events, both in terms of development and recovery, illustrate the inherent difficulty of interpreting the biological plausibility of O₃-induced cardiopulmonary health effects (EPA, 2006a, p. 8–48).

The interaction of O₃ with airway epithelial cell membranes and ELF to form lipid ozonation products and ROS is supported by numerous human, animal and in vitro studies. Ozonation products and ROS initiate a cascade of events that lead to oxidative stress, injury, inflammation, airway epithelial damage and increased epithelial damage and increased alveolar permeability to vascular fluids. Repeated respiratory inflammation can lead to a chronic inflammatory state with altered lung structure and lung function and may lead to chronic respiratory diseases such as fibrosis and emphysema (EPA, 2006a, section 8.6.2). Continued respiratory inflammation also can alter the ability to respond to infectious agents, allergens and toxins. Acute inflammatory responses to O₃ are well documented, and lung injury appears within 3 hours after exposure in humans.

Taken together, the 2006 Criteria Document concludes that the evidence from experimental human and animal toxicology studies indicates that acute O₃ exposure is causally associated with respiratory system effects. These effects include O₃-induced pulmonary function decrements; respiratory symptoms; lung inflammation and increased lung permeability; airway hyperresponsiveness; increased uptake of nonviable and viable particles; and consequent increased susceptibility to PM-related toxic effects and respiratory infections (EPA, 2006a, p. 8–48).

ii. Coherence and Plausibility of Effects on the Cardiovascular System

There is very limited experimental evidence of animals and humans that has evaluated possible mechanisms or physiological pathways by which acute O₃ exposures may induce cardiovascular system effects. Ozone induces lung injury, inflammation, and impaired mucociliary clearance, with a host of associated biochemical changes all leading to increased lung epithelial permeability. As noted above in section II.A.2.a, the generation of lipid ozonation products and ROS in lung tissues can influence pulmonary hemodynamics, and ultimately the cardiovascular system. Other potential mechanisms by which O₃ exposure may be associated with cardiovascular disease outcomes have been described. Laboratory animals exposed to relatively high O₃ concentrations (≥ 0.5 ppm) demonstrate tissue edema in the heart and lungs. Ozone-induced changes in heart rate, edema of heart tissue, and increased tissue and serum levels of ANF found with 8-hour 0.5 ppm O₃ exposure in animal toxicology studies (Vesely *et al.*, 1994a,b,c) also raise the possibility of potential cardiovascular effects of acute ambient O₃ exposures.

Animal toxicology studies have found both transient and persistent ventilatory responses with and without progressive decreases in heart rate (Arito *et al.*, 1997). Observations of O₃-induced vasoconstriction in a controlled human exposure study by Brook *et al.* (2002) suggests another possible mechanism for O₃-related exacerbations of preexisting cardiovascular disease. One controlled human study (Gong *et al.*, 1998) evaluated potential cardiovascular health effects of O₃ exposure. The overall results did not indicate acute cardiovascular effects of O₃ in either the hypertensive or control subjects. The authors observed an increase in rate-pressure product and heart rate, a decrement for FEV₁, and a > 10 mm Hg increase in the alveolar/arterial pressure difference for O₂ following O₃ exposure. Foster *et al.* (1993) demonstrated that even in relatively young healthy adults, O₃ exposure can cause ventilation to shift away from the well-perfused basal lung. This effect of O₃ on ventilation distribution may persist beyond 24-hours post-exposure (Foster *et al.*, 1997). These findings suggest that O₃ may exert cardiovascular effects indirectly by impairing alveolar-arterial O₂ transfer and potentially reducing O₂ supply to the myocardium. Ozone exposure may increase myocardial work and impair pulmonary gas exchange to a degree that could perhaps be clinically

important in persons with significant preexisting cardiovascular impairment.

As noted above in section II.A.2.a, a limited number of new epidemiological studies have reported associations between short-term O₃ exposure and effects on the cardiovascular system. Among these studies, three were population-based and involved relatively large cohorts; two of these studies evaluated associations between O₃ and HRV and the other study evaluated the association between O₃ levels and the relative risk of MI or heart attack. Such studies may offer more informative results based on their large subject-pool and design. Results from these three studies were suggestive of an association between O₃ exposure and the cardiovascular endpoints studied. In other recent studies on the incidence of heart attacks and some more subtle cardiovascular health endpoints, such as changes in HRV or cardiac arrhythmia, some but not all studies reported associations with short-term exposure to O₃ (EPA, 2006a, section 7.2.7.1). From these studies, the 2006 Criteria Document concludes that the “current evidence is rather limited but suggestive of a potential effect on HRV, ventricular arrhythmias, and MI incidence” (EPA, 2006a, p. 7–65).

An increasing number of studies have evaluated the association between O₃ exposure and cardiovascular hospital admissions. As discussed in section 7.3.4 of the 2006 Criteria Document, many reported negative or inconsistent associations, whereas other studies, especially those that examined the relationship when O₃ exposures were higher, have found positive and robust associations between O₃ and cardiovascular hospital admissions. The 2006 Criteria Document (p. 7–83) finds that the overall evidence from these studies remains inconclusive regarding the effect of O₃ on cardiovascular hospitalizations. The 2006 Criteria Document notes that the suggestive positive epidemiologic findings of O₃ exposure on cardiac autonomic control, including effects on HRV, ventricular arrhythmias and heart attacks, and reported associations between O₃ exposure and cardiovascular hospitalizations generally in the warm season gain credibility and scientific support from the results of experimental animal toxicology and controlled human exposure studies, which are indicative of plausible pathways by which O₃ may exert cardiovascular effects (EPA, 2006a, section 8.6.1).

iii. Coherence and Plausibility of Effects Related to Long-Term O₃ Exposure

Controlled human exposure studies cannot evaluate effects of long-term exposures to O₃; there is some evidence available from toxicological studies. While early animal toxicology studies of long-term O₃ exposures were conducted using continuous exposures, more recent studies have focused on exposures which mimic diurnal and seasonal patterns and more realistic O₃ exposure levels (EPA, 2006a, p. 8–50). Studies of monkeys that compared these two exposure scenarios found increased airway pathology only with the latter design. Persistent and irreversible effects reported in chronic animal toxicology studies suggest that additional complementary human data are needed from epidemiologic studies (EPA, 2006a, p. 8–50).

There is limited evidence from human studies for long-term O₃-induced effects on lung function. As discussed in section 8.6.2 of the 2006 Criteria Document, previous epidemiological studies have provided only inconclusive evidence for either mortality or morbidity effects of long-term O₃ exposure. The 2006 Criteria Document (p. 8–50) observes that the inconsistency in findings may be due to a lack of precise exposure information, the possibility of selection bias, and the difficulty of controlling for confounders. Several new longitudinal epidemiology studies have evaluated associations between long-term O₃ exposures and morbidity and mortality and suggest that these long-term exposures may be related to changes in lung function in children; however, little evidence is available to support a relationship between chronic O₃ exposure and mortality or lung cancer incidence (EPA, 2006a, p. 8–50).

The 2006 Criteria Document (p. 8–51) concludes that evidence from animal toxicology studies strongly suggests that chronic O₃ exposure is capable of damaging the distal airways and proximal alveoli, resulting in lung tissue remodeling leading to apparent irreversible changes. Such structural changes and compromised lung function caused by persistent inflammation may exacerbate the progression and development of chronic lung disease. Together with the limited evidence available from epidemiological studies, these findings offer some insight into potential biological mechanisms for suggested associations between long-term or seasonal exposures to O₃ and reduced lung function development in children which have been observed in

epidemiologic studies (EPA, 2006a, p. 8–51).

iv. Coherence and Plausibility of Short-Term Mortality-Related Health Endpoints

An extensive epidemiological literature on air pollution related mortality risk estimates from the U.S., Canada, and Europe is discussed in the 2006 Criteria Document (sections 7.4 and 8.6.3). These single- and multicity mortality studies coupled with results from meta-analyses generally indicate associations between acute O₃ exposure and elevated risk for all-cause mortality, even after adjustment for the influence of season and PM exposure. Several single-city studies that specifically evaluated the relationship between O₃ exposure and cardiopulmonary mortality also reported results suggestive of a positive association (EPA, 2006a, p. 8–51). These mortality studies suggest a pattern of effects for causality that have biologically plausible explanations, but our knowledge regarding potential underlying mechanisms is very limited at this time and requires further research. Most of the physiological and biochemical parameters investigated in human and animal studies suggest that O₃-induced biochemical effects are relatively transient and attenuate over time. The 2006 Criteria Document (p. 8–52) hypothesizes a generic pathway of O₃-induced lung damage, potentially involving oxidative lung damage with subsequent inflammation and/or decline in lung function leading to respiratory distress in some sensitive population groups (*e.g.*, asthmatics), or other plausible pathways noted below that may lead to O₃-related contributions to cardiovascular effects that ultimately increase risk of mortality.

The third National Health and Nutrition Examination Survey follow-up data analysis indicates that about 20 percent of the adult population has reduced FEV₁ values, suggesting impaired lung function in a significant portion of the population. Most of these individuals have COPD, asthma or fibrotic lung disease (Manino *et al.*, 2003), which are associated with persistent low-grade inflammation. Furthermore, patients with COPD are at increased risk for cardiovascular disease. Also, lung disease with underlying inflammation may be linked to low-grade systemic inflammation associated with atherosclerosis, independent of cigarette smoking (EPA, 2006a, p. 8–52). Lung function decrements in persons with cardiopulmonary disease have been associated with inflammatory markers,

such as C-reactive protein (CRP) in the blood. At a population level it has been found that individuals with the lowest FEV₁ values have the highest levels of CRP, and those with the highest FEV₁ values have the lowest CRP levels (Manino *et al.*, 2003; Sin and Man, 2003). This complex series of physiological and biochemical reactions following O₃ exposure may tilt the biological homeostasis mechanisms which could lead to adverse health effects in people with compromised cardiopulmonary systems.

Several other types of newly available data also support reasonable hypotheses that may help to explain the findings of O₃-related increases in cardiovascular mortality observed in some epidemiological studies. These include the direct effect of O₃ on increasing PAF in lung tissue that can then enter the general circulation and possibly contribute to increased risk of blood clot formation and the consequent increased risk of heart attacks, cerebrovascular events (stroke), or associated cardiovascular-related mortality. Ozone reactions with cholesterol in lung surfactant to form epoxides and oxysterols that are cytotoxic to lung and heart muscles and that contribute to atherosclerotic plaque formation in arterial walls represent another potential pathway. Stimulation of airway irritant receptors may lead to increases in tissue and serum levels of ANF, changes in heart rate, and edema of heart tissue. A few new field and panel studies of human adults have reported associations between ambient O₃ concentrations and changes in cardiac autonomic control (*e.g.*, HRV, ventricular arrhythmias, and MI). These represent plausible pathways that may lead to O₃-related contributions to cardiovascular effects that ultimately increase the risk of mortality.

In addition, O₃-induced increases in lung permeability allow more ready entry for inhaled PM into the blood stream, and thus O₃ exposure may increase the risk of PM-related cardiovascular effects. Furthermore, increased ambient O₃ levels contribute to ultrafine PM formation in the ambient air and indoor environments. Thus, the contributions of elevated ambient O₃ concentrations to ultrafine PM formation and human exposure, along with the enhanced uptake of inhaled fine particles, consequently may contribute to exacerbation of PM-induced cardiovascular effects in addition to those more directly induced by O₃ (EPA, 2006a, p. 8–53).

c. Summary

Judgments concerning the extent to which relationships between various health endpoints and ambient O₃ exposures are likely to be causal are informed by the conclusions and discussion in the 2006 Criteria Document as discussed above and summarized in section 3.7.5 of the 2007 Staff Paper. These judgments reflect the nature of the evidence and the overall weight of the evidence, and are taken into consideration in the quantitative risk assessment discussed below in section II.B.2.

For example, there is a very high level of confidence that O₃ induces lung function decrements in healthy adults and children due in part to the dozens of controlled human exposure and epidemiological studies consistently showing such effects. The 2006 Criteria Document (p. 8–74) states that these studies provide clear evidence of causality for associations between short-term O₃ exposures and statistically significant declines in lung function in children, asthmatics and adults who exercise outdoors. An increase in respiratory symptoms (*e.g.*, cough, shortness of breath) has been observed in controlled human exposure studies of short-term O₃ exposures, and significant associations between ambient O₃ exposures and a wide variety of respiratory symptoms have been reported in epidemiology studies (EPA, 2006a, p. 8–75). Population time-series studies showing robust associations between O₃ exposures and respiratory hospital admissions and emergency department visits are strongly supported by controlled human exposure, animal toxicological, and epidemiological evidence for O₃-related lung function decrements, respiratory symptoms, airway inflammation, and airway hyperactivity. The 2006 Criteria Document (p. 8–77) concludes that, taken together, the overall evidence supports the inference of a causal relationship between acute ambient O₃ exposures and increased respiratory morbidity outcomes resulting in increased emergency department visits and hospitalizations during the warm season. Further, recent epidemiologic evidence has been characterized in the 2006 Criteria Document (p. 8–78) as highly suggestive that O₃ directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality.

4. O₃-Related Impacts on Public Health

The following discussion draws from chapters 6 and 7 and section 8.7 of the 2006 Criteria Document and section 3.6 of the 2007 Staff Paper to characterize

factors which modify responsiveness to O₃, populations potentially at risk for O₃-related health effects, the adversity of O₃-related effects, and the size of the at-risk populations in the U.S. These considerations are all important elements in characterizing the potential public health impacts associated with exposure to ambient O₃.

a. Factors That Modify Responsiveness to Ozone

There are numerous factors that can modify individual responsiveness to O₃. These include: influence of physical activity; age; gender and hormonal influences; racial, ethnic and socioeconomic status (SES) factors; environmental factors; and oxidant-antioxidant balance. These factors are discussed in more detail in section 6.5 of the 2006 Criteria Document.

It is well established that physical activity increases an individual's minute ventilation and will thus increase the dose of O₃ inhaled (EPA, 2006a, section 6.5.4). Increased physical activity results in deeper penetration of O₃ into more distal regions of the lungs, which are more sensitive to acute O₃ response and injury. This will result in greater lung function decrements for acute exposures of individuals during increased physical activity. Research has shown that respiratory effects are observed at lower O₃ concentrations if the level of exertion is increased and/or duration of exposure and exertion are extended. Predicted O₃-induced decrements in lung function have been shown to be a function of exposure concentration, duration and exercise level for healthy, young adults (McDonnell *et al.*, 1997).

Most of the studies investigating the influence of age have used lung function decrements and symptoms as measures of response. For healthy adults, lung function and symptom responses to O₃ decline as age increases. The rate of decline in O₃ responsiveness appears greater in those 18 to 35 years old compared to those 35 to 55 years old, while there is very little change after age 55. In one study (Seal *et al.*, 1996) analyzing a large data set, a 5.4% decrement in FEV₁ on average was estimated for 20-year-old individuals exposed to 0.12 ppm O₃ for 2.3 hours, whereas similar exposure of 35-year-old individuals resulted in a 2.6% decrement on average. While healthy children tend not to report respiratory symptoms when exposed to low levels of O₃, for subjects 18 to 36 years old symptom responses induced by O₃ are observed but tend to decrease with increasing age within this range (McDonnell *et al.*, 1999).

Limited evidence of gender differences in response to O₃ exposure has suggested that females may be predisposed to a greater susceptibility to O₃. Lower plasma and NL fluid levels of the most prevalent antioxidant, uric acid, in females relative to males may be a contributing factor. Consequently, reduced removal of O₃ in the upper airways may promote deeper penetration. However, most of the evidence on gender differences appears to be equivocal, with one study (Hazucha *et al.*, 2003) suggesting that physiological responses of young healthy males and females may be comparable (EPA, 2006a, section 6.5.2).

A few studies have suggested that ethnic minorities might be more responsive to O₃ than Caucasian population groups (EPA, 2006a, section 6.5.3). This may be more the result of a lack of adequate health care and socioeconomic status (SES) than any differences in sensitivity to O₃. The limited data available, which have investigated the influence of race, ethnic or other related factors on responsiveness to O₃, prevent drawing any clear conclusions at this time.

Few human studies have examined the potential influence of environmental factors such as the sensitivity of individuals who voluntarily smoke tobacco (*i.e.*, smokers) and the effect of high temperatures on O₃ responsiveness. New controlled human exposure studies have confirmed that smokers are less responsive to O₃ than nonsmokers; however, time course of development and recovery of these effects, as well as reproducibility, was not different from nonsmokers (EPA, 2006a, section 6.5.5). Influence of ambient temperature on pulmonary effects induced by O₃ has been studied very little, but additive effects of heat and O₃ exposure have been reported.

Antioxidants, which scavenge free radicals and limit lipid peroxidation in the ELF, are the first line of defense against oxidative stress. Ozone exposure leads to absorption of O₃ in the ELF with subsequent depletion of antioxidant in the nasal ELF, but concentration and antioxidant enzyme activity in ELF or plasma do not appear related to O₃ responsiveness (EPA 2006a, section 6.5.6). Controlled studies of dietary antioxidant supplements have shown some protective effects on lung function decrements but not on symptoms and airway inflammatory responses. Dietary antioxidant supplements have provided some protection to asthmatics by attenuating post-exposure airway hyperresponsiveness. Animal studies

have also supported the protective effects of ELF antioxidants.

b. At-Risk Subgroups for O₃-Related Effects

Several characteristics may increase the extent to which a population group shows increased susceptibility or vulnerability. Information on potentially susceptible and vulnerable groups is summarized in section 8.7 of the 2006 Criteria Document. As described there, the term *susceptibility* refers to innate (e.g., genetic or developmental) or acquired (e.g., personal risk factors, age) factors that make individuals more likely to experience effects with exposure to pollutants. A number of population groups have been identified as potentially susceptible to health effects as a result of O₃ exposure, including people with existing lung diseases, including asthma, children and older adults, and people who have larger than normal lung function responses that may be due to genetic susceptibility. In addition, some population groups have been identified as having increased vulnerability to O₃-related effects due to increased likelihood of exposure while at elevated ventilation rates, including healthy children and adults who are active outdoors, for example, outdoor workers and joggers. Taken together, the susceptible and vulnerable groups make up “at-risk” groups.²⁴

i. Active People

A large group of individuals at risk from O₃ exposure consists of outdoor workers and children, adolescents, and adults who engage in outdoor activities involving exertion or exercise during summer daylight hours when ambient O₃ concentrations tend to be higher. This conclusion is based on a large number of controlled-human exposure studies and several epidemiologic field/panel studies which have been conducted with healthy children and adults and those with preexisting respiratory diseases (EPA 2006a, sections 6.2, 6.3, 7.2, and 8.4.4). The controlled human exposure studies show a clear O₃ exposure-response relationship with increasing spirometric and symptomatic response as exercise level increases. Furthermore, O₃-induced response increases as time of exposure increases. Studies of outdoor workers and others who participate in outdoor activities indicate that extended exposures to O₃ at elevated exertion

levels can produce marked effects on lung function, as discussed above in section IIA.2 (Brauer *et al.*, 1996; Höpfe *et al.*, 1995; Korrick *et al.*, 1998; McConnell *et al.*, 2002).

These field studies with subjects at elevated exertion levels support the extensive evidence derived from controlled human exposure studies. The majority of controlled human exposure studies has examined the effects of O₃ exposure in subjects performing continuous or intermittent exercise for variable periods of time and has reported significant O₃-induced respiratory responses. The epidemiologic studies discussed above also indicate that prolonged exposure periods, combined with elevated levels of exertion or exercise, may magnify O₃ effects on lung function. Thus, outdoor workers and others who participate in higher exertion activities outdoors during the time of day when high peak O₃ concentrations occur appear to be particularly vulnerable to O₃ effects on respiratory health. Although these studies show a wide variability of response and sensitivity among subjects and the factors contributing to this variability continue to be incompletely understood, the effect of increased exertion is consistent. It should be noted that this wide variability of response and sensitivity among subjects may be in part due to the wide range of other highly reactive photochemical oxidants coexisting with O₃ in the ambient air.

ii. People With Lung Disease

People with preexisting pulmonary disease are among those at increased risk from O₃ exposure. Altered physiological, morphological, and biochemical states typical of respiratory diseases like asthma, COPD, and chronic bronchitis may render people sensitive to additional oxidative burden induced by O₃ exposure. At the time of the 1997 review, it was concluded that these groups were at greater risk because the impact of O₃-induced responses on already-compromised respiratory systems would noticeably impair an individual's ability to engage in normal activity or would be more likely to result in increased self-medication or medical treatment. At that time there was little evidence that people with pre-existing disease were more responsive than healthy individuals in terms of the magnitude of lung function decrements or symptomatic responses. The new results from controlled exposure and epidemiologic studies continue to indicate that individuals with preexisting pulmonary disease are a sensitive population for O₃-related health effects.

Several controlled human exposure studies reviewed in the 1996 Criteria Document on atopic and asthmatic subjects have suggested but not clearly demonstrated enhanced responsiveness to acute O₃ exposure compared to healthy subjects. The majority of the newer studies reviewed in Chapter 6 of the 2006 Criteria Document indicate that asthmatics are more sensitive than normal subjects in manifesting O₃-induced lung function decrements. In one key study (Horstman *et al.*, 1995), the FEV₁ decrement observed in the asthmatics was significantly larger than in the healthy subjects (19% versus 10%, respectively). There was also a notable tendency for a greater group mean O₃-induced decrease in FEF₂₅₋₇₅ in asthmatics relative to the healthy subjects (24% versus 15%, respectively). A significant positive correlation in asthmatics was also reported between the magnitude of O₃-induced spirometric responses and baseline lung function, *i.e.*, responses increased with severity of disease.

Asthmatics present a differential response profile for cellular, molecular, and biochemical parameters (2006 Criteria Document, Figure 8–1) that are altered in response to acute O₃ exposure. Ozone-induced increases in neutrophils, IL–8 and protein were found to be significantly higher in the BAL fluid from asthmatics compared to healthy subjects, suggesting mechanisms for the increased sensitivity of asthmatics (Basha *et al.*, 1994; McBride *et al.*, 1994; Scannell *et al.*, 1996; Hiltermann *et al.*, 1999; Holz *et al.*, 1999; Bosson *et al.*, 2003). Neutrophils, or PMNs, are the white blood cells most associated with inflammation. IL–8 is an inflammatory cytokine with a number of biological effects, primarily on neutrophils. The major role of this cytokine is to attract and activate neutrophils. Protein in the airways is leaked from the circulatory system, and is a marker for increased cellular permeability.

Bronchial constriction following provocation with O₃ and/or allergens presents a two-phase response. The early response is mediated by release of histamine and leukotrienes that leads to contraction of smooth muscle cells in the bronchi, narrowing the lumen and decreasing the airflow. In people with allergic airway disease, including people with rhinitis and asthma, these mediators also cause accumulation of eosinophils in the airways (Bascom *et al.*, 1990; Jorres *et al.*, 1996; Peden *et al.*, 1995 and 1997; Frampton *et al.*, 1997; Michelson *et al.*, 1999; Hiltermann *et al.*, 1999; Holz *et al.*, 2002; Vagaggini *et al.*, 2002). In asthma, the eosinophil,

²⁴ In the Staff Paper and documents from previous O₃ NAAQS reviews, “at-risk” groups have also been called “sensitive” groups, to mean both groups with greater inherent susceptibility and those more likely to be exposed.

which increases inflammation and allergic responses, is the cell most frequently associated with exacerbations of the disease. A study by Bosson *et al.* (2003) evaluated the difference in O₃-induced bronchial epithelial cytokine expression between healthy and asthmatic subjects. After O₃ exposure the epithelial expression of IL-5 and GM-CSF increased significantly in asthmatics, compared to healthy subjects. Asthma is associated with Th2-related airway response (allergic response), and IL-5 is an important Th2-related cytokine. The O₃-induced increase in IL-5, and also in GM-CSF, which affects the growth, activation and survival of eosinophils, may indicate an effect on the Th2-related airway response and on airway eosinophils. The authors reported that the O₃-induced Th2-related cytokine responses that were found within the asthmatic group may indicate a worsening of their asthmatic airway inflammation and thus suggest a plausible link to epidemiological data indicating O₃-associated increases in bronchial reactivity and hospital admissions.

The accumulation of eosinophils in the airways of asthmatics is followed by production of mucus and a late-phase bronchial constriction and reduced airflow. In a study of 16 intermittent asthmatics, Hiltermann *et al.* (1999) found that there was a significant inverse correlation between the O₃-induced change in the percentage of eosinophils in induced sputum and the change in PC₂₀, the concentration of methacholine causing a 20% decrease in FEV₁. Characteristic O₃-induced inflammatory airway neutrophilia at one time was considered a leading mechanism of airway hyperresponsiveness. However, Hiltermann *et al.* (1999) determined that the O₃-induced change in percentage neutrophils in sputum was not significantly related to the change in PC₂₀. These results are consistent with the results of Zhang *et al.* (1995), which found neutrophilia in a murine model to be only coincidentally associated with airway hyperresponsiveness, *i.e.*, there was no cause and effect relationship. (2006 Criteria Document, AX 6–26). Hiltermann *et al.* (1999) concluded that the results point to the role of eosinophils in O₃-induced airway hyperresponsiveness. Increases in O₃-induced nonspecific airway responsiveness incidence and duration could have important clinical implications for asthmatics.

Two studies (Jörres *et al.*, 1996; Holz *et al.*, 2002) observed increased airway responsiveness to O₃ exposure with bronchial allergen challenge in subjects

with preexisting allergic airway disease. Jörres *et al.* (1996) found that O₃ causes an increased response to bronchial allergen challenge in subjects with allergic rhinitis and mild allergic asthma. The subjects were exposed to 0.25 ppm O₃ for 3 hours with IE. Airway responsiveness to methacholine was determined 1 hour before and after exposure; responsiveness to allergen was determined 3 hours after exposure. Statistically significant decreases in FEV₁ occurred in subjects with allergic rhinitis (13.8%) and allergic asthma (10.6%), and in healthy controls (7.3%). Methacholine responsiveness was statistically increased in asthmatics, but not in subjects with allergic rhinitis or healthy controls. Airway responsiveness to an individual's historical allergen (either grass and birch pollen, house dust mite, or animal dander) was significantly increased after O₃ exposure when compared to FA exposure. In subjects with asthma and allergic rhinitis, a maximum percent fall in FEV₁ of 27.9% and 7.8%, respectively, occurred 3 days after O₃ exposure when they were challenged with of the highest common dose of allergen. The authors concluded that subjects with asthma or allergic rhinitis, without asthma, could be at risk if a high O₃ exposure is followed by a high dose of allergen. Holz *et al.* (2002) reported an early phase lung function response in subjects with rhinitis after a consecutive 4-day exposure to 0.125 ppm O₃ that resulted in a clinically relevant (>20%) decrease in FEV₁. Ozone-induced exacerbation of airway responsiveness persists longer and attenuates more slowly than O₃-induced lung function decrements and respiratory symptom responses and can have important clinical implications for asthmatics.

A small number of *in vitro* studies corroborate the differences in the responses of asthmatic and healthy subject generally found in controlled human exposure studies. *In vitro* studies (Schierhorn *et al.*, 1999) of nasal mucosal biopsies from atopic and nonatopic subjects exposed to 0.1 ppm O₃ found significant differences in release of IL-4, IL-6, IL-8, and TNF- α . Another study by Schierhorn *et al.* (2002) found significant differences in the O₃-induced release of the neuropeptides neurokinin A and substance P for allergic patients in comparison to nonallergic controls, suggesting increased activation of sensory nerves by O₃ in the allergic tissues. Another study by Bayram *et al.* (2002) using *in vitro* culture of bronchial epithelial cells recovered from atopic and nonatopic asthmatics also

found significant increases in epithelial permeability in response to O₃ exposure.

The new data on airway responsiveness, inflammation, and various molecular markers of inflammation and bronchoconstriction indicate that people with asthma and allergic rhinitis (with or without asthma) comprise susceptible groups for O₃-induced adverse effects. This body of evidence indicates that controlled human exposure and epidemiological panel studies of lung function decrements and respiratory symptoms that evaluate only healthy, non-asthmatic subjects likely underestimate the effects of O₃ exposure on asthmatics and other susceptible populations. The effects of O₃ on lung function, inflammation, and increased airway responsiveness demonstrated in subjects with asthma and other allergic airway diseases, provide plausible mechanisms underlying the more serious respiratory morbidity effects, such as emergency department visits and hospital admissions, and respiratory mortality effects.

A number of epidemiological studies have been conducted using asthmatic study populations. The majority of epidemiological panel studies that evaluated respiratory symptoms and medication use related to O₃ exposures focused on children. These studies suggest that O₃ exposure is associated with increased respiratory symptoms and medication use in children with asthma. Other reported effects include respiratory symptoms, lung function decrements, and emergency department visits, as discussed in the 2006 Criteria Document (section 7.6.7.1). Strong evidence from a large multicity study (Mortimer *et al.*, 2002), along with support from several single-city studies indicate that O₃ exposure is associated with increased respiratory symptoms and medication use in children with asthma. With regard to ambient O₃ levels and increased hospital admissions and emergency department visits for asthma and other respiratory causes, strong and consistent evidence establishes a correlation between O₃ exposure and increased exacerbations of preexisting respiratory disease for 1-hour maximum O₃ concentrations <0.12 ppm. As discussed above and in the 2006 Criteria Document, section 7.3, several hospital admission and emergency department visit studies in the U.S., Canada, and Europe have reported positive associations between increase in O₃ and increased risk of emergency department visits and hospital admissions for asthma other

respiratory diseases, especially during the warm season.

In summary, based on a substantial new body of evidence from animal, controlled human exposure and epidemiological studies the 2006 Criteria Document (section x.x) concludes that people with asthma and other preexisting pulmonary diseases are among those at increased risk from O₃ exposure. Evidence from controlled human exposure studies indicates that asthmatics may exhibit larger lung function decrements and can have larger inflammatory responses in response to O₃ exposure than healthy controls. Asthmatics present a different response profile for cellular, molecular, and biochemical parameters that are altered in response to acute O₃ exposure. Asthmatics, and people with allergic rhinitis, are more likely to mount an allergic-type response upon exposure to O₃, as manifested by increases in white blood cells associated with allergy and related molecules, which increase inflammation in the airways. The increased inflammatory and allergic responses also may be associated with the larger late-phase responses that asthmatics can experience, which can include increased bronchoconstrictor responses to irritant substances or allergens and additional inflammation. Epidemiological studies have reported fairly robust associations between ambient O₃ concentrations and measures of lung function and daily respiratory symptoms (e.g., chest tightness, wheeze, shortness of breath) in children with moderate to severe asthma and between O₃ and increased asthma medication use. These more serious responses in asthmatics and others with lung disease provide biological plausibility for the respiratory morbidity effects observed in epidemiological studies, such as emergency department visits and hospital admissions. The body of evidence from controlled human exposure and epidemiological studies, which includes asthmatic as well as non-asthmatic subjects, indicates that controlled human exposure studies of lung function decrements and respiratory symptoms that evaluate only healthy, non-asthmatic subjects likely underestimate the effects of O₃ exposure on asthmatics and other susceptible populations.

Newly available reports from controlled human exposure studies (see chapter 6 in the 2006 Criteria Document) utilized subjects with preexisting cardiopulmonary diseases such as COPD, asthma, allergic rhinitis, and hypertension. The data generated from these studies that evaluated

changes in spirometry did not find clear differences between filtered air and O₃ exposure in COPD subjects. However, the new data on airway responsiveness, inflammation, and various molecular markers of inflammation and bronchoconstriction indicate that people with atopic asthma and allergic rhinitis comprise susceptible groups for O₃-induced adverse health effects.

Although controlled human exposure studies have not found evidence of larger spirometric responses to O₃ in people with COPD relative to healthy subjects, this may be due to the fact that most people with COPD are older adults who would not be expected to be as responsive based on their age. However, in section 8.7.1, the 2006 Criteria Document notes that new epidemiological evidence indicates that people with COPD may be more likely to experience other effects, including emergency room visits, hospital admissions, or premature mortality. For example, results from an analysis of five European cities indicated strong and consistent O₃ effects on unscheduled respiratory hospital admissions, including COPD (Anderson *et al.*, 1997). Also, an analysis of a 9-year data set for the whole population of the Netherlands provided risk estimates for more specific causes of mortality, including COPD (Hoek *et al.*, 2000, 2001; reanalysis Hoek, 2003); a positive, but nonsignificant, excess risk of COPD-related mortality was found to be associated with short-term O₃ concentrations. Moreover, as indicated by Gong *et al.* (1998), the effects of O₃ exposure on alveolar-arterial oxygen gradients may be more pronounced in patients with preexisting obstructive lung diseases. Relative to healthy elderly subjects, COPD patients have reduced gas exchange and low SaO₂. Any inflammatory or edematous responses due to O₃ delivered to the well-ventilated regions of the lung in COPD subjects could further inhibit gas exchange and reduce oxygen saturation. In addition, O₃-induced vasoconstriction could also acutely induce pulmonary hypertension. Inducing pulmonary vasoconstriction and hypertension in these patients would perhaps worsen their condition, especially if their right ventricular function was already compromised (EPA, 2006a, section 6.10). These controlled human exposure and epidemiological studies indicate that people with pre-existing lung diseases other than asthma are also at greater risk from O₃ exposure than people without lung disease.

iii. Children and Older Adults

Supporting evidence exists for heterogeneity in the effects of O₃ by age. As discussed in section 6.5.1 of the 2006 Criteria Document, children, adolescents, and young adults (<18 yrs of age) appear, on average, to have nearly equivalent spirometric responses to O₃, but have greater responses than middle-aged and older adults when exposed to comparable O₃ doses. Symptomatic responses to O₃ exposure, however, do not appear to occur in healthy children, but are observed in asthmatic children, particularly those who use maintenance medications. For adults (>17 yrs of age) symptoms gradually decrease with increasing age. In contrast to young adults, the diminished symptomatic responses in children and the diminished symptomatic and spirometric responses in older adults increases the likelihood that these groups continue outdoor activities leading to greater O₃ exposure and dose.

As described in the section 7.6.7.2 of the 2006 Criteria Document, many epidemiological field studies focused on the effect of O₃ on the respiratory health of school children. In general, children experienced decrements in lung function parameters, including PEF, FEV₁, and FVC. Increases in respiratory symptoms and asthma medication use were also observed in asthmatic children. In one German study, children with and without asthma were found to be particularly susceptible to O₃ effects on lung function. Approximately 20 percent of the children, both with and without asthma, experienced a greater than 10 percent change in FEV₁, compared to only 5 percent of the elderly population and athletes (Höppe *et al.*, 2003).

The American Academy of Pediatrics (2004) notes that children and infants are among the population groups most susceptible to many air pollutants, including O₃. This is in part because their lungs are still developing. For example, eighty percent of alveoli are formed after birth, and changes in lung development continue through adolescence (Dietert *et al.*, 2000). Children are also likely to spend more time outdoors than adults, which results in increased exposure to air pollutants (Wiley *et al.*, 1991a,b). Moreover, children have high minute ventilation rates and high levels of physical activity which also increases their dose (Plunkett *et al.*, 1992).

Several mortality studies have investigated age-related differences in O₃ effects (EPA, 2006a, section 7.6.7.2). Older adults are also often classified as

being particularly susceptible to air pollution. The 2006 Criteria Document (p. 8–60) concludes that the basis for increased O₃ sensitivity among the elderly is not known, but one hypothesis is that it may be related to changes in the respiratory tract lining fluid antioxidant defense network (Kelly *et al.*, 2003). Older adults have lower baseline lung function than younger people, and are also more likely to have preexisting lung and heart disease. Increased susceptibility of older adults to O₃ health effects is most clearly indicated in the newer mortality studies. Among the studies that observed positive associations between O₃ and mortality, a comparison of all age or younger age (≤65 years of age) O₃-mortality effect estimates to that of the elderly population (>65 years) indicates that, in general, the elderly population is more susceptible to O₃ mortality effects. The meta-analysis by Bell *et al.* (2005) found a larger mortality effect estimate for the elderly than for all ages. In the large U.S. 95 communities study (Bell *et al.*, 2004), mortality effect estimates were slightly higher for those aged 65 to 74 years, compared to individuals less than 65 years and 75 years or greater. The absolute effect of O₃ on premature mortality may be substantially greater in the elderly population because of higher rates of preexisting respiratory and cardiac diseases. The 2006 Criteria Document (p. 7–177) concludes that the elderly population (>65 years of age) appear to be at greater risk of O₃-related mortality and hospitalizations compared to all ages or younger populations.

The 2006 Criteria Document notes that, collectively, there is supporting evidence of age-related differences in susceptibility to O₃ lung function effects. The elderly population (>65 years of age) appear to be at increased risk of O₃-related mortality and hospitalizations, and children (<18 years of age) experience other potentially adverse respiratory health outcomes with increased O₃ exposure (EPA, 2006a, section 7.6.7.2).

iv. People With Increased Responsiveness to Ozone

New animal toxicology studies using various strains of mice and rats have identified O₃-sensitive and resistant strains and illustrated the importance of genetic background in determining O₃ susceptibility (EPA, 2006a, section 8.7.4). Controlled human exposure studies have also indicated a high degree of variability in some of the pulmonary physiological parameters. The variable effects in individuals have been found to be reproducible, in other

words, a person who has a large lung function response after exposure to O₃ will likely have about the same response if exposed again to the same dose of O₃. In controlled human exposure studies, group mean responses are not representative of this segment of the population that has much larger than average responses to O₃. Recent studies of asthmatics by David *et al.* (2003) and Romieu *et al.* (2004) reported a role for genetic polymorphism in observed differences in antioxidant enzymes and genes involved in inflammation to modulate lung function and inflammatory responses to O₃ exposure.²⁵

Biochemical and molecular parameters extensively evaluated in these experiments were used to identify specific loci on chromosomes and, in some cases, to relate the differential expression of specific genes to biochemical and physiological differences observed among these species. Utilizing O₃-sensitive and O₃-resistant species, it has been possible to identify the involvement of increased airway reactivity and inflammation processes in O₃ susceptibility. However, most of these studies were carried out using relatively high doses of O₃, making the relevance of these studies questionable in human health effects assessment. The genes and genetic loci identified in these studies may serve as useful biomarkers in the future.

v. Other Population Groups

There is limited, new evidence supporting associations between short-term O₃ exposures and a range of effects on the cardiovascular system. Some but not all, epidemiological studies have reported associations between short-term O₃ exposures and the incidence of heart attacks and more subtle cardiovascular health endpoints, such as changes in HRV and cardiac arrhythmia. Others have reported associations with hospitalization or emergency department visits for cardiovascular diseases, although the results across the studies are not consistent. Studies also report associations between short-term O₃ exposure and mortality from cardiovascular or cardiopulmonary causes. The 2006 Criteria Document (p. 7–65) concludes that current

²⁵ Similar to animal toxicology studies referred above, a polymorphism in a specific proinflammatory cytokine gene has been implicated in O₃-induced lung function changes in healthy, mild asthmatics and individuals with rhinitis. These observations suggest a potential role for these markers in the innate susceptibility to O₃, however, the validity of these markers and their relevance in the context of prediction to population studies requires additional research.

cardiovascular effects evidence from some field studies is rather limited but supportive of a potential effect of short-term O₃ exposure and HRV, cardiac arrhythmia, and heart attack incidence. In the 2006 Criteria Document's evaluation of studies of hospital admissions for cardiovascular disease (EPA 2006a, section 7.3.4), it is concluded that evidence from this growing group of studies is generally inconclusive regarding an association with O₃ in studies conducted during the warm season (EPA 2006a, p. 7–83). This body of evidence suggests that people with heart disease may be at increased risk from short-term exposures to O₃; however, more evidence is needed to conclude that people with heart disease are a susceptible population.

Other groups that might have enhanced sensitivity to O₃, but for which there is currently very little evidence, include groups based on race, gender and SES, and those with nutritional deficiencies, which presents factors which modify responsiveness to O₃.

c. Adversity of Effects

In the 2008 rulemaking, in making judgments as to when various O₃-related effects become regarded as adverse to the health of individuals, EPA looked to guidelines published by the American Thoracic Society (ATS) and the advice of CASAC. While recognizing that perceptions of “medical significance” and “normal activity” may differ among physicians, lung physiologists and experimental subjects, the ATS (1985)²⁶ defined adverse respiratory health effects as “medically significant physiologic changes generally evidenced by one or more of the following: (1) Interference with the normal activity of the affected person or persons, (2) episodic respiratory illness, (3) incapacitating illness, (4) permanent respiratory injury, and/or (5) progressive respiratory dysfunction.” During the 1997 review, it was concluded that there was evidence of causal associations from controlled human exposure studies for effects in the first of these five ATS-defined categories, evidence of statistically significant associations from epidemiological studies for effects in the second and third categories, and evidence from animal toxicology

²⁶ In 2000, the American Thoracic Society (ATS) published an official statement on “What Constitutes an Adverse Health Effect of Air Pollution?” (ATS, 2000), which updated its earlier guidance (ATS, 1985). Overall, the new guidance does not fundamentally change the approach previously taken to define adversity, nor does it suggest a need at this time to change the structure or content of the tables describing gradation of severity and adversity of effects described below.

studies, which could be extrapolated to humans only with a significant degree of uncertainty, for the last two categories.

For ethical reasons, clear causal evidence from controlled human exposure studies still covers only effects in the first category. However, for this review there are results from epidemiological studies, upon which to base judgments about adversity, for effects in all of the categories. Statistically significant and robust associations have been reported in epidemiology studies falling into the second and third categories. These more serious effects include respiratory events (*e.g.*, triggering asthma attacks) that may require medication (*e.g.*, asthma), but not necessarily hospitalization, as well as respiratory hospital admissions and emergency department visits for respiratory causes. Less conclusive, but still positive associations have been reported for school absences and cardiovascular hospital admissions. Human health effects for which associations have been suggested through evidence from epidemiological and animal toxicology studies, but have not been conclusively demonstrated still fall primarily into the last two categories. In the 1997 review of the O₃ standard, evidence for these more serious effects came from studies of effects in laboratory animals. Evidence from animal studies evaluated in the 2006 Criteria Document strongly suggests that O₃ is capable of damaging the distal airways and proximal alveoli, resulting in lung tissue remodeling leading to apparently irreversible changes. Recent advancements of dosimetry modeling also provide a better basis for extrapolation from animals to humans. Information from epidemiological studies provides supporting, but limited evidence of irreversible respiratory effects in humans than was available in the prior review. Moreover, the findings from single-city and multicity time-series epidemiology studies and meta-analyses of these epidemiological studies are highly suggestive of an association between short-term O₃ exposure and mortality particularly in the warm season.

While O₃ has been associated with effects that are clearly adverse, application of these guidelines, in particular to the least serious category of effects related to ambient O₃ exposures, involves judgments about which medical experts on the CASAC panel and public commenters have expressed diverse views in the past. To help frame such judgments, EPA staff have defined specific ranges of functional responses

(*e.g.*, decrements in FEV₁ and airway responsiveness) and symptomatic responses (*e.g.*, cough, chest pain, wheeze), together with judgments as to the potential impact on individuals experiencing varying degrees of severity of these responses, that have been used in previous NAAQS reviews. These ranges of pulmonary responses and their associated potential impacts are summarized in Tables 3–2 and 3–3 of the 2007 Staff Paper.

For active healthy people, moderate levels of functional responses (*e.g.*, FEV₁ decrements of ≥ 10 percent but < 20 percent, lasting up to 24 hours) and/or moderate symptomatic responses (*e.g.*, frequent spontaneous cough, marked discomfort on exercise or deep breath, lasting up to 24 hours) would likely interfere with normal activity for relatively few responsive individuals. On the other hand, EPA staff determined that large functional responses (*e.g.*, FEV₁ decrements ≥ 20 percent, lasting longer than 24 hours) and/or severe symptomatic responses (*e.g.*, persistent uncontrollable cough, severe discomfort on exercise or deep breath, lasting longer than 24 hours) would likely interfere with normal activities for many responsive individuals. EPA staff determined that these would be considered adverse under ATS guidelines. In the context of standard setting, CASAC indicated that a focus on the mid to upper end of the range of moderate levels of functional responses (*e.g.*, FEV₁ decrements ≥ 15 percent but < 20 percent) is appropriate for estimating potentially adverse lung function decrements in active healthy people. However, for people with lung disease, even moderate functional (*e.g.*, FEV₁ decrements ≥ 10 percent but < 20 percent, lasting up to 24 hours) or symptomatic responses (*e.g.*, frequent spontaneous cough, marked discomfort on exercise or with deep breath, wheeze accompanied by shortness of breath, lasting up to 24 hours) would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication. For people with lung disease, large functional responses (*e.g.*, FEV₁ decrements ≥ 20 percent, lasting longer than 24 hours) and/or severe symptomatic responses (*e.g.*, persistent uncontrollable cough, severe discomfort on exercise or deep breath, persistent wheeze accompanied by shortness of breath, lasting longer than 24 hours) would likely interfere with normal activity for most individuals and would increase the likelihood that these individuals would seek medical treatment. In the context of standard

setting, the CASAC indicated (Henderson, 2006c) that a focus on the lower end of the range of moderate levels of functional responses (*e.g.*, FEV₁ decrements ≥ 10 percent) is most appropriate for estimating potentially adverse lung function decrements in people with lung disease.

In judging the extent to which these impacts represent effects that should be regarded as adverse to the health status of individuals, an additional factor that has been considered in previous NAAQS reviews is whether such effects are experienced repeatedly during the course of a year or only on a single occasion. While some experts would judge single occurrences of moderate responses to be a “nuisance,” especially for healthy individuals, a more general consensus view of the adversity of such moderate responses emerges as the frequency of occurrence increases.

The new guidance builds upon and expands the 1985 definition of adversity in several ways. There is an increased focus on quality of life measures as indicators of adversity. There is also a more specific consideration of population risk. Exposure to air pollution that increases the risk of an adverse effect to the entire population is adverse, even though it may not increase the risk of any individual to an unacceptable level. For example, a population of asthmatics could have a distribution of lung function such that no individual has a level associated with significant impairment. Exposure to air pollution could shift the distribution to lower levels that still do not bring any individual to a level that is associated with clinically relevant effects. However, this would be considered to be adverse because individuals within the population would have diminished reserve function, and therefore would be at increased risk if affected by another agent.

Of the various effects of O₃ exposure that have been studied, many would meet the ATS definition of adversity. Such effects include, for example, any detectible level of permanent lung function loss attributable to air pollution, including both reductions in lung growth or acceleration of the age-related decline of lung function; exacerbations of disease in individuals with chronic cardiopulmonary diseases; reversible loss of lung function in combination with the presence of symptoms; as well as more serious effects such as those requiring medical care including hospitalization and, obviously, mortality.

d. Size of At-Risk Populations

Although O₃-related health risk estimates may appear to be small, their significance from an overall public health perspective is determined by the large numbers of individuals in the population groups potentially at risk for O₃-related health effects discussed above. For example, a population of concern includes people with respiratory disease, which includes approximately 11 percent of U.S. adults and 13 percent of children who have been diagnosed with asthma and 6 percent of adults with chronic obstructive pulmonary disease (chronic bronchitis and/or emphysema) in 2002 and 2003 (Table 8–4 in the 2006 Criteria Document, section 8.7.5.2). More broadly, individuals with preexisting cardiopulmonary disease may constitute an additional population of concern, with potentially tens of millions of people included in each disease category. In addition, populations based on age group also comprise substantial segments of the population that may be potentially at risk for O₃-related health impacts. Based on U.S. census data from 2003, about 26 percent of the U.S. population are under 18 years of age and 12 percent are 65 years of age or older. Hence, large proportions of the U.S. population are included in life stages that are most likely to have increased susceptibility to the health effects of O₃ and/or those with the highest ambient O₃ exposures.

The 2006 Criteria Document (section 8.7.5.2) notes that 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 100,000 deaths from chronic lower respiratory diseases. For respiratory health diseases, there are nearly 4 million hospital discharges per year, 14 million emergency department visits, 112 million ambulatory care visits, and an estimated 700 million restricted activity days per year due to respiratory conditions from all causes per year. Applying small risk estimates for the O₃-related contribution to such health effects with relatively large baseline levels of health outcomes can result in quite large public health impacts related to ambient O₃ exposure. Thus, even a small percentage reduction in O₃ health impacts on cardiopulmonary diseases would reflect a large number of avoided cases. In considering this information together with the concentration-response relationships that have been observed between exposure to O₃ and various health endpoints, the 2006

Criteria Document (section 8.7.5.2) concludes that exposure to ambient O₃ likely has a significant impact on public health in the U.S.

B. Human Exposure and Health Risk Assessments

To put judgments about health effects that are adverse for individuals into a broader public health context, EPA has developed and applied models to estimate human exposures and health risks. This broader context includes consideration of the size of particular population groups at risk for various effects, the likelihood that exposures of concern will occur for individuals in such groups under varying air quality scenarios, estimates of the number of people likely to experience O₃-related effects, the variability in estimated exposures and risks, and the kind and degree of uncertainties inherent in assessing the exposures and risks involved.

As discussed below there are a number of important uncertainties that affect the exposure and health risk estimates. It is also important to note that there have been significant improvements in both the exposure and health risk model. CASAC expressed the view that the exposure analysis represents a state-of-the-art modeling approach and that the health risk assessment was “well done, balanced and reasonably communicated (Henderson, 2006c). While recognizing and considering the kind and degree of uncertainties in both the exposure and health risk estimates, the 2007 Staff Paper (pp. 6–20 to 6–21) judged that the quality of the estimates is such that they are suitable to be used as an input to the Administrator’s decisions on the O₃ primary standard.

In modeling exposures and health risks associated with just meeting the current and alternative O₃ standards, EPA has simulated air quality to represent conditions just meeting these standards based on O₃ air quality patterns in several recent years and on how the shape of the O₃ air quality distribution have changed over time based on historical trends in monitored O₃ air quality data. As described in the 2007 Staff Paper (EPA, 2007b, section 4.5.8) and discussed below, recent O₃ air quality distributions have been statistically adjusted to simulate just meeting the current and selected alternative standards. These simulations do not reflect any consideration of specific control programs or strategies designed to achieve the reductions in emissions required to meet the specified standards. Further, these simulations do not represent predictions of when,

whether, or how areas might meet the specified standards.²⁷

As noted in section I.C above, around the time of the release of the final 2007 Staff Paper in January 2007, EPA discovered a small error in the exposure model that when corrected resulted in slight increases in the simulated exposures. Since the exposure estimates are an input to the lung function portion of the health risk assessment, this correction also resulted in slight increases in the lung function risk estimates as well. The exposure and risk estimates discussed in this notice reflect the corrected estimates, and thus are slightly different than the exposure and risk estimates cited in the January 31, 2007 Staff Paper.²⁸

1. Exposure Analyses

a. Overview

As part of the 2008 rulemaking, the EPA conducted exposure analyses using a simulation model to estimate O₃ exposures for the general population, school age children (ages 5–18), and school age children with asthma living in 12 U.S. metropolitan areas representing different regions of the country where the then current 8-hour O₃ standard is not met. The emphasis on children reflects the finding of the 1997 O₃ NAAQS review that children are an important at-risk group. The 12 modeled areas combined represent a significant fraction of the U.S. urban population, 89 million people, including 18 million school age children of whom approximately 2.6 million have asthma. The selection of urban areas to include in the exposure analysis took into consideration the location of O₃ epidemiological studies, the availability of ambient O₃ data, and the desire to represent a range of geographic areas, population demographics, and O₃ climatology. These selection criteria are discussed further in chapter 5 of the 2007 Staff Paper (EPA, 2007b). The geographic extent of each modeled area consists of the census tracts in the combined statistical area (CSA) as defined by OMB (OMB, 2005).²⁹

²⁷ Modeling that projects whether and how areas might attain alternative standards in a future year is presented in the Regulatory Impact Analysis being prepared in connection with this rulemaking.

²⁸ EPA made available corrected versions of the final 2007 Staff Paper, and human exposure and health risk assessment technical support documents in July 2007 on the EPA Web site listed in the Availability of Related Information section of this notice.

²⁹ The 12 CSAs modeled are: Atlanta-Sandy Springs-Gainesville, GA-AL; Boston-Worcester-Manchester, MA-NH; Chicago-Naperville-Michigan City, IL-IN-WI; Cleveland-Akron-Elyria, OH; Detroit-Warren-Flint, MI; Houston-Baytown-Huntsville, TX; Los Angeles-Long Beach-Riverside,

Exposure estimates were developed using a probabilistic exposure model that is designed to explicitly model the numerous sources of variability that affect people's exposures. As discussed below, the model estimates population exposures by simulating human activity patterns, air conditioning prevalence, air exchange rates, and other factors. The modeled exposure estimates were developed for three recent years of ambient O₃ concentrations (2002, 2003, and 2004), as well as for O₃ concentrations adjusted to simulate conditions associated with just meeting the then current NAAQS and various alternative 8-hour standards based on the three year period 2002–2004.³⁰ This exposure assessment is more fully described and presented in the 2007 Staff Paper and in a technical support document, *Ozone Population Exposure Analysis for Selected Urban Areas* (EPA, 2007c; hereafter Exposure Analysis TSD). The scope and methodology for this exposure assessment were developed over the last few years with considerable input from the CASAC Ozone Panel and the public.³¹

The goals of the O₃ exposure assessment were: (1) To provide estimates of the size of at-risk populations exposed to various levels associated with recent O₃ concentrations, and with just meeting the current O₃ NAAQS and alternative O₃ standards, in specific urban areas; (2) to provide distributions of exposure estimates over the entire range of ambient O₃ concentrations as an important input to the lung function risk assessment summarized below in section II.B.2; (3) to develop a better understanding of the influence of various inputs and assumptions on the exposure estimates; and (4) to gain insight into the distribution of exposures and patterns of exposure

reductions associated with meeting alternative O₃ standards.

The EPA recognizes that there are many sources of variability and uncertainty inherent in the inputs to this assessment and that there is uncertainty in the resulting O₃ exposure estimates. With respect to variability, the exposure modeling approach accounts for variability in ambient O₃ levels, demographic characteristics, physiological attributes, activity patterns, and factors affecting microenvironmental (e.g., indoor) concentrations. In EPA's judgment, the most important uncertainties affecting the exposure estimates are related to the modeling of human activity patterns over an O₃ season, the modeling of variations in ambient concentrations near roadways, and the modeling of air exchange rates that affect the amount of O₃ that penetrates indoors. Another important uncertainty that affects the estimation of how many exposures are associated with moderate or greater exertion is the characterization of energy expenditure for children engaged in various activities. As discussed in more detail in the 2007 Staff Paper (EPA, 2007b, section 4.3.4.7), the uncertainty in energy expenditure values carries over to the uncertainty of the modeled breathing rates, which are important since they are used to classify exposures occurring at moderate or greater exertion which are the relevant exposures since O₃-related effects observed in controlled human exposure studies only are observed when individuals are engaged in some form of exercise. The uncertainties in the exposure model inputs and the estimated exposures have been assessed using quantitative uncertainty and sensitivity analyses. Details are discussed in the 2007 Staff Paper (section 4.6) and in a technical memorandum describing the exposure modeling uncertainty analysis (Langstaff, 2007).

b. Scope and Key Components

Population exposures to O₃ are primarily driven by ambient outdoor concentrations, which vary by time of day, location, and peoples' activities. Outdoor O₃ concentration estimates used in the exposure assessment are provided by measurements and statistical adjustments to the measured concentrations. The current exposure analysis allows comparisons of population exposures to O₃ within each urban area, associated with current O₃ levels and with O₃ levels just meeting several potential alternative air quality standards or scenarios. Human exposure, regardless of the pollutant,

depends on where individuals are located and what they are doing. Inhalation exposure models are useful in realistically estimating personal exposures to O₃ based on activity-specific breathing rates, particularly when recognizing that large scale population exposure measurement studies have not been conducted that are representative of the overall population or at risk subpopulations.

The model EPA used to simulate O₃ population exposure is the Air Pollutants Exposure Model (APEX), the human inhalation exposure model within the Total Risk Integrated Methodology (TRIM) framework (EPA, 2006c,d). APEX is conceptually based on the probabilistic NAAQS exposure model for O₃ (pNEM/O₃) used in the last O₃ NAAQS review. Since that time the model has been restructured, improved, and expanded to reflect conceptual advances in the science of exposure modeling and newer input data available for the model. Key improvements to algorithms include replacement of the cohort approach with a probabilistic sampling approach focused on individuals, accounting for fatigue and oxygen debt after exercise in the calculation of breathing rates, and a new approach for construction of longitudinal activity patterns for simulated persons. Major improvements to data input to the model include updated air exchange rates, more recent census and commuting data, and a greatly expanded daily time-activities database.

APEX is a probabilistic model designed to explicitly model the numerous sources of variability that affect people's exposures. APEX simulates the movement of individuals through time and space and estimates their exposures to O₃ in indoor, outdoor, and in-vehicle microenvironments. The exposure model takes into account the most significant factors contributing to total human O₃ exposure, including the temporal and spatial distribution of people and O₃ concentrations throughout an urban area, the variation of O₃ levels within each microenvironment, and the effects of exertion on breathing rate in exposed individuals. A more detailed description of APEX and its application is presented in chapter 4 of the 2007 Staff Paper and associated technical documents (EPA, 2006b,c,d).

Several methods have been used to evaluate the APEX model and to characterize the uncertainty of the model estimates. These include conducting model evaluation, sensitivity analyses, and a detailed uncertainty analysis for one urban area.

CA; New York-Newark-Bridgeport, NY-NJ-CT-PA; Philadelphia-Camden-Vineland, PA-NJ-DE-MD; Sacramento-Arden-Arcade-Truckee, CA-NV; St. Louis-St. Charles-Farmington, MO-IL; Washington-Baltimore-N. Virginia, DC-MD-VA-WV.

³⁰ All 12 of the CSAs modeled did not meet the 0.084 ppm O₃ NAAQS for the three year period examined.

³¹ The general approach used in the human exposure assessment was described in the draft Health Assessment Plan (EPA, 2005d) that was released to the CASAC and general public in April 2005 and was the subject of a consultation with the CASAC O₃ Panel on May 5, 2005. In October 2005, OAQPS released the first draft of the Staff Paper containing a chapter discussing the exposure analyses and first draft of the Exposure Analyses TSD for CASAC consultation and public review on December 8, 2005. In July 2006, OAQPS released the second draft of the Staff Paper and second draft of the Exposure Analyses TSD for CASAC review and public comment which was held by the CASAC O₃ Panel on August 24–25, 2006.

These are discussed fully in the 2007 Staff Paper (section 4.6) and in Langstaff (2007). The uncertainty of model structure was judged to be of lesser importance than the uncertainties of the model inputs and parameters. Model structure refers to the algorithms in APEX designed to simulate the processes that result in people's exposures, for example, the way that APEX models exposures to individuals when they are near roads. The uncertainties in the model input data (e.g., measurement error, ambient concentrations, air exchange rates, and activity pattern data) have been assessed individually, and their impact on the uncertainty in the modeled exposure estimates was assessed in a unified quantitative analysis with results expressed in the form of estimated confidence ranges around the estimated measures of exposure. This uncertainty analysis was conducted for one urban area (Boston) using the observed 2002 O₃ concentrations and 2002 concentrations adjusted to simulate just meeting the current standard, with the expectation that the results would be similar for other cities and years. One significant source of uncertainty, due to limitations in the database used to model peoples' daily activities, was not included in the unified analysis, and was assessed through separate sensitivity analyses. This analysis indicates that the uncertainty of the exposure results is relatively small. For example, 95 percent uncertainty intervals were calculated for the APEX estimates of the percent of children or asthmatic children with exposures above 0.060, 0.070, or 0.080 ppm under moderate exertion, for two air quality scenarios (current 2002 and 2002 adjusted to simulate just meeting the current standard) in Boston (Langstaff, 2007, Tables 26 and 27). The 95 percent uncertainty intervals for this set of 12 exposure estimates indicate the possibility of underpredictions of the exposure estimates ranging from 3 to 25 percent of the modeled estimates, and overpredictions ranging from 4 to 11 percent of the estimates. For example, APEX estimates the percent of asthmatic children with exposures above 0.070 ppm under moderate exertion to be 24 percent, for Boston 2002 O₃ concentrations adjusted to simulate just meeting the current standard. The 95 percent uncertainty interval for this estimate is 23 – 30 percent, or – 4 to +25 percent of the estimate. These uncertainty intervals do not include the uncertainty engendered by limitations of the activity database, which is in the range of one to ten percent.

The exposure periods modeled here are the O₃ seasons in 2002, 2003, and 2004. The O₃ season in each area includes the period of the year where elevated O₃ levels tend to be observed and for which routine hourly O₃ monitoring data are available. Typically this period spans from March or April through September or October, or in some areas, spanning the entire year. Three years were modeled to reflect the substantial year-to-year variability that occurs in O₃ levels and related meteorological conditions, and because the standard is specified in terms of a three-year period. The year-to-year variability observed in O₃ levels is due to a combination of different weather patterns and the variation in emissions of O₃ precursors. Nationally, 2002 was a relatively high year with respect to the 4th highest daily maximum 8-hour O₃ levels observed in urban areas across the U.S. (EPA, 2007b, Figure 2–16), with the mean of the distribution of O₃ levels for the urban monitors being in the upper third among the years 1990 through 2006. In contrast, on a national basis, 2004 is the lowest year on record through 2006 for this same air quality statistic, and 8-hour daily maximum O₃ levels observed in most, but not all of the 12 urban areas included in the exposure and risk analyses were relatively low compared to other recent years. The 4th highest daily maximum 8-hour O₃ levels observed in 2003 in the 12 urban areas and nationally generally were between those observed in 2002 and 2004.

Regulatory scenarios examined in the 2008 rulemaking include the then current 0.08 ppm, average of the 4th daily maximum 8-hour averages over a three year period standard; standards with the same form but with alternative levels of 0.080, 0.074, 0.070, and 0.064 ppm; standards specified as the average of the 3rd highest daily maximum 8-hour averages over a three year period with alternative levels of 0.084 and 0.074 ppm; and a standard specified as the average of the 5th highest daily maximum 8-hour averages over a three year period with a level of 0.074 ppm.³² The then current standard used a rounding convention that allows areas to have an average of the 4th daily maximum 8-hour averages as high as

³² The 8-hour O₃ standard established in 1997 was 0.08 ppm, but the rounding convention specified that the average of the 4th daily maximum 8-hour average concentrations over a three-year period must be at 0.084 ppm or lower to be in attainment of this standard. When EPA staff selected alternative standards to analyze, it was presumed that the same type of rounding convention would be used, and thus alternative standards of 0.084, 0.074, 0.064 ppm were chosen.

0.084 ppm and still meet the standard. All alternative standards analyzed were intended to reflect improved precision in the measurement of ambient concentrations (in ppm), where the precision would extend to three instead of two decimal places.

The then current standard and all alternative standards were modeled using a quadratic rollback approach to adjust the hourly concentrations observed in 2002–2004 to yield a design value³³ corresponding to the standard being analyzed. The quadratic rollback technique reduces higher concentrations more than lower concentrations near ambient background levels.³⁴ This procedure was considered in a sensitivity analysis in the 1997 review of the O₃ standard and has been shown to be more realistic than a linear, proportional rollback method, where all of the ambient concentrations are reduced by the same factor.

c. Exposure Estimates and Key Observations

The exposure assessment, which provides estimates of the number of people exposed to different levels of ambient O₃ while at specified exertion levels,³⁵ serve two purposes. First, the entire range of modeled personal exposures to ambient O₃ is an essential input to the portion of the health risk assessment based on exposure-response functions from controlled human exposure studies, discussed in the next section. Second, estimates of personal exposures to ambient O₃ concentrations at and above specific benchmark levels provide some perspective on the public

³³ A design value is a statistic that describes the air quality status of a given area relative to the level of the NAAQS. Design values are often based on multiple years of data, consistent with specification of the NAAQS in Part 50 of the CFR. For the 8-hour O₃ NAAQS, the 3-year average of the annual 4th-highest daily maximum 8-hour average concentrations, based on the monitor within (or downwind of) an urban area yielding the highest 3-year average, is the design value.

³⁴ The quadratic rollback approach and evaluation of this approach are described by Johnson (1997), Duff *et al.* (1998) and Rizzo (2005, 2006).

³⁵ As discussed above in Section II.A, O₃ health responses observed in controlled human exposure studies are associated with exposures while engaged in moderate or greater exertion and, therefore, these are the exposure measures of interest. The level of exertion of individuals engaged in particular activities is measured by an equivalent ventilation rate (EVR), ventilation normalized by body surface area (BSA, in m²), which is calculated as VE/BSA, where VE is the ventilation rate (liters/minute). Moderate and greater exertion levels were defined as EVR > 13 liters/min-m² (Whitfield *et al.*, 1996) to correspond to the exertion levels measured in most subjects studied in the controlled human exposure studies that reported health effects associated with 6.6 hour O₃ exposures.

health impacts of health effects that cannot currently be evaluated in quantitative risk assessments that may occur at current air quality levels, and the extent to which such impacts might be reduced by meeting the current and alternative standards. This is especially true when there are exposure levels at which it is known or can reasonably be inferred that specific O₃-related health effects are occurring. In this notice, exposures at and above these benchmark concentrations are referred to as “exposures of concern.”

It is important to note that although the analysis of “exposures of concern” was conducted using three discrete benchmark levels (*i.e.*, 0.080, 0.070, and 0.060 ppm), the concept is more appropriately viewed as a continuum with greater confidence and less uncertainty about the existence of health effects at the upper end and less confidence and greater uncertainty as one considers increasingly lower O₃ exposure levels. The EPA recognizes that there is no sharp breakpoint within the continuum ranging from at and above 0.080 ppm down to 0.060 ppm. In considering the concept of exposures of concern, it is important to balance concerns about the potential for health effects and their severity with the increasing uncertainty associated with our understanding of the likelihood of such effects at lower O₃ levels.

Within the context of this continuum, estimates of exposures of concern at discrete benchmark levels provide some perspective on the public health impacts of O₃-related health effects that have been demonstrated in controlled human exposure and toxicological studies but cannot be evaluated in quantitative risk assessments, such as lung inflammation, increased airway responsiveness, and changes in host defenses. They also help in understanding the extent to which such impacts have the potential to be reduced by meeting the current and alternative standards. In the selection of specific benchmark concentrations for this analysis, staff first considered the exposure level of 0.080 ppm, at which there is a substantial amount of controlled human exposure evidence demonstrating a range of O₃-related health effects including lung inflammation and airway responsiveness in healthy individuals. Thus, as in the 1997 review, this level was selected as a benchmark level for this assessment of exposures of concern. Evidence newly available in this review is the basis for identifying additional, lower benchmark levels of 0.070 and 0.060 ppm for this assessment.

More specifically, as discussed above in section II.A.2, evidence available from controlled human exposure and epidemiological studies indicates that people with asthma have larger and more serious effects than healthy individuals, including lung function, respiratory symptoms, increased airway responsiveness, and pulmonary inflammation, which has been shown to be a more sensitive marker than lung function responses. Further, a substantial new body of evidence from epidemiological studies shows associations with serious respiratory morbidity and cardiopulmonary mortality effects at O₃ levels that extend below 0.080 ppm. Additional, but very limited new evidence from controlled human exposure studies shows lung function decrements and respiratory symptoms in healthy subjects at an O₃ exposure level of 0.060 ppm. The selected benchmark level of 0.070 ppm reflects the new information that asthmatics have larger and more serious effects than healthy people and therefore controlled human exposure studies done with healthy subjects may underestimate effects in this group, as well as the substantial body of epidemiological evidence of associations with O₃ levels below 0.080 ppm. The selected benchmark level of 0.060 ppm additionally reflects the very limited new evidence from controlled human exposure studies that show lung function decrements and respiratory symptoms in some healthy subjects at the 0.060 ppm exposure level, recognizing that asthmatics are likely to have more serious responses and that lung function is not likely to be as sensitive a marker for O₃ effects as is lung inflammation.

The estimates of exposures of concern were reported in terms of both “people exposed” (the number and percent of people who experience a given level of O₃ concentrations, or higher, at least one time during the O₃ season in a given year) and “occurrences of exposure” (the number of times a given level of pollution is experienced by the population of interest, expressed in terms of person-days of occurrences). Estimating exposures of concern is important because it provides some indication of the potential public health impacts of a range of O₃-related health outcomes, such as lung inflammation, increased airway responsiveness, and changes in host defenses. These particular health effects have been demonstrated in controlled human exposure studies of healthy individuals to occur at levels as low as 0.080 ppm O₃, but have not been evaluated at lower

levels in controlled human exposure studies. The EPA did not include these effects in the quantitative risk assessment due to a lack of adequate information on the exposure-response relationships.

The 1997 O₃ NAAQS review estimated exposures associated with 1-hour heavy exertion, 1-hour moderate exertion, and 8-hour moderate exertion for children, outdoor workers, and the general population. The EPA’s analysis in the 1997 Staff Paper showed that exposure estimates based on the 8-hour moderate exertion scenario for children yielded the largest number of children experiencing exposures at or above exposures of concern. Consequently, EPA chose to focus on the 8-hour moderate and greater exertion exposures in all and asthmatic school age children in the current exposure assessment. While outdoor workers and other adults who engage in moderate or greater exertion for prolonged durations while outdoors during the day in areas experiencing elevated O₃ concentrations also are at risk for experiencing exposures associated with O₃-related health effects, EPA did not focus on quantitative estimates for these populations due to the lack of information about the number of individuals who regularly work or exercise outdoors. Thus, the exposure estimates presented here and in the 2007 Staff Paper are most useful for making relative comparisons across alternative air quality scenarios and do not represent the total exposures in all children or other groups within the general population associated with the air quality scenarios.

Population exposures to O₃ were estimated in 12 urban areas for 2002, 2003, and 2004 air quality, and also using O₃ concentrations adjusted to just meet the then current and several alternative standards. The estimates of 8-hour exposures of concern at and above benchmark levels of 0.080, 0.070, and 0.060 ppm aggregated across all 12 areas are shown in Table 1 for air quality scenarios just meeting the current and four alternative 8-hour average standards.³⁶ Table 1 provides estimates of the number and percent of school age children and asthmatic school age children exposed, with daily 8-hour maximum exposures at or above each O₃ benchmark level of exposures of concern, while at intermittent moderate or greater exertion and based on O₃ concentrations observed in 2002 and

³⁶The full range of quantitative exposure estimates associated with just meeting the 0.084 ppm and alternative O₃ standards are presented in chapter 4 and Appendix 4A of the 2007 Staff Paper.

2004. Table 1 summarizes estimates for 2002 and 2004 because these years reflect years that bracket relatively higher and lower O₃ levels, with year 2003 generally containing O₃ levels in between when considering the 12 urban areas modeled. This table also reports the percent change in the number of persons exposed when a given alternative standard is compared with the then current standard.

Key observations important in comparing exposure estimates associated with just meeting the current NAAQS and alternative standards under consideration include:

(1) As shown in Table 6–1 of the 2007 Staff Paper, the patterns of exposure in

terms of percentages of the population exceeding a given exposure level are very similar for the general population and for asthmatic and all school age (5–18) children, although children are about twice as likely to be exposed, based on the percent of the population exposed, at any given level.

(2) As shown in Table 1 below, the number and percentage of asthmatic and all school-age children aggregated across the 12 urban areas estimated to experience one or more exposures of concern decline from simulations of just meeting the then current 0.084 ppm standard to simulations of alternative 8-hour standards by varying amounts

depending on the benchmark level, the population subgroup considered, and the year chosen. For example, the estimated percentage of school age children experiencing one or more exposures ≥ 0.070 ppm, while engaged in moderate or greater exertion, during an O₃ season is about 18 percent of this population when the 0.084 ppm standard is met using the 2002 simulation; this is reduced to about 12, 4, 1, and 0.2 percent of children upon meeting alternative standards of 0.080, 0.074, 0.070, and 0.064 ppm, respectively (all specified in terms of the 4th highest daily maximum 8-hour average), using the 2002 simulation.

TABLE 1—NUMBER AND PERCENT OF ALL AND ASTHMATIC SCHOOL AGE CHILDREN IN 12 URBAN AREAS ESTIMATED TO EXPERIENCE 8-HOUR OZONE EXPOSURES ABOVE 0.080, 0.070, AND 0.060 PPM WHILE AT MODERATE OR GREATER EXERTION, ONE OR MORE TIMES PER SEASON, AND THE NUMBER OF OCCURRENCES ASSOCIATED WITH JUST MEETING ALTERNATIVE 8-HOUR STANDARDS BASED ON ADJUSTING 2002 AND 2004 AIR QUALITY DATA^{1 2}

Benchmark levels of exposures of concern (ppm)	8-Hour air quality standards ³ (ppm)	All children, ages 5–18 Aggregate for 12 urban areas Number of children exposed (% of all) [% reduction from 0.084 ppm standard]		Asthmatic children, ages 5–18 Aggregate for 12 urban areas Number of children exposed (% of group) [% reduction from 0.084 ppm standard]	
		2002	2004	2002	2004
0.080	0.084	700,000 (4%)	30,000 (0%)	110,000 (4%)	0 (0%)
	0.080	290,000 (2%) [70%]	10,000 (0%) [67%]	50,000 (2%) [54%]	0 (0%)
	0.074	60,000 (0%) [91%]	0 (0%) [100%]	10,000 (0%) [91%]	0 (0%)
	0.070	10,000 (0%) [98%]	0 (0%) [100%]	0 (0%) [100%]	0 (0%)
	0.064	0 (0%) [100%]	0 (0%) [100%]	0 (0%) [100%]	0 (0%)
0.070	0.084	3,340,000 (18%)	260,000 (1%)	520,000 (20%)	40,000 (1%)
	0.080	2,160,000 (12%) [35%]	100,000 (1%) [62%]	330,000 (13%) [36%]	10,000 (0%) [75%]
	0.074	770,000 (4%) [77%]	20,000 (0%) [92%]	120,000 (5%) [77%]	0 (0%) [100%]
	0.070	270,000 (1%) [92%]	0 (0%) [100%]	50,000 (2%) [90%]	0 (0%) [100%]
	0.064	30,000 (0.2%) [99%]	0 (0%) [100%]	10,000 (0.2%) [98%]	0 (0%) [100%]
0.060	0.084	7,970,000 (44%)	1,800,000 (10%)	1,210,000 (47%)	270,000 (11%)
	0.080	6,730,000 (37%) [16%]	1,050,000 (6%) [42%]	1,020,000 (40%) [16%]	150,000 (6%) [44%]
	0.074	4,550,000 (25%) [43%]	350,000 (2%) [80%]	700,000 (27%) [42%]	50,000 (2%) [81%]
	0.070	3,000,000 (16%) [62%]	110,000 (1%) [94%]	460,000 (18%) [62%]	10,000 (1%) [96%]
	0.064	950,000 (5%) [88%]	10,000 (0%) [99%]	150,000 (6%) [88%]	0 (0%) [100%]

¹ Moderate or greater exertion is defined as having an 8-hour average equivalent ventilation rate ≥ 13 l-min/m².

² Estimates are the aggregate results based on 12 combined statistical areas (Atlanta, Boston, Chicago, Cleveland, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC). Estimates are for the ozone season which is all year in Houston, Los Angeles and Sacramento and March or April to September or October for the remaining urban areas.

³ All standards summarized here have the same form as the 8-hour standard established in 1997 which is specified as the 3-year average of the annual 4th highest daily maximum 8-hour average concentrations must be at or below the concentration level specified. As described in the 2007 Staff Paper (EPA, 2007b, section 4.5.8), recent O₃ air quality distributions have been statistically adjusted to simulate just meeting the 0.084 ppm standard and selected alternative standards. These simulations do not represent predictions of when, whether, or how areas might meet the specified standards.

(3) Substantial year-to-year variability in exposure estimates is observed over the three-year modeling period. For example, the estimated number of school age children experiencing one or more exposures ≥ 0.070 ppm during an O₃ season when a 0.084 ppm standard is met in the 12 urban areas included in the analysis is 3.3, 1.0, or 0.3 million for the 2002, 2003, and 2004 simulations, respectively.

(4) There is substantial variability observed across the 12 urban areas in the percent of the population subgroups

estimated to experience exposures of concern. For example, when 2002 O₃ concentrations are simulated to just meet a 0.084 ppm standard, the aggregate 12 urban area estimate is 18 percent of all school age children are estimated to experience O₃ exposures ≥ 0.070 ppm (Table 1 below), while the range of exposure estimates in the 12 urban areas considered separately for all children range from 1 to 38 percent (EPA, 2007b, p. 4–48, Exhibit 2). There was also variability in exposure estimates among the modeled areas

when using the 2004 air quality simulation for the same scenario; however it was reduced and ranged from 0 to 7 percent in the 12 urban areas (EPA, 2007b, p. 4–60, Exhibit 8).

(5) Of particular note, as discussed above in section II.A of this notice, high inter-individual variability in responsiveness means that only a subset of individuals in these groups who are exposed at and above a given benchmark level would actually be expected to experience such adverse health effects.

(6) In considering these observations, it is important to take into account the variability, uncertainties, and limitations associated with this assessment, including the degree of uncertainty associated with a number of model inputs and uncertainty in the model itself, as discussed above.

2. Quantitative Health Risk Assessment

This section discusses the approach used to develop quantitative health risk estimates associated with exposures to O₃ building upon a more limited risk assessment that was conducted during the last review.³⁷ As part of the 1997 review, EPA conducted a health risk assessment that produced risk estimates for the number and percent of children and outdoor workers experiencing lung function and respiratory symptoms associated with O₃ exposures for 9 urban areas.³⁸ The risk assessment for the 1997 review also included risk estimates for excess respiratory-related hospital admissions related to O₃ concentrations for New York City. In the last review, the risk estimates played a significant role in both the staff recommendations and in the proposed and final decisions to revise the O₃ standards. The health risk assessment conducted for the current review builds upon the methodology and lessons learned from the prior review.

a. Overview

The updated health risk assessment conducted as part of the 2008 rulemaking includes estimates of (1) risks of lung function decrements in all and asthmatic school age children, respiratory symptoms in asthmatic children, respiratory-related hospital admissions, and non-accidental and cardiorespiratory-related mortality associated with recent ambient O₃ levels; (2) risk reductions and remaining risks associated with just meeting the then current 0.084 ppm 8-hour O₃ NAAQS; and (3) risk reductions and remaining risks associated with just meeting various alternative 8-hour O₃ NAAQS in a number of example urban areas. This risk assessment is more fully described and presented in chapter 5 of the 2007 Staff Paper and in a technical support document (TSD), *Ozone Health Risk Assessment for Selected Urban*

³⁷ The methodology, scope, and results from the risk assessment conducted in the last review are described in Chapter 6 of the 1996 Staff Paper (EPA, 1996) and in several technical reports (Whitfield *et al.*, 1996; Whitfield, 1997) and publication (Whitfield *et al.*, 1998).

³⁸ The 9 urban study areas included in the exposure and risk analyses conducted during the last review were: Chicago, Denver, Houston, Los Angeles, Miami, New York City, Philadelphia, St. Louis, and Washington, DC.

Areas (Abt Associates, 2007a, hereafter referred to as "Risk Assessment TSD"). The scope and methodology for this risk assessment were developed over the last few years with considerable input from the CASAC O₃ Panel and the public.³⁹ The information contained in these documents included specific criteria for the selection of health endpoints, studies, and locations to include in the assessment. In a peer review letter sent by CASAC to the Administrator documenting its advice in October 2006 (Henderson, 2006c), the CASAC O₃ Panel concluded that the risk assessment was "well done, balanced, and reasonably communicated" and that the selection of health endpoints for inclusion in the quantitative risk assessment was appropriate.

The goals of the risk assessment are: (1) To provide estimates of the potential magnitude of several morbidity effects and mortality associated with current O₃ levels, and with meeting the then current 0.084 ppm standard and alternative 8-hour O₃ standards in specific urban areas; (2) to develop a better understanding of the influence of various inputs and assumptions on the risk estimates; and (3) to gain insights into the distribution of risks and patterns of risk reductions associated with meeting alternative O₃ standards. The health risk assessment is intended to be dependent on and reflect the overall weight and nature of the health effects evidence discussed above in section II.A and in more detail in the 2006 Criteria Document and 2007 Staff Paper. While not independent of the overall evaluation of the health effects evidence, the quantitative health risk assessment provides additional insights regarding the relative public health implications associated with just meeting a 0.084 ppm standard and several alternative 8-hour standards.

The risk assessment covers a variety of health effects for which there is adequate information to develop quantitative risk estimates. However, as noted by CASAC (Henderson, 2007) and in the 2007 Staff Paper, there are a number of health endpoints (*e.g.*, increased lung inflammation, increased

³⁹ The general approach used in the health risk assessment was described in the draft Health Assessment Plan (EPA, 2005d) that was released to the CASAC and general public in April 2005 and was the subject of a consultation with the CASAC O₃ Panel on May 5, 2005. In October 2005, OAQPS released the first draft of the Staff Paper containing a chapter discussing the risk assessment and first draft of the Risk Assessment TSD for CASAC consultation and public review on December 8, 2005. In July 2006, OAQPS released the second draft of the Staff Paper and second draft of the Risk Assessment TSD for CASAC review and public comment which was held by the CASAC O₃ Panel on August 24–25, 2006.

airway responsiveness, impaired host defenses, increased medication usage for asthmatics, increased emergency department visits for respiratory causes, and increased school absences) for which there currently is insufficient information to develop quantitative risk estimates, but which are important to consider in assessing the overall public health impacts associated with exposures to O₃. These additional health endpoints are discussed above in section II.A.2 and are also taken into account in considering the level of exposures of concern in populations particularly at risk, discussed above in this notice.

There are two parts to the health risk assessment: One based on combining information from controlled human exposure studies with modeled population exposure and the other based on combining information from community epidemiological studies with either monitored or adjusted ambient concentrations levels. Both parts of the risk assessment were implemented within a new probabilistic version of TRIM.Risk, the component of EPA's Total Risk Integrated Methodology (TRIM) model framework that estimates human health risks.

The EPA recognizes that there are many sources of uncertainty and variability in the inputs to this assessment and that there is significant variability and uncertainty in the resulting O₃ risk estimates. As discussed in chapters 2, 5, and 6 of the 2007 Staff Paper, there is significant year-to-year and city-to-city variability related to the air quality data that affects both the controlled human exposure studies-based and epidemiological studies-based parts of the risk assessment. There are also uncertainties associated with the air quality adjustment procedure used to simulate just meeting various alternative standards. In the prior review, different statistical approaches using alternative functional forms (*i.e.*, quadratic, proportional, Weibull) were used to reflect how O₃ air quality concentrations have historically changed. Based on sensitivity analyses conducted in the prior review, the choice of alternative air quality adjustment procedures had only a modest impact on the risk estimates (EPA, 2007b, p. 6–20). With respect to uncertainties about estimated background concentrations, as discussed below and in the 2007 Staff Paper (section 5.4.3), alternative assumptions about background levels have a variable impact depending on the location, standard, and health endpoint analyzed.

With respect to the lung function part of the health risk assessment, key uncertainties include uncertainties in the exposure estimates, discussed above, and uncertainties associated with the shape of the exposure-response relationship, especially at levels below 0.08 ppm, 8-hour average, where only very limited data are available down to 0.04 ppm and there is an absence of data below 0.04 ppm (EPA, 2007b, pp. 6–20 to 6–21). Concerning the part of the risk assessment based on effects reported in epidemiological studies, important uncertainties include uncertainties (1) surrounding estimates of the O₃ coefficients for concentration-response relationships used in the assessment, (2) involving the shape of the concentration-response relationship and whether or not a population threshold or non-linear relationship exists within the range of concentrations examined in the studies, (3) related to the extent to which concentration-response relationships derived from studies in a given location and time when O₃ levels were higher or behavior and/or housing conditions were different provide accurate representations of the relationships for the same locations with lower air quality distributions and/or different behavior and/or housing conditions, and (4) concerning the possible role of co-pollutants which also may have varied between the time of the studies and the current assessment period. An important additional uncertainty for the mortality risk estimates is the extent to which the associations reported between O₃ and non-accidental and cardiorespiratory mortality actually reflect causal relationships.

As discussed below, some of these uncertainties have been addressed quantitatively in the form of estimated confidence ranges around central risk estimates; others are addressed through separate sensitivity analyses (e.g., the influence of alternative estimates for policy-relevant background levels) or are characterized qualitatively. For both parts of the health risk assessment, statistical uncertainty due to sampling error has been characterized and is expressed in terms of 95 percent credible intervals. The EPA recognizes that these credible intervals do not reflect all of the uncertainties noted above.

b. Scope and Key Components

The health risk assessment is based on the information evaluated in the 2006 Criteria Document. The risk assessment includes several categories of health effects and estimates risks associated with just meeting a 0.084

ppm standard and alternative 8-hour O₃ NAAQS and with several individual recent years of air quality (i.e., 2002, 2003, and 2004). The risk assessment considers the same alternative air quality scenarios that were examined in the human exposure analyses described above. Risk estimates were developed for up to 12 urban areas selected to illustrate the public health impacts associated with these air quality scenarios.⁴⁰ As discussed above in section II.B.1, the selection of urban areas was largely determined by identifying areas in the U.S. which represented a range of geographic areas, population demographics, and climatology; with an emphasis on areas that did not meet the then current 0.084 ppm 8-hour O₃ NAAQS and which included the largest areas with O₃ nonattainment problems. The selection criteria also included whether or not there were acceptable epidemiological studies available that reported concentration-response relationships for the health endpoints selected for inclusion in the assessment.

The short-term exposure related health endpoints selected for inclusion in the quantitative risk assessment include those for which the 2006 Criteria Document or the 2007 Staff Paper concluded that the evidence as a whole supports the general conclusion that O₃, acting alone and/or in combination with other components in the ambient air pollution mix, is either clearly causal or is judged to be likely causal. Some health effects met this criterion of likely causality, but were not included in the risk assessment for other reasons, such as insufficient exposure-response data or lack of baseline incidence data.

As discussed in the section above describing the exposure analysis, in order to estimate the health risks associated with just meeting various alternative 8-hour O₃ NAAQS, it is necessary to estimate the distribution of hourly O₃ concentrations that would occur under any given standard. Since compliance is based on a 3-year average, the amount of control has been applied to each year of data (i.e., 2002 to 2004) to estimate risks for a single O₃ season or single warm O₃ season, depending on the health effect, based on a simulation that adjusted each of these individual

years so that the three year period would just meet the specified standard.

Consistent with the risk assessment approach used in the last review, the risk estimates developed for both recent air quality levels and just meeting the then current 0.084 ppm standard and selected alternative 8-hour standards represent risks associated with O₃ levels attributable to anthropogenic sources and activities (i.e., risk associated with concentrations above “policy-relevant background”). Policy-relevant background O₃ concentrations used in the O₃ risk assessment were defined in chapter 2 of the 2007 Staff Paper (pp. 2–48–2–55) as the O₃ concentrations that would be observed in the U.S. in the absence of anthropogenic emissions of precursors (e.g., VOC, NO_x, and CO) in the U.S., Canada, and Mexico. The results of a global tropospheric O₃ model (GEOS-CHEM) have been used to estimate monthly background daily diurnal profiles for each of the 12 urban areas for each month of the O₃ season using meteorology for the year 2001. Based on the results of the GEOS-CHEM model, the Criteria Document indicates that background O₃ concentrations are generally predicted to be in the range of 0.015 to 0.035 ppm in the afternoon, and they are generally lower under conditions conducive to man-made O₃ episodes.⁴¹

This approach of estimating risks in excess of background is judged to be more relevant to policy decisions regarding ambient air quality standards than risk estimates that include effects potentially attributable to uncontrollable background O₃ concentrations. Sensitivity analyses examining the impact of alternative estimates for background on lung function and mortality risk estimates have been developed and are included in the 2007 Staff Paper and Risk Assessment TSD and key observations are discussed below. Further, CASAC noted the difficulties and complexities associated with available approaches to estimating policy-relevant background concentrations (Henderson, 2007).

In the first part of the risk assessment, lung function decrement, as measured by FEV₁, is the only health response that is based on data from controlled human exposure studies. As discussed above, there is clear evidence of a causal relationship between lung function decrements and O₃ exposures for school age children engaged in moderate

⁴⁰ The 12 urban areas are the same urban areas evaluated in the exposure analysis discussed in the prior section. However, for most of the health endpoints based on findings from epidemiological studies, the geographic areas and populations examined in the health risk assessment were limited to those counties included in the original epidemiological studies that served as the basis for the concentration-response relationships.

⁴¹ EPA notes that the estimated level of policy-relevant background O₃ used in the prior risk assessment was a single concentration of 0.04 ppm, which was the midpoint of the range of levels for policy-relevant background that was provided in the 1996 Criteria Document.

exertion based on numerous controlled human exposure and summer camp field studies conducted by various investigators. Risk estimates have been developed for O₃-related lung function decrements (measured as changes in FEV₁) for all school age children (ages 5 to 18) and a subset of this group, asthmatic school age children (ages 5 to 18), whose average exertion over an 8-hour period was moderate or greater. The exposure period and exertion level were chosen to generally match the exposure period and exertion level used in the controlled human exposure studies that were the basis for the exposure-response relationships. A combined data set including individual level data from the Folinsbee *et al.* (1988), Horstman *et al.* (1990), and McDonnell *et al.* (1991) studies, used in the previous risk assessment, and more recent data from Adams (2002, 2003a, 2006) have been used to estimate probabilistic exposure-response relationships for 8-hour exposures under different definitions of lung function response (*i.e.*, ≥ 10 , 15, and 20 percent decrements in FEV₁). As discussed in the 2007 Staff Paper (p. 5–27), while these specific controlled human exposure studies only included healthy adults aged 18–35, findings from other controlled human exposure studies and summer camp field studies involving school age children in at least six different locations in the northeastern United States, Canada, and Southern California indicated changes in lung function in healthy children similar to those observed in healthy adults exposed to O₃ under controlled chamber conditions.

Consistent with advice from CASAC (Henderson, 2006c), EPA has considered both linear and logistic functional forms in estimating the probabilistic exposure-response relationships for lung function responses. A Bayesian Markov Chain Monte Carlo approach, described in more detail in the Risk Assessment TSD, has been used that incorporates both model uncertainty and uncertainty due to sample size in the combined data set that served as the basis for the assessment. The EPA has chosen a model reflecting a 90 percent weighting on a logistic form and a 10 percent weighting on a linear form as the base case for the risk assessment. The basis for this choice is that the logistic form provides a very good fit to the combined data set, but a linear model cannot be entirely ruled out since there are only very limited data (*i.e.*, 30 subjects) at the two lowest exposure levels (*i.e.*, 0.040 and 0.060 ppm). The EPA has conducted a sensitivity analysis which

examines the impact on the lung function risk estimates of two alternative choices, an 80 percent logistic/20 percent linear split and a 50 percent logistic/50 percent linear split.

As noted above, risk estimates have been developed for three measures of lung function response (*i.e.*, ≥ 10 , 15, and 20 percent decrements in FEV₁). However, the 2007 Staff Paper and risk estimates summarized below focus on FEV₁ decrements ≥ 15 percent for all school age children and ≥ 10 percent for asthmatic school age children, consistent with the advice from CASAC (Henderson, 2006c) that these levels of response represent indicators of adverse health effects in these populations. The Risk Assessment TSD and 2007 Staff Paper present the broader range of risk estimates including all three measures of lung function response.

Developing risk estimates for lung function decrements involved combining probabilistic exposure-response relationships based on the combined data set from several controlled human exposure studies with population exposure distributions for all and asthmatic school age children associated with recent air quality and air quality simulated to just meet the then current 0.084 ppm standard and alternative 8-hour O₃ NAAQS based on the results from the exposure analysis described in the previous section. The risk estimates have been developed for 12 large urban areas for the O₃ season.⁴² These 12 urban areas include approximately 18.3 million school age children, of which 2.6 million are asthmatic school age children.⁴³

In addition to uncertainties arising from sample size considerations, which are quantitatively characterized and presented as 95 percentile credible intervals, there are additional uncertainties and caveats associated with the lung function risk estimates. These include uncertainties about the shape of the exposure-response relationship, particularly at levels below 0.080 ppm, and about policy-relevant background levels, for which sensitivity analyses have been conducted. Additional important caveats and uncertainties concerning the lung function portion of the health risk assessment include: (1) The

⁴² As discussed above in section II.B.1, the urban areas were defined using the consolidated statistical areas definition and the total population residing in the 12 urban areas was approximately 88.5 million people.

⁴³ For 9 of the 12 urban areas, the O₃ season is defined as a period running from March or April to September or October. In 3 of the urban areas (Houston, Los Angeles, and Sacramento), the O₃ season is defined as the entire year.

uncertainties and limitations associated with the exposure estimates discussed above and (2) the inability to account for some factors which are known to affect the exposure-response relationships (*e.g.*, assigning healthy and asthmatic children the same responses as observed in healthy adult subjects and not adjusting response rates to reflect the increase and attenuation of responses that have been observed in studies of lung function responses upon repeated exposures). A more complete discussion of assumptions and uncertainties is contained in chapter 5 of the 2007 Staff Paper and in the Risk Assessment TSD.

The second part of the risk assessment is based on health effects observed in epidemiological studies. Based on a review of the evidence evaluated in the 2006 Criteria Document and 2007 Staff Paper, as well as the criteria discussed in chapter 5 of the 2007 Staff Paper, the following categories of health endpoints associated with short-term exposures to ambient O₃ concentrations were included in the risk assessment: respiratory symptoms in moderate to severe asthmatic children, hospital admissions for respiratory causes, and non-accidental and cardiorespiratory mortality. As discussed above, there is strong evidence of a causal relationship for the respiratory morbidity endpoints included in the risk assessment. With respect to nonaccidental and cardiorespiratory mortality, the 2006 Criteria Document concludes that there is strong evidence which is highly suggestive of a causal relationship between nonaccidental and cardiorespiratory-related mortality and O₃ exposures during the warm O₃ season. As discussed in the 2007 Staff Paper (chapter 5), EPA also recognizes that for some of the effects observed in epidemiological studies, such as increased respiratory-related hospital admissions and nonaccidental and cardiorespiratory mortality, O₃ may be serving as an indicator for reactive oxidant species in the overall photochemical oxidant mix and that these other constituents may be responsible in whole or part for the observed effects.

Risk estimates for each health endpoint category were only developed for areas that were the same or close to the location where at least one concentration-response function for the health endpoint had been estimated.⁴⁴

⁴⁴ The geographic boundaries for the urban areas included in this portion of the risk assessment were generally matched to the geographic boundaries used in the epidemiological studies that served as the basis for the concentration-response functions. In most cases, the urban areas were defined as

Thus, for respiratory symptoms in moderate to severe asthmatic children only the Boston urban area was included and four urban areas were included for respiratory-related hospital admissions. Nonaccidental mortality risk estimates were developed for 12 urban areas and 8 urban areas were included for cardiorespiratory mortality.

The concentration-response relationships used in the assessment are based on findings from human epidemiological studies that have relied on fixed-site ambient monitors as a surrogate for actual ambient O₃ exposures. In order to estimate the incidence of a particular health effect associated with recent air quality in a specific county or set of counties attributable to ambient O₃ exposures in excess of background, as well as the change in incidence corresponding to a given change in O₃ levels resulting from just meeting various 8-hour O₃ standards, three elements are required for this part of the risk assessment. These elements are: (1) Air quality information (including recent air quality data for O₃ from ambient monitors for the selected location, estimates of background O₃ concentrations appropriate for that location, and a method for adjusting the recent data to reflect patterns of air quality estimated to occur when the area just meets a given O₃ standard); (2) relative risk-based concentration-response functions that provide an estimate of the relationship between the health endpoints of interest and ambient O₃ concentration; and (3) annual or seasonal baseline health effects incidence rates and population data, which are needed to provide an estimate of the seasonal baseline incidence of health effects in an area before any changes in O₃ air quality.

A key component in the portion of the risk assessment based on epidemiological studies is the set of concentration-response functions which provide estimates of the relationships between each health endpoint of interest and changes in ambient O₃ concentrations. Studies often report more than one estimated concentration-response function for the same location and health endpoint. Sometimes models include different sets of co-pollutants and/or different lag periods between the ambient concentrations and reported health responses. For some health endpoints, there are studies that estimated multicity and single-city O₃ concentration-response functions. While the Risk Assessment TSD and chapter 5

of the 2007 Staff Paper present a more comprehensive set of risk estimates, EPA has focused on estimates based on multicity studies where available. As discussed in chapter 5 of the 2007 Staff Paper, the advantages of relying more heavily on concentration-response functions based on multicity studies include: (1) More precise effect estimates due to larger data sets, reducing the uncertainty around the estimated coefficient; (2) greater consistency in data handling and model specification that can eliminate city-to-city variation due to study design; and (3) less likelihood of publication bias or exclusion of reporting of negative or nonsignificant findings. Where studies reported different effect estimates for varying lag periods, consistent with the 2006 Criteria Document, single day lag periods of 0 to 1 days were used for associations with respiratory hospital admissions and mortality. For mortality associated with exposure to O₃ which may result over a several day period after exposure, distributed lag models, which take into account the contribution to mortality effects over several days, were used where available.

One of the most important elements affecting the uncertainties in the epidemiological-based portion of the risk assessment is the concentration-response relationships used in the assessment. The uncertainty resulting from the statistical uncertainty associated with the estimate of the O₃ coefficient in the concentration-response function was characterized either by confidence intervals or by Bayesian credible intervals around the corresponding point estimates of risk. Confidence and credible intervals express the range within which the true risk is likely to fall if the only uncertainty surrounding the O₃ coefficient involved sampling error. Other uncertainties, such as differences in study location, time period (*i.e.*, the years in which the study was conducted), and model uncertainties are not represented by the confidence or credible intervals presented, but were addressed by presenting estimates for different urban areas, by including risk estimates based on studies using different time periods and models, where available, and/or are discussed throughout section 5.3 of the 2007 Staff Paper. Because O₃ effects observed in the epidemiological studies have been more clearly and consistently shown for warm season analyses, all analyses for this portion of the risk assessment were carried out for the same time period, April through September.

The 2006 Criteria Document (p. 8–44) finds that no definitive conclusion can

be reached with regard to the existence of population thresholds in epidemiological studies. The EPA recognizes, however, the possibility that thresholds for individuals may exist for reported associations at fairly low levels within the range of air quality observed in the studies, but not be detectable as population thresholds in epidemiological analyses. Based on the 2006 Criteria Document's conclusions, EPA judged and CASAC concurred, that there is insufficient evidence to support use of potential population threshold levels in the quantitative risk assessment. However, EPA recognizes that there is increasing uncertainty about the concentration-response relationship at lower concentrations which is not captured by the characterization of the statistical uncertainty due to sampling error. Therefore, the risk estimates for respiratory symptoms in moderate to severe asthmatic children, respiratory-related hospital admissions, and premature mortality associated with exposure to O₃ must be considered in light of uncertainties about whether or not these O₃-related effects occur in these populations at very low O₃ concentrations.

With respect to variability within this portion of the risk assessment, there is variability among concentration-response functions describing the relation between O₃ and both respiratory-related hospital admissions and nonaccidental and cardiorespiratory mortality across urban areas. This variability is likely due to differences in population (*e.g.*, age distribution), population activities that affect exposure to O₃ (*e.g.*, use of air conditioning), levels and composition of co-pollutants, baseline incidence rates, and/or other factors that vary across urban areas. The risk assessment incorporates some of the variability in key inputs to the analysis by using location-specific inputs (*e.g.*, location-specific concentration-response functions, baseline incidence rates, and air quality data). Although spatial variability in these key inputs across all U.S. locations has not been fully characterized, variability across the selected locations is imbedded in the analysis by using, to the extent possible, inputs specific to each urban area.

c. Risk Estimates and Key Observations

The 2007 Staff Paper (chapter 5) and Risk Assessment TSD present risk estimates associated with just meeting the then current 0.084 ppm standard and several alternative 8-hour standards, as well as three recent years of air quality as represented by 2002,

either a single county or a few counties for this portion of the risk assessment.

2003, and 2004 monitoring data. As discussed in the exposure analysis section above, there is considerable city-to-city and year-to-year variability in the O₃ levels during this period, which results in significant variability in both portions of the health risk assessment.

In the 1997 risk assessment, risks for lung function decrements associated with 1-hour heavy exertion, 1-hour moderate exertion, and 8-hour moderate exertion exposures were estimated. Since the 8-hour moderate exertion exposure scenario for children clearly resulted in the greatest health risks in terms of lung function decrements, EPA

chose to include only the 8-hour moderate exertion exposures in the risk assessment for this health endpoint. Thus, the risk estimates presented here and in the 2007 Staff Paper are most useful for making relative comparisons across alternative air quality scenarios and do not represent the total risks for lung function decrements in children or other groups within the general population associated with any of the air quality scenarios. Thus, some outdoor workers and adults engaged in moderate exertion over multi-hour periods (e.g., 6–8 hour exposures) also

would be expected to experience similar lung function decrements. However, the percentage of each of these other subpopulations expected to experience these effects is expected to be smaller than all school age children who tend to spend more hours outdoors while active based on the exposure analyses conducted during the prior review.

Table 2 presents a summary of the risk estimates for lung function decrements for the 0.084 ppm standard set in 1997 and several alternative 8-hour standard levels with the same form.

TABLE 2—NUMBER AND PERCENT OF ALL AND ASTHMATIC SCHOOL AGE CHILDREN IN SEVERAL URBAN AREAS ESTIMATED TO EXPERIENCE MODERATE OR GREATER LUNG FUNCTION RESPONSES ONE OR MORE TIMES PER SEASON ASSOCIATED WITH 8-HOUR OZONE EXPOSURES ASSOCIATED WITH JUST MEETING ALTERNATIVE 8-HOUR STANDARDS BASED ON ADJUSTING 2002 AND 2004 AIR QUALITY DATA^{1 2}

8-Hour air quality standards ³	All children, ages 5–18 FEV ₁ ≥ 15 percent Aggregate for 12 urban areas Number of children affected (% of all) [% reduction from 0.084 ppm standard]		Asthmatic Children, ages 5–18 FEV ₁ ≥ 10 percent Aggregate for 5 urban areas Number of children affected (% of group) [% reduction from 0.084 ppm standard]	
	2002	2004	2002	2004
0.084 ppm (Standard set in 1997).	610,000 (3.3%)	230,000 (1.2%)	130,000 (7.8%)	70,000 (4.2%)
0.080 ppm	490,000 (2.7%) [20% reduction]	180,000 (1.0%) [22% reduction]	NA ⁴	NA
0.074 ppm	340,000 (1.9%) [44% reduction]	130,000 (0.7%) [43% reduction]	90,000 (5.0%) [31% reduction]	40,000 (2.7%) [43% reduction]
0.070 ppm	260,000 (1.5%) [57% reduction]	100,000 (0.5%) [57% reduction]	NA	NA
0.064 ppm	180,000 (1.0%) [70% reduction]	70,000 (0.4%) [70% reduction]	50,000 (3.0%) [62% reduction]	20,000 (1.5%) [71% reduction]

¹ Associated with exposures while engaged in moderate or greater exertion, which is defined as having an 8-hour average equivalent ventilation rate ≥ 13 l-min/m².

² Estimates are the aggregate central tendency results based on either 12 urban areas (Atlanta, Boston, Chicago, Cleveland, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC) or 5 urban areas (Atlanta, Chicago, Houston, Los Angeles, New York). Estimates are for the O₃ season which is all year in Houston, Los Angeles and Sacramento and March or April to September or October for the remaining urban areas.

³ All standards summarized here have the same form as the 8-hour standard set in 1997, which is specified as the 3-year average of the annual 4th highest daily maximum 8-hour average concentrations. As described in the 2007 Staff Paper (section 4.5.8), recent O₃ air quality distributions have been statistically adjusted to simulate just meeting the 0.084 ppm standard set in 1997 and selected alternative standards. These simulations do not represent predictions of when, whether, or how areas might meet the specified standards

⁴ NA (not available) indicates that EPA did not develop risk estimates for these scenarios for the asthmatic school age children population.

The estimates are for the aggregate number and percent of all school age children across 12 urban areas and the aggregate number and percent of asthmatic school age children across 5 urban areas⁴⁵ who are estimated to have at least 1 moderate or greater lung function response (defined as FEV₁ ≥ 15 percent in all children and ≥ 10 percent in asthmatic children) associated with 8-hour exposures to O₃ while engaged in moderate or greater exertion on average over the 8-hour period. The lung function risk estimates summarized in

Table 2 illustrate the year-to-year variability in both remaining risk associated with a relatively high year (i.e., based on adjusting 2002 O₃ air quality data) and relatively low year (based on adjusting 2004 O₃ air quality data) as well as the year-to-year variability in the risk reduction estimated to occur associated with various alternative standards relative to just meeting the then current 0.084 ppm standard. For example, it is estimated that about 610,000 school age children (3.2 percent of school age children) would experience 1 or more moderate lung function decrements for the 12 urban areas associated with O₃ levels just meeting a 0.084 ppm standard based on 2002 air quality data compared to 230,000 (1.2 percent of children)

associated with just meeting a 0.084 ppm standard based on 2004 air quality data.

As discussed in the 2007 Staff Paper, a child may experience multiple occurrences of a lung function response during the O₃ season. For example, upon meeting a 0.084 ppm 8-hour standard, the median estimates are that about 610,000 children would experience a moderate or greater lung function response 1 or more times for the aggregate of the 12 urban areas over a single O₃ season (based on the 2002 simulation), and that there would be almost 3.2 million total occurrences. Thus, on average it is estimated that there would be about 5 occurrences per O₃ season per responding child for air quality just meeting a 0.084 ppm 8-hour

⁴⁵ Due to time constraints, lung function risk estimates for asthmatic school age children were developed for only 5 of the 12 urban areas, and the areas were selected to represent different geographic regions. The 5 areas were: Atlanta, Chicago, Houston, Los Angeles, and New York City.

standard across the 12 urban areas. While the estimated number of occurrences per O₃ season is lower when based on the 2004 simulation than for the 2002 simulation, the estimated number of occurrences per responding child is similar. The EPA recognizes that some children in the population might have only 1 or 2 occurrences while others may have 6 or more occurrences per O₃ season. Risk estimates based on adjusting 2003 air quality to simulate just meeting the a 0.084 ppm standard and alternative 8-hour standards are intermediate to the estimates presented in Table 2 above in this notice and are presented in the 2007 Staff Paper (chapter 5) and Risk Assessment TSD.

For just meeting a 0.084 ppm 8-hour standard, Table 5–8 in the 2007 Staff Paper shows that median estimates across the 12 urban areas for all school age children experiencing 1 or more moderate lung function decrements ranges from 0.9 to 5.4 percent based on the 2002 simulation and from 0.8 to 2.2 percent based on the 2004 simulation. Risk estimates for each urban area included in the assessment, for each of the three years analyzed, and for additional alternative standards are presented in chapter 5 of the 2007 Staff Paper and in the Risk Assessment TSD.

For just meeting a 0.084 ppm 8-hour standard, the median estimates across the 5 urban areas for asthmatic school age children range from 3.4 to 10.9 percent based on the 2002 simulation and from 3.2 to 6.9 percent based on the 2004 simulation.

Key observations important in comparing estimated lung function risks associated with just meeting the 0.084 ppm NAAQS and alternative standards under consideration include:

(1) As discussed above, there is significant year to year variability in the range of median estimates of the number of school age children (ages 5–18) estimated to experience at least one FEV₁ decrement \geq 15 percent due to 8-hour O₃ exposures across the 12 urban areas analyzed, and similarly across the 5 urban areas analyzed for asthmatic school age children (ages 5–18) estimated to experience at least one FEV₁ decrement \geq 10 percent, when various 8-hour standards are just met.

(2) For asthmatic school age children, the median estimates of occurrences of FEV₁ decrements \geq 10% range from 52,000 to nearly 510,000 responses associated with just meeting a 0.084 ppm standard (based on the 2002 simulation) and range from 61,000 to about 240,000 occurrences (based on the 2004 simulation). These risk estimates would be reduced to a range of 14,000

to about 275,000 occurrences (2002 simulation) and to about 18,000 to nearly 125,000 occurrences (2004 simulation) upon just meeting the most stringent alternative 8-hour standard (0.064 ppm, 4th highest). The average number of occurrences per asthmatic child in an O₃ season ranged from about 6 to 11 associated with just meeting a 0.084 ppm standard (2002 simulation). The average number of occurrences per asthmatic child ranged from 4 to 12 upon meeting the most stringent alternative examined (0.064 ppm, 4th-highest) based on the 2002 simulation. The number of occurrences per asthmatic child is similar for the scenarios based on the 2004 simulation.

As discussed above, several epidemiological studies have reported increased respiratory morbidity outcomes (*e.g.*, respiratory symptoms in moderate to severe asthmatic children, respiratory-related hospital admissions) and increased nonaccidental and cardiorespiratory mortality associated with exposure to ambient O₃ concentrations. The results and key observations from this portion of the risk assessment are presented below:

(1) Estimates for increased respiratory symptoms (*i.e.*, chest tightness, shortness of breath, and wheeze) in moderate/severe asthmatic children (ages 0–12) were developed for the Boston urban area only. The median estimated number of days involving chest tightness (using the concentration-response relationship with only O₃ in the model) is about 6,100 (based on the 2002 simulation) and about 4,500 (based on the 2004 simulation) upon meeting a 0.084 ppm 8-hour standard and this is reduced to about 4,600 days (2002 simulation) and 3,100 days (2004 simulation) upon meeting the most stringent alternative examined (0.064 ppm, 4th-highest daily maximum 8-hour average). This corresponds to 11 percent (2002 simulation) and 8 percent (2004 simulation) of total incidence of chest tightness upon meeting a 0.084 ppm 8-hour standard and to about 8 percent (2002 simulation) and 5.5 percent (2004 simulation) of total incidence of chest tightness upon meeting a 0.064 ppm, 4th-highest daily maximum 8-hour average standard. Similar patterns of effects and reductions in effects are observed for each of the respiratory symptoms examined.

(2) The 2007 Staff Paper and Risk Assessment TSD present unscheduled hospital admission risk estimates for respiratory illness and asthma in New York City associated with short-term exposures to O₃ concentrations in excess of background levels from April

through September for several recent years (2002, 2003, and 2004) and upon just meeting a 0.084 ppm standard and alternative 8-hour standards based on simulating O₃ levels using 2002–2004 O₃ air quality data. For total respiratory illness, EPA estimates about 6.4 cases per 100,000 relevant population (2002 simulation) and about 4.6 cases per 100,000 relevant population (2004 simulation), which represents 1.5 percent (2002 simulation) and 1.0 percent (2004 simulation) of total incidence or about 510 cases (2002 simulation) and about 370 cases (2004 simulation) upon just meeting a 0.084 ppm 8-hour standard. For asthma-related hospital admissions, which are a subset of total respiratory illness admissions, the estimates are about 5.5 cases per 100,000 relevant population (2002 simulation) and about 3.9 cases per 100,000 relevant population (2004 simulation), which represents about 3.3 percent (2002 simulation) and 2.4 percent (2004 simulation) of total incidence or about 440 cases (2002) and about 310 cases (2004) for this same air quality scenario.

For increasingly more stringent alternative 8-hour standards, there is a gradual reduction in respiratory illness cases per 100,000 relevant population from 6.4 cases per 100,000 upon just meeting a 0.084 ppm 8-hour standard to 4.6 cases per 100,000 under the most stringent 8-hour standard (*i.e.*, 0.064 ppm, average 4th-highest daily maximum) analyzed based on the 2002 simulation. Similarly, based on the 2004 simulation there is a gradual reduction from 4.6 cases per 100,000 relevant population upon just meeting a 0.084 ppm 8-hour standard to 3.0 cases per 100,000 under a 0.064 ppm, average 4th-highest daily maximum standard.

Additional respiratory-related hospital admission estimates for three other locations are provided in the Risk Assessment TSD. The EPA notes that the concentration-response functions for each of these locations examined different outcomes in different age groups (*e.g.*, > age 30 in Los Angeles, > age 64 in Cleveland and Detroit, vs. all ages in New York City), making comparison of the risk estimates across the areas very difficult.

(3) Based on the median estimates for incidence for nonaccidental mortality (based on the Bell *et al.* (2004) 95 cities concentration-response function), meeting the most stringent standard (0.064 ppm) is estimated to reduce mortality by 40 percent of what it would be associated with just meeting a 0.084 ppm standard (based on the 2002 simulation). The patterns for cardiorespiratory mortality are similar.

The aggregate O₃-related cardiorespiratory mortality upon just meeting the most stringent standard shown is estimated to be about 42 percent of what it would be upon just meeting a 0.084 ppm standard, using simulated O₃ concentrations that just meet a 0.084 ppm standard and alternative 8-hour standards based on the 2002 simulation. Using the 2004 simulation, the corresponding reductions show a similar pattern but are somewhat greater.

(4) Much of the contribution to the risk estimates for non-accidental and cardiorespiratory mortality upon just meeting a 0.084 ppm 8-hour standard is associated with 24-hour O₃ concentrations between background and 0.040 ppm. Based on examining relationships between 24-hour concentrations averaged across the monitors within an urban area and 8-hour daily maximum concentrations, 8-hour daily maximum levels at the highest monitor in an urban area associated with these averaged 24-hour levels are generally about twice as high as the 24-hour levels. Thus, most O₃-related nonaccidental mortality is estimated to occur when O₃ concentrations are between background and when the highest monitor in the urban area is at or below 0.080 ppm, 8-hour average concentration.

The discussion below highlights additional observations and insights from the O₃ risk assessment, together with important uncertainties and limitations.

(1) As discussed in the 2007 Staff Paper (section 5.4.5), EPA has greater confidence in relative comparisons in risk estimates between alternative standards than in the absolute magnitude of risk estimates associated with any particular standard.

(2) Significant year-to-year variability in O₃ concentrations combined with the use of a 3-year design value to determine the amount of air quality adjustment to be applied to each year analyzed, results in significant year-to-year variability in the annual health risk estimates upon just meeting various 8-hour standards.

(3) There is noticeable city-to-city variability in estimated O₃-related incidence of morbidity and mortality across the 12 urban areas analyzed for both recent years of air quality and for air quality adjusted to simulate just meeting a 0.084 ppm standard and selected potential alternative standards. This variability is likely due to differences in air quality distributions, differences in exposure related to many factors including varying activity patterns and air exchange rates,

differences in baseline incidence rates, and differences in susceptible populations and age distributions across the 12 urban areas.

(4) With respect to the uncertainties about estimated policy-relevant background concentrations, as discussed in the 2007 Staff Paper (section 5.4.3), alternative assumptions about background levels had a variable impact depending on the health effect considered and the location and standard analyzed in terms of the absolute magnitude and relative changes in the risk estimates. There was relatively little impact on either absolute magnitude or relative changes in lung function risk estimates due to alternative assumptions about background levels. With respect to O₃-related non-accidental mortality, while notable differences (*i.e.*, greater than 50 percent)⁴⁶ were observed for nonaccidental mortality in some areas, particularly for more stringent standards, the overall pattern of estimated reductions, expressed in terms of percentage reduction relative to the 0.084 ppm standard, was significantly less impacted.

C. Reconsideration of the Level of the Primary Standard

1. Evidence and Exposure/Risk-Based Considerations

The approach used in the 2007 Staff Paper as a basis for staff recommendations on standard levels builds upon and broadens the general approach used by EPA in the 1997 review. This approach reflects the more extensive and stronger body of evidence available for the 2008 rulemaking on a broader range of health effects associated with exposure to O₃, including: (1) Additional respiratory-related endpoints; (2) new information about the mechanisms underlying respiratory morbidity effects supporting a judgment that the link between O₃ exposure and these effects is causal; (3) newly identified cardiovascular-related health endpoints from animal toxicology and controlled human exposures studies that are highly suggestive that O₃ can directly or indirectly contribute to cardiovascular morbidity, and (4) new U.S. multicity time series studies, single city studies, and several meta-analyses of these

⁴⁶ For example, assuming lower background levels resulted in increased estimates of non-accidental mortality incidence per 100,000 that were often 50 to 100 percent greater than the base case estimates; assuming higher background levels resulted in decreased estimates of non-accidental mortality incidence per 100,000 that were less than the base case estimates by 50 percent or more in many of the areas.

studies that provide relatively strong evidence for associations between short-term O₃ exposures and all-cause (nonaccidental) mortality, at levels below the current primary standard: As well as (5) a substantial body of new evidence of increased susceptibility in people with asthma and other lung diseases. In evaluating evidence-based and exposure/risk-based considerations, the 2007 Staff Paper considered: (1) The ranges of levels of alternative standards that are supported by the evidence, and the uncertainties and limitations in that evidence and (2) the extent to which specific levels of alternative standards reduce the estimated exposures of concern and risks attributable to O₃ and other photochemical oxidants, and the uncertainties associated with the estimated exposure and risk reductions.

a. Evidence-Based Considerations

In taking into account evidence-based considerations, the 2007 Staff Paper evaluated available evidence from controlled human exposure studies and epidemiological studies, as well as the uncertainties and limitations in that evidence. In particular, it focused on the extent to which controlled human exposure studies provide evidence of lowest-observed-effects levels and the extent to which epidemiological studies provide evidence of associations that extend down to the lower levels of O₃ concentrations observed in the studies or some indication of potential effect thresholds in terms of 8-hour average O₃ concentrations.

The most certain evidence of adverse health effects from exposure to O₃ comes from the controlled human exposure studies, as discussed above in section II.A.2, and the large bulk of this evidence derives from studies of exposures at levels of 0.080 ppm and above. At those levels, there is consistent evidence of lung function decrements and respiratory symptoms in healthy young adults, as well as evidence of inflammation and other medically significant airway responses.

Two studies by Adams (2002, 2006), newly available for consideration in the 2008 rulemaking, are the only available controlled human exposure studies that examine respiratory effects associated with prolonged O₃ exposures at levels below 0.080 ppm, which was the lowest exposure level that had been examined in the 1997 review. As discussed above in section II.A.2.a.i.(a)(i), the Adams (2006) study investigated a range of exposure levels, including 0.060 and 0.080 ppm O₃, and analyzed hour-by-hour changes in responses, including lung function (measured in term of decrements in FEV₁) and respiratory

symptoms, to investigate the effects of different patterns of exposure. At the 0.060 ppm exposure level, the author reported no statistically significant differences for lung function decrements; statistically significant responses were reported for total subjective respiratory symptoms toward the end of the exposure period for one exposure pattern. The EPA's reanalysis (Brown, 2007) of the data from the Adams (2006) study addressed the more fundamental question of whether there were statistically significant changes in lung function from a 6.6-hour exposure to 0.060 ppm O₃ versus filtered air and used a standard statistical method appropriate for a simple paired comparison. This reanalysis found small group mean lung function decrements in healthy adults at the 0.060 ppm exposure level to be statistically significantly different from responses associated with filtered air exposure.

Moreover, the Adams' studies also report a small percentage of subjects (7 to 20 percent) experienced lung function decrements (> 10 percent) at the 0.060 ppm exposure level. This is a concern because, for active healthy people, moderate levels of functional responses (e.g., FEV₁ decrements of > 10% but < 20%) and/or moderate respiratory symptom responses would likely interfere with normal activity for relatively few responsive individuals. However, for people with lung disease, even moderate functional or symptomatic responses would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication. In the context of standard setting, the CASAC indicated (Henderson, 2006c) that a focus on the lower end of the range of moderate levels of functional responses (e.g., FEV₁ decrements ≥ 10%) is most appropriate for estimating potentially adverse lung function decrements in people with lung disease. Therefore, the results of the Adams studies which indicate that a small percentage of healthy, non-asthmatic subjects are likely to experience FEV₁ decrements ≥ 10% when exposed to 0.060 ppm O₃ have implications for setting a standard that protects public health, including the health of sensitive populations such as asthmatics, with an adequate margin of safety.

In considering these most recent controlled human exposure studies, the 2007 Staff Paper concluded that these studies provide evidence of a lowest-observed-effects level of 0.060 ppm for potentially adverse lung function decrements and respiratory symptoms in some healthy adults while at prolonged moderate exertion. It further

concluded that since people with asthma, particularly children, have been found to be more sensitive and to experience larger decrements in lung function in response to O₃ exposures than would healthy adults, the 0.060 ppm exposure level also can be interpreted as representing a level likely to cause adverse lung function decrements and respiratory symptoms in children with asthma and more generally in people with respiratory disease.

In considering controlled human exposure studies of pulmonary inflammation, airway responsiveness, and impaired host defense capabilities, discussed above in section II.A.2.a.i, the 2007 Staff Paper noted that these studies provide evidence of a lowest-observed-effects level for such effects in healthy adults at prolonged moderate exertion of 0.080 ppm, the lowest level tested. Moreover there is no evidence that the 0.080 ppm level is a threshold for these effects. Studies reporting inflammatory responses and markers of lung injury have clearly demonstrated that there is significant variation in response of subjects exposed, even to O₃ exposures at 0.080 ppm. One study showed notable interindividual variability in young healthy adult subjects in most of the inflammatory and cellular injury indicators analyzed at 0.080 ppm. This inter-individual variability suggests that some portion of the population would likely experience such effects at exposure levels extending well below 0.080 ppm.

As discussed above, these physiological effects have been linked to aggravation of asthma and increased susceptibility to respiratory infection, potentially leading to increased medication use, increased school and work absences, increased visits to doctors' offices and emergency departments, and increased hospital admissions. Further, pulmonary inflammation is related to increased cellular permeability in the lung, which may be a mechanism by which O₃ exposure can lead to cardiovascular system effects, and to potential chronic effects such as chronic bronchitis or long-term damage to the lungs that can lead to reduced quality of life. These are all indicators of adverse O₃-related morbidity effects, which are consistent with and lend plausibility to the adverse morbidity effects and mortality effects observed in epidemiological studies.

Significant associations between ambient O₃ exposures and a wide variety of respiratory symptoms and other morbidity outcomes (e.g., asthma medication use, school absences, emergency department visits, and

hospital admissions) have been reported in epidemiological studies, as discussed above in section II.A.2.a.i. Overall, the 2006 Criteria Document concludes that positive and robust associations were found between ambient O₃ concentrations and various respiratory disease hospitalization outcomes, when focusing particularly on results of warm-season analyses. Recent studies also generally indicate a positive association between O₃ concentrations and emergency department visits for asthma during the warm season. These positive and robust associations are supported by the controlled human exposure, animal toxicological, and epidemiological evidence for lung function decrements, increased respiratory symptoms, airway inflammation, and increased airway responsiveness. Taken together, the overall evidence supports a causal relationship between acute ambient O₃ exposures and increased respiratory morbidity outcomes resulting in increased emergency department visits and hospitalizations during the warm season (EPA, 2006a, p. 8–77).

Moreover, many single- and multicity epidemiological studies observed positive associations of ambient O₃ concentrations with total nonaccidental and cardiopulmonary mortality. As discussed above in section II.A.2.b.i, the 2006 Criteria Document finds that the results from U.S. multicity time-series studies provide the strongest evidence to date for O₃ effects on acute mortality. Recent meta-analyses also indicate positive risk estimates that are unlikely to be confounded by PM; however, future work is needed to better understand the influence of model specifications on the magnitude of risk. The 2006 Criteria Document concludes that the "positive O₃ effects estimates, along with the sensitivity analyses in these three meta-analyses, provide evidence of a robust association between ambient O₃ and mortality" (EPA, 2006a, p. 7–97). In summary, the 2006 Criteria Document (p. 8–78) concludes that these findings are highly suggestive that short-term O₃ exposure directly or indirectly contribute to non-accidental and cardiopulmonary-related mortality, but additional research is needed to more fully establish underlying mechanisms by which such effects occur.

The 2007 Staff Paper considered the epidemiological studies to evaluate evidence related to potential effects thresholds at the population level for morbidity and mortality effects. As discussed above in section II.A.3.a (and more fully in the 2007 Staff Paper in chapter 3 and the 2006 Criteria

Document in chapter 7), a number of time-series studies have used statistical modeling approaches to evaluate potential thresholds at the population level. A few such studies reported some suggestive evidence of possible thresholds for morbidity and mortality outcomes in terms of 24-hour, 8-hour, and 1-hour averaging times. These results, taken together, provide some indication of possible 8-hour average threshold levels from below about 0.025 to 0.035 ppm (within the range of background concentrations) up to approximately 0.050 ppm. Other studies, however, observe linear concentration-response functions suggesting no effect threshold. The 2007 Staff Paper (p.6–60) concluded that the statistically significant associations between ambient O₃ concentrations and lung function decrements, respiratory symptoms, indicators of respiratory morbidity including increase emergency department visits and hospital admissions, and possibly mortality reported in a large number of studies likely extend down to ambient O₃ concentrations that are well below the level of the then current standard (0.084 ppm). These associations also extend well below the level of the standard set in 2008 (0.075 ppm) in that the highest level at which there is any indication of a threshold is approximately 0.050 ppm. Toward the lower end of the range of O₃ concentrations observed in such studies, ranging down to background levels (*i.e.*, 0.035 to 0.015 ppm), however, the 2007 Staff Paper stated that there is increasing uncertainty as to whether the observed associations remain plausibly related to exposures to ambient O₃, rather than to the broader mix of air pollutants present in the ambient atmosphere.

The 2007 Staff Paper also considered studies that did subset analyses, which included only days with ambient O₃ concentrations below the level of the then current standard, or below even lower O₃ concentrations, and continue to report statistically significant associations. Notably, as discussed above, Bell *et al.* (2006) conducted a subset analysis that continued to show statistically significant mortality associations even when only days with a maximum 8-hour average O₃ concentration below a value of approximately 0.061 ppm were included.⁴⁷ Also of note is the large multicity NCICAS (Mortimer *et al.*,

2002) that reported statistically significant associations between ambient O₃ concentrations and lung function decrements even when days with 8-hour average O₃ levels greater than 0.080 ppm were excluded (which consisted of less than 5 percent of the days in the eight urban areas in the study).

Further, as discussed above in section II.A.3.a, there are limitations in epidemiological studies that make discerning thresholds in populations difficult, including low data density in the lower concentration ranges, the possible influence of exposure measurement error, and interindividual differences in susceptibility to O₃-related effects in populations. There is the possibility that thresholds for individuals may exist in reported associations at fairly low levels within the range of air quality observed in the studies but not be detectable as population thresholds in epidemiological analyses.

Based on the above considerations, the 2007 Staff Paper recognized that the available evidence neither supports nor refutes the existence of effect thresholds at the population level for morbidity and mortality effects, and that if a population threshold level does exist, it would likely be well below the level of the then current standard and possibly within the range of background levels. Taken together, these considerations also support the conclusion that if a population threshold level does exist, it would likely be well below the level of the 0.075 ppm, 8-hour average, standard set in 2008.

In looking more broadly at evidence from animal toxicological, controlled human exposure, and epidemiological studies, the 2006 Criteria Document found substantial evidence, newly available in the 2008 rulemaking, that people with asthma and other preexisting pulmonary diseases are among those at increased risk from O₃ exposure. Altered physiological, morphological, and biochemical states typical of respiratory diseases like asthma, COPD, and chronic bronchitis may render people sensitive to additional oxidative burden induced by O₃ exposure (EPA, 2006a, section 8.7). Children and adults with asthma are the groups that have been studied most extensively. Evidence from controlled human exposure studies indicates that asthmatics may exhibit larger lung function decrements in response to O₃ exposure than healthy controls. As discussed more fully in section II.A.4 above, asthmatics present a different response profile for cellular, molecular, and biochemical parameters (EPA,

2006a, Figure 8–1) that are altered in response to acute O₃ exposure. They can have larger inflammatory responses, as manifested by larger increases in markers of inflammation such as white blood cells (*e.g.*, PMNs) or inflammatory cytokines. Asthmatics, and people with allergic rhinitis, are more likely to have an allergic-type response upon exposure to O₃, as manifested by increases in white blood cells associated with allergy (*i.e.*, eosinophils) and related molecules, which increase inflammation in the airways. The increased inflammatory and allergic responses also may be associated with the larger late-phase responses that asthmatics can experience, which can include increased bronchoconstrictor responses to irritant substances or allergens and additional inflammation.

In addition to the experimental evidence of lung function decrements, respiratory symptoms, and other respiratory effects in asthmatic populations, two large U.S. epidemiological studies as well as several smaller U.S. and international studies, have reported fairly robust associations between ambient O₃ concentrations and measures of lung function and daily respiratory symptoms (*e.g.*, chest tightness, wheeze, shortness of breath) in children with moderate to severe asthma and between O₃ and increased asthma medication use (EPA, 2007a, chapter 6). These more serious responses in asthmatics and others with lung disease provide biological plausibility for the respiratory morbidity effects observed in epidemiological studies, such as emergency department visits and hospital admissions.

The body of evidence from controlled human exposure and epidemiological studies, which includes asthmatic as well as non-asthmatic subjects, indicates that controlled human exposure studies of lung function decrements and respiratory symptoms that evaluate only healthy, non-asthmatic subjects likely underestimate the effects of O₃ exposure on asthmatics and other susceptible populations. Therefore, relative to the healthy, non-asthmatic subjects used in most controlled human exposure studies, including the Adams (2002, 2006) studies, a greater proportion of people with asthma may be affected, and those who are affected may have as large or larger lung function and symptomatic responses at ambient exposures to 0.060 ppm O₃. This indicates that the lowest-observed-effects levels demonstrated in controlled human exposure studies that use only healthy subjects may not

⁴⁷ Bell *et al.* (2006) referred to this level as being approximately equivalent to 120 µg/m³, daily 8-hour maximum, the World Health Organization guideline and European Commission target value for O₃.

reflect the lowest levels at which people with asthma or other lung diseases may respond.

Being mindful of the uncertainties and limitations inherent in interpreting the available evidence, the 2007 Staff Paper stated the view that the range of alternative O₃ standards for consideration should take into account information on lowest-observed-effects levels in controlled human exposure studies as well as indications of possible effects thresholds reported in some epidemiological studies and questions of biological plausibility in attributing associations observed down to background levels to O₃ exposures alone. Based on the evidence and these considerations, it concluded that the upper end of the range of consideration should be somewhat below 0.080 ppm, the lowest-observed-effects level for effects such as pulmonary inflammation, increased airway responsiveness and impaired host-defense capabilities in healthy adults while at prolonged moderate exertion. The 2007 Staff Paper also concluded that the lower end of the range of alternative O₃ standards appropriate for consideration should be the lowest-observed-effects level for potentially adverse lung function decrements and respiratory symptoms in some healthy adults, 0.060 ppm.

b. Exposure and Risk-Based Considerations

In addition to the evidence-based considerations informing staff recommendations on alternative levels, as discussed above in section II.B, the 2007 Staff Paper also evaluated quantitative exposures and health risks estimated to occur upon meeting the then current 0.084 ppm standard and alternative standards.⁴⁸ In so doing, it presented the important uncertainties and limitations associated with these exposure and risk assessments (discussed above in section II.B and more fully in chapters 4 and 5 of the 2007 Staff Paper).

The 2007 Staff Paper (and the CASAC) also recognized that the exposure and risk analyses could not provide a full picture of the O₃ exposures and O₃-related health risks

posed nationally. The EPA did not have sufficient information to evaluate all relevant at-risk groups (e.g., outdoor workers) or all O₃-related health outcomes (e.g., increased medication use, school absences, and emergency department visits that are part of the broader pyramid of effects discussed above in section II.A.4.d), and the scope of the 2007 Staff Paper analyses was generally limited to estimating exposures and risks in 12 urban areas across the U.S., and to only five or just one area for some health effects included in the risk assessment. Thus, national-scale public health impacts of ambient O₃ exposures are clearly much larger than the quantitative estimates of O₃-related incidences of adverse health effects and the numbers of children likely to experience exposures of concern associated with meeting the 0.084 ppm standard or alternative standards. On the other hand, inter-individual variability in responsiveness means that only a subset of individuals in each group estimated to experience exposures exceeding a given benchmark exposure of concern level would actually be expected to experience such adverse health effects.

The 2007 Staff Paper focused on alternative standards with the same form as the then current 0.084 ppm O₃ standard (i.e. the 0.074/4, 0.070/4 and 0.064/4 scenarios).⁴⁹ Having concluded in the 2007 Staff Paper that it was appropriate to consider a range of standard levels from somewhat below 0.080 ppm down to as low as 0.060 ppm, the 2007 Staff Paper looked to results of the analyses of exposure and risk for the 0.074/4 scenario to represent the public health impacts of selecting a standard in the upper part of the range, the results of analyses of the 0.070/4 scenario to represent the impacts in the middle part of the range, and the results of the analyses of the 0.064/4 scenario to represent the lower part of the range.

As discussed in section II.B.1 of this notice, the exposure estimates presented in the 2007 Staff Paper are for the number and percent of all children and asthmatic children exposed, and the number of person-days (occurrences) of exposures, with daily 8-hour maximum exposures at or above several benchmark levels while at intermittent moderate or greater exertion. Exposures above selected benchmark levels provide some perspective on the public

health impacts of health effects that cannot currently be evaluated in quantitative risk assessments but that may occur at existing air quality levels, and the extent to which such impacts might be reduced by meeting alternative standard levels. As described in section II.B.1.c above, the 2007 Staff Paper refers to exposures at and above these benchmark levels as “exposures of concern.” The 2007 Staff Paper notes that exposures of concern, and the health outcomes they represent, likely occur across a range of O₃ exposure levels, such that there is no one exposure level that addresses all public health concerns. As noted above in section II.B., EPA also has acknowledged that the concept is more appropriately viewed as a continuum with greater confidence and less uncertainty about the existence of health effects at the upper end and less confidence and greater uncertainty as one considers increasingly lower O₃ exposure levels.

Consistent with advice from CASAC, the 2007 Staff Paper estimates exposures of concern not only at 0.080 ppm O₃, a level at which there are clearly demonstrated effects, but also at 0.070 and 0.060 ppm O₃ levels where there is some evidence that health effects are likely to occur in some individuals. The 2007 Staff Paper recognizes that there will be varying degrees of concern about exposures at each of these levels, based in part on the population groups experiencing them. Given that there is clear evidence of inflammation, increased airway responsiveness, and changes in host defenses in healthy people exposed to 0.080 ppm and reason to infer that such effects will continue at lower exposure levels, but with increasing uncertainty about the extent to which such effects occur at lower O₃ concentrations, the 2007 Staff Paper and discussion below, focus on exposures of concern at or above benchmark levels of 0.070 and 0.060 ppm O₃ for purposes of evaluating alternative standards. The focus on these two benchmark levels reflects the following evidence-based considerations, discussed above in section II.C.1, that raise concerns about adverse health effects likely occurring at levels below 0.080 ppm: (1) That there is limited, but important, new evidence from controlled human exposure studies showing lung function decrements and respiratory symptoms in some healthy subjects at 0.060 ppm; (2) that asthmatics are likely to have more serious responses than healthy individuals; (3) that lung function is not likely to be as sensitive a marker for O₃

⁴⁸ As described in the 2007 Staff Paper (section 4.5.8) and discussed above in section II.B, recent O₃ air quality distributions have been statistically adjusted to simulate just meeting the then current 0.084 ppm standard and selected alternative standards. These simulations do not represent predictions of when, whether, or how areas might meet the specified standards. Modeling that projects whether and how areas might attain alternative standards in a future year is presented in the Regulatory Impact Analysis being prepared in connection with this rulemaking.

⁴⁹ The abbreviated notation used to identify the then current 0.084 ppm standard and alternative standards in this section and in the risk assessment section of the Staff Paper is in terms of ppm and the nth highest daily maximum 8-hour average. For example, the 8-hour standard established in 1997 is identified as “0.084/4.”

effects as lung inflammation; and (4) that there is epidemiological evidence which reports associations with O₃ levels that extend well below 0.080 ppm.

Table 3 below summarizes the exposure estimates for all children and asthmatic children for the 0.060 and 0.070 ppm health effect benchmark levels associated with O₃ levels adjusted to just meet 0.074/4, 0.070/4, and 0.064/4 alternative 8-hour standards based on a generally poorer year of air quality (2002) and based on a generally better year of air quality (2004). This table includes exposure estimates reflecting the aggregate estimate for the 12 urban areas as well as the range across these same 12 areas. As shown in Table 3 below, the percent of population exposed over the selected benchmark levels is very similar for all and asthmatic school age children. Thus, the following discussion focuses primarily on the exposure estimates for asthmatic children, recognizing that the pattern of exposure estimates is similar for all children when expressed in terms of percentage of the population.

As noted in section II.B.2 and shown in Tables 1 and 3 of this notice, substantial year-to-year variability is observed, ranging to over an order of magnitude at the higher alternative standard levels, in estimates of the number of children and the number of occurrences of exposures of concern at both the 0.060 and 0.070 ppm benchmark levels. As shown in Table 3, and discussed more fully below, aggregate estimates of exposures of concern for the 12 urban areas included in the assessment are considerably larger for the benchmark level of ≥ 0.060 ppm O₃, compared to the 0.070 ppm benchmark, while the pattern of year-to-year variability is fairly similar.

As shown in Table 3, aggregate estimates of exposures of concern for a 0.060 ppm benchmark level vary considerably among the three alternative standards included in this table, particularly for the 2002 simulations (a year with generally poorer air quality in most, but not all areas). For air quality just meeting a 0.074/4 standard approximately 27% of asthmatic children, based on the 2002 simulation, and approximately 2% of asthmatic children based on the 2004 simulation (a year with better air quality in most but not all areas), are estimated to experience one or more exposures of concern at the benchmark level of ≥ 0.060 ppm O₃. Considering a 0.070/4 standard using the same benchmark level (0.060 ppm), about 18% of asthmatic children are estimated to experience one or more exposures of

concern, in a year with poorer air quality (2002), and only about 1% in a year with better air quality (2004). For the most stringent standard examined (a 0.064/4 standard), about 6% of asthmatic children are estimated to experience one or more exposures of concern in the simulation based on the year with poorer air quality (2002), and exposures of concern at the 0.060 ppm benchmark level are essentially eliminated based on a year with better air quality (2004).

Table 3 also provides aggregate exposure estimates for the 12 urban areas where a benchmark level of ≥ 0.070 ppm is used. Based on the year with poorer air quality (2002), the estimate of the percent of asthmatic children exposed one or more times is about 5% when a 0.074/4 standard is just met; based on a year with better air quality (2004), exposures of concern are essentially eliminated. For this same benchmark (0.070 ppm), when a 0.070/4 standard is just met, estimates range from about 2% of asthmatic children exposed one or more times over this benchmark based on a year with poorer air quality (2002), and exposures of concern are essentially eliminated based on a year with better air quality (2004). At the 0.070 ppm benchmark, just meeting a 0.064/4 standard essentially eliminates exposures of concern regardless of the year that is used as the basis for the analysis.

The 2007 Staff Paper also notes that there is substantial city-to-city variability in these estimates, and notes that it is appropriate to consider not just the aggregate estimates across all cities, but also to consider the public health impacts in cities that receive relatively less protection from the alternative standards. As shown in Table 3, in considering the benchmark level of ≥ 0.060 ppm, while the aggregate percentage of asthmatic children estimated to experience one or more exposures of concern across all 12 cities for a 0.074/4 standard is about 27% based on the year with poorer air quality (2002), it ranges up to approximately 51% for asthmatic children in the city with the least degree of protection from that alternative standard. Similarly, for air quality just meeting a 0.070/4 standard, the aggregate percentage of asthmatic children estimated to experience one or more exposures of concern across all 12 cities is 18% based on the year with poorer air quality, but it ranges up to about 41% in the city with the least degree of protection associated with just meeting that alternative standard. For just meeting a 0.064/4 standard, the aggregate estimate of asthmatic children experiencing

exposures of concern for the 0.060 ppm benchmark is about 6% based on the year with poorer air quality and ranges up to 16% in the city with the least degree of protection.

This pattern of city-to-city variability also occurs at the benchmark level of ≥ 0.070 ppm associated with air quality just meeting these same three alternative standards (*i.e.*, 0.074/4, 0.070/4, and 0.064/4). While the aggregate percentage of asthmatic children estimated to experience such exposures of concern across all 12 cities is about 5% based on the year with poorer air quality for just meeting the 0.074/4 standard, it ranges up to 14% in the city with the least degree of protection associated with that alternative standard. For just meeting a 0.070/4 standard the aggregate estimate is 2% of asthmatic children experiencing exposures of concern for the 0.070 ppm benchmark based on the year with poorer air quality and ranges up to 6% in the city with the least degree of protection. The aggregate estimate for exposures of concern is further reduced to 0.2% of asthmatic children for this same benchmark level for air quality just meeting a 0.064/4 standard based on the year with poorer air quality and ranges up to 1% in the city with the least degree of protection.

In addition to observing the fraction of the population estimated to experience exposures of concern associated with just meeting alternative standards, EPA also took into consideration in the 2007 Staff Paper the percent reduction in exposures of concern and health risks associated with alternative standards relative to just meeting the then current 0.084/4 standards. For the current decision it is also informative to consider the incremental reductions in exposures of concern associated with more stringent alternative standards relative to the 0.075 ppm standard. As shown in Table 1 above, at the ≥ 0.060 ppm benchmark level based on a year with poorer air quality, the reduction in exposures of concern for asthmatic children in going from the 0.074/4 standard (which approximates the 0.075 ppm standard adopted in 2008) down to a 0.064/4 standard is observed to be very similar to the reduction estimated to occur in going from then current 0.084/4 standard down to a 0.074/4 standard. More specifically, the estimates for asthmatic children are reduced from 47% (about 1.2 million children) associated with meeting a 0.084/4 standard down to 27% (about 700,000 children) for just meeting a 0.074/4 standard and the estimates are reduced further to about 6% (about 150,000 children) associated with just meeting a

0.064/4 standard in the 12 urban areas included in the assessment. In a year with better air quality (2004), exposures estimated to exceed the 0.060 ppm benchmark in asthmatic children one or more times in a year are reduced from 11% associated with just meeting a 0.084/4 standard down to about 2% for a 0.074/4 standard and are essentially eliminated when a 0.064/4 standard is just met.

Turning to consideration of the risk assessment estimates, Table 2 above summarizes the risk estimates for moderate lung function decrements in both all school age children and asthmatic school age children associated with just meeting several alternative standards based on simulations involving a year with relatively poorer air quality (2002) and a year with relatively better air quality (2004). As shown in Table 2, for the 2002 simulation the reduction in the number of asthmatic children estimated to experience one or more moderate lung function decrements going from a 0.074/4 standard down to a 0.064/4 standard is roughly equivalent to the additional health protection afforded associated with just meeting a 0.074/4 standard relative to then current 0.084/4 standard. More specifically, for just 5 urban areas, it is estimated that nearly 8% of asthmatic children (130,000 children) would experience one or more occurrences of moderate lung function decrements per year at a 0.084/4 standard and this would be reduced to about 5% (90,000 children) at a 0.074/4 standard and further reduced down to about 3% (50,000 children) at a 0.064/4 standard. Based on the 2002 simulations, the percent reduction associated with just meeting a 0.064/4 standard relative to then current 0.084/4 standard is about 62% which is about twice the reduction in risk compared to the estimated 31% reduction associated

with just meeting a 0.074/4 standard. As shown in Table 2 above, similar patterns were observed in reductions in lung function risk for all school age children in 12 urban areas associated with these alternative standards.

Figures 6–5 and 6–6 in the 2007 Staff Paper (EPA, 2007b) show the percent reduction in non-accidental mortality risk estimates associated with just meeting the same alternative standards discussed above relative to just meeting the then current 0.084/4 standard for 12 urban areas, based on adjusting 2002 and 2004 air quality data. These figures also provide perspective on the extent to which the risks in these years (*i.e.*, 2002 and 2004) are greater than those estimated to occur upon meeting the then current 0.084/4 standard (in terms of a negative percent reduction relative to a 0.084/4 standard). Based on the 2002 simulations (EPA, 2007b, Figure 6–5), the estimated reduction in non-accidental mortality is about 30 to 70% across the 12 urban areas for just meeting a 0.064/4 standard relative to the then current 0.084/4 standard. This reduction is roughly twice the 15 to 30% estimated reduction across the 12 urban areas associated with just meeting a 0.074/4 standard relative to a 0.084/4 standard. While the estimated incidence is lower based on the 2004 simulations (EPA, 2007b, Figure 6–6), the pattern of risk reductions among alternative standards is roughly similar to that observed for the 2002 simulations.

In addition to the risk estimates for lung function decrements in all school age children and non-accidental mortality that were estimated for 12 urban areas and lung function decrements in asthmatic children for 5 urban areas, a similar pattern of incremental reductions in health risks was shown for two health outcomes where risks were estimated in one city only for each of these outcomes. These

included reductions in respiratory symptoms in asthmatic children (EPA, 2007b; Boston, Table 6–9) and respiratory-related hospital admissions (EPA, 2007a; New York City, Table 6–10) associated with just meeting alternative 8-hour standards set at 0.074 ppm, 0.070 ppm, and 0.064 ppm relative to just meeting the then current 0.084 ppm standard. Using the 2002 simulation, a standard set at 0.074/4 is estimated to reduce the incidence of symptom days in children with moderate to severe asthma in the Boston area by about 15 percent relative to a 0.084/4 standard. With this reduction, it is estimated that about 1 respiratory symptom day in 8 during the O₃ season would be attributable to O₃ exposure. A standard set at 0.064/4 is estimated, based on the 2002 simulation, to reduce the incidence of symptom days in children with moderate to severe asthma in the Boston area by about a 25 to 30 percent reduction relative to a 0.084 ppm standard, which is roughly twice the reduction compared to that provided by a 0.074/4 standard. But even with this reduction, it is estimated that 1 respiratory symptom day in 10 during the O₃ season is attributable to O₃ exposure.

As shown in Table 6–10 (EPA, 2007b) estimated incidence of respiratory-related hospital admissions in one urban area (New York City) was reduced by 14 to 17 percent by a standard set at 0.074/4 relative to then current 0.084/4 standard, in the year with relatively high and relatively low O₃ air quality levels, respectively. Similar to the pattern observed for the other health outcomes discussed above, the reduction in incidence of respiratory-related hospital admissions for a 0.064/4 standard relative to a 0.084/4 standard is about twice that associated with a 0.074/4 standard relative to a 0.084/4 standard.

TABLE 3—NUMBER AND PERCENT OF ALL AND ASTHMATIC SCHOOL AGE CHILDREN IN 12 URBAN AREAS ESTIMATED TO EXPERIENCE 8-HOUR OZONE EXPOSURES ABOVE 0.060 AND 0.070 PPM WHILE AT MODERATE OR GREATER EXERTION, ONE OR MORE TIMES PER SEASON ASSOCIATED WITH JUST MEETING ALTERNATIVE 8-HOUR STANDARDS BASED ON ADJUSTING 2002 AND 2004 AIR QUALITY DATA^{1 2}

Benchmark levels of exposures of concern (ppm)	8-Hour air quality standards ³ (ppm)	All children, ages 5–18 Aggregate for 12 urban areas Number of children exposed (% of all children) [Range across 12 cities, % of all children]		Asthmatic children, ages 5–18 Aggregate for 12 urban areas Number of children exposed (% of group) [Range across 12 cities, % of group]	
		2002	2004	2002	2004
		0.070	0.074	770,000 (4%) [0–13%]	20,000 (0%) [0–1%]
	0.070	270,000 (1%) [0–5%]	0 (0%) [0%]	50,000 (2%) [0–6%]	0 (0%) [0%]
	0.064	30,000 (0.2%) [0–1%]	0 (0%) [0%]	10,000 (0.2%) [0–1%]	0 (0%) [0%]

TABLE 3—NUMBER AND PERCENT OF ALL AND ASTHMATIC SCHOOL AGE CHILDREN IN 12 URBAN AREAS ESTIMATED TO EXPERIENCE 8-HOUR OZONE EXPOSURES ABOVE 0.060 AND 0.070 PPM WHILE AT MODERATE OR GREATER EXERTION, ONE OR MORE TIMES PER SEASON ASSOCIATED WITH JUST MEETING ALTERNATIVE 8-HOUR STANDARDS BASED ON ADJUSTING 2002 AND 2004 AIR QUALITY DATA^{1 2}—Continued

Benchmark levels of exposures of concern (ppm)	8-Hour air quality standards ³ (ppm)	All children, ages 5–18 Aggregate for 12 urban areas Number of children exposed (% of all children) [Range across 12 cities, % of all children]		Asthmatic children, ages 5–18 Aggregate for 12 urban areas Number of children exposed (% of group) [Range across 12 cities, % of group]	
		2002	2004	2002	2004
		0.060	0.074	4,550,000 (25%) [1–48%]	350,000 (2%) [0–9%]
	0.070	3,000,000 (16%) [1–36%]	110,000 (1%) [0–4%]	460,000 (18%) [0–41%]	10,000 (1%) [0–3%]
	0.064	950,000 (5%) [0–17%]	10,000 (0%) [0–1%]	150,000 (6%) [0–16%]	0 (0%) [0–1%]

¹ Moderate or greater exertion is defined as having an 8-hour average equivalent ventilation rate ≥ 13 l-min/m².

² Estimates are the aggregate results based on 12 combined statistical areas (Atlanta, Boston, Chicago, Cleveland, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC). Estimates are for the ozone season which is all year in Houston, Los Angeles and Sacramento and March or April to September or October for the remaining urban areas.

³ All standards summarized here have the same form as the 8-hour standard established in 1997 which is specified as the 3-year average of the annual 4th highest daily maximum 8-hour average concentrations must be at or below the concentration level specified. As described in the 2007 Staff Paper (EPA, 2007b, section 4.5.8), recent O₃ air quality distributions have been statistically adjusted to simulate just meeting the 0.084 ppm standard and selected alternative standards. These simulations do not represent predictions of when, whether, or how areas might meet the specified standards.

2. CASAC Views Prior to 2008 Decision

In comments on the second draft Staff Paper, CASAC stated in its letter to the Administrator, “the CASAC unanimously recommends that the current primary ozone NAAQS be revised and that the level that should be considered for the revised standard be from 0.060 to 0.070 ppm” (Henderson, 2006c, p. 5). This recommendation followed from its more general recommendation that the 0.084 ppm standard needed to be substantially reduced to be protective of human health, particularly in at-risk subpopulations.

The CASAC Panel noted that beneficial reductions in some adverse health effects were estimated to occur upon meeting the lowest standard level (0.064 ppm) considered in the risk assessment (Henderson, 2006c, p. 4). The lower end of this range reflects CASAC’s views that “[w]hile data exist that adverse health effects may occur at levels lower than 0.060 ppm, these data are less certain and achievable gains in protecting human health can be accomplished through lowering the ozone NAAQS to a level between 0.060 and 0.070 ppm.” (id.).

In a subsequent letter sent specifically to offer advice to aid the Administrator and Agency staff in developing the O₃ proposal, the CASAC reiterated that the Panel members “were unanimous in recommending that the level of the current primary ozone standard should be lowered from 0.08 ppm to no greater than 0.070 ppm” (Henderson, 2007, p. 2). Further, the CASAC Panel expressed

the view that the 2006 Criteria Document and 2007 Staff Paper, together with the information in its earlier letter, provide “overwhelming scientific evidence for this recommendation,” and emphasized the Clean Air Act requirement that the primary standard must be set to protect the public health with an adequate margin of safety (id.).

3. Basis for 2008 Decision on the Primary Standard

This section presents the rationale for the 2008 final decision on the primary O₃ standard as presented in the 2008 final rule (73 FR 16475). The EPA’s conclusions on the level of the standard began by noting that, having carefully considered the public comments on the appropriate level of the O₃ standard, EPA concluded that the fundamental scientific conclusions on the effects of O₃ reached in the 2006 Criteria Document and 2007 Staff Paper remained valid. In considering the level at which the primary O₃ standard should be set, EPA placed primary consideration on the body of scientific evidence available in the 2008 final rulemaking on the health effects associated with O₃ exposure, while viewing the results of exposure and risk assessments as providing information in support of the decision. In considering the available scientific evidence, EPA concluded that a focus on the proposed range of 0.070 to 0.075 ppm was appropriate in light of the large body of controlled human exposure and epidemiological and other scientific

evidence. The notice stated that this body of evidence did not support retaining the then current 0.084 ppm 8-hour O₃ standard, as suggested by some commenters, nor did it support setting a level just below 0.080 ppm, because, based on the entire body of evidence, such a level would not provide a significant increase in protection compared to the 0.084 ppm standard. Further, such a level would not be appreciably below the level in controlled human exposure studies at which adverse effects have been demonstrated (i.e., 0.080 ppm). The notice also stated that the body of evidence did not support setting a level of 0.060 ppm or below, as suggested by other commenters. In evaluating the information from the exposure assessment and the risk assessment, EPA judged that this information did not provide a clear enough basis for choosing a specific level within the range of 0.075 to 0.070 ppm.

In making a final judgment about the level of the primary O₃ standard, EPA noted that the level of 0.075 ppm is above the range recommended by the CASAC (i.e., 0.070 to 0.060 ppm). The notice stated that in placing great weight on the views of CASAC, careful consideration had been given to CASAC’s stated views and the scientific basis and policy views for the range it recommended. In so doing, EPA fully agreed that the scientific evidence supports the conclusion that the current standard was not adequate and must be revised.

With respect to CASAC's recommended range of standard levels, EPA observed that the basis for CASAC's recommendation appeared to be a mixture of scientific and policy considerations. While in general agreement with CASAC's views concerning the interpretation of the scientific evidence, EPA noted that there was no bright line clearly directing the choice of level, and the choice of what was appropriate was clearly a public health policy judgment entrusted to the EPA Administrator. This judgment must include consideration of the strengths and limitations of the evidence and the appropriate inferences to be drawn from the evidence and the exposure and risk assessments. In reviewing the basis for the CASAC Panel's recommendation for the range of the O₃ standard, EPA observed that it reached a different policy judgment than the CASAC Panel based on apparently placing different weight in two areas: The role of the evidence from the Adams studies and the relative weight placed on the results from the exposure and risk assessments. While EPA found the evidence reporting effects at the 0.060 ppm level from the Adams studies to be too limited to support a primary focus at this level, EPA observed that the CASAC Panel appeared to place greater weight on this evidence, as indicated by its recommendation of a range down to 0.060 ppm. It was noted that while the CASAC Panel supported a level of 0.060 ppm, they also supported a level above 0.060, which indicated that they did not believe that the results of Adams studies meant that the level of the standard had to be set at 0.060 ppm. The EPA also observed that the CASAC Panel appeared to place greater weight on the results of the risk assessment as a basis for its recommended range. In referring to the risk assessment results for lung function, respiratory symptoms, hospital admissions and mortality, the CASAC Panel concluded that: "beneficial effects in terms of reduction of adverse health effects were calculated to occur at the lowest concentration considered (*i.e.*, 0.064 ppm)" (Henderson, 2006c, p. 4). However, EPA more heavily weighed the implications of the uncertainties associated with the Agency's quantitative human exposure and health risk assessments. Given these uncertainties, EPA did not agree that these assessment results appropriately served as a primary basis for concluding that levels at or below 0.070 ppm were required for the 8-hour O₃ standard.

The notice stated that after carefully taking the above comments and

considerations into account, and fully considering the scientific and policy views of the CASAC, EPA decided to revise the level of the primary 8-hour O₃ standard to 0.075 ppm. The EPA judged, based on the available evidence, that a standard set at this level would be requisite to protect public health with an adequate margin of safety, including the health of sensitive subpopulations, from serious health effects including respiratory morbidity, that were judged to be causally associated with short-term and prolonged exposures to O₃, and premature mortality. The EPA also judged that a standard set at this level provides a significant increase in protection compared to the 0.084 ppm standard, and is appreciably below 0.080 ppm, the level in controlled human exposure studies at which adverse effects have been demonstrated. At a level of 0.075 ppm, exposures at and above the benchmark of 0.080 ppm are essentially eliminated, and exposures at and above the benchmark of 0.070 are substantially reduced or eliminated for the vast majority of people in at-risk groups. A standard set at a level lower than 0.075 would only result in significant further public health protection if, in fact, there is a continuum of health risks in areas with 8-hour average O₃ concentrations that are well below the concentrations observed in the key controlled human exposure studies and if the reported associations observed in epidemiological studies are, in fact, causally related to O₃ at those lower levels. Based on the available evidence, EPA was not prepared to make these assumptions. Taking into account the uncertainties that remained in interpreting the evidence from available controlled human exposure and epidemiological studies at very low levels, EPA noted that the likelihood of obtaining benefits to public health decreased with a standard set below 0.075 ppm O₃, while the likelihood of requiring reductions in ambient concentrations that go beyond those that are needed to protect public health increased. The EPA judged that the appropriate balance to be drawn, based on the entire body of evidence and information available in the 2008 final rulemaking, was to set the 8-hour primary standard at 0.075 ppm. The EPA expressed the belief that a standard set at 0.075 ppm would be sufficient to protect public health with an adequate margin of safety, and did not believe that a lower standard was needed to provide this degree of protection. The EPA further asserted that this judgment appropriately considered the

requirement for a standard that was neither more nor less stringent than necessary for this purpose and recognized that the CAA does not require that primary standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

4. CASAC Advice Following 2008 Decision

Following the 2008 decision on the O₃ standard, serious questions were raised as to whether the standard met the requirements of the CAA. In April 2008, the members of the CASAC Ozone Review Panel sent a letter to EPA stating "In our most-recent letters to you on this subject—dated October 2006 and March 2007—the CASAC unanimously recommended selection of an 8-hour average Ozone NAAQS within the range of 0.060 to 0.070 parts per million for the primary (human health-based) Ozone NAAQS" (Henderson, 2008). The letter continued: "The CASAC now wishes to convey, by means of this letter, its additional, unsolicited advice with regard to the primary and secondary Ozone NAAQS. In 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" (*id.*). Moreover, the CASAC Panel noted that "numerous medical organizations and public health groups have also expressed their support of these CASAC recommendations." (*id.*) The letter further stated the following strong, unanimous view:

[the CASAC did] "not endorse the new primary ozone standard as being sufficient 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. 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" (Henderson, 2008).

5. Administrator's Proposed Conclusions

For the reasons discussed below, the Administrator proposes to set a new level for the 8-hour primary O₃ within

the range from 0.060 to 0.070 ppm.⁵⁰ In reaching this proposed decision, the Administrator has considered: the evidence-based considerations from the 2006 Criteria Document and the 2007 Staff Paper; the results of the exposure and risk assessments discussed above and in the 2007 Staff Paper; CASAC advice and recommendations provided in CASAC's letters to the Administrator both during and following the 2008 rulemaking; EPA staff recommendations; and public comments received in conjunction with review of drafts of these documents and on the 2007 proposed rule. In considering what level of an 8-hour O₃ standard is requisite to protect public health with an adequate margin of safety, the Administrator is mindful that this choice requires judgments based on an interpretation of the evidence and other information that neither overstates nor understates the strength and limitations of the evidence and information.

The Administrator notes that the most certain evidence of adverse health effects from exposure to O₃ comes from the controlled human exposure studies, and that the large bulk of this evidence derives from studies of exposures at levels of 0.080 ppm and above. At those levels, there is consistent evidence of lung function decrements and respiratory symptoms in healthy young adults, as well as evidence of O₃-induced pulmonary inflammation, airway responsiveness, impaired host defense capabilities, and other medically significant airway responses. Moreover, there is no evidence that the 0.080 ppm exposure level is a threshold for any of these types of respiratory effects. Rather, there is now controlled human exposure evidence, including studies of lung function decrements and respiratory symptoms at the 0.060 ppm exposure level, that strengthens our previous understanding that this array of respiratory responses are likely to occur in some healthy adults at such lower levels.

In particular, the Administrator notes two studies by Adams (2002, 2006), newly available in the 2008 rulemaking, that examined lung function and respiratory symptom effects associated with prolonged O₃ exposures at levels below 0.080 ppm, as well as EPA's

reanalysis of the data from the Adams (2006) study at a 0.060 ppm exposure level. As discussed above, while the author's analysis focused on hour-by-hour comparisons of effects, for the purpose of exploring responses associated with different patterns of exposure, EPA's reanalysis focused on addressing the more fundamental question of whether the pre- to post-exposure change in lung function differed between a 6.6-hour exposure to 0.060 ppm O₃ versus a 6.6 hour exposure to clean filtered air. The Administrator notes that this reanalysis found small, but statistically significant group mean differences in lung function decrements in healthy adults at the 0.060 ppm exposure level, which is now the lowest-observed-effects level for these effects. Moreover, these studies also report a small percentage of subjects (7 to 20 percent) experienced moderate lung function decrements (≥ 10 percent) at the 0.060 ppm exposure level. While for active healthy people, moderate levels of functional responses (e.g., FEV₁ decrements of $\geq 10\%$ but $< 20\%$) and/or moderate respiratory symptom responses would likely interfere with normal activity for relatively few responsive individuals, the Administrator notes that for people with lung disease, even moderate functional or symptomatic responses would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication. Further, she notes that CASAC indicated that a focus on the lower end of the range of moderate levels of functional responses (e.g., FEV₁ decrements $\geq 10\%$) is most appropriate for estimating potentially adverse lung function decrements in people with lung disease (Henderson, 2006c).

The Administrator also notes that many public commenters on the 2007 proposed rule raised a number of questions about the weight that should be placed on the Adams studies and EPA's reanalysis of data from the Adams (2006) study. Some commenters expressed the view that the results of these studies and EPA's reanalysis provided support for setting a standard level below the proposed range, while others raised questions about EPA's reanalysis and generally expressed the view that the study results were not robust enough to reach conclusions about respiratory effects at the 0.060 ppm exposure level.⁵¹

Based on all the above considerations, the Administrator concludes that the Adams studies provide limited but

important evidence which adds to the overall body of evidence that informs her proposed decision on the range of levels within which a standard could be set that would be requisite to protect public health with an adequate margin of safety, including the health of at-risk populations such as people with lung disease.

In considering controlled human exposure studies reporting O₃-induced pulmonary inflammation, airway responsiveness, and impaired host defense capabilities at exposure levels down to 0.080 ppm, the lowest level at which these effects have been tested, the Administrator notes that these physiological effects have been linked to aggravation of asthma and increased susceptibility to respiratory infection, potentially leading to increased medication use, increased school and work absences, increased visits to doctors' offices and emergency departments, and increased hospital admissions, especially in people with lung disease. These physiological effects are all indicators of potential adverse O₃-related morbidity effects, which are consistent with and lend plausibility to the associations observed between O₃ and adverse morbidity effects and mortality effects in epidemiological studies.

With regard to epidemiological studies, the Administrator observes that statistically significant associations between ambient O₃ levels and a wide array of respiratory symptoms and other morbidity outcomes including school absences, emergency department visits, and hospital admissions have been reported in a large number of studies. More specifically, positive and robust associations were found between ambient O₃ concentrations and respiratory hospital admissions and emergency department visits, when focusing particularly on the results of warm season analyses. Taken together, the overall body of evidence from controlled human exposure, toxicological, and epidemiological studies supports the inference of a causal relationship between acute ambient O₃ exposures and increased respiratory morbidity outcomes resulting in increased emergency department visits and hospitalizations during the warm season. Further, the Administrator notes that recent epidemiological evidence is highly suggestive that O₃ directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality.

The Administrator also considered the epidemiological evidence with regard to considering potential effects thresholds at the population level for

⁵⁰ As discussed above at the beginning of section II, the Administrator has focused her reconsideration of the primary O₃ standard set in the 2008 final rule on the level of the standard, having decided not to reopen the 2008 final rule with regard to the need to revise the 1997 primary O₃ standard to provide increased public health protection nor with regard to the indicator, averaging period, and form of the 2008 standard.

⁵¹ The EPA responded to these comments in the 2008 final rule (73 FR 16454-5).

morbidity and mortality effects. As discussed above, while some studies provide some indication of possible 8-hour average threshold levels from below about 0.025 to 0.035 ppm (within the range of background concentrations) up to approximately 0.050 ppm, other studies observe linear concentration-response functions suggesting that there may be no effects thresholds at the population level above background concentrations. In addition, other studies conducted subset analyses that included only days with ambient O₃ concentrations below the level of the then current standard, or below even lower O₃ concentrations, including a level as low as 0.061 ppm, and continue to report statistically significant associations. The Administrator notes that the relationships between ambient O₃ concentrations and lung function decrements, respiratory symptoms, indicators of respiratory morbidity including increased respiratory-related emergency department visits and hospital admissions, and possibly mortality reported in a large number of studies likely extend down to ambient O₃ concentrations well below the level of the standard set in 2008 (0.075 ppm), in that the highest level at which there is any indication of a threshold is approximately 0.050 ppm. The Administrator notes as well that toward the lower end of the range of O₃ concentrations observed in such studies, ranging down to background levels (*i.e.*, 0.035 to 0.015 ppm), there is increasing uncertainty as to whether the observed associations remain plausibly related to exposures to ambient O₃, rather than to the broader mix of air pollutants present in the ambient atmosphere. She also notes that there are limitations in epidemiological studies that make discerning population thresholds difficult, as discussed above, such that there is the possibility that thresholds for individuals may exist in reported associations at fairly low levels within the range of air quality observed in the studies but not be detectable as population thresholds in epidemiological analyses.

In looking more broadly at evidence from animal toxicological, controlled human exposure, and epidemiological studies, the Administrator finds substantial evidence, newly available for consideration in the 2008 rulemaking, that people with asthma and other preexisting pulmonary diseases are among those at increased risk from O₃ exposure. As discussed above, altered physiological, morphological, and biochemical states typical of respiratory diseases like

asthma, COPD, and chronic bronchitis may render people sensitive to additional oxidative burden induced by O₃ exposure. Children and adults with asthma are the group that has been studied most extensively. Evidence from controlled human exposure studies indicates that asthmatics and people with allergic rhinitis may exhibit larger lung function decrements in response to O₃ exposure than healthy subjects and that they can have larger inflammatory responses. The Administrator also notes that two large U.S. epidemiological studies, as well as several smaller U.S. and international studies, have reported fairly robust associations between ambient O₃ concentrations and measures of lung function and daily symptoms (*e.g.*, chest tightness, wheeze, shortness of breath) in children with moderate to severe asthma and between O₃ and increased asthma medication use. These more serious responses in asthmatics and others with lung disease provide biological plausibility for the respiratory morbidity effects observed in epidemiological studies, such as respiratory-related emergency department visits and hospital admissions.

The Administrator also observes that a substantial body of evidence from controlled human exposure and epidemiological studies indicates that relative to the healthy, non-asthmatic subjects used in most controlled human exposure studies, a greater proportion of people with asthma may be affected, and those who are affected may have as large or larger lung function and symptomatic responses to O₃ exposures. Thus, the Administrator concludes that controlled human exposure studies of lung function decrements and respiratory symptoms that evaluate only healthy, non-asthmatic subjects likely underestimate the effects of O₃ exposure on asthmatics and other susceptible populations.

In addition to the evidence-based considerations discussed above, the Administrator also considered quantitative exposures and health risks estimated to occur associated with air quality simulated to just meet various standard levels to help inform judgments about a range of standard levels for consideration that could provide an appropriate degree of public health protection. In so doing, she is mindful of the important uncertainties and limitations that are associated with the exposure and risk assessments, as discussed in more detail in the 2007 Staff Paper, and above in sections II.B and II.C.1.b. Beyond these uncertainties, the Administrator also recognized important limitations related to the

exposure and risk analyses. For example, EPA did not have sufficient information to evaluate all relevant at-risk groups (*e.g.*, outdoor workers) or all O₃-related health outcomes (*e.g.*, increased medication use, school absences, emergency department visits), and the scope of the analyses was generally limited to estimating exposures and risks in 12 urban areas across the U.S., and to only five or just one area for some health effects. Thus, it is clear that national-scale public health impacts of ambient O₃ exposures are much larger than the quantitative estimates of O₃-related incidences of adverse health effects and the numbers of children likely to experience exposures of concern associated with meeting the then current standard or alternative standards. Taking these limitations into account, the CASAC advised EPA not to rely solely on the results of the exposure and risk assessments in considering alternative standards, but also to place significant weight on the body of evidence of O₃-related health effects in drawing conclusions about an appropriate range of levels for consideration. The Administrator agrees with this advice.

Turning first to the results of the exposure assessment, the Administrator focused on the extent to which alternative standard levels, approximately at and below the 0.075 ppm O₃ standard set in the 2008 final rule, are estimated to reduce exposures over the 0.060 and 0.070 ppm health effects benchmark levels, for all and asthmatic school age children in the 12 urban areas included in the assessment.⁵² The Administrator also took note that the lowest standard level included in the exposure and health risk assessments was 0.064 ppm and that additional reductions in exposures over the selected health benchmark levels would be anticipated for just meeting a 0.060 ppm standard.

As an initial matter, the Administrator recognized that the concept of "exposures of concern" is more appropriately viewed as a continuum, with greater confidence and less uncertainty about the existence of health effects at the upper end and less confidence and greater uncertainty as one considers increasingly lower O₃ exposure levels. In considering the concept of exposures of concern, the Administrator also noted that it is important to balance concerns about the potential for health effects and their

⁵² As noted in section II.C.1.b. above, the Administrator focused on alternative standards with different levels but the same form and averaging time as the primary standard set in 2008.

severity with the increasing uncertainty associated with our understanding of the likelihood of such effects at lower O₃ levels. Within the context of this continuum, estimates of exposures of concern at discrete benchmark levels provide some perspective on the public health impacts of O₃-related physiological effects that have been demonstrated in controlled human exposure and toxicological studies but cannot be evaluated in quantitative risk assessments, such as lung inflammation, increased airway responsiveness, and changes in host defenses. They also help in understanding the extent to which such impacts have the potential to be reduced by meeting alternative standards. As discussed in II.C.1.a above, these O₃-related physiological effects are plausibly linked to the increased morbidity seen in epidemiological studies (*e.g.*, as indicated by increased medication use in asthmatics, school absences in all children, and emergency department visits and hospital admissions in people with lung disease).

Estimates of the number of people likely to experience exposures of concern cannot be directly translated into quantitative estimates of the number of people likely to experience specific health effects, since sufficient information to draw such comparisons is not available—if such information were available, these health outcomes would have been included in the quantitative risk assessment. Due to individual variability in responsiveness, only a subset of individuals who have exposures at and above a specific benchmark level are expected to experience such adverse health effects, and susceptible population groups such as those with asthma are expected to be affected more by such exposures than healthy individuals.

For the reasons discussed in section II.C.1.b above, the Administrator has concluded that it is appropriate to focus on both the 0.060 and 0.070 ppm health effect benchmarks for her decision on the primary standard. In summary, the focus on these two benchmark levels reflects the following evidence-based considerations, discussed above in section II.C.1.a, that raise concerns about adverse health effects likely occurring at levels below 0.080 ppm: (1) That there is limited, but important, new evidence from controlled human exposure studies showing lung function decrements and respiratory symptoms in some healthy subjects at 0.060 ppm; (2) that asthmatics are likely to have more serious responses than healthy individuals; (3) that lung function is not likely to be as sensitive a marker for O₃

effects as lung inflammation; and (4) that there is epidemiological evidence which reports associations between ambient O₃ concentrations and respiratory symptoms, ED visits, hospital admissions, and premature mortality in areas with O₃ levels that extend well below 0.080 ppm.

Based on the exposure and risk considerations discussed in detail in the 2007 Staff Paper and presented in sections II.B and II.C.1.b above, the Administrator notes the following important observations from these assessments: (1) There is a similar pattern for all children and asthmatic school age children in terms of exposures of concern over selected benchmark levels when estimates are expressed in terms of percentage of the population; (2) the aggregate estimates of exposures of concern reflecting estimates for the 12 urban areas included in the assessment are considerably larger for the benchmark level of 0.060 ppm compared to the 0.070 ppm benchmark; (3) there is notable year-to-year variability in exposure and risk estimates with higher exposure and risk estimates occurring in simulations involving a year with generally poorer air quality in most areas (2002) compared to a year with generally better air quality (2004); and (4) there is significant city-to-city variability in exposure and risk estimates, with some cities receiving considerably less protection associated with air quality just meeting the same standard. As discussed above, the Administrator believes that it is appropriate to consider not just the aggregate estimates across all cities, but also to consider the public health impacts in cities that receive relatively less protection from alternative standards under consideration. Similarly, the Administrator believes that year-to-year variability should also be considered in making judgments about which standards will protect public health with an adequate margin of safety.

In addition, significant reductions in exposures of concern and risk have been estimated to occur across standard levels analyzed. The magnitudes of exposure and risk reductions estimated to occur in going from a 0.074 ppm standard to a 0.064 ppm standard are as large as those estimated to occur in going from the then current 0.084 ppm standard to a 0.074 ppm standard. Consequently, the reduction in risk that can be achieved by going from a standard of 0.074 ppm to a standard of 0.064 ppm is comparable to the risk reduction that can be achieved by moving from the 1997 O₃ standard,

effectively a 0.084 ppm standard, to a standard very close to the 2008 standard of 0.075 ppm.

The Administrator also observes that estimates of exposures of concern associated with air quality just meeting the alternative standards below 0.080 ppm (*i.e.*, 0.074, 0.070, and 0.064 ppm, the levels included in the assessment) are notably lower than estimates for alternative standards set at and above 0.080 ppm. As shown in Table 6–8 in the 2007 Staff Paper, just meeting a 0.080 ppm standard is associated with an aggregate estimate of exposures of concern of about 13% of asthmatic children at the 0.070 ppm benchmark level, ranging up to 31% in the city with the least degree of protection in a year with generally poorer air quality, and an aggregate estimate of exposures of concern of about 40% of asthmatic children, ranging up to 63% in the city with the least degree of protection at the 0.060 ppm benchmark level. Based on the exposure estimates presented in Table 3 in this notice, she observes that standards included in the assessment below 0.080 ppm (*i.e.*, 0.074, 0.070, and 0.064 ppm), are estimated to have substantially lower estimates of exposures of concern at the 0.070 ppm benchmark level. Similarly, she notes that exposures of concern at the 0.060 ppm benchmark associated with alternative standards below 0.080 ppm are appreciably lower than exposures associated with standards at or above 0.080 ppm, especially for standards set at 0.064 and 0.070 ppm.

As noted previously, the Administrator also recognizes that the risk estimates for health outcomes included in the risk assessment are limited and that the overall health effects evidence is indicative of a much broader array of O₃-related health effects that are part of a “pyramid of effects” that include various indicators of morbidity that could not be included in the risk assessment (*e.g.*, school absences, increased medication use, doctor’s visits, and emergency department visits), some of which have a greater impact on at-risk groups. Consideration of such unquantified risks for this array of health effects, taken together with the estimates of exposures of concern and the quantified health risks discussed above, supports the Administrator’s evidence-based conclusion that revising the standard level to a level well below 0.080 ppm will provide important increased public health protection, especially for at-risk groups such as people with asthma or other lung disease, as well as children and older adults, particularly those active outdoors, and outdoor workers.

Based on the evidence- and exposure/ risk-based considerations discussed above, the Administrator concludes that it is appropriate to set the level of the primary O₃ standard to a level well below 0.080 ppm, a level at which the evidence provides a high degree of certainty about the adverse effects of O₃ exposure in healthy people, to provide an adequate margin of safety for at-risk groups. In selecting a proposed range of levels, the Administrator believes it is appropriate to consider the following information: (1) The strong body of evidence from controlled human exposure studies evaluating healthy people at exposure levels of 0.080 ppm and above that demonstrated lung function decrements, respiratory symptoms, pulmonary inflammation, and other medically significant airway responses, as well as limited but important evidence of lung function decrements and respiratory symptoms in healthy people down to O₃ exposure levels of 0.060 ppm; (2) the substantial body of evidence from controlled human exposure and epidemiological studies indicating that people with asthma are likely to experience larger and more serious effects than healthy people; (3) the body of epidemiological evidence indicating associations are observed for a wide range of serious health effects, including respiratory-related emergency department visits and hospital admissions and premature mortality, across distributions of ambient O₃ concentrations that extend below the current standard level of 0.075 ppm, as well as questions of biological plausibility in attributing the observed effects to O₃ alone at the lower end of the concentration ranges extending down to background levels; and (4) the estimates of exposures of concern and risks for a range of health effects that indicate that important improvements in public health are very likely associated with O₃ levels just meeting alternative standards, especially for standards set at 0.070 and 0.064 ppm (the lowest levels included in the assessment), relative to standards set at and above 0.080 ppm.

The Administrator next considered what standard level well below 0.080 ppm would be requisite to protect public health, including the health of at-risk groups, with an adequate margin of safety that is sufficient but not more than necessary to achieve that result. The assessment of a standard level calls for consideration of both the degree of risk to public health at alternative levels of the standard as well as the certainty that such risk will occur at any specific level. Based on the information

available in the 2008 rulemaking, there is no evidence-based bright line that indicates a single appropriate level. Instead there is a combination of scientific evidence and other information that needs to be considered as a whole in making this public health policy judgment, and selecting a standard level from a range of potentially reasonable values.

As an initial matter, the Administrator considered whether the standard level of 0.075 ppm set in the 2008 final rule is sufficiently below 0.080 ppm to be requisite to protect public health with an adequate margin of safety. In considering this standard level, the Administrator looked to the rationale for selecting this level presented in the 2008 final rule, as summarized above in section II.C.3. In that rationale, EPA observed that a level of 0.075 ppm is above the range of 0.060 to 0.070 ppm recommended by CASAC, and that the CASAC Panel appeared to place greater weight on the evidence from the Adams studies and on the results of the exposure and risk assessments, whereas EPA placed greater weight on the limitations and uncertainties associated with that evidence and the quantitative exposure and risk assessments. Additionally, EPA's rationale did not discuss and thus placed no weight on exposures of concern relative to the 0.060 ppm benchmark. Further, EPA concluded that "[a] standard set at a lower level than 0.075 ppm would only result in significant further public health protection if, in fact, there is a continuum of health risks in areas with 8-hour average O₃ concentrations that are well below the concentrations observed in the key controlled human exposure studies and if the reported associations observed in epidemiological studies are, in fact, causally related to O₃ at those lower levels. Based on the available evidence, [EPA] is not prepared to make these assumptions" (73 FR 16483).

In reconsidering the entire body of evidence available in the 2008 rulemaking, including the Agency's own assessment of the epidemiological evidence in the 2006 Criteria Document, and placing significant weight on the views of CASAC, the Administrator now concludes that important and significant risks to public health are likely to occur at a standard level of 0.075 ppm. She judges that a standard level of 0.075 ppm is not sufficient to provide protection with an adequate margin of safety. In support of this conclusion, the Administrator finds that setting a standard that would protect public health, including the health of at-risk populations, with an adequate margin of

safety should reasonably depend upon giving some weight to the results of the Adams studies and EPA's reanalysis of the Adams's data, and to how effectively alternative standard levels would serve to limit exposures of concern relative to the 0.060 ppm benchmark level as well as to the 0.070 ppm benchmark level. The Administrator notes that EPA's risk assessment estimates comparable risk reductions in going from a 0.074 ppm standard to a 0.064 ppm standard as were estimated in going from the then current 0.084 ppm standard down to a 0.074 ppm standard for an array of health effects analyzed. These estimates include reductions in risk for lung function decrements in all and asthmatic school age children, respiratory symptoms in asthmatic children, respiratory-related hospital admissions, and non-accidental mortality.

Further, based on the exposure assessment estimates discussed above, the Administrator notes that for air quality just meeting a 0.074 ppm standard, approximately 27% of asthmatic school age children and 25% of all school age children are estimated to experience one or more exposures of concern over the 0.060 ppm benchmark level based on simulations for a year with generally poorer air quality; this estimate increases to about 50% of asthmatic and all children in the city with the least degree of protection. The Administrator judges that these estimates are large and strongly suggest significant public health impacts would likely remain in many areas with air quality just meeting a 0.075 ppm O₃ standard.

In light of these estimates and the available evidence, the Administrator agrees with CASAC's conclusion that important public health protections can be achieved by a standard set below 0.075 ppm, within the range of 0.060 to 0.070 ppm. In addition, based on both the evidence- and exposure/risk-based considerations summarized above, the Administrator concludes that a standard set as high as 0.075 would not be considered requisite to protect public health with an adequate margin of safety, and that consideration of lower levels is warranted. In considering such lower levels, the Administrator recognizes that the CAA requires her to reach a public health policy judgment as to what standard would be requisite to protect public health with an adequate margin of safety, based on scientific evidence and technical assessments that have inherent uncertainties and limitations. This judgment requires making reasoned decisions as to what weight to place on various types of

evidence and assessments and on the related uncertainties and limitations.

In selecting a level below 0.075 ppm that would serve as an appropriate upper end for a range of levels to propose, the Administrator has considered a more cautious approach to interpreting the available evidence and exposure/risk-based information—that is, an approach that places significant weight on uncertainties and limitations in the information so as to avoid potentially overestimating public health risks and protection likely to be associated with just meeting a particular standard level. In so doing, she notes that the most certain evidence of adverse health effects from exposure to O₃ comes from the controlled human exposure studies, and that the large bulk of this evidence derives from studies of exposures at levels of 0.080 ppm and above. At those levels, there is consistent evidence of lung function decrements and respiratory symptoms in healthy young adults, as well as evidence of inflammation and other medically significant airway responses. Further, she takes note of the limited but important evidence from controlled human exposure studies indicating that lung function decrements and symptoms can occur in healthy people at levels as low as 0.060 ppm, while also recognizing the limitations in that evidence, as discussed above in sections II.A.1 and II.C.1.a. She also notes that some people with asthma are likely to experience larger and more serious effects than the healthy subjects evaluated in the controlled exposure studies, while recognizing that there is uncertainty about the magnitude of such differences. In considering the available epidemiological studies, she recognizes that they provide evidence of serious respiratory morbidity effects, including respiratory-related emergency department visits and hospital admissions, and non-accidental mortality at levels well below 0.080 ppm, while also recognizing that there is increasing uncertainty associated with the likelihood that such effects occur at decreasing O₃ levels down to background levels. Considering the exposure/risk information, as shown in Table 3, the Administrator observes that a standard set at 0.070 ppm would likely substantially limit exposures of concern relative to the 0.070 ppm benchmark level, while affording far less protection against exposures of concern relative to the 0.060 ppm benchmark level. To the extent that more weight is placed on protection relative to the higher benchmark level, and more weight is placed on the

uncertainties associated with the epidemiological evidence, a standard set at 0.070 ppm might be considered to be adequately protective. Taken together, this type of cautious approach to interpreting the evidence and the exposure/risk information serves as the basis for the Administrator's conclusion that the upper end of the proposed range should be set at 0.070 ppm O₃.

In selecting a level that would serve as an appropriate lower end for a range of levels to propose, the Administrator has considered a more precautionary approach to interpreting the available evidence and exposure/risk-based information—that is, an approach that places less weight on uncertainties and limitations in the information so as to avoid potentially underestimating public health improvements likely to be associated with just meeting a particular standard level. In so doing, the Administrator notes the limited, but important evidence of a lowest-observed-effects level at 0.060 ppm O₃ from controlled human exposure studies reporting lung function decrements and respiratory symptoms in healthy subjects. Notably, these studies also report that a small percentage of subjects (7 to 20 percent) experienced moderate lung function decrements (≥ 10 percent) at the 0.060 ppm exposure level, recognizing that for people with lung disease, such moderate functional or symptomatic responses would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication. In addition, a substantial body of evidence indicates that people with asthma are likely to experience larger and more serious effects than healthy people and therefore controlled human exposure studies done with healthy subjects likely underestimate effects in this at-risk population.

Moreover, epidemiological studies provide evidence of serious respiratory morbidity effects, including respiratory-related emergency department visits and hospital admissions, and non-accidental mortality at O₃ levels that may plausibly extend down to at least 0.060 ppm even when considering the uncertainties inherent in such studies. The Administrator notes that the controlled human exposure studies conducted at 0.060 ppm provide some biological plausibility for associations between respiratory morbidity and mortality effects found in epidemiological studies and O₃ exposures down to 0.060 ppm. Considering the exposure information, as shown in Table 3, the Administrator observes that a standard set at 0.064 ppm would likely essentially eliminate

exposures of concern relative to the 0.070 ppm benchmark level, while appreciably limiting exposures of concern relative to the 0.060 ppm benchmark level to approximately 6 percent of asthmatic children in the aggregate across 12 cities and up to 16 percent in the city that would receive the least protection. While not addressed in the exposure assessment done as part of the 2008 rulemaking, a standard set at 0.060 ppm would be expected to provide somewhat greater protection from such exposures, which is important to the extent that more weight is placed on providing protection relative to the lower benchmark level. Taken together, the Administrator concludes that this precautionary approach to interpreting the evidence and the exposure/risk information supports a level of 0.060 ppm as the lower end of the proposed range.

The Administrator has also concluded that the lower end of the proposed range should not extend below 0.060 ppm O₃. In reaching this conclusion, she gives significant weight to the recommendation of the CASAC panel that 0.060 ppm should be the lower end of the range for consideration (Henderson, 2006c). In the Administrator's view, the evidence from controlled human exposure studies at the 0.060 ppm exposure level, the lowest level tested, is not robust enough to support consideration of a lower level. While some epidemiological studies provide evidence of serious respiratory morbidity effects and non-accidental mortality with no evidence of a threshold, the Administrator notes that other studies provide evidence of a potential threshold somewhat below 0.060 ppm. Moreover, there are limitations in epidemiological studies that make discerning population thresholds difficult, including fewer observations in the range of lower concentrations, concerns related to exposure measurement error, the possible role of copollutants and effects modifiers, and interindividual differences in susceptibility to O₃-related effects. In the Administrator's judgment, these limitations in epidemiological studies, including the limitations in judging the causality of observed associations at lower O₃ levels, and the lack of robust controlled human exposure data at 0.060 ppm make it difficult to interpret this evidence as a basis for a standard level set below 0.060 ppm. Thus, in selecting 0.060 ppm as the lower end of the range for the proposed level of the O₃ standard, the Administrator has taken into

account information on the lowest-observed-effects levels in controlled human exposure studies, indications of possible thresholds reported in some epidemiological studies, the increasing uncertainty in the epidemiological evidence at even lower levels, as well as evidence about increased susceptibility of people with asthma and also other lung diseases. In so doing, she concludes that a primary O₃ standard set below 0.060 ppm would be more than is necessary to protect public health with an adequate margin of safety for at-risk groups.

In reaching her proposed decision, the Administrator has also considered the public comments that were received on the 2007 proposed rule (72 FR 37818). The Administrator notes that there were sharply divergent views expressed by two general sets of commenters with regard to considering the health effects evidence, results of exposure and risk assessments, and the advice of the CASAC panel. On one hand, medical groups, health effects researchers, public health organizations, environmental groups, and some state, tribal and local air pollution control agencies strongly supported a standard set within the range recommended by the CASAC. These commenters generally placed significant weight on the more recent evidence from controlled human exposure studies, down to the 0.060 ppm exposure level, as well as on the epidemiological studies and the results of the exposure and risk assessment conducted for the 2008 rulemaking. Many of these commenters took a more precautionary view and supported a standard set at 0.060 ppm O₃, the lower end of the CASAC recommended range. The Administrator notes that these views are generally consistent with her proposed conclusions. On the other hand, another group of commenters primarily representing industry associations and businesses and some state environmental agencies, primarily expressed the view that the more recent evidence from controlled human exposure, the epidemiological studies, and the results of exposure and human health risk assessments were so uncertain that they did not provide a basis for making any changes to the then current 0.084 ppm O₃ standard set in 1997. This group of commenters generally argued that the health effects evidence newly available in the 2008 rulemaking, the results of the exposure and health risk assessments, and the advice of the CASAC were flawed. For the reasons discussed above, the Administrator does not agree with the

later group of commenters that essentially no weight should be placed on any of the new evidence or assessments that were available for consideration in the 2008 rulemaking.

Based on consideration of the entire body of evidence and information available in the 2008 rulemaking, including exposure and risk estimates, as well as the recommendations of CASAC, the Administrator proposes to set the level of the primary 8-hour O₃ standard to a level within the range of 0.060 to 0.070 ppm. A standard level within this range would reduce the risk of a variety of health effects associated with exposure to O₃, including the respiratory symptoms and lung function effects demonstrated in the controlled human exposure studies, and the respiratory-related emergency department visits, hospital admissions and mortality effects observed in the epidemiological studies. All of these effects are indicative of a much broader array of O₃-related health endpoints, such as school absences and increased medication use, that are plausibly linked to these observed effects. Depending on the weight placed on the evidence and information available in the 2008 rulemaking, as well as the uncertainties and limitations in the evidence and information, a standard could be set within this range at a level that would be requisite to protect public health with an adequate margin of safety.

In reaching this proposed decision, as discussed above, the Administrator has focused on the nature of the increased public health protection that would be afforded by a standard set within the proposed range of levels relative to the protection afforded by the standard set in 2008. Having considered the public comments received on the 2007 proposed rule in reaching this proposed decision that reconsiders the 2008 final rule, the Administrator is interested in again receiving public comment on the benefits to public health associated with a standard set at specific levels within the proposed range relative to the benefits associated with the standard set in 2008.

D. Proposed Decision on the Level of the Primary Standard

For the reasons discussed above, and taking into account information and assessments presented in the 2006 Criteria Document and 2007 Staff Paper, the advice and recommendations of CASAC, and public comments received during the 2008 rulemaking, the Administrator proposes to set a new level for the 8-hour primary O₃ standard. Specifically, the

Administrator proposes to set the level of the 8-hour primary O₃ standard to within a range of 0.060 to 0.070 ppm. The proposed 8-hour primary standard would be met at an ambient air monitoring site when the 3-year average of the annual fourth-highest daily maximum 8-hour average O₃ concentration is less than or equal to the level of the standard that is promulgated. Thus, the Administrator proposes to set a standard with a level within this range. She solicits comment on this range and on the appropriate weight to place on the various types of available evidence, the exposure and risk assessment results, and the uncertainties and limitations related to this information, as well as on the benefits to public health associated with a standard set within this range relative to the benefits associated with the standard set in 2008.

III. Communication of Public Health Information

Information on the public health implications of ambient concentrations of criteria pollutants is currently made available primarily through EPA's Air Quality Index (AQI) program. The current Air Quality Index has been in use since its inception in 1999 (64 FR 42530). It provides accurate, timely, and easily understandable information about daily levels of pollution (40 CFR 58.50). The AQI establishes a nationally uniform system of indexing pollution levels for O₃, carbon monoxide, nitrogen dioxide, particulate matter and sulfur dioxide. The AQI converts pollutant concentrations in a community's air to a number on a scale from 0 to 500. Reported AQI values enable the public to know whether air pollution levels in a particular location are characterized as good (0–50), moderate (51–100), unhealthy for sensitive groups (101–150), unhealthy (151–200), very unhealthy (201–300), or hazardous (300–500). The AQI index value of 100 typically corresponds to the level of the short-term NAAQS for each pollutant. An AQI value greater than 100 means that a pollutant is in one of the unhealthy categories (*i.e.*, unhealthy for sensitive groups, unhealthy, very unhealthy, or hazardous) on a given day; whereas an AQI value at or below 100 means that a pollutant concentration is in one of the satisfactory categories (*i.e.*, moderate or good). Decisions about the pollutant concentrations at which to set the various AQI breakpoints, that delineate the various AQI categories, draw directly from the underlying health information that supports the NAAQS review.

In the 2008 rulemaking, the AQI for O₃ was revised by setting an AQI value of 100 equal to 0.075 ppm, 8-hour average, the level of the revised primary O₃ standard. The other AQI breakpoints were also revised as follows: An AQI value of 50 is set at 0.059 ppm; an AQI value of 150 was set at 0.095 ppm; and an AQI value of 200 was set at 0.115 ppm. All these levels are averaged over 8 hours. These levels were developed by making proportional adjustments to the other AQI breakpoints (*i.e.*, AQI values of 50, 150 and 200).

The Agency recognizes the importance of revising the AQI in a timely manner to be consistent with any revisions to the NAAQS. Therefore, having proposed to set a new level for the 2008 primary 8-hour O₃ standard in this action, EPA also proposes to finalize conforming changes to the AQI in connection with the Agency's final decision on the level of the primary O₃ standard. These conforming changes would include setting the 100 level of the AQI at the same level as that set for the primary O₃ standard resulting from this rulemaking, and also making proportional adjustments to AQI breakpoints at the lower end of the range (*i.e.*, AQI values of 50, 150 and 200). EPA does not propose to change breakpoints at the higher end of the range (from 300 to 500), which would apply to state contingency plans or the Significant Harm Level (40 CFR 51.16), because the information from this reconsideration of the 2008 final rule does not inform decisions about breakpoints at those higher levels.

With respect to reporting requirements (40 CFR Part 58, § 58.50),

EPA proposes to require that the AQI be reported in all metropolitan and micropolitan statistical areas where O₃ monitoring is required, as discussed below in section VI. The Agency solicits comments on our proposed approach to AQI reporting requirements. We are also revising 40 CFR Part 58, § 58.50(c) to require the reporting requirements to be based on the latest available census figures, rather than the most recent decennial U.S. census. This change is consistent with our current practice of using the latest population figures to make monitoring requirements more responsive to changes in population.

IV. Rationale for Proposed Decision on the Secondary Standard

As an initial matter, the Administrator notes that the 2008 final rule concluded that (1) the protection afforded by the 1997 secondary O₃ standard was "not sufficient and that the standard needs to be revised to provide additional protection from known and anticipated adverse effects on sensitive natural vegetation and sensitive ecosystems, and that such a revised standard could also be expected to provide additional protection to sensitive ornamental vegetation" and (2) "that there is not adequate information to establish a separate secondary standard based on other effects of O₃ on public welfare" (73 FR 16497). The Administrator is not reconsidering these aspects of the 2008 decision, which are based on the reasons discussed in section IV.B of the 2008 final rule (73 FR 16489–16497). The Administrator also notes that the 2008 final rule concluded that it was appropriate to retain the O₃ indicator for

the secondary O₃ standard. The Administrator is not reconsidering this aspect of the 2008 decision, which was based on the reasons discussed in sections IV.B and IV.C of the 2008 final rule (73 FR 16489–16497). For these reasons, the Administrator is not reopening the 2008 decision with regard to the need to revise the 1997 secondary O₃ standard to provide additional protection from known and anticipated adverse effects on sensitive natural vegetation and sensitive ecosystems, nor with regard to the appropriate indicator for the secondary standard. Thus, the information that follows in this section specifically focuses on a reconsideration of the 8-hour secondary O₃ standard set in the 2008 final rule for the purpose of determining whether and, if so, how to revise the form, averaging time, and level of the standard to provide appropriate protection from known and anticipated adverse effects on sensitive natural vegetation and sensitive ecosystems.

This section presents the rationale for the Administrator's proposed decision that the secondary O₃ standard, which was set identical to the revised primary standard in the 2008 final rule, should instead be a new cumulative, seasonal standard. This standard is expressed in terms of a concentration-weighted form commonly called W126, which uses a sigmoidal weighting function to assign a weight to each hourly O₃ concentration within the 12-hour daylight period (8 am to 8 pm). This daily ozone index is defined as follows:

$$\text{daily W126} = \sum_{i=8am}^{i=8pm} w_i C_i, \text{ where } C_i = \text{hourly O}_3 \text{ at hour } i, \text{ and } w_i = \frac{1}{1 + 4403e^{-126C}}$$

The daily index values are then summed over each month within the O₃ season, and the annual highest consecutive three month sum is determined. The proposed standard consists of the three-year average of this highest three-month statistic, set at a level within the range of 7 to 15 ppm-hours.

As discussed more fully below, the rationale for this proposed new standard is based on a thorough review, in the 2006 Criteria Document, of the latest scientific information on vegetation, ecological and other public welfare effects associated with the presence of O₃ in the ambient air. This rationale also takes into account and is consistent with: (1) Staff assessments of the most

policy-relevant information in the 2006 Criteria Document and staff analyses of air quality, vegetation effects evidence, exposure, and risks, presented in the 2007 Staff Paper, upon which staff recommendations for revisions to the secondary O₃ standard are based; (2) CASAC advice and recommendations as reflected in discussions of drafts of the 2006 Criteria Document and 2007 Staff Paper at public meetings, in separate written comments, and in CASAC's letters to the Administrator, both before and after the 2008 rulemaking, and (3) public comments received during development of these documents, either in conjunction with CASAC meetings or separately; and on the 2007 proposed

rule, and (4) consideration of the degree of protection to vegetation potentially afforded by the 2008 8-hour standard.

In developing this rationale, the Administrator has again focused on direct O₃ effects on vegetation, specifically drawing upon an integrative synthesis of the entire body of evidence (EPA, 2006a, chapter 9), published through early 2006, on the broad array of vegetation effects associated with the presence of O₃ in the ambient air. In addition, because O₃ can also indirectly affect other ecosystem components such as soils, water, and wildlife, and their associated ecosystem goods and services, through its effects on vegetation, a qualitative discussion of these other indirect impacts is also

included, though these effects were not quantifiable at the time of the 2008 rulemaking. As discussed below in section IV.A, the peer-reviewed literature includes studies conducted in the U.S., Canada, Europe, and many other countries around the world.⁵³ In reconsidering this evidence, as was concluded in the 2008 rulemaking, and based on the body of scientific literature assessed in the 2006 Criteria Document, the Administrator continues to believe that it is reasonable to conclude that a secondary standard protecting the public welfare from known or anticipated adverse effects to trees and native vegetation would also afford increased protection from adverse effects to other environmental components relevant to the public welfare, including ecosystem services and function. Section IV.B focuses on considerations related to biologically relevant exposure indices. This rationale also draws upon the results of quantitative exposure and risk assessments, discussed below in section IV.C. Section IV.D focuses on the considerations upon which the Administrator's proposed conclusions are based. Considerations regarding a cumulative seasonal standard as well as an 8-hour standard are discussed, and the rationale for the 2008 decision on the secondary standard and CASAC advice, given both prior to the development of the 2007 proposed rule and following the 2008 final rule, are summarized. Finally, the Administrator's proposed conclusions on the secondary standard are presented. Section IV.E summarizes the proposed decision on the secondary O₃ standard and the solicitation of public comments.

As with virtually any policy-relevant vegetation effects research, there is uncertainty in the characterization of vegetation effects attributable to exposure to ambient O₃. As discussed below, however, research conducted since the 1997 review provides important information coming from field-based exposure studies, including free air, gradient, and biomonitoring surveys, in addition to the more traditional open top chamber (OTC) studies. Moreover, the newly available studies evaluated in the 2006 Criteria Document have undergone intensive scrutiny through multiple layers of peer review and many opportunities for public review and comment. While

important uncertainties remain, the review of the vegetation effects information has been extensive and deliberate. In the judgment of the Administrator, the intensive evaluation of the scientific evidence that has occurred provides an adequate basis for this reconsideration of the 2008 rulemaking.

A. Vegetation Effects Information

This section outlines key information contained in the 2006 Criteria Document (chapter 9) and in the 2007 Staff Paper (chapter 7) on known or anticipated effects on public welfare associated with the presence of O₃ in ambient air. The information highlighted here summarizes: (1) New information available in the 2008 rulemaking on potential mechanisms for vegetation effects associated with exposure to O₃; (2) the nature of effects on vegetation that have been associated with exposure to O₃ and consequent potential impacts on ecosystems; and (3) considerations in characterizing what constitutes an adverse welfare impact of O₃.

Exposures to O₃ have been associated quantitatively and qualitatively with a wide range of vegetation effects. The decision in the 1997 review to set a more protective secondary standard primarily reflected consideration of the quantitative information on vegetation effects available at that time, particularly growth impairment (*e.g.*, biomass loss) in sensitive forest tree species during the seedling growth stage and yield loss in important commercial crops. This information, derived mainly using the open top chamber (OTC) exposure method, found cumulative, seasonal O₃ exposures were most strongly associated with observed vegetation response. The 2006 Criteria Document discusses a number of additional studies that support and strengthen key conclusions regarding O₃ effects on vegetation and ecosystems found in the previous Criteria Document (EPA, 1996a, 2006a), including further clarification of the underlying mechanistic and physiological processes at the sub-cellular, cellular, and whole system levels within the plant. More importantly, however, in the context of this review, new quantitative information is now available across a broader array of vegetation effects (*e.g.*, growth impairment during seedlings, saplings and mature tree growth stages, visible foliar injury, and yield loss in annual crops) and across a more diverse set of exposure methods, including chamber, free air, gradient, model, and field-based observation. The non-chambered, field-based study results

begin to address one of the key data gaps cited by EPA in the 1997 review.

The following discussion of the policy-relevant science regarding vegetation effects associated with cumulative, seasonal exposures to ambient levels of O₃ integrates information from the 2006 Criteria Document (chapter 9) and the 2007 Staff Paper (chapter 7).

1. Mechanisms

Scientific understanding regarding O₃ impacts at the genetic, physiological, and mechanistic levels helps to explain the biological plausibility and coherence of the evidence for O₃-induced vegetation effects and informs the interpretation of predictions of risk associated with vegetation response at ambient O₃ exposure levels. In most cases, the mechanisms of response are similar regardless of the degree of sensitivity of the species. The evidence assessed in the 2006 Criteria Document (EPA, 2006a) regarding the O₃-induced changes in physiology continues to support the information discussed in the 1997 review (EPA, 1996a, 2006a). In addition, during the last decade understanding of the cellular processes within plants has been further clarified and enhanced. This section reviews the key scientific conclusions identified in 1996 Criteria Document (EPA, 1996a), and incorporates recent information from the Criteria Document (EPA, 2006a). This section describes: (1) Plant uptake of O₃, (2) O₃-induced cellular to systemic response, (3) plant compensation and detoxification mechanisms, (4) O₃-induced changes to plant metabolism, and (5) plant response to chronic O₃ exposures.

a. Plant Uptake of Ozone

To cause injury, O₃ must first enter the plant through openings in the leaves called stomata. Leaves exist in a three dimensional environment called the plant canopy, where each leaf has a unique orientation and receives a different exposure to ambient air, microclimatological conditions, and sunlight. In addition, a plant may be located within a stand of other plants which further modifies ambient air exchange with individual leaves. Not all O₃ entering a plant canopy is absorbed into the leaf stomata, but may be adsorbed to other surfaces *e.g.*, leaf cuticles, stems, and soil (termed non-stomatal deposition) or scavenged by reactions with intra-canopy biogenic VOCs and naturally occurring NO_x emissions from soils. Because O₃ does not typically penetrate the leaf's cuticle, it must reach the stomatal openings in the leaf for absorption to occur. The

⁵³ In its assessment of the evidence judged to be most relevant to making decisions on the level of the O₃ secondary standard, however, EPA has placed greater weight on U.S. studies, due to the often species-, site- and climate-specific nature of O₃-related vegetation response.

movement of O₃ and other gases such as CO₂ into and out of leaves is controlled by stomatal guard cells that regulate the size of the stomatal apertures. These guard cells respond to a variety of internal species-specific factors as well as external site specific environmental factors such as light, temperature, humidity, CO₂ concentration, soil fertility, water status, and in some cases, the presence of air pollutants, including O₃. These modifying factors produce stomatal conductances that vary between leaves of the same plant, individuals and genotypes within a species as well as diurnally and seasonally.

b. Cellular to Systemic Response

Once inside the leaf, O₃ can react with a variety of biochemical compounds that are exposed to the air spaces within the leaf or it can be dissolved into the water lining the cell wall of the air spaces. Once in the aqueous phase, O₃ can be rapidly altered to form oxidative products that can diffuse more readily into and through the cell and react with many biochemical compounds. Early steps in a series of O₃-induced events that can lead to leaf injury seems to involve alteration in cell membrane function, including membrane transport properties (EPA, 2006a) and/or reactions with organic molecules that in certain circumstances result in the generation of signaling compounds. The generation of such signaling compounds can lead to a cascade of events. One such signaling molecule is hydrogen peroxide (H₂O₂). The presence of higher-than-normal levels of H₂O₂ within the leaf is a potential trigger for a set of metabolic reactions that include those typical of the well documented "wounding" response or pathogen defense pathway generated by cutting of the leaf or by pathogen/insect attack. Ethylene is another compound produced when plants are subjected to biotic or abiotic stressors. Increased ethylene production by plants exposed to O₃ stress was identified as a consistent marker for O₃ exposure in studies conducted decades ago (Tingey *et al.*, 1976).

c. Compensation and Detoxification

Ozone injury will not occur if (1) the rate and amount of O₃ uptake is small enough for the plant to detoxify or metabolize O₃ or its metabolites or (2) the plant is able to repair or compensate for the O₃ impacts (Tingey and Taylor, 1982; U.S. EPA, 1996a). With regard to the first, a few studies have documented direct stomatal closure or restriction in the presence of O₃ in some species, which limits O₃ uptake and potential subsequent injury. This response may

be initiated ranging from within minutes to hours or days of exposure (Moldau *et al.*, 1990; Dann and Pell, 1989; Weber *et al.*, 1993). However, exclusion of O₃ simultaneously restricts the uptake of CO₂, which also limits photosynthesis and growth. In addition, antioxidants present in plants can effectively protect tissue against damage from low levels of oxidants by dissipating excess oxidizing power. Since 1996, the role of detoxification in providing a level of resistance to O₃ has been further investigated. A number of antioxidants have been found in plants. However, the pattern of changes in the amounts of these antioxidants varies greatly among different species and conditions. Most recent reports indicate that ascorbate within the cell wall provides the first significant opportunity for detoxification to occur. In spite of the new research, however, it is still not clear as to what extent detoxification protects against O₃ injury. Specifically, data are needed on potential rates of antioxidant production, sub-cellular location(s) of antioxidants, and whether generation of these antioxidants in response to O₃-induced stress potentially diverts resources and energy away from other vital uses. Thus, the 2006 Criteria Document concludes that scientific understanding of the detoxification mechanisms is not yet complete and requires further investigation (EPA, 2006a).

Regarding the second, once O₃ injury has occurred in leaf tissue, some plants are able to repair or compensate for the impacts. In general, plants have a variety of compensatory mechanisms for low levels of stress including reallocation of resources, changes in root/shoot ratio, production of new tissue, and/or biochemical shifts, such as increased photosynthetic capacity in new foliage and changes in respiration rates, indicating possible repair or replacement of damaged membranes or enzymes. Since these mechanisms are genetically determined, not all plants have the same complement of compensatory mechanisms or degree of tolerance, and these may vary over the life of the plant as not all stages of a plant's development are equally sensitive to O₃. At higher levels or over longer periods of O₃ stress, some of these compensatory mechanisms, such as a reallocation of resources away from storage in the roots in favor of leaves or shoots, could occur at a cost to the overall health of the plant. However, it is not yet clear to what degree or how the use of plant resources for repair or compensatory processes affects the

overall carbohydrate budget or subsequent plant response to O₃ or other stresses (EPA, 1996a, EPA, 2006a).

d. Changes to Plant Metabolism

Ozone inhibits photosynthesis, the process by which plants produce energy rich compounds (*e.g.*, carbohydrates) in the leaves. This impairment can result from direct impact to chloroplast function and/or O₃-induced stomatal closure resulting in reduced uptake of CO₂. A large body of literature published since 1996 has further elucidated the mechanism of effect of O₃ within the chloroplast. Pell *et al.* (1997) showed that O₃ exposure results in a loss of the central carboxylating enzyme that plays an important role in the production of carbohydrates. Due to its central importance, any decrease in this enzyme may have severe consequences for the plant's productivity. Several recent studies have found that O₃ has a greater effect as leaves age, with the greatest impact of O₃ occurring on the oldest leaves (Fiscus *et al.*, 1997; Reid and Fiscus, 1998; Noormets *et al.*, 2001; Morgan *et al.*, 2004). The loss of this key enzyme as a function of increasing O₃ exposure is also linked to an early senescence or a speeding up of normal development leading to senescence. If total plant photosynthesis is sufficiently reduced, the plant will respond by reallocating the remaining carbohydrate at the level of the whole organism (EPA, 1996a, 2006a). This reallocation of carbohydrate away from the roots into above ground vegetative components can have serious implications for perennial species, as discussed below.

e. Plant Response to Chronic Ozone Exposures

Though many changes that occur with O₃ exposure can be observed within hours, or perhaps days, of the exposure, including those connected with wounding, other effects take longer to occur and tend to become most obvious after chronic seasonal exposures to low O₃ concentrations. These lower chronic exposures have been linked to senescence or some other physiological response very closely linked to senescence. In perennial plant species, a reduction in carbohydrate storage in one year may result in the limitation of growth the following year (Andersen *et al.*, 1997). Such "carry-over" effects have been documented in the growth of tree seedlings (Hogsett *et al.*, 1989; Sasek *et al.*, 1991; Temple *et al.*, 1993; EPA, 1996a) and in roots (Andersen *et al.*, 1991; EPA, 1996a). Though it is not fully understood how chronic seasonal O₃ exposure affects long-term growth and resistance to other biotic and abiotic

insults in long-lived trees, accumulation of these carry-over effects over time could affect survival and reproduction.

2. Nature of Effects

Ozone injury at the cellular level can accumulate sufficiently to induce effects at the level of a whole leaf or plant. These larger scale effects can include: Reduced carbohydrate production and/or reallocation; reduced growth and/or reproduction; visible foliar injury and/or premature senescence; and reduced plant vigor. Much of what is now known about these O₃-related effects, as summarized below, is based on research that was available in the 1997 review. Studies available in the 2008 rulemaking continue to support and expand this knowledge (EPA, 2006a).

a. Carbohydrate Production and Allocation

When total plant photosynthesis is sufficiently reduced, the plant will respond by reallocating the remaining carbohydrate at the level of the whole organism. Many studies have demonstrated that root growth is more sensitive to O₃ exposure than stem or leaf growth (EPA, 2006a). When fewer carbohydrates are present in the roots, less energy is available for root-related functions such as acquisition of water and nutrients. In addition, by inhibiting photosynthesis and the amount of carbohydrates available for transfer to the roots, O₃ can disrupt the association between soil fungi and host plants. Fungi in the soil form a symbiotic relationship with many terrestrial plants. For host plants, these fungi improve the uptake of nutrients, protect the roots against pathogens, produce plant growth hormones, and may transport carbohydrates from one plant to another (EPA, 1996a). These below ground effects have recently been documented in the field (Grunke *et al.*, 1998; Grunke and Balduman, 1999). Data from a long-studied pollution gradient in the San Bernardino Mountains of southern California suggest that O₃ substantially reduces root growth in natural stands of Ponderosa pine (*Pinus ponderosa*). Root growth in mature trees was decreased at least 87 percent in a high-pollution site as compared to a low-pollution site (Grunke *et al.*, 1998), and a similar pattern was found in a separate study with whole-tree harvest along this gradient (Grunke and Balduman, 1999). Though effects on other ecosystem components were not examined, a reduction of root growth of this magnitude could have significant implications for the below-ground communities at those sites. Because effects on leaf and needle carbohydrate

content under O₃ stress can range from a reduction (Barnes *et al.*, 1990; Miller *et al.*, 1989), to no effect (Alscher *et al.*, 1989), to an increase (Luethy-Krause and Landolt, 1990), studies that examine only above-ground vegetative components may miss important O₃-induced changes below ground. These below-ground changes could signal a shift in nutrient cycling with significance at the ecosystem level (Young and Sanzone, 2002).

b. Growth Effects on Trees

Studies comparing the O₃-related growth response of different vegetation types (coniferous and deciduous) and growth stages (*e.g.*, seedling and mature) have established that on average, individual coniferous trees are less sensitive than deciduous trees, and deciduous trees are generally less sensitive to O₃ than most annual plants, with the exception of a few fast growing deciduous tree species (*e.g.*, quaking aspen, black cherry, and cottonwood), which are highly sensitive and, in some cases, as much or more sensitive to O₃ than sensitive annual plants. In addition, studies have shown that the relationship between O₃ sensitivity in seedling and mature growth stages of trees can vary widely, with seedling growth being more sensitive to O₃ exposures in some species, while in others, the mature growth stage is the more O₃ sensitive. In general, mature deciduous trees are likely to be more sensitive to O₃ than deciduous seedlings, and mature evergreen trees are likely to be less sensitive to O₃ than their seedling counterparts. Based on these results, stomatal conductance, O₃ uptake, and O₃ effects cannot be assumed to be equivalent in seedlings and mature trees.

In the 1997 review (EPA, 1996b), analyses of the effects of O₃ on trees were limited to 11 tree species for which concentration-response (C-R) functions for the seedling growth stage had been developed from OTC studies conducted by the National Health and Environmental Effects Research Lab, Western Ecology Division (NHEERL-WED). A number of replicate studies were conducted on these species, leading to a total of 49 experimental cases. The 2007 Staff Paper presented a graph of the composite regression equation that combines the results of the C-R functions developed for each of the 49 cases. The NHEERL-WED study predicted relative biomass loss at various exposure levels in terms of a 12-hour W126. For example, 50 percent of the tree seedling cases would be protected from greater than 10 percent biomass loss at a 3-month, 12-hour

W126 of approximately 24 ppm-hour, while 75 percent of cases would be protected from 10 percent biomass loss at a 3-month, 12-hour W126 level of approximately 16 ppm-hour.

Since the 1997 review, only a few studies have developed C-R functions for additional tree seedling species (EPA, 2006a). One such study is of particular importance because it documented growth effects in the field of a similar magnitude as those previously seen in OTC studies but without the use of chambers or other fumigation methods (Gregg *et al.*, 2003). This study placed eastern cottonwood (*Populus deltoides*) saplings at sites along a continuum of ambient O₃ exposures that gradually increased from urban to rural areas in the New York City area (Gregg *et al.*, 2003). Eastern cottonwood is a fast growing O₃ sensitive tree species that is important ecologically along streams and commercially for pulpwood, furniture manufacturing, and as a possible new source for energy biomass (Burns and Hankola, 1990). Gregg *et al.* (2003) found that the cottonwood saplings grown in urban New York City grew faster than saplings grown in downwind rural areas. Because these saplings were grown in pots with carefully controlled soil nutrient and moisture levels, the authors were able to control for most of the differences between sites. After carefully considering these and other factors, the authors concluded the primary explanation for the difference in growth was the gradient of cumulative O₃ exposures that increased as one moved downwind from urban to less urban and more rural sites. It was determined that the lower O₃ exposure within the city center was due to NO_x titration reactions which removed O₃ from the ambient air. The authors were able to reproduce the growth responses observed in the field in a companion OTC experiment, confirming O₃ as the stressor inducing the growth loss response (Gregg *et al.*, 2003).

Another recent set of studies employed a modified Free Air CO₂ Enrichment (FACE) methodology to expose vegetation to elevated O₃ without the use of chambers. This exposure method was originally developed to expose vegetation to elevated levels of CO₂, but was later modified to include O₃ exposure in Illinois (SoyFACE) and Wisconsin (AspenFACE) for soybean and deciduous trees, respectively (Dickson *et al.*, 2000; Morgan *et al.*, 2004). The FACE method releases gas (*e.g.*, CO₂, O₃) from a series of orifices placed along the length of the vertical pipes surrounding a circular field plot and uses the

prevailing wind to distribute it. This exposure method has many characteristics that differ from those associated with the OTC. Most significantly, this exposure method more closely replicates conditions in the field than do OTCs. This is because, except for O₃ levels which are varied across co-located plots, plants are exposed to the same ambient growing conditions that occur naturally in the field (e.g., location-specific pollutant mixtures; climate conditions such as light, temperature and precipitation; insect pests, pathogens). By using one of several co-located plots as a control (e.g., receives no additional O₃), and by exposing the other rings to differing levels of elevated O₃, the growth response signal that is due solely to the change in O₃ exposure can be clearly determined. Furthermore, the FACE system can expand vertically with the growth of trees, allowing for exposure experiments to span numerous years, an especially useful capability in forest research.

On the other hand, the FACE methodology also has the undesirable characteristic of potentially creating hotspots near O₃ gas release orifices or gradients of exposure in the outer ring of trees within the plots, such that averaging results across the entire ring potentially overestimates the response. In recognition of this possibility, researchers at the AspenFACE experimental site only measured trees in the center core of each ring, (e.g., at least 5–6 meters away from the emission sites of O₃) (Dickson *et al.*, 2000, Karnosky *et al.*, 2005). By taking this precaution, it is unlikely that their measurements were influenced by any potential hotspots or gradients of exposure within the FACE rings. Taking all of the above into account, results from the Wisconsin FACE site on quaking aspen appear to demonstrate that the detrimental effects of O₃ exposure seen on tree growth and symptom expression in OTCs can be observed in the field using this exposure method (Karnosky *et al.*, 1999; 2005).

The 2007 Staff Paper thus concluded that the combined evidence from the AspenFACE and Gregg *et al.* (2003) field studies provide compelling and important support for the appropriateness of continued use of the C–R functions derived using OTC from the NHEERL–WED studies to estimate risk to these tree seedlings under ambient field exposure conditions. These studies make a significant contribution to the coherence of the weight of evidence available in this review and provide additional evidence that O₃-induced effects observed in chambers also occur in the field.

Trees and other perennials, in addition to cumulating the effects of O₃ exposures over the annual growing season, can also cumulate effects across multiple years. It has been reported that effects can “carry over” from one year to another (EPA, 2006a). Growth affected by a reduction in carbohydrate storage in one year may result in the limitation of growth in the following year (Andersen *et al.*, 1997). Carry-over effects have been documented in the growth of some tree seedlings (Hogsett *et al.*, 1989; Simini *et al.*, 1992; Temple *et al.*, 1993) and in roots (Andersen *et al.*, 1991; EPA, 1996a). On the basis of past and recent OTC and field study data, ambient O₃ exposures that occur during the growing season in the United States are sufficient to potentially affect the annual growth of a number of sensitive seedling tree species. However, because most studies do not take into account the possibility of carry over effects on growth in subsequent years, the true implication of these annual biomass losses may be missed. It is likely that under ambient exposure conditions, some sensitive trees and perennial plants could experience compounded impacts that result from multiple year exposures.

c. Visible Foliar Injury

Cellular injury to leaves due to exposure to O₃ can and often does become visible. Acute injury usually appears within 24 hours after exposure to O₃ and, depending on species, can occur under a range of exposures and durations from 0.040 ppm for a period of 4 hours to 0.410 ppm for 0.5 hours for crops and 0.060 ppm for 4 hours to 0.510 ppm for 1 hour for trees and shrubs (Jacobson, 1977). Chronic injury may be mild to severe. In some cases, cell death or premature leaf senescence may occur. The significance of O₃ injury at the leaf and whole plant levels depends on how much of the total leaf area of the plant has been affected, as well as the plant's age, size, developmental stage, and degree of functional redundancy among the existing leaf area. As a result, it is not presently possible to determine, with consistency across species and environments, what degree of injury at the leaf level has significance to the vigor of the whole plant.

The presence of visible symptoms due to O₃ exposures can, however, by itself, represent an adverse impact to the public welfare. Specifically, it can reduce the market value of certain leafy crops (such as spinach, lettuce), impact the aesthetic value of ornamentals (such as petunia, geranium, and poinsettia) in urban landscapes, and affect the

aesthetic value of scenic vistas in protected natural areas such as national parks and wilderness areas. Many businesses rely on healthy looking vegetation for their livelihoods (e.g., horticulturalists, landscapers, Christmas tree growers, farmers of leafy crops) and a variety of ornamental species have been listed as sensitive to O₃ (Abt Associates Inc., 1995). Though not quantified, there is likely some level of economic impact to businesses and homeowners from O₃-related injury on sensitive ornamental species due to the cost associated with more frequent replacement and/or increased maintenance (fertilizer or pesticide application). In addition, because O₃ not only results in discoloration of leaves but can lead to more rapid senescence (early shedding of leaves) there potentially could be some lost tourist dollars at sites where fall foliage is less available or attractive.

The use of sensitive plants as biological indicators to detect phytotoxic levels of O₃ is a longstanding and effective methodology (Chappelka and Samuelson, 1998; Manning and Krupa, 1992). Each bioindicator exhibits typical O₃ injury symptoms when exposed under appropriate conditions. These symptoms are considered diagnostic as they have been verified in exposure-response studies under experimental conditions. In recent years, field surveys of visible foliar injury symptoms have become more common, with greater attention to the standardization of methods and the use of reliable indicator species (Campbell *et al.*, 2000; Smith *et al.*, 2003). Specifically, the United States Forest Service (USFS) through the Forest Health Monitoring Program (FHM) (1990–2001) and currently the Forest Inventory and Analysis (FIA) Program collects data regarding the incidence and severity of visible foliar injury on a variety of O₃ sensitive plant species throughout the U.S. (Coulston *et al.*, 2003, 2004; Smith *et al.*, 2003).

Since the conclusion of the 1997 review, the FIA monitoring program network and database has continued to expand. This network continues to document foliar injury symptoms in the field under ambient exposure conditions. Recent survey results show that O₃-induced foliar injury incidence is widespread across the country. The visible foliar injury indicator has been identified as a means to track O₃ exposure stress trends in the nation's natural plant communities as highlighted in EPA's most recent Report on the Environment (EPA, 2003a; <http://www.epa.gov/indicators/roe>).

Previous Criteria Documents have noted the difficulty in relating visible foliar injury symptoms to other vegetation effects such as individual tree growth, stand growth, or ecosystem characteristics (EPA, 1996a) and this difficulty remains to the present day (EPA, 2006a). It is important to note that direct links between O₃ induced visible foliar injury symptoms and other adverse effects are not always found. Therefore, visible foliar injury cannot serve as a reliable surrogate measure for other O₃-related vegetation effects because other effects (e.g., biomass loss) have been reported with and without visible injury. In some cases, visible foliar symptoms have been correlated with decreased vegetative growth (Karnosky *et al.*, 1996; Peterson *et al.*, 1987; Somers *et al.*, 1998) and with impaired reproductive function (Black *et al.*, 2000; Chappelka, 2002). Therefore, the lack of visible injury should not be construed to indicate a lack of phytotoxic concentrations of O₃ nor absence of other non-visible O₃ effects.

d. Reduced Plant Vigor

Though O₃ levels over most of the U.S. are not high enough to kill vegetation directly, current levels have been shown to reduce the ability of many sensitive species and genotypes within species to adapt to or withstand other environmental stresses. These O₃ effects may include increased susceptibility to freezing temperatures, increased vulnerability to pest infestations and/or root disease, and compromised ability to compete for available resources. As an example of the latter, when species with differing O₃-sensitivities occur together, O₃-sensitive species may experience a greater reduction in growth than more O₃-tolerant species, which then can better compete for available resources. The result of such above effects can produce a loss in plant vigor in O₃-sensitive species that over time may lead to premature plant death.

e. Ecosystems

Ecosystems are comprised of complex assemblages of organisms and the physical environment with which they interact. Each level of organization within an ecosystem has functional and structural characteristics. At the ecosystem level, functional characteristics include, but are not limited to, energy flow; nutrient, hydrologic, and biogeochemical cycling; and maintenance of food chains. The sum of the functions carried out by ecosystem components provides many benefits to humankind, as in the case of

forest ecosystems (Smith, 1992). Some of these benefits, also termed "ecosystem goods and services", include food, fiber production, aesthetics, genetic diversity, maintenance of water quality, air quality, and climate, and energy exchange. A conceptual framework for discussing the effects of stressors, including air pollutants such as O₃, on ecosystems was developed by the EPA Science Advisory Board (Young and Sanzone, 2002). In this report, the authors identify six essential ecological attributes (EEAs) of ecosystems including landscape condition, biotic condition, chemical/physical condition, ecological processes, hydrology/geomorphology, and natural disturbance regime. Each EEA is depicted as one of six triangles that together build a hexagon. On the outside of each triangle is a list of stressors that can act on the EEA. Tropospheric O₃ is listed as a stressor of both biotic condition and the chemical/physical condition of ecosystems. As each EEA is linked to all the others, it is clearly envisioned in this framework that O₃ could either directly or indirectly impact all of the EEAs associated with an ecosystem that is being stressed by O₃.

Vegetation often plays an influential role in defining the structure and function of an ecosystem, as evidenced by the use of dominant vegetation forms to classify many types of natural ecosystems, e.g., tundra, wetland, deciduous forest, and conifer forest. Plants simultaneously inhabit both above-and below-ground environments, integrating and influencing key ecosystem cycles of energy, water, and nutrients. When a sufficient number of individual plants within a community have been affected, O₃-related effects can be propagated up to ecosystem-level effects. Thus, through its impact on vegetation, O₃ can be an important ecosystem stressor.

i. Potential Ozone Alteration of Ecosystem Structure and Function

The 2006 Criteria Document outlines seven case studies where O₃ effects on ecosystems have either been documented or are suspected. The oldest and clearest example involves the San Bernardino Mountain forest ecosystem in California. This system experienced chronic high O₃ exposures over a period of 50 or more years. The O₃-sensitive and co-dominant species of ponderosa and Jeffrey pine demonstrated severe levels of foliar injury, premature senescence, and needle fall that decreased the photosynthetic capacity of stressed pines and reduced the production of carbohydrates resulting in a decrease in

radial growth and in the height of stressed trees. It was also observed that ponderosa and Jeffrey pines with slight to severe crown injury lost basal area in relation to competing species that are more tolerant to O₃. Due to a loss of vigor, these trees eventually succumbed to the bark beetle, leading to elevated levels of tree death. Increased mortality of susceptible trees shifted the community composition towards white fir and incense cedar, effectively reversing the development of the normal fire climax mixture dominated by ponderosa and Jeffrey pines, and leading to increased fire susceptibility. At the same time, numerous other organisms and processes were also affected either directly or indirectly, including successional patterns of fungal microflora and their relationship to the decomposer community. Nutrient availability was influenced by the heavy litter and thick needle layer under stands with the most severe needle injury and defoliation. In this example, O₃ appeared to be a predisposing factor that led to increased drought stress, windthrow, root diseases, and insect infestation (Takemoto *et al.*, 2001). Thus, through its effects on tree water balance, cold hardiness, tolerance to wind, and susceptibility to insect and disease pests, O₃ potentially impacted the ecosystem-related EEA of natural disturbance regime (e.g., fire, erosion). Although the role of O₃ was extremely difficult to separate from other confounding factors, such as high nitrogen deposition, there is evidence that this shift in species composition has altered the structure and dynamics of associated food webs (Pronos *et al.*, 1999) and carbon (C) and nitrogen (N) cycling (Arbaugh *et al.*, 2003). Ongoing and new research in this important ecosystem is needed to reveal the extent to which ecosystem services have been affected and to what extent strong causal linkages between historic and/or current ambient O₃ exposures and observed ecosystem-level effects can be made.

Ozone has also been reported to be a selective pressure among sensitive tree species (e.g., eastern white pine) in the east. The nature of community dynamics in eastern forests is different, however, than in the west, consisting of a wider diversity of species and uneven aged stands, and the O₃ levels are less severe. Therefore, lower level chronic O₃ stress in the east is more likely to produce subtle long-term forest responses such as shifts in species composition, rather than wide-spread community degradation.

Some of the best-documented studies of population and community response

to O₃ effects are the long-term studies of common plantain (*Plantago major*) in native plant communities in the United Kingdom (Davison and Reiling, 1995; Lyons *et al.*, 1997; Reiling and Davison, 1992c). Elevated O₃ significantly decreased the growth of sensitive populations of common plantain (Pearson *et al.*, 1996; Reiling and Davison, 1992a, b; Whitfield *et al.*, 1997) and reduced its fitness as determined by decreased reproductive success (Pearson *et al.*, 1996; Reiling and Davison, 1992a). While spatial comparisons of population responses to O₃ are complicated by other environmental factors, rapid changes in O₃ resistance were imposed by ambient levels and variations in O₃ exposure (Davison and Reiling, 1995). Specifically, in this case study, it appeared that O₃-sensitive individuals are being removed by O₃ stress and the genetic variation represented in the population could be declining. If genetic diversity and variation is lost in ecosystems, there may be increased vulnerability of the system to other biotic and abiotic stressors, and ultimately a change in the EEAs and associated services provided by those ecosystems.

Recent free-air exposure experiments have also provided new insight into how O₃ may be altering ecosystem structure and function (Karnosky *et al.*, 2005). For example, a field O₃ exposure experiment at the AspenFACE site in Wisconsin (described in section IV.A.2.b. above) was designed to examine the effects of both elevated CO₂ and O₃ on mixed stands of aspen (*Populus tremuloides*), birch (*Betula papyrifera*), and sugar maple (*Acer saccharum*) that are characteristic of Great Lakes aspen-dominated forests (Karnosky *et al.*, 2003; Karnosky *et al.*, 1999). They found evidence that the effects on above- and below-ground growth and physiological processes have cascaded through the ecosystem, even affecting microbial communities (Larson *et al.*, 2002; Phillips *et al.*, 2002). This study also confirmed earlier observations of O₃-induced changes in trophic interactions involving keystone tree species, as well as important insect pests and their natural enemies (Awmack *et al.*, 2004; Holton *et al.*, 2003; Percy *et al.*, 2002).

Collectively these examples suggest that O₃ is an important stressor in natural ecosystems, but it is difficult to quantify the contribution of O₃ due to the combination of other stresses present in ecosystems. In most cases, because only a few components in each of these ecosystems have been examined and characterized for O₃ effects, the full

extent of ecosystem changes in these example ecosystems is not fully understood. Clearly, there is a need for highly integrated ecosystem studies that specifically investigate the effect of O₃ on ecosystem structure and function in order to fully determine the extent to which O₃ is altering ecosystem services. Continued research, employing new approaches, will be necessary to fully understand the extent to which O₃ is affecting ecosystem services.

ii. Effects on Ecosystem Services and Carbon Sequestration

Since it has been established that O₃ affects photosynthesis and growth of plants, O₃ is most likely affecting the productivity of forest ecosystems. Therefore, it is desirable to link effects on growth and productivity to essential ecosystem services. However, it is very difficult to quantify ecosystem-level productivity losses because of the amount of complexity in scaling from the leaf-level or individual plant to the ecosystem level, and because not all organisms in an ecosystem are equally affected by O₃.

Terrestrial ecosystems are important in the Earth's carbon (C) balance and could help offset emissions of CO₂ by humans if anthropogenic C is sequestered in vegetation and soils. The annual increase in atmospheric CO₂ is less than the total inputs from fossil fuel burning and land use changes (Prentice *et al.*, 2001), and much of this discrepancy is thought to be attributable to CO₂ uptake by plant photosynthesis (Tans & White, 1998). Temperate forests of the northern hemisphere have been estimated to be a net sink of about 0.6 to 0.7 petagrams (Pg) C per year (Goodale *et al.* 2002). Ozone interferes with photosynthesis, causes some plants to senesce leaves prematurely, and in some cases, reduces allocation to stem and root tissue. Thus, O₃ decreases the potential for C sequestration. For the purposes of this discussion, C sequestration is defined as the net exchange of carbon by the terrestrial biosphere. However, long-term storage in the soil organic matter is considered to be the most stable form of C storage in ecosystems.

In a study including all ecosystem types, Felzer *et al.* (2004), estimated that U.S. net primary production (net flux of C into an ecosystem) was decreased by 2.6–6.8 percent due to O₃ pollution in the late 1980s to early 1990s. Ozone not only reduces C sequestration in existing forests, it can also affect reforestation projects (Beedlow *et al.* 2004). This effect, in turn, has been found to ultimately inhibit C sequestration in forest soils which act as long-term C

storage (Loya *et al.*, 2003; Beedlow *et al.* 2004). The interaction of rising O₃ pollution and rising CO₂ concentrations in the coming decades complicates predictions of future sequestration potential. Models generally predict that, in the future, C sequestration will increase with increasing CO₂, but often do not account for the decrease in productivity due to the local effects of current or potentially increasing levels of tropospheric O₃. In the presence of high O₃ levels, the stimulatory effect of rising CO₂ concentrations on forest productivity has been estimated to be reduced by more than 20 percent (Tingey *et al.*, 2001; Ollinger *et al.* 2002; Karnosky *et al.*, 2003).

In summary, it would be anticipated that meeting lower O₃ standards would increase the amount of CO₂ uptake by many ecosystems in the U.S. However, the amount of this improvement would be heavily dependent on the species composition of those ecosystems. Many ecosystems in the U.S. do have O₃ sensitive plants. For example, forest ecosystems with dominant species such as aspen or ponderosa pine would be expected to increase CO₂ uptake more with lower O₃ than forests with more O₃ tolerant species.

A recent critique of the secondary NAAQS review process published in the report by the National Academy of Sciences on Air Quality Management in the United States (NRC, 2004) stated that "EPA's current practice for setting secondary standards for most criteria pollutants does not appear to be sufficiently protective of sensitive crops and ecosystems * * *" This report made several specific recommendations for improving the secondary NAAQS process and concluded that "There is growing evidence that tighter standards to protect sensitive ecosystems in the United States are needed. * * *" An effort has been recently initiated within the Agency to identify indicators of ecological condition whose responses can be clearly linked to changes in air quality that are attributable to Agency environmental programs. Using a single indicator to represent the complex linkages and dynamic cycles that define ecosystem condition will always have limitations. With respect to O₃-related impacts on ecosystem condition, only two candidate indicators, foliar injury (as described above) and radial growth in trees, have been suggested. Thus, while at the present time, most O₃-related effects on ecosystems must be inferred from observed or predicted O₃-related effects on individual plants, additional research at the ecosystem level could identify new indicators and/or establish stronger causal linkages

between O₃-induced plant effects and ecosystem condition.

f. Yield Reductions in Crops

Ozone can interfere with carbon gain (photosynthesis) and allocation of carbon with or without the presence of visible foliar injury. As a result of decreased carbohydrate availability, fewer carbohydrates are available for plant growth, reproduction, and/or yield. Recent studies have further confirmed and demonstrated O₃ effects on different stages of plant reproduction, including pollen germination, pollen tube growth, fertilization, and abortion of reproductive structures, as reviewed by Black *et al.* (2000). For seed-bearing plants, these reproductive effects will culminate in reduced seed production or yield.

As described in the 1997 review and again in the 2006 Criteria Document and 2007 Staff Paper, the National Crop Loss Assessment Network (NCLAN) studies undertaken in the early to mid-1980s provide the largest, most uniform database on the effects of O₃ on agricultural crop yields. 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 O₃, sub-ambient O₃, and above ambient O₃. Robust C-R functions were developed for each of these crop species. These results showed that 50 percent of the studied cases would be protected from greater than 10 percent yield loss at a W126 level of 21 ppm-hour, while a W126 of 13 ppm-hour would provide protection for 75 percent of the cases studied from greater than 10 percent yield loss.

Recent studies continue to find yield loss levels in crop species studied previously under NCLAN that reflect the earlier findings. In other words, there has been no evidence that crops are becoming more tolerant of O₃ (EPA, 2006a). For cotton, some newer varieties have been found to have higher yield loss due to O₃ compared to older varieties (Olszyk *et al.*, 1993, Grantz and

McCool, 1992). In a meta-analysis of 53 studies, Morgan *et al.* (2003) found consistent deleterious effects of O₃ exposures on soybean from studies published between 1973 and 2001. Further, early results from the field-based exposure experiment SoyFACE in Illinois indicate a lack of any apparent difference in the O₃ tolerance of old and recent cultivars of soybean in a study of 22 soybean varieties (Long *et al.*, 2002). Thus, the 2007 Staff Paper concluded that the recent scientific literature continues to support the conclusions of the 1996 Criteria Document that ambient O₃ concentrations are reducing the yield of major crops in the U.S.

In addition to the effects described on annual crop species, several studies published since the 1997 review have focused on perennial forage crops (EPA, 2006a). These recent results confirm that O₃ is also impacting yields and quality of multiple-year forage crops at sufficient magnitude to have nutritional and possibly economic implications to their use as ruminant animal feed at O₃ exposures that occur in some years over large areas of the U.S.

3. Adversity of Effects

The 2007 Staff Paper recognized that the statute requires that a secondary standard be protective against "adverse" O₃ effects, not all identifiable O₃-induced effects. In considering what constitutes a vegetation effect that is adverse to the public welfare, the 2007 Staff Paper recognizes that O₃ can cause a variety of vegetation effects, beginning at the level of the individual cell and accumulating up to the level of whole leaves, plants, plant populations, communities and whole ecosystems, not all of which have been classified in past reviews as "adverse" to public welfare.

Previous reviews have classified O₃ vegetation effects as either "injury" or "damage" to help in determining adversity. Specifically, "injury" is defined as encompassing all plant reactions, including reversible changes or changes in plant metabolism (*e.g.*, altered photosynthetic rate), altered plant quality, or reduced growth, that does not impair the intended use or value of the plant (Guderian, 1977). In contrast, "damage" has been defined to include those injury effects that reach sufficient magnitude as to also reduce or impair the intended use or value of the plant. Examples of effects that are classified as damage include reductions in aesthetic values (*e.g.*, foliar injury in ornamental species) as well as losses in terms of weight, number, or size of the plant part that is harvested (reduced yield or biomass production). Yield loss also may include changes in crop

quality, *i.e.*, physical appearance, chemical composition, or the ability to withstand storage, while biomass loss includes slower growth in species harvested for timber or other fiber uses. While this construct has proved useful in the past, it appears to be most useful in the context of evaluating effects on single plants or species grown in monocultures such as agricultural crops or managed forests. It is less clear how it might apply to potential effects on natural forests or entire ecosystems when O₃-induced species level impacts lead to shifts in species composition and/or associated ecosystem services such as nutrient cycling or hydrologic cycles, where the intended use or value of the system has not been specifically identified.

A more recent construct for assessing risks to forests described in Hogsett *et al.* (1997) suggests that "adverse effects could be classified into one or more of the following categories: (1) Economic production, (2) ecological structure, (3) genetic resources, and (4) cultural values." This approach expands the context for evaluating the adversity of O₃-related effects beyond the species level. Another recent publication, *A Framework for Assessing and Reporting on Ecological Condition: An SAB report* (Young and Sanzone, 2002), provides additional support for expanding the consideration of adversity beyond the species level by making explicit the linkages between stress-related effects (*e.g.*, O₃ exposure) at the species level and at higher levels within an ecosystem hierarchy. Taking this recent literature into account, the 2007 Staff Paper concludes that a determination of what constitutes an "adverse" welfare effect in the context of the secondary NAAQS review can appropriately occur within this broader paradigm.

B. Biologically Relevant Exposure Indices

The 2006 Criteria Document concluded that O₃ exposure indices that cumulate differentially weighted hourly concentrations are the best candidates for relating exposure to plant growth responses. This conclusion follows from the extensive evaluation of the relevant studies in the 1996 Criteria Document (EPA, 1996a) and the recent evaluation of studies that have been published since that time. The following selections, taken from the 1996 Criteria Document (EPA, 1996a, section 5.5), further elucidate the depth and strength of these conclusions. Specifically, with respect to the importance of taking into account exposure duration, the 1996 Criteria Document stated, "when O₃ effects are the primary cause of variation

in plant response, plants from replicate studies of varying duration showed greater reductions in yield or growth when exposed for the longer duration” and “the mean exposure index of unspecified duration could not account for the year-to-year variation in response” (EPA, 1996a, pg. 5–96). Further, “because the mean exposure index treats all concentrations equally and does not specifically include an exposure duration component, the use of a mean exposure index for characterizing plant exposures appears inappropriate for relating exposure with vegetation effects” (EPA, 1996a, pg. 5–88). Regarding the relative importance of higher concentrations than lower in determining plant response, the 1996 Criteria Document concluded that “the ultimate impact of long-term exposures to O₃ on crops and seedling biomass response depends on the integration of repeated peak concentrations during the growth of the plant” (EPA, 1996a, pg. 5–104). Further, “at this time, exposure indices that weight the hourly O₃ concentrations differentially appear to be the best candidates for relating exposure with predicted plant response” (EPA, 1996a, pgs. 5–136).

At the conclusion of the 1997 review, the biological basis for a cumulative, seasonal form was not in dispute. There was general agreement between EPA and CASAC, based on their review of the air quality criteria, that a cumulative, seasonal form was more biologically based than the then current 1-hour and newly proposed 8-hour average form. However, in selecting a specific form appropriate for a secondary standard, there was less agreement. An evaluation of the performance of several cumulative seasonal forms in predicting plant response data taken from OTC experiments had found that all performed about equally well and was unable to distinguish between them (EPA, 1996a). In selecting between two of these cumulative forms, the SUM06⁵⁴ and W126, in the absence of biological evidence to distinguish between them, EPA based its decision on both science and policy considerations. Specifically, these were: (1) All cumulative, peak-weighted exposure indices considered, including W126 and SUM06, were about equally good as exposure measures to predict exposure-response relationships reported in the NCLAN crop studies; and (2) the SUM06 form would not be influenced by PRB O₃ concentrations (defined at the time as

0.03 to 0.05 ppm) under many typical air quality distributions. On the basis of these considerations, EPA chose the SUM06 as the most appropriate cumulative, seasonal form to consider when proposing an alternative secondary standard form (61 FR 65716).

Though the scientific justification for a cumulative, seasonal form was generally accepted in the 1997 review, an analysis undertaken by EPA at that time had shown that there was considerable overlap between areas that would be expected not to meet the range of alternative 8-hour standards being considered for the primary NAAQS and those expected not to meet the range of values (expressed in terms of the seasonal SUM06 index) of concern for vegetation. This result suggested that improvements in national air quality expected to result from attaining an 8-hour primary standard within the recommended range of levels would also be expected to significantly reduce levels of concern for vegetation in those same areas. Thus, in the 1996 proposed rule, EPA proposed two alternatives for consideration: one alternative was to make the secondary standard equal in every way to the proposed 8-hour, 0.08 ppm primary standard; and the second was to establish a cumulative, seasonal secondary standard in terms of a SUM06 form as also appropriate to protect public welfare from known or anticipated adverse effects given the available scientific knowledge and that such a seasonal standard “* * * is more biologically relevant * * *” (61 FR 65716).

In the 1997 final rule, EPA decided to make the secondary standard identical to the primary standard. The EPA acknowledged, however, that “it remained uncertain as to the extent to which air quality improvements designed to reduce 8-hr average O₃ concentrations averaged over a 3-year period would reduce O₃ exposures measured by a seasonal SUM06 index.” (62 FR 38876) In other words, it was uncertain as to whether the 8-hour average form would, in practice, provide sufficient protection for vegetation from the cumulative, seasonal and concentration-weighted exposures described in the scientific literature as of concern.

On the basis of that history, the 2007 Staff Paper (chapter 7) revisited the issue of whether the SUM06 was still the most appropriate choice of cumulative, seasonal form for a secondary standard to protect the public welfare from known and anticipated adverse vegetation effects in light of the new information available in this review. Specifically, the 2007 Staff

Paper considered: (1) The continued lack of evidence within the vegetation effects literature of a biological threshold for vegetation exposures of concern; and (2) new estimates of PRB that were lower than in the 1997 review. The W126 form, also evaluated in the 1997 review, was again selected for comparison with the SUM06 form. Regarding the first consideration, the 2007 Staff Paper noted that the W126 form, by its incorporation of a continuous sigmoidal weighting scheme, does not create an artificially imposed concentration threshold, yet also gives proportionally more weight to the higher and typically more biologically potent concentrations, as supported by the scientific evidence. Second, the index value is not significantly influenced by O₃ concentrations within the range of estimated PRB, as the weights assigned by the sigmoidal weighting scheme to concentrations in this range are near zero. Thus, it would also provide a more appropriate target for air quality management programs designed to reduce emissions from anthropogenic sources contributing to O₃ formation. On the basis of these considerations, the 2007 Staff Paper concluded that the W126 form was the most biologically-relevant cumulative, seasonal form appropriate to consider in the context of the 2008 rulemaking.

C. Vegetation Exposure and Impact Assessment

The vegetation exposure and impact assessment conducted for the 2008 rulemaking and described in the 2007 Staff paper, consisted of exposure, risk and benefits analyses and improved and built upon similar analyses performed in the 1997 review (EPA 1996b). The vegetation exposure assessment was performed using interpolation and included information from ambient monitoring networks and results from air quality modeling. The vegetation risk assessment included both tree and crop analyses. The tree risk analysis includes three distinct lines of evidence: (1) Observations of visible foliar injury in the field linked to monitored O₃ air quality for the years 2001–2004; (2) estimates of seedling growth loss under then current and alternative O₃ exposure conditions; and (3) simulated mature tree growth reductions using the TREGRO model to simulate the effect of meeting alternative air quality standards on the predicted annual growth of a single western species (ponderosa pine) and two eastern species (red maple and tulip poplar). The crop analysis includes estimates of the risks to crop yields from then current and alternative

⁵⁴ The SUM06 index is defined as the sum of all hourly O₃ concentrations greater or equal to 0.06 ppm over a specified time.

O₃ exposure conditions and the associated change in economic benefits expected to accrue in the agriculture sector upon meeting the levels of various alternative standards. Each element of the assessment is described below, including discussions of known sources and ranges of uncertainties associated with the elements of this assessment.

1. Exposure Characterization

Though numerous effects of O₃ on vegetation have been documented as discussed above, it is important in considering risk to examine O₃ air quality patterns in the U.S. relative to the location of O₃ sensitive species that have a known concentration-response in order to predict whether adverse effects are occurring at current levels of air quality, and whether they are likely to occur under alternative standard forms and levels.

The most important information about exposure to vegetation comes from the O₃ monitoring data that are available from two national networks: (1) Air Quality System (AQS; <http://www.epa.gov/ttn/airs/airsaqs>) and (2) Clean Air Status and Trends Network (CASTNET; <http://www.epa.gov/castnet/>). The AQS monitoring network currently has over 1100 active O₃ monitors which are generally sited near population centers. However, this network also includes approximately 36 monitors located in national parks. CASTNET is the nation's primary source for data on dry acidic deposition and rural, ground-level O₃. It consists of over 80 sites across the eastern and western U.S. and is cooperatively operated and funded with the National Park Service. In the 1997 O₃ NAAQS final rule, it was acknowledged that because the national air quality surveillance network for O₃ was designed principally to monitor O₃ exposure in populated areas, there was limited measured data available to characterize O₃ air quality in rural and remote sites. Since the 1997 review, there has been a small increase in the number of CASTNET sites (from approximately 52 sites in 1992 to 84 sites in 2004), however these monitors are not used for attainment designations.

National parks represent areas of nationally recognized ecological and public welfare significance, which have been afforded a high level of protection by Congress. Two recent reports presented some discussion of O₃ trends in a subset of national parks: The Ozone Report: Measuring Progress Through 2003 (EPA, 2004), and 2005 Annual Performance and Progress Report: Air

Quality in National Parks (NPS, 2005). Unfortunately, much of this information is presented only in terms of the current 8-hr average form. The 2007 Staff Paper analyzed available air quality data in terms of the cumulative 12-hour W126 form from 2001 to 2005 for a subset of national parks and other significant natural areas representing 4 general regions of the U.S. Many of these national parks and natural areas have monitored O₃ levels above concentrations that have been shown to decrease plant growth and above the 12-hour W126 levels analyzed in this review. For example, the Great Smokey Mountain, Rocky Mountain, Grand Canyon, Yosemite and Sequoia National Parks all had more than one year within the 2001–2005 period with a 12-hour W126 above 21 ppm-hour. This level of exposure has been associated with approximately no more than 10 percent biomass loss in 50 percent of the 49 tree seedling cases studied in the NHEERL–WED experiments (Lee and Hogsett, 1996). Black cherry (*Prunus serotina*), an important O₃-sensitive tree species in the eastern U.S., occurs in the Great Smoky Mountain National Park and is estimated to have O₃-related seedling biomass loss of approximately 40 percent when exposed to a 3 month, 12-hour W126 O₃ level greater than 21 ppm-hour. Ponderosa pine (*Pinus ponderosa*) which occurs in the Grand Canyon, Yosemite and Sequoia National Parks has been reported to have approximately 10 percent biomass losses at 3 month, 12 hour W126 O₃ levels as low as 17 ppm-hour (Lee and Hogsett, 1996). Impacts on seedlings may potentially affect long-term tree growth and survival, ultimately affecting the competitiveness of O₃-sensitive tree species and genotypes within forest stands.

In order to characterize exposures to vegetation at the national scale, however, the 2007 Staff Paper concluded that it could not rely solely on limited site-specific monitoring data, and that it was necessary to select an interpolation method that could be used to characterize O₃ air quality over broad geographic areas. The 2007 Staff Paper therefore investigated the appropriateness of using the O₃ outputs from the EPA/NOAA Community Multi-scale Air Quality (CMAQ)⁵⁵ model

⁵⁵ The CMAQ model is a multi-pollutant, multiscale air quality model that contains state-of-the-science techniques for simulating atmospheric and land processes that affect the transport, transformation, and deposition of atmospheric pollutants and/or their precursors on both regional and urban scales. It is designed as a science-based modeling tool for handling many major pollutants (including photochemical oxidants/O₃, particulate

system (<http://www.epa.gov/asmdnerl/CMAQ>, Byun and Ching, 1999; Arnold *et al.* 2003, Eder and Yu, 2005) to improve spatial interpolations based solely on existing monitoring networks. Due to the significant resources required to run CMAQ, model outputs were only available for a limited number of years. For the 2008 rulemaking, the most recent outputs available at the time from CMAQ version 4.5 were for the year 2001.

Based on the significant difference in monitor network density between the eastern and western U.S., the 2007 Staff Paper concluded that it was appropriate to use separate interpolation techniques in these two regions. Only AQS and CASTNET monitoring data were used for the eastern interpolation, since it was determined that enhancing the interpolation with CMAQ data did not add much information to the eastern U.S. interpolation. In the western U.S., however, where rural monitoring is more sparse, O₃ values generated by the CMAQ model were used to develop scaling factors to augment the interpolation.

In order to characterize uncertainties associated with the interpolation method, monitored O₃ concentrations were systematically compared to interpolated O₃ concentrations in areas where monitors were located. In general, the interpolation method used in the current review performed well in many areas in the U.S., although it under-predicted higher 12-hour W126 exposures in rural areas. Due to the important influence of higher exposures in determining risks to plants, this feature of the interpolated surface could result in an under-estimation of risks to vegetation in some areas. Taking these uncertainties into account, and given the absence of more complete rural monitoring data, this approach was used in developing national vegetation exposure and risk assessments that estimate relative changes in risk for the various alternative standards analyzed.

To evaluate changing vegetation exposures and risks under selected air quality scenarios, the 2007 Staff Paper utilized 2001 base year O₃ air quality distributions that had been adjusted with a rollback method (Horst and Duff, 1995; Rizzo, 2005, 2006) to reflect meeting the then current and alternative secondary standard options. This technique combines both linear and quadratic elements to reduce higher O₃

matter, and nutrient deposition) holistically. The CMAQ model can generate estimates of hourly O₃ concentrations for the contiguous U.S., making it possible to express model outputs in terms of a variety of exposure indices (e.g., W126, 8-hour average).

concentrations more than lower ones. In this regard, the rollback method attempts to account for reductions in emissions without greatly affecting lower concentrations. The following O₃ air quality scenarios were analyzed: (1) 4th-highest daily maximum 8-hour average: 0.084 ppm (the effective level of the then current standard) and 0.070 ppm levels; (2) 3-month, 12-hour. SUM06: 25 ppm-hour (proposed in the 1997 review) and 15 ppm-hour levels; and (3) 3-month, 12-hour W126: 21 ppm-hour and 13 ppm-hour levels.

The two 8-hour average levels were chosen as possible alternatives of the then current form for comparison with the cumulative, seasonal alternative forms. The SUM06 scenarios were very similar to the W126 scenarios. Since the W126 was judged to be the more biologically-relevant cumulative, seasonal form, only the results for the W126 scenarios are summarized below. For the W126 form, the two levels were selected on the basis of the associated levels of tree seedling biomass loss and crop yield loss protection identified in the NHEERL-WED and NCLAN studies, respectively. Specifically, the upper level of W126 (21 ppm-hour) was associated with a level of tree and crop protection of approximately no more than 10 percent growth or yield loss in 50 percent of cases studied. Alternatively, the lower level of W126 (13 ppm-hour) was associated with a level of tree seedling and crop protection of approximately no more than 10 percent growth or yield loss in 75 percent of studied cases.

The following discussion highlights key observations drawn from comparing predicted changes in interpolated air quality under each alternative standard form and level scenario for the base year, 2001:

(1) Under the base year (2001) "as is" air quality, a large portion of California had 12-hr W126 O₃ levels above 31 ppm-hour, which has been associated with approximately no more than 14 percent biomass loss in 50 percent of tree seedling cases studies. Broader multi-state regions in the east (NC, TN, KY, IN, OH, PA, NJ, NY, DE, MD, VA) and west (CA, NV, AZ, OK, TX) are predicted to have levels of air quality above the W126 level of 21 ppm-hour, which is approximately equal to the secondary standard proposed in 1996 and is associated with approximately no more than 10 percent biomass loss in 50 percent of tree seedling cases studied. Much of the east and Arizona and California have 12-hour W126 O₃ levels above 13 ppm-hour, which has been associated with approximately no more than 10 percent biomass loss in 75

percent of tree seedling cases studied. The results of the exposure assessment indicate that current air quality levels could result in significant impacts to vegetation in some areas.

(2) When 2001 air quality was rolled back to meet the then current 8-hour, 0.084 ppm secondary standard, the overall 3-month 12-hour W126 O₃ levels were somewhat improved, but not substantially. Under this scenario, there were still many areas in California with 12-hour W126 O₃ levels above 31 ppm-hour. A broad multi-state region in the east (NC, TN, KY, IN, OH, PA, MD) and west (CA, NV, AZ, OK, TX) were still predicted to have O₃ levels above the W126 level of 21 ppm-hour.

(3) Exposures generated for just meeting a 0.070 ppm, 4th-highest maximum 8-hour average alternative standard showed substantially improved O₃ air quality when compared to just meeting the then current 8-hour standard. Most areas were predicted to have O₃ levels below the W126 level of 21 ppm-hour, although some areas in the east (KY, TN, MI, AR, MO, IL) and west (CA, NV, AZ, UT, NM, CO, OK, TX) were still predicted to have O₃ levels above the W126 level of 13 ppm-hour.

These results suggest that meeting a 0.070 ppm, 8-hour secondary standard would provide substantially improved protection in some areas for vegetation from seasonal O₃ exposures of concern. The 2007 Staff Paper recognizes, however, that some areas meeting a 0.070 ppm 8-hour standard could continue to have elevated seasonal exposures, including forested park lands and other natural areas, and Class I areas which are federally mandated to preserve certain air quality related values. This is especially important in the high elevation forests in the Western U.S. where there are few O₃ monitors. This is because the air quality patterns in remote areas can result in relatively low 8-hour averages while still experiencing relatively high cumulative exposures.

To further characterize O₃ air quality in terms of various secondary standard forms, an analysis was performed in the 2007 Staff Paper to evaluate the extent to which county-level O₃ air quality measured in terms of various levels of the current 8-hour average form overlapped with that measured in terms of various levels of the 12-hour W126 cumulative, seasonal form. The 2007 Staff Paper presented this analysis using 2002–2004⁵⁶ county-level O₃ air quality

⁵⁶ This analysis was updated using 2003–2005 air quality as it became available, finding similar results.

data from AQS sites and the subset of CASTNET sites having the highest O₃ levels for the counties in which they are located. Since the current 8-hour average secondary form is a 3-year average, the analysis initially compared the 3-year averages of both the 8-hour and W126 forms. In addition, recognizing that some vegetation effects (e.g. crop yield loss and foliar injury) are driven solely by annual O₃ exposures and are typically evaluated with respect to exposures within the annual growing season, the 2007 Staff Paper also presented a comparison of the current 3-year average 8-hour form to the annual W126 form for the individual years, 2002 and 2004.

Results of the 3-year average comparisons showed that of the counties with air quality meeting the 3-year average form of a 0.084 ppm, 8-hour average standard, 7 counties showed 3-year average W126 values above the 21 ppm-hour level. At the lower W126 level of 13 ppm-hour, 135 counties with air quality meeting the 3-year average form of a 0.084 ppm, 8-hour average standard, would be above this W126 level. In addition, when the 3-year average of an 8-hour form was compared to annual W126 values, further variability in the degree of overlap between the 8-hour form and W126 form became apparent. For example, the relatively high 2002 O₃ air quality year showed a greater degree of overlap between those areas that would meet the levels analyzed for the current 8-hour and alternative levels of the W126 form than did the relatively low O₃ 2004 air quality year. This lack of a consistent degree of overlap between the two forms in different air quality years demonstrates that annual vegetation would be expected to receive widely differing degrees of protection from cumulative seasonal exposures in some areas from year to year, even when the 3-year average of the 8-hour form was consistently met.

It is clear that this analysis is limited by the lack of monitoring in rural areas where important vegetation and ecosystems are located, especially at higher elevation sites. This is because O₃ air quality distributions at high elevation sites often do not reflect the typical urban and near-urban pattern of low morning and evening O₃ concentrations with a high mid-day peak, but instead maintain relatively flat patterns with many concentrations in the mid-range (e.g., 0.05–0.09 ppm) for extended periods. These conditions can lead to relatively low daily maximum 8-hour averages concurrently with high cumulative values so that there is potentially less overlap between an 8-

hour average and a cumulative, seasonal form at these sites. The 2007 Staff Paper concluded that it is reasonable to anticipate that additional unmonitored rural high elevation areas important for vegetation may not be adequately protected even with a lower level of the 8-hour form.

The 2006 Criteria Document discusses policy relevant background (PRB) levels for high elevation sites and makes the following observations: (1) PRB concentrations of 0.04 to 0.05 ppm occur occasionally at high-elevation sites (*e.g.*, > 1.5 km) in the spring due to the free-tropospheric influence, including some limited contribution from hemispheric pollution (O₃ produced from anthropogenic emissions outside North America); and (2) while stratospheric intrusions might occasionally elevate O₃ at high-altitude sites, these events are rare at surface sites. Therefore, the 2007 Staff Paper concluded that springtime PRB levels in the range identified above and rare stratospheric intrusions of O₃ are unlikely to be a major influence on 3-month cumulative seasonal W126 values.

It further remains uncertain as to the extent to which air quality improvements designed to reduce 8-hour O₃ average concentrations would reduce O₃ exposures measured by a seasonal, cumulative W126 index. The 2007 Staff Paper indicated this to be an important consideration because: (1) The biological database stresses the importance of cumulative, seasonal exposures in determining plant response; (2) plants have not been specifically tested for the importance of daily maximum 8-hour O₃ concentrations in relation to plant response; and (3) the effects of attainment of a 8-hour standard in upwind urban areas on rural air quality distributions cannot be characterized with confidence due to the lack of monitoring data in rural and remote areas. These factors are important considerations in determining whether the current 8-hour form can appropriately provide requisite protection for vegetation.

2. Assessment of Risks to Vegetation

The 2007 Staff Paper presents results from quantitative and qualitative risk assessments of O₃ risks to vegetation (EPA, 2007). In the 1997 review, crop yield and seedling biomass loss OTC data provided the basis for staff analyses, conclusions, and recommendations (EPA, 1996b). Since then, several additional lines of evidence have progressed sufficiently to provide staff with a more complete and

coherent picture of the scope of O₃-related vegetation risks, especially those faced by seedling, sapling and mature tree species growing in field settings, and indirectly, forested ecosystems. Specifically, research published after the 1997 review reflects an increased emphasis on field-based exposure methods (*e.g.*, free air exposure and ambient gradient), improved field survey biomonitoring techniques, and mechanistic tree process models. Findings from each of these research areas are discussed separately below. In conducting these assessments, the Staff Paper analyses relied on both measured and modeled air quality information. For some effects, like visible foliar injury and modeled mature tree growth response, only monitored air quality information was used. For other effects categories (*e.g.*, crop yield and tree seedling growth), staff relied on interpolated O₃ exposures.

a. Visible Foliar Injury

As discussed above (section IV.A.2.c), systematic injury surveys have documented visible foliar injury symptoms diagnostic of phytotoxic O₃ exposures on sensitive bioindicator plants. These surveys have produced more expansive evidence than that available at the time of the 1997 review that visible foliar injury is occurring in many areas of the U.S. under current ambient conditions. The 2007 Staff Paper presents an assessment combining recent U.S. Forest Service Forest Inventory and Analysis (FIA) biomonitoring site data with the county level air quality data for those counties containing the FIA biomonitoring sites. This assessment showed that incidence of visible foliar injury ranged from 21 to 39 percent during the four-year period (2001–2004) across all counties with air quality levels at or below that of a 0.084 ppm, 8-hour standard. Of the counties that met an 8-hour level of 0.070 ppm in those years, 11 to 30 percent still had incidence of visible foliar injury. The magnitude of these percentages suggests that phytotoxic exposures sufficient to induce visible foliar injury would still occur in many areas after meeting the level of a 0.084 ppm secondary standard or alternative 0.070 ppm 8-hour standard. Additionally, the data showed that visible foliar injury occurrence was geographically widespread and occurring on a variety of plant species in forested and other natural systems. Linking visible foliar injury to other plant effects is still problematic. However, its presence indicates that other O₃-related vegetation effects could also be present.

b. Seedling and Mature Tree Biomass Loss

In the 1997 review, analyses of the effects of O₃ on trees were limited to 11 tree species for which C–R functions for the seedling growth stage had been developed from OTC studies conducted by the NHEERL–WED. Important tree species such as quaking aspen, ponderosa pine, black cherry, and tulip poplar were found to be sensitive to cumulative seasonal O₃ exposures. Work done since the 1997 review at the AspenFACE site in Wisconsin on quaking aspen (Karnosky *et al.*, 2005) and a gradient study performed in the New York City area (Gregg *et al.* 2003) has confirmed the detrimental effects of O₃ exposure on tree growth in field studies without chambers and beyond the seedling stage (King *et al.* 2005). These field studies are discussed above in section IV.A.

To update the seedling biomass loss risk analysis, C–R functions for biomass loss for available seedling tree species taken from the 2006 Criteria Document and information on tree growing regions derived from the U.S. Department of Agriculture's Atlas of United States Trees were combined with projections of O₃ air quality based on 2001 interpolated exposures, to produce estimated biomass loss for each of the seedling tree species individually. Maps of these biomass loss projections are presented in the 2007 Staff Paper. For example, quaking aspen had a wide range of O₃ exposures across its growing range and therefore, showed significant variability in percentages of projected seedling biomass loss across its range. Quaking aspen seedling biomass loss was projected to be greater than 4 percent over much of its geographic range, though it can reach above 10 percent in areas of Ohio, Pennsylvania, New York, New Jersey and California. Biomass loss for black cherry was projected to be greater than 20 percent in approximately half its range. Greater than 30 percent biomass loss for black cherry was projected in North Carolina, Tennessee, Indiana, Ohio, Pennsylvania, Arizona, Michigan, New York, New Jersey, Maryland and Delaware. For ponderosa pine, an important tree species in the western U.S., biomass loss was projected to be above 10 percent in much of its range in California. Biomass loss still occurred in many tree species when O₃ air quality was adjusted to meet the then current 8-hour standard of 0.084 ppm. For instance, black cherry, ponderosa pine, eastern white pine, and aspen had estimated median seedling biomass losses over portions of their growing

range as high as 24, 11, 6, and 6 percent, respectively, when O₃ air quality was rolled back to just meet a 0.084 ppm, 8-hour standard. The 2007 Staff Paper noted that these results are for tree seedlings and that mature trees of the same species may have more or less of a response to O₃ exposure. Due to the potential for compounding effects over multiple years, experts at a consensus workshop on O₃ vegetation effects and secondary standards, hereinafter referred to as the 1996 Consensus Workshop, reported in a subsequent 1997 Workshop Report, that a biomass loss greater than 2 percent annually can be significant (Heck and Cowling, 1997). Decreased seedling root growth and survivability could affect overall stand health and composition in the long term.

In addition to the estimation of O₃ effects on seedling growth, recent work available in the 2008 rulemaking has enhanced our understanding of risks beyond the seedling stage. In order to better characterize the potential O₃ effects on mature tree growth, a tree growth model (TREGRO) was used as a tool to evaluate the effect of changing O₃ air quality under just meet scenarios for selected alternative O₃ standards on the growth of mature trees. TREGRO is a process-based, individual tree growth simulation model (Weinstein *et al.*, 1991). This model has been used to evaluate the effects of a variety of O₃ exposure scenarios on several species of trees by incorporating concurrent climate data in different regions of the U.S. to account for O₃ and climate/meteorology interactions (Tingey *et al.*, 2001; Weinstein *et al.*, 1991; Retzlaff *et al.*, 2000; Laurence *et al.*, 1993; Laurence *et al.*, 2001; Weinstein *et al.*, 2005). The model provides an analytical framework that accounts for the nonlinear relationship between O₃ exposure and response. The interactions between O₃ exposure, precipitation and temperature are integrated as they affect vegetation, thus providing an internal consistency for comparing effects in trees under different exposure scenarios and climatic conditions. An earlier assessment of the effectiveness of national ambient air quality standards in place since the early 1970s took advantage of 40 years of air quality and climate data for the Crestline site in the San Bernardino Mountains of California to simulate ponderosa pine growth over time with the improving air quality using TREGRO (Tingey *et al.*, 2004).

The TREGRO model was used to assess growth of Ponderosa pine in the San Bernardino Mountains of California (Crestline) and the growth of yellow poplar and red maple in the

Appalachian mountains of Virginia and North Carolina, Shenandoah National Park (Big Meadows) and Linville Gorge Wilderness Area (Cranberry), respectively. Total tree growth associated with “as is” air quality, and air quality adjusted to just meet alternative O₃ standards was assessed. Ponderosa pine is one of the most widely distributed pines in western North America, a major source of timber, important as wildlife habitat, and valued for aesthetics (Burns and Honkala, 1990). Red maple is one of the most abundant species in the eastern U.S. and is important for its brilliant fall foliage and highly desirable wildlife browse food (Burns and Honkala, 1990). Yellow poplar is an abundant species in the southern Appalachian forest. It is 10 percent of the cove hardwood stands in southern Appalachians which are widely viewed as some of the country’s most treasured forests because the protected, rich, moist set of conditions permit trees to grow the largest in the eastern U.S. The wood has high commercial value because of its versatility and as a substitute for increasingly scarce softwoods in furniture and framing construction. Yellow poplar is also valued as a honey tree, a source of wildlife food, and a shade tree for large areas (Burns and Honkala, 1990).

The 2007 Staff Paper analyses found that just meeting a 0.084 ppm standard would likely continue to allow O₃-related reductions in annual net biomass gain in these species. This is based on model outputs that estimate that as O₃ levels are reduced below those of a 0.084 ppm standard, significant improvements in growth would occur. For instance, estimated growth in red maple increased by 4 and 3 percent at Big Meadows and Cranberry sites, respectively, when air quality was rolled back to just met a W126 value of 13 ppm-hour. Yellow poplar was projected to have a growth increase between 0.6 and 8 percent under the same scenario at the two eastern sites.

Though there is uncertainty associated with the above analyses, this information should be given careful consideration in light of several other pieces of evidence. Specifically, new evidence from experimental studies that go beyond the seedling growth stage continues to show decreased growth under elevated O₃ (King *et al.* 2005). Some mature trees such as red oak have shown an even greater sensitivity of photosynthesis to O₃ than seedlings of the same species (Hanson *et al.*, 1994). As indicated above, smaller growth loss increments may be significant for perennial species. The potential for

cumulative “carry over” effects as well as compounding also must be considered. The accumulation of such “carry-over” effects over time may affect long-term survival and reproduction of individuals and ultimately the abundance of sensitive tree species in forest stands.

c. Crops

As discussed in the 2007 Staff Paper, risk of O₃ exposure and associated monetized benefits were estimated for commodity crops, fruits and vegetables. Similar to the tree seedling analysis, this analysis combined C–R information on crops, crop growing regions and interpolated exposures during each crop growing season. NCLAN crop functions were used for commodity crops. According to USDA National Agricultural Statistical Survey (NASS) data, the 9 commodity crop species (*e.g.*, cotton, field corn, grain sorghum, peanut, soybean, winter wheat, lettuce, kidney bean, potato) included in the 2007 Staff Paper analysis accounted for 69 percent of 2004 principal crop acreage planted in the U.S. in 2004.⁵⁷ The C–R functions for six fruit and vegetable species (tomatoes-processing, grapes, onions, rice, cantaloupes, Valencia oranges) were identified from the California fruit and vegetable analysis from the 1997 review (Abt Associates Inc, 1995). The 2007 Staff Paper noted that fruit and vegetable studies were not part of the NCLAN program and C–R functions were available only in terms of seasonal 7-hour or 12-hour mean index. This index form is considered less effective in predicting plant response for a given change in air quality than the cumulative form used with other crops. Therefore, the fruit and vegetable C–R functions were considered more uncertain than those for commodity crops.

Analyses in the 2007 Staff Paper showed that some of the most important commodity crops such as soybean, winter wheat and cotton had some projected losses under the 2001 base year air quality. Soybean yield losses were projected to be 2–4 percent in parts of Pennsylvania, New Jersey, Maryland and Texas. Winter wheat was projected to have yield losses of 2–6 percent in parts of California.

⁵⁷ Principal crops as defined by the USDA include corn, sorghum, oats, barley, winter wheat, rye, Durum wheat, other spring wheat, rice, soybeans, peanuts, sunflower, cotton, dry edible beans, potatoes, sugar beets, canola, proso millet, hay, tobacco, and sugarcane. Acreage data for the principal crops were taken from the USDA NASS 2005 Acreage Report (<http://usda.mannlib.cornell.edu/reports/nassr/field/pcp-bba/acrg0605.pdf>).

Additionally, cotton was projected to have yield losses of above 6 percent in parts of California, Texas and North Carolina in 2001. The risk assessment estimated that just meeting the then current 0.084 ppm, 8-hour standard would still allow O₃-related yield loss to occur in some commodity crop species and fruit and vegetable species currently grown in the U.S. For example, based on median C-R function response, in counties with the highest O₃ levels, potatoes and cotton had estimated yield losses of 9–15 percent and 5–10 percent, respectively, when O₃ air quality just met the level of a 0.084 ppm, 8-hour standard. Estimated yield improved in these counties when the alternative W126 standard levels were met. The very important soybean crop had generally small yield losses throughout the country under just meeting the then current standard (0–4 percent).

The 2007 Staff Paper also presented estimates of monetized benefits for crops associated with a 0.084 ppm, 8-hour and alternative standards. The Agriculture Simulation Model (AGSIM) (Taylor, 1994; Taylor, 1993) was used to calculate annual average changes in total undiscounted economic surplus for commodity crops and fruits and vegetables when the then current and alternative standard levels were met. Meeting the various alternative standards did show some significant benefits beyond a 0.084 ppm, 8-hour standard. However, the 2007 Staff Paper recognized that the AGSIM economic benefits estimates also incorporate several sources of uncertainty, including: (1) Estimates of economic benefits derived from use of the more uncertain C-R relationships for fruits and vegetables; (2) uncertain assumptions about the treatment and effect of government farm payment programs; and (3) uncertain assumptions about near-term changes in the agriculture sector due to the increased use of crops as biofuels. Although the AGSIM model results provided a relative comparison of agricultural benefits between alternative standards, these uncertainties limited the utility of the absolute numbers.

D. Reconsideration of Secondary Standard

As discussed above at the beginning of section IV, this reconsideration of the secondary O₃ standard set in the 2008 rulemaking focuses on reconsidering certain elements of the standard, the form, averaging times, and level. The general approach for setting a secondary O₃ standard used in the 2008 rulemaking, and in the previous 1997

rulemaking, was to consider two basic policy options: Setting a distinct secondary standard with a biologically relevant form and averaging times, or setting a secondary standard identical to the primary standard. In the 2007 proposed rule, both such options were evaluated, commented on by CASAC and the public, and proposed, as discussed below in sections IV.D.1 and IV.D.2, respectively. In the 2008 final rule, EPA decided to set the secondary standard identical to the revised 8-hour primary standard, as discussed below in section IV.D.3. Section IV.D.4 summarizes comments received from CASAC following the 2008 decision. The Administrator's proposed conclusions based on this reconsideration are presented in section IV.D.5.

1. Considerations Regarding the 2007 Proposed Cumulative Seasonal Standard a. Form

The 2006 Criteria Document and 2007 Staff Paper concluded that the recent vegetation effects literature evaluated in the 2008 rulemaking strengthened and reaffirmed conclusions made in the 1997 review that the use of a cumulative exposure index that differentially weights ambient concentrations is best able to relate ambient exposures to vegetation response at this time (EPA, 2006a, b; see also discussion in IV.B above). The 1997 review focused in particular on two of these cumulative forms, the SUM06 and W126. In the 2008 rulemaking, the 2007 Staff Paper again evaluated these two forms in light of two key pieces of then recent information: Estimates of PRB that were lower than in the 1997 review, and continued lack of evidence within the vegetation effects literature of a biological threshold for vegetation exposures of concern. On the basis of those policy and science-related considerations, the 2007 Staff Paper concluded that the W126 form was more appropriate in the context of the 2008 rulemaking. Specifically, the W126 form, by its incorporation of a sigmoidal weighting scheme, does not create an artificially imposed concentration threshold, gives proportionally more weight to the higher and typically more biologically potent concentrations, and is not significantly influenced by O₃ concentrations within the range of estimated PRB. The Staff Paper further concluded that "it is not appropriate to continue to use an 8-hour averaging time for the secondary standard" and that "the 8-hour average form should be replaced with a cumulative, seasonal,

concentration weighted form" (EPA, 2007b; pg. 8–25).

The CASAC, based on its assessment of the same vegetation effects science, agreed with the 2006 Criteria Document and 2007 Staff Paper and unanimously concluded that it is not appropriate to try to protect vegetation from the known or anticipated adverse effects of ambient O₃ by continuing to promulgate identical primary and secondary standards for O₃. Moreover, the members of the CASAC and a substantial majority of the CASAC O₃ Panel agreed with 2007 Staff Paper conclusions and encouraged EPA to establish an alternative cumulative secondary standard for O₃ and related photochemical oxidants that is distinctly different in averaging time, form and level from the current or potentially revised 8-hour primary standard. The CASAC also stated that "the recommended metric for the secondary ozone standard is the (sigmoidally-weighted) W126 index" (Henderson, 2007).

The EPA agreed with the conclusions drawn in the 2006 Criteria Document, 2007 Staff Paper and by CASAC that the scientific evidence available in the 2008 rulemaking continued to demonstrate the cumulative nature of O₃-induced plant effects and the need to give greater weight to higher concentrations. Thus, EPA concluded that a cumulative exposure index that differentially weights O₃ concentrations represents a reasonable policy choice for a seasonal secondary standard to protect against the effects of O₃ on vegetation. The EPA further agreed with both the 2007 Staff Paper and CASAC that the most appropriate cumulative, concentration-weighted form to consider in the 2008 rulemaking was the sigmoidally weighted W126 form, due to EPA's recognition that there is no evidence in the literature for an exposure threshold that would be appropriate across all O₃-sensitive vegetation and that this form is unlikely to be significantly influenced by O₃ air quality within the range of PRB levels identified in this rulemaking. Thus, in 2007 EPA proposed as one option to replace the then current 0.084 ppm, 8-hour average secondary standard with a standard defined in terms of the cumulative, seasonal W126 form. The EPA also proposed the option of making the secondary identical to the proposed revised primary standard.

b. Averaging Times⁵⁸

The 2007 Staff Paper, in addition to form, also considered what exposure

⁵⁸ While the term "averaging time" is used, for the cumulative, seasonal standard the seasonal and

periods or durations are most relevant for vegetation, which, unlike people, is exposed to ambient air continuously throughout its lifespan. For annual species, this lifespan encompasses a period of only one year or less; while for perennials, lifespans can range from a few years to decades or centuries. However, because O₃ levels are not continuously elevated and plants are not equally sensitive to O₃ over the course of a day, season or lifetime, it becomes necessary to identify periods of exposure that have the most relevance for plant response. The 2007 Staff Paper discussed exposure periods relevant for vegetation in terms of a seasonal window and a diurnal window, and it also discussed defining the standard in terms of an annual index value versus a 3-year average of annual index values. The numbered paragraphs below present the 2007 Staff Paper discussions on these exposure periods, and the annual versus 3-year average index value, followed by a discussion of CASAC views and EPA proposed conclusions.

(1) In considering an appropriate seasonal window, the 2007 Staff Paper recognized that, in general, many annual crops are grown for periods of a few months before being harvested. In contrast, other annual and perennial species may be photosynthetically active longer, and for some species and locations, throughout the entire year. In general, the period of maximum physiological activity and thus, maximum potential O₃ uptake for annual crops, herbaceous species, and deciduous trees and shrubs coincides with some or all of the intra-annual period defined as the O₃ season, which varies on a state-by-state basis. This is because the high temperature and high light conditions that promote the formation of tropospheric O₃ also promote physiological activity in vegetation.

The 2007 Staff Paper noted that the selection of any single seasonal exposure period for a national standard would represent a compromise, given the significant variability in growth patterns and lengths of growing seasons among the wide range of vegetation species occurring within the U.S. that may experience adverse effects associated with O₃ exposures. However, the 2007 Staff Paper further concluded that the consecutive 3-month period within the O₃ season with the highest W126 index value (*e.g.*, maximum 3-month period) would, in most cases,

likely coincide with the period of greatest plant sensitivity on an annual basis. Therefore, the 2007 Staff Paper again concluded, as it did in the 1997 review, that the annual maximum consecutive 3-month period is a reasonable seasonal time period, when combined with a cumulative, concentration weighted form, for protection of sensitive vegetation.

(2) In considering an appropriate diurnal window, the Staff Paper recognized that over the course of the 24-hour diurnal period, plant stomatal conductance varies in response to changes in light level, soil moisture and other environmentally and genetically controlled factors. In general, stomata are most open during daylight hours in order to allow sufficient CO₂ uptake for use in carbohydrate production through the light-driven process of photosynthesis. At most locations, O₃ concentrations are also highest during the daytime, and thus, most likely to coincide with maximum stomatal uptake. It is also known however, that in some species, stomata may remain open sufficiently at night to allow for some nocturnal uptake to occur. In addition, at some rural, high elevation sites, the O₃ concentrations remain relatively flat over the course of the day, often at levels above estimated PRB. At these sites, nighttime W126 values can be of similar magnitude as daytime values, though the significance of these exposures is much less certain. This is because O₃ uptake during daylight hours is known to impair the light-driven process of photosynthesis, which can then lead to impacts on carbohydrate production, plant growth, reproduction (yield) and root function. It is less clear at this time to what extent and by what mechanisms O₃ uptake at night adversely impacts plant function. In addition, many species have not been shown to take up O₃ at night and/or do not occur in areas with elevated nighttime O₃ concentrations.

In reviewing the information on this topic that became available after the 1997 review, the 2007 Staff Paper considered the information compiled in a summary report by Musselman and Minnick (2000). This work reported that some species take up O₃ at night, but that the degree of nocturnal stomatal conductance varies widely between species and its relevance to overall O₃-induced vegetation effects remain unclear. In considering this information, the 2007 Staff Paper concluded that for the vast majority of studied species, daytime exposures represent the majority of diurnal plant O₃ uptake and are responsible for inducing the plant response of most significance to the

health and productivity of the plant (*e.g.*, reduced carbohydrate production). Until additional information is available about the extent to which co-occurrence of sensitive species and elevated nocturnal O₃ exposures exists, and what levels of nighttime uptake are adverse to affected species, the 2007 Staff Paper concluded that this information continues to be preliminary, and does not provide a basis for reaching a different conclusion regarding the diurnal window at this time. The 2007 Staff Paper further noted that additional research is needed to address the degree to which a 12-hour diurnal window may be under-protective in areas where elevated nighttime levels of O₃ co-occur with sensitive species with a high degree of nocturnal stomatal conductance. Thus, as in the 1997 review, the 2007 Staff Paper again concluded that based on the available science, the daytime 12-hour window (8 a.m. to 8 p.m.) is the most appropriate period over which to cumulate diurnal O₃ exposures, specifically those most relevant to plant growth and yield responses.

(3) In considering whether the standard should be defined in terms of an annual index value or a 3-year average of annual index values, the 2007 Staff Paper recognized that though most cumulative seasonal exposure levels of concern for vegetation have been expressed in terms of the annual timeframe, it may be appropriate to consider a 3-year average for purposes of standard stability. However, the 2007 Staff Paper noted that for certain welfare effects of concern (*e.g.*, foliar injury, yield loss for annual crops, growth effects on other annual vegetation and potentially tree seedlings), an annual time frame may be a more appropriate period in which to assess what level would provide the requisite degree of protection, while for other welfare effects (*e.g.*, mature tree biomass loss), a 3-year average may also be appropriate. Thus, the 2007 Staff Paper concluded that it is appropriate to consider both an annual and a 3-year average. Further, the 2007 Staff Paper concluded that should a 3-year average of the 12-hour W126 form be selected, a lower standard level should be considered to reduce the potential of adverse impacts to annual species from a single high O₃ year that could still occur while attaining a standard on average over 3 years.

The CASAC, in considering what seasonal, diurnal, and annual or multiyear time periods are most appropriate when combined with a cumulative, seasonal form to protect vegetation from exposures of concern, agreed that the 2007 Staff Paper

diurnal time periods at issue are those over which exposures during a specified period of time are cumulated, not averaged.

conclusion regarding the 3-month seasonal period and 12-hour daylight window was appropriate, with the distinction that both of these time periods likely represents the minimum time periods of importance. In particular, one O₃ Panel member commented that for some species, additional O₃ exposures of importance were occurring outside the 3-month seasonal and 12-hour diurnal windows. Further, the CASAC concluded that multi-year averaging to promote a "stable" secondary standard is less appropriate for a cumulative, seasonal secondary standard than for a primary standard based on daily maximum 8-hour concentrations. The CASAC further concluded that if multi-year averaging is employed to afford greater stability of the secondary standard, the level of the standard should be revised downward to assure that the desired degree of protection is not exceeded in individual years.

The EPA, in determining which seasonal and diurnal time periods are most appropriate to propose, took into account the 2007 Staff Paper and CASAC views. In being careful to consider what is needed to provide the requisite degree of protection, no more and no less, in 2007 EPA proposed that the 3-month seasonal period and 12-hour daylight period are appropriate. Based on the 2007 Staff Paper conclusions discussed above, EPA was mindful that there is the potential for under-protection with a 12-hour diurnal window in areas with sufficiently elevated nighttime levels of O₃ where sensitive species with a high degree of nocturnal stomatal conductance occur. On the other hand, EPA also recognized that a longer diurnal window (*e.g.*, 24-hour) has the possibility of over-protecting vegetation in areas where nighttime O₃ levels remain relatively high but where no species having significant nocturnal uptake exist. In weighing these considerations, EPA agreed with the 2007 Staff Paper conclusion that until additional information is available about the extent to which this co-occurrence of sensitive species and elevated nocturnal O₃ exposures exists, and what levels of nighttime uptake are adverse to affected species, this information does not provide a basis for reaching a different conclusion at this time. The EPA also considered to what extent the 3-month period within the O₃ season was appropriate, recognizing that many species of vegetation have longer growing seasons. The EPA further proposed that the maximum 3-month period is sufficient and appropriate to

characterize O₃ exposure levels associated with known levels of plant response. Therefore, EPA proposed that the most appropriate exposure periods for a cumulative, seasonal form is the daytime 12-hour window (8 a.m. to 8 p.m.) during the consecutive 3-month period within the O₃ monitoring season with the maximum W126 index value.

The EPA also proposed an annual rather than a multi-year cumulative, seasonal standard. In proposing this option, EPA also believed that it was appropriate to consider the benefits to the public welfare that would accrue from establishing a 3-year average secondary standard, and solicited comment on this alternative. In so doing, EPA also agreed with 2007 Staff Paper and CASAC conclusions that should a 3-year standard be finalized, the level of the standard should be set so as to provide the requisite degree of protection for those vegetation effects judged to be adverse to the public welfare within a single annual period.

c. Level

The 2007 Staff Paper, in identifying a range of levels for a 3-month, 12-hour W126 annual form appropriate to protect the public welfare from adverse impacts to vegetation from O₃ exposures, considered what information from the array of vegetation effects evidence and exposure and risk assessment results was most useful. Regarding the vegetation effects evidence, the 2007 Staff Paper found stronger support than what was available at the time of the 1997 review for an increased level of protection for trees and ecosystems. Specifically, this expanded body of support includes: (1) Additional field based data from free air, gradient and biomonitoring surveys demonstrating adverse levels of O₃-induced above and/or below-ground growth reductions on trees at the seedling, sapling and mature growth stages and incidence of visible foliar injury occurring at biomonitoring sites in the field at ambient levels of exposure; (2) qualitative support from free air (*e.g.*, AspenFACE) and gradient studies on a limited number of tree species for the continued appropriateness of using OTC-derived C-R functions to predict tree seedling response in the field; (3) studies that continue to document below-ground effects on root growth and "carry-over" effects occurring in subsequent years from O₃ exposures; and (4) increased recognition and understanding of the structure and function of ecosystems and the complex linkages through which O₃, and other stressors, acting at the organism and species level can

influence higher levels within the ecosystem hierarchy and disrupt essential ecological attributes critical to the maintenance of ecosystem goods and services important to the public welfare.

Based on the above observations and on the vegetation effects and the results of the exposure and impact assessment summarized above, the 2007 Staff Paper concluded that just meeting the then current standard would still allow adverse levels of tree seedling biomass loss in sensitive commercially and ecologically important tree species in many regions of the country. Seedling risk assessment results showed that some tree seedling species are extremely sensitive (*e.g.*, cottonwood, black cherry and aspen), with annual biomass losses occurring in the field of the same or greater magnitude that that of annual crops. Such information from the tree seedling risk assessment suggests that O₃ levels would need to be substantially reduced to protect sensitive tree seedlings like black cherry from growth and foliar injury effects.

In addition to the currently quantifiable risks to trees from ambient exposures, the 2007 Staff Paper also considered the more subtle impacts of O₃ acting in synergy with other natural and man-made stressors to adversely affect individual plants, populations and whole systems. By disrupting the photosynthetic process, decreasing carbon storage in the roots, increasing early senescence of leaves and affecting water use efficiency in trees, O₃ exposures could potentially disrupt or change the nutrient and water flow of an entire system. Weakened trees can become more susceptible to other environmental stresses such as pest and pathogen outbreaks or harsh weather conditions. Though it is not possible to quantify all the ecological and societal benefits associated with varying levels of alternative secondary standards, the 2007 Staff Paper concluded that this information should be weighed in considering the extent to which a secondary standard should be set so as to provide potential protection against effects that are anticipated to occur.

In addition, the 2007 Staff Paper also recognized that in the 1997 review, EPA took into account the results of a 1996 Consensus Workshop. At this workshop, a group of independent scientists expressed their judgments on what standard form(s) and level(s) would provide vegetation with adequate protection from O₃-related adverse effects. Consensus was reached with respect to selecting appropriate ranges of levels in terms of a cumulative, seasonal 3-month, 12-hr SUM06

standard for a number of vegetation effects endpoints. These ranges are identified below, with the estimated approximate equivalent W126 standard values shown in parentheses. For growth effects to tree seedlings in natural forest stands, a consensus was reached that a SUM06 range of 10 to 15 (W126 range of 7 to 13) ppm-hour would be protective. For growth effects to tree seedlings and saplings in plantations, the consensus SUM06 range was 12 to 16 (W126 range of 9 to 14) ppm-hour. For visible foliar injury to natural ecosystems, the consensus SUM06 range was 8 to 12 (W126 range of 5 to 9) ppm-hour.

Taking these consensus statements into account, EPA stated in the 1997 final rule (62 FR 38856) that "the report lends important support to the view that the current secondary standard is not adequately protective of vegetation * * * [and] * * * foreshadows the direction of future scientific research in this area, the results of which could be important in future reviews of the O₃ secondary standard" (62 FR 38856).

Given the importance EPA put on the consensus report in the 1997 review, the 2007 Staff Paper considered to what extent research published after 1997 provided empirical support for the ranges of levels identified by the experts as protective of different types of O₃-induced effects. With regard to O₃-induced biomass loss in sensitive tree seedlings/saplings growing in natural forest stands, the information discussed in the 2007 Staff Paper, including the evidence from free air and gradient studies, provides additional direct support for the conclusion that the 1996 Consensus Workshop approximate W126 range of 7–13 ppm-hour was an appropriate range to consider in selecting a protective level. With regard to visible foliar injury, the available evidence, including the 2007 Staff Paper analysis of incidence in counties with FIA monitoring sites and air quality data, showed significant levels of county-level visible foliar injury incidence at the W126 level of 13 ppm-hour. However, because this analysis did not address risks of this effect at lower levels of O₃ air quality, and because there is a significant uncertainty in predicting the degree of visible foliar injury symptoms expected for lower levels of O₃ air quality, the evidence provides less certain but qualitative directional support for the 1996 Consensus Workshop range of 5 to 9 ppm-hour to protect against this effect. With regard to O₃-induced effects on plantation trees, there is far less direct information available. Though some forest plantation trees are O₃-sensitive,

the monoculture nature of these stands makes uncertain the degree to which competition for resources might play a role and to what degree the variety of management practices applied would be expected to mitigate the O₃-induced effects. Thus, it is difficult to distinguish a protective range of levels for plantation trees from a range of levels that would be protective of O₃-sensitive tree seedlings and saplings in natural forest stands. Therefore, on the basis of the strength of the evidence available, the 2007 Staff Paper concluded that it was appropriate to consider a range for a 3-month, 12-hour, W126 standard that included the 1996 consensus recommendations for growth effects in tree seedlings in natural forest stands (*i.e.*, 7–13 ppm-hour in terms of a W126 form).

In considering the available information on O₃-related effects on crops in the 2008 rulemaking, the 2007 Staff Paper observed the following regarding the strength of the underlying crop science: (1) Nothing in the recent literature points to a change in the relationship between O₃ exposure and crop response across the range of species and/or cultivars of commodity crops currently grown in the U.S. that could be construed to make less appropriate the use of commodity crop C–R functions developed in the NCLAN program; (2) new field-based studies (*e.g.*, SoyFACE) provide qualitative support in a few limited cases for the appropriateness of using OTC-derived C–R functions to predict crop response in the field; and (3) refinements in the exposure, risk and benefits assessments in this review reduce some of the uncertainties present in the 1997 review. On the basis of these observations, the 2007 Staff Paper concluded that nothing in the newly assessed information called into question the strength of the underlying science upon which EPA based its proposed decision in the 1997 review to select a level of a cumulative, seasonal form associated with protecting 50 percent of crop cases from no more than 10 percent yield loss as providing the requisite degree of protection for commodity crops.

The 2007 Staff Paper then considered whether any additional information is available to inform judgments as to the adversity of various O₃-induced levels of crop yield loss to the public welfare. As noted above, the 2007 Staff Paper observed that agricultural systems are heavily managed, and that in addition to stress from O₃, the annual productivity of agricultural systems is vulnerable to disruption from many other stressors (*e.g.*, weather, insects, disease), whose

impact in any given year can greatly outweigh the direct reduction in annual productivity resulting from elevated O₃ exposures. On the other hand, O₃ can also more subtly impact crop and forage nutritive quality and indirectly exacerbate the severity of the impact from other stressors. Though these latter effects currently cannot be quantified, they should be considered in judging to what extent a level of protection selected to protect commodity crops should be precautionary.

Based on the above considerations, the 2007 Staff Paper concluded that the level of protection (no more than 10% yield or biomass loss in 50% of studied cases) judged requisite in the 1997 review to protect the public welfare from adverse levels of O₃-induced reductions in crop yields and tree seedling biomass loss, as provided by a W126 level of 21 ppm-hour, remains appropriate for consideration as an upper bound of a range of appropriate levels.

Thus, the 2007 Staff Paper concluded, based on all the above considerations, that an appropriate range of 3-month, 12-hour W126 levels was 7 to 21 ppm-hour, recognizing that the level selected is largely a policy judgment as to the requisite level of protection needed. In determining the requisite level of protection for crops and trees, and indirectly, ecosystems, the 2007 Staff Paper recognized that it is appropriate to weigh the importance of the predicted risks of these effects in the overall context of public welfare protection, along with a determination as to the appropriate weight to place on the associated uncertainties and limitations of this information.

The CASAC, in its final letter to the Administrator (Henderson, 2007), agreed with the 2007 Staff Paper recommendations that the lower bound of the range within which a seasonal W126 welfare-based (secondary) O₃ standard should be considered is approximately 7 ppm-hour; however, it did not agree with staff's recommendation that the upper bound of the range for consideration should be as high as 21 ppm-hour. Rather, CASAC recommended that the upper bound of the range considered should be no higher than 15 ppm-hour, which is just above the upper ends of the ranges identified in the 1996 Consensus Workshop as being protective of tree seedlings and saplings grown in natural forest stands and in plantations. The lower end of this range (7 ppm-hour) is the same as the lower end of the range identified in the 1996 Consensus Workshop as protective of tree seedlings

in natural forest stands from growth effects.

In the 2007 proposed rule, taking 2007 Staff Paper and CASAC views into account, EPA proposed a range of levels for a cumulative, seasonal secondary standard as expressed in terms of the maximum 3 month, 12-hour W126 form, in the range of 7 to 21 ppm-hour. This range encompasses the range of levels recommended by CASAC, and also includes a higher level as recommended for consideration in the 2007 Staff Paper. Given the uncertainty in determining the risk attributable to various levels of exposure to O₃, EPA believed, as a public welfare policy judgment, that this was a reasonable range to propose.

2. Considerations Regarding the 2007 Proposed 8-Hour Standard

In the 1997 review, the 1996 Staff Paper included an analysis to compare the degree of overlap between areas that would be expected not to meet the range of alternative 8-hour standards being considered for the primary NAAQS and those expected not to meet the range of values (expressed in terms of the seasonal SUM06 index) of concern for vegetation. This result suggested that improvements in national air quality expected to result from attaining an 8-hour primary standard within the recommended range of levels would also be expected to reduce levels of concern for vegetation in those same areas. In the 1997 final rule, the decision was made, on the basis of both science and policy considerations, to make the secondary identical to the primary standard. It acknowledged, however, that uncertainties remained “as to the extent to which air quality improvements designed to reduce 8-hour average O₃ concentrations averaged over a 3-year period would reduce O₃ exposures measured by a seasonal SUM06 index” (62 FR 38876).

On the basis of that history, the 2007 Staff Paper analyzed the degree of overlap expected between alternative 8-hour and cumulative seasonal secondary standards (as discussed above in section IV.C.1) using then recent air quality. Based on the results, the 2007 Staff Paper concluded that the degree to which the then current 8-hour standard form and level would overlap with areas of concern for vegetation expressed in terms of the 12-hour W126 standard is inconsistent from year to year and would depend greatly on the level of the 12-hour W126 and 8-hour standards selected and the distribution of hourly O₃ concentrations within the annual and/or 3-year average period.

Thus, though the 2007 Staff Paper recognized again that meeting the current or alternative levels of the 8-hour average standard could result in air quality improvements that would potentially benefit vegetation in some areas, it urged caution be used in evaluating the likely vegetation impacts associated with a given level of air quality expressed in terms of the 8-hour average form in the absence of parallel W126 information. This caution was due to the concern that the analysis in the 2007 Staff Paper may not be an accurate reflection of the true situation in non-monitored, rural counties due to the lack of more complete monitor coverage in many rural areas. Further, of the counties that did not show overlap between the two standard forms, most were located in rural/remote high elevation areas which have O₃ air quality patterns that are typically different from those associated with urban and near urban sites at lower elevations. Because the majority of such areas are currently not monitored, it is believed there are likely to be additional areas that have similar air quality distributions that would lead to the same disconnect between forms. Thus, the 2007 Staff Paper concluded that it remained problematic to determine the appropriate level of protection for vegetation using an 8-hour average form.

The CASAC recognized that an important difference between the effects of acute exposures to O₃ on human health and the effects of O₃ exposures on welfare is that vegetation effects are more dependent on the cumulative exposure to, and uptake of, O₃ over the course of the entire growing season (Henderson, 2006c). The CASAC O₃ Panel members were unanimous in concluding the protection of natural terrestrial ecosystems and managed agricultural crops requires a secondary O₃ standard that is substantially different from the primary O₃ standard in averaging time, level, and form (Henderson, 2007).

In considering the appropriateness of proposing a revised secondary standard that would be identical to the proposed primary standard, EPA took into account the approach used by the Agency in the 1997 review, the conclusions of the 2007 Staff Paper, CASAC advice, and the views of public commenters. The EPA first considered the 2007 Staff Paper analysis of the projected degree of overlap between counties with air quality expected to meet various alternative levels of an 8-hour standard and alternative levels of a W126 standard based on monitored air quality data. This analysis showed significant overlap within the proposed

range of the primary 8-hour form and selected levels of the W126 standard form being considered, with the degree of overlap between these two forms depending greatly on the levels selected and the distribution of hourly O₃ concentrations within the annual and/or 3-year average period. On this basis, EPA concluded that a secondary standard set identical to the proposed primary standard would provide a significant degree of additional protection for vegetation as compared to that provided by the current secondary standard. The EPA also recognized that lack of rural monitoring data made uncertain the degree to which the proposed 8-hour or W126 alternatives would be protective, and that there would be the potential for not providing the appropriate degree of protection for vegetation in areas with air quality distributions that result in a high cumulative, seasonal exposure but do not result in high 8-hour average exposures. While this potential for under-protection using an 8-hour standard was clear, the number and size of areas at issue and the degree of risk was hard to determine. On the other hand, EPA also considered at that time that there was a potential risk of over-protection with a cumulative, seasonal standard given the inherent uncertainties associated with moving to a new form for the secondary standard, in particular those associated with predicting exposure and risk patterns based on a limited rural monitoring network.

The EPA also considered the views and recommendations of CASAC, and agreed that a cumulative, seasonal standard is the most biologically relevant way to relate exposure to plant growth response. However, as reflected in the public comments, EPA also recognized that there remained significant uncertainties in determining or quantifying the degree of risk attributable to varying levels of O₃ exposure, the degree of protection that any specific cumulative, seasonal standard would produce, and the associated potential for error in determining the standard that will provide a requisite degree of protection—*i.e.*, sufficient but not more than what is necessary. Given this uncertainty, EPA also believed it was appropriate to consider the degree of protection that would be afforded by a secondary standard that was identical to the then proposed primary standard. Based on its consideration of the full range of views as described above, and in the 2007 proposed rule, EPA proposed as a second option to revise

the secondary standard to be identical in every way to the then proposed primary standard.

3. Basis for 2008 Decision on the Secondary Standard

In the 2008 final rule, EPA noted that deciding on the appropriate secondary standard involved making a choice between two possible alternatives, each with their strengths and weaknesses. The 2008 final rule reported that within the Administration at that time there had been a robust discussion of the same strengths and weaknesses associated with each option that were identified earlier. The process by which EPA reached its final conclusion is described in the final rule (73 FR 16497). The rationale for the 2008 decision presented in the final rule (73 FR 16499–16500) is described below.

In considering the appropriateness of establishing a new standard defined in terms of a cumulative, seasonal form, or revising the then current secondary standard by making it identical to the revised primary standard, EPA took into account the approach used by the Agency in the 1997 review, the conclusions of the 2007 Staff Paper, CASAC advice, and the views of public commenters. In giving consideration to the approach taken in the 1997 review, EPA first considered the 2007 Staff Paper analysis of the projected degree of overlap between counties with air quality expected to meet the revised 8-hour primary standard, set at a level of 0.075 ppm, and alternative levels of a W126 standard based on currently monitored air quality data. This analysis showed significant overlap between the revised 8-hour primary standard and selected levels of the W126 standard form being considered, with the degree of overlap between these alternative standards depending greatly on the W126 level selected and the distribution of hourly O₃ concentrations within the annual and/or 3-year average period.⁵⁹ On this basis, as an initial matter, EPA concluded that a secondary standard set identical to the proposed primary standard would provide a significant degree of additional protection for vegetation as compared to that provided by the then current 0.084 ppm secondary standard. In further considering the significant uncertainties that remain in the available body of evidence of O₃-related vegetation effects and in the exposure and risk analyses conducted for the 2008 rulemaking, and

the difficulty in determining at what point various types of vegetation effects become adverse for sensitive vegetation and ecosystems, EPA focused its consideration on a level for an alternative W126 standard at the upper end of the proposed range (*i.e.*, 21 ppm-hour). The 2007 Staff Paper analysis showed that at that W126 standard level, there would be essentially no counties with air quality that would be expected both to exceed such an alternative W126 standard and to meet the revised 8-hour primary standard—that is, based on this analysis of currently monitored counties, a W126 standard would be unlikely to provide additional protection in any monitored areas beyond that likely to be provided by the revised primary standard.

The EPA also recognized that the general lack of rural monitoring data made uncertain the degree to which the revised 8-hour standard or an alternative W126 standard would be protective in those areas, and that there would be the potential for not providing the appropriate degree of protection for vegetation in areas with air quality distributions that result in a high cumulative, seasonal exposure but do not result in high 8-hour average exposures. While this potential for under-protection using an 8-hour standard was clear, the number and size of areas at issue and the degree of risk was hard to determine. However, EPA concluded at that time that an 8-hour standard would also tend to avoid the potential for providing more protection than is necessary, a risk that EPA concluded would arise from moving to a new form for the secondary standard despite significant uncertainty in determining the degree of risk for any exposure level and the appropriate level of protection, as well as uncertainty in predicting exposure and risk patterns.

The EPA also considered the views and recommendations of CASAC, and agreed that a cumulative, seasonal standard was the most biologically relevant way to relate exposure to plant growth response. However, as reflected in some public comments, EPA also judged that there remained significant uncertainties in determining or quantifying the degree of risk attributable to varying levels of O₃ exposure, the degree of protection that any specific cumulative, seasonal standard would produce, and the associated potential for error in determining the standard that will provide a requisite degree of protection—*i.e.*, sufficient but not more than what is necessary. Given these significant uncertainties, EPA concluded at that time that establishing

a new secondary standard with a cumulative, seasonal form would result in uncertain benefits beyond those afforded by the revised primary standard and therefore may be more than necessary to provide the requisite degree of protection.

Based on its consideration of the views discussed above, EPA judged in the 2008 rulemaking that the appropriate balance to be drawn was to revise the secondary standard to be identical in every way to the revised primary standard. The EPA believed that such a standard would be sufficient to protect public welfare from known or anticipated adverse effects, and did not believe that an alternative cumulative, seasonal standard was needed to provide this degree of protection. The EPA believed that this judgment appropriately considered the requirement for a standard that is neither more nor less stringent than necessary for this purpose.

For the reasons discussed above, and taking into account information and assessments presented in the 2006 Criteria Document and 2007 Staff Paper, the advice and recommendations of the CASAC Panel, and the public comments to date, EPA decided to revise the existing 8-hour secondary standard. Specifically, EPA revised the then current 8-hour average 0.084 ppm secondary standard by making it identical to the revised 8-hour primary standard set at a level of 0.075 ppm.

4. CASAC Views Following 2008 Decision

Following the 2008 decision on the O₃ standards, serious questions were raised as to whether the standards met the requirements of the CAA. In April 2008, the members of the CASAC Ozone Review Panel sent a letter to EPA stating “In our most-recent letters to you on this subject—dated October 2006 and March 2007—* * * the Committee recommended an alternative secondary standard of cumulative form that is substantially different from the primary Ozone NAAQS in averaging time, level and form—specifically, the W126 index within the range of 7 to 15 ppm-hour, accumulated over at least the 12 “daylight” hours and the three maximum ozone months of the summer growing season” (Henderson, 2008). The letter continued: “The CASAC now wishes to convey, by means of this letter, its additional, unsolicited advice with regard to the primary and secondary Ozone NAAQS. In doing so, the participating members of the CASAC Ozone Review Panel are unanimous in strongly urging you or your successor as EPA Administrator to

⁵⁹ Prior to publication of the 2008 final rule, EPA did further analysis of the degree of overlap to extend the 2007 Staff Paper analyses, and that analysis was available in the docket.

ensure that these recommendations be considered during the next review cycle for the Ozone NAAQS that will begin next year" (id.). The letter further stated the following views:

The CASAC was * * * greatly disappointed that you failed to change the form of the secondary standard to make it different from the primary standard. As stated in the preamble to the Final Rule, even in the previous 1996 ozone review, "there was general agreement between the EPA staff, CASAC, and the Administrator, * * * that a cumulative, seasonal form was more biologically relevant than the previous 1-hour and new 8-hour average forms (61 FR 65716)" for the secondary standard. Therefore, in both the previous review and in this review, the Agency staff and its advisors agreed that a change in the form of the secondary standard was scientifically well-justified.

* * * * *

Unfortunately, this scientifically-sound approach of using a cumulative exposure index for welfare effects was not adopted, and the default position of using the primary standard for the secondary standard was once again instituted. Keeping the same form for the secondary Ozone NAAQS as for the primary standard is not supported by current scientific knowledge indicating that different indicator variables are needed to protect vegetation compared to public health. The CASAC was further disappointed that a secondary standard of the W126 form was not considered from within the Committee's previously-recommended range of 7 to 15 ppm-hour. The CASAC sincerely hopes that, in the next round of Ozone NAAQS review, the Agency will be able to support and establish a reasonable and scientifically-defensible cumulative form for the secondary standard. (Henderson, 2008)

5. Administrator's Proposed Conclusions

For the reasons discussed below, the Administrator proposes to set a cumulative seasonal standard expressed as an annual index of the sum of weighted hourly concentrations (*i.e.*, the W126 form), cumulated over 12 hours per day (8 am to 8 pm) during the consecutive 3-month period within the O₃ season with the maximum index value, set at a level within the range of 7 to 15 ppm-hour. This proposed decision takes into account the information and assessments presented in the 2006 Criteria Document and the 2007 Staff Paper and related technical support documents, the advice and recommendations of CASAC both during and following the 2008 rulemaking, and public comments received in conjunction with review of drafts of these documents and on the 2007 proposed rule.

a. Form

As discussed above in section IV.B, the 2006 Criteria Document and 2007

Staff Paper concluded that the recent vegetation effects literature evaluated in the 2008 rulemaking strengthens and reaffirms conclusions made in the 1997 review that the use of a cumulative exposure index that differentially weights ambient concentrations is best able to relate ambient exposures to vegetation response. The 1997 review focused in particular on two of these cumulative forms, the SUM06 and W126 (EPA, 1996). Given that the data available at that time were unable to distinguish between these forms, the EPA, based on the policy consideration of not including O₃ concentrations considered to be within the PRB, estimated at that time to be between 0.03 and 0.05 ppm, concluded that the SUM06 form would be the more appropriate choice for a cumulative, exposure index for a secondary standard.

In the 2008 rulemaking, the 2007 Staff Paper evaluated the continued appropriateness of the SUM06 form in light of new estimates of PRB that were lower than in the 1997 review, and the continued lack of evidence within the vegetation effects literature of a biological threshold for vegetation exposures of concern. On the basis of these policy and science-related considerations, the 2007 Staff Paper concluded that the W126 form was the more appropriate cumulative, concentration-weighted form. Specifically, the W126, by its incorporation of a sigmoidal weighting scheme, does not create an artificially imposed concentration threshold, gives proportionally more weight to the higher and typically more biologically potent concentrations, and is not significantly influenced by O₃ concentrations within the range of estimated PRB.

As discussed above, the CASAC, based on its assessment of the same vegetation effects science, agreed with the 2006 Criteria Document and 2007 Staff Paper and unanimously concluded that protection of vegetation from the known or anticipated adverse effects of ambient O₃ "requires a secondary standard that is substantially different from the primary standard in averaging time, level, and form," *i.e.* not identical to the primary standard for O₃ (Henderson, 2007). Moreover, the members of CASAC and a substantial majority of the other CASAC Panel members agreed with 2007 Staff Paper conclusions and encouraged EPA to establish an alternative cumulative secondary standard for O₃ and related photochemical oxidants that is distinctly different in averaging time, form and level from the then current or

potentially revised 8-hour primary standard (Henderson, 2006c). The CASAC Panel also stated that "the recommended metric for the secondary ozone standard is the (sigmoidally weighted) W126 index" (Henderson, 2007).

In reconsidering the 2008 final rule, the Administrator agrees with the conclusions drawn in the 2006 Criteria Document, 2007 Staff Paper and by CASAC that the scientific evidence available in the 2008 rulemaking continues to demonstrate the cumulative nature of O₃-induced plant effects and the need to give greater weight to higher concentrations. Thus, the Administrator concludes that a cumulative exposure index that differentially weights O₃ concentrations represents a reasonable policy choice for a secondary standard to protect against the effects of O₃ on vegetation. The Administrator further agrees with both the 2007 Staff Paper and CASAC that the most appropriate cumulative, concentration-weighted form to consider is the sigmoidally weighted W126 form.

The Administrator notes that in the 2007 proposed rule, EPA proposed a second option of revising the then current 8-hour average secondary standard by making it identical to the proposed 8-hour primary standard. The 2007 Staff Paper analyzed the degree of overlap expected between alternative 8-hour and cumulative seasonal secondary standards using recent air quality monitoring data. Based on the results, the 2007 Staff Paper concluded that the degree to which the current 8-hour standard form and level would overlap with areas of concern for vegetation expressed in terms of the 12-hour W126 standard is inconsistent from year to year and would depend greatly on the level of the 12-hour W126 and 8-hour standards selected and the distribution of hourly O₃ concentrations within the annual and/or 3-year average period. The 2007 Staff Paper also recognized that meeting the then current or alternative levels of the 8-hour average standard could result in air quality improvements that would potentially benefit vegetation in some areas, but urged caution be used in evaluating the likely vegetation impacts associated with a given level of air quality expressed in terms of the 8-hour average form in the absence of parallel W126 information. This caution was due to the concern that the analysis in the 2007 Staff Paper may not be an accurate reflection of the true situation in non-monitored, rural counties due to the lack of more complete monitor coverage in many rural areas. Further, of

the counties that did not show overlap between the two standard forms, most were located in rural/remote high elevation areas which have O₃ air quality patterns that are typically different from those associated with urban and near urban sites at lower elevations. Because the majority of such areas are currently not monitored, there are likely to be additional areas that have similar air quality distributions that would lead to the same disconnect between forms. Thus, the 2007 Staff Paper concluded that it remains problematic to determine the appropriate level of protection for vegetation using an 8-hour average form.

The Administrator also notes that CASAC recognized that an important difference between the effects of acute exposures to O₃ on human health and the effects of O₃ exposures on welfare is that vegetation effects are more dependent on the cumulative exposure to, and uptake of, O₃ over the course of the entire growing season (Henderson, 2006c). The CASAC O₃ Panel members were unanimous in concluding the protection of natural terrestrial ecosystems and managed agricultural crops requires a secondary O₃ standard that is substantially different from the primary O₃ standard in form, averaging time, and level (Henderson, 2007).

In reaching her proposed decision in this reconsideration of the 2008 final rule, the Administrator has considered the comments received on the 2007 proposed rule regarding revising the secondary standard either to reflect a new, cumulative form or by remaining equal to a revised primary standard. The commenters generally fell into two groups.

One group of commenters, including environmental organizations, strongly supported the proposed option of moving to a cumulative, seasonal standard, generally based on the reasoning explained in the 2007 proposal. Commenters in this group also expressed serious concerns with the other proposed option of setting a secondary O₃ standard in terms of the same form and averaging time (*i.e.*, daily maximum 8-hour average O₃ concentration) as the primary standard. These commenters expressed the view that such a standard would fail to protect public welfare because the maximum daily 8-hour average O₃ concentration failed to adequately characterize harmful O₃ exposures to vegetation. This view was generally based on the observation that there is no consistent relationship in areas across the U.S. between 8-hour peak O₃ concentrations and the longer-term cumulative exposures aggregated over a

growing season that are biologically relevant in characterizing O₃-related effects on sensitive vegetation. Thus, as EPA noted in the 2007 proposed rule, there is a lack of a rational connection between the level of an 8-hour standard and the requisite degree of protection required for a secondary O₃ NAAQS.

Another group of commenters, including industry organizations, agreed that a cumulative form of the standard may better match the underlying data, but expressed the view that remaining uncertainties associated with the vegetation effects evidence and/or EPA's exposure, risk and benefits assessments were so great that the available information did not provide an adequate basis to adopt a standard with a level based on a cumulative, seasonal form. These commenters asserted that because of the substantial uncertainties remaining at the time of the 2008 rulemaking, the benefits of changing to a W126 form were too uncertain to warrant revising the form of the standard at that time.

The Administrator notes that in both the 1997 and the 2008 decisions, EPA recognized that the risk to vegetation from O₃ exposures comes from cumulative exposures over a season or seasons. The CASAC has fully endorsed this view based on the available scientific evidence and assessments, and there is no significant disagreement on this issue by commenters. Thus, it is clear that the purpose of the secondary O₃ NAAQS should be to provide an appropriate degree of protection against cumulative, seasonal exposures to O₃ that are known or anticipated to harm sensitive vegetation or ecosystems. In reconsidering the 2008 final rule, the Administrator recognizes that the issue before the Agency is what form of the standard is most appropriate to perform that function.

Within this framework, the Administrator recognizes that it is clear that a cumulative, seasonal form has a distinct advantage in protecting against cumulative, seasonal exposures. Such a form is specifically designed to measure directly the kind of O₃ exposures that can cause harm to vegetation. In contrast, an 8-hour standard does not measure cumulative, seasonal exposures directly, and can only indirectly afford some degree of protection against such exposures. To the extent that clear relationships exist between 8-hour daily peak O₃ concentrations and cumulative, seasonal exposures, the 8-hour form and averaging time would have the potential to be effective as an indirect surrogate. However, as discussed in the 2007 proposed rule and the 2008 final rule, the evidence shows that there are

known types of O₃ air quality patterns that can lead to high levels of cumulative, seasonal O₃ exposures without the occurrence of high daily 8-hour peak O₃ concentrations. An 8-hour form and averaging time is an indirect way to measure biologically relevant exposure patterns, is poorly correlated with such exposure patterns, and therefore is less likely to identify and protect against the kind of cumulative, seasonal exposure patterns that have been determined to be harmful.

Past arguments or reasons for not moving to a cumulative, seasonal form, with appropriate exposure periods, have not been based on disagreement over the biological relevance of the cumulative, seasonal form, or the recognized disadvantages of an 8-hour standard in measuring and identifying a specified cumulative, seasonal exposure pattern. The reasons for not moving to such a form have been based on concerns over whether EPA has an adequate basis to identify the nature and magnitude of cumulative, seasonal exposure patterns that the standard should be designed to protect against, given the various uncertainties in the evidence and the lack of rural O₃ monitoring data. This most directly translates into a concern over whether EPA has an adequate basis to determine an appropriate level for a cumulative, seasonal secondary standard.

The Administrator has also considered issues associated with selection of the W126 cumulative form, as reflected in the following assertions made by some commenters on the 2007 proposed rule: (1) The W126 form lacks a biological basis, since it is merely a mathematical expression of exposure that has been fit to specific responses in OTC studies, such that its relevance for real world biological responses is unclear; (2) a flux-based model would be a better choice than a cumulative metric because it is an improvement over the many limitations and simplifications associated with the cumulative form; however, there is insufficient data to apply such a model at present; (3) the European experience with cumulative O₃ metrics has been disappointing and now Europeans are working on their second level approach, which will be flux-based; and (4) a second index that reflects the accumulation of peaks at or above 0.10 ppm (called N100) should be added to a W126 index to achieve appropriate protection.

With regard to whether the W126 index lacks a biological basis, the Administrator finds no basis for reaching such a conclusion. As discussed above in section IV.B, the

vegetation effects science is clear that exposures of concern to plants are not based on one discrete 8-hour period but on the repeated occurrence of elevated O₃ levels throughout the plant's growing season. The cumulative nature of the W126 is supported by the basic biological understanding that plants in the U.S. are generally most biologically active during the warm season and are exposed to ambient O₃ throughout this biologically active period. In addition, it has been shown in the scientific literature that all else being equal, plants respond more to higher O₃ concentrations, with no evidence of an exposure threshold for vegetation effects. The W126 sigmoidal weighting function reflects both of these understandings, by not including a threshold below which concentrations are not included, and by differentially weighting concentrations to give greater weight to higher concentrations and less weight to lower ones.

With regard to whether a flux-based model would be a better choice, the 2007 Staff Paper acknowledged that flux models may produce a more accurate calculation of dose to a specific plant species in a specific area. However, dose-response relationships have not been developed for these flux calculations for plants growing in the U.S. Further, flux calculations require large amounts of data for the physiology of each plant species and the local conditions for the growing range of each plant species. These exercises may be useful for limited small-scale risk assessments, but do not provide an appropriate basis for a national standard at this time.

With regard to dissatisfaction with the performance of a particular cumulative index in use in Europe,⁶⁰ and growing interest in development of flux-based models, the 2007 Staff Paper (Appendix 7A) noted that "because of a lack of flux-response data, a cumulative, cutoff concentration based (*e.g.*, AOT40) exposure index will remain in use in Europe for the near future for most crops and for forests and semi-natural herbaceous vegetation (Ashmore *et al.*, 2004a)." Further, like the SUM06 index, the AOT40 index incorporates a threshold below which concentrations are not considered. Though the AOT40 threshold is lower than the threshold value in SUM06, the 2007 Staff Paper concluded that the vegetation effects

information does not provide evidence of an effects threshold that applies to all species. Thus, the Administrator concludes neither of these forms is as biologically relevant as the W126 form.

With regard to consideration of coupling a W126 form with a separate N100 index, there was very little research on the N100 index or a coupled approach to be evaluated in the 2008 rulemaking. The CASAC, after reviewing all the information in the 2006 Criteria Document and the 2007 Staff Paper, did not recommend an additional N100 index for consideration. Therefore, there is no basis at this time to judge the extent to which such a coupled W126–N100 form would be a better choice than the proposed W126 form. Further, the W126 form incorporates a weighting scheme that places greater weight on increasing concentrations and gives every concentration of 0.10 ppm and above an equal weight of 1, which is the highest weight in this sigmoidal weighting function.

In summary, having considered the scientific information and assessment results available in the 2008 rulemaking as discussed above in this proposal notice, as well as the recommendations of the staff and CASAC, and having taken into consideration issues raised in public comments received as part of the 2008 rulemaking, and recognizing the determinations made below in section IV.D.5.c on level, the Administrator concludes that it is appropriate to set the secondary standard using a cumulative, seasonal form. The Administrator also concludes that the W126 form is best suited to reflect the biological impacts of O₃ exposure on vegetation, and that there is adequate certainty in the information available in the 2008 rulemaking to support such a change in form. Thus, the Administrator proposes to set the secondary standard using a cumulative, seasonal W126 form.

b. Averaging Times⁶¹

The Administrator, in addition to reconsidering what form of a secondary standard is most appropriate for protecting vegetation, is also reconsidering what exposure periods (*e.g.*, seasonal window, diurnal window), and what standard index, in terms of an annual index value versus a 3-year average of annual index values, are most appropriate when used in conjunction with the W126 cumulative

seasonal form. Based on the information set forth in the 2007 Staff Paper, as well as CASAC views, as discussed above in section IV.D.1.b, the Administrator has reached conclusions regarding exposure periods, and the annual versus 3-year average index, that have the most biological relevance for plant response, as discussed below.

In considering an appropriate seasonal window, the Administrator notes that the 2007 Staff Paper concluded that the consecutive 3-month period within the O₃ season with the highest W126 index value (*e.g.*, maximum 3-month period) was a reasonable seasonal time period to consider. The Administrator further notes that the 2007 Staff Paper acknowledged that the selection of any single seasonal exposure period for a national standard would necessarily represent a compromise, given the significant variability in growth patterns and lengths of growing seasons among the wide range of sensitive vegetation species occurring within the U.S. However, the Administrator also considered the Staff Paper conclusion that the period of maximum potential plant uptake of O₃ would also likely coincide with the period of highest O₃ occurring within the intra-annual period defined as the O₃ season, since the high temperature and light conditions conducive to O₃ formation are also conducive to plant activity. The Administrator also observes that the CASAC panel was supportive of the Staff Paper views, while recognizing that 3 months likely represented the minimum timeframe appropriate to consider. Therefore, the Administrator concludes, on these bases, that the consecutive 3-month period within the O₃ season with the highest W126 index value (*e.g.*, maximum 3-month period) remains an appropriate seasonal window to propose for the protection of sensitive vegetation.

With regard to consideration of an appropriate diurnal window, the Administrator has taken into account the 2007 Staff Paper conclusion that for the vast majority of studied species, daytime exposures represent the majority of diurnal plant O₃ uptake and are responsible for inducing the plant response of most significance to the health and productivity of the plant (*e.g.*, reduced carbohydrate production). The Administrator is also aware, based on discussions in the 2007 Staff Paper that there are some number of species that show non-negligible amounts of O₃ uptake at night due to incomplete stomatal closure. In reaching her conclusion that the 2007 Staff Paper recommendation of a 12-hour daytime

⁶⁰The AOT40 index used in Europe is a cumulative index that incorporates a threshold at 0.04 ppm (40 ppb). This index is calculated as the area over the threshold (AOT) by subtracting 40 ppb from the value of each hourly concentration above that threshold and then cumulating each hourly difference over a specified window.

⁶¹While the term "averaging time" is used, for the cumulative, seasonal standard the seasonal and diurnal time periods at issue are those over which exposures during a specified period of time are cumulated, not averaged.

window (8 a.m. to 8 p.m.) remains the most appropriate period over which to cumulate diurnal O₃ exposures, specifically those most relevant to plant growth and yield responses, the Administrator places weight on the fact that the CASAC comments were also supportive of this diurnal window, recognizing again that it likely represents a minimum period over which plants can be vulnerable to O₃ uptake. Therefore, the Administrator is again proposing the 12-hour daytime window (8 a.m. to 8 p.m.) as an appropriate diurnal window to protect against O₃-induced plant effects.

Lastly, in considering whether an annual or a 3-year average index is more appropriate, the Administrator notes that in addition to the available scientific evidence regarding plant effects that can be brought to bear, there are also other public welfare considerations that may be appropriate to consider. In taking this view, the Administrator notes that the 2007 Staff Paper recognized that though most cumulative seasonal exposure levels of concern for vegetation have been expressed in terms of the annual timeframe, it may be appropriate to consider a 3-year average for purposes of standard stability. The Administrator has considered that while the 2007 Staff Paper notes that for certain welfare effects of concern (*e.g.*, foliar injury, yield loss for annual crops, growth effects on other annual vegetation and potentially tree seedlings), an annual time frame may be a more appropriate period in which to assess what level would provide the requisite degree of protection, for other welfare effects (*e.g.*, mature tree biomass loss), it also points out that a 3-year average may also be appropriate. The Administrator further observes that in concluding that it was appropriate to consider both an annual and a 3-year average, the 2007 Staff Paper also concluded that should a 3-year average of the 3-month, 12-hour W126 form be selected, a potentially lower level should be considered to reduce the potential of adverse impacts to annual species from a single high O₃ year that could still occur while attaining a standard on average over 3-years. The Administrator also took note that the CASAC Panel, in addressing this issue of annual versus 3-year average concluded that multi-year averaging to promote a “stable” secondary standard is less appropriate for a cumulative, seasonal secondary standard than for a primary standard based on maximum 8-hour concentrations, and further concluded that if multi-year averaging is employed

to increase the stability of the secondary standard, the level of the standard should be revised downward to assure that the desired degree of protection is not exceeded in individual years. The Administrator, in considering the merits of both the annual and 3-year average, and taking into account both the 2007 Staff Paper and CASAC views, concludes that it is important to place more weight on the public welfare benefit in having a stable standard, and that appropriate protection for vegetation can be achieved using a 3-year average form. The Administrator is thus proposing a 3-year average. However, given the uncertain nature of the evidence and potential concerns with using a 3-year average form, the Administrator is proposing to take comment on the appropriateness of the specific seasonal and diurnal exposure periods proposed, as well as the use of a 3-year average, and, as discussed below, the impact that selection of these proposed seasonal and diurnal exposure periods would have, in conjunction with a 3-year average form, on the appropriateness of the proposed range of levels.

c. Level

i. Considerations Regarding 2007 Proposed Range of Levels

The 2007 Staff Paper, in identifying a range of levels for a 3-month, 12-hour (daytime) W126 standard appropriate for the Administrator to consider in protecting the public welfare from known or anticipated adverse effects to vegetation from O₃ exposures, considered what information from the array of vegetation effects evidence and exposure and risk assessment results was most useful. With respect to the vegetation effects evidence, the 2007 Staff Paper found stronger support than what was available at the time of the 1997 review for an increased level of protection for trees and forested ecosystems. Specifically, the expanded body of evidence included: (1) Additional field based data from free air, gradient and biomonitoring surveys demonstrating adverse levels of O₃-induced growth reductions on trees at the seedling, sapling and mature growth stages and incidence of visible foliar injury occurring at biomonitoring sites in the field at ambient levels of exposure; (2) qualitative support from free air (*e.g.*, AspenFACE) and gradient studies on a limited number of tree species for the continued appropriateness of using OTC-derived C-R functions to predict tree seedling response in the field; (3) studies that continued to document below-ground

effects on root growth and “carry-over” effects occurring in subsequent years from O₃ exposures; and (4) increased recognition and understanding of the structure and function of ecosystems and the complex linkages through which O₃, and other stressors, acting at the organism and species level can influence higher levels within the ecosystem hierarchy and disrupt essential ecological attributes critical to the maintenance of ecosystem goods and services important to the public welfare.

Based on the above sources of vegetation effects information and the results of the exposure and risk assessments summarized above, the 2007 Staff Paper concluded that just meeting the then current 0.084 ppm, 8-hour average standard would continue to allow adverse levels of O₃-induced effects to occur in sensitive commercially and ecologically important tree species in many regions of the country. The 2007 Staff Paper further concluded that air quality levels would need to be substantially reduced to protect sensitive tree seedlings, such as black cherry, aspen, and cottonwood, from these growth and foliar injury effects.

In addition to the currently quantifiable risks to trees from ambient exposures, the 2007 Staff Paper also considered the more subtle impacts of O₃ acting in synergy with other natural and man-made stressors to adversely affect individual plants, populations and whole systems. By disrupting the photosynthetic process, decreasing carbon storage in the roots, increasing early senescence of leaves and affecting water use efficiency in trees, O₃ exposures could potentially disrupt or change the nutrient and water flow of an entire system. Weakened trees can become more susceptible to other environmental stresses such as pest and pathogen outbreaks or harsh weather conditions. Though it is not possible to quantify all the ecological and societal benefits associated with varying levels of alternative secondary standards, the 2007 Staff Paper concluded that this information should be weighed in considering the extent to which a secondary standard should be set so as to provide potential protection against effects that are anticipated to occur.

The 2007 Staff Paper also recognized that in the 1997 review, EPA took into account the results of a 1996 Consensus Workshop. At this workshop, a group of independent scientists expressed their judgments on what standard form(s) and level(s) would provide vegetation with adequate protection from O₃-related adverse effects. Consensus was reached

on protective ranges of levels in terms of a cumulative, seasonal 3-month, 12-hr SUM06 standard for a number of vegetation effects endpoints. These ranges are identified below, with the estimated approximate equivalent W126 standard levels shown in parentheses. For growth effects to tree seedlings in natural forest stands, a consensus was reached that a SUM06 range of 10 to 15 (W126 range of 7 to 13) ppm-hour would be protective. For growth effects to tree seedlings and saplings in plantations, the consensus SUM06 range was 12 to 16 (W126 range of 9 to 14) ppm-hour. For visible foliar injury to natural ecosystems, the consensus SUM06 range was 8 to 12 (W126 range of 5 to 9) ppm-hour.

The 2007 Staff Paper then considered to what extent recent research provided empirical support for the ranges of levels identified by the experts as protective of different types of O₃-induced effects. As discussed above in section IV.D.1.c, the 2007 Staff Paper concluded on the basis of the available evidence that it was appropriate to consider a range for a 3-month, 12-hour, W126 standard level that included the 1996 Consensus Workshop recommendations regarding a range of levels protective against O₃-induced growth effects in tree seedlings in natural forest stands (*i.e.*, 7–13 ppm-hour in terms of a W126 form).

In considering the newly available information on O₃-related effects on crops in this review, the 2007 Staff Paper observed the following regarding the strength of the underlying crop science: (1) Nothing in the recent literature points to a change in the relationship between O₃ exposure and crop response across the range of species and/or cultivars of commodity crops currently grown in the U.S. that could be construed to make less appropriate the use of commodity crop C–R functions developed in the NCLAN program; (2) new field-based studies (*e.g.*, SoyFACE) provide qualitative support in a few limited cases for the appropriateness of using OTC-derived C–R functions to predict crop response in the field; and (3) refinements in the exposure, risk and benefits assessments in this review reduce some of the uncertainties present in 1996. On the basis of these observations, the 2007 Staff Paper concluded that nothing in the newly assessed information calls into question the strength of the underlying science upon which EPA based its proposed decision in the last review to select a level of a cumulative, seasonal form associated with protecting 50 percent of crop cases from no more than 10 percent yield loss as providing

the requisite degree of protection for commodity crops.

The 2007 Staff Paper then considered whether any additional information was available to inform judgments as to the adversity of various O₃-induced levels of crop yield loss to the public welfare. As noted above, the 2007 Staff Paper observed that agricultural systems are heavily managed, and that in addition to stress from O₃, the annual productivity of agricultural systems is vulnerable to disruption from many other stressors (*e.g.*, weather, insects, disease), whose impact in any given year can greatly outweigh the direct reduction in annual productivity resulting from elevated O₃ exposures. On the other hand, O₃ can also more subtly impact crop and forage nutritive quality and indirectly exacerbate the severity of the impact from other stressors. Since these latter effects could not be quantified at that time, they could only be considered qualitatively in reaching judgments about an appropriate degree of protection for commodity crops from O₃-related effects.

Based on the above considerations, the 2007 Staff Paper concluded that the level of protection judged requisite in the 1997 review to protect the public welfare from adverse levels of O₃-induced reductions in crop yields and tree seedling biomass loss, as approximately provided by a W126 level of 21 ppm-hour, remained appropriate for consideration as an upper bound of a range of appropriate levels. The 2007 Staff Paper also recognized that a standard set at this level would not protect the most sensitive species or individuals within a species from all potential effects related to O₃ exposures and further, that this level derives from the extensive and quantitative historic and recent crop effects database, as well as current staff exposure and risk analyses (EPA, 2007, pg. 8–22).

In identifying a lower bound for the range of alternative standard levels appropriate for consideration, staff concluded that several lines of evidence pointed to the need for greater protection for tree seedlings, mature trees, and associated forested ecosystems. Staff believed that tree growth was an important endpoint to consider because it is related to other aspects of societal welfare such as sustainable production of timber and related goods, recreation, and carbon (CO₂) sequestration. Impacts on tree growth can also affect ecosystems through shifts in species composition and the loss of genetic diversity due to the loss of O₃ sensitive individuals or species. In selecting an appropriate level

of protection for trees, staff considered the results of the 1996 Consensus Workshop which identified the SUM06 range of 10 to 15 (W126 of 7 to 13) ppm-hour for growth effects to tree seedlings in natural forest stands.

Because staff believed that O₃-related effects on forest tree species are important public welfare effects of concern, it therefore concluded, based on the above, that it was appropriate to include 7 ppm-hour as the lower bound of the recommended range, the lower end of the approximate range recommended by CASAC (Henderson, 2006c) and identified by the 1996 Consensus Workshop participants as protective of forest trees. At this lower end of the range, staff anticipated, based on its analyses of risks of tree seedling biomass loss and mature tree growth reductions and on the basis of the scientific effects literature, that adverse effects of O₃ on forested ecosystems would be substantially reduced. Further, staff anticipated that the lower end of this range would provide increased protection from the more subtle impacts of O₃ acting in synergy with other natural and man-made stressors to adversely affect individual plants, populations and whole systems. Staff also noted that by disrupting the photosynthetic process, decreasing carbon storage in the roots, increasing early senescence of leaves and affecting water use efficiency in trees, O₃ exposure could potentially disrupt or change the nutrient and water flow of an entire system. Such weakened trees can become more susceptible to other environmental stresses such as pest and pathogen outbreaks or harsh weather conditions. While recognizing that it is not possible to quantify all the ecological and societal benefits associated with varying levels of alternative secondary standards, staff believed that this information should be weighed in considering the extent to which a secondary standard should be precautionary in nature in protecting against effects that have not yet been adequately studied and evaluated.

Thus, the 2007 Staff Paper concluded, based on all the above considerations, that an appropriate range of levels, for an annual standard using a 3-month, 12-hour W126 form, for the Administrator to consider was 7 to 21 ppm-hour, recognizing that the level selected is largely a policy judgment as to the requisite level of protection needed. In determining the requisite level of protection for crops and trees, the 2007 Staff Paper recognized that it was appropriate to weigh the importance of the predicted risks of these effects in the overall context of

public welfare protection, along with a determination as to the appropriate weight to place on the associated uncertainties and limitations of this information.

ii. CASAC and Public Comments Prior to 2008 Decision

In considering the evidence described in both the 2006 Criteria Document and 2006 draft Staff Paper, CASAC, in its October 24, 2006 letter to the Administrator, expressed its view regarding the appropriate form and range of levels for the Administrator to consider. The CASAC preferred a seasonal 3-month W126 standard in a range that is the approximate equivalent of the SUM06 at 10 to 20 ppm-hour. Following the 2007 proposal, EPA received additional CASAC and public comments regarding an appropriate range of levels of a W126 form for the Administrator to consider in finalizing a revised secondary NAAQS for O₃. The CASAC, in its final letter to the Administrator (Henderson, 2007), agreed with the 2007 Staff Paper recommendations that the lower bound of the range within which a seasonal W126 secondary O₃ standard should be considered is approximately 7 ppm-hour; however, it did not agree with staff's recommendation that the upper bound of the range should be as high as 21 ppm-hour. Rather, as discussed above in section IV.D.1.c, the CASAC Panel recommended that the upper bound of the range considered should be no higher than a W126 of 15 ppm-hour for an annual standard.

The comments received from the public fell into two groups. One group of commenters supported the CASAC recommended range of 7–15 ppm-hour for a W126 standard. Many of these same commenters further emphasized the lower end of the proposed range as necessary to provide adequate protection for sensitive species. These commenters based their recommendation primarily on four sources of information: (1) Field-based evidence of foliar injury occurring on sensitive species at air quality levels well below that of the current standard; (2) the 1996 Consensus Workshop recommendations for protective levels in terms of cumulative exposures for different vegetation types; (3) CASAC advice and recommendations; and (4) studies published after the close of the 2006 Criteria Document that potentially strengthen the link between species level impacts and ecosystem response.

The other group of commenters did not support revising the current secondary standard. These commenters primarily focused on uncertainties

regarding the sources of information relied upon by the first group of commenters as support for a level within the range of levels recommended by CASAC. These uncertainties included: (1) potential confounders, such as soil moisture, on visible foliar injury and the lack of a clear relationship between visible foliar injury symptoms and other vegetation effects; (2) lack of documentation of the basis for the recommendations from the 1996 Consensus Workshop in selecting a range of levels, indicating that these recommendations should be used with great caution; (3) failure of CASAC and EPA to take into account the monitor height measurement gradient when making their recommendations concerning the level of the secondary standard; and (4) inability to quantitatively estimate ecosystem effects of O₃ or to extrapolate meaningfully from effects on individual plants to ecosystem effects due to inadequate data.

iii. Conclusions on Level

The Administrator is proposing to set a cumulative, seasonal standard expressed in terms of the maximum 3-month, 12-hour W126 form, in the range of 7 to 15 ppm-hour. In reaching this proposed decision about an appropriate range of levels for the secondary standard, the Administrator has considered the following: the evidence described in the 2006 Criteria Document and the 2007 Staff Paper; the results of the vegetation exposure and risk assessments discussed above and in the 2007 Staff Paper, giving weight to the assessments as judged appropriate; the CASAC Panel's advice and recommendations in the CASAC's letters to the Administrator; EPA staff recommendations; and public comments received during the development of these documents, either in connection with CASAC meetings or separately. In considering what range of levels of a cumulative 3-month standard to propose, the Administrator notes that this choice requires judgment as to what standard will protect the public welfare from any known or anticipated adverse effects. This choice must be based on an interpretation of the evidence and other information, such as the exposure and risk assessments, that neither overstates nor understates the strength and limitations of the evidence and information nor the appropriate inferences to be drawn. In taking all of the above into consideration, the Administrator also notes that there is no bright line clearly directing the choice of level for any of the effects of concern, and the choice of what is appropriate is

clearly a public welfare policy judgment entrusted to the Administrator.

In particular, the Administrator has given careful consideration to the following: (1) The nature and degree of effects of O₃ to the public welfare, including what constitutes an adverse effect; (2) the strengths and limitations of the evidence that is available regarding known or anticipated adverse effects from cumulative, seasonal exposures, and its usefulness in informing selection of a proposed range; and (3) CASAC's views regarding the strength of the evidence and its adequacy to inform a range of levels. Each of these topics is discussed in turn below.

In determining the nature and degree of effects of O₃ on the public welfare, the Administrator recognizes that the significance to the public welfare of O₃-induced effects on sensitive vegetation growing within the U.S. can vary, depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located. Any given O₃-related effect on vegetation and ecosystems (e.g., biomass loss, foliar injury), therefore, may be judged to have a different degree of impact on the public depending, for example, on whether that effect occurs in a Class I area, a city park, or commercial cropland. In her judgment, it is appropriate that this variation in the significance of O₃-related vegetation effects should be taken into consideration in judging the level of ambient O₃ that is requisite to protect the public welfare from any known or anticipated adverse effects. In this regard, the Administrator agrees with the definition of adversity as described above in section IV.A.3 and in the 2008 rulemaking. As a result, the Administrator concludes that of those known and anticipated O₃-related vegetation and ecosystem effects identified and discussed in this reconsideration, the highest priority and significance should be given to those that occur on sensitive species that are known to or are likely to occur in federally protected areas such as Class I areas⁶² or on lands set aside by States, Tribes and public interest groups to provide similar benefits to the public

⁶² For example, the level of protection granted by Congress under the Wilderness Act of 1964 for designated "wilderness areas" requires that these areas "shall be administered for the use and enjoyment of the American people in such manner as will leave them unimpaired for future use as wilderness, and so as to provide for the protection of these areas, the preservation of their wilderness character" (The Wilderness Act, 1964).

welfare, for residents on those lands, as well as visitors to those areas.

Likewise, the Administrator also notes that the same known or anticipated O₃-induced effects, occurring in other areas may call for less protection. For example, the maintenance of adequate agricultural crop yields is extremely important to the public welfare and is currently achieved through the application of intensive management practices, including in some cases, genetic engineering. These management practices, in conjunction with market forces and government programs, assure an appropriate balance is reached between costs of production and market availability. Thus, while research on agricultural crop species remains useful in illuminating mechanisms of action and physiological processes, information from this sector on O₃-induced effects is considered less useful in informing judgments on what level(s) would be sufficient but not more than necessary to protect the public welfare. With respect to commercial production of commodities, the Administrator notes that judgments about the extent to which O₃-related effects on commercially managed vegetation are adverse from a public welfare perspective are particularly difficult to reach, given that what is known about the relationship between O₃ exposures and agricultural crop yield response derives largely from data generated almost 20 years ago. The Administrator recognizes that there is substantial uncertainty at this time as to whether these data remain relevant to the majority of species and cultivars of crops being grown in the field today. In addition, the extensive management of such vegetation may to some degree mitigate potential O₃-related effects. The management practices used on these lands are highly variable and are designed to achieve optimal yields, taking into consideration various environmental conditions. Thus, the Administrator concludes there is no need for such additional protection for agricultural crops through the NAAQS.

The Administrator also recognizes that O₃-related effects on sensitive vegetation can occur in other areas that have not been afforded special Federal protections, ranging from effects on vegetation growing in residential or commercial settings, such as ornamentals used in urban/suburban landscaping, to vegetation grown in land use categories that are heavily managed for commercial production of commodities such as timber. For vegetation used for residential or commercial ornamental purposes, such

as urban/suburban landscaping, the Administrator believes that there is not adequate information at this time to establish a secondary standard based specifically on impairment of urban/suburban landscaping and other uses of ornamental vegetation, but notes that a secondary standard revised to provide protection for sensitive natural vegetation and ecosystems would likely also provide some degree of protection for such ornamental vegetation.

Based on the above, the Administrator finds that the types of information most useful in informing the selection of an appropriate range of protective levels is appropriately focused on information regarding exposures and responses of sensitive trees and other native species known or anticipated to occur in protected areas such as Class I areas or on lands set aside by States, Tribes and public interest groups to provide similar benefits to the public welfare, for residents on those lands, as well as visitors to those areas.

With regard to the available evidence, the Administrator finds the coherence and strength of the weight of evidence from the large body of available literature compelling. This evidence addresses a broad array of O₃-induced effects on a variety of tree species across a range of growth stages (*i.e.*, seedlings, saplings and mature trees) using diverse field-based (*e.g.* free air, gradient and ambient) and OTC exposure methods. It demonstrates that significant numbers of forest tree species are potentially experiencing O₃-induced stress under levels of ambient air quality, both at and below the level of the 1997 standard.

In particular, the Administrator notes the evidence from recent field-based studies and a gradient study of eastern cottonwood saplings (Gregg *et al.*, 2003). She observes that this study found that cottonwood saplings grown in urban New York City grew faster than saplings grown in downwind rural areas where cumulative O₃ exposures were higher, and the difference in biomass production between the urban site with the lowest cumulative exposure and the rural site with the highest cumulative exposure is dramatic (Figure 7–17 in the 2007 Staff Paper). The Administrator further notes that cottonwood is one of the most sensitive tree species studied to date and it is also important both from an ecological and public welfare perspective, as discussed above in section IV.A.2.b and in the 2007 Staff Paper.

The Administrator also notes the evidence related to the O₃-induced effect of visible foliar injury. The Administrator observes that the visible foliar injury database created from the

ambient field-based monitoring network managed by the United States Forest Service (USFS) Forest Inventory and Analysis (FIA) Program has continued to expand since the conclusion of the 1997 review. In utilizing this dataset, EPA staff collaborated with FIA staff to compare the incidence of visible foliar injury at different levels of air quality by county throughout the U.S. in counties with FIA monitoring sites. In considering the results of this analysis, depicted in Table 7–4 of the 2007 Staff Paper, the Administrator notes that for the 2001–2004 period, the percent of counties with documented foliar injury at a level approximately equivalent to the W126 of 21 ppm-hour, was 26 to 49 percent, while at the lower level approximately equivalent to a W126 of 13 ppm-hour, incidence values ranged from 12 to 35 percent. The Administrator believes it likely that some sensitive species occurring in specially protected areas would also exhibit visible foliar injury symptoms to a similar degree at these exposure levels. She further notes that while direct links between O₃ induced visible foliar injury symptoms and other adverse effects (*e.g.*, biomass loss) are not always found, visible foliar injury in itself is considered by the National Park Service (NPS) to affect adversely air quality related values (AQRV) in Class I areas.

The Administrator places significant weight on the judgments of CASAC. In so doing, the Administrator has carefully considered its stated views and the basis for the range of levels the CASAC O₃ Panel recommended. In its 2007 letter to the Administrator, the CASAC O₃ Panel agreed with EPA staff recommendations that the lower bound of the range within which a seasonal W126 O₃ standard should be considered is approximately 7 ppm-hour. However, “it does not agree with Staff’s recommendations that the upper bound of the range should be as high as 21 ppm-hour. Rather, the Panel recommends that the upper bound of the range considered should be no higher than 15 ppm-hour, which the Panel estimates is approximately equivalent to a seasonal 12-hour SUM06 level of 20 ppm-hour” (Henderson, 2007). The Administrator notes that CASAC views concerning an appropriate range of levels for the Administrator to consider were presented after CASAC had considered the entire body of evidence presented in both the 2006 Criteria Document and 2007 Staff Paper, and are generally consistent with the 1996 Consensus Workshop recommendations.

In considering the issues raised by commenters on the 2007 proposed rule, the Administrator noted that many public commenters supported the range of levels recommended by CASAC. The Administrator also considered the views expressed by the NPS as to what range of levels it identified as useful in helping it achieve its mandate to protect AQRVs in national parks and wilderness areas and to provide a level of protection for its resources in keeping with the Congressional mandate set forth in The Wilderness Act of 1964. In so doing, the Administrator notes that the NPS supported the range recommended by CASAC, while emphasizing that the lower end of the range was more appropriate. The NPS notes that though some visible foliar injury would still be expected to occur above the lower end of the CASAC recommended range (*i.e.* 7 ppm-hour), the potential for growth impacts at that level would be very low. It further notes that most of these parks contain aspen, black cherry, or ponderosa pine, all sensitive species predicted to have significant growth effects at current W126 levels.

The Administrator also considered those comments that highlighted sources of uncertainty in the evidence and risk assessments (summarized above in section IV.D.5.c.ii) to inform her judgments on how much weight to place on these associated uncertainties, as discussed below.

With regard to the issue of possible confounders of foliar injury information, the Administrator recognizes that visible foliar injury, like other O₃-induced plant effects, is moderated by environmental factors other than O₃ exposure. However, the Administrator also notes that the O₃-related visible foliar injury effect persisted across a four year period (2001–2004), despite year-to-year variability in meteorology and other environmental factors (see Table 7–4 in the 2007 Staff Paper). She also notes that approximately 26 to 49 percent of counties had visible foliar injury incidence at the approximate W126 level of 21 ppm-hour, while at a W126 level of 13 ppm-hour, this range of percentages dropped to approximately 12 to 23 percent. In an area such as a national park, where visitors come in part for the aesthetic quality of the landscape, the Administrator recognizes that visible foliar injury incidence is an important welfare effect which should be considered in determining an appropriately protective standard level.

With regard to the issues of what weight to place on the recommendations from the 1996 Consensus Workshop in

selecting a range of levels, as the 1997 Workshop Report did not clearly document the basis for its recommendations, the Administrator recognizes that the absence of such documentation does call for care in placing weight on such recommendations. However, the Administrator notes that the workshop participants were asked to review both the 1996 O₃ Criteria Document and Staff Paper, representing the most up to date compilation of the state of the science available at that time, in order to ensure that their expert judgments made were also informed by the latest science. She also notes that another group of experts, the CASAC O₃ Panel, reached a similar consensus based upon an expanded body of scientific evidence. In addition, the 2007 Staff Paper evaluated the same recommendations in the context of subsequent empirical evidence, and reached similar views, with the exception of the upper end of the recommended range, which in the 2007 Staff Paper was based on effects on commercial crops that had been considered in the 1997 review. While it would always be more useful to have documentation of the reasoning and basis for an expert's advice, in this case the Administrator judges that the 1996 Consensus Workshop recommendations should be given substantial weight.

With regard to other issues raised by some commenters related to uncertainties in the technical evidence and analyses, the Administrator notes that such issues had been addressed in the 2007 Staff Paper that reflected CASAC's advice on such issues. For example, while the Administrator recognizes that uncertainty remains as to what level of annual tree seedling biomass loss when compounded over multiple years should be judged adverse to the public welfare, she believes that the potential for such anticipated effects should be considered in judging to what degree a standard should be precautionary.

In considering all of the issues discussed above, the Administrator has decided to propose a range of 7–15 ppm-hour. In selecting as an upper bound a level of 15 ppm-hour, the Administrator notes that this level was specifically supported by the CASAC O₃ Panel and is just above the range identified in the 1996 Consensus Workshop report as needed to provide adequate protection for trees growing in natural areas. In addition, the NPS, along with many public commenters, were in support of the CASAC range, including the upper bound of 15 ppm-hour, and indicated that lower values within this range would be more

protective for sensitive trees in protected areas from biomass loss and visible foliar injury symptoms.

While the upper end of this range is lower than the upper end of 21 ppm-hour recommended in the 2007 Staff Paper, this upper level of 21 ppm-hour was originally put forward in the 1997 review in terms of a SUM06 of 25 ppm-hour (W126 of 21 ppm-hour) and was justified on the basis that it was predicted to allow up to 10% biomass loss annually in 50% of studied commercial crops and tree seedling species. Recognizing the significant uncertainties that are associated with evaluating effects on commercial crops from a public welfare perspective, the Administrator now concludes that commercial crop data are no longer useful for setting the upper level of the range for proposal.

With regard to her selection of a proposed range, the Administrator has considered that the direction from Congress to provide a high degree of protection in Class I areas creates a clearer target for gauging what types and magnitudes of effects would be known or anticipated to affect the intended use of these and other similarly protected areas, that would thus be considered adverse to the public welfare. Such similar areas include lands set aside by States, Tribes and public interest groups to provide similar benefits to the public welfare, for residents on those lands, as well as visitors to those areas. The Administrator also believes that in order to preserve wilderness areas in an unimpaired state for future generations, she must consider a level that affords substantial protection from known adverse O₃-related effects of biomass loss and foliar injury on sensitive tree species, as well as a level that takes into account potential "anticipated" adverse O₃-related effects, including effects that result in continued impairment in the year following O₃ exposure (*i.e.*, carry-over effects), below ground impacts, ecosystem level impacts, and reduced CO₂ sequestration.

While the Administrator acknowledges that growth effects and visible foliar injury can still occur in sensitive species at levels below the upper bound of the proposed range, the Administrator also recognizes that some significant uncertainties remain regarding the risk of these effects, as discussed above. For example, the Administrator concludes that remaining uncertainties make it difficult to judge the point at which visible foliar injury becomes adverse to the public welfare in various types of specially protected areas. Uncertainties associated with monitoring ambient exposures must be

considered in evaluating the strength of predictions regarding the degree of tree seedling growth impairment estimated to occur at varying ambient exposures. These uncertainties add to the challenge of judging which exposure levels are expected to be associated with levels of tree seedling growth effects considered adverse to public welfare. The Administrator believes that it is important to consider these uncertainties, and the weight to place on such uncertainties, in selecting a range of standard levels to propose. Establishing 15 ppm-hour as the upper end of the proposed range reflects her judgment regarding the appropriate weight to place on these uncertainties in determining the degree of protection that is warranted for known and anticipated adverse effects.

With regard to her selection of a lower bound for the proposed range, the Administrator believes that if weight is placed on taking a more precautionary approach, recognizing that the real world impacts on trees and ecosystems could, in some cases, be greater than predicted, then the lower end of the range of 7 ppm-hour could be warranted. There is clear evidence that higher cumulative exposures can occur in rural areas downwind of urban areas and potentially in Class I areas. Unmonitored high elevation sites would also likely have higher cumulative exposures than lower elevation sites that are currently monitored. All of these considerations lead the Administrator to propose 7 ppm-hour as the low end of the proposed range.

As discussed above in section IV.D.5.a, the main opposition to changing to a secondary standard with a cumulative, seasonal form has been the view that EPA does not have an adequate basis to identify the kinds and types of cumulative, seasonal exposure patterns that the standard should be designed to protect against, given the various uncertainties in the evidence, and whether EPA has an adequate basis to determine an appropriate level for a cumulative, seasonal secondary standard. While EPA agreed with this position in the 1997 review, the Administrator believes that the evidence before her appropriately supports a secondary standard that is distinctly different in form and averaging time from the 8-hour primary standard, and that such a standard is necessary to provide sufficient protection from cumulative, seasonal exposures to O₃.

While a different conclusion on this issue was reached in the 1997 review, the current conclusion that an exposure index that is cumulative and seasonal in nature, and therefore that setting a

standard based on such a form is necessary and appropriate, is based on information newly available in the 2008 rulemaking, which strengthens the information available in the 1997 review and reduces remaining uncertainties.

Such newly available information includes quantitative information for a broader array of vegetation effects (extending to sapling and mature tree growth stages) obtained using a more diverse set of field-based research study designs and improved analytical methods for assessing O₃-related exposures and risks as discussed above in sections IV.A–C.

These newly available studies also provide important support to the quantitative estimates of impaired tree growth based on earlier studies available in the 1997 review and address one of the key data gaps cited in the 1997 review. Additional qualitative information is also available regarding improved understanding of linkages between stress-related effects of O₃ exposures at the species level and those at higher levels within ecosystems. Finally, this information includes the use of new analytical methods, including a new multi-pollutant, multi-scale air quality model used to characterize exposures of O₃-sensitive tree and crop species further address uncertainties in the assessments done in the 1997 review. In total, this newly available information increases the Administrator's confidence in important aspects of this rulemaking.

The decision in 2008 to set the secondary O₃ standard identical to the 8-hour primary standard largely mirrored the decision in 1997, but failed to account for this significant increase in the body of knowledge available to support the 2008 rulemaking. This body of knowledge, while continuing to reflect significant uncertainties, provides an appropriate basis for determining a level of a cumulative, seasonal standard that, in the judgment of the Administrator, provides sufficient but not more than necessary protection from cumulative, seasonal exposures to O₃. This is clearly so when compared to a standard that uses an indirect form that is not biologically relevant, an 8-hour average standard aimed at peak daily exposures. This judgment is fully consistent with the advice provided by CASAC.

After carefully taking the above considerations into account, and giving significant weight to the views of CASAC, the Administrator has decided to propose a range of levels of 7–15 ppm-hour for a cumulative, seasonal secondary O₃ standard expressed as an index of the annual sum of weighted

hourly concentrations (*i.e.*, the W126 form), cumulated over 12 hours per day during the consecutive 3-month period within the O₃ season with the maximum index value, averaged over three years. In the Administrator's judgment, based on the information available in the 2008 rulemaking, a standard could be set within this range that would be requisite to protect public welfare from known or anticipated adverse effects to O₃-sensitive vegetation and ecosystems. In the Administrator's judgment, a standard set at a level below the lower end of the range is not now supported by the weight of evidence and would not give sufficient weight to the important uncertainties and limitations inherent in the available scientific evidence and in the quantitative assessments conducted for the 2008 rulemaking. A standard set at a level above the upper end of the range is also not now supported by the weight of evidence and would not give sufficient weight to the credible inferences that the Agency has drawn from the scientific evidence nor to the quantitative assessments conducted for the 2008 rulemaking. The Administrator judges that the appropriate balance to be drawn, based on the entire body of evidence and information available in the 2008 rulemaking, is a range between 7 and 15 ppm-hour. On balance, the Administrator believes that a standard could be set within this range that would be sufficient but not more than necessary to protect public welfare from known or anticipated adverse effects due to O₃.

In reaching this proposed decision, as discussed above, the Administrator has focused on the nature of the benefits associated with setting a distinct secondary standard with a cumulative, seasonal form relative to a standard with a peak daily 8-hour average form, as well as on assessments that quantify the degree of protection likely to be afforded by such standards. In so doing, the Administrator has acknowledged limitations in quantifying the expected benefits associated with the proposed cumulative seasonal standard relative to the secondary standard set in 2008. Having considered the public comments received on the 2007 proposed rule in reaching this proposed decision, the Administrator is interested in again receiving public comment on the benefits to public welfare associated with the proposed cumulative seasonal standard set at specific levels within the proposed range relative to the standard set in 2008.

E. Proposed Decision on the Secondary O₃ Standard

For the reasons discussed above, and taking into account information and assessments presented in the 2006 Criteria Document and 2007 Staff Paper, the advice and recommendations of CASAC, and the public comments received in conjunction with the 2008 rulemaking, the Administrator has decided to propose to set a new cumulative, seasonal secondary O₃ standard with a form expressed as an index of the annual sum of weighted hourly concentrations (*i.e.*, the W126 form), cumulated over 12 hours per day (8 a.m. to 8 p.m.) during the consecutive 3-month period within the O₃ season with the maximum index value, averaged over three years, set within a range of 7 to 15 ppm-hour. The Administrator solicits comment on the weight that is appropriately placed on the various types of evidence and analyses upon which this proposed standard is based, and on the appropriate weight to be placed on the uncertainties in this information, as well as on the benefits to public welfare associated with the proposed standard relative to the benefits associated with the standard set in 2008.

Data handling conventions for the proposed new secondary O₃ standard are specified in the proposed addition of a new section to 40 CFR 50 Appendix P, as discussed in section V below. Issues related to monitoring requirements for the proposed new secondary O₃ standard are discussed below in section VI.

V. Interpretation of the NAAQS for O₃ and Proposed Revisions to the Exceptional Events Rule

Appendix P to 40 CFR part 50, Interpretation of the Primary and Secondary National Ambient Air Quality Standards for Ozone, addresses data completeness requirements, data reporting, handling, and rounding conventions, and example calculations. The current Appendix P explains the computations necessary for determining when the current identical primary and secondary standards are met. The EPA is proposing to revise Appendix P to reflect the proposed revisions to the primary and secondary O₃ NAAQS discussed above and to make other changes described below.

As discussed below, the proposed revisions to Appendix P include the following: The addition of data interpretation procedures applicable to the proposed cumulative, seasonal secondary NAAQS (see section V.B); clarification of certain language in the

current provisions applicable to the primary NAAQS to reduce potential confusion (section V.C); revisions to the provisions regarding the use of incomplete data sets for purposes of the primary NAAQS and the data completeness requirements across three years (sections V.D and V.E); the addition of a provision providing the Administrator discretion to use incomplete data as if it were complete, for the purpose of the primary NAAQS (section V.F); a change from truncation to rounding of multi-hour and multi-year average O₃ concentrations for the purposes of the primary standard (section V.G); and the addition of provisions addressing data to be used in making comparisons to the NAAQS (section V.H). The proposed revisions also include changes in organization for greater clarity and consistency with other data interpretation appendices to 40 CFR part 50, which are not further described in this preamble.

The EPA is also proposing changes to the O₃-specific deadlines, in 40 CFR 50.14, by which states must flag ambient air data that they believe have been affected by exceptional events and submit initial descriptions of those events, and the deadlines by which states must submit detailed justifications to support the exclusion of that data from EPA determinations of attainment or nonattainment with the NAAQS. The O₃-specific deadlines in the current 40 CFR 50.14 would not be appropriate given the anticipated schedule for the designations of areas under the proposed revised O₃ NAAQS.

A. Background

The purpose of a data interpretation appendix in general is to provide the practical details on how to make a comparison between multi-day and possibly multi-monitor ambient air concentration data and the level of the NAAQS, so that determinations of compliance and violation are as objective as possible. Data interpretation guidelines also provide criteria for determining whether there are sufficient data to make a NAAQS level comparison at all. Appendix P was promulgated in March 2008 along with the most recent revisions to the primary and secondary O₃ NAAQS. It is very similar to Appendix I, Interpretation of the 8-Hour Primary and Secondary National Ambient Air Quality Standards for Ozone, which was adopted in 1997 when the O₃ NAAQS were first revised to have an 8-hour averaging period rather than the earlier 1-hour averaging period, along with other changes in form and level. The only substantive difference between Appendix I and the

current version of Appendix P is that Appendix P contains truncation procedures consistent with the additional decimal digit used to express the level of the 2008 NAAQS in parts per million (0.075 ppm) compared to the 1997 NAAQS (0.08 ppm). In July 2007, EPA had also proposed to include in Appendix P data interpretation procedures for the proposed cumulative, seasonal secondary O₃ NAAQS, but these procedures were not finalized given that the final secondary NAAQS was identical in all respects to the final primary NAAQS.

An exceptional event is defined in 40 CFR 50.1 as an event that affects air quality, is not reasonably controllable or preventable, is an event caused by human activity that is unlikely to recur at a particular location or a natural event, and is determined by the Administrator in accordance with 40 CFR 50.14 to be an exceptional event. Air quality data that are determined to have been affected by an exceptional event under the procedural steps and substantive criteria specified in section 50.14 may be excluded from consideration when EPA makes a determination that an area is meeting or violating the associated NAAQS. The key procedural deadlines in section 50.14 are that a state must notify EPA that data have been affected by an event, *i.e.*, “flag” the data in the Air Quality Systems (AQS) database, and provide an initial description of the event by July 1 of the year after the data are collected, and that the State must submit the full justification for exclusion within 3 years after the quarter in which the data were collected. However, if a regulatory decision based on the data, for example a designation action, is anticipated, the schedule is shortened and all information must be submitted to EPA no later than a year before the decision is to be made. This generic schedule presents problems when a NAAQS has been recently revised, as discussed in section V.I below. On May 15, 2009, EPA finalized a set of O₃-specific deadlines that corrected these problems at the time with respect to the 2008 NAAQS revisions (74 FR 23307). However, because of the anticipated effect of the current reconsideration on the schedule for O₃ designations, the schedule problems will resurface unless the deadlines are adjusted again.

B. Interpretation of the Secondary O₃ Standard

The EPA is proposing data interpretation procedures for the proposed secondary O₃ NAAQS, which is defined in terms of a specific cumulative, seasonal form, commonly

referred to as the W126 form, as described above in section IV. The proposed new section 4 of Appendix P on data interpretation for the secondary standard contains the following main features.

The "design value" for the secondary standard, the statistic for a monitoring site which would be compared to the level of the secondary standard to determine if the site meets the standard, would be the average of the annual maximum values of the three-month index value from three calendar years.

The new section would provide clear directions and examples for the calculation of the daily index value, the monthly cumulative index value, the annual maximum index value for a year, and the design value itself.

Only the data from the required O₃ monitoring season would be examined to determine the annual maximum index value; any additional period of monitoring undertaken voluntarily by a state would not be considered. The EPA believes that because of the recently proposed extension of the required monitoring seasons in many states (74 FR 34525, July 16, 2009), as discussed below in section VI, such a period of voluntary monitoring would be unlikely to have such high index values as to affect the annual maximum index value. Moreover, the proposed required monitoring season may encompass the most active growing season in many areas. The EPA invites comment on whether instead the entire actual O₃ monitoring period should be considered, to eliminate any possibility that the highest cumulative index value that can be determined with available data might be missed.

For each month in a three-month period, O₃ data would have to be available for at least 75 percent of daylight hours (defined for this purpose as 8 a.m.–7:59 p.m. LST). If data are available for at least 75 percent but fewer than 100 percent of these daylight hours in a month, the cumulative index value calculated from the available daylight hours in the month would be increased to compensate for the missing hours, based on an assumption that the missing hours would have the same distribution of O₃ concentrations as the available hours. A substitution test is also proposed, by which months in which fewer than 75 percent of daylight hours have O₃ concentration data might also be useable for calculating a valid cumulative index value. Such months would be used if the available O₃ concentrations are so high that even substituting low concentration values for enough missing data to meet the 75 percent requirement would result in a

design value greater than the level of the standard. The low value that would be substituted would be the lowest 1-hour O₃ concentration observed at the monitoring site during daylight hours during the required O₃ monitoring season, in that calendar year, or one-half the method detection limit (MDL) of the ozone instrument, whichever is higher.⁶³

The EPA notes that while this proposed approach to identifying the substitution value for the secondary standard is technically appropriate, it would necessitate data processing efforts during implementation that might be avoidable via some other approach that is also technically reasonable. We therefore invite comment on such alternative approaches, and we may adopt another approach in the final rule. For example, for simplicity the substituted 1-hour O₃ concentration value could instead simply always be zero or one-half the MDL of the O₃ instrument, noting that because of the sigmoidal weighting factor the exact magnitude of the low substitution value may typically make very little difference to the annual index value. Also, using the previous calendar year as the source of the substitution value instead of the current calendar year would have the advantage of allowing all parties to know early in each year what the substitution value will be.

The EPA is proposing that all decimal digits be retained in intermediate steps of the calculation of the cumulative index, with the result rounded to have no decimal digits when expressed in ppm-hours before comparison the level of the secondary NAAQS.

EPA expects that the three months over which the cumulative weighted index value is highest will generally occur in the middle of each year. Therefore, the proposed new section 4 of Appendix P presumes this, and does not address a situation in which the three months of maximum cumulative index spans two calendar years, for example December to February. The EPA invites comment on whether a provision addressing such a remote possibility is needed and what its terms

⁶³ Because only enough missing 1-hour ozone values would be substituted as needed to meet the 75 percent completeness requirement, to avoid unreasonable underestimation of the true W126 index, tying the the selection of the substitution value to the hour of the missing value, as is proposed for data substitution for the purpose of the primary standard (see section V.D), would introduce considerable complexity by requiring an algorithm for determining which specific missing values would be substituted. Therefore, EPA is proposing this simpler substitution approach for the secondary standard.

should be. For example, the process of checking each three month period in a calendar year to determine which gives the highest index value could include the combinations of December/January/February and November/December/January within one calendar year.

C. Clarifications Related to the Primary Standard

The EPA is proposing two clarifying changes to Appendix P to make unambiguous two aspects of data interpretation for the primary 8-hour standard. The first change clarifies that the standard data completeness requirement that valid daily maximum 8-hour values exist for 75 percent of all days refers to days within the required O₃ monitoring season only. The current wording of Appendix P is somewhat open to a reading that the requirement applies to all days in the actual monitoring record for the site in question, which could be longer than the required season if a state voluntarily monitors on additional days, or shorter than the required season if a monitor has started or ceased operation sometime during the required season. The O₃ data completeness requirement is intended to avoid a determination that an area has met the NAAQS when in fact more than a reasonable number of days with high O₃ potential were not successfully monitored. This purpose can be served if the data within the required O₃ monitoring season only are reasonably complete, because as mentioned above EPA has proposed to revise the required O₃ seasons so that they encompass all days with potential for an exceedance of even the lowest proposed level for the primary standard. Unsuccessful monitoring outside the required season should not be an obstacle to a finding of attainment. However, if an O₃ monitor has missed more than 25 percent of the required O₃ monitoring season, for example because it started or stopped operation mid-season, this should prevent a finding of attainment based on a three-year period that includes that season. The proposed clarifying language reflects EPA's actual intention and our past practice in applying Appendix P for regulatory purposes, and Appendix I as well.⁶⁴

⁶⁴ At present, EPA's Air Quality System (AQS) for storing and reporting air quality data provides a completeness report that is based on yet a third approach, in which the period for reporting data completeness is the required monitoring season plus any extension needed to encompass any exceedances that may have occurred outside the required season. However, EPA's practice for regulatory purposes has been to consider completeness only over the required ozone monitoring season.

The second proposed clarifying change would make it clear that when determining the fourth-highest daily maximum 8-hour O₃ concentration for a year, all days with monitoring data are to be considered, not just days within the required O₃ monitoring season. This proposed clarifying language also reflects EPA's actual intention and our past practice in applying Appendix P, and Appendix I as well. While EPA believes it to be quite unlikely that an exceedance will occur outside the proposed revised required O₃ monitoring seasons and have a high enough concentration to affect the selection of the fourth-highest concentration for the year, when and if such an occurrence does happen, the data should not be ignored.

D. Revision to Exceptions From Standard Data Completeness Requirements for the Primary Standard

The EPA is proposing to revise portions of Appendix P that describe certain exceptions to the standard data completeness requirements, under which a monitoring site can in some cases be determined to be meeting or violating the primary NAAQS despite not meeting the standard data completeness requirements. These changes would make Appendix P more logical in certain types of cases with incomplete data. While the particular types of cases whose outcome would be different with these changes have been rare historically, there may be more such affected cases in the future in conjunction with a primary O₃ standard revised to a level within the range of levels proposed in this action.

The standard data completeness requirements in Appendix P for the primary O₃ NAAQS apply a 75 percent requirement at each of three stages of data completeness testing. As discussed below, for each stage, there is an existing exception to the 75 percent requirement.

In the first stage, an 8-hour period can be considered to have a valid 8-hour average O₃ concentration only if at least 75 percent of the hours, *i.e.*, 6 or more hours, have a valid hourly O₃ value. The provided exception is that if there are 5 or fewer hours but if substituting a very low value (specifically, one-half the MDL of the O₃ instrument) for all the missing hours results in a hypothetical 8-hour average that is above the level of the primary standard, the 8-hour period is considered valid and is assigned the hypothetical level resulting from the data substitution.⁶⁵ For example, if the

O₃ concentration was 0.125 ppm for 5 hours, substituting a typical MDL/2 value of 0.0025 ppm for three missing hours would result in an 8-hour average of 0.079 ppm, which is an exceedance of the current primary standard, so the valid 8-hour average for the period would be taken to be 0.079 ppm. If this value is higher than one or more of the highest four daily maximum 8-hour concentrations otherwise calculated for the year, considering it to be valid affects the value identified as the fourth-highest for the year and thus also affects the final design value. The logical problem with this approach is that it is possible for a hypothetical 8-hour average with such substitution to be below the level of the NAAQS, thus not meeting the current condition for the exception, but for it to still make a critical difference in making the three-year design value be above the level of the NAAQS, because a three-year design value can include (and be sensitive to the exact value of) an annual fourth-highest daily maximum that is not above the level of the NAAQS. This could be the case if the hypothetical 8-hour average with substitution is the maximum concentration 8-hour period for its day, and the day is one of the highest four O₃ days of the year. Whether it actually is the case would further depend on the value of the 8-hour average itself, the values of the next highest daily maximum 8-hour average concentration in the year, and the values of the annual fourth-highest daily maximum 8-hour concentration from the other two years. If the substituted 8-hour average would make a critical difference, it should be treated as valid and used in the calculation of the three-year design value, even if it is not itself above the level of the standard. Another problem is that one-half of the MDL, which typically is about 0.0025 ppm, is very likely to be considerably lower than the actual O₃ concentrations that were not successfully measured. Thus, while the one-half-MDL-substituted value is prevented from being an overestimate of the actual 8-hour average concentration, it is an unreasonably low estimate of that concentration which may have the effect of allowing a site with actual O₃ levels above the standard to be found to meet the standard. The condition in the exception requiring a one-half-MDL-substituted "8-hour" average to be above

the level of the NAAQS is therefore inappropriate.

In the second stage of data completeness testing, 75 percent of the 24 possible 8-hour time blocks, which is 18 or more, must have valid 8-hour average concentrations values. The intent of this requirement is to make sure that most of the day was actually monitored, such that the highest concentration 8-hour period was likely to be captured in the data. When this is not the case, the day is not considered in selecting the annual fourth-highest daily maximum 8-hour concentration and no credit for the day's monitoring is given towards the third stage of data interpretation (see below). The provided exception in the current Appendix P is that a day is considered valid if at least one 8-hour period has an average concentration above the level of the standard. However, as in the first stage, it is possible for an 8-hour period with an average concentration at or below the level of the NAAQS to play a critical role in whether the three-year design value meets the standard. Invalidating the day could have the effect of causing a lower value to be selected as the annual fourth-highest daily maximum 8-hour concentration, leading to a three-year design value that does not exceed the NAAQS while it would have exceeded if the day and the 8-hour average value had been treated as valid. The condition in the exception requiring at least one 8-hour average during the day to be above the level of the NAAQS is therefore inappropriate.

In the third stage of data completeness testing, a completeness criterion is applied for the number of days in the required O₃ season that have a valid maximum 8-hour average, *i.e.*, days that have met the completeness conditions in the first two stages or have met the condition for an exception. Specifically, for each of the three years being used in the design value calculation, the number of valid days within the required O₃ monitoring season (with no credit for extra days outside the season) must be at least 75 percent of the days in the required O₃ season, and the number of valid days across all three years must be 90 percent of the days in the three seasons.⁶⁶ The provided exception to the 75/90 percent requirement is that data from a year with less than 75 percent of seasonal days can nevertheless be used if during the year at least one day's maximum 8-hour average O₃ concentration was

⁶⁵ Actually, it is an interpretation of the text of Appendix P, section 2.1, that the average resulting

from the data substitution is to be taken as the "8-hour" average, rather than the average of the available 5 or fewer hours of data, which would be higher. The text is not entirely clear on this point.

⁶⁶ EPA also is proposing eliminate this 90 percent requirement, see section V.E. The point made in this paragraph applies with or without the 90 percent requirement in place.

above the level of the standard and if the three-year design value is also above the standard.⁶⁷ The problem with this exception, similar to the problems with the exceptions in the first and second stages of data completeness testing, is that a daily maximum 8-hour concentration that is at or below the level of the NAAQS can nevertheless make a critical difference in making the three-year design value be above the level of the NAAQS. When it does, an incorrect final result will be reached if the year of data is not granted an exception to the 75/90 percent requirement. Specifically, there would be no valid three-year design value and no conclusion would be reached as to attainment or nonattainment, despite it being clear that the actual situation is nonattainment, in the sense that successful collection of additional hours and days of monitoring data could not possibly have resulted in a passing three-year design value. Moreover, since the three-year design value is the average of the fourth-highest daily maximum 8-hour concentration from each year, there is no logical connection between the design value and the existence of a single daily maximum concentration greater than the level of the standard, which is the current condition for the exception for this stage of testing for data incompleteness.

EPA proposes to remedy this situation by replacing the three separate statements of the exceptions to the three standard completeness requirements with a new data substitution step that addresses the root cause of the data incompleteness situation: missing hourly concentrations which make it doubtful whether actual maximum daily 8-hour concentrations were measured on a reasonably large percentage of the days during the required O₃ monitoring season of each year. In the event that only 1, 2, 3, 4, or 5 hourly averages are available for an 8-hour period, a partially substituted 8-hour average would be computed by substituting for all the hours without hourly averages a low hourly average value selected as follows, and then using 8 as the divisor.⁶⁸ For days within the required O₃ monitoring season, the substitution

value would be the lowest hourly average O₃ concentration observed for that hour of the day (local standard time) on any day during the required O₃ monitoring season of that year, or one-half the MDL, whichever is higher. Using this value makes it highly unlikely that the resulting partially substituted 8-hour average concentration is higher than the actual concentration. Therefore, using the partially substituted 8-hour average in the design value calculation procedure is highly unlikely to result in an incorrect finding that a site does not meet the standard, but it may lead to a correct finding that a site does not meet the standard in some cases in which there would be no finding possible or an incorrect finding under the current version of Appendix P. However, the use of the higher of the lowest observed same-hour concentration or one-half the MDL could be problematic if a robust set of hourly measurements is not available for the year, for example if a monitor began operation late in an ozone season. In such a case, the lowest observed same-hour concentration might not be low enough to eliminate all possibility that the value used for substitution is higher than the missing concentration value. To reduce this likelihood to essentially zero, we are proposing that if the number of same-hour concentration values available for the required O₃ monitoring season for the year is less than 50 percent of the number of days during the required O₃ monitoring season, one-half the MDL of the O₃ instrument would be used in the substitution instead of the lowest observed concentration. We invite comment on whether another percentage should be used for this purpose instead of 50 percent.

The EPA notes that while this proposed approach to identifying the substitution value for the primary standard is technically appropriate, it would necessitate new data processing efforts during implementation that might be avoidable via some other approach that is also technically reasonable. There may also be approaches which are more technically appropriate. We therefore invite comment on such alternative approaches, and we may adopt another approach in the final rule. Examples of simpler approaches would be to identify in the final rule a fixed substitution value other than one-half the MDL, to accept as valid 8-hour periods with only five measured hourly concentrations, to interpret between two hourly concentrations to obtain a substitute for a single missing hourly concentration,

or to use the previous calendar year as the source of the substitution value instead of the current calendar year (thereby allowing all parties to know early in each year what the substitution value will be). Examples of more complex approaches that might be more technically appropriate include selecting a low percentile of the available same-hour concentration data rather than the lowest value to be the substitution value, or selecting the lowest same-hour value from the same calendar quarter or month (of the current year or the most recent year) rather than from the entire required ozone monitoring season. We also invite comment on whether the proposed approach to substitution should be used at all and if not what other approach should be used to address the potential problem just described.

We propose that for simplicity and to further reduce any risk of a false finding that a site does not meet the standard, for days outside the required O₃ monitoring season, the substitution value would always be one-half the MDL of the O₃ instrument. We similarly invite comment on this aspect.

There would be no condition that a partially substituted 8-hour average exceed the level of the standard for it be used in calculating the design value, unlike is now the case. An 8-hour period with no available hourly averages at all would not have a valid 8-hour average, as is now the case.

In addition, to complete the solution to the problems described above, we are proposing that a design value that is greater than the level of the primary standard would be valid provided that in each year there were at least four days with at least one valid 8-hour concentration.⁶⁹ One or more of these 8-hour average concentrations could be the partially substituted 8-hour average concentration resulting from the above described substitution procedure. In such a case, there is essentially no possibility that more complete monitoring data would have shown the site to be meeting the NAAQS. It is appropriate to include all 8-hour averages including those involving substitution when testing for an exceedance of the standard, because those averages are extremely unlikely to

⁶⁷ EPA notes that in the current versions of Appendix I and P, it is not explicit that this provided exception also applies in the case of three years which each have 75 percent or more of days with valid data but less than 90 percent across three years. Because EPA is proposing to remove the 90 percent requirement (see section V.E) this ambiguity does not need correction.

⁶⁸ Appendix P now provides that in the event that only 6 or 7 hourly averages are available, the valid 8-hour average shall be computed on the basis of the hours available, using 6 or 7 as the divisor. We are not proposing to change this provision.

⁶⁹ The requirement that there be at least four days with at least one hourly measurement is actually redundant and is stated only for ease of understanding, since there would be no annual fourth-highest daily maximum 8-hour concentration unless there are at least four days with monitoring data, and a single hourly data point is necessary and sufficient (with the proposed substitution step) to generate a daily maximum 8-hour concentration.

be overestimates of actual concentrations.

Finally, a design value equal to or less than the level of the standard would be valid only if at least 75 percent of the days in the required O₃ monitoring season of each year have daily maximum 8-hour concentrations that are based on at least 18 periods with at least 6 hourly concentrations. This ensures that a site will be found to meet the standard only when a reasonably high percentage of the days in the required O₃ monitoring season have reasonably complete hourly data. In this situation, it would be inappropriate to count the 8-hour periods with five or fewer actual hourly measurement values towards the 75 percent requirement when testing for whether a site meets the standard, because those 8-hour averages will be based on substitution of low values and therefore will be underestimates of actual concentrations. The only way to be reasonably certain that no 8-hour period had a high enough concentration so as to contribute to a design value over the level of the standard is to have at least 18 periods in which substitution for missing O₃ values was not needed. This provision has the same effect as several elements of the current Appendix P considered together, and thus is not a substantive change.

E. Elimination of the Requirement for 90 Percent Completeness of Daily Data Across Three Years

As stated above in section VI.D, Appendix P currently requires that in order for a design value equal to or less than the standard to be valid, at least 75 percent of days in each of three years must have a valid daily maximum 8-hour average concentration value, *i.e.*, that many days must have at least 18 8-hour periods with at least 6 reported hourly concentrations each. Appendix P also requires that the average of the percentages from three consecutive years be at least 90 percent. The EPA is proposing to eliminate this 90 percent requirement for the average of three years and to retain only the requirement that each individual year have a percentage of at least 75 percent.

The 90 percent requirement was incorporated into Appendix I (the data interpretation appendix for the 0.08 ppm O₃ NAAQS) in 1997 with an explanation that EPA had observed that 90 percent of O₃ monitoring sites routinely achieved 90 percent data capture. The EPA now notes, however, that while the majority of monitoring sites do achieve 90 percent or better data capture in any given year, there are exceptions every year. The 90 percent

requirement applied to the average percentage over three years is quite unforgiving if there has been one year with relatively low data completeness. For example, if one year just met the 75 percent requirement, the remaining two years would have to achieve a 97.5 percent data capture rate in order for the three years to meet the 90 percent requirement. This would allow only 4 missed hours of measurements per week, which would be challenging. The consequences for states could be important, under the current requirement. One possible result could be that an area actually in nonattainment with the NAAQS might have to be designated unclassifiable, although the substitution procedure proposed for cases of incomplete data, as described above in section VI.D, provides a path to an appropriate nonattainment finding in at least some cases. Another possible result is that a nonattainment area which had actually come into attainment could be unable to receive an attainment determination until three more years of sufficiently complete data are collected. This might, for example, result in an area which has achieved needed emissions reductions by its attainment deadline nevertheless being bumped up to a higher classification.

The 90 percent requirement over three years has the potential to treat two areas disparately, for no obvious logical reason. Consider two areas with identical air quality. Suppose the first area has annual completeness percentages of 75, 95, and 95 percent (averaging to 85 percent and thus failing the 90 percent requirement) and the second area has annual completeness percentages of 75, 98, and 98 percent (averaging to 90 percent). Suppose that the three-year design values in both areas are below the level of the NAAQS. Practically speaking, the most important uncertainty about whether each area actually meets the NAAQS is the low data capture rate in the first year. There is no obvious logic why the fact that the second area achieves marginally better data capture in the second and third year should permit it to receive an attainment finding despite this uncertainty, while the first area may not.

The EPA also notes that for the other gaseous criteria pollutants—sulfur dioxide, carbon monoxide and nitrogen dioxide—the completeness requirement is for 75 percent completeness of hourly measurements in an individual year.⁷⁰

⁷⁰ EPA has recently proposed to amend the completeness requirements for sulfur dioxide and nitrogen dioxide to add quarterly 75 percent

For these reasons, EPA proposes to eliminate the 90 percent requirement across three years of data but to retain the 75 percent requirement for individual years. The EPA notes that as a practical matter, the current 90 percent requirement in effect requires a minimum data capture rate somewhat above 75 percent in each year, because if data capture in any one year were as low as 75 percent the required data capture in the other years would be very hard to achieve. The minimum annual data capture rate is effectively somewhere in the range of 80 percent (implying a requirement to achieve 95 percent data capture in the two remaining years in order to meet the 90 percent requirement across three years) and 85 percent (implying a requirement to achieve 92.5 percent data capture in the two remaining years). The EPA invites comment on whether instead of retaining the 75 percent completeness requirement in each individual year, the requirement should be 80 percent or 85 percent.

F. Administrator Discretion To Use Incomplete Data

The EPA is proposing that the Administrator have general discretion to use incomplete data to calculate design values that would be treated as valid for comparison to the NAAQS despite the incompleteness, either at the request of a state or at her own initiative. Similar provisions exist already for the PM_{2.5} and lead NAAQS, and EPA has recently proposed such provisions to accompany the proposed 1-hour NO₂ and SO₂ primary NAAQS. The Administrator would consider monitoring site closures/moves, monitoring diligence, and nearby concentrations in determining whether to use such data.

G. Truncation Versus Rounding

Almost all State agencies now report hourly O₃ concentrations in parts per million to three decimal places, since the typical incremental sensitivity of currently used O₃ monitors is 0.001 ppm. In the current Appendix P approach, in calculating 8-hour average O₃ concentrations from such hourly data any calculated digits past the third decimal place are truncated rather than retained or rounded back to three decimal places. Also, in calculating 3-year averages of the fourth-highest daily maximum 8-hour average concentrations, Appendix P currently requires the result to be reported to the

completeness requirements in connection with proposals to establish 1-hour primary NAAQS for these pollutants, still with no requirement for 90 percent completeness across three years.

third decimal place with digits to the right of the third decimal place truncated. In this regard, Appendix P follows the precedent of Appendix I, except that Appendix P is based on a NAAQS level specified to three decimal places (0.075 ppm) while Appendix I addressed the case of a NAAQS level specified to only two decimal places (0.08 ppm). In the rulemaking that concluded in 2008 by establishing the 0.075 ppm level, EPA noted that the 2007 Staff Paper demonstrated that taking into account the precision and bias in 1-hour O₃ measurements, the 8-hour design value had an uncertainty of approximately 0.001 ppm. Thus, EPA considered any value less than 0.001 ppm to be highly uncertain and, therefore, proposed and adopted truncation to the third decimal place for reporting 1-hour O₃ concentrations and for both the individual 8-hour averages used to determine the annual fourth maximum and the 3-year average of the fourth maxima.

The effect of this repeated truncation is that there is a consistent downward bias in the calculation of the three-year design value. The size of this bias can be notable. For example, seven hours with O₃ concentrations of 0.076 ppm plus one hour of 0.075 ppm results in an 8-hour average of 0.075 ppm after truncation, nearly a full 0.001 ppm below the actual 8-hour average of 0.075875 ppm. Seven hours with O₃ concentrations of 0.077 ppm plus one hour of 0.076 ppm results in an 8-hour average of 0.076 ppm after truncation. One year with the first pattern plus two years with the second pattern would give a three-year design value of 0.075 ppm, meeting the NAAQS, even though 23 of the 24 individual 1-hour concentrations involved in the calculation of the design value were above 0.075 ppm.

The EPA has decided to reconsider this aspect of O₃ data interpretation. Specifically, we are proposing that (1) 1-hour concentrations continue to be reported to only three decimal places, the same as is now specified in Appendix P, *i.e.*, that the current practice of truncation of the 1-hour data to the nearest 0.001 ppm be retained; (2) all digits resulting from the calculation of 8-hour averages be retained; and (3) the three-year average of annual fourth-highest daily maximum 8-hour concentrations be rounded to three decimal places before comparison to the NAAQS. The EPA continues to believe that given the uncertainty in individual 1-hour O₃ concentration measurements it is appropriate to truncate those measurements at three decimal places (many O₃ instruments are programmed

to only report three digits anyway). However, the calculations of 8-hour averages and three-year averages are mathematical steps, not a measurement process subject to uncertainties, and EPA perceives no logic in having a consistent downward bias by truncating the results of these mathematical steps. The EPA notes that the O₃ NAAQS is the only NAAQS for which multi-hour, multi-day, or multi-year averages of concentrations are truncated rather than rounded. The proposed change will make this aspect of O₃ data interpretation consistent with data interpretation procedures for the other criteria pollutants.

H. Data Selection

The current version of Appendix P does not explicitly address the issue of what ambient monitoring data for O₃ can and must be compared to the O₃ NAAQS. The EPA proposes to add to Appendix P language addressing this issue. This language is similar to provisions recently proposed to be included in new data interpretation appendices for nitrogen dioxide and sulfur dioxide. The new section of Appendix P would clarify that all quality assured data collected with approved monitoring methods and known to EPA shall be compared to the NAAQS, even if not submitted to EPA's Air Quality System. The section also addresses the question of what O₃ data should be used when two or more O₃ monitors have been operating and have reported data for the same period at one monitoring site.

I. Exceptional Events Information Submission Schedule

States are responsible for identifying air quality data that they believe warrant special consideration, including data affected by exceptional events. States identify such data by flagging (making a notation in a designated field in the electronic data record) specific values in the Air Quality System (AQS) database. States must flag the data and submit a justification that the data are affected by exceptional events if they wish EPA to consider excluding the data in determining whether or not an area is attaining the new O₃ NAAQS.

All states that include areas that could exceed the O₃ NAAQS and could therefore be designated as nonattainment for the O₃ NAAQS have the potential to be affected by this rulemaking. Therefore, this action applies to all states; to local air quality agencies to which a state has delegated relevant responsibilities for air quality management including air quality monitoring and data analysis; and to

Tribal air quality agencies where appropriate. The Exceptional Events Rule preamble describes in greater detail to whom the rule applies (72 FR 13562–13563, March 22, 2007).

The CAA Section 319(b)(2) authorizes EPA to promulgate regulations that govern the review and handling of air quality monitoring data influenced by exceptional events. Under this authority, EPA promulgated the Exceptional Events Rule (Treatment of Data Influenced by Exceptional Events (72 FR 13560, March 22, 2007) which sets a schedule for states to flag monitored data affected by exceptional events in AQS and for them to submit documentation to demonstrate that the flagged data values were caused by an exceptional event. Under this schedule, a state must initially notify EPA that data have been affected by an exceptional event by July 1 of the year after the data are collected; this is accomplished by flagging the data in AQS. The state must also include an initial description of the event when flagging the data. In addition, the state is required to submit a full demonstration to justify exclusion of such data within three years after the quarter in which the data were collected, or if a regulatory decision based on the data (such as a designation action) is anticipated, the demonstration must be submitted to EPA no later than one year before the decision is to be made.

The rule also authorizes EPA to revise data flagging and documentation schedules for data used in the initial designation of areas under a new NAAQS. The generic schedule, while appropriate for the period after initial designations have been made under a NAAQS, may need adjustment when a new NAAQS is promulgated because until the level and form of the NAAQS have been promulgated, a state would not have complete knowledge of the criteria for excluding data. In these cases, the generic schedule may preclude states from submitting timely flags and associated documentation for otherwise approvable exceptional events. This could, if not modified, result in some areas receiving a nonattainment designation when the NAAQS violations were legitimately due to exceptional events.

As a result of the Administrator's decision to reconsider the 2008 O₃ NAAQS, EPA is proposing to revise the exceptional events flagging and documentation schedule to correspond to the designations schedules that EPA is considering for the proposed revisions to the primary and secondary O₃ NAAQS. The designation schedules

under consideration are discussed in greater detail below in section VII.A and summarized here. The CAA requires EPA to promulgate the initial designations for all areas no later than 2 years from the promulgation of a new NAAQS. Such period may be extended for up to one year if EPA has insufficient information. (See CAA section 107(d).) For a new primary O₃ standard, EPA intends to issue designations on an accelerated schedule. For a new seasonal secondary O₃ standard, EPA is considering two alternative schedules for initial area designations.

Primary Standard: If, as a result of the reconsideration, EPA promulgates a new primary O₃ standard on August 31, 2010, EPA is proposing that state Governors would need to submit their initial designation recommendations to EPA by January 7, 2011. EPA would promulgate the final designations in July 2011 to allow sufficient time for the designations to be published and effective by August 31, 2011. EPA expects to base the final designations for the primary O₃ standard on three consecutive years of certified air quality monitoring data from the years 2007–2009 or 2008–2010, if available. EPA is proposing that for exceptional event claims made for data years 2007–2009, states must flag and provide an initial description and detailed documentation by November 1, 2010. For data collected during data year 2010, EPA is proposing that exceptional event data that states want EPA to exclude from consideration in the designations process must be

flagged with an initial description and fully documented by March 1, 2011 or 60 days after the end of the quarter when the event occurred, whichever date is first. To meet this proposed 60-day deadline, a state would also have to submit the O₃ concentration data to AQS sooner than the normal deadline for such submission, which is 90 days after the end of the calendar quarter. EPA believes this is a reasonable expectation given that most states currently submit O₃ data earlier than the 90-day deadline.

Secondary Standard: If, as a result of the reconsideration, EPA promulgates a new seasonal secondary O₃ standard by August 31, 2010, EPA is taking comment on two alternative designations schedules. Under the first alternative, EPA would designate areas for the secondary standard on the same accelerated schedule discussed above for the primary standard. Under the second alternative, EPA would designate areas for the secondary standard on the maximum 2-year schedule provided under the CAA.

Accelerated Schedule: Under the accelerated schedule for a seasonal secondary O₃ NAAQS, EPA is proposing that for exceptional event claims made for data years 2007–2009, states must flag and provide an initial description and detailed documentation by November 1, 2010. For data collected during data year 2010, EPA is proposing that exceptional event data that states want EPA to exclude from consideration in the designations process must be flagged with an initial description and

fully documented by March 1, 2011 or 60 days after the end of the quarter when the event occurred, whichever date is first.

2-year Schedule: Under the 2-year schedule, states would have 1 year, or by August 2011, to submit their designations recommendations and EPA would finalize designations under the new secondary standard by August 2012. EPA expects to base final designations for a new seasonal secondary standard on the most recent three years of certified air quality monitoring data, which would typically be from the years 2009–2011 in this case. Exceptional event data claims used from years 2008–2010 must be flagged with an initial description included in AQS and submitted with complete documentation supporting such claims by July 1, 2011. Exceptional event data claims from data year 2011 must be flagged with an initial description and submitted with complete documentation supporting such claims 60 days after the end of the calendar quarter when the event occurred or March 1, 2012, whichever occurs first.

Therefore, using the authority provided in CAA section 319(b)(2) and in the Exceptional Events Rule at 40 CFR 50.14(c)(2)(vi), EPA is proposing to modify the schedule for data flagging and submission of demonstrations for exceptional events data considered for initial designations under the proposed reconsidered O₃ primary and secondary NAAQS, as follows:

TABLE 1—SCHEDULE FOR EXCEPTIONAL EVENT FLAGGING AND DOCUMENTATION SUBMISSION FOR DATA TO BE USED IN DESIGNATIONS DECISIONS FOR NEW NAAQS

NAAQS Pollutant/standard/(level)/promulgation date	Air quality data collected for calendar year	Event flagging & initial description deadline	Detailed documentation submission deadline
Primary Ozone/8-Hr Standard (Level TBD)/promulgated by August 31, 2010.	2007–2009	November 1, 2010 ^b	November 1, 2010. ^b
	2010	60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b	60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b
Secondary Ozone/(Level TBD) Alternative 2-year Schedule—to be promulgated by August 31, 2010.	2008	July 1, 2011 ^b	July 1, 2011. ^a
	2009–2010 2011	July 1, 2011 ^b	July 1, 2011. ^b 60 Days after the end of the calendar quarter in which the event occurred or March 1, 2012, whichever occurs first. ^b
Secondary Ozone/(Level TBD)—Alternative Accelerated Schedule—to be promulgated by August 31, 2010.	2007–2009	November 1, 2010 ^b	November 1, 2010. ^b
	2010	60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b	60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b

^a These dates are unchanged from those published in the original rulemaking.

^b Indicates change from general schedule in 40 CFR 50.14.

Note: EPA notes that the table of revised deadlines only applies to data EPA will use to establish the final initial designations for new NAAQS. The general schedule applies for all other purposes, most notably, for data used by EPA for redesignations to attainment.

VI. Ambient Monitoring Related to Proposed O₃ Standards

Presently, States (including the District of Columbia, Puerto Rico, and the Virgin Islands, and including local agencies when so delegated by the State) are required to operate minimum numbers of EPA-approved O₃ monitors based on the population of each of their Metropolitan Statistical Areas (MSA) and the most recently measured O₃ levels in each area. Each State (or in some cases portions of a State) also has a required O₃ monitoring season based on historical experience on when O₃ levels are high enough to be of regulatory or public health concern. These requirements are contained in 40 CFR part 58 Appendix D, Network Design Criteria for Ambient Air Quality Monitoring. See section 4.1, especially Tables D-2 and D-3. These requirements were last revised on October 17, 2006 as part of a comprehensive review of ambient monitoring requirements for all criteria pollutants (71 FR 61236).

A. Background

In the 2007 proposed rule for the O₃ NAAQS (72 FR 37818), EPA did not propose specific changes to monitoring requirements to support the proposed NAAQS revisions, but instead solicited comment on several key matters that were expected to be important issues affecting the potential redesign of monitoring networks if revisions to the NAAQS were finalized. These matters included O₃ monitoring requirements in urban areas, the potential need for monitoring to support multiple objectives important to characterization in non-urban areas including the support of the secondary O₃ NAAQS, and the length of the required O₃ monitoring seasons. Comments on these monitoring issues were received during the ensuing public comment period, and these comments were summarized in the 2008 final rule for the O₃ NAAQS (73 FR 16501). As noted in that action, EPA stated its intention to propose, in a separate rulemaking, the specific changes to O₃ monitoring requirements that were deemed necessary to support the revised 2008 O₃ NAAQS which set the level of the primary 8-hour O₃ standard to 0.075 ppm and set the secondary standard identical in all respects to the primary standard. EPA published these proposed changes to O₃ monitoring requirements in a proposal dated July 16, 2009, Ambient Ozone

Monitoring Regulations: Revisions to Network Design Requirements (74 FR 34525). The EPA currently plans to finalize these changes in a final O₃ monitoring rule in 2010, either before or in conjunction with the final rule on the O₃ NAAQS.

In the following sections, the specific provisions of the 2009 O₃ monitoring proposal are briefly reviewed, and then discussed in the context of the proposed revisions of the 2008 O₃ NAAQS that have been discussed earlier in this notice.

B. Urban Monitoring Requirements

As noted earlier, current O₃ monitoring requirements for urban areas are based on two factors: MSA population and the most recent 3-year design value concentrations within each MSA. There are higher minimum monitoring requirements for areas that have most recent design values greater than or equal to 85 percent of the NAAQS (*i.e.*, design value concentrations that are greater than or equal to 85 percent of the level of the NAAQS), and lower requirements for areas that have design values less than 85 percent of the NAAQS. These minimum monitoring requirements for O₃ were revised during the 2006 monitoring rulemaking to ensure that additional monitors would be required in areas with higher design values and to also ensure that these requirements would remain applicable through future NAAQS reviews and potential revisions of the standards. Accordingly, these requirements do not need to be updated with the revisions of the O₃ NAAQS proposed in this action since the 85 percent threshold will be applied to the standard levels that are finalized for the primary and secondary standards.⁷¹ For example, given the range of levels of the primary standard being proposed, the level of the 85 percent threshold that requires greater minimum monitoring requirements ranges from 0.051 ppm (85 percent of 0.060 ppm) to 0.060 ppm (85 percent of 0.070 ppm).

EPA did propose one change to urban monitoring requirements in the 2009 O₃

⁷¹ The requirements specified in Table D-2 of Appendix D to part 58, as noted in the third footnote of Table D-2, are applicable to the levels of the O₃ NAAQS as defined in 40 CFR part 50. Accordingly, the 85 percent threshold for requiring higher minimum monitoring requirements within MSAs would apply to the proposed levels for the cumulative, seasonal secondary standard as well as to the proposed levels of the 8-hour primary standard.

monitoring proposal. Specifically, EPA proposed to modify the minimum O₃ monitoring requirements to require one monitor to be placed in MSAs of populations ranging from 50,000 to less than 350,000 in situations where there is no current monitor and no history of O₃ monitoring within the previous 5 years indicating a design value of less than 85 percent of the revised NAAQS.⁷² Since this proposed change to minimum requirements is also subject to the 85 percent threshold, EPA believes that the proposed change remains appropriate to support the revisions to the primary and secondary O₃ NAAQS proposed in this action.

C. Non-Urban Monitoring Requirements

In the 2007 proposed rule for the O₃ NAAQS, EPA solicited comment on the status of monitoring requirements for non-urban areas, specifically whether non-urban areas with sensitive vegetation that are only currently sparsely monitored for O₃ could experience undetected violations of the secondary NAAQS as a result of transport from urban areas with high precursor emissions and/or O₃ concentrations or from formation of additional O₃ from precursors emitted from sources outside urban areas.

Comments that were received in response to the 2009 O₃ NAAQS monitoring proposal noted the voluntary nature of most non-urban O₃ monitoring and the resulting relative lack of non-urban O₃ monitors in some areas. These commenters stated that EPA should consider adding monitoring requirements to support the secondary NAAQS by requiring O₃ monitors in locations that contain O₃-sensitive plants or ecosystems. These commenters also noted that the placement of current O₃ monitors may not be appropriate for evaluating issues such as vegetation exposure since many of these monitors were likely located to meet other objectives.

Based on these comments as well as analyses of O₃ concentrations from discretionary non-urban monitors located across the U.S., EPA included new proposed non-urban O₃ monitoring requirements in the 2009 O₃ monitoring proposal. These proposed requirements are intended to satisfy several important objectives including: (1) Better characterization of O₃ concentrations to which O₃-sensitive vegetation and

⁷² These MSAs are not currently required to monitor for O₃.

ecosystems are exposed in rural/remote areas to ensure that potential secondary NAAQS violations are measured; (2) assessment of O₃ concentrations in smaller communities located outside of the larger urban MSAs covered by urban monitoring requirements; and (3) the assessment of the location and severity of maximum O₃ concentrations that occur in non-urban areas and may be attributable to upwind urban sources. For reasons noted below, EPA believes that these proposed O₃ monitoring requirements are sufficient to support the revisions to the O₃ NAAQS proposed in this action.

With regard to the first objective, we note uncertainties will remain about the O₃ concentrations to which sensitive natural vegetation and ecosystems are exposed until additional monitors are sited in National Parks, State and/or tribal areas, wilderness areas, and other similar locations with sensitive ecosystems that are set aside to provide similar public welfare benefits. These monitors would support evaluation of the secondary NAAQS with a more robust data set than is now available. As noted in the 2009 O₃ monitoring proposal, EPA proposed that States operate at least one monitor to be located in areas such as some Federal, State, Tribal, or private lands, including wilderness areas that have O₃-sensitive natural vegetation and/or ecosystems. If EPA finalizes a cumulative, seasonal secondary standard at the lower end of the proposed range, then it is plausible that additional O₃ monitors, above the number required by the monitoring proposal, may be needed in such areas to provide adequate coverage of locations likely to experience violations of the revised secondary NAAQS. These additional monitors could be established through discretionary State initiatives to supplement minimum monitoring requirements, negotiated agreements between States and EPA Regional Administrators, or through a future rulemaking that addresses potential increased O₃ monitoring requirements to specifically address the need for additional monitoring to ensure detection of secondary standard violations.

With regard to the second objective of characterizing elevated ambient O₃ levels to which people are exposed in smaller communities located outside of the larger urban MSAs, the likelihood of measuring concentrations that approach or exceed the levels of the NAAQS due to the transport of O₃ from upwind areas and/or the formation of O₃ due to precursor emissions from industrial sources outside of urban areas is clearly increased due to the revised NAAQS

proposed in this action. Given that the analyses described in the 2009 O₃ monitoring proposal demonstrated that 50 percent of existing monitors located in such Micropolitan Statistical Areas⁷³ exceeded the current NAAQS and nearly all monitors recorded design values greater than or equal to 85 percent of the current NAAQS, the potential for violations in such areas can only be increased with the NAAQS revisions proposed in this action. As noted for the first non-urban monitoring objective, it is plausible that additional O₃ monitors, above the number required by the 2009 monitoring proposal may be needed in Micropolitan Statistical Areas to provide adequate coverage of locations likely to experience violations of the proposed lower primary NAAQS levels. These additional monitors could be established through discretionary State initiatives to supplement minimum monitoring requirements, negotiated requirements between States and EPA Regional Administrators, or through a future rulemaking that addresses potential increased O₃ monitoring requirements to specifically address the need for additional monitoring to ensure detection of primary standard violations in smaller communities.

The third proposed non-urban monitoring objective, requiring O₃ monitors to be located in the area of expected maximum O₃ concentration outside of any MSA, potentially including the far downwind transport zones of currently well-monitored urban areas, is not directly related to the level of the O₃ NAAQS. It is instead intended to ensure that all parts of a State meet the NAAQS and that all necessary emission control strategies have been included in State Implementation Plans. Accordingly, this proposed monitoring objective remains applicable independent of revisions to the O₃ NAAQS proposed in this action.

D. Revisions to the Length of the Required O₃ Monitoring Seasons

Ozone monitoring is only required during the seasons of the year that are conducive to O₃ formation. These seasons vary in length as the conditions that determine the likely O₃ formation (*i.e.*, seasonally-dependent factors such as ambient temperature, strength of solar insolation, and length of day) differ by location. In some locations, conditions conducive to O₃ formation are limited to a few summer months of the year while in other locations these

conditions occur year-round. As a result, the length of currently required O₃ monitoring seasons can vary from a length of 4 months in colder climates to a length of 12 months in warmer climates.

The 2009 O₃ monitoring proposal also addressed the issue of whether in some areas the required O₃ monitoring season should be made longer. The proposal also addressed the status of any currently effective Regional Administrator-granted waiver approvals to O₃ monitoring seasons, and the impact of proposed changes to monitoring requirements on such waiver approvals.

The EPA performed several analyses in support of proposed changes to the required O₃ monitoring seasons. The first analysis determined the number of observed exceedances of the 0.075 ppm level of the current 8-hour NAAQS in the months falling outside the currently required local O₃ monitoring season using monitors in areas that collected O₃ data year-round in 2004–2006. The second analysis examined observed occurrences of daily maximum 8-hour O₃ averages of at least 0.060 ppm. This threshold was chosen because it represented 80 percent of the current 0.075 ppm NAAQS level and provides an indicator of ambient conditions that may be conducive to the formation of O₃ concentrations that approach or exceed the NAAQS. While proposals for revising each State's required monitoring season were based on observed data in and surrounding each State, statistically predicted exceedances were also used to validate conclusions for each State.

The aforementioned analyses provided several results. The analysis of observed exceedances of the 0.075 ppm level of the current O₃ NAAQS indicated occurrences in eight States during months outside of the current required monitoring season. The eight States were Maine, Massachusetts, New Hampshire, New Jersey, New York, South Carolina, Vermont, and Wyoming. With the exception of Wyoming, these exceedances occurred in a very limited manner and timeframe, just before the beginning of these States' required O₃ monitoring season (beginning in these States on April 1). The frequency of observed occurrences of maximum 8-hour average O₃ levels of at least 0.060 ppm was quite high across the country in months outside of the current required monitoring season. A total of 32 States experienced such occurrences; 22 States had such levels only before the required monitoring season; 9 States had such levels both before and after the required monitoring

⁷³ Defined as areas having at least one urban cluster of at least 10,000 but less than a population of 50,000.

season; and 1 State had such levels only after the required monitoring season. In a number of cases, the frequency of such ambient concentrations was high, with some States experiencing between 31 to 46 out-of-season days during 2004 to 2006 at a high percentage of all operating year-round O₃ monitors.

Based on these analyses, EPA proposed a lengthening of the O₃ monitoring season requirements in many areas. The 2009 proposed changes were based not only on the goal of monitoring out-of-season O₃ NAAQS violations but also on the goal of ensuring monitoring when ambient O₃ levels reach 80 percent of the NAAQS so that persons unusually sensitive to O₃ would be alerted to potential NAAQS exceedances.

The EPA believes that the factors used to support the 2009 proposed changes to O₃ monitoring seasons are appropriate to support the revisions of the O₃ NAAQS proposed in this action. With regard to the primary standard, we note that the lower end of the range being proposed is an 8-hour level of 0.060 ppm, which is identical to the ambient O₃ level that was utilized in one of the analyses discussed above. Although that level was chosen to provide an indicator of ambient levels that were below but approaching the level of the NAAQS and hence to serve as an alert to potential exceedances, we note that EPA's traditional practice had been to base the length of required O₃ monitoring seasons on the likelihood of measuring exceedances of the level of the NAAQS. Therefore, if EPA finalizes the level of the primary standard at the lower end of the proposed range, the O₃ monitoring seasons that have been proposed as part of the 2009 O₃ monitoring proposal would provide sufficient monitoring coverage to ensure the goal of measuring potential violations of the primary standard.

One O₃ monitoring season issue that was not considered in the 2009 O₃ monitoring proposal was the question of whether analyses of ambient data based on 8-hour average statistics would also indicate whether the resulting proposed monitoring seasons would capture the cumulative maximum consecutive 3-month O₃ levels necessary to compute design values based on the secondary NAAQS proposed in this action, which is defined in terms of a W126 cumulative peak-weighted index, as discussed above in section IV. If areas experienced high cumulative index values during months outside of the required O₃ monitoring seasons (based on 8-hour statistics), then further revisions to the required monitoring seasons might be necessary to ensure

monitoring during all months important to the calculation of design values for the revised form proposed for the secondary NAAQS. A related issue is whether such high cumulative O₃ values also occurred during time periods that are biologically relevant for O₃-sensitive vegetation.

The EPA is not proposing additional revisions to O₃ monitoring seasons at this time. Additional analyses of the distribution of elevated cumulative W126 index values will be conducted, and the biologically relevant seasonal issue will be further reviewed. Based on the results of these analyses, EPA may consider proposing further revisions to the O₃ monitoring season as related to the secondary O₃ NAAQS.

VII. Implementation of Proposed O₃ Standards

A. Designations

After EPA establishes or revises a NAAQS, the CAA directs EPA and the states to take steps to ensure that the new or revised NAAQS are met. The first step is to identify areas of the country that do not meet the new or revised NAAQS. This step is known as the initial area designations.

The CAA provides that, "By such date as the Administrator may reasonably require, but not later than 1 year after promulgation of a new or revised national ambient air quality standard for any pollutant under section 109, the Governor of each state shall * * * submit to the Administrator a list of all areas (or portions thereof) in the state" that designates those areas as nonattainment, attainment, or unclassifiable. The CAA specifies that, "The Administrator may not require the Governor to submit the required list sooner than 120 days after promulgating a new or revised national ambient air quality standard." The CAA defines an area as nonattainment if it is violating the NAAQS or if it is contributing to a violation in a nearby area. (See CAA section 107(d)(1).)

The CAA further provides, "Upon promulgation or revision of a national ambient air quality standard, the Administrator shall promulgate the designations of all areas (or portions thereof) * * * as expeditiously as practicable, but in no case later than 2 years from the date of promulgation of the new or revised national ambient air quality standard. Such period may be extended for up to one year in the event the Administrator has insufficient information to promulgate the designations." EPA is required to notify states of any intended modifications to their recommendations that EPA may

deem necessary no later than 120 days prior to promulgating designations. States then have an opportunity to demonstrate why any such proposed modification is inappropriate. Whether or not a state provides a recommendation, EPA must promulgate the designation that the Agency deems appropriate. (See CAA section 107(d)(1)(B).)

On September 16, 2009, when the Administrator announced her decision to reconsider the 2008 O₃ NAAQS, she also indicated that the Agency would work with states to accelerate implementation of the standards adopted after reconsideration, including the initial area designations process. Acceleration of designations for the primary standard would help limit any delays in health protections associated with the reconsideration of the standards. If a secondary standard different from the primary standard is adopted, this would be the first time different primary and secondary standards would be in place for the O₃ standards. While welfare protection is also important, for the reasons provided below, we are providing alternative schedules for designating areas for the secondary standard.

If, as a result of the reconsideration, EPA determines that the record supports a primary standard different from that promulgated in 2008 and promulgates such different primary O₃ NAAQS in 2010, EPA intends to promulgate final designations on an accelerated schedule to allow the designations to be effective in 1 year. In order to meet such a schedule, EPA is proposing that the deadline for states to submit their designations recommendations for the revised 2010 primary standard be 129 days after promulgation of that primary standard. EPA recognizes that the proposed deadline would be an ambitious schedule. Therefore, EPA intends to provide technical information and guidance for states as early as possible to facilitate the development of their recommendations. Many of the areas that would be violating the proposed primary ozone standard are also violating the 2008 ozone standards. State Governors have provided recommendations on these areas pursuant to the 2008 standards and recommendations may not need much further evaluation.

Based on this proposed schedule, if EPA promulgates a new primary standard on August 31, 2010, state Governors would need to submit their initial designation recommendations to EPA by January 7, 2011. If the Administrator intends to modify any state recommendation, EPA would

notify the Governor no later than March 2011, 120 days prior to promulgating the final designations. States would then have an opportunity to comment on EPA's intended designations before EPA promulgates the final designations. EPA would promulgate the final designations in July 2011 to allow sufficient time for the designations to be published and effective by August 31, 2011. EPA expects to base the final designations for the primary O₃ standard on three consecutive years of certified air quality monitoring data from the years 2007–2009 or from 2008–2010, if available.

If, as a result of the reconsideration, EPA promulgates a distinct secondary standard that differs from that promulgated in 2008 and also differs from the 2010 primary standard, as proposed above, EPA is proposing two alternative deadlines for states to submit their designations recommendations. Under the first alternative, EPA would designate areas for the secondary standard on the same accelerated schedule discussed above for the primary standard. In order to meet that schedule, EPA is proposing that states submit their recommendations for the revised 2010 secondary standard 129 days after promulgation of that secondary standard. Accordingly, if EPA promulgates the new secondary standard on August 31, 2010, state Governors would need to submit their initial designation recommendations to EPA by January 7, 2011.

Weighing in favor of designating areas for the secondary standard at the same time as designations for the primary standard is that planning for both standards would occur on the same schedule. Our examination of current air quality data from the existing monitoring network indicates that for levels of the primary and secondary standards proposed in this action, it is likely that the vast majority of areas violating the secondary standard would overlap with areas violating the primary standard. In this case, implementing requirements for the primary and secondary standards on different schedules could present resource challenges to state and local agencies by requiring duplication of effort and hindering consideration of all factors when deciding which control strategies to adopt for each standard. For example, if designations for the secondary standard were delayed by a certain period (*e.g.*, a year) beyond the designations for the primary standard, then EPA would likely delay submission of attainment SIPs for the secondary standard for a similar period beyond the proposed date for

submission of the attainment SIPs for the primary standard. In this case, the initial transportation conformity determination for the secondary standard would be required later than the initial determination for the primary standard by the difference in time between the effective dates of the two designations.

Under the second alternative, EPA would designate areas for the secondary standard on the maximum 2-year schedule provided under the CAA. To meet that 2-year schedule, EPA is proposing that states submit their recommendations for the revised secondary standard no later than 1 year after promulgation of the 2010 secondary standard. Accordingly, if EPA promulgates a secondary standard on August 31, 2010, that differs from the primary standard, as proposed, under the alternative 2-year designations schedule, state Governors would need to submit their initial designation recommendations to EPA by August 31, 2011. If the Administrator intends to modify any state recommendation, EPA would notify the Governor no later than May 2012, 120 days prior to the 2-year deadline for promulgating the final designations. States would then have an opportunity to comment on EPA's intended designations before EPA promulgates the final designations. EPA would promulgate the final designations for the secondary standard by August 31, 2012. EPA expects to base the final designations in August 2012 for a different secondary standard on the most recent three consecutive years of certified air quality monitoring data, which would be from the years 2009–2011.

In the past, EPA has always set the secondary O₃ standard to be identical to the primary O₃ standard and the standards have embodied relatively short-term average concentrations (*e.g.*, 1-hour or 8-hour). In this action, EPA is proposing a cumulative, seasonal secondary standard that differs from the proposed primary standard. EPA has not previously set a seasonal secondary standard for O₃. Therefore, EPA and states do not have experience in implementing this type of secondary O₃ standard or in determining what area boundaries would be appropriate. As we further explore implementation considerations for the secondary standard, we may encounter unanticipated issues that may require additional time to address. Thus, EPA is considering whether an accelerated schedule for a seasonal secondary standard would provide adequate time for resolving issues that we cannot now anticipate. If EPA designates areas for

the secondary standard on a 2-year schedule, we note that we expect that accelerated implementation of the health-based primary standard would also result in accelerated welfare benefits. EPA requests comment on factors affecting the efficient and effective implementation of a secondary standard that differs from the primary standard in the context of establishing designations schedules.

EPA notes, as discussed in greater detail above in section VI, that it has proposed a monitoring rule that would increase the density of monitoring in National Parks and other non-urban and lesser populated areas (July 16, 2009; 74 FR 34525). The proposed requirements are intended to satisfy several important objectives, including better characterization of O₃ exposures to O₃-sensitive vegetation and ecosystems in rural/remote areas to ensure that potential secondary NAAQS violations are measured. As proposed, the new monitors would not be deployed until 2012 or 2013. Therefore, data from these monitors would not be available for use within the statutory timeframe for EPA to complete designations for a 2010 secondary standard regardless of which schedule EPA follows.

While CAA section 107 specifically addresses states, EPA intends to follow the same process for tribes to the extent practicable, pursuant to section 301(d) of the CAA regarding tribal authority, and the Tribal Authority Rule (63 FR 7254; February 12, 1998).

In a separate notice elsewhere in today's **Federal Register**, EPA is announcing that it is using its authority under the CAA to extend by 1 year the deadline for promulgating initial area designations for the O₃ NAAQS that were promulgated in March 2008. The new deadline is March 12, 2011. That notice explains the basis for the deadline extension. As mentioned above, on September 16, 2009, EPA notified the Court of its decision to initiate a rulemaking to reconsider the primary and secondary O₃ NAAQS set in March 2008 to ensure they satisfy the requirements of the CAA. In its notice to the Court, EPA stated that the final rule would be signed by August 31, 2010. Extending the deadline for promulgating designations for the 2008 O₃ NAAQS until March 12, 2011 will allow EPA to complete the reconsideration rulemaking for the 2008 O₃ NAAQS before determining whether it is necessary to finalize designations for those NAAQS or, instead, whether it is necessary to begin the designation process for different NAAQS promulgated pursuant to the reconsideration.

B. State Implementation Plans

The CAA section 110 provides the general requirements for SIPs. Within 3 years after the promulgation of new or revised NAAQS (or such shorter period as the Administrator may prescribe) each State must adopt and submit "infrastructure" SIPs to EPA to address the requirements of section 110(a)(1). Thus, States should submit these SIPs no later than August 21, 2013, three years after promulgation of the reconsidered ozone standard in 2010. These "infrastructure SIPs" provide assurances of State resources and authorities, and establish the basic State programs, to implement, maintain, and enforce new or revised standards.

In addition to the infrastructure SIPs, which apply to all States, CAA title I, part D outlines the State requirements for achieving clean air in designated nonattainment areas. These requirements include timelines for when designated nonattainment areas must attain the standards, deadlines for developing SIPs that demonstrate how the State will ensure attainment of the standards, and specific emissions control requirements. EPA plans to address how these requirements, such as attainment demonstrations and attainment dates, reasonable further progress, new source review, conformity, and other implementation requirements, apply to the O₃ NAAQS promulgated pursuant to the reconsideration in a subsequent rulemaking. Also in that rulemaking EPA will establish deadlines for submission of nonattainment area SIPs but anticipates that the deadlines will be no later than the end of December 2013, or 28 months after final designations.

C. Trans-Boundary Emissions

Cross border O₃ contributions from within North America (Canada and Mexico) entering the U.S. are generally thought to be small. Section 179B of the Clean Air Act allows designated nonattainment areas to petition EPA to consider whether such a locality might have met a clean air standard "but for" cross border contributions. To date, few areas have petitioned EPA under this authority. The impact of foreign emissions on domestic air quality in the United States is a challenging and complex problem to assess. EPA is engaged in a number of activities to improve our understanding of international transport. As work progresses on these activities, EPA will be able to better address the uncertainties associated with trans-

boundary flows of air pollution and their impacts.

VIII. Statutory and Executive Order Reviews

A. Executive Order 12866: Regulatory Planning and Review

Under section 3(f)(1) of Executive Order (EO) 12866 (58 FR 51735, October 4, 1993), the O₃ NAAQS action is an "economically significant regulatory action" because it is likely to have an annual effect on the economy of \$100 million or more. Accordingly, EPA submitted this action to the Office of Management and Budget (OMB) for review under EO 12866 and any changes made in response to OMB recommendations have been documented in the docket for this action. In addition, EPA prepared this regulatory impact analysis (RIA) of the potential costs and benefits associated with this action. This analysis is contained in the Regulatory Impact Analysis for the Ozone NAAQS Reconsideration, October 2009 (henceforth, "RIA"). A copy of the analysis is available in the RIA docket (EPA-HQ-OAR-2007-0225) and the analysis is briefly summarized here. The RIA estimates the costs and monetized human health and welfare benefits of attaining five alternative O₃ NAAQS nationwide. Specifically, the RIA examines the alternatives of 0.079 ppm, 0.075 ppm, 0.070 ppm, 0.065 ppm, and 0.060 ppm. The RIA contains illustrative analyses that consider a limited number of emissions control scenarios that States and Regional Planning Organizations might implement to achieve these alternative O₃ NAAQS. However, the Clean Air Act (CAA) and judicial decisions make clear that the economic and technical feasibility of attaining ambient standards are not to be considered in setting or revising NAAQS, although such factors may be considered in the development of State plans to implement the standards. Accordingly, although an RIA has been prepared, the results of the RIA have not been considered in issuing this proposed rule.

B. Paperwork Reduction Act

This action does not impose an information collection burden under the provisions of the Paperwork Reduction Act, 44 U.S.C. 3501 *et seq.* There are no information collection requirements directly associated with the establishment of a NAAQS under section 109 of the CAA.

Burden means the total time, effort, or financial resources expended by persons

to generate, maintain, retain, or disclose or provide information to or for a Federal agency. This includes the time needed to review instructions; develop, acquire, install, and utilize technology and systems for the purposes of collecting, validating, and verifying information, processing and maintaining information, and disclosing and providing information; adjust the existing ways to comply with any previously applicable instructions and requirements; train personnel to be able to respond to a collection of information; search data sources; complete and review the collection of information; and transmit or otherwise disclose the information.

An agency may not conduct or sponsor, and a person is not required to respond to a collection of information unless it displays a currently valid OMB control number. The OMB control numbers for EPA's regulations in 40 CFR are listed in 40 CFR part 9.

C. Regulatory Flexibility Act

The Regulatory Flexibility Act (RFA) generally requires an agency to prepare a regulatory flexibility analysis of any rule subject to notice and comment rulemaking requirements under the Administrative Procedure Act or any other statute unless the agency certifies that the rule will not have a significant economic impact on a substantial number of small entities. Small entities include small businesses, small organizations, and small governmental jurisdictions.

For purposes of assessing the impacts of today's proposed rule on small entities, small entity is defined as: (1) A small business that is a small industrial entity as defined by the Small Business Administration's (SBA) regulations at 13 CFR 121.201; (2) a small governmental jurisdiction that is a government of a city, county, town, school district or special district with a population of less than 50,000; and (3) a small organization that is any not-for-profit enterprise which is independently owned and operated and is not dominant in its field.

After considering the economic impacts of today's proposed rule on small entities, I certify that this action will not have a significant economic impact on a substantial number of small entities. This proposed rule will not impose any requirements on small entities. Rather, this rule establishes national standards for allowable concentrations of O₃ in ambient air as required by section 109 of the CAA. See also *American Trucking Associations v. EPA*, 175 F. 3d at 1044-45 (NAAQS do not have significant impacts upon small

entities because NAAQS themselves impose no regulations upon small entities). We continue to be interested in the potential impacts of the proposed rule on small entities and welcome comments on issues related to such impacts.

D. Unfunded Mandates Reform Act

Title II of the Unfunded Mandates Reform Act of 1995 (UMRA), Public Law 104–4, establishes requirements for Federal agencies to assess the effects of their regulatory actions on State, local, and Tribal governments and the private sector. Under section 202 of the UMRA, EPA generally must prepare a written statement, including a cost-benefit analysis, for proposed and final rules with “Federal mandates” that may result in expenditures to State, local, and Tribal governments, in the aggregate, or to the private sector, of \$100 million or more in any 1 year. Before promulgating an EPA rule for which a written statement is needed, section 205 of the UMRA generally requires EPA to identify and consider a reasonable number of regulatory alternatives and to adopt the least costly, most cost-effective or least burdensome alternative that achieves the objectives of the rule. The provisions of section 205 do not apply when they are inconsistent with applicable law. Moreover, section 205 allows EPA to adopt an alternative other than the least costly, most cost-effective or least burdensome alternative if the Administrator publishes with the final rule an explanation why that alternative was not adopted. Before EPA establishes any regulatory requirements that may significantly or uniquely affect small governments, including Tribal governments, it must have developed under section 203 of the UMRA a small government agency plan. The plan must provide for notifying potentially affected small governments, enabling officials of affected small governments to have meaningful and timely input in the development of EPA regulatory proposals with significant Federal intergovernmental mandates, and informing, educating, and advising small governments on compliance with the regulatory requirements.

Today’s proposed rule contains no Federal mandates (under the regulatory provisions of Title II of the UMRA) for State, local, or Tribal governments or the private sector. The proposed rule imposes no new expenditure or enforceable duty on any State, local or Tribal governments or the private sector, and EPA has determined that this proposed rule contains no regulatory requirements that might significantly or uniquely affect small governments.

Furthermore, as indicated previously, in setting a NAAQS EPA cannot consider the economic or technological feasibility of attaining ambient air quality standards, although such factors may be considered to a degree in the development of State plans to implement the standards. See also *American Trucking Associations v. EPA*, 175 F. 3d at 1043 (noting that because EPA is precluded from considering costs of implementation in establishing NAAQS, preparation of a Regulatory Impact Analysis pursuant to the Unfunded Mandates Reform Act would not furnish any information which the court could consider in reviewing the NAAQS). Accordingly, EPA has determined that the provisions of sections 202, 203, and 205 of the UMRA do not apply to this proposed decision. The EPA acknowledges, however, that any corresponding revisions to associated SIP requirements and air quality surveillance requirements, 40 CFR part 51 and 40 CFR part 58, respectively, might result in such effects. Accordingly, EPA will address, as appropriate, unfunded mandates if and when it proposes any revisions to 40 CFR parts 51 or 58.

E. Executive Order 13132: Federalism

Executive Order 13132, entitled “Federalism” (64 FR 43255, August 10, 1999), requires EPA to develop an accountable process to ensure “meaningful and timely input by State and local officials in the development of regulatory policies that have federalism implications.” “Policies that have federalism implications” is defined in the Executive Order to include regulations that have “substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government.”

This proposed rule does not have federalism implications. It will not have substantial direct effects on the States, on the relationship between the national government and the States, or on the distribution of power and responsibilities among the various levels of government, as specified in Executive Order 13132. The rule does not alter the relationship between the Federal government and the States regarding the establishment and implementation of air quality improvement programs as codified in the CAA. Under section 109 of the CAA, EPA is mandated to establish NAAQS; however, CAA section 116 preserves the rights of States to establish more stringent requirements if deemed necessary by a State. Furthermore, this

proposed rule does not impact CAA section 107 which establishes that the States have primary responsibility for implementation of the NAAQS. Finally, as noted in section E (above) on UMRA, this rule does not impose significant costs on State, local, or Tribal governments or the private sector. Thus, Executive Order 13132 does not apply to this rule.

However, as also noted in section D (above) on UMRA, EPA recognizes that States will have a substantial interest in this rule and any corresponding revisions to associated SIP requirements and air quality surveillance requirements, 40 CFR part 51 and 40 CFR part 58, respectively. Therefore, in the spirit of Executive Order 13132, and consistent with EPA policy to promote communications between EPA and State and local governments, EPA specifically solicits comment on this proposed rule from State and local officials.

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

Executive Order 13175, entitled “Consultation and Coordination with Indian Tribal Governments” (65 FR 67249, November 9, 2000), requires EPA to develop an accountable process to ensure “meaningful and timely input by tribal officials in the development of regulatory policies that have tribal implications.” This rule concerns the establishment of O₃ NAAQS. The Tribal Authority Rule gives Tribes the opportunity to develop and implement CAA programs such as the O₃ NAAQS, but it leaves to the discretion of the Tribe whether to develop these programs and which programs, or appropriate elements of a program, they will adopt.

This proposed rule does not have Tribal implications, as specified in Executive Order 13175. It does not have a substantial direct effect on one or more Indian Tribes, since Tribes are not obligated to adopt or implement any NAAQS. Thus, Executive Order 13175 does not apply to this rule.

Although Executive Order 13175 does not apply to this rule, EPA contacted tribal environmental professionals during the development of the March 2008 rule. The EPA staff participated in the regularly scheduled Tribal Air call sponsored by the National Tribal Air Association during the spring of 2007 as the proposal was under development. EPA specifically solicits additional comment on this proposed rule from Tribal officials.

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

Executive Order 13045, "Protection of Children from Environmental Health Risks and Safety Risks" (62 FR 19885, April 23, 1997) applies to any rule that: (1) Is determined to be "economically significant" as defined under Executive Order 12866, and (2) concerns an environmental health or safety risk that EPA has reason to believe may have a disproportionate effect on children. If the regulatory action meets both criteria, the Agency must evaluate the environmental health or safety effects of the planned rule on children, and explain why the planned regulation is preferable to other potentially effective and reasonably feasible alternatives considered by the Agency.

This proposed rule is subject to Executive Order 13045 because it is an economically significant regulatory action as defined by Executive Order 12866, and we believe that the environmental health risk addressed by this action may have a disproportionate effect on children. The proposed rule will establish uniform national ambient air quality standards for O₃; these standards are designed to protect public health with an adequate margin of safety, as required by CAA section 109. However, the protection offered by these standards may be especially important for children because children, especially children with asthma, along with other sensitive population subgroups such as all people with lung disease and people active outdoors, are potentially susceptible to health effects resulting from O₃ exposure. Because children are considered a potentially susceptible population, we have carefully evaluated the environmental health effects of exposure to O₃ pollution among children. Discussions of the results of the evaluation of the scientific evidence, policy considerations, and the exposure and risk assessments pertaining to children are contained in sections II.B and II.C of this preamble. A listing of the documents that contain the evaluation of scientific evidence, policy considerations, and exposure and risk assessments that pertain to children is found in the section on Children's Environmental Health in the Supplementary Information section of this preamble, and a copy of all documents have been placed in the public docket for this action. The public is invited to submit comments or identify peer-reviewed studies and data that assess effects of early life exposure to O₃.

H. Executive Order 13211: Actions That Significantly Affect Energy Supply, Distribution or Use

This proposed rule is not a "significant energy action" as defined in Executive Order 13211, "Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use" (66 FR 28355 (May 22, 2001)) because in the Agency's judgment it is not likely to have a significant adverse effect on the supply, distribution, or use of energy. The purpose of this rule is to establish revised NAAQS for O₃. The rule does not prescribe specific pollution control strategies by which these ambient standards will be met. Such strategies will be developed by States on a case-by-case basis, and EPA cannot predict whether the control options selected by States will include regulations on energy suppliers, distributors, or users. Thus, EPA concludes that this rule is not likely to have any adverse energy effects and does not constitute a significant energy action as defined in Executive Order 13211.

I. National Technology Transfer and Advancement Act

Section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104-113, section 12(d) (15 U.S.C. 272 note) directs EPA to use voluntary consensus standards in its regulatory activities unless to do so would be inconsistent with applicable law or otherwise impractical. Voluntary consensus standards are technical standards (e.g., materials specifications, test methods, sampling procedures, and business practices) that are developed or adopted by voluntary consensus standards bodies. The NTTAA directs EPA to provide Congress, through OMB, explanations when the Agency decides not to use available and applicable voluntary consensus standards.

This proposed rulemaking does not involve technical standards. Therefore, EPA is not considering the use of any voluntary consensus standards.

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

Executive Order 12898 (59 FR 7629 (Feb. 16, 1994)) establishes federal executive policy on environmental justice. Its main provision directs federal agencies, to the greatest extent practicable and permitted by law, to make environmental justice part of their mission by identifying and addressing, as appropriate, disproportionately high

and adverse human health or environmental effects of their programs, policies, and activities on minority populations and low-income populations in the United States.

EPA has determined that this proposed rule will not have disproportionately high and adverse human health or environmental effects on minority or low-income populations because it increases the level of environmental protection for all affected populations without having any disproportionately high and adverse human health or environmental effects on any population, including any minority or low-income population. The proposed rule will establish uniform national standards for O₃ air pollution.

References

- Abbey, D. E.; Nishino, N.; McDonnell, W. F.; Burchette, R. J.; Knutsen, S. F.; Beeson, W. L.; Yang, J. X. (1999) Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *Am. J. Respir. Crit. Care Med.* 159: 373-382.
- Abt Associates Inc. (1995) Ozone NAAQS benefits analysis: California crops. Report to U.S. EPA, July 1995. EPA Docket No. A-95-58 Item II-I-3.
- Abt Associates Inc. (2007a) Ozone Health Risk Assessment for Selected Urban Areas. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC, July 2007; EPA report no. EPA-452/R-07-009. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Abt Associates Inc. (2007b) Technical Report on Ozone Exposure, Risk, and Impacts Assessments for Vegetation: Final Report. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC, January 2007; EPA report no. EPA-452/R-07-002. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Adams, W. C. (2002) Comparison of chamber and face-mask 6.6-hour exposures to ozone on pulmonary function and symptoms responses. *Inhalation Toxicol.* 14: 745-764.
- Adams, W. C. (2003a) Comparison of chamber and face mask 6.6-hour exposure to 0.08 ppm ozone via square-wave and triangular profiles on pulmonary responses. *Inhalation Toxicol.* 15: 265-281.
- Adams, W. C. (2003b) Relation of pulmonary responses induced by 6.6 hour exposures to 0.08 ppm ozone and 2-hour exposures to 0.30 ppm via chamber and face-mask inhalation. *Inhalation Toxicol.* 15: 745-759.
- Adams, W. C. (2006) Comparison of chamber 6.6 hour exposures to 0.04-0.08 ppm ozone via square-wave and triangular

- profiles on pulmonary responses. *Inhalation Toxicol.* 18: 127–136.
- Adams, W.C. (2007) Comment on EPA memorandum: The effects of ozone on lung function at 0.06 ppm in healthy adults. October 9. EPA–HQ–OAR–2005–0172–4783.
- American Academy of Pediatrics, Committee on Environmental Health. (2004) Ambient air pollution: health hazards to children. *Pediatrics* 114: 1699–1707.
- American Thoracic Society. (1985) Guidelines as to what constitutes an adverse respiratory health effect, with special reference to epidemiologic studies of air pollution. *Am. Rev. Respir. Dis.* 131: 666–668.
- American Thoracic Society (2000) What constitutes an adverse effect of air pollution? *Am. J. Respir. Crit. Care Med.* 161: pp. 665–673.
- Andersen, C. P.; Hogsett, W. E.; Wessling, R.; Plocher, M. (1991) Ozone decreases spring root growth and root carbohydrate content in ponderosa pine the year following exposure. *Can. J. For. Res.* 21: 1288–1291.
- Anderson, H. R.; Spix, C.; Medina, S.; Schouten, J. P.; Castellsague, J.; Rossi, G.; Zmirou, D.; Touloumi, G.; Wojtyniak, B.; Ponka, A.; Bacharova, L.; Schwartz, J.; Katsouyanni, K. (1997) Air pollution and daily admissions for chronic obstructive pulmonary disease in 6 European cities: results from the APHEA project. *Eur. Respir. J.* 10: 1064–1071.
- Arbaugh, M.; Bytnerowicz, A.; Grulke, N.; Fenn, M.; Poth, M.; Temple, P.; Miller, P. (2003) Photochemical smog effects in mixed conifer forests along a natural gradient of ozone and nitrogen deposition in the San Bernardino Mountains. *Environ. Int.* 29: 401–406.
- Arnold J.R.; R. L. Dennis; G. S. Tonnesen, (2003) Diagnostic evaluation of numerical air quality models with specialized ambient observations: testing the Community Multiscale Air Quality modeling system (CMAQ) at selected SOS 95 ground sites, *Atmos. Environ.* 37: 1185–1198.
- Ashmore, M.; Emberson, L.; Karlsson, P. E.; Pleijel, H. (2004) New directions: a new generation of ozone critical levels for the protection of vegetation in Europe (correspondence). *Atmos. Environ.* 38: 2213–2214.
- Arito, H.; Takahashi, M.; Iwasaki, T.; Uchiyama, I. (1997) Age-related changes in ventilatory and heart rate responses to acute ozone exposure in the conscious rat. *Ind. Health* 35: 78–86.
- Avol, E. L.; Gauderman, W. J.; Tan, S. M.; London, S. J.; Peters, J. M. (2001) Respiratory effects of relocating to areas of differing air pollution levels. *Am. J. Respir. Crit. Care Med.* 164: 2067–2072.
- Awmack, C. S.; Harrington, R.; Lindroth, R. L. (2004) Aphid individual performance may not predict population responses to elevated CO₂ or O₃. *Global Change Biol.* 10: 1414–1423.
- Barnes, J. D.; Eamus, D.; Brown, K. A. (1990) The influence of ozone, acid mist and soilnutrient status on Norway spruce [*Picea abies* (L.) Karst.]: II. photosynthesis, dark respiration and soluble carbohydrates of trees during late autumn. *New Phytol.* 115: 149–156.
- Bascom, R.; Naclerio, R. M.; Fitzgerald, T. K.; Kagey-Sobotka, A.; Proud, D. (1990) Effect of ozone inhalation on the response to nasal challenge with antigen of allergic subjects. *Am. Rev. Respir. Dis.* 142: 594–601.
- Basha, M. A.; Gross, K. B.; Gwizdala, C. J.; Haidar, A. H.; Popovich, J., Jr. (1994) Bronchoalveolar lavage neutrophilia in asthmatic and healthy volunteers after controlled exposure to ozone and filtered purified air. *Chest* 106: 1757–1765.
- Beedlow P.A., Tingey D.T., Phillips D.L., Hogsett W.E. & Olszyk D.M. (2004) Rising atmospheric CO₂ and carbon sequestration in forests. *Frontiers in Ecology and the Environment* 2, 315–322.
- Beeson, W. L.; Abbey, D. E.; Knutsen, S. F. (1998) Long-term concentrations of ambient air pollutants and incident lung cancer in California adults: results from the AHSMOG study. *Environ. Health Perspect.* 106: 813–823.
- Bell, M. L.; McDermott, A.; Zeger, S. L.; Samet, J. M.; Dominici, F. (2004) Ozone and short-term mortality in 95 US urban communities, 1987–2000. *JAMA J. Am. Med. Assoc.* 292: 2372–2378.
- Bell, M. L.; Dominici, F.; Samet, J. M. (2005) A meta-analysis of time-series studies of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study. *Epidemiology* 16: 436–445.
- Bell, M. L.; Peng, R. D.; Dominici, F. (2006) The exposure-response curve for ozone and risk of mortality and the adequacy of current ozone regulations. *Environ. Health Perspect.*: doi:10.1289/ehp.8816. Available online at: <http://dx.doi.org/> [23 January, 2006].
- Bosson, J.; Stenfors, N.; Bucht, A.; Helleday, R.; Pourazar, J.; Holgate, S. T.; Kelly, F. J.; Sandström, T.; Wilson, S.; Frew, A. J.; Blomberg, A. (2003) Ozone-induced bronchial epithelial cytokine expression differs between healthy and asthmatic subjects. *Clin. Exp. Allergy* 33: 777–782.
- Brauer, M.; Blair, J.; Vedal, S. (1995) Effect of ambient ozone exposure on lung function in farm workers. *Am. J. Respir. Crit. Care Med.* 154: 981–987.
- Brauer, M.; Blair, J.; Vedal, S. (1996) Effect of ambient ozone exposure on lung function in farm workers. *Am. J. Respir. Crit. Care Med.* 154: 981–987.
- Brauer, M.; Brook, J. R. (1997) Ozone personal exposures and health effects for selected groups residing in the Fraser Valley. In: Steyn, D. G.; Bottenheim, J. W., eds. *The Lower Fraser Valley Oxidants/Pacific '93 Field Study*. *Atmos. Environ.* 31: 2113–2121.
- Black, V. J.; Black, C. R.; Roberts, J. A.; Stewart, C. A. (2000) Impact of ozone on the reproductive development of plants. *New Phytol.* 147: 421–447.
- Brook, R. D.; Brook, J. R.; Urch, B.; Vincent, R.; Rajagopalan, S.; Silverman, F. (2002) Inhalation of fine particulate air pollution and ozone causes acute arterial vasoconstriction in healthy adults. *Circulation* 105: 1534–1536.
- Brown, J. S. The effects of ozone on lung function at 0.06 ppm in healthy adults. June 14, 2007. Memo to the Ozone NAAQS Review Docket. EPA–HQ–OAR–2005–0172–0175. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Burnett, R. T.; Dales, R. E.; Raizenne, M. E.; Krewski, D.; Summers, P. W.; Roberts, G. R.; Raad-Young, M.; Dann, T.; Brook, J. (1994) Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals. *Environ. Res.* 65: 172–194.
- Burnett, R. T.; Brook, J. R.; Yung, W. T.; Dales, R. E.; Krewski, D. (1997a) Association between ozone and hospitalization for respiratory diseases in 16 Canadian cities. *Environ. Res.* 72: 24–31.
- Burnett, R. T.; Cakmak, S.; Brook, J. R.; Krewski, D. (1997b) The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. *Environ. Health Perspect.* 105: 614–620.
- Burns, R. M., Honkala, B. H., tech. coords. (1990) *Silvics of North America: 1. Conifers; 2. Hardwoods*. *Agriculture Handbook* 654. U.S. Department of Agriculture, Forest Service, Washington, DC. vol.2, p. 877.
- Byun, D.W., Ching, J.K.S. (Eds.), 1999. *Science Algorithms of the EPA Models-3 Community Multiscale Air Quality Model (CMAQ) Modeling System*. EPA/600/R-99/030, U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC 20460.
- Campbell, S.; Temple, P.; Pronos, J.; Rochefort, R.; Andersen, C. (2000) Monitoring for ozone injury in west coast (Oregon, Washington, California) forests in 1998. Portland, OR: U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station; general technical report no. PNW–GTR–495. Available: <http://www.fs.fed.us/pnw/gtrs.htm> [11 April, 2003].
- Centers for Disease Control and Prevention. (2004) The health consequences of smoking: a report of the Surgeon General. Atlanta, GA: U.S. Department of Health and Human Services, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. Available: http://www.cdc.gov/tobacco/sgr/sgr_2004/chapters.htm (18 August, 2004).
- Chappelka, A. H. (2002) Reproductive development of blackberry (*Rubus cuneifolius*) as influenced by ozone. *New Phytol.* 155: 249–255.
- Chappelka, A. H.; Samuelson, L. J. (1998) Ambient ozone effects on forest trees of the eastern United States: a review. *New Phytol.* 139: 91–108.
- Chen, L.; Jennison, B. L.; Yang, W.; Omaye, S. T. (2000) Elementary school absenteeism and air pollution. *Inhalation Toxicol.* 12: 997–101.
- Chen, C.-Y.; Bonham, A. C.; Plopper, C. G.; Joad, J. P. (2003) Plasticity in respiratory motor control: selected contribution:

- neuroplasticity in nucleus tractus solitarius neurons following episodic ozone exposure in infant primates. *J. Appl. Physiol.* 94: 819–827.
- Clean Air Scientific Advisory Committee (CASAC) (2006) Transcript of Public Meeting Held in Research Triangle Park, N.C. on August 24, 2006.
- Cox, W. M.; Camalier, L. (2006) The effect of measurement error on 8-hour ozone design concentrations. Memo to the Ozone NAAQS Review Docket. EPA–HQ–OAR–2005–0172–0026. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Coulston, J. W., Smith, G. C. and Smith, W. D. (2003) “Regional assessment of ozone sensitive tree species using bioindicator plants.” *Environmental Monitoring and Assessment* 83: 113–127.
- Coulston, J. W., K. H. Riitters and G. C. Smith (2004) A Preliminary Assessment of the Montréal Process Indicators of Air Pollution for the United States. *Environmental Monitoring and Assessment* 95: 57–74.
- Dann, M. S.; Pell, E. J. (1989) Decline of activity and quantity of ribulose biphosphatecarboxylase/oxygenase and net photosynthesis in ozone-treated potato foliage. *PlantPhysiol.* 91: 427–432.
- David, G. L.; Romieu, I.; Sienra-Monge, J. J.; Collins, W. J.; Ramirez-Aguilar, M.; Del Rio-Navarro, B. E.; Reyes-Ruiz, N. I.; Morris, R. W.; Marzec, J. M.; London, S. J. (2003) Nicotinamide adenine dinucleotide (phosphate) reduced:quinone oxidoreductase and glutathione s-transferase m1 polymorphism and childhood asthma. *Am. J. Respir. Crit. Care Med.* 168: 1199–1204.
- Davison, A. W.; Reiling, K. (1995) A rapid change in ozone resistance of *Plantago* major after summers with high ozone concentrations. *New Phytol.* 131: 337–344.
- Devlin, R. B.; McDonnell, W. F.; Koren, H. S.; Becker, S. (1990) Prolonged exposure of humans to 0.10 and 0.08 ppm ozone results in inflammation in the lung. Presented at: 83rd annual meeting of the Air & Waste Management Association; June; Pittsburgh, PA. Pittsburgh, PA: Air & Waste Management Association; paper no. 90–150.2.
- Devlin, R. B.; McDonnell, W. F.; Mann, R.; Becker, S.; House, D. E.; Schreinemachers, D.; Koren, H. S. (1991) Exposure of humans to ambient levels of ozone for 6.6 hrs causes cellular and biochemical changes in the lung. *Am. J. Respir. Cell Mol. Biol.* 4: 72–81.
- Dickson, R. E., Lewin K. F., Isebrands J. G., Coleman M. D., Heilman W. E., Riemenschneider D. E., Sober J., Host G. E., Zak D. F., Hendrey G. R., Pregitzer K. S. and Karnosky D. F. (2000) Forest atmosphere carbon transfer storage-II (FACTS II)—The aspen free-air CO₂ and O₃ enrichment (FACE) project in an overview. USDA Forest Service North Central Research Station. General Tech. Rep. NC–214. 68pp.
- Dietert, R. R.; Etzel, R. A.; Chen, D.; Halonen, M.; Holladay, S. D.; Jarabek, A. M.; Landreth, K.; Peden, D. B.; Pinkerton, K.; Smialowicz, R. J.; Zoetis, T. (2000) Workshop to identify critical window of exposure for children’s health: immune and respiratory systems work group summary. *Environ. Health Perspect. Suppl.* 108(3): 483–490.
- Dockery, D. W.; Pope, C. A., III; Xu, X.; Spengler, J. D.; Ware, J. H.; Fay, M. E.; Ferris, B. G., Jr.; Speizer, F. E. (1993) An association between air pollution and mortality in six U.S. cities. *N. Engl. J. Med.* 329: 1753–1759.
- Dominici, F.; McDermott, A.; Daniels, M.; Zeger, S. L.; Samet, J. M. (2003) Mortality among residents of 90 cities. In: Revised analyses of time-series studies of air pollution and health. Special report. Boston, MA: Health Effects Institute; pp. 9–24. Available: <http://www.healtheffects.org/Pubs/TimeSeries.pdf> [12 May, 2004].
- Duff, M., Horst, R. L., Johnson, T. R. (1998) “Quadratic Rollback: A Technique to Model Ambient Concentrations Due to Undefined Emission Controls.” Presented at the Air and Waste Management Annual Meeting. San Diego, California. June 14–18, 1998.
- Eder, B. and S. Yu, 2006: A performance evaluation of the 2004 release of Models-3 CMAQ. *Atmos. Environ.* 40: 4811–4824. Special issue on Model Evaluation: Evaluation of Urban and Regional Eulerian Air Quality Models.
- Environmental Protection Agency (1986) Air quality criteria for ozone and other photochemical oxidants. Research Triangle Park, NC: Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office; report nos. EPA–600/8–84–020aF–eF. 5v. Available from: NTIS, Springfield, VA; PB87–142949.
- Environmental Protection Agency (1996a) Air quality criteria for ozone and related photochemical oxidants. Research Triangle Park, NC: Office of Research and Development; EPA report no. EPA/600/AP–93/004aF–cF. 3v. Available from: NTIS, Springfield, VA; PB96–185582, PB96–185590, and PB96–185608. Available online at: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=2831>.
- Environmental Protection Agency (1996b) Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. OAQPS Staff Paper (Final) Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA/452/R–96–007. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_sp.html.
- Environmental Protection Agency (2002) Project Work Plan for Revised Air Quality Criteria for Ozone and Related Photochemical Oxidants. Research Triangle Park, NC: National Center for Environmental Assessment; EPA report no. NCEA–R–1068.
- Environmental Protection Agency (2004) The Ozone Report: Measuring Progress through 2003. EPA/454/K–04–001. Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- Environmental Protection Agency (2005a) Air Quality Criteria for Ozone and Related Photochemical Oxidants (First External Review Draft). Washington, DC: National Center for Environmental Assessment; EPA report no. EPA/600/R–05/004aA–cA. Available online at: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=114523>.
- Environmental Protection Agency (2005b) Air Quality Criteria for Ozone and Related Photochemical Oxidants (Second External Review Draft) Washington, DC: National Center for Environmental Assessment; EPA report no. EPA/600/R–05/004aB–cB. Available online at: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=137307>.
- Environmental Protection Agency (2005c) Review of the national ambient air quality standards for ozone: assessment of scientific and technical information. OAQPS staff paper (First Draft). Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA–452/D–05–002. Available online at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html.
- Environmental Protection Agency (2005d) Health Assessment Plan for Review of the National Ambient Air Quality Standards for Ozone. Office of Air Quality Planning and Standards, Research Triangle Park, NC. April 2005. Available electronically on the internet at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_pd.html.
- Environmental Protection Agency (2006a) Air Quality Criteria for Ozone and Related Photochemical Oxidants. (Final) Washington, DC: National Center for Environmental Assessment; EPA report no. EPA/600/R–05/004aB–cB. Available online at: <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=149923>.
- Environmental Protection Agency (2006b) Review of the national ambient air quality standards for ozone: assessment of scientific and technical information. OAQPS staff paper. (Second Draft). Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA–452/D–05–002. Available online at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html.
- Environmental Protection Agency (2007a) Review of the national ambient air quality standards for ozone: assessment of scientific and technical information. OAQPS staff paper. (Final) January 2007. Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA–452/R–07–003. Available online at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html.
- Environmental Protection Agency (2007b) Review of the national ambient air quality standards for ozone: assessment of scientific and technical information. OAQPS staff paper. (Updated Final) July 2007. Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA–452/R–07–007. Available online at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html.
- Environmental Protection Agency (2007c) Ozone Population Exposure Analysis for

- Selected Urban Areas. (Updated Final) July 2007. Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA-452/R-07-010. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Environmental Protection Agency (2008) Responses to Significant Comments on the 2007 Proposed Rule on the National Ambient Air Quality Standards for Ozone (July 11, 2007; 72 FR 37818). March 2008. Research Triangle Park, NC: Office of Air Quality Planning and Standards. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_rc.html.
- Environmental Protection Agency (2009) Provisional Assessment of Recent Studies on Health and Ecological Effects of Ozone Exposure. September 2009. Research Triangle Park: National Center for Environmental Assessment; EPA report no. EPA/600/R-09/101.
- Evans, M. J.; Fanucchi, M. V.; Baker, G. L.; Van Winkle, L. S.; Pantle, L. M.; Nishio, S. J.; Schelegle, E. S.; Gershwhin, L. J.; Miller, L. A.; Hyde, D. M.; Sannes, P. L.; Plopper, C. G. (2003) Atypical development of the tracheal basement membrane zone of infant rhesus monkeys exposed to ozone and allergen. *Am. J. Physiol.* 285: L931-L939.
- Felzer, B.; Kickligher, D.; Melillo, J.; Wang, C.; Zhuang, Q.; Prinn, R. (2004) Effects of ozone on net primary production and carbon sequestration in the conterminous United States using a biogeochemistry model. *Tellus B* 56 (3), 230-248.
- Fiore, A. M.; Jacob, D. J.; Bey, I.; Yantosca, R. M.; Field, B. D.; Fusco, A. C.; Wilkinson, J. G. (2002) Background ozone over the United States in summer: origin, trend, and contribution to pollution episodes. *J. Geophys. Res. (Atmos.)* 107(D15): 10.1029/2001JD000982.
- Fiore, A. M.; Jacob, D. J.; Liu, H.; Yantosca, R. M.; Fairlie, T. D.; Fusco, A. C.; Li, Q. (2003) Variability in surface ozone background over the United States: implications for Air Quality Policy. *J. of Geophysical Research*, 108(D24)19-1-19-12.
- Fiscus, E. L.; Reid, C. D.; Miller, J. E.; Heagle, A. S. (1997) Elevated CO₂ reduces O₃ flux and O₃-induced yield losses in soybeans: possible implications for elevated CO₂ studies. *J. Exp. Bot.* 48: 307-313.
- Folinsbee, L. J.; McDonnell, W. F.; Horstman, D. H. (1988) Pulmonary function and symptom responses after 6.6-hour exposure to 0.12 ppm ozone with moderate exercise. *JAPCA* 38: 28-35.
- Folinsbee, L. J.; Horstman, D. H.; Kehrl, H. R.; Harder, S.; Abdul-Salaam, S.; Ives, P. J. (1994) Respiratory responses to repeated prolonged exposure to 0.12 ppm ozone. *Am. J. Respir. Crit. Care Med.* 149: 98-105.
- Folinsbee, L. J.; Hazucha, M. J. (2000) Time course of response to ozone exposure in healthy adult females. *Inhalation Toxicol.* 12: 151-167.
- Foster, W. M.; Silver, J. A.; Groth, M. L. (1993) Exposure to ozone alters regional function and particle dosimetry in the human lung. *J. Appl. Physiol.* 75: 1938-1945.
- Foster, W. M.; Weinmann, G. G.; Menkes, E.; Macri, K. (1997) Acute exposure of humans to ozone impairs small airway function. *Ann. Occup. Hyg.* 41(suppl. 1): 659-666.
- Frampton, M. W.; Morrow, P. E.; Torres, A.; Cox, C.; Voter, K. Z.; Utell, M. J.; Gibb, F. R.; Speers, D. M. (1997) Ozone responsiveness in smokers and nonsmokers. *Am. J. Respir. Crit. Care Med.* 155: 116-121.
- Galizia, A.; Kinney, P. L. (1999) Long-term residence in areas of high ozone: associations with respiratory health in a nationwide sample of nonsmoking young adults. *Environ. Health Perspect.* 107: 675-679.
- Gauderman, W. J.; McConnell, R.; Gilliland, F.; London, S.; Thomas, D.; Avol, E.; Vora, H.; Berhane, K.; Rappaport, E. B.; Lurmann, F.; Margolis, H. G.; Peters, J. (2000) Association between air pollution and lung function growth in southern California children. *Am. J. Respir. Crit. Care Med.* 162: 1383-1390.
- Gauderman, W. J.; Gilliland, G. F.; Vora, H.; Avol, E.; Stram, D.; McConnell, R.; Thomas, D.; Lurmann, F.; Margolis, H. G.; Rappaport, E. B.; Berhane, K.; Peters, J. M. (2002) Association between air pollution and lung function growth in southern California children: results from a second cohort. *Am. J. Respir. Crit. Care Med.* 166: 76-84.
- Gauderman, W. J.; Avol, E.; Gilliland, F.; Vora, H.; Thomas, D.; Berhane, K.; McConnell, R.; Kuenzli, N.; Lurmann, F.; Rappaport, E.; Margolis, H.; Bates, D.; Peters, J. (2004a) The effect of air pollution on lung development from 10 to 18 years of age. *N. Engl. J. Med.* 351: 1057-1067.
- Gauderman, W. J.; Avol, E.; Gilliland, F. (2004b) Air pollution and lung function [reply letter]. *N. Engl. J. Med.* 351: 2653.
- Gent, J. F.; Triche, E. W.; Holford, T. R.; Belanger, K.; Bracken, M. B.; Beckett, W. S.; Leaderer, B. P. (2003) Association of low-level ozone and fine particles with respiratory symptoms in children with asthma. *JAMA J. Am. Med. Assoc.* 290: 1859-1867.
- Gilliland, F. D.; Berhane, K.; Rappaport, E. B.; Thomas, D. C.; Avol, E.; Gauderman, W. J.; London, S. J.; Margolis, H. G.; McConnell, R.; Islam, K. T.; Peters, J. M. (2001) The effects of ambient air pollution on school absenteeism due to respiratory illnesses. *Epidemiology* 12: 43-54.
- Goldstein, A. H.; Millet, D. B.; McKay, M.; Jaegle, L.; Horowitz, L.; Cooper, O.; Hudman, R.; Jacob, D.; Oltmans, S.; Clarke, A. (2004) Impact of Asian emissions on observations at Trinidad Head, California, during ITCT 2K2. *J. of Geophysical Research*, 109(D23S17), doi: 10.1029/2003JD004406.
- Gong, H., Jr.; Wong, R.; Sharma, R. J.; Linn, W. S.; Sullivan, E. D.; Shamo, D. A.; Anderson, K. R.; Prasad, S. B. (1998) Cardiovascular effects of ozone exposure in human volunteers. *Am. J. Respir. Crit. Care Med.* 158: 538-546.
- Goodale, C. L., Apps, M. J., Birdsey, R. A., Field, C. B., Heath, L. S., Houghton, R. A., Jenkins, J. C., Kohlmaier, G. H., Kurz, W., Liu, S., Nabuurs, G.-J., Nilsson, S. and Shvidenko, A. Z. (2002) Forest carbon sinks in the northern hemisphere. *Ecol. Appl.* 12, 891-899.
- Grantz, D.A., McCool, P.H. (1992) Effect of ozone on Pima and Acala cottons in the San Joaquin Valley. In: Herber, D.J., Richter, D.A. (Eds.), *Proceedings 1992 Beltwide Cotton Conferences*, vol. 3. National Cotton Council of America, Memphis, TN, pp. 1082-1084.
- Greer, J.R.; Abbey, D.E.; Burchette, R.J. (1993) Asthma related to occupational and ambient air pollutants in nonsmokers. *J. Occup. Med.* 35: 909-915.
- Gregg, J.W.; Jones, C.G.; Dawson, T.E. (2003) Urbanization effects on tree growth in the vicinity of New York City. *Nature* 424: 183-187.
- Gulke, N.E.; Andersen, C.P.; Fenn, M.E.; Miller, P.R. (1998) Ozone exposure and nitrogen deposition lowers root biomass of ponderosa pine in the San Bernardino Mountains, California. *Environ. Pollut.* 103: 63-73.
- Gulke, N.E.; Balduman, L. (1999) Deciduous conifers: high N deposition and O₃ exposure effects on growth and biomass allocation in ponderosa pine. *Water Air Soil Pollut.* 116: 235-248.
- Gryparis, A.; Forsberg, B.; Katsouyanni, K.; Analitis, A.; Touloumi, G.; Schwartz, J.; Samoli, E.; Medina, S.; Anderson, H.R.; Niciu, E. M.; Wichmann, H.-E.; Kriz, B.; Kosnik, M.; Skorkovsky, J.; Vonk, J.M.; Dörtbudak, Z. (2004) Acute effects of ozone on mortality from the "air pollution and health: a European approach" project. *Am. J. Respir. Crit. Care Med.* 170: 1080-1087.
- Guderian, R. (1977) Discussion of the suitability of plant responses as a basis for air pollution control measures. In: Billings, W.D.; Golley, F.; Lange, O.L.; Olson, J.S., eds. *Air pollution: phytotoxicity of acidic gases and its significance in air pollution control*. Berlin, Federal Republic of Germany: Springer Verlag; pp. 75-97.
- Hanson, P., Samuelson, L., Wullschlegel, S., Tabberer, T.; Edwards, G. (1994) "Seasonal patterns of light-saturated photosynthesis and leaf conductance for mature and seedling *Quercus rubra* L. foliage: differential sensitivity to ozone exposure." *Tree Physiology* 14:1351-1366.
- Hazucha, M.J.; Folinsbee, L.J.; Seal, E., Jr. (1992) Effects of steady-state and variable ozone concentration profiles on pulmonary function. *Am. Rev. Respir. Dis.* 146: 1487-1493.
- Hazucha, M.J.; Folinsbee, L.J.; Bromberg, P. A. (2003) Distribution and reproducibility of spirometric response to ozone by gender and age. *J. Appl. Physiol.* 95: 1917-1925.
- Heck, W.W.; Cowling, E.B. (1997) The need for a long term cumulative secondary ozone standard—an ecological perspective. *EM (January)*: 23-33.

- Henderson, R. (2006a) Letter from CASAC Chairman Rogene Henderson to EPA Administrator Stephen Johnson. February 16, 2006, EPA-CASAC-06-003.
- Henderson, R. (2006b) Letter from CASAC Chairman Rogene Henderson to EPA Administrator Stephen Johnson. June 5, 2006, EPA-CASAC-06-007.
- Henderson, R. (2006c) Letter from CASAC Chairman Rogene Henderson to EPA Administrator Stephen Johnson. October 24, 2006, EPA-CASAC-07-001.
- Henderson, R. (2007) Letter from CASAC Chairman Rogene Henderson to EPA Administrator Stephen Johnson. March 26, 2007, EPA-CASAC-07-002.
- Henderson, R. (2008) Letter from CASAC Chairman Rogene Henderson to EPA Administrator Stephen Johnson. April 7, 2008, EPA-CASAC-08-009.
- Hill, A.B. (1965) The environment and disease: association or causation? *Proc. R. Soc. Med.* 58: 295-300.
- Hiltermann, J.T.N.; Lapperre, T.S.; Van Bree, L.; Steerenberg, P.A.; Brahim, J.J.; Sont, J.K.; Sterk, P.J.; Hiemstra, P.S.; Stolk, J. (1999) Ozone-induced inflammation assessed in sputum and bronchial lavage fluid from asthmatics: a new noninvasive tool in epidemiologic studies on air pollution and asthma. *Free Radical Biol. Med.* 27: 1448-1454.
- Hoek, G. (2003) Daily mortality and air pollution in The Netherlands. In: Revised analyses of time-series studies of air pollution and health. Special report. Boston, MA: Health Effects Institute; pp. 133-142. Available: <http://www.healtheffects.org/Pubs/TimeSeries.pdf> [12 May, 2004].
- Hoek, G.; Brunekreef, B.; Verhoeff, A.; Van Wijnen, J.; Fischer, P. (2000) Daily mortality and air pollution in the Netherlands. *J. Air Waste Manage. Assoc.* 50: 1380-1389.
- Hoek, G.; Brunekreef, B.; Fischer, P.; Van Wijnen, J. (2001) The association between air pollution and heart failure, arrhythmia, embolism, thrombosis, and other cardiovascular causes of death in a time series study. *Epidemiology* 12: 355-357.
- Hogsett, W.E.; Tingey, D.T.; Hendricks, C.; Rossi, D. (1989) Sensitivity of western conifers to SO₂ and seasonal interaction of acid fog and ozone. In: Olson, R.K.; Lefohn, A.S., eds. Effects of air pollution on western forests [an A&WMA symposium; June; Anaheim, CA]. Air Pollution Control Association; pp. 469-491 (APCA transactions series: no. 16).
- Holton, M.K.; Lindroth, R.L.; Nordheim, E.V. (2003) Foliar quality influences tree-herbivore-parasitoid interactions: effects of elevated CO₂, O₃, and plant genotype. *Oecologia* 137: 233-244.
- Holz, O.; Mücke, M.; Paasch, K.; Böhme, S.; Timm, P.; Richter, K.; Magnussen, H.; Jörres, R.A. (2002) Repeated ozone exposures enhance bronchial allergen responses in subjects with rhinitis or asthma. *Clin. Exp. Allergy*. 32: 681-689.
- Höppe, P.; Praml, G.; Rabe, G.; Lindner, J.; Fruhmann, G.; Kessel, R. (1995) Environmental ozone field study on pulmonary and subjective responses of assumed risk groups. *Environ. Res.* 71: 109-121.
- Horst, R.; Duff, M. (1995). Concentration data transformation and the quadratic rollback methodology (Round 2, Revised). Unpublished memorandum to R. Rodríguez, U.S. EPA, June 8.
- Horstman, D.H.; Folinsee, L.J.; Ives, P.J.; Abdul-Salaam, S.; McDonnell, W.F. (1990) Ozone concentration and pulmonary response relationships for 6.6-hr exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm. *Am. Rev. Respir. Dis.* 142: 1158-1163.
- Horstman, D.H.; Ball, B.A.; Brown, J.; Gerrity, T.; Folinsee, L.J. (1995) Comparison of pulmonary responses of asthmatic and nonasthmatic subjects performing light exercise while exposed to a low level of ozone. *Toxicol. Ind. Health* 11: 369-385.
- Huang, Y.; Dominici, F.; Bell, M.L. (2005) Bayesian hierarchical distributed lag models for summer ozone exposure and cardio-respiratory mortality. *Environmetrics* 16: 547-562.
- Isebrands, J.G.; Dickson, R.E.; Rebbeck, J.; Karnosky, D.F. (2000) Interacting effects of multiple stresses on growth and physiological processes in northern forest trees. In: Mickler, R.A.; Birsdey, R.A.; Hom, J., eds. Responses of northern U.S. forests to environmental change. New York, NY: Springer-Verlag; pp. 149-180. (Ecological studies: v. 139).
- Isebrands, J.G.; McDonald, E.P.; Kruger, E.; Hendrey, G.; Percy, K.; Pregitzer, K.; Sober, J.; Karnosky, D.F. (2001) Growth responses of *Populus tremuloides* clones to interacting carbon dioxide and tropospheric ozone. *Environ. Pollut.* 115: 359-371.
- Ito, K.; De Leon, S.F.; Lippmann, M. (2005) Associations between ozone and daily mortality, analysis and meta-analysis. *Epidemiology* 16: 446-457.
- Jacobson, J.S. (1977) The effects of photochemical oxidants on vegetation. In: Ozon und Begleitstoffen im photochemischen Smog: das Kolloquium [Ozone and related substances in photochemical smog: the colloquium]; September 1976; Dusseldorf, Federal Republic of Germany. Dusseldorf, Federal Republic of Germany: VDI-Verlag GmbH; pp. 163-173. (VDI-Berichte nr. 270).
- Johnson, T. (1997) "Sensitivity of Exposure Estimates to Air Quality Adjustment Procedure." Letter to Harvey Richmond, Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina.
- Jörres, R.; Nowak, D.; Magnussen, H.; Speckin, P.; Koschky, S. (1996) The effect of ozone exposure on allergen responsiveness in subjects with asthma or rhinitis. *Am. J. Respir. Crit. Care Med.* 153: 56-64.
- Karnosky, D.F., Z.E. Gagnon, R.E. Dickson, M.D. Coleman, E.H. Lee, J.G. Isebrands, (1996) "Changes in growth, leaf abscission, biomass associated with seasonal tropospheric ozone exposures of *Populus tremuloides* clones and seedlings." *Can. J. For. Res.* 26: 23-37.
- Karnosky, D.F., B. Mankovska, K. Percy, R.E. Dickson, G.K. Podila, J. Sober, A. Noormets, G. Hendrey, M.D. Coleman, M. Kubiske, K.S. Pregitzer, and J.G. Isebrands (1999) "Effects of tropospheric O₃ on trembling aspen and interaction with CO₂: Results from an O₃-gradient and a FACE experiment." *J. Water, Air and Soil Pollut.* 116: 311-322.
- Karnosky, D.F.; Zak, D.R.; Pregitzer, K. S.; Awmack, C.S.; Bockheim, J.G.; Dickson, R.E.; Hendrey, G.R.; Host, G.E.; King, J.S.; Kopper, B.J.; Kruger, E.L.; Kubiske, M.E.; Lindroth, R. L.; Mattson, W.J.; McDonald, E.P. (2003) Tropospheric O₃ moderates responses of temperate hardwood forests to elevated CO₂: A synthesis of molecular to ecosystem results from the Aspen FACE project. *Funct. Ecol.* 17: 289-304.
- Karnosky, D.F., Pregitzer, K.S., Zak, D.R., Kubiske, M.E., Hendrey, G.R., Weinstein, D., Nosal, M. & Percy, K.E. (2005) Scaling ozone responses of forest trees to the ecosystem level in a changing climate. *Plant Cell Environ.* 28, 965-981.
- Kelly, F.J.; Dunster, C.; Mudway, I. (2003) Air pollution and the elderly: oxidant/antioxidant issues worth consideration. *Eur. Respir. J. Suppl.* 40: 70S-75S.
- Kim, S.-Y.; Lee, J.-T.; Hong, Y.-C.; Ahn, K.-J.; Kim, H. (2004) Determining the threshold effect of ozone on daily mortality: an analysis of ozone and mortality in Seoul, Korea, 1995-1999. *Environ. Res.* 94: 113-119.
- King, J.S., M.E. Kubiske, K.S. Pregitzer, G.R. Hendrey, E.P. McDonald, C.P. Giardina, V.S. Quinn, D.F. Karnosky. (2005) Tropospheric O₃ compromises net primary production in young stands of trembling aspen, paper birch and sugar maple in response to elevated atmospheric CO₂. *New Phytologist*. 168:623-636.
- Kinney, P.L.; Aggarwal, M.; Nikiforov, S.V.; Nadas, A. (1998) Methods development for epidemiologic investigations of the health effects of prolonged ozone exposure. Part III: an approach to retrospective estimation of lifetime ozone exposure using a questionnaire and ambient monitoring data (U.S. sites). Cambridge, MA: Health Effects Institute; research report no. 81; pp. 79-108.
- Korrick, S.A.; Neas, L.M.; Dockery, D.W.; Gold, D.R.; Allen, G.A.; Hill, L.B.; Kimball, K.D.; Rosner, B.A.; Speizer, F.E. (1998) Effects of ozone and other pollutants on the pulmonary function of adult hikers. *Environ. Health Perspect.* 106: 93-99.
- Koutrakis, P.; Suh, H.H.; Sarnat, J.A.; Brown, K.W.; Coull, B.A.; Schwartz, J. (2005) Characterization of particulate and gas exposures of sensitive subpopulations living in Baltimore and Boston. HEI Research Report 131.
- Kreit, J.W.; Gross, K.B.; Moore, T.B.; Lorenzen, T.J.; D'Arcy, J.; Eschenbacher, W.L. (1989) Ozone-induced changes in pulmonary function and bronchial responsiveness in asthmatics. *J. Appl. Physiol.* 66: 217-222.

- Krewski, D.; Burnett, R.T.; Goldberg, M.S.; Hoover, K.; Siemiatycki, J.; Jerrett, M.; Abrahamowicz, M.; White, W.H. (2000) Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of particulate air pollution and mortality. A special report of the Institute's particle epidemiology reanalysis project. Cambridge, MA: Health Effects Institute.
- Künzli, N.; Lurmann, F.; Segal, M.; Ngo, L.; Balmes, J.; Tager, I.B. (1997) Association between lifetime ambient ozone exposure and pulmonary function in college freshmen—results of a pilot study. *Environ. Res.* 72: 8–23.
- Langstaff, J. (2007) Analysis of Uncertainty in Ozone Population Exposure Modeling. January 31, 2007. Memo to the Ozone NAAQS Review Docket. EPA–HQ–OAR–2005–0172–0174. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Larson, J.L.; Zak, D.R.; Sinsabaugh, R.L. (2002) Extracellular enzyme activity beneath temperate trees growing under elevated carbon dioxide and ozone. *Soil Sci. Soc. Am. J.* 66: 1848–1856.
- Laurence, J.A., Kohut, R.J., Amundson, R.G., (1993). Use of TREGRO to simulate the effects of ozone on the growth of red spruce seedlings. *Forest Science.* 39: 453–464.
- Laurence, J.A.; Retzlaff, W.A.; Kern, J.S.; Lee, E.H.; Hogsett, W.E.; Weinstein, D.A. (2001) Predicting the regional impact of ozone and precipitation on the growth of loblolly pine and yellow poplar using linked TREGRO and ZELG models. *For. Ecol. Manage.* 146: 247–263.
- 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. EPA Docket No. A–95–58 Item II–1–265.
- Lefohn, A.S.; Runeckles, V.C.; Krupa, S.V.; Shadwick, D.S. (1989) Important considerations for establishing a secondary ozone standard to protect vegetation. *JAPCA* 39, pp. 1039–1045.
- Levy, J.I.; Chemerynski, S.M.; Sarnat, J.A. (2005) Ozone exposure and mortality, an empiric Bayes metaregression analysis. *Epidemiology* 16: 458–468.
- Linn, W.S.; Shamoo, D.A.; Anderson, K.R.; Peng, R.-C.; Avol, E.L.; Hackney, J.D.; Gong, H., Jr. (1996) Short-term air pollution exposures and responses in Los Angeles area schoolchildren. *J. Exposure Anal. Environ. Epidemiol.* 6: 449–472.
- Lipfert, F.W.; Perry, H.M., Jr.; Miller, J.P.; Baty, J.D.; Wyzga, R.E.; Carmody, S.E. (2000) The Washington University-EPRI veterans' cohort mortality study: preliminary results. In: Grant, L.D., ed. PM2000: particulate matter and health. *Inhalation Toxicol.* 12(suppl. 4): 41–73.
- Lipfert, F.W.; Perry, H.M., Jr.; Miller, J.P.; Baty, J.D.; Wyzga, R.E.; Carmody, S.E. (2003) Air pollution, blood pressure, and their long-term associations with mortality. *Inhalation Toxicol.* 15: 493–512.
- Long, S., Nelson, R.L., Ainsworth, L., Hollis, K., Mies, T., Morgan, P., Naidu, S., Ort, D.R., Webster, R., Zhu, X. Adapting Soybean To Current And Future Change In Atmospheric Composition. Do We Need More Than Field Selection Under Current Conditions. Cellular and Molecular Biology of Soybean Biennial Conference. (2002) p. 401. http://www.ars.usda.gov/research/publications/publications.htm?SEQ_NO_115=142752
- Loya W.M., Pregitzer K.S., Karberg N.J., King J.S. & Giardina C.P. (2003) Reduction of soil carbon formation by tropospheric ozone under elevated carbon dioxide. *Nature* 425, 705–707.
- Luethy-Krause, B.; Landolt, W. (1990) Effects of ozone on starch accumulation in Norway spruce (*Picea abies*). *Trees* 4: 107–110.
- Lyons, T.M.; Barnes, J.D.; Davison, A.W. (1997) Relationships between ozone resistance and climate in European populations of *Plantago major*. *New Phytol.* 136: 503–510.
- Manning, W.J.; Krupa, S.V. (1992) Experimental methodology for studying the effects of ozone on crops and trees. In: Lefohn, A.S., ed. Surface level ozone exposures and their effects on vegetation. Chelsea, MI: Lewis Publishers, Inc.; pp. 93–156.
- Mannino, D.M.; Ford, E.S.; Redd, S.C. (2003) Obstructive and restrictive lung disease and markers of inflammation: data from the Third National Health and Nutrition Examination. *Am. J. Med.* 114: 758–762.
- Marty, M. (2007a) Letter from CHPAC Chair to the Administrator. March 23. EPA–HQ–OAR–2005–0172–0105.
- Marty, M. (2007b) Letter from CHPAC Chair to the Administrator. September 4. EPA–HQ–OAR–2005–0172–2031.
- McBride, D.E.; Koenig, J.Q.; Luchtel, D.L.; Williams, P.V.; Henderson, W.R., Jr. (1994) Inflammatory effects of ozone in the upper airways of subjects with asthma. *Am. J. Respir. Crit. Care Med.* 149: 1192–1197.
- McCluney, L. (2007) Ozone 1-Hour to 8-Hour Ratios for the 2002–2004 Design Value Period. January 18, 2007. Memo to the Ozone NAAQS Review Docket. EPA–HQ–OAR–0172–0073.
- McConnell, R.; Berhane, K.; Gilliland, F.; London, S.J.; Islam, T.; Gauderman, W.J.; Avol, E.; Margolis, H.G.; Peters, J.M. (2002) Asthma in exercising children exposed to ozone: a cohort study. *Lancet* 359: 386–391.
- McDonnell, W.F.; Kehrl, H.R.; Abdul-Salaam, S.; Ives, P.J.; Folinsbee, L.J.; Devlin, R.B.; O'Neil, J.J.; Horstman, D.H. (1991) Respiratory response of humans exposed to low levels of ozone for 6.6 hours. *Arch. Environ. Health* 46: 145–150.
- McDonnell, W.F. (1996) Individual variability in human lung function responses to ozone exposure. *Environ. Toxicol. Pharmacol.* 2: 171–175.
- McDonnell, W.F.; Stewart, P.W.; Andreoni, S.; Seal, E., Jr.; Kehrl, H.R.; Horstman, D.H.; Folinsbee, L.J.; Smith, M.V. (1997) Prediction of ozone-induced FEV1 changes: effects of concentration, duration, and ventilation. *Am. J. Respir. Crit. Care Med.* 156: 715–722.
- McDonnell, W.F.; Abbey, D.E.; Nishino, N.; Lebowitz, M.D. (1999) Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the ahsmog study. *Environ. Res.* 80: 110–121.
- McLaughlin, S.B., Nosal, M., Wullschlegler, S.D., Sun, G. (2007a) Interactive effects of ozone and climate on tree growth and water use in a southern Appalachian forest in the USA. *New Phytologist* 174: 109–124.
- McLaughlin, S.B., Wullschlegler, S.D., Sun, G. and Nosal, M. (2007b) Interactive effects of ozone and climate on water use, soil moisture content and streamflow in a southern Appalachian forest in the USA. *New Phytologist* 174: 125–136.
- Michelson, P.H.; Dailey, L.; Devlin, R.B.; Peden, D.B. (1999) Ozone effects on the immediate-phase response to allergen in the nasal airways of allergic asthmatic subjects. *Otolaryngol. Head Neck Surg.* 120: 225–232.
- Miller, P.R.; McBride, J.R.; Schilling, S.L.; Gomez, A.P. (1989) Trend of ozone damage to conifer forests between 1974 and 1988 in the San Bernardino Mountains of southern California. In: Olson, R.K.; Lefohn, A.S., eds. Effects of air pollution on western forests [an A&WMA symposium; June; Anaheim, CA]. Air and Waste Management Association; pp. 309–323. (APCA transactions series, no. 16).
- Moldau, H.; Söber, J.; Söber, A. (1990) Differential sensitivity of stomata and mesophyll to sudden exposure of bean shoots to ozone. *Photosynthetica* 24: 446–458.
- Morgan, P.B.; Ainsworth, E.A.; Long, S.P. (2003) How does elevated ozone impact soybean? A meta-analysis of photosynthesis, growth and yield. *Plant Cell Environ.* 26: 1317–1328.
- Morgan, P.B., Bernacchi, C.J., Ort, D.R., Long, S.P. (2004) An in vivo analysis of the effect of season-long open-air elevation of ozone to anticipated 2050 levels on photosynthesis in soybean. *Plant Physiology* 135: 2348–2357.
- Mortimer, K.M.; Neas, L.M.; Dockery, D.W.; Redline, S.; Tager, I.B. (2002) The effect of air pollution on inner-city children with asthma. *Eur. Respir. J.* 19: 699–705.
- Mudway, I.S.; Kelly, F.J. (2004) An investigation of inhaled ozone dose and the magnitude of airway inflammation in healthy adults. *Am. J. Respir. Crit. Care Med.* 169: 1089–1095.
- Musselman, R.C.; Lefohn, A.S.; Massman, W.J.; Heath, R.L. (2006) A critical review and analysis of the use of exposure- and flux-based ozone indices for predicting vegetation effects. *Atmos. Environ.* 40: 1869–1888.
- National Association of Clean Air Agencies (NACAA) (2007) Letter and Comments Sent to Docket No. OAR–2005–0172 re: Proposed Rule—National Ambient Air Quality Standards for Ozone. Docket No. OAR–2005–0172–4274. October 9, 2007.

- NPS (2005) 2005 Annual Performance & Progress Report: Air Quality in National Parks. National Park Service. http://www2.nature.nps.gov/air/Pubs/pdf/gpra/Gpra2005_Report_03202006_Final.pdf
- National Park Service (NPS) Letter and Comments Sent to Docket No. OAR-2005-0172 re: Proposed Rule—National Ambient Air Quality Standards for Ozone. Docket No. OAR-2005-0172-4934. September 27, 2007.
- Navidi, W.; Thomas, D.; Langholz, B.; Stram, D. (1999) Statistical methods for epidemiologic studies of the health effects of air pollution. Cambridge, MA: Health Effects Institute; research report no. 86.
- Noormets, A.; Sober, A.; Pell, E.J.; Dickson, R.E.; Posila, G.K.; Sober, J.; Isebrands, J.G.; Karnosky, D.F. (2001) Stomatal and non-stomatal limitation to photosynthesis in two trembling aspen (*Populus tremuloides* Michx) clones exposed to elevated CO₂ and/or O₃. *Plant Cell Environ.* 24: 327–336.
- Ollinger, S.V., Aber, J.D., Reich, P.B. and Freuder, R.J. (2002) Interactive effects of nitrogen deposition, tropospheric ozone, elevated CO₂ and land use history on the carbon dynamics of northern hardwood forests. *Glob. Change Biol.* 8(6), 545–562.
- Olszyk, D., Bytnerowicz, A., Kats, G., Reagan, C., Hake, S., Kerby, T., Millhouse, D., Roberts, B., Anderson, C., Lee, H. (1993) Cotton yield losses and ambient ozone concentrations in California's San Joaquin Valley. *Journal of Environmental Quality* 22, 602–611.
- OMB (2005). Update of Statistical Area Definitions and Guidance on Their Uses. U.S. Office of Management and Budget, Bulletin No. 05–02. February 22, 2005.
- O'Neill, M.S.; Loomis, D.; Borja-Aburto, V.H. (2004) Ozone, area social conditions, and mortality in Mexico City. *Environ. Res.* 94: 234–242.
- Pearson, S.; Davison, A.W.; Reiling, K.; Ashenden, T.; Ollerenshaw, J.H. (1996) The effects of different ozone exposures on three contrasting populations of *Plantago major*. *New Phytol.* 132: 493–502.
- Peden, D.B.; Setzer, R.W., Jr.; Devlin, R.B. (1995) Ozone exposure has both a priming effect on allergen-induced responses and an intrinsic inflammatory action in the nasal airways of perennially allergic asthmatics. *Am. J. Respir. Crit. Care Med.* 151: 1336–1345.
- Peden, D.B.; Boehlecke, B.; Horstman, D.; Devlin, R. (1997) Prolonged acute exposure to 0.16 ppm ozone induces eosinophilic airway inflammation in asthmatic subjects with allergies. *J. Allergy Clin. Immunol.* 100: 802–808.
- Pell, E.J.; Schlagnhauser, C.D.; Arteca, R.N. (1997) Ozone-induced oxidative stress: mechanisms of action and reaction. *Physiol. Plant.* 100: 264–273.
- Percy, K.E.; Awmack, C.S.; Lindroth, R.L.; Kubiske, M.E.; Kopper, B.J.; Isebrands, J.G.; Pregitzer, K.S.; Hendry, G.R.; Dickson, R.E.; Zak, D.R.; Oksanen, E.; Sober, J.; Harrington, R.; Karnosky, D.F. (2002) Altered performance of forest pests under atmospheres enriched with CO₂ and O₃. *Nature (London)* 420: 403–407.
- Percy, K. E.; Nosal, M.; Heilman, W.; Dann, T.; Sober, J.; Legge, A. H.; Karnosky, D. F. (2007) New exposure-based metric approach for evaluating O₃ risk to North American aspen forests. *Environmental Pollution* 147:3 554–566.
- Peters, J. M.; Avol, E.; Navidi, W.; London, S. J.; Gauderman, W. J.; Lurmann, F.; Linn, W. S.; Margolis, H.; Rappaport, E.; Gong, H., Jr.; Thomas, D. C. (1999a) A study of twelve southern California communities with differing levels and types of air pollution. I. Prevalence of respiratory morbidity. *Am. J. Respir. Crit. Care Med.* 159: 760–767.
- Peters, J. M.; Avol, E.; Gauderman, W. J.; Linn, W. S.; Navidi, W.; London, S. J.; Margolis, H.; Rappaport, E.; Vora, H.; Gong, H., Jr.; Thomas, D. C. (1999b) A study of twelve southern California communities with differing levels and types of air pollution. II. Effects on pulmonary function. *Am. J. Respir. Crit. Care Med.* 159: 768–775.
- Peterson, D. L.; Arbaugh, M. J.; Wakefield, V. A.; Miller, P. R. (1987) Evidence of growth reduction in ozone-injured Jeffrey pine (*Pinus jeffreyi* Grev and Balf) in Sequoia and Kings Canyon National Parks. *JAPCA* 37: 906–912.
- Phillips, R. L.; Zak, D. R.; Holmes, W. E.; White, D. C. (2002) Microbial community composition and function beneath temperate trees exposed to elevated atmospheric carbon dioxide and ozone. *Oecologia* 131: 236–244.
- Plopper, C. G.; Fanucchi, M. V. (2000) Do urban environmental pollutants exacerbate childhood lung diseases? *Environ. Health Perspect.* 108: A252–A253.
- Plunkett, L. M.; Turnbull, D.; Rodricks, J. V. (1992) Differences between adults and children affecting exposure assessment. In: Guzelian, P. S.; Henry, D. J.; Olin, S. S., eds. Similarities and differences between children and adults: implications for risk assessment. Washington, DC: ILSI Press, pp. 79–96.
- Pope, C. A., III; Thun, M. J.; Namboodiri, M. M.; Dockery, D. W.; Evans, J. S.; Speizer, F. E.; Heath, C. W., Jr. (1995) Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. *Am. J. Respir. Crit. Care Med.* 151: 669–674.
- Pope, C. A., III; Burnett, R. T.; Thun, M. J.; Calle, E. E.; Krewski, D.; Ito, K.; Thurston, G. D. (2002) Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *JAMA J. Am. Med. Assoc.* 287: 1132–1141.
- Prentice IC, Farquhar GD, Fasham MJR, *et al.* (2001) The Carbon Cycle and Atmospheric Carbon Dioxide. In *Climate Change 2001: The Scientific Basis. Contribution of Working Group I to the Third Assessment Report of the Intergovernmental Panel on Climate Change.* (ed J. T. Houghton YD, D. J. Griggs, M. Noguier, P. J. van der Linden, X. Dai, K. Maskell, and C. A. Johnson), pp. 241–280. Cambridge University Press, Cambridge, United Kingdom and New York, NY, USA.
- Pronos, J.; Merrill, L.; Dahlsten, D. (1999) Insects and pathogens in a pollution-stressed forest. In: Miller, P. R.; McBride, J. R., eds. Oxidant air pollution impacts in the montane forests of southern California. Springer, pp. 317–337.
- Reid, C. D.; Fiscus, E. L. (1998) Effects of elevated [CO₂] and/or ozone on limitations to CO₂ assimilation in soybean (*Glycine max*). *J. Exp. Bot.* 49: 885–895.
- Reiling, K.; Davison, A. W. (1992a) Effects of a short ozone exposure given at different stages in the development of *Plantago major* L. *New Phytol.* 121: 643–647.
- Reiling, K.; Davison, A. W. (1992b) The response of native, herbaceous species to ozone: growth and fluorescence screening. *New Phytol.* 120: 29–37.
- Retzlaff, W. A.; Arthur, M. A.; Grulke, N. E.; Weinstein, D. A.; Gollands, B. (2000) Use of a single-tree simulation model to predict effects of ozone and drought on growth of a white fir tree. *Tree Physiol.* 20: 195–202.
- Rizzo, M (2005). Evaluation of a quadratic approach for adjusting distributions of hourly ozone concentrations to meet air quality standards. November 7, 2005. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Rizzo, M. (2006). A distributional comparison between different rollback methodologies applied to ambient ozone concentrations. August 23, 2006. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.
- Romieu, I.; Meneses, F.; Ruiz, S.; Sienna, J. J.; Huerta, J.; White, M. C.; Etzel, R. A. (1996) Effects of air pollution on the respiratory health of asthmatic children living in Mexico City. *Am. J. Respir. Crit. Care Med.* 154: 300–307.
- Romieu, I.; Meneses, F.; Ruiz, S.; Huerta, J.; Sienna, J. J.; White, M.; Etzel, R.; Hernandez, M. (1997) Effects of intermittent ozone exposure on peak expiratory flow and respiratory symptoms among asthmatic children in Mexico City. *Arch. Environ. Health* 52: 368–376.
- Romieu, I.; Sienna-Monge, J. J.; Ramírez-Aguilar, M.; Moreno-Macias, H.; Reyes-Ruiz, N. I.; Estela del Rio-Navarro, B.; Hernández-Avila, M.; London, S. J. (2004) Genetic polymorphism of GSTM1 and antioxidant supplementation influence lung function in relation to ozone exposure in asthmatic children in Mexico City. *Thorax* 59: 8–10.
- Sarnat, J. A.; Schwartz, J.; Catalano, P. J.; Suh, H. H. (2001) Gaseous pollutants in particulate matter epidemiology: confounders or surrogates? *Environ. Health Perspect.* 109: 1053–1061.
- Sarnat, J. A.; Brown, K. W.; Schwartz, J.; Coull, B. A.; Koutrakis, P. (2005) Ambient gas concentrations and personal particulate matter exposures: implications for studying the health effects of particles. *Epidemiology* 16: 385–395.

- Sarnat, J. A.; Coull, B. A.; Schwartz, J.; Gold, D. R.; Suh, H. H. (2006) Factors affecting the association between ambient concentrations and personal exposure to particles and gases. *Environ. Health Perspect.* 114(5): 649–654.
- Sasek, T. W.; Richardson, C. J.; Fendick, E. A.; Bevington, S. R.; Kress, L. W. (1991) Carryover effects of acid rain and ozone on the physiology of multiple flushes of loblolly pine seedlings. *For. Sci.* 37: 1078–1098.
- Scannell, C.; Chen, L.; Aris, R. M.; Tager, I.; Christian, D.; Ferrando, R.; Welch, B.; Kelly, T.; Balmes, J. R. (1996) Greater ozone-induced inflammatory responses in subjects with asthma. *Am. J. Respir. Crit. Care Med.* 154: 24–29.
- Schwartz, J. (2005) How sensitive is the association between ozone and daily deaths to control for temperature? *Am. J. Respir. Crit. Care Med.* 171: 627–631.
- Schildcrout, J. S.; Sheppard, L.; Lumley, T.; Slaughter, J. C.; Koenig, J. Q.; Shapiro, G. G. (2006) Ambient air pollution and asthma exacerbations in children: an eight city analysis. *Am. J. Epidemiol.* 164(5): 505–517.
- Schierhorn, K.; Hanf, G.; Fischer, A.; Umland, B.; Olze, H.; Kunkel, G. (2002) Ozone-induced release of neuropeptides from human nasal mucosa cells. *Int. Arch. Allergy Immunol.* 129: 145–151.
- Sheppard, L.; Slaughter, J. C.; Schildcrout, J.; Liu, L.-J. S.; Lumley, T. (2005) Exposure and measurement contributions to estimates of acute air pollution effects. *J. Exposure Anal. Environ. Epidemiol.* 15: 366–376.
- Simini, M.; Skelly, J. M.; Davis, D. D.; Savage, J. E.; Comrie, A. C. (1992) Sensitivity of four hardwood species to ambient ozone in north central Pennsylvania. *Can. J. For. Res.* 22: 1789–1799.
- Sin, D. D.; Man, S. F. P. (2003) Why are patients with chronic obstructive pulmonary disease at increased risk of cardiovascular diseases? *Circulation* 107: 1514–1519.
- Sitch, S.; Cox, P. M.; Collins, W. J.; Huntingford, C. (2007) Indirect radiative forcing of climate change through ozone effects on the land-carbon sink. *Nature (London, U.K.)* 448: 791–794.
- Smith, W. H. (1992) Air pollution effects on ecosystem processes. In: Barker, J. R.; Tingey, D. T., eds. *Air pollution effects on biodiversity*. Van Nostrand Reinhold; pp. 234–260.
- Smith, G.; Coulston J., Jepsen, J. and Prichard, T. (2003) “A national ozone biomonitoring program: Results from field surveys of ozone sensitive plants in northeastern forest (1994–2000)” *Environmental Monitoring and Assessment* 87(3): 271 – 291.
- Somers, G. L.; Chappelka, A. H.; Rosseau, P.; Renfro, J. R. (1998) Empirical evidence of growth decline related to visible ozone injury. *For. Ecol. Manage.* 104: 129–137.
- Tager, I. B.; Künzli, N.; Lurmann, F.; Ngo, L.; Segal, M.; Balmes, J. (1998) Methods development for epidemiologic investigations of the health effects of prolonged ozone exposure. Part II: an approach to retrospective estimation of lifetime ozone exposure using a questionnaire and ambient monitoring data (California sites). Cambridge, MA: Health Effects Institute; research report no. 81; pp. 27–78.
- Takemoto, B. K.; Bytnerowicz, A.; Fenn, M. E. (2001) Current and future effects of ozone and atmospheric nitrogen deposition on California’s mixed conifer forests. *For. Ecol. Manage.* 144: 159–173.
- Tans P.P., White J.W.C. (1998) In balance, with a little help from the plants. *Science*, 281, 183–184.
- Taylor, C.R. “AGSIM: Model Description and Documentation.” *Agricultural Sector Models for the United States*. C.R. Taylor, K.H. Reichelderfer, and S.R. Johnson, eds. Ames IA: Iowa State University Press, (1993).
- Taylor R. (1994) “Deterministic versus stochastic evaluation of the aggregate economic effects of price support programs” *Agricultural Systems* 44: 461–473.
- Temple, P. J.; Riechers, G. H.; Miller, P. R.; Lennox, R. W. (1993) Growth responses of ponderosa pine to longterm exposure to ozone, wet and dry acidic deposition, and drought. *Can. J. For. Res.* 23: 59–66.
- Thurston, G.D.; Ito, K.; Kinney, P.L.; Lippmann, M. (1992) A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. *J. Exposure Anal. Environ. Epidemiol.* 2:429–450.
- Tingey, D. T.; Standley, C.; Field, R. W. (1976) Stress ethylene evolution: a measure of ozone effects on plants. *Atmos. Environ.* 10: 969–974.
- Tingey, D. T.; Taylor, G. E., Jr. (1982) Variation in plant response to ozone: a conceptual model of physiological events. In: Unsworth, M. H.; Ormrod, D. P., eds. *Effects of gaseous air pollution in agriculture and horticulture*. London, United Kingdom: Butterworth Scientific; pp. 113–138.
- Tingey, D. T.; Laurence, J. A.; Weber, J. A.; Greene, J.; Hogsett, W. E.; Brown, S.; Lee, E. H. (2001) Elevated CO₂ and temperature alter the response of *Pinus ponderosa* to ozone: A simulation analysis. *Ecol. Appl.* 11: 1412–1424.
- Tingey, D. T.; Hogsett, W. E.; Lee, E. H.; Laurence, J. A. (2004) Stricter ozone ambient air quality standard has beneficial effect on *Ponderosa* pine in California. *Environ. Manage.* 34: 397–405.
- Touloumi, G.; Katsouyanni, K.; Zmirou, D.; Schwartz, J.; Spix, C.; Ponce de Leon, A.; Tobias, A.; Quennel, P.; Rabczenko, D.; Bacharova, L.; Bisanti, L.; Vonk, J. M.; Ponka, A. (1997) Short-term effects of ambient oxidant exposure on mortality: a combined analysis within the APHEA project. *Am. J. Epidemiol.* 146: 177–185.
- U.S. Department of Agriculture, 2006. The PLANTS Database (<http://plants.usda.gov>, December 2006). National Plant Data Center, Baton Rouge, LA.
- Ultman, J. S.; Ben-Jebria, A.; Arnold, S. F. (2004) Uptake distribution of ozone in human lungs: intersubject variability in physiologic response. Boston, MA: Health Effects Institute.
- Vagaggini, B.; Taccola, M.; Clanchetti, S.; Carnevali, S.; Bartoli, M. L.; Bacci, E.; Dente, F. L.; Di Franco, A.; Giannini, D.; Paggiaro, P. L. (2002) Ozone exposure increases eosinophilic airway response induced by previous allergen challenge. *Am. J. Respir. Crit. Care Med.* 166: 1073–1077.
- Vedal, S.; Brauer, M.; White, R.; Petkau, J. (2003) Air pollution and daily mortality in a city with low levels of pollution. *Environ. Health Perspect.* 111: 45–51.
- Vesely, D. L.; Giordano, A. T.; Raska-Emery, P.; Montgomery, M. R. (1994a) Ozone increases amino- and carboxy-terminal atrial natriuretic factor prohormone peptides in lung, heart, and circulation. *J. Biochem. Toxicol.* 9: 107–112.
- Vesely, D. L.; Giordano, A. T.; Raska-Emery, P.; Montgomery, M. R. (1994b) Increase in atrial natriuretic factor in the lungs, heart, and circulatory system owing to ozone. *Chest* 105: 1551–1554.
- Vesely, D. L.; Giordano, A. T.; Raska-Emery, P.; Montgomery, M. R. (1994c) Ozone increases atrial natriuretic peptides in heart, lung and circulation of aged vs. adult animals. *Gerontology (Basel)* 40: 227–236.
- Weber, J. A.; Clark, C. S.; Hogsett, W. E. (1993) Analysis of the relationship(s) among O₃ uptake, conductance, and photosynthesis in needles of *Pinus ponderosa*. *Tree Physiol.* 13: 157–172.
- Weinstein, D.A., Beloin, R.M., R.D. Yanai (1991) “Modeling changes in red spruce carbon balance and allocation in response to interacting ozone and nutrient stress.” *Tree Physiology* 9: 127–146.
- Weinstein, D.A., J.A. Laurence, W.A. Retzlaff, J.S. Kern, E.H. Lee, W.E. Hogsett, J. Weber (2005) Predicting the effects of tropospheric ozone on regional productivity of ponderosa pine and white fir. *Forest Ecology and Management* 205: 73–89.
- Whitfield, R.; Biller, W.; Jusko, M.; and Keisler, J. (1996) A Probabilistic Assessment of Health Risks Associated with Short- and Long-Term Exposure to Tropospheric Ozone. Argonne National Laboratory, Argonne, IL.
- Whitfield, R. (1997) A Probabilistic Assessment of Health Risks Associated with Short-term Exposure to Tropospheric Ozone: A Supplement. Argonne National Laboratory, Argonne, IL.
- Whitfield, C. P.; Davison, A. W.; Ashenden, T. W. (1997) Artificial selection and heritability of ozone resistance in two populations of *Plantago major*. *New Phytol.* 137: 645–655.
- Whitfield, R.G.; Richmond, H.M.; and Johnson, T.R. (1998) “Overview of Ozone Human Exposure and Health Risk Analyses Used in the U.S. EPA’s Review of the Ozone Air Quality Standard,” pp.483–516 in: T. Schneider, ed. *Air Pollution in the 21st Century: Priority Issues and Policy* Elsevier; Amsterdam.
- Wolff, G.T. (1995) Letter from Chairman of Clean Air Scientific Advisory Committee

to the EPA Administrator, dated November 30, 1995. EPA-SAB-CASAC-LTR-96-002.

Wolff, G.T. (1996) Letter from Chairman of Clean Air Scientific Advisory Committee to the EPA Administrator, dated April 4, 1996. EPA-SAB-CASAC-LTR-96-006.

Xu, X.; Ding, H.; Wang, X. (1995) Acute effects of total suspended particles and sulfur dioxides on preterm delivery: a community-based cohort study. Arch. Environ. Health 50: 407-415.

Young, T. F.; Sanzone, S., eds. (2002) A framework for assessing and reporting on ecological condition: an SAB report. Washington, DC: U.S. Environmental Protection Agency, Science Advisory Board; report no. EPA-SAB-EPEC-02-009. Available online at: [http://yosemite.epa.gov/sab/sabproduct.nsf/C3F89E598D843B58852570CA0075717E/\\$File/epec02009a.pdf](http://yosemite.epa.gov/sab/sabproduct.nsf/C3F89E598D843B58852570CA0075717E/$File/epec02009a.pdf).

Zeger, S. L.; Thomas, D.; Dominici, F.; Samet, J. M.; Schwartz, J.; Dockery, D.; Cohen, A. (2000) Exposure measurement error in time-series studies of air pollution: concepts and consequences. Environ. Health Perspect. 108: 419-426.

Zhang, L.-Y.; Levitt, R. C.; Kleeberger, S. R. (1995) Differential susceptibility to ozone-induced airways hyperreactivity in inbred strains of mice. Exp. Lung Res. 21: 503-518.

Zidek, J. V.; White, R.; Le, N. D.; Sun, W.; Burnett, R. T. (1998) Imputing unmeasured explanatory variables in environmental epidemiology with application to health impact analysis of air pollution. Environ. Ecol. Stat. 5: 99-115.

List of Subjects in 40 CFR Parts 50 and 58

Environmental protection, Air pollution control, Carbon monoxide,

Lead, Nitrogen dioxide, Ozone, Particulate matter, Sulfur oxides.

Dated: January 6, 2010.

Lisa P. Jackson,
Administrator.

For the reasons set forth in the preamble, parts 50 and 58 of chapter 1 of title 40 of the code of Federal regulations are proposed to be amended as follows:

PART 50—NATIONAL PRIMARY AND SECONDARY AMBIENT AIR QUALITY STANDARDS

1. The authority citation for part 50 continues to read as follows:

Authority: 42 U.S.C. 7401 *et seq.*

2. Section 50.15 is revised to read as follows:

§ 50.15 National primary and secondary ambient air quality standards for ozone.

(a) The level of the national 8-hour primary ambient air quality standard for O₃ is (0.060–0.070) parts per million (ppm), daily maximum 8-hour average, measured by a reference method based on Appendix D to this part and designated in accordance with part 53 of this chapter or an equivalent method designated in accordance with part 53 of this chapter.

(b) The 8-hour primary O₃ ambient air quality standard is met at an ambient air quality monitoring site when the average of the annual fourth-highest daily maximum 8-hour average O₃ concentration is less than or equal to

(0.060–0.070) ppm, as determined in accordance with appendix P to this part.

(c) The level of the national secondary ambient air quality standard for O₃ is a cumulative index value of (7–15) ppm-hours, measured by a reference method based on Appendix D to this part and designated in accordance with part 53 of this chapter or an equivalent method designated in accordance with part 53 of this chapter.

(d) The secondary O₃ ambient air quality standard is a seasonal standard expressed as a sum of weighted hourly concentrations, cumulated over the 12 hour daylight period from 8 a.m. to 8 p.m. local standard time, during the consecutive 3-month period within the O₃ monitoring season with the maximum index value. The secondary O₃ standard is met at an ambient air quality monitoring site when the annual maximum consecutive 3-month cumulative index value (W126) is less than or equal to (7–15) ppm-hours, as determined in accordance with appendix P to this part.

3. Section 50.14 is amended by adding entries for primary and secondary ozone standards to the end of Table 1 in paragraph (c)(2)(vi) to read as follows:

§ 50.14 Treatment of air quality monitoring data influenced by exceptional events.

- * * * * *
- (c) * * *
- (2) * * *
- (vi) * * *

TABLE 1—SCHEDULE FOR EXCEPTIONAL EVENT FLAGGING AND DOCUMENTATION SUBMISSION FOR DATA TO BE USED IN DESIGNATIONS DECISIONS FOR NEW NAAQS

NAAQS pollutant/ standard/(level)/ promulgation date	Air quality data collected for calendar year	Event flagging & initial description deadline	Detailed documentation submission deadline
* * * * *	* * * * *	* * * * *	* * * * *
Primary Ozone/8-Hr Standard (Level TBD)/promulgated by August 31, 2010.	2007–2009 2010	November 1, 2010 ^b 60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b	November 1, 2010. ^b 60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b
Secondary Ozone/(Level TBD) Alternative 2-year Schedule—to be Promulgated by August 31, 2010.	2008 2009–2010 2011	July 1, 2011 ^b July 1, 2011 ^b 60 Days after the end of the calendar quarter in which the event occurred or March 1, 2012, whichever occurs first. ^b	July 1, 2011. ^a July 1, 2011. ^b 60 Days after the end of the calendar quarter in which the event occurred or March 1, 2012, whichever occurs first. ^b
Secondary Ozone/(Level TBD)—Alternative Accelerated Schedule—to be promulgated by August 31, 2010.	2007–2009 2010	November 1, 2010 ^b 60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b	November 1, 2010. ^b 60 Days after the end of the calendar quarter in which the event occurred or March 1, 2011, whichever date occurs first. ^b

TABLE 1—SCHEDULE FOR EXCEPTIONAL EVENT FLAGGING AND DOCUMENTATION SUBMISSION FOR DATA TO BE USED IN DESIGNATIONS DECISIONS FOR NEW NAAQS—Continued

NAAQS pollutant/ standard/(level)/ promulgation date	Air quality data collected for calendar year	Event flagging & initial description deadline	Detailed documentation submission deadline
*	*	*	*

^a These dates are unchanged from those published in the original rulemaking.

^b Indicates change from general schedule in 40 CFR 50.14.

Note: EPA notes that the table of revised deadlines only applies to data EPA will use to establish the final initial designations for new NAAQS. The general schedule applies for all other purposes, most notably, for data used by EPA for redesignations to attainment.

4. Appendix P to part 50 is revised to read as follows:

Appendix P to Part 50—Interpretation of the Primary and Secondary National Ambient Air Quality Standards for Ozone

1. General

(a) This appendix explains the data handling conventions and computations necessary for determining whether the 8-hour primary and secondary national ambient air quality standards for ozone specified in § 50.15 are met at an ambient ozone air quality monitoring site. Ozone is measured in the ambient air by a reference method based on Appendix D of this part, as applicable, and designated in accordance with part 53 of this chapter, or by an equivalent method designated in accordance with part 53 of this chapter. Data reporting, data handling, and computation procedures to be used in making comparisons between reported ozone concentrations and the levels of the ozone standards are specified in the following sections.

(b) Whether to exclude, retain, or make adjustments to the data affected by exceptional events, including stratospheric ozone intrusion and other natural events, is determined by the requirements under §§ 50.1, 50.14 and 51.930.

(c) The terms used in this appendix are defined as follows:

8-hour average is the rolling average of eight hourly ozone concentrations as explained in section 3 of this appendix.

Annual fourth-highest daily maximum refers to the fourth-highest value measured at a monitoring site during a particular year.

Annual Cumulative W126 Index is the maximum sum over three consecutive calendar months of the monthly W126 index in a year, as explained in section 4 of this appendix.

Daily maximum 8-hour average concentration refers to the maximum calculated 8-hour average for a particular day as explained in section 3 of this appendix.

Daily W126 Index is the sum of the sigmoidally weighted hourly ozone concentrations during the 12-hour daylight period, 8 a.m. to 7:59 p.m. local standard time (LST).

Design values are the metrics (*i.e.*, statistics) that are compared to the primary and secondary NAAQS levels to determine compliance, calculated as shown in sections 3 and 4 of this appendix.

Monthly W126 Index is the sum of the daily W126 index over one calendar month

during the required ozone monitoring season, adjusted for incomplete data if appropriate, as explained in section 4 of this appendix.

Required ozone monitoring season refers to the span of time within a calendar year when individual States are required to measure ambient ozone concentrations as listed in part 58 Appendix D to this chapter.

Year refers to calendar year.

2. Requirements for Data Used for Comparisons With the Ozone NAAQS

(a) All valid FRM/FEM ozone data submitted to EPA's Air Quality System (AQS), or otherwise available to EPA, meeting the requirements of part 58 of this chapter including appendices A, C, and E shall be used in design value calculations.

(b) When two or more ozone monitors are operated at a site, the state may in advance designate one of them as the primary monitor. If the state has not made this designation, the Administrator will make the designation, either in advance or retrospectively. Design values will be developed using only the data from the primary monitor, if this results in a valid design value. If data from the primary monitor do not allow the development of a valid design value, data solely from the other monitor(s) will be used in turn to develop a valid design value, if this results in a valid design value. If there are three or more monitors, the order for such comparison of the other monitors will be determined by the Administrator. The Administrator may combine data from different monitors in different years for the purpose of developing a valid primary or secondary standard design value, if a valid design value cannot be developed solely with the data from a single monitor. However, data from two or more monitors in the same year at the same site will not be combined in an attempt to meet data completeness requirements, except if one monitor has physically replaced another instrument permanently, in which case the two instruments will be considered to be the same monitor, or if the state has switched the designation of the primary monitor from one instrument to another during the year.

(c) Hourly average concentrations shall be reported in parts per million (ppm) to the third decimal place, with additional digits to the right of the third decimal place truncated. The start of each hour shall be identified in local standard time (LST).

3. Comparison to the Primary Standard for Ozone

(a) Computing 8-Hour Averages

Running 8-hour averages shall be computed from the hourly ozone concentration data for each hour of the year and shall be stored in the first, or start, hour of the 8-hour period. In the event that only 6 or 7 hourly averages are available, the valid 8-hour average shall be computed on the basis of the hours available, using 6 or 7 as the divisor. In the event that only 1, 2, 3, 4, or 5 hourly averages are available, the 8-hour average shall be computed on the basis of substituting for all the hours without hourly averages a low hourly average value selected as follows, using 8 as the divisor. For days within the required ozone monitoring season, the substitution value shall be the lowest hourly average ozone concentration observed during the same hour (local standard time) of any day in the required ozone monitoring season of that year, or one-half of the method detection limit of the ozone instrument, whichever is higher. However, if the number of same-hour concentration values available for the required ozone monitoring season for the year, from which the lowest observed hourly concentration would be identified for purposes of this substitution, is less than 50% of the number of days during the required ozone monitoring season, one-half the method detection limit of the ozone instrument shall be used in the substitution. For days outside the required ozone monitoring season, the substitution value shall be one-half the method detection limit of the ozone instrument. An 8-hour period with no available hourly averages does not have a valid 8-hour average. The computed 8-hour average ozone concentrations are not rounded or truncated.

(b) Daily Maximum 8-Hour Average Concentrations

There are 24 8-hour periods in each calendar day. Some of these may not have valid 8-hour averages, under section 3(a). The daily maximum 8-hour concentration for a given calendar day is the highest of the valid 8-hour average concentrations computed for that day. This process is repeated, yielding a daily maximum 8-hour average ozone concentration for each day with ambient ozone monitoring data, including days outside the required ozone monitoring season if data are available. The daily maximum 8-hour concentrations from two consecutive days may have some hourly concentrations in common. Generally, overlapping daily maximum 8-hour averages are not likely,

except in those non-urban monitoring locations with less pronounced diurnal variation in hourly concentrations. In these cases, the maximum 8-hour average concentration from each day is used, even if the two averages have some hours in common.

(c) Primary Standard Design Value

The primary standard design value is the annual fourth-highest daily maximum 8-hour ozone concentration considering all days with monitoring data including any days outside the required ozone monitoring season, expressed in parts per million, averaged over three years. The 3-year average shall be computed using the three most recent, consecutive years of monitoring data that can yield a valid design value. For a design value to be valid for comparison to the standard, the monitoring data set on which it is based must meet the data completeness requirements described in section 3(d). The computed 3-year average of the annual fourth-highest daily maximum 8-hour average ozone concentrations shall be rounded to three decimal places. Values equal to or greater than 0.0xx5 ppm shall round up.

(d) Data Completeness Requirements for a Valid Design Value

(i) A design value greater than the standard is valid if in each of the three years there are

at least four days with a daily maximum 8-hour average concentration. Under sections 3(a) and 3(b), there will be a daily maximum 8-hour average concentration on any day with at least one hourly concentration. One or more of these four days may be outside the required ozone monitoring season.

(ii) A design value less than or equal to the standard is valid if for at least 75% of the days in the required ozone monitoring season in each of the three years there are at least 18 8-hour averages in the day that are based on at least 6 measured hourly average concentrations.

(iii) When computing whether the minimum data completeness requirement in section 3(d)(ii) has been met for the purpose of showing that a design value equal to or less than the standard is valid, meteorological or ambient data may be sufficient to demonstrate that ozone levels on days with missing data would not have affected the design value. At the request of the state, the Regional Administrator may consider demonstrations that meteorological conditions on one or more days in the required ozone monitoring season which do not have at least 18 8-hour averages in the day that are based on at least 6 measured hourly average concentrations could not have caused a daily maximum 8-hour concentration high enough to have been one

of the four highest daily maximum 8-hour concentrations for the year. At the request of the state, days so demonstrated may be counted towards the 75% requirement for the purpose of validating the design value, subject to the approval of the Regional Administrator.

(vi) Years that do not meet the completeness criteria stated in 3(d)(ii) may nevertheless be used to calculate a design value that will be deemed valid with the approval of, or at the initiative of, the Administrator, who may consider factors such as monitoring site closures/moves, monitoring diligence, the consistency and levels of the valid concentration measurements that are available, and nearby concentrations in determining whether to use such data.

(e) Comparison With the Primary Ozone Standard

(i) The primary ozone ambient air quality standard is met at an ambient air quality monitoring site when the design value is less than or equal to [0.075] ppm.

(ii) Comparison with the primary ozone standard is demonstrated by examples 1 and 2 as follows:

Example 1. Ambient monitoring site attaining the primary ozone standard.

Year	Percent valid days (within the required monitoring season)	1st Highest daily max 8-hour conc. (ppm)	2nd Highest daily max 8-hour conc. (ppm)	3rd Highest daily max 8-hour conc. (ppm)	4th Highest daily max 8-hour conc. (ppm)	5th Highest daily max 8-hour conc. (ppm)
2006	80	0.092500	0.090375	0.085125	0.078375	0.078125
2007	96	0.084750	0.083500	0.075375	0.071875	0.070625
2008	98	0.080875	0.079750	0.077625	0.075500	0.060375
Average					0.075250	
Rounded					0.075	

As shown in Example 1, this monitoring site meets the primary ozone standard because the 3-year average of the annual fourth-highest daily maximum 8-hour average ozone concentrations (*i.e.*, 0.075256 ppm, rounded to 0.075 ppm) is less than or

equal to [0.075] ppm. The data completeness requirement is also met because no single year has less than 75% data completeness. In Example 1, the individual 8-hour averages and the 3-year average are shown with six decimal digits. In actual calculations, all

digits supported by the calculator or calculation software must be retained.

Example 2. Ambient monitoring site failing to meet the primary ozone standard.

Year	Percent valid days (within the required monitoring season) (percent)	1st Highest daily max 8-hour conc. (ppm)	2nd Highest daily max 8-hour conc. (ppm)	3rd Highest daily max 8-hour conc. (ppm)	4th Highest daily max 8-hour conc. (ppm)	5th Highest daily max 8-hour conc. (ppm)
2006	96	0.105125	0.103500	0.101125	0.078625	0.072375
2007	74	0.104250	0.103625	0.093000	0.080250	0.069500
2008	98	0.103125	0.101875	0.101750	0.075375	0.074625
Average					0.078083	
Rounded					0.078	

As shown in Example 2, the data capture in 2007 is less than 75%. The primary ozone standard is not met for this monitoring site because the 3-year average of the fourth-

highest daily maximum 8-hour average ozone concentrations (*i.e.*, 0.078083 ppm, rounded to 0.078 ppm) is greater than [0.075] ppm and is therefore valid despite this

incompleteness. In Example 2, the individual 8-hour averages and the 3-year average are shown with six decimal digits. In actual calculations, all digits supported by the

calculator or calculation software must be retained.

4. Secondary Ambient Air Quality Standard for Ozone

(a) *Computing the daily W126 index value.*

The secondary ozone ambient air quality standard is a seasonal standard expressed as a sum of weighted hourly concentrations, cumulated over the 12 hour daylight period from 8 a.m. to 8 p.m. local standard time, during the consecutive 3-month period within the ozone monitoring season with the maximum index value. The first step in determining whether the standard is met at

a monitoring site is to compute the daily W126 index value for each day by applying the sigmoidal weighting function in Equation 1 to each reported measurement of hourly average concentration.

Equation 1

$$daily\ W126 = \sum_{i=8am}^{i<8pm} w_{ci} C_i$$

Where:

C_i = hourly O₃ at hour i, and

$$w_c = \frac{1}{1 + 4403e^{-126C}}$$

The computed value of the sigmoidally weighted hourly concentration is not rounded or truncated. The daily W126 index is formed by summing the twelve computed hourly values, retaining all decimal places. An illustration of computing a daily W126 index value is below:

Example 3. Daily W126 index value calculation for an ambient ozone monitoring site.

Start of hour	Concentration (ppm)	Weighted concentration (ppm)
8:00 a.m.	0.045	0.002781
9:00 a.m.	0.060	0.018218
10:00 a.m.	0.075	0.055701
11:00 a.m.	0.080	0.067537
12:00 p.m.	0.079	0.065327
1:00 p.m.	0.082	0.071715
2:00 p.m.	0.085	0.077394
3:00 p.m.	0.088	0.082448
4:00 p.m.	0.083	0.073683
5:00 p.m.	0.081	0.069667
6:00 p.m.	0.065	0.029260
7:00 p.m.	0.056	0.011676
Sum=Daily W126 index value	* 0.625406

* ppm-hours.

In Example 3, the individual weighted concentrations and their sum are shown with six decimal digits. In actual calculations, all digits supported by the calculator or calculation software must be retained. There are no data completeness requirements for the daily index. If fewer than 12 hourly values are available, only the available hours are weighted and summed. However, there are data completeness requirements for the monthly W126 index values and a required adjustment for incomplete data, as describe in the next section.

(b) *Computing the Monthly W126 Index*

As described in section 4(a), the daily index value is computed at each monitoring site for each calendar day in each month during the required ozone monitoring season with no rounding or truncation. The monthly W126 index is the sum of the daily index values over one calendar month. At an individual monitoring site, a monthly W126 index is valid if hourly average ozone concentrations are available for at least 75% of the possible daylight hours in the month. For months with more than 75% but less than 100% data completeness, the monthly W126 value shall be adjusted for incomplete data by multiplying the unadjusted monthly W126 index value by the ratio of the number of possible reporting hours to the number of hours with reported ambient hourly concentrations using Equation 2 in this appendix:

Equation 2

$$M.I. = \left[\sum_{j=1}^n (D.I.) \right] * (n * 12) / v$$

where

- M.I. = the adjusted monthly W126 index,
- D.I. = daily W126 index (i.e., the daily sum of the sigmoidally weighted daylight hourly concentrations),
- n = the number of days in the calendar month,
- v = the number of daylight reporting hours (8 a.m.–7:59 p.m. LST) in the month with reported valid hourly ozone concentrations.

The resulting adjusted value of the monthly W126 index shall not be rounded or truncated.

(c) Secondary Standard Design Value

The secondary standard design value is the 3-year average of the annual maximum consecutive 3-month sum of adjusted monthly W126 index values expressed in ppm-hours. Specifically, the annual W126 index value is computed on a calendar year basis using the three highest, consecutive adjusted monthly W126 index values. The 3-year average shall be computed using the most recent, consecutive three calendar years of monitoring data meeting the data completeness requirements described in section 4(c). The computed 3-year average of the annual maximum consecutive 3-month sum of adjusted monthly W126 index values in ppm-hours shall be rounded to a whole

number with decimal values equal to or greater than 0.500 rounding up.

(c) Data Completeness Requirement

(i) The annual W126 index is valid for purposes of calculating a 3-year design value if each full calendar month in the required ozone monitoring season has at least 75% data completeness for daylight hours.

(ii) If one or more months during the ozone monitoring seasons of three successive years has less than 75% data completeness, the three years shall nevertheless be used in the computation of a valid design value for the site if substituting the lowest ozone concentration observed during daylight hours in the required ozone monitoring season of each year, or one-half of the method detection limit of the ozone instrument, whichever is higher, for enough of the missing hourly concentrations within each incomplete month to make the month 75% complete, and then adjusting for the remaining missing data using Equation 2, above results in a design value greater than the level of the standard.

(d) Comparisons With the Secondary Ozone Standard

(i) The secondary ambient ozone air quality standard is met at an ambient air quality monitoring site when the design value is less than or equal to [15] ppm-hours.

(ii) Comparison with the secondary ozone standard is demonstrated by example 4 as follows:

Example 4. Ambient Monitoring Site Failing to Meet the Secondary Ozone Standard

	April	May	June	July	August	September	October	Overall
2006								
Adjusted monthly W126 index	4.442	9.124	12.983	16.153	13.555	4.364	1.302
3-Month sum	na	na	26.549	38.260	42.691	34.072	19.221
2006 Maximum	42.691	42.691
2007								
Adjusted monthly W126 index	3.114	7.214	8.214	8.111	7.455	7.331	5.115
3-Month sum	na	na	18.542	23.539	23.780	22.897	19.901
2007 Maximum	23.780	23.780
2008								
Adjusted monthly W126 index	4.574	5.978	6.786	8.214	5.579	4.331	2.115
3-Month sum	na	na	17.338	20.978	20.579	18.124	12.025
2008 Maximum	20.978	20.978
3-Year average W126 index	29.149666
Rounded	29

As shown in example 4, the secondary ozone standard is not met for this monitoring site because the 3-year average of the annual W126 index value for this site is greater than [15] ppm-hours:

$$3\text{-year average W126 index} = (42.691 + 23.780 + 20.978) / 3 = 29.149666, \text{ which rounds to } 29 \text{ ppm-hours.}$$

In Example 4, the adjusted monthly W126 index values and the 3-month sums of the adjusted monthly W126 index values are shown with three decimal digits. In actual calculations, all digits supported by the calculator or calculation software must be retained.

PART 58—AMBIENT AIR QUALITY SURVEILLANCE

5. The authority citation for part 58 continues to read as follows:

Authority: 42 U.S.C. 7410 7403, 7410, 7601(a), 7611, and 7619.

6. Section 58.50 is amended by revising paragraph (c) and adding paragraph (d) to read as follows:

§ 58.50 Index reporting.
* * * * *

(c) The population of a metropolitan statistical area for purposes of index reporting is the latest available U.S. census population.

(d) For O₃, reporting is required in metropolitan and micropolitan statistical areas wherever monitoring is required under Appendix D to Part 58—SLAMS Minimum O₃ Monitoring Requirements.

7. Appendix G of Part 58 is amended by revising section 3. to read as follows:

Appendix G to Part 58—Uniform Air Quality Index (AQI) and Daily Reporting

* * * * *

3. Must I Report the AQI?

You must report the AQI daily if yours is a metropolitan statistical area (MSA) with a population over 350,000. For O₃, reporting is required in metropolitan and micropolitan statistical areas wherever monitoring is required under Appendix D to Part 58—SLAMS Minimum O₃ Monitoring Requirements.

* * * * *

[FR Doc. 2010-340 Filed 1-15-10; 8:45 am]

BILLING CODE 6560-50-P