ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 50

[EPA–HQ–OAR–2005–0172; FRL–8331–5]

RIN 2060–AN24

National Ambient Air Quality Standards for Ozone

AGENCY: Environmental Protection Agency (EPA).

ACTION: Proposed rule.

SUMMARY: Based on its review of the air quality criteria for ozone (O_3) and related photochemical oxidants and national ambient air quality standards (NAAQS) for O_3, EPA proposes to make revisions to the primary and secondary NAAQS for O_3 to provide requisite protection of public health and welfare, respectively, and to make corresponding revisions in data handling conventions for O_3.

With regard to the primary standard for O_3, EPA proposes to revise the level of the 8-hour standard to a level within the range of 0.070 to 0.075 parts per million (ppm), to provide increased protection for children and other ''at risk'' populations against an array of O_3-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 nonaccidental and cardiopulmonary mortality. The EPA also proposes to specify the level of the primary standard to the nearest thousandth ppm. The EPA solicits comment on alternative levels down to 0.060 ppm and up to and including retaining the current 8-hour standard of 0.08 ppm (effectively 0.084 ppm using current data rounding conventions).

With regard to the secondary standard for O_3, EPA proposes to revise the current 8-hour standard with one of two options to provide increased protection against O_3-related adverse impacts on vegetation and forested ecosystems. One option is to replace the current standard with a cumulative, seasonal standard expressed as an index of the annual sum of weighted hourly concentrations, cumulated over 12 hours per day (8 a.m. to 8:00 p.m.) during the consecutive 3-month period within the O_3 season with the maximum index value, set at a level within the range of 7 to 21 ppm-hours. The other option is to make the secondary standard identical to the proposed primary 8-hour standard. The EPA solicits comment on specifying a cumulative, seasonal standard in terms of a 3-year average of the annual sums of weighted hourly concentrations; on the range of alternative 8-hour standard levels for which comment is being solicited for the primary standard, including retaining the current secondary standard, which is identical to the current primary standard; and on an alternative approach to setting a cumulative, seasonal secondary standard(s).

DATES: Written comments on this proposed rule must be received by October 9, 2007.

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

- *www.regulations.gov:* Follow the on-line instructions for submitting comments.
- *E-mail: a-and-r-Docket@epa.gov.*
- *Fax:* 202–566–1741.
- *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.

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. For additional information about EPA's public docket, visit the EPA Docket Center homepage at *http://www.epa.gov/epahome/dockets.htm.*

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.

Public Hearings: The EPA intends to hold public hearings around the end of August to early September in several cities across the country, and will announce in a separate **Federal Register** notice the dates, times, and addresses of the public hearings on this proposed rule.

FOR FURTHER INFORMATION CONTACT: Dr. David J. McKee, 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: 919–541–5288; fax: 919–541–0237; e-mail: *mckee.dave@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 *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.
- If you estimate potential costs or burdens, explain how you arrived at your estimate in sufficient detail to allow for it to be reproduced.
- 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 (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.'' The 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 Staff Paper is available at *http://www.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.* EPA will be making available corrected versions of the final Staff Paper and human exposure and health risk assessment technical support documents on these same EPA Web sites on or around July 16, 2007. These and other related documents are also available for inspection and copying in the EPA docket identified above.

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The following topics are discussed in this preamble:

I. Background

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 his judgment, may reasonably be anticipated to endanger public health and welfare'' and 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 identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in ambient air * * *.''

Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate ''primary'' and ''secondary'' NAAQS for pollutants listed under section 108. 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 [the] pollutant in the ambient air.'' [2]

[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-

Continued

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 (D.C. 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 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) (Breyer, J., concurring in part and concurring in judgment).

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. As discussed by Justice Breyer in *Whitman* v. *American Trucking Associations,* however, ''this interpretation of § 109 does not require the EPA to eliminate every health risk, however slight, at any economic cost, however great, to the point of ''hurtling'' industry over ''the brink of ruin,'' or even forcing ''deindustrialization.'' *Id.* at 494 (Breyer J., concurring in part and concurring in judgment) (*citations omitted*). Rather, as Justice Breyer explained:

> The statute, by its express terms, does not compel the elimination of *all* risk; and it grants the Administrator sufficient flexibility to avoid setting ambient air quality standards ruinous to industry.
>
> Section 109(b)(1) directs the Administrator to set standards that are ''requisite to protect the public health'' with ''an adequate margin of safety.'' But these words do not describe a world that is free of all risk—an impossible and undesirable objective. (*citation omitted*). Nor are the words ''requisite'' and ''public health'' to be understood independent of context. We consider football equipment ''safe'' even if its use entails a level of risk that would make drinking water ''unsafe'' for consumption. And what counts as ''requisite'' to protecting the public health will similarly vary with background circumstances, such as the public's ordinary tolerance of the particular health risk in the particular context at issue. The Administrator can consider such background circumstances when ''deciding what risks are acceptable in the world in which we live.'' (*citation omitted*).
>
> The statute also permits the Administrator to take account of comparative health risks. That is to say, she may consider whether a proposed rule promotes safety overall. A rule likely to cause more harm to health than it prevents is not a rule that is ''requisite to protect the public health.'' For example, as the Court of Appeals held and the parties do not contest, the Administrator has the authority to determine to what extent possible health risks stemming from reductions in tropospheric ozone (which, it is claimed, helps prevent cataracts and skin cancer) should be taken into account in setting the ambient air quality standard for ozone. (*citation omitted*).
>
> The statute ultimately specifies that the standard set must be ''requisite to protect the public health'' ''*in the judgment of the Administrator,*'' § 109(b)(1), 84 Stat. 1680 (emphasis added), a phrase that grants the Administrator considerable discretionary standard-setting authority.
>
> The statute's words, then, authorize the Administrator to consider the severity of a pollutant's potential adverse health effects, the number of those likely to be affected, the distribution of the adverse effects, and the uncertainties surrounding each estimate. (*citation omitted*). They permit the Administrator to take account of comparative health consequences. They allow her to take account of context when determining the acceptability of small risks to health. And they give her considerable discretion when she does so.
>
> This discretion would seem sufficient to avoid the extreme results that some of the industry parties fear. After all, the EPA, in setting standards that ''protect the public health'' with ''an adequate margin of safety,'' retains discretionary authority to avoid regulating risks that it reasonably concludes are trivial in context. Nor need regulation lead to deindustrialization. Preindustrial society was not a very healthy society; hence a standard demanding the return of the Stone Age would not prove ''requisite to protect the public health.''
>
> Although I rely more heavily than does the Court upon legislative history and alternative sources of statutory flexibility, I reach the same ultimate conclusion. Section 109 does not delegate to the EPA authority to base the national ambient air quality standards, in whole or in part, upon the economic costs of compliance.

Id. at 494–496.

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 NO_X and VOC emissions that contribute to O_3 formation in the United States come from three types of sources: mobile sources, industrial processes (which include consumer and commercial products), and the electric

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.''

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 O_3 season, which are considered natural background emissions.

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 O_3 by decreasing emissions of NO_X and VOC. These programs complement State and local efforts to improve O_3 air quality and meet current national standards. Under the Federal Motor Vehicle Control Program (FMVCP, see 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. Recently EPA proposed new standards for locomotive and marine diesel engines. 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. The EPA is currently working to finalize new federal rules, or amendments to existing rules, that will establish new nationwide VOC content limits for several categories of consumer and commercial products, including aerosol coatings, architectural and industrial maintenance coatings, and household and institutional commercial products. These rules will take effect in 2009, and will yield significant new reductions in nationwide VOC emissions—about 200,000 tons per year. Additionally, in O_3 nonattainment areas, we anticipate reductions of an additional 25,000 tons per year following completion of control technique recommendations for 3 additional consumer and commercial product categories. 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 and some large industrial boilers and turbines. The EPA's landmark Clean Air Interstate Rule (CAIR), issued on March 10, 2005, permanently caps power industry emissions of NO_X in the eastern United States. The first phase of the cap begins in 2009, and a lower second phase cap begins in 2015. By 2015, EPA projects that the CAIR and other programs in the Eastern U.S. will reduce power industry O_3 season NO_X emissions in that region by about 50 percent and annual NO_X emissions by about 60 percent from 2003 levels.

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 O_3 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 O_3 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 O_3 NAAQS.

C. Review of Air Quality Criteria and Standards for O_3

Tropospheric (ground-level) O_3 is formed from biogenic and anthropogenic precursor emissions. Naturally occurring O_3 in the troposphere can result from biogenic organic precursors reacting with naturally occurring nitrogen oxides (NO_X) and by stratospheric O_3 intrusion into the troposphere. Anthropogenic precursors of O_3, specifically NO_X and volatile organic compounds (VOC), originate from a wide variety of stationary and mobile sources. Ambient O_3 concentrations produced by these emissions are directly affected by temperature, solar radiation, wind speed and other meteorological factors.

The last review of the O_3 NAAQS was completed on July 18, 1997, based on the 1996 O_3 CD (U.S. EPA, 1996a) and 1996 O_3 Staff Paper (U.S. EPA, 1996b). EPA revised the primary and secondary O_3 standards on the basis of the then latest scientific evidence linking exposures to ambient O_3 to adverse health and welfare effects at levels allowed by the 1-hour average standards (62 FR 38856). The O_3 standards were revised by replacing the existing primary 1-hour average standard with an 8-hour average O_3 standard set at a level of 0.08 ppm, which is equivalent to 0.084 ppm using the standard rounding conventions. The form of the primary standard was changed to the annual fourth-highest daily maximum 8-hour average concentration, averaged over three years. The secondary O_3 standard was changed by making it identical in all respects to the revised primary standard.

Following promulgation of the revised O_3 NAAQS, petitions for review were

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

filed addressing a broad range of issues. In May 1999, in response to those challenges, the U.S. Court of Appeals for the District of Columbia Circuit held that EPA's approach to establishing the level of the standards in 1997, both for the O_3 and for the particulate matter (PM) NAAQS promulgated on the same day, effected ''an unconstitutional delegation of legislative authority.'' *American Trucking Associations* v. *EPA,* 175 F.3d 1027 (DC Cir., 1999). Although the D.C. Circuit stated that ''factors EPA uses in determining the degree of public health concern associated with different levels of O_3 and PM are reasonable,'' it remanded the rule to EPA, stating that when EPA considers these factors for potential non-threshold pollutants ''what EPA lacks is any determinate criterion for drawing lines'' to determine where the standards should be set. *Id.* at 1034. Consistent with EPA's long-standing interpretation and DC Circuit precedent, the court also reaffirmed prior rulings holding that in setting the NAAQS, it is ''not permitted to consider the cost of implementing those standards.'' *Id.* at 1040–41. The DC Circuit further directed EPA to consider on remand the potential indirect beneficial health effects of O_3 pollution in shielding the public from the effects of solar ultraviolet (UV) radiation, as well as the direct adverse health effects of O_3 pollution.

Both sides filed cross appeals on the constitutional and cost issues to the United States Supreme Court, and the Court granted *certiorari.* On February 27, 2001, the U.S. Supreme Court issued a unanimous decision upholding the EPA's position on both the constitutional and the cost issues. *Whitman* v. *American Trucking Associations,* 531 U.S. at 464, 475–76. On the constitutional issue, the Court held that the statutory requirement that NAAQS be ''requisite'' to protect public health with an adequate margin of safety sufficiently guided EPA's discretion, affirming EPA's approach of setting standards that are neither more nor less stringent than necessary. The Supreme Court remanded the case to the D.C. Circuit for resolution of any remaining issues that had not been addressed by that Court's earlier decisions. *Id.* at 475–76. On March 26, 2002, the D.C. Circuit Court rejected all remaining challenges to the NAAQS, holding under traditional standard of review that EPA ''engaged in reasoned decision-making'' in setting the 1997 O_3 NAAQS. *Whitman* v. *American Trucking Associations,* 283 F.3d 355 (DC Cir. 2002).

In response to the DC Circuit Court's remand to consider the potential indirect beneficial health effects of O_3 in shielding the public from the effects of solar (UV) radiation, on November 14, 2001, EPA proposed to leave the 1997 8-hour NAAQS unchanged (66 FR 57267). After considering public comment on the proposed decision, EPA reaffirmed the 8-hour O_3 NAAQS set in 1997 (68 FR 614). Finally, on April 30, 2004, EPA issued an 8-hour implementation rule that, among other things, provided that the 1-hour O_3 NAAQS would no longer apply to areas one year after the effective date of the designation of those areas for the 8-hour NAAQS (69 FR 23966).[4] For most areas, the date that the 1-hour NAAQS no longer applied was June 15, 2005. (See 40 CFR 50.9 for details.)

The EPA initiated this current review in September 2000 with a call for information (65 FR 57810) for the development of a revised Air Quality Criteria Document for O_3 and Other Photochemical Oxidants (henceforth the ''Criteria Document''). A project work plan (U.S. EPA, 2002) for the preparation of the Criteria Document was released in November 2002 for CASAC and public review. 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, the CASAC offered final comments on all chapters of the Criteria Document (Henderson, 2006a), and the final Criteria Document (EPA, 2006a) was released on March 21, 2006. In a June 8, 2006 letter (Henderson, 2006b) to the Administrator, the CASAC offered additional advice to the Agency concerning chapter 8 of the final 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 and 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 Staff Paper (EPA, 2007) was released on January 31, 2007. Around the time of the release of the final Staff Paper in January 2007, EPA discovered a small error in the exposure model that when corrected resulted in slight increases in the human exposure estimates. 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.[5] 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 O_3 NAAQS.

The schedule for completion of this review is 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 current review within the period provided by statute.[6] The modified consent decree that governs this review, entered by the court on December 16, 2004, provides that EPA sign for publication notices of proposed and final rulemaking concerning its review of the O_3 NAAQS no later than March 28, 2007 and December 19, 2007, respectively. This 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.

This action presents the Administrator's proposed decisions on the review of the current primary and secondary O_3 standards. Throughout this preamble a number of conclusions, findings, and determinations proposed by the Administrator are noted. While

[4] On December 22, 2006, the D.C. Circuit vacated the April 30, 2004 implementation rule. *South Coast Air Quality Management District* v. *EPA*, 472 F.3d 882. In March 2007, EPA requested the Court to reconsider its decision.

[5] EPA plans to make available corrected versions of the final Staff Paper and the human exposure and health risk assessment technical support documents on or around July 16, 2007 on the EPA web site listed in the Availability of Related Information section of this notice.

[6] *American Lung Association* v. *Whitman* (No. 1:03CV00778, D.D.C. 2003).

they identify the reasoning that supports this proposal, they are not intended to be final or conclusive in nature. The EPA invites general, specific, and/or technical comments on all issues involved with this proposal, including all such proposed judgments, conclusions, findings, and determinations.

II. Rationale for Proposed Decision on the Primary Standard

This section presents the rationale for the Administrator's proposed decision to revise the existing 8-hour O_3 primary standard by lowering the level of the standard to within a range from 0.070 to 0.075 ppm, and to specify the standard to the nearest thousandth ppm (*i.e.*, to the nearest parts per billion). As discussed more fully below, this rationale is based on a thorough review, in the Criteria Document, of the latest scientific information on human health effects associated with the presence of O_3 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 Criteria Document and staff analyses of air quality, human exposure, and health risks, presented in the Staff Paper, upon which staff recommendations for revisions to the primary O_3 standard are based; (2) CASAC advice and recommendations, as reflected in discussions of drafts of the Criteria Document and Staff Paper at public meetings, in separate written comments, and in CASAC's letters to the Administrator; and (3) public comments received during the development of these documents, either in connection with CASAC meetings or separately.

In developing this rationale, EPA has drawn upon an integrative synthesis of the entire body of evidence, published through early 2006, on human health effects associated with the presence of O_3 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 O_3 (EPA, 2006a, chapter 8), and includes over one hundred epidemiologic studies conducted in the U.S., Canada, and many countries around the world.[7] In considering this evidence, EPA focuses on those health endpoints that have been demonstrated to be caused by exposure to O_3, or for which the Criteria Document judges associations with O_3 to be causal, likely causal, or for which the evidence is highly suggestive that O_3 contributes to the reported effects. This rationale also draws upon the results of quantitative exposure and risk assessments, discussed below in section II.B. Evidence- and exposure/risk-based considerations that form the basis for the Administrator's proposed decisions on the adequacy of the current standard and on the elements of the range of proposed alternative standards are discussed below in sections II.C and II.D, respectively.

Judgments made in the Criteria Document and Staff Paper about the extent to which relationships between various health endpoints and short-term exposures to ambient O_3 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 O_3, it is clear that human studies provide the most directly applicable information for determining causality because they are not limited 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 effects data under closely monitored conditions and can provide exposure-response relationships. Epidemiological data provide evidence of associations between ambient O_3 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 confounding variables (*e.g.*, other pollutants, temperature) and other factors affects the level of confidence that the health effects being investigated are attributable to O_3 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 O_3, 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 O_3-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, O_3 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 O_3 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 O_3, or for which the Criteria Document judges associations with O_3 to be causal, likely causal, or for which the evidence is highly suggestive that O_3 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 O_3-related physiological health effects would experience ''exposures of concern'' and specific health endpoints

[7] In its assessment of the epidemiological evidence judged to be most relevant to making decisions on the level of the O_3 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.

under varying air quality scenarios (*e.g.*, just meeting the current or alternative standards), as well as characterizations of the kind and degree of uncertainties inherent in such estimates.

In this review, the term ''exposures of concern'' is defined as personal exposures while at moderate or greater exertion to 8-hour average ambient O_3 levels at and above specific benchmark levels which represent exposure levels at which O_3-related health effects are known or can reasonably be inferred to occur in some individuals, as discussed below in section II.B.1.[8] EPA emphasizes 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 O_3 exposure levels. 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 O_3 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 O_3-related health effects that have been demonstrated in human clinical 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. These O_3-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 O_3 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 this review 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 O_3 levels, as well as risk reductions and remaining risks associated with just meeting the current and various alternative O_3 standards in a number of example urban areas. There were two parts to this risk assessment: one part was based on combining information from controlled human exposure studies with modeled population exposure, and the other part was based on combining information from community epidemiological studies with either monitored or adjusted ambient concentrations levels. This assessment not only provided estimates of the potential magnitude of O_3-related health effects, as well as a characterization of the uncertainties and variability inherent in such estimates. This assessment also provided insights into the distribution of risks and patterns of risk reductions associated with meeting alternative O_3 standards.

As discussed below, a substantial amount of new research has been conducted since the last review of the O_3 NAAQS, with important new information coming from epidemiologic studies as well as from controlled human exposure, toxicological, and dosimetric studies. The newly available research studies evaluated in the Criteria Document and the exposure and risk assessments presented in the 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 O_3, the review of this information has been extensive and deliberate. In the judgment of the Administrator, this intensive evaluation of the scientific evidence has provided an adequate basis for regulatory decision making. This review also provides important input to EPA's research plan for improving our future understanding of the effects of ambient O_3 at lower levels, especially in at-risk population groups.

A. Health Effects Information

This section outlines key information contained in the Criteria Document (chapters 4–8) and in the Staff Paper (chapter 3) on known or potential effects on public health which may be expected from the presence of O_3 in ambient air. The information highlighted here summarizes: (1) New information available on potential mechanisms for health effects associated with exposure to O_3; (2) the nature of effects that have been associated directly with exposure to O_3 and indirectly with the presence of O_3 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 O_3, including the identification of ''at risk'' subpopulations.

The decision in the last 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

[8] Exposures of concern were also considered in the last review of the O_3 NAAQS, and were judged by EPA to be an important indicator of the public health impacts of those O_3-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 to potential aggravation of chronic bronchitis or long-term damage to the lungs). (62 FR 38868)

hospital admissions and emergency department (ED) visits for respiratory causes. The Criteria Document prepared for this review emphasizes a 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 O_3 on lung function decrements and enhanced respiratory symptoms in asthmatic individuals, school absences, and premature mortality. It also emphasizes 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 O_3 exposures at levels below 0.080 ppm, which was the lowest exposure level that had been examined in the last review.

(2) Numerous controlled human-exposure studies have examined indicators of O_3-induced inflammatory response in both the upper respiratory tract (URT) and lower respiratory tract (LRT), while other studies have examined changes in host defense capability following O_3 exposure of healthy young adults and increased airway responsiveness to allergens in subjects with allergic asthma and allergic rhinitis exposed to O_3.

(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 O_3-related lung function decrements and respiratory symptoms in physically active healthy subjects and asthmatic subjects, as well as evidence on new health endpoints, such as the relationships between ambient O_3 concentrations and school absenteeism and between ambient O_3 and cardiac-related physiological endpoints.

(5) Several additional studies have been published over the last decade examining the temporal associations between O_3 exposures and emergency department visits for respiratory diseases and on respiratory-related hospital admissions.

(6) A large number of newly available epidemiological studies have examined the effects of acute exposure to PM and O_3 on mortality, notably including large multicity studies that provide much more robust and credible information than was available in the last review, as well as recent meta-analyses that have evaluated potential sources of heterogeneity in O_3-mortality associations.

1. Overview of Mechanisms

Evidence on possible mechanisms by which exposure to O_3 may result in acute and chronic health effects is discussed in chapters 5 and 6 of the Criteria Document.[9] Evidence from dosimetry, toxicology, and human exposure studies has contributed to an understanding of the mechanisms that help to explain the biological plausibility and coherence of evidence for O_3-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 O_3 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 O_3 exposure to premature mortality or cardiovascular effects. As discussed more fully in section II.A.3.b.ii below, since the last review an emerging body of animal toxicology and human clinical evidence is beginning to suggest mechanisms that may mediate acute O_3 cardiovascular effects. While much is known about mechanisms that play a role in O_3-related respiratory effects, additional research is needed to more clearly understand the role that O_3 may have in contributing to cardiovascular effects.

With regard to the mechanisms related to short-term respiratory effects, scientific evidence discussed in the Criteria Document (section 5.2) indicates that reactions of O_3 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 O_3. 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 to respond to infectious agents, allergens and toxins. Acute inflammatory responses to O_3 in some healthy people are well documented, and precursors to lung injury can become apparent 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 individual susceptibility to O_3, as discussed below. At the same O_3 dose, individuals who are more susceptible to O_3 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 O_3.

The inhaled dose is the product of O_3 concentration (C), minute ventilation or ventilation rate, and duration of exposure (T), or (C x ventilation rate x 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 O_3 Criteria Documents. In an attempt to describe O_3 dose-response characteristics, acute responses were modeled as a function of total inhaled O_3 dose which was generally found to be a better predictor of response than O_3 concentration, ventilation rate, or duration of exposure, alone, or as a combination of any two of these factors (EPA 2006a, section 6.2). Predicted O_3-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 O_3 dose.

The 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 have been identified as potentially susceptible to health effects as a result of O_3 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

[9] While most of the available evidence addresses mechanisms for O_3, O_3 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 O_3.

vulnerability to O_3-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 [10], as discussed more fully below in section II.A.4.b.

Based on new evidence from animal, human clinical and epidemiological studies the Criteria Document concludes that people with preexisting pulmonary disease are likely to be among those at increased risk from O_3 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 O_3 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 O_3 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 (CD, Figure 8–1) that are altered in response to acute O_3 exposure. They can have larger inflammatory responses, as manifested by larger increases in markers of inflammation such as white bloods 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 O_3, 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. These more serious responses in asthmatics and others with lung disease provide biological plausibility for the respiratory morbidity effects observed in epidemiological studies.

Children with and without asthma were found to be particularly susceptible to O_3 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 O_3. 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 O_3 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 O_3 effects (EPA, 2006a, section 7.6.7.2), primarily in the older adult population. Among the studies that observed positive associations between O_3 and mortality, a comparison of all age or younger age (65 years of age) O_3-mortality effect estimates to that of the elderly population (>65 years) indicates that, in general, the elderly population is more susceptible to O_3 mortality effects. There is supporting evidence of age-related differences in susceptibility to O_3 lung function effects. The Criteria Document concludes that the elderly population (>65 years of age) appears to be at greater risk of O_3-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 O_3 exposure (EPA, 2006a, section 7.6.7.2).

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 O_3 will likely have about the same response if exposed again to the same dose of O_3 (EPA 2006a, p. 6–2). In human clinical studies, group mean responses are not representative of this segment of the population that has much larger than average responses to O_3. Recent studies, discussed in section II.A.4.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 O_3 exposure. These observations suggest a potential role for these markers in the innate susceptibility to O_3, however, the validity of these markers and their relevance in the context of prediction to population studies needs additional experimentation.

Clinical studies that provide information about mechanisms of the initial response to O_3 (*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 O_3 exposure, given the rapid onset of lung function changes and their persistence for 24 to 48 hours among more responsive human subjects in clinical 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 O_3 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 O_3-induced increases in clotting agents arising from the cascade of events, starting with cell injury described above, occurring within 12 to 24 hours of O_3 exposure. The time course for many of these initial responses to O_3 is highly variable. Moreover these observations pertain only to the initial response to O_3. Consequent responses can follow. For example, Jörres *et al.*, (1996) found that in subjects with

[10] In previous Staff Papers and **Federal Register** notices announcing proposed and final decisions on the O_3 and other NAAQS, EPA has used the phrase ''sensitive population groups'' to include both population groups that are at increased risk because they are more susceptible and population groups that are at increased risk due to increased vulnerability or exposure. In this notice, we use the phrase, ''at risk'' populations to include both types of population groups.

asthma and allergic rhinitis, a maximum percent fall in FEV_1 of 27.9% and 7.8%, respectively, occurred 3 days after O_3 exposure when they were challenged with the highest common dose of allergen.

2. Nature of Effects

The 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 O_3 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 Staff Paper, which is based on scientific evidence critically reviewed in chapters 5, 6, and 7 of the Criteria Document, as well as the Criteria Document's integration of scientific evidence contained in chapter 8.[11] Conclusions drawn about O_3-related health effects are based upon the full body of evidence from controlled human exposure, epidemiological and toxicological data contained in the Criteria Document.

a. Morbidity

This section summarizes scientific information on the effects of inhalation of O_3, 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 Criteria Document and chapter 3 of the 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 O_3, as discussed in chapter 10 of the Criteria Document and chapter 3 of the Staff Paper.

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

i. Effects on the Respiratory System From Short-Term and Prolonged O_3 Exposures

Controlled human exposure studies have shown that O_3 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 O_3 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 O_3 exposure health effects on the respiratory system from short-term O_3 exposures were reviewed in the 1986 and 1996 Criteria Documents (EPA, 1986, 1996). In the last review, 0.50 ppm was the lowest O_3 concentration at which statistically significant reductions in forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) were reported in sedentary subjects. During exercise, spirometric (lung function) and symptomatic responses were observed at much lower O_3 exposures. When minute ventilation was considerably increased by continuous exercise (CE) during O_3 exposures lasting 2 hour or less at $\geq$ 0.12 ppm, healthy subjects generally experienced decreases in FEV_1, 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 O_3 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 O_3 concentrations $\geq$ 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 O_3 exposure relative to young adults; (3) O_3-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 O_3, but responses tend to be reproducible within a given individual over a period of several months; (5) subjects exposed repeatedly to O_3 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 O_3 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 O_3 levels near 0.120 ppm, lung function responses largely resolve within 4 to 6 hours post-exposure, 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 hours to return to baseline. The majority of these responses are attenuated after repeated consecutive exposures, but such attenuation to O_3 is lost one week post-exposure.

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 Staff Paper (Appendix 3C). As summarized in more detail in the Staff Paper (section 3.3.1.1), among the more important of the recent studies that examined changes in FEV_1 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 FEV_1 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 O_3 using moderate intermittent 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, O_3 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 FEV_1 decrements and respiratory symptoms than square-wave exposures (when the overall O_3 doses are equal). These results suggest that peak exposures, reflective of the pattern of ambient O_3 concentrations in some locations, are important in terms of O_3 toxicology.

McDonnell (1996) used data from a series of studies to investigate the frequency distributions of FEV_1 decrements following 6.6 hour exposures and found statistically significant but relatively small group mean decreases in average FEV_1 responses (between 5 and 10 percent) at 0.080 ppm O_3.[12] 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, 2007, 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).[13] In these studies, 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 FEV_1 decrements (comparing pre- and post-exposures) when the results were not corrected for the effects of exercise alone in filtered air (EPA, 2007, Figure 3–1B) and with 23 percent of subjects (7 of 30) experiencing such effects when the results were corrected (EPA, 2007, p. 3–6).[14]

These studies by Adams (2002, 2006) are notable in that they are the only available controlled exposure human studies that examine respiratory effects associated with prolonged O_3 exposures at levels below 0.080 ppm, which was the lowest exposure level that had been examined in the last review. The Adams (2006) study investigated a range of exposure levels (0.000, 0.040, 0.060, and 0.080 ppm O_3) using square-wave and triangular exposure patterns. The study was designed to examine multiple comparisons of pulmonary function (FEV_1) 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. At the 0.060 ppm exposure level, the author reported no statistically significant differences for FEV_1 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, comparing FEV_1 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.[15] Notably, these studies report a small percentage of subjects experiencing lung function decrement (≥ 10 percent) at the 0.060 ppm exposure level.[16]

(ii) Results of Epidemiological and Field Studies

A relatively large number of field studies investigating the effects of ambient O_3 concentrations, in combination with other air pollutants, on lung function decrements and respiratory symptoms have 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 FEV_1 or peak expiratory flow (PEF), and respiratory symptoms in healthy adults and asthmatic children are closely correlated to ambient O_3 concentrations. Pre-1996 field studies focused primarily on children attending summer camps and found O_3-related impacts on measures of lung function, but not respiratory symptoms, in healthy children. The newer studies have expanded to evaluate O_3-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 O_3 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 O_3 exposures were estimated by averaging hourly O_3 concentrations from ambient monitors located at the base and summit. The mean 8-hour average O_3 concentration was 0.040 ppm (8-hour average concentration range of 0.021 ppm to 0.074 ppm O_3). Decreased lung function was associated with O_3 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 O_3 exposures among asthmatic and healthy persons. One large U.S. multicity study, the National Cooperative Inner City Asthma Study or NCICAS, (Mortimer *et al.*, 2002) examined O_3-related changes in PEF in 846 asthmatic children from 8 urban areas and reported that the incidence of ≥ 10 percent decrements in morning PEF are associated with increases in 8-hour average O_3 for a 5-day cumulative lag, suggesting that O_3 exposure may be associated with clinically significant changes in PEF in

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

[13] These studies, conducted at a facility at the University of California, in Davis, CA, reported O_3 concentrations to be accurate within +/−0.003 ppm over the range of concentrations included in these studies.

[14] These distributional results presented in the Criteria Document and Staff Paper for the Adams studies are based on study data that were not included in the publication but were obtained from the author.

[15] Brown, J.S. (2007). EPA Office of Research and Development memorandum to Ozone NAAQS Review Docket (OAR–2005–0172); Subject: The effects of ozone on lung function at 0.06 ppm in healthy adults, June 14, 2007.

[16] Based on study data (Adams, 2006) provided by the author, 7 percent of the subjects (2 of 30 subjects) experienced notable FEV_1 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, 2007, p. 3–6).

asthmatic children; however, no associations were reported with evening PEF. The mean 8-hour average O_3 was 0.048 ppm across the 8 cities. Excluding days when 8-hour average O_3 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 O_3 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 O_3-related reductions in PEF, with variations in effect depending on lag time and time of day. Mean 1-hour maximum O_3 concentrations in these locations ranged from 0.190 ppm (SD 80) in northern Mexico City to 0.196 ppm (SD 78) in southwestern Mexico City. While several studies report statistically significant associations between O_3 exposure and reduced PEF in asthmatics, other studies did not, possibly due to low levels of O_3 exposure. EPA concludes that these studies collectively indicate that O_3 may be associated with short-term declines in lung function in asthmatic individuals and that the Mortimer *et al.* (2002) study showed statistically significant effect at concentrations in the range below 0.080 ppm O_3.

Most of the panel studies which have investigated associations between O_3 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 O_3 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 O_3 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 O_3 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 O_3 and 8-hour max O_3 concentrations were 0.0586 ppm (SD 19.0) and 0.0513 ppm (SD 15.5), 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 O_3 were observed on a variety of respiratory symptoms only in the medication user group. Both daily 1-hour max and 8-hour max O_3 concentrations were similarly related to symptoms such as chest tightness and shortness of breath. Effects of O_3, 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 O_3 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 O_3, and provides supportive evidence for effects of O_3 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 provides an asthmatic population representative of the United States, and several single-city studies indicate a robust association of O_3 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 O_3 exposure, these studies had less statistical power and/or were conducted in areas with relatively low 1-hour maximum average O_3 levels, in the range of 0.03 to 0.09 ppm. Even so, the evidence has continued to expand since 1996 and now is considered to be much stronger than in the previous review. The 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 previous review of the O_3 primary standard.

School absenteeism is another potential surrogate for the health implications of O_3 exposure in children. The association between school absenteeism and ambient O_3 concentrations was assessed in two relatively large field studies. 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 PM_{10} and CO concentrations) and found that ambient O_3 concentrations with a distributed lag of 14 days were statistically significantly associated with an increased rate of school absences. Gilliland *et al.* (2001) studied O_3-related absences among about 2,000 4th grade students in 12 southern California communities and found statistically significant associations between 8-hour average O_3 concentrations (with a distributed lag out to 30 days) and all absence categories, and particularly for respiratory causes. Neither PM_{10} nor NO_2 were associated with any respiratory or nonrespiratory illness-related absences in single pollutant models. The Criteria Document concludes that these studies of school absences suggest that ambient O_3 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 O_3 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 Criteria Document (section 6.8) and 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 O_3 causes an increase in airway responsiveness as indicated by a reduction in the concentration of stimuli required to produce a given reduction in FEV_1 or airway obstruction. Increased airway responsiveness is an important consequence of exposure to O_3 because its presence means that the airways are predisposed to narrowing on inhalation of various stimuli, such as specific allergens, cold air or SO_2. 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 O_3 (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 O_3 (0.160 ppm for 7.6 hours) was observed. These observations, based on O_3 exposures to levels much higher than the current standard level suggest that O_3 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, O_3 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 O_3 can induce increased airway responsiveness in asthmatic subjects to O_3, 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 O_3 levels; other studies reported similar results. According to one study (Folinsbee and Hazucha, 2000), changes in airway responsiveness after O_3 exposure resolve more slowly than changes in FEV_1 or respiratory symptoms. Other studies of repeated exposure to O_3 suggest that changes in airway responsiveness tend to be somewhat less affected by attenuation with consecutive exposures than changes in FEV_1 (EPA, 2006a, p. 6–31).

The Criteria Document (section 6.8) concludes that O_3 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 O_3 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 (Criteria Document, p. 8–21). As such, increased airway responsiveness after O_3 exposure represents a plausible link between O_3 exposure and increased hospital admissions.

(c) Respiratory Inflammation and Increased Permeability

Based on evidence from the previous review, acute inflammatory responses in the lung have been observed subsequent to 6.6 hour O_3 exposures to the lowest tested level—0.080 ppm—in healthy adults engaged in moderately high exercise (section 6.9 of the Criteria Document and section 3.3.1.3 of the Staff Paper). Some of these prior studies suggest that inflammatory responses may be detected in some individuals following O_3 exposures in the absence of O_3-induced pulmonary decrements in those subjects. These studies also demonstrate that short-term exposures to O_3 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 Criteria Document (pp. 8–21 to 8–24) consistent with the previous review, but they provide better characterization of the physiological mechanisms by which O_3 causes these effects.

Lung inflammation and increased permeability, which are distinct events controlled by different mechanisms, are two commonly observed effects of O_3 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)[17] 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 O_3. In addition to their role in inflammation, many of these compounds have bronchoconstrictive properties and may be involved in increased airway responsiveness following O_3 exposure. An in vitro study of epithelial cells from nonatopic and atopic asthmatics exposed to 0.010 to 0.100 ppm O_3 showed significantly increased permeability compared to cells from normal persons. This indicates a potentially inherent susceptibility of cells from asthmatic individuals for O_3-induced permeability.

In the 1996 Criteria Document, assessment of controlled human exposure studies indicated that a single, acute (1 to 4 hours) O_3 exposure ($\geq$ 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 O_3 exposures repeated up to 5 days indicate that there is ongoing cellular damage irrespective of attenuation of

[17] Graham and Koren (1990) compared inflammatory mediators present in NL and BAL fluids of humans exposed to 0.4 ppm O_3 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).

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 O_3 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 O_3 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 O_3 exposures at 0.080 and 0.100 ppm. To provide some perspective on the public health impact for these effects, the 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 O_3-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 O_3 dose (product of O_3 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 Criteria Document (chapter 8) concludes that interaction of O_3 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, O_3 reactivity is not eliminated at environmentally relevant exposures (Criteria Document, p. 8–24). Further, antioxidant reactivity with O_3 is both species-specific and dose-dependent.

(d) Increased Susceptibility to Respiratory Infection

As discussed in more detail in the Criteria Document (sections 5.2.2, 6.9.6, and 8.4.2), short-term exposures to O_3 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. Short-term O_3 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 O_3 exposures suppress alveolar phagocytosis and immune system functions. Dysfunction of host defenses and subsequent increased susceptibility to bacterial lung infection in laboratory animals has been induced by short-term exposures to O_3 levels as low as 0.080 ppm.

A single controlled human exposure study reviewed in the 1996 Criteria Document reported that exposure to 0.080 to 0.100 ppm O_3 for 6.6 hours (with moderate exercise) induced decrements in the ability of AMs to phagocytose microorganisms (EPA, 2006a, p. 8–26). Integrating the recent animal study results with human exposure evidence available in the 1996 Criteria Document, the Criteria Document concludes that available evidence indicates that short-term O_3 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 O_3 effects, the effects of particles, and respiratory infections (EPA, 2006a, p. 8–26).

(e) Morphological Effects

The 1996 Criteria Document found that short-term O_3 exposures cause similar alterations in lung morphology in all laboratory animal species studied, including primates. As discussed in the 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 O_3-induced damage (epithelial cell necrosis and remodeling of respiratory bronchioles), possibly because epithelium in this region receives the greatest dose of O_3 delivered to the lower respiratory tract. Following chronic O_3 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 O_3 exposures can cause epithelial cell proliferation and fibrolitic changes in the CAR, these changes appear to be transient with recovery time after exposure, depending on species and O_3 dose. The potential impacts of repeated short-term and chronic morphological effects of O_3 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 of respiratory bronchiolitis, which have the potential to progress to fibrotic lung disease (Criteria Document, p. 8–25).

Recent studies continue to show that short-term and sub-chronic exposures to O_3 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 (Criteria Document, p. 8–25). Based on evidence from animal toxicological studies, short-term and sub-chronic exposures to O_3 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 O_3. As discussed in section 3.3.1.1.6 of the Staff Paper, numerous studies conducted in various locations in the U.S. and Canada consistently have shown a relationship between ambient O_3 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 O_3 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 last review of the O_3 NAAQS, the Criteria Document evaluated emergency department visits and hospital admissions as possible outcomes following exposure to O_3 (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 O_3 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 O_3) and at higher levels (above 0.120 ppm 1-hour max O_3), was found in the group of studies which investigated summertime[18] daily hospital admissions for respiratory causes in different eastern North American cities. These studies consistently demonstrated that ambient O_3 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 O_3, after adjustment for possible confounding effects of temperature and copollutants. Overall, the 1996 Criteria Document concluded that there was strong evidence that ambient O_3 exposures can cause significant exacerbations of preexisting respiratory disease in the general public. Excess respiratory-related hospital admissions associated with O_3 exposures for the New York City area (based on Thurston *et al.*, 1992) were included in the quantitative risk assessment in the prior 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 (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 last or the current O_3 NAAQS review.

In the past decade, a number of studies have examined the temporal pattern associations between O_3 exposures and emergency department visits for respiratory causes (EPA, 2006a, section 7.3.2). These studies are summarized in the 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. Among studies with adequate controls for seasonal patterns, many reported at least one significant positive association involving O_3.

In reviewing evidence for associations between emergency department visits for asthma and short-term O_3 exposures, the Criteria Document notes that in general, O_3 effect estimates from summer only analyses tended to be positive and larger compared to results from cool season or all year analyses (Figure 7–8, EPA, 2006a, p. 7–68). Several of the studies reported significant associations between O_3 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 O_3 concentrations, length of the study period, and statistical methods used to control confounding by seasonal patterns and copollutants appear to affect the observed O_3 effect on emergency department visits. Thus, the Criteria Document has concluded that stratified analyses by season generally supported a positive association between O_3 concentrations and emergency department visits for asthma in the warm season.

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 O_3 levels. Results of a fairly large number of these studies published during the past decade are summarized in 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 O_3 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 O_3 concentrations and respiratory hospital admissions in the warm season. However, not all studies found a statistically significant relationship with O_3, possibly because of very low ambient O_3 levels. Analyses for confounding using multipollutant regression models suggest that copollutants generally do not confound the association between O_3 and respiratory hospitalizations. Ozone effect estimates were robust to PM adjustment in all-year and warm-season only data.

Overall, the Criteria Document concludes that positive and robust associations were found between ambient O_3 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 O_3 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 human clinical, 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 O_3 exposures and increased respiratory morbidity outcomes resulting in increased emergency department visits and hospitalizations during the warm season (EPA, 2006a, p. 8–77).

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

ii. Effects on the Respiratory System of Long-Term O_3 Exposures

The 1996 Criteria Document concluded that there was insufficient evidence from the limited number of studies to determine whether long-term O_3 exposures resulted in chronic health effects at ambient levels observed in the U.S. However, the aggregate evidence suggested that O_3 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 pathophysiologic 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 Criteria Document (p. 7–114) summarizes these studies collectively indicate that seasonal O_3 exposure is associated with smaller growth-related increases in lung function in children than they would have experienced living in areas with lower O_3 levels and that there is some limited, as yet uncertain, evidence that seasonal O_3 also may affect lung function in young adults, although the uncertainty about the role of copollutants makes it difficult to attribute the effects to O_3 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 O_3 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 O_3 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 O_3 exposures might have a negative impact on baseline lung function, particularly during childhood when these exposures might have long-term risks.

Several epidemiological studies published since 1996 have examined the relationship between lung function development and long-term O_3 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 O_3 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 O_3 (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 O_3 levels were associated with decreases in FVC, FEV_1, 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 O_3 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 O_3 levels also experienced smaller increases in various lung function parameters. The strongest relationship with O_3 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 O_3 and NO_2 were significant joint predictors of FEV_1 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 O_3 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 O_3 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 O_3 exposures on lung function development.

Evidence for a significant relationship between long-term O_3 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 O_3 exposures during childhood. A similar study of college freshmen at University of California at Berkeley also reported significant effects of long-term O_3 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 O_3 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 O_3 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 O_3 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 O_3 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-O_3 and six low-O_3 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-O_3 communities versus the six low-O_3 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-O_3 communities. This association was absent in the communities with lower O_3 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 O_3 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 O_3 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 O_3 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 O_3 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 O_3 exposures suggesting that these processes are only partially reversible and may progress following cessation of O_3 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 O_3 exposure on respiratory health outcomes. Limited evidence from recent long-term morbidity studies have suggested in some cases that chronic exposure to O_3 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 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 O_3 Exposure

At the time of the 1997 review, the possibility of O_3-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 O_3 exposures might exert cardiovascular system effects, however much needs to be done to substantiate these potential mechanisms. Possible mechanisms may involve O_3-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 O_3-induced decreases in heart rate, mean arterial pressure, and core temperature. One controlled human exposure study that evaluated effects of O_3 exposure on cardiovascular health outcomes found no significant O_3-induced differences in ECG or blood pressure in healthy or hypertensive subjects but did observe a significant O_3-induced increase the alveolar-to-arterial PO_2 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 O_3 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 O_3-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 O_3 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.

Epidemiologic panel and field studies that examined associations between O_3 and various cardiac physiologic endpoints have yielded limited evidence suggestive of a potential association between acute O_3 exposure and altered heart rate variability, ventricular arrhythmias, and incidence of heart attacks. 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 Criteria Document, many of the studies reported negative or inconsistent associations. Some other studies, especially those that examined the relationship when O_3 exposures were higher, have found robust positive associations between O_3 and cardiovascular hospital admissions (EPA, 2006a, p. 7–82). For example, one study reported a positive association between O_3 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 adjusting 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./Canadian studies showing statistically significant positive associations of cardiovascular hospitalizations with warm-season O_3 concentrations. Most of the available European and Australian studies, all of which conducted all-year O_3 analyses, did not find an association between short-term O_3 concentrations and cardiovascular hospitalizations. Overall, the currently available evidence is inconclusive regarding an association between cardiovascular hospital admissions and ambient O_3 exposure (EPA, 2006a, p. 7–83).

In summary, based on the evidence from animal toxicology, human controlled exposure, and epidemiologic studies, from the Criteria Document concludes that this generally limited body of evidence is suggestive that O_3 can directly and/or indirectly contribute to cardiovascular-related morbidity, but that much needs to be done to more fully integrate links between ambient O_3 exposures and adverse cardiovascular outcomes (EPA, 2006a, p. 8–77).

b. Mortality

i. Mortality and Short-Term O_3 Exposure

The 1996 Criteria Document concluded that an association between daily mortality and O_3 concentration for areas with high O_3 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 current Criteria Document includes results from numerous epidemiological analyses of the relationship between O_3 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 O_3 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 multi-city time-series studies that report associations between O_3 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 PM_{10} on mortality. A significant association was reported between mortality and 24-hour average O_3 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 O_3 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 O_3 measured on the same day, and approximately 1.04 percent per 0.020 ppm change in 24-hour average O_3 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 O_3-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 O_3 exposure (24-hour average) during the summer season only. The authors report an approximate 1.47 percent increase per 0.020 ppm change in O_3 concentration measured on the same day and an approximate 2.52 percent increase per 0.020 ppm change in O_3 concentration using a 7-day distributed lag model. These findings suggest that the effect of O_3 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 O_3 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 O_3 (1-hour maximum) was similar to time-series analysis results with adjustment for temperature (approximately 0.76 percent per 0.040 ppm O_3), suggesting that associations between O_3 and mortality were robust to the different adjustment methods for temperature.

An initial publication from APHEA, a European multi-city study, reported statistically significant associations between daily maximum O_3 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 O_3 in a full year analysis. Gryparis *et al.* (2004) noted that there was a considerable seasonal difference in the O_3 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 with respiratory mortality (EPA, 2006a, p. 7–93, 7–99).

Numerous single-city analyses have also reported associations between mortality and short-term O_3 exposure, especially for those analyses using warm season data. As shown in Figure 7–21 of the Criteria Document, the results of recent publications show a pattern of positive, often statistically significant associations between short-term O_3 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 O_3 and mortality. As described in section 7.4.4 of the 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 O_3 (EPA, 2006a, Figure 7–20). Three recent meta-analyses evaluated potential sources of heterogeneity in O_3-mortality associations (Bell *et al.*, 2005; Ito *et al.*, 2005; Levy *et al.*, 2005). The

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 O_3-mortality associations remained after accounting for that potential bias. The Criteria Document concludes that the ''positive O_3 effects estimates, along with the sensitivity analyses in these three meta-analyses, provide evidence of a robust association between ambient O_3 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. multi-city 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 O_3-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 multi-city studies observed positive associations of ambient O_3 concentrations with total nonaccidental and cardiopulmonary mortality. The Criteria Document finds that the results from U.S. multi-city time-series studies provide the strongest evidence to date for O_3 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 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 Criteria Document (p. 8–78) concludes that these findings are highly suggestive that short-term O_3 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.[19]

ii. Mortality and Long-Term O_3 Exposure

Little evidence was available in the last review on the potential for associations between mortality and long-term exposure to O_3. In the Harvard Six City prospective cohort analysis, the authors report that mortality was not associated with long-term exposure to O_3 (Dockery *et al.*, 1993). The authors note that the range of O_3 concentrations across the six cities was small, which may have limited the power of the study to detect associations between mortality and O_3 levels (EPA, 2006a, p. 7–127).

As discussed in section 7.5.8 of the 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).[20] 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 Staff Paper (section 3.3.2.2) but not in the 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 O_3 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 O_3 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 O_3 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 O_3 exposure and increased risk of lung cancer mortality among males only (Beeson *et al.*, 1998; Abbey

[19] 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).

[20] 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).

et al., 1999). No significant associations were reported between long-term O_3 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 Criteria Document discussed concerns about the plausibility of the reported association with lung cancer (EPA, 2006a, p. 7–130).

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 O_3 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 O_3 exposure estimates concurrently across the study period, there were positive, statistically significant associations between peak O_3 and mortality (EPA, 2006a, p. 7–129).

Overall, the Criteria Document concludes that consistent associations have not been reported between long-term O_3 exposure and all-cause, cardiopulmonary or lung cancer mortality (EPA, 2006a, p. 7–130).

c. Role of Ground-Level O_3 in Solar Radiation-Related Human Health Effects

Beyond the direct health effects attributable to inhalation exposure to O_3 in the ambient air discussed above, the Criteria Document also assesses potential indirect effects related to the presence of O_3 in the ambient air by considering the role of ground-level O_3 in mediating human health effects that may be directly attributable to exposure to solar ultraviolet radiation (UV–B). The 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 Criteria Document provides a thorough analysis of the current understanding of the relationship between reducing ground-level O_3 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 O_3 as a result of any revision to the O_3 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 Criteria Document (section 10.2.3.6) also discusses protective effects of UV–B radiation. Recent reports indicate the necessity of UV–B in producing vitamin D, and that 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 Criteria Document concludes that any assessment that attempts to quantify the consequences of increased UV–B exposure on humans due to reduced ground-level O_3 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 O_3 concentrations on UV-induced health outcomes cannot yet be critically assessed within reasonable uncertainty (Criteria Document, p. 10–36).

The Agency last considered indirect effects of O_3 in the ambient air in its 2003 final response to a remand of the Agency's 1997 decision to revise the O_3 NAAQS. In so doing, based on the available information in the last review, the Administrator determined that the information linking (a) Changes in patterns of ground-level O_3 concentrations likely to occur as a result of programs implemented to attain the 1997 O_3 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 O_3 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 in this review, the Criteria Document and Staff Paper conclude that the effect of changes in ground-level O_3 concentrations, likely to occur as a result of revising the O_3 NAAQS, on UV-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. EPA requests comment on available studies or data that would be relevant to conducting a critical assessment with reasonable certainty of UV-induced health outcomes and how evidence of UV-induced health outcomes might inform the Agency's review of the primary O_3 standard.

3. Interpretation and Integration of Health Evidence

As discussed below, in assessing the new health evidence, the 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 O_3. 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 O_3-related effects. Integrating more broadly across epidemiological and experimental evidence, the Criteria Document also focuses on the *coherence* and *plausibility* of observed O_3-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 O_3 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 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 O_3 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 O_3 or equivalent, whereas associations for hospitalization range up to 50 percent increases per standardized O_3 increment. However, the Criteria Document notes that there are large multicity studies that find small associations between short-term O_3 exposure and mortality or morbidity and have done so with great precision due to the statistical power of the studies (EPA, 2006a, 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 Criteria Document (sections 7.1.3 and 8.4.4.3) and 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 O_3 concentrations measured at centrally-located ambient monitoring stations are generally used to represent a community's exposure to ambient O_3. 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 O_3 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 O_3; 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 Criteria Document (section 3.9), only a limited number of studies have examined the relationship between ambient O_3 concentrations and personal exposures to ambient O_3. 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; Xue *et al.*, 2005). There is some concern about the extent to which ambient concentrations are representative of personal O_3 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 O_3 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 O_3 concentrations and personal exposures to O_3. Collectively, these studies observed that the daily averaged personal O_3 exposures tend to be well correlated with ambient O_3 concentrations despite the substantial variability that existed among the personal measurements. These studies provide supportive evidence that ambient O_3 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 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 O_3 exposures determined by several methods, finding that O_3 exposures based on the use of ambient monitoring data overestimate the individual's O_3 exposure and thus generally result in O_3 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 O_3 concentrations may obscure the presence of thresholds in epidemiologic studies (EPA, 2006a, p. 7–9).

As discussed in the Criteria Document (section 3.9), using ambient concentrations to determine exposure generally overestimates true personal O_3 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 O_3 is greater than these effects estimates indicate. As very few studies evaluating O_3 health effects with personal O_3 exposure measurements exist in the literature, effect estimates determined from ambient O_3 concentrations must be evaluated and used with caution to assess the health risks of O_3. In the absence of available data on personal O_3 exposure, the use of routinely monitored ambient O_3 concentrations as a surrogate for personal exposures is not generally expected to change the principal conclusions from O_3 epidemiologic studies. Therefore, population health risk estimates derived using ambient O_3 levels from currently available observational studies, with appropriate caveats about personal exposure considerations, remain useful. The Criteria Document recommends caution in the quantitative use of effect estimates calculated using ambient O_3 concentrations as they may lead to underestimation of the potency of O_3. However, the Staff Paper observes that the use of these risk estimates for comparing relative risk reductions between alternative ambient O_3 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, SO_2 and NO_2) can meet the criteria for potential confounding in O_3-health associations if they are potential risk factors for the health effect under study and are correlated with O_3. 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 Criteria Document (p. 7–148) observes that O_3 is generally not highly correlated with other criteria pollutants (*e.g.*, PM_{10}, CO, SO_2 and NO_2), but may be more highly correlated with secondary fine particles, especially during the summer months, and that the degree of correlation between O_3 and other pollutants may vary across seasons. For example, positive associations are observed between O_3 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 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 O_3-health relationships. The 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 O_3 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 multi-city and single-city studies indicate that O_3-mortality associations do not appear to be substantially changed in multipollutant models including PM_{10} or $PM_{2.5}$ (EPA, 2006a, p. 7–101; Figure 7–22). Focusing on results of warm season analyses, effect estimates for O_3-mortality associations are fairly robust to adjustment for PM in multipollutant models (EPA, 2006a, p. 7–102; Figure 7–23). The Criteria Document concludes that in the few multipollutant analyses conducted for these endpoints, copollutants generally do not confound the relationship between O_3 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 O_3, but the Criteria Document reports that results of available analyses indicate that such associations generally were robust to adjustment for $PM_{2.5}$ (EPA, 2006a, p. 7–154). For example, in a large multi-city study of asthmatic children (Mortimer *et al.*, 2002), the O_3 effect was attenuated, but there was still a positive association; in Gent *et al.* (2003), effects of O_3, 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 Criteria Document concludes: ''Multipollultant regression analyses indicated that O_3 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 O_3 on respiratory health outcomes appear to be robust and independent of the effects of other copollutants (EPA, 2006a, p. 7–154).''

The Criteria Document observes that another challenge of time-series epidemiological analysis is assessing the relationship between O_3 and health outcomes while avoiding bias due to confounding by other time-varying factors, particularly seasonal trends and weather variables (EPA, 2006a, p. 7–14). These variables are of particular interest because O_3 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 O_3 or with seasonal or weather variables in statistical analyses.

The 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 O_3 coefficients to the extent of adjustment for temporal trends and weather factors. In addition, several recent studies, including U.S. multi-city 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 O_3-mortality associations. As discussed in the Criteria Document (section 7.6.3.1), these studies generally report that associations reported with O_3 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 multi-city analysis was presented that found that alternative adjustments for weather resulted in up to 2-fold difference in the O_3 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 O_3 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 multi-city studies and single-city studies in different areas, the Criteria Document (p. 8–41) observes general consistency in effects of short-term O_3 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 O_3, as well as varying concentrations and composition of copollutants present in different regions. Thus, the 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 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 O_3 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 O_3 and health outcomes after removal of days with higher O_3 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 O_3 exposure and mortality was little changed when days exceeding 0.060 ppm (24-hour average) were excluded in the analysis. Bell *et al.* (2006) found no difference in estimated effect even when all days with 24-hour O_3 concentrations <0.020 ppm were excluded (EPA, 2006a, p. 8–43). Using data from 8 U.S. cities, Mortimer and colleagues (2002) also reported that associations between O_3 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 O_3 exposure and mortality. As discussed in section 7.6.5 of the Criteria Document, one European multi-city study included evaluation of the shape of the concentration-response curve, and observed no deviation from a linear function across the range of O_3 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 O_3 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 O_3 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 O_3-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 O_3 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 O_3 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 O_3 and mortality in British Columbia where O_3 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 O_3 concentrations suggests that any potential threshold level would be quite low in this data set.

In summary, the Criteria Document finds that, taken together, the available evidence from clinical and epidemiological studies suggests that no clear conclusion can now be reached with regard to possible threshold levels for O_3-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 O_3-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 Staff Paper emphasizes the role of O_3 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 O_3 could, either directly or through initiation, interfere with basic cellular oxidation processes responsible for inflammation, reduced antioxidant capacity, atherosclerosis and other effects. Reasoning that O_3 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 O_3 may also contribute to a number of adverse respiratory and cardiovascular health effects. Consequently, the role of O_3 should be considered more broadly as O_3 behaves as a generator of numerous oxidative species in the atmosphere.

In considering the biological plausibility of reported O_3-related effects, the Staff Paper (section 3.4.6) considers this broader question of health effects of pollutant mixtures containing O_3. The potential for O_3-related enhancements of PM formation, particle uptake, and exacerbation of PM-induced cardiovascular effects underscores the importance of considering contributions of O_3 interactions with other often co-occurring air pollutants to health effects due to O_3-containing pollutant mixes. The Staff Paper summarizes some examples of important pollutant mixture effects from studies that evaluate interactions of O_3 with other co-occurring pollutants, as discussed in chapters 4, 5, and 6 of the Criteria Document.

All of the types of interactive effects of O_3 with other co-occurring gaseous and nongaseous viable and nonviable PM components of ambient air mixes noted above argue that O_3 acts not only alone but that O_3 also is a surrogate indicator for air pollution mixes which may enhance the risk of adverse effects due to O_3 acting in combination with other pollutants. Viewed from this perspective, those epidemiologic findings of morbidity and mortality associations, with ambient O_3 concentrations extending to quite low levels in many cases, become more understandable and plausible.

The 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 O_3-related health effects to reach judgments about the causal nature of observed associations. As summarized below, coherence and biological plausibility are discussed for each of the following types of O_3-related effects: short-term effects on the respiratory system, effects on the cardiovascular system, effects related to long-term O_3 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 O_3 include such health endpoints as decrements in lung function, 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 O_3 NAAQS review on associations between ambient O_3 exposure and decline in lung function for children. The Criteria Document (p. 8–34) concludes that exposure to ambient O_3 has a significant effect on lung function and is associated with increased respiratory symptoms and medication use, particularly in asthmatics. Short-term exposure to O_3 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 O_3 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 O_3-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.[21] Positive effect estimates indicate increases in the health outcome with O_3 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.

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[21] 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.

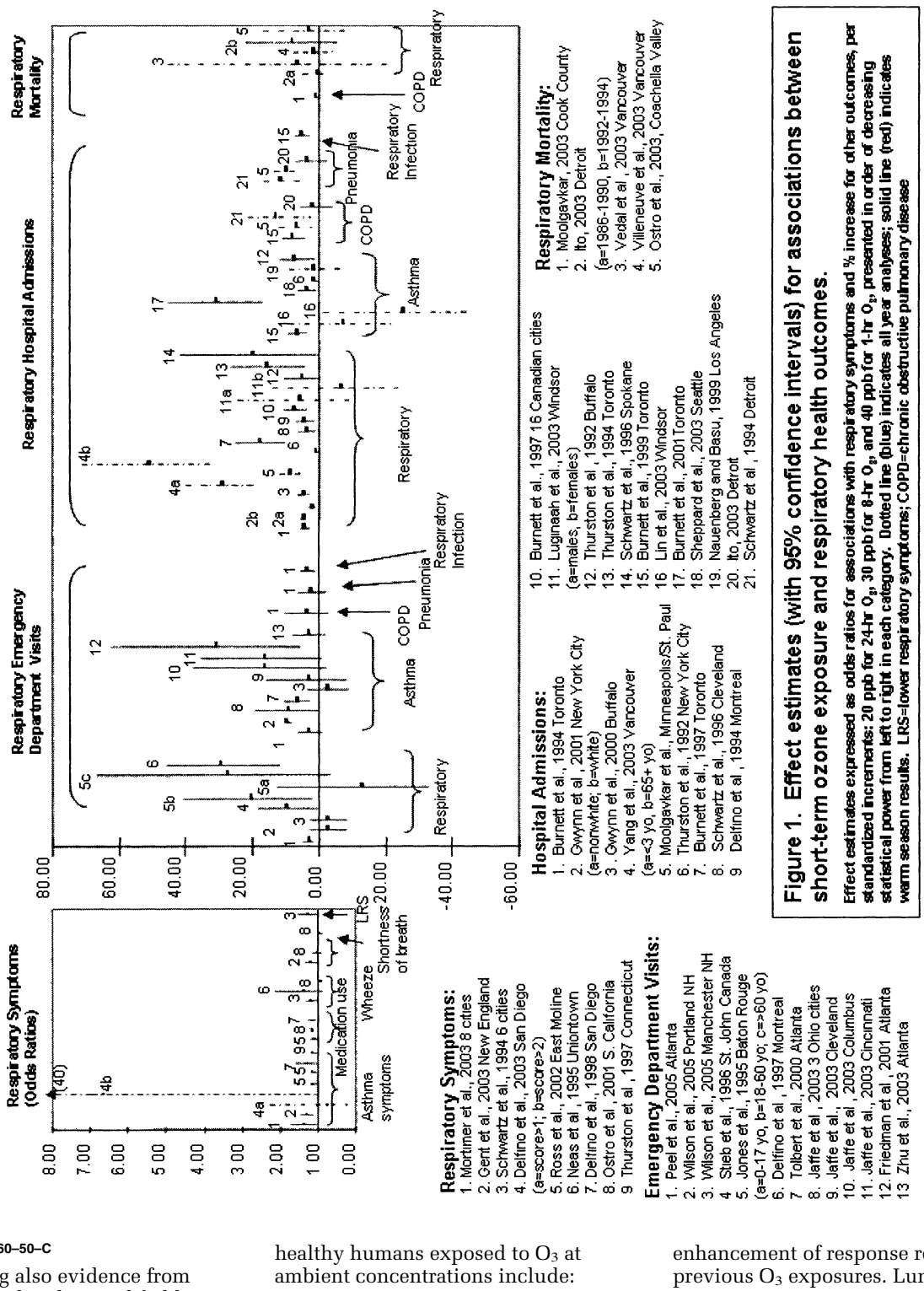

Figure 1. Effect estimates (with 95% confidence intervals) for associations between short-term ozone exposure and respiratory health outcomes.

Effect estimates expressed as odds ratios for associations with respiratory symptoms and % increase for other outcomes, per standardized increments: 20 ppb for 24-hr O_3, 30 ppb for 8-hr O_3, and 40 ppb for 1-hr O_3, 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

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Considering also evidence from toxicological, chamber, and field studies, the Criteria Document (section 8.6) discusses biological plausibility and coherence of evidence for acute O_3-induced respiratory health effects. Inhalation of O_3 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 O_3 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 O_3 sensitivity, and the degree of attenuation or enhancement of response resulting from previous O_3 exposures. Lung function studies of several animal species acutely exposed to relatively low O_3 levels (0.25 to 0.4 ppm) show responses similar to those observed in humans, including increased breathing frequency, decreased 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 O_3 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 O_3-induced cardiopulmonary health effects (EPA, 2006a, p. 8–48).

The interaction of O_3 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 O_3 are well documented, and lung injury can become apparent within 3 hours after exposure in humans.

Taken together, the Criteria Document concludes that the evidence from experimental human and animal toxicology studies indicates that acute O_3 exposure is causally associated with respiratory system effects, including O_3-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 O_3 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.b, 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 O_3 exposure may be associated with cardiovascular disease outcomes have been described. Laboratory animals exposed to relatively high O_3 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 O_3 exposure in animal toxicology studies (Vesely *et al.*, 1994a, b, c) also raise the possibility of potential cardiovascular effects of acute ambient O_3 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 O_3-induced vasoconstriction in a controlled human exposure study by Brook *et al.* (2002) suggests another possible mechanism for O_3-related exacerbations of preexisting cardiovascular disease. One controlled human study (Gong *et al.*, 1998) evaluated potential cardiovascular health effects of O_3 exposure. The overall results did not indicate acute cardiovascular effects of O_3 in either the hypertensive or control subjects. The authors observed an increase in rate-pressure product and heart rate, a decrement for FEV_1, and a >10 mm Hg increase in the alveolar/arterial pressure difference for O_2 following O_3 exposure. Foster *et al.* (1993) demonstrated that even in relatively young healthy adults, O_3 exposure can cause ventilation to shift away from the well-perfused basal lung. This effect of O_3 on ventilation distribution may persist beyond 24-hours post-exposure (Foster *et al.*, 1997). These findings suggest that O_3 may exert cardiovascular effects indirectly by impairing alveolar-arterial O_2 transfer and potentially reducing O_2 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.b, a limited number of new epidemiological studies have reported associations between short-term O_3 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 O_3 and heart rate variability (HRV) and the other study evaluated the association between O_3 levels and the relative risk of myocardial infarction (MI). 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 O_3 exposure and the cardiovascular endpoints studied. In other recent studies on the incidence of MI 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 O_3 (EPA, 2006a, section 7.2.7.1). From these studies, the 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 O_3 exposure and cardiovascular hospital admissions. As discussed in section 7.3.4 of the Criteria Document, many reported negative or inconsistent associations, whereas other studies, especially those that examined the relationship when O_3 exposures were higher, have found positive and robust associations between O_3 and cardiovascular hospital admissions. The Criteria Document finds that the overall evidence from these studies remains inconclusive regarding the effect of O_3 on cardiovascular hospitalizations (EPA, 2006a, p. 7–83).

The Criteria Document notes that the suggestive positive epidemiologic findings of O_3 exposure on cardiac autonomic control, including effects on HRV, ventricular arrhythmias and MI, and reported associations between O_3 exposure and cardiovascular hospitalizations generally in the warm season gain credibility and scientific support from the results of experimental animal toxicology and human clinical studies, which are indicative of plausible pathways by which O_3 may exert cardiovascular effects (EPA, 2006a, section 8.6.1).

iii. Coherence and Plausibility of Effects Related to Long-Term O_3 Exposure

Human chamber studies can not evaluate effects of long-term exposures to O_3; there is some evidence available from toxicological studies. While early animal toxicology studies of long-term O_3 exposures were conducted using continuous exposures, more recent studies have focused on exposures which mimic diurnal and seasonal patterns and more realistic O_3 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 O_3-induced effects on lung function. As discussed in section 8.6.2 of the Criteria Document, previous epidemiological studies have provided only inconclusive evidence for either mortality or morbidity effects of long-term O_3 exposure. The Criteria Document 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 (EPA, 2006a, p. 8–50). Several new longitudinal epidemiology studies have evaluated associations between long-term O_3 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 O_3 exposure and mortality or lung cancer incidence (EPA, 2006a, p. 8–50).

The Criteria Document (p. 8–51) concludes that evidence from animal toxicology studies strongly suggests that chronic O_3 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 pulmonary 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 O_3 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 Criteria Document (sections 7.4 and 8.6.3). These single- and multi-city mortality studies coupled with meta-analyses generally indicate associations between acute O_3 exposure and elevated risk for all-cause mortality, even after adjustment for the influence of season and PM. Several single-city studies that specifically evaluated the relationship between O_3 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 O_3-induced biochemical effects are relatively transient and attenuate over time. The Criteria Document (p. 8–52) hypothesizes a generic pathway of O_3-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 O_3-related contributions to cardiovascular effects that ultimately increase risk of mortality.

The third National Health and Nutrition Examination Follow-up data analysis indicates that about 20 percent of the adult population has reduced FEV_1 values, suggesting impaired lung function in some 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 FEV_1 values have the highest levels of CRP, and those with the highest FEV_1 values have the lowest CRP levels (Manino *et al.*, 2003; Sin and Man, 2003). This complex series of physiological and biochemical reactions following O_3 exposure may tilt the biological homeostasis mechanisms which could lead to adverse health effects in people with compromised cardiopulmonary systems.

Of much interest are several other types of newly available data that support reasonable hypotheses that may help to explain the findings of O_3-related increases in cardiovascular mortality observed in some epidemiological studies. These include the direct effect of O_3 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 MI, 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 O_3 concentrations and changes in cardiac autonomic control (*e.g.*, HRV, ventricular arrhythmias, and MI). These represent plausible pathways that may lead to O_3-related contributions to cardiovascular effects that ultimately increase the risk of mortality.

In addition, O_3-induced increases in lung permeability allow more ready entry for inhaled PM into the blood stream, and O_3 exposure may increase the risk of PM-related cardiovascular effects. Furthermore, increased ambient O_3 levels contribute to ultrafine PM formation in the ambient air and indoor environments. Thus, the contributions of elevated ambient O_3 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 O_3 (EPA, 2006a, p. 8–53).

c. Summary

Judgments concerning the extent to which relationships between various health endpoints and ambient O_3

exposures are likely causal are informed by the conclusions and discussion in the Criteria Document as discussed above and summarized in section 3.7.5 of the Staff Paper. These judgments reflect the nature of the evidence and 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 O_3 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 Criteria Document (p. 8–74) states that these studies provide clear evidence of causality for associations between short-term O_3 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 O_3 exposures, and significant associations between ambient O_3 exposures and a wide variety of symptoms have been reported in epidemiology studies (EPA, 2006a, p. 8–75). Aggregate population time-series studies showing robust associations with respiratory hospital admissions and emergency department visits are strongly supported by human clinical, animal toxicologic, and epidemiologic evidence for O_3-related lung function decrements, respiratory symptoms, airway inflammation, and airway hyperreactivity. The Criteria Document (p. 8–77) concludes that, taken together, the overall evidence supports the inference of a causal relationship between acute ambient O_3 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 Criteria Document (p. 8–78) as highly suggestive that O_3 directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality.

4. O_3-Related Impacts on Public Health

The following discussion draws from chapters 6 and 7 and section 8.7 of the Criteria Document and section 3.6 of the Staff Paper to characterize factors which modify responsiveness to O_3, subpopulations potentially at risk for O_3-related health effects, the adversity of O_3-related effects, and the size of the at-risk subpopulations in the U.S. These considerations are all important elements in characterizing the potential public health impacts associated with exposure to ambient O_3.

a. Factors That Modify Responsiveness to Ozone

There are numerous factors that can modify individual responsiveness to O_3. 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 Criteria Document.

It is well established that physical activity increases an individual's minute ventilation and will thus increase the dose of O_3 inhaled (EPA, 2006a, section 6.5.4). Increased physical activity results in deeper penetration of O_3 into more distal regions of the lungs, which are more sensitive to acute O_3 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 O_3 concentrations if the level of exertion is increased and/or duration of exposure and exertion are extended. Predicted O_3-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 O_3 decline as age increases. The rate of decline in O_3 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 was estimated for 20 year old individuals exposed to 0.12 ppm O_3, whereas similar exposure of 35 year old individuals were estimated to have a 2.6% decrement. While healthy children tend not to report respiratory symptoms when exposed to low levels of O_3, for subjects 18 to 36 years old symptom responses induced by O_3 tend to decrease with increasing age (McDonnell *et al.*, 1999).

Limited evidence of gender differences in response to O_3 exposure has suggested that females may be predisposed to a greater susceptibility to O_3. 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 O_3 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 O_3 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 O_3. The limited data available, which have investigated the influence of race, ethnic or other related factors on responsiveness to O_3, 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. New controlled human exposure studies have confirmed that smokers are less responsive to O_3 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 O_3 has been studied very little, but additive effects of heat and O_3 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 O_3 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 O_3 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 O_3-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

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 O_3 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 O_3-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.[22]

i. Active People

A large group of individuals at risk from O_3 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 O_3 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 O_3 exposure-response relationship with increasing spirometric and symptomatic response as exercise level increases. Furthermore, O_3-induced response increases as time of exposure increases. Studies of outdoor workers and others who participate in outdoor activities indicate that extended exposures to O_3 at elevated exertion levels can produce marked effects on lung function, as discussed above in section IIA.2 (Brauer *et al.*, 1996; Höppe *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 human chamber studies have examined the effects of O_3 exposure in subjects performing continuous or intermittent exercise for variable periods of time. Significant O_3-induced respiratory responses have been observed in clinical studies of exercising individuals. The epidemiologic studies discussed above also indicate that prolonged exposure periods, combined with elevated levels of exertion or exercise, may magnify O_3 effects on lung function. Thus, outdoor workers and others who participate in higher exertion activities outdoors during the time of day when high peak O_3 concentrations occur appear to be particularly vulnerable to O_3 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 O_3 in the ambient air.

ii. People With Lung Disease

People with preexisting pulmonary disease are likely to be among those at increased risk from O_3 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 O_3 exposure. At the time of the last review, it was concluded that this group was at greater risk because the impact of O_3-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 pulmonary 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 subpopulation for O_3 health effects.

Several clinical studies reviewed in the 1996 Criteria Document on atopic and asthmatic subjects had suggested but not clearly demonstrated enhanced responsiveness to acute O_3 exposure compared to healthy subjects. The majority of the newer studies reviewed in Chapter 6 of the Criteria Document indicate that asthmatics are as sensitive as, if not more sensitive than, normal subjects in manifesting O_3-induced pulmonary 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 O_3-induced decrease in FEF_{25-75} in asthmatics relative to the healthy subjects (24% versus 15%, respectively). A significant positive correlation in asthmatics was also reported between O_3-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 (Criteria Document, Figure 8–1) that are altered in response to acute O_3 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 cell 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 O_3 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.*, 1997a; Michelson *et al.*, 1999; Hiltermann *et al.*, 1999; Holz *et al.*, 2002; Vagaggini *et al.*, 2002). In asthma, the eosinophil, 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 O_3-induced bronchial epithelial cytokine expression between healthy and asthmatic subjects. After O_3 exposure the epithelial expression of IL–5 and GM-CSF increased significantly in

[22] In the Staff Paper and documents from previous O_3 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.

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 O_3-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 O_3-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 O_3-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 O_3-induced change in the percentage of eosinophils in induced sputum and the change in PC_{20}, the concentration of methacholine causing a 20% decrease in FEV_1. Characteristic O_3-induced inflammatory airway neutrophilia at one time was considered a leading mechanism of airway hyperresponsiveness. However, Hiltermann *et al.* (1999) determined that the O_3-induced change in percentage neutrophils in sputum was not significantly related to the change in PC_{20}. 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. (Criteria Document, AX 6–26). Hiltermann *et al.* (1999) concluded that the results point to the role of eosinophils in O_3-induced airway hyperresponsiveness. Increases in O_3-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 O_3 exposure with bronchial allergen challenge in subjects with preexisting allergic airway disease. Jörres *et al.* (1996) found that O_3 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 O_3 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 FEV_1 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 O_3 exposure when compared to FA exposure. In subjects with asthma and allergic rhinitis, a maximum percent fall in FEV_1 of 27.9% and 7.8%, respectively, occurred 3 days after O_3 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 O_3 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 O_3 that resulted in a clinically relevant (>20%) decrease in FEV1. Ozone-induced exacerbation of airway responsiveness persists longer and attenuates more slowly than O_3-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 O_3 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 O_3-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 O_3 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 O_3 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 O_3-induced adverse effects. This body of evidence indicates that human clinical and epidemiological panel studies of lung function decrements and respiratory symptoms that evaluate only healthy, non-asthmatic subjects likely underestimate the effects of O_3 exposure on asthmatics and other susceptible populations. The effects of O_3 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 O_3 exposures focused on children. These studies suggest that O_3 exposure may be 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 Criteria Document (section 7.6.7.1). Strong evidence from a large multi-city study (Mortimer *et al.*, 2002), along with support from several single-city studies suggest that O_3 exposure may be associated with increased respiratory symptoms and medication use in children with asthma. With regard to ambient O_3 levels and increased hospital admissions and emergency department visits for asthma and other respiratory causes, strong and consistent evidence establishes a correlation between O_3 exposure and increased exacerbations of preexisting respiratory disease for 1-hour maximum O_3 concentrations <0.12 ppm. As discussed in the 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 O_3 and increased risk of emergency department visits and hospital admissions for asthma and other respiratory diseases, especially during the warm season. Finally, from epidemiological studies that included assessment of associations with specific causes of death, some studies have observed larger effects estimates for respiratory mortality and others have observed larger effects estimates for cardiovascular mortality. The apparent inconsistency regarding the effect size of O_3-related respiratory mortality may be due to reduced statistical power in this

subcategory of mortality (EPA, 2006a, p. 7–108).

Newly available reports from controlled human exposure studies (see chapter 6 in the 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 O_3 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 O_3-induced adverse health effects.

Although controlled human exposure studies have not found evidence of larger spirometric changes 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 have such changes based on their age. However, in section 8.7.1, the 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 O_3 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 O_3 concentrations. Moreover, as indicated by Gong *et al.* (1998), the effects of O_3 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 O_3 delivered to the well-ventilated regions of the COPD lung could further inhibit gas exchange and reduce oxygen saturation. In addition, O_3-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).

iii. Children and Older Adults

Supporting evidence exists for heterogeneity in the effects of O_3 by age. As discussed in section 6.5.1 of the Criteria Document, children, adolescents, and young adults (<18 yrs of age) appear, on average, to have nearly equivalent spirometric responses to O_3, but have greater responses than middle-aged and older adults when exposed to comparable O_3 doses. Symptomatic responses to O_3 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 O_3 exposure and dose.

As described in the section 7.6.7.2 of the Criteria Document, many epidemiological field studies focused on the effect of O_3 on the respiratory health of school children. In general, children experienced decrements in pulmonary function parameters, including PEF, FEV_1, 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 O_3 effects on lung function. Approximately 20% of the children, both with and without asthma, experienced a greater than 10% change in FEV_1, compared to only 5% 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 O_3. 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 O_3 effects (EPA, 2006a, section 7.6.7.2). Older adults are also often classified as being particularly susceptible to air pollution. The basis for increased O_3 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). (EPA 2006a, p. 8–60) 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 O_3 health effects is most clearly indicated in the newer mortality studies. Among the studies that observed positive associations between O_3 and mortality, a comparison of all age or younger age (≤65 years of age) O_3-mortality effect estimates to that of the elderly population (>65 years) indicates that, in general, the elderly population is more susceptible to O_3 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 O_3 on premature mortality may be substantially greater in the elderly population because of higher rates of preexisting respiratory and cardiac diseases. The Criteria Document concludes that the elderly population (>65 years of age) appear to be at greater risk of O_3-related mortality and hospitalizations compared to all ages or younger populations (EPA, 2006a, p. 7–177).

The Criteria Document notes that, collectively, there is supporting evidence of age-related differences in susceptibility to O_3 lung function effects. The elderly population (>65 years of age) appear to be at increased risk of O_3-related mortality and hospitalizations, and children (<18 years of age) experience other potentially adverse respiratory health outcomes with increased O_3 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 O_3-sensitive and resistant strains and illustrated the importance of genetic background in determining O_3 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 O_3 will likely have about the same response if exposed again to the same dose of O_3. In human clinical studies, group mean responses are not representative of this segment of the population that has much larger than average responses to O_3. 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 pulmonary function and inflammatory responses to O_3 exposure.[23]

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 O_3-sensitive and O_3-resistant species, it has been possible to identify the involvement of increased airway reactivity and inflammation processes in O_3 susceptibility. However, most of these studies were carried out using relatively high doses of O_3, 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 and, ultimately, can likely be integrated with epidemiological studies.

v. Other Population Groups

There is limited, new evidence supporting associations between short-term O_3 exposures and a range of effects on the cardiovascular system. Some but not all, epidemiological studies have reported associations between short-term O_3 exposures and the incidence of MI 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 O_3 exposure and mortality from cardiovascular or cardiopulmonary causes. The Criteria Document concludes that current cardiovascular effects evidence from some field studies is rather limited but supportive of a potential effect of short-term O_3 exposure and HRV, cardiac arrhythmia, and MI incidence (EPA, 2006a, p. 7–65). In the 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 O_3 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 O_3; however, more evidence is needed to conclude that people with heart disease are a susceptible population.

Other groups that might have enhanced sensitivity to O_3, 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 O_3.

c. Adversity of Effects

In making judgments as to when various O_3-related effects become regarded as adverse to the health of individuals, the Administrator has 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)[24] 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 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 last review of the O_3 standard, evidence for these more serious effects came from studies of effects in laboratory animals. Evidence from animal studies evaluated in this Criteria Document strongly suggests that O_3 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 multi-city time-series epidemiology studies and meta-analyses of these epidemiology studies are highly suggestive of an association between short-term O_3 exposure and mortality particularly in the warm season.

While O_3 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 O_3 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

[23] Similar to animal toxicology studies referred above, a polymorphism in a specific proinflammatory cytokine gene has been implicated in O_3-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 O_3, however, the validity of these markers and their relevance in the context of prediction to population studies needs additional experimentation.

[24] 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.

(*e.g.*, decrements in FEV_1 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 Staff Paper.

For active healthy people, moderate levels of functional responses (*e.g.*, FEV_1 decrements of ≥10% but <20%, 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.*, FEV_1 decrements ≥20%, 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.*, FEV_1 decrements ≥15% but <20%) is appropriate for estimating potentially adverse lung function decrements in active healthy people. However, for people with lung disease, even moderate functional (*e.g.*, FEV_1 decrements ≥10% but <20%, 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.*, FEV_1 decrements ≥20%, 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.*, FEV_1 decrements ≥10%) is most appropriate for estimating potentially adverse lung function decrements in active healthy people.

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 O_3 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 Subpopulations

Although O_3-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 subpopulations potentially at-risk for O_3-related health effects discussed above. For example, a population of concern includes people with respiratory disease, including 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 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, subpopulations based on age group also comprise substantial segments of the population that may be potentially at risk for O_3-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 age groups include those most likely to have increased susceptibility to the health effects of O_3 and or those with the highest ambient O_3 exposures.

The 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 millions 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 O_3-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 O_3 exposure. Thus, even a small percentage reduction in O_3 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 O_3 and various health endpoints, the Criteria

Document (section 8.7.5.2) concludes that exposure to ambient O_3 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 O_3-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 Staff Paper 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 O_3 primary standard (Staff Paper, p. 6–20—6–21).

In modeling exposures and health risks associated with just meeting the current and alternative O_3 standards, EPA has simulated air quality to represent conditions just meeting these standards based on O_3 air quality patterns in several recent years and on how the shape of the O_3 air quality distribution has changed over time based on historical trends in monitored O_3 air quality data. As described in the Staff Paper (section 4.5.8) and discussed below, recent O_3 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.[25]

As noted in section I.C above, around the time of the release of the final 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.[26]

1. Exposure Analyses

a. Overview

The EPA conducted exposure analyses using a simulation model to estimate O_3 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 current 8-hour O_3 standard is not met. The emphasis on children reflects the finding of the last O_3 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 O_3 epidemiological studies, the availability of ambient O_3 data, and the desire to represent a range of geographic areas, population demographics, and O_3 climatology. These selection criteria are discussed further in chapter 5 of the Staff Paper. The geographic extent of each modeled area consists of the census tracts in the combined statistical area (CSA) as defined by OMB (OMB, 2005).[27]

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 O_3 concentrations (2002, 2003, and 2004), as well as for O_3 concentrations adjusted to simulate conditions associated with just meeting the current NAAQS and various alternative 8-hour standards based on the three year period 2002–2004.[28] This exposure assessment is more fully described and presented in the Staff Paper and in a technical support document, *Ozone Population Exposure Analysis for Selected Urban Areas* (US EPA, 2006b; 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.[29]

The goals of the O_3 exposure assessment were: (1) To provide estimates of the size of at-risk populations exposed to various levels associated with recent O_3 concentrations, and with just meeting the current O_3 NAAQS and alternative O_3 standards, in specific urban areas; (2) to provide distributions of exposure estimates over the entire range of ambient O_3 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

[25] 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.

[26] EPA plans to make available corrected versions of the final Staff Paper, and human exposure and health risk assessment technical support documents on or around July 16, 2007 on the EPA web site listed in the Availability of Related Information section of this notice.

[27] 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, 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.

[28] All 12 of the CSAs modeled did not meet the current O_3 NAAQS for the three year period examined.

[29] The general approach used in the current exposure assessment was described in the draft Health Assessment Plan (EPA, 2005a) that was released to the CASAC and general public in April 2005 and was the subject of a consultation with the CASAC O_3 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 O_3 Panel on August 24–25, 2006.

reductions associated with meeting alternative O_3 standards.

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 O_3 exposure estimates. With respect to variability, the exposure modeling approach accounts for variability in ambient O_3 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 O_3 season, the modeling of variations in ambient concentrations near roadways, and the modeling of air exchange rates that affect the amount of O_3 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 Staff Paper (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 O_3-related effects observed in clinical 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 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 O_3 are primarily driven by ambient outdoor concentrations, which vary by time of day, location, and peoples' activities. Outdoor O_3 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 O_3 within each urban area, associated with current O_3 levels and with O_3 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 O_3 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 O_3 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 O_3 (pNEM/O_3) used in the last O_3 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 O_3 in indoor, outdoor, and in-vehicle microenvironments. The exposure model takes into account the most significant factors contributing to total human O_3 exposure, including the temporal and spatial distribution of people and O_3 concentrations throughout an urban area, the variation of O_3 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 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. These are discussed fully in the 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 O_3 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 (Tables 26 and 27 in Langstaff, 2007). 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 O_3 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 O_3 seasons in 2002, 2003, and

2004. The O_3 season in each area includes the period of the year where elevated O_3 levels tend to be observed and for which routine hourly O_3 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 O_3 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 O_3 levels is due to a combination of different weather patterns and the variation in emissions of O_3 precursors. Nationally, 2002 was a relatively high year with respect to the 4th highest daily maximum 8-hour O_3 levels observed in urban areas across the U.S. (EPA, 2007, Figure 2–16), with the mean of the distribution of O_3 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 O_3 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 O_3 levels observed in 2003 in the 12 urban areas and nationally generally were between those observed in 2002 and 2004.

Regulatory scenarios examined include the 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.[30] The current standard uses a rounding convention that allows areas to have an average of the 4th daily maximum 8-hour averages as high as 0.084 ppm and still meet the standard. All alternative standards analyzed were intended to reflect improved precision in the measurement of ambient concentrations, where the precision would extend to three instead of two decimal places (in ppm).

The 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 value[31] corresponding to the standard being analyzed. The quadratic rollback technique reduces higher concentrations more than lower concentrations near ambient background levels.[32] This procedure was considered in a sensitivity analysis in the last review of the O_3 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 O_3 while at specified exertion levels[33] serve two purposes. First, the entire range of modeled personal exposures to ambient O_3 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 O_3 concentrations at and above specific benchmark levels provide some perspective on the public health impacts of health effects that we cannot currently evaluate 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 we know or can reasonably infer that specific O_3-related health effects are occurring. We refer to exposures at and above these benchmark concentrations as ''exposures of concern.''

EPA emphasizes 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 O_3 exposure levels. 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 O_3 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 O_3-related health effects that have been demonstrated in human clinical 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, we first considered the exposure level of 0.080 ppm, at which there is a substantial amount of clinical evidence demonstrating a range of O_3-related health effects including lung inflammation and airway responsiveness in healthy individuals. Thus, as in the last 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 epidemiology studies indicates that people with asthma have larger and more serious effects than healthy individuals, including lung function, respiratory symptoms, increased airway

[30] The current O_3 standard is 0.08 ppm, but the current rounding convention specifies 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 the 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.

[31] 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 current O_3 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.

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

[33] As discussed above in Section II.A., O_3 health responses observed in human clinical 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 m^2), 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-m^2 (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 O_3 exposures.

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 epidemiology studies shows associations with serious respiratory morbidity and cardiopulmonary mortality effects at O_3 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 O_3 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 O_3 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 O_3 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 O_3 concentrations, or higher, at least one time during the O_3 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 O_3-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 O_3, but have not been evaluated at lower levels in controlled human exposure studies. EPA has not included these effects in the quantitative risk assessment due to a lack of adequate information on the exposure-response relationships.

The 1997 O_3 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. 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 has chosen 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 O_3 concentrations also are at risk for experiencing exposures associated with O_3-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 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 O_3 were estimated in 12 urban areas for 2002, 2003, and 2004 air quality, and also using O_3 concentrations adjusted to just meet the 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.[34] 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 O_3 benchmark level of exposures of concern, while at intermittent moderate or greater exertion and based on O_3 concentrations observed in 2002 and 2004. Table 1 summarizes estimates for 2002 and 2004, because these years reflect years that bracket relatively higher and lower O_3 levels, with year 2003 generally containing O_3 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 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 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 1 or more exposures of concern decline from simulations of just meeting the current 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 $\geq$ 0.070 ppm, while engaged in moderate or greater exertion, during an O_3 season is about 18 percent of this population when the current 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.

[34] The full range of quantitative exposure estimates associated with just meeting the current and alternative O_3 standards are presented in chapter 4 and Appendix 4A of the Staff Paper.

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 current standard]		Asthmatic children, ages 5–18 Aggregate for 12 urban areas, number of children exposed (% of group) [% reduction from current 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/m^2.

[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 current 8-hour standard 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 Staff Paper (section 4.5.8), recent O_3 air quality distributions have been statistically adjusted to simulate just meeting the current 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 O_3 season when the current 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 O_3 concentrations are simulated to just meet the current standard, the aggregate 12 urban area estimate is 18 percent of all school age children are estimated to experience O_3 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, 2007, Exhibit 2, p. 4–48). 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, 2007, Exhibit 8, p. 4–60).

(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 O_3 building upon a more limited risk assessment that was conducted during the last review.[35] As part of the last 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 O_3 exposures for 9 urban areas.[36] The risk assessment for the last review also included risk estimates for excess respiratory-related hospital admissions related to O_3 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 O_3 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 this review 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 O_3 levels; (2) risk reductions and remaining

[35] 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).

[36] 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, Philadephia, St. Louis, and Washington, DC.

risks associated with just meeting the current 8-hour O_3 NAAQS; and (3) risk reductions and remaining risks associated with just meeting various alternative 8-hour O_3 NAAQS in a number of example urban areas. This risk assessment is more fully described and presented in the Staff Paper (EPA, 2007, chapter 5) and in a technical support document (TSD), *Ozone Health Risk Assessment for Selected Urban Areas* (Abt Associates, 2006, 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 O_3 Panel and the public.[37] 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 O_3 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 O_3 levels, and with meeting the current and alternative 8-hour O_3 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 O_3 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 Criteria Document and 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 the current 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 Staff Paper, there are a number of health endpoints (*e.g.*, increased lung inflammation, increased 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 O_3. 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.

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 O_3 risk estimates. As discussed in chapters 2, 5, and 6 of the 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 the current and selected alternative standards In the prior review, different statistical approaches using alternative functional forms (*i.e.*, quadratic, proportional, Weibull) were used to reflect how O_3 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, 2007, p. 6–20). With respect to uncertainties about estimated background concentrations, as discussed below and in the Staff Paper (EPA 2007b, 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, 2007, pp. 6–20—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 O_3 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 O_3 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 O_3 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. EPA recognizes that these credible intervals do not reflect all of the uncertainties noted above.

[37] The general approach used in the current risk assessment was described in the draft Health Assessment Plan (EPA, 2005a) that was released to the CASAC and general public in April 2005 and was the subject of a consultation with the CASAC O_3 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 O_3 Panel on August 24–25, 2006.

b. Scope and Key Components

The current health risk assessment is based on the information evaluated in the final Criteria Document. The risk assessment includes several categories of health effects and estimates risks associated with just meeting the current and alternative 8-hour O_3 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.[38] 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 do not meet the current 8-hour O_3 NAAQS and which included the largest areas with O_3 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 final Criteria Document and or Staff Paper concluded that the evidence as a whole supports the general conclusion that O_3, 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 the current and alternative 8-hour O_3 NAAQS, it is necessary to estimate the distribution of hourly O_3 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 O_3 season or single warm O_3 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 current and selected alternative 8-hour standards represent risks associated with O_3 levels attributable to anthropogenic sources and activities (*i.e.*, risk associated with concentrations above ''policy-relevant background''). Policy-relevant background O_3 concentrations used in the O_3 risk assessment were defined in chapter 2 of the Staff Paper (EPA, 2007, pp. 2–48—2–55) as the O_3 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 O_3 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 O_3 season using meteorology for the year 2001. Based on the results of the GEOS–CHEM model, the Criteria Document indicates that background O_3 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 O_3 episodes.[39]

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 O_3 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 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). Recognizing these complexities, EPA requests comments on the new approach used in this review for estimating these levels as an input to the health risk assessment.[40]

In the first part of the current risk assessment, lung function decrement, as measured by FEV_1, 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 O_3 exposures for school age children engaged in moderate exertion based on numerous controlled human exposure and summer camp field studies conducted by various investigators. Risk estimates have been developed for O_3-related lung function decrements (measured as changes in FEV_1) 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, 2003, 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 FEV_1). As discussed in the Staff Paper (EPA, 2007, 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 O_3 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

[38] 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.

[39] EPA notes that the estimated level of policy-relevant background O_3 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.

[40] Recognizing the importance of this issue, EPA intends to conduct additional sensitivity analyses related to policy-relevant background and its implications for the risk assessment.

to sample size in the combined data set that served as the basis for the assessment. 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 current 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). 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 FEV_1). However, the Staff Paper and risk estimates summarized below focus on FEV_1 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 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 current and alternative 8-hour O_3 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 O_3 season.[41] These 12 urban areas include approximately 18.3 million school age children, of which 2.6 million are asthmatic school age children.[42]

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 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 Staff Paper and in the Risk Assessment TSD (Abt Associates, 2006).

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 Criteria Document and Staff Paper, as well as the criteria discussed in chapter 5 of the Staff Paper, the following categories of health endpoints associated with short-term exposures to ambient O_3 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 current risk assessment. With respect to nonaccidental and cardiorespiratory mortality, the Criteria Document concludes that there is strong evidence which is highly suggestive of a causal relationship between nonaccidental and cardiorespiratory-related mortality and O_3 exposures during the warm O_3 season. As discussed in the 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, O_3 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.[43] 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 O_3 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 O_3 exposures in excess of background, as well as the change in incidence corresponding to a given change in O_3 levels resulting from just meeting the current or alternative 8-hour O_3 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 O_3 from ambient monitors for the selected location, estimates of background O_3 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 O_3 standard); (2) relative risk-based concentration-response functions that provide an estimate of the relationship between the health endpoints of interest and ambient O_3 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 O_3 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

[41] 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.

[42] For 9 of the 12 urban areas, the O_3 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 O_3 season is defined as the entire year.

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

between each health endpoint of interest and changes in ambient O_3 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 multi-city and single-city O_3 concentration-response functions. While the Risk Assessment TSD and chapter 5 of the Staff Paper present a more comprehensive set of risk estimates, EPA has focused on estimates based on multi-city studies where available. The advantages of relying more heavily on concentration-response functions based on multi-city 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 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 O_3 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 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 O_3 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 O_3 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 Staff Paper. Because O_3 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 Criteria Document finds that no definitive conclusion can be reached with regard to the existence of population thresholds in epidemiological studies (Criteria Document, pp. 8–44). 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 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 O_3 must be considered in light of uncertainties about whether or not these O_3-related effects occur in these populations at very low O_3 concentrations.

With respect to variability within this portion of the risk assessment, there is variability among concentration-response functions describing the relation between O_3 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 O_3 (*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 current 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 Staff Paper (chapter 5) and Risk Assessment TSD present risk estimates associated with just meeting the current and several alternative 8-hour standards, as well as three recent years of air quality as represented by 2002, 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 O_3 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 has chosen to include only the 8-hour moderate exertion exposures in the current risk assessment for this health endpoint. Thus, the risk estimates presented here and in the 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 current standard and several alternative 8-hour standard levels with the same form as the current 8-hour standard. 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 areas [44] who are estimated to have at least 1 moderate or greater lung function response (defined as FEV_1 ≥15 percent in all children and ≥10 percent in asthmatic children) associated with 8-hour exposures to O_3 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 O_3 air quality data) and relatively low year (based on adjusting 2004 O_3 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 current 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 O_3 levels just meeting the current standard based on 2002 air quality data compared to 230,000 (1.2 percent of children) associated with just meeting the current standard based on 2004 air quality data.

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 1 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 current standard]		Asthmatic children, ages 5–18, FEV_1 ≥10 percent, aggregate for 5 urban areas, number of children affected (% of group) [% reduction from current standard]	
	2002	2004	2002	2004]
0.084 ppm (Current standard).	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/m^2.

[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 O_3 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 current 8-hour standard 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 stated concentration level. As described in the Staff Paper (section 4.5.8), recent O_3 air quality distributions have been statistically adjusted to simulate just meeting the current 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.

As discussed in the Staff Paper, a child may experience multiple occurrences of a lung function response during the O_3 season. For example, upon meeting the current 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 O_3 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 O_3 season per responding child for air quality just meeting the current 8-hour standard across the 12 urban areas. While the estimated number of occurrences per O_3 season is lower when based on the 2004 simulation than for the 2002 simulation, the estimated number of occurrences per responding child is similar. EPA recognizes that some children in the population might have only 1 or 2 occurrences while others may have 6 or more occurrences per O_3 season. Risk estimates based on adjusting 2003 air quality to simulate just meeting the current and alternative 8-hour standards are intermediate to the estimates presented in Table 2 above in this notice and are presented in the Staff Paper (chapter 5) and Risk Assessment TSD.

For just meeting the current 8-hour standard, Table 5–8 in the 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 Staff Paper and in the Risk Assessment TSD.

For just meeting the current 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 attainment of the current 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 FEV_1 decrement ≥15 percent due to 8-hour O_3 exposures across the 12 urban

[44] 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.

areas analyzed, and similarly across the 5 urban areas analyzed for asthmatic school age children (ages 5–18) estimated to experience at least one FEV_1 decrement ≥10 percent, when the current and alternative 8-hour standards are just met.

(2) For asthmatic school age children, the median estimates of occurrences of FEV_1 decrements ≥10% range from 52,000 to nearly 510,000 responses associated with just meeting the current 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 O_3 season ranged from about 6 to 11 associated with just meeting the current 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 O_3 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 O_3 in the model) is about 6,100 (based on the 2002 simulation) and about 4,500 (based on the 2004 simulation) upon meeting the current 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 the current 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 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 O_3 concentrations in excess of background levels from April through September for several recent years (2002, 2003, and 2004) and upon just meeting the current and alternative 8-hour standards based on simulating O_3 levels using 2002–2004 O_3 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 the current 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 the current 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 the current 8-hour standard to 3.0 cases per 100,000 under the 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. 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 the current standard (based on the 2002 simulation). The patterns for cardiorespiratory mortality are similar. The aggregate O_3-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 the current standard, using simulated O_3 concentrations that just meet the current 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 the current 8-hour standard is associated with 24-hour O_3 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 O_3-related nonaccidental mortality is estimated to occur when O_3 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 O_3 risk assessment, together with important uncertainties and limitations.

(1) As discussed in the 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 O_3 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 the current and potential alternative 8-hour standards.

(3) There is noticeable city-to-city variability in estimated O_3-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 the current 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 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 O_3-related non-accidental mortality, while notable differences (*i.e.*, greater than 50 percent)[45] 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 current standard, was significantly less impacted.

C. Conclusions on the Adequacy of the Current Primary Standard

1. Background

The initial issue to be addressed in the current review of the primary O_3 standard is whether, in view of the advances in scientific knowledge and additional information, the existing standard should be revised. In evaluating whether it is appropriate to retain or revise the current standard, the Administrator builds upon the last review and reflects the broader body of evidence and information now available. The Administrator has taken into account both evidence-based and quantitative exposure- and risk-based considerations in developing conclusions on the adequacy of the current primary O_3 standard. Evidence-based considerations include the assessment of evidence from controlled human exposure, animal toxicological, field, and epidemiological studies for a variety of health endpoints. For those endpoints based on epidemiological studies, greater weight has been placed on associations with health endpoints that are causal or likely causal based on an integrative synthesis of the entire body of evidence, including not only all available epidemiological evidence but also evidence from animal toxicological and controlled human exposure studies. Less weight has been placed on evidence of associations that were judged to be only suggestive of possible causal relationships. Consideration of quantitative exposure- and risk-based information draws from the results of the exposure and risk assessments described above. More specifically, estimates of the magnitude of O_3-related exposures and risks associated with recent air quality levels, as well as the exposure and risk reductions likely to be associated with just meeting the current 8-hour primary O_3 NAAQS, have been considered.

In this review, a series of general questions frames the approach to reaching a decision on the adequacy of the current standard, such as the following: (1) To what extent does newly available information reinforce or call into question evidence of associations of O_3 exposures with effects identified in the last review?; (2) to what extent has evidence of new effects and/or at-risk populations become available since the last review?; (3) to what extent have important uncertainties identified in the last review been reduced and have new uncertainties emerged?; (4) to what extent does newly available information reinforce or call into question any of the basic elements of the current standards?

The question of whether the available evidence supports consideration of a standard that is more protective than the current standard includes consideration of: (1) Whether there is evidence that associations, especially likely causal associations, extend to ambient O_3 concentration levels that are as low as or lower than had previously been observed, and the important uncertainties associated with that evidence; (2) the extent to which exposures of concern and health risks are estimated to occur in areas upon meeting the current standard and the important uncertainties associated with the estimated exposures and risks; and (3) the extent to which the O_3-related health effects indicated by the evidence and the exposure and risk assessments are considered important from a public health perspective, taking into account the nature and severity of the health effects, the size of the at-risk populations, and the kind and degree of the uncertainties associated with these considerations.

The current primary O_3 standard is an 8-hour standard, which was set at a level of 0.08 ppm,[46] with a form of the annual fourth-highest daily maximum 8-hour average concentration, averaged over three years. This standard was chosen to provide protection to the public, especially children and other at-risk populations, against a wide range of O_3-induced health effects. As an introduction to this discussion of the adequacy of the current O_3 standard, it is useful to summarize the key factors that formed the basis of the decision in the last review to revise the averaging time, level, and form of the then current 1-hour standard.

In the last review, the key factor in deciding to revise the averaging time of the primary standard was evidence from controlled human exposure studies of healthy young adult subjects exposed for 1 to 8 hours to O_3. The best documented health endpoints in these studies were decrements in indices of lung function, such as forced expiratory volume in 1 second (FEV_1), and respiratory symptoms, such as cough and chest pain on deep inspiration. For short-term exposures of 1 to 3 hours, group mean FEV1 decrements were statistically significant for O_3 concentrations only at and above 0.12 ppm, and only when subjects engaged in very heavy exertion. By contrast, evidence available in the prior review showed that prolonged exposures of 6 to 8 hours produced statistically significant group mean FEV_1 decrements at the lowest O_3 concentrations evaluated in those studies, 0.080 ppm, even when experimental subjects were engaged in more realistic intermittent moderate exertion levels. The health significance of this newer evidence led to the conclusion in the 1997 final decision that the 8-hour averaging time is more directly associated with health effects of concern at lower O_3 concentrations than is the 1-hour averaging time.

[45] 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.

[46] If the standard were to be specified to the nearest thousandth ppm, the current 0.08 ppm 8-hour standard would be equivalent to a standard set at 0.084 ppm, reflecting the data rounding conventions that are part of the definition of the current 8-hour standard.

Based on the available evidence of O_3-related health effects, the following factors were of particular importance in the last review in informing the selection of the level and form of a new 8-hour standard: (1) Quantitative estimates of O_3-related risks to active children, who were judged to be an at-risk subgroup of concern, in terms of transient and reversible respiratory effects judged to be adverse, including moderate to large decreases in lung function and moderate to severe pain on deep inspiration, and the uncertainty and variability in such estimates; (2) consideration of both the estimated percentages, total numbers of children, and number of times they were likely to experience such effects; (3) epidemiological evidence of associations between ambient O_3 and increased respiratory hospital admissions, and quantitative estimates of percentages and total numbers of asthma-related admissions in one example urban area that were judged to be indicative of a pyramid of much larger effects, including respiratory-related hospital admissions, emergency department visits, doctor visits, and asthma attacks and related increased medication use; (4) quantitative estimates of the number of ''exposures of concern[47]'' (defined as exposures ≥ 0.080 ppm for 6 to 8 hour) that active children are likely to experience, and the uncertainty and variability in such estimates; (5) the judgment that such exposures are an important indicator of public health impacts of O_3-related effects for which information is too limited to develop quantitative risk estimates, including increased nonspecific bronchial responsiveness (*e.g.*, related to aggravation of asthma), decreased pulmonary defense mechanisms (suggestive of increased susceptibility to respiratory infection), and indicators of pulmonary inflammation (related to potential aggravation of chronic bronchitis or long-term damage to the lungs); (6) the broader public health perspective of the number of people living in areas that would breathe cleaner air as a result of the revised standard; (7) consideration of the relative seriousness of various health effects and the relative degree of certainty in both the likelihood that people will experience various health effects and their medical significance; (8) the relationship of a standard level to estimated ''background'' levels associated with nonanthropogenic sources of O_3; and (9) CASAC's advice and recommendations. Additional factors considered in selecting the form of the standard included balancing the public health implications of the estimated number of times in an O_3 season that the standard level might be exceeded in an area that is in attainment with the standard with the year-to-year stability of the air quality statistic, which can be particularly affected by years with unusual meteorology. A more stable air quality statistic serves to avoid disruptions to ongoing control programs that could result from moving into and out of attainment, thereby interrupting the public health protection afforded by such control programs.

In reaching a final decision in the last review, the Administrator was mindful that O_3 exhibits a continuum of effects, such that there is no discernible threshold above which public health protection requires that no exposures be allowed or below which all risks to public health can be avoided. The final decision reflected a recognition that important uncertainties remained, for example with regard to interpreting the role of other pollutants co-occurring with O_3 in observed associations, understanding biological mechanisms of O_3-related health effects, and estimating human exposures and quantitative risks to at-risk populations for these health effects.

2. Evidence- and Exposure/Risk-Based Considerations in the Staff Paper

The Staff Paper (section 6.3.1) considers the evidence presented in the Criteria Document as discussed above in section II.A as a basis for evaluating the adequacy of the current O_3 standard, recognizing that important uncertainties remain. The extensive body of human clinical, toxicological, and epidemiological evidence serves as the basis for the judgments about O_3-related health effects discussed above, including judgments about causal relationships with a range of respiratory morbidity effects, including lung function decrements, increased respiratory symptoms, airway inflammation, increased airway responsiveness, and respiratory-related hospitalizations and emergency department visits in the warm season, and about the evidence being highly suggestive that O_3 directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality.

These judgments take into account important uncertainties that remain in interpreting this evidence. For example, with regard to the utility of time-series epidemiological studies to inform judgments about a NAAQS for an individual pollutant, such as O_3, within a mix of highly correlated pollutants, such as the mix of oxidants produced in photochemical reactions in the atmosphere, the Staff Paper notes that there are limitations especially at ambient O_3 concentrations below levels at which O_3-related effects have been observed in controlled human exposure studies. The Staff Paper (section 3.4.5) also recognizes that 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 variability in susceptibility to O_3-related effects in populations.

While noting these limitations in the interpretation of the findings from the epidemiological studies, the Staff Paper (section 3.4.5) concludes that if a population threshold level does exist, it would likely be well below the level of the current O_3 standard and possibly within the range of background levels. As discussed above in section II.A.3.a, this conclusion is supported by several epidemiological studies that have explored the question of potential thresholds directly, either using a statistical curve-fitting approach to evaluate whether linear or non-linear models fit the data better using sub-sets of the data, where days over or under a specific cutpoint (*e.g.*, 0.080 ppm or even lower O_3 levels) were excluded and then evaluating the association for statistical significance. In addition to direct consideration of the epidemiological studies, findings from controlled human exposure studies discussed above in section II.A.2.a.i(a)(i) indicate that prolonged exposures produced statistically significant group mean FEV_1 decrements and symptoms in healthy adult subjects at levels down to at least 0.060 ppm, with a small percentage of subjects experiencing notable effects (*e.g.*, >10 percent FEV_1 decrement, pain on deep inspiration). Controlled human exposure studies evaluated in the last review also found significant responses in indicators of lung inflammation and cell injury at 0.080 ppm in healthy adult subjects. The effects in these controlled human exposure studies were observed in healthy young adult subjects, and it is likely that more serious responses, and

[47] In the last review, ''exposures of concern'' referred to exposures at and above 0.08 ppm, 8-hour average, at which a range of health effects have been observed in controlled human studies, but for which data were too limited to allow for quantitative risk assessment. (62 FR 38860, July 18, 1997).

responses at lower levels, would occur in people with asthma and other respiratory diseases. 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. The observations provide additional support for the conclusion in the Staff Paper that the associations observed in the epidemiological studies, particularly for respiratory-related effects and potentially for cardiovascular effects, extend down to O_3 levels well below the current standard (*i.e.*, 0.084 ppm) (EPA, 2007, p. 6–7).

As discussed above in section II.A and in the Staff Paper (section 3.7), the newly available information reinforces the judgments about the likelihood of causal relationships between O_3 exposure and respiratory effects observed in the last review and broadens the evidence of O_3-related associations to include additional respiratory-related endpoints, newly identified cardiovascular-related health endpoints, and mortality. Newly available evidence also has shown that people with asthma are likely to experience more serious effects than people who do not have asthma (section II.A.4.b.ii above). The Staff Paper also concludes that substantial progress has been made since the last review in advancing the understanding of potential mechanisms by which ambient O_3, alone and in combination with other pollutants, is causally linked to a range of respiratory-related health endpoints, and may be causally linked to a range of cardiovascular-related health endpoints. Thus, the Staff Paper (section 6.3.6) finds strong support in the evidence developed since the last review, for consideration of an O_3 standard that is at least as protective as the current standard and finds no support for consideration of an O_3 standard that is less protective than the current standard. This conclusion is consistent with the advice and recommendations of CASAC and with the views expressed by all interested parties who provided comments on drafts of the Staff Paper. While CASAC and some commenters supported revising the current standard to provide increased public health protection and other commenters supported retaining the current standard, no one who provided comments supported a standard that would be less protective than the current standard.

a. Evidence-Based Considerations

In looking more specifically at the controlled human exposure and epidemiological evidence (which is summarized in chapter 3 and Appendix 3B of the Staff Paper), the Staff Paper first notes that controlled human exposure studies provide the clearest and most compelling evidence for an array of human health effects that are directly attributable to acute exposures to O_3 *per se.* Evidence from such human studies, together with animal toxicological studies, help to provide biological plausibility for health effects observed in epidemiological studies. In considering the available evidence, the Staff Paper focuses on studies that examined health effects that have been demonstrated to be caused by exposure to O_3, or for which the Criteria Document judges associations with O_3 to be causal or likely causal, or for which the evidence is highly suggestive that O_3 contributes to the reported effects. In considering the epidemiological evidence as a basis for reaching conclusions about the adequacy of the current standard, the Staff Paper focuses on studies reporting effects in the warm season, for which the effect estimates are more consistently positive and statistically significant than those from all-year studies. The Staff Paper (section 6.3.1.1) considers the extent to which such studies provide evidence of associations that extend down to ambient O_3 concentrations below the level of the current standard, which would thereby call into question the adequacy of the current standard. In so doing, the Staff Paper notes, as discussed above, that if a population threshold level does exist for an effect observed in such studies, it would likely be at a level well below the level of the current standard. The Staff Paper (section 6.3.1.1) also attempts to characterize whether the area in which a study was conducted likely would or would not have met the current standard during the time of the study, although it recognizes that the confidence that would appropriately be placed on the associations observed in any given study, or on the extent to which the association would likely extend down to relatively low O_3 concentrations, is not dependent on this distinction. Further, the Staff Paper considered studies that examined subsets of data that include only days with ambient O_3 concentrations below the level of the current O_3 standard, or below even lower O_3 concentrations, and continue to report statistically significant associations. The Staff Paper (section 6.3.1.1) judges that such studies are directly relevant to considering the adequacy of the current standard, particularly in light of reported responses to O_3 at levels below the current standard found in controlled human exposure studies.

i. Lung Function, Respiratory Symptoms, and Other Respiratory Effects

Health effects for which the Criteria Document continues to find clear evidence of causal associations with short-term O_3 exposures include lung function decrements, respiratory symptoms, pulmonary inflammation, and increased airway responsiveness. In the last review, these O_3-induced effects were demonstrated with statistical significance down to the lowest level tested in controlled human exposure studies at that time (*i.e.*, 0.080 ppm). As discussed in chapter 3 of the Staff Paper, and in section II.A.2.a.i.(a)(i) above, two new studies are notable in that they are the only controlled human exposure studies that examined respiratory effects, including lung function decrements and respiratory symptoms, in healthy adults at lower exposure levels than had previously been examined. EPA's reanalysis of the data from the most recent study shows small group mean decrements in lung function responses to be statistically significant at the 0.060 ppm exposure level, while the author's analysis did not yield statistically significant lung function responses but did yield some statistically significant respiratory symptom responses toward the end of the exposure period. Notably, these studies report a small percentage of subjects experiencing lung function decrements ($\geq$ 10 percent) at the 0.060 ppm exposure level. These studies provide very limited evidence of O_3-related lung function decrements and respiratory symptoms at this lower exposure level.

The Staff Paper (section 3.3.1.1.1) notes that evidence from controlled human exposures studies indicates that people with moderate-to-severe asthma have somewhat larger decreases in lung function in response to O_3 relative to healthy individuals and that lung function responses in people with asthma appear to be affected by baseline lung function (*i.e.*, magnitude of responses increases with increasing disease severity). As discussed in the Criteria Document (p.8–80), this newer information expands our understanding of the physiological basis for increased sensitivity in people with asthma and other airway diseases, recognizing that

people with asthma present a different response profile for cellular, molecular, and biochemical responses than people who do not have asthma. New evidence indicates that some people with asthma have increased occurrence and duration of nonspecific airway responsiveness, which is an increased bronchoconstrictive response to airway irritants. Controlled human exposure studies also indicate that some people with allergic asthma and rhinitis have increased airway responsiveness to allergens following O_3 exposure. Exposures to O_3 exacerbated lung function decrements in people with pre-existing allergic airway disease, with and without asthma. Ozone-induced exacerbation of airway responsiveness persists longer and attenuates more slowly than O_3-induced lung function decrements and respiratory symptom responses and can have important clinical implications for asthmatics.

The Staff Paper (p.6–10) also concludes that newly available human exposure studies suggest that some people with asthma also have increased inflammatory responses, relative to non-asthmatic subjects, and that this inflammation may take longer to resolve. 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 O_3-induced adverse effects. This body of evidence qualitatively informs the Staff Paper's (pp.6–10 to 6–11) evaluation of the adequacy of the current O_3 standard in that it indicates that human clinical and epidemiological panel studies of lung function decrements and respiratory symptoms that evaluate only healthy, non-asthmatic subjects likely underestimate the effects of O_3 exposure on asthmatics and other susceptible populations.

The Staff Paper (p.6–11) notes that in addition to the experimental evidence of lung function decrements, respiratory symptoms, and other respiratory effects in healthy and asthmatic populations discussed above, epidemiological studies have reported associations of lung function decrements and respiratory symptoms in several locations (Appendix 3B; also Figure 3–4 for respiratory symptoms). As discussed in the Staff Paper (section 3.3.1.1.1) and above, two large U.S. panel studies which together followed over 1000 asthmatic children on a daily basis (Mortimer *et al.*, 2002, the National Cooperative Inner-City Asthma Study, or NCICAS; and Gent *et al.*, 2003), as well as several smaller U.S. and international studies, have reported robust associations between ambient O_3 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 O_3 and increased asthma medication use. Overall, the multi-city NCICAS (2002), Gent *et al.* (2003), and several other single-city studies indicate a robust positive association between ambient O_3 concentrations and increased respiratory symptoms and increased medication use in asthmatics.

In considering the large number of single-city epidemiological studies reporting lung function or respiratory symptoms in healthy or asthmatic populations (Staff Paper, Appendix 3B), the Staff Paper (p.6–11) notes that most such studies that reported positive and often statistically significant associations in the warm season were conducted in areas that likely would not have met the current standard. In considering the large multi-city NCICAS (Mortimer *et al.*, 2002), the Staff Paper notes that the 98th percentile 8-hour daily maximum O_3 concentrations at the monitor reporting the highest O_3 concentrations in each of the study areas ranged from 0.084 ppm to >0.10 ppm. However, the authors indicate that less than 5 percent of the days in the eight urban areas had 8-hour daily O_3 concentrations exceeding 0.080 ppm. Moreover, the authors observed that when days with 8-hour average O_3 levels greater than 0.080 ppm were excluded, similar effect estimates were seen compared to estimates which included all of the days. There are also a few other studies in which the relevant air quality statistics provide some indication that lung function and respiratory symptom effects may be occurring in areas that likely would have met the current standard (EPA, 2007, p.6–12).

ii. Respiratory Hospital Admissions and Emergency Department Visits

At the time of the last review, many time-series studies indicated positive associations between ambient O_3 and increased respiratory hospital admissions and emergency room visits, providing strong evidence for a relationship between O_3 exposure and increased exacerbations of preexisting lung disease at O_3 levels below the level of the then current 1-hour standard (EPA 2007, section 3.3.1.1.6). Analyses of data from studies conducted in the northeastern U.S. indicated that O_3 air pollution was consistently and strongly associated with summertime respiratory hospital admissions.

Since the last review, new epidemiological studies have evaluated the association between short-term exposures to O_3 and unscheduled hospital admissions for respiratory causes. Large multi-city studies, as well as many studies from individual cities, have reported positive and often statistically significant O_3 associations with total respiratory hospitalizations as well as asthma- and COPD-related hospitalizations, especially in studies analyzing the O_3 effect during the summer or warm season. Analyses using multipollutant regression models generally indicate that copollutants do not confound the association between O_3 and respiratory hospitalizations and that the O_3 effect estimates were robust to PM adjustment in all-year and warm-season only data. The Criteria Document (p.8–77) concludes that the evidence supports a causal relationship between acute O_3 exposures and increased respiratory-related hospitalizations during the warm season.

In looking specifically at U.S. and Canadian respiratory hospitalization studies that reported positive and often statistically significant associations (and that either did not use GAM or were reanalyzed to address GAM-related problems), the Staff Paper (p.6–12) notes that many such studies were conducted in areas that likely would not have met the current O_3 standard, with many providing only all-year effect estimates, and with some reporting a statistically significant association in the warm season. Of the studies that provide some indication that O_3-related respiratory hospitalizations may be occurring in areas that likely would have met the current standard, the Staff Paper notes that some are all-year studies, whereas others reported statistically significant warm-season associations.

Emergency department visits for respiratory causes have been the focus of a number of new studies that have examined visits related to asthma, COPD, bronchitis, pneumonia, and other upper and lower respiratory infections, such as influenza, with asthma visits typically dominating the daily incidence counts. Among studies with adequate controls for seasonal patterns, many reported at least one significant positive association involving O_3. However, inconsistencies were observed which were at least partially attributable to differences in model specifications and analysis approach among various studies. In general, O_3 effect estimates from summer-only analyses tended to be positive and larger compared to results from cool season or all-year analyses. Almost all of the studies that reported

statistically significant effect estimates were conducted in areas that likely would not have met the current standard. The Criteria Document (section 7.3.2) concluded that analyses stratified by season generally supported a positive association between O_3 concentrations and emergency department visits for asthma in the warm season. These studies provide evidence of effects in areas that likely would not have met the current standard and evidence of associations that likely extend down to relatively low ambient O_3 concentrations.

iii. Mortality

The 1996 Criteria Document concluded that an association between daily mortality and O_3 concentrations for areas with high O_3 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, and thus the possibility that O_3 exposure may be associated with mortality was not relied upon in the 1997 decision on the O_3 primary standard.

Since the last review, as described above, the body of evidence with regard to O_3-related health effects has been expanded by animal, human clinical, and epidemiological studies and now includes biologically plausible mechanisms by which O_3 may affect the cardiovascular system. In addition, there is stronger information linking O_3 to serious morbidity outcomes, such as hospitalization, that are associated with increased mortality. Thus, there is now a coherent body of evidence that describes a range of health outcomes from lung function decrements to hospitalization and premature mortality.

Newly available large multi-city studies (Bell *et al.*, 2004; Huang *et al.*,2005; and Schwartz 2005) designed specifically to examine the effect of O_3 and other pollutants on mortality have provided much more robust and credible information. Together these studies have reported significant associations between O_3 and mortality that were robust to adjustment for PM and different adjustment methods for temperature and suggest that the effect of O_3 on mortality is immediate but also persists for several days. One recent multi-city study (Bell *et al.*, 2006) examined the shape of the concentration-response function for the O_3-mortality relationship in 98 U.S. urban communities for the period 1987 to 2000 specifically to evaluate whether a ''safe'' threshold level exists. Results from various analytic methods all indicated that any threshold, if it exists, would likely occur at very low concentrations, far below the level of the current O_3 NAAQS and nearing background levels.

New data are also available from several single-city studies conducted world-wide, as well as from several meta-analyses that have combined information from multiple studies. Three recent meta-analyses evaluated potential sources of heterogeneity in O_3-mortality associations. All three analyses reported common findings, including effect estimates that were statistically significant and larger in warm season analyses. Reanalysis of results using default GAM criteria did not change the effect estimates, and there was no strong evidence of confounding by PM. The Criteria Document (p.7–175) finds that the majority of these studies suggest that there is an elevated risk of total nonaccidental mortality associated with acute exposure to O_3, especially in the summer or warm season when O_3 levels are typically high, with somewhat larger effect estimate sizes for associations with cardiovascular mortality.

Overall, the Criteria Document (p.8–78) finds that the results from U.S. multi-city time-series studies, along with the meta-analyses, provide relatively strong evidence for associations between short-term O_3 exposure and all-cause mortality even after adjustment for the influence of season and PM. The results of these analyses indicate that copollutants generally do not appear to substantially confound the association between O_3 and mortality. In addition, several single-city studies observed positive associations of ambient O_3 concentrations with total nonaccidental and cardiopulmonary mortality.

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; effect estimates for respiratory mortality are less consistent in size, possibly due to reduced statistical power in this subcategory of mortality. For cardiovascular mortality, the Criteria Document (p.7–106) suggests that effect estimates are consistently positive and more likely to be larger and statistically significant in warm season analyses. The Criteria Document (p.8–78) concludes that these findings are highly suggestive that short-term O_3 exposure directly or indirectly contributes to nonaccidental and cardiopulmonary-related mortality, but additional research is needed to more fully establish underlying mechanisms by which such effects occur.

b. Exposure- and Risk-Based Considerations

As discussed above in section II.B, the Staff Paper also estimated quantitative exposures and health risks associated with recent air quality levels and with air quality that meets the current standard to help inform judgments about whether or not the current standard provides adequate protection of public health. In so doing, it presented the important uncertainties and limitations associated with the exposure and risk assessments (discussed above in section II.B and more fully in chapters 4 and 5 of the Staff Paper).

The Staff Paper (and the CASAC) also recognized that the exposure and risk analyses could not provide a full picture of the O_3 exposures and O_3-related health risks posed nationally. The Staff Paper did not have sufficient information to evaluate all relevant at-risk groups (*e.g.*, outdoor workers) or all O_3-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 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 O_3 exposures are clearly much larger than the quantitative estimates of O_3-related incidences of adverse health effects and the numbers of children likely to experience exposures of concern associated with recent air quality or air quality that just meets the current 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.

As described above in section II.B, the Staff Paper estimated exposures and risks for the three most recent years (2002–2004) for which data were available at the time of the analyses. Within this 3-year period, 2002 was a year with relatively higher O_3 levels in most, but not all, areas and simulation of just meeting the current standard based on 2002 air quality data provides a generally more upper-end estimate of exposures and risks, while 2004 was a year with relatively lower O_3 levels in

most, but not all, areas and simulation of just meeting the current standard using 2004 air quality data provides a generally more lower-end estimate of exposures and risks.

i. Exposure Assessment Results

As discussed above in section II.B.1, the Staff Paper estimates personal exposures to ambient O_3 levels at and above specific benchmark levels to 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 current air quality levels, and the extent to which such impacts might be reduced by meeting the current and alternative standards. As described in greater detail in section II.B.1.c above, the Staff Paper refers to exposures at and above these benchmark levels as ''exposures of concern.'' The Staff Paper notes that exposures of concern, and the health outcomes they represent, likely occur across a range of O_3 exposure levels, such that there is no one exposure level that addresses all relevant public health concerns. Therefore, with the concurrence of the CASAC, the Staff Paper estimated exposures of concern not only at 0.080 ppm O_3, a level at which there are demonstrated effects, but also at 0.070 and 0.060 ppm O_3. The Staff Paper recognized that there will be varying degrees of concern about exposures at each of these levels, based in part on the population subgroups 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 O_3 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 O_3 concentrations, the Staff Paper, and the discussion below, focuses on exposures of concern at or above benchmark levels of 0.070 and 0.060 ppm O_3 for purposes of evaluating the adequacy of the current standard.

The exposure estimates presented in the Staff Paper are for the number and percent of all school age children and asthmatic school age 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. As shown in the Table 1 in this notice, the percent of population exposed at any given level is very similar for all and asthmatic school age children. Substantial year-to-year variability in exposure estimates is observed, ranging to over an order of magnitude at the current standard level, in estimates of the number of children and, as shown in Table 6–1a and b of the Staff Paper, the number of occurrences of exposures of concern at both of these benchmark levels. The Staff Paper states that it is appropriate to consider not just the average estimates across all years, but also to consider public health impacts in year with relatively higher O_3 levels. The 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 upon meeting the current standard.

As discussed in the Staff Paper (EPA, 2007b, see section 6.3.1.2), about 50 percent of asthmatic or all school age children, representing nearly 1.3 million asthmatic children and about 8.5 million school age children in the 12 urban areas examined, are estimated to experience exposures of concern at or above the 0.070 ppm benchmark level (*i.e.*, these individuals are estimated to experience 8-hour O_3 exposures at or above 0.070 ppm while engaged in moderate or greater exertion 1 or more times during the O_3 season) associated with 2002 O_3 air quality levels. In contrast, about 17 percent of asthmatic and all school age children are estimated to experience exposures of concern at or above the 0.070 ppm benchmark level associated with 2004 O_3 air quality levels. Just meeting the current standard results in an aggregate estimate of about 20 percent of asthmatic or 18 percent or all school age children likely to experience exposures of concern at or above the 0.070 ppm benchmark level using the 2002 simulation. The exposure estimates for this benchmark level range up to about 40 percent of asthmatic or all school age children in the single city with the least degree of protection from this standard. Just meeting the current standard based on the 2004 simulation, results in an aggregate estimate of about 1 percent of asthmatic or all school age children experiencing exposures at the 0.07 ppm benchmark level.

At the benchmark level of 0.060 ppm, about 70 percent of all or asthmatic school age children are estimated to experience exposures of concern at this benchmark level for the aggregate of the 12 urban areas associated with 2002 O_3 levels. Just meeting the current standard would result in an aggregate estimate of about 45 percent of asthmatic or all school age children likely to experience exposures of concern at or above the 0.060 ppm benchmark level using the 2002 simulation. The exposure estimates for this benchmark level range up to nearly 70 percent of all or asthmatic school age children in the single city with the least degree of protection associated with just meeting the current standard using the 2002 simulation. The Staff Paper indicates an aggregate estimate of about 10 percent of asthmatic or all school age children would experience exposures at or above the 0.06 ppm benchmark level associated with just meeting the current standard using the 2004 simulation.

ii. Risk Assessment Results

As described in more detail in section II.B.2 above and in chapters 5 and 6 of the Staff Paper, risk estimates have been developed for several important health endpoints, including: (1) Lung function decrements (*i.e.*, ≥15 percent and ≥20 percent reductions in FEV_1) in all school age children for 12 urban areas; (2) lung function decrements (*i.e.*, ≥10 percent and ≥20 percent reductions in FEV_1) in asthmatic school age children for 5 urban areas (a subset of the 12 urban areas); (3) respiratory symptoms (*i.e.*, chest tightness, shortness of breath, wheeze) in moderate to severe asthmatic children for the Boston area; (4) respiratory-related hospital admissions for 3 urban areas; and (5) nonaccidental and cardiorespiratory mortality for 12 urban areas for three recent years (2002 to 2004) and for just meeting the current standard using a 2002 simulation and a 2004 simulation.

With regard to estimates of moderate lung function decrements, as shown in Tables 6–2 of the Staff Paper, meeting the current standard substantially reduces the estimated number of school age children experiencing one or more occurrences of FEV_1 decrements ≥15 percent for the 12 urban areas, going from about 1.3 million children (7 percent of children) under 2002 air quality to about 610,000 (3 percent of children) based on the 2002 simulation, and from about 620,000 children (3 percent of children) to about 230,000 (1 percent of children) using the 2004 simulation. In asthmatic children, the estimated number of children experiencing one or more occurrences of FEV_1 decrements ≥10 percent for the 5 urban areas goes from about 250,000 children (16 percent of asthmatic children) under 2002 air quality to about 130,000 (8 percent of asthmatic children) using the 2002 simulation, and from about 160,000 (10 percent of asthmatic children) to about 70,000 (4 percent of asthmatic children) using the 2004 simulation. Thus, even when the

current standard is met, about 4 to 8 percent of asthmatic school age children are estimated to experience one or more occurrences of moderate lung function decrements, resulting in about 1 million occurrences (using the 2002 simulation) and nearly 700,000 occurrence (using the 2004 simulation) in just 5 urban areas. Moreover, the estimated number of occurrences of moderate or greater lung function decrements per child is on average approximately 6 to 7 in all children and 8 to 10 in asthmatic children in an O_3 season, even when the current standard is met, depending on the year used to simulate meeting the current standard. In the 1997 review of the O_3 standard a general consensus view of the adversity of such moderate responses emerged as the frequency of occurrences increases, with the judgment that repeated occurrences of moderate responses, even in otherwise healthy individuals, may be considered adverse since they may well set the stage for more serious illness.

With regard to estimates of large lung function decrements, the Staff Paper notes that FEV_1 decrements *>20 percent* would likely interfere with normal activities in many healthy individuals, therefore single occurrences would be considered to be adverse. In people with asthma, large lung function responses would likely interfere with normal activities for most individuals and would also increase the likelihood that these individuals would use additional medication or seek medical treatment. Not only would single occurrences be considered to be adverse to asthmatic individuals under the ATS definition, but they also would be cause for medical concern. While the current standard reduces the occurrences of large lung function decrements in all children and asthmatic children from about 60 to 70%, in a year with relatively higher O_3 levels (2002), there are estimated to be about 500,000 occurrences in all school children across the entire 12 urban areas, and about 40,000 occurrences in asthmatic children across just 5 urban areas. As noted above, it is clear that even when the current standard is met over a three-year period, O_3 levels in each year can vary considerably, as evidenced by relatively large differences between risk estimates based on 2002 to 2004 air quality. The Staff Paper expressed the view that it was appropriate to consider this yearly variation in O_3 levels allowed by the current standard in judging the extent to which impacts on members of at-risk groups in a year with relatively higher O_3 levels remains of concern from a public health perspective.

With regard to other O_3-related health effects, as shown in Tables 6–4 through 6–6 of the Staff Paper, the estimated risks of respiratory symptom days in moderate to severe asthmatic children, respiratory-related hospital admissions, and non-accidental and cardiorespiratory mortality, respectively, are not reduced to as great an extent by meeting the current standard as are lung function decrements. For example, just meeting the current standard reduces the estimated average incidence of chest tightness in moderate to severe asthmatic children living in the Boston urban area by 11 to 15%, based on 2002 and 2004 simulations, respectively, resulting in an estimated incidence of about 23,000 to 31,000 per 100,000 children attributable to O_3 exposure (Table 6–4). Just meeting the current standard is estimated to reduce the incidence of respiratory-related hospital admissions in the New York City urban area by about 16 to 18%, based on 2002 and 2004 simulations, respectively, resulting in an estimated incidence per 100,000 population of 4.6 to 6.4, respectively (Table 6–5). Across the 12 urban areas, the estimates of non-accidental mortality incidence per 100,000 relevant population range from 0.4 to 2.6 (for 2002) and 0.5 to 1.5 (for 2004) (Table 6–6). Meeting the current standard results in a reduction of the estimated incidence per 100,000 population to a range of 0.3 to 2.4 based on the 2002 simulation and a range of 0.3 to 1.2 based on the 2004 simulation. Estimates for cardiorespiratory mortality show similar patterns.

In considering the estimates of the proportion of population affected and the number of occurrences of the health effects that are included in the risk assessment, the Staff Paper notes that these limited estimates are indicative of a much broader array of O_3-related health endpoints 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, emergency department visits) and which primarily affect members of at-risk groups. While the Staff Paper had sufficient information to estimate and consider the number of symptom days in children with moderate to severe asthma, it recognized that there are many other effects that may be associated with symptom days, such as increased medication use, school and work absences, or visits to doctors' offices, for which there was not sufficient information to estimate risks but which are important to consider in assessing the adequacy of the current standard. The same is true for more serious, but less frequent effects. The Staff Paper estimated hospital admissions, but there was not sufficient information to estimate emergency department visits in a quantitative risk assessment. Consideration of such unquantified risks in the Staff Paper reinforces the Staff Paper conclusion that consideration should be given to revising the standard so as to provide increased public health protection, especially for at-risk groups such as people with asthma or other lung diseases, as well as children and older adults, particularly those active outdoors, and outdoor workers.

c. Summary

Based on the available information and taking into account the views of CASAC and public comments, the Staff Paper initially notes that all parties commenting on the NAAQS review agree that the standard should be at least as protective as the current standard, as no party suggested it should be revised to provide less protection. The Staff Paper concludes that the overall body of evidence clearly calls into question the adequacy of the current standard in protecting at-risk groups, notably including asthmatic children and other people with lung disease, as well as all children and older adults, especially those active outdoors, and outdoor workers,[48] against an array of adverse health effects that range from decreased lung function to serious indicators of respiratory morbidity including emergency department visits and hospital admissions for respiratory causes, nonaccidental mortality, and possibly cardiovascular effects. The available information provides strong support for consideration of an O_3 standard that would provide increased health protection for these at-risk groups. The Staff Paper also concludes that risks projected to remain upon meeting the current standard, based on the exposure and risk estimates discussed above and in more detail in the Staff Paper, are indicative of risks to at-risk groups that can be judged to be important from a public health perspective, which reinforces the Staff Paper conclusion that consideration should be given to revising the level of the standard so as to provide increased

[48] In defining at-risk groups this way we are including both groups with greater inherent sensitivity and those more likely to be exposed.

public health protection (EPA, 2007, section 6.3.6).

3. CASAC Views

In its letter to the Administrator, the CASAC O_3 Panel, with full endorsement of the chartered CASAC, unanimously concluded that there is ''no scientific justification for retaining'' the current primary O_3 standard, and the current standard ''needs to be substantially reduced to protect human health, particularly in sensitive subpopulations'' (Henderson, 2006c, pp. 1–2). In its rationale for this conclusion, the CASAC Panel concluded that ''new evidence supports and build-upon key, health-related conclusions drawn in the 1997 Ozone NAAQS review'' (id., p. 3). The Panel points to studies discussed in chapter 3 and Appendix 3B of the Staff Paper in noting that several new single-city studies and large multi-city studies have provided more evidence for adverse health effects at concentrations lower than the current standard, and that these epidemiological studies are backed-up by evidence from controlled human exposure studies. The Panel specifically noted evidence from the recent Adams (2006) study that reported statistically significant decrements in the lung function of healthy, moderately exercising adults at a 0.080 ppm exposure level, and importantly, also reported adverse lung function effects in some healthy individuals at 0.060 ppm. The Panel concluded that these results indicate that the current standard ''is not sufficiently health-protective with an adequate margin of safety,'' noting that that while similar studies in sensitive groups such as asthmatics have yet to be conducted, ''people with asthma, and particularly children, have been found to be more sensitive and to experience larger decrements in lung function in response to O_3 exposures than would healthy volunteers (Mortimer *et al.*, 2002)'' (Henderson, 2006c, p. 4).

The CASAC Panel also highlighted a number of O_3-related adverse health effects, that are associated with exposure to ambient O_3, below the level of the current standard, based on a broad range of epidemiological studies (Henderson, 2006c). These adverse health effects include increases in school absenteeism, respiratory hospital emergency department visits among asthmatics and patients with other respiratory diseases, hospitalizations for respiratory illnesses, symptoms associated with adverse health effects (including chest tightness and medication usage), and premature mortality (nonaccidental, cardiorespiratory deaths) reported at exposure levels well below the current standard. ''The CASAC considers each of these findings to be an important indicator of adverse health effects'' (Henderson, 2006c).

The CASAC Panel expressed the view that more emphasis should be placed on the subjects in controlled human exposure studies with FEV_1 decrements greater than 10 percent, which can be clinically significant, rather than on the relatively small average decrements. The Panel also emphasized significant O_3-related inflammatory responses and markers of injury to the epithelial lining of the lung that are independent of spirometric responses. Further, the Panel expressed the view that the Staff Paper did not place enough emphasis on serious morbidity (*e.g.*, hospital admissions) and mortality observed in epidemiology studies. On the basis of the large amount of recent data evaluating adverse health effects at levels at and below the current O_3 standard, it was the unanimous opinion of the CASAC Panel that the current primary O_3 standard is not adequate to protect human health, that the relevant scientific data do not support consideration of retaining the current standard, and that the current standard needs to be substantially reduced to be protective of human health, particularly in sensitive subpopulations (Henderson, 2006c, pp. 4–5).

Further, the CASAC letter noted that ''there is no longer significant scientific uncertainty regarding the CASAC's conclusion that the current 8-hour primary NAAQS must be lowered'' (Henderson, 2006c, p. 5). The Panel noted that a ''large body of data clearly demonstrates adverse human health effects at the current level'' of the standard, such that ''[R]etaining this standard would continue to put large numbers of individuals at risk for respiratory effects and/or significant impact on quality of life including asthma exacerbations, emergency room visits, hospital admissions and mortality'' (Henderson, 2006c). The Panel also noted that ''scientific uncertainty does exist with regard to the lower level of O_3 exposure that would be fully protective of human health,'' concluding that ''it is possible that there is no threshold for an O_3-induced impact on human health and that some adverse events may occur at policy-relevant background'' (Henderson, 2006c, p.5).

4. Administrator's Proposed Conclusions Concerning Adequacy of Current Standard

Based on the large body of evidence concerning the public health impacts of O_3 pollution, including significant new evidence concerning effects at O_3 concentrations below the level of the current standard, the Administrator proposes that the current standard does not protect public health with an adequate margin of safety and should be revised to provide additional public health protection. In considering whether the primary standard should be revised, the Administrator has carefully considered the conclusions contained in the Criteria Document, the rationale and recommendations contained in the Staff Paper, the advice and recommendations from the CASAC, and public comments to date. The Administrator notes that evidence of a range of respiratory-related morbidity effects seen in the last review has been considerably strengthened, both through toxicological and controlled human exposure studies as well as through many new panel and epidemiological studies.

In addition, new evidence from controlled human exposure and epidemiological studies identifies people with asthma as an important susceptible population for which estimates of respiratory effects in the general population likely underestimate the magnitude or importance of these effects. New evidence about mechanisms of toxicity more completely explains the biological plausibility of O_3-induced respiratory effects and is beginning to suggest mechanisms that may link O_3 exposure to cardiovascular effects. Further, there is now relatively strong evidence for associations between O_3 and total nonaccidental and cardiopulmonary mortality, even after adjustment for the influence of season and PM. Relative to the information that was available to inform the Agency's 1997 decision to set the current standard, the newly available evidence increases the Administrator's confidence that respiratory morbidity effects such as lung function decrements and respiratory symptoms are causally related to O_3 exposures, that indicators of respiratory morbidity such as emergency department visits and hospital admissions are causally related to O_3 exposures, and that the evidence is highly suggestive that O_3 exposures during the O_3 season contribute to premature mortality.

The Administrator judges that there is important new evidence demonstrating that exposures to O_3 at levels below the level of the current standard are associated with a broad array of adverse health effects, especially in at-risk populations. These at-risk populations include people with asthma or other lung diseases who are likely to experience more serious effects from

exposure to O_3. As discussed in section II.A.4 above, these groups also include children and older adults with increased susceptibility, as well as those who are likely to be vulnerable as a result of spending a lot of time outdoors engaged in physical activity, especially active children and outdoor workers.

Examples of this important new evidence include demonstration of O_3-induced lung function effects and respiratory symptoms in some healthy individuals down to the previously observed exposure level of 0.080 ppm, as well as very limited new evidence at exposure levels well below the level of the current standard. In addition, there is now epidemiological evidence of statistically significant O_3-related associations with lung function and respiratory symptom effects, respiratory-related emergency department visits and hospital admissions, and increased mortality, in areas that likely would have met the current standard. There are also many epidemiological studies done in areas that likely would not have met the current standard but which nonetheless report statistically significant associations that generally extend down to ambient O_3 concentrations that are below the level of the current standard. Further, there are a few studies that have examined subsets of data that include only days with ambient O_3 concentrations below the level of the current standard, or below even much lower O_3 concentrations, and continue to report statistically significant associations with respiratory morbidity outcomes and mortality. The Administrator recognizes that the evidence from controlled human exposure studies, together with animal toxicological studies, provides considerable support for the biological plausibility of the respiratory morbidity associations observed in the epidemiological studies and for concluding that the associations extend below the level of the current standard.

Based on the strength of the currently available evidence of adverse health effects, and on the extent to which the evidence indicates that such effects result from exposures to ambient O_3 concentrations below the level of the current standard, the Administrator judges that the current standard does not protect public health with an adequate margin of safety and that the standard should be revised to provide such protection, especially for at-risk groups, against a broad array of adverse health effects.

In reaching this judgment, the Administrator has also considered the results of both the exposure and risk assessments conducted for this review, to provide some perspective on the extent to which at-risk groups would likely experience ''exposures of concern'' [49] and on the potential magnitude of the risk of experiencing various adverse health effects when recent air quality data (from 2002 to 2004) are used to simulate meeting the current standard and alternative standards in a number of urban areas in the U.S.[50] In considering the exposure assessment results, the Administrator is relying on analyses that define exposures of concern by three benchmark exposure levels: 0.080, 0.070, and 0.060 ppm. Estimates of exposures of concern in at-risk groups at and above these benchmark levels, using O_3 air quality data in 2002 and 2004, provide some indication of the potential magnitude of the incidence of health outcomes that cannot currently be evaluated in a quantitative risk assessment, such as increased airway responsiveness, increased pulmonary inflammation, including increased cellular permeability, and decreased pulmonary defense mechanisms. These physiological effects have been demonstrated to occur in healthy people at O_3 exposures as low as 0.080 ppm, the lowest level tested. They are associated with aggravation of asthma, increased medication use, increased school and work absences, increased susceptibility to respiratory infection, increased visits to doctors' offices and emergency departments, increased admissions to hospitals, and possibly to cardiovascular system effects and chronic effects such as chronic bronchitis or long-term damage to the lungs that can lead to reduced quality of life.

In considering these various benchmark levels for exposures of concern, the Administrator has focused primarily on estimated exposures at and above the 0.070 ppm benchmark level as an important surrogate measure for potentially more serious health effects in at-risk groups such as people with asthma. This judgment is based on the strong evidence of effects in healthy people at the 0.080 ppm exposure level and the new evidence that people with asthma are likely to experience larger and more serious effects than healthy people at the same level of exposure. In the Administrator's view, this evidence does not support a focus on exposures at and above the benchmark level of 0.080 ppm O_3, as it would not adequately account for the increased risk of harm from exposure for members of at-risk groups, especially people with asthma. The Administrator also judges that the evidence of demonstrated effects is too limited to support a primary focus on exposures down to the lowest benchmark level considered of 0.060 ppm. The Administrator particularly notes that although the analysis of ''exposures of concern'' was conducted to estimate exposures at and above three discrete benchmark levels (0.080, 0.070, and 0.060 ppm), the concept is appropriately viewed as a continuum. As discussed at the outset in section II.A above, the Administrator strives to balance concern 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 O_3 exposure levels.

The Administrator observes that based on the aggregate exposure estimates for the 2002 simulation summarized above in Table 1 (section II.B.1) and in the Staff Paper (EPA, 2007b, Table 6–7) for the 12 U.S. urban areas included in the exposure analysis, upon just meeting the current standard up to about 20 percent of asthmatic or all school age children are likely to experience one or more exposures of concern at and above the 0.070 ppm benchmark level; the 2004 simulation yielded an estimate of about 1 percent of such children. The Administrator notes from this comparison that there is substantial year-to-year variability, ranging up to an order of magnitude or more in estimates of the number of people and the number of occurrences of exposures of concern at and above this benchmark level. Moreover, within any given year, the exposure assessment indicates that there is substantial city-to-city variability in the estimates of the children exposed or the number of occurrences of exposure at and above this benchmark level. For example, city-specific estimates of the percent of asthmatic or all school age children likely to experience exposures at and above the benchmark level of 0.070 ppm

[49] As discussed in section II.B.1.c above, ''exposures of concern'' are estimates of personal exposures while at moderate or greater exertion to 8-hour average ambient O_3 levels at and above specific benchmark levels which represent exposure levels at which O_3-related health effects are known or can with varying degrees of certainty be inferred to occur in some individuals. Estimates of exposures of concern provide some perspective on the public health impacts of health effects that may occur in some individuals at recent air quality levels but cannot be evaluated in quantitative risk assessments, and the extent to which such impacts might be reduced by meeting the current and alternative standards.

[50] As described in the Staff Paper (section 4.5.8) and discussed above, recent O_3 air quality distributions have been statistically adjusted to simulate just meeting the current and selected alternative standards. These simulations do not represent predictions of when, whether, or how areas might meet the specified standards.

ranges from about 1 percent up to about 40 percent across the 12 urban areas upon just meeting the current standard based on the 2002 simulation; the 2004 simulation yielded estimates that range from about 0 up to about 7 percent. The Administrator judges it is important to recognize the substantial year-to-year and city-to-city variability in considering these estimates.

With regard to the results of the risk assessment, as discussed above, the Administrator recognizes that a simulation of just meeting the current standard in the cities included in the assessment indicate that the estimated risk is lower for all of the health endpoints evaluated. In considering the adequacy of the current standard, the Administrator has focused on the risks estimated to remain upon just meeting the current standard. Based on the aggregate risk estimates summarized above in Table 2 (section II.B.2 of this notice), the Administrator observes that upon just meeting the current standard based on the 2002 simulation, approximately 8 percent of asthmatic school age children across 5 urban areas (ranging up to about 11 percent in the city that receives relatively less protection) and approximately 3 percent of all school age children across 12 urban areas (ranging up to over 5 percent in the city that receives relatively less protection) would still be estimated to experience moderate or greater lung function decrements one or more times within an O_3 season. The Administrator recognizes that, as with the estimates of exposures of concern, there is substantial year-to-year and city-to-city variability in these risk estimates.

In addition to the percentage of asthmatic or all children estimated to experience 1 or more occurrences of an effect, the Administrator recognizes that some individuals are estimated to have multiple occurrences. For example, across all the cities in the assessment, approximately 6 to 7 occurrences of moderate or greater lung function decrements per child are estimated to occur in all children and approximately 8 to 10 occurrences are estimated to occur in asthmatic children in an O_3 season, even upon just meeting the current standard. In the last review, a general consensus view of the adversity of such responses emerged as the frequency of occurrences increases, with the judgment that repeated occurrences of moderate responses, even in otherwise healthy individuals, may be considered adverse since they may well set the stage for more serious illness. The Administrator continues to support this view.

Large lung function decrements (*i.e.*, ≥20 percent FEV_1 decrement) would likely interfere with normal activities in many healthy individuals, therefore single occurrences would be considered to be adverse. In people with asthma, large lung function responses (*i.e.*, ≥ 20 percent FEV_1 decrement), would likely interfere with normal activities for most individuals and would also increase the likelihood that these individuals would use additional medication or seek medical treatment. Not only would single occurrences be considered to be adverse to asthmatic individuals under the ATS definition, but they also would be cause for medical concern for some individuals. Upon just meeting the current standard based on the 2002 simulation, close to 1 percent of asthmatic and all school age children are estimated to experience one or more occurrences of large lung function decrements in the aggregate across 5 and 12 urban areas, respectively, with close to 2 percent of both asthmatic and all school age children estimated to experience such effects in the city that receives relatively less protection from this standard. These estimates translate into approximately 500,000 occurrences of large lung function decrements in all children across 12 urban areas, and about 40,000 occurrences in asthmatic children across just 5 urban areas upon just meeting the current standard based on the 2002 simulation; the 2004 simulation yielded estimates that translate into approximately 160,000 and 10,000 such occurrences in all children and asthmatic children, respectively.

Upon just meeting the current standard based on the 2002 simulation, the estimate of the O_3-related risk of respiratory symptom days in moderate to severe asthmatic children in the Boston area is about 8,000 symptom days; the 2004 simulation yielded an estimate of about 6,000 such symptoms days. These estimates translate into as many as one symptom day in 6, and one symptom day in 8, respectively, that are attributable to O_3 exposure during the O_3 season of the total number of symptom days associated with all causes of respiratory symptoms in asthmatic children during those years.

The estimated O_3-related risk of respiratory-related hospital admissions upon just meeting the current standard based on the 2002 simulation is greater than 500 hospital admissions in the New York City area alone, or about 1.5 percent of the total incidence of respiratory-related admissions associated with all causes; the 2004 simulation yielded an estimate of approximately 400 such hospital admissions. For nonaccidental mortality, just meeting the current standard based on the 2002 simulation results in an estimated incidence of from 0.3 to 2.4 per 100,000 population; the 2004 simulation resulted in an estimated incidence of from 0.3 to 1.2 per 100,000 population. Estimates for cardiorespiratory mortality show similar patterns. (Abt Associates, 2007, Table 4–26).

The Administrator recognizes that in considering the estimates of the proportion of population affected and the number of occurrences of those specific health effects that are included in the risk assessment, these limited estimates based on 2002 and 2004 simulations are indicative of a much broader array of O_3-related health endpoints that are part of a ''pyramid of effects'' (discussed above in section II.A.4.d) that include various indicators of morbidity that could not be included in the risk assessment (*e.g.*, school absences, increased medication use, emergency department visits) and which primarily affect members of at-risk groups. Moreover, the Administrator notes that the CASAC Panel supported a qualitative consideration of the much broader array of O_3-related health endpoints, and specifically referred to respiratory emergency department visits in asthmatics and people with other lung diseases, increased medication use, and increased respiratory symptoms reported at exposure levels well below the current standard.

The Administrator believes the exposure and risk estimates discussed in the Staff Paper and summarized above are important from a public health perspective and are indicative of potential exposures and risks to at-risk groups. In reaching this proposed judgment, the Administrator considered the following factors: (1) The estimates of numbers of persons exposed at and above the 0.070 ppm benchmark level; (2) the risk estimates of the proportion of the population and number of occurrences of various health effects in areas upon just meeting the current standard; (3) the year-to-year and city-to-city variability in both the exposure and risk estimates; (4) the uncertainties in these estimates; and (5) recognition that there is a broader array of O_3-related adverse health outcomes for which risk estimates could not be quantified (that are part of a broader ''pyramid of effects'') and that the scope of the assessment was limited to just a sample of urban areas and to some but not all at-risk populations, leading to an incomplete estimation of public health impacts associated with O_3 exposures

across the country. The Administrator also notes that it was the unanimous conclusion of the CASAC Panel that there is no scientific justification for retaining the current primary O_3 standard, that the current standard is not sufficiently health-protective with an adequate margin of safety, and that the standard needs to be substantially reduced to protect human health, particularly in at-risk subpopulations.

Based on all of these considerations, the Administrator proposes that the current O_3 standard is not requisite to protect public health with an adequate margin of safety because it does not provide sufficient protection and that revision would result in increased public health protection, especially for members of at-risk groups.

D. Conclusions on the Elements of the Primary Standard

1. Indicator

In the last review EPA focused on a standard for O_3 as the most appropriate surrogate for ambient photochemical oxidants. In this review, while the complex atmospheric chemistry in which O_3 plays a key role has been highlighted, no alternative to O_3 has been advanced as being a more appropriate surrogate for ambient photochemical oxidants.

The Staff Paper (section 2.2.2) notes that it is generally recognized that control of ambient O_3 levels provides the best means of controlling photochemical oxidants. Among the photochemical oxidants, the acute exposure chamber, panel, and field epidemiological human health database provides specific evidence for O_3 at levels commonly reported in the ambient air, in part because few other photochemical oxidants are routinely measured. However, recent investigations on copollutant interactions have used simulated urban photochemical oxidant mixes. These investigations suggest the need for similar studies to help in understanding the biological basis for effects observed in epidemiological studies that are associated with air pollutant mixtures, where O_3 is used as the surrogate for the mix of photochemical oxidants. Meeting the O_3 standard can be expected to provide some degree of protection against potential health effects that may be independently associated with other photochemical oxidants but which are not discernable from currently available studies indexed by O_3 alone. Since the precursor emissions that lead to the formation of O_3 generally also lead to the formation of other photochemical oxidants, measures leading to reductions in population exposures to O_3 can generally be expected to lead to reductions in population exposures to other photochemical oxidants.

The Staff Paper notes that while the new body of time-series epidemiological evidence cannot resolve questions about the relative contribution of other photochemical oxidant species to the range of morbidity and mortality effects associated with O_3 in these types of studies, control of ambient O_3 levels is generally understood to provide the best means of controlling photochemical oxidants in general, and thus of protecting against effects that may be associated with individual species and/or the broader mix of photochemical oxidants, independent of effects specifically related to O_3.

In its letter to the Administrator, the CASAC O_3 Panel noted that O_3 is ''the key indicator of the extent of oxidative chemistry and serves to integrate multiple pollutants.'' CASAC also stated that ''although O_3 itself has direct effects on human health and ecosystems, it can also be considered as an indicator of the mixture of photochemical oxidants and of the oxidizing potency of the atmosphere'' (Henderson, 2006c, p. 9).

Based on the available information, and consistent with the views of EPA staff and the CASAC, the Administrator proposes to continue to use O_3 as the indicator for a standard that is intended to address effects associated with exposure to O_3, alone or in combination with related photochemical oxidants. In so doing, the Administrator recognizes that measures leading to reductions in population exposures to O_3 will also reduce exposures to other photochemical oxidants.

2. Averaging Time

a. Short-Term and Prolonged (1 to 8 Hours)

The current 8-hour averaging time for the primary O_3 NAAQS was set in 1997. At that time, the decision to revise the averaging time of the primary standard from 1 to 8 hours was supported by the following key observations and conclusions:

(1) The 1-hour averaging time of the previous NAAQS was originally selected primarily on the basis of health effects associated with short-term (*i.e.*, 1- to 3-hour) exposures.

(2) Substantial health effects information was available for the 1997 review that demonstrated associations between a wide range of health effects (*e.g.*, moderate to large lung function decrements, moderate to severe symptoms and pulmonary inflammation) and prolonged (*i.e.*, 6- to 8-hour) exposures below the level of the then current 1-hour NAAQS.

(3) Results of the quantitative risk analyses showed that reductions in risks from both short-term and prolonged exposures could be achieved through a primary standard with an averaging period of either 1 or 8 hours. Thus establishing both a 1-hour and an 8-hour standard would not be necessary to reduce risks associated with the full range of observed health effects.

(4) The 8-hour averaging time is more directly associated with health effects of concern at lower O_3 concentrations than the 1-hour averaging time. It was thus the consensus of CASAC ''that an 8-hour standard was more appropriate for a human health-based standard than a 1-hour standard.'' (Wolff, 1995)

(5) An 8-hour averaging results in a significantly more uniformly protective national standard than the then current 1-hour standard.

(6) An 8-hour averaging time effectively limits both 1- and 8-hour exposures of concern.

In looking at the new information that is discussed in section 7.6.2 of the current Criteria Document, the Staff Paper noted that epidemiological studies have used various averaging periods for O_3 concentrations, most commonly 1-hour, 8-hour and 24-hour averages. As described more specifically in sections 3.3 and 3.4 of the Staff Paper, in general the results presented from U.S. and Canadian studies show no consistent difference for various averaging times in different studies. Because the 8-hour averaging time continues to be more directly associated with health effects of concern from controlled human exposure studies at lower concentrations than do shorter averaging periods, the Staff Paper did not evaluate alternative averaging times in this review and did not conduct exposure or risk assessments for standards with averaging times other than 8 hours.

The Staff Paper discusses an analysis of a recent three-year period of air quality data (2002 to 2004) which was conducted to determine whether the comparative 1- and 8-hour air quality patterns that were observed in the last review continue to be observed based on more recent air quality data. This updated air quality analysis (McCluney, 2007) is very consistent with the analysis done in the last review in that it indicates that only two urban areas of the U.S. have such ''peaky'' air quality patterns such that the ratio of 1-hour to 8-hour design values is greater than 1.5. This suggests that, based on recent air quality data, it is reasonable to again conclude that an 8-hour average

standard at or below the current level would generally be expected to provide protection equal to or greater than the previous 1-hour standard of 0.12 ppm in almost all urban areas. Thus, the Staff Paper again concluded that setting a standard with an 8-hour averaging time can effectively limit both 1- and 8-hour exposures of concern and is appropriate to provide adequate and more uniform protection of public health from both short-term and prolonged exposures to O_3 in the ambient air.

In its letter to the Administrator, the CASAC O_3 Panel supported the continued use of an 8-hour averaging time for the primary O_3 standard (Henderson, 2006c, p. 2), as did many commenters. Some other commenters expressed the view that consideration should be given to setting or reinstating a 1-hour standard, in addition to maintaining the use of an 8-hour averaging time, to protect people in those parts of the country with relatively more ''peaky'' exposure profiles. These commenters point out that when controlled exposure studies using triangular exposure patterns (with relatively higher 1-hour peaks) have been compared to constant exposure patterns with the same aggregate O_3 dose (in terms of concentration x time), ''peaky'' exposure patterns are seen to lead to higher risks. The California Air Resources Board made particular note of this point, expressing the view that a 1-hour standard would more closely represent actual exposures, in that many people spend only 1 to 2 hours a day outdoors, and that it would be better matched to O_3 concentration profiles along the coasts where O_3 levels are typically high for shorter averaging periods than 8 hours.

b. Long-term

During the last review, there was a large animal toxicological database for consideration that provided clear evidence of associations between long-term (*e.g.*, from several months to years) exposures and lung tissue damage, with additional evidence of reduced lung elasticity and accelerated loss of lung function. However, there was no corresponding evidence for humans, and the state of the science had not progressed sufficiently to allow quantitative extrapolation of the animal study findings to humans. For these reasons, consideration of a separate long-term primary O_3 standard was not judged to be appropriate at that time, recognizing that the 8-hour standard would act to limit long-term exposures as well as short-term and prolonged exposures.

Taking into consideration the currently available evidence on long-term O_3 exposures, discussed above in section II.A.2.a.ii, the Staff Paper concludes that a health-based standard with a longer-term averaging time than 8 hours is not warranted at this time. The Staff Paper notes that, while potentially more serious health effects have been identified as being associated with longer-term exposure studies of laboratory animals and in epidemiology studies, there remains substantial uncertainty regarding how these data could be used quantitatively to develop a basis for setting a long-term health standard. Because long-term air quality patterns would be improved in areas coming into attainment with an 8-hour standard, the potential risk of health effects associated with long-term exposures would be reduced in any area meeting an 8-hour standard. Thus, the Staff Paper did not recommend consideration of a long-term, health-based standard at this time.

In its final letter to the Administrator, the CASAC O_3 Panel offered no views on the long-term exposure evidence, nor did it suggest that consideration of a primary O_3 standard with a long-term averaging time was appropriate. In fact, the CASAC O_3 Panel agreed with the choice of an 8-hour averaging time for the primary O_3 NAAQS suggested by Agency staff (Henderson, 2007). Similarly, no commenters expressed support for considering such a long-term standard.

c. Administrator's Conclusions on Averaging Time

In considering the information discussed above, CASAC views and public comments, the Administrator concludes that a standard with an 8-hour averaging time can effectively limit both 1- and 8-hour exposures of concern and that an 8-hour averaging time is appropriate to provide adequate and more uniform protection of public health from both short-term (1- to 3-hour) and prolonged (6- to 8-hour) exposures to O_3 in the ambient air. This conclusion is based on the observations summarized above, particularly: (1) The fact that the 8-hour averaging time is more directly associated with health effects of concern at lower O_3 concentrations than are averaging times of shorter duration and (2) results from quantitative risk analyses showing that attaining an 8-hour standard reduces the risk of experiencing health effects associated with both 8-hour and shorter duration exposures. Furthermore, the Administrator observes that the CASAC O_3 Panel agreed with the choice of averaging time (Henderson, 2007). Therefore, the Administrator proposes to retain the 8-hour averaging time and is not proposing a separate 1-hour standard. The Administrator also concludes that a standard with a long-term averaging time is not warranted at this time.

3. Form

In 1997, the primary O_3 NAAQS was changed from a ''1-expected-exceedance'' form per year over three years [51] to a concentration-based statistic, specifically the 3-year average of the annual fourth-highest daily maximum 8-hour concentrations. The principal advantage of the concentration-based form is that it is more directly related to the ambient O_3 concentrations that are associated with the health effects. With a concentration-based form, days on which higher O_3 concentrations occur would weigh proportionally more than days with lower concentrations, since the actual concentrations are used in determining whether the standard is attained. That is, given that there is a continuum of effects associated with exposures to varying levels of O_3, the extent to which public health is affected by exposure to ambient O_3 is related to the actual magnitude of the O_3 concentration, not just whether the concentration is above a specified level.

During the 1997 review, consideration was given to a range of alternative forms, including the second-, third-, fourth- and fifth-highest daily maximum 8-hour concentrations in an O_3 season, recognizing that the public health risks associated with exposure to a pollutant without a clear, discernable threshold can be appropriately addressed through a standard that allows for multiple exceedances to provide increased stability, but that also significantly limits the number of days on which the level may be exceeded and the magnitude of such exceedances. Consideration was given to setting a standard with a form that would provide a margin of safety against possible, but uncertain chronic effects, and would also provide greater stability to ongoing control programs. The fourth-highest daily maximum was selected because it was decided that the differences in the degree of protection against potential chronic effects afforded by the alternatives within the range were not well enough understood to use any such differences as a basis for

[51] The 1-expected-exceedance form essentially requires that the fourth-highest air quality value in 3 years, based on adjustments for missing data, be less than or equal to the level of the standard for the standard to be met at an air quality monitoring site.

choosing the most restrictive forms. On the other hand, the relatively large percentage of sites that would experience O_3 peaks well above 0.080 ppm and the number of days on which the level of the standard may be exceeded, even when attaining a fifth-highest 0.080 ppm concentration-based standard, argued against choosing that form.

As an initial matter, the Staff Paper considered whether it is appropriate to continue to specify the level of the O_3 standard to the nearest hundredth (two decimal places) ppm, or whether the precision with which ambient O_3 concentrations are measured supports specifying the standard level to the thousandth ppm (*i.e.*, to the part per billion (ppb)). The Staff Paper discusses an analysis conducted by EPA staff to determine the impact of ambient O_3 measurement error on calculated 8-hour average O_3 design value concentrations, which are compared to the level of the standard to determine whether the standard is attained (Cox and Camalier, 2006). The results of this analysis suggest that instrument measurement error, or possible instrument bias, contribute very little to the uncertainty in design values. More specifically, measurement imprecision was determined to contribute less than 1 ppb to design value uncertainty, and a simulation study indicated that randomly occurring instrument bias could contribute approximately 1 ppb. EPA staff interpreted this analysis as being supportive of specifying the level of the standard to the nearest thousandth ppm. If the current standard were to be specified to this degree of precision, the current standard would effectively be at a level of 0.084 ppm, reflecting the data rounding conventions that are part of the definition of the current 0.080 ppm 8-hour standard. This information was provided to the CASAC O_3 Panel and made available to the public.

In evaluating alternative forms for the primary standard in conjunction with specific standard levels, the Staff Paper considered the adequacy of the public health protection provided by the combination of the level and form to be the foremost consideration. In addition, the Staff Paper recognized that it is important to have a form of the standard that is stable and insulated from the impacts of extreme meteorological events that are conducive to O_3 formation. Such instability can have the effect of reducing public health protection, because frequent shifting in and out of attainment due to meteorological conditions can disrupt an area's ongoing implementation plans and associated control programs. Providing more stability is one of the reasons that EPA moved to a concentration-based form in 1997.

The Staff Paper considered two concentration-based forms of the standard: the nth-highest maximum concentration and a percentile-based form. A percentile-based statistic is useful for comparing datasets of varying length because it samples approximately the same place in the distribution of air quality values, whether the dataset is several months or several years long. However, a percentile-based form would allow more days with higher air quality values in locations with longer O_3 seasons relative to places with shorter O_3 seasons. An nth-highest maximum concentration form would more effectively ensure that people who live in areas with different length O_3 seasons receive the same degree of public health protection. For this reason, the exposure and risk analyses were based on a form specified in terms of an nth-highest concentration, with n ranging from 3 to 5.

The results of some of these analyses are shown in the Staff Paper (Figures 6–1 through 6–4) and specifically discussed in chapter 6. These figures illustrate the estimated percent change in risk estimates for the incidence of moderate or greater decrements in lung function (≥15 percent FEV_1) in all school age children and moderate or greater lung function decrements (≥10 percent FEV_1) in asthmatic school age children, associated with going from meeting the current standard to meeting alternative standards with alternative forms based on the 2002 and 2004 simulations. Figures 6–5 and 6–6 illustrate the estimated percent of change in the estimated incidence of non-accidental mortality, associated with going from meeting the current standard to meeting alternative standards, based on the 2002 and 2004 simulations. These results are generally representative of the patterns found in all of the analyses. The estimated reductions in risk associated with different forms of the standard, ranging from third- to fourth-highest daily maximum concentrations at 0.084 ppm, and from third- to fifth-highest daily maximum concentrations at 0.074 ppm, are generally less than the estimated reductions associated with the different levels that were analyzed. As seen in these figures, there is much city-to-city variability, particularly in the percent changes associated with going from a fourth-highest to third-highest form at the current level of 0.084 ppm, and with estimated reductions associated with the fifth-highest form at a 0.074 ppm level. In most cities, there are generally only small differences in the estimated reductions in risks associated with the third- to fifth-highest forms at a level of 0.074 ppm simulated using 2002 and 2004 O_3 monitoring data.

The Staff Paper noted that there is not a clear health-based threshold for selecting a particular nth-highest daily maximum form of the standard from among the ones analyzed. It also noted that the changes in the form considered in the analyses result in only small differences in the estimated reductions in risks in most cities, although in some cities larger differences are estimated. The Staff Paper concluded that a range of concentration-based forms from the third- to the fifth-highest daily maximum 8-hour average concentration is appropriate for consideration in setting the standard. Given that there is a continuum of effects associated with exposures to varying levels of O_3, the extent to which public health is affected by exposure to ambient O_3 is related to the actual magnitude of the O_3 concentration, not just whether the concentration is above a specified level. The principal advantage of a concentration-based form is that it is more directly related to the ambient O_3 concentrations that are associated with health effects. Robust, concentration-based forms, in the range of the third- to fifth-highest daily maximum 8-hour average concentration, including the current 4th-highest daily maximum form, minimize the inherent lack of year-to-year stability of exceedance-based forms and provide insulation from the impacts of extreme meteorological events. Such instability can have the effect of reducing public health protection by disrupting ongoing implementation plans and associated control programs.

With regard to the precision of the standard, in their letter to the Administrator, the CASAC concluded that current monitoring technology ''allows accurate measurement of O_3 concentrations with a precision of parts per *billion*'' (Henderson, 2006c). The CASAC recommended that the specification of the level of the O_3 standard should reflect this degree of precision (Henderson, 2006c). Some public comments supported specifying the standard in terms of parts per billion, or to three decimal places if specified in terms of parts per million.[52] Other public commenters stated that the

[52] The Staff Paper notes that the 8-hour O_3 standard adopted by the State of California in 2006 is specified to the nearest thousandth part per million (at a level of 0.070 ppm) (*http://www.arb.ca.gov/research/aaqs/ozone-rs/ozone-rs.htm*).

basis for changing the current rounding procedures is not supported by a complete analysis of the O_3 compliance monitoring procedures, including consideration of uncertainty related to humidity effects and interferences from aromatic compounds in the monitoring of O_3 levels.

With regard to the form of the standard, in their letter to the Administrator, CASAC recommended that ''*a range of concentration-based forms from the third-to the fifth-highest daily maximum 8-hour average concentration*'' be considered (Henderson, 2006c, p. 5). Some public commenters that expressed the view that the current primary O_3 standard is not adequate also submitted comments that supported a more health-protective form of the standard than the current form (*e.g.*, a second-or third-highest daily maximum form). Commenters who expressed the view that the current standard is adequate did not provide any views on alternative forms that would be appropriate for consideration should the Administrator consider revisions to the standard.

The Administrator proposes that the level of the standard be specified to the nearest thousandth ppm, based on the staff's analysis and conclusions discussed in the Staff Paper that current monitoring technology allows accurate measurement of O_3 to support specifying the 8-hour standard to this degree of precision, and on CASAC's recommendation with respect to this aspect of the standard. The Administrator invites comment on this proposal to specify the standard to the thousandth ppm.

The Administrator recognizes that there is not a clear health-based threshold for selecting a particular nth-highest daily maximum form of the standard from among the ones analyzed in the Staff Paper, and that the current form of the standard provides a stable target for implementing programs to improve air quality. The Administrator also agrees that the adequacy of the public health protection provided by the combination of the level and form is a foremost consideration. Based on this, the Administrator proposes to retain the form of the current standard, 4th-highest daily maximum 8-hour average concentration, recognizing that the public health protection that would be provided by the standard is based on combining this form with the level discussed below. Mindful of the recommendation of the O_3 CASAC Panel and the view expressed by commenters, the Administrator also invites comment on two alternative forms of the standard, the third- and the fifth-highest daily maximum 8-hour average concentrations.

4. Level

a. Evidence and Exposure/Risk Based Considerations in the Staff Paper

The approach used in the Staff Paper as a basis for staff recommendations on standard levels builds upon and broadens the general approach used by EPA in the last review. This approach reflects the more extensive and stronger body of evidence now available on a broader range of health effects associated with exposure to O_3, including: (1) Additional respiratory-related endpoints; (2) new information about the mechanisms underlying respiratory morbidity effects supporting a judgment that the link between O_3 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 O_3 can directly or indirectly contribute to cardiovascular morbidity, and (4) new U.S. multi-city time series studies, single city studies, and several meta-analyses of these studies that provide relatively strong evidence for associations between short-term O_3 exposures and all-cause (nonaccidental) mortality, at levels below the current primary standard: as well as (5) evidence of increased susceptibility in people with asthma and other lung diseases. In evaluating evidence-based and exposure/risk-based considerations, the 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 O_3 and other photochemical oxidants, and the uncertainties associated with the estimated exposure and risk reductions.

In taking into account evidence-based considerations, the 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 O_3 concentrations observed in the studies or some indication of potential effect thresholds in terms of 8-hour average O_3 concentrations.

In considering the available controlled human exposure studies, as discussed above in section II.A.2.a.i(a)(i), two new studies are notable in that they are the only controlled human exposure studies that examined respiratory effects, including lung function decrements and respiratory symptoms, in healthy adults at lower exposure levels than had previously been examined. EPA's reanalysis of the data from the most recent study shows small group mean decrements in lung function responses to be statistically significant at the 0.060 ppm exposure level, while the author's analysis did not yield statistically significant lung function responses (but did yield some statistically significant respiratory symptom responses toward the end of the exposure period). Notably, these studies report a small percentage of subjects experiencing lung function decrements (> 10 percent) at the 0.060 ppm exposure level. These studies provide very limited evidence of O_3-related lung function decrements and respiratory symptoms at this lower exposure level.

In considering controlled human exposure studies of pulmonary inflammation, airway responsiveness, and impaired host defense capabilities, the Staff Paper notes 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. 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 O_3 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 O_3-related morbidity effects, which are consistent with and lend plausibility to the adverse morbidity effects and mortality effects observed in epidemiological studies.

In considering epidemiological studies, the Staff Paper first recognizes 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 current O_3 standard and

possibly within the range of background levels. As discussed above (and more fully in the Staff Paper in chapter 3 and the Criteria Document in chapter 7), a number of 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 up to approximately 0.050 ppm. Other studies, however, observe linear concentration-response functions suggesting no effect threshold. The Staff Paper concludes that the statistically significant associations between ambient O_3 concentrations and lung function decrements, respiratory symptoms, indicators of respiratory morbidity including increased emergency department visits and hospitals admissions, and possibly mortality reported in a large number of studies likely extend down to ambient O_3 concentrations that are well below the level of the current standard (EPA, 2007, p. 6–60). Toward the lower end of the range of O_3 concentrations observed in such studies, ranging down to background levels, however, the Staff paper states that there is increasing uncertainty as to whether the observed associations remain plausibly related to exposures to ambient O_3, rather than to the broader mix of air pollutants present in the ambient atmosphere.

The Staff Paper also considered studies that did subset analyses that include only days with ambient O_3 concentrations below the level of the current standard, or below even lower O_3 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 associations even when only days with a maximum 8-hour average O_3 concentration below a value of approximately 0.061 ppm were included.[53] Also of note is the large multi-city NCICAS (Mortimer *et al.*, 2002) that reported statistically significant associations between ambient O_3 concentrations and lung function decrements even when days with 8-hour average O_3 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).

Being mindful of the uncertainties and limitations inherent in interpreting the available evidence, the Staff Paper states the view that the range of alternative O_3 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 O_3 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 Staff Paper also concludes that the lower end to the range of alternative O_3 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.

In addition to the evidence-based considerations informing staff recommendations on alternative levels, the Staff Paper also evaluated quantitative exposures and health risks estimated to occur upon meeting the current and alternative standards.[54] In so doing, it presented the important uncertainties and limitations associated with these exposure and risk assessments. For example, the Staff Paper noted important uncertainties affecting the exposure estimates are related to modeling human activity patterns over an O_3 season (especially repetitive exposures), modeling ambient concentrations near roadways and modeling building air exchange rates which impact estimates of indoor O_3 concentrations. With regard to the risk assessment, important uncertainties include, for example, those related to exposure estimates for children engaged in moderate or greater exertion, as well as those related to estimation of concentration-response functions, specification of concentration-response models, the possible role of copollutants in interpreting reported associations with O_3, and inferences of a likely causal relationship between O_3 exposure and nonaccidental mortality (for risk estimates based on epidemiological studies).

Beyond these uncertainties, the Staff Paper also recognized important limitations to the exposure and risk analyses. For example, the Staff Paper did not have sufficient information to evaluate all relevant at-risk groups (*e.g.*, outdoor workers) or all O_3-related health outcomes (*e.g.*, increased medication use, school absences, emergency department visits), and the scope of the 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 risk analyses. The Staff Paper notes that it is clear that national-scale public health impacts of ambient O_3 exposures are much larger than the quantitative estimates of O_3-related incidences of adverse health effects and the numbers of children likely to experience exposures of concern associated with meeting the current or alternative standards. On the other hand, due to individual variability in responsiveness, only a subset of individuals who are estimated to experience exposures of concern at and above a specific benchmark level can be expected to experience certain adverse health effects, although susceptible subpopulations such as those with asthma are expected to be affected more by such exposures than healthy individuals. In taking these limitations into account, the Staff Paper reflected CASAC's advice 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 O_3-related health effects in drawing conclusions about an appropriate range of levels for consideration.

The Staff Paper focused on alternative standards with the same form as the current O_3 standard (*i.e.* the 0.074/4, 0.070/4 and 0.064/4 scenarios).[55] Having concluded in the 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 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

[53] Bell *et al.* (2006) referred to this level as being approximately equivalent to 120 $\mu g/m^3$, daily 8-hour maximum, the World Health Organization guideline and European Commission target value for O_3.

[54] As described in the Staff Paper (section 4.5.8) and discussed above, recent O_3 air quality distributions have been statistically adjusted to simulate just meeting the current 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.

[55] The abbreviated notation used to identify the current 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 current standard is identified as ''0.084/4.''

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 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. For reasons discussed above in section II.C.2, the Staff Paper focused on exposures of concern at the 0.070 and 0.060 ppm benchmark levels for the purpose of evaluating alternative standard levels. As shown in the Table 1 in this notice, the percent of population exposed at any given level is very similar for all and asthmatic school age children. Substantial year-to-year variability in exposure estimates 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 of these benchmark levels. The 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 discussed in the Staff Paper, a standard set at the upper part of the range recommended by EPA staff (*e.g.*, the 0.074/4 scenario) would result in an aggregate estimate of about 4 percent of all or asthmatic school age children likely to experience exposures of concern at the ≥0.070 ppm benchmark level based on the 2002 simulation, a year with relatively high O_3 levels, while the estimates range up to 12 percent of all or asthmatic school age children in the single city with the least degree of protection from this standard. Using the 2004 simulation, a year with relatively low O_3 levels, exposures of concern at this level are essentially eliminated. At the benchmark level of ≥0.060 ppm, in aggregate using the 2002 simulation about 22 percent of all or asthmatic school age children are estimated to experience exposures of concern; this estimate ranges up to about 46 percent of all or asthmatic school age children in the single city with the least degree of protection from this standard. Using the 2004 simulation, exposures of concern at this level are estimated to be substantially lower. A standard set at this level is estimated to reduce the number of all and asthmatic school age children estimated to experience one or more moderate lung function decrements by about 30 to 50 percent relative to the current standard, with city-to-city differences accounting for most of the variability in estimates. A standard set at this level is estimated to reduce non-accidental mortality by about 10 to 40 percent, with most of the variability occurring across the 12 city estimates.

Using the 2002 simulation, a standard set at this level (the 0.074/4 scenario) is estimated to reduce the incidence of symptom days in children with moderate to severe asthma in the Boston area by about 1,000 days, a 15 percent reduction relative to the current standard. With this reduction, it is estimated that about 1 respiratory symptom day in 8 during the O_3 season would be attributable to O_3 exposure. Estimated incidence of respiratory-related hospital admissions was reduced by 14 to 17 percent by a standard set at this level relative to the current standard, in the year with relatively high and relatively low O_3 air quality levels respectively.

The Staff Paper notes that a standard set at the middle part of the staff-recommended range, as indicated by the estimates for the 0.070/4 scenario, would reduce the exposures of concern at the 0.070 ppm level substantially over the current standard, resulting in an aggregate estimate of about 1.5 to nearly 2 percent of all or asthmatic school age children likely to experience exposures of concern even using the 2002 simulation, and leaving approximately 5 percent or less of children likely to experience exposures of concern in the city with the least degree of protection. Using the 2004 simulation, it essentially eliminates exposures of concern at this level. It reduces exposures of concern at the 0.060 ppm benchmark level less so, leaving larger percentages of all school age children unprotected using the 2002 simulation (about 15 percent in aggregate) or in the city with the least protection from this standard (about 33 percent). However, using the 2004 simulation, it is estimated to reduce exposures of concern at this benchmark level to approximately 5 percent or less of children even in the city with the least degree of protection. It provides considerable additional protection for members of at-risk groups, over the current O_3 standard, against respiratory morbidity effects such as lung function decrements, respiratory symptom days and hospital admissions, as well as non-accidental mortality.

A standard set at lower part of the staff-recommended range (*e.g.*, the 0.064/4 scenario), would result in an aggregate estimate of less than 0.5 percent of all and asthmatic school age children likely to experience exposures of concern at the 0.070 ppm benchmark level using the 2002 simulation and only about 1 percent of all and asthmatic school age children in the city with the least degree of protection from this standard. At the benchmark level of 0.060 ppm, in aggregate using the 2002 simulation about 5 percent of all and asthmatic school age children are estimated to experience exposures of concern; this number ranges up to 15 percent of all and asthmatic school age children in the city with the least degree of protection from this standard. A standard set at this level is estimated to reduce the number of all and asthmatic school age children estimated to experience one or more moderate lung function decrements by about 40 to 75 percent over the current standard, and non-accidental mortality by about 25 to 75 percent, with most of the variability occurring across the 12 city estimates.

A standard set at the 0.064/4 scenario 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 1,900 days, about a 25 to 30 percent reduction over the current standard. But even with this reduction, it is estimated that 1 respiratory symptom day in 10 during the O_3 season is attributable to O_3 exposure. Estimated incidence of respiratory-related hospital admissions would be reduced by 30 to 35 percent over the current standard, a reduction of 125 to 150 hospital admissions in the New York City area alone, using the 2002 and 2004 simulations, respectively.

b. CASAC Views

As 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 follows from its more general recommendation, discussed above, that the current standard of 0.084 ppm needs 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 to the Administrator, 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 Criteria Document and 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.).

c. Administrator's Proposed Conclusions on Level

For the reasons discussed below, and taking into account information and assessments presented in the Criteria Document and Staff Paper, the advice and recommendations of CASAC, and the public comments to date, the Administrator proposes to revise the existing 8-hour primary O_3 standard. Specifically, the Administrator proposes to revise (1) The level of the primary O_3 standard to within a range from 0.070 to 0.075 ppm and (2) the degree of precision to which the level of the standard is specified to the thousandth ppm.

However, in recognition of alternative views of the science, the exposure and risk assessments and the uncertainties inherent in these assessments, and the appropriate policy responses based on the currently available information, the Administrator also solicits comments on whether to proceed instead with: (1) Alternative levels of the 8-hour primary O_3 standard, within ranges of below 0.070 ppm down to 0.060 ppm and above 0.075 ppm up to and including retaining the current standard; (2) alternative forms of the standard, including the 3-year average of the annual third- and fifth-highest daily maximum 8-hour average O_3 concentrations; and (3) retaining the degree of precision of the current standard (to the nearest hundredth ppm). Based on the comments received and the accompanying rationales, the Administrator may adopt other standards within the range of the alternative levels and forms identified above in lieu of the standards he is proposing today.

The Administrator's consideration of alternative levels of the primary O_3 standard builds on his proposal, discussed above, that the overall body of evidence indicates that the current 8-hour O_3 standard is not requisite to protect public health with an adequate margin of safety because it does not provide sufficient protection and that revision would result in increased public health protection, especially for members of at-risk groups, notably including asthmatic children and other people with lung disease, as well as all children and older adults, especially those active outdoors, and outdoor workers, against an array of adverse health effects. These effects range from health outcomes that could be quantified in the risk assessment, including decreased lung function, respiratory symptoms, serious indicators of respiratory morbidity such as hospital admissions for respiratory causes, and nonaccidental mortality, to health outcomes that could not be directly estimated, including pulmonary inflammation, increased medication use, emergency department visits, and possibly cardiovascular-related morbidity effects. In reaching a proposed decision about the level of the O_3 primary standard, the Administrator has considered: the evidence-based considerations from the Criteria Document and the Staff Paper; the results of the exposure and risk assessments discussed above and in the Staff Paper, giving weight to the exposure and risk assessments as judged appropriate; CASAC advice and recommendations, as reflected in discussions of drafts of the Criteria Document and Staff Paper at public meetings, in separate written comments, and in 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 8-hour standard is requisite to protect public health with an adequate margin of safety, the Administrator is mindful that this choice requires judgment based on an interpretation of the evidence and other information that neither overstates nor understates the strength and limitations of the evidence and information nor the appropriate inferences to be drawn.

The Administrator notes that the most certain evidence of adverse health effects from exposure to O_3 comes from the clinical studies, and that the large bulk of this evidence derives from studies of exposures at levels of 0.080 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. Moreover there is no evidence that the 0.080 ppm level is a threshold for these effects. Although the Administrator takes note of the very limited new evidence of lung function decrements and respiratory symptoms in some healthy individuals at the 0.060 ppm exposure level, he judges this evidence too limited to support a primary focus at this level. The Administrator also notes that clinical studies, supported by epidemiological studies, provide important new evidence that people with asthma are likely to experience larger and more serious effects than healthy people from exposure to O_3. There are also epidemiological studies that provide evidence of statistically significant associations between short-term O_3 exposures and more serious health effects such as emergency department visits and hospital admissions, and premature mortality, in areas that likely would have met the current standard. There are also many epidemiological studies done in areas that likely would not have met the current standard but which nonetheless report statistically significant associations that generally extend down to ambient O_3 concentrations that are below the level of the current standard. Further, there are a few studies that have examined subsets of data that include only days with ambient O_3 concentrations below the level of the current standard, or below even much lower O_3 concentrations, and continue to report statistically significant associations with respiratory morbidity outcomes and mortality. In considering this evidence, the Administrator notes that the extent to which these studies provide evidence of causal relationships with exposures to O_3 alone down to the lowest levels observed remains uncertain. To further inform the interpretation of this evidence, EPA seeks comment on the degree to which associations observed in epidemiological studies reflect causal relationships between important health endpoints and exposure to O_3 alone at ambient O_3 levels below the current standard.

Therefore, the Administrator judges that revising the current standard to protect public health with an adequate margin of safety is warranted, and

would reduce risk to public health, based on: (1) The strong body of clinical evidence in healthy people at exposure levels of 0.080 and above of lung function decrements, respiratory symptoms, pulmonary inflammation, and other medically significant airway responses, as well as some indication of lung function decrements and respiratory symptoms at lower levels; (2) the substantial body of clinical and epidemiological evidence indicating that people with asthma are likely to experience larger and more serious effects than healthy people; and (3) the body of epidemiological evidence indicating associations are observed for a wide range of serious health effects, including respiratory emergency department visits and hospital admissions, and premature mortality, at and below 0.080 ppm. The Administrator also judges that the estimates of exposures of concern and risks remaining upon just meeting the current standard or a standard at the 0.080 ppm level provide additional support for this view. For the same reasons, and the reasons discussed above in section II.C on the adequacy of the current standard, the Administrator judges that the standard should be set below 0.080 ppm, a level at which the evidence provides a high degree of certainty about the adverse effects of O_3 exposure even in healthy people.

The Administrator next considered what standard level below 0.080 ppm would be requisite to protect public health with an adequate margin of safety, that is sufficient but not more than necessary to achieve that result, recognizing that such a standard would result in increased public health protection. The assessment of a standard level calls for consideration of both the degree of additional protection that alternative levels of the standard might be expected to provide as well as the certainty that any specific level will in fact provide such protection. In the circumstances present in this review, 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 holistically in making this public health policy judgment, and selecting a standard level from a range of reasonable values.

The Administrator notes that at exposure levels below 0.080 ppm there is only a very limited amount of evidence from clinical studies indicating effects in some healthy individuals at levels as low as 0.060 ppm. The great majority of the evidence concerning effects below 0.080 ppm is from epidemiological studies. The epidemiological studies do not identify any bright-line threshold level for effects. At the same time, the epidemiological studies are not themselves direct evidence of a causal link between exposure to O_3 and the occurrence of the effects. The Administrator considers these studies in the context of all the other available evidence in evaluating the degree of certainty that O_3-related adverse health effects would occur at various ambient levels below 0.080 ppm, including the strong human clinical studies and the toxicological studies that demonstrate the biological plausibility and mechanisms for the effects of O_3 on airway inflammation and increased airway responsiveness at exposure levels of 0.080 ppm and above.

Based on consideration of the entire body of evidence and information available at this time, as well as the recommendations of CASAC, the Administrator proposes that a standard within the range of 0.070 to 0.075 ppm would be requisite to protect public health with an adequate margin of safety. A standard level within this range would reduce the risk of a variety of health effects associated with exposure to O_3, including the respiratory symptoms and lung function effects demonstrated in the clinical studies, and the emergency department visits, hospital admissions and mortality effects indicated in the epidemiological studies. All of these effects are indicative of a much broader array of O_3-related health endpoints, such as school absences and increased medication use, that are plausibly linked to these observed effects.

The Administrator considered the degree of improvements in public health that potentially could be achieved by a standard of 0.070 to 0.075 ppm, giving weight to the exposure and risk assessments as he judged appropriate, as discussed below. In considering the results of the exposure assessment, as discussed above (section II.C.4), the Administrator has primarily focused on exposures at and above the 0.070 ppm benchmark level as an important surrogate measure for potentially more serious health effects for at-risk groups, including people with asthma. In so doing, the Administrator particularly notes that although the analysis of ''exposures of concern'' was conducted to estimate exposures at and above three discrete benchmark levels, the concept is appropriately viewed as a continuum. As discussed above, the Administrator strives to balance concern 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 O_3 exposure levels. In focusing on this benchmark, the Administrator notes that upon just meeting a standard within the range of 0.070 to 0.075 ppm based on the 2002 simulation, the number of school age children likely to experience exposures at and above this benchmark level in aggregate (for the 12 cities in the assessment), is estimated to be approximately 2 to 4 percent of all and asthmatic children, and generally less than 10 percent of children even in cities that receive the least degree of protection from such a standard in a recent year with relatively high O_3 levels. A standard within the 0.070 to 0.075 ppm range would thus substantially reduce exposures of concern by about 90 to 80 percent, respectively, from those estimated to occur upon just meeting the current standard. While placing less weight on the results of the risk assessment, in light of the important uncertainties inherent in the assessment, the Administrator notes that the results indicate that a standard set within this range would likely reduce risks to at-risk groups from the O_3-related health effects considered in the risk assessment, and by inference across the much broader array of O_3-related health effects that can only be considered qualitatively, relative to the level of protection afforded by the current standard. This lends support to the proposed range.

The Administrator judges that a standard set within the range of 0.070 to 0.075 ppm would provide a degree of reduction in risk that is important from a public health perspective, and that a standard within this range would be requisite to protect public health, including the health of at-risk groups, with an adequate margin of safety. EPA's evaluation of the body of scientific evidence and quantitative estimates of exposures and risks indicates that substantial reductions in public health risks would occur throughout this range. Because there is no bright line clearly directing the choice of level within this reasonable range, the choice of what is appropriate, considering the strengths and limitations of the evidence, and the appropriate inferences to be drawn from the evidence and the exposure and risk assessments, is a public health policy judgment. To further inform this judgment, EPA seeks comment on the extent to which the epidemiological and clinical evidence provides guidance as to the level of a standard that would be requisite to protect public health with

an adequate margin of safety, especially for at-risk groups.

In considering the available information, the Administrator also judges that a standard level below 0.070 ppm would not be appropriate. In reaching this judgment, the Administrator notes that there is only quite limited evidence from clinical studies at exposure levels below 0.080 ppm O_3. Moreover, the Administrator recognizes that in the body of epidemiological evidence, many studies report positive and statistically significant associations, while others report positive results that are not statistically significant, and a few do not report any positive O_3-related associations. In addition, the Administrator judges that evidence of a causal relationship between adverse health outcomes and O_3 exposures becomes increasingly uncertain at lower levels of exposure.

The Administrator also has considered the results of the exposure assessments in reaching his judgment that a standard level below 0.070 ppm would not be appropriate. The Administrator notes that in considering the results from the exposure assessment, a standard set at the 0.070 ppm level, with the same form as the current standard, is estimated to provide substantial reductions in exposures of concern (*i.e.*, approximately 90 to 92 percent reductions in the numbers of school age children and 94 percent reduction in the total number of occurrences) for both all and asthmatic school age children relative to just meeting the current standard based on a simulation of a recent year with relatively high O_3 levels (2002). Thus, a 0.070 ppm standard would be expected to provide protection from the exposures of concern that the Administrator has primarily focused on for over 98 percent of all and asthmatic school age children even in a year with relatively high O_3 levels, increasing to over 99.9 percent of children in a year with relatively low O_3 levels (2004).

In considering the results of the health risk assessment, as discussed in section II.B above, the Administrator notes that there are important uncertainties and assumptions inherent in the risk assessment and that this assessment is most appropriately used to simulate trends and patterns that can be expected as well as providing informed but still imprecise estimates of the potential magnitude of risks. The Administrator particularly notes that as lower standard levels are modeled, including a standard set at a level below 0.070 ppm, the risk assessment continues to assume a causal link between O_3 exposures and the occurrence of the health effects examined, such that the assessment continues to indicate reductions in O_3-related risks upon meeting a lower standard level. As discussed above, however, the Administrator recognizes that evidence of a causal relationship between adverse health effects and O_3 exposures becomes increasingly uncertain at lower levels of exposure. Given all of the information available to him at this time, the Administrator judges that the increasing uncertainty of the existence and magnitude of additional public health protection that standards below 0.070 ppm might provide suggests that such lower standard levels would likely be below what is necessary to protect public health with an adequate margin of safety.

In addition, the Administrator judges that a standard level higher than 0.075 ppm would also not be appropriate. This judgment takes into consideration the information discussed above in section II.B, and is based on the strong body of clinical evidence in healthy people at exposure levels of 0.080 ppm and above, the substantial body of clinical and epidemiological evidence indicating that people with asthma are likely to experience larger and more serious effects than healthy people, the body of epidemiological evidence indicating that associations are observed for a wide range of more serious health effects at levels below 0.080 ppm, and the estimates of exposure and risk remaining upon just meeting a standard set at 0.080 ppm. The much greater certainty of the existence and magnitude of additional public health protection that such levels would forego provides the basis for judging that levels above 0.075 ppm would be higher than what is requisite to protect public health, including the health of at-risk groups, with an adequate margin of safety.

For the reasons discussed above, the Administrator proposes to revise the level of the primary O_3 standard to within the range of 0.070 to 0.075 ppm.

Having reached this decision based on the approach to interpreting the evidence described above, the Administrator recognizes that other approaches to selecting a standard level have been presented to the Agency. As described above, the CASAC has stated in two letters to the Administrator (Henderson, 2006c; Henderson, 2007) its unanimous recommendation that the current primary O_3 NAAQS be revised to within the range from 0.060 to 0.070 ppm. The CASAC Panel noted that while data exist that adverse health effects may occur at levels lower than 0.060 ppm, these data are less certain and that achievable gains in protecting human health can be accomplished through lowering the O_3 NAAQS to a level between 0.060 and 0.070 ppm. In addition to the views of CASAC described above, the Agency received the public comments described below.

One group of commenters submitted comments that supported revising the level of the primary O_3 standard from 0.070 ppm down to or even below 0.060 ppm, consistent with or below the range recommended by CASAC. In considering the available evidence as a basis for their views, these commenters generally noted that the controlled human exposure studies, showing statistically significant declines in lung function, and increases in respiratory symptoms, airway inflammation and airway responsiveness at a 0.080 ppm exposure level, were conducted with healthy adults, not members of at-risk groups including people with asthma and active children generally. Further, recognizing the substantial variability in response between subjects, some of these commenters felt that the number of subjects included in these studies was too small to ascertain the full range of responses, especially for at-risk groups. Such considerations in part were the basis for these commenters' view that an O_3 standard set at 0.080 ppm is not protective of public health and has no margin of safety for at-risk groups. In addition, some of these commenters also noted that the World Health Organization's guidelines for O_3 air quality are in the range of 0.061 to 0.051 ppm.

In considering the results of the human exposure and health risk assessment, this group of commenters generally expressed the view that these assessments substantially underestimate the public health impacts of exposure to O_3. For example, several commenters noted that the assessments are done for a limited number of cities, they do not address risks to important at-risk subpopulations (*e.g.*, outdoor workers, active people who spend their summers outdoors, children up to 5 years of age), and they do not include many health effects that are important from a public health perspective (*e.g.*, school absences, restricted activity days). Further, some of these commenters expressed the view that the primary O_3 standard should be set to protect the most exposed and most vulnerable groups, and the fact that some children are frequently indoors, and thus at lower risk, should not weigh against setting a standard to protect those children who are active outdoors. To the extent the exposure and risk estimates

are considered, some of these commenters felt that primary consideration should be given to the estimates based on 2002 air quality, for which most areas had relatively higher O_3 levels than in 2004, so as to ensure public health protection even in years with relatively worse O_3 air quality levels. Some commenters also felt that the exposure analysis should focus on a benchmark concentration for exposures of concern at the 0.060 ppm level, the lower end of the range of alternative standards advocated by the CASAC Panel.

In sharp contrast to the views discussed above, other public commenters supported retaining the current standard. In considering the available evidence as a basis for their views, these commenters challenged a number of aspects of the interpretation of the evidence presented in the Criteria Document. For example, some of these commenters asserted that EPA generally overestimates the magnitude and consistency of the results of short-term exposure epidemiological studies (*e.g.*, for respiratory symptoms, school absences, hospital admissions, mortality), mistakenly links statistical significance and consistency with strength of associations, and underestimates the uncertainties in interpreting the results of such studies. Further, these commenters generally express the view that there is significant uncertainty related to the reliability of estimates from time-series studies, in that ambient monitors do not provide reliable estimates of personal exposures, such that the small reported morbidity and mortality risks are unlikely to be attributable to people's exposures to O_3. Rather, these commenters variously attribute the reported risks to the inability of time series studies to account for key model specification factors such as smoothing for time-varying parameters, meteorological factors, and removal of O_3 by building ventilation systems, and confounding by co-pollutants. In particular, these commenters generally asserted that reported associations between short-term O_3 exposure and mortality are not causal, in that the reported relative risks are too small to provide a basis for inferring causality and the associations are most likely due to confounding, inappropriately specified statistical models, or publication bias.

In considering the results of the human exposure and health risk assessment, this group of commenters generally expressed the view that these assessments are based on a number of studies that should not be used in quantitative risk assessment. For example, some commenters asserted that the results of time-series studies should not be used at all in quantitative risk assessments, that risk estimates from single city time-series studies should not be used since they are highly heterogeneous and influenced by publication bias, and that risk estimates from multi-city studies should not be used in estimating risk for individual cities. This group of commenters also generally expressed the view that the assessments generally overestimate the public health impacts of exposure to O_3. Noting that the risk assessment used a nonlinear exposure-response function to estimate decreased lung function risks, some commenters expressed the view that a nonlinear approach should also be used to assess other acute morbidity effects and mortality. This view was in part based on judgments that it is not possible to determine if thresholds exist using time-series analyses and that the lack of association of O_3 to mortality in the winter season is highly supportive of the likelihood of the existence of an effect threshold. With regard to the risk assessment based on controlled human exposure studies of lung function decrements, some commenters expressed the view that the assessment should not rely on what they characterized as ''outlier'' information to define exposure-response relationships, with reference to the data in the Adams (2006) study at the 0.060 and 0.040 ppm exposure levels, but rather should focus on group central tendency response levels. Further, some commenters expressed the view that the air quality rollback algorithm used introduces significant uncertainty, especially when applied to areas requiring very large reductions in air quality to meet the alternative standards examined, and may result in overestimates in benefits from emission reductions. Some commenters noted that potential beneficial effects of O_3 in shielding from UV–B radiation are not quantified in the assessment, and that the assessment should discuss the evidence for both adverse and beneficial effects with the same objectivity. Finally, some of these commenters asserted that since estimates of exposures of concern (which they defined as the benchmark concentration of 0.080 ppm) and lung function decrements are substantially below the estimates available when the current O_3 standard was set in 1997, retaining the current standard is the most appropriate policy alternative.

Some commenters also have raised concerns about potential uncertainties with regard to estimating policy-relevant background O_3 levels including: (1) Stratospheric O_3 contributions to the mid- and upper troposphere, which are relatively long-lived (1 to 2 months), and are transported downward to the surface over time; (2) potential trends in stratospheric O_3 levels due to changes in stratospheric circulation or to reduction of O_3 depleting chemicals; (3) O_3 levels due to lightning strikes in estimating policy-relevant background concentrations; and (4) potential uncertainty with regard to policy-relevant background O_3 levels having to do with increases in O_3 precursors elsewhere in the world. EPA asks for comments on these issues and on how they may relate to the estimation and consideration of policy-relevant background levels in setting the O_3 standards.

Several Governors, State Legislators, and other local officials have expressed concerns related to a more stringent standard. These officials recognize that State and local governments have important roles in developing and implementing policy that improve air quality while at the same time achieving economic and quality of life objectives. In addition, these officials note that States are just beginning to implement current air quality standards and raise concern with moving forward on revised standards without first realizing the results from the last revision.

As a related concern, a number of areas—including some of the cities involved in the risk assessment—will have difficulty in complying with the current 8-hour standard within the next decade. As a result, the full public health gains in these areas from a more stringent 8-hour standard are unlikely to be realized for a number of years. In light of the fact that these public health gains may not fully materialize within the attainment date structure set forth in the Clean Air Act, some commenters question whether the Agency can or should consider these projected gains as a health based criterion for its decisionmaking. EPA requests comment on this view.

The Administrator is mindful that the country has important goals related to the increased production and use of renewable energy, and that these new energy sources can have important public health, environmental and other benefits, such as national security benefits. In some contexts and situations, however, the use of renewable fuels may impact compliance with a lowered ozone NAAQS standard. For example, the Agency recently promulgated final regulations pursuant to section 211(o) of the Clean Air Act,

which was enacted as part of the Energy Policy Act of 2005. This provision requires the use of 7.5 billion gallons of renewable fuel by 2012, a level which will be greatly exceeded in practice. In the Regulatory Impact Analysis which accompanied the renewable fuel regulations, the Agency recognized the impact of this program on emissions related to ozone, toxics and greenhouse gases and otherwise reviewed the impacts on energy security. The Administrator requests comment on such factors and any relationship to this rulemaking, including the extent of EPA's discretion under the Clean Air Act to take such factors into account (see section I.A).

In general, these commenters' concerns are consistent with the view that adopting a more stringent 8-hour standard now, without a better understanding of the health effects associated with O_3 exposure at lower levels, would have an uncertain public health benefit. The Administrator recognizes that commenters have raised numerous concerns regarding various types of uncertainties in the available information, including for example uncertainties in (1) The assessment of exposures, (2) the estimation of concentration-response associations in the epidemiological studies, (3) the potential role of co-pollutants in interpreting the reported associations in epidemiological studies, and (4) the estimation of background concentrations. The Administrator has heard these concerns from Governors and other commenters and invites comment on whether it would be appropriate to retain the existing standard and delay considering modification of the 8-hour standard until the next NAAQS review, when a more complete body of information is expected to be available.

Consistent with the goal of soliciting comment on a wide array of views, the Administrator also solicits comments on these alternative approaches and views, and on related standard levels, including levels down to 0.060 ppm and up to retaining the level of the current 8-hour standard (*i.e.*, effectively 0.084 ppm with the current rounding convention). The Administrator recognizes that these sharply divergent views on the appropriate level of the standard are based on very different interpretations of the science itself, including its relative strengths and limitations, very different judgments as to how such scientific evidence should be used in making policy decisions on proposed standards, and very different public health policy judgments.

E. Proposed Decision on the Primary Standard

For the reasons discussed above, and taking into account information and assessments presented in the Criteria Document and Staff Paper, the advice and recommendations of CASAC, and the public comments to date, the Administrator proposes to revise the existing 8-hour primary O_3 standard. Specifically, the Administrator proposes to revise: (1) The level of the primary O_3 standard to within a range from 0.070 to 0.075 ppm and (2) the degree of precision to which the level of the standard is specified to the thousandth ppm. The proposed 8-hour primary standard, with a level in the range of 0.070 to 0.075 ppm, would be met at an ambient air monitoring site when the 3-year average of the annual forth-highest daily maximum 8-hour average O_3 concentration is less than or equal to the level of the standard that is promulgated. Data handling conventions are specified in the proposed creation of Appendix P, as discussed in section V below.

However, in recognition of alternative views of the science, the exposure and risk assessments and the uncertainties inherent in these assessments, and the appropriate policy responses based on the currently available information, the Administrator also solicits comments on whether to proceed instead with: (1) Alternative levels of the 8-hour primary O_3 standard, within ranges of below 0.070 ppm down to 0.060 ppm and above 0.075 ppm up to and including retaining the current standard; (2) alternative forms of the standard, including the 3-year average of the annual third- and fifth-highest daily maximum 8-hour average O_3 concentrations; and (3) retaining the degree of precision of the current standard (to the nearest hundredth ppm). Based on the comments received and the accompanying rationales, the Administrator may adopt other standards within the range of the alternative levels and forms identified above in lieu of the standards he is proposing today.

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 O_3, 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. For the current O_3 NAAQS, an 8-hour average concentration of 0.084 ppm corresponds to an AQI value of 100. 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; 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.

The Agency recognizes the importance of revising the AQI in a timely manner to be consistent with any revisions to the NAAQS. Therefore EPA proposes to finalize conforming changes to the AQI, in connection with the Agency's final decision on the O_3 NAAQS if revisions to the primary standard are promulgated. These conforming changes would include setting the 100 level of the AQI at the same level as the revised primary O_3 NAAQS, 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 review does not inform decisions about breakpoints at those higher levels.

IV. Rationale for Proposed Decision on the Secondary Standard

This section presents the rationale for the Administrator's proposed decision to revise the existing 0.08 ppm, 8-hour O_3 secondary NAAQS. The Administrator proposes to revise the current secondary standard by replacing it with one of two standard options. One

option is to adopt a new cumulative, seasonal concentration-weighted form, set at an annual level in the range of 7 to 21 ppm-hours. This standard would be expressed as a sum of weighted hourly concentrations, cumulated over the 12-hour daylight period (8 a.m. to 8 p.m.) during the consecutive 3-month period within the O_3 monitoring season with the maximum index value. This concentration-weighted form is commonly called W126, and is defined as the sum of sigmoidally weighted hourly O_3 concentrations over a specified period, where the daily sigmoidal weighting function is defined as:

$$W126 = \sum_{i=8AM}^{i<7PM} w_{C_i} C_i, \text{ where } C_i = \text{hourly } O_3 \text{ at hour i, and } w_{C_i} = \frac{1}{1+4403e^{-126C_i}}.$$

The other option is to revise the current secondary standard by making it identical to the proposed 8-hour primary standard, within the proposed range of 0.070 to 0.075 ppm. For this option, EPA also solicits comment on a wider range of 8-hour secondary standard levels, including down to 0.060 ppm and up to and including retaining the current 8-hour secondary standard of 0.08 ppm. The Administrator has also considered and solicits comment on an alternative approach to setting a cumulative, seasonal standard(s).

As discussed more fully below, the rationale for these proposed options is based on a thorough review of the latest scientific information on vegetation effects associated with exposure to ambient levels of O_3, as assessed in the Criteria Document. This rationale also takes into account: (1) Staff assessments of the most policy-relevant information in the Criteria Document regarding the evidence of adverse effects of O_3 to vegetation and ecosystems, information on biologically-relevant exposure metrics, and staff analyses of air quality, vegetation exposure and risks, presented in the Staff Paper and described in greater detail in the associated Technical Report on Ozone Exposure, Risk, and Impact Assessments for Vegetation (Abt, 2007), upon which staff recommendations for revisions to the secondary O_3 standard are based; (2) CASAC advice and recommendations as reflected in discussion of drafts of the Criteria Document and Staff Paper at public meetings, in separate written comments, and in CASAC's letters to the Administrator (Henderson, 2006a, b, c; 2007); (3) public comments received during development of these documents either in conjunction with CASAC meetings or separately; and (4) consideration of the degree of protection to vegetation potentially afforded by the proposed 8-hour primary standard.

In developing this rationale, EPA has again focused on direct O_3 effects on vegetation, specifically drawing upon an integrative synthesis of the entire body of evidence, published through early 2006, on the broad array of vegetation effects associated with exposure to ambient levels of O_3 (EPA, 2006a, chapter 9). In addition, because O_3 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 are not quantifiable at this time. As was concluded in the 1997 review, and based on the body of scientific literature assessed in the current Criteria Document, the Administrator believes that it is reasonable to conclude that a secondary standard protecting the public welfare from known or anticipated adverse effects to trees, native vegetation and crops would also afford increased protection from adverse effects to other environmental components relevant to the public welfare, including ecosystem services and function. The peer-reviewed literature includes studies conducted in the U.S., Canada, Europe, and many other countries around the world. In its assessment of the evidence judged to be most relevant to making decisions on the level of the O_3 secondary standard, however, EPA has placed greater weight on U.S. studies, due to the often species-, site- and climate-specific nature of O_3-related vegetation response.

As with virtually any policy-relevant vegetation effects research, there is uncertainty in the characterization of vegetation effects attributable to exposure to ambient O_3. As discussed below, however, research conducted since the last review provides important information coming from field-based exposure studies, including free air, gradient and biomonitoring surveys, in addition to the more traditional controlled open top chamber (OTC) studies. Moreover, the newly available studies evaluated in the 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 in this review has provided an adequate basis for regulatory decision-making at this time. This review also provides important input to EPA's research plan for improving our future understanding of the effects of ambient O_3 at lower levels.

A. Vegetation Effects Information

This section outlines key information contained in the Criteria Document (chapter 9) and in the Staff Paper (chapter 7) on known or potential effects on public welfare which may be expected from the presence of O_3 in ambient air. The information highlighted here summarizes: (1) New information available on potential mechanisms for vegetation effects associated with exposure to O_3; (2) the nature of effects on vegetation that have been associated with exposure to O_3 and consequent potential impacts on ecosystems; and (3) considerations in characterizing what constitutes an adverse welfare impact of O_3.

Exposures to O_3 have been associated quantitatively and qualitatively with a wide range of vegetation effects. The decision in the last 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 OTC exposure method, found cumulative, seasonal O_3 exposures were most strongly associated with observed vegetation response. The Criteria Document prepared for this review discusses a number of additional studies that support and strengthen key conclusions regarding O_3 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 subcellular, 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. These non-chambered, field-based study results begin to address one of the key data gaps cited by the Administrator in the last review.

The following discussion of the policy-relevant science regarding vegetation effects associated with cumulative, seasonal exposures to ambient levels of O_3 integrates information from the Criteria Document (chapter 9) and the Staff Paper (chapter 7).

1. Mechanisms Governing Plant Response to Ozone

The interpretation of predictions of risk associated with vegetation response at ambient O_3 exposure levels can be informed by scientific understanding regarding O_3 impacts at the genetic, physiological, and mechanistic 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 O_3-induced changes in physiology continues to support the information discussed in the last review (EPA, 1996a, 2006a). In addition, during the last decade understanding of the cellular processes within plants has been further clarified and enhanced. Therefore, this section reviews the key scientific conclusions identified in 1996 Criteria Document (EPA, 1996a), and incorporates new information from the current Criteria Document (EPA, 2006a). This section describes: (1) Plant uptake of O_3, (2) O_3-induced cellular to systemic response, (3) plant compensation and detoxification mechanisms, (4) O_3-induced changes to plant metabolism, and (5) plant response to chronic O_3 exposures.

a. Plant Uptake of Ozone

To cause injury, O_3 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 O_3 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 O_3 does not penetrate the leaf's cuticle, it must reach the stomatal openings in the leaf for absorption to occur. The movement of O_3 and other gases such as CO_2 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, CO_2 concentration, soil fertility and water status, and in some cases the presence of air pollutants, including O_3. These modifying factors produce stomatal conductance that vary between leaves of the same plant, individuals and genotypes within a species and diurnally and seasonally.

b. Cellular to Systemic Response

Once inside the leaf, O_3 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. Having entered the aqueous phase, O_3 can be rapidly altered to form oxidative products that can diffuse more readily into and through the cell and react with many biochemical compounds. An early step in a series of O_3-induced events that leads to leaf injury seems to involve alteration in cell membrane function, including membrane transport properties (EPA, 2006a). One such signaling molecule is hydrogen peroxide (H_2O_2). The presence of higher-than-normal levels of H_2O_2 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 O_3 stress was identified as a consistent marker for O_3 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 O_3 uptake is small enough for the plant to detoxify or metabolize O_3 or its metabolites or (2) the plant is able to repair or compensate for the O_3 impacts (Tingey and Taylor, 1982; U.S. EPA, 1996a). A few studies have documented direct stomatal closure or restriction in the presence of O_3 in some species. 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 O_3 simultaneously restricts the uptake of CO_2, 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 O_3 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 O_3 injury. Specifically, data are needed on potential rates of antioxidant production, subcellular location(s) of antioxidants, and whether generation of these antioxidants in response to O_3-induced stress potentially diverts resources and energy away from other vital uses. Thus, the Criteria Document concludes that scientific understanding of the detoxification mechanisms is not yet complete and requires further investigation (EPA, 2006a).

Once O_3 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 or degree of tolerance, nor are all stages of a plant's development equally sensitive to O_3. At higher levels or over longer periods of O_3 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 O_3 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 O_3-induced stomatal closure resulting in reduced uptake of CO_2. A large body of literature published since 1996 has further elucidated the mechanism of the effect of O_3 within the chloroplast. Pell *et al.* (1997) showed that O_3 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 O_3 has a greater effect as leaves age, with the greatest impact of O_3 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 O_3 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.

e. Plant Response to Chronic Ozone Exposures

Though many changes that occur with O_3 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 exposures to low O_3 concentrations. These 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 O_3 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, when it has accumulated sufficiently, will be propagated to the level of the 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 O_3-related effects, as summarized below, is based on research that was available in the last review. Recent studies 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 O_3 exposure than stem or leaf growth (EPA, 2006a). When fewer carbohydrates are present in the roots, less energy will be 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 O_3 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 (Grulke *et al.*, 1998; Grulke and Balduman, 1999). Data from a long-studied pollution gradient in the San Bernardino Mountains of southern California suggest that O_3 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 (Grulke *et al.*, 1998), and a similar pattern was found in a separate study with whole-tree harvest along this gradient (Grulke 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 O_3 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 O_3-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 O_3-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 O_3 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 O_3 than sensitive annual plants. In addition, studies have shown that the relationship between O_3 sensitivity in seedling and mature growth stages of trees can vary widely, with seedling growth being more sensitive to O_3 exposures in some species, while in others, the mature growth stage is the more O_3 sensitive. In general, mature deciduous trees are likely to be more sensitive to O_3 than deciduous seedlings, and mature evergreen trees are likely to be less sensitive to O_3 than their seedling counterparts. Based on these results, stomatal conductance, O_3 uptake, and O_3 effects cannot be assumed to be equivalent in seedlings and mature trees.

In the last review (EPA, 1996b), analyses of the effects of O_3 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 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 yield 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-hrs, 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-hrs.

Since the 1996 review, only a few studies have developed C–R functions for additional tree seedling species (EPA, 2006a). One such study is of particular importance in that it documented growth effects from O_3 exposure in the field without the use of chambers or other fumigation methods that were as great as those seen in OTC studies (Gregg *et al.*, 2003). This study placed Eastern cottonwood (*Populus deltoides*) saplings at sites along a continuum of ambient O_3 exposures that gradually increased from urban to rural areas in the New York City area (Gregg *et al.*, 2003). Eastern cottonwood was selected because it is fast growing, O_3 sensitive and important ecologically, along stream banks, 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 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 O_3 exposures that increased as one moved downwind from urban to less urban and more rural sites. It was determined that the lower O_3 exposure within the city center was due to NO_X titration reactions which removed O_3 from the ambient air. The authors were able to reproduce the growth responses observed in the field in a companion OTC experiment, confirming O_3 as the stressor inducing the growth loss response (Gregg *et al.*, 2003).

Another recent set of studies employed a modified Free Air CO_2 Enrichment (FACE) methodology to expose vegetation to elevated O_3 without the use of chambers. This exposure method was originally developed to expose vegetation to elevated levels of CO_2, but has been modified to include O_3 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.*, CO_2, O_3) 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 did OTCs. This is because, except for O_3 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 O_3), and by exposing the other rings to differing levels of elevated O_3, the growth response signal that is due solely to the change in O_3 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 O_3 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 O_3) (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 O_3 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 Staff Paper thus concluded that the combined evidence from the AspenFACE[56] 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 in the weight of evidence available in this review and provide additional evidence that O_3-induced effects observed in chambers also occur in the field.

Trees and other perennials, in addition to cumulating the effects of O_3 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 O_3 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 can and often does become visible. Acute injury usually appears within 24 hours after exposure to O_3 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 O_3 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

[56] Only a few northern forest types in the U.S. have been well studied with respect to O_3 exposures using the FACE method, though these systems are being used to expose numerous other ecosystem types to elevated levels of CO_2. Additional FACE studies with O_3 on other U.S. forest types would provide a better understanding of whether these results can be extrapolated to other forest types and mature forest stands.

the leaf level has significance to the vigor of the whole plant.

The presence of visible symptoms due to O_3 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 O_3 (Abt, 1993). Though not quantified, there is likely some level of economic impact to businesses and homeowners from O_3-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 O_3 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 O_3 is a longstanding and effective methodology (Chappelka and Samuelson, 1998; Manning and Krupa, 1992). Each bioindicator exhibits typical O_3 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 O_3 sensitive plant species throughout the U.S. (Coulston *et al.* 2003, 2004; Smith *et al.* 2003).

Since the conclusion of the 1996 NAAQS 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 O_3-induced foliar injury incidence is widespread across the country. The visible foliar injury indicator has been identified as a means to track O_3 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 O_3 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 O_3-related vegetation effects because other effects (*e.g.*, biomass loss) have been reported with and without visible injury. In a few 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 O_3 nor absence of other non-visible O_3 effects.

d. Reduced Plant Vigor

Though O_3 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 may include increased susceptibility to freezing temperatures, pest infestations and/or root disease, and compromised ability to compete for available resources. For example, when species with differing O_3-sensitivities occur together, the resulting decrease in growth in O_3-sensitive species may lead to an increase in growth of more O_3-tolerant species, which are now able to better compete for available resources. The result of such above effects can produce a loss in plant vigor in O_3-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 O_3 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) include 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 O_3 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 O_3 could either directly or indirectly impact all of the EEAs associated with an ecosystem that is being stressed by O_3.

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, O_3-related effects can be propagated up to ecosystem-level effects. Thus, through its impact on vegetation, O_3 can be an important ecosystem stressor.

i. Potential Ozone Alteration of Ecosystem Structure and Function

The Criteria Document (EPA, 2006a) outlines seven case studies where O_3 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 O_3 exposures over a period of 50 or more years. The

O_3-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 O_3. 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. The composition of lichens was significantly reduced. In this example, O_3 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, O_3 potentially impacted the ecosystem-related EEA of natural disturbance regime (*e.g.*, fire, erosion). Although the role of O_3 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 O_3 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 O_3 levels are less severe. Therefore, lower level chronic O_3 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.

One of the best-documented studies of population and community response to O_3 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 O_3 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 O_3 are complicated by other environmental factors, rapid changes in O_3 resistance were imposed by ambient levels and variations in O_3 exposure (Davison and Reiling, 1995). Specifically, in this case study, it appeared that O_3-sensitive individuals are being removed by O_3 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 O_3 may be altering ecosystem structure and function (Karnosky *et al.*, 2005). For example, a field O_3 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 CO_2 and O_3 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 O_3-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 O_3 is an important stressor in natural ecosystems, but it is difficult to quantify the contribution of O_3 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 O_3 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 O_3 on ecosystem structure and function in order to fully determine the extent to which O_3 is altering ecosystem services. Continued research, employing new approaches, will be necessary to fully understand the extent to which O_3 is affecting ecosystem services.

ii. Effects on Ecosystem Services and Carbon Sequestration

Since it has been established that O_3 affects photosynthesis and growth of plants, O_3 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 O_3.

Terrestrial ecosystems are important in the Earth's carbon (C) balance and could help offset emissions of CO_2 by humans if anthropogenic C is sequestered in vegetation and soils. The annual increase in atmospheric CO_2 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 CO_2 uptake by plant photosynthesis (Tans & White, 1998). Temperate forests of the northern hemisphere have been estimated to be a net sink of 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, O_3 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 O_3 pollution in the late 1980's to early 1990's. 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 O_3 pollution and rising CO_2 concentrations in the coming decades complicates predictions of future sequestration potential. Models generally predict that, in the future, C sequestration will increase with increasing CO_2, but often do not account for the decrease in productivity due to the local effects of tropospheric O_3. In the presence of high O_3 levels, the stimulatory effect of rising CO_2 concentrations on forest productivity has been estimated to be reduced by more that 20 percent (Tingey *et al.*, 2001; Ollinger *et al.* 2002; Karnosky *et al.*, 2003).

In summary, it would be anticipated that meeting lower O_3 standards would increase the amount of CO_2 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 O_3 sensitive plants. For, example forest ecosystems with dominant species such as aspen or ponderosa pine would be expected to increase CO_2 uptake more with lower O_3 than forests with more O_3 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 O_3-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 O_3-related effects on ecosystems must be inferred from observed or predicted O_3-related effects on individual plants, additional research at the ecosystem level could identify new indicators and/or establish stronger causal linkages between O_3-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 O_3 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 last review and again in the current Criteria Document and Staff Paper, the National Crop Loss Assessment Network (NCLAN) studies undertaken in the early to mid-1980's provide the largest, most uniform database on the effects of O_3 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 O_3 and increased O_3 above ambient (*i.e.*, modified ambient). 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[57]. In other words, there has been no evidence that crops are becoming more tolerant of O_3 (EPA, 2006a). For cotton, some newer varieties have been found to have higher yield loss due to O_3 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 O_3 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 O_3 tolerance of old and recent cultivars of soybean in a study of 22 soybean varieties (Long *et al.*, 2002). Thus, the Staff Paper concluded that the recent scientific literature continues to support the conclusions of the 1996 Criteria Document that ambient O_3 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 last review have focused on perennial forage crops (EPA, 2006a). These recent results confirm that O_3 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 O_3 exposures that occur in some years over large areas of the U.S.

3. Adversity of Effects

The Staff Paper recognized that the statute requires that a secondary standard be protective against ''adverse'' O_3 effects, not all identifiable effects. In considering what constitutes a vegetation effect that is adverse to the public welfare, the Staff Paper recognizes that O_3 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 O_3 vegetation effects as either ''injury'' or ''damage'' to help in determining adversity. Specifically, ''injury'' is

[57] Given the usefulness of generating robust C–R functions such as have been developed under NCLAN, it would be beneficial to employ a similar protocol to update and expand this research to include more recent and additional crop species and varieties, such as fruit and vegetable species, as well as recent O_3 air quality.

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 O_3-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 O_3-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.*, O_3 exposure) at the species level and at higher levels within an ecosystem hierarchy. Taking this recent literature into account, the 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 Criteria Document concluded that O_3 exposure indices that cumulate differentially weighted hourly concentrations are the best candidates for relating exposure to plant growth responses (EPA, 2006a). 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 (EPA, 2006a). The following selections, taken from section 5.5 the 1996 Criteria Document (EPA, 1996a), 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 O_3 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 O_3 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 O_3 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 last review, the biological basis for a cumulative, seasonal form was not in dispute. There was general agreement between the EPA staff, CASAC, and the Administrator, 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 seasonal cumulative 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 SUM06 [58] and W126, in the absence of biological evidence to distinguish between them, the Administrator based her 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 O_3 concentrations (defined at the time as 0.03 to 0.05 ppm) under many typical air quality distributions. On the basis of these considerations, the Administrator 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 last 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 notice of proposed rulemaking, the Administrator 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, the Administrator decided to make the secondary standard identical to the primary standard. She acknowledged,

[58] SUM06: Sum of all hourly O_3 concentrations greater or equal to 0.06 ppm over a specified time.

however, that ''it remained uncertain as to the extent to which air quality improvements designed to reduce 8-hr average O_3 concentrations averaged over a 3-year period would reduce O_3 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, chapter 7 of the current Staff Paper 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 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 are lower than in the last review. The W126 form, also evaluated in the last review, was again selected for comparison with the SUM06 form. Regarding the first consideration, the Staff Paper noted that 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 O_3 concentrations within the range of estimated PRB, as the weights assigned to concentrations in this range are very small. Thus, it would also provide a more appropriate target for air quality management programs designed to reduce emissions from anthropogenic sources contributing to O_3 formation. On the basis of these considerations, the Staff Paper concludes that the W126 form is the most biologically-relevant cumulative, seasonal form appropriate to consider in the context of the secondary standard review.

C. Vegetation Exposure and Impact Assessment

The vegetation exposure and impact assessment conducted for the current review and described in the Staff paper, consisted of exposure, risk and benefits analyses and improves and builds upon similar analyses performed in the last 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 recent monitored O_3 air quality for the years 2001–2004; (2) estimates of seedling growth loss under current and alternative O_3 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 current and alternative O_3 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 O_3 on vegetation have been documented as discussed above, it is important in considering risk to examine O_3 air quality patterns in the U.S. relative to the location of O_3 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 O_3 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 O_3 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 O_3. 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 final O_3 rule, it was acknowledged that because the national air quality surveillance network for O_3 was designed principally to monitor O_3 exposure in populated areas, there was limited measured data available to characterize O_3 air quality in rural and remote sites. Since the last 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 are afforded a higher level of protection. Two recent reports presented some discussion of O_3 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 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 O_3 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 O_3-sensitive tree species in the eastern U.S., occurs in the Great Smoky Mountain National Park and is estimated to have O_3-related seedling biomass loss of approximately 40 percent when exposed to a 3-month, 12-hour W126 O_3 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 O_3 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 O_3-sensitive tree species and genotypes within forest stands.

In order to characterize exposures to vegetation at the national scale,

however, the 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 O_3 air quality over broad geographic areas. The Staff Paper therefore investigated the appropriateness of using the O_3 outputs from the EPA/NOAA Community Multi-scale Air Quality (CMAQ)[59] model 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 this review, 2001 outputs from CMAQ version 4.5 were the most recent available.

Based on the significant difference in monitor network density between the eastern and western U.S., the Staff Paper concluded that it was appropriate to use separate interpolation techniques in these two regions. AQS and CASTNET monitoring data were solely 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., where rural monitoring is more sparse, O_3 values generated by the CMAQ model were used to develop scaling factors to augment the interpolation.

In order to characterize uncertainty in the interpolation method, monitored O_3 concentrations were systematically compared to interpolated O_3 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 Staff Paper utilized adjusted 2001 base year O_3 air quality distributions with a rollback method (Horst and Duff, 1995; Rizzo, 2005 & 2006) to reflect meeting the current and alternative secondary standard options. This technique combines both linear and quadratic elements to reduce higher O_3 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 O_3 air quality scenarios were analyzed: (1) 4th-highest daily maximum 8-hour average: 0.084 ppm (the effective level of the current standard) and 0.070 ppm levels; (2) 3-month, 12-hour. SUM06: 25 ppm-hour (proposed in the 1996 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 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 O_3 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 O_3 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 is rolled back to meet the current 8-hour secondary standard, the overall 3-month 12-hour W126 O_3 levels were somewhat improved, but not substantially. Under this scenario, there were still many areas in California with 12-hour W126 O_3 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 O_3 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 O_3 air quality when compared to just meeting the current 0.08 ppm, 8-hour standard. Most areas were predicted to have O_3 levels below the W126 level of 21 ppm-hr, 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 O_3 levels above the W126 level of 13 ppm-hour.

These results suggest that meeting a proposed 0.070 ppm, 8-hour secondary standard would provide substantially improved protection in some areas for vegetation from seasonal O_3 exposures of concern. The 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 O_3 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.

[59] The CMAQ model is a multi-pollutant, multiscale air quality model that contains state-of-the-science techniques for simulating all 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/O_3, particulate matter, and nutrient deposition) holistically. The CMAQ model can generate estimates of hourly O_3 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).

To further characterize O_3 air quality in terms of current and alternative secondary standard forms, an analysis was performed in the Staff Paper to evaluate the extent to which county-level O_3 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 Staff Paper presented this analysis using recent (2002–2004) [60] county-level O_3 air quality data from AQS sites and the subset of CASTNET sites having the highest O_3 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 O_3 exposures and are typically evaluated with respect to exposures within the annual growing season, the 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 the 0.08 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-hours, 135 counties with air quality meeting the 3-year average form of the 0.08 ppm, 8-hour average standard, would be above this W126 level. In addition, when the 3-year average of the 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 O_3 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 O_3 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.

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

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 O_3 air quality distributions at high elevation sites often do not reflect the typical urban and near-urban pattern of low morning and evening O_3 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 Staff Paper concludes 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 Criteria Document (EPA, 2006a), 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 (O_3 produced from anthropogenic emissions outside North America); and (2) stratospheric intrusions might occasionally elevate O_3 at high-altitude sites, however, these events are rare. Therefore, the Staff Paper concludes that springtime PRB levels in the range identified above and rare stratospheric intrusions of O_3 are unlikely to influence 3 month cumulative seasonal W126 values significantly.

It further remains uncertain as to the extent to which air quality improvements designed to reduce 8-hour O_3 average concentrations would reduce O_3 exposures measured by a seasonal, cumulative W126 index. The 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 O_3 concentrations in relation to plant response; and (3) the effects of attainment of an 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 Risk to Vegetation

The Staff Paper presents results from quantitative and qualitative risk assessments of O_3 risks to vegetation (EPA, 2007). In the last 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 O_3-related vegetation risks, especially those currently faced by seedling, sapling and mature tree species growing in field settings, and indirectly, forested ecosystems. Specifically, new research 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 O_3 exposures.

a. Visible Foliar Injury

As discussed earlier (Section A), recent systematic injury surveys continue to document visible foliar injury symptoms diagnostic of phytotoxic O_3 exposures on sensitive bioindicator plants. These surveys produced more expansive evidence than that available at the time of the last review that visible foliar injury is occurring in many areas of the U.S. under current ambient conditions. The 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 the current 0.08 8-hour standard. Of the counties that met an 8-hour level of 0.07 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 the current secondary standard or alternative 0.07 ppm 8-hour standard. Additionally, the data show that visible foliar injury occurrence is geographically widespread and is 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 O_3-related vegetation effects could also be present.

b. Seedling and Mature Tree Biomass Loss

In the last review (EPA, 1996b), analyses of the effects of O_3 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, tulip poplar were found to be sensitive to cumulative seasonal O_3 exposures. Work done since the 1996 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 O_3 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 analysis, C–R functions for biomass loss for available seedling tree species taken from the CD 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 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 Staff Paper. For example, quaking aspen had a wide range of O_3 exposure across its growing range and therefore, showed significant variability in 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 O_3 air quality was adjusted to meet the current 8-hour standard. 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 O_3 air quality was rolled back to just meet the current 8-hour standard. The 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 O_3 exposure. Due to the potential for compounding effects over multiple years, a consensus workshop on O_3 effects reported 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 O_3 effects on seedling growth, recent work has enhanced our understanding of risks beyond the seedling stage. In order to better characterize the potential O_3 effects on mature tree growth, a tree growth model (TREGRO) was used as a tool to evaluate the effect of changing O_3 air quality scenarios from just meeting alternative O_3 standards on the growth of mature trees. TREGRO is a process-based, individual tree growth simulation model (Weinstein *et al.,* 1991) and has been used to evaluate the effects of a variety of O_3 scenarios and linked with concurrent climate data to account for O_3 and climate/meteorology interactions on several species of trees in different regions of the U.S. (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 O_3 exposure and response. The interactions between O_3 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 O_3 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 Staff Paper analyses found that just meeting the current standard would likely continue to allow O_3-related reductions in annual net biomass gain in these species. This is based on model outputs that estimate that as O_3 levels are reduced below those of the current 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 O_3 (King *et al.,* 2005). Some mature trees such as red oak have shown an even greater sensitivity of photosynthesis to O_3 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 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 Staff Paper, risk of O_3 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 Staff Paper analysis accounted for 69 percent of 2004 principal crop acreage planted in the U.S. in 2004.[61] 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 last review (Abt 1995). The 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 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. 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 current 8-hour standard would still allow O_3-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 O_3 levels, potatoes and cotton had estimated yield losses of 9–15 percent and 5–10 percent, respectively, when O_3 air quality just met the level of the current 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 current standard (0–4 percent).

The Staff Paper also presented estimates of monetized benefits for crops associated with the current 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 current and alternative standard levels were met. Meeting the various alternative standards did show some significant benefits beyond the current 8-hour standard. However, the Staff Paper recognized the AGSIM modeled economic benefits had many uncertainties: For example, much of the economic benefits were from the fruits and vegetables which had uncertain C–R relationships, there was uncertainty in assumptions about the treatment and effect of government farm payment programs, and there was also uncertainty about near-term changes in 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, the uncertainties limited the utility of the absolute numbers.

D. Conclusions on the Adequacy of the Current Standard

1. Background

The initial issue to be addressed in the current review of the secondary O_3 standard is whether, in view of the advances in scientific knowledge reflected in the Criteria Document and additional information on exposure and risk discussed in the Staff Paper, the existing standard should be revised. The current secondary standard is a 3-year average of the annual 4th-highest maximum 8-hour average O_3 concentration set at a level of 0.08 ppm. In evaluating whether it is appropriate to retain or revise the current secondary O_3 standard, the Administrator adopts an approach in this review that builds upon the general approach used in the last review and reflects the broader body of evidence now available.

In developing proposed conclusions on the adequacy of the current secondary O_3 standard, the Administrator has considered a weight-of-evidence approach that evaluated information across the variety of vegetation-related research areas described in the Criteria Document (*e.g.*, seedling, sapling and mature forest tree species growth stages and commodity, fruit, vegetable and forage crop species), and included the assessments of air quality, exposures, and qualitative and quantitative risks associated with alternative air quality scenarios. Evidence-based considerations included assessment of vegetation effects evidence obtained from chamber, free air, gradient, model and field-based observation studies across an array of vegetation effects endpoints. Exposure- and risk-based considerations were drawn from exposure and risk assessments that relied upon both monitored and interpolated O_3 exposures as described in the Staff Paper. These assessments reflect the availability of new tools and assessment methods, as well as the larger and more diverse body of evidence available since the last review. Specifically, estimates of exposures and risks associated with recent O_3 air quality levels, as well as estimates of the relative magnitude of exposure and risk reductions potentially associated with meeting the current 8-hour secondary O_3 NAAQS and alternative standards, have also been considered, along with all known associated uncertainties.

In this review, a series of general questions frames the approach to reaching a proposed decision on the adequacy of the current standard, beginning with: (1) To what extent does newly available information reinforce or call into question evidence of associations of O_3 exposures with effects identified in the last review?; (2) to what extent does newly available information reinforce or call into question any of the basic elements of the current standard?;

[61] 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*).

and (3) to what extent have important uncertainties identified in the last review been reduced and have new uncertainties emerged? To the extent the available information suggests that revision of the current standard may be appropriate, the question of whether the available information supports consideration of a standard that is either more or less protective than the current standard is addressed, including: (1) Whether there is evidence that vegetation effects extend to ambient O_3 concentration levels that are as low as or lower than had previously been observed, and what are the important uncertainties associated with that evidence?; (2) whether vegetation exposures and risks of concern estimated to occur in areas upon meeting the current standard are considered important from a public welfare perspective; and (3) what are the important uncertainties associated with the estimated risks?

The current secondary standard was selected to provide protection to the public welfare against a range of O_3-induced vegetation effects, particularly yield loss in agricultural crops and biomass loss in tree seedlings. As an introduction to the discussion in this section of the adequacy of the current O_3 standard, it is useful to summarize the key factors that formed the basis of the decision in the last review to revise the averaging time, level and form of the then current 1-hour secondary standard.

In the 1996 proposal notice (61 FR 65716), the Administrator proposed to replace the then existing 1-hour O_3 secondary NAAQS with one of two alternative new standards: a standard identical to the proposed and now current 0.08 ppm, 8-hour primary standard (described above), or alternatively, a new seasonal standard, SUM06, expressed as a sum of hourly concentrations greater than or equal to 0.06 ppm, cumulated daily over a 12 hour daylight window (8 am to 8 pm) during the maximum consecutive 3-month period (*e.g.*, the consecutive 3 month period with the highest SUM06 index value) during the O_3 monitoring season, set at a level of 25 ppm-hours. The latter form and level were selected to provide protection to vegetation on the basis of annual, rather than 3-year average, exposures.

In the final rule for the O_3 NAAQS published in July 1997 (62 FR 38877), the Administrator decided to replace the then current 1-hour, 0.12-ppm secondary NAAQS with a standard that was identical in every way to the new revised primary standard of an 0.08 ppm annual 4th-highest maximum 8-hour average standard averaged over 3 years. Her decision was based on: (1) Her judgment that the then existing secondary standard did not provide adequate protection for vegetation against the adverse welfare effects of O_3; (2) CASAC advice ''that a secondary NAAQS, more stringent than the present primary standard, was necessary to protect vegetation from O_3'' (Wolff, 1996); (3) her judgment that the new 8-hour average standard would provide substantially improved protection for vegetation from O_3-related adverse effects as compared to the level of protection provided by the then current 1-hour, 0.12-ppm secondary standard; (4) recognition that significant uncertainties remained with respect to exposure dynamics, air quality relationships, and the exposure, risk, and monetized valuation analyses presented in the proposal, resulting in only rough estimates of the increased public welfare likely to be afforded by each of the proposed alternative standards; (5) her judgment that there was value in allowing more time to obtain additional information to better characterize O_3-related vegetation effects under field conditions from additional research and to develop a more complete rural monitoring network and air quality database from which to evaluate the elements of an appropriate seasonal secondary standard; and (6) her judgment that there was value in allowing more time to evaluate more specifically the improvement in rural air quality and in O_3-related vegetation effects resulting from measures designed to attain the new primary standard (62 FR 38877–78).

The Administrator further concluded (62 FR 38877–78) that continued research on the effects of O_3 on vegetation under field conditions and on better characterizing the relationship between O_3 exposure dynamics and plant response would be important in the next review because: (1) The available biological database highlighted the importance of cumulative, seasonal exposures as a primary determinant of plant responses; (2) the association between daily maximum 8-hour O_3 concentrations and plant responses had not been specifically examined in field tests; (3) the impacts of attaining an 8-hour, 0.08 ppm primary standard in upwind urban areas on rural air quality distributions could not be characterized with confidence due to limited monitoring data and air quality modeling in rural and remote areas.

2. Evidence- and Exposure/Risk-Based Considerations

The new evidence available in this review as described in the Criteria Document continues to support and strengthen key policy-relevant conclusions drawn in the previous review (EPA, 2006a). Based on this new evidence, the current Criteria Document once more concludes that: (1) A plant's response to O_3 depends upon the cumulative nature of ambient exposure as well as the temporal dynamics of those concentrations; (2) current ambient concentrations in many areas of the country are sufficient to impair growth of numerous common and economically valuable plant and tree species; (3) the entrance of O_3 into the leaf through the stomata is the critical step in O_3 effects; (4) effects can occur with only a few hourly concentrations above 0.08 ppm; (5) other environmental biotic and abiotic factors are also influential to the overall impact of O_3 on plants and trees; and (6) a high degree of uncertainty remains in our ability to assess the impact of O_3 on ecosystem services.

In light of the new evidence, as described in the Criteria Document, the Staff Paper evaluates the adequacy of the current standard based on assessments of both the most policy-relevant vegetation effects evidence and exposure and risk-based information, as summarized above in sections IV.A and IV.C, respectively. In evaluating the strength of this information, the Staff Paper takes into account the uncertainties and limitations in the scientific evidence and analyses as well as the views of CASAC. The Staff Paper concludes that progress has been made since the last review and generally finds support in the available effects- and exposure/risk-based information for consideration of an O_3 standard that is more protective than the current standard. The Staff Paper further concludes that there is no support for consideration of an O_3 standard that is less protective than the current standard. This general conclusion is consistent with the advice and recommendations of CASAC.

a. Evidence-Based Considerations

In the last review, crop yield and tree seedling biomass loss data obtained in OTC studies provided the basis for the Administrator's judgment that the then current 1-hour, 0.12 ppm secondary standard was inadequate (EPA, 1996b). Since then, several additional lines of evidence have progressed sufficiently to provide a more complete and coherent picture of the scope of O_3-related

vegetation risks, especially those currently faced by sensitive seedling, sapling and mature growth stage tree species growing in field settings, and their associated forested ecosystems. Specifically, new research reflects an increased emphasis on field-based exposure methods (*e.g.*, free air, ambient gradient, and biomonitoring surveys). In reaching conclusions regarding the adequacy of the current standard, the Staff Paper has considered the combined information from all these areas together, along with associated uncertainties, in an integrated, weight-of-evidence approach.

Regarding the O_3-induced effect of visible foliar injury, observations for the years 2001 to 2004 at USDA FIA biomonitoring sites showed widespread O_3-induced leaf injury occurring in the field, including in forested ecosystems, under current ambient O_3 conditions. For a few studied species, it has been shown that the presence of visible foliar injury is further linked to the presence of other vegetation effects (*e.g.*, reduced plant growth and impaired below ground root development) (EPA, 2006), though for most species, this linkage has not been specifically studied or where studied, has not been found. Nevertheless, when visible foliar injury is present, the possibility that other O_3-induced vegetation effects could also be present for some species should be considered. Likewise, the absence of visible foliar injury should not be construed to demonstrate the absence of other O_3-induced vegetation effects. The Staff Paper concludes that it is not possible at this time to quantitatively assess the degree of visible foliar injury that should be judged adverse in all settings and across all species, and that other environmental factors can mitigate or exacerbate the degree of O_3-induced visible foliar injury expressed at any given concentration of O_3. However, the Staff Paper also concludes that the presence of visible foliar injury alone can be adverse to the public welfare, especially when it occurs in protected areas such as national parks and wilderness areas. Thus, on the basis of the available information on the widespread distribution of O_3-sensitive species within the U.S. including in areas, such as national parks, which are afforded a higher degree of protection, the Staff Paper concludes that the current standard continues to allow levels of visible foliar injury in some locations that could reasonably be considered to be adverse from a public welfare perspective. Additional monitoring of both O_3 air quality and foliar injury levels are needed in these areas of national significance to more fully characterize the spatial extent of this public welfare impact.

With respect to O_3-induced biomass loss in trees, the Staff Paper concludes that the significant new body of field-based research on trees strengthens the conclusions drawn on tree seedling biomass loss from earlier OTC work by documenting similar seedling responses in the field. For example, recent empirical studies conducted on quaking aspen at the AspenFACE site in Wisconsin have confirmed the detrimental effects of O_3 exposure on tree growth in a field setting without chambers (Isebrands *et al.*, 2000, 2001). In addition, results from an ambient gradient study (Gregg *et al.*, 2003), which evaluated biomass loss in cottonwood along an urban-to-rural gradient at several locations, found that conditions in the field were sufficient to produce substantial biomass loss in cottonwood, with larger impacts observed in downwind rural areas due to the presence of higher O_3 concentrations. These gradients from low urban to higher rural O_3 concentrations occur when O_3 precursors generated in urban areas are transported to downwind sites and are transformed into O_3. In addition,O_3 concentrations typically fall to near 0 ppm at night in urban areas due to scavenging of O_3 by NO_X and other compounds. In contrast, rural areas, due to a lack of nighttime scavenging, tend to maintain elevated O_3 concentrations for longer periods. On the basis of such key studies, the Staff Paper concludes that the expanded body of field-based evidence, in combination with the substantial corroborating evidence from OTC data, provides stronger evidence than that available in the last review that ambient levels of O_3 are sufficient to produce visible foliar injury symptoms and biomass loss in sensitive vegetative species growing in natural environments. Further, the Staff Paper judges that the consistency in response in studied species/genotypes to O_3 under a variety of exposure conditions and methodologies demonstrates that these sensitive genotypes and populations of plants are susceptible to adverse impacts from O_3 exposures at levels known to occur in the ambient air. Due to the potential for compounded risks from repeated insults over multiple years in perennial species, the Staff Paper concludes that these sensitive subpopulations are not afforded adequate protection under the current secondary O_3 standard. Despite the fact that only a relatively small portion of U.S. plant species have been studied with respect to O_3 sensitivity, those species/genotypes shown to have O_3 sensitivity span a broad range of vegetation types and public use categories, including direct-use categories like food production for human and domestic animal consumption; fiber, materials, and medicinal production; urban/private landscaping. Many of these species also contribute to the structure and functioning of natural ecosystems (*e.g.*, the EEAs) and thus, to the goods and services those ecosystems provide (Young and Sanzone, 2002), including non-use categories such as relevance to public welfare based on their aesthetic, existence or wildlife habitat value.

The Staff Paper therefore concludes that the current secondary standard is inadequate to protect the public welfare against the occurrence of known adverse levels of visible foliar injury and tree seedling biomass loss occurring in tree species (*e.g.*, ponderosa pine, aspen, black cherry, cottonwood) that are sensitive and clearly important to the public welfare.

b. Exposure- and Risk-Based Considerations

The Staff Paper also presents the results of exposure and risk assessments. Due to multiple sources of uncertainty, both known and unknown, that continue to be associated with these analyses, the Staff Paper put less weight on this information in drawing conclusions on the adequacy of the current standard. However, the Staff Paper also recognizes that some progress has been made since the last review in better characterizing some of these associated uncertainties and, therefore concluded that the results of the exposure and risk assessments continue to provide information useful to informing judgments as to the relative changes in risks predicted to occur under exposure scenarios associated with the different standard alternatives considered. Importantly, with respect to two key uncertainties, the uncertainty associated with continued reliance on C–R functions developed from OTC exposure systems to predict plant response in the field and the potential for changes in tree seedling and crop sensitivities in the intervening period since the C–R functions were developed, the Staff Paper concluded that recent research has provided information useful in judging how much weight to put on these concerns. Specifically, new field-based studies, conducted on a limited number of tree seedling and crop species to date, demonstrate plant growth and visible foliar injury responses in the field that

are similar in nature and magnitude to those observed previously under OTC exposure conditions, lending qualitative support to the conclusion that OTC conditions do not fundamentally alter the nature of the O_3-plant response. Second, nothing in the recent literature suggests that the O_3 sensitivity of crop or tree species studied in the last review and for which C–R functions were developed has changed significantly in the intervening period. Indeed, in the few recent studies where this is examined, O_3 sensitivities were found to be as great as or greater than those observed in the last review.

i. Seedling and Mature Tree Biomass Loss

Biomass loss in sensitive tree seedlings is predicted to occur under O_3 exposures that meet the level of the current secondary standard. For instance, black cherry, ponderosa pine, eastern white pine, and aspen had estimated median seedling biomass losses as high as 24, 11, 6, and 6 percent, respectively, over some portions of their growing ranges when air quality was rolled back to meet the current 8-hr standard with the 10 percent downward adjustment for the potential O_3 gradient between monitor height and short plant canopies applied. The Staff Paper notes that these results are for tree seedlings and that mature trees of the same species may have more or less of a response to O_3 exposure. Decreased root growth associated with biomass loss has the potential to indirectly affect the vigor and survivability of tree seedlings. If such effects occur on a sufficient number of seedlings within a stand, overall stand health and composition can be affected in the long term. Thus, the Staff Paper concludes that these levels of estimated tree seedling growth reduction should be considered significant and potentially adverse, given that they are well above the 2 percent level of concern identified by the 1997 consensus workshop (Heck and Cowling, 1997).

Though there is significant uncertainty associated with this analysis, the Staff Paper recommends that this information should be given careful consideration in light of several other pieces of evidence. Specifically, limited evidence from experimental studies that go beyond the seedling growth stage continues to show decreased growth under elevated O_3 levels (King *et al.*, 2005). Some mature trees such as red oak have shown an even greater sensitivity of photosynthesis to O_3 than seedlings of the same species (Hanson *et al.*, 1994). The potential for effects to ''carry over'' to the following year or cumulate over multiple years, including the potential for compounding, must be considered. The accumulation of such ''carry-over'' effects over time may affect long-term survival and reproduction of individual trees and ultimately the abundance of sensitive tree species in forest stands.

ii. Qualitative Ecosystem Risks

In addition to the quantifiable risk categories discussed above, the Staff Paper presents qualitative discussions on a number of other public welfare effects categories. In so doing, the Staff Paper concludes that the quantified risks to vegetation estimated to be occurring under current air quality or upon meeting the current secondary standard likely represent only a portion of actual risks that may be occurring for a number of reasons.

First, as mentioned above, out of the over 43,000 plant species catalogued as growing within the U.S. (USDA PLANTS database, USDA, NRCS, 2006), only a small percentage have been studied with respect to O_3 sensitivity. Most of the studied species were selected because of their commercial importance or observed O_3-induced visible foliar injury in the field. Given that O_3 impacts to vegetation also include less obvious but often more significant impacts, such as reduced annual growth rates and below ground root loss, the paucity of information on other species means the number of O_3-sensitive species that exists within U.S., could be greater than what is now known. Since no state in the lower 48 states has less than seven known O_3-sensitive plant species, with the majority of states having between 11 and 30 (see Appendix 7J–2 in Staff Paper), protecting O_3 sensitive vegetation is clearly important to the public welfare at the national scale.

Second, the Staff Paper also takes into consideration the possibility that more subtle and hidden risks to ecosystems are potentially occurring in areas where vegetation is being significantly impacted. Given the importance of these qualitative and anticipated risks to important public welfare effects categories such as ecosystem impacts leading to potential losses or shifts in ecosystem goods and services (*e.g.*, carbon sequestration, hydrology, and fire disturbance regimes), the Staff Paper concludes that any secondary standard set to protect against the known and quantifiable adverse effects to vegetation should also consider the anticipated, but currently unquantifiable, potential effects on natural ecosystems.

iii. Crop Yield Loss

Exposure and risk assessments in the Staff Paper estimated that meeting the current 8-hour standard would still allow O_3-related yield loss to occur in several fruit and vegetable and commodity crop species currently grown in the U.S. These estimates of crop yield loss are substantially lower than those estimated in the last review as a result of several factors, including adjusted exposure levels to reflect the presence of a variable O_3 gradient between monitor height and crop canopies, and use of a different econometric agricultural benefits model updated to reflect more recent agricultural policies (EPA, 2006b). Though these sources of uncertainty associated with the crop risk and benefits assessments were better documented in this review, the Staff Paper concludes that the presence of these uncertainties make the risk estimates suitable only as a basis for understanding potential trends in relative yield loss and economic benefits. The Staff Paper further recognizes that actual conditions in the field and management practices vary from farm to farm, that agricultural systems are heavily managed, and that adverse impacts from a variety of other factors (*e.g.*, weather, insects, disease) can be orders of magnitude greater than that of yield impacts predicted for a given O_3 exposure. Thus, the relevance of such estimated impacts on crop yields to the public welfare are considered highly uncertain and less useful as a basis for assessing the adequacy of the current standard. The Staff Paper notes, however, that in some experimental cases, exposure to O_3 has made plants more sensitive or vulnerable to some of these other important stressors, including disease, insect pests, and harsh weather (EPA, 2006a). The Staff Paper therefore concluded that this remains an important area of uncertainty and that additional research to better characterize the nature and significance of these interactions between O_3 and other plant stressors would be useful.

c. Summary

In summary, the Staff Paper concludes that the current secondary O_3 standard is inadequate. This conclusion is based on the extensive vegetation effects evidence, in particular the recent empirical field-based evidence on biomass loss in seedlings, saplings and mature trees, and foliar injury incidence that has become available in this review, which demonstrates the occurrence of adverse vegetation effects at ambient

levels of recent O_3 air quality, as well as evidence and exposure- and risk-based analyses indicating that adverse effects would be predicted to occur under air quality scenarios that meet the current standard.

3. CASAC Views

In a letter to the Administrator (Henderson, 2006c), the CASAC O_3 Panel, with full endorsement of the chartered CASAC, unanimously concluded that ''despite limited recent research, it has become clear since the last review that adverse effects on a wide range of vegetation including visible foliar injury are to be expected and have been observed in areas that are below the level of the current 8-hour primary and secondary ozone standards.'' Therefore, ''based on the Ozone Panel's review of Chapters 7 and 8 [of the Staff Paper], the CASAC unanimously agrees that it is not appropriate to try to protect vegetation from the substantial, known or anticipated, direct and/or indirect, adverse effects of ambient O_3 by continuing to promulgate identical primary and secondary standards for O_3. Moreover, the members of the Committee and a substantial majority of the Ozone Panel agree with EPA staff conclusions and encourage the Administrator to establish an alternative cumulative secondary standard for O_3 and related photochemical oxidants that is distinctly different in averaging time, form and level from the currently existing or potentially revised 8-hour primary standard'' (Henderson, 2006c).[62]

4. Administrator's Proposed Conclusions Concerning Adequacy of Current Standard

The Administrator recognizes that the secondary standard is to protect against ''adverse'' O_3 effects, discussed above in section IV.A.3. In considering what constitutes a vegetation effect that is also adverse to the public welfare, the Administrator took into account the Staff Paper conclusions regarding the nature and strength of the vegetation effects evidence, the exposure and risk assessment results, the degree to which the associated uncertainties should be considered in interpreting the results, and the views of CASAC and members of the public. On these bases, the Administrator proposes that the current secondary standard is inadequate to protect the public welfare from known and anticipated adverse O_3-related effects on vegetation and ecosystems. Ozone levels that would be expected to remain after meeting the current secondary standard are sufficient to cause visible foliar injury, seedling and mature tree biomass loss, and crop yield reductions to degrees that could be considered adverse depending on the intended use of the plant and its significance to the public welfare, and the current secondary standard does not provide adequate protection from such effects. Other O_3-induced effects described in the literature, including an impaired ability of many sensitive species and genotypes within species to adapt to or withstand other environmental stresses, such as freezing temperatures, pest infestations and/or disease, and to compete for available resources, would also be anticipated to occur. In the long run, the result of these impairments (*e.g.*, loss in vigor) could lead to premature plant death in O_3 sensitive species. Though effects on other ecosystem components have only been examined in isolated cases, effects such as those described above could have significant implications for plant community and associated species biodiversity and the structure and function of whole ecosystems. These considerations also support the proposed conclusion that the current secondary standard is not adequate and that revision is needed to provide additional public welfare protection.

E. Conclusions on the Elements of the Secondary Standard

Given his proposed conclusion that the current secondary standard is inadequate, the Administrator then considered what revisions to the standard are appropriate. In so doing, the Administrator has focused on revisions to the key standard elements of indicator, form, averaging time, and level. On the basis of the strength and coherence of the vegetation effects evidence suggesting that a biologically-based standard for vegetation, at a minimum, should cumulate exposures and differentially-weight higher O_3 concentrations, the Administrator judges that it is appropriate to consider revisions to the secondary standard that reflect this understanding. In addition, the Administrator also judges that the current 8-hour average form, though not based on the most biologically relevant and coherent vegetation effects literature, can also provide substantially improved protection to vegetation when set at an appropriate level. Therefore, the Administrator also considered whether revision to the level of the current 8-hour secondary standard might provide the requisite level of public welfare protection. In light of these considerations, as discussed below, the Administrator is proposing two options for revising the current secondary standard: one option is a cumulative seasonal standard (section IV.E.2) and the other option is an 8-hour average standard consistent with the revised 8-hour average standard proposed above for the primary standard (section IV.E.3). The Administrator has also considered an alternative approach to setting a cumulative, seasonal standard(s) as described below in section IV.E.2.

1. Indicator

In the last review, EPA focused on a standard for O_3 as the most appropriate surrogate for ambient photochemical oxidants. In this review, while the complex atmospheric chemistry in which O_3 plays a key role has been highlighted, no alternatives to O_3 have been advanced as being a more appropriate surrogate for ambient photochemical oxidants. Thus, as is the case for the primary standard, (discussed above in section II.D.1.), the Administrator proposes to continue to use O_3 as the indicator for a standard that is intended to address effects associated with exposure to O_3, alone and in combination with related photochemical oxidants. In so doing, the Administrator recognizes that measures leading to reductions in vegetation exposures to O_3 will also reduce exposures to other photochemical oxidants.

2. Cumulative, Seasonal Standard

The Administrator proposes to replace the current secondary standard with a new cumulative, seasonal standard expressed as an index of the annual sum of weighted hourly concentrations (using the W126 form), set at a level in the range of 7 to 21 ppm-hours. The index would be cumulated over the 12-hour daylight period (8 a.m. to 8 p.m.) during the consecutive 3-month period within the O_3 season with the maximum index value. In addition, as discussed below, the Administrator is considering an alternative approach to setting a cumulative, seasonal standard(s) that would afford differing degrees of protection for O_3-related impacts on different types of vegetation with different intended uses.

[62] One CASAC Panel member reached different conclusions from those of the broader Panel regarding certain aspects of the vegetation effects information and the appropriate degree of emphasis that should be placed on the associated uncertainties. These concerns related to how the results of O_3/vegetation exposure experiments carried out in OTC can be extrapolated to the ambient environment and how C–R functions developed in the 1980's can be used today given that he did not expect that current crop species/cultivars in use in 2002 would have the same O_3 sensitivity as those studied in NCLAN (Henderson, 2007, pg. C–18).

a. Form

The current Criteria Document and Staff Paper concluded that the recent vegetation effects literature evaluated in this review strengthens and reaffirms conclusions made in the last 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 1996 review focused in particular on two of these cumulative forms, the SUM06 and W126. As described in the last review (EPA, 1996a, b) it was concluded that, based on statistical reanalysis of the NCLAN data, these different cumulative forms performed equally well in predicting crop yield loss response to O_3 exposure. Given that the data available at that time were unable to distinguish between these forms, the Administrator, based on the policy consideration of not including O_3 concentrations considered to be within the PRB, concluded that the SUM06 form was the more appropriate choice for a secondary standard.

In this review, the Staff Paper evaluated the continued appropriateness of the SUM06 form in light of two key pieces of information: new estimates of PRB that are lower than in the last 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 Staff Paper concluded that the W126 form was more appropriate in the context of this review. 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 O_3 concentrations within the range of estimated PRB.

The CASAC, based on its assessment of the same vegetation effects science, agreed with the Criteria Document and Staff Paper and unanimously concluded that it is not appropriate to try to protect vegetation from the known or anticipated adverse effects of ambient O_3 by continuing to promulgate identical primary and secondary standards for O_3. Moreover, the members of CASAC and a substantial majority of the CASAC O_3 Panel agreed with Staff Paper conclusions and encouraged the Administrator to establish an alternative cumulative secondary standard for O_3 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).[63]

The Administrator agrees with the conclusions drawn in the Criteria Document, Staff Paper and by CASAC that the scientific evidence available in the current review continues to demonstrate the cumulative nature of O_3-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 O_3 concentrations represents a reasonable policy choice for a seasonal secondary standard to protect against the effects of O_3 on vegetation. The Administrator further agrees with both the Staff Paper and CASAC that the most appropriate cumulative, concentration-weighted form to consider in this review is the sigmoidally weighted W126 form, due to his recognition that there is no evidence in the literature for an exposure threshold that would be appropriate across all O_3-sensitive vegetation and that this form is unlikely to be significantly influenced by O_3 air quality within the range of PRB levels identified in this review. Thus, the Administrator proposes as one option to replace the current 8-hour average secondary standard form with the cumulative, seasonal W126 form.

b. Averaging Times [64]

The Staff Paper, in addition to form, also considers what ''averaging'' periods or exposure 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 O_3 levels are not continuously elevated and plants are not equally sensitive to O_3 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. Exposure periods are discussed below in terms of a seasonal window, a diurnal window, and an annual versus 3-year average standard.

(1) In considering an appropriate seasonal window, the Staff Paper recognizes 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 O_3 uptake for annual crops, herbaceous species, and deciduous trees and shrubs coincides with some or all of the intra-annual period defined as the O_3 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 O_3 also promote physiological activity in vegetation.

The Staff Paper notes 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 O_3 exposures. However, the Staff Paper further concludes that the consecutive 3-month period within the O_3 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 Staff Paper again concludes, as it did in 1996, 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 recognizes 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 CO_2 uptake for use in carbohydrate production through the light-driven process of photosynthesis. At most locations, O_3 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

[63] One CASAC Panel member expressed the view that the O_3 exposure indices, SUM06 and W126, are simply mathematical expressions of exposure and, thus, cannot be said to have a biological basis (Henderson, 2007, pg. C–18).

[64] While the term ''averaging time'' is used, for the cumulative, seasonal standard the time period at issue is one over which exposures during a specified period of time are cumulated, not averaged.

open sufficiently at night to allow for some nocturnal uptake to occur. In addition, at some rural, high elevation sites, the O_3 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 O_3 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 O_3 uptake at night adversely impacts plant function. In addition, many species do not take up O_3 at night or occur in areas with elevated nighttime O_3 concentrations.

In light of a recent work on this topic conducted by Musselman and Minnick (2000), the Staff Paper again revisited the issue of what diurnal period is of most relevance in influencing O_3-induced effects on vegetation. This work reports that some species take up O_3 at night, but that the degree of nocturnal stomatal conductance varies widely between species and its relevance to overall O_3-induced vegetation effects remain unclear. In considering this information, the Staff Paper concludes that for the vast majority of studied species, daytime exposures represent the majority of diurnal plant O_3 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 O_3 exposures exists, and what levels of nighttime uptake are adverse to affected species, the Staff Paper concludes that this information continues to be preliminary, and does not provide a basis for reaching a different conclusion at this time. The Staff Paper further notes 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 O_3 co-occur with sensitive species with a high degree of nocturnal stomatal conductance. Thus, as in the last review, the Staff Paper again concludes 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 O_3 exposures, specifically those most relevant to plant growth and yield responses.

(3) In considering whether an annual or 3-year averaging period is more appropriate, the 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 averaging period for purposes of standard stability. However, the 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, while for other welfare effects (*e.g.*, mature tree biomass loss), a 3-year averaging period may also be appropriate. Thus, the Staff Paper concludes that it is appropriate to consider both an annual and a 3-year averaging period. Further, the Staff Paper concludes that should a 3-year average of the 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 O_3 year that could still occur while attaining a standard on average over 3-years.

The CASAC, in considering what seasonal and diurnal time periods are most appropriate when combined with a cumulative, concentration-weighted form to protect vegetation from exposures of concern, agreed that the Staff Paper conclusion regarding the 3-month seasonal period and 12-hour daylight window was appropriate, with the distinction that both time designations likely represents the minimum time periods of importance. In particular, one O_3 Panel member commented that for some species, additional O_3 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 maximum 8-hour concentrations. CASAC 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 determining which seasonal and diurnal time periods are most appropriate to propose, took into account Staff Paper and CASAC views. The Administrator, in being careful to consider what is needed to provide the requisite degree of protection, no more and no less, proposes that the 3-month seasonal period and 12-hour daylight period are appropriate. Based on the Staff Paper conclusions discussed above, the Administrator is mindful that there is the potential for under-protection with a 12-hour diurnal window in areas with sufficiently elevated nighttime levels of O_3 where sensitive species with a high degree of nocturnal stomatal conductance occur. On the other hand, the Administrator also recognizes that a longer diurnal window (*e.g.*, 24-hour) has the possibility of over-protecting vegetation in areas where nighttime O_3 levels remain relatively high but where no species having significant nocturnal uptake exist. In weighing these considerations, the Administrator agrees with the Staff Paper conclusion that until additional information is available about the extent to which this co-occurrence of sensitive species and elevated nocturnal O_3 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 Administrator also considered to what extent the 3-month period within the O_3 season was appropriate, recognizing that many species of vegetation have longer growing seasons. The Administrator further proposes that the maximum 3-month period is sufficient and appropriate to characterize O_3 exposure levels associated with known levels of plant response. Therefore, the Administrator proposes 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 O_3 monitoring season with the maximum W126 index value.

The Administrator also proposes an annual rather than a multi-year cumulative, seasonal standard. In proposing this alternative, the Administrator also believes that it is appropriate to consider the benefits to the public welfare that would accrue from establishing a 3-year average secondary standard, and solicits comment on this alternative. In so doing, the Administrator also agrees with 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 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 O_3 exposures, considers what information from the array of vegetation effects evidence and exposure and risk assessment results was most useful. In regards to the vegetation effects evidence, the Staff Paper finds stronger support than what was available at the time of the last 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 O_3-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 O_3 exposures; and (4) increased recognition and understanding of the structure and function of ecosystems and the complex linkages through which O_3, 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 Staff Paper concludes that just meeting the 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 air quality 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 Staff Paper also considers the more subtle impacts of O_3 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, O_3 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 Staff Paper concludes 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 Staff Paper also recognizes that in the last review, the Administrator took into account the results of a 1996 consensus-building workshop as described in a January 1997 report (Heck and Cowling, 1997). 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 O_3-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 range of 10 to 15 (7 to 13) ppm-hours would be protective. For growth effects to tree seedlings and saplings in plantations, the consensus range was 12 to 16 (9 to 14) ppm-hours. For visible foliar injury to natural ecosystems, the consensus range was 8 to 12 (5 to 9) ppm-hours (Heck and Cowling, 1997).

Taking these consensus statements into account, the Administrator stated in the 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 O_3 secondary standard'' (62 FR 38856).

Given the importance the Administrator put on the consensus report in the last review, the Staff Paper considered to what extent new research provided empirical support for the ranges of levels identified by the experts as protective of different types of O_3-induced effects. On the basis of new field-based tree seedling growth loss and foliar injury data, and including both the above quantitative and qualitative information regarding O_3-induced effects on sensitive trees and forested ecosystems, the Staff Paper concludes that it is appropriate to consider a range for a 3-month, 12-hour, W126 standard that includes the consensus recommendations for growth effects in tree seedlings in natural forest stands.

In considering the newly available information on O_3-related effects on crops in this review, the Staff Paper observes the following regarding the strength of the underlying crop science: (1) Nothing in the recent literature points to a change in the relationship between O_3 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 Staff Paper concludes that nothing in the newly assessed information calls into question the strength of the underlying science upon which the Administrator based her 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 Staff Paper then considered whether any additional information is available to inform judgments as to the adversity of various O_3-induced levels of crop yield loss to the public welfare. As noted above, the Staff Paper observes that agricultural systems are heavily managed, and that in addition to stress from O_3, 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 O_3 exposures. On the other hand, O_3 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 Staff Paper concludes that the level of protection judged requisite in the last review to protect the public welfare from adverse levels of O_3-induced reductions in crop yields, as provided by a W126 level of 21 ppm-hours, remains appropriate for consideration as an upper bound of a range of appropriate levels.

Thus, the Staff Paper concludes, based on all the above considerations, that an appropriate range of 3-month, 12-hour W126 levels is 7 to 21 ppm-hours, 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 Staff Paper recognizes 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 Staff Paper recommendations that the lower bound of the range within which a seasonal W126 welfare-based (secondary) O_3 standard should be considered is approximately 7 ppm-hours; however, it did not agree with Staff's recommendation that the upper bound of the range should be as high as 21 ppm-hours. Rather, CASAC recommended that the upper bound of the range considered should be no higher than 15 ppm-hours, which the Panel estimates is approximately equivalent to a seasonal 12-hour SUM06 level of 20 ppm-hours. The lower end of this range (7 ppm-hours) is the same as the lower end of the range identified in the 1997 Consensus Workshop as protective of tree seedlings in natural forest stands from growth effects (Heck and Cowling, 1997).

The Administrator, taking Staff Paper and CASAC views into account, proposes 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-hours. This range encompasses the range of levels recommended by CASAC, and also includes a higher level as recommended in the Staff Paper. Given the uncertainty in determining the risk attributable to various levels of exposure to O_3, the Administrator believes as a public welfare policy judgment that this is a reasonable range to propose.

In taking into account the uncertainty associated with the above, the Administrator has also considered an alternative approach to establishing a secondary standard(s). This alternative approach would establish a cumulative, seasonal standard(s) that would afford differing degrees of protection for O_3-related impacts on different types of vegetation with different intended uses.

The Administrator recognizes that known O_3-sensitive plant species growing within the U.S experience a variety of O_3-induced effects, including visible foliar injury, biomass loss and yield loss, and that the public welfare significance of each of these effects can vary significantly, depending on the nature of the effect, the intended use of the plant, and/or the type of environment or location in which the plant grows. Any given O_3-related effect on vegetation (*e.g.*, biomass loss, or foliar injury) may be judged to have a different degree of impact on public welfare depending, for example, on whether that effect occurs in a Class I area, commercial cropland, or a city park. This variation in the significance of O_3-related vegetation effects from a public welfare perspective across type of effect, intended plant use, and area grown means that the level of ambient O_3 that is requisite to protect the public welfare may also vary. The level of ambient O_3 that is requisite in a federally designated Class I area may be lower than the level that is requisite in a cropland area. EPA is therefore considering and soliciting comment on an alternative approach for the secondary O_3 standard, with the aim of reasonably reflecting these variations.

Specifically, the Administrator seeks comment on an alternative approach that would establish a suite of secondary standards. The suite of standards would contain different ambient levels, with each standard at a level that is requisite to protect public welfare for that variation in plant effect, use, and/or location. For example, a secondary standard intended to provide protection to natural systems valued for their aesthetic beauty and/or important ecological functions they might serve could be set at a lower, more protective level to provide the requisite degree of protection against a broad array of O_3-related effects on important sensitive species in such areas. In contrast, while negative impacts on yield production in sensitive agricultural crops is also an important public welfare effect, O_3-related reductions in yield may be considered less significant or adverse to the public welfare, depending on the degree of impact, since the intended use of such land is to produce optimum yields and croplands are already heavily managed to achieve that goal. Thus, a secondary standard set to provide the requisite degree of crop protection for such an area could be set at a higher level.

The Administrator recognizes that variation in vegetation type and location, intended use, and impacts related to O_3 exposure can be diverse, and believes that it is appropriate to consider whether it is appropriate and feasible to establish a suite of standards that accounts more broadly for such variation. EPA recognizes that this approach is unique with regard to secondary standards and will pose unique challenges, including how to classify areas according to intended use. Some geographic areas have already been identified for specific uses, such as Federal Class I areas,[65] which are intended to conserve unimpaired natural ecosystems and their associated species for the enjoyment of future generations. Likewise, the USDA has classified cultivated areas in the U.S. into certain categories of intended use (such as cropland, rangeland, timberland) that could help inform the setting of a suite of standards.

EPA is taking comment on all aspects of this alternative approach, including whether it is appropriate to set a suite of secondary standards that varies depending on use, location, and type of effect on vegetation. EPA invites comment on the appropriateness of this approach, from the scientific, legal, and policy perspectives, and on other factors that should be considered in determining the applicability of any one level within a suite of standards.

[65] The Clean Air Act defines Class I areas as national parks over 6,000 acres, national wilderness areas and national memorial parks over 5,000 acres, and international parks. The National Park Service was created in 1916 by Congress through the National Park Service Organic Act in order to ''conserve the scenery and the natural and historic objects and the wild life therein and to provide for the enjoyment of the same in such manner and by such means as will leave them unimpaired for the enjoyment of future generations.''

3. 8-Hour Average Standard

The Administrator is also proposing to revise the current secondary standard by making it identical to the proposed 8-hour primary standard, which is proposed to be within the range of 0.070 to 0.075 ppm. For this option, EPA also solicits comment on a wider range of 8-hour standard levels, including levels down to 0.060 ppm and up to the current standard (*i.e.*, effectively 0.084 ppm with the current rounding convention).

In the last review, the 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 O_3 concentrations averaged over a 3-year period would reduce O_3 exposures measured by a seasonal SUM06 index'' (62 FR 38876).

On the basis of that history, the current 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 recent air quality. Based on the results, the 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 O_3 concentrations within the annual and/or 3-year average period.

Thus, though the 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 urges 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 is due to the concern that the analysis in the 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 O_3 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 Staff Paper concluded that it remains 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 O_3 on human health and the effects of O_3 exposures on welfare is that vegetation effects are more dependent on the cumulative exposure to, and uptake of, O_3 over the course of the entire growing season (Henderson, 2006c). The CASAC O_3 Panel members were unanimous in concluding the protection of natural terrestrial ecosystems and managed agricultural crops requires a secondary O_3 standard that is substantially different from the primary O_3 standard in averaging time, level, and form (Henderson, 2007).

A number of public commenters also presented views for the Administrator's consideration regarding the adequacy of the current standard and whether or not revisions to that standard were warranted. These commenters did not support adopting an alternative, cumulative form for the secondary standard. These commenters stated that ''though directionally a cumulative form of the standard may better match the underlying data,'' they believed further work is needed to determine whether a cumulative exposure index for the form of the secondary standard is necessary. These commenters identified a number of key concerns regarding the available evidence that, in their view, make it inappropriate to revise the secondary standard at this time. In particular they assert that (1) The key uncertainties, cited by the Administrator in the 1997 review as reasons for deciding it was not appropriate to move forward with a seasonal secondary, have not been materially reduced in the current review; and (2) the exposure assessment is inaccurate and too uncertain due to the use of low estimates of PRB, an arbitrary rollback method that is uninformed by atmospheric chemistry from photochemical models, and the use of the CMAQ model in the west, whose biases and uncertainties are insufficiently characterized and evaluated.

In considering the appropriateness of proposing a revised secondary standard that would be identical to the proposed primary standard, the Administrator took into account the approach used by the Agency in the last review, the conclusions of the Staff Paper, CASAC advice, and the views of public commenters. The Administrator first considered the 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 O_3 concentrations within the annual and/or 3-year average period. On this basis, the Administrator recognizes 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 Administrator also recognizes that lack of rural monitoring data makes 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 is clear, the number and size of areas at issue and the degree of risk is hard to determine. However, such a standard would also tend to avoid the potential for providing more protection than is necessary, a risk that 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 Administrator also considered the views and recommendations of

CASAC, and agrees 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, the Administrator also recognizes that there remain significant uncertainties in determining or quantifying the degree of risk attributable to varying levels of O_3 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, the Administrator also believes it is appropriate to consider the degree of protection that would be afforded by a secondary standard that is identical to the proposed primary standard. Based on his consideration of the full range of views as described above, the Administrator proposes as a second option to revise the secondary standard to be identical in every way to the proposed primary standard.

F. Proposed Decision on the Secondary Standard

The Administrator proposes to replace the current secondary standard with one of two options. One option is a new cumulative, seasonal standard expressed as an index of the annual sum of weighted hourly concentrations (using the W126 form), set at a level in the range of 7 to 21 ppm-hours. The index would be cumulated over the 12-hour daylight period (8 a.m. to 8 p.m.) during the consecutive 3-month period within the O_3 season with the maximum index value. The other option is to revise the current secondary standard by making it identical to the proposed 8-hour primary standard, which is proposed to be within the range of 0.070 to 0.075 ppm. For this option, EPA also solicits comment on a wider range of 8-hour standard levels, including levels down to 0.060 ppm and up to the current standard (*i.e.*, effectively 0.084 ppm with the current rounding convention. The Administrator is also soliciting comment on an alternative approach for a setting cumulative, seasonal standard(s) that would afford differing degrees of protection for O_3-related impacts on different types of vegetation with different intended uses.

V. Creation of Appendix P—Interpretation of the NAAQS for Ozone

The EPA is proposing to create Appendix P to 40 CFR part 50 to reflect the proposed revisions to the primary and secondary standards discussed above. This Appendix would explain the computations necessary for determining when the proposed primary and secondary standards are met. More specifically, Appendix P addresses data completeness requirements, data reporting, handling, and rounding conventions, and example calculations. Although EPA is proposing two alternative secondary standards, the proposed Appendix has been written to address a seasonal secondary standard expressed in the W126 form. If EPA adopts a secondary standard identical to the primary standard, Appendix P will be modified accordingly. The proposed Appendix also reflects the final rule promulgated on March 22, 2007 for the treatment of data influenced by exceptional events (72 FR 13560).

Key elements of the proposed revisions to Appendix P are outlined below.

A. Data Completeness

The data completeness requirements in Appendix P proposed here for the proposed 8-hr primary standard secondary standards are the same as those in Appendix I to 40 CFR part 50 required for the current standard. To satisfy the date completeness requirement, Appendix P would require 90% data completeness, on average, for the 3-year period at a monitoring site, with no single year within the period having less than 75% data completeness. This data completeness requirement would have to be satisfied in order to determine that the standard(s) have been met at a monitoring site. A site could be found not to have met the standard(s) with less than complete data. EPA concluded in adopting these same data completeness requirements in Appendix I in 1997 that these proposed requirements are reasonable based on its earlier analysis of available air quality data that showed that 90% of all monitoring sites that are operated on a continuous basis routinely meet this objective. The EPA is seeking comment, however, on whether meteorological data would provide an objective basis for determining, on a day for which there is missing data, that the meteorological conditions were not conducive to high O_3 concentrations, and therefore, that the day could be assumed to have an O_3 concentration less than 0.070 to 0.075 ppm.

We are proposing separate data completeness requirements for the proposed seasonal secondary standard expressed in the W126 form. For such a standard, Appendix P would require a site to have 75% data completeness in a given month. Appendix P would also provide a mechanism for adjusting for missing data. Because this alternative is a seasonal cumulative index, representing a distribution of O_3 values under a range of meteorological conditions, rather than a peak statistic, the EPA is proposing a missing data procedure that would require the monthly total index to be adjusted for incomplete data by multiplying the unadjusted W126 value by the ratio of the number of possible daylight hours (8:00 a.m. to 8:00 p.m.) to the number of hours with valid ambient hourly concentrations. This adjustment is analogous to calculating an estimated number of exceedances contained within part 50 Appendix I for the one hour O_3 standard.

B. Data Handling and Rounding Conventions

Almost all State agencies now report hourly O_3 concentrations to three decimal places, in ppm, since the typical incremental sensitivity of currently used O_3 monitors is 0.001 ppm. Consistent with the current approach for computing 8-hr averages, in calculating 8-hr average O_3 concentrations from such hourly data, any calculated digits past the third decimal place would be truncated to preserve the number of significant digits in the reported data. In calculating 3-year averages of the fourth highest maximum 8-hr average concentrations, EPA is proposing to require the result to be reported to the third decimal place with digits to the right of the third decimal place truncated to preserve the number of significant digits in the reported data, as prescribed by the current standard. Analyses discussed in the Staff Paper demonstrated that taking into account the precision and bias in 1-hour O_3 measurements, the 8-hour design value had an uncertainty of approximately 0.001 ppm. Thus, EPA considers any value less than 0.001 ppm to be highly uncertain and, therefore, proposes truncating both the individual 8-hour averages used to determine the annual fourth maximum as well as the 3-year average of the fourth maxima to the third decimal place. Nevertheless, EPA solicits comment on the appropriateness of rounding to the third decimal place as well as the policy reasons behind either truncating or rounding the 3-year average to the third decimal place (with 0.0005 and greater rounding up). EPA is also seeking comment on the scientific validity of truncating the three year average as opposed to rounding it as well as the policy reasons behind either truncating or rounding the average to the third decimal place.

To determine whether the proposed standard is met, the calculated value of the fourth highest maximum 8-hour average concentrations, averaged over three years, would be compared to the level of the standard. As discussed in section II, the EPA is proposing to issue an 8-hr standard extending to three decimal places, based on the staff's analysis and conclusions discussed in the Staff paper that expressing the proposed standard to the third decimal place is consistent with the precision requirements of the current O_3 monitoring technology. Given that both the proposed standard and the calculated value of the 3-year average of the fourth highest maximum 8-hr O_3 concentration are expressed to three decimal places, the two values can be compared directly. This is different than the approach for determining compliance with the current standard O_3 standard. In comparing the calculated 3-year average (which is expressed to three decimal places) to the current standard O_3 standard (which is expressed to only two decimal places), Appendix I requires the calculated 3-year average to be rounded to two decimal places. This additional step would not be necessary for the proposed standard given that the standard and the 3-year average are each expressed to three decimal places.

For the proposed seasonal secondary standard, the annual maximum 3-month W126 value computed on a calendar year basis using the three highest, consecutive monthly W126 values would be used as the summary statistic. The resulting value would then be compared to the level of the secondary O_3 standard. The Agency is also interested in receiving comments regarding a 3-year average form summary statistic.

VI. Ambient Monitoring Related to Proposed Revised O_3 Standards

The EPA is not proposing any specific changes to existing requirements for monitoring of O_3 in the ambient air. However, we invite comment on a number of issues which naturally arise in connection with the proposed revision of the O_3 NAAQS. The EPA may propose changes to some of the existing requirements at a later date.

Current requirements regarding EPA-approved measurement methods for ambient O_3 are stated in 40 CFR part 50 Appendix D, Measurement Principle and Calibration Procedure for the Measurement of Ozone in the Atmosphere, and in 40 CFR part 53, Ambient Air Monitoring Reference and Equivalent Methods. The EPA does not intend to propose any changes to these requirements, because we believe these requirements would continue to be appropriate to support implementation of a revised O_3 NAAQS.

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 O_3 monitors based on the population of each of their Metropolitan Statistical Areas (MSA) and the most recently measured O_3 levels in each area. Each State (or in some cases portions of a State) also has a required O_3 monitoring season based on historical experience on when O_3 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) Certain deviations including minimum monitoring requirements and/or monitoring season requirements may be approved by the EPA Regional Administrator on a case-by-case basis.

Required O_3 monitoring seasons range from four to 12 months. The minimum number of monitors in an MSA ranges from zero (for an area with population under 350,000 and no recent history of an O_3 design value greater than 85 percent of the NAAQS) to four (for an area with population greater than 10 million and an O_3 design value greater than 85 percent of the NAAQS). Because these requirements apply at the MSA level, large urban areas consisting of multiple MSAs can require more than four monitors. For example, the New York-Newark-Bristol NY-NJ-CT-PA combined statistical area requires about 14 monitors. In total, about 400 monitors are required in MSAs, but about 1100 are actually operating in MSAs because most States operate more than the minimum required number of monitors.

There are no EPA requirements for O_3 monitoring in less populated areas outside of MSA boundaries (*e.g.*, Metropolitan Statistical Areas) or in rural areas. However, there are about 250 O_3 monitors in counties that are not part of MSAs. Some required State monitors are placed downwind of the urban center of the MSA of interest in locations that are in some cases in a county outside the MSA itself; some States also operate a few rural monitors for research purposes. The EPA operates a network of about 56 O_3 monitors as part of its Clean Air Status and Trends Network (CASTNET). The National Park Service (NPS) operates about 27 monitors at other CASTNET sites. The NPS also has O_3 monitoring stations in parks that are not part of the CASTNET dry deposition monitoring effort including multiple O_3 stations in Great Smoky Mountains, Sequoia, Yosemite, and Joshua Tree National Parks.

Required quality assurance procedures for O_3 monitoring are given in 40 CFR Part 58 Appendix A, Quality Assurance Requirements for State and local air monitoring stations (SLAMS), special purpose monitors (SPM), and prevention of significant deterioration (PSD) Air Monitoring. The EPA does not intend to propose any changes to these quality assurance requirements, because we believe that the current measurement uncertainty goals and related procedures for assessing precision and bias as documented in paragraph 2.3.1.2 of Appendix A are appropriate to support the implementation of a revised O_3 NAAQS.

States are required to report O_3 data quarterly to EPA's Air Quality System (AQS), and most also voluntarily report their pre-validated O_3 data on an hourly basis to EPA's real time AirNow data system, where the data are used to forecast O_3 concentrations and to provide public advisories. The National Park Service and many other organizations also report their O_3 data to AQS and/or AirNow. The locations of currently operating O_3 monitors which report data to EPA's Air Quality System are available through the EPA AirData Web site *http://www.epa.gov/air/data/index.html.*

Data from O_3 monitors at CASTNET stations are currently kept in a separate national data base.[66]

The EPA invites comments on O_3 monitoring issues (other than O_3 monitoring methods and quality assurance requirements), including the following:

(1) Ozone monitoring network requirements in urban areas. Table D–2 of 40 CFR Part 58 Appendix D is based on the percentage of the O_3 NAAQS, with a break point at 85 percent of the NAAQS. Therefore, a revision of the NAAQS would automatically increase the required number of O_3 monitors. For example, assuming a final NAAQS of

[66] At present, not all ozone monitors at CASTNET sites are operated in full compliance with the quality assurance requirements of 40 CFR Part 58 Appendix D, as they have not been primarily intended for regulatory use. The EPA is working towards such compliance in the near future and towards making CASTNET ozone data available through AQS.

0.070 ppm for purposes of illustration only, about 70 MSAs with current O_3 design values in the range of about 0.060 ppm (about 85 percent of the current NAAQS) to 0.070 ppm (about 85 percent of 0.070 ppm) would be affected, with most changing from no required monitors to one, or from one required monitor to two. Because most of these areas already are operating at least as many monitors as the possible new requirement, the number of monitors which would need to be initiated (or moved from a location of excess monitors) would be only about five monitors. About 100 MSAs with populations less than 350,000 presently are without any O_3 monitors, and hence they do not have an O_3 design value for use with Table D–2. If for the purpose of applying Table D–2, these areas are treated as if they have O_3 concentrations below 85 percent of the revised NAAQS, then a NAAQS revision would not automatically result in a requirement for O_3 monitoring in these MSAs.[67] EPA invites comments on the appropriateness of the existing minimum monitoring requirements for purposes of implementing the proposed revised NAAQS, including the automatic changes to minimum monitoring requirements that would be triggered by a NAAQS revision.

(2) Ozone monitoring seasons. As mentioned, the currently required O_3 monitoring seasons range from four to 12 months of the year. In some cases, O_3 monitoring may start a couple of weeks before and may end a couple of weeks after the required season. With a lower O_3 NAAQS, the issue arises of whether in some areas the required O_3 monitoring season should be made longer. The EPA notes that under the existing regulations, the Regional Administrator may approve State-requested deviations from the established O_3 monitoring season but EPA may not increase the length of the season for an area at EPA's own initiative other than by notice and comment rulemaking.

(3) Monitoring to support implementation of a secondary O_3 NAAQS. It is fair to say that the existing O_3 monitoring requirements and current State monitoring practices are primarily oriented towards protecting against health effects in people, *i.e.*, towards implementation of the primary NAAQS. This accounts for the focus on urban areas, which can combine large populations, large emissions of O_3-forming precursors, and O_3 concentrations of concern. The purpose of the secondary NAAQS is to protect against vegetation damage and other welfare effects, which can occur in both urban and rural areas. States have largely been given discretion on whether to add additional monitors aimed specifically at achieving the objectives of the previous and current secondary NAAQS. In urban areas, EPA in general believes that an O_3 monitoring network (and monitoring season) appropriate to support implementation of the primary NAAQS will also be appropriate for implementing the secondary NAAQS. However, rural areas are presently only sparsely monitored for O_3 so violations of the secondary NAAQS in areas with sensitive vegetation may occur undetected, as a result of transport from urban areas with high precursor emissions and/or O_3 concentrations or from formation of additional O_3 from precursors emitted from sources outside urban areas. It is conceivable that rural violations of a secondary NAAQS could occur in areas with sensitive vegetation even though urban monitoring networks are showing compliance with the primary NAAQS, whether the forms and levels of the two standards are the same or different. The EPA invites comment on the likelihood of this occurring under the possible combinations of primary and secondary standards proposed in this notice, and on whether, where, and how EPA should require monitoring in rural areas specifically aimed at implementation of the secondary NAAQS (and/or promote more voluntary monitoring or conduct monitoring itself in rural areas).

[67] EPA might instead treat one or more of these counties as having a design value based on a monitor in a nearby monitored county, in which case ozone monitoring might become required in certain currently unmonitored MSAs and the number of new required monitors would increase in the illustrative NAAQS example stated above.

VII. 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 O_3 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 prepared this regulatory impact analysis (RIA) of the potential costs and benefits associated with this action. The RIA estimates the costs and monetized human health and welfare benefits of attaining three alternative O_3 NAAQS nationwide. Specifically, the RIA examines the alternatives of 0.075 ppm, 0.070 ppm, and 0.065 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 O_3 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 final 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 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 O_3 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 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 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 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 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 E (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 O_3 NAAQS. The Tribal Authority Rule gives Tribes the opportunity to develop and implement

CAA programs such as the O_3 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 this 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 this 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 & 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 O_3; 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 O_3 exposure. Because children are considered a potentially susceptible population, we have carefully evaluated the environmental health effects of exposure to O_3 pollution among children. These effects and the size of the population affected are summarized in section 8.7 of the Criteria Document and section 3.6 of the Staff Paper, and the results of our evaluation of the effects of O_3 pollution on children are discussed in sections II.A–C of this preamble.

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 O_3. 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 No. 104–113, § 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 O_3 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. (2006) 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. December 2006. Available electronically on the internet at: *http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.*

Abt Associates, Inc. (2007) 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. Available electronically on the internet at: *http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html. EPA–HQ–OAR–2005–0172–0176.*

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.

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.

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.

Alscher, R. G.; Amundson, R. G.; Cumming, J. R.; Fellows, S.; Fincher, J.; Rubin, G.; van Leuken, P.; Weinstein, L. H. (1989) Seasonal changes in the pigments, carbohydrates and growth of red spruce as affected by ozone. New Phytol. 113: 211–223.

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 health effect of air pollution? Am. J. Respir. Crit. Care Med. 161: 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, Atmospheric Environment 37: 1185–1198.

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 CO_2 or O_3. 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.

Bayram, H.; Rusznak, C.; Khair, O. A.; Sapsford, R. J.; Abdelaziz, M. M. (2002) Effect of ozone and nitrogen dioxide on the permeability of bronchial epithelial cell cultures of nonasthmatic and asthmatic subjects. Clin. Exp. Allergy 32: 1285–1292.

Beedlow P.A., Tingey D.T., Phillips D.L., Hogsett W.E. & Olszyk D.M. (2004) Rising atmospheric CO_2 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 U.S. 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: *http://dx.doi.org/* [23 January, 2006].

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.

Borja-Aburto, V. H.; Loomis, D. P.; Bangdiwala, S. I.; Shy, C. M.; Rascon-Pacheco, R. A. (1997) Ozone, suspended particulates, and daily mortality in Mexico City. Am. J. Epidemiol. 145: 258–268.

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.

Bremner, S. A.; Anderson, H. R.; Atkinson, R. W.; McMichael, A. J.; Strachan, D. P.; Bland, J. M.; Bower, J. S. (1999) Short term associations between outdoor air pollution and mortality in London 1992–4. Occup. Environ. Med. 56: 237–244.

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.

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., B. H. Honkala, 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, 877 p.

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.; Samuelson, L. J. (1998) Ambient ozone effects on forest trees of the eastern United States: a review. New Phytol. 139: 91–108.

Chappelka, A. H. (2002) Reproductive development of blackberry (Rubus cuneifolius) as influenced by ozone. New Phytol. 155: 249–255.

Chen, L.; Jennison, B. L.; Yang, W.; Omaye, S. T. (2000) Elementary school

absenteeism and air pollution. Inhalation Toxicol. 12: 997–1016.
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.
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.
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.
Dann, M. S.; Pell, E. J. (1989) Decline of activity and quantity of ribulose bisphosphatecarboxylase/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 CO_2 and O_3 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.; Yu, S. (2005) A performance evaluation of the 2004 release of Models–3 CMAQ, Atmos. Environ. 40: 4811–4824.
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; report nos. EPA/600/AP–93/004aF–cF. 3v. Available from: NTIS, Springfield, VA; PB96–185582, PB96–185590, and PB96–185608. Available: *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. EPA/452/R–96–007. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Available electronically on the internet at: *http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_pr_sp.html.*
Environmental Protection Agency. Technical Assistance Document for the Calibration of Ambient Ozone Monitors, EPA publication number EPA–600/4–79–057, EPA, National Exposure Research Laboratory, Department E, Research Triangle Park, NC 27711.
Environmental Protection Agency. Transfer Standards for Calibration of Ambient Air Monitoring Analyzers for Ozone, EPA publication number EPA–600/4–79–056, EPA, National Exposure Research Laboratory, Department E, Research Triangle Park, NC 27711.
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-RTP Report no. NCEA–R–1068.
Environmental Protection Agency (2003a) Ozone Injury to Forest Trees. In: EPA's Draft Report on the Environment Technical Document. EPA–600–R–03–050. U.S. EPA, ORD, Washington, DC, page 5–19.
Environmental Protection Agency (2003b) Clean Air Status and Trends Network (CASTNet) 2001 Quality Assurance Report; Research Triangle Park, NC: Office of Air Quality Planning and Standards. Report from EPA Contract No. 68–D–98–112.
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, 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, 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. First draft OAQPS staff paper. Research Triangle Park, NC: Office of Air Quality Planning and Standards. 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, 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. Second draft OAQPS staff paper. Research Triangle Park, NC: Office of Air Quality Planning and Standards. Available online at: *http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html.*
Environmental Protection Agency (2006c) Ozone Population Exposure Analysis for Selected Urban Areas. Office of Air Quality Planning and Standards, Research Triangle Park, NC. July 2006.

Available electronically on the internet at: *http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.*

Environmental Protection Agency (2006d) Ozone Population Exposure Analysis for Selected Urban Areas. Office of Air Quality Planning and Standards, Research Triangle Park, NC. December 2006. Available electronically on the internet at: *http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.*

Environmental Protection Agency (2007) Review of the 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–07–003. Available online at: *http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html.*

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.

Fiscus, E. L.; Reid, C. D.; Miller, J. E.; Heagle, A. S. (1997) Elevated CO_2 reduces O_3 flux and O_3-induced yield losses in soybeans: possible implications for elevated CO_2 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.

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.

Gong, H., Jr.; Wong, R.; Sarma, R. J.; Linn, W. S.; Sullivan, E. D.; Shamoo, 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.

Gouveia, N.; Fletcher, T. (2000) Time series analysis of air pollution and mortality: effects by cause, age and socioeconomic status. J. Epidemiol. Community Health 54: 750–755.

Graham, D. E.; Koren, H. S. (1990) Biomarkers of inflammation in ozone-exposed humans: comparison of the nasal and bronchoalveolar lavage. Am. Rev. Respir. Dis. 142: 152–156.

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., C.G. Jones and T.E. Dawson (2003) ''Urbanization effects on tree growth in the vicinity of New York City.'' Nature 424: 183–187.

Grulke, 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.

Grulke, N. E.; Balduman, L. (1999) Deciduous conifers: high N deposition and O_3 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., Wullschleger, S., Tabberer, T. and 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.

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 SO_2 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).

Hogsett, W. E., Weber, J. E., Tingey, D., Herstrom, A., Lee, E. H., Laurence, J. A. (1997) ''Environmental auditing: an approach for characterizing tropospheric ozone risk to forests.'' *Environ. Manage.* 21: 105–120.

Holton, M. K.; Lindroth, R. L.; Nordheim, E. V. (2003) Foliar quality influences tree-herbivore-parisitoid interactions: effects of elevated CO_2, O_3, 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.

Höppe, P.; Peters, A.; Rabe, G.; Praml, G.; Lindner, J.; Jakobi, G.; Fruhmann, G.; Nowak, D. (2003) Environmental ozone effects in different population subgroups. Int. J. Hyg. Environ. Health 206: 505–516.

Horst, R.; Duff, M. (1995) Concentration data transformation and the quadratic rollback methodology (Round 2, Revised). Unpublished memorandum to R. Rodrguez, U.S. EPA, June 8, 1995. EPA Docket No. A–95–58 Item II–I–199.

Horstman, D. H.; Folinsbee, 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.

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 Begleitsubstanzen 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.; Koschyk, 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 O_3 on trembling aspen and interaction with CO_2: Results from an O_3-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 O_3 moderates responses of temperate hardwood forests to elevated CO_2: 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.

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 O_3 compromises net primary production in young stands of trembling aspen, paper birch and sugar maple in response to elevated atmospheric CO_2. New Phytologist. 168: 623–636.

Kinney, P. L.; Thurston, G. D.; Raizenne, M. (1996) The effects of ambient ozone on lung function in children: a reanalysis of six summer camp studies. Environ. Health Perspect. 104: 170–174.

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.

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.

Lamb, C.; Dixon, R. A. (1997) The oxidative burst in plant disease resistance. Ann. Rev. Plant Physiol. Mol. Biol. 48: 251–275.

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.

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–I–265.

Levy, J. I.; Chemerynski, S. M.; Sarnat, J. A. (2005) Ozone exposure and mortality, an empiric Bayes metaregression analysis. Epidemiology 16: 458–468.

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.

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.

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.

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.

Moldau, H.; Söber, J.; Söber, A. (1990) Differential sensitivity of stomata and mesophyll tosudden 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.

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.

Neufeld, H. S.; Lee, E. H.; Renfro, J. R.; Hacker, W. D. (2000) Seedling insensitivity to ozone for three conifer species native to Great Smoky Mountains National Park. Environ. Pollut. 108: 141–151.

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 CO_2 and/or O_3. Plant Cell Environ. 24: 327–336.

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.*

NRC (2004) Air quality management—United States. I. National Research Council (U.S.). Committee on Air Quality Management in the United States. TD883.2.A64325 2004 363.739′25′0973—dc222004014594 *http://www.nap.edu/openbook/0309089328/html/.*

Odum, E. P. (1963) Ecology. New York, NY: Holt, Rinehart and Winston. (Modern biology series).

Ollinger, S. V., Aber, J. D., Reich, P. B. and Freuder, R. J. (2002) Interactive effects of nitrogen deposition, tropospheric ozone, elevated CO_2 and land use history on the carbon dynamics of northern hardwood forests. Glob. Change Biol. 8(6), 545–562.

Olszyk, D., Bytnerowlez, 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.

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.

Peel, J. L.; Tolbert, P. E.; Klein, M.; Metzger, K. B.; Flanders, W. D.; Knox, T.; Mulholland, J. A.; Ryan, P. B.; Frumkin, H. (2005) Ambient air pollution and respiratory emergency department visits. Epidemiology 16: 164–174.

Pell, E. J.; Schlagnhaufer, 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 CO_2 and O_3. Nature (London) 420: 403–407.

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.

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 I. C., Farquhar G. D., Fasham M. J. R., *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. Noguer, P. J. van der Linder, 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 [CO_2] and/or ozone on limitations to CO_2 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.

Reiling, K.; Davison, A. W. (1992c) Spatial variation in ozone resistance of British populations of Plantago major. L. New Phytol. 122: 699–708.

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. Memo to the Ozone NAAQS Review Docket. EPA–HQ–OAR–0172–0005.

Rizzo, M. (2006) A distributional comparison between different rollback methodologies applied to ambient ozone concentrations. May 31, 2006. Memo to the Ozone NAAQS Review Docket. EPA–HQ–OAR–0172–0027.

Romieu, I.; Meneses, F.; Ruiz, S.; Sienra, 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.; Sienra, 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.; Sienra-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.

Samet, J. M.; Zeger, S. L.; Dominici, F.; Curriero, F.; Coursac, I.; Dockery, D. W.; Schwartz, J.; Zanobetti, A. (2000) The national morbidity, mortality, and air pollution study. Part II: morbidity, mortality, and air pollution in the United States. Cambridge, MA: Health Effects Institute; research report no. 94, part II.

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.

Sartor, F.; Snacken, R.; Demuth, C.; Walckiers, D. (1995) Temperature, ambient ozone levels, and mortality during summer, 1994, in Belgium. Environ. Res. 70: 105–113.

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.

Schelegle, E. S.; Miller, L. A.; Gershwin, L. J.; Fanucchi, M. V.; Van Winkle, L. S.; Gerriets, J. E.; Walby, W. F.; Mitchell, V.; Tarkington, B. K.; Wong, V. J.; Baker, G. L.; Pantle, L. M.; Joad, J. P.; Pinkerton, K. E.; Wu, R.; Evans, M. J.; Hyde, D. M.; Plopper, C. G. (2003) Repeated episodes of ozone inhalation amplifies the effects of allergen sensitization and inhalation on airway immune and structural development in Rhesus monkeys. Toxicol. Appl. Pharmacol. 191: 74–85.

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.

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.

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.

Simpson, R. W.; Williams, G.; Petroeschevsky, A.; Morgan, G.; Rutherford, S. (1997) Associations between outdoor air pollution and daily mortality in Brisbane, Australia. Arch. Environ. Health 52: 442–454.

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.

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.

Sunyer, J.; Basagaña, X.; Belmonte, J.; Antó, J. M. (2002) Effect of nitrogen dioxide and ozone on the risk of dying in patients with severe asthma. Thorax 57: 687–693.

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 long-term 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 CO_2 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.
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.
Weber, J. A.; Clark, C. S.; Hogsett, W. E. (1993) Analysis of the relationship(s) among O_3 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.
Wiley, J. A.; Robinson, J. P.; Piazza, T.; Garrett, K.; Cirksena, K.; Cheng, Y.-T.; Martin, G. (1991a) Activity patterns of California residents. Final report. Sacramento, CA: California Air Resources Board; report no. ARB/R93/487. Available from: NTIS, Springfield, VA.; PB94–108719.
Wiley, J. A.; Robinson, J. P.; Cheng, Y.-T.; Piazza, T.; Stork, L.; Pladsen, K. (1991b) Study of children's activity patterns: final report. Sacramento, CA: California Air Resources Board; report no. ARB–R–93/489.
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.
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: http://www.epa.gov/sab/pdf/epec02009.pdf [9 December, 2003].
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.
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 Part 50

Environmental protection, Air pollution control, Carbon monoxide, Lead, Nitrogen dioxide, Ozone, Particulate matter, Sulfur oxides.

Dated: June 20, 2007.

Stephen L. Johnson,

Administrator.

For the reasons stated in the preamble, title 40, chapter I of the code of Federal regulations is 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 added 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 O_3 is (0.070–0.075) 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.

(b) The 8-hour primary O_3 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 O_3 concentration is less than or equal to (0.070–0.075) ppm, as determined in accordance with appendix P to this part.

(c) The level of the national secondary ambient air quality standard for O_3 is a cumulative index value of (7–21) ppm-hours, measured by a reference method based on Appendix D to this part and designated in accordance with part 53 of this chapter.

(d) The secondary O_3 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 O_3 monitoring season with the maximum index value. The secondary O_3 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 (7–21) ppm-hours, as

determined in accordance with appendix P to this part.

3. Appendix P is added 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 national 8-hour primary and secondary ambient air quality standards for O_3 specified in § 50.14 are met at an ambient O_3 air quality monitoring site. Ozone is measured in the ambient air by a Federal reference method (FRM) based on appendix D of this part, as applicable, and designated in accordance with part 53 of this chapter, or by a Federal equivalent method (FEM) designated in accordance with part 53 of this chapter, or by an Approved Regional Method (ARM) designated in accordance with part 58 of this chapter. Data reporting, data handling, and computation procedures to be used in making comparisons between reported O_3 concentrations and the level of the O_3 standard are specified in the following sections. Whether to exclude, retain, or make adjustments to the data affected by exceptional events, including stratospheric O_3 intrusion and other natural events, is subject to the requirements under § 50.1, § 50.14 and § 51.930.

(b) The terms used in this appendix are defined as follows:

8-hour average is the rolling average of hourly O_3 concentrations as explained in section 2 of this appendix.

Annual fourth highest daily maximum refers to the fourth highest value measured at a monitoring location during the O_3 season for a particular year.

Daily maximum 8-hour average concentration refers to the maximum calculated 8 hour average for a particular day as explained in section 2 of this appendix.

Design values are the metrics (*i.e.*, statistics) that are compared to the NAAQS levels to determine compliance, calculated as shown in sections 3 and 4 of this appendix.

Ozone monitoring season refers to the span of time within a calendar year when individual States are required to measure ambient O_3 concentrations as listed in part 58 appendix D to this chapter.

W126 is the weighted hourly O_3 concentrations based on seasonal measurements as explained in section 4 of this appendix.

Year refers to calendar year.

2. Primary Ambient Air Quality Standard for Ozone

2.1 Data Reporting and Handling Conventions

Computing 8-hour averages. Hourly average concentrations shall be reported in parts per million (ppm) to the third decimal place, with additional digits to the right being truncated. Running 8-hour averages shall be computed from the hourly O_3 concentration data for each hour of the year and the result shall be stored in the first, or start, hour of the 8-hour period. An 8-hour average shall be considered valid if at least 75% of the hourly averages for the 8-hour period are available. In the event that only 6 (or 7) hourly averages are available, the 8-hour average shall be computed on the basis of the hours available using 6 (or 7) as the divisor (8-hour periods with three or more missing hours shall not be ignored if, after substituting one-half the minimum detectable limit for the missing hourly concentrations, the 8-hour average concentration is greater than the level of the standard). The computed 8-hour average O_3 concentrations shall be reported to three decimal places (the insignificant digits to the right of the third decimal place are truncated, consistent with the data handling procedures for the reported data).

Daily maximum 8-hour average concentrations. (a) There are 24 possible running 8-hour average O_3 concentrations for each calendar day during the O_3 monitoring season. The daily maximum 8-hour concentration for a given calendar day is the highest of the 24 possible 8-hour average concentrations computed for that day. This process is repeated, yielding a daily maximum 8-hour average O_3 concentration for each calendar day with ambient O_3 monitoring data. Because the 8-hour averages are recorded in the start hour, 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.

(b) An O_3 monitoring day shall be counted as a valid day if valid 8-hour averages are available for at least 75% of possible hours in the day (*i.e.*, at least 18 of the 24 averages). In the event that less than 75% of the 8-hour averages are available, a day shall also be counted as a valid day if the daily maximum 8-hour average concentration for that day is greater than the level of the ambient standard.

2.2 Primary Standard-Related Summary Statistic

The standard-related summary statistic is the annual fourth-highest daily maximum 8-hour O_3 concentration, expressed in parts per million, averaged over three years. The 3-year average shall be computed using the three most recent, consecutive calendar years of monitoring data meeting the data completeness requirements described in this appendix. The computed 3-year average of the annual fourth-highest daily maximum 8-hour average O_3 concentrations shall be reported to three decimal places (the insignificant digits to the right of the third decimal place are truncated, consistent with the data handling procedures for the reported data).

2.3 Comparisons With the Primary Ozone Standard

(a) The primary O_3 ambient air quality standard is met at an ambient air quality monitoring site when the 3-year average of the annual fourth-highest daily maximum 8-hour average O_3 concentration is less than or equal to [0.070 to 0.075] ppm.

(b) This comparison shall be based on three consecutive, complete calendar years of air quality monitoring data. This requirement is met for the three year period at a monitoring site if daily maximum 8-hour average concentrations are available for at least 90%, on average, of the days during the designated O_3 monitoring season, with a minimum data completeness in any one year of at least 75% of the designated sampling days. When computing whether the minimum data completeness requirements have been met, meteorological or ambient data may be sufficient to demonstrate that meteorological conditions on missing days were not conducive to concentrations above the level of the standard. Missing days assumed less than the level of the standard are counted for the purpose of meeting the data completeness requirement, subject to the approval of the appropriate Regional Administrator.

(c) Years with concentrations greater than the level of the standard shall not be ignored on the ground that they have less than complete data. Thus, in computing the 3-year average fourth maximum concentration, calendar years with less than 75% data completeness shall be included in the computation if the average annual fourth maximum 8-hour concentration is greater than the level of the standard.

(d) Comparisons with the primary O_3 standard is demonstrated by examples 1 and 2 in paragraphs (d)(1) and (d)(2) respectively as follows:

EXAMPLE 1.—AMBIENT MONITORING SITE ATTAINING THE PRIMARY O_3 STANDARD

Year	Percent valid days (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)
2004	100	0.092	0.090	0.085	0.079	0.078
2005	96	0.084	0.083	0.075	0.072	0.070
2006	98	0.080	0.079	0.073	0.061	0.060

EXAMPLE 1.—AMBIENT MONITORING SITE ATTAINING THE PRIMARY O_3 STANDARD

Year	Percent valid days (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)
Average	98				0.070	

(1) As shown in example 1, the primary standard is met at this monitoring site because the 3-year average of the annual fourth-highest daily maximum 8-hour average O_3 concentrations (*i.e.*, 0.0707 ppm, truncated to 0.070 ppm) is less than or equal to [0.070 to 0.75] ppm. The data completeness requirement is also met because the average percent of days with valid ambient monitoring data is greater than 90%, and no single year has less than 75% data completeness. In Example 1, the individual 8-hour averages used to determine the annual fourth maximum are truncated to the third decimal place.

EXAMPLE 2.—AMBIENT MONITORING SITE FAILING TO MEET THE PRIMARY O_3 STANDARD

Year	Percent valid days (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)
2004	96	0.105	0.103	0.103	0.102	0.102
2005	74	0.104	0.103	0.092	0.091	0.088
2006	98	0.103	0.101	0.101	0.095	0.094
Average	89				0.096	

As shown in example 2, the primary standard is not met at this monitoring site because the 3-year average of the fourth-highest daily maximum 8-hour average O_3 concentrations (*i.e.*, 0.0960 ppm, truncated to 0.096 ppm) is greater than [0.070 to 0.075] ppm. Note that the O_3 concentration data for 2005 is used in these computations, even though the data capture is less than 75%, because the average fourth-highest daily maximum 8-hour average concentration is greater than [0.070 to 0.075] ppm. In Example 2, the individual 8-hour averages used to determine the annual fourth maximum are truncated to the third decimal place.

3. Design Values for Primary Ambient Air Quality Standards for Ozone

The air quality design value at a monitoring site is defined as that concentration that when reduced to the level of the standard ensures that the site meets the standard. For a concentration-based standard, the air quality design value is simply the standard-related test statistic. Thus, for the primary standard, the 3-year average annual fourth-highest daily maximum 8-hour average O_3 concentration is also the air quality design value for the site.

4. Secondary Ambient Air Quality Standard for Ozone

4.1 Data Reporting and Handling Conventions

Computing the daily index value (D.I.). The secondary O_3 standard is a seasonal standard expressed as the sum of weighted hourly concentrations, cumulated over the 12 hour daylight period, 8 a.m. to 8 p.m. local standard time (LST), during the maximum consecutive 3-month period within the O_3 monitoring season. Hourly average concentrations for each hour from 8 a.m. to 8 p.m. LST shall be reported in parts per million (ppm) to the third decimal place, with additional digits to the right being truncated. The first step in computing the daily index value, D.I., for the daylight hours is to apply a sigmoidal weighting function in the form of Equation 1 in this appendix:

Equation 1

$$O_3 * \left(\frac{1}{1 + \left(4403 * e^{-126 * O_3} \right)} \right)$$

to each measurement of hourly average concentration, where O_3 is the average hourly O_3 concentration expressed in ppm. The computed value of the sigmoidally weighted hourly concentration shall be expressed to three decimal places (the remaining digits to the right are truncated). An illustration of computing a daily index value is below:

EXAMPLE 3.—DAILY INDEX VALUE CALCULATION FOR AN AMBIENT O_3 MONITORING SITE

Start hour	Concentration (ppm)	Weighted concentration (ppm)
8:00 AM	0.045	0.002
9:00 AM	0.060	0.018
10:00 AM	0.075	0.055
11:00 AM	0.080	0.067
12:00 PM	0.079	0.065
1:00 PM	0.082	0.071
2:00 PM	0.085	0.077
3:00 PM	0.088	0.082
4:00 PM	0.083	0.073
5:00 PM	0.081	0.069
6:00 PM	0.065	0.029
7:00 PM	0.056	0.011

Daily index value (D.I.) = 0.002 + 0.018 + 0.055 + 0.067 + 0.065 + 0.071 + 0.077 + 0.082 + 0.073 + 0.069 + 0.029 + 0.011 = 0.619 ppm-hours

Computing the monthly cumulative index (W126). The daily index value is computed at each monitoring site for each calendar day in each month during the O_3 monitoring. At an individual monitoring site, a month is counted as a valid O_3 monitoring month if hourly average O_3 concentrations are available for at least 75% of the possible index hours in the month. For months with less than 75% data completeness, the monthly cumulative index value shall be adjusted for incomplete sampling by multiplying the unadjusted W126 cumulative index value by the ratio of the number of possible daylight hours to the number of hours with valid ambient hourly concentrations using Equation 2 in this appendix:

Equation 2

$$M.I. = \sum_{j=1}^{n} (D.I.) * (n * 12) / v$$

Where,

M.I. = the monthly sum of the weighted daylight hours,

D.I. = the daily sum of the weighted daylight hours,

n = the number of days in the calendar month,

v = the number of daylight hours (8:00 a.m.—8:00 p.m. LST) with valid hourly O_3 concentrations.

4.2 Secondary Standard-related Summary Statistic

The standard-related summary statistic is the annual maximum consecutive 3-month W126 value expressed in ppm-hours. Specifically, the annual W126 value is computed on a calendar year basis using the three highest, consecutive monthly W126 values.

4.3 Comparisons with the Secondary Ozone Standard

The secondary ambient O_3 air quality standard is met when the annual maximum W126 value based on a consecutive 3-month period at an O_3 air quality monitoring site is less than or equal to [7 to 21] ppm-hours. The number of significant figures in the level of the standard dictates the rounding convention for comparing the computed W126 value with the level of the standard. The first decimal place of the computed W126 value is rounded, with values equal to or greater than of 0.5 rounding up.

EXAMPLE 4.—CALCULATION OF THE MAXIMUM 3-MONTH W126 VALUE AT AN AMBIENT AIR QUALITY MONITORING SITE FAILING TO MEET THE SECONDARY O_3 STANDARD

	April	May	June	July	August	September	October
Monthly W126	4.442	9.124	12.983	16.153	13.555	4.364	1.302
3-Month Total	na	na	26.549	38.260	42.691	34.072	19.221

As shown in example 4, the maximum consecutive 3-month W126 value for this site is 43 ppm-hours. Because 43 ppm-hours is greater than [7 to 21] ppm-hours, the secondary standard is not met at this ambient air quality monitoring site.

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