Responses to Significant Comments on the 2007 Proposed Rule on the National Ambient Air Quality Standards for Ozone

(July 11, 2007; 72 FR 37818)

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List of Acronyms

The following acronyms have been used for the sake of brevity in this document:

Act	Clean Air Act
AAM	Alliance of Automobile Manufacturers
AAP	American Academy of Pediatrics
ACC	American Chemistry Council
ACCP	American College of Chest Physicians
AHA	American Heart Association
ALA	American Lung Association
AMA	American Medical Association
ANA	American Nurses Association
APHA	American Public Health Association
API	American Petroleum Institute
AQI	Air Quality Index
AQS	Air Quality System
ARMs	Approved Regional Methods
ATS	American Thoracic Society
CAA	Clean Air Act
CARB	California Air Resources Board
CASAC	Clean Air Scientific Advisory Committee
CASTNET	Clean Air Status and Trends Network
CDC	Centers for Disease Control
CFR	Code of Federal Regulations
CHPAC	Children's Health Protection Advisory Committee
CI	Confidence interval
CO	Carbon monoxide
COPD	Chronic obstructive pulmonary disease
EPA	Environmental Protection Agency
FEM	Federal Equivalent Method
FEV_1	Forced Expiratory Volume for 1 second
FRM	Federal Reference Method
GAM	Generalized additive model
GLM	Generalized linear model
HEI	Health Effects Institute
HML	Highest measured level
IQG	Information Quality Guidelines
LML	Lowest measured level
MSA	Metropolitan Statistical Area
NACAA	National Association of Clean Air Agencies
NACCHO	National Association of County and City Health Officials
NAAQS	National ambient air quality standards
NAM	National Association of Manufacturers
NESCAUM	Northeast States Coordinated Air Use Management
NMMAPS	National Morbidity, Mortality, and Air Pollution Study

NO_2	Nitrogen Dioxide
NPS	National Park Service
NRC	National Research Council
NRDC	Natural Resources Defense Council
NTAA	National Tribal Air Association
O_3	Ozone
OAP	Office of Atmospheric Programs
OR	Odds ratio
OTC	Ozone Transport Commission
PAMS	Photochemical Assessment Monitoring Stations
PEFR	Peak Expiratory Flow Rate
ppm	Parts per million
ppb	Parts per billion
PM	Particulate matter
$PM_{2.5}$	Particles generally less than or equal to 2.5 μm in diameter
PM_{10}	Particles generally less than or equal to 10 micrometers (μm) in diameter
PRB	Policy relevant background
QA	Quality assurance
RIA	Regulatory Impact Analysis
RR	Relative risk
SAB	Science Advisory Board
SO_2	Sulfur Dioxide
TSD	Technical support document
UARG	Utility Air Regulatory Group
USDA	U.S. Department of Agriculture
US PIRG	U.S. Public Interest Research Group
WHO	World Health Organization

Frequently Cited Documents

The following documents are frequently cited throughout EPA's response to comments, often by means of the short names listed below:

Criteria Document: Environmental Protection Agency (2006) 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.

Preamble to the final rule: Preamble to the Final Rule on the Review of the National Ambient Air Quality Standards for Ozone; to be published in the *Federal Register* in March 2008.

Proposal notice: National Ambient Air Quality Standards for Ozone: Proposed Rule. 72 FR 37818, July 11, 2007.

Staff Paper: Environmental Protection Agency (2007a) Review of the national ambient air quality standards for ozone: assessment of scientific and technical information. OAQPS staff paper. (Final) July 2007. Research Triangle Park, NC: Office of Air Quality Planning and Standards; EPA report no. EPA-452/R-07-003. Available online at: http://epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_sp.html.

Health Risk TSD: Abt Associates Inc. (2007a) Ozone Health Risk Assessment for Selected Urban Areas. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. July 2007. Available electronically on the internet at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.

Human Exposure TSD: Environmental Protection Agency (2007b) Ozone Population Exposure Analysis for Selected Urban Areas. Office of Air Quality Planning and Standards, Research Triangle Park, NC. (Final) July 2007. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.

Vegetation Risk TSD: Abt Associates Inc. (2007b) Technical Report on Ozone Exposure, Risk, and Impacts Assessments for Vegetation: Final Report. Prepared for Office of Air Quality Planning and Standards, U.S. Environmental Protection Agency, Research Triangle Park, NC. January 2007. Available online at: http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_cr_td.html.

Responses to Significant Comments on the 2007 Proposed Rule on the National Ambient Air Quality Standards for Ozone

I. INTRODUCTION

This document, together with the preamble to the final rule on the review of the national ambient air quality standards (NAAQS) for ozone (O_3), presents the responses of the Environmental Protection Agency (EPA) to some of the thousands of public comments received on the 2007 O_3 NAAQS proposal notice (72 FR 37818). All significant issues raised in the public comments have been addressed.

Due to the large number of comments that addressed similar issues, as well as the sheer volume of the comments received, this response-to-comments document does not generally cross-reference each response to the commenter(s) who raised the particular issue involved, although commenters are identified in some cases where they provided particularly detailed comments that were used to frame the overall response on an issue.

The responses presented in this document are intended to augment the responses to comments that appear in the preamble to the final rule or to address comments not discussed in the preamble to the final rule. Although portions of the preamble to the final rule are paraphrased in this document where useful to add clarity to responses, the preamble itself remains the definitive statement of the rationale for the revisions to the standards adopted in the final rule.

In many instances, particular responses presented in this document include cross references to responses on related issues that are located either in the preamble to the O_3 NAAQS final rule, or in this Response to Comments document. All issues on which the Administrator is taking final action in the O_3 NAAQS final rule are addressed in the O_3 NAAQS rulemaking record. Issues on which the Administrator is taking final action in the monitoring final rule are addressed in that rulemaking record.

Accordingly, this Response to Comments document, together with the preamble to the O_3 NAAQS final rule and the information contained in the Criteria Document (EPA, 2006) and the Staff Paper (EPA, 2007), should be considered collectively as EPA's response to all of the significant comments submitted on EPA's 2007 O_3 NAAQS proposed rule. This document incorporates directly or by reference the significant public comments addressed in the preamble to the O_3 NAAQS final rule as well as other significant public comments that were submitted on the proposed rule.

Various commenters have referred to and discussed a number of "new" scientific studies on the health and welfare effects of O_3 that had been published recently and therefore were not

included in the Criteria Document.[1] EPA has provisionally considered any significant "new" studies, including those submitted during the public comment period. The purpose of this effort was to ensure that the Administrator was fully aware of the "new" science before making a final decision on whether to revise the current O_3 NAAQS. EPA provisionally considered these studies to place their results in the context of the findings of the Criteria Document. EPA's provisional consideration of "new" studies is included as an Appendix to this document and is referred to throughout this document with regard to specific comments related to "new" studies. EPA concludes that, taken in context, the "new" information and findings do not materially change any of the broad scientific conclusions regarding the health effects of O_3 exposure made in the Criteria Document.

Consistent with the final decisions presented in the notice of final rulemaking, comments on the primary O_3 standard are addressed in section II.A. Comments on the secondary O_3 standard are addressed below in section II.B. Comments on data handling procedures are addressed in II.C. Comments on monitoring related issues are addressed in section II.D. Section III includes responses to legal, administrative, procedural, or misplaced comments.

II. RESPONSES TO SIGNIFICANT COMMENTS ON PROPOSED O_3 STANDARDS

A. Primary O_3 Standards

1. General Comments on Proposed Primary O_3 standard

General comments based on relevant factors that either support or oppose any change to the current O_3 primary standard are addressed in this section. The responses to these comments are also discussed in section II.B.2 of the preamble to the final rule. Specific comments on the proposed primary standard, including comments on the indicator, averaging time, form and level are addressed in sections II.C in the preamble to the final rule and discussed more fully below in section II.A.2. Specific comments about the health effects evidence and the results of the human exposure and health risk assessments are addressed in sections II.B.2 in the preamble to the final rule and discussed in sections II.A.3 through II.A.5 below. Incorporating responses contained in sections II.B of the preamble to the final rule, EPA provides the following responses to general comments related to the need to revise the O_3 standard.

a. Support for Revising the Current Standard

[1] For ease of reference, these studies will be referred to as "new" studies or "new" science, using quotation marks around the word *new*. Referring to studies that were published too recently to have been included in the 2004 Criteria Document as "new" studies is intended to clearly differentiate such studies from those that have been published since the last review and are included in the 2004 Criteria Document (these studies are sometimes referred to as new (without quotation marks) or more recent studies, to indicate that they were not included in the 1996 Criteria Document and thus are newly available in this review.

Many public comments received on the proposal asserted that, based on the available scientific information, the current O_3 standard is insufficient to protect public health with an adequate margin of safety and revisions to the standard are appropriate. Among those calling for revisions to the current standards are medical groups, including the American Thoracic Society (and other health organizations, including American Medical Association (AMA), American College of Chest Physicians (ACCP), American College of Preventive Medicine, American Association of Cardiovascular and Pulmonary Rehabilitation, American College of Occupational and Environmental Medicine, National Association for the Medical Direction of Respiratory Care) (ATS et al.), and the American Academy of Pediatrics (AAP), as well as medical doctors and academic researchers. Similar conclusions were also submitted in comments from many national, state, and local public health organizations, including, for example, the American Lung Association (and other environmental organizations, including Environmental Defense, Sierra Club, Earthjustice) (ALA et al.), the American Heart Association (and other health organizations, including, National Association of County and City Health Officials (NACCHO), American Lung Association, American Nurses Association (ANA), American Public Health Association (APHA), Physicians for Social Responsibility) (AHA et al.), and the American Nurses Association (ANA), as well as in letters to the Administrator from EPA's Children's Health Protection Advisory Committee (CHPAC) (Marty, 2007a, 2007b). Environmental groups also commented in support of revising the standard, including the Sierra Club, Environmental Defense, the Natural Resources Defense Council (NRDC), Earthjustice, and the US Public Interest Research Group (US PIRG). All of these medical, public health and environmental commenters stated that the current O_3 standard needs to be revised and that an even more protective standard than proposed by EPA is needed to protect the health of sensitive population groups. Many individual commenters also expressed such views.

The majority of State and local air pollution control authorities who commented on the O_3 standard supported revision of the current O_3 standard, as did the National Tribal Air Association (NTAA). Environmental agencies that supported revising the standard include agencies from: Arkansas; California; Delaware; Iowa; Illinois; Michigan; North Carolina; New Mexico; New York; Oklahoma; Oregon; Pennsylvania; Utah; Wisconsin; and Washington, DC. State organizations, including the National Association of Clean Air Agencies (NACAA), Northeast States for Coordinated Air Use Management (NESCAUM), and the Ozone Transport Commission (OTC) urged that EPA revise the O_3 standard. All of these commenters supported revisions to the current standard, with most supporting a standard consistent with CASAC's recommendations.

> *Comment:* Many public comments received on the proposal asserted that the current O_3 standard is insufficient to protect public health, especially the health of sensitive groups, with an adequate margin of safety and revisions to the standard are appropriate. For example, the ATS stated:
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> > We believe that the Administrator has correctly stated that, beyond any degree of scientific uncertainty, convincing and compelling evidence has demonstrated that exposure to ozone at levels below the current standard is responsible for measurable and significant adverse health effects, both in terms of morbidity and mortality. ..... The known respiratory, cardiac and perinatal effects of ozone

> pollution are each in their own right major public health issues. In combination they provide immediate, actionable information and require a meaningful public health policy response from the EPA. (ATS et al., pp. 1, 11)

All of these commenters supported revisions to the current standard, with most supporting a standard consistent with CASAC's recommendations. In general, the commenters noted above primarily based their views on the body of evidence assessed in the Criteria Document as articulated in sections II.A and II.C of the preamble to proposal, finding it to be stronger and more compelling than in the last review. Some specifically agreed with the weight of evidence approach taken by the Criteria Document. These commenters generally placed much weight on CASAC's interpretation of the body of available evidence and the results of EPA's exposure and risk assessments, both of which formed the basis for CASAC's recommendation to revise the O_3 standard to provide increased public health protection.

These commenters also noted that in recent years, a broad scientific consensus has emerged that EPA's current air quality standards for ozone are not sufficient to protect public health, and that the levels and form must be greatly tightened. This consensus is evidenced by the strong unanimous comments of the CASAC, which was backed by the endorsement of over 100 leading independent air quality scientists, EPA's Children's Health Protection Advisory Committee, and many others. In the face of this strong scientific consensus, one commenter stated that it is untenable to cite "uncertainty" as a rationale for failing to propose tighter standards. [ALA et al., p. 15]

Medical and public health commenters also expressed the view that EPA must not use uncertainty in the scientific evidence as justification for retaining the current O_3 standard. For example, the ATS stated:

> We note with concern that throughout the standard setting process, senior EPA officials have take a very conservative approach to reviewing the scientific literature on the health effects of ozone. We find the science on the health effects of ozone to be robust and compelling. (ATS et al., p. 11)

Response: EPA generally agrees with these commenters' conclusion regarding the need to revise the O_3 primary standard. The scientific evidence noted by these commenters was generally the same as that assessed in the Criteria Document and the Staff Paper, and EPA agrees that this evidence provides a basis for concluding that the current O_3 standard is not sufficient to protect public health with an adequate margin of safety. However, for reasons discussed in section II.B of the preamble to the final rule and in sections II.A.2 and II.A.3 below, EPA disagrees with aspects of these commenters' views on the level of protection that is appropriate and supported by the available scientific information. The Administrator's overall conclusions about the need for revisions to the primary O_3 standard, which include consideration of these general comments, are presented in the response to comment (1) in section II.A.1.b below.

b. Support for Retaining the Current Standard

Another group of commenters representing industry associations and businesses opposed revising the current primary O_3 standard. These views were extensively presented in comments from the Utility Air Regulatory Group (UARG), representing a group of electric generating companies and organizations and several national trade associations, and in comments from other industry and business associations including, for example: Exxon Mobil Corporation; the Alliance of Automobile Manufacturers (AAM); the National Association of Manufacturers (NAM), and the American Petroleum Institute (API). The API sponsored a workshop at the University of Rochester in June 2007 to review the scientific information and health risk assessment considered by EPA during the review of the O_3 NAAQS. Although the report (hereafter, "Rochester Report") from this workshop does not offer judgments on the specific elements of the current or proposed standard, it has been cited in a number of public comments that opposed revision of the current 8-hour standard. The Annapolis Center for Science-Based Public Policy issued a report (hereafter, "Annapolis Center") on the science and health effects of O_3, which explicitly opposed revising the current O_3 primary standard. Several State environmental agencies also opposed revising the current O_3 primary standard, including agencies from: Georgia; Indiana; Kentucky; Louisiana; Nevada; and Texas.

As discussed more fully below in sections dealing with specific comments, these and other commenters in this group generally mentioned many of the same studies that were cited by the commenters noted above who supported revising the standard, as well as other studies, but highlighted different aspects of these studies in reaching substantially different conclusions about their strength and the extent to which progress has been made in reducing uncertainties in the evidence since the last review.

> *Comment:* These commenters generally expressed the view that the current standard provides the requisite degree of public health protection. In so doing, they considered whether the evidence that has become available since the last review has established a more certain risk or a risk of effects that are significantly different in character to those that provided a basis for the current standard, or whether the evidence demonstrates that the risk to public health upon attainment of the current standard would be greater than was understood when EPA established the current standard in 1997.
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> In supporting their view that the present primary O_3 standard continues to provide the requisite public health protection and should not be revised, UARG and others generally stated that:
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> - the effects of concern have not changed significantly since 1997
> - the uncertainties in the underlying health science are as great or greater than in 1997
> - the estimated risk upon attainment of the current O_3 standard has decreased since 1997
> - "new" studies not included in the Criteria Document continue to increase uncertainty about possible health risks associated with exposure to O_3.

Response: As noted in the response to comment (1) in section II.A.1.a above, EPA, believes, contrary to the view fo the commenters, that the available information provides a basis for concluding that the current O3 standard does not protect public health with an adequate margin of safety. EPA has responded to the specific points noted above as well as additional specific issues raised by those contending that the current 8-hour primary standard should not be revised in sections II.A.3 through II.A.5 below and in the Appendix to this document.

More generally, the rationale for EPA's position regarding the need for revision of the current primary O_3 standard is necessarily based upon consideration of all the comments, including the specific comments that are presented in sections II.A.3 through II.A.5. The Administrator's overall conclusions about the need for revisions to the primary O_3 standard, which include consideration of these general comments, are presented below.

Having carefully considered all of the public comments related to the need for revision of the current O_3 primary standard, the Administrator believes the fundamental scientific conclusions on the effects of O_3 reached in the Criteria Document and Staff Paper, briefly summarized below in section II.A.2 and discussed more fully in section II.A of the proposal, remain valid. In considering whether the primary O_3 standard should be revised, the Administrator places primary consideration on the body of scientific evidence available in this review on the health effects associated with O_3 exposure, as summarized in section II.B.1 of the preamble to the final rule,. The Administrator notes that there is much new evidence that has become available since the last review, including an especially large number of new epidemiological studies. The Administrator believes that this body of scientific evidence is very robust, recognizing that it includes large numbers of various types of studies, including toxicological studies, controlled human exposure studies, field panel studies, and community epidemiological studies, that provide consistent and coherent evidence of an array of O_3-related respiratory morbidity effects and possibly cardiovascular-related morbidity as well as total nonaccidental and cardiorespiratory mortality. The Administrator observes that (1) the 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; (2) newly available 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; (3) newly available 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; and (4) 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. The Administrator believes that this very robust body of evidence, taken together, enhances our understanding of O_3-related effects relative to what was known at the time of the last review. Further, he believes that the available evidence provides increased 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 warm O_3 season contribute to premature mortality.

Further, 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. This is especially true in at-risk populations that include people with asthma or other lung diseases, who are likely to experience more serious effects from exposure to O_3, 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. The Administrator notes that this important new evidence demonstrates 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, the Administrator notes that (1) 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; (2) 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; (3) 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; and (4) 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 available evidence, the Administrator agrees with the CASAC Panel and the majority of public commenters that the current standard is not requisite to protect public health with an adequate margin of safety because it is does not provide sufficient protection and that revision of the current O_3 standard is needed to provide increased public health protection. The Administrator notes that extensive critical review of this body of evidence and related uncertainties during the criteria and standard review process, including review by the CASAC Panel and the public of the basis for EPA's proposed decision to revise the primary O_3 standard, has identified a number of issues about which different reviewers disagree and for which additional research is warranted. Nonetheless, on balance, the Administrator believes that the remaining uncertainties in the available evidence do not diminish confidence in the causal relationships between O_3 exposures and indicators of serious respiratory morbidity effects, or the highly suggestive evidence of associations between O_3 exposures and premature mortality, nor do they diminish confidence in the conclusion that the associations extend below the level of the current standard.

Beyond a primary consideration of the available evidence, the Administrator has also taken into consideration the Agency's exposure and risk assessments to help inform his evaluation of the adequacy of the current standard. As at the time of proposal, the Administrator believes the results of those assessments inform his judgment on the adequacy of the current standard to protect against health effects of concern. In considering the exposure analysis results at this time, the Administrator recognizes that there is a risk of confusion in the term "exposure of concern" that was used at the time of proposal, as it could be read to imply a determination that a certain benchmark level of exposure has been shown to be causally associated with adverse health effects. As a consequence, the Administrator believes that it is more appropriate to consider such exposure estimates in the context of a continuum rather than focusing on any one discrete benchmark level, as was done at the time of proposal, since the Administrator does not believe that the underlying scientific evidence is certain enough to support a focus on any bright-line benchmark level. In so doing, the Administrator recognizes that associations between O_3 exposures and health effects of concern become increasingly uncertain at lower O_3 exposure levels. Thus, the Administrator has taken into consideration the pattern of such exposure estimates across the range of discrete benchmark levels considered in EPA's exposure assessment to provide some indication of the potential magnitude of the incidence of health outcomes that could not be evaluated in the Agency's quantitative risk assessment but which have been demonstrated to occur in healthy people at O_3 exposures as low as 0.080 ppm, the lowest level at which such health outcomes have been tested.[2]

More specifically, the Administrator has considered the pattern of reductions in such exposures across the benchmark levels of 0.080, 0.070, and 0.060 ppm, which span the level at which there is strong evidence of effects in healthy people down to a level at which the Administrator judges the evidence of effects to be very limited. The Administrator observes that based on the aggregated exposure estimates for the 2002 simulation for the 12 urban areas included in the exposure analysis, upon just meeting the current standard, the percentages of asthmatic or all school age children likely to experience one of more exposures at and above these benchmark levels of 0.080, 0.070, and 0.060 ppm (while at moderate or greater exertion) are approximately 4%, 20%, and 45%, respectively. As noted at the time of proposal, the Administrator recognizes that there is substantial year-to-year and city-to-city variability in these estimates and that it is important to recognize this variability in considering these estimates. For example, for the 0.080, 0.070, and 0.060 ppm benchmark levels, these percentages are estimated to range from approximately 1 to 10%, 1 to 40%, and 7 to 65%, respectively, across each of

[2] As noted above, such health outcomes include increased airway responsiveness, increased pulmonary inflammation, increased cellular permeability, and decreased pulmonary defense mechanisms, which have been 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.

the 12 urban areas based on the 2002 simulation, and from approximately 0 to 1%, 0 to 7%, and 1 to 25%, respectively, based on the 2004 simulation.

With regard to the results of the risk assessment, the Administrator again considered the risks estimated to remain upon just meeting the current standard. The Administrator takes note of the estimated magnitudes of such risks, which are presented in section II.B.1.c of the preamble to the final rule for a range of health effects including moderate and large lung function decrements (including percentages of children and number of occurrences), respiratory symptom days, respiratory-related hospital admissions, and nonaccidental and cardiorespiratory mortality, as well as year-to-year and city-to-city variability, and the uncertainties in these estimates. Further, the Administrator recognizes that these estimated risks for the specific health effects that could be analyzed in the Agency's risk assessment 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.

In considering these quantitative exposure and risk estimates, as well as the broader array of O_3-related health endpoints that could not be quantified, the Administrator believes that they are important from a public health perspective and indicative of potential exposures and risks to at-risk groups. The Administrator thus finds that the exposure and risk estimates provide additional support to the evidence-based conclusion, reached above, that the current standard needs to be revised. Based on these considerations, and consistent with CASAC Panel's unanimous conclusion that there is no scientific justification for retaining the current standard, the Administrator concludes that the current primary O_3 standard is not sufficient and thus not requisite to protect public health with an adequate margin of safety, and that revision is needed to provide increased public health protection. It is important to note that this conclusion, and the reasoning on which it is based, do not address the question of what specific revisions are appropriate. That requires looking specifically at the current indicator, averaging time, form, and level of the O_3 standard, and evaluating the evidence relevant to determining whether and to what extent any of these elements should be revised. Comments on the proposed elements of the primary standard are addressed below in section II.A.2.

2. Specific Comments on Proposed Primary O_3 Standards

a. Averaging Time

The EPA received limited public comments on the issue of averaging time for the O_3 primary standard. In addition to the discussion contained in section II.C.2 of the preamble to the final rule, EPA provides the following response to a specific issue related to the averaging time for O_3.

Comment: A few comments specifically supported keeping only the 8-hour averaging time. However, there were other comments in support of setting a separate 1-hour

standard along with am 8-hour standard to better protect against peak exposures (California EPA, ALA et al.).

Response: EPA has conducted extensive air quality analyses to determine the extent to which meeting the 8-hour standard provides protection from 1-hour and longer-term ambient air concentrations of O_3. It was concluded from these analyses that adequate protection from both 1-hour and longer-term ambient O_3 concentrations would be provided in areas meeting the 8-hour primary standard.

b. Form of the Standard

The EPA received a limited number of public comments on the appropriate form for the O_3 standard. Incorporating responses contained in sections II.C.3 of the preamble to the final rule, EPA provides the following responses to specific comments related to the form of the 8-hour O_3 standard.

(1) *Comment:* 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)(e.g., ALA et al.). Most commenters who expressed the view that the current standard should not be revised did not provide any views on alternative forms that would be appropriate for consideration should the Administrator consider revisions to the standard. A few industry association and business commenters supported changing to a 5^{th} highest form (e.g., Dow Chemical, AAM). One commenter (Oklahoma Department of Transportation) suggested the use of a 6^{th} or 7^{th} highest daily maximum form.

Response: 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, recognizing that the 4^{th}-highest daily maximum, 8-hour average concentration form of the standard provides a stable target for implementing programs to improve air quality. 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. EPA also believes that the adequacy of the public health protection provided by the combination of the level and form is a foremost consideration. Based on this, EPA detemined that the form of the current standard, 4^{th}-highest daily maximum 8-hour average concentration, averaged over 3 years, should be retained, recognizing that the public health protection that would be provided by this standard is based on combining

this form with the increased health protection provided by the lower level of the standard discussed in the section below.

(2) *Comment:* While the CASAC Panel unanimously supported specifying the level of the standard to a three decimal places (i.e., ppb) degree of precision, public comments were mixed. Environmental organizations (e.g., ALA et al.) and some State/regional agencies (e.g., NESCAUM, PA Department of Environmental Protection) supported the proposed increased precision but did not support truncating to the third decimal. However, several industry associations (e.g., API, EMA, AAM) suggested that there is not sufficient evidence to modify the 1997 decision to round to two decimal places.

Response: EPA concludes that the level of the standard should be specified to the thousandth ppm (three decimal places), 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 the CASAC Panel's reasoning and recommendation with respect to this aspect of the standard. A discussion of the issue of truncating vs. rounding can be found in section II.C.1 of this Response to Comments document.

c. Level

A large number of comments on the proposed range of levels for the primary O_3 standard basically expressed one of two substantively different views: (1) support for a more health protective standard at or below the range of levels proposed by EPA or (2) opposition to any modification of the current O_3 standard. Many of these commenters simply expressed their views without stating any rationale, while others gave general reasons for their views but without reference to the factual evidence or rationale presented in the proposal notice as a basis for the Agency's proposed decision regarding the level of the primary O_3 standard.

With regard to the evaluation and consideration of the health effects evidence and how such information should be considered in the decision on the standard level, EPA notes that the commenters fell into the same two groups discussed in section II.B.2 of the preamble to the final rule. The two groups often cited the same studies and evidence, but they reached sharply divergent conclusions as to what standard level is supported by the health effects evidence. The general views of both groups on the interpretation and use of the health effects evidence are presented in section II.B.2.a of the preamble, with most comments from one group arguing that this evidence supports a decision to revise the 8-hour standard to 0.060 ppm or below, and the other group arguing that it supports a decision not to revise the current 8-hour standard.

A number of commenters, including many States and Tribes, who supported selecting a standard in the range 0.060 to 0.070 ppm generally placed great weight on the recommendation of CASAC. Section II.C.4 of the preamble to the final rule presents the Agency's response to these very general views. In addition to the discussion contained in that section, EPA provides the following responses to specific issues related to the level for the primary O_3 standard. The first section below addresses comments on health evidence considerations, and the next section

addresses comments on exposure and risk considerations related to consideration of the level for the primary standard. Additional specific comments on the exposure analysis and health risk assessment are included in sections II.A.4 and II.A.5 below.

i. Comments on Health Evidence Considerations

(1) *Comment:* With regard to the evidence from controlled human exposure studies, commenters that included public health and environmental groups who supported revising the current standard expressed the view that the large body of evidence available at the time of the last review, demonstrating an array of adverse health effects (i.e., reduced lung function, respiratory symptoms, increased airway responsiveness, inflammation, and increased susceptibility to respiratory infection), at concentrations of 0.080 ppm O_3, indicated that the standard should have been set at a lower level. These commenters noted that standards must be set below the level shown to cause effects in healthy subjects in order to protect sensitive populations with an adequate margin of safety. As discussed in section II.B.2.a above, these commenters focused on the results of the Adams studies (2002, 2006) as evidence that exposure to 0.060 ppm O_3 will result in a significant proportion (i.e., 7%) of the adult population who do not have asthma or other lung diseases experiencing notable lung function decrements (FEV_1 decrement ≥ 10%), and furthermore that larger decrements in FEV_1 would be expected in more susceptible populations. This evidence caused these commenters to reject EPA's proposed range:

> Clearly, EPA's proposed standard of 0.070 to 0.075 ppm cannot be considered protective of public health in light of experimental evidence demonstrating adverse respiratory effects in healthy individuals exposed to 0.060 ppm, and the legal requirements to protect sensitive populations with an adequate margin of safety. (ALA et al., p. 51)

The second group of commenters, who opposed revision of the standard, expressed the view that the group mean changes reported in the Adams studies (2002, 2006) were small, that such decrements should not be considered to be adverse, and that the individuals who experienced larger responses were too few to serve as a basis for a revised O_3 standard. This group included virtually all commenters representing industry associations and businesses. These general comments are also addressed in section II.B.2.a in the preamble to the final rule.

Response: In considering comments received on controlled human exposure studies, and how these studies support a focus on particular standard levels, the Administrator observes that in general the comments support his original view that these studies provide the most certain evidence of adverse health effects, and that the large bulk of evidence derives from studies of exposures at levels of 0.080 ppm and above. The Administrator notes that since the last review important new evidence includes demonstration of O_3-induced lung function effects and respiratory symptoms in some healthy adults down to the previously observed exposure level of 0.080 ppm, as well as very limited new evidence of the same effects at exposure levels well below the level of the current

standard (Adams, 2002, 2006). Based on careful consideration of the comments, the Administrator again concludes that while the Adams studies provide evidence that some healthy individuals will experience lung function decrements and respiratory symptoms at the 0.060 ppm exposure level, this evidence is too limited to support a primary focus at this level. Moreover, the Administrator notes that while the CASAC panel supported a level of 0.060 ppm, they also supported a level above 0.060, indicating that they disagree with the commenters view that the results of Adams studies mean that the level of the standard has to be set at 0.060 ppm.

(2) *Comment:* With regard to the information from epidemiological studies, commenters representing public health, environmental, and medical organizations generally asserted that the large body of new epidemiological studies provides evidence of causal associations between O_3 exposures and a wide array of respiratory and cardiovascular morbidity effects, including emergency department visits and hospital admissions. They expressed the view that a significant body of strong, consistent evidence links short-term exposures to premature mortality and noted that this evidence is supported by new research that provides biological plausibility for such effects. These commenters noted that various approaches, including air quality assessments which show that statistically significant associations occurred in areas that likely would have met the current standard, or statistical approaches that examined subsets of the data which indicate that statistically significant associations remain down to very low ambient O_3 levels, show effects well below the level of the current standard. Moreover they identified particular studies, including some "new" studies not considered in the Criteria Document, that indicated there are additional sub-populations that are likely to be sensitive to O_3, including infants, women, and African-Americans, that should be considered in deciding the requisite level of protection. They asserted that this information supports a standard set at a level no higher than 0.060 ppm O_3.

With regard to the information from epidemiological studies, the second group of commenters focused strongly on EPA's interpretation of the epidemiological evidence and the uncertainties they saw in this evidence as a basis for concluding that no change to the current level of the 8-hour O_3 standard is warranted. In commenting on the proposed range of levels, these commenters generally relied on the same arguments presented above in section II.A.1.b as to why they believed it would be inappropriate for EPA to make any revisions to the primary O_3 standard. That is, they asserted that the health effects of concern associated with short-term or prolonged exposures to O_3 have not changed significantly since 1997; that the inconsistencies and uncertainties inherent in these studies as a whole should preclude any reliance on them as justification for a more stringent standard; and that "new" science not included in the Criteria Document continues to increase uncertainty about possible health risks associated with exposure to O_3. Specific methodological issues cited as additional support for their conclusions included: adequacy of exposure data; potential confounding by copollutants; model selection; inconsistent evidence relating O_3 exposure to mortality, and "new" studies that provide additional evidence of inconsistencies. These general comments are also addressed in section II.B.2.a of the preamble to the final rule.

Response: In considering these comments on the epidemiological evidence with regard to the interpretation of the epidemiological evidence and methodological issues, the Administrator notes that in general, most of the issues and concerns raised by those who do not support any revisions to the primary O_3 standard with regard to the interpretation of the epidemiological evidence and methodological issues, are essentially restatements if issues raised during the review of the Criteria Document and Staff Paper. The same is true of the views of commenters who supported a level of the standard no higher than 0.060 ppm O_3. EPA presented and the CASAC Panel reviewed the interpretation of the epidemiological evidence in the Criteria Document and the integration of the evidence with policy considerations in the development of the policy options presented in the Staff Paper for consideration by the Administrator. CASAC reviewed the scientific content of both the Criteria Document and Staff Paper and advised the Administrator that these documents provided an appropriate basis for use in regulatory decision making. Therefore, these comments do not provide a basis for the Administrator to reach fundamentally different conclusions than he reached at the time of proposal.

Moreover, the Administrator notes that epidemiological evidence is most appropriately evaluated in the context of all available evidence, including evidence from controlled human exposure and toxicological studies. The Administrator agrees with the weight of evidence approach used in the Criteria Document and believes that this body of scientific evidence is very robust, recognizing that it includes a large number of various types of studies that provide consistent and coherent evidence of an array of O_3-related respiratory morbidity effects and possibly cardiovascular-related morbidity as well as total nonaccidental and cardiorespiratory mortality. Therefore, the Administrator judges that the body of epidemiological evidence indicating associations with a wide range of serious health effects, including respiratory emergency department visits and hospital admissions and premature mortality, at and below 0.080 ppm supports revising the current standard to protect public health. While the great majority of evidence concerning effects below 0.080 ppm was 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. Therefore, Administrator has considered 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. In that context, there is only quite limited evidence from controlled human exposure studies at exposure levels below 0.080 ppm O_3. The Administrator recognizes that in the body of epidemiological evidence, many studies reported positive and statistically significant associations, while others reported positive results that were not statistically significant, and a few did not report any positive O_3-related associations. In addition, the Administrator judged that evidence of a causal relationship between adverse health outcomes and O_3 exposures became increasingly uncertain at lower levels of exposure. Based on this the Administrator continues to believe that the body of epidemiological evidence does not support setting a standard as low as 0.060 as suggested by some commenters.

The Administrator also notes the 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 were below the level of the current standard. Further, there were 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 continued to report statistically significant associations with respiratory morbidity outcomes and mortality. In the context of the strong clinical evidence of adverse effect in healthy adults at 0.080, the Administrator finds that the body of epidemiological evidence does not support retaining a standard of 0.08, as suggested by commenters.

(3) Comment: Both groups of commenters also considered evidence from controlled human exposure and epidemiological studies of increased susceptibility in people with lung disease, especially people with asthma, but they reached sharply divergent conclusions about what standard level is supported by this evidence. As discussed above in section II.B.2.a, medical organizations and public health and environmental groups agreed with EPA that, based on evidence from controlled human exposure and epidemiological studies, people with asthma, especially children, are likely to have greater lung function decrements and respiratory symptoms in response to O_3 exposure than people who do not have asthma, and are likely to respond at lower levels. Furthermore, these commenters noted that epidemiological studies have identified other potentially sensitive subpopulations, including for example, infants, women and African-Americans, and that effects in these groups should be part of the consideration in providing an adequate margin of safety. These commenters concluded that the appropriate level for the primary O_3 standard is 0.060 ppm, to provide protection for members of sensitive groups, especially people with asthma, who are likely to have more serious responses and to respond at lower levels that healthy people. They also contend that a standard set at this level also would provide protection against anticipated, but as yet unproven effects in the additional groups cited.

Response: The Administrator agrees with these commenters that important new evidence shows that asthmatics have more serious responses, and are more likely to respond at lower O_3 levels, than healthy individuals. Moreover, he agrees that this evidence supports a standard set at a level below 0.080 ppm O_3, based on the strong evidence from human clinical studies in healthy adults at this level. However, for the reasons described in preamble to the final rule and in the response to comment number (2) above, he does not agree that the controlled human exposure and epidemiological evidence provide support for a standard set at 0.060 ppm.

(4) Comment: Industry association and business commenters asserted that EPA is wrong to claim that new evidence indicates that the current standard does not provide adequate health public health protection for people with asthma. In support of this position, these commenters made the following major comments: (1) the lung function decrements and respiratory symptoms observed in clinical studies of asthmatics are not clinically important; (2) EPA postulates that asthmatics would likely experience more serious responses and responses at lower levels than the subjects of controlled human exposure

experiments, but that hypothesis is not supported by scientific evidence; and, (3) EPA recognized asthmatics as a sensitive subpopulation in 1997, and new information does not suggest greater susceptibility than was previously believed.

Response: EPA has specifically responded to these comments in the response to comment (2) in section II.3.c below. After careful consideration of these comments and based on the response given below, the Administrator continues to judge 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 that include people with asthma or other lung diseases who are likely to experience more serious effects from exposure to O_3, as well as children and older adults with increased susceptibility, and 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.

ii. Comments on Exposure and Risk Considerations

(1) *Comment:* With regard to considering how the quantitative exposure and health risk assessments should factor into a decision on the standard level, EPA notes that the comments generally fell into two groups that reached sharply divergent conclusions as to what standard level is supported by these assessments. The general views of both groups on the implications of the exposure and risk assessment are presented in the preamble to the final rule (section II.B.2.b), with one group arguing that it supports a decision to revise the 8-hour standard to 0.060 ppm or below, and the other group arguing that it supports a decision not to revise the current 8-hour standard.

A joint set of comments from ALA and several environmental groups expressed the view that EPA cannot use exposures of concern to justify a standard in the range of 0.070 to 0.075 ppm. These commenters contend that standards in the proposed range would continue to expose too many asthmatic children, as well as other at risk groups such as outdoor workers and preschool children, to "demonstrably unhealthy levels of ozone pollution" in only 12 cities which does not represent a national estimate (ALA et al., p. 106). These same commenters asserted that if EPA were to consider exposures of concern, then the benchmark level must be defined as 0.060 ppm based on the considerable evidence of adverse health effects occurring at this level. They also cited various reasons why the exposure estimates were underestimated, including: only 12 cities were included in the assessment, various at risk groups including outdoor workers and preschool children were not included in the assessment, and EPA's exposure assessment underestimated exposures since it considers average children, not active children who spend more time outdoors and repeated exposures also were underestimated.

In contrast, industry association and business group commenters expressed the view that the concept of exposures of concern should not be considered as a basis for revising the level of the standard because it provided no indication of the probability that individuals would actually experience an adverse health effect. These same commenters also

provided various reasons why the exposure estimates were overestimated based on specific methodological choices made by EPA including, for example, O_3 measurements at fixed-site monitors can be higher than other locations where individuals are exposed, the exposure estimates do not account for O_3 avoidance behaviors, and the exposure model overestimates elevated breathing rates. Finally, these commenters also contend that the estimates of exposures of concern associated with just meeting the current standard, using the 0.080 ppm benchmark levels, have not appreciably changed since the prior review and, thus provide no support for revising the current standard.

Response: EPA has responded below to the criticisms from both groups of commenters related to concerns that the exposure estimates are either underestimated or overestimated in sections II.A.4 and II.A.5 of this document. EPA also has addressed the issues raised by both groups of commenters concerning the appropriateness of considering exposures at and above various benchmark levels as an element in the decision on the level of the standard in the preamble to the final rule (section II.C.4) and in the response to comment (2) below.

(2) *Comment:* Environmental and public health group comments expressed the view that if exposures of concern were considered, then the Administrator should focus only on the 0.060 ppm benchmark based on the contention that adverse health effects had been demonstrated down to this level. These commenters generally argued that significant exposure reductions could be achieved by setting the standard level at 0.060 ppm or lower. In contrast, other commenters, primarily industry and business groups focused on comparisons of the exposures of concern at the 0.080 ppm benchmark level based on their view that there was no convincing evidence demonstrating adverse health effects at levels below this benchmark.

Response: In view of the comments received related to the definition and use of the term "exposure of concern" at the time of proposal, the Administrator recognizes that that there is a risk of confusion, as it could be read to imply a determination that a certain benchmark level of exposure has been shown to be causally associated with adverse health effects. As a consequence, the Administrator believes that it is more appropriate to consider such exposure estimates in the context of a continuum rather than focusing on any one discrete benchmark level, as was done at the time of proposal, since the Administrator does not believe that the underlying scientific evidence is certain enough to support a focus on any single bright-line benchmark level. Thus, the Administrator believes it is appropriate to consider a range of benchmark levels from 0.080 down to 0.060 ppm, recognizing that concentrations at and above specified benchmark levels while at elevated exertion must be considered in the context of a continuum of the potential for health effects of concern, and their severity, with increasing uncertainty associated with the likelihood of such effects at lower O_3 exposure levels.

EPA recognizes that the 0.080 ppm benchmark level represents a level at which several health outcomes including lung inflammation, increased airway responsiveness, and decreased resistance to infection have been shown to occur in health adults. The Administrator places relatively great weight on the public health significance of

exposures exceeding this benchmark level given the greater certainty that these adverse health responses are likely to be observed in a significant fraction of the at-risk population. With respect to his decision on the level of the 8-hour standard, 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 asthmatic children likely to experience exposures at and above the 0.080 ppm benchmark level in aggregate (for the 12 cities in the assessment) is estimated to range from 0.1 to 0.4 percent of asthmatic school age children. Based on the 2004 simulation, the estimates are even lower, with no asthmatic children estimated to experience exposures at and above the 0.080 ppm benchmark level. Similar patterns are observed for all school age children. Recognizing the uncertainties inherent in the exposure assessment, the Administrator concludes that the exposure assessment suggests that exposures at and above the 0.080 ppm level, where several health effects have been shown to occur in healthy individuals, are eliminated or nearly eliminated depending on the modeling year.

However, the Administrator does not agree with those commenters who would only consider this single benchmark level. While the Administrator places less weight on exposures exceeding the 0.070 pm benchmark level, given the increased uncertainty about the fraction of the population and severity of the health responses that might occur associated with exposures above this level, he believes that it is appropriate to consider exposures at this benchmark as well in judging what level will protect public health with an adequate margin of safety. Consideration of the 0.070 ppm benchmark level recognizes that the effects observed at 0.080 ppm were in healthy adult subjects and sensitive population groups, such as asthmatics, are expected to respond at lower O_3 levels than healthy individuals. 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 asthmatic school age children likely to experience exposures at and above the 0.070 ppm benchmark level in aggregate (for the 12 cities in the assessment) is estimated to range from about 2 to 5 percent of asthmatic school age children. Based on the 2004 simulation, the estimates are substantially lower, with 0 to 0.6 percent of asthmatic children estimated to experience exposures at and above the 0.070 ppm benchmark level.

The Administrator considered but placed very little weight on exposures at and above the 0.060 ppm benchmark given the very limited scientific evidence supporting a conclusion that O_3 is causally related to various health outcomes at this exposure level.

Considering the uncertainties associated with the exposure assessment, the Administrator concludes that the exposure estimates associated with each of the benchmark levels are not appreciably different, between a 0.070 or 0.075 ppm standard, and therefore, the exposure assessment does not provide a clear enough basis for choosing a specific level within the proposed range.

(3) *Comment:* With regard to considering how the quantitative risk assessment should factor into a decision on the standard level, both groups of commenters generally considered the risk assessment in their comments on the standard level, but they reached sharply

divergent conclusions as to what standard level is supported by the risk assessment. More specifically, the environmental, public health, most medical organizations, and some State and regional air pollution agencies (e.g., California, NESCAUM) contend that EPA's proposed range of 0.070 to 0.075 ppm would result in significant residual public health risks. As articulated most fully in the joint set of comments from ALA and several environmental organizations, these commenters expressed the view that EPA's risk assessment clearly demonstrates that a more stringent 8-hour O_3 standard of 0.065 ppm, the most stringent standard analyzed by EPA, would significantly decrease O_3-related lung function decrements, respiratory symptoms, hospital admissions, and mortality and that "EPA must adopt a more stringent ozone standard of 0.060 ppm or below – a level that incorporates a more adequate margin of safety" (ALA et al., p. 108). These same commenters also cited various reasons for asserting that the risk assessment likely underestimates health risks to a substantial degree, including the limited nature of the assessment with respect to number of cities, populations covered, and health endpoints analyzed.

Response: EPA has responded to the comments concerning the scope of the risk assessment and assertion that health risks are likely underestimated both in the preamble to the final rule (section II.B.2.b) and in more detail in section II.A.5 of this document. While the Administrator places less weight on the results of the risk assessment, he notes that the results indicate that a standard set within the proposed range would likely reduce risks to at-risk groups from the O_3-related health effects considered in the 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. Moreover, he notes that the results of the assessment suggest a gradual reduction in risks with no clear breakpoint as increasingly lower standard levels are considered. However, in light of the important uncertainties inherent in the assessment discussed above and in the proposal, the Administrator concludes that the risk assessment does not provide a basis for choosing a level within the proposed 0.075 to 0.070 ppm range.

(4) *Comment:* Industry association and business group commenters who supported not revising the level of the current 8-hour standard generally asserted the following points: (1) that risk estimates have not changed significantly since the prior review in 1997; (2) that uncertainties and limitations underlying the risk assessments make them too speculative to be used in supporting a decision to revise the standard; (3) that EPA should have defined PRB differently and that EPA underestimated PRB levels which results in health risk reductions associated with more stringent standards being overestimated; and (4) that health risks are overestimated based on specific methodological choices made by EPA including, for example, selection of inappropriate effect estimates from health effect studies, EPA's approach to addressing the shape of exposure-response relationships, and whether or not to incorporate thresholds into its models for the various health effects analyzed.

Response: EPA has responded to the comments concerning the scope of the risk assessment and assertion that health risks are likely underestimated both in the preamble to the final rule (section II.B.2.b) and in more detail in section II.A.5 of this document.

3. Specific Comments on the Interpretation of Scientific Evidence

More specific comments on the EPA's interetation of the scientific evidence and EPA's responses are discussed below. The summary of comments and responses follows the discussion of these topics in the preamble to the final rule. The first section below contains comments on evidence from controlled human exposure studies (preamble section II.B.2.a.i); the second section below contains comments on evidence from epidemiological studies, including interpretation of the evidence and specific methodological issues (preamble section II.B.2.a.ii); the third section below contains comments on evidence pertaining to at-risk subgroups for O_3-related effects (preamble section II.B.2.a.iii). A fourth section contains comments on the adversity of health effects, a topic that was discussed generally in the preamble and also as part of some responses to comments on at-risk subgroups for O_3-related effects and the fifth section below contains comments on the role of ground-level O_3 in solar radiation-related health effects.

EPA notes here that most of the issues and concerns raised by commenters concerning the health effects evidence, including both the interpretation of the evidence and specific technical or methodological issues, were essentially restatements of issues raised during the review of the Criteria Document and the Staff Paper. Most of these issues were highlighted and thoroughly discussed during the review of these documents by the CASAC. Incorporating responses contained in section II.B.2.a of the preamble to the final rule, EPA provides the following responses to specific issues related to the need to revise the O_3 standard.

a. Evidence from Controlled Human Exposure Studies

(1) *Comment:* NAM contended that EPA did not disclose critical studies and analyses (NAM, p. 17). This commenter stated that a noticeable instance of bias is EPA's failure to disclose at least one critical collection of information within its control: its reanalysis of the data obtained by Adams (2006a). NAM contends that this reanalysis (Brown 2007, "Brown Memorandum") is a crucial element of Agency staff policy recommendations and that EPA did not disclose enough information to make it reproducible. NAM contends that it reaches conclusions opposite of the researcher, such that it is equivalent to a new study inserted into the record in a discriminatory fashion.

Response: EPA has disclosed all critical studies and analyses. As an initial matter, the Brown Memorandum is not a crucial element of the staff's policy recommendations, as it was prepared after completion of the Staff Paper, or the Administrator's final decision. The Brown Memorandum represents a logical progression in the interpretation of a study, not published until 2006, that was included in the final Criteria Document in February 2006. In Chapter 8 of the Criteria Document, it was noted that the FEV_1 responses during exposure to 0.06 ppm ozone appeared to diverge away from the responses of

filtered air and 0.04 ppm ozone after about 5.6 hr. Subsequently, in the Staff Paper, it was noted that a statistically significant difference in FEV_1 responses was suggested by a lack of overlap in the standard error of the responses following 6.6 hours of exposure to 0.06 ppm ozone versus filtered air. That interpretation of the data was supported by CASAC review. In late February 2007, public comments were submitted by a Dr. Richard Smith. In his comments, he provided a table comparing changes in FEV_1 following all of the six exposure protocols utilized by Adams (2006) and the data for 0.04 ppm O_3 (corrected for filtered air responses) from Adams (2002). His table specifically indicated that the FEV_1 responses, in the Adams (2006) study, following the two 0.06 ppm O_3 exposures were statistically different from the FEV_1 responses following filtered air exposures using a paired t test. The Brown Memorandum presents these same comparisons. The table provided in the Brown Memorandum discloses all information necessary to reproduce the standard statistical paired t test which was utilized. Thus, it was a public commenter that first placed the analysis of FEV_1 responses following exposure to 0.06 ppm O_3 versus filtered air in the public rulemaking docket. In short, the Brown Memorandum confirms Dr. Smith's analyses, frames the analysis in terms of the analysis performed by Dr. Adams versus prior O_3 studies, and discusses the biological significance of FEV_1 responses.

(2) *Comment:* NAM also contends that the Brown Memorandum itself exemplifies multiple types of violations of the information quality standard of objectivity (pp. 18-19). First, NAM contends that it is a post hoc statistical analysis conducted on data whose initial analysis did not support the declared policy preferences of Agency staff. Second, NAM contends that it was prompted by a low-quality analytic review ("visual comparison" and "cursory evaluation," p. 3). Ioannidis (2005) shows that positive results are usually false even when researchers exercise normal restraint with respect to Type I error (rejecting the no-effect hypothesis when in fact it is true). NAM also contends that EPA has included or excluded data or studies based on the extent to which they support stated or unstated risk management objectives (NAM, p. 19). In the proposal notice, NAM contends that EPA acknowledges implicitly that Agency staff used the Adams data for purposes that were never intended by the study design. It is reported that two of the 30 healthy adult subjects experienced exercise-adjusted FEV_1 decrements exceeding 10% at 0.060 ppm using one of the two exposure patterns examined (but not the other) (72 FR 37828, n. 16). It is inappropriate to obtain a sample, subject its members to a well designed test, learn that the sample does not yield hoped-for outcome, and in response, abandon the sample in favor of focusing on selected individuals within it. Federal information quality guidelines require transparency. When, as in this case, the disclosed portion of information shows the hallmarks of purposeful bias, the only responsible default inference is that the reanalysis does not meet applicable information quality standards. Unless EPA can show otherwise, the reanalysis should not be disseminated and the Brown Memorandum should be withdrawn.

Response: EPA rejects NAM's contention that the Brown Memorandum exemplifies any violation of the information quality standard of objectivity. The API, which funded the Adams (2006) study, asked that the data from that study be included in the EPA risk assessment. Dr. Adams then provided his data to the EPA. Thus, the funding

organization and the author provided their data for the express purpose that it be included in EPA's analyses. Reporting that two of 30 subjects in the Adams (2006) study experienced exercise-adjusted FEV_1 decrements exceeding 10% at 0.060 ppm is merely a statement of fact by EPA. Adams (2002) reported that 6 of 30 subjects exposed to 0.06 ppm had greater than a 10% decrement in FEV_1. API has refused to provide Dr. Adams technical report describing that data. In the reference cited by NAM, Ioannidis (2005) notes "… that true research findings may occasionally be annulled because of reverse bias. For example, with large measurement errors relationships are lost in noise [12], or investigators use data inefficiently or fail to notice statistically significant relationships, or there may be conflicts of interest that tend to "bury" significant findings [13]."

The Brown Memorandum confirms analyses completed by Dr. Smith who was funded by API to perform his analyses and to provide comments to CASAC. In the Brown Memorandum EPA used the same statistical approach as used by Dr. Smith. In both Dr. Brown's and Dr. Smith's analyses a standard statistical test was used which is appropriate for the type of comparison made. EPA rejects NAM's contention that this approach has the "peculiar quality of dramatically increasing Type I error." Thus, EPA has not included or excluded data that has a stated policy purpose.

The Brown Memorandum objectively describes the FEV_1 responses following exposure to 0.06 ppm ozone relative to filtered air in terms of both statistical and biological significance. The Brown Memorandum discusses relevant public and CASAC comments. The table provided in the Brown Memorandum discloses all information necessary to reproduce the standard statistical paired t test which was utilized. In EPA's judgment, its weight-of-the-evidence approach looks at all of the available evidence and the Brown Memorandum is transparent and without purposeful bias.

(3) *Comment:* NAM contends that EPA draws inferences from a study that are not supported by the data and analysis reported (NAM, p. 27**).** Five clinical studies have been performed since the last review, but only three involved exposures below the current standard level (Adams 2002, 2003b, 2006a). EPA's interpretation of Adams' work diverges, and is inconsistent with, that of the author (Adams 2007). Adams reports no statistically significant effects from O_3 at 0.040 ppm, but EPA finds them in its reanalysis. This poses special information quality issues. EPA's interpretation of Adams' work does not enjoy a rebuttable presumption of objectivity. Moreover, to utilize Adams' work in contrary ways, the Agency must first rebut the presumption of objectivity that attaches to these studies. Furthermore, EPA's reanalysis of Adams' data in the final Staff Paper and Brown Memorandum is neither transparent nor capable of being substantially reproduced. This is an essential prerequisite for adherence to the objectivity standard and inconsistent with EPA's information quality guidelines.

Response: EPA did not draw inferences that were not supported by the data and the reported analysis as NAM contends. The Adams (2007) teleconference comments to CASAC concerned the description of his 2006 publication in the Staff Paper. CASAC heard Dr. Adams comments and did not object to the discussion of the Adams (2006)

publication in the Staff Paper. With regard to the effects of O_3 at 0.040 ppm, NAM is incorrect. Neither the Adams (2006) publication nor EPA describe statistically significant effects of exposure to 0.04 ppm O_3 on lung function. As already discussed, the table provided in the Brown Memorandum discloses all information necessary to reproduce the standard statistical paired t test which was utilized.

(4) *Comment:* NAM also contends (NAM, p. 29) that EPA's analysis of clinical data on cardiac effects is problematic with respect to information quality standards. In the proposed rule, NAM contends that EPA emphasizes the increase in AaPO2 and interprets it as evidence that O_3 exposure "result[s] in an overall increase in myocardial work and impairment in pulmonary gas exchange" (72 FR 38734). EPA says nothing about the relevance of the exposure level, which was 0.3 ppm -- 3.75 times greater than the current NAAQS, or the uncertainties implied by extrapolating to the population clinical data obtained from a sample of 16.

Response: proposal notice. The quote provided by NAM, which appeared in the Staff Paper and proposal notice was based on the conclusion of Dr. Henry Gong stated in the original article. Dr. Gong was a physician and first author of the study in question. The details of his study were provided in Chapter 6 and Annex 6 of the Criteria Document. Furthermore, in the paragraph to which NAM has referred (72 FR 38734), the EPA specifically states, "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*" (emphasis added). Absent evidence to the contrary, there is no reason to believe that there would be a threshold O_3 concentration for such effects. Additionally, the Gong et al. (1998) study was ethically restricted to patients with mild hypertension and could not examine these effects in individuals more severely affected by cardiovascular disease. EPA explicitly stated that mechanistic data was limited and needed to be substantiated. In that context, no further discussion of limitations or extrapolations was warranted

(5) *Comment:* Some commenters cited the discussion of the controlled human exposure studies in the Rochester Report as supporting their views that the current standard should be retained. The following passages from the Rochester Report summarize the points made in that report:

> Adams (2006a), seeking differences in patterns of response among the different exposures, utilized a Scheffe post hoc test for controlling study-wide level of alpha while making multiple comparisons among the many data points. This test (which is not particularly powerful for detecting specific differences in the context of large numbers of comparisons) did not identify the response of the 0.06 ppm exposure as statistically different from that of the FA exposure. However, alternative statistical tests suggest that the observed small group mean response in FEV_1 induced by exposure to 0.06 ppm compared to FA is not the result of chance alone. The mean difference in the FEV_1 decrements between the two exposures at 6.6 hours was approximately 2.9% which was statistically different

($p<0.001$) from zero when tested using a t-statistic without correction for multiple comparisons.

Further examination of the post-exposure FEV data and mean data at other time points and concentrations also suggest a pattern of response at 0.06 ppm that is consistent with a dose-response rather than random variability. For example, the response at 5.6 hours was similar to that of the post-exposure 6.6 hour response and appeared to also differ from the FA response. The volunteers in this study did not appear to be more responsive to ozone than volunteers in previous studies as the observed response at 0.08 ppm in this study was similar to that of previous studies. Although of much smaller magnitude, the temporal pattern of the 0.06 ppm response was generally consistent with the temporal patterns of response to higher concentrations of ozone in this and other studies. Responses below 0.08 ppm ozone have not previously been observed, but this finding is not totally unexpected because the previously observed FEV responses to 0.08 ppm were in the range of 6-9% suggesting that exposure to lower concentrations of ozone would result in smaller, but real FEV decrements. The EPA re-analysis and re-interpretation of the studies of Adams has been questioned by Adams (2007) and by Smith (2007) in presentations to the Clean Air Scientific Advisory Committee. Thus, the public health significance of responses at 0.06 ppm ozone is still being debated. The Panel recognizes that uncertainty necessarily surrounds a secondary analysis and the integration of results from a single study in one laboratory with 0.06 ozone exposures and results obtained in studies at higher concentrations by other investigators. Resolution of this uncertainty will require that further research be conducted to clarify the issue. (Rochester Report, pp. 56-57)

Response: The EPA generally agrees with the Rochester Report's evaluation of the Adams (2006) study and the interpretation of the EPA's secondary analysis of the data. EPA agrees that there is a small group mean response in FEV_1 induced by exposure to 0.060 ppm ozone compared to FA and that the observed response is not the result of chance alone. Furthermore, EPA agrees that the pattern of response at 0.060 ppm ozone is consistent with a dose-response curve rather than random variability. Given that the 0.060 ozone exposures and results have not been replicated, some uncertainty exists and EPA agrees that further research is needed to clarify the issue.

(6) *Comment:* One commenter submitted a statistical analysis and evaluation (Exxon Mobil) of the Adams (2006) study and data by Dr. Nicolich. Dr. Nicolich reached four conclusions from his analysis:
1) Based on the additional statistical analyses detailed in this report, we have shown that the basic results of Adams (2006) ozone chamber study were replicated.
2) Based on analyses of the response pattern over the 6.6 hours of experimentation, the only percent changes in FEV_1 responses that are statistically significantly different from the first period fresh air exposures were the 0.08 ppm triangle exposure at hours 4.6, 5.6 and 6.6 hours and the 0.08 ppm square wave exposure at 6.6 hours.
3) The analysis of only the 6.6 hour readings indicated the 0.08 ppm square wave and 0.08 ppm triangle wave exposures were different from the fresh air exposure.

4) The magnitude of the estimated changes for all individuals in all exposure groups are less than the 15 percent decrement considered to be of clinicall significance by the American Thoracic Society.

Response: Dr. Nicolich conducted a statistical reanalysis of the Adams (2006) data. To correct for the multiple comparisons, the significance level was adjusted by the Dunnett's test which "is not as conservative as Scheffé's test or as extreme as individual t-tests." It should be recognized that Dr. Nicolich ran multiple comparisons and had to correct for these multiple comparisons in order to avoid type 1 error (falsely rejecting the null hypothesis when there is no effect). Thus, the probability of type 2 error (falsely accepting the null hypothesis when a real effect exists) was increased just as in the case of Dr. Adams' analysis. Furthermore, Dr. Nicolich did not provide estimates of the power of his statistical analysis to detect an effect. Therefore, the lack of being able to find a statistically significant effect at 0.060 ppm ozone does not mean that no effect exists. On page A-3, Dr. Nicolich states "that the residuals are not normally distributed and the observations do not meet the assumptions required for the model" and that "the subject-based errors are not independently, identically and normally distributed and the subjects do not meet the assumptions required for the model." Later on page A-5, Dr. Nicolich states that his "results are essentially correct, but the significance levels may not be exactly correct." Regarding the analysis of responses at 6.6 hours, Dr. Nicolich again stated that the "significance levels may not be exact because of the failure to meet the normality assumption." Therefore, given that the underlying statistical assumptions required for his analysis were not met and that significance levels are questionable, in EPA's judgment the analyses presented by Dr. Nicolich are ambiguous.

On page A-5 of his comments, Dr. Nicolich states that "The model predicted response for individuals in Figure 3 shows that none of the predicted points shows more than about a 6 percent decrement and the lower confidence intervals (not shown) are all greater than 10 percent and, therefore, do not include the 15 percent decrement considered to be of clinical significance by the ATS (Wang and Petsonk 2004)." On inspection of the Adams (2006) data for the 0.080 ppm exposures, a greater than 15% ozone-induced decrement in FEV_1 is seen in 2 individuals for the square-wave exposure protocol and in 3 individuals for the triangular exposure protocol. It is unclear why Dr. Nicolich would utilize a statistical model that violates basic statistical assumptions to support his assertion. The commenter does not accurately characterize the cited ATS guidelines on the magnitude of lung function (FEV_1) change. The cited guidelines were not defining the magnitude of lung function (FEV_1) change that can be considered to be clinically adverse. The cited guidelines were established to help determine whether a change in lung function, between two tests made a year or more apart, is "real" or only the result of test variability (ATS 1991). The guideline goes on to note that all lung function measurements tend to be more variable when made weeks to months apart than when repeated at the same test session or even daily. EPA's discussion of adversity in the Staff Paper, NPRM, preamble to the final rule and this Response to Comments document appropriately characterizes the ATS adversity guidelines (ATS, 2000). Therefore, Dr. Nicolich's comment regarding clinical significance of FEV_1 responses in the Adams (2006) study is without merit.

(7) *Comment:* Dr. Adams in his October 2007 comments states that the FEV_1 response in healthy young adults to 6.6 h exposure to 0.060 ppm O_3 in his study (Adams, 2006a) do not demonstrate a significant mean effect by ordinarily acceptable statistical analysis and then references the analysis of Dr. Mark Nicolich in support of this assertion. He considers the response to be in somewhat of a gray area, both in terms of a biologically meaningful response and a statistically significant response. Further, he feels that more studies of human pulmonary function (and measurements of related physiological mechanisms) should be conducted in prolonged 0.060 ppm O_3 exposures as soon as possible.

Response: Both Dr. Adams and Dr. Nicolich ran multiple comparisons which increases probability of type 2 error (falsely accepting the null hypothesis when a true effect exists) when evaluating the simple issue of whether post-exposure lung function was decreased in subjects exposed to 0.060 ppm ozone versus filtered air in the Adams (2006) study. The two-factor analysis of variance and Scheffé method cited by Adams (2006) assumes that data are normally distributed and that variances are equal within cells. From previous studies of higher O_3 exposures, we know that FEV_1 responses become skewed and variance increases (McDonnell 1996). This increase in variance is clearly apparent in the Adams (2006) data with the standard deviation of FEV_1 responses increasing from 2.98% for filtered air to 4.24% for exposures at 0.060 ppm O_3 to 8.65% for exposures at 0.080 ppm. Dr. Nicolich's analysis also found that the underlying statistical assumptions of data normality required for Dr. Adams' analysis were violated. Thus, it is very possible that the wide range in variances among the cells could have resulted in wider confidence levels and inaccuracies of the reported p-values for a subset of the comparisons. Dr. Nicolich even stated that "significance levels may not be exact because of the failure to meet the normality assumption." Therefore, as addressed in response to Dr. Nicolich's comments, not finding a statistically significant effect at 0.06 ppm ozone does not mean that no effect exist.

Dr. Adams contends that, "it appears that paired *t* tests were applied to my data without an initial ANOVA to examine whether there was statistical significance in pre- versus post-exposure FEV_1 response across multiple exposure conditions. If this were done, it would necessitate a post-hoc correction of the paired t test results that might or might not result in the statistical significance for the FEV_1 response reported in the memorandum. Statistical texts (e.g., Neter et al., 1996) routinely recommend that ANOVA is preferable because the t-test is too extreme (non-conservative)" (Adams, 2007, p. 2). Dr. Adams is not accurate with regard to the analysis presented in the Brown Memorandum. The effects of other exposure protocols (0.040 and 0.080 ppm) were not considered. The goal of the memorandum was to address whether there was a pre- to postexposure change in the lung function of individuals exposed to 0.060 ppm ozone versus filtered air. A repeated measures ANOVA of the change in FEV_1 following exposure to 0.060 ppm ozone versus filtered air provides the same result as the paired t test which was utilized in the Brown Memorandum, i.e., a relatively small but statistically significant decrease in group mean FEV_1 responses following 6.6 h of exposure to 0.060 ppm ozone compared to filtered air. Dr. Adams' comments (page 2) regarding the magnitude of lung function decrements due to exposure to 0.080 ppm versus 0.060 ppm ozone or other ozone

exposure protocols are not relevant other than to establish that a trend in responses exists. Therefore, there was no need for an initial repeated measures ANOVA of all the exposure protocols in the Adams (2006) study to discern if ozone effected post-exposure lung function at 0.060 ppm versus filtered air.

As presented in the Brown Memorandum, paired *t* test were conducted for the two 0.060 ppm exposure protocols versus filtered air responses. The reported two-tailed significance levels were 0.009 and 0.001 for the triangular and square-wave exposure protocols, respectively. Dr. Adams contends (Adams, 2007, p. 3) that "no correction for post hoc application is used (which would result in their increase by 5 times)" and thereby lead to a critical p-value of 0.01 (i.e., 0.05/5). However, it should be noted that in actuality, only two comparisons were made in the Brown Memorandum. Clearly, the significance levels reported in the Brown Memorandum which were <0.01 should remain statistically significant following any reasonable multiple comparison correction. Dr. Adams also generally objected to the use of a *t* test for evaluating the significance of results from his study. However, Dr. Richard Smith (a statistician at the University of North Carolina) also utilized *t* tests to evaluate the statistical significance of the Adams data in his public comments to CASAC (March 5, 2007). Therefore, we conclude that ordinarily acceptable statistical analyses such as presented in the Brown Memorandum show that exposure to 0.060 ppm O_3 causes a relatively small but statistically significant decrease in group mean FEV_1 responses compared to filtered air.

Dr. Adams agrees in his October 2007 comments (p. 3) that pre- to postexposure responses may be an acceptable means of evaluating effects on lung function for the square-wave exposure protocols. He does not, however, consider it appropriate for analysis of triangular exposure protocols. Dr. Adams' concern arises because during triangular exposures to 0.080 and 0.120 ppm ozone, subjects have been observed to experience their maximal pulmonary function decrements at one to two hours before the end of the exposure. Hence, by examining only pre- to postexposure pulmonary function changes, the maximal effects would be missed. However, as noted by Dr. Adams (Adams, 2007, p. 2) "no such tendency is apparent for FEV_1 response with time in the two 0.06 ppm exposures in Fig. 1 of the memorandum. Rather, there is a non-significantly greater drop from 4.6 h to 5.6 h for the 0.06 ppm square-wave exposure than for the 0.06 ppm triangular exposure." Therefore, for the 0.060 ppm ozone protocols, it appears that the use of pre- to postexposure responses are an acceptable means of evaluating effects on lung function for the square-wave and triangular exposure protocols.

Dr. Adams did correctly note, contrary to what is stated in the Brown Memorandum, that Horstman et al. (1995) used an ANOVA for a split-plot design to test the hypothesis that pre- minus post-exposure differences in FEV_1 for the air and O_3 exposures were the same for a group of asthmatics as for a group of nonasthmatics. The t tests were used in the Horstman et al. (1995) study to evaluate the differences between an aerosol test of small airways function, not FEV_1, between the asthmatics and nonasthmatics.

Overall, there appears to be general agreement among Dr. Adams, the Rochester Report, and the EPA that the effects on pulmonary function at 0.060 ppm O_3 appear to be consistent with the trend in FEV_1 responses observed for exposures ranging from to 0.04 ppm to 0.08 ppm O_3 and that further studies below 0.08 ppm are warranted.

b. Evidence from Epidemiological Studies

This section contains comments on EPA's assessment of epidemiological studies in the proposal and the Agency's general responses to those comments. Comments on EPA's interpretation and assessment of the body of epidemiological evidence are discussed first and then comments on methodological issues and particular study designs are discussed. EPA notes here that most of the issues and concerns raised by commenters on the interpretation of the epidemiological evidence and methodological issues are essentially restatements of issues raised during the review of the Criteria Document and Staff Paper. EPA presented and the CASAC Panel reviewed the interpretation of the epidemiological evidence in the Criteria Document and the integration of the evidence with policy considerations in the development of the policy options presented in the Staff Paper for consideration by the Administrator. CASAC reviewed both the Criteria Document and Staff Paper and approved of the scientific content and accuracy of both documents. The CASAC chairman sent to the Administrator one letter (Henderson, 2006a) for the Criteria Document and another letter for the Staff Paper (Henderson, 2006c) indicating that these documents provided an appropriate basis for use in regulatory decision making regarding the O_3 NAAQS.

As with evidence from controlled human exposure studies, comments on the evidence from epidemiological studies, including EPA's interpretation of the evidence, were highly polarized. One group of commenters from medical, public health and environmental organizations, in general, supported EPA's interpretation of the epidemiological evidence (72 FR 37838, sections II.a.3.a-c) with regard to whether the evidence for associations is consistent and coherent and whether there is biological plausibility for judging whether exposure to O_3 is causally related to respiratory and cardiovascular morbidity and mortality effects.

(1) *Comment:* Comments of public health and environmental groups, including a joint set of comments from ALA and several environmental groups (ALA et al.), note that more than 250 new epidemiological studies, published from 1996 to 2005, were included in the Criteria Document and point to a figure from the Staff Paper and proposal (72 FR 37842, Figure 1) of short-term O_3 exposures and respiratory health outcome showing consistency in an array of positive effects estimates and health endpoints observed in multiple locations in Canada and the U.S.

Medical commenters, including ATS and AMA, stated that these "real world" studies support the findings of chamber studies to show adverse respiratory health effects at levels below the current 8-hour O_3 standard. These commenters generally expressed agreement with the weight of evidence approach taken by the Criteria Document and the conclusions reached, which were reviewed by CASAC, that the effects of O_3 on respiratory symptoms, lung function changes, emergency department visits for respiratory and cardiovascular effects, and hospital admissions can be considered causal.

However, in contrast with EPA, these commenters assert that the causal associations extend down to the lowest ambient O_3 concentrations reported in these studies. These commenters also expressed the view that the respiratory and cardiovascular system effects are well-supported by the Hill criteria[3] of judging causality: strength of association, consistency between studies, coherence among studies, and biological plausibility (ALA et al., pp. 51-52). Medical commenters, including ATS and AMA, state that these "real world" studies support the findings of chamber studies to show adverse respiratory health effects at levels below the current 8-hour O_3 standard. They also note that recent studies provide compelling evidence that exposure to O_3 results in adverse cardiovascular health effects (ATS, pp. 6-7).

Response: EPA generally agrees with this interpretation of the epidemiological evidence. The Criteria Document concludes that positive and robust associations were found between ambient O_3 concentrations and various respiratory disease hospitalization outcomes and emergency department visits for asthma, when focusing particularly on results of warm-season analyses. 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 (Criteria Document, p. 8-77).

However, EPA disagrees with the assertion of these commenters that the causal associations extend down to the low ambient O_3 concentrations reported in these studies. The biological plausibility of the epidemiological associations is generally supported by controlled human exposure and toxicological evidence of respiratory morbidity effects for levels at and below 0.080 ppm, but that biological plausibility becomes increasingly uncertain especially below 0.060 ppm, the lowest level at which effects were observed in controlled human exposure studies. Further, at lower levels, it becomes increasingly uncertain as to whether the reported associations are related to O_3 alone rather than to the broader mix of air pollutants present in the ambient air. EPA notes that the multi-city times series studies evaluated in this review do not resolve this issue. It also becomes increasingly uncertain as to whether effect thresholds exist but can not be clearly discerned by statistical analyses. Thus, when considering the epidemiological evidence in light of the other available information, it is reasonable to judge that at some point the epidemiological associations can not be interpreted with confidence as providing evidence that the observed health effects can be attributed to O_3 alone. For a fuller discussion of EPA's approach to making judgments about causal relationships see response to comment 6 below.

[3] The Hill criteria, published by Sir Bradford Hill (1965), are commonly used criteria for reaching judgments about causality from observed associations, and these criteria were the basis for the critical assessment of the epidemiological evidence presented in the Criteria Document (pp. 7-3 – 7-4).

With regard to cardiovascular health outcomes, the Criteria Document concludes that the generally limited body of evidence from animal toxicology, human controlled exposure, and epidemiologic studies is suggestive that O_3 can directly and/or indirectly contribute to cardiovascular-related morbidity, and that for cardiovascular mortality the Criteria Document suggests that effects estimates are more consistently positive and statistically significant in warm season analyses but that additional research is needed to more fully establish the underlying mechanisms by which such mortality effects occur (EPA, 2006a, pp. 8-77-78).

The second group of commenters, mostly representing industry associations and some businesses opposed to revising the primary O_3 standard, disagreed with EPA's interpretation of the epidemiological evidence.

(2) *Comment:* These commenters expressed the view that while many new epidemiological studies have been published since the current primary O_3 standard was promulgated, the inconsistencies and uncertainties inherent in these studies as a whole should preclude any reliance on them as justification for a more stringent primary O_3 NAAQS. They contend that the purported consistency is the result of inappropriate selectivity in focusing on specific studies and specific results within those studies (UARG, p. 15). With regard to daily mortality, the proposal emphasizes the multi-city studies, suggesting that they have the statistical power to allow the authors to reliably distinguish even weak relationships from the null hypothesis with statistical confidence. However, these commenters note that these studies are not consistent, with regard to the findings concerning individual cities analyzed in the multi-city analyses. One commenter asserted that each of the multi-city studies and meta-analyses cited by EPA involves cities for which the city-specific estimates of O_3 effects have been observed to vary over a wide range that includes negative [i.e., beneficial] effects (API, p. 15). To illustrate this point, many commenters point to EPA's use of the study by Bell et al., 2004. They note that in focusing on the national estimate from Bell of the association between 24-hour average O_3 levels and daily mortality, the Administrator overlooks the very significant and heterogeneous information of the individual analyses of the 95 cities used to produce the national estimate and, based on this inconsistency, question whether what is being seen is actually an O_3 mortality association at all (UARG, p. 16).

Response: EPA has accurately characterized the inconsistencies and uncertainties in the epidemiological evidence and strongly denies that it has inappropriately focused on specific positive studies or specific positive results within those studies. EPA's assessment of the health effects evidence in the Criteria Document has been reviewed by the CASAC Panel. EPA has appropriately characterized the heterogeneity in O_3 health effects in assessing the results of the single-city and multi-city studies and the meta-analyses, as discussed in section 7.6.6 of the Criteria Document In general, the Administrator recognizes that in the body of epidemiological evidence, many studies reported positive and statistically significant associations, while others reported positive results that were not statistically significant, and a few did 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 became increasingly uncertain at lower levels of exposure.

More specifically, the Bell et al. (2004) study observed a statistically significant, positive association between short-term O_3 concentrations (24-hour average) and all-cause mortality using data from 95 U.S. National Morbidity, Mortality, and Air Pollution Study (NMMAPS) communities. The objective of the NMMAPS was to develop an overall national effect estimate using multi-city time-series analyses, by drawing on information from all of the individual cities. The strength of this approach is the use of a uniform analytic methodology, avoidance of selection bias, and larger statistical power. Significant intercity heterogeneity was noted in the Bell et al. and other multi-city studies, probably due to many factors, including city-specific differences in pollution characteristics, the use of air conditioning, time spent indoors versus outdoors, and socioeconomic factors. Levy et al. (2005) found suggestive evidence that air conditioning prevalence was a predictor of heterogeneity in O_3 effect estimates in their meta-analysis.

(3) *Comment:* NAM contends that EPA has not considered publication bias in the presentation of reported results (NAM, pp. 11-13).

Response: Nam's contention is not true, since EPA recognized the potential impact of publication bias on the conclusions that may be drawn from a body of studies, as indicated in section 7.1.3.6 of the Criteria Document. NAM describes the theoretical basis for publication bias, but EPA was able to draw upon the findings of several studies that assessed the potential for publication bias for O_3-related health effects studies. As described in section 7.4.4 of the Criteria Document, two meta-analyses investigating the association between short-term exposure to O_3 and mortality also examined the evidence for publication bias in the available literature. Bell et al. (2005) concluded that the results provided strong evidence of an association between O_3 and mortality that was not sensitive to adjustment for PM or for model specifications. However, they suggested that, based on comparisons between the meta-analysis results and NMMAPS results from 95 U.S. communities (Bell et al., 2004), there was evidence of publication bias (1.75% [95% CI: 1.10, 2.37] per 20 ppb increase in 24-h avg O_3 for meta-analysis versus 0.50% [95% CI: 0.24, 0.78] for NMMAPS 0-day lag results). Ito et al. (2005) also observed a statistically significant association between O_3 and mortality that was generally robust to adjustment for PM. They found suggestive evidence of publication bias (a significant asymmetry in the funnel plot), but adjusting for the asymmetry reduced the combined estimate only slightly (from 1.6% [95% CI: 1.1, 2.0] to 1.4% [95% CI: 0.9, 1.9] per 20 ppb increase in 24-h avg O_3). The extent of potential bias implicated in this study differed compared to that reported by Bell et al. (2005). The source of this difference is not clear, but Ito et al. stated that sensitivity analyses comparing estimates from commonly used weather model specifications suggest that the stringent weather model used in NMMAPS may tend to yield smaller risk estimates than those used in other studies.

NAM also contends that EPA should, for each critical study, determine the extent to which nonpositive outcomes were not reported and include that information in its presentation. EPA rejects NAM's contention that it should determine the extent to which nonpositive outcomes were not reported and include that information in its presentation of each critical study. First, there is no evidence to show that researchers are not reporting all results. Second, EPA can not include in its assessment results that were not reported. Third, EPA uses a weight of evidence approach to evaluate evidence that does not depend on a few critical studies.

Specifically, NAM (p. 13) cites three studies of respiratory symptoms (Gent et al., 2003; Korrick et al., 1998; Mortimer et al., 2002) as example of studies that "report only the most statistically significant results." This is clearly not the case, as Gent et al. (2003), for example, reported that while effects were observed in asthmatic children using maintenance medication, no effects were observed among asthmatics not using maintenance medication. In the Criteria Document, EPA recognized the post-hoc nature of the population stratification by medication use as one of the limitations of this study.

NAM contends that Gent et al. (2003) exaggerates their findings in their conclusions regarding the susceptibility of asthmatic children using maintenance medication. NAM states "Peak ozone exposures, which logically drive the results observed, exceeded current standards" (NAM, p. 13). However, there is no reason to believe that the peak O_3 concentrations drive the findings of this study. The Mortimer et al. (2002) study observed that excluding days when 8-h avg O_3 levels were greater than 80 ppb provided effect estimates that were similar to those when all days were included in the analysis, indicating that the negative effect of O_3 on morning PEF were not driven by the peak O_3 concentrations.

NAM also contends that Mortimer et al. (2002) selectively reported study results, by focusing on specific average lag periods for the various pollutants. However, as discussed in section 7.2.3.2 of the Criteria Document, Mortimer et al. examined the association between O_3 and respiratory effects using single day lags from lag day 1 to lag day 6. Small morning effects were observed at 1- and 2-day lags. The effect of O_3 on morning outcomes increased over several days. Examination of these single lag day effects led to the consideration of a multiday lag period of 1 to 5 days in the case of PEF and 1 to 4 days in the case of respiratory symptoms to estimate the cumulative effect of O_3. The different multiday lag periods used for the various pollutants likely reflects the different relationship between the individual pollutant and the health effects.

NAM further states that the odds ratios in the Mortimer et al. (2002) study were "barely statistically significant" and it cannot be discerned whether any of these ratios would have retained statistical significance or declined in magnitude in a multipollutant model. In fact, Mortimer et al. does report multipollutant model results, as presented in section 7.2.4 of the Criteria Document. The odds ratios for the incidence of symptoms per 30 ppb increase in 8-h avg O_3 were 1.23 (95% CI: 0.94, 1.61) with SO_2 and 1.14 (95% CI: 0.85, 1.59) with NO_2. In the three urban areas with PM_{10} data, the odds ratios were 1.21 (95% CI: 0.61, 2.40) in the O_3-only model and 1.08 (95% CI: 0.41, 2.40) when PM_{10} also

was included in the model. Though the O_3 effect was shown to be slightly diminished and did not retain statistically significance in multipollutant models, there was considerable overlap in the 95% confidence intervals between the single-pollutant and multipollutant model results, leading EPA to conclude that the association was generally robust.

Finally, NAM contends that EPA did not recognize the fundamental data quality problems with self-reported respiratory testing found by Kamps et al. (2001). In section 7.2.3 of the Criteria Document, EPA does in fact state that PEF measurements have been shown to be more variable than FEV_1 in some studies (Vaughan et al., 1989; Cross and Nelson, 1991) and can have an element of uncertain reliability when self-administered by study subjects. However, Lippmann and Spektor (1998) state that PEF measurements from small inexpensive flow meters, which are more feasible to use in field studies, can produce similar results to PEF measured spirometrically.

In conclusion, EPA does not agree that reported associations between O_3 and health effects are an artifact of publication bias. The EPA acknowledges that publication bias can result in potential overestimation of the estimated risk in a body of literature. However, for an individual study, factors such as exposure error or selection of results from an individual lag period from among several positive associations can result in underestimation of an effect estimate.

(4) *Comment:* NAM contends that EPA inappropriately assumes confidence intervals adequately describe variability and uncertainty (NAM, p. 49).

Response: EPA does not inappropriately assume that confidence intervals fully describe variability and uncertainty. As an initial matter, EPA notes that while statistical significance (i.e., confidence intervals) is considered in the evaluation of the scientific evidence, EPA has emphasized the importance of examining the pattern of results across various studies and not focusing solely on statistical significance as a criterion (discussed in further detail in response to comments below and in section II.A.2 of the preamble to the proposed decision). Second, the point that the uncertainty ranges reported in the risk assessment do not reflect all of the uncertainty in the risk estimates is explicitly discussed in the Staff Paper (section 5.3.2.5).

NAM contends that epidemiologists tend to use convenience samples and other non-random research designs, which result in reported confidence intervals representing "best case" conditions. EPA disagrees that most of the epidemiologic literature evaluated in the O_3 Criteria Document is based on non-random research designs. Not all epidemiologic studies evaluated in the O_3 Criteria Document use study populations that are *generalizable* to the entire population, but this does not mean that the study population was non-random. In fact, these epidemiologic studies tend to randomly select subjects into their study based on the study selection criteria. The study selection criteria would limit the generalizability of the study results, for example, results from a study of asthmatic children are likely not generalizable to healthy children or asthmatic adults, but this does not imply a lack of randomization. In time-series studies that use population-

level data, randomization is also not an issue as the available data on the entire population (or within certain age groups of the population) in the specified location are utilized.

(5) *Comment:* Several commenters argued that EPA overstates the probability of causal links between health effects and exposure to O_3, especially at the lower concentrations examined, and that the statistical associations found in the cited epidemiological studies do not automatically imply that a causal relationship exists. These commenters expressed the view that the correlation between health effects and O_3 exposure must be rigorously evaluated according to a standard set of criteria before concluding that there is a causal link and that EPA fails to articulate and follow the weight of the evidence or established causality criteria for evaluating epidemiological studies in drawing conclusion regarding causality (Exxon Mobil, pp. 10-11).

Response: In the proposal, EPA explicitly stated that epidemiological studies are not themselves direct evidence of a causal link between exposure to O_3 and the occurrence of effects (72 FR 37879). Throughout the O_3 review, a standard set of criteria have been used to evaluate evidence of a causal link. The critical assessment of epidemiological evidence presented in the Criteria Document was conceptually based upon consideration of salient aspects of the evidence of associations so as to reach fundamental judgments as to the likely causal significance of the observed associations in accordance with the Hill criteria (Criteria Document, pp. 7-3 - 7-4). Moreover, consistent with the proposal the Administrator has specifically considered evidence from epidemiological studies in the context of all the other available evidence in evaluating the degree of certainty that O_3-related adverse health effects occur at various levels at and below 0.080 ppm, including the strong evidence from controlled human exposure studies and the toxicological studies that demonstrate biological plausibility and mechanisms for effects. More detailed discussion of the criteria used to evaluate evidence with regard to judgments about causality can be found in section d. below.

(6) *Comment:* NAM contends that EPA uses policy judgment, rather than scientific judgment in assessing causality (NAM, pp. 37-39).

Response: EPA strongly disagrees with this comment by NAM that EPA's approach to judging causality is "unambiguously and transparently policy directed" (NAM, p. 38) EPA's approach to assessing the epidemiologic evidence is presented in section 7.1.2 of the Criteria Document. The critical assessment of epidemiologic evidence presented in the Criteria Document is conceptually based upon consideration of salient aspects of the evidence of associations so as to reach fundamental judgments as to the likely causal significance of the observed associations, as described by Hill (1965).

NAM further alleges that EPA's analysis and presentation of the scientific evidence does not follow a "plausibly objective analysis." (p. 38) In developing an integrated assessment of the health effects evidence for O_3, EPA has emphasized the importance of examining the pattern of results across various studies, and not focusing solely on statistical significance as a criterion. In doing so, EPA recognizes the distinction between evaluation of individual study results and integration of a body of evidence. Individual

studies are discussed and evaluated to assess their relative scientific quality. Statistical significance is an indicator of the precision of that study's results, which is influenced by the size of the study, as well as exposure and measurement error and other such factors. It is important not to focus the on results of statistical tests to the exclusion of other information. As observed by Rothman (1998):

> Many data analysts appear to remain oblivious to the qualitative nature of significance testing. Although calculations based on mountains of valuable quantitative information may go into it, statistical significance is itself only a dichotomous indicator. As it has only two values, significant or not significant, it cannot convey much useful information. . . . Nevertheless, P-values still confound effect size with study size, the two components of estimation that we believe need to be reported separately. Therefore, we prefer that P-values be omitted altogether, provided that point and interval estimates, or some equivalent, are available. (Rothman, 1998, p. 334)

The concepts underlying EPA's approach to integrated assessment of statistical associations have been discussed in numerous publications, including a recent report by the U.S. Surgeon General on the health consequences of smoking (Centers for Disease Control and Prevention, 2004). This report also cautions against over-reliance on statistical significance in evaluating the overall evidence for an exposure-response relationship.

Hill made a point of commenting on the value, or lack thereof, of statistical testing in the determination of cause: "No formal tests of significance can answer those [causal] questions. Such tests can, and should, remind us of the effects the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that, they contribute nothing to the 'proof' of our hypothesis" (Hill, 1965, p. 299).

Hill's warning was in some ways prescient, as the reliance on statistically significant testing as a substitute for judgment in causal inference remains today (Savitz et al., 1994; Holman et al., 2001; Poole 2001). To understand the basis for this warning, it is critical to recognize the difference between inductive inferences about the truth of underlying hypotheses, and deductive statistical calculations that are relevant to those inferences, but that are not inductive statements themselves. The latter include p values, confidence intervals, and hypothesis tests (Greenland 1998; Goodman 1999). The dominant approach to statistical inference today, which employs those statistical measures, obscures this important distinction between deductive and inductive inferences (Royall 1997), and has produced the mistaken view that inferences flow directly and inevitably from data. There is no mathematic formula that can transform data into a probabilistic statement about the truth of an association without introducing some formal quantification of external knowledge, such as in Bayesian approaches to inference (Goodman 1993; Howson and Urbach 1993). Significance testing and the complementary estimation of confidence intervals remain useful for characterizing the role of chance in producing the association in hand (CDC, pp. 23-24).

Accordingly, the statistical significance of individual study findings has played an important role in EPA's evaluation of the study's results, and EPA has placed greater emphasis on studies reporting statistically significant results. However, in the broader evaluation of the evidence from many epidemiologic studies, EPA has also emphasized the *pattern* of results for drawing conclusions on the relationship between air pollutants and health outcomes, as well as consideration of the integration of epidemiologic evidence with findings of laboratory studies.

It is also important to reiterate that the EPA's evaluation of the scientific evidence was reviewed in detail by CASAC and the public. Two drafts of the Criteria Document were released for CASAC and public review at public meetings, and an additional teleconference meeting was held with CASAC for review of Chapter 8. Evidence related to the substantive issues raised by the commenters were evaluated in the Criteria Document drafts, and discussed at length in public CASAC meetings. This process ensured that overemphasis or underemphasis on any study or group of studies was addressed.

(7) *Comment:* Several commenters made the point that the results of the new epidemiological studies included in this review are not coherent. They state that although EPA notes that estimates of risk from cardiovascular mortality are higher than those for total mortality and indicates that these findings are highly suggestive that short-term O_3 exposure directly or indirectly contributes to cardiovascular mortality, the Agency fails to contrast the mortality studies to studies of hospital admissions for cardiovascular causes. Most studies of cardiovascular causes have not found statistically significant associations with O_3 exposures (UARG, pp. 16-17).

Response: EPA strongly disagrees that it has failed to appropriately characterize the association between O_3 exposure and potential cardiovascular morbidity and mortality effects. As noted above, the Criteria Document characterizes the overall body of evidence as limited, but highly suggestive, and concludes that much needs to be done to more fully integrate links between ambient O_3 exposures and adverse cardiovascular outcomes (Criteria Document, p. 8-77). Some field/panel studies that examined associations between O_3 and various cardiac physiologic endpoints have yielded limited epidemiological evidence suggestive of a potential association between acute O_3 exposure and altered HRV, ventricular arrhythmias, and incidence of myocardial infarction (Criteria Document, section 7.2.7). In addition, there were approximately 20 single-city studies of emergency department visits and hospital admissions for all cardiovascular diseases or specific diseases (i.e., myocardial infarction, congestive heart failure, ischemic heart disease, dysrhythmias). In the studies using all year data, many showed positive results but few were statistically significant. Given the strong seasonal variations in O_3 concentrations and the changing relationship between O_3 and other copollutants by season, inadequate adjustment for seasonal effects might have masked or underestimated the associations. In the limited number of studies that analyzed data by season (6 studies), statistically significant associations were observed in all but one study (Criteria Document, section 7.3.4). Newly available animal toxicology data provide some plausibility for the observed associations between O_3 and cardiovascular outcomes.

EPA believes that its characterization of the evidence for O_3-related cardiovascular system effects is appropriate. It is clear that coherence is stronger in the much larger body of evidence of O_3-related respiratory morbidity and mortality effects.

Many commenters who did not support revising the current O_3 primary standard also submitted comments on specific methodological issues related to the epidemiological evidence, including: the adequacy of exposure data; confounding by copollutants; model selection; evidence of mortality; and, new studies not included in the Criteria Document. The comments on methodological issues raised by these commenters are discussed below.

(8) *Comment:* Many commenters, mostly representing industry associations and some businesses opposed to revising the primary O_3 standard, expressed concern about the adequacy of exposure data both for time-series and panel studies. These commenters argued that almost all of the epidemiological studies on which EPA relies in recommending a more stringent O_3 standard are based on data from ambient monitors for which there is a poor correlation with the actual personal exposure subjects receive during their daily activities. They questioned the Administrator's conclusion that 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 epidemiological studies. These commenters also note that, in its June 2006 letter, the CASAC Panel raised the issue of exposure error, concluding that it called into question whether observed associations could be attributed to O_3 alone (API, p. 17). One of these commenters cited studies (e.g., Sarnat et al., 2001; Sarnat et al., 2005) that show a lack of correlation between personal exposures and ambient concentrations (NAM, p. 22). Another cited studies (Sarnat et al., 2001, 2005, and 2006; and Koutrakis et al., 2005) that have found that the ability of ambient gas monitors to represent personal exposure to such gases is similarly quite limited, including: (1) most personal exposures are so low as to be not detectable at a level of 5 parts per billion (ppb), resulting in very low correlation between concentrations reported from central ambient monitors and personal monitors; (2) O_3 measurements from ambient monitors are a better surrogate for personal exposure to $PM_{2.5}$ than to O_3; and (3) populations expected to be potentially susceptible to O_3, including children, the elderly, and those with COPD, are at the low end of the population exposure distribution (Exxon Mobil, pp. 15-16). These commenters contended that without such a correlation there is no legitimate way for EPA to conclude that O_3 exposure has caused the reported health effects, or to conclude that use of routinely monitored ambient O_3 concentrations as a surrogate for personal exposures is adequate. Some of these commenters also contended that EPA incorrectly concludes that the exposure error in epidemiological studies results in an underestimate of risk (Exxon Mobil, p. 20).

Response: With regard to the views on exposure measurement error expressed by CASAC, while the commenter is correct that the CASAC Panel raised the question of exposure error and whether observed associations could be attributed to O_3 alone, the commenter failed to note that CASAC's comment was focused on the association between O_3 and mortality, at very low O_3 concentrations and in the group of people most susceptible to premature mortality. The CASAC Panel stated:

> The population that would be expected to be potentially susceptible to dying from exposure to ozone is likely to have ozone exposures that are at the lower end of the ozone population distribution, in which case the population would be exposed to very low ozone concentrations, and especially so in winter. Therefore it seems unlikely that the observed associations between short-term ozone concentrations and daily mortality are due solely to ozone itself. (Henderson 2006b, pp. 3-4)

This section of the quote, which was not addressed in the comment submitted by API, together with the conclusions in the final CASAC letter (Henderson, 2007), leads EPA to conclude that contrary to the commenters' assertion, the CASAC Panel was not calling into question the association between O_3 exposure and the full range of morbidity effects found in panel or time-series studies that rely on ambient monitoring data as a surrogate for personal exposure data. It is important to note that EPA agrees that the evidence is only highly suggestive that O_3 directly or indirectly contributes to mortality, as compared to the stronger evidence of causality for respiratory morbidity effects.

EPA agrees that exposure measurement error may result from the use of stationary ambient monitors as an indicator of personal exposure in population studies. There is a full discussion of measurement error and its effect on the estimates of relative risk in section 7.1.3.1 of the Criteria Document. However, the possibility of measurement error does not preclude the use of ambient monitoring data as a surrogate for personal exposure data in time-series or panel studies. It simply means that in some situations where the likelihood of measurement error is greatest, effects estimates must be evaluated carefully and that caution must be used in interpreting the results from these studies. Throughout this review, EPA has recognized this concern. The Criteria Document states that there is supportive evidence that ambient O_3 concentrations from central monitors may serve as valid surrogate measures for *mean* personal O_3 exposures experienced by the population, which is of most relevance to time-series studies, in which individual variations in factors affecting exposure tend to average out across the study population. This is especially true for respiratory hospital admission studies for which much of the response is attributable to O_3 effects on asthmatics. In children, for whom asthma is more prevalent than adults, ambient monitors are more likely to correlate reasonably well with personal exposure to O_3 of ambient origin because children tend to spend more time outdoors than adults in the warm season. EPA does not agree that the correlation between personal exposure and ambient monitoring data is necessarily poor, especially in children. Moreover, the CASAC Panel supported this view as they noted that "[p]ersonal exposures most likely correlate better with central site values for those subpopulations that spend a good deal of time outdoors, which coincides, for example, with children actively engaged in outdoor activities, and which happens to be a group that the ozone risk assessment focuses upon." (Henderson, 2006c. p. 10). However, the Criteria Document notes that there is some concern in considering certain mortality and hospitalization time-series studies regarding the extent to which ambient O_3 concentrations are representative of personal O_3 exposures in another particularly susceptible group of individuals, the debilitated elderly, as the correlation between the two measurements has not been examined in this population. A better understanding of the relationship between ambient concentrations and personal exposures, as well as of the factors that affect the relationship, will improve

the interpretation of observed associations between ambient concentration and population health response.

With regard to the specific comments that reference the findings of studies by Sarnat et al. (2001, 2005, 2006) and Koutrakis et al. (2005), the fact that personal exposure monitors cannot detect O_3 levels of 5 ppb and below may in part explain why there was a poor correlation between personal exposure measurements and ambient monitoring data in the winter relative to the correlation in the warm season, along with differences in activity patterns and building ventilation. In one study conducted in Baltimore, Sarnat et al. (2001) observed that ambient O_3 concentrations showed stronger associations with personal exposure to $PM_{2.5}$ than to O_3; however, in a later study conducted in Boston (Sarnat et al., 2005), ambient O_3 concentrations and personal O_3 exposures were found to be significantly associated in the summer. Another study cited by the commenter, but not included in the Criteria Document, conducted in Steubenville (Sarnat et al., 2006), also observed significant associations between ambient O_3 concentrations and personal O_3. The authors noted that the city-specific discrepancy in the results may be attributable to differences in ventilation. Though the studies by Sarnat et al. (2001, 2005, and 2006) included senior citizens, the study selection criteria required them to be nonsmoking and physically healthy. EPA is not relying on studies that are not in the Criteria Document, such as Sarnat et al. (2006), to refute the commenters. However, EPA notes that Sarnat et al. (2006) does not support the conclusion drawn by the commenters that this study shows very limited associations between ambient O_3 concentrations and personal exposures.

Existing epidemiologic models may not fully take into consideration all the biologically relevant exposure history or reflect the complexities of all the underlying biological processes. Using ambient concentrations to determine exposure generally overestimates true personal O_3 exposures (by approximately 2- to 4- fold in the various studies described in the Criteria Document, section 3.9), which assuming the relationship is causal, would result in biased descriptions of underlying concentration-response relationships (i.e., in attenuated effect estimates). From this perspective, the implication is that the effects being estimated in relationship to ambient levels occur at fairly low personal exposures and the potency of O_3 is greater than these effect estimates indicate. On the other hand, 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 (Criteria Document, pp. 7-8 to 7-10). Nonetheless, as noted in section II.C.3 of the proposal, 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 risk estimates derived using ambient O_3 concentrations from currently available observational studies, with appropriate caveats about personal exposure considerations, remain useful. (72 FR 37839)

(9) *Comment:* NAM contends that EPA characterizes a study as reporting something when it reports the opposite, specifically referring to the work by Sarnat and colleagues (2001, 2005) (NAM, pp. 22-23) NAM states that in Sarnat et al. (2001), "the authors concluded

that ambient concentrations did not provide a surrogate for personal exposures, and in 2005 they reconfirmed that result" (NAM, p. 22). NAM then contends that EPA has "the study sites reversed and the results reported incorrectly" (NAM, p. 23).

Response: EPA has correctly characterized the conclusions of Sarnat et al. (2001) while NAM has the study sites reversed. Sarnat et al. (2001) was conducted in Baltimore, MD while the 2005 study was conducted in Boston, MA. As reported in the Criteria Document, in the study conducted in Baltimore, Sarnat et al. (2001) observed no relationship between ambient O_3 concentrations and personal O_3 exposures in both the summer and winter. The ambient O_3 concentrations showed stronger associations with personal exposure to $PM_{2.5}$ than to O_3; thus authors noted that "ambient concentrations of gaseous pollutants cannot be considered as surrogates for their respective personal exposure without site-specific evidence to support that assumption." They also noted, however, that a limitation of their study was that it was only conducted in one city. In the later study conducted in Boston (Sarnat et al., 2005), ambient O_3 concentrations and personal O_3 exposures were found to be significantly associated in the summer. From this they noted that these results suggest that "there may be differences, by location in the strength of the personal-ambient association for the gases." and that "it is incorrect to assume that ambient gas measurements are consistent surrogates for PM exposures."

(10) *Comment:* Many commenters, mostly representing industry associations and some businesses opposed to revising the primary O_3 standard, argued that known confounders are inadequately controlled in the epidemiological studies of O_3 and various health outcomes and that the health effects of O_3 are often not statistically significant when epidemiological studies consider the effects of confounding air pollutants (e.g., $PM_{2.5}$, CO, nitrogen dioxide (NO_2) in multi-pollutant models. Many commenters cited Mortimer et al. (2002), a large multi-city asthma panel study as an example, and indicated that it found that when other pollutants, i.e., sulfur dioxide (SO_2), NO_2, and particles with an aerodynamic diameter less than or equal to a nominal 10 micrometers (PM_{10}), were placed in a multi-pollutant model with O_3, the O_3-related associations with respiratory symptoms and lung function became non-significant.

Response: EPA has thoroughly reviewed issues related to confounding and the evidence of potential confounding by copollutants in sections 7.1.3 and 7.6.4 of the Criteria Document. EPA recognizes that a major methodological issue affecting O_3 epidemiologic studies concerns the evaluation of the extent to which other air pollutants may confound or modify O_3-related effect estimates, and that the changing relationship between O_3 and copollutants across seasons further complicates the issue. The use of multipollutant regression models is the prevailing approach for controlling potential confounding by copollutants in O_3 health effects studies (Criteria Document, p. 7-24). In section 7.6.4.2, the Criteria Document reviews the evidence from studies that use multipollutant models to evaluate confounding by copollutants for effects ranging from mortality and respiratory hospitalizations to lung function measures and symptoms. It concluded that multipollutant regression analyses indicated that O_3 risk estimates, in general, were not sensitive to the inclusion of copollutants, including $PM_{2.5}$ and sulfate. As can be seen in the Figure 1 below (Figure 7-22 from the Criteria Document) the ozone

effect estimates for mortality are generally unchanged upon inclusion of PM in the models. These results suggest that the effect of O_3 on respiratory health outcomes appears to be robust and independent of the effects of other copollutants (Criteria Document, p. 7-154).

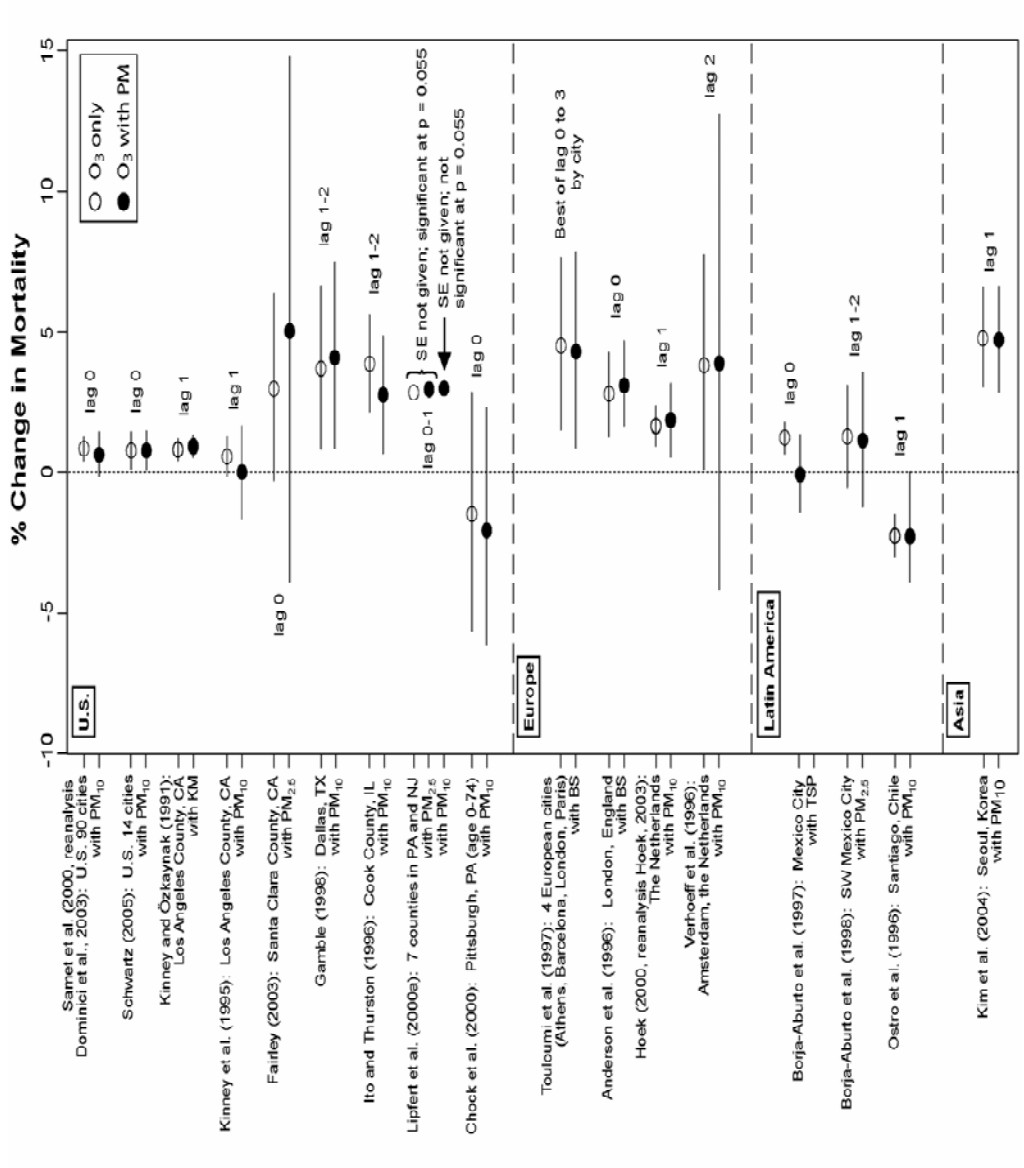

Figure 1. All-cause (nonaccidental) O_3 excess mortality risk estimates (95% CI) with adjustment for PM indices for all-year analyses per standardized increment (see Section 7.1.3.2). Analyses include all ages unless otherwise noted.

The National Cooperative Inner-City Asthma Study (Mortimer et al., 2002) evaluated air pollution health effects in 846 asthmatic children in 8 urban areas. The pollutants evaluated included O_3, PM_{10}, SO_2, and NO_2. Three effects were evaluated: (1) daily percent change in lung function, measured as peak expiratory flow rate (PEFR); (2) incidence of ≥ 10% reduction in lung function (PEFR); and, (3) incidence of symptoms

(i.e., cough, chest tightness, and wheeze). EPA notes that in this study, O_3 was the only pollutant associated with reduction in lung function. Nitrogen dioxide had the strongest effect on morning symptoms, and the authors concluded it "…may be a better marker for the summer-pollutant mix in these cities" but had no association with morning lung function. In a two-pollutant model with NO_2, the O_3 effect on morning symptoms remained relatively unchanged. Sulfur dioxide had statistically significant effects on morning symptoms but no association with morning lung function. Particulate matter (PM_{10}), which was measured daily in 3 cities, had no statistically significant effect on morning lung function. In a two-pollutant model with O_3, the PM_{10} estimate for morning symptoms was slightly reduced and there was a larger reduction in the O_3 estimate, which remained positive but not statistically significant.

(11) *Comment:* NAM contends that EPA did not consider non-air pollutant confounders that are not adequately controlled in the literature (NAM, pp. 32-33). NAM cites a number of factors that have been reported to be associated with asthma, and argues that failure to control for all of these factors "yields upwardly biased estimates of risk." (NAM, p. 32).

Response: Contrary to NAM's contention, EPA thoroughly reviewed the issues related to confounding and the evidence of potential confounding in the Criteria Document. A number of the factors listed by NAM would not appropriately be identified as potential confounders for a relationship between O_3 and health outcomes. For example, the NAM comment discusses cockroach and house dust mite allergens and exercise, none of which would be likely to confound a relationship with O_3 in a time-series study. To be a confounder, the variable must be correlated with both the health outcome and the exposure under study. In a time-series analysis, only variables that are temporally correlated with O_3 can truly confound an O_3-health outcome relationship. It is highly unlikely that exercise behaviors are correlated with O_3 concentrations – that people choose to exercise when O_3 concentrations are highest. It is known that house dust mite and cockroach allergen concentrations in a home do not vary from day to day as do ambient O_3 concentrations. Thus, these factors may be independently associated with exacerbation of asthma, but would not be confounders in a relationship between ozone and asthma exacerbation.

NAM also observes that control for meteorological variables is important. EPA agrees, and carefully evaluated the potential for confounding by temperature and humidity, as discussed in section 7.1.3.4 and further in section 7.6.3 of the Criteria Document. EPA concluded that O_3 risk estimates were generally more sensitive to alternative weather models than to varying degrees of freedom for temporal trend adjustment. In addition, careful consideration was given to whether studies had considered seasonality, as many epidemiologic studies observed differences in O_3-related health effects in the warm versus cool season. Seasonality influences the relationship between O_3 and health outcomes, as it may serve as an indicator for time-varying factors, such as temperature, copollutant concentrations, infiltration, and human activity patterns. Given the potentially significant influence of season, EPA noted that season-specific analyses were more informative in assessing O_3-related health risks and only estimated health risks for the O3 warm season in its health risk assessment.

The Administrator acknowledges that uncertainties concerning other potential confounders may be an important source of uncertainty affecting the specific risk estimates included in EPA's risk assessment and that these quantitative risk estimates must be used with appropriate caution, keeping in mind these important uncertainties. As discussed in the preamble to the final rule, the Administrator is not relying on any specific quantitative effect estimates from the time-series studies or any risk estimates based on the time-series studies in reaching his judgment about the need to revise the current 8-hour O_3 standard.

(12) *Comment:* Commenters who did not support revision of the primary O_3 standard raised issues regarding the adequacy of model specification including control of temporal and weather variables in the time-series epidemiological studies that EPA has claimed support the finding of O_3-related morbidity and mortality health outcomes. Specifically, concerns were expressed regarding the following issues: (1) commenters noted that recent meta-analyses have confirmed the important effects of model selection in the results of the time-series studies, including the choice of models to address weather and the degree of smoothing, in direct contradiction of the Staff Paper's conclusion on the robustness of the models used in the O_3 time-series studies (Exxon Mobil, p. 41); (2) commenters contended that there were no criteria for how confounders such as temperature or other factors were to be addressed, resulting in arbitrary model selection potentially impacting the resulting effect estimates; and (3) commenters expressed the view that to appropriately address concerns about model selection in the O_3 time-series studies, EPA should rely on an alternative statistical approach, Bayesian model averaging, that incorporates a range of models addressing confounding variables, pollutants, and lags rather than a single model.

Response: In response to the first issue, EPA agrees that the results of the meta-analyses do support the conclusion that there are important effects of model selection and that, for example, alternative models to address weather might make a difference of a factor of two in the effect estimates. However, as noted in the Criteria Document, one of the meta-analyses (Ito et al., 2005) suggested that the stringent weather model used in the Bell et al. (2004) NMMAPS study may tend to yield smaller effect estimates than those used in other studies (Criteria Document, p. 7-96), and, thus concerns about appropriate choice of models could result in either higher or lower effect estimates than reported. In addressing this issue, the Criteria Document concluded,

> Considering the wide variability in possible study designs and statistical model specification choices, the reported O_3 risk estimates for the various health outcomes are in reasonably good agreement. In the case of O_3-mortality time-series studies, combinations of choices in model specifications … alone may explain the extent of difference in O_3 risk estimates across studies. (Criteria Document, p. 7-174)

Second, the issues surrounding sensitivity to model specifications were thoroughly discussed in the Criteria Document (see section 7.1.3.6) and evaluated in some of the

meta-analyses reviewed in the Criteria Document and Staff Paper. As stated in the Criteria Document, O_3 effect estimates "were generally more sensitive to alternative weather models than to varying degrees of freedom for temporal trend adjustment" (Criteria Document, p. 7-176). The Criteria Document also concluded that "although there is some concern regarding the use of multipollutant models … results generally suggest that the inclusion of copollutants into the models do not substantially affect O_3 risk estimates" and the results of the time-series studies are "robust and independent of the effects of other copollutants" (Criteria Document, p. 7-177). Overall, EPA continues to believe that based on its integrated assessment, the time-series studies provide strong support for concluding there are O_3-related morbidity effects, including respiratory-related hospital admissions and emergency department visits during the warm season and that the time-series studies provide findings that are highly suggestive that short-term O_3 exposure directly or indirectly contributes to non-accidental and cardiorespiratory-related mortality. The Administrator acknowledges that uncertainties concerning appropriate model selection are an important source of uncertainty affecting the specific risk estimates included in EPA's risk assessment and that these quantitative risk estimates must be used with appropriate caution, keeping in mind these important uncertainties. As discussed in the preamble to the final rule, the Administrator is not relying on any specific quantitative effect estimates from the time-series studies or any risk estimates based on the time-series studies in reaching his judgment about the need to revise the current 8-hour O_3 standard or on the appropriate level of the standard.

Third, in response to commenters who suggested that EPA adopt an alternative statistical approach, i.e., Bayesian model averaging, to address concerns about potential arbitrary selection of models, the Criteria Document evaluated the strengths and weaknesses of such methods in the context of air pollution epidemiology. The Criteria Document noted several limitations, especially where there are many interaction terms and meteorological variables and where variables are highly correlated, as is the case for air pollution studies, which makes it very difficult to interpret the results using this alternative approach. EPA believes further research is needed to address concerns about model selection and to develop appropriate methods addressing these concerns.

(13) *Comment:* NAM asserts that EPA selected models known to yield upwardly biased risk estimates, such as single-pollutant models and models that do not control for known confounders (NAM, pp. 31-32, 34). NAM also criticizes the use of single-pollutant model results in EPA's risk assessment, which is addressed in comment numbers (7) and (9) in section II.A.5.

Response: EPA rejects NAM's assertion that it selected models known to yield upwardly biased risk estimates, such as single-pollutant models and models that do not correct for confounders in the Criteria Document, Staff Paper, or risk assessment. EPA's approach in all of these documents was to include and discuss results from both single- and multi-pollutant models when available. EPA's Criteria Document rigorously and thoroughly evaluated the potential for confounding in O_3 epidemiological studies. EPA discussed the merits and issues with both single- and multipollutant models in section 7.1.3.5 of the Criteria Document. EPA further evaluated the potential confounding of the association

between O_3 and various health outcomes by copollutants in section 7.6.4 of the Criteria Document. EPA stated that although there was some concern regarding the use of multipollutant models given the varying concurvity across pollutants, results generally suggested that the inclusion of copollutants into the models did not substantially affect O_3 risk estimates. From these findings, EPA concluded that effects of O_3 on various health outcomes were robust and independent of the effects of other copollutants. Additional response to this comment with respect to the issue of alleged upward bias in the context of the health risk assessment is addressed in the response to comment number (7) and number (9) in section II.A.5 of this document.

(14) *Comment:* NAM also contends that EPA disseminates results from models known to yield risk estimates that are upwardly biased and more uncertain, such as Generalized Additive Models conducted with insufficient convergence criteria (NAM, pp. 36-37).

Response: NAM incorrectly states "For years EPA has relied on studies utilizing Generalized Additive Models (GAMs) and software (S-PLUS) that yielded estimates of health effects from air pollution that were upwardly biased and excessively uncertain." (p. 36). In fact, that has not been the case in recent years. NAM cites results from the Special Report prepared by the Health Effects Institute (HEI, 2003); in fact EPA has been a leader in examining this issue. Upon first learning of the questions surrounding the use of GAM, EPA funded a special workshop and supported the HEI in a project to reanalyze dozens of studies to fully investigate this issue. EPA has always carefully evaluated the methodology used in epidemiologic studies in the process of its scientific assessments. EPA discusses the issue of GAM and other methodologies in section 7.1.3.7 of the Criteria Document. The Criteria Document generally only includes results from studies that used methods other than GAM or the newly developed convergence criteria that addressed the issues found with the initial use of GAM in SPlus. Thus EPA's Criteria Document and the risk assessment *do not* rely on studies that are impacted by the "GAM issues."

(15) *Comment:* NAM contends (NAM, pp. 34-35) that for lags to be biologically plausible, at least three critical biological principles must be respected: (1) all health effects must occur after exposure; (2) for each health effect, the gradient of risk must be biologically appropriate; and (3) more severe health effects must occur subsequent to minor effects. NAM contends that the time-series studies EPA relies upon do not respect these fundamental biological requirements. NAM specifically notes that biologically implausible lags are reported by Mortimer et al. (2002).

Response: EPA disagrees with NAM's charge that lags for specific health effects have been selected based on statistical strength without regard for the underlying biology. All the epidemiologic studies included in the Criteria Document examined O_3 health effects associated with exposure from the same day or previous days; thus, in these studies the health effect did not precede the exposure. In section 7.1.3.3 of the Criteria Document, EPA recognized that analyzing a large number of lags and simply choosing the largest and most significant results may bias the air pollution risk estimates away from the null. However, most time-series analyses have shown that O_3 has a fairly consistent,

immediate effect on emergency department visits, hospitalizations, and mortality. For the respiratory and cardiovascular outcomes investigated, the "most significant" lags were generally 0- or 1-day lags, suggesting that the majority of the single-day associations were immediate, not a random pattern in which associations could be observed on any of the lags examined with equal probabilities.

EPA further recognized that effects can occur acutely with exposure on the same or previous day, cumulatively over several days, or after a delayed period of a few days. Several studies also observed significant O_3 effects over longer cumulative lag periods, suggesting that in addition to single-day lags, multiday lags should be investigated to fully capture a delayed O_3 effect on health outcomes. Due to these findings, in the Criteria Document, discussion largely focused on effect estimates from same day and previous day exposures, with some consideration of cumulative, multiday lag effects.

NAM specifically uses Mortimer et al. (2002) as an example to note that multiple lag times are used without biological justification. As discussed briefly in response to a previous comment #(7), Mortimer et al. examined the association between O_3 and respiratory effects using single day lags from lag day 1 to lag day 6. Small morning effects were observed at a 1-day lag. The effect of O_3 on morning outcomes increased over several days. A single-day lag model calculates a risk estimate that assumes dependence only on exposure from the specified day. In contrast, a multiday lag model provides an estimate that is a summary measure of the cumulative lag effect from all included lag days. The effect estimates were not all statistically significant for each single-day lag, but the pattern of the effect estimates was generally consistent with considerable overlap in the confidence intervals for the multiple days considered, suggesting that the gradient of risk was, in general, biologically appropriate. Examination of these single lag day effects led to the consideration of a multiday lag period of 1 to 5 days in the case of PEF and 1 to 4 days in the case of respiratory symptoms to estimate the cumulative effect of O_3. The selected cumulative lag period was biologically plausible and consistent with previous findings that asthma exacerbation is generally associated with pollutant exposures over a several day period. The observation of greater effects in the morning and the change in effects following adjustment for copollutants were addressed in responses to previous comments (see comments #(16) and #(10) in this section).

NAM also comments that more severe health effects must occur subsequent to more minor effects for a lag to be biologically plausible. NAM notes that respiratory symptoms should occur before emergency department visits and hospital admissions. Though not specified in the comments, NAM seems to be implying that the longer cumulative lag observed for respiratory symptoms in the Mortimer et al. study compared to the shorter, more immediate lags observed in the time-series analyses of emergency department visits and hospital admissions indicate that the observed association between respiratory health outcomes and O_3 is biologically implausible. EPA disagrees that a direct comparison can be made in the exposure lags between these two very different types of studies. Mortimer et al. is a panel study of asthmatic children, in which individual-level health outcome data is linked with ambient O_3 concentrations for a

limited number of subjects. In contrast, time-series analyses link daily community-level health outcome data (i.e., counts of emergency department visits or hospitalizations) with ambient concentrations. By design, a population with a much wider variability in susceptibility is considered in these time-series analyses. It is important to note that while several studies consistently observed associations between O_3 and respiratory emergency department visits and hospitalizations at lag 0- or 1-day, strong associations were also observed in studies that examined longer multiday, cumulative exposures. In addition, it should be noted that there are potentially different mechanisms between the different endpoints described above. For example, asthmatics may experience severe acute exacerbations that result in emergency department visits, or they may have inflammatory responses that develop over a longer period of time. It is well known that asthma is a complex disease likely to have multiple etiologic pathways for health responses.

(16) *Comment:* NAM contends that methodological errors, e.g., repeated statistical tests, biologically implausible lags, and inadequate control for confounders, have not been properly considered by EPA in the evaluation of the evidence for O_3 health effects (NAM, pp. 14-16).

Response: In the Criteria Document, EPA conducted a rigorous assessment of potential methodological error in epidemiologic analyses, as can be seen in section 7.1.3. Issues such as those cited by NAM, including control for copollutants and other potential confounders, consideration of exposure lag periods, and measurement error, were evaluated by EPA in its assessment of the scientific evidence. EPA does not agree that methodological errors exist in these studies that are "so severe that they have a material effect on utility, particularly for regulatory decision-making." (p. 14).

With regard to NAM's statement that repeated statistical tests are performed without apparent regard for the resulting increase in the rate of false positive, EPA discusses the issue of multiple hypothesis testing in Section 7.1.3.6 of the Criteria Document. In particular, EPA notes that multiple hypotheses may need to be developed for researchers to explore more thoroughly potential associations for an O_3-related health effect. NAM specifically cites Korrick et al. (1998) and Mortimer et al. (2002) in reference to this comment. In Korrick et al. (1998), though multiple hypotheses are tested, these hypotheses can be divided into confirmatory vs exploratory hypotheses. The main confirmatory hypothesis is whether O_3 concentrations are associated with pulmonary function. In their models adjusting for multiple covariates, they observe a clear inverse association between O_3 and FEV_1 and FVC. In their sensitivity or exploratory analyses, they observe that the O_3 effect estimate is generally robust to adjustment for $PM_{2.5}$ and acidity. They further observed that the effect of O_3 may be modified by asthma status, but not by smoking status, gender, or age. In Mortimer et al. (2002) the issue pertains to the examination of multiple lags. As discussed above, the examination of multiple lags may be viewed as the testing of exploratory hypotheses. As noted in the response to comment (15) above, in section 7.1.3.3 of the Criteria Document, EPA recognized that analyzing a large number of lags and simply choosing the largest and most significant results may bias the air pollution risk estimates away from the null. In the response

above and in the response to comment (3) the issue of lags is further discussed specifically with regard to Mortimer et al. (2002)

Other methodological errors that NAM mention include the reliability of self-administered PEF testing (Mortimer et al., 2002), selective use of data from different times of day (Mortimer et al., 2002), reliance on subjective symptoms rather than objective signs (Gent et al., 2003), and the use of a nonstandard measure of incremental change (Korrick et al., 1998).

One issue that NAM discusses in some detail in this section is the error related to self-reported peak expiratory flow data. NAM presents the results of Kamps et al. (2001) which found that compliance and reliability of self-reported data was less than optimal. EPA recognizes that PEF measurements have been shown to be more variable than FEV_1 in some studies (Vaughan et al., 1989; Cross and Nelson, 1991) and can have an element of uncertain reliability when self-administered by study subjects. As increased variability in the PEF measurement will add noise to the relationship between O_3 and PEF, it will likely lead to bias towards the null rather than a spurious significant association. Furthermore, the observed association between O_3 and PEF concur with the strong associations found between O_3 and measures of spirometry, which is a more consistent test of lung function and typically conducted by a trained individual.

NAM also comments on the selective use of data from different times of day. In Mortimer et al. (2002), while associations were found between O_3 and morning PEF and respiratory symptoms, no associations were observed with evening PEF or respiratory symptoms. The fact that associations were more evident with asthma symptoms measured in the morning is consistent with the understanding that the development of asthma exacerbation through an inflammatory mechanism would occur over time, with symptoms manifested hours after the exposure period. Mortimer et al. further explain that the most severe bronchoconstriction occurs in the morning, when measurable differences between and within individuals may be greatest. Therefore, there are biologically plausible reasons as to why associations would be observed at different times of day.

The NAM statement that Gent et al. (2003) relied on a subjective measure of symptoms rather than an objective measure is incorrect. In addition to respiratory symptoms, Gent et al. also observed an association between O_3 and rescue medication use, which is an objective measure. Regardless, EPA deems respiratory symptoms to be a valuable health outcome, which considered in conjunction with various other more "objective" measures, allows a more complete depiction of the potential respiratory health effects of pollutants.

NAM further contends that Korrick et al. (1998) uses a nonstandard measure of incremental change (% per 50-100 ppb O_3) that is too large to have any practical utility for making policy decisions over small exposure ranges. EPA disagrees with this contention as these effect estimates can be standardized to any incremental change based on the linear concentration-response function. Discussion of this approach can be found in section 7.1.3.2 of the Criteria Document (Criteria Document, p. 7-10).

(17) *Comment:* With regard to evidence of O_3-related mortality, many commenters, including those that argued for revising the current O_3 standard as well as those that argued against revisions, focused on the new evidence from multi-city time-series analyses and meta-analyses linking O_3 exposure with mortality. Again, sharply divergent comments were received. One group of commenters, including medical, public health, and environmental organizations argued that recent published research has provided more robust, consistent evidence linking O_3 to cardiovascular and respiratory mortality. The ATS, AMA, and others stated that data from single-city studies, multiple-city studies, and meta-analyses show a consistent relationship between O_3 exposure and mortality from respiratory and cardiovascular causes. These commenters noted that this effect was observed after controlling for co-pollutants and seasonal impacts. These commenters stated that research has demonstrated that exposure to O_3 pollution is causing premature deaths, and has also provided clues on the possible mechanisms that lead to premature mortality (ATS, p. 4). These commenters noted that people may die from O_3 exposure even when the concentrations are well below the current standard. They pointed to a study (Bell et al., 2006) in which the authors followed up on their 2004 multi-city study to estimate the exposure-response curve for O_3 and the risk of mortality and to evaluate whether a threshold exists below which there is no effect. The authors applied several statistical models to data on air pollution, weather, and mortality for 98 U.S. urban communities for the period 1987 to 2000. The study reported that O_3 and mortality results did not appear to be confounded by temperature or PM and showed that any threshold, if it existed, would have to be at very low concentrations, far below the current standard (ALA et al., p. 74). Another approach also indicated that the mortality effect is unlikely to be confounded by temperature. A case-crossover study (Schwartz 2005) of over one million deaths in 14 U.S. cities, designed to control for the effect of temperature on daily deaths attributable to O_3, found that the association between O_3 and mortality risk reported in the multi-city studies is unlikely to be due to confounding by temperature (ALA et al., p. 76). These commenters argue that meta-analyses also provide compelling evidence that the O_3-mortality findings are consistent. They point to three independent analyses conducted by separate research groups at Johns Hopkins University, Harvard University and New York University, using their own methods and study criteria, which reported a remarkably consistent link between daily O_3 levels and total mortality.

Response: In response, EPA notes that the Criteria Document states that the results from the 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). The Criteria Document concludes that these findings are highly suggestive that short-term O_3 exposure directly or indirectly contributes to non-accidental and cardiorespiratory-related mortality but that additional research is needed to more fully establish the underlying mechanisms by which such effects occur (72 FR 37836). Thus while EPA generally agrees with the direction of the comment, EPA believes the evidence supports a view as noted above. In addition, it must be noted that the Administrator did not focus on mortality as a basis for proposing that the current O_3

standard was not adequate or on determining the appropriate level of the standard. In the proposal, the Administrator focused on the very strong evidence of respiratory morbidity effects in healthy people at the 0.080 ppm exposure level and new evidence that people with asthma are likely to experience larger and more serious effects than healthy people at the same level of exposure and evidence that epidemiological studies indicated an association with health effects at the current level of the standard (72 FR 37870). With regard to the ambient concentrations at which O_3-related mortality effects may be occurring, EPA recognized in the proposal that evidence of a causal relationship between adverse health effects and O_3 exposures becomes increasingly uncertain at lower levels of exposure (72 FR 37880). This is discussed more fully in other sections.

(18) *Comment:* Another group of commenters, including several industry organizations argued against placing any reliance on the time-series epidemiological studies, especially those studies related to mortality effects. The Annapolis Center (p. 46) makes the point that although there may be somewhat more positive associations than negative associations, there is so much noise or variability in the data that identifying which positive associations may be real health effects and which are not is beyond the capability of current methods. They cite the view that the CASAC Panel expressed in a June 2006 letter (Henderson, 2006b), noting that "Because results of time-series studies implicate all of the criteria pollutants, findings of mortality time-series studies do not seem to allow us to confidently attribute observed effects specifically to individual pollutants."

Several of these commenters focused on the O_3 mortality multi-city studies in particular, arguing that, although these studies have the statistical power to distinguish weak relationships between daily O_3 and mortality, they do not provide reliable or consistent evidence implicating O_3 exposures as a cause of mortality. Several reasons were given, including: (a) the multi-city studies cited by EPA involve a wide range of city-specific effects estimates, including some large cities that have very slight or negligible effects (e.g., Los Angeles) (Bell et al., 2004), thus causing several commenters to question the relevance of a "national" effect of O_3 on mortality and argue that a single national O_3 concentration-mortality coefficient should be used and interpreted with caution (Rochester Report p. 4); (b) the multi-city mortality studies did not sufficiently account for other pollutants, for example, Bell et al. (2004) adjusted for PM_{10} but did not have the necessary air quality data to adequately adjust for $PM_{2.5}$, which EPA has concluded also causes mortality and is correlated with O_3, especially in the summer months (Annapolis Center, p. 42); and (c) these studies contain several findings that are inconsistent or implausible, such as premature mortality reported at such low levels as to imply that O_3-related mortality is occurring at levels well within natural background, which is not biologically plausible (Annapolis Center, p. 42).

Response: Evidence supporting an association between short-term O_3 exposure and premature mortality is not limited to multi-city time-series studies. Most single-city studies show elevated risk of total, non-accidental mortality, cardiorespiratory, and respiratory mortality (> 20 studies), including one study in an area that would have met current standard (Vedal et al., 2003). Three large meta-analyses, which pool data from many single-city studies to increase statistical power, reported statistically significant associations and examined sources of heterogeneity in those associations (Bell et al.,

2005; Ito et al., 2005; Levy et al. 2005). These studies found: (1) larger and more significant effects in the warm season than in the cool season or all year; (2) no strong evidence of confounding by PM; and (3) suggestive evidence of publication bias, but significant associations remain even after adjustment for the publication bias.

In the letter cited, the CASAC Panel did raise the issue of the utility of time-series studies in the standard setting process with regard to time-series mortality studies. Nevertheless, in a subsequent letter to the Administrator, CASAC noted these mortality studies as evidence to support a recommendation to revise the current primary O_3 standard. "Several new single-city studies and large multi-city studies designed specifically to examine the effects of ozone and other pollutants on both morbidity and mortality have provided more evidence for adverse health effects at concentrations lower than the current standard (Henderson, 2006c, p. 3)."

With regard to the specific issues raised in the comments as to why the times-series mortality studies do not provide reliable or consistent evidence implicating O_3 exposure as a cause of mortality, EPA has the following responses:

(1) The purpose of the NMMAPS approach is not to single out individual city results but rather to estimate the overall effect from the 95 communities. It was designed to provide a general, nationwide estimate. With regard to the very slight or negligible effects estimates for some large cities (e.g., Los Angeles), an important factor to consider is that the Bell et al. (2004) study used all available data in their analyses. Bell et al. reported that the effect estimate for all available (including 55 cities with all year data) and warm season (April-October) analyses for the 95 U.S. cities were similar in magnitude; however, in most other studies, larger excess mortality risks were reported in the summer season (generally June-August when O_3 concentrations are the highest) compared to all year or the cold season. Though the effect estimate for Los Angeles is small compared to some of the other communities, it should be noted that all year data (combined warm and cool seasons) was used in the analyses for this city, which likely resulted in a smaller effect estimate. Because all year data was used for Los Angeles, the median O_3 concentration for Los Angeles is fairly low compared to the other communities, ranked 23rd out of 95 communities. The median 24-hour average O_3 concentration for Los Angeles in this dataset was 22 ppb, with a 10^{th} percentile of 8 ppb to a 90^{th} percentile of 38 ppb. The importance of seasonal differences in O_3-related health outcomes has been well documented.

(2) In section 7.4.6, O_3 mortality risk estimates adjusting for PM exposure, the Criteria Document states that the main confounders of interest for O_3, especially for the northeast U.S., are "summer haze-type" pollutants such as acid aerosols and sulfates. Since very few studies included these chemical measurements, PM (especially $PM_{2.5}$) data, may serve as surrogates. However, due to the expected high correlation among the constituents of the "summer haze mix," multipollutant models including these pollutants may result in unstable coefficients; and, therefore, interpretation of such results requires some caution.

In section 7.4.6 of the Criteria Document, Figure 7-22 shows the O_3 risk estimates with and without adjustment for PM indices using all-year data in studies that conducted two-pollutant analyses. Approximately half of the O_3 risk estimates increased slightly, whereas the other half decreased slightly with the inclusion of PM in the models. In general, the O_3 mortality risk estimates were robust to adjustment for PM in the models.

The U.S. 95 communities study by Bell et al. (2004) examined the sensitivity of acute O_3-mortality effects to potential confounding by PM_{10}. Restricting analysis to days when both O_3 and PM_{10} data were available, the community-specific O_3-mortality effect estimates as well as the national average results indicated that O_3 was robust to adjustment for PM_{10} (Bell et al., 2004). As commenters noted, there were insufficient data available to examine potential confounding by $PM_{2.5}$. One study (Lipfert et al., 2000) reported O_3 risk estimates with and without adjustment for sulfate, a component of $PM_{2.5}$. Lipfert et al. (2000a) calculated O_3 risk estimates based on mean (45 ppb) less background (not stated) levels of 1-hour max O_3 in seven counties in Pennsylvania and New Jersey. The O_3 risk estimate was not substantially affected by the addition of sulfate in the model (3.2% versus 3.0% with sulfate) and remained statistically significant.

Several O_3 mortality studies examined the effect of confounding by PM indices in different seasons (Figure 7-23, section 7.4.6, Criteria Document). In analyses using all-year data and warm-season only data, O_3 risk estimates were once again fairly robust to adjustment for PM indices, with values showing both slight increases and decreases with the inclusion of PM in the model. In the analyses using cool season data only, the O_3 risk estimates all increased slightly with the adjustment of PM indices, although none reached statistical significance.

The three recent meta-analyses (Bell et al., 2005; Ito et al., 2005; Levy et al. 2005) all examined the influence of PM on O_3 risk estimates. No substantial influence was observed in any of these studies. In the analysis by Bell et al. (2005), the combined estimate without PM adjustment was 1.75% (95% PI: 1.10, 2.37) from 41 estimates, and the combined estimate with PM adjustment was 1.95% (95% PI: -0.06, 4.00) from 11 estimates per 20 ppb increase in 24-hour average O_3. In the meta-analysis of 15 cities by Ito et al. (2005), the combined estimate was 1.6% (95% CI: 1.1, 2.2) and 1.5% (95% CI: 0.8, 2.2) per 20 ppb in 24-hour average O_3 without and with PM adjustment, respectively. The additional time-series analysis of six cities by Ito et al. found that the influence of PM by season varied across alternative weather models but was never substantial. Levy et al. (2005) examined the regression relationships between O_3 and PM indices (PM_{10} and $PM_{2.5}$) with O_3-mortality effect estimates for all year and by season. Positive slopes, which might indicate potential confounding, were observed for $PM_{2.5}$ on O_3 risk estimates in the summer and all-year periods, but the relationships were weak. The effect of one causal variable (i.e., O_3) is expected to be overestimated when a second causal variable (e.g., PM) is excluded from the analysis, if the two variables are positively correlated and act in the same direction. However, EPA notes that the results from these meta-analyses, as well as several single- and multiple-city studies, indicate that copollutants, including PM, generally do not appear to substantially confound the association between O_3 and mortality.

(3) Moreover, EPA asserts that the biological plausibility of the epidemiological mortality associations is generally supported by controlled human exposure and toxicological evidence of respiratory morbidity effects for levels at and below 0.080 ppm, but that biological plausibility becomes increasingly uncertain especially below 0.060 ppm, the lowest level at which effects were observed in controlled human exposure studies. Further, at lower levels, it becomes increasingly uncertain as to whether the reported associations are related to O_3 alone rather than to the broader mix of air pollutants present in the ambient air. EPA notes that the multi-city times series studies evaluated in this review can not resolve this issue. It also becomes increasingly uncertain as to whether effect thresholds exist but can not be clearly discerned by statistical analyses. Thus, when considering the epidemiological evidence in light of the other available information, it is reasonable to judge that at some point the epidemiological associations can not be interpreted with confidence as providing evidence that the observed health effects can be attributed to O_3 alone.

(19) *Comment:* NAM contends that EPA assumes that associations observed in time-series studies are significant but the absence of associations in long-term cohort studies is not. NAM states that EPA arrives at the conclusion that "ozone causes premature mortality in the short-term that cannot be observed over the long-term" by "leap of faith, not scientific inference" (NAM, p.50)

Response: EPA rejects NAM's contention that it has reached inappropriate conclusions about associations between O_3 exposure and premature mortality. In the Criteria Document, EPA presented numerous recent epidemiologic studies conducted in the United States and abroad which have investigated the association between short-term exposure to O_3 and mortality. Results from several large U.S. multicity studies as well as several single-city studies indicated a positive association between increases in ambient O_3 levels and excess risk of all-cause (nonaccidental) daily mortality. Newly available experimental data from both animal and human studies provide evidence suggestive of plausible pathways by which risk of respiratory or cardiovascular morbidity and mortality could be increased by ambient O_3 either acting alone or in combination with copollutants in ambient air mixes. These overall findings led EPA to conclude that there is highly suggestive evidence that O_3 directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality, but additional research is needed to more fully establish underlying mechanisms by which such effects occur. As few epidemiologic studies were available on long-term exposure to O_3 and mortality, and the results from these studies were not consistent, EPA further concluded that there was insufficient evidence to suggest a causal relationship between long-term O_3 exposure and increased risk for mortality in humans.

Kunzli et al. (2001) state that air pollution may play a role both in increasing the decedent's underlying susceptibility or frailty and in triggering the event. In another case, the underlying frailty may be related to long term air pollution, but the event or the occurrence of death itself is unrelated to the levels of air pollution shortly before death. In the third case, reduced health status or frailty is not related to air pollution, but ambient

air pollution experienced before death may trigger the terminal event. Thus, mortality from long-term exposure to a pollutant is not simply the accumulation of mortality from short-term exposures. Different mechanisms are at play for mortality associated with short-term versus long-term exposures; therefore, the inability to assess causality of the effect of long-term O_3 exposure on mortality does not have bearing on the EPA's finding that there is highly suggestive evidence that short-term exposure to O_3 contributes to mortality.

Further, in a letter to the Administrator, CASAC noted these mortality studies as evidence to support a recommendation to revise the current primary O_3 standard. "Several new single-city studies and large multi-city studies designed specifically to examine the effects of ozone and other pollutants on both morbidity and mortality have provided more evidence for adverse health effects at concentrations lower than the current standard" (Henderson, 2006c, p. 3).

(20) *Comment:* NAM contends that EPA characterizes in the proposal notice that results from "numerous" multi-city and single-city studies show that the association between O_3 and mortality do not appear to be changed in multipollutant models including PM_{10} or $PM_{2.5}$ (72 FR 37839). NAM contends that these studies are NMMAPS studies, none of which has daily $PM_{2.5}$ data. These associations "do not appear to be changed" because they only measure PM_{10} (NAM, pp. 21).

Response: EPA disagrees with NAM's characterization of the basis for the statements made in the proposal notice with respect to what the scientific evidence shows with respect to the influence of PM in affecting the relationship observed between O_3 and mortality. NAM mistakenly states that the conclusion in the proposal notice was solely based on results of the NMMAPS; in fact, it is based on the results of many other studies as presented in Figures 7-22 and 7-23 in section 7.4.6 of the Criteria Document. The studies used different PM indices, including $PM_{2.5}$, PM_{10}, BS, as well as TSP, to adjust for potential confounding by PM of the O_3 relationship with mortality. Furthermore, while not relying on "new" studies published since completion of the Criteria Document, EPA notes that specifically in response to the use of PM_{10} by NMMAPS, a recently published study (not included in the Criteria Document) by Bell et al. (2007) reported that the national and community-specific effect estimates of the short-term effects of O_3 on mortality were robust to inclusion of PM_{10} or $PM_{2.5}$ in time-series models.

(21) *Comment:* NAM contends that EPA has misinterpreted two epidemiologic studies by Moolgavkar and colleagues (1995; 2000) in EPA's risk assessment and that EPA's interpretation is different than that of the primary author and states that "EPA represents the results of these studies in ways that the corresponding author says are says are incorrect (Moolgavkar 2007, 4-5)" (NAM, p.21).

Response: EPA disagrees with NAM's contention that it has interpreted the two cited studies by Moolgavkar differently than the primary author. Moolgavkar (2007) on pages 4 and 5 states that EPA included the positive association that he reported in his 1995 Philadelphia paper but that EPA failed to cite his 2000 study in Los Angeles. EPA notes

that there is nothing on pp. 4-5 of Moolgavkar that supports NAM's contention that EPA has interpreted these two studies in a way different than the primary author. Furthermore, there is no discussion of EPA's risk assessment or the use of Moolgavkar's studies in the risk assessment on pp.4-5 of the Moolgavkar (2007) document.

(22) *Comment:* Many commenters identified "new" studies that were not included in the Criteria Document that they stated support arguments both for and against the revision of the current O_3 standard. Commenters who supported revising the current O_3 standard identified new studies that generally supported EPA's conclusions about the associations between O_3 exposure and a range of respiratory and cardiovascular health outcomes. These commenters also identified new studies that provide evidence for associations with health outcomes that EPA has not linked to O_3 exposure, such as cancer, and populations that EPA has not identified as being susceptible or vulnerable to O_3 exposure, including African-American and women. Commenters who did not support revision of the current O_3 standard often submitted the same "new" studies, but focused on different aspects of the findings. Commenters who did not support revision of the current O_3 standard stated that these "new" studies provide inconsistent and sometimes conflicting findings that do little to resolve uncertainties regarding whether O_3 has a causal role in the reported associations with adverse health outcomes, including premature mortality and various morbidity outcomes.

Response: To the extent that these commenters included "new" scientific studies, studies that were published too late to be considered in the Criteria Document, in support of their arguments for revising or not revising the standards, EPA notes, as discussed in section II of the preamble to the final rule, that as in past NAAQS reviews, it is basing the final decisions in this review on the studies and related information included in the O_3 air quality criteria that have undergone CASAC and public review and will consider newly published studies for purposes of decision making in the next O_3 NAAQS review. In provisionally evaluating commenters' arguments, EPA notes that its provisional consideration of "new" science found that such studies did not materially change the conclusions in the Criteria Document. For more information about this provisional consideration see the Appendix.

(23) *Comment:* NAM contends that EPA displays a systematic preference for studies that show positive associations even among studies that have important information quality limitations (NAM, pp. 30-31). NAM asserts that it has been unable to identify any epidemiological studies that EPA has identified as dispositive but which did not find a positive association. NAM claims that EPA "consistently selects studies that show positive associations with ozone (e.g., Gent et al., 2003; Mortimer et al., 2002) over studies that do not (e.g., Schildcrout et al., 2006) but does not establish an information quality basis for its selections" (NAM, p. 30).

Responses: EPA rejects NAM's contention that it displays a systematic preference for studies that show positive associations. EPA has clearly presented the results of Mortimer et al. (2002) and Gent et al. (2003), including results that were not positive. EPA has described studies that do not report positive or statistically significant results.

The reason that NAM is unable to find any epidemiological studies that EPA has identified as dispositive but which did not find a positive association is that there is no single study or group of studies that is "dispositive." EPA asserts that this would be an inappropriate way to represent the entire body of evidence. EPA uses a weight of evidence approach that integrates evidence from different types of studies including: animal toxicology; controlled human exposure; and epidemiological studies.

Schildcrout et al. (2006) is not included in the Criteria Document as it was published after the date specified for inclusion (new studies accepted for publication by December 2004). In fact, it was published on-line in June of 2006, after the Criteria Document had been finalized. As noted above, EPA is basing the final decisions in this review on the studies and related information included in the O_3 air quality criteria that have undergone CASAC and public review and will consider the newly published studies for purposes of decision making in the next O_3 NAAQS review. This study has not been relied upon as it is one of the "new" studies that have been published since the cutoff date.

NAM cites only the Schildcrout study to support its claim that "EPA's risk assessment for ozone includes a large measure of study selection bias" (NAM, p. 31). In fact, clearly the converse is true. EPA's evaluation of the scientific evidence has been rigorous and thorough, and the process for selection of studies for inclusion in the assessment has been transparent and subject to review by the CASAC and the public.

c. Evidence Pertaining to At-Risk Subgroups for O_3-Related Effects

This section contains major comments on EPA's assessment of the body of evidence, including controlled human exposure and epidemiological studies, related to the effects of O_3 exposure on sensitive subpopulations. Since new information about the increased responsiveness of people with lung disease, especially children and adults with asthma, was an important consideration in the Administrator's proposed decision that the current O_3 standard is not adequate, many of the comments focused on this information and the conclusions drawn from it. There were also comments on other sensitive groups identified by EPA, as well as comments suggesting that additional groups should be considered at increased risk from O_3 exposure. As with the comments on controlled human exposure and epidemiological studies, upon which judgments about sensitive subpopulations were based, the comments about EPA's delineation of these groups were highly polarized.

(1) *Comment:* In general, one group of commenters who supported revising the current O_3 primary standard, including medical associations, public health and environmental groups, agreed in part with EPA's assessment of the subpopulations that are at increased risk from O_3 exposure, but commented that there are additional groups that need to be considered. A comment from ATS, AMA and other medical associations noted:

> Within this population exists a number of individuals uniquely at much higher risk for adverse health effects from ozone exposures, including children, people

> with respiratory illness, the elderly, outdoor workers and healthy children and adults who exercise outdoors. (ATS, p. 2)

These commenters agreed with EPA that, based on evidence from controlled human exposure and epidemiology studies, people with asthma, especially children, are likely to have greater lung function decrements and respiratory symptoms in response to O_3 exposure than people who do not have asthma, and are likely to respond at lower levels. Because of this, these commenters make the point that controlled human exposure studies that employ healthy subjects will underestimate the effects of O_3 exposures in people with asthma.

These commenters also agreed with EPA's assessment that epidemiological studies provide evidence of increased morbidity effects, including lung function decrements, respiratory symptoms, emergency department visits and hospital admissions, in people with asthma and that controlled human exposure studies provide biological plausibility for these morbidity outcomes. Further, the Rochester Report evaluated some of the same the studies that EPA did and found similar results with regard to the increased inflammatory responses and increased airway responsiveness of people with asthma when exposed to O_3. The Rochester Report reached the same conclusion that EPA did, that this increased responsiveness provides biological plausibility for the respiratory morbidity effects found in epidemiological studies.

> Several new studies have demonstrated that exposure of individuals with atopic asthma to sufficient levels of ozone produces an increase in specific airway responsiveness to inhaled allergens……These findings, in combination with previously observed effects of ozone on nonspecific airway responsiveness and airway inflammation, supports the idea that ambient ozone exposure could result in exacerbation of asthma several days following exposure, and provides biological plausibility for the epidemiologic studies in which ambient ozone concentration has been associated with increased asthma symptoms, medication use, emergency room visits, and hospitalizations for asthma. (Rochester Report, pp. 57-58)

Commenters also often mentioned the increased susceptibility of people with COPD and in this case cited "new" studies not considered in the Criteria Document.

They identify that one potentially susceptible subpopulation that EPA did not focus on in the proposal is infants. Commenters from medical associations, and environmental and public health groups expressed the view that O_3 exposure can have important effects on infants, including reduced birth weight, pre-term birth, and increased respiratory morbidity effects in infants. Exposure to O_3 during pregnancy, especially during the second and third trimesters, was associated with reduced birth weight in full-term infants. Although this effect was noted at relatively low O_3 exposure levels, the ATS notes that, "…the reduced birth weight in infants in the highest ozone exposures communities equaled the reduced birth weight observed in pregnant women who smoke" (ATS, p. 7).

Response: In general, EPA agrees with comments that there is very strong evidence from controlled human exposure and epidemiological studies that people with lung disease, especially children and adults with asthma, are susceptible to O_3 exposure and are likely to experience more serious effects than those people who do not have lung disease. This means that controlled human exposure studies that employ subjects who do not have lung disease will likely underestimate effects in those people that do have asthma or other lung diseases.

In summarizing the epidemiological evidence related to birth-related health outcomes, the Criteria Document (p. 7-133) concludes that O_3 was not an important predictor of several birth-related outcomes including premature births and low birth weight. Birth-related outcomes generally appeared to be associated with air pollutants that tend to peak in the winter and are possibly traffic-related. However, given that most of these studies did not analyze the data by season, seasonal confounding may have therefore influenced the reported associations. One study reported some results suggestive of associations between exposures to O_3 in the second month of pregnancy and birth defects, but further evaluation of such potential associations is needed. With regard to comments about effect in infants, EPA notes that some of the studies cited by commenters were "new" studies too late for inclusion in the Criteria Document and thus were not considered in the Criteria Document.

(2) *Comment:* The second group of commenters, mostly representing industry associations and some businesses opposed to revising the primary O_3 standard, asserted that EPA is wrong to claim that new evidence indicates that the current standard does not provide adequate health public health protection for people with asthma. In support of this position, these commenters made the following major comments: (1) lung function decrements and respiratory symptoms observed in controlled human exposure studies of asthmatics are not clinically important; (2) EPA postulates that asthmatics would likely experience more serious responses and responses at lower levels than the subjects of controlled human exposure experiments, but that hypothesis is not supported by scientific evidence; and, (3) EPA recognized asthmatics as a sensitive subpopulation in 1997, and new information does not suggest greater susceptibility than was previously believed.

With regard to the first point, these commenters expressed the view that asthmatics are not likely to experience medically significant lung function changes or respiratory symptoms at ambient O_3 concentrations at or even above the level of the current standard. Many of these commenters cited the opinion of one physician who was asked on behalf of a group of trade associations and companies to provide his views on the health significance for asthmatics of the types of responses that have been reported in controlled human exposure studies of O_3. This commenter (McFadden) reviewed controlled human exposure studies of asthmatics from the last review as well as the recent controlled human exposure studies of healthy individuals (Adams 2002, 2003, and 2006) at 0.12, 0.08, 0.06, and 0.04 ppm and expressed the view that "…these studies on asthmatics indicate that ozone exposures at ~ 0.12 ppm do not produce medically significant functional changes and are right around the inflection point where one begins to see an increase in symptoms; however, that increase is small" (McFadden, p. 3). This

commenter went on to express the view that responses to O_3 exposure at levels < 0 .08 ppm would be even less and that the available data are not sufficiently robust to indicate that such exposures would present a significant health concern even to sensitive people like asthmatics.

With regard to the third point, commenters note that there is no significant new evidence establishing greater risk to asthmatics than was accepted in 1997, when EPA concluded that the existing NAAQS was sufficiently stringent to protect public health – including asthmatics – with and adequate margin of safety (UARG, pp. 22-23). To support this view, these commenters note the points made above, that the effects of O_3 at and below 0.080 ppm are not clinically significant and that asthmatics do not have more serious responses and responses at lower levels. They also express the view that epidemiological studies of asthmatics that provide new evidence of respiratory symptoms and medication use in asthmatic children are subject to the limitations of epidemiological studies discussed above (e.g., confounding by co-pollutants, and heterogeneity of results). In addition, these commenters identified a new, large multi-city panel study not included in the Criteria Document, by Schildcrout et al. (2006), which reported no association between O_3 concentrations and exacerbation of asthma.

Response: With regard to the first point, EPA notes that this commenter based his comment on the group mean functional and respiratory symptom changes in the studies he reviewed. EPA agrees that group mean changes at these levels are relatively small and has described them as such in both the previous review and this one (72 FR 37828). The importance of group mean changes is to evaluate the statistical significance of the association between the exposures and the observed effects, to try to determine if the observed effects are likely due to O_3 exposure rather than chance. In the previous review as well as in this one, EPA has also focused on the fact that some individuals experience more severe effects that may be clinically significant. With regard to the significance of individual responses, this commenter (McFadden, p. 2) states "...transient decreases in FEV_1 of 10-20% are not by themselves significant or meaningful to asthmatics…. It has been my experience from examining and studying thousands of patients for both clinical and research purposes that asthmatics typically will not begin to sense bronchoconstriction until their FEV_1 falls about 50% from normal." EPA strongly disagrees with this assessment. As stated in the Criteria Document (Table 8-3, p. 8-68) for people with lung disease, even moderate functional responses (e.g., FEV_1 decrements $\geq$ 10% but < 20%) would likely interfere with normal activities for many individuals, and would likely result in more frequent medication use. EPA notes that in the context of standard setting, CASAC indicated (Henderson, 2006c) that a focus on the lower end of the range of moderate functional responses (e.g., FEV_1 decrements $\geq$ 10%) is most appropriate for estimating potentially adverse lung function decrements in people with lung disease.

With regard to the second point, whether asthmatics would likely experience more serious responses and responses at lower levels than the subjects of controlled human exposure experiments and EPA's discussion of the relationship of increased airway responsiveness and inflammation experienced by asthmatics to exacerbation of asthma,

this commenter stated that "there simply are no data to support the sequence described" and that "the assumption that these responses would lead to clinical manifestations in terms of exacerbations of asthma or other adverse health effects remains unproven theory"(McFadden, p. 3). In the proposal (72 FR 37826, and 37846-37847), EPA describes the evidence that people with asthma are as sensitive as, if not more sensitive than, normal subjects in manifesting O_3-induced pulmonary function decrements. Controlled human exposure studies show that asthmatics present a differential response profile for cellular, molecular, and biochemical parameters that are altered in response to acute O_3 exposure. Asthmatics have greater O_3-induced inflammatory responses and increased O_3-induced airway responsiveness (both incidence and duration) that could have important clinical implications.

There are two ways to interpret these comments. One way to interpret them is that because these controlled human exposure studies have not produced exacerbations of asthma in study subjects resulting in the need for medical attention, there are no data to support the clinical significance of the results. EPA rejects this interpretation because it would be unethical to knowingly conduct a controlled human exposure study that would lead to exacerbation of asthma. Controlled human exposure studies are specifically designed to avoid these types of responses. The other interpretation is that the commenter does not agree that the differences in lung function, inflammation and increased airway responsiveness found in these controlled human exposure studies support the inference that asthmatics are likely to have more serious responses than healthy subjects, and that these responses could have important clinical implications. EPA rejects this interpretation as well. EPA did not base its increased concern for asthmatics solely on the results of the controlled human exposure studies, but has appropriately used a weight of evidence approach, integrating evidence from animal toxicological, controlled human exposure and epidemiological studies as a basis for this concern. The Criteria Document evaluated a number of epidemiological studies that have been conducted using asthmatic study populations (72 FR 37847). These studies suggest that O_3 exposure may be associated with increased respiratory symptoms and medication use in children with asthma, and also respiratory symptoms, lung function decrements, emergency department visits and hospital admissions for respiratory causes and respiratory mortality. The Criteria Document concludes that the positive and robust epidemiological associations between O_3 exposure and emergency department visits and hospitalizations in the warm season are supported by the human clinical, animal toxicological and epidemiological evidence for lung function decrements, increased respiratory symptoms, airway inflammation, and increased airway responsiveness (72 FR 37832). The CASAC Panel itself expressed the view that people with asthma, especially children, have been found to be more sensitive to O_3 exposure, and indicated that EPA should place more weight on inflammatory responses and serious morbidity effects, such as increased respiratory-related emergency department visits and hospitalizations (Henderson, p. 4). Moreover, the Rochester Report, cited above, reaches essentially the same conclusions as EPA did, that the evidence from controlled human exposure studies provides biological plausibility for the epidemiological studies in which ambient O_3 concentrations have been associated with increased asthma symptoms, medication use, emergency room visits, and hospitalizations for asthma. Therefore, EPA continues to

assert that there is strong evidence that asthmatics likely have more serious responses to O_3 exposure than people without asthma, and that these responses have the potential to lead to exacerbation of asthma as indicated by the serious morbidity effects, such as increased respiratory-related emergency department visits and hospitalizations found in epidemiological studies.

With regard to the third point, at the time of the last review EPA concluded that people with asthma were 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 indicate that individuals with preexisting lung disease, especially people with asthma, are likely to have more serious responses than people who do not have lung disease and therefore are at greater risk for O_3 health effects than previously judged in the 1997 review. EPA notes that comments on the limitations of epidemiological studies and the evidence from "new" studies (not in the Criteria Document) have been addressed above.

d. Adversity of Health Effects

This section contains comments on EPA's assessment of the body of evidence particularly related to judgments about the adversity of the health effects associated with O_3 exposure, primarily estimates of moderate lung function decrements (FEV_1 decrements ≥ 10%) from controlled human exposure studies.

(1) *Comment:* NAM contends that there is no consensus concerning where to draw the line distinguishing adverse from non-adverse effects, such as effects that are transient and reversible over short periods of time.

NAM contends that EPA increasingly treats as adverse mere exposure and that when exposure cannot be detected, EPA increasingly looks for biomarkers of exposure - irrespective of whether they are associated with symptoms or signs; their selectivity with respect to the hazard of concern; or the capacity to detect them without complex analytic techniques. As an example of this, the commenter points to the definition of a "key event" in EPA's Guidelines of Carcinogenic Risk Assessment. A *"key event"* is an empirically observable precursor step that is itself a necessary element of the mode of action or is a biologically based marker for such an element. The term *"mode of action"* is defined as a sequence of key events and processes, starting with interaction of an agent with a cell, proceeding through operational and anatomical changes, and resulting in cancer formation. The scientific task is a very limited one, consisting only of the assignment of phenomena into categories defined by policy. Commenter notes that information quality guidelines require that the Agency be transparent in its scientific description of these phenomena and the available data.

NAM contends that this same problem infects EPA's description of effects to asthmatics. Transient and reversible effects, such as chest tightness or wheezing, have a dozen reported triggers, including laughter. How similar are the effects of O_3 to the effects caused by exposure to humor? (NAM, p. 42)

Response: EPA strongly rejects NAM's assertion that there is no consensus in distinguishing adverse from non-adverse effects, with regard to the Administrator's judgments as to when O_3-related effects become regarded as adverse to the health of individuals. Evaluating the adversity of health effects in individuals and the resulting impacts on public health is a main focus of all NAAQS reviews. The issue of adversity was clearly addressed in this review and the judgments about adversity are consistent with the previous review. In this review, as in the 1997 review of the O_3 standard, the Administrator has looked to guidelines published by ATS and the advice of the CASAC panel. While recognizing that perceptions of "medical significance" and "normal activity" may differ among physicians, lung physiologists and experimental subjects, the ATS (1985)[4] 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

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

studies, 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. It is these effects that are the focus of this comment, and have been an important focus in the current and the 1997 review of the primary O_3 standard. To help frame such judgments, EPA staff have defined specific ranges of functional responses (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 (EPA, 2007).

For active healthy people, moderate levels of functional responses (e.g., FEV_1 decrements of $\geq$ 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 $\geq$ 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 $\geq$ 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 $\geq$ 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 $\geq$ 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 people with lung disease. There is more discussion of the focus on the lower end of moderate levels of functional responses (e.g., FEV_1 decrements ≥ 10%) in the next comment below.

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. This is the reason that EPA focuses not only on single, but also on repeated occurrences of moderate lung function decrements in school-age children.

As indicated by the discussion above and the additional discussions of adversity in the rationale for finding that the current standard needs revision and rationale for the level of the standard that is appropriate, EPA has clearly addressed the issue of the adversity of the transient and reversible effects attributable to O_3 exposure, specifically respiratory symptoms and lung function decrements, in both healthy people and people who have asthma. Moreover, EPA has evaluated the adversity of these effects in the context of daily activities. Its evaluation has been subjected to peer-review and supported by the CASAC in both the 1997 and current reviews of the O_3 standard. In this review of the O_3 standard EPA has not evaluated the adversity of any biomarkers of exposure, so with regard to the discussion of biomarkers the comment is not accurate.

(2) *Comment:* NAM contends that the 10% threshold for FEV_1 decrements is a post hoc threshold apparently chosen for compatibility with EPA staff policy recommendations in the Staff Paper. This threshold differs significantly from the clinical thresholds recommended by the ATS (15%) and CASAC (15-20%). While EPA says CASAC agrees with its 10% threshold, there is "strong evidence suggesting that any such agreement was at best a minority view based on policy considerations not science" (NAM, p.19). An individual CASAC panel member stated:

> While most attributions to CASAC are correct, I don't believe it was a written opinion of CASAC that "more emphasis should be placed on numbers of subjects in controlled human exposure studies with FEV_1 decrements greater than 10%, which can be clinically significant, rather than on the relatively small decrements" (p. 6-43). While this may have merit in some (or even many) situation, for example when noting that 26% of individuals had > 10% FEV1 decrements at 0.08 ppm …, in other cases, such as the specific case of 0.060 or 0.040 ppm exposures, this approach amounts to attempting to find effects in a very few

> individuals when the statistical tests are not significant, which is a dangerous precedent – especially in this case where we are looking at small effects in 3 of 30 vs. 1 of 30, a pitiful number on which to attempt to base policy … (CASAC, 2007, C-30) (NAM, pp. 19-20)

NAM states that EPA did not correct the misstatement in the final Staff Paper on p.6-43.

Response: The focus on FEV_1 decrements ≥ 10% came up in the context of evaluation of the adversity of transient and reversible effects, as discussed in the comment above. There was an extensive discussion of this topic at the August 24, 2006 meeting of the CASAC O_3 Panel in Research Triangle Park, NC (see transcript p. 142-150). During the discussion of Chapter 3 of the Staff Paper, one of the CASAC Panel members noted that in Chapter 6 of the Staff Paper, EPA staff focused on the lower end of the moderate range (i.e., FEV_1 decrements ≥ 10%) as an indicator of adverse effects in people with asthma, given the new evidence that people with asthma have more serious responses to O_3 exposure than healthy people. The CASAC panel, including Dr. Vedal the author of the opinion cited in the comment above, agreed that this focus on a 10% reduction in FEV_1 was an appropriate one and should also be included in the discussion of adversity in Chapter 3 of the Staff Paper. Clearly the CASAC Panel agreed that more weight should be placed on lung function decrements (FEV_1 decrements ≥ 10%) as an indictor of adversity for people with asthma. Since FEV_1 is measured primarily in controlled human exposure studies, EPA asserts that this statement "more emphasis should be placed on numbers of subjects in controlled human exposure studies with FEV_1 decrements greater than 10%, which can be clinically significant," is appropriately attributed to the CASAC Panel.

The commenter also incorrectly asserts that ATS has recommended a clinical threshold of a 15% decrement in FEV_1. In fact, ATS has not done this. The ATS guidelines indicate that a reduction in FEV_1 or FVC associated with clinical symptoms is adverse. The guidelines also indicate that a small but significant reduction in a population mean FEV_1 is probably medically significant:

> Exposure could also enhance risk for a population to an unacceptable degree, perhaps without shifting the risks of any particular individuals to an unacceptable level. Effects on persons with asthma are illustrative. A population of children with asthma could have a distribution of lung function such that no individual child has a level associated with significant impairment. Exposure to air pollution could shift the distribution toward lower levels without bringing *any individual child to a level that is associated with clinically relevant consequences*. Individuals within the population would, however, have diminished reserve function and are at potentially increased risk if affected by another agent, e.g., a viral infection. Assuming that the relationship between the risk factor and the disease is causal, the committee considered that such a shift in the risk factor distribution, and hence the risk profile of the exposed population, should be considered adverse, even in the absence of the immediate occurrence of frank illness. (ATS, 2000)

ATS also defines as adverse increased exacerbations of disease (i.e., shortness of breath) in people with chronic lung disease that could be reflected in a variety of ways, including for example, being less able to cope with daily activities. Clearly this is entirely consistent with EPA's definition of adversity, as discussed above.

e. Comments on Role of Ground-Level O_3 in Solar Radiation-Related Health Effects

Comment: A few commenters responded to EPA's request for comments, including 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 potential preventative effect ground level ozone may have in reducing exposure to ultraviolet radiation.

ATS, AMA and other medical organizations expressed the view that given the known adverse respiratory health effects of O_3, it would appear more prudent to focus on current approaches to reducing ultraviolet radiation by preventing UV exposures, rather than permitting the harmful respiratory effects of O_3. These commenters conclude that there is "no compelling evidence that should persuade the Administrator to consider this issue when setting the NAAQS O_3 standard." (ATS et al., p. 9)

AAM and a few other commenters that did not support revising the current primary O_3 standard expressed the opposite view that tropospheric O_3 has a beneficial effect along with stratospheric O_3 in protecting public health from solar UV radiation. A reduction in tropospheric O_3 would act to increase UV exposures and UV-induced health effects, including both non-melanoma and melanoma skin cancer. These commenters noted that when EPA responded to a court's remand (68 FR 614, Jan 2003) to consider UV-related effects of ground level O_3 there was no disagreement with the fact that ground-level O_3 has beneficial effects. The only disagreements centered on the magnitude of the effect or the certainty with which it can be estimated.

Response: EPA asserts that it has appropriately assessed the UV-B shielding effects of tropospheric O_3. The Criteria Document has assessed 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) focused 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 provided 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 (1) 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 (2) 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.

4. Specific Comments on the Population Exposure Analyses

Comments related to the population exposure analyses conducted for O_3 are addressed in this section. Incorporating responses contained in section II.B.2.b and II.C.4 of the preamble to the final rule, EPA provides the following responses to specific issues related to the exposure analyses. Comments related to the overall weight placed on the exposure analyses in reaching decisions on the need to revise the current standard and on an appropriate standard level are addressed in the preamble to the final rule and in section II.A.2.c.ii of this document.

(1) *Comment:* Several commenters (e.g., UARG, API, AAM) contend that EPA has failed to adequately address the significant uncertainties with its exposure assessment, including those associated with EPA's APEX exposure model.

Response: As discussed in the proposal notice, EPA recognizes that the exposure assessment necessarily contains many sources of uncertainty including those noted by these commenters, and EPA has accounted for such uncertainties to the extent possible. EPA developed and presented an uncertainty analysis addressing the most significant uncertainties affecting the exposure estimates (Langstaff, 2007). The CASAC Panel reviewed in detail the approach used to assess exposure and the presentation of the results in the Staff Paper. EPA believes, and the CASAC Panel concurred, that the model used to estimate exposures represents a state-of-the-art approach and that "there is an explicit discussion of the limitations of the APEX model in terms of variability and quality of the input data, which is appropriate and fine" (Henderson, 2006c, p. 11). Although EPA agrees that important limitations and uncertainties remain, and that future research directed toward addressing these uncertainties is warranted, EPA believes that overall uncertainties of short-term O_3 population exposure assessment have diminished since the last review. The Administrator has carefully considered the limitations and uncertainties associated with these quantitative assessments but continues to believe that they provide general support for concluding that exposures and health risks associated with meeting the current 8-hour standard are important from a public health perspective and that the 8-hour standard needs to be revised to provide additional protection in order to protect public health with an adequate margin of safety.

(2) *Comment:* One commenter (AAM) stated that O_3 monitors are often sited so as to capture the highest O_3 concentrations expected in an area. Since downwind sites are usually the design value sites, they will dominate the upper tail of the O_3 distribution and yet may not reflect the overall outdoor exposures in the area. This phenomenon will tend to bias the modeled exposures high.

Response: EPA agrees that O_3 concentrations can vary spatially across an urban area. In the exposure modeling conducted, EPA used several O_3 monitors in each area to take into account the spatial variations of O_3 concentrations. The geographic variation of O_3

concentration is accounted for by using measurements from the closest O_3 monitor to represent concentrations in a neighborhood and the measurements at downwind monitors are applied only to the downwind areas. Therefore this is not a source of bias in the modeled exposures.

(3) *Comment:* One commenter (AAM) stated that O_3 concentrations are lower at person's breathing height compared to measurement height. (Wisbeth et al. measured the increment between O_3 at 2 and 10 meters and reported an average 13 percent difference.) This phenomenon will tend to bias the modeled exposures high.

Response: EPA recognizes that there can be differences in O_3 concentrations between breathing and monitor heights and that this will result in some uncertainty in the modeled exposures, with some exposures underestimated and some overestimated as a result. However, as discussed in the exposure uncertainty analysis, data were not available to quantify the potential biases of differences between O_3 concentrations at a person's breathing height compared to the heights of nearby monitors. EPA believes that these biases, to the extent that they exist, are relatively small during warm summer afternoons when the atmosphere is well-mixed and O_3 concentrations tend to be higher.

(4) *Comment:* One commenter (AAM) stated that if people spend time outdoors in closer proximity to streets or in areas with more surface area (buildings, etc.) to quench O_3, their exposures will be below that measured at the monitor. This commenter contends that the APEX model assumes that whatever O_3 is interpolated from the monitor measurement is the actual O_3 exposure in the outdoor microenvironment, which will tend to bias the modeled exposures high.

Response: This statement about APEX is not true. The reduction in O_3 concentrations near roadways due to titration of O_3 from automobile emissions of NO is accounted for and explicitly modeled in APEX and thus does not bias estimates of exposures. This phenomenon was modeled through the use of "proximity factors," which adjust the monitored concentrations to account for the titration of O_3 by NO emissions (the monitored concentrations are multiplied by the proximity factors). Three proximity factor distributions were developed, one for local roads, one for urban roads, and one for interstates, with mean factors of 0.75, 0.75, and 0.36 respectively (section 3.10.2, Human Exposure TSD). Furthermore, the uncertainty of these proximity factor distributions was included in the exposure uncertainty analysis.

(5) *Comment:* Some commenters (API, AAM) noted that the upper tail of the distribution of breathing rates (equivalent ventilation rates (EVR)) is particularly important because it is a critical factor in determining the number of exposures of concern. These commenters contend that the APEX model predicts more elevated ventilation rate occurrences than observed in real world data. They argue that this results in an overestimation of the number of exposures of concern and of the risk estimates.

Response: EPA does not agree that the APEX model overestimates the number of exposures at specified exertion levels across all age groups. EPA did a comparison

(Langstaff, 2007) of ventilation rates predicted by APEX to measurements which showed APEX over-predicting ventilation rates for ages 5 to 10, under-predicting ventilation rates for ages 11 to 29 and greater than 39, and in close agreement for ages 30 to 39. The overall agreement was judged favorable, and the errors of the predicted ventilation rates were partially incorporated into the overall uncertainty analysis as part of the uncertainties of the metabolic equivalents (MET), which are the primary drivers of ventilation rates.

(6) *Comment:* One set of comments (ALA et al., pp. 104-105) contends that school absences, increased use of asthma medication, emergency room visits, and hospital admissions are not accurately reflected in EPA's use of "exposures of concern." Contrary to EPA's assertion, the exposure estimates cannot be generalized beyond the effects studied in the controlled human exposure studies -- that is, primarily lung function decrements and respiratory symptoms. Other health endpoints, such as school absences, increased use of asthma medications, long-term deficits in lung function and associated risk of illness, emergency room visits, hospital admissions, and premature deaths, have been characterized principally in epidemiological studies, where increased risks are a function of the ambient concentrations. The exposure analysis, which relies on activity profiles to minimize assumed population exposures, is not relevant to the estimation of those health endpoints which have been reported in epidemiological studies. This limitation contradicts EPA's desired use of the exposure assessment to "provide some perspective on the public health impacts of health effects that we cannot currently evaluate in quantitative risk assessments." 72 FR 37853. (ALA et al., pp. 104-105)

Response: The exposure analysis is not intended to be used to characterize potential health risks for health endpoints which have been reported in epidemiological studies. It also is not true that the exposure modeling "relies on activity profiles to minimize assumed population exposures" (ALA, p. 105). In discussing exposures at specified exertion levels EPA has referred to evidence of inflammation, increased airway responsiveness, and changes in host defenses in healthy people exposed to 0.080 ppm O_3 in controlled human exposure studies.

(7) *Comment:* EPA's exposure model underestimates repeated outdoor exposures. As EPA acknowledges, CHAD underestimates the frequency of occurrences of "repeated routine behavior." This results in underestimates of exposures to children who spend large portions of their summers playing outside or in summer camps. The "exposures of concern" assessment does not include outdoor workers or outdoor recreation enthusiasts who receive higher inhaled doses of O_3 due to their increased ventilation rates. Outdoor workers experience more frequent exposure to O_3 than the general population, due to the time spent outdoors, and the increased breathing rate under physical exertion. Several studies have examined the association between O_3 exposure and health outcomes in outdoor workers, including farm workers, mail carriers, and others. The Exposure Assessment and Risk Assessment completely ignore health risks to outdoor workers, a population that is exposed to ambient O_3 while under exertion. In the United States, this population constitutes more than 9 million people. Outdoor workers include a diverse set of occupations, ranging from construction workers to farm workers. (ALA et al., p. 105)

Response: This is discussed in the assessment of uncertainty of the exposure analysis. While EPA has not conducted an exposure analysis for outdoor workers since it was judged that school aged children presented the greatest likelihood of being outdoors and exposed under moderate exertion averaged over an 8-hour period based on the exposure analyses conducted for outdoor workers and children in the prior O_3 NAAQS review. EPA has recognized in the Staff Paper and in the final notice that outdoor workers would also be potentially affected by O_3 exposure. The Administrator has concluded that the revised standard will protect this population as well with an adequate margin of safety.

(8) *Comment:* One set of comments (ALA et al., p.105) contends that the exposure assessment focuses on the average child rather than the highly exposed and that active children are not well characterized. The exposure and risk assessments do not adequately capture risks to active children. EPA had initially profiled exposures of "active" children as a separate subpopulation, but subsequently dropped this category, and considers only exposures to average children. This analytical approach is then used to argue against adequate protection for those active children or adults that spend a lot of time outdoors. Because EPA averages the activity patterns of active and sedentary people, standards may not protect the most exposed individuals. (ALA et al., p. 105)

Response: This assertion is incorrect; all types of individuals are modeled, not the "average child." The exposure modeling does not average activity patterns. Active children are a subset of all children, which are modeled.

(9) *Comment:* The exposure analysis does not consider the effect of O_3 avoidance behavior on activity profiles. People living in the 12 cities examined experience frequent O_3 alerts warning them of unhealthy air quality and the need to avoid exercising outdoors. Schools, day cares and day camps routinely confine children indoors on code red days. The analysis fails to consider the extent to which O_3 avoidance behavior has diminished the estimates of outdoor exercise in children. (ALA et al., p. 105)

Response: EPA concurs that behavior changes in response to O_3 pollution or in response to AQI notification alerts ("avoidance behavior") were not explicitly taken into account in the exposure modeling. EPA recognizes that this is a limitation of the exposure analysis and discusses this in the exposure uncertainty analysis. There is not much information about the extent to which people currently modify their activities in response to O_3 alerts; in particular the activity data (CHAD) used in the analysis does not have sufficient information to allow avoidance behavior to be modeled. However, under the scenarios modeled for just meeting alternative standards, O_3 alerts would be infrequent relative to the number of alerts that currently occur in the nonattainment areas modeled. Consequently, EPA does not feel that this is an influential factor in the estimation of exposure for the scenarios simulating just meeting the current or proposed standards.

(10) *Comment:* Babies, toddlers, and preschoolers, an important segment of the population that spends lots of time playing outdoors, are not factored into the analysis. Only school-aged children are included in EPA's estimates. (ALA et al., p. 105)

Response: The exposure analysis focused on school age children and adults due to the lack of health effects evidence explicitly addressing O_3-related respiratory effects in pre-school age children. In addition, there is a paucity of data for human activity data for infants and toddlers that would make any exposure analysis applied to these populations very uncertain. The Administrator has taken into consideration that additional populations, including pre-school age children, are potentially at risk for the O_3-related respiratory effects observed in school-age children.

(11) *Comment:* Several commenters (e.g., ALA et al., p. 106) noted that most of the country is excluded from the analysis. The geographic scope of EPA's analysis is limited to just 12 metropolitan areas.

Response: See response to comment (1) in section II.A.5.

(12) *Comment:* One set of comments (ALA et al., p. 106) noted that the exposure assessment does not account for exposures and health impacts that result from O_3 transported from the 12 MSAs analyzed, which can actually result in people downwind being exposed to higher concentrations.

Response: The urban areas modeled are CSAs, not MSAs, and are large geographically (e.g., the Boston CSA extends into New Hampshire). However, the increase in O_3 downwind may extend beyond the CSA for some of the areas modeled, and this is not captured in the analysis. The design of the exposure analysis intentionally focused on 12 urban areas.

(13) *Comment:* O_3 concentrations vary from year to year with different weather conditions. Estimates of "exposures of concern" are subject to great variability depending on whether the baseline year for comparison is 2002, a relatively dirty year, or 2004, a relatively clean year. The risk and exposure analysis must focus on 2002 as a baseline year. Use of a year with favorable meteorology as the baseline year distorts exposure estimates. (ALA et al., p. 106)

Response: EPA accounted for this variability by analyzing all three years (2002-2004) with a focus on the year with higher exposures, 2002.

(14) *Comment:* One commenter (API) contends that "while EPA conducted an analysis of alternative adjustment procedures before choosing the quadratic over a linear one … their two-parameter quadratic rollback procedure is still suspect" (API, p.27). API expressed concern that the quadratic method was developed from data spanning only six to eight years which would mean that ozone differences could have been caused more by extreme meteorologic events than from actual reductions in ozone precursor emissions. Thus, it was stated that air quality data spanning ten to fifteen years should be used. The commenter also cited an alternative analysis which utilized ten to fifteen years worth of data and proposed alternative optimized adjustment procedures which better fit the ozone data in four cities.

Response: Regarding the commenter's objection that EPA's approach was developed from data spanning only six to eight years, EPA believes this time span was long enough to reflect sizable emission reductions and thus was an appropriate period to use for developing the approach. Also, use of a longer time span would have introduced uncharacteristically high O_3 concentrations from the early part of the examined period when compared to those seen in more recent years. Since the commenters analysis is based on a longer period, up to 15 years, its conclusions about ranking alternative air quality adjustment approaches may have introduced uncharacteristically high O_3 concentrations from the early part of the examined period. Further, EPA notes that the commenter's analysis offered no evidence or analysis that its proposed alternative air quality adjustment procedures would have made any meaningful difference in the exposure or risk estimates associated with just meeting the current or alternative 8-hour standards.

5. Specific Comments on the Health Risk Assessment

Comments related to the health risk assessment conducted for O_3 are addressed in this section. Incorporating responses contained in section II.B.2.b and II.C.4 of the preamble to the final rule, EPA provides the following responses to specific issues related to the quantitative health risk assessment. Comments related to the overall weight placed on the health risk assessment in reaching decisions on the need to revise the current standard and on an appropriate standard level are addressed in the preamble and in sections II.A.2.a and II.A.2.d above.

(1) *Comment:* Several commenters (ALA et al., NESCAUM) argued that EPA's risk assessment understated the total health benefits of reducing the O_3 standard. Reasons given included:

- It considered only 12 metropolitan statistical areas (MSA), and didn't consider benefits to adjacent areas beyond the MSA boundaries, that are likely because of the regional character of O_3;
- It didn't include all relevant health outcomes and populations; among the health outcomes and populations that were omitted are: lung function decrements and respiratory symptoms in adults; school absences for respiratory illness among children; asthma-related ER visits and increased medication usage among asthmatics; doctor visits, lung inflammation, and decreased resistance to infection among children and adults.
- A number of the health effects that EPA omitted in its risk assessment have been quantified in other analyses, including EPA's RIA, and in the benefits assessment conducted by California for the review of the state ambient air quality standard for O_3, both of which used EPA's BenMAP model. They concluded that the methodology for quantitatively assessing these other health endpoints is well established.
- EPA's risk assessment excluded vulnerable subpopulations that are either more susceptible to O_3 health effects, experience greater exposure, or both. Specific populations they identified that are not quantitatively assessed include children under five years old, active children, outdoor workers, and senior citizens.

- One commenter expressed the concern that EPA's risk assessment, being focused on 12 U.S. cities, may have underestimated O_3 impacts since it has been shown that O_3 concentrations downwind of an urban area are often higher than within a city. (Minister of the Environment, Province of Ontario, Canada)

  Response: EPA agrees that the exposure and health risk assessments are limited to certain urban areas and do not capture all of the populations at risk for O_3-related effects, and that the risk assessment does not include all potential O_3-related health effects. The criteria and rationale for selecting the populations and health outcomes included in the quantitative assessments were presented in the draft Health Assessment Plan, Staff Paper, and technical support documents for the exposure and health risk assessments that were reviewed by the CASAC Panel and the public. The CASAC Panel indicated in its letter that the health outcomes included in the quantitative risk assessment were appropriate, while recognizing that other health outcomes such as emergency department visits, increased doctors' visits should be addressed qualitatively (Henderson, 2006c). The Staff Paper (and the CASAC Panel) clearly 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 proposal notice made note of this important point and stated that "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" (72 FR 37866). However, as stated in the proposal notice, EPA also recognizes that inter-individual variability in responsiveness to O_3 shown in controlled human exposure studies for a variety of effects means that only a subset of individuals in any population group estimated to experience exposures exceeding a given benchmark exposure of concern level would actually be expected to experience such adverse health effects. The Administrator continues to recognize 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 is fully mindful of these limitations, along with the uncertainties in these estimates, in reaching his conclusion that observations from the exposure and health risk assessments provide additional support for his judgment that the current 8-hour standard does not protect public health with an adequate margin of safety and must be revised.

  In considering the level at which the primary O_3 standard should be set, the Administrator continues to place primary consideration on the body of scientific evidence available in this review on the health effects associated with O_3 exposure, as summarized in section II.C.4.a of the preamble to the final rule, while viewing the results of exposure and risk assessment, as providing information in support of his decision. While the Administrator places less weight on the results of the risk assessment, he notes that the results indicate that a standard set within the proposed range would likely reduce risks to at-risk groups from the O_3-related health effects considered in the 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. Moreover, he notes that the results of the assessment suggest a gradual reduction in risks with no clear breakpoint as increasingly lower standard levels are considered. However, in light of the important uncertainties inherent in the assessment discussed in the preamble to the final rule and in the proposal notice, the Administrator concludes that the risk assessment does not provide a basis for choosing a level within the proposed range.

To the extent the commenters suggest that EPA is legally bound to base quantitative features of the standards (such as levels) on the results of the risk assessment, or that the standards must be established at a lower level because the risk assessment quantifies morbidity and mortality effects below the levels the Administrator selected, the commenter is mistaken. See ATA III, 283 F. 3d at 373-74 (EPA not obliged to use the numerical results of the risk assessment to establish the 24-hour $PM_{2.5}$ standard at a lower level when it provided a reasonable basis for not using the assessment).

(2) *Comment:* A group of commenters mostly representing industry associations, businesses, and some State and local officials opposed to revising the 8-hour standard, and most extensively presented in comments from UARG, API, Exxon-Mobil, AAM, NAM, and the Annapolis Center raised the concern that exposures of concern and health risk estimates have not changed significantly since the prior review in 1997.

Response: In asserting that the estimated exposures and risks associated with air quality just meeting the current standard have not appreciably changed since the prior review, comments from Exxon-Mobil, the Annapolis Center and others have compared results of EPA's lung function risk assessment done in the last review with those from the Agency's risk assessment done as part of this review and have concluded that lung function risks upon attainment of the current O_3 standard are below those that were predicted in 1997 and that uncertainties about other health effects based on epidemiological studies remain the same. These commenters used this conclusion as the basis for a claim that there is no reason to depart from the Administrator's 1997 decision that the current 8-hour standard is requisite to protect public health.

EPA believes that this claim is fundamentally flawed for three reasons, as discussed in turn below: (1) it is factually inappropriate to compare the quantitative risks estimated in 1997 with those estimated in the current rulemaking; (2) it fails to take into account that with similar risks, increased certainty in the risks presented by O_3 implies greater concern than in the last review, and (3) it fails to recognize that the Administrator has used these estimates in a supportive role, in light of significant uncertainties in the exposure and risk estimates, to inform the conclusions drawn primarily from integrative assessment of the controlled human exposure and epidemiological evidence on whether ambient O_3 levels allowed under the current standard present a serious public health problem warranting revision of the O_3 standard.

With respect to the first point, the 1997 risk estimates, or any comparison of the 1997 risk estimates to the current estimates, are irrelevant for the purpose of judging the adequacy

of the current 8-hour standard, as the 1997 estimates reflect outdated analyses that have been updated in this review to reflect the current science. Just comparing the results for lung function decrements ignores these differences. In particular, as discussed in section 4.6.1 of the Staff Paper, there have been significant improvements to the exposure model and the model inputs since the last review that make comparisons inappropriate between the prior and current review. For example, the geographic areas modeled are larger than in the previous review and when modeling a larger area, extending well beyond the urban core, there will be more people exposed, but a smaller percentage of the modeled population will be exposed at high levels, if O_3 concentrations are lower in the extended areas. In the prior review, only typical years, in terms of O_3 air quality were modeled, while the current review used the most recent three year period (i.e., 2002-2004). Also, the prior review estimated exposures for children who spent more time outdoors, while the assessment for the current review included all school age and all asthmatic school age children. Therefore, the population groups examined in the exposure assessment are different between those considered in the 1997 and current review, making comparison of the resulting estimates inappropriate. Another important difference making comparison between the 1997 health risk assessment and the current assessment inappropriate is that a number of additional health effects were included in the current review (e.g., respiratory symptoms in moderate/severe asthmatic children, non-accidental and cardiorespiratory mortality) based on health effects observed in epidemiological studies that were not included in the risk assessment for the prior review. These commenters only compare the risk estimates with respect to lung function decrement and fail to account for differences in additional and more severe health endpoints not covered in the 1997 assessment, as well as the fact that there are somewhat different and more urban areas included in the current assessment.

Second, it is important to take into account EPA's increased level of confidence in the associations between short-term O_3 exposures and morbidity and mortality effects. In comparing the scientific understanding of the risk presented by exposure to O_3 between the last and current reviews, one must examine not only the quantitative estimate of risk from those exposures (e.g. the numbers of increased hospital admissions at various levels) but also the degree of confidence that the Agency has that the observed health effects are causally linked to O_3 exposure at those levels. As documented in the Criteria Document and the recommendations and conclusions of CASAC, EPA recognizes significant advances in our understanding of the health effects of O_3 based on new epidemiological studies, new human and animal studies documenting effects, new laboratory studies identifying and investigating biological mechanisms of O_3 toxicity, and new studies addressing the utility of using ambient monitors to assess population exposures to ambient O_3. As a result of these advances, EPA is now more certain that ambient O_3, alone or in combination with other pollutants, presents a significant risk to public health at levels at or above the range of levels that the Agency had considered for these standards in 1997. From this more comprehensive perspective, since the risks presented by O_3 are more certain and the current quantitative risk estimates include additional important health effects, O_3-related risks for a wider range of health effects are now of greater concern at the current level of the standard than in the last review.

Third, quantitative risk estimates were not the only basis for EPA's decision in setting a level for the O_3 standard in 1997, and they do not set any quantified "benchmark" for the Agency's decision to revise the O_3 standard at this time. While EPA believes that confidence in the causal relationships between short-term exposures to O_3 and various health effects reported in epidemiological studies has increased markedly since 1997, the Administrator also recognizes that the risk estimates for these effects must be considered in the light of uncertainties about whether or not these O_3-related effects occur at very low O_3 concentrations. The Administrator continues to believe that the exposure and risk estimates associated with just meeting the current standard discussed in the Staff Paper and summarized in the proposal notice are important from a public health perspective and are indicative of potential exposures and risks to at-risk groups. In considering the exposure and risk estimates, the Administrator has considered the year-to-year and city-to-city variability in both the exposure and risk estimates, the uncertainties in these estimates, and 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.

(3) *Comment:* Several commenters, mostly representing industry associations, businesses, and some State and local officials opposed to revising the 8-hour standard (e.g., UARG, API, Exxon-Mobil, AAM), contend that, given the uncertainties and limitations underlying the risk assessments, the health risk estimates are too speculative to support lowering the primary standard level. Specific uncertainties and limitations cited include:

- uncertainties in the exposure estimates;
- uncertainties about the concentration-response relationship (and especially the assumption of a linear non-threshold relationship between O_3 exposures and effects) particularly at levels below 0.08 ppm, so that the extrapolations assumed in the risk assessment may not be valid;
- uncertainties related to the use of single pollutant models rather than multi-pollutant models to estimate O_3-related health risks;
- the risk estimates based on lung function decrements use a benchmark of FEV1 declines of $\geq$ 15% for healthy individuals and $\geq$ 10% for asthmatics; these types of changes are transient, routinely experienced and well-tolerated, and not of adverse health consequence for either healthy or asthmatic individuals;
- the estimated risks of respiratory symptoms, hospital admissions, and non-accidental mortality are based on epidemiological studies, which do not reliably implicate O_3, and certainly not O_3 at any specific level, as the causal agent for the effects reported;
- the evidence for a causal relationship is particularly uncertain at lower O_3 levels; to the extent that the assumption of a causal link at lower O_3 levels is invalid, estimated reductions in O_3-related risk are artificially inflated;
- the evidence from single-city epidemiological studies reporting relationships between O3 and cardiorespiratory mortality are too unreliable and mixed to be included in the health risk assessment.

Response: EPA's response concerning uncertainties in the exposure estimates is discussed in the previous section (II.A.4) on exposure analysis. EPA's response to comments concerning the assumption of a linear non-threshold concentration-response relationship is addressed under comment number 5 in this section. EPA's response to comments concerning the use of single vs. multi-pollutant models to estimate O_3-related health risks is addressed under comment number (9) in this section. EPA's response to comments asserting that the lung function decrement benchmarks included in the assessment do not represent adverse health consequences for either healthy or asthmatic individuals is addressed above in section II.A.3.d (comment number (2)).

In response to the assertion that the results of time-series studies should not be used at all in quantitative risk assessments, EPA notes that the selection of specific studies and effect estimates was based on a careful evaluation of the evidence evaluated in the Criteria Document and that the criteria and rationale for selection of studies and effect estimates were presented and extensively reviewed and discussed by the CASAC Panel and in public comments presented to the CASAC Panel. EPA notes that the CASAC Panel judged the selection of the endpoints based on the epidemiological studies for inclusion in the quantitative risk assessment to be "appropriate" and that the risk assessment chapter of the Staff Paper and its accompanying risk assessment were "well done, balanced and reasonably communicated" (Henderson, 2006c, p. 12).

In response to comments that O_3-related risks for mortality and other endpoints based on the epidemiological evidence are artificially inflated due to the assumption of a causal relationship which is particularly uncertain at lower O_3 levels, EPA has responded to comments related to EPA's characterization of the weight-of-the-evidence with respect to judgments about causality in section II.A.3 above. The Administrator has carefully considered the epidemiological evidence and has taken into consideration the implications of the uncertainties regarding whether or not there is a causal relationship which extends to lower O_3 levels, particularly below 0.075 ppm. As discussed in the preamble to the final rule, this concern is one of the principle reasons that the Administrator has not placed much weight on the results of the risk assessment in his decision on the final level of the O_3 primary standard.

In response to the comments that risk estimates from single-city time-series studies should not be used since they are highly heterogeneous and influenced by publication bias, EPA notes that while two of the meta-analyses, Bell et al. (2005) and Ito et al. (2005), provided suggestive evidence of publication bias, O_3-mortality associations remained after accounting for that potential bias. The Criteria Document (p. 7-97) 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." Concerns about the heterogeneity of responses observed across different urban areas, particularly for O_3-related mortality, are addressed above in section II.A.3.b (comment number (2)).

As discussed in more detail in the Staff Paper (section 5.3.2.3), there are different advantages associated with use of single-city and multi-city effect estimates as the basis

for estimating health risks in specific urban areas. Therefore, the risk assessment included estimates based on both types of effect estimates where such information was available.

(4) *Comment:* One commenter (NAM, pp. 10-11 and p. 28) argued that the portions of the health risk assessment that EPA generated itself or sponsored are not sufficiently transparent, i.e., EPA does not provide enough information for these results to be replicated.

Response: EPA strongly disagrees with this comment. EPA has carefully documented the health risk assessment methods in the Health Risk TSD and has made available, upon request, the models and data inputs, including the exposure-response data from the controlled human exposure studies, used to develop both the exposure and health risk assessments as indicated on EPA's FERA website (http://www.epa.gov/ttn/fera/risk_ozone.html) which states:

> **Program and input files used in the Health Risk Assessment:** Because of the number and size of the program and data files, they are not available for interactive download. To receive a DVD containing all of the code and data files, contact Harvey Richmond via email at Richmond.harvey@epa.gov

In fact, comments submitted from another industry group (UARG) indicating that it was able to closely replicate the risk assessment results reported in the Health Risk TSD (UARG, Attachment 3) clearly refute the claim made by NAM that the health risk assessment is not transparent enough to be replicated.

(5) *Comment:* Some commenters (e.g., API, Lefohn) contend that EPA's risk assessment assumes a linear concentration-response relationship but that there is ample evidence in the epidemiological literature that the relationship between ambient O_3 levels and health effects is nonlinear. Similarly, one commenter contends, there is evidence that the relationship between O_3 and health endpoints in the human clinical literature is also nonlinear and that EPA's assumption of linearity in its risk assessment is therefore invalid, it was argued, and the resulting risk estimates are also invalid (API, p.23) NAM contends that EPA assumed a constant risk per unit of O_3 for all levels of O_3 exposure in its health risk assessment and that this assumption is not biologically plausible and that EPA offers no scientific evidence to support this proposition (NAM, p.40).

Response: The portion of the risk assessment based on epidemiological studies used studies that satisfied selection criteria that were clearly laid out (see section 4.1.5 in the Health Risk TSD), and the form of the concentration-response function was not one of the selection criteria – i.e., the risk assessment did not make its own assumptions about the form of the relationship between ambient O_3 levels and a given health endpoint but simply used the best epidemiological evidence available. The vast majority of O_3 epidemiological studies estimated log-linear concentration-response functions. There is not "ample evidence" in the epidemiological literature of a non-linear relationship between ambient O_3 levels and health effects (or, specifically, of a threshold in that

relationship). In fact, as discussed in the Staff Paper (p. 6-15), one recent multi-city study (Bell et al., 2006) examined the threshold question for the O_3-mortality concentration-response function using several different approaches and concluded that "all results indicate that any threshold would exist at very low concentrations, far below current U.S. and international regulations and nearing background levels."

The portion of the risk assessment based on controlled human exposure studies also did not assume linearity in the relationship between personal exposure to O_3 and lung function response. In this portion of the risk assessment, EPA fit two different models – a linear (hockeystick) model and a logistic model – to the data from the available controlled human exposure studies. The hockeystick model was derived by fitting a linear regression to the data but not constraining the intercept to be zero. Thus, if the regression line crossed the x-axis, the data "dictated" a threshold value – EPA set the response rate to zero for all O_3 exposures less than or equal to the exposure at which the regression line crossed the x-axis in this model, forming a "hockeystick" model. The logistic model is also nonlinear, and would constitute a smooth approximation to the hockeystick model. Both models allow for decreasing (or zero) response rates at lower levels of exposure. Because of the uncertainty about the correct functional form, EPA derived exposure-response functions that were mixtures of the two models. One such mixture hypothesized that the exposure-response relationship is logistic with 90% probability and linear (hockeystick) with 10% probability. The other two mixtures were 80%/20% and 50%/50%. Thus, even if the linear model was truly linear (i.e., even if it did not cross the x-axis), none of the final models used in this portion of the risk assessment were linear, because each assigned a substantial probability (90%, in the base case model) to the (nonlinear) logistic form of the relationship between personal exposure to O_3 and lung function response.

In response to NAM's comment, as indicated above, EPA did not assume a constant risk per unit of O_3 for the lung function portion of the health risk assessment. For the health outcomes based on epidemiological studies, as discussed above, EPA did not make its own assumptions about the form of the relationship between ambient O_3 levels and a given health endpoint but simply used the best epidemiological evidence available. The vast majority of O_3 epidemiological studies estimated log-linear concentration-response functions and EPA did offer scientific evidence, the analysis by Bell et al. (2006) which found only a very small change in the size of the effect estimate down to very low O_3 levels (i.e., 10 ppb). EPA did acknowledge the uncertainties about the shape of the concentration-response relationships for the health outcomes based on the epidemiological studies and stated in the Staff Paper that the risk estimates for these effects "must be considered in the light of uncertainties about whether or not these O_3-related effects occur in the population at very low concentrations" (Staff Paper, p.5-45).

(6) *Comment:* Several commenters (e.g., ACC) noted that only a single coefficient used in the risk assessment is based on 8-hour O_3 concentrations, the averaging time of the current and proposed NAAQS. They argued that therefore the calculated excess risks are likely not relevant to setting a NAAQS with an 8-hour averaging time.

Response: As with many of the criteria air pollutants, health effects have been reported in studies using a variety of averaging times. In this review, as in other NAAQS reviews, EPA has assessed risks arising from exposure to the pollutant associated with concentrations or exposures for various averaging times resulting from air quality just meeting the current and alternative standards for a specified averaging time. There is no requirement or logical argument that the averaging time of the models used to estimate specific health effects must be the same as the averaging time for the standard. It has long been recognized that air quality associated with just meeting a NAAQS with a specific averaging time will also limit air quality and exposures for other averaging times as well. For example, in 1997 EPA and the CASAC clearly concluded that an 8-hour O_3 standard set at an appropriate level would provide protection against both 1-hour and 8-hour O3-related effects. Therefore, EPA does not agree the calculated excess risks for health effects based on averaging times other than 8-hours are irrelevant to the setting of an 8-hour averaging time standard.

(7) *Comment:* One commenter (NAM, pp. 30-32) contends that both study selection bias (consistently selecting studies that show positive associations) and model selection bias (selecting models known to yield upwardly biased risk estimates, such as single-pollutant models that do not control for known confounders) can be found in EPA's risk assessment. Several sources of bias were given, including

- publication bias (the predominance of positive studies reported);
- the choice of which study to emphasize (with preference given to those with larger risk coefficients);
- inadequate control for confounders (leading to their effects being attributed to O_3);
- the choice of statistical methods (with some more likely to reject the no-effect hypothesis than others); and
- the choice of which estimate from which reported model to rely upon.

The result, they contend, is "a cascade of bias such that the resulting risk assessment is much higher than would have been obtained using objective methods."
Another comment (the Rochester Report) asserted that EPA ignored model uncertainty in its health risk assessment.

Response: EPA has addressed the issues of alleged publication bias, selection bias, control of confounders, and choice of statistical methods in section II.A.3 above and EPA does not agree with the conclusion that there is a "cascade of bias" resulting in higher risk estimates. EPA takes exception to the implication that it used methods with some known bias. As stated in EPA's IQG (p.22), "if data are subjected to formal, independent, external peer review the information may generally be presumed to be of acceptable objectivity." EPA's risk assessment underwent extensive peer review by the CASAC O_3 Panel, and in EPA's judgment the commenter has not rebutted the presumed objectivity of the methods used.

With respect to the selection of studies included, it is important to recognize that EPA's criteria for selection of studies and concentration-response relationships were described in the draft "Health Assessment Plan" (EPA, 2005) and in draft Health Risk TSD reports (Abt Associates, 2005; Abt Associates, 2006) and drafts of the Staff Paper that were

reviewed by the CASAC and made available to the public for comment at several stages during the review. The CASAC O_3 Panel did not express any concerns about EPA's selection of studies to be included in the O_3 risk assessment and in its October 2006 letter (Henderson, 2006c, p. 12)) to the EPA Administrator stated, "… the panel found Chapter 5 [the chapter in the Staff Paper that discusses the risk assessment] and its accompanying risk assessment to be well done, balanced and reasonably communicated." The CASAC O_3 Panel (Henderson, 2006c, p. 12) also explicitly stated that it judged the selection of health outcomes "for inclusion in the quantitative risk assessment to be appropriate."

Contrary to the statement in the Rochester Report, EPA did not ignore the issue of model uncertainty in its risk assessment. While we did not include a quantitative treatment of model uncertainty, EPA's risk assessment and Staff Paper clearly discussed the potential impact of model uncertainty and the importance of considering this issue in interpreting the results of the risk assessment. In particular, EPA's Staff Paper acknowledged that "other uncertainties, such as differences in study location, time period, and model uncertainties are not represented by confidence or credible intervals presented" (p.5-44, OAQPS Staff Paper). As discussed further in the Staff Paper, "Based on the CD's conclusions, we judge that there is insufficient evidence to support use of potential threshold levels in the quantitative risk assessment [we note that CASAC supported this choice during its review of the scope and methods to be used in the risk assessment], but we do recognize that there is increasing uncertainty about the concentration-response relationship at lower concentrations that is not captured by the characterization of the statistical uncertainty due to sampling error. Therefore, … the risk estimates for premature mortality, respiratory symptoms in moderate to severe asthmatic children, and respiratory-related hospital admissions associated with exposure to O_3 must be considered in the light of uncertainties about whether or not these O_3-related effects occur in the population at very low concentrations" (p. 5-45, OAQPS Staff Paper, emphasis added).

With respect to choice of models to rely upon in the risk assessment, EPA agrees that recent work on model sensitivity has raised new concerns and the Agency has given much attention to this issue. In so doing, EPA recognizes, as does HEI and other researchers, that there is no clear consensus at this time as to what constitutes appropriate control of weather and temporal trends in time-series studies, and that no single statistical modeling approach is likely to be most appropriate in all cases (EPA, 2004, p. 8-238).

The recent time-series epidemiologic studies evaluated in the Criteria Document have included some degree of control for variations in weather and seasonal variables. However, as summarized in the HEI review panel commentary, selecting a level of control to adjust for time-varying factors, such as temperature, in time-series epidemiologic studies involves a trade-off. For example, if the model does not sufficiently adjust for the relationship between the health outcome and temperature, some effects of temperature could be falsely ascribed to the pollution variable. Conversely, if an overly aggressive approach is used to control for temperature, the result would possibly underestimate the pollution-related effect and compromise the ability to detect a small but true pollution effect (EPA, 2004, p. 8-236; HEI, 2003, p. 266). The selection of

approaches to address such variables depends in part on prior knowledge and judgments made by the investigators, for example, about weather patterns in the study area and expected relationships between weather and other time-varying factors and health outcomes considered in the study. EPA is unaware of any information that supports the commenters' contention that the models selected by the original investigators are biased.

In considering these issues related to uncertainties in the underlying health science, on balance, EPA believes that the available evidence interpreted in light of these remaining uncertainties does provide increased confidence relative to the last review in the reported associations between short-term O_3 exposures and mortality and morbidity effects, alone and in combination with other pollutants, and generally supports stronger inferences as to the causal nature of the associations. EPA also believes that this increased confidence, when taken in context of the entire body of available health effects evidence, adds support to its conclusion that the current 8-hour standard needs to be revised to provide increased public health protection.

(8) *Comment:* Several commenters contend that EPA inappropriately uses data from ambient monitors as proxies for personal exposures in studies estimating effects that are used in the risk assessment. These commenters noted that the literature consistently shows that personal O_3 exposure is generally less than the values obtained from ambient monitors. Further, they stated that the only subpopulation for which ambient monitors may closely track personal exposure are outdoor workers during the O_3 season and there is virtually no overlap between this subpopulation and the sensitive subpopulations. To adhere to the information quality substantive objectivity standard, one commenter (NAM, p.32) contends that, "EPA must provide unbiased estimates of exposure for each subpopulation of concern."

Response: Since the relationships used in the risk assessment for the health outcomes based on epidemiological studies are *concentration-response* relationships that relate ambient concentrations to a population response there is no bias introduced in the health risk estimates by not having an exposure assessment. The fact that ambient concentrations may overstate actual personal exposure does not imply that the risk estimates are biased. EPA does not agree that there is any requirement to provide unbiased estimates of exposure for each subpopulation group of concern before it can use concentration-response relationships in its risk assessments.

(9) *Comment:* One commenter (NAM, p. 34) contends that EPA's decision to disseminate risk estimates derived from single-pollutant models as estimates of risk, even when multi-pollutant models are available, is an unambiguous violation of the objectivity standard. NAM contends that EPA has utilized the study by Gent et al. (2003) in ways that systematically overstate risk estimates and that EPA assumes asthmatics are exposed at the same level as other children (NAM, p. 34).

Response: EPA rejects NAM's contention that disseminating risk estimates derived from single-pollutant models is a violation of the objectivity standard. As discussed in the Criteria Document and section 3.3.2.1 of the Staff Paper, O_3 epidemiological studies have

reported results based on both single and/or multi-pollutant models. As stated in the Staff Paper (p. 5-35), "To the extent that any of the co-polluants present in the ambient air may have contributed to the health effects attributed to O_3 in single pollutant models, risks attributed to O_3 might be overestimated where concentration-response functions are based on single pollutant models. However if co-pollutants are highly correlated with O_3, their inclusion in an O_3 health effects model can lead to misleading conclusions in identifying a specific causal pollutant. When collinearity exists, inclusion of multiple pollutants in models often produces unstable and statistically insignificant effect estimates for both O_3 and the co-pollutants. Given that single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other in all cases, risk estimates based on both single-and multiple-pollutant models have been included in the risk assessment where both are available." The CASAC O_3 Panel generally agreed with this approach in its review of the risk assessment scope and methods plan, Health Risk TSD, and final Staff Paper. Further, EPA notes that CASAC's overall assessment of the risk assessment was that it was "well done, balanced and reasonably communicated" (Henderson, 2006c) which supports EPA's view that the approach used did not violate the objectivity standard under the IQG.

For the reasons discussed in the paragraph above, EPA does not agree that it has used the Gent et al. (2003) study in ways that systematically overstate risk estimates. Further, EPA did not make any assumption about the exposure of asthmatics relative to other children. EPA applied concentration-response relationships reported in this study, which are relationships between levels observed at ambient monitors and a given response, to ambient data in the Boston area. Thus, NAM is incorrect in stating that EPA assumed asthmatics had the same exposure as other children in the risk assessment.

(10) *Comment:* Several commenters (e.g., AAM, NAM, p. 50) contend that, because EPA failed to show that the associations between O_3 and health endpoints are causal, the risk assessment should include a probabilistic consideration that the associations may not be causal. They contend that, in contrast to the acute respiratory effects in clinical studies where we know the observed effects are due to the exposure of the pollutant being assessed, "the most important uncertainty is the extent to which the associations between O_3 and the health endpoints included in the assessment actually reflect causal relationships" (AAM, citing Second Draft SP at 5-76). Noting the "biologically implausible" wide range of associations for mortality, one commenter questioned the practice of using Bayesian techniques to shrink the (highly heterogeneous) city-specific estimates towards the overall mean, especially when the heterogeneity includes both no effect and protective effects (AAM). They concluded that the risk assessment should include a probabilistic consideration that the associations may not be causal, pointing out that the Regulatory Impact Analysis (RIA) does evaluate the case assuming that O_3 mortality associations are not causal.

Response: EPA does not agree that it must include a probabilistic characterization of causality for the health effects included in the risk assessment. EPA's characterization of likely causality for the various O_3-related health endpoints included in the risk assessment is presented in the Criteria Document and Staff Paper (see section 3.7.5). As stated in the

Staff Paper, EPA has only included health endpoints in the quantitative risk assessment for which there is, "at a minimum, a likely causal relationship with either short-term O_3 exposure itself or with O_3 serving as an indicator for itself and other components of the photochemical oxidant mix, especially during the warm O_3 season" (p.5-42), EPA has not assigned a probability to causation. The Administrator has taken into consideration qualitatively uncertainties related to whether or not there is a causal relationship in general and particularly the extent to which there may or may not be a causal relationship at increasingly lower O_3 levels.

(11) *Comment:* One commenter (NAM) contends that EPA abandoned its commitment "to provide the best possible scientific characterization of risks based on a rigorous analysis of available information and knowledge" by declaring, in the following statement, that its practices were biased, and purposefully so: "EPA's risk assessments are conducted in support of its mission to protect public health and the environment. Given the uncertainty, variability, and data gaps encountered when conducting any risk assessment, a key objective for EPA's risk assessments is that they avoid both underestimation of risk and gross overestimation of risk. … EPA seeks to adequately protect public and environmental health by ensuring that risk is not likely to be underestimated." (NAM, pp. 39-40)

Response: Contrary to the claim made in the comment, EPA has not abandoned its commitment to provide the best possible scientific characterization of risks. As noted previously, EPA's approach and analytical assumptions were carefully documented and subject to review by the CASAC O_3 Panel and the public and were based on the scientifically peer-reviewed final Criteria Document. The CASAC O_3 Panel clearly supported EPA's view that the health risk assessment was not biased when it stated that the assessment was "well done, balanced, and reasonably communicated" and that the selection of health endpoints for inclusion in the quantitative risk assessment was appropriate (Henderson, 2006c). As the quoted passage in the comment indicates, EPA seeks to avoid both underestimation of risk and gross overestimation of risk. EPA notes that in conducting any health risk assessment, analytical choices have to be made and EPA has carefully discussed any known directional influences of these choices in the Health Risk TSD and Chapter 5 of the Staff Paper.

(12) *Comment:* One commenter (NAM) contends that when EPA says that its estimates of risk from O_3 (or a peer reviewed study on which it relies) has "controlled for PM," it is not clear whether that control addressed only PM_{10} or $PM_{2.5}$ as well. $PM_{2.5}$ is correlated in space and time with O_3; PM_{10} is not. Thus, controlling for PM_{10} is, in effect, functionally equivalent to no control at all (NAM, p. 41).

Response: For each concentration-response function used in the risk assessment, the specifics of the function, including co-pollutants in the model, are listed in Table 4-2 of the Health Risk TSD. For those models that included a measure of PM, the exact measure used (i.e., $PM_{2.5}$ or PM_{10}) is given in Table 4-2, so there should be no lack of clarity about this.

Moreover, there is some evidence that the influence of PM on the O_3-mortality effect estimate may not be very substantial. One study, Levy et al. (2005), ran univariate regressions at a number of sites with O_3 as the dependent variable and another criteria pollutant as the independent variable, for each of the other criteria pollutants, including PM_{10} and $PM_{2.5}$. They then examined the extent to which the relationship (correlation) between O_3 and the other pollutant at a site influenced the O_3-mortality effect estimate at the site. They found only a weak relationship between the O_3-$PM_{2.5}$ correlation at a site and the O_3-mortality effect estimate at the site. Further analysis of a possible $PM_{2.5}$ impact on the O_3-mortality effect estimate did not alter this finding (see Levy et al., 2005, p. 463).

(13) *Comment:* Several commenters (e.g., NAM, Rochester Report, UARG) argued that EPA should conduct an "integrated uncertainty analysis" – or, as it was described in one comment quoting the National Research Council (NRC, 2002), "EPA should begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple source uncertainty analysis. This shift will require specification of probability distributions for major sources of uncertainty, which should be based on available data and expert judgment" (Rochester Report, p. 19; NAM, p.46) Such an "integrated uncertainty analysis" would include the effects of additional sources of uncertainty, "including model selection, non-linearity of the concentration response function at lower and lower concentrations, and the role of other pollutants" (UARG, p. 28). Additional related comments included:

- It was suggested that, the preponderant effect of all of the sources of uncertainty is to create an upward bias in EPA's risk estimates. "Even when leaving aside the uncertainty about causality, analyses produced by other researchers have demonstrated that when an integrated uncertainty analysis is performed, the large majority of probability in the estimates falls far below the primary estimates that EPA reports (Rochester Report, p. 75)."
- EPA has presented the Administrator data and analyses that led him to be much more confident than is scientifically justified that O_3 exposure below the current NAAQS poses actual human health risks. EPA's risk estimates capture only statistical variability for the selected models, not scientific uncertainty about their validity and reliability as estimates of human health risk. Information about variability, which is small relative to the magnitude of variability and uncertainty combined, has no utility to the Administrator unless it is placed in proper context with information about uncertainty" (NAM, pp. 44-45).
- One group of commenters contends that EPA sometimes reports "confidence bounds" or "uncertainty intervals" for its primary estimates, but these ranges are based solely on the standard error of the single relative risk coefficient estimate. "In other words," they contend, "EPA presents to the public a measure of variability as if it were a measure of uncertainty. This is highly misleading to the non-technical audience that may not take the time to learn all of the details of EPA's analysis methods. Thus, this audience is easily swayed into believing that these ranges are a reasonably complete representation of the uncertainties about the level of risk. Given that some of these ranges are very wide (in many cases falling into the negative numbers) it would be quite understandable if people

were to think these ranges present a comprehensive view of uncertainty, but they do not even start to do so" (Rochester Report, p. 73).

Response: First, EPA notes that the 2002 NRC report cited by several commenters made recommendations with respect to EPA's regulatory impact analyses which are required under E.O. 120266 and not EPA's health risk assessments. Even in this separate context, the NRC recognized that it would require a transition over time to implement its recommendations to incorporate more probabilistic assessments in health benefits assessments.

Second, the point that the uncertainty ranges reported in the risk assessment do not reflect all of the uncertainty in the risk estimates is explicitly discussed in the Staff Paper (EPA, 2007, section 5.3.2.5). As indicated in the Staff Paper, statistical uncertainty surrounding the estimated O_3 coefficients in the reported concentration-response functions is reflected in the confidence intervals and additional uncertainties "have been addressed quantitatively through sensitivity analyses and/or have been discussed throughout section 5.3" (Staff Paper, p. 5-40). Given the existing data gaps in the scientific evidence and associated uncertainties, a more comprehensive integrated assessment of uncertainties, would have be desirable, but in the staff's judgment would require use of techniques involving elicitation of probabilistic judgments from health scientists. While the Agency is currently developing these approaches, such comprehensive assessments of uncertainty are not available for the current O_3 risk assessment for this NAAQS review.

Third, EPA does not agree that the preponderant effect of all of the sources of uncertainty is to create an upward bias in EPA's risk estimates and EPA does not agree that other researchers have presented a credible, balanced, peer-reviewed integrated uncertainty analysis that shows the large majority of probability in the estimates falls far below the primary estimates that EPA reported. The analysis conducted by A. Smith for UARG cited by the commenter only examined one health endpoint (i.e., premature mortality) and is based on the assumption that there is no O_3-related response associated with levels below 0.070 ppm. As discussed in the Staff Paper (section 5.3.2.5) and section II.A.2 of the preamble to the final rule, there is no scientific basis to conclude that a population threshold exists at this level.

Fourth, EPA has defined very carefully in the Staff Paper and Health Risk TSD (sections 3.1.5 and 4.1.9) the concepts of uncertainty and variability and has extensively discussed important sources of both uncertainty and variability and their impact on the health risk assessment.

(14) *Comment:* One commenter (NAM) contends that EPA has violated the fundamental risk assessment principle that double counting is not acceptable by attributing to O_3 health risks that have been accounted for previously by EPA -- most prominently, premature mortality risks in its analysis of fine PM (NAM, pp. 51-52).

Response: In discussing the selection of concentration-response functions to use in the portion of the O_3 risk assessment based on epidemiological studies, EPA noted that,

"single and multi-pollutant models each have both potential advantages and disadvantages, with neither type clearly preferable over the other in all cases…" and, as a result, EPA made the decision to "report risk estimates based on both single- and multi-pollutant models where both are available" (Health Risk TSD, p. 4-11). It is possible that a single pollutant model could falsely attribute some portion of mortality risk reduction (or avoided cases of mortality) to O_3 that should instead be attributed to fine PM, and if those avoided cases of mortality were already attributed to fine PM in a $PM_{2.5}$ analysis, then there would be some degree of double counting. However, the evidence that this is the case to any substantial degree for PM as the copollutant is weak. One study, Levy et al. (2005), ran univariate regressions at a number of sites with O_3 as the dependent variable and another criteria pollutant as the independent variable, for each of the other criteria pollutants, including PM_{10} and $PM_{2.5}$. They then examined the extent to which the relationship (correlation) between O_3 and the other pollutant at a site influenced the O_3-mortality effect estimate at the site. They found only a weak relationship between the O_3-$PM_{2.5}$ correlation at a site and the O_3-mortality effect estimate at the site. Further analysis of a possible $PM_{2.5}$ impact on the O_3-mortality effect estimate did not alter this finding (see Levy et al., 2005, p. 463). This suggests that, if there is any "double counting" across EPA O_3 and $PM_{2.5}$ analyses, it will be fairly small. Given that there is a different price to be paid in using only multi-pollutant models (i.e., in reduced stability of the O_3 coefficient estimate in such models), EPA continues to believe that the application of both single- and multi-pollutant models, where both were available, is an appropriate approach.

(15) *Comment:* One commenter (UARG), "recognizing that the Administrator proposes to reject any possible standard level below 0.070 ppm because of the uncertainty about a causal effect of low levels of O_3," commissioned an analysis of the distribution of the risk in EPA's risk assessment attributable to O_3 exposures above and below 0.070 ppm. The analysis found that "very little of EPA's estimated mortality and morbidity risk is attributable to days when the maximum 8-hour average level of O_3 is more than 70 ppb" and that attainment of the existing NAAQS "would actually provide very significant protection against exposures to O_3 right down to 70 ppb." They concluded that there is no reason to believe that promulgation of a more stringent NAAQS would result in meaningful reduction in health risk. (UARG, pp. 24-25)

Response: EPA had conducted its own analysis for non-accidental and cardiorespiratory mortality that partitioned the contribution to overall O_3-related risk estimates from specified O_3 concentration range intervals as displayed in figure 5-15 of the Staff Paper (p.5-79) and figures 4-14 and 4-15 of the Health Risk TSD (pp. 4-70 to 4-71). UARG's analysis makes the assumption that there is population threshold at 0.070 ppm. The Administrator is very mindful of the uncertainties related to whether the observed associations between O_3 concentrations at very low levels, well below 0.080 ppm, and the health outcomes reported in the epidemiological studies reflect actual causal relationships and has taken this into account in considering the risk assessment estimates in his decision.

The Administrator does not agree with the commenter's conclusion that a more stringent NAAQS would not result in meaningful reductions in health risk. The Administrator has taken into consideration the pattern of exposure estimates across the range of discrete benchmark levels considered in EPA's exposure assessment to provide some indication of the potential magnitude of the incidence of health outcomes that could not be evaluated in the Agency's quantitative risk assessment but which have been demonstrated to occur in healthy people at O_3 exposures as low as 0.080 ppm, the lowest level at which such health outcomes have been tested.[5] As discussed in the preamble to the final rule, the Administrator concludes that the exposure assessment suggests that exposures at above the 0.080 ppm benchmark level, where several health effects have been shown to occur in healthy individuals, are essentially eliminated for standards in the range 0.070 to 0.075 ppm. He also concludes that at the 0.070 ppm benchmark level, the exposures are substantially reduced and eliminated for the vast majority of people in at-risk groups, and that the very low estimates of such exposures are not appreciably different, from a public health perspective, between those exposures associated with just meeting a standard set at 0.070 ppm or 0.075 ppm. Further, the Administrator places relatively little weight on the exposures using the 0.060 ppm benchmark level given the very limited scientific evidence supporting a conclusion that O_3 is causally related to health outcomes at this exposure level. Considering the uncertainties associated with the exposure assessment, the Administrator concludes that the exposure estimates associated with each of the benchmark levels are not appreciably different, between a 0.070 or 0.075 ppm standard, and therefore, the exposure assessment does not provide a basis for choosing a level within the proposed range.

While the Administrator places less weight on the results of the risk assessment, he notes that the results indicate that a standard set within the proposed range would likely reduce risks to at-risk groups from the O_3-related health effects considered in the 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. Moreover, he notes that the results of the assessment suggest a gradual reduction in risks with no clear breakpoint as increasingly lower standard levels are considered. However, in light of the important uncertainties inherent in the assessment discussed above and in the proposal, the Administrator concludes that the risk assessment does not provide a basis for choosing a level within the proposed range.

(16) *Comment:* Several commenters (e.g., API, UARG, Rochester Report) contend that the approach EPA used to adjust hourly O_3 concentrations to simulate just attaining an alternative standard, the quadratic rollback approach, may overstate the air quality

[5] Such health outcomes include increased airway responsiveness, increased pulmonary inflammation, increased cellular permeability, and decreased pulmonary defense mechanisms, which have been 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.

improvements resulting from emissions reductions and thus overstate the benefits of stricter standards. One commenter (API) stated that the quadratic air quality adjustment approach "was still suspect" (API, p. 27) and submitted an analysis examining alternative air quality adjustment procedures. In addition, there is uncertainty about whether the alternative standards in the proposed range are even attainable, given EPA's focus on the mean background and failure to consider the extremes of background. One group of commenters contends that considerable uncertainty remains on the impact of reducing the 8-hour O_3 standard from the current level of 0.08 ppm to a lower level, because EPA has assumed for its analysis that shifts in the probability distribution of exposure to O_3 will occur not just in the upper tail of the distribution (the days on which O_3 levels will exceed the existing 8-hour standard of 0.08 ppm or proposed alternative standards of 0.060 to 0.074 ppm), but also the lower and middle portions of this distribution, levels below 0.060 ppm. It is these changes in the lower and middle portion of the distribution, they pointed out, that provide most of the calculated benefits in terms of reduced short term-mortality and reduced hospital admissions associated with the more stringent alternative proposed standard (Rochester Report, p. 19). One commenter contends that EPA's "proportional rollback" approach assumes that States will reduce O3 levels by a "proportionate amount throughout the distribution of 8-hour measurement periods" (NAM, p. 59).

Response: First, with respect to the air quality adjustment approach used in the current review to simulate air quality just meeting the current and alternative O_3 standards, as discussed in the Staff Paper (section 4.5.6) and in more detail in a staff memorandum (Rizzo, 2006), EPA concluded that the quadratic air quality adjustment approach generally best represented the pattern of reductions across the O_3 air quality distribution observed over an 8-year period in areas implementing control programs designed to attain the O_3 NAAQS. See the response to comment number (14) in section II.A.4 in this document for additional response to API's analysis of alternative air quality adjustment procedures. While EPA recognizes that future changes in air quality distributions are area-specific, and will be affected by whatever specific control strategies are implemented in the future to attain a revised NAAQS, there is no empirical evidence to suggest that future reductions in ambient O_3 will be significantly different from past reductions with respect to impacting the overall shape of the O_3 distribution.

Second, it is not permissible for EPA to consider whether or not a given alternative standard is or is not attainable under the provisions of section 109 of the Clean Air Act.

Third, EPA believes that the commenters' implied alternative approach of only reducing peak 8-hour daily maximum values that are at or near the standard level is unrealistic in that most O_3-related air pollution control measures are continuous in nature and have an impact on the entire distribution of 8-hour O_3 concentrations. In contrast, EPA's use of a quadratic adjustment procedure that reduces not only the upper end of the distribution, but also to a lesser extent the middle and lower end of the distribution has been shown to match reductions observed in O_3 concentrations over nearly a decade.

Fourth, EPA rejects NAM's contention that EPA used a "proportional rollback" approach and that the approach was not justified, when in fact, as described above, EPA used a quadratic air quality adjustment approach that reduced the upper end of the O_3 distribution more than the middle or lower-end of the distribution. Further, as discussed above, EPA's air quality adjustment approach was based on an analysis of historical changes in O_3 concentrations and EPA's approach is conistent with past changes in O_3 concentrations resulting from a myriad set of national, state, and local control programs reducing O_3 precursors.

Finally, some commenters appear to interpret attainment of the standard using a single year. This is not consistent with the form of the current standard or the alternative standards that EPA analyzed. Thus, EPA does not agree that its approach assumed an arbitrary degree of further reduction, but rather EPA's approach reflects that the reductions required are based on a three-year period, not just a single year.

(17) *Comment:* One commenter contends that, because 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, 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 this, this commenter questioned whether the Agency can or should consider these projected gains as a health-based criterion for its decision making (Dow Chemical). Another commenter states that "it is technically infeasible to eliminate all U.S. anthropogentic emissions" and that "it is also technically infeasible to achieve the revised ozone standards proposed" (NAM, p. 63).

Response: EPA notes that the Administrator in reviewing and making decisions with respect to the NAAQS is prohibited from considering the ability of areas to attain the NAAQS. In addition, while cost and technological feasibility are taken into account in the implementation of the NAAQS – even if an area is delayed in meeting the standard due to cost or other implementation difficulties -- this does not reduce the benefits to public health that could be expected from full attainment of a new standard, which is the focus of EPA's risk assessment.

(18) *Comment:* A group of environmental and health organizations contends that EPA "has illegally understated the health effects of O_3 by basing its risk assessment solely on risks in excess of policy-related background levels. Section 109 of the [Clean Air] Act requires the primary NAAQS to be set at a level requisite to protect public health with an adequate margin of safety: it does not allow EPA to protect only against health risks presented by O_3 attributable to anthropogenic sources. A person breathing the air is exposed to the total concentration of O_3 in the air, including background, and the NAAQS must protect against that total exposure. There is nothing in the Act that allows EPA to ignore or discount the risk presented by the background component of such exposures." (ALA et al., p. 110)

Response: EPA does not agree that it is inappropriate or impermissible to assess risks that are in excess of PRB or that EPA must focus on total risks when using a risk assessment to inform decisions on the primary standard. Consistent with the approach

used in the risk assessment for the prior O_3 standard review and consistent with the approach used in risk assessments for other prior NAAQS reviews, estimating risks in excess of PRB is judged to be relevant to policy decisions regarding the ambient air quality standard as it provides useful information regarding the risks attributable to anthropogenic emissions. EPA also notes that with respect to the adequacy of the current standard taking total risks into account would not impact the Administrator's decision as he judges that the current standard is not adequate even when risks in excess of PRB are considered. In addition, EPA notes that consideration of the evidence itself, as well as exposures at and above benchmark levels in the range of 0.060 to 0.080 ppm are not impacted at all by consideration of PRB. In this review, EPA's use of PRB in the risk assessment had little impact on the Administrator's determination regarding the level of the standard given the relative weight placed on the risk assessment and the range of PRB values compared to the standards under consideration.

(19) *Comment:* While disagreeing with EPA's approach of estimating risks only above PRB, ALA et al. supported the use of the GEOS-CHEM model as the "best tool available to derive background concentrations" should EPA continue to pursue this approach.

Response: EPA agrees with this comment.

(20) *Comment:* Many industry commenters argued that EPA's (flawed) approach to estimating PRB concentrations has lead to underestimates of PRB and thus to an overestimation of the numbers of cases of health effects associated with O_3 concentrations above PRB. Some commenters also pointed out that the major difference between this review and the previous one is the lower background level; others pointed out that if the same background level were used, almost all the health benefits would be reduced by up to 90% – or, as one commenter put it, "the choice of the PRB dominates EPA's estimates of O_3 health risks" (NAM, p. 62). These commenters expressed concerns that PRB levels were estimated by EPA solely by modeling using the GEOS-CHEM model, which they argued underestimates PRB. Related arguments made related to the role of PRB in the risk assessment included:

- A few commenters contend that EPA's current PRB estimates inappropriately excluded certain emissions contributions that should be included – notably, anthropogenic precursor emissions from Canada and Mexico. They argued that this approach artificially inflates the risk estimates.
- One commenter argued that, based on EPA's limited sensitivity analysis to examine how sensitive the risk estimates are to differing assumptions about PRB, the understatement of PRB can result in substantial overstatements of risk. For example, assuming PRB was 0.005 ppm higher than that predicted by the GEOS-CHEM model decreased estimates of nonaccidental mortality by 50% or more – and resulted in a 62% decrease in predicted incidence of nonaccidental mortality in Los Angeles upon attainment of the present standard when 2002 O_3 air quality data were used (API).
- One commenter contends that the risk estimates were "highly sensitive (in the downward direction) to any of several alternative possible assumptions on PRB. The commenter found that using the monitored air quality data from the Trinidad Head site, "believed by many to represent PRB better than do the results of the GEOS-CHEM modeling," when

estimating the risk remaining after attainment of the existing NAAQS reduced the average risk estimates by from 36% to 72% from the risk levels predicted in EPA's analysis. For Sacramento and Los Angeles, the two cities considered in EPA's risk assessment that are closest to the Trinidad Head site, the estimates of risk fell by 65% and 72%, respectively. The commenter concluded that the absolute risk estimates are highly sensitive to alternative plausible PRB assumptions, preventing meaningful comparisons of health risks at alternative possible standard levels to that upon attainment of the existing NAAQS (UARG, pp. 25-27).

- One commenter argued that the sensitivity to 0.005 ppm changes in background demonstrates that the bulk of estimated mortality comes from days with O_3 concentrations that are very low. This commenter contends that this is not biologically plausible (AAM).
- One commenter argued that EPA had ignored an important comment from the CASAC Panel which undermines the approach used in EPA's risk assessment. Citing CASAC's August teleconference, this commenter quoted the CASAC letter (Henderson, 2006b) which noted that PRB is highly problematic to calculate and is, in some sense, "unknowable." CASAC went on to say, "One can avoid this problem, it was contended, by calculating the change in incidence of the health effect associated with a change in O_3 from the current standard to some other specified concentration. This approach, it was argued, would allow focus on the question, 'What is the difference in the expected number of health effects that will occur at various concentrations of O_3, relative to the current standard of 0.08?' A key advantage of this approach is that it does not depend on the choice of PRB, and thus is free of the uncertainties surrounding estimation of PRB" (NAM, p. 26).
- One commenter argued that the sensitivity of clinical respiratory effects to background assumptions, while not as large as for other health effects, is still very important. Citing Tables 3-28 and 3-31 in the Health Risk TSD, this commenter noted that the numbers of occurrences of lung function response of healthy and asthmatic children are reduced with a 0.005 ppm increase in PRB almost as much as a lowering of the standard from 0.084 to 0.074 ppm. The commenter concluded that, given the importance of the choice of background, the Agency should reevaluate its assumed background and then re-evaluate the clinical risks (AAM).
- One commenter contended that EPA had not considered one literature review (Vingarzan, 2004) and one research study (Ortmans et al., 2006) and that "both articles indicate that there is significant spatial and seasonal variability, with springtime peak" (NAM, p. 62).

Response: First, the U.S. government has influence over emissions at our borders that affect ambient O_3 concentrations entering the U.S. from Canada and Mexico through either regulations or international agreements, and therefore EPA does not agree that these emissions are uncontrollable. PRB is currently designed to identify O_3 levels that result from emissions that are considered uncontrollable because the U.S. has little if any influence on their control, and in that context anthropogenic emissions from Mexico or Canada should be excluded from PRB. EPA has consistently defined PRB as excluding anthropogenic emissions from Canada and Mexico in NAAQS reviews over more than two decades and sees no basis in the comments to alter this current definition.

Second, the criticisms raised concerning the use of a modeling approach (GEOS-CHEM) and the potential alternative approach of using remote monitoring data to estimate PRB were considered by EPA's scientific staff and the CASAC Panel during the course of reviewing the Criteria Document. Both EPA's experts and CASAC endorsed the use of the peer-reviewed, thoroughly evaluated modeling approach (GEOS-CHEM) described in the Criteria Document as the best approach for estimating PRB levels. The Criteria Document reviewed detailed evaluations of GEOS-CHEM with O_3 observations at U.S. surface sites (Fiore et al., 2002, 2003) and comparisons of GEOS-CHEM predictions with observations at Trinidad Head, CA (Goldstein et al., 2004) and found no significant differences between the model predictions and observations for all conditions, including those given in the current PRB definition. The Criteria Document (p. 3-49) states that the current model estimates indicate that PRB in the U.S. is generally 0.015 to 0.035 ppm that declines from spring to summer and is generally < 0.025 ppm under conditions conducive to high O_3 episodes. The Criteria Document acknowledges that PRB can be higher, especially at elevated sites in the spring due to stratospheric exchange. However, unusually high springtime O_3 episodes tied to stratospheric intrusion are rare and generally occur at elevated locations and these can be readily identified and excluded under EPA's exceptional events rule (72 FR 13560) to avoid any impact on attainment/non-attainment status of an area.

Third, many of the commenters who raised the concern that EPA's current estimates of PRB were too low and had the impact of exaggerating the risks associated with the current standard ignored the fact that the risk assessment included a sensitivity analysis which showed the potential impact of both lower and higher estimates of PRB or only focused on the impact of higher estimates of PRB. The choices of lower and higher estimates of PRB included in the risk assessment sensitivity analyses were based on the peer-reviewed evaluation of the accuracy of GEOS-CHEM model. As discussed in the Staff Paper (p. 2-54), the Criteria Document refers to a number of GEOS-CHEM publications (Bey et al., 2001; Liu et al., 2002; Martin et al., 2002; Fusco and Logan, 2003, Li et al., 2002; 2005), summarizing their conclusions as "results indicate no significant bias, and agreement to generally within 5 ppbv (parts per billion volume) for monthly mean concentrations at different altitudes"(Criteria Document, p. 3-51 to 3-53). The Criteria Document (p. 3-53) also states "in conclusion, we estimate that the PRB O_3 values reported by Fiore et al. (2003) for afternoon surface air over the United States are likely 10 parts per billion by volume (ppbv) too high in the southeast in summer, and accurate within 5 ppbv in other regions and seasons." These error estimates are based on comparison of model output with observations for conditions which most nearly reflect those given in the PRB definition, i.e., at the lower end of the probability distribution. As discussed in the Criteria Document and Staff Paper, it can be seen that GEOS-CHEM overestimates O_3 for the southeast and underestimates it by a small amount for the northeast. These commenters generally ignored the scientific conclusion presented in the Criteria Document that for some regions of the country the evidence suggests that the model actually overestimates PRB. Thus, the influence of alternative estimates of PRB on risks in excess of PRB associated with meeting the current standard can be to lower or increase the risk estimates. While the choice of estimates for PRB contributes to the

uncertainty in the risk estimates, EPA does not agree that the approach currently used is biased since peer-reviewed evaluations of the model have shown relatively good agreement (i.e., generally within 5 ppb for most regions of the country).

Fourth, with respect to commenters who pointed to the CASAC comment (Henderson, 2006c, p. 12) that EPA could avoid problems about PRB by focusing on risk reductions relative to just meeting the current standard, EPA notes the following. An O_3 risk assessment calculates the risk reductions associated with changing from O_3 concentrations estimated to exist under one scenario to O_3 concentrations estimated to exist under another scenario. For example, we can calculate

- the risk reductions that would result from changing from "as is" (ambient) O_3 concentrations to PRB O_3 concentrations; or
- the risk reductions that would result from changing from O_3 concentrations under the current standard to PRB O_3 concentrations; or
- the risk reductions that would result from changing from O_3 concentrations under the current standard to O_3 concentrations under some more stringent standard.

EPA can avoid using estimates of PRB in its calculations only if all O_3 concentrations in both scenarios are above PRB. Since, by definition, we cannot get O_3 concentrations to be less than PRB concentrations (i.e., we cannot get O_3 concentrations to be lower than what they would be in the absence of human activity), we must know what PRB concentrations are (or have estimates of these concentrations) to know what the lower bound for feasibly reducing O_3 concentrations is.

The only way EPA could avoid using estimates of PRB concentrations in our risk assessment is if we were reasonably sure that all of the O_3 concentrations in both scenarios (i.e., before reduction and after reduction) were above PRB concentrations – i.e., even if we don't know the PRB concentrations, our estimated O_3 concentrations are sufficiently high that we can be reasonably sure they are not below whatever PRB concentrations are. However, in practice, this doesn't happen – i.e., we typically have O_3 concentrations that are sufficiently low that we cannot be sure they are above PRB.

EPA believes that some commenters have misread the CASAC Panel concern "that the current approach to determining PRB is the best method to make this estimation" (Henderson, 2007, p. 2) as a criticism of the use of the GEOS-CHEM modeling approach and/or support for primary reliance on estimates based on remote monitoring sites. However, the CASAC Panel went on to state that one reason for its concern was that the contribution to PRB from beyond North America was uncontrollable by EPA and that "a better scientific understanding of intercontinental transport of air pollutants could serve as the basis for a more concerted effort to control its growth ..." (Henderson, 2007, p. 3). Hence, CASAC's concern appeared to be more with defining what emissions to include in defining PRB, and the role that PRB should play, as compared to the technical question of the best way to estimate PRB levels.

In addition, EPA notes that Dr. Armistead Russell, the atmospheric modeling expert on the CASAC O_3 Panel, stated the following about EPA's current use of PRB in his comments on the second draft Staff Paper:

> In regards to PRB, given the contentious nature of the issue, the use of the PRB should be discussed in Chapter 2, along with other measures. However, their current approach has been peer-reviewed, and is appropriate. They should, before the next analyses, continue to refine and evaluate their approach to setting the PRB, and how it is used in the assessments. For example, might they use the PRB in the roll-back formula? This could be done by analyzing CMAQ simulations where the boundary conditions are set to those conditions representative of PRB conditions (Henderson, 2006c, p. D-48).

Dr. Russell clearly endorsed EPA's current approach to PRB and was discussing potential improvements for the next review of the O_3 NAAQS. EPA notes that much of the spatial variability in O_3 concentrations is due to spatial variability in the distribution of anthropogenic sources of O_3 precursors. This source of variability is not a factor in the PRB simulations using the GEOS-CHEM model. While Dr. Russell suggested that higher resolution models such as CMAQ might be needed, it is not clear that results will be significantly improved by going to the much higher spatial resolution used in regional scale air quality models given the good agreement of GEOS-CHEM estimates with observations.

In conclusion, the model used, GEOS-CHEM has been extensively evaluated and is one of the key models used in major international assessments including the Intergovernmental Panel on Climate Change and Long-Range Transboundary Air Pollution Converntion assessments. As such it is highly suitable for gauging the influence of intercontinental transport on air quality in the U.S. Thus, its predictions reflect state of the science estimates of intercontinental transport. In reviewing the Staff Paper, the atmospheric modeling expert on the CASAC Panel in his comments on how PRB had been estimated using the GEOS-CHEM model concluded that the "current approach has been peer-reviewed, and is appropriate" (Henderson, 2006b, p. D-48).

Finally, in response to the NAM comment that EPA had ignored two studies related to PRB, the first study (Vingarzan, 2004) was considered by EPA and is discussed in the Annex to the final Criteria Document (see p. AX3-136). The other study (Oltmans et al., 2006 – NAM misspelled the author of this study in its comment) was published after completion of the Criteria Document. EPA has already discussed the fact that there is spatial and seasonal variability in PRB in the Criteria Document and Staff Paper and the GEOS-CHEM model runs also show this spatial and seasonal variability.

(21) *Comment:* Some commenters (e.g., Lefohn, API) asserted that the EPA made an administrative (arbitrary) decision in its choice of methods for estimating PRB levels.

Response: EPA extensively evaluated the usefulness of different methods for estimating PRB levels. The methodology for estimating PRB levels was explained in section AX3.9

of the Criteria Document. EPA noted in the Criteria Document and Staff Paper that the GEOS-CHEM model has been extensively evaluated for O_3 and other species. Simulations at a number of relatively remote sites have indicated no significant bias in the model predictions (See comment #20 above). Ozone is a regional pollutant and it is thus highly problematic to separate the regional pollution contribution from PRB. Even the Annapolis Report acknowledges this point – at least for the eastern U.S. However, the same considerations also apply to the western U.S. As noted in the Criteria Document, it is impossible to tell whether or not a given site is subject to regional or continental anthropogenic influence without ancillary measurements. These include measurements of O_3 precursors from which photochemical production rates can be calculated or of tracers of transport from source regions in North America. Either the requisite analyses have not been performed or data are not available for the monitoring sites suggested in the comments. On the other hand, the GEOS-CHEM model has been evaluated with respect to O_3 precursors in addition to O_3 itself.

(22) *Comment:* One commenter (NAM, p. 63) contends that "presentational objectivity requires that EPA accurately and clearly state the proportion of reduced health effects properly attributable to each of the following factors: (a) the increase in EPA's estimate of unit health risks; (b) the proposed reduction in the allowable peak O_3 level; (c) the rollback procedure; and (d) the lowering of the Policy Relevant Baseline." Without such disaggregation, this commenter contends, policymakers and the public will be misled to believe that all or virtually all of the projected health benefits are attributable to the reduction in the standard.

Response: Contrary to the commenter's contention, there is no requirement that EPA has to partition its risk estimates with respect to the factors cited. It is not clear what the commenter is referring to with respect to "the increase in EPA's estimate of unit health risks." Most of the health effects based on epidemiological studies were not included in the 1997 risk assessment, so these are new quantitative estimates, not increased risk estimates. EPA has appropriately discussed the air quality adjustment procedure in the Health Risk TSD and Staff Paper. Similarly, EPA has discussed its new estimates for PRB, which are based on the best available science as discussed in Chapter 2 of the Criteria Document (section 3.7) and has presented the results of sensitivity analyses that show the impacts on the risk estimates of both lower and higher estimates for PRB in several of the risk assessment locations.

(23) *Comment:* One commenter (NAM, p. 29) contends that EPA has used the data from the Adams' clinical studies (Adams 2002, 2003a, 2003b, 2006a, 2006b) for the purpose of estimating individual or population variability in response, which was not the original purpose of the authors. This commenter further asserted that under the IQG EPA must provide an analysis supporting use of data for a purpose other than the original one.

Response: As in the 1997 risk assessment, EPA obtained individual data from several 6.6-hour O_3 controlled human exposure studies from the author. API, the funding sponsor of the Adams studies, urged EPA to use the data from these studies, particularly the most recent study by Dr. Adams in its health risk assessment in its comments on the

draft Staff Paper and draft health risk assessment in January 2006 (API, 2006). EPA obtained the individual data used in the health risk assessment directly from the author and explained that the data would be combined with other individual data from the Horstman, Folinsbee, and McDonnell 6.6-hour O_3 studies. The health risk assessment for lung function responses was reviewed by the CASAC O_3 Panel and there were no objections expressed by CASAC panel members or by Dr. Adams in either his oral or written comments to EPA concerning EPA's use of the Adams data as part of the basis for estimating the exposure-response relationships used in the health risk assessment.

(24) *Comment:* One commenter (API) urged EPA to use a dynamic response model developed by McDonnell to estimate minute-by-minute FEV1 decrements in its health risk assessment based on all of the controlled human exposure study data. This commenter also urged EPA to break the wide range of EVR cutpoints used in the exposure modeling into a number of smaller bins to test the hypothesis that the use of such a wide EVR range biases the overall result. This commenter asserted that the sensitivity of the results to the use of the API model should be evaluated prior to the issuance of the final rule.

Response: The recommendation to replace the lung function exposure-response relationships developed for EPA's risk assessment was first made in August 2006 in a presentation to the CASAC O3 Panel. EPA does not agree that it is appropriate or necessary to change the lung function exposure-response model used in its health risk assessment for the following reasons: (1) the proposed alternative model had not been peer-reviewed or published at the time EPA's assessment was completed; (2) while there are clearly described criteria about the relative adversity of lung function decrements associated with 1- to 8-hour lung function decrements as discussed in the Criteria Document and Staff Paper and in the previous O3 NAAQS review, there is no consensus about the adversity of 1-minute decrements in lung function response, and (3) CASAC reviewed two drafts of the Health Risk TSD and did not recommend that EPA adopt the alternative approach suggested by the commenter. In EPA's judgment the alternative approach recommended by the commenter requires further examination and review and should be investigated for potential use in the next O3 NAAQS review.

(25) *Comment:* EPA assumes that all exposures between 13 and 27 EVR respond as though they were at 20 EVR, substantially overestimating the risk since there are many more 8-hour occurrences of ozone at 13 EVR than at 20 and many more at 20 EVR than at 27. This results in an overestimation of the number of exposures of concern and of the risk estimates. (API, AAM)

Response: The commenter incorrectly asserts that EPA assumed that all exposures between 13 and 27 EVR respond as though they were at 20 EVR. As in the lung function risk assessment conducted in the prior O_3 NAAQS review, EPA matched the exposure-response relationships from controlled human exposure studies where the vast majority of subjects (>99% of subjects) had EVR's in the range of 13 to 27 $l\text{-}min^{-1}m^2$ with 8-hour average exposures corresponding to averaged EVR's in this same range. The individuals in the 6.6-hour controlled human exposure studies did not all have the same EVR and in

fact there were individuals throughout the range from 13 to 27 l-min^{-1}m^{2}. In EPA's judgment there is not adequate data from 6 to 8-hour exposure studies to justify specifying lung function decrements for a more detailed breakdown of EVRs. Therefore, EPA does not agree that there is necessarily an overestimation of the number of exposures at specified exertion levels or that the risk estimates are overstated. In addition, as noted in the response to comment number (23) above, CASAC reviewed two drafts of the Health Risk TSD and did not recommend changing the approach implemented by EPA in its risk assessment.

(26) *Comment:* One commenter contends that information quality principles require that scientific information be presented "within a proper context" and that "a starting point for context would be to compare the severity of respiratory effects from ozone to effects associated with the most relevant of perhaps a dozen confounders" (NAM, p.51)

Response: EPA believes it has provided sufficient context in its discussion of respiratory effects in the Criteria Document and Staff Paper and that there is no specific requirement to make the type of comparison suggested by the commenter. Further, EPA is unaware of scientific evidence that would allow it to present an equivalent difference in allergen exposure, exercise, temperature or humidity to a specified change in ozone levels.

(27) *Comment:* One commenter contends that EPA characterized its risk estimates as "primary" or "secondary" and that EPA's primary estimates were those that tend to support a policy preference for a more stringent NAAQS and its secondary estimates were those that did not. "To adhere to information quality requirements, EPA must accurately and clearly describe what distinguishes between these two classes of risk estimates" (NAM, p. 53). EPA's treatment of alternative risk estimates also show defects in presentational objectivity by "failing to carry forward all risk estimates of similar likelihood – and in this case, giving greater presentational attention to risk estimates of lower likelihood" (NAM, p. 53).

Response: NAM's contention that EPA's risk estimates are characterized as "primary" or "secondary" in the Staff Paper or proposal notice is incorrect. EPA did use the term "base case" for its lung function risk estimates and provided its rationale for distinguishing between its base case and alternative assumptions about the shape of the exposure-response relationship in the proposal notice:

> 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. (72 FR 37858)

EPA strongly disagrees that its rationale for choosing its base case was influenced by a policy preference. As shown in Figures 5-17 and 5-18 in the Staff Paper (pp. 5-88 to 5-91), the pattern and, in many cases, the actual percent reduction in lung function risk is very similar across the base case and alternative assumptions about the shape of the exposure-response relationship.

With respect to the risk estimates for the health outcomes based on epidemiological studies, EPA presented its rationale for presenting certain risk estimates and noted where alternative estimates could be found in the Staff Paper and Risk Assessment TSD. For example, EPA stated in the proposal,

> 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 (72 FR 37859).

In conclusion, EPA did not divide its risk estimates into "primary" and "secondary" categories: EPA clearly articulated its rationale for presenting some risk estimates over risk estimates in the proposal notice; its rationale was not influenced by any policy preference; and EPA discussed where alternative risk estimates could be found in its documents. Finally, the Administrator in his final decision focused on the overall pattern of the risk estimates, which is very similar between the base case estimates presented in the proposal notice and the alternative estimates more fully presented in the Staff Paper and Risk Assessment TSD.

(28) *Comment:* One commenter contends that EPA characterized its risk estimates as "primary" or "secondary" and that EPA's primary estimates were those that tend to support a policy preference for a more stringent NAAQS and its secondary estimates were those that did not. "To adhere to information quality requirements, EPA must accurately and clearly describe what distinguishes between these two classes of risk estimates" (NAM, p. 53). EPA's treatment of alternative risk estimates also show defects in presentational objectivity by "failing to carry forward all risk estimates of similar likelihood – and in this case, giving greater presentational attention to risk estimates of lower likelihood" (NAM, p. 53).

Response: EPA rejects NAM's contention that EPA characterized its risk estimates as "primary" or "secondary" in the Staff Paper or proposal notice. EPA did use the term "base case" for its lung function risk estimates and provided its rationale for distinguishing between its base case and alternative assumptions about the shape of the exposure-response relationship in the proposal notice:

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> 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. (72 FR 37858)

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> In conclusion, EPA did not divide its risk estimates into "primary" and "secondary" categories, EPA clearly articulated its rationale for presenting some risk estimates over over risk estimates in the proposal notice and its rationale was not influenced by any policy preference, and EPA discussed where alternative risk estimates could be found in its documents. Finally, the Administrator in his final decision focused on the overall pattern of the risk estimates, which is very similar between the base case estimates presented in the proposal notice and the alternative estimates more fully presented in the Staff Paper and Risk Assessment TSD.

6. Specific Comments Related to 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 (40 CFR 58.50). 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. The Agency recognized the importance of revising the AQI in a timely manner to be consistent with any revisions to the NAAQS. Therefore, EPA proposed 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 were promulgated. This section responds to comments received on the proposed conforming changes to the AQI.

Comment: EPA received relatively few comments on the proposed changes to the AQI. Three major issues came up in the comments, including: (1) whether the AQI should be revised at all, even if the primary standard is revised; (2) whether the AQI should be revised in conjunction with this rulemaking, or in a separate rulemaking; and, (3) whether an AQI value of 100 should be set equal to or lower than the level of the short-term primary O_3 standard, and the other breakpoints adjusted accordingly. UARG asserted that EPA should not revise the AQI at all, even if EPA does revise the primary O_3 standard. In support of this view, UARG noted that there is no requirement for EPA to set an AQI value of 100 equal to the level of the short-term standard, and cited the 1999 decision to set the an AQI value of 100 for $PM_{2.5}$ equal to 40 $\mu g/m^3$, when the level of the short-term standard was then 65 $\mu g/m^3$. UARG also expressed the view that lowering the ambient concentrations associated with different AQI values would confuse and mislead the public about actual trends in air quality, which UARG asserted are improving. ALA and other environmental groups in a joint set of comments did not support revising the AQI in conjunction with this rulemaking. ALA et al. expressed the view that since EPA did not propose specific breakpoints in its proposed revisions to the AQI, EPA should conduct a separate rulemaking, specifying the proposed breakpoints to allow the public an opportunity to comment on them. Several State agencies, including agencies from Pennsylvania, Wisconsin and Oklahoma, and State organizations, including NACAA and NESCAUM, supported revising the AQI at the same time that the standard is revised. NACAA expressed the view that: "The effectiveness of the AQI as a public health tool will be undermined if EPA undertakes regulatory changes to the ozone NAAQS without simultaneously revising the AQI." (NACAA, p. 5) The Wisconsin Department of Natural Resources (WI DNR) further noted that:

> "…when the 24-hour $PM_{2.5}$ standard was revised, EPA missed an opportunity to adopt conforming changes to the AQI. The Administrator signed the Federal Register notice promulgating a revised fine-particle standard in September 2006, but EPA still has not changed the AQI to reflect the revised standard. We recommend that the AQI be amended to be consistent with the revised ozone and $PM_{2.5}$ standards." (WI DNR, p. 3)

Finally, ALA et al. and NESCAUM expressed the view that an AQI value of 100 should be set at an ambient concentration below the range for the proposed primary standard. These commenters cited the health evidence showing adverse health effects below the proposed range of the standard, the recommended range of CASAC, and also cited the 1999 decision to set an AQI value of 100 for $PM_{2.5}$ equal to 40$\mu g/m^3$ when the level of the short-term standard was 65 $\mu g/m^3$, as support for this view. Most other State commenters supported setting an AQI value of 100 equal to the level of the primary O_3 standard.

Response: Recognizing the importance of the AQI as a communication tool that allows the public to take exposure reduction measures when air quality may pose health risks,

EPA agrees with State agencies and organizations that favored revising the AQI at the same time as the primary standard. EPA also agrees with State agency commenters that its historical approach of setting an AQI value of 100 equal to the level of the revised primary standard is appropriate, both from a public health and a communication perspective.

Both UARG and ALA et al. cite the 1999 AQI rulemaking, which set an AQI value of 100 for $PM_{2.5}$ equal to 40 $\mu g/m^3$ a lower level than the level of the short-term $PM_{2.5}$ standard, as support for their view that an AQI value of 100 does not need to be set at the level of the revised O_3 standard. However, the sub-index for $PM_{2.5}$ was developed using an approach that was conceptually consistent with past practice for selecting the air quality concentrations associated with the AQI breakpoints. The Agency's historical approach to selecting index breakpoints had been to simply set the AQI value of 100 at the level of the short-term standard (e.g., 24 hours) for a pollutant. This method of structuring the index is appropriate in the case where a short-term standard is set to protect against the health effects associated with short-term exposures and/or an annual standard is set to protect against health effects associated with long-term exposures. In such cases, the short-term standard in effect defines a level of health protection provided against short-term risks and thus can be a useful benchmark against which to compare daily air quality concentrations.

In the case of the 1997 $PM_{2.5}$ standards, EPA took a different approach to protecting against the health risks associated with short-term exposures. The intended level of protection against short-term risk was not defined by the 24-hour standard (set at a level of 65 $\mu g/m^3$) but by the combination of the 24-hour and the annual standards working in concert. In fact, the annual standard (set at a level of 15 $\mu g/m^3$) was intended to serve as the principal vehicle for protecting against both long-term and short-term $PM_{2.5}$ exposures by lowering the entire day-by-day distribution of $PM_{2.5}$ concentrations in an area throughout the year. See generally 62 FR at 38668-70 (July 18, 1997). Because the 24-hour standard served to provide additional protection against very high short-term concentrations, localized "hotspots," or risks arising from seasonal emissions that would not be well-controlled by a national annual standard, EPA consequently concluded that it would be appropriate to caution members of sensitive groups exposed to concentrations below the level of the 24-hour standard. EPA also concluded that it would be inappropriate to compare daily air quality concentrations directly with the level of the annual standard by setting an AQI value of 100 at that level. EPA wanted to set the AQI value of 100 to reflect the general level of health protection against short-term risks offered by the annual and 24-hour standards combined, consistent with the underlying logic of the historical approach to establishing AQI 100 levels. Therefore EPA set the AQI value of 100 at the midpoint of the range between the annual and the 24-hour $PM_{2.5}$ standards (i.e., 40 $\mu g/m^3$) in order to reflect the combined role of the 24-hour and the annual $PM_{2.5}$ standards in protecting against short-term risks. Therefore, this approach for defining and AQI value of 100 is conceptually consistent with the proposed decision to set an AQI value of 100 equal to the level of the primary O_3 standard.

Therefore, EPA is revising the AQI for O_3 by setting an AQI value of 100 equal to 0.075 ppm, 8-hour average, the level of the revised O_3 standard. EPA is also revising the following breakpoints: an AQI value of 50 is set at 0.059 ppm, an AQI value of 150 is set at 0.095 ppm; and an AQI value of 200 is set at 0.115 ppm. All these levels are averaged over 8 hours. As indicated in the proposal, these levels were developed by making proportional adjustments to the other AQI breakpoints (i.e., AQI values of 50, 150 and 200). The proportional adjustments were modified slightly to allow for each category to span at least a 0.015 ppm range to allow for more accurate forecasting. So, for example, simply making a proportional adjustment to the level of an AQI value of 150 (0.104 ppm) would result in a level of about 0.092 ppm. Since most of these ranges are rounded to the nearest 5 thousandths of a ppm, that rounding would have resulted in a 0.014 ppm range (i.e., 0.076 to 0.090 ppm). So, the number was rounded upward to the nearest 5 thousandths of a ppm, to allow for at least a 0.015 ppm range for forecasting. The same principle applies to the calculation of an AQI value for 200 (0.115 ppm). EPA believes that the finalized breakpoints provide a balance between proportional adjustments to reflect the revised O_3 standard and providing category ranges that are large enough to be forecasted accurately, so that the new AQI for O_3 can be implemented more easily in the public forum for which the AQI ultimately exists.

B. Secondary O_3 Standards

Public comments on a range of issues related to the proposed secondary O_3 standard options are addressed in the preamble to the final rule and/or in this document. In particular, general public comments related to whether or not the current secondary O_3 standard should be revised are addressed in section IV.B of the preamble and below in section II.B.1. Specific comments addressing basic elements of the standard, including form, averaging times, and level, are addressed in section IV.C of the preamble and below in section II.B.2. Comments on the interpretation of the scientific evidence and EPA's vegetation exposure and risk assessments for O_3 are addressed in sections IV.B and IV.C of the preamble and below in sections II.B.3 and II.B.4.

1. General Comments on Proposed Alternative Secondary O_3 Standards

A large number of comments on the proposed secondary standards for O_3 were very general in nature, basically expressing one of two sharply divergent views: (1) support for revising the current secondary to provide additional protection for vegetation and ecosystems by establishing a separate and distinct cumulative, seasonal secondary standard with a biologically relevant form or (2) support for keeping the secondary standard identical to the 8-hour primary standard, either with revision as proposed, or without any revision. In general, commenters who supported a revised primary standard also supported adopting a distinct cumulative, seasonal secondary standard. Commenters who supported retaining the current primary standard generally also supported retaining the current secondary standard. Many of these commenters simply expressed their views without stating any rationale, while others gave general reasons for

their views but without reference to the factual evidence or rationale presented in the proposal notice as a basis for the Agency's proposed decision.

General comments based on relevant factors that either support or oppose any change to the current O_3 secondary standard are addressed in this section. The biggest difference between the two main groups of commenters related to their views regarding the form of the standard; that is, whether a secondary standard with a form separate and distinct from that of the current or revised primary standard is appropriate, given the information available in this review.

a. Support for Distinct Cumulative, Seasonal Secondary Standard

Many public comments received on the proposal asserted that, based on the available scientific information, the current secondary O_3 standard is insufficient in both form and level to protect vegetation and ecosystems from known or anticipated adverse O_3-induced effects and revisions to the standard are necessary and appropriate. Among those calling for revisions to the current secondary standard are national and local environmental organizations (e.g., Environmental Defense, Appalachian Mountain Club, Rocky Mountain Clean Air Action), individual States and State environmental/public health agencies, State and local air pollution control authorities, NESCAUM, NACAA, Tribal Associations, and the National Park Service (NPS).

(1) *Comment:* These commenters stated that the available science clearly showed that O_3-induced vegetation and ecosystem effects are occurring at and below levels that meet the current 8-hour standard and, therefore, provides a strong basis and support for the conclusion that the current secondary standard is inadequate. In support of their view, these commenters relied on the entire body of evidence available for consideration in this review, including evidence assessed previously in the last review. These commenters pointed to the information and analyses in the Staff Paper and the conclusions and recommendations of CASAC as providing a clear basis for concluding that the current standard does not adequately protect vegetation from an array of O_3-related effects. This group of commenters strongly supported revising the current standard, not only because in their view the available evidence conclusively demonstrates that the current standard is inadequate to protect sensitive vegetation, but also because the Staff Paper provides abundant evidence that it is appropriate to establish an alternative cumulative, seasonal secondary standard that is distinctly different in form from the current or revised primary standard. For example, NESCAUM states that "… the option of equating the ozone secondary NAAQS with the 8-hour primary is inappropriate and clearly not supported by the weight of scientific evidence."

Response: EPA agrees with these commenters that when evaluated as a whole, the information on vegetation and ecosystem effects available in this review supports the need to revise the current standard to provide increased protection from an array of O_3-related effects on sensitive vegetation and ecosystems. For reasons discussed below in sections II.B.2, however, EPA disagrees with these commenters' views that use of a

cumulative form and related averaging time(s) and level of such a standard is appropriate in light of all of the available scientific information.

(2) *Comment:* These same commenters also presented new information for the Administrator's consideration, including a number of "new" studies published after completion of the Criteria Document. These commenters encouraged the Administrator to consider these "new" studies in making his final decision.

Response: EPA notes, as discussed in section I of the preamble to the final rule and in section I above, that as in past NAAQS reviews, it is basing the final decisions in this review on the studies and related information included in the O_3 air quality criteria that have undergone CASAC and public review and will consider newly published studies for purposes of decision making in the next O_3 NAAQS review. In evaluating commenters' arguments, as discussed below in section II.B.3, EPA notes that its provisional consideration of "new" science found that such studies did not materially change the conclusions in the Criteria Document (See further discussion in the Appendix.)

b. Support for Secondary Identical to Current or Revised 8-Hour Standard

The other main group of commenters, which included Exxon-Mobil, AAM, UARG, API, other industry groups, The Annapolis Center for Science Based Public Policy, individual States and other organizations representing local energy, agriculture or business interests, expressed the contrasting view that the limited number of studies published since the last review and addressed in the Criteria Document provided insufficient evidence to support a conclusion different than what was reached in the last review. In addition, these commenters also generally asserted that the evidence that has become available since the last review does not materially reduce the uncertainties that were present and cited by the Administrator in the last review as important factors in her decision to set the secondary standard identical to the revised primary standard.

(1) *Comment:* These commenters asserted that the types of vegetation effects evaluated in the last review have not changed, and that the Criteria Document, Staff Paper, and CASAC have acknowledged that the information that has become available since the last review does not fundamentally change the conclusions reached in the last review. As a result, they argued that the currently available evidence fails to show that revision to the standard is requisite to provide additional protection from these effects. In particular, UARG states that "…the effects of interest now are for all practical purposes, identical to those that EPA considered in the last review" and "…furthermore, recent science has not significantly changed what is known quantitatively about these effects in association with O_3." Thus, UARG asserts that since "the record provides no new scientific insight concerning possible effects of O_3 on vegetation, it supplies no information to call into question the Agency's determination during the last review of the secondary O_3 NAAQS that the existing standard provides the requisite protection to public welfare."

Response: While EPA agrees with the above commenters that the array of known or anticipated vegetation effects evaluated in the last review is generally the same as in the current review, EPA strongly disagrees with the commenters' assertion that the currently available evidence has not materially reduced key uncertainties present in the last review that factored into the Administrator's decision. EPA notes that the information that has become available since the last review, though limited, strengthens the scientific basis supporting the need for revision to the secondary standard. The recent expansion of field-based evidence across a broad array of vegetation effects categories, as discussed in the Criteria Document, Staff Paper, and the preamble to the final rule, has significantly increased EPA's confidence in extrapolating predictions based on chamber studies to the field. It was this uncertainty, e.g. applying chamber results to the field that in part, led the Administrator to conclude that it was not appropriate to go beyond the increased level of protection that would be expected to occur from an 8-hour revised primary and secondary in the 1997 review. There was not, as implied by the above comment, important uncertainties remaining with respect to the types of O_3-induced effects on vegetation at the time of the last review, though significant uncertainties did and still do remain on the significance of these vegetation effects at the ecosystem level. This increased confidence in the likelihood that predicted effects based on open top chamber (OTC) data are and would continue to occur in the field at and below ambient levels was significant in forming EPA's conclusion that revision to the secondary standard to provide increased protection to sensitive vegetation and associated ecosystems, is both necessary and appropriate at this time.

A number of commenters also expressed the view that EPA has overstated conclusions that could be drawn from the vegetation data and in particular, questioned the Administrator's conclusion that the vegetation effects that are occurring should be judged "adverse" to the public welfare. They assert that there remain considerable uncertainties about the degree to which and levels at which O_3 produces adverse effects on the public welfare, as well as the extent to which the proposed revised secondary standard would protect the public welfare from such effects. These commenters make a number of specific points related to this issue of adversity, as described below.

(2) *Comment:* One commenter stated "The extent to which the predicted effects on vegetation from ozone exposure at levels below the current standard would be adverse to the public welfare depends on the intended use of the vegetation and its significance to the public. These factors have not been adequately explored."

Response: EPA agrees that a judgment of adversity regarding any O_3-induced vegetation effects takes into account the intended use of the vegetation and the significance of any O_3-induced impairment of that use to the public (See following comment). However, EPA disagrees that these factors have not been adequately explored. EPA explicitly addressed the relationship between adversity and intended use with respect to the definition of adversity in section IV.A.3 of the proposal notice. EPA further considered in the Staff Paper and described in the proposal notice (72 FR 37902/3) what information could be brought to bear to help inform judgments pertaining to the adversity of various O_3-induced levels of crop yield loss to the public welfare. In

addition, EPA recognized in the proposal notice that "…the level selected is largely a policy judgment as to the requisite level of protection needed, ….and that it is appropriate to weigh the importance of the predicted risks of these effects in the overall context of public welfare protection." Finally, EPA recognized in the proposal notice (72 FR 37903) that "… the public welfare significance of O_3-related 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."

(3) *Comment:* "The Administrator notes that '[T]he Staff Paper concludes that a determination of what constitutes an 'adverse' welfare effect in the context of the secondary NAAQS review' can be viewed in the broader context of 'linkages between stress-related effects …at the species level and at higher levels with an ecosystem hierarchy.' He does not, however, explicitly indicate that he is adopting this revised definition. Nor should he adopt it. The AQC [sic] is clear that we still lack sufficient scientific information to extrapolate meaningfully from effects on individual plants to ecosystem effects."

Response: EPA does not agree that it is inappropriate to expand considerations of adversity to include impacts on the intended use of ecosystems or the associated ecosystem goods and services. Such impacts, at sufficient levels, could clearly prove adverse to the public welfare. The preamble to the final rule states EPA "…believes it is appropriate to continue to rely on the definition of "adverse," discussed in section IV.A.3 of the proposal that imbeds the concept of "intended use" of the ecological receptors and resources that are affected, and applies that concept beyond the species level to the ecosystem level." EPA further believes that such a broad definition is in keeping with the broad definition of welfare effects defined in section 302 (h) of the CAA, which includes effects on many interrelated components of ecosystems, (e.g., soils, water, wildlife, vegetation). Further, as stated in section IV.A.3 of the proposal notice, "…a recent publication …(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 …at the species level and at higher levels within an ecosystem hierarchy." While EPA agrees that there still remains a high degree of uncertainty in assessing the linkages between O_3 effects on vegetation and those at the ecosystem level, including ecosystem services, EPA believes such linkages have and continue to be made, and as such can reasonably be anticipated.

2. Specific Comments on Proposed Alternative Secondary O_3 Standards

a. Form

Comments received following proposal regarding the appropriate form for the secondary standard fell generally into two groups. One group expressed support for a new, cumulative seasonal form while the other group expressed support for retaining the current 8-hour form.

These comments were similar to those raised prior to the proposal during earlier phases of the NAAQS review. Discussions of these comments are included in sections IV.B.2 and IV.C.2 of the preamble to the final rule, and in more detail below.

(1) *Comment:* One group of commenters, including the National Park Service, Environmental Defense, NESCAUM, NACAA, individual States, Tribal Associations, and local environmental organizations, asserted that the weight of scientific evidence was unambiguous with regard to the need for a cumulative form, and specifically supported the proposed W126 exposure index. For example, New York State DEC explained that "…scientific research recognizes that exposure-based indices considering seasonal time period, exposure duration, diurnal dynamics, peak hourly ozone concentrations, and cumulative effects are important when assessing vegetation effects of ozone exposure (Musselman et al., 2006). The W126 exposure index has long been recognized as a biologically meaningful and useful way to summarize hourly ozone data as a measure of ozone exposure to vegetation (Lefohn et al., 1989)." Similarly, Environmental Defense stated "…[f]or reasons amply explained by CASAC and the Staff, neither the existing secondary standard for ozone nor the proposed primary standards are requisite to protect against adverse welfare effects on vegetation and forested ecosystems. CASAC and Staff further amply justified the need for a separate cumulative seasonal welfare standard to protect against these effects, rather than relying solely on the primary standards to provide such protection."

Response: For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

(2) *Comment:* In addition to expressing strong support for the W126 cumulative seasonal form, commenters in this group also expressed serious concerns with EPA's other proposed option of setting the secondary standard equal to a revised primary standard. For example, NPS agreed with CASAC that "retaining the current form of the 8-hour standard for the secondary NAAQS is inappropriate and inadequate for characterizing ozone exposures to vegetation." NESCAUM stated "…we also strongly encourage EPA to avoid the flawed rationale employed in the previous 1997 ozone NAAQS review, i.e., that many of the benefits of a secondary NAAQS would be achieved if the primary NAAQS were attained. This rationale is flawed in at least two ways: first, ozone damage to vegetation persists in areas that attain the primary NAAQS; and second, the relationship between short-term 8-hour peak concentrations and longer-term seasonal aggregations is not constant, but varies over space and time…as EPA notes at 72 FR 37904…. EPA should set a secondary NAAQS on its own independent merits based on adverse welfare effects. Real or perceived relationships between primary and secondary non-attainment areas are irrelevant to setting the appropriate form and level of the secondary NAAQS."

Response: For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

(3) *Comment:* The other set of commenters, including UARG, API, Exxon-Mobil, The Annapolis Center, ASL and Associates, and AAM, did not support adopting an alternative, cumulative form for the secondary standard. Some of these commenters, while agreeing that "directionally a cumulative form of the standard may better match the underlying data," believe that further work is needed to determine whether a cumulative exposure index for the form of the secondary standard is requisite to protect public welfare.

Response: EPA agrees with the commenters who state that a cumulative form of the standard better matches the underlying data but for the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative, seasonal standard.

(4) *Comment:* These commenters also asserted that EPA's analysis of the impact of the nation's O_3 control program for the 8-hour standard on W126 exposures is not scientifically sound due to the use of low estimates of PRB and an arbitrary rollback method that is uninformed by atmospheric chemistry from photochemical models. They argue that EPA must first realistically evaluate the total O_3 reductions that would occur by using a state-of-the-art photochemical model and perform an analysis of the exposure-response data to determine if effects are observed for exposures which do not exceed the 8-hour standard.

Response: EPA disagrees that the methods used are not scientifically sound and has provided a more detailed discussion under section IV.B.2 in the preamble to the final rule and in section II.A.4 of this Response to Comments document. Regarding the remaining uncertainties in the exposure and risk assessments and the reduction in uncertainties since the last review, these comments have already been addressed in section IV.B.2 of the preamble to the final rule and are further discussed in sections II.B.4 below.

(5) *Comment:* These commenters also stated that without producing C-R functions for the 8-hour form of the standard, EPA has failed to show that the current 8-hour standard would provide less than requisite protection. These commenters asserted that substantial uncertainties remain in this review, and that the benefits of changing to a W126 form are too uncertain to warrant revising the form of the standard at this time.

Response: EPA emphasizes that the Criteria Document has reviewed hundreds of studies that support the conclusion that cumulative metrics, such as W126, are the most biologically relevant concentration-based metrics for vegetation available at this time (EPA 2006). EPA also notes that examples of crop concentration-response functions with the 8-hour form were provided (Staff Paper, Figure 7E-1 of Appendix 7E) and refers the reader to section II.B.3 below for a fuller discussion of this topic. For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

(6) *Comment:* This group of commenters also addressed perceived limitations associated with selection of the W126 cumulative form. Commenters asserted that: (a) the W126 form lacks a biological basis, since it is merely a mathematical expression of exposure that has been fit to specific responses in OTC studies, such that its relevance for real world biological responses is unclear; (b) a flux-based model would be a better choice than a cumulative metric because it is an improvement over the many limitations and simplifications associated with the cumulative form; however, there is insufficient data to apply such a model at present; (c) the European experience with cumulative O_3 metrics has been disappointing and now Europeans are working on their second level approach, which will be flux-based; and (d) the W126 form cannot provide nationally uniform protection, as the same value of an exposure index may relate to different vegetation responses; some commenters support adding a second index that reflects the accumulation of peaks at or above 0.10 ppm (called N100). These concerns are addressed in turn below.

Response: (a) With respect to the comment that the W126 index lacks a biological basis, EPA disagrees. EPA concludes that the vegetation effects science provides evidence that exposures of concern to plants are not based on discrete 8-hour periods but on the repeated occurrence of elevated O_3 levels throughout the plant's growing season. The cumulative nature of the W126 is supported by the basic biological understanding of how most plants in the U.S. are most biologically active during the warm season and are exposed to ambient O_3 throughout this biologically active period. In addition, it has been clearly shown in the scientific literature that, all else being equal, plants respond disproportionately more to higher concentrations, though there continues to be no evidence of an exposure threshold for vegetation effects. The W126 sigmoidal weighting function reflects both of these understandings, by not including a threshold below which concentrations are not included, and by differentially weighting concentrations to give greater weight to higher concentrations and less weight to lower ones. In addition, it has been clearly shown in the scientific literature that, all else being equal, plants respond more to higher concentrations, though there continues to be no evidence of an exposure threshold for vegetation effects. The W126 sigmoidal weighting function reflects both of these understandings, by not including a threshold below which concentrations are not included, and by differentially weighting concentrations to give greater weight to higher concentrations and less weight to lower ones. While recognizing that a cumulative, seasonal form is the most biologically relevant way to relate exposure to plant growth response, EPA's reasons for adopting an 8-hour average standard instead of a cumulative seasonal standard are explained in the preamble.

(b) With regard to the comment that a flux-based model would be a better choice, EPA acknowledges that flux models may produce a more accurate calculation of dose to a specific plant species in a specific area at a specific time, when detailed species-specific and site-specific information is known. However, flux calculations require large amounts of data on the physiology of each plant species and the local growing conditions for the growing range of each plant species. The EPA recognizes that the selection of an appropriate form of exposure index that can be nationally applied will necessarily represent a simplification of the multiple factors that can potentially affect specific plant

response across the wide variety of species and ecosystems/conditions that occur within the U.S.

(c) With regard to European dissatisfaction with the performance of a particular cumulative index in use in Europe[6] and growing interest in development of flux-based models, the Staff Paper (Appendix 7A) noted that "because of a lack of flux-response data, a cumulative, cutoff concentration-based (e.g., AOT40) exposure index will remain in use in Europe for the near future for most crops and for forests and seminatural herbaceous vegetation (Ashmore et al., 2004a)."

(d) EPA disagrees with the comment that a W126 form, if it is to be used, must be coupled with a separate N100 index. EPA has found very little research on the N100 index or a coupled approach. The Criteria Document did not evaluate this approach in the discussion of exposure indices (EPA 2006), and the CASAC, after reviewing all the information in the Criteria Document and the Staff Paper, did not recommend an additional N100 index for consideration. Therefore, the EPA has no basis at this time to judge the extent to which such a coupled W126-N100 form would be a better choice than the proposed W126 form. In addition, EPA notes that the W126 form already has built in a weighting scheme that places greater weight on increasing concentrations and gives every concentration of 0.10 ppm and above an equal weight of 1, which is the highest weight in this sigmoidal weighting function. As has already been discussed, there is no threshold of exposure that is considered adverse to all plants under all conditions. By putting such an emphasis on concentrations at or above 0.10 ppm, the commenters seem to imply that there is a sudden shift in importance between concentrations that fall just below and above 0.10 ppm. This has not been shown in the vegetation effects literature. In addition, in areas that typically have no occurrences of peaks above 0.10 ppm, yet still experience high cumulative exposures, it is not clear what purpose an N100 metric would serve, since it would not improve predictions of O_3-induced effects.

With respect to the related concern that foliar injury in not well correlated with the W126 metric alone, EPA is aware that visible foliar injury can be caused by both acute and chronic exposures. The proposal notice states that "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." Clearly, with visible injury that occurs in response to an acute exposure, a 3-month cumulative value will have less meaning. However, as Jacobson, 1977 makes clear, foliar injury effects can also occur under chronic low level exposure conditions.

[6] The AOT40 index used in Europe is a cumulative index that incorporates a threshold at 0.04 ppm (40 ppb). This index is calculated as the area over the threshold (AOT) by subtracting 40 ppb from the value of each hourly concentration above that threshold and then cumulating each hourly difference over a specified window.

Several commenters, however, expressed the view that a W126 coupled with an N100 is necessary to better predict visible foliar injury in the field. EPA notes, however, that none of these commenters makes a recommendation as to what number of N100 is important to protect against different degrees of foliar injury so as to inform judgments concerning what constitutes requisite protection. One commenter, A.S.L. and Associates also mentions numerous other modifying factors that are important in accurately predicting levels of visible foliar injury observable in the field, including soil moisture, diurnal/nocturnal variation in stomatal conductance and detoxification potential. EPA was aware of these confounders. For example, the Criteria Document, Staff Paper, and proposal notice state that "a major confounding effect for O_3 induced visible foliar injury is the amount of soil moisture (local rainfall) available to a plant during the year that the visible foliar injury is being assessed." Given these complexities, it is not clear why this commenter concludes that adding the N100 alone will provide the stability this commenter asserts is needed to make the W126 a more appropriate form for predicting foliar injury. Lacking additional data to the contrary, and given the full discussion of these issues above, EPA disagrees that adding the N100 metric alone would significantly address the complexities and uncertainties that remain related to the relationship between available soil moisture, timing of peak O_3 exposures and plant.

b. Averaging Times

i. Seasonal Window

(1) <u>*Comment:*</u> Only a few commenters specifically stated support for the 3-month seasonal window. In one example, the NPS stated that "…we agree that the maximum consecutive 3-month period within the ozone season is a reasonable averaging time for vegetation in many areas of the country." Other commenters simply stated support for the recommendations of the Staff Paper and CASAC with regard to the W126 form which included a 3-month seasonal window.

<u>*Response:*</u> For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

(2) <u>*Comment:*</u> In contrast, other commenters recommended a longer seasonal averaging time. In particular, the Appalachian Mountain Club stated that "…the ozone season for the secondary standard should range from May to September to fully protect plant and ecosystem health" and further, that "…the standard should be based on the full growing season of a region and this should be re-evaluated over time. Growing seasons are expanding due to climate change."

<u>*Response:*</u> EPA agrees that many plants, including tree species, have growing seasons longer than three months and that the selection of any single seasonal exposure period for a national standard must necessarily 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. For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

ii. Diurnal Window

(1) *Comment:* With respect to the 12-hour diurnal window, the NPS stated that "…for many areas of the country, the daytime 12-hour window is an appropriate period over which to cumulate diurnal ozone exposures. However, as the Staff Paper points out, there is evidence to suggest that in some species in some areas, ozone uptake occurs outside this 12-hour window….Nevertheless, NPS agrees with EPA and CASAC that, for most areas and species, the 12-hour daytime period is sufficient and appropriate to characterize ozone uptake for the cumulative standard." The NPS further noted that "…there may be specific areas (e.g., very dry, hot areas where stomates are primarily open at night) where a different window may better characterize ozone exposure. It may be appropriate to give States discretion, in these instances, to shift or extend the 12-hour window for calculating exposure in these areas."

Response: For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

(2) *Comment:* Some commenters, however, including NESCAUM, NYSDEQ, AMC, and ADEQ, contended that the appropriate diurnal window for vegetation exposure is longer than 12 hours and typically recommend a 24-hour window. Some of these commenters provided additional air quality analyses and cited both published and unpublished sources of data that document the co-occurrence of sensitive species and elevated nighttime exposures. NESCAUM stated that it "…believes the literature on nighttime adverse ozone impacts is strong and can support an ozone secondary NAAQS that encompasses nighttime hours. This is important to the NESCAUM states as elevated nighttime ozone concentrations occur in many [high elevation] locations throughout the region." NESCAUM also noted that the number of daylight hours during EPA's presumed 3-month growing season is greater than 12 hours at the latitudes of the NESCAUM region." The Appalachian Mountain Club stated that "…we strongly disagree with the Staff and Administrator's opinion that more evidence is needed 'about the extent to which this co-occurrence of sensitive species and elevated nocturnal O_3 exposure exists' …to warrant a 24-hour standard." AMC also provided four examples of National Parks and other federal lands where both elevated nighttime O_3 exposure and sensitive species are present, some of which have been identified as showing nocturnal stomatal conductance in the review by Musselman and Minnick (2000). Arizona DEQ stated "…cacti and other desert succulents breathe at night when they lose less water. One of the impacts of ozone exposure identified in the literature is the inability of the guard cells to fully close stomata. If cacti and succulents cannot close stomata during the daytime, the resulting water loss may substantially weaken the plants or directly kill them. In addition, ozone

damage could make stomata stay open overnight for most vegetative species, which exchange gases diurnally. Therefore, ADEQ supports a 24-hour time horizon for the Secondary Standard."

Response: For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

iii. Annual vs. Three Year Average

With respect to comments concerning the annual versus three-year averaging period for the cumulative form, comments are fairly evenly divided between support for an annual and support for a three year average. However, many of those commenters who agreed that a three-year average could be appropriate only did so with the condition that the level of the standard selected should be lower to protect against adverse levels in one year.

(1) *Comment:* Some commenters expressed support only for the annual averaging time in order to provide the appropriate degree of protection from effects occurring to vegetation on an annual basis.

Response: EPA disagrees that only an annual form of the secondary standard could provide the appropriate degree of protection for annual plants. EPA recognizes and agrees that the adverse impact of some O_3-induced vegetation effects are realized within an annual timeframe and are based on the cumulative O_3 exposure that occurs in that same year. These effects can include growth and reproductive effects in annual species, crop yield loss, and foliar injury symptoms on both annuals and perennial species, including trees growing in protected national areas. EPA notes, however, that with regard to crop yield loss, foliar injury symptoms, and tree seedling biomass loss, determining what degree of vegetation impact is adverse, and, therefore, for which appropriate protection is required in any given year, is more uncertain. As discussed in the preamble, EPA believes that appropriate protection for vegetation can be achieved using a 3-year average of the 8-hour average form.

(2) *Comment:* As stated above, many commenters, while preferring an annual standard, also gave conditional support to a three-year averaging time, on the condition that the level of the standard is lowered to achieve the same level of protection judged requisite on an annual basis. For example, NESCAUM stated that "…adverse vegetation damage occurs on an annual basis… If multi-year averaging is employed to promote a more 'stable' NAAQS (as opposed to more stable ecological health), the level should be set lower than what would otherwise have been set for an annual NAAQS. A reduction of the needed annual level by at least one-third can help assure that the intended threshold is not exceeded in individual years."

Response: EPA agrees that the degree of protection provided by a standard is based on the combination of averaging time and level and thus should be considered together in establishing a standard that provides the requisite degree of protection. For the reasons

discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

(3) *Comment:* In contrast, several other commenters, including NC DFR, NC DAQ, Mid-Ohio Regional Planning Commission, IDNR and IDPH, expressed the view that the cumulative, seasonal standard should be based on a 3-year average, rather than a 1-year average, regardless of level, due to concerns regarding the volatility of using a one-year averaging time with the W126 form. For example, Iowa DNR stated "…using only one year of monitoring data to compute the W126 makes it particularly volatile, as infrequent high hourly ozone spikes make it through the sigmoid filter and contribute to the three month sum but frequently occurring low ozone values do not."

Response: EPA agrees that stability in a standard is a desirable trait that confers benefit to the public welfare. Because EPA concluded that the appropriate degree of protection for vegetation can be achieved using a 3-year average form, EPA further concludes that a three year average form is both desirable and appropriate.

c. Level

(1) *Comment:* One group of commenters, which included the National Park Service, Environmental Defense, NESCAUM, NACAA, State, Tribal and local environmental groups, stated support for the CASAC range of 7- 15 ppm-hours. Many of these same commenters further emphasized the lower end of the proposed range as necessary to provide adequate protection for sensitive species. For example, the NPS "…strongly supports CASAC's recommendation that the upper bound of the range for the standard should not exceed a W126 of 15 ppm-hours. Further, the NPS strongly recommends a value for the secondary standard that is representative of the low end of the range recommended by CASAC." Similarly, NESCAUM states that it "…does not support a secondary NAAQS above 15 ppm-hrs…" and "…furthermore, based on observed ozone damage to forests in the NESCAUM region at current ozone levels, a secondary NAAQS of the W126 form towards the lower end of the CASAC-recommended range would provide better protection in the NESCAUM region." The Appalachian Mountain Club stated "…we urge the more protective 7 ppm-hours level, proposed by EPA, be adopted for areas with known sensitive species and areas under special federal protection related to air quality. This protective approach should be used to ensure that Federal Land Managers are able, as directed by Congress, to protect the air quality related values in our National Parks and Forests and Wilderness areas for future generations."

These commenters who recommended a level at the mid- to lower end of the proposed range based their recommendation primarily on four sources of information: (a) field-based evidence of foliar injury occurring on sensitive species at air quality levels well below that of the current standard; (b) the 1996 consensus workshop recommendations for protective levels in terms of cumulative exposures for different vegetation types; (c) CASAC advice and recommendations; and (d) "new" studies published after the close of

the 2006 CD that potentially point to a stronger link between species level impacts and ecosystem response.

Response: EPA notes that in considering what standard is requisite to protect public welfare from known or anticipated adverse effects, judgment is required, based on an interpretation of the evidence and other information, that neither overstates or understates the strength and limitations of the evidence and information nor the appropriate inferences to be drawn. In light of significant remaining uncertainties, described by other commenters below, EPA disagrees that the appropriate level of protection for vegetation lies within the CASAC range. For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard.

The other group of commenters, including Exxon-Mobil, the Annapolis Center, AAM, and State and local organizations, did not support revising the current secondary standard. While many of their comments regarding sources of uncertainties associated with the effects evidence and the exposure and risk assessments have been addressed in section IV.B.2 of the preamble and in sections II.B.3 and II.B.4 below, some of their comments also identify uncertainties regarding the sources of information relied upon by the first group of commenters as support for a level within the range of levels recommended by CASAC.

(2) *Comment:* With regard to the usefulness of foliar injury evidence to inform selection of a level of protection, Exxon-Mobil stated "…EPA has not addressed potential confounders, such as soil moisture, or related the visible foliar injury symptoms to other vegetation effects." Georgia DNR also identified uncertainties associated with visible foliar injury including "…a lack of linkage between visible foliar injury and other vegetation effects, and the inability to quantitatively assess the degree of visible foliar injury that should be judged adverse in all settings and across all species."

Response: In the proposal notice and preamble to the final rule, EPA concluded that foliar injury information available in this review provides compelling evidence that the current standard is inadequate to protect against this vegetation effect. However, EPA also concludes, given the many sources of uncertainty that have been raised regarding using the foliar injury data as a basis for determining an appropriate level of public welfare protection, that the foliar injury data available at this time is insufficient to specifically inform quantitative judgments regarding the selection of an appropriate standard and should only be considered qualitatively.

(3) *Comment:* With regard to comments received stating concern with EPA's and/or CASAC's apparent reliance on the recommendations from the 1996 consensus workshop in selecting a range of levels, both the Annapolis Center and AAM state that "…[i]f the workshop recommendations are to be used for standard setting, then the studies underlying their basis, as well as the method for reaching the recommended values, should be stated in more detail than in the cited workshop summary. The basis for establishing a standard should be transparent and reproducible." More specifically, Exxon-Mobil stated "…this workshop was by invitation only, and documentation is not

available to the public. The reference [sic] cited is short and provides consensus recommendations, but without details as to their basis. In particular, the basis for the recommendation to consider a 2 percent effect level to be significant is not provided. It is not clear how this effect level could be experimentally determined, if it is discernable from biological variability, or what references suggest the potential compounding effect over multiple years, ….” If available, a more detailed report of the consensus workshop should be released. If studies to support the recommendations cannot be provided, then the recommendations should be used with great caution.”

Response: With regard to comments stating concern with EPA’s apparent reliance on the recommendations from the 1996 consensus workshop in selecting a range of levels, EPA agrees that though the workshop participants were asked to review, and thus were aware of, the scientific literature available at the time, as described in both the 1996 O_3 Criteria Document and Staff Paper, the workshop discussion of ranges of levels recommended as protective of different vegetation types for different effects falls short of providing a scientific basis for determining at what level these effects become adverse to the public welfare. In addition, EPA agrees that the scientific basis for establishing a 2% level of biomass loss in tree seedlings as a benchmark of concern to take into account the possibility of compounding is unclear, though there was and continues to be, evidence of the potential for compounding effects to occur over multiple years. As the proposal states (72 FR 37885, 37886) with respect to potential compounding, there is important evidence that “…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)”. However, EPA concludes that significant uncertainty remains as to what level of annual tree seedling biomass loss when compounded over multiple years should be judged adverse to the public welfare. Since tree seedling risk numbers were often compared to this level of biomass loss derived from expert judgment, EPA also notes that this issue adds additional uncertainty in judging the adversity of relative risks of tree seedling biomass loss predicted under different alternative air quality scenarios.

(4) *Comment:* With regard to the strength of the scientific basis for CASAC’s views, the Annapolis Center and AAM stated that “…if ozone monitoring is continued under the current guidelines, the ranges for a seasonal secondary standard must be adjusted upward. Based on the reduction in ozone exposure at plant height of a factor of two compared to that at measurement height, the range should be increased by roughly a factor of two. Neither CASAC nor EPA took this into account in making their recommendations concerning the level of the secondary standard.” AAM also stated “…[i]t is now widely accepted that ozone concentrations at the ‘standard measurement height’ are not the same as the ozone exposures at plant height. When this factor was corrected in the vegetation risk assessment, the SUM06 and W126 exposures at plant height were approximately half those at measurement height.”

Response: With regard to the uncertainty associated with monitor height gradients and its consideration by CASAC and EPA in making recommendations on an appropriate range of levels, EPA notes that the risk assessments conducted by EPA on both tree seedling biomass loss and crop yield loss included an exposure adjustment factor to better account for this issue. As a result, both EPA and the CASAC were fully aware of this issue when making recommendations on a range of levels for the Administrator's consideration. However, the Staff Paper also "…recognizes that a 10% adjustment to hourly monitoring data across the country is a very simple method to deal with a complicated issue." Thus, because there still remains significant uncertainty associated with selecting an appropriate adjustment method and because the impact of this issue on establishing the appropriate relationship between exposures measured at monitor height and predicted adverse levels of plant response for short vegetation (e.g., crops and tree seedlings) has not been fully evaluated, EPA believes caution should be used in selecting a level of air quality that would not be more protective than necessary in areas where the height of vegetation canopies are significantly lower than that of the monitor probe height (e.g., grasslands, croplands).

EPA believes that this important uncertainty should be taken into account in establishing a standard that is not more protective than necessary. In so doing, EPA recognizes that the degree of protection afforded by a standard is determined by the combination of the various elements of a standard, including form, averaging time and level. Thus, EPA believes that this uncertainty can be addressed in part by coupling a standard level in the proposed range with a three year average standard, rather than an annual standard.

(5) *Comment:* The Annapolis Center stated an additional concern that the CASAC made its recommendations based on the draft Staff Paper that contained errors overstating the magnitude of vegetation effects. In particular, the Annapolis Center states "…[t]here were major errors in the translation of the results of the vegetation risk assessment into the second draft Staff Paper, overstating the magnitude of crop loss from ozone….While this was corrected in the final SP, CASAC made its recommendations concerning the secondary standard based on the draft text that contained numerous errors overstating the magnitude of vegetation effects."

Response: EPA disagrees that this error factored significantly into CASAC's recommendations. CASAC considered the entire body of information available during the review to inform its recommendations. In addition, CASAC reviewed the final Staff Paper and submitted an additional letter (Henderson, 2007) restating and in some cases elaborating on their earlier advice to the Administrator on the second draft Staff Paper (Henderson, 2006). Had the change in risk results significantly factored into their reasons for recommending a revised secondary, they would have revised their advice to the Administrator in their final letter following their review of the final Staff Paper to reflect this change. As they did not do so, it can be concluded that their final advice to the Administrator did not depend on the vegetation risk numbers.

3. Specific Comments on the Interpretation of Scientific Evidence

Comments related to the interpretation of the vegetation and ecosystems effects evidence for O_3 are addressed in this section. Incorporating responses contained in Section IV.B.2.a and IV.C.4 of the preamble to the final rule, EPA provides the following responses to specific issues related to the interpretation of the vegetation and ecosystems effects evidence. Comments related to the overall weight placed on the vegetation and ecosystem effects evidence in reaching decisions on the need to revise the current standard and on an appropriate standard level are addressed in the preamble and in Sections II.B.1 and II.B.2.c above.

(1) *Comment:* A number of commenters reference information from "new" studies published after the close of the 2006 Criteria Document in support of their positions, both for and against revision of the secondary standard.

Response: EPA notes that as in past NAAQS reviews, it is basing the final decisions in this review on the studies and related information included in the O_3 air quality criteria that have undergone CASAC and public review and will consider newly published studies for purposes of decision making in the next O_3 NAAQS review. In evaluating commenters' arguments, as discussed below in section II.B.3, EPA notes that its provisional consideration of "new" science found that such studies did not materially change the conclusions in the Criteria Document (See Appendix).

(2) *Comment:* Several commenters questioned EPA's basis for concluding that adverse effects on vegetation would be expected to occur under air quality that met or was below that of the current 8-hour secondary standard. In particular, Exxon-Mobil stated that "EPA is incorrect in concluding vegetation impacts at the current standard" and further that the "…newer field-based evidence EPA cites for ozone impacts on seedlings, saplings and mature trees indicates ozone impacts but at exposures that are likely in exceedence of the current secondary standard." This commenter also asserted that "…the analysis is not transparent because different indices are not directly correlated, but the mean or seasonal ozone exposures provided appear to be sufficiently high that they would include multiple days with an 8-hour daily maximum average exceeding 80 ppb. Therefore, while these studies demonstrate ozone impact, they do not appear to provide additional evidence for adverse vegetation impacts under air quality scenarios meeting the current standard". Exxon-Mobil also provided specific comment on a number of studies cited by EPA in the proposal, including: (a) King et al., 2005; (b) Gregg et al., 2003; (c) Karnosky et al., 1999; and (d) Isebrand et al., 2001, and states that "…[w]hile these studies provide additional support for ozone impact on vegetation, including observance of ozone effects in field settings without chambers, they do not provide support for the conclusion that ambient levels in compliance with the current standard would result in significant ozone impact." The specific comments on each study are presented individually below. The comment regarding transparency is addressed in a separate comment below.

(a) The commenter stated that King et al. (2005) "…provided ozone concentrations as seasonal daily averages (49-55 ppb) and total seasonal sums (81-97 ppm-hrs for 4.5 to 5

months. Given expected daily fluctuations, it is likely that the current secondary standard was exceeded for the ozone levels specified, as would the W126 values. This study does not demonstrate impact from ozone levels at or below the current standard."

(b) The commenter stated that "…the ambient areas where effects were reported had ozone concentrations that likely exceeded the current secondary standard…." However, "…[i]n Gregg et al. the ambient concentrations are NOT presented as exceedance of 8-hour daily average or even as a cumulative metric, but rather as an annual seasonal 12-hour mean ozone…. Whereas the data of Gregg et al. indicate that ambient levels of O_3 are sufficient to produce biomass loss, it does not address if these ambient levels fall above or below the current standard."

(c) The commenter stated that "Karnosky et al. (1999) reports foliar injury in two higher ozone ambient sites and no injury in one low ozone site. Symptoms were worse in FACE plots than in ambient gradient sites. Ozone exposure concentrations were: ambient higher ozone sites - seasonal sum over 12 hr/day = 47-70 ppm hr (5-43% leaves show injury); FACE exposures with symptoms – seasonal sum over 12 hr/day = 57-61 ppm hr, 12 hr seasonal mean = 54-56 ppb, and 24 hr AOT40 = 27-31 ppm-hrs (11-55% leaves show injury); OTC exposures that produced symptoms – seasonal sum over 12 hr/day = 50-60 ppm hr (50 percent leaves show injury of sensitive clones). These exposures are likely to include exceedances of the current secondary standard."

(d) The commenter stated that "Isebrand et al. (2001) reports results for FACE plots of elevated ozone, although the levels in ozone addition treatments are indicated to be representative of current conditions in many areas surrounding midwestern [sic] cities. The exposures show episodic total seasonal exposures of 90 ppm-hr. The five clones studied showed significant variability in response to ozone, with one showing increased growth under elevated ozone. The results of this study support the potential of ozone to impact vegetation, but again the ozone exposure level is sufficiently high to indicate that there were likely multiple exceedances of the current secondary standard."

Response: The commenter is mistaken in its assertion that EPA is claiming that the above named studies, in and of themselves, demonstrate support for concluding that adverse O_3-induced impacts on vegetation would occur at air quality levels that meet or are below the standard. Rather the conclusion reached by EPA regarding these field based studies, as the following excerpts from the proposal notice show, is that the new field based studies provided limited qualitative support to the use of the OTC derived C-R functions to describe plant response in the field. Specifically, the proposal notice states "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)…. 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. 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 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" (72 FR 37886). As a result of this primary conclusion, the proposal then states "[o]n 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" (72 FR 37897). In regard to comments on particular studies, EPA notes the following:

(a) EPA agrees that the findings of King et al., 2005 do not demonstrate effects below the level of the current standard. However, the most important contribution of this study is not the level of exposures that produced the response, but that the response occurring in the field was comparable to that of previous OTC studies, thus strengthening the coherence across the available weight of evidence.
(b) EPA agrees that Gregg et al., 2003 did not directly address whether the ambient concentrations experienced in the study fell above or below the current standard. However, EPA obtained the raw data from the authors in order to calculate the W126 values for those same ambient exposures (Figure 7-17 on page 7-57 of the Staff Paper). From this raw data, EPA observed that significant biomass loss was observed at O_3 air quality ranging from the level of the current standard to 4th highest daily maximum 8-hour averages well below (e.g., 0.072 ppm) the current 8-hr standard.
(c) EPA agrees that while not demonstrating effects below the level of the current standard, Karnosky et al. (1999) demonstrated that across FACE, gradient and OTC studies using the same tree species, the magnitude of O_3-induced effects were similar, increasing EPA's confidence in using OTC C-R functions and strengthening the coherence across the available weight of evidence.
(d) EPA agrees that while not demonstrating effects below the level of the current standard Isebrand et al. (2001) further demonstrated that the response occurring in the field was comparable to that of previous OTC studies, thus strengthening the coherence across the available weight of evidence.

(3) *Comment:* Exxon-Mobil asserted that the "…FACE and gradient studies show that ozone can impact tree species, but specific provision and/or comparison of the concentration-response functions from these studies with those from OTC studies is limited. Thus, these studies, while very informative, do not reduce the uncertainties in the OTC concentration-response functions…. EPA should develop concentration-response functions based upon the newer data in the same form as the existing ones, to allow direct comparison."

Response: Because the C-R functions developed in OTC included multiple exposure levels including charcoal filtered air representing below ambient conditions, ambient and above ambient, it is not currently possible to create directly comparable C-R functions based on the FACE or gradient study data, since they cannot exclude ambient O_3 concentrations. However, in these field studies (e.g., the FACE studies) the observed vegetation response from O_3 exposures at ambient or above was similar to that observed in OTC studies at similar levels of exposure. Though these studies are still limited in scope, it is nevertheless EPA's conclusion that such field-based evidence reduces the uncertainties associated with the C-R functions generated in OTC studies that were noted by the Administrator in the last review. Thus, the current body of evidence increases EPA's confidence in the results from the OTC studies which demonstrate O_3-related effects at exposure levels below that of the current standard.

(4) *Comment:* Exxon-Mobil stated that "[s]everal times in the proposed rule, EPA indicates the potential for a compounding effect of ozone exposures on perennial species, including studies that document carryover effects. (72 Fed. Reg. at 37,898). EPA fails to provide documentation that supports this claim. Neither the proposal nor the CD and SP reference studies that provide evidence of carry-over effects. The multiple-year studies on tree species cited by EPA do not demonstrate a carry-over or compounding effect, but rather that ozone resulted in a similar percent change across study years (Figure 4 in Isebrand et al. 2001; Figure 2 in King et al. 2005; …). EPA should conduct analyses on other available data sets to determine if the results of the two studies cited above are generally representative.

Response: The commenter is mistaken in asserting that EPA has not provided documentation for its claim of the possibility of carry-over effects. EPA directs the commenter to pp. 9-6 and 9-9 of the Criteria Document, 7-6 of the Staff Paper and pp. 72 FR 37886, 37894, 37897, and 37898. For example, the proposal notice (72 FR 37886), in citing back to the Criteria Document states "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."

(5) *Comment:* Exxon-Mobil stated that "[w]hile EPA is correct in stating that the field data indicate some level of visible ozone injury at ambient levels that meet the current

secondary standard (Jacobson, 1977), EPA has not addressed potential confounders, such as soil moisture or related the visible foliar injury symptoms to other vegetation effects. EPA also indicates a major confounder for ozone induced visible foliar injury is soil moisture – with dry periods in local areas decreasing incidence and severity of visible foliar injury (p. 7-61). These same conditions would favor higher ozone levels. Furthermore, the W126 values for these sites are not provided, so it cannot be assessed if the proposed W126 standard would provide additional protection. EPA should provide the comparable W126 values and compare foliar injury at 'non-background' sites with 'background' sites, to better understand the impact of ozone exposures above background levels."

Response: EPA disagrees with the commenter that it has not addressed potential confounders such as soil moisture. EPA discusses this issue pg. 7-61 of the Staff Paper and clearly takes this into account in considering how much weight to put on foliar injury data (see also discussion under Form above). EPA further notes that it assessed incidence of visible foliar injury across a four year period (e.g., 2001-2004). Though not specifically evaluated, within that four year period it is likely that a variety of soil moisture conditions existed, both temporally and spatially. Even so, at no point in that four year period, did incidence of foliar injury drop below 21% for areas that met the level of 0.084 ppm (see Table 7-4, Staff Paper p. 7-64). At the time this analysis was conducted, EPA was still considering using the SUM06 form, so only SUM06 comparisons are included in the table. However, EPA notes that a SUM06 of 25 ppm-hrs is approximately equivalent to a W126 of 21 ppm-hrs so that an approximate comparison could be made.

(6) *Comment:* Exxon-Mobil stated that "EPA also indicates that Morgan et al. 2003 show consistent deleterious effects of ozone exposures on soybean from 1973-2001." The commenter goes on to assert however that "…the Morgan et al. 2003 meta-analysis is not presented by year and provides results by time, so it cannot be used to support the statement of consistent deleterious effects over time. Morgan et al. provide ranges of ozone impact for ozone concentrations in three average daily concentration categories: 30-59 pb [sic], 60-79 ppb, 90-120 ppb. For the highest two categories and the upper range of the lowest category, it is likely that there were multiple exceedences of the current 8-hour secondary standard. EPA should follow its own recommendation included in footnote number 57 on page 37889 and develop new concentration-response functions for new studies that are updated to meet recent air quality conditions."

Response: The commenter is correct in that Morgan et al. (2003) does not list the studies by date. However, in Figure 2 of the paper O_3 is shown to have a negative effect on nearly all the variables measured on soybean. Most importantly, seed yield was consistently lower under elevated O_3 concentrations. This study was not cited to address whether effects were still occurring at the current standard level. It was cited to point out that across many cultivars in studies conducted over many years there is an apparent consistency of negative effects on soybean yield.

(7) *Comment:* Several commenters argued that because the C-R functions used in this review were developed under the NCLAN program which used exposure regimes containing numerous hourly values greater than 100 ppm (Lefohn and Foley, 1992), a concentration response function should be developed that includes components for both the cumulative seasonal ozone exposure and frequency of hourly peaks. For example, Exxon-Mobil states that "Researchers have documented that the NCLAN studies contain numerous hourly values greater than 100 ppb (Lefohn and Foley, 1992). Failure to include these values known to be key determinants of plant effects in the exposure-response function is a significant oversight for a proposed national standard. The number of hourly values greater than 100 ppb …indicates that many of the NCLAN exposures would have multiple exceedences of an 8-hour standard of 80 ppb. Therefore, reliance on NCLAN data to support the conclusion that exposures below an 8-hour standard would result in ozone impact has significant limitations if the response function fails to consider these peak concentrations.

Response: EPA agrees with these commenters that a biologically relevant concentration response function should take into account both cumulative and peak exposures. However, EPA does not agree with the commenters that the W126 alone fails to do so. The W126 was designed to cumulate exposures while giving greater weight to peak concentrations. The Criteria Document and CASAC recommended a cumulative weighted metric (such as W126) as the most appropriate index and did not recommend the addition of N100 or any other modifications to W126.

(8) *Comment:* Exxon-Mobil stated "Despite previous requests, EPA has not provided the concentration-response functions for the 8-hour form of the standard. Therefore, a comparative analysis of the relative protection afforded by an 8-hour form to that provided by the proposed W126 form cannot be completed… EPA has failed to show that the current 8-hour standard would provide less than requisite protection. EPA should develop concentration response relationships using the 8-hour form of the standard for the same datasets that form the basis for the proposed W126 standard. Frequency of peak occurrence should also be included in the analysis. This is the only meaningful way to compare the level of protection afforded by the different standards." This commenter further stated that without such a comparison, EPA fails to show that the currently proposed W126 standard would result in additional protection than the current 8-hour secondary standard.

Response: For the reasons discussed in the preamble, EPA has determined that it is appropriate to adopt an 8-hour average secondary standard instead of a cumulative seasonal standard. In response to the comment that EPA has failed to produce C-R functions in terms of the 8-hour form, EPA points the reader to Appendix 7E (Figure 7E-1) of the Staff Paper that shows the NCLAN crop yield loss data recalculated in terms of the 8-hour form. This graph shows that a 4th highest maximum daily 8-hour average level that corresponds to the protection level used in the last review (e.g., no more than 10% yield loss in 50% of the crop cases) would be close to 0.06 ppm. Therefore, the current 8-hour average secondary standard set at the current level (0.084 ppm) clearly does not provide the level of protection expected and judged to be requisite by the

Administrator in the last review as expressed in terms of a SUM06 of 25 ppm-hrs (approximately equivalent to W126 of 21 ppm-hrs evaluated in this review). Field-based information that shows effects occurring in the field at ambient air quality levels at and below the level of the current 8-hour standard support this conclusion. For example, EPA staff obtained the raw hourly data that produced the results in Gregg, et al., 2003 in order to calculate the W126 values associated with the cottonwood tree seedling biomass loss as graphed in Figure 7-17 on page 7-57 of the Staff Paper. Though the 4th highest maximum daily 8-hour averages were not presented in the Staff Paper, the raw data to calculate this are available in the docket.[7] From this raw data, EPA observed that significant biomass loss was observed at O_3 air quality ranging from the level of the current 8-hour standard to 4th highest daily maximum 8-hour averages well below (e.g., 0.072 ppm) the current 8-hour standard. Taken together with the foliar injury data which occurred at areas meeting and/or below the level of the current 8-hour standard (see Staff Paper p. 7-61 to 7-64; Table 7-4), EPA concluded that significant vegetation effects can occur at air quality levels that meet or are below the level of the current 8-hour, 0.08 ppm secondary standard.

(9) *Comment*: Exxon-Mobil stated that "[t]he basis for a regulatory standard should be transparent." This commenter then identifies three areas where it alleges a lack of transparency: (a) "Inconsistent use and reporting of metrics and, in some cases, reference to unavailable information, result in a general lack of transparency in the science supporting a revised secondary standard. The studies presented differ in metrics and some studies fail to present all metrics. EPA has failed to conduct a re-evaluation of the studies in order to report results in a consistent format….EPA should provide re-analysis of data and reporting in common metrics as appropriate, particularly to support the key points of its rulemaking." (b) "…EPA cites the recommendations of a consensus workshop (Heck and Cowling, 1997) several times. However, this workshop was by invitation only, and documentation is not available to the public. The reference [sic] cited is short and provides consensus recommendations, but without details as to their basis. In particular, the basis for the recommendation to consider a 2 percent effect level to be significant is not provided. It is not clear how this effect level could be experimentally determined, if it is discernible from biological variability, or what references suggest the potential compounding effect over multiple years, given the results of Isebrand et al. (2001) and King et al (2005) cited above…. If available, a more detailed report of the consensus workshop should be released. If studies to support the recommendations cannot be provided, then the recommendations should be used with great caution." (c) "An additional reference cited in the SP (section 7.5.1) as the source of key information on crop and seedling concentration-response, Lee and Hogsett (1996), is not available. This document is an EPA report, and its release was requested in previous comments to EPA. However, it could not be found on a search of the EPA

[7] Data for Figure 7-17 on pg. 7-57 of the final ozone staff paper: The hourly ozone data in this file was used to calculate the 12-hr W126 ozone exposures for the cottonwood trees grown in the New York City area. Docket No. EPA-HQ-OAR-2005-0172-6753. November 29, 2007.

website or a specific search of EPA publications….EPA should also make the Lee and Hogsett (1996) report available to the public"

Response: (a) EPA agrees that different studies have used different metrics for reporting results and that this has resulted in a lack of direct comparability between these studies. However, in some of these studies, the authors have directly compared their results in the field to those from OTC studies and reported that results are similar between plant response in OTCs and in the field. It is this latter point of comparison that EPA felt was most informative in the context of this review. Thus, EPA does not agree that converting all exposure metrics to a common metric for comparability is a critical activity for the purpose of this review.

(b) EPA agrees that insufficient information is available to judge the scientific basis for the consensus statements from the Heck and Cowling, 1997 consensus workshop. The preamble to the final rule states "With regard to comments stating concern with EPA's apparent reliance on the recommendations from the 1996 consensus workshop in selecting a range of levels, EPA agrees that though the workshop participants were asked to review, and thus were aware of, the scientific literature available at the time, as described in both the 1996 O_3 Criteria Document and Staff Paper, the workshop discussion of ranges of levels recommended as protective of different vegetation types for different effects falls short of providing a scientific basis for determining at what level these effects become adverse to the public welfare. In addition, EPA agrees that the scientific basis for establishing a 2% level of biomass loss in tree seedlings as a benchmark of concern to take into account the possibility of compounding is unclear, though there was and continues to be evidence of the potential for compounding effects to occur over multiple years (72 FR 37885, 37886).

(c) EPA notes that the 1996 article by Lee and Hogsett is available in the docket (Docket No. EPA-HQ-OAR-2005-0172-0069. January 25, 2007.)

(10) *Comment:* A number of commenters expressed concern that the possible impact of O_3-related reductions in plant productivity could result in a reduced capacity of vegetation to serve as a carbon sink to mitigate the impacts of rising CO_2 in a changing climate, citing to a "new" study on that topic (Sitch et al., 2007). Many of these same commenters also cited to "new" field-based studies in the Great Smoky Mountain National Park that find a relationship between O_3 exposure, tree stem growth loss, tree water use and stream flow as evidence that current ambient O_3 levels can impact ecosystems and that ecosystems should be afforded protection from such potential effects. For example, some of these commenters note that "new" studies in the Great Smoky Mountain National Park (McLaughlin, et al., 2007a,b) have found that (1) ambient O_3 caused substantial growth reductions in mature trees in a mixed deciduous forest, which was due in part to increased O_3-induced water loss and led to seasonal losses in stem growth of 30 - 50 percent for most species in a high-ozone year; (2) increasing ambient O_3 levels also resulted in depletion of soil moisture in the rooting zone and reduced late-season stream flow in the watershed; and (3) O_3 may amplify the adverse effects of increasing temperature on forest growth and forest hydrology and may exacerbate the effects of drought on forest

growth and stream health. Other "new" research noted by these commenters as supporting EPA's findings that current O_3 exposures cause significant biomass losses in sensitive seedlings of various tree species include a study that predicted up to 31 percent growth loss in aspen in certain areas of its North American range in 2001-2003 (Percy, et al., 2007).

Response: EPA agrees that O_3 exposure can decrease the potential for plants to sequester carbon. This topic is discussed in the Criteria Document, Staff Paper and proposal notice. The proposal states (72 FR 37888/9) "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. 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. With regard to the other effects described in the "new" studies, EPA refers the reader to the Appendix.

4. Specific Comments on the Vegetation Exposure and Risk Assessments

Comments related to the quantitative vegetation exposure and risk assessments conducted for O_3 are addressed in this section. Incorporating responses contained in Section IV.B.2.b and IV.C.4 of the preamble to the final rule, EPA provides the following responses to specific issues related to these assessments. Most comments received on these assessments were from those commenters opposed to revising the current secondary standard, including Exxon-Mobil, UARG, the Annapolis Center, AAM, and some State agencies. Comments related to views regarding the appropriate weight to place on the vegetation exposure and risk assessments in reaching decisions on the need to revise the current standard and on an appropriate standard level are addressed in the preamble and in section II.B.2.a above.

(1) *Comment:* In general, as stated by AAM, these commenters asserted that "there are problems, flaws and concerns with the Staff Paper analysis and vegetation risk assessment that precludes the establishment of a separate secondary standard to protect vegetation.

Response: EPA agrees that there are uncertainties and limitations associated with the vegetation exposure and risk assessments. Comments regarding specific concerns will be addressed individually below. However, EPA disagrees that such limitations would necessarily "preclude the establishment of a separate secondary to protect vegetation."

(2) *Comment:* A number of commenters asserted that EPA used unrealistically low levels of PRB that resulted in an overestimate of risks and benefits associated with just meeting alternative standards. In particular, the Annapolis Center states that "…[t]he same analyses, arguments, and data that apply to consideration of background for the primary standard also apply to the secondary standard."

Response: EPA strongly disagrees with the assertion of these commenters that EPA used unrealistically low levels of PRB, for the reasons documented in section II.A.5 above, and in the preamble to the final rule in section II.B.2.b, which addresses this and other comments related to EPA's approach to estimating PRB and its role in exposure and risk assessments related to the primary standard.

(3) *Comment:* The Annapolis Center and AAM state that "…the global model EPA relied on is not capable of resolving the phenomena that contribute to attainment or nonattainment of secondary ozone standards throughout the country. In fact, even a more refined, higher resolution model, the Community Multi-Scale Air Quality (CMAQ) modeling system… performed poorly in predicting the observed 2001 SUM06 and W126 in the west…. This means that the sources and processes that contribute to substantial levels of both SUM06 and W126 in rural and remote areas in the Western U.S. are not well-captured in state-of-the-art chemical transport models…." These commenters further state that "…[t]he map of 'as is' W127 levels included as Figure 7-6 of the SP shows W126 levels exceeding 7 ppm-hrs throughout many areas of the country…The overall pattern of W126 suggests both a contribution from U.S. man-made emissions and a background component that could involve biogenic emissions, agricultural emissions, long-range transport from non-U.S. sources, and a stratospheric component. In particular, W126 levels greater than 7 ppm-hr exist in many locations throughout the country, including many rural and remote Western sites well-removed from anthropogenic emission source areas. AAM further elaborates that "…[t]hese levels could be explained in part by NOx emissions from soils due to microbial processes. Jaegle, et al. [(2005)], using satellite observations of NO_2 columns, found that soil NOx emissions were twice as high as assumed in the GEOSS-CHEM model. This will result in a misallocation of ozone between man-made and natural sources. The presence of a large soil NOx source in the Great Plains also may help explain the discrepancy between the locations of elevated SUM06 and W126 and the major populated areas of the country. Thus, "EPA and CASAC assume, incorrectly, based on the Fiore et al. 2003 global modeling analysis, that SUM06 or W126 is not confounded by background ozone.

Response: EPA's conclusions regarding the appropriateness of using the GEOSS-CHEM model predictions of PRB are discussed in detail in section II.A.5 above. Because PRB is defined as including those sources of ozone or ozone precursors that are uncontrollable by the U.S., the natural sources of O_3 precursors such as soil NOx concentrations and stratospheric intrusions, as well as long-range transport from non-U.S. sources have already been included in these estimates. Based on the range of PRB values estimated in this review (e.g., 0.015 to 0.035 ppm), EPA calculated the maximum PRB background contribution that would be possible using the 3-month 12-hr W126 exposure index (see

Staff Paper, p.7-22). Using a constant hourly O_3 concentration of 0.035 ppm (the upper end of the PRB range), multiplied by the very low weight assigned by the W126 weighting scheme and cumulated for 12 hours a day for 3 months (92 days), the PRB value is less than 1 ppm-hr. Thus, on this basis, EPA concludes that areas experiencing W126 values of 7 ppm-hrs or greater, as depicted in Figure 7-6 of the Staff Paper, likely have a very low contribution from PRB and a substantial contribution from non-PRB sources. It is unclear why the commenter views long-range transport of anthropogenic precursors of O_3 or O_3 itself from source regions within the U.S. improbable, since the possibility of long-range transport of these pollutants from non-U.S. sources in contributing to PRB has already been acknowledged. On a final note, the study cited by the commenter (e.g., Jaegle et al., 2005) was not evaluated in the Criteria Document or Staff Paper and is considered a "new" study (see discussion under II.B.3, above).

The proposal notice further discusses considerations regarding the potential influence of PRB on the W126 value at high elevation sites and states "[t]he 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…" that are calculated for the maximum 3 month period during the O_3 season.

(4) *Comment:* Another commenter disputed EPA's calculation of the maximum PRB contribution to 3-month, 12-hour W126 values. Specifically, UARG asserts that "…[i]f, however, one takes monitored air quality at the remote Trinidad Head, California monitoring site as indicative of PRB, the contribution to the 3-month, 12-hour W126 could be as much as 3.50 ppm-hour." In a footnote to the comment, UARG explains that this value was arrived at by assuming that two-thirds of the 24-hour W126 value calculated for the 3-month period April to June 2003 would be equal to the 12 hour value.

Response: As EPA has already stated in II.A.5, above, both EPA's experts and CASAC endorsed the use of the peer-reviewed, thoroughly evaluated modeling approach (GEOS-CHEM) described in the Criteria Document as the best approach for estimating PRB levels. The Criteria Document reviewed detailed evaluations of GEOS-CHEM with O_3 observations at U.S. surface sites (Fiore et al., 2002, 2003) and comparisons of GEOS-CHEM predictions with observations at Trinidad Head, CA (Goldstein et al., 2004) and found no significant differences between the model predictions and observations for all conditions, including those reflecting those given in the PRB definition. The Criteria Document (p. 3-49) states that the current model estimates indicate that PRB in the U.S. is generally 0.015 to 0.035 ppm that declines from spring to summer and is generally < 0.025 ppm under conditions conducive to high O_3 episodes. The Criteria Document acknowledges that PRB can be higher, especially at elevated sites in the spring due to

stratospheric exchange. However, unusually high springtime O_3 episodes tied to stratospheric intrusion are rare and generally occur at elevated locations and these can be readily identified and excluded under EPA's exceptional events rule (72 FR 13560) to avoid any impact on attainment/non-attainment status of an area. Thus, EPA concludes that such a high PRB value would be extremely rare, and highly unlikely to occur during the maximum 3-month daytime period evaluated against the level of the W126 standard. If due to stratospheric exchange, this value would be excluded from consideration.

(5) *Comment:* Many commenters also asserted that there was a lack of new information since the last review that would, in their judgment, materially reduce the uncertainties present in the assessments conducted for the last review. For example, the Annapolis Center asserted that "…[s]ome of the most important caveats and uncertainties concerning the exposure and risk assessments for crop yield that were listed in the [1996] proposal included (a) extrapolating from exposure-response functions generated in open-top chambers to ambient conditions; (b) the lack of a performance evaluation of the national air quality extrapolation; (c) the methodology to adjust modeled air quality to reflect attainment of various alternative standard options; and (d) inherent uncertainties in models to estimate economic values associated with attainment of alternative standard. . . . Because of the lack of new data or substantive improvements in the risk assessment, these same issues remain today, contributing a similar degree of uncertainty, as was the case in the prior review."

Response: While EPA recognizes that important uncertainties remain in estimates of vegetation exposure and O_3-related risk to vegetation, especially with regard to O_3-related effects on crop yields, EPA disagrees with comments that assert that the uncertainties in such assessments remain unchanged or generally have not been materially reduced since the last review. EPA discusses each of the uncertainties from the 1997 review identified by the commenters in turn below.

(a) With regard to the uncertainties associated with using the OTC C-R functions, the Annapolis Center further stated that "CASAC characterized the estimates as rough estimates because the open-top chambers alter the ambient microclimate conditions introducing uncertainty. The concern was raised that in these studies, plant response to ozone has been optimized under conditions that do not reflect real-life field conditions….The Panel concluded that 'five years from now, if we do not have the results of research coupling ozone air quality and plant biology under conditions more representative of ambient field conditions, then we will continue to be hampered by our inability to come to consensus on the levels of air quality that are protective of vegetation and ecosystems…' ". In addition, the Annapolis Center states that "…[t]en years have now elapsed, and the same concentration-response functions from the OTC studies of the 1980's are still the only viable data to use to estimate crop loss …. The 1996 CASAC Panel agreed that the estimates of crop loss at that time were highly uncertain. It raised specific issues concerning the procedures and noted that the background estimate of 0.025 ppm was too low and would over-inflate the crop loss estimates."

While EPA agrees that important uncertainties continue to be associated with the use of the C-R functions generated many years ago using OTC studies for crop yield loss, EPA does not agree that the new information available in this review has not reduced the uncertainties identified above and in the last review. EPA concludes that the uncertainties associated with continued use of the C-R functions derived from the NCLAN OTC studies have been materially reduced in three important ways by the recent information. First, and most importantly, as described above and in the Staff Paper and proposal, the results of significant new field based studies (e.g., AspenFACE, SoyFACE and gradient) show that the levels of vegetation response that have been observed in the field are of similar magnitude as those observed in OTC experiments, thus providing qualitative evidence that use of C-R functions generated in OTC would not be expected to overestimate risk to vegetation in the field. Second, the lower estimates of PRB in this review (e.g., 0.015 to 0.035 ppm) now bracket the 0.025 ppm level used as the lowest exposure level representing background in the OTC studies. This further reduces the concern that these C-R functions result in an overestimation of risks in the field. Third, limited information available in this review on the O_3 sensitivities of some crop species (e.g., soybean, cotton) suggests that O_3 sensitivity has not changed significantly in the intervening years. Taking all the above into account, EPA's level of confidence in the applicability of the OTC generated C-R functions to represent ambient conditions in the field has materially increased.

(b) With regard to the lack of a performance evaluation of the national air quality extrapolation, EPA disagrees and notes that there have been advancements in the tools and methods used for such extrapolations and the characterization of associated uncertainties since the last review. With respect to the generation of interpolated O_3 exposure surfaces, EPA employed a different approach than that used in the last review and undertook a quantitative assessment of the uncertainties associated with the use of this method. This uncertainty assessment was accomplished by sequentially dropping out of the interpolation each monitoring site and then recalculating the exposure surface using the remaining monitoring sites. As discussed in the Staff Paper, this method of evaluation may result in a slight overestimation of error and bias for the exposure surface, since dropping out monitors loses information that the interpolation uses in that local area. As another point of comparison, EPA also examined the subset of rural CASTNET sites to illustrate how the interpolation technique predicted air quality in that rural monitoring network. For this subset, the evaluation indicated that in general, the interpolation technique slightly overestimated W126 exposures at relatively low levels and underestimated W126 exposure at relatively high levels. This aspect of the estimation method potentially resulted in an underestimation of the more important risks associated with higher cumulative exposures in some areas. Based on this evaluation, EPA reiterates the conclusion in the Staff Paper that "…the calculation of error and bias metrics for the interpolation represents a notable improvement over the 1996 assessment which did not have such an evaluation." EPA further concludes that in general, the sources and likely direction of uncertainties associated with the exposure and risk assessments have been better accounted for and characterized than in the last review.

(c) With regard to criticisms of the methodology used to adjust modeled air quality to reflect attainment of various alternative standard options, the Annapolis Center further states that "EPA's analysis to date does not evaluate the impact of the nation's ozone control program on W126 in a scientifically sound manner." EPA strongly disagrees and notes that this issue has been raised in the context of both the primary and secondary standards. As noted above in section II.B.2.b, based on information in the Staff Paper (section 4.5.6) and in more detail in a staff memorandum (Rizzo, 2006), EPA concluded that the quadratic air quality adjustment approach used in this assessment generally best represented the pattern of reductions across the O_3 air quality distribution observed over the last decade in areas implementing control programs designed to attain the O_3 NAAQS. While EPA recognizes that future changes in air quality distributions are area-specific and will be affected by whatever specific control strategies are implemented in the future to attain a revised NAAQS, there is no empirical evidence to suggest that future reductions in ambient O_3 will be significantly different from past reductions with respect to impacting the overall shape of the O_3 distribution.

(d) With regard to comments that asserted that inherent uncertainties in models to estimate economic values of crop loss have not been reduced since the last review, EPA acknowledges that while an updated state of the art model, the AGSIM benefits model, was used in this review, substantial uncertainties remain in these estimates of economic crop loss. Further, EPA notes that these estimates were not relied on as a basis for reaching a decision on the need to revise the current standard.

(6) *Comment:* Some commenters also asserted that the estimated exposures and risks associated with air quality just meeting the current standard have not appreciably changed since the last review. These commenters used this conclusion as the basis for a claim that there is no reason to depart from the Administrator's 1997 decision that the current secondary standard is requisite to protect public welfare.

Response: EPA believes that this claim is fundamentally flawed for three reasons, discussed in turn below: (a) it is factually inappropriate to compare quantitative vegetation risks estimated in 1997 with those estimated in the current rulemaking; (b) it fails to take into account that with similar risks, increased certainty in the risks presented by O_3 implies greater concern than in the last review; and (c) it fails to recognize that the Administrator has used these estimates in a supportive role, in light of the significant uncertainties in the exposure and risk estimates, to inform the conclusions drawn primarily from the integrative assessment of the vegetation effects information on whether adverse O_3-induced effects to vegetation observed under ambient O_3 levels allowed under the current standard are sufficiently adverse to the public welfare to warrant a revision of the O_3 standard.

(a) The 1997 risk estimates, or any comparison of the 1997 risks estimates to the current estimates, are irrelevant for the purpose of judging the adequacy of the current 8-hour standard, as the 1997 estimates reflect outdated analyses that have been updated in this review to reflect the current science. In particular, as discussed in section 7 of the Staff Paper, there have been significant improvements to the exposure and risk methods used,

incorporation of additional sources of information, more sophistication in application of approach and methods evaluation/uncertainty characterization that make comparisons inappropriate between the prior and current review.

(b) It is important to take into account EPA's increased level of confidence in use of OTC derived C-R functions to predict risks in the field based on important new field data and the lower estimates of PRB estimated in this review, since in judging the weight to place on quantitative risk estimates it is important to examine not only the magnitude of the estimated risks but also the degree of confidence in those estimates.

(c) Quantitative vegetation risk estimates were not the main basis for EPA's decision in setting a level for the secondary standard in 1997, and they do not set any quantified "benchmark" for the Agency's decision to revise the current standard at this time. The proposal notice made clear that decisions about the need to revise the current standard are mainly based on an integrated evaluation of evidence available across a broad array of vegetation effects, while the more uncertain exposure, risk and benefits estimates were used in a supportive role. Both the Staff Paper and proposal clearly distinguished the roles that these different types of information played in informing the Administrator's proposed decision. The proposal states that "…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." In determining the requisite level of protection, the Staff Paper recognized that it is appropriate to weigh the importance of the predicted risks of these effects in the overall context of public welfare protection, along with a determination as to the appropriate weight to place on the associated uncertainties and limitations of this information. Thus, while EPA is fully mindful of the uncertainties associated with the estimates of exposure, risk and benefits, as discussed above, it judges that these estimates are still useful in providing additional support for his judgment that the current 8-hour secondary standard does not adequately protect sensitive vegetation.

C. Specific Comments Related to Data Handling (Appendix P)

The primary and secondary ozone NAAQS are specified in terms of an indicator (i.e., the specific pollutant to be measured), a level that defines the highest ambient concentration for a monitoring site to be considered to be in attainment with the NAAQS, and a statistical form. To determine whether an ambient monitoring site violates the NAAQS, the relevant portion of the historical record of ambient concentrations of the indicator pollutant at the monitoring site must be selected, evaluated for suitability for making a determination, and if suitable summarized into

a statistic appropriate for comparison to the adopted form and level of the NAAQS. An important aspect of the suitability evaluation is to determine whether the ozone monitoring data set is complete enough to represent the actual air quality situation well enough to allow any determination of current attainment or nonattainment to be made. These steps collectively are referred to as data handling. Precisely how these steps are performed affects the stringency of the NAAQS, so the detailed requirements for these steps are generally proposed and adopted at the same time as the NAAQS. In this case, they were proposed in the form of a new Appendix P to 40 CFR 50, and were discussed in the preamble to the notice of proposed rulemaking.

Appendix P as proposed was a modification of the existing Appendix I which applies to the ozone NAAQS adopted in 1997. The major modification consisted of conforming revisions to reflect that the proposed 3-decimal-places form of the proposed revised 8-hour primary standard simplifies the steps for comparing the monitoring data to the level of the NAAQS (because rounding is no longer needed). This issue is not fully separable from the choice of the level of the NAAQS. Comments on the level issue per se are addressed elsewhere in this document. The second set of modifications were new provisions for all aspects of data handling for the proposed secondary NAAQS using the cumulative peak weighted index (because the 1997 primary and secondary NAAQS were identical and so no special provisions were needed for the secondary NAAQS).

In the preamble, EPA also invited comment on one particular issue related to data completeness, on possible objective criteria for considering ozone levels to have been low during a period of hours or days when ozone monitoring data was not successfully obtained.

Comments regarding data handling for the 8-hour NAAQS were mostly about the issue of using 3 decimal places in the level of the NAAQS. A majority of the comments this issue supported EPA's proposal. Some of the favorable commenters noted that monitor precision allowed rounding to the third decimal place; others argued that the proposal would close an existing "loophole" that allowed communities to claim attainment at concentrations higher than the standard. Other commenters wrote that the standard should not be expressed to three decimal places. These commenters generally asserted, implicitly or explicitly, that ozone monitoring data has too much uncertainty to allow comparison to a pass/fail limit expressed with three decimal places. There were a few comments on other topics, which are included below.

Only a small number of comments were received on the portions of Appendix P that are relevant to a secondary standard non-identical to the primary standard. These generally concerned the proposed data completeness rules, including whether and how to compensate for an incomplete monitoring record.

1. Specific Comments on Data Handling Related to the 8-hour Standard

(1) *Comment:* Specifying the standard to the third decimal place implies a degree of precision in the underlying health effects data that does not exist. EPA recognized this when it adopted the current standard in 1997. It stated, at that time, that it was specifying the standard to two decimal places "in part to reflect uncertainties in the health effects evidence upon which the proposed standard is based," including "the measurement

uncertainty and representativeness inherent in the reported O_3 concentrations used in field and epidemiological studies and the uncertainty in the exposure estimates upon which the quantitative risk estimates have been based" (62 FR 38886). Those uncertainties remain. EPA has not explained why those uncertainties should not lead to the same conclusion reached in 1997.

Response: The body of scientific evidence supporting EPA's current revision of the primary O_3 NAAQS is different than the evidence considered in 1997, so EPA's statements in 1997 cannot be taken to necessarily apply to the newer body of evidence and the new level of the NAAQS. The interpretation of that evidence is discussed in the final rule preamble.

EPA notes that we are not asserting that monitoring precision in the field has broadly and substantially improved since 1997, although improvements have been made. We do not believe that major improvement is a precondition for the adoption of a NAAQS specified with three digits.

EPA also notes that as a practical matter, given current O_3 concentration measuring equipment and reporting practices, it is inevitable that three digits will be used in making comparisons to the NAAQS, effectively making the NAAQS have three significant digits even if nominally expressed with only two. Almost all States report hourly O_3 concentrations with three digits. It is necessary, therefore, for the NAAQS rule to specify how those reported digits will be treated through all the steps leading up to and including the final comparison to the NAAQS. If the NAAQS were to be nominally expressed with only 2 digits, it would still be necessary to specify which final 3-year average values are considered to be in compliance. So, for example, a hypothetical NAAQS of 0.07 ppm functionally would be either 0.074 ppm (if the rule were to specify that the third digit of the final 3-year average value is rounded, as Appendix I specifies) or 0.079 ppm (if the third reported digit of the final 3-year average value were truncated)[8]. That is, the effective level of the NAAQS depends on the combination of the number of digits specified and the data handling and final comparison instructions. EPA believes that explicit use of three digits in the definition of the NAAQS is the more transparent approach, as well as the only approach that allows the NAAQS to be set so that 0.075 ppm is treated as complying and 0.076 ppm is treated as noncomplying.

(2) *Comment:* EPA's ambient air monitoring reference methods specify a precision of two decimal places (0.01 ppm) for O_3 concentrations (40 C.F.R. § 53.20, Table B-1).

Response: The Table cited in CFR represents the minimum performance specifications and the 0.01 ppm precision is literally a pass/fail test to determine whether it is appropriate to employ a method in the field. Actual monitor precision is much better than the pass/fail limit (about 3 percent precision and about 0 percent bias).

[8] In 1997, we considered establishing a NAAQS level of 0.08 ppm and data handling procedures that would result in any values above 0.080 ppm being considered a violation; in effect that would have been setting a NAAQS of 0.080 ppm.

(3) *Comment:* In 1997, EPA also stated its belief that "…expressing the proposed standard to the second decimal place is also consistent with the quality assurance guidelines that indicate the precision for such O_3 measurements shall be within +15%...." Both of those concepts seem valid and appropriate.

Response: The precision limit stated by quality assurance guidelines reflects a maximum limit of tolerance and does not at all represent what is occurring in the field. As of 2006, the precision limit for O_3 has been lowered to 7% from the original 15% benchmark (40 CFR Part 58, p.61304, section 2.3.1.2).

(4) *Comment:* The Agency's calculation of measurement uncertainty is at odds with research showing that instrument imprecision can represent 7-40% of the temporal variation in daily measurements for pollutants and that the population-weighted variation in daily ambient air pollutant levels can be as high as 20% for both instrument imprecision and spatial variability.

Response: The statistics cited by the commenters were taken from an AWMA publication (Wade et al) and were not confined to O_3 monitoring precision and variability but included many other pollutant gases and particulate species. A close examination of the Wade publication (Table 3) shows that the estimated "nugget" parameter for O_3 was approximately 0.10 which the authors define to be the ratio of the standard deviation of the difference between two collocated monitors to the standard deviation of their mean. Since the ratio of the standard deviation of daily maximum 8-hour O_3 to the mean concentration is approximately 0.3 in many urban areas, the nugget value reported by the authors (i.e. 0.10) translates into a coefficient of variation of approximately 3 percent for the mean difference between two collocated O_3 monitors (0.10 X 0.30 about 0.03). The value of 3 percent is very close to the median precision associated with EPA's assessment of the precision of O_3 monitors nationally. The statistics cited that relate to the spatial variability of pollutant concentrations are not relevant to this issue.

(5) *Comment:* The analysis cited by EPA in 1997 (Systems Analysis, International –SAI) concluded that "a conservative estimate for the typical error associated with an O_3 concentration measurement in the real world is on the order of 4 ppb." It is unclear what has changed with respect to O_3 monitor performance since the 1997 assessment. Moreover, questions have been raised about the recent analysis done by EPA staff and its applicability to the older monitors that make up much of the ambient monitoring network. The recent analysis should not, therefore, be taken as resolving the issue of whether to express the 8-hour NAAQS to the thousandth ppm.

Response: EPA is not asserting that the national monitoring network is necessarily producing more accurate data now than in 1997. In fact, subsequent analysis (see comment below) confirms that theoretically derived O_3 measurement error is still approximately 4 ppb, based on the analysis by Mikel (2006).

(6) *Comment:* A number of commenters made reference to the analysis performed by SAI (1997) and a subsequent reanalysis (Mikel 2006) to estimate the "systematic error"

involved in the collection of O_3 data. Since each analysis concluded that "systematic error" for hourly O_3 measurements is approximately 4 ppb, these commenters assumed the term "systematic error" meant that O_3 measurements have an inherent error of 4 ppb for each hour measured during the O_3 season.

Response: The SAI (1997) and Dennis Mikel analysis (used in Cox and Camalier, 2006) independently assessed the potential for measurement uncertainty in the total monitoring system, which consists of combinations of error due to drift, detector non-linearity, precision and instrument calibration, and NIST standard calibration. The term "systematic error" is used in these documents to emphasize that the error estimate reflects all the components of the overall O_3 monitoring system. It was not intended and does not mean that the O_3 monitoring system always produces errors of 4 ppb (or larger). Rather, random errors are centered on zero, thus producing equally probable positive and negative errors, with most errors being less than 4 ppb.

(7) *Comment:* Several commenters argued that EPA should not base its proposal on the Cox and Camalier (2006) analysis. The instrument bias shown has not been proved to be random.

Response: With regard to the Cox and Camalier (2006) simulation analysis, the reference to "random bias", was perhaps misleading and should have been referred to as "random error". Bias connotes a constant deviation between truth and observation which certainly does not apply to O_3 ambient monitors operating in the field. An assessment of the precision and bias of the reported data from the national network of O_3 monitors has shown a median precision to be about 3 to 4% and bias to be about 0%. Since these statistics are based on individual hourly measurements, the precision and bias of an individual 8-hour daily maximum can be no larger and almost certainly smaller due to averaging of errors. Based on the national O_3 performance statistics and additional information regarding the performance of individual O_3 monitors, the simulation study assumed a "random error" of 0.004 ppm to be conservative (larger than necessary) but reasonably representative of an upper bound for a single individual daily maximum 8-hour concentration. As demonstrated in the simulation study, this level of concentration uncertainty has a minimal impact (about 0.001 ppm) on the accuracy of the O_3 design

(8) *Comment:* EPA has access to considerable data on the site-specific precision and bias estimates, such as Langstaff, who reports bias estimates for individual Boston monitors varying between -7 and + 11 % in 2003

Response: The values of precision and bias used in John Langstaff's report were taken from a Quality Assurance assessment report which uses one-sided, upper confidence bound precision and bias indicators intended to trigger a quality assurance response by protecting against calibration issues and instrument malfunctions. While this conservatism may be useful to feed into the exposure models employed by Langstaff, the precision and bias results are overestimated and do not reflect the true uncertainty that is occurring at the particular monitor. Furthermore, the bias noted above by Langstaff represents the very high end of the bias values at individual sites across the country. Most

sites have bias values close to zero. Therefore, the Boston monitor example is unrepresentative of the performance of the network overall.

(9) *Comment:* Regarding quality assurance procedures, one commenter wrote that, "Determination of O_3 NAAQS attainment requires three years of data but of the more than 13,000 hourly observations taken over three years at a typical O_3 monitoring site, less than 0.75% of the data receives significant scrutiny during the attainment determination process. Although statistical approaches utilizing all available precision and audit data are valuable for assessing overall O_3 data quality EPA should seek and investigate concepts that focus on QA measures taken shortly before and after (e.g., 3-7 days) peak 8-hour O_3 concentrations are recorded. This approach would better assess the quality of O_3 data in the higher concentration range which, ultimately, drives the attainment process."

Response: EPA appreciates the comment; however it is outside the scope of this rulemaking.

(10) *Comment:* One commenter suggested that EPA add a site-specific "method detection level" (MDL) requirement to O_3 measurement QA procedures: "this could be similar to the currently proposed MDL test for precursor trace gases11 in which SLT organizations challenge field-based monitors repeatedly with low concentration gases. The instrument's MDL is calculated as the standard deviation of the average response multiplied by the Student's t-test value for the number of test values. By performing this test under "as installed" conditions a more reliable gauge of an instrument's sensitivity is generated. Additionally, monitoring organizations may discover and correct siting- or installation-related problems while performing such tests and thus generate higher quality data."

Response: Although it is not a requirement, monitoring organizations do have the option to report their derived MDL or the vendor MDL to the Air Quality System (AQS). EPA appreciates the comment; however it is outside the scope of this rulemaking.

(11) *Comment:* Regarding data completeness, one commenter generally supported EPA's proposal and also urged EPA to allow State or local air pollution control agencies the flexibility to determine that certain days were not conducive to high O_3 levels, should a monitor not be operational. They stated that the decisions may need to be made on a case-by-case basis, but listed the types of meteorological conditions that are generally not associated with high O_3 levels: days with significant rain between 10 a.m. to 6 p.m.; days with cooler temperatures (< 60°F); days with north winds and frontal passages.

Response: EPA recognizes the comment, however, EPA believes there are too many meteorological conditions to list; therefore, EPA will continue to handle this issue on a case-by-case basis as explained in Appendix P.

(12) *Comment:* One commenter suggested that the rule allow for averaging the monitoring results among O_3 monitors that are located in close proximity to each other in order to determine compliance. The commenter suggests that States have the option to average the

O_3 measurements between monitors that are located within 20 miles of each other [suggested regulatory text included in comment].

Response: EPA disagrees with this comment since local O_3 concentrations may be impacted by local NO_x emissions from sources such as motor vehicles. Thus, local concentrations averaged together may not adequately characterize the true exposure to the local population.

(13) *Comment:* One commenter wrote that the potential impact of stratospheric O_3 intrusion needs to be thoroughly evaluated.

Response: EPA's Exceptional Event Rule handles the impact of stratospheric O_3 intrusion.

(14) *Comment:* In calculation of using raw data (such as computation of eight hour averages or design values) we recommend rounding the results of these calculations instead of truncating them, as truncation may result in low bias in the final design value.

Response: EPA appreciates the comment. However, it has been standard practice since the 1997 NAAQS to report and handle O_3 data by truncating. Consequently, our interpretation of the relationship between reported O_3 concentrations and health effects in epidemiology studies inherently corrects for the small downward bias in multi-hour and multi-day average concentrations. In all or nearly all cases, data available to health effects researchers were truncated to three decimal places when submitted to AQS or stored in state monitoring agency or other data bases. Reports of prevailing multi-hour O_3 concentrations in study areas were also made on the basis of truncated averages of hourly concentrations. Hence, the small downward bias in O_3 statistics that will be compared to the NAAQS is the same as the small bias in nominal O_3 concentrations examined in the assessment of the health studies and in the O_3 risk assessment. The net effect should be nearly the same as if all O_3 concentration averaging were done with rounding.

2. Specific Comments Related to Non-identical Secondary Standard Provisions of Appendix P

(1) *Comment:* Since the proposed W126 method "weights" certain hours more than others, not all missing hours would be of equal impact. The usual standard of 75% data completeness may therefore not be appropriate in this case. EPA should consider this further and address data completeness requirements in a conservative manner.

Response: EPA appreciates the comment. Given the Administrator's final decision to set the secondary standard identical to the primary standard, the comment is moot.

(2) *Comment:* One commenter presented specific recommendations about missing data adjustment and data handling conventions:

In place of Equation 2, the commenter suggests the following missing data substitution scheme for all months with less than 100 percent completeness:

1. For one or two hour missing data gaps, use a simple interpolation scheme.

2. For three or more hour missing data gaps:
   -- Fill in using data from the closest representative monitoring site. "Representative" means that both sites measure O_3 plumes from identical source regions;
   -- If data from a representative monitoring site are not available, then fill in using a technique approved by the administrator.

Response: EPA appreciates the comment. Given the Administrator's final decision to set the secondary standard identical to the primary standard, the comment is moot.

(3) *Comment:* To be consistent with data handling conventions for the primary O_3 NAAQS, NESCAUM recommends the following revisions to section 4.3 of Appendix P to Part 50:

1. 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 15] 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.

2. This requirement is met for the three month period at a monitoring site if O_3 concentrations are available (before substituting for missing data) for at least 90% of the possible index hours with a minimum data completeness in any one month of at least 75% of the possible index hours.

3. Months with W126 values 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-month W126 value, months with less than 75% data completeness (before substituting for missing data) shall be included in the computation if the 3-month W126 value is greater than the level of the standard."

Response: EPA appreciates the comment. Given the Administrator's final decision to set the secondary standard identical to the primary standard, the comment is moot.

D. Comments Related to Monitoring

1. Specific Comments Related to Monitoring and the Primary Standard

(1) *Comment:* Several commenters stated that EPA's revision of the primary ozone NAAQS will be "seriously compromised" unless it is supported by accurate data establishing current ambient levels of ozone. For example, NACAA stated: "The proposed rule, however, ignores the needs that states and localities will have for additional monitors to measure ozone levels in currently under-monitored areas and, in particular, in unmonitored areas that have populations under 350,000. Unless this latter deficiency is corrected in the final rule, the health benefits of EPA's ozone NAAQS revision will likely be limited to those living in MSAs with populations greater than 350,000."(NACAA, p. 9)

Response: EPA recognizes that the issues raised by the commenters are important and will likely require changes in the monitoring requirements to address MSAs with a population lower than 350,000. As noted in section VI of the preamble to the final rule, EPA intends to address these issues as part of its proposed monitoring rule. EPA will likely consider proposed revisions to the minimum monitoring requirements that apply to smaller MSAs, as well as options for EPA Regional Administrators to consider in deviating from such revised minimum monitoring requirements, as is now permitted in certain sections of 40 CFR part 58, Appendix D.

(2) *Comment:* Several commenters raised similar concerns, adding that the current breakpoint of 85 percent of the standard to determine if an ozone monitor is required in an MSA is "sorely inadequate." They also questioned how EPA determines if an area is below the 85 percent threshold without monitoring. While one commenter supports the current breakpoint, they also contend that all areas, including rural areas that have the potential to exceed this breakpoint at least four times per year will need to have a large number of additional ozone monitors in order to demonstrate attainment with a new lower standard.

Response: EPA notes that the current breakpoint of 85 percent of the standard (to require additional monitors in an MSA) provides a margin of safety by increasing minimum monitoring requirements as the design value for an MSA approaches any of the applicable NAAQS. This approach was adopted for O_3 and $PM_{2.5}$ minimum monitoring requirements on October 17, 2006 as part of a comprehensive review of ambient monitoring requirements for all criteria pollutants. (See 71 FR 61236). Specifically for O_3, an 8-hour O_3 design value of 0.068 ppm triggered increased minimum monitoring requirements for an MSA based on the pre-existing primary standard level of 0.08 ppm. With the decision to revise the 8-hour primary standard to a level of 0.075 ppm, the 8-hour O_3 design value that will trigger increased minimum monitoring requirements for an MSA has decreased from 0.068 ppm to 0.064 ppm. Therefore, MSAs with 8-hour design values between 0.064 ppm and 0.067 ppm are now required to increase the number of monitors operating to meet minimum requirements. EPA notes that in practice, however, virtually all of these areas already are operating at least as many monitors as the new requirements based on the revised primary standard, so the number

of monitors which would need to be initiated (or moved from a location of excess monitors) would be negligible. With regard to the specific comment about how EPA could determine if an area is below the 85 percent threshold without monitoring, EPA recognizes that current monitoring requirements do not require O_3 monitors to be added in smaller MSAs in the absence of a O_3 design value, and that this issue has become more important with the decision to revise the level of the primary standard to 0.075 ppm. As a result, EPA intends to issue a proposed monitoring rule in June 2008 and a final rule by March 2009, as stated in the preamble to the final rule, to deal with monitoring issues related to revisions of the primary and secondary standards.

(3) *Comment:* Several commenters advocated for an expanded ozone monitoring network. They wrote that EPA needs to provide adequate funding for monitoring and the development of more appropriate ways to inform and display that data to the public and natural resource managers. One commenter contends that EPA must expand the national ozone monitoring network in areas with populations of less than 150,000 to identify natural background levels and the nature and patterns of domestic and international ozone transport.

Response: As noted elsewhere in this Response to Comments document, EPA intends to issue a proposed monitoring rule in June 2008 and a final rule by March 2009 to deal with monitoring issues related to revisions of the primary and secondary standards. EPA notes that about 400 monitors are presently required in MSAs, but about 1100 are actually operating in MSAs because most States operate more than the minimum required number of monitors. Of these approximately 700 monitors that are operating in excess of minimum requirements, EPA expects that opportunities will exist for relocations of monitors to smaller MSAs that are presently unmonitored, allowing for more efficient use of existing O_3-related resources without increasing the overall size of an already robust O_3 network. EPA also expects to work with the Office of Atmospheric Programs (OAP) to finalize quality assurance upgrades to CASTNET monitors operating primarily in rural areas, to meet some of the objectives (monitoring background levels, transport patterns) noted by the commenter.

(4) *Comment:* The New York State Department of Environmental Conservation noted that the proposed monitoring regulations essentially overlook the issue of the adequacy of the existing ozone monitoring network for determining compliance with the alternate proposed levels. They noted that in the section of the proposed regulations related to ambient monitoring, the EPA states that "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." This commenter noted that this statement is accurate only if the new primary standard remains at or near the current effective standard of 0.084 ppm or if the standard is dropped to as low as 0.060 ppm. At levels in the range that the EPA is proposing, 0.070 - 0.075 ppm, the monitoring network becomes less than adequate….."

Response: As noted elsewhere in this Response to Comments document, EPA intends to issue a proposed monitoring rule in June 2008 and a final rule by March 2009 to deal with monitoring issues related to the revision of the primary and secondary standards, including the specific issue noted by this commenter.

(5) *Comment:* Several comments were received on possible changes to the required O_3 monitoring season if the primary standard is revised downward. For example, The New York State Department of Environmental Conservation stated: "The season for measuring Ozone is selected on a State by State basis on historical data and the standards that the data will be compared to. The season in New York (April through October) is appropriate if the proposed standard is set at a level at or near the current standard. If the standards are dropped to the low end of the proposed standards, it is likely that Ozone concentrations could be elevated in relation to the standards during periods prior to and following the currently designated Ozone season." NESCAUM commented: "Moving to a primary ozone NAAQS of 0.070 ppm or lower may result in the need for additional sites to properly reflect non-urban population exposures. In addition, depending on the final NAAQS level, the ozone (and possibly the PAMS (photochemical assessment monitoring stations)) season may need to be extended. NESCAUM supports efforts that would better characterize public exposure to ozone, and urges that EPA be prepared to provide funding support for states to carry out such efforts."

Response: EPA agrees that the length of the required O_3 monitoring should be reviewed in light of the Administrator's decision to revise the level of the primary and secondary standard. EPA does not agree that the O_3 season issue is linked to a primary standard level of 0.070 ppm or lower, and notes that the revision to a level of 0.075 ppm necessitates such a review as part of the proposed monitoring rule discussed elsewhere in this Response to Comments document. EPA has done a preliminary analysis of 2004-2006 ambient data to address the issue of whether extensions of currently required O_3 monitoring seasons are appropriate in light of the revised primary and secondary O_3 standard level and the revised breakpoints for the AQI. The results of the analysis demonstrated that out-of-season exceedances of the revised primary standard level occurred in eight states during the study period. Additionally, the frequency of days with O_3 concentrations that reached the revised Moderate AQI category (based on a breakpoint of 0.060 ppm) was much greater compared with the frequency of days with concentrations that reached the pre-existing Moderate AQI category (based on a breakpoint of 0.065 ppm). This increased frequency of days with Moderate AQI levels was noted to occur during periods before and after the currently required O_3 seasons. Based on these preliminary analyses, EPA intends to consider changes to the length of the required O_3 season for the coming monitoring rulemaking. Such changes could be based solely on the frequency of exceedances of the revised primary and secondary standards, or could also consider the frequency of concentrations in the Moderate category of the AQI.

2. Specific Comments Related to Monitoring and the Secondary Standard

(1) *Comment:* Several commenters questioned whether the current monitoring system will be sufficient for monitoring a new standard, such as W126, particularly in rural areas. For example, the ACC noted that "there is also the very practical problem of the lack of a rural monitor network to support a W126 standard. Thus, even if evidence is directionally pointing to establishing a cumulative standard, it is premature to do so now." With regard to how to address the lack of a rural monitoring network, Alan Leston noted that "EPA should more fully address the coverage and quality of non-urban ozone monitoring by regulation. As a starting point EPA might require that 10% of a State's ozone monitoring sites (or at least 2 sites, whichever is greater) be located in non-urban areas." The Iowa Department of Natural Resources urged EPA to include monitoring requirements regarding the secondary standard – in particular requiring monitoring in areas that contain ozone-sensitive crops or ecosystems.

Response: In light of the Administrator's decision to revise the 8-hour secondary standard by setting it identical in all respects to the 8-hour primary standard, EPA believes that it is appropriate to consider whether the existing urban-based monitoring requirements are adequate and appropriate to characterize the exposure in more rural areas where O_3-sensitive plant species and more sensitive ecosystems exist, and where resulting vegetation damage would adversely affect land usage. Such areas would likely include public lands that are protected areas of national interest (e.g., national parks, wilderness areas). In consideration of the spatial gaps that currently exist in the rural ozone monitoring network, and to the extent that the existence of such gaps has contributed to the overall uncertainty that exists in the understanding of ambient O_3 levels that occur throughout rural areas, EPA believes that there is merit in considering whether additional monitoring requirements in certain rural areas would help support ongoing ecosystem research studies as well as future reviews of the O_3 NAAQS by providing a more robust data set with which to assess the relationship of vegetation damage to O_3 concentrations. Accordingly, as part of its separate monitoring rulemaking, EPA intends to consider specific requirements for a minimum number of rural monitors per State, with detailed rule language to ensure that States locate such monitors in appropriate areas. For example, these areas could include Federal, State, or Tribal lands characterized by areas of sensitive vegetation species subject to visible foliar injury, seedling and mature tree biomass loss, and other adverse impacts to a degree that could be considered adverse depending on the intended use of the plant and its significance to the public welfare.

(2) *Comment:* NESCAUM recommended that EPA and the states explore together how the CASTNET (Clean Air Status and Trends Network) program might be augmented and made more efficient and cost-effective to enhance the monitoring network, especially in rural areas. Some commenters expressed concern about the quality assurance practices at CASTNET sites with regard to certain aspects of O_3 monitoring. They recommended that EPA upgrade such practices to meet the 40 CFR Part 58 Appendix A quality assurance requirements already followed by the States so that the resulting data could be used in assessing compliance with the revised secondary standard.

Response: EPA agrees that CASTNET sites represent a valuable resource to help provide rural monitoring coverage in support of the revised level of the secondary O_3

NAAQS. EPA notes that O_3 monitoring technology and procedural upgrades have been completed at some of the CASTNET sites, and that such upgrades will be completed at all CASTNET sites by 2009. The resulting O_3 ambient data from the upgraded sites will meet Appendix A requirements as is presently the case for O_3 data from State operated monitors and NPS monitors. These data will be deemed acceptable for NAAQS-comparison objectives and available in the AQS database beginning in 2008.

(3) *Comment:* The National Park Service noted that the level of monitoring in non-urban and natural areas, such as Class I areas in national parks and wilderness areas, is inadequate to identify all areas that might violate a secondary standard. NPS urged EPA to consider alternative monitoring approaches since typical monitoring may not be possible in some areas due to lack of electricity and other restrictions.

Response: While EPA notes the value of non-traditional monitoring approaches to meet certain research oriented objectives, we believe that monitors intended for demonstrating compliance with the primary and secondary NAAQS must employ federally approved reference or equivalent methods to insure that data of known and acceptable quality are used in the designation of areas as attainment or non-attainment. EPA is aware of the potential logistical difficulty of locating monitors in remote areas and that the lack of monitoring data may complicate the assessment of areas with the regard to compliance with the revised level of the secondary NAAQS. Future guidance or rulemaking on O_3 implementation issues may deal with this issue.

III. RESPONSES TO LEGAL, ADMINISTRATIVE, AND PROCEDURAL ISSUES AND MISPLACED COMMENTS

A number of comments were received that addressed a wide range of issues including legal, administrative, and procedural issues. Many legal issues are addressed generally throughout the preamble to the final rule. Specific legal issues are more fully addressed below in section III.A. In addition, a number of comments were submitted related to issues that are not germane to the review of the NAAQS, including implementation issues. Implementation issues are addressed generally in section I.C of the preamble to the final rule. Comments on implementation and other issues that have been categorized as "misplaced" comments are included in section III.B below.

A. Legal, Administrative and Procedural Issues

(1) *Comment:* By law, the Clean Air Scientific Advisory Committee (CASAC) is charged with performing both a scientific review and policy advice function (Clean Air Act, section 109(d)(2)). This means that EPA must be extraordinarily careful in how it listens to CASAC to ensure that it clearly distinguishes scientific insight from policy prescription (NAM, pp. 53-54).

CASAC's primary scientific responsibility is to perform a scientific peer review of EPA's various risk assessment documents, including Criteria Documents and Staff Papers. CASAC is directed to "complete a review of the criteria published under section 108," which requires that air pollution criteria "accurately reflect the latest scientific knowledge." CASAC's primary duty is to ensure that EPA's risk assessment is accurate, clear and unbiased. CASAC is not charged with performing a de novo synthesis of the science, which is EPA's job through preparation of the Criteria Document. Since the Criteria Document should be completely free of policy considerations, there is no reason why CASAC should ever stray into policy matters (NAM, p. 55).

CASAC's review of the EPA Staff Paper is necessarily different, for the Staff Paper is by design a complex mix of science and policy recommendations from Agency staff. In principle, the design of the Staff Paper should make it relatively easy for CASAC to maintain a clear distinction between its scientific review and policy advocacy roles. CASAC does not seem to have adhered to that principle. It is difficult to discern where it is commenting on science and opining about policy (NAM, p. 55).

CASAC's members also are invited to provide the Administrator with their opinions regarding how he ought to exercise his statutory discretion in revising or retaining NAAQS. Because their principal charge is scientific, the public might reasonably expect CASAC members to limit their advice to matters of a strictly scientific nature, as befitting their technical expertise. However, the law does not limit CASAC to advising on matters of science, nor does it constrain them from providing pure policy advice reflecting their personal values and preferences (NAM, p. 56).

The law invites CASAC to provide policy advice several ways. First, it specifies that one member of the committee must represent State air pollution agencies. Like EPA, these agencies are regulatory rather than scientific in nature, function, or organization, and they are populated with personnel who quite reasonably share their agency's (and EPA's) air pollution control mission. Furthermore the act of representation is inherently a stakeholder role (NAM, p. 56).

CASAC members are asked to "recommend to the Administrator any new national ambient air quality standards and revisions of existing criteria and standards as may be appropriate" (section 109(d)(2)(B)). In short, they are invited to speculate as to how they think they would exercise the Administrator's statutory discretion if they were standing in his shoes. Despite the fact that CASAC members all have scientific training and have distinguished themselves in one or more scientific fields, there is nothing scientific about this assignment (NAM, p. 57).

Response: Under section 109(d)(2)(B) of the Clean Air Act, CASAC "shall complete a review of the [air quality criteria] and [the NAAQS] and shall recommend to the Administrator any new national ambient air quality standards and revisions of existing criteria and standards as may be appropriate under section 108 and subsection (b) of this section." Thus CASAC has two primary responsibilities: to review the air quality criteria and advise EPA on appropriate revisions to the criteria, and to review the

NAAQS and advise EPA on appropriate new or revised NAAQS. The Act does not distinguish between these obligations and does not establish different ranking or priority between them; instead they are of equal statutory status.

The Act does not specify how CASAC is to perform its statutory function. For example, it neither requires nor prohibits CASC from conducting a "de novo synthesis" of the science. For many years, as in this review, the method employed by CASAC in discharging its advisory role has been to provide comments, advice, and recommendations to EPA on draft and final versions of the air quality criteria document, Staff Paper, and related exposure and/or risk assessments. In certain cases, as it has here, CASAC has also provided advice and recommendations on a range of potential alternative standards. EPA has found this overall approach to be a very constructive and useful method for CASAC to provide its advice. Since passage of the CAA Amendments in 1977, EPA has carefully considered all such advice and recommendations provided by CASAC.

EPA recognizes that CASAC serves in an advisory capacity and that the judgments required under section 109(b) are made by the Administrator and not CASAC. Advice of the CASAC is available to the Administrator to use as deemed appropriate in making decisions on the proposal and final rulemaking. The Administrator has followed that approach in this rulemaking.

In providing advice and recommendations to EPA on these various documents, CASAC has properly provided both their views on the science as well as their views on appropriate new or revised NAAQS. It is the latter advice, on new or revised NAAQS, that EPA understands NAM to mean by the term "policy advice." Providing advice and recommendations on new or revised NAAQS calls for CASAC to apply its understanding of the science to the criteria in section 109(b) for setting the NAAQS. This by definition means providing advice and recommendations on the public health and welfare policy judgments inherent in judging, for example, what primary standard would be requisite to protect public health with an adequate margin of safety. This is fully appropriate based on the charge to CASAC in the CAA. In general, as in this case, CASAC has provided such advice by recommending a range of possible new or revised standards, as compared to recommending a single standard, recognizing that section 109 calls for the Administrator to exercise his own public health and welfare policy judgment.

In considering CASAC's advice and recommendations EPA fully agrees that it is very important to pay close attention to the content of the advice and weigh it carefully, whether it be advice on the science or advice on policy matters related to new or revised NAAQS. EPA in fact does this, as seen in the many and varied revisions made to the draft documents CASAC has reviewed and commented on, such as the air quality criteria document, the Staff Paper, and the related exposure and risk assessments. It can also be seen in the careful attention paid by the Administrator to CASAC's advice in both the proposed rule and in this final rule. The Administrator has carefully explained where he has accepted CASAC's advice and where he has not, and explained in detail his reasoning.

In general, EPA has found CASAC's science and policy advice to be very important and helpful. The result has been a robust and comprehensive peer review of the various documents noted above, all of which have informed EPA's proposed and final rule in this review. EPA recognizes that the nature of the advice and recommendations from CASAC, whether on the science or on the policy judgments in applying the science to the criteria for the NAAQS, does vary depending whether the focus of the document at issue is on the science, as in the Criteria Document, or on applying the science to the standard-setting criteria of section 109(b), as in the Staff Paper or the proposal. EPA also agrees that advice and recommendations from CASAC are most useful when the science and/or policy basis of the advice are clearly expressed and identified.

In this rulemaking, EPA is confident that it has been able to clearly differentiate CASAC's science advice from the policy advice on the appropriateness of new or revised NAAQS. NAM has not identified examples where it believes EPA has failed to so differentiate, nor examples where CASAC has improperly mixed science and policy in providing its advice. For example NAM has not identified where CASAC's science advice on the air quality criteria was improper because it was based on policy instead of science considerations. Nor has NAM identified any area where it believes CASAC is speculating about new or revised NAAQS, as compared to exercising their judgment. EPA fully agrees that it needs to pay close attention when evaluating the advice of CASAC to properly understand its scientific basis as well as its policy basis; however, NAM has provided no example where EPA has failed to do so or where EPA has confused or misunderstood the basis for CASAC's advice. In fact, this rule indicates that EPA is fully capable of disagreeing with CASAC on its advice and recommendations, and explaining why EPA either was not persuaded or did not agree with CASAC's reasoning or conclusions.

(2) *Comment:* Because it is an independent body outside of the Agency's control, CASAC is exempt from federal information quality guidelines. However, EPA is not exempt when it disseminates or uses information provided by CASAC. EPA cannot simply cite CASAC as a scientific authority without regard for whether their content adheres to applicable information quality standards. Where EPA disseminates covered information obtained from CASAC in a manner that a reasonable person would construe as Agency agreement, EPA must ensure that the information satisfies applicable information quality standards as if the Agency itself had produced the information (NAM, p. 54).

EPA's Notice of Proposed Rulemaking contains numerous subsections in which the input it received from CASAC is summarized. In some places, this input is clearly described as scientific information, subject to applicable information quality guidelines. But in most instances, EPA does not carefully distinguish CASAC's scientific review from its policy advice. This is entirely understandable insofar as CASAC itself did not make these distinctions clear. However, adherence to information quality guidelines is EPA's responsibility and not that of CASAC. Moreover, in its charge to CASAC, EPA did not ask the committee to clearly distinguish between its scientific review and its policy recommendations. For example, EPA did not ask CASAC to apply the Agency's

information quality guidelines, and CASAC didn't do so. Nor did EPA disclose any pre-dissemination review of the input it received from CASAC (or any other third party) to ensure that applicable information quality requirements were met (NAM, p. 57).

To minimize the number of error correction requests they receive, agencies are required by OMB's government-wide information quality guidelines to establish effective procedures for dissemination review. In EPA's own guidelines the Agency states that it has in place sufficient pre-dissemination review procedures to ensure that information quality error is rare. The problem, though, is that none of the documents subject to this RFC contain any text suggesting that pre-dissemination review actually occurred. Information quality and its associated concepts and definitions simply don't appear (NAM, pp. 57-58).

Response: CASAC is a separate entity from EPA and, as such, assesses scientific and other documents produced by EPA independently of Agency oversight. While EPA gives careful consideration to the advice provided by CASAC members, EPA cannot subject CASAC recommendations to information quality standards. In using CASAC panel advice, however, EPA agrees that it is subject to following information quality guidelines.

EPA's Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated does not require the Agency to discuss, separately, whether the pre-dissemination review actually occurred. The Guidelines, rather, provide a process for developing quality actions, of which the pre-dissemination review procedures are a part. This process is also a part of EPA's Action Development Process (ADP). EPA's ADP is a mechanism that assists the Agency in achieving the objectivity and transparency of information used in developing regulations. The stakeholder involvement and consultation that is a part of the ADP are a valuable component of pre-dissemination review. In fact, NAM's comments are also a part of this process to ensure that the information EPA disseminates is of appropriate quality.

Throughout this O_3 NAAQS review there has been public review and comment of drafts of the Criteria Document, the Staff Paper, the risk and exposure assessments, and comment on the proposal notice. In addition, the CASAC meetings were open to the public, and interested parties had the opportunity to submit comments and/or present oral testimony. In EPA's view, this process has ensured a robust review of the science. EPA believes that this lengthy and transparent process is an effective procedure for dissemination review.

The discussions in both the proposed and final rule of the science, the risk and exposure assessment, and the public health and welfare judgments supporting the proposed and final standards make clear not only the basis for the Administrator's decision but also what advice was received form CASAC, and how EPA differentiated between advice on the science and advice on the policy matters relevant to setting the NAAQS. It also shows where EPA disagreed with CASAC and the basis for any such disagreement.

NAM has not identified any example where EPA failed to properly differentiate between science and policy advice from CASAC.

(3) *Comment:* NAM contends that EPA demonstrates presentational bias through selective citation of statements made by the CASAC for the purpose of conveying the impression that CASAC endorsed all or virtually all of the EPA staff analyses. In the first set of examples, NAM contends that EPA demonstrates this presentational bias by citing text from a paragraph that supports its analyses, but does not cite other text in the same paragraph that does not. In the second set of examples that allegedly demonstrate presentational bias, NAM finds important scientific comments from the CASAC O_3 Panel that it contends do not support EPA's interpretation of the data and also contends that these comments do not appear in the NPRM. (NAM, pp. 23-27)

First set of examples:

1. NAM contends that EPA did not fully present the CASAC O_3 Panel's concerns about the utility of time-series mortality studies and whether use of such studies allows us to confidently attribute observed effects specifically to individual pollutants.

2. NAM contends that EPA did not fully present the CASAC O_3 Panel's concerns about the limited information on the specific chemical composition, toxicity and, equally importantly, the population exposure of oxidant pollutants other than O_3.

3. NAM contends that EPA did not fully present the CASAC O_3 Panel's comments concerning inclusion of health endpoints in the risk assessment and the CASAC advice to qualitatively discuss other health endpoints.

Second set of examples:

4. NAM contends that EPA did not fully include in the proposal notice CASAC's comments concerning exposure measurement error which were expressed in CASAC's letter on the final Criteria Document (Henderson, 2006b, pp.3-4) and that in NAM's view these CASAC comments support the view that 1) data from ambient monitors yield upwardly biased estimates of risk, 2) personal exposure cannot be correlated with ambient exposures and thus associations between data from ambient monitors and mortality are spurious, and 3) that short-term studies do not provide supportive evidence that positive associations in time series results are true.

5. NAM contends that EPA did not present the CASAC O_3 Panel's comment that O_3 measurement error would be expected to upwardly bias estimates of risk and have a substantial impact on the ability to detect a threshold of the concentration-response relationship below which no ozone effects are discernible. NAM also contends that this aspect of O_3 measurement error would undermine EPA's risk assessment.

6. NAM contends that EPA did not present in the proposal notice CASAC's comments recommending an alternative approach to calculate risks relative to the current standard which would avoid the problem of estimating PRB.

7. NAM contends that EPA did not present in the proposal notice CASAC's comments on the final Criteria Document (Henderson, 2006b, p. 4) that NAM states support conclusions that "attributing the observed associations between ozone and mortality in time-series studies masks the underlying factor(s) actually responsible" (NAM, p.27) and "If ozone is not the relevant factor, and its control will not serendipitously reduce the relevant factor(s) then the risk reductions predicted by EPA will not be realized" (NAM, p. 27).

Response: EPA strongly denies NAM's contention that it demonstrated presentational bias. As an initial matter, EPA is not required to quote verbatim all of an important comment made by the CASAC O_3 Panel. Doing so in the Staff Paper or NPRM could have the effect of obstructing clear communication of the concepts involved rather than facilitating communication. Most of the examples listed above were comments made by the CASAC O_3 Panel to point out issues that were not fully addressed in the final Criteria Document that it wanted to have addressed in the final Staff Paper. The issues were fully addressed in the final Staff Paper and thoroughly discussed in the NPRM. If these issues had not been fully addressed, the CASAC O_3 Panel would have noted that in its final review of the Staff Paper, but it did not. Instead it praised the final Staff Paper in its March 26, 2007 letter:

> Members of the CASAC Ozone Review Panel were pleased to review EPA's Final Ozone Staff Paper. The members of CASAC and the Ozone Panel were unanimous in their praise of both the *responsiveness of the Agency to our previous recommendations* and of the clarity of this document. (Henderson, 2007) (emphasis added)

With respect to the specific examples cited by NAM, EPA's responses are given below.

First set of examples:
1. Contrary to NAM's contention, EPA fully presented the CASAC O_3 Panel's views on the utility of time-series studies, and addressed this issue in both the Staff Paper and the NPRM. In section 3.4 of the Staff Paper, EPA described important issues related to assessment of epidemiological studies and this included discussion of NAM's examples 1, 5 and 2 above, respectively: the general issue of the utility of time-series epidemiological studies in assessing the risks from exposure to O_3 and other criteria pollutants, as well as related issues about exposure measurement error in O_3 mortality time-series studies and O_3 as a surrogate for the broader mix of photochemical oxidant pollution in time-series studies. EPA further goes on to note that the implications of these three issues for staff conclusions about the adequacy of the current O_3 NAAQS and the identification of options for consideration are considered in Chapter 6.

With regard to whether use of time-series studies allows us to confidently attribute observed effects specifically to individual pollutants, in section 6.3.4.1 of the Staff Paper, on the evidence-based considerations in selecting the level of the standard, staff generally concludes that:

Toward the lower end of the range of O_3 concentrations observed in such studies, ranging down to background levels, however, we conclude 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. (EPA 2007a, p.6-61)

This conclusion is carried over to section II.D.4.a, the discussion of the evidence based considerations from the Staff Paper the level section in the NPRM (72 FR 37875). The uncertainty about whether observed associations remain plausibly related to exposures to ambient O_3 at the lower end of the range was among the uncertainties that were included in the rationale for concluding that a standard level below 0.070 ppm would not be appropriate. In this rationale the Administrator judged that evidence of a causal relationship between adverse health outcomes and O3 exposures becomes increasingly uncertain at lower levels of exposure. (72 FR 37880)

Moreover, EPA highlights this issue in section 6.4 of the Staff Paper as a key uncertainty that merits more research in the future. In this section staff notes:

Most epidemiological studies of short-term exposure effects have been time-series studies in large populations. Time-series studies remain subject to uncertainty due to use of ambient fixed-site data serving as a surrogate for ambient exposures, to the difficulty of determining the impact of any single pollutant among the mix of pollutants in the ambient air, to limitations in existing statistical models, or to a combination of all of these factors. Independent variables for air pollution have generally been measurements made at stationary outdoor monitors, but the accuracy with which these measurements actually reflect subjects' exposure is not yet fully understood. Also, additional research is needed to improve the characterization of the degree to which discrepancy between stationary monitor measurements and actual pollutant exposures introduces error into statistical estimates of pollutant effects in time-series studies. (EPA 2007a, p. 6-89)

2. Contrary to NAM's contention, EPA fully presented the CASAC O_3 Panel's views on the limited information on the specific chemical composition, toxicity and the population exposure of oxidant pollutants other than O_3, and addressed this issue in both the Staff Paper and the NPRM. As noted above, EPA first described this issue in section 3.4 of the Staff Paper. In section 6.3.2 of the Staff Paper, in the discussion of the most appropriate surrogate for ambient photochemical oxidants, EPA again discusses this issue. In section 6.3.6 of the Staff Paper, summary of staff conclusions and recommendations on the primary O_3 NAAQS, staff concludes:

> It is appropriate 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. Based on the available information, and consistent with the views of CASAC and public commenters, we concluded that there is no basis for considering any alternative indicator at this time. Staff 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. (Staff Paper, p. 6-85)

The CASAC O_3 Panel obviously endorsed this assessment because in its letter on the final Staff Paper it notes that the panel, "agreed with the choice of indicator, statistical form and averaging time for the primary Ozone NAAQS suggested by Agency staff." (Henderson, 2007)

However, the CASAC O_3 Panel further noted that "There is an urgent need to fund more research on the effects on sensitive subpopulations of low levels of the photochemical oxidant mixture for which ozone is used as a surrogate. More information on the effects of low levels of oxidant mixtures on public health is essential to inform the future decision-making process." (Henderson, 2007) This view about the need for additional research mirrored the view expressed by EPA Staff in Section 6.4 of the Staff Paper, summary of key uncertainties and research recommendations related to setting a primary O_3 standard, which noted:

> The extent to which the broad mix of photochemical oxidants and more generally other copollutants in the ambient air (e.g., PM, NO_2, SO_2, etc.) may play a role in modifying or contributing to the observed associations between ambient O_3 and various morbidity effects and mortality continues to be an important research question. Ozone has long been known as an indicator of health effects of the entire photochemical oxidant mix in the ambient air and has served as a surrogate for control purposes. A better understanding of sources of the broader pollutant mix, of human exposures, and of how other pollutants may modify or contribute to the health effects of O_3 in the ambient air, and vice versa, is needed to better inform future NAAQS reviews. (Staff Paper, p. 6-88)

In the NPRM, the issue of the limited information on the specific chemical composition, toxicity and the population exposure of oxidant pollutants other than O_3 was discussed in section II.D.1 on the indicator for the primary standard. (72 FR 37872)

3. EPA rejects NAM's contention that EPA did not present the CASAC O_3 Panel's comments in the proposal notice regarding CASAC's advice on health effects which it should discuss qualitatively. In the proposal notice EPA states,

However as noted by CASAC (Henderson, 2007) and in the Staff Paper, there are a number of health endpoints (e.g., increased lung inflammation, …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 exposure to O_3. (72 FR 37856)

With respect to the first part of the CASAC letter quote that EPA omitted, CASAC was simply agreeing that the health endpoints based on epidemiological evidence that EPA included in its risk assessment were appropriate. EPA stated in the proposal notice that "the CASAC O3 Panel concluded "… that the selection of health endpoints for inclusion in the quantitative risk assessment was appropriate" (72 FR 37856)

Second set of examples:
4. EPA rejects NAM's contention that EPA did not fully discuss the issues raised by CASAC with respect to the role of exposure measurement error in O3 mortality time-series studies. EPA does not agree with the set of conclusions that NAM draws from the CASAC comments with regard to whether this issue undermines the use of epidemiological studies in risk assessment. Rather, EPA concludes that until more data on personal O_3 exposure becomes available, 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. As noted in the general response to this comment, evidently the CASAC O_3 Panel agreed with this assessment in the final Staff Paper. Far from downplaying this issue, EPA highlights it again in section 6.4 of the Staff Paper as a key uncertainty that merits more research in the future. (EPA 2007a, p. 6-88) The discussion of measurement error and the overall conclusion about the usefulness of epidemiological studies in estimating population risk can be found in section II.A.3.a, assessment of evidence from epidemiological studies, in the proposal notice (72 FR 37838).

5 and 7. EPA rejects NAM's contentions that EPA did not present the CASAC O_3 Panel's comments that O_3 measurement error would be expected to have a substantial impact on the ability to detect a threshold of the concentration-response relationship below which no ozone effects are discernible and that attributing the observed associations between O3 and mortality in time-series studies masks the underlying factor(s) actually responsible. EPA also disagrees with NAM's contention that this issue undermines EPA's risk assessment.

EPA has thoroughly discussed the issue of measurement error and the impact on the ability to detect a threshold in section 3.4 of the Staff Paper. In this section, EPA notes that:

> …the use of ambient O3 concentrations may obscure the presence of thresholds in epidemiologic studies (CD p. 7-9). Brauer et al. (2002) concluded that surrogate measures of exposure, such as those from centrally located ambient monitors, that were not highly correlated with personal exposures obscured the presence of thresholds in epidemiologic studies at the population level, even if a common threshold exists for individuals within the population. (Staff Paper, p. 3-41)

With regard to whether the issue of exposure measurement error undermines the use of epidemiological studies in risk assessment, EPA reaches a different conclusion than

NAM. EPA concludes that until more data on personal O_3 exposure becomes available, 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. As noted in the general response to this comment, evidently the CASAC O_3 Panel agreed with this assessment in the final Staff Paper. Far from downplaying this issue, EPA highlights it again in section 6.4 of the Staff Paper as a key uncertainty that merits more research in the future. (EPA 2007a, p. 6-88) The discussion of measurement error obscuring population thresholds and the overall conclusion about the usefulness of epidemiological studies in estimating population risk can be found in section II.A.3.a, assessment of evidence from epidemiological studies, in the proposal notice. (72 FR 37838)

6. EPA has included CASAC's comments related to PRB and its suggested alternative approach and EPA's response to this comment above in section II.A.5 (see comment number (20)).

(4) *Comment:* NAM claims that it is a violation of the information quality standard of objectivity to use terms such as "likely" in ways that conflict with their actual use in appropriate context or without clear definition. NAM contends that EPA must establish clear rules and procedures for how such terms will be used based on the following principals: (1) where possible assign quantitative values to statements about likelihood to reduce interpretative heterogeneity, (2) the values assigned by EPA to likelihood statements and probability descriptors must be consistent with both intuition and scientific research about such terms, (3) to adhere to applicable information quality standards EPA must at a minimum make transparent what it means when it uses likelihood statements and probability descriptors, and (4) EPA must have full and complete documentation of what it proposes and subject its work to pre-dissemination review (such as peer review by qualified psychologists).

Response: EPA agrees that where available information provides a basis for assigning quantitative values to probabilistic statements that it is generally appropriate to do so. On the other hand, EPA does not agree that it is appropriate to interpret information in quantitative terms if available information does not provide a basis to do so, which would have the effect of communicating a higher degree of precision than is warranted, or that the Information Quality Guidelines either require such an interpretation or that EPA establish clear rules and procedures for how commonly used probabilistic terms will be used in a NAAQS review. The commenter specifically refers to EPA's use of the term "likely" as an example of a probabilistic statement that should be assigned quantitative values. EPA disagrees. EPA has used this and other similar types of words throughout the relevant documents based on their common definitions when appropriate, in the absence of information that would support a more quantitative statement. In so doing, EPA has been consistent in using such words and has explained the scientific and logical basis for the statements in which such words appear. For example, the word "likely" is intended to convey its common meaning, i.e., having the qualities or characteristics that

make something probable. This meaning reflects a judgment, for which EPA provides a reasoned basis in these documents. NAM has not identified any specific instance in which the Agency's use of terms such as "likely" or "unlikely" is not consistent with the common meaning of these terms. EPA does not agree that the process that NAM suggests would add to the accuracy, clarity, completeness, or unbiased dissemination of information in NAAQS reviews or would be an appropriate use of Agency resources.

(5) *Comment:* One commenter (NAM, pp. 47-49) contends that EPA used default values in its assessments and that this violates information quality principles.

Response: EPA rejects NAM's contention that it used default values in its assessments. More specifically, EPA used the best available scientific information and included distributions based on available scientific and technical data in its exposure and health risk assessments to characterize variability. Further, the commenter offered no specific examples of where EPA used default values.

(6) *Comment:* One commenter (NAM, pp. 20-21, 56 fn. 31) states that EPA needs to draw a distinction between studies included in the last review ("old" studies) and subsequent studies ("new" studies). EPA should discuss old studies only to set the stage for the review of new studies, since the Administrator's charge under section 108 and 109 is to utilize the best available new scientific information. None of the old studies has utility in deciding in this review whether to change the standard, absent some finding of error or other reason to change the prior evaluation of the old study. CASAC's advice includes reference to various studies published between 1988 and 1993, noting that the relevance of these studies is unclear given CASAC's charge to focus on new research.

Response: EPA disagrees with the construct suggested by the commenter, on both legal and scientific grounds. Section 108 calls for the air quality criteria to "accurately reflect the latest scientific knowledge useful in indicating the kinds and extent of all identifiable effects on public health or welfare." EPA implements this charge by reviewing the newest scientific information, and conducting this review not in isolation but by synthesizing and integrating the newest information with the prior scientific knowledge. An integrated synthesis of the entire body of evidence allows all of the evidence to be evaluated in context, without artificially segregating new from old information. It allows EPA to draw the most appropriate implications and conclusions from the evidence when seen as a whole. The commenter's approach would instead call for freezing our understanding of the information gained from the "old" studies, and would not allow EPA to fully evaluate what the old studies teach us in drawing proper implications from the entire body of evidence. The commenter's approach is not grounded in scientific principles, and would interfere with developing the most useful analysis of the evidence. The commenter's approach is neither required nor appropriate.

(7) *Comment:* One commenter (NAM, pp. 45, 65) stated that the overall result of various errors in the risk assessment is that the Administrator has been misled to believe that the risk estimates are much more certain than they actually are. For these and various other errors, the risk assessment should only be used for purposes of a screening level

assessment, and should not be used for making the important public health decision in this review.

Response: In both the preamble and this Response to Comments document EPA has responded to a wide variety of comments on the risk assessment, as well as comments on the Staff Paper and Criteria Document. The exposure and risk assessment, the Staff paper and the Criteria Document have all carefully identified the many sources of uncertainty in the science or the analysis reported. Throughout the development of these documents EPA received comments on many of these same issues, and addressed them appropriately in many cases by recognizing and describing or otherwise characterizing the uncertainties.

As in prior NAAQS reviews, the degree of uncertainty in the science and the analyses developed using the science is a critical component of decision making by the Administrator. In this review the Administrator has been fully aware of all of these sources of uncertainty. He has taken them into account in making provisional decisions in the proposal, as well as in making decisions in the final rule. For example, uncertainties associated with the exposure and risk assessment results was discussed extensively in the proposal (*See e.g*. 72 FR 37851, 37852, 37856, and 37862). The Administrator explained that he took these uncertainties into consideration when evaluating the adequacy of the current standard, and when evaluating what range of standards would be appropriate (72 FR 37871, 37880). As described in the preamble to the final rule, the Administrator has fully considered the uncertainties in the science and in the exposure and risk assessment. Based on this consideration, he has placed primary emphasis on the scientific evidence and implications drawn from it. He has also placed more emphasis on the exposure assessment than the risk assessment. This reflects a reasoned approach that considers and evaluates the uncertainties, and places more weight where there is more certainty and less weight where there is less certainty. The lengthy public process leading to this final rule has provided a full and comprehensive review of both the science and the various analyses based on the science, as well as the type and degree of uncertainties in each of these areas.

(8) *Comment:* One commenter, NAM, submitted a Request for Correction (RFC) concerning certain information it claims was disseminated by EPA in its proposal notice. In its RFC, NAM describes numerous alleged information quality errors in the description, analysis, and presentation of scientific information and information quality errors in the assessment of human health risks. NAM also claims the existence of information quality errors in the consideration of reports from CASAC, in the rollback assumption, and in the description of PRB.

Response: The RFC has been treated as a comment on our proposal notice. EPA has reviewed NAM's RFC and finds that there is no merit to their objections. EPA disagrees with NAM's allegations that EPA has not complied with the requirements of the Information Quality Act or the Agency's policies for ensuring information quality. EPA has responded to NAM's significant comments in the preamble to the final rule or in this document.

(9) *Comment:* Some commenters argued that EPA has not provided any rational justification for disagreeing with CASAC's recommendations, in violation of the requirements of section 307 (d) (ALA et al.).

Response: The EPA disagrees with this characterization of its explanation at proposal, and refers also to its detailed response in sections II.A regarding decisions not to accept CASAC's recommendations regarding the level of the 8-hour standard for O_3.

(10) *Comment:* The standards must protect these vulnerable populations – including persons with heart disease, hypertension, diabetes, and chronic bronchitis or asthma -- with an adequate margin of safety, the same standards that apply to the general population (ALA et al.).

Response: The EPA agrees with this comment, and has carefully considered effects on vulnerable subpopulations in considering whether and how to revise the O_3 NAAQS (See e.g. section II.A.2).

(11) *Comment:* Some commenters (e.g., ALA et al.) argued that EPA must adopt a precautionary approach to the standard setting process, and set standards in a manner that deals with uncertainty not by ignoring uncertain effects but rather by protecting against adverse health effects even where those effects may be uncertain. In support of its argument, the ALA et al. specifically cited ATA III, 283 F. 3d at 369 (EPA must promulgate protective NAAQS even where risks cannot be qualified or precisely identified) and Lead Industries, 647 F. 2d at 1155 (requiring EPA to wait until it can conclusively demonstrate that a particular effect is adverse to human health is inconsistent with the statute's preventative and precautionary orientation).

Response: The EPA agrees generally with this comment, but notes further that a general invocation of precautionary principles does not determine where in the range of reasonable values EPA could establish the level of a standard --in this case, the 8-hour average O_3 standard. Section II.C.4 to the preamble to the final rule explains in detail why it is appropriate to set the level of the 8-hour standard at 0.075 ppm. The EPA notes further that this choice is consistent with case law in the D.C. Circuit (not cited by the commenter) that the Administrator is to carefully examine all of the relevant studies in the record, but need not base the level of the standard on either the highest or lowest value in these studies. Rather, an informed judgment is called for. API, 665 F. 2d at 1187; NRDC v. EPA, 902 F. 2d 962, 970. Section II.C.4 to the preamble to the final rule states the basis for the Administrator's exercise of informed judgment here, in particular, setting the level of the 8-hour standard below the level at which health effects related to O3 have been clearly demonstrated to occur (i.e. at and above 0.080 ppm) in controlled human exposure studies.

Far from "ignoring uncertain effects," the Administrator has focused closely on the issue of "uncertain effects" in determining where the 8-hour level should be set, recognizing the significant uncertainty over whether adverse effects occur from exposure at various

ambient levels of O_3. As discussed above, Lead Industries is not on point as the issue in that case concerned when an effect should be considered adverse, and the authority to determine that an effect was adverse before there was clear evidence that the effect was harmful. Here there is no issue that mortality and serious morbidity are adverse.

(12) *Comment:* Environmental Defense commented that EPA's proposed alternative to set the secondary standards at the same level as the primary standard flouts the recommendations of both CASAC and Staff, and is scientifically unsupportable. The commenter stated that EPA provides no reasoned basis for rejecting CASAC's recommendation on this score, and that EPA concedes that the cumulative seasonal W126 standard is the most biologically relevant way to protect against adverse welfare effects on vegetation. Environmental Defense claims that EPA cites no scientific or other rational relationship between the proposed primary standards and protection of vegetation against adverse welfare effects. Because there is no rational connection between the proposed primary standard and the level of protection needed to protect vegetation against adverse O_3-induced welfare effects, any EPA finding that the primary standards would be sufficient for secondary standards purposes would be arbitrary. The most that EPA asserts is that there could be significant overlap in the areas protected by the 8-hour and W126 forms, and that the proposed primary standards.

Response: EPA agrees that the W126 form is currently the most biologically relevant form available for reflecting vegetation response to O_3 exposures. However, EPA does not agree that a biologically relevant form is a mandatory requirement in setting a secondary standard, under all circumstances. Indeed, EPA has a long history of judging that a secondary standard set identical to the primary standard form and level will be requisite to protect public welfare, based on the entire body of information available at that time. This judgment has often been made, in large part, as a result of the often significant uncertainties that remain regarding what combination of form and level are most appropriate for a secondary standard, as was the case at the conclusion of the last O_3 NAAQS review in 1997. EPA's reasons for adopting an 8-hour average standard are discussed in the preamble.

(13) *Comment:* Environmental Defense commented that EPA's proposal to set a W126 secondary standard in the range of 7-21 ppm-hrs unlawfully and arbitrarily departs from CASAC's recommendation and the supporting science. The commenter stated that the Act and settled principles of administrative law require EPA to provide a reasoned justification for departing from CASAC's recommendation with respect to the upper bound of the proposed W126 standard and EPA has failed to provide such a justification here. The Agency merely asserts that "[g]iven 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" 72 Fed. Reg. 37,903. The commenter claimed that this bare assertion is grossly deficient as a basis for departing from CASAC's recommendation, as described in paragraphs (a) through (c) below.

(a) The commenter asserted that EPA fails to identify its basis for claiming uncertainty in determining the risk attributable to various O_3 levels or explain why the degree of alleged uncertainty is so great as to render the findings of adverse effects at those levels improbable. The agency cites no facts supporting the claim of uncertainty and does not explain any rational connection between the degree of alleged uncertainty and specified levels of the standard. The agency does not show, nor does the record support a finding that uncertainty of the evidence of adverse effects at 15 ppm-hours is materially greater than at 21 ppm-hours, or that any alleged uncertainty of adverse effects at 15 ppm-hours is so great as to render adverse effects at that level improbable. To the contrary, the record shows that conclusions of a consensus workshop of independent scientists that limits below 15 ppm-hours are requisite to protect against adverse effects to vegetation. 72 Fed. Reg. 37902/2. Evidence in the Criteria Document and analysis in the Staff paper provides further support for these consensus recommendations, and indeed the Staff found nothing in more recent information to call these recommendations into question.

(b) EPA does not meet its burden of rationally justifying its choice of standards and its rejection of CASAC's recommendation merely by asserting that its decision is a "policy judgment." The Criteria Document and other evidence in the record support a finding of adverse welfare effects on vegetation at W126 levels at and below 15 ppm-hours. To justify setting the secondary standard at a higher level than that, EPA must provide a reasoned justification for disregarding or giving limited weight to the studies and scientific findings of adverse effects at and below 15, and for rejecting CASAC's recommendation.

(c) Based on the evidence of adverse welfare effects on vegetation from O_3 levels as low as 7 ppm-hours, EPA must set the secondary standard at the lowest end of the range. Section 109 (b) (2) of the Act requires EPA to set the secondary standard at a level "requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air." Because the record documents known or anticipated adverse O_3 welfare effects on vegetation at levels of 7 ppm-hours (and even below), EPA must set the standard at or below that level.

Response: Congress explicitly recognized that in making judgments about how to use uncertain evidence to set standards, the Administrator may not always agree with his scientific advisors, s*ee* CAA 307(d)(3), but EPA agrees that where the NAAQS differs in any important respect from the advice of CASAC, it must provide an explanation of the reasons for such differences. EPA fully considered CASAC's advice with regard to the range of W126 levels proposed, as is clear from the expansion of the original range identified in the second draft Staff Paper (range 13 to 21 ppm-hours) to include the lower end of the CASAC recommended range (7 ppm-hours), and explained that the level selected within the range of 7 to 21 ppm-hrs is largely a policy judgment as to the requisite level of protection needed. Specifically, the Staff Paper states "In the absence of any information regarding a threshold of O_3 exposures for vegetation, staff recognizes 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 Administrator will need 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." (EPA, 2007a)

(a) With regard to the Commenter's assertion that EPA fails to identify its basis for claiming uncertainty in determining the risks attributable to various O_3 levels, EPA refers the Commenter to the Staff Paper discussions regarding the nature and scope of uncertainties associated with the exposure (Staff Paper, pp.7-34 to 7-37), risks (Staff Paper pp. 7-43 to 7-47 for crops; 7-69 to 7-70 for trees,) and benefits (Staff Paper, pp. 7-52 to 7-53) assessments as well as similar discussions in parallel sections of the proposal notice (e.g., 72 FR 37897). The uncertainties associated with each of these assessments translate into uncertain levels of risks to vegetation for each of the different air quality scenarios evaluated. EPA, in being careful to discharge its mandate to provide the requisite level of public welfare protection, i.e. sufficient but not more than necessary, took into account to the best of its ability, all the information it had available to help inform its judgments on requisite protection. EPA's intention to do so is clearly articulated throughout the proposal notice (e.g., 72 FR 37904/5). EPA notes that the uncertainties associated with the exposure and risk assessments affect the predictions of risk to vegetation at different levels equally, so they do not, by themselves, make the risks at one level of exposure any more or less probable than another. What they do show, however, is that at lower and lower levels of O_3 air quality, remaining risks to vegetation appear to decline so that the relative risks among the different air quality scenarios can be compared to each other.

However, in addition to comparing the relative risks of effects occurring at any given level, EPA must make a further determination as to the significance of those remaining risks to the public welfare and at what level those risks could reasonably be considered adverse to the public welfare. Thus, it is not only the uncertainty associated with reductions in the presence of effects but the uncertainties associated with the degree of public welfare impact of those reduced levels of effect that must be weighed.

(b) Based on the statutory language in the section 109(b) (2) of the CAA, EPA notes that Congress clearly envisioned a role for "the judgment of the Administrator" in identifying the "requisite level of protection" for the public welfare. As stated in the proposal notice, "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." (72 FR 37903). Comments received following proposal further helped inform EPA's judgments both as to the likelihood of predicted risks occurring and potential adversity in the context of public welfare. In particular, comments regarding the significance of the uncertainties and limitations associated with the exposure and risk assessments, as well as the field based data, helped inform EPA's judgments regarding how much weight to place on these uncertainties, in reaching a decision regarding what standard is requisite to protect the public welfare.

EPA disagrees that it has not provided a reasoned justification for departing from the CASAC recommended form and range. This justification is described in the proposal notice and further refined in the preamble to the final rule to take into account comments received on the proposal notice. The Administrator notes that he is in general agreement with the CASAC Panel's views concerning the interpretation of the scientific evidence. The Administrator also notes that there is no bright line clearly directing the choice of standard for any of the effects of concern, and the choice of what standard is appropriate is clearly a public welfare policy judgment entrusted to the Administrator. This judgment must include consideration of the strengths and limitations of the evidence and the appropriate inferences to be drawn from the evidence and the exposure and risk assessments. In reviewing the basis for the CASAC Panel's recommendations for the form and range of the secondary O_3 standard, the Administrator observes that the CASAC Panel apparently placed appreciable weight on the combined science and policy views contained in a report from a 1996 consensus building workshop held by a group of independent scientists to inform its policy judgments on standard levels that would be protective against various types of O_3-related effects. While the Administrator finds the views expressed in that report to be relevant and of interest, he does not see in that report clear linkages between the underlying scientific information and the recommended standard levels. Lacking such linkages, the Administrator can find no appropriate scientific basis for deferring to the policy judgments reflected in that report. Similarly, in presenting their recommendations, the CASAC Panel did not provide clear linkages between their interpretation of the scientific evidence, the substantial uncertainties associated with that evidence, and their recommended range of levels. The Administrator more heavily weighs the implications of the uncertainties associated with the scientific evidence and with the Agency's vegetation exposure and risk assessments than the CASAC Panel apparently does, and disagrees with CASAC that the evidence and assessment results appropriately serve as a basis for concluding that a seasonal standard with a level no higher than 15 ppm-hours is required for an annual standard, or that an even lower level is required for a standard averaged over three years.

After carefully taking the above comments and considerations into account, and fully considering the scientific and policy views of the CASAC, the Administrator has decided to set the secondary standard equal to the primary standard, as discussed in the preamble.

(c) EPA disagrees that it must set the standard at the lowest level for which there are known adverse effects. As stated above in (b), the CAA does not require that secondary standards be set at a zero-risk level, but rather at a level that reduces risk sufficiently but not more than what is necessary to protect public welfare from known or anticipated adverse effects. EPA further notes that the same effect may have different degrees of adversity to the public welfare, depending on the intended use of the plant and where it is growing. In addition, in concluding that the appropriate range for EPA to consider was between 7-15 ppm-hours, the CASAC Panel, as stated in (b) above, apparently placed appreciable weight on the combined science and policy views contained in a report from a 1996 consensus building workshop held by a group of independent scientists to inform its policy judgments on standard levels that would be protective against various types of

O_3-related effects. However, though EPA found the views expressed in that report to be relevant and of interest, as stated in section II.B.2.c above, it concludes that these views fall far short of providing a scientific basis for determining at what level these effects become adverse to the public welfare.

(14) *Comment:* EPA also asks for comment on setting differing levels of protection for different types of vegetation. Because EPA has not proposed the specific levels for such a proposal, or methods for implementing it, the agency cannot pursue it further without additional notice and comment.

Response: EPA agrees that should such an alternative approach have been chosen, EPA may have needed to issue a supplemental proposal providing more detail on a proposed approach. EPA notes that since the Agency did not choose this alternative path, such a supplemental proposal is not necessary.

(15) *Comment:* UARG states in a footnote that "EPA has previously noted that 'striking anomalies arise in attempting to set welfare-based air quality standards according to some welfare effects but not others.' 49 Fed. Reg. 10,408, 10,417 (March 20, 1984). The Agency did not then resolve how such anomalies should be addressed. Nor has the Agency ever considered whether and how the beneficient [sic] effects of a pollutant (e.g., protection provided by tropospheric O_3 against UV-B radiation-related health effects such as cataracts and melanoma) should be considered in selecting a secondary NAAQS that differs from the primary one. Should the Administrator decide in this rulemaking to set a secondary standard different from the primary one, these are issues that he would be compelled to address.

Response: In the 1984 notice of proposed rulemaking cited by UARG, the question as to whether EPA could consider the costs of implementation in setting the secondary standard had not been resolved by a court. Although EPA explained that the better view of the CAA was that EPA could not consider costs in setting the standard, it noted that it was uncertain whether a court would reach the same conclusion. The "striking anomalies" noted by EPA arose from the possibility that EPA could consider the costs of implementation in setting a secondary NAAQS. As the Supreme Court has now resolved the issue of whether EPA may consider the costs of implementation in setting a secondary standard, the Agency need not resolve how such "anomalies" described in the 1984 NPRM should be addressed.

B. Misplaced Comments

Some comments received on the proposed O_3 NAAQS addressed issues that are not relevant for consideration in the review of the NAAQS. These include several comments that address implementation issues. In addition to comments related to implementation of the O_3 NAAQS that are generally addressed in section I.C of the preamble to the final rule, these comments and other "misplaced" comments are discussed more fully below.

(1) *Comment:* Many commenters noted that the current standard has not yet been fully implemented and felt that EPA should allow time for existing regulations to work before revising the ozone standard. State agencies and industry have spent years planning and implementing strategies for how best to implement reductions mandated by current regulatory programs, and a revised standard could undo much of that planning. Numerous commenters expressed concern over the substantial burden placed on states to adopt a new set of implementation plans. Further, state plans to achieve a revised standard would probably be due in 2013, which is in the middle of the current attainment effort and before the results of current activities will be known. Several commenters believed that more significant public health gains can be achieved by focusing all resources on meeting the current 8-hour standard versus starting the SIP process over again. One commenter felt that EPA should not propose to lower the current 8-hour ozone standard until such time as NOx reductions from federal programs are realized. One commenter stated that a tighter ozone standard could discourage the use and development of greenhouse-gas-friendly energy and biofuels. The commenter believes that reducing reliance on imported fossil fuels and developing technologies to reduce greenhouse gas emissions is more important than a "marginal change in ozone levels."

(2) *Comment:* Numerous commenters from state environmental organizations, industry, and private citizens stated that they did not believe that the proposed standard was attainable. Several commenters expressed concern over the impact of additional nonattainment areas on regulatory agencies charged with planning for attainment of the new standards. Several commenters expressed concern that neither state agencies nor industry will be able to devise a successful compliance strategy for the proposed standard because the control technology does not exist. Others commented that as nonattainment counties become more rural, the choice of sources that can be controlled becomes very limited and offsets would not be as available, which would hinder the economy.

(3) *Comment:* Several commenters representing industry and economic development associations felt that if the lower standard is not achievable, that it calls into question the validity of the asserted health benefits of such a standard. The Gulf Coast Lignite Coalition stated that the proposed standard was overly conservative and would not benefit the health of residents in many newly designated nonattainment areas that are relatively rural, large in size, and have a low population density. The Coalition did not believe that monitoring data was appropriate for estimating exposure.

(4) *Comment:* Several organizations believed that international and interstate impacts on ambient ozone levels will likely result in more stringent requirements placed on facilities that are not significant contributors to local air quality. Other commenters stressed that improving air quality in areas affected by transport of ozone and ozone precursors depends largely on reducing ozone levels in areas currently in nonattainment. In some cases, reducing local production of ozone would not attain a lower standard. For example, the Associated Industries of Massachusetts indicated that Massachusetts will be in nonattainment for the foreseeable future if the standard was lowered to 0.070 ppm. The issue is a result of upwind pollution transported into Massachusetts and there are no

further reductions that can be made. Similarly, the Gila River Indian Community asked that EPA acknowledge and address in the final rule the problem of identifying the Community as a nonattainment area when the largest source of ozone precursors is one over which the Community does not have jurisdiction.

(5) *Comment:* Several commenters questioned EPA's focus on mean background rather than the extremes of the background, believing that it resulted in EPA's failure to propose an attainable standard. Further, others mentioned that when EPA established the current primary standard for ozone, the EPA Administrator specifically rejected a standard of 0.070 ppm as being too close to background levels. The AAM stated that reliance on one particular global model is not a scientifically sound way to estimate the contribution of uncontrollable ozone to an extreme value standard. Some industry groups believed that the proposed welfare standard should be dropped until the issue of background ozone levels and achievable ozone limits is better understood and assessed.

Response to Comments 1-5: Questions of implementation or attainability consequences of a NAAQS are legally irrelevant in determining which standards are requisite to protect public health with an adequate margin of safety. API v. Costle, 665 F. 2d at 1185-86. In addition, section 109(d) requires EPA to reevaluate the NAAQS no later than five years after completion of the last review, and determine whether new or revised NAAQS are appropriate in light of current science, as reflected in the updated air quality criteria. Issues of implementation or attainability are not a basis to delay conducting and finishing the review and making the decisions required under section 109(d). The EPA therefore did not consider these comments in its decision-making process.

(6) *Comment:* Several organizations, including the American Association of State Highway and Transportation Officials and the Association of Metropolitan Planning Organizations, recommend that EPA initiate research to identify innovative new strategies and technologies including new federal emission control programs that will assist regional or multi-state nonattainment areas in meeting the existing standard or any new standard that is adopted. They also suggest that EPA address regional pollutant transport issues, including research, guidance on offsets, and attainment dates. The Fertilizer Institute recommended that chemical interactions that lead to ground-level ozone formation clearly need to be further studied prior to mandating further source reductions in precursor compounds.

(7) *Comment:* The Texas Department of Transportation commented that EPA should evaluate the effects and address the applicability of W126 on transportation and general conformity. If conformity does not apply, the commenter recommended that EPA provide a mechanism to exempt transportation programs from the conformity provision in areas where on-road mobile sources are not a significant contributor to the nonattainment problems, and also in areas where nonattainment is largely caused by ozone transport. The Washington Department of Ecology also commented about the implementation of the secondary standard, and stated that EPA needs to consult with and assist states that are inexperienced with dealing with a secondary standard. The National Association of Regional Councils also noted that creating two standards of evaluation

would make planning infrastructure complicated; the same primary and secondary standard would be a simpler approach.

(8) *Comment:* Several organizations expressed concerns about how the implementation of a revised standard would be coordinated with other policies and programs. The U.S. Chamber of Commerce and several other organizations commented that the proposed NAAQS standard would be in force at the "Twenty in Ten" target date set for a 35-billion-gallon renewable fuel standard and will last eight years past that date. The commenter believes that ozone precursors will rise significantly with the renewable fuels standard, which will impact compliance with the ozone NAAQS.

(9) *Comment:* Several commenters, including the Indian Nations Council of Governments and others, recommended that EPA provide for better synchronization of various programs and policies at the federal level, including the Regional Haze Program, Heavy-Duty Highway Rule, and Regional Transport from Ground-Level Ozone Program. Several commenters were concerned about the impacts that a revised standard would have on current NOx emission control activities under EPA's NOx SIP call, CAIR, and Regional Haze Program (e.g., Best Available Retrofit Technology and reasonable progress). One commenter requested that EPA provide funding commensurate with that provided to these other air quality programs.

(10) *Comment:* Several commenters stated that designation of additional nonattainment areas would significantly impact the Congestion Mitigation and Air Quality Program. One commenter predicted that the revision could possibly create unfunded mandates for many regions. Local governments do not have the resources or controls to address the changing requirements. Another commenter suggested that program funding be increased and protected from budget cuts.

(11) *Comment:* With respect to implementing a revised standard, many commenters expressed that EPA needs to issue timely implementation guidance, identifying reasonable practical measures that can aid stakeholders, and work with state and local governments to develop national strategies to address ozone. They also noted that EPA should provide the maximum time allowed under the CAA to set the standards and implement and attain such standards. The South Carolina Department of Health and Environmental Control felt that the implementation process should reward and provide flexibility to programs that attain the standards on an accelerated basis.

(12) *Comment:* Numerous commenters asked that EPA consider simultaneous strengthening of other national policies/measures that affect ozone nationwide. Several felt that more stringent controls on mobile sources would be appropriate to help achieve the standard and avoid undue burden on stationary sources. One commenter stated that because mobile source emission controls rest solely with the federal government, the reductions needed to meet attainment under the current standard are greater than the amount of emissions that the State of Texas currently regulates. One commenter suggested that EPA further tighten standards for on-road heavy-duty and off-road new diesel engines to further reduce ozone levels.

(13) *Comment:* One commenter recommended that if EPA revises the current standard that it should adopt SIP criteria to allow states to factor out background ozone and transported ozone from emission rollback requirements in their nonattainment SIPs. The commenter further suggested that EPA adopt an optional form of identifying reasonable progress toward attainment based on making reductions in the population and magnitude-weighted exposure to ozone levels remaining above the standard.

(14) *Comment:* One commenter recommended that if EPA revises the current standard that it should adopt SIP criteria to allow states to factor out background ozone and transported ozone from emission rollback requirements in their nonattainment SIPs. The commenter further suggested that EPA adopt an optional form of identifying reasonable progress toward attainment based on making reductions in the population and magnitude-weighted exposure to ozone levels remaining above the standard.

(15) *Comment:* Several commenters, including public citizens and industry organizations, suggested technologies and practices to help reduce ozone: hybrid cars, low emission engines, solar energy, low-carbon fuel, and a carbon tax. Other commenters called on EPA to make educational, modeling or policy changes to promote the integration of land-use and transportation planning and implementation. Another commenter was concerned about the potential consequences of expanded use of reformulated gasoline on refiners and consumers.

Response to Comments 6-15: As noted above, comments concerning implementation of a NAAQS are legally irrelevant in determining which standards are requisite to protect public health with an adequate margin of safety. API v. Costle, 665 F. 2d at 1185-86. The EPA therefore did not consider this comment in its decision making process. For the purpose of informing the public, a variety of information on control strategies is discussed in the RIA.

(16) *Comment:* Numerous comments were received regarding significant issues associated with the Regulatory Impact Analysis (RIA), including the scope, assumptions and methodology, and additional analyses suggested.

Response: Because the costs of implementation cannot be considered in setting or revising the NAAQS (see section I.B of the preamble to the final rule), the results of the RIA were not considered in EPA's decisions on the O_3 standards. For the same reason, comments on the RIA were not considered in the decisions. Comments on the Interim RIA were considered, as appropriate, in developing the RIA for the final rule.

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Appendix

Provisional Consideration of Recent Studies

EPA has provisionally considered the recent literature related to the health and ecological effects of ozone (O_3) to identify pertinent "new" studies that were not included in the O_3 Criteria Document. The O_3 Criteria Document included a rigorous and thorough review of the pertinent literature that included studies accepted for publication through December 2004. More recent studies accepted for publication in 2005 and 2006 were also included in the 2006 Criteria Document. These papers were identified by EPA staff, or by public comments and the Clean Air Scientific Advisory Committee (CASAC) as adding significantly to the existing body of data on critically important topics. Typically, these studies examined effects at lower O3 levels than previously reported or discussed epidemiologic methodological issues. The Criteria Document forms the scientific basis for this review of the national ambient air quality standards (NAAQS) for O_3.

A number of commenters submitted more recently published studies along with their public comments on the proposed revisions to the NAAQS. In addition, EPA has screened and surveyed the recent literature and provisionally considered those "new" studies of potentially greatest relevance to place them in the context of the findings of the 2006 O_3 Criteria Document. The intent of this effort is to determine if the new studies published since the Criteria Document materially change the conclusions of that document. Overall, EPA's provisional consideration of "new" studies, as discussed below, concludes that, taken in context, the "new" information and findings do not materially change any of the broad scientific conclusions regarding the health and ecological effects of ozone exposure made in the O_3 Criteria Document.

This effort cannot be considered a complete literature review. While the papers have been individually peer-reviewed, this provisional consideration has not been subjected to peer review or the public comment process, and it has not been reviewed by the CASAC, as is done in the development of the Criteria Document and Staff Paper.

The following sections highlight findings of recent studies from three scientific disciplines that were the major focus for the relevant recent studies submitted by public commenters: (1) epidemiology, (2) human exposure, and (3) ecology.

1. Epidemiologic Studies of Ozone-Related Health Effects

EPA has screened and surveyed the recent epidemiologic literature and identified a number of "new" studies on the health effects associated with O_3 exposure. This process has identified approximately 90 epidemiologic studies that encompass the majority of health outcomes addressed in the O_3 Criteria Document. The following sections summarize the results of EPA's provisional consideration of these epidemiologic studies for a range of health outcomes; the

overall conclusions from the O_3 Criteria Document are presented at the beginning of each section.

1.1. Human Health Effects Associated with Short-Term Exposures to Ozone

1.1.1 Mortality

The analysis of several large multi-city studies, single-city studies, and additional meta-analyses of these studies in the O_3 Criteria Document found a "positive association between increasing ambient O_3 concentrations and excess risk for non-accidental and cardio-pulmonary-related daily mortality (EPA, 2006)." The O_3 Criteria Document, therefore, concluded that the literature is "highly suggestive that O_3 directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality," but the underlying mechanisms by which such effects occur are not entirely clear (EPA, 2006).

This provisional consideration identified a number of "new" short-term O_3 exposure mortality studies; however, not all of them focused solely on O_3-related health effects.[9] These are summarized below.

One of the studies examined potential confounding in the O_3-mortality relationship using the multicity data from the National Morbidity, Mortality, and Air Pollution Study (NMMAPS). In Bell et al. (2007), confounding was investigated by analyzing the effect of PM on the association between short-term exposure to O_3 and mortality using data from 98 U.S. communities. By estimating the correlation between daily PM and O_3 concentrations, along with including PM as a covariate in various models, Bell et al. (2007) concluded that neither PM_{10} nor $PM_{2.5}$ is a likely confounder of the observed O_3 and mortality relationship.

The additional short-term exposure mortality studies identified consisted of studies that were conducted in: Italy (Forastiere et al. (2005), Parodi et al. (2005)), China (Qian et al. (2007)), South America (Cakmak et al. (2007), Australia (Simpson et al. (2005)), and one that was conducted in the U.S. (Zanobetti and Schwartz (2007)). Forastiere et al. (2005) and Qian et al. (2007) did not find an association between short-term O_3 exposure and out-of hospital coronary deaths in Rome, and non-accidental mortality in Wuhan, China, respectively. The lack of an association in these two studies may be attributed to the fact neither study focused specifically on the "O_3 season." Several studies reported associations between O_3 and mortality:

- Zanobetti and Schwartz (2007) examined mortality displacement, which postulates that deaths associated with exposure to air pollutants are occurring in frail individuals, by studying an unconstrained and smooth distributed lag of O_3 with 21 lags. The authors found that the effect size estimate was greatest when observing 21 days of ozone instead of a single day, which indicates that the effect observed is not due to mortality

[9] Two of studies identified did not focus on O_3-related mortality: Kan et al. (2007) studied the effect of short-term exposures to PM on mortality; whereas, Medina-Ramon et al. (2007) focused solely on the temperature-mortality relationship.

displacement. As a result, the authors concluded that single day lags may underestimate the true effect of short-term O_3 exposure on mortality.

- Parodi et al. (2005) and Cakmak et al. (2007) both investigated the association between O_3 and mortality in populations consisting primarily of elderly inhabitants. In Genoa, Italy, Parodi et al. found an association between both total and cardiovascular mortality throughout the whole year, but the effects were more pronounced during the warm season (May-October). Similarly, Cakmak et al. in their study in Chile concluded that the very elderly, >85, had the largest percent increase in mortality due to O_3 exposure compared to rest of the population.

- Simpson et al. (2005) assessed the effect of air pollution on mortality in four Australian cities (Brisbane, Melbourne, Perth, and Sydney). The authors found an association between short-term exposure to O_3 and respiratory-related mortality.

The remaining short-term exposure mortality studies identified (Ren et al. (2007a), Ren et al. (2007b), Rainham et al. (2005), Gonçalves et al. (2007)), analyzed the potential modification of the effect of O_3 on mortality due to various weather variables, including temperature.[10] In two separate studies, Ren et al. (2007a, 2007b) analyzed whether temperature modified the O_3-mortality effect and whether O_3 modified the temperature-mortality effect, respectively. Ren et al. (2007a) found in a study of 60 large eastern U.S. communities that temperature synergistically modifies the O_3-mortality effect, but the modification varies depending on the geographic location. On the other hand, Ren et al. (2007b) found in a study of 95 large U.S. communities, using the NMMAPS data, that O_3 modified the temperature effect on cardiovascular mortality across all regions of the U.S. Unlike the two Ren et al. studies, Rainham et al. (2005) and Gonçalves et al. (2007) analyzed the overall effect of weather on the air pollution-mortality association. Rainham et al. (2005), in a study in Toronto, Canada, did not find a systematic pattern of modification, but a modification effect seems dependent on the type of synoptic climatology category[11] analyzed. Similarly, Gonçalves et al. (2007), in a study of cardiovascular mortality in Sao Paulo, Brazil, did not find an association between weather variables and O_3 concentrations on cardiovascular mortality.

1.1.2 Respiratory Morbidity

Results from controlled human exposure studies and animal toxicological studies analyzed during the completion of the O_3 Criteria Document "provide clear evidence of causality for the associations observed between acute ($\leq$ 24 h) O_3 exposure and relatively small, but statistically

[10] In addition to the studies that focused on the potential modification of the O_3 effect, the literature search identified three studies that investigated the effect of ambient O_3 concentrations and temperature on mortality in response to the European heat wave that occurred in the summer of 2003 (Dear et al. (2003), Filleul et al. (2006), Keatinge et al. (2006)).

[11] Synoptic categories, which are also referred to as air mass categories were devised through a complex statistical approach that classifies various meteorological components (i.e., temperature, dew point, components of wind, cloud cover, and sea level pressure) into six categories.

significant declines in lung function observed in numerous recent epidemiologic studies. Declines in lung function were particularly noted in children, asthmatics, and adults who work or exercise outdoors (EPA, 2006)."

Respiratory morbidity studies analyzed in the O_3 Criteria Document provide evidence of associations between acute exposure to ambient O_3 and an increase in respiratory symptoms in asthmatic children. This consideration identified four studies that analyzed the effect of ambient O_3 concentrations on asthma symptoms (Feo Brito et al. (2007), Ho et al. (2007), Kim et al. (2007), Schildcrout et al. (2006)). The first three report an association, which is consistent with the 2006 Criteria Document; however, one - Schildcrout et al. (2006) - did not observe an association between O_3 and respiratory symptoms.

The following observations were made from those studies that reported an association:

- Ho et al. (2007) investigated the relationship between air pollution, including O_3, and weather on asthma prevalence and asthma attack rate in a study of junior high school students in Taiwan. The authors found in models examining only the effect of air pollution on each outcome, that air pollution, including O_3, is associated with asthma attack rate.

- Kim et al. (2007) examined the effect of ambient O_3 on pulmonary function and asthmatic symptoms in 17 moderate to severe asthmatics in Korea. The authors observed an association between O_3 and asthma symptoms even at O_3 concentrations below 80 ppb.

- Feo Brito et al. (2007), in a cohort of asthmatic patients in two Spanish towns, found an association between O_3 concentrations and asthma symptoms. However, the study focused on the development of asthma symptoms during the pollen season, which also influences the presentation of asthma symptoms.

Schildcrout et al. (2006) investigated the relation between ambient criteria pollutant concentrations and asthma exacerbations in a cohort of children in 8 U.S. cities. Schildcrout et al. included a population of children in which the severity of their asthma was not clearly identified. Other studies described in the 2006 Criteria Document, specifically Gent et al. (2003) and Mortimer et al. (2002), observed associations in children clearly defined as having severe asthma. Although the study population in Schildcrout et al. encompasses children that do not have severe asthma, the study results are generally consistent with the pattern of results observed in the larger body of evidence on the relation between O_3 exposure and respiratory symptoms as reviewed in the O_3 Criteria Document. The following issues were brought to the forefront by the primary author of the study, which further indicate that the lack of an association found in Schildcrout et al. is not inconsistent with the conclusions of the Criteria Document[12]:

[12] Jonathan Schildcrout provided an email to EPA discussing the results of the 2007 study in which he was the lead author on January 4, 2008.

- Shildcrout et al. included a study population of 990 children with, on average, 12 children being examined every day. The O_3 analysis included the months May through September, which resulted in the study population being less than the 990 children observed during the course of the full study. As a result, the total number of children observed by Schildcrout et al. is not comparable to other large multi-city studies that examined the effect of O_3 concentrations on asthma exacerbation, such as Mortimer et al. (2002).

- Although Schildcrout et al. did not find an association between O_3 concentrations and asthma exacerbation, Shildcrout does not imply the results are inconsistent with those previously found because a thorough evaluation of study populations, uncertainty in parameter estimates, precise scientific questions, and additional comparisons between studies that examined the effect of O_3 exposure on asthma exacerbations has not been conducted.

A few "new" studies were identified during the provisional consideration that addressed the effect of short-term O_3 exposure on lung function. In two U.S. studies, Lagorio et al. (2006) a small panel study of individuals with co-morbid conditions (Chronic Obstructive Pulmonary Disease (COPD), asthma, or Ischemic Heart Disease (IHD)), and Girardot et al. (2006) an observational study of healthy hikers, no association was found between exposure to ambient O_3 concentrations and a decrease in lung function (i.e., FEV_1 or FVC). However, in the Girardot et al. study all measured O_3 concentrations were below the current O_3 NAAQS, which is below the concentration in which lung decrements have been consistently observed in exercising individuals. The final lung function study identified, Lewis et al. (2005), did find an association between increasing ambient O_3 concentrations and reduced lung function (i.e., FEV_1) in a study of 86 school age children in Detroit, but greater than 75% of the children included in the study were classified as having persistent asthma.

1.1.2 Cardiovascular Morbidity

The "generally limited body of evidence is highly suggestive that O_3 directly and/or indirectly contributes to cardiovascular-related morbidity," including physiologic effects (i.e., release of platelet activating factor (PAF)), heart rate variability (HRV), arrhythmias, and myocardial infarctions (EPA, 2006). However, the available body of evidence reviewed during the O_3 Criteria Document does not "fully substantiate links between ambient O_3 exposure and adverse cardiovascular outcomes (EPA, 2006)."

Four "new" studies were identified (Sarnat et al. (2006), Rich et al. (2006a), Rich et al. (2006b), Metzger et al. (2007)) that investigated the effect of O_3 on arrhythmias. Each study used different cardiac episodes to identify an arrhythmia event: Sarnat et al. (2006) used supraventricular and ventricular ectopy, Rich et al. (2006a) used paroxysmal atrial fibrillation episodes (PAF), Rich et al. (2006b) used ventricular arrhythmias, and Metzger et al. (2007) used tachyarrhythmic events. Of these studies, Sarnat et al. (2006) and Rich et al. (2006a) found an association between O_3 concentrations and the onset of arrhythmias in a study of 32 non-smoking older adults, and in a study of 203 patients with implantable cardiac devices (ICDs) in Boston, respectively. However, Rich et al. (2006b) in a study of 56 patients with ICD in St.

Louis observed a weak association between O_3 concentrations and arrhythmias, while Metzger et al. (2007) did not find any association in a study 518 patients with tachyarrhythmia that had ICDs in Atlanta.

In addition, two studies were identified that examined physiologic effects, the release of PAF - a potential stroke precursor, and B-type natriuretic peptide (BNP) - a marker of congestive heart failure (HF) severity, in response to O_3 exposure (Chuang et al. (2007), Wellenius et al. (2007)). Chuang at al. (2007) found an increase in PAF along with other cellular proteins (i.e., high-sensitivity C-reactive protein (hs-CRP), 8-hydroxy-2'-deoxyguanosine (8-OHdG)) and HRV, in response to an increase in O_3 concentrations. Two additional studies, one assessing the effect of O_3 concentrations on HRV in a cohort of men in Boston (Park et al. (2007))[13], and another observing the impact of O3 on stroke in France (Henrotin et al. (2007)) along with Chuang et al. (2007) contribute to the body of evidence suggesting a potential effect of O_3 on HRV and stroke. In the Wellenius et al. (2007) study the authors did not observe any fluctuations in BNP, but this could be due to within-person variability in BNP levels.

1.1.3 Respiratory/Cardiovascular Hospital Admissions and Emergency Department Visits

Numerous population time-series studies have "observed that ambient O_3 concentrations are positively and robustly associated with respiratory-related hospitalization and asthma emergency department (ED) visits during the warm season. These observations are strongly supported by the human clinical, animal toxicologic, and epidemiologic evidence for lung function decrements, increased respiratory symptoms, airway inflammation, and airway hyperreactivity. Taken together, the overall evidence supports a causal relationship between acute ambient O_3 exposures and increased respiratory morbidity resulting in increased ED visits and hospitalizations during the warm season (EPA, 2006)."

This provisional consideration identified numerous studies that focus on respiratory hospitalization and emergency department visits, of which one (Chen et al. (2007)) did not assess the effect of ozone, and, as a result, was excluded from this consideration. The remaining studies were conducted in the U.S. (Medina-Ramón et al. (2006), Babin et al. (2007)), Canada (Cakmak et al. (2006)), Australia (Erbas et al. (2005), Barnett et al. (2005)), Hong Kong (Ko et al. (2007), Lee et al. (2006)), Korea (Kim et al. (2007)), and Taiwan (Bell et al. (2007), Yang et al. (2007), Tsai et al. (2006)). All of these studies, except Barnett et al. (2005) found an association between ambient O_3 concentrations and respiratory hospital admissions. A brief description of each of the multi-city analyses is outlined below:

- Medina-Ramón et al. (2006) evaluated the effect of ambient O_3 on respiratory hospital admissions in 36 U.S. cities. The authors found an association between O_3 exposure and chronic obstructive pulmonary disease (COPD) and pneumonia hospital admissions during the warm season.

[13] Park et al. (2007) examined the effect of O_3 on HRV, and the originating location of the ambient air. An association was only found when the air originated in the west.

- Cakmak et al. (2006) examined whether community income and education modified the effect of gaseous pollutants, including O_3, on respiratory hospitalizations in 10 large Canadian cities. Although the analysis focused on income and education variables, the study did find an association between O_3 exposure and respiratory hospital admissions in a single pollutant model, which excluded both variables.

"Highly suggestive evidence for O_3-induced cardiovascular effects [has been] provided by a few population studies of cardiovascular hospital admissions, which reported positive O_3 associations during the warm season between ambient O_3 concentrations and cardiovascular hospitalizations [and ED visits] (EPA, 2006)." The O_3 Criteria Document, therefore, concluded, that the "generally limited body of evidence is highly suggestive that O3 directly and/or indirectly contributes to cardiovascular morbidity, but more research is needed to further substantiate the links between ambient O_3 exposure and adverse cardiovascular outcomes (EPA, 2006)."

Five "new" cardiovascular hospital admission and ED visit studies were identified (Peel et al. (2007), Ballester et al. (2006), Chan et al. (2006), von Klot et al. (2005), Barnett et al. (2006)), all of which found an association between ambient O_3 concentrations and either hospital admissions or ER visits except Barnett et al. (2006). Individual observations for two of these studies are presented below:

- Peel et al. (2007) examined the effect of ambient O_3 concentrations on cardiovascular ED visits from 31 hospitals in Atlanta for individuals inflicted with co-morbid conditions (e.g., hypertension, diabetes, COPD, etc.). The authors observed an increase in cardiovascular ED visits due to effect modification in individuals co-morbid with COPD in response to ambient O_3 levels. These results add to the evidence that individuals co-morbid with various conditions, including COPD, have an increased susceptibility to ambient air pollution.

- Ballester et al. (2006) evaluated the effect of ambient O_3 concentrations on cardiovascular ED visits in 14 Spanish cities. Focusing specifically on the warm season when analyzing O3, the authors found an association between O_3 concentrations and ED visits for both cardiovascular disease (CVD) and heart disease (HD).

In addition to the respiratory and cardiovascular specific hospital admission and ED visit studies already presented, four studies examined the effect of ambient O_3 concentrations on both respiratory and cardiovascular hospital admissions and ED visits (Zanobetti and Schwartz (2006), Hinwood et al. (2006), Chan et al. (2006), Simpson et al. (2005)). Hinwood et al. (2006) and Zanobetti and Schwartz (2006) both found that O_3 was not associated with an increase in cardiovascular and respiratory, or myocardial and pneumonia hospital admissions, respectively. In contrast, Simpson et al. (2005) in a study conducted in four Australian cities found an association between O_3 concentrations and hospital admissions for both asthma and COPD in the elderly for three of the four cities; whereas, Chan et al. (2006) found an association between O_3 concentrations and cerebrovascular disease in Taiwan.

1.2 Health Effects Associated with Long-Term Exposures to Ozone[14]

1.2.1 Mortality

Few epidemiologic studies have assessed the relationship between long-term exposure to O_3 and mortality. As a result, the O_3 Criteria Document concluded that an insufficient amount of evidence exists "to suggest a causal relationship between chronic O_3 exposure and increased risk for mortality in humans (EPA, 2006)."

This provisional consideration identified a few studies that examined the association between long-term exposure to O_3 and mortality. Two of these studies focused specifically on traffic density (Lipfert et al. (2006a), Lipfert et al. (2006b)), and therefore, were not addressed in this analysis. The remaining studies (Krewski et al. (2005), Jerret et al. (2005)) did analyze the effect of long-term exposure to O_3 on mortality, but consistent with the conclusions of previous long-term O_3 exposure studies, did not find a clear association with mortality.

1.2.2 Lung Function

"The epidemiologic data, collectively, indicates that the current evidence is suggestive, but inconclusive for respiratory health effects from long-term O_3 exposure (EPA, 2006)." This provisional review identified two[15] well conducted cohort studies that assessed the effect of long-term exposure to O_3 on lung function development (Islam et al. (2007), Rojas-Martinez et al. (2007)). A description of each of the aforementioned studies and their findings are presented below:

- Islam et al. (2007) investigated the relationship between air pollution, lung function, and the subsequent development of asthma in a cohort of 9 and 10 year old children without asthma or wheeze from the Children's Health Study. The authors found that long-term O_3 exposure did not have any observable effect on forced expiratory flow (FEF), and, therefore, was not associated with lung damage or asthma development.

- Rojas-Martinez et al. (2007) evaluated the association between long-term exposure to O_3 and lung function development in Mexico City schoolchildren. In this study, the authors found that deficits in FVC and FEV_1 were associated with O_3 exposure.

[14] In addition to the studies presented in this section, three long-term O_3 exposure studies were identified that did not warrant analysis because they either focused specifically on proximity to traffic (Meng et al. (2007), McConnell et al. (2006a)), or the impact of cat and/or dog ownership along with air pollution levels on asthmatic occurrences (McConnell et al. (2006b)).

[15] Morgan et al. (2005) also studied lung function and the development of wheeze; however, the study does not take into consideration the role of air pollution on both. As a result, it was excluded from this analysis.

1.2.3 Lung Cancer

"The weight of evidence from recent animal toxicological studies and a very limited number of epidemiologic studies do not support ambient O_3 as a pulmonary carcinogen (EPA, 2006)." This provisional consideration identified two studies (Chen et al. (2006), Huen et al. (2006)), which both observed cytogenic damage (i.e., micronuclei formation and degenerated cells) in response to an increase in O_3 exposure. Although cytogenic damage could potentially lead to cancer development, neither study concluded that O_3 is a pulmonary carcinogen.

1.2.4 Prenatal and Neonatal Outcomes

A limited number of studies have examined the relationship between O_3 exposure and birth-related outcomes, including mortality, premature births, low birth weights, and birth defects. The O_3 Criteria Document concluded that "O_3 [is] not an important predictor of several birth-related outcomes including intrauterine and infant mortality, premature births, and low birth weight (EPA, 2006)."

This provisional consideration identified "new" studies that analyzed the effect of O_3 exposure on various birth outcomes, including pre-term birth (Hansen et al. (2007)); low birth weight (LBW) (Salam et al. (2005), Dugandzic et al. (2006), Hansen et al. (2007)); respiratory effects/hospitalizations (Triche et al. (2006), Dales et al. (2006)); and mortality (Tsai et al. (2006a), Tsai et al. (2006b), Hajat et al. (2007)). A synopsis of the findings for each birth outcome is presented below:

- Pre-Term Birth: Hansen et al. (2007) analyzed the association between O_3 exposure during pregnancy and pre-term birth in 28,000 singleton births in Brisbane, Australia. The authors found that an increase in O_3 exposure during the 1st trimester was associated with pre-term birth.

- Low Birth Weight (LBW): Salam et al. (2005) assessed the effect of increasing O_3 concentrations on LBW in a population of infants born in California from 1975 – 1987. The authors concluded that a positive association exists between an increase in O_3 concentrations and LBW over the entire pregnancy with the association being the strongest in the 2nd and 3rd trimesters. Dugandzic et al (2006) and Hansen et al. (2007)[16] also analyzed the effect of O_3 on LBW in Canada and Australia, respectively. In both instances, neither study observed an association between O_3 exposure and LBW during any trimester.

- Respiratory: Triche et al. (2006) examined respiratory effects of O_3 in infants of asthmatic mothers. The authors found that infants of asthmatic mothers had a greater likelihood of wheeze and difficulty breathing compared to infants whose mother did not have asthma for every interquartile-range increase in 24-hr

[16] Hansen et al. (2007) also observed the effect of O_3 on Head Circumference and Crown-Heel Length. The authors also found no association between O_3 exposure and these health effects.

average ozone.[17] In addition, Dales et al. (2006) tested the association between daily neonatal respiratory hospitalizations and ambient O_3 concentrations in 11 large Canadian cities. The authors concluded that current O_3 levels are responsible for a significant proportion of hospitalizations in neonates.

- Mortality: Tsai et al. (2006a), Tsai et al. (2006b), and Hajat et al. (2007) in studies conducted in Taiwan, Taiwan, and the United Kingdom, respectively, all found a weak association between O_3 exposure and infant mortality.

1.3 Vulnerability/Susceptibility

1.3.1 Heightened Vulnerability

Epidemiologic studies reviewed in the O_3 Criteria Document suggest that "exercising (moderate to high physical exertion) children and adolescents appear to demonstrate increased responsiveness to ambient concentrations of O_3 and may be more likely to experience O_3-induced health effects (EPA, 2006)." During this provisional consideration, a study was identified that examined the effect of exercise on O_3-related health effects (Wong et al. (2007)), however, the study focused on mortality in individuals > 30, instead of more benign health effects in children and adolescents. In this study, Wong et al. (2007) examined the relationship between habitual exercise and air pollution associated mortality in Hong Kong. The authors found an association between O_3 exposure and mortality in people ≥ 65 who never exercised. As a result, the authors concluded that habitual exercise may reduce the risk of premature death attributed to air pollution.

1.3.2. Genetic Susceptibility

Human clinical and epidemiologic studies analyzed in the O_3 Criteria Document demonstrated that "genetic polymorphisms for antioxidant enzymes and inflammatory genes (GSTM1, NQO1, and *Tnf-α*) may modulate the effect of O_3 exposure on pulmonary function and airway inflammation (EPA, 2006)." This provisional consideration identified three studies (Islam et al. (2007), Chen et al. (2007), Romieu et al. (2006)) along with two review papers (London (2007), McCunney (2005)), which found that genetic polymorphisms in antioxidant genes can lead to a decrease in lung function upon exposure to O_3.

[17] An association was not observed for wheeze for an interquartile-range increase in 8-hr maximum or 1-hr maximum.

1.4 Provisional Consideration of Epidemiologic Studies Summary

EPA emphasizes that this is a provisional consideration of the recent literature, and it is not intended to serve as a supplement to the Criteria Document. This summary of "new" studies has not undergone the detailed and extensive review process entailed in the development of a Criteria Document, and it has not been discussed by CASAC. Overall, the "new" study results support and expand upon findings in the 2006 O_3 Criteria Document. The essential conclusions of this provisional consideration are that the science supporting evaluation of the potential health impacts of O_3 on human health continues to expand and hence provides a larger knowledge base for better characterizing the relationships between O_3 and health effects. The "new" studies provide additional insights on the health effects of O_3 exposure, but the results do not materially change any of the broad scientific conclusions regarding the health effects of O_3 exposure made in the 2006 O_3 Criteria Document.

2. Human Exposure Studies

Three "new" studies were identified that addressed the relationship of personal exposures to ambient O_3 concentrations: Koutrakis et al. (2005), Sarnat et al. (2006), and McConnell et al. (2006c). The Koutrakis et al. (2005) study is a research report summarizing results presented in peer-reviewed publications that were reviewed in the O_3 Criteria Document (Sarnat et al., 2000, 2001, 2005). As described in these publications, the report found that ambient O_3 concentrations were significantly associated with personal O_3 exposures in a study in Boston during the summer, although a similar study conducted in Baltimore found that ambient O_3 concentrations showed stronger associations with personal exposures to $PM_{2.5}$ than to O_3. The poor correlations observed during the winter between ambient concentrations and personal exposure measurements may be due to the inability of personal monitors to detect low O_3 concentrations (below 5 ppb), along with differences in activity patterns and building ventilation compared to the summer. The study conducted in Steubenville, OH by Sarnat et al. (2006) observed significant associations between ambient O_3 concentrations and personal O_3 exposures in both summer and fall seasons, with higher associations observed for subjects spending time under high building ventilation conditions. A modeling study to quantitatively estimate the effect of NO_x emissions on O_3 concentrations near roadways (McConnell et al. (2006c)) predicted decreased O_3 concentrations near roadways, contributing to increased spatial variability and poor associations between residential outdoor concentrations and measured at ambient monitors; however, mean O_3 concentrations measured outside homes were within 3 ppb of fixed-site monitor values (33 ppb vs. 36 ppb).

The results of these studies are generally consistent with, and do not materially change, the broad scientific conclusions reached in the 2006 O_3 Criteria Document regarding the spatial variability of ambient O_3 concentrations and relationships between personal exposures and ambient concentrations of O_3. In one case (Koutrakis et al. (2005)), data from the study had been presented in peer-reviewed publications that were considered during preparation of the Criteria Document.

3. Ecological Studies

EPA has screened and surveyed the recent vegetation and ecological literature and identified a number of "new" studies on effects associated with O_3 exposure. The following discussion summarizes the results of EPA's provisional consideration of these studies for a range of issues related to the effects of O_3 on vegetation and ecosystems.

Two recently published companion papers (McLaughlin et al 2007a, McLaughlin et al 2007b) investigated the effects of ambient O_3 on tree growth and hydrology at forest sites in the southern Appalachian Mountains. The authors reported that the cumulative effects of ambient levels of O_3 decreased seasonal stem growth by 30-50% for most trees species in a high ozone year in comparison to a low O_3 year (McLaughlin et al 2007a). The authors also report that high ambient O_3 concentrations can disrupt whole tree water use and in turn, reduce late-season stream-flow (McLaughlin et al 2007b).

Several new studies were published since the O_3 Criteria Document from the Aspen FACE "free air" O_3 and carbon dioxide exposure experiment in a forest in Wisconsin (Kubiske et al. 2006a, Kubiske et al. 2006b, Liu et al. 2007, Percy et al. 2007, Darbah et al. 2007). Kubiske et al. (2006b) reported that elevated O_3 may change the intra- and inter-species competition. For example, O_3 treatments increased the rate of conversion from a mixed aspen-birch community to a birch dominated community. In another study at this site, Percy et al. (2007) showed that negative growth effects were seen below the current 8-hour O_3 standard level (0.084 ppm). The authors also attempted to compare different O_3 metrics to predict effects on tree growth by using trees repeatedly measured over 5 years. The authors suggested that 4th highest maximum metric performed the best, but they did not include the 3-month 12-hr W126 in their analysis.

Several new studies have been published on the incidence of foliar injury in the field due to ambient O_3 concentrations (Kohut 2007, Chappelka et al. 2007, Davis 2007a, Davis 2007b, Davis & Orendovici 2006). Kohut (2007) presented a foliar injury assessment for 244 National parks over 5 years. The author reported that risk of foliar injury was high in 65 parks, moderate in 46 parks, and low in 131 parks. Chappelka et al. (2007) reported that the average incidence of O_3-induced foliar injury of was 73% on milkweed in the Great Smokey Mountain National Park in the years 1992-1996. Three papers (Davis 2007a, Davis 2007b, Davis & Orendovici 2006) reported O_3-induced foliar injury in several plants species in National Wildlife Refuges in Maine, Michigan and New Jersey.

In a large-scale modeling analysis, Sitch et al. (2007) suggested that increasing ambient O_3 concentrations across the globe suppresses the land carbon sink because O_3 decreases plant productivity. As a consequence, more CO_2 is accumulating in the atmosphere. The authors suggest that the radiative forcing of this extra CO_2 is greater than the direct radiative forcing of O_3 as a greenhouse gas alone.

Overall, EPA's provisional consideration of these "new" studies concludes that, taken in context, the new information and findings do not materially change any of the broad scientific conclusions regarding the effects of O_3 exposure on vegetation and ecosystems made in the O_3 Criteria Document.

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