Responses to Significant Comments on the 2014 Proposed Rule on the National Ambient Air Quality Standards for Ozone

(December 17, 2014; 79 FR 75234)

Docket Number OAR-2008-0699

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List of Acronyms

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

AAM	Alliance of Automobile Manufacturers
AAP	American Academy of Pediatrics
AASHTO	American Association of State Highway and Transportation Officials
Act	Clean Air Act
AGCA	Associated General Contractors of America
AHEF	Atmospheric and Health Effects Framework
AHR	Airway hyperresponsiveness
ALA	American Lung Association
AMA	American Medical Association
ANPR	Advanced Notice of Proposed Rulemaking
APA	Administrative Procedure Act
APEX	Air Pollution Exposure model
APHEA	Air Pollution and Health: A European Approach
APHENA	Air Pollution and Health: A European and North American Approach
API	American Petroleum Institute
AQCD	Air Quality Criteria Document
AQI	Air Quality Index
AQRV	Air quality related values
AQS	Air Quality System
AspenFACE	Aspen Free Air gas Concentration Enrichment Facility
ATS	American Thoracic Society
BALF	Bronchoalveolar Lavage Fluid
BMI	Body mass index
BSA	Body surface area
CAA	Clean Air Act
CAPs	Concentrated ambient particles
CARB	California Air Resources Board
CASAC	Clean Air Scientific Advisory Committee
CASTNet	Clean Air Status and Trends Network
CBSA	Core-based statistical area
CCLP	Clara cell secretory protein
CD	Criteria Document
CDC	Centers for Disease Control
CFR	Code of Federal Regulations
CHAD	Consolidated Human Activity Database
CI	Confidence interval
CMAQ	Community Multi-scale Air Quality Model
CO	Carbon monoxide
CO_2	Carbon dioxide
COPD	Chronic obstructive pulmonary disease
C-R	Concentration-response
CRF	Concentration-response function
CRP	C-reactive protein

CV	Cardiovascular
CVD	Cardiovascular disease
DEARS	Detroit Exposure Aerosol Research Study
df	Degrees of freedom
DLW	Doubly-labeled water
Dow	Dow Chemical Company
DV	Design value
ELF	Extracellular Lining Fluid
EMPs	Enhanced Monitoring Programs
EO	Executive Order
EPA	Environmental Protection Agency
EPOC	Excess post exercise oxygen consumption
EPRI	Electric Power Research Institute
E-R	Exposure-response
ER	Emergency room
EVR	Equivalent ventilation rate
FA	Filtered air
FACE	Free-air CO_2 (and ozone) enrichment system
FEM	Federal Equivalent Method
FEV_1	Forced Expiratory Volume for 1 second
FHM	Forest Health Monitoring
FHM/FIA	Forest Health Monitoring/Forest Inventory and Analysis Program
FIA	USDA Forest Inventory and Analysis Program
FR	Federal Register
FRM	Federal Reference Method
FVC	Forced Vital Capacity
GEOS	Goddard Earth Observing System
GSTM1	Glutathione-S-transferase polymorphism M1 genotypes
GSTP1	Glutathione-S-transferase polymorphism P1 genotypes
HA	Hospital admission
HDDM	Higher Order Direct Decoupled Method
HEI	Health Effects Institute
HMOX1	Heme oxygenase-1 polymorphism
HREA	Health Risk and Exposure Assessment
HRV	Heart rate variability
ICNIRP	International Commission on Non-Ionizing Radiation Protection
IL	Interleukin
IOM	Institute of Medicine
IQR	Interquartile range
ISA	Integrated Science Assessment
IUA	Integrated uncertainty analysis
LML	Lowest measured level
LVOS	Las Vegas Ozone Study
Max	Maximum
MDA8	Maximum daily 8-hour ozone average
MDL	Method detection limit

METs	Metabolic equivalents of work
MSA	Metropolitan Statistical Area
MSS	McDonnell-Stewart-Smith
NAAQS	National ambient air quality standards
NAB	North America background
NACAA	National Association of Clean Air Agencies
NAM	National Association of Manufacturers
NAS	National Academy of Sciences
NB	Natural background
NCore sites	National Core multi-pollutant monitoring sites
NERA	National Economic Research Associates
NESCAUM	Northeast States for Coordinated Air Use Management
NFS	National Forest Service
NHAPS	National Human Activity Pattern
NHEERL-WED	National Health and Environmental Effects Research Laboratory – Western Ecology Division
NMA	National Mining Association
NMMAPS	National Morbidity, Mortality, and Air Pollution Study
NMOGA	New Mexico Oil and Gas Association
NO	Nitric oxide
NO_2	Nitrogen dioxide
NO_X	Nitrogen oxides
NOAA	National Oceanic and Atmospheric Administration
NPS	National Park Service
NQO1	NAD(P)H-quinone oxidoreductase genotype
NSR	New Source Review
NTAA	National Tribal Air Agency
O_3	Ozone
OCR	Office of Civil Rights
ODS	Ozone depleting substance
OEHHA	Office of Environmental Health Hazard Assessment
OH EPA	Ohio Environmental Protection Agency
OSIPC	8-Hour Ozone State Implementation Plan Coalition
PA	Policy Assessment
PACF	Partial autocorrelation function
PAMS	Photochemical Assessment Monitoring Stations
PEM	Personal exposure measurement
ppm	Parts per million
ppm-hrs	Parts per million-hours
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 μm in diameter
PMN	Polymorphonuclear leukocyte
pNEM	Probabilistic NAAQS exposure model
PSD	Prevention of Significant Deterioration

PSI	Pollution Standards Index
PSR	Physicians for Social Responsibility
QA	Quality assurance
QC	Quality control
RBL	Relative biomass loss
RIA	Regulatory Impact Analysis
RPO	Regional Planning Organization
RTC	Response to Comments
RYL	Relative yield loss
SCAQMD	South Coast Air Quality Management District
SIP	State Implementation Plan
SO_2	Sulfur dioxide
SOP	Standard operating procedure
SUM06	Seasonal sum of all hourly average concentrations $\geq$ 0.06 ppm
TCEQ	Texas Commission on Environmental Quality
TLNISE	Two-level normal independent sampling estimation
TNF	Tumor Necrosis Factor
TPA	Texas Pipeline Association
TSS	Total subjective symptoms score
TTP	Through-the-probe
UARG	Utility Air Regulatory Group
U.S.	United States
USB	United States background
USDA	U.S. Department of Agriculture
UV	Ultraviolet
Var(ε)	Variability
VNA	Voronoi Neighbor Averaging
VO_2	Oxygen consumption rate
VOC	Volatile organic compound
VQ	Ventilator equivalent ratio for oxygen
W126	Cumulative integrated exposure index with a sigmoidal weighting function
WESTAR	Western States Air Resources Council
WoE	Weight of evidence
wt	Wild type
WREA	Welfare Risk and Exposure Assessment

Frequently Cited Documents

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

Integrated Science Assessment (ISA)

U.S. EPA (2013). Integrated Science Assessment of Ozone and Related Photochemical Oxidants (Final). U.S. Environmental Protection Agency, Washington, DC. EPA/600/R-10/076F. http://www.epa.gov/ttn/naaqs/standards/ozone/s_o3_2008_isa.html

Health Risk and Exposure Assessment for Ozone (HREA)

U.S. EPA (2014a). Health Risk and Exposure Assessment for Ozone (Final). U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, Research Triangle Park, NC, 27711. EPA-452/R-14-004a. http://www.epa.gov/ttn/naaqs/standards/ozone/data/20140829healthrea.pdf

Welfare Risk and Exposure Assessment for Ozone (WREA)

U.S. EPA (2014b). Welfare Risk and Exposure Assessment for Ozone (Final). U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, EPA-452/R-14-005a. http://www.epa.gov/ttn/naaqs/standards/ozone/data/20141021welfarerea.pdf

Policy Assessment (PA)

U.S. EPA (2014c). Policy Assessment for the Review of the Ozone National Ambient Air Quality Standards. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards, EPA-452/R-14-006. http://www.epa.gov/ttn/naaqs/standards/ozone/data/20140829pa.pdf

Proposed Rule (Proposal)

National Ambient Air Quality Standards for Ozone: Proposed Rule. 79 FR 75234, December 17, 2014.

Preamble to the Final Rule

Preamble to the Final Rule on the Review of the National Ambient Air Quality Standards for Particulate Matter: Final Rule. To be published in the Federal Register on October 1, 2015.

Responses to Significant Comments on the 2014 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 public comments received on the 2014 O_3 NAAQS proposal notice (79 FR 75234). All significant issues raised in timely public comments have been addressed. Where comments were submitted after the close of the public comment period, the EPA has responded to the extent practicable.

More than 430,000 written comments were received from various commenters during the public comment period on the proposal, approximately 428,000 of which were part of mass mail campaigns. Among the unique submissions, comments were received from four national and regional organizations of air agencies (National Association of Clean Air Agencies (NACAA), Northeast States for Coordinated Air Use Management (NESCAUM), South Coast Air Quality Management Division (SCAQMD), and Western States Air Resources Council (WESTAR)); approximately 47 state environmental or health agencies; more than 50 federal, state, and local elected officials; 13 national environmental or public health organizations, including the American Lung Association (ALA), American Thoracic Society (ATS), the American Academy of Pediatrics (AAP), and the Sierra Club; more than 250 industry organizations, including the Alliance of Automobile Manufacturers (AAM), the American Petroleum Institute (API), the Utility Air Regulatory Group (UARG), and the U.S. Chamber of Commerce; nine tribes and tribal agencies; an organization of state highway officials (American Association of State Highway and Transportation Officials (AASHTO)); and approximately 430,000 individuals including those submissions as a part of a mass mail campaign.

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

The responses presented in this document are intended to augment the responses to comments that appear in the preamble to the final rule and 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 RTC document, to the extent such paraphrasing introduces any confusion or apparent inconsistency, the preamble itself remains the definitive statement of the rationale for the revisions to the standards adopted in the final rule. This document, together with the preamble to the O_3 NAAQS final rule and the information contained in the Integrated Science Assessment (ISA, U.S. EPA, 2013), the Risk and Exposure Assessments (REAs, U.S. EPA, 2014a, 2014b), the Policy Assessment (PA, U.S. EPA, 2014c), and related technical support documents, should be considered collectively as EPA's response to all of the significant comments submitted on EPA's 2014 O_3 NAAQS proposal.

Sections II.A and II.B address public comments on the primary and secondary O_3 standards, respectively. Comments on the interpretation of the primary and secondary NAAQS for O_3 are addressed in section II.C. Section II.D includes comments on the ambient monitoring requirements related to the O_3 NAAQS, and section II.E addresses comments on the implementation of the O_3 NAAQS. Section III includes responses to legal, administrative, procedural, or misplaced comments.

II. Responses to Comments on the Proposed O_3 Standards

This section addresses public comments on the primary (II.A) and secondary (II.B) O_3 standards, on the interpretation of the primary and secondary NAAQS for O_3 (II.C), on the ambient monitoring requirements related to the O_3 NAAQS (II.D), and on the implementation of the O_3 NAAQS (II.E).

A. Primary O_3 Standard

1. Comments on the Need for Revision of the Primary Standard

a. General and Overarching Comments on the Need for Revision

(1) *Comment:* Many commenters asserted that the current primary O_3 standard is not sufficient to protect public health, especially the health of sensitive groups, with an adequate margin of safety. These commenters agreed with the EPA's proposed decision to revise the current standard to increase public health protection. Among those calling for revisions to the current primary standard were medical groups (e.g., American Academy of Pediatrics (AAP), American Medical Association (AMA), American Lung Association (ALA), American Thoracic Society (ATS), American Heart Association, and the American College of Occupational and Environmental Medicine); national, state, and local public health and environmental organizations (e.g., the National Association of County and City Health Officials, American Public Health Association, Physicians for Social Responsibility (PSR), Sierra Club, Natural Resources Defense Council, Environmental Defense Fund, Center for Biological Diversity, and Earthjustice); the majority of state and local air pollution control authorities that submitted comments (e.g., agencies from California (California Air Resources Board (CARB) and Office of Environmental Health Hazard Assessment (OEHHA)), Connecticut, Delaware, Iowa, Illinois, Maryland, Minnesota, New Hampshire, New York, North Dakota, Oregon, Pennsylvania, Tennessee, and Wisconsin); a number of tribes and tribal organizations, including the National Tribal Air Association (NTAA); state and regional organizations (e.g., National Association of Clean Air Agencies (NACAA), Northeast States for Coordinated Air Use Management (NESCAUM), Ozone Transport Commission). While all of these commenters agreed with the EPA that the current O_3 standard needs to be revised, many supported a more protective standard than proposed by EPA, as discussed in more detail in section II.C.4 of the preamble to the final rule. Many individual commenters also expressed similar views.

(2) Commenters who expressed support for the EPA's proposed decision to revise the current primary O_3 standard generally concluded that the body of scientific evidence assessed in the ISA is much stronger and more compelling than in the last review. These commenters also generally emphasized CASAC's interpretation of the body of available evidence, which formed an important part of the basis for CASAC's reiterated recommendations to revise the O_3 standard to provide increased public health protection. In some cases, these commenters

supported their positions by citing studies published since the completion of the ISA.

Response: The EPA generally agrees with these commenters regarding the need to revise the current primary O_3 standard in order to increase public health protection though, in many cases, not with their conclusions about the degree of protection that is appropriate (II.C.4.b and II.C.4.c, below). The scientific evidence noted by these commenters was generally the same as that assessed in the ISA and the proposal,[1] and their interpretation of the evidence was often, though not always, consistent with the conclusions of the ISA and CASAC. The EPA agrees that the evidence available in this review provides a strong basis for the conclusion that the current O_3 standard is not adequately protective of public health, and that it is therefore appropriate to revise the standard (within the meaning of CAA section 109 (d)(1)) in order to provide requisite protection with an adequate margin of safety. In reaching this conclusion, the EPA places a large amount of weight on the scientific advice of CASAC (including reiterated advice that there is "clear scientific support to revise the [current] standard", and that "the current primary [NAAQS] for ozone is not protective of human health" (Frey, 2014a, p. 3; Frey, 2014b, p. ii), and on CASAC's endorsement of the assessment of the evidence in the ISA (Frey and Samet, 2012a).

(3) *Comment:* A second group of commenters, representing industry associations, businesses and some state agencies, opposed the proposed decision to revise the current primary O_3 standard, expressing the view that the current standard is adequate to protect public health, including the health of sensitive groups, and to do so with an adequate margin of safety. Industry and business groups expressing this view included the American Petroleum Institute (API), the Alliance of Automobile Manufacturers (AAM), the American Forest and Paper Association, the Dow Chemical Company, the National Association of Manufacturers (NAM), the National Mining Association (NMA), the U.S. Chamber of Commerce (in a joint comment with other industry groups), and the Utility Air Regulatory Group (UARG). State environmental agencies opposed to revising the current primary O_3 standard included agencies from Arkansas, Georgia, Louisiana, Kansas, Michigan, Mississippi, Nebraska, North Carolina, Ohio, Texas, Virginia, and West Virginia.

While commenters who opposed the proposed decision to revise the primary O_3 standard generally focused on many of the same studies assessed in the ISA, these commenters highlighted different aspects of these studies and reached 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. These commenters generally concluded that information about the health

[1] As discussed in section I.C in the preamble to the final rule, the EPA has provisionally considered studies that were highlighted by commenters and that were published after the ISA. These studies are generally consistent with the evidence assessed in the ISA, and they do not materially alter our understanding of the scientific evidence or the Agency's conclusions based on that evidence.

effects of concern has not changed significantly since 2008 and that the uncertainties in the underlying health science have not been reduced since the 2008 review. In some cases, these commenters specifically questioned the EPA's approach to assessing the scientific evidence and to reaching conclusions on the strength of that evidence in the ISA. For example, several commenters (e.g., AAM, API, TCEQ) asserted that the EPA's causal framework, discussed in detail in the ISA, is flawed and that it has not been applied consistently across health endpoints. Commenters also noted departures from other published causality frameworks (Samet and Bodurow, 2008) and from the criteria for judging causality put forward by Sir Austin Bradford Hill (Hill, 1965).

Response: The EPA disagrees with comments questioning the ISA's approach to assessing the evidence, the causal framework established in the ISA, and the consistent application of that framework across health endpoints. While the EPA acknowledges the ISA's approach departs from assessment and causality frameworks that have been developed for other purposes, such departures reflect appropriate adaptations for the NAAQS. As with other ISAs, the O_3 ISA uses a five-level hierarchy that classifies the weight of evidence for causation. In developing this hierarchy, the EPA has drawn on the work of previous evaluations, most prominently the Institute of Medicine's (IOM) *Improving the Presumptive Disability Decision-Making Process for Veterans* (Samet and Bodurow, 2008), EPA's Guidelines for Carcinogen Risk Assessment (U.S. EPA, 2005), and the U.S. Surgeon General's smoking report (CDC, 2004). The ISA's weight of evidence evaluation is based on the integration of findings from various lines of evidence from across the health and environmental effects disciplines. These separate judgments are integrated into a qualitative statement about the overall weight of the evidence and causality. The ISA's causal framework has been developed over multiple NAAQS reviews, based on extensive interactions with CASAC and based on the public input received as part of the CASAC review process. In the current review, the causality framework, and the application of that framework to causality determinations in the ISA, have been reviewed and endorsed by CASAC (Frey and Samet, 2012a).

Given these views on the assessment of the evidence in the ISA, it is relevant to note that many of the issues and concerns raised by commenters on the EPA's interpretation of the evidence, and on the EPA's conclusions regarding the extent to which uncertainties have been reduced since the 2008 review, are essentially restatements of issues raised during the development of the ISA, HREA, and/or PA. The CASAC O_3 Panel reviewed the interpretation of the evidence, and the EPA's use of information from specific studies, in drafts of these documents. In CASAC's advice to the Administrator, which incorporates its consideration of many of the issues raised by commenters, CASAC approved of the scientific content, assessments, and accuracy of the ISA, HREA, and PA, and indicated that these documents provide an appropriate basis for use in regulatory decision making for the O_3 NAAQS (Frey and Samet, 2012a, 2012b; Frey, 2014a, 2014b). Therefore, the EPA's responses to many of the comments on the evidence rely heavily on the process established in the ISA for assessing the evidence, which is

the product of extensive interactions with CASAC over a number of different reviews, and on CASAC advice received as part of this review of the O_3 NAAQS.

(4) *Comment:* Some commenters maintained that the proposed rule (and by extension the final rule) is fundamentally flawed because it does not quantify, or otherwise define, what level of protection is "requisite" to protect the public health. These commenters asserted that "EPA has not explained how far above zero-risk it believes is appropriate or how close to background is acceptable. EPA has failed to explain how the current standard is inadequate on this specific basis" (e.g., UARG). These commenters further maintained that the failure to quantify a requisite level of protection "drastically reduces the value of public participation" since "the public does not understand what is driving EPA's decision" (e.g., UARG).

Response: The EPA disagrees with these comments and notes that industry petitioners made virtually the same argument before the D.C. Circuit in *ATA III,* on remand from the Supreme Court, arguing that unless EPA identifies and quantifies a degree of acceptable risk, it is impossible to determine if a NAAQS is requisite (i.e., neither too stringent or insufficiently stringent to protect the public health). The D.C. Circuit rejected petitioners' argument, holding that "[a]lthough we recognize that the Clean Air Act and circuit precedent require EPA qualitatively to describe the standard governing its selection of particular NAAQS, we have expressly rejected the notion that the Agency must 'establish a measure of the risk to safety it considers adequate to protect public health every time it establish a [NAAQS]'" *ATA III,* 283 F. 3d at 369 (quoting *NRDC v. EPA,* 902 F.2d 962, 973 (DC Cir. 1990)). The court went on to explain that the requirement is only for EPA to engage in reasoned decision-making, "not that it definitively identify pollutant levels below which risks to public health are negligible." *ATA III,* 283 F. 3d at 370.

Thus, the Administrator is required to exercise her judgment in the face of scientific uncertainty to establish the NAAQS to provide appropriate protection against risks to public health, both known and unknown. As discussed in the preamble to the final rule, in the current review, the Administrator judges that the existing primary O_3 standard is not requisite to protect public health with an adequate margin of safety, a judgment that is consistent with CASAC's conclusion that "there is clear scientific support for the need to revise the standard" (Frey, 2014b, p. ii). Further, in section II.C.4 of the preamble to the final rule, the Administrator has provided a thorough explanation of her rationale for concluding that a standard with a level of 70 ppb is requisite to protect public health with an adequate margin of safety, explaining the various scientific uncertainties which circumscribe the range of potential alternative standards, and how she exercised her "judgment" (per section 109(b)(1) of the CAA) in selecting a standard from within that range of scientifically reasonable choices. Most obviously, evidence from both controlled human exposure studies and epidemiologic studies indicates that adverse effects can occur among both at-risk and the general population upon exposure to concentrations of O_3 less than

allowed by the current standard, or (in the case of the epidemiologic evidence) air quality distributions which would have met the current standard. This type of evidence has been held repeatedly to allow (if not compel) revision of the standard to afford requisite protection of public health. This "reasoned decision making" is what the Act requires, 283 F. 3d at 370, not the quantification advocated by these commenters.

The EPA further disagrees with the comment that a failure to quantify a requisite level of protection impaired or impeded public notice and comment opportunities. In fact, the EPA clearly gave adequate notice of the basis both for determining that the current standard does not afford requisite protection,[2] and for determining how the standard should be revised. In particular, the EPA explained in detail which evidence it considered critical, and the scientific uncertainties that could cause the Administrator to weight that evidence in various ways (79 FR 75308-75310). There were robust comments submitted by commenters from a range of viewpoints on these issues, supporting the adequacy of notice. See *National Association of Manufacturers v.* EPA, 750 F. 3d 921, 926 (D.C. Cir. 2014) (notice adequate where petitioners' comments show that they "had no problem understanding the scope of the issues up for consideration"). The public was also afforded multiple opportunities to comment to the EPA and CASAC during the development of the ISA, HREA, and PA. The EPA, thus, does not agree that lack of quantification of a risk level that is "requisite" (which is not required in any case, as stated above) has deprived commenters of adequate notice and opportunity to comment in this proceeding.

(5) *Comment:* Some commenters asserted that the proposed revision to the primary standard is based on a change in EPA's interpretation of the scientific evidence (i.e. the level of protection "requisite" to protect human health and welfare) and *not on the evidence itself*. Without new or unchanged scientific evidence supporting a lowered standard, these commenters claim that EPA has not sufficiently demonstrated or explained its change in judgment to support reversing its previous decision "that the data did not warrant adoption of a lower standard due to the 'limited' human clinical evidence and the uncertainties in epidemiological studies."

Response: While the final decision in any NAAQS review is left to the judgment of the Administrator, commenters are not correct in their conclusion that the evidence available in the current review is unchanged from the last review. In fact, in the current review the ISA considered over 1,000 new studies that have been published since the last review. As discussed in detail in the proposal and in

[2] See 79 FR 75287-91 (noting, among other things, that exposure to ambient O_3 concentrations below the level of the current standard has been associated with diminished lung function capacity, respiratory symptoms, and respiratory health effects resulting in emergency room visits or hospital admissions). See also Frey, 2014b, p. 5 (CASAC reiterated its conclusion, after multiple public comment opportunities, that as a matter of science the current standard "is not protective of public health" and provided the basis for that conclusion).

the preamble to the final rule (e.g., throughout sections II.A and II.B of the preamble to the final rule), these included critical new controlled human exposure studies conducted at O_3 exposure concentrations below 75 ppb, including new studies showing effects at 60 ppb,[3] and a number of new epidemiologic studies reporting associations with a wide range of adverse morbidity outcomes and premature mortality. In some cases, epidemiologic studies report such associations with ambient O_3 concentrations that met, or that were likely to have met, the current standard with its level of 75 ppb. Thus, we do not agree with commenters' assertion that the lack of new evidence supports retaining the existing standard.

(6) *Comment:* Some commenters highlighted the need for additional research to inform the O_3 NAAQS review. These commenters suggested additional health-based research and analysis to better understand the relationship between health effects and O_3 exposure at levels within the proposed range of 65 to 70 ppb.

Response: While additional research can reduce uncertainties, as identified in Chapter 4 of the PA (section 4.7), the EPA disagrees that further research is needed prior to revising the 2008 standard of 75 ppb down to a level of 70 ppb. The Administrator thoroughly considered the adequacy of the public health protection provided by the current 75 ppb standard. As discussed in sections II.B.2 and II.B.3 of the preamble to the final rule, a strong body of health effects evidence, along with the results of exposure and risk assessments, support the Administrator's decision that the current standard does not protect public health with an adequate margin of safety. A prime purpose of the NAAQS is to provide protection not only against effects which are clearly harmful, but also to provide a margin of safety to guard against effects which may not be fully understood. See, e.g., *Lead Industries*, 647 F. 2d at 1156 (rejecting argument that NAAQS is only to provide protection against effects shown to be clearly harmful); see also section I.A to the preamble to the final rule. It is therefore not "appropriate", within the meaning of CAA section 109(d)(1), to eschew revision of the O_3 NAAQS due to future research possibilities and objectives.

In addition to the evidence and exposure/risk information, the Administrator took note of the CASAC advice in the current review, in the 2008 decision establishing the current standard, and in the 2010 reconsideration of the 2008 decision. As discussed in section II.B of the preamble to the final rule, the current CASAC "finds that the current NAAQS for ozone is not protective of human health" and "unanimously recommends that the Administrator revise the current primary ozone standard to protect public health" (Frey, 2014b, p. 5). The prior CASAC O_3 Panel likewise recommended revision of the current standard to one with a lower level. This earlier recommendation was based entirely on the evidence and information in the record for the 2008 standard decision, which, as discussed in

[3] See *State of Mississippi, 744 F. 3d at 1350* ("[p]erhaps more studies like the Adams studies will yet reveal that the [60 ppb] level produces significant adverse decrements that simply cannot be attributed to normal variation in lung function").

sections II.A and II.B of the preamble to the final rule, has been substantially strengthened in the current review (Samet, 2011; Frey and Samet, 2012a).

In consideration of all of the above, the Administrator concludes that the current primary O_3 standard is not requisite to protect public health with an adequate margin of safety, and that it should be revised to provide increased public health protection. This decision is based on the Administrator's conclusions that the available evidence and exposure and risk information clearly call into question the adequacy of public health protection provided by the current primary standard such that it is not appropriate, within the meaning of section 109(d)(1) of the CAA, to retain the current standard.

b. Comments on the Health Effects Evidence

The remainder of this section discusses public comments and the EPA's responses on controlled human exposure studies (I.A.1.b.i); epidemiologic studies (I.A.1.b.ii); and at-risk populations (I.A.1.b.iii).

i. Evidence from Controlled Human Exposure Studies

This section discusses major comments on the evidence from controlled human exposure studies and provides the Agency's responses to those comments. To support their views on the adequacy of the current standard, commenters often highlighted specific aspects of the scientific evidence from controlled human exposure studies. Key themes discussed by these commenters included the following: (1) the adversity of effects demonstrated in controlled human exposure studies, especially studies conducted at exposure concentrations below 80 ppb; (2) representativeness of different aspects of the controlled human exposure studies for making inferences to the general population and at-risk populations; (3) results of additional analyses of the data from controlled human exposure studies; (4) evaluation of a threshold for effects; and (5) importance of demonstration of inflammation at 60 ppb. This section discusses these key comment themes as discussed in the preamble to the final rule, as well as other comments not addressed in the preamble to the final rule, and provides the EPA's responses.

Adversity

(1) *Comment:* Some commenters who disagreed with the EPA's proposed decision to revise the current primary O_3 standard disputed the Agency's characterization of the adversity of the O_3-induced health effects shown to occur in controlled human exposure studies. Some of these commenters contended that the proposal does not provide a clear definition of adversity or that there is confusion concerning what responses the Administrator considers adverse.

Response: The EPA disagrees with these comments, and notes that section II.E.4.d of the proposal describes the Administrator's proposed approach to considering the adversity of effects observed in controlled human exposure studies. Her final approach to considering the adversity of these effects, and her conclusions on adversity, are described in detail in sections II.A.1.c, II.B.2.b.i, II.C.4.b and II.C.4.c of the preamble to the final rule.

(2) *Comment:* Some commenters disagreed with the EPA's judgments regarding adversity and expressed the view that the effects observed in controlled human exposure studies following 6.6-hour exposures to O_3 concentrations below the level of the current standard (i.e., 75 ppb) are not adverse.[4] This group of commenters cited several reasons to support their views, including that: (1) the lung function decrements and respiratory symptoms observed at 72 ppb in the study by Schelegle et al. (2009) were not correlated with each other, and therefore were not adverse; and (2) group mean FEV_1 decrements observed following exposures below 75 ppb are small (e.g., < 10%, as highlighted by some commenters), transient and reversible, do not interfere with daily activities, and do not result in permanent respiratory injury or progressive respiratory dysfunction. Some of these commenters specifically contended that the evidence of adverse health effects from O_3 exposure at 0.060 and 0.072 ppm from the Schelegle et al. (2009) and Kim et al. (2011) studies is weak and does not justify lowering the standard.

Response: While the EPA agrees that not all effects reported in controlled human exposure studies following exposures below 75 ppb can reasonably be considered to be adverse, the Agency strongly disagrees with comments asserting that none of these effects are adverse. As an initial matter, the Administrator notes that, when considering the extent to which the current or a revised standard could allow adverse respiratory effects, based on information from controlled human exposure studies, she considers not only the effects themselves, but also quantitative estimates of the extent to which the current or a revised standard could allow such effects. Quantitative exposure and risk estimates provide perspective on the extent to which various standards could allow populations, including at-risk populations such as children and children with asthma, to experience the types of O_3 exposures that have been shown in controlled human exposure studies to cause respiratory effects. As discussed further in the preamble to the final rule in sections II.B.3, II.C.4.b, and II.C.4.c, to the extent at-risk populations are estimated to experience such exposures repeatedly, the Administrator becomes increasingly concerned about the potential for adverse responses in the exposed population. Thus, even though the Administrator concludes there is important uncertainty in the adversity of some of the effects observed in controlled human exposure studies based on the single exposure periods evaluated in these studies (e.g., FEV_1 decrements observed following exposures to 60 ppb O_3, as discussed in sections II.C.4.b and II.C.4.c in the preamble to the final rule), she judges that the potential for adverse effects increases as the number of exposures increases. Contrary to the commenters' views noted above, the Administrator considers the broader body of available information (i.e., including quantitative exposure and risk estimates) when

[4] Commenters who supported revising the primary O_3 standard often concluded that there is clear evidence for adverse effects following exposures to O_3 concentrations at least as low as 60 ppb, and that such adverse effects support setting the level of a revised primary O_3 standard at 60 ppb. These comments, and the EPA's responses, are discussed in the preamble to the final rule in section II.C.4.b within the context of the Administrator's decision on a revised level.

considering the extent to which the current or a revised standard could allow adverse respiratory effects (see sections II.B.3, II.C.4.b, and II.C.4.c in the preamble to the final rule).

In further considering commenters' views on the potential adversity of the respiratory effects themselves (i.e., without considering quantitative estimates), the EPA notes that although the results of controlled human exposure studies provide a high degree of confidence regarding the occurrence of health effects following exposures to O_3 concentrations from 60 to 80 ppb, there are no universally accepted criteria by which to judge the adversity of the observed effects. Therefore, as in the proposal, the Administrator relies upon recommendations from the ATS and advice from CASAC to inform her judgments on adversity.

In particular, the Administrator focuses on the ATS recommendation that "reversible loss of lung function in combination with the presence of symptoms should be considered adverse" (ATS, 2000). The study by Schelegle et al. (2009) reported a statistically significant decrease in group mean FEV_1 and a statistically significant increase in respiratory symptoms in healthy adults following 6.6-hour exposures to average O_3 concentrations of 72 ppb. In considering these effects, CASAC noted that "the combination of decrements in FEV_1 together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014b, p. 5). The EPA's response on this point is discussed in more detail in section II.B.2.b.i of the preamble to the final rule.

(3) *Comment:* As mentioned above, some commenters concluded that the effects observed in Schelegle et al. (2009) following exposure to 72 ppb O_3 were not adverse because the magnitudes of the FEV_1 decrements and the increases in respiratory symptoms were not well-correlated in individual study subjects. These commenters submitted an analysis of the individual-level data from the study by Schelegle et al. (2009). This analysis indicated that, while some study volunteers did experience both lung function decrements and increased respiratory symptoms following 6.6-hour exposures to 72 ppb O_3, others did not (e.g., Gradient). Additionally, commenters pointed out that this is consistent with previous studies that found lung function decrements were only weakly associated or not associated at all with symptoms (Frampton et al., 1997a, 1997b; McDonnell et al. 1999).

Some commenters further stated that in the proposal, moderate function changes, even those with more symptoms than have been reported at exposures below the current standard, could be viewed as a nuisance and would not interfere with daily

activities. In support of this, some cited Goodman et al. (2014a), who referred to such effects as being of low severity.[5]

Response: Although the results of controlled human exposure studies provide a high degree of confidence regarding the occurrence of health effects following exposures to O_3 concentrations from 60 to 80 ppb, there are no universally accepted criteria by which to judge the adversity of the observed effects. Therefore, as in the proposal, the Administrator relies upon recommendations from the ATS and advice from CASAC to inform her judgments on adversity. In particular, the Administrator focuses on the ATS recommendation that "reversible loss of lung function in combination with the presence of symptoms should be considered adverse" (ATS, 2000). The study by Schelegle et al. (2009) reported a statistically significant decrease in group mean FEV_1 and a statistically significant increase in respiratory symptoms in healthy adults following 6.6-hour exposures to average O_3 concentrations of 72 ppb. In considering these effects, CASAC noted that "the combination of decrements in FEV_1 together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014b, p. 5).

As mentioned above, some commenters nonetheless maintained that the effects observed in Schelegle et al. (2009) following exposure to 72 ppb O_3 (average concentration) were not adverse because the magnitudes of the FEV_1 decrements and the increases in respiratory symptoms (as measured by the total subjective symptoms score, TSS) were not correlated across individual study subjects. A commenter submitted an analysis of the individual-level data from the study by Schelegle et al. (2009) to support their position. This analysis indicated that, while the majority of study volunteers (66%) did experience both lung function decrements and increased respiratory symptoms following 6.6-hour exposures to 72 ppb O_3, some (33%) did not (e.g., Figure 3 in comments from Gradient).[6] In addition, the study subjects who experienced relatively large lung function decrements did not always also experience relatively large increases in respiratory symptoms. These commenters interpreted the lack of a statistically significant correlation between the magnitudes of decrements and symptoms as meaning that the effects reported by Schelegle et al. (2009) at 72 ppb did not meet the ATS criteria for an adverse response.

However, the ATS recommendation that the combination of lung function decrements and symptomatic responses be considered adverse is not restricted to effects of a particular magnitude nor a requirement that individual responses be

[5] The study's authors (Goodman et al. 2014a) also considered effects observed at less than 72 ppb as not being caused by O_3.They concluded, "The FEV_1 decrements observed at 60 (or 63) ppb are isolated and may be attributable to other factors unrelated to ozone." Thus, the authors ignored the well-established dose-response relationship between FEV_1 and O_3.

[6] The figure provided in comments by Gradient only clearly illustrated the responses of 30 out of 31 subjects.

correlated. Similarly, CASAC made no such qualifications in its advice on the combination of respiratory symptoms and lung function decrements (See e.g., Frey, 2014b, p. 5). Therefore, as in the proposal and consistent with both CASAC advice and ATS recommendations, the EPA continues to conclude that the finding of both statistically significant decrements in lung function and significant increases in respiratory symptoms following 6.6-hour exposures to an average O_3 concentration of 72 ppb provides a strong indication of the potential for exposed individuals to experience this combination of effects.[7]

In particular, the Administrator notes that lung function provides an objective measure of the respiratory response to O_3 exposure while respiratory symptoms are subjective, and as evaluated by Schelegle et al. (2009) were based on the subjective TSS score. If an O_3 exposure causes increases in both objectively measured lung function decrements and subjective respiratory symptoms, which indicate that people may modify their behavior in response to the exposure, then the effect is properly viewed as adverse. As noted above, the commenter's analysis shows that the majority of study volunteers exposed to 72 ppb O_3 in the study by Schelegle et al. (2009) did, in fact, experience both a decrease in lung function and an increase in respiratory symptoms.

In further considering this comment, the EPA recognizes that, consistent with commenter's analysis, some individuals may experience large decrements in lung function with minimal to no respiratory symptoms (McDonnell et al., 1999), and vice versa. As indicated above and discussed in the proposal (79 FR 75289), the Administrator acknowledges such interindividual variability in responsiveness in her interpretation of estimated exposures of concern. Specifically, she notes that not everyone who experiences an exposure of concern, including for the 70 ppb benchmark, is expected to experience an adverse response. However, she further judges that the likelihood of adverse effects increases as the number of occurrences of O_3 exposures of concern increases. In making this judgment, she notes that the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O_3 may cause other more serious effects.[8] Therefore, her decisions on the primary standard emphasize the public health importance of limiting the occurrence of repeated exposures to O_3 concentrations at or above those shown to cause adverse effects in controlled human exposure studies (see sections II.B.3, II.C.4.b, and II.C.4.c in the preamble to the final rule). The Administrator views

[7] Indeed, the finding of statistically significant decreases in lung function and increases in respiratory symptoms in the same study population likewise indicates that, on average, study volunteers did experience both effects.

[8] For example, as discussed in the proposal (79 FR 75252) and the ISA (p. 6-76), inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely. However, repeated occurrences of airway inflammation could potentially result in the induction of a chronic inflammatory state; altered pulmonary structure and function, leading to diseases such as asthma; altered lung host defense response to inhaled microorganisms; and altered lung response to other agents such as allergens or toxins (ISA, section 6.2.3).

this approach to considering the evidence from controlled human exposure studies as being consistent with commenter's analysis indicating that, while the majority did, not all study volunteers exposed to 72 ppb O_3 experienced the adverse combination of lung function decrements and respiratory symptoms following the single exposure period evaluated by Schelegle et al. (2009).

(4) *Comment:* Commenters urging revision of the standard to a level of 60 ppb noted the EPA's reliance on certain aspects of the ATS (2000) definition, but noted that it was incomplete. In particular, these commenters quoted ATS (2000) at 671, stating that "[a]t the population level, any detectable increment in symptom frequency should be considered as constituting an adverse health effect." They further quoted, "[t]he present committee shared the view of the previous group: detectable effects of air pollution on clinical measures should be considered adverse" (ATS, 2000). The commenter noted that epidemiological literature has linked O_3 at levels of 60 ppb with numerous clinical measures, and that statistically significant group mean decrease in FEV_1 observed in controlled human exposure studies as low as 60 ppb is an adverse effect at the population level. The commenter pointed to the EPA review of the SO_2 NAAQS, and the finding there that "diminished reserve lung function in a population that is attributable to air pollution is considered an adverse effect under ATS guidance" (quoting 75 FR 35226/2). In this regard, the commenter noted a substantial percentage – at least 10% – of the healthy, study subjects experienced a 10% decrement at exposure to 60 ppb, that at-risk populations would be expected to experience at least the same degree of decrements, that these results are consistent with those predicted by the McDonnell-Stewart-Smith (MSS) model, and that these exposures consequently constitute a population level adverse effect. One commenter noted that EPA indicated that not every lung function decrement at the 10 or 15% level will be adverse, but maintained that certainly some of those exposures will result in adverse effects, and that adverse effects can result from single occurrence decrements of this magnitude.

Commenters further noted that the PA itself states that "60-ppb is a short-term exposure concentration that may be reasonably concluded to elicit adverse effects in at-risk groups" (PA, p. 4-12). Commenters asserted that the EPA has not explained its deviation from this conclusion (citing caselaw from the D.C. Circuit that deviations from PA conclusions require rational explanation). Some commenters further asserted that exposures to 60 ppb O_3 cause adverse effects in at-risk populations and in some healthy individuals, so it is irrelevant that not all exposed will experience adverse effects.

Response: The Administrator disagrees that her consideration of the potential for O_3 exposures to result in adverse responses is inconsistent with ATS recommendations, CASAC advice, or with the EPA's past practices (i.e., in the review of the primary SO_2 NAAQS, as highlighted by commenters).[9] As

[9] Her consideration of ATS recommendations and CASAC advice is discussed extensively in sections II.B.2, II.B.3, II.C.4.b, and II.C.4.c in the preamble to the final rule.

discussed in the final rule, an important part of the Administrator's consideration of exposure estimates is the extent to which she judges that adverse effects could occur following specific O_3 exposures.[10] While controlled human exposure studies provide a high degree of confidence regarding the extent to which specific health effects occur following exposures to O_3 concentrations from 60 to 80 ppb and above, the Administrator notes that there are no universally accepted criteria by which to judge the adversity of the observed effects. Therefore, in making judgments about the extent to which the effects observed in controlled human exposure studies have the potential to be adverse, the Administrator considers the recommendations of ATS and advice from CASAC (see sections II.B.2, II.B.3, II.C.4.b, and II.C.4.c in the preamble to the final rule).

As an initial matter, with regard to the effects shown in controlled human exposure studies following O_3 exposures, the Administrator notes the following:

- The largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures to 80 ppb O3 or higher, with most exposure studies conducted at these higher concentrations. Specifically, 6.6-hour exposures of healthy young adults to 80 ppb O3, while engaged in quasi-continuous, moderate exertion, can decrease lung function, increase airway inflammation, increase respiratory symptoms, result in airway hyperresponsiveness, and decrease lung host defenses.

- Exposures of healthy young adults for 6.6 hours to O3 concentrations as low as 72 ppb, while engaged in quasi-continuous, moderate exertion, have been shown to both decrease lung function and result in respiratory symptoms, including in the same individuals.

- Exposures of healthy young adults for 6.6 hours to O3 concentrations as low as 60 ppb, while engaged in quasi-continuous, moderate exertion, have been shown to decrease lung function and to increase airway inflammation.

To inform her judgments on the potential adversity to public health of these effects reported in controlled human exposure studies, as in the proposal, the Administrator focuses on the ATS recommendation that "reversible loss of lung function in combination with the presence of symptoms should be considered adverse" (ATS, 2000). She notes that this combination of effects has been shown to occur following 6.6-hour exposures to O_3 concentrations at or above 72 ppb. In

[10] These conclusions focus on effects reported in controlled human exposure studies, which the Administrator is emphasizing in her decisions in this review of the primary O_3 standard. Regarding the epidemiologic studies cited by commenters, as discussed elsewhere in this response to comments document and in the preamble to the final rule (section II.C.4.b.ii), it is not clear that the study area O_3 concentrations cited by commenters are relevant for direct comparison to the level of the primary O_3 standard.

considering these effects, CASAC observed that "the combination of decrements in FEV_1 together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014b, p. 5).

Regarding the potential for adverse effects following exposures to lower concentrations, the Administrator notes the CASAC judgment that the adverse combination of lung function decrements and respiratory symptoms "almost certainly occur in some people" following exposures to O_3 concentrations below 72 ppb (Frey, 2014b, p. 6). In particular, when commenting on the extent to which the study by Schelegle et al. (2009) suggests the potential for adverse effects following O_3 exposures below 72 ppb, CASAC judged that:

> [I]f subjects had been exposed to ozone using the 8-hour averaging period used in the standard [rather than the 6.6-hour exposures evaluated in the study], adverse effects could have occurred at lower concentration. Further, in our judgment, the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma (Frey, 2014b, p. 5).

Though CASAC did not provide advice as to how far below 72 ppb adverse effects would likely occur, the Administrator agrees that such effects could occur following exposures at least somewhat below 72 ppb.

The Administrator notes that while adverse effects could occur following exposures at least somewhat below 72 ppb, the combination of statistically significant increases in respiratory symptoms and decrements in lung function has not been reported following 6.6-hour exposures to average O_3 concentrations of 60 ppb or 63 ppb, though studies have evaluated the potential for such effects (Adams, 2006; Schelegle et al., 2009; Kim et al., 2011). In the absence of this combination, the Administrator looks to additional ATS recommendations and CASAC advice in order to inform her judgments regarding the potential adversity of the effects that have been observed following O_3 exposures as low as 60 ppb.

With regard to ATS, she first notes the recommendations that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" and that "[f]ew…biomarkers have been validated sufficiently that their responses can be used with confidence to define the point at which a response should be equated to an adverse effect warranting preventive measures" (ATS, 2000).[11] Based on these recommendations, compared to effects following exposures at or

[11] With regard to this latter recommendation, as discussed in section II.A.1.c in the preamble to the final rule, the ATS concluded that elevations of biomarkers such as cell numbers and types, cytokines, and reactive oxygen species may signal risk for ongoing injury and more serious effects or may simply represent transient responses, illustrating the lack of clear boundaries that separate adverse from nonadverse events.

above 72 ppb, the Administrator has less confidence in the adversity of the respiratory effects that have been observed following exposures to 60 or 63 ppb.

She further notes that some commenters who advocated for a level of 60 ppb also focused on ATS recommendations regarding population-level risks. These commenters specifically stated that lung function decrements "may be adverse in terms of 'population risk,' where exposure to air pollution increases the risk to the population even though it might not harm lung function to a degree that is, on its own, 'clinically important' to an individual" (e.g., ALA et al.). These commenters asserted that the EPA has not appropriately considered the potential for such population-level risk.[12] Contrary to the views expressed by these commenters, the Administrator carefully considers the potential for population risk, particularly within the context of the ATS recommendation that "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). Given that exposures to 60 ppb O_3 have been shown in controlled human exposure studies to cause transient and reversible decreases in group mean lung function, the Administrator notes the potential for such exposures to result in similarly transient and reversible shifts in the risk profile of an exposed population. However, in contrast to commenters who advocated for a level of 60 ppb, the Administrator also notes that the available evidence does not provide information on the extent to which a short-term, transient decrease in lung function in a population, as opposed to a longer-term or permanent decrease, could affect the risk of other, more serious respiratory effects (i.e., change the risk profile of the population). This uncertainty, together with the additional ATS recommendations noted above, indicates to the Administrator that her judgment that there is uncertainty in the adversity of the effects shown to occur at 60 ppb is consistent with ATS recommendations.[13]

With regard to CASAC advice, the Administrator notes that, while CASAC clearly advised the EPA to consider the health effects shown to occur following exposures to 60 ppb O_3, its advice regarding the adversity of those effects is less clear. In particular, she notes that CASAC was conditional about whether the lung function decrements observed in some people at 60 ppb (i.e., FEV_1 decrements $\geq$ 10%) are adverse. Specifically, CASAC stated that these decrements "*could* be adverse in individuals with lung disease" (Frey, 2014b, p. 7, emphasis added) and

[12] For example, the commenters who highlighted what they interpreted as inconsistencies with the most recent review of the primary SO_2 NAAQS, noted above, focused on the EPA's consideration of ATS recommendations regarding population risks. However, consideration of population-level risk in SO_2 was focused on FEV_1 decrements $\geq$ 15%, rather than the 10% decrements highlighted by these commenters, and shown to occur in some people following 60 ppb O_3 exposures. It is reasonable to conclude that, as the magnitude of FEV_1 decrements decreases, there is increasing uncertainty in the potential for such effects to be adverse.

[13] ATS provided additional recommendations to help inform judgments regarding the adversity of air pollution-related effects (e.g., related to "quality of life"), though it is not clear whether, or how, such recommendations should be applied to the respiratory effects observed in controlled human exposure studies following 6.6-hour O_3 exposures (ATS, 2000, p. 672).

that they provide a "*surrogate* for adverse health outcomes for people with asthma and lung disease" (Frey, 2014b, p. 3, emphasis added). Further, CASAC did not recommend considering standard levels low enough to eliminate O_3-induced FEV_1 decrements ≥ 10% (Frey, 2014b). With regard to the full range of effects shown to occur at 60 ppb (i.e., FEV_1 decrements, airway inflammation), CASAC stated that exposures of concern for the 60 ppb benchmark are "*relevant for consideration*" with respect to people with asthma (Frey, 2014b, p. 6, emphasis added). In addition, "[t]he CASAC concurs with EPA staff regarding the finding based on scientific evidence that a level of 60 ppb corresponds to the lowest exposure concentration demonstrated to result in lung function decrements large enough to be judged an abnormal response by ATS and that *could be adverse* in individuals with lung disease" (Frey, 2014b, p. 7, emphasis added).[14] The Administrator contrasts these statements with CASAC's clear advice that "the combination of decrements in FEV_1 together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014b, p. 5).

Based on her consideration of all of the above recommendations and advice noted above, the Administrator judges that, compared to exposure concentrations at and above 72 ppb, there is greater uncertainty with regard to the adversity of effects shown to occur following O_3 exposures as low as 60 ppb. However, based on the effects that have been shown to occur at 60 ppb (i.e., lung function decrements, airway inflammation), and CASAC advice indicating the importance of considering these effects (though its advice regarding the adversity of effects at 60 ppb is less clear), she concludes that it is appropriate to give some consideration to the extent to which a revised standard could allow such effects. Her consideration of such effects is discussed in the preamble to the final rule (sections II.C.4.b and II.C.4.c).

(5) *Comment:* Commenters primarily representing industry groups, business associations, and some states asserted that the effects reported in the controlled human exposure studies at concentrations below the current standard level are not adverse and are not important from a public health perspective. Many asserted that the lung function decrements observed in controlled human exposure studies are transient, reversible, and not likely to interfere with normal activities. These commenters also objected to using a 10% decrement in FEV_1 to identify adverse responses, contending that such a cutoff for adversity is arbitrary and that a 10% decrement is not likely to be adverse. Commenters pointed out that the ATS

[14] CASAC also noted "findings of adverse effects, including clinically significant lung function decrements and airway inflammation, after exposures to 60 ppb ozone in healthy adults with moderate exertion" (Frey, 2014b, p. 7). However, it is not clear from its letter how CASAC intended the EPA to interpret this aspect of its advice, within the context of its broader advice on the potential adversity of effects at 60 ppb. In particular, this advice was offered in support of a level of 60 ppb as the lower bound of the range of standard levels that could be supported by the scientific information, a range that included an upper bound of 70 ppb (Frey, 2014b, p. ii).

guidelines do not specify 10% lung function decrement as a cutpoint for adversity and cautioned against considering FEV_1 decrements themselves as adverse. Commenters contended that the clinical significance of a 10% decrement is dependent on the study subjects and that, in healthy individuals, a 10% decrement, and possibly even a 15% decrement, would likely not be adverse.

Response: While the Administrator has concluded that there is some uncertainty in using lung function decrements alone as a basis for characterizing O_3 responses as adverse, she does not agree with the industry comments that lung function decrements at or above 10% raise no issues of potential public health concern. Her consideration of such decrements is discussed in this section above, and in the preamble to the final rule (sections II.B.2, II.B.3, II.C.4.b, and II.C.4.c of the final rule).

(6) *Comment:* Some commenters who supported revision of the primary standard also asserted that the proposed definitions of adversity are inconsistent with approaches taken by EPA in past O_3 reviews, as well as CASAC advice in past reviews. In some cases, these commenters cited CASAC advice from previous reviews regarding the potential adversity of FEV_1 decrements $\geq$ 10% in people with lung diseases such as asthma (e.g., Henderson, 2006; Samet, 2011). For example, one commenter stated that in the 2008 O_3 NAAQS, EPA stated that a lung function decrement equal to or greater than 10% FEV_1 "represent[s] a level that should be considered adverse for asthmatic individuals" (quoting 73 FR at 16455). The commenter also quotes the 2011 CASAC O_3 Review panel as stating that "clinically relevant effects are decrements greater than 10%, a decrease in lung function considered clinically relevant by the American Thoracic Society" (Samet, 2011, p. 2). The commenter further quotes the CASAC panel as stating that "[A] 10% decrement in FEV_1 can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e. decreased baseline FEV_1) such that a 10% decrement could lead to moderate to severe respiratory symptoms" (Samet, 2011, p. 7). The commenter stated that EPA has abandoned its past position regarding the adversity of this magnitude of lung function decrement without providing an explanation for its change of position.

Other commenters opposing revision also noted examples of where they interpreted the Administrator's judgments as being inconsistent with past practice. For example, one commenter asserted that in previous reviews, isolated FEV_1 decrements even with mild symptoms were not considered a concern. This commenter also suggested that effects reported below the current standard are arguably somewhere between the mild and moderate categories for functional changes used by EPA in previous reviews.

Response: As an initial matter, we note that in this review the Agency's consideration of the potential adversity of various O_3-related responses is based on an updated body of scientific evidence, updated exposure and risk estimates,

and updated CASAC advice. The Administrator fully considered all of this updated information and staff conclusions, as well as ATS recommendations and CASAC advice, in making judgments about the potential adversity of O_3-related effects.

As in past reviews, a consideration of FEV_1 decrements $\geq$ 10% in the current review is based in part on ATS criteria, as well as on CASAC advice. Based on ATS guidelines for assessing bronchoconstriction, the ISA states that "[a] 10% FEV_1 decrement is…generally accepted as an abnormal response" (ISA, p. 6-19). In this context, "abnormal" indicates that the decrement is outside the normal range of day-to-day variability, and is not meant to indicate that such a response is invariably "adverse." In fact, in its recommendations on adversity, the ATS did not speak specifically to FEV_1 decrements of any particular magnitude, and stated that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" (ATS, 2000, p. 670).

Consistent with this characterization, in the current and past reviews CASAC and the EPA have noted the potential for O_3-induced FEV_1 decrements $\geq$ 10% to be adverse in people with lung disease, but acknowledged that there is some uncertainty in this judgment. For example, in the 2008 final rule the EPA noted that "in the context of standard setting, CASAC indicated 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*" (73 FR 16463, March 27, 2008; internal citations omitted, emphasis added). In the 2010 proposal, which proposed to reconsider the 2008 decision on the primary O_3 standard, a different Administrator again noted this same point (75 FR 2993, January 19, 2010). Beyond this characterization of the potential adversity of O_3-induced FEV_1 decrements $\geq$ 10%, and the characterization provided by commenters as noted above, in the past the EPA has also stated that such decrements "can be clinically significant" (73 FR 16449, March 27, 2008), that they "may be clinically significant" (75 FR 2950, January 19, 2010), and that they are "notable" (75 FR 2950, January 19, 2010). In addition, in the 2008 review CASAC stated that 10% FEV_1 decrements "can be clinically significant" (Henderson, 2006, p.4) and that "in asthmatic children, a 10% change is indicative of adverse effects" (Henderson, 2006, p. 12). Consistent with these characterizations, in the current review the Administrator notes the CASAC advice that "an FEV_1 decrement of $\geq$ 10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease" (e.g., see sections II.B.2.b.i, II.B.3, II.C.4.b, and II.C.4.c of the preamble to the final rule). In the current review, the Administrator further agrees with the judgment made in past reviews (e.g., see 75 FR 2973, January 19, 2010) that a more general consensus view of the potential adversity of such decrements emerges as the frequency of occurrences increases (sections II.B.3 and II.C.4.c of the preamble to the final rule). In addition, consistent with past and current CASAC recommendations, in both the 2008 final decision and in the Administrator's final decision in the current review, the level of the primary O_3

standard was set to reduce, but not eliminate, the estimated occurrence of O_3-induced FEV_1 decrements ≥ 10%.

Beyond consideration of O_3-induced FEV_1 decrements by themselves, the updated evidence available in this review provides additional insight into the potential for O_3 exposures to result in the combination of lung function decrements and respiratory symptoms. As discussed further in this section above, one study published since the last review found this combination of effects following 6.6-hour exposures to average O_3 concentrations as low as 72 ppb (Schelegle et al., 2009). CASAC agreed with the PA conclusion that this combination of effects meets the ATS criteria for an adverse response. The combination of statistically significant increases in respiratory symptoms and significant decreases in FEV_1 has not been shown to occur following 6.6-hour exposures to average O_3 concentrations of 60 or 63 ppb, though several studies have evaluated the potential for such effects (Adams, 2006; Schelegle et al., 2009; Kim et al., 2011).

Given the above, and as discussed in sections II.B.2.b.i, II.B.3, II.C.4.b, and II.C.4.c of the preamble to the final rule, the Administrator judges that her conclusions regarding the adversity of O_3-induced effects in the current review are consistent with the updated scientific evidence, exposure/risk information, and CASAC advice. In addition, as described above, she does not agree with commenters that her judgments regarding the adversity of O_3-induced effects, including FEV_1 decrements ≥ 10%, are inconsistent with those in the 2008 final decision or in the 2010 proposed reconsideration of the decision made in the 2008, and notes that her judgments and conclusions in the current review are based on an expanded body of information and takes into account the most recent CASAC advice.

Representativeness

A number of commenters raised issues concerning the representativeness of controlled human exposure studies considered by the Administrator in this review, based on different aspects of these studies. These commenters asserted that since the controlled human exposure studies were not representative of real-world exposures, they should not be relied upon as a basis for finding that the current standard is not adequate to protect public health. Some issues highlighted by commenters include: small size of the study populations; unrealistic activity levels used in the studies; unrealistic exposure scenarios (i.e., triangular exposure protocol) used in some studies, including Schelegle et al. (2009); and differences in study design that limit comparability across studies.

(7) *Comment:* Some commenters asserted that the controlled human exposure studies were not designed to have individuals represent portions of any larger group, and that group means do not mask individual responses (e.g., citing Goodman et al., 2015b for support). Commenters stated that the results from new controlled human exposure studies that report lung function effects below 75 ppb should not be extrapolated to the U.S. population. Moreover, these commenters noted that the impacts on a small number of people do not implicate the health of an entire

subpopulation, particularly when the FEV_1 decrements are small, temporary, and reversible. These commenters also noted that the Administrator failed to provide an explanation or justification for why the individuals in these studies can be viewed as representatives of a subpopulation. Further, they asserted that EPA's use of results from individuals, rather than the group mean responses, contradicts the intent of CAA section 109 to protect groups of people, not just the most sensitive individuals in any group (79 FR 75237).

Response: Consistent with CASAC advice (Frey, 2014b, p. 5), the EPA concludes that the body of controlled human exposure studies are sufficiently representative to be relied upon as a basis for finding that the current standard is not adequate to protect public health. These studies generally recruit healthy young adult volunteers, and often expose them to O_3 concentrations found in the ambient air under real-world exposure conditions. As described in more detail in section II.A.1.b of the preamble to the final rule, the evidence from controlled human exposure studies to date makes it clear that there is considerable variability in responses across individuals, even in young healthy adult volunteers, and that group mean responses are not representative of more responsive individuals. It is consequently important to look beyond group mean responses to the responses of these individuals to evaluate the potential impact on more responsive members of the population. Contrary to commenters' assertions and the conclusions of Goodman et al. (2015b),[15] relying on group mean changes to evaluate lung function responses to O_3 exposures would mask the responses of the most sensitive groups, particularly where, as here, the group mean reflects responses solely among the healthy young adults who were the study participants. Thus, the studies of exposures below 80 ppb O_3 show that 10% of young healthy adults experienced FEV_1 decrements > 10% following exposures to 60 ppb O_3, and 19% experienced such decrements following exposures to 72 ppb (under the controlled test conditions involving moderate exertion for 6.6 hours). These percentages would likely have been higher had people with asthma or other at-risk populations been exposed (ISA, pp. 6-17 and 6-18; Frey, 2014a, p. 14; Frey 2014b, p. 7[16]). Therefore, the EPA disagrees with the conclusion of some commenters that average responses adequately reflect those of sensitive individuals (either members of at-risk groups, or more responsive healthy members of the population).

Legislative history dating from the inception of the NAAQS supports the EPA's conclusion that it legitimately view**s** the individuals in these studies as representatives of the larger subpopulation of at-risk or sensitive groups, and not just as isolated, unrepresentative individuals. As stated in the Senate Report to the

[15] Responders were identified at concentrations where group mean was significant. In their analyses, the authors incorrectly assumed that all responses were normally distributed.

[16] See also *National Environmental Development Associations Clean Action Project v. EPA*, 686 F. 3d 803, 811 (D.C. Cir.2012) (EPA drew legitimate inference that serious asthmatics would experience more serious health effects than clinical test subjects who did not have this degree of lung function impairment).

1970 legislation establishing the NAAQS statutory provisions, "the Committee emphasizes that included among these persons whose health should be protected by the ambient standard are particularly sensitive citizens such as bronchial asthmatics and emphysematics who in the normal course of daily activity are exposed to the ambient environment. In establishing an ambient standard necessary to protect the health of these persons, reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group….For purposes of this description, a statistically related sample is the number of persons necessary to test in order to detect a deviation in the health of any person within such sensitive group which is attributable to the condition of the ambient air." S. Rep. No. 11-1196, 91st Cong. 2d sess. at 10. As just noted above, 10% of healthy young adults in these studies experienced > 10% FEV_1 decrements following exposure to 60 ppb O_3, and the proportion of individuals experiencing such decrements increases with increasing O_3 exposure concentrations. This substantial percentage certainly can be viewed as "a representative sample of persons" and as a sufficient number to "detect a deviation in the health of any person within such sensitive group," especially given that it reflects the percentage of *healthy adults* who experienced decrements > 10%.

The percentage of individuals experiencing effects in the controlled human subject clinical tests are also consistent with estimates from the McDonnell-Stewart-Smith (MSS) model, which makes reliable quantitative predictions of the lung function response to O_3 exposures, and reasonably predicts the magnitude of individual lung function responses following such exposures. As described in section II.A.2.c in the preamble to the final rule, and documented in the HREA, when the MSS model was used to quantify the risk of O_3-induced FEV_1 decrements in 15 urban study areas, the current standard was estimated to allow about 8 to 12% of children to experience two or more O_3-induced FEV_1 decrements ≥ 10%, and about 2 to 3% to experience two or more decrements ≥ 15% (see Table 2 in the preamble to the final rule). These percentages correspond to hundreds of thousands of children in the urban study areas, and tens of thousands of asthmatic children. While the Administrator judges that there is some uncertainty with regard to the adversity of these O_3-induced lung function decrements (see sections II.C.4.b.i and II.C.4.c in the preamble to the final rule), such risk estimates clearly indicate that they are a matter of public health importance on a broad scale, not isolated effects on idiosyncratically responding individuals. See also response under "threshold" in this section.

(8) *Comment:* Other commenters considered the ventilation rates used in controlled human exposure studies to be unreasonably high and at the extreme of prolonged daily activity. Some of these commenters noted that these scenarios are unrealistic for sensitive populations, such as asthmatics and people with COPD, whose conditions would likely prevent them from performing the intensity of exercise, and therefore experiencing the ventilation rates, required to produce decrements in lung function observed in experimental settings.

Response: The EPA disagrees with these commenters. The activity levels used in controlled human exposure studies were summarized in Table 6-1 of the ISA. The exercise level in the 6.6-hour exposure studies by Adams (2006), Schelegle et al. (2009), and Kim et al. (2011) of young healthy adults was moderate and ventilation rates are typically targeted for 20 L/min-m^2 BSA.[17] Following the exposures to 60 ppb at this activity level, 10% of the individuals had greater than a 10% decrement in FEV_1 (ISA, p. 6-18). Similar 6.6-hour exposure studies of individuals with asthma are not available to assess either the effects of O_3 on their lung function or their ability to perform the required level of moderate exercise.

However, referring to Tables 6-9 and 6-10 of the HREA, between 42% and 45% of FEV_1 decrements ≥ 10% were estimated to occur at exercise levels of <13 L/min-m^2 BSA. This corresponds to light exercise, and this level of exercise has been used in a 7.6-hour study of healthy people and people with asthma exposed to 160 ppb O_3 (Horstman et al., 1995). In that study, people with asthma exercised with an average minute ventilation of 14.2 L/min-m^2 BSA. Adjusted for filtered air responses, an average 19% FEV_1 decrement was seen in the people with asthma versus an average 10% FEV_1 decrement in the healthy people. In addition, the EPA noted in the HREA that the data underlying the exposure assessment indicate that "activity data for asthmatics [is] generally similar to [that for] non-asthmatics" (HREA, p. 5-75, Tables 5G-2 and 5G-3). Thus, contrary to the commenters' assertion, based on both the HREA and the Horstman et al. (1995) study, people with respiratory disease such as asthma can exercise for a prolonged period under conditions where they would experience >10% FEV_1 decrements in response to O_3 exposure.

(9) *Comment:* Additionally, a number of commenters asserted that the exposure scenarios in Schelegle et al. (2009), which are based on a so-called triangular study protocol, where O_3 concentrations ramp up and down as the study is conducted, are not directly generalizable to most healthy or sensitive populations because of large changes in the O_3 concentrations from one hour to the next. Commenters stated that although large fluctuations in O_3 are possible in certain locations due to meteorological conditions (e.g., in valleys on very hot, summer days), they believe that, in general, concentrations of O_3 do not fluctuate by more than 20-30 ppb from one hour to the next. Thus, commenters suggested the Schelegle et al. (2009) study design could happen in a "worst-case" exposure scenario, but that the exposure protocol was not reflective of conditions in most cities and thus not informative with regard to the adequacy of the current standard.

Response: The EPA disagrees with the comment that these triangular exposure scenarios are not generalizable because of hour-to-hour fluctuations. Adams

[17] Exercise consisted of alternating periods walking on a treadmill at a pace of 17-18 minutes per mile inclined to a grade of 4-5% or cycling at a load of about 72 watts. Typical heart rates during the exercise periods were between 115-130 beats per minute. This activity level is considered moderate (ISA, p. 6-18, Table 6-1).

(2003, 2006) showed that FEV_1 responses following 6.6 hours of exposure to 60 and 80 ppb average O_3 exposures do not differ between triangular (i.e. ramping concentration up and down) and square-wave (i.e. constant concentration). Schelegle et al. (2009) used the 80 ppb triangular protocol and a slightly modified 60 ppb triangular protocol (concentrations during the third and fourth hours were reversed) from Adams (2006). Therefore, in considering pre- to post-exposure changes in lung function, concerns about the hour-by-hour changes in O_3 concentrations at 60 and 80 ppb in the Schelegle et al. (2009) study are unfounded. EPA additionally notes that the magnitude of FEV_1 responses at the target exposures of 60 and 80 ppb are similar to those observed in other studies as illustrated in Figure 6-1B of the ISA. This consistency in responses between the Schelegle et al. (2009) study and prior studies at 60 and 80 ppb provides assurance that the effects observed at 72 ppb are also reliable and generalizable. This is further supported by Figure 6 (panel 1A) of McDonnell et al. (2012) which shows that the MSS model well predicts the mean FEV_1 responses of the Schelegle et al. (2009) study at 72 ppb.

(10) *Comment:* Some commenters also stated that the Kim et al. (2011) study is missing critical information and its study design makes comparison to the other studies difficult. That is, the commenter suggests that data at times other than pre- and post-exposure should have been provided.

Response: The EPA disagrees with this comment. With regard to providing data at other time points besides pre- and post-exposure, there is no standard that suggests an appropriate frequency at which lung function should be measured in prolonged 6.6-hour exposure studies. The Adams (2006) study showed that lung function decrements during O_3 exposures with moderate exercise become most apparent following the third hour of exposure. As such, it makes little sense to measure lung function during the first couple hours of exposure. However, having data at multiple time points toward the end of an exposure can provide evidence that the mean post-exposure FEV_1 response is not a single anomalous data point. The FEV_1 response data for the 3-, 4.6-, 5.6-, and 6.6-hour time points of the Kim et al. (2011) study are available in Figure 6 of the McDonnell et al. (2012) paper where they are plotted with the Adams (2006) data for 60 ppb. Similar to the Adams (2006) study, the responses at 5.6 hours are only marginally smaller than the response at 6.6 hours in the Kim et al. (2011) study. This indicates that the post-exposure FEV_1 responses in both studies are consistent with responses at an earlier time point and thus not likely to be anomalous data.

Additional Studies

Several commenters analyzed the data from controlled human exposure studies, or they commented on the EPA's analysis of the data from some of these studies (Brown et al., 2008), to come to a different conclusion than the EPA's interpretation of these studies thereby questioning the proposed decision that the current standard is not adequate to protect public health.

(11) *Comment:* One commenter submitted an independent assessment of the scientific evidence and risk, and used this analysis to assert that there are multiple flaws in the underlying studies and their interpretation by the EPA. This commenter stated that the EPA's discussion of the spirometric responses of children and adolescents and older adults to O_3 was misleading. They claimed that the EPA did not mention that "the responses of children and adolescents are equivalent to those of young adults (18-35 years old; McDonnell et al., 1985a) and that this response diminishes in middle-aged and older adults (Hazucha, 1985[18])" (e.g., TCEQ).

Response: The EPA notes that the commenter misrepresented our characterization of the effect of age on FEV_1 responses to O_3 and asserted mistakenly that EPA did not mention diminished responses on older adults. In fact, the proposal clearly states that, "Respiratory symptom responses to O_3 exposure appears to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996b); lung function responses to O_3 exposure also decline from early adulthood (U.S. EPA, 1996b)" (79 FR 75267) (see also PA, p. 3-82). With regard to differences between children and adults, it was clearly stated in the ISA (p. 6-21) that healthy children exposed to filtered air and 120 ppb O_3 experienced similar spirometric responses, but lesser symptoms than similarly exposed young healthy adults (McDonnell et al., 1985a). In addition, the EPA's approach to modeling the effect of age on responses to O_3 is clearly provided in the HREA (Table 6-2).

(12) *Comment:* Some commenters asserted that it is improper to compare responses following O_3 exposures to those following filtered air. For example, commenters stated that the EPA's treatment of filtered air responses in the dose-response curve was incorrect. These commenters claimed that when creating a dose-response curve, it is most appropriate to include a zero-dose point and not to subtract the filtered air response from responses to O_3. Other commenters also claimed that it was improper to compare responses following O_3 exposure to those following filtered air exposure.

Response: Contrary to this assertion, EPA correctly adjusted FEV_1 responses to O_3 by responses following filtered air, as was also done in the McDonnell et al. (2012) model. As indicated in the ISA (p. 6-4), the majority of controlled human exposure studies investigating the effects of O_3 are of a randomized, controlled, crossover design in which subjects were exposed, without knowledge of the exposure condition and in random order, to clean filtered air and, depending on the study, to one or more O_3 concentrations. The filtered air control exposure provides an unbiased estimate of the effects of the experimental procedures on the outcome(s) of interest. Comparison of responses following this filtered air exposure to those following an O_3 exposure allows for estimation of the effects of O_3 itself on an outcome measurement while controlling for independent effects of the experimental procedures, such as ventilation rate. Thus, the commenter's

[18] The citation submitted by the commenter (Hazucha et al., 1985) appears to be incorrect. In responding to this comment, EPA assumes that the commenter meant the citation to be Hazucha et al. (2003).

approach does not provide an estimate of the effects of O_3 alone. Furthermore, as illustrated in these comments, following "long" filtered air exposures, there is about a 1% improvement in FEV_1. By not accounting for this increase in FEV_1, the commenter underestimated the FEV_1 decrement due to O_3 exposure. The commenters' approach thus is fundamentally flawed.

(13) *Comment:* The commenter also asserted that the McDonnell et al. (2012) model and exposure-response (E-R) models incorrectly used only the most responsive people and that EPA's reliance on data from clinical trials that use only the most responsive people irrationally ignores large portions of relevant data.

Response: The EPA rejects this assertion that the McDonnell et al. (2012) model and the E-R analysis ignored large portions of relevant data, as this assertion is demonstrably incorrect. The McDonnell et al. (2012) model was fit to all of the available O_3-induced (i.e., corrected for filtered air response) FEV_1 response data from EPA and University of California at Davis studies. In total, data were available for 741 individuals and the model was fit to their 8,477 O_3-induced FEV_1 responses from all time points where measurements made in studies (i.e., reflecting all available data for O_3-induced changes in FEV_1). Subsequently, as illustrated by the figures in the McDonnell et al. (2012) paper and described in the text of paper, the model was fit to all available FEV_1 data measured during the course of O_3 exposures, including exposures shorter than 6.6 hours. Thus, the model predicts temporal dynamics of FEV_1 response to any set of O_3 exposure conditions that might reasonably be experienced in the ambient environment, predicting the mean responses and the distribution of responses around the mean. For the HREA, the proportion of individuals, under variable exposure conditions, predicted to have FEV_1 decrements $\geq$ 10, 15 and 20% was estimated. Finally, the commenter referenced the exposure-response model on p. 6-18 of the HREA. However, they neglected to note that this was in a section describing the exposure-response function approach used in prior reviews (HREA, starting on p. 6-17). Thus, the commenter confused the exposure-response model used in the last review with the updated approach used in this review.

(14) *Comment:* The commenter also stated that EPA did not properly consider O_3 dose when interpreting the human clinical data. Ozone total dose includes three factors: duration of exposure, concentration, and ventilation rate. The commenter claimed the EPA emphasized only concentration without properly considering and communicating duration of exposure and ventilation rate. Further, they asserted that because people are not exposed to the same dose, they cannot be judged to have the same exposure and would therefore not be expected to respond consistently.

Response: The EPA rejects this claim that we emphasized only concentration without properly incorporating the other two factors. As noted in the ISA, total O_3 dose does not describe the temporal dynamics of FEV_1 responses as a function of concentration, ventilation rate, time and age of the exposed individuals (ISA, p. 6-5). Thus, the use of total O_3 dose is antiquated and the EPA therefore conducted a

more sophisticated analysis of FEV_1 response to O_3 in the HREA. In this review, the HREA estimates risks of lung function decrements in school-aged children (ages 5 to 18), asthmatic school-aged children, and the general adult population for 15 urban study areas. A probabilistic model designed to account for the numerous sources of variability that affect people's exposures was used to simulate the movement of individuals through time and space and estimate their exposure to O_3 while occupying indoor, outdoor, and in-vehicle locations. That information was linked with the McDonnell et al. (2012) model to estimate FEV_1 responses over time as O_3 exposure concentrations and ventilation rates changed. As noted earlier, CASAC agreed that this approach is both scientifically valid and a significant improvement over approaches used in past O_3 reviews (Frey, 2014a, p. 2).

(15) *Comment:* Several commenters criticized the EPA analysis published by Brown et al. (2008). One commenter suggested that the EPA needed to state why the Brown et al. (2008) analysis was relied on rather than Nicolich (2007) or Lefohn et al. (2010). Further, commenters stated that the analysis of the Adams (2006) data in Brown et al. (2008) was flawed. Among other reasons, commenters expressed the opinion that it was not appropriate for Brown et al. (2008) to only examine a portion of the Adams (2006) data. The commenters argued that Brown et al. (2008) inappropriately excluded all pulmonary function data at interim hourly time points and exposure levels within the 6.6-hour exposure and only used the final response information at 6.6 hours. Commenters contended that the approach is not appropriate and that the statistically significant results were mostly likely due to a majority of the data being selectively omitted from the analysis. Commenters suggested that there is little certainty that the lung function decrements reported in Brown et al. (2008) were anything more than normal inter-individual variability. Commenters argued that EPA should have placed more weight on analyses that used methods and approaches that incorporated all of the exposure concentrations and time points.

Response: The EPA disagrees with these commenters.[19] As an initial matter, Nicolich (2007) was a public comment and is not a peer-reviewed publication that would be used to assess the scientific evidence for effects of O_3 on lung function in the ISA. The Nicolich (2007) comments were specifically addressed by the EPA on pp. 24-25 in the Response to Comments Document for the 2007 proposed rule (U.S. EPA, 2008a). On page A-3 of his comments (Nicolich, 2007), Dr. Nicolich stated "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." The EPA

[19] The D.C. Circuit has held that EPA reasonably used and interpreted the Brown (2007) analysis in the last review. *Mississippi*, 744 F. 3d at 1347. In this review, there is now additional corroborative evidence supporting the Brown (2007) analysis, in the form of further controlled human clinical studies finding health effects in young, healthy adults at moderate exercise at O_3 concentrations of 60 ppb over a 6.6 hour exposure period.

reasonably chose not to rely on this analysis: "Therefore, given that the underlying statistical assumptions required for his analyses were not met and that significance levels are questionable, in EPA's judgment the analyses presented by Dr. Nicolich are ambiguous" (U.S. EPA, 2008a). It is likely that the Lefohn et al. (2010) analysis of the Adams (2006) data would similarly not meet the statistical assumptions of the model (e.g., homoscedasticity). In contrast, recognizing the concerns related to the distribution of responses, Brown et al. (2008) conservatively used a nonparametric sign test to obtain a p-value of 0.002 for the comparison responses following 60 ppb O_3 versus filter air. Other common statistical tests also showed significant effects on lung function. In addition, the effects of 60 ppb O_3 on FEV_1 responses in Brown et al. (2008) remained statistically significant even following the exclusion of three potential outliers.

EPA disagrees with the comment stating that it was not appropriate for Brown et al. (2008) to only examine a portion of the Adams (2006) data. In fact, there is no established single manner or protocol decreeing that data throughout the protocol must be analyzed and included. Furthermore, Brown et al. (2008) was a peer-reviewed journal publication. CASAC also expressed favorable comments in their March 30, 2011, letter to Administrator Jackson. With reference to a memorandum (Brown, 2007) that preceded the Brown et al. (2008) publication, on p. 6 of the CASAC Consensus Responses to Charge Questions it was stated, "The results of the Adams et al. study also have been carefully reanalyzed by EPA investigators (Brown et. al., [2008]), and this reanalysis showed a statistically significant group effect on FEV_1 after 60 ppb ozone exposure." On p. A-13, a CASAC panelist and biostatistician stated, "Thus, from my understanding of the statistical analyses that have been conducted, I would argue that the analysis by EPA should be preferred to that of Adams for the specific comparison of the FEV_1 effects of 0.06 ppm exposure relative to filtered air exposure." (Samet, 2011, p. A-13).

Threshold

(16) *Comment:* Several commenters used the new McDonnell et al. (2012) and Schelegle et al. (2012) models to support their views about the O_3 concentrations associated with a threshold for adverse lung function decrements. For example, one commenter who supported retaining the current standard noted that McDonnell et al. (2012) found that the threshold model fit the observed data better than the original (no-threshold) model, especially at earlier time points and at the lowest exposure concentrations. The commenter expressed the view that the threshold model showed that the population mean FEV_1 decrement did not reach 10% until exposures were at least 80 ppb, indicating that O_3 exposures of 80 ppb or higher may cause lung function decrements and other respiratory effects.[20]

[20] Conversely, another group of commenters who supported revising the standard to a level of 60 ppb noted that the results of these models are consistent with the results of controlled human exposure studies

Response: As described in the preamble to the final rule in section II.A.1.b, the McDonnell et al. (2012) and Schelegle et al. (2012) models represent a significant technical advance in the exposure-response modeling approach since the last review, and these models indicate that a dose-threshold model fits the data better than a non-threshold model. However, the EPA disagrees that using the predicted group mean response from the McDonnell model provides support for retaining the current standard. As discussed in the preamble to the final rule, the group mean responses do not convey information about interindividual variability, or the proportion of the population estimated to experience the larger lung function decrements (e.g., 10 or 15% FEV_1 decrements) that could be adverse. In fact, it masks this variability. These variable effects in individuals have been found to be reproducible. In other words, a person who has a large lung function response after exposure to O_3 will likely have about the same response if exposed again in a similar manner. Group mean responses are not representative of this segment of the population that has much larger than average responses to O_3.

Inflammation

(17) *Comment:* Some commenters asserted that the pulmonary inflammation observed following exposure to 60 ppb in the controlled human exposure study by Kim et al. (2011) was small and unlikely to result in airway damage. Commenters argued that the immune system responses discussed in the Proposed Rule as the first indications of inflammation are physiological processes that occur in all living organisms under the stimuli of daily life. These first reported changes are small and reversible and well within the range of physiological variability. These changes are considered biochemical markers that the ATS guidelines indicate do not necessarily imply adversity. Another commenter contended that pulmonary inflammation as an indicator for long-term O_3 effects remains unreliable depending on the variability and range of lung changes.

Response: The EPA recognized in the proposal (79 FR 75252) and the ISA (p. 6-76) that inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely. Thus, the inflammatory response observed following the single exposure to 60 ppb in the study by Kim et al. (2011) is not necessarily an adverse response. However, the EPA notes that it is also important to consider the potential for continued acute inflammatory responses to evolve into a chronic inflammatory state and to affect the structure and function of the lung.[21] The Administrator considers this possibility through her consideration of estimated exposures of concern for the 60 ppb benchmark

finding adverse health effects at 60 ppb. These comments are discussed in the preamble to the final rule (section II.C.4.b), within the context of the Administrator's decision on a revised standard level.

[21] Inflammation induced by exposure of humans to O_3 can have several potential outcomes, ranging from resolving entirely following a single exposure to becoming a chronic inflammatory state (ISA, section 6.2.3). Lung injury and the resulting inflammation provide a mechanism by which O_3 may cause other more serious morbidity effects (e.g., asthma exacerbations) (ISA, section 6.2.3). See generally section II.A.1.a in the preamble to the final rule.

(see sections II.B.3 and II.C.4 in the preamble to the final rule). As discussed in detail in section II.C.4.b of the preamble to the final rule, while she judges that there is important uncertainty in the adversity of the effects shown to occur following exposures to 60 ppb O_3, including the inflammation reported by Kim et al. (2011), she gives some consideration to estimates of two or more exposures of concern for the 60 ppb benchmark (i.e., as a health-protective surrogate for repeated exposures of concern at or above 60 ppb), particularly when considering the extent to which the current and revised standards incorporate a margin of safety.

(18) *Comment:* One commenter cited a California Air Resources Board (CARB) report by Balmes et al. (2011) that reported that exercise of the same intensity as in the controlled human exposure studies alone produced systemic inflammation that was on the same order of magnitude as the pulmonary inflammation reported in the Kim et al. (2011) study.

Response: Balmes et al. (2011) stated, "The repeated-measure cross-over design used in many of inhalational exposure studies may control for the confounding effects of exercise; however, the exercise effects may still overwhelm the pollutant signal and make it undetectable." Restated, the authors' concern was that the effect of exercise on systemic inflammation would prevent the detection of a pollutant's effect on inflammation. However, it is not clear why the commenter compared systemic inflammation to pulmonary inflammation. Regardless, the EPA notes that a statistically significant increase in lung inflammation was observed in the Kim et al. (2011) following exposure to 60 ppb O_3 relative to filtered air, thus demonstrating that the response was not the result of exercise.

(19) *Comment:* One commenter quoted CASAC advice that lung function decrements of 10% FEV_1 "are usually associated with inflammatory changes, such as more neutrophils in the bronchoalveolar lavage fluid" (Frey, 2014a, p. 2). The commenter views both of these effects as adverse and supporting a standard set at a level of 60 ppb.

Response: With respect to CASAC advice on the adversity of lung function decrements, as discussed in the comments on controlled human exposure studies in the preamble to the final rule (section II.C.3.b.i), the Administrator notes that, while CASAC clearly advised the EPA to consider the health effects shown to occur following exposures to 60 ppb O_3, its advice regarding the adversity of those effects is less clear. In particular, she notes that CASAC was conditional about whether the lung function decrements observed in some people at 60 ppb (i.e., FEV_1 decrements $\geq$ 10%) are adverse. Specifically, CASAC stated that these decrements "could be adverse in individuals with lung disease" (Frey, 2014b, p. 7) and that they provide a "surrogate for adverse health outcomes for people with asthma and lung disease" (Frey, 2014b, p. 3). Further, CASAC did not recommend considering standard levels low enough to eliminate O_3-induced FEV_1 decrements $\geq$ 10% (Frey, 2014b). With regard to the full range of effects

shown to occur at 60 ppb (i.e., FEV1 decrements, airway inflammation), CASAC stated only that exposures of concern for the 60 ppb benchmark are "relevant for consideration" with respect to people with asthma (Frey, 2014b, p. 6). The Administrator contrasts these statements with CASAC's clear advice that "the combination of decrements in FEV_1 together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014b, p. 5).

With respect to the adversity of neutrophils in lavage fluid, in its most recent adversity guidelines the ATS (2000) concluded that elevations of biomarkers such as cell numbers and types, cytokines, and reactive oxygen species may signal risk for ongoing injury and more serious effects or may simply represent transient responses, illustrating the lack of clear boundaries that separate adverse from nonadverse events. With regard to ATS, the Administrator first notes the recommendations that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" and that "[f]ew…biomarkers have been validated sufficiently that their responses can be used with confidence to define the point at which a response should be equated to an adverse effect warranting preventive measures" (ATS, 2000). Based on these recommendations, compared to effects following exposures at or above 72 ppb, the Administrator has less confidence in the adversity of the respiratory effects that have been observed following exposures to 60 or 63 ppb.

Additional Technical Comments

In addition to the comments addressed above and in the preamble to the final rule, EPA received a number of comments on the assessment of controlled human exposure studies in the proposal. Comments on EPA's interpretation and assessment of the body of controlled human exposure evidence are discussed first, followed by comments on specific controlled human exposure studies.

(20) *Comment:* Some commenters, primarily representing medical, public health, and environmental groups, noted that controlled human exposure studies have shown a variety of effects following acute O_3 exposures. Respiratory effects highlighted by commenters include decreased breathing capacity, rapid and shallow breathing, painful inhalation, increased respiratory tract inflammation, and increased epithelial permeability. With regard to inflammation, these commenters specifically noted increased airway inflammation following exposures at or above 80 ppb O_3, based on earlier studies, and following 6.6 hour exposures to 60 ppb O_3, based on a more recent study. Commenters indicated that inflammation is a host response to injury and that the presence of inflammation is an indication that injury occurred. With regard to lung function, these commenters also highlighted findings that some healthy adults experience 10% FEV_1 decrements following exposures to 60 ppb O_3. These commenters also noted that controlled human exposure studies indicate negative cardiovascular effects in response to short-term ozone exposure, including changes in heart rate variability and blood markers of

systemic inflammation and oxidative stress, further supporting effects observed in toxicological studies. These commenters further maintained that individuals with pre-existing pulmonary disease are likely to experience adverse health impacts based on O_3 exposures at concentrations of 60 ppb (citing CASAC advice in the 2008 review) (e.g., ALA et al.). The same commenters indicated that a lung function decrement of 10% would be adverse to individuals with asthma, citing CASAC advice from the current review in support.

Commenters that highlighted these effects generally concluded that evidence from controlled human exposure studies necessitates a standard no higher than 60 ppb. In reaching this conclusion, these commenters noted that controlled human exposure studies evaluate healthy adults, not sensitive populations such as children or individuals with asthma, that have been shown to experience larger decrements in lung function in response to O_3 exposure than healthy adults. They also note that the significant amount of variation in individual responses may mean that, in the general population, a large subset of healthy adults will have similar significant enhanced responses. These commenters generally argued that the evidence from controlled human exposure studies supports lowering the level of the primary standard to 60 ppb or below, and that a standard level within EPA's proposed range of 65 to 70 ppb cannot be considered protective of public health and that there is a legal requirement to protect sensitive populations with an adequate margin of safety.

Other commenters, primarily representing industry, businesses, and some states, expressed concern regarding uncertainty in the evidence from controlled human exposure studies. These commenters often noted that there is greater confidence and less uncertainty in the upper end of the range of O_3 exposure concentrations discussed in the Proposed Rule. These commenters generally contended that the available evidence does not support setting a standard level as low as 70 ppb (e.g., in one case stating that there is a lack of statistically significant data for setting the standard level below 72 ppb). In some cases, such commenters asserted that the Administrator did not justify her selection of *"two or more"* exposures as the appropriate criteria for judging risk for adverse effects. These commenters claimed that the EPA failed to present any scientific evidence documenting this effect or supporting the selection of *"two"* as the critical value that could lead to adverse effects.

Response: A wide range of health effects have been reported in controlled human exposure studies following O_3 exposures. We agree with commenters from medical, public health, and environmental groups that these are important studies and that they have important implications for the EPA's decision on the standard level. We also agree with these commenters that NAAQS are to be set to protect the public health, including the health of at-risk populations. However, as described below, we do not agree with these commenters' conclusion that controlled human exposure studies necessitate a standard level no higher than 60 ppb.

As discussed in sections II.B.2, II.B.3, and II.C.4 of the preamble to the final rule, when considering the extent to which alternative standard levels would be expected to limit population exposures to the range of O_3 concentrations shown to cause respiratory effects, the Administrator considers the extent to which such a standard would be expected to limit the occurrence of O_3 exposures of concern at or above 60, 70, and 80 ppb.[22] In doing so, she notes that an O_3 standard established at a particular level can provide protection against a range of exposure concentrations, including concentrations below the standard level. This is because the degree of protection provided by any NAAQS is due to the combination of all of the elements of the standard (i.e., indicator, averaging time, form, level). In the case of the 4th maximum form of the O_3 NAAQS, which the Administrator is retaining in the current review (section II.C.3 of the preamble to the final rule), the large majority of days in areas that meet the revised standard will have 8-hour O_3 concentrations below the level of the standard.

In considering exposures of concern at or above 60, 70, and 80 ppb, the Administrator judges that the evidence supporting the occurrence of adverse respiratory effects is strongest for exposures at or above the 70 and 80 ppb benchmarks (see "adversity" section above, and sections II.B.2.b.i, II.C.4.b, and II.C.4.c of the preamble to the final rule). While the Administrator has less confidence that adverse effects will occur following exposures to O_3 concentrations as low as 60 ppb, she notes the possibility for adverse effects following such exposures given that (1) CASAC has indicated the moderate lung function decrements (i.e., FEV_1 decrements $\geq$ 10%) that occur in some healthy adults following exposures to 60 ppb O_3, which are large enough to be judged an abnormal response by ATS, could be adverse to people with lung disease (see "adversity" section above, and sections II.A.1.c, II.B.2, II.B.3, and II.C.4 of the preamble to the final rule), and that (2) airway inflammation has been reported following exposures as low as 60 ppb O_3. She also takes note of CASAC advice that the occurrence of exposures of concern at or above 60 ppb is an appropriate consideration for people (including children) with asthma (Frey, 2014b, p. 6).

Due to interindividual variability in responsiveness, the Administrator further notes that not every occurrence of an exposure of concern will result in an adverse effect.[23] Repeated occurrences of some of the effects demonstrated following exposures of concern could increase the likelihood of adversity. For example, as discussed in the ISA (section 6.2.3), while the airway inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve

[22] Unlike respiratory effects, controlled human exposure studies have not shown cardiovascular effects at O_3 exposure concentrations near or below the level of the current standard (ISA, Chapter 6; e.g., section 6.3.4). Thus, compared to studies of respiratory effects following exposures from 60 to 80 ppb, the available controlled human exposure studies of cardiovascular effects are less informative to decisions on the current and alternative O_3 standards.

[23] For most of the effects demonstrated in controlled human exposure studies (e.g., airway inflammation, AHR, decreased lung host defense, respiratory symptoms) the available data are not sufficient to quantify the number of people who would experience adverse effects due to O_3 exposures.

entirely, continued inflammation could potentially result in adverse effects, including the induction of a chronic inflammatory state; altered pulmonary structure and function, leading to diseases such as asthma; altered lung host defense response to inhaled microorganisms, particularly in potentially at-risk populations such as the very young and old; and altered lung response to other agents such as allergens or toxins. The Administrator notes that the types of lung injury that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O_3 may cause other more serious effects. Therefore, the Administrator is most concerned about protecting at-risk populations against repeated occurrences of exposures of concern.

In considering the appropriate metric for evaluating repeated occurrences of exposures of concern, the Administrator acknowledges that it is not clear from the evidence, or from the ATS recommendations, CASAC advice, or public comments, how particular numbers of exposures of concern could impact the seriousness of the resulting effects, especially at lower exposure concentrations. Therefore, the Administrator judges that focusing on HREA estimates of two or more exposures of concern provides a health-protective approach to considering the potential for repeated occurrences of exposures of concern that could result in adverse effects. She notes that other possible metrics for considering repeated occurrences of exposures of concern (e.g., 3 or more, 4 or more, etc.) would result in smaller exposure estimates.

Based on the above considerations, the Administrator focuses primarily on the extent to which a revised standard would be expected to protect populations from experiencing two or more O_3 exposures of concern (i.e., as a surrogate for repeated exposures). While she emphasizes the importance of limiting two or more exposures and reducing their occurrence, compared to the current standard, she balances this emphasis by noting that (1) not all exposures of concern will result in adverse effects; and (2) she has less confidence in the occurrence of adverse effects at the 60 ppb benchmark than at the 70 or 80 ppb benchmarks. Therefore, in using estimates of exposures of concern to inform her decisions on alternative standard levels, the Administrator judges that it would not be appropriate to set a standard intended to eliminate all exposures of concern for all benchmarks, particularly the 60 ppb benchmark. Her consideration of specific estimates of exposures of concern is discussed below.

As illustrated in Table 1 of the preamble to the final rule, the Administrator notes that, in the urban study areas, a revised standard with a level of 70 ppb would be expected to eliminate the occurrence of two or more exposures of concern to O_3 concentrations at and above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O_3 concentrations at and above 70 ppb, even in the worst-case urban study area and year. For the 70 ppb benchmark, this reflects about a 90% reduction in the number of children experiencing two or more exposures of concern, compared to the current standard (see Table 1 of the preamble to the final rule).

Although the Administrator is less concerned about single occurrences of exposures of concern, she acknowledges that even single exposures to O_3 concentrations at or above benchmark concentrations (particularly for the 70 and 80 ppb benchmarks) could potentially result in adverse effects. To the extent this may be the case, the Administrator notes that a standard with a level of 70 ppb would also be expected to virtually eliminate exposures of concern at or above 80 ppb and to protect about 99% or more children from experiencing even single exposures of concern at or above 70 ppb. For the 70 ppb benchmark, this reflects more than a 70% reduction in the number of children experiencing one or more exposures of concern, compared to the current standard (Table 1 of the preamble to the final rule).

Though the Administrator also acknowledges greater uncertainty with regard to the occurrence of adverse effects following exposures of concern at or above 60 ppb, she notes that a revised standard with a level of 70 ppb would be expected to protect the large majority of children in the urban study areas (i.e., about 96% to more than 99% of children in individual urban study areas) from experiencing two or more exposures of concern at or above 60 ppb. Compared to the current standard, this represents a reduction of more than 60% in the occurrence of two or more exposures of concern (Table 1 of the preamble to the final rule). A level of 70 ppb is also estimated to achieve important reductions, compared to the current standard, in the occurrence of one or more exposures of concern at or above 60 ppb (i.e., almost a 50% reduction in the number of children estimated to experience such exposures) (Table 1 of the preamble to the final rule).

Based on the above information, the Administrator concludes that a revised O_3 standard with a level of 70 ppb would be expected to eliminate, or almost eliminate, O_3 exposures of concern for the 70 and 80 ppb benchmarks; to protect the large majority of children from experiencing two or more exposures of concern at or above 60 ppb; and to substantially reduce the occurrence of one or more O_3 exposures of concern for the 60 ppb benchmark, compared to the current standard.

For the reasons discussed above, the Administrator does not agree with commenters who argue that controlled human exposure studies necessitate a standard level of 60 ppb or below. In particular, she notes that a decision to set the primary O_3 standard level at 60 ppb would place a large amount of weight on the potential public health importance of virtually eliminating even single occurrences of exposures of concern at and above 60 ppb, despite uncertainties in the adversity of effects at 60 ppb. Thus, although the Administrator agrees that it is appropriate to consider estimated exposures of concern for the 60 ppb benchmark (based on the evidence and CASAC advice, described above), particularly when such exposures occur repeatedly, she does not agree that even single occurrences of these exposures need to be eliminated in order to protect public health with an adequate margin of safety.

(21) *Comment:* Other commenters (e.g., ALA et al.) argued that controlled human exposure studies are not representative of real-world exposures. These commenters noted that such studies underestimate effects because they do not take into account other pollutants and environmental conditions that could exacerbate the effects of O_3.

Response: As discussed in the preamble to the final rule (sections II.B.1.e, II.B.3, and II.C.4), the Administrator places the greatest weight on the results of controlled human exposure studies and on exposure and risk analyses based on information from these studies. In doing so, she notes that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following exposures to specific O_3 concentrations. The effects reported in these studies are due solely to O_3 exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). She further notes the CASAC judgment that "the scientific evidence supporting the finding that the current standard is inadequate to protect public health is strongest based on the controlled human exposure studies of respiratory effects" (Frey, 2014b, p. 5). Consistent with this emphasis, the HREA conclusions reflect relatively greater confidence in the results of the exposure and risk analyses based on information from controlled human exposure studies (i.e., exposures of concern and risk of lung function decrements) than the results of epidemiology-based risk analyses, given the greater uncertainties in the epidemiology-based risk estimates (HREA, section 9.6). For all of these reasons, the Administrator has the most confidence in using the information from controlled human exposure studies to reach proposed conclusions on alternative standard levels.

While the Administrator acknowledges that co-exposures to other air pollutants could potentially impact the O_3-attributable health effects shown to occur in controlled human exposure studies, she judges that the very limited amount of evidence on this issue does not provide a basis for reaching conclusions on what those impacts may be. She notes that the evidence is particularly limited for co-pollutants other than PM, for O_3 concentrations at or near those present in the ambient air, and for effects on the respiratory system (ISA, section 5.4.2.6). In addition to these limitations, in assessing co-pollutant studies the ISA states that "[r]esults are highly variable and depend on whether exposures are simultaneous or sequential, the type of PM employed and the endpoint examined" (ISA, p. 5-69).

(22) *Comment:* One commenter pointed to a study by Carranza Rosenzweig et al. (2004) to support the contention that lung function was not well correlated with symptoms in asthmatics.

Response: While true for baseline conditions (r=0.09), Carranza Rosenzweig et al. (2004) found that following 12 weeks of asthma treatment (e.g., inhaled corticosteroids), there was a moderate correlation between improvement in FEV_1 and overall quality of life scores (r=0.38). These authors also noted (p. 1163) that

patients live with their disease by avoiding or omitting from their lifestyle those things that impact their disease and have the potential to exacerbate their symptoms. Thus, for individuals with asthma, it is important to consider the potential effects air pollution on lung function and quality of life.

(23) *Comment:* Commenters questioned EPA's evaluation of repeated occurrences of exposures of concern, suggesting that the data presented is limited to one or more and two or more occurrences, rather than also presenting the data for five or more, ten or more, etc. These commenters contend that such data could be used to evaluate the likelihood of responses repeatedly during the course of the year that might lead to a more serious health condition.

Response: The Administrator's consideration of "repeated" exposures of concern is discussed in sections II.B.3 and II.C.4 of the preamble to the final rule. Due to interindividual variability in responsiveness, the Administrator agrees that not every occurrence of an exposure of concern will result in an adverse effect. Repeated occurrences of some of the effects demonstrated following exposures of concern could increase the likelihood of adversity. For example, as discussed in the ISA (section 6.2.3), repeated occurrences of airway inflammation could lead to the induction of a chronic inflammatory state; altered pulmonary structure and function, leading to diseases such as asthma; altered lung host defense response to inhaled microorganisms, particularly in potentially at-risk populations such as the very young and old; and altered lung response to other agents such as allergens or toxins. The Administrator notes that the types of lung injury that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O_3 may cause other more serious effects. Therefore, the Administrator is most concerned about protecting at-risk populations against repeated occurrences of exposures of concern.

To provide insight into the potential for repeated exposures of concern, the Administrator focuses on HREA estimates of two or more such exposures. In doing so, she recognizes that there is no single definition of the term "repeated" that would appropriately characterize the potential for adverse effects for all at-risk populations and health endpoints. Therefore, in order to be confident that the revised O_3 standard appropriately protects against repeated occurrences of exposures of concern, the Administrator focuses her considerations on estimates of two or more such exposures rather than on estimates of a larger number of occurrences.

(24) *Comment:* Some commenters, primarily industry groups and business associations, contended that the controlled human exposure studies were not designed to assess ozone effects in individuals. Some commenters suggested that respiratory effects are more consistent with exposure greater than 0.080 ppm, that group mean responses at 0.072 ppm are not adverse, and that it is not appropriate to extrapolate the results from the few individuals in the controlled human exposure studies to the entire U.S. population. A few commenters argued that the Administrator failed to recognize that group mean effects at 0.060 ppm and 0.072

are not adverse and are inconsistent, and that airway inflammation results are inconsistent among individuals. One commenter specifically stated that the controlled human exposure studies are insufficient to show that a sensitive group will experience a statistically significant adverse effect following exposure below the current standard, and that none of the studies found statistically significant group mean responses that meet EPA's definition of adversity.

Response: In assessing the evidence from controlled human exposure studies, the ISA notes the following:

> Consideration of group mean changes is important in discerning if observed effects are due to O_3 exposure rather than chance alone. Inter-individual variability in responses is, however, considerable and pertinent to assessing the fraction of the population that might actually be affected during an O_3 exposure.

Thus, when characterizing the potential for health impacts following O_3 exposures, the ISA evaluated group mean responses and, when available, distributions of responses of the individual study participants.[24] In particular, the ISA combined the individual data from multiple studies of healthy adults exposed for 6.6 hours to 60 ppb O_3 (Kim et al., 2011; Schelegle et al., 2009; Adams, 2006, 2002, 1998). Based on these data, the ISA reports that 10% of exposed subjects experienced moderate FEV_1 decrements $\geq$ 10% (i.e., abnormal, based on ATS criteria, and large enough to be potentially adverse for people with pulmonary disease, based on CASAC advice) (ISA, section 6.2.1.1).

Consistent with these findings, recently developed empirical models based on data from individual study participants predict the onset of O_3-induced FEV_1 decrements in some healthy adults that can occur following exposures to 60 ppb O_3 for 4 to 5 hours while at moderate, intermittent exertion (Schelegle et al., 2012), with the models estimating that 9% of healthy adults exposed to 60 ppb O_3 for 6.6 hours would experience FEV_1 decrements greater than or equal to 10% (McDonnell et al., 2012) (ISA, section 6.2.1.1). When the evidence for O_3-induced lung function decrements was taken together, the ISA concluded that (1) "mean FEV_1 is clearly decreased by 6.6-h exposures to 60 ppb O_3 and higher concentrations in subjects performing moderate exercise" (ISA, p. 6-9) and (2) although group mean decrements following exposures to 60 ppb O_3 are

[24] With regard to group mean FEV_1 responses, the ISA concluded that "there is a smooth intake dose-response curve without evidence of a threshold for exposures between 40 and 120 ppb O_3" (ISA, p. 6-9). This conclusion is based on the consistent results reported by the available studies that evaluated 6.6-hour O_3 exposures in healthy adults engaged in moderate, quasi-continuous exertion (i.e., Kim et al., 2011; Schelegle et al., 2009; Adams, 2006, 2003, 2002; McDonnell et al., 2001; Horstman et al., 1990; Folinsbee, 1988). Figure 6-1 of the ISA illustrates the consistency of results across these studies, and their consistency with model predictions based on a larger body of studies (i.e., compare Figure 6-1 with Figure 6-3 in the ISA).

biologically small, "a considerable fraction of exposed individuals experience clinically meaningful decrements in lung function" (ISA, p. 6-20).

As noted in previous responses, looking only at group mean responses masks responses of more sensitive healthy individuals. Yet more sensitive individuals exist among the population of healthy adults, and they are a considerable percentage of individuals exposed at 60 ppb in controlled human exposure studies. As noted in the preamble to the final rule (section II.A.2.b) compared to the healthy young adults included in the controlled human exposure studies, members of at-risk populations could be more likely to experience adverse effects, could experience larger and/or more serious effects, and/or could experience effects following exposures to lower O_3 concentrations. The primary standard is required to provide requisite protection to these at-risk populations.

Given the above information, and CASAC's endorsement of the assessment of the evidence in the ISA and the consideration of this evidence in the PA, the EPA disagrees with commenters who assert that it is not appropriate to consider data on individual participants in controlled human exposure studies.

(25) *Comment:* A commenter who expressed the view that lung function decrements and inflammation have been shown to occur with exposure to 60 ppb, noted limitations to the controlled human exposure studies relied upon by the EPA, including: (1) individuals in controlled human exposure studies are generally young, healthy, nonsmokers - not at-risk subpopulations (e.g., people with asthma, particularly children) who have been found to experience larger lung decrements than healthy adults; (2) exposures were for 6.6 hours, and since effects clearly increase with cumulative dose, the level of an 8-hour standard must be somewhat lower than the level at which effects are observed in the studies due to the greater accumulated dose of ozone; (3) full range of human responses cannot be detected in studies with a small number of young, healthy adults, which are generally unable to access the full range of human responses and individual sensitivity; and, (4) controlled human exposure studies do not fully capture the potential adverse effects of real world ambient O_3 exposures that occur in combination with other pollutants and environmental conditions.

This commenter goes on to note that it is because of the studies' limitations that it is also critical to consider evidence from other types of studies, including toxicological studies and epidemiological studies, in the discussion of health science. Consideration of all these types of studies and evidence together, as in the ISA and by CASAC, provides the most complete picture of ozone-related health effects and physiological mechanisms.

Response: The EPA generally agrees with this comment about the effects found in controlled human exposure studies at 60 ppb, the uses and limitations of controlled human exposure studies, as well as the weight-of-evidence approach to

assess O_3-related effects (as discussed in section II.A of the proposal).[25] While the EPA agrees that this evidence supports the Administrator's decision that the current standard is not adequate to provide requisite public health protection, for the reasons discussed in section II.C.4.c of the preamble to the final rule and in section II.A.2 below, the Administrator asserts that a standard level of 70 ppb will provide adequate protection of public health against the exposure to 60 ppb. As noted in the preamble to the final rule (section II.C.4.c), while she is less confident that adverse effects will occur following exposures to O_3 concentrations as low as 60 ppb, the Administrator also considered estimates of exposures of concern for the 60 ppb benchmark. Consistent with this judgment, CASAC did clearly advise the EPA to consider the extent to which a revised standard is estimated to limit the effects observed following 60 ppb exposures (Frey, 2014b). Therefore, the Administrator considers estimated exposures of concern for the 60 ppb benchmark, and notes that a revised standard with a level of 70 ppb is estimated to protect the vast majority of children in urban study areas (i.e., about 96% to more than 99% of children in individual areas) from experiencing two or more exposures of concern at or above 60 ppb. Compared to the current standard, this represents a reduction of more than 60%.

(26) *Comment:* Some commenters pointed to evidence of a threshold in responses. One commenter suggested for exposures at rest or typical levels of exertion that the threshold for the first mild, transient effects is well above the current standard, between 300 and 500 ppb. These first effects (e.g., small FEV_1 decrements, neutrophilic inflammation, and mild respiratory symptoms) all exhibit threshold behavior and only O_3 exposures of sufficient duration and concentration will being to trigger or activate other defenses. This commenter also pointed to a threshold for inflammatory and symptom responses as well. Additionally, this commenter referenced the Mudway and Kelly (2004) study that the PA and Proposed Rule indicated had reported that PMN influx in healthy subjects is linearly associated with total O_3 dose, stating that the actual paper indicates a threshold in the dose-response. Another commenter cited a review by Honeycutt and Shirley (2014) that suggested for the low concentration studies considered by the EPA (Adams, 2002; Adams, 2006; Schelegle et al., 2009; Kim et al., 2011), a threshold below 70 ppb exists at which there are no statistically significant adverse effects associated with O_3.

Response: The EPA disagrees with the commenters' characterization of thresholds in response to O_3 based on evidence from controlled human exposure studies. It is untrue, for resting or "typical" exposures of healthy individuals, that a level of 300–500 ppb O_3 might represent a threshold for health effects over the 8 hour averaging time of the O_3 NAAQS. In fact, two studies described in the ISA (p. 6-5) reported statistically significant FEV_1 decrements (6-7% group mean) in young health adults after only 2 hours of resting exposure to 500 ppb O_3. One of those studies (Folinsbee et al., 1978), also found a statistically significant FEV_1

[25] See also response above in this section about the representativeness of the study populations in controlled human exposure studies.

decrement (4% group mean) after 2 hours of exposure to 300 ppb O_3 during light exercise (walking 15 minutes with alternating 15 minute periods of rest). Thus, 300–500 ppb O_3 is well beyond any potential threshold for health effects in young healthy adults briefly exposed to for 2 hours. As discussed in the ISA (p. 6-5), the magnitude of respiratory effects (e.g., FEV_1 decrements) is a function of O_3 concentration, minute ventilation, and exposure duration (time). Greater effects of O_3 on lung function will occur as the exposure period is increased beyond 2 hours. Therefore, 300–500 ppb O_3 cannot rationally be viewed as a potential threshold for health effects over an 8 hour period relevant to the NAAQS.

EPA considered thresholds in the context of FEV_1 responses to O_3. The results presented in the HREA are based on an updated dose-threshold model (McDonnell et al., 2012) that estimates FEV_1 responses for individuals following short-term exposures to O_3. The impact of the dose threshold is that O_3-induced FEV_1 decrements result primarily from exposures on days with average ambient O_3 concentrations above about 40 ppb (HREA, section 6.3.1, Figure 6-9). Tables 6-9 and 6-10 of the HREA show that between 42% and 45% of ≥10% FEV_1 decrements were estimated to occur with light exercise levels (<13 L/min-m^2 body surface area). Described in more detail in the HREA discussion accompanying these figures, using a dose-threshold model, ≥10% FEV_1 decrements are likely to occur under realistic ambient exposure concentrations and levels of activity. Moreover, "[t]here is very little difference in response between the threshold and non-threshold models" (HREA, p. 6-15).

The EPA is aware of thresholds discussed by Mudway and Kelly (2004) and Honeycutt and Shirley (2014).[26] The EPA properly described inflammatory responses (i.e. PMN influx) in healthy subjects as linearly associated with total O_3 dose (79 FR 75252; ISA, p. 6-76). Although the Mudway and Kelly (2004) defined a PMN threshold at the 95% confidence interval for responses observed following filtered air exposures, the authors nonetheless used a linear model to fit the increase in PMN with total O_3 dose without regard to the threshold, consistent with the EPA's analysis and approach. The Honeycutt and Shirley (2014) suggestion that 70 ppb O_3 may represent a threshold is overly simplistic. These authors simply pointed to a study showing an adverse response (i.e., the combination of statistically significant FEV_1 decrements and respiratory symptoms) at 72 ppb and other studies not showing adverse responses at 60 ppb following 6.6 hours of exposure. The authors made no attempt to quantify how far below 70 ppb adverse effects may be observed or at what concentrations adverse responses may be expected following an 8 hour period (i.e., the averaging time of the O_3 NAAQS). Nor did the authors account for the fact that the controlled human exposure studies involved healthy subjects rather than at-risk populations, nor that the studies were conducted for 6.6 hours rather than longer exposure periods. Most obviously, the study failed to account for the controlled human exposure studies showing statistically significant health effects (lung function

[26] Honeycutt and Shirley (2014) is a non-peer-reviewed opinion piece included as an appendix to the comments of the Ohio EPA.

decrements, inflammation) having the potential to be adverse at exposure to O_3 concentrations of 60 ppb (in young healthy adults at moderate exercise for 6.6 hours). In summary, EPA properly described the Mudway and Kelly (2004) model, and the Honeycutt and Shirley (2014) assessment of an O_3 threshold concentration is without merit.

(27) *Comment:* Some commenters disagreed with the ISA conclusion that the evidence indicates there is "likely to be a causal relationship between short-term exposures to O_3 and cardiovascular effects" (e.g., ISA, p. 1-8). Some of these commenters contended that the systemic inflammation and cardiac effects reported in controlled exposure studies are not statistically significant or clinically relevant at exposure concentrations below the level of the current standard. To support their position, these commenters pointed to a systematic review (Goodman et al., 2015b) of studies that evaluated biomarkers of systemic inflammation and indicated that, in controlled human exposure studies with O_3, almost no findings were statistically or clinically significant, even with high exposures. For example, one commenter argued that significant increases in three biomarkers of inflammation in the Devlin et al. (2012) study were not clinically relevant and the increased levels were still within normal reference ranges. This commenter argued that uncertainty remained whether inflammatory or cardiovascular effects would be observed at current ambient exposures, given that these exposures are up to an order of magnitude lower than the concentrations used in the controlled human exposure studies.

Response: As an initial matter we agree that, unlike effects in the respiratory system, systemic inflammation and cardiac effects have not been reported in controlled human exposure studies following exposures to O_3 concentrations below the level of the current standard. However, we disagree with commenters who questioned the ISA conclusion that there is likely to be a causal relationship between short-term exposures to O_3 and cardiovascular effects.

The ISA concludes that new toxicological studies, although limited in number, have provided evidence of O_3-induced cardiovascular effects. These effects may, in part, correspond to changes in the autonomic nervous system or to the development and maintenance of oxidative stress and inflammation throughout the body that resulted from inflammation in the lungs. Controlled human exposure studies also suggest cardiovascular effects in response to short-term O_3 exposure, including changes in heart rate variability and blood markers of systemic inflammation and oxidative stress, which provide some coherence with the effects observed in animal toxicology studies. Collectively, the experimental studies provide initial biological plausibility for the consistently positive associations observed in epidemiologic studies of short-term O_3 exposure and cardiovascular mortality. However, the ISA acknowledges that studies in the epidemiologic literature generally have not observed a relationship between short-term exposure to O_3 and cardiovascular morbidity including studies that examined the association between short-term O_3 exposure and cardiovascular-related hospital admissions and emergency department visits and other various cardiovascular

effects. The lack of coherence between the results from studies that examined associations between short-term O_3 exposure and cardiovascular morbidity and cardiovascular mortality complicate the interpretation of the overall evidence for O_3-induced cardiovascular effects. Although there is a lack of coherence with epidemiologic studies of cardiovascular morbidity, the ISA concludes that animal toxicological studies demonstrate O_3-induced cardiovascular effects, and provide support to the strong body of evidence indicating O_3-induced cardiovascular mortality. The ISA concludes that, overall, the body of evidence indicates that there is likely to be a causal relationship between short-term exposures to O_3 and cardiovascular effects, including cardiovascular mortality (e.g., see ISA, pp. 1-7 to 1-8; sections 2.5.2 and 6.3.4).

CASAC endorsed this conclusion. More specifically, based studies discussed in the ISA, CASAC determined the animal toxicological and controlled human exposure studies provided direct evidence for cardiovascular effects and recommended a likely causal determination over a suggestive determination (Samet, 2011, p. 3; Frey and Samet, 2012a, p. 2).

Two papers cited by commenters (Goodman et al. 2014b, 2015a) provided a weight-of-evidence evaluation and concluded that the evidence did not provide a convincing case for a causal relationship, but that the evidence could not provide definitive evidence for a lack of causation. As peer-reviewed publications, the Goodman et al. (2014b, 2015a) papers warrant consideration, but the conclusions of the authors were clearly not subjected to the level of CASAC review and comment of the ISA.[27] As raised by the commenters, the EPA acknowledges that most effects have been observed at high O_3 exposure concentrations with exercise and that some new studies using lower concentrations with resting exposures have not observed statistically significant effects. Although this is an area of uncertainty, the current evidence base is insufficient to conclude that systemic and cardiovascular effects would not occur in some individuals following ambient exposures.

The EPA has provisionally considered new studies, including the studies by Goodman et al. that were highlighted by commenters, in the context of those assessed in the ISA (Appendix A). Based on this provisional consideration, the EPA concludes that the new studies are not sufficient to alter the conclusions reached in the ISA regarding O_3 and cardiovascular effects. The EPA's provisional consideration of these studies did not and could not provide the kind of in-depth critical assessment of the evidence that is provided by the ISA, which has undergone extensive review by CASAC, and has been discussed by CASAC and the public at a series of public meetings. The Goodman et al. papers

[27] Weight-of-evidence (WoE) analysis used by Goodman et al. (2014b and 2015a) greatly reduces the evidence considered in evaluating effects. CASAC did not support this approach of rating studies at the June 2, 2015 public meeting reviewing the 2nd draft NOx ISA (e.g., see Diez Roux and Frey, 2015, p. A-9).

submitted by commenters, together with other new evidence, will be fully assessed in the next review of the O_3 NAAQS.

(28) *Comment:* Commenters from industry (e.g., API, UARG, Dow, TPA) and some states (e.g., OH EPA), pointed to the small change in FEV_1 and suggested that there is large variability in lung function responses between individuals. In the Schelegle et al. (2009) study, five of 31 participants had FEV_1 decrements greater than 10% following exposure to 63 ppb. In an independent analysis of this data, API found that four of the five participants had decrements between 10.2% and 12%, while one had >15% decrement. Eight of 31 participants had better lung function after exposure to 63 ppb O_3 and others had very small decrements. API observed similar trends for exposures to 72 ppb O_3 with only six of 31 participants having FEV_1 decrements of > 10% and five having increased FEV_1. Because O_3 exposure is not expected to be beneficial, commenters suggested that these results indicate that there is substantial inter-individual variability in FEV_1. TPA claimed that in considering the proportion of individuals having >10% FEV_1 decrements, EPA did not adequately account for other factors potentially affecting FEV_1 responses such as exercise. Commenters also stated that lung function returned to normal in all participants within one to four hours following exposure.

Response: The EPA recognizes the importance of intersubject variability in FEV_1 responses to O_3. As described in more detail in this section above and in section II.A.1.b of the preamble to the final rule, the evidence from controlled human exposure studies to date makes it clear that there is considerable variability in responses across individuals, even in young healthy adult volunteers, and that group mean responses are not representative of more responsive individuals. It is consequently important to look beyond group mean responses to the responses of these individuals to evaluate the potential impact on more responsive members of the population. Moreover, relying on group mean changes to evaluate lung function responses to O_3 exposures would mask the responses of the most sensitive groups, particularly where the group mean reflects responses solely among the healthy young adults who were the study participants. The studies of exposures below 80 ppb O_3 show that 10% of young healthy adults experienced FEV_1 decrements > 10% following exposures to 60 ppb O_3, and 19% experienced such decrements following exposures to 72 ppb (under the controlled test conditions involving moderate exertion for 6.6 hours).[28] These percentages would likely have been higher had people with asthma or other at-risk populations been exposed (ISA, pp. 6-17 and 6-18; Frey, 2014a, p. 14; Frey 2014b, p. 7[29]).

[28] These results are consistent with the predictions of a model based on a broader range of studies and O_3 exposure concentrations, as illustrated in Figure 6-3 of the ISA. Figure 6-3 illustrates the proportion of individuals expected to experience >10, 15, and 20% FEV_1 decrements based on McDonnell et al. (2012).

[29] See also *National Environmental Development Associations Clean Action Project v. EPA*, 686 F. 3d 803, 811 (D.C. Cir.2012) (EPA drew legitimate inference that serious asthmatics would experience more serious health effects than clinical test subjects who did not have this degree of lung function impairment).

Such intersubject variability likely reflects real differences in the intrinsic responsiveness of individuals to O_3, as well as some variability in the experimental procedures. Put another way, some healthy individuals are more responsive to O_3 exposure. The fact that other individuals are less responsive does not mean that the effects seen in more responsive individuals are due to chance. It reflects variation in responsiveness. Although the existence of intersubject variability may make it more difficult to find statistical significance, it by no means negates the very real effect of O_3 on lung function of young healthy adults in these studies.

Moreover, that some individuals may have improvements in FEV_1 following O_3 exposure is not surprising given that there is a distribution of responses. Those same individuals having improvements in FEV_1 following O_3 exposure may have had even larger FEV_1 improvements in following filtered air exposures. Thus, even an individual showing some small degree of improvement in FEV_1 following an O_3 exposure may still be experiencing a decrement in function relative to their response following filter air exposure. This is consistent with data illustrated in Figure 6-2 of the ISA. Although there are some individuals at all of the illustrated O_3 exposure concentrations showing some apparent improvement in FEV_1, the distribution of individual responses and the group mean shifts toward larger FEV_1 decrements with increasing O_3 concentration. Furthermore, relative to responses following filtered air exposures, the increases in the group mean FEV_1 decrements following O_3 exposure were statistically significant (i.e., unlikely due to chance) for exposures at and above 72 ppb in the Schelegle et al. (2009) study. An apparent improvement FEV_1 in some individuals following O_3 exposure is consistent with well recognized variability in responses and does not negate findings of statistically significant group mean decrements due to O_3 exposures.

Contrary to commenters' assertions, the controlled human exposure studies relied upon by the EPA did properly control for other factors that could affect FEV_1 responses. In particular, the finding of statistically significant decrements in FEV_1 following O_3 exposure, compared to filtered air controls, indicates that the decrements are due to O_3 exposure itself, and not to other factors. If observed decrements were due to factors other than the O_3 exposure, they would have been evident in the filtered air control condition as well. The importance of filtered air control exposures are discussed in the ISA (pp. 6-4 and 6-5).

With regard to some commenters' observation that lung function decrements return to normal several hours following exposure, EPA has clearly referred to the lung function decrements due to O_3 observed in controlled human exposure studies as transient and reversible. The EPA's consideration of the potential adversity of these decrements is described in detail in this section above and in sections II.B.2.b.i, II.B.3, II.C.4.2, and II.C.4.3 of the preamble to the final rule.

(29) *Comment:* Some commenters objected to the study design used in the Schelegle et al. (2009) study. Specifically, the commenters pointed to the separation

between exposures to filtered air (FA) and O_3, with varying amounts of time for each individual. Commenters argued that the baseline conditions in the two exposure scenarios can be quite different when the FA exposure occurs much earlier than or much later than the O_3 exposure, and that FA may not have been an appropriate control in this study. Commenters contended that the effect of time between the FA and O_3 exposures introduces some degree of uncertainty regarding the magnitude of observed lung function changes. Commenters also argued that the Schelegle et al. (2009) may have overlooked other sources of variability. They provide the example of recent respiratory infections (within 3 weeks of exposure) that may have influenced lung function during the O_3 exposure but not during FA exposure.

Response: Although Schelegle et al. (2009) did not specify in their paper whether subjects were required to be free of respiratory infections within 3 weeks of exposure, the EPA otherwise believe the investigators' experimental design and protocol were appropriate. The Schelegle et al. (2009) used a randomized, controlled, crossover design in which subjects were exposed, without knowledge of the exposure conditions and in random order, to clean filtered air (FA) and four O_3 concentrations. The FA control provides an unbiased estimate of the effects of the experimental procedures on the outcomes of interest. Comparison of responses following this FA control to those following the O_3 exposures allows for estimation of the effects of O_3 itself while controlling for independent effects such as those raised by commenters. Issues such as the length of time between exposures and/or respiratory infection could add variability to experimental results. However, due to the randomized order of exposures, it is unlikely that across all subjects and exposure conditions in the study that increased variability of the results would affect the overall magnitude of responses. It is more probable that random variability of the results would bias results of the Schelegle et al. (2009) study toward the null, i.e., not finding a statistically significant effect of O_3 on lung function.

(30) *Comment:* A number of commenters contended that the group mean FEV_1 and FVC decrements in the Kim et al. (2011) study were small (1.71% and 1.19%, respectively) across the study population and was far less than the 10-15% FEV_1 decrement that is recognized as a moderate function response in asthmatics and healthy adults, respectively. One commenter stated that at exposure to 0.060 ppm, only three of 59 subjects in the Kim et al. (2011) had FEV_1 decrements greater than 10%. Another commenter suggested that these decrements are consistent with normal variability, are not clinically significant, and that the severity of symptoms was not different between exposed and controls. Similarly, other commenters noted that the average FEV_1 decrement in Adams (2006) was 2.8%, less than the 5% daily variation in FEV_1 experienced by healthy adults (per ATS).

Response: The group mean O_3-induced FEV_1 decrement observed in the study by Kim et al. (2011) was 1.8%. However, as described in more detail in this section above and in section II.A.1.b of the preamble to the final rule, the evidence from controlled human exposure studies to date makes it clear that there is considerable

variability in responses across individuals, and that group mean responses are not representative of more responsive individuals. Therefore, it is important to look beyond group mean responses to the responses of individuals to evaluate the potential impact on more responsive members of the population. Moreover, relying on group mean changes to evaluate lung function responses to O_3 exposures (i.e., as has been done by the commenters noted above) would mask the responses of the most sensitive groups, particularly where the group mean reflects responses solely among the healthy young adults who were the study participants. Based on individual-level data from 150 study volunteers evaluated in controlled human exposure studies that examined exposures to 60 ppb O_3 (including the studies by Kim et al., 2011 and Adams, 2006), the ISA reported that 10% of young healthy adults experienced FEV_1 decrements > 10% following exposures to 60 ppb O_3 (ISA, p. 6-18).

As discussed in this section above, based on ATS guidelines for assessing bronchoconstriction, the ISA states that "[a] 10% FEV_1 decrement is…generally accepted as an abnormal response" (ISA, p. 6-19). In this context, "abnormal" indicates that the decrement is outside the normal range of day-to-day variability. In considering the individual-level data at 60 ppb, the ISA concludes that "[t]hough group mean decrements are biologically small and generally do not attain statistical significance, a considerable fraction of exposed individuals [in the clinical studies] experience clinically meaningful decrements in lung function" when exposed for 6.6 hours to 60 ppb O_3 during quasi-continuous, moderate exertion (ISA, section 6.2.1.1, p. 6-20). The Administrator's consideration of such decrements in the current review are discussed in detail in sections II.B.2.b.i, II.B.3, II.C.4.b, and II.C.4.c of the preamble to the final rule.

(31) *Comment:* Some commenters asserted that the majority of chamber studies have been conducted during winter, or times when ambient ozone exposure levels are lower. They claimed that this creates a potentially unrealistic response scenario in that the lungs are exposed to sudden, rapid changes in ozone concentration. These commenters pointed out that, according to the ISA, during the ozone season, the lungs may mediate concentration effects through acclimatizing to weather and ozone conditions. Accordingly, by conducting studies during times of low ozone concentrations when the lungs have no opportunity to acclimate to the conditions, commenters concluded the studies may be measuring the effect associated with an unrealistic magnitude of change (in factors such as ozone, temperature, or humidity) and not demonstrating clinical responses to a dose-specific level of ozone exposure.

Response: As an initial matter with regard to this comment, we note that the key O_3 controlled human exposure studies held factors other than O_3 concentration constant across the filtered air and the various O_3 exposure conditions. Therefore, if the measured responses were due to sudden changes in temperature, humidity or other factor besides the O_3 concentration, those changes would also be expected to occur during the filtered air exposures. Thus, we do not agree that the O_3-induced responses shown to occur in controlled human exposure studies could

be due to changes in factors other than the O_3 exposure concentration (e.g., factors such as temperature or humidity).

Contrary to these commenters' assertions, attenuation of some responses does not mean that continued O_3 exposures cease to affect the respiratory system. As discussed in the ISA, "inflammation within the airways persists following repeated exposure to O_3" (ISA, p. 6-81) and markers of cellular integrity indicate ongoing tissue damage with repeated O_3 exposures (e.g., see ISA, sections 6.2.3.1 and 6.2.9). The ISA notes that "[t]he continued presence of cellular injury markers indicates a persistent effect that may not necessarily be recognized due to the attenuation of spirometric and symptom responses" (ISA, p. 6-81). That is, attenuation of some responses could actually mask ongoing tissue damage due to repeated O_3 exposures. In addition, the O_3 exposure scenarios evaluated in controlled human exposure studies, and the measured responses, are not unrealistic. While commenters are correct that, over the course of the O_3 season, O_3-induced lung function and symptomatic responses can become attenuated, relying on attenuated responses would not appropriately characterize the potential for O_3-induced effects under a number of common scenarios (e.g., responses early in the O_3 season, when recent O_3 exposures are relatively low; responses in people who travel from a less polluted region to a more polluted region).

(32) *Comment:* Some commenters noted that scientists have conducted most lung impairment human exposure studies at temperatures substantially lower than the temperature at which potentially unhealthy levels of ozone most often occur. These commenters pointed out that CASAC has identified the need to study the interaction between O_3 and temperature to determine whether there might be a mediating effect (or contributing effect). Commenters claim that the Administrator failed to consider this uncertainty in determining the requisite level of protection.

Response: We agree that an important area for future research is the potential for temperature to confound or modify the effects of O_3 exposure. In the PA,the need for this type of research is identified by EPA staff as it pertains to epidemiologic studies. Specifically, the PA states the following (section 4.7, p. 4-71):

> As epidemiologic research has continued to be an important factor in assessing the public health impacts of O_3, methodological issues in epidemiologic studies have received greater visibility and scrutiny. There remains a need to further examine alternative modeling specifications and control of time-varying factors, and to better understand the role of copollutants in the ambient air. Additionally, there remains uncertainty around the role of temperature as a potential confounder or effect modifier in epidemiologic models.

CASAC agreed with EPA staff recommendations regarding the need for future research, stating:

> For the health-based standard, we note that the Second Draft PA outlines key uncertainties and research that needs to be addressed for future reviews of the health-based standards. Specifically, we underscore the need for research to address the characterization of the exposure-response function; the identification of population thresholds; the role of co-pollutants and temperature in modifying or contributing to ozone effects; alternative modeling specifications; population-based information on human exposure for at-risk populations; time-activity data to improve population-based exposure and risk assessment; and the characterization of background levels (Frey, 2014b, p. iv).

To the extent ongoing or future research addresses this issue, the resulting studies will be considered in future reviews of the O_3 NAAQS. In the current review, CASAC clearly was aware of this issue and the need for additional research. Based on the available scientific information, including the uncertainties and limitations in that information, CASAC recommended setting the level of the primary O_3 standard to within the range of 60 to 70 ppb (Frey, 2014b). Therefore, while we agree that an important area for future research is the potential for temperature to confound or modify the effects of O_3 exposure, we do not agree with these commenters that this uncertainty has not been appropriately considered or that a different decision on standard level should be reached because of it.

(33) *Comment:* One commenter states that there is no clear justification for the choice of an FEV_1 decrement of 10% as being adverse. This commenter cites EPA's justification from a study of exercise-induced bronchoconstriction (EIB) (Dryden, 2010) but criticizes the lack of clarity regarding the appropriateness of EIB for establishing an O_3-induced adversity cut-off. TCEQ claims:

> "The EPA states that the 10% cut-off is an appropriate threshold for those with lung disease such as asthma or COPD, but…applies the 10% cut-off to the entire population for expected FEV_1 decrements. The FEV_1 decrement of 10% should not be modeled in healthy children, and by an inappropriate FEV_1 decrement cut-off, the EPA is misleading the readers of this document, as well as the Administrator" (e.g., TCEQ).

Response: As an initial matter, we note that the commenter has incorrectly characterized the Administrator's consideration of O_3-induced FEV_1 decrements $\geq$ 10%. As discussed in the preamble to the final rule, the percentages of all children and children with asthma estimated to experience various O_3-induced lung function decrements were virtually indistinguishable from each other, though the estimated numbers of all children were different from the estimated numbers of children with asthma. Therefore, when discussing percentages of children at risk of O_3 induced decrements $\geq$ 10% (or 15%), the same percentages apply regardless of whether the focus is on all children or on children with asthma. In contrast, in instances where the Administrator considers the numbers of children estimated to be at risk, she focuses specifically on the numbers of children with asthma when discussing decrements $\geq$ 10% (see section II.B.3 of the preamble to the final

rule). This is consistent with CASAC advice in the current review that "an FEV_1 decrement of ≥ 10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease" (Frey, 2014b, p. 3) and its advice in the last review that "people with chronic obstructive pulmonary disease have decreased ventilatory reserve (*i.e.,* decreased baseline FEV_1) such that a ≥10% decrement could lead to moderate to severe respiratory symptoms" (Samet, 2011).

With regard to commenters' more general assertion that there is no justification for characterizing 10% FEV_1 decrements as adverse, we note that, as in past reviews, the consideration of FEV_1 decrements ≥ 10% in the current review is based in part on ATS criteria, as well as on CASAC advice. Based on ATS guidelines for assessing bronchoconstriction, the ISA states that "[a] 10% FEV_1 decrement is…generally accepted as an abnormal response" (ISA, p. 6-19). In this context, "abnormal" indicates that the decrement is outside the normal range of day-to-day variability, and is not meant to indicate that such a response is invariably "adverse." In fact, in its recommendations on adversity, the ATS did not speak specifically to FEV_1 decrements of any particular magnitude, and stated that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" (ATS, 2000, p. 670).

Consistent with this characterization, in the current and past reviews CASAC and the EPA have noted the potential for O_3-induced FEV_1 decrements ≥ 10% to be adverse in people with lung disease, but acknowledged that there is some uncertainty in this judgment. For example, in the 2008 final rule the EPA noted that "in the context of standard setting, CASAC indicated that a focus on the lower end of the range of moderate functional responses (e.g., FEV_1 decrements ≥ 10%) is most appropriate for estimating *potentially adverse lung function decrements in people with lung disease*" (73 FR 16463, March 27, 2008; internal citations omitted, emphasis added). In the 2010 proposal, which proposed to reconsider the 2008 decision on the primary O_3 standard, a different Administrator again noted this same point (75 FR 2993, January 19, 2010). In the current review the Administrator notes the CASAC advice that "an FEV_1 decrement of ≥ 10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease" (e.g., see sections II.B.2.b.i, II.B.3, II.C.4.b, and II.C.4.c of the preamble to the final rule). In the current review, the Administrator further agrees with the judgment made in past reviews (e.g., see 75 FR 2973, January 19, 2010) that a more general consensus view of the potential adversity of such decrements emerges as the frequency of occurrences increases (sections II.B.3 and II.C.4.c of the preamble to the final rule).

Thus, the Administrator considered risk estimates of 10% decrement in FEV_1. As noted in the preamble to the final rule (section II.C.4.c), the Administrator judges that a standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O_3-induced lung function decrements (≥10% as well as 15%) in children, including

children with asthma. Specifically, a revised standard with a level of 70 ppb is estimated to reduce the risk of two or more O_3-induced decrements by about 30% and 20% for decrements ≥ 15 and 10%, respectively (as noted in this section above, estimated percent reductions were the same for all children and for children with asthma).

However, as discussed in the proposal and in the preamble to the final rule, the Administrator judges that estimates of the risk of O_3-induced lung function decrements provide a less certain basis for distinguishing between specific standard levels than exposures of concern, given the considerable overlap between risk estimates for various standard levels. In addition, as discussed in section II.C.4.b.i of the preamble to the final rule, the Administrator judges that there are important uncertainties in using lung function risk estimates as a basis for considering the occurrence of adverse effects in the population given (1) the ATS recommendation that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" (ATS, 2000); (2) uncertainty in the extent to which a transient population-level decrease in FEV_1 would increase the risk of other, more serious respiratory effects in that population (i.e., per ATS recommendations on population-level risk); and (3) that CASAC did not advise EPA to consider a standard that would be estimated to eliminate O_3-induced lung function decrements ≥ 10 or 15% (Frey, 2014b). Moreover, as at proposal, the Administrator notes that the variability in lung function risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels.[30] Given this, and the resulting considerable overlap between the ranges of lung function risk estimates for different standard levels, the Administrator puts limited weight on the lung function risk estimates for distinguishing between the degrees of public health protection provided by alternative standard levels. Therefore, the Administrator judges that while a standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O_3-induced lung function decrements (≥10%, 15%) in children, including children with asthma, she also judges that estimated risks of O_3-induced lung function decrements provide a much more limited basis than exposures of concern for distinguishing between the appropriateness of the health protection afforded by a standard level of 70 ppb versus lower levels.

Thus, as discussed in sections II.B.2, II.B.3, II.C.4.b, and II.C.4.c of the preamble to the final rule, the Administrator judges that her conclusions regarding the potential adversity of O_3-induced FEV_1 decrements ≥ 10% in the current review

[30] For example, the average percentage of children estimated to experience two or more decrements ≥ 10% ranges from approximately 6 to 11% for a standard level of 70 ppb, up to about 9% for a level of 65 ppb, and up to about 6% for a level of 60 ppb (Table 2 in the preamble to the final rule). In contrast, the average percentage of children estimated to experience two or more exposures of concern for the 60 ppb benchmark ranges from approximately 0.5 to 3.5% for a level of 70 ppb, up to 0.8% for a level of 65 ppb, and up to 0.2% for a level of 60 ppb (Table 1 in the preamble to the final rule).

are consistent with the updated scientific evidence, exposure/risk information, and CASAC advice.

ii. Evidence from Epidemiologic Studies

This section discusses key comments on the EPA's assessment of the epidemiologic evidence and provides the Agency's responses to those comments. The focus in this section is on comments related to the EPA's approach to assessing and interpreting the epidemiologic evidence as a whole. Comments addressed in the preamble to the final rule are presented first, followed by detailed comments on specific studies, or specific methodological or technical issues. Many of the issues and concerns raised by commenters on the interpretation of the epidemiologic evidence are essentially restatements of issues raised during the development of the ISA, HREA, and/or PA, and in many instances were considered by CASAC in the development of its advice on the current standard. The EPA's responses to these comments rely heavily on the process established in the ISA for assessing the evidence, and on CASAC advice received as part of this review of the O_3 NAAQS.

(1) *Comment:* As with evidence from controlled human exposure studies, commenters expressed sharply divergent views on the evidence from epidemiologic studies, and on the EPA's interpretation of that evidence. One group of commenters, representing medical, public health and environmental organizations, and some states, generally supported the EPA's interpretation of the epidemiologic evidence with regard to the consistency of associations, the coherence with other lines of evidence, and the support provided by epidemiologic studies for the causality determinations in the ISA. These commenters asserted that the epidemiologic studies evaluated in the ISA provide valuable information supporting the need to revise the level of the current primary O_3 standard in order to increase public health protection. In reaching this conclusion, commenters often cited studies (including a number from the past review) which they interpreted as showing health effect associations in locations with O_3 air quality concentrations below the level of the current standard. In some cases, these commenters also cited PA analyses of air quality in locations of epidemiologic studies. A second group of commenters, mostly representing industry associations, businesses, and states opposed to revising the primary O_3 standard, expressed the general view that while many new epidemiologic studies have been published since the last review of the O_3 NAAQS, inconsistencies and uncertainties inherent in these studies as a whole, and in the EPA's assessment of study results, should preclude any reliance on them as justification for a more stringent primary O_3 standard. To support their views, these commenters often focused on specific technical or methodological issues that contribute to uncertainty in epidemiologic studies, including the potential for exposure error, confounding by copollutants and by other factors (e.g., weather, season, disease, day of week, etc.), and heterogeneity in results across locations. In some cases, commenters highlighted these and other specific limitations in individual epidemiologic studies, and asserted that O_3 epidemiologic studies report only weak health effect associations.

Response: The EPA agrees with certain aspects of each of these views. Specifically, while the EPA agrees that epidemiologic studies are an important part of the broader body of evidence that supports the ISA's causality determinations, and that these studies provide support for the decision to revise the current primary O_3 standard, the Agency also acknowledges that there are important uncertainties and limitations associated with these epidemiologic studies that should be considered when reaching decisions on the current standard.

In particular, the available O_3 epidemiologic studies provided strong support for the ISA's determination that "there is a causal relationship between short-term O_3 exposure and respiratory effects" (ISA, p. 1-6). The ISA specifically concluded the following (ISA, 2013, p. 1-6):

> [F]indings from experimental studies provided support for epidemiologic evidence, in which short-term increases in O_3 concentration were consistently associated with increases in respiratory symptoms and asthma medication use in children with asthma, respiratory-related hospital admissions, and [emergency department] visits for chronic obstructive pulmonary disease (COPD) and asthma. Additionally, recent epidemiologic evidence supports the range of respiratory effects induced by O_3 by demonstrating that short-term increases in ambient O_3 concentrations can lead to respiratory mortality.

These conclusions in the ISA were the product of extensive interactions between the EPA and CASAC at multiple public meetings. At these meetings, there were opportunities for members of the public to express their views on the evidence to CASAC and to the EPA. Many of the issues considered by CASAC and the EPA during the development of the ISA (and the HREA and PA) were issues that have been raised again in public comments on the proposal. Thus, while the EPA agrees that any individual epidemiologic study has limitations (many of which have been highlighted by industry commenters), we conclude that, taken together, these studies provide strong support for the causality determinations in the ISA.

Although O_3 epidemiologic studies show consistent associations between O_3 exposures and serious health effects, including morbidity and mortality, and some of these studies reported such associations with ambient O_3 concentrations below the level of the current standard, the EPA also concludes that important limitations in these studies should be considered with reaching specific decisions on the primary O_3 standard. Uncertainties and limitations in the epidemiologic evidence were considered by the Administrator in the proposal, and contributed to her decision to place less weight on information from epidemiologic studies than on information from controlled human exposure studies when considering the adequacy of the current primary O_3 standard (see 79 FR 75281-83). The Administrator adheres to these conclusions in her final determination, as discussed in the preamble to the final rule (sections II.B.2.b.ii, II.C.4.a.ii, and II.C.4.c).

In doing so, she noted that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following exposures to specific O_3 concentrations. The effects reported in these studies are due solely to O_3 exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in the O_3 epidemiologic studies). She further noted the CASAC judgment that "the scientific evidence supporting the finding that the current standard is inadequate to protect public health is strongest based on the controlled human exposure studies of respiratory effects" (Frey, 2014b, p. 5).

Despite receiving less weight, the EPA does not agree with commenters who asserted that uncertainties in the epidemiologic evidence provide a basis for concluding that the current primary standard does not need revision. As in the proposal, in her final decisions the Administrator specifically considers the extent to which available studies support the occurrence of O_3 health effect associations with air quality likely to be allowed by the current standard, while also considering the implications of important uncertainties, as discussed below.

In considering information from epidemiologic studies within the context of her conclusions on the adequacy of the current standard, the Administrator specifically considers analyses in the PA that evaluate the extent to which O_3 health effect associations have been reported for air quality concentrations likely to be allowed by the current standard. She notes that such analyses can provide insight into the extent to which the current standard would allow the distributions of ambient O_3 concentrations that provided the basis for these health effect associations. While the majority of O_3 epidemiologic studies evaluated in the PA were conducted in areas that would have violated the current standard during study periods, as discussed in the preamble to the final rule (section II.B.2.b.ii), the Administrator observes that the study by Mar and Koenig (2009) reported associations between short-term O_3 concentrations and asthma emergency department visits in children and adults in a U.S. location that would have met the current O_3 standard over the entire study period.[31] Based on this, she notes the conclusion from the PA that the current primary O_3 standard would have allowed the distribution of ambient O_3 concentrations that provided the basis for the statistically significant associations with asthma emergency department visits reported by Mar and Koenig (2009) (PA, section 3.1.4.2).

In addition, even in some single-city study locations where the current standard was violated (i.e., those evaluated in Silverman and Ito, 2010; Strickland et al., 2010), the Administrator notes that PA analyses of reported concentration-response functions and available air quality data support the occurrence of O_3-attributable hospital admissions and emergency department visits on subsets of

[31] The large majority of locations evaluated in U.S. epidemiologic studies of long-term O_3 would have violated the current standard during study periods. Although these studies support the ISA's causality determinations, they provide limited insight into the adequacy of the current standard (PA, section 3.1.4.3).

days with virtually all ambient O_3 concentrations below the level of the current standard. PA analyses of study area air quality further support the conclusion that exposures to the ambient O_3 concentrations present in the locations evaluated by Strickland et al. (2010) and Silverman and Ito (2010) could have plausibly resulted in the respiratory-related emergency department visits and hospital admissions reported in these studies (PA, section 3.1.4.2). The Administrator agrees with the PA conclusion that these analyses indicate a relatively high degree of confidence in reported statistical associations with respiratory health outcomes on days when virtually all monitored 8-hour O_3 concentrations were 75 ppb or below. She further agrees that though these analyses do not identify true design values, the presence of O_3-associated respiratory effects on such days provides insight into the types of health effects that could occur in locations with maximum ambient O_3 concentrations below the level of the current standard.

Compared to the single-city epidemiologic studies discussed above, the Administrator notes additional uncertainty in interpreting the relationships between short-term O_3 air quality in individual study cities and reported O_3 multicity effect estimates. In particular, she judges that the available multicity effect estimates in studies of short-term O_3 do not provide a basis for considering the extent to which O_3 health effect associations persist in individual locations with ambient O_3 concentrations low enough to meet the current O_3 standard, versus locations with O_3 concentrations that violate this standard.[32] While such uncertainties limit the extent to which the Administrator bases her conclusions on air quality in locations of multicity epidemiologic studies, she does note that statistically significant O_3 associations with respiratory morbidity or mortality have been reported in several multicity studies when the substantial majority of study locations (though not all study locations) would likely have met the current O_3 standard (PA, pp. 3-62 and 3-63).

Looking across the body of epidemiologic evidence, the Administrator reaches the conclusion that analyses of air quality in study locations support the occurrence of adverse O_3-associated effects at ambient O_3 concentrations likely to have met the current standard. She further concludes that the strongest support for this conclusion comes from single-city studies of respiratory-related hospital

[32] As noted in the proposal (II.E.4.d), this uncertainty applies specifically to interpreting air quality analyses within the context of multicity effect estimates for short-term O_3 concentrations, where effect estimates for individual study cities are not presented. That is, the health information across the entire study cannot be disaggregated, so even though air quality in each of the study cities is known, there is uncertainty in connecting air quality in each city with the documented, but not dis-aggregable, adverse health effects. This uncertainty exists for all of the key multi-city O_3 studies analyzed in the PA, with the exception of the study by Stieb et al. (2009) where none of the city-specific effect estimates for asthma emergency department visits were statistically significant. This specific uncertainty does not apply to multicity epidemiologic studies of long-term O_3 concentrations, where multicity effect estimates are based on comparisons across cities. For example, see discussion of study by Jerrett et al. (2009) in the PA (section 3.1.4.3).

admissions and emergency department visits associated with short-term O_3 concentrations.

With regard to her decision to revise the level of the primary O_3 standard to 70 ppb, the Administrator notes analyses in the PA (section 4.4.1) indicating that a revised standard with a level of 70 ppb would be expected to require additional reductions, beyond those required by the current standard, in the short- and long-term ambient O_3 concentrations that provided the basis for statistically significant O_3 health effect associations in both the single-city and multicity epidemiologic studies evaluated. As discussed further in the preamble to the final rule, while the Administrator concludes that these analyses support a level at least as low as 70 ppb, based on a study reporting health effect associations in a location that met the current standard over the entire study period but that would have violated a revised standard with a level of 70 ppb,[33] she further judges that they are of much more limited utility for distinguishing between the appropriateness of the health protection estimated for a standard level of 70 ppb and the protection estimated for lower levels. Thus, the Administrator notes that a revised standard with a level of 70 ppb will provide additional public health protection, beyond that provided by the current standard, against the clearly adverse effects reported in epidemiologic studies. She judges that a standard with a level of 70 ppb strikes an appropriate balance between setting the level to require reductions in the ambient O_3 concentrations associated with statistically significant health effects in epidemiologic studies, while not being more protective than necessary in light of her considerable uncertainty in the extent to which studies show O_3-attributable effects at ambient O_3 concentrations lower than 70 ppb.

(2) *Comment:* As part of a larger set of comments criticizing the EPA's interpretation of the evidence from time series epidemiologic studies, some commenters objected to the EPA's reliance on the studies by Strickland et al. (2010), Silverman and Ito (2010), and Mar and Koenig (2009). These commenters highlighted what they considered to be key uncertainties in interpreting these studies, including uncertainties due to the potential for confounding by co-pollutants, aeroallergens, or the presence of upper respiratory infections; and

[33] As discussed in the preamble to the final rule (sections II.B.2 and II.B.3), the study by Mar and Koenig (2009) reported positive and statistically significant associations with respiratory emergency department visits in a location that would have met the current standard over the entire study period, but would have violated a standard with a level of 70 ppb over the entire study period (see PA, 3-62). In addition, air quality analyses in the locations of two additional studies highlighted in sections II.B.2 and II.B.3 (Silverman and Ito, 2010; Strickland et al., 2010) were used in the PA to inform staff conclusions on the adequacy of the current primary O_3 standard. However, the appropriate interpretation of these analyses became less clear for standard levels below 75 ppb. Unlike the situation when virtually every monitor during the study period was less than 75 ppb, increasing numbers of monitors exceeded levels of 70 ppb and lower such that the air quality in question could have violated a standard set at these lower levels (see PA, section 4.4.1; Appendix 3B, Tables 3B-6 and 3B-7). Therefore, these analyses were not used in the PA to inform conclusions on potential alternative standard levels lower than 75 ppb (PA, Chapters 3 and 4). See *Mississippi*, 744 F. 3d at 1352-53 (study appropriate for determining causation may not be probative for determining level of a revised standard).

uncertainties in the interpretation of zero-day lag models (i.e., specifically for Mar and Koenig, 2009).

Response: While the EPA agrees that there are uncertainties associated with interpreting the O_3 epidemiologic evidence, as discussed in the preamble to the final rule, we disagree with commenters' assertion that these uncertainties should preclude the use of the O_3 epidemiologic evidence in general, or the studies by Silverman and Ito (2010), Strickland et al. (2010), or Mar and Koenig (2009) in particular, as part of the basis for the Administrator's decision to revise the current primary standard. As a general point, when considering the potential importance of uncertainties in epidemiologic studies, we rely on the broader body of evidence, not restricted to these three studies, and the ISA conclusions based on this evidence. The evidence, the ISA's interpretation of specific studies, and the use of information from these studies in the HREA and PA, was considered by CASAC in its review of drafts of the ISA, HREA, and PA. Based on the assessment of the evidence in the ISA, and CASAC's endorsement of the ISA conclusions, as well as CASAC's endorsement of the approaches to using and considering information from epidemiologic studies in the HREA and PA, we do not agree with these commenters' conclusions regarding the usefulness of the epidemiologic studies by Strickland et al. (2010), Silverman and Ito (2010), and Mar and Koenig (2009).

More specifically, with regard to confounding by co-pollutants, we note the ISA conclusion that, in studies of O_3-associated hospital admissions and emergency department visits "O_3 effect estimates remained relatively robust upon the inclusion of PM…and gaseous pollutants in two-pollutant models" (ISA, pp. 6-152 and 6-153). This conclusion was supported by several studies that evaluated co-pollutant models including, but not limited to, two of the studies specifically highlighted by commenters (i.e., Silverman and Ito, 2010; Strickland et al., 2010) (ISA, section 6.2.7.5; Figure 6-20 and Table 6-29).

Other potential uncertainties highlighted by commenters have been evaluated less frequently (e.g., confounding by allergen exposure, respiratory infections). However, we note that Strickland et al. (2010) did consider the potential for pollen (a common airborne allergen) to confound the association between ambient O_3 and emergency department visits. While quantitative results were not presented, the authors reported that "estimates for associations between ambient air pollutant concentrations and pediatric asthma emergency department visits were similar regardless of whether pollen concentrations were included in the model as covariates" (Strickland et al., 2010, p. 309). This suggests a limited impact of aeroallergens on O_3 associations with asthma-related emergency department visits and hospital admissions.

With respect to the comment about epidemiologic studies not controlling for respiratory infections in the model, the EPA disagrees with the commenters' assertion. We recognize that asthma is a multi-etiologic disease and that air pollutants, including O_3, represent only one potential agent capable of triggering

an asthma exacerbation. Strickland et al. (2010) attempted to further clarify the relationship between short-term O_3 exposures and asthma emergency department visits by controlling for the possibility that respiratory infections may lead to an asthma exacerbation. By including the daily count of upper respiratory visits as a covariate in the model, Strickland et al. (2010) were able to account for the possibility that respiratory infections contribute to the daily counts of asthma emergency department visits, and to identify the O_3 effect on asthma emergency department visits. In models that controlled for upper respiratory infection visits, associations between O_3 and emergency department visits remained statistically significant (see Table 4 in Strickland et al., 2010), demonstrating a relatively limited influence of respiratory infections on the association observed between short-term O_3 exposures and asthma emergency department visits, contrary to the commenters' claim.

In addition, with regard to the criticism of the results reported by Mar and Koenig (2009), the EPA disagrees with commenters who questioned the appropriateness of a zero-day lag. These commenters specifically noted uncertainty in the relative timing of the O_3 exposure and the emergency department visit when they occurred on the same day. However, based on the broader body of evidence, the ISA concludes that the strongest support is for a relatively immediate respiratory response following O_3 exposures. Specifically, the ISA states that "[t]he collective evidence indicates a rather immediate response within the first few days of O_3 exposure (i.e., for lags days averaged at 0-1, 0-2, and 0-3 days) for hospital admissions and [emergency department] visits for all respiratory outcomes, asthma, and chronic obstructive pulmonary disease in all-year and seasonal analyses" (ISA, p. 2-32). Thus, the use of a zero-day lag is consistent with the broader body of evidence supporting the occurrence of O_3-associated health effects. In addition, while Mar and Koenig reported the strongest associations for zero-day lags, they also reported positive associations for lags ranging from zero to five days (see Table 5 in Mar and Koenig, 2009). In considering this study, the ISA stated that Mar and Koenig (2009) "found consistent positive associations across individual lag days" and that "[f]or children, consistent positive associations were observed across all lags…with the strongest associations observed at lag 0 (33.1% [95% CI: 3.0, 68.5]) and lag 3 (36.8% [95% CI: 6.1, 77.2])" (ISA, p. 6-150). Given support for a relatively immediate response to O_3 and given the generally consistent results in analyses using various lags, we disagree with commenters who asserted that the use of a zero-day lag represents an important uncertainty in the interpretation of the study by Mar and Koenig (2009).

Given all of the above, we do not agree with commenters who asserted that uncertainties in the epidemiologic evidence in general, or in specific key studies, should preclude the Administrator from relying on those studies to inform her decisions on the primary O_3 standard.

(3) *Comment:* Some commenters also objected to the characterization in the ISA and the proposal that the results of epidemiologic studies are consistent. These

commenters contended that the purported consistency of results across epidemiologic studies is the result of inappropriate selectivity on the part of the EPA in focusing on specific studies and specific results within those studies. In particular, several commenters contended that EPA favors studies that show positive associations and selectively ignores certain studies that report null results. They also cite a recent study published after completion of the ISA (Goodman et al., 2013) suggesting that, in papers where the results of more than one statistical model are reported, the EPA tends to report the results with the strongest associations.

Response: The EPA disagrees that it has inappropriately focused on specific positive studies or specific positive results within individual studies. The ISA appropriately builds upon the assessment of the scientific evidence presented in previous AQCDs and ISAs.[34] When evaluating new literature, "[s]election of studies for inclusion in the ISA is based on the general scientific quality of the study, and consideration of the extent to which the study is informative and policy-relevant" (ISA, p. liii). In addition, "the intent of the ISA is to provide a concise review, synthesis, and evaluation of the most policy-relevant science to serve as a scientific foundation for the review of the NAAQS, not extensive summaries of all health, ecological and welfare effects studies for a pollutant" (ISA, p. lv). Therefore, not all studies published since the previous review would be appropriate for inclusion in the ISA.[35] With regard to the specific studies that are included in the ISA, and the analyses focused upon for given studies, the EPA notes that the ISA undergoes extensive peer review in a public setting by the CASAC. This process provides ample opportunity for CASAC and the public to comment on studies not included in the ISA, and on the specific analyses focused upon within individual studies. In endorsing the final O_3 ISA as adequate for rule-making purposes, CASAC agreed with the selection and presentation of analyses on which to base the ISA's key conclusions.

In addition to the comments addressed above and in the preamble to the final rule, EPA received a number of detailed and technical comments on the evidence from epidemiologic studies, including EPA's interpretation of the evidence. These comments are addressed below.

[34] Cf. *Coalition for Responsible Regulation v. EPA*, 684 F. 3d 102, 119 (D.C. Cir. 2012) ("EPA simply did here what it and other decision-makers often must do to make a science-based judgment: it sought out and reviewed existing scientific evidence to determine whether a particular finding was warranted. It makes no difference that much of the scientific evidence in large part consisted of 'syntheses' of individual studies and research. Even individual studies and research papers often synthesize past work in an area and then build upon it. That is how science works").

[35] See also section II.C.4.b in the preamble to the final rule responding to comments from environmental interests that EPA inappropriately omitted many studies which (in their view) support establishing a revised standard at a level of 60 ppb or lower. Although, as explained there, the EPA disagrees with these comments, the comments illustrate that the EPA was even-handed in its consideration of the epidemiologic evidence, and most certainly did not select merely studies favorable to the point of view of revising the current standard.

(4) *Comment:* Some commenters claimed that EPA arbitrarily disregarded panel studies cited in Section 4.4.1 of the PA, which show associations in places where O_3 concentrations never exceeded various levels below 70 ppb.

Response: Contrary to commenters' claims, the EPA has not disregarded the panel studies cited in the PA. In the PA, epidemiologic panel studies are viewed as providing support for the real-world occurrence, in a broader range of populations (including children), of the types of respiratory effects observed in healthy adults in controlled human exposure studies (PA, sections 3.1.4.1 and 4.4.1). However, when considering the types of respiratory effects reported in both controlled human exposure studies and epidemiologic panel studies, the Administrator places the most emphasis on the results of controlled human exposure studies. As discussed in the proposal, controlled human exposure studies provide more certain evidence for the relationship between specific O_3 exposures and respiratory effects. In particular, the proposal notes the following (79 FR 75249, December 17, 2014):

> An advantage of O_3 controlled human exposure studies (*i.e.*, compared to the epidemiologic panel studies discussed below) is that reported effects necessarily result from exposures to O_3 itself. To the extent studies report statistically significant decrements in mean lung function following O_3 exposures after controlling for other factors, these studies provide greater confidence that measured decrements are due to the O_3 exposure itself, rather than to chance alone.

In contrast to controlled human exposure studies, interpretation of epidemiologic panel studies within the context of a decision on the level of the primary O_3 standard is complicated by several factors. For example, the ISA notes that the use of filtered air responses as a control for the assessment of responses following O_3 exposure in controlled human exposure studies serves to eliminate alternative explanations other than O_3 itself in causing the measured responses (ISA, section 6.2.1.1). No such controls are used in epidemiologic panel studies. Therefore, unlike controlled human exposure studies, it is not clear in epidemiologic panel studies the extent to which various characteristics of the O_3 exposure conditions (i.e., exposure concentrations, durations of exposure, degree of activity) could have contributed to the reported effects. In addition, the key O_3 panel studies discussed in the PA, the proposal, and the preamble to the final rule have reported health effect associations with averaging periods across studies ranging from 10 minutes to 12 hours (PA, Table 3-2). Within some individual studies, the averaging periods for the reported O_3 concentration also varied widely across individual study volunteers (PA, Table 3-2). Thus, the fact that some of these panel studies reported health effect associations for O_3 concentrations below 70 ppb (based on ambient O_3 concentrations measured for minutes to hours) does not necessarily indicate the occurrence of adverse effects for air quality distributions that would be allowed by the revised primary O_3 standard. Also, some of the associations reported in epidemiologic panel studies, particularly those based on

the lower ambient O_3 concentrations, were not statistically significant, adding to the uncertainty in interpreting these results.

Therefore, consistent with the assessment of the evidence in the ISA and the consideration of these studies in the PA, when considering the specific O_3 exposures that elicit respiratory effects within the context of the current and alternative standards, the Administrator places the most emphasis on information from controlled human exposure studies (i.e., see sections II.B.2, II.B.3, II.C.4 of the preamble to the final rule) and views panel studies as providing supporting evidence in a broader range of populations. The EPA consequently does not accept commenters' reading of the panel studies that would accord these studies strong weight in determining a level of a revised standard.

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

(5) *Comment:* A number of commenters contended that EPA has not considered publication bias in the presentation of the reported results. Publication bias results in an overestimation of positive associations of O_3 and mortality, as well as inflated risk estimates. Therefore, any analysis performed on the air pollution epidemiologic literature utilizes biased inputs and the results are thus biased. In particular, commenters cited a study by Goodman (2005) that noted that there was more than a factor of three difference between the results of the O_3 meta-analyses and the NMMAPS individual city results, which were not affected by publication bias. Commenters also cited a report of a separate review by a panel of ten air pollution health effect experts that concluded "taken together, the meta-analyses provide evidence of a disturbingly large publication bias and model selection bias" (Rochester Conference Report, 2007). Commenters suggested that EPA should have taken the impact of publication bias into account when evaluating the body of epidemiologic literature to make causal determinations.

Response: Contrary to these commenters' assertion, in the ISA the EPA recognized the potential impact of publication bias on the conclusions that may be drawn from a body of studies, as indicated in the preamble (ISA, p. lix). The potential for publication bias is one reason that the ISA emphasizes multicity epidemiologic studies, which are less prone to such bias, in its assessment of the evidence. Furthermore, EPA has repeatedly drawn 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 2006 O_3 AQCD, 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). They concluded that analyses not subject to this publication bias generally have effect estimates that are reduced in magnitude, but that do not disappear completely. 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 (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. (2005) 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. This comment was addressed in the previous review, and we point again to the fact that the previous CASAC O_3 Panel did not express any concerns about EPA's selection of studies to be included in the O_3 risk assessment. In fact, CASAC's October 2006 letter (Henderson, 2006, 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, 2006, p.12) also explicitly stated that it judged the selection of health outcomes "for inclusion in the quantitative risk assessment to be appropriate."

Many commenters who did not support revising the current O_3 standard also submitted comments on specific methodological issues and limitations related to individual studies and to the epidemiologic evidence as a whole. These issues and limitations included uncertainties related to the exposure surrogates used in epidemiologic studies; the potential for confounding by copollutants or other factors; issues related to model selection; uncertainties in the evidence of mortality or hospital admissions and emergency department visits; and the implications of new studies not included in the ISA. The comments on methodological issues raised by these commenters are discussed below.

(6) *Comment:* Some commenters 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 epidemiologic studies on which EPA relies in recommending a more stringent O_3 standard are based on data from ambient monitors for which there is 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 epidemiologic studies. For example, 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. Others also cited a study (Meng et al. 2005) that found biases in personal versus ambient exposures as a result of pollutant infiltration behavior. Some commenters also suggest that the

assumption that exposure to O_3 is relatively constant potentially overestimates an individual's true exposure to an extreme degree. Two sources of overestimation errors highlighted by these commenters included time spent indoors versus outdoors and exposure consequences of avoidance or aversion behavior. One commenter suggests that the average American adult, senior citizen, and child will only spend 5.3%, 5.8%, and 7.9% of their time outdoors, respectively, and therefore will not often be exposed to O_3. Industry commenters also contended that EPA did not thoroughly consider exposure measurement error in its evaluation of the epidemiologic evidence. Commenters noted that, in the previous review, 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. CASAC further questioned the likelihood of O_3 itself causing mortality and noted the limitation that measurement error obscures thresholds in time-series studies. Commenters cited a study (Rhomberg et al., 2011) that has shown that measurement error can give a false linear result. The AAM also suggested that the CASAC concerns and the Rhomberg et al. (2011) findings were consistent with several points made by the Special Panel of the HEI Review Committee (Special Panel of the Health Review Committee, 2004) that raised cautions in the interpretation of the NMMAPS concentration-response result, particularly that city-specific concentration-response curves exhibited a variety of shapes, that measurement error could obscure any threshold that might exist, and that the use of Akaike Information Criterion may not be an appropriate criterion for choosing between models.

Response: With regard to the views on exposure measurement error expressed by CASAC in the previous review, while the commenters are correct that the CASAC Panel raised the question of exposure error and whether observed associations could be attributed to O_3 alone, the commenters 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 2006, pp. 3-4)

This section of the quote, which was not addressed in the comment submitted by industry commenters, together with the conclusions in the final CASAC letter from the last review (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.

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 4.6 of the ISA. 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 the current and previous reviews, EPA has recognized the concern surrounding exposure measurement error. The ISA states (p. 4-14) that that study results "generally indicate that personal exposures are moderately well correlated with ambient concentrations, and that the ratio of personal exposure to ambient concentration is higher in outdoor microenvironments and during the summer season." The 2006 O_3 AQCD 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, 2006, p.10). Of concern in interpreting results from mortality and hospitalization time-series studies is the extent to which the ambient O_3 concentrations are representative of personal O_3 exposures in a particularly susceptible group of individuals, the debilitated elderly, as the correlation between the two measurements have not been examined in this population. However, to the extent that relative changes in central-site monitor concentration are associated with relative changes in exposure concentration, ambient monitor concentrations are representative of day-to-day changes in average total personal exposure and in personal exposure to ambient O_3 (ISA, pp. 4-64).

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, characterized in the ISA, conducted in Steubenville, OH (Sarnat et al., 2006), also observed statistically significant associations between ambient O_3 concentrations and personal O_3 exposures. 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, 2006) included senior citizens, the study selection criteria required them to be nonsmoking and physically healthy.

As initially discussed in the 2006 AQCD and updated in section 4.3.3 of the 2013 ISA, 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. Moreover, results from studies examining relationships between measured ambient O_3 concentrations from fixed monitoring sites and personal O_3 exposure (Avol et al., 1998; Brauer and Brook, 1995, 1997; Chang et al., 2000; Delfino et al., 1996; Lee et al., 2004; Liard et al., 1999; Linn et al., 1996; Liu et al., 1995, 1997; O'Neill et al., 2003; Sarnat et al., 2001) indicate that the relationship between ambient O_3 concentrations and personal exposure will vary depending on individual- or city-specific factors such as time activity patterns, indoor air exchange rates, and housing conditions, creating potential measurement errors. Therefore, with respect to the concentration-response analyses detailed in NMMAPS, EPA recognizes that all of the aforementioned factors that can contribute to exposure differences between cities can also potentially influence the observed city-specific concentration-response relationships. Additionally, it is highly likely that other modifiers in addition to exposure measurement error that vary regionally (e.g., temperature) may influence the shape of the concentration-response curve (ISA, p. 6-257). In light of these potential limitations of concentration-response analyses, EPA concluded that "the studies evaluated support a linear O_3-mortality C-R relationship and continue to support the conclusions from the 2006 O_3 AQCD, which stated that "if a population threshold level exists in O_3 health effects, it is likely near the lower limit of ambient O_3 concentrations in the United States" (ISA, p. 6-257; 2006 AQCD).

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 section 3.9 of the 2006 AQCD), 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 relatively 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 (2006 AQCD, pp.7-8 to 7-10). Nonetheless, as noted in section II.A.2.b of the preamble to the final rule, 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 2985-2988).

(7) *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 epidemiologic studies of O_3 and various health outcomes and that the health effects of O_3 are often not statistically significant when epidemiologic studies consider the effects of confounding air pollutants (e.g., $PM_{2.5}$, CO, nitrogen dioxide (NO_2)) in multi-pollutant models. One commenter cited the NMMAPS study by Dominici et al. (2003) that showed that in single pollutant models O_3 had a significantly positive association with mortality. However, when models with two or more criteria pollutants were used, the single pollutant coefficients were attenuated and, in most cases, lost statistical significance. This commenter also cited a study by Smith et al. (2009) that found evidence of confounding in their study and conclude that the appearance of an association between low O_3 concentrations and mortality may be due to the effect of copollutants. Moreover, this commenter pointed out multiple examples of PM confounding in the APHENA study (Katsouyanni et al. 2009) for O_3 and mortality associations in the U.S., Canada, and Europe. When PM was added to the models, the associations between O_3 and mortality from all-cause, cardiovascular, or respiratory effects were either attenuated or lost statistical significance.

Response: The EPA disagrees with these commenters and points to the ISA, specifically Chapter 6, to demonstrate the thorough evaluation of issues related to confounding. It is important to make a distinction, which some commenters have confused, with respect to EPA's position on interpreting the results from epidemiologic studies that use multipollutant models (i.e., models including 3 or more pollutants) versus copollutant models (i.e., models including 2 pollutants). EPA focuses the evaluation of potential confounding of the O_3 associations by other pollutants through an assessment of copollutant models because of the difficulty encountered in interpreting multipollutant results due to the multicollinearity often observed between pollutants.

There are two issues the commenters are addressing within the context of this comment: (1) the lack of statistical significance in the O_3 association in copollutant models; and (2) whether or not the copollutant models suggest

evidence of confounding. With respect to first issue, it is important to recognize that statistical significance is an indicator of the precision of a study's results, which is influenced by the size of the study, as well as by exposure and measurement error. It is important not to focus only 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 report by the U.S. Surgeon General on the health consequences of smoking (CDC, 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, 2004, 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. However, in the broader evaluation of the evidence from many epidemiologic studies, EPA focuses on the *pattern* of results for drawing conclusions on the relationship between air pollutants and health outcomes, as well as consideration of the integration of observational epidemiologic evidence with findings of experimental laboratory studies. This is not only permissible, but appropriate.[36]

With respect to point (2) on the assessment of confounding in the epidemiologic literature evaluated in the ISA, 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 copollutant regression models is the prevailing approach for controlling potential confounding by copollutants in O_3 health effects studies as has been detailed in previous assessments (2006 AQCD, p.7-24) and in the ISA (section 4.3.4).

The commenters specifically highlight a number of studies that evaluate the relationship between short-term O_3 exposures and mortality. Section 6.6.2.1 of the ISA specifically focuses on the evaluation of potential confounders of the O_3-mortality relationship, including some of the studies the commenters cite. As detailed in the ISA (p. 6-224 to 225), it is important to consider when evaluating potential confounding specifically by PM or PM components, the temporal correlation among PM components and O_3, and their possible interactions. These relationships contribute to the challenge in interpreting the results from copollutant models that attempt to disentangle the health effects associated with each pollutant. Further complicating the interpretation of copollutant results, at times, is the every-3rd or -6th day PM sampling schedule employed in most locations, which limits the number of days where both PM and O_3 data is available, and subsequently the uncertainty estimate surrounding any copollutant model result. The ISA recognizes both of these issues, but still concludes that "across studies, the potential impact of PM indices on O_3-mortality risk estimates tended to be much smaller than the variation in O_3-mortality risk estimates across cities suggesting that O_3 effects are independent of the relationship between PM and mortality" (ISA, p. 6-262).[37]

[36] See. e.g. *American Farm Bureau v. EPA*, 559 F. 2d 512, 525 (EPA erred in rejecting consideration of epidemiologic study on grounds of statistical significance where results of that study were consistent with results of other studies).

[37] See also CASAC's conclusion in Samet (2011) p. 10: "While it may be difficult to disentangle the effect of a single pollutant in epidemiological studies, the evidence regarding ozone-related health effects from epidemiological studies is consistent with the evidence from controlled exposure studies that involve ozone alone." CASAC also stated that "[o]ur confidence that the effects from epidemiological studies are

The commenters point to Dominici et al. (2003) to support their claim of copollutant confounding of the O_3-mortality relationship; however, a recent study evaluated in the 2013 O_3 ISA (Bell et al., 2007) also using the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) data provides additional evidence supporting that the O_3-mortality association is robust to the inclusion of other pollutants in the models (ISA, section 6.6.2.1). Bell et al. (2007) focused exclusively on examining whether there was evidence of potential confounding of the association between short-term O_3 exposure and mortality by PM. As detailed above, the sampling schedule for PM played a role in the amount of data available for the analysis focusing on $PM_{2.5}$, only 9.2% of days and 62 out of 91 communities measuring $PM_{2.5}$ could be used for the analysis. In an analysis that examined for evidence of confounding by either PM_{10} or $PM_{2.5}$ along a range of O_3 concentrations (i.e., <10 ppb, 10-20, 20-40, 40-60, 60-80, and >80 ppb), O_3 associations remained robust when including either PM size fraction in the model. Additionally, even with the small number of days in which both O_3 and $PM_{2.5}$ data were available, "the percent increases in nonaccidental deaths per 10 ppb increase 24-h avg O_3 concentrations at lag 0-1 day were 0.22% (95% CI: -0.22, 0.65) without $PM_{2.5}$ and 0.21% (95% CI: -0.22, 0.64) with $PM_{2.5}$ in 62 communities (p. 6-225)," which further supports the overall conclusions of the ISA with respect to copollutant confounding.

The commenters also cite a study conducted by Smith et al. (2009), which further examined the potential confounding effects of PM on the O_3-mortality relationship using NMMAPS data for 98 U.S. cities for the years 1987-2000 (ISA, section 6.6.2.1). This study is similar to the dataset used by Bell et al. (2004). In copollutant analyses, Smith et al. (2009) observed an approximate 22-33% reduction in O_3 mortality risk estimates. It is also important to point out that Smith et al. (2009) question the interpretability of these results due to the 6-day sampling of PM_{10}, which reduced the overall quantity of data available for the analysis. However, unlike Bell et al. (2004), the air quality data used in this analysis was not detrended and trimmed means were not used; therefore, it is unclear how similar the underlying data is between the two studies. Overall, the potential bias in O_3 mortality risk estimates in Smith et al. (2009) due to PM is much smaller than the observed city-to-city variation in risk estimates.

Finally, the commenters cite a study conducted by Katsouyanni et al. (2009), the Air Pollution and Health: A European and North American Approach (APHENA) study (ISA, section 6.6.2.1). The findings of APHENA generally confirm those presented throughout the ISA, but again are limited by the primarily every-6^{th}-day PM_{10} sampling schedule in both the U.S. and Canadian datasets. In this study the authors do not specify which spline model results to focus on, but based off previous time-series analyses alternative spline models result in relatively similar effect estimates (HEI, 2003). Additionally, the authors do not specify the extent of smoothing deemed to be most appropriate when examining the study results.

attributable to ozone is also bolstered by the recognition that the endpoints of concern do not change at the lower levels of the proposed range" (Samet, 2011, p.10).

However, through an evaluation of the results across each of the three datasets by EPA, the inconsistency in effect estimates within the same lag period for different spline models suggests that the 3 df/yr and PACF models do not adequately control for seasonal trends. Therefore, when focusing on either the 8 or 12 df/yr results, in copollutant models the O_3 mortality effect estimates remain relatively robust to the inclusion of PM_{10} in datasets that use both every-day and every-6th day PM_{10} data.[38]

(8) *Comment:* Commenters also pointed to other confounding factors that may influence the air pollution epidemiology, including weather, temporal effects such as season, cyclic diseases, and day-of-the-week patterns. Commenters also identified subject characteristics, such as socioeconomic status, that are included in models as proxy measures for unmeasured confounders, such as access to health care or overall health status, and can therefore contribute an additional source of uncertainty or error. Errors in confounder measurement can affect associations between O_3 and health outcomes in either a positive or negative direction. Commenters noted that associations with short-term O_3 are less susceptible to time-invariant subject characteristics, such as smoking history, but are vulnerable to factors that co-vary in time with O_3 exposure and health, including meteorology, copollutants, pollen and other aeroallergens, and respiratory infections. One commenter argued that in cases of childhood asthma, the epidemiologic studies fail to adequately examine indoor allergens, such as dust mites, pet dander, the presence of cockroaches and rodents, combined with other allergens such as pollen and mold. Moreover, genetic predisposition, exposure to tobacco smoke, cold air, physical exercise, and excess weight all remain critical initiation factors confounding any examination of asthma rates in children and adults. Additionally, the commenter suggested that while some attention was given to socioeconomic status, geographic location, and other simplistic indicators of overall health quality as confounding factors, the studies did not sufficiently examine the details to warrant a reduction in the current standard. One commenter contended that EPA dismissed the possibility of confounding by pollen in the Proposed Rule by stating that studies adjusted for pollen generally showed that results are not confounded. However, the commenter argued that relatively few studies of respiratory morbidity and mortality account for pollen or other aeroallergens and the few that did generally indicated that associations between respiratory effects and pollen are stronger than those with O_3 and that associations with O_3 are attenuated following adjustment for pollen. Commenters suggested that EPA should have fully accounted for these factors in its evaluation of the epidemiologic evidence, especially considering that biases such as confounding can have effects that as large as, if not larger than, true associations between air pollutants and health.

Response: Contrary to the commenters' contention, EPA thoroughly reviewed the issues related to confounding and the evidence of potential confounding in the

[38] See also ISA at 6-162, discussing how "co-pollutant-adjusted findings across respiratory endpoints provide support for the independent effects of short-term exposures to ambient O_3".

ISA. A number of the factors listed by the commenters would not appropriately be identified as potential confounders for a relationship between O_3 and health outcomes. 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. Moreover, it is unlikely that house dust mite and cockroach allergen concentrations in a home would be correlated with ambient O_3 concentrations since ambient O_3 does not readily infiltrate into the indoor environment. Thus, these factors may be independently associated with exacerbation of asthma, but would not be confounders in a relationship between O_3 and asthma exacerbation.

The commenters also observed that control for meteorological variables is important. EPA agrees, and carefully evaluated the potential for confounding by temperature and humidity, as discussed in section 6.6.2.1 of the ISA (pp. 6-224 to 6-234). EPA concluded that O_3 effect estimates were generally more sensitive to alternative weather models than to varying degrees of freedom for temporal trend adjustment. However, more recently a study conducted by Katsouyanni et al. (2009) extensively examined a variety of approaches to adjust for seasonal trends when examining the association between short-term O_3 exposures and mortality and hospital admissions. 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. EPA noted in the ISA that 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 important 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 O_3 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.

(9) *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 time-series epidemiologic 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, (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; (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; and (4) model specification and citing Katsouyanni et al. (2009).

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 up to a factor of two in the effect estimates. However, as noted in the 2006 AQCD, 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 (2006 AQCD, 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 2006 AQCD 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. (2006 AQCD, p.7-174)

Second, the issues surrounding sensitivity to model specifications were thoroughly discussed in the 2006 AQCD (see section 7.1.3.6), evaluated in some of the meta-analyses reviewed in the 2006 AQCD and 2007 Staff Paper, and detailed in Section 6.6.2.1 of the ISA. As stated in the 2006 AQCD, O_3 effect estimates "were generally more sensitive to alternative weather models than to varying degrees of freedom for temporal trend adjustment" (2006 AQCD, p.7-176). However, more recently a study conducted by Katsouyanni et al. (2007) extensively examined a variety of approaches to adjust for seasonal trends when examining the association between short-term O_3 exposures and mortality and hospital admissions. In this analysis, the authors demonstrated that inadequate control of seasonal trends can dramatically affect the O_3 risk estimate. Specifically, "the results [of Katsouyani et al. 2007) show that the methods used to combine single-city estimates did not influence the overall results, and that neither 3 df/year nor choosing the df/year by minimizing the sum of absolute values of PACF of regression residuals was sufficient to adjust for the seasonal negative relationship between O_3 and mortality" (p. 6-234). With respect to copollutant models, the 2006 AQCD 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" (2006 AQCD, p.7-177), which is further supported by the more recent studies evaluated in the ISA. Overall, EPA's integrated assessment demonstrates that 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 O_3-related mortality.

The EPA 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 considering the effect estimates from the time-series studies as providing supporting information, keeping in mind the uncertainties and limitations associated with these studies, in reaching her judgment about the need to revise the current 8-hour O_3 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 2006 AQCD evaluated the strengths and weaknesses of such methods in the context of air pollution epidemiology. The 2006 AQCD 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.

Fourth, Katsouyanni et al. (2009) conducted an extensive sensitivity analysis of data from the large multi-city studies conducted in the U.S. (NMMAPS), Europe (APHEA-2), and Canada with the goal of "develop[ing] more reliable estimates of the potential acute effects of air pollution on human health [and] provid[ing] a common basis for [the] comparison of risks across geographic areas". As detailed in Section 6.6 of the ISA, in this analysis the authors examined the robustness of O_3-mortality risk estimates to different model specifications (i.e., penalized spline and natural spline modes) and methods of smoothing to adjust for temporal trends (i.e., 3 df/yr, 8 df/yr, 12 df/yr and degrees of freedom selected using the absolute sum of the residuals of the partial autocorrelation function [PACF]). In the study the authors do not specify the extent of smoothing deemed to be most appropriate, but based off previous analyses, alternative spline models result in relatively similar effect estimates (HEI, 2003). As detailed in the ISA, Table 6-46 presents the results of the degrees of freedom analysis using alternative methods to calculate a combined estimate: the Berkey et al. (1998) meta-regression and the two-level normal independent sampling estimation (TLNISE) hierarchical method. The results show that the methods used to combine single-city estimates did not influence the overall results, and that neither 3 df/year nor choosing the df/year by minimizing the sum of absolute values of PACF of regression residuals was sufficient to adjust for the seasonal negative relationship between O_3 and

mortality. However, it should be noted, the majority of studies in the literature that examined the mortality effects of short-term O_3 exposure, particularly the multicity studies, used 7 or 8 df/year to adjust for seasonal trends, and in both methods a positive association was observed between O_3 exposure and mortality" (p. 6-234).

(10) *Comment:* Commenters contended that EPA favors studies that show positive associations and selectively ignores certain studies that report nulls results. They also cited a recent study that suggested that, in papers where the results of more than one statistical model are reported EPA tends to report the results with the strongest associations (Goodman et al., 2013). From this study, the commenters point to inconsistencies in the consideration for inclusion of studies and they quote,

For example, in the ISA, EPA states, "[l]iterature searches have been conducted routinely since then to identify studies published since the last review, focusing on studies published from 2005 (closing date for the previous scientific assessment) through July 2011." EPA included the study by Zanobetti & Schwartz (2011) in the ozone ISA but omitted a study by Lipsett et al. (2011) that was published online the same day (23 June 2011). EPA also omitted a study by Spencer-Hwang et al. (2011), which was published online on 21 July 2011. In addition, there were several studies of both ozone and PM that were not included in the ozone ISA but played a prominent role in EPA's PM evaluation (e.g., Jerrett et al. 2005; Miller et al. 2007). This indicates that not all relevant studies were captured by the literature search strategy (e.g., AAM).

Commenters pointed to the Goodman et al. (2013) claim that EPA emphasized studies with positive associations over studies with null associations, rather than emphasizing studies of greater quality over those of less quality. They also state that different statistical model formulations used in the APHENA study produced different results that would not have supported a causal relationship, had the outcomes been viewed collectively. Commenters also suggested that EPA ignored limitations of the epidemiology that might mean that O_3 is not a causal factor.

Response: EPA disagrees with the commenters on the approach used by EPA in the presentation of results and the types of studies included within the ISA. It is important to recognize that the ISA builds off the scientific evidence presented in previous AQCDs and ISAs. When evaluating new literature, "[s]election of studies for inclusion in the ISA is based on the general scientific quality of the study, and consideration of the extent to which the study is informative and policy-relevant" (ISA, p. liii). Additionally, "the intent of the ISA is to provide a concise review, synthesis, and evaluation of the most policy-relevant science to serve as a scientific foundation for the review of the NAAQS, not extensive summaries of all health, ecological and welfare effects studies for a pollutant" (ISA, p. lv); therefore, not all studies published since the previous review would be discussed within an ISA. It is important to note that the ISA undergoes extensive peer review in a public setting by the Clean Air Scientific Advisory

Committee (CASAC). This process provides ample opportunity for the public to comment on studies not included within the ISA during different stages of ISA development. If EPA missed key studies there was ample opportunity for both CASAC and the public to comment and recommend the inclusion of specific studies into the ISA. In the end, CASAC endorsed the final O_3 ISA as adequate for rule-making purposes without the inclusion of the studies mentioned in Goodman et al. (2013).

Furthermore, EPA has accurately characterized the inconsistencies and uncertainties in the epidemiologic 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 ISA has been favorably 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 in the ISA. In general, EPA recognizes that in the body of epidemiologic 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.

EPA also disagrees with the commenters that studies included in the PM ISA were excluded from the O_3 ISA as detailed in Goodman et al. (2013). In reference to Miller et al. (2007), this study only focused on PM and did not include an analysis for O_3, while Jerrett et al. (2005) did not present quantitative results for O_3, making it difficult to fully evaluate the study in the context of exposure to O_3, resulting in its exclusion from the ISA.

Additionally, EPA disagrees with the commenter on the interpretation of results from the APHENA study. In APHENA, Katsouyanni et al. (2009) conducted an extensive sensitivity analysis of data from the large multi-city studies conducted in the U.S. (NMMAPS), Europe (APHEA-2), and Canada with the goal of "develop[ing] more reliable estimates of the potential acute effects of air pollution on human health [and] provid[ing] a common basis for [the] comparison of risks across geographic areas." As detailed in Section 6.6 of the ISA, in this analysis the authors examined the robustness of O_3-mortality risk estimates to different model specifications (i.e., penalized spline and natural spline modes) and methods of smoothing to adjust for temporal trends (i.e., 3 df/yr, 8 df/yr, 12 df/yr and degrees of freedom selected using the absolute sum of the residuals of the partial autocorrelation function [PACF]). In the study the authors do not specify the extent of smoothing deemed to be most appropriate, but based off previous analyses, alternative spline models result in relatively similar effect estimates (HEI, 2003). As detailed in the ISA, Table 6-46 presents the results of the degrees of freedom analysis using alternative methods to calculate a combined estimate: the Berkey et al. (1998) meta-regression and the two-level normal independent sampling estimation (TLNISE) hierarchical method. The results show that the methods used to combine single-city estimates did not influence the overall results, and that neither 3 df/year nor choosing the df/year by minimizing the sum

of absolute values of PACF of regression residuals was sufficient to adjust for the seasonal negative relationship between O_3 and mortality. However, it should be noted, the majority of studies in the literature that examined the mortality effects of short-term O_3 exposure, particularly the multicity studies, used 7 or 8 df/year to adjust for seasonal trends, and in both methods a positive association was observed between O_3 exposure and mortality" (p. 6-234).

(11) *Comment:* Some commenters contended that the EPA failed to explain why asthma rates in the United States have been increasing at the same time that there have been significant decreases in O_3 concentrations in most areas of the United States. These commenters claimed that this fact alone should require a higher degree of scrutiny of studies purporting to show a link between asthma incidence and ozone levels. Some commenters pointed to areas with relatively low O_3 concentrations that have relatively high incidence rates for asthma, and vice versa. These commenters also pointed out strong correlations between poverty and asthma rates.

Response: A large number of studies conducted over a period of decades have examined the potential linkages between O_3 exposures and asthma-related health outcomes. Of these studies, the strongest evidence indicates that short-term (e.g., hours to days) O_3 exposures can trigger adverse respiratory effects in people who already have asthma (ISA, pp. 1-4 to 1-7 and Chapter 6). In some cases, these respiratory effects are severe enough to result in emergency room visits and/or hospital admissions (ISA, section 6.2).

In addition, some studies have reported associations between long-term exposures to ambient O_3 and respiratory outcomes, including the development of asthma. Asthma is a complex disease and there are a number of genetic, lifestyle, and environmental factors that are likely to contribute to its development. Thus, while some studies indicate that long-term O_3 exposures can contribute to the development of asthma, these studies do not indicate that O_3 is the only, or even the predominant, factor in the development of the disease. The ISA assesses the evidence for O_3 exposures and asthma development within the context of other respiratory effects that have also been linked with long-term O_3 exposures. The ISA concludes that "[t]aken together, the recent epidemiologic studies of respiratory health effects (including symptoms, new-onset asthma and mortality) combined with toxicological studies in rodents and nonhuman primates, provide biologically plausible evidence that there is likely to be a causal relationship between long-term exposure to O_3 and respiratory effects" (ISA, pp. 1-6 to 1-7). Thus, while we agree that O_3 exposure is not the only factor that contributes to the development of asthma, or even the most important factor, the available evidence does provide support for a link between long-term O_3 exposures and asthma development. Therefore, given the multiple potential etiologies for asthma, it is

overly simplistic to assume that asthma rates will fall in direct correspondence with reduced ambient levels of O_3.[39]

(12) *Comment:* Some commenters suggested that there is a disconnect between the controlled human exposure studies and the epidemiologic studies. Some commenters stated that the human clinical studies demonstrate that the first O_3 effects are mild and transient and occur above a threshold dose as a result of the protective effects of antioxidants in the epithelial lining fluid. Commenters contended that the dose approaches effects that may be considered adverse only at concentrations of O_3 above the current standard and with vigorous exercise. They contended that EPA assumes that O_3 causes premature mortality and hospital admissions down to zero O_3 levels, which is not consistent with the general principles of toxicology or the specific findings of the controlled human exposure studies.

Response: We do not agree that there is a disconnect between controlled human exposure and epidemiologic studies. Controlled human exposure studies have generally evaluated healthy adults. If the effects observed in these studies are experienced by members of at-risk populations (e.g., children, people with asthma), they could become serious enough to result in the types of outcomes reported in epidemiologic studies (e.g., emergency department visits, hospital admissions). The ISA assesses evidence from across disciplines, including controlled human exposure, epidemiologic, and toxicology studies, to support its causal determinations.[40] The ISA specifically states the following (ISA, p. 2-17):

> Together, the evidence integrated across controlled human exposure, epidemiologic, and toxicological studies and across the spectrum of respiratory health endpoints continues to demonstrate that there is a causal relationship between short-term O_3 exposure and respiratory health effects.

In addition, PA analyses of epidemiologic study area air quality, and comparison of air quality to O_3 exposure concentrations in controlled human exposure studies, are "consistent with the occurrence of O_3-attributable respiratory hospital admissions, even when virtually all monitored concentrations were below the level of the current standard" (PA, p. 3-71).

With regard to risk estimates, commenters are correct that the HREA estimated O_3-associated health risks for the full distribution of ambient O_3 concentrations (i.e., down to zero O_3 as indicated by some commenters). However, these

[39] Cf. *ATA III*, 283 F. 3d at 380 (rejecting argument that secondary standard for ozone is unlawful because other factors such as temperature and pests cause more damage to crop yield than exposure to ozone, and stating "[t]he Clean Air Act direct ERPA to protect public welfare from adverse effects of ozone and other pollutants; the Agency cannot escape that directive because ozone wreaks less havoc than temperature, rainfall, and pests").

[40] Comments regarding the adversity of effects observed in controlled human exposure studies are addressed in sections II.B.2 and II.C.4 of the preamble to the final rule.

commenters fail to acknowledge that, due in large part to the types of uncertainties that they raise, in giving some consideration to risk estimates the Administrator focuses on the risks associated with O_3 concentrations in the upper portions of ambient distributions (e.g., see preamble to the final rule, sections II.B.2.b.iii and II.B.3). In doing so, she notes the increasing uncertainty associated with the shapes of concentration-response curves for O_3 concentrations in the lower portions of ambient distributions and the evidence from controlled human exposure studies, which provide the strongest support for O_3-induced effects following exposures to O_3 concentrations corresponding to the upper portions of typical ambient distributions (i.e., 60 ppb and above). Thus, consistent with the concerns raised by these commenters, the Administrator's consideration of epidemiologic-based risk estimates reflects her increasing uncertainty in the occurrence of O_3-attributable effects at relatively low ambient O_3 concentrations.

The EPA also carefully discussed whether, how, and when extracellular lining fluid (ELF) can quench or mitigate O_3 effects. In short, ELF has attenuative properties, but can be overwhelmed (PA, p. 3-3; preamble to the final rule, section II.A.1.a). The mechanism by which O_3 can cause effects accounts for both the attenuative potential, that it is finite, and that it can be overwhelmed by exposure to elevated concentrations of O_3. Secondary oxidation products formed as a result of O_3 exposure initiate numerous responses at the cellular, tissue and whole organ level of the respiratory system (PA, pp. 3-3 to 3-6 and Fig. 3-1).

(13) *Comment:* A number of commenters pointed to difficulties interpreting the results of multi-city studies in light of substantial between-city heterogeneity. Although commenters recognized the transition from single-city studies to multi-city studies to address disadvantages of single-city studies, these commenters argued that multi-city and multi-continent studies are also limited in validity. They suggested that systematic reviews of single-city studies demonstrate that a substantial amount of between-city heterogeneity exists and that attempts to explain this variability in terms of regional characteristics (e.g., climate or population attributes) have been unsuccessful. Others specifically highlighted differences in relationships observed in Canada versus the relationships observed in U.S. and European cities in the APHENA study. Some commenters claimed that while regional heterogeneity was recognized in the proposal, EPA failed to explain how regional heterogeneity impacts the conclusions that can be drawn from these studies.

Response: EPA agrees that epidemiologic studies provide evidence of regional and city-to-city heterogeneity in O_3 risk estimates. However, EPA disagrees with the assertions of the commenters that multi-city and multi-continent studies are flawed and that systematic reviews of single-city studies should be considered. Within the ISA the focus tends to be on multicity studies in an attempt to reduce the potential for publication bias (ISA, p. lix), which has been demonstrated in single-city studies (Bell et al. 2005). Additionally, a comparison of single-city studies that have all been conducted using different exposure assignment approaches, study populations, time periods, and statistical methodologies further

complicates the ability to adequately assess heterogeneity of O_3-risk estimates. The ISA recognizes that multi-city studies provide evidence of heterogeneity in O_3-risk estimates and this is detailed in section 2.5.4.5, as well as in sections of the ISA focusing on specific health categories (e.g., short-term O_3 exposure and mortality, and respiratory-related hospital admissions). These studies have identified a variety of factors that may modify the O_3-mortality or –respiratory hospital admission relationship, but overall "studies have not consistently identified specific community characteristics that explain the observed heterogeneity" (ISA, pp. 2-34 - 2-35).

With respect to APHENA, EPA disagrees with the commenter's contention that the results from Canada are implausible due to EPA recognizing within section 6.2.7 of the ISA that by standardizing risk estimates from the Canadian dataset to a 40-ppb increase in 1-h max O_3 concentrations misrepresents the magnitude, not direction, of the risk estimate. Specifically, on p. 6-136, EPA states: "Because O_3 concentrations across the cities included in the Canadian dataset are low (median concentrations ranging from 6.7-8.3 ppb (Table 6-26)), the standardized increment of 40 ppb for a 1-h max increase in O_3 concentrations represents an unrealistic increase in O_3 concentrations in Canada and increases the magnitude, not direction, of the observed risk estimate. As a result, calculating the O_3 risk estimate using the 40 ppb increment does not accurately reflect the observed risk of O_3-related respiratory hospital admissions. Although this increment adequately characterizes the distribution of 1-h max O_3 concentrations across the U.S. and European datasets, it misrepresents the observed O_3 concentrations in the Canadian dataset. As a result in summary figures, for comparability, effect estimates from the Canadian dataset are presented for both a 5.1 ppb increase in 1-h max O_3 concentrations (i.e., an approximate interquartile range [IQR] increase in O_3 concentrations across the Canadian cities) as well as the 40 ppb increment used throughout the ISA."

Additionally, in section 6.6, when discussing the mortality results from the APHENA study, the ISA presented results for both an IQR increase in O_3 concentrations within Canada as well as using the standardized 40-ppb for 1-h max increase in O_3 concentrations.

We further disagree with the comment that the final notice fails to explain how regional heterogeneity impacts the conclusions that can be drawn from these studies. As discussed in section II.B.2.c.iii of the preamble to the final rule, in reaching decisions on the primary O_3 NAAQS the Administrator places the greatest weight on the results of controlled human exposure studies and on quantitative analyses based on information from these studies (particularly exposures of concern, as discussed below in II.B.3 and II.C.4), and less weight on risk analyses based on information from epidemiologic studies. In doing so, the Agency continues to note that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O_3 exposures. In addition, the effects reported in these studies are due solely to O_3 exposures, and interpretation of study results is not

complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). The Agency further notes the CASAC judgment that "the scientific evidence supporting the finding that the current standard is inadequate to protect public health is strongest based on the controlled human exposure studies of respiratory effects" (Frey, 2014b, p. 5). Consistent with this emphasis, the HREA conclusions reflect relatively greater confidence in the results of the exposure and risk analyses based on information from controlled human exposure studies than the results of epidemiology-based risk analyses. As discussed in the HREA (section 9.6), several key uncertainties complicate the interpretation of epidemiology-based risk estimates, including the heterogeneity in O_3 effect estimates between locations, the potential for exposure measurement errors in these epidemiologic studies, and uncertainty in the interpretation of the shape of concentration-response functions at lower O_3 concentrations.

(14) *Comment:* Some commenters contended that stochastic variability produces biologically implausible results. This commenter pointed to the individual city-specific raw estimates of the mortality increase attributed to O_3 exposure. Smith et al. (2009) found individual estimates ranged from -2% to approximately +3.5% change in mortality per 10 ppb increase in O_3, with about 25% of the cities experiencing a protective (negative) effect and 75% a slight increase in mortality. The commenter summarized by saying that the data suggest that in 25% of the cities, increased O_3 resulted in lower mortality and that is was not biologically plausible. The commenter further discussed the stochastic variability evident when comparing individual risk estimates from Zanobetti and Schwartz (2008) and Bell et al. (2004), which have cities in common. The commenter stated that nine of the cities had a negative association in one study and a positive association in the other, while eight cities had negative associations in both studies, and suggested that the results are not plausible.

Response: With respect to understanding the nature and magnitude of O_3-related mortality risks, the EPA agrees that epidemiologic studies evaluating health effects associated with short-term O_3 exposures have reported heterogeneity in risk estimates between cities and geographic regions (including some negative estimates) as summarized in section 2.5.4.5 of the ISA. However, focusing only on the individual city-specific risk estimates in multi-city studies is simplistic and does not take into account a variety of factors that may influence the city-specific risk estimates such as individual- and community-level characteristics (e.g., section 6.6.2.2), and exposure error (section 4.6).

As detailed in the ISA there are a number of possible explanations for city-to-city heterogeneity in O_3-mortality risk estimates such as differences in community characteristics (individual- or community-level) across cities that could modify the O_3 effect (e.g., activity patterns, housing type and age distribution, prevalence and use of air conditioning); or effect modification by concentrations of other air pollutants or interactions with temperature or other meteorological factors that vary regionally in the U.S. (ISA, p. 2-34). An evaluation of studies that examined a variety of these factors found that "studies have not consistently identified

specific community characteristics that explain the observed heterogeneity" (ISA, p. 2-35).

Additionally, when comparing risk estimates between cities it is important to take into consideration exposure assignment and the potential implications of exposure measurement error on the results from epidemiologic studies. In the majority of studies that have provided evidence of city-to-city or regional heterogeneity in O_3 risk estimates "community-averaged concentration of an air pollutant measured at central-site monitors is typically used as a surrogate for individual or population ambient exposure" (ISA, p. 4-50). "[T]he use of a community-averaged O_3 concentration in a time-series epidemiologic study may be adequate to represent the day-to-day temporal concentration variability used to evaluate health effects, but may not capture differences in the magnitude of exposure due to spatial variability. Other factors that could influence exposure estimates include nonambient exposure, topography of the natural and built environment, meteorology, measurement errors, use of ambient O_3 concentration as a surrogate for ambient O_3 exposure, and the presence of O_3 in a mixture of pollutants" (ISA, p. 4-51). Exposure measurement error may "under- or over-estimate epidemiologic associations between ambient pollutant concentrations and health outcomes by biasing effect estimates toward or away from the null, and tends to widen confidence intervals around those estimates (Sheppard et al., 2005; Zeger et al. 2000)."

As a result of the heterogeneity in O_3 risk estimates in multi-city studies and potential implications of exposure measurement error, as detailed in the proposal, the PA places relatively less weight on epidemiologic-based risk estimates. In doing so, the PA notes that the overall conclusions from the HREA likewise reflect less confidence in estimates of epidemiologic-based risks than in estimates of exposures and lung function risks. The determination to attach less weight to the epidemiologic based estimates reflects the uncertainties associated with mortality and morbidity risk estimates, including the heterogeneity in effect estimates between epidemiologic study areas, [and] the potential for epidemiologic-based exposure measurement error."

(15) *Comment*: Beyond the comments on technical or methodological aspects of the O_3 epidemiologic evidence, some commenters also contended that the purported consistency of results across epidemiologic studies is the result of inappropriate selectivity on the part of the EPA in focusing on specific studies and specific results within those studies. In particular, commenters contended that EPA favors studies that show positive associations and selectively ignores certain studies that report null results. They also cite a recent study (Goodman et al. 2013) suggesting that, in papers where the results of more than one statistical model are reported, the EPA tends to report the results with the strongest associations.

Response: The EPA strongly disagrees that it has inappropriately focused on specific positive studies or specific positive results within individual studies. The ISA builds upon the assessment of the scientific evidence presented in previous

AQCDs and ISAs. When evaluating new literature, "[s]election of studies for inclusion in the ISA is based on the general scientific quality of the study, and consideration of the extent to which the study is informative and policy-relevant" (ISA, p. liii). Additionally, "the intent of the ISA is to provide a concise review, synthesis, and evaluation of the most policy-relevant science to serve as a scientific foundation for the review of the NAAQS, not extensive summaries of all health, ecological and welfare effects studies for a pollutant" (ISA, p. lv). Therefore, not all studies published since the previous review would be appropriate for inclusion in the ISA. With regard to the specific studies that are included in the ISA, and the analyses focused upon for given studies, the EPA notes that the ISA undergoes extensive peer review in a public setting by the CASAC. This process provides ample opportunity for CASAC and the public to comment on studies not included in the ISA, and on the specific analyses focused on within individual studies. In endorsing the final O_3 ISA as adequate for rule-making purposes, CASAC agreed with the selection and presentation of analyses on which to base the ISA's key conclusions.

The EPA strongly disagrees that the results with the strongest associations are preferentially reported in the ISA. When there are multiple results presented from a study, the EPA focuses on results from the most appropriate statistical model and for the lag period with the most biological relevance. The magnitude or statistical significance of an association is not included in the judgment of which result(s) to present. The EPA also notes that commenters from the environmental community castigated the EPA for ignoring results of studies they regard as favorable, notably panel studies and certain epidemiologic studies. Although the EPA believes there are reasonable responses to these comments and does not accept the conclusions therein voiced, the fact that both industry and environmental groups accuse the EPA of selectivity here suggests that EPA's approach is reasonable and even-handed. CASAC's endorsement of the body of evidence in the ISA, as well as EPA's interpretation of that evidence in the PA, further supports that conclusion.

(16) *Comment:* Some commenters argued that only one out of 12 studies considered by EPA showed an association between long-term exposure to O_3 and premature mortality, but EPA uses this study to justify lowering the standard despite other studies that show the current standard is protective of public health.

Response: These commenters are incorrect. As discussed extensively in the proposal and in the preamble to the final rule, the strongest support for the need to revise the current primary O_3 standard comes from the extensive body of evidence supporting the conclusion that there is a causal relationship between short-term O_3 exposures and respiratory effects. These effects include decreased lung function, increased airway inflammation, increased respiratory symptoms, and respiratory effects that can result in O_3-associated hospital admissions, emergency department visits, and premature mortality. In addition, a large body of evidence supports the ISA conclusion that there is likely to be a causal relationship between long-term O_3 exposures and respiratory effects, including new onset asthma and

respiratory mortality. In contrast, there is considerably greater uncertainty in the evidence supporting a relationship between long-term O_3 exposures and total (i.e., not limited to respiratory) mortality, as reflected in the ISA conclusion that the evidence is suggestive of a causal relationship. These causality determinations, and the evidence supporting them, are discussed in the ISA (e.g., see ISA, Chapter 1 for summaries).

(17) *Comment:* One commenter questioned whether the evidence of the health effects of O_3 exposures apply to Wichita, KS. This commenter argued that more scientific evidence showing the local health effects of sporadic, short-term spikes in O_3 is needed, as opposed to clinical trials or studies in locations with sustained high O_3 levels.

Response: The EPA disagrees with this comment. As discussed in the preamble to the final rule (e.g., see sections II.B, II.C.4), the Administrator places the most emphasis on information from controlled human exposure studies. These studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O_3 exposures, regardless of where those exposures occur. The effects reported in these studies are due solely to O_3 exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). In addition, the O_3 epidemiologic evidence includes studies conducted over broad range of locations, impacted by various types of sources, mixtures of co-occurring pollutants, and patterns of O_3 air quality. The body of epidemiologic evidence provides a broad perspective on the occurrence of O_3-associated health effects across the U.S.

(18) *Comment*: Many commenters on both sides of the issue of the adequacy of the current primary O_3 standard identified "new" studies that were not included in the ISA. Commenters who supported revising the current O_3 standard identified 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 health outcomes for at-risk subgroups that EPA has not identified as being susceptible or vulnerable to O_3 exposure, including pregnant women and their fetuses and individuals with preexisting conditions (e.g., organ transplants, obesity, stroke, cystic fibrosis). Some commenters also pointed to new studies that reported O_3 health effect associations for ambient O_3 concentrations below the current standard (and often below 65 ppb). Some commenters who did not support revision of the current O_3 standard also submitted new studies, but reached different conclusions. These commenters stated that 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: As in prior NAAQS reviews, the EPA is basing its decision in this review on studies and related information included in the ISA, REA, and PA, which have undergone CASAC and public review. The studies included in these documents, and the integration of the scientific evidence presented in them, have undergone extensive critical review by EPA, CASAC, and the public. The rigor of that review makes these studies, and their integrative assessment, the most reliable source of scientific information on which to base decisions on the NAAQS, decisions that all parties recognize as of great importance. NAAQS decisions can have profound impacts on public health and welfare, and these decisions should be based on studies that have been rigorously assessed in an integrative manner not only by EPA but also by the statutorily mandated independent advisory committee, and that have undergone the public review that accompanies this process. This approach is consistent with EPA's practice in prior NAAQS reviews and its interpretation of the requirements of the CAA.

Since the 1970 CAA amendments, the EPA has taken the view that NAAQS decisions are to be based on scientific studies and related information that have been assessed as a part of the pertinent air quality criteria, and has consistently followed this approach. This longstanding interpretation was strengthened by new legislative requirements enacted in 1977, which added section 109(d)(2) of the Act concerning CASAC review of air quality criteria.[41] As discussed in the EPA's 1993 decision not to revise the NAAQS for O_3, new studies may sometimes be of such significance that it is appropriate to delay a decision on revision of a NAAQS and to supplement the pertinent air quality criteria so the studies can be taken into account (58 FR at 13013–13014, March 9, 1993). In the present case, in light of the Administrator's focus on respiratory effects attributable to short-term O_3 exposures, and her focus on information from controlled human exposure studies of ambient or near-ambient O_3 concentrations, the EPA notes that studies of such significance have not been submitted by commenters. For this reason, reopening the air quality criteria review would not be warranted even if there were time to do so under the court order governing the schedule for this rulemaking.

Additionally, EPA has provisionally considered all of the peer-reviewed evidence mentioned by public commenters (Appendix A) and determined that the additional evidence does not substantially change the conclusions reached in the 2013 O_3 ISA.

Accordingly, the EPA is basing the final decisions in this review on the studies and related information included in the ISA, REA, and PA that have undergone CASAC and public review. EPA will consider "new" studies for purposes of decision-making in the next periodic review of the O_3 NAAQS, which will

[41] See 71 FR 61144, 61148 (October 17, 2006) (final decision on review of PM NAAQS) for a detailed discussion of this issue and EPA's past practice.

provide the opportunity to fully assess these studies through a more rigorous review process involving EPA, CASAC, and the public.

iii. Comments on At-Risk Populations

A number of groups submitted comments on the EPA's identification of at-risk populations and lifestages.

(1) *Comment:* Some industry commenters who opposed revising the current standard disagreed with the EPA's identification of people with asthma or other respiratory diseases as an at-risk population for O_3-attributable effects, citing controlled human exposure studies that did not report larger O_3-induced FEV_1 decrements in people with asthma than in people without asthma.

Response: We disagree with comments that the evidence does not support the identification of asthmatics as an at-risk population. As summarized in the proposal, the EPA's identification of populations at risk of O_3 effects is based on a systematic approach that assesses the current scientific evidence across the relevant scientific disciplines (i.e., exposure sciences, dosimetry, controlled human exposure, toxicology, and epidemiology), with a focus on studies that conducted stratified analyses allowing for an evaluation of different populations exposed to similar O_3 concentrations within the same study design (ISA, pp. 8-1 to 8-3). Based on this established process and framework, the ISA identifies individuals with asthma among the populations and lifestages for which there is "adequate" evidence to support the conclusion of increased risk of O_3-related health effects. Other populations for which the evidence is adequate are individuals with certain genotypes, younger and older age groups, individuals with reduced intake of certain nutrients, and outdoor workers. These conclusions are based on consistency in findings across studies and evidence of coherence in results from different scientific disciplines.

For example, with regard to people with asthma, the ISA notes a number of epidemiologic and controlled human exposure studies reporting larger and/or more serious effects in people with asthma than in people without asthma or other respiratory diseases. These include epidemiologic studies of lung function, respiratory symptoms, and medication use, as well as controlled human exposure studies showing larger inflammatory responses and markers indicating altered immune functioning in people with asthma, and also includes evidence from animal models of asthma that informs the EPA's interpretation of the other studies. We disagree with the industry commenters' focus solely on the results of certain studies without an integrated consideration of the broader body of evidence, and wider range of respiratory endpoints. It is such an integrated approach that supports EPA's conclusion that "there is adequate evidence for asthmatics to be an at-risk population" (ISA, section 8.2.2).

We also disagree with commenters' misleading reference to various studies cited to support the claim that asthmatics are not at increased risk of O_3-related health

effects. One of the controlled human studies cited (Mudway et al. 2001) involved asthmatic adults who were older than the healthy controls, and it is well-recognized that responses to O_3 decrease with age.[42] Another study (Alexis et al. 2000) used subjects with mild asthma who are unlikely to be as responsive as people with more severe disease (Horstman et al., 1995) (PA, p. 3-80). Controlled human exposure studies and epidemiologic studies of adults and children amply confirm that "there is adequate evidence for asthmatics to be an at-risk population" (PA, p. 3-81).

(2) *Comment:* Some industry commenters further contended that there is no evidence that patients with asthma or COPD, or children, are any more responsive to acute O_3 exposure than healthy adults. These commenters argued that there is evidence that these populations have even shown smaller O_3-induced decrements than healthy people. They pointed to studies that have shown that transient declines of 10-20% are common in asthmatics, especially at night, and that FEV_1 and FVC values generally are not well correlated with symptoms in asthmatics.

Response: With regard to children, we disagree with these commenters' position. As discussed in the ISA (section 4.4.1) and in section II.B.3 of the preamble to the final rule, children spend more time than adults being physically active outdoors and are more likely to experience the types of O_3 exposures that have been shown to cause respiratory effects. Compared to adults, children also have higher ventilation rates relative to their lung volumes, which tends to increase the O_3 dose when normalized to lung surface area (e.g., ISA, section 8.3.1.1). In addition, as noted section II.A.1.c with respect to lung function decrements, responsiveness to O_3 exposure decreases with increasing age. Evidence from controlled human exposure studies indicate that children are at least as responsive as healthy young adults (18 year olds), which is why lung function responses of 18 year olds are used to estimate lung function risk in the HREA. Given these factors related to exposure and dose, together with evidence from some animal toxicology studies reporting larger effects in younger animals and some epidemiologic studies reporting larger associations with respiratory hospital admissions and emergency department visits in children than adults (ISA, section 8.3.1.1),[43] we do not agree with commenters that the evidence does not support children as an at-risk population.

For the reasons noted above (see previous comment), we also disagree with these commenters' position on people with asthma (see also ISA, section 8.2.2).[44] In addition to the discussion of this issue above, we note that while lung function

[42] Indeed, elsewhere in its comments, they in fact stated (correctly) that spirometric responses diminish in middle-aged and older adults.

[43] As indicated, the ISA considers a wider range of studies and endpoints than just the FEV_1 decrements that seem to have provided the basis for these commenters' conclusions (ISA, section 8.3.1.1).

[44] Though we agree that there is greater uncertainty with regard to people with COPD. The ISA concludes that the "small number of studies provides inadequate evidence to determine whether COPD results in increased risk of O_3-related health effects" (ISA, section 8.2.3).

was not well correlated with symptoms in asthmatics under baseline conditions (r=0.09) (i.e., based on Carranza Rosenzweig et al., 2004, as cited by one commenter), study authors found that following 12 weeks of asthma treatment (e.g., inhaled corticosteroids) there was a moderate correlation between improvement in FEV_1 and improvement in overall quality of life scores (r=0.38). These authors noted that patients live with their disease by avoiding or omitting from their lifestyle those things that impact their disease and that have the potential to exacerbate their symptoms (Carranza Rosenzweig et al., 2004, p. 1163). See also response to comments in Inflammation section above.

(3) *Comment:* In contrast, comments from medical, environmental, and public health groups generally agreed with the at-risk populations identified by EPA, and also identified other populations that they stated should be considered at risk, including people of lower socio-economic status; people with diabetes or who are obese; pregnant women (due to reproductive and developmental effects); people with COPD; recipients of organ transplants; people with cystic fibrosis; and African American, Asian or Hispanic/Latino communities. They also urged that adult "responders" – healthy adults with a special sensitivity to O_3 exposure – be considered an at-risk population. In some cases, commenters cited recent scientific studies to support their positions. In addition, a number of tribal groups submitted comments suggesting that tribal communities should also be considered at-risk, given the greater prevalence of asthma and more time spent outdoors compared to non-tribal communities.

As support for the additional populations, some of these commenters cited scientific studies, including studies that were not included in the ISA (discussed in section I.C of the preamble to the final rule and above in section II.A.1.b).

Response: We agree with the comments that are consistent with the conclusions of the ISA regarding at-risk populations. As such, we agree with commenters who asserted that children, people with asthma, and people who spend a large amount of time being active outdoors are important at-risk populations. However, we do not agree that there is sufficient evidence to support the identification of additional populations (i.e., beyond those identified in the ISA) as at risk of O_3-attributable health effects.[45] The EPA has relied on the ISA's conclusions regarding populations at increased risk of O_3-related effects (ISA, Chapter 8), which have been reviewed and endorsed by CASAC (Frey and Samet, 2012a).

To identify factors that potentially lead to some populations being at greater risk to air pollutant-related health effects, the evidence across relevant scientific

[45] Though, as highlighted by commenters, we agree that some groups (e.g., some racial, ethnic, tribal groups) include larger proportions of at-risk individuals, such as people with asthma or people who are active outdoors for long periods of time, than the general population. As discussed in the preamble to the final rule (II.A.2.b, II.A.2.c), people with asthma and people who are active outdoors for prolonged periods of time were evaluated as part of the HREA's exposure and risk analyses. The results of these analyses inform the Administrator's decisions on the current, and a revised, primary O_3 standard (see sections II.B.2, II.B.3, II.C.4.b, II.C.4.c in the preamble to the final rule).

disciplines (i.e., exposure sciences, dosimetry, controlled human exposure, toxicology, and epidemiology) was evaluated in the ISA. In this systematic approach, the collective evidence was used to examine coherence of effects across disciplines and determine biological plausibility. By first focusing on studies that conducted stratified analyses (i.e., epidemiologic or controlled human exposure) it is possible to identify factors that may result in some populations being at greater risk of an air pollutant related health effect. These types of studies allowed for an evaluation of populations exposed to similar air pollutant (e.g., O_3) concentrations within the same study design. Experimental studies also provide important lines of evidence in the evaluation of factors that may lead to increased risk of an air pollutant related-health effect. Toxicological studies conducted using animal models of disease and controlled human exposure studies that examine individuals with underlying disease or genetic polymorphisms may provide evidence to inform whether a population is at increased risk of an air pollutant related health effect in the absence of stratified epidemiologic analyses. Additionally these studies can provide support for coherence with the health effects observed in epidemiologic studies as well as an understanding of biological plausibility. Information on factors that may result in increased risk of O_3-related health effects can also be obtained from studies that examine exposure differences between populations. The collective results across the scientific disciplines comprise the overall weight of evidence used in the ISA to determine whether a specific factor results in a population being at increased risk of an air pollutant related health effect.

The ISA presents conclusions regarding the strength of evidence, based on the evaluation and synthesis across scientific disciplines, for each factor that may contribute to increased risk of an O_3-related health effect. The conclusions were drawn while considering the "Aspects to Aid in Judging Causality" discussed in Table 1 of the Preamble to the ISA. The categories considered for evaluating the potential increased risk of an air pollutant-related health effect are "adequate evidence," "suggestive evidence," "inadequate evidence," and "evidence of no effect." They are described in more detail in Table 8-1 of the ISA.

The populations and lifestages identified in the ISA as having "adequate evidence" for increased O_3-related health effects are individuals with certain genotypes, individuals with asthma, younger and older age groups, individuals with reduced intake of certain nutrients, and outdoor workers, based on consistency in findings across studies and evidence of coherence in results from different scientific disciplines. Other populations were identified in the ISA as having either "suggestive evidence" or "inadequate evidence." These included sex, socioeconomic status, obesity, influenza/infection, chronic obstructive pulmonary disease, cardiovascular disease, diabetes, hyperthyroidism, race/ethnicity, smoking, and air conditioning use.

Based on the EPA's provisional consideration of studies published since the completion of the ISA (Appendix A), recent studies that examine other groups or

effects highlighted by commenters are not sufficient to materially change the ISA's conclusions on at-risk populations.

Specifically with regard to pregnant women, the ISA concluded that the "evidence is suggestive of a causal relationship between exposures to O_3 and reproductive and developmental effects" including birth outcomes, noting that "the collective evidence for many of the birth outcomes examined is generally inconsistent" (ISA, pp. 7-74 and 7-75). At the time of the completion of the ISA, no studies had been identified that examined the relationship between exposure to O_3 and the health of pregnant women (e.g., studies on pre-eclampsia, gestational hypertension). Due to the generally inconsistent epidemiologic evidence for effects on birth outcomes, the lack of studies on the health of pregnant women, and the lack of studies from other disciplines to provide biological plausibility for the effects examined in epidemiologic studies, pregnant women were not considered an at-risk population. Based on the EPA's provisional consideration of studies published since the completion of the ISA (Appendix A), recent studies that examine exposure to O_3 and pre-eclampsia and other health effects experienced by pregnant women are not sufficient to materially change the ISA's conclusions on at-risk populations. In addition, as summarized in the proposal, the ISA concluded that the evidence for other populations was either suggestive of increased risk, with further investigation needed (e.g., other genetic variants, obesity, sex, and socioeconomic status), or was inadequate to determine if they were of increased risk of O_3-related health effects (e.g., influenza/infection, COPD, CVD, diabetes, hyperthyroidism, smoking, race/ethnicity, and air conditioning use) (ISA, section 2.5.4.1). The CASAC has concurred with the ISA conclusions (Frey, 2014b).

With respect to the comment that tribal communities should also be considered an at-risk population, given the greater prevalence of asthma and more time spent outdoors compared to non-tribal communities, consistent with the assessment conducted in each NAAQS review, the EPA has evaluated the available evidence with regard to populations that may be at greater risk of O_3 health effects than the general population. That assessment, described in the ISA, identified people with asthma and people who are active outdoors as groups at greater risk. As discussed in section III below, the commenter has provided no evidence that revision of the O_3 standard to a level of 70 ppb will result in a disproportionate impact on Native Americans or Alaska natives, other than has been assessed and considered by the Administrator in this review.

In addition to the comments addressed above and in the preamble to the final rule, a number of commenters provided additional comments regarding EPA's characterization of at-risk populations.

(4) *Comment:* One group of commenters, primarily representing the medical associations, public health groups and environmental groups, suggested that, because the number of individuals with certain genetic variants and reduced intake of certain nutrients are currently unknown, the addition of these groups to

the at-risk populations indicates that more people may be at risk of O_3-related health effects and support stronger standards than were adopted in 2008.

Response: The EPA agrees that the evidence and the exposure and risk information available in this review supports a stronger primary O_3 standard than was set in 2008. Based on this evidence and information, we anticipate that the revised standard with its level of 70 ppb will result in important improvements in public health broadly across the population, including in at-risk populations such as children, people with asthma, older adults, and other groups for which exposures and risks have not been quantified.

(5) *Comment:* Another group of commenters, primarily representing industry associations and businesses opposed to revising the primary O_3 standard, asserted that the sensitivity of at-risk populations is not accurately represented by EPA. With regard to asthmatics as an at-risk population, these commenters emphasized purported inconsistencies in the supporting evidence. In some cases, these commenters pointed to studies comparing spirometric responses between people with asthma and non-asthmatic healthy individuals, highlighting that only one of nine studies found that asthmatics have a larger O_3-induced response. Commenters also criticized the lack of healthy controls in studies considered for non-spirometric endpoints, claiming that it is unclear if responses are enhanced in asthmatics or if it is a standard response. Additionally, commenters suggested that, while the studies used mild asthmatics, individuals with moderate and severe asthma would not be able to sustain the exercise levels required to reach an O_3 dose at which an effect would be seen, and therefore would not be able to attain that dose in a real-life exposure situation.

Response: Evidence for people with asthma as an at-risk population is discussed in detail in section 8.2.2 of the ISA and is summarized in section II.B.4 of the proposal. Though there is variability in results for some specific endpoints, as the commenters note, the collective evidence from controlled human exposure studies, epidemiologic studies and animal toxicological studies supports the increased risk of O_3-related health effects among individuals with asthma. The comment about individuals with moderate asthma not being able to sustain the exertion levels required to reach an O_3 dose at which an effect would be seen, has been addressed in responses in section A.1.b.i above and in section II.B.2.b.iii of the preamble to the final rule. Responses to related comments are discussed in this section. The extent to which more severe asthmatics could sustain an exertion level adequate to reach an O_3 dose sufficient to cause an effect is an uncertainty since this group has not been experimentally evaluated. However, it should be emphasized that the activity level used in prolonged exposure studies (i.e., 6.6 hours or longer) is equivalent to a brisk walk, not jogging or running (see Table 6-1 of the ISA). Additionally, the positive association observed by Horstman et al. (1995) between asthma severity and FEV_1 response to O_3 exposure indicates that severe asthmatics could potentially experience effects at lower O_3 doses than mild or moderate asthmatics.

In the 2006 AQCD, the potential for individuals with asthma to have greater risk of O_3-related health effects was supported by a number of controlled human exposure studies, evidence from toxicological studies, and a limited number of epidemiologic studies. Overall, in the recent epidemiologic literature some, but not all, studies report greater risk of health effects among individuals with asthma. Studies examining effect measure modification of the relationship between short-term O_3 exposure and altered lung function by corticosteroid use provided limited and inconsistent evidence of O_3-related health effects. Additionally, recent studies of behavioral responses have found that studies do not take into account individual behavioral adaptations to forecasted air pollution levels (such as avoidance and reduced time outdoors), which may underestimate the observed associations in studies that examined the effect of O_3 exposure on respiratory health (Neidell and Kinney, 2010). This could explain some inconsistency observed among recent epidemiologic studies. The evidence from controlled human exposure studies provides support for increased decrements in FEV_1 and greater inflammatory responses to O_3 in individuals with asthma than in healthy individuals without a history of asthma. These studies are often performed among individuals with mild asthma and therefore it is possible that individuals with severe asthma may have an even greater risk of O_3-related health effects. The collective evidence for increased risk of O_3-related health effects among individuals with asthma from controlled human exposure studies is supported by recent toxicological studies which provide biological plausibility for heightened risk of asthmatics to respiratory effects due to O_3 exposure. Evidence indicating O_3-induced respiratory effects among individuals with asthma is further supported by additional studies of O_3-related respiratory effects (ISA, section 6.2). Overall, the ISA concludes that there is adequate evidence for asthmatics to be an at-risk population based on the substantial, consistent evidence among controlled human exposure studies and coherence from epidemiologic and toxicological studies.

Multiple epidemiologic studies assessed in the ISA evaluated the potential for increased risk of O_3-related health effects among individuals with asthma. A study of lifeguards in Texas reported decreased lung function with short-term O_3 exposure among both individuals with and without asthma; however, the decrease was greater among those with asthma (Thaller et al., 2008). A Mexican study of children ages 6-14 detected an association between short-term O_3 exposure and wheeze, cough, and bronchodilator use among asthmatics but not non-asthmatics, although this may have been the result of a small non-asthmatic population (Escamilla-Nuñez et al., 2008). A study of modification by airway hyperresponsiveness (AHR) (a condition common among asthmatics) reported greater short-term O_3-associated decreases in lung function in elderly individuals with AHR, especially among those who were obese (Alexeeff et al., 2007). However, no evidence for increased risk was found in a study performed among children in Mexico City that examined the effect of short-term O_3 exposure on respiratory health (Barraza-Villarreal et al., 2008). In this study, a positive association was reported for airway inflammation among asthmatic children, but the observed association was similar in magnitude to that of non-asthmatics. Similarly, a study of children in California reported an association between O_3

concentration and exhaled nitric oxide fraction (FeNO) that persisted both among children with and without asthma as well as those with and without respiratory allergy (Berhane et al., 2011). Finally, Khatri et al. (2009) found no association between short-term O_3 exposure and altered lung function for either asthmatic or non-asthmatic adults, but did note a decrease in lung function among individuals with allergies.

Evidence for differences in effects among asthmatics has been observed in studies that examined the association between O_3 exposure and altered lung function by asthma medication use. A study of children with asthma living in Detroit reported a greater association between short-term O_3 and lung function for corticosteroid users compared with noncorticosteroid users (Lewis et al., 2005). Conversely, another study of children found decreased lung function among noncorticosteroid users compared to corticosteroid users, although in this study, a large proportion of non-users were considered to be persistent asthmatics (Hernández-Cadena et al., 2009). Lung function was not related to short-term O_3 exposure among corticosteroid users and non-users in a study taking place among children during the winter months in Canada (Liu et al., 2009). Additionally, a study of airway inflammation among individuals aged 12-65 years old reported a counterintuitive inverse association with O_3 of similar magnitude for all groups of corticosteroid users and non-users (Qian et al., 2009).

Controlled human exposure studies that have examined the effects of O_3 on individuals with asthma and healthy controls are limited. Based on studies reviewed in the 1996 and 2006 O_3 AQCDs, subjects with asthma appeared to be at least as sensitive to acute effects of O_3 in terms of FEV_1 and inflammatory responses as healthy non-asthmatic subjects. For instance, Horstman et al. (1995) observed that mild-to-moderate asthmatics, on average, experienced double the O_3-induced FEV_1 decrement of healthy subjects (19% versus 10%, respectively, $p = 0.04$). Moreover, a statistically significant positive correlation between FEV_1 responses to O_3 exposure and baseline lung function was observed in individuals with asthma, i.e., responses increased with severity of disease. Kreit et al. (1989) performed a short duration study in which asthmatics also showed a considerably larger average O_3-induced FEV_1 decrement than the healthy controls (25% versus 16%, respectively) following exposure to O_3 with moderate-heavy exercise. Alexis et al. (2000) and Jorres et al. (1996) also reported a tendency for slightly greater FEV_1 decrements in asthmatics than healthy subjects. Minimal evidence exists suggesting that individuals with asthma have smaller O_3-induced FEV_1 decrements than healthy subjects (3% versus 8%, respectively) (Mudway et al., 2001). However, the asthmatics in that study also tended to be older than the healthy subjects, which could partially explain their lesser response since FEV_1 responses to O_3 exposure diminish with age (as noted above). Individuals with asthma also had more neutrophils in the BALF (18 hours postexposure) than similarly exposed healthy individuals (Scannell et al., 1996; Basha et al., 1994).

Some studies (e.g., Peden et al., 1997) have demonstrated eosinophilic responses to O_3 in individuals with asthma, but did not include a group healthy controls.

With respect to the eosinophil infiltration, the proposal (79 FR 75266) states that in asthma, the eosinophil, which increases inflammation and allergic responses, is the cell most frequently associated with exacerbations of the disease and that the accumulation of eosinophils in the airways of asthmatics is followed by production of mucous and a late-phase bronchial constriction. Clearly this cascade of responses is not beneficial. Elevated eosinophil levels within the respiratory tract are associated with allergy and asthma making the inclusion of a healthy control group unimportant. Healthy controls are not necessarily needed to interpret the effects of O_3 on other non-spirometric endpoints such as air responsiveness in asthmatics. As clearly indicated in the ISA (p. 6-73), people with asthma are generally more sensitive to bronchoconstricting agents than those without asthma, and the use of an airway challenge to inhaled bronchoconstricting agents is a diagnostic test in asthma. That is, at baseline individuals with asthma are more responsive to bronchial challenge than healthy controls. The study by Kreit et al. (1989) showed a similar percent increase in airway responsiveness following O_3 exposure in healthy and asthmatic adults. This finding could lead to the erroneous conclusion that asthmatic response to O_3 is no different from that of healthy individuals. However, the asthmatics in the Kreit et al. (1989) study were 60- to 70-times more reactive than the healthy controls at baseline. Thus, the individuals with asthma were at-risk at baseline relative to healthy adults without O_3 further increasing their airway responsiveness. Therefore, for health endpoints where asthmatics are sufficiently different from healthy individuals at baseline, comparison against the responses of healthy individuals is not necessarily needed to establish asthmatics as an at-risk group.

Furthermore, a study examining the effects of O_3 on individuals with atopic asthma and healthy controls reported that greater numbers of neutrophils, higher levels of cytokines and hyaluronan, and greater expression of macrophage cell-surface markers were observed in induced sputum of atopic asthmatics compared with healthy controls (Hernandez et al., 2010). Differences in O_3-induced epithelial cytokine expression were noted in bronchial biopsy samples from asthmatics and healthy controls (Bosson et al., 2003). Cell-surface marker and cytokine expression results, and the presence of hyaluronan, are consistent with O_3 having greater effects on innate and adaptive immunity in these asthmatic individuals. In addition, studies have demonstrated that O_3 exposure leads to increased bronchial reactivity to inhaled allergens in mild allergic asthmatics (Kehrl et al., 1999; Jorres et al., 1996) and to the influx of eosinophils in individuals with pre-existing allergic disease (Vagaggini et al., 2002; Peden et al., 1995).

Taken together, these results point to several mechanistic pathways which could account for increased risk of O_3–related health effects in subjects with asthma (see ISA, section 5.4.2.2). Toxicological studies provide biological plausibility for greater effects of O_3 among those with asthma or AHR. In animal toxicological studies, an asthmatic phenotype is modeled by allergic sensitization of the respiratory tract. Many of the studies that provide evidence that O_3 exposure is an inducer of AHR and remodeling utilize these types of animal models. For

example, a series of experiments in infant rhesus monkeys have shown these effects, but only in monkeys sensitized to house dust mite allergen (Fanucchi et al., 2006; Joad et al., 2006; Schelegle et al., 2003). Similarly, Funabashi et al. (2004) demonstrated changes in pulmonary function in mice exposed to O_3, and Wagner et al. (2007) demonstrated enhanced inflammatory responses in rats exposed to O_3, but only in animals sensitized to allergen. In general, it is the combined effects of O_3 and allergic sensitization which result in measurable effects on pulmonary function. In a bleomycin induced pulmonary fibrosis model, exposure to 250 ppb O_3 for 5 days increased pulmonary inflammation and fibrosis, along with the frequency of bronchopneumonia in rats (Oyarzún et al., 2005). Thus, short-term exposure to O_3 may enhance damage in a previously injured lung.

(6) *Comment:* With regard to lifestages, some commenters focused on EPA's consideration of children as an at-risk population. Commenters stated that the evidence that children are at increased risk is not consistently supported by the literature and that the body of evidence is not suggestive of a dose-response relationship for effects in children. Commenters agreed that children are more likely to be exposed to O_3 because they spend more time outdoors at higher ventilation rates. However, this commenter disagreed with EPA's conclusions regarding the spirometric response of children and adolescents to O_3. These commenters pointed to EPA's conclusion that children, adolescents, and young adults (<18 years old) have equivalent spirometric responses, which are greater than middle-aged and older adults, and suggested that EPA failed to mention that the responses of children and adolescents are equivalent to the responses of young adults (18-35 years old) and diminishes in middle-aged and older adults.

Response: With regard to spirometric responses, the EPA agrees that O_3-induced FEV_1 responses decrease with increasing age. As discussed in the ISA (e.g., p. 6-22), in healthy individuals, the fastest rate of decline in O_3 responsiveness appears between the ages of 18 and 35 years. During the middle age period (35-55 years), O_3 sensitivity continues to decline, but at a much lower rate. Beyond this age (>55 years), acute O_3 exposure elicits minimal spirometric changes. With regard to differences between children and adults, the ISA (p. 6-21) clearly states that healthy children exposed to filtered air and 120 ppb O_3 experienced similar spirometric responses as young healthy adults (McDonnell et al., 1985a). The implications of this evidence for the HREA's FEV_1 risk assessment in children are discussed in section II.A.2.c.i of the preamble to the final rule.

However, the evidence for children as an at-risk population goes well-beyond studies of spirometric responses.[46] Children are considered to be at greater risk from O_3 exposure in part because their respiratory systems undergo lung growth until about 18-20 years of age and are therefore thought to be intrinsically more at

[46] Whether the same age-dependent pattern of O_3 sensitivity decline also holds for nonspirometric pulmonary function, airway reactivity or inflammatory endpoints has not been determined (ISA, section 6.2.1.1).

risk for O_3-induced damage (U.S. EPA, 2006a). It is also generally recognized that children spend more time outdoors than adults, and, therefore, would be expected to have higher exposure to O_3 than adults. Children aged 11 years and older and adults have higher absolute ventilation rates than younger children aged 1-11 years. However, younger children have higher ventilation rates relative to their lung volumes, which tends to increase dose normalized to lung surface area. In all ages, exercise intensity has a substantial effect on ventilation rate, high intensity activity results in nearly double the ventilation rate for moderate activity. For more information on time spent outdoors and ventilation rate differences by age group, see section 4.4.1 in the ISA. Children are also more likely than adults to have asthma.

The 1996 AQCD reported clinical evidence that children, adolescents, and young adults (<18 years of age) appear, on average, to have nearly equivalent spirometric responses to O_3 exposure, but have greater responses than middle-aged and older adults (U.S. EPA, 1996a). Symptomatic responses (e.g., cough, shortness of breath, pain on deep inspiration) to O_3 exposure, however, appear to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996a); see also other responses in this section above. Complete lung growth and development is not achieved until 18-20 years of age in women and the early 20s for men; pulmonary function is at its maximum during this time as well (see also PA, section 3.1.5 and ISA, chapter 8).

Recent epidemiologic studies have examined different age groups and their risk of O_3-related respiratory hospital admissions and emergency department visits. Evidence for greater risk in children was reported in several studies. A study in Cyprus of short-term O_3 concentrations and respiratory hospital admissions detected possible effect measure modification by age with a larger association among individuals < 15 years of age compared with those > 15 years of age; the effect was apparent only with a 2-day lag (Middleton et al., 2008). Similarly, a Canadian study of asthma-related emergency department visits reported the strongest O_3-related associations among 5- to 14-year olds compared to the other age groups (ages examined 0-75+) (Villeneuve et al., 2007). Greater O_3-associated risk in asthma-related emergency department visits were also reported among children (<15 years) as compared to adults (15 to 64 years) in a study from Finland (Halonen et al., 2009). A study of New York City hospital admissions demonstrated an increase in the association between O_3 exposure and asthma-related hospital admissions for 6- to 18-year olds compared to those < 6 years old and those > 18 years old (Silverman and Ito, 2010). When examining long-term O_3 exposure and asthma-related hospital admissions among children, associations were determined to be larger among children 1 to 2 years old compared to children 2 to 6 years old (Lin et al., 2008). A few studies reported positive associations among both children and adults and no modification of the effect by age.

The evidence reported in epidemiologic studies is supported by recent toxicological studies which observed O_3-induced health effects in immature

animals. Early life exposures of multiple species of laboratory animals, including infant monkeys, resulted in changes in conducting airways at the cellular, functional, ultra-structural, and morphological levels. The studies conducted on infant monkeys are most relevant for assessing effects in children. Carey et al. (2007) conducted a study of O_3 exposure in infant rhesus macaques, whose respiratory tract closely resemble that of humans. Monkeys were exposed either acutely or in episodes designed to mimic human exposure. All monkeys acutely exposed to O_3 had moderate to marked necrotizing rhinitis, with focal regions of epithelial exfoliation, numerous infiltrating neutrophils, and some eosinophils. The distribution, character, and severity of lesions in episodically exposed infant monkeys were similar to that of acutely exposed animals. Neither exposure protocol for the infant monkeys produced mucous cell metaplasia proximal to the lesions, an adaptation observed in adult monkeys exposed in another study (Harkema et al., 1987). Functional and cellular changes in conducting airways were common manifestations of exposure to O_3 among both the adult and infant monkeys (Plopper et al., 2007). In addition, the lung growth of the distal conducting airways in the infant monkeys was significantly stunted by O_3 and this aberrant development was persistent 6 months post-exposure (Fanucchi et al., 2006).

Age may also affect the inflammatory response to O_3 exposure. Toxicological studies reported that the difference in effects among younger lifestage test animals may be due to age-related changes in antioxidants levels and sensitivity to oxidative stress. Further discussion of these studies may be found in section 8.3.1.1 of the ISA (p. 8-18).

The previous and recent human clinical and toxicological studies reported evidence of increased risk from O_3 exposure for younger ages, which provides coherence and biological plausibility for the findings from epidemiologic studies. Although there was some inconsistency, generally, the epidemiologic studies reported positive associations among both children and adults or just among children. The interpretation of these studies is limited by the lack of consistency in comparison age groups and outcomes examined. However, overall, the epidemiologic, controlled human exposure, and toxicological studies provide adequate evidence that children are at increased risk of O_3-related health effects.

(7) *Comment:* Some commenters also questioned the EPA's characterization of the elderly as an at-risk population. Commenters asserted that while there is some support for an increased risk of mortality, the evidence is not consistent for other endpoints, particularly for an increased risk of O_3-related CV morbidity in older adults. Other commenters stated that the difference in the average time spent outdoors between the elderly and younger adults is not a big enough difference to consider the elderly at-risk for O_3 exposure.

Response: The ISA notes that older adults are at greater risk of health effects associated with O_3 exposure through a variety of intrinsic pathways (ISA, section 8.3.1.2). In addition, older adults may differ in their exposure and internal dose.

Older adults were outdoors for a slightly longer proportion of the day than adults aged 18-64 years. For more information on time spent outdoors by age group, see section 4.4 in the ISA. The gradual decline in physiological processes that occurs with aging may lead to increased risk of O_3-related health effects (U.S. EPA, 2006a). Respiratory symptom responses to O_3 exposure appears to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996a); lung function responses to O_3 exposure also decline from early adulthood (U.S. EPA, 1996a). The reductions of these responses with age may put older adults at increased risk for continued O_3 exposure (i.e., a lack of symptoms may result in their not avoiding or ceasing exposure) (ISA, p. 6-22). In addition, older adults, in general, have a higher prevalence of preexisting diseases compared to younger age groups and this may also lead to increased risk of O_3-related health effects (ISA, section 8.3.1.2). With the number of older Americans increasing in upcoming years (estimated to increase from 12.4% of the U.S. population to 19.7% between 2000 to 2030, which is approximately 35 million and 71.5 million individuals, respectively) this group represents a large population potentially at risk of O_3-related health effects (SSDAN CensusScope, 2010; U.S. Census Bureau, 2010).

The majority of recent studies reported greater effects of short-term O_3 exposure and mortality among older adults, which is consistent with the findings of the 2006 AQCD. A study (Medina-Ramón and Schwartz, 2008) conducted in 48 cities across the U.S. reported larger effects among adults ≥65 years old compared to those < 65 years. Further investigation of this study population revealed a trend of O_3-related mortality risk that gets larger with increasing age starting at age 51 (Zanobetti and Schwartz, 2008). Another study conducted in 7 urban centers in Chile reported similar results, with greater effects in adults ≥65 years old (Cakmak et al., 2007). More recently, a study conducted in the same area reported similar associations between O_3 exposure and mortality in adults aged < 64 years old and 65 to 74 years old, but the risk was increased among the older age group (Cakmak et al., 2011). A study performed in China reported greater effects in populations ≥45 years old (compared to 5 to 44 year olds), with statistically significant effects present only among those ≥65 years old (Kan et al., 2008). An Italian study reported higher risk of all-cause mortality associated with increased O_3 concentrations among individuals ≥85 year old as compared to those 35 to 84 years old (Stafoggia et al., 2010). The Air Pollution and Health: A European and North American Approach (APHENA) project examined the association between O_3 exposure and mortality for those <75 and ≥ 75 years of age. In Canada, the associations for all-cause and cardiovascular mortality were greater among those ≥75 years old. In the U.S., the association for all-cause mortality was slightly greater for those <75 years of age compared to those ≥75 years old in summer-only analyses. No consistent pattern was observed for CVD mortality. In Europe, slightly larger associations for all-cause mortality were observed in those <75 years old in all-year and summer-only analyses. Larger associations were reported among those <75years for CVD mortality in all-year analyses, but the reverse was true for summer-only analyses (Katsouyanni et al., 2009).

With respect to epidemiologic studies of O_3 exposure and hospital admissions, a positive association was reported between short-term O_3 exposure and respiratory hospital admissions for adults ≥ 65 years old but not for those adults aged 15 to 64 years (Halonen et al., 2009). In the same study, no association was observed between O_3 concentration and respiratory mortality among those ≥65 years old or those 15 to 64 years old. No modification by age (40 to 64 year olds versus >64 year olds) was observed in a study from Brazil examining O_3 levels and COPD-related emergency department visits.

Although some outcomes reported mixed findings regarding an increase in risk for older adults, recent epidemiologic studies report consistent positive associations between short-term O_3 exposure and mortality in older adults. The evidence from mortality studies is consistent with the results reported in the 2006 AQCD and is supported by toxicological studies providing biological plausibility for increased risk of effects in older adults. Also, older adults may be experiencing increased exposure compared to younger adults. Overall, the ISA concludes adequate evidence is available indicating that older adults are at increased risk of O_3-related health effects.[47]

(8) *Comment:* With regard to individuals with certain genetic variants, commenters note that the evidence from the studies reviewed in the ISA is insufficient to support the Administrator's conclusion that there is adequate evidence for populations with certain genotypes being more at-risk than others to the effects of O_3 exposure. Commenters specifically point to limitations in the studies evaluated in the ISA, particularly related to potential confounders in the communities that were studied and that only one study (Romieu et al., 2006) found an effect modification in asthma symptoms in children following O_3 exposure with specific genotype variants compared to those without the genotype.

Response: The potential effects of air pollution on individuals with specific genetic characteristics have been examined; studies often target polymorphisms in already identified candidate susceptibility genes or in genes whose protein products are thought to be involved in the biological mechanism underlying the health effect of an air pollutant (Sacks et al., 2011). As a result, multiple studies that examined the effect of short- and long-term O_3 exposure on respiratory function have focused on whether various gene profiles lead to an increased risk of O_3-related health effects. For more details on the function and mode of action of the genetic factors, see Section 5.4.2.1 of the ISA.

Additionally, a limited number of toxicological studies have examined the joint effects of nutrition and genetics. Details on these toxicological studies of nutrition and genetics can be found in Section 5.4.2.3 of the ISA. Multiple genes, including glutathione S-transferase Mu 1 (GSTM1) and tumor necrosis factor-α (TNF-α)

[47] Note that although older adults are less susceptible to asthma than younger adults and children, the remaining evidence relating to other health end points in the response above amply supports the determination that older adults comprise an at-risk population for ozone exposure.

were evaluated in the 2006 AQCD and found to have a "potential role… in the innate susceptibility to O_3" (U.S. EPA, 2006a). Epidemiologic, controlled human exposure, and toxicological studies performed since the 2006 AQCD have continued to examine the roles of GSTM1 and TNF-α in modifying O_3-related health effects and have examined other gene variants that may also increase risk. Due to small sample sizes, many controlled human exposure studies are limited in their ability to test genes with low frequency minor alleles and therefore, some genes important for O_3-related health effects may not have been examined in these types of studies. A summary of effect measure modification findings from epidemiologic and controlled human exposure studies is included in Table 8-2 of the ISA and from animal toxicology studies in Table 8-3 of the ISA.

Epidemiologic studies that examined the effects of short-term exposure to O_3 on lung function included analyses of potential gene-environment interactions. Romieu et al. (2006) reported an association between O_3 and respiratory symptoms that were larger among children with GSTM1 null or glutathione S-transferase P 1 (GSTP1) Val/Val genotypes compared with children with GSTM1 positive or GSTP1 Ile/Ile or Ile/Val genotypes, respectively. However, results suggested that O_3-associated decreases in lung function may be greater among children with GSTP1 Ile/Ile or Ile/Val compared to GSTP1 Val/Val. Alexeeff et al. (2008) reported greater O_3-related decreases in lung function among GSTP1 Val/Val adults than those with GSTP1 Ile/Ile or GSTP1 Ile/Val genotypes. In addition, they detected greater O_3-associated decreases in lung function for adults with long GT dinucleotide repeats in heme-oxygenase-1 (HMOX1) promoters.

Several controlled human exposure studies have reported that genetic polymorphisms of antioxidant enzymes may modulate pulmonary function and inflammatory responses to O_3 challenge. Healthy carriers of NAD(P)H quinone oxidoreductase 1 (NQO1) wild type (wt) in combination with GSTM1 null genotype had greater decreases in lung function parameters with exposure to O_3 (Bergamaschi et al., 2001). Vagaggini et al. (2010) exposed mild-to-moderate asthmatics to O_3 during moderate exercise. In subjects with NQO1 wt and GSTM1 null, there was no evidence of changes in lung function or inflammatory responses to O_3. Kim et al. (2011) also recently conducted a study among young adults, about half of whom were GSTM1-null and half of whom were GSTM1-sufficient. They detected no difference in the FEV_1 responses to O_3 exposure by GSTM1 genotype and did not examine NQO1. In another study that examined GSTM1 but not NQO1, asthmatic children with GSTM1 null genotype (Romieu et al., 2004) were reported to have greater decreases in lung function in relation to O_3 exposure. Additionally, supplementation with antioxidants (Vitamins C and E) had a slightly more beneficial effect among GSTM1 null children (for more on modification by diet, see section 8.4.1 of the ISA).

In a study of healthy volunteers with GSTM1 sufficient and GSTM1 null genotypes exposed to O_3 with exercise, Alexis et al. (2009) found genotype effects on inflammatory responses but not lung function responses to O_3. At 4 hours post-O_3 exposure, individuals with either GSTM1 genotype had statistically

significant increases in sputum neutrophils with a tendency for a greater increase in GSTM1 sufficient than GSTM1 nulls. At 24 hours postexposure, neutrophils had returned to baseline levels in the GSTM1 sufficient individuals. In the GSTM1 null subjects, neutrophil levels increased from 4 to 24 hours and were significantly greater than both baseline levels and levels at 24 hours in the GSTM1 sufficient individuals. In addition, O_3 exposure increased the expression of the surface marker CD14 in airway neutrophils of GSTM1 null subjects compared with GSTM1 sufficient subjects. CD14 and TLR4 are co-receptors for endotoxin, and signaling through this innate immune pathway has been shown to be important for a number of biological responses to O_3 exposure in toxicological studies (Garantziotis et al., 2010; Hollingsworth et al., 2010; Hollingsworth et al., 2004; Kleeberger et al., 2000). Alexis et al. (2009) also demonstrated decreased numbers of airway macrophages at 4 and 24 hours following O_3 exposure in GSTM1 sufficient subjects. Airway macrophages in GSTM1 null subjects were greater in number and found to have greater oxidative burst and phagocytic capability following O_3 exposure than those of GSTM1 sufficient subjects. Airway macrophages and dendritic cells from GSTM1 null subjects exposed to O_3 expressed higher levels of the surface marker HLA-DR; again suggesting activation of the innate immune system. Since there was no FA control in the Alexis et al. (2009) study, effects of the exposure other than O_3 cannot be ruled out. In general, the findings between these studies are inconsistent. It is possible that different genes may be important for different phenotypes. Additional studies, which include appropriate controls, are needed to clarify the influence of genetic polymorphisms on O_3 responsiveness in humans.

In general, toxicological studies have reported differences in cardiac and respiratory effects after O_3 exposure among different mouse strains, which alludes to differential risk among individuals due to genetic variability (Tankersley et al., 2010; Chuang et al., 2009; Hamade and Tankersley, 2009; Hamade et al., 2008). Thus strains of mice which are prone to or resistant to O_3-induced effects have been used to systematically identify candidate genes that may increase risk of O_3-related health effects. Genome wide linkage analyses have identified quantitative trait loci for O3-induced lung inflammation and hyperpermeability on chromosome 17 (Kleeberger et al., 1997) and chromosome 4 (Kleeberger et al., 2000), respectively, using recombinant inbred strains of mice. More specifically, these studies found that TNF (protein product is the inflammatory cytokine TNF-α) and Tlr4 (protein product is TLR4, involved in endotoxin responses) were candidate susceptibility genes (Kleeberger et al., 2000; Kleeberger et al., 1997). The TNF receptors 1 and 2 have also been found to play a role in injury, inflammation, and airway hyperreactivity in studies of O_3-exposed knockout mice (Cho et al., 2007; Cho et al., 2001) through NF-κB and MAPK/AP-1 (Jnk) signaling pathways (Cho et al., 2007). In addition to Tlr4, other innate immune pattern recognition signaling pathway genes, including Tlr2 and Myd88, appear to be important in responses to O_3, as demonstrated by Williams et al. (2007). A role for the inflammatory cytokine IL-6 has been demonstrated in gene-deficient mice with respect to inflammation and injury, but not AHR (Johnston et al., 2005b; Yu et al., 2002). Other studies have demonstrated a key role for CXCR2, the

chemokine receptor for the neutrophil chemokines KC and MIP-2, (Johnston et al., 2005a) and CD44, the major receptor for the extracellular matrix component hyaluronan (Garantziotis et al., 2009) in O_3-mediated AHR. Mice deficient in IL-10, an anti-inflammatory cytokine, demonstrated increased pulmonary inflammation in response to O_3 exposure (Backus et al., 2010). Thus genes related to innate immune signaling and pro- and anti-inflammatory genes are important for O_3-induced responses.

Altered O_3 responses between mouse strains could be due to genetic variability in nuclear factor erythroid 2-related factor 2 (Nrf-2), suggesting a role for genetic differences in altering the formation of ROS (Hamade et al., 2010). Additionally, some studies have reported O_3-related effects to vary by Inf-1 and Inf-2 quantitative trait loci (Tankersley and Kleeberger, 1994) and a gene coding for Clara cell secretory protein (CCSP) (Broeckaert et al., 2003; Wattiez et al., 2003). Other investigations in inbred mouse strains found that differences in expression of certain proteins, such as CCSP (Broeckaert et al., 2003) and MARCO (Dahl et al., 2007), are responsible for phenotypic characteristics, such as epithelial permeability and scavenging of oxidized lipids, respectively, which confer sensitivity to O_3. Nitric oxide (NO), derived from activated macrophages, is produced upon exposure to O_3 and is thought to participate in lung damage. Mice deficient in the gene for inducible nitric oxide synthase (NOS2/NOSII/iNOS) are partially protected against lung injury (Kleeberger et al., 2001), and it appears that O_3-induced iNOS expression is tied to the TLR4 pathway described above. Similarly, iNOS deficient mice do not produce reactive nitrogen intermediates after O_3 exposure, in contrast to their wild-type counterparts, and also produce less PGE2 comparatively (Fakhrzadeh et al., 2002). These gene-deficient mice were protected from O_3-induced lung injury and inflammation. In contrast, another study using a similar exposure concentration but longer duration of exposure found that iNOS deficient mice were more at risk of O_3-induced lung damage (Kenyon et al., 2002). Therefore, the role of iNOS in mediating the response to O_3 exposure is likely dependent on the exposure concentration and duration.

Voynow et al. (2009) have shown that NQO1 deficient mice, like their human counterparts, are resistant to O_3-induced AHR and inflammation. NQO1 catalyzes the reduction of quinones to hydroquinones, and is capable of both protective detoxification reactions and redox cycling reactions resulting in the generation of reactive oxygen species. Reduced production of inflammatory mediators and cells and blunted AHR were observed in NQO1 null mice after exposure to O_3. These results correlated with those from in vitro experiments in which human bronchial epithelial cells treated with an NQO1 inhibitor exhibited reduced inflammatory responses to exposure to O3. This study may provide biological plausibility for the increased biomarkers of oxidative stress and increased pulmonary function decrements observed in O_3-exposed individuals bearing both the wild-type NQO1 gene and the null GSTM1 gene (Bergamaschi et al., 2001). Deletion of the gene for MMP9 also conferred protection against O_3-induced airways inflammation and injury (Yoon et al., 2007).

The role of TNF-α signaling in O_3-induced responses has been previously established through depletion experiments, but a more recent toxicological study investigated the effects of combined O_3 and PM exposure in transgenic TNF overexpressing mice. Kumarathasan et al. (2005) found that subtle effects of these pollutants were difficult to identify in the midst of the severe pathological changes caused by constitutive TNF-α overexpression. However, there was evidence that TNF transgenic mice were at increased risk of O_3/PM-induced oxidative stress, and they exhibited elevation of a serum creatine kinase after pollutant exposure, which may suggest potential systemic or cardiac related effects. Differential risk of O_3 among inbred strains of animals does not seem to be dose dependent since absorption of 18O in various strains of mice did not correlate with resistance or sensitivity (Vancza et al., 2009).

Defects in DNA repair mechanisms may also confer increased risk of O_3-related health effects. Cockayne syndrome, a rare autosomal recessive disorder in humans, is characterized by UV sensitivity abnormalities, neurological abnormalities, and premature aging. The same genetic defect in mice (Csb-/-) makes them sensitive to oxidative stressors, including O_3. Kooter et al. (2007) demonstrated that Csb-/- mice produced significantly more TNF-α after exposure to O_3 than their wild-type counterparts. However, there were no statistically significant differences in other markers of inflammation or lung injury between the two strains of mice. Overall, for variants in multiple genes there is adequate evidence for involvement in populations being more at-risk than others to the effects of O_3 exposure on health. Controlled human exposure and epidemiologic studies have reported evidence of O_3-related increases in respiratory symptoms or decreases in lung function with variants including GSTM1, GSTP1, HMOX1, and NQO1. NQO1 deficient mice were found to be resistant to O_3-induced AHR and inflammation, providing biological plausibility for results of studies in humans. Additionally, studies of rodents have identified a number of other genes that may affect O3-related health outcomes, including genes related to innate immune signaling and pro- and anti-inflammatory genes, which have not been investigated in human studies.

(9) *Comment:* Commenters also provided comments on individuals with reduced intake of certain nutrients (i.e., vitamins C and E). They noted that the available evidence with regard to the level of protection provided from dietary supplements, the concentrations of O_3 were not relevant to human exposures and that many of the biomarkers were of unknown clinical significance. They suggested that the human and toxicological studies do not support a strong and consistent relationship between nutrient intake and O_3-related health effects.

Response: Diet was not examined as a factor potentially affecting risk in previous O_3 AQCDs, but recent studies have examined modification of the association between O_3 and health effects by dietary factors. Because O_3 mediates some of its toxic effects through oxidative stress, the antioxidant status of an individual is an important factor that may contribute to increased risk of O_3-related health effects.

Supplementation with vitamins C and E has been investigated in a number of studies as a means of inhibiting O_3-mediated damage.

Two epidemiologic studies have examined effect modification by diet and found evidence that certain dietary components are related to the effect O_3 has on respiratory outcomes. In one recent study, the effects of fruit/vegetable intake and Mediterranean diet were examined. Increases in these food patterns, which have been noted for their high vitamins C and E and omega-3 fatty acid content, were positively related to lung function in asthmatic children living in Mexico City, and modified by O_3 exposure (Romieu et al., 2009). Another study examined supplementation of the diets of asthmatic children in Mexico with vitamins C and E (Sienra-Monge et al., 2004). Associations were detected between short-term O_3 exposure and nasal airway inflammation among children in the placebo group but not in those receiving the supplementation.

The epidemiologic evidence is supported by controlled human exposure studies, discussed in section 8.4.1 of the ISA, that have shown that the first line of defense against oxidative stress is antioxidants-rich extracellular lining fluid (ELF) which scavenges free radicals and limit lipid peroxidation. Exposure to O_3 depletes antioxidant levels in nasal ELF probably due to scrubbing of O_3; however, the concentration and the activity of antioxidant enzymes either in ELF or plasma do not appear to be related to O_3 responsiveness. Controlled studies of dietary antioxidant supplementation have demonstrated some protective effects of α-tocopherol (a form of vitamin E) and ascorbate (vitamin C) on spirometric measures of lung function after O_3 exposure but not on the intensity of subjective symptoms and inflammatory responses. Dietary antioxidants have also afforded partial protection to asthmatics by attenuating postexposure bronchial hyperresponsiveness. Toxicological studies discussed in section 8.4.1 of the ISA provide evidence of biological plausibility to the epidemiologic and controlled human exposure studies.

Overall, the ISA concludes adequate evidence is available indicating that individuals with diets lower in vitamins C and E are at risk for O_3-related health effects. The evidence from epidemiologic studies is supported by controlled human exposure and toxicological studies.

(10) *Comment:* Some commenters asserted that the requirement to provide an adequate margin of safety means that NAAQS must be sufficiently stringent to protect everyone. These commenters particularly emphasized the need to set a standard that protects minority children living in the most impacted urban neighborhoods, the elderly and people with compromised lung function.

Response: The EPA agrees that the NAAQS are meant to protect the public health with an adequate margin of safety, including the health of members of at-risk populations. See, e.g., *Coalition of Battery Recyclers v. EPA*, 604 F. 3d 613, 617-18 (D.C. Cir. 2010). We also agree with these commenters that the evidence is adequate to characterize children, the elderly and people with asthma (among

others) as being at increased risk of O_3-related effects (ISA, section 2.5.4.1). As discussed in the HREA (Chapters 5 through 7), the EPA has explicitly assessed O_3 exposures and O_3-related health risks in these populations in urban study areas across the U.S. As discussed in the preamble to the final rule (II.C.4.b and II.C.4.c), based in part on these assessments and on the large body of evidence for O_3-related effects in a variety of populations, the Administrator judges that a revised standard with a level of 70 ppb is requisite to protect public health with an adequate margin of safety. Compared to the current standard, this revised standard is expected to increase public health protection broadly across the U.S., including in both urban and non-urban areas (preamble to the final rule, II.C.4.b and II.C.4.c).

We do not agree that NAAQS must protect every individual. Rather, as discussed in the preamble to the final rule (II.C.4.b), NAAQS must be "requisite" (i.e., "sufficient, but not more than necessary" (*Whitman*, 531 U.S. at 473)) to protect the "public health" ("the health of the public" (*Whitman*, 531 U.S. at 465)). Thus, NAAQS are meant to be neither under- nor over-protective, and to address issues of public health rather than health issues pertaining only to isolated individuals.

(11) *Comment:* A number of commenters agreed with EPA that, based on the evidence reviewed in the ISA and the fact that outdoor workers are exposed to higher levels of ambient ozone than indoor workers, that they may be more vulnerable to O_3.

Response: Studies included in the 2006 AQCD reported that individuals who participate in outdoor activities or work outside to be a population at increased risk based on consistently reported associations between O_3 exposure and respiratory health outcomes in these groups. Outdoor workers are exposed to ambient O_3 concentrations for a greater period of time than individuals who spend their days indoors. As discussed in section 4.7 of the ISA outdoor workers sampled during the work shift had a higher ratio of personal exposure to fixed-site monitor concentrations than health clinic workers who spent most of their time indoors. Additionally, an increase in dose to the lower airways is possible during outdoor exercise due to both increases in the amount of air breathed (i.e., minute ventilation) and a shift from nasal to oronasal breathing. The association between FEV_1 responses to O_3 exposure and minute ventilation is discussed more fully in section 6.2.3.1 of the 2006 AQCD.

Previous studies have shown that increased exposure to O_3 due to outdoor work leads to increased risk of O_3-related health effects, specifically decrements in lung function. The strong evidence from the 2006 AQCD, which demonstrated increased exposure, dose, and ultimately risk of O_3-related health effects in this population, supports the conclusion that there is adequate evidence to indicate that increased exposure to O_3 through outdoor work increases the risk of O_3-related health effects.

(12) *Comment:* As demonstrated in controlled human exposure studies among healthy adults, some people are especially sensitive to O_3 exposures. These people, experience markedly larger decrements in lung function in response to O_3 exposure, and their larger responses are consistent over time. A high level of inter-individual variability is also evidenced in airway inflammation following O_3 exposure. In this review, the EPA recognizes that a certain genetic makeup predisposes some individuals to be especially responsive to O_3 exposures, although other characteristics may also play a role that is not currently clear. The commenter asserts that the standard should be set at levels that will protect these responders, and not merely the population on average.

Response: In this review (discussed in section A.1.b.i above and section II.B.2.b.i in the preamble to the final rule) as in previous reviews, the EPA has recognized that the evidence from controlled human exposure studies to date makes it clear that there is considerable variability in responses across individuals, even in young healthy adult volunteers, and that group mean responses are not representative of more responsive individuals. It is consequently important to look beyond group mean responses to the responses of these individuals to evaluate the potential impact on more responsive members of the population. Thus, the studies of exposures below 80 ppb O_3 show that 10% of young healthy adults experienced FEV_1 decrements > 10% following exposures to 60 ppb O_3, and 19% experienced such decrements following exposures to 72 ppb (under the controlled test conditions involving moderate exertion for 6.6 hours). The EPA recognizes the importance of considering more responsive healthy individuals and in the HREA, PA, proposed and final rule, the proportions of the population predicted to experience one or more >10, 15, and 20% FEV_1 decrement were specifically considered. The Administrator has fully considered these responders in setting a primary O_3 standard that is requisite to protect public health.

c. Comments on Exposure and Risk Assessments

This section discusses major comments on the EPA's quantitative assessments of O_3 exposures and health risks, presented in the HREA and considered in the PA, and the EPA's responses to those comments. The focus in this section is on overarching comments related to the EPA's approach to assessing exposures and risks, and to interpreting the exposure/risk results within the context of the adequacy of the current primary O_3 standard. Comments addressed in the preamble to the final rule are presented first, followed by more detailed and technical comments that were not addressed in the preamble to the final rule. Section II.A.1.c.i discusses comments on estimates of O_3 exposures of concern, section II.A.1.c.ii discusses comments on estimates of the risk of O_3-induced lung function decrements, and section II.A.1.c.iii discusses comments on estimates of the risk of O_3-associated mortality and morbidity, section II.A.1.c.iv discusses comments on the air quality characterization, and section II.A.1.c.v discusses other comments on the exposure and risk assessments.

i. O_3 Exposures of Concern

The EPA received a number of comments expressing divergent views on the estimation of, and interpretation of, O_3 exposures of concern.

(1) *Comment:* In general, comments from industry, business, and some state groups opposed to revising the current primary O_3 standard asserted that the approaches and assumptions that went into the HREA assessment result in overestimates of O_3 exposures. These commenters highlighted several aspects of the assessment, asserting that the HREA overestimates the proportion of the population expected to achieve ventilation rates high enough to experience an exposure of concern; that the use of out-of-date information on activity patterns results in overestimates of the amount of time people spend being active outdoors; and that exposure estimates do not account for the fact that people spend more time indoors on days with bad air quality (i.e., they engage in averting behavior). In contrast, comments from medical, public health, and environmental groups that supported revision of the current standard asserted that the HREA assessment of exposures of concern, and the EPA's interpretation of exposure estimates, understates the potential for O_3 exposures that could cause adverse health effects. These commenters claimed that the EPA's focus on 8-hour exposures understates the O_3 impacts on public health since effects in controlled human exposure studies were shown following 6.6-hour exposures; that the HREA exposure estimates do not capture the most highly exposed populations, such as highly active children and outdoor workers; evaluates an overly narrow subset of health endpoints; is too limited in geographic scope; places too much emphasis on a three-year study period with better air quality than the other period evaluated; and that the EPA's interpretation of estimated exposures of concern impermissibly relies on the assumption that people stay indoors to avoid dangerous air pollution (i.e., that they engage in averting behavior).

Response: In considering these comments, the EPA first notes that as discussed in the HREA, PA, and the proposal, there are aspects of the exposure assessment that, considered by themselves, can result in either overestimates or underestimates of the occurrence of O_3 exposures of concern. Commenters tended to highlight the aspects of the assessment that supported their positions, including aspects that were discussed in the HREA and/or the PA and that were considered by CASAC. In contrast, commenters tended to ignore the aspects of the assessment that did not support their positions. The EPA has carefully described and assessed the significance of the various uncertainties in the exposure analysis (HREA, Table 5-10), noting that, in most instances, the uncertainties could result in either overestimates or underestimates of exposures and that the magnitudes of the impacts on exposure results were either "low," "low to moderate," or "moderate" (HREA, Table 5-10).

Consistent with the characterization of uncertainties in the HREA, PA, and the proposal, the EPA agrees with some, though not all, aspects of these commenters' views. For example, the EPA agrees with the comment by groups opposed to revision that the equivalent ventilation rate (EVR) used to characterize individuals as at moderate or greater exertion in the HREA likely leads to overestimates of

the number of individuals experiencing exposures of concern (HREA, Table 5-10, p. 5-79). In addition, we note that other physiological processes that are incorporated into exposure estimates are also identified in the HREA as likely leading to overestimates of O_3 exposures, based on comparisons with the available scientific literature (HREA, Table 5-10, p. 5-79). These aspects of the exposure assessment are estimated to have either a "moderate" (EVR) or a "low to moderate" (physiological processes) impact on exposure estimates (HREA, Table 5-10, p. 5-79). Focusing on these aspects of the assessment, by themselves, could lead to the conclusion that the HREA overstates the occurrence of O_3 exposures of concern.

However, the EPA notes that there are also aspects of the HREA exposure assessment that, taken by themselves, could lead to the conclusion that the HREA understates the occurrence of O_3 exposures of concern. For example, as noted above, some medical, public health, and environmental groups asserted that the exposure assessment could underestimate O_3 exposures for highly active populations, including outdoor workers and children who spend a large portion of time outdoors during summer. In support of these assertions, commenters highlighted sensitivity analyses conducted in the HREA. However, as noted in the HREA (Table 5-10), this aspect of the assessment is likely to have a "low to moderate" impact on exposure estimates (i.e., a smaller impact than uncertainty associated with the EVR, and similar in magnitude to uncertainties related to physiological processes, as noted above). Therefore, when considered in the context of all of the uncertainties in exposure estimates, it is unlikely that the HREA's approach to using data on activity patterns leads to overall underestimates of O_3 exposures. The implications of this uncertainty are discussed in more detail in the preamble to the final rule (section II.C.4.b), within the context of the Administrator's decision on a revised standard level.

(2) *Comment:* In addition, medical, public health, and environmental groups pointed out that the controlled human exposures studies that provided the basis for health effect benchmarks were conducted in healthy adults, rather than at-risk populations, and these studies evaluated 6.6 hour exposures, rather than the 8-hour exposures evaluated in the HREA exposure analyses. They concluded that adverse effects would occur at lower exposure concentrations in at-risk populations, such as people with asthma, and if people were exposed for 8 hours, rather than 6.6 hours.

Response: In its review of the PA, CASAC clearly recognized these uncertainties, which provided part of the basis for CASAC's advice to consider exposures of concern for the 60 ppb benchmark. For example, when considering the results of the study by Schelegle et al. (2009) for 6.6-hour exposures to an average O_3 concentration of 72 ppb, CASAC judged that if subjects had been exposed for eight hours, the adverse combination of lung function decrements and respiratory symptoms "could have occurred" at lower O_3 exposure concentrations (Frey, 2014b, p. 5). With regard to at-risk populations, CASAC concluded that "based on results for clinical studies of healthy adults, and scientific considerations of differences in

responsiveness of asthmatic children compared to healthy adults, there is scientific support that 60 ppb is an appropriate exposure of concern for asthmatic children" (Frey, 2014b, p. 8). As discussed in the preamble to the final rule (sections II.B.3, II.C.4.b, II.C.4.c of the final rule), based in large part on CASAC advice, the Administrator does consider exposure results for the 60 ppb benchmark and which played a role in the Administrator's determination that it is appropriate to revise the current primary standard (for example, based on the degree a revised standard reduces exposures at the 60 ppb benchmark), and the choice of a level for a revised standard (based on the degree to which a standard at a level of 70 ppb also reduces the number of multiple exposures at the 60 ppb benchmark).

Thus, rather than viewing the potential implications of various aspects of the HREA exposure assessment in isolation, as was done by many commenters, the EPA considers them together, along with other issues and uncertainties related to the interpretation of exposure estimates. As discussed above, CASAC recognized the key uncertainties in exposure estimates, as well as in the interpretation of those estimates in the HREA and PA (Frey, 2014a, c). In its review of the 2nd draft REA, CASAC concluded that "[t]he discussion of uncertainty and variability is comprehensive, appropriately listing the major sources of uncertainty and their potential impacts on the APEX exposure estimates" (Frey, 2014a, p. 6). Even considering these and other uncertainties, CASAC emphasized estimates of O_3 exposures of concern as part of the basis for their recommendations on the primary O_3 NAAQS. In weighing these uncertainties, which can bias exposure results in different directions but tend to have impacts that are similar in magnitude (HREA, Table 5-10), and in light of CASAC's advice based on its review of the HREA and the PA, the EPA continues to conclude that the approach to considering estimated exposures of concern in the HREA, PA, and the proposal reflects an appropriate balance, and provides an appropriate basis for considering the public health protectiveness of the primary O_3 standard.

(3) *Comment:* Commenters on both sides of the issue objected to the EPA's handling of averting behavior in exposure estimates. Some commenters who supported retaining the current standard claimed that the HREA overstates exposures of concern because available time-location-activity data do not account for averting behavior. These commenters noted sensitivity analyses in the HREA that estimated fewer exposures of concern when averting behavior was considered. In contrast, commenters supporting revision of the standard criticized the EPA's estimates of exposures of concern, claiming that the EPA "emphasizes the role of averting behavior, noting that it may result in an overestimation of exposures of concern, and cites this behavior (essentially staying indoors or not exercising) in order to reach what it deems an acceptable level of risk" (e.g., ALA et al.).

Response: The EPA disagrees with both of these comments. In brief, the NAAQS must "be established at a level necessary to protect the health of persons," not the health of persons refraining from normal activity or resorting to medical interventions to ward off adverse effects of poor air quality (S. Rep. No. 11-1196, 91st Cong. 2d Sess. at 10). On the other hand, ignoring normal activity patterns for

a pollutant like O_3, where adverse responses are critically dependent on ventilation rates, will result in a standard which provides more protection than is requisite (see generally section II.C.4.b.iii of the preamble to the final rule).

These commenters also misconstrue the EPA's limited sensitivity analyses on impacts of averting behavior in the HREA. The purpose of the HREA sensitivity analyses was to provide perspective on the potential role of averting behavior in modifying O_3 exposures. These sensitivity analyses were limited to a single urban study area, a 2-day period, and a single air quality adjustment scenario (HREA, section 5.4.3.3). In addition, the approach used in the HREA to simulate averting behavior was itself uncertain, given the lack of actual activity pattern data that explicitly incorporated this type of behavioral response. In light of these important limitations, sensitivity analyses focused on averting behavior were discussed in the proposal within the context of the discussion of uncertainties in the HREA assessment of exposures of concern (II.C.2.b in the proposal) and, contrary to the claims of some commenters, they were not used to support the proposed decision.

(4) *Comment:* Some industry groups also claimed that the time-location-activity diaries used by APEX to estimate exposures are out-of-date, and do not represent activity patterns in the current population. These commenters asserted that the use of out-of-date diary information leads to overestimates in exposures of concern.

Response: This issue was explicitly addressed in the HREA and the EPA disagrees with commenters' conclusions. In particular, diary data was updated in this review to include data from studies published as late as 2010, directly in response to CASAC concerns. In their review of this data, CASAC stated that "[t]he addition of more recent time activity pattern data addresses a concern raised previously by the CASAC concerning how activity pattern information should be brought up to date" (Frey, 2014a, p. 8). As indicated in the HREA (HREA, Appendix 5G, Figures 5G-7 and Figure 5G-8), the majority of diary days used in exposure simulations of children originate from the most recently conducted activity pattern studies (HREA, Table 5-3). In addition, evaluations included in the HREA indicated that there were not major systematic differences in time-location-activity patterns based on information from older diaries versus those collected more recently (HREA, Appendix 5G, Figures 5G-1 and 5G-2). Given all of the above, the EPA does not agree with commenters who claimed that the time-location-activity diaries used by APEX are out-of-date, and result in overestimates of exposures of concern.

In addition to the comments addressed above, and in the preamble to the final rule, EPA received a number of technical comments on O_3 exposures of concern. These comments are addressed below.

Comments on Activity Pattern Data Used by APEX

(5) *Comment*: One commenter expressed concern regarding how children's exposures and risk, particularly during the summer days may be underestimated. More

specifically in a section titled "EPA's Proposed Range of 65 to 70 ppb Undervalues Exposure Risk for Children and Outdoor Workers" is found the comment "[e]stimating exposures for children who spend large portions of time outdoors during the summer when school is traditionally out of session, EPA found that exposures of concern could be underestimated by 33%" and also "During the summer, many children spend significant amounts of time outdoors and being active at camps and at play. Summertime is also when ozone levels are highest, thus putting children at greater risk" (e.g., ALA, et al.).

Response: EPA generally agrees with the commenter regarding how children face a greater potential risk than most other study groups considered in the HREA. See response in section II.A.b.iii above. However, EPA generally disagrees with the commenter's interpretation of the APEX sensitivity analysis performed in the HREA and the results cited that "exposures of concern could be underestimated by 33%". The limited sensitivity analysis informing this O_3 HREA reported result (a single study area (Detroit) for year 2007) was designed to investigate a hypothetical alternative exposure scenario to estimate school-age (5-18) children's exposures "assuming all children were on a traditional calendar year summer vacation" (HREA, Appendix 5G, pp. 5G-29 to 5G-30). Additional clarity is added below regarding the overall exposure scenario objectives for the identified APEX exposure simulation, the input data used, and the broad context for interpreting the exposure results related to this particular hypothetical exposure scenario.

The activity pattern data input to APEX for this "traditional summer vacation" scenario were derived as a subset of the complete Consolidated Human Activity Database (CHAD) data set used by APEX in the main body HREA simulations to reflect only diary data from school-age children that did not have any time spent indoors while at school and those that did not perform a paid work activity for that day. Exposure results generated using this subset of CHAD diaries were compared with exposures generated using all available CHAD diaries (i.e., the data set used to generate the main body HREA results). The comparison of exposures generated from these two simulations, and as reported in the HREA, "suggests that, for urban study areas having a traditional school calendar (i.e., school not in session during the months of June, July and August), exposures at or above selected benchmark levels could be underestimated by about 33%" (HREA, p. 5-49). The key terms stressed in our discussion of the results presented in the O_3 HREA and that follows here when interpreting the scenario results are "suggests" and "could be".

The exposure scenario for the sensitivity analysis was designed to evaluate the effect of the diary data subset used for the purposes described above and, by its design, represents a potential upper bounding for exposures when assuming all simulated individuals in the study area behave in a prescriptive, rigid, and perhaps unusual manner (i.e., no child in the exposure simulation performs paid work, no child in the exposure simulation spends any time indoors at a school). More specifically, the approach used to estimate exposures for this particular scenario

subsumed a degree of conservatism biased towards estimating high exposures that was not taken into account by the commenter when interpreting the entirety of the HREA main body exposure results. For example:

1. In this limited exposure simulation, 2007 was Detroit's worst (highest) O_3 concentration year in the collection of years of air quality evaluated in the HREA. All other years of air quality for Detroit would have substantially fewer children exposed, considering any hypothetical scenario. That said, the difference in the number (not percent) of children exposed would be of greater practical importance when evaluating any lower ambient concentration scenario (i.e., lower ambient concentration years and lower alternative standard levels). For instance, the percent difference cited above of 33% was approximated using the information from REA Figure 5-13; whereas about 15% and 20% of school-age children were estimated to be exposed at least once to an 8-hr average O_3 concentration for air quality that just meets the existing standard, and using the main body HREA simulation approach and the "traditional summer vacation" simulation, respectively (i.e., [20-15]/15×100=33%). Using the actual number of simulated individuals in this comparison to similarly calculate this percent difference of course remains as 33% ([200,695-150,665]/150,665×100), while the difference in the number of persons exposed between the two approaches is about 50,000. When considering 2007 air quality just meeting a standard level of 70 ppb and the same 60 ppb benchmark (REA Figure 5-13), the percent difference between the two approaches may be greater (11% exposed vs 7.8% exposed, respectively, or a percent difference of 41%), but the difference in the number of persons is much less (about 110,000 exposed vs 80,000 exposed, respectively, or a difference of 30,000). However, these potential (upper bound) numbers decrease substantially under different assumptions, as discussed below.

2. Not accounted for by the "traditional summer vacation" simulation is that approximately 50% of children ≥16 years old are estimated to perform paid work during summer months,[48] and mostly for retail and hospitality services,[49] jobs that typically take place indoors. In our simulations of school-age children (ages 5-18), children ages 16-18 comprise approximately 20% of the total study group population, and assuming they equivalently comprised those exposed at or above the 60 ppb benchmark level and that about half performed paid work activities indoors, the percent difference between the two approaches using the same 75 ppb air quality standard level described above would be reduced to about 20% ([200,695×.9-150,665]/150,665*100), while the difference in the number of persons exposed would be reduced to about 40,000 for this particular scenario.

[48] http://www.bls.gov/news.release/youth.t01.htm
[49] http://www.bls.gov/news.release/youth.t03.htm

3. Our "traditional summer vacation" simulation does not include an adjustment for when potential "summer camp" activities do not occur outdoors (i.e., academic activities are common and typically occur indoors), a likelihood of occurring even considering those programs focusing largely on outdoor recreation.[50] It is entirely possible that a CHAD survey participant attended an outdoor recreational camp and recorded an academic activity occurring "at school" rather than at a camp building, excluding that particular diary from being used by APEX in this simulation, and thus not being represented in exposure results. Further, many of these "camps" or other types of summer instructional services have summer activities occurring largely indoors at schools (not at outdoor parks), particularly in urban areas such as Detroit.[51] In our "traditional summer vacation" simulations, absolutely no children spent any time indoors at a school, thus selecting for a unique and smaller subgroup of simulated individuals that would be among those experiencing the highest O_3 exposures.

4. The "traditional summer vacation" simulation comparison does not account for the proportion of students that may attend year-round schools (the actual number of which is largely uncertain though evidence exists for continued expansion),[52] those having other potential alternative school schedules, or children attending school during the summer months to avoid retainment. Not accounting for these individuals in our "traditional summer vacation" simulations also selects for a reduced subgroup of simulated individuals that could be highly exposed to O_3.

5. The three months of the "traditional summer vacation" simulation (June, July, August) were chosen generally out of modeling convenience and do not directly correspond the actual days of the Detroit[53] traditional school summer vacation (or likely other study areas). In considering the actual schedule for Detroit public schools, there were a greater number of days simulated for June (21 days), fewer days simulated for September (13 days) than actual, an amount when combined shows overall the simulated "traditional summer vacation" was longer than the actual schedule by 8 days, possibly leading to a greater number of estimated exposure events.

Considering these additional factors, the commenter's suggestion that children's exposures could underestimated by 33% is particularly conditioned to the constructed simulation, the exposure scenario is likely only applicable to a small fraction of children comprising the population in any study area, and not meant to quantitatively relate directly to the main body HREA results.

[50] http://find.acacamps.org/
[51] http://detroitk12.org/content/summer-academy/
[52] http://nayre.org/YRE%20Schools%20on%20the%20Web.html
[53] http://www.nctq.org/docs/22-05_7093.pdf

(6) *Comment*: One commenter expressed concern regarding the potential for averting behavior that may already be expressed in the activity diary data that were used to estimate exposures to underestimate the health risks. Specifically, "[a]lthough, in the HREA, EPA recognizes evidence that many people, including children, avert outdoor activity and that the exposure estimates may be too high due to averting behavior, it is also unclear whether there is double-counting of the impact of averting behavior. In a footnote, EPA notes that "we do not know if any diary day represents the activities of an individual who averted. Thus it is entirely possible that the 'no averting' simulation includes, to an unknown extent, individuals who spent less time outdoors than would have occurred if absolutely no individuals averted." In other words, EPA does not know if the activity data it is using already includes double-counting averting behavior, but chooses to assume it does not in their basis for calculating exposures of concern and, consequently, reduces its estimates of risks" (e.g., ALA et al.).

Response: EPA generally disagrees with the commenter's interpretation of the activity pattern data used by APEX in simulating population-based study groups. In general, the comment pertains to the commenter's interpretation of activity data used to perform a limited sensitivity analysis (HREA, Appendix 5G, pp. 5G-39 to 5G-44) and the associated simulated exposure results (HREA, section 5.4.3.3, pp. 5-53 to 5-54). The specific concern by the commenter regards information provided in footnote 27 of the HREA and is related to a limited sensitivity analysis conducted to approximate the impact averting behavior (e.g., people actively reducing time spent outdoors to avoid air pollution events) has on estimated exposures. HREA footnote 27 states, "because most activity diaries are limited to a single day and the survey participants were not directly asked if they altered their daily activities in response to a high air pollution event, we do not know if any diary day represents the activities of an individual who averted. Thus it is entirely possible that the 'no averting' simulation includes, to an unknown extent, individuals who spent less time outdoors than would have occurred if absolutely no individuals averted." (HREA, p. 5-33). Additional clarity is provided below regarding the objective of this hypothetical scenario, the development of the input data used to estimate exposures for the sensitivity analysis, the overall representativeness of the CHAD data base, and context regarding the hypothetical scenario results.

The particular exposure simulation performed and summarized in HREA section 5.4.3.3 was designed to estimate the potential impact of reducing time spent outdoors by a fraction of the study population on high O_3 concentration days. Three APEX simulations were performed, identical in all model inputs and settings except differing by the activity pattern data used to represent time spent outdoors. To design reasonable parameters to inform this sensitivity analysis, the available averting behavior literature was first reviewed and an amount of outdoor time reduced (20-40 minutes) and fraction of the population participating in averting (15% and 30% for general population and people with asthma, respectively) were approximated based on analysis of the reviewed published studies (Graham, 2012). This information was then used to inform a manual

adjustment of the CHAD diary data used by APEX to reflect such a reduction in afternoon time spent outdoors and the fraction of the population participating in outdoor activities. The upper bound on the proposed reduction in average time spent outdoors (~40 minutes) was selected as a conservative target to simulate the averting by each study group considered in this hypothetical exposure scenario.

Thus, three identical sets of school-age children's diaries were used, differing only by the fraction of the population having a reduction in their time spent outdoors (i.e., one set of diaries targeted to have no change, the second having 15% of all school-age children reduce time by 40 minutes, and the third having 30% of school-age asthmatic children reduce time by 40 minutes). Following the three simulations in APEX, the afternoon time spent outdoors was evaluated to determine how closely the manually adjusted data, and hence the simulated population in for each study group, came to the target rates of averting and reduction in afternoon time spent outdoors. Of course and by design, the simulation using the collection of unadjusted diaries had no reduction in time spent outdoors. The two simulations having CHAD diaries manually adjusted to reflect averting in the two study groups of interest generally met the planned, hypothetical averting targets for the simulated population (i.e., whereas 15.3% of all school-age children and 30.4% or children with asthma, each and on average, spent 44 minutes less afternoon time outdoors – see HREA, pp. 5-53 and 5-54). Therefore in actuality, this hypothetical scenario reflects the potential impact to the number of benchmark exposures according to these resulting simulated averting rates and reduced afternoon time spent outdoors.

The selection of air quality data used in the hypothetical averting scenario is also an important consideration in the interpretation of results. Unadjusted (base) air quality for a two day period (August 1-2, 2007) in one study area (Detroit) was specifically selected for this sensitivity analysis because that two-day period had some of the highest ambient O_3 concentrations and number of people exposed in that study area for that year (HREA, Appendix 5G, p. 5G-42). Again, the intent was to see the potential impact of simulated averting when there was an actual high concentration exposure event, and as such, the three simulations used these specifically selected high ambient O_3 conditions (i.e., and possibly above those that would exist when just meeting the existing standard or lower alternative standards).

In any APEX simulation, the CHAD diaries are used "as is" or unadjusted in APEX to reflect actual variability in human activities, though considering their most important influential personal attributes (e.g., age, sex) and other variables (e.g., daily temperature) affecting what people do and where they go in developing the individual and longitudinal profiles for the simulated population (Graham and McCurdy, 2004). There is no direct information available in CHAD or otherwise to determine whether the surveyed person actively reduced their outdoor time on the surveyed day beyond that considered typical for that particular person. For example, it is possible the activity pattern study participant reduced their time spent outdoors unusually, when compared with what may be

considered as their typical daily activities, because of an air pollution event, or for other more likely reasons such as an illness, extreme outdoor temperatures, a rain event, etc. Similarly, there is no direct information in CHAD or the original study data set to determine if the activity pattern survey participant actually spent more time outdoors than they would have usually spent on the recorded day (e.g., more time spent outdoors attending or participating in a unique outdoor sporting event). The fundamental assumption in using the CHAD data "as is" is that the diary day reflects the typical activities that might be observed for that person and that the collection of diaries used in our modeling appropriately reflects the activities performed by the simulated population in the exposure study area (with appropriate adjustments made by APEX in accounting for age/sex distributions in a census tract, having corresponding daily temperatures between the diary day selected and the simulated individual, appropriate matching of weekday/weekend day-types, and so on).

That said, it is entirely possible that for as many individuals that may have actually and atypically spent less time outdoors on that diary day, there could also be a similar number of individuals that had actually and atypically spent more time outdoors on their recorded diary day. In the targeted exposure simulation to evaluate averting in the HREA, the actual degree of existing averting (and time outdoors atypically above that considered usual) that may or may not have occurred by CHAD activity pattern survey participants within the baseline simulations is not a factor of concern in the scenario, as we are observing the impact of averting by a specified fraction of the population for a specified mean amount of time spent outdoors. It is also not particularly concerning regarding any exposure model simulation that uses CHAD "as is", because the CHAD diaries (past and present) are largely from studies that randomly selected study subjects, some of which were nationally representative, and collectively, likely represent the places people visit and the activities performed throughout a typical day.

Further, context regarding the duration of the hypothetical scenario is also an important consideration in interpreting the exposure results. Results of this sensitivity analysis indicated that for the two-day simulation period, a one to two percentage point or fewer school-age children (without asthma and children with asthma, respectively) experienced exposures at or above any of the selected benchmark levels when compared with the baseline scenario (HREA, Figure 5-15). Note also, these results likely do not directly correlate with the estimated number or percent of children exposed for an entire O_3 season. Averting one or two days of the year does not automatically extend to behavior realized over longer periods, particularly given the potential for desensitization by the number of air quality alerts, sequentially occurring (Zivin and Neidell, 2009), or perhaps that could occur in total over an entire O_3 season. Thus, it is entirely possible that the approximated reduction in the percent of school-age children exposed considering this limited 2-day averting scenario could be an overestimate of what might actually occur across an entire O_3 season and in actuality, would likely approach the range of exposures generated for the main body HREA.

(7) *Comment*: A few commenters suggested that by not accounting for averting behavior, exposures and health risks might be overestimated. Specifically, "[t]he APEX model runs also included a number of assumptions regarding the activities of the simulated individuals. First, the APEX model did not consider averting behavior in children. Sensitivity analyses conducted for one high-ozone period in Detroit found that 8-hour ozone exposures decreased up to 30% when averting behavior was considered (HREA, Figure 5-15)" (e.g., API).

"Further, the results presented in the Proposed Rule likely overestimate ozone exposure and risk because they focus on children with "moderate or greater exertion level at the time of exposure" who do not exhibit averting behavior (i.e., staying indoors when there are high ozone concentrations) (HREA, p. 5-2)" (e.g., Gradient).

"Aversion or avoidance behavior within the potentially exposed population, especially sensitive subgroups, also factors into the over-estimation of total population ozone exposure. This could be especially significant in larger urban areas where ozone action days are widely advertised in the local media and educational systems. This fundamental over-estimation of true ozone exposure complicates the ability to discern subtle distinctions from these epidemiologic studies that discount any support for a reduced ozone standard" (e.g., OH EPA).

Response: EPA disagrees with the commenters' interpretation of the exposure results generated from the HREA's hypothetical simulation of averting. Much of what has been described in EPA's response above noting the limited nature of the sensitivity analysis performed in the HREA (section 5.4.3.3) applies to this comment as well. EPA adds that while time spent outdoors is an important variable in understanding how individuals are exposed to high concentrations, the hypothetical averting investigation performed in the HREA was limited in scope (a single study area using high concentration (base) air quality for a two-day period) and designed to estimate the impact on exposure, of having some known fraction of the simulated population reduce their afternoon time spent outdoors by some known amount during a high ambient concentration event. The limited hypothetical exposure scenario did not take into account other additional factors perhaps important in further evaluating this scenario (e.g., the occurrence of multiple high concentration events over the entire O_3 season and its relationship with longitudinal averting behavior), nor were the results meant to serve as a prediction of how averting time spent outdoors may comprehensively impact O_3 exposures in the future.

EPA agrees with the commenter that averting behavior can impact the interpretation epidemiologic study information, particularly "the potential negative impact it could have on O_3 concentration-response (CR) functions used to estimate health risk" (HREA, section 5.4.3.3). To clarify this statement here, existing concentration-response functions, if developed during study periods when study participants avoided outdoor time due to high ambient pollution

events, would tend to underestimate actual health risk if used in a quantitative assessment and presumed that people would freely choose to spend time outdoors.

We also reiterate that, the NAAQS must "be established at a level necessary to protect the health of persons," not the health of persons refraining from normal activity or resorting to medical interventions to prevent or abate adverse effects of poor air quality (S. Rep. No. 11-1196, 91st Cong. 2d Sess. at 10). See also ATS (2000) discussion of adversity, noting that changes in normal behavior patterns in response to exposure to the etiologic agent is a quality of life change which can be considered to be an adverse health effect.

(8) *Comment:* A few commenters attempted to link statistics regarding time spent indoors alone as an indicator of reduced exposure. Specifically, "EPA should explain the limitations of setting standard for ambient air based on clinical exposures when HREA states that most people spend the majority of their time indoors (see quote from CASAC above)" (e.g., OSIPC).

"A majority of Americans spend most of their hours indoors. Children and senior citizens spend on average less than 10% of their time outdoors (ISA, page 4-31). Therefore, true average individual ozone exposure measures substantially less than presumed by outdoor air quality monitoring" (e.g., OH EPA).

"Ozone is primarily an outdoor pollutant with ventilation and indoor structures scavenging it and removing it from indoor air. The average American adult, senior citizen, and child will spend only 5.3%, 5.8%, and 7.9% of their time outdoors, respectively (ISA, page 4-31), and therefore they will often not be exposed to ozone" (e.g., TCEQ).

Response: EPA agrees with the commenters in that most people spend the majority of their time indoors (e.g., see Graham and McCurdy, 2004). However, EPA emphasizes that individuals experiencing O_3 exposures at or above health effect benchmark levels are those that spend a significant amount of time outdoors (HREA, Appendix 5G, section 5G-2). While this study group may not constitute a majority of the population *per se*, they do represent a significant number and percent of the overall population exposed to the highest O_3 concentrations (e.g., HREA, Figures 5-5 through 5-11). By law, the NAAQS must afford requisite protection, with an adequate margin of safety, to such groups. See, e.g. *Coalition of Battery Recyclers v. EPA*, 604 F. 3d at 617-18. Children are among these groups.

Further, when estimating O_3 exposures for simulated individuals, APEX appropriately accounts for air exchange rates and expected O_3 decay that occurs within indoor microenvironments (HREA, Appendix 5B, section 5B-6.6). Indoor microenvironmental O_3 concentrations estimated by APEX are much less than those occurring in outdoor microenvironments (e.g., HREA, Appendix A, Figure 5A-2). We agree with the commenter that when time-averaged O_3 exposures

include times-of-day when an individual was indoors, estimated O_3 exposures will be lower than that measured by outdoor air quality monitoring.

(9) *Comment:* One commenter expressed concern regarding the year activity pattern survey data were obtained and its potential influence to activities performed. Specifically, "APEX uses time-activity diaries from the 1980s through the 2000s. EPA compared the ranges of time that children spent outdoors during the 1980s, 1990s, and 2000s in the time-activity diary data used in APEX and found that the range decreased over time. EPA assumed that this decrease would not impact the model results, because there was a large degree of uncertainty in the calculated ranges (HREA, Appendix 5-G), but this assumption was not rigorously tested using the APEX model" (e.g., API).

Response: In general, we disagree with the commenter's interpretation of the activity pattern data and the informative evaluations performed in the O_3 HREA. CHAD is the most comprehensive collection of activity pattern data available for appropriately modeling short-term human exposures. The CHAD database used in the HREA's exposure assessment contains over 53,000 individual daily diaries including time-location-activity patterns for individuals of both sexes across a wide range of ages (HREA, Chapter 5). The CHAD data are an accurate record of the locations where actual surveyed individuals visited (e.g., outdoors at school) and their activities performed (e.g., play sports). While CHAD was originally developed a few decades ago, EPA has continuously increased the number of diaries in CHAD since its inception, making noteworthy increases to the database specifically for this review, partly in response to CASAC requests to do so.[54] EPA performed an analysis in the HREA section 5.4.1.3 indicating that there is little difference in the key exposure variable, time spent outdoors, when comparing the CHAD data set across three decades (1980's, 1990's, 2000's). While we did not perform a direct sensitivity analysis of how the older CHAD diary data might affect exposure results generated by APEX, the need to perform such a simulation was minimized by analyses provided in HREA, Appendix G, Figures 5G-7 and Figure 5G-8, showing that the overwhelming majority of diary days used in the simulations of school-age children originate from the most recently conducted Institute for Social Research (ISR; 1997-2008) and Ozone Averting Behavior (OAB; 2002-2003) activity pattern studies (HREA, Table 5-3) and not activity patterns recorded from the older CHAD studies.

(10) *Comment:* A few commenters suggested that the time spent outdoors modeled by APEX was greater than that expected. Specifically, "In the ozone ISA (Table 4-4), EPA noted that the National Human Activity Pattern survey data (NHAPS) show that the average time a 5- to 17-year-old spends outdoors is 7.88% of the day, or 1.9 hours. We would expect that many of the APEX-simulated children

[54] CASAC "commend[ed] the EPA for substantial revision to the first Draft HREA based on its prior advice…and notes tremendous improvement in the Second Draft HREA. Overall, the document is well-written, founded based upon comprehensive analyses and adequate for its intended purpose" (Frey 2014a, p. 1).

experiencing FEV_1 decrements > 10% would spend more than the average amount of outdoors, since there is a greater probability of high ozone exposures outdoors than indoors. However, API Figure II.1[55] demonstrates that a number of the modeled children spent an unrealistically high amount of time outdoors (e.g., up to 24 hours), which suggests that the model does not always accurately simulate the daily activity patterns of children and the exposures that result from these activities" (e.g., API).

"[S]ome of the children with FEV_1 decrements greater than 10% were simulated by APEX to spend a large percent of the day outdoors (e.g., up to 24 hours per day on the days when they experienced the lung function decrements). This can be contrasted with the National Human Activity Pattern survey data that show that 5- to 17-year-olds, on average, spend 7.88%, or 1.9 hours, of their day outside. These data demonstrate that not all of the children in the APEX model are realistically simulated, and that the number of simulated children with FEV_1 decrements greater than 10% is likely overestimated (e.g., Gradient).

Response: We disagree with the commenter's interpretation of activity pattern data and their conclusions drawn regarding what is an "unrealistic" amount of time spent outdoors for children or any study group modeled by APEX. The CHAD is the most comprehensive collection of activity pattern data available for appropriately modeling short-term human inhalation exposures. The CHAD data are an accurate record of the locations where actual surveyed individuals visited (e.g., outdoors at school) and their activities performed (e.g., play sports). The CHAD data are used in APEX "as is" or unadjusted from when the activity pattern information was originally collected from the surveyed individuals. There are many diary days in CHAD where study participants had absolutely no time spent outdoors (i.e., 0 minutes) and an extremely limited number of diary days where survey participants spent the entire day (i.e., 24 hours) outdoors, while other diary days fall somewhere in between. This is the nature of human activity patterns and the data recorded to represent them.

Comparing time budgets of select individual diary days used by APEX for when children experienced FEV decrements to aggregated, time-averaged diary data data (i.e., all of NHAPS data) as was done by the commenters is inappropriate. Graham (2015) performed a more appropriate comparison of time expenditure using all of the CHAD diaries for children ages 5-17 used by APEX and compared this with the NHAPS reported mean time spent outdoors. Considering the full set of children's diaries age 5-17 in CHAD and used by APEX, less time is spent outdoors on average (i.e., mean = 1.4 hours, standard deviation = 2.1 hours, Table 1 of Graham, 2015) than when compared with the NHAPS data alone (mean = 1.9 hours, reported by the commenter). Note also that the NHAPS

[55] The commenter performed APEX simulations and "reproduced EPA's APEX model simulation for the base air quality in Los Angeles in 2006, and further analyzed the profiles of the simulated individuals who experienced FEV1 decrements > 10% and > 15%. This simulation was based on the same input files and parameters as the simulation reported in the HREA."

activity pattern survey data contribute to a large portion of the diary data included in CHAD and are used by APEX in estimating exposures (~20% of all CHAD diaries, see Table 5-3, HREA).

Further, often overlooked when performing time-averaged comparisons or when focusing only on maximum values is whether the individual activity survey participants spent any or little time outdoors and what proportion of the population these people comprise (i.e., considering the full distribution of time expended for the population). Graham (2015) Table 1 shows that approximately 45% of CHAD diary days for children ages 5-17 have 0 minutes of outdoor time, just over half did not spend greater than 30 minutes outdoors, with nearly 60 percent having less than an hour outdoors. Regarding time spent outdoors for an entire 24 hour period, a concern expressed by the commenter, there were only four diary days that were used by APEX having that amount of time spent outdoors, comprising 0.025% of the total number of available CHAD diaries for children ages 5-17 (Table 1 of Graham, 2015). The location information recorded on these four diary days indicated that most of these surveyed children spent time outdoors at a park or by a pool/river/lake. It is certainly reasonable and entirely not "unrealistic" that when these diary days were initially recorded by the study participants, the children surveyed were participating in an outdoor camping event, as some of the recorded activities include hiking/fishing or other recreational sports. Further, given that these are recorded events from actual children, it is certainly not at all "unrealistic" to also include these diaries having time spent outdoors as long as 24 hours in the APEX exposure simulations.

Thus, not only are the CHAD diary data accurate representations of what actual people do and appropriately indicate the locations that people visit and the amount of time associated with these events, the meant time spent outdoors for CHAD survey participants is also consistent with the NHAPS data statistics cited by the commenter. Moreover, the commenter's approach of highlighting group means alone masks the importance of considering observed interindividual variability in daily time expenditure and associated pollutant exposures – in this case, disregarding children spending longer amounts of time outdoors than the average and that are likely to experience the highest O_3 exposures.[56] The Clean Air Act does not deny requisite protection to children attending summer camps, to those children playing outdoors for multiple hours, or to those who go camping.

(11) *Comment:* One commenter suggested that the influence of age of individuals and historical trends have on physical activity level was not accounted for in the exposure analysis. Specifically, "studies have reported a significant decline in physical activity from childhood to adolescence, and the rate of decline has

[56] Similarly, as pointed out in section II.B.2.c.i in the preamble to the final rule and II.A.1.b.i.(b) in this document, criticisms of controlled human exposure studies based on group mean levels in those studies served to obscure the effects demonstrated to occur in more susceptible study members.

increased in recent years (Dumith et al., 2011), but this was not accounted for in the analysis" (e.g., API).

Response: EPA disagrees with the commenter's interpretation of how physical activity data (and the CHAD diaries themselves) are used by the APEX model in estimating exposures for the HREA. EPA certainly acknowledges that there is variability in activity patterns that is dependent on age, such as time spent outdoors and associated activities performed (e.g., Graham and McCurdy, 2004; U.S. EPA, 2011). This is reason why the APEX model assigns age-specific diaries from CHAD to simulate age-specific individuals' locations visited and activities performed and thus accounts for when time expenditure varies due to influential personal attributes such as age. In short, EPA does account for the "decline in physical activity from childhood to adolescence" and, in largely using the most recent CHAD data[57] to simulate children's activities in the HREA, does account for the proposed "decline [that] has increased in recent years", where such a rate of decline does exist in the recorded diary data for the activity pattern study participants.

Comments on APEX Estimated Ventilation Rates

(12) *Comment:* A few commenters suggested that APEX overestimated ventilation rates. One commenter suggested "the APEX model predicts more elevated ventilation rate occurrences than observed in real world data. In the previous review, Langstaff acknowledged that the 'values produced by the ventilation rate algorithm may exhibit an excessive degree of variability' [(Langstaff, 2007)]. An excessive degree of variability will produce an excessive number of extreme values of ventilation rate. The 1997 EPA analysis had also over-estimated the number of high ventilation rates in the population by using an algorithm to assign ventilation rates based on individuals who exercised regularly and were motivated to reach a high ventilation rate. As a result, the 1996 Staff Paper acknowledged that the analysis allowed more high ventilation rates (hence greater risk) than would actually occur in the populations of interest - outdoor workers, outdoor children, etc. The final HREA includes a comparison of predicted ventilation rates with mean values in the literature, but the upper tails of the distribution which impact the risk estimates were not compared. This was an important oversight because the upper percentiles of ventilation rate are responsible for the exposures that cause the perceived risk." (e.g., AAM; U.S. Chamber of Commerce).

"EPA acknowledges that the ventilation rates used in the APEX model can be greater than published measurements by 2-3 m^3/day (HREA), but does not

[57] HREA, Appendix G, Figures 5G-7 and Figure 5G-8 show that the overwhelming majority of diary days used in the simulations of school-age children originate from the most recently conducted Institute for Social Research (ISR; 1997-2008) and Ozone Averting Behavior (OAB; 2002-2003) activity pattern studies (HREA, Table 5-3)

acknowledge that this contributes to overestimated exposures and lung function decrement risks" (e.g., API).

"In addition, according to the HREA (p. 5-64), APEX ventilation rates can be overstated by 2-3 m^3/day, which is a significant overestimation in comparison to typically assumed daily inhalation rates of 20 m^3/day (i.e. 10-15%)" (e.g., TPA).

Response: EPA disagrees with the commenters' use of information provided in former and recent O_3 NAAQS-related exposure assessments and the conclusions drawn by the commenters regarding the current APEX modeling approach used to estimate ventilation rates. A few corrections to the commenter's statement are first needed here. Regarding the reference to an assessment performed for the 2007 O_3 NAAQS review, EPA specifically notes here that the statement regarding 'excessive variability' was not directly made by Langstaff (2007) but actually refers to a statement made by Johnson (2002, 2003) pertaining to estimating metabolic equivalents of work (METs) that would lead to "VO_2 [*oxygen consumption*] values which exceed limits based on activity duration and the physiological characteristics of the cohort" (Johnson 2002). A commenter's mention that "the 1997 EPA analysis had also over-estimated the number of high ventilation rates in the population" actually refers to a statement made in the 1996 O_3 exposure assessment (Johnson et al., 1996) regarding the approach used to estimate ventilation rates at that time by the probabilistic NAAQS exposure model (pNEM), where eight lognormal distributions[58] of equivalent ventilation rate (EVR) were randomly sampled to estimate breathing in simulated exposure cohorts (i.e., *not individuals* as is done currently by APEX). Johnson et al. (1996) states, "[c]onsequently, the EVR limiting algorithm may permit more high EVR values to occur in the pNEM/O_3 simulation than would occur in the actual population. This potential bias may be corrected in future versions of pNEM/O_3 by distinguishing cohorts by gender, age, and physical conditioning" (see Johnson et al., 1996).

Assessments of older approaches used to estimate ventilation in earlier NAAQS reviews are not relevant to evaluating the ventilation approach used in the current APEX model. EPA has since specifically addressed the above identified issue of controlling for physiologically unusual VO_2 by appropriately modeling fatigue in APEX simulated individuals that could occur with sequentially-repeated high-exertion activities, while also accounting for *increased* ventilation that is expected to occur following completion of high-exertion activities (i.e., excess post exercise oxygen consumption, EPOC) (see Isaacs et al., 2008; U.S. EPA, 2012a; U.S. EPA, 2012b). Furthermore, both the 1996 EPA exposure assessment (Johnson et al., 1996) and Johnson (2002, 2003) used/assessed a different algorithm used by APEX to estimate ventilation rates, an algorithm that has since been updated and currently accounts for important influential variables such as age, sex, and body mass (Graham and McCurdy, 2005). While the Johnson (2002,

[58] Each distribution is specific to an age group (children or adults) and breathing rate category (sleeping, slow, medium, or fast) and derived from a limited study using 36 participants aged 10 to 50 years old.

2003) algorithm used an individual-based activity-specific ventilation approach much like the approach used for the HREA, Graham and McCurdy (2005) expanded the clinical data set originally used in Johnson (2002, 2003) by approximately 600 measurement data points (now comprising 6,284 observations) and extended the algorithm to improve the estimation of ventilation rates in children less than 18 years old. Therefore, any comments made that are supported by an assessment of obsolete modeling approaches, and which pertain to older, more limited data sets, are not relevant to the current ventilation algorithm used by APEX.

EPA also disagrees with the commenters' interpretation of available literature-reported ventilation rates including how they compare to APEX estimated ventilation rates. In their evaluation, the commenter emphasizes data reported in Table 25 of Langstaff (2007) that compares mean (± sd) daily ventilation rates estimated by APEX with ventilation rates estimated by Brochu et al. (2006). The commenter concluded that, based on the results presented in these two selected sources of information and because APEX simulated daily mean (and standard deviation) ventilation rates were higher than Brochu et al. (2006), the APEX results are over-estimated. Interestingly, in their comment, the commenter references the first part of a statement provided in Table 5-10 of the HREA that begins with "the APEX estimated daily ventilation rates can be greater (2-3 m^3/day) than literature reported measurement values (Table 25 of Langstaff, 2007)," but then fails to acknowledge the latter and more important portion of the sentence "though if accounting for measurement bias this minimizes the discrepancy (Graham and McCurdy, 2005; see Figure 5-23 and Figure 5-24)" (see HREA). Additional clarity is provided below regarding the Brochu et al. (2006) ventilation data set and the Langstaff (2007) reported results for APEX ventilation rates, then followed with additional analysis to respond to the above comments.

The Brochu et al. (2006) data were based on doubly-labeled water (DLW) consumption/elimination to estimate energy expenditure in healthy normal-weight males and females over a 7-day to 21-day period (Brochu et al., 2006). This extended period used to estimate energy expenditure is standard with this approach, resulting from the several hours it takes for the administered ^{18}O and ^{2}H isotopes to distribute throughout the bodies of study subjects and the daily measurements needed to evaluate the progressive decrease in isotope concentrations over time (McArdle et al., 2001). Thus, within person day-to-day variability in ventilation rates that is expected to occur is not accounted for by this method, immediately unrealistically constraining variability in the reported results, even when considering these daily mean values reported by Brochu et al. (2006).

More so ,however, the principal issue commonly ignored when evaluating the Brochu et al. (2006) data, but stressed in the HREA (section 5.4.4.2) and the Graham (2009) evaluation of the Brochu et al. (2006) reported data, is that the Brochu et al. (2006) study did not directly measure ventilation rates. Ventilation rates in the Brochu et al. (2006) study is a calculated value, an approximation

constrained by its own set of assumptions (and differing from the assumptions used to simulate ventilation rates in APEX). The most important assumption made by Brochu et al. (2006) and discussed in the HREA is their assuming a single ventilatory equivalent ratio for oxygen (VQ) equal to 27, a factor used to calculate ventilation rate for all persons, both sexes, and all ages without consideration of exertion level. A VQ point estimate of 27 could be a generally reasonable approximation for estimating a mean ventilation rate in adults while performing low exertion activities (e.g., LeMura and Von Duvillard, 2004), however it is less appropriate for use in estimating ventilation rates associated with moderate or greater exertion activities (e.g., LeMura and Von Duvillard, 2004), is less than values more commonly used in estimating ventilation in children (e.g., Arcus-Arth and Blaisdell, 2007), and is entirely unable to realistically capture short-term intra- and inter-personal variability in ventilation rates.

With this in mind, Graham (2015) corrected the Brochu et al. (2006) reported mean ventilation estimates and associated standard deviations (and as was done for a similar evaluation in the section 5.4.4.2 of the HREA) using an independent, more appropriate VQ estimate of 30.6 offered by Arcus-Arth and Blaisdell (2007) and used to re-calculate ventilation rates for children ages 7-10. When the Brochu et al. (2006) reported distribution of daily mean ventilation rates are corrected, it is nearly identical to that estimated by APEX, both regarding the mean and the standard deviation (see Figure 2 of Graham, 2015). Further, a simulation performed by Graham (2015) using the corrected Brochu et al. (2006) parameters that would more appropriately describe the distribution of daily mean ventilation rates, indicated that even the maximum estimated daily ventilation rate closely matched that generated using APEX (means of 20.5 m^3/day versus 20.8 m^3/day, respectively). Based on this analysis it is likely that the choice of incorrectly limiting VQ to a single value of 27 in the Brochu et al. (2006) study led to an underestimation of daily ventilation rates at all percentiles of the distribution, particularly for children.

The above discussion and results presented here and those included in the HREA indicate that APEX does not predict more elevated ventilation occurrences than observed in 'real world' data as implied by the commenters. The results presented here indicate that the Brochu et al. (2006) calculated daily ventilation rates are likely systematically underestimated. Further, the Brochu et al. (2006) data, even when corrected using a more appropriate VQ, could not be used to appropriately evaluate variability in ventilation rates having durations of less than 24-hours. In addition, when considering that the energy expenditure measurements used to approximate ventilation were collected over 7-14 days, an approach that compresses day-to-day variability, the Brochu et al. (2006) study likely underestimates the actual variance associated with their reported daily ventilation rates.

(13) *Comment:* A few commenters suggested that APEX estimates of moderate and greater exertion ventilation rates are above those reported in literature sources. Specifically, "The U.S. EPA Exposure Factors Handbook (U.S. EPA, 2011, Table

6-27) presents literature values for inhalation rates at different activity levels. For children aged 3-16, the Handbook lists the following mean ventilation rates: 21-25 L/min for moderate intensity activities and 37-49 L/min for high intensity activities. Figure II.2 demonstrates that a number of the modeled children have ventilation rates that are well above those rates for high intensity activities, and in addition, it is not realistic to assume that a large number of the simulated children would approach these high ventilation rates while engaging in typical outdoor play and sports" (e.g., API).

"[M]any of the simulated children had ventilation rates above 21-25 L/min, and some had rates above 37-49 L/min, which are the ranges of ventilation rates given by the U.S. EPA Exposure Factors Handbook (U.S. EPA, 2011, Table 6-27) for children engaging in medium- and high-intensity activities, respectively. It is not realistic to assume that such a large number of children would approach or exceed these high ventilation rates while engaging in typical outdoor play and sports" (e.g., Gradient).

Response: We disagree with the commenters' characterization of APEX estimated ventilation rates and the comparisons the commenters made with literature provided values. A correction and clarification are needed prior to providing additional response to the comment.

As an initial matter, a correction to the comment is needed regarding the source of information used when comparing APEX estimated ventilation rates with that reported in the Exposure Factors Handbook (U.S. EPA, 2011). The commenters appear to have used data from Table 6-2 of U.S. EPA, 2011 rather than Table 6-27 as they stated. Table 6-2 of U.S. EPA (2011) contains "Recommended Short-Term Exposure Values for Inhalation (males and females combined)" and includes the range of mean ventilation rates cited by the commenter for moderate (21-25 L/min) and high (37-49 L/min) exertion activities for children aged 3 to <16. EPA contends these reported data from Table 6-2 of U.S. EPA (2011) are what was used by the commenters to compare with the ventilation rates output from an APEX model simulation performed by the commenters and summarized in their Figure II.2 (and provided in Figure 3 of Graham, 2015). Based on that comparison, the commenters suggested the APEX-estimated ventilation rates were "not realistic" because the distribution of values estimated by APEX extends beyond the range of values provided by U.S. EPA (2011).

First, the commenters do not provide any published (or other) evidence to support their statement that "it is not realistic to assume that such a large number of children would approach or exceed these high ventilation rates while engaging in typical outdoor play and sports", rendering this statement merely as an opinion. To the best of our knowledge, there is no population-based database available that accurately represents the complete distribution of ventilation rates that exists for all people residing across an entire urban study area(s) and over an entire year, a database that, if available, could provide insight into how many and how often children might engage in moderate or high exertion activities. The U.S. EPA

(2011) data tables are not designed to address this issue. In the absence of having a population-based database of ventilation rates, EPA reasonably uses the CHAD data (i.e., activity pattern survey data based on real people that accounts for influential variables such as age, sex, day-of-week, temperature) and associated energy expenditure estimated by APEX to inform this proportion.

Second, understanding and distinguishing mean and upper percentile estimates for ventilation rates is an important consideration here. The commenter's focus on highest ventilation events addresses less than 1 per cent of the data simulated by APEX for each simulated individual. As summarized in the responses below and detailed in Graham (2015), this leads to a significant low bias in all of the commenter's analyses and comparisons when using a time-averaged data to evaluate upper percentiles of a distribution. Further, Graham (2015) provides the detailed responses to the comment, specifically addressing instances where a number of influential variables (i.e., sex, age, means vs. upper percentile values, event duration) were not accounted for in their comparison of APEX estimates and the U.S. EPA (2011) recommended ventilation rates. In short,

1. The commenter's ventilation rate comparison is biased by their using U.S. EPA (2011) reported data for children of both sexes, while the APEX distribution of ventilation rates is based on data from individuals, i.e., the distribution of ventilation rates are from simulated females and males separately. Overall, the comparison provided by the commenter by not considering differences in ventilation rates for males and females separately would contribute to an underestimation bias of about 4-5%.

2. The commenter biased their comparison of ventilation rates by using U.S. EPA (2011) reported data for children ages 3 to <16 to compare with the APEX estimated ventilation rates of children ages 5 to 18. Using this approach, the comparison provided by the commenter by not considering differences in mean ventilation rates for the appropriate age groupings would contribute to an underestimation bias of about 2-6%.

3. Perhaps most importantly, the commenters biased their evaluation by comparing mean values meant to describe ventilation rates for an average individual in a population to a distribution of values representing a population of individuals. The portion of the U.S. EPA (2011) Table 6-2 cited by the commenter contains recommended mean values and are not meant to be applicable to an entire population, particularly the suggestion that the mean values are appropriate to use for all individuals comprising the population. The commenter neglected to include the additional information provided in Table 6-2 U.S. EPA (2011), which also recommends 95th percentile ventilation rates for each of the selected age groups. For activities involving moderate and high exertion, U.S. EPA (2011) recommends using 27-

35 L/min and 48-70 L/min, respectively, as upper bound (95th percentile) ventilation rates for children ages 3 to <16 years old. The majority of APEX ventilation rates estimated are within these 95th percentile values (see Figure 3 of Graham, 2015).

And finally, in their comparison the commenter selected for individuals not considered as typical or representative of an average population by reducing the total population of simulated individuals to only the few that had two or more lung function decrements. The activity pattern events used, the ventilation rates estimated, evaluated and then contended by the commenter as "not realistic" and presented in the commenter's assessment are those that have been simulated with extremely limited frequency comprising only a tiny fraction of all ventilation rates (e.g., 0.02%) estimated by APEX for each simulated individual (Graham, 2015).

Thus, the data presented by the commenter are atypical of the simulated population in the study area as a whole, and are not comparable with the mean values reported in U.S. EPA (2011), particularly those ignoring observed differences across sex and age. Further, when considering an upper range value for ventilation rate of 70 L/min (i.e., the 95th percentile) reported by U.S. EPA (2011), as expected there are only few individuals simulated by APEX (and illustrated by the commenter's results) that are above this level (i.e., approximately 250 person days out of the 4,546 children, or 5.5% of all children that had at least two lung function decrements; see Figure 3 of Graham, 2015). Even if the commenters ventilation rate comparison were completely appropriate (which it is not due to the age and sex biases discussed above), the overall difference in U.S. EPA (2011) reported and APEX modeled ventilation rates, considering upper percentile values is minimal.

(14) *Comment:* A few commenters suggested the level of equivalent ventilation rate (EVR) used to indicate moderate or greater exertion leads to an overestimation in the number of individuals at or above health effect benchmark levels. Specifically, "A second way the counts of benchmark exposures are biased high relates to how EPA defines moderate or greater exercise over 8 hours. The HREA follows the approach begun in 1996 of defining Equivalent Ventilation Rates (EVR, L/min-m^2 body surface area) between 13 and 27 as moderate. The counts in Chapter 5 thus accumulate exposures accompanied by 8-hour EVRs of 13 or greater. In Chapter 6, the risks are calculated for individuals with daily 8-hour average EVR greater than 13 using response functions developed from chamber study data conducted at a significantly higher EVR, ~ 20. In comments on the first draft HREA, AIR, Inc. presented data that showed the EPA algorithm predicts that the 95th percentile 8-hour EVR is between 14 and 15 while the EVR used in the clinical studies of 20 is about the 99th percentile. AIR included figures showing the distribution of mean EVR, maximum 2-hour EVR and maximum 8-hour EVR for both asthmatics and non-asthmatics. AIR noted that APEX accumulates headcounts for subjects that are associated with 8-hour EVRs in the low 90s of percentiles while the EVR used in the clinical studies represents the 99th percentile. Thus, the resulting benchmark headcounts overestimate the

number of subjects at potential risk and the FEV_1 risks calculated with the E-R method are unreasonably high" (e.g., AAM).

"The HREA defines an equivalent ventilation rate of 13 L/min-m^2 (p. 5-18 of HREA) as the lower-bound equivalent ventilation rate to categorize persons engaged in moderate exertion activities for an 8-hr period. Yet, lung function decrements are calculated for individuals with daily 8-hour average equivalent ventilation rates greater than 13 L/min/m^2 using concentration response functions developed from controlled human exposure study data conducted at significantly higher equivalent ventilation rates of approximately 20 L/min/m^2 (23 in some studies). The 95th percentile 8-hour equivalent ventilation rate is between 14 and 15 L/min/m^2, while the equivalent ventilation rates used in the clinical studies of 20 L/min/m^2 is about the 99^{th} percentile. Thus, the resulting headcounts and risks from the lung function risk assessment are overestimated (Heuss, 2012)" (e.g., TPA).

Response: In general we agree with the commenters to the extent that having a conservative estimate of EVR could overestimate the number of persons at or above the health effect benchmark levels, considering this influential factor alone. EPA noted that using the lower bound of the mean EVR is an important uncertainty in the HREA Table 5-10, stating, "[g]iven that the EVR serves as a cut point for selecting individuals performing moderate or greater exertion activities and is a lower bound value (~5^{th} percentile), the simulated number of people achieving this level of exercise could be overestimated." The latter part of this statement could be better clarified here to state that the number of people identified at or above moderate exertion could be overestimated.

Additional context is needed to clarify the selection of the EVR value used by APEX. In the HREA exposure Chapter 5, APEX uses the 8-hr average equivalent ventilation rate (EVR), along with the 8-hr health effect benchmarks, to identify when simulated individuals could be experiencing exposures of concern. The value used by APEX (13 liters/min-m^2), and discussed in the HREA section 5.2.8, was originally developed by Whitfield (1996) using EVR data reported in a controlled human exposure study conducted by McDonnell et al. (1991). Specifically, the mean EVR in that study is reported as 19.9 ± 3.3 L/min-m^2 for study subjects exposed to 0.10 ppm of O_3 (Table 1, McDonnell et al., 1991). In recognizing that individuals will have an FEV_1 response at variable ventilation rates, it had been assumed by Whitfield (1996) that two standard deviations would approximate a 95^{th} percentile range of the mean EVR, yielding a confidence interval about the mean that extends downward to this selected value of 13 liters/min-m^2 (i.e., 19.9-[2×3.3]). This EVR level has been used in each of the O_3 NAAQS reviews since 1996 (including the current review) to characterize when individuals are at or above moderate or greater exertion.

An analysis of EVRs is presented in Chapter 6 of the HREA (Figure 6-11) and shows that the distribution of EVRs for the APEX simulated individuals in Atlanta at or above 13 have a greater number of persons at the lower end of the

distribution of EVRs, as expected, with a progressively decreasing number of persons having higher EVRs, and having a general distribution form much like an exponential decay. Considering the EVR cut-point described above in defining when an individual may be at or above moderate or greater exertion and the estimated distribution of EVRs in simulated individuals, the selection of this lower bound of 13 liters/min-m^2 could be considered conservative in that it would likely capture a greater number of individuals characterized as at moderate or greater exertion than ought to be because, by design, it uses a lower exertion level than would likely be needed to potentially characterize this activity level in most individuals (i.e. 95%).

While alternative, higher EVR values could be used for characterizing this threshold used to identify the point at which an individual is at or above a moderate exertion level, doing so would then increase the number of simulated individuals not meeting the threshold, a proportion of which would be inappropriately mischaracterized as not being at or above moderate exertion, resulting in a potential underestimation in the number of people identified as experiencing exposures of concern. Therefore, EPA feels the EVR cut-point selected, while possibly conservative, remains a reasonable approximation to identify when individuals may be at moderate or greater exertion.

Comments on Differences between Exposures of Concern and FEV_1 Decrement Results

(15) *Comment:* A few commenters suggested that because there were differences between the number of individuals experiencing exposures of concern (i.e., concentrations at or above the selected health effect benchmark levels) and those estimated to have FEV_1 decrements, the number of people having FEV_1 decrements are overestimated. Specifically "in all but 1 of the 15 APEX study areas, there are no simulated children with more than two exposures greater than 0.080 ppm, and that, in all of the 15 areas, less than 1% of children have two or more exposures greater than 0.070 ppm. Therefore, most of the simulated children have exposures less than the 0.075 ppm, even at the current standard. The APEX model also calculates that many of these children have FEV_1 decrements > 10% (discussed further below). This inconsistency between the modeled results and observations in the controlled exposure studies indicates that EPA likely overestimated the number of children with FEV_1 decrements > 10% using the APEX model" (e.g., API, Gradient).

"The HREA estimates more FEV_1 decrements than exposures of concern. Using the HDDM model to estimate decreases in ozone levels, the EPA also estimates the number of people (and the Proposed Rule focuses on children) at exposures of concern of 60, 70, or 80 ppb ozone, as well as the number of people expected to experience FEV_1 decrements of <10%, <15%, or <20%. However, there is a discrepancy between the FEV_1 and the exposure of concern risk estimates (presented on pp. 75272, 73 & 75 of the HREA). For example, at a modeled standard of 60 ppb, 70,000 children are predicted to be exposed to a benchmark concentration of ≥ 60 ppb one or more times. Yet, also at a modeled standard of

60 ppb, 1.4 million children are predicted to experience at least one FEV_1 decrement of ≥ 10%" (e.g., TCEQ).

Response: EPA disagrees with the commenters' suggestion that because there is a difference in the potential health risk results generated using the two approaches (i.e., exposure related exceedances of health effect benchmarks and modeled lung function decrements) then the approach generating the higher risk values (the FEV_1 decrements) is in error. The two approaches are generally similar (developing counts of individuals estimated to potentially experience an adverse respiratory outcome) though they are not directly comparable. As described in the HREA, the approach used in Chapter 5 (exposure) assumes both an 8-hour exposure concentration and ventilation rate are needed to be exceeded simultaneously, with the health effects data used to inform the benchmark levels derived from more than adverse health endpoint. The main approach used in Chapter 6 uses the McDonnell-Stewart-Smith (MSS) model to estimate FEV_1 decrements (HREA, section 6.2.4), an approach that incorporates the complete time-series of both the exposure concentration and ventilation rate for simulated individuals, in addition to the estimated dose levels (and decrements) for prior time periods, to calculate the corresponding time-series of FEV_1 reductions for every exposure event in each individual.

The latest formulation of the MSS model (McDonnell et al., 2012), acknowledged by CASAC as appropriate for estimating risk of lung function decrements and used in the HREA,[59] was developed from the 15 controlled human exposure studies using different exposure durations and different exertion levels (and hence breathing rates). The MSS model "allows for modeling a delay in response until accumulated dose (taking into account decreases over time according to first order reaction kinetics) reaches a threshold value. The threshold is not a concentration threshold and does not preclude responses at low concentration exposures" (HREA, p. 6-9). Thus, FEV_1 reductions simulated by the MSS are not always directly linked with the occurrence of high concentration exposures alone, and limited to their occurring across an entire 8-hour period. Further, an evaluation of the influence EVR has on FEV_1 decrements indicates a large portion of the decrements (upwards to about 45%) occur when individuals are at an EVR of less than 13 (HREA, Tables 6-9 and 6-10). Thus, FEV_1 reductions simulated by the MSS are not always directly linked with the occurrence of high ventilation rates alone and limited to their occurring across an entire 8-hour period. Based on the model formulation that accounts for decrements that may occur at lower concentrations, lower ventilation rats, and shorter durations, the MSS model captures a greater number of individuals at risk than would be estimated when

[59] See, e.g., Frey 2014a p. 2 ("The CASAC finds that the MSS model to be scientifically and biologically defensible. The incorporation of time--dependent inhaled ozone dose and detoxification dynamics represent a substantial improvement over the mean population response analyses at a fixed level of exertion that were done in the previous risk assessments").

considering peak 8-hour exposure concentrations concomitant with moderate or greater exertion alone.

Comments on Urban Core vs Outside Urban Core Exposure Estimates

(16) *Comment:* One commenter contends that "suburban and rural areas will gain more benefits of decreasing ozone, while in the urban areas there will be fewer benefits. This is demonstrated by the comparison of urban and non-urban exposures of concern and FEV_1 decrements shown in Appendix 9 of the HREA (Figures 9A-1 to 9A-24) – as the ozone levels are pushed lower, the discrepancy in ozone exposures between urban core and non-urban areas becomes greater (Figure 3)" (e.g., TCEQ)

Response: EPA generally agrees with statement made by the commenter, although a correction is needed to the figure provided by the commenter. The figure provided by the commenter does not represent data from Appendix 9 Figure 9A-7 for 2007 as there is no observable difference between the urban study area and outer study area 1-hour exposures of 80 ppb (see Figure 9A-7 of the HREA). However, a representation of this general pattern described by the commenter can be seen using the 2009 exposure results for Houston (i.e., Figure 9A-7 of the HREA), whereas approximately 38% of simulated individuals residing in the urban portion of the study area experienced a maximum 1-hour concentration at or above 80 ppb in the urban portion of the study area, while approximately 21% of simulated individuals residing in the outer portion of the study area experienced a similar 1-hour concentration. The appropriate results for each of the 12 study areas can be found in Appendix 9 Figures 9A-1 to 9A-12 (maximum 1-hour exposures) and Figures 9A-13 to 9A-24 (maximum FEV_1 decrements).

EPA acknowledged the presence of this general pattern in concentrations in the O_3 HREA Appendix 9, stating "when we compare patterns of risk reduction for the urban core and outer ring (across urban study areas), we generally see larger degrees of risk reduction for the outer rings. This may reflect two factors: (a) design monitors (targeted for ozone reductions under simulated attainment of the current and alternative standard levels) tend to be located in the outer ring and consequently ozone levels near these monitors are likely to experience greater degrees of reduction and (b) there may be a degree of dampening of risk reduction in the urban core reflecting the non-linear nature of ozone formation which can result in increase in ozone on lower ozone days following simulation of both current and alternative standard levels (see section 7.1.1 for additional discussion)."

However, there are important qualifications associated with this evaluation which the commenter does not acknowledge. First, within the urban study areas exhibiting this pattern, including Houston, the model-based air quality adjustments show reduced O_3 levels at the highest ambient concentrations and increases in the O_3 levels at the lower ends of those distributions (HREA, section

4.3.3.2, Figures 4-9 and 4-10). Analyses of trends in monitored O_3 indicate that over such a time period, the upper end of the distribution of monitored O_3 concentrations (i.e., indicated by the 95th percentile) generally decreased in urban and non-urban locations across the U.S. (HREA, Figure 8-29). This is significant because the evidence from controlled human exposure studies, which provide the strongest support for O_3-induced effects following exposures to O_3 concentrations corresponding to the upper portions of typical ambient distributions (60 ppb and above), make it appropriate to focus on risks associated with O_3 concentrations in these upper portions of the air quality distribution (79 FR 75291/1). Likewise, the epidemiologic evidence, including the shape of C-R functions, provides stronger support for the occurrence of O_3-attributable health effects following exposures to O_3 concentrations corresponding to the upper ends of typical ambient distributions (79 FR 75278-79; preamble to the final rule, section II.B.3). Consequently, the EPA believes that both urban core and downwind areas will experience significant reductions in risk from lowering O_3 through NO_X reductions because the portion of the air quality distribution most linked to risk will be reduced relatively uniformly in both the urban core and outlying areas.

Second, as pointed out by the EPA and endorsed by CASAC, NO_X reductions will lead to reductions in formation of nitrate PM and will do so in both urban core and outlying areas (79 FR 75285 and 75287 n. 107; Frey, 2014a, p. 10). This reduction will likewise increase public health protection in urban core and outlying areas.

Third, as explained at 79 FR 75277-78, representativeness analyses in the HREA indicate that the majority of the U.S. population lives in locations where reducing NO_X emissions would be expected to result in decreases in warm season averages of daily maximum 8-hour ambient O_3 concentrations. These areas include suburbs and urban areas outside the urban center. The HREA analysis of urban study areas thus underrepresents the larger populations living in such areas, and likely understates the average reductions in O_3 associated-mortality and morbidity risks that would be experienced across the U.S. population as a whole.

Comments on Personal Exposure Measurements and APEX Modeled Concentrations

(17) *Comment:* One commenter contends "[a]mbient concentrations are not representative of personal exposures" and followed with a comment on the Detroit Exposure Aerosol Research Study (DEARS) daily personal O_3 exposure data as "well below any of the benchmarks suggested" and later "in figure 5-15 of the HREA that the upper end of daily average ozone personal exposure are well less than 20 ppb, well below the current standard and the range of proposed alternate standards" (e.g., OSIPC).

Response: In general, we agree with the comment that ambient O_3 concentrations do not equal personal exposures. This is why EPA performed an exposure assessment using an exposure model that not only accounts for spatially and temporally variable ambient concentrations, but also includes pollutant removal

within indoor microenvironments and time activity profiles to better establish contact of the simulated study population with the pollutant of interest. As such, the distribution of exposure concentrations generated in Chapter 5 of the O_3 HREA are simulated personal O_3 exposures (not ambient O_3 concentrations) for each of the study groups of interest and in each of the study areas.

EPA disagrees with the commenter's interpretation of the daily average DEARS study data relative to the 8-hour benchmark exposures and consideration of the existing and alternative 8-hour standards. Additional context is provided regarding the DEARS study sampling protocol, the study period, and personal exposure measurement data set. The personal O_3 exposure sample collection protocol for the 36 DEARS study participants was staggered using 5-day consecutive sampling over a two month period (7 sampling events for ~ 5 persons each in July and August 2006) (HREA, Appendix 5G, section 5G-5). The DEARS two-month exposure sampling (and the corresponding APEX exposure simulations) is not considered in context with air quality that just meets the standard, which considers three complete years of air quality, but considers existing air quality conditions for that year. Design values calculated for Detroit that include 2006 air quality were all above the existing 8-hour standard of 75 ppb.[60] While it is possible that an actual short-term high ambient O_3 concentration event occurred during this two-month period in 2006,[61] at most only five study participants out of the total 36 study participants (14%) had the potential to experience these highest concentrations, if it occurred at that time and if the study participants spent time outdoors during that possible short-term high ambient O_3 concentration event.

Furthermore, the daily average is the mean of all 24 one-hour concentrations that vary throughout the day. This differs from the averaging time and form of the health effect benchmark of interest which is the daily maximum 8-hour average (the value calculated by averaging the sequential 8-highest concentrations in the day). Unless all of the 1-hour concentrations in a day were exactly the same (which is highly unlikely), the 8-hour daily maximum will always be greater than the daily average. Therefore, directly comparing a daily mean with an 8-hour daily maximum is inappropriate.

(18) *Comment:* One commenter suggested based on a comparison of personal exposure measurement data and APEX exposure estimates provided in the HREA, there is greater variability in APEX estimated exposures. Specifically, "[a]nother comparison can provide further insight into this issue. A comparison of personal O_3 exposure measurements from Detroit with an APEX simulation reported in the HREA showed that the outdoor concentrations and time outdoors tracked well between the simulation and the observations, but that there were

[60] http://www.epa.gov/airtrends/values_previous.html

[61] In the APEX exposure simulation that was performed for this particular period in Detroit, there were a number of person-days where the simulated adults had an 8-hr exposure at or above 60 ppb, just not occurring at or above moderate or greater exertion levels.

major differences in the mean daily ozone exposures and, importantly, the maximum daily ozone exposures, as shown in Figure 5-18 from the HREA. This comparison clearly shows the influence of the excessive variability in the APEX model" (e.g., AAM).

Response: EPA generally disagrees with the commenter's interpretation of the DEARS study data relative to the APEX-estimated exposures, but agrees to some extent that APEX better models variability in exposures compared with time-averaged exposure measurements. In evaluating differences in DEARS personal O_3 exposure measurements with that of APEX simulated O_3 exposures, the HREA discusses at length the possibility for a systematic low concentration bias in the DEARS measurements related to either detection limits of the passive sampler device used and/or the generally limited amount of time spent outdoors by study participants (HREA section 5.4.4.1). Thus, considering the staggered sampling protocol for the DEARS study, the potential for underestimating of personal exposures due to passive sampler detection limits, lack of precision in personal measurements when individuals transition across low and high concentration microenvironments contrary to APEX which can precisely simulate concentrations on a minute-by-minute basis, and the limited time spent outdoors by DEARS study participants combined would limit the likelihood of capturing high O_3 exposure events.

(19) *Comment:* One commenter contends that EPA "is aware that there are differences between ambient concentrations of ozone and personal exposure, but effectively ignores this difference in the O3 HREA when deriving quantitative estimates of risk" and cites two published papers (O'Neill et al., 2003; Lee et al., 2004) (e.g., OSIPC).

Response: EPA disagrees with this comment as EPA has reported the observed differences in exposure and ambient concentrations in the ISA (e.g., section 4.3.3, Table 4-3) using 8 distinct studies, one of which was the commenter- mentioned O'Neill et al. (2003) study. EPA also acknowledged the expected difference in exposure and ambient concentrations when calculating exposures (HREA, Chapter 5) and health risk (HREA, Chapters 6 and 7).

(20) *Comment:* One commenter contends that "studies that have investigated ozone personal exposure and compared it to ambient concentrations have found that personal exposure is much lower than ambient exposure (about 10% of the measured ambient level; Lee et al., 2004), and that there may not even be a correlation between personal and ambient concentrations (Sarnat et al., 2001; Sarnat et al., 2005). Because of this, not only will an assumption of ambient concentrations not necessarily accurately represent the individuals in the study, it also grossly overestimates their exposure" (e.g., TCEQ).

Response: EPA disagrees with the commenter's interpretation regarding the relevance of ambient O_3 concentrations to exposures. EPA is well aware of the differences in personal exposure measurements and ambient concentrations,

largely the result of removal processes that reduce O_3 concentrations when penetrating into indoor microenvironments. See, e.g. the immediately preceding response. The ISA presents a wide range of values reported from 10 studies having indoor (I) and outdoor (O) concentration measurements (ISA, section 4.3.2, Table 4-1), with I/O ratios generally between 0.10 to 0.40, along with having a similar range of personal exposure to ambient concentration ratios (generally between 0.10 to 0.30, see ISA, section 4.3.3, Table 4-3). This of course indicates the importance of the indoor microenvironment in estimating overall exposure concentrations, though it perhaps is more relevant when considering exposure durations only as short as a day or more.

However, in understanding how people may be exposed to concentrations of concern, particularly when considering an 8-hour average concentration, the time spent outdoors (and hence the outdoor ambient concentrations) is of much greater importance than time spent indoors (HREA, section 5.4.2). In support of this finding, note the higher personal exposure measurement to ambient concentration ratios for Farmworkers (0.96) and Camp Counselors (0.53) reported by Brauer and Brook (1997), individuals expected to spend a few to several hours per day outdoors, particularly during the afternoon hours when O_3 concentrations are highest. Therefore, when accounting for people's transitions through varying microenvironments, as is done in our APEX exposure modeling on a minute by minute basis (where applicable based on the CHAD diary data used to simulate activity patterns), short-term peak ambient concentrations are of paramount relevance to estimating exposures.

(21) *Comment:* One commenter suggests "for several reasons, described in more detail below, personal exposure modeled by the APEX model is likely to be overestimated. Based on a personal monitoring study conducted using a miniaturized UV absorption monitor (PEM) to measure ozone concentrations in a series of microenvironments in Raleigh, North Carolina, Long et al. (2005) found that the APEX model underestimates concentrations in indoor and in-vehicle microenvironments when windows are open (modeled concentrations are approximately ½ the measured concentrations) and results in an 8-fold overestimate of concentrations when windows are closed. The median ratio of indoor PEM concentrations to APEX concentrations was 1.87 for open windows and 0.13 for closed windows" (e.g., TPA).

Response: EPA generally disagrees with the commenter's limited interpretation of how personal exposure measurement studies can be used to evaluate APEX estimated exposures. A small clarification is needed prior to responding to the comment: the commenter has not provided a reference for the study cited "Long et al. (2005)" upon which they have based their comment and no such study could be found upon a literature search; however, EPA has found a conference presentation (not peer-reviewed) containing similar findings to that cited by the commenter (i.e., Long et al., 2008). The response below assumes the commenter intended to cite to Long et al. (2008).

EPA has used many types of concentration measurement data (microenvironmental, exposure, and ambient) among other data (e.g., O_3 decay rates) in both developing and evaluating the APEX model. Input data generation, algorithm formulation, and model evaluation using the highest quality data available are fundamental to the APEX model development and applications, as evidenced by its incremental, significant improvements made over time (U.S. EPA, 2012a; U.S. EPA, 2012b) and increasing usage in assessing population based exposures to ambient pollutants (HREA, U.S. EPA, 2008b, 2009a, 2010a). In particular for this review to evaluate model performance, EPA used personal exposure measurement data from the DEARS study and compared the distribution of daily average concentrations to the APEX estimated exposures (HREA, section 5.4.4.1). There were differences between the DEARS measurement and APEX modeled data and EPA justifiably found in the O_3 HREA that bias can exist in both the measurement reported and modeled data. In particular, regarding the APEX concentrations estimated for the Detroit study area, "it is possible that indoor O_3 concentrations could be biased high on occasion due to a wider range of variability in AER equally applied across simulated individuals' than should be as well as the potential for underestimation of indoor O_3 decay" (HREA, page 5-61). This discussion in the HREA is generally consistent with the reported findings by Long et al. (2008) and mentioned by the commenter, i.e., when the scripted indoor microenvironmental scenario measured concentrations with windows closed, APEX modeled indoor concentrations were higher than the measured indoor concentrations in that study. Also, as mentioned by the commenter, when indoor microenvironmental scripted conditions called for windows to be open, the APEX modeled indoor concentrations were *lower* than those measured. In general, these variable results (indicated by Long et al., 2008 and in the HREA evaluation) indicate there could be future investigations into evaluating the types of new measurement sampling methods, input data used, and algorithms that might be needed to incrementally improve the agreement between the APEX model estimated concentrations, specifically those pertaining to the indoor microenvironments where people may experience O_3 exposures.

However, when considering the most important factor influencing individuals who experience the highest O_3 exposures, that is, large amounts of afternoon time spent outdoors and high ambient O_3 concentrations (HREA, section 5.4.2), these discrepancies in estimating indoor microenvironmental concentrations are of far lesser importance. Consider first, for example, the Long et al. (2008) findings reported by the commenter that the greatest discrepancies were actually those where the measurement concentrations were lowest. The measured O_3 concentrations in indoor microenvironments were almost always less than 10 ppb, regardless of whether windows were closed or open, while those within residential microenvironments were barely detected[62] when windows were closed. As was discussed in the HREA (section 5.4.4.1), these discrepancies could result

[62] The Long et al. (2008) study does not include detection limits, however, according to Figure 2 of that study, concentrations as low as 1 ppb or less were reported. Manufacturer reported detection limits for the sampler used in the Long et al. (2008) study are 3.0 ppb (see http://www.twobtech.com/model_202.htm).

from an underestimation bias by the measurement device due to indoor microenvironmental concentrations existing at or below sampling detection limits. Consider also, the most relevant findings reported by Long et al. (2008) are those using the outdoor O_3 concentrations, specifically "In outdoor microenvironments, good agreement was observed between PEM concentrations and ambient concentrations (median ambient/PEM ratio = 0.92) and between PEM concentrations and APEX estimates (median APEX/PEM ratio = 0.97)" (taken from Figure 4 of Long et al., 2008). In addition to the observed strong agreement, median concentrations outdoors using either measurement or modeled data were about 60 ppb, much higher and thus more relevant to 8-hour average exposure concentrations of concern than concentrations occurring indoors.

Comments Regarding Health Benefits with Reducing Standard Level

(22) *Comment:* A few commenters suggested that reducing the NAAQS does not lead to significant decreases in the number of individuals exposed to O_3. Specifically, "EPA used extremely conservative assumptions in the exposure and risk assessments and still did not demonstrate that the lower ozone NAAQS would be more protective of health than the current standard. Using the Air Pollutants Exposure (APEX) model based on controlled exposure studies, EPA modeled the current conditions, meeting 0.075 ppm, and meeting alternative possible standards (0.070, 0.065, and 0.060). EPA showed the largest benefits come from just meeting 0.075 ppm, with little gains from a lower standard" (e.g., NMOGA).

"The APEX exposure assessment demonstrates that lowering the NAAQS will not lead to a significant decrease in the number of individuals experiencing multi-day, high-ozone exposures" (e.g., Gradient).

Response: EPA disagrees with the commenter regarding the degree of conservatism in the assumptions used in the exposure modeling. All exposure model input data and results generated were identified as appropriate by CASAC, without mention of extreme conservatism. EPA also disagrees with both commenters regarding their characterization of exposure reductions associated with meeting alternative standard levels more stringent than the existing standard of 75 ppb. The reduction in exposures to the 60 ppb benchmark is substantial in each HREA study area (HREA Figure 9-8), whereas on average, about 50% fewer people are exposed to that level when the standard is lowered from 75 ppb to 70 ppb, with consistently greater reductions in exposures with increasing stringency of the standard downwards to a level of 60 ppb.

Other Exposure-related Influential Factors

(23) *Comment:* One commenter contends that high air conditioning usage in the U.S., "as much as 98.4%" of the population", will "remove the vast majority of ambient ozone" (commenter provides an illustration from the U.S. Energy Information Administration) (e.g., OSIPC).

Response: We agree with this comment and add that the APEX model accounts for air conditioning prevalence (HREA, Appendix 5B-6.2) in calculating O_3 exposures all study areas, and appropriately estimates the infiltration of outdoor ambient concentrations (HREA, Appendix 5B-6.3) and resultant decay (HREA, Appendix 5B-6.6) within indoor microenvironments.

(24) *Comment:* One commenter contends "[t]he third way the counts of benchmark exposures are biased high relates to the fact that ozone exposure is lower at "breathing" height compared to "measurement" height (3-15 meters) as acknowledged in the 2006 Ozone Criteria Document" (e.g., AAM).

Response: EPA disagrees with this comment regarding the potential impact of monitor probe heights relative to counts of benchmark exposures in the HREA study areas. First, there is limited information available to quantitatively inform the variation in ozone concentrations with vertical height in urban areas. Most of the discussion in the 2006 AQCD[63] that indicated the presence of a vertical gradient, also indicated it was largely attenuated by unstable atmospheric conditions, conditions likely present during daytime hours in urban areas (2006 O_3 AQCD). In addition, nearly 70% of the inlet heights for the monitors used in the HREA study areas are typically situated at a height of 3 to 5 meters (see Figure 4 of Graham, 2015). Taken together, while there is some information indicating decreasing O_3 concentration with decreasing monitor probe height, there are too few studies available with having appropriate data to develop a reasonable quantitative relationship in urban areas, but more importantly, the overall expected impact on the HREA estimated exposures is likely be negligible based on the probe heights of the majority of the O_3 monitors used in estimating exposures and the atmospheric conditions that typically exist during periods of high ambient concentrations.

ii. Risk of O_3-Induced FEV_1 Decrements

[63] The 2006 Ozone Criteria Document (O_3 AQCD, U.S. EPA, 2006a) states in a section titled "Vertical Variations in Ozone Concentrations" (pages 3-15 to 3-17) that "[m]ost work characterizing the vertical profile of O_3 near the surface has been performed in nonurban areas with the aim of calculating fluxes of O_3 and other pollutants through forest canopies and to crops and short vegetation, etc. Corresponding data are sparse for urban areas." The section in the 2006 O_3 AQCD discusses the variability in concentration with respect to vertical height at some length including influence by vegetation present and atmospheric stability and concludes, based on the information derived from non-urban studies, that the degree of atmospheric stability is important as "there was a decrease of about 20% in going from a height of 4 m down to 0.5 m above the surface during stable conditions, but O_3 decreased by only about 7% during unstable conditions" and that "the stability regime during the day in urban areas tends more toward instability because of the urban heat island effect." Later the 2006 AQCD very briefly mentions in a section titled, "Factors Affecting the Relationship between Ambient Concentrations and Personal Exposures to O_3", "[s]tudies on the effect of elevation on O_3 concentrations found that concentrations increased with increasing elevation (Väkevä et al., 1999; Johnson, 1997)."

The EPA also received a large number of comments on the FEV_1 risk assessment presented in chapter 6 of the HREA and summarized in the proposal (II.C.3.a in the proposal).

(1) *Comment:* Commenters representing medical, public health, and environmental groups generally expressed the view that these risk estimates support the need to revise the current primary O_3 standard in order to increase public health protection, though these groups also questioned some of the assumptions inherent in the EPA's interpretation of those risk estimates. For example, ALA et al. stated that "[t]he HREA uses a risk function derived from a controlled human exposure study of healthy young adults to estimate lung function decrements in children, including children with asthma. This assumption could result in an underestimate of risk." On this same issue, commenters representing industry groups opposed to revising the standard also asserted that assumptions about children's responses to O_3 exposures are highly uncertain. In contrast to medical and public health groups, these commenters concluded that this uncertainty, along with others discussed below, call into question the use of FEV_1 risk estimates to support a decision to revise the current primary O_3 standard.

Response: The EPA agrees that an important source of uncertainty is the approach to estimating the risk of FEV_1 decrements in children and in children with asthma based on data from healthy adults. However, this issue is discussed at length in the HREA and the PA, and was considered carefully by CASAC in its review of draft versions of these documents. The conclusions of the HREA and PA, and the advice of CASAC, were reflected in the Administrator's interpretation of FEV_1 risk estimates in the proposal, as described below. Commenters have not provided additional information that changes the EPA's views on this issue.

As discussed in the proposal (II.C.3.a.ii in the proposal), in the near absence of controlled human exposure data for children, risk estimates are based on the assumption that children exhibit the same lung function response following O_3 exposures as healthy 18-year olds (i.e., the youngest age for which sufficient controlled human exposure data is available) (HREA, section 6.5.3). As noted by CASAC (Frey, 2014a, p. 8), this assumption is justified in part by the findings of McDonnell et al. (1985a), who reported that children (8-11 years old) experienced FEV_1 responses similar to those observed in adults (18-35 years old). The HREA concludes that this approach could result in either over- or underestimates of O_3-induced lung function decrements in children, depending on how children compare to the adults used in controlled human exposure studies (HREA, section 6.5.3). With regard to people with asthma, although the evidence has been mixed (ISA, section 6.2.1.1), several studies have reported statistically larger, or a tendency for larger, O_3-induced lung function decrements in asthmatics than in non-asthmatics (Kreit et al., 1989; Horstman et al., 1995; Jorres et al., 1996; Alexis et al., 2000). On this issue, CASAC noted that "[a]sthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting O_3-induced pulmonary function decrements" (Frey, 2014b, p. 4). To the extent asthmatics experience larger O_3-induced lung function decrements than the healthy adults used to develop exposure-response relationships, the HREA

could underestimate the impacts of O_3 exposures on lung function in asthmatics, including asthmatic children (HREA, section 6.5.4). As noted above, these uncertainties have been considered carefully by the EPA and by CASAC during the development of the HREA and PA. In addition, the Administrator has appropriately considered these and other uncertainties in her interpretation of risk estimates, as discussed further in the preamble to the final rule (sections II.B.3, II.C.4.b, and II.C.4.c).

(2) *Comment:* Some commenters additionally asserted that the HREA does not appropriately characterize the uncertainty in risk estimates for O_3-induced lung function decrements. Commenters pointed out that there is statistical uncertainty in model coefficients that is not accounted for in risk estimates. One commenter presented an analysis of this uncertainty, and concluded that there is considerable overlap between risk estimates for standard levels of 75, 70, and 65 ppb, undercutting the confidence in estimated risk reductions for standard levels below 75 ppb.

Response: The Agency recognizes that there are important sources of uncertainty in the FEV_1 risk assessment. In some cases, these sources of uncertainty can contribute to substantial variability in risk estimates, complicating the interpretation of those estimates. For example, as discussed in the proposal, the variability in FEV_1 risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels (Table 2 in the preamble to the final rule and 79 FR 75306 n. 164). Given this, and the resulting considerable overlap between the ranges of FEV_1 risk estimates for different standard levels, the Administrator views these risk estimates as providing a more limited basis than exposures of concern for distinguishing between the degree of public health protection provided by alternative standard levels. Thus, although the EPA does not agree with the overall conclusions of industry commenters, their analysis of statistical uncertainty in risk estimates, and the resulting overlap between risk estimates for standard levels of 75, 70, and 65 ppb, tends to reinforce the Administrator's approach, which places greater weight on estimates of O_3 exposures of concern than on risk estimates for O_3-induced FEV_1 decrements.

In addition to the comments addressed above, and in the preamble to the final rule, EPA received a number of technical comments on the risk of O_3-induced FEV_1 decrements. These comments are addressed below.

Measurement Error in the Clinical Data and MSS Model

(3) *Comment:* Several commenters (e.g., AAM, API) pointed out that there is measurement error in the clinical data and that EPA's risk estimates do not properly take that into account.

- The MSS model, as applied in the HREA, overestimates FEV_1 decrements by assuming that there is no measurement error in the clinical data, in contradiction of the MSS author's acknowledgement that the data is noisy.

The FEV_1 decrements in Table 2 of the proposal are overestimates due to this assumption (e.g., AAM).

- "The MSS model predicts substantially more occurrences of various decrements, about a factor of three higher than the E-R approach. As shown in Figures 6-9 and 6-10 of the HREA, the MSS model predicts FEV1 ≥ 10 % decrements at exposures as low as 10 to 20 ppb and predicts substantial decrements below 60 ppb. Also as shown in Tables 6-9 and 6-10, almost half of the profiles with instances with FEV1 ≥ 10 % never experience 8-hour EVR ≥ 13. Table 2 in the proposal reports the decrements calculated using the MSS model, and the discussion of lung function effects in the proposal relies on the data in Table 2.

- "The question arises as to why the MSS model predicts FEV_1 decrements at low ozone concentrations and mild exercise rates even though the model includes consideration of a threshold. First, McDonnell et al. acknowledge that the data from the individual lung function measurements are noisy. The model was developed from a dataset of 8477 lung function measurements during ozone exposure. There is also a dataset of 2948 measurements made during filtered air exposures. The fit of the individual model predictions versus the observations for the 8477 individual measurements during ozone exposure is shown in Figures 2a and 3a from McDonnell et al. 2012 and reproduced here as Figure 13. The noise in the individual response data is evident in these figures with the range of the data as the predictions approach zero being roughly between a 10% improvement in FEV_1 to a 10% decrement. In fact, the HREA acknowledges that the model does not have good predictive ability for individuals, with $r^2 = 0.28$. McDonnell et al. point out:

    > All within-subject variability is currently lumped into a single term E as a result of limitations of the model fitting program. It is likely that some of the within-subject variability is due to true changes in responsiveness to ozone over time while much is simply noise.

    - Second, in contrast to McDonnell's acknowledgement that the lung function measurements are noisy, the Agency assumes that there is zero measurement error, noting:

        > The MSS model estimated intra-individual variability Var(ε) has two basic components: (1) the intra-individual variability of the true response to O_3 (both within-day and between-day) and (2) measurement error. These cannot be distinguished based on the available data. We are assuming that all of this variability is due to the true response, which will (absent other uncertainties) tend to overestimate the response to O_3…. The assumption of no measurement error in Var(ε) has the potential to significantly affect the risk results" (e.g., AAM).

> "Another limitation of the APEX model is associated with the version of the MSS model that EPA used. EPA used the threshold version of the MSS model, which assumes that the intra-subject variability parameter has a Gaussian distribution centered on zero, and truncated this parameter to ± 2 standard deviations (HREA). EPA conducted a number of sensitivity analyses on the intra-subject variability parameter by varying the truncation of the parameter. These analyses showed that different approaches had a significant impact on the model-predicted percent of the population with FEV_1 decrements > 10% and > 15% (but less of an effect on the percent with FEV_1 decrements > 20%) (HREA)" (e.g., API).

Response: The commenter states that the MSS model predicts FEV_1 decrements at low ozone concentrations and mild exercise rates even though the model includes consideration of a threshold, based on Figures 6-9 and 6-10 and Tables 6-9 and 6-10 of the HREA. EPA would like to point out that while Figures 6-9 and 6-10 show that some FEV_1 decrements ≥ 10% are seen at low exposures, these are likely at high exercise levels with O_3 dose remaining from the previous day; and while Tables 6-9 and 6-10 show that some FEV_1 decrements ≥ 10% occur when 8-hour EVR ≤ 13, these are likely at high O_3 concentrations. These figures and tables do not indicate that there are FEV_1 decrements occurring when O_3 concentrations are low at the same time that EVR ≤ 13. Note that the MSS threshold model was used for these analyses, and so these results are consistent with the MSS dose threshold. Therefore EPA disagrees with the commenter's statement that "the MSS model predicts FEV_1 decrements at low ozone concentrations and mild exercise rates." The response to comment above (in the section titled Comments on Differences between Exposures of Concern and FEV_1 Decrement Results) provides additional discussion of the threshold in the MSS model.

Although EPA agrees that there can be measurement error in the clinical data, the MSS model applied in the HREA takes this uncertainty into account. Specifically, the variability term (Var(ε)) includes measurement error and within-individual variability not otherwise captured by the model (HREA, Section 6.2.4). EPA used the parameters for that term given in McDonnell et al. (2012). EPA acknowledged the significance of this term in the HREA. As stated in the HREA, "Clearly the intra-individual variability Var(ε) in the MSS model is a key parameter and is influential in predicting the proportions of the population with FEV_1 decrements > 10 and 15%," (HREA Section 6.5.1.2).

Despite this acknowledgement, the HREA assumes that there is zero measurement error, due to gaps in the available data. In HREA Section 6.5.1.2, EPA explains that:

> The MSS model estimated intra-individual variability Var(ε) has two basic components: (1) the intra-individual variability of the true response to O_3 (both within-day and between-day) and (2) measurement error. These cannot be distinguished based on the available data. We are assuming that

all of this variability is due to the true response, which will (absent other uncertainties) tend to overestimate the response to O_3.

To address the potential uncertainty associated with this assumption, EPA conducted a sensitivity analyses reducing the standard deviation of ε by 50%. This reduces Var(ε) to 25% of the base case value, which corresponds to assigning 25% of Var(ε) to intra-individual variability and 75% to measurement error. This reduced the percent of children ages 5 to 18 with one or more FEV_1 decrements ≥ 10% from 32% to 20%, for the scenario modeled (Atlanta, 2006 O_3 season) (HREA, section 6.5.1.2).

Since the components of Var(ε) cannot be separately quantified based on the available data, EPA employed the conservative assumption that all of this variability is due to the true response. However, the claim that the FEV_1 decrements in Table 2 of the proposal are overestimates does not follow from this, since there are a number of other uncertainties in these numbers. EPA agrees that this assumption (absent other uncertainties) has the potential to overestimate the response to O_3. However, this does not preclude EPA from using the results of the modeling to inform considerations of the adequacy of the existing standard or the protectiveness of alternative standards.

Accounting for individual variability of FEV_1

(4) *Comment:* "One limitation not discussed in the Proposed Rule is that the APEX model does poorly when predicting individual decrements in FEV_1. The Proposed Rule reports results for the number of children who experience at least one or two FEV_1 decrements per year (HREA, Table 2), but highlights results primarily for at least two FEV_1 decrements in a year. Determining the number of individuals who experience at least one or two FEV_1 decrements over a cutoff value likely overestimates the significance of individual responses, particularly at lower ozone exposure levels, because these results are based on controlled exposure studies that may not have accounted for individual variability of FEV_1 when measured by diagnostic spirometry. For example, in a study that took repeated FEV_1 measurements from several healthy individuals exposed to clean air, the observed variation in FEV_1 was up to ± 5% in some subjects (Lefohn et al., 2010). Because APEX relies on controlled exposure studies that have this inherent variability, it is likely that a portion of the modeled individuals exposed to low ozone concentrations were identified as responders only because they were just over a cutoff that could be explained by normal variability" (e.g., API).

Response: EPA disagrees with this characterization. Individual variability in the controlled exposure studies could result in misclassification of some study participants as being over or under a cutoff when their response is near the cutoff. However, the MSS model is not intended to predict individual decrements in FEV_1. Rather, it predicts a distribution of FEV_1 responses in the population modeled. The MSS model explicitly treats individual variability and the distributions predicted by the MSS model in APEX take this into account. In

addition, >10% decrements in FEV_1 at low ozone doses were avoided by constraining var(U) and var(ε) to be within ±2 standard deviations from the means (when samples are outside of this range, they were discarded and resampled). Figure 6-2 of the HREA shows that the distribution of responses of 20 year olds exposed to 100 ppb with moderate exercise. The 99 percentile of the distribution did not reach a 10% FEV_1 decrement until after 2 hours of exposure. Consequently, the HREA does not overestimate responses at lower ozone exposure levels.

(5) *Comment:* One commenter (e.g., UARG) contended that EPA is inconsistent as to which individual results it uses, and that EPA is chooses individual results not because it is scientifically sound to do so, but rather because the results provide a convenient way to support a standard below 75 ppb. They further argued that EPA did not extrapolate the percentage of individual participants in controlled human exposure studies who experienced FEV_1 improvements, as was done for decrements, to estimate population-wide effects.

Response: The model EPA used to estimate FEV_1 decrements was based on all data from controlled human exposure studies that were available at the time (McDonnell et al., 2012) and includes over 700 exposed subjects from 22 studies. The data on FEV_1 improvements, as well as decrements, were used in modeling O_3-induced FEV_1 decrements as well as the individual variability in measurement error. For further discussion of this point, refer to section II.A.1.b above.

Influence of Background on Risk Estimates

(6) *Comment:* Commenters (e.g., Lefohn and Oltmans) demonstrated that background ozone concentrations have a significant effect on the lung function risk assessment. These commenters performed analyses which demonstrate that a large percentage of the risks are associated with 1-hour average ambient concentrations in the 25-55 ppb range, which they assert is heavily influenced by background O_3.

Response: EPA agrees that ozone concentrations in the 25-55 ppb range can be heavily influenced by background O_3. For example, Table 2-2 on page 2-25 of the PA provides estimates of background ozone for each of these cities. The estimates of seasonal mean maximum daily 8-hour average background ozone concentrations for Los Angeles, Denver, Houston, Philadelphia, and Boston are respectively 51, 47, 48, 49, and 43 ppb. Since these are seasonal means, the daily background ozone concentrations will fluctuate around these numbers. Therefore, estimates of background concentrations can extend well into the 25-55 ppb range.

This commenter's finding that a large percentage of the risks are associated with 1-hour average ambient concentrations in the 25-55 ppb range is consistent with similar results presented in the HREA (Figure 6-9, Section 6.3.1). In consideration of these analyses, EPA agrees that background concentrations can have significant influence on the absolute risk estimates, but it would not

significantly affect the incremental risk estimates between alternative standard levels. The air quality modeling incorporates emissions from background sources, and the adjustments to ozone to reflect just meeting alternative standard levels are based only on reductions in U.S. anthropogenic NO_X emissions, and therefore differences between alternative standard levels only reflect those emissions.

However, there are important qualifications which the commenter does not acknowledge. First, as explained at proposal and in the final rule preamble, risks are most likely associated with the upper portions of the air quality distribution. This is true both for exposures of concern (benchmarks of 60 ppb and higher, based on a large body of evidence from controlled human exposure studies) and epidemiologic evidence (reflecting, among other things, the shape of the CR function). See, e.g., 79 FR 75278-79, 291. Second, modeling analyses indicate that these highest O_3 days generally have similar daily maximum 8-hour average background concentrations as the seasonal means of this metric, but have larger contributions from U.S. anthropogenic sources. As summarized in the PA, "the highest modeled O_3 site-days tend to have background O_3 levels similar to mid-range O_3 days … [T]he days with highest O_3 levels have similar distributions (i.e. means, inter-quartile ranges) of background levels as days with lower values, down to approximately 40 ppb. *As a result, the proportion of total O_3 that has background origins is smaller on high O_3 days (e.g. greater than 60 ppb) than on the more common lower O_3 days that tend to drive seasonal means*" (PA, p. 2-21, emphasis added). [64] When averaged over the entire U.S., the models estimate that the mean USB fractional contribution to daily maximum 8-hour average O_3 concentrations above 70 ppb is less than 35 percent. Putting these two facts together, U.S. anthropogenic emission sources are thus the dominant contributor to the higher portions of the air quality distribution most associated with risks.

Uncertainty in APEX-modeled FEV_1

(7) *Comment:* Some commenters (e.g., API) noted that the threshold version of the MSS model has been shown to overestimate the number of individuals experiencing FEV_1 decrements > 10%, especially at low levels of exposure (McDonnell et al., 2013; McDonnell and Stewart, 2014). McDonnell et al. (2013) proposed a new version of the MSS model that addresses these issues, but this version was not used by EPA.

Response: Although EPA agrees that a newer version of the MSS model exists, this model version was not available in time to use for the main risk analysis. However, EPA was able to compare the results of the newer model with the model that was used. In Section 6.5.1 of the HREA, EPA states:

> McDonnell et al. (2013) have introduced another version of their model which assumes that Var(ε) increases with median response. So, with a fixed ventilation rate, Var(ε) will be larger for higher exposure

[64] This phenomenon holds true for areas in the intermountain west (see Henderson, 2012).

concentrations and smaller for lower exposure concentrations. Simulations using their preferred model (Model 3) yield risk results higher than results based on the threshold model used for this HREA; those results are not presented here.

Therefore, EPA disagrees that the newer model would show that EPA's HREA overstated risk.

(8) *Comment:* Some commenters noted that EPA used the age term but not the BMI term in the MSS model, both of which were statistically insignificant, leading to additional uncertainties and overestimation of numbers of FEV_1 decrements in children.

One commenter (e.g., API) provided a sensitivity analysis and discussion regarding the BMI term in the MSS model. They state that McDonnell et al. (2012) proposed an alternative version of the threshold MSS model that alters the model results based on individuals' body mass index (BMI), and the BMI version fits the controlled exposure study data better than the standard version of the model. They state that the better-fitting BMI version produces substantially lower projected numbers of FEV_1 decrements in children and that EPA does not provide adequate justification for using the standard version of the threshold MSS model instead of the BMI version.

- "While it is true that the coefficient on BMI is statistically insignificant in the MSS model, it should be noted that the coefficient on age is also statistically insignificant, regardless of whether BMI is included in the model. The HREA discusses this point on page 6-40. Despite this, the HREA attaches some importance to the coefficient on age, and develops a piecewise linear function of age to allow estimation of an age effect for age groups outside of those studied in McDonnell et al. No explanation is provided in the HREA for why one statistically insignificant coefficient is treated as important, while the insignificance of another is used as grounds to prefer a model that excludes that variable.

  Further, the version of the MSS model that includes BMI as a variable clearly fits the data better than the version that omits BMI. The Akaike Information Criterion (AIC) for the model that includes BMI is 49,583, while the AIC for the model that excludes BMI is 49,594 – a lower AIC indicates a better fit. We also note that the latest publication from McDonnell et al. (2013) only considers the version of the model that includes BMI, suggesting that this is their preferred specification" (e.g., API).

- "The estimates of lung function benefits presented in the HREA do not include any measures of uncertainty. As we have seen in the analysis above, this uncertainty is considerable. Statistical uncertainty in the

estimation of the MSS model and model specification uncertainty in selecting which version of the MSS model to use both have large influences on the HREA results for lung function decrement incidences.

In particular, model specification uncertainty has a dramatic impact on the potential lung function benefits for children, with the estimates of lung function decrement from the version of the MSS model that includes BMI at 75 ppb statistically significantly smaller than the estimates produced using the version of the MSS model that excludes BMI at 70 ppb.

The opposite pattern is observed for older age groups, with the BMI version of the MSS model producing estimates of lung function decrement that are significantly higher than those produced by the version of the MSS model that excludes BMI at an ozone level that is 5 ppb higher.

Put another way, model specification uncertainty has more influence on the estimates of lung function decrement than a 5 ppb change in the ozone NAAQS. As both versions of the MSS model are regarded as credible by researchers, this represents a dramatic source of uncertainty that is completely ignored in the HREA. Alternate assumptions about how to extend the MSS model to children that are based on observed data also lead to dramatically lower estimates of lung function decrement for children" (e.g., API).

Response: EPA agrees that the coefficients on age and BMI are statistically insignificant in the MSS model. In the HREA EPA presents a rationale supporting the extrapolation of the MSS model to younger ages, and compared the predictions of the MSS model to the outcomes of a clinical study of children, lending support to the extrapolation of the MSS model to children (HREA, Appendix 6D). There are no studies of how BMI would enter into the prediction of lung function response for children, and no evidence that the BMI term in the MSS model is appropriate for children.

If the BMI model were used, it would predict lower projected numbers of FEV_1 decrements in children; however, the differences in these numbers between alternative standards analyzed would be very small, as shown in Langstaff (2015). For example, when risk is taken to be the percent of children experiencing FEV_1 decrements > 10%, then the MSS model in APEX without the BMI term estimates risks of 15.85% for the 70 ppb air quality scenario and 19.21% for the 75 ppb (current standard) scenario for APEX simulations of Atlanta, 2006. The corresponding estimates of risk based on the MSS model with the BMI term are 13.95% and 17.11%. The risk reduction from the 75 ppb scenario to the 70 ppb scenario is 3.4% for the MSS model without the BMI term and 3.2% for the MSS model with the BMI term. These differences are very close. If looking at the the

percent of children experiencing FEV_1 decrements > 15%, the risk differences are 1.5% for the MSS model without the BMI term and 1.4% for the MSS model with the BMI term.

(9) *Comment:* Some commenters noted that there is a lack of data supporting EPA's extrapolation of the age term in the MSS model to children, and that therefore the FEV_1 estimates for children are highly uncertain.

One commenter (e.g., API) stated that the MSS model used to calculate FEV_1 decrements included an extrapolation of FEV_1 effects in adults to children. The MSS model was developed using data from healthy individuals aged 18-35 years, and, because there are no data for younger individuals, EPA assumed that a child's FEV_1 decrements are the same as those of an 18-year-old. This assumption adds a potentially large degree of uncertainty to the model results for children, and these are the results highlighted by the Administrator in the PR.

"The EPA justifies the decision to assume that all individuals from ages 5-18 are equally responsive to ozone as follows:

Clinical studies data for children which could be used to fit the model for children are not available at this time. In the absence of data, we are extending the model to ages 5 to 18 by holding the age term constant at the age 18 level (HREA, p. 6-12).

However, there *is* published data on the effect of ozone on children, and this information should be considered when extending the MSS model to ages 5-17 years. McDonnell et al. (1985b) conducted a chamber study of 23 children between the ages of 8 and 11, and found they had a reduced responsiveness to ozone when compared to an identical chamber exposure protocol of 18-30 year old subjects" (e.g., API).

Response: EPA agrees that this assumption (holding the age term constant at the age 18 level) results in uncertainties, and the HREA discussed the rationale and uncertainties for the age term in Chapter 6. In Section 6.2.4 the modified age term is described; in Section 6.4.2 and Appendix 6D model results are compared with a clinical study with children; in Section 6.5.1.1 the statistical significance of the age term is discussed; Section 6.5.3 and Appendix 6E have results about the effect of the age term, age-relevant factors (e.g., time spent outdoors, ventilation rates), and a sensitivity analysis with an alternative age term.

The statement "McDonnell et al. (1985b) conducted a chamber study of 23 children between the ages of 8 and 11, and found they had a reduced responsiveness to ozone when compared to an identical chamber exposure protocol of 18-30 year old subjects" is not accurate. The authors concluded that "These data indicate that, as a percent of baseline, the mean magnitude of decrements in pulmonary function due to O_3 exposure are similar in children and adults." In another paper in the same year (McDonnell et al., 1985a), the authors

"concluded that with exercise normalized for body size, children appear to be no more responsive to O_3 exposure as measured by pulmonary function than are adults and may experience fewer symptoms."

(10) *Comment:* One commenter focused on a source of uncertainty in the lung function risk results comes from the statistical uncertainty in the MSS model estimates, and that is represented by the standard errors on the model coefficients. In the APEX model the EPA uses the MSS model coefficients to calculate lung function risk, but does not account for these standard errors. The commenter (e.g., API provided new analyses on determining how the statistical uncertainty in the MSS model influences the predictions that come out of the APEX model, and presented distributions reflecting uncertainty in the estimates of risk (e.g., the number of children with lung function decrements of ≥10%). The commenter states that the 95% confidence intervals (from this source of uncertainty alone) of the risk estimates are comparable to the difference in risk between the 70 and 65 ppb alternative standard scenarios.

The commenter states that across all age groups and frequencies of decrements one sees significant overlap in the uncertainty distributions for each ozone scenario, indicating there is uncertainty as to whether a change in ozone standards would lead to a significant reduction in the percentage experiencing this level of lung decrement.

"For example, for the one-day decrement for ages 5-18, 43 out of 100 APEX simulations using the 75 ppb ozone scenario were less than or equal to the maximum value estimated by the 100 APEX simulations using the 70 ppb ozone scenario. Similarly, 23 out of 100 APEX simulations using the 70 ppb ozone scenario were less than the maximum value estimated by the 100 APEX simulations using the 65 ppb ozone scenario." "This overlap becomes especially pronounced when considering decrements for two or more or six or more days, and in some cases the distributions become visually indistinguishable" (e.g., API).

Response: EPA strongly disagrees that overlap in the distributions of the 100 random MSS coefficients indicates uncertainty as to whether a change in O_3 standards would lead to a significant reduction in the proportion of individuals (especially children) experiencing specific lung function decrements.

One cannot do a statistical test of differences in the probability densities between the standards in the standard way because the distributions are completely correlated, e.g. you get the same realization of the uncertainty distribution for each of the standards, thus, when you are at the 5^{th} percentile value for the 75 standard, you are also at the 5^{th} percentile for the 70 ppb and 65 ppb standards, so there will never be an overlap.

Although the percent predicted to experience a single or multiple >10, 15, or 20% FEV_1 decrements varies depending on which MSS coefficients are used, the distributions of the 100 MSS coefficients clearly show a shift toward a larger

proportion affected with increasing O_3 concentration for children (5-18 years) in all of the simulations in the analysis provided by the commenter. This indicates that the proportion of children predicted to experience a given decrement in FEV_1 is affected by both O_3 concentration and which of the MSS coefficients are selected. Although which of the MSS coefficients are selected affects the predicted proportion experiencing a specific FEV_1 decrement, for any given selected set of the MSS coefficients there is a clear increase in the proportion of children having given FEV_1 decrement with increasing O_3 concentrations. For example, the right tail (or similarly the mode) of each density plot would be from the same set of MSS coefficients, comparison of these points across the three O_3 concentrations clearly illustrates the effect of O_3.

The distributions of >10% FEV_1 decrements also clearly shift to a greater proportion affected with increasing O_3 concentration in the 19-35 and 36-55 year age groups. As discussed above for children, although which of the MSS coefficients are selected affects the predicted proportion experiencing specific FEV_1 decrements, for any given selected set of MSS coefficients there is a clear increase in the proportion of individuals having given FEV_1 decrement with increasing O_3 concentrations.

(11) *Comment:* One commenter (e.g., API) pointed out that "simulation noise" adds to the uncertainty of the lung function risk estimates, and presents the results of APEX simulations to quantify the simulation noise.

"The results of each APEX simulation rely in part on a set of random variables that are created by taking draws from probability distributions. These random variables include characteristics of the simulated individuals, such as age, location of residence, and individual responsiveness to ozone (*U* in the MSS model), as well as characteristics that vary across time, such as activity level. This means that the results of each APEX run will depend in part on the seed used to generate these random distributions, even if all of the input files for the APEX simulation are identical. This random variability is commonly known as simulation noise, or jitter" (e.g., API).

Response: EPA agrees with the commenter on this point, and discussed this in Chapter 6 (Convergence of APEX Results) of the HREA. As the commenter pointed out, EPA also presented the results of APEX simulations to quantify the simulation noise, and these results agreed with the commenter's. The range of the simulation noise was less than 1%.

The difference in risk estimates between levels of alternative standards is an important measure and the simulation convergence uncertainty is much less for the differences than for the absolute risks for each scenario. This results from the cancellation of the simulation noise when subtracting the risk estimates. This is demonstrated in Langstaff (2015).

(12) *Comment:* Some commenters (e.g., API, NERA, AAM) contended that the uncertainties of the lung function risk assessment are substantial and asserted that the HREA does not appropriately characterize the uncertainty in risk estimates for O_3-induced lung function decrements. Commenters pointed out that there is statistical uncertainty in model coefficients that is not accounted for in risk estimates. One commenter (e.g., API) presented an analysis of this uncertainty, and concluded that there is considerable overlap between risk estimates for standard levels of 75, 70, and 65 ppb, undercutting the confidence in estimated risk reductions for standard levels below 75 ppb.

- "Any decision to tighten ozone standards must be informed by an analysis of the uncertainty in the benefits such a tightening would bring. Unfortunately, this uncertainty was ignored in the HREA. Our analysis reveals that uncertainty plays a major role in our understanding of the potential benefits from tightening ozone standards. These represent meaningful limitations in the scientific evidence and information that affect the strength of inferences that can be drawn regarding the lung function decrement risk estimates that are under consideration in the Proposed Rule" (e.g., API).

- **"**CASAC members did not question why EPA did not present confidence bounds (*i.e.*, confidence intervals, or CIs) for any of the exposure or lung function risk estimates. Importantly, none of the CASAC members commented on how uncertainty should be considered in the interpretation of the risk results, particularly when comparing across different proposed levels of the standard. These are important issues that CASAC should have asked EPA to address" (e.g., U.S. Chamber of Commerce, NAM, Memorandum from J. Goodman and S. Sax to CASAC, *Comments on the CASAC Review of the Health Risk and Exposure Assessment and the Policy Assessment for Ozone*, May 14, 2014. p. 2-3.).

(13) *Comment:* "For the overall estimates of exposures of concern and FEV_1 decrements, the EPA acknowledges that there is substantial variability in these numbers (HREA, pg. 75274), but no confidence intervals are provided in the Proposed Rule – this is misleading and unscientific" (e.g., TCEQ).

Response: The Agency recognizes that there are important sources of uncertainty in the FEV_1 risk assessment. In some cases, these sources of uncertainty can contribute to substantial variability in risk estimates, complicating the interpretation of those estimates. For example, as discussed in the proposal, unlike exposures of concern, the variability in FEV_1 risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels. Given this, and the resulting considerable overlap between the ranges of lung function risk estimates for different standard levels, in the proposal the Administrator viewed lung function risk estimates as providing a more limited

basis than exposures of concern for distinguishing between the degree of public health protection provided by alternative standard levels.

Comparing risk with exposure benchmarks

(14) *Comment:* Some commenters noted that the percentage of children with FEV_1 decrements ≥ 10% was the same as the percentage of children exposed at least once above 60 ppb in an ozone season and asserted that this is an indication that the model is overestimating the risks in children.

> "In pre-meeting comments, Dr. Vedal noted that "it was difficult to accept" that the percentage of children with FEV_1 decrements > 10% was the same as the percentage of children exposed at least once above 60 ppb in an ozone season. Although he did not elaborate on this point, the results appear to reflect the conservative nature of the model and the high likelihood that the model is overestimating the risks in children and in the urban populations as a whole" (e.g., U.S. Chamber of Commerce, NAM, Memorandum from J. Goodman and S. Sax to CASAC, *Comments on the CASAC Review of the Health Risk and Exposure Assessment and the Policy Assessment for Ozone*, May 14, 2014. p. 11.)

Response: EPA agrees that, in certain scenarios, the risk percentages can be equal to or higher than the exposure benchmark percentages. This can occur because the exposure-response function is non-zero for exposures less than 60 ppb. In addition, the lung function risk model shows responses for all combinations of ozone and activity levels, not just moderate exertion for 8 hours as is used in the exposure benchmark analyses.

iii. Risk of O_3-Associated Mortality and Morbidity

In the proposal, the Administrator placed the greatest emphasis on the results of controlled human exposure studies and on quantitative analyses based on information from these studies, and less weight on mortality and morbidity risk assessments based on information from epidemiology studies. The EPA received a number of comments on its consideration of epidemiology-based risks, with some commenters expressing support for the Agency's approach and others expressing opposition.

(1) *Comment:* In general, commenters representing industry organizations or states opposed to revising the current primary O_3 standard agreed with the Administrator's approach in the proposal to viewing epidemiology-based risk estimates, though these commenters reached a different conclusion than the EPA regarding the adequacy of the current standard. In supporting their views, these commenters highlighted a number of uncertainties in the underlying epidemiologic studies, and concluded that risk estimates based on information from such studies do not provide an appropriate basis for revising the current standard. For example, commenters noted considerable spatial heterogeneity in health effect associations; the potential for co-occurring pollutants (e.g., $PM_{2.5}$) to

confound O_3 health effect associations; and the lack of statistically significant O_3 health effect associations in many of the individual cities evaluated as part of multicity analyses. In contrast, some commenters representing medical, public health, or environmental organizations placed greater emphasis than the EPA on epidemiology-based risk estimates. These commenters asserted that risk estimates provide strong support for a lower standard level, and pointed to CASAC advice to support their position.

Response: As in the proposal, the EPA continues to place the greatest weight on the results of controlled human exposure studies and on quantitative analyses based on information from these studies (particularly exposures of concern, as discussed in sections II.B.3 and II.C.4 of the preamble to the final rule), and less weight on risk analyses based on information from epidemiologic studies. In doing so, the Agency continues to note that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following specific O_3 exposures. In addition, the effects reported in these studies are due solely to O_3 exposures, and interpretation of study results is not complicated by the presence of co-occurring pollutants or pollutant mixtures (as is the case in epidemiologic studies). The Agency further notes the CASAC judgment that "the scientific evidence supporting the finding that the current standard is inadequate to protect public health is strongest based on the controlled human exposure studies of respiratory effects" (Frey, 2014b, p. 5). Consistent with this emphasis, the HREA conclusions reflect relatively greater confidence in the results of the exposure and risk analyses based on information from controlled human exposure studies than the results of epidemiology-based risk analyses. As discussed in the HREA (section 9.6), several key uncertainties complicate the interpretation of epidemiology-based risk estimates, including the heterogeneity in O_3 effect estimates between locations, the potential for exposure measurement errors in these epidemiologic studies, and uncertainty in the interpretation of the shape of concentration-response functions at lower O_3 concentrations. Commenters who opposed the EPA's approach in the proposal to viewing the results of quantitative analyses tended to highlight aspects of the evidence and CASAC advice that were considered by the EPA at the time of proposal and nothing in these commenters' views has changed those considerations. Therefore, the EPA continues to place the most emphasis on using the information from controlled human exposure studies to inform consideration of the adequacy of the primary O_3 standard.

However, while the EPA agrees that there are important uncertainties in the O_3 epidemiology-based risk estimates, the Agency disagrees with industry commenters that these uncertainties support a conclusion to retain the current standard. As discussed below, the decision to revise the current primary O_3 standard is based on the EPA's consideration of the broad body of scientific evidence, quantitative analyses of O_3 exposures and risks, CASAC advice, and public comments. While recognizing uncertainties in the epidemiology-based risk estimates here, and giving these uncertainties appropriate consideration, the Agency continues to conclude that these risk estimates contribute to the broader

body of evidence and information supporting the need to revise the primary O_3 standard.

(2) *Comment:* Some commenters opposed to revising the current $_{O3}$ standard highlighted the fact that, in a few urban study locations, larger risks are estimated for standard levels below 75 ppb than for the current standard with its level of 75 ppb. For example, TCEQ states that "differential effects on ozone in urban areas also lead to the EPA's modeled increases in mortality in Houston and Los Angeles with decreasing ozone standards." These commenters cited such increases in estimated risk as part of the basis for their conclusion that the current standard should be retained. Some commenters additionally asserted that, "most of the study areas display relatively limited reduction in ozone-attributable risk across the three alternative standards" and concluded that reducing the ozone NAAQS will "not have a health benefit" (e.g., Texas Pipeline Association).

Response: For communities across the U.S. (including in the Houston and Los Angeles areas), exposure and risk analyses indicate that reducing emissions of O_3 precursors (NO_X, VOCs) to meet a revised standard with a level of 70 ppb will substantially reduce the occurrence of adverse respiratory effects and mortality risk attributable to high O_3 concentrations (HREA, Appendix 9A; PA, sections 4.4.2.1 to 4.4.2.3). However, because of the complex chemistry governing the formation and destruction of O_3, some NO_X control strategies designed to reduce the highest ambient O_3 concentrations can also result in increases in relatively low ambient O_3 concentrations. As a result of the way the EPA's epidemiology-based risk assessments were conducted (HREA, Chapter 7), increases estimated in low O_3 concentrations impacted mortality and morbidity risks, leading to the estimated risk increases highlighted by some commenters. However, while the EPA is confident that reducing the highest ambient O_3 concentrations will result in substantial improvements in public health, including reducing the risk of O_3-associated mortality, the Agency is far less certain about the public health implications of the changes in relatively low ambient O_3 concentrations (79 FR at 75278/3, 75291/1, and 75308/2). Therefore, reducing precursor emissions to meet a lower O_3 standard is expected to result in important reductions in O_3 concentrations from the part of the air quality distribution where the evidence provides the strongest support for adverse health effects.

Specifically, for area-wide O_3 concentrations at or above 40 ppb,[65] a revised standard with a level of 70 ppb is estimated to reduce the number of premature deaths associated with short-term O_3 concentrations by about 10%, compared to the current standard. In addition, for area-wide concentrations at or above 60 ppb, a revised standard with a level of 70 ppb is estimated to reduce O_3-associated

[65] The ISA concludes that there is less certainty in the shape of concentration-response functions for area-wide O_3 concentrations at the lower ends of warm season distributions (i.e., below about 20 to 40 ppb) (ISA, section 2.5.4.4).

premature deaths by about 50% to 70%.[66] The EPA views these results, which focus on the portion of the air quality distribution where the evidence indicates the most certainty regarding the occurrence of adverse O_3-attributable health effects, not only as supportive of the need to revise the current standard (section II.B.3 of the preamble to the final rule), but also as showing the benefits of reducing the peak O_3 concentrations associated with air quality distributions meeting the current standard (section II.C.4 of the preamble to the final rule).

In addition, even considering risk estimates based on the full distribution of ambient O_3 concentrations (i.e., estimates influenced by decreases in higher concentrations and increases in lower concentrations), the EPA notes that, compared to the current standard, standards with lower levels are estimated to result in overall reductions in mortality risk across the urban study areas evaluated (PA, Figure 4-10). As discussed in the preamble to the final rule (sections II.A.2.a and II.A.2.c), analyses in the HREA indicate that these overall risk reductions could understate the actual reductions that would be experienced by the U.S. population as a whole.

For example, the HREA's national air quality modeling analyses indicate that the HREA urban study areas tend to underrepresent the populations living in areas where reducing NO_X emissions would be expected to result in decreases in warm season averages of daily maximum 8-hour ambient O_3 concentrations.[67] Given the strong connection between these warm season average O_3 concentrations and risk, risk estimates for the urban study areas are likely to understate the average reductions in O_3-associated mortality and morbidity risks that would be experienced across the U.S. population as a whole upon reducing NO_X emissions (HREA, section 8.2.3.2).

In addition, in recognizing that the reductions in modeled NO_X emissions used in the HREA's core analyses are meant to be illustrative, rather than to imply a particular control strategy for meeting a revised O_3 NAAQS, the HREA also conducted sensitivity analyses in which both NO_X and VOC emissions reductions were evaluated. In all of the urban study areas evaluated in these analyses, the increases in low O_3 concentrations were smaller for the NO_X/VOC emission reduction scenarios than the NO_X only emission reduction scenario (HREA, Appendix 4D, section 4.7). This was most apparent for Denver, Houston, Los Angeles, New York, and Philadelphia. These results suggest that in some locations, optimized emissions reduction strategies could result in larger

[66] Available experimental studies provide the strongest evidence for O_3-induced effects following exposures to O_3 concentrations corresponding to the upper portions of typical ambient distributions. In particular, as discussed above, controlled human exposure studies showing respiratory effects following exposures to O_3 concentrations at or above 60 ppb.

[67] Specifically, the HREA urban study areas tend to underrepresent populations living in suburban, smaller urban, and rural areas, where reducing NO_X emissions would be expected to result in decreases in warm season averages of daily maximum 8-hour ambient O_3 concentrations (HREA, section 8.2.3.2).

reductions in O_3-associated mortality and morbidity than indicated by HREA's core estimates.

Thus, the patterns of estimated mortality and morbidity risks across various air quality scenarios and locations have been evaluated and considered extensively in the HREA and the PA, as well as in the proposal. Epidemiology-based risk estimates have also been considered by CASAC, and those considerations are reflected in CASAC's advice. Specifically, in considering epidemiology-based risk estimates in its review of the HREA, CASAC stated that "[a]lthough these estimates for short-term exposure impacts are subject to uncertainty, the CASAC is confident that that the evidence of health effects of O_3 presented in the ISA and Second Draft HREA in its totality, indicates that there are meaningful reductions in mean, absolute, and relative premature mortality associated with short-term exposures to O_3 levels lower than the current standard" (Frey, 2014a, p. 3). Commenters' views on this issue are not based on new information, but on an interpretation of the analyses presented in the HREA that is different from the EPA's, and CASAC's, interpretation. Given this, the EPA's considerations and conclusions related to this issue, as described in the proposal and as summarized briefly above, remain valid. Therefore, the EPA does not agree with commenters who cited increases in estimated risk in some locations as supporting a conclusion that the current standard should be retained.

(3) *Comment:* For risk estimates of respiratory mortality associated with long-term O_3, several industry commenters supported placing more emphasis on threshold models, and including these models as part of the core analyses rather than as sensitivity analyses.

Response: The EPA agrees with these commenters that an important uncertainty in risk estimates of respiratory mortality associated with long-term O_3 stems from the potential for the existence of a threshold. Based on sensitivity analyses included in the HREA in response to CASAC advice, the existence of a threshold could substantially reduce estimated risks. CASAC discussed this issue at length during its review of the REA and supported the EPA's approach to including a range of threshold models as sensitivity analyses (Frey, 2014a, p. 3). Based in part on uncertainty in the existence and identification of a threshold, the HREA concluded that lower confidence should be placed in risk estimates for respiratory mortality associated with long-term O_3 exposures (HREA, section 9.6). This uncertainty was also a key part of the Administrator's rationale for placing only limited emphasis on risk estimates for long-term O_3 exposures. In her final decisions, discussed in the preamble to the final rule (sections II.B.3, II.C.4.b, and II.C.4.c), the Administrator continues to place only limited emphasis on these estimates. The EPA views this approach to considering risk estimates for respiratory mortality as generally consistent with the approach supported by the commenters noted above.

In addition to the comments addressed above, and in the preamble to the final rule, EPA received a number of technical comments on the risk of O_3-associated mortality and morbidity. These comments are addressed below.

(4) *Comment:* Several commenters state that the heterogeneity and sensitivity of ozone effect estimates to a variety of covariates calls into question whether short-term ozone exposure is causally related to mortality and consequently whether available effect estimates can be used to reliably estimate reductions in mortality associated with specific ozone reductions. Commenters also point to the fact that unadjusted city-specific effects used in modeling mortality range from positive to negative, as support for the assertion that there is significant model uncertainty associated with specifying these effects. They also note that there is a spatial and temporal pattern to the data (mortality effects) that is not consistent with ozone causality. Furthermore, commenters point to non-statistically significant mortality estimates and increase in mortality in some cities (specifically in modeling the 70ppb standard in the HREA) as suggesting that there would not be any mortality benefit from a 70ppb standard. Commenters also assert that EPA's modeling of risk at low ozone concentrations cannot be justified since it is not possible to identify a threshold in modeling ozone risk due to exposure uncertainty. Furthermore, commenters cite language from the ozone ISA stating that reduced sampling at low ozone concentrations combined with the potential for exposure measurement error (and other sources of uncertainty identified) can obscure the existence of a threshold should one be present. They further assert that these sources of uncertainty can also result in application of a linear response model even when in reality, a non-linear model would be more appropriate. Collectively, these factors point to there being less confidence in the size of the ozone effect at lower concentrations, which they feel is not acknowledged sufficiently by EPA.

Response: EPA disagrees with this comment. Based on the body of scientific evidence, the ISA concludes that there is likely to be a causal association between short-term ozone exposure and mortality (ISA, p. 1-5). This conclusion was supported by CASAC (Frey and Samet, 2012b, p. 2). In the HREA we acknowledge that spatial heterogeneity is a key source of variability associated with mortality effect estimates (HREA, p. 7-36) from the epidemiology studies and can be related to a number of factors including differences in O_3-attributable factors (including confounders), the degree of averting behavior, and variation in sample sizes which can impact stability of effect estimates. We also acknowledge that variation in sample size, among other factors (e.g., exposure measurement error), can result in wider confidence intervals and in some cases, negative estimates. For that reason, the HREA included Bayes-adjusted city-specific effect estimates reflecting application of both a regional- and national-prior, both of which are intended to capture cross-city differences in effect estimates the mortality endpoint, while still reflecting input from the more stable regional, or national-level signal. Regarding increases in mortality risk generated for some cities (with the 70ppb standard level), as discussed in the HREA, these reflect non-linearities related to ozone formation. Furthermore, we note in the HREA (p. 7-75) that decreases relative to baseline in risk tend to occur on days with

composite monitor O_3 concentrations ranging from 40-70ppb, while increases in risk tend to occur on days with composite monitor values in the range at or below 30-40 ppb (with most risk increases falling in the range of 15ppb to 40ppb). As noted in section 7.1.1 of the HREA, there is less confidence in characterizing the nature of the C-R function (and therefore less confidence in specifying risk) in the range below 20 ppb, relative to estimates of mortality at higher ozone concentrations.

EPA disagrees that ozone-related effects at lower concentrations are driven primarily by copollutants and/or a mismatch between personal and ambient exposures. While we acknowledge in the ISA that there is reduced confidence in specifying the shape of the C-R function for short-term O_3-attributable respiratory morbidity and mortality in the range generally below 20 ppb (for both 8-hr maximum and 24-hr metrics) (ISA, section 2.5.4.4) due to a reduced number of observations at those lower concentrations, the linear no-threshold model is still the best fit for the observed data. For that reason, we employ non-threshold models in modeling ozone reflecting the discussion of the relevant studies in the ISA (section 2.5.4.4).

EPA acknowledges that specific factors identified in the ISA (p. 2-32 and 2-33) reduce confidence in the characterization of the concentration-response function (including potential thresholds) for ozone in the lower range of exposure. These factors include heterogeneity in the ozone effect and reduced data density in the lower ozone concentration range reflected in studies. However, when all of the evidence is considered together, the ISA concludes that there is support for a linear, no threshold C-R relationship (for multiple health effects reflecting both short- and long-term ozone exposure) across the range of 8-h max and 24-h avg O_3 on concentrations most commonly observed in the U.S. during the O_3 season (i.e., greater than 20 ppb). As acknowledged in the HREA, there is less certainty in specification of the shape of the C-R curve at the lower end of the distribution of O_3 data, which corresponds to 8-h max and 24-h avg O_3 concentrations generally below 20 ppb.

(5) *Comment:* Some commenters support the Administrator's decision to place reduced emphasis on epidemiology-based risk estimates and identify a number of sources of uncertainty that they say significantly impact the risk estimates, including: use of concentration-response functions without threshold, the influence of regional heterogeneity in mortality effects, the use of area-wide averages of air monitoring data, and the influence of co-pollutants on model results.

Response: EPA agrees in part with the commenter's description of the Administrator's rationale for reduced emphasis on the epidemiology-based risk estimates; however, the commenter is incorrect in calling out co-pollutant modeling as a factor in that decision. In placing reduced emphasis on the epidemiology-based risk estimates in the current review, the Administrator cites the following factors: heterogeneity in effect estimates between epidemiologic

study areas, the potential for epidemiologic-based exposure measurement error, uncertainty in the interpretation of the shape of concentration-response functions at lower O_3 concentrations, and the use of concentration-response relationship developed for a particular population in a particular location to estimate health risks in different populations and locations (Proposed Rule, 79 FR 75276). However, we note that the influence of co-pollutants or exclusion of threshold models did not factor into this decision to deemphasize ozone-epidemiology-based risk estimates in this review. Regarding copollutants, the ISA states that short-term mortality associations remained relatively robust to inclusion of PM (as seen in Stafoggia et al., 2010 and Katsouyanni et al., 2009) although the ISA acknowledges that the interpretation of these studies was complicated by the different PM sampling schedules (ISA, p. 6-258). Regarding the issue of threshold models, the ISA concludes that the relationship between concentration and response is linear along the range of O_3 concentrations observed in the U.S., with no indication of a threshold within that range. However, the ISA acknowledges that there is less certainty in specifying the nature of the concentration-response function at O_3 concentrations generally below 20 ppb (ISA, section 2.5.4.4).

(6) *Comment:* Several commenters note that many of the effect estimates used in the risk assessment include zero in their confidence intervals, which suggests ozone could have no effect.

Response: EPA disagrees with the commenter that the presence of non-statistically significant effect estimates suggests a lack of ozone-related health impacts for the set of health endpoints modeled in the HREA. For each of the health endpoints modeled in the HREA, the relationship with O_3 has been classified by EPA as being either a *causal relationship* or *likely to be a causal relationship* based on a comprehensive review of the available evidence (HREA, p. 3-19). These causal classifications have been reviewed by CASAC as part of their review of the ISA. Therefore, it is incorrect to interpret non-significant effect estimates for a particular endpoint included in the HREA as suggesting that there is not an actual association between exposure and that health effect. Other factors, such as a lack of statistical power, can play a role in widening confidence intervals for effects such that they encompass zero. Furthermore, it is important to point out that EPA places greatest emphasis on central tendency estimates of risk in the HREA and relies on the associated 95 percent confidence interval to provide additional perspective on overall confidence related to that specific point estimate (specifically uncertainty related to the statistical fit of that estimate).

(7) *Comment:* Commenters cite uncertainty resulting from our application of CRFs to study areas (CBSAs) larger than those used in the original epi studies. The commenter also states that EPA should calculate population weighted ozone responses to lower proposed standards and recalculate health benefits associated with lower proposed standards.

Response: Although EPA agrees that the approach of modeling risk at the CBSA-level adds uncertainty, the Agency disagrees that this significantly compromises

the risk estimates generated. As discussed in detail in section 7.1.1 of the HREA, the decision to model risk at the larger CBSA-level reflects a desire to more fully capture the spatial patterns of changes in risk in the broader urban area when simulating alternative standard levels (for some of the urban study areas modeled, core urban areas experience can experience net increases in risk reflecting non-linearities in ozone formation, while the remainder of those urban areas experience reductions in risk). Modeling risk at the broader CBSA-level insures that the complete picture of changes in ozone-related risk at the urban-level is reflected. However, we also acknowledge (and discuss in section 7.1.1 of the HREA), the uncertainty that is introduced into the assessment of risk through application of effect estimates (originally derived using smaller core urban areas) to larger CBSAs. Ultimately, the decision to model risk at the broader CBSA-level reflected a greater weight placed on reducing a potentially large source of recognized bias (i.e., only focusing on urban core-related risk) than on any additional uncertainty introduced into the analysis. The CASAC supported the EPA's decision to model risk at the CBSA-level rather than at the central urban area-level (Frey, 2014a, p. 2). Regarding the recommendation that EPA derive population-weighted responses to ozone at lower exposure levels, we would point out that derivation of de-novo effect estimates (essentially a new epidemiology study), if that is what is intended by the comment is not feasible.

(8) *Comment:* Several commenters criticize our use of nationally-adjusted city-specific Bayes CRFs for short-term mortality. They cite Smith's comments and recommendation that we should have focused on regional adjustment-Bayes CRFs. While these commenters acknowledge that we did include regionally-adjusted effect estimates in the sensitivity analyses completed for the risk assessment, they assert that they should have been used in generating core risk estimates (in place of the nationally-adjusted effect estimates that were used).

Response: EPA disagrees with the assertion that we should not have used nationally-adjusted city-specific effect estimates in modeling short-term mortality. The decision to use Bayes-adjusted effect estimates obtained from Smith (HREA, section 7.3.2) reflecting a national prior in the core analysis (rather than a regional prior) reflects consideration for the tradeoff between (a) generating more stable city-specific effect estimates which still capture a degree of spatial heterogeneity (application of the national prior with its greater sample size) and (b) generating less stable effect estimates which may capture more fully regional heterogeneity (application of the regional prior). For the final HREA, we decided to focus on effect estimates reflecting the national prior for the core analysis. The CASAC in their review of the 1st draft HREA expressed support for the effect estimates used in modeling risk (which included effect estimates for short-term mortality obtained from Bell, reflecting application of a national prior). Given that the Smith et al. (2009) analysis essentially reproduced the Bell et al. (2004) analysis (albeit with application of additional models and diagnostic analyses), we decided for the core analysis in the 2nd draft (and final HREA) to use effect estimates from Smith et al. (2009) reflecting application of national priors.

(9) *Comment:* Commenters suggest that there is evidence for a threshold in ozone-related health effects (including short-term mortality) and they point to controlled human exposure studies for support as well as a series of epidemiology studies including Stylianou and Nicolich (2009), Xia and Tong (2006), Smith et al. (2009), Bell et al. (2006), and Katsouyanni et al. (2009). Some of the commenters also cite Rhomberg et al. (2011) in making the point that exposure measurement error associated with ozone can mask the presence of a threshold. Commenters further argue that in modeling long-term exposure-related mortality, EPA should include threshold-based models in the core analysis. They also conduct analyses incorporating thresholds into short-term mortality functions and demonstrating substantial reductions in mortality estimates when thresholds are included. Commenters also note that the Proposed Rule does not present the results of sensitivity analyses where EPA incorporated thresholds into its modeling of long-term mortality.

Response: EPA disagrees that the threshold model for short-term mortality should have been in the core HREA analysis. Regarding the modeling of the relationship between short-term O_3 exposure and mortality and the issue of a potential threshold, we first note the conclusion drawn in the ISA following a rigorous evaluation of existing literature, which was itself subjected to peer-review by the CASAC. The ISA states, "In conclusion, the evaluation of the O_3-mortality C-R relationship did not find any evidence that supports a threshold in the relationship between short-term exposure to O_3 and mortality within the range of O_3 concentrations observed in the United States. Additionally, recent evidence suggests that the shape of the O_3-mortality C-R curve remains linear across the full range of O_3 concentrations." (ISA, p. 6-257). However, the ISA does note that efforts to identify whether population-level thresholds exist through national analyses are complicated due to the heterogeneity in the O_3-mortality association observed across cities and regions and the tendency of potential effect modifiers to vary regionally.

Turning specifically to the studies cited by the commenters. Both Xia and Tong (2006) and Stylianou and Nicholich (2009), used a new statistical model developed by Xia and Tong (2006) to examine for evidence of potential thresholds by accounting for the cumulative and nonlinear effects of air pollution using a weighted cumulative sum for each pollutant, with the weights (non-increasing further into the past) derived by a restricted minimization method. As detailed in the ISA, while Xia and Tong (2006) did note that there was evidence of a threshold effect around 24 h avg concentrations of 25 ppb, the threshold values estimated in the analysis were sometimes in the range where data density was low and thus may have been unable to identify modest increases in risk, indicative of a non-threshold relationship. Stylianou and Nicolich (2009) found that the estimated O_3 mortality risks varied across the nine cities examined in the study with the models exhibiting apparent thresholds, in the 10-45 ppb range for O_3 (3-day accumulation). It should be noted that Xia and Tong (2006) did not include a smooth function of days to adjust for unmeasured temporal confounders, and instead adjusted for season using a temperature term. As a

result, these results need to be viewed with caution because some potential temporal confounders (e.g., influenza) do not always follow seasonal patterns of temperature; whereas, Stylianou and Nicolich (2009) included a smooth function of time to adjust for seasonal/temporal confounding, which could explain the difference in results between the two studies. Overall, the results of both Stylianou and Nicolich (2009) and Xia and Tong (2006) are consistent with the conclusions of the ISA regarding potential thresholds. Smith et al. (2009) conducted an analysis similar to that detailed in Bell et al. (2006), but used a reverse subset approach to examine the mortality C-R function relationship. Specifically, Smith et al. (2009) removed days with 24-h avg O_3 concentrations below 20, 30, 40, 50, and 60 ppb. The authors reported consistent positive associations at all cutpoints up to 60 ppb where the total number of days with 24-h avg O_3 concentrations above are so limited that the variability around the central estimate is increased substantially. Regarding the observation from Rhomberg et al. (2011) provided by the commenter, we note that this paper is a review paper that does not provide any new evidence regarding the ozone-mortality C-R relationship.

Regarding potential thresholds in long-term exposure-related respiratory mortality, EPA's decision to include a non-threshold model in the core analysis (and include a range of threshold models as sensitivity analyses) was supported by CASAC (Frey, 2014a, p. 3). Specifically, CASAC noted that while the model including a threshold at 56 ppb was shown to be a better predictor using a less stringent statistical test, none of the threshold models performed better than a linear model when more stringent tests were employed. Furthermore, they noted that confidence intervals associated with the suite of threshold models considered suggest that if a threshold exists, it could fall anywhere between 0 and 60 ppb. Given these results, CASAC concurred with EPA's planned approach to include a non-threshold model as a core analysis. In addition, the ISA states (in relation to the Jerrett exploration of potential thresholds) that, "Ozone threshold analyses indicated that the threshold model was not a better fit to the data ($p > 0.05$) than a linear representation of the overall O_3-mortality association." (ISA, p. 7-88). This further supports observations made by CASAC in relation to this issue.

(10) *Comment:* Commenters argue that EPA should use NAS's IUA (integrated uncertainty analysis) to evaluate overall uncertainty in modeling mortality risk. Commenters further assert that when an IUA is completed for long-term respiratory mortality, 10 of the 12 urban study areas are projected to experience no risk reduction. This they argue, suggests that there is little support for reducing the standard. Note, also that the commenter state that when risk estimates are shown to be variable (based on sensitivity analyses), it is "…standard practice in risk analysis is to provide an IUA that incorporates uncertainty on each sensitive input assumption, and brings results of that analysis to the forefront of the report" (e.g., EPRI).

Response: EPA agrees that given sufficient time and resources, application of an IUA has the potential to provide a more rigorous and complete characterization of

uncertainty. However, as the commenter observes, application of IUA requires specification of confidence distributions of key input parameters representing important sources of uncertainty. The specification of these confidence distributions is a critical step in implementing an IUA since these drive the outcome of the simulation. In addition, as also noted by the commenter, it is also important to specify any correlations between key inputs since these can also significantly impact the uncertainty analysis. . The method used to establish confidence distributions (and any potential correlations between parameters) is critical to both the outcome of the IUA as well as the scientific defensibility of the overall analysis and can be both time- and resource-intensive. This reflects the fact that, depending on analysis involved, experts can differ in their characterization of confidence for key input factors. The commenter recognizes this when they state that, "it should be noted that other professionals familiar with uncertainty analysis and subjective judgment might draw different conclusions [compared with subjective judgement exercised by the commenter in their IUA example] from a review of the same information, or may bring in additional information that has not been considered" (e.g., Association of Electric Companies). The commenter notes (and EPA agrees) that expert elicitation can provide a method for developing these confidence intervals and defining any correlations between parameters. In fact, EPA used this type of approach in the last PM NAAQS review in developing a suite of distributions characterizing uncertainty in the long-term mortality effect for $PM_{2.5}$. However, conducting an expert elicitation was beyond the scope of the HREA. As a result, for many important inputs to the HREA analyses, we found that there was insufficient information available to provide defensible specifications of distributions around those key input parameters. Thus we determined that a defensible IUA would not be possible for the HREA.

(11) *Comment:* Several commenters note that the HREA presents positive risk estimates without indicating how or whether those estimates are scientifically or statistically significant. Furthermore, they assert that given EPA's reliance on a few conflicting studies, the commenters assert that EPA does not have a solid body of evidence on which to base a lowering of the standard.

Response: EPA disagrees with this comment. The CASAC supported EPA's approach including the studies selected (Frey and Samet, 2012b, p. 3). The HREA present core risk estimates for short-term mortality and morbidity including confidence intervals that reflect the statistical fit of the underlying effect estimates (HREA, Table 7-7 for mortality and Appendix 7B for morbidity). As we discuss in the final HREA (p. 7-65), when an effect estimate is drawn from a study with low statistical power, confidence intervals can be wide, and can include negative values because of the assumptions of normality in the distribution of the effect estimate. Negative lower-confidence bounds do not imply that additional exposure to O_3 has a beneficial effect, but rather that the estimated O_3 effect estimate in the C-R function was not statistically significantly different from zero, and thus has a higher degree of uncertainty as to the magnitude of the estimated risk.

Contrary to the commenters' assertion, EPA has provided clear indication that risk estimates are scientifically significant. All of the health effects endpoints included in the risk assessment were classified as having either a causal or likely causal association with ozone exposure in the ozone ISA (Table 1-1), which was peer-reviewed by CASAC. In addition, the specific suite of health endpoints selected for the HREA was also supported by CASAC (Frey and Samet, 2012b).

(12) *Comment:* Commenters criticize the EPA's modeling of risk below background and down to zero and assert that this approach towards risk modeling represents a break from past EPA practice and is not supported by available science. Other commenters have noted that a significant portion of EPA's modeled epidemiology-based risk occurs at ozone concentrations below background (0.04 to 0.06 ppm). Other commenters have also referenced background, asserting that from 51-74% of modeled mortality risk occurs at ozone levels below background. Furthermore, commenters assert that the CAA charges EPA with addressing pollution in setting NAAQS and that naturally-occurring ozone does not fit into this category and therefore, modeling risk for areas dominated by naturally-sourced ozone is not supported (they cite data form Big Bend and Houston Tx which, the assert, shows elevated ozone concentrations and significant contributions from both naturally-occurring and internationally-transported precursors).

Response: EPA agrees that this HREA has a different treatment of background concentrations than previous REAs. EPA adopted the new approach based on the recognition that individuals and populations are exposed to total O_3 from all sources, and risks associated with O_3 exposure are due to total O_3 exposure and do not vary for O_3 exposure associated with any specific source including background sources, regardless of how they may be defined. (HREA, section 2.5.2). The approach taken in the HREA was supported by CASAC (Frey and Samet, 2012b, p.2). In addition, because of the methodology we used to adjust O_3 air quality, estimates of risk changes resulting from just meeting alternative standards only reflect changes in U.S. anthropogenic emissions. As discussed in section 2.2.3 of the Final HREA, for this assessment, we have employed a sophisticated approach (Higher-Order Decoupled Direct Method (HDDM) capabilities in the Community Multi-scale Air Quality (CMAQ) model) to simulate attainment of both the current and alternative standard levels. This modeling incorporates all known emissions, including emissions from both natural and anthropogenic sources within and outside of the U.S. By using the model-adjustment methodology we are able to more realistically simulate the temporal and spatial patterns of O_3 response to precursor emissions. Therefore, O_3 concentrations associated with background sources, e.g. nonanthropogenic and international emissions are accounted for by the modeling approach used to simulate each of the air quality scenarios, which removes the need for considering them separately during calculation of changes in risk as had been done in assessments for earlier reviews.

(13) *Comment:* Commenters note that, following CASAC advice, EPA is not applying the LML from a particular epidemiology study (used in deriving CR functions) as a threshold in modeling risk. The commenters observe, however that in past NAAQS risk assessments, EPA has used this approach because of the greater confidence associated with applying an effect estimate across the range of ambient ozone associated with the study underlying that function.

Response: EPA agrees that in the HREA completed for the last $PM_{2.5}$ NAAQS review (U.S. EPA, 2010b), EPA utilized LMLs to generate higher-confidence estimates of long-term mortality because effect estimates for this endpoint were based on an air metric (annual average) that readily lent itself to identification of a clear LML. However, in the previous $PM_{2.5}$ NAAQS HREA, we noted, that a similar approach could not be readily used for short-term endpoints (characterized using time series studies), since they are derived using distributions of daily air metrics which do not have clearly identifiable LMLs (or where LMLs are so low as to make little difference when incorporated into risk modeling). That same observation holds for the current ozone HREA, which focuses primarily on short-term endpoints, including mortality (i.e., application of LMLs as a lower bound for generating higher confidence risk estimates, would not have a significant impact on risk). In the case of long-term respiratory mortality modeled for the current ozone HREA, we did consider the potential for thresholds in effect (rather than LMLs) as part of our sensitivity analysis (HREA, section 7.1.1). Furthermore, EPA acknowledges in the HREA that we have less certainty in specification of the shape of the C-R curve at the lower end of the distribution of O_3 data, which corresponds to 8-h max and 24-h avg O_3 concentrations generally below 20 ppb (HREA, section 7.1.1). In addition, as acknowledged by the commenter, CASAC recommended that we not incorporate LMLs into risk modeling for the current ozone HREA (section 7.1.1).

(14) *Comment:* Several commenters point to evidence from controlled human exposure studies of a 40 ppb threshold as support for a potential threshold in more serious endpoints including mortality. Addressing short-term mortality specifically, commenters note that Smith et al. (2009) suggests the potential for a reduced mortality effect at lower ozone concentrations (15-40 ppb). Commenters note that the Smith et al. (2009) findings suggest the potential for a non-linear ozone effect and go on to assert that the study findings call into question the causality of ozone in relation to short-term mortality.

Response: EPA disagrees that the results of controlled human exposure studies can be used to infer the potential for thresholds in effect estimates obtained from epidemiologic studies. Clinical exposure studies typically involve relatively small human study populations that do not include individuals with pre-existing conditions that would put them at greater risk for the health effect of interest. By contrast, epidemiology studies typically involve large populations of diverse individuals, likely including those with greater susceptibility/vulnerability to the pollutant of interest. For this reason, depending on the health effect, epidemiology studies have the potential to capture health effects that are less frequent in the

population relative to clinical studies. In addition, clinical studies for ozone typically focus on respiratory effects (i.e., FEV_1) which makes them less applicable directly in interpreting epidemiology-based mortality studies, including the potential shape of exposure-response functions.

Regarding the commenters observation that the Smith et al., 2009 study suggests a reduced ozone mortality effect at lower ozone levels (15-40 ppb), EPA would point out that this is based on the *reversed subset approach* which considers the fit of the response function based on data above a given ozone level (as contrasted with considering the fit of a function based on data below a given level as is often used with threshold evaluations). As noted in the Smith study, with this approach, estimates of the effect at higher ozone levels have reduced confidence (which can be seen with the wider confidence intervals in Figure 7 in the study – bottom plot) since they are based on an increasingly smaller subset of the overall data from the study. In fact, likely reflecting the larger confidence interval associated with the beta for the highest ozone range, the Smith study states that "…there is no clear indication that $Beta^3$ [the effect estimate for the highest ozone range] is the largest of the three coefficients (as one might expect if it were true that ozone toxicity is greater at higher concentrations) and it has by far the largest posterior standard deviation of the three estimates." Although the Smith study does present the possibility of a piece-wise linear model (with 2-3 break points and a slope which increases with higher ozone), EPA notes that the bayesian-adjusted city-specific effect estimates that were available for risk modeling based on the study each had a fixed slope and were not piece-wise in form.

(15) *Comment:* Commenters assert that in modeling long-term exposure-related mortality using effect estimates obtained from Jerrett et al. (2009), EPA should have used a model with a threshold at 56 ppb in the core analysis, since this was shown to have the best fit. Furthermore, they argue that, by citing study results based on a p value of 0.06 (rather than 0.05) EPA was able to make a stronger (but incorrect) assertion that the threshold models did not fit any better than the non-threshold model. Commenters also point to work done by Dr. Anne Smith demonstrating that a threshold of 56ppb is equivalent (depending on the urban study area) to a NAAQS level of 70 to 75 ppb (given the form of the standard) and thereby, when applied in modeling risk, results in virtually of the risk disappearing form the estimate.

Response: EPA disagrees that a threshold model should have been used in the core analysis for long-term respiratory mortality. As discussed in the HREA, based on review of the Krewski et al. (2009) study and communications with the study authors, it is not clear whether any of the threshold models considered in the study perform better than the linear model. In addition, it is not possible to clearly differentiate between the different threshold models in terms of fit, leading the study authors to conclude that considerable caution should be exercised in using any specific threshold model (see HREA, p. 7-22). EPA does recognize the significant reduction in estimates of risk that are associated with application of these thresholds, as reflected in the sensitivity analysis results referenced here

(HREA, Figure 7-9). However, the ISA makes the statement that, "Generally, both short- and long-term exposure studies indicate a linear, no threshold C-R relationship when examining the association between O_3 exposure and multiple health effects across the range of 8-h max and 24-h avg O_3 concentrations most commonly observed in the U.S. during the O_3 season (i.e., greater than 20 ppb)" (U.S. EPA, 2012, section 2.5.4.4). EPA acknowledges potential uncertainty associated with the existence and location of a threshold for long-term respiratory mortality and for that reason included consideration for several thresholds as sensitivity analyses, while the linear non-threshold model was used in the core analysis (HREA, section 7.1.1). CASAC supported our approach of including a non-threshold model as the core analysis and including threshold-based models as sensitivity analyses (Frey, 2014b, p. 13-14)

(16) *Comment:* Commenters asserts that EPA's risk estimates are unreliable because the Agency selectively picked data from the studies for use in the HREA by: 1) using concentration-response relationships that represent the average across all cities evaluated (and that the set of cities reflected in the short-term mortality studies are not nationally-representative, which means the averaged effect estimates are not nationally-representative); 2) choosing concentration-response relationships for lag times that resulted in positive associations; and/or 3) using concentration-response relationships not corrected for the influence of particulate matter which often made the associations negative and almost always made them statistically insignificant. In particular, commenters note that there is significant spatial heterogeneity in the ozone-mortality effect due to a number of factors (e.g., activity patterns, air conditioner use, presence of other urban pollutants). The commenters then assert that by using national-average effect estimates for both short- and long-term exposure-related mortality, EPA downplays that spatial heterogeneity and obtains uniformly positive effect estimates for use in modeling risk. The commenters also note that the Agency's approach downplays overall uncertainty by not considering the range of city-specific effect estimates (including negative values) reflected in the underlying epidemiology studies (other commenters specifically pointed to regional heterogeneity, including negative effect estimates presented in Jerrett et al., 2009). The commenters also note that several studies reported no association between ozone and short- and long-term mortality and were not included in EPA's review (Dominici et al., 2005; Goldberg et al., 2006; Lipsett et al., 2011).

Response: EPA disagrees with the commenter that the risk estimates presented in the HREA are unreasonable for the reasons presented. Regarding the use of effect estimates averaged across cities and the potential masking of heterogeneity, for short-term mortality (one of the key endpoints included in the HREA), we did not use nationally-averaged effect estimates. For this endpoint, we used Bayesian-adjusted city-specific effect estimates that reflect both consideration for the city-specific effect (i.e., spatial heterogeneity in the effect) combined with the overall (higher powered) national effect (see HREA, section 7.1.1). As a sensitivity analysis for short-term mortality, we used Bayesian adjusted city specific estimates incorporating regional priors (rather than national priors as used in the

core analysis). Regarding the criticism that the short-term mortality studies providing the Bayesian adjusted effect estimates are based on a set of cities that are not nationally-representative: the number of cities is still relatively large and diverse and consequently is likely to do a reasonable job of capturing urban variation in the ozone-effect across the U.S. For long-term respiratory mortality, the core estimate was based on application of a national-level effect estimate (copollutant model with ozone and $PM_{2.5}$). However, as a sensitivity analysis, we generated risk estimates using regionally-differentiated effect estimates to more fully explore spatial heterogeneity associated with this endpoint.

Regarding consideration for negative effect estimates relating ozone exposure to a reduction in risk (e.g., subset of raw un-adjusted effect estimates for short-term mortality presented in Smith et al., 2009), EPA's believes that it would not be appropriate to use these negative effect estimates in the core risk analysis. Given that each of the health effect endpoints included in the HREA has strong evidence supporting an association between ozone exposure and the health effect of interest (based on causal determinations presented in the ISA, Table 1-1), it is not reasonable to use an effect estimate which would predict a reduction in risk (for that health effect) linked to ozone exposure. In those instances where there is a raw (unadjusted) effect estimate that is negative, it is likely that this reflects some underlying limitation in the input data used in the epidemiology study (e.g., exposure measurement error) and not a true health benefit linked to ozone. For that reason, the use of Bayesian-adjusted effect estimates (that reflect consideration for the full set of city-specific estimates in addition to the city-level effect estimate) is appropriate.

Regarding consideration for lag structures in the selection of effect estimates, as stated the HREA, based on review of available evidence, EPA concluded that there is increased confidence in modeling both short-term O_3-attributable mortality and respiratory morbidity risk based on exposures occurring up to a few days prior to the health effect, with less support for associations over longer exposure periods or effects lagged more than a few days from the exposure (see ISA, section 2.5.4.3,). Consequently, we favored effect estimates reflecting relatively shorter lag structures and within that subset, we favored effect estimates that were larger and had smaller confidence intervals since those effect estimates are more strongly associated with the effect of interest.

Regarding consideration for copollutant models in selecting effect estimates, as noted in the HREA, EPA recognizes the tradeoff between single and copollutants models and for that reason, when possible, have considered including both in modeling specific endpoints (HREA, section 7.3.2). However, as noted in the HREA, copollutants sampling limitations (specifically lower sampling rates for PM relative to ozone) can mean that effect estimates in copollutants models are subject to less precision relative to single pollutant models, which argues for an emphasis on single pollutant models, particularly for short-term exposure-related endpoints. In developing the HREA technical approach, EPA (a) was clear about which types of models (single or copollutant) were used in modeling each

endpoint (b) to the extent possible given available literature, attempted to evaluate uncertainty related to this issue quantitatively through sensitivity analyses including copollutant models and (c) discussed this issue rigorously as part of our qualitative treatment of uncertainty impacting risk estimates (HREA, Table 7-4).

Regarding the set of additional epidemiology studies the commenter asserts does not support an association between ozone and short-term and long-term exposure-related mortality (and was not considered by the EPA in their review), EPA disagrees with one or both of these assertions depending on the study being considered. The Goldberg et al. (2006) study is included in the ISA and therefore, was considered by EPA in selecting studies to use as the basis for risk modeling. Specifically with regard to ozone, this study indicates uniformly positive central estimates (with generally stronger associations in the warmer months) but in the majority of cases, these associations were not statistically significant. However, as discussed earlier, non-statistically significant associations do not necessarily imply a lack of causal association, but rather reduced precision which can result from a number of factors (e.g., low sample size, exposure measurement error). Had EPA attempted to model mortality using effect estimates from Goldberg et al. (2006) it is not clear that the needed baseline incidence rates (and population counts) would have been available at the study area-level for the at-risk populations considered in the study (i.e., individuals within specified age ranges with diabetes and cardiovascular disease). The Dominici et al. (2005) study has been superseded by other, more recent studies based on the National Morbidity, Mortality and Air Pollution Study (NMMAPS) in particular the Smith et al. (2009) study which provided effect estimates used in the HREA. However, consistent with the studies EPA used in the risk assessment, the Dominici et al. (2005) study showed an association between ozone and total mortality in the summer months. The Lipsett et al. (2011) study of long-term exposure (including ozone) and cardiorespiratory disease in California, reports an association between ozone exposure and mortality, stating, "Both measures of ozone in our study suggested an association with nonmalignant respiratory mortality…" However, given our need to identify an effect estimate for long-term respiratory mortality which could be used for the set of 12 urban study areas, we focused on Jerrett et al. (2009) which provides broader national-scale coverage (by contrast, the Lipsett et al. (2011) study is limited to southern California and focused only on women, which limits its applicability in supporting the HREA). Finally, the set of epidemiology studies that were selected for supporting risk modeling was subject to review by CASAC and received broad support initially in CASAC's review of the 1st draft HREA (Frey and Samet, 2012b, p. 2) and following refinements to the methodology including substitution of Smith et al. (2009) for Bell et al. (2004) in modeling short-term mortality (Frey, 2014a, p. 2).

(17) *Comment:* Commenters disagree with the EPA's assertion that risk estimates would have been biased low had modeling been confined to the smaller study areas reflected in the underlying epidemiology studies (supporting derivation of the CR functions) rather than modeling at the larger CBSA level. Instead, the commenter argues that by modeling at the CBSA level, we biased overall risk

estimates (i.e., total incidence) higher by using a larger study area with larger population. Other commenters make a similar assertion, noting that, as EPA acknowledges extrapolating effect estimates to the larger CBSA introduces exposure measurement error. Further they add that, different communities within a larger metropolitan area can vary with regard to socioeconomic class and environmental variables which can modify ozone-related effects.

Response: EPA disagrees with the commenter that modeling risk at the CBSA has resulted in estimates of risk that are biased high. As discussed in the HREA (section 7.1.1), the use of the larger CBSA study areas allows us to better reflect how the change in air quality affects risk across the entire urban area and to avoid introducing known bias into the HREA by focusing risk estimates on that subpopulation living in areas likely to experience potential increases in O_3 (and excluding the larger population of urban and suburban areas likely to experience reductions in ambient O_3 levels). EPA provides estimates of risks using several metrics that normalize for the total population size, including risks per 100,000 population and percent risk. These risk metrics are not affected by the total size of the population in an urban area. EPA did present estimates of risk modeled at the smaller study areas (reflected in the underlying epidemiology study area) as a sensitivity analysis in order to fully explore heterogeneity in the estimates of risk that result from non-linearities in ozone formation (see Figure 7-7 in the HREA). EPA acknowledges that the use of the larger CBSA-based study areas (to addresses this source of known bias), introduces uncertainty into the HREA since the HREA study areas are not matched to the study areas in the epidemiological studies providing the effect estimates used in modeling health effects endpoints (see HREA, Table 7-4).

(18) *Comment:* Commenter point out limitations and uncertainties associated with individual epidemiological studies used as the source of effect estimates used in modeling short-term exposure-related risk in the HREA; the implication being that these factors significantly reduce overall confidence in the risk estimates generated. These limitations, as referenced by the commenters, include: (a) inconsistencies in the patterns of lag effects, (b) instances in which the study areas (cities) used in an epidemiology study were not randomly selected and/or application of study based on a particular urban location to a different location modeled in the risk assessment, (c) sensitivity of effect estimates to ozone seasonality, inclusion of copollutants and model-form (e.g., spline structure), (d) spatial heterogeneity in effect estimates (across regions modeled in a study), (e) value of using multiple studies in modeling risk for the same health effect endpoint (when those studies utilizes the same air metric), (f) evidence that in some epidemiology studies, ambient concentrations (used in deriving effect estimates) are not associated with corresponding personal exposures, (g) subjective reporting of symptoms by mothers of children under study (also involving reporting of non-standard symptoms by mothers using symptom calendars), (h) relatively small study samples involving families where a health condition of concern in the context of the study (asthma) is already present in the family possibly resulting in genetic pre-disposition, and (i) application of effect

estimates generated for a particular set of urban/residential areas to a different urban location modeled in the HREA.

Response: EPA acknowledges that there is uncertainty associated with selection and application of the effect estimates used in the HREA, including uncertainty associated with the initial derivation of those effect estimates as described in the underlying epidemiology studies. In fact, many of the limitations/uncertainties identified by the commenter have also been identified by EPA and are either reflected in the set of sensitivity analyses completed in support of the HREA, or included in the qualitative discussion of uncertainty presented in Table 7-4 of the HREA. It is also important to point out that EPA utilized a systematic process for selecting both the epidemiology studies and associated effect estimates used in both the core and supporting sensitivity analyses completed for the HREA (see section 7.3.2 of the HREA). Not only did EPA describe that approach in detail, it also presented the specific set of epidemiology studies and associated effect estimates chosen for the HREA. Both of the elements of the HREA design (the framework for effect estimates selection and the set of effect estimates chosen) were subjected to CASAC review. The specific set of endpoints and associated effect estimates for the HREA received broad support initially in relation to the 1st draft HREA (Frey and Samet, 2012b, p. 2) and following refinements to the methodology including substitution of Smith et al. (2009) for Bell et al. (2004) in modeling short-term mortality (Frey, 2014a, p. 2). Furthermore, in their review of the 2nd draft HREA, CASAC concluded that the risk assessment completed for short-term mortality (a key risk metric generated in the HREA) was sufficiently robust to support policy analysis in the NAAQS review context. Specifically, they stated that, "Although these estimates for short-term exposure impacts are subject to uncertainty, the CASAC is confident that that the evidence of health effects of ozone presented in the ISA and Second Draft HREA in its totality, indicates that there are meaningful reductions in mean, absolute, and relative premature mortality associated with short-term exposures to ozone levels lower than the current standard and that the mean estimates presented in the Second Draft HREA are useful for policy analysis." (Frey, 2014a).

(19) *Comment:* Several commenters criticize the use of Jerrett et al. (2009) in modeling long-term exposure-related cardiopulmonary mortality. Specifically, they state that: (a) the study did not adequately control for $PM_{2.5}$ (only two years of $PM_{2.5}$ data, versus 24 years for ozone) and did not control for other pollutants (namely SO_2), (b) the study found an inverse association with all-cause and cardiovascular mortality which is counter-intuitive and (c) cities included in the study were not randomly selected which means that the results are not nationally-representative. In addition, commenters also assert that in the HREA, EPA incorrectly states that ISA identified respiratory-related effects including respiratory mortality as likely causal (to support this, they point to Table 2-3 in the ISA).

Response: EPA disagrees with the commenter's criticism of Jerrett et al. (2009) and the use of its effect estimates in the HREA. While we acknowledge that a

shorter (two-year) period of monitored $PM_{2.5}$ data was used in the co-pollutant model (and that the model did not include control for SO_2 specifically) it important to note that the endpoint modeled in the HREA was long-term respiratory mortality, not cardiovascular mortality. Available evidence suggests that long-term ozone exposure is more strongly associated with long-term respiratory-related effects (ISA concluded a "likely to be causal relationship" for long-term ozone exposures and respiratory effects) while long-term $PM_{2.5}$ exposures are more closely strongly associated with cardiovascular effects (2009 PM ISA concluded a "causal relationship" for long-term $PM_{2.5}$ exposures and cardiovascular effects, U.S. EPA, 2009b) than long-term ozone exposures. Consequently, Jerrett et al. (2009), in differentiating cardiopulmonary effects into separate respiratory and cardiovascular components, allowed a stronger differentiation between long-term ozone-related (respiratory) mortality and $PM_{2.5}$-related (cardiovascular) mortality, which allowed for a better examination of the potential impact of long-term exposure on respiratory mortality. As detailed in the ISA, there is greater consistency and coherence in the effect of long-term ozone exposures on respiratory effects than cardiovascular effects. Additionally, due to the relatively small number of respiratory deaths that encompass a combined cardiopulmonary mortality outcome, the use of a combined cardiopulmonary grouping complicates the ability to examine the relationship between long-term ozone exposures and only respiratory mortality. Furthermore, the smaller $PM_{2.5}$-related monitoring period, relative to ozone, is not as much of a concern since the relative rank-order of the locations in the study by long-term average $PM_{2.5}$ concentration is more important that the actual $PM_{2.5}$ concentrations measured in these locations when adjusting for $PM_{2.5}$ as a potential confounder (and it has been shown that these rank orders do not change substantially over time). Regarding the mixed finding for cardiovascular mortality referenced by the commenter, this is also of less of a concern given emphasis placed in the HREA on modeling ozone-related respiratory mortality (with ozone having the stronger association with this endpoint compared with $PM_{2.5}$). Furthermore, given the stronger evidence-based support for long-term exposure to ozone and long-term respiratory effects relative to SO_2 (SO_2 was classified as *inadequate to assign a causal association* for long-term respiratory effects, U.S. EPA, 2008c), there is less concern for not having controlled explicitly for SO_2 in the multipollutant ozone model addressing respiratory mortality. EPA also disagrees with the commenter that we mistakenly assigned long-term respiratory effects including mortality a *likely to be causal* relationship. That is the classification given to this mix of respiratory endpoints (including mortality) in the ISA (see ISA, p. 1-6 and 1-7).

(20) *Comment*: Commenters assert that despite the fact that "much debate continues regarding the level at which truly adverse health effects occur and the relationship between monitored ozone concentrations and hospitalizations/mortality reported" (e.g., Texas Pipeline Association), EPA continues to presume that ozone as low as 60 ppb causes hospitalizations/death. Furthermore other commenters state that in modeling these endpoints, the EPA uses epidemiology studies that do not measure actual exposures not adequately control for other factors which can confound

study results such as other pollutants, population characteristics (age, sex, race), health status (pre-existing conditions, obesity, blood pressure, lack of exercise), and living conditions. Commenters also criticize the assumption in epidemiological studies that people are exposed to a uniform level of air pollution when such factors as behavior lead to variation in exposure (this resulting in measurement error in epidemiological studies). The commenters also note that the effects used by EPA are often uniformly weak, inconsistent and mostly statistically insignificant. And finally, the commenters assert that EPA does not sufficiently evaluate the impact of uncertainty (they use qualitative assessments biased towards identifying factors that could result in under-prediction of risk and they do not included probabilistic quantitative uncertainty analyses).

Response: EPA disagrees that points raised by the commenter call into question the ability of risk estimates presented in the HREA to be used in support of the ozone NAAQS review. All of the endpoints modeled in the HREA are well supported by available evidence with the ISA assigning each of the endpoints either a causal or likely causal association (ISA, section Table 1-1). Regarding the use of epidemiology studies employing composite monitors (or similar exposure surrogates) in risk assessment, the ISA states that specifically with regard to short-term mortality, "…In conclusion, the recent epidemiologic studies [published since the last ozone NAAQS review] build upon and confirm the associations between short-term O_3 exposure and all-cause and cause-specific mortality reported in the 2006 O_3 AQCD" (ISA, p. 6-264). Furthermore, EPA bases the application of linear non-threshold models (in modeling both morbidity and mortality related to short-term ozone exposure) on a rigorous review of the available evidence as presented in the ISA (p. 2-32). CASAC expressed support both for the set of epidemiology studies used in the HREA as well as the application of non-threshold based models in evaluating risk for short-term exposure-related endpoints included in the HREA (Frey and Samet, 2012b, pp. 2 and 15). Furthermore, in their review of the 2nd draft HREA, CASAC concluded that the risk assessment completed for short-term mortality (a key risk metric generated in the HREA), even given uncertainty acknowledged by EPA, was sufficiently robust to support policy analysis in the NAAQS review context. Specifically, CASAC stated that, "Although these estimates for short-term exposure impacts are subject to uncertainty, the CASAC is confident that that the evidence of health effects of ozone presented in the ISA and Second Draft HREA in its totality, indicates that there are meaningful reductions in mean, absolute, and relative premature mortality associated with short-term exposures to ozone levels lower than the current standard and that the mean estimates presented in the Second Draft HREA are useful for policy analysis." (Frey, 2014a).

Regarding the assertion that EPA did not sufficiently evaluate the impact of uncertainty on the risk estimates generated (including failure to complete a probabilistic uncertainty analysis), EPA would point out that, to the extent supported by available data, we included a range of quantitative sensitivity analyses intended to look at key sources of uncertainty (e.g., spatial heterogeneity in effect, model choice and specification including copollutant/single pollutant

modeling, methods used in simulating attainment of alternative standards) (HREA, section 7.4.3). EPA also included probabilistic simulation to integrate statistical fit (in the effect estimates) into estimates of risk. Note, however, that we did not have the data necessary to complete a full probabilistic uncertainty analysis (that would have required use of expert elicitation to derive confidence levels for all key inputs which was not feasible). In addition to the range of quantitative sensitivity analyses completed, EPA also included a rigorous qualitative analysis of key sources of uncertainty including discussion of the potential nature and magnitude of the impact form individual sources of uncertainty on the risk estimates generated (see HREA, Table 7-14).

(21) *Comment*: Commenters questions the EPA's decision to model HA, ER and respiratory symptoms in several cities [using single-city studies] when the Agency had already used multi-city studies (covering all 12 urban study areas) to model HA and ER endpoints. Commenters note that single city studies, while typically providing higher effects are also more variable.

Response: EPA disagrees that single-city studies were used to model specific morbidity endpoints, when multi-city studies were available (and should have been used). Specifically, EPA would point out that the use of single-city studies allows coverage for a range of additional factors that are relevant in providing a more complete picture of risk for these morbidity endpoints, including: (a) coverage for distinct endpoints within these broader endpoint categories (e.g., HA for chronic lung disease, asthma, COPD, respiratory and ED for asthma, respiratory), (b) single versus multipollutant models (e.g., ozone alone, ozone with CO, NO_2, PM_{10}), (c) different model forms (e.g., lag structures, application of different spline models). Together this range of modeling approaches allows the risk assessment to more fully capture the impact of different sources of uncertainty for these morbidity categories (as well as a greater number of endpoints within each category).

(22) *Comment:* Commenters assert that, based on our characterization of epi-based risk, which reads "…most of the study areas display relatively limited reduction in ozone-attributable risk across the three alternative standards," we should conclude that reducing the ozone NAAQS will "not have a health benefit" (e.g., Texas Pipeline Association). The commenters also note that some cities (Houston) have short-term exposure-related mortality estimates reflecting a moderate increase in risk in going from baseline to the current and alternate standards (they also note that this same pattern is seen in Los Angeles for HA). These observations regarding a potential risk disbenefit were also made by other commenters. Based on these observations, the commenters conclude that in Houston and Los Angeles, decreasing the ozone NAAQS has the potential to worsen public health.

Response: The interpretation of the magnitude of predicted risk reductions and implications in terms of public health protection is a policy-relevant comment and

does not represent a technical criticism of the methodologies or data used in the HREA and consequently will not be addressed here.

Regarding the presence of potential increases in risk under simulation of alternative standard levels in several of the cities including Houston, EPA has acknowledged the presence of these predicted potential risk increases (see HREA, section 7.5.2). For communities across the U.S. (including in the Houston and Los Angeles areas), exposure and risk analyses indicate that reducing emissions of O_3 precursors (NO_X, VOCs) to meet a revised standard with a level of 70 ppb will substantially reduce the occurrence of adverse respiratory effects and mortality risk attributable to high O_3 concentrations (HREA, Appendix 9A; and sections 4.4.2.1 to 4.4.2.3). However, because of the complex chemistry governing the formation and destruction of O_3, some NO_X control strategies designed to reduce the highest ambient O_3 concentrations can also result in increases in relatively low ambient O_3 concentrations. As a result of the way the EPA's epidemiology-based risk assessments were conducted (HREA, Chapter 7), increases estimated in low O_3 concentrations impacted mortality and morbidity risks, leading to the estimated risk increases highlighted by some commenters. However, while the EPA is confident that reducing the highest ambient O_3 concentrations will result in substantial improvements in public health, including reducing the risk of O_3-associated mortality, the Agency is far less certain about the public health implications of the changes in relatively low ambient O_3 concentrations (Proposed Rule, 79 FR at 75278/3, 75291/1, and 75308/2). Therefore, reducing precursor emissions to meet a lower O_3 standard is expected to result in important reductions in O_3 concentrations from the part of the air quality distribution where the evidence provides the strongest support for adverse health effects.

In addition, it is important to reiterate that in considering epidemiology-based risk estimates presented in the HREA, CASAC stated that "[a]lthough these estimates for short-term exposure impacts are subject to uncertainty, the CASAC is confident that that the evidence of health effects of O_3 presented in the ISA and Second Draft HREA in its totality, indicates that there are meaningful reductions in mean, absolute, and relative premature mortality associated with short-term exposures to O_3 levels lower than the current standard" (Frey, 2014a, p. 3). Given these comments by CASAC and the additional information presented earlier in this response (addressing greater confidence in risk reductions at higher ozone levels), the EPA does not agree with commenters who cited increases in estimated risk in some locations as supporting a conclusion that the current standard should be retained (see also response above regarding comments on urban core versus outside urban core exposure estimates).

(23) *Comment:* Commenters highlight issues raised by CASAC in their review of the 2nd draft PA. In particular, they noted key uncertainties and areas of potential future research identified by the CASAC Chair including research to address the characterization of the exposure-response function; the identification of population thresholds; the role of co-pollutants and temperature in modifying or contributing to ozone effects; alternative modeling specifications; population-

based information on human exposure for at-risk populations; time-activity data to improve population-based exposure and risk assessment; and the characterization of background levels. Commenters then state that these areas of uncertainty, like the exposure-response function, are critical elements necessary to establish a scientifically defensible standard. They assert that there is greater confidence and less uncertainty in the upper end of the ranges that were relied upon in the proposed rule. They further assert that there is a lack of statistically significant data for establishing a NAAQS at concentrations less than 72 ppb. These comments were provided by the commenter to support the argument that, due to a lack of evidence of effects below 72 ppb and due to reduced confidence in predicting ozone effects at lower levels, the current standard should not be lowered.

Response: EPA disagrees with the commenter's assertion that uncertainty in the risk assessment (and a lack of support for modeling risk at lower ozone levels) compromises the utility of the HREA in supporting consideration of lower alternative standard levels for ozone. The HREA addresses many of the sources of uncertainty (and areas of future research) identified by the commenter through either quantitative sensitivity analyses, or (when data did not allow) rigorous qualitative analysis (see section 7.4). Regarding the assertion that the epidemiology risk assessment did not support establishing a NAAQS at concentrations less than 72 ppb, we disagree and would point to CASAC comments on the 2nd draft HREA where CASAC stated that "[a]lthough these estimates for short-term exposure impacts are subject to uncertainty, the CASAC is confident that that the evidence of health effects of O_3 presented in the ISA and Second Draft HREA in its totality, indicates that there are meaningful reductions in mean, absolute, and relative premature mortality associated with short-term exposures to O_3 levels lower than the current standard" (Frey, 2014a, p. 3). We would point out that this observation was based on their review of the 2nd draft HREA, which included risk estimates generated for both the current standard and risk reductions associated with 70, 65 and 60 ppb, although it is not clear that the CASAC comment applied to all three alternative standard levels under consideration.[68]

(24) *Comment:* Commenters assert that EPA has ignored a number of source of evidence supporting the potential for thresholds in modeling epidemiology-based risk. These include (a) the role of antioxidants in the epithelial lining of the lung in scavaging ozone and the fact that these antioxidants can be replenished resulting in a lower impact from ozone exposure at lower levels (commenter cites Schelegle, 2007), (b) potential for regional heterogeneity and exposure measurement error in multi-city studies to obstruct identification of a threshold

[68] See also Samet (2011), p. 10: "While epidemiological studies are inherently more uncertain as exposures and risk estimates decrease (due to the greater potential for biases to dominate small effect estimates), specific evidence in the literature does not suggest that our confidence on the specific attribution of the estimated effects of ozone on health outcomes differs over the proposed range of 60-70 ppb" and further describing specific epidemiological studies supporting this conclusion.

(commenter acknowledges that EPA has mentioned this, but still points to it as support for a threshold), and (c) evidence in a single city study of an effect threshold (Atkinson, 2012).

Response: EPA has not ignored available evidence related to the potential for a threshold in ozone-related effects. Regarding the Schelegle (2007) study, the ISA explicitly discusses this study in the context of antioxidants (of which ozone is an example) modulating the neuronal reflex resulting in delays in the onset of respiratory symptoms (ISA, p. 5-32). EPA has also acknowledged that exposure measurement error can interfere with efforts to identify potential thresholds, especially at lower ozone levels (ISA, p. 2-11). In addition the ISA points out that interindividual variability in factors related to ozone exposure can mean that thresholds (if present) would be distributed across the population, making it difficult to identify a single population-level threshold. Regarding the Atkinson 2012 study, the commenter did not provide the full citation and consequently we were not able to definitively identify and review. However, based on a comprehensive review of available evidence, EPA has concluded in the ISA (p. 2-32 and 2-33) that epidemiologic studies examining the shape of the C-R curve and the potential presence of a threshold have indicated a generally linear C-R function with no indication of a threshold in analyses that have examined 8-h max and 24-h avg O_3 concentrations. However, EPA acknowledges that there is less certainty in the shape of the C-R curve at the lower end of the distribution of O_3 concentrations (below background concentrations, 29-40 ppb) due to the low density of data in this range.

(25) *Comment:* Commenters point to a possible seasonal relationship between ozone exposure and health effects with studies suggesting a positive association existing in warm months and no association in cold months (Dales et al., 2006; Ito et al., 2007; Medina-Ramón et al., 2006; Stieb et al., 2009; Strickland et al., 2010; Villeneuve et al., 2007; Zanobetti and Schwartz, 2006). Commenters point to an ozone-health threshold as one possible explanation for this seasonal effect. An alternative explanation provided by commenters could be that ozone levels are higher in the summer months and people spend more time outdoors when it's warmer. Commenters describe potential research initiatives to utilize these seasonal patterns in ozone response to investigate further, the potential for a threshold in the ozone effect.

Response: EPA has noted seasonal differences in the magnitude of ozone-related health effects with larger effects often associated with the warmer season (see ISA, section 6.2.7.5). There are a number of factors which could be responsible for this seasonal variation (e.g., variations in the mix of ozone precursors linked to seasonal patterns of urban emissions, seasonal differences in activity outdoor activity levels, seasonal differences in baseline incidence rates). EPA agrees that research to further investigate seasonal variation in ozone-related health impacts could be useful in the future and could provide information relevant to future reviews of the ozone NAAQS.

(26) *Comment:* Commenters state that EPA has incorrectly calculated sensitivity analysis results focusing on the potential impact of thresholds related to short-term mortality. Specifically, they contend that threshold values should not only result in exclusion of mortality values for days below those threshold, but that also, in calculating absolute risk for a day above the threshold, the effect estimate should be applied only to the increment of that day above the threshold being considered and not all the way down to zero (i.e., not to the full ozone level for that day) as they assert was done by EPA in completing the referenced sensitivity analysis. When implemented using the approach described by the commenter, they point to the significantly larger impact on risk that the thresholds would have (relative to the values presented by EPA in their sensitivity analysis). Note, that other commenters have also identified the significant impact that potential threshold related to short-term mortality would have on the estimates or risk.

Response: EPA disagrees with the commenter that the data presented in Table 3 of the Proposed Rule (79 FR 75277) was incorrectly calculated. The risk estimates in Table 3 do not reflect formal application of thresholds (at 20, 40 and 60ppb) in modeling short-term ozone-related mortality, but rather a more generalized look at the fraction of total simulated risk associated with days falling above a particular ozone level. In this way, Table 3 addresses uncertainty in a more general sense asking the question, 'what fraction of total simulated risk is associated with higher-confidence upper-end ozone days - e.g., days with basecase levels of 60ppb or greater'. Had we implemented a formal simulation of potential thresholds for this endpoint, the appropriate way to calculate risk (given a particular threshold) would depend largely on the way that threshold had been characterized in the underlying epidemiology study.

(27) *Comment*: One commenter asserts that the EPA has significantly underestimated the health impacts associated with ozone by only evaluating some health effects (respiratory ER visits and respiratory symptoms) in a subset of urban study areas modeled in the analysis and excluding other ozone-related health endpoints form quantification (note, no specific endpoints identified in the comment).

Response: EPA disagrees with the commenter that we have significantly underestimated risk due to exclusion of key health effects in our analysis. The set of health effects endpoint that we modeled is comprehensive and reflects a balance between fully capturing risk and providing risk estimates that are scientifically defensible (additional endpoints could have been modeled, but in most cases, those did not have the degree of evidence support we had established for inclusion in the HREA). Regarding the specific set of urban study areas modeled in the HREA, here we utilized a rigorous framework for selecting study areas, which balanced a desire to capture a diverse array or urban areas (with a range of ozone risk-related attributes) with practical consideration of resource and time restraints. As part of the HREA, we completed a representativeness analysis intended to evaluate the degree to which the set of modeled urban study areas is representative of the broader set of urban areas across the United States (HREA, section 8.2). Based on that analysis, we concluded that, "These three factors

[evaluated as part of the representativeness analysis] suggest that the urban study areas capture overall risk for the nation well, with a potential for better characterization of the high end of the risk distribution" (HREA, section 8.2.4). However, we readily acknowledge that these urban study areas do not fully capture broader suburban areas beyond their CBSA delineations that are likely to experience substantial risk reductions under implementation of alternative standards (see also response below in Risk of O_3 Associated Mortality and Morbidity section, and 79 FR 75277-78).

(28) *Comment:* Some commenters asserted that there are important uncertainties in the epidemiologic evidence due to model selection. For example, one commenter stated that the selection of an appropriate statistical model for epidemiologic analysis of the air pollution data is an extremely important process that can significantly affect the outcome of the study and can make the difference between finding a positive association, a negative association, or no association. They note that EPA used a relative risk value from the Smith et al. (2009) study to develop the concentration-response for non-accidental mortality that was an increase of 0.32% ± 0.08 for a 10 ppb increase in maximum daily 8-hour average O_3 concentration. The commenter contended that, in selecting this value, EPA pulled just one of hundreds of risk estimates from the Smith et al. (2009) study "because it met their criteria and ignored many others" (e.g., AAM). The commenter also contended that EPA does not mention Smith et al. (2009)'s conclusions when all of their results are considered in context.

Response: EPA disagrees with the commenter that we ignored many of the effect estimates for short-term mortality presented in Smith et al. (2009). EPA identified a clear set of criteria for selecting the set of effect estimates used in the core analysis (as well as the additional set included as sensitivity analyses to explore overall confidence in modeling this endpoint). In selecting the set of Bayes-adjusted effect estimates (reflecting application of a national-prior) for the core analysis, EPA clearly discussed the tradeoff between (a) national and regional priors (noting the reduced power associated with regionally-adjusted effect estimates), (b) copollutants versus multi-pollutant models (discussing the reduced number of copollutants monitoring data associated with the later models) and (c) full ozone-year versus summer-only models (noting the potential for the latter models to exclude ozone effect during cooler months) (see section 7.3.2 of the HREA, for a discussion of criteria considered and rationale used in selecting effect estimates for the core and sensitivity analysis; see section 7.4.1 and 7.4.2 of the HREA for discussion of the degree to which the effect estimates selected provided coverage for key sources of variability and are subject to uncertainty, respectively). In completing the sensitivity analyses and qualitative discussions of uncertainty/variability referenced here, we paid careful attention to critical issues raised in the Smith et al. (2009) study. Furthermore, we would point out that, in commenting on the short-term risk modeling presented in the HREA, CASAC was supportive of our approach, stating that, "Taking into account the body of scientific information and the scientifically based approach to inference of exposure and risk employed in the HREA, CASAC finds that the exposure and

risk estimates based on short-term exposure are of sufficient scientific quality to serve as a basis for decision making regarding the adequacy of the current primary standard and possible levels of a revised primary standard (Frey, 2014a, p. 10-11).

(29) *Comment:* Some commenters claimed that the EPA has not considered the effects of higher elevation, and the consequent reduction in exposure mass, on health risks.

Response: The EPA disagrees with this comment. Neglecting the changes in ventilation that occur at high altitude, the assertion that the number of ozone molecules per volume of air decreases with increasing elevation is correct. It is incorrect, however, to conclude that minute ventilation in humans/animals is unaffected by changes in air density. In acclimatized individuals, for a given metabolic rate, minute ventilation increases to compensate for less oxygen per unit volume with increasing altitude. This increase in minute ventilation results in similar ozone "exposures" at sea level vs. at higher altitudes. For someone not acclimatized to high altitude, the acute increase in ventilation would be much greater during the first days/week after ascending to altitude so that the ozone exposure for this person would be much higher during that time than someone at sea level.

In addition, modern ozone monitors based on UV absorption compute ozone concentration based on Beer's Law expressed as: $T = I / I_o = e^{-\alpha C l}$ where T is the transmission, I and I_o are the intensity of the UV light passing through the cell with and without ozone respectively, α is the absorption coefficient of ozone at 254 nm, C is the concentration of ozone in the absorption cell and l is the optical path length. The absorption coefficient is slightly pressure and temperature dependent so an internal pressure and temperature sensor measures the cell pressure and is used to correct the concentration. The monitors report out the concentration in mixing ratio (v/v) as specified by the EPA requirements. The net result is that the computed mixing ratio is independent of local atmospheric pressure.

iv. Air Quality Characterization

(1) *Comment:* Commenters (e.g., Clean Air Task Force) assert that the EPA Voronoi Neighbor Averaging (VNA) interpolation technique results in underestimates of ozone exposure in Boston. Specifically, they note that "(i)n the Boston area, the nine monitors outside of the design value monitor had three-year average fourth high values ranging from 0.058 to 0.069 ppm, with an overall average of 0.067 ppm. Seven of the nine were within 0.003 ppm of the regional DV. In other words, the ozone gradient across the metro-Boston area is minimal. This implies the potential exposures across the metropolitan area are not substantially lower than the levels determined from the maximum monitor in the region. The relatively uniform concentrations are important and may not be capture [sic] by

the technique relied upon by EPA in its Risk Assessment. . . An obvious drawback of the VNA approach is that it ignores transport in its calculation. CATF reviewed the monitors included in the VNA for the Boston area. One monitor – located in Dudley Square, near one of the busiest bus depots of the MBTA – was an outlier in behavior as compared to the rest of the region's monitors, presumably due to scavenging. The ozone levels measured at Dudley represent concentrations very near the traffic center, a large NO_x source. A nearby monitor on Long Island, a few miles downwind of the Dudley monitor, has ozone levels that track with the rest of the regional monitors, showing that ozone levels recover very quickly to the regional values just a few miles downwind. Due to its central location, this Dudley monitor was likely chosen by VNA as a major contributor to the determined spatial gradient across the region, effectively blocking contribution from the Long Island monitor. The Long Island monitor shows that the Dudley results do no [sic] accurately represent ambient concentrations throughout Boston. In this instance EPA's application of VNA clearly mischaracterizes the ozone levels. As a result, the estimate ozone levels for the Boston Risk Assessment would be lower than their true value. Equally important, the greatest influence of the Dudley monitor may also overlap with the most densely populated communities, further exacerbating the exposure underestimation."

Response: EPA disagrees that applying the VNA technique results in a mischaracterization of O_3 levels in Boston. The spatial pattern seen in the VNA results for Boston were similar to that of several other urban areas analyzed in the HREA, where O_3 concentrations observed by monitors near the urban center were lower than those measured in surrounding suburban areas. In these cities, application of the VNA technique resulted in a smooth surface transitioning from a local minimum near the urban center to higher concentrations in surrounding areas, especially downwind from the urban center. In the specific case of Boston, the Dudley Square monitor is located in a heavily urbanized area near the city center, while the Long Island monitor is located on a small island approximately 4 miles from the coast. These monitors reflect the typical pattern seen in the REA analyses, where the Dudley Square monitor experiences lower O_3 concentrations due to titration from local precursor emissions, while the Long Island monitor experiences higher concentrations due to its position directly downwind from the urban center and the lack of nearby precursor emissions sources. Additionally, the EPA provided a cross-validation evaluation of the VNA methodology in three urban areas in the HREA (Appendix 4-A), including Philadelphia, which had an urban gradient similar to that of Boston. The evaluation in Philadelphia showed that the VNA technique performed well with a mean bias of -0.01 ppb and an R^2 value of 0.868. Therefore, EPA believes that application of the VNA technique provides ozone values that are appropriate for estimating O_3 exposures for the Boston area as well as other urban areas evaluated in the HREA.

(2) *Comment:* Commenters (e.g., Clean Air Task Force) also assert that the model-based adjustment methodology "significantly overstated" the amount of NO_x reductions necessary to achieve the 70 ppb standard level. They base this

assertion on the fact that the model-based technique used 49% and 40% NO_x reductions from 2007 levels to achieve 70 ppb in 2006-2008 and 2008-2010 time period respectively, yet 2012-2014 ambient data already showed Boston in compliance with the 70 ppb standard with less than 28% reductions of national NO_x compared to earlier time periods. They further state that "By modeling NO_x reduction nearly twice the level observed to achieve the ambient DV of 0.070 ppm, EPA estimated future ozone levels to be much lower than what have actually occurred in Boston. As a result, there would have been many fewer days at the considered benchmark levels, leading to lower estimated modeled exposures relative to what is likely occurring."

Response: EPA disagrees with the assertions of the commenters and believes that their argument stems from a misunderstanding of how the modeling was applied in the REA and the relationship between model predictions and ambient concentrations in subsequent years. The air quality modeling started with ambient ozone concentrations from 2006-2010 and estimated how they would change as a result of emissions reductions given that all other factors remained constant. This is standard procedure for modeling-based projections. The measured 2012-2014 ozone values are not directly comparable to an analysis of what 2006-2010 ozone would look like if ONLY emissions were adjusted, as other factors such as changes in meteorological conditions will significantly affect the observations. In addition, the modeling methodology used for the REA is not intended to be a precise prediction of the future. Rather, the REA states that the technique allowed the EPA "to estimate how O_3 would respond to changes in ambient NO_x and/or VOC concentrations without simulating a specific control strategy…The purpose of these reductions scenarios is…to develop internally consistent estimates of spatial and temporal variability in O_3 associated with specified levels of possible standards". Extensive evaluations of the methodology provided in the REA and associated appendices support the credibility of this methodology. In addition, the model adjustments have undergone peer review both in the scientific literature and by CASAC.

(3) *Comment:* Commenters (e.g., ALA et al.) argue that "NO_x scavenging does not counsel a higher ozone standard" and provide several specific air quality and health related arguments. The air quality related comment are summarized here, while the health implications are dealt with separately in this document. Comments about air quality aspects include: 1) "ozone scavenging is not a pollution control mechanism – it is a pollution moving mechanism…In other words, NO_x scavenging does not actually destroy ozone, it merely delays its formation, and shifts it downwind," 2) "NO_x scavenging…results in the creation of NO_2, which is another harmful criteria pollutant [and] NO_x itself…is a key precursor not only of ozone, but also of particulate matter and other compounds with health and environmental concerns," 3) "The ozone scavenging implications of NO_x emissions reductions accompanying lower standards is extremely limited, both temporally and geographically…Further, the ozone scavenging implications of NO_x emissions reductions in urban cores was extremely restricted: NO_x reductions in urban cores generally decreased peak concentrations and increased

lower concentrations (as they did in the larger study areas) but they only increased concentrations near the center of the ozone distribution in a small subset of cities…while reductions in NO_x precursors led to increases in ozone in certain locations, those increases were restricted to the middle and lower ends of the ozone distribution and a small number of urban cores, and they did not get progressively larger as alternative standards were lowered,." and 4) "The effects of reduced ozone scavenging can be mitigated with a combined VOCs/ NO_x reduction strategy."

Response: We agree with several but not all of the commenters' points about ozone scavenging and atmospheric chemistry. First, we agree with the general point that, in most cases, NO_x emissions which lead to ozone scavenging (resulting in increases of ozone concentrations) near emission sources will eventually lead to ozone formation downwind as it mixes with VOC and sunlight. In addition, we agree that NO_x scavenging (i.e. the reaction of NO + O_3) results in the formation of NO2 which is both a criteria pollutant and can be a precursor to PM. On the commenters' third point, we disagree with the characterization of the ozone scavenging being "extremely" limited both temporally and geographically. The extent of ozone scavenging varied by time of year and location. In winter months the scavenging was more widespread. Also, in certain urban areas the scavenging was more widespread. A full characterization of this phenomenon was described and explored in the HREA and associated appendices. We do agree that modeled NO_x reductions generally reduced ozone on high ozone days even in locations that were most prone to scavenging and that mid-range ozone decreased in most cities as a result of NO_x reductions. We also agree that the ozone increases generally occurred at times and locations where ozone concentrations were low to begin with. Finally, as the commenters point out, results from sensitivity analysis in the HREA showed that in some cases combining VOC and NO_x reductions could mitigate ozone increases that occurred as the result of NO_x emissions reductions alone. The sensitivity analysis predicted that the mitigating effect on ozone was more pronounced in some urban areas and less pronounced in others. In some cases the VOC reductions reversed the ozone increase (i.e. resulted in net ozone decreases) but in other cases the VOC reductions did not entirely eliminate the ozone increases. However, the sensitivity analysis did not explore all possible emissions control strategies and did not attempt to optimize reductions for greatest ozone or health benefits so it is not possible to derive definitive conclusions from that analysis on whether more targeted emissions controls might result in a larger mitigating impact on the ozone disbenefits. Health implications of the mitigated ozone disbenefits in the NO_x/VOC sensitivity analyses are discussed separately in the response to comments in section I.A.1.c.iii above.

v. Other Risk and Exposure Assessment Comments

(1) *Comment:* Some commenters (e.g., AAM) asserted that the EPA had not appropriately considered the beneficial effects of O_3, including the beneficial effects of shielding from ultraviolet radiation. For example, one commenter noted

that in 2003 the EPA responded to the court's remand to consider the potential beneficial shielding effects of ozone by deciding that any plausible changes in ultraviolet (UV–B) radiation exposures from changes in patterns of ground-level O_3 were too uncertain to quantify and would likely be very small from a public health perspective.

The commenter noted that in the current review, the EPA again reaches the same conclusion, stating in the ISA (Chapter 10.5.2) that:

> EPA has found no published studies that adequately examine the incremental health or welfare effects (adverse or beneficial) attributable specifically to changes in UV-B exposure resulting from perturbations in tropospheric O_3 concentrations. While the effects are expected to be small, they cannot yet be critically assessed within reasonable uncertainty. Overall, the evidence is inadequate to determine if a causal relationship exists between changes in tropospheric O_3 concentrations and effects on health and welfare related to UV-B shielding.

As discussed below, the commenter makes specific points to back up its assertion that EPA can, in fact, estimate probable increases in skin cancer mortality and incidences due to the proposed changes to the O_3 standard. In support of this claim the commenter focuses on several points.

> 1. The main concern is for increased skin cancer incidences and deaths. The commenter notes that the ISA states that exposure to UV radiation is considered to be a major risk factor for all forms of skin cancer.
>
> 2. The ISA discusses a study by Madronich et al. (2011) that used the CMAQ model to estimate the UV radiation response to changes in tropospheric ozone concentrations in the southeastern U.S. under different control scenarios. Madronich et al. (2011) did not attempt to link their predicted increase in UV radiation to a predicted increase in skin cancer incidence, however, due to several remaining and substantial uncertainties.
>
> 3. The EPA has developed and applied models to link increases in UV radiation due to stratospheric ozone depletion to skin cancer incidences and mortality. A 2006 EPA report indicates that the Agency uses its Atmospheric and Health Effects Framework (AHEF) to evaluate certain human health impacts associated with reduced emissions of ozone-depleting substances (ODS) under the Montreal Protocol and associated amendments (U.S. EPA, 2006b). The AHEF model is easily adapted to predict changes in skin cancer incidence and mortality resulting from almost any scenario involving a change in ozone concentrations. The results for various scenarios involving the Montreal Protocol and its amendments estimate incidences of melanoma, basal cell, and squamous cell carcinoma, and deaths from melanoma. Such calculations have been

used by the Agency in rulemakings for ozone-depleting substances and for a proposed fleet of supersonic aircraft.

The commenter goes on to express the view that a comparison of the substantial effort to estimate premature mortality from ozone in the HREA with the lack of even passing reference to potential increases in skin cancer morbidity and mortality from a revised standard demonstrates, again, a double standard. In both cases, the effects are small and uncertain. However, the UV-related skin cancer effects at current ozone column levels are acknowledged by the scientific community and EPA as real, while the assumption of ambient ozone causing mortality with no threshold is not biologically plausible. The uncertainty over whether a revised standard will have a net benefit or dis-benefit for morbidity and mortality is, therefore, an additional reason to heavily discount the observational studies in the final decision.

Response: EPA asserts that it has appropriately assessed the UV-B shielding effects of tropospheric O_3. The ISA 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 UV-B radiation. The ISA (Chapter 10.4) focused this assessment on three key factors, including those factors that govern (1) human exposure and susceptibility to UV-B radiation, (2) human health effects due to UV-B radiation, and (3) UV-B shielding effects associated with changes in tropospheric O_3 concentrations. In doing so, the ISA 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.

With respect to human exposure and susceptibility, the factors that potentially influence UV radiation exposure were discussed in detail in Chapter 10 of the O_3 AQCD (U.S. EPA, 2006a) and section 10.4.2 of the ISA. These factors included outdoor activity, occupation, age, sex, geography, and protective behavior. Outdoor activity and occupation both influenced the amount of time people spend outdoors during daylight hours, the predominant factor for exposure to solar UV radiation. Age and sex were found to be factors that influence human exposure to UV radiation, particularly by influencing other factors of exposure such as outdoor activity and risk behavior. Studies indicated that females generally spent less time outdoors and, consequently, had lower UV radiation exposure on average compared to males. Geography influences the degree of solar UV flux to the surface, with higher solar flux at lower latitudes increasing the annual UV radiation dose for people living in southern states relative to northern states. Altitude was also found to influence personal exposure to UV radiation. Protective behaviors such as using sunscreen, wearing protective clothing, and spending time in shaded areas were shown to reduce exposure to UV radiation. Unlike the well-characterized factors affecting exposure and risk associated with damaging effects of inhaling tropospheric O_3, these factors are still not well-characterized or captured in models.

With respect to UV-B shielding effects associated with changes in tropospheric O_3 concentrations, there are multiple complexities in attempting to quantify the relationship between changes in tropospheric O_3 concentrations and UV-B exposure. The 2006 O_3 AQCD (U.S. EPA, 2006a) described a handful of studies addressing this relationship, but none reported quantifiable effects of tropospheric O_3 concentration fluctuations on UV-B exposure at the surface. Further quantifying the relationship between UV-B exposure and health effects is complicated by the uncertainties involved in the selection of an action spectrum and appropriate characterization of dose (e.g., peak or cumulative levels of exposure, timing of exposures, etc.). The lack of published studies that critically examined these issues together--that is the incremental health effects attributable specifically to UV-B changes resulting from changes in tropospheric O_3 concentrations--lead to the prior conclusion that the effect of changes in surface-level O_3 concentrations on UV-induced health outcomes could not be critically assessed within reasonable uncertainty (U.S. EPA, 2006a).

More specifically, as noted in the ISA and by the commenter, a recent study by Madronich et al. (2011) used CMAQ to estimate UV radiation response to changes in tropospheric O_3 concentrations under different control scenarios projected out to 2020. This study focused on southeastern U.S. and accounted for spatial and temporal variation in tropospheric O_3 concentration reductions. The average relative change in skin cancer-weighted surface UV radiation between the two scenarios was $0.11 \pm 0.03\%$ over June, July and August. Weighting by population, this estimate increased to $0.19 \pm 0.06\%$. Madronich et al. (2011) report that their estimated UV radiation increment is an *order of magnitude less* than that reported in an earlier study by Lutter and Wolz (1997), a key study cited in the court remand of the 1997 O_3 standards. Madronich et al. (2011) did not attempt to link their predicted increase in UV radiation to a predicted increase in skin cancer incidence, however, due to several remaining and substantial uncertainties.

The commenter also mentions the AHEF model, which is not discussed in the ISA, but is used by the EPA to predict the changes in skin cancer incidence and mortality and cataract incidence for different ozone-depleting substance emissions scenarios. The commenter suggests that it is a tool that the EPA could use in estimating the potential increases in skin cancer morbidity and mortality from a revised O_3 standard. However, this model does not address the multiple complexities associated with attempting to quantify the relationship between changes in tropospheric O_3 concentrations and UV-B exposure. The AHEF model's focus on well-mixed stratospheric gases does not allow for the estimation of local 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 the human exposure and susceptibility factors to adequately estimate UV-B exposure levels. However, little is known about the impact of these factors on individual exposure to UV-B.

With respect to the health effects of UV-B exposure, the ISA (section 10.4.3) notes that the most conspicuous and well-recognized acute response to UV radiation is erythema, or the reddening of the skin. Erythema is likely caused by direct damage to DNA by UV radiation. Many studies discussed in Chapter 10 of the 2006 O_3 AQCD (U.S. EPA, 2006a) found skin type to be a significant risk factor for erythema. Skin cancer is another prevalent health effect associated with UV radiation. Exposure to UV radiation is considered to be a major risk factor for all forms of skin cancer. Ocular damage from UV radiation exposure includes effects on the cornea, lens, iris, and associated epithelial and conjunctival tissues. The region of the eye affected by exposure to UV radiation depends on the wavelength of the incident UV radiation. Depending on wavelength, common health effects associated with UV radiation include photokeratitis (snow blindness; short wavelengths) and cataracts (opacity of the lens; long wavelengths).

Experimental studies reviewed in Chapter 10 of the 2006 O_3 AQCD (U.S. EPA, 2006a) have shown that exposure to UV radiation may suppress local and systemic immune responses to a variety of antigens. Results from controlled human exposure studies suggest that immune suppression induced by UV radiation may be a risk factor contributing to skin cancer induction. There is also evidence that UV radiation has indirect involvement in viral oncogenesis through the human papillomavirus, dermatomyositis, human immunodeficiency virus, and other forms of immunosuppression.

Beyond these well recognized adverse health effects associated with various wavelengths of UV radiation, the ISA (section 10.4.3) also discusses the potential health benefits of increased UV-B exposure related to the production of vitamin D in humans. The health benefits of UV-B exposure are an important consideration that are completely ignored by the commenter. Most humans depend on sun exposure to satisfy their requirements for vitamin D. Vitamin D deficiency can cause metabolic bone disease among children and adults, and also may increase the risk of many common chronic diseases, including type I diabetes mellitus and rheumatoid arthritis. Substantial in vitro and toxicological evidence also support a role for vitamin D activity against the incidence or progression of various forms of cancer. In some studies, UV-B related production of vitamin D had potential beneficial immunomodulatory effects on multiple sclerosis, insulin-dependent diabetes mellitus, and rheumatoid arthritis.

Therefore, in establishing guidelines on limits of exposure to UV radiation, the International Commission on Non-Ionizing Radiation Protection (ICNIRP) agreed that some low-level exposure to UV radiation has health benefits (ICNIRP, 2004). However, the adverse health effects of higher UV exposures necessitated the

development of exposure limits for UV radiation. The ICNIRP recognized the challenge in establishing exposure limits that would achieve a realistic balance between beneficial and adverse health effects. As concluded by ICNIRP (2004), "[t]he present understanding of injury mechanisms and long-term effects of exposure to [UV radiation] is incomplete, and awaits further research." This conclusion is entirely consistent with the conclusion of the ISA, as cited by the commenter.

Based on its review in the ISA of potential beneficial and non-beneficial shielding effects of tropospheric O_3, and consideration of public comments received on the change in shielding effects of tropospheric O_3 related to proposed changes in the O_3 standard, the EPA again concludes that information linking (a) changes in patterns of ground-level O_3 concentrations likely to occur as a result of revising the O_3 NAAQS to (b) changes in relevant patterns of exposures to UV-B radiation that may be of concern to public health is too uncertain at this time to warrant any relaxation in the level of public health protection determined by the Administrator to be requisite to protect against demonstrated direct adverse respiratory effects of exposure to O_3 in the ambient air. Further as noted in the ISA, it is the Agency's view that associated changes in UV-B radiation exposures, using plausible but highly uncertain assumptions about likely changes in patterns of ground-level ozone concentrations, would likely be very small from a public health perspective.

2. Comments on the Elements of a Revised Primary Standard

a. Indicator

The EPA received very few comments on the indicator of the primary standard. Those who did comment supported the proposed decision to retain O_3 as the indicator, noting the rationale put forward in the preamble to the proposed rule. These commenters generally expressed support for retaining the current indicator in conjunction with retaining other elements of the current standard, such as the averaging time and form. After considering the available evidence, CASAC advice, and public comments, the Administrator concludes that O_3 remains the most appropriate indicator for a standard meant to provide protection against photochemical oxidants. Therefore, she is retaining O_3 as the indicator for the primary standard in this final rule.

b. Averaging Time

The EPA established the current 8-hour averaging time[69] for the primary O_3 NAAQS in 1997 (62 FR 38856). The decision on averaging time in that review was based on numerous controlled human exposure and epidemiologic studies reporting associations between adverse respiratory effects and 6- to 8-hour O_3 concentrations (62 FR 38861). It was also noted that a standard with a maximum 8-hour averaging time is likely to provide substantial protection against respiratory effects associated with 1-hour peak O_3 concentrations. Similar conclusions

[69] This 8-hour averaging time reflects daily maximum 8-hour average O_3 concentrations.

were reached in the last O_3 NAAQS review and thus, the 8-hour averaging time was retained in 2008.

The EPA received limited public comments on the issue of averaging time for the O_3 primary standard. These comments are discussed in section II.C.2 of the preamble to the final rule. The EPA provides the following responses to specific comments on the averaging time for the primary O_3 standard.

(1) *Comment:* Most public commenters did not address the issue of whether the EPA should consider additional or alternative averaging times. Of those who did address this issue, some commenters representing state agencies or industry groups agreed with the proposed decision to retain the current 8-hour averaging time, generally noting the supportive evidence discussed in the preamble to the proposed rule. In contrast, several medical organizations and environmental groups questioned the degree of health protection provided by a standard based on an 8-hour averaging time. For example, one commenter asserted that "[a]veraging over any time period, such as 8 hours, is capable of hiding peaks that may be very substantial if they are brief enough" (e.g., Physicians for Social Responsibility). Another similar comment suggested that having an alternative averaging time may be beneficial under certain conditions, stating that "a four-hour standard (or a standard for some time period shorter than eight hours) would allow for better protection of human health" (e.g., Kentuckiana Planning and Development Agency).

Response: The EPA agrees with these commenters that an important issue in the current review is the appropriateness of using a standard with an 8-hour averaging time to protect against adverse health effects that are attributable to a wide range of O_3 exposure durations, including those shorter and longer than 8 hours. This is an issue that has been thoroughly evaluated by the EPA in past reviews, as well as in the current review.

The 8-hour O_3 NAAQS was originally set in 1997, as part of revising the then-existing standard with its 1-hour averaging time, and was retained in the review completed in 2008 (73 FR 16472). In both of these reviews, several lines of evidence and information provided support for an 8-hour averaging time rather than a shorter averaging time. For example, substantial health evidence demonstrated associations between a wide range of respiratory effects and 6- to 8-hour exposures to relatively low O_3 concentrations (i.e., below the level of the 1-hour O_3 NAAQS in place prior to the review completed in 1997). A standard with an 8-hour averaging time was determined to be more directly associated with health effects of concern at lower O_3 concentrations than a standard with a 1-hour averaging time. In addition, results of quantitative analyses showed that a standard with an 8-hour averaging time can effectively limit both 1- and 8-hour exposures of concern, and that an 8-hour averaging time results in a more uniformly protective national standard than a 1-hour averaging time. In past reviews, CASAC has agreed that an 8-hour averaging time is appropriate.

In reaching her proposed decision to retain the 8-hour averaging time in the current review, the Administrator again considered the body of evidence for adverse effects attributable to a wide range of O_3 exposure durations, including studies specifically referenced by public commenters who questioned the protectiveness of a standard with an 8-hour averaging time. For example, as noted above substantial health effects evidence from controlled human exposure studies demonstrates that a wide range of respiratory effects occur in healthy adults following 6.6-hour exposures to O_3 (ISA, section 6.2.1.1). Compared to studies evaluating shorter exposure durations (e.g., 1-hour), studies evaluating 6.6-hour exposures in healthy adults have reported respiratory effects at lower O_3 exposure concentrations and at more moderate levels of exertion. The Administrator also noted the strength of evidence from epidemiologic studies that evaluated a number of different averaging times, with the most common being the maximum 1-hour concentration within a 24-hour period (1-hour max), the maximum 8-hour average concentration within a 24-hour period (8-hour max), and the 24-hour average. Evidence from time-series and panel epidemiologic studies comparing risk estimates across averaging times does not indicate that one exposure metric is more consistently or strongly associated with respiratory health effects or mortality (ISA, section 2.5.4.2; p. 2-31). For single- and multi-day average O_3 concentrations, lung function decrements were associated with 1-hour max, 8-hour max, and 24-hour average ambient O_3 concentrations, with no strong difference in the consistency or magnitude of association among the averaging times (ISA, p. 6-71). Similarly, in studies of short-term exposure to O_3 and mortality, Smith et al. (2009) and Darrow et al. (2011) have reported high correlations between risk estimates calculated using 24-hour average, 8-hour max, and 1-hour max averaging times (ISA, p. 6-253). Thus, the epidemiologic evidence does not provide a strong basis for distinguishing between the appropriateness of 1-hour, 8-hour, and 24-hour averaging times.

In addition, quantitative exposure and risk analyses in the HREA are based on an air quality adjustment approach that estimates hourly O_3 concentrations, and on scientific studies that evaluated health effects attributable to a wide range of O_3 exposure durations. For example, the risk of lung function decrements is estimated using a model based on controlled human exposure studies with exposure durations ranging from 2 to 7.6 hours (ISA, section 6.2.1.1). Epidemiology-based risk estimates are based on studies that reported health effect associations with short-term ambient O_3 concentrations ranging from 1-hour to 24-hours and with long-term seasonal average concentrations (HREA, Table 7-2). Thus, the HREA estimated health risks associated with a wide range of O_3 exposure durations and the Administrator's conclusions on averaging time in the current review are based, in part, on consideration of these estimates.

When taken together, the evidence and analyses indicate that a standard with an 8-hour averaging time, coupled with the current 4th-high form and an appropriate level, would be expected to provide appropriate protection against the short- and long-term O_3 concentrations that have been reported to be associated with

respiratory morbidity and mortality. The CASAC agreed with this, stating the following (Frey, 2014b, p. 6):

> The current 8-hour averaging time is justified by the combined evidence from epidemiologic and clinical studies referenced in Chapter 4. Results from clinical studies, for example, show a wide range of respiratory effects in healthy adults following 6.6 hours of exposure to ozone, including pulmonary function decrements, increases in respiratory symptoms, lung inflammation, lung permeability, decreased lung host defense, and airway hyperresponsiveness. These findings are supported by evidence from epidemiologic studies that show causal associations between short-term exposures of 1, 8 and 24-hours and respiratory effects and "likely to be causal" associations for cardiovascular effects and premature mortality. The 8-hour averaging window also provides protection against the adverse impacts of long-term ozone exposures, which were found to be "likely causal" for respiratory effects and premature mortality.

Given all of the above, the EPA disagrees with commenters who question the protectiveness of an O_3 standard with an 8-hour averaging time, particularly for an 8-hour standard with the revised level of 70 ppb that is being established in this review, as discussed in section II.C.4 of the preamble to the final rule.

c. Form of the Primary Standard

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

(1) *Comment:* Several commenters focused on the stability of the standard to support their positions regarding form. Some industry associations and state agencies support changing to a form that would allow a larger number of exceedances of the standard level than are allowed by the current 4th-high form. In some cases, these commenters argued that a standard allowing a greater number of exceedances would provide the same degree of public health protection as the current standard. Some commenters advocated a percentile-based form, such as the 98th percentile. These commenters cited a desire for consistency with short-term standards for other criteria pollutants (e.g., $PM_{2.5}$, NO_2), as well as a desire to allow a greater number of exceedances of the standard level, thus making the standard less sensitive to fluctuations in background O_3 concentrations and to extreme meteorological events.

Other commenters submitted analyses purporting to indicate that a 4th-high form provides only a small increase in stability, relative to forms that allow fewer exceedances of the standard level (i.e., 1st-high, 2nd-high). These commenters also called into question the degree of health protection achieved by a standard with a

4th-high form and a level in the proposed range (i.e., 65 to 70 ppb). They pointed out that a 4th-high form will, by definition, allow 3 days per year, on average, with 8-hour O_3 concentrations above the level of the standard. Commenters further stated that "[i]f ozone levels on these peak days are appreciably higher than on the 4th-highest day, given EPA's acknowledged concerns regarding single or multiple (defined by EPA as 2 or more) exposures to elevated ozone concentrations, EPA must account for the degree of under-protection in setting the level of the NAAQS;" that is, by lowering the level of the standard (e.g., ALA et al.).

Response: For the reasons discussed in the proposal, and summarized in sections II.C.3.a through II.C.3.b of the preamble to the final rule, the EPA disagrees with commenters who supported a percentile-based form, such as the 98th percentile, for the O_3 NAAQS. As noted above, a percentile-based statistic would not be effective in ensuring the same degree of public health protection across the country. Rather, a percentile-based form would allow more days with higher air quality values in locations with longer O_3 seasons relative to locations with shorter O_3 seasons. Thus, as in the 2008 review, in the current review the EPA concludes that a form based on the nth-highest maximum O_3 concentration would more effectively ensure that people who live in areas with different length O_3 seasons receive the same degree of public health protection.

In considering various nth-high values, as in past reviews (e.g., 73 FR 16475, March 27, 2008), the EPA recognizes that there is not a clear health-based threshold for selecting a particular nth-highest daily maximum form. Rather, the primary consideration is the adequacy of the public health protection provided by the combination of all of the elements of the standard, including the form. Environmental and public health commenters are correct that a standard with the current 4th-high form will allow 3 days per year, on average, with 8-hour O_3 concentrations higher than the standard level. However, the EPA disagrees with these commenters' assertion that using a 4th-high form results in a standard that is under-protective. The O_3 exposure and risk estimates that informed the Administrator's consideration of the degree of public health protection provided by various standard levels were based on air quality that "just meets" various standards with the current 8-hour averaging time and 4th-high, 3-year average form (HREA, section 4.3.3). Therefore, air quality adjusted to meet various levels of the standard with the current form and averaging time will include days with concentrations above the level of the standard, and these days contribute to exposure and risk estimates. In this way, the Administrator has considered the public health protection provided by the combination of all of the elements of the standard, including the 4th-high form.

In past reviews, EPA selected the 4th-highest daily maximum form in recognition of the public health protection provided by this form, when coupled with an appropriate averaging time and level, and recognizing that such a form can provide stability for ongoing implementation programs. As noted above, some commenters submitted analyses suggesting that a 4th high form provides only a small increase in stability, relative to a 1st- or 2nd-high form. The EPA has

conducted analyses of ambient O_3 monitoring data to further consider these commenters' assertions regarding stability. The EPA's analyses of n^{th}-high concentrations ranging from 1^{st}-high to 5^{th}-high have been summarized in a memo to the docket (Wells, 2015a). Consistent with commenters' analyses, Wells (2015a) indicates a progressive decrease in the variability of O_3 concentrations, and an increase in the stability of those concentrations, as "n" increases. Based on these analyses, there is no clear threshold for selecting a particular n^{th}-high form based on stability alone. Rather, as in past reviews, the decision on form in this review focuses first and foremost on the Administrator's judgments on public health protection, with judgments regarding stability of the standard being a legitimate, but secondary consideration.

In reaching a final decision on the form of the primary O_3 standard, as described in the proposal and in the preamble to the final rule, the Administrator recognizes that there is not a clear health-based rationale for selecting a particular n^{th}-highest daily maximum form. Her foremost consideration is the adequacy of the public health protection provided by the combination of all of the elements of the standard, including the form. In this regard, the Administrator recognizes the support from analyses in previous reviews, and from the CASAC in the current review, for the conclusion that the current 4^{th}-high form of the standard, when combined with a revised level as discussed below, provides an appropriate balance between public health protection and a stable target for implementing programs to improve air quality. In particular, she notes that the CASAC concurred that the O_3 standard should be based on the 4^{th}-highest, daily maximum 8-hour average value (averaged over 3 years), stating that this form "provides health protection while allowing for atypical meteorological conditions that can lead to abnormally high ambient ozone concentrations which, in turn, provides programmatic stability" (Frey, 2014b, p. 6). Based on these considerations, and on consideration of public comments on form as discussed in the preamble to the final rule and above, the Administrator judges it appropriate to retain the current 4^{th}-high form (4^{th}-highest daily maximum 8-hour O_3 concentration, averaged over 3 years) in this final rule.

(2) *Comment:* Some commenters (e.g., ALA et al.) maintain that the effective level of a standard with an nth-percentile form are quantifiable, and allow multiple days of air quality shown from the controlled human exposure studies to be injurious even at exposure times of 6.6 hours, and specifically, would allow air quality levels producing the combination of lung function decrements and symptoms which the EPA identifies as clearly adverse. Specifically, the commenter indicates that a standard set at 70 ppb with the current form is the equivalent of establishing a 1^{st} max standard of between 77.2 and 78.1 ppb; the current form even with a standard set at a level of 65 ppb would allow concentrations of 72.2 to 73.1 ppb. The form also allows numerous exposures identified by both the EPA and CASAC as levels of concern.

Response: As discussed in section II.A.2.d below, while the EPA does not dispute the results of air quality analyses submitted by these commenters, and agrees that

4th-high form allows multiple days per year with ambient O_3 concentrations above the level of the standard (3 days per year, on average over a 3-year period), the Agency disagrees with commenters' assertion that, because of this, the level of the primary O_3 standard should be set below 70 ppb. As also discussed in section II.A.2.d below, the number of days above the level of the standard is not the same as estimates of exposure of concern and health risk, which are indicators of potential public health impacts. As discussed in the preamble to the final rule (section II.A.2), the quantitative assessments that informed the Administrator's proposed decision, presented in the HREA and considered in the PA and by CASAC, estimated O_3 exposures of concern and health risks associated with air quality that "just meets" various standards with the current 8-hour averaging time and 4th-high, 3-year average form. Thus, in considering the degree of public health protection appropriate for the primary O_3 standard, the Administrator has considered quantitative exposure and risk estimates that are based a 4th-high form, and therefore on a standard that, as these commenters point out, allows multiple days per year with ambient O_3 concentrations above the level of the standard. The EPA judges that this approach is reasonable when considering the public health protection provided by a standard with a 4^{th} high form.

The Administrator's consideration of exposure and risk estimates within the context of her decision on the level of the primary standard is discussed in detail in sections II.C.4.b and II.C.4.c of the preamble to the final rule. As discussed in those sections, contrary to the conclusions of commenters who advocated for a level below 70 ppb, the Administrator judges that a revised standard with a level of 70 ppb, when combined with the current 8-hour averaging time and 3-year average 4^{th} high form, will effectively limit the occurrence of the O_3 exposures for which she is most confident in the adversity of the resulting effects (i.e., based on estimates for the 70 and 80 ppb benchmarks). She further concludes that such a standard will provide substantial protection against the occurrence of O_3 exposures for which there is greater uncertainty in the adversity of effects (i.e., based on estimates for the 60 ppb benchmark). The Administrator also notes the important public health improvements estimated for a revised standard with a level of 70 ppb, based on the lung function and epidemiology-based risk assessments included in the HREA (sections II.C.4.b and II.C.4.c of the preamble to the final rule).

(3) *Comment:* Commenters (e.g., ALA et al.) argue that because of the truncating convention in the form of the standard in Appendix P to Part 50, and O_3 concentration is, as a practical matter, essentially 1 ppb higher. The EPA has failed to account for (or acknowledge) this practical effect, which results in the top end of the range being even less protective (for example, the average concentration in Schelegle et al., 2009, was 72 ppb, and if a standard of 70 ppb is practically 71 ppb, there is even less of a margin between that standard and this key study).

Response: The analyses of ambient O_3 concentration data provided in the HREA and PA documents, and the risk and exposure estimates provided in the HREA

document, were based on adjusting O_3 air quality concentrations to just meet the current and alternative standards using the same truncation provisions that are being retained in this review.[70] That is, the existing truncation procedure is already implicitly accounted for in the Administrator's decision regarding the level of the revised O_3 standards. Thus, the Administrator took into consideration estimates that incorporated the precision level of the standard (using truncation) in evaluating the margin of safety of the revised primary standard. As above, the EPA judges that this approach is reasonable when considering the public health protection provided by the standard that the Agency is adopting in this review.

d. Level

A number of groups representing medical, public health, or environmental organizations; some state agencies; and many individuals submitted comments on the appropriate level of a revised primary O_3 standard.[71]

Virtually all of these commenters supported setting the standard level within the range recommended by CASAC (i.e., 60 to 70). Some expressed support for the overall CASAC range, without specifying a particular level within that range, while others expressed a preference for the lower part of the CASAC range, often emphasizing support for a level of 60 ppb. Some of these commenters stated that if the EPA does not set the level at 60 ppb, then the level should be set no higher than 65 ppb (i.e., the lower bound of the proposed range of standard levels).

To support their views on the level of a revised standard, some commenters focused on overarching issues related to the statutory requirements for the NAAQS.

(1) *Comment:* Some commenters maintained that the primary NAAQS must be set at a level at which there is an absence of adverse effects in sensitive individuals.

Response: While this argument has some support in the case law and in the legislative history to the 1970 CAA (see *Lead Industries Ass'n v. EPA*, 647 F. 2d 1147, 1153 (D.C. Cir. 1980)), it is well established that the NAAQS are not meant to be zero risk standards. See *Lead Industries v. EPA*, 647 F.2d at 1156 n.51; *Mississippi v. EPA*, 744 F. 3d at 1351. From the inception of the NAAQS standard-setting process, the EPA and the courts have acknowledged that scientific uncertainties in general, and the lack of clear thresholds in pollutant effects in particular, preclude any such definitive determinations. *Lead Industries*, 647 F. 2d at 1156 (setting standard at a level which would remove most but not all sub-clinical effects). Likewise, the House report to the 1977 amendments

[70] Truncation was applied for purposes of adjusting air quality. The resulting ambient O_3 concentrations that served as inputs to exposure and risk analyses were not truncated.

[71] In general, commenters who expressed the view that the EPA should retain the current O_3 NAAQS (i.e., commenters representing industry and business groups, and some states) did not provide comments on alternative standard levels. As a result, this section focuses primarily on comments from commenters who expressed support for the proposed decision to revise the current primary O_3 standard.

addresses this question, indicating that NAAQS are not intended to be zero-risk (H. Rep. 95-294, 95th Cong. 1st sess. 127):[72]

> Some have suggested that since the standards are to protect against all known or anticipated effects and since no safe threshold can be established, the ambient standards should be set at zero or background levels. Obviously, this no-risk philosophy ignores all economic and social consequences and is impractical. This is particularly true in light of the legal requirement for mandatory attainment of the national primary standards within 3 years.

Thus, post-1970 jurisprudence makes clear the impossibility, and lack of legal necessity, for NAAQS removing all health risk. See *ATA III*, 283 F. 3d at 360 ("[t]he lack of a threshold concentration below which these pollutants are known to be harmless makes the task of setting primary NAAQS difficult, as EPA must select standard levels that reduce risks sufficiently to protect public health even while recognizing that a zero-risk standard is not possible"); *Mississippi*, 744 F. 3d at 1351 (same); see also id. at 1343 ("[d]etermining what is 'requisite' to protect the 'public health' with an 'adequate' margin of safety may indeed require a contextual assessment of acceptable risk. See *Whitman*, 531 U.S. at 494-95 (Breyer J. concurring)").

In this review, EPA is setting a standard based on a careful weighing of available evidence, including a weighing of the strengths and limitations of the evidence and underlying scientific uncertainties therein. The Administrator's choice of standard level is rooted in her evaluation of the evidence, which reflects her legitimate uncertainty as to the O_3 concentrations at which the public would experience adverse health effects. This is a legitimate, and well recognized, exercise of "reasoned decision-making." *ATA III*. 283 F. 3d at 370; see also id. at 370 ("EPA's inability to guarantee the accuracy or increase the precision of the … NAAQS in no way undermines the standards' validity. Rather, these limitations indicate only that significant scientific uncertainty remains about the health effects of fine particulate matter at low atmospheric concentration…."); *Mississippi*, 744 F. 3d at 1352-53 (appropriate for EPA to balance scientific uncertainties in determining level of revised O_3 NAAQS).

(2) *Comment:* In an additional overarching comment, some commenters also fundamentally objected to the EPA's consideration of exposure estimates in reaching conclusions on the primary O_3 standard. These commenters' general assertion was that NAAQS must be established so as to be protective, with an adequate margin of safety, regardless of the activity patterns that feed into

[72] Similarly, Senator Muskie remarked during the floor debates on the 1977 Amendments that "there is no such thing as a threshold for health effects. Even at the national primary standard level, which is the health standard, there are health effects that are not protected against". 123 Cong. Rec. S9423 (daily ed. June 10, 1977).

exposure estimates. They contended that "[a]ir quality standards cannot rely on avoidance behavior in order to protect the public health and sensitive groups" and that "[i]t would be unlawful for EPA to set the standard at a level that is contingent upon people spending most of their time indoors" (e.g., ALA et al.). In support of these comments, ALA et al. analyzed ambient monitoring data from Core-Based Statistical Areas (CBSAs) with design values between 66-70 ppb and 62-65 ppb and pointed out that there are many more days with ambient concentrations above the benchmark levels than were estimated in the EPA's exposure analysis (i.e., at and above the benchmark level of 60, 70 and 80 ppb).

Response: The EPA disagrees with these commenters' conclusions regarding the appropriateness of considering exposure estimates, and notes that NAAQS must be "requisite" (i.e., "sufficient, but not more than necessary" (*Whitman*, 531 U.S. at 473)) to protect the "public health" ("the health of the public" (*Whitman*, 531 U.S. at 465)). Estimating exposure patterns based on available data[73] is a reasonable means of ascertaining that standards are neither under- nor over-protective, and that standards address issues of public health rather than health issues pertaining only to isolated individuals.[74] Behavior patterns are critical in assessing whether ambient concentrations of O_3 may pose a public health risk.[75] Exposures to ambient or near-ambient O_3 concentrations have only been shown to result in potentially adverse effects if the ventilation rates of people in the exposed populations are raised to a sufficient degree (e.g., through physical exertion) (ISA, section 6.2.1.1). Ignoring whether such elevated ventilation rates are actually occurring, as advocated by these commenters, would not provide an accurate assessment of whether the public health is at risk. Indeed, a standard established without regard to behavior of the public would likely lead to a standard which is more stringent than necessary to protect the public health.

While setting the primary O_3 standard based only on ambient concentrations, without consideration of activity patterns and ventilation rates, would likely result in a standard that is over-protective, the EPA also concludes that setting a standard based on the assumption that people will adjust their activities to avoid exposures on high-pollution days would likely result in a standard that is under-protective. The HREA's exposure assessment does not make this latter

[73] The CHAD database used in the HREA's exposure assessment contains over 53,000 individual daily diaries including time-location-activity patterns for individuals of both sexes across a wide range of ages (HREA, Chapter 5). See also various responses relating to the CHAD database in section Comments on Activity Pattern Data Used by APEX above.

[74] CASAC generally agreed with the EPA's methodology for characterizing exposures of concern (Frey, 2014a, pp. 5-6).

[75] See 79 FR 75269. "The activity pattern of individuals is an important determinant of their exposure. Variation in O_3 concentrations among various microenvironments means that the amount of time spent in each location, as well as the level of activity, will influence an individual's exposure to ambient O_3. Activity patterns vary both among and within individuals, resulting in corresponding variations in exposure across a population and over time" (internal citations omitted).

assumption.[76] The time-location-activity diaries that provided the basis for exposure estimates reflect actual variability in human activities. While some diary days may reflect individuals spending less time outdoors than would be typical for them, it is similarly likely that some days reflect individuals spending more time outdoors than would be typical. Considering the actual variability in time-location-activity patterns is at the least a permissible way of identifying standards that are neither over- nor under-protective.[77]

Further, the EPA sees nothing in the CAA that prohibits consideration of the O_3 exposures that could result in effects of public health concern. While a number of judicial opinions have upheld the EPA's decisions in other NAAQS reviews to place little weight on particular risk or exposure analyses (i.e., because of scientific uncertainties in those analyses), none of these opinions have suggested that such analyses are irrelevant because actual exposure patterns do not matter. See, e.g. *Mississippi*, 744 F. 3d at 1352-53; *ATA III*, 283 F. 3d at 373-74. Therefore, because behavior patterns are critical in assessing whether ambient concentrations of O_3 may pose a public health risk, the EPA disagrees with the views expressed by the commenters, objecting to the consideration of O_3 exposures in reaching decisions on the primary O_3 standard.

In addition to these overarching comments, a number of commenters supported their views on standard level by highlighting specific aspects of the scientific evidence, exposure/risk information, and/or CASAC advice. Key themes expressed by these commenters included the following: (1) controlled human exposure studies provide strong evidence of adverse lung function decrements and airway inflammation in healthy adults following exposures to O_3 concentrations as low as 60 ppb, and at-risk populations would be likely to experience more serious effects or effects at even lower concentrations; (2) epidemiologic studies provide strong evidence for associations with mortality and morbidity in locations with ambient O_3 concentrations below 70 ppb, and in many cases in locations with concentrations near and below 60 ppb; (3) quantitative analyses in the HREA are biased such that they understate O_3 exposures and risks, and the EPA's interpretation of lung function risk estimates is not appropriate and not consistent with other NAAQS; and (4) the EPA must give deference to CASAC advice, particularly CASAC's policy advice to set the standard level below 70 ppb. The next sections discuss comments related to each of these key themes, and provide the EPA's responses to those comments.

Effects in Controlled Human Exposure Studies

[76] The EPA was aware of the possibility of averting behavior during the development of the HREA, and that document includes sensitivity analyses to provide perspective on the potential role of averting behavior in modifying O_3 exposures. As discussed in section II.B.2.c in the preamble to the final rule, as well as in responses to comments in the O_3 Exposures of Concern section above, these sensitivity analyses were limited and the results were discussed in the proposal within the context of uncertainties in the HREA assessment of exposures of concern.

[77] See *Mississippi*, 744 F. 3d at 1343 ("[d]etermining what is 'requisite' to protect the 'public health' with an 'adequate' margin of safety may indeed require a contextual assessment of acceptable risk. See *Whitman*, 531 U.S. at 494-95 (Breyer, J. concurring…))"

(3) *Comment:* Some commenters who advocated for a level of 60 ppb (or absent that, for 65 ppb) asserted that controlled human exposure studies have reported adverse respiratory effects in healthy adults following exposures to O_3 concentrations as low as 60 ppb. These commenters generally based their conclusions on the demonstration of FEV_1 decrements $\geq$ 10% and increased airway inflammation following exposures of healthy adults to 60 ppb O_3. They concluded that even more serious effects would occur in at-risk populations exposed to 60 ppb O_3, and that such populations would experience adverse effects following exposures to O_3 concentrations below 60 ppb.

Response: While the EPA agrees that information from controlled human exposure studies conducted at 60 ppb can help to inform the Administrator's decision on the standard level, the Agency does not agree that this information necessitates a level below 70 ppb. In fact, as discussed both at proposal and in the preamble to the final rule, a revised O_3 standard with a level of 70 ppb can be expected to provide substantial protection against the effects shown to occur following various O_3 exposure concentrations, including those observed following exposures to 60 ppb.[78] This is because the degree of protection provided by any NAAQS is due to the combination of all of the elements of the standard (i.e., indicator, averaging time, form, level). In the case of the 4th-high form of the O_3 NAAQS, which the Administrator is retaining in the current review (section II.C.3 in the preamble to the final rule), the large majority of days in areas that meet the standard will have 8-hour O_3 concentrations below the level of the standard, with most days well-below the level. Therefore, in considering the degree of protection provided by an O_3 standard with a particular level, it is important to consider the extent to which that standard would be expected to limit population exposures of concern to the broader range of O_3 exposure concentrations shown in controlled human exposure studies to result in health effects. The Administrator's consideration of such exposures of concern is discussed in section II.C.4.c in the preamble to the final rule.

An important part of the Administrator's consideration of exposure estimates is the extent to which she judges that adverse effects could occur following specific O_3 exposures. While controlled human exposure studies provide a high degree of confidence regarding the extent to which specific health effects occur following exposures to O_3 concentrations from 60 to 80 ppb, the Administrator notes that there are no universally accepted criteria by which to judge the adversity of the observed effects. Therefore, in making judgments about the extent to which the effects observed in controlled human exposure studies have the potential to be adverse, the Administrator considers the recommendations of ATS and advice from CASAC (section II.A.1.c in the preamble to the final rule).

[78] See, e.g. Table 1 in the preamble to final rule (two or more exposures at 60 ppb benchmark reduced to 0.5-3.5% in urban study areas by a standard with a level of 70 ppb).

As an initial matter, with regard to the effects shown in controlled human exposure studies following O_3 exposures, the Administrator notes the following:

1. The largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures to 80 ppb O_3 or higher, with most exposure studies conducted at these higher concentrations. Specifically, 6.6-hour exposures of healthy young adults to 80 ppb O_3, while engaged in quasi-continuous, moderate exertion, can decrease lung function, increase airway inflammation, increase respiratory symptoms, result in airway hyperresponsiveness, and decrease lung host defenses.

2. Exposures of healthy young adults for 6.6 hours to O_3 concentrations as low as 72 ppb, while engaged in quasi-continuous, moderate exertion, have been shown to both decrease lung function and result in respiratory symptoms.

3. Exposures of healthy young adults for 6.6 hours to O_3 concentrations as low as 60 ppb, while engaged in quasi-continuous, moderate exertion, have been shown to decrease lung function and to increase airway inflammation.

To inform her judgments on the potential adversity to public health of these effects reported in controlled human exposure studies, as in the proposal, the Administrator considers the ATS recommendation that "reversible loss of lung function in combination with the presence of symptoms should be considered adverse" (ATS, 2000). She notes that this combination of effects has been shown to occur following 6.6-hour exposures to O_3 concentrations at or above 72 ppb. In considering these effects, CASAC observed that "the combination of decrements in FEV_1 together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014b, p. 5).

Regarding the potential for adverse effects following exposures to lower concentrations, the Administrator notes the CASAC judgment that the adverse combination of lung function decrements and respiratory symptoms "almost certainly occur in some people" following exposures to O_3 concentrations below 72 ppb (Frey, 2014b, p. 6). In particular, when commenting on the extent to which the study by Schelegle et al. (2009) suggests the potential for adverse effects following O_3 exposures below 72 ppb, CASAC judged that:

> [I]f subjects had been exposed to ozone using the 8-hour averaging period used in the standard [rather than the 6.6-hour exposures evaluated in the study], adverse effects could have occurred at lower concentration. Further, in our judgment, the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma (Frey, 2014b, p. 5).

Though CASAC did not provide advice as to how far below 72 ppb adverse effects would likely occur, the Administrator agrees that such effects could occur following exposures at least somewhat below 72 ppb.

The Administrator notes that while adverse effects could occur following exposures at least somewhat below 72 ppb, the combination of statistically significant increases in respiratory symptoms and decrements in lung function has not been reported following 6.6-hour exposures to average O_3 concentrations of 60 ppb or 63 ppb, though studies have evaluated the potential for such effects (Adams, 2006; Schelegle et al., 2009; Kim et al., 2011). In the absence of this combination, the Administrator looks to additional ATS recommendations and CASAC advice in order to inform her judgments regarding the potential adversity of the effects that have been observed following O_3 exposures as low as 60 ppb.

With regard to ATS, she first notes the recommendations that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" and that "[f]ew…biomarkers have been validated sufficiently that their responses can be used with confidence to define the point at which a response should be equated to an adverse effect warranting preventive measures" (ATS, 2000).[79] Based on these recommendations, compared to effects following exposures at or above 72 ppb, the Administrator has less confidence in the adversity of the respiratory effects that have been observed following exposures to 60 or 63 ppb.

(4) *Comment:* Some commenters who advocated for a level of 60 ppb also focused on ATS recommendations regarding population-level risks. These commenters specifically stated that lung function decrements "may be adverse in terms of 'population risk,' where exposure to air pollution increases the risk to the population even though it might not harm lung function to a degree that is, on its own, 'clinically important' to an individual" (e.g., ALA et al.). These commenters asserted that the EPA has not appropriately considered the potential for such population-level risk.

Response: Contrary to the views express by these commenters, the Administrator carefully considers the potential for population risk, particularly within the context of the ATS recommendation that "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). Given that exposures to 60 ppb O_3 have been shown in controlled human exposure studies to cause transient and reversible decreases in group mean lung function, the Administrator notes the potential for such exposures to result in similarly transient and reversible shifts in the risk profile of an exposed population. However, in contrast to commenters who advocated for a level of 60 ppb, the Administrator also notes that the available evidence does not provide information on the extent

[79] With regard to this latter recommendation, as discussed in section II.A.1.c of the preamble to the final rule, the ATS concluded that elevations of biomarkers such as cell numbers and types, cytokines, and reactive oxygen species may signal risk for ongoing injury and more serious effects or may simply represent transient responses, illustrating the lack of clear boundaries that separate adverse from nonadverse events.

to which a short-term, transient decrease in lung function in a population, as opposed to a longer-term or permanent decrease, could affect the risk of other, more serious respiratory effects (i.e., change the risk profile of the population). This uncertainty, together with the additional ATS recommendations noted above, indicates to the Administrator that her judgment that there is uncertainty in the adversity of the effects shown to occur at 60 ppb is consistent with ATS recommendations.[80]

With regard to CASAC advice, the Administrator notes that, while CASAC clearly advised the EPA to consider the health effects shown to occur following exposures to 60 ppb O_3, its advice regarding the adversity of those effects is less clear. In particular, she notes that CASAC was conditional about whether the lung function decrements observed in some people at 60 ppb (i.e., FEV_1 decrements $\geq$ 10%) are adverse. Specifically, CASAC stated that these decrements "*could* be adverse in individuals with lung disease" (Frey, 2014b, p. 7, emphasis added) and that they provide a "*surrogate* for adverse health outcomes for people with asthma and lung disease" (Frey, 2014b, p. 3, emphasis added). Further, CASAC did not recommend considering standard levels low enough to eliminate O_3-induced FEV_1 decrements $\geq$ 10% (Frey, 2014b). With regard to the full range of effects shown to occur at 60 ppb (i.e., FEV_1 decrements, airway inflammation), CASAC stated only that exposures of concern for the 60 ppb benchmark are "*relevant for consideration*" with respect to people with asthma (Frey, 2014b, p. 6, emphasis added). The Administrator contrasts these statements with CASAC's clear advice that "the combination of decrements in FEV_1 together with the statistically significant alterations in symptoms in human subjects exposed to 72 ppb ozone meets the American Thoracic Society's definition of an adverse health effect" (Frey, 2014b, p. 5).

Based on her consideration of all of the above recommendations and advice, the Administrator judges that, compared to exposure concentrations at and above 72 ppb, there is greater uncertainty with regard to the adversity of effects shown to occur following O_3 exposures as low as 60 ppb. However, based on the effects that have been shown to occur (i.e., lung function decrements, airway inflammation), and CASAC advice indicating the importance of considering these effects (though its advice regarding their adversity is less clear), she concludes that it is appropriate to give some consideration to the extent to which a revised standard could allow such effects, and has done so particularly in considering the substantial elimination of multiple exposures of concern at the 60 ppb benchmark resulting from a standard set at a level of 70 ppb. Moreover, the EPA expects that a revised standard with a level of 70 ppb will also reduce the occurrence of

[80] ATS provided additional recommendations to help inform judgments regarding the adversity of air pollution-related effects (e.g., related to "quality of life"), though it is not clear whether, or how, such recommendations should be applied to the respiratory effects observed in controlled human exposure studies following 6.6-hour O_3 exposures (ATS, 2000, p. 672).

exposures to O_3 concentrations at least somewhat *below* 60 ppb (HREA, Figures 4-9 and 4-10).[81]

(5) *Comment:* As discussed in section II.B.2.b of the preamble to the final rule, some commenters who opposed revision of the current standard based on their analysis of effects shown to occur following exposures to 72 ppb O_3, made the point that not every occurrence of an exposure of concern will result in an adverse effect. An aspect of this point was also highlighted by some commenters who advocated for a level of 60 ppb, based on the discussion of O_3-induced inflammation in the proposal. In particular, this latter group of commenters highlighted discussion from the proposal indicating that "[i]nflammation induced by a single O_3 exposure can resolve entirely but, as noted in the ISA (p. 6-76), 'continued acute inflammation can evolve into a chronic inflammatory state'" (e.g., ALA et al.).

Response: In considering estimates of exposures of concern for the 60, 70, and 80 ppb benchmarks within the context of her judgments on adversity, the Administrator notes that, as commenters stated (echoing repeated findings by the EPA at proposal, e.g. 79 FR 75246/1), due to interindividual variability in responsiveness, not every occurrence of an exposure of concern will result in an adverse effect. Consistent with these comments, and with her consideration of estimated exposures of concern in the proposal, the Administrator judges that the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O_3 may cause other more serious effects. Because of this, the Administrator is most concerned about protecting at-risk populations against repeated occurrences of exposures of concern.

The Administrator's consideration of estimated exposures of concern is discussed in more detail in the preamble to the final rule in sections II.C.4.b.iv and II.C.4.c. In summary, contrary to the conclusions of commenters who advocated for a level of 60 ppb, the Administrator judges that a revised standard with a level of 70 ppb will effectively limit the occurrence of the O_3 exposures for which she is most confident in the adversity of the resulting effects (i.e., based on estimates for the 70 and 80 ppb benchmarks). She further concludes that such a standard will provide substantial protection against the occurrence of O_3 exposures for which there is greater uncertainty in the adversity of effects (i.e., based on estimates for the 60 ppb benchmark). Not only will such occurrences be limited but, as just noted, due to inter-individual variability, exposure does not automatically mean that the individual exposed will experience an adverse effect.

(6) *Comment:* Some commenters also pointed out that benchmark concentrations are based on studies conducted in healthy adults, whereas at-risk populations are

[81] Air quality analyses in the HREA indicate that reducing the level of the primary standard from 75 ppb to 70 ppb will result in reductions in the O_3 concentrations in the upper portions of ambient distributions. This includes 8-hour ambient O_3 concentrations at, and somewhat below, 60 ppb (HREA, Figures 4-9 and 4-10).

likely to experience more serious effects and effects at lower O_3 exposure concentrations. In considering this issue, the EPA notes CASAC's endorsement of 60 ppb as the lower end of the range of benchmarks for evaluation, and its advice that "the 60 ppb-8hr exposure benchmark is relevant for consideration with respect to adverse effects on asthmatics" (Frey, 2014b, p. 6).

Response: As discussed in detail in section II.C.4.c in the preamble to the final rule and in other responses to comments in this document (including the responses immediately preceding), the Administrator has carefully considered estimated exposures of concern for the 60 ppb benchmark. In addition, though the available information does not support the identification of specific benchmarks below 60 ppb that could be appropriate for consideration for at-risk populations, and though CASAC did not recommend consideration of any such benchmarks, the EPA expects that a revised standard with a level of 70 ppb will also reduce the occurrence of exposures to O_3 concentrations at least somewhat below 60 ppb (HREA, Figures 4-9 and 4-10).[82]

Thus, even if some members of at-risk populations may experience effects following exposures to O_3 concentrations somewhat below 60 ppb, a revised level of 70 ppb would be expected to reduce the occurrence of such exposures.[83] Therefore, the EPA has considered O_3 exposures that could be relevant for at-risk populations such as children and people with asthma, and does not agree that controlled human exposure studies reporting respiratory effects in healthy adults following exposures to 60 ppb O_3 necessitate a standard level below 70 ppb.

(7) *Comment:* One commenter noted that the averaging period for the controlled human exposure studies was 6.6 hours, whereas the averaging period for the NAAQS is 8 hours. The commenter explains that because O_3 harm increases with cumulative dose, the level at which O_3 would cause adverse effects would be lower than seen in the controlled human exposure studies. The commenter concludes that the level of the 8-hour standard must accordingly be adjusted lower.

Response: As this commenter points out, the revised primary O_3 standard includes an 8-hour averaging time. In reaching her decision to retain the 8-hour averaging time in the current review, the Administrator considered the body of evidence for adverse effects attributable to a wide range of O_3 exposure durations, including studies specifically referenced by public commenters who questioned the protectiveness of a standard with an 8-hour averaging time. For example, as noted by commenters, a substantial body of health effects evidence from controlled human exposure studies demonstrates that a wide range of respiratory effects

[82] Air quality analyses in the HREA indicate that reducing the level of the primary standard from 75 ppb to 70 ppb will result in reductions in the O_3 concentrations in the upper portions of ambient distributions. This includes 8-hour ambient O_3 concentrations at, and somewhat below, 60 ppb (HREA, Figures 4-9 and 4-10).

[83] The uncertainty associated with the potential adversity of any such effects would be even greater than that discussed above for the 60 ppb benchmark.

occur in healthy adults following 6.6-hour exposures to O_3 (ISA, section 6.2.1.1). Compared to studies evaluating shorter exposure durations (e.g., 1-hour), studies evaluating 6.6-hour exposures in healthy adults have reported respiratory effects at lower O_3 exposure concentrations and at more moderate levels of exertion. The Administrator also notes the evidence from epidemiologic studies that evaluated a number of different averaging times, with the most common being the maximum 1-hour concentration within a 24-hour period (1-hour max), the maximum 8-hour average concentration within a 24-hour period (8-hour max), and the 24-hour average. Evidence from time-series and panel epidemiologic studies comparing risk estimates across averaging times does not indicate that one exposure metric is more consistently or strongly associated with respiratory health effects or mortality (ISA, section 2.5.4.2; p. 2-31). For single- and multi-day average O_3 concentrations, lung function decrements were associated with 1-hour max, 8-hour max, and 24-hour average ambient O_3 concentrations, with no strong difference in the consistency or magnitude of association among the averaging times (ISA, p. 6-71). Similarly, in studies of short-term exposure to O_3 and mortality, Smith et al. (2009) and Darrow et al. (2011) have reported high correlations between risk estimates calculated using 24-hour average, 8-hour max, and 1-hour max averaging times (ISA, p. 6-253). Thus, the epidemiologic evidence does not provide a strong basis for distinguishing between the appropriateness of 1-hour, 8-hour, and 24-hour averaging times**.**

In addition, quantitative exposure and risk analyses in the HREA are based on an air quality adjustment approach that estimates hourly O_3 concentrations, and on scientific studies that evaluated health effects attributable to a wide range of O_3 exposure durations. For example, the risk of lung function decrements is estimated using a model based on controlled human exposure studies with exposure durations ranging from 2 to 7.6 hours (ISA, section 6.2.1.1). Epidemiology-based risk estimates are based on studies that reported health effect associations with short-term ambient O_3 concentrations ranging from 1-hour to 24-hours and with long-term seasonal average concentrations (HREA, Table 7-2). Thus, the HREA estimated health risks associated with a wide range of O_3 exposure durations and the Administrator's conclusions on averaging time in the current review are based, in part, on consideration of these estimates.

The comments noted above are an issue primarily within the context of interpreting quantitative estimates of O_3 exposures of concern, which were based on 8-hour exposure estimates. As discussed in the preamble to the final rule (see II.B.2.b and II.C.4.b of the final rule), there are aspects of the exposure assessment that, considered by themselves, can result in either overestimates or underestimates of the occurrence of O_3 exposures of concern. The EPA has carefully considered these various aspects of the assessment above (section II.A.1.c.i.) and in section II.B.2.c.i of the preamble to the final rule.

(8) *Comment:* Commenters maintained that finding of 10 percent FEV_1 decrements and pulmonary inflammation at 60 ppb in the controlled human exposure studies

necessitates a standard established at a level of 60 ppb. They based this on the following:

- The studies were conducted with healthy adults, not sensitive populations
- Even among healthy adults, some sizeable percentage of the population are more sensitive to O_3 effects ("responders")
- ATS views a 10% decrement in FEV_1 as an abnormal response and a reasonable criteria for assessing exercise-induced bronchoconstriction
- In previous NAAQS reviews, the EPA itself has judged that for people with lung disease, moderate decrement in FEV_1 greater than 10 percent but less than 20 percent lasting up to 24 hours would likely interfere with normal activity for many individuals, and would likely result in more frequent use of medication, and that ATS consider more frequent use of medication as a change in clinical status viewed by ATS as adverse
- In previous reviews, CASAC advised that FEV decrements of 10 percent should be considered adverse in people with lung disease, especially asthmatic children (Henderson, 2006).
- CASAC has also advised that a 10 percent decrement in FEV_1 can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease (Samet, 2011). "For example, people with chronic obstructive pulmonary disease have decreased ventilator reserve (i.e. decreased baseline FEV_1) such that a 10 percent decrement could lead to moderate to severe respiratory symptoms." (Henderson, 2006).

Response: The EPA has addressed much of the substance of these comments in other responses. Discussion of the use of a 10% reduction in lung function as an abnormal response by ATS and the adversity of a 10% reduction in lung function is discussed in responses to comments in section II.A.1.b.i above and in this section below. Discussion of the consistency of adversity judgments by the EPA between this review and the 2008 review and 2010 reconsideration, and with past CASAC advice, can also be found in comments and responses in sections II.A.1.b.i above and in this section below.

As an initial matter, we note that in this review the Agency's consideration of the potential adversity of various O_3-related responses is based on an updated body of scientific evidence, updated exposure and risk estimates, and updated CASAC advice. The Administrator fully considered all of this updated information and all of the staff conclusions, as well as ATS recommendations and CASAC advice, in making judgments about the potential adversity of O_3-related effects.

As in past reviews, a consideration of FEV_1 decrements $\geq$ 10% in the current review is based in part on ATS criteria, as well as on CASAC advice. Based on

ATS guidelines for assessing bronchoconstriction, the ISA states that "[a] 10% FEV_1 decrement is…generally accepted as an abnormal response" (ISA, p. 6-19). In this context, "abnormal" indicates that the decrement is outside the normal range of day-to-day variability, and is not meant to indicate that such a response is invariably "adverse" or that it would necessarily be accompanied by symptoms or lifestyle changes such as alterations in medication use. In fact, in its recommendations on adversity, the ATS did not speak specifically to FEV_1 decrements of any particular magnitude, and stated that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" (ATS, 2000, p. 670).

With regard to advice from CASAC in previous reviews cited by the commenter, we note that, as in this review, CASAC spoke conditionally when discussing adversity of 10 percent lung function decrements, indicating the same uncertainty the Administrator sees in CASAC's advice on the same issue in this review: "a 10 percent decrement *could* lead to moderate to severe respiratory symptoms" (Samet, 2011, emphasis added). That panel also advised that "'[c]linically relevant' effects are decrements greater than 10%, a decrease in lung function considered clinically relevant by the American Thoracic Society" (Samet, 2011, p. 2), but as noted above, the ATS itself did not determine that this decrement was an adverse health effect. Consistent with these characterizations, in the current review CASAC advised that "an FEV_1 decrement of $\geq$ 10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease" (e.g., see sections II.B.2.b.i, II.B.3, II.C.4.b, and II.C.4.c of the preamble to the final rule). In the current review, the Administrator further agrees with the judgment made in past reviews (e.g., see 75 FR 2973, January 19, 2010) that a more general consensus view of the potential adversity of such decrements emerges as the frequency of occurrences increases (sections II.B.3 and II.C.4.c of the preamble to the final rule). In addition, consistent with past and current CASAC recommendations, in both the 2008 final decision and in the Administrator's final decision in the current review, the level of the primary O_3 standard was set to reduce, but not eliminate, the estimated occurrence of O_3-induced FEV_1 decrements $\geq$ 10%, reflecting some uncertainty as to the adversity of this effect.

As discussed in section II.C.4.c in the preamble to the final rule, in reaching her decision to set the primary standard at a level of 70 ppb, the Administrator fully considered the potential adversity of10% decrements in FEV_1, and estimates of the occurrence of such decrements in the population. However, as discussed in section II.C.4.b in the preamble to the final rule and in responses in this section above, it is well established that the NAAQS are not meant to be zero risk standards. The EPA agrees that an important consideration when reaching a decision on level is the extent to which a revised standard is estimated to allow the types of exposures shown in controlled human exposure studies to cause respiratory effects discussed by these commenters. In reaching her final decision that a level of 70 ppb is requisite to protect public health with an adequate margin of safety (II.C.4.c, preamble to the final rule), the Administrator carefully

considers the potential for such exposures and effects. In doing so, she emphasizes the importance of setting a standard that limits the occurrence of the exposures about which she is most concerned (i.e., those for which she has the most confidence in the adversity of the resulting effects, which are repeated exposures of concern at or above 70 or 80 ppb, as discussed in section II.C.4.b.i of the preamble to the final rule). Based on her consideration of information from controlled human exposure studies in light of CASAC advice and ATS recommendations, the Administrator additionally judges that there is important uncertainty in the extent to which the effects shown to occur following exposures to 60 ppb O_3 are adverse to public health (discussed in sections II.C.4.b.i and II.C.4.b.iii of the preamble to the final rule). However, based on the effects that have been shown to occur, CASAC advice indicating the importance of considering these effects, and ATS recommendations indicating the potential for adverse population-level effects (II.C.4.b.i, II.C.4.b.iii), she concludes that it is appropriate to give some consideration to the extent to which a revised standard could allow the respiratory effects that have been observed following exposures to 60 ppb O_3.

When considering the extent to which a revised standard could allow O_3 exposures that have been shown in controlled human exposures studies to result in respiratory effects such as the ones that commenters discuss, the Administrator is most concerned about protecting the public, including at-risk populations, against repeated occurrences of such exposures of concern (section II.C.4.b.i in the preamble to the final rule). In considering the appropriate metric for evaluating repeated occurrences of exposures of concern, the Administrator acknowledges that it is not clear from the evidence, or from the ATS recommendations, CASAC advice, or public comments, how the number of exposures of concern could impact the seriousness of the resulting effects, especially at lower exposure concentrations. Therefore, the Administrator judges that focusing on HREA estimates of two or more exposures of concern provides a health-protective approach to considering the potential for repeated occurrences of exposures of concern that could result in adverse effects. She notes that other possible metrics for considering repeated occurrences of exposures of concern (e.g., 3 or more, 4 or more, etc.) would result in smaller exposure estimates.

As discussed further (in section II.C.4.c of the preamble to the final rule), the Administrator notes that a revised standard with a level of 70 ppb is estimated to eliminate the occurrence of two or more exposures of concern to O_3 concentrations at or above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O_3 concentrations at or above 70 ppb (Table 1, preamble to the final rule). For the 70 ppb benchmark, this reflects about a 90% reduction in the number of children estimated to experience two or more exposures of concern, compared to the current standard. Even considering the worst-case urban study area and worst-case year evaluated in the HREA, a standard with a level of 70 ppb is estimated to protect more than 99% of children from experiencing two or more exposures of concern to O_3 concentrations at or above 70 ppb (Table 1, preamble to the final rule).

Though the Administrator judges that there is greater uncertainty with regard to the occurrence of adverse effects following exposures as low as 60 ppb, she notes that a revised standard with a level of 70 ppb is estimated to protect the vast majority of children in the urban study areas (i.e., about 96% to more than 99% in individual areas) from experiencing two or more exposures of concern at or above 60 ppb. Compared to the current standard, this represents a reduction of more than 60% in exposures of concern for the 60 ppb benchmark (Table 1, preamble to the final rule). Given the Administrator's uncertainty regarding the adversity of the effects following exposures to 60 ppb O_3, and her health-protective approach to considering repeated occurrences of exposures of concern as of special import, the Administrator judges that this degree of protection is appropriate and that it reflects substantial protection against the occurrence of O_3-induced effects, including effects for which she judges the adversity to public health is uncertain.

Contrary to the conclusions of commenters who advocated for a level below 70 ppb, the Administrator notes that her final decision is consistent with CASAC's advice in this review and in prior reviews (see II.A.1.b.i. above), based on the scientific evidence, and with CASAC's focus on setting a revised standard to further limit the occurrence of the respiratory effects observed in controlled human exposure studies, including effects observed following exposures to 60 ppb O_3. Given her judgments and conclusions discussed above, and given that the CAA reserves the choice of the standard that is requisite to protect public health with an adequate margin of safety for the judgment of the EPA Administrator (while based on the air quality criteria), she disagrees with commenters who asserted that CASAC advice necessitates a level below 70 ppb, and as low as 60 ppb.

Epidemiologic Studies

(9) *Comment:* Commenters representing environmental and public health organizations also highlighted epidemiologic studies that, in their view, provide strong evidence for associations with mortality and morbidity in locations with ambient O_3 concentrations near and below 60 ppb. These commenters focused both on the epidemiologic studies evaluated in the PA's analyses of study location air quality (PA, Chapter 4) and on studies that were not explicitly analyzed in the PA, and in some cases on studies that were not included in the ISA.

Response: The EPA agrees that epidemiologic studies can provide perspective on the degree to which O_3-associated health effects have been identified in areas with air quality likely to have met various standards. However, as discussed in the preamble to the final rule, we do not agree with the specific conclusions drawn by these commenters regarding the implications of epidemiologic studies for the standard level. As an initial matter in considering epidemiologic studies, the EPA notes its decision, consistent with CASAC advice, to place the most emphasis on information from controlled human exposure studies (sections II.B.2 and II.B.3 in the preamble to the final rule). This decision reflects the greater certainty in using information from controlled human exposure studies to link specific O_3 exposures

with health effects, compared to using air quality information from epidemiologic studies of O_3 for this purpose.

While being aware of the uncertainties discussed in the preamble to the final rule (section II.B.2.b.ii), in considering what epidemiologic studies can tell us, the EPA notes analyses in the PA (section 4.4.1) indicating that a revised standard with a level at or below 70 ppb would be expected to maintain distributions of short-term ambient O_3 concentrations below those present in the locations of all of the single-city epidemiologic studies analyzed. As discussed in the PA (section 4.4.1), this includes several single-city studies conducted in locations that would have violated the current standard, and the study by Mar and Koenig (2009) that reported positive and statistically significant associations with respiratory emergency department visits with children and adults in a location that would have met the current standard over the entire study period.[84] While these analyses provide support for a level at least as low as 70 ppb, the Administrator judges that they do not provide a compelling basis for distinguishing between the appropriateness of 70 ppb and lower standard levels.

As in the proposal, the EPA acknowledges additional uncertainty in interpreting air quality in locations of multicity epidemiologic studies of short-term O_3 for the purpose of evaluating alternative standard levels (PA, sections 3.1.4.2 and 4.4.1). In particular, the PA concludes that interpretation of such air quality information is complicated by uncertainties in the extent to which multicity effect estimates (i.e., which are based on combining estimates from multiple study locations) can be attributed to ambient O_3 in the subset of study locations that would have met a particular standard, versus O_3 in the study locations that would have violated the standard.[85] While giving only limited weight to air quality analyses in these study areas because of this uncertainty, the EPA also notes PA analyses indicating that a standard level at or below 70 ppb would require additional reductions, beyond those required by the current standard, in the ambient O_3 concentrations that provided the basis for statistically significant O_3 health effect associations in multicity epidemiologic studies. As was the case for the single-city studies, and contrary to the views expressed by the commenters noted above, the Administrator judges that these studies do not provide a compelling basis for

[84] As noted in the preamble to the final rule (sections II.B.2 and II.B.3), the studies by Silverman and Ito (2010) and Strickland et al. (2010) provided support for the Administrator's decision to revise the current primary O_3 standard, but do not provide insight into the appropriateness of specific standard levels below 75 ppb.

[85] As noted in the proposal (section II.E.4.d), this uncertainty applies specifically to interpreting air quality analyses within the context of multicity effect estimates for short-term O_3 concentrations, where effect estimates for individual study cities are not presented (as is the case for the key O_3 studies analyzed in the PA, with the exception of the study by Stieb et al. (2009) where none of the city-specific effect estimates for asthma emergency department visits were statistically significant). This specific uncertainty does not apply to multicity epidemiologic studies of long-term O_3 concentrations, where multicity effect estimates are based on comparisons across cities. For example, see discussion of study by Jerrett et al. (2009) in the PA (section 3.1.4.3).

distinguishing between the appropriateness of alternative standard levels at or below 70 ppb.

(10) *Comment:* In some cases, commenters highlighted studies that were assessed in the 2008 review of the O_3 NAAQS, but were not included in the ISA in the current review. These commenters asserted that such studies support the occurrence of O_3 health effect associations in locations with air quality near or, in some cases, below 60 ppb. Specifically, they highlighted a number of studies included in the 2007 Staff Paper that were not included in the ISA, claiming that these studies support a standard level below 70 ppb, and as low as 60 ppb.

Response: As an initial matter with regard to these studies, the EPA notes that the focus of the ISA is on assessing the most policy-relevant scientific evidence. In the current review, the ISA considered over 1,000 new studies that have been published since the last review. Thus, it is not surprising that, as the body of evidence has been strengthened since the last review, some of the studies considered in the last review are no longer among the most policy relevant. However, based on the information included in the 2007 Staff Paper, the EPA does not agree that the studies highlighted by commenters provide compelling support for a level below 70 ppb. In fact, as discussed in the Staff Paper in the last review (U.S. EPA, 2007, p. 6-9; Appendix 3B), the O_3 concentrations reported for these studies, and the concentrations highlighted by commenters, were based on averaging across multiple monitors in study areas. Given that the highest monitor in an area is used to determine whether that area meets or violates the NAAQS, the averaged concentrations reported in the Staff Paper are not appropriate for direct comparison to the level of the O_3 standard. When the Staff Paper considered the O_3 concentrations measured at individual monitors for the subset of these study areas with particularly low concentrations, they were almost universally found to be above, and in many cases well above, even the current standard level of 75 ppb.[86] Based on the above considerations, and consistent with the Administrator's overall decision to place less emphasis on air quality in locations of epidemiologic studies to select a standard level, the EPA disagrees with commenters who asserted that epidemiologic studies included in the last review, but not cited in the ISA or PA in this review, necessitate a level below 70 ppb. In fact, the EPA notes that these studies are consistent with the majority of the U.S. studies evaluated in the PA in the current review, in that most were conducted in locations that would have violated the current O_3 NAAQS over at least part of the study periods.

[86] For one study conducted in Vancouver, where data from individual monitors did indicate ambient concentrations below the level of the current standard (Vedal et al., 2003), the Staff Paper noted that the study authors questioned whether O3, other gaseous pollutants, and PM in this study may be acting as surrogate markers of pollutant mixes that contain more toxic compounds, "since the low measured concentrations were unlikely, in their opinion, to cause the observed effects" (U.S. EPA, 2007, p. 6-16). See also PA p. 3-62 n. 47 referring to Vedal et al., 2003, but not providing the interpretive context given here. The 2007 Staff Paper further noted that another study conducted in Vancouver failed to find statistically significant associations with O3 (Villeneuve et al., 2003).

(11) *Comment:* Other commenters highlighted studies that were not included in the ISA, and that did not report associations with O_3. These commenters asserted that the EPA should consider these and other studies where no associations were found. Some commenters noted that they submitted to CASAC a study that showed no association with O_3. These commenters encouraged the EPA to consider this study before making a decision in the current review.

Response: As noted above, the focus of the ISA is on assessing the most policy-relevant scientific evidence and, in the current review, the ISA considered thousands of studies. Not all of the studies considered were judged to be among the most policy relevant. The process for identifying specific studies to include in the ISA was transparent, as discussed in the preamble to the ISA, and was subject to CASAC review. In addition, CASAC and members of the public had ample opportunity for identifying additional studies that should be included in the ISA. Therefore, we do not agree with commenters who asserted that studies not included in the ISA (particularly studies submitted by commenters to CASAC for consideration, but not recommended for inclusion by CASAC), provide a basis for reaching a different decision on the primary O_3 standard than that reached by the Administrator in this review.

(12) *Comment:* With respect to studies included in the ISA, one commenter noted that multicity studies provide strong evidence for the need for a revised standard at a level of 60 ppb. In particular, the commenter pointed to Katsouyanni (2009) where 9 of 12 cities would have met a standard of 65 ppb, and 5 of 12 cities would have met a standard of 60 ppb where the study showed positive and statistically significant associations with respiratory hospital admissions. Stieb et al. (2009) likewise showed nearly half of the study cities meeting a standard of 60 ppb where the study showed positive and statistically significant association with respiratory emergency department visits. The commenter further states more generally that the multi-city studies are highly probative here, given that "effect estimates are largely influenced by locations meeting the current standard", suggesting "a relatively high degree of confidence in the presence of associations with mortality and morbidity for ambient O_3 concentrations meeting the current standard" (quoting PA, p. 3-64).

One commenter indicated that even accepting EPA's metric, numerous studies have 98th percentile 8-hour daily maximum concentrations below 70, 65, and 60 ppb, citing studies from the last review and the current review, including Mortimer (2002), Delfino et al. (1997), Koken et al. (2003), Delfino et al. (1994) and Burnett et al. (1997) and (1999).

Some commenters also noted CASAC advice from the last review, where the panel stated that it had equal confidence in epidemiologic studies at the lower levels of the potential range (60 ppb-70 ppb) (citing to Samet, 2011, pp. 10-11). The basis for the panel's statement (as quoted by the commenter) was:

- Adverse effects in studies observed below the level of the current standard, citing Vedal et al., (2003) (mortality); Korrick et al. (1998) (FEV_1 decreases); Spektor et al. (1988) (panel study; lung function decrements), and; Thurston et al. (1997) (various responses).
- Endpoints of concern do not change at the lower level of the range, showing consistency with the results of the controlled human exposure studies

These commenters concluded from this advice that EPA's statement of increased uncertainty of effects at lower levels such as 60 ppb to be unfounded.

Response: Studies from the last review that were cited by commenters to support standard levels below 70 ppb are addressed in responses just above (see also responses to comments in Evidence from Epidemiologic Studies section above discussing the Administrator's consideration of panel studies in this review. In considering information from epidemiologic studies within the context of her conclusions on the adequacy of the current standard in the current review, the Administrator specifically considers analyses in the PA that evaluate the extent to which O_3 health effect associations have been reported for air quality concentrations likely to be allowed by the current standard. She notes that such analyses can provide insight into the extent to which the current standard would allow the distributions of ambient O_3 concentrations that provided the basis for these health effect associations. While the majority of O_3 epidemiologic studies evaluated in the PA were conducted in areas that would have violated the current standard during study periods, as discussed in section II.B.2.b.ii of the preamble to the final rule, the Administrator observes that the study by Mar and Koenig (2009) reported associations between short-term O_3 concentrations and asthma emergency department visits in children and adults in a U.S. location that would have met the current O_3 standard over the entire study period.[87] Based on this, she notes the conclusion from the PA that the current primary O_3 standard would have allowed the distribution of ambient O_3 concentrations that provided the basis for the associations with asthma emergency department visits reported by Mar and Koenig (2009) (PA, section 3.1.4.2).

In addition, even in some single-city study locations where the current standard was violated (i.e., those evaluated in Silverman and Ito, 2010; Strickland et al., 2010), the Administrator notes that PA analyses of reported concentration-response functions and available air quality data support the occurrence of O_3-attributable hospital admissions and emergency department visits on subsets of days with virtually all ambient O_3 concentrations below the level of the current standard. PA analyses of study area air quality further support the conclusion that exposures to the ambient O_3 concentrations present in the locations evaluated by Strickland et al. (2010) and Silverman and Ito (2010) could have plausibly

[87] The large majority of locations evaluated in U.S. epidemiologic studies of long-term O3 would have violated the current standard during study periods. Although these studies support the ISA's causality determinations, they provide limited insight into the adequacy of the current standard (PA, section 3.1.4.3).

resulted in the respiratory-related emergency department visits and hospital admissions reported in these studies (PA, section 3.1.4.2). The Administrator agrees with the PA conclusion that these analyses indicate a relatively high degree of confidence in reported statistical associations with respiratory health outcomes on days when virtually all monitored 8-hour O_3 concentrations were 75 ppb or below. She further agrees with the PA conclusion that although these analyses do not identify true design values, the presence of O_3-associated respiratory effects on such days provides insight into the types of health effects that could occur in locations with maximum ambient O_3 concentrations below the level of the current standard.

Compared to the single-city epidemiologic studies discussed above, the Administrator notes additional uncertainty in interpreting the relationships between short-term O_3 air quality in individual study cities and reported O_3 multicity effect estimates. In particular, she judges that the available multicity effect estimates in studies of short-term O_3 do not provide a basis for considering the extent to which reported O_3 health effect associations are influenced by individual locations with ambient O_3 concentrations low enough to meet the current O_3 standard, versus locations with O_3 concentrations that violate this standard.[88] While such uncertainties limit the extent to which the Administrator bases her conclusions on air quality in locations of multicity epidemiologic studies, she does note that O_3 associations with respiratory morbidity or premature mortality have been reported in several multicity studies when the majority of study locations (though not all study locations) would have met the current O_3 standard (PA, section 3.1.4.2) and that this information provides some support for the determination that it is appropriate to revise the current primary standard to afford requisite public health protection.

Looking across the body of epidemiologic evidence, the Administrator thus reaches the conclusion that analyses of air quality in study locations support the occurrence of adverse O_3-associated effects at ambient O_3 concentrations that met, or are likely to have met, the current standard. She further concludes that the strongest support for this conclusion comes from single-city studies of respiratory-related hospital admissions and emergency department visits associated with short-term O_3 concentrations, with some support also from multicity studies of morbidity or mortality. Given uncertainties in linking reported O_3 health effect associations with individual locations with ambient O_3 concentrations low enough to meet the current O_3 standard, versus locations with

[88] As noted in the proposal (see section II.E.4.d of the proposal), this uncertainty applies specifically to interpreting air quality analyses within the context of multicity effect estimates for short-term O_3 concentrations, where effect estimates for individual study cities are not presented (as is the case for the key O_3 studies analyzed in the PA, with the exception of the study by Stieb et al. (2009) where none of the city-specific effect estimates for asthma emergency department visits were statistically significant). This specific uncertainty does not apply to multicity epidemiologic studies of long-term O_3 concentrations, where multicity effect estimates are based on comparisons across cities. For example, see discussion of study by Jerrett et al. (2009) in the PA (section 3.1.4.3).

O_3 concentrations that violate this standard, the Administrator does not accept the commenter's contention that this body of evidence, and the Canadian multi-city studies in particular, compels adoption of a revised standard with a level below 70 ppb.

(13) *Comment:* One commenter maintains that EPA must base the level of the standard on concentrations of O_3 below the mean in epidemiological studies, rather than looking to highest concentration day in a study or some other metric drawn from the form of the NAAQS. This is because, in the commenter's view, health effects occur at concentrations both above and below the mean, with the bulk of effects occurring within one standard deviation of the mean. The commenter cites CASAC advice in support of its argument, referring to Frey and Samet (2012c) as well as statements of individual panel members.

Response: The EPA agrees that the information in the epidemiologic studies should be interpreted based on the total air quality distribution of the studies. However, given that the O_3 standard is based on the 3-year average of the 4th high 8-hour maximum daily O_3 concentration, the EPA does not agree with the commenter that it is appropriate to set level of the primary O_3 standard at or below the mean O_3 concentration reported in particular studies.[89] Rather, it is reasonable to ascertain whether the air quality in a given study area (or, for multi-city studies, areas) would have been allowed by the current standards. See, e,g, *State of Mississippi*, 744 F. 3d at 1345. If the air quality in the study areas would not be allowed by the current standard, then there is no necessary inference that whatever air quality caused the observed effects would be allowed had the standard been attained.

CASAC advice is not to the contrary. The statement from Frey and Samet (2012c) quoted by the commenter was in the context of causation, not level of a standard. The consensus letter thus states that "the EPA can utilize information from various studies regardless of whether the way in which ozone was assessed directly matches the form or averaging period used in the standard. The purpose is to infer the general causal relationship (i.e. shape of and magnitude of the concentration-response function) between exposure levels and risk of various occurrences." Similarly, the quoted statements of individual panel members do not support the commenter's contention (even putting aside the issue that these comments are individual, not consensus panel statements). These comments from individual Panel members were provided as part of CASAC's review of the first draft PA. These individual comments, which focused on the PA's analyses of air quality in locations of epidemiologic studies, were considered during the development of the second draft PA and the final PA. Thus, the approach to

[89] Note that this is a different situation than the primary annual $PM_{2.5}$ standard, which is based on an annual average form. In that situation, annual mean $PM_{2.5}$ concentrations are most relevant for consideration with regard to the standard level. Thus, in the most recent review of the PM NAAQS (78 FR 3086, January 15, 2013), the Administrator's decision on the level of the annual $PM_{2.5}$ standard was informed by the mean $PM_{2.5}$ concentrations present in locations of epidemiologic studies.

considering and interpreting air quality in locations of epidemiologic studies in the final PA reflects staff's consideration of the Panel members' advice that was highlighted by the commenters noted above. In their letter to the Administrator on the 2nd draft PA, CASAC cited PA analyses of air quality in study locations as part of its rationale for its recommendations on the existing primary standard. For example, CASAC stated that "[w]ith regard to epidemiologic studies the CASAC concurs with the EPA staff finding in the Second Draft PA that, compared to the current standard, a revised standard with a level of 70 ppb would be more effective in maintaining short-term ambient O_3 concentrations below those present in locations that provided the basis for positive and statistically significant health effect associations" (Frey, 2014b, p. 7). Thus, we do not agree with commenters who asserted that CASAC advice, including the advice of individual Panel members, supports an approach to viewing O_3 air quality in locations of epidemiologic studies that is different from the approach adopted in the final PA, and discussed in the proposed rule and in the preamble to the final rule.

Exposure and Risk Assessments

(14) *Comment:* Some commenters supporting levels below 70 ppb also asserted that quantitative analyses in the HREA are biased such that they understate O_3 exposures of concern and risks of O_3-induced FEV_1 decrements. Many of these comments are discussed above within the context of the adequacy of the current standard (section II.B.2.b in the preamble to the final rule), including comments pointing out that exposure and risk estimates are based on information from healthy adults rather than at-risk populations; comments noting that the exposure assessment evaluates 8-hour O_3 exposures rather than the 6.6-hour exposures used in controlled human exposure studies; and comments asserting that the EPA's exposure and risk analyses rely on people staying indoors on high pollution days (i.e., averting behavior).

Response: As discussed in section II.B.2.b in the preamble to the final rule, while the EPA agrees with certain aspects of these commenters' assertions, we do not agree with their overall conclusions. In particular, there are aspects of the HREA's quantitative analyses that, if viewed in isolation, would tend to either overstate or understate O_3 exposures and/or health risks. While commenters tended to focus on those aspects of the assessments that support their position, they tended to ignore aspects of the assessments that do not support their position (points that were often raised by commenters on the other side of the issue). Rather than viewing the potential implications of these aspects of the HREA assessments in isolation, the EPA considers them together, along with other issues and uncertainties related to the interpretation of exposure and risk estimates.

(15) *Comment:* Some commenters who advocated for a level below 70 ppb asserted that the exposure assessment could underestimate O_3 exposures for highly active populations, including outdoor workers and children who spend a large portion of time outdoors during summer. In support of these assertions, commenters highlighted sensitivity analyses conducted in the HREA.

Response: As noted in the HREA (Table 5-10), this aspect of the assessment is likely to have only a "low to moderate" impact on the magnitude of exposure estimates. To put this magnitude in perspective, HREA sensitivity analyses conducted in a single urban study area indicate that, regardless of whether exposure estimates for children are based on all available diaries or on a subset of diaries restricted to simulate highly exposed children, a revised standard with a level of 70 ppb is estimated to protect more than 99% of children from experiencing two or more exposures of concern at or above 70 ppb (HREA, Chapter 5 Appendices, Figure 5G-9).[90, 91] In contrast to the focus of commenters who supported a level below 70 ppb, other aspects of quantitative assessments, some of which were highlighted by commenters who opposed revising the current standard (section II.B.2 in the preamble to the final rule), tend to result in overestimates of O_3 exposures. These aspects are characterized in the HREA as having either a "low," a "low-to-moderate," or a "moderate" impact on the magnitudes of exposure estimates.

In its reviews of the HREA and PA, CASAC recognized many of the uncertainties and issues highlighted by commenters. Even considering these uncertainties, CASAC endorsed the approaches adopted by the EPA to assess O_3 exposures and health risks, and CASAC used exposure and risk estimates as part of the basis for their recommendations on the primary O_3 NAAQS (Frey, 2014b). Thus, as discussed in section II.B.2.b in the preamble to the final rule, the EPA disagrees with commenters who claim that the aspects of the quantitative assessments that they highlight lead to overall underestimates of exposures or health risks.[92]

(16) *Comment:* Some commenters contended that the level of the primary O_3 standard should be set below 70 ppb in order to compensate for the use of a form that allows multiple days with concentrations higher than the standard level. These groups submitted air quality analyses to support their point that the current 4th-high form allows multiple days per year with ambient O_3 concentrations above the level of the standard.

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[90] More specifically, based on all children's diaries, just under 0.1% of children are estimated to experience two or more exposures of concern at or above 70 ppb. Based on simulated profiles of highly exposed children, this estimate increased to just over 0.1% (HREA, Chapter 5 Appendices, Figure 5G-9).

[91] In addition, when diaries were selected to mimic exposures that could be experienced by outdoor workers, the percentages of modeled individuals estimated to experience exposures of concern were generally similar to the percentages estimated for children (i.e., using the full database of diary profiles) in the worst-case cities and years (i.e., cities and years with the highest exposure estimates) (HREA, section 5.4.3.2, Figure 5-14).

[92] As discussed in II.B.2.b in the preamble to the final rule, in weighing the various uncertainties, which can bias exposure results in different directions but tend to have impacts that are similar in magnitude (HREA, Table 5-10), and in light of CASAC's advice based on its review of the HREA and the PA, the EPA continues to conclude that the approach to considering estimated exposures of concern in the HREA, PA, the proposal and the preamble to the final rule reflects an appropriate balance, and provides an appropriate basis for considering the public health protectiveness of the primary O_3 standard.

Response: While the EPA does not dispute the results of air quality analyses submitted by these commenters, and agrees that 4th-high form allows multiple days per year with ambient O_3 concentrations above the level of the standard (3 days per year, on average over a 3-year period), the Agency disagrees with commenters' assertion that, because of this, the level of the primary O_3 standard should be set below 70 ppb. As discussed in the preamble to the final rule (section II.A.2), the quantitative assessments that informed the Administrator's proposed decision, presented in the HREA and considered in the PA and by CASAC, estimated O_3 exposures and health risks associated with air quality that "just meets" various standards with the current 8-hour averaging time and 4th-high, 3-year average form. Thus, in considering the degree of public health protection appropriate for the primary O_3 standard, the Administrator has considered quantitative exposure and risk estimates that are based a 4th-high form, and therefore on a standard that, as these commenters point out, allows multiple days per year with ambient O_3 concentrations above the level of the standard.

CASAC Advice

(17) *Comment:* Many commenters, including those representing major medical, public health, or environmental groups; some state agencies; and a large number of individual commenters, focused on CASAC advice in their rationale supporting levels below 70 ppb, and as low as 60 ppb. These commenters generally asserted that the EPA must give deference to CASAC. In some cases, these commenters expressed strong objections to a level of 70 ppb, noting CASAC policy advice that such a level would provide little margin of safety.

One commenter questioned the EPA's statement that "CASAC did not provide advice as to how far below 72 ppb adverse effects would likely occur" quoting 79 FR at 75305, which refers to Frey (2014b) (p. 5). The commenter maintained that CASAC in fact addressed this issue explicitly, in its statements that concentrations of O_3 down to 60 ppb "result in lung function decrements large enough to be judged an abnormal response by ATS and that could be adverse in individuals with lung disease" and that a standard set at a level of 70 ppb is still "of significant concern", citing to Frey (2014b) (p. 7). The commenter concluded that CASAC expressly found that there is substantial scientific certainty of adverse effects at 70 ppb, and that effects are expected to occur at levels below 70 ppb.

Commenters maintain that the proposal is inconsistent with CASAC advice in that it does not include 60 ppb within the range of a level for a primary standard, and that this deviation from CASC advice is not rationally explained. Specifically, the commenter states that:

- The proposal fails to address CASAC's own reasoning – namely, that adverse effects (including lung function decreases and inflammation) occur upon exposure of healthy adults at moderate exercise to O_3 concentrations of 60 ppb. CASAC also noted that a standard of 60 ppb

would eliminate single and multiple exposures of concern at the 60 ppb benchmark.

Response: The EPA agrees that CASAC advice is an important consideration in reaching a decision on the standard level (see e.g. CAA section 307 (d)(3)),[93] though not with commenters' conclusion that CASAC advice necessitates a standard level below 70 ppb. As discussed in the preamble to the final rule (section II.C.4.a), the Administrator carefully considered CASAC advice in the proposal, and she judged that her proposed decision to revise the level to within the range of 65 to 70 ppb was consistent with CASAC advice, based on the available science.

As in the proposal, in her final decision on level the Administrator notes CASAC's overall conclusion that "based on the scientific evidence from clinical studies, epidemiologic studies, animal toxicology studies, as summarized in the ISA, the findings from the exposure and risk assessments as summarized in the HREA, and the interpretation of the implications of all of these sources of information as given in the Second Draft PA…there is adequate scientific evidence to recommend a range of levels for a revised primary ozone standard from 70 ppb to 60 ppb" (Frey, 2014b, p. 8). Thus, CASAC used the health evidence and exposure/risk information to inform its range of recommended standard levels, a range that included an upper bound of 70 ppb based on the scientific evidence, and it did not use the evidence and information to recommend setting the primary O_3 standard at any specific level within the range of 70 to 60 ppb. In addition, CASAC further stated that "the choice of a level within the range recommended based on scientific evidence [i.e., 70 to 60 ppb] is a policy judgment under the statutory mandate of the Clean Air Act" (Frey, 2014b, p. ii).

In addition to its advice based on the scientific evidence, CASAC offered the "policy advice" to set the level below 70 ppb, stating that a standard level of 70 ppb "may not meet the statutory requirement to protect public health with an adequate margin of safety" (Frey, 2014b, p. ii). In supporting its policy advice to set the level below 70 ppb, CASAC noted the respiratory effects that have been shown to occur in controlled human exposure studies following exposures from 60 to 80 ppb O_3, and the extent to which various standard levels are estimated to allow the occurrence of population exposures that can result in such effects (Frey, 2014b, pp. 7-8).

The EPA agrees that an important consideration when reaching a decision on level is the extent to which a revised standard is estimated to allow the types of exposures shown in controlled human exposure studies to cause respiratory effects. In reaching her final decision that a level of 70 ppb is requisite to protect public health with an adequate margin of safety (in section II.C.4.c in the

[93] The EPA notes, of course, that the CAA places the responsibility for judging what standard is requisite with the Administrator and only requires that, if her decision differs in important ways from CASAC's advice, she explain her reasoning for differing.

preamble to the final rule), the Administrator carefully considers the potential for such exposures and effects. In doing so, she emphasizes the importance of setting a standard that limits the occurrence of the exposures about which she is most concerned (i.e., those for which she has the most confidence in the adversity of the resulting effects, which are repeated exposures of concern at or above 70 or 80 ppb, as discussed in II.C.4.b.i in the preamble to the final rule). Based on her consideration of information from controlled human exposure studies in light of CASAC advice and ATS recommendations, the Administrator additionally judges that there is important uncertainty in the extent to which the effects shown to occur following exposures to 60 ppb O_3 are adverse to public health (discussed in sections II.C.4.b.i and II.C.4.b.iii in the preamble to the final rule). However, based on the effects that have been shown to occur, CASAC advice indicating the importance of considering these effects, and ATS recommendations indicating the potential for adverse population-level effects (sections II.C.4.b.i and II.C.4.b.iii in the preamble to the final rule), she concludes that it is appropriate to give some consideration to the extent to which a revised standard could allow the respiratory effects that have been observed following exposures to 60 ppb O_3.

When considering the extent to which a revised standard could allow O_3 exposures that have been shown in controlled human exposures studies to result in respiratory effects, the Administrator is most concerned about protecting at-risk populations against repeated occurrences of such exposures of concern (section II.C.4.b.i, in the preamble to the final rule). In considering the appropriate metric for evaluating repeated occurrences of exposures of concern, the Administrator acknowledges that it is not clear from the evidence, or from the ATS recommendations, CASAC advice, or public comments, how the number of exposures of concern could impact the seriousness of the resulting effects, especially at lower exposure concentrations. Therefore, the Administrator judges that focusing on HREA estimates of two or more exposures of concern provides a health-protective approach to considering the potential for repeated occurrences of exposures of concern that could result in adverse effects. She notes that other possible metrics for considering repeated occurrences of exposures of concern (e.g., 3 or more, 4 or more, etc.) would result in smaller exposure estimates.

As discussed further in section II.C.4.c of the preamble to the final rule, the Administrator notes that a revised standard with a level of 70 ppb is estimated to eliminate the occurrence of two or more exposures of concern to O_3 concentrations at or above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O_3 concentrations at or above 70 ppb (see Table 1 in the preamble to the final rule). For the 70 ppb benchmark, this reflects about a 90% reduction in the number of children estimated to experience two or more exposures of concern, compared to the current standard.[94] Even considering the worst-case urban study area and worst-case year evaluated in the HREA, a

[94] Percent reductions in this section refer to reductions in the number of children in HREA urban study areas (averaged over the years evaluated in the HREA) estimated to experience exposures of concern, based on the information in Table 1 in the preamble to the final rule.

standard with a level of 70 ppb is estimated to protect more than 99% of children from experiencing two or more exposures of concern to O_3 concentrations at or above 70 ppb (see Table 1 in the preamble to the final rule).

Though the Administrator judges that there is greater uncertainty with regard to the occurrence of adverse effects following exposures as low as 60 ppb, she notes that a revised standard with a level of 70 ppb is estimated to protect the vast majority of children in urban study areas (i.e., about 96% to more than 99% in individual areas) from experiencing two or more exposures of concern at or above 60 ppb. Compared to the current standard, this represents a reduction of more than 60% in exposures of concern for the 60 ppb benchmark (Table 1 in the preamble to the final rule). Given the Administrator's uncertainty regarding the adversity of effects following exposures to 60 ppb O_3, and her health-protective approach to considering repeated occurrences of exposures of concern, the Administrator judges that this degree of protection is appropriate and that it reflects substantial protection against the occurrence of O_3-induced effects, including effects for which she judges the adversity to public health is uncertain.

With respect to exposures of concern at 60 ppb, the commenter is mistaken in stating CASAC indicated that a standard set at the level of 60 ppb would eliminate exposures of concern at that level, since the estimates show otherwise. See Table 1 in the preamble to the final rule, showing exposures of concern remaining in both the 2006-2010 study period, and in worst case years. Nor does a standard established at that level eliminate estimated occurrences of single or multiple lung function decrements (FEV_1 decrements $\geq$ 10%, Table 2 in the preamble to the final rule).

While being less concerned about single occurrences of exposures of concern, especially at lower exposure concentrations, the Administrator also notes that a standard with a level of 70 ppb is estimated to (1) virtually eliminate all occurrences of exposures of concern at or above 80 ppb; (2) protect $\geq$ about 99% of children in urban study areas from experiencing any exposures of concern at or above 70 ppb; and (3) to achieve substantial reductions (i.e., about 50%), compared to the current standard, in the occurrence of one or more exposures of concern at or above 60 ppb (Table 1,preamble to the final rule).

Given the information and advice noted in the preamble to the final rule in sections II.C.4.b.i and II.C.4.b.iii, the Administrator judges that a revised standard with a level of 70 ppb will effectively limit the occurrence of the O_3 exposures for which she has the most confidence in the adversity of the resulting effects (i.e., based on estimates for the 70 and 80 ppb benchmarks). She further judges that such a standard will provide a large degree of protection against O_3 exposures for which there is greater uncertainty in the adversity of effects (i.e., those observed following exposures to 60 ppb O_3), contributing to the margin of safety of the standard. Given the considerable protection provided against repeated exposures of concern for all of the benchmarks evaluated, including the 60 ppb benchmark, the Administrator judges that a standard with a level of 70 ppb will provide a

substantial margin of safety against the adverse O_3-induced effects shown to occur following exposures at or above 72 ppb, and judged by CASAC likely to occur following exposures somewhat below 72 ppb.[95]

Contrary to the conclusions of commenters who advocated for a level below 70 ppb, the Administrator judges that her final decision is consistent with CASAC's advice, based on the scientific evidence, and consistent with CASAC's focus on setting a revised standard to further limit the occurrence of the respiratory effects observed in controlled human exposure studies, including effects observed following exposures to 60 ppb O_3. Given her judgments and conclusions discussed in the preamble to the final rule, and given that the CAA reserves the choice of the standard that is requisite to protect public health with an adequate margin of safety for the judgment of the EPA Administrator (and, of course, "based on … [the air quality] criteria") (CAA section 109 (b)(1)), she disagrees with commenters who asserted that CASAC advice necessitates a level below 70 ppb, and as low as 60 ppb. The Administrator's final conclusions on level are discussed in more detail in section II.C.4.c of the preamble to the final rule.

(18) *Comment:* The same commenter asserts that in rejecting CASAC advice to include 60 ppb within the range of potential revised standards, the Agency indicated that a standard of 60 ppb would place a large amount of weight on the potential public health importance of further reducing occurrence of O_3-induced lung function decrements of 10 and 15% (citing to 79 FR 75309/3). The commenter asserts that "potential public health importance" is merely a policy consideration which is not responsive to CASAC's science-based judgment that adverse effects occur in healthy adults at exposures to 60 ppb O_3, and exposures of at-risk populations to even lower concentrations could result in adverse health effects. In any case, the commenter asserts that the EPA has offered no basis for questioning why reducing exposures that result in reductions of decrements of 10 and 15% is not of public health importance.

Response: As noted above, CASAC concluded that "based on the scientific evidence from clinical studies, epidemiologic studies, animal toxicology studies, as summarized in the ISA, the findings from the exposure and risk assessments as summarized in the HREA, and the interpretation of the implications of all of these sources of information as given in the Second Draft PA…there is adequate scientific evidence to recommend a range of levels for a revised primary ozone standard from 70 ppb to 60 ppb" (Frey, 2014b, p. 8). The Administrator's

[95] As discussed in the preamble to the final rule (II.C.4.b.i), when commenting on the extent to which the study by Schelegle et al. (2009) suggests the potential for adverse effects following O_3 exposures below 72 ppb, CASAC stated the following: "[I]f subjects had been exposed to ozone using the 8-hour averaging period used in the standard [rather than the 6.6-hour exposures evaluated in the study], adverse effects could have occurred at lower concentration. Further, in our judgment, the level at which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma" (Frey, 2014b, p. 5).

consideration of CASAC advice is discussed in detail in the immediately preceding responses.

In addition, the Administrator's consideration of risk estimates for O_3-induced FEV_1 decrements ≥ 10% and 15% is discussed in sections II.B.2, II.B.3, II.C.4.b, and II.C.4.c of the preamble to the final rule. For example, the Administrator judges that a revised standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O_3-induced lung function decrements (≥ 10%, ≥ 15%) in children, including children with asthma. Specifically, a revised standard with a level of 70 ppb is estimated to reduce the risk of two or more O_3-induced decrements by about 30% and 20% for decrements ≥ 15% and 10%, respectively (Table 2 in the preamble to the final rule). However, as discussed in section II.C.4.b.i of the preamble to the final rule, the Administrator judges that there are important uncertainties in using lung function risk estimates as a basis for considering the occurrence of adverse effects in the population given (1) the ATS recommendation that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" (ATS, 2000); (2) uncertainty in the extent to which a transient population-level decrease in FEV_1 would increase the risk of other, more serious respiratory effects in that population (i.e., per ATS recommendations on population-level risk); and (3) that CASAC did not advise considering a standard that would be estimated to eliminate O_3-induced lung function decrements ≥ 10 or 15% (Frey, 2014b). Moreover, as at proposal, the Administrator notes that the variability in lung function risk estimates across urban study areas is often greater than the differences in risk estimates between various standard levels (Table 2 in the preamble to the final rule). Given this, and the resulting considerable overlap between the ranges of lung function risk estimates for different standard levels,[96] the Administrator puts limited weight on the lung function risk estimates for distinguishing between the degrees of public health protection provided by alternative standard levels. Therefore, the Administrator judges that while a standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O_3-induced lung function decrements (≥10%, 15%) in children, including children with asthma, she also judges that estimated risks of O_3-induced lung function decrements provide a more limited basis than exposures of concern for distinguishing between the appropriateness of the health protection afforded by a standard level of 70 ppb versus lower levels.

Adequate Margin of Safety

[96] For example, the average percentage of children estimated to experience two or more decrements ≥ 10% ranges from approximately 6 to 11% for a standard level of 70 ppb, up to about 9% for a level of 65 ppb, and up to about 6% for a level of 60 ppb (Table 2 in the preamble to the final rule). In contrast, the average percentage of children estimated to experience two or more exposures of concern for the 60 ppb benchmark ranges from approximately 0.5 to 3.5% for a level of 70 ppb, up to 0.8% for a level of 65 ppb, and up to 0.2% for a level of 60 ppb (Table 1 in the preamble to the final rule).

(19) *Comment:* Some commenters who supported revising the current standard disputed that a standard with a level of 70 ppb could provide an adequate margin of safety. To support this conclusion, such commenters often contended that a level of 70 ppb would allow adverse effects, based on the evidence for effects in healthy adults below 75 ppb ppb and on CASAC advice. In some cases, these commenters asserted that the proposal failed to justify the conclusion that a standard with a level of 70 ppb can provide an adequate margin of safety.

Response: The EPA disagrees with these commenters' assertion that a level of 70 ppb fails to provide an adequate margin of safety. As discussed in section II.C.4.c of the preamble to the final rule, we note that the determination of what constitutes an adequate margin of safety is expressly left to the judgment of the EPA Administrator. See *Lead Industries Association v. EPA*, 647 F.2d at 1161-62; *Mississippi*, 744 F. 3d at 1353. We further note that in evaluating how particular standards address the requirement to provide an adequate margin of safety, it is appropriate to consider such factors as the nature and severity of the health effects, the size of sensitive population(s) at risk, and the kind and degree of the uncertainties present (e.g., see I.B of the preamble to the final rule). Consistent with past practice and long-standing judicial precedent, the Administrator takes the need for an adequate margin of safety into account as an integral part of her decision-making on the appropriate level, averaging time, form, and indicator of the standard.[97]

Taken together, the Administrator concludes that the evidence from controlled human exposure studies provides strong support for her conclusion that a revised standard with a level of 70 ppb is requisite to protect the public health with an adequate margin of safety. She bases this conclusion, in part, on the fact that such a standard level would be well below the O_3 exposure concentration shown to result in the widest range of respiratory effects (i.e., 80 ppb), and below the lowest O_3 exposure concentration shown to result in the adverse combination of lung function decrements and respiratory symptoms (i.e., 72 ppb). See *Lead Industries*, 647 F. 2d at 1160 (setting NAAQS at level well below the level where the clearest, more serious adverse effects occur, and at a level eliminating most "sub-clinical effects" provides an adequate margin of safety).

As discussed in section II.C.4.b.i of the preamble to the final rule, the Administrator also notes that a revised O_3 standard with a level of 70 ppb can provide substantial protection against the broader range of O_3 exposure concentrations that have been shown in controlled human exposure studies to result in respiratory effects, including exposure concentrations below 70 ppb. Therefore, as discussed in the proposal, in considering the degree of protection provided by a revised primary O_3 standard, the Administrator considers the extent to which that standard would be expected to limit population exposures of

[97] See, e.g. *NRDC v. EPA*, 902 F. 2d 962, 973-74 (D.C. Cir. 1990).

concern to the broader range of O_3 exposure concentrations shown to result in health effects.

Due to interindividual variability in responsiveness, the Administrator notes that not every occurrence of an exposure of concern will result in an adverse effect (II.C.4.b.i of the preamble to the final rule). Moreover, repeated occurrences of some of the effects demonstrated following exposures of concern could increase the likelihood of adversity (ISA, Section 6.2.3, p. 6-76). In particular, she notes that the types of respiratory effects that can occur following exposures of concern, particularly if experienced repeatedly, provide a plausible mode of action by which O_3 may cause other more serious effects. Therefore, as in the proposal, the Administrator is most concerned about protecting at-risk populations against repeated occurrences of exposures of concern. In considering the appropriate metric for evaluating repeated occurrences of exposures of concern, the Administrator acknowledges that it is not clear from the evidence, or from the ATS recommendations, CASAC advice, or public comments, how particular numbers of exposures of concern could impact the seriousness of the resulting effects, especially at lower exposure concentrations. Therefore, the Administrator judges that focusing on HREA estimates of two or more exposures of concern provides a health-protective approach to considering the potential for repeated occurrences of exposures of concern that could result in adverse effects.

Based on her consideration of adversity discussed above (II.A.1.b.i) and in the preamble to the final rule (e.g., II.B.2.b.i, II.B.3, II.C.4.b, II.C.4.c of the final rule), the Administrator places the most emphasis on setting a standard that appropriately limits repeated occurrences of exposures of concern at or above the 70 and 80 ppb benchmarks. She notes that a revised standard with a level of 70 ppb is estimated to eliminate the occurrence of two or more exposures of concern to O_3 concentrations at or above 80 ppb and to virtually eliminate the occurrence of two or more exposures of concern to O_3 concentrations at or above 70 ppb for all children and children with asthma, even in the worst-case year and location evaluated.

While she is less confident that adverse effects will occur following exposures to O_3 concentrations as low as 60 ppb, as discussed above and in the preamble to the final rule, the Administrator judges that it is also appropriate to consider estimates of exposures of concern for the 60 ppb benchmark. Consistent with this judgment, although CASAC advice regarding the potential adversity of effects at 60 ppb was less definitive than for effects at 72 ppb, CASAC did clearly advise the EPA to consider the extent to which a revised standard is estimated to limit the effects observed following 60 ppb exposures (Frey, 2014b). Therefore, the Administrator considers estimated exposures of concern for the 60 ppb benchmark, particularly considering the extent to which the health protection provided by a revised standard includes a margin of safety against the occurrence of adverse O_3-induced effects. The Administrator notes that a revised standard with a level of 70 ppb is estimated to protect the vast majority of children in urban study areas (i.e., about 96% to more than 99% of children in individual areas) from experiencing two or

more exposures of concern at or above 60 ppb. Compared to the current standard, this represents a reduction of more than 60%.

Given the considerable protection provided against repeated exposures of concern for all of the benchmarks evaluated, including the 60 ppb benchmark, the Administrator judges that a standard with a level of 70 ppb will incorporate a margin of safety against the adverse O_3-induced effects shown to occur following exposures at or above 72 ppb, and judged likely to occur following exposures somewhat below 72 ppb.

While the Administrator is less concerned about single occurrences of O_3 exposures of concern, especially for the 60 ppb benchmark, she judges that estimates of one or more exposures of concern can provide further insight into the margin of safety provided by a revised standard. In this regard, she notes that a standard with a level of 70 ppb is estimated to (1) virtually eliminate all occurrences of exposures of concern at or above 80 ppb; (2) protect the vast majority of children in urban study areas from experiencing any exposures of concern at or above 70 ppb (i.e., ≥ about 99%, based on mean estimates; Table 1); and (3) to achieve substantial reductions, compared to the current standard, in the occurrence of one or more exposures of concern at or above 60 ppb (i.e., about a 50% reduction; Table 1 in the preamble to the final rule). The Administrator judges that these results provide further support for her conclusion that a standard with a level of 70 ppb will incorporate an adequate margin of safety against the occurrence of O_3 exposures that can result in effects that are adverse to public health.

The Administrator additionally judges that a standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O_3-induced lung function decrements (≥ 10%, ≥ 15%) in children, including children with asthma. Specifically, a revised standard with a level of 70 ppb is estimated to reduce the risk of two or more O_3-induced decrements by about 30% and 20% for decrements ≥ 15 and 10%, respectively (Table 2 in the preamble to the final rule). However, as discussed in section II.C.4.b.i of the preamble to the final rule, the Administrator judges that there are important uncertainties in using lung function risk estimates as a basis for considering the occurrence of adverse effects in the population given (1) the ATS recommendation that "a small, transient loss of lung function, by itself, should not automatically be designated as adverse" (ATS, 2000); (2) uncertainty in the extent to which a transient population-level decrease in FEV_1 would increase the risk of other, more serious respiratory effects in that population (i.e., per ATS recommendations on population-level risk); and (3) that CASAC did not advise considering a standard that would be estimated to eliminate O_3-induced lung function decrements ≥ 10 or 15% (Frey, 2014b). Moreover, as at proposal, the Administrator notes that the variability in lung function risk estimates across urban study areas is often greater than the differences in risk estimates between

various standard levels (Table 2 in the preamble to the final rule).[98] Given this, and the resulting considerable overlap between the ranges of lung function risk estimates for different standard levels, the Administrator puts limited weight on the lung function risk estimates for distinguishing between the degrees of public health protection provided by alternative standard levels. Therefore, the Administrator judges that while a standard with a level of 70 ppb would be expected to result in important reductions, compared to the current standard, in the population-level risk of O_3-induced lung function decrements ($\geq$10%, 15%) in children, including children with asthma, she also judges that estimated risks of O_3-induced lung function decrements provide a more limited basis than exposures of concern for distinguishing between the appropriateness of the health protection afforded by a standard level of 70 ppb versus lower levels.

The Administrator also considers the epidemiologic evidence and the quantitative risk estimates based on information from epidemiologic studies. As discussed in the proposal, and above in the EPA's responses to significant comments, although the Administrator acknowledges the important uncertainties in using the O_3 epidemiologic studies as a basis for selecting a standard level, she notes that these studies can provide perspective on the degree to which O_3-associated health effects have been identified in areas with air quality likely to have met various standards. Specifically, the Administrator notes analyses in the PA (section 4.4.1) indicating that a revised standard with a level of 70 ppb would be expected to require additional reductions, beyond those required by the current standard, in the short- and long-term ambient O_3 concentrations that provided the basis for statistically significant O_3 health effect associations in both the single-city and multicity epidemiologic studies evaluated. As discussed in the preamble to the final rule, while the Administrator concludes that these analyses support a level at least as low as 70 ppb, based on a study reporting health effect associations in a location that met the current standard over the entire study period but that would have violated a revised standard with a level of 70 ppb,[99] she further judges that they are of more limited utility for distinguishing between the appropriateness of

[98] For example, the average percentage of children estimated to experience two or more decrements $\geq$ 10% ranges from approximately 6 to 11% for a standard level of 70 ppb, up to about 9% for a level of 65 ppb, and up to about 6% for a level of 60 ppb (Table 2 in the preamble to the final rule). In contrast, the average percentage of children estimated to experience two or more exposures of concern for the 60 ppb benchmark ranges from approximately 0.5 to 3.5% for a level of 70 ppb, up to 0.8% for a level of 65 ppb, and up to 0.2% for a level of 60 ppb.

[99] As discussed above (II.B.2.c.ii and II.B.3 of the preamble to the final rule), the study by Mar and Koenig (2009) reported positive and statistically significant associations with respiratory emergency department visits in a location that would have met the current standard over the entire study period, but violated a standard with a level of 70 ppb. In addition, air quality analyses in the locations of two additional studies highlighted in sections II.B.2 and II.B.3 of the final rule (Silverman and Ito, 2010; Strickland et al., 2010) were used in the PA to inform staff conclusions on the adequacy of the current primary O_3 standard. However, they did not provide insight into the appropriateness of standard levels below 75 ppb and, therefore, these analyses were not used to inform conclusions on potential alternative standard levels lower than 75 ppb (PA, Chapters 3 and 4). See *Mississippi*, 744 F. 3d at 1352-53 (study appropriate for determining causation may not be probative for determining level of a revised standard).

the health protection estimated for a standard level of 70 ppb and the protection estimated for lower levels. Thus, the Administrator notes that a revised standard with a level of 70 ppb will provide additional public health protection, beyond that provided by the current standard, against the clearly adverse effects reported in epidemiologic studies, and would not allow the air quality distribution associated with adverse effects in the Mar and Koenig (2009) study. She judges that a standard with a level of 70 ppb strikes an appropriate balance between setting the level to require reductions in the ambient O_3 concentrations associated with statistically significant health effects in epidemiologic studies, while not being more protective than necessary in light of her considerable uncertainty in the extent to which studies clearly show O_3-attributable effects at lower ambient O_3 concentrations. This judgment is consistent with the Administrator's conclusions based on information from controlled human exposure studies, as discussed above.

With regard to epidemiology-based risk estimates, the Administrator takes note of the CASAC conclusion that "[a]lthough the estimates for short-term exposure impacts are subject to uncertainty, the data supports a conclusion that there are meaningful reductions in mean premature mortality associated with ozone levels lower than the current standard" (Frey, 2014a, p. 10). While she concludes that epidemiology-based risk analyses provide only limited support for any specific standard level, consistent with CASAC advice the Administrator judges that, compared to the current standard, a revised standard with a level of 70 ppb will result in meaningful reductions in the mortality and respiratory morbidity risk that is associated with short-or long-term ambient O_3 concentrations.

Given all of the evidence and information discussed above, the Administrator judges that a standard with a level of 70 ppb is requisite to protect public health with an adequate margin of safety, and that a level below 70 ppb would be more than "requisite" to protect the public health. In reaching this conclusion, she notes that a decision to set a lower level would place a large amount of emphasis on the potential public health importance of (1) further reducing the occurrence of O_3 exposures of concern, though the exposures about which she is most concerned are estimated to be almost eliminated with a level of 70 ppb, and lower levels would be expected to achieve virtually no additional reductions in these exposures (see Table 1 in the preamble to the final rule); (2) further reducing the risk of O_3-induced lung function decrements $\geq$ 10 and 15%, despite having less confidence in judging the potential adversity of lung function decrements alone and the considerable overlap between risk estimates for various standard levels that make it difficult to distinguish between the risk reductions achieved; (3) further reducing ambient O_3 concentrations, relative to those in locations of epidemiologic studies, though associations have not been reported for air quality that would have met a standard with a level of 70 ppb across all study locations and over entire study periods, and despite her consequent judgment that air quality analyses in epidemiologic study locations are not informative regarding the additional degree of public health protection that would be afforded by a standard set at a level below 70 ppb; and (4) further reducing epidemiology-based

risk estimates, despite the important uncertainties in those estimates. As discussed in this section and in the responses to significant comments above, the Administrator does not agree that it is appropriate to place significant weight on these factors or to use them to support the appropriateness of standard levels below 70 ppb O_3. Compared to an O_3 standard level of 70 ppb, the Administrator concludes that the extent to which lower standard levels could result in further public health improvements becomes notably less certain.

For all of the above reasons, the Administrator concludes that a primary O_3 standard with an 8-hour averaging time; a 3-year average, 4th-high form; and a level of 70 ppb is requisite to protect public health, including the health of at-risk populations, with an adequate margin of safety.

(20) *Comment:* Some commenters disputed the EPA's view that NAAQS are not meant to be zero-risk standards. These commenters asserted that *State of Mississippi* does not stand for the proposition that NAAQS are not meant to be zero-risk standards, and that any suggestive statements in the opinion are dicta (citing to 744 F. 3d at 1343).

Response: This issue is discussed in more detail above and in the preamble to the final rule (section II.C.4.b). In summary, *State of Mississippi* holds (not states in *dicta,* as the commenter would have it) that *"*we have previously acknowledged the impossibility of eliminating all risk of health effects from 'non-threshold' pollutants like ozone" (744 F. 3d at 1351, referring to *ATA III*, 283 F. 3d at 360. The same court's approving citation to Justice Breyer's concurrence in Whitman *(744 F. 3d at 1343)* likewise indicates that the D.C. Circuit does not read the Act as requiring the elimination of all adverse effects. Indeed, the very case that the commenter cites in support of NAAQS having to be zero-risk, in fact holds that EPA established a primary NAAQS with an adequate margin of safety where the standard eliminates most but not all subclinical effects (effects found by the court to be adverse) and provided an adequate margin of safety because it was substantially below the level at which more serious adverse effects occurred. *Lead Industries*, 647 F. 2d at 1156, 1157, 1158, 1160, 1161. This is very similar to the approach adopted here, where the standard eliminates all of the clearly adverse effects (i.e. eliminates multiple exposures at the 70 ppb and 80 ppb benchmarks and virtually all individual annual exposures at those benchmarks (Table 1 to final preamble),[100] and establishes a standard resulting in air quality with lower distributions of O_3 than found in the sole epidemiologic study where an area not meeting the current standard exhibited statistically significant associations with adverse respiratory effects), eliminates nearly all of the exposures of concern about which there are scientific uncertainties as to adversity of effects but certainty that not all persons exposed will experience any effects, and eliminates

[100] Single exposures could leave 0-0.1% of children in the urban study areas exposed to single annual exposures at the 80 ppb benchmark, and 0.1-1.2% of children exposed to single annual exposures at the 70 ppb benchmark. Table 1 to preamble to the final rule. It is to be remembered that exposures do not necessarily lead to effects of any type, much less to adverse effects, due to interindividual variability.

occurrence of the great percentage of lung function decrements, again an effect about which there is uncertainty as to its adversity.

3. Communication of Public Health Information

Recognizing the importance of revising the AQI in a timely manner to be consistent with any revisions to the NAAQS, EPA proposed conforming changes to the AQI, in connection with the Agency's proposed decision on revisions to the O_3 NAAQS. The EPA proposed to revise the AQI for O_3 by setting an AQI value of 100 equal to the level of the revised O_3 standard (65-70 ppb). The EPA also proposed to revise the following breakpoints: an AQI value of 50 to within a range from 49-54 ppb; an AQI value of 150 to 85 ppb; an AQI value of 200 to 105 ppb, and an AQI value of 300 to 200 ppb. All these levels are averaged over 8 hours. The EPA did not propose to change the level at the top of the index (i.e., AQI value of 500) that typically is set equal to the Significant Harm Level (40 CFR 51.16), which would apply to state contingency plans.

With respect to reporting requirements (40 CFR Part 58, section 58.50), EPA proposed to revise 40 CFR Part 58, section 58.50 (c) to determine the areas subject to AQI reporting requirements based on the latest available census figures, rather than the most recent decennial U.S. census.[101] This change is consistent with our current practice of using the latest population figures to make monitoring requirements more responsive to changes in population.

EPA received many comments on the proposed changes to the AQI. Three 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 an AQI value of 100 should be set equal to the level of the primary standard and the other breakpoints adjusted accordingly; and, (3) whether the AQI reporting requirements should be based on the latest available census figures rather than the most recent decennial census.

(1) *Comment:* Some industry commenters stated that the AQI should not be revised at all, even if the level of the primary O_3 standard is revised. In support of this position, these commenters stated that the proposed conforming changes to the AQI would lower O_3 levels in each category, and would mean that air quality that is actually improving would be reported as less healthy. According to commenters, the revised AQI would fail to capture these improvements and potentially mislead the public into thinking that air quality has degraded and that EPA and state regulators are not doing their jobs. These commenters noted that there is no requirement to revise the AQI, and that the CAA does not tie the AQI to the standards, stating that the purpose of section 319(a) of the CAA is to provide a consistent, uniform means of gauging air quality. These commenters further asserted that EPA's proposed changes run counter to that uniformity by changing the air quality significance of a given index value and category and that retention of the current AQI breakpoints would allow continued uniform information on air quality. Commenters stated that it is important that the EPA

[101] Under 40 CFR 58.50, any MSA with a population exceeding 350,000 is required to report AQI data.

clearly communicates that the immediate increases in moderate rated days are due to AQI breakpoint adjustment and not due to a sudden decline in air quality.

Response: EPA disagrees with commenters who stated that the AQI should not be linked to the primary standards. As noted in the August 4, 1999, rulemaking (64 FR 149, 42531) that established the current AQI, the EPA established the nationally uniform air quality index, called the Pollutant Standards Index (PSI), in 1976 to meet the needs of state and local agencies with the following advantages: it sends a clear and consistent message to the public by providing nationally uniform information on air quality; it is keyed as appropriate to the NAAQS and the Significant Harm Level which have a scientific basis relating air quality and public health; it is simple and easily understood by the public while communicating information reflecting detailed scientific basis; it provides a framework for reflecting changes to the NAAQS; and it can be forecasted to provide advance information on air quality.

Both the PSI and AQI have historically been normalized across pollutants by defining an index value of 100 as the numerical level of the short-term (i.e., averaging time of 24-hours or less) primary NAAQS for each pollutant. Moreover, this approach does not mislead the public. Just the opposite. Since the establishment of the AQI, the EPA and state and local air agencies and organizations have developed experience in educating the public about changes in the standards and, concurrently, related changes to AQI breakpoints and advisories. When the standards change, EPA and state and local agencies have tried to help the public understand that air quality is not getting worse, it's that the health evidence underlying the standards and the AQI has changed. EPA's Air Quality System (AQS), the primary repository for air quality monitoring data, is also adjusted to reflect the revised breakpoints. Specifically, all historical AQI values in AQS are recomputed with the revised breakpoints, so that all data queries and reports downstream of AQS will show appropriate trends in AQI values over time.

(2) *Comment:* Commenters (e.g., API) estimated the increased proportion of days in the moderate category and above in 10 metropolitan areas for 2013 and also for 2025 for 4 cities from the original 10 that were estimated to attain a standard below 70 ppb, to compare with 2013. This commenter noted that the change in the proposed AQI breakpoint between "good" and "moderate" would result in a larger number of days that did not meet the "good" criteria. They went further to claim that the change in breakpoints would result in fewer "good" days in the year 2025 (using the new breakpoint) than occurred in 2013 (using the old breakpoints) despite substantial improvement in air quality over that time period.

Response: Although we do not contest the assertion that the new AQI breakpoints will lead to fewer green days in the near future, we do not agree that commenters' analysis sufficiently demonstrates that there would be fewer green days in 2025 than in 2013. In their analysis, they compared observed 2013 data with modeled 2025 data without doing any model performance evaluation for AQI categories or

comparison of current year modeled and observed data. The current year observations are not directly comparable to the future-year modeling data without some such evaluation and, as such, we cannot support their quantitative conclusions.

(3) *Comment:* State and local agencies and their organizations, environmental and medical groups, and members of the public overwhelmingly supported revising the AQI when the level of the standard is revised. Even state agencies that did not support revising the standard, expressed support for revising the AQI at the same time as the standard, if the standard is revised.

Response: Recognizing the importance of the AQI as a communication tool that allows members of the public to take exposure reduction measures when air quality poses health risks, the EPA agrees with these comments about revising the AQI at the same time as the primary standard. The EPA agrees with state and local agency commenters that its historical approach of setting an AQI value of 100 equal to the level of the revised 8-hour primary O_3 standard is appropriate, both from a public health and a communication perspective.

(4) *Comment:* In general, commenters who supported revising the AQI when the standard is revised, also supported setting an AQI value of 100 equal to the level of the 8-hour primary O_3 standard.

Response: The EPA agrees with these commenters. With respect to an AQI value of 100, the EPA is taking final action to set an AQI value of 100 equal to the level of the 8-hour primary standard at 70 ppb O_3.

(5) *Comment:* With respect to proposed changes to other AQI breakpoints, some state and local agency commenters expressed general support for all the changes in O_3 breakpoints (in Table 2 of Appendix G in the preamble to the final rule). In addition, we received a few comments specifically about the breakpoint between the good and moderate categories. One state expressed the view that forecasting the AQI for O_3 is not an exact science, so it is important to provide a range large enough to reasonably predict O_3 concentrations for the following day ($\geq$ 20 ppb). Although not supporting revision of the standard, this state recommended that if the primary standard was revised to 70 ppb, the lower end of moderate category should be set at 50 ppb to allow for a 20 ppb spread in that category. Several commenters recommending a breakpoint between the good and moderate categories of no higher than 50 ppb stated that this breakpoint should be set on health information, pointing to epidemiologic data and the World Health Organization guidelines.

Response: The Agency agrees that AQI breakpoints should take into consideration health information when possible, and also that it is important for AQI categories to span ranges large enough to support accurate forecasting. The EPA is setting the breakpoint at the lower end of the moderate category at 55 ppb, which is 15 ppb below the level of the standard of 70 ppb. This is consistent with

past practice of making a proportional adjustment to this AQI breakpoint, relative to an AQI value of 100 (i.e., 70 ppb), and also retains the current practice of providing a 15 ppb range in the moderate category to allow for accurate forecasting. This level is below the lowest concentration (i.e., 60 ppb) that has been shown in controlled human exposure studies of healthy adults to cause moderate lung function decrements (i.e., FEV_1 decrements $\geq$ 10%, which could be adverse to people with lung disease), large lung function decrements (i.e., FEV_1 decrements $\geq$ 20%) in a small proportion of people, and airway inflammation.

(6) *Comment:* There were fewer comments on proposed changes to the AQI values of 150, 200 and 300. Again, some state and local agency commenters expressed general support for proposed changes to the AQI. Some states specifically supported these breakpoints. However, a commenter suggested setting an AQI value at the lower end of the unhealthy category, at a level much lower than 85 ppb, since they state that it is a key threshold that is often used in air quality action day programs as a trigger to encourage specific behavior modifications or reduce emissions of O_3 precursors (e.g., by taking public transportation to work). This commenter stated that setting the breakpoint at 85 ppb would, in the Agency's own rationale, not require the triggering of these pollution reduction measures until air quality threatened to impact 25% of people exposed.

Response: We disagree with this commenter because EPA does not have any requirements for voluntary programs. State and local air agencies have discretion to set the trigger for voluntary action programs at whatever level they choose, and they are currently set at different levels, not just at the unhealthy breakpoint specified in the comment. For example, Houston, Galveston and Brazoria, TX metropolitan area calls ozone action days when air quality reaches the unhealthy for sensitive groups category. For more information about action days programs across the U.S. see the AirNow website (www.airnow.gov) and click on the link to AirNow Action Days. The unhealthy category represents air quality where there are general population-level effects. We believe that setting the breakpoint between the unhealthy for sensitive groups and unhealthy categories, at 85 ppb where, as discussed in section III.A of the preamble to the final rule, controlled human exposure studies of young, healthy adults exposed to O_3 while engaged in quasi-continuous moderate exercise for 6.6 hours indicate that up to 25% of exposed people are likely to have moderate lung function decrements and up to 7% are likely to have large lung function decrements (McDonnell et al., 2012; Figure 7) is appropriate. A smaller proportion of inactive or less active individuals would be expected to experience lung function decrements at 85 ppb. Moreover, a breakpoint at 85 ppb allows for category ranges large enough for accurate forecasting. Accordingly, the EPA is adopting the proposed revisions to the AQI values of 150, 200 and 300.

As noted earlier, the EPA proposed to revise 40 CFR Part 58, section 58.50 (c) to determine the areas subject to AQI reporting requirements based on the latest available census figures, rather than the most recent decennial U.S. census.

(7) *Comment:* A total of five state air monitoring agencies provided comments on this proposed change. Four agencies supported the proposal. One state commenter did not support the proposal, noting that the change would unnecessarily complicate AQI reporting and possibly increase reporting burdens in an unpredictable manner.

Response: The EPA notes that the majority of monitoring network minimum requirements listed in Appendix D to Part 58 include a reference to "latest available census figures." Minimum network requirements for O_3, $PM_{2.5}$, SO_2, and NO_2 all include this language in the regulatory text and monitoring agencies have successfully adopted these processes into their planning activities and the subsequent revision of their annual monitoring network plans which are posted for public review. Annual population estimates are easily obtainable from the U.S. Census Bureau and the EPA does not believe the burden in tracking these annual estimates is excessive or complicated.[102] Although the changes in year to year estimates are typically modest, there are MSAs that are approaching (or have recently exceeded) the 350,000 population AQI reporting limit and there is great value in having the AQI reported for these areas when the population threshold is exceeded versus waiting potentially up to 10 years for a revision to the decennial census. Accordingly, the EPA is finalizing the proposed revision to 40 CFR Part 58, section 58.50 (c) to require the AQI reporting requirements to be based on the latest available census figures.

(8) *Comment:* One state requested additional guidance on the frequency of updating the AQI reporting threshold, and recommended linking the AQI reporting requirement evaluation with the annual air monitoring network plan requirements, and recommended requiring AQI reporting to begin no later than January 1 of the following year.

Response: The EPA notes that the census bureau estimates appear to be released around July 1 of each year which would not provide sufficient time for monitoring agencies to incorporate AQI reporting in their annual plans for that year, which are also due by July 1 each year. The EPA believes that it should be unnecessary for monitoring agencies to wait until the implementation of the following year's annual plan (i.e., approximately 18 months later) to begin AQI reporting. Accordingly, the EPA is not at this time including a specific deadline for commencement of AQI reporting for newly-subject areas in 40 CFR part 58, but will work with agencies to implement additional AQI reporting as needed to ensure that information is being disseminated in a timely fashion.

[102] http://www.census.gov/popest/data/metro/totals/2014/CBSA-EST2014-alldata.html.

B. Secondary O_3 Standard

Public comments on topic areas related to the proposed secondary standard are addressed in the preamble to the final rule and/or in this document. General comments based on relevant factors that either support or oppose revising the current O_3 secondary standard are addressed in section IV.B of the preamble to the final rule and/or in section II.B.1 below. Specific comments on revisions to the secondary standard, including key aspects of the Administrator's rationale for those revisions are addressed in section IV.C of the preamble and/or in section II.B.2 below. Additional comments about the welfare effects evidence and results of the welfare risk and exposure analyses are addressed in sections II.B.3 and II.B.4 below.

1. General Comments on the Need for Revision

Comments on the proposed decision to revise the secondary O_3 standard were divided between those that supported the proposed decision to revise the standard to achieve additional public welfare protection, and those that asserted that the evidence and quantitative information did not support a decision to revise. In addition to the commenters that referenced specific evidence and quantitative information, many commenters simply expressed their views without stating any rationale, others gave general reasons for their views but without reference to specific factual evidence or the rationale presented in the proposal notice as a basis for the Agency's proposed decision.

a. *Support for the Proposed Decision to Revise the Standard*

Many state and local environmental agencies or government bodies, tribal agencies and organizations, and environmental organizations concurred with the EPA's proposed conclusion on the need to revise the current standard, stating that the available scientific information shows that O_3-induced vegetation and ecosystem effects are occurring under air quality conditions allowed by the current standard, and that this provides a strong basis and support for the conclusion that the current secondary standard is not adequate. In support of their view, these commenters generally relied on the body of evidence available for consideration in this review, including evidence assessed previously in the last review. These commenters variously pointed to the information and analyses in the PA and the conclusions and recommendations of CASAC as providing a clear basis for concluding that the current standard does not provide adequate protection of public welfare from O_3-related effects. Many of these commenters generally noted their agreement with the rationale provided in the proposal with regard to the Administrator's proposed conclusion on adequacy of the current standard, and some gave additional emphasis to several aspects of that rationale, including the appropriateness of the EPA's attention to sensitive vegetation and ecosystems in Class I areas and other public lands that provide similar public welfare benefits and of the EPA's reliance on the strong evidence of impacts to tree growth and growth-related effects.

Many commenters agree with EPA's conclusion that the current standard does not provide adequate public welfare protection, variously stating the following.

- The EPA appropriately gives particular attention to sensitive vegetation and ecosystems in Class I areas and other public lands providing similar public welfare benefits.
- The proposal for revision is supported by the current evidence, including studies newly available since the last review. Information described in the proposal indicates harmful effects to public welfare from O_3 allowed by the current standard.
- The Administrator appropriately relies on strong evidence of impacts to tree growth and gives attention to other growth-related effects.

Tribal organizations and agencies additionally noted that many Class I areas, and other similarly protected areas, are of sacred value to tribes or provide treaty-protected benefits to tribes, including the exercise of gathering rights. Tribal groups also noted the presence in these areas of large numbers of culturally important plant species, which they indicated may be impacted by air quality conditions allowed by the current standard. The impacts of concern described include visible foliar injury, loss in forest growth and crop yield loss, which these groups describe as especially concerning when occurring on lands set aside for the benefit of the public or that are of sacred value to tribes or provide treaty-protected benefits to tribes.

These comments received in support of the EPA's conclusion that the standard should be revised are generally addressed in section IV.B.2 of the preamble to the final rule.

b. ***Comments Recommending Retaining the Current Standard***

A number of industries, industry associations, or industry consultants, as well as some state governors, attorneys general and environmental agencies disagreed with the EPA's proposed conclusion on the adequacy of the current standard and recommended against revision. In support of their position, these commenters variously stated the following.

- The available evidence is little changed from that available at the time of the 2008 decision, and the evidence is too uncertain, including with regard to growth-related effects and visible foliar injury, to support revision, and does not demonstrate adverse effects to public welfare for conditions associated with the current standard.
- The EPA analysis of Class I areas did not document adverse effects to public welfare, and it includes monitors that are outside of Class I areas, thereby mischaracterizing air quality in Class I areas.
- While the EPA has indicated a particular focus on Class I areas in its rationale for the need for revision of the current standard, the EPA does not specifically describe the occurrence of adverse effects on sensitive species present in such areas.
- Relative biomass loss (RBL) is not adverse to public welfare and RBL in tree seedlings is not informative of impacts on mature trees, such as commenters state comprise the ecosystems of Class I and other similarly protected areas.
- It is not appropriate to evaluate protection of the current standard under current conditions due to long-range transport of O_3 and precursors;
- Modeling analyses indicate that under conditions where the current standard is met throughout the U.S., the associated W126 index values would all be below the upper

end of the range proposed as providing requisite public welfare protection and many would be below the lower end of 13 ppm-hrs.

- In the WREA air quality scenarios, the sparseness of monitors in the mountain west and in rural areas results in more weight being given to urban monitors and contributes to an overestimation of W126 values (and associated exposure and risks).
- The WREA modeling analyses indicate that any welfare improvements associated with a revised standard would be marginal, in particular compared to the benefits of achieving the current standard.
- The EPA is inappropriately considering visible foliar injury with regard to adversity to public welfare.
- For policy reasons, EPA should not focus on crops, and more research is needed on air quality and crops.

Comments received that recommended retaining the current standard are generally addressed in section IV.B.2 of the preamble to the final rule. Specific aspects to some of these comments are additionally discussed in the sections below.

2. Comments on Aspects of the Proposed Secondary O_3 Standard

a. *Comments on Consideration of Growth-related Effects*

(1) *Comment:* In disagreeing with the EPA's focus on RBL and growth-related effects, one comment expresses the view that RBL is not necessarily adverse to public welfare. In so doing, the comment suggests that dead and slow-growing trees are positive to forest health and the public welfare, citing various considerations, including the role of brush in wildlife habitat and forest structure, municipality costs of tree pruning, interprets some studies to indicate that reduced growth improves survival without detriment to ecosystem productivity, and interprets other studies (e.g., study on root biomass response, and study on regrowth in response to moose browsing) to indicate that ecosystems adjust to O_3 exposure over longer durations and that seedling contribution to net primary productivity is not adversely affected by continued exposure as trees age and reproduce. The comment additionally describes tree seedling RBL as unimportant because the carbon storage ability of tree seedlings is substantially less than that of mature trees, implies that increased carbon fixation at the expense of forest community composition is positive to public welfare, and claims recent USDA forestry data document a lack of effect of O_3 on U.S. forests.

Response: We disagree with this comment with regard to the weight that should be accorded RBL and growth-related effects in this review of the secondary standard. The focus on growth-related effects, and the use of RBL as a surrogate or proxy of the broad array of plant-related effects of potential public welfare significance, is consistent with advice from the CASAC, including their statement that "biomass loss is a scientifically valid surrogate of a variety of adverse effects to public welfare" (Frey, 2014b, p. 10). We describe our consideration of specific aspects of the comment below, based on which we conclude that the comment has not provided information that contradicts the evidence on RBL and the broad

array of vegetation growth-related effects assessed in this review in the ISA, analyzed in the WREA and PA, and summarized in the proposal and preamble to the final rulemaking, and on which the Administrator's conclusions regarding the public welfare impacts of such effects is based.

We disagree with the implication of the comment that O_3 exposures that would result in dead or slow-growing trees are a benefit to the public welfare. While, as the comment notes, smaller trees survive transplantation (and perhaps some other risks) better than larger trees, the timber industry is not, as the comment implies, benefited by air quality that reduces tree growth. Further, while we agree that the growth of invasive species or species that contribute allergens to the atmosphere may have undesirable aspects, we disagree with the implication of the comment that elevated O_3 in ambient air is an appropriate protection mechanism.

The statements made by the commenter that rapid growth of some tree stands results in increased mortality (citing Jimenez and Lugo, 1985; and Coomes and Allen, 2006)[103] appear to be based on consideration of the early development of stands of some trees in which there is strong competition for space and accordingly high rate of mortality of the young trees and which results in a stand dominated by a smaller number of larger trees. We disagree that this occurrence in young stands supports the view implied by the comment that mitigation of O_3-attributable reductions in growth would increase the mortality of young seedlings during this phase of forest stand growth. Rather, to the extent the stands are comprised of O_3-sensitive species, reduction in O_3 exposures would be expected to result in larger surviving trees in the stand.

The study cited by the comment as indicating that tree seedlings adjust to O_3 exposure, such that it does not result in sustained reduction of root mass over multiple years is discussed in the ISA (ISA, sections 9.4.3.1, 9.4.6.3; Pregitzer et al., 2008). The ISA states that the increased fine root production observed in later years of the exposure in the study by Pregitzer (2008) was "due to changes in community composition, such as better survival of the O_3-tolerant aspen genotype, birch, and maple, rather than changes in C allocation at the individual tree level" (ISA, p. 9-45). The moose grazing study cited by the comment did not assess the effect of O_3 on biomass (Persson et al., 2007).[104] Rather it reported on a variation across several species with regard to their regenerative response to years of significant grazing activity by moose. Thus, in some tree species,[105] the grazing transferred carbon up the trophic chain, while not reducing the subsequent primary production. This differs from the effect of O_3 on biomass which reduces primary production in the exposure year.

[103] Although the studies by Jimenez and Lugo (1985) and Coomes and Allen (2007) are not focused on the effects of O_3 and are outside the scope of the air quality criteria, we have provisionally considered them in the context of the comment.
[104] Although the study by Persson et al (2007) is not focused on the effects of O_3 and is outside the scope of the air quality criteria, we have provisionally considered it in the context of the comment.
[105] In other tree species, the regenerative capacity of the tree was reduced by the grazing activity.

In claiming RBL of tree seedlings to be unimportant because the carbon storage ability of tree seedlings is substantially less than that of mature trees, the comment appears to be presuming that O_3 does not also cause RBL in mature trees. As discussed in response to a comment below (and in the preamble to the final rule), the evidence does not support the conclusion that the growth of mature trees is unaffected by O_3. Rather, as discussed in other responses in the evidence indicates that in some situations, mature trees may be more affected than seedlings. Further, tree seedlings are not, as the comment implies, unimportant. Rather, they are the future of the forest, providing for continued productivity after the loss of the mature, elderly trees. For example, following a disturbance affecting the established trees, seedlings affected by O_3 exposure can slow down recovery of the ecosystem net primary productivity.

In suggesting that altered community composition may not be adverse to public welfare if the change in composition does not reduce net primary productivity, the comment is not considering the full array of O_3 effects at the species and ecosystem level. Net primary productivity, while important to the public welfare, is one of multiple ecosystem services of public welfare importance. Community composition is also important (e.g., "[b]iodiversity is a supporting service that is increasingly recognized to sustain many of the goods and services that humans enjoy from ecosystems" [ISA, p. 9-37]). And, the studies cited by the comment[106] as evidence that O_3 may not affect net primary productivity generally reported shifts in the presence or abundance of specific genotypes or species. These shifts in species or genotype abundance and the loss of species or genotypes represent changes in community composition and biodiversity that have potential consequences for ecosystem services of public welfare importance.

Lastly, we disagree with the comment's interpretation of the USDA report describing the state of forests in the U.S.[107] and do not interpret the report's positive findings regarding U.S. forests to mean that O_3 does not cause effects on forests. Evidence in this and past reviews clearly demonstrates the effects of O_3 on forests. Additionally, the Forest Health Monitoring O_3 bioindicator response data, referenced in the comment (and in the cited USDA report) as not being linked to a specific tree health problem or regional decline, are data documenting the occurrence of visible foliar injury in U.S. forests. As noted in the ISA, the proposal and the preamble to the final rule, visible foliar injury "is not always a reliable indicator of other negative effects on vegetation" (ISA, p. 9-39). Thus, the statement in the USDA report regarding these data that is cited by the comment about a lack of evidence linking the FHM visible foliar injury data to a specific

[106] The cited studies have been reviewed in the current or prior O_3 NAAQS reviews or are extending research which was reviewed in the current or prior reviews (Heagle et al., 1991; Moran and Kubiske, 2013; the WI FACE aspen research).

[107] Although the USDA report (*National Report on Sustainable Forests*, 2010) is not focused on the effects of O_3 and is outside the scope of the air quality criteria, we have provisionally considered it in the context of the comment.

tree health problem or regional decline is consistent with the EPA's finding in this review.

(2) *Comment:* A comment states that if EPA is to continue to use RBL in judging adverse effects of O_3 and appropriate protection targets for the secondary standard, the EPA should focus on a much higher percent RBL, suggesting 10%, and only rely on estimates for aspen and ponderosa pine, the two of the 11 species for which established robust E-R functions are available that the commenters consider pertinent to Class I and other similarly protected areas. In support of this view, the commenters disagree with the proposal's consideration of other species for which established robust E-R functions exist as they consider them to occur in too few areas. The commenters further make a number of claims with regard to aspen and ponderosa pine, which in their view raise uncertainty regarding the RBL in Class I areas and leads the comment to suggest a target of 10% RBL.

Response: We disagree with the comment. While we agree that there are uncertainties with regard to the precise magnitude of RBL that would occur in forests in the natural environment, and we disagree that the evidence indicates such uncertainty to contribute to a downward bias in RBL or that a higher RBL should be considered acceptable.[108] As noted by the CASAC, "there is quite a lot of certainty in estimates of biomass loss for forest tree seedling species … for which E-R functions have been developed" (Frey, 2014b). Additionally, we note that the use of RBL in the Administrator's decisions on the secondary standard goes beyond being a predictor of a quantitative reduction in growth of some tree seedlings. Rather, RBL is being used as a surrogate or proxy of the broad array of plant-related effects of potential public welfare significance, consistent with the CASAC advice. Further, in consideration of various uncertainties, such as the relatively greater uncertainty in the functions for species for which fewer studies are available, the EPA focused in the proposal on the median E-R function across the 11 species, rather than a single function for a particular species that might be much more (or less) sensitive than the median. Accordingly, the Administrator's consideration of RBL in this manner has been on the median estimate of RBL, derived from the estimates across the 11 established, robust E-R functions. Contrary to the comment's statement that these species occur in too few areas, the studied species include both deciduous and coniferous tree species with a wide range of sensitivities and species native to every region across the U.S. and in most cases are resident across multiple states and NOAA climatic regions (WREA, Appendix 6A). Thus, the EPA has concluded that all 11 species, as represented by the median, are relevant to informing the identification of a

[108] The commenters provide no evidence for their view that aspen has a "naturally low rate" of seedling growth in Class I areas or that such an occurrence reduces the probability of "extensive" aspen seedling RBL in these areas, or that having wide adaptability and suitability for a range wide of sites or being browsed by elk would be expected to reduce or negate O_3-attributable RBL and associated productivity. They also provide no evidence for their view that any RBL in ponderosa pine seedlings is in response to drought and not O_3.

secondary standard providing protection in these areas, as discussed in section II.B.2 of the preamble to the final rule.

While the CASAC stated that there is "considerable uncertainty in extrapolating from the [studied] forest tree species to all forest tree species in the U.S.," it additionally expressed the view that it should be anticipated that there are highly sensitive vegetation species for which we do not have E-R functions and others that are insensitive. In so doing, the CASAC stated that it "should not be assumed that species of unknown sensitivity are tolerant to ozone" and "[i]t is more appropriate to assume that the sensitivity of species without E-R functions might be similar to the range of sensitivity for those species with E-R functions" (Frey, 2014b, p. 11). Accordingly, we conclude that we have appropriately considered the strength of the scientific evidence and the associated uncertainties in considering revision of the secondary standard, and we find no evidence-based support provided with the comment for the view that identification of an appropriate level of protection to be afforded by the secondary standard should focus on exposure conditions associated with a 10% RBL (or higher) for two species, rather than a RBL estimate below 6%, as the median estimate across the 11 established, robust E-R functions, consistent with recommendations from the CASAC.

(3) *Comment:* Some commenters state that it is inappropriate to consider RBL estimates associated with E-R functions for tree seedlings in drawing conclusions regarding impacts in Class I and similarly protected areas, stating that the E-R functions overestimate responses of mature forests. In support of their position, some commenters take issue with the EPA's interpretation of the current evidence, including the conclusion from the ISA analysis of the Aspen free-air carbon-dioxide and ozone enrichment (FACE) experiment data that "the function based on one year of growth was applicable to subsequent years" (ISA, p. 9-135; PA, p. 5-16), and claim that mature canopy trees likely have greater detoxification ability and as a result, experience reduced O_3 effects compared with tree seedlings.

- Commenters state that findings from Kubiske et al. (2007) and King et al. (2005) for monospecies and mixed-species exposures refute the ISA conclusions, and that the ISA analysis based on aspen response in King et al. (2005) does not reduce uncertainties for subsequent growth stages of multiple tree species. Commenters cite Kubiske et al. (2007) as reporting no significant effects of O_3 or of O_3 with CO_2 exposures on height or diameter after 7-year exposures of mixed species communities, and cite King et al. (2005) as reporting that biomass in the aspen monoculture stand was reduced only in the first two to three years of the seven years studied and as concluding that "[m]onospecific responses to O_3 are not simply additive."
- Commenters claim that mature canopy trees likely have greater detoxification ability, stating that mature canopy trees have increased photosynthesis rates and such increased rates can increase anti-oxidant abilities which influences

the effect of O3 exposure. In support of this claim, the commenters cite a study by Fredericksen et al. (1996).

Response: In section IV.B.2 of the preamble to the final rule, the EPA has addressed the overarching comment on uncertainty related to interpretation of RBL estimates (based on tree seedling studies) with regard to older tree lifestages. We recognize the generally more limited availability of evidence for mature trees as compared to that for tree seedlings and the associated uncertainty for older lifestages and that there is variation in tree species biomass response across different studies, across different genotypes, and whether exposure is for monospecies and mixed-species stand exposures,[109] as well as across species. The focus in the proposal, as well as in the final rule, has, however, been on the relationship between O_3 exposure and RBL for the median studied species, for the reasons described in the proposal and the preamble to the final rule. That focus is consistent with comments from the CASAC. We address some additional specific aspects of the comments here.

We disagree that the two studies cited by the commenters (Kubiske et al., 2007; King et al., 2005) contradict the ISA conclusion that the Aspen FACE results have reduced uncertainty associated with the application of the tree seedling E-R functions to predict response in subsequent years. The ISA conclusion is based on the comparison of the above-ground aspen biomass observed for the same trees in six years of the Aspen FACE O_3 exposure experiment with biomass predicted for those years by the median composite function based on the EPA's National Health and Environmental Effects Research Laboratory – Western Ecology Division (NHEERL-WED) 11 aspen six-year studies (ISA, section 9.6.3.2; King et al., 2005). There was "very close" agreement leading the ISA to conclude that the methodology used for obtaining the median composite function is "capable of deriving a predictive model despite potential confounders, and despite the added measurement error that is expected from calculating biomass using allometric equations" (ISA, p. 9-135). Thus, the ISA conclusion that "the function based on one year of growth was shown to be applicable to subsequent years" (ISA, p. 9-135). Contrary to the implication of the comment, the ISA does not state that the aspen E-R function from monospecies exposures could represent aspen response in mixed-species stands. In fact, the ISA recognizes that O_3 affects competition in mixed-species stands, thus leading to differing response for the same species in mixed stands compared to monospecies stands (ISA, section 9.4.7.1). The CASAC has additionally stated that "competition among species with different sensitivity [in mixed-species forest stands] will reduce overall stand growth losses, but also exacerbate effects on sensitive species" (Frey, 2014b, p. 11). Such effects in mixed-stands lead to alteration of terrestrial community composition, an

[109] Contrary to the implication of the comment, the ISA does not state that the aspen E-R function from monospecies exposures could represent aspen response in mixed-species stands. In fact, the ISA recognizes that O_3 affects competition in mixed-species stands, thus leading to differing response for the same species in mixed stands compared to monospecies stands (ISA, section 9.4.7).

ecosystem services effect recognized by the EPA in this review (ISA, sections 9.4.1.1 and 9.4.7).

Further, the commenters' statement that King et al. (2005) reported biomass in the monospecies culture to be reduced only in the first 2-3 years of the study is not supported by that publication. Figure 2 of the paper shows that for each of the years of treatment (and not only the first 2-3 years), the biomass of the O_3 treatment trees in the monoculture was more than 20% reduced compared to controls, with the percentage reduction being slightly smaller in the first two to three years of study (King et al., 2005). Additionally, the commenters' selective quoting from King et al. (2005) regarding additivity erroneously implies that the authors' statement is with regard to additivity across years. However, the statement instead refers to additivity of response across species and appears to concern the study authors' conclusion that results from monospecies exposures of two different species would not predict the responses, in additive manner, of the two species in a mixed-species exposure (King et al., 2005, p. 632). Preceding the quoted statement, the authors observed that in some studies a monospecies stand had shown little biomass response to O_3 treatment, while the same species when grown in a stand with a second less sensitive species exhibited significant O_3-related biomass reduction (King et al., 2005). The authors' statement was not in reference to additivity across subsequent years in a multi-year exposure, and accordingly does not provide support for the commenters' view regarding reduced RBL in older lifestages. Additionally, with regard to the study by Kubiske et al. (2007), we disagree that the mixed-species O3 exposures did not show reduced growth. In both mixed stands, both tree diameter and volume of aspen was reduced, as was maple. Further, it can be seen that in all but one of the different aspen clones, the O_3 treatment resulted in reduced height and diameter, with many of the differences being statistically significant (Kubiske et al., 2007).

With regard to the comment's claim that mature, overstory trees have greater detoxification ability and, as a result, experience reduced O_3 effects compared with tree seedlings, the EPA agrees that light is a critical factor in plant photosynthesis and that the unshaded portions of canopy trees may have faster rates of photosynthesis than the shaded portions and than shaded, understory trees of the same species and lifestage. We disagree, however, with the implication of the comment that there is evidence showing that mature canopy trees have greater detoxification ability and as a result, experience reduced O_3 effects compared with tree seedlings. The study by Fredericksen et al. (1996), cited by the comment, did not measure anti-oxidant levels in the black cherry leaves and did not compare the growth effects of O_3 between mature black cherry trees and seedlings. The lack of visible foliar injury symptoms in high light leaves of black cherry does not demonstrate that those leaves were not affected by ozone as "the lack of visible injury does not always indicate a lack of … non-visible O_3 effects" (ISA, p. 9-39). Further, a single study of individual leaves in a single stand of a single species does not provide the basis for such a general statement about all canopy or mature trees. Even if the growth rate of understory tree seedlings was lower than that of mature canopy trees of the same species, the commenters have not provided

evidence that the O_3-related RBL (which is a percentage reduction in growth rate) would differ. Further, we note that the studies on which the E-R functions are based were OTC field studies in which the plants were not shaded. So to the extent that anti-oxidant capability is increased with high light conditions, that increased capability and any associated effects on the plant response to O_3 is captured in the studies and the resultant E-R functions.

We additionally note that among the limited number of studies that have directly compared the O_3 effects on canopy trees to effects on smaller, younger trees, some have reported a greater (not lesser) response in older trees. For example, OTC studies over two seasons found O_3 exposure to result in a greater reduction in photosynthesis in mature red oak trees than in seedlings of that species (2006 AQCD, p. AX9-251), and a meta-analysis newly available in this review found older trees to be more affected by O_3 than younger trees (ISA, p. 9-42; Wittig et al., 2007). Additionally, contrary to the comment's hypothesis that greater light and associated photosynthetic activity reduces the effect of O3, photosynthetic efficiency in Norway spruce was more adversely affected by O_3 in high than in low light (2006 AQCD, p. AX9-108). Thus, we disagree with the view expressed in the comment that the evidence supports a conclusion that the E-R functions from tree seedling studies overestimate RBL in mature forest trees. We additionally disagree with the implication of the comment that tree seedling growth is not relevant to ecosystems in Class I and other similarly protected areas. Such areas contain dynamic ecosystems, in which all lifestages play an integral role, and are also affected by natural disturbances, in which seedlings play an important recovery role.

(4) *Comment:* In support of the view regarding uncertainties associated with the use of E-R functions for RBL derived from tree seedling studies, one comment emphasizes that the functions are based on studies of monocultures and that O_3 effects on growth in mixed species stands, such as may be found in the natural environment differs from that in monocultures, citing two studies in the ISA, King et al. (2005) and Kubiske et al. (2007).

Response: As an initial matter, we note that, as described in section IV.C.3 of the preamble to the final rule, the Administrator has focused on the median species RBL, the metric recommended by CASAC, and has used this metric as a surrogate for the broad array of growth-related effects of O_3, the use recommended by the CASAC. We don't disagree with the comment that growth responses to O_3 in mixed-species forests may differ from those observed in monocultures.[110] However, the evidence from mixed species cultures, including

[110] With regard to our WREA characterization of RBL estimates for multiple species that may be in mixed-species forests, we have recognized a number of sources of uncertainty (WREA, Table 6-27). Differences seen across studies of the same species is one area of uncertainty; the WREA characterization of uncertainty for these estimates indicates that the direction of the influence of this uncertainty on the estimates is unknown. The WREA also notes that the absence of E-R functions for many O_3-sensitive species results in an underestimate of the total tree biomass loss in urban areas and Class I areas in those

the studies cited by the comment, indicates an array of responses that go beyond RBL in individual species. These responses include a reduced competitive capacity of some species in the presence of other species, as in the case of aspen in studies of aspen-birch and aspen-maple communities by Kubiske et al. (2007), and an increased susceptibility to O_3 under interspecific competition as in the study of beech in mixed culture with spruce by Kozovits et al. (2005). Another example in a natural forest is the loss of O_3-sensitive pine species (ponderosa pine and Jeffery pine) in forests in the San Bernardino Mountains, converting the forest composition to predominantly white fir (ISA, p. 9-81). As described in section IV.C.3 of the preamble to the final notice, the Administrator's use of RBL in determining appropriate revisions to the secondary standard, as suggested by the CASAC, is as a surrogate or proxy for the broader array of vegetation-related effects, including alterations in community composition, such as these.

(5) *Comment:* One comment stated that the EPA failed to account for the potential of trees to adapt to O_3 exposure, in that the PA does not mention such a potential and the proposal does not consider it. In support of the view regarding such an adaptation, the comment cites Moran and Kubiske (2013). They additionally cite a study by Nakamura et al. (2011), and claim there to be a finding in the RBL results in later years of the aspen study by King et al. (2005), which they state to be consistent with a hypothesis of O_3 tolerance over extended exposures.

Response: We disagree with the comment that the cited studies provide evidence that individual trees adapt to O_3 exposure and with the implication that changes in community composition that result in a reduced prevalence of more-sensitive species or genotypes is not an ecosystem effect with which the EPA should be concerned in considering the adequacy of the secondary standard. We note that, as described in the ISA, the full body of evidence supports the conclusion of a likely causal relationship between O_3 exposure and the alteration of forest community composition in some ecosystems, and the ISA describes the evidence with regard to changes in intra- and inter-species composition that can result from exposure to elevated O_3 (ISA, section 9.4.7). The PA and proposal summarize these effects. Such changes in intra-and inter-species composition, which are the result of competition between species of differing O_3 sensitivities, result in a changed forest community. For example, the study by Kubiske et al. (2007) reported that elevated O_3 changed intra- and inter-species competition, increasing the rate of conversion from a mixed aspen-birch community to a birch dominated community (ISA, p. 9-43). A later study coauthored by Kubiske and cited by the comment (Moran and Kubiske, 2013) further explores the effect of differing sensitivities among several aspen clones by using five more years of growth and survival data, as well as simulated forest genotypic composition.

analyses (WREA, Table 6-27). We additionally note, however, that for species that grow naturally in monocultures in many areas, such as aspen, monoculture results are particularly informative of their responses in those natural forests.

The study by Moran and Kubiske (2013), cited by the comment, is of forest stands in which species composition has been affected by O_3 such that the presence of more sensitive species or genotypes have been diminished. The next-generation forest community modeled by Moran and Kubiske (2013) was predicted to be somewhat less sensitive to O_3 because of the shift in community composition. However, the predicted decrease in ozone sensitivity was relatively minor: the O_3 effect changed from a -7.8% change in height growth in the first generation to between -6.9% and -6.4% in the modeled second generation.[111]

The findings of relative lesser RBL during later years of the multi-year exposure reported by King et al. (2005) are for a mixed stand of aspen and birch. As discussed in that paper, such responses may be caused by compensatory growth of less-O_3 sensitive species, and may lead to changed community composition. Thus, this study is not demonstrating an adaptation of an individual tree or species. Rather, it is describing how O3 can affect a specific mixed-species forest community under specific conditions.

The study by Nakamura et al. (2011) cited in the comment is not a study on O_3 and does not draw conclusions regarding O_3 effects.[112] Rather, the study by Nakamura et al. (2011) is cited within the study by Moran and Kubiske (2014) in making a point regarding evolution resulting from elevated CO_2 exposure. Accordingly, the study by Nakamura et al. (2011) does not support the comment.

(6) *Comment:* In support of the view that the established E-R functions overstate O_3 effects, some commenters said that the EPA, in characterizing the effect of O_3 on plant growth, failed to consider offsetting effects on plant growth of rising CO_2, variously citing to Karnosky et al. (2003), King et al. (2005) and Temperton et al. (2003). One comment stated that this finding substantially increases the uncertainty associated with the E-R functions for RBL such that it is disingenuous for the ISA to claim that O3 exposure will result in RBL.

Response: Contrary to the comment, the EPA has considered the evidence with regard to O_3 effects under conditions of elevated atmospheric CO_2, including studies cited by the comment.[113] The ISA summarizes findings of the 2006 comprehensive review of the influence of CO_2, among the many factors that

[111] While the study cited by the comment (Moran and Kubiske, 2013) was published after the ISA, the EPA has provisionally considered it and concludes that it does not materially change any of the broad scientific conclusions regarding effects associated with O_3 exposure made in the 2013 O3 ISA and thus does not warrant reopening the air quality criteria review.

[112] Although the study by Nakamura et al. (2011) is not focused on the effects of O_3 and is outside the scope of the air quality criteria, we have provisionally considered it in the context of the comment.

[113] The studies by Karnosky et al. (2003) and King et al. (2005), cited by the comment are both considered in the ISA. The third study (Temperton, et al., 2003), that is referenced by the comment as an example of a study that shows that increased carbon dioxide exposure increases biomass growth even in the presence of O_3, does not, in fact, describe any consideration of O_3. Although this study is not focused on the effects of O_3 and is outside the scope of the air quality criteria, we have provisionally considered it in the context of the comment.

influence or alter plant response to O_3, and also considers more recent studies that have added to the understanding of the role of these interactions in modifying O_3-induced plant responses (ISA, section 9.4.8.4). As described in the ISA, several decades of research has shown that exposure to elevated CO_2 generally increases the growth of plants, effects in contrast to the decrease in photosynthesis and growth observed in many plants exposed to elevated O_3.

Thus, we agree with the comment that exposure to elevated CO_2 concentration in ambient air increases photosynthetic rates and generally increases plant growth (ISA, p. 9-90). We disagree, however, with the view that the evidence is unequivocal with regard to an offsetting effect of rising CO_2 on O_3 effects and that the evidence, including the studies cited by the comment, provide information that is relevant to ambient air in the U.S. today. For example, while studies indicate contrasting effects on plant growth of O_3 and CO_2, the evidence is not clear regarding how forest tree growth or the composition and functioning of forests might be influenced by different combinations of these two gases (ISA, pp. 9-90 to 9-91). Additionally, the CO_2 concentrations studied, are well above those common in today's atmosphere. Rather, they are concentrations estimated to be relevant to the year 2050, well after the next several CAA required O_3 NAAQS reviews. Further, several aspects of the exposure conditions for the two gases are not representative of the relevant circumstances, as noted below.

With regard to the study conditions, we note, as an initial matter, that the study atmospheres for the exposures on which the E-R functions are based include the presence of CO_2 at levels it currently occurs in the atmosphere. In contrast, the studies cited by the comment of O_3 exposures in the presence of elevated CO_2 are of little relevance to current ambient conditions, which are the focus of the NAAQS review, because the levels of elevated CO_2 in many of the studies would not be expected to be experienced in the field for 30 or 40 years, while elevated levels of O_3 can occur presently in several areas of the United States. Further, almost all of the evidence, including the studies cited by the comment, comes from experimentation involving plants subjected to an abrupt step increase to a higher, steady CO_2 concentration. In contrast, the O_3 exposure concentrations usually varied from day to day. In the context of climate change, however, CO_2 levels increase relatively slowly (globally 2 ppm/year) and may change little over several seasons of growth. On the other hand, O_3 presents a fluctuating stressor with considerable hour-to-hour, day-to-day and regional variability. Accordingly, there are difficulties in predicting the likely effects of a gradual CO_2 increase from experiments involving a step increase or those using a range of CO_2 concentrations (ISA, section 9.4.8.4).

In recognizing uncertainty in interpreting these results with regard to responses to future elevated CO_2 conditions, the ISA also notes that O_3 and CO_2 interact with other climatic variables, such as temperature and precipitation. Given the key role played by temperature in regulating physiological processes and modifying plant response to increased CO_2 levels and the knowledge that relatively modest increases in temperature may lead to dramatic consequences in terms of plant

development, studies of CO_2 and O_3 interactions alone may not create a complete understanding of effects on plants under future climate change (ISA, section 9.4.8.4).

In summary, we disagree with the comment that O_3 does not cause RBL under current atmospheric conditions relevant to this O_3 NAAQS review and that the established E-R functions are not appropriate descriptors of this effect. The studies cited by the comment on O_3 and CO_2 co-exposures included CO_2 levels of a magnitude projected by some modeling for the year 2050 (i.e., 35 years into the future). We additionally note that, beyond consideration of growth effects on a single species, one of the studies cited by the comment reports effects of O_3 on species competition which has implications for forest composition (as changes in competition influences community composition), with and without co-exposure to elevated CO_2.

(7) *Comment:* In disagreeing with the EPA's reliance on the established E-R functions, some commenters raised concerns about uncertainties associated with the functions, variously stating that there are insufficient studies supporting these functions, as a whole and/or for some species and that E-R functions are established for only 13 species that occur primarily in the two regions of the U.S.

Response: This comment is addressed this comment in section IV.B.2 of the preamble to the final notice.

(8) *Comment:* With regard to consideration of crop relative yield loss (RYL), one comment stated that various growth factors in crop production make quantification of the yield reduction associated with the current or alternative standards difficult for actual conditions difficult, and associated with related uncertainty. Another comment stated that crop yield loss does not rise to the importance of an adverse public welfare effect under CAA, and it is inappropriate to try to address this effect of O_3 thru the NAAQS given the active management of crops and competing market interests.

Response: As indicated in the preamble to the final rule (sections IV.B.2 and IV.C.3), the EPA recognizes that factors such as those listed by the comment may complicate judgments on the extent to which O_3-related effects on commercially managed vegetation are adverse from a public welfare perspective. Thus, while recognizing the public welfare significance of crop yield effects, Administrator gives greater weight to the CASAC's comments on RBL as a surrogate for an array of growth-related effects, and finds protection of public welfare from crop yield impacts to be a less important consideration in this review for reasons including the extensive management of crop yields and the dynamics of agricultural markets. In so doing, however, she notes that a standard revised to increase protection for forested ecosystems would also be expected to provide some increased protection for agricultural crops. With regard to the adversity to public welfare of crop yield loss, we note that as recognized in section IV.C.3 of the preamble to the final rule, the maintenance of adequate agricultural crop

yields is extremely important to the public welfare. In considering this area of the evidence available in this review, however, the Administrator notes that judgments about the extent to which O_3-related effects on commercially managed vegetation are adverse from a public welfare perspective are particularly difficult to reach, given the extensive management of such vegetation. The Administrator's decision gave less weight to this area of the evidence, noting that a standard revised to increase protection for forested ecosystems would also be expected to provide some increased protection for agricultural crops and other commercial commodities, such as timber (Final Rule, section IV.C.3).

(9) *Comment:* A comment states that EPA considers urban forests as Class I areas for protection but that several factors confound attribution of O_3 damage to urban trees (restricted root zone, poor soil, road salt).

Response: As a point of clarification, Class I areas are defined by section 162 of the CAA to include all international parks, national wilderness areas which exceed 5,000 acres in size, national memorial parks which exceed 5,000 acres in size, and national parks which exceed six thousand acres in size, provided the park or wilderness area was in existence on August 7, 1977. Other areas may also be designated as Class I if designated as Class I consistent with the Act. Contrary to the comment's implication, few if any of these areas are within the boundaries of an urban area. Further, while the EPA agrees that there are a number of potential confounders that would be relevant to a field study investigating the role of O_3 in tree damage or reduced tree growth in urban areas, such studies have not been the basis for the evidence on which the decision in this review is primarily based[114].

b. ***Comments on Consideration of Visible Foliar Injury***

(10) *Comment:* Some comments state that visible foliar injury cannot be reliably evaluated for adversity given a lack of information or that it is not an adverse effect on public welfare that must be addressed through a secondary standard, citing to statements in the proposal and ISA regarding the difficulty in relating it to other vegetation effects such as growth and productivity at the plant or ecosystem level, and noting difficulties in its use for policy and in quantifying benefits. Other comments present the alternate view that the information available in this review and advice from CASAC and the National Park Service (NPS) indicate that a secondary standard limiting cumulative exposures to 7-9 ppm-hrs is needed to protect against visible foliar injury effects that are adverse to public welfare, stating that control to such W126 index levels is needed in order to protect national parks and public recreation lands (e.g., ALA et al.). Specific aspects of the latter comments include the following.

[114] Additionally, the studies on which the 11 established robust E-R functions are based were designed to assess O_3 effects specifically, controlling for any relevant confounders.

- Some commenters contend that contrary to the statement in the proposal that there is "a lack of guidance for federal land managers regarding what spatial scale or degree of severity of visible foliar injury is considered sufficient to trigger protective actions for O_3 sensitive AQRVs" (79 FR 75334), an NPS document (U.S. NPS, 2011), provides such guidance. The commenters further state that this document considers W126 exposures greater than 7 ppm-hrs to represent moderate to major impacts on O_3-sensitive vegetation and that NPS comments to EPA (in this review and the last review) have recommended adoption of a W126-based secondary standard with a level in the range of 7 to 9 ppm-hrs, which the comment states to be based in part on concerns about foliar injury.
- The commenters contend that the EPA wrongly states in the proposal that the CASAC did not provide any guidance on foliar injury benchmarks as they did for biomass loss and crop yields. The comment states that the CASAC frames its overall recommendation of 7-15 ppm-hrs on the basis of all evidence including the foliar injury information, points to the CASAC statement that "a level below 10 ppm-hrs is required to reduce foliar injury" (Frey, 2014b, p. iii) and states that since the CASAC's advice on this point was based on its scientific judgment, EPA must adopt a standard at least as protective as 10 ppm-hrs unless it can articulate a scientific basis for not doing so, which the commenters consider EPA not to have done in the proposal. The comment further states that the CASAC and the PA specifically identified visible foliar injury as an adverse welfare effect and that the EPA offers no reasoned basis for rejecting these conclusions.
- Some commenters state that wetlands "are especially at risk from the stress of foliar damage from ozone," that foliar injury is an indicator of stress and that likely air pollution-caused stresses, which they suggest include acid deposition, nitrogen, insect infestations and drought, must be considered
- In support of their disagreement with EPA's consideration of aspects of the available evidence on O_3-induced visible foliar injury, one group of commenters state that in weighing variability in response to O_3 exposures, EPA should not overlook the positive observations of foliar injury and its relationship with a W126 index level of exposure, citing to a recent study (Kohut et al., 2012) and to the WREA cumulative analysis. They further state that to suggest visible foliar injury is not an adverse effect on welfare would be unlawful and arbitrary. These commenters further describe EPA's consideration of foliar injury in the proposal as contrary to EPA statements in past reviews and object to the Administrator's recognition in the proposal of "significant challenges in judging the extent to which such effects should be considered adverse to the public welfare, in light of the variability and the lack of clear quantitative relationship with other effects on vegetation, as well as the lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this vegetation effect" (79 FR 75349).

- The commenters state that EPA's statements regarding visible foliar injury are similar to EPA's position on visibility in the 2006 rulemaking on the $PM_{2.5}$ NAAQS, which a 2009 court decision found to be inadequate in that rulemaking, and for that reason, the EPA needs to identify a level of protection against foliar injury on which to base a decision on the secondary standard.

Response: The EPA disagrees to some extent with aspects of both sets of comments. As described in the proposal and preamble to the final rule, the EPA recognizes a causal relationship to exist between O_3 concentrations and visible foliar injury in sensitive vegetation. The EPA further has emphasized protection of ecosystems in Class I and other similarly protected areas, such as national parks, as an important consideration in this review of the secondary standard[115] and the EPA recognizes that "[d]epending on the extent and severity, O_3-induced visible foliar injury might be expected to have the potential to impact the public welfare in scenic and/or recreational areas during the growing seasons, particularly in areas with special protection" with "the ecosystem services most likely to be affected [being] cultural services, including aesthetic value and outdoor recreation" (79 FR, 75321), services also emphasized by the latter group of commenters. While the currently available evidence demonstrates that O_3 causes visible foliar injury in sensitive vegetation under certain conditions, we are limited in our ability to describe an exposure-response relationship that could inform characterizations of the extent and severity of impact and associated judgments regarding the public welfare significance, as well as, conversely, the level of protection associated with different O_3 exposure conditions. Accordingly, while recognizing the potential for this effect to affect the public welfare in the context of affecting values pertaining to natural forests, particularly those afforded special government protection, the Administrator recognizes significant challenges in judging the specific extent and severity at which such effects should be considered adverse to public welfare, in light of the variability in the occurrence of visible foliar injury and the lack of clear quantitative relationships with other effects on vegetation, as well as the lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this vegetation effect. Thus, visible foliar injury was not the primary focus in the Administrator's identification of the appropriate revisions to the secondary standard. Accordingly, the Administrator has provided a reasoned basis for her consideration of visible foliar injury and its potential public welfare impacts.

With regard to guidance for federal land managers regarding what spatial scale or degree of severity of visible foliar injury is considered sufficient to trigger

[115] While the EPA notes that increased availability of soil moisture contributes to a predisposing environment for O_3-induced visible foliar injury (ISA, p. 9-39), the available studies do not include comparisons across wetland and non-wetland ecosystems and the ISA has not identified wetlands as an ecosystem type at particular risk of foliar injury.

protective action for O_3 sensitive AQRVs, the 2011 NPS document cited by the commenters does not, as had been indicated by the comment, provide such guidance. Rather than linking action triggers to visible foliar injury spatial extent or severity,[116] the NPS document, in providing approaches for assessing air quality-related impacts of proposed development projects, specifies ranges of cumulative O_3 exposure (in terms of W126 and SUM06 indices) for different types of impacts (negligible or minor, moderate, and major), with a range of W126 $\leq$ 7 ppm-hrs or SUM06 $\leq$ 8 ppm-hrs for negligible or minor impacts (NPS, 2011). As to the comment that the 2011 NPS document describes visible foliar injury as a basis for identifying W126 above 7 ppm-hrs as a moderate to major impact on O_3-sensitive vegetation, we disagree. In focusing on this W126 level, the 2011 NPS document cites a 1996 workshop report (Heck and Cowling, 1997), stating that the workshop participants noted that a W126 range of 7-13 ppm-hrs would be protective for growth effects and "a W126 of 5-9 ppm-hrs, would protect plants in natural ecosystems against foliar injury" (NPS, 2011, p. 14). We note that the workshop report actually provided the foliar injury exposure range in terms of SUM06 (rather than W126), stating "for foliar injury to natural ecosystems – a range of 8 to 12 ppm-hrs," which the 2007 Staff Paper suggested would roughly translate (depending on air quality patterns) to a W126 index range of 5 to 9 ppm-hrs (PA, p. 6-17). Thus, in focusing on a W126 of 7 ppm-hrs and a SUM06 of 8 ppm-hrs as the cumulative exposure breakpoint between the negligible or minor impacts category and the moderate category, if those breakpoints were based on foliar injury impacts, the NPS document would appear to be emphasizing, without explanation, the lower end of the 1996 workshop's SUM06 range and the central point of that range when converted to W126. We additionally note that the 1996 workshop report provides no specific quantitative information and cites no studies as the basis for its recommendation (Heck and Cowling, 1997). As noted in the preamble to the final notice, the EPA suggests that the 1996 workshop range may, at the low end, relate to a benchmark derived for the highly sensitive species, black cherry, for growth effects (Kohut, 2007; Lefohn et al., 1997). This would mean that the basis for the 2011 NPS cumulative O_3 exposure breakpoint identified for the lowest impact categories is not visible foliar injury, in contrast to the latter commenters' suggestion.

With regard to advice from CASAC on visible foliar injury, while we agree with the latter commenters that the CASAC described their basis for the lower end of their recommended W126 index range as including consideration of visible foliar injury (Frey, 2014b, pp. iii, 15), the EPA continues to hold the view that, as the proposal stated, this did not constitute a recommendation similar to the benchmarks the CASAC identified for RBL and RYL. That is, the CASAC did not identify an extent or severity of visible foliar injury as a benchmark for EPA's consideration in judging that effect, as they did by the identification of RBL and RYL benchmarks for effects on tree growth and crop yield (79 FR 75334). Further, while the CASAC letter on the second draft PA stated that the CASAC

[116] Additionally, the 2011 document states that it "does not specify the impact level that might constitute an impairment" (U.S. NPS, 2011).

concurred that visible foliar injury, along with relative biomass loss and crop yield loss, are "appropriate surrogates" for adverse welfare effects, its view with regard to exposure levels associated with protection from this effect appears to be based on interpretation of the WREA cumulative proportion analysis (as evidenced by their statement that "W126 values below 10 ppm-hr [are] required to reduce the number of sites showing visible foliar symptoms" [Frey, 2014b, pp. 10, 14]). Additional description of this WREA analysis and additional observations from the same dataset indicate that foliar injury declines with declining exposures of a magnitude higher than 10 ppm-hrs (final rule, section IV.C.2.b; Smith and Murphy, 2015). This scientific information is provided in the preamble to the final rule in describing why the information available on visible foliar injury in this review does not lead the Administrator to focus on a standard that would limit cumulative seasonal exposures to 10 ppm-hrs or lower. Thus, the EPA does not agree that it has not provided a reasoned basis for its disagreement with the CASAC regarding such conclusions drawn from the WREA cumulative analyses.

Commenters do not provide specific information concerning their view that wetlands are especially at risk from the stress of foliar damage from O_3 or clarification as to the stress-related impacts on wetlands (or public welfare) for which they consider there to be a risk. Nor do they indicate how such a status leads to their conclusion on the need for a standard of 7 ppm-hrs in terms of W126. We note that wetlands are not identified in the ISA as an ecosystem type at particular risk of prevalent or severe visible foliar injury.While soil moisture availability, which would be expected to be appreciable in wetland systems, is recognized to be a factor in conditions predisposing the occurrence of visible foliar injury, the ISA recognizes that the occurrence of visible foliar injury depends both on conditions predisposing to visible foliar injury, which include soil moisture, and on the presence of sensitive plants (ISA, p. 9-39, "visible foliar injury occurs only when sensitive plants are exposed to elevated O_3 concentrations in a predisposing environment"). The commenters provide no information about differences in visible foliar impacts in wetland systems as compared to other ecosystems. With regard to processes such as acid deposition, nitrogen, insect infestations and drought, as identified by the commenters,[117] the EPA has considered the impacts of such stressors on O3-related responses (e.g., ISA, sections 9.4.8; 2006 AQCD, sections AX9.3.3.1, AX9.3.4.2, AX9.3.4.4, AX9.3.6.5). This evidence, however, does not include quantitative information that might inform development of exposure-response relationships for O_3-related effects that reflect such influences, and the commenters do not provide such information.

Contrary to implications of the commenters, the EPA has not ignored positive observations of foliar injury and its relationship with O_3 exposure in this review.

[117] Although the studies cited by the commenters for such stresses (Duarte et al., 2013; Likens and Buso, 2012; Clark et al., 2013; Knight et al., 2013; Anderegg et al., 2013) are not focused on the effects of O_3 and are outside the scope of the air quality criteria, we have provisionally considered them in the context of the comment.

For example, the proposal and final rule explicitly recognize the causal relationship of O_3 in ambient air and the occurrence of visible foliar injury in sensitive vegetation and note the ISA statement that "[e]xperimental evidence has clearly established a consistent association of visible injury with O_3 exposure, with greater exposure often resulting in greater and more prevalent injury" (ISA, p. 9-41). Additionally, however, contrary to the implication of the commenters' statement that the study they cite[118] finds the seasonal W126 index to be a consistent predictor of O_3 foliar injury, this study did not identify greater visible foliar injury prevalence or severity across years with greater exposures. Rather, the study found that W126 index values in each year of the study were above two predetermined levels[119] and also that foliar injury was observed in each year.

We disagree with the comment that consideration of visible foliar injury in this review is inconsistent with its consideration in past reviews. Rather, as was done in past reviews, this review has recognized visible foliar injury as an effect, along with growth effects, that is associated with O_3 in ambient air, and appropriate to consider in reviewing the level of protection provided by the secondary standard. Also as was the case in past reviews, this review did not relate levels of protection for visible foliar injury with alternative levels for the standard, or describe the level of protection from this effect afforded by the revised standard. Further, contrary to the suggestion of this comment that EPA's views of visible foliar injury in the current review are inconsistent with those with regard to the 2010 proposed reconsideration, descriptions of the Administrator's conclusions in the two notices are not dissimilar in their consideration of this effect. Both recognize uncertainties that affect our characterization abilities for this effect.[120] In this case of a revised standard, the Administrator further "notes that the evidence is not conducive to use for identification of a specific quantitative public welfare protection objective, due to uncertainties and complexities" and "concludes that her judgments above, reached with a focus on RBL estimates, would also be

[118] The EPA has provisionally considered the study cited by the commenter (Kohut, 2012, listed in Appendix B) and concludes that it does not materially change any of the broad scientific conclusions regarding effects associated with O_3 exposure made in the 2013 O_3 ISA and thus does not warrant reopening the air quality criteria review.

[119] Two predetermined thresholds were identified for W126 index, one from a paper by Lefohn et al. (1997) that was related to a magnitude of predicted RBL (rather than some relationship with visible foliar injury), and a second based on the range of W126 secondary standard levels proposed by EPA in 2010 (based on recommendations related to a combination of effects on vegetation).

[120] For example, the 2010 proposal which proposed a range of levels for a revised secondary standard stated that "[w]hile the Administrator acknowledges that growth effects and visible foliar injury can still occur in sensitive species at levels below the upper bound of the proposed range, the Administrator also recognizes that some significant uncertainties remain regarding the risk of these effects … For example, the Administrator concludes that remaining uncertainties make it difficult to judge the point at which visible foliar injury becomes adverse to the public welfare in various types of specially protected areas" (75 FR 3025). Similarly, the final rule in this review states that "the Administrator takes note of the current lack of robust exposure-response functions that would allow prediction of visible foliar injury severity and incidence under varying air quality and environmental conditions" (preamble to final rule, section IV.C.3).

expected to provide an additional desirable degree of protection against visible foliar injury in sensitive vegetation" (preamble to final rule, section IV.C.3). She, "[a]ccordingly … considers a conclusion on the appropriateness of selecting a standard that will generally limit cumulative exposures above 17 ppm-hrs to be additionally supported by evidence for visible foliar injury, while not based on specific consideration of this effect" (preamble to the final rule, section IV.C.3). Moreover, we note that in this review the Agency's consideration of the potential adversity of various O_3-related responses is based on an updated body of scientific evidence, updated quantitative analyses, and updated CASAC advice. The Administrator fully considered all of this updated information and staff conclusions, as well as CASAC advice, in making judgments about the potential adversity of O_3-related effects.

We additionally disagree with commenters' view of a similarity between EPA's consideration of visibility effects in the 2006 review of the secondary standard for $PM_{2.5}$ and EPA's consideration of visible foliar injury in this review of the secondary standard for O_3. In the PM review, the evidence included "direct, quantitative relationships between PM in ambient air and light extinction, and thus visibility impairment," in addition to information on impacts of urban visibility impairment on public welfare (based in part on valuation studies of benefits associated with improvements in visibility and in part on recognition of a number of programs, standards and planning efforts that illustrate the value that the public places on improved visibility), and information on approaches to evaluating public perceptions and attitudes about visibility impairment (71 FR 61203). With regard to visible foliar injury, however, there remains a lack of robust exposure-response functions that would allow prediction of visible foliar injury severity and incidence under varying air quality and environmental conditions, and as noted in the ISA, "visible foliar injury is not always a reliable indicator of other negative effects on vegetation." Previous AQCDs have also noted "the difficulty in relating visible foliar injury symptoms to other vegetation effects such as individual plant growth, stand growth, or ecosystem characteristics" (ISA, section 9.4.2). As noted in the proposal and final rule, there is a lack of established criteria or objectives that might inform consideration of potential public welfare impacts related to this vegetation effect. However, even assuming the Administrator did reach conclusions as to precise degrees of visible foliar injury that are adverse to public welfare, the scientific evidence to link such degrees of injury with the elements of a secondary standard is lacking. Thus, the Administrator considered the potential for visible foliar injury under a revised standard, but did not attempt to set a standard based specifically on visible foliar injury.

For these reasons, we disagree with latter commenters' view that the evidence bases for visibility-related effects of PM on the public welfare and O_3-attributable visible foliar injury-related effects on the public welfare provide comparable support for identifying a level of protection appropriate to be afforded by the secondary standards for these two pollutants. Thus, while we disagree with the industry commenters' view that protection against visible foliar injury is not

appropriately considered in the context of reviewing the secondary standard, we have concluded that the information available for this effect and its potential impacts on public welfare is sorely limited, leading the Administrator to not give a primary focus to this effect in identifying the appropriate revisions to the standard, as described in section IV.C.3 of the preamble to the final rule.

In addition, we disagree with the latter commenters' assertion that the decision on the secondary standard for O_3 in this final rule is comparable to the decision on the $PM_{2.5}$ secondary standard in the 2006, which was found inadequate in *American Farm Bur. Fd. v. EPA*, 559 F.3d 512, 530 (D.C. Cir. 2009). In that case, the court found the EPA's determination that visibility protection would be afforded by a secondary standard set equal to the primary standard inadequate on the basis that the EPA had not adequately described the Administrator's objectives for visibility-related public welfare protection under the standard (*American Farm Bur. Fd. v. EPA*, 559 F.3d 512, 530 [D.C. Cir. 2009]). In this review, as explained in detail in the preamble to the final rule (section IV.C.3), the Administrator has identified a desired level of welfare protection, and explained the basis for that protection (including how visible foliar injury was taken into consideration, and the reasons therefor) and how that protection is provided by the revised secondary standard.

In summary, the EPA disagrees with the latter commenters that the NPS document and the WREA analyses support their view that a focus on 7 to 9 ppm-hrs is needed to address visible foliar injury effects of O_3 on sensitive vegetation. As described in the preamble to the final rule, the evidence for visible foliar injury at FIA/FHM biosites across the U.S., including in national parks, indicates that reductions in O_3 across cumulative exposure levels extending at the high end well above 20 ppm-hrs, down past and including 17 ppm-hrs, are associated with a lower proportion of sites with injury and a lower severity of injury. While the Administrator notes that the evidence is not conducive to use for identification of a specific quantitative public welfare protection objective related to visible foliar injury, she concludes that the revised standard is expected to provide an additional desirable degree of protection against visible foliar injury in sensitive vegetation.

(11) *Comment:* Some industry commenters also state that the WREA foliar injury W126-based benchmark of 10.46 ppm-hrs is an unsuitable metric for judging whether the secondary standard should be revised.

Response: The EPA agrees with the commenters that this benchmark is not a suitable basis for such judgments and EPA is not using it in that way in this NAAQS review. This benchmark was derived from the WREA cumulative proportion analysis of the complete biosite dataset, as described in the WREA (section 7.3.1.5 and Appendix 7A). It was used for the "base scenario" in the WREA national park screening-level assessment, not to indicate a threshold, but to provide an indication of the risk of foliar injury based on analysis of the USFS FHM/FIA data. As described further in section IV.C.2 of the final rule, additional observations from the WREA dataset, as well as the full body of currently

available information, show that the occurrence of visible foliar injury varies across a larger range of W126 exposure index values (Smith and Murphy, 2015). Further, a number of associated complexities and limitations (described in sections IV.D.1 and IV.D.3 of the proposal), pose challenges to judgments regarding adversity to public welfare based on the occurrence of this effect.

c. ***Comments on Other Welfare Effects***

(12) *Comment:* Some commenters stated that the EPA should give emphasis to the climate change-related effects of O_3 in considering revisions to the secondary standard, while others expressed the contrary view that the information on O_3 and climate was too uncertain to be used as a basis for revising the standard. Among the former, some commenters stated that O_3 is a potent greenhouse gas (the third strongest) and EPA should consider the direct as well as indirect climate impacts of O_3 in setting a level for the secondary standard. Some of these commenters objected to EPA's characterization of welfare impacts of radiative forcing from O_3 in the final rule, stating that O_3 has a strong warming impact, especially in the Northern mid-latitudes and the Arctic, and that it has been established that O_3 can be reduced through decreases in various precursor chemicals. Another comment indicated that a W126-based standard of 7 ppm-hrs was needed to address a "climate-change feedback loop" (involving increased heat, drought and elevated O_3 contributions to forest fires which contribute to increases in O_3) which is described as having been created in the Mountain West and for which the comment ascribes a key role to O_3, stating that a secondary standard of 7 ppm-hours with narrowly drawn exceptions for extraordinary events would do the most to dampen this feedback loop.

Response: Although the EPA has determined causal and likely causal relationships to exist between changes in tropospheric O_3 concentrations and radiative forcing and effects on climate as quantified through surface temperature response, respectively (ISA, section 10.5.1), quantitative uncertainties associated with relating the magnitude of such effects with differing tropospheric O_3 conditions are such that the Administrator has not made these a primary focus in her decision on the secondary standard. As summarized in sections IV.C.2 and IV.C.3 of the preamble to the final rule, she judges the quantification uncertainties to be too great to support identification of a standard specific to such effects. Rather, given the availability of well-established exposure-response functions for tree growth and the concurrence of CASAC with use of these functions as a surrogate or proxy for the array of related effects, including ecosystem services such as carbon storage, the decision on the revised secondary standard has focused on the use of RBL in this manner, with the Administrator's decision on the revised secondary standard based primarily on consideration of O_3 effects on plant growth. In reaching her decision in this way, she has focused on setting a standard based on providing protection against vegetation-related effects which would be expected to also have positive implications for climate change protection through the protection of ecosystem carbon storage (preamble to the final rule, section IV.C.3).

With regard to the specific comments on the direct effects of O_3 on climate, we concur that O_3 ranks third in importance as a greenhouse gas and radiative forcing agent, after carbon dioxide and methane (ISA, p. 10-8), and have concluded there is a causal relationship between O_3 and radiative forcing (ISA, p. 10-30). As O_3 is only one of many pollutants contributing to warming, however, any welfare impacts resulting from increases in temperature cannot be directly linked to tropospheric O_3 alone. Additionally, estimates of tropospheric O_3 radiative forcing are relatively more uncertain than the other long-lived greenhouse gases (ISA, p. 10-8, Figure 10-3). The ways in which O_3 differs from these other gases contribute to this uncertainty.[121] These differences likely contribute to the fact that "few studies" have "calculated the climate response to changes in tropospheric O_3 concentrations alone in the future atmosphere" (ISA, p. 10-24). The limited availability of models and studies evaluating the wide range of potential downstream effects on ecosystems resulting from O_3-induced climate change limit the ability to quantitatively evaluate the effects of O_3-induced climate change, particularly at the level of detail required to assess the full range of potential climate change impacts of O_3 in Class I areas. Thus, limitations in currently available studies and models have precluded the EPA from assessing all downstream impacts on ecosystems resulting from O_3 contributions to climate change, including those potentially impacting Class I areas and other protected areas in the U.S., including those in the Arctic.

With regard to the comments emphasizing feedbacks, the EPA agrees that feedbacks from both the response of climate to radiative forcing and downstream effects can affect the abundance of O_3 and O_3 precursors in ambient air (ISA, Figure 10-2 and section 10.3.2.4). We are not, however, aware of evidence, and the comments did not provide any, that indicates the specific impacts of different O_3 W126 index values on radiative forcing, climate, or climate feedbacks. As recognized in the preamble to the final rule, there are "large uncertainties in the magnitude of the radiative forcing estimate attributed to tropospheric O_3" (ISA, p. 2-47). There are multiple ways in which changes in concentrations of O_3 precursors can affect the radiative balance of the atmosphere and some of these ways are competing (ISA, section 10.3.4), and as noted above, "few studies" have "calculated the climate response to changes in tropospheric O_3 concentrations alone in the future atmosphere" (ISA, p. 10-24). The limited availability of models and studies evaluating the wide range of potential downstream effects on

[121] The important ways in which tropospheric O_3 differs from other greenhouse gases (ISA, section 10.3.2.4) include the following: (a) it is not emitted directly, but is produced through photochemical oxidation of CO, CH_4, and nonmethane VOCs in the presence of NO_X; (b) it is also supplied by vertical transport from the stratosphere; (c) the lifetime of O_3 in the troposphere is typically a few weeks, resulting in an inhomogeneous distribution that varies seasonally, while the distribution of the long-lived greenhouse gases like CO_2 and CH_4 are much more uniform (d) the longwave radiative forcing by O_3 is mainly due to absorption in the 9.6 μm window, where absorption by water vapor is weak, making it less sensitive to local humidity than the radiative forcing by CO_2 or CH_4, for which there is much more overlap with the water absorption bands; (e) unlike other major greenhouse gases, O_3 absorbs in the shortwave as well as the longwave part of the spectrum (ISA, p. 10-7).

ecosystems resulting from O_3-induced climate change limit our ability to evaluate the "climate-change feedback loop" referenced by the comment or any other downstream effect of O_3-induced climate change. Additionally, these models do not focus on cumulative exposures in quantifying O_3 inputs. While changes in climate can affect O_3 concentrations, and changes in tropospheric O_3 concentrations affect radiative forcing, the evidence does not provide a basis for reaching conclusions regarding a relative difference between different alternative secondary standards based on this environmental effect. We also agree that O_3 affects plant growth and carbon storage in forests, as discussed in the ISA, PA, proposal and preamble to the final notice. Based on the current scientific evidence, however, we disagree with the comment that the role of O_3 in such climate feedbacks supports a specific level for the secondary standard or a cumulative exposure level to target.

d. *Comments on Use of W126-based Metric in Evaluating Vegetation Effects and Public Welfare Protection*

(13) *Comment:* Some commenters that supported use of a W126-based metric as the form for the revised standard, disagreed with EPA's derivation of that metric by summing only during the daylight hours of 8 a.m. to 8 p.m. and across a 3-month period, expressing the view that concentrations over all 24 hours in a day and all days in a year should be summed, and generally indicating that O_3 exposure can cause vegetation damage outside of the windows identified by EPA and concluding that without inclusion of the longer summation period, the pertinent exposure would be underestimated. Without such a change, these commenters state that EPA should consider a lower value of a W126-based standard. In support of summing over a 24-hour daily period, some specific comments raised are summarized here: (1) commenters stated that plants are photosynthetically active beyond the hours of 8:00 a.m. and 8:00 p.m., citing three studies from the ISA; (2) commenters stated that sensitive species have been documented to exhibit nocturnal conductance, listing such species that they state are also present in national parks; (3) commenters provided estimates for 28 national parks of the average percent by which a 12-hour monthly W126 index value underestimated a 24-hour monthly W126 index value, based on 18 months from April to September from 2006 to 2008, and state that overnight or early morning exposures can be high in mountain locations ; (4) commenters stated that there is evidence of O_3 uptake and injury from nighttime exposures, citing three studies from the ISA; (5) commenters stated that an anti-oxidant defensive compound, ascorbate is produced largely in daytime and depleted in late afternoon leaving plants less protected from nighttime and early morning elevated O_3 concentrations, citing to two studies in the ISA.

Response: Although this comment was made in the context of recommendations for a revised secondary standard with a W126 form, the Administrator's decision is not to revise the form of the secondary standard. Accordingly, we have considered the comment in the context of the EPA's use of W126 index values in consideration of the relationships between cumulative exposures and tree seedling

RBL for purposes of describing the objectives for the revised standard with regard to limitations on such exposures, and additionally note that the air quality analyses that characterized W126 exposures associated with alternative levels for the 4th high metric, focused on a W126 metric using the same summation periods.[122]

As described in the preamble to the final notice, in our use of the W126 index, we have relied on the established robust E-R functions for 11 tree species, the studies for which were generally of 3-month duration and the controlled exposures occurred during the daylight period. Since the studies on which the E-R functions are based are generally not longer than three months in duration, they cannot inform our understanding of potential impacts of exposures over longer periods. Further, no E-R functions have been established for a W126 index derived by cumulating 24 hours per day across a full year. Rather, the established E-R functions are based on the cumulation of O_3 exposure across the hours of 8 a.m. through 8 p.m. and three consecutive months.

With regard to the comments regarding photosynthetic activity, conductance, O_3 uptake and injury during nighttime hours, while the cited studies variously suggested such processes may be involved in effects related to cumulative O_3 concentrations that may occur across hours during the darkness as well as the daylight, the studies on which the E-R functions are based will have inherently captured these processes. The E-R functions themselves simply attribute the full resultant response to the W126 index derived through summing across only the 12-hour period. Similarly, to the extent that ascorbate is modifying plant response to O_3, that response is captured in the studies on which the E-R functions (which relate daytime O_3 concentrations during the study to the full growth response observed in the study), are based. We additionally agree with commenters that a W126 index derived by summing across 24 hours in a day will be higher than a W126 index derived by summing across 12 hours in a day, and that the difference will vary with meteorological and other factors. Thus, E-R functions derived for a 12-hour W126 index will differ from those for a 24-hour index. The established E-R functions used in this review, with concurrence from the CASAC, are based on summing across the 12 hours of 8 a.m. to 8 p.m.; in its focus on the W126 index, it is this definition with which the CASAC concurred, in light of the currently available information (Frey, 2014b).

With regard to comments concerning nocturnal exposures in mountain locations, we note that the majority of the experiments that support the E-R functions were not performed at high elevation and did not involve peak O_3 concentrations during the night-time hours (or hours outside of the 8 a.m. to 8 p.m. window). Further,

[122] We additionally note that using a W126 index that was derived using 24 hours/day concentrations in the air quality analyses would not be relevant to the W126 index target identified by the Administrator in consideration of the relationships between RBL and W126 index values based on the established E-R functions because the W126 index for the E-R functions is derived through the 12-hour and 3-month summation periods.

while we recognize an array of plant processes that may not occur solely within the summation periods used, we have concluded that there is insufficient evidence available at this time to characterize the potential effects associated with cumulative exposures derived either solely from nighttime exposures or from a combination of daytime and nighttime exposures in order to provide support for derivation and use of an exposure index based on 24-hour O_3 concentrations (e.g., ISA, section 9.5.3.2). We note that while the E-R functions were derived from exposures estimated through summing concentrations for just the hours from 8 a.m. through 8 p.m., they also are based on the quantified growth response to 3 months of 24 hours-per-day exposures in which the controlled exposures occurred during the daylight period, but the nighttime hours, while not a major part of the experimental exposure periods, were not completely without O_3 exposure because there was O_3 in the ambient air.[123] Thus, we have continued to focus on a W126 index that cumulates hourly O_3 concentrations across the 12-hour period from 8 a.m. to 8 p.m. in identifying exposures of concern for vegetation-related effects.

(14) *Comment:* Some comments generally supported EPA's proposed conclusions related to target W126 index exposures for the secondary standard, stating that a W126 index of 13-17 ppm would provide requisite protection, and that support was lacking for a standard intended to control to lower cumulative seasonal exposures. Some comments further stated that a target W126 index value as low as 7 ppm-hrs would not be appropriate because the WREA results indicate that timber gains projected for such an exposure level would be essentially undetectable and not significant.

Response: The EPA generally agrees with these commenters that it is not necessary for the revised secondary standard to control cumulative seasonal exposures to W126 index values as low as 7 ppm-hrs across the country. For the reasons described in the preamble to the final notice, the Administrator concluded it is appropriate to identify a standard that would restrict cumulative seasonal exposures to 17 ppm-hrs or lower, in terms of a 3-year W126 index, in nearly all instances (preamble to the final rule, section IV.C.3).

(15) *Comment:* In support of the view that EPA should set a secondary standard with a W126 form and with a level below 15 ppm-hrs, one comment stated that a W126 value below 15 ppm-hrs is needed because O_3 concentrations in much of the northeast U.S. have been below a W126 of 15 ppm-hrs and U.S. NFS-trained observers have routinely observed forest damage (foliar injury) in sensitive tree species in some parts of region and elsewhere in the eastern U.S., citing a study from the ISA (Smith et al., 2003).

Response: Although this comment was made in the context of recommendations for a revised secondary standard with a W126 form, the Administrator's decision

[123] The commenters provided no evidence for the statement that overnight or early morning O3 can be "high" in mountain locations, and the information about diurnal patterns of O3 concentrations assessed in the ISA indicates the highest concentrations to occur during the daylight hours (e.g., ISA, section 3.6.3.2).

is not to revise the form of the secondary standard. Accordingly, we have considered the comment in the context of the Administrator's considerations in her decision on revisions to the standard.

As an initial matter, we note that while the study cited by the comment describes O_3 for the New England study area as ranging down to SUM06 values expected to correspond to W126 index values below 15 ppm-hrs (W126 values are not reported in the study), it also states that SUM06 values ranged as high as 31.86 ppm-hrs (Smith et al., 2003). This magnitude of SUM06 would be expected to be well above 15 ppm-hrs, in terms of a W126 index, based on the relationship to W126 developed in the 2007 Staff Paper, which found a SUM06 of 25 ppm-hrs to be approximately equivalent to 21 ppm-hrs in terms of a 12-hour seasonal W126 index (PA, Appendix 6A; U.S. EPA, 2007, Appendix 7B). Further, the study cited by the comment does not describe a specific quantitative relationship of visible foliar injury occurrence, prevalence or severity with W126 index of O_3 exposures that might inform consideration of effects occurring at alternative magnitudes of cumulative exposure. As noted by the ISA with reference to this study, "the degree and extent of visible foliar injury development varies from year to year and site to site" (ISA, p. 9-38) and "many studies have shown that dry periods in local areas tend to decrease the incidence and severity of O_3-induced visible foliar injury; therefore, the incidence of visible foliar injury is not always higher in years and areas with higher O_3, especially with co-occurring drought" (ISA, p. 9-39). Thus, the cited study does not support the comment's implication that a standard controlling air quality to cumulative exposures below 15 ppm-hrs, in terms of a W126 index, will achieve conditions in which visible foliar does not occur. Nor does the comment describe a prevalence or severity of visible foliar injury that should be considered to be adverse to public welfare, or criteria or objectives that might inform such consideration. Thus, the information cited by the comment does not appear to provide information additional to that which was available to and considered by the EPA at the time of proposal, which stated that "the Administrator takes note of the complexities and limitations in the evidence base regarding characterizing air quality conditions with respect to the magnitude and extent of risk for visible foliar injury" and "recognizes the challenges of associated judgments with regard to adversity of such effects to the public welfare" (79 FR 75336). Accordingly, for the reasons described in section IV.C.3 of the preamble to the final rule and in section II.B.2.b of this RTC, the Administrator has not given primary focus to visible foliar injury in identifying the appropriate revisions to the standard, instead primarily basing her decision for a revised standard on her consideration of RBL as a surrogate or proxy for the broad array of vegetation-related effects of potential public welfare significance, that include effects on growth of individual sensitive species and extend to ecosystem-level effects. In so doing, she recognizes that her decision on the revised standard would also be expected to provide an additional desirable degree of protection against visible foliar injury in sensitive vegetation.

(16) *Comment:* In support of their view that the EPA did not adequately consider a secondary standard that would limit cumulative exposures to 7 ppm-hrs in terms

of a W126 index, one group of commenters states that the EPA has no basis for claiming greater uncertainty as to welfare benefits at lower levels, stating that the CASAC cautioned against overstating such uncertainty, indicated there to be "quite a lot of certainty in the estimates of biomass loss for forest tree seedling species for which E-R functions have been developed" (Frey, 2014b, p. 15), and "expressly relied on those estimates to recommend 7 ppm-hrs as the low end of the range" (e.g., ALA et al.). These commenters additionally state that the EPA did not provide adequate explanation in the proposal for departing from CASAC and PA recommendations for 7 ppm-hrs, suggesting that the EPA's consideration of the CASAC recommendation regarding 7 ppm-hrs was not adequate because in their view the EPA interpreted the recommendation to be related to the CASAC's policy judgment, rather than scientific judgment which was the commenters' interpretation.

Response: In considering the CASAC comments regarding the certainty in estimates of biomass loss for forest tree seedling species, we take note of their context, which is in consideration of the certainty of the RBL estimates based on the E-R functions developed from controlled exposure studies. In the proposal and final notice, the EPA's recognition of uncertainty with regard to lower exposure levels is not focused on such RBL estimates themselves. Rather it is focused on the use of estimates as a surrogate or proxy in the context of the judgments required of the Administrator in reaching a decision on a secondary standard that provides the appropriate level of protection. Accordingly, the proposal noted uncertainty "associated with the extent to which estimates of benefits in terms of ecosystem services and reduced effects on vegetation at lower O_3 exposures might be judged significant to the public welfare" (79 FR 75349), and the preamble to the final rule recognized uncertainties "regarding the extent to which associated effects on vegetation at lower O_3 exposures would be adverse to public welfare" (final rule, section IV.C.3). Thus, we have not dismissed the CASAC advice regarding certainty associated with RBL observed in the controlled exposure studies and estimates derived from the associated E-R functions. Rather, the Administrator has made consideration of RBL and the relationships between cumulative O_3 exposures and RBL derived from those studies a central part of her consideration of both the adequacy of the existing (2008) standard and of the appropriate revisions to the standard.

With regard to EPA's consideration in the proposal of the CASAC recommendation regarding 7 ppm-hrs (a recommendation which was also reflected in the PA conclusions), we disagree with several aspects of the commenters' characterization. As an initial matter, we disagree with the implication of the commenters that the CASAC recommendation regarding a W126 index value on which to focus for the revised standard was that EPA should revise the secondary standard to 7 ppm-hrs. The CASAC recommendation was not for EPA to consider only that possibility for a revised secondary standard. Rather, the CASAC recommended that the EPA consider a range of nine alternatives, among which 7 ppm-hrs was one (i.e., "[t]he CASAC recommends that the level associated with this form be within the range of 7 ppm-hrs to 15

ppm-hrs" [Frey, 2014b, p. iii]). Further, while the CASAC clearly described the "range of levels" as based on its "scientific judgment" (Frey, 2014b, p. iii), the letter from the CASAC to the Administrator conveying this recommendation, additionally clearly states that "[t]he CASAC acknowledges that the choice of a level within the range recommended … is a policy judgment under the statutory mandate of the Clean Air Act" (Frey, 2014b, p. iii).

Thus, not only did the CASAC not recommend only one W126 level for the EPA's consideration, but the CASAC recognized that acceptance or rejection of that specific level or any other within the CASAC range was a matter of policy. Even so, the proposal fully considered the entire W126 index range identified by the CASAC and the PA, as described in sections E.2.b and E.3 (79 FR 75347-75349), including "the CASAC policy view regarding protection provided for trees and associated ecosystem services from a W126 index value of 7 ppm-hrs" (79 FR 75349). Additionally, to insure full consideration of this aspect of the CASAC advice in her final decision on the revised standard, the Administrator further solicited comment on W126 index values across the complete CASAC range (including 7 ppm-hrs), both with regard to the appropriate cumulative exposure target for public welfare protection and with regard to the form and level for the revised standard (79 FR 75351). The EPA's consideration of those comments, and of CASAC advice is described in the preamble to the final rule and in this RTC, with the EPA's consideration of the CASAC recommendations for a secondary standard, including the reasons for departures from them, described in detail in section IV.C.3 of the preamble to the final rule, consistent with CAA section 307(d)(3) and 307(d)(6)(A). For example, this section includes discussion of the Administrator's reasons for departing from CASAC advice regarding revision of the form of the secondary standard and with regard to a focus on a 3-year average W126 index for assessing RBL estimates, as well as discussion of the CASAC comments regarding protection associated with W126 index values below 10 ppm-hrs. In summary, the Administrator's reasons for selecting the secondary standard she did, including the scientific considerations and policy judgments supporting that decision, are described in section IV.C.3 of the preamble to the final rule.

(17) *Comment:* Some commenters, who supported a secondary standard with a W126 form, disagreed with the Administrator's decision to focus on a 3-year average W126 exposure index in her decision. One of these comments stated that, although the EPA referenced the PA finding of greater significance for effects associated with multi-year exposures, the PA did not indicate that a multi-year average would provide better protection than a single-year W126 form and that the PA noted that a 3-year average W126 may lead to underestimation of RBL and EPA doesn't explain how a 3-year average addresses annual year effects. Another comment stated that adverse vegetation damage can occur on an annual basis and indicated concern that high O_3 in a single year might not get attention if O_3 was relatively low in adjacent years, citing a study they state to indicate significant year-to-year variations in the extent of observed vegetation damage due to ozone (McLaughlin et al., 2007a).

Response: Although these comments were made in the context of recommendations for a revised secondary standard with a W126 form, the Administrator's decision is not to revise the form of the secondary standard. Accordingly, we have considered the comment in the context of the EPA's use of the W126 metric in consideration of the relationships between cumulative exposures and tree seedling RBL, as a surrogate or proxy for the array of vegetation-related effects on public welfare, for purposes of describing the objectives for the revised standard with regard to limitations on such exposures. While the bulk of the comments related to focus on a 3-year average W126 index are addressed in the preamble to the final rule, we further address here the specific points described above.

Regarding the 1st comment, the EPA considered the issue of a single vs multi-year W126 index from a diverse array of perspectives, summarized E.2.a and E.5 of the proposal and IV.C.2c and IV.C.3 of the preamble to the final rule, including those implied by these comments. These comments do not reflect the full picture of the PA's statements on a 3-year average W126 index. The PA, in summarizing a WREA analysis noted that the RBL estimated from three years of the same W126 will be slightly lower than the RBL estimated from three years of differing W126 index but for which the 3-year average W126 is the same as that in the three years of the first case. In summarizing this PA finding, the proposal additionally states that "the PA notes that this limited analysis does not account for moisture levels, and other environmental factors that could affect plant growth and that vary from year to year" and that "the PA recognizes the importance of considering the extent to which the cumulative effects of different average W126 exposures across the three-year period would be judged adverse" (79 FR 75338). The proposal summary of the PA on this topic went on to recognize that the PA concluded a 3-year form might be "appropriate for a standard intended to achieve the desired level of protection from longer-term effects, including those associated with potential compounding" (79 FR 75339). As noted in section IV.C.3 of the preamble to the final rule, the Administrator took note of the PA considerations on this point and others, the evidence in the ISA, as well as advice from CASAC and recognized the role of her judgment with regard to uncertainties and limitations in the available information. Based on all of this, she concluded it is appropriate to use an index averaged across three years for judging public welfare protection afforded by a revised secondary standard.

With regard to the second comment, the EPA does not disagree with the comment that growth inhibition can result from a year of O_3 exposure. The study cited by the comment, McLaughlin et al. (2007a), reports on growth rates of studied species at two sites over a 3-year period. Rates were reduced during the 2nd year of the period during which O_3 was relatively higher compared to the 1st and 3rd y ears (which were similar to each other). Contrary to the commenter's concern, however, the relatively lower O_3 concentration years did not dilute the high year as the design values (for the 2008 standard which has a 3-year average form), were both in exceedance of the 2008 standard at 92 and 76 ppb (http://www3.epa.gov/airtrends/values_previous.html). Further, we note that

while the growth rate in the 3rd year at the site with the higher O_3 concentrations is still substantially reduced from the 1st year's rate, the growth rate in the 3rd year at the lower concentration site recovered to near that of the 1st year. And, both sites were in exceedance of the 2008 standard in the subsequent 3-year periods also (e.g., 2002-2004, 2003-2005). Thus, while we do not consider this study to necessarily inform consideration of protection afforded by the revised standard of 70 ppb, we find this study to be supportive of the Administrator's judgment that the potential for impacts of concern associated with a single year exposure can be addressed through use of a 3-year average metric with attention to the magnitude of exposure (section IV.C.3 of the preamble to the final rule).

(18) *Comment:* Some commenters stated that the proposal did not adequately explain EPA's divergence from CASAC advice with regard to a use of a single-year W126 index.

Response: Section IV.C.2 of the preamble to the final rule discusses the EPA's consideration of comments in this area and section IV.C.3 describes the Administrator's consideration of CASAC advice in this area.

(19) *Comment:* One comment stated that EPA had based its selection of the W126 exposure index values describing cumulative exposures of importance for public welfare protection by a revised secondary standard on estimated equivalency with the range proposed for the primary standard (65-70 ppb) and that the EPA had failed to "rationally justify" such options for the secondary standard.

Response: The EPA disagrees with this characterization of its consideration of public welfare protection objectives. In the preamble to the final rule, the EPA has described the rationale for the decision on the revised secondary standard, including a detailed explanation of the public welfare protection considerations and judgments associated with that decision.

(20) *Comment:* In expressing disagreement with a secondary standard focused on cumulative exposures in terms of a W126 index value of 17 ppm-hrs, one comment stated that the CASAC has called a W126 index of 17 ppm "unacceptably high" and identified six species that the comment stated are "at risk" at this exposure level. Another comment claimed that in not including 17 ppm-hrs in its recommended range, the CASAC also considered effects at 10 and 7 ppm-hrs that the comment termed "adverse".

Response: As an initial matter, the EPA disagrees with the comment that 17 ppm-hrs was what the CASAC was calling "unacceptably high" (Frey, 2014b, pp. iii, 14). The full context for this phrase on page 14 of the CASAC letter on the second draft PA, is that "[w]e do not consider a value of 17 ppm-hrs from Table 6-1 because … the median species has relative biomass loss of 6.0 percent, which is *{emphasis added}* unacceptably high." Thus, it is the RBL magnitude that the CASAC is judging. Further, the commenter's statement that six species are "at risk" with a cumulative seasonal exposure of 17 ppm-hrs implies that the

comment is interpreting an RBL estimate above 2% to put a species "at risk," although the comment does not describe the impact for which the species are at risk. EPA would agree that these six species (with an RBL above 2% at 17 ppm-hrs) will be at greater risk of growth effects than the other five species for which E-R functions (and associated RBL estimates) are available. For purposes of setting the secondary standard, however, the Administrator has focused on the median species RBL, the metric recommended by CASAC, and has used this metric as a surrogate for the broad array of growth-related effects of O_3, as recommended by CASAC.

The second comment cites page 12 of the CASAC letter, where CASAC concurs with the PA consideration of and conclusion that the current standard is not adequate. In the paragraph mentioning 10 and 7 ppm-hrs, the CASAC does not, as the comment misstates, term effects at these exposures to be "adverse." Rather, the CASAC refers to WREA findings ("effects") for those two exposure levels as augmenting their immediately prior statement that "the correlative similarity between the current standard and a level of the W126 index of 15 ppm-hrs must not be interpreted to mean that just meeting the current standard is equivalent to just meeting a W126 level of 15 ppm-hrs" (Frey, 2014b, p. 12). Thus, we do not conclude their mention of 10 ppm-hrs and 7 ppm-hrs to be central to their conclusion that their range should not include 17 ppm-hrs. The subsequent sentence in the same paragraph appears to then turn away from the conclusion on adequacy of the current standard to consideration of the range that should be considered for a revised standard by stating that the CASAC "concludes that the upper bound of the range … should not exceed 15 ppm-hrs." The next two sentences repeat this sentiment and are followed by the statement that "[f]or example, at 17 ppm-hrs, the median tree species has 6% relative biomass loss" and "[t]hese levels are unacceptably high" (Frey, 2014b, p. 13).

(21) *Comment:* Some state agency commenters in western states indicated that in their states the highest O_3 concentrations during a year have on occasion occurred in the winter months and expressed concern that a W126 standard that might cumulate across the highest three months in the year could inappropriately include winter months. Some of these commenters requested that if the EPA were to adopt a secondary standard with a W126 form, the EPA should include a mechanism that would avoid a focus on the highest 3-month period without attention to whether or not it occurs within the growing season for an area.

Response: Although these comments were made in the context of recommendations for a revised secondary standard with a W126 form, the Administrator's decision is not to revise the form of the secondary standard. So the commenters' concern of judging attainment with the secondary standard in their states based on air quality during winter months is moot. We have, however, considered this issue in the context of the derivation of W126 index values for use in the air quality analyses summarized in section IV.E.4 of the proposal and described in detail in Wells (2014a), with expanded analyses in Wells (2015). In

so doing, we are providing some clarification here of our consideration of exposure periods including winter months.

The derivation of W126 estimates across the U.S. in this context did not include the wintertime 3-month periods that span across two years (November - January and December - February). Further, in not considering a fixed 3-month window, such as June through August, we allowed for the fact that the length and timing of the growing season varies among plants, including tree species, that occur in areas across the U.S. For example, a 1-month growing season in April, as well as, some portion of a 5-month growing season extending from April through August, can be captured by the EPA's focus on the 3-month consecutive period with the highest index value. This provides a greater likelihood that at the national scale the three months with the maximum W126 will coincide with some or all of the same months that plants are susceptible to O_3 exposure since, in most cases, both elevated O_3 concentrations and biological activity occur during the warm season.

The EPA recognizes that "to be vulnerable to O_3 pollution [a plant] must have foliage and be physiologically active" (ISA, p. 9-112), and agrees with the need to assess O_3 exposure that may place sensitive vegetation at risk of effects. We do not agree, however, that the maximum 3-month period must overlap exactly with the most sensitive part of the growing season for each species to do so. We note that "[v]egetation across the U.S. has widely varying periods of physiological activity during the year due to variability in climate and phenology" (ISA, p. 9-112). Further, studies of some sensitive species have shown that the period of highest O_3 uptake does not always coincide with the late summer season (in which O_3 concentrations are traditionally the highest), and a study of ponderosa pine in some locations has reported higher O_3 uptake in the winter than in the late summer, citing factors such as lower soil moisture during late summer (ISA, section 9.5.3.2; Panek 2002, 2004). Given the "significant variability in growth patterns and lengths of growing season among the wide range of vegetation species that may experience adverse effects associated with O_3 exposure, no single time window of exposure can work perfectly for all types of vegetation" (ISA, p. 9-112). Thus, while we note that, as described above, our W126 calculations have omitted the two mid-winter 3-month periods (November-January and December-February), the inclusion of periods with one or two winter months may still be of relevance to sensitive species (although we would expect an elevated W126 based on these periods to be rare). Thus, the approach used tends to include periods in which there may be a species that is biologically active during that time. We recognize the potential to include months where vegetation is not biologically active as an uncertainty in our assessment approach that may, in some locations, contribute to an overestimate of the potential for effect. The weight placed on this uncertainty, among the various uncertainties that may contribute to different directions of bias, is, as summarized in section IV.A of the preamble to the final rule, a judgment made by the Administrator in reaching her decision on the appropriate secondary standard.

e. ***Comments on Form and Averaging Time***

(22) One comment states that in revising the secondary standard, the Agency has "arbitrarily rejected or ignored advice and input from the CASAC and the National Park Service," has apparently not based the target range of cumulative exposures on matters of public welfare protection and must select a revised standard that reflects recommendations "of its scientific advisors and federal land managers."

Response: We disagree that we have arbitrarily rejected advice and input from these entities and have not based the secondary standards decision on matters of public welfare protection. The NPS comments received and considered over the course of the review are similar to some comments from the public and those comments on the secondary standard are also considered in the preamble to the final rule and in this RTC. With regard to consideration of matters of public welfare protection, as described in the preamble to the final rule, the Administrator's decision on a revised secondary standard that provides the requisite protection to the public welfare has taken into account the information and assessments presented in the ISA and PA, the advice and recommendations of CASAC, the public comments, and public welfare judgments. With regard to federal land managers, we note that in discussing potential public welfare impacts, the EPA considered NPS guidance materials developed for federal land managers in both the proposal and preamble to the final rulemaking. Lastly, we note that, contrary to the comment's implication, the CAA explicitly recognizes that the Administrator may not always agree with CASAC. Thus, consistent with the EPA's statutory responsibilities in considering advice from the CASAC, this advice has been given full consideration and any departures from it are described in the preamble to the final rule (especially in section IV.C.3 with regard to advice on the standard) and this RTC.[124]

(23) *Comment:* Some commenters stated that the proposal didn't adequately explain EPA's divergence from CASAC's recommendation that the revised secondary standard have a W126 index form.

Response: Section IV.C.2 of the preamble to the final rule discusses the comments received with regard to the form for the revised standard, while section IV.C.3 describes the Administrator's consideration of CASAC advice in this area and the basis for her conclusion that a standard of the current form and averaging time, with a revised level, would be requisite.

(24) *Comment:* In stating support for a W126 form for the standard, one comment claimed that while the current form and averaging time may often be sufficiently protective as compared with the target W126 index values, this may not always be

[124] Where the standard differs in an important respect from CASAC advice, EPA is to provide an explanation of the reasons for such differences. CAA sections 307(d)(3) and 307(d)(6)(A); see also Mississippi v. EPA, 744 F.3d 1334, 1354 (D.C. Cir. 2013).

the case, "especially as the NAAQS becomes lower" (e.g., comments from New Hampshire Department of Environmental Services).

Response: The comment did not provide information to explain the basis for their comment or clarify the magnitude intended by "lower NAAQS." The EPA notes, however, that at the levels of 3-year W126 index on which the Administrator focused in her decision in this review, the revised level for the standard was shown in the air quality analyses to provide the desired degree of control of cumulative exposures. To the extent the comment is suggesting that the current form and averaging time might not provide sufficient protection if the EPA were to revise and lower the standard in a future review, we note that the decision in that review as to the adequacy of protection provided by such a revised standard would be based on consideration of the information available at that time.

(25) *Comment:* As support for the view that EPA needs to revise the form and averaging time, some commenters note that the proposal recognized the relatively lesser density of monitors in rural areas which they claim makes uncertain the degree to which a revised level for the current standard would provide the appropriate degree of protection for vegetation-related effects on public welfare in those areas.

Response: The EPA recognizes that there is a lesser density of monitors in rural as compared to urban areas. We note, however, the analysis of rural sites in the ISA (pp. 3-131 to 3-132), which indicated that the O_3 concentrations at the rural sites in the west, which had the relatively higher concentrations, were influenced by upwind air quality. Thus, as noted in the proposal, such rural areas would be expected to benefit from O_3 precursor emissions reductions to attain the revised standard in urban areas, which will contribute to reduced concentrations over the season in downwind rural areas. Further, the EPA's analysis of a more extensive O_3 monitoring data set, for the period from 2001 to 2013, found that monitors in the West and Southwest regions highlighted by the comment, in expressing concern for the extent to which a 4^{th} high standard would be expected to control cumulative seasonal exposure in terms of W126 index values, actually exhibited the greatest response in W126 values per unit change in 4^{th} max values (Wells, 2015b).

(26) *Comment:* In support of their view that a secondary standard based on the current form and averaging time would not adequately control cumulative seasonal exposures, some comments pointed to statements in the PA regarding ISA observations on six rural mountain sites and statements from documents in the 2008 review as support for their concern that rural areas, including those in mountain areas, might not be adequately protected, additionally noting the relatively lesser density of monitors in such areas.

Response: We agree that there are fewer O_3 monitoring sites in rural areas and that, as described in section 3.6.3.2 of the ISA, air quality patterns at a subset of the six rural mountain sites considered do not show the diel pattern of O_3

concentrations that is common in urban areas and that the PA states would suggest that such sites "may have increased cumulative seasonal values coincident with increased daily 8-hour peak O_3 concentrations" (PA, p. 6-37). The PA then notes that consideration of these six sites indicates that this does not occur at the sites without the urban influence, which are the three eastern mountain sites. In considering the findings of this analysis, the PA goes on to note that the cumulative seasonal values for the eastern sites are generally lower than those of the western sites. While these site-by-site observations are interesting and may be informative regarding sources of O_3 at the sites considered, they do not address the issue raised by the commenters of the adequacy of control on cumulative seasonal exposures likely to be achieved by a secondary standard for which the level is revised and the form and averaging time retained.

To address that issue, the EPA has considered more extensive analyses of a much larger dataset (than the six rural sites in the ISA)[125] that are documented in the technical memoranda to the docket (Wells, 2014a, 2015b). As described in sections IV.C.2 and IV.C.3 of the preamble to the final rule, these air quality analyses of 13 years of monitoring data identify the occurrence of cumulative exposures of a magnitude of interest (e.g., 17 ppm-hrs) at sites that meet various alternative standard levels (e.g., 70 ppb). These analyses involving nearly 4000 3-year W126 index values from across the most recently available 11 3-year periods of data at monitors for which the 4th high metric is at or below 70 ppb find fewer than a handful of occurrences of a 3-year W126 index value above 17 ppm-hrs at sites meeting a revised standard level of 70 ppb.[126]

Further, we note that the statements from the 2008 review with regard to potential for lack of adequate protection appear to relate to analyses in the 2007 Staff Paper that considered the magnitude of W126 index at monitoring sites that met different alternative levels for the 4th high metric, although these analyses did not distinguish rural or high-elevation sites from urban or low-elevation sites. In these analyses, however, W126 index values down to 13 ppm-hrs were considered in describing exposures of interest. Thus, these observations do not address the EPA's consideration in this review of control that might be achieved of higher cumulative exposure levels, including 17 ppm-hrs.

Moreover, while the relatively lesser density of monitors in rural areas of the U.S. than in urban areas may indicate some greater uncertainty for conclusions reached regarding unmonitored rural (as compared to urban) areas, the analyses of air quality data from both rural and urban areas, across the past 13 years, as summarized in the proposal and the preamble to the final rule, and described in technical memoranda to the docket indicate that reductions in 4th high metric (the

[125] As noted in the ISA, "[t[hese six areas investigated were selected as illustrative examples and do not represent all rural areas in the U.S." (ISA, p. 3-144).

[126] We note that these occurrences are all at western locations and not eastern locations as might be expected based on the sentences in the PA regarding the six sites considered in the ISA on which the commenters focused.

design value for the 2008 secondary standard) are associated with reductions in W126 index (Wells, 2014a, Wells, 2015b). These analyses further find that across the 11 3-year periods analyzed, sites meeting a 4th high metric of 70 ppb have cumulative exposures at or below 17 ppm-hrs in all but a handful of instances (Wells, 2015b) - isolated, rare instances which the Administrator does not judge to be indicative of adverse effects to the public welfare, as explained in section IV.C.3 of the preamble to the final rule.

(27) *Comment:* Some commenters disagree with EPA's reliance on the air quality analysis of 4th high and W126 metrics documented in the technical memo to the rulemaking docket (Wells, 2014a), variously stating that the memo had no CASAC review, and objecting to the memo's use of a 3-year average W126 index, noting the preference of the CASAC for an annual W126 index. Other comments expressed the alternate view that the air quality analyses described in Wells (2014a) were appropriately developed by EPA after CASAC review of the second draft PA and are responsive to CASAC comments regarding the biological relevance of the W126 metric for the key welfare effects in this review.

Response: We disagree with the former commenters regarding CASAC review of the air quality analyses of design values for the existing standard and the W126 metric. As described in the proposal (section IV.E.4), the specific air quality analyses described in Wells (2014a), which have been updated and expanded in Wells (2015b), are similar to and an expanded version of analyses included in the second draft PA (PA, Appendix 2B), which was reviewed by the CASAC. As discussed in the proposal (and in the preamble to the final rule), the Administrator judged it appropriate to focus on a W126 index, averaged over three years, for her purposes in considering a level of protection appropriate for the revised standard. Her reasons for doing so, even as the CASAC favored a single-year metric, are discussed in the preamble to the final rule (sections IV.C.2.c and IV.C.3). Accordingly, the air quality analyses documented in the final PA, as well as the two technical memoranda, have informed the Administrator's judgment regarding the cumulative seasonal exposures to be expected with air quality conditions associated with alternative levels for the secondary standard and thus her decision on the appropriate revision to the secondary standard.

(28) *Comment:* A few commenters stated that EPA should retain the same form as primary, but make both forms the 5th high. As support for this view, these commenters provide health-based reasons (see section II.A above with regard to comments on the form for the primary standard) or suggest that the EPA's air quality analysis indicates that target W126 index values can be achieved with such a form and an unrevised level of the standard.

Response: The EPA disagrees with the commenters that the reasons cited by the comments related to public health protection are relevant to consideration of the secondary standard. The EPA additionally disagrees that the air quality analyses support the commenters' statement that a standard defined as the 3-year average of the 5th highest 8-hour daily maximum concentrations would provide control of

W126 index values of interest in this decision. The EPA's air quality analyses do not include a 5th high metric and so cannot support (or refute) such a statement. Further, the air quality analysis indicates that a level lower than the 2008 level of 75 ppb is needed in order to reduce cumulative seasonal exposures to the levels that the Administrator judges to be associated with the requisite protection for the secondary standard.

(29) *Comment:* One comment which indicated support for a secondary standard that will protect public welfare and is "closely aligned" with the form of the primary standard, recommended that as an alternative to consideration of the W126 index EPA might consider lengthening the averaging time to something like a weekly or monthly maximum daily average. This comment expressed the view that such an approach would minimize the weight natural background has on a design value, while still reflecting the influence of controllable anthropogenic sources, and it would provide a framework for future revisions without a risk of setting the standard to a value within "background"

Response: As summarized in the ISA and past AQCDs, exposures over a month or longer (e.g., cumulative exposures over a season) are of relevance to vegetation effects of O_3. Studies of O_3 effects on plants are generally on the order of three months in length in order to assess the effects over a growing season (ISA, p. 9-112). Accordingly, E-R functions have been developed from these studies based on a seasonal or three-month W126 index. The W126 index also reflects an emphasis on multiple occasions of higher concentrations (ISA, section 9.5.3.2). Depending on the level and form for a standard of a weekly or monthly averaging time, such an approach might also be used to provide control of the cumulative exposures to relatively higher concentrations. As discussed in sections IV.C.2 and IV.C.3 of the notice, however, the EPA has concluded that protection against cumulative exposures over seasonal periods can be provided by a standard with an 8-hour averaging time. Such a standard, in addition to limiting occurrences of maximum 8-hour concentrations, also controls the broader distribution of O_3 concentrations including the magnitude of concentrations (and cumulative exposures, such as those quantified by the W126 index) for longer time periods. Thus, the current evidence does not indicate that a longer averaging time is needed to provide the level of protection judged to be appropriate by the Administrator for the secondary standard. Additionally, the EPA finds no support in the current evidence for the commenter's view that a longer averaging time is less influenced by background sources of O_3, and the commenter provided no such evidence.

(30) *Comment:* In support of EPA's proposal to retain the current form and averaging time, one comment stated that the W126 index, which it described as a one-size-fits all approach that attempts to quantify effects to diverse vegetation over large distances, terrains and climates with a single index, is not comprehensive enough to accurately capture the secondary effects of O_3 equally.

Response: As described in the preamble to the final notice, the EPA is retaining the current form and averaging time of the secondary standard. The EPA is additionally revising the level for the standard based on consideration of O_3 effects related to cumulative seasonal exposures, which are considered using the W126 exposure index. While the EPA recognizes that index may not capture every aspect of the exposure circumstances that influence all welfare effects of O_3 across the U.S., this index is the best supported by the current evidence for the Agency's purposes in this review. For those effects with most substantial evidence bases (e.g., growth reduction), the evidence demonstrates that an index such as the W126 index, which accumulates the hourly concentrations and preferentially weights the higher concentrations, provides improved explanatory power of E-R models over other indices based simply on mean or peak exposure values (ISA, section 9.5.5). The CASAC concurred with EPA that the W126 index is a biologically relevant exposure metric for considering effects on vegetation (Frey, 2014b).

(31) *Comment:* Some commenters stated that there is insufficient justification to set a secondary standard with a W126 index form or to establish a level for the current 4th high form based on targeting a given level of the W126 index, stating that EPA, in concluding that the W126 index was the most appropriate metric for considering vegetation effects of O_3, did not give adequate consideration to the evidence for alternative metrics. For example, these commenters state that a long-term study considered in the ISA (Percy et al., 2007, and a subsequent analysis of, Percy et al., 2009), conducted at the Aspen FACE facility using aspen and birch trees, found that a metric based on the 4th highest daily maximum 8-hour O_3 concentration and several meteorological variables, which the commenters describe as biologically-based, predicted growth of mean basal area well, with strong R^2 and very low *p* value. The commenters also identified other reasons in support of their view that the W126 index is not an appropriate metric for characterizing exposures of concern for plant growth: (a) W126 gives increased weight to higher O_3 concentrations but does not consider the role of moderate concentrations, stomatal conductance, plant defenses, and their diel patterns (which will also influence plant response) and (b) a paper in the ISA by Panek (2004) concluded that cumulative metrics based on concentration, such as the W126 index, are not appropriate for some species, such as Ponderosa Pine in Sierra Nevada.

Response: While, as described in the preamble to the final rule, the Administrator has concluded that the requisite protection for the secondary standard can be achieved by revising the level and retaining the current form of standard, the EPA has concluded that the W126 index is an appropriate metric for assessment of vegetation effects of O_3 and has used it for that purpose in this review. In so doing, the EPA recognizes that the W126 index may not capture every aspect of the exposure circumstances that influence all welfare effects of O_3. With regard to consideration of the evidence for alternative metrics, however, the ISA and past AQCDs have evaluated such evidence in reviews dating back to the 1997 review (ISA, section 9.5; 2006 AQCD, sections AX9.4 and AX9.5; 1996 AQCD, section

5.5). For those effects with most substantial evidence bases (e.g., growth reduction), the currently available evidence demonstrates that an index, such as the W126 index, which accumulates the hourly concentrations and preferentially weights the higher concentrations, provides improved explanatory power of E-R models over other indices based simply on mean or peak exposure values (ISA, section 9.5.5). The analysis referenced by the comment (Percy et al., 2007),[127] which matched "12 yearly exposures and 12 yearly tree sizes, while disregarding age as if size did not also depend on it" and "compares the size of trees of various ages as if they were all the same age," does not take into account the fact that "[n]ot only does the size of the tree at the beginning of each year of exposure increase, but size is also dependent on the exposure from previous years," so that "the relationship of response and exposure must be analyzed either one year at a time, or by standardizing the response as a yearly increment relative to size at the beginning of each year" (ISA, pp. 9-128 to 9-129). Accordingly, the ISA concluded the analysis was "not informative" to consideration of E-R functions (ISA, p. 9-129).[128]

With regard to flux-based approaches, the ISA concludes that "[t]he lack of data in the U.S. and the lack of understanding of detoxification processes have made this technique less viable for vulnerability and risk assessments in the U.S." (ISA, p. 9-116). In comments during this review, the CASAC additionally stated with regard to flux-based metrics that "excessive uncertainty remains at this time" (Frey, 2014b, p. 9). With regard to the conclusion in Panek (2004), that study found that periods of highest gas exchange during the study year did not correspond to periods of peak O_3 concentrations, particularly in areas experiencing drought. This study did not measure effects (e.g., growth), making it unclear whether a metric not based on concentration (e.g., one based on O_3 uptake) would better predict O_3 effects than cumulative metrics in this situation. Thus, we have concluded that the currently available evidence, particularly with regard to growth effects, which are a primary focus in this review, supports a focus on the W126 index, a conclusion with which the CASAC concurred (Frey, 2014b).

(32) *Comment:* In comments regarding the option of establishing a secondary standard in the form of a W126 index, some commenters described a number of changes to data handling, monitoring, modeling and implementation procedures that they stated would be needed for such a standard, and challenges related to

[127] The comment also references a second publication by Percy et al. (2009), which is a book chapter describing the same results published in Percy et al. (2007). The 2007 study has been considered in the ISA.

[128] In considering this study, the ISA additionally stated that "[t]he Aspen FACE experiment has provided extensive data on responses of trees beyond the seedling stage under long-term exposure, and also on ecosystem-level responses (Section 9.4), but the only attempt to use those data in a continuous model of the response of tree growth to O_3 exposure (Percy et al., 2007) suffered from severe methodological problems" (ISA, p. 9-119).

such changes, including those associated with the burdens and costs of making such changes.

Response: As discussed in the preamble, the costs of implementation of a new standard cannot be considered in setting or revising the NAAQS (see section I.A of the preamble to the final rule describing the relevant case law). Accordingly, EPA did not consider the costs and burdens of procedural or programmatic changes that would be needed to implement a secondary standard in a W126 form, such as those raised by commenters, in its decision on the secondary standard. Moreover, as EPA is not revising the secondary standard to the form of a W126 index, it is unnecessary to address data handling, monitoring and implementation procedures that would be required for a standard using such a form. For these reasons, EPA is not further addressing these comments.

(33) *Comment:* One comment that supported setting the secondary standard equal to the primary standard suggested as an alternative that EPA could leave it to the states to determine if the secondary standard should be the same as the primary standard or based on the W126 metric.

Response: Contrary to the suggested alternative, the final rule for the O_3 NAAQS does not allow states to select the form of the revised secondary standard. We note that separate from any state action, section 109 of the CAA entrusts the Administrator with the responsibility to establish secondary national ambient air quality standards, which she has done with this rulemaking. To the extent that a state has authority under state law to establish additional air quality standards that would apply only to that state, it could consider doing so using a different form than that used by the NAAQS, if such an approach was appropriate under the applicable legal authority.

f. *Comments on Revisions to the Standard Level*

(34) *Comment:* One comment claims that it is unlikely that the difference in the two proposed levels of 0.065 and 0.070 ppm (0.005 ppm) can be reliably demonstrated as a true differences given that MDL for number of monitors are within this range.

Response: We disagree with this comment. A method detection limit (MDL) or lower detectable limit (LDL) for a monitor is the lowest concentration that a monitor can detect. The ability to detect the difference between 0.065 ppm and 0.070 ppm is related to the manufacturer's stated resolution for a monitor and not a monitor's MDL or LDL. Modern O_3 monitor manufacturers have a stated resolution of 0.1 ppb (U.S. EPA, 2014d). The resolution is the manufacturer's stated ability to be able to detect a change in concentration. Therefore, a monitor with a resolution of 0.1 ppb can detect a change in concentration between 70.0 and 70.1 ppb. Some much older monitor manufacturer's had stated resolutions of 1 ppb. Without knowing exactly what model and age of ozone monitor is being used at each of the more than 1300 sites in the O_3 NAAQS network, an estimate

of 1 ppb resolution would be a conservative estimate of a monitor's ability to detect a change, which is much less than 5 ppb. Thus, current O_3 analytical methods are more than adequate to detect differences between 0.065 and 0.070 ppm.

(35) *Comment:* One comment stated that EPA must reduce the secondary standard to 0.065 ppm in order to protect urban sycamore trees because research from the USDA Forest Service in Syracuse, NY demonstrates that we must reduce the level of the standard to 0.065 ppm in order to protect our urban inner cities' sycamore trees that cleanse our air of various pollutants. In expressing this view, this comment cites Nowak et al. (2002, 2006).

Response: The papers cited by the comment develop estimates of pollutant removal by urban trees. These papers provide no information related to whether O_3 has a detrimental effect on the ability of urban trees to remove pollutants from the atmosphere, nor do they assess the extent to which such a function would be protected by a secondary standard of 0.065 ppm.[129] Further, while the WREA for the current review included analyses that estimated changes in pollutant removal estimates for different air quality scenarios, and the PA recognized appreciable uncertainty in these estimates, the WREA analyses did not indicate substantial differences among the different W126 exposure scenarios assessed (WREA, section 6.7; PA, section 6.3), and these quantitative estimates were not given primary consideration by the Administrator in reaching her decision on the revised secondary standard.

(36) *Comment:* In support of the view that the secondary standard level should be revised to 0.065 ppm, one comment stated that the U.S. Department of Agriculture (USDA) had reported that "satellite views of the Midwestern United States show that ground ozone levels above 50 ppb (0.050 ppm) could reduce soybean yields by at least 10 percent." As support for this statement, the comment cites a USDA press release which references a study by Fishman et al., (2010).[130] This comment further stated that "soybean yields decrease to about 75 percent of normal, while wheat, corn and alfalfa yields to about 90 percent of normal" at 0.060 ppm, referencing Adams et al. (1989), as cited by Chameides et al. (1999).

Response: The papers cited by the comment in support of a standard level of 0.065 ppm (which were assessed in the ISA or a prior AQCD) did not address air

[129] Although the studies cited by the comment are not focused on the effects of O_3, we have provisionally considered them in the context of the comment (Appendix B).

[130] The comment referenced this USDA-ARS news website link with a press release titled "Monitoring Ground-Level Ozone from Space" (http://www.ars.usda.gov/is/pr/2011/110829.htm). The news article describes a study led by NASA in collaboration with USDA-ARS researchers, Fitz Booker and Lisa Ainsworth. The article reports that the research is published in the Journal Atmospheric Environment. A simple search of journal's website for Ainsworth or Booker as authors results in finding the research article described in the USDA press release (Fishman et al., 2010, http://www.sciencedirect.com/science/article/pii/S1352231010000415)

quality conditions associated with such a level. Rather, these papers summarized O_3 concentrations in ambient air as seasonal monthly average concentrations, in the case of Fishman et al. (2010), or seasonal daytime average concentrations, as in the case of Adams et al. (1989). In both cases, the three year average of fourth highest daily maximum 8-hour concentration would be higher than the concentrations of the metrics used (seasonal monthly and seasonal daytime averages). Ambient monitoring data summarized into design values on the EPA's web site indicate that many locations in the tri-county study area over the study period (2002-2006) for Fishman et al. (2010) exceeded 0.070 and the highest values extended above 0.080 ppm (http://www.epa.gov/airtrends/values.html). The study by Adams et al. (1989) employed E-R functions, based on the NCLAN studies and quantifying exposures as seasonal 7-hour or 12-hour average concentrations, to estimate impacts on crop yield and associated monetary impacts for specific air quality scenarios, including a potential secondary standard of 50 ppb as a 3-month average of daily 7-hour average concentration, with the three months being the three consecutive months resulting in the highest average. Such a form and averaging time differs substantially from those considered in this review. Air quality conditions corresponding to a 50 ppb 3-month average of daily 7-hour average concentration would have a three year average of fourth highest daily maximum 8-hour concentration that was higher than 50 ppb. Further, the study by Adams et al. (1989) involved comparisons to a base scenario that used O_3 concentrations in the 1981-1983 time period, a time in which O_3 concentrations were much different than today. Accordingly, the consideration of a standard level of 50 ppb (for a form much different than the current standard form) in the study by Adams et al. (1989) is not informative to the current review. Thus, the materials cited by the comment do not provide evidence regarding a 0.065 ppm level for a standard of the current form.

(37) *Comment:* In support of their recommendation for a standard level of 65 ppb, some tribal commenters generally noted concern about the effects of O_3 on plant species of cultural importance to native American tribes, with some commenters citing the PA list of O_3-sensitive plants used by some tribes (PA, Appendix 5A), stating that protection of these plant uses necessitates a level no higher than 65 ppb.

Response: These commenters did not provide specific evidence regarding the impact of a standard level of 65 ppb (or alternative levels) on the species identified in the PA as O_3 sensitive due to foliar injury effects (PA, Appendix 5A) and EPA is not aware of any specific basis for such a conclusion. As noted in the preamble to the final rule and elsewhere this RTC, while the Administrator concludes that her judgments on the revised secondary standard, reached with a focus on growth-related effects, would also be expected to provide an additional desireable degree of protection against visible foliar injury in sensitive vegetation.

(38) *Comment:* One comment suggested that studies describing the importance to public health of the natural environment and the presence of vegetation provided

support for their view that EPA should revise the secondary standard level to 65 ppb.

Response: This comment did not provide evidence regarding the impact of a standard level of 65 ppb (or alternative levels) on benefits to human wellbeing (and public health) related to the presence of vegetation that might be considered pertinent to the Administrator's decisions on protection of the public welfare, and the EPA is not aware of evidence in support of this specific conclusion.

3. Additional Comments on Interpretation of Welfare Effects Evidence

(1) *Comment:* As support for the view that the scientific evidence does not support a revised secondary standard, one comment stated that the ISA relies in part on a study by Wittig et al. (2009), a paper that the comment describes as unreliable due to its reliance on other studies that used what the comment describes to be unreliable models to establish pre-industrial O_3 concentrations.

Response: We disagree with the comment's characterization of the study by Wittig et al. (2009). This study is one of several meta-analyses newly available in this O_3 NAAQS review and the ISA states that this analysis "demonstrates the coherence of O_3 effects across numerous studies and species that used a variety of experimental techniques, and these results support the conclusion of the previous AQCD that exposure to O_3 decreases plant growth" (ISA, p. 9-43). The meta-analysis estimated differences in plant physiology and growth associated with differences in O_3 concentration based on "quantitatively compiled peer reviewed studies from the past 40 years on the effect of current and future O_3 exposures on the physiology and growth of forest species" (ISA, p. 9-43). In summarizing the findings, the authors compared the measures of plant physiology and growth from ambient air with those from charcoal-filtered (CF) air (a common method for removing O_3 from air for purposes of a control exposure) as an approach that "provides a measure of how the elevation of [O_3] that has occurred since the Industrial Revolution" (Wittig et al., 2009, pp. 399-400). Contrary to the comment's statement, the pre-industrial O_3 concentrations referenced in the paper were based on measurements, not modeling. The comment provides no reason that would lead us to discount the conclusions we have drawn from the paper and summarized in the ISA and above.

(2) *Comment:* As support for their view that the scientific evidence does not support a revised secondary standard, some commenters stated that the studies cited for the ISA crop yield analysis (ISA, pp. 9-57 - 9-67) did not account for agricultural management and policy factors, which the commenter states have the potential to confound or mitigate the impacts of O_3.

Response: We disagree with this comment, noting that many of the studies cited in the ISA were designed with the use of standard agricultural practices in order to reflect "real world" conditions. For example, the ISA notes that "[t]he cultural conditions used in the NCLAN studies approximated typical agronomic

practices," with one of the primary objectives of the research being "to assess the national economic consequences resulting from O_3 exposure of major agricultural crops" (ISA, p. 9-119). The NCLAN experiments yielded 54 exposure-response curves for 12 crop species, some of which were represented by multiple cultivars at several of 6 locations throughout the United States" (ISA, p. 9-119). The comment provided no examples of the factors claimed to not be accounted for in the studies or any supporting documentation.

(3) *Comment:* In questioning EPA's use of tree seedling RBL estimates in considering growth effects in the field and the proposal's citing of McLaughlin et al. (2007a) as support for this, one comment stated that the findings of McLaughlin et al. (2007a) may be confounded by the co-occurrence of dry conditions during high O_3 years (citing Fiore et al., 2002 as indicating that dry summers will reduce growth and high O_3 is more likely in hot dry summers), leading to false attribution of growth effects to O_3. This comment states that a more appropriate approach would be to directly compare growth for the same year(s) between similar sites but where O_3 levels differ (e.g., urban vs nearby similar rural sites).

Response: We disagree with this comment, and do not find the results of McLaughlin et al. (2007a), that were cited in the proposal, to be affected by co-occurring dry conditions and high O_3. The high O_3 year (2002) in this study was noted by the authors to have similar rainfall patterns to the low O_3 year (2001). The temperatures were similar between years and the Palmer drought index was in the mid-range of values for both years (McLauglin et al., 2007a). The authors determined the major explanation for the differences in growth to be attributable to effects of cumulative O_3 exposure (McLauglin et al., 2007a). Depending on the details, the study design mentioned by the comment of comparing similar sites during the same year where only O_3 differs may be more or less informative. Where such studies are available they have also been assessed as part of the air quality criteria in the ISA (e.g., the cottonwood study by Gregg et al., 2003). However, both types of studies are informative and add to the evidence base documenting the effects of O_3 in the natural environment. Thus, where available such studies have been considered in this NAAQS review.

(4) *Comment:* One comment states that the EPA does not consider that factors other than O_3 (such as availability of nitrogen and water) are likely the limiting factors on ecosystem net primary productivity, citing Huang et al. (2007).

Response: While we recognize that abiotic factors other than O_3 limit the net primary productivity in some ecosystems, we disagree with the suggestion that nitrogen and water availability are the limiting factors on productivity in all ecosystems and do not find support for this view in the paper cited in the comment.[131] Further, contrary to the implication of the comment, the EPA has

[131] The EPA has provisionally considered the paper by Huang et al. (2007) cited in the comment (and listed in Appendix B). Based on this provisional consideration, we conclude that the information provided

assessed an array of abiotic factors, including nitrogen and water availability, with regard to the extent of their impact on plant susceptibility to O_3 effects (e.g., ISA, sections 9.4.8.3 and 9.4.8.4). With regard to ecosystems during times of limited water availability, multiple effects of O_3 have been reported. For example, while effects of O_3 have been less pronounced in drought conditions in some field experiments, drought has also been indicated to exacerbate the effects of O_3 on plants (ISA, section 9.4.8.3). Similarly, the limited number of studies published since the last review indicated that, consistent with the conclusions in the last review, the interactive effects of N and O_3 varied among species and ecosystems (ISA, section 9.4.8.4). Thus, we find no basis in the evidence for the implication of the comment that O_3 does not have an influence on ecosystem productivity. Rather, we have concluded there to be a causal relationship between O_3 and reduced productivity in terrestrial ecosystems (ISA, p. 9-148).

4. Additional Comments on the Welfare Exposure and Risk Assessment

(1) *Comment:* In support of the view that the current standard provides adequate public welfare protection, some commenters point to what they characterize as substantial uncertainties in the WREA analyses, which are said to pertain particularly to the west and southwest regions and which the comment says should limit EPA's consideration of the WREA results, particularly in those regions. In this regard they variously state: (1) emissions reductions to meet different air quality scenarios do not reflect spatial and temporal heterogeneity that may occur with local and regional reductions; (2) concentration estimates in west and southwest may be influenced by wildfire smoke interference with O_3 instruments, the seasonal use of some monitors and potential for some to be missing time intervals; (3) W126 values are influenced by significant contributions from stratospheric O_3 sources in high-elevation western U.S. areas (citing Lin et al., 2012); and (4) plant exposure is overestimated by W126 in regions characterized by "non-average" conditions such as drought years or low soil moisture periods when O_3 may be higher but vegetation may have closed stomata. With regard to the last statement, the commenters additionally state that the W126 metric assumes that vegetation is physiologically active during the entire exposure duration (i.e., the 3 months of the year with the highest ozone concentrations), thus likely overestimating exposures, particularly in regions with "non-average" conditions.

Response: The EPA agrees that some of the items cited by the commenters are uncertainties in some of the WREA analyses. The first two items are recognized in the WREA's summary of the qualitative uncertainty analysis of key air quality elements (WREA, Table 4-5). With regard to the first, the conclusion from this uncertainty analysis is that the potential influence of this area of uncertainty on the risk estimates associated with the air quality scenarios is a tendency toward overestimation (WREA, p. 4-42). With regard to the second, any influence of

by this study does not materially change the conclusions reached in the ISA. Accordingly, as discussed in section I.C of the preamble, EPA is not re-opening the air quality criteria for this review.

uncertainties associated with these and other aspects of the ambient air monitoring data is concluded to go in both directions and the overall impact is believed to be minimal (WREA, p. 4-39). We also agree with commenters that W126 index values can be influenced by stratospheric O_3 contributions under some circumstances, particularly in the intermountain western U.S" (PA, section 2.4.5). Since the WREA air quality scenarios simply used model-adjusted, measured concentrations, without separating estimated O_3 concentrations from background and U.S. emissions, however, it is not clear why the comment is describing the occurrence of this phenomenon as a particular uncertainty in the air quality scenario O_3 concentrations. Additionally, we note, however, that the results of WREA analyses for the current standard or W126 air quality scenarios were not among the Administrator's primary considerations in reaching her proposed and final decisions on the secondary standard.

With regard to plant uptake during drought or low soil moisture periods, the EPA agrees that the evidence indicates uptake may be lower during periods of drought that during normal periods, but the EPA disagrees with the statement that the W126 index assumes vegetation is physiologically active during the entire exposure duration. The W126 index is simply an estimate of cumulative seasonal exposure. In using this metric, the EPA has defined it to cumulate hourly O_3 concentrations across the 12 hours from 8 a.m. to 8 p.m. for each day in the three consecutive months that result in the highest result. A key use of the W126 index estimates in the WREA is in combination with E-R functions for RBL and these estimates are based on studies documenting RBL associated with the thus-quantified cumulative O_3 exposures; there is no assumption regarding O_3 uptake. Thus, the EPA disagrees that WREA results (e.g., related to RBL) are overestimated in regions with "non-average" conditions.

(2) *Comment:* One comment stated that the results of the WREA county RBL analysis are overestimates that do not accurately represent the potential impact of O_3 on forests. In support of this view it makes three assertions claimed to bias the results: (a) each county was given maximum O_3 level within that county, which may overestimate O_3 in rural (forested) areas, (b) the majority of monitoring sites are in urban areas where O_3 is higher, and (c) biomass results are estimated relative to zero O_3 rather than to policy relevant background.

Response: We disagree with the comment that the estimates from the county RBL analysis are overestimates. Contrary to statements made with the comment, each county was not assigned the highest monitored O_3 value within the county. Rather, O_3 was estimated in 12 kilometer x 12 kilometer grid cells using spatial interpolation (the Voronoi Neighbor Averaging [VNA] interpolation technique) of the observed and model-adjusted monitoring data. The RBL was then estimated for the species with E-R functions indicated to occur in each grid cell based on estimated O_3 concentrations for that grid cell. Then the number of counties in which one or more species was estimated to have an RBL value above 2% (in any of the grid cells occurring within the county), and the number of counties in which the median RBL for a grid cell in the county was above 2% were

enumerated. This analysis was developed directly in response to advice from CASAC on the second draft PA (Frey, 2014b, p. 11). With regard to the commenter's statement about concentrations in rural as compared to urban areas, we note, as discussed in section II.B.2 of the preamble to the final rule in response to a similar comment concerning O_3 estimates in the WREA air quality scenarios for unmonitored rural areas, we have no reason to conclude that the interpolation method provided overestimates in such areas. In fact, a cross-validation evaluation of the VNA interpolation technique for W126 index values in all monitored areas across the contiguous U.S., which was performed as part of the WREA (Appendix 4A, section 4A.3), showed that the method predicted monitored W126 index with a mean bias of just -0.15 ppm-hrs (a slight under-prediction).

Lastly, the WREA estimates RBL for the cumulative seasonal O_3 exposures derived from model adjustment and interpolation of concentrations at monitors, with no subtraction of an estimate of policy-relevant background. While we agree that the results are estimates attributable to the total exposures rather than to a portion of the total exposure that might be attributable to the exposures above policy-relevant background, we disagree that this use of total O_3 concentrations estimated in this way impart an upward bias to the RBL estimates, as these concentrations contribute to the estimated vegetation exposures, and are accordingly relevant to this analysis. Further, the CASAC concurred with the air quality modeling approach employed in the WREA (Frey, 2014b, p. 2). We additionally note, however, that the WREA estimates were not a primary consideration in the Administrator's judgments regarding the adequacy of the current standard and appropriate revisions (as described in the preamble to the final rule, sections IV.B.3 and IV.C.3).

(3) *Comment:* As support for the view that estimates associated with the WREA analyses should not be used as a basis for decisions on the secondary standard, some commenters raise concerns over the use of models designed for urban applications and urban monitoring data to estimate O_3 concentrations in rural areas, with limited monitor coverage. Additional comments state that EPA's statement that higher W126 index values are underestimated in the air quality scenarios is unsupported, and that EPA should conduct further analysis of its model's effectiveness before using it as the basis for a revised standard.

Response: With regard to underestimation of the highest W126 index values, as summarized in section IV.A.2 of the preamble to the final rule, the application of the VNA interpolation method to estimate W126 index values at the centroid of every 12 kilometer x 12 kilometer grid cell, rather than only at each monitor location, results in a lowering of the highest values in each region, as can be seen from the information in Table 4-3 and Figure 4-7 of the WREA. With regard to the influence of urban monitoring data on O_3 estimates for rural areas, as described in the WREA (section 4A.2.1), and sections IV.B.2 of the final rule, monitors in areas with greater monitor density (such as urban areas) have the potential for influence over much smaller areas than monitors in areas with lesser

monitor density (e.g., rural areas). With regard to evaluation of model effectiveness, the WREA includes a characterization of the uncertainty associated with the different aspects of the methods used to estimate W126 at the grid cell (WREA, section 4.4 and Appendix 4-A). These evaluations show that the potential influence of uncertainty on risk estimates goes in both directions for nearly all of the evaluated aspects (WREA, Table 4-5). For the evaluated aspect for which that is not the case, the uncertainty characterization concluded that the potential influence of uncertainty on risk estimates of the assumption of regionally-determined across-the-board emissions reductions is to produce overestimates of W126 benefits (i.e., by underestimating W126 for the model-adjusted scenarios). Additionally, a cross-validation evaluation of the VNA interpolation technique for W126 index values in all monitored areas across the contiguous U.S., indicated a mean underprediction bias of -0.15 ppm-hrs (WREA, Figure 4A-4). Further, as described in section IV.A.2 of the final notice, the VNA technique also has the effect of reducing the highest W126 values estimated for monitor locations in each region. Thus, our statements regarding underestimates of the highest values are supported, and substantial evaluation of the modeling effectiveness has been performed. We additionally note, however, that the WREA air quality modeling and analyses based on model estimates were not a primary consideration of the Administrator in her judgments on the adequacy of the current secondary standard or the appropriate revisions.

(4) *Comment:* One comment expresses the view that the WREA national park screening-level assessment estimates of W126 exposure index values for the current standard scenario do not support the need for a revised standard.

Response: As summarized in section IV.A.2 of the preamble to the final rule, section IV.C.1 of the proposal, chapter 5 of the PA and chapter 4 of the WREA, the EPA recognizes uncertainties associated with the W126 index estimates for the WREA air quality scenarios and notes that the highest W126 estimates in these scenarios are underestimated. In light of these types of uncertainties, the Administrator did not give primary consideration to WREA results for model-adjusted air quality scenarios in reaching conclusions regarding the adequacy of the 2008 secondary standard.

(5) *Comment:* Some commenters express the view that the WREA national park case studies do not support a revised standard.

Response: EPA agrees with the comment that the national park case studies do not provide information supporting a revised standard. These case studies simply describe the scope and magnitude (including in monetary terms) of ecosystem services related three national parks and also report the estimates of W126 index values in these parks for the WREA modeled air quality scenarios. The Administrator's conclusion that the secondary standard should be revised, as described in section IV.B.3, is not based on these analyses. As summarized in section IV.A.2 of the preamble to the final rule, section IV.C.1 of the proposal, chapter 5 of the PA and chapter 4 of the WREA, the EPA recognizes uncertainties

associated with the W126 index estimates for the WREA air quality scenarios. In light of these types of uncertainties, the Administrator did not give primary consideration to WREA results for model-adjusted air quality scenarios in reaching conclusions regarding the adequacy of the 2008 secondary standard.

5. Additional Comments on Air Quality Analyses

(1) *Comment:* Some commenters expressed the view that the 13-year dataset (inclusive of eleven 3-year periods) used in the air quality analysis documented in Wells (2014a) inappropriately includes data from the U.S. recession of 2008-2010, which they state is not a representative period as industrial activity and emissions declined.

Response: The EPA disagrees that it is inappropriate to include the full dataset, including periods of economic recession, in the air quality analyses considered in the review of the secondary standard. It is reasonable for the EPA, in considering the extent to which a 4th high metric may provide control of W126 index values, to consider the range of conditions that may have relevance to future conditions. So, to the extent that a recession period has the potential to occur in the future and the impact of such a period on O_3 concentrations in the past may be informative to our consideration of patterns likely to occur in the future, it is useful to include these data. That said, EPA notes that the proposal's characterization of findings from the dataset related to numbers of locations that met different values for a 4th high metric and had W126 index values above values of interest, we focused on the most recent period, from 2011-2013, rather than earlier years in which the recession period occurred as well as periods of appreciably different emissions prior to several regulatory actions to control O_3 precursor emissions (79 FR 75345-75346; Wells, 2014a). Additionally, the component of the air quality analyses that compared changes in the two metrics that occurred as a result of nationwide control programs also did not include the 2008-2010 period. Rather it assessed changes between the 2001-2003 and 2011-2013 periods.

In consideration of comments on the proposal, we have additionally summarized findings related to numbers of occurrences of sites meeting specific 4^{th} high metric values and having W126 index values above values of interest for all 3-year periods in the full 13-year dataset (Wells, 2014b), as described in sections IV.C.2.d and IV.C.3 of the preamble to the final rule, as well as section II.B.2.e of this RTC. The overall conclusions reached based on these additional summaries are not changed.

(2) *Comment:* One comment, in claiming the EPA approach in assessing the adequacy of the existing standard to be weak, states that the data used for comparing the design value for the existing standard with W126 index values are "undocumented, with unknown statistical accuracy."

Response: We disagree with the comment that the data in the analysis referenced are undocumented. The analysis is described in a 17-page technical memorandum

to the rulemaking docket, with approximately a page and a half of it devoted to describing the dataset and derivation of the metrics analyzed (Wells, 2014a). Datasets associated with the memorandum were made available to all requesters during the public comment period on the proposal. We further note that an updated and expanded version of this memo has subsequently been developed (Wells, 2015b) and is available in the docket for this rulemaking and on the Agency's website, along with the full dataset. Although the comment provides no clarification of the intended meaning of "statistical accuracy", we note that the calculation of the design values and W126 index values are described in the "Data Handling" section of the memo. As indicated there, a precision of 1 ppb is assumed for design values and 1 ppm-hr for W126 index values. Additionally, we note that that ambient measurements used for this analysis were quality assured and certified by the monitoring agencies that submit these data to AQS, as required by 40 CFR Part 58. Accordingly, we have no reason to conclude there are inaccuracies or biases associated with these data.

(3) *Comment:* In support of the view that EPA should revise the form of the secondary standard, one comment notes variation in the 4th high metric values at sites meeting any particular W126 metric value, suggesting that strategies focused on reducing peak levels may not effectively reduce the W126 metric to desired levels and citing to a recent modeling analysis (Nopmongcol et al., 2014) that investigated different precursor reduction approaches to meet potential future 4th high and W126 standards at selected urban and rural locations.

Response: For the purposes of considering the control of cumulative exposures, in terms of W126 index, expected in response to reductions implemented to meet a 4th high standard with a level revised downward, the EPA finds that the air quality analyses based on actual O_3 measurements (summarized in the technical memorandum by Wells [2015b], which expands on Wells [2014a]) provide more relevant information than air quality modeling analyses, such as those in the study cited by the comment. Various aspects of the modeling analysis study make it less well suited for these purposes. For example, the modeling study involved estimating a single year metric (e.g., the 4th highest daily maximum 8-hour average concentration) for comparison to the various alternative standard levels (e.g., 70 ppb as the fourth-highest daily maximum 8-hour concentration, averaged across three consecutive years), rather than a metric consistent with the 3-year form of the standards the Administrator considered (and adopted), and the analysis also relied on modeling (of O_3 precursor emissions estimates and meteorological conditions) to represent the baseline for the single year, rather than observed O_3 concentrations. As a result the modeling analysis does not capture the large variation in O_3 concentrations that can occur across years. Also the modeling study does not replicate a realistic emissions reduction scenario, using instead a sensitivity analysis approach in which emissions reductions are distributed evenly across all precursor sources and hours of the year. As a result we do not find the modeling study to be suited to address the issue considered here. Rather we find the EPA's air quality monitoring analyses, based on nearly

4000 ozone measurements from across the most recently available 11 3-year periods of data at monitors across the country, to be appropriate for this task.

The EPA analyses of O_3 measurements in locations across the U.S. demonstrate the reductions in W126 index that have resulted over the period between 2001-2003 and 2011-2013 from reductions for the purposes of achieving a reduced level of the standards set in terms of the 4th high metric (Wells, 2015b). These EPA analyses of recent air quality monitoring data, summarized in the proposal and preamble to the final rule include an analysis of reductions in the W126 index metric that have occurred over the period between 2001-2003 and 2011-2013 in response to nationwide controls intended to reduce the 4^{th} high metric. As summarized in section II.C.2.d of the preamble to the final rule, the regression analysis of these changes in 3-year W126 index with changes in the 4^{th} high metric across the same period indicates a fairly linear and positive relationship, with, on average, a change of approximately 0.7 ppm-hr in the W126 values per unit ppb change in 4^{th} high values (Wells, 2015b). The EPA air quality analyses additionally support the conclusion also stated in the preamble to the final rule that W126 exposures above 17 ppm-hrs at sites for which the 4^{th} high metric is at or below 70 ppb would be expected to continue to be rare in the future, particularly as steps are taken to meet a 70 ppb standard.

C. Appendix U: Interpretation of the Primary and Secondary NAAQS for O_3

This section contains EPA's responses to public comments on section V of the preamble to the proposed rule and the associated proposed rule text in 40 CFR Part 50. Comments on the proposed data handling procedures in Appendix U to 40 CFR Part 50 are addressed in section V.B and V.C of the preamble to the final rule and/or in sections II.C.1-II.C.4 below. Comments on the proposed exceptional events submission schedule in 40 CFR Part 50.14 are addressed in section V.D of the preamble to the final rule and/or in section II.C.5 below. Additional comments related to data handling and interpretation of the primary and secondary O_3 NAAQS are addressed in section II.C.6 below.

1. Comments on Combining Data at Sites with Multiple Monitors

EPA proposed to combine data at monitoring sites with two or more O_3 monitoring instruments operating simultaneously into a single site-level data record for determining compliance with the NAAQS, and proposed an analytical approach to perform this combination. Several commenters supported the EPA's proposed approach, including the State of Iowa, where 15 of the 20 monitoring sites currently operating two O_3 monitors simultaneously are located. Commenters supporting the proposal noted that a similar approach is already being used for lead and particulate monitoring, and that the proposed approach will help states meet data completeness requirements.

(1) *Comment:* A few commenters supported the EPA's proposed approach with the additional restrictions that the monitoring instruments must use identical methods and be operated by the same monitoring agency.

Response: At the time of this rulemaking, all monitors reporting O_3 concentration data to EPA for regulatory use were Federal Equivalent Methods (FEMs). All current O_3 FEMs use an ultraviolet photometry sampling methodology and have been found to meet the performance criteria in 40 CFR Part 53. Therefore, EPA has no reason to believe that O_3 concentration data should not be combined across monitoring methods at the site level. Regarding the commenters' suggestion that data should not be combined when two or more monitors at the same site are operated by different monitoring agencies, EPA is aware of only one instance where this presently occurs. In this instance, the monitors have been assigned distinct site ID numbers in the AQS database, so that data will not be combined across these monitors. Should future instances arise where two or more monitoring agencies decide to operate O_3 monitors at the same site, EPA encourages these agencies to work together to establish a plan for how the data collected from these monitors should be used in regulatory decision making.

(2) *Comment:* One state objected to combining data across monitors because the secondary monitors at their sites were used only for quality assurance purposes, and data from those monitors should not be combined with data reported from the primary monitors.

Response: Concentration data collected to meet quality assurance requirements (i.e. precision and bias data) are reported and stored in a separate location within the AQS database and are not used for determining compliance with the NAAQS. The required quality assurance data is derived from O_3 standards and not from a separate O_3 monitor. However, if a separate O_3 monitor is used strictly for quality assurance purposes and does not meet the applicable 40 CFR part 50, 53, and 58 requirements, it can be distinguished in AQS in such a manner that data from the secondary monitor would not be combined with data from the primary monitor.

(3) *Comment:* EPA is proposing to no longer consider design values from monitors other than the primary monitor that are located at multi-monitor sites. Not considering design values from monitors at multi-monitor sites is the same as reducing the number of monitors. EPA presents no reason why the number of monitors should be decreased while the standard is being made more protective, nor is there one. Rather, it is arbitrary to ignore data for monitors other than the primary monitor which EPA has in making regulatory decisions.

EPA seems to imply that there is consistency across monitors at the same site. That is not always true. For example, AQS reports the 4th high for 2013 for monitor 1 at site 060430003 in Yosemite National Park at 0.073 ppm, or above the proposed NAAQS. However, AQS reports the 4th high for 2013 for monitor 2 at that same site as 0.056 ppm or below the proposed NAAQS. This is a 30 percent spread which could not be fairly described as consistent. Nor is this an isolated example.

Response: While it is true that the total number of comparisons with the NAAQS may decrease under the proposed approach, the number of physical locations where valid NAAQS comparisons are made will not decrease, and in fact may increase due to additional sites meeting the data completeness requirements. Furthermore, EPA's proposal does not ignore data from monitors other than the primary monitor. Appendix U contains provisions for substituting data from secondary monitors at a site whenever hourly concentration values are not available for the primary monitor. Regarding the specific example provided by the commenter, EPA notes that monitor 2 collected data only during the first four months of 2013 while monitor 1 collected data for the full calendar year. Thus, one cannot make a valid comparison between their annual 4^{th} highest daily maximum values.

(4) *Comment:* Two commenters submitted similar comments citing EPA's evaluation of collocated O_3 monitoring data and precision data in the ISA (section 3.5.2), and stated that although the median differences in concentrations reported by the pairs of monitoring instruments were near zero, the extreme values were close to +/- 3.5%. The commenters argued that since the O_3 NAAQS are based on the 4^{th} highest annual value, data should not be combined across monitors because of the imprecision in the extreme values.

Response: The EPA disagrees, noting that the data presented in the ISA are based on hourly concentrations, while design values for the O_3 NAAQS are based on a 3-year average of 8-hour average concentrations. Thus, the random variability in the hourly O_3 concentration data due to monitoring imprecision will be reduced when concentrations are averaged for comparison with the NAAQS. Additionally, the precision data are typically collected at concentrations at or above the level of the NAAQS, thus the EPA expects that the level of precision documented in the ISA analysis is consistent with the level of precision in the 4^{th} highest daily maximum concentrations used for determining compliance with the NAAQS.

(5) *Comment:* In proposing to average data from two or more monitors at a given site EPA must address the issues of bias, precision, and zero offset present in all monitors from which data might be combined, developing guidance which is complete and concise. Since EPA notes that only 20 U.S. sites had even two collocated monitors, developing such guidance may require more effort than is justified. EPA should drop this proposal absent compelling evidence that it will substantially reduce the number of sites for which attainment is unknown or improve overall O_3 data completeness.

Response: The commenter appears to have misunderstood EPA's proposed approach. EPA did not propose to average data across monitors at a site, but to substitute missing data from a secondary monitor whenever data were not available for the primary monitor. EPA's proposal did state that if two or more secondary monitors were present, the data would be averaged across the secondary monitors and then substituted for missing values at the primary monitor. However, as noted in the proposal, there are currently no O_3 monitoring sites in the U.S. where three or more monitors are in simultaneous operation.

2. Comments on Site Combinations

EPA proposed to add a provision in Appendix U that would allow the Regional Administrator to approve "site combinations", or to combine data across two nearby monitors for the purpose of calculating a valid design value. Public commenters unanimously expressed general support for this proposed addition. A few commenters submitted additional considerations in their comments, which are listed and addressed below.

(1) *Comment:* Given the difficulties states have in procuring high quality monitoring sites in urban/suburban areas, where open areas are sparse and multiple emission sources are numerous, it is sensible to combine data from nearby sites shut down for reasons beyond a monitoring agency's control, but only provided that the proposal retains enough safeguards documenting the site similarities proposed for "combination" and that EPA requires simultaneous monitoring at both sites, to the extent possible.

Response: EPA believes that approval of site combinations should be handled on a case-by-case basis, and that any requests for supporting documentation should be left to the discretion of the Regional Administrator. EPA strongly encourages

states to perform simultaneous monitoring whenever it is known in advance that a monitor will need to be relocated. However, as the commenter implies, this is not always possible and shall therefore not be required.

(2) *Comment:* While there is validity to this approach in some cases, EPA has not described in detail the acceptability criteria for the formal procedure that it would use for this purpose. Combining sites may be appropriate in some instances but the existing practice of establishing a new and unique DV for a new site that does not meet the criteria for consideration of combining data with another site should be considered as the default.

Response: EPA may issue future guidance providing general criteria for determining an acceptable level of similarity in air quality concentrations between monitored locations, but will not prescribe detailed criteria for approval of site combinations in this rulemaking. EPA agrees that establishing a new and unique design value for a new site shall continue to be considered the default approach.

(3) *Comment:* Site combinations should be permitted as long as they are requested by a monitoring agency and EPA's Regional Administrator finds that both sites are representative of the area. This process avoids critical gaps in data due to unavoidable site issues. The process should be transparent and the site combination request and approval process should be documented in the agency's Annual Network Monitoring Plan. This information does not need to be included in EPA's Air Quality System because the public has limited access to this resource.

Response: EPA agrees that the site combination process should be transparent and documented in the monitoring agency's Annual Monitoring Network Plan. However, EPA disagrees with the commenter's statement that the information does not need to be included in the AQS database. Public access to the site combination information in the AQS database is necessary for transparency.

(4) *Comment:* EPA should not create a regulatory burden with overly prescriptive requirements laid out in regulations, but rather ensure they maintain flexibility so this option remains a useable tool for states.

Response: EPA agrees with this comment.

3. Comments on Change from ½ MDL to 0 in Data Substitution Tests

EPA proposed a change in Appendix U to the pre-existing 8-hour average data substitution test in Appendix P which is used to determine if a site would have had a valid 8-hour average greater than the NAAQS when fewer than 6 hourly O_3 concentration values are available for a given 8-hour period. EPA proposed to change the value substituted for the missing hourly concentrations from one-half of the method detection limit of the O_3 monitoring instrument (½ MDL) to zero (0.000 ppm). Several commenters supported the proposed change, stating that the use of a constant substitution value instead of ½ MDL, which can vary across O_3 monitoring methods, would simplify design value calculations.

(1) *Comment:* Substituting missing hourly values is irrelevant when the sum of the six or fewer hours in a given 8-hour period is equal to or greater than 0.60 ppm, the sum of eight hourly values of 0.075 ppm which will always result in an "exceedance" of the current average 8-hour NAAQS of 0.075 ppm regardless of how small an amount is substituted for the missing hours. Eight-hour data relevant to newly adopted NAAQS should be handled in the same manner (e.g., if a 0.060 ppm NAAQS is adopted EPA need only specify that 8-hour averages, where the sum of available valid hours is 0.48 ppm or greater, constitutes an exceedance regardless of the number of missing hours).

Response: EPA agrees with this comment and has added a clause in the final version of Appendix U making note of this equivalent approach to the data substitution test.

(2) *Comment:* Substituting zero is arbitrary because there is no reason to believe the value is actually zero. Rather, a review of AQS shows hours with zero ozone levels are extremely rare. The most rational approach is to extrapolate the most reasonable approximation of the hourly value based on trajectory of the hourly values closest in time to the missing hourly value or some other mathematically acceptable way to approximate. This approach is also more consistent with Congress' intent for a precautionary implementation of the NAAQS. This approach also moves the actual implementation of the standard closer to providing the protection against exposures of concerns which EPA claims to the public and CASAC that EPA is providing. Moreover, this need not be complicated, time consuming or resource intensive. It can be done using automated computer programs.

Response: The intent of the data substitution test is not to provide the most accurate estimate of the 8-hour average in the presence of missing data, but to identify situations where the available concentrations are so high that a violation of the NAAQS would have occurred regardless of the values of the missing hourly concentrations. EPA believes that zero, being the lowest concentration value physically possible, is the most appropriate value to substitute in this situation. In general, EPA does not support the use of interpolation or other means of filling in missing monitoring data for O_3 NAAQS comparisons. Such an approach would be contrary to EPA's long-standing policy of using only quality-assured and certified ambient air quality measurement data to determine compliance with the O_3 NAAQS.

4. Comments on Proposed Daily Maximum 8-hour Average Calculations

EPA proposed a new procedure in Appendix U for determining daily maximum 8-hour O_3 concentrations for the revised NAAQS. The EPA proposed to determine the daily maximum 8-hour O_3 concentration based on 17 consecutive moving 8-hour periods in each day, beginning with the 8-hour period from 7:00 AM to 3:00 PM, and ending with the 8-hour period from 11:00 PM to 7:00 AM. In addition, the EPA proposed that a daily maximum value would be considered valid if 8-hour averages were available for at least 13 of the 17 consecutive moving 8-hour

periods, or if the daily maximum value was greater than the level of the NAAQS. This procedure is designed to eliminate "double counting" exceedances of the NAAQS based on overlapping 8-hour periods from two consecutive days with up to 7 hours in common, which was allowed under previous 8-hour O_3 NAAQS. A dozen public commenters expressed support for the proposed procedure, including several states.

(1) *Comment:* One regional air quality management organization and three of its member states submitted similar comments stating that they agreed with the principle of eliminating "double counting" exceedances of the NAAQS based on overlapping 8-hour periods, but suggested an alternative calculation procedure that would accomplish the same objective. The alternative procedure iteratively finds the highest 8-hour period in a given year, then removes this 8-hour period and all other 8-hour periods associated with that day, including any overlapping 8-hour periods on adjacent days, from the data until a daily maximum value is determined for each day of the year with sufficient monitoring data.

Response: The EPA examined a similar iterative procedure in a previous data analysis supporting the proposal (Wells, 2014b, Method 1). The EPA compared this procedure to the procedure proposed by the commenters using the data from the original analysis and found the resulting daily maximum 8-hour values to be nearly identical (Wells, 2015a). Additionally, the commenters' procedure suffers from the same limitations the EPA identified previously in the original analysis: added complexity in design value calculations, longer computational time, and challenges to real-time O_3 data reporting systems, which would have to re-calculate daily maximum 8-hour values for the entire year each time the system was updated with new data.

(2) *Comment:* Three states submitted comments stating that they agreed with the proposed calculation procedure, but disagreed with the proposed requirements for determining a valid daily maximum 8-hour O_3 concentration. These states were primarily concerned that the proposed requirements would only allow a monitoring site to have four missing 8-hour averages during a day before the entire day would be invalidated, compared with six missing 8-hour averages allowed previously. Two of these states also stated concerns that the proposed requirements would be more difficult to meet while maintaining compliance with existing monitoring requirements such as biweekly quality assurance checks.

Response: The EPA compared annual data completeness rates calculated using the Appendix U requirements to annual data completeness rates calculated using the requirements under the previous O_3 standards across all U.S. monitoring sites based on data from 2011-2013 (Wells, 2015a). The national mean annual data completeness rate was 0.1% higher under the proposed Appendix U requirements than under the previous O_3 standards, and the median annual data completeness rates were identical. In addition, EPA notes that the Appendix U requirements allow for biweekly quality assurance checks and other routine maintenance to be performed between 5:00 AM and 9:00 AM local time without affecting data completeness. Therefore, EPA does not believe that the daily data completeness

requirements in Appendix U will be more difficult for monitoring agencies to meet.

(3) *Comment:* Two public commenters opposed the proposed procedures for determining daily maximum 8-hour concentrations. These commenters expressed similar concerns, primarily that not considering 8-hour periods starting midnight to 6:00 AM is less protective of public health than the procedure used to determine daily maximum 8-hour concentrations for the previous O_3 standards.

Response: EPA believes that the proposed approach, which we are adopting in the final rule, provides the appropriate degree of protection for public health. We note that the hourly concentrations starting midnight to 6:00 AM are covered under the 8-hour period from 11:00 PM to 7:00 AM, which is included in the design value calculations in Appendix U. This approach also ensures that individual hourly concentrations may not contribute to multiple exceedances of the NAAQS, which EPA believes is inappropriate given that people are only exposed once.

(4) *Comment:* This change is acceptable, however, it will require changes to the procedures currently in place to calculate the ozone design values and will also make comparisons to previous years DV's more difficult as the data handling protocol will have changed. The best option for this will be to only employ the new calculation procedures for the years post enactment of the standard and use those years/datasets for subsequent NAAQS comparisons.

Response: EPA notes that this procedure will be adopted only for the revised O_3 NAAQS. Design values for the 1997 8-hour O_3 NAAQS and the 2008 8-hour O_3 NAAQS will continue to be calculated according to Appendix I and Appendix P to 40 CFR Part 50, respectively. However, design values for the revised O_3 NAAQS based on data from years prior to the enactment of the standards will be calculated according to Appendix U to CFR Part 50.

(5) *Comment:* A consequence of the new procedures for determining daily maximum 8-hour average ozone concentrations is that this would create additional complexity to established calculation methods. The EPA needs to ensure that the new procedures for determining daily maximum 8-hour average ozone concentrations are properly coded into the EPA's AQS database in order for state, local, and tribal agencies to generate the resulting design values in an expeditious fashion. In addition, state, local, and tribal air agencies will need to devote additional resources to re-program established calculation methods to compute the 8-hour average ozone concentrations.

Response: After promulgation of the revised O_3 NAAQS, EPA will ensure that the new O_3 design value calculations in Appendix U are properly coded in AQS. Additionally, EPA will reach out to state, local, and tribal agencies to assist with their transition to the new data handling procedures in Appendix U.

(6) *Comment:* EPA also proposes that there must be 13 of 17 8-hour periods in a day in order to determine a valid daily maximum. EPA says that it is including this requirement because 13/17 is consistent with the 75% data completion requirement used for daily and annual NAAQS-related statistics. However, EPA proposes to keep the provision in 40 CFR § 50, Appendix P which says that a daily maximum 8-hour average is valid if it is greater than the NAAQS. *Id.* The Appendix P language is mandatory: "a day shall be also be counted as valid". 40 CFR. § 50, Appendix P 2.1. The preamble used an unclear term "allowing." We believe that the preamble was just lacking in precision and that the language in Appendix P 2.1 will remain mandatory. We support and believe the Clean Air Act mandates that any day with an 8-hour average above the level of the NAAQS must be included in calculating the design value.

Response: The existing language in Appendix P stating that "a day shall also be counted as a valid day if the daily maximum 8-hour average concentration for that day is greater than the level of the standard" has been retained in Appendix U and will remain mandatory under the revised O_3 NAAQS.

5. Exceptional Events Information Submission Schedule

EPA proposed exceptional events scheduling provisions in 40 CFR 50.14 that will apply to the submission of information supporting claimed exceptional events affecting pollutant data that is intended to be used in the initial area designations for any new or revised NAAQS. The new scheduling provisions will apply to initial area designations for the revised O_3 NAAQS.

(1) *Comment:* Several commenters (e.g., Nevada Division of Environmental Protection, South Carolina Department of Health and Environmental Control, Manufacturers Association of Florida, the American Fuel and Petrochemical Manufacturers) supported promulgating the data flagging and exceptional events demonstration submittal schedule as proposed. Other commenters noted that that the schedule by which agencies must flag ambient air data that they believe have been affected by exceptional events, submit initial descriptions of those events, and submit detailed justification to support the exclusion of those data from EPA-monitoring-based determinations of attainment or nonattainment with the primary and secondary O_3 NAAQS is insufficient. Commenters noted that the proposed schedule is particularly burdensome for agencies needing to submit exceptional events packages for the third year to be used in a 3-year design value (e.g., the EPA would presumably use 2014-2016 data for initial area designation decisions anticipated in October of 2017). Several commenters recommended that the EPA either establish no defined schedule for data flagging and exceptional events demonstration submittal or allow a minimum of 2 years from the setting of any new NAAQS for air agencies to provide a complete exceptional events demonstration. Agencies noting the difficulty associated with meeting the proposed schedule included, for example, the New Mexico Environment Department, Georgia Department of Natural Resources, the Minnesota Pollution Control Agency, the Minnesota Department of Health, the Ohio Environmental Protection Agency, the Texas Commission on Environmental Quality, the

Wisconsin Department of Natural Resources, the Wyoming Department of Environmental Quality, and several trade associations, planning organizations, and industry groups.

Response: The EPA has promulgated the data flagging and exceptional events documentation submission schedule as proposed. The EPA recognizes that the promulgated schedule is compressed, particularly for the third year of data to be used in a 3-year design value, and we will work cooperatively with air agencies to accommodate this scenario. Under the promulgated schedule and assuming initial area designation decisions in October 2017 for the revised O_3 NAAQS, affected air agencies would need to flag data, submit initial event descriptions and submit demonstrations for exceptional events occurring in 2016 by May 31, 2017. This schedule provides approximately 5 months between the EPA's receipt of the demonstration package and the expected date of designation decisions and approximately 1 month between the EPA's receipt of a package and the date by which the EPA must notify states and tribes of intended modifications to the Governors' recommendations for designations (*i.e.*, 120-day letters). Given the CAA requirement that the EPA follow a 2-year designations schedule, the EPA cannot remove submittal schedules entirely for data influenced by exceptional events or provide a minimum 2-year period from the setting of a new or revised NAAQS for documentation submittal. Neither of these options would ensure that the EPA has time to consider event-influenced data in initial area designation decisions. Rather, the EPA has promulgated an exceptional events data flagging and demonstration submission schedule that provides air agencies with the maximum amount of time available to prepare exceptional events demonstrations and still allows the EPA sufficient time to consider such exceptional events demonstrations in the designations process in advance of the date by which the EPA must send 120-day notification letters to states.

While for the third year of data anticipated to be used in a 3-year design value for the revised O_3 NAAQS, the promulgated schedule provides for demonstration submission 5 months after the end of the calendar year, the EPA expects that most submitting agencies will have additional time to prepare documentation as we expect the majority of potential O_3-related exceptional events to occur during the warmer months (*e.g.*, March through October). Also, as noted in the promulgated NAAQS, the EPA expects to propose and promulgate revisions to the Exceptional Events Rule in an upcoming notice and comment rulemaking effort anticipated in advance of the date by which Governors must submit their recommendations for the revised O_3 NAAQS.

6. Other Comments

(1) *Comment:* Three states requested that EPA change the reporting units for hourly O_3 concentration data from parts per million (ppm) to parts per billion (ppb). One state also requested that the hourly concentrations be reported with a precision of 0.1 ppb, instead of the current precision of 0.001 ppm or 1 ppb.

Response: Currently, states have the option of reporting hourly O_3 concentration data to the AQS database in either ppm or ppb. Concentration values reported in ppb are automatically converted to ppm in the database prior to computing design values and other summary statistics. Regarding the precision of reported hourly concentrations, states have the option to report data to AQS to whatever precision they feel is appropriate. However, EPA believes it is inappropriate to require a standard precision smaller than 0.001 ppm, since concentrations measured by O_3 FEMs are typically accurate to the nearest 0.001 ppm.

(2) *Comment:* Three commenters made comments referring to the measurement uncertainty in O_3 concentration data to EPA's acceptability criteria of +/-7% for one-point quality control checks. Two commenters stated that this uncertainty warrants returning to the two-digit rounding convention for design values used under the 1997 O_3 NAAQS (i.e., a 3-year average of 0.074 ppm would round to 0.07 ppm, which would meet the revised O_3 NAAQS). The third commenter argued that because of measurement uncertainty, areas with violating monitors that have design values within 7% of the NAAQS (i.e., 0.071 to 0.074 ppm) should be designated as "unclassifiable" rather than "nonattainment".

Response: EPA disagrees, noting that the three decimal digit reporting convention for design values in Appendix U is consistent with the convention established in Appendix P for the previous O_3 NAAQS. During the 2008 O_3 NAAQS review, EPA conducted an analysis which determined that the uncertainty in the design value metric due to measurement error was approximately 1 ppb (or 0.001 ppm; Camalier and Cox, 2006). EPA continues to believe that a precision of 0.001 ppm is appropriate for determining compliance with the NAAQS, and will continue to require design values to be calculated in ppm to three decimal places. Regarding the use of the "unclassifiable" designation, EPA does not believe it is appropriate to use this classification for any areas that have design values greater than the level of the NAAQS. Under section 107(d)(1)(A)(iii) of the Clean Air Act, the "unclassifiable" designation is reserved for areas that cannot be classified on the basis of available information as meeting or not meeting the NAAQS.

(3) *Comment:* Two commenters submitted similar comments that EPA's proposal in Appendix U would result in the unequal application of data completeness requirements. As proposed, any design value above the O_3 NAAQS would result in a nonattainment designation even if there are known data completeness issues. But if the design value is below the NAAQS, the area may not be designated in attainment if there are data completeness issues. All data should be required to meet the same requirements regardless if it supports a design value greater or less than the standard. EPA should revise the proposed Appendix U to state that "All design values must meet minimum data completeness requirements in order to be considered valid."

Response: EPA disagrees that the data completeness requirements need to be met in order for a monitor to show a violation of the O_3 NAAQS. Once a monitor has collected data showing that the 3-year average of the annual 4^{th} highest daily

maximum 8-hour concentration is greater than the NAAQS, it is impossible for collection of additional data to result in a lower design value. On the other hand, it is quite possible for collection of additional data at a monitor with a design value below the NAAQS to result in a violation. Therefore, EPA has set minimum data completeness requirements which must be met in order for a monitoring site to show that it attains the standards.

(4) *Comment:* One state commented that all "valid" 8-hour averages should be used in determining exceedances of the NAAQS, seasonal daily maximum 4th high 8-hour concentrations and design values. The daily maximum validity test should only be used in determining data completeness and should also include all valid 8-hour averages. If the daily data completeness is less than 18 of 24 valid hourly concentrations, the maximum 8-hr concentration for that day shall nevertheless be used in the computation of a valid design value for that site.

Response: EPA did not propose to change the long-standing requirement that daily maximum 8-hour average concentrations must meet the daily validity criteria in order be included in design value calculations. EPA believes that it is inappropriate for a day to be used in the calculation of the annual 4th highest daily maximum value, but not be counted toward the 75% annual and 90% 3-year average data completeness requirements.

(5) *Comment:* The proposed Section 2 in Appendix U of 40 CFR §50 outlines criteria for monitoring method, placement, quality assurance, and data submittal that must be adhered to before measurement data can be used for NAAQS comparison. The section also states that data "otherwise available to the EPA shall be used in design value calculations." The EPA needs to clarify that any data used to calculate a design value should also meet the same monitoring method, placement, and quality assurance requirements as data submitted to AQS, and that any other data "otherwise available to the EPA" only be used in design value calculations if the EPA provides that data to states by a specific date that allows for adequate time for states to refute its use or appropriately consider it in design value calculations. Without these restrictions, the EPA's discretion would be wholly unfettered and leave states without administrative or legal recourse to rebut the use of "other data" in design value calculations.

Response: Data "otherwise available to the EPA" must be verified to meet the same monitoring and quality assurance requirements as regulatory data submitted to the AQS database. In the event that such data were to be used in a regulatory action, EPA would provide notice of the intended use of this data as part of the proposal and public comment process.

(6) *Comment:* Referring to the Wells (2014b) technical memo cited in the preamble, one commenter submitted the following:

'Table 2 of the EPA memo notes that 8.3% of the 1,261 sites (i.e., 105 sites) had "overlapping" MDA8 values of "greater than 75 ppb" (the current NAAQS) for

any single year during the 2004-2013 period. The memo does not indicate that any "overlapping MDA8" from those 105 sites actually impacted any site's design value (DV). Table 2 also notes that 2.1% of the sites (i.e., 26 sites) had "overlapping" MDA8 of the current O_3 NAAQS on an annual basis. Having overlapping MDA8 values above the NAAQS every year certainly increases the likelihood that the DV will be impacted because in order to exceed the O_3 NAAQS a site must record a 4th high MDA8 for three consecutive years. EPA should note the number of occurrences in which overlapping MDA8 values actually caused an increase in a site's DV.

The report notes that "double counting" is more likely in sparsely populated rural areas without the NO titration protection from elevated nighttime O_3 afforded to urban areas by mobile source emissions. This likelihood also increases as the O_3 NAAQS level is lowered. The report should note also that many of the rural area O_3 levels are monitored at CASTNet sites where 10 meter monitor inlet heights above ground level are mandated, in contrast to the lower 2-15 meter heights, typically 4-6 meters, allowed at urban compliance sites. This height difference is important given recent CASTNet evidence that measured O_3 levels increase with inlet height above 2 meters, a height below which most outdoor human O_3 exposure occurs. The higher rural O_3 monitor inlets likely result from the fact that CASTNet locations were not originally designed as O_3 compliance sites and only a recent EPA decision has made them so, beginning with the 2011 ozone season.'

Response: Regarding the comment on design values, EPA has extended the analysis in Wells (2014b) to include an assessment of the impact of overlapping MDA8 values on O_3 design values. In the extended analysis, EPA compared design values from eight consecutive 3-year periods (2004-2006 to 2011-2013) using the data handling criteria in Appendix U to 40 CFR Part 50 to the previous criteria in Appendix P. The results of the extended analysis are presented in a technical memo to the docket (Wells, 2015a).

Regarding the comment on CASTNet monitoring, in citing "recent CASTNet evidence that measured O_3 levels increase with inlet height above 2 meters," the commenter is apparently referring to data collected during a short-term study at the Howland Research Forest AmeriFlux site in Howland, Maine. This unpublished study was specifically designed to determine the feasibility of measuring nitrogen, sulfur, and ozone deposition within a tree canopy using low-frequency measurements and an instrument configuration that is quite different from the standard CASTNet configuration for long-term measurements. The siting criteria outlined in Appendix E to 40 CFR Part 58 excludes the Howland study and any other monitor located within a tree canopy for use in regulatory determinations because of the influence of vegetation on measurements. Because this study is not representative of conditions at regulatory monitors, it is inappropriate to extrapolate results from the study to claim that O_3 levels increase with inlet height above 2 meters at other CASTNet monitors. CASTNet monitors were originally designed to measure representative regional atmospheric conditions with minimal influence from local emission sources. An inlet height of

10 meters was chosen to minimize the transient influences from vegetation or other surface characteristics that are more apparent at lower inlet heights.

(7) *Comment:* The language of the NAAQS should not be limited to monitoring sites; the language needs to be broad enough to take into account the potential use of modeling for evaluating attainment, which should utilize a full receptor grid reflecting the fact the NAAQS are National Ambient Air Quality Standards.

Response: EPA does not believe it is appropriate to change the long-standing requirement that comparisons with the O_3 NAAQS be based on quality assured and certified data collected at ambient air quality monitoring sites in accordance with 40 CFR Part 58. Although the EPA considers other forms of information for purposes of evaluating areas with sources that contribute to monitored violations for inclusion within the nonattainment area boundaries, the fundamental basis for designating an area as nonattainment for the O_3 NAAQS is the presence of one or more FRM or FEM monitors with data showing violations of the NAAQS. Model predictions in unmonitored areas are more uncertain due to the lack of monitors with which to evaluate or validate the model results.

(8) *Comment:* EPA needs to create a methodology that fills in missing data, similar to 40 CFR. § Part 75 of the Acid Rain program. Without this, the NAAQS cannot ensure the protection that EPA says it is choosing to provide. The current and proposed form and averaging time do not make adjustments for missing data. However, the old one-hour average did. While there is a minimum number of days that monitoring sites are required to collect, there is no consequence if the monitoring site fails to meet this standard. In any event, because the minimum standard is not 100% of the required days or hours, people can be and are exposed to significantly more short term periods about the level that EPA says is the appropriate level. EPA should add a data filling mechanism based on protective assumptions.

Response: Compliance with the 1-hour O_3 NAAQS was based on "expected exceedances", or the number of observed exceedances adjusted for missing data. However, design values for the 8-hour O_3 NAAQS are concentration based, and EPA believes it is inappropriate to adjust concentration-based design values for missing data, because it is physically impossible for missing data values to affect concentrations on days with complete monitoring data. In general, EPA does not support the use of interpolation or other means of filling in missing monitoring data for O_3 NAAQS comparisons. Such an approach would be contrary to EPA's long-standing policy of using only quality-assured and certified ambient air quality measurement data to determine compliance with the O_3 NAAQS.

(9) *Comment:* EPA's proposed data handling convention would require that any decimal digits reported beyond three decimal digits will be truncated. EPA's stated reasons for this are (1) consistency with past practice and (2) typical measurement uncertainty. EPA must, at a minimum round the third decimal place rather than truncate. Past practices do not provide a rational basis to truncate in

this context because monitoring equipment has changed over the decades since EPA started truncating. As to measurement uncertainty, truncating and rounding both address this uncertainty. However, rounding is more consistent with Congress' clearly expressed will that NAAQS be addressed in a precautionary manner.

Response: EPA did not propose to change the long-standing convention of truncating decimal digits beyond the third decimal place in the calculation of 8-hour average O_3 concentrations and 3-year average design values. In addition, EPA notes that the analyses of ambient O_3 concentration data provided in the HREA and PA documents, as well as the risk and exposure estimates provided in the HREA document were based on truncated O_3 concentration values. Thus, the existing truncation procedure is already implicitly accounted for in the Administrator's decision regarding the level of the revised O_3 standards.

D. Ambient Monitoring Related to the Proposed O_3 Standards

Public comments on ambient monitoring related topics (O_3 monitoring seasons, PAMS requirements, the new FRM and revisions to the FEM testing requirements) are addressed in the preamble to the final rule or in this document. Other comments on monitoring that were not specifically related to O_3 seasons, PAMS, or the FRM/FEM are also included below.

(10) *Comment*: An analysis of the difference in the monitored concentrations from collocated ozone monitors at Rocky Mountain National Park, which are part of the CASTNET monitoring network, was conducted. These sites are operated by different contractors and were the subject of an EPA report examining the persistent bias between the sites, which was very large. EPA and the NPS attempted to bring the monitors into closer agreement through a thorough QA/QC process, but were unable to identify what caused the differences in monitored concentrations and resolution was reached by replacing one monitor which eliminated the difference between the monitors. While the problem has been resolved, the cause of the discrepancy was never identified. This situation raises concerns regarding ozone monitoring in general, because these two monitors were both operated using EPA QA/QC procedures, with no identified issues with the data, yet there was a large difference in monitored levels. This difference points to a large, unidentified uncertainty in the monitoring network that has the potential to cause uncertainty in attainment designations.

Response: The EPA appreciates the comment. The EPA does not agree that the discrepancy between the collocated monitors at Rocky Mountain National Park is indicative of the uncertainty across the entire ozone monitoring network. The CASTNET monitoring network staff identified some operational and procedural differences between these independently operated sites, implemented additional data validation steps to their data review process, and updated the procedures for on-site through-the-probe (TTP) calibrations when using transfer standard instruments that can experience pressure imbalances between the detector cells. Most CASTNET monitors are calibrated using transfer standards with an internal scrubber and are not affected by this potential issue. It was noted that when a TTP calibration was performed using a transfer standard with a pressure imbalance between the detector cells, the monitor was biased high. As a result, the CASTNET standard operating procedures (SOPs) were updated to indicate that when a multi-cell transfer standard is used for a TTP calibration, the pressure imbalance must be minimized to avoid calibration bias. Biases between the CASTNET monitors have been less than one percent of the instrument calibration range since the SOPs were revised. The recommended instrument calibration criterion is two percent.

(11) *Comment*: The commenter provided a study of precision and bias. The study of the precision of a number of instruments shows that while the precision of individual analyzers, as well as the group, was quite acceptable, a small percentage of measurements (e. g. 5%) can show significant errors. An analysis was done both for individual monitors and for all monitors collectively. The

precision data indicate the following: 1) In general, the precision of the individual monitors is very similar and indicates that none of the monitors are outliers; 2) the mean difference is close to zero and implies the precision error is normally distributed; 3) for the combined precision data, the standard deviation of the error is approximately +/- 2 ppb. This means that 68% of the errors lie within that range. However, this also suggests that, 32% of the time, the error in ozone precision (reproducibility) is in excess of +/- 2 ppb; and 4) at a level of two standard deviations from the mean (95% of the data), the precision difference is in excess of +/- 4 ppb and the overall range is -6 to 7 ppb (13 ppb range). The study of accuracy bias in field monitors showed that current ozone monitoring data are accurate and reproducible, but uncertainty errors may be significant for low-level standards. For areas at the threshold of non-attainment, the uncertainty inherent in the monitoring data may lead to misclassifications of non-attainment areas. The commenter presents the results of an analysis in terms of bias in the data for all monitors collectively. The analysis indicates that, although current ozone monitoring data are accurate and reproducible, there are limitations in the data that must be considered in policy assessments.

Response: We are confident, based on quality assurance (QA) and quality control (QC) requirements in 40 CFR part 58, Appendix A and data quality assessments, that the vast majority of ozone monitoring data collected nationwide are suitable for attainment/nonattainment determinations. There are uncertainties associated with the UV ozone method, as there are with other methods for measuring air quality. The EPA evaluated hourly precision from collocated monitors in at two sites in Missouri as part of the ISA and the precision from biweekly single point QC checks. The average concentration measured at the two sites in Missouri was 34 ppb and the average concentration of the single point QC checks was 90 ppb. The mean relative percent difference between the collocated monitors in Missouri and the single point QC check data from all ozone sites were less than 1 percent. The EPA operates collocated monitors at its on-site monitoring station in RTP and the data from the two monitors for June – July 2015 shows an average of less than 1 ppb absolute difference between them. A memo to the docket (Camalier and Cox, 2006) evaluated the effect of measurement error on the 8-hour ozone design value concentrations. Values of daily bias were assumed to arise from a normal distribution (as noted in the comment above) with zero mean and (no average bias) and standard deviation of ±4 ppb. The ±4 ppb was used as a reasonable estimate of the maximum hourly measurement bias expected from instruments operating under routine conditions. Results from the Camalier and Cox analysis indicate that measurement bias could contribute approximately 1 ppb to the 8-hour design value uncertainty.

(12) *Comment:* One environmental group comment states that a lack of ozone monitors in an areas of oil and gas exploration is contrary to EO 12898. "Inadequate monitoring in the Eagle Ford shale play is not only a problem for the 10.5 million citizens of the four major metropolitan areas surrounding it. The 23-county area that the Eagle Ford shale underlies has a population of 907,844 on its own. The ozone pollution issues in these rural areas raise not only public health

concerns, but also environmental justice concerns. 54.72% of the residents of the 23-county Eagle Ford area are Hispanic. In Webb, Zavala, and Maverick Counties, 93% of the population is Hispanic. Under Executive Order 12898, EPA, as a federal agency, must "collect, maintain, and analyze information assessing and comparing environmental and human health risks borne by populations identified by race, national origin, or income." The near-complete lack of ozone monitoring data in the 20,000- square-mile, majority-Hispanic Eagle Ford shale region constitutes a failure to address the requirements of E.O. 12898. Until adequate monitoring data is available, it will be impossible to know how ozone pollution from oil and gas drilling is affecting public health in the region.

Response: We require that monitoring agencies review the monitoring network on an annual basis to ensure that the network is adequate and representative of the entire area, and to document any changes in their annual monitoring network plans. The monitoring agencies should be factoring the growth of oil and gas development in their area in determining the appropriate distributions of monitors in their state. It would be important for the commenter to work with the state in the monitoring network review process to convey their concerns. The EPA sets standards to protect all populations even the most sensitive populations.

1. Comments on Revisions to the Length of the Required O_3 Monitoring Seasons

(1) *Comment:* The EPA analysis (Rice memo Nov 19, 2014) of high "off season" O_3 concentrations confirms an earlier memo to the docket (Camalier and Weinstock, 2008) which determined that elevated (i.e. > 60ppb MDA8) O_3 concentrations were being recorded outside the normal O_3 "season" in many areas of the country. The EPA should expand their recent analysis to include an analysis of stratospheric intrusions and other exceptional events since occurrences of high ozone concentrations over multi-State regions on several dates were demonstrated in that monitoring season analysis.

Response: The data retrieved from AQS (2010-2013) for the ozone season analysis excluded regionally-concurred exceptional events. Basing O_3 monitoring season requirements on the goal of ensuring monitoring when ambient O_3 levels approach or exceed the level of the NAAQS supports established monitoring network objectives described in Appendix D of Part 58, including the requirement to provide air pollution data to the general public in a timely manner and to support comparisons of an area's air pollution levels against the NAAQS.

(2) *Comment:* No changes to the ozone season were proposed for Nevada which is currently January through December. However, the Nevada DEP was granted a waiver to adjust the monitoring season to April through October. Based on the proposed preamble language, the existing waiver would be revoked. The Nevada DEP would need to request a new waiver. The Nevada DEP is requesting that a new waiver be granted for the April through October ozone monitoring season. If waiver is not approved, conducting year-round monitoring would add additional costs and resource needs to the Nevada DEP's monitoring program, including

more quality controls and more data entry into the AQS, without any additional funding from the EPA.

Response: When the final rule becomes effective 60 days after publication, existing waivers are revoked. It is beyond the scope of this rulemaking to grant specific waivers to any particular states. Waivers must be discussed with and requested from the relevant EPA Regional Office, in light of the requirements finalized in paragraph 4.1(i) of 40 CFR part 58, Appendix D.

(3) *Comment:* Some commenters noted that monitoring should not be required during the cooler months (e.g., March) due to the relationship between warm seasons only and health effects.

Response: Within the U.S. the monitoring of ozone concentrations varies across cities with some cities monitoring O_3 year round and others only during the warm season when O_3 concentrations are known to be higher. As a result, epidemiologic studies either use all data from every city or only focus on examining the relationship between O_3 exposures and health effects during the warm season. Analyses using all-year O_3 concentrations demonstrate consistent positive associations between short-term O_3 exposures and health effects, such as respiratory-related hospital admissions and emergency department visits, though associations are often stronger and larger in magnitude during the warm season (ISA, section 6.2.7.5). Based on the epidemiologic evidence for health effect associations using all-year O_3 concentrations, and based on effects shown in controlled human exposure studies (i.e., which are relevant for consideration regardless of the season), the EPA disagrees with the commenters that O_3-realted health effects only occur during the warm season resulting in the elimination of the need for monitoring during the cold season.

(4) *Comment:* One state commenter noted that the "U.S. EPA's Cross State Air Pollution Rule, Clean Air Interstate Rule, and NO_x SIP Call imposes requirements on sources based on a cap and trade program during the ozone season. Under these programs the ozone season runs from May 1st to September 30th. It is imperative that any changes made under this proposal to a state's ozone season do not impact the compliance periods under these rules." One industry group requested that EPA more clearly articulate the potential regulatory impacts of proposed ozone season changes on current and future interstate transport rules.

Response: The ozone season, as defined in the transport rule, is simply based on the time of year when ozone is typically highest in the East. We therefore need specific emissions controls to reduce ozone transport during that time of year. This is accomplished by setting a statewide NO_x emissions budget for the May-September period. This is different than the purpose of the monitoring ozone season. The monitoring season is based on the time of year when monitors in a state could potentially exceed the level of the NAAQS. All states affected by these rules have monitoring seasons longer than the May-September period. Any future transport rules for the 2015 NAAQS will consider the appropriate ozone

season for transport based on the location of the nonattainment problems and the level of the NAAQS.

(5) *Comment:* One industry commented that, "If a larger portion of the year is considered important for health impacts, the difference between integrated background ozone and integrated ambient ozone become smaller – *i.e.*, background is a larger component of cooler-season ozone. EPA has presented background ozone data for the May to September time period, but these fractions are not representative of a full year. EPA needs to take this difference into account in the promulgation of the monitoring season".

Response: The ozone season lengths help to ensure that we are monitoring in the months when we expect ozone concentrations to approach or exceed the level of the NAAQS. Background ozone considerations are not relevant to the question of the appropriate ozone monitoring season.

(6) *Comment:* One state commented that, "Before implementing the new ozone season proposal, EPA should more closely consider multi-state nonattainment areas and areas where upwind states significantly contribute to downwind nonattainment (under Good Neighbor provisions) to insure contributing emissions are properly controlled during downwind state ozone seasons".

Response: The EPA believes it is important that O_3 monitors operate during periods when there is a reasonable likelihood of ambient levels approaching the level of the NAAQS irrespective of the sources contributing to ambient O_3 concentrations. Basing O_3 monitoring season requirements on the goal of ensuring monitoring when ambient O_3 levels approach or exceed the level of the NAAQS supports established monitoring network objectives described in Appendix D of Part 58. While we appreciate that considering upwind contributors is relevant when designating nonattainment areas or developing attainment strategies, we have concluded that the revisions to the monitoring season are justified based on the available monitoring data and the level of the revised standard and that implementation issues related to interstate transport of O_3 do not provide a basis for delaying these revisions.

(7) *Comment:* Some commenters support an extended monitoring season for the secondary standard. Two commenters wanted the O_3 seasons to reflect regional seasonal differences in the growing season. Photosynthesis in conifers and early emerging forest floor species begins before deciduous canopy leaf-out which should be considered in setting the length of the monitoring season. Further, the growing season can vary greatly across the U.S. EPA also must account for the extended timing of elevated ozone concentrations in the context of climate change. The increase in seasons should also be consistent across regions with the same growing seasons. This might be evaluated using the USDA hardiness growing zones.

Response: The EPA appreciates the comment. Given that an important objective of ozone surveillance monitoring is identification of areas where there is a potential for the NAAQS to be exceeded, our primary consideration in reviewing the appropriate length for the monitoring season at sites across the U.S. was whether the available data indicate the likelihood of O_3 concentrations with the potential to contribute to an exceedance. Accordingly, we have based the ozone season length on the number of days that were ≥0.060 ppm in the months outside a state's current O_3 season. This threshold serves as an appropriate indicator of ambient conditions that may be conducive to the formation of O_3 concentrations that approach or exceed the level of the NAAQS. While more extensive data collection may be useful for other purposes, such as future welfare or health assessments, they are not the primary objective of the required ozone monitoring network. Accordingly, we did not evaluate or propose extending the monitoring seasons based on growing seasons or climate change.

(8) Comment: One commenter was "concerned that EPA is not taking the necessary steps to ensure that monitoring will be adequate to effectively implement any new secondary standard. EPA has long acknowledged that uncertainties will remain about ozone concentrations affecting sensitive natural vegetation and ecosystems until additional monitors are sited in National Parks wilderness areas and other public lands. Yet EPA does not propose to address these concerns. EPA should identify monitoring needs in parallel to finalizing this proposal. EPA has the information necessary to identify ecosystems of concern for impacts from ozone and plan an appropriate distribution of monitors. This information should be used to outline the monitoring that will be required to protect these areas. Moreover, while additional monitors are of great importance it is critical that existing monitors be maintained. Funding cuts in recent years have led to the removal of important monitors. Monitored data is the lifeblood of NAAQS and EPA should ensure that funding for monitors be a priority for the agency."

Response: The EPA appreciates the comment; however, we did not propose any expansion of the existing ozone monitoring network. We note that there are about 120 non-urban ozone monitors operated by the National Park Service, CASTNET, and at rural NCore sites (ISA, Section 3.5.6.1).

(9) *Comment:* One environmental group commented that, "EPA's proposal does not go far enough. EPA is trying to set the ozone season to monitor when conditions are conducive to ozone formation. EPA tries to do this by looking at data from monitors which were operated outside of their ozone season. But, this approach almost certainly misses many situations in which ozone exceedances occur outside the traditional ozone season because approximately 700 monitors do not operate year round. In addition, EPA's current methods for determining what conditions are conducive to ozone formation are overly conservative. Climate change is also likely to increase the length and severity of ozone seasons. To assure more complete identification of the periods in which ozone exceedances occur, EPA needs to require all monitors to operate year round."

Response: The EPA's proposal did include a requirement for the NCore multipollutant monitoring sites to operate year-round regardless of the state's required O_3 season. The NCore has about 80 monitoring locations across the U.S. The EPA based the O_3 season proposal on the number of days that were ≥0.060 ppm at monitors that were operating outside their current monitoring season, including monitors that were operating year-round. This threshold serves as an appropriate indicator of ambient conditions that may be conducive to the formation of O_3 concentrations that approach or exceed the level of the NAAQS. The proposal was based on the all available data in AQS, including monitors operating outside the required O_3 season. In states where monitors were operating year-round, the data did not support monitoring year-round.

(10) *Comment:* One commenter called for further expansion of the ozone monitoring network in addition to year-round monitoring at all sites and that the number and locations of ozone monitors are inadequate to capture ozone levels in many areas undergoing heavy oil and gas development.

Response: EPA did not propose a further expansion of the ozone monitoring network. In regards to the comment on all sites operating on a year-round schedule: The proposal was based on the all available data in AQS, including monitors operating outside the required O_3 season. The EPA's analysis found that the number of days that were ≥0.060 ppm did not support the requirement for all ozone monitors in all states and the District of Columbia to operate year-round. In states where monitors were operating year-round, the data did not support monitoring year-round. Monitors operating in areas undergoing oil and gas development were included in this analysis. Many of the monitors in oil and gas development areas (e.g., Wyoming, North Dakota, South Dakota, and Montana) are already operate a large percentage (71% in Wyoming, 89% in North Dakota, 100% in South Dakota, and 78% in Montana) of their monitors year-round.

(11) *Comment:* One environmental group commented that, "the regulations cannot allow the Regional Administrators to change the regulations regarding ozone season without notice and comment. This would be an APA and/or CAA violation. If an ozone season is changed without public notice, people may assume that ozone levels are safe when in fact they are not safe but there is no monitoring being conducted to report ozone levels to the public. Note, however, that we do fully support revocation of previous Regional Administrator-granted approvals".

Response: The commenter does not explain why the process is inconsistent with the APA and/or CAA. The current ambient monitoring rule, in paragraph 4.1(i) of 40 CFR part 58, Appendix D (71 FR 61319, October 17, 2006), allows the EPA Regional Administrators to approve changes to the O_3 monitoring season without rulemaking. The EPA is retaining the rule language allowing such deviations from the required O_3 monitoring seasons to be approved by the EPA Regional Administrator without rulemaking. Any modifications to the O_3 monitoring

network, include a waiver or deviation from the required O_3 season, per 40 CFR Subpart B §58.10 would require the opportunity for public comment.

(12) *Comment:* One industry group commented that EPA cannot justify extending Florida's ozone monitoring season to year-round when its analysis found that there were no days that were ≥ 0.060 ppm in December.

Response: Florida operated 95% (59 of 62) of the ozone monitors in the state year-round in 2010-2013. In the EPA's analysis, a year-round monitor was identified as "year-round" if it had at least 20 daily observations in all 12 months, for at least 1 year of the 4 year period (2010-2013). Shutting 62 ozone monitors down for one month of the year could add more burden on the state monitoring agency. Ozone monitors are automated, as well as the QA/QC checks, and keeping the monitors operating for one additional month adds relatively small incremental cost/burden especially since Florida is already operating 95% of the monitors year-round.

(13) *Comment:* One industry group commenter stated that, "If EPA extends the current monitoring season as proposed, EPA should also revise the form of the standard from the 4th highest daily maximum 8-hour average to the 8th highest daily maximum 8-hour standard."

Response: This commenter suggest that if EPA adopts a longer monitoring season, then EPA should revise the form of the standard to allow for more exceedances of the level of the standard without causing a violation of the standard. In effect, commenters are suggesting that EPA adopt a percentile approach (where having more days with monitoring data allows for more exceedances of the levels) rather than an nth-high approach (where a fixed number of exceedances is allowed, regardless of the length of the monitoring season) as the form of the standard. As discussed in the preamble, EPA considered a percentile-based statistic for the form of the standard, but concluded (as in past reviews) that using a 4th-high statistic for the form is more appropriate because it more effectively ensures that people who live in areas with different length monitoring seasons receive the same degree of public health protection.

(14) *Comment:* One industry group commented that, "EPA's cost estimate only focuses on monitoring costs and not costs associated with areas that may have limited monitored exceedances outside traditional ozone season that would now either be moved into non-attainment status or chained to it.

Response: The purpose of cost estimates is to estimate the cost of information collection. The fact that the information collected may ultimately result in a determination that an area is not attaining a standard or contributing to a nearby area not attaining the standard does not change EPA's estimates of what it costs to collect the information. EPA's goal in estimating costs is to support a judgment whether the proposed monitoring changes are worthwhile as part of EPA's ongoing obligations to assess, as accurately as possible, taking into account costs,

the air quality of the entire country, to support the development of control strategies, and to inform the public of air quality.

(15) *Comment:* Some industry groups commented that, "Extending ozone seasons could lead to increased number of NAAs, which EPA does not appear to have analyzed in its proposal or RIA. EPA ignores potential impact of proposed longer seasons. Implications of an area failing the standard are substantial". And that, "EPA's cost estimate only focuses on monitoring costs and not costs associated with areas that may have limited monitored exceedances outside traditional ozone season that would now either be moved into non-attainment status or chained to it".

Response: As noted in the proposed and final rulemaking, basing O_3 monitoring season requirements on the goal of ensuring monitoring when ambient O_3 levels approach or exceed the level of the NAAQS supports established monitoring network objectives described in Appendix D of Part 58, including the requirement to provide air pollution data to the general public in a timely manner and to support comparisons of an area's air pollution levels to the NAAQS. Thus, the goal is to accurately identify areas that may be exceeding the NAAQS in order to enable areas to take action to protect public health. The increase in the number of nonattainment areas was not relevant to the analysis of whether a state's season should be extended. Costs and benefits related to potential nonattainment areas were presented in the RIA (2014). Regarding the RIA, the results of the RIA were not considered in EPA's decisions on the O_3 seasons.

(16) *Comment:* One industry group commented, that winter time temperature inversions should be considered exceptional events, especially if we are subject to an extended season that includes the winter months.

Response: The CAA specifically excludes stagnation of air masses or meteorological inversions from being considered as exceptional events. *See* 42 U.S.C. § 7619(b)(1)(B).

(17) *Comment:* One commenter stated that an "Increase of 1 month in Northern Texas would impact the amount of resources required to re-educate the public and develop new marketing and outreach materials.

Response: We acknowledge that the commenter may need to adjust their outreach materials to add one month. However, we expect that the commenter is reporting the AQI and providing public outreach for ozone and $PM_{2.5}$, which is an annual standard instead of a seasonal one. We expect that the impact on the resources for re-education, marketing, and outreach would be minimal. The new ozone season will not become effective until January 1, 2017 which gives the commenter some time to adjust their outreach materials.

2. Comments on Revisions to the Photochemical Assessment Monitoring Stations (PAMS)

a. *Network Design*

(1) *Comment:* Two commenters stated that due to the proposed compliance schedule, data from the revised PAMS network "will play a minor role in ozone standard attainment in most areas of the US." As such, they also state that "EPA should seek to minimize the costs associated with the PAMS program and seek the smallest PAMS network practicable."

Response: The EPA agrees that due to the timing of when the network changes will occur, the data from the revised network will likely not be available for states as they evaluate control options for attainment of the revised NAAQS. However, the intent of the current network changes are to improve the network for future NAAQS reviews and model development and evaluation efforts beyond the immediate review cycle.

(2) *Comment:* Two commenters suggested "EPA should consider a tiered approach to PAMS monitoring" that places PAMS sites at NCore sites in existing ozone non-attainment areas that have CBSA populations of 1,000,000 or more" and in "non-attainment areas that measure MDA8 values 15% or more above the NAAQS."

Response: The EPA considered this comment in developing the final network requirements. The final requirements reflect a number of the elements suggested in this comment. For the reasons discussed in the preamble, EPA has finalized requirements for fixed PAMS sites will be required in CBSAs with a population of 1,000,000 or more (regardless of attainment status), and Enhanced Monitoring Plans (EMPs) will only be required in moderate or above ozone non-attainment areas.

(3) *Comment:* One commenter suggested that "EPA should ensure the goals of the PAMS network are met through this proposal and give further thought to whether such a resource-intensive network design accomplishes those goals."

Response: As discussed in the preamble, the EPA evaluated the historical objectives of the PAMS program and attempted to determine which of the PAMS objectives were being met. The proposed and final requirements were developed based on recognizing that the existing network design was overly resource-intensive and therefore limited the local monitoring agencies abilities to explore alternative monitoring data collection efforts. The final network design requirements dramatically reduce the cost of the fixed PAMS network freeing up resources for monitoring agencies to use in developing and implementing their EMPs.

(4) *Comment:* One commenter stated that "in order to function properly, a PAMS network requires an upwind/background site to determine background and

transported ozone, two maximum emissions impacts sites, a maximum ozone concentration site, and an extreme downwind ozone site."

Response: The EPA agrees that in some locations upwind and downwind data are important to understand ozone formation and transport. However, the EPA believes the EMP will allow monitoring agencies to identify more cost effective methods of collecting necessary upwind and downwind data than the existing fixed site PAMS requirements.

(5) *Comment:* One commenter suggested that "EPA permit Regional Planning Organizations (RPOs) to coordinate Enhanced Monitoring Plans for monitoring agencies that desire to coordinate their monitoring efforts."

Response: The EPA agrees that RPOs could play an important role in assisting and coordinating EMPs and strongly encourages monitoring agencies to consider working with RPOs and EPA as they develop their EMPs. However, the EPA does not believe it is appropriate to require a state to work with RPOs, and as such, has not incorporated any regulatory language to that effect.

(6) *Comment:* One commenter suggested that "additional considerations for retaining or shutting down sites should include continuing data collection from sites with a long history of trend data, the start-up time required for new PAMS site and the need to site PAMS both upwind and downwind of metropolitan areas."

Response: The EPA agrees that these considerations should be taken into account as monitoring agencies evaluate alternative monitoring locations to meet the revised fixed site network requirements. The final waiver provision for alternative locations has been broadened to allow for alternative locations that may be outside of the CBSA or even the state. The EPA believes that the final requirements will allow for more historical sites to be continued as alternatives to the newly required fixed PAMS sites.

(7) *Comment:* One commenter recommended that EPA work with states to refine the network adding that some states may want to divest while others may not. The commenter suggested that since ozone is a regional pollutant, states and EPA regional offices should be encouraged to develop a regional plan to refine the PAMS network in consideration of EPA objectives and the strengths and capabilities of each state involved.

Response: The EPA agrees that the states should work with EPA in developing regionally appropriate plans that take into account such considerations as historical participation in the PAMS program and interest and ability to continue participation. As noted above, the final waiver provision for alternative locations has been broadened to allow for alternative locations that may be outside of the CBSA or even the state.

(8) *Comment:* One commenter stated it was unclear if the full suite of pollutant monitoring required at existing NCore sites would be required in each new

nonattainment area or how many monitoring sites are sufficient to adequately understand ozone formation.

Response: The final requirements intentionally do not specify minimum monitoring requirements for the EMPs, recognizing that monitoring agencies are in a better position to identify the data needed to understand ozone formation in their area. However, the EPA does not expect each EMP site to be a full suite of PAMS measurements.

(9) *Comment:* One commenter stated that, while they agreed that attainment status along with population should determine applicable monitoring requirements for an area, the preamble did not discuss the potential population thresholds that would apply.

Response: The preamble to the proposed rule requested comments on alternative frameworks for the fixed PAMS network requirement which could include attainment status and population thresholds. The preamble also referenced an EPA memorandum to the Ozone NAAQS docket entitled "Network Design Considerations for the PAMS Network" that discussed several options including attainment status and population thresholds between 250,000 and 1,000,000. For the reasons discussed in the preamble, EPA finalized a requirement for PAMS monitors that includes a population threshold of 1,000,000.

(10) *Comment:* One commenter questioned if the monitoring performed as part of the EMPs would be "as immoveable as monitors that have historically determined compliance with the current ozone NAAQS, and stated that the monitors should be considered special purpose monitors and that states should be provided explicit ability to move or decommission monitors, including those that may have historically been used for compliance purposes.

Response: The final requirements do not include any limitations on moving or closing sites as part of the EMP. However, criteria pollutant monitoring (O_3 or NO_2) conducted as part of the EMP would be subject to the requirements of 40 CFR part 58.14(c)(1) unless the sites were also identified as a special purpose monitor and were operated under the conditions of 40 CFR part 58.20.

(11) *Comment:* One commenter stated that the proposed changes would likely not save states money and resources because each new nonattainment area would need a network of PAMS monitors to understand the particulars of ozone formation chemistry and regional transportation and that the EPA's proposed plan oversimplifies the monitoring necessary to understand ozone formation.

Response: Neither the proposed or final requirements call for a network of PAMS monitors in every ozone nonattainment area. The final requirements reduce the total number of required fixed PAMS sites to approximately 40 sites, with the sites being predominately located at existing multi-pollutant NCore sites. In addition to the fixed sites, states with moderate ozone nonattainment areas are

required to develop an area specific EMP. The EMP may include additional PAMS measurements in areas where upwind and downwind data are desirable, however, a full suite of PAMS measurements would not necessarily be required at each EMP site.

(12) *Comment:* One commenter stated that they believed that the requirement to develop an EMP in ozone nonattainment areas that do not have an NCore site places a significant undue burden on the state and its resources.

Response: The EMP requirements have been reduced such that they only apply to areas that are designated as moderate O_3 nonattainment or above. The EPA made this change from the proposed rule recognizing that most marginal O_3 nonattainment areas are expected to attain the NAAQS without additional state directed controls. Those areas required to develop and implement an EMP will be provided grant funding as part of EPA's section 105 grants.

(13) *Comment:* One commenter suggested EPA allow consolidation of Enhanced Monitoring Plans for adjacent nonattainment areas of similar type.

Response: The EMP requirements are at the state level. States with multiple moderate O_3 nonattainment areas can develop a single EMP for the entire state. States are also encouraged to coordinate EMPs as a means of improving information on regional ozone issues.

b. ***VOC Measurements***

(1) *Comment:* A number of commenters raised concerns that the addition of PAMS measurements would require states to run research grade equipment that they felt would detract from their compliance monitoring activities.

Response: AutoGCs have been used in the PAMS program since the mid-1990s. Many improvements have been made to the hardware and software since. As such, the EPA believes autoGCs are not research grade equipment and are appropriate for continued use in the PAMS network.

(2) *Comment:* One commenter suggested that EPA develop procedures that would ensure that the auto-GCs collect the full TO-15 suite of air toxic VOCs in addition to the PAMS suite. Another commenter stated that the required target list should be consistent with the capabilities of the instrumentation.

Response: While the EPA agrees it is a worthwhile goal to develop procedures for the auto-GCs to be able to collect the full TO-15 suite, it is uncertain if improvements can be made that would allow for the full suite of compounds. Due to moisture control requirements, polar compounds may not be measurable by auto-GCs. In addition, many air toxic VOCs may be present at concentrations too low to measure with auto-GCs. The EPA has recently completed an exercise to identify priority compounds for measurement at the PAMS sites. The priority list includes a number of compounds that were identified as important hazardous air

pollutants (HAPs). A number of candidate compounds were identified for further consideration, but, to date, these compounds have not been added to the PAMS target list due to the issues discussed above.

(3) *Comment:* One commenter stated that EPA should not assume that NCore operators will have the skills necessary to operate an autoGC and that autoGCs require significantly more skill and training to operate and maintain than do the continuous analyzers typically used at NCore sites. Several other commenters noted the need for training on autoGCs.

Response: While the EPA believes that modern autoGCs can be operated by typical monitoring staff, we agree training in the operation of specific instruments, data acquisition, and data validation will be necessary to ensure successful deployment of autoGCs at the required PAMS sites. The EPA commits to assist in the development and production of training materials and courses to assist monitoring agencies as they develop the necessary skills and knowledge.

(4) *Comment:* One commenter suggested that canister sampling be allowed until staff are properly trained with the equipment and data are of sufficient quality to support hourly measurements.

Response: The final rule requirements allow for the use of canisters in limited situations. The EPA agrees that in some places it may be appropriate to begin sampling with canisters and then move to autoGCs as the necessary skills are developed or acquired.

c. ***Carbonyl Measurements***

(1) *Comment:* A number of commenters suggested that EPA delay the addition of carbonyl sampling at PAMS sites until current method improvements are completed.

Response: Based on the final requirements, monitoring agencies will not be required to begin carbonyl sampling until June 2019. As stated in the proposal, the EPA has been evaluating improvements to the current method TO-11A that will reduce the uncertainty associated with that method. The EPA is confident that a revised method will be available by the time monitoring agencies are required to begin sampling.

(2) *Comment:* One commenter raised concerns with EPA's statement that the need for carbonyl data outweighs the concerns over the uncertainty of the data.

Response: Formaldehyde has been identified as both a highly important O_3 precursor but also a highly important air toxic. The statement made in the preamble to the proposed rule was based on the importance of formaldehyde data and on the knowledge that method improvements were underway that will reduce uncertainty in the carbonyl data.

d. ***Nitrogen Measurements***

(1) *Comment:* One commenter stated that if true NO_2 monitoring is desired at the PAMS sites, the photolytic oxide nitrogen analyzer may not be the best choice. They note that the FEM NO_2 by cavity attenuated phase shift (CAPS) may be a better choice.

Response: The EPA provided two examples of technologies that could be used collect true NO_2. We also noted that since NCore sites are currently equipped with a NO_y monitor, that a direct reading NO_2 instrument, such as the CAPS identified by the commenter, may be a preferred approach to collecting the true NO_2 measurement.

(2) *Comment:* Because NCore sites would have an NO_y monitoring in addition to the newly required NO2 monitor, one commenter suggested with the EPA provide clear and specific guidance on how agencies can request that the NO_y monitoring be eliminated.

Response: Adding a true NO_2 measurement to the existing NO_y measurement will provide a more complete characterization of reactive nitrogen compounds than either a single NO_y or NO_x analyzer. As such, the EPA does intend to allow waivers to remove the NO_y monitor at PAMS sites.

e. ***PAMS Season***

(1) *Comment:* One commenter stated that due to programmatic needs and logistical and technical capabilities, it would not be technically feasible for them to conduct enhanced monitoring activities during the months of June through August, and requested that a non-attainment area specific enhanced monitoring plan season be approved.

Response: The EPA agrees that for EMP monitoring activities, the sampling season should reflect the programmatic needs of the agency. As such, the sampling season for EMP activities was not defined. For the fixed monitoring sites, a minimum of June-August was maintained as the PAMS monitoring season to ensure consistent data availability for national and regional scale modeling efforts.

(2) Comment: One commenter stated that EPA proposed to extend the PAMS season beyond the June-August timeframe.

Response: The EPA did not propose to extend the PAMS season. We did request comments on extending the season, and indicated that as part of the EMP activities, monitoring agencies should consider monitoring for additional periods to meet local programmatic data needs. The final rule maintains the current PAMS monitoring season for the required PAMS sites.

(3) *Comment*: One commenter stated that EPA addressed extending the PAMS season by stating that the extension of the PAMS season should be part of an enhanced monitoring plan and that this assumes that the enhanced monitoring plan includes auto-GC as part of the plan, which is inconsistent with earlier language which discusses PAMS as one possible part of the enhanced plan and suggests that EPA should offer further clarification in the final rule.

Response: The EPA was referring to extending the monitoring season at the required PAMS site as one element that should be considered as the agency develops their EMP.

(4) *Comment*: One commenter stated that EPA only points to "the potential burden associated with lengthening of the PAMS season and the value of a uniform season to provide a consistent data set. The commenter states that these reasons are not a rational explanation for refusing to gather more, better data.

Response: The EPA did not propose changes to the PAMS monitoring season. The primary reason for maintaining a consistent 3-month monitoring season for the required PAMS sites was to ensure data availability from all PAMS sites for the months of June through August. Monitoring during this period of time will meet EPA's data needs for model development and evaluation. Because each state's ozone issues are likely to be different (as evidenced by the various ozone monitoring seasons) we recognized that monitoring agencies are in the best position to determine their data needs for local objectives and suggested that extending the season to additional months would be an appropriate consideration for the EMP.

f. ***Implementation and Timing***

(1) *Comment*: Several commenters raised concerns with the proposed compliance timeline stating that two years was not enough time to plan, purchase, install, and begin making PAMS measurements.

Response: The EPA addressed this concern in the final rule. The final rule allows for over three years from the time of the final rulemaking until monitoring agencies are required to begin making PAMS measurements at the newly required PAMS sites.

(2) *Comment*: Several commenters noted that existing NCore sites may not have sufficient space to accommodate the additional equipment needed to make PAMS measurements.

Response: The EPA agrees that in some cases existing infrastructure may not be sufficient to meet needs. The EPA is committed to work with monitoring agencies to identify actual needs and work on a funding strategy that will take infrastructure needs into consideration. In addition, the EPA included a waiver provision that would allow for making PAMS measurements at an alternative

location. Insufficient space would be a valid consideration in requesting an alternative location.

(3) *Comment*: A number of commenters argued that EPA should fund the PAMS program through 103 grants rather than 105 grants because 103 grants provide better access and control over the grant funds.

Response: The source of funding by EPA for state PAMS monitors is beyond the scope of this rulemaking. However, EPA notes that the PAMS program has historically been funded in part under the section 105 grant program and no change is expected in the grant mechanisms used to fund the PAMS program.

(4) *Comment*: Several commenters argued that if the PAMS revisions were finalized that the EPA would need to provide funding for the purchase of the equipment necessary to comply with the new requirements. One commenter noted that if an alternative site is selected to make PAMS measurements, rather than the NCore site, the costs of installation and operation would be dramatically greater.

Response: The PAMS program is funded in part through the section 105 grant program. The EPA recognizes that the changes being made as part of the final rule will result in an increase in monitoring burden in some areas and a decrease in monitoring burden in other areas. The EPA will work with the monitoring agencies to develop an appropriate funding strategy that will take into consideration these increases and decreases.

(5) *Comment*: Two commenters raised concerns regarding changes to the quality assurance requirements for criteria and non-criteria pollutants that were proposed in an earlier action. The commenters state that "the main problem with the proposal is the reduction in QA-related activities for non-criteria PAMS parameters" and that these changes "will degrade modeling results."

Response: The QA revisions being discussed by the commenter were proposed under a separate rulemaking (79 FR 54356). This rulemaking did not revise any of the QA requirements for the PAMS program, and as such, this comment is not germane to this rulemaking. Nonetheless, the EPA is fully invested in ensuring all environmental measurements meet minimum quality standards. Because the PAMS program is partially funded through the section 105 grants, all monitoring agencies making PAMS measurements will need to implement or have implemented a quality system conforming to the American National Standard ASQ/ANSI E4:2014: Quality management systems for environmental information and technology programs. Additional information on EPA's requirements for quality systems for environmental data collected using EPA grant funds can be found here - http://www.epa.gov/ogd/grants/assurance.htm

3. Comments on the Addition of a New Federal Reference Method (FRM) for O_3

a. *Comments on the Addition of a New FRM for O_3*

We received several comments on EPA's supporting report EPA/600/R-14/432 that are irrelevant to the establishment of the new FRM in this rulemaking. The EPA will not make any changes to the report as part of this rulemaking.

(1) *Comment*: The water vapor interference in the current ET-CL FRM is not minor, as EPA indicated in EPA's supporting report. Several sources indicate that such interference can be significant at high, summertime humidity. With reference to the supporting report EPA/600/R-14/432 (pages 3 and 5), the statement that the ET-CL FRM has no significant interferences should be corrected, as water vapor is a substantial interference.

Response: The proposed revised FRM for ozone specifies that FRM analyzers are to have a system for drying the sampled ambient air. This requirement would apply to both the original ET-CL as well as the proposed new NO-CL methodologies. All designated analyzers have been tested and have demonstrated compliance with the limits for water vapor interference equivalent specified in Table B-1 of Part 53. Moreover, EPA has proposed that this interference equivalent limit be reduced from 0.02 to 0.005 ppm. Also proposed was that the water vapor interference equivalent test specification for NO-CL analyzers in Table B-3 of Part 53 would require that water vapor be mixed with ozone so that the test more thoroughly determines water vapor interference (mixing is not currently required for testing ET-CL candidate analyzers). Further, in response to this and similar comments about water vapor interference, EPA intends to extend this same mixing requirement to apply to ET-CL analyzers, as well.

(2) *Comment*: With reference to the supporting report EPA/600/R-14/432 (page 6), the statement that the ET-CL and NO-CL methods behave in the same manner regarding the interferents listed in 40 CFR 53, Table B-3 is incorrect and should be corrected because the ET-CL method shows significant water vapor interference while the NO-CL method, equipped with a Nafion dryer, shows little response to water vapor. Further, the water vapor bias in the ET-CL method is positive while it is negative for the NO-CL method.

Response: The EPA disagrees with this comment. Data collected during laboratory-based and field studies conducted by EPA and documented in the EPA report have shown that very similar results were obtained from both the ET-CL and NO-CL methods even in high humidity environments.

(3) *Comment*: With reference to the supporting report EPA/600/R-14/432 (page 7), the statement that, in general, the UV photometric measurements compare very well with those obtained from FRM analyzers does not consider that the NAAQS are based on less than 1% of monitoring data rather than general, long-term average data. The report should focus more on instrument performance under typical meteorological conditions.

Response: The EPA appreciates this comment. However, this comment refers directly to the EPA report and its focus, rather than the approval of the new NO-CL FRM. The EPA will not make changes to the report as part of this rulemaking.

(4) *Comment*: With reference to the supporting report EPA/600/R-14/432 (page 10), the statement that FEM test results [submitted in an FEM application] are considered as confidential business information and can't be presented does not indicate that such results may be available on vendor websites.

Response: The EPA appreciates this comment. However, this comment refers directly to the EPA report and the commenter's suggestion of additional material that should be included in the report does not address promulgation of the new NO-CL FRM. The EPA will not make changes to the report as part of this rulemaking.

(5) *Comment*: The supporting report EPA/600/R-14/432 (page 14), notes that a slightly elevated offset (~2 ppb) was observed during the Houston study between the candidate FRM Model 211 UV and Model 265 NO-CL instruments and ascribes the offset to the performance of the Model 211. The EPA report should note that removal of moisture by the Nafion dryer in the Model 265 enhances O_3 concentrations relative to the Model 211's Nafion humidity control, which equalizes the water content between its sample and reference streams rather than drying them, and that this humidity difference between the 265 and 211 units may have played a role in creating the offset.

Response: The EPA disagrees with this comment as the data collected and documented in the report indicate an enhancement (~2ppb) of the scrubberless UV (SL-UV) results as compared to the NO-CL and ET-CL results. Enhancement of the NO-CL results would result in a negative intercept in the regression line as both chemiluminescence method results occupied the abscissa (x-axis) during the comparisons.

(6) *Comment*: Quenching may not explain the positive water vapor interference in the ET-CL method as EPA indicated in a referenced report. EPA/600/R-14/432 (page 5), points out that quenching of the chemiluminescence reaction causes the water vapor interference in the ET-CL method. The commenter disagrees and recommends that this statement should be removed, as quenching causes a negative, not positive, bias.

Response: The EPA appreciates this comment. However, this comment refers directly to the EPA report and the commenter's suggestion does not affect promulgation of the new NO-CL FRM. The EPA will not make changes to the report as part of this rulemaking. However, the referenced report does not list quenching as a source of the positive water interference as suggested by the commenter. Furthermore, the proposed revised FRM for ozone specifies that FRM analyzers are to have a system for drying the sampled ambient air. This

requirement would apply to both the original ET-CL as well as the proposed new NO-CL methodologies.

(7) *Comment*: With reference to the supporting report EPA/600/R-14/432 (page 6), the statement that the ET-CL and NO-CL methods behave in the same manner regarding the interferents listed in 40 CFR 53, Table B-1 is incorrect and should be corrected because the ET-CL method shows significant water vapor interference while the NO-CL method, equipped with a Nafion dryer, shows little response to water vapor. Further, the water vapor bias in the ET-CL method is positive while it is negative for the NO-CL method.

Response: The EPA disagrees with this comment. Data collected during laboratory-based and field studies conducted by EPA and documented in the EPA report have shown that very similar results were obtained from both the ET-CL and NO-CL methods even in high humidity environments.

(8) *Comment*: The supporting report EPA/600/R-14/432 (page 13), indicated that the Bendix 8002 FRM analyzer used for ambient evaluations was quite old and possibly not performing completely up to original factory specifications, which could have caused its lack of water vapor sensitivity. The report should address this possibility and its disagreement with the ASTM Method 5149 characterizations of the ET-CL FRM's humidity sensitivity.

Response: The EPA appreciates this comment; however, the comment refers directly to the EPA report and the commenter’s suggestion of focus does not affect promulgation of the new NO-CL FRM. The EPA will not make changes to the report as part of this rulemaking.

(9) *Comment*: Because the NO-CL technique requires removal of water vapor from the air sample, the rules should allow the use of a Nafion® dryer in the inlet line of a chemiluminescence analyzer.

Response: The proposed revised FRM for ozone calls for an internal dryer for both ET-CL and NO-CL FRM analyzers, so it appears unnecessary for another dryer in the inlet. Also, a proposed amendment to Part 53, Table B-3 would require an interference test for water vapor mixed with NO2 for NO-CL analyzers. Further, in response to another comment, EPA intends to amend Table B-3 to extend this mixing requirement to ET-CL FRM analyzers, as well. Therefore, there appears to be no need for an additional dryer in the inlet of these FRM analyzers. An older ET-CL FRM analyzer (if any are still in operation) that does not have an internal dryer may be equipped with an external inlet air dryer by requesting approval for a user-modification (40 CFR Part 58, Appendix C, Section 2.8).

(10) *Comment*: The EPA should clarify whether the proposed NAAQS for ozone is meant to be a "dry" or "wet" standard (presumably meaning whether or not the monitor uses a dryer), as removal of water vapor from the inlet stream will

increase the measured ozone concentration. A correction for humidity may be needed.

Response: The proposed changes to the ozone FRM require a dryer for FRM analyzers, and the proposed revised water interference test of Part 53 should obviate this issue.

(11) *Comment*: There is no need for trace-level ozone measurements, it's too resource intensive.

Response: The EPA disagrees with this comment and believes that there are situations where very low level measurements are needed.

(12) *Comment*: Suggests that the EPA failed to note that cylinder-to-cylinder differences in impurities in the pressurized ethylene gas cylinders required for the ET-CL method require recalibration of the monitor after each cylinder replacement. The EPA provided no information regarding the effect of impurities in the NO cylinder gas required for the NO-CL method.

Response: The EPA appreciates this comment and will consider further investigations regarding cylinder impurities and their impact on both the ET-CL and NO-CL FRMs.

(13) *Comment*: With reference to the supporting report EPA/600/R-14/432 (page 1), suggests that the EPA should reconsider NAAQS for gas-phase criteria pollutants in µg/m3 rather than ppm and allowances for adjustments for local barometric pressure, similar to NAAQS for particulate matter.

Response: The EPA disagrees with this comment, the referenced report EPA/600/R-14/432 (page 1), does not suggests that the EPA should reconsider NAAQS for gas-phase criteria pollutants in µg/m3 rather than ppm. In addition, modern ozone monitors allow for pressure compensation and as such, the computed mixing ratio is independent of local atmospheric pressure.

(14) *Comment*: One commenter suggests that EPA does not address whether the proposed new FRM is valid with respect to the correlation of past data utilized in health effect studies. In addition, monitored air quality itself may not correlate well with personal exposure to ozone levels.

Response: Air quality monitoring methods used in the past to collect monitoring data were either the ET-CL FRM or approved FEMs. The proposed FRM NO-CL methodology has been designated in two current FEMs. These FEMs have been shown per 40 CFR Part 53, subpart B to meet all performance limit specifications and also per 40 CFR Part 53, subpart C to show comparability to the current FRM (ET-CL). Also, as noted in the preamble, "a substantial number of laboratory tests have confirmed the excellent performance of the NO–CL analyzers as well as very close agreement with both ET–CL and UV analyzers in collocated field tests." Regarding correlation of monitoring data with personal exposure, we agree

that ambient concentrations can be different from personal exposures. The implications of such exposure measurement error for O3 health studies are discussed extensively in the ISA (e.g., see the Preamble and sections 2.3, 4.3). In addition, analyses in the HREA (HREA, Chapters 5 and 6) use the APEX model, which takes into account human time-activity patterns, along with spatially and temporally varying O_3 concentrations, to estimate actual exposures.

4. Comments on Revisions to the Procedures for Testing Performance Characteristics and Determining Comparability Between Candidate Methods (FEMs) and Reference Methods

a. *Comments on the Revisions to the Analyzer Performance Requirements*

(1) *Comment*: Table B-3 must be revised to include gaseous mercury since it is a major interferent in the conventional UV-absorption FEM (currently used by virtually all SLT monitoring agencies).

Response: The EPA appreciates this comment. Insufficient information is available on correct procedures to safely use high concentration of gaseous mercury in interference testing. The EPA will not finalize the commenters suggested change to 40 CFR Part 53, with this rulemaking.

E. Implementation of Proposed O_3 Standards

This section addresses comments concerning the EPA's proposed transition scheme for PSD permit applications pending when the EPA finalizes the 2015 revisions to the O_3 NAAQS. The proposal included two grandfathering milestones within the Prevention of Significant Deterioration (PSD) permitting process in order to provide a reasonable transition for certain PSD permits already in the review process when the revised O_3 NAAQS are finalized. Specifically, the EPA proposed PSD permit application grandfathering for the following milestones and corresponding dates by which the milestone must be reached to qualify for grandfathering: (1) applications for which the reviewing authority has formally determined that the application is complete on or before the signature date of the revised NAAQS; and (2) applications for which the reviewing authority has first published a public notice of a draft permit or preliminary determination before the effective date of the revised NAAQS (see proposed revisions to 40 CFR 51.166(i)(11) and 40 CFR 52.21(i)(12)). The proposed grandfathering provision would enable eligible PSD applications to continue to rely upon the demonstration that the proposed project would not cause or contribute to a violation of the O_3 NAAQS in effect at the time the grandfathering milestone is reached, rather than the revised O_3 NAAQS published in this final rule.

Comments supporting the PSD grandfathering provision as proposed are discussed in section II.E.1.a. Comments supporting grandfathering but recommending modification to the proposal are discussed in section II.E.1.b; while opposing comments are discussed in section II.E.1.c.

1. Comments supporting the proposed grandfathering provision for PSD

(1) *Comment:* Many commenters, largely representing state air or environmental agencies but also some industry commenters, supported the grandfathering provision for PSD permits as proposed.

Response: Consistent with these commenters, the EPA has decided to adopt the grandfathering provision as proposed in the final rule.

2. Comments supporting grandfathering for PSD with variations in the eligibility criteria

Some commenters who generally supported the concept of grandfathering pending PSD applications disagreed with particular aspects of the EPA's proposed approach and recommended varied ways to write the final grandfathering provision. These comments ranged from using only one milestone to grandfather applications submitted before the revised NAAQS becomes effective to grandfathering all permit applications submitted before the effective date of area designations for O_3. These comments are discussed below based on the approach as specifically recommended.

(1) *Comment:* Some industry and state commenters suggested using either a single milestone or a single date for grandfathering purposes. Some of these commenters recommended grandfathering only applications for which a completeness determination has been made on or before the signature date, while one

recommended grandfathering applications for which a completeness determination was made on or before the effective date of the revised O_3 NAAQS. One of these state commenters explained that that it may take months of consultation to produce a satisfactory air quality analysis, and that to go back and deem the application incomplete based on the air quality analysis after such a completeness determination would lead to additional delay. This commenter further noted that, because a complete permit application "is the basis of the specific statutory deadline in section 165(c) of the CAA for processing PSD applications within one year (or possibly shorter time periods …), it insinuates that a completeness determination is an important step in moving the permitting process forward" (e.g., MS DEQ).

Response: The EPA's response to these comments is included in section VII.B of the preamble to the final rule.

(2) *Comment:* Some commenters recommended changing the proposed approach to use the effective date of the revised O_3 NAAQS as the date for both grandfathering milestones. Several commenters reasoned that the distinction between the two proposed dates for the corresponding milestones was not fully explained or justified by the EPA in the proposal. One of the commenters explained that the effective date of the revised NAAQS is published in the Federal Register. Thus, commenters stated: "States and permit applicants therefore have some advanced notice of it. By contrast, public notice is not necessarily provided before the signature date for a final action at the completion of a NAAQS review." (e.g., UARG) One of these commenters indicated a preference that all PSD applications be grandfathered up to the effective date of the revised O_3 NAAQS. Other commenters (e.g., AGCA) was less specific in that they recommended that the EPA grandfather "certain NSR permit applications" that are pending on the effective date of the revised O_3 NAAQS.

Response: The EPA disagrees with these comments with respect to using the effective date of the final revised O_3 NAAQS for both milestones, for the reasons explained in section VII.B of the preamble to the final rule and further below. While the EPA is using the effective date of the revised O_3 NAAQS as the date for one of the grandfathering milestones, in sections VII.B and C of the preamble to the final rule the EPA explains its justification for why it is finalizing the grandfathering milestone based on a completeness determination as proposed, using the signature date of the final rule as the date, rather than the effective date of the revised O_3 NAAQS.

The EPA does not agree that the lack of advance notice to permitting authorities and permit applicants of the signature date for a final rule makes the date of signature an inappropriate date for the milestone based on completeness determinations. In general, the lack of such advance notice could help ensure that state permitting authorities do not prematurely issue completeness determinations to qualify for the grandfathering provision, thus helping to ensure that the integrity of the completeness determination process is preserved under the

grandfathering provision. Further, in this instance, the EPA is subject to a court order to sign for publication a notice of final rulemaking concerning its review of the O3 NAAQS no later than October 1, 2015. While the actual date that the Administrator signs the final rule may precede the deadline in the court order, permit applicants and permitting authorities do have some idea in this case of when the final NAAQS rule could be signed.

The EPA also declines to extend grandfathering to all PSD applications up to the effective date of the revised O3 NAAQS for the same reasons that it is not using the effective date of the final revised O3 NAAQS for both milestones, as explained in section VII.B of the preamble to the final rule. In addition the EPA does not believe it is appropriate to grandfather all permit applications, as that approach could delay implementation of the revised standards under the PSD program more than is necessary to accommodate the goals of the grandfathering provision. Therefore, the grandfathering provision finalized in this rulemaking is crafted to draw a reasonable balance that accommodates the requirements under both sections 165(a)(3) and 165(c). Permit applications that have not yet been determined complete can be supplemented or revised to address the revised O3 standards before the completeness determination is issued and the one-year deadline for processing permits in section 165(c) applies.

(3) *Comment:* Many commenters recommended that the EPA adopt a grandfathering provision that would allow PSD applications to be grandfathered with respect to the revised O_3 NAAQS based on milestones occurring beyond the effective date of the revised O_3 NAAQS. These commenters offered a variety of recommended approaches, as explained below. Nevertheless, many of these commenters shared a general concern that sources have put a lot of effort into the development of a permit application and believed that it would be unreasonable to require applicants to have to revise their applications due to the revised NAAQS. Some commenters (e.g., Axiall) stated that "[a] project that has a substantial PSD permit application submitted close to the time of the NAAQS finalization will have invested years of engineering and resources associated with securing a viable project. A permit application submitted near the date of the final NAAQS revision will have rightfully premised and sited the project based upon the current NAAQS and associated designation at the time of those activities, well before the final rule, and even before the proposal was made." Some of these commenters indicated that the two proposed milestones—complete application determination and publication of public notice—should continue to be used but that only one date should be applied to such milestones, the effective date of the area designations for the revised O_3 NAAQS. Some commenters recommended that grandfathering be based only on the publication of a public notice of the draft permit by the effective date of area designations for the revised O_3 NAAQS. Other commenters recommended grandfathering PSD/NSR permit applications that have completed the public participation process by the time of any designation of the area pursuant to any revised NAAQS. Another commenter recommended that the EPA allow the grandfathering of any application for which a PSD permit would be issued prior to the effective date of the area designations

for the revised O_3 NAAQS. Finally, several commenters recommended that the EPA grandfather all PSD permit applications submitted before the effective date of the area designations for the revised O_3 NAAQS.

Response: For the reasons explained in section VII.B of the preamble to the final rule, the EPA disagrees with these comments recommending that the EPA use dates after the effective date of the NAAQS as the date by which the permit application must meet the relevant milestone to qualify for grandfathering, as well as with the comments suggesting that the time period for the grandfathering provision should be expanded to allow a source located in an area designated as nonattainment for a pollutant at the time of permit issuance to obtain a PSD permit for that pollutant rather than a NNSR permit.

In addition, the EPA does not agree with the comment that it would be unreasonable to require applicants to revise their permit applications due to the revised NAAQS. While the EPA understands that applicants invest time and resources in a project prior to submitting a permit application, they should be aware of the possibility that NAAQS will be revised while project planning is underway. The Act requires the existing NAAQS to be reviewed every five years, through a public process that allows multiple opportunities for public participation, and that when NAAQS are revised, area designations may also need to be revised under the Act. The EPA publishes notice of its proposed action well before completing a revision to a NAAQS.

Further, as explained in section VII.A of the preamble to the final rule, absent a regulatory grandfathering provision, the EPA has long interpreted section 165(a)(3)(B) of the CAA and the implementing PSD regulations at 40 CFR 52.21(k)(1) and 51.166(k)(1) to require that PSD permit applications must include a demonstration that emissions from the proposed facility will not cause or contribute to a violation of any NAAQS that is in effect as of the date the PSD permit is issued. Thus, without a grandfathering provision, the CAA and the implementing regulations would require all permit applications that are pending at the time a revised NAAQS becomes effective to be revised to address the revised NAAQS.

The EPA has designed the grandfathering provision to balance these competing considerations and provide a reasonable transition mechanism for PSD applications for which both the applicant and the reviewing authority have committed substantial resources and in situations where the need to satisfy the demonstration requirement under CAA section 165(a)(3) could impact the reviewing authority's ability to meet the statutory deadline for issuing a permit within one year of the completeness determination, as required under CAA section 165(c). The EPA does not believe, however, that reliance interests of applicants or permitting authorities support a grandfathering provision that would excuse all permit applications submitted near the date of the revised NAAQS from the requirement to update their applications based on the revised NAAQS. For the same reasons explained in section VII.B of the preamble to the final rule

for not using later dates for the proposed milestones, such an approach would upset the reasonable balance that the EPA has drawn in the grandfathering provision as finalized to accommodate the requirements under both CAA sections 165(a)(3) and 165(c).

Moreover, because the revised O_3 NAAQS does not take effect immediately upon signature of the rule, but rather not until 60 days after publication in the Federal Register, when a pending permit application has not met one of the milestones in the grandfathering provision by the corresponding date, the EPA believes that permit applicants and permitting authorities will have sufficient notice and opportunity to update permit applications to address the revised O_3 NAAQS without causing undue delay in the permitting process.

Finally, the EPA does not agree that submission of a permit application should be used as a milestone for this grandfathering provision, because doing so could potentially result in a barrage of hastily assembled permit applications submitted before the specified deadline.

(4) *Comment:* Some commenters generally complained that the proposed grandfathering provision applies to only a limited number of sources or offers a very limited period of applicability, but did not recommend an alternative approach for the EPA's consideration.

Response: The EPA acknowledges that the grandfathering provision is limited in scope by design. As described in section VII of the preamble to the final rule and elsewhere in this section of the Response to Comments document, it is based on two milestones that represent stages in the permit review process at which substantial resources have been committed by both the applicant and the reviewing authority and situations where the need to satisfy the demonstration requirement under CAA section 165(a)(3) could impact the reviewing authority's ability to meet the statutory deadline for issuing a permit within one year of the completeness determination, as required under CAA section 165(c). Additionally, the proposed grandfathering provision for O_3 is consistent with prior grandfathering provisions that the EPA has promulgated through regulation, such as the one adopted in the final 2012 PM NAAQS rule. (78 FR 3086, January 15, 2013.) Accordingly, the EPA is finalizing the grandfathering provision as proposed.

3. Comments opposing PSD grandfathering

One group of commenters representing a number of environmental groups and health organizations submitted comments stating that the EPA does not have the legal authority to grandfather PSD sources, raising several legal arguments. The commenter disagreed with the EPA's interpretation that the CAA authorizes the agency to establish by rulemaking transition procedures for implementing new requirements that include the grandfathering of certain sources. The EPA provides a general response to this comment in section VII.B of the preamble to the final rule and further explains its authority and justification for the grandfathering

provision in section VII.C of the preamble to the final rule. In the comments and responses below, the EPA is providing a more detailed response to specific points made by these commenters.

(1) *Comment:* These commenters stated that the plain language of CAA section 165 forecloses the EPA's proposed grandfathering approach. The comment asserted that the Act does not confer authority on the EPA to exempt or grandfather permit applicants from the statutory PSD requirements. The comment quotes section 165(a)(3) of the CAA and cites section 168(b). The comment characterizes the grandfathering provision as a waiver of the statutory requirements. The commenter asserted that the statutory language applies PSD requirements based on commencement of construction and not on any stage of the permit application process. According to the commenter, because Congress limited the applicability of the PSD requirements in several ways when it adopted the PSD permitting program, including by providing specific grandfathering to sources that had commenced construction before the 1977 CAA Amendments, the EPA lacks authority to waive otherwise applicable, unambiguous statutory requirements (citing *Andrus v. Glover Constr. Co*., 446 U.S. 608, 616-17 (1980)). The commenter asserts that the EPA does not suggest that there is an ambiguity or gap in the language of CAA section 165(a)(3).

In support of its interpretation, this commenter also asserted that the grandfathering provision was inconsistent with the purposes of the PSD program in CAA section 160. The commenter pointed to statements the EPA had made in declining to establish grandfathering provisions in adopting the 1980 implementing regulations for the PSD program. The comment also pointed to the statutory structure and the legislative history, claiming that grandfathering was inconsistent with policy choices that Congress made in establishing the PSD program to prevent air pollution problems by limiting pollution from new sources and to require that controls be installed during initial construction rather than as retrofits. The comment further claimed that if emissions from grandfathered projects are determined later to lead to violations of the standards, states would be responsible for developing plans to meet the standards, which could lead to requirements for grandfathered sources to address these emissions in a less cost-effective manner through retro-fit controls. The comment asserted this outcome would undermine Congress' policy goals in the PSD program.

Response: The EPA does not agree with the interpretations of the CAA offered by the commenters who argue the EPA lacks authority for the proposed grandfathering provisions. To begin, the EPA has previously exercised its discretion to establish grandfathering provisions in regulations. Indeed, as far back as 1980, the EPA has asserted its authority to grandfather through rulemaking where provisions of the CAA contradict each other or are ambiguous. The EPA has cited the Administrator's authority under section 301(a)(1) "to set transitional rules which accommodate reasonably the purpose and concerns behind the two contradictory provisions" (45 FR 52676 at 52683 (August 7, 1980)). Consistent with this historical interpretation, as also explained in the

preamble to the final rule, the EPA has identified an ambiguity or gap in section 165. This provision does not address how the requirements of CAA section 165(a)(3)(B) should be met where the EPA issues a revised NAAQS while permit applications are pending, particularly when the EPA also determines that complying with the demonstration requirement for the revised NAAQS could hinder compliance with section 165(c)'s requirement to issue a permit within one year of the completeness determination for a certain subset of pending permits.

As explained in the proposal for this rulemaking, sections 165(a)(3) and 165(c), read together with the rulemaking authority in CAA section 301, provide the EPA the discretion to grandfather pending permit applications from addressing a revised NAAQS in certain circumstances. Such circumstances are those where grandfathering is necessary to achieve both the objective under section 165(a)(3) of protecting the NAAQS as well as the objective under section 165(c) of avoiding delays in processing permit applications. 79 FR 75234 at 75377 (December 17, 2014). The prior EPA actions cited in the proposal reflect the EPA's position, which is maintained in this rule, that in some circumstances, a conflict or tension may result between sections 165(a)(3) and 165(c). *Id.* at 75377-78 (citing the legal rationale and basis for grandfathering in the $PM_{2.5}$ rulemaking at 78 FR 3087 at 3253-59 (January 15, 2013), which includes a description of the tension between sections 165(a)(3) and 165(c)). More specifically, section 165(a)(3) requires that a permit applicant demonstrate that its proposed project will not cause or contribute to a violation of any NAAQS. At the same time, section 165(c) requires that a PSD permit be granted or denied within 1 year after the permitting authority determines the application for such permit to be complete. Thus, while the EPA generally agrees with the commenters that the requirements of section 165(a)(3) apply directly to sources, the CAA does not provide clear direction on how to apply section 165(a)(3) to NAAQS that become effective in circumstances where efforts to update a complete permit application to address the new or revised NAAQS would be time consuming and thus could impede compliance with section 165(c)'s requirement to issue a permit within one year of the completeness determination. As Congress has not spoken precisely to this issue, the EPA has the discretion to apply a permissible interpretation of the Act that balances the requirements in the Act to make a decision on a permit application within one year and to ensure the new and modified sources will only be authorized to construct after showing they can meet the substantive permitting criteria. *Chevron, U.S.A., Inc. v. Natural Res. Def. Council, Inc*., 467 U.S. 837, 843-44 (1984). In addressing the tension that exists in section 165 in some situations, the EPA also turns to section 301 of the CAA, where the Administrator is authorized "to prescribe such regulations as are necessary to carry out his functions under this chapter." When read in combination, it is reasonable to interpret these three provisions of the CAA to provide the EPA with the discretion to issue grandfathering regulations such as this one.

The EPA also disagrees with the commenter's assertion that targeted grandfathering applicable to a specific NAAQS is a waiver of the statutory requirements in section 165(a)(3). Rather, the grandfathering provision makes

clear which NAAQS are covered by this provision of the Act when it is applied to a permit application that has reached a specific stage in the review process before a specified date. The grandfathering provision resolves the question of how the EPA and other reviewing authorities should interpret and apply section 165(a)(3) of the Act in the case of today's revisions to the O_3 NAAQS, considering the requirement of section 165(c) of the Act that reviewing authorities make a decision on a permit application within one year of the date the application was determined complete. This is not a question of whether section 165(a)(3) applies; it is a question of which NAAQS this requirement should cover in the case of a PSD permit that is pending and has reached a particular milestone at the time the revised NAAQS takes effect. *See Sierra Club v. EPA*, 762 F.3d 971, 983 (9th Cir. 2014) (recognizing that the EPA's traditional exercise of grandfathering authority through rulemaking was consistent with statutory requirement to "enforce whatever regulations are in effect at the time the agency makes a final decision" because it in effect identified an operative date or dates, incident to setting the new substantive standard, explicitly building the grandfathering of pending permit applications into the new PSD regulations). In issuing such a regulatory grandfathering provision at the time of revising a NAAQS, the EPA is determining which O_3 NAAQS apply to certain permit applications that are pending when the EPA finalizes the 2015 revisions to the O_3 NAAQS, and thus clarifying, for the limited purpose of satisfying the requirements under section 165(a)(3)(B) for those permits, which O_3 NAAQS are applicable to that permit application and must be addressed in the source's demonstration that its emissions do not cause or contribute to a violation of the NAAQS.

The EPA agrees that as a general rule, section 165(a)(3) applies to "any NAAQS" that is effective as of the date a final PSD permit is initially issued (before any administrative appeal proceeding commences). However, with regard to the statutory structure, these provisions cannot be read in isolation and should be construed in the context of other provisions in section 165 of the Act, such as section 165(c). Legislative history further illustrates congressional intent to avoid delays in permit processing. S. Rep. No. 94–717, at 26 (1976) ("nothing could be more detrimental to the intent of this section and the integrity of this Act than to have the process encumbered by bureaucratic delay"). The commenters' interpretation also fails to take into account the EPA's ability to define through regulation when a new substantive standard becomes applicable for purposes of PSD requirements, as recognized in *Sierra Club v. EPA*, 762 F.3d at 983, and to enforce those requirements. Thus, the EPA does not agree with the view expressed by this comment that section 165(a)(3) must be read strictly in all circumstances to apply to all NAAQS in effect on the date the EPA issues a final permit decision, regardless of other requirements or provisions of the CAA. Similarly, the EPA does not agree that 165(a)(3) forecloses its ability to issue a grandfathering provision through rulemaking as it is doing in this rule.

The EPA is also not persuaded that the presence of a grandfathering provision in section 168(b) precludes the EPA from establishing grandfathering provisions in other circumstances. The EPA is not persuaded by the commenters' reference to

the Supreme Court's observation that when "Congress expressly enumerates certain exceptions to a general prohibition, additional exceptions are not to be implied in the absence of evidence of a contrary legislative intent," *Andrus*, 446 U.S. at 616–17. The Court applied this principle in a circumstance where there was a provision of law "expressly relating to contracts of the sort at issue here." *Id.* These are not the circumstances here. Section 168(b) of the Act does not expressly relate to the application of PSD permitting requirements to an application pending at the time of the promulgation of a new or revised NAAQS. Section 168(b) exempted facilities that were subject to permitting requirements under an earlier version of the PSD program created solely by the EPA regulation prior to the enactment of section 165 of the CAA and other provisions that expressly authorized and established the requirements of the PSD permitting program applicable today. This exemption operated to continue existing requirements for certain sources after a fundamental change in the statutory and regulatory regime under which such sources were required to obtain authorization to construct or modify major stationary sources of air pollutants. Such an exemption does not expressly relate to the incorporation of a new requirement into the PSD program, under existing statutory authority, when the EPA promulgates a regulation that creates such a requirement. In this case, the EPA is not grandfathering permit applications from the general prohibition in section 165(a) against commencing construction in the absence of a permit issued "in accordance with the requirements of this part." The CAA does not contain any express exemptions to the phrase "the requirements of this part" or from section 165(a)(3) of the Act that apply when the EPA promulgates a new or revised NAAQS.

The EPA also does not agree with the implication in the comment that the inclusion of the language "[n]o major emitting facility on which construction is commenced after August 7, 1977" in section 165(a) identifies the only sources that may be exempted from the requirements of 165(a). The EPA interprets this provision as identifying sources to which the requirements of section 165(a) would otherwise apply, but it does not address what requirements apply to such sources when the EPA promulgates a new or revised NAAQS. Furthermore, section 168(b) applied to sources that had commenced construction before new provisions of the CAA were enacted, whereas the grandfathering that the EPA proposed for purposes of this revised ozone NAAQS is applicable to changes in regulatory requirements prior to the issuance of a permit. Thus, the adoption of a one-time grandfathering provision upon enactment of the statutory PSD program is clearly different from grandfathering when the EPA promulgates a new or revised NAAQS, which the Act does not address. The fact that Congress expressly enumerated an exemption in section 168 intended to ease transition upon enactment of the PSD provisions in the Act does not constrain the Agency with respect to offering reasonable transitional provisions when EPA regulations create new PSD program requirements under those statutory provisions.

The EPA agrees that the purposes of the PSD program include the goals of protecting public health and welfare, preserving and protecting air quality in

protected areas, and assuring that a decision to permit increased air pollution is made after careful evaluation of that decision and an opportunity for informed public participation. In general terms, the EPA also agrees the PSD program is based on the goals of preventing air pollution problems and imposing control requirements when new projects are being constructed. Section 160(3) of the Act, however, also states that a purpose of the PSD program is to "insure that economic growth will occur in a manner consistent with the preservation of existing clean air resources." The EPA continues to construe this provision to call for a balancing of economic growth and protection of air quality. *See* 70 FR 59582 at 59587–88 (October 12, 2005); 78 FR 3086 at 3255 (January 15, 2013). As noted earlier, the legislative history further illustrates Congressional intent to avoid a moratorium on construction and delays in permit processing. The House Committee report describes how "the committee went to extraordinary lengths to assure that this legislation and the time needed to develop and implement regulations would not cause current construction to be halted or clamp even a temporary moratorium on planned industrial and economic development." H.R. Rep. No. 95–294, 95th Cong., 1st Sess., at 171 (1977). As an illustration of the lengths to which the committee went, the report lists five elements of the legislation, including the following statement: "To prevent disruption of present or planned sources, the committee has authorized extensive 'grandfathering' of both existing and planned sources." *Id.* Furthermore, the Senate Committee report specifically discusses concerns about delays in program implementation. S. Rep. No. 94–717, at 26 (1976) ("nothing could be more detrimental to the intent of this section and the integrity of this Act than to have the process encumbered by bureaucratic delay"). In the EPA's view, a limited grandfathering provision, such as the one in today's rulemaking, reconciles these various and competing purposes by striking an appropriate balance between the different goals of the Clean Air Act. The grandfathering provision in this rule ensures that new and modified major sources are authorized to construct only after they comply with the substantive PSD requirements, including the requirements to demonstrate that the source will not cause or contribute to a violation of the NAAQS that apply to that permit application. Under the grandfathering provision finalized in this action, grandfathered permit applications are still required to demonstrate that emissions from the proposed facility will not cause or contribute to a violation of the O_3 NAAQS, but that demonstration will be made with respect to the O_3 NAAQS that was in effect at the time the permitting milestone that qualifies the source for grandfathering was achieved. The rule also provides an opportunity for informed public participation concerning the analysis done for each permit application.

With respect to the EPA's actions in the 1980 PSD regulation, in those regulations the EPA sought to strike a balance between competing goals of the CAA in promulgating a grandfathering provision, as it is here (45 FR 52,676 at 52,683 (August 7, 1980)). In that rulemaking, the EPA explained that delaying certain construction "by imposing new PSD requirements could frustrate economic development" and noted that the grandfathered projects "have a relatively minor effect on air quality." *Id.* As a result, the EPA adopted a grandfathering provision

that "would strike a rough balance between the benefits and costs of applying PSD to those projects." *Id.* Although the EPA used issuance of permits previously required under the SIP in that case to determine eligibility for grandfathering, that decision in that rulemaking does not preclude the EPA from using other milestones in the permit process in this rulemaking to determine eligibility in order to strike the appropriate balance in a different situation.

(2) *Comment:* The commenters disagreed with the EPA's interpretation that there is a conflict between sections 165(a)(3) and 165(c). The comment claimed that if a permit cannot be approved within the required time frame and also meet its air quality demonstration obligation, the appropriate course is to deny the permit application or determine that the permit is no longer complete. Even if it were impossible to comply with both sections, the commenters indicated that the Act should be construed to give effect to all of its provisions, citing *Hibbs v. Winn*, 542 U.S. 88, 101 (2004) and *Bernier v. Bernier*, 147 U.S. 242, 245 (1893), rather than ignoring some, and also stating that the EPA does not have authority to revise clear statutory terms, citing *Utility Air Regulatory Grp. v. EPA*, 134 S.Ct. 2427, 2446 (2014). The comment also asserts that the Supreme Court has already rejected the EPA's reasoning that we must reconcile the statutory obligations to issue a permit within one year and to ensure that the permitting requirements of section 165 are fulfilled. They cite the holding in *General Motors Corp. v. United States*, 496 U.S. 530 (1990) that delay on the part of the EPA does not affect the Agency's ability to enforce the other requirements of the Act. The commenter additionally says that any problem that might exist could be managed if permit applicants assessed compliance with an ozone standard down to 60 ppb, pending final promulgation of the ozone standard. If a particular proposed project would cause or contribute to a violation of this lower standard, the applicant could prepare accordingly without delaying review of the permit.

Response: As the EPA explained in the preamble to the final rule and the preceding comment response, as well as in prior actions, its view is that there is a tension or conflict between sections 165(a)(3) and 165(c) that it must reconcile in situations where the ability of air agencies to complete action on a permit application within the statutory one-year deadline is likely to be impeded if a new or revised NAAQS becomes applicable during the review of a pending permit application. The commenters' suggestion that the EPA could resolve this tension by generally denying permit applications or determining applications are no longer complete, and presumably requiring further analysis from permit applicants, does not adequately address the issue because both of these approaches would lead to additional delay in permitting. As explained in the prior comment response, the legislative history shows Congress' intent to avoid delays in permit processing and construction moratoriums, and indicates that Congress saw grandfathering as a means of accomplishing that goal. That congressional intent is also reflected in the one-year deadline to issue or deny a permit in section 165(c).

The commenters' suggestion that it would avoid delay if permit applicants assessed compliance with a possible lower standard is unsupported and effectively imposes the lower end of a proposed NAAQS on permit applicants before such a standard is adopted and the EPA considers public comments. Such an approach could also lead to delay because the demonstration that a source would not cause a violation of the more stringent requirement could well require additional resources and time. In addition, if a higher standard is ultimately selected, an applicant or permitting authority might decide to conduct additional assessments to see if a higher emission limitation would be adequate to protect the standard selected.

The EPA is cognizant of the principles of statutory construction cited by the comment and has respected those. The EPA's interpretation does not render section 165(a)(3) void, inoperative, insignificant, or superfluous. This grandfathering provision will affect only one requirement, the one in the regulations implementing section 165(a)(3)(B), and grandfathering continues to give this provision effect by requiring permit applications eligible for grandfathering to demonstrate compliance with the O_3 NAAQS that was in effect at the time of the relevant milestone that makes the application eligible for grandfathering. Moreover, the comment cited *Hibbs v. Winn*, 542 U.S. 88, 101 (2004), but that case also declined to read the relevant statutory term in isolation. In that case, the Supreme Court recognized the "cardinal rule that statutory language must be read in context." *Id.* (citation and internal quotations omitted).[132] As explained above, the EPA is construing section 165(a)(3) in the context of other provisions in section 165, such as 165(c), as well as section 301(a). In this way, the EPA's interpretation does not revise the Act, but rather balances the requirements of sections 165(c) and 165(a)(3), giving effect to each. The commenters' reliance on *Utility Air Regulatory Grp. v. EPA*, 134 S.Ct. 2427, 2446 (2014) for the proposition that the EPA does not have authority to "revise clear statutory terms that turn out not to work in practice" is inapposite. The EPA is not seeking to change clear statutory terms, but rather to resolve an ambiguity in the Act. The fact that the commenter would draw a different balance between these provisions does not make the EPA's interpretation impermissible. *See Chevron U.S.A., Inc. v. Natural Resources Defense Council, Inc.*, 467 U.S. 837, 842-43 (1984) (where the "statute is silent or ambiguous with respect to the specific issue," the agency's interpretation is upheld so long as it constitutes "a permissible construction of the statute.").

[132] The comment also cites *Bernier v. Bernier*, 147 U.S. 242, 245 (1893), for the principle of statutory construction that it "is a general rule, without exception, in construing statutes, that effect must be given to all their provisions if such a construction is consistent with the general purposes of the act and the provisions are not necessarily conflicting." The Court in that case went on to say that "[w]hen a provision admits of more than one construction, that one will be adopted which best serves to carry out the purposes of the Act." *Id.* While the EPA respects the general principles of statutory construction articulated in this case, we note that this case was decided long before *Chevron* and thus does not account for the deference that present day courts give to administrative agencies' construction of the statutes that they administer.

The EPA also disagrees with the comment's interpretation of *General Motors Corp v. United States*, 496 U.S. 530, 540-41 (1990). *General Motors Corp* concerned enforcement of an existing SIP provision, not promulgation of a regulation for PSD permitting. In *General Motors Corp. v. United States*, the Supreme Court held that enforcement of an existing SIP is not barred by the EPA's unreasonable delay in acting on a proposed revision of the SIP. The situation presented in that case is distinguishable from the one addressed by the EPA in this rule. In *General Motors*, the court addressed a different question, which was whether the Agency's delay in approving a change in law (a revision of a SIP) barred the Agency from applying the previously applicable law (the earlier version of the SIP). In contrast, the EPA does not here contend in this instance that any delay on its part justifies declining to apply the permitting requirements in effect at an earlier time. The requirements applicable to the permit at the time of the applicable milestone continue to apply to the permit application. The question the EPA is addressing in this rule is when in the application review process a new requirement should supersede the previously-applicable requirement.

In addition, under the grandfathering provision, a grandfathered permit application would still be required to obtain a PSD permit and to meet all the substantive PSD obligations in section 165(a) that apply to it before constructing. The grandfathering provision merely reflects the interpretation that one of these substantive obligations (section 165(a)(3)) should not be read to incorporate a new standard where that would affect the ability of the permitting authority to satisfy another statutory requirement (section 165(c)). In addition, *General Motors* supports the general principle that a reviewing authority should apply the regulations that are in effect at the time it takes final action. Since the EPA would promulgate a regulatory grandfathering provision in this situation, any reviewing authority applying this provision would be acting consistent with the regulatory requirements in effect at the time it makes its final decision, consistent with *General Motors*.

(3) *Comment:* The commenters also challenged the EPA's use of its general rulemaking authority section 301 to alter what the commenter sees as the plain requirements of the Act. The comment stated that courts have rejected the notion that the EPA's authority to issue regulations under section 301 is open-ended, particularly when there is statutory language on point, citing several cases: *NRDC v. EPA*, 749 F.3d 1055, 1063-64 (D.C. Cir. 2014); *Am. Petroleum Inst. v. EPA*, 52 F.3d 1113, 1119 (D.C. Cir.1995); *NRDC v. Reilly*, 976 F.2d 36, 41 (D.C. Cir.1992); *Sierra Club v. EPA*, 719 F.2d 436, 453 (D.C. Cir. 1983).

Response: Section 301 authorizes the EPA's Administrator to "prescribe such regulations as are necessary to carry out his functions under" the Act. In the grandfathering proposal and in the preamble to the final rule, the EPA did not rely solely on section 301 to support its authority for the grandfathering provision, but rather read this section in conjunction with the tension or ambiguity between the requirements of sections 165(a)(3) and 165(c). In that context, the EPA interprets

the authority under section 301 to support its ability to promulgate a regulation that reasonably addresses the ambiguity of how to apply the revised O_3 NAAQS in PSD permitting and that balances the competing requirements of sections 165(a)(3) and 165(c). The grandfathering provision establishes when the revised NAAQS will apply for certain regulatory requirements and certain PSD permit applications that are pending at the time that the revised NAAQS is finalized. As noted in an earlier response in this document, in promulgating the 1980 PSD regulations, the EPA cited authority under 301(a)(1) "to set transitional rules which accommodate reasonably the purpose and concerns behind … two contradictory [statutory] provisions." 45 FR 52676, 52683 (Aug. 7, 1980). In addition, the EPA noted that even in the absence of a conflict between sections of the Act, "EPA would have the authority under section 301(a)(1) to exempt those projects in order to phase-in new requirements on a reasonable schedule." *Id.* at 52683 n. 5. The EPA maintains those interpretations of section 301(a)(1) in this action.

The statements in the cases that the comment cited are inapposite because they address situations where there is no ambiguity or conflict in the relevant statutory provision. For example, the comment cites *NRDC v. EPA*, 749 F.3d 1055, 1063-64 (D.C. Cir. 2014), for the principle that "EPA cannot rely on its gap-filling authority [in section 301] to supplement the Clean Air Act's provisions when Congress has not left the agency a gap to fill." Similarly, *NRDC v. Reilly*, 976 F.2d 36, 41 (D.C. Cir.1992), and *Sierra Club v. EPA*, 719 F.2d 436, 453 (D.C. Cir. 1983), both concerned situations where the court determined that the relevant provision of the Act spoke specifically or clearly to an issue, and that the EPA could not rely on authority under section 301 to trump that clear direction. *See NRDC*, 976 F.2d at 41 (where the CAA mandated a "highly circumscribed schedule" for the promulgation of certain regulations, section 301 did not provide the EPA with authority to stay regulations that were subject to those deadlines); *Sierra Club*, 719 F.2d at 453 (where CAA section 123 expressly lists three statutory criteria for determining what good engineering practice is with respect to stack heights, the EPA did not have authority under section 301 to add a fourth). *Am. Petroleum Inst. v. EPA*, 52 F.3d 1113, 1119 (D.C. Cir.1995), interprets a different CAA provision, section 211(k)(1), which gives the EPA the authority to promulgate regulations establishing requirements for reformulated gasoline and which is not applicable to this situation. The court found that this provision unambiguously precluded the EPA from adopting regulations that were not directed at the reduction of VOCs and toxic emissions, and thus held that the general grant of authority in section 211(k)(1) did not authorize regulations that were independent of such reductions and that could lead to increases of those pollutants. *Id.* ("EPA cannot rely on its general authority to make rules necessary to carry out its functions when a specific statutory directive defines the relevant functions of EPA in a particular area.") These cases are all distinguishable from the situation here because in this case the EPA is using its general regulatory authority in section 301 to address the ambiguity in the Act that results from the tension between sections 165(a)(3) and 165(c).

(4) *Comment:* The commenter also disagreed that *Sierra Club v. EPA*, 762 F.3d 971 (9th Cir. 2014) supported the EPA's authority to grandfather PSD applications when the agency does so through rulemaking. The commenter stated that the court found the statute unambiguous on the question of whether section 165(a)(3) requires sources to demonstrate compliance with the NAAQS in effect at the time of permit issuance and that this ends the statutory analysis. Referring to the court's discussion of the EPA's previous grandfathering provisions, which were established through rulemaking, the commenter asserted that the court misunderstood those actions because the court seemed to believe that the EPA had adjusted the operative dates of those NAAQS in the prior grandfathering provisions. The commenter disagreed with that interpretation, saying that in those actions the EPA was actually waiving the statutory requirement to enforce whatever NAAQS are in effect at the time the agency makes a final decision. The comment concluded that the Ninth Circuit found such a waiver exceeded the EPA's statutory authority.

Response: The EPA disagrees with this comment and, as made clear in the preamble to the final rule and in prior comment responses in this document, believes that the *Sierra Club v. EPA* decision provides support for the grandfathering authority it is using here. In that case, the U.S. Court of Appeals for the Ninth Circuit vacated a decision by the EPA to issue an individual PSD permit after grandfathering a permit applicant from certain requirements without first amending its regulations to include a grandfathering provision. The requirements at issue included newly effective NAAQS that had become effective while the permit application was pending. 762 F.3d 971, 973-74 (9th Cir. 2014). The court contrasted that permit-specific, non-regulatory grandfathering with the regulatory approach that the EPA is taking in this rulemaking and made clear that the court's vacatur of that permit decision did not limit the EPA's authority to grandfather through rulemaking. *Id.*, at 982, n. 7 & 982–983 (stating that the court did "not doubt, or express any opinion on, the EPA's traditional authority to employ formal rulemaking to implement grandfathering").

Contrary to the comment's characterization, the court did not hold that section 165(a)(3) requires a source to demonstrate compliance with the NAAQS in effect at the time of permit issuance. Rather, the court stated "we hold that the [CAA] unambiguously requires [the permit applicant] to demonstrate that [the project] complies with *the regulations in effect* at the time the Permit is issued." *Id.* at 973-74 (emphasis added, internal citation omitted). The court determined that the EPA does not have "authority to simply waive the newly effective regulations on an ad hoc basis." *Id.* at 982. Accordingly, the court concluded that "at least *without applicable grandfathering provisions in the relevant regulations*, the EPA must enforce the regulations in effect at the time each Permit is issued, as the [CAA] clearly requires." *Id.* at 983 (emphasis added). The court saw a "significant difference" between the EPA's prior applications of grandfathering through rulemaking, which the court said did not "on its face, violate the plan statutory mandate to enforce whatever regulations are in effect at the time an agency makes a final decision," and "its waiver of the currently applicable regulations" in that

case. *Id.* at 983. Thus, the court's holding and its interpretation of the CAA's requirements are both carefully crafted to preserve the EPA's discretion to issue a grandfathering provision through rulemaking, as it does in this action.

In addition, the court's analysis favorably discussed prior actions in which the EPA had grandfathered permit applications through rulemaking, including actions that are directly analogous to the action the EPA is taking today, such as the grandfathering provision that the EPA issued when it revised the NAAQS for $PM_{2.5}$ in 2013. *Id.* at 983. While the comment asserts that the court misunderstood those prior actions, the court's interpretation is not inconsistent with those actions. The court recognized that NAAQS and other CAA requirements are implemented by the EPA through regulations, so it understood the EPA's prior regulatory grandfathering actions as in effect "specifying an operative date (or dates) for each new regulation," as they were adopted through notice-and-comment rulemaking, such that the grandfathering of pending permit applications was expressly part of the new regulations. *Id.* Even if the EPA did not expressly frame its prior actions in those precise terms, the EPA believes that it is reasonable to view the grandfathering provision adopted in this rule as specifying the operative date of the NAAQS for certain PSD permit applications and requirements. At the same time that the EPA is otherwise adopting the NAAQS and specifying the effective date, the EPA is addressing the extent to which the revised standards apply to pending PSD permit applications and for purposes of specific PSD requirements. As such, the EPA is adopting this view as part of its rationale for today's action.

(5) *Comment:* The commenters asserted that the EPA's statements that it has adopted similar exemptions, which they characterized as illegal, in the past also does not provide any authority to continue such practices here, citing *New Jersey v. EPA*, 517 F.3d 574, 583 (D.C. Cir. 2008) ("[P]revious statutory violations cannot excuse the one now before the court.") and *F.J. Vollmer Co. v. Magaw*, 102 F.3d 591, 598 (D.C. Cir. 1996) ("[W]e do not see how merely applying an unreasonable statutory interpretation for several years can transform it into a reasonable interpretation.").

Response: The EPA has explained at length in the preceding responses why it believes that its interpretation of the Act is both legal and reasonable. Accordingly, the EPA does not agree with the premise of these comments that its interpretation is illegal or unreasonable. The EPA's view is not simply based on past practice, but rather based on its interpretation of the Act articulated in the record for this rule. The reasonableness of this interpretation is bolstered by the fact that it is consistent with the EPA's long-standing interpretation of the Clean Air Act to allow for grandfathering under circumstances like these, dating back to its application of a grandfathering provision in the 1980 PSD regulations, as described above. The EPA has maintained this interpretation, applying it most recently before this action, by establishing a similar grandfathering provision for the $PM_{2.5}$ NAAQS issued in 2013. 78 FR 3086 (January 15, 2013). No court has ever found that the EPA's interpretation is inconsistent with the Clean Air Act,

and Congress has taken no action to amend the Act to preclude the EPA's reading.

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, as well as issues that are not germane to the setting of the NAAQS. Many legal issues are addressed generally throughout the preamble to the final rule. Specific responses to other comments are presented below.

(1) *Comment:* In support of their view that the NAAQS should not be revised to be more stringent, some commenters stated that peak natural background levels in several states would make it impossible for those states to attain and maintain the revised standards and that the EPA's analyses were underestimating background concentrations. These commenters identified several aspects of the EPA's analyses which they claim contributed to underestimates of background O_3. These include: a) use of stationarity in boundary conditions and meteorology given increasing trends and climate change, b) use of zero out modeling and/or use of source apportionment modeling, c) model performance issues (e.g., underestimate frequency and magnitudes of stratospheric intrusions and international transport), d) the EPA doesn't provide persuasive data to support the statement that background O_3 contributions are similar between high O_3 days and other O_3 days and e)the EPA's emphasis on model mean background levels is inappropriate, given that the standard has a 4th highest daily average form (meaning that maximum days, or at least 4th highest days should be the focus**)**. Finally, some commenters also cited existing non-EPA analyses in support of their view that background O_3 would prevent attainment of revised NAAQS.

Response: In section II.B.2 of the preamble to the final rule, the EPA has responded to the aspects of this comment related to consideration of attainability in decisions on the NAAQS and generally responded to aspects of the commenters' characterization of background O_3. Based on that discussion, the EPA's ultimate conclusion is that commenters have at most shown that there are infrequent instances, almost exclusively in rural locations in the intermountain west, where the revised NAAQS might be exceeded, in large part, but not exclusively, due to levels of background O_3. The NAAQS, of course, are national standards, and "the agency need not tailor national regulations to fit each region or locale" (*API v. EPA*, 665 F. 2d at 1185 (rejecting argument of city of Houston that EPA had acted arbitrarily in establishing a (purportedly) unattainable standard due to natural factors in the Houston area, and holding further that "attainability and technological feasibility are not relevant considerations in the promulgation of national ambient air quality standards"). Moreover, the record (including studies submitted by commenters) indicates that the background levels which might exceed revised O_3 NAAQS typically result from stratospheric intrusions or wildfires (see e.g., Attachment G of NMA comments [Las Vegas Ozone Study], p. 14). The CAA and the EPA's implementing regulations, specifically the Exceptional Events Rule at 40 CFR 50.14, allow for the exclusion of air quality monitoring data from design value calculations when there are exceedances caused by certain event-related U.S. background influences, notably

wildfires and stratospheric intrusions. The resulting design values, when considered with an area-specific analysis of multiple factors, could lead to regulatory relief from an initial area designation as nonattainment.

We address other specific technical aspects of these comments related to the EPA modeling analyses here, in items (a) through (e) below. In many cases, the comments reference background O_3 only in the generic sense. Background O_3 can be generically defined as the portion of O_3 that comes from sources outside the jurisdiction of an area and can include natural sources as well as transported O_3 of anthropogenic origin. Two specific definitions of background O_3 are relevant to this discussion: natural background (NB) and United States background (USB). Natural background is defined as the O_3 that would exist in the absence of any manmade precursor emissions, and USB is defined as that O_3 that would exist in the absence of any manmade emissions inside the U.S. (ISA, p. 3-31). This includes anthropogenic emissions outside the U.S. as well as naturally occurring O_3. It is important to note that both of these quantities are theoretical constructs and therefore there is no way to definitively measure or validate these numbers. Despite the theoretical nature of NB and USB, they can provide useful information and context for policy decisions. Unless explicitly noted otherwise, we have assumed all references to background in the comments are intended to refer to USB. Interstate and intrastate transport of domestically-generated ozone can influence ozone concentrations in downwind rural areas and is not part of USB. In items (f) through (i) below, we also address the characterization of existing non-EPA studies of background O_3 that some commenters also cited in support of their view that background O_3 would prevent attainment of revised NAAQS.

a) We agree with commenters that stated that emissions upwind of the U.S. contribute to USB, and that climate change has the potential to impact O_3 concentrations in the future. However, we disagree that, in developing O_3 concentration estimates for the present day from estimates for a 2007 scenario,[133] the EPA has underestimated background by not accounting for increased international emissions and for future climate change. With regard to international emissions, we consider projections of O_3 precursor emissions from upwind countries (e.g., Asia) to be highly uncertain (Klimont et al., 2009), making it difficult to assess the representativeness of the 2007 scenario relative to more current conditions. Further, even if we were to modify the EPA estimates by recent estimates of Asian emissions impacts, the result would not make the estimates appreciably closer to the level of the current or revised NAAQS. For example, ambient monitoring data collected over the past 15-20 years suggest that the seasonal mean concentrations of O_3 in the free troposphere over remote portions of the U.S., which some researchers use as a surrogate for intercontinentally transported O_3, has increased at a rate of approximately 0.4 ppb per year (Cooper et al., 2010; Cooper et al., 2012).

[133] The EPA estimates of USB for the present day time period were informed by global model outputs for a 2007 scenario (PA, section 2.4).

Additionally, there is some evidence that this trend has slowed over the most recent period (Cooper et al., 2012). The EPA's median seasonal mean USB maximum-daily 8-hour (MDA8) O_3 concentration is 32.7 ppb (PA, section 2.4). Even if we were to extrapolate the ambient free-tropospheric trend of 0.4 ppb/year (i.e., approximately 3.0 - 3.5 ppb over an 8-year period), USB concentrations would still typically be well below, and therefore would not preclude, attainment of the 70 ppb level of the revised standard.[134] With regard to climate change impacts, we note that the time period for such impacts is typically over longer scales (Jacob and Winner, 2009). For shorter projections, such as from 2007 to the current period, natural inter-annual variability, which the EPA estimates have addressed, tends to be a more significant factor. Thus, we consider the assessment of background O_3 based on a 2007 modeling scenario to provide a sound, state-of-the-science estimate of present-day USB.

b) Some commenters objected to the use of zero-out modeling methodology to estimate USB O_3 because they regard zero-out simulations as inherently unrealistic scenarios that do not properly account for nonlinear O_3 chemistry. At the same time, other commenters objected to USB estimates based on source apportionment modeling because it does not reflect the increasing role of USB in the future as domestic emissions are reduced. Both the zero-out and source apportionment techniques have a long history of supporting regulatory decision-making and the EPA utilized both methodologies in its estimates of USB concentrations. When averaged over all sites, O_3 from sources other than U.S. anthropogenic emissions is estimated to comprise 66 (zero-out) and 59 (source apportionment) percent of the total seasonal O_3 mean (PA, p. 2-20). The spatial patterns of USB and apportionment-based USB are also similar across the two modeling approaches. The EPA believes that both methodologies provide valuable and consistent insight into USB concentrations across the country and that both approaches are well-suited for this use.

c) The EPA disagrees with commenters who suggested that the EPA estimates of USB were low due to model performance issues, including underestimating the frequency and magnitude of stratospheric intrusions, as well as underestimating the importance of international transport. Because USB is an entity that cannot be directly measured, it is evidently not possible to evaluate via direct measurement how well any model simulates USB. Nor is it possible to directly evaluate how well the model can simulate components of USB like stratospheric contributions or international transport. Thus, we have attempted to evaluate USB indirectly. First, we evaluated the model base case simulations against observations. As shown in Appendix 4b of the HREA, the operational evaluation of the base CMAQ modeling exhibited relatively low bias when compared against observed O_3 data in 2007. In another indirect evaluation, we

[134] Even some of the materials cited by commenters emphasize that international contributions to background are relatively insignificant in comparison with contribution due to stratospheric intrusions and wildfires (see, e.g., NMA Attachment G [Las Vegas Ozone Study], p. 14).

assessed whether there is any relationship between USB estimates and daily model performance. The EPA analyses suggest (Dolwick et al., 2015) that there is no significant correlation between USB and model bias at the vast majority of sites. However, for a subset of sites at high elevation (sites located at elevations above 1 km), there is a slight tendency for the model to produce lower USB estimates on days when the model is underestimating the observed O_3. At these same sites, the model also shows a tendency toward higher USB estimates on days where the model is known to have overestimated observed O_3. Based on these findings, the EPA concludes that the modeling used to characterize USB across the U.S. has performed consistent with the state-of-the-science, and with performance that is comparable to other regional modeling exercises. Accordingly, the EPA finds the associated estimates sound and appropriate for their use here.

The EPA agrees with the commenters that described the values of USB O_3 on possible exceedance days to be a more meaningful consideration from an implementation perspective than seasonal mean concentrations. We have described this aspect of the analysis in Section 2.4.3 of the PA by analyzing the differences between USB estimates on high O_3 days versus other days (PA, Figures 2-14 and 2-15). The modeling indicated that the proportion of total O_3 that originates from USB is smaller on high O_3 days (e.g., days > 60 ppb) than on the more common lower O_3 days that tend to drive seasonal means. Thus, we disagree with commenters questioning the sufficiency of data showing that background O_3 concentrations remain relatively constant on high and median O_3 days. In fact, the highest modeled O_3 site-days tend to have background O_3 levels similar to mid-range O_3 days. Figures 2-14 and 2-15 of the PA show that days with highest O_3 levels have similar distributions (in the sense of similar means and similar inter-quartile ranges) of background levels as days with lower values. These results are similar whether zero-out or source apportionment modeling is utilized (PA, p. 2-21). The results are likewise consistent with an analysis of two earlier non-EPA modeling studies, Zhang et al. (2011) and Emery et al. (2012).[135] In a 2012 technical memorandum, EPA staff analyzed the results of the modeling analyses from Zhang et al. (2011) and Emery et al. (2012) using data provided by the study authors and showed that background O_3 does not vary significantly as 8-hour peak O_3 levels increase above 55-60 ppb (Henderson et al., 2012, pp. 8-10). This result was observed nationally as well as in individual regions, including locations within the intermountain west where background concentrations typically represent a higher percentage of the total modeled O_3. Even at these locations, however, the air quality modeling indicates that background represents a smaller fraction of total O_3 on days with the maximum 8-hour average concentration greater than 60 ppb than on days representative of the seasonal mean of such concentrations.

[135] These two studies are also referenced by some commenters (e.g., see section f below).

The EPA has thus demonstrated with multiple modeling analyses and multiple modeling approaches that the relative contribution of background O_3 is less on high days, than on low or median O_3 days. This fact has significant implications. First, it indicates that, in most locations, domestic anthropogenic sources are "largely responsible for the 4th highest 8-hour daily maximum O_3 concentrations" which determine attainment status (PA, pp. 2-20 to 21). This is because when looking across regions, domestic anthropogenic emissions contribute higher percentages of O_3 on higher O_3 days (since background is shown to be similar between mean and high O_3 days). Second, the anthropogenic domestic emissions contribution is important, not only with respect to attainment considerations, but also with respect to public health considerations. As noted in the PA, the proposal and the preamble to the final rule, the EPA and the Administrator have greatest confidence in O_3-attributable health effects at the upper ends of the distributions of ambient O_3 concentrations (79 FR 75291; preamble to the final rule, section II.B.2.c.iii; PA, p. 3-70 and 4-39). These occur on the days that domestic anthropogenic emissions typically contribute greater proportions, meaning that the days with the higher potential for risk to public health are those with the higher percentages of anthropogenic domestic emissions.

Again, for the relatively infrequent cases where background O_3 does contribute strongly to high O_3 values, the CAA and the EPA implementing regulations, specifically the Exceptional Events Rule at 40 CFR 50.14, allow for the exclusion of air quality monitoring data from design value calculations when there are exceedances caused by certain event-related U.S. background influences, notably wildfires and stratospheric intrusions. This exclusion of data could produce a design value that meets the level of the NAAQS and result in an initial area designation decision indicating that the affected area meets the NAAQS.

d) The EPA strongly disagrees with commenters that conclude that the USB analyses focus disproportionately on seasonal means and not on days with high O_3 concentrations. As discussed above, the PA presents both analysis perspectives (PA, section 2.4.3).

e) A number of commenters (e.g. UARG) indicated that the Langford et al (2015) summary of the Las Vegas Ozone Study (LVOS) showed that this area would frequently exceed a standard of 70 ppb due to background concentrations, and suggested that this situation is illustrative of broader conditions in the western U.S. Some cited this study as evidence that the city of Las Vegas would exceed the proposed standard (set at any part of the proposed range) due "almost entirely" to background emissions (e.g., Murray Energy comments, citing to December 2014 Congressional testimony by NAM).

The LVOS was conducted over 43 days during May and June 2013. During that period there were 14 days in which at least one O_3 monitor in Clark Co., NV exceeded 70 ppb. Additionally, the LVOS suggested that some of these exceedance days had contributions from background sources such as

stratospheric air or wildfire plumes. It is generally inappropriate to assume that monitored O_3 concentrations can be used as a proxy for background O_3, and particularly inappropriate to do so with respect to the study area of the LVOS. According to the 2010 census, nearly two million people live in Clark Co., NV. Not surprisingly, the paper by Langford et al (2015) identifies multiple causes for high O_3 values in this region, including stratospheric air, wildfires, and transport from Asia; but also including locally-generated pollution and urban pollution transported from the Los Angeles Basin (Langford et al., 2015, p. 308). Further, at no point does the Langford paper use the phrase "almost entirely" in describing the role of background O_3 in exceedances of the NAAQS. Instead, it concludes that certain elements of background O_3 significantly increased surface O_3 values in Clark Co., NV during the late spring and early summer of 2013. It did conclude that stratospheric intrusions and, to a lesser extent, wildfires, significantly increased concentrations in this area during this period (Langford et al., 2015, p. 320). The EPA agrees that these sources can influence O_3 concentrations (at times, significantly) and has noted that throughout the rulemaking process. Again, the Exceptional Events Rule allows for the exclusion of air quality monitoring data from design value calculations when monitored exceedances are caused by events such as wildfires and stratospheric intrusions, even if there are multiple instances of these impacts within a season.

f) Some commenters (e.g., NMA) pointed to various studies and claimed they showed exceedances of the NAAQS in various areas based on modelled results exclusively. The studies cited include Zhang et al (2011) which estimated USB to be greater than 60 ppb in some locations; Emery et al (2012) which estimated USB in the "West" to occasionally exceed 60 ppb; and Lefohn et al (2014) which estimated that modeled concentrations at national parks, including Yellowstone, show O_3 concentrations of 90 ppb, of which 90% is contributed by USB.

   First, the EPA modeling has also shown that there can be relatively infrequent events in which background O_3 concentrations can approach or exceed even the level of the current NAAQS (PA, Section 2.4). Similarly, the EPA modeling has also shown that the seasonal mean background can be a large proportion of total O_3 (e.g., 70-80 percent) at certain high-elevation, western locations (PA, Section 2.4). The key consideration is that the locations that are most strongly influenced by background O_3 are relatively limited in scope, i.e., rural areas in the intermountain western U.S. And even despite the large proportional contribution at these limited remote sites, many of these rural locations are still expected to attain a 70 ppb standard. For example, the 2012-2014 O_3 design value at Yellowstone National Park is currently 63 ppb (http://www3.epa.gov/airtrends/values.html). Moreover, the EPA source apportionment modeling estimates that 17 to 26% of the O_3 that is measured at locations in Wyoming originates from U.S. anthropogenic sources (U.S. EPA, 2015). Second, the EPA believes that many of the characterizations of background O_3 in these comments or their original source material are

inaccurate. For instance, the NMA comments reiterate findings from the Zhang et al. (2011) and Emery et al. (2012) modeling studies which show some occurrences of days where background O_3 levels exceed 60 ppb and restate the authors' assertion that States "will have little or no ability to reach compliance through North American regulatory controls" if the NAAQS are lowered into the 60-70 ppb range (Zhang et al., 2011). While these modeling studies certainly advanced our ability to estimate background O_3 levels across the U.S. via the application of global and regional air quality models with finer horizontal resolution, this particular policy conclusion is overly simplistic and demonstrably inaccurate. Again, the CAA contains several provisions to provide regulatory flexibility for States with air quality problems that they cannot control. In particular, any air quality data affected by an "exceptional event" (e.g., a natural event that affects air quality that is not reasonably controllable or preventable) can be excluded from consideration if an air agency can demonstrate that the event clearly caused the exceedance. The Zhang et al (2011) study (p. 6774) indicates that at least some of highest background values are in areas associated with wildfires ("a secondary maximum of 59 ppb over Idaho due to large wildfires"), which would be eligible for exceptional event consideration. It should also be noted that the largest annual 4th-high North American background (NAB)[136] value estimated in the Zhang et al (2011) analysis (p. 6774) is lower than the 70 ppb final standards. Even without consideration of the CAA relief provisions, this particular modeling analysis thus does not show any areas for which 70 ppb NAAQS would be unattainable due to USB.

g) To support the contention that levels of O_3 are high in pristine areas, such as national parks, and that this can only be attributed to background O_3, one commenter refers to a monitoring study prepared by the State of Alaska and characterizes it as showing that O_3 levels in Denali National Park are higher than levels in certain Alaska cities (e.g., NMA comment). Attachment J (p. 45) to NMA's comments shows monitored levels of O_3 in Denali National Park tend to range between 20 and 25 ppb. The study suggests that this is an estimate of naturally occurring O_3 in this region. The EPA does not believe this study provides significant support for any of the propositions asserted in the NMA comments.

h) Several commenters cite to monitoring results in a study prepared by the Utah Department of Air Quality (e.g., NMA comments, Attachment D) and characterize the information as showing "recent background trends demonstrat[ing] significant exceedances of 70 ppb … in rural areas throughout these states." As noted in section II.B.2.a of the preamble to the final rule, attributing monitored exceedances at these Utah sites to background emissions is improper. The study itself refers to emissions from Salt Lake City as being a

[136] While Zhang et al. (2011) estimated NAB levels, as opposed to the more relevant USB, we do not expect that the impacts from emissions in Canada and Mexico would lead to 4th high values greater than 70 ppb, as impacts from Canada and Mexico are typically less than 3 ppb, except in the immediate vicinity of the border (Wang et al., 2009).

significant contributor to O_3 levels in several of the study areas (e.g., NMA comments, Attachment D). Other areas (Toole) reflected monitoring results from a monitor on Badger Island in the Great Salt Lake, which is proximate to Salt Lake City and reflects emissions from that city (e.g., NMA comments, Attachment D). The study likewise notes the proximity of O_3 levels in other monitored rural areas to Provo, Utah, shows that O_3 levels in Provo and the rural areas are correlated, and demonstrates how O_3 concentrations in the rural areas increased when prevailing winds from the direction of Provo were stronger (e.g., NMA comments, Attachment D). The study also notes that O_3 transport from Las Vegas and southern California could be contributing to observed O_3 in the Utah rural sites (e.g., NMA comments, Attachment D). The first bullet on slide 18 of the Utah presentation summarizing the study (e.g., NMA comments, Attachment F) likewise states, under the heading "Factors affecting Rural O_3," that "regional transport" from Las Vegas, Los Angeles and from other areas harder-to-identify is a source of these O_3 levels. Slide 14 of that same presentation indicates that the Great Salt Lake contributes to O_3 formation in rural areas as well. Since the Great Salt Lake abuts many population centers, including Salt Lake City itself, this is another example of regional transport of domestic anthropogenic O_3 contributing as an important source of downwind O_3 levels. The Utah study consequently does not support the commenter's contention that nonattainment results from background O_3 in the enumerated Utah monitoring locations.

i) Finally, a commenter (e.g., NMA) refers to the Nevada Rural Ozone Initiative study (Gustin et al., 2015) as supporting the contention that a revised O_3 standard at levels 70 ppb and lower will prove unattainable in many areas due to background emissions. The commenter notes that the Nevada study monitored in various rural areas, and that these areas are particularly susceptible to high background O_3 levels due to such factors as high elevation, complex terrain, and deep convective mixing (e.g., NMA comments, including Attachment H). The commenter also views the study as confirming the results of the Las Vegas O_3 study discussed in (f) above. The commenter correctly quotes the study as providing information that elevation, meteorological conditions, terrain, and convective mixing were factors in the monitored O_3 levels at these rural sites. The commenter is incorrect in asserting that this study supports its proposition that background emissions result in NAAQS exceedances in these areas. The study indicates that domestic anthropogenic emissions, specifically O_3 transported from heavily-polluted California areas, are a key source of the monitored O_3 levels monitored in these Nevada locations. Thus, the study by Gustin et al (2015) indicated (p. 467) that "regions of California with significant air pollution", such as the San Joaquin Valley and the South Coast Air Basin can impact monitors in rural Nevada.

(2) *Comment:* In expressing the view that EPA should not revise the standard to a level of 70 ppb (or lower), some commenters note a statement made by the Administrator in revising the O_3 NAAQS in 1997 to 80 ppb that a level of 70 ppb was approaching background, stating that the 1997 NAAQS were upheld in part

because of this judgment by EPA. These commenters state they see no reason why the same conclusion is not reached in the current review and based on the same conclusion, they state that EPA cannot set the standard at or below background.

Response: The statement in the 1997 notice of final rulemaking for the primary standard to which the commenters refer was made after the finding by the Administrator that the public health impacts, based on the information available in that review, to be important and sufficiently large as to warrant a standard set as a level of 0.08 ppm, as proposed. The notice described that the Administrator, in considering the views of commenters who argued for a standard level of 0.07 ppm, first recognized that no member of the CASAC panel had supported a standard level lower than 0.08 ppm and that the health effects evidence did not provide strong support for a lower standard level. Lastly, the notice recognized that, as commenters at that time noted, a standard set at a 0.07 ppm level would be "closer" to peak background concentrations that infrequently occur in some areas than the proposed level of 0.08 ppm. We don't disagree with this statement, just as we note that a level of 0.065 ppm is closer to such concentrations than a level of 0.070 ppm. Although it is true that the Administrator in 1997 considered the proximity to background as one factor in not selecting a level of 0.07 ppm, the decision on the primary standard in that review, as in this one, was based primarily on the health effects evidence and the Administrator looked to proximity to background as one reason not to set the standard at the lower end of the range of reasonable levels supported by the air quality criteria and judgments of the Administrator. In this review, the Administrator concluded, based on the updated body of scientific evidence, updated exposure and risk estimates, and updated CASAC advice, that it was appropriate not to set the standard at the lower end of the range of levels supported by the air quality criteria, without considering the issue of proximity to O_3 concentrations from background sources in some areas. Further, as discussed in section II.B.1 of the final rule and expanded upon in the first comment in this section of the RTC, we disagree with the commenters' implications that O_3 concentrations resulting from background sources are of a magnitude as to affect attainment of the revised standard.

(3) *Comment:* Noting the proposal's discussion of the potential for climate change and increased temperatures to increase summertime O_3 concentrations, one comment stated that EPA should not consider the relationship between climate change and O_3 in revising the 2008 O_3 standards. The comment additionally stated that EPA should not propose revisions to the 2008 O_3 standards to address potential future increases in summertime O_3 concentrations related to future increases in temperature.

Response: While the proposal (and final rule) does contain discussion of how climate change may influence future O_3 concentrations, including discussions of modeling studies, this discussion is part of a larger discussion related to understanding and characterizing O_3 air quality, including uncertainties relating to predicting future O_3 levels. The EPA's decisions on revisions to the primary and

secondary standards are not based on possible impacts of climate change on future O_3 concentrations. Rather, consistent with the CAA, the standards are based on the current air quality criteria in this review.

(4) *Comment:* One group of commenters stated that EPA must follow through on its 2011 statement to the D.C. Circuit that it was deferring the completion of its voluntary rulemaking to reconsider the 2008 decision until it completes its statutorily- required periodic review, which was made as part of its motion to dismiss litigation challenging EPA's decision to halt the reconsideration. EPA Mot. to Dismiss at 2, *American Lung Ass'n v. EPA*, No. 11-1396 (D.C. Cir. Dec. 8, 2011). The court granted that motion, accepting EPA's characterization of its action as a "non-final decision to defer action on the 2008 voluntary revision of the national ambient air quality standards for ozone." Order, American Lung Ass'n, No. 11-1396 (D.C. Cir. Feb. 17, 2012); see also *Mississippi*, 744 F.3d at 1341-42 (summarizing history). These commenters assert that EPA must conclude the reconsideration rulemaking, consistent with its representations to the court.

Response: The EPA consolidated the reconsideration with its statutorily required periodic review of the O_3 NAAQS. This rulemaking concludes the reconsideration process. Under CAA section 109, the EPA is required to base its review of the NAAQS on the current air quality criteria, and thus the record and decision for this review also serve for the reconsideration.

(5) *Comment:* Some commenters express the view that CASAC advice should be given less weight by EPA, stating that EPA did not advise CASAC of the role that background O_3 can play in determining the standards. One commenter further states that "[h]ad CASAC been properly advised of the role that background O_3 can – indeed, should- be considered in determining the appropriate level for the NAAQS, the Committee might well have offered different recommendations to EPA concerning revision of the NAAQS" (e.g., UARG, p. 53).

Response: We disagree with the commenters' view that we failed to provide CASAC with the relevant information regarding considerations in setting the O_3 NAAQS that may be relevant to O_3 in ambient air from background sources. In the June 26, 2014 letter from the CASAC to Administrator McCarthy, the CASAC devotes roughly one third of the first page to comments associated with consideration of background in selecting a revised standard, specifically noting that the second draft PA describes both the 2002 court decision on the 1997 standards and the 1981 court decision on the 1979 standards, and describing them as "two legal guidelines" (Frey, 2014b). Further, the transcript of the public meeting on the first draft PA indicates that the issue was discussed prominently by both EPA staff in their presentation to the CASAC, and by the CASAC in its public deliberations (Transcript of September 11-12, 2012 CASAC public meeting in the docket, Document ID EPA-HQ-ORD-2011-0050-0053). This included a discussion of the potential legal bases for considering, and not considering, proximity of a standard to background O_3 levels. Thus, the comment is mistaken in its presumption regarding information provided and considered by

the CASAC. Even if the CASAC had lacked information on background O_3, the EPA does not believe that would warrant less consideration for the Committee's views on the health effects of O_3, especially as "background ozone" is indistinguishable as a chemical matter (and in terms of biological responses) from other O_3.

(6) *Comment:* A number of commenters stated that the CASAC has a statutory obligation to advise the EPA regarding "any adverse public health, welfare, social, economic, or energy effects which may result from various strategies for attainment and maintenance of such national ambient air quality standards", citing section 109(d)(2)(C)(iv) of the Act. At least one commenter seemed to suggest, obliquely, that any such advice must (or should) occur as part of the review of the NAAQS. This issue was also raised in public comments at the public meetings with the CASAC.

Response: The EPA agrees that the Act states that the CASAC should advise the Administrator on potential adverse impacts which may result from various strategies for attainment. The EPA, however, disagrees that such advice must be coincident with, or a pre-condition for, EPA action to revise a NAAQS where revision is "appropriate" (within the meaning of section 109 (d)(1)). Most obviously, there is no such requirement in section 109 (d)(2)(C)(iv). The provision is silent as to when CASAC is to provide such advice. In contrast, where Congress wanted CASAC to provide advice at a particular time and to coordinate timing with NAAQS review cycles, it said so explicitly. See section 109 (d)(2)(B) ("not later than January 1, 1980, and at five-year intervals thereafter, the committee referred to in subparagraph (A) [CASAC] shall complete a review of the criteria published under section [108] … and the national primary and secondary ambient air quality standards promulgated under this section and shall recommend to the Administrator any new national ambient air quality standards and revisions of existing criteria and standards as may be appropriate…"). Moreover, temporal correlation with the NAAQS review is specified when CASAC is to give advice relating to air quality criteria or standard setting (see section 109 (d)(2)(B)). Because the advice in section 109 (d)(2)(C)(iv) does not relate to either air quality criteria or standard setting, no time for providing the advice is specified.

Furthermore, the EPA is legally prohibited from considering costs of implementing the NAAQS in establishing the standards (*Whitman*, 531 U.S. at 456). The Congress, thus, could not have intended for the EPA to receive CASAC advice on "adverse, social, economic, or energy effects which may result from various strategies for attainment and maintenance" during the standard setting process. In this regard, the Supreme Court in *Whitman,* in rejecting arguments that costs may or must be considered in establishing NAAQS, stated that section 109 (d)(2)(C)(iv) "enables the Administrator to assist the States in carrying out their statutory role as primary *implementers* of the NAAQS … It would be impossible to perform that task intelligently without considering which abatement technologies are most efficient, and most economically feasible …." (emphasis

original); see *Whitman* at n. 2 (rejecting argument that CASAC advice on costs generated under section 109 (d)(2)(C)(iv) must be included in NAAQS rulemaking record); see also *ATA I*, 175 F. 3d at 1041 ("[t]he advice required in section 109 (d)(2)(C)(iv) is pertinent only to the EPA's duty under section 108 to provide the States with control strategy information").

The EPA notes further that the issue of potential adverse public health effects which might conceivably result from various alternative standard scenarios that reduce NOx and VOC emissions was considered by the EPA, discussed with CASAC, and addressed by CASAC in its advice to the EPA in this review. See Frey 2014a, pp. 10 and 11; see also 79 FR at 75271, 75279, 75285 at n. 102 and 105, and 75287 n. 107.

(7) *Comment:* Several commenters stated that a revised standard set at a more stringent level than the current standard would result in adverse health impacts, with one citing "socio-economic disruption" with "attendant health impacts" and that "[t]here is no legal basis for EPA to disregard such adverse health impacts" (e.g., NMA, pp. 21, 24).

Response: We disagree with these commenters and note that a unanimous Supreme Court has rejected this argument. In holding that "the text of section 109 (b) [the NAAQS standard-setting provision] unambiguously bars cost considerations from the NAAQS-setting process", the Court explained that "the economic cost of implementing a very stringent standard might produce health losses sufficient to offset the health gains achieved in cleaning the air – for example, by closing down whole industries and thereby impoverishing the workers and consumers dependent upon those industries. That is unquestionably true, and Congress was unquestionably aware of it" (*Whitman*, 531 U.S. at 471, 465). The D.C. Circuit reached the identical conclusion: "AISI next contends that EPA erred in refusing to consider the health consequences of unemployment in determining the primary standard for particulate matter. This claim is entirely without merit. In three previous cases the court has emphatically stated that section 109 does not permit EPA to consider such costs in promulgating national ambient air quality standards" (*Natural Resources Defense Council v. EPA*, 902 F. 2d 962, 973 [D.C. Cir. 1990]); see also *Whitman*, 531 U.S. at 464 (approvingly citing this portion of *NRDC*)). Accordingly, the EPA has not considered the costs suggested by the commenters

In fact, were the EPA to act on the commenter's suggestion it would be grounds for vacating the NAAQS, since the EPA would have considered an impermissible factor and thereby not followed the law (*Whitman*, 531 U.S. at 472 n. 4). The EPA has not considered the costs of attainment in its determination to revise the O_3 NAAQS. However, we have considered whether there is a factual predicate for the commenter's assertion that a revised standard would lead to socio-economic disruption. To support this assertion, the commenter cites studies of NERA, *Economic Consulting, Assessing Economic Impacts of a Stricter NAAQS for O_3* (July 2014) and *Economic Impacts of a 65 ppb National Ambient Air Quality*

Standard for Ozone (February 2015) (e.g., NMA Comment, attachments T and U, evaluating only economic costs of standards established at levels of 60 ppb and 65 ppb). However, these studies do not establish a predicate for the commenter's argument. For example, the studies consider impacts of standards stricter than the one EPA is adopting and estimate economic impacts using a proprietary model that is not transparent. Furthermore, this model is not appropriate for estimating involuntary unemployment because the model is based on the assumption of full employment, i.e. it assumes that everyone who wants a job can find one, and therefore reallocates labor displaced from one sector to other sectors. Because the model gives no direct estimate of unemployment, the NERA studies use an alternate approach, estimating changes in overall labor income and dividing this by the average wage to estimate losses of so-called "job equivalents". Some have misinterpreted these estimates as actual losses in jobs expected in the economy. This is not an appropriate interpretation of the results of the NERA studies. Moreover, NERA made unreasonable assumptions about control strategies available to states to reach attainment with more stringent standards. NERA assumed states would pursue only two policies to achieve emissions reductions needed beyond known measures: coal plant retirements and passenger vehicle retirements. There are many options available to states to reach attainment with the standards, however, these studies appear to have chosen some of the least cost-effective measures possible for their analyses. The commenter's factual premise for its legal argument thus is lacking.

(8) *Comment:* A few commenters stated that EPA should identify and propose a specific standard, rather than a range, for public comment. In stating this, these commenters indicated their view that the Administrator lacked conclusive science to select new NAAQS.

Response: There is no requirement in CAA section 109 or 307(d) that the EPA propose a single standard or that it propose a range. Rather, what matters is that the EPA provides clear notice of the range of alternative views under consideration and the reasoning behind a potential choice. The Agency did so in this proposal (e.g., 79 FR 675308-310) and also explained how the reasoning is supported by the air quality criteria.[137] Furthermore, as a matter of administrative practice, although we recognize that there are times when the Administrator may propose a single option, we also note that it is quite common in NAAQS reviews for the Administrator to propose a range of options for a revised standard. Examples of this latter approach including the most recent proposals for the NAAQS for lead and particulate matter and the primary NAAQS for nitrogen dioxide and sulfur dioxide (73 FR 29184, May 20, 2008; 74 FR 34404, July 15, 2009; 74 FR 64810, December 8, 2009; 77 FR 38890; June 29, 2012).

[137] It is well-established that the Administrator is not to wait for "conclusive" science to set new NAAQS. Rather, EPA is to set NAAQS in part to protect "against hazards which research has not yet identified" (*Natural Res. Def. Council v. EPA*, 824 F.2d 1146, 1152 (D.C. Cir. 1987) (en banc) (quoting S. Rep. No. 91-1196, at 10 (1970))).

(9) *Comment:* One commenter claims that the EPA's use of the PA in the process for reviewing the NAAQS contributes to a lack of transparency, which in their view would be addressed by its replacement with an Advance Notice of Proposed Rulemaking. Based on this view, they recommend that EPA reinstate the ANPR, which EPA used in one NAAQS review in the past few decades. They state that the ANPR approach ensured that decisions about the NAAQS were informed by the best available science and provided for broad participation among experts in the scientific community. In contrast, they indicate their view that the PA does not provide for oversight or input from the outside scientific community and allows bias to enter the process, biasing the information that the Administrator is provided for her consideration.

Response: We disagree with the commenters' view that the PA does not provide for oversight or input from the scientific community and the public, and note that two drafts of the O_3 PA were made available for public comment and reviewed by the CASAC at multiple public meetings. This extent of public process and scientific review is well beyond that associated with release of an ANPR. The resultant final PA as well as CASAC's advice regarding it are provided to the Administrator to inform her decisions in the review. The commenters provide no documentation or basis for their view that the use of the PA allows bias to enter the process, and given the extent of public process and scientific review, we find no evidence for that assertion.

(10) *Comment:* Some commenters have stated that the EPA should make all the scientific assessments and data on which those assessments are based that it relied upon in deciding to revise the O_3 NAAQS, available to the public and/or for peer review, claiming that the EPA has not complied with federal law and Presidential directives that require this. One commenter additionally stated their view that the EPA has not made all pertinent data and studies public, citing a U.S. House of Representatives, Science, Space and Technology Committee subpoena with which the commenter claims the EPA has not complied.

Response: Contrary to the implication of these comments, the EPA has made available all scientific analyses on which it based its decision for review of the NAAQS. The process that the EPA follows in each NAAQS review provides for transparency, including with regard to the information on which decisions are based. The NAAQS review process includes extensive external scientific peer review in the public eye, and multiple public comment periods. For example, three drafts of the ISA, and two drafts each of the HREA, WREA and PA were released for public comment and review by the CASAC. The CASAC conducted their review in multiple public meetings at which they provided time for public comment. The draft documents, letters from the CASAC with its reviews, more than 100 comments from the public on the draft documents, and transcripts from the CASAC public meetings on the draft ISAs are in the docket for this rulemaking. The final HREA, WREA and PA include extensive appendices providing detailed information regarding analyses, and EPA has provided underlying datasets for these analyses explicitly within the documents, by

submissions to the regulatory docket on the EPA website and/or by request to interested parties.

With regard to the U.S. House committee subpoena concerning data owned by third parties who conducted several epidemiological studies on particulate matter and O_3 that the EPA partially funded, the EPA has, contrary to the implications of the commenter, fully responded to the subpoena. Consistent with the Shelby Amendment and implementing regulations, the EPA requested from the third parties the study data that were developed with EPA funding and provided Congress with all of the third-party data that the Agency received. In addition, the EPA has identified approaches that are available to interested researchers for collaborating with the third-party owners of the datasets or for gaining access to the datasets for the purposes of further analyses.

(11) *Comment:* Some commenters state that it is unreasonable to revise the O_3 NAAQS before the 2008 standards have been fully implemented, noting that many states are still addressing milestones associated with the 2008 rulemaking. Additionally, one comment states that the EPA's proposal relies on modeled estimates potential exposures and risks that may result from the current standards, which the EPA would not have to rely on –in lieu of data- if it would fully implement the current standards.

Response: The only issue before the EPA in considering whether revision of current NAAQS is needed is the adequacy of the current standards. The state of implementation of the existing (2008) standards is irrelevant in making that determination since it has nothing to do with whether the air quality allowed by the current standards provides the requisite public health and public welfare protection. As noted in section I.C of the preamble for the final rule, the decisions in this NAAQS review are based on the current air quality criteria and associated quantitative analyses, consistent with CAA section 109. Under the commenters' logic, no matter how inadequate the public health or public welfare protection provided by existing standards, the associated air quality would be allowed to continue, without improvement, based on the state of the implementation process. The Act does not allow such considerations (*Whitman*, 531 U.S. at 468).

(12) *Comment:* One commenter states that the EPA has not appropriately considered and complied with several executive orders. More specifically, this commenter states that although EPA is capable of modeling the likely extent of nonattainment areas and analyzing the impact of the proposed standards within relevant timeframe, EPA has chosen not provide "any more than an 'illustrative analysis'", which this commenter states to be inapposite to the letter and spirit of executive orders (EOs) 12866 and 13563. As a result, this commenter states that the proposed rule violates the goals of these two EOs. The commenter also states that EPA should reassess its position with respect to the applicability of EOs 13132, 13211 and 13604.

Response: The commenter raises concerns about how and whether the executive orders would apply to actions that could be taken to implement the revised ozone NAAQS. However, this rule merely establishes the NAAQS, and EPA has fully complied with relevant executive orders to the extent they apply to the establishment of NAAQS (see section VIII of the proposal preamble and section IX of the final rule preamble). States have the primary role under the Clean Air Act in selecting and implementing strategies to attain the NAAQS. Because the States have broad discretion in adopting strategies to implement the NAAQS, the possible future effects of such implementation activities are beyond the scope of this rulemaking, and EPA is only able to provide an illustrative analysis of potential costs and benefits in the RIA. See also, *American Trucking Assoc. vs. EPA*, 175 F.3d 1027, 1034-1045 (D.C. Cir. 1999).

(13) *Comment:* Some tribal commenters object to the EPA's approach to considering tribal implications under EO 13175, expressing the view that no CAA regulatory action would ever be found to have tribal implications under the approach used by EPA, and recommending that the EPA consult with Tribes on a government-to-government basis regarding the proposed rule. This commenter states their view that the proposed rule has implications to Indian tribes because, in the view of the commenter, the proposed standards may not be stringent enough for Tribes who the commenter describes to be at greater risk of impacts to their health or welfare.

Response: Executive Order 13175, entitled ''Consultation and Coordination with Indian Tribal Governments'' (65 FR 67249, November 9, 2000), requires the EPA to develop an accountable process to ensure ''meaningful and timely input by tribal officials in the development of regulatory policies that have tribal implications.'' It provides that "'policies that have tribal implications' refers to regulations, legislative comments or proposed legislation, and other policy statements or actions that have substantial direct effects on one or more Indian tribes, on the relationship between the Federal Government and Indian tribes, or on the distribution of power and responsibilities between the Federal Government and Indian tribes." This NAAQS rulemaking establishes stronger national standards to address the health and welfare effects of O_3, providing increased protection for sensitive groups from adverse effects to public health, with an adequate margin of safety, and increased protection of public welfare from known or anticipated adverse effects. Tribes are not obligated to implement these standards, or to conduct monitoring or adopt monitoring requirements, such that no direct requirements are placed on tribes by this action. This NAAQS rulemaking does not have tribal implications, as specified in Executive Order 13175, and is therefore not subject to the Executive Order.[138] Even if this action were determined to have tribal implications within the meaning of that Executive Order, it will neither impose substantial direct compliance costs on tribal

[138] Contrary to some commenters' assertion that EPA's approach would result in no CAA action being found to have tribal implications, some CAA regulatory actions have been found to have tribal implications under E.O. 13175.

governments, nor preempt tribal law, and therefore consultation under the Executive Order was not required.

Nonetheless, under the "EPA Policy on Consultation and Coordination with Indian Tribes," EPA consults on a government-to-government basis with federally recognized tribal governments when EPA actions and decisions may affect tribal interests. Accordingly, we offered to consult with tribes by sending letters to tribal leadership and inviting consultation on the proposed rule. No tribe requested a formal government-to-government consultation with the EPA.

Although no tribe requested formal consultation, we undertook a number of outreach activities to inform tribal environmental professionals about the O_3 NAAQS review, which are described in the "OAR Handbook for Interacting with Tribal Governments." We conducted outreach and information calls to tribal environmental staff and other stakeholders on December 2 and 17, 2015, and we participated in the National Tribal Air Association call on December 18, 2014.

In addition, during the public comment period, we received comments on the proposed rule from seven tribes and three tribal organizations. All significant comments are addressed in the preamble to the final rule or in this RTC.[139]

(14) *Comment:* Some tribal commenters state that under EO 12898, the EPA needs to conduct an environmental justice analysis of the proposed rule and its impacts to Tribes, or claim that revision to even the lowest level in proposed range (65ppb) "will still result in a disproportionate impact on Native Americans and Alaska Natives with Asthma", that revision to 60 ppb would allow EPA to comply with its EJ policies to better protect Native American Tribes and that EPA should complete a thorough EJ analysis to ensure reviewed NAAQS will provide best protection of Tribes and indigenous peoples.

Response: The commenter has provided no evidence that revision of the O_3 standards to a level of 70 or 65 ppb will result in a disproportionate impact on native Americans and Alaska natives with asthma or that revision to 60 ppb would not, and the EPA is not aware of such evidence. Further, contrary to the comment, and consistent with the assessment conducted in each NAAQS review, the EPA has evaluated the available evidence with regard to populations that may be at greater risk of O_3 health effects than the general population. That assessment, described in the ISA, identified asthmatics as an at-risk population bud did not identify native Americans as an at-risk population. The term "at-risk populations" is used to recognize populations that have a greater likelihood of experiencing O_3-related health effects (sometimes referred to as sensitive groups). Thus, revision of the standard to protect the identified at-risk populations would be expected to also provide protection for other groups.

[139] The aspect of this comment involving the tribal groups' view that tribal members are at increased risk of health effects from ozone is addressed below and in section II.A above.

(15) *Comment:* A few commenters state that EPA's environmental justice analysis is inadequate or that the O_3 proposal fails to address the requirements of E.O. 12898 on Environmental Justice and fails to follow EPA's strategy to achieve Environmental Justice. One comment additionally states that the proposal does not include a meaningful evaluation of the EJ implications of alternative regulatory proposals and formally requests EPA to conduct a more detailed analysis of the impact of different regulatory alternatives on racial minority and low-income pops and to "incorporate" these findings into its final regulatory proposal. This comment additionally states that EPA fails (under Title VI of 1964 Civil Rights Act) to provide guidance to federally-funded state partners regarding their obligations to incorporate EJ into their activities to reduce O_3 forming pollution.

Response: As described in section I.A of the final rule, the NAAQS must protect public health with an adequate margin of safety, including for sensitive groups (or populations) as well as the general populace. Minority populations, low-income populations and/or indigenous peoples are often such sensitive populations. The primary O_3 standard established in today's final rule is a nationally uniform standard which in the Administrator's judgment is requisite to protect public health, including the health of sensitive groups (also termed at-risk populations), with an adequate margin of safety. As discussed in section II of the preamble to the final rule, in other comment responses, and as summarized in section IX.J of the preamble to the final rule, the EPA expressly considered the available information regarding O_3 exposure and health effects among sensitive populations, including low income and minority populations, in making this determination. The ISA, HREA, and PA for this review, which include identification of populations at risk from O3-related health effects, are available in the docket, EPA-HQ-OAR-2008-0699. In accordance with E.O. 12898, EPA has considered whether the decisions promulgated in the final rule may have disproportionate negative impacts on minority populations, low-income populations or indigenous peoples. This rule establishes a primary O_3 standard is more protective than the current standard and this rule is not expected to have disproportionate negative impacts on minority or low-income populations. Rather, the EPA expects that the revised O_3 standards will reduce health risks in the areas subject to the highest ambient air concentrations of O_3.

To the extent any of the commenters is suggesting E.O. 12898 requires additional quantitative analysis or assessment of environmental justice issues related to revising the O_3 NAAQS, or that the standard should be set more stringent than necessary to protect the health of sensitive and other groups with an adequate margin of safety, EPA disagrees.

This rule establishes the revised O_3 NAAQS. States have primary response for implementing the NAAQS and implementation plans are beyond the scope of this rulemaking. However, EPA notes that recipients of EPA financial assistance must comply with all federal nondiscrimination statutes that together prohibit discrimination on the bases of race, color, national origin (including limited-

English proficiency), disability, sex and age. These laws include: Title VI of the Civil Rights Act of 1964; Section 504 of the Rehabilitation Act of 1973; Section 13 of the Federal Water Pollution Control Act Amendments of 1972; Title IX of the Education Act Amendments of 1972; and the Age Discrimination Act of 1975.

EPA's Office of Civil Rights (OCR) is responsible for carrying out compliance with these federal nondiscrimination statutes and does so through a variety of means including: complaint investigation; agency-initiated compliance reviews; pre-grant award assurances and audits; and technical assistance and outreach activities. Anyone who believes that any of the federal nondiscrimination laws enforced by OCR have been violated by a recipient of EPA financial assistance may file an administrative complaint with EPA's OCR.

(16) *Comment*: Numerous comments were received regarding significant issues associated with the RIA, including the scope, assumptions and methodology, and additional analyses suggested. Additionally, in support of their view that the current standard should not be changed, many commenters state that costs of new standards will impose detrimental effect on economy, that net O_3 benefits are negative, only 30% of RIA benefits are from O_3. The EPA understates cost and overstates benefits. Further, some commenters state that fact sheets, the RIA and summary materials released to the public with the notice of proposed rulemaking provided a misleading presentation of health benefits and how revised standards will be attained and that EPA must correct this. Citing to the RIA, some of these commenters further state that the O_3 NAAQS rulemaking is inappropriately based on inadequately established assumptions (unsubstantiated assumptions of benefits obtained from other rules that are unlikely to survive judicial review), improper reliance on waivers, reliance on unknown controls vis-à-vis the States, and EPA's failure to adequately assess the effects of the Proposed Rule on other proposed rules.

Response: As noted in section I.A of the preamble, the CAA bars consideration of costs in determining whether it is appropriate to revise a NAAQS or how to revise it if implementation is appropriate (within the meaning of the CAA section 109(d)(1)). Accordingly, the EPA has not considered costs, including the costs or economic impact of implementation or compliance, in revising the O_3 NAAQS. For the same reason, comments on the RIA were not considered in those decisions. Similarly, any fact sheets and summary materials drawn from the RIA were not considered in EPA's decisions on the O_3 standards, and comments on the RIA were not considered in the decisions. Indeed, fact sheets are public communication tools only, and are not part of the administrative record for the decision whether to revise the NAAQS and if so, how. Comments on the proposal RIA were considered, as appropriate, in developing the RIA for the final rule, and materials associated with the RIA for the final rule will reflect that consideration.

IV. References

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Appendix A. Studies cited in public comments related to the primary standard that were not included in the 2013 O_3 ISA or prior AQCDs and are provisionally considered in responding to the comments

Studies cited in public comments or otherwise identified by EPA while responding to comments related to the primary standard that were not included in the 2013 O_3 ISA (e.g., published after document closure) or prior AQCDs. These studies were provisionally considered by EPA, as discussed in section I.C of the preamble to the final rule and in this RTC.

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**Non peer-reviewed reports, review articles, commentaries, and editorials are not generally considered for inclusion in an ISA.*

Appendix B. Studies cited in public comments related to the secondary standard that were not included in the 2013 O_3 ISA or prior AQCDs and are provisionally considered in responding to the comments

Studies cited by public commenters related to the secondary standard that were not included in the 2013 O_3 ISA (e.g., published after document closure) or prior AQCDs. These studies were provisionally considered by EPA, as discussed in section I.C of the preamble to the final rule and in this document

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**Although these studies are not focused on the effects of O_3 and are outside the scope of the air quality criteria, we have provisionally considered them in the context of the comments.*