



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

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EPA-452/R-14-006

August 2014

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

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List of Acronyms and Abbreviations

Act	Clean Air Act
ACS	American Cancer Society
AHR	Airway hyperresponsiveness
ANF	Atrial natriuretic factor
AOT40	Seasonal sum of the difference between an hourly concentration at the threshold value of 40 ppb, minus the threshold value of 40 ppb
AOT60	Seasonal sum of the difference between an hourly concentration at the threshold value of 60 ppb, minus the threshold value of 60 ppb
APEX	Air Pollutants Exposure model
APHEA	Air Pollution and Health: A European Approach
APHENA	Air Pollution and Health: A European and North American Approach
AQCD	Air Quality Criteria Document
AQI	Air Quality Index
AQRV	Air quality related value
AQS	Air Quality System
ARG	Arginase
AspenFACE	Aspen Free Air gas Concentration Enrichment Facility
ATS	American Thoracic Society
BALF	Bronchoalveolar Lavage Fluid
BI	Biosite Index
BRFSS	Behavioral Risk Factor Surveillance System
C	Concentration
CAA	Clean Air Act
CAL FIRE	California Department of Forestry and Fire Protection
CAMx	Comprehensive Air Quality Model with Extensions
CAMP	Childhood Asthma Management Program
CAR	Centriacinar region
CASAC	Clean Air Scientific Advisory Committee
CASTNET	Clean Air Status and Trends Network
CAT	Catalase
CBSA	Core-based statistical area
CD	Criteria Document
CDC	Centers for Disease Control
CFR	Code of Federal Regulations
CH ₄	Methane
CHD	Coronary Heart Disease
CHF	Congestive Heart Failure
CHS	Children's Health Study
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

CSA	Combined Statistical Area
CSTR	Continuous stirred tank reactor
CVD	Cardiovascular disease
DHEW	Department of Health, Education, and Welfare
ED	Emergency department
ELF	Extracellular Lining Fluid
EPA	Environmental Protection Agency
E-R	Exposure-response
eVNA	Enhanced Voronoi Neighbor Averaging
FACE	Free-air CO ₂ (and ozone) enrichment system
FASOMGHG	Forest and Agricultural Optimization Model – Greenhouse gas version
FEM	Federal Equivalent Method
FeNO	Exhaled nitric oxide fraction
FEV ₁	Forced Expiratory Volume for 1 second
FHM/FIA	Forest Health Monitoring /Forest Inventory and Analysis Program
FHWAR	National Survey of Fishing, Hunting, and Wildlife-Associated Recreation
FIA	USDA Forest Inventory and Analysis Program
FLAG	Federal Land Managers' Air Quality Related Values Workgroup
FR	Federal Register
FRM	Federal Reference Method
FVC	Forced Vital Capacity
GEOS	Goddard Earth Observing System
GIS	Geographic Information Systems
GRSM	Great Smoky Mountains National Park
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
HO	Heme oxygenase
HR	Heart rate
HREA	Health Risk and Exposure Assessment
HRV	Heart rate variability
ICD-9	International Classification of Disease - 9 th revision
ICU	Intensive care unit
IgE	Immunoglobulin E
IL	Interleukin
I/R	Ischemia-reperfusion
ISA	Integrated Science Assessment
Max	maximum
MDA8	Maximum daily 8-hour ozone average
MMTCO ₂ e	Million metric tonnes of carbon dioxide equivalents
MPO	Myeloperoxidase
MSA	Metropolitan Statistical Area
NAAQS	National ambient air quality standards

NAB	North American background
NB	Natural background
NCDC	National Climatic Data Center
NCLAN	National Crop Loss Assessment Network
NCORE sites	National Core multi-pollutant monitoring sites
NHLBI	National Heart, Lung, and Blood Institute
NMMAAPS	National Morbidity, Mortality, and Air Pollution Study
NO	Nitric oxide
NO ₂	Nitrogen Dioxide
NO _x	Nitrogen Oxides
NOAA	National Oceanic and Atmospheric Administration
NQO1	NAD(P)H-quinone oxidoreductase genotype
NRC	National Research Council
NRCS	Natural Resources Conservation Service
NSRE	National Survey on Recreation and the Environment
NWR	National wildlife refuges
O ₃	Ozone
OC	Organic carbon
OIF	Outdoor Industry Foundation
OIRA	Office of Information and Regulatory Affairs
OMB	Office of Management and Budget
OR	Odds ratio
OTC	Open-top chamber
PA	Policy Assessment
PAMS	Photochemical Assessment Monitoring Stations
PAN	Peroxyacetyl nitrate
PAPA	Public Health and Air Pollution in Asia
PDSI	Palmer Drought Severity Index
PEFR	Peak Expiratory Flow Rate
ppm	Parts per million
ppm-hrs	part 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 micrometers (μm) in diameter
PMN	Polymorphonuclear leukocyte
POMS	Portable Ozone Monitoring System
PRB	Policy relevant background
QA	Quality assurance
RBL	Relative biomass loss
ROMO	Rocky Mountain National Park
RR	Relative risk
SEKI	Sequoia and Kings Canyon National Parks
SES	Socioeconomic status
SIP	State Implementation Plans
SLAMS	State and Local Monitoring Stations

SO ₂	Sulfur Dioxide
SO _x	Sulfur Oxides
SoyFACE	Soybean Free Air gas Concentration Enrichment Facility
SP	Staff Paper
SPMS	Special Purpose Monitoring Stations
STE	Stratospheric-tropospheric exchange
SUM00	Season sum of all hourly average concentrations
SUM06	Seasonal sum of all hourly average concentrations ≥ 0.06 ppm
TLC	Total Lung Capacity
TNF	Tumor Necrosis Factor
UNEP	United Nations Environmental Programme
U.S.	United States
USB	United States background
UV	Ultraviolet
\dot{V}_E	Ventilation rate
VNA	Voronoi neighbor Averaging
VOC	Volatile Organic Compounds
W126	Cumulative integrated exposure index with a sigmoidal weighting function
WHO	World Health Organization
wRBL	Weighted relative biomass loss
WREA	Welfare Risk and Exposure Assessment
WTP	Willingness to pay

EXECUTIVE SUMMARY

This Policy Assessment (PA) has been prepared by staff in the Environmental Protection Agency's (EPA) Office of Air Quality Planning and Standards (OAQPS) as part of the Agency's review of the primary (health-based) and secondary (welfare-based) national ambient air quality standards (NAAQS) for ozone (O₃). The current O₃ standards were established in 2008 at the end of the previous review cycle. These standards include a primary O₃ standard of 75 ppb,¹ and a secondary O₃ standard set identical to the primary standard. These 2008 standards are now under review, as required by sections 108 and 109 of the Clean Air Act (Act). The PA presents analyses and staff conclusions regarding the policy implications of the key scientific and technical information that informs this review. Staff conclusions are presented regarding the adequacy of the current standards and potential alternative standards appropriate for consideration. Staff analyses in this PA are based on the scientific and technical information, including the uncertainties and limitations related to this information, assessed and presented in the Integrated Science Assessment for Ozone (ISA), the Health Risk and Exposure Assessment for Ozone (HREA), and the Welfare Risk and Exposure Assessment for Ozone (WREA). The PA is intended to "bridge the gap" between the relevant scientific evidence and technical information and the judgments required of the EPA Administrator in determining whether to retain or revise the current standards. Development of the PA is also intended to facilitate advice and recommendations on the standards to the Administrator from an independent scientific review committee, the Clean Air Scientific Advisory Committee (CASAC), as provided for in the Act. Staff considerations and conclusions in this final PA have been informed by comments and recommendations from CASAC, and by public comments.

Health Effects and Review of the Primary Standard

A longstanding and comprehensive evidence base, stronger now than in the last review, documents the effects of O₃ exposures on human health. It is well-understood that secondary oxidation products, which develop as a result of O₃ exposure, initiate numerous responses at the cellular, tissue, and whole organ level of the respiratory system. These key initiating events have the potential to result in a variety of adverse respiratory effects, as well as effects outside the respiratory system (e.g., cardiovascular effects). Ozone inhalation poses the greatest risk to people in certain lifestages (i.e., children, older adults), people with asthma, people with certain genetic variants (related to oxidative stress and inflammation), people with diets limited in

¹ The level of the O₃ standard is specified as 0.075 ppm rather than 75 ppb. However, in this PA we refer to ppb, which is most often used in the scientific literature and in the ISA, in order to avoid the confusion that could result from switching units when discussing the evidence in relation to the standard level.

certain nutrients (antioxidant vitamins C and E), and people experiencing the largest exposures (e.g., outdoor workers, children). The evidence from animal toxicology and controlled human exposure studies indicates that higher exposure concentrations and repeated exposures lead to a greater prevalence of effects and increasingly severe effects, including increased susceptibility to other respiratory stressors, among exposed populations, especially these at-risk populations.

As an initial matter in this PA, staff concludes that reducing ambient O₃ concentrations to meet the current standard of 75 ppb will provide important improvements in public health protection. This initial conclusion is based on (1) the strong body of scientific evidence indicating a wide range of adverse health outcomes attributable to exposures to O₃ concentrations found in the ambient air and (2) estimates indicating decreased O₃ exposures and health risks upon meeting the current standard, compared to recent air quality.

Strong support for this initial conclusion is provided by controlled human exposure studies of respiratory effects, and by quantitative estimates of exposures of concern and lung function decrements based on information in these studies. Analyses in the HREA estimate that the percentages of children (i.e., all children and children with asthma) in urban case study areas² experiencing exposures of concern, or experiencing abnormal and potentially adverse lung function decrements, are consistently lower for air quality that just meets the current O₃ standard than for recent air quality. The HREA estimates such reductions consistently across the urban case study areas and across years evaluated, and throughout various portions of individual urban case study areas, including in urban cores and in the portions of case study areas surrounding urban cores. These reductions in exposures of concern and O₃-induced lung function decrements reflect consistent reductions in relatively high O₃ concentrations (i.e., those in the upper portions of the distribution of ambient concentrations) following reductions in precursor emissions to meet the current standard. Thus, populations in both urban and non-urban areas would be expected to experience important reductions in O₃ exposures and O₃-induced lung function risks upon meeting the current standard.

Support for this initial conclusion is also provided by estimates of O₃-associated mortality and morbidity based on application of concentration-response relationships from epidemiologic studies to air quality adjusted to just meet the current standard. While these estimates are associated with uncertainties that complicate their interpretation, they suggest that O₃-associated mortality and morbidity would be expected to decrease nationwide following reductions in precursor emissions to meet the current O₃ standard.

² HREA analyses for exposures of concern and for risk of moderate or large lung function decrements covered 15 urban case study areas. HREA analyses of mortality and morbidity endpoints from epidemiologic studies covered 12 urban case study areas. Exposures and risks were evaluated for the years 2006 through 2010.

While meeting the current O₃ standard is estimated to result in important public health improvements compared to recent air quality, staff further concludes that the O₃-attributable health effects estimated to be allowed by air quality that meets the current primary standard can reasonably be judged important from a public health perspective. This conclusion is based on consideration of: (1) the scientific evidence discussed in the ISA, including controlled human exposure studies reporting abnormal or adverse respiratory effects following exposures to O₃ concentrations below the level of the current standard and epidemiologic studies indicating associations with morbidity and mortality for air quality that would likely meet the current standard; (2) HREA estimates of O₃ exposures of concern, O₃-induced lung function risks, and O₃-associated morbidity and mortality risks; (3) advice received from CASAC based on their review of draft versions of the ISA, HREA, and PA, and advice received in previous reviews; and (4) staff consideration of public comments. Staff reaches the overall conclusion that the available health evidence and exposure/risk information call into question the adequacy of the public health protection provided by the current standard.

Given this conclusion regarding the adequacy of the current standard, staff also reaches conclusions for the Administrator's consideration regarding the elements of potential alternative primary O₃ standards that could be supported by the available evidence and exposure/risk information. Any such potential alternative standards should protect public health against effects associated with exposures to O₃, alone or in combination with related photochemical oxidants, taking into account the available scientific evidence and exposure/risk information. In reaching conclusions about the range of potential alternative standards appropriate for consideration, staff is mindful that the Act requires primary standards that, in the judgment of the Administrator, are requisite to protect public health with an adequate margin of safety. In setting a primary standard that is "requisite" to protect public health, the EPA's task is to establish standards that are neither more nor less stringent than necessary. The requirement that primary standards provide an "adequate margin of safety" is intended to address uncertainties associated with inconclusive scientific and technical information. Thus, the Act does not require that primary NAAQS be set at zero-risk levels, but rather at levels that reduce risk sufficiently to protect public health with an adequate margin of safety.

The degree of public health protection provided by any NAAQS results from the collective impact of the elements of the standard, including the indicator, averaging time, form, and level. Staff's conclusions on each of these elements are summarized below.

- (1) **Indicator:** It is appropriate to continue to use O₃ as the indicator for a standard that is intended to address effects associated with exposure to O₃, alone or in combination with related photochemical oxidants. Based on the available information, staff concludes that

there is no basis for considering any alternative indicator at this time. Meeting an O₃ standard can be expected to provide some degree of protection against potential health effects that may be independently associated with other photochemical oxidants, even though such effects are not discernible from currently available studies indexed by O₃ alone. Staff notes that control of ambient O₃ concentrations is generally understood to provide the best means of controlling photochemical oxidants, and thus of protecting against effects that may be associated with individual species and/or the broader mix of photochemical oxidants. CASAC concurred with these conclusions.

(2) **Averaging time:** It is appropriate to consider retaining the current 8-hour averaging time for the primary O₃ standard.

(a) Staff concludes that an 8-hour averaging time remains appropriate for addressing health effects associated with short-term exposures to ambient O₃. An 8-hour averaging time is similar to the exposure periods evaluated in controlled human exposure studies, including recent studies reporting respiratory effects following exposures to O₃ concentrations below the level of the current standard. In addition, epidemiologic studies provide evidence for health effect associations with 8-hour O₃ concentrations, as well as with 1-hour and 24-hour concentrations. Staff concludes that a standard with an 8-hour averaging time (combined with an appropriate standard form and level) would be expected to provide substantial protection against health effects attributable to 1- and 24-hour exposures. CASAC concurred, concluding that the current 8-hour averaging time is justified by the combined evidence from epidemiologic and clinical studies.

(b) Staff also concludes that a standard with an 8-hour averaging time can provide protection against respiratory effects associated with longer term O₃ exposures. Air quality analyses indicate that just meeting an 8-hour standard with an appropriate level (i.e., 70 to 60 ppb, as discussed below) would be expected to maintain long-term O₃ concentrations (i.e., seasonal average of 1-hour daily max) below those where a key study indicates the most confidence in the concentration-response relationship with respiratory mortality. In addition, risk analyses in the HREA estimate that just meeting such alternative 8-hour standards would be expected to decrease the incidence of respiratory mortality associated with long-term O₃ concentrations. In considering other long-term O₃ metrics evaluated in recent health studies, analyses in the HREA indicate that the large majority of the U.S. population lives in locations where reducing precursor emissions would be

expected to decrease warm season averages of daily 8-hour ambient O₃ concentrations, a long-term metric used in several recent studies reporting associations with respiratory morbidity. Taken together, these analyses suggest that a standard with an 8-hour averaging time, coupled with the current 4th-highest form and an appropriate level (discussed below), could provide appropriate protection against the long-term O₃ concentrations reported to be associated with respiratory morbidity and mortality. CASAC concurred, concluding that the 8-hour averaging time provides protection against the adverse impacts of long-term O₃ exposures.

- (3) **Form:** For an 8-hour O₃ standard with a revised level, as described below, it is appropriate to consider retaining the current form, defined as the 3-year average of the annual 4th-highest daily maximum concentration. Staff notes that this form was selected in 1997 and 2008 in recognition of the public health protection provided, when coupled with an appropriate averaging time and level, combined with the stability provided for implementation programs. The currently available evidence and exposure/risk information do not call into question these conclusions from previous reviews. CASAC concurred with this conclusion, agreeing that the current form, combined with the current 8-hour averaging time, provides health protection while allowing for atypical meteorological conditions that can lead to abnormally high ambient O₃ concentrations which, in turn, provides programmatic stability.
- (4) **Level:** The available scientific evidence and exposure/risk information provide strong support for considering a primary O₃ standard with a revised level in order to increase public health protection, including for at-risk populations and lifestyles. Staff concludes that it is appropriate in this review to consider a revised primary O₃ standard level within the range of 70 ppb to 60 ppb. A standard set within this range would result in important improvements in public protection, compared to the current standard, and could reasonably be judged to provide an appropriate degree of public health protection, including for at-risk populations and lifestyles. In its advice to the Administrator, CASAC also concluded that the scientific evidence and exposure/risk information support consideration of standard levels from 70 to 60 ppb. Within this range, CASAC concluded that a level of 70 ppb would provide little margin of safety and, therefore, provided the policy advice that the level of the O₃ standard should be set below 70 ppb.

The Administrator's consideration of specific standard levels will reflect her judgments as to the appropriate weight to be given to various aspects of the scientific evidence and exposure/risk information, including the appropriate weight to be given to important uncertainties. To inform these judgments, staff considers what the evidence and information indicate with regard to the degree of public health protection that could be achieved with levels from the upper (70 ppb), middle (65 ppb), and lower (60 ppb) parts of the range.

A level of 70 ppb is below the O₃ exposure concentration that has been reported to elicit a broad range of respiratory effects that includes airway hyperresponsiveness and decreased lung host defense, in addition to lung function decrements, airway inflammation, and respiratory symptoms (i.e., 80 ppb). A level of 70 ppb is also just below the lowest exposure concentration at which the combined occurrence of respiratory symptoms and lung function decrements have been reported (i.e., 72 ppb), a combination judged adverse by the ATS (section 3.1.3). A level of 70 ppb is above the lowest exposure concentration demonstrated to result in lung function decrements and pulmonary inflammation (i.e., 60 ppb). Compared to the current standard, a revised O₃ standard with a level of 70 ppb would be expected to (1) reduce the occurrence of exposures of concern to O₃ concentrations that result in respiratory effects in healthy adults (at or above 60 and 70 ppb) by about 45 to 95%, almost eliminating the occurrence of multiple exposures at or above 70 ppb; (2) reduce the occurrence of moderate-to-large O₃-induced lung function decrements (FEV₁ decrements \geq 10, 15, 20%) by about 15 to 35%, most effectively limiting the occurrence of multiple decrements and decrements \geq 15, 20%; (3) more effectively maintain short- and long-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard;³ and (4) reduce the risk of O₃-associated mortality and morbidity, particularly the risk associated with the upper portions of the distributions of ambient O₃ concentrations.

A level of 65 ppb is well below the O₃ exposure concentration that has been reported to elicit the wide range of potentially adverse respiratory effects noted above, and is below the lowest exposure concentration at which the combined occurrence of respiratory

³ Though epidemiologic studies also provide evidence for O₃ health effect associations in locations likely to have met a standard with a level of 70 ppb, as discussed below for lower standard levels.

symptoms and lung function decrements has been reported. As noted above for 70 ppb, a level of 65 ppb is above the lowest exposure concentration demonstrated to result in lung function decrements and pulmonary inflammation. Compared to a standard with a level of 70 ppb, a revised standard with a level of 65 ppb would be expected to (1) further reduce the occurrence of exposures of concern (by about 80 to 100% compared to the current standard), decreasing exposures at or above 60 ppb and almost eliminating exposures at or above 70 and 80 ppb; (2) further reduce the occurrence of FEV₁ decrements \geq 10, 15, and 20% (by about 30 to 65%, compared to the current standard); (3) more effectively maintain short- and long-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard;⁴ and (4) further reduce the risk of O₃-associated mortality and morbidity, particularly the risk associated with the upper portion of the distribution of ambient O₃ concentrations.

A level of 60 ppb is well below the O₃ exposure concentration shown to result in the combined occurrence of respiratory symptoms and lung function decrements, and corresponds to the lowest exposure concentration demonstrated to result in lung function decrements and pulmonary inflammation. Compared to a standard with a level of 70 or 65 ppb, a revised standard with a level of 60 ppb would be expected to (1) further reduce the occurrence of exposures of concern (by about 95 to 100% compared to the current standard), almost eliminating exposures at or above 60 ppb; (2) further reduce the occurrence of FEV₁ decrements \geq 10, 15, and 20%, (by about 45 to 85% compared to the current standard); (3) more effectively maintain short- and long-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard;⁵ and (4) further reduce the risk of O₃-associated mortality and morbidity, particularly the risk associated with the upper portion of the distribution of ambient O₃ concentrations.

Welfare Effects and Review of the Secondary Standard

The longstanding and comprehensive evidence base, stronger than in the last review, documents the vegetation and ecosystem-related effects of O₃ in ambient air. In particular, recent controlled studies at the molecular, biochemical and cellular scales have increased the

⁴ Though epidemiologic studies also provide evidence for O₃ health effect associations in locations likely to have met a standard with a level of 65 ppb.

⁵ Epidemiologic studies have not evaluated O₃ health effect associations based primarily on air quality in locations likely to have met a standard with a level of 60 ppb.

mechanistic understanding of the basic biology of how plants are affected by oxidative stress. These studies have focused on a variety of plant responses to O₃ including: 1) reduced carbon dioxide uptake due to stomatal closure; 2) the upregulation of genes associated with plant defense, signaling, hormone synthesis and secondary metabolism; 3) the down regulation of genes related to photosynthesis and general metabolism; 4) the loss of carbon assimilation capacity due to declines in the quantity and activity of key proteins and enzymes; and 5) the negative impacts on the efficiency of the photosynthetic light reactions. In addition, these effects at the plant scale can be linked to an array of effects at larger scales, as shown in recent field studies, together with previously available evidence. Specifically, plant-scale effects, such as altered rates of leaf gas exchange, growth, and reproduction at the individual plant level, can result in larger scale effects in ecosystems, such as alterations in productivity, carbon storage, water cycling, nutrient cycling, and community composition. The available information also demonstrates a relationship between changes in tropospheric O₃ concentrations and radiative forcing, and between changes in tropospheric O₃ concentrations and effects on climate.

The long-standing body of available evidence also provides a wealth of information on aspects of O₃ exposure that are important in influencing plant response. These include support for the conclusions that: O₃ effects in plants are cumulative; higher O₃ concentrations appear to be more important than lower concentrations in eliciting a response; plant sensitivity to O₃ varies with time of day and plant development stage; and quantifying exposure with indices that cumulate hourly O₃ concentrations and preferentially weight the higher concentrations improves the explanatory power of exposure/response models for growth and yield, over using indices based on mean and peak exposure values.

As an initial matter in this PA, staff concludes that reducing ambient O₃ concentrations to meet the current standard of 75 ppb will provide important improvements in public welfare protection. This initial conclusion is based on (1) the strong body of scientific evidence indicating a wide range of effects to sensitive vegetation, including tree biomass loss, crop yield loss, and visible foliar injury, and associated ecosystems and services attributable to cumulative exposures to O₃ concentrations found in the ambient air and (2) estimates indicating decreased cumulative O₃ exposures and welfare risks upon meeting the current standard, compared to recent air quality. Strong support for this conclusion is provided by the available welfare evidence; by WREA estimates of cumulative exposures to O₃ concentrations shown to result in decreased biomass loss, crop yield loss, and visible foliar injury incidence under just meeting the current secondary standard; and by WREA estimates of improvements in carbon storage and air pollution removal in urban and commercial forests. Support for this conclusion is also provided by WREA estimates of increased protection for Class I areas from O₃-associated visible foliar injury and tree biomass loss.

Staff further concludes that the O₃-attributable welfare effects estimated to be allowed by air quality that meets the current secondary standard call into question the adequacy of the public welfare protection provided by the current standard. In addition, staff also concludes that the public welfare protection is most appropriately judged through the use of a more biologically relevant form, such as the cumulative, seasonal W126-metric. These conclusions are based on consideration of: (1) the scientific evidence, including controlled exposure studies reporting effects on plant growth, productivity and carbon storage, crop yield loss, and visible foliar injury following exposures to O₃ concentrations below the level of the current standard and field based studies that support these conclusions for air quality that would likely meet the current standard; (2) the longstanding and extensive evidence demonstrating that the risk to vegetation comes from cumulative seasonal exposures; (3) evidence suggesting that in Class I areas meeting the current standard, cumulative seasonal O₃ exposures occur that are associated with estimates of tree growth impacts of a magnitude that are reasonably considered important to public welfare; (4) WREA estimates of reductions in biomass loss, crop yield loss, and visible foliar injury incidence, and improvements in carbon storage and air pollution removal in urban and commercial forests when meeting alternative W126 levels; (5) advice received from CASAC based on their review of draft versions of the ISA, WREA, and PA, and advice received in previous reviews; and (6) public comments. Staff reaches the overall conclusion that the available vegetation and ecosystem effects evidence and exposure/risk information, including for associated ecosystem services important from a public welfare perspective, call into question the adequacy of the public welfare protection provided by the current standard. Based on the evaluation presented in this PA, staff concludes that consideration should be given to revising the standard to provide increased public welfare protection. CASAC agreed with this conclusion.

Given this conclusion regarding the adequacy of the current standard, staff also reaches conclusions for the Administrator's consideration regarding the elements of potential alternative secondary O₃ standards that could be supported by the available evidence and exposure/risk information. Any such potential alternative standards should protect public welfare against known or anticipated adverse environmental effects associated with exposures to O₃, alone or in combination with related photochemical oxidants, taking into account the available scientific evidence and exposure/risk information. In reaching conclusions about the range of potential alternative standards appropriate for consideration, staff is mindful that the Act requires secondary standards that are at "a level of air quality the attainment and maintenance of which" in the "judgment of the Administrator", are "requisite to protect public welfare from any known or anticipated adverse effects". In setting a secondary standard that is "requisite" to protect public welfare, the EPA's task is to establish standards that are neither more nor less stringent

than necessary. Thus, the Act does not require that NAAQS be set at zero-risk levels, but rather at levels that reduce risk sufficiently to protect public welfare from adverse effects.

The degree of public welfare protection provided by any NAAQS results from the collective impact of the elements of the standard, including the indicator, averaging time, form, and level. Staff's conclusions on each of these elements are summarized below.

- (1) **Indicator:** Staff concludes that it is appropriate to continue to use O₃ as the indicator for a standard that is intended to address welfare effects associated with exposure to O₃, alone or in combination with related photochemical oxidants. Based on the available information, staff concludes that there is no basis for considering an alternative indicator at this time. CASAC concurred with these conclusions.

- (2) **Averaging time and form:** Staff concludes that it is appropriate to consider a revised secondary standard in terms of the cumulative, seasonal, concentration-weighted form, called the W126 index. This is supported by strong scientific evidence that cumulative O₃ exposures drive plant response and can cause reduced tree growth, productivity, and carbon storage; crop yield loss; visible foliar injury; and other welfare effects. With regard to the appropriate form and averaging times, staff reaches the following additional conclusions:
 - (a) It is appropriate to consider the consecutive 3-month period within the O₃ season with the maximum index value as the seasonal period over which to cumulate hourly O₃ exposures. Staff notes that the maximum 3-month period generally coincides with maximum biological activity for most vegetation, making the 3-month duration a suitable surrogate for longer growing seasons.

 - (b) It is appropriate to cumulate daily exposures for the 12-hour period from 8:00 am to 8:00 pm, generally representing the daylight period during the 3-month period identified above.

To the extent the Administrator finds it useful to consider the extent of public welfare protection that might be afforded by a revised primary standard, staff concludes that public welfare protection is appropriately judged through the use of the cumulative, seasonal W126 index form, as described above. CASAC agreed that it was

appropriate to establish a revised form of the secondary standard and that the W126 index was a more biologically relevant form than the current form of the standard.

With regard to the number of years over which it is appropriate to average, staff notes that there is limited information to discern between the level of protection provided by an annual form or a 3-year average form of a W126 standard for crop yield loss or foliar injury, and that a multiple year form could be considered to provide a more consistent target level of protection for this endpoint. Such a form might also be appropriate for a standard intended to achieve the desired level of protection from longer-term effects, including those associated with potential compounding of biomass loss over multiple years. Further, such a form might be concluded to contribute to greater stability in air quality management programs, and thus, greater effectiveness in achieving the desired level of public welfare protection, than that that might result from a single year form. Therefore, to the extent that the greater emphasis is placed on protecting against effects associated with multi-year exposures and maintaining more year-to-year stability of public welfare protection, staff concludes that it is appropriate to consider a secondary standard form that averages the seasonal W126 index values across three consecutive years. CASAC recommended that if a 3-year averaging period is selected, the level should be set lower than if a 1-year averaging period is selected in order to provide greater protection for annual crops and against cumulative effects on perennial species.

- (3) **Level:** With regard to level for a revised secondary standard, staff concludes that it is appropriate to give consideration to a range of levels from 17 to 7 ppm-hrs, expressed in terms of the W126 index. In so doing, we primarily consider the evidence- and exposure/risk-based information for cumulative seasonal O₃ exposures represented by W126 index values (including those represented by the WREA average W126 scenarios) associated with biomass loss in studied tree species, both in and outside areas that have been afforded special protections. We note CASAC's advice that a 6% median RBL is unacceptably high, and that the 2% median RBL is an important benchmark to consider. We further note that for the lower level of 7 ppm-hrs the median tree species biomass loss is at or below 2% and that for the upper level of 17 ppm-hrs the median tree biomass loss is below 6%.⁶ We also note that a level of 17 ppm-hrs reduces the percent of total area having weighted RBL greater than 2% to

⁶ We note that a W126 index value of 19 ppm-hrs is estimated to result in a median RBL value of 6%.

0.2%, and reduces the number of Class I areas with weighted RBL greater than 2% to 2 of the 145 assessed nationally protected Class I areas.

We also note that tree biomass loss can be an indicator of more significant ecosystem-wide effects which might reasonably be concluded to be significant to public welfare. For example, when it occurs over multiple years at a sufficient magnitude, biomass loss is linked to an array of effects on other ecosystem-level processes such as nutrient and water cycles, changes in above and below ground communities, and carbon storage and air pollution removal. These effects have the potential to be adverse to the public welfare.

In addition, a range of levels from 17 to 7 ppm-hrs would protect at least half of the crop species from a yield loss of greater than 5%. A W126 level of 10 ppm-hrs or less would also reduce prevalence of visible foliar injury and promote appreciable gains in carbon sequestration and pollutant removal.

CASAC recommended a range of W126 values of 15 ppm-hrs to 7 ppm-hrs and did not recommend levels above 15 ppm-hrs. CASAC noted that a level of 15 ppm-hrs is requisite to protect median crop yield loss to no more than 5% and that a level below 10 ppm-hrs is required to reduce foliar injury prevalence. CASAC also noted that a W126 level of 7 ppm-hrs limits median relative biomass loss for trees to no greater than 2% and offers additional protection against crop yield loss and foliar injury.

The Administrator's consideration of a particular level within the range of 17 to 7 ppm-hrs would reflect judgments as to the appropriate weight to be given to various aspects of the scientific evidence and exposure/risk information, with appropriate weight given to important uncertainties and with particular consideration of the support provided by this evidence and information regarding the protection of public welfare. To the extent the Administrator finds it useful to consider the extent of public welfare protection that might be afforded by a revised primary standard, staff concludes that public welfare protection is appropriately judged through the use of the cumulative seasonal W126-based metric.

1 INTRODUCTION

1.1 PURPOSE

The U.S. Environmental Protection Agency (EPA) is presently conducting a review of the primary (health-based) and secondary (welfare-based) national ambient air quality standards (NAAQS) for ozone (O₃). The overall plan for this review was presented in the *Integrated Review Plan for the O₃ National Ambient Air Quality Standards* (IRP, U.S. EPA, 2011a). The IRP also identified key policy-relevant issues to be addressed in this review and discussed the key documents that generally inform NAAQS reviews, including an Integrated Science Assessment (ISA), Risk and Exposure Assessments (REAs), and a Policy Assessment (PA). The PA is prepared by the staff in EPA's Office of Air Quality Planning and Standards (OAQPS). It presents a staff evaluation of the policy implications of the key scientific and technical information in the ISA and REAs for EPA's consideration.¹ The PA provides a transparent evaluation, and staff conclusions, regarding policy considerations related to reaching judgments about the adequacy of the current standards, and if revision is considered, what revisions may be appropriate to consider.

The PA is intended to help “bridge the gap” between the Agency's scientific assessments presented in the ISA and REAs, and the judgments required of the EPA Administrator in determining whether it is appropriate to retain or revise the NAAQS.² In evaluating the adequacy of the current standard and whether it is appropriate to consider potential alternative standards, the PA focuses on information that is most pertinent to evaluating the basic elements of the NAAQS: indicator,³ averaging time, form,⁴ and level. These elements, which together serve to define each standard, must be considered collectively in evaluating the health and welfare protection afforded by the O₃ standards. The PA integrates and interprets the information from the ISA and REAs to frame policy options for consideration by the Administrator. In so doing, the PA recognizes that the selection of a specific approach to reaching final decisions on the primary and secondary O₃ standards will reflect the judgments of the Administrator.

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

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

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

⁴The “form” of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. For example, the form of the current 8-hour O₃ NAAQS is the 3-year average of the annual fourth-highest daily maximum 8-hour average.

The development of the PA is also intended to facilitate advice to the Agency and recommendations to the Administrator from an independent scientific review committee, the Clean Air Scientific Advisory Committee (CASAC), as provided for in the Clean Air Act. As discussed below in section 1.2.1, the CASAC is to advise not only on the Agency's assessment of the relevant scientific information, but also on the adequacy of the existing standards, and to make recommendations as to any revisions of the standards that may be appropriate. The EPA facilitates CASAC advice and recommendations, as well as public input and comment, by requesting CASAC review and public comment on one or more drafts of the PA.

In this PA for the review of the O₃ NAAQS, we⁵ consider the scientific and technical information available in this review as assessed in the *Integrated Science Assessment for O₃ and Related Photochemical Oxidants* (ISA, U.S. EPA, 2013), prepared by EPA's National Center for Environmental Assessment (NCEA), and the quantitative human exposure and health risk assessment and welfare risk assessment documents (HREA, U.S. EPA, 2014a; WREA, U.S. EPA, 2014b). The evaluation and staff conclusions presented in this PA have been informed by comments and advice received from CASAC in their reviews of draft versions of the PA, and in their reviews of the other draft Agency documents prepared for this NAAQS review.

Beyond informing the EPA Administrator and facilitating the advice and recommendations of CASAC and the public, the PA is also intended to be a useful reference to all parties interested in the NAAQS review. In these roles, it is intended to serve as a single source of the most policy-relevant information that informs the Agency's review of the NAAQS, and it is written to be understandable to a broad audience.

The remainder of chapter 1 summarizes information on the NAAQS legislative requirements and on the history of the O₃ NAAQS (section 1.2), and summarizes our general approaches to reviewing the current O₃ NAAQS (section 1.3). Chapter 2 of this PA provides an overview of the O₃ ambient monitoring network and O₃ air quality, including estimates of O₃ concentrations attributable to background sources. The remaining chapters are organized into two main parts. Chapters 3 and 4 focus on the review of the primary O₃ NAAQS while chapters 5 and 6 focus on the review of the secondary O₃ NAAQS. Staff's considerations and conclusions related to the current primary and secondary standards are discussed in chapters 3 and 5, respectively. Staff's considerations and conclusions related to potential alternative primary and secondary standards are discussed in chapters 4 and 6, respectively. Key uncertainties in the review and areas for future research and data collection are additionally identified in chapters 4 and 6 for the two types of standards.

⁵As noted above, the term "we" through this document refer to personnel in the EPA's Office of Air Quality Planning and Standards (OAQPS).

1.2 BACKGROUND

1.2.1 Legislative Requirements

Two sections of the Clean Air Act (CAA) govern the establishment and revision of the NAAQS. Section 108 (42 U.S.C. section 7408) directs the Administrator to identify and list certain air pollutants and then to issue air quality criteria for those pollutants. The Administrator is to list those air pollutants that in her “judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare;” “the presence of which in the ambient air results from numerous or diverse mobile or stationary sources;” and “for which . . . [the Administrator] plans to issue air quality criteria. . . .” Air quality criteria are intended to “accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of [a] pollutant in the ambient air . . .” 42 U.S.C. § 7408(b). Section 109 (42 U.S.C. 7409) directs the Administrator to propose and promulgate “primary” and “secondary” NAAQS for pollutants for which air quality criteria are issued. Section 109(b)(1) defines a primary standard as one “the attainment and maintenance of which in the judgment of the Administrator, based on such criteria and allowing an adequate margin of safety, are requisite to protect the public health.”⁶ A secondary standard, as defined in section 109(b)(2), must “specify a level of air quality the attainment and maintenance of which, in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air.”⁷

The requirement that primary standards provide an adequate margin of safety was intended to address uncertainties associated with inconclusive scientific and technical information available at the time of standard setting. It was also intended to provide a reasonable degree of protection against hazards that research has not yet identified. See State of Mississippi v. EPA, 744 F. 3d 1334, 1353 (D.C. Cir. 2012) (“By requiring an ‘adequate margin of safety’, Congress was directing EPA to build a buffer to protect against uncertain and unknown dangers to human health”). See also Lead Industries Association v. EPA, 647 F.2d 1130, 1154 (D.C. Cir. 1980); American Petroleum Institute v. Costle, 665 F.2d 1176, 1186 (D.C. Cir. 1981); American Farm Bureau Federation v. EPA, 559 F. 3d 512, 533 (D.C. Cir. 2009); Association of Battery

⁶ The legislative history of section 109 indicates that a primary standard is to be set at “the maximum permissible ambient air level . . . which will protect the health of any [sensitive] group of the population,” and that for this purpose “reference should be made to a representative sample of persons comprising the sensitive group rather than to a single person in such a group” S. Rep. No. 91-1196, 91st Cong., 2d Sess. 10 (1970).

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

Recyclers v. EPA, 604 F. 3d 613, 617-18 (D.C. Cir. 2010). Both kinds of uncertainties are components of the risk associated with pollution at levels below those at which human health effects can be said to occur with reasonable scientific certainty. Thus, in selecting primary standards that provide an adequate margin of safety, the Administrator is seeking not only to prevent pollution levels that have been demonstrated to be harmful but also to prevent lower pollutant levels that may pose an unacceptable risk of harm, even if the risk is not precisely identified as to nature or degree. The CAA does not require the Administrator to establish a primary NAAQS at a zero-risk level or at background concentration levels, see Lead Industries v. EPA, 647 F.2d at 1156 n.51; State of Mississippi v. EPA, 744 F. 3d at 1343, 1351, but rather at a level that reduces risk sufficiently so as to protect public health with an adequate margin of safety.

In addressing the requirement for an adequate margin of safety, the EPA considers such factors as the nature and severity of the health effects, the size of sensitive population(s)⁸ at risk, and the kind and degree of the uncertainties that must be addressed. The selection of any particular approach for providing an adequate margin of safety is a policy choice left specifically to the Administrator's judgment. See Lead Industries Association v. EPA, 647 F.2d at 1161-62; State of Mississippi, 744 F. 3d at 1353.

In setting primary and secondary standards that are "requisite" to protect public health and welfare, respectively, as provided in section 109(b), EPA's task is to establish standards that are neither more nor less stringent than necessary for these purposes. In so doing, the EPA may not consider the costs of implementing the standards. See generally, Whitman v. American Trucking Associations, 531 U.S. 457, 465-472, 475-76 (2001). Likewise, "[a]ttainability and technological feasibility are not relevant considerations in the promulgation of national ambient air quality standards." American Petroleum Institute v. Costle, 665 F. 2d at 1185.

Section 109(d)(1) requires that "not later than December 31, 1980, and at 5-year intervals thereafter, the Administrator shall complete a thorough review of the criteria published under section 108 and the national ambient air quality standards . . . and shall make such revisions in such criteria and standards and promulgate such new standards as may be appropriate" Section 109(d)(2) requires that an independent scientific review committee "shall complete a review of the criteria . . . and the national primary and secondary ambient air quality standards . . . and shall recommend to the Administrator any new . . . standards and revisions of existing

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

criteria and standards as may be appropriate” Since the early 1980's, the Clean Air Scientific Advisory Committee (CASAC) has performed this independent review function.⁹

1.2.2 History of O₃ NAAQS Reviews

Table 1-1 summarizes the O₃ NAAQS that the EPA has promulgated to date. In each review, the EPA set the secondary standard at a level identical to the primary standard. These reviews are briefly described below.

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

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

The EPA first established primary and secondary NAAQS for photochemical oxidants in 1971 (36 FR 8186, April 30, 1971). The EPA set both primary and secondary standards at a level of 0.08 parts per million (ppm), 1-hr average, total photochemical oxidants, not to be exceeded more than one hour per year. The EPA based the standards on scientific information contained in the 1970 *Air Quality Criteria for Photochemical Oxidants* (U.S. DHEW, 1970). We initiated the first periodic review of the NAAQS for photochemical oxidants in 1977. Based on the 1978 *Air*

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

Quality Criteria for Ozone and Other Photochemical Oxidants (U.S. EPA, 1978), the EPA published proposed revisions to the original NAAQS in 1978 (43 FR 16962) and final revisions in 1979 (44 FR 8202). At that time, the EPA revised the level of the primary and secondary standards from 0.08 to 0.12 ppm and changed the indicator from photochemical oxidants to O₃, and the form of the standards from a deterministic to a statistical form. This statistical form defined attainment of the standards as occurring when the expected number of days per calendar year with maximum hourly average concentration greater than 0.12 ppm equaled one or less.

Following the final decision in the 1979 review, the City of Houston challenged the Administrator's decision arguing that the standard was arbitrary and capricious because natural O₃ concentrations and other physical phenomena in the Houston area made the standard unattainable in that area. The U.S. Court of Appeals for the District of Columbia Circuit (D.C. Circuit) rejected this argument, holding (as noted above) that attainability and technological feasibility are not relevant considerations in the promulgation of the NAAQS. The court also noted that the EPA need not tailor the NAAQS to fit each region or locale, pointing out that Congress was aware of the difficulty in meeting standards in some locations and had addressed this difficulty through various compliance related provisions in the Act. See *API v. Costle*, 665 F.2d 1176, 1184-6 (D.C. Cir. 1981).

In 1982, we announced plans to revise the 1978 Air Quality Criteria document (47 FR 11561), and in 1983, we initiated the second periodic review of the O₃ NAAQS (48 FR 38009). We subsequently published the 1986 *Air Quality Criteria for Ozone and Other Photochemical Oxidants* (U.S. EPA, 1986) and the 1989 Staff Paper (U.S. EPA, 1989). Following publication of the 1986 Air Quality Criteria Document (AQCD), a number of scientific abstracts and articles were published that appeared to be of sufficient importance concerning potential health and welfare effects of O₃ to warrant preparation of a Supplement. On August 10, 1992, under the terms of a court order, the EPA published a proposed decision to retain the existing primary and secondary standards (57 FR 35542). The notice explained that the proposed decision would complete EPA's review of information on health and welfare effects of O₃ assembled over a 7-year period and contained in the 1986 AQCD and its 1992 Supplement. The proposal also announced EPA's intention to proceed as rapidly as possible with the next review of the air quality criteria and standards for O₃ in light of emerging evidence of health effects related to 6- to 8-hour O₃ exposures. On March 9, 1993, the EPA concluded the review by affirming its proposed decision to retain the existing primary and secondary standards. (58 FR 13008).

In August 1992, we announced plans to initiate the third periodic review of the air quality criteria and O₃ NAAQS (57 FR 35542). In December 1996, the EPA proposed to replace the then existing 1-hour primary and secondary standards with 8-hour average O₃ standards set at a level of 0.08 ppm (equivalent to 0.084 ppm using standard rounding conventions) (61 FR 65716). The

EPA also proposed to establish a new distinct secondary standard using a biologically-based cumulative, seasonal form. The EPA completed this review on July 18, 1997 (62 FR 38856) by setting the primary standard at a level of 0.08 ppm, based on the annual fourth-highest daily maximum 8-hr average concentration, averaged over three years, and setting the secondary standard identical to the revised primary standard. In reaching this decision, the EPA identified several reasons supporting its decision to reject a potential alternate standard set at 0.07 ppm. Most importantly, the EPA pointed out the scientific uncertainty at lower concentrations and placed significant weight on the fact that no CASAC panel member supported a standard level set lower than 0.08 ppm (62 FR 38868). In addition to noting the uncertainties in the health evidence for exposure concentrations below 0.08 ppm and the advice of CASAC, the EPA noted that a standard set at a level of 0.07 ppm would be closer to peak background concentrations that infrequently occur in some areas due to nonanthropogenic sources of O₃ precursors (62 FR 38856, 38868; July 18, 1997).

On May 14, 1999, in response to challenges by industry and others to EPA's 1997 decision, the U.S. Court of Appeals for the District of Columbia Circuit remanded the O₃ NAAQS to the EPA, finding that section 109 of the Act, as interpreted by the EPA, effected an unconstitutional delegation of legislative authority. American Trucking Assoc. vs. EPA, 175 F.3d 1027, 1034-1040(D.C. Cir. 1999) (“ATA I”). In addition, the court directed that, in responding to the remand, the EPA should consider the potential beneficial health effects of O₃ pollution in shielding the public from the effects of solar ultraviolet (UV) radiation, as well as adverse health effects. Id. at 1051-53. In 1999, the EPA petitioned for rehearing *en banc* on several issues related to that decision. The court granted the request for rehearing in part and denied it in part, but declined to review its ruling with regard to the potential beneficial effects of O₃ pollution. 195 F3d 4, 10 (D.C Cir., 1999) (“ATA II”). On January 27, 2000, the EPA petitioned the U.S. Supreme Court for certiorari on the constitutional issue (and two other issues), but did not request review of the ruling regarding the potential beneficial health effects of O₃. On February 27, 2001, the U.S. Supreme Court unanimously reversed the judgment of the D.C. Circuit on the constitutional issue. Whitman v. American Trucking Assoc., 531 U. S. 457, 472-74 (2001) (holding that section 109 of the CAA does not delegate legislative power to the EPA in contravention of the Constitution). The Court remanded the case to the D.C. Circuit to consider challenges to the O₃ NAAQS that had not been addressed by that court's earlier decisions. On March 26, 2002, the D.C. Circuit issued its final decision on remand, finding the 1997 O₃ NAAQS to be “neither arbitrary nor capricious,” and so denying the remaining petitions for review. American Trucking Associations, Inc. v EPA, 283 F.3d 355, 379 (D.C Cir., 2002) (“ATA III”).

Specifically, in ATA III, the D.C. Circuit upheld EPA's decision on the 1997 O₃ standard as the product of reasoned decision-making. The Court made clear that the most important support for EPA's decision was the health evidence and the concerns it raised about setting a standard level below 0.08 ppm. ("the record is replete with references to studies demonstrating the inadequacies of the old one-hour standard", as well as extensive information supporting the change to an 8-hour averaging time). 283 F 3d at 378. The Court also pointed to the significant weight that the EPA properly placed on the advice it received from CASAC. Id. at 379. The court further noted that "although relative proximity to peak background ozone concentrations did not, in itself, necessitate a level of 0.08, EPA could consider that factor when choosing among the three alternative levels." Id.

Independently of the litigation, the EPA also responded to the Court's remand to consider the potential beneficial health effects of O₃ pollution in shielding the public from effects of solar (ultraviolet or UV-B) radiation. The EPA provisionally determined that the information linking changes in patterns of ground-level O₃ concentrations to changes in relevant patterns of exposures to ultraviolet (UV-B) radiation of concern to public health was too uncertain, at that time, to warrant any relaxation in 1997 O₃ NAAQS. The EPA also expressed the view that any plausible changes in UV-B radiation exposures from changes in patterns of ground-level O₃ concentrations would likely be very small from a public health perspective. In view of these findings, the EPA proposed to leave the 1997 8-hour NAAQS unchanged (66 FR 57268, Nov. 14, 2001). After considering public comment on the proposed decision, the EPA published its final response to this remand on January 6, 2003, re-affirming the 8-hour O₃ NAAQS set in 1997 (68 FR 614).

The EPA initiated the fourth periodic review of the air quality criteria and O₃ standards in September 2000 with a call for information (65 FR 57810). The schedule for completion of that review was ultimately governed by a consent decree resolving a lawsuit filed in March 2003 by plaintiffs representing national environmental and public health organizations, who maintained that EPA was in breach of a mandatory legal duty to complete review of the O₃ NAAQS within a statutorily-mandated deadline. On July 11, 2007, the EPA proposed to revise the level of the primary standard within a range of 0.075 to 0.070 ppm. (72 FR 37818). Documents supporting this proposed decision included the *Air Quality Criteria for Ozone and Other Photochemical Oxidants* (U.S. EPA, 2006) and the Staff Paper (U.S. EPA, 2007) and related technical support documents. The EPA also proposed two options for revising the secondary standard: (1) replace the current standard with a cumulative, seasonal standard, expressed as an index of the annual sum of weighted hourly concentrations cumulated over 12 daylight hours during the consecutive 3-month period within the O₃ season with the maximum index value, set at a level within the range of 7 to 21 ppm-hrs, and (2) set the secondary standard identical to the proposed primary

standard. The EPA completed the review with publication of a final decision on March 27, 2008 (73 FR 16436). In that final rule, the EPA revised the NAAQS by lowering the level of the 8-hour primary O₃ standard from 0.08 ppm to 0.075 ppm, not otherwise revising the primary standard, and adopting a secondary standard identical to the revised primary standard. In May 2008, state, public health, environmental, and industry petitioners filed suit challenging EPA's final decision on the 2008 O₃ standards. On September 16, 2009, the EPA announced its intention to reconsider the 2008 O₃ standards, and initiated a rulemaking to do so. At EPA's request, the Court held the consolidated cases in abeyance pending EPA's reconsideration of the 2008 decision.

On January 19, 2010 (75 FR 2938), the EPA issued a notice of proposed rulemaking to reconsider the 2008 final decision. In that notice, the EPA proposed that further revisions of the primary and secondary standards were necessary to provide a requisite level of protection to public health and welfare. The EPA proposed to decrease the level of the 2008 8-hour primary standard from 0.075 ppm to a level within the range of 0.060 to 0.070 ppm, and to change the secondary standard to a new cumulative, seasonal standard expressed as an annual index of the sum of weighted hourly concentrations, cumulated over 12 hours per day (8 am to 8 pm), during the consecutive 3-month period within the O₃ season, with a maximum index value set at a level within the range of 7 to 15 ppm-hours. The Agency also solicited CASAC review of the proposed rule on January 25, 2010 and solicited additional CASAC advice on January 26, 2011. After considering comments from CASAC and the public, the EPA prepared a draft final rule, which was submitted for interagency review pursuant to Executive Order 12866. On September 2, 2011, consistent with the direction of the President, the Administrator of the Office of Information and Regulatory Affairs ("OIRA"), Office of Management and Budget ("OMB"), returned the draft final rule to the EPA for further consideration. In view of this return and the timing of the Agency's ongoing periodic review of the O₃ NAAQS required under Clean Air Act section 109 (as announced on September 29, 2008), the EPA decided to coordinate further proceedings on its voluntary rulemaking on reconsideration with that ongoing periodic review, by deferring the completion of its voluntary rulemaking on reconsideration until it completes its statutorily-required periodic review.

In light of EPA's decision to consolidate the reconsideration with the current review, the Court proceeded with the litigation on the 2008 final decision. On July 23, 2013, the D.C. Circuit Court of Appeals upheld EPA's 2008 primary O₃ standard, but remanded the 2008 secondary standard to the EPA. State of Mississippi v. EPA, 744 F. 3d 1334. With respect to the primary standard, the court first held that the EPA reasonably determined that the existing standard was not requisite to protect public health with an adequate margin of safety, and consequently required revision. Specifically, the court noted that there were "numerous epidemiological

studies linking health effects to exposure to ozone levels below 0.08 ppm and clinical human exposure studies finding a causal relationship between health effects and exposure to ozone levels at and below 0.08 ppm”. 744 F. 3d at 1345. The court also specifically endorsed the weight of evidence approach utilized by EPA in its deliberations. *Id.* at 1344.

The court went on to reject arguments that EPA should have adopted a more stringent primary standard. Dismissing arguments that a clinical study (as properly interpreted by EPA) to show effects at 0.06 ppm necessitated a standard level lower than that selected, the court noted that this was a single, limited study. *Id.* at 1350. With respect to the epidemiologic evidence, the court accepted EPA’s argument that there could be legitimate uncertainty that a causal relationship between O₃ and 8-hour exposures less than 0.075 ppm exists, so that associations at lower levels reported in epidemiologic studies did not necessitate a more stringent standard. *Id.* at 1351-52.¹⁰

The court also rejected arguments that an 8-hour primary standard of 0.075 ppm failed to provide an adequate margin of safety, noting that margin of safety considerations involved policy judgments by the agency, and that by setting a standard “appreciably below” the level of the current standard (0.08 ppm), the agency had made a reasonable policy choice. *Id.* Finally, the court rejected arguments that EPA’s decision was inconsistent with CASAC’s scientific recommendations because CASAC had been insufficiently clear in its recommendations whether it was providing scientific or policy recommendations, and EPA had reasonably addressed CASAC’s policy recommendations. *Id.* at 1357-58.

With respect to the secondary standard, the court held that because EPA had failed to identify a level of air quality requisite to protect public welfare, EPA’s comparison between the primary and secondary standards for determining if requisite protection for public welfare was afforded by the primary standard was inherently arbitrary. The court thus rejected EPA’s determination that the revised 8-hour primary standard afforded sufficient protection of public welfare, and remanded the standard to EPA. *Id.* at 1360-62.

1.2.3 Current O₃ NAAQS Review

On September 29, 2008, the EPA announced the initiation of a new periodic review of the air quality criteria for O₃ and related photochemical oxidants and issued a call for information in the Federal Register (73 FR 56581, Sept. 29, 2008). A wide range of external

¹⁰ The court cautioned, however, that “perhaps more [clinical] studies like the Adams studies will yet reveal that the 0.060 ppm level produces significant adverse decrements that simply cannot be attributed to normal variation in lung function”, and further cautioned that “agencies may not merely recite the terms ‘substantial uncertainty’ as a justification for their actions”. *Id.* at 1350, 1357 (internal citations omitted).

experts, as well as EPA staff, representing a variety of areas of expertise (e.g., epidemiology, human and animal toxicology, statistics, risk/exposure analysis, atmospheric science, ecology, biology, plant science, ecosystem services) participated in a workshop. This workshop was held on October 28-29, 2008 in Research Triangle Park, NC. The workshop provided an opportunity for a public discussion of the key policy-relevant issues around which the EPA would structure this O₃ NAAQS review and the most meaningful new science that would be available to inform our understanding of these issues.

Based in part on the workshop discussions, the EPA developed a draft Integrated Review Plan outlining the schedule, process, and key policy-relevant questions that would guide the evaluation of the air quality criteria for O₃ and the review of the primary and secondary O₃ NAAQS. A draft of the IRP was released for public review and comment in September 2009. This IRP was the subject of a consultation with the CASAC on November 13, 2009 (74 FR 54562; October 22, 2009).¹¹ We considered comments received from that consultation and from the public in finalizing the plan and in beginning the review of the air quality criteria. The EPA's overall plan and schedule for this review is presented in the *Integrated Review Plan for the Ozone National Ambient Air Quality Standards*.¹²

As part of the process of preparing the O₃ ISA, NCEA hosted a peer review workshop in October 29-30, 2008 (73 FR 56581, September 29, 2008) on preliminary drafts of key ISA chapters. The CASAC and the public reviewed the first external review draft ISA (U.S. EPA, 2011b; 76 FR 10893, February 28, 2011) at a meeting held in May 19-20, 2011 (76 FR 23809; April 28, 2011). Based on CASAC and public comments, NCEA prepared a second draft ISA (U.S. EPA, 2011c; 76 FR 60820, September 30, 2011). CASAC and the public reviewed this draft at a January 9-10, 2012 (76 FR 236, December 8, 2011) meeting. Based on CASAC and public comments, NCEA prepared a third draft ISA (U.S. EPA 2012a; 77 FR 36534; June 19, 2012), which was reviewed at a CASAC meeting in September 2012. The EPA released the final ISA in February 2013.

The EPA presented its plans for conducting the Risk and Exposure Assessments (REAs) that build on the scientific evidence presented in the ISA, in two planning documents titled *Ozone National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment* and *Ozone National Ambient Air Quality Standards: Scope and Methods*

¹¹ See <http://yosemite.epa.gov/sab/sabproduct.nsf/WebProjectsbyTopicCASAC!OpenView> for more information on CASAC activities related to the current O₃ NAAQS review.

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

Plan for Welfare Risk and Exposure Assessment (henceforth, Scope and Methods Plans).¹³

These planning documents outlined the scope and approaches that staff planned to use in conducting quantitative assessments, as well as key issues that would be addressed as part of the assessments. We released these documents for public comment in April 2011, and consulted with CASAC on May 19-20, 2011 (76 FR 23809; April 28, 2011). In designing and conducting the initial health risk and welfare risk assessments, we considered CASAC comments (Samet 2011) on the Scope and Methods Plans and also considered public comments. In May 2012, we issued a memo titled *Updates to Information Presented in the Scope and Methods Plans for the Ozone NAAQS Health and Welfare Risk and Exposure Assessments* that described changes to elements of the scope and methods plans and provided a brief explanation of each change and the reason for it.

In July 2012, EPA made the first drafts of the Health and Welfare REAs available for CASAC review and public comment (77 FR 42495, July 19, 2012). The first draft PA was made available for CASAC review and public comment in August 2012. These documents were reviewed by the CASAC O₃ Panel at a public meeting in September 2012. The second draft REAs and PA were prepared by EPA in consideration of CASAC (Frey and Samet, 2012a, 2012b) and public comment and were reviewed by the CASAC O₃ Panel at a public meeting on March 25-27, 2014. This final PA reflects staff's consideration of the comments and recommendations made by CASAC, and comments made by members of the public, in their review of draft versions of the PA.

1.3 GENERAL APPROACH FOR REVIEW OF THE STANDARDS

As described in section 1.1 above, this PA presents a transparent evaluation and staff conclusions regarding policy considerations related to reaching judgments about the adequacy of the current standards and the revisions that are appropriate to consider. Staff considerations and conclusions in this document are based on the available body of scientific evidence assessed in the ISA (U.S. EPA, 2013), exposure and risk analyses presented in the REAs (U.S. EPA, 2014a, b), advice and recommendations from CASAC on the first and second draft REAs and PA and other draft and final EPA documents in this review, as well as on public comments. This evaluation and associated conclusions on the range of policy options that, in staff's view, are supported by the available scientific evidence and exposure/risk information will inform the Administrator's decisions as to whether the existing primary and/or secondary O₃ standards should be revised and, if so, what revised standard or standards is/are appropriate.

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

Staff's considerations and conclusions related to the current and alternative primary and secondary O₃ standards are framed by a series of key policy-relevant questions, expanding upon those presented in the IRP at the outset of this review (U.S. EPA, 2011a). Answers to these questions in this final PA will inform the Administrator's decisions as to whether, and if so how, to revise the current O₃ standards. The first overarching question is as follows.

- **Do the currently available scientific evidence and exposure/risk information, as reflected in the ISA and REAs, support or call into question the adequacy of the protection afforded by the current O₃ standards?**

If the answer to this question, which is informed by staff's consideration of more specific questions related to the primary and secondary standards, suggests that revision of the current standards may be appropriate, then staff further considers the currently available evidence and information with regard to the following question.

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

The general approaches for consideration of these overarching questions in review of the primary and secondary standards are described separately in sections 1.3.1 and 1.3.2 below.

1.3.1 Approach for the Primary Standard

Staff's approach in this review of the current primary O₃ standard takes into consideration the approaches used in previous O₃ NAAQS reviews. The past and current approaches described below are both based, fundamentally, on using EPA's assessment of the current scientific evidence and associated quantitative analyses to inform the Administrator's judgment regarding a primary standard for O₃ that is "requisite" (i.e., neither more nor less stringent than necessary) to protect public health with an adequate margin of safety.

In reaching conclusions on options for the Administrator's consideration, we note that the final decision to retain or revise the current primary O₃ standard is a public health policy judgment to be made by the Administrator. This final decision by the Administrator will draw upon the available scientific evidence for O₃-attributable health effects, and on analyses of population exposures and health risks, including judgments about the appropriate weight to assign the range of uncertainties inherent in the evidence and analyses. Our general approach to informing these judgments, discussed more fully below, recognizes that the available health effects evidence reflects a continuum from relatively higher O₃ concentrations, at which scientists generally agree that health effects are likely to occur, through lower concentrations, at which the likelihood and magnitude of a response become increasingly uncertain. Therefore, in developing conclusions in this PA, we are mindful that the Administrator's ultimate judgments

on the primary standard will most appropriately reflect an interpretation of the available scientific evidence and exposure/risk information that neither overstates nor understates the strengths and limitations of that evidence and information. This approach is consistent with the requirements of sections 108 and 109 of the Act, as well as with how the EPA and the courts have historically interpreted the Act.

Section 1.3.1.1 below provides an overview of the general approach taken in the last review of the primary O₃ NAAQS (i.e., the 2008 review), and a summary of the rationale for the decision on the level of the standard in that review (73 FR 16436). Section 1.3.1.2 presents our approach in the current review, including our approach to considering the health evidence and exposure/risk information, and considerations regarding ambient O₃ concentrations attributable to background sources.

1.3.1.1 Approach Used in the Last Review

In the 2008 review of the O₃ NAAQS, the Administrator considered the available scientific evidence and exposure/risk information, the advice and recommendations of CASAC, and comments from the public. Based on this, he revised the level of the 8-hour primary O₃ standard from 0.08 ppm¹⁴ to 0.075 ppm (75 ppb¹⁵). In reaching a decision to revise the 1997 8-hour primary O₃ standard, the Administrator noted that much new evidence had become available since the 1997 review. He noted that this body of scientific evidence was very robust and provided consistent and coherent evidence of an array of O₃-related respiratory morbidity effects, and possibly cardiovascular-related morbidity, as well as total nonaccidental and cardiorespiratory mortality. The Administrator specifically observed that (1) the evidence of a range of respiratory-related morbidity effects had been considerably strengthened; (2) newly available evidence from controlled human exposure and epidemiologic studies identified people with asthma as an important susceptible population for which estimates of respiratory effects in the general population likely underestimate the magnitude or importance of these effects; (3) newly available evidence about mechanisms of toxicity more completely explained the biological plausibility of O₃-induced respiratory effects and was beginning to suggest mechanisms that may link O₃ exposure to cardiovascular effects; and (4) there was relatively strong evidence for associations between short-term O₃ concentrations and total nonaccidental and cardiopulmonary mortality. In the opinion of the Administrator, this very robust body of evidence enhanced our understanding of O₃- related effects and provided increased confidence

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

¹⁵ The level of the O₃ standard is specified as 0.075 ppm rather than 75 ppb. However, in this PA we refer to ppb, which is most often used in the scientific literature and in the ISA, in order to avoid the confusion that could result from switching units when discussing the evidence in relation to the standard level.

that various respiratory morbidity effects and other effects marked by indicators of respiratory morbidity are causally related to O₃ exposures, and the evidence was highly suggestive that O₃ exposures during the warm O₃ season contribute to premature mortality.¹⁶

The Administrator also noted important new health evidence reporting a broad array of adverse effects following short-term exposures to O₃ concentrations below the level of the 1997 standard, and concerns for such or related effects in at-risk populations,¹⁷ including people with asthma or other lung diseases, older adults with increased susceptibility, and those who are likely to be vulnerable as a result of spending a lot of time outdoors engaged in physical activity (e.g., especially active children and outdoor workers).

He specifically noted new scientific evidence, which built upon existing evidence, demonstrating O₃-induced lung function effects and respiratory symptoms in some healthy individuals following exposures down to 80 ppb. He also noted very limited new evidence demonstrating such effects at exposure concentrations well below 80 ppb. In addition, the Administrator noted (1) epidemiologic evidence of statistically significant associations with O₃-related health effects in areas that likely would have met the then-current standard; (2) epidemiologic studies conducted in areas that likely would have violated the existing standard but which nonetheless reported statistically significant associations that generally extended down to ambient O₃ concentrations below the level of that standard; (3) the few studies that had reported statistically significant associations with respiratory morbidity outcomes and mortality in subsets of data that included only days with ambient O₃ concentrations below the level of the existing standard; and (4) controlled human exposure studies, together with animal toxicological studies, that provided considerable support for the biological plausibility of the respiratory morbidity associations observed in the epidemiologic studies. Based on the available evidence, the Administrator agreed with the CASAC and the majority of public commenters that the existing standard was not requisite to protect public health with an adequate margin of safety (73 FR 16471).

¹⁶ 73 FR 16470-16471 (March 27, 2008)

¹⁷ Here, as in the ISA, the term “at-risk population” is used to encompass populations or lifestages that have a greater likelihood of experiencing health effects related to exposure to an air pollutant due to a variety of factors; other terms used in the literature include susceptible, vulnerable, and sensitive. These factors may be intrinsic, such as genetic factors, lifestage, or the presence of preexisting diseases, or they may be extrinsic, such as socioeconomic status (SES), activity pattern and exercise level, or increased pollutant exposures (U.S. EPA 2013, p. lxx, 8-1, 8-2). The courts and the Act’s legislative history refer to these at-risk subpopulations as “susceptible” or “sensitive” populations. See, e.g., *American Lung Ass’n v. EPA*, 134 F. 3d 388, 389 (D.C. Cir. 1998) (“NAAQS must protect not only average health individuals, but also ‘sensitive citizens’ – children, for example, or people with asthma, emphysema, or other conditions rendering them particularly vulnerable to air pollution” (quoting S. Rep. No. 91-1196 at 10)).

Beyond this focus on the available health evidence, the Administrator also considered estimates of O₃ exposures and health risks based on analyses where air quality was adjusted to simulate just meeting the existing and potential alternative standards. For the various air quality simulations, he specifically considered the pattern of estimated reductions in O₃ exposures across health benchmark concentrations of 80, 70, and 60 ppb. The 80 ppb benchmark reflected an exposure concentration for which there was strong evidence for respiratory effects in healthy people, including airway inflammation, respiratory symptoms, airway hyperresponsiveness, and impaired lung host defense (U.S. EPA, 2007, section 4.7). The 60 ppb benchmark reflected an exposure concentration for which the Administrator judged the evidence of such effects to be very limited (73 FR 16471).

The Administrator took note of the magnitudes of estimated health risks for a range of health effects, including moderate and large lung function decrements, respiratory symptoms, respiratory-related hospital admissions, and nonaccidental and cardiorespiratory mortality. He recognized that these quantitative risk estimates for a limited number of specific health effects were indicative of a much broader array of O₃-related effects, including various indicators of morbidity in at-risk populations that we could not analyze in the risk assessment (e.g., school absences, increased medication use, emergency department visits). The Administrator concluded that quantitative exposure and risk estimates, as well as the broader array of O₃-related health endpoints that could not be quantified, provided additional support for the evidence-based conclusion that the existing standard needed to be revised (73 FR 16472).

Based on the above considerations, and consistent with CASAC's unanimous conclusion that there was no scientific justification for retaining the existing standard, the Administrator concluded that the primary O₃ standard set in 1997 was not sufficient and thus not requisite to protect public health with an adequate margin of safety. He further concluded that revision of this standard was needed to provide increased public health protection (73 FR 16472).

Throughout the 2008 review, CASAC supported a standard level in the range of 60 to 70 ppb (without change to the form, indicator, or averaging time). In a letter to the Administrator on the second draft Staff Paper, CASAC unanimously recommended "that the current primary ozone standard be revised and that the level that should be considered for the revised standard be from 0.060 to 0.070 ppm" (60 to 70 ppb) (Henderson, 2006, p. 5). This recommendation, based in part on the placement of more weight on the evidence for effects following exposures to 60 ppb O₃, followed from the CASAC's more general recommendation that the 1997 standard needed to be made substantially more protective of human health, particularly for at-risk populations. In a subsequent letter sent specifically to offer advice to aid the Administrator and Agency staff in developing the 2007 O₃ proposal, CASAC reiterated that Panel members "were

unanimous in recommending that the level of the current primary ozone standard should be lowered from 0.08 ppm to no greater than 0.070 ppm” (Henderson, 2007, p. 2).¹⁸

After considering CASACs comments, the Administrator judged that the appropriate balance to draw, based on the entire body of evidence and information available in the 2008 review, was a standard set at a level of 75 ppb (and leaving all other elements of the NAAQS unchanged). In making this decision, the Administrator placed primary emphasis on the body of available scientific evidence, while viewing the results of exposure and risk assessments as providing supporting information. Specifically, the Administrator judged that a standard set at 75 ppb would be appreciably below 80 ppb, the level in controlled human exposure studies at which adverse effects had been demonstrated at the time, and would provide a significant increase in protection compared to the then-current standard. Based on results of the exposure assessment, he also noted that exposures to O₃ concentrations at and above a benchmark level of 80 ppb would be essentially eliminated with a standard level of 75 ppb, and that exposures at and above a 70 ppb benchmark level would be substantially reduced or eliminated for the vast majority of people in at-risk groups. In addition, the Administrator concluded that the body of evidence did not support setting a lower standard level, specifically judging that the available evidence for effects following exposures to O₃ concentrations of 60 ppb was “too limited to support a primary focus at this level” (75 FR 2938). With respect to the epidemiologic evidence, the Administrator stated that a standard set at a level lower than 75 ppb “would only result in significant further public health protection if, in fact, there is a continuum of health risks in areas with 8-hour average O₃ concentrations that are well below the concentrations observed in the key controlled human exposure studies and if the reported associations observed in the epidemiological studies are, in fact, causally related to O₃ at those lower levels” (73 FR 16483).

In making his final decision about the level of the primary O₃ standard, the Administrator noted that the level of 75 ppb was above the range recommended by CASAC (i.e., 70 to 60 ppb). He concluded that “CASAC’s recommendation appeared to be a mixture of scientific and policy considerations” (75 FR 2992). The Administrator reached a different policy judgment than the CASAC Panel, placing less weight than CASAC on the available controlled human exposure studies reporting effects following exposures to 60 ppb O₃ and less weight on the results from exposure and risk assessments, particularly on estimates of exposures to O₃ concentrations at or above 60 ppb (73 FR 16482-3).

¹⁸ The D.C. Circuit, in its review of the 2008 primary standard, stated that it was unclear whether CASAC’s advice reflected issues of pure science or issues of science and policy. That is, the court was unable to determine whether CASAC’s conclusion in its 2007 letter that the standard be set no higher than 70 ppb “was based on its scientific judgment that adverse effects would occur at that level or instead based on its more qualitative judgment that the range it proposed would be more appropriately protective of human health with an adequate margin of safety.” *Mississippi*, 744 F. 3d at 1357.

1.3.1.2 Approach for the Current Review

To identify the range of options appropriate for the Administrator to consider in the current review, we apply an approach that builds upon the general approach used in the last review (and in the 2010 reconsideration proposal) and that reflects the broader body of scientific evidence, updated exposure/risk information, and advances in O₃ air quality modeling now available. As summarized above, the Administrator's decisions in the prior review were based on an integration of information on health effects associated with exposure to O₃, judgments on the adversity and public health significance of key health effects, and expert and policy judgments as to when the standard is requisite to protect public health with an adequate margin of safety.

Staff's conclusions on the primary O₃ standard reflect our consideration of the available scientific evidence, exposure/risk information, and air quality modeling information, within the context of the overarching questions related to: (1) the adequacy of the current primary O₃ standard to protect against effects associated with both short- and long-term exposures and (2) potential alternative standards that are appropriate to consider in this review. In addressing these broad questions, we organize the discussions in chapters 3 and 4 of this document around a series of more specific questions reflecting different aspects of each overarching question. When evaluating the health protection afforded by the current and potential alternative standards, we take into account the four basic elements of the NAAQS: the indicator, averaging time, form, and level.

Figure 1-1 below provides an overview of our approach in this review. We believe that the general approach summarized in this section, and outlined in Figure 1-1, provides a comprehensive basis to help inform the judgments required of the Administrator in reaching decisions about the current and potential alternative primary O₃ standards. In the subsections below, we describe our general approaches to considering the scientific evidence (evidence-based considerations) and to considering the human exposure- and health risk information (exposure- and risk-based considerations). We also recognize considerations related to ambient O₃ attributable to background sources.

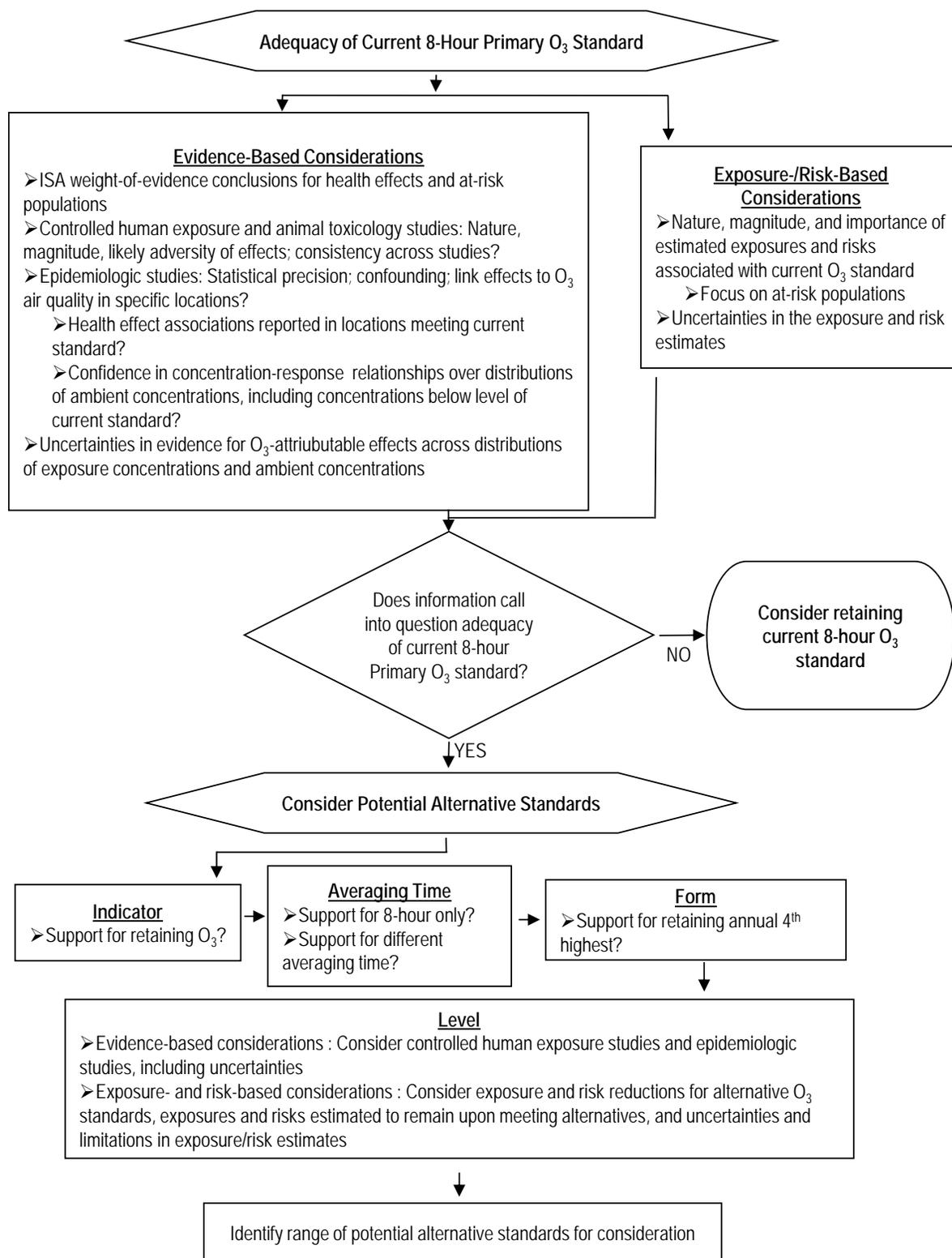


Figure 1-1. Overview of approach to reviewing the primary standard.

1.3.1.2.1 Consideration of the Scientific Evidence

Our approach in this review draws upon an integrative synthesis of the entire body of available scientific evidence for O₃-related health effects, including the evidence newly available in the current review and the evidence from previous reviews, as presented in the ISA (U.S. EPA, 2013).¹⁹ Our approach to considering the scientific evidence is based fundamentally on using information from controlled human exposure and epidemiologic studies, supplemented by information from animal toxicology studies. Such evidence informs our consideration of the health endpoints and at-risk populations on which to focus the current review, and our consideration of the O₃ concentrations at which various health effects can occur.

Since the 2008 review of the O₃ NAAQS, the Agency has developed formal frameworks for characterizing the strength of the scientific evidence with regard to health effects associated with exposures to O₃ in ambient air and factors that may increase risk in some populations or lifestages (U.S. EPA, 2013, Preamble; Chapter 8). These frameworks provide the basis for robust, consistent, and transparent processes for evaluating the scientific evidence, including uncertainties in the evidence, and for drawing weight-of-evidence conclusions on air pollution-related health effects and at-risk populations.

With regard to characterization of health effects, the ISA uses a five-level hierarchy to classify the overall weight-of-evidence into one of the following categories: causal relationship, likely to be a causal relationship, suggestive of a causal relationship, inadequate to infer a causal relationship, and not likely to be a causal relationship (U.S. EPA, 2013, Preamble Table II). In using the weight of evidence approach to inform judgments about the degree of confidence that various health effects are likely to be caused by exposure to O₃, confidence increases as the number of studies consistently reporting a particular health endpoint grows and as other factors, such as biological plausibility and strength, consistency and coherence of evidence increases. Conclusions about biological plausibility, consistency and coherence of O₃-related health effects are drawn from the integration of epidemiologic studies with mechanistic information from controlled human exposure and animal toxicological studies, as discussed in the ISA (U.S. EPA, 2013, EPA Framework for Causal Determination, p. 1viii). In this PA, we place the greatest

¹⁹Selection 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. Policy relevant and informative studies include those that provide a basis for or describe the relationship between the criteria pollutant and effects. This includes studies that offer innovation in method or design and studies that reduce uncertainty on critical issues, such as analyses of confounding or effect modification by copollutants or other variables, analyses of concentration-response or dose-response relationships, or analyses related to time between exposure and response. Review articles, by contrast, are generally not included because they typically present summaries or interpretations of existing studies. The specific criteria applied to the various types of studies are discussed in more detail in the Preamble to the ISA (U.S. EPA, 2013, Preamble).

weight on the health effects for which the evidence has been judged in the ISA to demonstrate a causal or a “likely to be” causal relationship with O₃ exposures. Our consideration of the available evidence for such effects is presented below in Chapter 3 (consideration of the adequacy of the current standard) and in Chapter 4 (consideration of potential alternative standards).

As discussed below, we further consider the evidence base assessed in the ISA with regard to the types and levels of exposure at which health effects are indicated. This further consideration of the evidence, which directly informs EPA’s conclusions regarding the adequacy of current or potential alternative standards in providing requisite public health protection, differs from consideration of the evidence in the ISA with regard to overarching determinations of causality. Therefore, studies that inform determinations of causality may or may not be concluded to be informative with regard to the adequacy of the current or potential alternative standards.²⁰

As with health endpoints, the ISA’s characterization of the weight-of-evidence for potential at-risk populations is based on the evaluation and synthesis of evidence from across scientific disciplines. The ISA characterizes the evidence for a number of “factors” that have the potential to place populations at increased risk for O₃-related effects. The categories considered in evaluating the evidence for these potential at-risk factors are “adequate evidence,” “suggestive evidence,” “inadequate evidence,” and “evidence of no effect.” These categories are discussed in more detail in the ISA (U.S. EPA, 2013, chapter 8, Table 8-1). In this PA, we focus our consideration of potential at-risk populations on those factors for which the ISA judges there is “adequate” evidence (U.S. EPA, 2013, Table 8-6). At-risk populations are discussed in more detail in section 3.2.1, below.

Using the available scientific evidence to inform conclusions on the current and potential alternative standards is complicated by the recognition that a population-level threshold has not been identified, below which it can be concluded with confidence that O₃-attributable effects do not occur (U.S. EPA, 2013, section 2.5.4.4). In the absence of a discernible threshold, our general approach to considering the available O₃ health evidence involves characterizing our confidence in the extent to which O₃-attributable effects occur, and the extent to which such effects are adverse, over the ranges of O₃ exposure concentrations evaluated in controlled human exposure studies and over the distributions of ambient O₃ concentrations in locations where epidemiologic studies have been conducted. As noted above, we recognize that the available

²⁰For example, as discussed further in this section and in Chapters 3 and 4 of this PA, we judge that health studies evaluating exposure concentrations near or below the level of the current standard and epidemiologic studies conducted in locations meeting the current standard are particularly informative when considering the adequacy of the public health protection provided by the current standard.

health effects evidence reflects a continuum from relatively high O₃ concentrations, at which scientists generally agree that adverse health effects are likely to occur, through lower concentrations, at which the likelihood and magnitude of a response become increasingly uncertain. Aspects of our approach particular to evidence from controlled human exposure and epidemiologic studies, respectively, are discussed below.

Controlled Human Exposure Studies

Controlled human exposure studies provide direct evidence of relationships between pollutant exposures and human health effects (U.S. EPA, 2013, p.lx). Controlled human exposure studies provide data with the highest level of confidence since they provide human effects data under closely monitored conditions and can provide exposure response relationships. Such studies are particularly useful in defining the specific conditions under which pollutant exposures can result in health impacts, including the exposure concentrations, durations, and ventilation rates under which effects can occur. As discussed in the ISA, controlled human exposure studies provide clear and compelling evidence for an array of human health effects that are directly attributable to acute exposures to O₃ *per se* (i.e., as opposed to O₃ and other photochemical oxidants, for which O₃ is an indicator, or other co-occurring pollutants) (U.S. EPA, 2013, Chapter 6). Together with animal toxicological studies, which can provide information about more serious health outcomes as well as the effects of long-term exposures and mode of action, controlled human exposure studies also help to provide biological plausibility for health effects observed in epidemiologic studies.

In this PA, we consider the evidence from controlled human exposure studies in two ways. First, we consider the extent to which controlled human exposure studies provide evidence for health effects following exposures to different O₃ concentrations, down to the lowest-observed-effects levels in those studies. Second, we use such studies to inform our evaluation of the extent to which we have confidence in health effect associations reported in epidemiologic studies down through lower ambient O₃ concentrations, where the likelihood and magnitude of O₃-attributable effects become increasingly uncertain.

We consider the range of O₃ exposure concentrations evaluated in controlled human exposure studies, including concentrations near or below the level of the current standard. We consider both group mean responses, which provide insight into the extent to which observed changes are due to O₃ exposures rather than to chance alone, and inter-individual variability in responses, which provides insight into the fraction of the population that might be affected by such O₃ exposures (U.S. EPA, 2013, section 6.2.1.1). When considering the relative weight to place on various controlled human exposure studies, we consider the exposure conditions evaluated (e.g., exercising versus resting, exposure duration); the nature, magnitude, and likely adversity of effects over the range of reported O₃ exposure concentrations; the statistical

precision of reported effects; and the consistency of results across studies for a given health endpoint and exposure concentration. In addition, because controlled human exposure studies typically involve healthy individuals and do not evaluate the most sensitive individuals in the population (U.S. EPA, 2013, Preamble p. lx), when considering the implications of these studies for our evaluation of the current and potential alternative standards, we also consider the extent to which reported effects are likely to reflect the magnitude and/or severity of effects in at-risk groups.

Epidemiologic Studies

We also consider epidemiologic studies of short- and long-term O₃ concentrations in ambient air. Epidemiologic studies provide information on associations between variability in ambient O₃ concentrations and variability in various health outcomes, including lung function decrements, respiratory symptoms, school absences, hospital admissions, emergency department visits, and premature mortality (U.S. EPA, 2013, Chapters 6 and 7). Epidemiologic studies can inform our understanding of the effects in the study population (which may include at-risk groups) of real-world exposures to the range of O₃ concentrations in ambient air, and can provide evidence of associations between ambient O₃ levels and serious acute and chronic health effects that cannot be assessed in controlled human exposure studies. For these studies, the degree of uncertainty introduced by confounding variables (e.g., other pollutants, temperature) and other factors affects the level of confidence that the health effects being investigated are attributable to O₃ exposures, alone and in combination with copollutants.

Available studies have generally not indicated a discernible population threshold, below which O₃ is no longer associated with health effects (U.S. EPA, 2013, section 2.5.4.4). However, the currently available epidemiologic evidence indicates decreased confidence in reported concentration-response relationships for O₃ concentrations at the lower ends of ambient distributions (U.S. EPA, 2013, section 2.5.4.4). Therefore, our general approach to considering the results of epidemiologic studies within the context of the current and potential alternative standards focuses on characterizing the range of ambient O₃ concentrations over which we have the most confidence in O₃-associated health effects, and the concentrations below which our confidence in such health effect associations becomes appreciably lower. In doing so, we consider the statistical precision of O₃ health effect associations reported in study locations with various ambient O₃ concentrations; confidence intervals around concentration-response functions reported over distributions of ambient O₃ (where available); and the extent to which the biological plausibility of associations at various ambient O₃ concentrations is supported by evidence from controlled human exposure and/or animal toxicological studies.

We consider both multi-city and single-city studies assessed in the ISA, each of which have strengths and limitations. Multi-city studies evaluate large populations and provide greater

statistical power than single-city studies. Multi-city studies also reflect O₃-associated health impacts across a range of diverse locations, providing spatial coverage for different regions and reflecting differences in exposure-related factors that could impact O₃ risks. In addition, compared to single-city studies, multi-city studies are less prone to publication bias and they afford the possibility of generalizing to the broader national population (U.S. EPA, 2004, p. 8-30). In contrast, while single-city studies are more limited than multicity studies in terms of statistical power and geographic coverage, conclusions regarding the extent to which air quality met the current or potential alternative standards in the cities for which associations have been reported can be made with greater certainty for single-city studies (compared to multicity studies reporting only multicity effect estimates) because the associations are reported for city-specific analyses (U.S. EPA, 2011d, section 2.3.4.1).²¹ In some cases, single-city studies can also provide evidence for locations or population-specific characteristics not reflected in multicity studies (U.S. EPA, 2013, section 6.2.7.1). Therefore, when considering available epidemiologic studies we evaluate both multi-city and single-city studies, recognizing the strengths and limitations of each.

In placing emphasis on specific epidemiologic studies, we focus on studies conducted in the U.S. and Canada. Such studies reflect air quality and exposure patterns that are likely more typical of the U.S. population than the air quality and exposure patterns reflected in studies conducted outside the U.S. and Canada.²² We also focus on studies reporting associations with effects judged in the ISA to be robust to confounding by other factors, including co-occurring air pollutants.

1.3.1.2.2 Consideration of Exposure and Risk Estimates

To put judgments about O₃-related health effects into a broader public health context, we consider exposure and risk estimates from the HREA, which develops and applies models to estimate human exposures to O₃ and O₃-related health risks in urban case study areas across the United States (U.S. EPA, 2014a). The HREA estimates exposures of concern, based on interpreting quantitative exposure estimates within the context of controlled human exposure study results; lung function risks, based on applying exposure-response relationships from controlled human exposure studies to quantitative estimates of exposures; and epidemiologic-based risk estimates, based on applying concentration-response relationships drawn from epidemiologic studies to adjusted air quality. Each of these types of assessments is discussed briefly below.

²¹ Though in some cases multicity studies present single-city effect estimates in addition to multi-city estimates.

²² Though we recognize that a broader body of studies, including international studies, inform the causal determinations in the ISA.

As in the 2008 review, the HREA estimates exposures at or above benchmark concentrations of 60, 70, and 80 ppb, reflecting exposure concentrations of concern based on the available health evidence.²³ Estimates of exposures of concern, defined as personal exposures while at moderate or greater exertion to 8-hour average ambient O₃ levels, at or above these discrete benchmark concentrations provide perspective on the public health impacts of O₃-related health effects that are plausibly linked to the more serious effects seen in epidemiological studies, but cannot be evaluated in quantitative risk assessments. They also help elucidate the extent to which such impacts may be reduced by meeting the current and alternative standards. Estimates of the number of people likely to experience exposures of concern cannot be directly translated into quantitative estimates of the number of people likely to experience specific health effects due to individual variability in responsiveness. Only a subset of individuals can be expected to experience such adverse health effects, and at-risk populations or lifestages, such as people with asthma or children, are expected to be affected more by such exposures than healthy adults. Though this analysis is conducted using discrete benchmark concentrations, health-relevant exposures are more appropriately viewed as a continuum with greater confidence and less uncertainty about the existence of health effects at higher O₃ exposure concentrations and less confidence and greater uncertainty at lower exposure concentrations. This approach recognizes that there is no sharp breakpoint within the exposure-response relationship for exposure concentrations at and above 80 ppb down to 60 ppb.

The HREA also generates quantitative estimates of O₃ health risks for air quality adjusted to just meet the current and potential alternative standards. As noted above, one approach to estimating O₃ health risks is to combine modeled exposure estimates with exposure-response relationships derived from controlled human exposure studies of O₃-induced health effects. The HREA uses this approach to estimate the occurrence of O₃-induced lung function decrements in at-risk populations in urban case study areas, including school-age children, school-age children with asthma, adults with asthma, and older adults. The available exposure-response information does not support this approach for other endpoints evaluated in controlled human exposure studies (U.S. EPA, 2014a, section 2.2.5 to 2.2.7).

Another approach to estimating O₃-associated health risks is to apply concentration-response relationships derived from short- and/or long-term epidemiologic studies to air quality adjusted to just meet current and potential alternative standards. The concentration-response relationships drawn from epidemiologic studies are based on population exposure surrogates, such as 8-hour concentrations averaged across monitors and over more than one day

²³ For example, see 75 FR 2945-2946 (January 19, 2010) and 73 FR 16441-16442 (March 27, 2008) discussing “exposures of concern”.

(incorporation of lag) (U.S. EPA, 2013, Chapter 6). The HREA presents epidemiologic-based risk estimates for O₃-associated mortality, hospital admissions, emergency department visits, and respiratory symptoms (U.S. EPA, 2014a, Chapter 7). These estimates are derived from the full distributions of ambient O₃ concentrations estimated for the study locations.²⁴ In addition, the HREA estimates mortality risks associated with various portions of distributions of short-term O₃ concentrations (U.S. EPA, 2014a). In this PA we consider risk estimates based on the full distributions of ambient O₃ concentrations and, when available, estimates of the risk associated with various portions of those ambient distributions.²⁵ In doing so, we take note of the ISA conclusions regarding confidence in linear concentration-response relationships over distributions of ambient concentrations, and of the extent to which health effect associations at various ambient O₃ concentrations are supported by the evidence from experimental studies for effects following specific O₃ exposures.

1.3.1.2.3 Considerations Regarding Ambient O₃ Concentration Estimates Attributable to Background Sources

As noted above, our approach in this review utilizes recent advances in modeling techniques to estimate the contributions of U.S. anthropogenic, international anthropogenic, and natural sources to ambient O₃ (discussed in detail in Chapter 2 of this PA). Such model estimates can provide insights into the extent to which different types of background emissions sources contribute to total ambient O₃ concentrations. Consideration of this issue in the current review is informed by the approaches taken in previous reviews, as well as by court decisions in subsequent litigation.

In 1979, the EPA set a 1-hour O₃ standard with a level of 0.12 ppm. Following the final decision in that review, the City of Houston argued that the standard was arbitrary and capricious because natural O₃ concentrations and other physical phenomena in the Houston area made the standard unattainable in that area. The D.C. Circuit rejected this argument, stating that attainability and technological feasibility are not relevant considerations in the promulgation of the NAAQS. The Court also noted that the EPA need not tailor the NAAQS to fit each region or locale, pointing out that Congress was aware of the difficulty in meeting standards in some

²⁴ In previous reviews, including the 2008 review and reconsideration, such risks were separately estimated for O₃ concentrations characterized as above policy-relevant background concentrations. Policy-relevant background concentrations were defined as the distribution of ozone concentrations attributable to sources other than anthropogenic emissions of ozone precursor emissions (e.g., VOC, CO, NO_x) in the U.S., Canada, and Mexico. The decision to estimate total risk across the full range of O₃ concentrations reflects current OAQPS views and consideration of advice from CASAC (Frey and Samet, 2012b).

²⁵ In a series of sensitivity analyses, the HREA also evaluates a series of threshold models for respiratory mortality associated with long-term O₃ concentrations. In this PA we consider these risk estimates based on threshold models, in addition to HREA core estimates based on the linear model (sections 3.2.3.2, 4.4.2.3).

locations and had addressed this difficulty through various compliance related provisions in the Act. See API v. Costle, 665 F.2d 1176, 1184-6 (D.C. Cir. 1981).

More recently, in the 1997 review of the O₃ NAAQS, the Administrator set an 8-hour standard with a level of 0.08 ppm (84 ppb). In reaching this decision, the EPA identified several reasons supporting its decision to reject a more stringent standard of 0.07 ppm. Most importantly, the EPA pointed out the scientific uncertainty at lower concentrations and placed significant weight on the fact that no CASAC panel member supported a standard level set lower than 0.08 ppm (62 FR 38868). In addition to noting the uncertainties in the health evidence for exposure concentrations below 0.08 ppm and the advice of CASAC, the EPA noted that a standard set at a level of 0.07 ppm would be closer to peak background concentrations that infrequently occur in some areas due to nonanthropogenic sources of O₃ precursors (62 FR 38856, 38868; July 18, 1997).

In subsequent litigation, the D.C. Circuit upheld the EPA's decision as the product of reasoned decision-making. The Court made clear that the most important support for the EPA's decision was the health evidence and the concerns it raised about setting a standard level below 0.08 ppm. The Court also pointed to the significant weight that the EPA properly placed on the advice it received from CASAC. Finally (as discussed in section 1.2.2 above), the Court noted that the EPA could also consider relative proximity to peak natural background O₃ when considering alternatives within the range of reasonable values supported by the scientific evidence and judgments of the Administrator. See ATA III, 283 F.3d at 379 (D.C. Cir. 2002).

These cases provide a framework for considering the contributions of U.S. anthropogenic, international anthropogenic, and natural sources within the context of considering the health evidence and CASAC advice, when evaluating various potential alternative standards. Consistent with such a framework, this PA identifies the range of policy options for the primary O₃ standard that staff concludes are appropriate to consider in light of the available scientific evidence and exposure/risk information, and the advice of CASAC. In identifying the range of policy options supported by the evidence and information, staff has not considered proximity to background O₃ concentrations. The Administrator, when evaluating the range of possible standards that are supported by the scientific evidence, could consider proximity to background O₃ concentrations as one factor in selecting the appropriate standard.

1.3.2 Approach for the Secondary Standard

Staff's approach in this review of the current secondary standard takes into consideration aspects of the approaches used in past O₃ NAAQS reviews. The past and current approaches, generally described below, are both based fundamentally on using EPA's assessment of the current scientific evidence and associated quantitative analyses to inform the Administrator's

judgment regarding a secondary standard for O₃ that is requisite (i.e., neither more nor less stringent than necessary) to protect public welfare.

In reaching conclusions on options for the Administrator's consideration, we note that the final decision to retain or revise the current secondary O₃ standard is a public welfare policy judgment to be made by the Administrator. This final decision will draw upon the available scientific evidence for O₃-attributable welfare effects and on analyses of vegetation and ecosystem exposures and public welfare risks based on impacts to vegetation, ecosystems and their associated services, including judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses. In determining the requisite level of protection for crops and trees, the Administrator will need to weigh the importance of the predicted risks of these effects in the overall context of public welfare protection, along with a determination as to the appropriate weight to place on the associated uncertainties and limitations of this information. Our general approach to informing these judgments, discussed more fully below, recognizes that the available evidence demonstrates a range of O₃ sensitivity across studied plant species and documents an array of O₃-induced effects that extend from lower to higher levels of biological organization. These effects range from those affecting cell processes and individual plant leaves to effects on the physiology of whole plants, species effects and effects on plant communities to effects on related ecosystem processes and services. Given this evidence, it is not possible to generalize across all studied species regarding which cumulative exposures are of greatest concern, as this can vary by situation due to differences in exposed species sensitivity, the importance of the observed or predicted O₃-induced effect, the role that the species plays in the ecosystem, the intended use of the affected species and its associated ecosystem and services, the presence of other co-occurring predisposing or mitigating factors, and associated uncertainties and limitations. At the same time, the evidence also demonstrates that though effects of concern can occur at very low exposures in sensitive species, at higher cumulative exposures those effects would likely occur at a greater magnitude and/or higher levels of biological organization and additional species would likely be impacted. It is important to note, however, due to the variability in the importance of the associated ecosystem services provided by different species at different exposures and in different locations, as well as differences in associated uncertainties and limitations, adverse effects observed or predicted at lower exposures along the exposure continuum may or may not have less public welfare significance than those observed at higher cumulative exposures. Therefore, in developing conclusions in this final PA, we take note of the complexity of judgments to be made by the Administrator regarding the adversity of known and anticipated effects to the public welfare and are mindful that the Administrator's ultimate judgments on the secondary standard will most appropriately reflect an interpretation of the available scientific evidence and exposure/risk

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

Section 1.3.2.1 below provides an overview of the general approach taken in the last review of the secondary standard for O₃ (i.e., the 2008 review), and a summary of the rationale for the decision on the standard in that review (73 FR 16436). Section 1.3.2.2 presents our approach in the current review, including our approach to considering the vegetation effects evidence and exposure/risk information, and considerations regarding ambient O₃ concentrations attributable to background sources.

1.3.2.1 Approach Used in the Last Review

In the 2008 review of the secondary NAAQS for O₃, the Administrator relied upon consideration of the available scientific evidence and exposure/risk information, information regarding biologically-relevant exposure indices, air quality information regarding the degree of overlap between different exposure index forms, the advice and recommendations of CASAC, considerations regarding adversity, and comments from the public. Based on all of this, he revised the level of the secondary O₃ standard from 0.08 ppm²⁶ to 0.075 ppm (75 ppb²⁷).

In reaching a decision to revise the 1997 8-hour secondary standard, the Administrator found, after carefully considering the public comments, that the fundamental scientific conclusions on the effects of O₃ on vegetation and sensitive ecosystems reached in the 2006 Criteria Document and 2007 Staff Paper, as discussed in section IV.A of the final rule remained valid (73 FR 16496). He further recognized that several additional lines of evidence had progressed sufficiently since the 1997 review to provide a more complete and coherent picture of the scope of O₃-related vegetation risks (i.e., visible foliar injury, tree biomass loss, crop yield loss, and others), especially those faced by sensitive seedling, sapling and mature growth stage tree species growing in field settings, and their associated forested ecosystems. This new research reflected an increased emphasis on field-based exposure methods (e.g., free-air, ambient gradient and biomonitoring surveys) (73 FR 16490) in addition to the more traditional controlled open-top chamber (OTC) studies (73 FR 16485), and began to address one of the key data gaps cited by the Administrator in the 1997 review (73 FR 16486). Specifically, by providing additional evidence that O₃-induced crop yield loss and tree seedling biomass loss effects observed in chambers also occurs in the field, this new research qualitatively increased support

²⁶ As noted earlier, due to rounding convention, the 1997 standard level of 0.08 ppm corresponded to 0.084 ppm (84 ppb).

²⁷ As explained above, the level of the O₃ standard is specified as 0.075 ppm rather than 75 ppb. However, in this draft PA we refer to ppb, which is most often used in the scientific literature and in the ISA, in order to avoid the confusion that could result from switching units when discussing the evidence in relation to the standard level.

for, and confidence in, the continued use of OTC-derived crop and tree seedling exposure-response (E-R) functions developed in the National Crop Loss Assessment Network (NCLAN) and National Health and Environmental Effects Research Laboratory – Western Ecology Division (NHEERL-WED) studies, respectively, to predict O₃-induced impacts on crops and tree seedlings in the field (72 FR 37886). All of these areas were considered together, along with associated uncertainties, in an integrated weight-of-evidence approach (73 FR 16490).

Beyond the available vegetation effects evidence, the Administrator also considered estimates of O₃ exposures and risks when air quality was adjusted to simulate just meeting the existing and potential alternative standards. On the basis of these assessments, the Administrator concluded that O₃ exposures that would be expected to remain after meeting the existing standard would be sufficient to cause visible foliar injury and seedling and mature tree biomass loss in O₃-sensitive vegetation (73 FR 16496) and would still allow O₃-related yield loss to occur in some commodity crop species and fruit and vegetable species grown in the U.S. (73 FR 16489). Other O₃-induced effects described in the literature, including an impaired ability of many sensitive species and genotypes within species to adapt to or withstand other environmental stresses, such as freezing temperatures, pest infestations and/or disease, and to compete for available resources, would also be anticipated to occur. In the long run, the result of these impairments (e.g., loss in vigor) could lead to premature plant death in O₃ sensitive species. Though effects on other ecosystem components had only been examined in isolated cases, the Administrator noted effects such as those described above could have significant implications for plant community and associated species biodiversity and the structure and function of whole ecosystems (73 FR 16496).

Although the Administrator concluded that the then-current standard was not sufficient to protect against the known and anticipated effects described above, he also recognized that the secondary standard is not meant to protect against all known observed or anticipated O₃-related effects, but only those that can reasonably be judged to be adverse to the public welfare. The Administrator found that the degree to which such effects should be considered to be adverse depended on the intended use of the vegetation and its significance to the public welfare (73 FR 16496). In this regard, he took note of a number of actions taken by Congress to establish public lands that are set aside for specific uses that are intended to provide benefits to the public welfare, including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment of future generations. Based on these considerations, and taking into consideration the advice and recommendations of CASAC, the Administrator concluded that the protection afforded by the existing standard was not sufficient, and that the standard needed to be revised to provide

additional protection from known and anticipated adverse effects on sensitive natural vegetation and ecosystems (73 FR 16497).

Given this judgment, the Administrator then considered what revisions to the standard were requisite to protect public welfare. Regarding the form of the standard, the Administrator took note that at the conclusion of the 1997 review, the biological basis for a cumulative, seasonal form was not in dispute²⁸ and that the 2006 Criteria Document also concluded that O₃ exposure indices that cumulate differentially-weighted hourly concentrations are the best candidates for relating exposure to plant growth responses (EPA, 2006) (61 FR 65716; 73 FR 16486). The CASAC, in its letter to the Administrator following its review of the second draft Staff Paper, stated that “there is a clear need for a secondary standard which is distinctly different from the primary standard in averaging time, level and form” and that “the CASAC unanimously agrees that it is not appropriate to try to protect vegetation from the substantial, known or anticipated, direct and/or indirect, adverse effects of ambient ozone by continuing to promulgate identical primary and secondary standards for ozone” (Henderson, 2006, pp. 5-7). Although many possible cumulative, seasonal concentration-weighted exposure metrics exist, the Staff Paper and the CASAC Panel concluded that the W126²⁹ form is the most biologically-relevant cumulative, seasonal form appropriate to consider in the context of the secondary standard review (73 FR 16486-87).³⁰

Although agreeing with the Criteria Document, Staff Paper and CASAC conclusions that a cumulative exposure index that differentially weights O₃ concentrations could represent a reasonable policy choice for a seasonal secondary standard to protect against the effects of O₃ on vegetation and that the most appropriate cumulative, concentration-weighted form to consider was the sigmoidally-weighted W126 form (73 FR 16498), the Administrator also took note of the 1997 decision to make the revised secondary standard identical to a revised primary standard after similar considerations (73 FR 16498). In considering the rationale for the 1997 decision, the Administrator observed that it was based in part on an analysis that compared the degree of

²⁸ In the 1997 review, a different cumulative metric (SUM06) was proposed.

²⁹ W126 is a cumulative exposure index that is biologically based. The W126 index focuses on the higher hourly average concentrations, while retaining the mid-and lower-level values. It is defined as the sum of sigmoidally-weighted hourly O₃ concentrations over a specified period, where the daily sigmoidal weighting function is defined as: $1 - \exp[-(W126/\eta)^\beta]$

³⁰ In a subsequent letter offering unsolicited advice to the Administrator and Agency staff on development of the proposed rulemaking, the CASAC reiterated that Panel members “were unanimous in supporting the recommendation in the Final Ozone Staff Paper that protection of managed agricultural crops and natural terrestrial ecosystems requires a secondary Ozone NAAQS that is substantially different from the primary ozone standard in averaging time, level and form”...and “[t]he recommended metric for the secondary ozone standard is the (sigmoidally-weighted) W126 index, accumulated over at least the 12 ‘daylight’ hours and over at least the three maximum ozone months of the summer ‘growing season’” (Henderson, March 26, 2007, p.3).

overlap in county-level air quality measured in terms of alternative standard forms (62 FR 38876). Recognizing that significant uncertainty remained in 1997 regarding conclusions drawn from such analyses, the Administrator also considered the results of a similar analysis of recent monitoring data undertaken in the 2007 Staff Paper to assess the degree of overlap expected between the existing standard (4th high, daily maximum 8-hour concentration averaged over three years) and potential alternative standards based on W126 cumulative seasonal forms.

The Administrator noted that this analysis showed significant overlap between the 8-hour secondary standard and selected levels of W126 standard forms, with the degree of overlap between these potential alternative standards depending greatly on the W126 level selected and the distribution of hourly O₃ concentrations within the annual and/or 3-year average period. From this analysis, the Administrator recognized that a secondary standard set identical to a revised primary standard would provide a significant degree of additional protection for vegetation as compared to that provided by the existing secondary standard. In further considering the significant uncertainties in the available body of evidence and in the exposure and risk analyses, and the difficulty in determining at what point various types of vegetation effects become adverse for sensitive vegetation and ecosystems, the Administrator focused his consideration on a level for an alternative W126 standard (with an annual form) at the upper end of the proposed range (i.e., 21 ppm-hours). The Staff Paper analysis showed that at a W126 level of 21 ppm-hours, there would be essentially no counties with air quality expected both to exceed such an alternative W126 standard and to meet the revised 8-hour primary standard—that is, based on this analysis of counties with ambient O₃ monitors, a W126-based level of 21 ppm-hours would be unlikely to provide additional protection in any areas beyond that likely to be provided by the revised 2008 primary standard (73 FR 16499/500).

The Administrator also considered the Staff Paper finding that the degree of overlap between counties (with areas of concern for vegetation) expected to meet an 8-hour level for the form of the existing standard and potential alternative levels of a W126-based standard was inconsistent across years analyzed. This variation depended greatly on levels selected for a W126-based standard and a 3-year average 4th high daily maximum 8-hour standard, respectively, and the distribution of hourly O₃ concentrations within the annual and/or 3-year average period. From this, the Staff Paper recognized the need for caution in evaluating the likely vegetation impacts associated with a given level of air quality expressed in terms of the existing 8-hour average standard in the absence of parallel W126 information. In considering these findings, the Administrator “recognize[d] that the general lack of rural monitoring data made uncertain the degree to which the revised 8-hour standard or an alternative W126 standard would be protective, and that there was the potential for not providing the appropriate degree of protection for vegetation in areas with air quality distributions that resulted in a high cumulative,

seasonal exposure but did not result in high 8-hour average exposures” (73 FR 16500). With regard to the 8-hour standard, he also noted that “[w]hile this potential for under-protection was clear, the number and size of areas [then] at issue and the degree of risk [was] hard to determine. However, such a standard would also tend to avoid the potential for providing more protection than is necessary, a risk that would have arisen from moving to a new form for the secondary standard despite the significant uncertainty in determining the degree of risk for any exposure level and the appropriate level of protection, as well as uncertainty in predicting exposure and risk patterns” (73 FR 16500).

Thus, although the Administrator agreed with the views and recommendations of CASAC that a cumulative, seasonal standard was the most biologically relevant way to relate exposure to plant growth response, he also recognized that there remained significant uncertainties in determining or quantifying the degree of risk attributable to varying levels of O₃ exposure, the degree of protection that any specific cumulative, seasonal standard would produce, and the associated potential for error in determining the secondary standard that would provide a requisite degree of protection—i.e., sufficient but not more than what is necessary. Given these significant uncertainties, the Administrator concluded that establishing a new secondary standard with a cumulative, seasonal form, at that time, would have resulted in uncertain benefits beyond those afforded by the revised primary standard, and therefore, might have been more than necessary to provide the requisite degree of protection (73 FR 16500). Based on his consideration of these issues (73 FR 16497), the Administrator judged that the appropriate balance to be drawn was to set a secondary standard identical in every way to the revised 8-hour primary standard of 0.075 ppm. The Administrator believed that such a standard would be sufficient to protect public welfare from known or anticipated adverse effects, and did not believe that an alternative cumulative, seasonal standard was needed to provide this degree of protection (73 FR 16500).

As noted above, on July 23, 2013 the D.C. Circuit found this approach to be contrary to law because the EPA had failed to identify a level of air quality requisite to protect public welfare and, therefore, the EPA’s comparison between the primary and secondary standards for determining if requisite protection for public welfare was afforded by the primary standard was inherently arbitrary. The court remanded the secondary standard to the EPA for further consideration. 744 F. 3d at 1360-62.

1.3.2.2 Approach for the Current Review

To identify the range of options appropriate for the Administrator to consider in the current review, we apply an approach that builds upon the general approach used in the 2008 review (and in the 2010 reconsideration proposal), and that reflects the broader body of scientific

evidence, updated exposure/risk information, and advances in O₃ air quality modeling now available. As summarized above, the Administrator's decisions in the prior review were based on an integration of information on welfare effects associated with exposure to O₃, judgments on the adversity and public welfare significance of key effects, and, expert and policy judgments as to when the standard is requisite to protect public welfare. These considerations were informed by air quality and related analyses, quantitative exposure and risk assessments, and qualitative assessment of impacts that could not be quantified. In performing the evaluation in this document, we are additionally mindful of the recent remand of the secondary standard by the D.C. Circuit and our approach in the current review incorporates our response to this remand.

Our approach in this review of the secondary O₃ standard also reflects our consideration of the available scientific evidence, information on biologically-relevant exposure indices, exposure/risk information, and air quality modeling information, within the context of overarching questions related to: (1) the adequacy of the current secondary O₃ standard to protect against effects associated with cumulative, seasonal exposures and (2) potential alternative standards, if any, that are appropriate to consider in this review. In addressing these broad questions, we have organized the discussions in chapters 5 and 6 of this document around a series of more specific questions reflecting different aspects of each overarching question. When evaluating the welfare protection afforded by the current or potential alternative standards, we take into account the four basic elements of the NAAQS: the indicator, averaging time, form, and level.

Figure 1-2 below provides an overview of our approach in this review. We believe that the general approach summarized in this section, and outlined in Figure 1-2, provides a comprehensive basis to help inform the judgments required of the Administrator in reaching decisions about the current and potential alternative secondary O₃ standards. In the subsections below, we summarize our general approaches to considering the scientific evidence (evidence-based considerations) and to considering the exposure and risk information (exposure- and risk-based considerations).

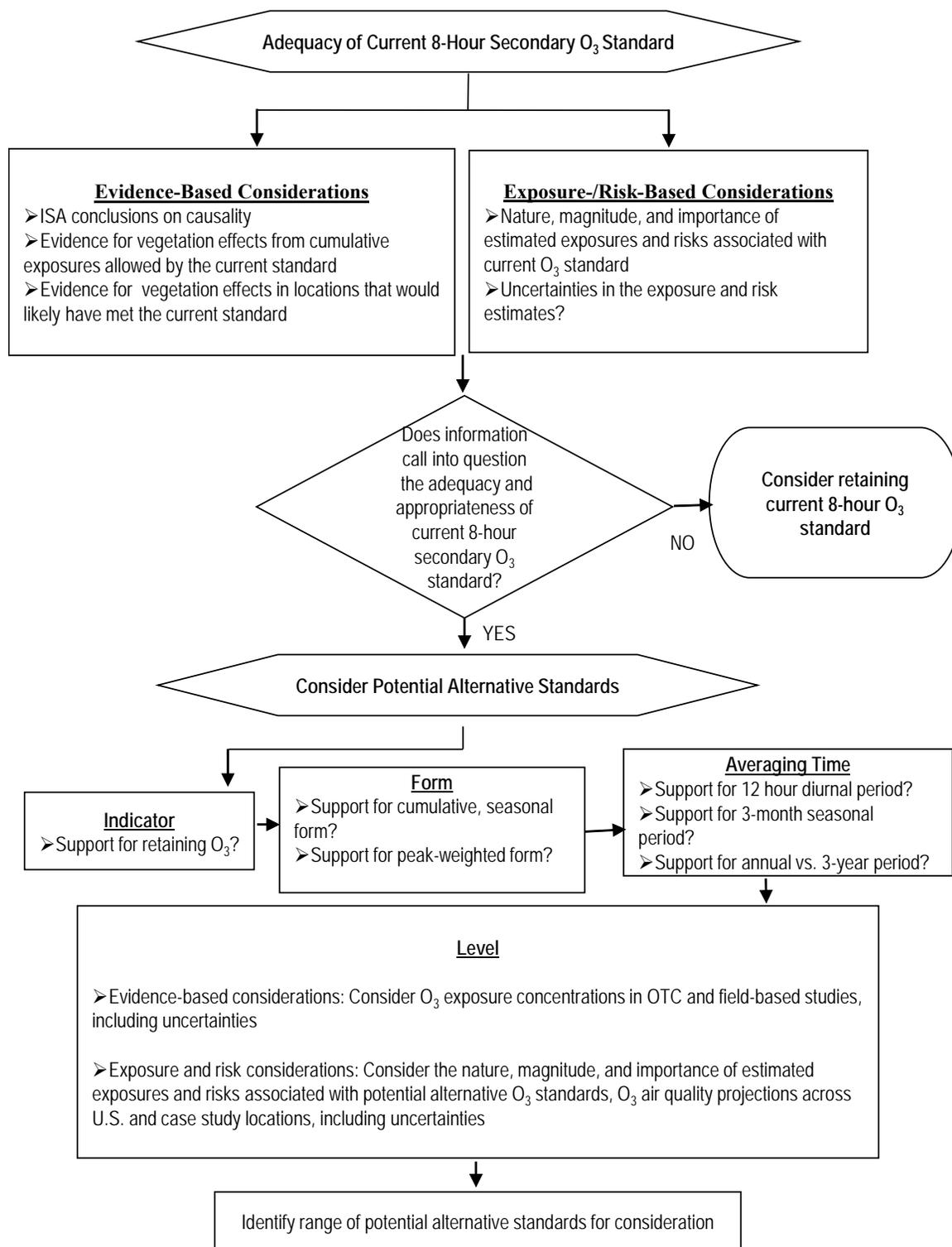


Figure 1-2. Overview of approach to reviewing the secondary standard.

1.3.2.2.1 Consideration of the Scientific Evidence

Our approach in this review draws upon an integrative synthesis of the entire body of available scientific evidence for O₃-related welfare effects, including the evidence newly available in the current review and the evidence from previous reviews, as presented in the ISA (U.S. EPA, 2013). Our approach to considering the scientific evidence for effects on vegetation is based fundamentally on using information from controlled chamber studies and field-based studies. Such evidence informs our consideration of welfare endpoints and at-risk species and ecosystems on which to focus the current review, and our consideration of the ambient O₃ conditions under which various welfare effects can occur.

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

Since the 2008 review of the O₃ NAAQS, the Agency has developed a formal framework for characterizing the strength of the scientific evidence with regard to a causal relationship between ambient O₃ and welfare effects (U.S. EPA, 2013, Preamble; Chapter 9). This framework provides the basis for a robust, consistent, and transparent process for evaluating the scientific evidence, including uncertainties in the evidence, and for drawing weight-of-evidence conclusions regarding air pollution-related welfare effects. In so doing, the ISA uses a five-level hierarchy, classifying the overall weight of evidence into one of the following categories: causal relationship, likely to be a causal relationship, suggestive of a causal relationship, inadequate to infer a causal relationship, and not likely to be a causal relationship (U.S. EPA, 2013, Preamble Table II). In our approach here, we place the greatest weight on the evidence for welfare effects that have been judged in the ISA to be caused by, or likely to be caused by, O₃ exposures. Our consideration of the available evidence for such effects is presented below in Chapter 5 (consideration of the adequacy of the current standard) and in Chapter 6 (consideration of potential alternative standards).

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

Studies that have informed determinations of causality may or may not be concluded to be informative with regard to the adequacy of the current or potential alternative standards.

Our approach in this review, as in past reviews, included recognition that the available evidence has not provided identification of a threshold in exposure or ambient O₃ concentrations below which it can be concluded with confidence that O₃-attributable vegetation effects do not occur across the broad range of O₃-sensitive plant species growing within the U.S. This is due in part to the fact that research shows that there is variability in sensitivity between and within species and that numerous factors, i.e. chemical, physical, biological, and genetic, can influence the direction and magnitude of the studied effect (U.S. EPA, 2013, section 9.4.8). In the absence of a discernible threshold, our general approach to considering the available O₃ welfare evidence involves characterizing our confidence in conclusions regarding O₃-attributable vegetation effects over the ranges of cumulative seasonal O₃ exposure values evaluated in chamber studies and in field studies in areas where O₃-sensitive vegetation are known to occur, as well as characterizing the extent to which these effects can be considered adverse. In addition, because O₃ can indirectly affect other ecosystem components (such as soils, water, and wildlife, and their associated goods and services, through its effects on vegetation) our approach also considers those indirect effects for which the ISA concludes, based on multiple lines of evidence, including mechanistic and physiological processes, to have a causal or likely to be a causal relationship. With respect to ecosystem services for which we may have only limited or qualitative information regarding an association with O₃ exposures, our approach is to consider their policy-relevance in the context of section 109(b) (2) of the CAA which specifies that secondary standards provide requisite protection of “public welfare from any ... known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air”. As noted above, our approach to informing these judgments, discussed more fully below, recognizes that the available welfare effects evidence demonstrates a wide range in O₃ sensitivities across studied plant species. As a result, at relatively high cumulative O₃ exposures, a greater number of plant species will show effects, and the magnitude of the observed effects will be greater, particularly on the more sensitive species, while at lower cumulative O₃ exposures, fewer species will demonstrate effects and the magnitude of those observed effects will be less.

In this review, the evidence base includes quantitative information across a broad array of vegetation effects (e.g., growth impairment during seedlings, saplings and mature tree growth stages, visible foliar injury, and yield loss in annual crops) and across a diverse set of exposure methods from laboratory and field studies. These methods include the more traditional OTC studies, as well as field-based exposure studies. While we consider the full breadth of information available, we place greater weight on U.S. studies due to the often species-, site-, and climate-specific nature of O₃-related vegetation responses. We especially weight those

studies that include O₃ exposures that fall within the range of those likely to occur in the ambient air. Further, our approach in the context of the quantitative exposure and risk assessments (discussed below), places greatest emphasis on studies that have evaluated plant response over multiple exposure levels and developed exposure-response relationships that allow the prediction (estimation) of plant responses over the range of potential alternative standards being assessed.

In considering the evidence, we recognize differences across different study types in what information they provide. For example, because conditions can be controlled in laboratory studies, responses in such studies may be less variable and smaller differences may be easier to detect. However, the control conditions may limit the range of responses or incompletely reflect pollutant bioavailability, so they may not reflect responses that would occur in the natural environment. Alternatively, field data can provide important information for assessments of multiple stressors or where site-specific factors significantly influence exposure. They are also often useful for analyses of larger geographic scales and higher levels of biological organization. However, because most field study conditions cannot be controlled, variability is expected to be higher and differences harder to detect. The presence of confounding factors can also make it difficult to attribute observed effects to specific stressors.

In considering information from across multiple lines of evidence, our approach is to first integrate the evidence from both controlled and field-based studies and assess the coherence and consistency across the available evidence for each effect. We then consider the extent to which these identified effects should be considered adverse to the public welfare, relying largely on the paradigm used in the 2008 review and 2010 proposed reconsideration (e.g., 75 FR 3006). This paradigm recognizes that the significance to the public welfare of O₃-induced effects on sensitive vegetation growing within the U.S. can vary depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located. Accordingly, any given O₃-related effect on vegetation and ecosystems (e.g., biomass loss, crop yield loss, foliar injury) may be judged to have a different degree of impact on the public welfare depending, for example, on whether that effect occurs in a Class I area, a city park, or commercial cropland. Our approach takes this variation in the significance of O₃-related vegetation effects into account in evaluating the currently available evidence with regard to the extent to which it calls into question the adequacy of the current standard and, as appropriate, indicates potential alternative standards that would be appropriate for the Administrator to consider. In the 2010 proposed reconsideration, the Administrator proposed to place the highest priority and significance on vegetation and ecosystem effects to sensitive species that are known to or are likely to occur in federally protected areas such as national parks and other Class I areas, or on lands set aside by States, Tribes and public interest groups to provide similar benefits to the public welfare (75 FR 3023/24). Our approach in this

review considers whether newly available information would suggest any evolution to this paradigm, in particular in the context of considering associated ecosystem services.

Finally, our approach continues to give great weight to the scientific evidence available in this and previous reviews indicating the relevance of cumulative, seasonal, concentration-weighted exposures in inducing vegetation effects. More specifically, in the 2008 and 2010 reviews, the EPA concluded and the CASAC agreed that the W126 cumulative exposure metric was the most appropriate to use in this review to evaluate both the adequacy of the current secondary standard and the appropriateness of any potential revisions. As discussed in chapter 5 in this PA, the information available in this review continues to support the use of such a metric and does not call into question the appropriateness of using the W126 metric in this context. Therefore, both the WREA and PA continue to express exposures in terms of the W126 index, and continue to consider the important policy implications regarding selection of an appropriate exposure index for vegetation. Our approach also places primary emphasis on studies that evaluated plant response to exposures that were or can be described using such an index. The policy-relevant discussions in chapters 5 and 6 focus on vegetation effects evidence and exposure/risk information that can be associated with cumulative, seasonal peak-weighted exposures, where possible. Discussions pertaining to the adequacy of the current secondary standard will consider what cumulative seasonal exposures would be allowed under air quality that would just meet the current standard.

1.3.2.2.2 Consideration of Exposure and Risk Estimates and Air Quality Analyses

To put judgments about O₃-related vegetation and ecosystem effects and services into a broader public welfare context, we consider national scale exposure and risk assessments described in the WREA (U.S. EPA, 2014b). We particularly focused on the WREA quantitative risks related to three types of vegetation effects: foliar injury, biomass loss, and crop yield loss. These risks were assessed in a range of WREA analyses variously involving recent O₃ monitoring data and/or national-scale adjusted air quality scenarios for the current secondary standard and, in some analyses, for a cumulative, seasonal W126 form at one or more levels (15, 11 and 7 ppm-hours). Our consideration of these WREA results provide insight into the extent to which the current or potential alternative standards would be expected to maintain distributions of cumulative, seasonal O₃ exposures below those associated with adverse vegetation effects.

With regard to quantitative O₃ risks related to welfare effects and ecosystem services for foliar injury, we consider two main analyses in the WREA: a screening-level assessment of 214 National Parks and a case study focused on three National Parks. In the screening-level assessment, O₃ concentrations in national parks are assessed using criteria developed from a U.S. Forest Service nationwide dataset on foliar injury, ambient O₃ concentrations (in terms of W126

index) and soil moisture (which can influence susceptibility of vegetation to foliar injury). Additionally, we consider a case study for three Class I areas (Great Smoky Mountain National Park, Rocky Mountain National Park, and Sequoia/Kings Canyon National Park). We consider results from this case study for three metrics: 1) percent of vegetation cover affected by foliar injury; 2) percent of trails affected by foliar injury; 3) estimates of species specific biomass loss within the case study area. We also consider qualitative analyses on ecosystem services effects for this endpoint. For example, the WREA uses GIS mapping to illustrate where effects may be occurring and relates those areas to national scale statistics for recreational use and data on hiking trails, campgrounds and other park amenities that intersect with potentially affected areas. These are used to identify impacts on ecosystem services associated with recreation in national parks. We additionally consider analyses showing associations between elevated O₃ concentrations and increased vulnerability to fire risk regimes, insect attacks and impacts on hydrological cycles.

With regard to risks related to biomass and crop yield loss, we consider WREA results based on exposure-response functions for tree and crop species that predict the growth or yield response of each species, based on the exposure patterns estimated within its growing region. To compare exposure-response across species, genotypes or experiments for which absolute response values may vary greatly, the WREA instead uses estimates of relative biomass loss for trees or yield loss for crops. The WREA develops such estimates nationally and separately for more than 100 federally designated Class I areas. Additionally, we consider WREA-developed estimates of associated impacts on the agriculture and forestry sectors quantifying how O₃ exposure to vegetation is estimated to affect the provision of timber and crops and carbon sequestration. We consider estimates for impacts related to tree biomass loss on ecosystem services such as pollution removal, carbon storage and sequestration in five urban case study areas. We consider biomass and crop yield loss estimates in light of advice from CASAC, as discussed in sections 5.3 and 5.4 below.

In considering the amount of weight to place on the estimates of exposures and risks at or above specific W126 values described in the WREA, our approach: 1) evaluates the weight of the scientific evidence concerning vegetation effects associated with those O₃ exposures; 2) considers the importance, from a public welfare perspective, of the O₃-induced effects on sensitive vegetation and associated ecosystem services that are known or anticipated to occur as a result of exposures at selected W126 values; and, 3) recognizes that predictions of effects associated with any given O₃ exposure may be mitigated or exacerbated by actual conditions in the field (i.e., co-occurring modifying environmental and genetic factors). When considering analyses in the WREA that involve discrete exposure levels or varying levels of severity of effects, our approach to informing these judgments recognizes that the available welfare effects

evidence demonstrates a wide range in O₃ sensitivities across studied plant species. As a result, at relatively high cumulative O₃ exposures, a greater number of plant species will show effects, and the magnitude of the observed effects will be greater, particularly on the more sensitive species, while at lower cumulative O₃ exposures, fewer species will demonstrate effects and the magnitude of those observed effects will be less. We recognize that there is no sharp breakpoint along this continuum of effects incidence and severity, ranging from concentrations at and above the level of the current secondary standard down to the lowest cumulative, seasonal W126 value assessed. In considering these results in this PA, we consider both the potential for welfare effects and their severity and our understanding of the likelihood of such effects at different O₃ exposures.

1.3.2.2.1 Considerations Regarding Ambient O₃ Concentration Estimates Attributable to Background Sources

As noted above, our approach in this review utilizes recent advances in modeling techniques to estimate the contributions of U.S. anthropogenic, international anthropogenic, and natural sources to ambient O₃ (discussed in detail in Chapter 2 of this document). Such model estimates can provide insights into the extent to which different types of emissions sources contribute to total ambient O₃ concentrations. Our consideration of this issue in the current review is informed by the approaches taken in previous reviews, and by court decisions on subsequent litigation, as discussed in section 1.3.1.2.3 above. Further, in the 1996 proposal, O₃ background concentrations were one of the factors the Administrator considered in selecting the SUM06 index as a form for an alternative secondary standard. This and other cumulative exposure indices under consideration were judged to be equally capable at estimating exposures relevant to vegetation, given the lack of evidence for a discernible threshold for vegetation effects in general (U.S. EPA 1996, p. 225), which might have provided a scientific basis for selecting among different cumulative exposure indices. At that time, the SUM06 metric was selected over the W126 metric because it focused on the policy-relevant (above background) portion of the total cumulative seasonal exposures reaching plants (62 FR 38856). At the conclusion of that review, the Administrator ultimately chose to set the secondary standard identical to the primary standard, including using the 8-hour average instead of a cumulative seasonal form (62 FR 38868). In the 2008 review, staff analyses concluded that the W126 index was more biologically-relevant based on the available science; staff additionally noted, based on then-available estimates of background, that this form was also not likely to be significantly impacted by background concentrations given the very low weight assigned to lower O₃ concentrations by the W126 index (U.S. EPA, 2007 SP, 7-22; 72 FR 37893). In this review, the degree to which the total value of the W126 index could be contributed by background concentrations was again assessed. Based on a limited analysis, described in chapter 2 of the

PA, background O₃ (BGO₃) can comprise a non-negligible portion of W126 across the U.S., have the greatest contributions to W126 in the intermountain western U.S., and because of the sigmoidal weighting function that emphasizes the background contributions to the highest hourly ozone values (when BGO₃ contributions are generally lowest), proportionally contribute slightly less for the W126 than for seasonal means of maximum daily 8 hour average values. As with the primary standard, in identifying the range of policy options supported by the evidence and information, staff has not considered proximity to background O₃ concentrations. The Administrator, when evaluating the range of possible standards that are supported by the scientific evidence, could consider proximity to background O₃ concentrations as one factor in selecting the appropriate standard.

1.3.3 Organization of this Document

Chapter 2 of this PA provides an overview of the O₃ ambient monitoring network and O₃ air quality, including estimates of O₃ concentrations attributable to background sources. The remaining chapters are organized into two main parts. Chapters 3 and 4 focus on the review of the primary O₃ NAAQS while chapters 5 and 6 focus on the review of the secondary O₃ NAAQS. Staff's considerations and conclusions related to the current primary and secondary standards are discussed in chapters 3 and 5, respectively. Staff's considerations and conclusions related to potential alternative primary and secondary standards are discussed in chapters 4 and 6, respectively. Key uncertainties in the review and areas for future research and data collection are additionally identified in chapters 4 and 6 for the two types of standards.

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2 O₃ MONITORING AND AIR QUALITY

This section provides overviews of ambient O₃ monitoring in the U.S. (section 2.1); O₃ precursor emissions and atmospheric chemistry (section 2.2); ambient O₃ concentrations (section 2.3); and available evidence and information related to background O₃ (section 2.4). These issues are also discussed in detail in chapter 3 of the Integrated Science Assessment (ISA) (US EPA, 2013).

2.1 O₃ MONITORING

2.1.1 O₃ Monitoring Network

To monitor compliance with the NAAQS, state and local environmental agencies operate O₃ monitoring sites at various locations, depending on the population of the area and typical peak O₃ concentrations.¹ All of the state and local monitoring stations that report data to the EPA AQS use ultraviolet (UV) Federal Equivalent Methods (FEMs). The Federal Reference Method (FRM) is no longer used due to lack of availability and safety concerns.² In 2010, there were over 1,300 state, local, and tribal O₃ monitors reporting concentrations to EPA. The “State and Local Monitoring Stations” (SLAMS) minimum monitoring requirements to meet the O₃ design criteria are specified in 40 CFR Part 58, Appendix D. The requirements are both population and design value based.³ The minimum number of O₃ monitors required in a Metropolitan Statistical Area (MSA) ranges from zero for areas with a population of at least 50,000 and under 350,000 with no recent history of an O₃ design value greater than 85 percent of the NAAQS, to four for areas with a population greater than 10 million and an O₃ design value greater than 85 percent of the NAAQS. Within an O₃ network, at least one site for each MSA, or Combined Statistical Area (CSA) if multiple MSAs are involved, must be designed to record the maximum concentration for that particular metropolitan area. Since highest O₃ concentrations tend to be associated with particular seasons for various locations, EPA requires ozone monitoring during specific ozone monitoring seasons which vary by state.⁴

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

² EPA is developing a new O₃ Federal Reference Method (FRM) and proposed changes to the FEM testing requirements to reflect new and improved measurement technology.

³ A design value is a statistic that describes the air quality status of a given area relative to the level of the NAAQS. Design values are typically used to classify nonattainment areas, assess progress towards meeting the NAAQS, and develop control strategies. See <http://epa.gov/airtrends/values.html> (U, 2010, 677582) for guidance on how these values are defined.

⁴ The required O₃ monitoring seasons for each state are listed in Table D-3 of Appendix D to 40 CFR Part 58. EPA plans to complete an analysis using certified data for the years of 2010-2012 to determine if any changes to the length of the required O₃ monitoring seasons would be needed to support a revised NAAQS.

Figure 2-1 shows the locations of the U.S. ambient O₃ monitoring sites reporting data to EPA at any time during the 2006-2010 period. The gray dots which make up over 80% of the O₃ monitoring network are SLAMS monitors, which are operated by state and local governments to meet regulatory requirements and provide air quality information to public health agencies. Thus, the SLAMS monitoring sites are largely focused on urban and suburban areas. The blue dots highlight two important subsets of monitoring sites within the SLAMS network: the “National Core” (NCore) multi-pollutant monitoring network and the “Photochemical Assessment Monitoring Stations” (PAMS) network.⁵

While the existing U.S. O₃ monitoring network has a largely urban focus, to address ecosystem impacts of O₃ such as biomass loss and foliar injury, it is equally important to focus on O₃ monitoring in rural areas. The green dots in Figure 2-1 represent the Clean Air Status and Trends Network (CASTNET) monitors which are located in rural areas. There were about 80 CASTNET sites operating in 2010, with sites in the eastern U.S. being operated by EPA and sites in the western U.S. being operated by the National Park Service (NPS). Finally, the black dots represent “Special Purpose Monitoring Stations” (SPMS), which include about 20 rural monitors as part of the “Portable O₃ Monitoring System” (POMS) network operated by the NPS. Between the CASTNET, NCore, and POMS networks, there were about 120 rural O₃ monitoring sites operating in the U.S. in 2010.

⁵ EPA is currently developing proposed revisions to the PAMS network design intended to increase coverage and allow for more locale-specific flexibility.

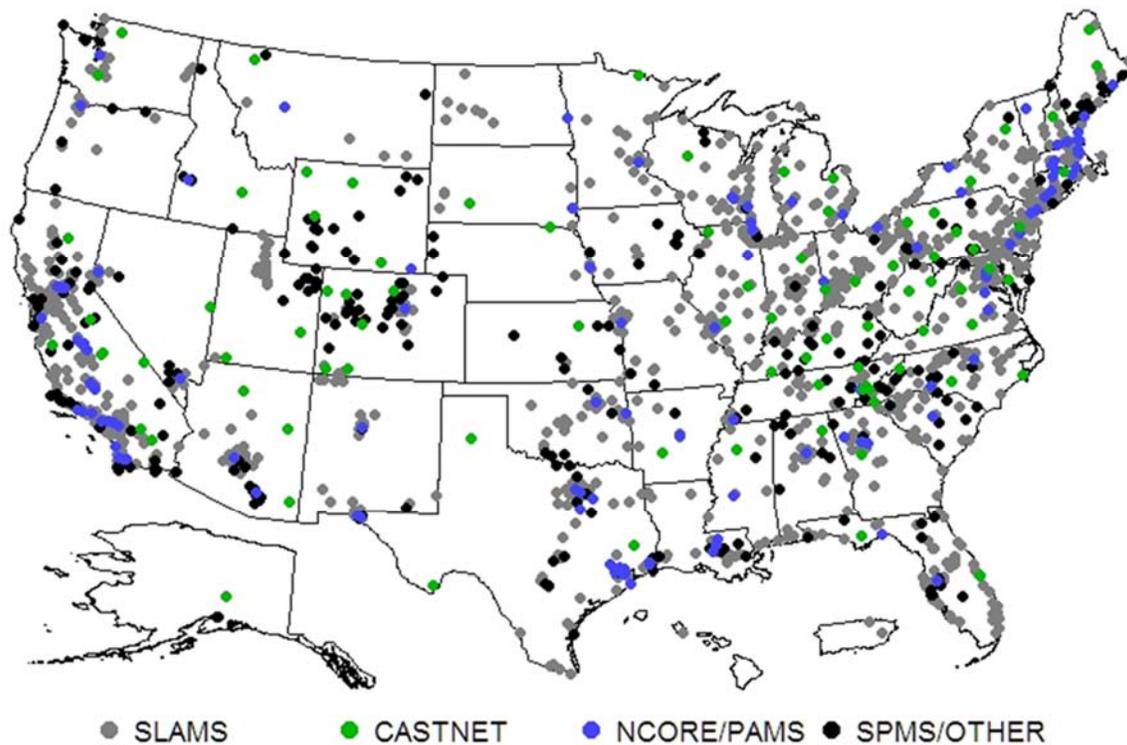


Figure 2-1. Map of U.S. ambient O₃ monitoring sites reporting data to EPA during the 2006-2010 period.

2.1.2 Recent O₃ Monitoring Data and Trends

To determine whether or not the O₃ NAAQS has been met at an ambient monitoring site, a statistic commonly referred to as a “design value” must be calculated based on three consecutive years of data collected from that site. The form of the existing O₃ NAAQS design value statistic is the 3-year average of the annual 4th highest daily maximum 8-hour O₃ concentration in parts per billion (ppb), with decimal digits truncated. The existing primary and secondary O₃ NAAQS are met at an ambient monitoring site when the design value is less than or equal to 75 ppb.⁶ In counties or other geographic areas with multiple monitors, the area-wide design value is defined as the design value at the highest individual monitoring site, and the area is said to have met the NAAQS if all monitors in the area are meeting the NAAQS.

Figure 2-2 shows the trend in the annual 4th highest daily maximum 8-hour O₃ concentrations in ppb based on 933 “trends” sites with complete data records over the 2000 to 2012 period. The center line in this figure represents the median value across the trends sites, while

⁶ For more details on the data handling procedures used to calculate design values for the existing O₃ NAAQS, see 40 CFR Part 50, Appendix P.

the dashed lines represent the 25th and 75th percentiles, and the bottom and top lines represent the 10th and 90th percentiles. Figures 2-3 and 2-4 show maps of the O₃ design values (ppb) at all U.S. monitoring sites for the 2009-2011 and 2010-2012 periods, respectively. The trend figure shows that the annual 4th highest daily maximum values decreased for the vast majority of monitoring sites in the U.S. between 2000 and 2009. The decreasing trend is especially sharp from 2002 to 2004, when EPA implemented the “NO_x SIP Call”, a program designed to reduce summertime emissions of NO_x in the eastern U.S., but has been relatively flat since then.

The trends also show a modest increase in the 4th highest daily maximum values from 2009 to 2012. This is reflected in the design value maps, which show an increase in the number of monitors violating the existing O₃ standard in 2010-2012 relative to 2009-2011. Meteorology played an important role in these short-term trends. O₃ concentrations tend to be higher on days with hot and stagnant conditions and lower on days with cool or wet conditions. According to the National Oceanic and Atmospheric Administration’s National Climatic Data Center (NOAA-NCDC), the summer of 2009 was cooler and wetter than average over most of the eastern U.S., while conversely the summers, of 2010, 2011, and 2012 were all much warmer than average. In particular, the central and eastern U.S. experienced a 2-week period of record-breaking heat in late June and early July of 2012, which contributed to hundreds of violations of the existing O₃ standard. In contrast, the most recent climatological information available from NOAA-NCDC (<http://www.ncdc.noaa.gov/sotc/>) shows that the summer of 2013 was cooler and wetter than average for much of the U.S. Thus, EPA does not expect the recent increasing trend in the 4th highest daily maximum O₃ concentrations to continue in 2013.

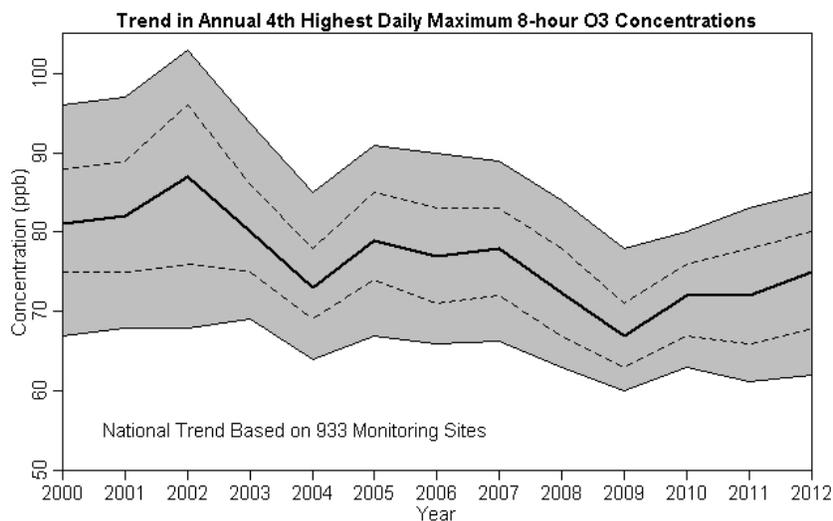


Figure 2-2. Trend in U.S. annual 4th highest daily maximum 8-hour O₃ concentrations in ppb, 2000 to 2012. Solid center line represents the median value across monitoring sites, dashed lines represent 25th and 75th percentile values, and top/bottom lines represent 10th and 90th percentile values.

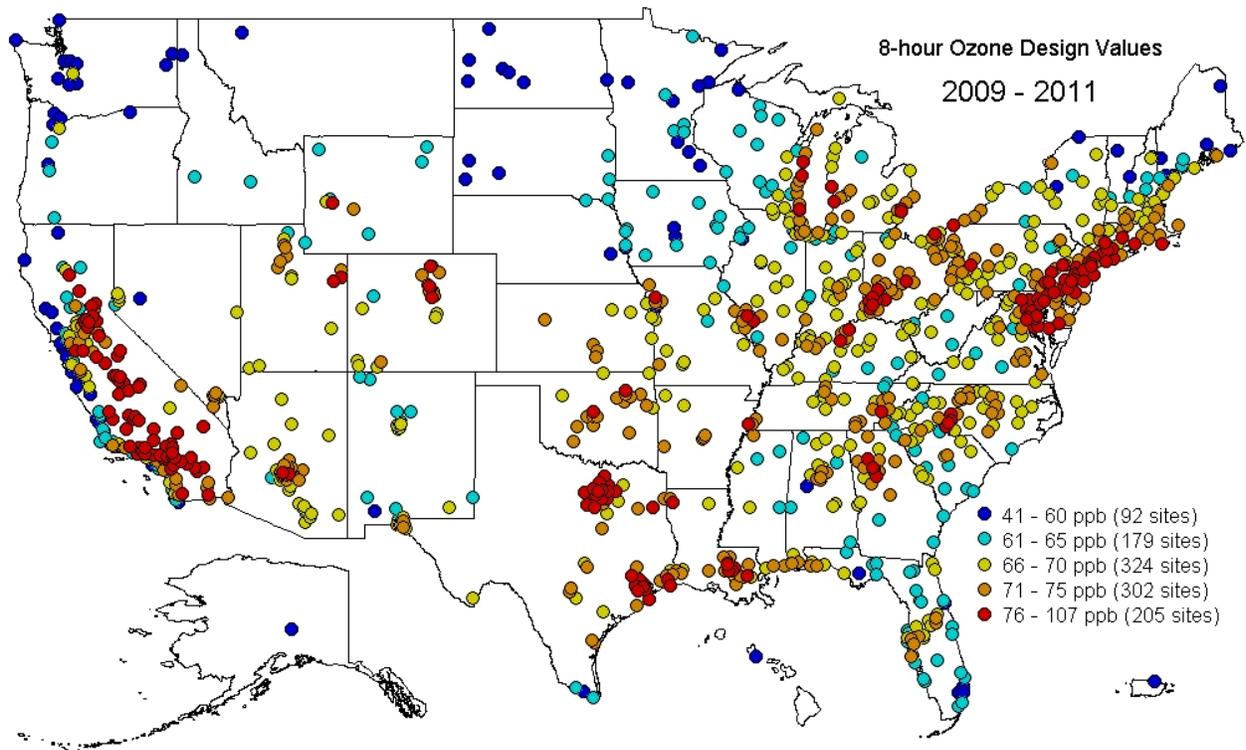


Figure 2-3. Map of 8-hour O₃ design values in ppb for the 2009-2011 period.

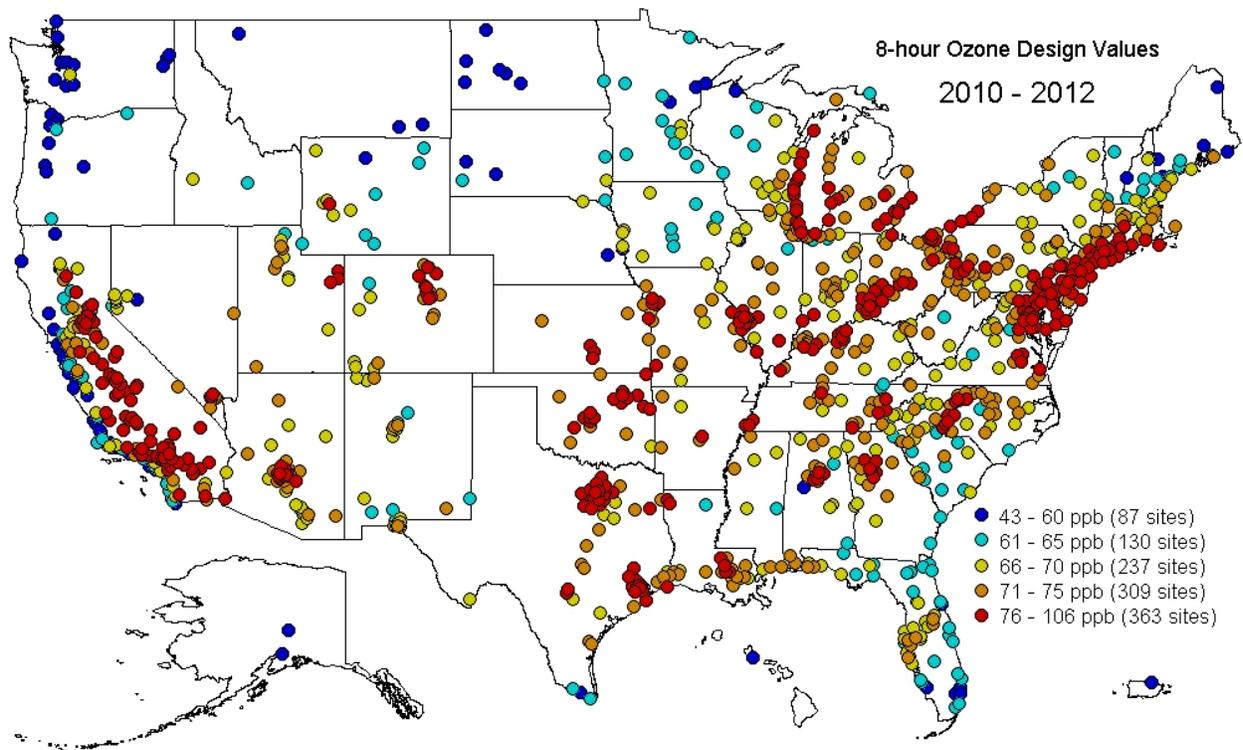


Figure 2-4. Map of 8-hour O₃ design values in ppb for the 2010-2012 period.

In addition, EPA focused our analyses of welfare and ecosystem effects on a W126 O₃ exposure metric in this review. The W126 metric⁷ is a seasonal aggregate of daytime (8:00 AM to 8:00 PM) hourly O₃ concentrations, designed to measure the cumulative effects of O₃ exposure on plant and tree species, with units in parts per million-hours (ppm-hrs). The W126 metric uses a logistic weighting function to place less emphasis on exposure to low hourly O₃ concentrations and more emphasis on exposure to high hourly O₃ concentrations (Lefohn et al, 1988).

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

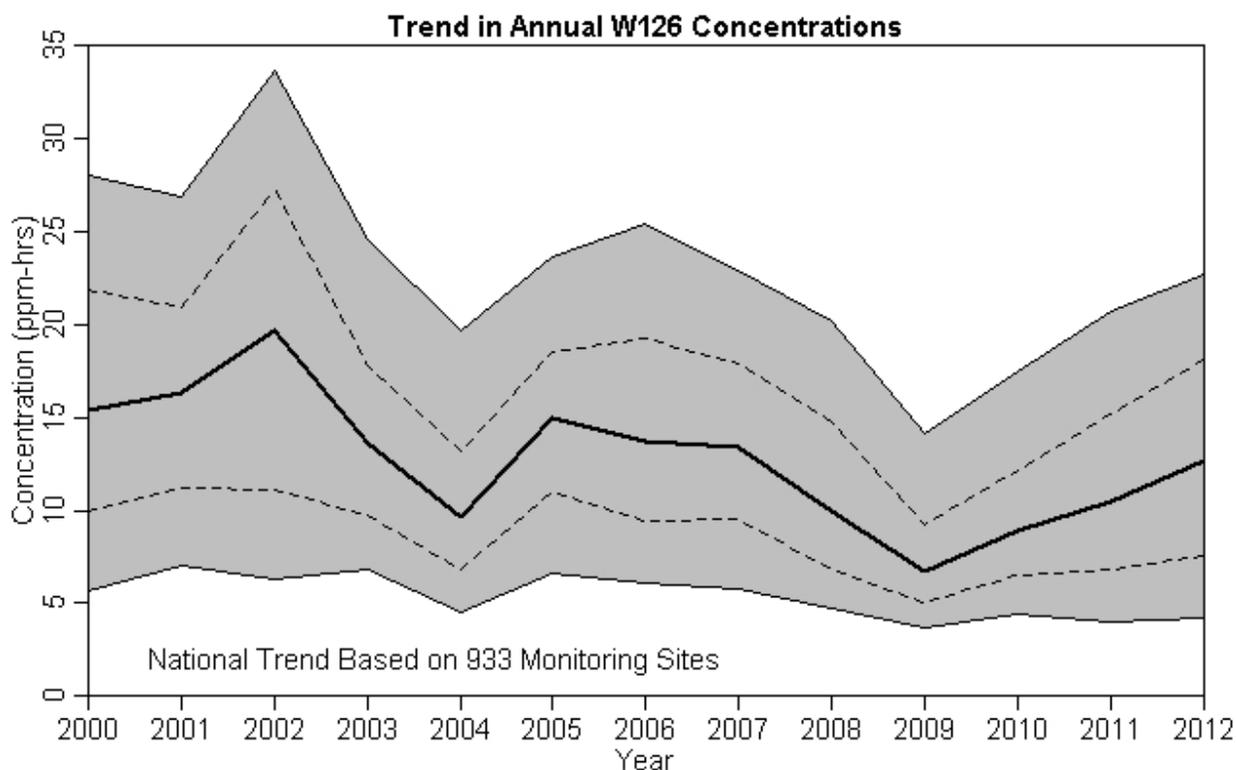


Figure 2-5. Trend in U.S. annual W126 concentrations in ppm-hrs, 2000 to 2012. Solid center line represents the median value across monitoring sites, dashed lines represent 25th and 75th percentile values, and top/bottom lines represent 10th and 90th percentile values.

⁷ Details on the procedure used to calculate the W126 metric are provided in Chapter 4 of the welfare Risk and Exposure Assessment.

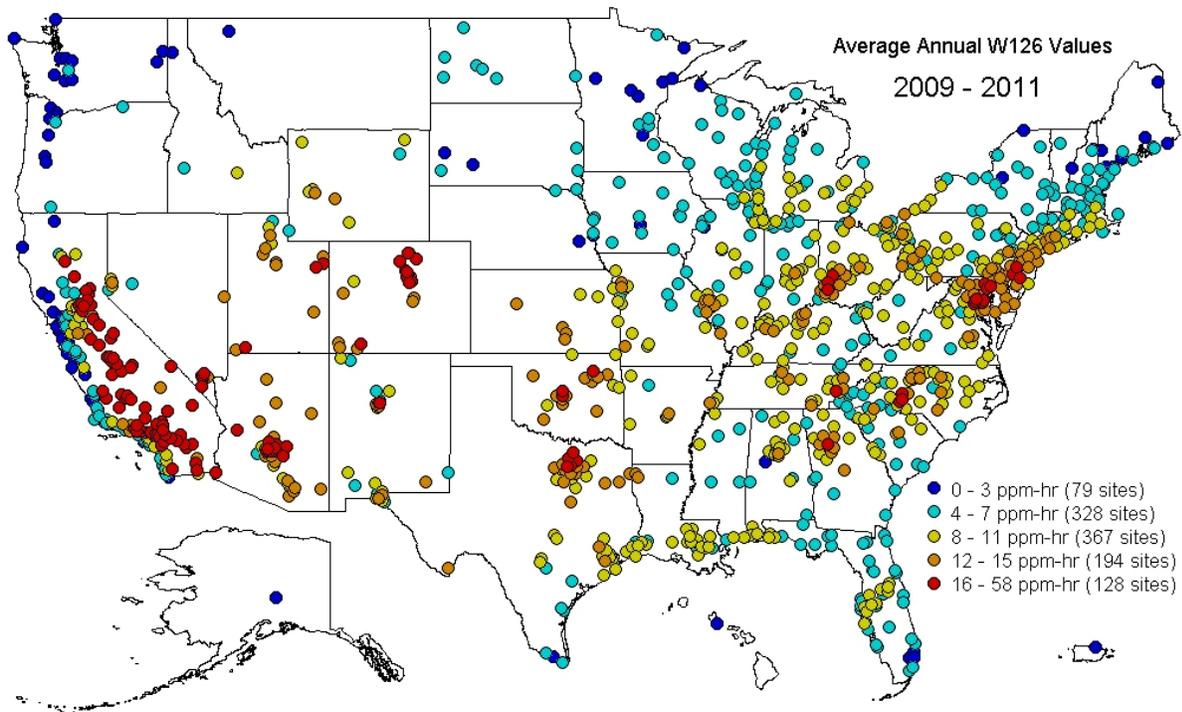


Figure 2-6. Map of 2009-2011 average annual W126 values in ppm-hrs.

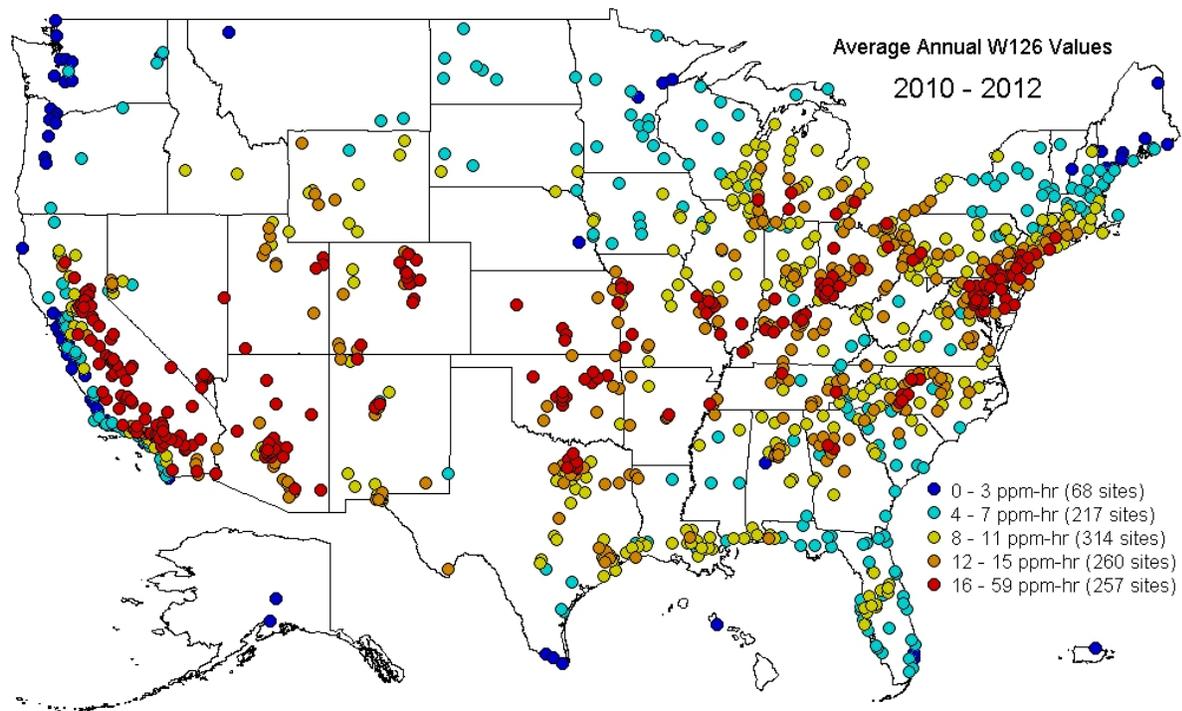


Figure 2-7. Map of 2010-2012 average annual W126 values in ppm-hrs.

Variations in meteorological conditions play an important role in determining ozone concentrations. Ozone is more readily formed on warm, sunny days when the air is stagnant. Conversely, ozone generation is more limited when it is cool, rainy, cloudy, or windy. EPA uses a statistical model to adjust for the variability in seasonal average ozone concentrations due to weather conditions to provide a more accurate assessment of the underlying trend in ozone caused by emissions (Camalier, 2007). Figure 2-8 shows the national trend in the May to September average of the daily maximum 8-hour ozone concentrations from 2000 to 2012 in 112 urban locations. The dotted red line shows the trend in observed ozone concentrations at selected monitoring sites, while the solid blue line shows the underlying ozone trend at those sites after removing the effects of weather. The solid blue lines represent ozone levels anticipated under “typical” weather conditions and serve as a more accurate assessment of the trend in ozone due to changes in precursor emissions.

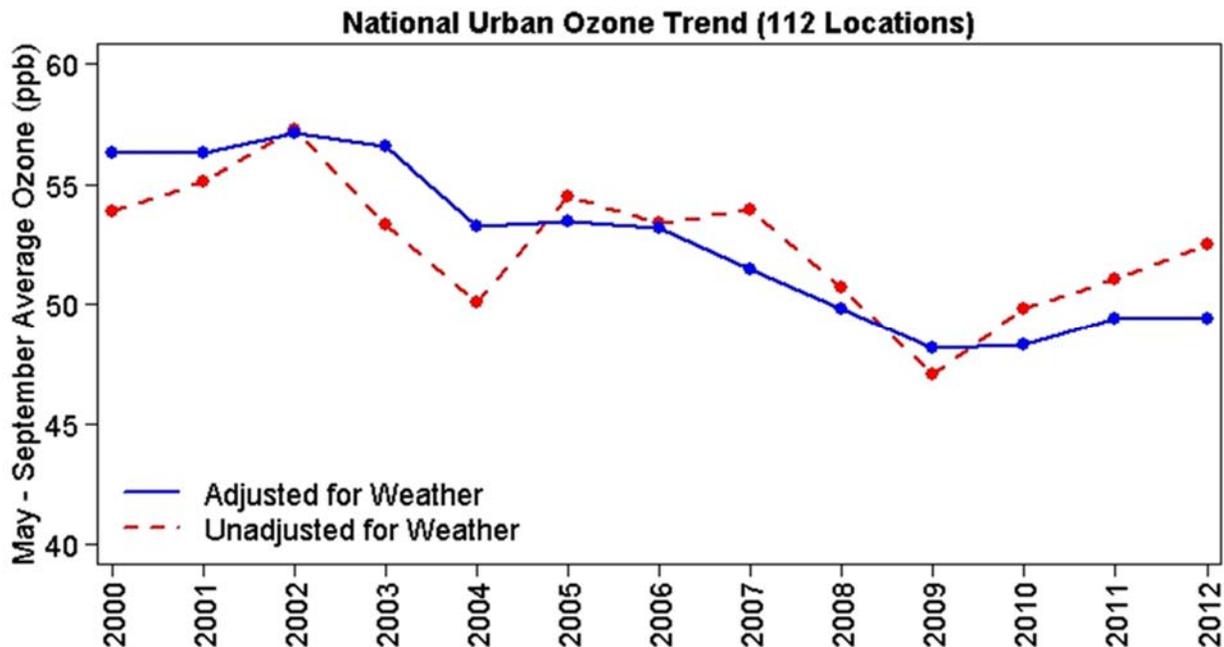


Figure 2-8. Trend in the May to September mean of the daily maximum 8-hour ozone concentrations before (dotted red line) and after (solid blue line) adjusting for year-to-year variability in meteorology⁸.

Figure 2-8 shows that after adjusting for the year-to-year variability in meteorology, the overall trend in seasonal average ozone concentrations is much smoother. The adjusted trend

⁸ More detailed information on these trends is available at: <http://www.epa.gov/airtrends/weather.html>

clearly shows that the NO_x SIP Call program resulted in a sharp decrease in summertime ozone concentrations starting in 2004. The adjusted trend also indicates that ozone levels continued to decrease between 2004 and 2009, and while there is still some evidence of an increasing trend from 2009 to 2012, there is also evidence that much of the recent increase in ozone levels is due to meteorological conditions which were more favorable to ozone formation than normal.

2.2 EMISSIONS AND ATMOSPHERIC CHEMISTRY

O₃ is formed by photochemical reactions of precursor gases and is not directly emitted from specific sources. In the stratosphere, O₃ occurs naturally and provides protection against harmful solar ultraviolet radiation. In the troposphere, near ground level, O₃ forms through atmospheric reactions involving two main classes of precursor pollutants: volatile organic compounds (VOCs) and nitrogen oxides (NO_x). Carbon monoxide (CO) and methane (CH₄) are also important for O₃ formation over longer time periods (US EPA, 2013, section 3.2.2).

Emissions of O₃ precursor compounds can be divided into anthropogenic and natural source categories, with natural sources further divided into biogenic emissions (from vegetation, microbes, and animals) and abiotic emissions (from biomass burning, lightning, and geogenic sources). Anthropogenic sources, including mobile sources and power plants, account for the majority of NO_x and CO emissions. Anthropogenic sources are also important for VOC emissions, though in some locations and at certain times of the year (e.g., southern states during summer) the majority of VOC emissions come from vegetation (US EPA, 2013, section 3.2.1). In practice, the distinction between natural and anthropogenic sources is often unclear, as human activities directly or indirectly affect emissions from what would have been considered natural sources during the preindustrial era. Thus, emissions from plants, animals, and wildfires could be considered either natural or anthropogenic, depending on whether emissions result from agricultural practices, forest management practices, lightning strikes, or other types of events (US EPA, 2013, sections 3.2 and 3.7.1).

Rather than varying directly with emissions of its precursors, O₃ changes in a nonlinear fashion with the concentrations of its precursors. NO_x emissions lead to both the formation and destruction of O₃, depending on the local quantities of NO_x, VOC, radicals, and sunlight. In areas dominated by fresh emissions of NO_x, radicals are removed, which lowers the O₃ formation rate. In addition, the scavenging of O₃ by reaction with NO is called “titration” and is often found in downtown metropolitan areas, especially near busy streets and roads, as well as in power plant plumes. This short-lived titration results in localized areas in which O₃ concentrations are suppressed compared to surrounding areas, but which contain NO₂ that contributes to subsequent O₃ formation further downwind. Consequently, O₃ response to reductions in NO_x emissions is complex and may include O₃ decreases at some times and

locations and increases of O₃ at other times and locations. In areas with relatively low NO_x concentrations, such as those found in remote continental areas and rural and suburban areas downwind of urban centers, O₃ production typically varies directly with NO_x concentrations (e.g. decreases with decreasing NO_x emissions). The NO_x titration effect is most pronounced in urban core areas which have higher volume of mobile source NO_x emissions from vehicles than do the surrounding areas. It should be noted that such locations, which are heavily NO_x saturated (or radical limited), tend to have much lower observed O₃ concentrations than downwind areas. As a general rule, as NO_x emissions reductions occur, one can expect lower O₃ values to increase while the higher ozone values would be expected to decrease. NO_x reductions are expected to result in a compressed O₃ distribution, relative to current conditions.

The formation of O₃ from precursor emissions is also affected by meteorological parameters such as the intensity of sunlight and atmospheric mixing. Major episodes of high ground-level O₃ concentrations in the eastern United States are associated with slow-moving high pressure systems. High pressure systems during the warmer seasons are associated with the sinking of air, resulting in warm, generally cloudless skies, with light winds. The sinking of air results in the development of stable conditions near the surface which inhibit or reduce the vertical mixing of O₃ precursors. The combination of inhibited vertical mixing and light winds minimizes the dispersal of pollutants, allowing their concentrations to build up. In addition, in some parts of the United States (e.g., in Los Angeles), mountain barriers limit mixing and result in a higher frequency and duration of days with elevated O₃ concentrations. Photochemical activity involving precursors is enhanced during warmer seasons because of the greater availability of sunlight and higher temperatures (US EPA, 2013, section 3.2).

O₃ concentrations in a region are affected both by local formation and by transport of O₃ and its precursors from upwind areas. O₃ transport occurs on many spatial scales including local transport between cities, regional transport over large regions of the U.S. and international/long-range transport. In addition, O₃ can be transferred into the troposphere from the stratosphere, which is rich in O₃, through stratosphere-troposphere exchange (STE). These intrusions usually occur behind cold fronts, bringing stratospheric air with them and typically affect O₃ concentrations in higher elevation areas (e.g. > 1500 m) more than areas at lower elevations (U.S. EPA, 2012, section 3.4.1.1). The role of long-range transport of ozone and other elements of ozone background is discussed in more detail in Section 2.4.

2.3 AIR QUALITY CONCENTRATIONS

Because O₃ is a secondary pollutant formed in the atmosphere from precursor emissions, concentrations are generally more regionally homogeneous than concentrations of primary pollutants emitted directly from stationary and mobile sources (US EPA, 2013, section 3.6.2.1).

However, variation in local emissions characteristics, meteorological conditions, and topography can result in daily and seasonal temporal variability in ambient O₃ concentrations, as well as local and national-scale spatial variability.

Temporal variation in ambient O₃ concentrations results largely from daily and seasonal patterns in sunlight, precursor emissions, atmospheric stability, wind direction, and temperature (US EPA, 2013, section 3.7.5). On average, ambient O₃ concentrations follow well-recognized daily and seasonal patterns, particularly in urban areas. Specifically, daily maximum 1-hr O₃ concentrations in urban areas tend to occur in mid-afternoon, with more pronounced peaks in the warm months of the O₃ season than in the colder months (US EPA, 2013, Figures 3-54, 3-156 to 3-157). Rural sites also follow this general pattern, though it is less pronounced in colder months (US EPA, 2013, Figure 3-55). With regard to day-to-day variability, median maximum daily 8-hour average (MDA8) O₃ concentrations in U.S. cities in 2007-2009 were approximately 47 ppb, with typical ranges between 35 to 60 ppb and the highest MDA8 concentrations above 100 ppb in several U.S. cities (as noted further below).

In addition to temporal variability, there is considerable spatial variability in ambient O₃ concentrations within cities and across different cities in the United States. With regard to spatial variability within a city, local emissions characteristics, geography, and topography can have important impacts. For example, as noted above, fresh NO emissions from, for example, motor vehicles titrate O₃ present in the urban background air, resulting in an O₃ gradient around roadways with O₃ concentrations increasing as distance from the road increases (US EPA, 2013, section 3.6.2.1). In comparing urban areas, the ISA notes that measured O₃ concentrations are relatively uniform and well-correlated within some cities (e.g., Atlanta) while they are more variable in others (e.g., Los Angeles) (US EPA, 2013, section 3.6.2.1 and Figures 3-28 to 3-36).

With regard to variability across cities, when the ISA evaluated the distributions of 8-hour O₃ concentrations for the years 2007 to 2009 in 20 cities, the highest concentrations were reported in Los Angeles, with high concentrations also reported in several eastern and southern cities. The maximum recorded MDA8 was 137 ppb in Los Angeles, and was near or above 120 ppb in Atlanta, Baltimore, Dallas, New York City, Philadelphia, and St. Louis (US EPA, 2013, Table 3-10). The pattern was similar for the 98th percentile of the distribution of MDA8 concentrations⁹, with Los Angeles recording the highest 98th percentile concentration (91 ppb) and many eastern and southern cities reporting 98th percentile concentrations near or above 75 ppb. In contrast, somewhat lower 98th percentile O₃ concentrations were recorded in cities in the western United States outside of California (US EPA, 2013, Table 3-10).

⁹ Table 3-10 in the ISA analyzes the warm season. Therefore, the 98th percentile values would be an approximation of the 4th highest value.

Rural sites can be affected by transport of O₃ or O₃ precursors from upwind urban areas and by local anthropogenic sources such as motor vehicles, power generation, biomass combustion, or oil and gas operations (US EPA, 2013, section 3.6.2.2). In addition, O₃ tends to persist longer in rural than in urban areas due to lower rates of chemical scavenging in non-urban environments. At higher elevations, increased O₃ concentrations can also result from stratospheric intrusions (US EPA, 2013, sections 3.4, 3.6.2.2). As a result, O₃ concentrations measured in some rural sites can be higher than those measured in nearby urban areas (US EPA, 2013, section 3.6.2.2), and the ISA concludes that cumulative exposures for humans and vegetation in rural areas can be substantial, often higher than cumulative exposures in urban areas (US EPA, 2013, section 3.7.5).

2.4 BACKGROUND O₃

One of the aspects of ozone that is unusual relative to the other pollutants with National Ambient Air Quality Standards (NAAQS) is that, periodically, in some locations, an appreciable fraction of the observed ozone results from sources or processes other than local and domestic regional anthropogenic emissions of ozone precursors (Fiore *et al.*, 2002). Any ozone formed by processes other than the chemical conversion of local or regional ozone precursor emissions is generically referred to as “background” ozone. Background O₃ can originate from natural sources of O₃ and O₃ precursors, as well as from manmade international emissions of O₃ precursors. Natural sources of O₃ precursor emissions such as wildfires, lightning, and vegetation can lead to O₃ formation by chemical reactions with other natural sources. Another important component of background is O₃ that is naturally formed in the stratosphere through interactions of ultraviolet light with molecular oxygen. Stratospheric O₃ can mix down to the surface at high concentrations in discrete events called intrusions, especially at higher-altitude locations. The manmade portion of the background includes any O₃ formed due to anthropogenic sources of O₃ precursors emitted far away from the local area (e.g., international emissions). Finally, both biogenic and international anthropogenic emissions of methane, which can be chemically converted to O₃ over relatively long time scales, can also contribute to global background O₃ levels. Away from the surface, ozone can have an atmospheric lifetime on the order of weeks. As a result, background ozone can be transported long distances in the upper troposphere and, when meteorological conditions are favorable, be available to mix down to the surface and add to the ozone loading from non-background sources.

As indicated in the first draft policy assessment (US EPA, 2012, sections 1.3.4 and 3), EPA has updated several aspects of our methodology for estimating the change in health risk and exposure that would result from a revision to the O₃ NAAQS. First, risk estimates are now based on total O₃ concentrations, as opposed to previous reviews which only considered risk above

background levels. Second, EPA is now using air quality modeling to estimate the spatial patterns of O₃ that would result from attaining various levels of the NAAQS, as opposed to a quadratic rollback approach that required the estimation of a background “floor” beyond which the rollback would not take place. Both of these revisions have had the indirect effect of reducing the need for estimates of background O₃ levels as part of the O₃ risk and exposure assessment (REA). Regardless, EPA expects that a well-founded understanding of the fractional contribution of background sources and processes to surface O₃ levels will be valuable. Accordingly, in this section, we briefly summarize existing results on background O₃ from the ISA (US EPA, 2013, section 3.4) as supplemented by additional EPA modeling recently conducted for a 2007 base year. The summary will focus on national estimates of the:

- seasonal mean background O₃ values for three specific definitions of background O₃,
- relative proportion of background O₃ to total O₃ for the same three definitions from a seasonal mean perspective,
- distributions of background O₃ within a seasonal mean,
- ratio of background O₃ to total modeled ozone in the 12 REA case study areas,
- relative proportion of background O₃ concentrations to total W126 ozone, and
- relative contribution of different components of background to total background O₃.

The definition of background O₃ can vary depending upon context, but it generally refers to O₃ that is formed by sources or processes that cannot be influenced by actions within the jurisdiction of concern. In the first draft policy assessment document (US EPA, 2012), EPA identified three specific definitions of background O₃: natural background (NB), North American background (NAB), and United States background (USB). Natural background is the narrowest definition of background, and it is defined as the O₃ that would exist in the absence of any manmade O₃ precursor emissions. The other two definitions of background are based on a presumption that the U.S. has little influence over anthropogenic emissions outside either our continental or domestic borders. North American background is defined as that O₃ that would exist in the absence of any manmade O₃ precursor emissions from North America. U.S. background is defined as that O₃ that would exist in the absence of any manmade emissions inside the United States.

Each of these three definitions of background O₃ requires photochemical modeling simulations to estimate what the residual O₃ concentrations would be were the various anthropogenic emissions to be removed. EPA exclusively uses modeling estimates to characterize background, as opposed to using observed concentrations from a remote site, because even the most remote monitors within the U.S. can be periodically affected by U.S.

anthropogenic emissions. In most situations, without special monitoring it is impossible to determine how much of the ozone measured by a monitor originated from background sources. Prior to using the new 2007-based model simulations to estimate background O₃ levels over the U.S., EPA confirmed that this modeling was able to reproduce historical O₃ levels and that there was limited correlation between model errors and the background estimates. This evaluation is described more fully in the appendix (Appendix A) to this chapter.

Previous modeling studies have estimated what background levels would be in the absence of certain sets of emissions by simply assessing the remaining O₃ in a simulation in which certain emissions were removed (Zhang et al. (2011), Emery et al. (2012)). This basic approach is often referred to as “zero-out” modeling or “emissions perturbation” modeling. While the zero-out approach has traditionally been used to estimate natural background, North American background, and U.S. background, the methodology has an acknowledged limitation. It cannot answer the question of how much of the existing observed ozone results from background sources or processes.

A separate modeling technique can be used to estimate the contribution of background ozone and other contributing source terms to total O₃ within a model. This approach, referred to as “source apportionment” modeling, has been described and evaluated in the peer-reviewed literature (Dunker et al., 2002; Kemball-Cook et al., 2009). Source apportionment modeling has frequently been used in other regulatory settings to estimate the “contribution” to ozone of certain sets of emissions (EPA 2005, EPA 2011). The source apportionment technique provides a means of estimating the contributions of each user-identified source category to ozone formation in a single model simulation. This is achieved by using multiple tracer species to track the fate of ozone precursor emissions (VOC and NO_x) and the ozone formation resulting from these emissions. The methodology is designed so that all ozone and precursor concentrations are tracked and apportioned to the selected source categories at all times without perturbing the inherent chemistry. The primary limitation of the source apportionment modeling is that its estimations of background are explicitly linked to the emissions scenarios modeled and would change with different emissions scenarios.

EPA recently completed updated zero-out and source apportionment modeling for a 2007 base year to supplement the characterization of background O₃ that was provided in the ISA. Both of these approaches have value in assessing the potential impacts of background O₃, but they are used separately as described in Table 2-1.

Estimation Methodology	Question addressed	Background Quantities	Strengths and Limitations
Zero-out	How much ozone would remain if controllable emissions were completely removed?	NB / NAB / USB	<p><u>Strength:</u> The approach is simple to implement and provides an estimate of the lowest O₃ levels that can be attained by eliminating all U.S. anthropogenic emissions.</p> <p><u>Limitation:</u> Estimates are based on a counterfactual, represents a quantity never to occur in real atmosphere. Additionally, sensitivity approaches can be unreliable for evaluating mass contributions to O₃ production because of non-linearity in the chemistry.</p>
Source Apportionment	How much of the current ozone can be attributed to sources other than U.S. anthropogenic sources?	Apportionment-based USB	<p><u>Strength:</u> Provides a direct estimate of the amount of O₃ contributed by each source category while avoiding artifacts caused by non-linearity in the chemistry.</p> <p><u>Limitation:</u> While this approach identifies important sources that contribute to O₃, it does not predict quantitatively how O₃ will respond to specific emissions reduction scenarios.</p>

Table 2-1 Comparison of the two model methodologies used to characterize background ozone levels.

The key configuration elements of the updated modeling are described below; a more detailed description of the modeling is provided in Appendix A. The zero-out modeling was based on a model configuration that nested a regional-scale air quality model (CMAQ at 12 km horizontal grid resolution) within a global scale air quality model (GEOS-Chem at 2.0 x 2.5

degree horizontal grid resolution). The lateral boundary conditions from the global model were used as inputs for the regional simulation. Four scenarios were modeled:

- a 2007 base case simulation which was also the basis of the air quality modeling performed for the 2nd draft ozone REA and is described in more detail in Appendix 4b of EPA (2013b)
- a natural background run with anthropogenic ozone precursor emissions¹⁰ removed in both the global and regional models¹¹
- a North American background run with anthropogenic ozone precursor emissions removed across North America in both the global and regional model simulations
- a U.S. background run with anthropogenic ozone precursor emissions removed over the U.S. in both the global and regional model simulations

The source apportionment modeling was also based on a regional scale air quality model (CAMx at 12 km horizontal grid resolution) that used the same lateral boundary conditions from the 2007 base global modeling scenario. EPA used the Anthropogenic Precursor Culpability Assessment (APCA) tool in this analysis. The APCA tool attributes ozone production to manmade sources whenever ozone is determined to result from a combination of anthropogenic and biogenic emissions (Environ, 2011). The APCA methodology calculates natural ozone as the production resulting from the interaction of biogenic VOC with biogenic NO_x emissions. Eleven separate source categories were tracked in the EPA source apportionment analysis, including five boundary condition terms and six in-domain sectors:

- Boundary condition terms:¹²
 - Northern edge
 - Eastern edge
 - Southern edge
 - Western edge
 - Top boundary

¹⁰ In the global model, only emissions from natural sources were used (i.e., VOC, NO_x, CO) and methane was reset to pre-industrial levels (700 ppb) to reflect natural contributions. In the regional modeling, the methane levels were left unchanged.

¹¹ Note that methane is not modeled as an explicit species in CMAQ but instead is treated as having a constant concentration. Therefore, although methane was reduced to pre-industrial levels in the global GEOS-Chem run used to create boundary conditions, methane was assumed to be equal to modern-day levels in the CMAQ model runs. Since methane reactions occur on relatively long timescales, this discrepancy is not expected to have a large impact on modeled background ozone levels.

¹² It should be noted that although boundary conditions are treated as part of apportionment-based USB for this analysis, in some cases they may be influenced by US anthropogenic emissions that are advected out of the model domain and recirculated back into the U.S. This is not expected to make a substantial impact on results.

- In-domain sectors:
 - U.S. anthropogenic emissions
 - Point sources located within the Gulf of Mexico
 - Category 3 marine vessels outside State boundaries
 - Climatologically-averaged wildfire emissions
 - Biogenic emissions
 - Canada/Mexico emissions (only those sources within the domain)

2.4.1 Seasonal Mean Background O₃ in the U.S.

The ISA (US EPA 2013, section 3.4) previously established that background concentrations vary spatially and temporally and that simulated mean background concentrations are highest at high-elevation sites within the western U.S. Background levels typically are greatest over the U.S. in the spring and early summer. The updated EPA modeling focused on the months from April to October. Figure 2-9 displays the spatial patterns of seasonal mean natural background O₃ as estimated by a 2007 zero-out scenario. Seasonal means are computed over those seven months. This figure shows the average daily maximum 8-hour O₃ concentration (MDA8) that would exist in the absence of any anthropogenic O₃ precursor emissions at monitor locations. As shown, seasonal mean NB levels range from approximately 15-35 ppb (i.e., +/- 1 standard deviation) with the highest values at higher-elevation sites in the western U.S. The median value over these locations is 24.2 ppb, and more than 50 percent of the locations have natural background levels of 20-25 ppb. The highest modeled estimate of seasonal average, natural background, MDA8 O₃ is 34.3 ppb at the high-elevation CASTNET site (Gothic) in Gunnison County, CO. Natural background levels are higher at these high-elevation locations primarily because natural stratospheric O₃ impacts and international transport impacts increase with altitude (where O₃ lifetimes are longer).

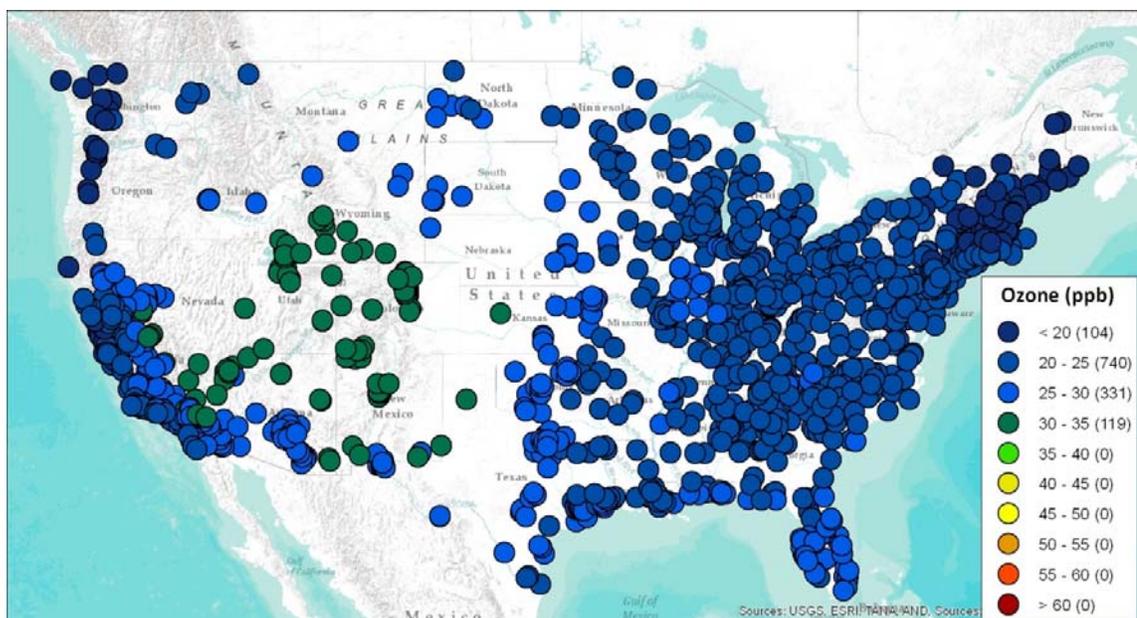


Figure 2-9. Map of 2007 CMAQ-estimated seasonal mean natural background O₃ levels (ppb) from zero-out modeling.

Figures 2-10 and 2-11 show the same information for the NAB and USB scenarios. In these model runs, all anthropogenic O₃ precursor emissions were removed from the U.S., Canada, and Mexico portions of the modeling domain (NAB scenario) and then only from the U.S. (USB scenario). The figures show that there is not a large difference between the NAB and USB scenarios. Seasonal mean NAB and USB O₃ levels range from 25-50 ppb, with the most frequent values estimated in the 30-35 ppb bin. The median seasonal mean background levels are 31.5 and 32.7 ppb (NAB and USB, respectively). Again, the highest levels of seasonal mean background are predicted over the intermountain western U.S. Locations with NAB and USB concentrations greater than 40 ppb are confined to Colorado, Nevada, Utah, Wyoming, northern Arizona, eastern California, and parts of New Mexico. The 2007 EPA modeling suggests that seasonal mean USB concentrations are on average 1-3 ppb higher than NAB background. These results were similar to those reported by Wang et al. (2009). From a seasonal mean perspective, background levels are typically well-below the NAAQS thresholds.

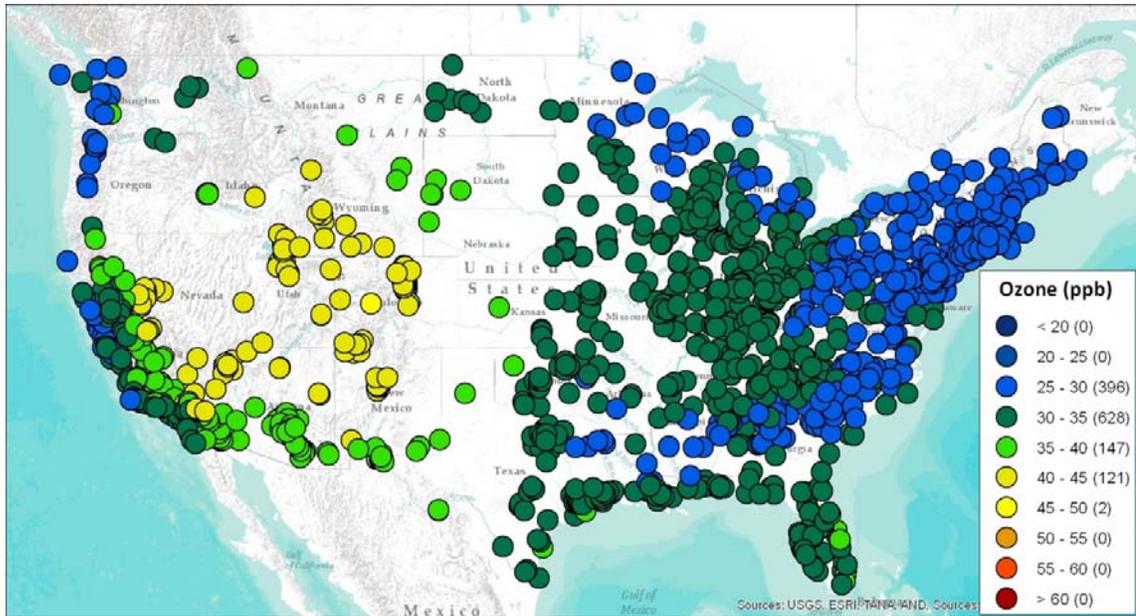


Figure 2-10. Map of 2007 CMAQ-estimated seasonal mean North American background O₃ levels (ppb) from zero-out modeling.

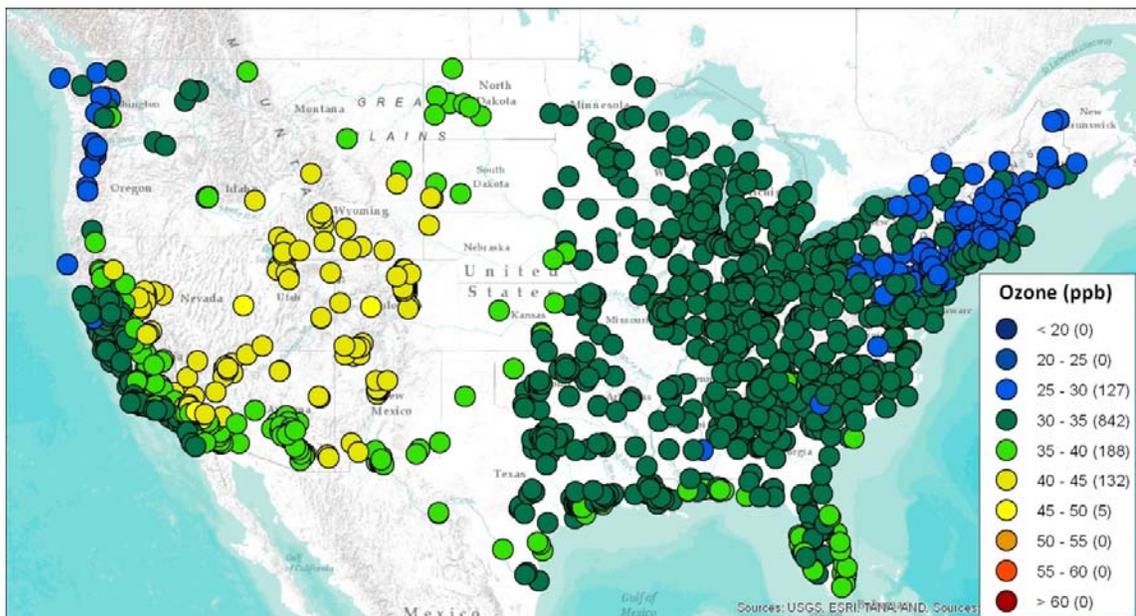


Figure 2-11. Map of 2007 CMAQ-estimated seasonal mean United States background O₃ levels (ppb) from zero-out modeling.

2.4.2 Seasonal Mean Background O₃ in the U.S. as a Proportion of Total O₃

Another informative way to assess the importance of background as part of seasonal mean O₃ levels across the U.S. is to consider the ratios of NB, NAB, and USB to total modeled O₃ at each monitoring location. Considering the proportional impact of background allows for an

initial assessment of the relative importance of background and non-background sources. Because ozone chemistry is non-linear, one should not assume that individual perturbations (e.g., zero out runs) are additive in all locations. Figures 2-12 and 2-13 show the ratio of U.S. anthropogenic sources to total O₃ using the metric of the seasonal mean MDA8 O₃ concentrations as estimated by both the zero-out and source apportionment modeling methodologies. Recall that the terms NB, NAB, and USB are explicitly linked to the zero-out modeling approach. For comparison sake, in Figure 2-13 we are extending the definition of USB to also include the source apportionment model estimates of the O₃ that is *attributable to sources other than U.S. anthropogenic emissions*. To preserve the original definition of USB, this second term will be hereafter referred to as “apportionment-based USB”. As noted earlier, the advantage of the source apportionment modeling is that all of the modeled O₃ is attributed to various source terms without perturbing the inherent chemistry. Thus, this approach is not affected by the confounding occurrences of background O₃ values exceeding the base O₃ values as can happen in the zero-out modeling (i.e., background proportions > 100%). Consequently, one would expect the fractional background levels to be lower in the source apportionment methodology as a result of removing this artifact.

When averaged over all sites, O₃ from sources other than U.S. anthropogenic emissions is estimated to comprise 66 (zero-out) and 59 (source apportionment) percent of the total seasonal O₃ mean. The spatial patterns of USB and apportionment-based USB are similar across the two modeling exercises. Background O₃ is a relatively larger percentage (e.g., 70-80%) of the total seasonal mean O₃ in locations within the intermountain western U.S. and along the U.S. border. In locations where O₃ levels are generally higher, like California and the eastern U.S., the seasonal mean background fractions are relatively smaller (e.g., 40-60%). The additional 2007 modeling confirms that background ozone, while generally not approaching levels of the ozone standard, can comprise a considerable fraction of total seasonal mean ozone across the U.S.

2.4.3 Daily Distributions of Background O₃ within the Seasonal Mean

As a first-order understanding, it is valuable to be able to characterize seasonal mean levels of background O₃. However, it is well established that background levels can vary substantially from day-to-day within the seasonal mean. From an implementation perspective, the values of background O₃ on possible exceedance days are a more meaningful consideration. The first draft policy assessment (US EPA, 2012) considered this issue in detail, via summaries of the existing 2006 zero-out modeling (Henderson et al., 2012), and concluded that “results suggest that background concentrations on the days with the highest total O₃ concentrations are not dramatically higher than typical seasonal average background concentrations.” Based on this finding, EPA determined that “anthropogenic sources within the U.S. are largely responsible for

4th highest 8-hour daily maximum O₃ concentrations.” The recent EPA modeling using a 2007 base year and the two distinct modeling methodologies described above, supports the finding from the previous 2006-based modeling analyses. That is, the highest modeled O₃ site-days tend to have background O₃ levels similar to mid-range O₃ days. Figure 2-14 and 2-15 show the distribution of daily MDA8 apportionment-based USB levels (absolute magnitudes and relative fractions, respectively) from the source apportionment simulation¹³. Again, the 2007 modeling shows that the days with highest O₃ 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 O₃ that has background origins is smaller on high O₃ days (e.g., days > 60 ppb) than on the more common lower O₃ days that tend to drive seasonal means. This helps put the results from section 2.4.2 into better context. For example, for site-days in which base O₃ is between 70-75 ppb, the source apportionment modeling estimates that approximately 37 percent of those O₃ levels originate from sources other than U.S. anthropogenic emissions (i.e., apportionment-based USB). Figure 2-15 also indicates that there are cases in which the model predicts much larger background proportions, as shown by the upper outliers in the figure. These infrequent episodes usually occur in relation to a specific event, and occur more often in specific geographical locations, such as at high elevations or wildfire prone areas during the local dry season.

It should be noted here that EPA has policies for treatment of air quality monitoring data affected by these types of events. EPA’s exceptional events policy allows exclusion of certain air quality monitoring data from regulatory determinations if a State adequately demonstrates that an exceptional event has caused the exceedance or violation of a NAAQS. In addition, Section 179B of the CAA also provides for treatment of air quality data from international transport when an exceedance or violation of a NAAQS would not have occurred but for the emissions emanating from outside of the United States.

¹³ Similar plots from the zero-out modeling for natural background, North American background, and U.S. background are provided in Appendix A.

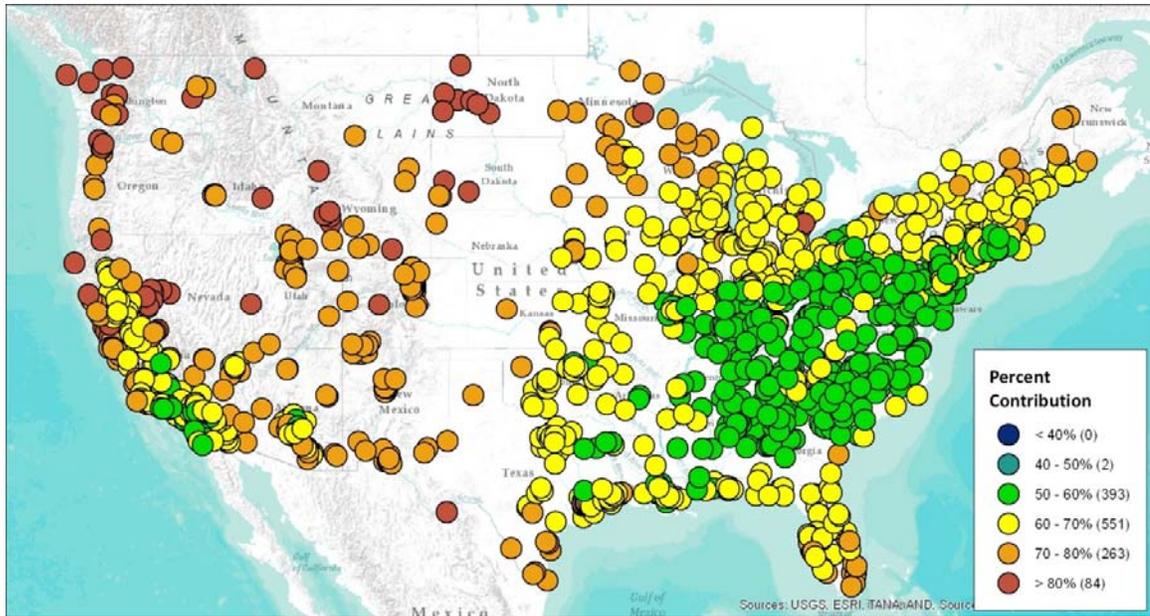


Figure 2-12. Map of site-specific ratios of U.S. background to total seasonal mean O₃ based on 2007 CMAQ zero-out modeling.

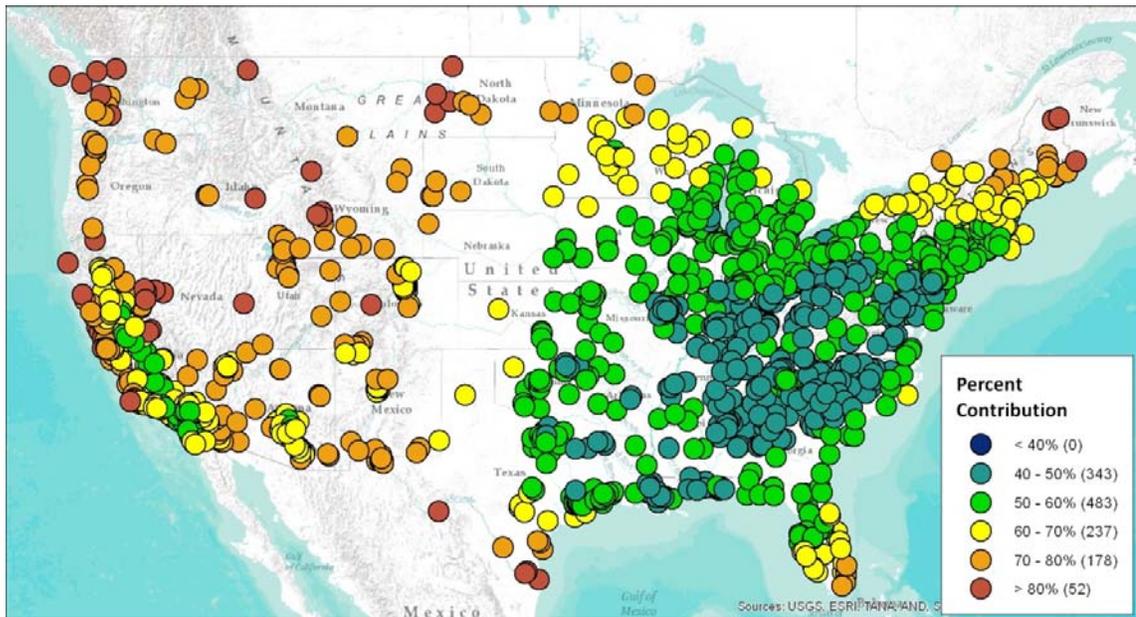


Figure 2-13. Map of site-specific ratios of apportionment-based U.S. background to seasonal mean O₃ based on 2007 CAMx source apportionment modeling.

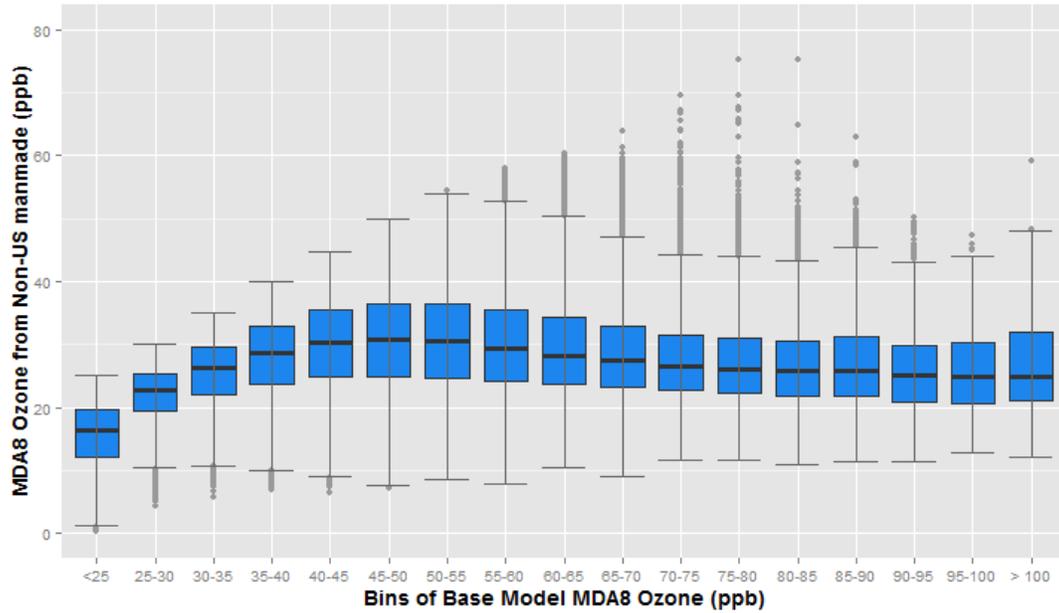


Figure 2-14. Distributions of absolute estimates of apportionment-based U.S. Background (all site-days), binned by modeled MDA8 from the 2007 source apportionment simulation.

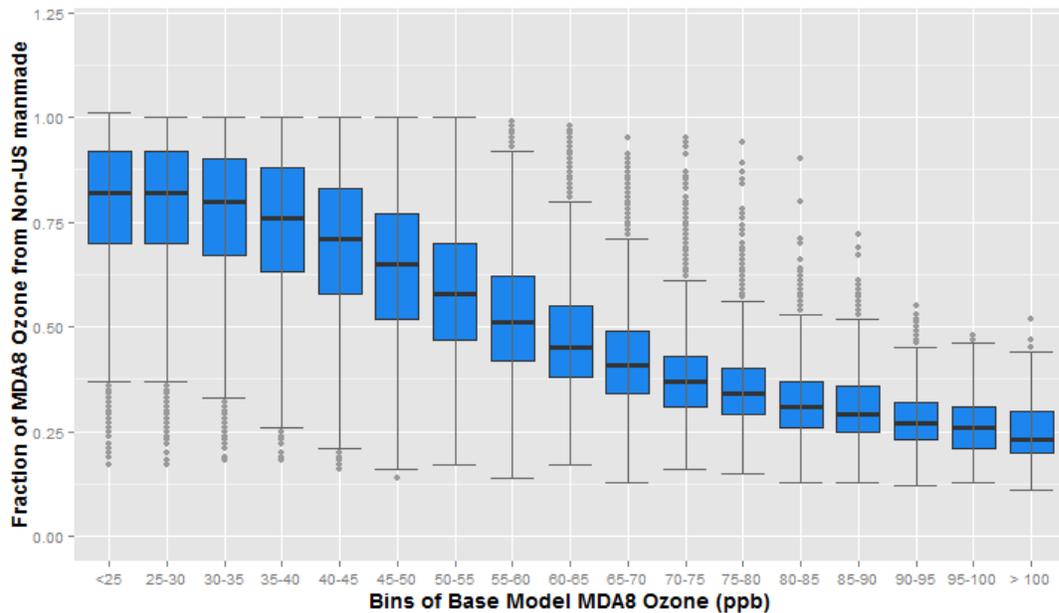


Figure 2-15. Distributions of the relative proportion of apportionment-based U.S. Background to total O₃ (all site-days), binned by modeled MDA8 from the 2007 source apportionment simulation.

2.4.4 Proportion of Background O₃ in 12 Urban Case Study Areas

This section presents estimates of the overall fraction of O₃ that is estimated to result from background sources or processes in each of the 12 urban case study areas considered in the epidemiological-based risk assessment of the REA (US EPA 2014, Chapter 7). The results are based on the recent EPA 2007 source apportionment modeling. Table 2-1 summarizes the estimated ratios of sources other than U.S. anthropogenic emissions (i.e., apportionment-based USB) to total seasonal mean MDA8 O₃ in each of the 12 urban case study areas. The table shows that the fractional contributions from sources other than anthropogenic emissions within the U.S. can range from 43 to 66 percent across these 12 urban areas. These fractions are consistent with the national ratios summarized in section 2.4.2, although the fractions of background are generally smaller at urban sites than at rural sites.

As shown in section 2.4.3, the background-to-total ratios are smaller on days with high modeled O₃ (i.e., days that may exceed the level of the NAAQS). Table 2-2 provides the fractional contributions from apportionment-based USB, only considering days in which base model MDA8 O₃ was greater than 60 ppb. As expected, the fractional background contributions are smaller, ranging from 31 to 55 percent.

Rather than taking the fractions of the seasonal means (as in Table 2-1), Table 2-3 displays the mean and median daily MDA8 background fractions. These metrics may be more appropriate for application to health studies. The fractional contributions to background calculated via this approach are very similar to the Table 2-1 calculations. For completeness, we also provide USB ratios based on the zero-out modeling for the 12 cities (see Table 2-4). The results are similar to the source apportionment findings (Table 2-1), though the zero-out technique provides slightly higher background proportions, as expected. It should be noted that all fractional contributions are based on recent conditions from 2007. These ratios would be expected to change as anthropogenic emissions and O₃ levels are lowered.

Based on the source apportionment modeling for these 12 areas, there is evidence that background levels comprise a non-negligible fraction of the total ozone observed within these locations. However, for site-days in which model MDA8 ozone exceeds 60 ppb, ozone formed from U.S. anthropogenic emissions comprise a larger fraction of the total ozone in 11 of the 12 areas (all but Denver). The major metropolitan areas in the eastern U.S. (e.g., Atlanta, New York City, Philadelphia) are less influenced by background sources than a higher-elevation, western U.S., location like Denver. Even in Denver, though, U.S. anthropogenic emissions have a large influence on total ozone (45 percent).

Table 2-2 Seasonal mean MDA8 O₃ (ppb), seasonal mean apportionment-based USB contribution (ppb), and fractional apportionment-based USB contribution to total O₃ (all site-days) in the 12 REA urban case study areas (%).

All days, CAMx	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	59.3	54.4	43.0	48.9	47.3	39.1	48.5	51.1	45.4	48.7	46.4	49.8
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.3	25.9	26.2	25.7	31.3	23.3	27.0	29.1	24.5	24.2	29.7	24.3
Fractional contribution from background	0.43	0.48	0.61	0.53	0.66	0.60	0.56	0.57	0.54	0.50	0.64	0.49

Table 2-3 Seasonal mean MDA8 O₃ (ppb), seasonal mean apportionment-based USB contribution (ppb), and fractional apportionment-based USB contribution to total O₃ (site-days > 60 ppb) in the 12 REA urban study areas (%).

Only days w/ base MDA8 > 60 ppb	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	74.0	75.3	70.7	72.0	67.5	68.9	70.3	74.4	74.1	74.0	68.3	70.0
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.4	23.7	24.4	25.4	37.3	24.4	28.0	31.9	23.5	22.9	32.1	25.4
Fractional contribution from background	0.34	0.31	0.35	0.35	0.55	0.35	0.40	0.43	0.32	0.31	0.47	0.36

Table 2-4 Fractional contribution of apportionment-based USB in the 12 REA urban study areas (%), using the means and medians of daily MDA8 fractions (instead of fractions of seasonal means).

	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Mean of daily MDA8 background fractions	0.46	0.53	0.68	0.58	0.69	0.64	0.59	0.61	0.61	0.56	0.67	0.52
Median of daily MDA8 background fractions	0.43	0.51	0.73	0.54	0.69	0.66	0.59	0.60	0.63	0.54	0.66	0.49

Table 2-5 Seasonal mean MDA8 O₃ (ppb), seasonal mean USB (ppb), and USB/Total ratio (all site-days) in the 12 REA urban case study areas (%).

All days, CMAQ	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	58.6	55.6	45.2	51.8	57.1	43.5	49.4	54.8	47.7	50.5	51.9	52.6
Model MDA8 seasonal mean USB	30.0	29.9	28.5	31.6	42.2	31.7	33.0	33.3	29.1	29.4	34.4	32.0
Ratio of USB/Total MDA8 O ₃	0.51	0.54	0.63	0.61	0.74	0.73	0.67	0.61	0.61	0.58	0.66	0.61

2.4.5 Influence of Background O₃ on W126 levels

EPA also conducted a limited assessment of the impacts of background O₃ sources on the W126 metric. The W126 metric (LeFohn et al., 1988) is a cumulative peak-weighted index designed to estimate longer-term effects of daytime ozone levels on sensitive vegetation and ecosystems. EPA used the 2007 zero-out modeling to assess NB, NAB, and USB influences at

four sample locations: Atlanta GA, Denver CO, Farmington NM, and Riverside CA. Each of the four analyses locations had relatively high observed values of W126 in 2007, as averaged over all sites in the area: Atlanta (25.1 ppm-hrs), Denver (19.6 ppm-hrs), Farmington (20.2 ppm-hrs), and Riverside (36.0 ppm-hrs).

As discussed above, in the current review, EPA is supplementing the counterfactual assessment used in previous reviews (zero out modeling) with analyses that estimate the portion of the existing ozone that is due to background sources (source apportionment). This has important ramifications for assessing the influence of background on W126 concentrations, because of the non-linear weighting function used in the metric, which emphasizes high ozone hours (e.g., periods in which ozone is greater than ~60 ppb). As an example, consider a sample site in the intermountain western U.S. region with very high modeled estimates of U.S. background (e.g., seasonal mean of 45 ppb with some days as high as 65 ppb). Even at this high background location, the USB simulations estimate annual W126 (USB) values that are quite low, on the order of 3 ppm-hrs. Sites in the domain with lower U.S. background levels have even smaller USB W126 values, on the order of the 1 ppm-hrs, which is consistent with values mentioned in past reviews (US EPA, 2007). Using the counterfactual scenarios, background ozone has a relatively small impact on W126 levels across the U.S.

However, because of the non-linear weighting function used in the W126 calculation, the sum of the W126 from the USB scenario and the W126 resulting from U.S. anthropogenic sources will not equal the total W126. In most cases, the sum of those two components will be substantially less than total W126. As a result, EPA believes it is more informative to estimate the fractional influence of background ozone to W126 levels. Using a methodology that is described in more detail in Appendix A, EPA considered the fractional influence of background ozone on annual W126 levels in four locations. The fractional influence methodology utilizes the 2007 zero-out modeling but places higher weights on background fractions on days that are going to contribute most substantially to the yearly W126 value. Figure 2-16 shows the results. Based on the fractional influence methodology, natural background sources are estimated to contribute 29-50% of the total modeled W126 with the highest relative influence in the intermountain western U.S. (i.e., Farmington NM) and the lowest relative influence in the eastern U.S. (i.e., Atlanta). U.S. background is estimated to contribute 37-65% of the total modeled W126. The proportional impacts of background are slightly less for the W126 metric than for seasonal mean MDA8 (discussed in section 2.4.2), because of the sigmoidal weighting function that places more emphasis on higher ozone days when background fractions are generally lower.

The key conclusion from this cursory analysis is that background ozone can comprise a non-negligible portion of current W126 levels across the U.S. These fractional influences are greatest in the intermountain western U.S. and are slightly smaller than the seasonal mean

MDA8 metric. This conclusion was also reached in a separate analysis recently completed by Lapina et al. (2014).

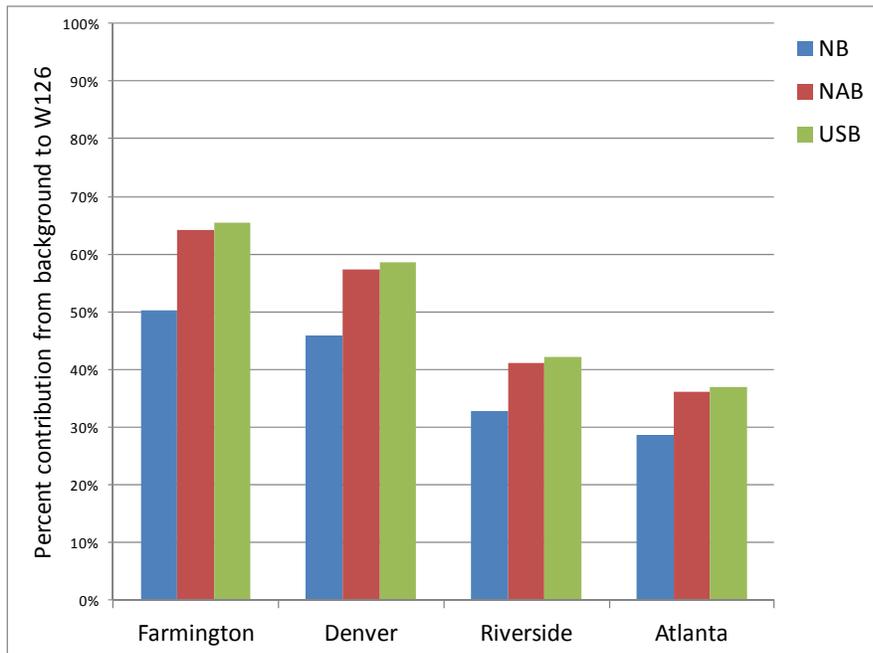


Figure 2-16. Fractional influence of background sources to W126 levels in four sample locations. Model estimates based on 2007 CMAQ zero-out modeling.

2.4.6 Estimated Magnitude of Individual Components of Background O₃

To provide a fuller characterization of background O₃ levels, it is useful to develop an understanding of the relative contributions of various background elements to total background O₃. This section will utilize the supplemental 2007 air quality modeling estimates to consider the relative contribution of specific elements of background O₃. Several background elements were isolated in either the zero-out or source apportionment modeling. Appendix A provides more detail on these analyses. In conjunction with the previous analyses summarized in the ISA, some broad characterizations of the individual components of background O₃ can be developed.

The recent 2007 EPA modeling confirms the importance of methane emissions and international ozone precursor emissions in contributing to background O₃. Methane has an atmospheric lifetime of about a decade and can be an important contributor to ozone on longer time scales. Anthropogenic methane emission sources include agriculture, coal mines, landfills, and natural gas and oil systems. The difference between the NAB and NB zero-out scenarios provides an estimate of contributions from international anthropogenic emissions and anthropogenic methane, which is modeled by reducing model concentrations from present-day values to pre-industrial levels. The ISA (US EPA, 2013, section 3.4) estimated that roughly half of the difference between the NB and NAB scenarios resulted from the removal of anthropogenic

methane emissions and that the other half resulted from international anthropogenic emissions of shorter-lived O₃ precursors (e.g., NO_x and VOC). Figure 2-17 shows the difference in seasonal mean MDA8 O₃ levels between the NB and NAB scenarios. North American seasonal mean background is 6-15 ppb higher than comparable natural background levels. The most frequent increment is an 8-10 ppb increase when the methane is increased and the non-North American emissions are added. These estimates of seasonal-mean external enhancement are similar to previous estimates summarized in the ISA (e.g., Fiore et al., 2009; Zhang et al., 2011). It is not possible via the EPA 2007 modeling to parse out what fraction of this change is due to emissions outside of North America, as opposed to what fraction is due to anthropogenic methane emissions, but the modeling suggests that any control measures to reduce emissions from either of these terms have the potential to contribute in an important way to reduce average background O₃ levels in the U.S.

The difference between the NAB and USB scenarios is easier to interpret as it only involves one change, the inclusion of anthropogenic emissions from the in-domain portion of Canada and Mexico. These emissions (not shown here, but depicted in Appendix A) contribute less than 2 ppb to the seasonal mean MDA8 O₃ levels over most of the U.S. There are 70 sites, near an international border, where the modeling estimates Canadian/Mexican seasonal average impacts of 2-4 ppb. Peak single-day MDA8 impacts from these specific international emissions sources can approach 25 ppb (e.g., San Diego, Buffalo NY).

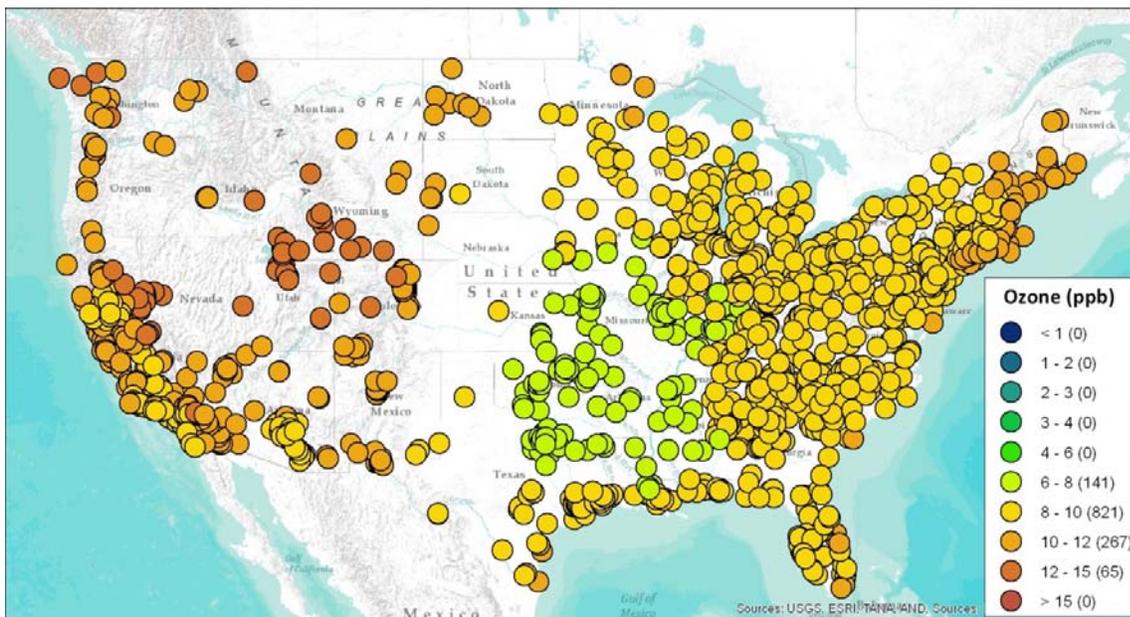


Figure 2-17. Differences in seasonal mean O₃ (ppb) between the NAB and NB scenarios.

Eleven separate source categories were tracked in the source apportionment modeling, including five boundary condition terms (East, South, West, North, and top) and six emissions sectors within the domain. The contributions of each of these terms is provided in the Appendix and summarized below. At most locations, the five model boundary terms contributed an aggregate 40-60 percent of the total seasonal mean MDA8 O₃ across the U.S. The highest proportional impacts from the boundary conditions are along the coastlines and the intermountain West. The O₃ entering the model domain via the boundary conditions can have a variety of origins including: a) natural sources of O₃ and precursors emanating from outside the domain, b) anthropogenic sources of O₃ precursors emanating from outside the domain, and c) some fraction of U.S. emissions (natural and anthropogenic) which exit the regional model domain but get re-imported into the domain via synoptic-scale recirculation. Accordingly, it is not possible to relate the boundary condition contribution to any specific background element. The single largest sector that was tracked in the source apportionment modeling was U.S. anthropogenic emissions. Figure 2-18 shows the contributions from this sector to seasonal mean MDA8 O₃ levels. Over most of the U.S. this term contributes 40-60 percent to the total seasonal mean O₃. As discussed in section 2.4.3, these contributions are even higher when only high O₃ days are considered. International shipping emissions, as well as fires and other biogenic emissions also contribute in a non-negligible way to background O₃ over the U.S. The key finding from this analysis is that air quality planning efforts to reduce anthropogenic methane emissions and international NO_x/VOC emissions (e.g., migrating from Asia, Canada, and Mexico; and from commercial shipping) have the potential to lower background O₃ levels.

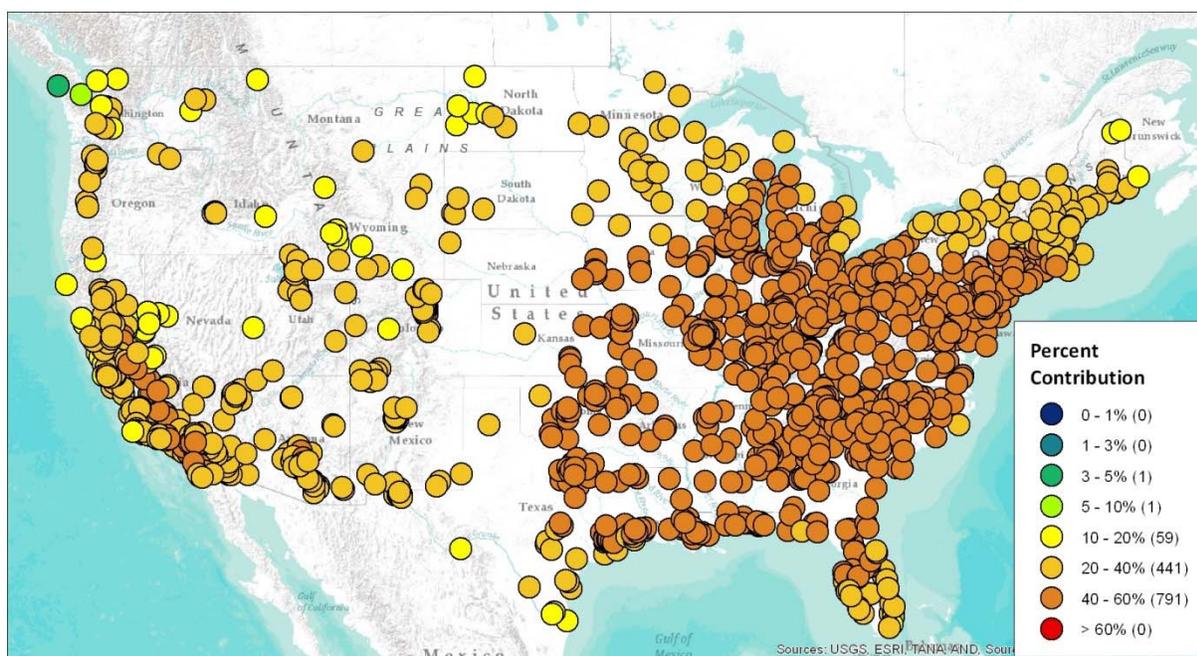


Figure 2-18. Percent contribution of U.S. anthropogenic emissions to total seasonal mean MDA8 O₃ levels, based on 2007 source apportionment modeling.

2.4.7 Summary

For a variety of reasons, it is challenging to present a comprehensive summary of all the components and implications of background O₃. In many forums the term “background” is used generically and the lack of specificity can lead to confusion as to what sources are being considered. Additionally, it is well established that the impacts of background sources can vary greatly over space and time which makes it difficult to present a simple summary of background O₃ levels. Further, background O₃ can be generated by a variety of processes, each of which can lead to differential patterns in space and time, and which often have different regulatory ramifications. Finally, background O₃ is difficult to measure and thus, typically requires air quality modeling which has inherent uncertainties and potential errors and biases.

That said, EPA believes the following concise and three step summary of the implications of background O₃ on the NAAQS review is appropriate, as based on previous modeling exercises and the more recent EPA analyses summarized herein. First, background O₃ exists and can comprise a considerable fraction of total seasonal mean MDA8 O₃ and W126 across the U.S. Air quality models can estimate the fractional contribution of background sources to total O₃ in an individual area. The largest absolute values of background (NB, NAB, USB, or apportionment-

based USB) are modeled to occur at locations in the intermountain western U.S. and are maximized in the spring and early summer seasons. Second, the modeling indicates that U.S. anthropogenic emission sources are the dominant contributor to the majority of modeled O₃ exceedances of the NAAQS. Higher O₃ days generally have smaller fractional contributions from background. This finding indicates that the relative importance of background O₃ would increase were O₃ concentrations to decrease with a lower level of the O₃ NAAQS. Third and finally, while the majority of modeled O₃ exceedances have local and domestic regional emissions as their primary cause, there can be events where O₃ levels approach or exceed 60-75 ppb due to the influence of background sources. These events are relatively infrequent and EPA has policies that could allow for the exclusion of air quality monitoring data affected by these types of events from design value calculations.

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3 Adequacy of the Current Primary Standard

This chapter presents staff's considerations and conclusions regarding the adequacy of the current primary O₃ NAAQS. In doing so, we pose the following overarching question:

Does the currently available scientific evidence and exposure/risk information, as reflected in the ISA and HREA, support or call into question the adequacy of the current O₃ standard?

As discussed more fully in this chapter, staff reaches the conclusion that the available evidence and exposure and risk information clearly calls into question the adequacy of public health protection provided by the current primary standard. This evidence and information provides strong support for the occurrence of a range of adverse respiratory effects, and mortality, under air quality conditions that would meet the current standard. Based on the analyses in the HREA, we conclude that the exposures and risks projected to remain upon meeting the current standard are indicative of risks that can reasonably be judged to be important from a public health perspective. Thus, staff concludes that the evidence and information provides strong support for giving consideration to revising the current primary standard in order to provide increased public health protection against an array of adverse health effects that range from decreased lung function and respiratory symptoms to more serious indicators of morbidity (e.g., including emergency department visits and hospital admissions), and mortality. The remainder of this chapter discusses the evidence and exposure/risk information, and the considerations and conclusions based on that evidence and information, supporting staff's overarching conclusion regarding the adequacy of the current primary O₃ standard.

In addressing the overarching question for this chapter, we pose a series of more specific questions, as discussed in sections 3.1 through 3.4 below. Section 3.1 presents our consideration of the available scientific evidence (i.e., evidence-based considerations) about the health effects associated with short- and long-term O₃ exposures. Section 3.2 presents our consideration of available estimates of O₃ exposures and health risks (exposure- and risk-based considerations). Section 3.3 discusses the advice and recommendations that we have received from the CASAC on the first draft O₃ PA, and on documents from previous reviews of the O₃ NAAQS. Section 3.4 revisits the overarching question of this section, and presents staff's conclusions regarding the adequacy of the current primary O₃ NAAQS.

3.1 EVIDENCE-BASED CONSIDERATIONS

This section presents our consideration of the available scientific evidence with regard to the adequacy of the current O₃ standard. Our approach, as summarized in section 1.3.1 above, is based on the full body of evidence in this review. We use information from the full evidence base to characterize our confidence in the extent to which O₃-attributable effects occur, and the extent to which such effects are adverse, over the ranges of O₃ exposure concentrations evaluated in controlled human exposure studies and over the distributions of ambient O₃ concentrations in locations where epidemiologic studies have been conducted. In doing so, we recognize that the available health effects evidence reflects a continuum from relatively high O₃ concentrations, at which scientists generally agree that adverse health effects are likely to occur, through lower concentrations, at which the likelihood and magnitude of a response become increasingly uncertain.

Section 3.1.1 summarizes a mode of action framework for understanding the effects of both short- and long-term O₃ exposures, based on Chapter 5 of the ISA (U.S. EPA, 2013). Section 3.1.2 presents our consideration of the evidence for health effects attributable to short-term and long-term O₃ exposures. Section 3.1.3 discusses the adversity of the effects. Section 3.1.4 presents our consideration of evidence with regard to concentrations associated with health effects and section 3.1.5 presents our consideration of the public health implications of exposures to O₃, including the adversity of effects and evidence for at-risk populations and lifestages.¹

3.1.1 Modes of Action

Our consideration of the evidence of effects attributable to short-and long-term exposures and the factors that increase risk in populations and lifestages builds upon evidence about the modes of action by which O₃ exerts effects (U.S. EPA, 2013; section 5.3). Mode of action refers to a sequence of key events and processes that result in a given toxic effect; elucidation of mechanisms provides a more detailed understanding of these key events and processes. The purpose of this section is to describe the key events and pathways that contribute to health effects resulting from both short-term and long-term exposures to O₃. The extensive research carried out

¹ Here, as in the ISA, the term “at-risk population” is used to encompass populations or lifestages that have a greater likelihood of experiencing health effects related to exposure to an air pollutant due to a variety of factors; other terms used in the literature include susceptible, vulnerable, and sensitive. These factors may be intrinsic, such as genetic factors, lifestage, or the presence of preexisting diseases, or they may be extrinsic, such as socioeconomic status (SES), activity pattern and exercise level, or increased pollutant exposures (U.S. EPA, 2013, p. lxx, 8-1, 8-2). The courts and the Act’s legislative history refer to these at-risk subpopulations as “susceptible” or “sensitive” populations. See, e.g., *American Lung Ass’n v. EPA*, 134 F. 3d 388, 389 (D.C. Cir. 1998) (“NAAQS must protect not only average health individuals, but also ‘sensitive citizens’ – children, for example, or people with asthma, emphysema, or other conditions rendering them particularly vulnerable to air pollution” (quoting S. Rep. No. 91-1196 at 10)).

over several decades in humans and animals has yielded numerous studies on mechanisms by which O₃ exerts its effects. It is well-understood that secondary oxidation products, which form as a result of O₃ exposure, initiate numerous responses at the cellular, tissue and whole organ level of the respiratory system. These responses include the activation of neural reflexes, initiation of inflammation, alteration of barrier epithelial function, sensitization of bronchial smooth muscle, modification of lung host defenses, and airways remodeling, as discussed below. These key events have the potential to affect other organ systems such as the cardiovascular system. It has been proposed that secondary oxidation products, which are bioactive and cytotoxic in the respiratory system, are also responsible for systemic effects. Recent studies in animal models show that inhalation of O₃ results in systematic oxidative stress.

Figure 3.1 below, adapted from Figure 5-8 of the ISA (ISA, Section 5.3.10, U.S. EPA, 2013), shows key events in the toxicity pathway of O₃ that are described in more detail below. The initial key event in the toxicity pathway of O₃ is the formation of secondary oxidation products in the respiratory tract (ISA, section 5.3, U.S. EPA, 2013). This mainly involves direct reactions with components of the extracellular lining fluid (ELF). Although the ELF has inherent capacity to quench (based on individual antioxidant capacity), this capacity can be overwhelmed, especially with exposure to elevated concentrations of O₃.² The resulting secondary oxidation products transmit signals to the epithelium, pain receptive nerve fibers and, if present, immune cells (i.e., eosinophils, dendritic cells and mast cells) involved in allergic responses. Thus, the effects of O₃ are mediated by components of ELF and by the multiple cell types found in the respiratory tract. Further, oxidative stress³ is an implicit part of this initial key event.

² The ELF is a complex mixture of lipids (fats), proteins, and antioxidants that serve as the first barrier and target for inhaled O₃. The quenching ability of antioxidant substances present in the ELF appear in most cases to limit interaction of O₃ with underlying tissues and to prevent penetration of O₃ deeper into the lung. However, as the ELF thickness decreases and becomes ultra thin in the alveolar region, it may be possible for direct interaction of O₃ with the underlying epithelial cells to occur. The formation of secondary oxidation products is likely related to the concentration of antioxidants present and the quenching ability of the lining fluid.

³ Oxidative stress reflects an imbalance between the systemic manifestation of reactive oxygen species, such as superoxide, and a biological system's ability to readily detoxify the reactive intermediates or to repair the resulting damage.

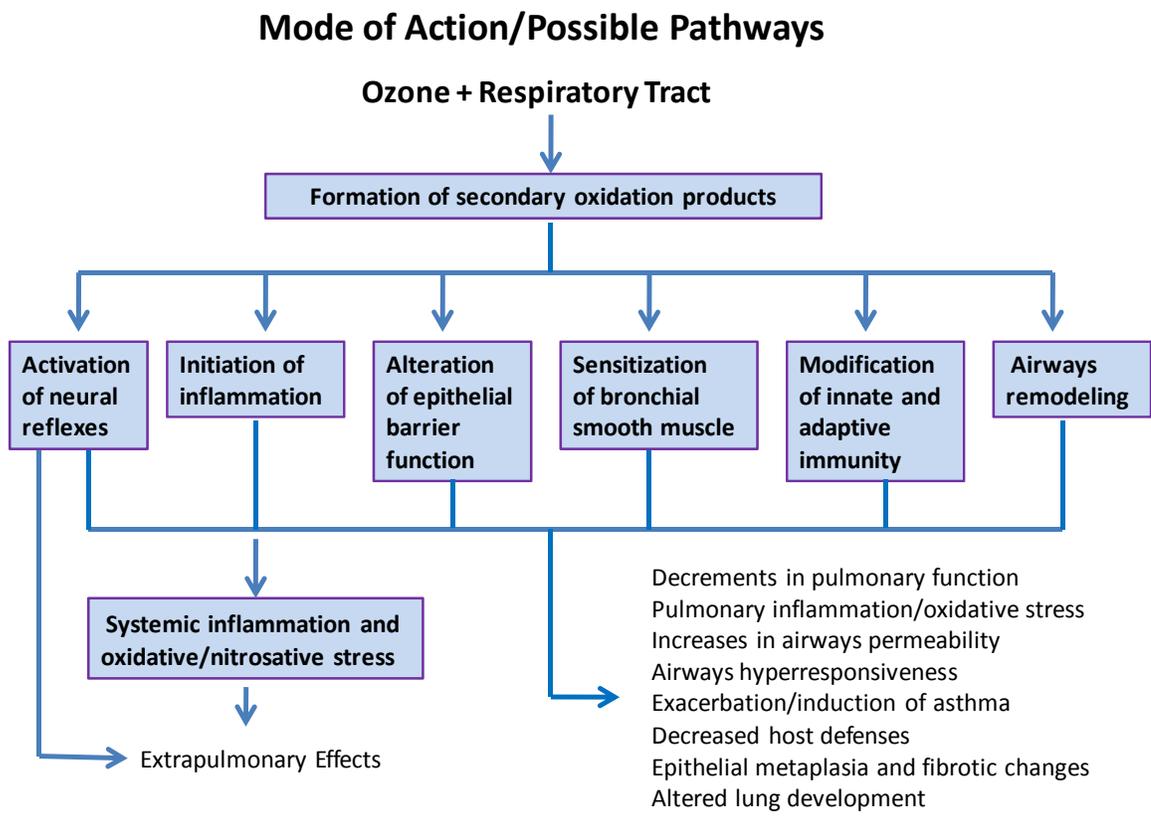


Figure 3-1. Modes of action/possible pathways underlying the health effects resulting from inhalation exposure to O₃. (Adapted from U.S. EPA, 2013, Figure 5-8)

Another key event in the toxicity pathway of O₃ is the activation of neural reflexes which lead to lung function decrements. Evidence is accumulating that secondary oxidation products are responsible for this effect. Different receptors on bronchial sensory nerves (i.e., C-fibers) have been shown to mediate separate effects of O₃ on pulmonary function. For example, pain (i.e., nociceptive) sensory nerves are involved in the involuntary truncation of inspiration which results in decreases in FVC, FEV₁, tidal volume and pain upon deep inspiration. Ozone exposure also results in activation of vagal sensory nerves and a mild increase in airway obstruction measured as increased sRaw. Activation of neural reflexes also results in extrapulmonary effects such as slow resting heart rate (i.e., bradycardia).

Initiation of inflammation is also a key event in the toxicity pathway of O₃. Secondary oxidation products, as well as cell signaling molecules (i.e., chemokines and cytokines) from airway epithelial cells and white blood cells (i.e., macrophages), have been implicated in the initiation of inflammation. Airways neutrophilia has been demonstrated in bronchoalveolar lavage fluid (BALF), mucosal biopsy and induced sputum samples. Influx of other cells (i.e., mast cells, monocytes and macrophages) also occur. Inflammation further contributes to

O₃-mediated oxidative stress. It should be noted that inflammation, as measured by airways neutrophilia, is not correlated with decrements in pulmonary function as measured by spirometry.

A fourth key event in the toxicity pathway of O₃ is alteration of epithelial barrier function. Increased permeability⁴ occurs as a result of damage to tight junctions between epithelial cells subsequent to O₃-induced injury and inflammation. It may play a role in allergic sensitization and in airway hyperresponsiveness (AHR). Genetic susceptibility has been associated with this pathway.

A fifth key event in the toxicity pathway of O₃ is the sensitization of bronchial smooth muscle. Airway hyperresponsiveness, or increased bronchial reactivity, can be both a rapidly occurring and a persistent response. The mechanisms responsible for AHR are not well-understood. Tachykinins, peptides that can excite neurons and cause smooth muscle contraction, and the secondary oxidation products of O₃ have been proposed as mediators of the early response and inflammation-derived products have been proposed as mediators of the later response. Other chemical signaling molecules (i.e., cytokines and chemokines) have been implicated in the AHR response to O₃ in animal models. Antioxidants may confer protection.

A sixth key event in the toxicity pathway of O₃ is the modification of innate/adaptive immunity. While the majority of evidence for this key event comes from animal studies, there are several studies suggesting that this pathway may also be relevant in humans. Ozone exposure of human subjects resulted in recruitment of activated innate immune cells to the airways. Animal studies further linked O₃-mediated activation of the innate immune system to the development of nonspecific AHR, demonstrated an interaction between allergen and O₃ in the induction of nonspecific AHR, and found that O₃ acted as an adjuvant for allergic sensitization through the activation of both innate and adaptive immunity. These studies provide evidence that O₃ can alter host immunologic response and lead to immune system dysfunction. These mechanisms may underlie the exacerbation and induction of asthma, as well as decreases in lung host defense.

Another key event in the toxicity pathway of O₃ is airways remodeling. Persistent inflammation and injury, which are observed in animal models of chronic and intermittent

⁴ Cells in epithelium are very densely packed together, leaving very little intercellular space. All epithelial cells rest on a basement membrane, a thin sheet of fibers that acts as scaffolding on which epithelium can grow and regenerate after injuries. Epithelial tissue is innervated but avascular; it must be nourished by substances diffusing from the blood vessels in the underlying tissue. Injury to epithelial cells, such as caused by oxidative stress, can cause the epithelium to become more permeable to substances in the underlying vasculature.

exposure to O₃, are associated with morphologic changes such as mucous cell metaplasia⁵ of nasal epithelium, bronchiolar metaplasia of alveolar ducts and fibrotic changes in small airways (see Section 7.2.3 of the ISA, U.S. EPA, 2013). Mechanisms responsible for these responses are not well-understood. However, a recent study in mice demonstrated a key role for a signaling pathway in the deposition of collagen in the airway wall following chronic intermittent exposure to O₃. Chronic intermittent exposure to O₃ has also been shown to result in effects on the developing lung and immune system.

Systemic inflammation and vascular oxidative/nitrosative stress are also key events in the toxicity pathway of O₃. Extrapulmonary effects of O₃ occur in numerous organ systems, including the cardiovascular, central nervous, reproductive, and hepatic systems (see Sections 6.3 to 6.5 and Sections 7.3 to 7.5 of the ISA, U.S. EPA, 2013). It has been proposed that lipid oxidation products resulting from reaction of O₃ with lipids and/or cellular membranes in the ELF are responsible for systemic responses; however, it is not known whether they gain access to the circulation. Alternatively, release of diffusible mediators from the lung into the circulation may initiate or propagate inflammatory responses in the circulation or other organ systems.

Responses to O₃ exposure are variable within the population. Although studies have shown a large range of pulmonary function (i.e., spirometric) responses to O₃ among healthy young adults, responses within an individual are relatively consistent over time. Other responses to O₃ have also been characterized by a large degree of inter-individual variability. For example, a 3- to 20-fold difference among subjects in their studies in airways inflammation (i.e., neutrophilia influx) following O₃ exposure has been reported (Schelegle et al., 1991 and Devlin et al., 1991, respectively). Reproducibility of an individual's inflammatory response to O₃ exposure in humans, measured as sputum neutrophilia, was demonstrated by Holz et al. (1999). Since individual inflammatory responses were relatively consistent across time, it was thought that inflammatory responsiveness reflected an intrinsic characteristic of the subject (Mudway and Kelly, 2000). While the basis for the observed inter-individual variability in responsiveness to O₃ is not clear, section 5.4.2 of the ISA (U.S. EPA, 2013) discusses mechanisms that may underlie the variability in responses seen among individuals. Certain functional genetic polymorphisms, pre-existing conditions or diseases, nutritional status, lifestages, and co-exposures contribute to altered risk of O₃-induced effects.

⁵ Metaplasia is the reversible replacement of one differentiated cell type with another mature differentiated cell type. The change from one type of cell to another may generally be a part of normal maturation process or caused by some sort of abnormal stimulus. In simplistic terms, it is as if the original cells are not robust enough to withstand the new environment, and so they change into another type more suited to the new environment. If the stimulus that caused metaplasia is removed or ceases, tissues return to their normal pattern of differentiation.

Experimental evidence for such O₃-induced changes contributes to our understanding of the biological plausibility of adverse O₃-related health effects, including a range of respiratory effects as well as effects outside the respiratory system (e.g., cardiovascular effects) (U.S. EPA, 2013, Chapters 6 and 7).

3.1.2 Nature of Effects

- **To what extent does the currently available scientific evidence alter or strengthen our conclusions from the last review regarding health effects attributable to O₃ exposure in ambient air? Are previously identified uncertainties reduced or do important uncertainties remain?**

The health effects of ozone are described in detail in the assessment of the evidence available in this review which is largely consistent with conclusions of past Air Quality Criteria Documents (AQCD). In some categories of health effects, there is newly available evidence regarding some aspects of the effects described in the last review or that strengthens our conclusions regarding aspects of O₃ toxicity on a particular physiological system (U.S. EPA, 2013, Table 1-1). A sizeable number of studies on O₃ health effects are newly available in this review and are critically assessed in the ISA as part of the full body of evidence. Based on this assessment, the ISA determined that a causal relationship⁶ exists between short-term exposure to O₃ in ambient air⁷ and effects on the respiratory system and that a likely to be causal relationship⁸ exists between long-term exposure to O₃ in ambient air and respiratory effects (U.S. EPA, 2013, pp. 1-6 to 1-7). As stated in the ISA, “[c]ollectively, a very large amount of evidence spanning several decades supports a relationship between exposure to O₃ and a broad range of respiratory effects” (ISA, p. 1-6). Additionally, the ISA determined that the relationships between short-term exposures to O₃ in ambient air and both total mortality and cardiovascular effects are likely to be causal, based on expanded evidence bases in the current review (U.S. EPA, 2013, pp. 1-7 to 1-8). In the ISA, EPA additionally determined that the currently available evidence for additional endpoints is suggestive of causal relationships between short-term (central nervous system effects) and long-term exposure (cardiovascular effects, central nervous system effects and total mortality) to ambient O₃. Consistent with emphasis in past reviews on O₃ health effects for which

⁶ Since the last O₃ NAAQS review, the ISAs which have replaced CDs in documenting each review of the scientific evidence (or air quality criteria) employ a systematic framework for weighing the evidence and describing associated conclusions with regard to causality, using established descriptors, as summarized in section 1.3.1 above (U.S. EPA, 2013, Preamble).

⁷ In determining that a causal relationship exists for O₃ with specific health effects, EPA has concluded that “[e]vidence is sufficient to conclude that there is a causal relationship with relevant pollutant exposures” (ISA, p. lxiv).

⁸ In determining a likely to be a causal relationship exists for O₃ with specific health effects, EPA has concluded that “[e]vidence is sufficient to conclude that a causal relationship is likely to exist with relevant pollutant exposures, but important uncertainties remain” (ISA, p. lxiv).

the evidence is strongest, we place the greatest emphasis on studies of health effects that have been judged in the ISA to be caused by, or likely to be caused by, O₃ exposures (U.S. EPA, 2013, section 2.5.2).

This section presents our consideration of the evidence for health effects attributable to O₃ exposures, including respiratory morbidity and mortality effects attributable to short- and long-term exposures, and cardiovascular system effects (including mortality) and total mortality attributable to short-term exposures. We focus particularly on considering the extent to which the scientific evidence available in the current review has been strengthened since the last review, and the extent to which important uncertainties and limitations in the evidence from the last review have been addressed. In section 3.1.2.2, we then consider the extent to which the available evidence indicates health effects may be attributable to ambient O₃ concentrations likely to be allowed by the current O₃ NAAQS. In this section, we address the following specific question for each category of health effects considering the evidence available in the 2008 review of the standard as well as evidence that has become available since then. The ISA summarizes the longstanding body of evidence for O₃ respiratory effects as follows (U.S. EPA, 2013, p. 1-6).

The clearest evidence for health effects associated with exposure to O₃ is provided by studies of respiratory effects. Collectively, a very large amount of evidence spanning several decades supports a relationship between exposure to O₃ and a broad range of respiratory effects (see Section 6.2.9 and Section 7.2.8). The majority of this evidence is derived from studies investigating short-term exposures (i.e., hours to weeks) to O₃, although animal toxicological studies and recent epidemiologic evidence demonstrate that long-term exposure (i.e., months to years) may also harm the respiratory system.

The extensive body of evidence supporting a causal relationship between short-term O₃ exposures and respiratory effects is discussed in detail in Chapter 6 of the ISA (U.S. EPA, 2013), while evidence for respiratory effects associated with long-term or repeated O₃ exposures are discussed in chapter 7 of that document (U.S., EPA, 2013).

3.1.2.1 Respiratory Effects – Short-term Exposures

- **To what extent does the currently available scientific evidence, including related uncertainties, strengthen or alter our understanding from the last review of respiratory effects attributable to short-term O₃ exposures?**

The 2006 O₃ AQCD concluded that there was clear, consistent evidence of a causal relationship between short-term O₃ exposure and respiratory effects (U.S. EPA, 2006). This conclusion was substantiated by evidence from controlled human exposure and toxicological studies indicating a range of respiratory effects in response to short-term O₃ exposures, including pulmonary function decrements and increases in respiratory symptoms, lung inflammation, lung

permeability, and airway hyperresponsiveness. Toxicological studies provided additional evidence for O₃-induced impairment of host defenses. Combined, these findings from experimental studies provided support for epidemiologic evidence, in which short-term increases in ambient O₃ concentration were consistently associated with decreases in lung function in populations with increased outdoor exposures, especially children with asthma and healthy children; increases in respiratory symptoms and asthma medication use in children with asthma; and increases in respiratory-related hospital admissions and asthma-related ED visits (U.S. EPA, 2013, pp. 6-1 to 6-2).

As discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.9), studies evaluated since the completion of the 2006 O₃ AQCD support and expand upon the strong body of evidence that, in the last review, indicated a causal relationship between short-term O₃ exposures and respiratory health effects. Recent controlled human exposure studies conducted in young, healthy adults with moderate exertion have reported FEV₁ decrements and pulmonary inflammation following prolonged exposures to O₃ concentrations as low as 60 ppb, and respiratory symptoms following exposures to concentrations as low as 70 ppb.⁹ Epidemiologic studies provide evidence that increases in ambient O₃ exposures can result in lung function decrements, increases in respiratory symptoms, and pulmonary inflammation in children with asthma; increases in respiratory-related hospital admissions and emergency department visits; and increases in respiratory mortality. Some of these studies report such associations even for O₃ concentrations at the low end of the distribution of daily concentrations. Recent epidemiologic studies report that associations with respiratory morbidity and mortality are stronger during the warm/summer months and remain robust after adjustment for copollutants. Recent toxicological studies reporting O₃-induced inflammation, airway hyperresponsiveness, and impaired lung host defense continue to support the biological plausibility and modes of action for the O₃-induced respiratory effects observed in the controlled human exposure and epidemiologic studies. Further support is provided by recent studies that found O₃-associated increases in indicators of airway inflammation and oxidative stress in children with asthma (U.S. EPA, 2013, section 6.2.9). Together, epidemiologic and experimental studies support a continuum of respiratory effects associated with O₃ exposure that can result in respiratory-related emergency department visits, hospital admissions, and/or mortality (U.S. EPA, 2013, section 6.2.9).

Across respiratory endpoints, evidence indicates antioxidant capacity may modify the risk of respiratory morbidity associated with O₃ exposure (U.S. EPA, 2013, section 6.2.9, p. 6-161) (section 3.1.1, above). The potentially elevated risk of populations with diminished

⁹ Schelegle et al. (2009) reported a statistically significant increase in respiratory symptoms in healthy adults at a target O₃ exposure concentration of 70 ppb. For this 70 ppb target, Schelegle et al. (2009) reported an actual exposure concentration, averaged over the study period, of 72 ppb.

antioxidant capacity and the reduced risk of populations with sufficient antioxidant capacity is supported by epidemiologic and controlled human exposure studies. Additional evidence characterizes O₃-induced decreases in antioxidant levels as a key event in the mode of action for downstream effects.

We describe key aspects of this evidence below with regard to lung function decrements; pulmonary inflammation, injury, and oxidative stress; airway hyperresponsiveness; respiratory symptoms and medication use; lung host defense; allergic and asthma-related responses; hospital admissions and emergency department visits; and respiratory mortality.

Lung Function Decrements

In the 2008 review, a large number of controlled human exposure studies reported O₃-induced lung function decrements in young, healthy adults engaged in intermittent, moderate exertion following 6.6 hour exposures to O₃ concentrations at or above 80 ppb. Although two studies also reported effects following exposures to lower concentrations, an important uncertainty in the last review was the extent to which exposures to O₃ concentrations below 80 ppb result in lung function decrements. In addition, in the last review epidemiologic panel studies had reported O₃-associated lung function decrements in a variety of different populations (e.g., children, outdoor workers) likely to experience increased exposures. In the current review, additional controlled human exposure studies are available that have evaluated exposures to O₃ concentrations of 60 or 70 ppb. The available evidence from controlled human exposure and panel studies is assessed in detail in the ISA (U.S. EPA, 2013, section 6.2.1) and is summarized below.

Controlled exposures to O₃ concentrations that can be found in the ambient air can result in a number of lung function effects, including decreased inspiratory capacity; mild bronchoconstriction; and rapid, shallow breathing patterns during exercise. Reflex inhibition of inspiration results in a decrease in forced vital capacity (FVC) and total lung capacity (TLC) and, in combination with mild bronchoconstriction, contributes to a decrease in the forced expiratory volume in 1 second (FEV₁) (U.S. EPA, 2013, section 6.2.1.1).¹⁰ Accumulating evidence indicates that such effects are mediated by activation of sensory nerves, resulting in the involuntary truncation of inspiration and a mild increase in airway obstruction due to bronchoconstriction (U.S. EPA, 2013, section 5.3.10).

¹⁰ The controlled human exposure studies emphasized in this PA utilize only healthy adult subjects. In the near absence of controlled human exposure data for children, HREA estimates of lung function decrements are based on the assumption that children exhibit the same lung function responses following O₃ exposures as healthy 18 year olds (U.S. EPA, 2014, sections 6.2.4 and 6.5). This assumption is justified in part by the findings of McDonnell et al. (1985), who reported that children 8-11 year old experienced FEV₁ responses similar to those observed in adults 18-35 years old. Thus, the conclusions about the occurrence of lung function decrements that follow generally apply to children as well as to adults.

Data from controlled human exposure studies indicate that increasing the duration of O₃ exposures and increasing ventilation rates decreases the O₃ exposure concentrations required to impair lung function. Ozone exposure concentrations well above those typically found in ambient air are required to impair lung function in healthy resting adults, while exposure to O₃ concentrations at or below those in the ambient air have been reported to impair lung function in healthy adults exposed for longer durations while undergoing intermittent, moderate exertion (U.S. EPA, 2013, section 6.2.1.1). With repeated O₃ exposures over several days, FEV₁ responses become attenuated in both healthy adults and adults with mild asthma, though this attenuation of response is lost after about a week without exposure (U.S. EPA, 2013, section 6.2.1.1; page 6-27).

When considering controlled human exposures studies of O₃-induced lung function decrements we evaluate both group mean changes in lung function and the interindividual variability in the magnitude of responses. An advantage of O₃ controlled human exposure studies (i.e., compared to the epidemiologic panel studies discussed below) is that reported effects necessarily result from exposures to O₃ itself.¹¹ To the extent studies report statistically significant decrements in mean lung function following O₃ exposures after controlling for other factors, we have more confidence that measured decrements are due to the O₃ exposure itself, rather than to chance alone. As discussed below, group mean changes in lung function are often small, especially following exposures to relatively low O₃ concentrations (e.g., 60 ppb). However, even when group mean decrements in lung function are small, some individuals could experience decrements that are “clinically meaningful” (Pellegrino et al., 2005; ATS, 1991) with respect to criteria for spirometric testing, and/or that could be considered “adverse” with respect to public health policy decisions (section 3.1.3 below).

At the time of the last review, a number of controlled human exposure studies had reported lung function decrements in young, healthy adults following prolonged (6.6-hour) exposures while at moderate exertion to O₃ concentrations at and above 80 ppb. In addition, there were two controlled human exposure studies by Adams (2002, 2006) that examined lung function effects following exposures to O₃ concentrations of 60 ppb. The EPA’s analysis of the data from the Adams (2006) study reported a small but statistically significant O₃-induced decrement in group mean FEV₁ following exposures of young, healthy adults, while at moderate exertion, to 60 ppb O₃, when compared with filtered air controls (Brown, 2008).¹² Further

¹¹ The ISA notes that the use of filtered air responses as a control for the assessment of responses following O₃ exposure in controlled human exposure studies serves to eliminate alternative explanations other than O₃ itself in causing the measured responses (U.S. EPA, 2013, section 6.2.1.1).

¹² Adams (2006) did not find effects on FEV₁ at 60 ppb to be statistically significant. In an analysis of the Adams (2006) data, even after removal of potential outliers, Brown et al. (2008) found the average effect on FEV₁ at 60 ppb to be small, but highly statistically significant ($p < 0.002$) using several common statistical tests.

examination of the post-exposure FEV₁ data, and mean data for other time points and other concentrations, indicated that the temporal pattern of the response to 60 ppb O₃ was generally consistent with the temporal patterns of responses to higher O₃ concentrations in this and other studies. (75 FR 2950, January 19, 2010). This suggested a pattern of response following exposures to 60 ppb O₃ that was consistent with a dose-response relationship, rather than random variability. See also *State of Mississippi v. EPA*, 744 F. 3d at 1347 (upholding EPA's interpretation of the Adams studies).

Figure 6-1 in the ISA summarizes the currently available evidence from multiple controlled human exposure studies evaluating group mean changes in FEV₁ following prolonged O₃ exposures (i.e., 6.6 hours) in young, healthy adults engaged in moderate levels of physical activity (U.S. EPA, 2013, section 6.2.1.1). With regard to the group mean changes reported in these studies, the ISA specifically notes the following (U.S. EPA, 2013, section 6.2.1.1, Figure 6-1):

1. Prolonged exposure to 40 ppb O₃ results in a small decrease in group mean FEV₁ that is not statistically different from responses following exposure to filtered air (Adams, 2002; Adams, 2006).
2. Prolonged exposure to an average O₃ concentration of 60 ppb results in group mean FEV₁ decrements ranging from 1.8% to 3.6% (Adams 2002; Adams, 2006;¹³ Schelegle et al., 2009;¹⁴ Kim et al., 2011). Based on data from multiple studies, the weighted average group mean decrement was 2.7%. In some analyses, these group mean decrements in lung function were statistically significant (Brown et al., 2008; Kim et al., 2011), while in other analyses they were not (Adams, 2006; Schelegle et al., 2009).¹⁵
3. Prolonged exposure to an average O₃ concentration of 70 ppb results in a statistically significant group mean decrement in FEV₁ of about 6% (Schelegle et al., 2009).¹⁶
4. Prolonged square-wave exposure to average O₃ concentrations of 80 ppb, 100 ppb, or 120 ppb O₃ results in statistically significant group mean decrements in FEV₁ ranging from 6 to 8%, 8 to 14%, and 13 to 16%, respectively (Folinsbee et al., 1988; Horstman et al., 1990; McDonnell et al., 1991; Adams, 2002; Adams, 2003; Adams, 2006).

¹³ Adams (2006; 2002) both provide data for an additional group of 30 healthy subjects that were exposed via facemask to 60 ppb (square-wave) O₃ for 6.6 hours with moderate exercise ($\dot{V}_E = 23$ L/min per m² BSA). These subjects are described on page 133 of Adams (2006) and pages 747 and 761 of Adams (2002). The FEV₁ decrement may be somewhat increased due to a target \dot{V}_E of 23 L/min per m² BSA relative to other studies having the target \dot{V}_E of 20 L/min per m² BSA. The facemask exposure is not expected to affect the FEV₁ responses relative to a chamber exposure.

¹⁴ Schelegle et al. (2009) reported an actual mean exposure concentration of 63 ppb for the target of 60 ppb.

¹⁵ Adams (2006) did not find effects on FEV₁ at 60 ppb to be statistically significant. In an analysis of the Adams (2006) data, Brown et al. (2008) addressed the more fundamental question of whether there were statistically significant differences in responses before and after the 6.6 hour exposure period and found the average effect on FEV₁ at 60 ppb to be small, but highly statistically significant using several common statistical tests, even after removal of potential outliers.

¹⁶ Schelegle et al. (2009) reported an actual mean exposure concentration of 72 ppb for the target of 70 ppb.

As illustrated in Figure 6-1 of the ISA, there is a smooth dose-response curve without evidence of a threshold for exposures between 40 and 120 ppb O₃ (U.S. EPA, 2013, Figure 6-1). When these data are taken together, the ISA concludes that “mean FEV₁ is clearly decreased by 6.6-h exposures to 60 ppb O₃ and higher concentrations in [healthy, young adult] subjects performing moderate exercise” (U.S. EPA, 2013, p. 6-9).

With respect to interindividual variability in lung function, in an individual with relatively “normal” lung function, with recognition of the technical and biological variability in measurements, within-day changes in FEV₁ of $\geq 5\%$ are clinically meaningful (Pellegrino et al., 2005; ATS, 1991). The ISA (U.S. EPA, 2013, section 6.1.) focuses on individuals with $>10\%$ decrements in FEV₁ for two reasons. A 10% FEV₁ decrement is accepted by the American Thoracic Society (ATS) as an abnormal response and a reasonable criterion for assessing exercise-induced bronchoconstriction (Dryden et al., 2010; ATS, 2000). (U.S. EPA, 2013, section 6.2.1.1). Also, some individuals in the Schelegle et al. (2009) study experienced 5-10% FEV₁ decrements following exposure to filtered air.

In previous NAAQS reviews, the EPA has made judgments regarding the potential implications for individuals experiencing FEV₁ decrements of varying degrees of severity.¹⁷ For people with lung disease, the EPA judged that moderate functional decrements (e.g., FEV₁ decrements ≥ 10 percent but < 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 (75 FR 2973, January 19, 2010). In previous reviews CASAC has endorsed these conclusions. In the context of standard setting, in the last review O₃ review CASAC indicated that it is appropriate to focus on the lower end of the range of moderate functional responses (e.g., FEV₁ decrements ≥ 10 percent) when estimating potentially adverse lung function decrements in people with lung disease, especially children with asthma (Henderson, 2006). More specifically, CASAC stated that “[a] 10% decrement in FEV₁ can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV₁) such that a $\geq 10\%$ decrement could lead to moderate to severe respiratory symptoms” (Samet, 2011). In this review, CASAC reiterated its support for this conclusion, stating that “[a]n FEV₁ decrement of $\geq 10\%$ is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014 p. 3). Therefore, in considering interindividual variability in

¹⁷ Such judgments have been made for decrements in FEV₁ as well as for increased airway responsiveness and symptomatic responses (e.g., cough, chest pain, wheeze). Ranges of pulmonary responses and their associated potential impacts are presented in Tables 3-2 and 3-3 of the Staff Paper (U.S. EPA, 2007).

O₃-induced lung function decrements in the current review, we also focus on the extent to which individuals were reported to experience FEV₁ decrements of 10% or greater.

New studies (Schelegle et al., 2009; Kim et al., 2011) add to the previously available evidence for interindividual variability in the responses of healthy adults following exposures to O₃. Following prolonged exposures to 80 ppb O₃ while at moderate exertion, the proportion of healthy adults experiencing FEV₁ decrements greater than 10% was 17% by Adams (2006), 26% by McDonnell (1996), and 29% by Schelegle et al. (2009). Following exposures to 60 ppb O₃, that proportion was 20% by Adams (2002), 3% by Adams (2006), 16% by Schelegle et al. (2009), and 5% by Kim et al. (2011). Based on these studies, the weighted average proportion of young, healthy adults with >10% FEV₁ decrements is 25% following exposure to 80 ppb O₃ and 10% following exposure to 60 ppb O₃ (U.S. EPA, 2013, page 6-19).¹⁸ The ISA notes that responses within an individual tend to be reproducible over a period of several months, indicating that interindividual differences reflect differences in intrinsic responsiveness. Given this, the ISA concludes that “a considerable fraction” of healthy individuals experience clinically meaningful decrements in lung function when exposed for 6.6 hours to 60 ppb O₃ during quasi continuous, moderate exertion (U.S. EPA, 2013, section 6.2.1.1, p. 6-20).

As discussed above (Figure 3-1) and in the ISA (U.S EPA, 2013, Section 5.3.2), secondary oxidation products formed following O₃ exposures can activate neural reflexes leading to decreased lung function. Two new quantitative models, discussed in section 6.2.1.1 of the ISA (U.S. EPA, 2013, p. 6-15), included mathematical approaches to simulate the protective effect of antioxidants in the ELF at lower ambient O₃ concentrations, and include a threshold below which changes in lung function do not occur (McDonnell et al., 2012; Schelegle et al., 2012).

McDonnell et al. (2012) and Schelegle et al. (2012) developed models using data on O₃ exposure concentrations, ventilation rates, duration of exposures, and lung function responses from a number of controlled human exposure studies. The McDonnell et al. (2012) and Schelegle et al. (2012) studies analyzed large datasets to fit compartmental models that included the concept of a dose of onset in lung function response or a response threshold based upon the inhaled O₃ dose. The first compartment in the McDonnell et al. (2012) model considers the level of oxidant stress in response to O₃ exposure to increase over time as a function of dose rate ($C \times \dot{V}_E$) and decrease by clearance or metabolism over time. In the second compartment of the McDonnell model, once oxidant stress reaches a threshold level the decrement in FEV₁ increases

¹⁸ The ISA also notes that by considering responses uncorrected for filtered air exposures, during which lung function typically improves (which would increase the size of the change, pre-and post-exposure), 10% is an underestimate of the proportion of healthy individuals that are likely to experience clinically meaningful changes in lung function following exposure for 6.6 hours to 60 ppb O₃ during intermittent moderate exertion (U.S. EPA, 2013, section 6.2.1.1).

as a sigmoid-shaped function. In the Schelegle et al. (2012) model, a first compartment acts as a reservoir in which oxidant stress builds up until the dose of onset, at which time it spills over into a second compartment. The second compartment is identical to the first compartment in McDonnell et al. (2012) model. The oxidant levels in the second compartment were multiplied by a responsiveness coefficient to predict FEV₁ responses for the Schelegle et al. (2012) model.

The McDonnell et al. (2012) model was fit to a large dataset consisting of the FEV₁ responses of 741 young, healthy adults (18-35 years of age) from 23 individual controlled exposure studies. Concentrations across individual studies ranged from 40 ppb to 400 ppb, activity level ranged from rest to heavy exercise, duration of exposure was from 2 to 7.6 hours. The extension of the McDonnell et al. (2012) model to children and older adults is discussed in section 6.2.1 of the ISA (U.S. EPA, 2013). Schelegle et al. (2012) also analyzed a large dataset with substantial overlap to that used by McDonnell et al. (2012). The Schelegle et al. (2012) model was fit to the FEV₁ responses of 220 young healthy adults (taken from a dataset of 704 individuals) from 21 individual controlled exposure studies. The resulting empirical models can estimate the frequency distribution of individual lung function responses for any exposure scenario as well as summary measures of the distribution such as the mean or median response and the proportions of individuals with FEV₁ decrements > 10%, 15%, and 20%.

The predictions of the McDonnell and Schelegle models are consistent with the observed results from the individual studies of O₃-induced FEV₁ decrements. Specifically, the model developed by McDonnell et al. (2012) predicts that 9% of healthy exercising adults would experience FEV₁ decrements greater than 10% following 6.6 hour exposure to 60 ppb O₃, and that 22% would experience such decrements following exposure to 80 ppb O₃ (U.S. EPA, 2013, p. 6-18 and Figure 6-3). The model developed by Schelegle et al. (2012) predicts that, for a prolonged (6.6 hours) O₃ exposure with moderate, quasi continuous exercise, the average dose of onset for FEV₁ decrement would be reached following 4 to 5 hours of exposure to 60 ppb, and following 3 to 4 hours of exposure to 80 ppb. However, 14% of the individuals had a dose of onset that was less than 40% of the average. Those individuals would reach their dose of onset following 1 to 2 hours of exposure to 50 to 80 ppb O₃ (U.S. EPA, 2013, p. 6-16), which is consistent with the threshold FEV₁ responses reported by McDonnell et al. (2012).

Epidemiologic studies¹⁹ have consistently linked short-term increases in ambient O₃ concentrations with lung function decrements in diverse populations and lifestages, including children attending summer camps, adults exercising or working outdoors, and groups with pre-existing respiratory diseases such as asthmatic children (U.S. EPA, 2013, section 6.2.1.2). Some

¹⁹ Unless otherwise specified, the epidemiologic studies discussed in this PA evaluate only adults.

of these studies reported ozone-associated lung function decrements accompanied by respiratory symptoms²⁰ in asthmatic children (Just et al., 2002; Mortimer et al., 2002; Ross et al., 2002; Gielen et al., 1997; Romieu et al., 1997; Thurston et al., 1997; Romieu et al., 1996). In contrast, studies of children in the general population have reported similar O₃-associated lung function decrements but without accompanying respiratory symptoms (Ward et al., 2002; Gold et al., 1999; Linn et al., 1996) (U.S. EPA, 2013, section 6.2.1.2).

Several epidemiologic panel studies reported that associations with lung function decrements persisted at relatively low ambient O₃ concentrations. For outdoor recreation or exercise, associations were reported in analyses restricted to 1-hour average O₃ concentrations less than 80 ppb (Spektor et al., 1988a; Spektor et al., 1988b), 60 ppb (Brunekreef et al., 1994; Spektor et al., 1988a), and 50 ppb (Brunekreef et al., 1994). Among outdoor workers, Brauer et al. (1996) found a robust association using daily 1-hour max O₃ concentrations less than 40 ppb. Ulmer et al. (1997) found a robust association in schoolchildren using 30-minute maximum O₃ concentrations less than 60 ppb. For 8-hour average O₃ concentrations, associations with lung function decrements in children with asthma were found to persist at concentrations less than 80 ppb in a U.S. multicity study (Mortimer et al., 2002) and less than 51 ppb in a study conducted in the Netherlands (Gielen et al., 1997).

Epidemiologic panel studies investigating the effects of short-term exposure to O₃ provided information on potential confounding by copollutants such as PM_{2.5}, PM₁₀, NO₂, or SO₂. These studies varied in how they evaluated confounding. Some studies of subjects exercising outdoors indicated that ambient concentrations of copollutants such as NO₂, SO₂, or acid aerosol were low, and thus not likely to confound associations observed for O₃ (Hoppe et al., 2003; Brunekreef et al., 1994; Hoek et al., 1993). In other studies of children with increased outdoor exposures, O₃ was consistently associated with decreases in lung function, whereas other pollutants such as PM_{2.5}, sulfate, and acid aerosol individually showed variable associations across studies (Thurston et al., 1997; Castillejos et al., 1995; Berry et al., 1991; Avol et al., 1990; Spektor et al., 1988a). Studies that conducted copollutant modeling generally found O₃-associated lung function decrements to be robust (i.e., most copollutant-adjusted effect estimates fell within the 95% CI of the single-pollutant effect estimates) (U.S. EPA, 2013, Figure 6-10 and Table 6-14). Most O₃ effect estimates for lung function were robust to adjustment for temperature, humidity, and copollutants such as PM_{2.5}, PM₁₀, NO₂, or SO₂. Although examined

²⁰ Reversible loss of lung function in combination with the presence of symptoms meets the ATS definition of adversity (ATS, 2000).

in only a few epidemiologic studies, O₃ also remained associated with decreases in lung function with adjustment for pollen or acid aerosols (U.S. EPA, 2013, section 6.2.1.2).

Several epidemiologic studies demonstrated the protective effects of vitamin E and vitamin C supplementation, and increased dietary antioxidant intake, on O₃-induced lung function decrements (Romieu et al., 2002) (U.S. EPA, 2013, Figure 6-7 and Table 6-8).²¹ These results provide support for the new, quantitative models (McDonnell et al., 2012; Schelegle et al., 2012), discussed above, which make use of the concept of oxidant stress to estimate the occurrence of lung function decrements following exposures to relatively low O₃ concentrations.

In conclusion, new information from controlled human exposure studies considerably strengthens the evidence and reduces the uncertainties, relative to the evidence that was available at the time of the 2008 review, regarding the presence and magnitude of lung function decrements in healthy adults following prolonged exposures to O₃ concentrations below 80 ppb. As discussed in Section 6.2.1.1 in the ISA (U.S. EPA, 2013, p. 6-12), there is information available from four separate studies that evaluated exposures to 60 ppb O₃ (Kim et al., 2011; Schelegle et al., 2009; Adams 2002; 2006). Although not consistently statistically significant, group mean FEV₁ decrements following exposures to 60 ppb O₃ are consistent among these studies. Moreover, as is illustrated in Figure 6-1 of the ISA (U.S. EPA, 2013), the group mean FEV₁ responses at 60 ppb fall on a smooth intake dose-response curve for exposures between 40 and 120 ppb O₃. These studies also indicate that, on average, 10% of young, healthy adults experience clinically meaningful decrements in lung function when exposed for 6.6 hours to 60 ppb O₃ during intermittent, moderate exertion. One recent study has also reported statistically significant decrements following exposures to 70 ppb O₃ (Schelegle et al., 2009). Predictions from newly developed quantitative models, based on the concept that O₃-induced oxidation results in lung function decrements, are consistent with these experimental results. Additionally, as discussed in more detail in section 3.1.4 below, epidemiologic studies continue to provide evidence of lung function decrements in people who are active outdoors, including people engaged in outdoor recreation or exercise, children, and outdoor workers, at low ambient O₃ concentrations. While few new epidemiologic studies of O₃-associated lung function decrements are available in this review, previously available studies have reported associations with decrements, including at relatively low ambient O₃ concentrations.

Pulmonary Inflammation, Injury, and Oxidative Stress

Ozone exposures result in increased respiratory tract inflammation and epithelial permeability. Inflammation is a host response to injury, and the induction of inflammation is

²¹ Evidence from controlled human exposure studies is mixed, suggesting that supplementation may be ineffective in the absence of antioxidant deficiency (U.S. EPA, 2013, p. 5-63).

evidence that injury has occurred. Oxidative stress has been shown to play a key role in initiating and sustaining O₃-induced inflammation. Secondary oxidation products formed as a result of reactions between O₃ and components of the ELF can increase the expression of molecules (i.e., cytokines, chemokines, and adhesion molecules) that can enhance airway epithelium permeability (U.S. EPA, 2013, Sections 5.3.3 and 5.3.4). As discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.3), O₃ exposures can initiate an acute inflammatory response throughout the respiratory tract that has been reported to persist for at least 18-24 hours after exposure.

Inflammation induced by exposure of humans to O₃ can have several potential outcomes: (1) inflammation induced by a single exposure (or several exposures over the course of a summer) can resolve entirely; (2) continued acute inflammation can evolve into a chronic inflammatory state; (3) continued inflammation can alter the structure and function of other pulmonary tissue, leading to diseases such as asthma; (4) inflammation can alter the body's host defense response to inhaled microorganisms, particularly in potentially at-risk populations or lifestages such as the very young and old; and (5) inflammation can alter the lung's response to other agents such as allergens or toxins (U.S. EPA, 2013, Section 6.2.3). Thus, lung injury and the resulting inflammation provide a mechanism by which O₃ may cause other more serious morbidity effects (e.g., asthma exacerbations).

In the last review, controlled human exposure studies reported O₃-induced airway inflammation following exposures at or above 80 ppb. In the current review, the link between O₃ exposures and airway inflammation and injury has been evaluated in additional controlled human exposure studies, as well as in recent epidemiologic studies. Controlled human exposure studies have generally been conducted in young, healthy adults or in adults with asthma using lavage (proximal airway and bronchoalveolar), bronchial biopsy, and more recently, induced sputum. These studies have evaluated one or more indicators of inflammation, including neutrophil²² (PMN) influx, markers of eosinophilic inflammation, increased permeability of the respiratory epithelium, and/or prevalence of proinflammatory molecules (U.S. EPA, 2013, section 6.2.3.1). Epidemiologic studies have generally evaluated associations between ambient O₃ and markers of inflammation and/or oxidative stress, which plays a key role in initiating and sustaining inflammation (U.S. EPA, 2013, section 6.2.3.2).

There is an extensive body of evidence from controlled human exposure studies indicating that short-term exposures to O₃ can cause pulmonary inflammation. Previously

²² Referred to as either neutrophils or polymorphonuclear neutrophils (or PMNs), these are the most abundant type of white blood cells in mammals. PMNs are recruited to the site of injury following trauma and are the hallmark of acute inflammation. The presence of PMNs in the lung has long been accepted as a hallmark of inflammation and is an important indicator that O₃ causes inflammation in the lungs. Neutrophilic inflammation of tissues indicates activation of the innate immune system and requires a complex series of events, that then are normally followed by processes that clear the evidence of acute inflammation.

available evidence indicated that O₃ causes an inflammatory response in the lungs (U.S. EPA, 1996). A single acute exposure (1-4 hours) of humans to moderate concentrations of O₃ (200-600 ppb) while exercising at moderate to heavy intensities resulted in a number of cellular and biochemical changes in the lung, including inflammation characterized by increased numbers of PMNs, increased permeability of the epithelial lining of the respiratory tract, cell damage, and production of proinflammatory molecules (i.e., cytokines and prostaglandins, U.S. EPA, 2006). A meta-analysis of 21 controlled human exposure studies (Mudway and Kelly, 2004) using varied experimental protocols (80-600 ppb O₃ exposures; 1-6.6 hours exposure duration; light to heavy exercise; bronchoscopy at 0-24 hours post-O₃ exposure) reported that PMN influx in healthy subjects is linearly associated with total O₃ dose. Animal toxicological studies also provided evidence for increases in inflammation and permeability in rabbits at levels as low as 100 ppb O₃ (Section 2.5.3.1, ISA, U.S. EPA, 2013).

Several studies, including one published since the last review (Alexis et al., 2010), have reported O₃-induced increases in PMN influx and permeability following exposures at or above 80 ppb (Alexis et al., 2010; Peden et al., 1997; Devlin et al., 1991), and eosinophilic inflammation following exposures at or above 160 ppb (Scannell et al., 1996; Peden et al., 1997; Hiltermann et al., 1999; Vagaggini et al., 2002). In addition, one recent controlled human exposure study has reported O₃-induced PMN influx following exposures of healthy adults to O₃ concentrations of 60 ppb (Kim et al., 2011), the lowest concentration at which inflammatory responses have been evaluated in human studies.

As with FEV₁ responses to O₃, inflammatory responses to O₃ are generally reproducible within individuals, with some individuals experiencing more severe O₃-induced airway inflammation than indicated by group averages (Holz et al., 2005; Holz et al., 1999). Unlike O₃-induced decrements in lung function, which are attenuated following repeated exposures over several days (U.S. EPA, 2013, section 6.2.1.1), some markers of O₃-induced inflammation and tissue damage remain elevated during repeated exposures, indicating ongoing damage to the respiratory system (U.S. EPA, 2013, section 6.2.3.1, p. 6-81).

Most controlled human exposure studies have reported that asthmatics experience larger O₃-induced inflammatory responses than non-asthmatics. Specifically, asthmatics exposed to 200 ppb O₃ for 4-6 hours with exercise show significantly more neutrophils in bronchoalveolar lavage fluid (BALF) than similarly exposed healthy individuals (Scannell et al., 1996; Basha et al., 1994). Bosson et al. (2003) reported significantly greater expression of a variety of pro-inflammatory cytokines in asthmatics, compared to healthy subjects, following exposure to 200 ppb O₃ for 2 hours. In addition, research available in the last review, combined with a recent study newly available in this review, indicates that pretreatment of asthmatics with corticosteroids can prevent the O₃-induced inflammatory response in induced sputum, though

pretreatment did not prevent FEV₁ decrements (Vagaggini et al., 2001; 2007). In contrast, Stenfors et al. (2002) did not detect a difference in the O₃-induced increases in neutrophil numbers between 15 subjects with mild asthma and 15 healthy subjects by bronchial wash at the 6 hours postexposure time point, although the neutrophil increase in the asthmatic group was on top of an elevated baseline.

In people with allergic airway disease, including people with rhinitis and asthma, evidence available in the last review indicated that proinflammatory mediators also cause accumulation of eosinophils in the airways (Jorres et al., 1996; Peden et al., 1995 and 1997; Frampton et al., 1997; Hiltermann et al., 1999; Holz et al., 2002; Vagaggini et al., 2002). The eosinophil, which increases inflammation and allergic responses, is the cell most frequently associated with exacerbations of asthma (75 CFR 2969, January 19, 2010).

Studies reporting inflammatory responses and markers of lung injury have clearly demonstrated that there is important variation in the responses of exposed subjects (75 FR 2953, January 19, 2010). Some individuals also appear to be intrinsically more susceptible to increased inflammatory responses from O₃ exposure (Holz et al., 2005). In healthy adults exposed to each 80 and 100 ppb O₃, Devlin et al. (1991) observed group average increases in neutrophilic inflammation of 2.1- and 3.8-fold, respectively. However, there was a 20-fold range in inflammatory responses between individuals at both concentrations. Relative to an earlier, similar study conducted at 400 ppb (Koren et al., 1989), Devlin et al. (1991) noted that although some of the study population showed little or no increase in inflammatory and cellular injury indicators analyzed after exposures to lower levels of O₃ (i.e., 80 and 100 ppb), others had changes that were as large as those seen when subjects were exposed to 400 ppb O₃. The data suggest that as a whole the healthy population, on average, may have small inflammatory responses to near-ambient levels of O₃, though there may be a substantial subpopulation that is very sensitive to low levels of O₃. Devlin et al. (1991) expressed the view that “susceptible subpopulations such as the very young, elderly, and people with pulmonary impairment or disease may be even more affected.”

A number of studies report that O₃ exposures increase epithelial permeability. Increased BALF protein, suggesting O₃-induced changes in epithelial permeability, has been reported at 1 hour and 18 hours postexposure (Devlin et al., 1997; Balmes et al., 1996). A meta-analysis of results from 21 publications (Mudway and Kelly, 2004) for varied experimental protocols (80-600 ppb O₃; 1-6.6 hours duration; light to heavy exercise; bronchoscopy at 0-24 hours post-O₃ exposure; healthy subjects), showed that increased BALF protein is associated with total inhaled O₃ dose (i.e., the product of O₃ concentration, exposure duration, and \dot{V}_E). As noted in the 2009 PM ISA (U.S. EPA, 2009), it has been postulated that changes in permeability associated with acute inflammation may provide increased access of inhaled antigens, particles, and other

inhaled substances deposited on lung surfaces to the smooth muscle, interstitial cells, immune cells underlying the epithelium, and the blood (U.S. EPA, 2013, sections 5.3.4, 5.3.5). Animal toxicology studies have provided some support for this hypothesis (Adamson and Prieditis, 1995; Chen et al., 2006), though these studies did not specifically evaluate O₃ exposures (U.S. EPA, 2009). Because of this potentially increased access, it has been postulated that increases in epithelial permeability following O₃ exposure might lead to increases in airway responsiveness to specific and nonspecific agents. In a recent study, Que et al. (2011) investigated this hypothesis in healthy young adults (83M, 55 F) exposed to 220 ppb O₃ for 2.25 hours (alternating 15 min periods of rest and brisk treadmill walking). As has been observed for FEV₁ responses, within-individual changes in permeability were correlated between sequential O₃ exposures, indicating intrinsic differences among individuals in susceptibility to epithelial damage following O₃ exposures. However, increases in epithelial permeability at 1 day post-O₃ exposure were not correlated with changes in airway responsiveness assessed 1 day post-O₃ exposure. The authors concluded that changes in epithelial permeability is relatively constant over time in young healthy adults, although changes in permeability and AHR appear to be mediated by different physiologic pathways.

The limited epidemiologic evidence reviewed in the 2006 O₃ AQCD (U.S. EPA, 2006) demonstrated an association between short-term increases in ambient O₃ concentrations and airways inflammation in children (1-hour max O₃ of approximately 100 ppb). In the 2006 O₃ AQCD (U.S. EPA, 2006), there was limited evidence for increases in nasal lavage levels of inflammatory cell counts and molecules released by inflammatory cells (i.e., eosinophilic cationic protein, and myeloperoxidases). Since 2006, as a result of the development of less invasive methods, there has been a large increase in the number of studies assessing ambient O₃-associated changes in airway inflammation and oxidative stress, the types of biological samples collected, and the types of indicators. Most of these recent studies have evaluated biomarkers of inflammation or oxidative stress in exhaled breath, nasal lavage fluid, or induced sputum (U.S. EPA, 2013, section 6.2.3.2). These recent studies form a larger database to establish coherence with findings from controlled human exposure and animal studies that have measured the same or related biological markers. Additionally, results from these studies provide further biological plausibility for the associations observed between ambient O₃ concentrations and respiratory symptoms and asthma exacerbations.

A number of epidemiologic studies provide evidence that short-term increases in ambient O₃ exposure increase pulmonary inflammation and oxidative stress in children, including those with asthma (Sienra-Monge et al., 2004; Barraza-Villarreal et al., 2008; Romieu et al., 2008;

Berhane et al., 2011). Multiple studies examined and found increases in exhaled NO (eNO)²³ (Berhane et al., 2011; Khatri et al., 2009; Barraza-Villarreal et al., 2008). In some studies of subjects with asthma, increases in ambient O₃ concentration at the same lag were associated with both increases in pulmonary inflammation and respiratory symptoms (Khatri et al., 2009; Barraza-Villarreal et al., 2008). Although more limited in number, epidemiologic studies also found associations with cytokines such as IL-6 or IL-8 (Barraza-Villarreal et al., 2008; Sienna-Monge et al., 2004), eosinophils (Khatri et al., 2009), antioxidants (Sienna-Monge et al., 2004), and indicators of oxidative stress (Romieu et al., 2008) (ISA, Section 6.2.3.2, U.S. EPA, 2013). Because associations with inflammation were attenuated with higher antioxidant intake the study by Sienna-Monge et al. (2004) provides additional evidence that inhaled O₃ is likely to be an important source of reactive oxygen species in airways and/or may increase pulmonary inflammation via oxidative stress-mediated mechanisms among all age groups. Limitations in some recent studies have contributed to inconsistent results in adults (U.S. EPA, 2013, section 6.2.3.2).

Exposure to ambient O₃ on multiple days can result in larger increases in pulmonary inflammation and oxidative stress, as discussed in section 6.2.3.2 of the ISA (U.S. EPA, 2013). In studies that examined multiple O₃ lags, multiday averages of 8-hour maximum or 8-hour average concentrations were associated with larger increases in pulmonary inflammation and oxidative stress (Berhane et al., 2011; Delfino et al., 2010a; Sienna-Monge et al., 2004), consistent with controlled human exposure (U.S. EPA, 2013, section 6.2.3.1) and animal studies (U.S. EPA, 2013, section 6.2.3.3) reporting that some markers of pulmonary inflammation remain elevated with O₃ exposures repeated over multiple days. Evidence from animal toxicological studies also clearly indicates that O₃ exposures result in damage and inflammation in the lung (ISA, Section 5.3, U.S. EPA, 2013). In the few studies that evaluated the potential for confounding, O₃ effect estimates were not confounded by temperature or humidity, and were robust to adjustment for PM_{2.5} or PM₁₀ (Barraza-Villarreal et al., 2008; Romieu et al., 2008; Sienna-Monge et al., 2004).

In conclusion, a relatively small number of controlled human exposure studies evaluating O₃-induced airway inflammation have become available since the last review. For purposes of reviewing the current O₃ NAAQS, the most important of these recent studies reported a statistically significant increase in airway inflammation in healthy adults at moderate exertion following exposures to 60 ppb O₃, the lowest concentration that has been evaluated for inflammation. In addition, a number of recent epidemiologic studies report O₃-associated

²³ Exhaled NO has been shown to be a useful biomarker for airway inflammation in large population-based studies (ISA, U.S. EPA, 2013, Section 7.2.4).

increases in markers of pulmonary inflammation, particularly in children. Thus, recent studies continue to support the evidence for airway inflammation and injury that was available in previous reviews, with new evidence for such effects following exposures to lower concentrations than had been evaluated previously.

Airway Hyperresponsiveness

Airway hyperresponsiveness (AHR) refers to a condition in which the conducting airways undergo enhanced bronchoconstriction in response to a variety of stimuli. Airway hyperresponsiveness is an important consequence of exposure to ambient O₃ because its presence reflects a change in airway smooth muscle reactivity, and indicates that the airways are predisposed to narrowing upon inhalation of a variety of ambient stimuli including specific triggers (i.e., allergens) and nonspecific triggers (e.g., SO₂, and cold air). 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. Standards for airway responsiveness testing have been developed for the clinical laboratory (ATS, 2000), although variation in the methodology for administering the bronchoconstricting agent may affect the results (Cockcroft et al., 2005). There is a wide range of airway responsiveness in people without asthma, and responsiveness is influenced by a number of factors, including cigarette smoke, pollutant exposures, respiratory infections, occupational exposures, and respiratory irritants. Dietary antioxidants have been reported to attenuate O₃-induced bronchial hyperresponsiveness in people with asthma (Trenga et al., 2001).

Evidence for airway hyperresponsiveness following O₃ exposures is derived primarily from controlled human exposure and toxicological studies (U.S. EPA, 2013, section 6.2.2). Airway responsiveness is often quantified by measuring changes in pulmonary function following the inhalation of an aerosolized allergen or a nonspecific bronchoconstricting agent (e.g., methacholine), or following exposure to a bronchoconstricting stimulus such as cold air. In the last review, controlled human exposure studies of mostly adults (≥ 18 years of age) had shown that exposures to O₃ concentrations at or above 80 ppb increase airway responsiveness, as indicated by a reduction in the concentration of specific (e.g., ragweed) and non-specific (e.g., methacholine) agents required to produce a given reduction in lung function (e.g., as measured by FEV₁ or specific airway resistance) (U.S. EPA, 2013, section 6.2.2.1). This O₃-induced AHR has been reported to be dose-dependent (Horstman et al., 1990). Animal toxicology studies have reported O₃-induced airway hyperresponsiveness in a number of species, with some rat strains exhibiting hyperresponsiveness following 4-hour exposures to O₃ concentrations as low as 50 ppb (Depuydt et al., 1999). Since the last review, there have been relatively few new controlled human exposure and animal toxicology studies of O₃ and airway hyperresponsiveness, and no

new studies have evaluated exposures to O₃ concentrations at or below 80 ppb (U.S. EPA, 2013, section 6.2.2.1)

Airway hyperresponsiveness is linked with the accumulation and/or activation of eosinophils in the airways of asthmatics, which is followed by production of mucus and a late-phase asthmatic response (75 FR 2970, January 19, 2010). In a study of 16 intermittent asthmatics, Hiltermann et al. (1999) found that there was a significant inverse correlation between the O₃-induced change in the percentage of eosinophils in induced sputum and the concentration of methacholine causing a 20% decrease in FEV₁. Hiltermann et al. (1999) concluded that the results point to the role of eosinophils in O₃-induced airway hyperresponsiveness. Increases in O₃-induced nonspecific airway responsiveness incidence and duration could have important clinical implications for children and adults with asthma, such as exacerbations of their disease.

Airway hyperresponsiveness after O₃ exposure appears to resolve more slowly than changes in FEV₁ or respiratory symptoms (Folinsbee and Hazucha, 2000). Studies suggest that O₃-induced AHR usually resolves 18 to 24 hours after exposure, but may persist in some individuals for longer periods (Folinsbee and Hazucha, 1989). Furthermore, in studies of repeated exposure to O₃, changes in AHR tend to be somewhat less susceptible to attenuation with consecutive exposures than changes in FEV₁ (Gong et al., 1997; Folinsbee et al., 1994; Kulle et al., 1982; Dimeo et al., 1981) (U.S. EPA, 2013, section 6.2.2). In animal studies a 3-day continuous exposure resulted in attenuation of O₃-induced airway hyperresponsiveness (Johnston et al., 2005) while repeated exposures for 2 hours per day over 10 days did not (Chhabra et al., 2010), suggesting that attenuation could be lost when repeated exposures are interspersed with periods of rest (U.S. EPA, 2013, section 6.2.2.2).

Increases in airway responsiveness do not appear to be strongly associated with decrements in lung function or increases in symptoms (Aris et al., 1995). Recently, Que et al. (2011) assessed methacholine responsiveness in healthy young adults (83M, 55 F) one day after exposure to 220 ppb O₃ and filtered air for 2.25 hours (alternating 15 minute periods of rest and brisk treadmill walking). Increases in airways responsiveness at 1 day post-O₃ exposure were not correlated with FEV₁ responses immediately following the O₃ exposure or with changes in epithelial permeability assessed 1-day post-O₃ exposure. This indicates that airway hyperresponsiveness also appears to be mediated by a differing physiologic pathway.

As mentioned above, in addition to human subjects a number of species, including nonhuman primates, dogs, cats, rabbits, and rodents, have been used to examine the effect of O₃ exposure on airway hyperresponsiveness (see Table 6-14, (U.S. EPA, 1996) of the 1996 O₃ AQCD and Annex Table AX5-12 on page AX5-36 (U.S. EPA, 2006) of the 2006 O₃ AQCD). A body of animal toxicology studies, including some recent studies conducted since the last review,

provides support for the O₃-induced AHR reported in humans (U.S. EPA, 2013, section 6.2.2.2). Although most of these studies evaluated O₃ concentrations above those typically found in ambient air in cities in the United States (i.e., most studies evaluated O₃ concentrations of 100 ppb or greater), one study reported that a very low exposure concentration (50 ppb for 4 hours) induced AHR in some rat strains (Depuydt et al., 1999). Additional recent rodent studies reported O₃-induced AHR following exposures to O₃ concentrations from 100 to 500 ppb (Johnston et al., 2005; Chhabra et al., 2010; Larsen et al., 2010). In characterizing the relevance of these exposure concentrations, the ISA noted that a study using radiolabeled O₃ suggests that even very high O₃ exposure concentrations in rodents could be equivalent to much lower exposure concentrations in humans. Specifically, a 2000 ppb (2 ppm) O₃ exposure concentration in resting rats was reported to be roughly equivalent to a 400 ppb exposure concentration in exercising humans (Hatch et al., 1994). Given this relationship, the ISA noted that animal data obtained in resting conditions could underestimate the risk of effects for humans (U.S. EPA, 2013, section 2.4, p. 2-14).

The 2006 AQCD (U.S. EPA, 2006, p. 6-34) concluded that spirometric responses to O₃ are independent of inflammatory responses and markers of epithelial injury or integrity (Balmes et al., 1996; Blomberg et al., 1999; Torres et al., 1997). Significant inflammatory responses to O₃ exposures that did not elicit significant spirometric responses have been reported (Holz et al., 2005). A recent study (Que et al., 2011) indicates that airway hyper-responsiveness also appears to be mediated by a differing physiologic pathway. These results from controlled human exposure studies indicate that sub-populations of healthy study subjects consistently experience larger than average lung function decrements, greater than average inflammatory responses and pulmonary injury as expressed by increased epithelial permeability, and greater than average airway responsiveness, and that these effects are mediated by apparently different physiologic pathways. Except for lung function decrements, we do not have the concentration- or exposure-response function information about the other, potentially more sensitive,²⁴ clinical endpoints (i.e., inflammation, increased epithelial permeability, airway hyperresponsiveness) that would allow us to quantitatively estimate the size of the population affected and the magnitude of their responses. Moreover, some uncertainties about the exact physiological pathways underlying these endpoints prevents us from knowing whether the exaggerated responses are distributed in sub-populations evenly across the population, or may be clustered with more than one type of exaggerated response in particular sub-populations, or both.

²⁴ CASAC noted that "...[W]hile measures of FEV₁ are quantitative and readily obtainable in humans, they are not the only measures — and perhaps not the most sensitive measures — of the adverse health effects induced by ozone exposure." (Henderson, 2006).

In summary, a strong body of controlled human exposure and animal toxicological studies, most of which were available in the last review of the O₃ NAAQS, report O₃-induced airway hyperresponsiveness after either acute or repeated exposures (U.S. EPA, 2013, section 6.2.2.2). People with asthma often exhibit increased airway responsiveness at baseline relative to healthy controls, and they can experience further increases in responsiveness following exposures to O₃. Studies reporting increased airway responsiveness after O₃ exposure contribute to a plausible link between ambient O₃ exposures and increased respiratory symptoms in asthmatics, and increased hospital admissions and emergency department visits for asthma (U.S. EPA, 2013, section 6.2.2.2).

Respiratory Symptoms and Medication Use

Because respiratory symptoms are associated with adverse outcomes such as limitations in activity, and are the primary reason for people with asthma to use quick relief medication and seek medical care, studies evaluating the link between O₃ exposures and such symptoms allow a more direct characterization of the clinical and public health significance of ambient O₃ exposure than measures of lung function decrements and pulmonary inflammation. Controlled human exposure and toxicological studies have described modes of action through which short-term O₃ exposures may increase respiratory symptoms by demonstrating O₃-induced airway hyperresponsiveness (U.S. EPA, 2013, section 6.2.2) and pulmonary inflammation (U.S. EPA, 2013, section 6.2.3).

The link between subjective respiratory symptoms and O₃ exposures has been evaluated in both controlled human exposure and epidemiologic studies, and the link with medication use has been evaluated in epidemiologic studies. In the last review, several controlled human exposure studies reported respiratory symptoms following exposures to O₃ concentrations at or above 80 ppb. In addition, one study reported such symptoms following exposures to 60 ppb O₃, though the increase was not statistically different from filtered air controls. Epidemiologic studies reported associations between ambient O₃ and respiratory symptoms and medication use in a variety of locations and populations, including asthmatic children living in U.S. cities. In the current review, additional controlled human exposure studies have evaluated respiratory symptoms following exposures to O₃ concentrations below 80 ppb and recent epidemiologic studies have evaluated associations with respiratory symptoms and medication use (U.S. EPA, 2013, sections 6.2.1, 6.2.4).

In controlled human exposure studies available in the last review as well as newly available studies, statistically significant increases in respiratory symptoms have been consistently reported in healthy adult volunteers engaged in intermittent, moderate exertion following 6.6 hour exposures to average O₃ concentrations at or above 80 ppb (Adams, 2003; Adams, 2006; Schelegle et al., 2009). Such symptoms have been reported to increase with

increasing O₃ exposure concentrations, duration of exposure, and activity level (McDonnell et al., 1999). For example, in a study available during the last review, Adams (2006) reported an increase in respiratory symptoms in healthy adults during a 6.6 hour exposure protocol with an average O₃ exposure concentration of 60 ppb. This increase was significantly different from initial respiratory symptoms, but not from filtered air controls. Two recent controlled human exposure studies that have become available since the last review did not report statistically significant increases in respiratory symptoms following exposures of healthy adults to 60 ppb O₃ (Schelegle et al., 2009; Kim et al., 2011). A recent study by Schelegle et al. (2009) did report a statistically significant increase in respiratory symptoms in healthy adults following 6.6 hour exposures to an average O₃ concentration of 70 ppb. The findings for O₃-induced respiratory symptoms in controlled human exposure studies, and the evidence integrated across disciplines describing underlying modes of action, provide biological plausibility for epidemiologic associations observed between short-term increases in ambient O₃ concentration and increases in respiratory symptoms (U.S. EPA, 2013, section 6.2.4).

In epidemiologic studies of respiratory symptoms, data typically are collected by having subjects (or their parents) record symptoms and medication use in a diary without direct supervision by study staff. Several limitations of symptom reports are well recognized, as described in the ISA (U.S. EPA, 2013, section 6.2.4). Nonetheless, symptom diaries remain a convenient tool to collect individual-level data from a large number of subjects and allow modeling of associations between daily changes in O₃ concentration and daily changes in respiratory morbidity over multiple weeks or months. Importantly, many of the limitations in these studies are sources of random measurement error that can bias effect estimates to the null or increase the uncertainty around effect estimates (U.S. EPA, 2013, Section 6.2.4). Because respiratory symptoms are associated with limitations in activity and daily function and are the primary reason for using medication and seeking medical care, the evidence is directly coherent with the associations consistently observed between increases in ambient O₃ concentration and increases in asthma emergency department visits, discussed below (U.S. EPA, 2013, Section 6.2.4).

Most epidemiologic studies of O₃ and respiratory symptoms and medication use have been conducted in children and/or adults with asthma, with fewer studies, and less consistent results, in non-asthmatic populations (U.S. EPA, 2013, section 6.2.4). The 2006 AQCD (U.S. EPA, 2006, U.S. EPA, 2013, section 6.2.4) concluded that the collective body of epidemiologic evidence indicated that short-term increases in ambient O₃ concentrations are associated with increases in respiratory symptoms in children with asthma. A large body of single-city and single-region studies of asthmatic children provides consistent evidence for associations between

short-term increases in ambient O₃ concentrations and increased respiratory symptoms and asthma medication use in children with asthma (U.S. EPA, 2013, Figure 6-12, Table 6-20).

Methodological differences among studies make comparisons across recent multicity studies of respiratory symptoms difficult. Because of fewer person-days of data (Schildcrout et al., 2006) or examination of 19-day averages of ambient O₃ concentrations (O'Connor et al., 2008), the ISA did not give greater weight to results from recent multicity studies than results from single-city studies (U.S. EPA, 2013, section 6.2.4.5). While evidence from the few available U.S. multicity studies is less consistent (O'Connor et al., 2008; Schildcrout et al., 2006; Mortimer et al., 2002), the overall body of epidemiologic evidence with respect to the association between exposure to O₃ and respiratory symptoms in asthmatic children remains compelling (U.S. EPA, 2013, section 6.2.4.1). Findings from a small body of studies indicate that O₃ is also associated with increased respiratory symptoms in adults with asthma (Khatri et al., 2009; Feo Brito et al., 2007; Ross et al., 2002) (U.S. EPA, 2013, section 6.2.4.2).

Available evidence indicates that O₃-associated increases in respiratory symptoms are not confounded by temperature, pollen, or copollutants (primarily PM) (U.S. EPA, 2013, section 6.2.4.5; Table 6-25; Romieu et al., 1996; Romieu et al., 1997; Thurston et al., 1997; Gent et al., 2003). However, identifying the independent effects of O₃ in some studies was complicated due to the high correlations observed between O₃ and PM or different lags and averaging times examined for copollutants. Nonetheless, the ISA noted that the robustness of associations in some studies of individuals with asthma, combined with findings from controlled human exposure studies for the direct effects of O₃ exposure, provide substantial evidence supporting the independent effects of short-term ambient O₃ exposure on respiratory symptoms (U.S. EPA, 2013, section 6.2.4.5).

Epidemiologic studies of medication use have reported associations with 1-hour maximum O₃ concentrations and with multiday average O₃ concentrations (Romieu et al., 2006; Just et al., 2002). Some studies reported O₃ associations for both respiratory symptoms and asthma medication use (Escamilla-Nuñez et al., 2008; Romieu et al., 2006; Schildcrout et al., 2006; Jalaludin et al., 2004; Romieu et al., 1997; Thurston et al., 1997) while others reported associations for either respiratory symptoms or medication use (Romieu et al., 1996; Rabinovitch et al., 2004; Just et al., 2002; Ostro et al., 2001).

In summary, both controlled human exposure and epidemiologic studies have reported respiratory symptoms attributable to short-term O₃ exposures. In the last review, the majority of the evidence from controlled human exposure studies in young, healthy adults was for symptoms following exposures to O₃ concentrations at or above 80 ppb. Although studies that have become available since the last review have not reported respiratory symptoms in young, healthy adults following exposures with moderate exertion to 60 ppb, one recent study has reported increased

symptoms in young, healthy adults while at moderate exertion following exposures to O₃ concentrations as low as 70 ppb.²⁵ As was concluded in the 2006 O₃ AQCD (U.S. EPA, 2006, 1996), the collective body of epidemiologic evidence indicates that short-term increases in ambient O₃ concentration are associated with increases in respiratory symptoms in children with asthma (U.S. EPA, 2013, section 6.2.4). Recent studies of respiratory symptoms and medication use, primarily in asthmatic children, add to this evidence. In a smaller body of studies, increases in ambient O₃ concentration were associated with increases in respiratory symptoms in adults with asthma.

Lung Host Defense

The mammalian respiratory tract has a number of closely integrated defense mechanisms that, when functioning normally, provide protection from the potential health effects of exposures to a wide variety of inhaled particles and microbes. These defense mechanisms include mucociliary clearance, alveolobronchiolar transport mechanism, alveolar macrophages²⁶, and adaptive immunity²⁷ (U.S. EPA, 2013, section 6.2.5). The previous O₃ AQCD (U.S. EPA, 2006) concluded that animal toxicological studies provided evidence that acute exposure to O₃ concentrations as low as 100 to 500 ppb can increase susceptibility to infectious diseases due to modulation of these lung host defenses. This conclusion was based in large part on animal studies of alveolar macrophage functioning and mucociliary clearance (U.S. EPA, 2013, section 6.2.5).

With regard to alveolar macrophage functioning, the previous O₃ AQCD (U.S. EPA, 2006) concluded that short periods of O₃ exposure can cause a reduction in the number of free alveolar macrophages available for pulmonary defense, and that these alveolar macrophages are more fragile, less able to engulf particles (i.e., phagocytic), and exhibit decreased lysosomal²⁸ enzyme activities (U.S. EPA, 2013, section 6.2.5). These conclusions were based largely on studies conducted in animals exposed for several hours up to several weeks to O₃ concentrations from 100 to 250 ppb (Hurst et al., 1970; Driscoll et al., 1987; Cohen et al., 2002). Consistent with the animal evidence, a controlled human exposure study available in the last review had reported decrements in the ability of alveolar macrophages to phagocytize yeast following exposures of healthy volunteers to O₃ concentrations of 80 and 100 ppb for 6.6-hour during

²⁵As noted above, for the 70 ppb exposure concentration Schelegle et al. (2009) reported that the actual mean exposure concentration was 72 ppb.

²⁶ Phagocytic white blood cells within the alveoli of the lungs that ingest inhaled particles.

²⁷ The adaptive immune system, is also known as the acquired immune system. Acquired immunity creates immunological memory after an initial response to a specific pathogen, leading to an enhanced response to subsequent encounters with that same pathogen.

²⁸ Lysosomes are cellular organelles that contain acid hydrolase enzymes that break down waste materials and cellular debris.

moderate exercise (Devlin et al., 1991). Integrating the animal study results with human exposure evidence available in the 1996 Criteria Document, the 2006 Criteria Document concluded that available evidence indicates that short-term O₃ exposures have the potential to impair host defenses in humans, primarily by interfering with alveolar macrophage function. Any impairment in alveolar macrophage function may lead to decreased clearance of microorganisms or nonviable particles. Compromised alveolar macrophage functions in asthmatics may increase their susceptibility to other O₃ effects, the effects of particles, and respiratory infections (EPA, 2006, p. 8–26).

With regard to mucociliary clearance, in the last review a number of studies conducted in different animal species had reported morphological damage to the cells of the tracheobronchial tree from acute and sub-chronic exposure to O₃ concentrations at or above 200 ppb. The cilia were either completely absent or had become noticeably shorter or blunt. In general, functional studies of mucociliary transport had observed a delay in particle clearance soon after acute exposure, with decreased clearance more evident at higher doses (1 ppm) (U.S. EPA, 2013, section 6.2.5.1).

Alveolobronchiolar transport mechanisms refers to the transport of particles deposited in the deep lung (alveoli) which may be removed either up through the respiratory tract (bronchi) by alveolobronchiolar transport or through the lymphatic system. The pivotal mechanism of alveolobronchiolar transport involves the movement of alveolar macrophages with ingested particles to the bottom of the conducting airways. These airways are lined with ciliated epithelial cells and cells that produce mucous, which surrounds the macrophages. The ciliated epithelial cells move the mucous packets up the respiratory tract, hence the term “mucociliary escalator.” Although some studies show reduced tracheobronchial clearance after O₃ exposure, alveolar clearance of deposited material is accelerated, presumably due to macrophage influx, which in itself can be damaging.

With regard to adaptive immunity, a limited number of epidemiologic studies have examined associations between O₃ exposure and hospital admissions or ED visits for respiratory infection, pneumonia, or influenza. Results have been mixed, and in some cases conflicting (U.S. EPA, 2013, Sections 6.2.7.2 and 6.2.7.3). With the exception of influenza, it is difficult to ascertain whether cases of respiratory infection or pneumonia are of viral or bacterial etiology. A recent study that examined the association between O₃ exposure and respiratory hospital admissions in response to an increase in influenza intensity did observe an increase in respiratory hospital admissions (Wong et al., 2009), but information from toxicological studies of O₃ and viral infections is ambiguous.

In summary, relatively few studies conducted since the last review have evaluated the effects of O₃ exposures on lung host defense. When the available evidence is taken as a whole,

the ISA concludes that acute O₃ exposures impair the host defense capability of animals, primarily by depressing alveolar macrophage function and perhaps also by decreasing mucociliary clearance of inhaled particles and microorganisms. Coupled with limited evidence from controlled human exposure studies, this suggests that humans exposed to O₃ could be predisposed to bacterial infections in the lower respiratory tract (EPA, 2013, section 6.2.5.5). The seriousness of such infections may depend on how quickly bacteria develop virulence factors and how rapidly PMNs are mobilized to compensate for the deficit in alveolar macrophage function.

Allergic and Asthma-Related Responses

Effects resulting from combined exposures to O₃ and allergens have been studied in a variety of animal species, generally as models of experimental asthma. Pulmonary function and AHR in animal models of asthma are discussed in detail in Section 6.2.1.3 and Section 6.2.2.2, respectively, in the ISA (U.S. EPA, 2013). Studies of allergic and asthma-related responses are discussed in detail in sections 5.3.6 and 6.2.6 of the ISA (U.S. EPA, 2013).

Evidence available in the last review indicates that O₃ exposure skews immune responses toward an allergic phenotype. For example, Gershwin et al. (1981) reported that O₃ (800 and 500 ppb for 4 days) exposure caused a 34-fold increase in the number of IgE (allergic antibody)-containing cells in the lungs of mice. In general, the number of IgE-containing cells correlated positively with levels of anaphylactic sensitivity. In humans, allergic rhinoconjunctivitis symptoms are associated with increases in ambient O₃ concentrations (Riediker et al., 2001). Controlled human exposure studies have observed O₃-induced changes indicating allergic skewing. Airway eosinophils, which are white blood cells that participate in allergic disease and inflammation, were observed to increase in volunteers with atopy²⁹ and mild asthma (Peden et al., 1997). In a more recent study, expression of IL-5, a cytokine involved in eosinophil recruitment and activation, was increased in subjects with atopy but not in healthy subjects (Hernandez et al., 2010). Epidemiologic studies describe associations between eosinophils in both short- (U.S. EPA, 2013, Section 6.2.3.2) and long-term (U.S. EPA, 2013, Section 7.2.5) O₃ exposure, as do chronic exposure studies in non-human primates. Collectively, findings from these studies suggest that O₃ can induce or enhance certain components of allergic inflammation in individuals with allergy or allergic asthma.

Evidence available in the last review indicates that ozone may also increase AHR to specific allergen triggers (75 FR 2970, January 19, 2010). Two studies (Jörres et al., 1996; Holz et al., 2002) observed increased airway responsiveness to O₃ exposure with bronchial allergen

²⁹ Atopy is a predisposition toward developing certain allergic hypersensitivity reactions. A person with atopy typically presents with one or more of the following: eczema (atopic dermatitis), allergic rhinitis (hay fever), allergic conjunctivitis, or allergic asthma.

challenge in subjects with preexisting allergic airway disease. Ozone-induced exacerbation of airway responsiveness persists longer and attenuates more slowly than O₃-induced lung function decrements and respiratory symptom responses and can have important clinical implications for asthmatics.

Animal toxicology studies indicate that O₃ enhances inflammatory and allergic responses to allergen challenge in sensitized animals. In addition to exacerbating existing allergic responses, toxicology studies indicate that O₃ can also act as an adjuvant to produce sensitization in the respiratory tract. Along with its pro-allergic effects (inducing or enhancing certain components of allergic inflammation in individuals with allergy or allergic asthma), O₃ could also make airborne allergens more allergenic. When combined with NO₂, O₃ has been shown to enhance nitration of common protein allergens, which may increase their allergenicity Franze et al. (2005).

Hospital Admissions and Emergency Department Visits

The 2006 O₃ AQCD evaluated numerous studies of respiratory-related emergency department visits and hospital admissions. These were primarily time-series studies conducted in the U.S., Canada, Europe, South America, Australia, and Asia. Based on such studies, the 2006 O₃ AQCD concluded that “the overall evidence supports a causal relationship between acute ambient O₃ exposures and increased respiratory morbidity resulting in increased ED visits and [hospital admissions] during the warm season³⁰” (U.S. EPA, 2006). This conclusion was “strongly supported by the human clinical, animal toxicologic[al], and epidemiologic evidence for [O₃-induced] lung function decrements, increased respiratory symptoms, airway inflammation, and airway hyperreactivity” (U.S. EPA, 2006).

The results of recent studies largely support the conclusions of the 2006 O₃ AQCD (U.S. EPA, 2013, section 6.2.7). Since the completion of the 2006 O₃ AQCD, relatively fewer studies conducted in the U.S., Canada, and Europe have evaluated associations between short-term O₃ concentrations and respiratory hospital admissions and emergency department visits, with a growing number of studies conducted in Asia. This epidemiologic evidence is summarized in Appendix 3A and discussed in detail in the ISA (U.S. EPA, 2013, section 6.2.7).

In considering this body of evidence, the ISA focused primarily on multicity studies because they examine associations with respiratory-related hospital admissions and emergency department visits over large geographic areas using consistent statistical methodologies (U.S. EPA, 2013, section 6.2.7.1). The ISA also focused on single-city studies that encompassed a large number of daily hospital admissions or emergency department visits, included long study-

³⁰Epidemiologic associations for O₃ are more robust during the warm season than during cooler months (e.g., smaller measurement error, less potential confounding by copollutants). Rationale for focusing on warm season epidemiologic studies for O₃ can be found at 72 FR 37838-37840.

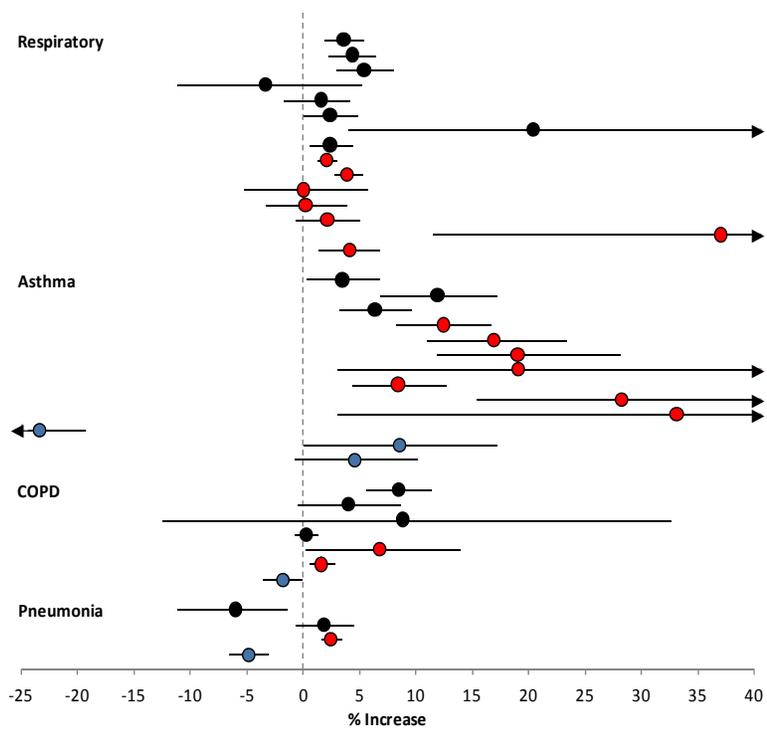
durations, were conducted in locations not represented by the larger studies, or examined population-specific characteristics that may increase the risk of O₃-related health effects but were not evaluated in the larger studies (U.S. EPA, 2013, section 6.2.7.1). When examining the association between short-term O₃ exposure and respiratory health effects that require medical attention, the ISA distinguishes between hospital admissions and emergency department visits because it is likely that a small percentage of respiratory emergency department visits will be admitted to the hospital; therefore, respiratory emergency department visits may represent potentially less serious, but more common outcomes (U.S. EPA, 2013, section 6.2.7.1).

Several recent multicity studies (e.g., Cakmak et al., 2006; Dales et al., 2006) and a multi-continent study (Katsouyanni et al., 2009) report associations between short-term O₃ concentrations and increased respiratory-related hospital admissions and emergency department visits. These multicity studies are supported by single-city studies also reporting consistent positive associations using different exposure assignment approaches (i.e., average of multiple monitors, single monitor, population-weighted average) and averaging times (i.e., 1-hour max and 8-hour max) (U.S. EPA, 2013, sections 6.2.7.1 to 6.2.7.5). When examining cause-specific respiratory outcomes, recent studies report positive associations with hospital admissions and emergency department visits for asthma (Strickland et al., 2010; Stieb et al., 2009) and COPD (Stieb et al., 2009; Medina-Ramon et al., 2006), with more limited evidence for pneumonia (Medina-Ramon et al., 2006; Zanobetti and Schwartz, 2006). In seasonal analyses (Figure 3-2 below; U.S. EPA, 2013, Figure 6-19, Table 6-28), stronger associations were reported in the warm season or summer months (red circles), when O₃ concentrations are higher, compared to the cold season (blue circles), particularly for asthma (Strickland et al., 2010; Ito et al., 2007) and COPD (Medina-Ramon et al., 2006).³¹ The available evidence indicates that children are at greatest risk for O₃-induced respiratory effects (Silverman and Ito, 2010; Strickland et al., 2010; Mar and Koenig, 2009; Villeneuve et al., 2007; Dales et al., 2006).

Although the collective evidence across studies indicates a mostly consistent positive association between O₃ exposure and respiratory-related hospital admissions and ED visits, the magnitude of these associations may be underestimated due to behavioral modification in response to air quality forecasts (U.S. EPA, 2013, Section 4.6.6).

³¹ The study by Strickland et al. (2010) is discussed in more detail in section 3.1.4.2, below.

Study	Location	Visit Type	Age	Lag
Wong et al. (2009)	Hong Kong	HA	All	0-1
Cakmak et al. (2006)	10 Canadian cities	HA	All	1,2
Dales et al. (2006)	11 Canadian cities	HA	0-27 days	2
Orazio et al. (2009) ^a	6 Italian cities	ED	0-2	0-6
Katsouyanni et al. (2009)	APHENA-Europe	HA	65+	0-1
	APHENA-U.S.	HA	65+	0-1
	APHENA-Canada	HA	65+	DL (0-2)
	APHENA-Canada	HA	65+	DL (0-2) ^b
Darrow et al. (2009)	Atlanta	ED	All	1
Tolbert et al. (2007)	Atlanta	ED	All	0-2
Biggeri et al. (2005) ^c	8 Italian cities	HA	All	0-3
Katsouyanni et al. (2009)	APHENA-Europe	HA	65+	0-1
	APHENA-U.S.	HA	65+	0-1
	APHENA-Canada	HA	65+	DL (0-2)
	APHENA-Canada	HA	65+	DL (0-2) ^b
Stieb et al. (2009)	7 Canadian Cities	ED	All	2
Villeneuve et al. (2007)	Alberta, CAN	ED	> 2	0-2
Strickland et al. (2010)	Atlanta	ED	Children	0-2
Silverman and Ito (2010) ^d	New York	HA	All	0-1
Ito et al. (2007)	New York	ED	All	0-1
Villeneuve et al. (2007)	Alberta, CAN	ED	> 2	0-2
Mar and Koenig (2009)	Seattle, WA	ED	18+	2
Strickland et al. (2010)	Atlanta	ED	Children	0-2
Silverman and Ito (2010) ^d	New York	HA	6-18	0-1
Mar and Koenig (2009)	Seattle, WA	ED	<18	0
Ito et al. (2007)	New York	ED	All	0-1
Villeneuve et al. (2007)	Alberta, CAN	ED	> 2	0-2
Strickland et al. (2010)	Atlanta	ED	Children	0-2
Wong et al. (2009)	Hong Kong	HA	All	0-1
Stieb et al. (2009)	7 Canadian Cities	ED	All	2
Yang et al. (2006)	Vancouver	HA	65+	0-3
Medina-Ramon et al. (2006)	36 U.S. cities	HA	65+	DL (0-1)
Stieb et al. (2009) ^e	7 Canadian Cities	ED	All	NR
Medina-Ramon et al. (2006)	36 U.S. cities	HA	65+	DL (0-1)
	36 U.S. cities	HA	65+	DL (0-1)
Zanobetti and Schwartz (2006)	Boston	HA	65+	0-1
Medina-Ramon et al. (2006)	36 U.S. cities	HA	65+	DL (0-1)
	36 U.S. cities	HA	65+	DL (0-1)
	36 U.S. cities	HA	65+	DL (0-1)



Note: Effect estimates are for a 20 ppb increase in 24-hour; 30 ppb increase in 8-hour max; and 40 ppb increase in 1-hour max O₃ concentrations. HA=hospital admission; ED=emergency department. Black=All-year analysis; Red=Summer only analysis; Blue=Winter only analysis.

^a Wheeze used as indicator of lower respiratory disease.

^b APHENA-Canada results standardized to approximate IQR of 5.1 ppb for 1-h max O₃ concentrations.

^c Study included 8 cities; but of those 8, only 4 had O₃ data.

^d non-ICU effect estimates.

^e The study did not specify the lag day of the summer season estimate.

Figure 3-2. Percent increase in respiratory-related hospital admission and emergency department visits in studies that presented all-year and/or seasonal results.

Studies examining the potential confounding effects of copollutants have reported that O₃ effect estimates remained relatively robust upon the inclusion of PM and gaseous pollutants in two-pollutant models (U.S. 2013, Figure 6-20, Table 6-29). Additional studies that conducted copollutant analyses, but did not present quantitative results, also support these conclusions (Strickland et al., 2010; Tolbert et al., 2007; Medina-Ramon et al., 2006) (U.S. 2013, section 6.2.7.5).

In the last review, studies had not evaluated the concentration-response relationship between short-term O₃ exposure and respiratory-related hospital admissions and emergency department visits. A preliminary examination of this relationship in studies that have become available since the last review found no evidence of a deviation from linearity when examining the association between short-term O₃ exposure and asthma hospital admissions (U.S. EPA, 2013, page 6-157, and Silverman and Ito, 2010). In addition, an examination of the concentration-response relationship for O₃ exposure and pediatric asthma emergency department visits found no evidence of a threshold at O₃ concentrations as low as 30 ppb (for daily maximum 8-hour concentrations) (Strickland et al., 2010). However, in both studies there is uncertainty in the shape of the concentration-response curve at the lower end of the distribution of O₃ concentrations due to the low density of data in this range (U.S. 2013, page 6-157).

Respiratory Mortality

The controlled human exposure, epidemiologic, and toxicological studies discussed in section 6.2 of the ISA (U.S. EPA, 2013, section 6.2) provide strong evidence for respiratory morbidity effects, including ED visits and hospital admissions, in response to short-term O₃ exposures. Moreover, evidence from experimental studies indicates multiple potential pathways of respiratory effects from short-term O₃ exposures, which support the continuum of respiratory effects that could potentially result in respiratory-related mortality in adults (U.S. EPA, 2013, section 6.2.8). The 2006 O₃ AQCD found inconsistent evidence for associations between short-term O₃ concentrations and respiratory mortality (U.S. EPA, 2006). Although some studies reported a strong positive association between O₃ and respiratory mortality, additional studies reported small associations or no associations. New epidemiologic evidence for respiratory mortality is discussed in detail in section 6.2.8 of the ISA (U.S. EPA, 2013). The majority of recent multicity studies have reported positive associations between short-term O₃ exposures and respiratory mortality, particularly during the summer months (U.S. EPA, 2013, Figure 6-36).

Specifically, recent multicity studies from the U.S. (Zanobetti and Schwartz, 2008b), Europe (Samoli et al., 2009), Italy (Stafoggia et al., 2010), and Asia (Wong et al., 2010), as well as a multi-continent study (Katsouyanni et al., 2009), reported associations between short-term O₃ concentrations and respiratory mortality (U.S. EPA, 2013, Figure 6-37, page 6-259). With

respect to respiratory mortality, summer-only analyses were consistently positive and most were statistically significant. In all-year analyses associations were positive, but smaller in magnitude.

Of the studies evaluated, only the studies by Katsouyanni et al. (2009) and by Stafoggia et al. (2010) analyzed the potential for copollutant confounding of the O₃-respiratory mortality relationship. Based on the results of these analyses, the ISA concluded that O₃ respiratory mortality risk estimates appear to be moderately to substantially sensitive (e.g., increased or attenuated) to inclusion of PM₁₀. However, in the APHENA study (Katsouyanni et al., 2009), the mostly every-6th-day sampling schedule for PM₁₀ in the Canadian and U.S. datasets greatly reduced their sample size and limits the interpretation of these results (U.S. EPA, 2013, section 6.2.8).

In summary, recent epidemiologic studies support and reinforce the epidemiologic evidence for O₃-associated respiratory hospital admissions and emergency department visits from the last review. In addition, the evidence for associations with respiratory mortality has been strengthened considerably since the last review, with the addition of several large multicity studies. The biological plausibility of the associations reported in these studies is supported by the experimental evidence for respiratory effects.

3.1.2.2 Respiratory Effects – Long-term Exposures

- **To what extent does the currently available scientific evidence, including related uncertainties, strengthen or alter our understanding from the last review of respiratory effects attributable to long-term O₃ exposures?**

As recognized in section 3.1.2.1, “the clearest evidence for health effects associated with exposure to O₃ is provided by studies of respiratory effects” (U.S. EPA, 2013, section 1, p. 1-6). Collectively, there is a vast amount of evidence spanning several decades that supports a causal association between exposure to O₃ and a continuum of respiratory effects (U.S. EPA, 2013, section 2.5). While the majority of this evidence is derived from studies investigating short-term exposures, evidence from animal toxicological studies and recent epidemiologic evidence indicate that long-term exposures (i.e., months to years) may also be detrimental to the respiratory system. Across this evidence, particularly the epidemiologic evidence, the exposures of focus vary, often involving repeated short concentrations extending over a long period, rather than a continuous long-term exposure period.

In the 2006 O₃ AQCD, evidence was examined for relationships between long-term O₃ exposure and effects on respiratory health outcomes including declines in lung function, increases in inflammation, and development of asthma in children and adults. Animal toxicology data provided a clearer picture indicating that long-term O₃ exposure may have lasting effects.

Chronic³² exposure studies in animals have reported biochemical and morphological changes suggestive of irreversible long-term O₃ impacts on the lung. In contrast to supportive evidence from chronic animal studies, the epidemiologic studies on longer-term (annual) lung function declines, inflammation, and new asthma development remained inconclusive.

Several epidemiologic studies collectively indicated that O₃ exposure averaged over several summer months was associated with smaller increases in lung function growth in children. For longer averaging periods (annual), the analysis in the Children's Health Study (CHS) reported by Gauderman et al. (2004) provided little evidence that such long-term exposure to ambient O₃ was associated with significant deficits in the growth rate of lung function in children. Limited epidemiologic research examined the relationship between long-term O₃ exposures and inflammation. Cross-sectional studies detected no associations between long-term O₃ exposures and asthma prevalence, asthma-related symptoms or allergy to common aeroallergens in children. However, longitudinal studies provided evidence that long-term O₃ exposure influences the risk of asthma development in children and adults.

The currently available body of evidence supporting a relationship between long-term O₃ exposures and adverse respiratory health effects that is likely to be causal is discussed in detail in the ISA (EPA 2013, section 7.2). New evidence reports interactions between genetic variants and long-term O₃ exposure affect the occurrence of new-onset asthma in multi-community, U.S. cohort studies where protection by specific oxidant gene variants was restricted to children living in low O₃ communities. A new line of evidence reports a positive concentration-response relationship between first asthma hospitalization and long-term O₃ exposure. Related studies report coherent relationships between asthma severity and control, and respiratory symptoms among asthmatics and long-term O₃ exposure. There is also limited evidence for an association between long-term exposure to ambient O₃ concentrations and respiratory mortality. These studies are summarized briefly below for new-onset asthma and asthma prevalence, asthma hospital admissions and other morbidity effects, pulmonary structure and function, and respiratory mortality.

Currently available scientific evidence of the adverse health effects attributable to long-term O₃ exposures, even considering related uncertainties, is much stronger than the body of evidence available at the time of the 2008 review of the O₃ standard. The 2006 O₃ AQCD (U.S. EPA, 2006) concluded that epidemiologic studies provided no evidence of associations between long-term (annual) O₃ exposures and asthma-related symptoms, asthma prevalence, or allergy to common allergens after controlling for covariates. It found limited evidence for a relationship

³² Unless otherwise specified, the term "chronic" generally refers to an annual exposure duration for epidemiologic studies and a duration of greater than 10% of the lifespan of the animal in toxicological studies.

between long-term exposures to ambient O₃ and deficits in the growth rate of lung-function in children, pulmonary inflammation and other endpoints. Episodic exposures were also known to cause more severe pulmonary morphological changes than continuous exposure.

The evidence base available in this review includes additional epidemiologic studies using a variety of designs and analysis methods evaluating the relationship between long-term O₃ exposures and measures of respiratory morbidity and mortality effects conducted by different research groups in different locations. The ISA (U.S. EPA, 2013, p. 7-33), in Table 7-2 presents selected key new longitudinal and cross-sectional studies of respiratory health effects and associated O₃ concentrations. The positive results from various designs and locations support a relationship between long-term exposure to ambient O₃ and respiratory health effects and mortality.

In this review, the evidence of effects associated with long-term exposures strengthens the relationship between O₃ exposure and health effects defined as adverse by the ATS, a definition that has been used in previous reviews of the O₃ standard. As discussed in more detail in section 3.1.3 below, the ATS (1985) defined adverse as “medically significant physiologic or pathologic changes generally evidenced by one or more of the following: (1) interference with the normal activity of the affected person or persons, (2) episodic respiratory illness, (3) incapacitating illness, (4) permanent respiratory injury, and/or (5) progressive respiratory dysfunction.” As discussed below, in this review there is now credible evidence of respiratory health effects associated with long-term O₃ exposures that would fall in to each of these five categories that define adversity.

From a policy perspective, the recent epidemiologic studies from the CHS of long-term O₃ exposures that shed light on the interaction between genetic variability, O₃ exposures, and health effects in children are important, not only because they help clarify previous findings, but also because the effects evaluated, such as new-onset asthma, are clearly adverse. The ISA (U.S. EPA, 2013, p. 7-12) notes that the collective evidence from CHS provides an important demonstration of gene-environment interactions. It further notes that in the complex gene-environment setting a modifying effect might not be reflected in an exposure main effect and that the simultaneous occurrence of main effect and interaction effect can occur. Moreover, the study of gene-environment interactions elucidates disease mechanisms in humans by using information on susceptibility genes to focus on the biological pathways that are most relevant to that disease.

In the CHS cohort of children in 12 Southern California communities, long-term exposure to O₃ concentrations was not associated with increased risk of developing asthma (McConnell et al., 2010); however, greater outdoor exercise was associated with development of asthma in children living in communities with higher ambient O₃ concentrations (McConnell et

al., 2002). Recent CHS studies examined interactions among genetic variants, long-term O₃ exposure, and new onset asthma in children. These prospective cohort studies are methodologically rigorous epidemiologic studies, and evidence indicates gene-O₃ interactions. These studies have provided data supporting decreased risk of certain genetic variants on new onset asthma (e.g., HMOX-1, ARG) that is limited to children either in low (Islam et al., 2008) or high (Salam et al., 2009) O₃ communities. Gene-environment interaction also was demonstrated with findings that greater outdoor exercise increased risk of asthma in GSTP1 Ile/Ile children living in high O₃ communities (Islam et al., 2009). Biological plausibility for these gene-O₃ environment interactions is provided by evidence that these enzymes have antioxidant and/or anti-inflammatory activity and participate in well recognized modes of action in asthma pathogenesis. As O₃ is a source of oxidants in the airways, oxidative stress serves as the link among O₃ exposure, enzyme activity, and asthma. Cross-sectional studies by Akinbami et al. (2010) and Hwang et al. (2005) provide further evidence relating O₃ exposures with asthma prevalence.

Studies using a cross-sectional design provide support for a relationship between long-term O₃ exposure and adverse health effects in asthmatics, including: bronchitic symptoms (related to TNF-308 genotype in asthmatic children) (Lee et al., 2009); asthma severity (Rage et al., 2009) and asthma control (Jacquemin et al., in press) in an adult cohort; respiratory-related school absences (related to CAT and MPO variant genes) (Wenten et al., 2009); asthma ED visits in adults (Meng et al., 2010); and, asthma hospital admissions in adults and children (Lin et al., 2008b; Meng et al., 2010; Moore et al., 2008). Several studies, shown in Table 7-3 (ISA, U.S. EPA, 2013, p. 7-35), provide results adjusted for potential confounders presenting results for both O₃ and PM (in single and multipollutant models) as well as other pollutants where PM effects were not provided. As shown in this table, O₃ associations were generally robust to adjustment by potential confounding by PM.

Information from toxicological studies in nonhuman primates indicates that long term exposure to O₃ during gestation or development can result in irreversible morphological changes in the lung, which in turn can influence the function of the respiratory tract. This nonhuman primate evidence of an O₃-induced change in airway responsiveness supports the biologic plausibility of long term exposure to O₃ contributing to effects of asthma in children. However, results from epidemiologic studies examining long-term O₃ exposure and pulmonary function effects are inconclusive with some new studies relating effects at higher exposure levels.

The ISA (U.S. EPA, 2013, p. 7-31) concludes that there is limited evidence for an association between long-term exposure to ambient O₃ concentrations and respiratory mortality in adults (Jerrett et al., 2009). This effect was robust to the inclusion of PM_{2.5} and insensitive to a number of different model specifications. Moreover, there is evidence that long-term exposure to

O₃ is associated with mortality among individuals that had previously experienced an emergency hospital admission due to COPD (Zanobetti and Schwartz, 2011).

In conclusion, since the last review, the body of evidence about the effects of long-term O₃ exposure has been considerably strengthened. The scientific evidence available for this review, including related uncertainties, provides an overall strong body of evidence of adverse health effects attributable to long-term O₃ exposures. These include a coherent range of asthma morbidity effects such as new-onset asthma, asthma prevalence, symptoms, school absences, ED visits and hospital admissions. There is also new evidence of respiratory mortality associated with long-term O₃ exposure. Further discussion of key studies is below.

New-onset Asthma and Asthma Prevalence

Asthma is a heterogeneous disease with a high degree of temporal variability. The on-set, progression, and symptoms can vary within an individual's lifetime, and the course of asthma may vary markedly in young children, older children, adolescents, and adults. In the previous review, longitudinal cohort studies that examined associations between long-term O₃ exposures and the onset of asthma in adults and children indicated a direct effect of long-term O₃ exposures on asthma risk in adults (McDonnell et al., 1999, 15-year follow-up; Greer et al., 1993, 10-year follow-up) and effect modification by O₃ in children (McConnell et al., 2002). Since that review, new evidence has become available about the association between long-term exposures to O₃ and new-onset asthma that has increased our understanding of the gene-environment interaction and the mechanisms and biological pathways most relevant to assessing O₃-related effects.

In children, the relationship between long-term O₃ exposure and new-onset asthma has been extensively studied in the CHS; a long-term study that was initiated in the early 1990's which has evaluated effects in several cohorts of children. The CHS was initially designed to examine whether long-term exposure to ambient pollution was related to chronic respiratory outcomes in children in 12 communities in southern California. In the CHS, new-onset asthma was classified as having no prior history of asthma at study entry with subsequent report of physician-diagnosed asthma at follow-up, with the date of onset assigned to be the midpoint of the interval between the interview date when asthma diagnosis was first reported and the previous interview date. The results of one study (McConnell et al., 2002) available in the previous review indicated that within high O₃ communities, asthma risk was 3.3 times greater for children who played three or more outdoor sports as compared with children who played no sports.

For this review, as discussed in section 7.2.1.1 of the ISA (U.S. EPA, 2013), recent studies from the CHS provide evidence for gene-environment interactions in effects on new-onset asthma by indicating that the lower risks associated with specific genetic variants are found in children who live in lower O₃ communities. These studies indicate that the risk for new-onset

asthma is related in part to genetic susceptibility, as well as behavioral factors and environmental exposure. The onset of a chronic disease, such as asthma, is partially the result of a sequence of biochemical reactions involving exposures to various environmental agents metabolized by enzymes related to a number of different genes. Oxidative stress has been proposed to underlie the mechanistic hypotheses related to O₃ exposure. Genetic variants may impact disease risk directly, or modify disease risk by affecting internal dose of pollutants and other environmental agents and/or their reaction products, or by altering cellular and molecular modes of action. Understanding the relation between genetic polymorphisms and environmental exposure can help identify high-risk subgroups in the population and provide better insight into pathway mechanisms for these complex diseases.

The CHS analyses (Islam et al., 2008; Islam et al. 2009; Salam et al., 2009) have found that asthma risk is related to interactions between O₃ and variants in genes for enzymes such as heme-oxygenase (HO-1), arginases (ARG1 and 2), and glutathione S transferase P1 (GSTP1). Biological plausibility for these findings is provided by evidence that these enzymes have antioxidant and/or anti-inflammatory activity and participate in well-recognized modes of action in asthma pathogenesis. Further, several lines of evidence demonstrate that secondary oxidation products of O₃ initiate the key modes of action that mediate downstream health effects (ISA, Section 5.3, U.S. EPA, 2013). For example, HO-1 responds rapidly to oxidants, has anti-inflammatory and anti-oxidant effects, relaxes airway smooth muscle, and is induced in the airways during asthma. Gene-environment interactions are discussed in detail in Section 5.4.2.1 in the ISA (U.S. EPA, 2013).

Asthma Hospital Admissions

In the 2006 AQCD, studies on O₃-related hospital discharges and emergency department (ED) visits for asthma and respiratory disease mainly looked at short-term (daily) metrics. The short-term O₃ studies presented in section 6.2.7.5 of the ISA (U.S. EPA, 2013) and discussed above in section 3.1.2.1 continue to indicate that there is evidence for increases in both hospital admissions and ED visits in children and adults related to all respiratory outcomes, including asthma, with stronger associations in the warm months. New studies, discussed in section 7.2.2 of the ISA (U.S. EPA, 2013) also evaluated long-term O₃ exposure metrics, providing a new line of evidence that suggests a positive exposure-response relationship between the first hospital admission for asthma and long-term O₃ exposure, although the ISA cautions in attributing the associations in that study to long-term exposures since there is potential for short-term exposures to contribute to the observed associations.

Evidence associating long-term O₃ exposure to first asthma hospital admission in a positive concentration-response relationship is provided in a retrospective cohort study (Lin et al., 2008b). This study investigated the association between chronic exposure to O₃ and

childhood asthma admissions by following a birth cohort of more than 1.2 million babies born in New York State (1995-1999) to first asthma admission or until 31 December 2000. Three annual indicators (all 8-hour maximum from 10:00 a.m. to 6:00 p.m.) were used to define chronic O₃ exposure: (1) mean concentration during the follow-up period (41.06 ppb); (2) mean concentration during the O₃ season (50.62 ppb); and (3) proportion of follow-up days with O₃ levels >70 ppb. The effects of co-pollutants were controlled, and interaction terms were used to assess potential effect modifications. A positive association between chronic exposure to O₃ and childhood asthma hospital admissions was observed, indicating that children exposed to high O₃ levels over time are more likely to develop asthma severe enough to be admitted to the hospital. The various factors were examined and differences were found for younger children (1-2 years), poor neighborhoods, Medicaid/self-paid births, geographic region and others. As shown in the ISA, Figure 7-3 (U.S. EPA, 2013, p. 7-16), positive concentration-response relationships were observed. Asthma admissions were significantly associated with increased O₃ levels for all chronic exposure indicators.

In considering the relationship between long-term pollutant exposures and chronic disease health endpoints, where chronic pathologies are found with acute expression of chronic disease, Künzli (2012) hypothesizes that if the associations of pollution with events are much larger in the long-term studies, it provides some indirect evidence that air pollution increases the pool of subjects with chronic disease, and that more acute events are to be expected to be seen for higher exposures. The results of Lin et al. (2008b) for first asthma hospital admission, presented in Figure 7-3 (U.S. EPA, 2013, p. 7-16), show effects estimates that are larger than those reported in a study of childhood asthma hospital admission in New York state (Silverman and Ito, 2010), discussed in section 3.1.2.1 and 3.1.2.2 above. The ISA (U.S. EPA, 2013, p. 7-16) notes that this provides some support for the hypothesis that O₃ exposure may not only have triggered the events but also increased the pool of asthmatic children, but cautions in attributing the associations in Lin et al. (2008b) study to long-term exposures since there is potential for short-term exposures to contribute to the observed associations.

Pulmonary structure and function

In the 2006 O₃ AQCD, few epidemiologic studies had investigated the effect of chronic O₃ exposure on pulmonary function. The definitive 8-year follow-up analysis of the first cohort of the CHS (U.S. EPA, 2013, section 7.2.3.1) provided little evidence that long-term exposure to ambient O₃ was associated with significant deficits in the growth rate of lung function in children. The strongest evidence was for medium-term effects of extended O₃ exposures over several summer months on lung function (FEV₁) in children, i.e., reduced lung function growth being associated with higher ambient O₃ levels. Short-term O₃ exposure studies presented in ISA (U.S. EPA, 2013, Section 6.2.1.2), and above in section 3.1.2.1, provide a cumulative body of

epidemiologic evidence that strongly supports associations between ambient O₃ exposure and decrements in lung function among children. A later CHS study (Islam et al., 2007) included in this review (U.S. EPA, 2013, section 7.2.3.1) also reported no substantial differences in the effect of O₃ on lung function. However, in a more recent CHS study, Breton et al. (2011) hypothesized that genetic variation in genes on the glutathione metabolic pathway may influence the association between ambient air pollutant exposures and lung function growth in children, and found that variation in the GSS locus was associated with differences in risk of children for lung function growth deficits associated ambient air pollutants, including O₃. A recent study (Rojas-Martinez et al., 2007) of long-term exposure to O₃, described in section 7.2.3.1 of the ISA (U.S. EPA, 2013, p. 7-19), observed a relationship with pulmonary function declines in school-aged children where O₃ and other pollutant levels were higher (90 ppb at high end of the range) than those in the CHS. Two studies of adult cohorts provide mixed results where long-term exposures were at the high end of the range.

Long-term studies in animals allow for greater insight into the potential effects of prolonged exposure to O₃ that may not be easily measured in humans, such as structural changes in the respiratory tract. Despite uncertainties, epidemiologic studies observing associations of O₃ exposure with functional changes in humans can attain biological plausibility in conjunction with long-term toxicological studies, particularly O₃-inhalation studies performed in non-human primates whose respiratory systems most closely resembles that of the human. An important series of studies, discussed in section 7.2.3.2 of the ISA (U.S. EPA, 2013), have used nonhuman primates to examine the effect of O₃ alone, or in combination with an inhaled allergen, house dust mite antigen (HDMA), on morphology and lung function. These animals exhibit the hallmarks of allergic asthma defined for humans, including: a positive skin test for HDMA with elevated levels of IgE in serum and IgE-positive cells within the tracheobronchial airway walls; impaired airflow which is reversible by treatment with aerosolized albuterol; increased abundance of immune cells, especially eosinophils, in airway exudates and bronchial lavage; and development of nonspecific airway responsiveness (NHLBI, 2007). These studies and others have demonstrated changes in pulmonary function and airway morphology in adult and infant nonhuman primates repeatedly exposed to environmentally relevant concentrations of O₃ (ISA, U.S. EPA, 2013, section 7.2.3.2).

The initial observations in adult nonhuman primates have been expanded in a series of experiments using infant rhesus monkeys repeatedly exposed to 0.5 ppm O₃ starting at 1 month of age (Plopper et al., 2007). The purpose of these studies was to determine if a cyclic regimen of O₃ inhalation would amplify the allergic responses and structural remodeling associated with allergic sensitization and inhalation in the infant rhesus monkey. After several episodic exposures of infant monkeys to O₃, they observed a significant increase in the baseline airway

resistance, which was accompanied by a small increase in airway responsiveness to inhaled histamine (Schelegle et al., 2003), although neither measurement was statistically different from filtered air control values. Exposure of animals to inhaled house dust mite antigen alone also produced small but not statistically significant changes in baseline airway resistance and airway responsiveness, whereas the combined exposure to both (O₃ + antigen) produced statistically significant and greater than additive changes in both functional measurements. This nonhuman primate evidence of an O₃-induced change in airway resistance and responsiveness provides biological plausibility of long-term exposure, or repeated short-term exposures, to O₃ contributing to the effects of asthma in children.

To understand which conducting airways and inflammatory mechanisms are involved in O₃-induced airway hyperresponsiveness in the infant rhesus monkey, results of a follow-up study (Joad et al., 2006) suggest that effect of O₃ on airway responsiveness occurs predominantly in the smaller bronchioles, where dosimetric models indicate the dose would be higher. The functional changes in the conducting airways were accompanied by a number of cellular and morphological changes, including a significant 4-fold increase in eosinophils. Thus, these studies demonstrate both functional and cellular changes in the lung of infant monkeys after cyclic exposure to 0.5 ppm O₃, providing relevant information to understanding the potentially damaging effects of ambient O₃ exposure on the respiratory tract of children.

In addition, noteworthy structural changes in the respiratory tract development, during which conducting airways increase in diameter and length, have been observed in infant rhesus monkeys after cyclic exposure to O₃ (Fanucchi et al., 2006). Observed changes included more proximal first alveolar outpocketing, decreases in the diameter and length of the terminal and respiratory bronchioles, increases in mucus-producing goblet cell mass, alterations in smooth muscle orientation in the respiratory bronchioles, epithelial nerve fiber distribution, and basement membrane zone morphometry. The latter effects are important because of their potential contribution to airway obstruction and airway hyperresponsiveness which are central features of asthma. A number of studies in both non-human primates and rodents demonstrate that O₃ exposure can increase collagen synthesis and deposition, including fibrotic-like changes in the lung (ISA, U.S. EPA, 2013, section 7.2.3.2.).

Collectively, evidence from animal studies strongly suggests that chronic O₃ exposure is capable of damaging the distal airways and proximal alveoli, resulting in lung tissue remodeling and leading to apparent irreversible changes. Potentially, persistent inflammation and interstitial remodeling play an important role in the progression and development of chronic lung disease. Further discussion of the modes of action that lead to O₃-induced morphological changes can be found in Section 5.3.7 of the ISA (U.S. EPA, 2013). Discussion of mechanisms involved in lifestage susceptibility and developmental effects can be found in Section 5.4.2.4 of the ISA

(U.S. EPA, 2013). The findings reported in chronic animal studies offer insight into potential biological mechanisms for the suggested association between seasonal O₃ exposure and reduced lung function development in children as observed in epidemiologic studies (see Section 7.2.3.1).

Respiratory Mortality

A limited number of epidemiologic studies have assessed the relationship between long-term exposure to O₃ and mortality in adults. The 2006 O₃ AQCD concluded that an insufficient amount of evidence existed “to suggest a causal relationship between chronic O₃ exposure and increased risk for mortality in humans” (U.S. EPA, 2006). Though total and cardio-pulmonary mortality were considered in these studies, respiratory mortality was not specifically considered. In the most recent follow-up analysis of the ACS cohort (Jerrett et al., 2009), cardiopulmonary deaths were separately subdivided into respiratory and cardiovascular deaths, rather than combined as in the Pope et al. (2002) work. Increased O₃ exposure was associated with the risk of death from respiratory causes, and this effect was robust to the inclusion of PM_{2.5}. The association between increased O₃ concentrations and increased risk of death from respiratory causes was insensitive to the use of different models and to adjustment for several ecologic variables considered individually. Additionally, a recent multi-city time series study (Zanobetti and Schwartz, 2011), which followed (from 1985 to 2006) four cohorts of Medicare enrollees with chronic conditions that might predispose to O₃-related effects, observed an association between long-term (warm season) exposure to O₃ and elevated risk of mortality in the cohort that had previously experienced an emergency hospital admission due to COPD. A key limitation of this study is the inability to control for PM_{2.5}, because data were not available in these cities until 1999.

3.1.2.3 Total Mortality – Short-term Exposures

- **To what extent does the currently available scientific evidence, including related uncertainties, strengthen or alter our understanding from the last review of mortality attributable to short-term O₃ exposures?**

The 2006 O₃ AQCD concluded that the overall body of evidence was highly suggestive that short-term exposure to O₃ directly or indirectly contributes to nonaccidental and cardiopulmonary-related mortality in adults, but additional research was needed to more fully establish underlying mechanisms by which such effects occur (U.S. EPA, 2006; U.S. EPA, 2013, p. 2-18). In building on the 2006 evidence, the ISA states the following (U.S. EPA, 2013, p. 6-261).

The evaluation of new multicity studies that examined the association between short-term O₃ exposures and mortality found evidence that supports the conclusions of the 2006 AQCD. These new studies reported consistent positive associations between short-term O₃ exposure and all-cause (non-accidental)

mortality, with associations persisting or increasing in magnitude during the warm season, and provide additional support for associations between O₃ exposure and cardiovascular and respiratory mortality

The 2006 O₃ AQCD reviewed a large number of time-series studies of associations between short-term O₃ exposures and total mortality including single- and multicity studies, and meta-analyses. In the large U.S. multicity studies that examined all-year data, effect estimates corresponding to single-day lags ranged from a 0.5-1% increase in all-cause (nonaccidental) total mortality per a 20 ppb (24-hour), 30 ppb (8-hour maximum), or 40 ppb (1-hour maximum) increase in ambient O₃ (U.S. EPA, 2013, section 6.6.2). Available studies reported some evidence for heterogeneity in O₃ mortality risk estimates across cities and across studies. Studies that conducted seasonal analyses reported larger O₃ mortality risk estimates during the warm season. Overall, the 2006 O₃ AQCD identified robust associations between various measures of daily ambient O₃ concentrations and all-cause mortality, which could not be readily explained by confounding due to time, weather, or copollutants. With regard to cause-specific mortality, consistent positive associations were reported between short-term O₃ exposure and cardiovascular mortality, with less consistent evidence for associations with respiratory mortality. The majority of the evidence for associations between O₃ and cause-specific mortality were from single-city studies, which had small daily mortality counts and subsequently limited statistical power to detect associations. The 2006 O₃ AQCD concluded that “the overall body of evidence is highly suggestive that O₃ directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality” (U.S. EPA, 2013, section 6.6.1).

Recent studies have strengthened the body of evidence that supports the association between short-term O₃ concentrations and mortality in adults. This evidence includes a number of studies reporting associations with non-accidental as well as cause-specific mortality. Multi-continent and multicity studies have consistently reported positive and statistically significant associations between short-term O₃ concentrations and all-cause mortality, with evidence for larger mortality risk estimates during the warm or summer months (Figure 3-3 below, reprinted from the ISA) (U.S. EPA, 2013, Figure 6-27; Table 6-42). Similarly, evaluations of cause-specific mortality have reported consistently positive associations with O₃, particularly in analyses restricted to the warm season (U.S. EPA, 2013, Figure 6-37; Table 6-53).³³

³³ Respiratory mortality is discussed in more detail above.

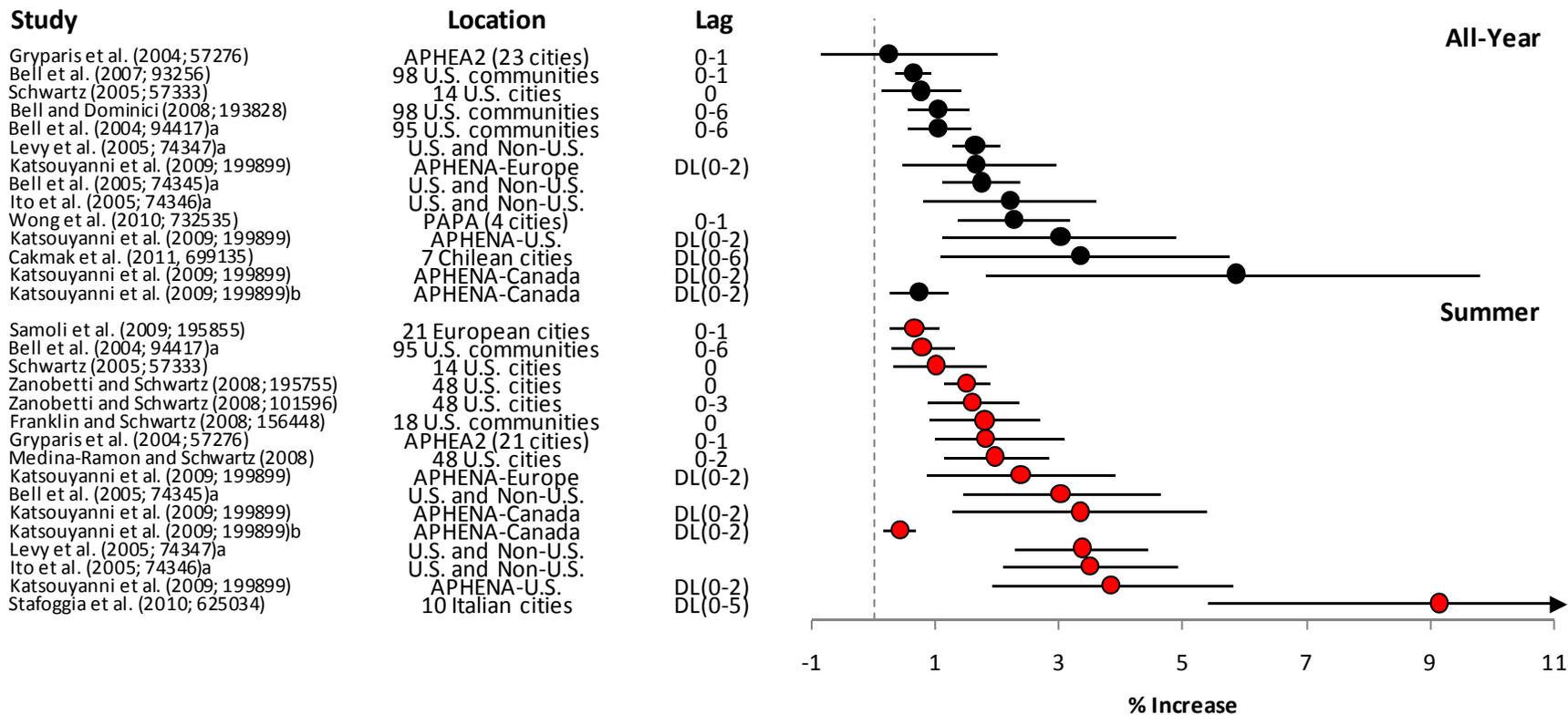


Figure 3-3. Summary of mortality risk estimates for short-term O₃ and all-cause (nonaccidental) mortality.³⁴

³⁴ Reprinted from the ISA (U.S. EPA, 2013, Figure 6-27).

In assessing the evidence for O₃-related mortality, the 2006 AQCD also noted that multiple uncertainties remained regarding the relationship between short-term O₃ concentrations and mortality, including the extent of residual confounding by co-pollutants; characterization of the factors that modify the O₃-mortality association; the appropriate lag structure for identifying O₃-mortality effects; and the shape of the O₃-mortality concentration-response function and whether a threshold exists. Many of the studies, published since the last review, have attempted to address one or more of these uncertainties. The ISA (U.S. EPA, 2013, Section 6.6.2) discusses the extent to which recent studies have evaluated these uncertainties in the relationship between O₃ and mortality.

In particular, recent studies have evaluated different statistical approaches to examine the shape of the O₃-mortality concentration-response relationship and to evaluate whether a threshold exists for O₃-related mortality. In an analysis of the NMMAPS data, Bell et al. (2006) evaluated the potential for a threshold in the O₃-mortality relationship. The authors reported positive and statistically significant associations with mortality in a variety of subset analyses, including analyses restricted to days with 24-hour area-wide average O₃ concentrations below 60, 55, 50, 45, 40, 35, and 30 ppb. In these restricted analyses O₃ effect estimates were of similar magnitude, were statistically significant, and had similar statistical precision. In analyses restricted to days with 24-hour average O₃ concentrations below 25 ppb, the O₃ effect estimate was similar in magnitude to the effect estimates resulting from analyses with the higher cutoffs, but had somewhat lower statistical precision, with the estimate approaching statistical significance (i.e., based on observation of Figure 2 in Bell et al., 2006). In analyses restricted to days with lower 24-hour average O₃ concentrations, effect estimates were not statistically significant (i.e., based on observation of Figure 2 in Bell et al., 2006).

Bell et al. (2006) also evaluated the shape of the concentration-response relationship between O₃ and mortality. Although the results of this analysis suggested the lack of threshold in the O₃-mortality relationship, the ISA noted that it is difficult to interpret such a curve because: (1) there is uncertainty around the shape of the concentration-response curve at 24-hour average O₃ concentrations generally below 20 ppb and (2) the concentration-response curve does not take into consideration the heterogeneity in O₃-mortality risk estimates across cities (U.S. EPA, 2013, section 6.6.2.3).

Several additional studies have used the NMMAPS dataset to evaluate the concentration-response relationship between short-term O₃ concentrations and mortality. For example, using the same data as Bell et al. (2006), Smith et al. (2009) conducted a subset analysis, but instead of restricting the analysis to days with O₃ concentrations below a cutoff the authors only included days *above* a defined cutoff. The results of this analysis were consistent with those reported by Bell et al. (2006). Specifically, the authors reported consistent positive associations for all cutoff

concentrations up to concentrations where the total number of days available were so limited that the variability around the central estimate was increased (U.S. EPA, 2013, section 6.6.2.3). In addition, using NMMAPS data for 1987-1994 for Chicago, Pittsburgh, and El Paso, Xia and Tong (2006) reported evidence for a threshold around a 24-hour average O₃ concentration of 25 ppb, though the threshold values estimated in the analysis were sometimes in the range of where data density was low (U.S. EPA, 2013, section 6.6.2.3). Stylianou and Nicolich (2009) examined the existence of thresholds following an approach similar to Xia and Tong (2006) using data from NMMAPS for nine major U.S. cities (i.e., Baltimore, Chicago, Dallas/Fort Worth, Los Angeles, Miami, New York, Philadelphia, Pittsburgh, and Seattle) for the years 1987-2000. The authors reported that the estimated O₃-mortality risks varied across the nine cities, with the models exhibiting apparent thresholds in the 10-45 ppb range for O₃ (24-hour average). Additional studies in Europe, Canada, and Asia did not report evidence for a threshold (Katsouyanni et al., 2009).

3.1.2.4 Cardiovascular effects – Short-term Exposure

- **To what extent does the currently available scientific evidence, including related uncertainties, strengthen or alter our understanding from the last review of cardiovascular effects attributable to short-term O₃ exposures?**

A relatively small number of studies have examined the potential effect of short-term O₃ exposure on the cardiovascular system. The 2006 O₃ AQCD (U.S. EPA, 2006, p. 8-77) concluded that “O₃ directly and/or indirectly contributes to cardiovascular-related morbidity” but added that the body of evidence was limited. This conclusion was based on a controlled human exposure study that included hypertensive adult males; a few epidemiologic studies of physiologic effects, heart rate variability, arrhythmias, myocardial infarctions, and hospital admissions; and toxicological studies of heart rate, heart rhythm, and blood pressure.

More recently, the body of scientific evidence available that has examined the effect of O₃ on the cardiovascular system has expanded. There is an emerging body of animal toxicological evidence demonstrating that short-term exposure to O₃ can lead to autonomic nervous system alterations (in heart rate and/or heart rate variability) and suggesting that proinflammatory signals may mediate cardiovascular effects. Interactions of O₃ with respiratory tract components result in secondary oxidation product formation and subsequent production of inflammatory mediators, which have the potential to penetrate the epithelial barrier and to initiate toxic effects systemically. In addition, animal toxicological studies of long-term exposure to O₃ provide evidence of enhanced atherosclerosis and ischemia/reperfusion (I/R) injury, corresponding with development of a systemic oxidative, proinflammatory environment. Recent experimental and epidemiologic studies have investigated O₃-related cardiovascular events and

are summarized in Section 6.3 of the ISA (U.S. EPA, 2013, Section 6.3). Overall, the ISA summarized the evidence in this review as follows (U.S. EPA, 2013, p. 6-211).

In conclusion, animal toxicological studies demonstrate O₃-induced cardiovascular effects, and support the strong body of epidemiologic evidence indicating O₃-induced cardiovascular mortality. Animal toxicological and controlled human exposure studies provide evidence for biologically plausible mechanisms underlying these O₃-induced cardiovascular effects. However, a lack of coherence with epidemiologic studies of cardiovascular morbidity remains an important uncertainty.

Animal toxicological studies support that short-term O₃ exposure can lead to cardiovascular morbidity. Animal studies provide evidence for both increased and decreased heart rate (HR), however it is uncertain if O₃-induced reductions in HR are relevant to humans. Animal studies also provide evidence for increased heart rate variability (HRV), arrhythmias, vascular disease and injury following short-term O₃ exposure. In addition, a series of studies highlight the role of genetic variability and age in the induction of effects and attenuation of responses to O₃ exposure.

Biologically plausible mechanisms have been described for the cardiovascular effects observed in animal exposure studies (U.S. EPA, 2013, Section 5.3.8). Evidence that parasympathetic pathways may underlie cardiac effects is described in more detail in Section 5.3.2 of the ISA (U.S. EPA, 2013). Recent studies suggest that O₃ exposure may disrupt the endothelin system that constricts blood vessels and increase blood pressure, which can result in an increase in HR, HRV; and disrupt the NO system and the production of atrial natriuretic factor (ANF), vasodilators that reduce blood pressure. Additionally, O₃ may increase oxidative stress and vascular inflammation promoting the progression of atherosclerosis and leading to increased susceptibility to I/R injury. As O₃ reacts quickly with the ELF and does not translocate to the heart and large vessels, studies suggest that the cardiovascular effects exhibited could be caused by secondary oxidation products resulting from O₃ exposure. However, direct evidence of translocation of O₃ reaction products to the cardiovascular system has not been demonstrated *in vivo*. Alternatively, extrapulmonary release of diffusible mediators (such as cytokines or endothelins) may initiate or propagate inflammatory responses throughout the body leading to the cardiovascular effects reported in toxicology studies. Ozone reacts within the lung to induce pulmonary inflammation and the influx and activation of inflammatory cells, resulting in a cascading proinflammatory state, and may lead to the extrapulmonary release of diffusible mediators that could result in cardiovascular injury.

Controlled human exposures studies discussed in previous AQCDs have not demonstrated any consistent extrapulmonary effects. In this review, evidence from controlled human exposure studies suggests cardiovascular effects in response to short-term O₃ exposure

(see ISA, U.S. EPA, 2013, Section 6.3.1) and provides some coherence with evidence from animal toxicology studies. Controlled human exposure studies also support the animal toxicological studies by demonstrating O₃-induced effects on blood biomarkers of systemic inflammation and oxidative stress, as well as changes in biomarkers that can indicate a prothrombogenic response to O₃. Increases and decreases in high frequency HRV have been reported following relatively low (120 ppb during rest) and high (300 ppb with exercise) O₃ exposures, respectively. These changes in cardiac function observed in animal and human studies provide preliminary evidence for O₃-induced modulation of the autonomic nervous system through the activation of neural reflexes in the lung (see ISA, U.S. EPA, 2013, Section 5.3.2).

Overall, the ISA concludes that the available body of epidemiologic evidence examining the relationship between short-term exposures to O₃ concentrations and cardiovascular morbidity is inconsistent (U.S. EPA, 2013, Section 6.3.2.9). Across studies, different definitions, (i.e., ICD-9 diagnostic codes) were used for both all-cause and cause-specific cardiovascular morbidity (ISA, U.S. EPA, 2013, see Tables 6-35 to 6-39), which may contribute to inconsistency in results. However, within diagnostic categories, no consistent pattern of association was found with O₃. Generally, the epidemiologic studies used nearest air monitors to assess O₃ concentrations, with a few exceptions that used modeling or personal exposure monitors. The inconsistencies in the associations observed between short-term O₃ and cardiovascular disease (CVD) morbidities are unlikely to be explained by the different exposure assignment methods used (see Section 4.6, ISA, U.S. EPA, 2013). The wide variety of biomarkers considered and the lack of consistency among definitions used for specific cardiovascular disease endpoints (e.g., arrhythmias, HRV) make comparisons across studies difficult.

Despite the inconsistent evidence for an association between O₃ concentration and CVD morbidity, mortality studies indicate a consistent positive association between short-term O₃ exposure and cardiovascular mortality in multicity studies and in a multicontinent study. When examining mortality due to cardiovascular disease, epidemiologic studies consistently observe positive associations with short-term exposure to O₃. Additionally, there is some evidence for an association between long-term exposure to O₃ and mortality, although the association between long-term ambient O₃ concentrations and cardiovascular mortality can be confounded by other pollutants as evident by a study of cardiovascular mortality that reported no association after adjustment for PM_{2.5} concentrations. The ISA (U.S. EPA, 2013, section 6.3.4) states that taken together, the overall body of evidence across the animal and human studies is sufficient to conclude that there is likely to be a causal relationship between relevant short-term exposures to O₃ and cardiovascular system effects.

3.1.3 Adversity of Effects

In this section we address the following question:

- **To what extent does the currently available scientific evidence expand our understanding of the adversity of O₃-related health effects?**

In making judgments as to when various O₃-related effects become regarded as adverse to the health of individuals, in previous NAAQS reviews staff has relied upon the guidelines published by the ATS and the advice of CASAC. In 2000, the ATS published an official statement on “What Constitutes an Adverse Health Effect of Air Pollution?” (ATS, 2000), which updated and built upon its earlier guidance (ATS, 1985). The earlier guidance defined adverse respiratory health effects as “medically significant physiologic changes generally evidenced by one or more of the following: (1) interference with the normal activity of the affected person or persons, (2) episodic respiratory illness, (3) incapacitating illness, (4) permanent respiratory injury, and/or (5) progressive respiratory dysfunction”, while recognizing that perceptions of “medical significance” and “normal activity” may differ among physicians, lung physiologists and experimental subjects (ATS, 1985). The 2000 ATS guidance builds upon and expands the 1985 definition of adversity in several ways. The guidance concludes that transient, reversible loss of lung function in combination with respiratory symptoms should be considered adverse. There is also a more specific consideration of population risk (ATS, 2000). Exposure to air pollution that increases the risk of an adverse effect to the entire population is adverse, even though it may not increase the risk of any individual to an unacceptable level. For example, a population of asthmatics could have a distribution of lung function such that no individual has a level associated with clinical impairment. Exposure to air pollution could shift the distribution to lower levels that still do not bring any individual to a level that is associated with clinically relevant effects. However, this would be considered to be adverse because individuals within the population would have diminished reserve function, and therefore would be at increased risk to further environmental insult (U.S. EPA, 2013, p. lxxi; and 75 FR at 35526/2, June 22, 2010).

The ATS also concluded that elevations of biomarkers such as cell 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. More subtle health outcomes also may be connected mechanistically to health effects that are clearly adverse, so that small changes in physiological measures may not appear clearly adverse when considered alone, but may be part of a coherent and biologically plausible chain of related health outcomes that include responses that are clearly adverse, such as mortality (section 3.1.2.1, above).

In this review, the new evidence provides further support for relationships between O₃ exposures and a spectrum of health effects, including effects that meet the ATS criteria for being adverse (ATS, 1985 and 2000). The ISA judgment that there is a causal relationship between short-term O₃ exposure and a full range of respiratory effects, including respiratory morbidity (e.g., lung function decrements, respiratory symptoms, inflammation, hospital admissions, and emergency department visits) and mortality, provides support for concluding that short-term O₃ exposure is associated with adverse effects (U.S. EPA, 2013, section 2.5.2). Overall, including new evidence of cardiovascular system effects, the evidence supporting an association between short-term O₃ exposures and total (non-accidental, cardiopulmonary) respiratory mortality is stronger in this review (U.S. EPA, 2013, section 2.5.2). And the judgment of likely causal associations between long-term measures of O₃ exposure and respiratory effects such as new-onset asthma, prevalence of asthma, asthma symptoms and control, and asthma hospital admissions provides support for concluding that long-term O₃ exposure is associated with adverse effects ranging from episodic respiratory illness to permanent respiratory injury or progressive respiratory decline (U.S. EPA, 2013, section 7.2.8).

This review provides additional evidence of O₃-attributable effects that are clearly adverse, including premature mortality. Application of the ATS guidelines to the least serious category of effects related to ambient O₃ exposures, which are also the most numerous and therefore are also potentially important from a public health perspective, involves judgments about which medical experts on CASAC panels and public commenters have in the past expressed diverse views. To help frame such judgments, EPA staff defined gradations of individual functional responses (e.g., decrements in FEV₁ and airway responsiveness) and symptomatic responses (e.g., cough, chest pain, wheeze), together with judgments as to the potential impact on individuals experiencing varying degrees of severity of these responses. These gradations were used in the 1997 O₃ NAAQS review and slightly revised in the 2008 review (U.S. EPA, 1996, p. 59; 2007, p. 3-72; 72 FR 37849, July 11, 2007). These gradations and impacts are summarized in Tables 3-2 and 3-3 in the 2007 O₃ Staff Paper (U.S. EPA, 2007, pp. 3-74 to 3-75).

For active healthy people, including children, moderate levels of functional responses (e.g., FEV₁ decrements of $\geq 10\%$ but $< 20\%$, lasting 4 to 24 hours) and/or moderate symptomatic responses (e.g., frequent spontaneous cough, marked discomfort on exercise or deep breath, lasting 4 to 24 hours) would likely interfere with normal activity for relatively few sensitive individuals (U.S. EPA, 2007, p. 3-72; 72 FR 37849, July 11, 2007); whereas large functional responses (e.g., FEV₁ decrements $\geq 20\%$, lasting longer than 24 hours) and/or severe symptomatic responses (e.g., persistent uncontrollable cough, severe discomfort on exercise or deep breath, lasting longer than 24 hours) would likely interfere with normal activities for many

sensitive individuals (U.S. EPA, 2007, p. 3-72; 72 FR 37849, July 11, 2007) and therefore would be considered adverse under ATS guidelines. For the purpose of estimating potentially adverse lung function decrements in active healthy people in the 2008 O₃ NAAQS review, the CASAC panel for that review indicated that a focus on the mid to upper end of the range of moderate levels of functional responses is most appropriate (e.g., FEV₁ decrements $\geq 15\%$ but $< 20\%$) (Henderson, 2006; U.S. EPA, 2007, p. 3-76). In this review, CASAC concurred that the “[e]stimation of FEV₁ decrements of $\geq 15\%$ is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults” (Frey, 2014, p. 3). However, for children and adults with lung disease, even moderate functional (e.g., FEV₁ decrements $\geq 10\%$ but $< 20\%$, lasting up to 24 hours) or symptomatic responses (e.g., frequent spontaneous cough, marked discomfort on exercise or with deep breath, wheeze accompanied by shortness of breath, lasting up to 24 hours) would likely interfere with normal activity for many individuals, and would likely result in additional and more frequent use of medication (U.S. EPA, 2007, p.3-72; 72 FR 37849, July 11, 2007). For people with lung disease, large functional responses (e.g., FEV₁ decrements $\geq 20\%$, lasting longer than 24 hours) and/or severe symptomatic responses (e.g., persistent uncontrollable cough, severe discomfort on exercise or deep breath, persistent wheeze accompanied by shortness of breath, lasting longer than 24 hours) would likely interfere with normal activity for most individuals and would increase the likelihood that these individuals would seek medical treatment (U.S. EPA, 2007, p.3-72; 72 FR 37849, July 11, 2007). In the last O₃ NAAQS review, for the purpose of estimating potentially adverse lung function decrements in people with lung disease the CASAC panel indicated that a focus on the lower end of the range of moderate levels of functional responses is most appropriate (e.g., FEV₁ decrements $\geq 10\%$) (Henderson, 2006; U.S. EPA, 2007, p. 3-76). In addition, in the reconsideration of the 2008 final decision, CASAC stated that “[a] 10% decrement in FEV₁ can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV₁) such that a $\geq 10\%$ decrement could lead to moderate to severe respiratory symptoms” (Samet, 2011) (section 3.1.2.1, above). In this review, CASAC concurred that “[a]n FEV₁ decrement of $\geq 10\%$ is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014, p. 3).

In judging the extent to which these impacts represent effects that should be regarded as adverse to the health status of individuals, in previous NAAQS reviews we also considered whether effects were experienced repeatedly during the course of a year or only on a single occasion (Staff Paper, U.S. EPA, 2007). Although some experts would judge single occurrences of moderate responses to be a “nuisance,” especially for healthy individuals, a more general consensus view of the adversity of such moderate responses emerges as the frequency of

occurrence increases. Thus it has been judged that repeated occurrences of moderate responses, even in otherwise healthy individuals, may be considered to be adverse since they could well set the stage for more serious illness (61 FR 65723). The CASAC panel in the 1997 NAAQS review expressed a consensus view that these “criteria for the determination of an adverse physiological response were reasonable” (Wolff, 1995). In the review completed in 2008, estimates of repeated occurrences continued to be an important public health policy factor in judging the adversity of moderate lung function decrements in healthy and asthmatic people (72 FR 37850, July 11, 2007).

Evidence new to this review indicates that 6.6-hour exposures to 60 ppb O₃ during moderate exertion can result in pulmonary inflammation in healthy adults. As discussed in section 3.1.2 above, the initiation of inflammation can be considered as evidence that injury has occurred. Inflammation induced by a single O₃ exposure can resolve entirely, but continued acute inflammation can evolve into a chronic inflammatory state (ISA, U.S. EPA, 2013, p. 6-76), which is clearly adverse. Therefore, like moderate lung function decrements, whether inflammation is experienced repeatedly during the course of a year or only on a single occasion is judged by staff to be reasonable criteria for determining adverse inflammatory effects attributable to O₃ exposures at 60 ppb.

Responses measured in controlled human exposure studies indicate that the range of effects elicited in humans exposed to ambient O₃ concentrations include: decreased inspiratory capacity; mild bronchoconstriction; rapid, shallow breathing pattern during exercise; and symptoms of cough and pain on deep inspiration (EPA, 2013, section 6.2.1.1). Some young, healthy adults exposed to O₃ concentrations \geq 60 ppb, while engaged in 6.6 hours of intermittent moderate exertion, develop reversible, transient decrements in lung function, symptoms of breathing discomfort, and inflammation if minute ventilation or duration of exposure is increased sufficiently (EPA, 2013, section 6.2.1.1). Among healthy subjects there is considerable interindividual variability in the magnitude of the FEV₁ responses, but averaged across studies at 60 ppb (EPA, 2013, pp. 6-17 to 6-18), 10% of healthy subjects had >10% FEV₁ decrements. Moreover, consistent with the findings of the ISA (EPA, 2013, section 6.2.1.1), CASAC concluded that “[a]sthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting ozone-induced pulmonary function decrements” (Frey, 2014, p. 4). The combination of lung function decrements and respiratory symptoms, which has been considered adverse in previous reviews, has been demonstrated in healthy adults following prolonged (6.6 hour) exposures, while at intermittent moderate exertion, to 70 ppb. For these types of effects, information from controlled human exposure studies, which provides an indication of the magnitude and thus adversity of effects at different O₃ concentrations,

combined with estimates of occurrences in the population from the HREA, provide information about their importance from a policy perspective.

3.1.4 Ozone Concentrations Associated With Health Effects

In evaluating O₃ exposure concentrations reported to result in health effects, within the context of the adequacy of the current standard, we first consider the following specific question:

- **To what extent does the currently available scientific evidence indicate morbidity and/or mortality attributable to exposures to O₃ concentrations lower than previously reported or that would meet the current standard?**

In addressing this question, we characterize the extent to which O₃-attributable effects have been reported over the ranges of O₃ exposure concentrations evaluated in controlled human exposure studies and over the distributions of ambient O₃ concentrations in locations where epidemiologic studies have been conducted.

3.1.4.1 Concentrations in Controlled Human Exposure Studies and in Epidemiologic Panel Studies

In considering what the currently available evidence indicates with regard to effects associated with exposure concentrations lower than those identified in the last review, or that could meet the current standard, we first consider the evidence from controlled human exposure studies and epidemiologic panel studies. This evidence is assessed in section 6.2 of the ISA and is summarized in section 3.1.2 above. Controlled human exposure studies have generally been conducted with young, healthy adults, and have evaluated exposure durations less than 8 hours. Epidemiologic panel studies have evaluated a wider range of study populations, including children, and have generally evaluated associations with O₃ concentrations averaged over several hours (U.S. EPA, 2013, section 6.2.1.2).³⁵

As summarized above (section 3.1.2.1), and as discussed in detail in the ISA (U.S. EPA, 2013, section 6.2), a large number of controlled human exposure studies have reported lung function decrements, respiratory symptoms, airway inflammation, airway hyperresponsiveness, and/or impaired lung host defense in young, healthy adults engaged in moderate, intermittent exertion, following 6.6-hour O₃ exposures. These studies have consistently reported such effects following exposures to O₃ concentrations of 80 ppb or greater. Available studies have also evaluated some of these effects (i.e., lung function decrements, respiratory symptoms, airway inflammation) following exposures to O₃ concentrations below 75 ppb. Table 3-1 highlights the

³⁵ In this section we focus on panel studies that used on-site monitoring, and that are highlighted in the ISA for the extent to which monitored ambient O₃ concentrations reflect exposure concentrations in their study populations (U.S. EPA, 2013, section 6.2.1.2).

group mean results of individual controlled human exposure studies that have evaluated exposures of healthy adults to O₃ concentrations below 75 ppb. The studies included in Table 3-1 indicate lung function decrements, airway inflammation, and respiratory symptoms in healthy adults following exposures to O₃ concentrations below 75 ppb.

Table 3-1. Group mean results of controlled human exposure studies that have evaluated exposures to ozone concentrations below 75 ppb in young, healthy adults.

Endpoint	O ₃ Exposure Concentration	Study	Statistically Significant O ₃ -Induced Effect ³⁶
FEV ₁ decrements	70 ppb	Schelegle et al., 2009 ³⁷	yes
	60 ppb	Kim et al., 2011	yes
		Schelegle et al., 2009 ³⁸	no
		Adams, 2006	yes ³⁹
		Adams, 2002	no
	40 ppb	Adams, 2006	no
Adams, 2002		no	
Respiratory Symptoms	70 ppb	Schelegle et al., 2009	yes
	60 ppb	Kim et al., 2011	no
		Schelegle et al., 2009	no
		Adams, 2006	no ⁴⁰
	40 ppb	Adams, 2006	no
		Adams, 2002	no
Airway Inflammation (neutrophil influx)	60 ppb	Kim et al., 2011	yes

In further evaluating O₃-induced FEV₁ decrements following exposures to O₃ concentrations below 75 ppb, the ISA also combined the individual data from multiple studies of healthy adults exposed for 6.6 hours to 60 ppb O₃ (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 FEV₁ decrements of 10% or more (i.e., abnormal and large enough to be potentially adverse for people with pulmonary disease, based on past CASAC advice (section 3.1.3, above))⁴¹ (U.S. EPA, 2013, section 6.2.1.1). Consistent with these findings, recently developed

³⁶ Based on study population means.

³⁷ As noted above, for the 70 ppb exposure concentration Schelegle et al. (2009) reported that the actual mean exposure concentration was 72 ppb.

³⁸ As noted above, for the 60 ppb exposure concentration Schelegle et al. (2009) reported that the actual mean exposure concentration was 63 ppb.

³⁹ In an analysis of the Adams (2006) data for square-wave chamber exposures, even after removal of potential outliers, Brown et al. (2008) reported the average effect on FEV₁ at 60 ppb to be statistically significant (p < 0.002) using several common statistical tests (U.S. EPA, 2013, section 6.2.1.1) (section 3.1.2.1, above).

⁴⁰ Adams (2006) reported increased respiratory symptoms during a 6.6 hour exposure protocol with an average O₃ exposure concentration of 60 ppb. The increase in symptoms was reported to be statistically different from initial respiratory symptoms, though not statistically different from filtered air controls.

⁴¹ As noted above (section 3.1.3), CASAC has previously stated that “[a] 10% decrement in FEV₁ can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV₁) such that a ≥10% decrement could lead to moderate to severe respiratory symptoms” (Samet, 2011).

empirical models predict that the onset of O₃-induced FEV₁ decrements in healthy adults occurs following exposures to 60 ppb O₃ for 4 to 5 hours while at moderate, intermittent exertion (Schelegle et al., 2012), and that 9% of healthy adults exposed to 60 ppb O₃ for 6.6 hours would experience FEV₁ decrements greater than or equal to 10% (McDonnell et al., 2012) (U.S. EPA, 2013, section 6.2.1.1; section 3.1.2.1, above). When the evidence for O₃-induced lung function decrements was taken together, the ISA concluded that (1) “mean FEV₁ is clearly decreased by 6.6-h exposures to 60 ppb O₃ and higher concentrations in subjects performing moderate exercise” (U.S. EPA, 2013, p. 6-9) and (2) although group mean decrements following exposures to 60 ppb O₃ are biologically small, “a considerable fraction of exposed individuals experience clinically meaningful decrements in lung function” (U.S. EPA, 2013, p. 6-20).

In considering the specific question above, controlled human exposure studies have reported decreased lung function, increased airway inflammation, and increased respiratory symptoms in healthy adults following exposures to O₃ concentrations below 75 ppb. Such impairments in respiratory function have the potential to be adverse, based on ATS guidelines for adversity and based on previous advice from CASAC (section 3.1.3, above). In addition, if they become serious enough, these respiratory effects could lead to the types of clearly adverse effects commonly reported in O₃ epidemiologic studies (e.g., respiratory emergency department visits, hospital admissions). Therefore, following exposures to O₃ concentrations lower than 75 ppb, controlled human exposure studies have reported respiratory effects that could be adverse in some individuals, particularly if experienced by members of at-risk populations (e.g., asthmatics, children).⁴²

In further considering effects following exposures to O₃ concentrations below 75 ppb, we also note that the ISA highlights some epidemiologic panel studies for the extent to which monitored ambient O₃ concentrations reflect exposure concentrations in their study populations (U.S. EPA, 2013, section 6.2.1.2). Specifically, Table 3-2 below includes O₃ panel studies that have evaluated associations with lung function decrements for O₃ concentrations at or below 75 ppb, and that measured O₃ concentrations with monitors located in the areas where study subjects were active (e.g., on site at summer camps or in locations where exercise took place) (U.S. EPA, 2013, section 6.2.1.2 and Table 6-6). Epidemiologic panel studies have evaluated a wider range of populations and lifestages than controlled human exposure studies of O₃ concentrations below 75 ppb (e.g., including children).

⁴² These effects were reported in healthy individuals. Consistent with past CASAC advice (Samet, 2011), and evidence in the ISA (U.S. EPA, 2013, p. 6-77), it is a reasonable inference that the effects would be greater in magnitude and potential severity for at-risk groups. See *National Environmental Development Ass'n Clean Air Project v. EPA*, 686 F. 3d 803, 811 (D.C. Cir. (2012) (making this point).

Table 3-2. Panel studies of lung function decrements with analyses restricted to O₃ concentrations below 75 ppb.

Study	Population	O ₃ Concentrations	Statistically Significant Association with Lung Function Decrements
Spektor et al. (1988a)	Children at summer camp	Restricted to 1-hour concentrations below 60 ppb	Yes
Chan and Wu (2005)	Mail carriers	Maximum 8-hour average was 65 ppb	Yes
Korrick et al. (1998)	Adult hikers	2- to 12-hour average from 40 to 74 ppb during hikes	Yes
Brauer et al. (1996)	Farm workers	Restricted to 1-hour maximum below 40 ppb	Yes
		Restricted to 1-hour maximum below 30 ppb	No
Brunekreef et al. (1994)	Exercising adults	Restricted to 10-minute to 2.4-hour averages below 61 ppb	No
		Restricted to 10-minute to 2.4-hour averages below 51 ppb	No
		Restricted to 10-minute to 2.4-hour averages below 41 ppb	No

Although these studies report health effect associations for different averaging times, and it is not clear the extent to which specific O₃ exposure conditions (i.e., concentrations, durations of exposure, degrees of activity) were responsible for eliciting reported decrements, they are consistent with the findings of the controlled human exposure studies discussed above. Specifically, the epidemiologic panel studies in Table 3-2 indicate O₃-associated lung function decrements when on-site monitored concentrations (ranging from minutes to hours) were below 75 ppb, with the evidence becoming less consistent at lower O₃ concentrations.

3.1.4.2 Concentrations in Epidemiologic Studies – Short-term Metrics

We next consider distributions of ambient O₃ concentrations in locations where epidemiologic studies have evaluated O₃-associated hospital admissions, emergency department visits, and/or mortality. When considering epidemiologic studies within the context of the current standard, we emphasize those studies conducted in the U.S. and Canada. Such studies reflect air quality and exposure patterns that are likely more typical of the U.S. population than the air quality and exposure patterns reflected in studies conducted outside the U.S. and Canada (section 1.3.1.2, above).⁴³ We also emphasize studies reporting associations with effects judged in the ISA to be robust to confounding by other factors, including co-occurring air pollutants. In addition to these factors, we consider the statistical precision of study results, the extent to which

⁴³ Nonetheless, we recognize the importance of all studies, including international studies, in the ISA's assessment of the weight of the evidence that informs causality determinations.

studies report associations in at-risk populations, and the extent to which the biological plausibility of associations at various ambient O₃ concentrations is supported by controlled human exposure and/or animal toxicological studies. These considerations help inform the range of ambient O₃ concentrations over which we have the most confidence in O₃-associated health effects, and the range of concentrations over which our confidence in such associations is appreciably lower. We place particular emphasis on characterizing those portions of distributions of ambient O₃ concentrations likely to meet the current standard.

In our consideration of these issues, we first address the following question:

- **To what extent have U.S. and Canadian epidemiologic studies reported associations with mortality or morbidity in locations that would have met the current O₃ standard during the study period?**

Addressing this question can provide important insights into the extent to which O₃-health effect associations are present for distributions of ambient O₃ concentrations that would be allowed by the current standard. To the extent O₃ health effect associations are reported in study areas that would have met the current standard, we have greater confidence that the current standard could allow the clearly adverse O₃-associated effects indicated by those studies (e.g., mortality, hospital admissions, emergency department visits).⁴⁴

Epidemiologic studies evaluate statistical associations between variation in the incidence of health outcomes and variation in ambient O₃ concentrations. In many of the O₃ epidemiologic studies assessed in the ISA, ambient concentrations are averaged across multiple monitors within study areas, and in some cases over multiple days. These averages are used as surrogates for the spatial and temporal patterns of O₃ exposures in study populations. In this PA, we refer to these averaged concentrations as “area-wide” O₃ concentrations.

The area-wide concentrations reported in many epidemiologic studies do not identify the actual O₃ exposures that may be eliciting the observed health outcomes. Thus, in considering epidemiologic studies of mortality and morbidity, we are not drawing conclusions regarding single short-duration O₃ concentrations in ambient air that, alone, are eliciting the reported health outcomes. Rather, our focus in this section is to consider what these studies convey regarding the extent to which health effects may be occurring (i.e., as indicated by associations) under air quality conditions meeting the current standard.

⁴⁴ See *ATA III*, 283 F.3d at 370 (EPA justified in revising NAAQS when health effect associations are observed at levels allowed by the NAAQS).

In order to facilitate consideration of the question above, we have identified U.S. and Canadian studies of respiratory hospital admissions, respiratory emergency department visits,⁴⁵ and mortality (total, respiratory, cardiovascular) from the ISA (studies identified from U.S. EPA, 2013, Tables 6-28, 6-42, and 6-53, and section 6.2.8) (Appendix 3B). For each monitor in the areas evaluated by these studies, we have identified the 3-year averages of the annual 4th highest daily maximum 8-hour O₃ concentrations (Appendix 3B).⁴⁶ To provide perspective on whether study cities would have met or violated the current O₃ NAAQS during the study period, these O₃ concentrations were compared to the level of the current standard. Based on this approach, a study city was judged to have met the current standard during the study period if all of the 3-year averages of annual 4th highest 8-hour O₃ concentrations in that area were at or below 75 ppb.

Based on these analyses, the large majority of epidemiologic study areas evaluated would have violated the current standard during study periods (Appendix 3B). Table 3-3 below highlights the subset of U.S. and Canadian studies that evaluated O₃ health effect associations in locations that would have met the current standard during study periods. This includes a U.S. single-city study that would have met the current standard over the entire study period (Mar and Koenig, 2009) and four Canadian multicity studies for which the majority of study cities would have met the current standard over the entire study periods (Cakmak et al., 2006; Dales et al., 2006; Katsouyanni et al., 2009; Stieb et al., 2009).⁴⁷

⁴⁵ Given the inconsistency in results across cardiovascular morbidity studies (U.S. EPA, 2013, section 6.3.2.9), our consideration of the morbidity evidence in this section focuses on studies of respiratory hospital admissions and emergency department visits.

⁴⁶ These concentrations are referred to as “design values.” A design value is a statistic that is calculated at individual monitors and based on 3 consecutive years of data collected from that site. In the case of O₃, the design value for a monitor is based on the 3-year average of the annual 4th highest daily maximum 8-hour O₃ concentration in parts per billion (ppb). For U.S. study areas, we used EPA’s Air Quality System (AQS) (<http://www.epa.gov/ttn/airs/airsaqs/>) to identify design values. For Canadian study areas, we used publically available air quality data from the Environment Canada National Air Pollution Surveillance Network (<http://www.etc-cte.ec.gc.ca/napsdata/main.aspx>). We followed the data handling protocols for calculating design values as detailed in 40 CFR Part 50, Appendix P.

⁴⁷ In addition, a study by Vedal et al. (2003) was included in the 2006 CD (U.S. EPA, 2006). This study reported positive and statistically significant associations with mortality in Vancouver during a time period when the study area would have met the current standard (U.S. EPA, 2007). This study was not highlighted in the ISA in the current review (U.S. EPA, 2013).

Table 3-3. U.S. and Canadian epidemiologic studies reporting O₃ health effect associations in locations that would have met the current standard during study periods.

Authors	Study Results	Cities	Number of cities meeting the current standard over entire study period
Cakmak et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	10 Canadian cities	7
Dales et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	11 Canadian cities	7
Katsouyanni et al. (2009)	Positive and statistically significant associations with respiratory hospital admissions	12 Canadian cities	10
Katsouyanni et al. (2009)	Positive and statistically significant associations with all-cause and cardiovascular mortality ⁴⁸	12 Canadian cities	8
Mar and Koenig (2009)	Positive and statistically significant associations with asthma emergency department visits in children (< 18 years) and adults (> 18 years)	Seattle	1
Stieb et al. (2009)	Positive and statistically significant association with asthma emergency department visits	7 Canadian cities	5

As illustrated in Table 3-3, Mar and Koenig reported health effect associations with asthma emergency department visits in a location that would have met the current standard over the entire study period. This analysis indicates that the current standard would allow the distribution of ambient O₃ concentrations that provided the basis for reported associations with respiratory emergency department visits.

In addition, four multicity studies reported associations with mortality or morbidity when the majority of study locations would have met the current standard over the entire study periods. Thus, the current standard would allow the majority of the distributions of ambient O₃ concentrations that provided the basis for positive and statistically significant associations with mortality or morbidity. Our interpretation of these results 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 O₃ in the subset of locations that would have met the current standard, versus O₃ in the smaller number of locations that would have violated the standard. While there is uncertainty in ascribing the multicity effect estimates reported in these Canadian studies to ambient concentrations that would have met the current

⁴⁸ Katsouyanni et al. (2009) report a positive and statistically significant association with cardiovascular mortality for people aged 75 years or older.

standard, the information in Table 3-3 suggests that reported multicity effect estimates are largely influenced by locations meeting the current standard (i.e., given that most study areas would have met this standard). Together, these U.S. and Canadian epidemiologic studies suggest a relatively high degree of confidence in the presence of associations with mortality and morbidity for ambient O₃ concentrations meeting the current standard.

We next consider the extent to which additional epidemiologic studies of mortality or morbidity, specifically those conducted in locations that violated the current standard, can also inform our consideration of adequacy of the current standard. In doing so, we note that health effect associations reported in epidemiologic studies are influenced by the full distributions of ambient O₃ concentrations, including concentrations below the level of the current standard. We focus on studies that have explicitly characterized such O₃ health effect associations, including confidence in those associations, for various portions of distributions of ambient O₃ concentrations. In doing so, we consider the following question:

- **To what extent do analyses from epidemiologic studies indicate confidence in health effect associations over distributions of ambient O₃ concentrations, including at concentrations lower than previously identified or below the current standard?**

We first focus on those studies that have reported confidence intervals around concentration-response functions over distributions of ambient O₃ concentrations. Confidence intervals around concentration-response functions can provide insights into the range of ambient concentrations over which the study indicates the most confidence in the reported health effect associations (i.e., where confidence intervals are narrowest), and into the range of ambient concentrations below which the study indicates that uncertainty in the nature of such associations becomes notably greater (i.e., where confidence intervals become markedly wider). The concentrations below which confidence intervals become markedly wider in such analyses are intrinsically related to data density, and do not necessarily indicate the absence of an association.

The ISA identifies several epidemiologic studies that have reported confidence intervals around concentration-response functions in U.S. cities. The ISA concludes that studies generally indicate a linear concentration-response relationship “across the range of 8-h max and 24 h avg O₃ concentrations most commonly observed in the U.S. during the O₃ season” and that “there is less certainty in the shape of the C-R curve at the lower end of the distribution of O₃ concentrations” (U.S. EPA, 2013, pp. 2-32 to 2-34). In characterizing the O₃ concentrations below which such certainty decreases, the ISA discusses area-wide O₃ concentrations as low as 20 ppb and as high as 40 ppb (U.S. EPA, 2013, section 2.5.4.4).

Consistent with these conclusions, the range of ambient concentrations over which the evidence indicates the most certainty in concentration-response relationships can vary across

studies. Such variation is likely due at least in part to differences in the O₃ metrics evaluated and differences in the distributions of ambient concentrations and health events. Thus, although consideration of confidence intervals around concentration-response functions can provide valuable insights into the ranges of ambient concentrations over which studies indicate the most confidence in reported health effect associations, there are limitations in the extent to which these analyses can be generalized across O₃ metrics, study locations, study populations, and health endpoints.

The ISA emphasizes two U.S. single-city studies that have reported confidence intervals around concentration-response functions (Silverman and Ito, 2010; Strickland et al., 2010). These studies, and their associated O₃ air quality, are discussed below.

Silverman and Ito (2010) evaluated associations between 2-day rolling average O₃ concentrations⁴⁹ and asthma hospital admissions in New York City from 1999 to 2006 (a time period when the study area would have violated the current standard, Appendix 3B). As part of their analysis, the authors evaluated the shape of the concentration-response relationship for O₃ using a co-pollutant model that included PM_{2.5} (reprinted in Figure 3-4, below). Based on their analyses, Silverman and Ito (2010) concluded a linear relationship between O₃ and hospital admissions is a reasonable approximation of the concentration-response function throughout much of the range of ambient O₃ concentrations. Based on visual inspection of Figure 3-4 below (Figure 3 from published study), we note that confidence in the reported concentration-response relationship is highest for area-wide average O₃ concentrations around 40 ppb (i.e., near the reported median of 41 ppb), and decreases notably for concentrations at and below about 20 ppb.

⁴⁹ 2-day rolling averages of daily maximum 8-hour O₃ concentrations were calculated throughout the study period, averaged across study monitors.

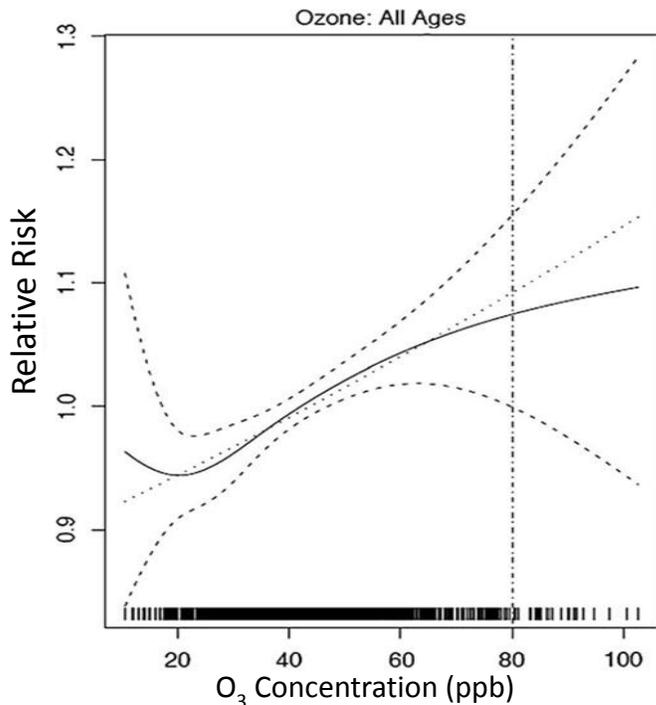


Figure 3-4. Concentration-response function for asthma hospital admissions over the distribution of area-wide averaged O₃ concentrations (adapted from Silverman and Ito, 2010).⁵⁰

In considering the concentration-response function presented by Silverman and Ito (2010) within the context of the adequacy of the current standard, we recognize that true design values cannot be identified for the subsets of air quality data contributing to various portions of the concentration-response function.⁵¹ Therefore, to use this analysis to inform our consideration of the adequacy of the current standard we evaluate the extent to which the concentration-response function indicates a relatively high degree of confidence in the reported health effect association on days when all monitored 8-hour O₃ concentrations were below 75 ppb (Table 3-4, below). This approach can provide insight into the extent to which the reported O₃ health effect association is present when all monitored O₃ concentrations are below the level of the current standard.

Based on the information in Table 3-4 below, when 2-day averaged O₃ concentrations ranged from 36 to 45 ppb (i.e., around the median, where confidence intervals are narrowest), there were 3 days (out of 432) with at least one monitor recording a daily maximum 8-hour O₃ concentration above the level of the current standard (approximately 0.7% of days). When 2-day

⁵⁰This figure was also reprinted in the ISA (U.S. EPA, 2013; Figure 6-16).

⁵¹As discussed above, O₃ design values are calculated using all data available from a monitor.

averaged O₃ concentrations ranged from 26 to 45 ppb (i.e., extending to concentrations below the median, but still above the concentrations where confidence intervals widen notably), there were 4 days (out of 816) with at least one monitor recording a daily maximum 8-hour O₃ concentration above the level of the current standard (approximately 0.5% of days). Thus, on over 99% of the days when area-wide “averaged” O₃ concentrations were between 26 and 45 ppb, the highest daily maximum 8-hour O₃ concentrations were below 75 ppb. For comparison, the annual 4th highest daily maximum 8-hour O₃ concentration generally corresponds to the 98th or 99th percentile of the seasonal distribution, depending on the length of the O₃ season.

Table 3-4. Distributions of daily 8-hour maximum ozone concentrations from highest monitors over range of 2-day moving averages from composite monitors (for study area evaluated by Silverman and Ito, 2010)

Distribution of 8-hr max from highest monitors	2-day moving average across monitors (ppb)								
	11 to 20 (62 days)	21 to 25 (92 days)	26 to 30 (178 days)	31 to 35 (206 days)	36 to 40 (236 days)	41 to 45 (196 days)	46 to 50 (153 days)	51 to 55 (111 days)	56 to 60 (71 days)
Min	15	21	19	26	25	15	31	30	41
5th	16	23	25	33	34	37	38	37	46
25th	20	28	32	38	42	46	51	54	60
50th	24	31	36	43	47	52	59	62	68
75th	29	36	42	47	52	59	65	69	78
95th	37	49	50	55	61	72	77	80	90
98th	41	56	60	71	67	75	85	89	93
99th	41	57	67	75	69	87	91	94	93
Max	42	59	80	75	79	91	97	94	93
Days > 75 ppb	0	0	1	0	1	2	9	15	20

In a separate study, Strickland et al. (2010) evaluated associations between 3-day rolling average O₃ concentrations⁵² and asthma hospital admissions in Atlanta during the warm season from 1994 to 2004 (a time period when the study area would have violated the current standard, Appendix 3B). As part of this analysis, Strickland et al. (2010) evaluated the concentration-response relationship for O₃ and pediatric asthma emergency department visits. The authors reported the shape of the concentration-response function to be approximately linear with no evidence of a threshold when 3-day averaged daily maximum 8-hour O₃ concentrations were approximately 30 to 80 ppb (Figure 3-5 below and U.S. EPA, 2013, Figure 6-18). Figure 3-5 below illustrates that the confidence intervals around the concentration-response function are

⁵² Three-day rolling averages of population-weighted daily maximum 8-hour O₃ concentrations were calculated throughout the study period (Strickland et al., 2010).

narrowest around the study mean (i.e., 55 ppb), and that these confidence intervals do not widen notably for “averaged” O₃ concentrations as low as about 30 ppb.

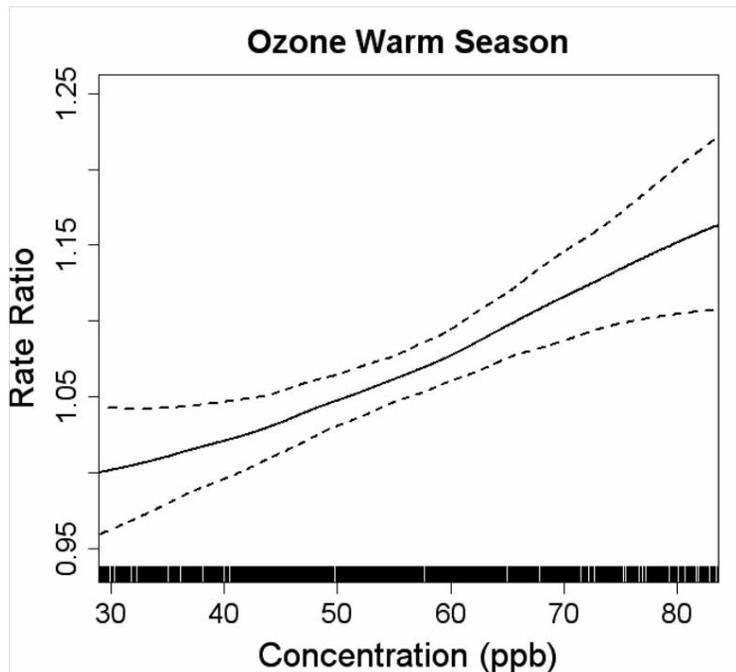


Figure 3-5. Concentration-response function for pediatric asthma emergency department visits over the distribution of averaged, population-weighted 8-hour O₃ concentrations (reprinted from Strickland et al., 2010).⁵³

Similar to the study by Silverman and Ito (2010), we consider the extent to which the reported concentration-response function indicates a relatively high degree of confidence in health effect associations on days when all monitored 8-hour O₃ concentrations are below 75 ppb (Table 3-5, below).⁵⁴ In considering the information presented in Table 3-5, we first note that when 3-day averaged O₃ concentrations were in the range of the mean (i.e., 51 to 60 ppb), there were 77 days (out of 516; 14.9%) with at least one monitor recording a daily maximum 8-hour O₃ concentration above the level of the current standard. In contrast, during the 519 days when averaged O₃ concentrations were in the lower portion of the distribution where study authors indicate relatively high confidence in the reported concentration-response relationship (i.e., between 31 and 45 ppb), there were 4 days with at least one monitor in the study area measuring a daily maximum 8-hour O₃ concentration greater than 75 ppb (approximately 0.8% of days).

⁵³ This figure was also reprinted in the ISA (U.S. EPA, 2013; Figure 6-18).

⁵⁴ The study by Strickland et al. (2010) used five monitors. For our evaluation of highest daily maximum 8-hour concentrations (i.e., from the individual monitor recording the highest such concentration), we obtained information from the four of these study area monitors that report data to AQS (Appendix 3B).

Thus, on over 99% of the days when “averaged” O₃ concentrations were between 31 and 45 ppb, all monitors measured daily maximum 8-hour O₃ concentrations below 75 ppb.

Table 3-5. Distribution of daily 8-hour maximum ozone concentrations from highest monitors over range of 3-day moving averages of population-weighted concentrations (for study area evaluated by Strickland et al., 2010)

Distribution of 8-hr max from highest monitors	3-day moving average across monitors (ppb)						
	26-30 (75 days)	31-35 (144 days)	36-40 (165 days)	41-45 (210 days)	46-50 (235 days)	51-55 (244 days)	56-60 (272 days)
Min	15	18	21	19	25	23	25
5th	20	22	28	27	35	38	39
25th	29	31	35	37	46	52	54
50th	33	37	42	47	52	60	63
75th	40	44	49	56	62	67	73
95th	53	55	68	69	75	80	88
98th	67	59	74	72	83	88	95
99th	68	63	78	78	85	95	99
Max	70	64	82	90	92	97	106
Days > 75	0	0	2	2	10	24	53

In summary, analyses of air quality data from the study locations evaluated by Silverman and Ito (2010) and Strickland et al. (2010) indicate a relatively high degree of confidence in reported statistical associations with respiratory health outcomes on days when virtually all monitored 8-hour O₃ concentrations were 75 ppb or below. Though these analyses do not identify true design values, the presence of O₃-associated respiratory effects on such days provides insight into the types of health effects that could occur in locations with maximum ambient O₃ concentrations below the level of the current standard.

We next consider the following question:

- **To what extent are there important uncertainties in analyses of confidence in concentration-response functions?**

There are several important uncertainties that are specifically related to our analyses of distributions of O₃ air quality in the study locations evaluated by Silverman and Ito (2010) and Strickland et al. (2010). Although these studies report health effect associations with two-day (Silverman and Ito) and three-day (Strickland) averages of daily O₃ concentrations, it is possible that the respiratory morbidity effects reported in these studies were also at least partly attributable to the days immediately preceding these two- and three-day periods. In support of this possibility, Strickland et al. reported positive and statistically significant associations with emergency department visits for multiple lag periods, including lag periods exceeding three days.

Our analysis of highest monitored concentrations focuses on two- and three- day periods, as used in the published study to generate concentration-response functions. This could have important implications for our interpretation of the reported concentration-response functions if a 2-day period with no monitors measuring 8-hour concentrations at or above 75 ppb is immediately preceded by one or more days with monitors that do exceed 75 ppb. Although we do not know the extent to which O₃ concentrations on a larger number of days could have contributed to reported health effect associations, we note this as a potentially important uncertainty in our consideration of concentration-response functions within the context of the current standard.

In addition, an important uncertainty that applies to epidemiologic studies in general is the extent to which reported health effects are caused by exposures to O₃ itself, as opposed to other factors such as co-occurring pollutants or other pollutant mixtures. Although both of the studies evaluated above reported health effect associations in co-pollutant models, this uncertainty becomes an increasingly important consideration as health effect associations are evaluated at lower ambient O₃ concentrations (i.e., presumably corresponding to lower exposure concentrations).

One approach to considering the potential importance of this uncertainty in epidemiologic studies is to evaluate the extent to which there is coherence with the results of experimental studies (i.e., in which the study design dictates that exposures to O₃ itself are responsible for reported effects). Therefore, in further considering uncertainties associated with the above air quality analyses for the study areas evaluated by Silverman and Ito (2010) and Strickland et al. (2010), we evaluate the following question:

- **To what extent is there coherence between evidence from controlled human exposure studies and epidemiologic studies supporting the occurrence of O₃-attributable respiratory effects when daily maximum 8-hour ambient O₃ concentrations are at or below 75 ppb?**

As summarized above and as discussed in the ISA (U.S. EPA, 2013, section 6.2), controlled human exposure studies demonstrate the occurrence of respiratory effects in an appreciable percentage of healthy adults following single short-term exposures to O₃ concentrations as low as 60 ppb. As O₃ exposure concentrations exceed 60 ppb: 1) effects in healthy adults become larger and more serious; 2) a broader range of effects are observed in a greater percentage of exposed individuals; and 3) effects are reported more consistently across studies. In addition, exposure concentrations below 60 ppb could potentially result in respiratory effects, particularly in at-risk populations such as children and asthmatics. Thus, as the potential increases for portions of epidemiologic study populations to be exposed to O₃ concentrations approaching or exceeding 60 ppb, our confidence increases that reported respiratory health effects could be caused by exposures to the ambient O₃ concentrations present in study locations.

As discussed above, for the study by Silverman and Ito (2010), 26 to 45 ppb represents the lower end of the range of “averaged” concentrations over which the study indicates a relatively high degree of confidence in the statistical association with respiratory hospital admissions (and for which virtually all monitored concentrations were 75 ppb or below). As averaged concentrations increase from 26 to 45 ppb, the number of days with maximum monitored concentrations approaching or exceeding 60 ppb increases (Table 3-4, above).⁵⁵ For example, of the 178 days with area-wide average concentrations from 26 to 30 ppb, only about 5% had monitors recording ambient concentrations of 50 ppb or greater and about 2% had monitors recording concentrations of 60 ppb or greater. In contrast, of the 196 days with area-wide average concentrations from 41 to 45 ppb, about half had one or more monitors recording ambient concentrations above 50 ppb and about 25% had monitors recording concentrations at or above 60 ppb. On a small number of these days, at least one monitored concentration exceeded 70 or 80 ppb. Thus as averaged concentrations approach 45 ppb there is an increasing likelihood that at least some portion of the Silverman and Ito study population could have been exposed to O₃ concentrations near or above those shown to cause respiratory effects in healthy adults. If these effects become serious enough (e.g., in people with asthma) they could lead to the respiratory-related hospital admissions reported in the study. This analysis is consistent with the occurrence of O₃-attributable respiratory hospital admissions, even when virtually all monitored concentrations were below the level of the current standard. Similar results were obtained for the study by Strickland et al. (2010) (Table 3-5, above).

In further evaluating O₃ concentration-response relationships within the context of the adequacy of the current standard, we note that some epidemiologic studies report health effect associations for air quality subsets restricted to ambient pollutant concentrations below one or more predetermined cut points. Such “cut point” analyses can provide information on the magnitude and statistical precision of effect estimates for defined distributions of ambient concentrations, which may in some cases include distributions that would meet the current standard. Therefore, we next consider the following question:

- **To what extent do cut-point analyses from epidemiologic studies report health effect associations at ambient O₃ concentrations lower than previously identified or that would likely meet the current standard?**

By considering the magnitude and statistical significance of effect estimates for restricted air quality distributions, cut-point analyses can provide insight into the extent to which health

⁵⁵ Though, as noted above, the epidemiologic studies by Silverman and Ito (2010) and Strickland et al. (2010) do not provide information on the extent to which reported health effects result from exposures to any specific O₃ concentrations.

effect associations are driven by ambient concentrations above the cut point, versus concentrations below the cut point. For studies that evaluate multiple cut points, these analyses can provide insights into the magnitude and statistical precision of health effect associations for different portions of the distribution of ambient concentrations, including insights into the ambient concentrations below which uncertainty in reported associations becomes notably greater. As with analyses of concentration-response functions, discussed above, the cut points below which confidence intervals become notably wider depend in large part on data density.⁵⁶

In the U.S. multicity study by Bell et al. (2006), study authors used the NMMAPS data set to evaluate associations between 2-day rolling average O₃ concentrations⁵⁷ and total (non-accidental) mortality in 98 U.S. cities from 1987 to 2000. Based on the full distributions of ambient O₃ concentrations in study cities, the large majority of the NMMAPS cities would have violated the current standard during the study period (Appendix 3B). However, Bell et al. (2006) also reported health effect associations in a series of cut-point analyses, with effect estimates based only on the subsets of days contributing to “averaged” O₃ concentrations below cut points ranging from 5 to 60 ppb (see Figure 2 in Bell et al., 2006). The lowest cut-point for which the association between O₃ and mortality was reported to be statistically significant was 30 ppb (based on visual inspection of Figure 2 in the published study). As with the studies by Silverman and Ito (2010) and Strickland et al. (2010), discussed above, we consider what these cut point analyses indicate with regard to the potential for health effect associations to extend to ambient O₃ concentrations likely to be allowed by the current O₃ NAAQS.

We attempted to recreate the subsets of air quality data used in the cut point analyses presented by Bell et al. (2006). In doing so, we applied the criteria described in the published study to generate air quality subsets corresponding to those defined by the cut points evaluated by study authors.⁵⁸ From the days with averaged O₃ concentrations below each cut point, we identified 3-year averages of annual 4th highest daily maximum 8-hour O₃ concentrations in each study area. We then compared these 4th highest O₃ concentrations to the level of the current standard in order to provide insight into the extent to which the air quality distributions included in various cut point analyses would likely have met the current standard.

⁵⁶ As such, these analyses provide insight into the ambient concentrations below which the available air quality information becomes too sparse to support conclusions about the nature of concentration-response relationships, with a high degree of confidence.

⁵⁷ Two-day rolling averages of 24-hour average O₃ concentrations were calculated throughout the study period. This calculation was done across study monitors in study cities with multiple monitors.

⁵⁸ We were unable to obtain the air quality data used to generate the cut-point analyses in the study published by Bell et al. (2006). Therefore, we generated 2-day averages of 24-hour O₃ concentrations in study locations using the air quality data available in AQS, combined with the published description of study area definitions. In doing so, we did not recreate the trimmed means used by Bell. As discussed below, this represents an important uncertainty in our analysis.

We particularly focus on the lowest cut-point for which the association between O₃ and mortality was reported in this study to be statistically significant (i.e., 30 ppb, as noted above). Based on the O₃ air quality concentrations that met the criteria for inclusion in the 30 ppb cut point analysis, 95% of study areas had 3-year averages of annual 4th highest daily maximum 8-hour O₃ concentration at or below 75 ppb over the entire study period. For the 35 ppb cut point, which also resulted in a statistically significant association with mortality, 68% of study areas had 3-year averages of annual 4th highest daily maximum 8-hour O₃ concentration at or below 75 ppb. This suggests that the large majority of air quality distributions that provided the basis for positive and statistically significant associations with mortality (i.e., for the 30 and 35 ppb cut points) would likely have met the current O₃ standard. For higher cut points, all of which also resulted in statistically significant associations with mortality, the majority of study cities had 3-year averages of annual 4th highest daily maximum 8-hour concentrations greater than 75 ppb.

Table 3-6. Number of study cities with 4th highest daily maximum 8-hour concentrations greater than 75 ppb, for various cut-point analyses presented in Bell et al. (2006)

	Cut-point for 2-day moving average across monitors and cities (24-hour average)								
	25	30	35	40	45	50	55	60	All
Number (%) of Cities with 4 th highest ≥ 75 (any 3-yr period; 1987-2000)	0 (0%)	5 (5%)	31 (32%)	70 (71%)	86 (88%)	88 (90%)	92 (94%)	92 (94%)	92 (94%)

In addition to the uncertainties noted above for our analysis of the single-city studies by Silverman and Ito (2010) and Strickland et al. (2010) (e.g., attributing effects specifically to air quality included in various subsets), an important uncertainty related to this analysis is that we were unable to obtain the air quality data used to generate the cut-point analyses in the study published by Bell et al. (2006). Therefore, as noted above, we generated 2-day averages of 24-hour O₃ concentrations in study locations using the air quality data available in AQS, combined with the published description of study area definitions. In doing so, we did not recreate the trimmed means used by Bell. An important uncertainty in this approach is the extent to which we were able to appropriately recreate the cut-point analyses in the published study.

The ISA also notes important uncertainties inherent in multicity studies that evaluate the potential for thresholds to exist, as was done in the study by Bell et al. (2006). Specifically, the ISA highlights the regional heterogeneity in O₃ health effect associations as a factor that could obscure the presence of thresholds, should they exist, in multicity studies (U.S. EPA, 2013, sections 2.5.4.4 and 2.5.4.5). The ISA notes that community characteristics (e.g., activity patterns, housing type, age distribution, prevalence of air conditioning) could be important contributors to reported regional heterogeneity (U.S. EPA, 2013, section 2.5.4.5). Given this heterogeneity, the ISA concludes that “a national or combined analysis may not be appropriate to identify whether a threshold exists in the O₃-mortality C-R relationship” (U.S. EPA, 2013, p. 2-33). This represents an important source of uncertainty when characterizing our confidence in reported concentration-response relationships over distributions of ambient O₃ concentrations, based on multicity studies. This uncertainty becomes increasingly important when interpreting concentration-response relationships at lower ambient O₃ concentrations, particularly those concentrations corresponding to portions of distributions where data density decreases notably.

3.1.4.3 Concentrations in Epidemiologic Studies – “Long-term” Metrics

We next consider the extent to which epidemiologic studies employing longer-term ambient O₃ concentration metrics inform our understanding of the air quality conditions

associated with O₃-attributable health effects, and specifically inform consideration of the extent to which such effects could occur under air quality conditions meeting the current standard. Unlike for the studies of short-term O₃ discussed above, the available U.S. and Canadian epidemiologic studies evaluating long-term ambient O₃ concentration metrics have not been conducted in locations likely to have met the current 8-hour O₃ standard during the study period (Appendix 3B). Therefore, although these studies contribute to our understanding of health effects associated with long-term or repeated exposures to ambient O₃ (as summarized in section 3.1.2 above), consideration of study area design values does not inform our consideration of the extent to which those health effects may be occurring in locations that met the current standard.

In further considering epidemiologic studies of long-term O₃ concentrations, we also evaluate the extent to which concentration-response functions, including associated confidence intervals, have been characterized for distributions of ambient O₃, and what those functions can tell us about health effect associations for O₃ concentrations likely to be allowed by the current standard. Specifically, we consider the following question:

- **To what extent do confidence intervals around concentration-response functions indicate O₃-associated health outcomes at ambient concentrations meeting the current O₃ standard?**

The ISA identifies a single epidemiologic study reporting confidence intervals around a concentration-response function for “long-term” O₃ concentrations and respiratory mortality (Jerrett et al., 2009; U.S. EPA, 2013, sections 7.2.7, 7.2.8 and 7.7). Jerrett et al. (2009) reported that when seasonal averages of 1-hour daily maximum O₃ concentrations⁵⁹ ranged from 33 to 104 ppb, there was no statistical deviation from a linear concentration-response relationship between O₃ and respiratory mortality across 96 U.S. cities (U.S. EPA, 2013, section 7.7). However, the authors reported “limited evidence” for an effect threshold at an O₃ concentration of 56 ppb (p=0.06).⁶⁰ Visual inspection of this concentration-response function (Figure 3-6) confirms the possibility of an inflection point just below 60 ppb, which is close to the median concentration across cities (i.e., 57 ppb).

⁵⁹ Jerrett et al. (2009) evaluated the April to September averages of 1-hour daily maximum O₃ concentrations across 96 U.S. metropolitan areas from 1977- 2000. In urban areas with multiple monitors, April to September 1-hour daily maximum concentrations from each individual monitor were averaged. This step was repeated for each year in the study period. Finally, each yearly averaged O₃ concentrations was then averaged again to yield the single averaged 1-hour daily maximum O₃ concentration depicted on the x axis of Figure 3-6 below.

⁶⁰ This issue is discussed further in section 3.2.3.2, below.

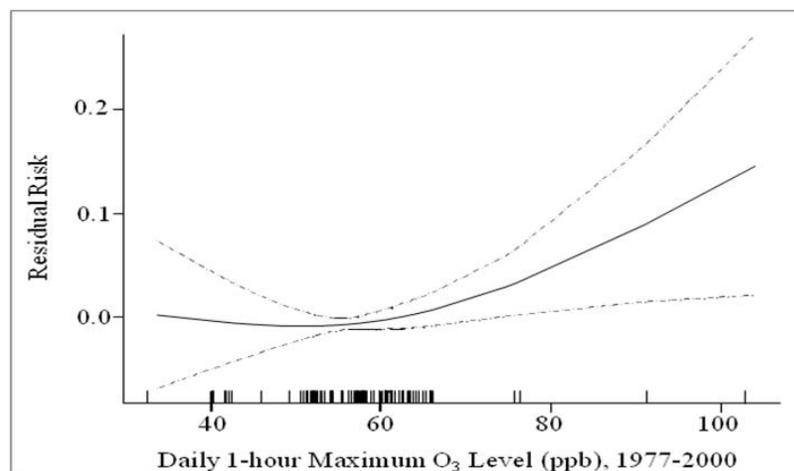


Figure 3-6. Exposure-Response relationship between risk of death from respiratory causes and ambient O₃ concentration study metric (Jerrett et al., 2009).

We consider the extent to which this concentration-response function indicates confidence in the reported health effect association at various ambient O₃ concentrations. In identifying the concentrations over which we have the greatest confidence, we note the following: (1) most of the study cities had O₃ concentrations above 53.1 ppb (i.e., the upper bound of the first quartile), accounting for approximately 72% of the respiratory deaths in the cohort (Table 2 in Jerrett et al. 2009); (2) confidence intervals widen notably for O₃ concentrations in the first quartile (based on visual inspection of Figure 3-6); and (3) study authors noted limited evidence for a threshold at 56 ppb.⁶¹ In considering this information, we conclude that the analysis reported by Jerrett indicates a relatively high degree of confidence in the linear concentration-response function for “long-term” O₃ concentrations at least as low as 56 ppb, and notably decreased confidence in the linear function for concentrations at or below about 53 ppb (i.e., the upper bound of the first quartile of O₃ concentrations).

Based on information in the published study (Figure 1 in Jerrett et al., 2009), we identified 72 of the 96 study cities as having ambient O₃ concentrations in the highest three quartiles (Appendix 3B). As noted above, these 72 cities account for approximately 72% of the respiratory deaths in the cohort (Table 2 in Jerrett et al. 2009). Of these 72 cities, 71 had 3-year averages of annual 4th highest daily maximum 8-hour O₃ concentrations above 75 ppb (Appendix 3B). Thus, the current 8-hour NAAQS would have been violated during the study period in virtually all of the study cities that contribute to the range of long-term O₃ concentrations over which we have the greatest confidence in the reported relationship with respiratory mortality.

⁶¹ The ISA does not reach conclusions regarding the potential for a threshold in the association between “long-term” O₃ concentrations and respiratory mortality.

Thus, while the study by Jerrett et al. (2009) contributes to our understanding of health effects associated with ambient O₃ (as summarized in section 3.1.2 above), it is less informative regarding the extent to which those health effects may be occurring under air quality conditions allowed by the current standard.

3.1.5 Public Health Implications

In this section, we address the public health implications of O₃ exposures with respect to the factors that put populations at increased risk from exposures (section 3.1.5.1), the size of at-risk populations (section 3.1.5.2), and the potential effects of averting behavior on reducing O₃ exposures and associated health effects (section 3.1.5.3). Providing appropriate public health protection requires consideration of the factors that put populations at greater risk from O₃ exposure. In order to estimate potential public health impacts, it is important to consider not only the adversity of the health effects, but also the populations at greater risk and potential behaviors that may reduce exposure.

3.1.5.1 At-Risk Populations

In this section we address the following question:

- **To what extent does the currently available scientific evidence expand our understanding of at-risk populations?**

The currently available evidence expands our understanding of populations that were identified to be at greater risk of O₃-related health effects at the time of the last review (i.e., people who are active outdoors, people with lung disease, children and older adults and people with increased responsiveness to O₃) and supports the identification of additional factors that may lead to increased risk (U.S. EPA, 2006, section 3.6.2; U.S. EPA, 2013, chapter 8). Populations and lifestages may be at greater risk for O₃-related health effects due to factors that contribute to their susceptibility and/or vulnerability to ozone. The definitions of susceptibility and vulnerability have been found to vary across studies, but in most instances “susceptibility” refers to biological or intrinsic factors (e.g., lifestage, sex, preexisting disease/conditions) while “vulnerability” refers to non-biological or extrinsic factors (e.g., socioeconomic status [SES]) (U.S. EPA, 2013, p. 8-1). In some cases, the terms “at-risk” and “sensitive” have been used to encompass these concepts more generally. In the ISA and this PA, “at-risk” is the all-encompassing term used to define groups with specific factors that increase their risk of O₃-related health effects. Further discussion of at-risk populations can be found below.

There are multiple avenues by which groups may experience increased risk for O₃-related health effects. A population or lifestage⁶² may exhibit greater effects than other populations or lifestages exposed to the same concentration or dose, or they may be at greater risk due to increased exposure to an air pollutant (e.g., time spent outdoors). A group with intrinsically increased risk would have some factor(s) that increases risk through a biological mechanism and, in general, would have a steeper concentration-risk relationship, compared to those not in the group. Factors that are often considered intrinsic include pre-existing asthma, genetic background, and lifestage. A group of people could also have extrinsically increased risk, which would be through an external, non-biological factor, such as socioeconomic status (SES) and diet. Some groups are at risk of increased internal dose at a given exposure concentration, for example, because of breathing patterns. This category would include people who work or exercise outdoors. Finally, there are those who might be placed at increased risk for experiencing greater exposures by being exposed to higher O₃ concentrations. This would include, for example, groups of people with greater exposure to ambient O₃ due to less availability or use of home air conditioners such that they are more likely to be in locations with open windows on high ozone days. Some groups may be at increased risk of O₃-related health effects through a combination of factors. For example, children tend to spend more time outdoors when O₃ levels are high, and at higher levels of activity than adults, which leads to increased exposure and dose, and they also have biological, or intrinsic, risk factors (e.g., their lungs are still developing) (U.S. EPA, 2013, Chapter 8). An at-risk population or lifestage is more likely to experience adverse health effects related to O₃ exposures and/or, develop more severe effects from exposure than the general population.

People with Specific Genetic Variants

Overall, for variants in multiple genes there is adequate evidence for involvement in populations being more at-risk than others to the effects of O₃ exposure on health (U.S. EPA, 2013, section 8.1). Controlled human exposure and epidemiologic studies have reported evidence of O₃-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 O₃-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.

People with Asthma

⁶² Lifestages, which in this case includes childhood and older adulthood, are experienced by most people over the course of a lifetime, unlike other factors associated with at-risk populations.

Previous O₃ AQCDs identified individuals with asthma as a population at increased risk of O₃-related health effects. Multiple new epidemiologic studies included in the ISA have evaluated the potential for increased risk of O₃-related health effects in people with asthma, including: lung function; symptoms; medication use; airway hyperresponsiveness (AHR); and airway inflammation (also measured as exhaled nitric oxide fraction, or FeNO). A study of lifeguards in Texas reported decreased lung function with short-term O₃ 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 O₃ 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 AHR (an obligate condition among asthmatics) reported greater short-term O₃-associated decreases in lung function in elderly individuals with AHR, especially among those who were obese (Alexeeff et al., 2007). With respect to airway inflammation, in one study, a positive association was reported for airway inflammation among asthmatic children following short-term O₃ exposure, but the observed association was similar in magnitude to that of non-asthmatics (Barraza-Villarreal et al., 2008). Similarly, another study of children in California reported an association between O₃ concentration and 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 O₃ exposure and altered lung function for either asthmatic or non-asthmatic adults, but did note a decrease in lung function among individuals with allergies.

New evidence for difference in effects among asthmatics has been observed in studies that examined the association between O₃ 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 O₃ and lung function for corticosteroid users compared with noncorticosteroid users (Lewis et al., 2005). Conversely, another study found decreased lung function among noncorticosteroid users compared to 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 O₃ exposure among corticosteroid users and non-users in a study taking place during the winter months in Canada (Liu et al., 2009). Additionally, a study of airway inflammation reported a counterintuitive inverse association with O₃ 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 O₃ on adults with asthma and healthy controls are limited. Based on studies reviewed in the 1996 and 2006 O₃ AQCDs, subjects with asthma appeared to be more sensitive to acute effects of O₃ in terms of

FEV₁ and inflammatory responses than healthy non-asthmatic subjects. For instance, Horstman et al. (1995) observed that mild-to-moderate asthmatics, on average, experienced double the O₃-induced FEV₁ decrement of healthy subjects (19% versus 10%, respectively, $p = 0.04$). Moreover, a statistically significant positive correlation between FEV₁ responses to O₃ exposure and baseline lung function was observed in individuals with asthma, i.e., responses increased with severity of disease. Minimal evidence exists suggesting that individuals with asthma have smaller O₃-induced FEV₁ 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 FEV₁ responses to O₃ exposure diminish with age. Individuals with asthma also had significantly more neutrophils in the BALF (18 hours postexposure) than similarly exposed healthy individuals (Peden et al., 1997; Scannell et al., 1996; Basha et al., 1994). Furthermore, a study examining the effects of O₃ 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 O₃-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 O₃ having greater effects on innate and adaptive immunity in these asthmatic individuals. In addition, studies have demonstrated that O₃ 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 the enhanced sensitivity to O₃ in subjects with asthma (see Section 5.4.2.2 in the ISA).

Toxicological studies provide additional evidence of the biological basis for the greater effects of O₃ among those with asthma or AHR (U.S. EPA, 2013, section 8.2.2). In animal toxicological studies, an asthmatic phenotype is modeled by allergic sensitization of the respiratory tract. Many of the studies that provide evidence that O₃ 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. Similarly, adverse changes in pulmonary function were demonstrated in mice exposed to O₃; enhanced inflammatory responses were in rats exposed to O₃, but only in animals sensitized to allergen. In general, it is the combined effects of O₃ and allergic sensitization which result in measurable effects on pulmonary function. In a pulmonary fibrosis model, exposure O₃ for 5 days increased pulmonary inflammation and fibrosis, along with the frequency of

bronchopneumonia in rats. Thus, short-term exposure to O₃ may enhance damage in a previously injured lung (U.S. EPA, 2013, section 8.2.2).

In the 2006 O₃ AQCD, the potential for individuals with asthma to have greater risk of O₃-related health effects was supported by a number of controlled human exposure studies, evidence from toxicological studies, and a limited number of epidemiologic studies. In section 8.2.2, the ISA reports that 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 O₃ exposure and altered lung function by corticosteroid use provided limited evidence of O₃-related health effects. However, 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 O₃ 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 detriments in FEV₁ and greater inflammatory responses to O₃ in individuals with asthma than in healthy individuals without a history of asthma. The collective evidence for increased risk of O₃-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 O₃ exposure. Overall, the ISA finds there is adequate evidence for asthmatics to be an at-risk population.

Children

Children are considered to be at greater risk from O₃ exposure because their respiratory systems undergo lung growth until about 18-20 years of age and are therefore thought to be intrinsically more at risk for O₃-induced damage (U.S. EPA, 2006). It is generally recognized that children spend more time outdoors than adults, and therefore would be expected to have higher exposure to O₃ than adults. The ventilation rates also vary between children and adults, particularly during moderate/heavy activity. Children aged 11 years and older and adults have higher absolute ventilation rates than children aged 1-11 years. However, children have higher ventilation rates relative to their lung volumes, which tends to increase dose normalized to lung surface area. Exercise intensity has a substantial effect on ventilation rate, with high intensity activities resulting in nearly double the ventilation rate during moderate activity among children and those adults less than 31 years of age. For more information on time spent outdoors and ventilation rate differences by age group, see Section 4.4.1 in the ISA (U.S. EPA, 2013).

The 1996 O₃ AQCD reported clinical evidence that children, adolescents, and young adults (<18 years of age) appear, on average, to have nearly equivalent spirometric responses to

O₃ exposure, but have greater responses than middle-aged and older adults (U.S. EPA, 1996). Symptomatic responses (e.g., cough, shortness of breath, pain on deep inspiration) to O₃ exposure, however, appear to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996). 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.

Recent epidemiologic studies have examined different age groups and their risk to O₃-related respiratory hospital admissions and emergency department (ED) visits. Evidence for greater risk in children was reported in several studies. A study in Cyprus of short-term O₃ concentrations and respiratory hospital admissions (HA) 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-ED visits reported the strongest O₃-related associations among 5- to 14-year olds compared to the other age groups (ages examined 0-75+) (Villeneuve et al., 2007). Greater O₃-associated risk in asthma-related ED 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 O₃ 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 O₃ exposure and asthma HA 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., 2008b). 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 O₃-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 O₃ 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 O₃ 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 O₃ among both the adult and infant monkeys (Plopper et al., 2007). In addition, the lung structure of the conducting airways in the infant monkeys was significantly stunted by O₃ and this aberrant development was persistent 6 months postexposure (Fanucchi et al., 2006).

Age may also affect the inflammatory response to O₃ 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 (U.S. EPA, 2013, p. 8-18).

The previous and recent human clinical and toxicological studies reported evidence of increased risk from O₃ 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 potentially at increased risk of O₃-related health effects.

Older Adults

The ISA notes that older adults are at greater risk of health effects associated with O₃ exposure through a variety of intrinsic pathways (U.S. EPA, 2013, 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. Older adults also have somewhat lower ventilation rates than adults aged 31 - less than 61 years. For more information on time spent outdoors and ventilation rate differences by age group, see Section 4.4 in the ISA (U.S. EPA, 2013). The gradual decline in physiological processes that occur with aging may lead to increased risk of O₃-related health effects (U.S. EPA, 2006). Respiratory symptom responses to O₃ exposure appears to increase with age until early adulthood and then gradually decrease with increasing age (U.S. EPA, 1996); lung function responses to O₃ exposure also decline from early adulthood (U.S. EPA, 1996). The reductions of these responses with age may put older adults at increased risk for continued O₃ exposure. 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 O₃-related health effects (U.S. EPA, 2013, 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 O₃-related health effects (SSDAN CensusScope, 2010; DeNavas-Walt et al., 2011).

The majority of recent studies reported greater effects of short-term O₃ exposure and mortality among older adults, which is consistent with the findings of the 2006 O₃ 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 O₃-related mortality risk that gets larger with increasing age starting at age 51 (Zanobetti and Schwartz, 2008a). 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 O₃ exposure and mortality in adults aged < 64 years old and 65 to 74 years old, but the risk was increased among older age groups (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 O₃ 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 O₃ 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 < 75 years 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 O₃ exposure and hospital admissions, a positive association was reported between short-term O₃ 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 O₃ 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 O₃ levels and COPD ED 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 O₃ exposure and mortality in older adults. The evidence from mortality studies is consistent with the results reported in the 2006 O₃ 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 (U.S. EPA, 2013)

concludes adequate evidence is available indicating that older adults are at increased risk of O₃-related health effects.

People with Diets Lower in Vitamins C and E

Diet was not examined as a factor potentially affecting risk in previous O₃ AQCDs, but recent studies have examined modification of the association between O₃ and health effects by dietary factors. Because O₃ 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 O₃-related health effects. Supplementation with vitamins C and E has been investigated in a number of studies as a means of inhibiting O₃-mediated damage.

Two epidemiologic studies have examined effect measure modification by diet and found evidence that certain dietary components are related to the effect O₃ 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 O₃ 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 O₃ 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 (U.S. EPA, 2013), that have shown that the first line of defense against oxidative stress is antioxidants-rich extracellular lining fluid (ELF) which scavenge free radicals and limit lipid peroxidation. Exposure to O₃ depletes antioxidant levels in nasal ELF probably due to scrubbing of O₃; however, the concentration and the activity of antioxidant enzymes either in ELF or plasma do not appear to be related to O₃ 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 O₃ 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 (U.S. EPA, 2013) provide evidence of biological plausibility to the epidemiologic and controlled human exposure studies.

There is adequate evidence that individuals with diets lower in vitamins C and E are at risk for O₃-related health effects. The evidence from epidemiologic studies is supported by controlled human exposure and toxicological studies.

Outdoor Workers

Studies included in the 2006 O₃ 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 O₃ exposure and respiratory health outcomes in these groups (U.S. EPA, 2006). Outdoor workers are exposed to ambient O₃ concentrations for a greater period of time than individuals who spend their days indoors. As discussed in Section 4.7 of the ISA (U.S. EPA, 2013) 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 FEV₁ responses to O₃ exposure and minute ventilation is discussed more fully in Section 6.2.3.1 of the 2006 O₃ AQCD.

Previous studies have shown that increased exposure to O₃ due to outdoor work leads to increased risk of O₃-related health effects, specifically decrements in lung function (U.S. EPA, 2006). The strong evidence from the 2006 O₃ AQCD which demonstrated increased exposure, dose, and ultimately risk of O₃-related health effects in this population supports the conclusion that there is adequate evidence to indicate that increased exposure to O₃ through outdoor work increases the risk of O₃-related health effects.

In some cases, it is difficult to determine a factor that results in increased risk of effects. For example, previous assessments have included controlled human exposure studies in which some healthy individuals demonstrate greater O₃-related health effects compared to other healthy individuals. Intersubject variability has been observed for lung function decrements, symptomatic responses, pulmonary inflammation, AHR, and altered epithelial permeability in healthy adults exposed to O₃ and these results tend to be reproducible within a given individual over a period of several months indicating differences in the intrinsic responsiveness. In many cases the reasons for the variability is not clear. This may be because one or some of the factors described above have not been evaluated in studies, or it may be that additional, unidentified factors influence individual responses to O₃ (U.S. EPA, 2013, section 8.5).

As discussed in chapter 8 of the ISA the challenges and limitations in evaluating the factors that can increase risk for experiencing O₃-related health effects may contribute to a lack of information about the factors that may increase risk from O₃ exposures. This lack of information may contribute to conclusions that evidence for some factors, such as sex, SES, and obesity provided “suggestive” evidence of increased risk, or that for a number of factors the evidence was inadequate to draw conclusions about potential increase in risk of effects. Overall, the factors for which the ISA concludes there is adequate evidence of increased risk for

experiencing O₃-related effects were related to asthma, lifestage (children and older adults), genetic variability, dietary factors, and working outdoors.

3.1.5.2 Size of At-Risk Populations and Lifestages in the United States

One consideration in the assessment of potential public health impacts is the size of various population groups for which there is adequate evidence of increased risk for health effects associated with O₃-related air pollution exposure. The factors for which the ISA judged the evidence to be “adequate” with respect to contributing to increased risk of O₃-related effects among various populations and lifestages included: asthma; childhood and older adulthood; diets lower in vitamins C and E; certain genetic variants and, working outdoors (U.S. EPA, 2013, section 8.5).

With regard to asthma, Table 3-7 below summarizes information on the prevalence of current asthma by age in the U.S. adult population in 2010 (Schiller et al., 2012; children - Bloom et al., 2011). Individuals with current asthma constitute a fairly large proportion of the population, including more than 25 million people. Asthma prevalence tends to be higher in children than adults.

Within the U.S., approximately 8.2% of adults have reported currently having asthma (Schiller et al., 2012) and 9.5% of children have reported currently having asthma (Bloom et al., 2011). Table 3-12 below provides more detailed information on prevalence of asthma by age in the U.S.

Table 3-7. Prevalence of asthma by age in the U.S.

Age (years)	N (in thousands)	Percent
0-4	1,285	6.0
5-11	3,020	10.5
12-17	2,672	10.9
18-44	8,902	8.1
45-64	6,704	8.4
65-74	1,849	8.7
75+	1,279	7.4

Asthma prevalence is reported for “still has asthma”

Source: Statistics for adults: Schiller et al. (2012); Statistics for children: Bloom et al. (2011)

With regard to lifestyles, based on U.S. census data from 2010 (Howden and Meyer, 2011), about 74 million people, or 24% of the U.S. population, are under 18 years of age and more than 40 million people, or about 13% of the U.S. population, are 65 years of age or older. Hence, a large proportion of the U.S. population, more than 33%, is included in age groups that are considered likely to be at increased risk for health effects from ambient O₃ exposure.

With regard to dietary factors, no statistics are available to estimate the size of an at-risk population based on nutritional status.

With regard to outdoor workers, in 2010 approximately 11.7% of the total number of people (143 million people) employed, or about 16.8 million people, worked outdoors one or more day per week (based on worker surveys).⁶³ Of these approximately 7.4% of the workforce, or about 7.8 million people, worked outdoors three or more days per week.

The health statistics data illustrate what is known as the “pyramid” of effects. At the top of the pyramid, there are approximately 2.5 million deaths from all causes per year in the U.S. population, with about 250 thousand respiratory-related deaths (CDC-WONDER⁶⁴). For respiratory health diseases, there are nearly 3.3 million hospital discharges per year (HCUP⁶⁵),

⁶³ The O*NET program is the nation's primary source of occupational information. Central to the project is the O*NET database, containing information on hundreds of standardized and occupation-specific descriptors. The database, which is available to the public at no cost, is continually updated by surveying a broad range of workers from each occupation. <http://www.onetcenter.org/overview.html>

http://www.onetonline.org/find/descriptor/browse/Work_Context/4.C.2/

⁶⁴ <http://wonder.cdc.gov/>

⁶⁵ <http://www.hcup-us.ahrq.gov/>

8.7 million respiratory ED visits (HCUP, 2007), 112 million ambulatory care visits (Woodwell and Cherry, 2004), and an estimated 700 million restricted activity days per year due to respiratory conditions (Adams et al., 1999). Combining small risk estimates with relatively large baseline levels of health outcomes can result in quite large public health impacts. Thus, even a small percentage reduction in O₃ health impacts on cardiopulmonary diseases would reflect a large number of avoided cases.

3.1.5.3 Averting Behavior

The activity pattern of individuals is an important determinant of their exposure (ISA, U.S. EPA, 2013, section 4.4.1). Variation in O₃ 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 O₃. Activity patterns vary both among and within individuals, resulting in corresponding variations in exposure across a population and over time. Individuals can reduce their exposure to O₃ by altering their behaviors, such as by staying indoors, being active outdoors when air quality is better, and by reducing their activity levels or reducing the time being active outdoors on high-O₃ days (U.S. EPA, 2013, section 4.4.2). The evidence in this topic area, while not addressed in the 2006 AQCD, is evaluated in the ISA for this review.

The widely reported Air Quality Index (AQI) conveys advice to the public, and particularly at-risk populations, on reducing exposure on days when ambient levels of common air pollutants are elevated (www.airnow.gov). The AQI describes the potential for health effects from O₃ (and other individual pollutants) in six color-coded categories of air-quality, ranging from Good (green), Moderate (yellow), Unhealthy for Sensitive Groups (orange), Unhealthy (red), and Very Unhealthy (purple), and Hazardous (maroon). Levels in the unhealthy ranges (i.e., Unhealthy for Sensitive Groups and above) come with recommendations about reducing exposure. Forecasted and actual AQI values for O₃ are reported to the public during the O₃ season. The AQI advisories explicitly state that children, older adults, people with lung disease, and people who are active outdoors, may be at greater risk from exposure to O₃. People are advised to reduce exposure depending on the predicted O₃ levels and the likelihood of risk. This advice includes being active outdoors when air quality is better, and reducing activity levels or reducing the time being active outdoors on high-O₃ days. Staying indoors to reduce exposure is not recommended until air quality reaches the Very Unhealthy or Hazardous categories.

Evidence of individual averting behaviors in response to AQI advisories has been found in several studies, including activity pattern and epidemiologic studies, especially for the at-risk populations, such as children, older adults, and people with asthma, who are targeted by the advisories. Such effects are less pronounced in the general population, possibly due to the opportunity cost of behavior modification. Epidemiologic evidence from a study (Neidell and

Kinney, 2010) conducted in the 1990's in Los Angeles, CA reports increased asthma hospital admissions among children and older adults when O₃ alert days (1-hour max O₃ concentration >200 ppb) were excluded from the analysis of daily hospital admissions and O₃ concentrations (presumably thereby eliminating averting behavior based on high O₃ forecasts). The lower rate of admissions observed when alert days were included in the analysis suggests that estimates of health effects based on concentration-response functions that do not account for averting behavior may be biased towards the null (U.S. EPA, 2013, section 4.4.2).

3.2 AIR QUALITY-, EXPOSURE-, AND RISK-BASED CONSIDERATIONS

In order to inform judgments about the public health impacts of O₃-related health effects, the HREA has developed and applied models to estimate human exposures to O₃ and O₃-associated health risks across the United States, with a specific focus on urban case study areas (U.S. EPA, 2014).⁶⁶ The HREA uses photochemical modeling to adjust air quality from the 2006-2010 O₃ seasons to just meet the current and alternative standards for the 2006-2008 and 2008-2010 periods.⁶⁷ In this section, staff considers estimates of short-term O₃ exposures and estimates of health risks associated with short- and long-term O₃ exposures, for air quality adjusted to just meet the current O₃ standard. In section 3.2.1, we consider the implications for exposure and risk estimates of the approach used in the HREA to adjust air quality. Sections 3.2.2 and 3.2.3 discuss our exposure-based and risk-based considerations, respectively. In these sections we specifically consider the following question:

- **What are the nature and magnitude of O₃ exposures and health risks remaining upon adjusting recent air quality to just meet the current O₃ standard, and what are the important uncertainties associated with those exposure and risk estimates?**

3.2.1 Consideration of the Adjusted Air Quality Used in Exposure and Risk Assessments

In the first draft HREA for this review, as in the last review, the EPA relied upon quadratic rollback to adjust hourly O₃ concentrations in urban case study areas to just meet the current O₃ standard (U.S. EPA, 2014). Although the quadratic rollback method reproduces

⁶⁶ The 15 urban case study areas analyzed for exposures are Atlanta, Baltimore, Boston, Chicago, Cleveland, Dallas, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC. Morbidity and mortality risk estimates are presented for these same areas, with the exception of Chicago, Dallas, and Washington, DC. The HREA also presents a national scale mortality risk assessment for unadjusted (recent) air quality. This national-scale assessment, which focuses on existing air quality conditions and does not estimate the health risks associated with just meeting the current or alternative standards, can provide perspective on the relationship between national-scale O₃ public health impacts and impacts estimated in specific urban areas.

⁶⁷ Three-year periods are used recognizing that the current standard is the average across three years of the annual fourth-highest daily maximum 8-hour average concentration.

historical patterns of air quality changes better than some alternative methods, it relies on statistical relationships without explicitly accounting for atmospheric chemistry and precursor emissions (U.S. EPA, 2014, Chapter 4). An important drawback of the quadratic rollback approach, recognized in the first draft HREA (U.S. EPA, 2012b), is that it forces all monitors in an assessment area to exhibit the same response when air quality is adjusted. It does not allow for the spatial or temporal heterogeneity in responses that result from the non-linear atmospheric chemistry that influences ambient O₃ concentrations (U.S. EPA, 2014, Chapter 4). Because quadratic rollback does not account for physical and chemical atmospheric processes, or the sources of emissions precursors that lead to O₃ formation, a backstop or “floor” must be used when applying quadratic rollback to just meet current or alternative standards to ensure that estimated O₃ is not reduced in a manner inconsistent with O₃ chemistry, such as to reduce concentrations below that associated with background sources (U.S. EPA, 2014, Chapter 4).

Consistent with recommendations from the National Research Council of the National Academies (NRC, 2008), the HREA uses a photochemical model to estimate sensitivities of O₃ to changes in precursor emissions, in order to estimate ambient O₃ concentrations that would just meet the current and alternative standards (U.S. EPA, 2014, Chapter 4).⁶⁸ For the urban case study areas evaluated in the HREA, this model-based adjustment approach was set up to estimate hourly O₃ concentrations at each monitor location when modeled U.S. anthropogenic precursor emissions (i.e., NO_x, VOC)⁶⁹ were reduced to estimate air quality that just meets the current and alternative O₃ standards.⁷⁰

As discussed in Chapter 4 of the HREA (U.S. EPA, 2014), this approach models the physical and chemical atmospheric processes that influence ambient O₃ concentrations. Compared to the quadratic rollback approach, it provides more realistic estimates of the spatial and temporal responses of O₃ to reductions in precursor emissions. These improved estimates avoid many of the limitations inherent in the quadratic rollback method, including the requirement that all monitors in an assessment area exhibit the same response upon air quality

⁶⁸ The HREA uses the CMAQ photochemical model instrumented with the higher order direct decoupled method (HDDM) to estimate ozone concentrations that would occur with the achievement of the current and alternative O₃ standards (U.S. EPA, 2014, Chapter 4).

⁶⁹ Exposure and risk analyses for most urban case study areas focus on reducing NO_x emissions alone (NO_x emissions were reduced by about 10 to 85% for the current standard, and up to about 95% for alternatives). In most of the urban case study areas, reducing VOC emissions did not alter the NO_x emissions reductions required to just meet the current or alternative standards. However, in Chicago and Denver, reductions in VOC emissions allowed for smaller NO_x emissions reductions. Therefore, exposure and risk analyses for Chicago and Denver are based on reductions in emissions of both NO_x and VOC (U.S. EPA, 2014, section 4.3.3.1, Table 4-3).

⁷⁰ Although this chapter focuses on the current standard, our overarching considerations regarding adjusted air quality also apply to alternative standards simulated in the HREA. Alternative standards are discussed in chapter 4 of this PA.

adjustment to the current and/or alternative standards. Because adjusted air quality scenarios are based on reducing only U.S. anthropogenic emissions, this approach also does not require the specification of background concentrations as a rollback “floor” (U.S. EPA, 2014, section 4.3.3).

The use of this model-based air quality adjustment approach in the HREA has important implications for the patterns of ambient O₃ concentrations estimated in urban case study areas. Specifically, in locations and time periods when NO_x is predominantly contributing to O₃ formation (e.g., downwind of important NO_x sources, where the highest O₃ concentrations often occur), model-based adjustment to the current and alternative standards decreases estimated ambient O₃ concentrations compared to recent monitored concentrations (U.S. EPA, 2014, section 4.3.3.2). In contrast, in locations and time periods when NO_x is predominantly contributing to O₃ titration (e.g., in urban centers with high concentrations of NO_x emissions, where ambient O₃ concentrations are often suppressed and thus relatively low⁷¹), model-based adjustment increases ambient O₃ concentrations compared to recent measured concentrations (U.S. EPA, 2014, section 4.3.3.2) (Chapter 2, above).

Within urban case study areas, the overall impacts of model-based air quality adjustment are to reduce relatively high ambient O₃ concentrations (i.e., concentrations at the upper ends of ambient distributions) and to increase relatively low O₃ concentrations (i.e., concentrations at the lower ends of ambient distributions) (U.S. EPA, 2014, section 4.3.3.2, Figures 4-9 and 4-10). Seasonal means of daily concentrations generally exhibit only modest changes upon air quality adjustment, reflecting the seasonal balance between daily decreases and increases in ambient concentrations (U.S. EPA, 2014, Figures 4-9 and 4-10). The resulting compression in distributions of ambient O₃ concentrations is evident in all of the urban case study areas evaluated, though the degree of compression varies considerably across areas (U.S. EPA, 2014, Figures 4-9 and 4-10).

Adjusted patterns of O₃ air quality have important implications for exposure and risk estimates in urban case study areas. Estimates influenced largely by the upper ends of the distribution of ambient concentrations (i.e., exposures of concern and lung function risk estimates, as discussed in sections 3.2.2 and 3.2.3.1 below) will decrease with model-adjustment to the current and alternative standards. In contrast, seasonal risk estimates influenced by the full distribution of ambient O₃ concentrations (i.e., epidemiology-based risk estimates, as discussed in section 3.2.3.2 below) either increase or decrease in response to air quality adjustment,

⁷¹ Titration is also prominent during time periods when photochemistry is limited, such as at night and on cool, cloudy days.

depending on the balance between the daily decreases in high O₃ concentrations and increases in low O₃ concentrations.⁷²

We further consider the implications of the spatial and temporal patterns of adjusted air quality within the context of exposure (section 3.2.2) and risk (section 3.2.3) estimates for O₃ concentrations adjusted to just meet the current standard. As discussed below (section 3.2.3.2), these altered patterns are particularly important to consider when interpreting epidemiology-based risk estimates.

3.2.2 Exposure-Based Considerations

The exposure assessment presented in the HREA (U.S. EPA, 2014, Chapter 5) provides estimates of the number of people exposed to various concentrations of ambient O₃, while at specified exertion levels. The HREA estimates exposures in 15 urban case study areas for school-age children (ages 5 to 18), asthmatic school-age children, asthmatic adults, and older adults, reflecting the strong evidence indicating that these populations are potentially at increased risk for O₃-attributable effects (EPA, 2013, Chapter 8; section 3.1.2, above). An important purpose of these exposure estimates is to provide perspective on the extent to which air quality adjusted to just meet the current O₃ NAAQS could be associated with exposures to O₃ concentrations reported to result in respiratory effects.⁷³ Estimates of such “exposures of concern” provide perspective on the potential public health impacts of O₃-related effects, including for effects that cannot currently be evaluated in a quantitative risk assessment (e.g., airway inflammation).

In the absence of large scale exposure studies that encompass the general population, as well as at-risk populations, modeling is the preferred approach to estimating exposures to O₃. The use of exposure modeling also facilitates the estimation of exposures resulting from ambient air concentrations differing from those in exposure studies (e.g., concentrations just meeting the current standard). In the HREA, population exposures to ambient O₃ concentrations are estimated using the current version of the Air Pollutants Exposure (APEX) model. The APEX model simulates the movement of individuals through time and space and estimates their exposures to a given pollutant in indoor, outdoor, and in-vehicle microenvironments (U.S. EPA, 2014, section 5.1.3). APEX takes into account the most critical factors that contribute to total

⁷² In addition, because epidemiology-based risk estimates use “area-wide” average O₃ concentrations, calculated by averaging concentrations across multiple monitors in urban case study areas (section 3.2.3.2 below), risk estimates on a given day depend on the daily balance between increasing and decreasing O₃ concentrations at individual monitors.

⁷³ In addition, the range of modeled personal exposures to ambient O₃ provide an essential input to the portion of the health risk assessment based on exposure-response functions (for lung function decrements) from controlled human exposure studies. The health risk assessment based on exposure-response information is discussed in section 3.2.3, below.

human exposure to ambient O₃, including the temporal and spatial distributions of people and O₃ concentrations throughout an urban area, the variation of O₃ concentrations within various microenvironments, and the effects of exertion on breathing rate in exposed individuals (U.S. EPA, 2014, section 5.1.3). To the extent spatial and/or temporal patterns of ambient O₃ concentrations are altered upon air quality adjustment, as discussed above, exposure estimates reflect population exposures to those altered patterns.

The HREA estimates 8-hour exposures at or above benchmark concentrations of 60, 70, and 80 ppb for individuals engaged in moderate or greater exertion. Benchmarks reflect exposure concentrations at which O₃-induced respiratory effects are known to occur in some healthy adults engaged in moderate, intermittent exertion, based on evidence from controlled human exposure studies (section 3.1.2.1 above and U.S. EPA, 2013, section 6.2). The amount of weight to place on the estimates of exposures at or above specific benchmark concentrations depends in part on the weight of the scientific evidence concerning health effects associated with O₃ exposures at that concentration. It also depends on judgments about the importance, from a public health perspective, of the health effects that are known or can reasonably be inferred to occur as a result of exposures at benchmark concentrations (sections 3.1.3, 3.1.5 above).

As discussed in more detail above (section 3.1.2.1), the health evidence that supports evaluating exposures of concern at or above benchmark concentrations of 60, 70, and 80 ppb comes from a large body of controlled human exposure studies reporting a variety of respiratory effects in healthy adults. The lowest O₃ exposure concentration for which controlled human exposure studies have reported respiratory effects in healthy adults is 60 ppb, with more evidence supporting this benchmark concentration in the current review than in the last review. In healthy adults, exposures to 60 ppb O₃ have been reported to decrease lung function and to increase airway inflammation. Exposures of healthy adults to 70 ppb O₃ have been reported to result in larger lung function decrements, compared to 60 ppb, as well as in increased respiratory symptoms.⁷⁴ Exposures of healthy adults to 80 ppb O₃ have been reported to result in larger lung function decrements than following exposures to 60 or 70 ppb, increased airway inflammation, increased respiratory symptoms, increased airways responsiveness, and decreased lung host defense (section 3.1.2.1, above). As discussed above (section 3.1.3), respiratory effects reported following exposures to O₃ concentrations of 60, 70, or 80 ppb meet ATS criteria for adverse effects, result in effects judged important by CASAC in past reviews, and/or could contribute to the clearly adverse effects reported in epidemiologic studies evaluating broader populations. Compared to the healthy individuals included in the studies that provided the basis for the

⁷⁴ As noted above, for the 70 ppb exposure concentration Schelegle et al. (2009) reported that the actual mean exposure concentration was 72 ppb.

benchmarks, at-risk populations (e.g., asthmatics, children) are more likely to experience larger and/or more serious effects (e.g., U.S. EPA, 2013, p. 6-21).

In considering estimates of O₃ exposures of concern at or above benchmarks of 60, 70, and 80 ppb, within the context of the adequacy of the current standard, we first address the following specific question:

- **What are the nature and magnitude of the short-term O₃ exposures of concern remaining upon adjustment of air quality to just meet the current O₃ standard?**

In addressing this question, we focus on modeled exposures for school-age children (ages 5-18) and asthmatic school-age children, two of the at-risk populations identified in the ISA (section 3.1.5 above). The percentages of children estimated to experience exposures of concern are larger than the percentages estimated for adult populations (i.e., approximately 3-fold larger across cities) (U.S. EPA, 2014, sections 5.3.2, 5.3.3 and Figures 5-5 to 5-8). The larger exposure estimates for children are due primarily to the larger percentage of children estimated to spend an extended period of time being physically active outdoors when O₃ concentrations are elevated (U.S. EPA, 2014, sections 5.3.2 and 5.4.1).

Although exposure estimates differ between children and adults, the patterns of results across the cities and years are similar among all of the populations evaluated (U.S. EPA, 2014, Figures 5-5 to 5-8). Therefore, while we highlight estimates in children, we also note that the patterns of exposures estimated for children represent the patterns estimated for adult asthmatics and older adults.

Key results for children are summarized below for air quality adjusted to simulate just meeting the current O₃ NAAQS (Figures 3-7 to 3-10).⁷⁵ Estimates for all children and asthmatic children are virtually indistinguishable (U.S. EPA, 2014, section 5.3.2). The estimates presented in Figures 3-7 to 3-10 below reflect consistent reductions in estimated exposures of concern across urban case study areas, relative to recent (i.e., unadjusted) air quality (U.S. EPA, 2014, Appendix 5F). When averaged over the years evaluated in the HREA, reductions of up to about 70% were estimated, compared to recent air quality. These reductions in estimated exposures of concern, relative to unadjusted air quality, reflect the consistent reductions in the highest ambient O₃ concentrations upon air quality adjustment to just meet the current standard (section 3.2.1 above; U.S. EPA, 2014, Chapter 4). Such reductions in estimated exposures of concern are evident throughout urban case study areas, including in urban cores and in surrounding areas (U.S. EPA, 2014, section 9.6, Appendix 9A). Figures 3-7 (Average over years) and 3-8 (Worst-Case Years) present estimates of one or more exposures of concern. Figures 3-9 (Average over years) and 3-10 (Worst-Case Years) present estimates of two or more exposures of concern.

⁷⁵ Figures 3-7 and 3-8 present estimates of one or more exposures of concern. Figures 3-9 and 3-10 present estimates of two or more exposures of concern.

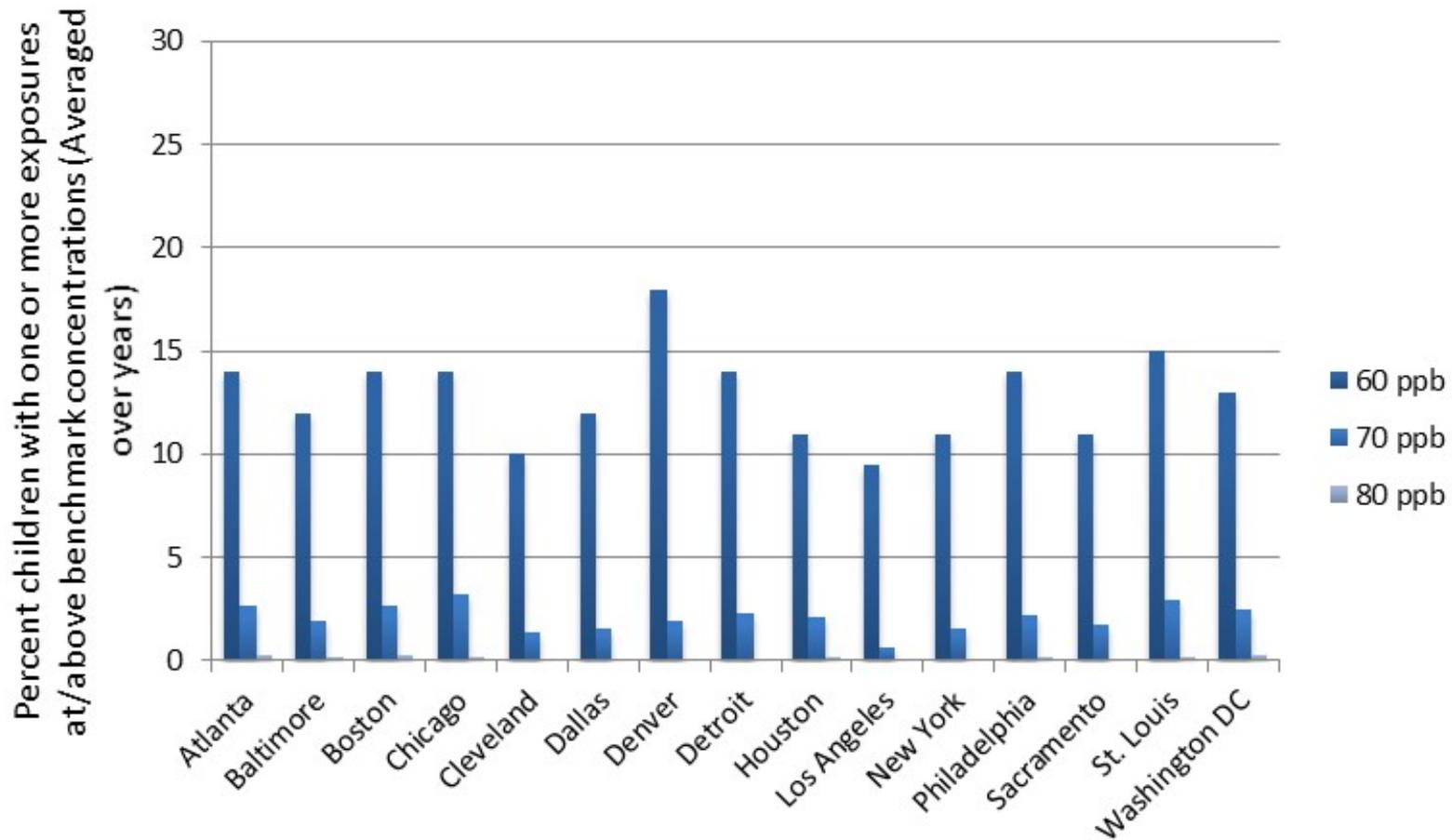


Figure 3-7. Percent of children estimated to experience one or more exposures of concern at or above 60, 70, 80 ppb with air quality adjusted to just meet the current standard - Averaged Over 2006 to 2010.

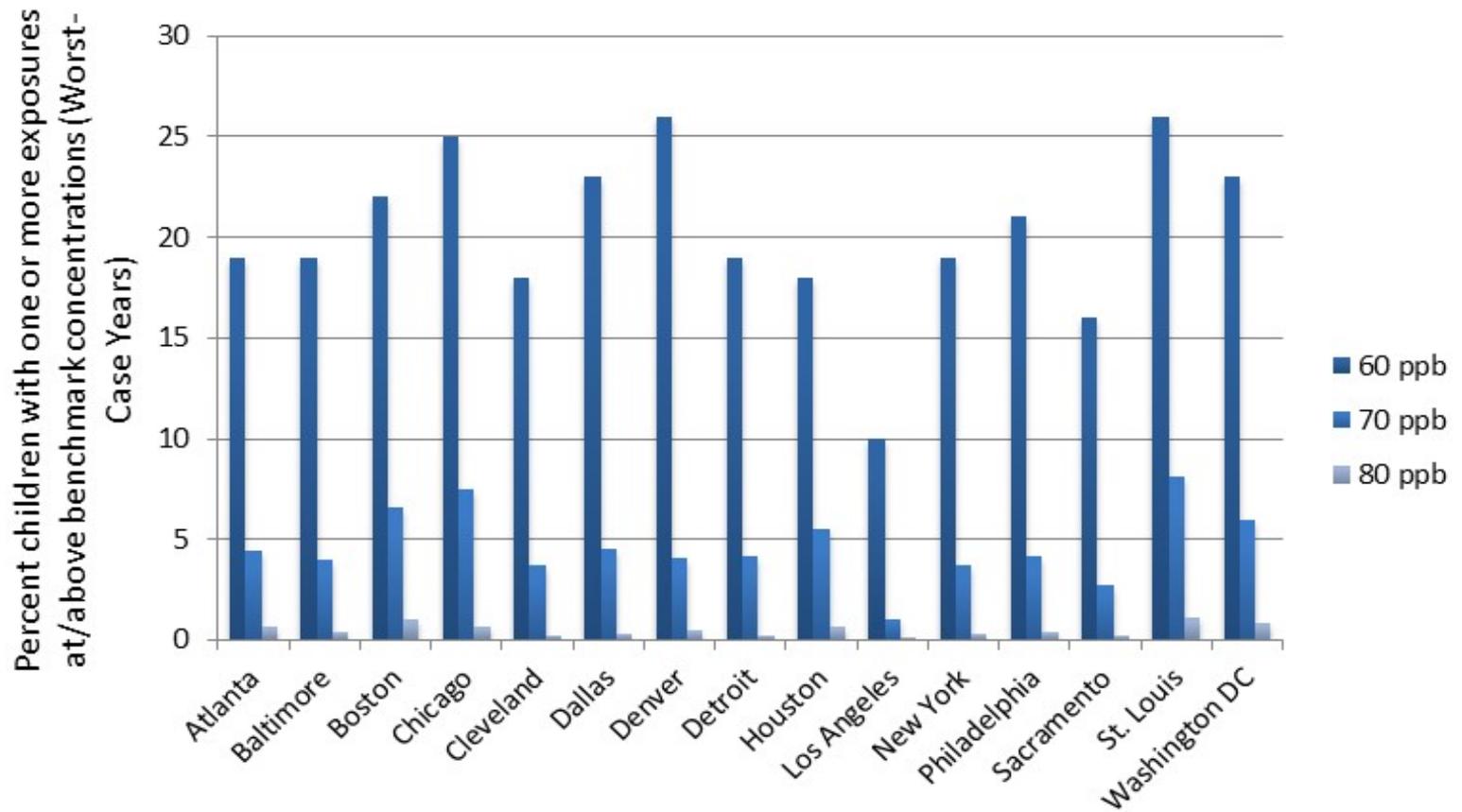


Figure 3-8. Percent of children estimated to experience one or more exposures of concern at or above 60, 70, 80 ppb with air quality adjusted to just meet the current standard - Worst-Case Year from 2006 to 2010.

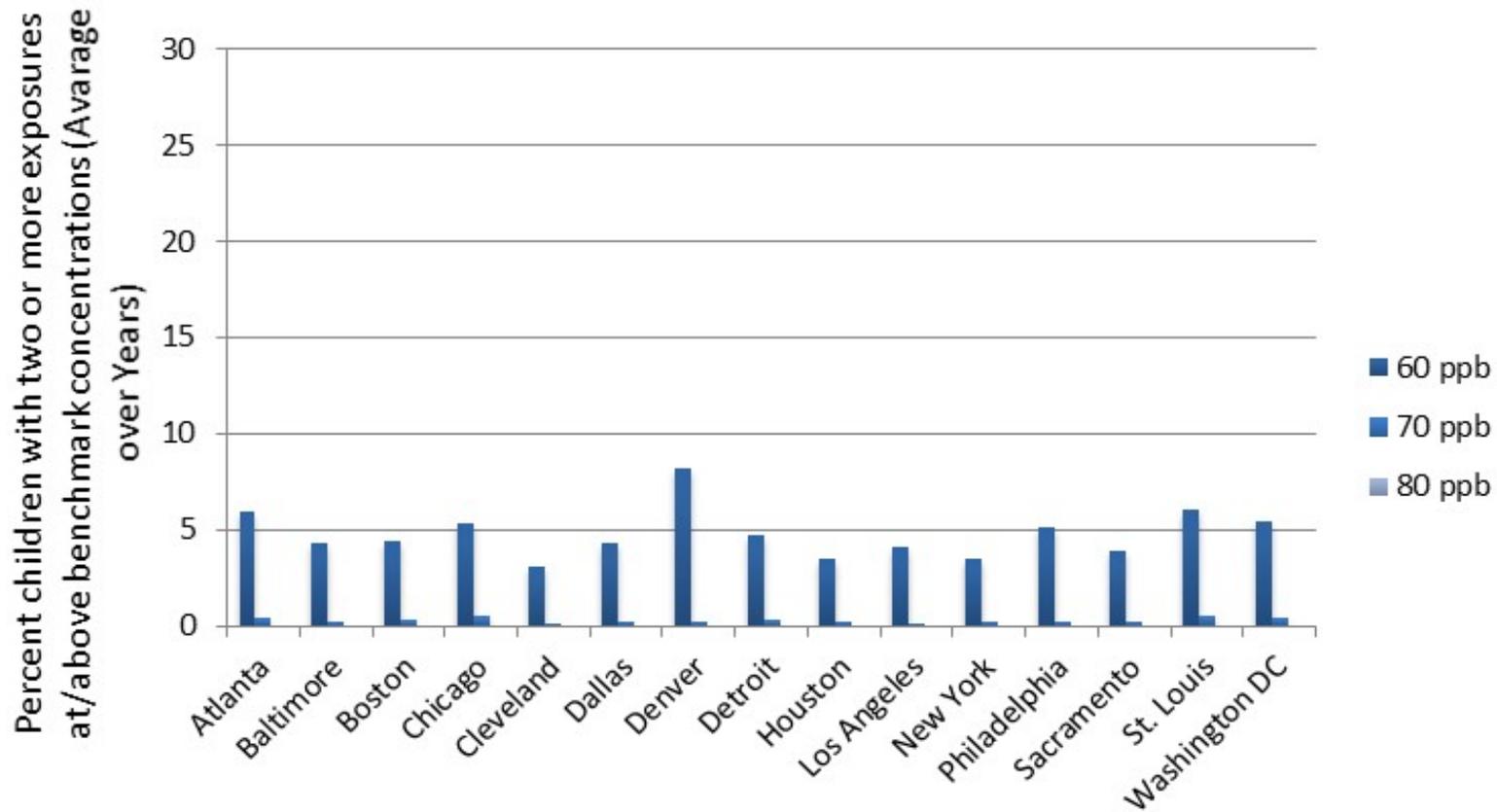


Figure 3-9. Percent of children estimated to experience two or more exposures of concern at or above 60, 70, 80 ppb with air quality adjusted to just meet the current standard - Averaged Over 2006 to 2010.

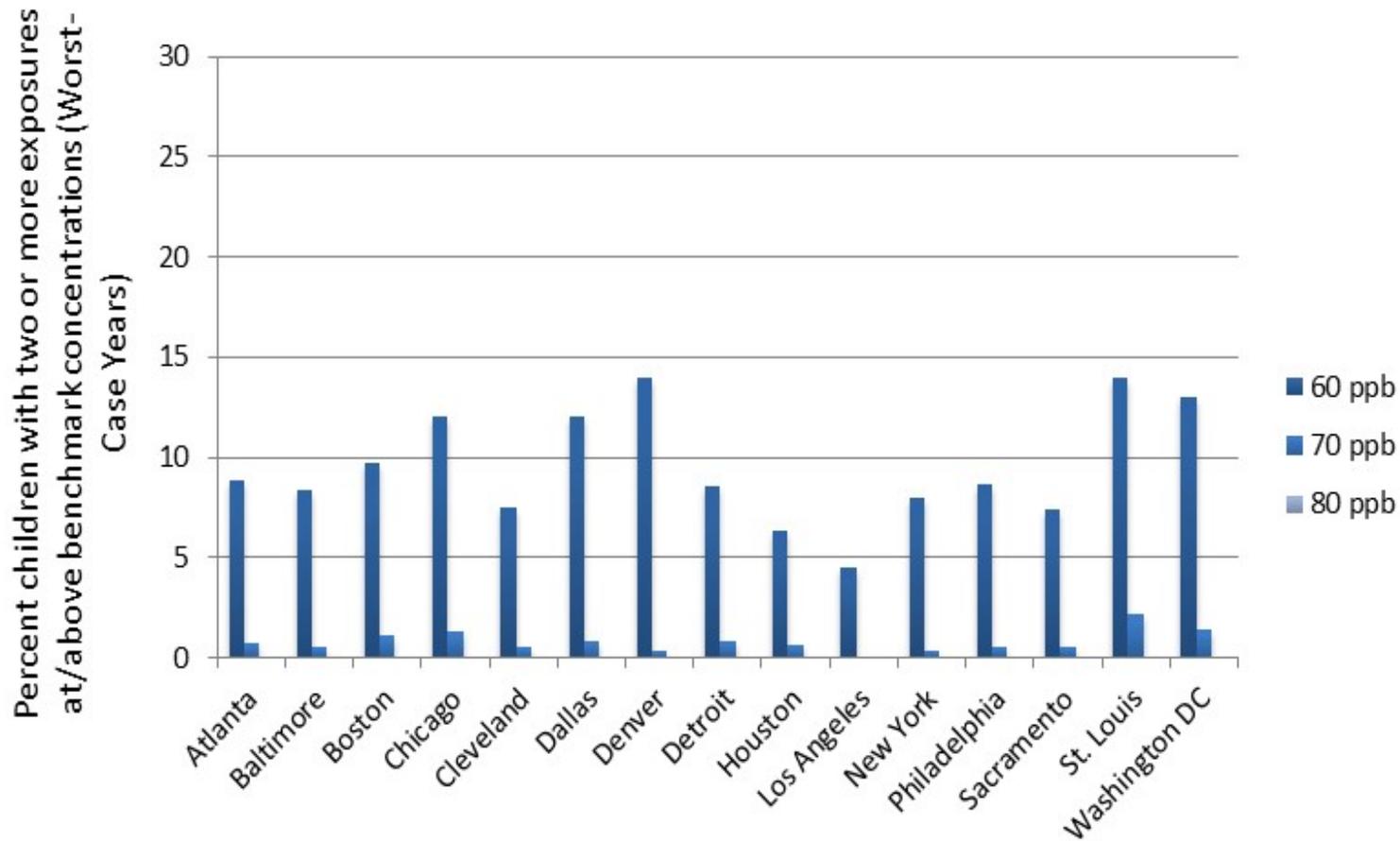


Figure 3-10. Percent of children estimated to experience two or more exposures of concern at or above 60, 70, 80 ppb with air quality adjusted to just meet the current standard - Worst-Case Year from 2006 to 2010.

Based on Figures 3-7 to 3-10 and the associated details described in the HREA (U.S. EPA, 2014, Chapter 5), we take note of the following with regard to exposures that are estimated to be allowed by the current standard:

1. For exposures of concern at or above 60 ppb:
 - a. On average over the years 2006 to 2010, the current standard is estimated to allow approximately 10 to 17% of children in urban case study areas to experience one or more exposures of concern at or above 60 ppb. Summing across urban case study areas, these percentages correspond to almost 2.5 million children experiencing approximately 4 million exposures of concern at or above 60 ppb during a single O₃ season. Of these children, almost 250,000 are asthmatics.
 - b. On average over the years 2006 to 2010, the current standard is estimated to allow approximately 3 to 8% of children in urban case study areas to experience two or more exposures of concern to O₃ concentrations at or above 60 ppb. Summing across the urban case study areas, these percentages correspond to almost 900,000 children (including about 90,000 asthmatic children) estimated to experience at least two O₃ exposure concentrations at or above 60 ppb during a single O₃ season.
 - c. In the worst-case years (i.e., those with the largest exposure estimates), the current standard is estimated to allow approximately 10 to 26% of children to experience one or more exposures of concern at or above 60 ppb, and approximately 4 to 14% to experience two or more exposures of concern at or above 60 ppb.
2. For exposures of concern at or above 70 ppb:
 - a. On average over the years 2006 to 2010, the current standard is estimated to allow up to approximately 3% of children in urban case study areas to experience one or more exposures of concern at or above 70 ppb. Summing across urban case study areas, more than 350,000 children (including about 40,000 asthmatic children) are estimated to experience O₃ exposure concentrations at or above 70 ppb during a single O₃ season.
 - b. On average over the years 2006 to 2010, the current standard is estimated to allow less than 1% of children in urban case study areas to experience two or more exposures of concern to O₃ concentrations at or above 70 ppb.
 - c. In the worst-case years, the current standard is estimated to allow approximately 1 to 8% of children to experience one or more exposures of concern at or above 70 ppb, and up to approximately 2% to experience two or more exposures of concern, at or above 70 ppb.
3. For exposures of concern at or above 80 ppb: The current standard is estimated to allow about 1% or fewer children in urban case study areas to experience exposures of concern at or above 80 ppb, even in years with the highest exposure estimates.

In further evaluating estimated exposures of concern from the HREA, we next consider the following question:

- **What are the important sources of uncertainty associated with exposure estimates?**

Due to variability in responsiveness, only a subset of individuals who experience exposures at or above a benchmark concentration can be expected to experience health effects. Given the lack of sufficient exposure-response information for most of the health effects that informed benchmark concentrations, estimates of the number of people likely to experience exposures at or above benchmark concentrations generally cannot be translated into quantitative estimates of the number of people likely to experience specific health effects.⁷⁶ We view health-relevant exposures as a continuum with greater confidence and certainty about the existence of adverse health effects at higher O₃ exposure concentrations, and less confidence and greater uncertainty as one considers lower exposure concentrations. This view draws from the overall body of available health evidence, which indicates that as exposure concentrations increase the incidence, magnitude, and severity of effects increases.

Though we have less confidence in the likelihood of adverse health effects as O₃ exposure concentrations decrease, we also note that the controlled human exposure studies that provided the basis for health benchmark concentrations have not evaluated at-risk populations. Compared to the healthy individuals included in controlled human exposure studies, members of at-risk populations (e.g., asthmatics, children) 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 O₃ concentrations. In considering estimated exposures of concern within the context of drawing conclusions on the adequacy of the current standard (section 3.4, below), we balance concerns about the potential for adverse health effects, including effects in at-risk populations, with our increasing uncertainty regarding the likelihood of such effects following exposures to lower O₃ concentrations.

Uncertainties associated with the APEX exposure modeling also have the potential to be important in our consideration of the adequacy of the current standard (U.S. EPA, 2014, section 5.5.2, Table 5-10). For example, the HREA concludes that exposures of concern could be underestimated for some individuals who are frequently and routinely active outdoors during the warm season (U.S. EPA, section 5.5.2). This could include outdoor workers and children who are frequently active outdoors. The HREA specifically notes that long-term diary profiles (i.e., monthly, annual) do not exist for such populations, limiting the extent to which APEX outputs reflect people who follow similar daily routines resulting in high exposures, over extended

⁷⁶ The exception to this is lung function decrements, as discussed below (section 3.2.3.1).

periods of time. Thus, exposure estimates generated from the general pool of available diary profiles likely do not reflect the most highly exposed individuals in the population.

In order to evaluate the potential implications of this uncertainty for exposure estimates, the HREA reports the results of limited sensitivity analyses using subsets of diaries specifically selected to reflect groups spending a larger proportion of time being active outdoors during the O₃ season. When diaries were selected to mimic exposures that could be experienced by outdoor workers, the percent of modeled individuals estimated to experience exposures of concern increased compared to other adult populations evaluated. The percent of outdoor workers 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) (U.S. EPA, 2014, section 5.4.3.2, Figure 5-14). In addition, when diaries were restricted to children who did not report any time spent inside a school or performing paid work (i.e., to mimic children spending particularly large portions of their time outdoors during the summer), the number experiencing exposures of concern increased by approximately 30% (U.S. EPA, 2014, section 5.4.3.1). Though these sensitivity analyses are limited to single urban case study areas, and though there is uncertainty associated with diary selection approaches to mimic highly exposed populations, they suggest the possibility that some at-risk groups could experience more frequent exposures of concern than indicated by estimates based on the full database of activity diary profiles.

In further considering activity diaries, the HREA also notes growing evidence indicating that people can change their behavior in response to high O₃ concentrations, reducing the time spent being active outdoors (U.S. EPA, 2014, section 5.4.3.3). Commonly termed “averting behaviors,” these altered activity patterns could reduce personal exposure concentrations. Therefore, the HREA also performed limited sensitivity analyses to evaluate the potential implications of averting behavior for estimated exposures of concern. These analyses suggest that averting behavior could reduce the percentages of children estimated to experience exposures of concern at or above the 60 or 70 ppb benchmark concentrations by approximately 10 to 30%, with larger reductions possible for the 80 ppb benchmark (U.S. EPA, 2014, Figure 5-15). As discussed above for other sensitivity analyses, these analyses are limited to a single urban case study area and are subject to uncertainties associated with assumptions about the prevalence and duration of averting behaviors. However, the results suggest that exposures of concern could be overestimated, particularly in children (Neidell et al., 2009; U.S. EPA, 2013, Figures 4-7 and 4-8), if the possibility for averting behavior is not incorporated into estimates.

3.2.3 Risk-Based Considerations

For some health endpoints, there is sufficient scientific evidence and information available to support the development of quantitative estimates of O₃-related health risks. In the last review of the O₃ NAAQS, the quantitative health risk assessment estimated O₃-related lung function decrements, respiratory symptoms, respiratory-related hospital admissions, and non-accidental and cardiorespiratory-related mortality (U.S. EPA, 2007). In those analyses, both controlled human exposure and epidemiologic studies were used for the quantitative assessment of O₃-related human health risks.

In the current review, for short-term O₃ concentrations the HREA estimates lung function decrements; respiratory symptoms in asthmatics; hospital admissions and emergency department visits for respiratory causes; and all-cause mortality (U.S. EPA, 2014, Chapters 6 and 7). For “long-term” O₃ concentrations, the HREA estimates respiratory mortality (U.S. EPA, 2014, Chapter 7).⁷⁷ Estimates of O₃-induced lung function decrements are based on exposure modeling, as noted above, combined with exposure-response relationships from controlled human exposure studies (U.S. EPA, 2014, Chapter 6). Estimates of O₃-associated respiratory symptoms; hospital admissions and emergency department visits; and mortality are based on concentration-response relationships from epidemiologic studies (U.S. EPA, 2014, Chapter 7). As with the exposure assessment discussed above, O₃-associated health risks are estimated for recent air quality and for ambient concentrations adjusted to just meet the current 8-hour O₃ NAAQS, based on 2006-2010 air quality and adjusted precursor emissions.

Section 3.2.3.1 below discusses risk results for O₃-induced lung function decrements following short-term exposures, based on exposure modeling results and exposure-response relationships from controlled human exposure studies. Section 3.2.3.2 discusses epidemiology-based risk estimates, with a focus on all-cause mortality (short-term O₃ concentrations); respiratory-related morbidity outcomes (short-term O₃ concentrations); and respiratory mortality (long-term O₃ concentrations).

3.2.3.1 Risk of Lung Function Decrements

In the last review, EPA conducted a health risk assessment that produced risk estimates for the number and percent of school-aged children, asthmatic school-aged children, and the general population experiencing lung function decrements associated with O₃ exposures for 12 urban areas. These estimates were based on exposure-response relationships developed from

⁷⁷ Risk estimates for “long-term” concentrations are based on the concentration-response relationship identified in the study by Jerrett et al. (2009). As discussed above, study authors used April to September averages of 1-hour daily maximum O₃ concentrations as surrogates for “long-term” exposures.

analysis of data from several controlled human exposure studies, combined with exposure estimates developed for children and adults (U.S. EPA, 2007).

In the current 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 case study areas.⁷⁸ The results presented in the HREA are based on an updated dose-threshold model that estimates FEV₁ responses for individuals following short-term exposures to O₃ (McDonnell et al., 2012), reflecting methodological improvements since the last review (U.S. EPA, 2014, section 6.2.4; section 3.1.2.1, above). The impact of the dose threshold is that O₃-induced FEV₁ decrements result primarily from exposures on days with ambient O₃ concentrations above about 40 ppb (U.S. EPA, 2013, section 6.3.1, Figure 6-9).⁷⁹

As discussed above (section 3.1.3), for effects such as lung function decrements, which are transient and reversible, aspects such as the likelihood that these effects would occur repeatedly and would interfere with normal activities are important to consider in making judgments about adversity to individuals. As stated in the 2006 Criteria Document (Table 8-3, p. 8-68), for people with lung disease even moderate functional responses (e.g., FEV₁ decrements \geq 10% but $<$ 20%) would likely interfere with normal activities for many individuals, and would likely result in more frequent medication use. Moreover, as noted above, in a recent letter to the Administrator, the CASAC O₃ Panel stated that “[c]linically relevant effects are decrements $>$ 10%, a decrease in lung function considered clinically relevant by the American Thoracic Society” (Samet, 2011, p.2). The CASAC O₃ Panel also stated that:

[A] 10% decrement in FEV₁ can lead to respiratory symptoms, especially in individuals with pre-existing pulmonary or cardiac disease. For example, people with chronic obstructive pulmonary disease have decreased ventilatory reserve (i.e., decreased baseline FEV₁) such that a \geq 10% decrement could lead to moderate to severe respiratory symptoms (Samet, 2011, p.7).

Consistent with this advice from the last review, in the current review CASAC has concluded that “estimation of FEV₁ decrements of \geq 15% is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, whereas an FEV₁ decrement of \geq 10% is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014, p. 3).

⁷⁸As noted for the exposure assessment above, the 15 cities assessed are Atlanta, Baltimore, Boston, Chicago, Cleveland, Dallas, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, St. Louis, and Washington, DC.

⁷⁹**Error! Reference source not found.** in the HREA shows that more than 90% of daily instances of FEV₁ decrements \geq 10% occur when 8-hr average ambient concentrations are above 40 ppb for 2006 air quality in Los Angeles. The distribution of decrements will be different for different study areas, years, and air quality scenarios (U.S. EPA, 2014, section 6.3.1).

In judging the extent to which moderate lung function decrements represent effects that should be regarded as adverse to the health status of individuals, in previous NAAQS reviews we have also considered the extent to which decrements were experienced repeatedly during the course of a year (U.S. EPA, 2007). Although some experts would judge single occurrences of moderate responses to be a “nuisance,” especially for healthy individuals,⁸⁰ a more general consensus view of the adversity of such moderate responses emerges as the frequency of occurrence increases. Thus in the past EPA has judged that repeated occurrences of moderate responses, even in otherwise healthy individuals, may be considered to be adverse since they could well set the stage for more serious illness (61 FR 65723). The CASAC panel in the 1997 NAAQS review expressed a consensus view that these “criteria for the determination of an adverse physiological response were reasonable” (Wolff, 1995).

The HREA estimates risks of moderate to large lung function decrements, defined as FEV₁ decrements $\geq 10\%$, $\geq 15\%$, or $\geq 20\%$. In evaluating these lung function risk estimates within the context of considering the adequacy of the current O₃ standard, we first consider the following specific question:

- **What are the nature and magnitude of lung function risks remaining upon just meeting the current O₃ standard?**

In considering risks of O₃-induced FEV₁ decrements, we focus on the percent of children estimated to experience decrements ≥ 10 , 15, and 20%, noting that the percentage of asthmatic children estimated to experience such decrements is virtually indistinguishable from the percentage estimated for all children.⁸¹ Compared to children, smaller percentages of adults are estimated to experience O₃-induced FEV₁ decrements (U.S. EPA, 2014, section 6.3.1, Table 6-4). As for exposures of concern (see above), the patterns of results across urban case study areas and over the years evaluated are similar in children and adults (U.S. EPA, 2014, Appendix 6E). Therefore, while we highlight estimates in children, we note that these results are also representative of the patterns estimated for adult populations.

Key results for children are summarized below for air quality adjusted to just meet the current O₃ NAAQS (Figures 3-11 to 3-14).⁸² The estimates presented in Figures 3-11 to 3-14 below reflect consistent reductions across urban case study areas in the percent of children estimated to experience O₃-induced lung function decrements, relative to recent (i.e., unadjusted) air quality (U.S. EPA, 2014, Appendix 6B). When averaged over the years evaluated in the

⁸⁰Though not all experts, as indicated by the advice received on this issue from past CASAC O₃ Panels (Samet, 2011).

⁸¹ Though see below for discussion of uncertainty in lung function responses of children and asthmatics.

⁸² Figures 3-11 and 3-12 present estimates of one or more decrements. Figures 3-13 and 3-14 present estimates of two or more decrements.

HREA, reductions of up to about 40% were estimated compared to recent air quality. These reductions reflect the consistent decreases in relatively high ambient O₃ concentrations upon adjustment to just meet the current standard (section 3.2.1 above; U.S. EPA, 2014, Chapter 4).⁸³ Such reductions in estimated lung function risks are evident throughout urban case study areas, including in urban cores and in surrounding areas (U.S. EPA, 2014, section 9.6). Figures 3-11 (Average over years) and 3-12 (Worst-Case Years) present estimates of one or more O₃-induced lung function decrements. Figures 3-13 (Average over years) and 3-14 (Worst-Case Years) present estimates of two or more decrements.

⁸³ As noted above, the impact of the dose threshold is that O₃-induced FEV₁ decrements result primarily from days with average ambient O₃ concentrations above about 40 ppb.

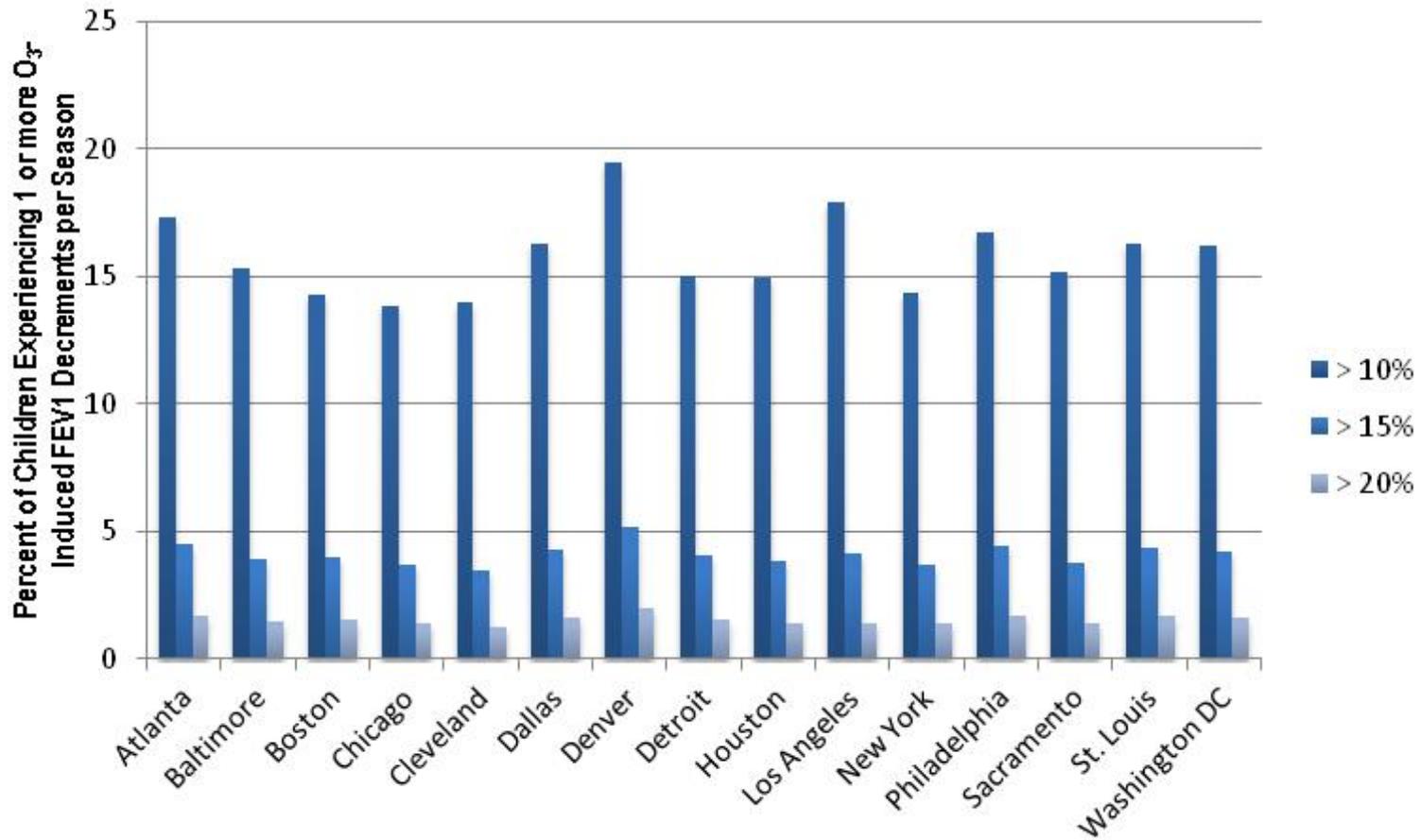


Figure 3-11. Percent of school-aged children (5-18 yrs) estimated to experience one or more days with FEV₁ decrements \geq 10, 15, or 20% with air quality adjusted to just meet the current standard – Averaged over 2006 to 2010

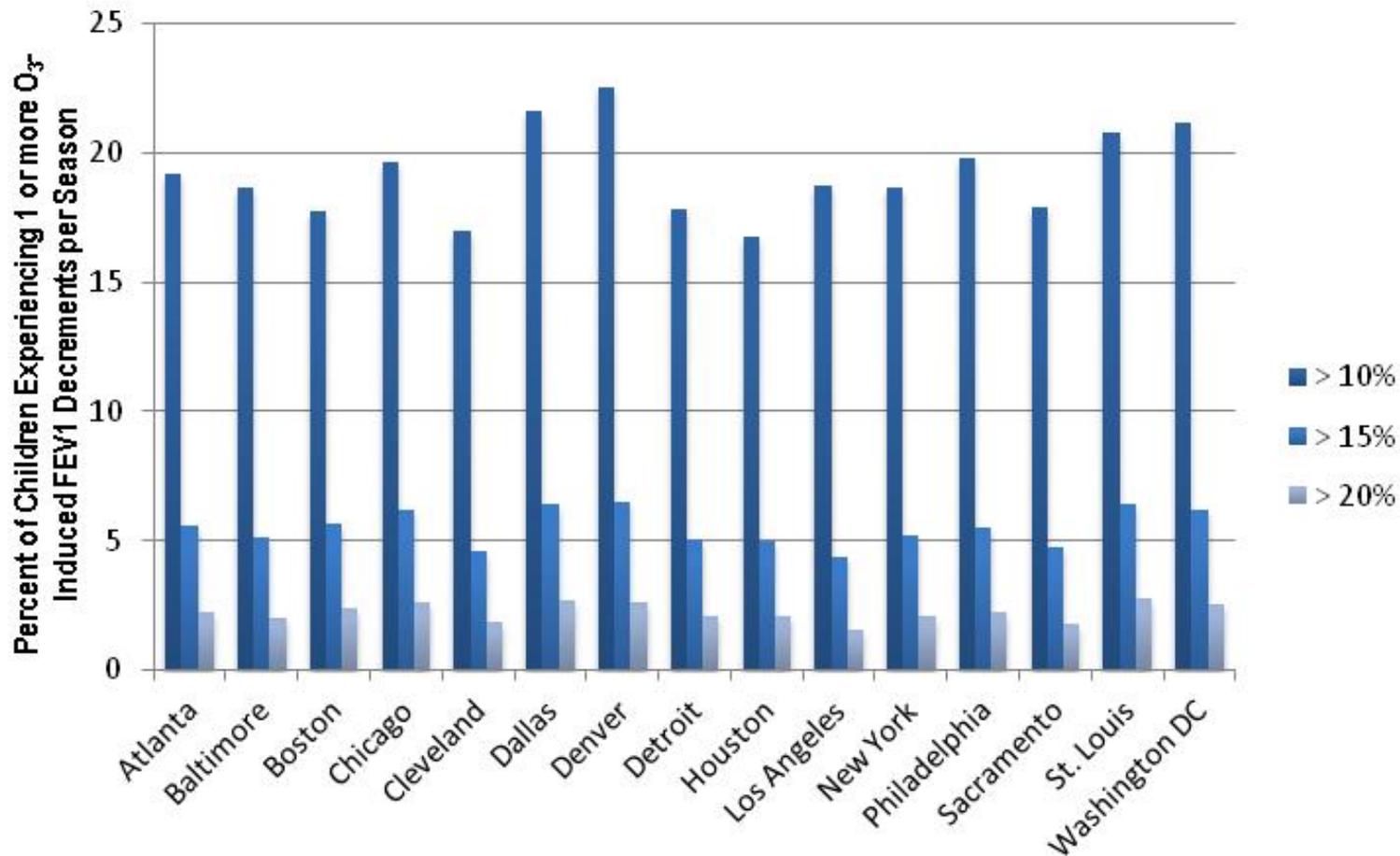


Figure 3-12. Percent of school-aged children (5-18 yrs) estimated to experience one or more days with FEV₁ decrements \geq 10, 15, or 20% with air quality adjusted to just meet the current standard – Worst-Case Year from 2006 to 2010

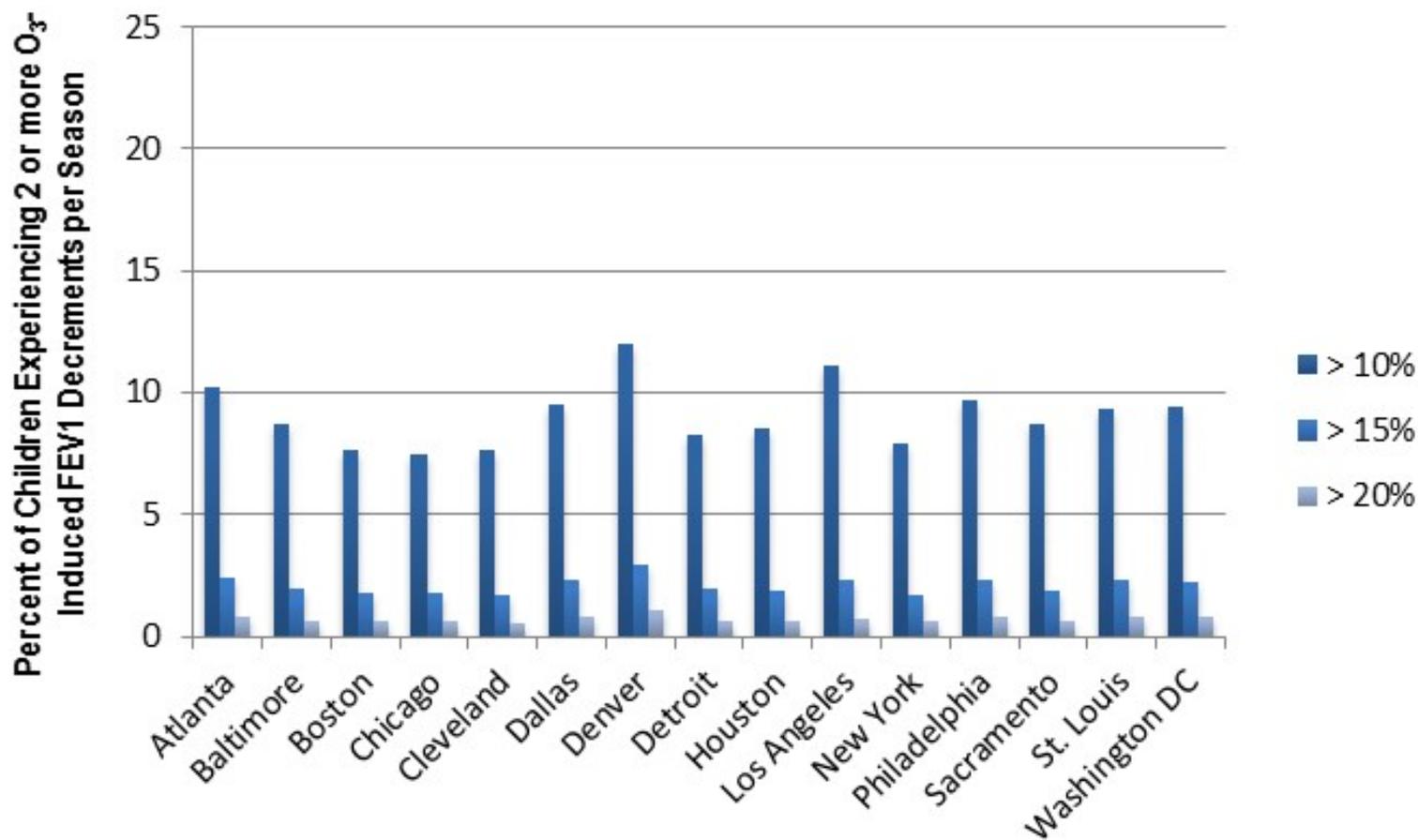


Figure 3-13. Percent of school-aged children (aged 5-18 yrs) estimated to experience two or more days with FEV₁ decrements \geq 10, 15, or 20% with air quality adjusted to just meet the current standard – Averaged over 2006 to 2010

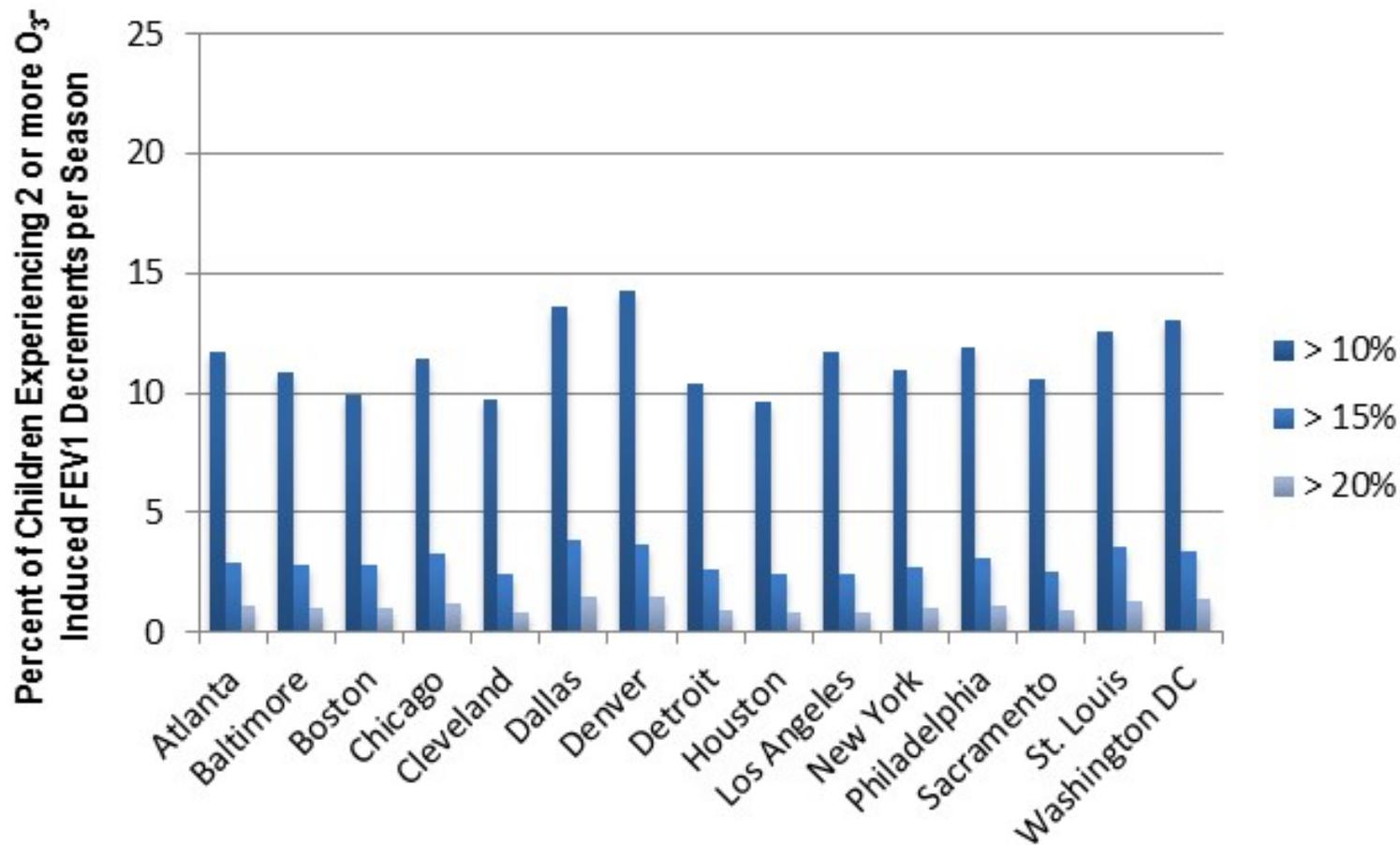


Figure 3-14. Percent of school-aged children (5-18 yrs) estimated to experience two or more days with FEV₁ decrements \geq 10, 15, or 20% with air quality adjusted to just meet the current standard - Worst-Case Year from 2006 to 2010

Based on Figures 3-11 to 3-14 and the associated details described in the HREA (U.S. EPA, 2014, Chapter 6), we take note of the following with regard to lung function decrements estimated to be allowed by the current standard:

1. For FEV₁ decrements $\geq 10\%$:
 - a. On average over the years 2006 to 2010, the current standard is estimated to allow approximately 14 to 19% of children in urban case study areas to experience one or more lung function decrements $\geq 10\%$. Summing across urban case study areas, this corresponds to approximately 3 million children experiencing 15 million O₃-induced lung function decrements $\geq 10\%$ during a single O₃ season. Of these children, about 300,000 are asthmatics.
 - b. On average over the years 2006 to 2010, the current standard is estimated to allow approximately 8 to 12% of children in urban case study areas to experience two or more O₃-induced lung function decrements $\geq 10\%$. Summing across the urban case study areas, this corresponds to almost 2 million children (including almost 200,000 asthmatic children) estimated to experience two or more O₃-induced lung function decrements greater than 10% during a single O₃ season.
 - c. In the worst-case years, the current standard is estimated to allow approximately 17 to 22% of children in urban case study areas to experience one or more lung function decrements $\geq 10\%$, and approximately 10 to 14% to experience two or more O₃-induced lung function decrements $\geq 10\%$.
2. For FEV₁ decrements $\geq 15\%$:
 - a. On average over the years 2006 to 2010, the current standard is estimated to allow approximately 3 to 5% of children in urban case study areas to experience one or more lung function decrements $\geq 15\%$. Summing across urban case study areas, this corresponds to over 750,000 children (including approximately 80,000 asthmatic children) estimated to experience at least one O₃-induced lung function decrement $\geq 15\%$ during a single O₃ season.
 - b. On average over the years 2006 to 2010, the current standard is estimated to allow approximately 2 to 3% of children in urban case study areas to experience two or more O₃-induced lung function decrements $\geq 15\%$.
 - c. In the worst-case years, the current standard is estimated to allow approximately 4 to 7% of children in urban case study areas to experience one or more lung function decrements $\geq 15\%$, and approximately 2 to 4% to experience two or more O₃-induced lung function decrements $\geq 15\%$.
3. For FEV₁ decrements $\geq 20\%$:
 - a. On average over the years 2006 to 2010, the current standard is estimated to allow approximately 1 to 2% of children in urban case study areas to experience one or more lung function decrements $\geq 20\%$. Summing across urban case study areas, this corresponds to almost 300,000 children (including approximately 30,000 asthmatic

children) estimated to experience at least one O₃-induced lung function decrement \geq 20% during a single O₃ season.

- b. On average over the years 2006 to 2010, the current standard is estimated to allow less than about 1% of children in urban case study areas to experience two or more O₃-induced lung function decrements \geq 20%.
- c. In the worst-case years, the current standard is estimated to allow approximately 2 to 3% of children to experience one or more lung function decrements \geq 20%, and less than 2% to experience two or more O₃-induced lung function decrements \geq 20%.

In further considering estimated lung function risks from the HREA, we next consider the following question:

- **What are the important sources of uncertainty associated with lung function risk estimates?**

In addition to the uncertainties noted above for exposure estimates, the HREA identifies several key uncertainties associated with estimates of O₃-induced lung function decrements. An uncertainty with particular potential to impact our consideration of risk estimates in this Policy Assessment stems from the lack of exposure-response information in children. In the absence of controlled human exposure data for children, risk estimates are based on the assumption that children exhibit the same lung function response following O₃ exposures as healthy 18 year olds (i.e., the youngest age for which controlled human exposure data is available) (U.S. EPA, 2014, section 6.5.3). This assumption was justified in part by the findings of McDonnell et al. (1985), who reported that children 8-11 year old experienced FEV₁ responses similar to those observed in adults 18-35 years old. In addition, as discussed in the ISA (U.S. EPA, 2013, section 6.2.1), summer camp studies of school-aged children reported O₃-induced lung function decrements similar in magnitude to those observed in controlled human exposure studies using adults. In extending the risk model to children, the HREA fixes the age term in the model at its highest value, the value for age 18. This approach could result in either over- or underestimates of O₃-induced lung function decrements in children, depending on how children compare to the adults used in controlled human exposure studies (U.S. EPA, 2014, section 6.5.3).

A related source of uncertainty is that the risk assessment estimates O₃-induced decrements in asthmatics using the exposure-response relationship developed from data collected from healthy individuals. Although the evidence has been mixed (U.S. EPA, 2013, section 6.2.1.1), several studies have reported larger O₃-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). Consistent with the findings of the ISA (U.S. EPA, 2013, section 6.2.1.1), CASAC noted that “[a]sthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting ozone-induced pulmonary function decrements” (Frey, 2014,

p. 4). To the extent asthmatics experience larger O₃-induced lung function decrements than the healthy adults used to develop exposure-response relationships, the HREA could underestimate the impacts of O₃ exposures on lung function in asthmatics, including asthmatic children. The HREA notes that the magnitude this uncertainty might have on risk estimates remains unknown at this time (U.S. EPA, 2014, section 6.5.4).

3.2.3.2 Estimated Health Risks Associated with Short- or Long-Term O₃ Exposures, Based on Epidemiologic Studies

Risk estimates based on epidemiologic studies can provide perspective on the most serious O₃-associated public health outcomes (e.g., mortality, hospital admissions, emergency department visits) in populations that often include at-risk groups. The HREA estimates O₃-associated risks in 12 urban case study areas⁸⁴ using concentration-response relationships drawn from epidemiologic studies. These concentration-response relationships are based on “area-wide” average O₃ concentrations.⁸⁵ The HREA estimates risks for the years 2007 and 2009 in order to provide estimates of risk for a year with generally higher O₃ concentrations (2007) and a year with generally lower O₃ concentrations (2009) (U.S. EPA, 2014, section 7.1.1).

In the last review, epidemiologic-based risks were estimated for O₃ concentrations above mean “policy-relevant background concentrations.” As discussed above (Chapter 2), policy-relevant background (PRB) concentrations were defined as the distribution of ozone concentrations that would be observed in the U.S. in the absence of anthropogenic (man-made) emissions of ozone precursor emissions (e.g., VOC, CO, NO_x) in the U.S., Canada, and Mexico. This approach provided a focus on O₃ concentrations “that can be controlled by U.S. regulations (or through international agreements with neighboring countries)” (U.S. EPA, 2007, pp. 2-48 to 2-54).

As in the last review, we recognize that ambient O₃ concentrations, and therefore O₃-associated health risks, result from precursor emissions from various types of sources. Based on the air quality modeling discussed above in chapter 2, approximately 30 to 60% of average daytime O₃ during the warm season (i.e., daily maximum 8-hour concentrations averaged from April to October) is attributable to precursor emissions from U.S. anthropogenic sources (section 2.4.4). The remainder is attributable to precursor emissions from international anthropogenic

⁸⁴ The 12 urban areas evaluated are Atlanta, Baltimore, Boston, Cleveland, Denver, Detroit, Houston, Los Angeles, New York, Philadelphia, Sacramento, and St. Louis.

⁸⁵ In the epidemiologic studies that provide the health basis for HREA risk assessments, concentration-response relationships are based on daytime O₃ concentrations, averaged across multiple monitors within study areas. These daily averages are used as surrogates for the spatial and temporal patterns of exposures in study populations. Consistent with this approach, the HREA epidemiologic-based risk estimates also utilize daytime O₃ concentrations, averaged across monitors, as surrogates for population exposures. In this PA, we refer to these averaged concentrations as “area-wide” O₃ concentrations. Area-wide concentrations are discussed in more detail in section 3.1.4, above.

sources and natural sources. Because the HREA characterizes health risks from all O₃, regardless of source, risk estimates reflect emissions from U.S. anthropogenic, international anthropogenic, and natural sources.

In evaluating epidemiology-based risk estimates within the context of the adequacy of the current standard, we first consider the following question:

- **What are the nature and magnitude of the O₃-associated mortality and morbidity risks remaining upon adjustment of air quality to just meet the current O₃ standard?**

In addressing this question, we note that the HREA estimates mortality and morbidity risks associated with just meeting the current standard by applying concentration-response relationships from epidemiologic studies to the entire distributions of adjusted “area-wide” average O₃ concentrations present in urban case study areas (U.S. EPA, 2014, Chapter 7). Implicit in this approach to estimating risks is the assumption that concentration-response relationships are linear over those distributions. Therefore, as noted in section 3.2.1, when air quality is adjusted to just meet the current standard, risk estimates are influenced by the decreases in area-wide O₃ concentrations at the upper ends of warm season distributions and the increases in area-wide O₃ concentrations at the lower ends of those distributions (U.S. EPA, 2014, section 4.3.3.2, Figures 4-9 and 4-10).⁸⁶ When the decreases and increases are of the same magnitude, they result in the same degree of change in estimated risks, though opposite in direction. Therefore, seasonal estimates of O₃-associated mortality and morbidity risks either increase or decrease in response to air quality adjustment, depending on the seasonal balance between the modeled daily decreases in high area-wide O₃ concentrations and increases in low area-wide O₃ concentrations. One consequence is that the estimated impacts on mortality and morbidity risks of adjusting air quality to just meet the current standard are more modest, and less directionally consistent across urban case study areas, than on either exposures of concern or O₃-induced lung function decrements.

In the remainder of this section, we consider estimates of total (non-accidental) mortality and respiratory morbidity associated with short-term O₃ concentrations, and respiratory mortality associated with “long-term” O₃ concentrations.

Total Mortality – Short-Term O₃

Risk estimates for total mortality are based on concentration-response relationships described by Smith et al. (2009). To generate risk estimates, the HREA uses “area-wide”

⁸⁶ On a given day, area-wide O₃ concentrations and estimated risks decrease when the sum of the changes at monitors with decreasing O₃ (e.g., downwind of important NO_x sources, where the highest O₃ concentrations often occur) are larger than the sum of the changes at monitors with increasing O₃ (e.g., often in urban centers with high concentrations of NO_x emissions, where ambient O₃ concentrations are suppressed and thus relatively low). Area-wide O₃ concentrations and estimated risks increase when the opposite occurs.

averages of daily maximum 8-hour O₃ concentrations over the full monitoring periods in urban case study areas. When 2007 air quality was adjusted to the current standard (the year with generally “higher” O₃-associated risks), 10 of 12 urban case study areas exhibited either decreases or virtually no change in estimates of the number of O₃-associated deaths (U.S. EPA, 2014, Appendix 7B). Increases were estimated in two of the urban case study areas (Houston, Los Angeles) (U.S. EPA, 2014, Appendix 7B).⁸⁷

Figure 3-15 below presents estimates of O₃-associated all-cause mortality in urban case study areas for 2007 and 2009, with air quality adjusted to just meet the current O₃ standard. The HREA estimates that upon just meeting the current standard, O₃ could be associated with from 0.8 to 4.1% of all-cause mortality across the urban case study areas. This corresponds to approximately 60 to 3,200 O₃-associated deaths per season in individual urban case study areas, and approximately 7,000 to 7,500 O₃-associated deaths per season summed over the 12 urban case study areas (U.S. EPA, 2014, Tables 7-7 and 7-8).

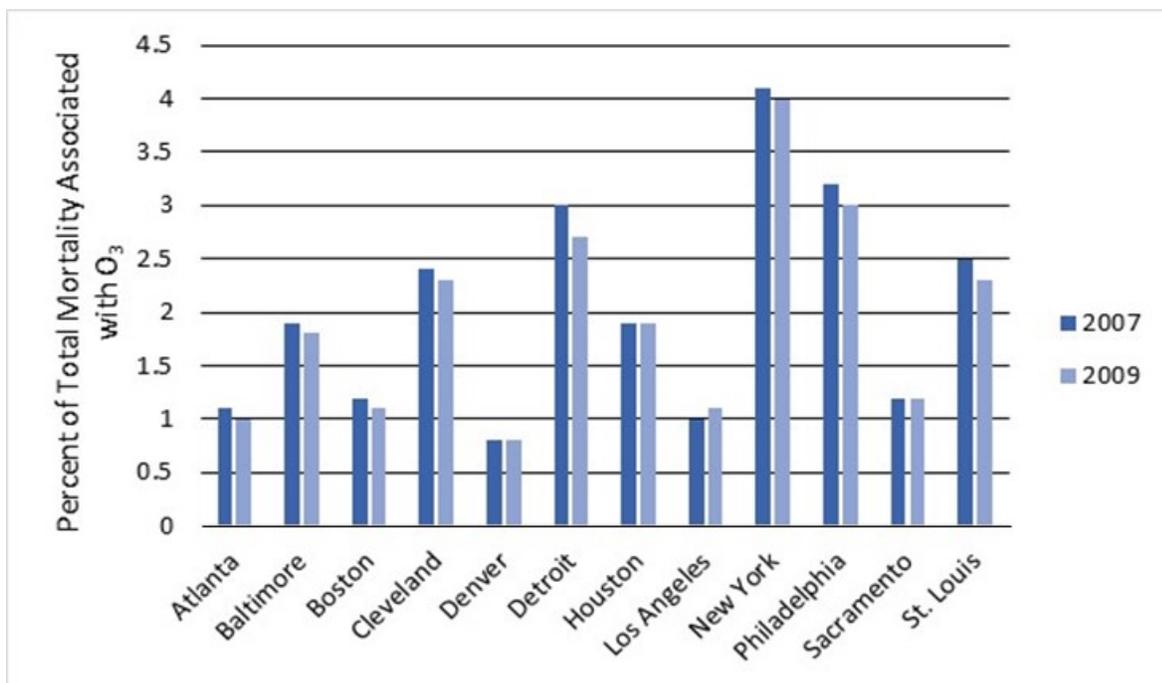


Figure 3-15. Percent of all-cause mortality associated with O₃ for air quality adjusted to just meet the current standard.

In considering the risk estimates presented in Figure 3-15, which are based on applying linear concentration-response relationships to the full distributions of daily 8-hour “area-wide”

⁸⁷ For 2009 (i.e., the year with generally lower O₃ concentrations), changes in risk were generally smaller than in 2007 (i.e., most changes about 2% or smaller). Increases were estimated for Houston, Los Angeles, and New York City.

O₃ concentrations, we note the ISA conclusion that there is less certainty in the shape of concentration-response functions for area-wide O₃ concentrations at the lower ends of warm season distributions (i.e., below about 20 to 40 ppb depending on the O₃ metric, health endpoint, and study population) (U.S. EPA, 2013, section 2.5.4.4). We also recognize that for the range of health endpoints evaluated, controlled human exposure and animal toxicological studies provide greater certainty in the increased incidence, magnitude, and severity of effects at higher exposure concentrations (discussed in sections 3.1.2.2 and 3.1.4.2, above).⁸⁸ Thus, in addition to considering estimates of total O₃-associated risks, we also consider the extent to which risks are associated with days with higher, versus lower, area-wide O₃ concentrations.

Figure 3-16 presents risk estimates, summed across urban case study areas, for days with area-wide concentrations at or above 20, 40, and 60 ppb. Daytime O₃ concentrations in the upper portion of the distribution of area-wide concentrations (e.g., at or above 40 or 60 ppb) are estimated to be associated with hundreds to thousands of deaths per year in urban case study areas.⁸⁹

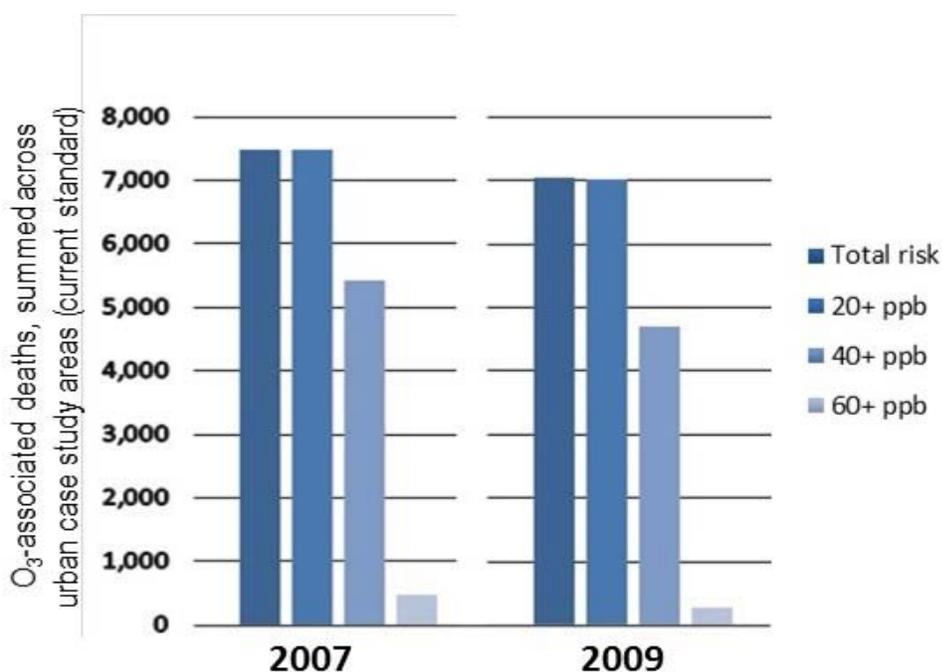


Figure 3-16. Estimated O₃-associated deaths attributable to various area-wide average O₃ concentrations, with air quality adjusted to just meet current standard.

⁸⁸ As discussed in section 3.1.4.2, as ambient concentrations increase the potential for exposures to higher O₃ concentrations also increases. Thus with increasing ambient concentrations, controlled human exposure and animal toxicological studies support the increased incidence, magnitude, and severity of O₃-attributable effects.

⁸⁹ The relatively small proportion of O₃-associated deaths attributable to days with area-wide concentrations of 60 ppb or greater reflects the relatively small proportion of days with such elevated area-wide concentrations.

Respiratory Mortality – “Long-Term” O₃

The HREA estimates the risk of respiratory mortality associated with long-term O₃ exposures, based on the study by Jerrett et al. (2009) (U.S. EPA, 2014, Chapter 7). As discussed above (section 3.1.4.3), Jerrett et al. (2009) reported that when seasonal averages of 1-hour daily maximum O₃ concentrations ranged from 33 to 104 ppb, there was no statistically significant deviation from a linear concentration-response relationship between O₃ and respiratory mortality across 96 U.S. cities (U.S. EPA, 2013, section 7.7). However, the authors reported “limited evidence” for an effect threshold at an O₃ concentration of 56 ppb (p=0.06). In communications with EPA staff (described in Sasser, 2014), the study authors indicated that it is not clear whether a threshold model is a better predictor of respiratory mortality than the linear model, and that “considerable caution should be exercised in accepting any specific threshold.” Consistent with this communication, the HREA estimated respiratory mortality associated with long-term O₃ concentrations based on the linear model from the published study, and in a series of sensitivity analyses with models that included thresholds ranging from 40 to 60 ppb (U.S. EPA, 2014, Figure 7-9).

To generate risk estimates, the HREA uses “area-wide” averages of 1-hour daily maximum O₃ concentrations during the warm season (April to September). When 2007 air quality was adjusted to just meet the current standard (i.e., the year with generally higher O₃ concentrations) all 12 of the urban case study areas exhibited decreases in estimated O₃-associated respiratory mortality (i.e., compared to recent, unadjusted air quality). For 2009 adjusted air quality (i.e., the year with generally lower O₃ concentrations), urban case study areas exhibited either no change in estimated risk, or decreases in risk that were smaller than those for 2007 (U.S. EPA, 2014, Appendix 7B, Tables 7B-6 and 7B-7). Risk estimates based on the linear model, for air quality adjusted to just meet the current standard, are presented below in Figure 3-17.

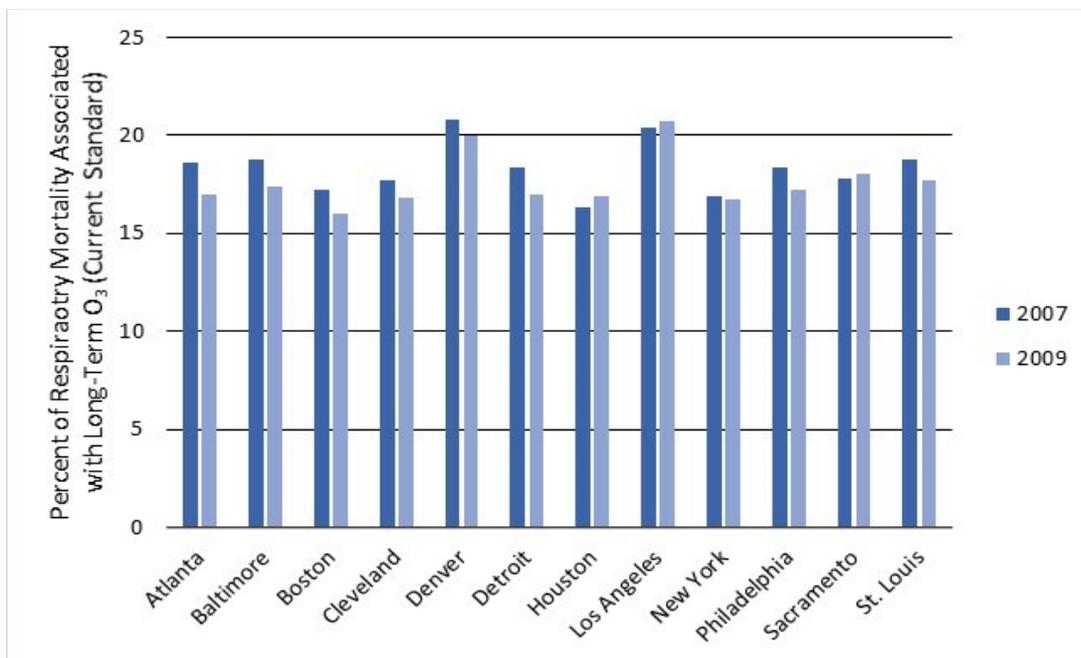


Figure 3-17. Percent of baseline respiratory mortality estimated to be associated with long-term O₃.

Based on a concentration-response function that is linear over the entire distribution of long-term O₃ concentrations, O₃ is estimated to be associated with approximately 16 to 21% of respiratory deaths in urban case study areas during the warm season. This corresponds to approximately 300 to 2,100 O₃-associated deaths per season in individual urban case study areas, and a total of approximately 8,000 to 9,000 O₃-associated deaths summed across all 12 case study areas. Based on threshold models, HREA sensitivity analyses indicate that the number of respiratory deaths associated with long-term O₃ concentrations could potentially be considerably lower (i.e., by more than 75% if a threshold exists at 40 ppb, and by about 98% if a threshold exists at 56 ppb) (U.S. EPA, 2014, Figure 7-9).

Hospital Admissions, Emergency Department Visits, and Asthma Exacerbations

Risk estimates for respiratory-related hospital admissions, emergency department visits, and asthma exacerbations associated with air quality adjusted to just meet the current standard are based on several studies, as presented in Table 7-2 of the HREA (U.S. EPA, 2014).⁹⁰ Estimates indicate that O₃-associated respiratory-related hospital admissions generally account for approximately 2 to 3% of total respiratory-related admissions in urban case study locations. Depending on the city, this corresponds to 10's to 100's of O₃-associated hospital admissions per season. Estimates indicate that O₃-associated respiratory-related emergency department visits

⁹⁰As with respiratory mortality above, the HREA does not characterize distributions of respiratory morbidity risks over distributions of ambient O₃ concentrations. Therefore, in considering respiratory morbidity risks we evaluate estimates of total risk.

account for approximately 3 to 20% of total respiratory-related emergency department visits in Atlanta and New York City (corresponding to thousands of visits per season in these two cities), and that O₃-associated asthma exacerbations account for approximately 15 to 30% of total exacerbations in Boston (30,000 to 80,000 exacerbations per season). Full estimates are presented in Tables 7-9 to 7-11 in the HREA (U.S. EPA, 2014).

Based on the detailed information presented in Chapter 7 of the HREA (U.S. EPA, 2014), we note the following key observations:

1. In focusing on total risk, the current standard is estimated to allow thousands of O₃-associated deaths per year in the urban case study areas. These estimates are based on concentration-response functions from epidemiologic studies that used either 8-hour daily O₃ concentrations (total mortality associated with short-term O₃) or seasonal averages of 1-hour daily O₃ concentrations (respiratory mortality associated with long-term O₃).
2. In focusing on the risks associated with the upper portions of distributions of ambient concentrations, the current standard is estimated to allow hundreds to thousands of O₃-associated deaths per year in the urban case study areas. These estimates are based on concentration-response functions from an epidemiologic study that evaluated associations between 8-hour daily O₃ concentrations and total mortality.
3. In urban case study areas, the current standard is estimated to allow tens to thousands of O₃-associated morbidity events per year. Distributions of O₃-associated morbidity over distributions of ambient O₃ concentrations would likely be similar to mortality, though the HREA did not analyze such distributions for morbidity endpoints.

In further considering estimated O₃-associated mortality and morbidity risks from the HREA, we next consider the following question:

- **What are the important sources of uncertainty associated with mortality and morbidity risk estimates?**

Compared to estimates of O₃ exposures of concern and estimates of O₃-induced lung function decrements (discussed above), the HREA conclusions reflect somewhat lower confidence in epidemiologic-based risk estimates, given important uncertainties. In particular, the HREA highlights the unexplained heterogeneity in effect estimates between locations, the potential for exposure measurement errors, and uncertainty in the interpretation of the shape of concentration-response functions at lower O₃ concentrations (U.S. EPA, 2014, section 9.6). The HREA also concludes that lower confidence should be placed in the results of the assessment of respiratory mortality risks associated with long-term O₃ exposures, primarily because that analysis is based on only one study (even though that study is well-designed) and because of the uncertainty in that study about the existence and level of a potential threshold in the concentration-response function (U.S. EPA, 2014, section 9.6). This section discusses some of the key uncertainties in epidemiologic-based risk estimates, with a focus on uncertainties that can

have particularly important implications for our consideration of epidemiology-based risk estimates in this PA

Estimating air quality that just meets the current standard based on modeled responses to reductions in NO_x emissions generally reduces O₃-associated mortality and morbidity risks in locations and time periods with relatively high ambient O₃ concentrations and increases risks in locations and time periods with relatively low concentrations. When evaluating uncertainties in epidemiologic risk estimates, it is important to consider the extent to which the pattern of air quality changes in response to reductions in NO_x emissions is representative of trends in ambient O₃; the extent to which estimated changes in risks in urban case study areas are representative of the changes that would be experienced broadly across the U.S. population; and the extent to which the O₃ response to reductions in precursor emissions could differ with emissions reduction strategies that are different from those used in REA risk estimates.

To evaluate the first issue, the HREA conducted a national analysis evaluating trends in monitored ambient O₃ concentrations during a time period when the U.S. experienced large-scale reductions in NO_x emissions (i.e., 2001 to 2010). Analyses of trends in monitored O₃ indicate that over such a time period, the upper end of the distribution of monitored O₃ concentrations (i.e., indicated by the 95th percentile) generally decreased in urban and non-urban locations across the U.S. (U.S. EPA, 2014, Figure 8-29). During this same time period, median O₃ concentrations decreased in suburban and rural locations, and in some urban locations. However, median concentrations increased in some large urban centers (U.S. EPA, 2014, Figure 8-28). As discussed in the REA, and above (II.C.1), these increases in median concentrations likely reflect the increases in relatively low O₃ concentrations that can occur near important sources of NO_x upon reductions in NO_x emissions (U.S. EPA, 2014, section 8.2.3.1). These patterns of monitored O₃ during a period when the U.S. experienced large reductions in NO_x emissions are qualitatively consistent with the modeled responses of O₃ to reductions in NO_x emissions.

To evaluate the second issue, the HREA conducted national air quality modeling analyses. These analyses estimated the proportion of the U.S. population living in locations where seasonal averages of daily O₃ concentrations are estimated to decrease in response to reductions in NO_x emissions, and the proportion living in locations where such seasonal averages are estimated to increase. Given the strong relationship between changes in seasonal averages of daily O₃ concentrations and changes in seasonal risk estimates, this analysis informs consideration of the extent to which the risk results in urban case study areas represent the U.S. population as a whole. This representativeness analysis indicates that the 12 urban case study areas may not represent the response of O₃ in other populated areas of the U.S., including suburban areas, smaller urban areas, and rural areas. This analysis also indicates 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 O₃ concentrations. One implication of these results is that HREA risk estimates for the urban case study areas may understate the average reduction in O₃-associated mortality and morbidity risk that would be experienced across the U.S. population upon reducing NO_x emissions (U.S. EPA, 2014, sections 8.2.3.2 and 8.4).

To evaluate the third issue, the HREA assesses the O₃ air quality response to reducing both NO_x and VOC (i.e., in addition to assessing reductions in NO_x emissions alone) for a subset of seven urban case study areas. As noted above (section 3.2.1), in most of these urban case study areas the inclusion of VOC emissions reductions did not alter the NO_x emissions reductions required to meet the current or alternative standards.⁹¹ However, the addition of VOC reductions generally resulted in larger decreases in mid-range O₃ concentrations (25th to 75th percentiles) (U.S. EPA, 2014, Appendix 4D, section 4D-4.7).⁹² In addition, in all seven of the urban case study areas evaluated, the increases in low O₃ concentrations were smaller for the NO_x/VOC scenarios than the NO_x alone scenarios (U.S. EPA, 2014, Appendix 4D, section 4D-4.7). This was most apparent for Denver, Houston, Los Angeles, New York, and Philadelphia. Given the impacts on total risk estimates of increases in low O₃ concentrations, these results suggest that in some locations better-optimized emissions reduction strategies could result in larger reductions in O₃-associated mortality and morbidity than indicated in the HREA core estimates.

Section 7.4 of the HREA also highlights some additional uncertainties associated with epidemiologic-based risk estimates (U.S. EPA, 2014). This section of the HREA identifies and discusses sources of uncertainty and presents a qualitative evaluation of key parameters that can introduce uncertainty into risk estimates (U.S. EPA, 2014, Table 7-4). For several of these parameters the HREA also presents quantitative sensitivity analyses (U.S. EPA, 2014, section 7.5.3). Of the uncertainties discussed in Chapter 7 of the HREA, those related to the application of concentration-response functions from epidemiologic studies can have particularly important implications for our consideration of epidemiology-based risk estimates in this PA, as discussed below.

An important uncertainty is the shape of concentration-response functions at low ambient O₃ concentrations (U.S. EPA, 2014, Table 7-4).⁹³ Consistent with the ISA conclusion that there

⁹¹ The exception is Chicago and Denver, for which the HREA risk estimates are based on reductions in both NO_x and VOC (U.S. EPA, 2014, section 4.3.3.1). Emissions of NO_x and VOC were reduced by equal percentages, a scenario not likely to reflect the optimal combination for reducing risks.

⁹² This was the case for all of the urban case study areas evaluated, with the exception of New York (U.S. EPA, 2014, Appendix 4D, section 4D-4.7).

⁹³ A related uncertainty is the existence, or not, of a threshold. The HREA addresses this issue for long-term O₃ by evaluating risks in models that include potential thresholds (see above).

is no discernible population threshold in O₃-associated health effects, the HREA estimates epidemiology-based mortality and morbidity risks for entire distributions of ambient O₃ concentrations, with the assumption that concentration-response relationships remain linear over those distributions. In addition, in recognition of the ISA conclusion that certainty in the shape of O₃ concentration-response functions decreases at low ambient concentrations, the HREA also estimates distributions of total mortality incidence for various portions of the distribution of ambient O₃ concentrations. In this PA, we consider both types of risk estimates while recognizing that we have greater certainty in the increased incidence and severity of O₃-attributable effects at higher ambient O₃ concentrations (which drive higher exposure concentrations, section 3.2.2 above), as compared to lower concentrations.⁹⁴

The HREA also notes important uncertainties associated with using a concentration-response relationship developed for a particular population in a particular location to estimate health risks in different populations and locations (U.S. EPA, 2014, Table 7-4). As discussed above, concentration-response relationships derived from epidemiologic studies reflect the spatial and temporal patterns of population exposures during the study. The HREA applies concentration-response relationships from epidemiologic studies to adjusted air quality in study areas that are different from, and often larger in spatial extent than, the areas used to generate the relationships. This approach ensures the inclusion of the actual non-attainment monitors that often determine the magnitude of emissions reductions for the air quality adjustments throughout the urban case study areas. This approach also allows the HREA to estimate patterns of health risks more broadly across a larger area, including a broader range of air quality concentrations and a larger population. The HREA notes that it is not possible to quantify the impacts of this uncertainty on risk estimates in most urban case study locations, though the HREA notes that mortality effect estimates for different portions of the New York City CBSA-based assessment area vary by a factor of almost 10 (U.S. EPA, 2014, section 7.5.3).

An additional, related uncertainty is that associated with applying concentration-response functions from epidemiologic studies to adjusted air quality. Concentration-response functions from the O₃ epidemiologic studies used in the HREA are based on associations between day to day variation in “area-wide” O₃ concentrations (i.e., averaged across multiple monitors) and variation in health effects. Epidemiologic studies use these area-wide O₃ concentrations, which reflect the particular spatial and temporal patterns of ambient O₃ present in study locations, as surrogates for the pattern of O₃ exposures experienced by study populations. To the extent adjusting O₃ concentrations to just meet the current standard results in important alterations in

⁹⁴ As discussed above, we also consider the potential implications of the existence of a threshold in the association between long-term O₃ and respiratory mortality.

the spatial and/or temporal patterns of ambient O₃, there is uncertainty in the appropriateness of applying concentration-response functions from epidemiologic studies to estimate health risks associated with adjusted O₃ air quality.⁹⁵ In particular, this uncertainty could be important to the extent that (1) factors associated with space modify the effects of O₃ on health or (2) spatial mobility is a key driver of individual-level exposures. Although the impact of this uncertainty on risk estimates cannot be quantified (U.S. EPA, 2014, Table 7-4), it has the potential to become more important as air quality adjustment results in larger changes in spatial and temporal patterns of ambient O₃ concentrations across urban case study areas.

There is also uncertainty related specifically to the public health importance of the increases in relatively low O₃ concentrations following air quality adjustment. This uncertainty relates to the fact that HREA risk estimates are equally influenced by decreasing high concentrations and increasing low concentrations, when the increases and decreases are of equal magnitude. Even on days with increases in relatively low area-wide average concentrations, resulting in increases in estimated risks, some portions of the urban case study areas could experience decreases in high O₃ concentrations. To the extent it is reasonable to conclude that O₃-attributable effects are more strongly supported for higher ambient concentrations (see above), likely resulting in higher exposure concentrations for some portions of study areas, the impacts on risk estimates of increasing low O₃ concentrations reflect an important source of uncertainty.

The use of a national concentration-response function to estimate respiratory mortality associated with long-term O₃ is a source of uncertainty. Risk estimates generated in sensitivity analyses using region-specific effect estimates differ substantially from the core estimates based on a single national-level effect estimate (U.S. EPA, 2014; Table 7-14). Furthermore, the risk estimates generated using the regional effect estimates display considerable variability across urban case study areas (U.S. EPA, 2014; Table 7-14), reflecting the substantial variability in the underlying effect estimates (see Jerrett et al., 2009, Table 4). While the results of the HREA sensitivity analyses evaluating this uncertainty point to the potential for regional heterogeneity in the long-term risk estimates, the relatively large confidence intervals associated with regional effect estimates resulted in the HREA conclusion that staff does not have confidence in the regionally-based risk estimates themselves.

Finally, we note the HREA does not quantify any reductions in risk that could be associated with reductions in the ambient concentrations of pollutants other than O₃, resulting from control of NO_x. For example as discussed in chapter 2 of this PA, NO_x emissions contribute to ambient NO₂, and NO_x and VOCs can contribute to secondary formation of PM_{2.5}

⁹⁵ As discussed above (section 3.2.1), decreasing modeled NO_x emissions to just meet the current standard can dramatically alter the spatial and temporal patterns of ambient O₃ concentrations across urban case study areas.

constituents, including ammonium sulfate (NH₄SO₄), ammonium nitrate (NH₄NO₃), and organic carbon (OC). Therefore, at some times and in some locations, control strategies that would reduce NO_x emissions (i.e., to meet an O₃ standard) could reduce ambient concentrations of NO₂ and PM_{2.5}, resulting in health benefits beyond those directly associated with reducing ambient O₃ concentrations.⁹⁶

3.3 CASAC ADVICE AND PUBLIC COMMENTERS' VIEWS ON THE ADEQUACY OF THE CURRENT STANDARD

Beyond the evidence- and risk/exposure-based information discussed above, staff has also taken into account the comments and advice of CASAC, based on their reviews of the ISA, the HREA and PA, as well as comments provided by public commenters. The range of views summarized here generally reflects differing judgments as to the relative weight to place on various types of evidence, the exposure- and risk-based information, and the associated uncertainties, as well as differing judgments about the importance of various O₃-related health effects from a public health perspective.

Following the 2008 decision to revise the primary O₃ standard by setting the level at 0.075 ppm (75 ppb), CASAC strongly questioned whether the standard met the requirements of the CAA, further described below. In September 2009, EPA announced its intention to reconsider the 2008 standards, issuing a notice of proposed rulemaking in January 2010 (FR 75 2938). Soon after, EPA solicited CASAC review of that proposed rule and in January 2011 solicited additional advice. This proposal was based on the scientific and technical record from the 2008 rulemaking, including public comments and CASAC advice and recommendations. As further described in section 1.2.2 above, EPA in the fall of 2011 did not revise the standard as part of the reconsideration process but decided to coordinate further proceedings on the reconsideration rulemaking with this ongoing periodic review. Accordingly, in this section we describe CASAC's advice related to the 2008 final decision and the subsequent reconsideration, as well as its advice on the NAAQS review that was initiated in September 2008.

In April 2008, the members of the CASAC Ozone Review Panel sent a letter to EPA stating “[I]n our most-recent letters to you on this subject—dated October 2006 and March 2007—the CASAC unanimously recommended selection of an 8-hour average Ozone NAAQS within the range of 0.060 to 0.070 parts per million [60 to 70 ppb] for the primary (human health-based) Ozone NAAQS” (Henderson, 2008). The letter continued:

⁹⁶ We expect little focus by states on controlling NO_x for purposes of controlling PM_{2.5} given the more efficient control of PM_{2.5} through reduction of SO₂ and direct PM_{2.5} emissions in most locations. Thus, consideration in this review of reductions in ambient PM_{2.5} resulting from putative NO_x control would not double-count PM_{2.5} emission reductions.

The CASAC now wishes to convey, by means of this letter, its additional, unsolicited advice with regard to the primary and secondary Ozone NAAQS. In doing so, the participating members of the CASAC Ozone Review Panel are unanimous in strongly urging you or your successor as EPA Administrator to ensure that these recommendations be considered during the next review cycle for the Ozone NAAQS that will begin next year ... numerous medical organizations and public health groups have also expressed their support of these CASAC recommendations' ... [The CASAC did] not endorse the new primary ozone standard as being sufficiently protective of public health. The CASAC—as the Agency's statutorily-established science advisory committee for advising you on the national ambient air quality standards—unanimously recommended decreasing the primary standard to within the range of 0.060–0.070 ppm [60 to 70 ppb]. It is the Committee's consensus scientific opinion that your decision to set the primary ozone standard above this range fails to satisfy the explicit stipulations of the Clean Air Act that you ensure an adequate margin of safety for all individuals, including sensitive populations.

In response to EPA's solicitation of their advice on the Agency's proposed rulemaking as part of the reconsideration, CASAC conveyed support (Samet, 2011).

CASAC fully supports EPA's proposed range of 0.060 – 0.070 parts per million (ppm) for the 8-hour primary ozone standard. CASAC considers this range to be justified by the scientific evidence as presented in the Air Quality Criteria for Ozone and Related Photochemical Oxidants (March 2006) and Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper (July 2007). As stated in our letters of October 24, 2006, March 26, 2007 and April 7, 2008 to former Administrator Stephen L. Johnson, CASAC unanimously recommended selection of an 8-hour average ozone NAAQS within the range proposed by EPA (0.060 to 0.070 ppm). In proposing this range, EPA has recognized the large body of data and risk analyses demonstrating that retention of the current standard would leave large numbers of individuals at risk for respiratory effects and/or other significant health impacts including asthma exacerbations, emergency room visits, hospital admissions and mortality.

In response to EPA's request for additional advice on the reconsideration in 2011, CASAC reaffirmed their conclusion that “the evidence from controlled human and epidemiological studies strongly supports the selection of a new primary ozone standard within the 60 – 70 ppb range for an 8-hour averaging time” (Samet, 2011). As requested by EPA, CASAC's advice and recommendations were based on the scientific and technical record from the 2008 rulemaking. In considering the record for the 2008 rulemaking, CASAC stated the following to summarize the basis for their conclusions (Samet, 2011, pp. ii to iii).

- *The evidence available on dose-response for effects of ozone shows associations extending to levels within the range of concentrations currently experienced in the United States.*

- *There is scientific certainty that 6.6-hour exposures with exercise of young, healthy, non-smoking adult volunteers to concentrations ≥ 80 ppb cause clinically relevant decrements of lung function.*
- *Some healthy individuals have been shown to have clinically relevant responses, even at 60 ppb.*
- *Since the majority of clinical studies involve young, healthy adult populations, less is known about health effects in such potentially ozone sensitive populations as the elderly, children and those with cardiopulmonary disease. For these susceptible groups, decrements in lung function may be greater than in healthy volunteers and are likely to have a greater clinical significance.*
- *Children and adults with asthma are at increased risk of acute exacerbations on or shortly after days when elevated ozone concentrations occur, even when exposures do not exceed the NAAQS concentration of 75 ppb.*
- *Large segments of the population fall into what EPA terms a “sensitive population group,” i.e., those at increased risk because they are more intrinsically susceptible (children, the elderly, and individuals with chronic lung disease) and those who are more vulnerable due to increased exposure because they work outside or live in areas that are more polluted than the mean levels in their communities.*

With respect to evidence from epidemiologic studies, CASAC stated “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.” (Samet, 2011, p. 10).

Following their review of the second draft PA in the current review, which considers an updated scientific and technical record since the 2008 rulemaking, CASAC concluded that “there is clear scientific support for the need to revise the standard” (Frey, 2014, p. ii). In particular, CASAC noted the following (Frey, 2014, p. 5):

[T]he scientific evidence provides strong support for the occurrence of a range of adverse respiratory effects and mortality under air quality conditions that would meet the current standard. Therefore, CASAC unanimously recommends that the Administrator revise the current primary ozone standard to protect public health.

In supporting these conclusions, CASAC judged that the strongest evidence comes from controlled human exposure studies of respiratory effects. The Committee specifically noted that “the combination of decrements in FEV₁ 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, 2014, p. 5). CASAC further judged that “the level at

which adverse effects might be observed would likely be lower for more sensitive subgroups, such as those with asthma” (Frey, 2014, p. 5).

With regard to lung function risk estimates based on information from controlled human exposure studies, CASAC concluded that “estimation of FEV1 decrements of $\geq 15\%$ is appropriate as a scientifically relevant surrogate for adverse health outcomes in active healthy adults, whereas an FEV1 decrement of $\geq 10\%$ is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014, p. 3). The Committee further concluded that “[a]sthmatic subjects appear to be at least as sensitive, if not more sensitive, than non-asthmatic subjects in manifesting O₃-induced pulmonary function decrements” (Frey, 2014, p. 4). In considering estimates of the occurrence of these decrements in urban case study areas, CASAC specifically noted that the current standard is estimated to allow 11 to 22% of school age children to experience at least one day with an FEV₁ decrement $\geq 10\%$.

While CASAC judged that controlled human exposure studies of respiratory effects provide the strongest evidence supporting their conclusion on the current standard, the Committee judged that there is also “sufficient scientific evidence based on epidemiologic studies for mortality and morbidity associated with short-term exposure to ozone at the level of the current standard” (Frey, 2014, p. 5). In support of the biological plausibility of the associations reported in these epidemiologic studies, CASAC noted that “[r]ecent animal toxicological studies support identification of modes of action and, therefore, the biological plausibility associated with the epidemiological findings” (Frey, 2014, p. 5).

Consistent with the advice of CASAC, several public commenters supported revising the primary O₃ standard to provide increased public health protection. In considering the available evidence as a basis for their views, these commenters generally noted that the health evidence is stronger in the current review than in past reviews. These commenters often noted that causal determinations were strengthened to “likely causal” for total mortality and cardiovascular effects from short-term O₃ exposures, and for respiratory effects from long-term O₃ exposures. These commenters also noted the increase in controlled human exposure studies showing lung function decrements and new evidence of inflammation in healthy young adults at 60 ppb O₃, as well as the increase in the number of epidemiologic studies showing consistent, positive associations between O₃ exposures and hospital admissions, emergency department visits, and premature mortality. Some commenters noted that children have long been known to be more vulnerable than adults to the effects of air pollution due to ongoing lung development, the greater permeability of their airways epithelial layer, and greater resting minute ventilation (when normalized to body mass or lung volume) resulting in increased exposure compared with adults. These commenters noted that adverse effects have been described on early lung development and the evidence for O₃ as a contributor to childhood respiratory disease is extremely strong. They

expressed the view that O₃ in particular has long been known to induce asthma exacerbations in children and, in one well characterized population-based cohort study in California, exposure to ozone was associated with the development of asthma. Some commenters expressed the view that young children and small infants should be included in the exposure and risk assessment. Other commenters noted that the health endpoints considered in the HREA are limited, and do not represent the comprehensive array of health effects attributable to O₃ exposure.

In contrast to the views discussed above, other public commenters opposed considering revised standards. These commenters discussed a variety of reasons for their views. A number of commenters expressed the view that EPA should not lower the level of the standard because a lower level would be closer to background O₃ concentrations. In addition, several commenters challenged the interpretation of the evidence presented in the ISA. For example, some commenters questioned the ISA's judgments regarding the strength of evidence for cardiovascular system effects from short-term O₃ exposures. With respect to the risk assessment, several commenters expressed the view that the EPA should only estimate risks above O₃ background concentrations, or above threshold concentrations. In some cases these commenters noted that (1) the O₃ mode of action indicates that there are thresholds for O₃ effects; (2) that these thresholds are considered in the lung function risk assessment; and (3) that there is no reason to believe that similar thresholds would not also be associated with other health effects, particularly more serious effects. Some commenters also expressed the view that, based on the mortality and morbidity risk estimates in the HREA, there is little to no difference between the risks estimated for the current O₃ standard and the risks estimated for revised standards with lower levels. These commenters concluded that the HREA and PA have not shown that the public health improvements likely to be achieved by a revised O₃ standard would be greater than the improvements likely to be achieved by the current standard.

3.4 STAFF CONCLUSIONS ON ADEQUACY OF PRIMARY STANDARD

This section presents staff's conclusions for the Administrator to consider in deciding whether it is appropriate to revise the existing primary O₃ standard. Staff conclusions are based on our consideration of the assessment and integrative synthesis of the evidence presented in the ISA, the air quality distributions in locations of selected epidemiologic studies, exposure and risk analyses in the HREA, the advice of CASAC, and comments received from members of the public.

As an initial matter, staff concludes that reducing precursor emissions to achieve O₃ concentrations that meet the current standard will provide important improvements in public health protection. This initial conclusion is based on (1) the strong body of scientific evidence indicating a wide range of adverse health outcomes attributable to exposures to O₃

concentrations commonly found in the ambient air and (2) estimates indicating decreased occurrences of O₃ exposures of concern and decreased health risks upon meeting the current standard, compared to recent air quality.

Strong support for this initial conclusion is provided by controlled human exposure studies of respiratory effects, and by quantitative estimates of exposures of concern and lung function decrements based on information in these studies. Analyses in the HREA estimate that the percentages of children (i.e., all children and children with asthma) in urban case study areas experiencing exposures of concern, or experiencing abnormal and potentially adverse lung function decrements, are consistently lower for air quality that just meets the current O₃ standard than for recent air quality. The HREA estimates such reductions consistently across the urban case study areas evaluated and throughout various portions of individual urban case study areas, including in urban cores and the portions of case study areas surrounding urban cores. These reductions in exposures of concern and O₃-induced lung function decrements reflect the consistent decreases in the highest O₃ concentrations following reductions in precursor emissions to meet the current standard. Thus, populations in both urban and non-urban areas would be expected to experience important reductions in O₃ exposures and O₃-induced lung function risks upon meeting the current standard.

Support for this initial conclusion is also provided by estimates of O₃-associated mortality and morbidity based on application of concentration-response relationships from epidemiologic studies to air quality adjusted to just meet the current standard. These estimates, which are based on the assumption that concentration-response relationships are linear over entire distributions of ambient O₃ concentrations, are associated with uncertainties that complicate their interpretation (discussed below). However, risk estimates for effects associated with short- and long-term O₃ exposures, combined with the HREA's national analysis of O₃ responsiveness to reductions in precursor emissions and the consistent reductions estimated for the highest ambient O₃ concentrations, suggest that O₃-associated mortality and morbidity would be expected to decrease nationwide following reductions in precursor emissions to meet the current O₃ standard.

As discussed in section 3.2.3.2, reductions in O₃ precursor emissions (i.e., NO_x) could also increase public health protection by reducing the ambient concentrations of pollutants other than O₃. For example, NO_x emissions contribute to ambient NO₂, and NO_x and VOCs can contribute to secondary formation of PM_{2.5} constituents, including ammonium sulfate (NH₄SO₄), ammonium nitrate (NH₄NO₃), and organic carbon (OC). Therefore, at some times and in some locations, control strategies that would reduce NO_x emissions (i.e., to meet an O₃ standard) could reduce ambient concentrations of NO₂ and PM_{2.5}, resulting in health benefits beyond those directly associated with reducing ambient O₃ concentrations.

We next revisit the overarching policy question for this chapter, taking into consideration the responses to the specific questions focused on the adequacy of the current primary O₃ standard, as discussed above.

- **Does the currently available scientific evidence and exposure/risk information, as reflected in the ISA and HREA, support or call into question the adequacy of the protection afforded by the current primary O₃ standard?**

In considering the available evidence and information, staff concludes that the O₃-attributable health effects estimated to be allowed by air quality that meets the current primary standard for O₃ can reasonably be judged important from a public health perspective. Thus, we conclude that the available health evidence and exposure/risk information call into question the adequacy of the public health protection provided by the current standard. We further conclude that it is appropriate in this review to consider alternative standards that would increase public health protection, compared to the current standard. The basis for these conclusions is discussed below.

Studies evaluated since the completion of the 2006 O₃ AQCD support and expand upon the strong body of evidence that, in the last review, indicated a causal relationship between short-term O₃ exposures and respiratory health effects. Together, experimental and epidemiologic studies support conclusions regarding a continuum of O₃ respiratory effects ranging from small reversible changes in pulmonary function to more serious effects that can result in respiratory-related emergency department visits, hospital admissions, and/or mortality. Recent animal toxicological studies support descriptions of modes of action for these respiratory effects and augment support for biological plausibility for the role of O₃ in reported effects. With regard to mode of action, evidence indicates that antioxidant capacity may modify the risk of respiratory morbidity associated with O₃ exposure. In addition, based on the consistency of findings across studies and evidence for the coherence of results from different scientific disciplines, strong evidence indicates that certain populations are at increased risk of experiencing O₃-related effects. These include populations and lifestages identified in previous reviews (i.e., people with asthma, children, older adults, outdoor workers) and populations identified since the last review (i.e., people with certain genotypes related to anti-oxidant and/or anti-inflammatory status; people with reduced intake of certain nutrients, such as Vitamins C and E).

Evidence for adverse respiratory health effects attributable to “long-term” or repeated daily O₃ exposures is much stronger than in previous reviews, and the ISA concludes that the evidence supports a “likely to be” causal relationship between such O₃ exposures and adverse respiratory health effects. Uncertainties related to the extrapolation of data generated by rodent toxicology studies to the understanding of health effects in humans have been reduced by studies

in non-human primates and by recent epidemiologic studies. The evidence available in this review includes new epidemiologic studies using a variety of designs and analysis methods, conducted by different research groups in different locations, evaluating the relationships between long-term O₃ exposures and measures of respiratory morbidity and mortality. New evidence supports associations between long-term or repeated O₃ exposures and the development of asthma in children, with several studies reporting interactions between genetic variants and such O₃ exposures. Studies also report associations between long-term or repeated O₃ exposure and asthma prevalence, asthma severity and control, respiratory symptoms among asthmatics, and respiratory mortality.

In considering the O₃ exposure concentrations reported to elicit respiratory effects, we note that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following exposures to specific O₃ concentrations. Consistent with this, CASAC also concluded 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, 2014, p. 5). As discussed above, recent evidence includes controlled human exposure studies reporting lung function decrements and pulmonary inflammation in healthy adults engaged in intermittent, moderate exertion following 6.6 hour exposures to O₃ concentrations as low as 60 ppb, and lung function decrements and respiratory symptoms following exposures to concentrations as low as 72 ppb.⁹⁷ Compared to the evidence available in the last review, these studies have strengthened support for the occurrence of abnormal and adverse respiratory effects attributable to short-term exposures to O₃ concentrations below 80 ppb.⁹⁸ Consistent with CASAC advice, we conclude that exposures to such O₃ concentrations are potentially important from a public health perspective given the following:

1. The respiratory effects reported to occur in healthy adults following exposures to O₃ concentrations of 60 and 72 ppb, while at moderate exertion, can reasonably be judged adverse based on ATS criteria and advice from CASAC. In considering the 72 ppb exposure concentration, CASAC noted that “the combination of decrements in FEV₁ 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, 2014, p. 5). With regard to 60 ppb O₃, CASAC agreed that “a level of 60 ppb corresponds to the lowest exposure concentration demonstrated to result in

⁹⁷ As noted above, for the 70 ppb exposure concentration Schelegle et al. (2009) reported that the actual mean exposure concentration was 72 ppb.

⁹⁸ Cf. *Mississippi*, 744 F.3d at 1350 (“Perhaps more studies like the Adams studies will yet reveal that the 0.060 ppm level produces significant adverse decrements that simply cannot be attributed to normal variation in lung function.”).

lung function decrements large enough to be judged an abnormal response by ATS and that could be adverse in individuals with lung disease” (Frey, 2014, p. 7). CASAC further noted that “a level of 60 ppb also corresponds to the lowest exposure concentration at which pulmonary inflammation has been reported” (Frey, 2014, p. 7).

2. The controlled human exposure studies reporting these respiratory effects were conducted in healthy adults, while at-risk groups (e.g., children, people with asthma) could experience larger and/or more serious effects. In their advice to the Administrator, CASAC concurred with this conclusion (Frey, 2014, p. 5).
3. These respiratory effects are coherent with the serious health outcomes that have been reported in epidemiologic studies (e.g., respiratory-related hospital admissions, emergency department visits, and mortality).

Given the above considerations, our conclusions regarding the adequacy of the current primary O₃ standard place a large amount of weight on the results of controlled human exposure studies conducted at 60 and 72 ppb, and on HREA analyses based on information from controlled human exposure studies (i.e., exposures of concern to O₃ concentrations at or above 60, 70, and 80 ppb and O₃-induced FEV₁ decrements \geq 10%, 15%, and 20%).

Recent epidemiologic studies also provide support, beyond that available in the last review, for associations between short-term O₃ exposures and a wide range of adverse respiratory outcomes (including respiratory-related hospital admissions, emergency department visits, and mortality) and with total mortality. Associations with morbidity and mortality are stronger during the warm or summer months, and remain robust after adjustment for co-pollutants. Many epidemiologic studies of morbidity effects and mortality were conducted in locations that did not meet the current standard. However, in one U.S. single-city study associations with respiratory morbidity were reported in a location that would likely have met the current O₃ standard over the entire study period, suggesting that health effect associations persist in locations meeting the current standard. In addition, associations with respiratory morbidity or mortality were reported in several Canadian multicity studies, and in cut point analyses included in a U.S. multicity study, when the majority of study locations would likely have met the current O₃ standard. While there is additional uncertainty in interpreting the relationship between air quality meeting the current standard and health effects in these multicity studies (i.e., compared to single-city studies), they provide supporting evidence for the occurrence of health effect associations in locations that meet the current standard. Even in some study locations where the current standard was likely not met, considering reported concentration-response functions in the context of available air quality data support the occurrence of O₃-health effect associations on the subsets of days with ambient O₃ concentrations below the level of the current standard. Taken together, these studies and associated air quality data support the occurrence of O₃-associated

hospital admissions, emergency department visits, and mortality at ambient concentrations that meet the current standard.

Beyond our consideration of the evidence, we also consider the results of the HREA exposure and risk analyses in reaching conclusions regarding the adequacy of the current primary O₃ standard. In doing so, we focus primarily on estimates of the occurrence of exposures of concern to O₃ concentrations at or above 60 and 70 ppb and lung function decrements \geq 10%, 15% and 20%. We place relatively less weight on epidemiologic-based risk estimates, noting 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 (U.S. EPA, 2014, section 9.6). Our 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 locations, the potential for exposure measurement errors, and uncertainty in the interpretation of the shape of concentration-response functions at lower O₃ concentrations. The HREA also concludes that lower confidence should be placed in the results of the assessment of respiratory mortality risks associated with long-term O₃ exposures, primarily because that analysis is based on only one study (even though that study is well-designed) and because of the uncertainty in that study about the existence and level of a potential threshold in the concentration-response function (U.S. EPA, 2014, section 9.6).

With regard to HREA estimates of exposures of concern we note the CASAC conclusion that 60 ppb is an appropriate exposure of concern for asthmatic children (Frey, 2014, p. 8). Exposure estimates from the HREA indicate that, if the 15 urban case study areas were to just meet the current O₃ standard, approximately 10 to 20% of children (on average over the years of analysis) in those areas, including asthmatic children, could experience one or more exposures of concern to O₃ concentrations of 60 ppb or above. In the case study areas evaluated, this corresponds to over 2 million children (including over 200,000 asthmatic children) experiencing approximately 4 million such exposures. Nationally, far more children would be expected to experience such exposures of concern. On average over the years evaluated in the HREA, approximately 3 to 8% of children are estimated to experience two or more exposures of concern to O₃ concentrations of 60 ppb or greater. For the worst-case years in the worst-case locations (i.e., years and locations with air quality patterns resulting in the largest exposure estimates), approximately 25% of children are estimated to experience one or more exposures of concern at or above 60 ppb, and about 14% are estimated to experience two or more such exposures. Although the current standard more effectively limits exposures of concern at or above higher O₃ concentrations (i.e., 70, 80 ppb), we note that in the worst-case year and location about 8% of children are estimated to experience one or more exposures of concern at or above 70 ppb and about 2% of children are estimated to experience two or more such exposures.

Though we focus on children in these analyses of O₃ exposures, we also recognize that exposures to 8-hour average O₃ concentrations at or above 60, 70, or 80 ppb could be of concern for adult populations as well. As discussed above, the patterns of exposure estimates over years and across cities are similar in adult asthmatics, older adults, and children, though smaller percentages of adult populations are estimated to experience exposures of concern. Thus, the results for children are one part of a broader range of at-risk populations that also includes asthmatic adults and older adults.

Consistent with estimates of exposures of concern, the HREA also estimates that under air quality conditions just meeting the current O₃ NAAQS, hundreds of thousands of asthmatic children would be expected to experience O₃-induced lung function decrements that are large enough to be potentially adverse in people with lung disease. On average over the years evaluated in the HREA, the current standard is estimated to allow about 14% to 19% of children in the 15 urban case study areas, including asthmatic children, to experience one or more O₃-induced lung function decrements $\geq 10\%$ (a decrement judged by CASAC to be a “scientifically-relevant surrogate for adverse health outcomes for people with asthma and lung disease” (Frey, 2014, p. 4)). This corresponds to about 300,000 asthmatic children. Nationally, far more children would be expected to experience such O₃-induced lung function decrements. Across the 15 urban areas, about 8% to 12% of children are estimated to experience two or more decrements $\geq 10\%$, on average over the analysis years. In the worst-case year and location, approximately 22% of children are estimated to experience one or more decrements $\geq 10\%$ and about 14% are estimated to experience two or more such decrements. As with exposures of concern, the current standard more effectively limits the larger O₃-induced lung function decrements evaluated (i.e., $\geq 15\%$, 20%). However, about 7% of children are estimated to experience one or more O₃-induced decrements $\geq 15\%$ in the worst-case city and year analyzed in the HREA, and about 4% are estimated to experience two or more such decrements. As discussed above, CASAC judged decrements $\geq 15\%$ to be an appropriate “surrogate for adverse health outcomes in active healthy adults” (Frey, 2014, p. 4).

As noted above, compared to the weight given to HREA estimates of exposures of concern and lung function risks, we place relatively less weight on epidemiologic-based risk estimates. For epidemiology-based risk estimates, we consider total risks (i.e., based on the full distributions of ambient O₃ concentrations) and risks associated with O₃ concentrations in the upper portions of ambient distributions. A focus on estimates of total risks places greater weight on the possibility that concentration-response relationships remain linear over the entire distributions of ambient O₃ concentrations. With regard to total risks, the HREA estimates thousands of O₃-associated hospital admissions, emergency department visits, and deaths per year for air quality conditions associated with just meeting the current standard in the 12 urban

case study areas evaluated. A focus on risks associated with O₃ concentrations in the upper portions of ambient distributions places greater weight on the uncertainty associated with the shapes of concentration-response curves for O₃ concentrations in the lower portions of ambient distributions (section 3.2.3.2). Based on area-wide O₃ concentrations from the upper portions of seasonal distributions, the current standard is estimated to allow hundreds to thousands of O₃-associated deaths per year in urban case study areas. As with the exposures of concern and lung function risks, this number would be much greater if risks were assessed across the entire U.S. population.

Although we note the HREA conclusions indicating somewhat less confidence in estimates of O₃-associated mortality and morbidity risks compared to estimates of exposures of concern and lung function risks, we conclude that the general magnitude of mortality and morbidity risk estimates suggests the potential for a substantial number of O₃-associated deaths and adverse respiratory events nationally when the current standard is met. This is the case even based on the risks associated with the upper ends of distributions of ambient O₃ concentrations, where experimental evidence indicates increasing support for the occurrence of adverse effects attributable to O₃ exposures.

In addition to the evidence and exposure/risk information discussed above, we also take note of the CASAC advice provided to the EPA Administrator on the proposed reconsideration of the 2008 decision establishing the current standard and the advice of CASAC in the current review. In commenting on the proposed reconsideration, the prior CASAC O₃ Panel recommended revision of the standard to one with a lower level based on the evidence and information in the record for the 2008 standard (Samet, 2011), which has been substantially strengthened in the current review. As discussed in more detail above, the current CASAC also “unanimously recommends that the Administrator revise the current primary ozone standard to protect public health” (Frey, 2014, p. 6).

In consideration of all of the above, staff reaches the conclusion that the available evidence and exposure and risk information clearly calls into question the adequacy of public health protection provided by the current primary standard. The evidence from controlled human exposure studies provides strong support for the occurrence of adverse respiratory effects following exposures to O₃ concentrations below the level of the current standard. Epidemiologic studies provide support for the occurrence of adverse respiratory effects and mortality under air quality conditions that would likely meet the current standard. In addition, based on the analyses in the HREA, we conclude that the exposures and risks projected to remain upon meeting the current standard are indicative of risks that can reasonably be judged to be important from a public health perspective. Thus, staff concludes that the evidence and information provides strong support for giving consideration to revising the current primary standard in order to

provide increased public health protection against an array of adverse health effects that range from decreased lung function and respiratory symptoms to more serious indicators of morbidity (e.g., including emergency department visits and hospital admissions), and mortality. In consideration of all of the above, staff draws the conclusion that it is appropriate for the Administrator to consider revision of the current primary O₃ standard to provide increased public health protection.

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4 CONSIDERATION OF ALTERNATIVE PRIMARY STANDARDS

Having reached the conclusion that the currently available scientific evidence and exposure/risk information calls into question the adequacy of the current O₃ standard, we next consider the following overarching question:

- **What is the range of potential alternative standards that are supported by the currently available scientific evidence and exposure/risk information, as reflected in the ISA and HREA respectively?**

To address this overarching question, in the sections below we evaluate a series of more specific questions related to the major elements of the NAAQS: indicator (section 4.1), averaging time (section 4.2), form (section 4.3), and level (section 4.4). In addressing these questions, we consider the currently available scientific evidence and exposure/risk information, including the evidence and information available at the time of the last review and that newly available in the current review, as assessed in the ISA and the HREA. In so doing, we note that the final decision by the Administrator in this review will consider these elements collectively in evaluating the health protection afforded by the primary standard.¹

4.1 INDICATOR

In the last review, EPA focused on O₃ as the most appropriate indicator for a standard meant to provide protection against ambient photochemical oxidants. In this review, while the complex atmospheric chemistry in which O₃ plays a key role has been highlighted, no alternatives to O₃ have been advanced as being a more appropriate indicator for ambient photochemical oxidants. More specifically, the ISA noted that O₃ is the only photochemical oxidant (other than NO₂) that is routinely monitored and for which a comprehensive database exists (ISA section 3.6). Data for other photochemical oxidants (e.g., PAN, H₂O₂, etc.) typically have been obtained only as part of special field studies. Consequently, no data on nationwide patterns of occurrence are available for these other oxidants; nor are extensive data available on the relationships of concentrations and patterns of these oxidants to those of O₃ (U.S. EPA, 2013, section 3.6). In its review of the second draft PA, CASAC concurred, stating “The indicator of ozone is appropriate based on its causal or likely causal associations with multiple adverse health outcomes and its representation of a class of pollutants known as photochemical oxidants” (Frey, 2014, p. ii).

¹We also take note of the 1997 review (discussed in section 1.3.1.2.3), in which O₃ background concentrations were an additional consideration in EPA’s selection of a standard from among a range of scientifically acceptable alternatives. Background O₃ is discussed in more detail in chapter 2 of this PA.

We further note that meeting an O₃ standard can be expected to provide some degree of protection against potential health effects that may be independently associated with other photochemical oxidants, even though such effects are not discernible from currently available studies indexed by O₃ alone. That is, since the precursor emissions that lead to the formation of O₃ generally also lead to the formation of other photochemical oxidants, measures leading to reductions in population exposures to O₃ can generally be expected to lead to reductions in population exposures to other photochemical oxidants. Taken together, we conclude that O₃ remains the most appropriate indicator for a standard meant to provide protection against photochemical oxidants.²

4.2 AVERAGING TIME

The EPA established the current 8-hour averaging time³ for the primary O₃ 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 6 to 8 hour O₃ concentrations and adverse respiratory effects (62 FR 38861). It was also noted that a standard with a max 8-hour averaging time is likely to provide substantial protection against respiratory effects associated with 1-hour peak O₃ concentrations. Similar conclusions were reached in the last O₃ NAAQS review and thus, the 8-hour averaging time was retained in 2008.

In the current review, we first consider the following question related to averaging time:

- **To what extent does the available evidence continue to support the appropriateness of a standard with an 8-hour averaging time?**

In reaching conclusions related to this question, staff considers causality judgments from the ISA, as well as results from the specific controlled human exposure and epidemiologic studies that informed those judgments. These considerations are described below in more detail.

As an initial consideration with respect to the most appropriate averaging time for the O₃ NAAQS, we note that the strongest evidence for O₃-associated health effects is for respiratory effects following short-term exposures. More specifically, the ISA concludes that evidence relating short-term O₃ exposures to respiratory effects is “sufficient to infer a causal relationship.” The ISA also judges that the evidence for short-term exposures to O₃ indicates “likely to be” causal relationships with both cardiovascular effects and mortality (U.S. EPA, 2013, section 2.5.2). Therefore, as in past reviews, the strength of the available scientific

²The D.C. Circuit upheld the use of O₃ as the indicator for photochemical oxidants based on these same considerations. *American Petroleum Inst. v. Costle*, 665 F. 2d 1176, 1186 (D.C. Cir. 1981).

³This 8-hour averaging time reflects daily max 8-hour average O₃ concentrations.

evidence provides strong support for a standard that protects the public health against short-term exposures to O₃.

In first considering the level of support available for specific short-term averaging times, we note the evidence available from controlled human exposure studies. As discussed in more detail in chapter 3 of this PA, substantial health effects evidence from controlled human exposure studies demonstrates that a wide range of respiratory effects (e.g., pulmonary function decrements, increases in respiratory symptoms, lung inflammation, lung permeability, decreased lung host defense, and airway hyperresponsiveness) occur in healthy adults following 6.6 hour exposures to O₃ (EPA 2013, section 6.2.1.1). Compared to shorter exposure durations (e.g., 1-hour), studies evaluating 6.6 hour exposures in healthy adults have reported respiratory effects at lower O₃ exposure concentrations and at more moderate levels of exertion.

We also note the strength of evidence from epidemiologic studies that have evaluated a wide variety of populations (e.g., including at-risk lifestages and populations, such as children and people with asthma, respectively). A number of different averaging times are used in O₃ epidemiologic studies, with the most common being the max 1-hour concentration within a 24-hour period (1-hour max), the max 8-hour average concentration within a 24-hour period (8-hour max), and the 24-hour average. These studies are discussed in chapter 3 of this PA, and are assessed in detail in chapter 6 of the ISA (U.S. EPA, 2013). Limited 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, though the ISA notes some evidence for “smaller O₃ risk estimates when using a 24-hour average exposure metric” (EPA 2013, section 2.5.4.2; p. 2-31). For single- and multi-day average O₃ concentrations, lung function decrements were associated with 1-hour max, 8-hour max, and 24-hour average ambient O₃ concentrations, with no strong difference in the consistency or magnitude of association among the averaging times (EPA 2013, p. 6-71). Similarly, in studies of short-term exposure to O₃ 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 (EPA 2013, p. 6-253). Thus, the epidemiologic evidence alone does not provide a strong basis for distinguishing between the appropriateness of 1-hour, 8-hour, and 24-hour averaging times.

Considering the health information discussed above, we conclude that an 8-hour averaging time remains appropriate for addressing health effects associated with short-term exposures to ambient O₃. An 8-hour averaging time is similar to the exposure periods evaluated in controlled human exposure studies, including recent studies that provide evidence for respiratory effects following exposures to O₃ concentrations below the level of the current standard. In addition, epidemiologic studies provide evidence for health effect associations with

8-hour O₃ concentrations, as well as with 1-hour and 24-hour concentrations. As in previous reviews, we note that a standard with an 8-hour averaging time (combined with an appropriate standard form and level) would also be expected to provide substantial protection against health effects attributable to 1-hour and 24-hour exposures (e.g., 62 FR 38861, July 18, 1997). In its review of the second draft PA, CASAC concurred stating that “the current 8-hour averaging time is justified by the combined evidence from epidemiologic and clinical studies” (Frey, 2014, p. 6).

The ISA also concludes that the evidence for long-term O₃ exposures indicates that there is “likely to be a causal relationship” with respiratory effects (US EPA, 2013, chapter 7). Thus, in this review we also consider the extent to which currently available evidence and exposure/risk information suggests that a standard with an 8-hour averaging time can provide protection against respiratory effects associated with longer term exposures to ambient O₃. In doing so, staff considers the following question:

- **To what extent does the available evidence and exposure/risk information indicate that a standard with the current 8-hour averaging time could provide protection against long-term exposures to ambient O₃?**

In considering this issue in the last review of the O₃ NAAQS, staff noted that “because long-term air quality patterns would be improved in areas coming into attainment with an 8-hr standard, the potential risk of health effects associated with long-term exposures would be reduced in any area meeting an 8-hr standard” (U.S. EPA, 2007, p. 6-57).

In the current review, we further evaluate this issue, with a focus on the “long-term” O₃ metrics reported to be associated with mortality or morbidity in recent epidemiologic studies. As discussed in section 3.1.3, much of the recent evidence for such associations is based on studies that defined long-term O₃ in terms of seasonal averages of daily max concentrations (e.g., seasonal averages of 1-hour or 8-hour daily max concentrations).

As an initial consideration, we note the risk results from the HREA for respiratory mortality associated with long-term O₃ concentrations. As discussed in section 3.2.3.2, HREA analyses indicate that as air quality is adjusted to just meet the current 8-hour standard, most urban case study areas are estimated to experience reductions in respiratory mortality associated with long-term O₃ concentrations based on the seasonal averages of 1-hour daily max O₃ concentrations evaluated in the study by Jerrett et al. (2009) (U.S. EPA, 2014, chapter 7). As air quality is adjusted to meet lower potential alternative standard levels, for standards based on 3-year averages of the annual fourth-highest daily max 8-hour O₃ concentrations, respiratory mortality risks are estimated to be reduced further in urban case study areas (section 4.4.2.3, below). This analysis indicates that an O₃ standard with an 8-hour averaging time, when coupled with an appropriate form and level, can reduce respiratory mortality reported to be associated with “long-term” O₃ concentrations.

In further considering the study by Jerrett et al. (2009), we compare long-term O₃ concentrations following air quality adjustment in urban case study areas (i.e., adjusted to meet the current and potential alternative 8-hour standards) to the concentrations present in study cities that provided the basis for the positive and statistically significant association with respiratory mortality. As indicated below (Table 4-3), this comparison suggests that a standard with an 8-hour averaging time can decrease seasonal averages of 1-hour daily max O₃ concentrations, and can maintain those O₃ concentrations below the seasonal average where we have the most confidence in the reported concentration-response relationship with respiratory mortality (see section 4.4.1 for further discussion).

The HREA also conducted analyses evaluating the impacts of reducing regional NO_x emissions on the seasonal averages of 8-hour daily max O₃ concentrations.⁴ Seasonal averages of 8-hour daily max O₃ concentrations reflect long-term metrics that have been reported to be associated with respiratory morbidity effects in several recent O₃ epidemiologic studies (e.g., Islam et al., 2008; Lin et al., 2008; Salam et al., 2009). The HREA analyses indicate that the large majority of the U.S. population lives in locations where reducing NO_x emissions would be expected to result in decreases in seasonal averages of daily max 8-hour ambient O₃ concentrations (U.S. EPA, 2014, section 8.2.3.2). Thus, consistent with the respiratory mortality risk estimates noted above, this analysis suggests that reductions in O₃ precursor emissions in order to meet a standard with an 8-hour averaging time would also be expected to reduce the long-term O₃ concentrations that have been reported in recent epidemiologic studies to be associated with respiratory morbidity.

Taken together, we conclude 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 long-term O₃ concentrations that have been reported to be associated with respiratory morbidity and mortality. In its review of the second draft PA, CASAC concurred, stating that “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” (Frey, 2014, p. 6). This issue is considered further, within the context of specific potential alternative standard levels, in section 4.4 below.

4.3 FORM

The “form” of a standard defines the air quality statistic that is to be compared to the level of the standard in determining whether an area attains the standard. The foremost consideration in selecting a form for potential alternative primary standards is the adequacy of

⁴Analyses are based on regional NO_x reductions, which are effective in bringing down peak ambient O₃ concentrations, but can have variable impacts on seasonal mean concentrations.

the public health protection provided by the combination of the form and the other elements of the standard. As such, in reaching staff conclusions regarding the appropriate form(s) to consider for a potential alternative primary O₃ standard, we consider the following question:

- **To what extent do the available evidence and/or information continue to support the appropriateness of a standard with a form defined by the 3-year average of annual 4th-highest 8-hour daily max O₃ concentrations?**

The EPA established the current form of the primary O₃ NAAQS in 1997 (62 FR 38856). Prior to that time, the standard had a “1-expected-exceedance” form.⁵ An advantage of the current concentration-based form recognized in the 1997 review is that such a form better reflects the continuum of health effects associated with increasing ambient O₃ concentrations. Unlike an expected exceedance form, a concentration-based form gives proportionally more weight to years when 8-hour O₃ concentrations are well above the level of the standard than to years when 8-hour O₃ concentrations are just above the level of the standard. It was judged appropriate to give more weight to higher O₃ concentrations, given that available health evidence indicated a continuum of effects associated with exposures to varying concentrations of O₃, and given that the extent to which public health is affected by exposure to ambient O₃ is related to the actual magnitude of the O₃ concentration, not just whether the concentration is above a specified level.

During the 1997 review, EPA considered a range of alternative “concentration-based” forms, including the second-, third-, fourth- and fifth-highest daily max 8-hour concentrations in an O₃ season. The fourth-highest daily max was selected, recognizing that a less restrictive form (e.g., fifth highest) would allow a relatively large percentage of sites to experience O₃ peaks well above the level of the standard, and would allow more days on which the level of the standard may be exceeded when attaining the standard (62 FR 38856). Consideration was also given to setting a standard with a form that would provide a margin of safety against possible but uncertain chronic effects, and would provide greater stability to ongoing control programs.⁶ A more restrictive form was not selected, recognizing that the differences in the degree of protection afforded by the alternatives were not well enough understood to use any such differences as a basis for choosing the most restrictive forms (62 FR 38856).

In the 2008 review, EPA additionally considered the potential value of a percentile-based form. In doing so, EPA recognized that such a statistic is useful for comparing datasets of

⁵For a standard with a 1-expected-exceedance form to be met at an air quality monitoring site, the fourth-highest air quality value in 3 years, given adjustments for missing data, must be less than or equal to the level of the standard.

⁶ See *American Trucking Assn’s v. EPA*, 283 F. 3d 355, 374-75 (D.C. Cir. 2002) (less stable implementation programs may be less effective, and therefore EPA can consider programmatic stability in determining the form of a NAAQS).

varying length because it samples approximately the same place in the distribution of air quality values, whether the dataset is several months or several years long. However, EPA concluded that a percentile-based statistic would not be effective in ensuring the same degree of public health protection across the country. Specifically, a percentile-based form would allow more days with higher air quality values in locations with longer O₃ seasons relative to places with shorter O₃ seasons. Thus, in the 2008 review EPA concluded that a form based on the nth-highest max O₃ concentration would more effectively ensure that people who live in areas with different length O₃ seasons receive the same degree of public health protection.

Based on analyses for forms specified in terms of an nth-highest concentration (n ranged from 3 to 5), advice from CASAC, and public comment,⁷ the Administrator concluded that a 4th-highest daily max should be retained (73 FR 16465). In reaching this decision, the Administrator recognized that “there is not a clear health-based threshold for selecting a particular nth-highest daily maximum form of the standard” and that “the adequacy of the public health protection provided by the combination of the level and form is a foremost consideration” (73 FR 16475). Based on this, the Administrator judged that the existing form (4th-highest daily maximum 8-hour average concentration) should be retained, recognizing the increase in public health protection provided by combining this form with a lower standard level (i.e., 75 ppb).

The Administrator also recognized that it is important to have a form that provides stability with regard to implementation of the standard. In the case of O₃, for example, he noted the importance of a form insulated from the impacts of the meteorological events that are conducive to O₃ formation. Such events could have the effect of reducing public health protection, to the extent they result in frequent shifts in and out of attainment due to meteorological conditions. The Administrator noted that such frequent shifting could disrupt an area’s ongoing implementation plans and associated control programs (73 FR 16474). In his final decision, the Administrator judged that a “4th high form provides a stable target for implementing programs to improve air quality” (73 FR 16475).

In the current review, we consider the extent to which newly available information provides support for consideration of alternative forms. In so doing, we take note of the conclusions of prior reviews summarized above. We recognize the value of an nth-high statistic over that of an expected exceedance or percentile-based form in the case of the O₃ standard, for

⁷In the 2008 review, one group of commenters expressed the view that the standard was not adequate and supported a more health-protective form (e.g., a second- or third-highest daily max form). Another group of commenters expressed the view that the standard was adequate and did not provide any views on alternative forms that would be appropriate should the Administrator consider revisions to the standard. The Administrator considered the protection afforded by the combination of level and form in revising the standard in 2008 to 75 ppb, as a 3-year average of the annual fourth-highest daily max 8-hour concentrations (73 FR 16475).

the reasons summarized above. We additionally take note of the importance of stability in implementation to achieving the level of protection specified by the NAAQS. Specifically, we note that to the extent that areas engaged in implementing the O₃ NAAQS frequently shift from meeting to violating the standard, it is possible that ongoing implementation plans and associated control programs could be disrupted, thereby reducing public health protection.

In light of this, while giving foremost consideration to the adequacy of public health protection provided by the combination of all elements of the standard, including the form, we consider particularly findings from prior reviews with regard to the use of the nth-high metric. As noted above, the 4th-highest daily max was selected 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 implementation programs. The currently available evidence and information does not call into question these conclusions from previous reviews. Moreover, in its review of the second draft PA, CASAC concurred that the O₃ standard should be based on the fourth highest, daily maximum 8-hour average value (averaged over three 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, 2014, p. 6). Thus a standard with the current 4th high form, coupled with a level lower than 75 ppb as discussed below, would be expected to increase public health protection relative to the current standard while continuing to provide stability for implementation programs. Therefore, we conclude that it would be appropriate to consider retaining the current 4th-highest daily max form for an O₃ standard with an 8-hour averaging time and a revised level, as discussed below.

4.4 LEVEL

In considering potential alternative standards levels to provide greater protection than that afforded by the current standard against O₃-related adverse health effects, we address the following overarching question.

- **For an O₃ standard defined in terms of the current indicator, averaging time, and form, what alternative levels are appropriate to consider in order to provide adequate public health protection against short- and long- term exposures to O₃ in ambient air?**

In considering this question, we take into account the experimental and epidemiologic evidence as presented in the ISA, as well as the uncertainties and limitations associated with this evidence (section 4.4.1). In addition, we consider the quantitative estimates of exposure and risk provided by the HREA, as well as the uncertainties and limitations associated with these risk estimates (section 4.4.2).

4.4.1 Evidence-based Considerations

In this section, we consider the available evidence from controlled human exposure and epidemiologic studies, including the uncertainties and limitations associated with that evidence, within the context of potential alternative standard levels. We consider both the exposure concentrations at which controlled human exposure studies provide evidence for health effects, and the ambient O₃ concentrations present in locations where epidemiologic studies have reported health effect associations (see also section 3.1).

Controlled human exposure studies and epidemiologic panel studies

We consider the following question related to controlled human exposure studies and panel studies:

- **To what extent does the available evidence from controlled human exposure studies and panel studies provide support for consideration of potential alternative standard levels lower than 75 ppb?**

To inform our conclusions regarding this question, we consider the lowest O₃ concentrations at which various effects have been evaluated and statistically significant effects reported. We also consider the potential for reported effects to be adverse, including in at-risk populations.

As discussed in section 3.1.2.1, data from controlled human exposure studies show that group mean O₃-induced lung function decrements in healthy adults exhibit a smooth dose-response relationship without evidence of a threshold from 40 to 120 ppb O₃ (US EPA, 2013, Figure 6-1). The lowest O₃ exposure concentration for which statistically significant decrements have been reported is 60 ppb (Brown, 2008; Kim et al., 2011). The ISA concludes that mean FEV₁ is clearly decreased by 6.6-hour exposures to O₃ concentrations of 60 ppb and higher in young, healthy adults during moderate exertion (US EPA, 2013, p. 6-9). As discussed in section 3.1.3, such a decrease in mean lung function meets the ATS criteria for an adverse response given that a downward shift in the distribution of FEV₁ would result in diminished reserve function, and therefore would increase risk from further environmental insult. In addition, based on data from studies by Kim et al. (2011), Schelegle et al. (2009), Adams (2006), and Adams (1998), the ISA notes that following exposures to 60 ppb O₃ 10% of healthy adults experience FEV₁ decrements > 10% (U.S. EPA, 2013, page 6-19).⁸ A 10% decrement in FEV₁ is accepted

⁸As discussed in Chapter 3 of this PA (section 3.1.2.1), these estimates are consistent with the predictions of quantitative models developed by McDonnell et al. (2012) and Schelegle et al. (2012). The McDonnell model, as discussed in McDonnell et al. (2010), provides the basis for lung function risk estimates in the HREA (section 4.4.2.2, below). For the target of 60 ppb, Schelegle et al. (2009) reported an actual mean exposure concentration of 63 ppb.

by ATS as an abnormal response. Based on advice received from CASAC in this (Frey, 2014, p. 3) and previous reviews, such decrements could be adverse in people with lung disease (section 3.1.3). Moreover, as discussed in section 3.1.3 of this PA, repeated occurrences of moderate responses may be considered adverse since they could set the stage for more serious effects.

One recent controlled human exposure study has reported O₃-induced pulmonary inflammation (PMN increased in sputum from lower airways) following exposures of young, healthy adults to O₃ concentrations of 60 ppb (Kim et al., 2011), the lowest concentration at which inflammatory responses have been evaluated in human studies (see discussion in section 3.1.2.1). Induction of pulmonary inflammation is evidence that injury has occurred. The possibility of chronic effects due to repeated inflammatory events has been evaluated in animal studies. Repeated events of acute inflammation can have several potentially adverse outcomes including: 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 (U.S. EPA, 2013, Section 6.2.3). Thus, lung injury and the resulting inflammation, particularly if experienced repeatedly, provide a mechanism by which O₃ may cause other more serious respiratory effects (e.g., asthma exacerbations) and possibly extrapulmonary effects.

With respect to respiratory symptoms, a recent study by Schelegle et al. (2009) reported a statistically significant increase in respiratory symptoms in young, healthy adults following 6.6 hour exposures to an average O₃ concentration of 70 ppb.⁹ This study also reported a statistically significant decrease in FEV₁ following such exposures. As discussed in section 3.1.3, the occurrence of both lung function decrements and respiratory symptoms meets criteria established by the ATS defining an “adverse” respiratory response. Although some studies have reported that respiratory symptoms develop during exposures at 60 ppb, the increases in symptoms in these studies have not reached statistical significance by the end of the 6.6 hour exposures (Adams 2006; Schelegle et al., 2009).¹⁰

Based on the results discussed above and in section 3.1.2.1, we conclude that controlled human exposure studies provide evidence of potentially adverse lung function decrements and airway inflammation in healthy adults following exposures to 60 ppb O₃, and evidence of

⁹ For the target of 70 ppb, Schelegle et al. (2009) reported an actual mean exposure concentration of 72 ppb.

¹⁰ Adams (2006) reported an increase in respiratory symptoms in healthy adults during a 6.6 hour exposure protocol with an average O₃ exposure concentration of 60 ppb. This increase was significantly different from initial respiratory symptoms, but not from the filtered air control day. For the target of 60 ppb, Schelegle et al. (2009) reported an actual mean exposure concentration of 63 ppb and did not observe a statistically significant increase in respiratory symptoms.

respiratory symptoms combined with lung function decrements (an “adverse” response based on ATS criteria) following exposures to 70 ppb. In reaching these conclusions, we recognize that most studies have not evaluated exposure concentrations below 60 ppb, and that 60 ppb does not necessarily reflect an exposure concentration below which effects no longer occur. Specifically, given the occurrence of airway inflammation in healthy adults following exposures to 60 ppb and higher, it may be reasonable to expect that inflammation would also occur following exposures to O₃ concentrations somewhat below 60 ppb. Although some studies show that respiratory symptoms develop during exposures at 60 ppb, they have not reached statistical significance by the end of the 6.6 hour exposures (Adams 2006; Schelegle et al. 2009). Thus, respiratory symptoms combined with lung function decrements are likely to occur to some degree in healthy adults with 6.6-hour exposures to concentrations below 70 ppb, and are more likely to occur with 8-hour exposures to 70 ppb and below. Further, we note that these controlled human exposure studies were conducted in healthy adults and that people with asthma, including asthmatic children, are likely to be more sensitive to O₃-induced respiratory effects. Therefore, these exposure concentrations are more likely to cause adverse respiratory effects in children and adults with asthma, and more generally in people with respiratory disease.

With regard to other O₃-induced effects, we note that airway hyperresponsiveness and impaired lung host defense capabilities have been reported in healthy adults engaged in moderate exertion following exposures to O₃ concentrations as low as 80 ppb, the lowest concentration evaluated for these effects.¹¹ As discussed in section 3.1.2.1, these physiological effects have been linked to aggravation of asthma and increased susceptibility to respiratory infection, potentially leading to increased medication use, increased school and work absences, increased visits to doctors’ offices and emergency departments, and increased hospital admissions. These are all indicators of adverse O₃-related morbidity effects, which are consistent with, and provide plausibility for, the adverse morbidity effects and mortality effects observed in epidemiologic studies.

In further considering effects following exposures to O₃ concentrations below 75 ppb, in section 3.1.4.1 we discuss panel studies highlighted in the ISA for the extent to which monitored ambient O₃ concentrations reflect exposure concentrations in their study populations (U.S. EPA, 2013, section 6.2.1.2). These panel studies used on-site monitoring to evaluate O₃-attributable lung function decrements in people engaged in outdoor recreation, exercise, or work. Table 3-2 includes O₃ panel studies that report analyses of O₃-attributable lung function decrements for O₃ concentrations at or below 75 ppb, and that measure O₃ concentrations with monitors located in the areas where study subjects were active (e.g., on site at summer camps or in locations where

¹¹There is no evidence that 80 ppb is a threshold for these effects (72 FR 37878, July 11, 2007).

exercise took place). Consistent with the results of controlled human exposure studies discussed above, these panel studies report associations with lung function decrements for subjects exposed to on-site monitored O₃ concentrations below 75 ppb. Associations in panel studies have been reported for a wider range of populations than has been evaluated in controlled human exposure studies, including children.

With regard to the question above, we conclude that the available controlled human exposure evidence and evidence from panel studies supports an upper end of the range of potential alternative standard levels for consideration no higher than 70 ppb. As just discussed, 6.6-hour exposures of healthy adults to 70 ppb O₃ result in lung function decrements and respiratory symptoms, a combination of effects that meet ATS criteria for an adverse response (as discussed in section 3.1.3).¹² In addition, while 70 ppb is below the 80 ppb concentration shown in 6.6-hour exposure studies to cause potentially adverse respiratory effects such as airway hyperresponsiveness and impaired host-defense capabilities, these effects have not been evaluated at exposure concentrations below 80 ppb and there is no reason to believe that 80 ppb represents a threshold for such effects. As discussed in section 3.1.2.1 of this PA, the physiological effects reported in controlled human exposure studies down to 60 ppb O₃ have been linked to aggravation of asthma and increased susceptibility to respiratory infection, potentially leading to increased medication use, increased school and work absences, increased visits to doctors' offices and emergency departments, and increased hospital admissions.

Based on the above considerations, we also conclude that the evidence from controlled human exposure studies and panel studies supports considering alternative O₃ standard levels at least as low as 60 ppb. Potentially adverse lung function decrements and pulmonary inflammation have been demonstrated to occur in healthy adults at 60 ppb, with little evidence for potentially adverse effects following exposures to O₃ concentrations below 60 ppb. Thus, 60 ppb is a short-term exposure concentration that may be reasonably concluded to elicit adverse effects in at-risk groups. Pulmonary inflammation, particularly if experienced repeatedly, provides a mechanism by which O₃ may cause other more serious respiratory morbidity effects (e.g., asthma exacerbations) and possibly extrapulmonary effects.

Epidemiologic evidence

We also consider what the information from epidemiologic studies indicates with regard to potential alternative standard levels appropriate for consideration. Based on the information in

¹² Based on the Schelegle et al. (2009) study, CASAC observed that, "adverse health effects in young healthy adults occur with exposures to 72 ppb of ozone for 6.6 hours" and that "It is the judgment of CASAC that if subjects had been exposed to ozone using the 8-hour averaging period used in the standard, adverse effects could have occurred at [a] 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, 2014, p. 5).

section 3.1.4.2 of this PA (see Table 3-3), we first note that several epidemiologic studies have reported positive and statistically significant associations with hospital admissions, emergency department visits, and/or mortality in study areas where ambient O₃ concentrations would have met the current standard (i.e., with its level of 75 ppb). This includes Canadian multicity studies in which the majority of study cities would have met the current standard over entire study periods (Cakmak et al., 2006; Dales et al., 2006; Katsouyanni et al., 2009; Stieb et al., 2009), and a U.S. single-city study conducted in a location likely to have met the current standard over the entire study period (Mar and Koenig, 2009).

In further evaluating these studies, and building upon our conclusions based on controlled human exposures studies, as discussed above, we consider the following question related to the epidemiologic evidence:

- **To what extent have U.S. and Canadian epidemiologic studies reported associations with mortality or morbidity in locations likely to have met potential alternative O₃ standards with levels from 70 to 60 ppb?**

Our focus in addressing this question is on what epidemiologic studies convey regarding the extent to which O₃-associated health effects may be occurring (i.e., as indicated by associations) under air quality conditions allowed by potential alternative standards with levels of 70, 65, and 60 ppb (Table 4-1).¹³

¹³See *ATA III*, 283 F.3d at 370 (EPA justified in revising NAAQS when health effect associations are observed at levels allowed by the NAAQS).

Table 4-1 Numbers of epidemiologic study locations likely to have met potential alternative standards with levels of 70, 65, and 60 ppb

Study	Result	Cities	Number of study cities meeting potential alternative standards during entire study period		
			70 ppb	65 ppb	60 ppb
Cakmak et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	10 Canadian cities	7	6	2
Dales et al. (2006)	Positive and statistically significant association with respiratory hospital admissions	11 Canadian cities	5	4	0
Katsouyanni et al. (2009)	Positive and statistically significant associations with respiratory hospital admissions	12 Canadian cities	9	9	5
Katsouyanni et al. (2009)	Positive and statistically significant associations with total and cardiovascular mortality	12 Canadian cities	7	5	1
Mar and Koenig (2009)	Positive and statistically significant associations with asthma emergency department visits	Single city: Seattle	0	0	0
Stieb et al. (2009)	Positive and statistically significant association with respiratory emergency department visits	7 Canadian cities	5	4	3

As discussed in section 3.1.4.2, the single-city study by Mar and Koenig reported associations with respiratory emergency department visits in a location that would have met the current standard over the entire study period. In contrast, over at least part of the study period this area would have violated alternative O₃ standards with levels of 70 ppb or below. Thus, while this study indicates that the current standard would allow the reported associations with respiratory emergency department visits, it does not provide information on the extent to which those health effect associations would be present if ambient O₃ concentrations were reduced to meet a revised standard with a level at or below 70 ppb.

With regard to the multicity studies included in Table 4-1, none were conducted in study locations that all would have met an O₃ standard with a level at or below 70 ppb. However, for the studies by Cakmak et al. (2006), Katsouyanni et al. (2009), and Stieb et al. (2009), the majority of study locations would likely have met a standard with a level of either 70 or 65 ppb (Cakmak et al., 2006; Katsouyanni et al., 2009; Stieb et al., 2009). Thus the majority of the distributions of ambient O₃ concentrations that provided the basis for positive and statistically significant associations with mortality or morbidity in these studies would likely be allowed

under alternative standards with levels of 70 or 65 ppb, though not 60 ppb. However, our interpretation of these results is complicated by uncertainties in the extent to which multicity effect estimates can be attributed to ambient O₃ in the majority of locations, which would have met alternative standards, versus O₃ in the smaller number of locations that would have violated those alternatives.

As with our consideration of the current standard (section 3.1.4.2), we next consider the extent to which epidemiologic studies have characterized O₃ health effect associations, including confidence in those associations, for various portions of distributions of ambient O₃ concentrations. In considering such analyses within the context of potential alternative standards, we focus on the extent to which epidemiologic studies report health effect associations for air quality distributions restricted to ambient pollutant concentrations below one or more predetermined cut-points. As discussed in section 3.1.4.2, such “cut-point” analyses can provide information on the magnitude and statistical precision of effect estimates for defined distributions of ambient concentrations, which may in some cases include distributions that would be allowed by potential alternative standards. Specifically, we consider the following question:

- **To what extent do cut-point analyses from epidemiologic studies report health effect associations at ambient O₃ concentrations that are likely to be allowed by potential alternative standards with levels from 70 to 60 ppb?**

As with our consideration of the current standard in section 3.1.4.2 of this PA, we evaluate the cut-point analyses presented in the U.S. multicity study by Bell et al. (2006). These cut-point analyses can provide insights into the magnitude and statistical precision of health effect associations for different portions of the distribution of ambient concentrations, including insights into the ambient concentrations below which uncertainty in reported associations becomes notably greater. Our analysis of air quality data associated with the cut-points evaluated by Bell et al., and uncertainties associated with that analysis, is described elsewhere in this document (section 3.1.4.2). In this section, we consider what these cut-point analyses indicate with regard to the potential for health effect associations to extend to ambient O₃ concentrations likely to be allowed by a revised O₃ NAAQS with a level below 75 ppb.

We particularly focus on the lowest cut-point for which the association between O₃ and mortality was reported to be statistically significant (i.e., 30 ppb, as discussed in section 3.1.4.2). Based on the O₃ air quality concentrations that met the criteria for inclusion in the 30 ppb cut-point analysis, 84% of study areas had 3-year averages of annual 4th highest 8-hour daily max O₃ concentrations at or below 70 ppb over the entire study period (Table 4-2). In addition, 64% of study areas had 3-year averages of annual 4th highest 8-hour daily max O₃ concentrations at or

below 65 ppb (Table 4-2). In contrast, the majority of study areas had 4th highest concentrations above 60 ppb.

Consistent with our interpretation of multicity effect estimates discussed above, these results suggest that the majority of the air quality distributions included in the 30 ppb O₃ cut point would have been allowed by a standard with a level of 70 or 65 ppb. Thus the majority of the distributions of ambient O₃ concentrations that provided the basis for a positive and statistically significant association with mortality would be allowed by alternative standards with levels of 70 or 65 ppb, but not 60 ppb. However, as discussed below our interpretation of these cut point analyses is complicated by important uncertainties.

Table 4-2 Number of study cities with 3-year averages of 4th highest 8-hour daily max concentrations greater than 70, 65, or 60 ppb, for various cut-point analyses presented in Bell et al. (2006)

	Cut-point for 2-day moving average across monitors and cities (24-h avg) ¹⁴								
	25	30	35	40	45	50	55	60	All
Number (%) of Cities with 4th highest >70 (any 3-yr period; 1987-2000)	0 (0%)	16 (16%)	55 (56%)	82 (84%)	89 (91%)	92 (94%)	94 (96%)	95 (97%)	95 (97%)
Number (%) of Cities with 4th highest >65 (any 3-yr period; 1987-2000)	3 (3%)	35 (36%)	77 (79%)	89 (91%)	94 (96%)	95 (97%)	95 (97%)	95 (97%)	95 (97%)
Number (%) of Cities with 4th highest >60 (any 3-yr period; 1987-2000)	16 (16%)	61 (62%)	86 (88%)	94 (96%)	95 (97%)	96 (8%)	96 (8%)	96 (8%)	96 (8%)

In further considering the implications of Tables 4-1 and 4-2 for potential alternative standard levels, we also note the important uncertainties described in section 3.1.4 of this document. General uncertainties include the geographic heterogeneity in effect estimates, which could obscure presence of potential thresholds in multicity studies; uncertainty in the extent to which multicity effect estimates can be attributed to ambient O₃ in the majority of locations, which would have met alternative standards with levels of 70 or 65 ppb, versus O₃ in the smaller number of locations that would have violated those alternatives; and uncertainty in the extent to which the relatively low ambient O₃ concentrations present in some study areas caused or

¹⁴Cut point analyses presented in the study by Bell et al. (2006) are described in more detail in sections 3.1.2.3 and 3.1.4.2 of this document.

contributed to reported effects. Additional uncertainties specific to our analysis of the cut points presented by Bell et al. (2006) include the appropriateness of identifying 4th highest concentrations from air quality subsets, rather than the entire air quality distributions that existed in study locations, and uncertainty associated with the air quality data used to re-create the cut-point analyses from the published study. With regard to this second uncertainty, as described in more detail in section 3.1.4.2 of this document, our re-creation of the cut points was based on air quality data available in AQS, combined with the published descriptions of cut point criteria and study area definitions. In doing so, we did not recreate the trimmed means used by Bell. Therefore, an important uncertainty in this approach is the extent to which we were able to appropriately re-create the cut-point analyses in the published study.

Overall, our analyses of air quality in U.S. and Canadian epidemiologic study locations indicate that (1) single-city studies have not been conducted in locations that would have met alternative O₃ standards with levels of 70 ppb or below and that (2) multicity epidemiologic studies report positive and statistically significant associations with mortality and morbidity based largely on distributions of ambient O₃ that would have been allowed by alternative standards with levels of 70 or 65 ppb, but not 60 ppb. While important uncertainties, mentioned above, complicate our interpretation of the multicity studies, at a minimum these results suggest that an alternative standard level of 60 ppb would not allow the distributions of ambient O₃ concentrations present in the majority of study locations that provided the basis for statistically significant health effect associations. While the potential implications for alternative standard levels of 70 and 65 ppb are less clear, given the important uncertainties in these analyses, the results suggest that positive and statistically significant associations with mortality or morbidity in some studies were largely influenced by air quality distributions that would be allowed under alternative standards with such levels.

We next consider the extent to which epidemiologic studies employing longer-term ambient O₃ concentration metrics can inform our consideration of potential alternative standard levels. In doing so, we consider the following question:

- **To what extent does the available evidence indicate that an O₃ standard with a level from 70 to 60 ppb, combined with the current 8-hour averaging time and 4th high form, could provide protection from long-term exposures to ambient O₃ concentrations for which there is evidence of health effects?**

We first note that, as discussed in section 3.1.4.3 of this PA, virtually all of the study cities that provided the basis for the positive and statistically significant association between long-term O₃ and respiratory mortality (Jerrett et al., 2009) would have violated the current standard, and therefore potential alternative standards with lower levels. Thus, as with our

consideration of the current standard in section 3.1.4.3, while the study by Jerrett et al. (2009) contributes to our understanding of health effects associated with ambient O₃ (summarized in section 3.1.2), it is less informative regarding the extent to which those health effects may be occurring under air quality conditions that would meet potential alternative standards.

To further evaluate this issue, we use the adjusted air quality in urban case study areas, as described in the HREA, to consider the extent to which just meeting alternative O₃ standards with levels of 70, 65, and 60 ppb could maintain long-term O₃ concentrations below those in the cities that provided the basis for the positive and statistically significant association with respiratory mortality reported by Jerrett et al. (2009).¹⁵ Upon adjustment of air quality in U.S. urban case study areas to meet the current and potential alternative 8-hour standards, seasonal average 1-hour daily max concentrations were calculated and compared to the concentrations in study cities.

As discussed in section 3.1.4.3, Jerrett et al. (2009) reported that when seasonal averages of 1-hour daily max O₃ concentrations¹⁶ ranged from 33 to 104 ppb, there was no statistical deviation from a linear concentration-response relationship between O₃ and respiratory mortality across 96 U.S. cities (U.S. EPA, 2013, section 7.7). However, as discussed in section 3.1.4.3, the study suggests notably decreased confidence in the reported linear concentration-response function for “long-term” O₃ concentrations in the first quartile (i.e., at or below about 53 ppb), given the widening in confidence intervals for lower concentrations (based on visual inspection of Figure 3-6 in section 3.1.4.3); the fact that most study cities contributing to the linear function had O₃ concentrations in the highest three quartiles, accounting for approximately 72% of the respiratory deaths in the cohort (based on Table 2 in the published study); and the limited evidence presented in the published study for a threshold at or near 56 ppb.¹⁷

Given the above, we note the extent to which long-term O₃ concentrations (i.e., seasonal average of 1-hour daily max) in urban case study areas are estimated to be at or below 53 ppb following air quality adjustment to meet potential alternative standards with levels of 70, 65, and 60 ppb. To the extent air quality adjustment to just meet potential alternative short-term standards results in long-term concentrations near or below 53 ppb, we have greater confidence

¹⁵Air quality in U.S. urban case study areas was adjusted to just meet the current 8-hour standard at 75 ppb, as well as potential potential alternative 8-hour standards at 70 ppb, 65 ppb, and 60 ppb, as described in the HREA (chapter 4). After a given adjustment, seasonal average 1-hour daily max concentrations were calculated.

¹⁶Jerrett et al. (2009) evaluated the April to September averages of 1-hour daily max O₃ concentrations across 96 U.S. metropolitan areas from 1977- 2000. In urban areas with multiple monitors, April to September 1-hour daily max concentrations from each individual monitor were averaged. This step was repeated for each year in the study period. Finally, each yearly averaged O₃ concentrations was then averaged again to yield the single averaged 1-hour daily max O₃ concentration depicted on the x-axis of Figure 3-6 below.

¹⁷The issue of potential thresholds based on the Jerrett study is discussed in more detail in section 3.2.3.2 of this PA.

in the degree to which those short-term standards could protect against the health effects associated with longer term O₃ exposures. Though there is uncertainty associated with these comparisons (e.g., due to uncertainty in the potential for a threshold to exist; uncertainty in the identification of such a threshold, should one exist; uncertainty in the long-term concentration below which confidence intervals widen notably, based on visual inspection of concentration-response function in the published study; and the limited number of urban case study areas for which adjusted air quality is available), this analysis can provide insight into the extent to which various alternative short-term standards would be expected to maintain long-term O₃ concentrations below those where we have the most confidence in the reported concentration-response relationship with respiratory mortality.

Table 4-3 indicates that when considering recent (i.e., unadjusted) air quality, 2 of 12 urban case study areas had seasonal average 1-hour daily max O₃ concentrations at or below 53 ppb in all of the years examined. When air quality was adjusted to just meet the current 8-hour standard (75 ppb in Table 4-3), 6 of 12 urban case study areas had seasonal average 1-hour daily max O₃ concentrations at or below 53 ppb in all of the years examined. When air quality is further adjusted to just meet potential alternative standards with lower levels, seasonal averages of 1-hour daily max O₃ concentrations are estimated to be at or below 53 ppb in 9 of 12 urban case study areas (70 ppb level), 10 of 12 urban case study areas (65 ppb level), and 11 of 11 urban case study areas (60 ppb level).¹⁸ Though as noted above there are important uncertainties associated with interpreting these comparisons, they suggest that in many locations across the U.S. a standard with an 8-hour averaging time, when combined with the current 4th high form and an appropriate standard level, would be expected to maintain seasonal averages of 1-hour daily max O₃ concentrations below those where analyses indicate the most confidence in the concentration-response relationship with respiratory mortality reported by Jerrett et al. (2009).

¹⁸As described in the HREA, a standard level of 60 ppb was not evaluated in New York City (U.S. EPA, 2014, chapter 4).

Table 4-3 Seasonal averages of 1-hour daily max O₃ concentrations in U.S. urban case study areas for recent air quality and air quality adjusted to just meet the current and potential alternative standards.

	Air Quality Adjusted to:	2006 (Adj Yrs 2006-2008)	2007 (Adj Yrs 2006-2008)	2008 (Adj Yrs 2008-2010)	2009 (Adj Yrs 2008-2010)	2010 (Adj Yrs 2008-2010)
Atlanta	Recent	65	63	57	50	56
	75	53	52	53	47	52
	70	50	49	49	44	49
	65	47	46	46	42	46
	60	45	44	44	40	44
Baltimore	Recent	60	59	57	52	60
	75	54	54	53	49	55
	70	52	51	51	48	53
	65	49	49	48	46	50
	60	46	46	46	44	48
Boston	Recent	49	50	46	45	49
	75	48	49	49	45	48
	70	46	47	48	44	48
	65	44	45	46	43	46
	60	43	43	44	41	44
Cleveland	Recent	51	52	53	49	54
	75	49	50	51	47	51
	70	47	48	48	45	48
	65	45	45	45	43	45
	60	41	41	41	40	42
Denver	Recent	63	63	63	58	60
	75	62	61	63	58	60
	70	60	59	62	58	58
	65	58	58	59	56	55
	60	53	53	53	51	50
Detroit	Recent	50	54	51	48	52
	75	50	52	NA	NA	NA
	70	48	50	51	49	52
	65	47	49	49	47	50
	60	45	46	46	45	47
Houston	Recent	53	48	47	47	46
	75	48	46	47	48	46
	70	47	45	46	47	46
	65	46	44	45	46	45
	60	45	43	43	44	44
Los Angeles	Recent	65	61	64	62	57
	75	58	59	60	60	58
	70	55	56	57	58	56
	65	52	53	54	54	53
	60	50	51	52	52	50
New York City	Recent	53	54	55	48	55
	75	47	47	51	47	51
	70	44	45	48	45	48
	65	36	36	39	38	39
	60	NA	NA	NA	NA	NA
Philadelphia	Recent	56	59	57	51	58
	75	51	52	54	49	54
	70	49	50	51	47	52
	65	47	48	49	45	49
	60	45	46	47	43	47
Sacramento	Recent	66	59	65	61	55
	75	55	50	54	51	48
	70	52	48	51	49	46
	65	50	46	49	47	44
	60	47	44	46	44	42
Saint Louis	Recent	58	58	52	51	55
	75	53	53	51	50	54
	70	50	51	50	48	52
	65	47	48	48	46	49
	60	44	45	45	43	46

Based on the above analyses, we conclude that the available epidemiologic evidence is consistent with the available evidence from controlled human exposure studies in providing support for consideration of an O₃ standard level in the range of 70 to 60 ppb. Compared to the current standard, a standard level from within this range would be expected to be more effective at maintaining short-term and long-term ambient O₃ concentrations below those present in studies reporting O₃-associated mortality and/or morbidity.

In reaching overall staff conclusions about an appropriate range of standard levels for consideration, we further evaluate the results of the exposure and risk assessments that are based on modeling changes in the entire distribution of ambient O₃ concentrations to simulate just meeting potential alternative standards. These results are discussed below in section 4.4.2.

4.4.2 Air Quality-, Exposure-, and Risk-Based Considerations

Beyond considering the available evidence, we also consider the extent to which specific potential alternative standard levels, in conjunction with the current averaging time and form (3-year average of annual 4th highest 8-hour daily max), could reduce estimated O₃ exposures and health risks. In the first draft PA (U.S. EPA, 2012b), we concluded that the available evidence supports conducting further exposure and risk analyses of potential alternative O₃ standard levels in the range of 70 down to 60 ppb. Based on these conclusions, the HREA evaluates exposures and risks estimated to be associated with potential alternative standard levels from the upper (70 ppb), middle (65 ppb), and lower (60 ppb) portions of this range. In considering these analyses in this PA, we consider the following question:

- **To what extent does the available exposure and risk information provide support for considering potential alternative standard levels from 70 to 60 ppb, when combined with the current 8-hour averaging time and 4th high form?**

In considering exposure and risk analyses, we emphasize the nature and magnitude of the O₃ exposures and health risks estimated to remain upon just meeting each alternative standard level, and the changes in exposures and risks estimated for each alternative level when compared to the current standard. Section 4.4.2.1 below discusses our exposure-based considerations. Sections 4.4.2.2 and 4.4.2.3 discuss our consideration of estimates of lung function risks and estimates of epidemiology-based mortality/morbidity risks, respectively.

4.4.2.1 Exposure-Based Considerations

As discussed in more detail in section 3.2.2 of this PA, the exposure assessment presented in the HREA (U.S. EPA, 2014, Chapter 5) provides estimates of the number and percent of people exposed to O₃ concentrations at or above benchmark concentrations of 60, 70, and 80 ppb, while at moderate or greater exertion. Estimates of such “exposures of concern”

provide perspective on the potential public health impacts of O₃-related effects, including for effects that cannot currently be evaluated in a quantitative risk assessment. The approach taken in the HREA to estimating exposures of concern, and the key uncertainties associated with exposure estimates, are summarized in section 3.2.2 for air quality adjusted to just meet the current standard and are discussed in more detail in chapter 5 of the HREA (U.S. EPA, 2014). As discussed in section 3.2.2, when evaluating potential alternative standard levels we focus on modeled exposures for school-age children (ages 5-18), noting that percentages of asthmatic school-age children estimated to experience exposures of concern are virtually indistinguishable from those for all children, and that patterns of exposure in children represent a broader range of at-risk populations, which includes adult asthmatics and older adults. In this review, CASAC advised EPA to focus on the 60 ppb benchmark as being relevant for considering adverse effects on people with asthma (Frey, 2014, p. 6).

In this section, we consider the following question:

- **To what extent are potential alternative standards with revised levels estimated to reduce the occurrence of O₃ exposures of concern, compared to the current standard, and what are the nature and magnitude of the exposures remaining for each alternative standard level evaluated?**

Key results related to this question are summarized below (Figures 4-1 to 4-4). Figures 4-1 (estimates averaged over years) and 4-2 (estimates from worst-case years) present estimates of one or more exposures of concern, and Figures 4-3 (estimates averaged over years) and 4-4 (estimates from worst-case years) present estimates of two or more exposures of concern.

Figure 4-1. Percent of children estimated to experience one or more exposures of concern at or above 60, 70, or 80 ppb for air quality adjusted to just meet the current and potential alternative standards (averaged over 2006 to 2010)

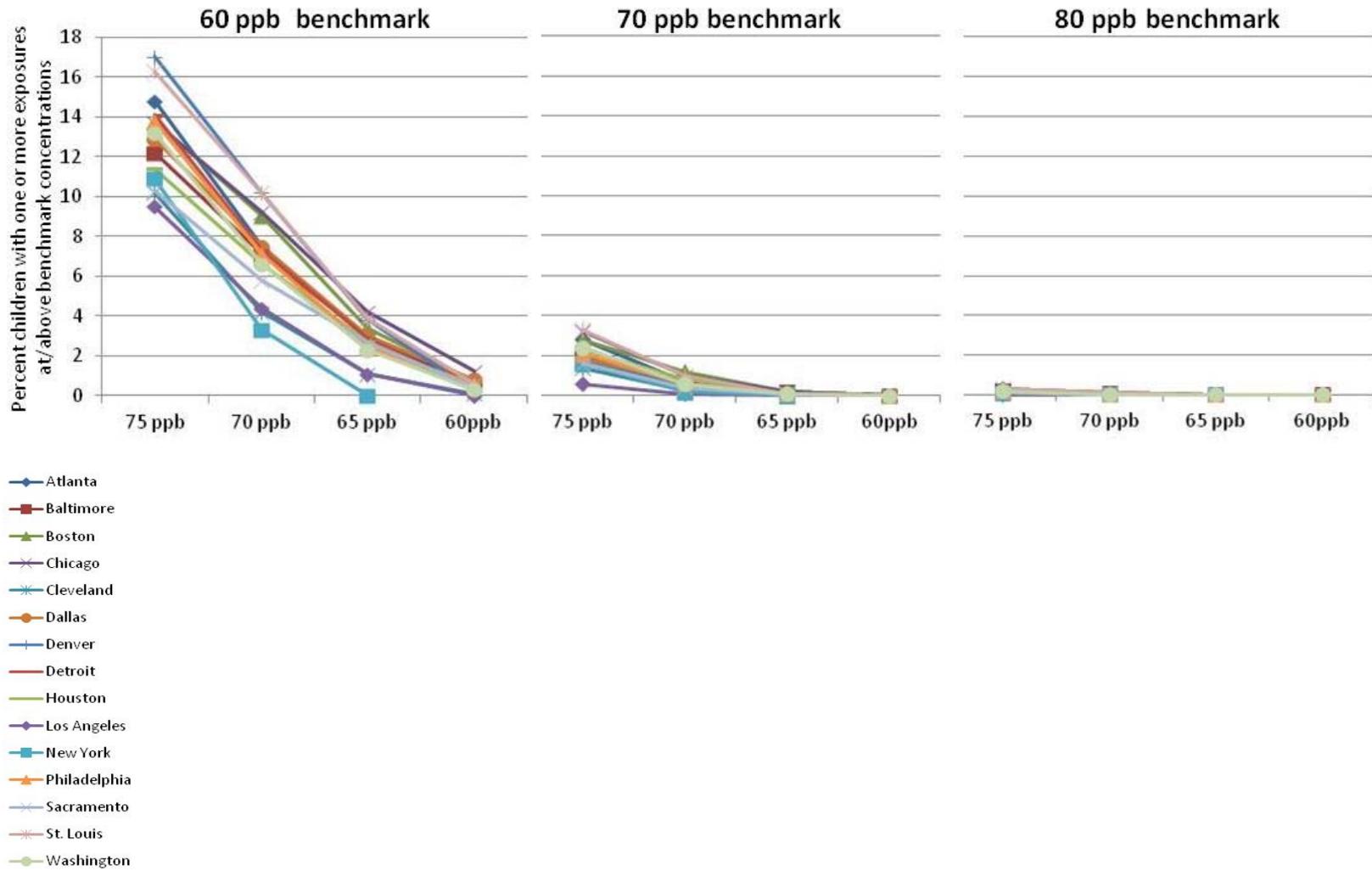
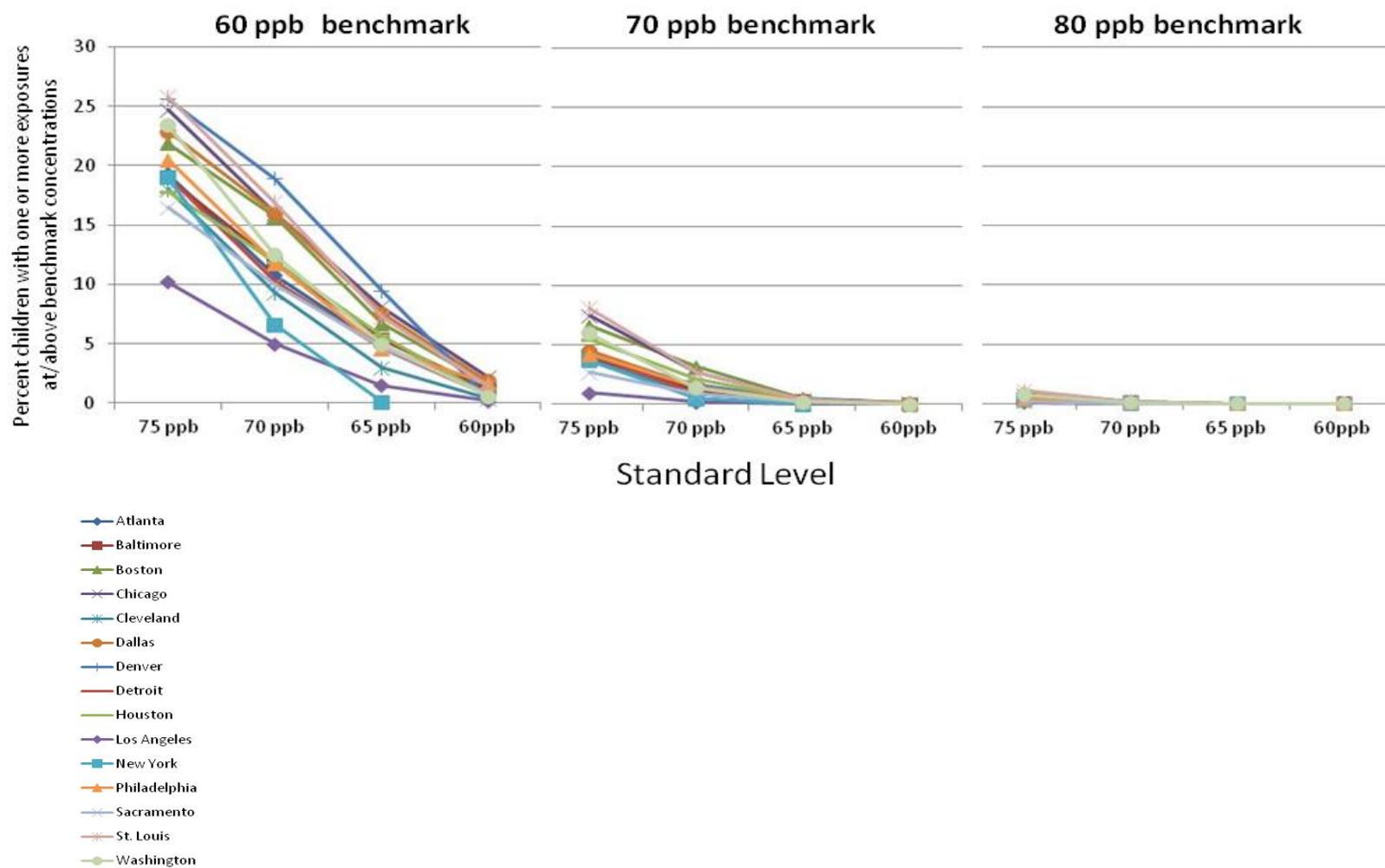


Figure 4-2. Percent of children estimated to experience one or more exposures of concern at or above 60, 70, or 80 ppb for air quality adjusted to just meet the current and potential alternative standards (worst-case year from 2006 to 2010¹⁹)



¹⁹“Worst-case” year refers to the year in each urban case study area with the largest percentage of children estimated to experience exposures of concern.

Figure 4-3. Percent of children estimated to experience two or more exposures of concern at or above 60, 70, or 80 ppb for air quality adjusted to just meet the current and potential alternative standards (averaged over 2006 to 2010)

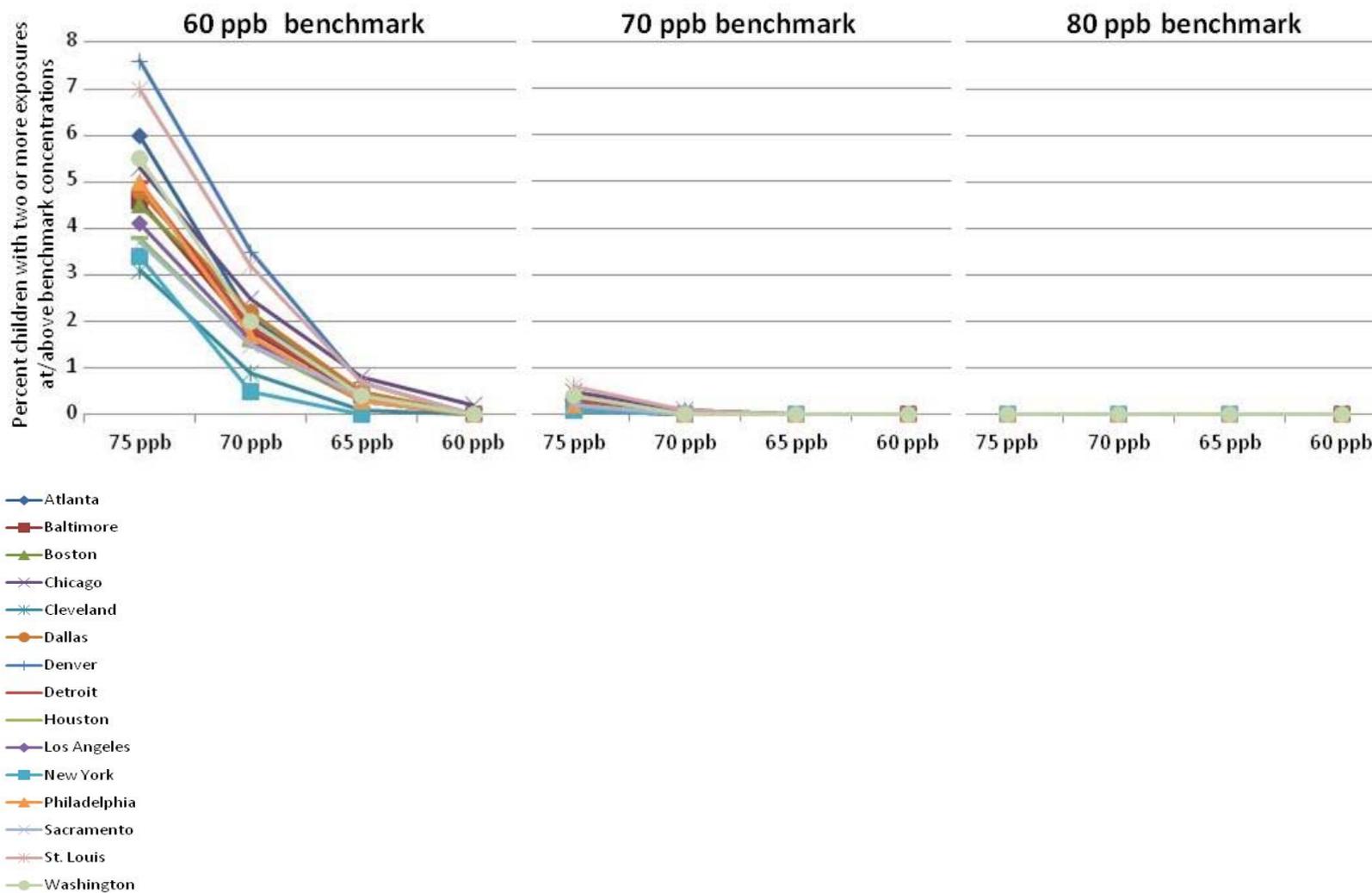
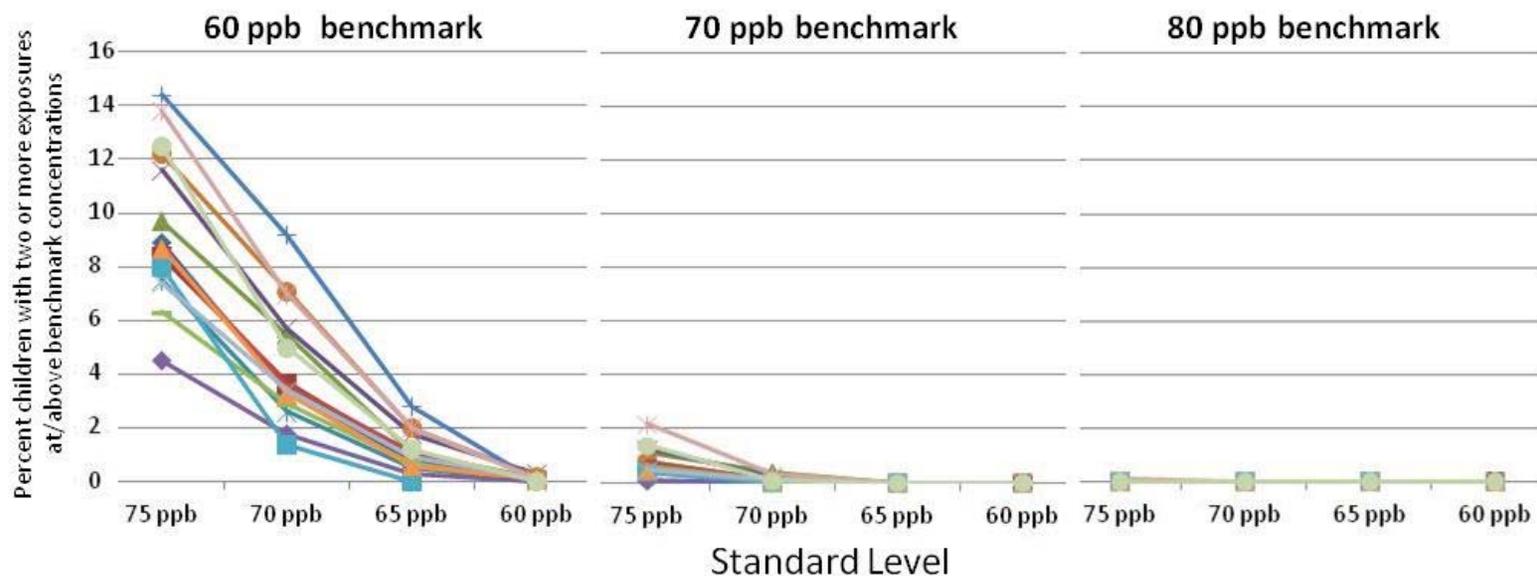


Figure 4-4. Percent of children estimated to experience two or more exposures of concern at or above 60, 70, or 80 ppb for air quality adjusted to just meet the current and potential alternative standards (worst-case year from 2006 to 2010)



- Atlanta
- Baltimore
- Boston
- Chicago
- Cleveland
- Dallas
- Denver
- Detroit
- Houston
- Los Angeles
- New York
- Philadelphia
- Sacramento
- St. Louis
- Washington

As illustrated above in Figures 4-1 to 4-4, adjusting air quality to just meet progressively lower potential alternative standard levels reduces estimated exposures of concern consistently across urban case study areas. These results reflect the consistent reductions in the highest ambient O₃ concentrations upon air quality adjustment, as summarized in section 3.2.1 and as discussed in more detail in the HREA (U.S. EPA, 2014, chapter 4). Based on Figures 4-1 to 4-4 and the associated details described in the HREA (U.S. EPA 2014, chapter 5), we take note of the following with regard to exposures of concern for specific potential alternative standard levels:

1. For an O₃ standard level of 70 ppb:
 - a. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow approximately 3 to 10% of children in urban case study areas to experience one or more exposures of concern at or above 60 ppb (approximately 30 to 70% reduction, relative to current standard). Summing across urban case study areas, these percentages correspond to over 1 million children experiencing over 1.5 million exposures of concern at or above 60 ppb during a single O₃ season. Of these children, over 100,000 are asthmatics.
 - b. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow approximately 0.5 to 3.5% of children in urban case study areas to experience two or more exposures of concern at or above 60 ppb (approximately 50 to 85% reduction, relative to current standard).
 - c. In the worst-case years (i.e., those with the largest exposure estimates), a standard with a level of 70 ppb is estimated to allow approximately 5 to 19% of children in urban case study areas to experience one or more exposures of concern at or above 60 ppb, and approximately 2 to 9% to experience two or more.
 - d. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow approximately 1% or less of children to experience one or more exposures of concern at or above 70 ppb (approximately 55 to 90% reduction, relative to current standard), and far less than 1% to experience two or more such exposures (approximately 65 to 100% reduction, relative to current standard).
 - e. In the worst-case years, approximately 3% or less of children are estimated to experience one or more exposures of concern at or above 70 ppb, and less than 1% are estimated to experience two or more such exposures.
 - f. A standard with a level of 70 ppb is estimated to allow less than 1% of children to experience one or more exposures of concern at or above 80 ppb, even in the worst-case years. No children are estimated to experience two or more such exposures.
2. For an O₃ standard level of 65 ppb:

- a. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow approximately 4% or less of children in urban case study areas to experience one or more exposures of concern at or above 60 ppb (approximately 70 to 100% reduction, relative to current standard). Summing across urban case study areas, these percentages correspond to almost 400,000 children experiencing almost 500,000 exposures of concern at or above 60 ppb during a single O₃ season. Of these children, about 40,000 are asthmatics.
 - b. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow less than 1% of children to experience two or more exposures of concern at or above 60 ppb (approximately 85 to 100% reduction, relative to current standard).
 - c. In the worst-case years, a standard with a level of 65 ppb is estimated to allow approximately 10% or less of children to experience one or more exposures of concern at or above 60 ppb, and approximately 3% or less to experience two or more such exposures.
 - d. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow approximately 1% or less of children to experience one or more exposures of concern at or above 70 ppb (approximately 90 to 100% reduction, relative to current standard), and almost no children to experience two or more such exposures. Even in the worst-case years, a level of 65 ppb is estimated to allow less than 1% of children to experience exposures of concern at or above 70 ppb.
 - e. A standard with a level of 65 ppb is estimated to allow virtually no children to experience exposures of concern at or above 80 ppb, even in the worst-case years.
3. For an O₃ standard level of 60 ppb:
- a. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to allow approximately 1% or less of children to experience one or more exposures of concern at or above 60 ppb (approximately 90 to 100% reduction, relative to current standard), and virtually no children to experience multiple such exposures.
 - b. In the worst-case years, a standard with a level of 60 ppb is estimated to allow approximately 2% or less of children to experience one or more exposures of concern at or above 60 ppb, and almost no children to experience multiple such exposures.
 - c. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to almost eliminate exposures of concern at or above 70 ppb or 80 ppb. Even in years with the highest exposure estimates, virtually no children are estimated to experience such exposures.

In further considering these exposure estimates, we take note of the associated uncertainties, as discussed in more detail in section 3.2.2 of this PA (and in Chapter 5 of the

HREA, U.S. EPA, 2014). These include (1) individual variability in responsiveness to O₃ exposures such that only a subset of individuals who experience exposures at (or above) a benchmark concentration would experience health effects; (2) potential to underestimate exposures in most highly exposed populations; and (3) potential to overestimate exposures in populations who alter behavior in response to high O₃ days (i.e., spend less time being active outdoors). The implications of estimated exposures of concern for potential alternative standard levels are discussed below in section 4.6.

4.4.2.2 Risk-Based Considerations: Lung Function

As discussed above in more detail in section 3.2.3.1 of this PA, the assessment of lung function risks presented in the HREA (U.S. EPA, 2014, Chapter 6) provides estimates of the number and percent of people experiencing O₃-induced lung function decrements greater than or equal to 10, 15, and 20%. In the current and past reviews, CASAC has advised EPA to focus on decrements of 10% or greater when considering people with pre-existing lung disease (Frey, 2014; Samet, 2011).

Lung function risk estimates are based on an updated dose-threshold model that estimates FEV₁ responses for healthy adults following short-term exposures to O₃ (McDonnell, Stewart, and Smith, 2010), reflecting methodological improvements since the last review (U.S. EPA, 2014, section 6.2.4). The approach taken in the HREA to estimating O₃-induced lung function decrements, and the key uncertainties associated with these estimates, are summarized in section 3.2.3.1 for air quality adjusted to just meet the current standard and are discussed in more detail in chapter 6 of the HREA (U.S. EPA, 2014).

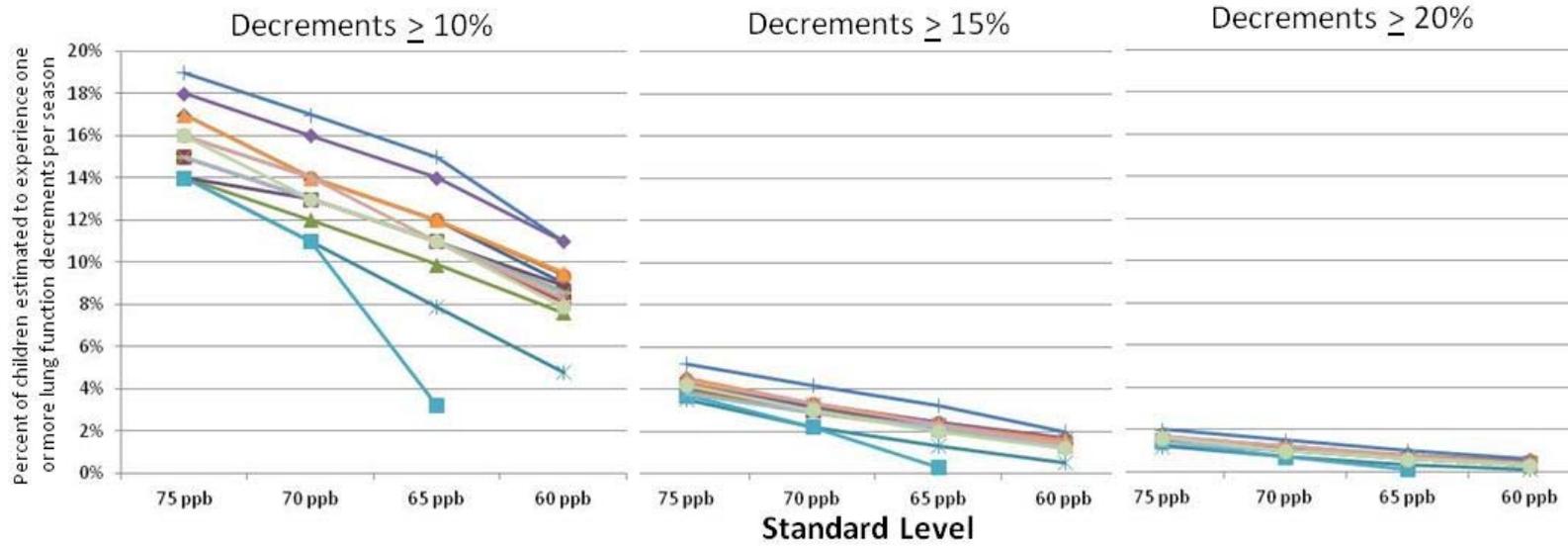
As discussed in section 3.2.3.1, in evaluating potential alternative standard levels we focus on modeled exposures for school-age children, with an emphasis on asthmatic children. As with exposures of concern, the percentages of all school age children and asthmatic school age children estimated to experience particular O₃-induced lung function decrements are virtually indistinguishable.

In this section, we consider the following question:

- **To what extent are potential alternative standards with revised levels estimated to decrease the occurrence of O₃-induced lung function decrements, compared to the current standard, and what are the nature and magnitude of the decrements remaining for each alternative standard level evaluated?**

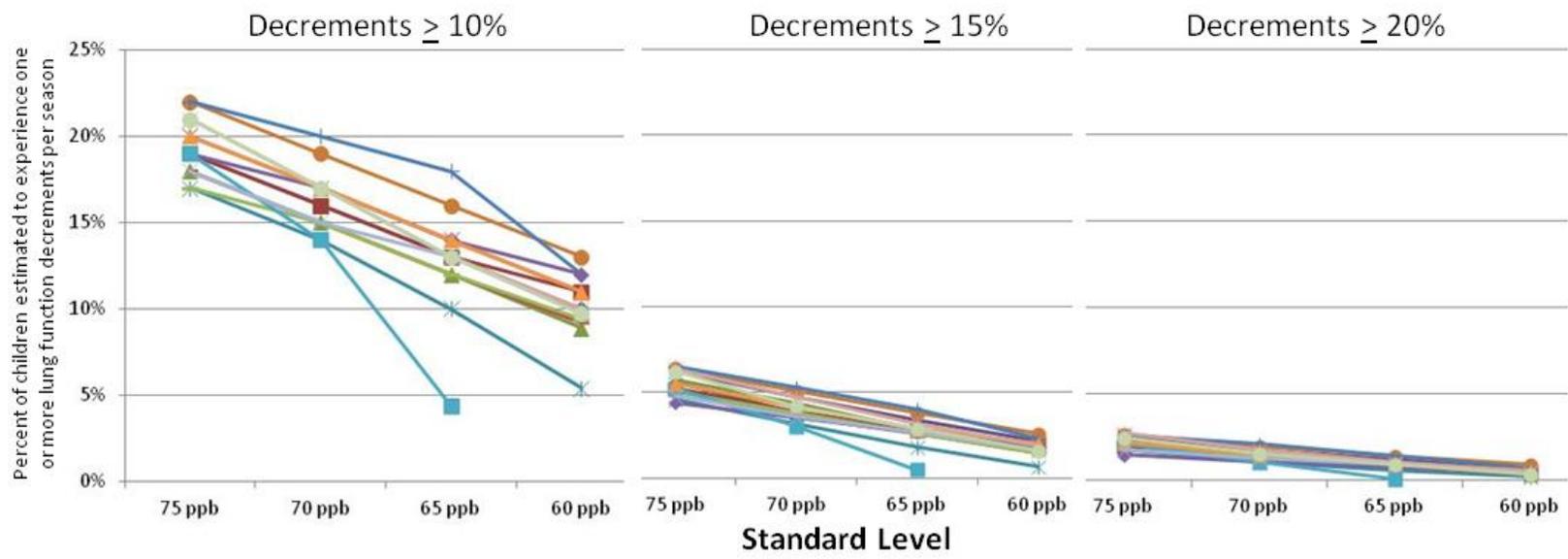
Key results related to this question are summarized below (Figures 4-5 to 4-8). Figures 4-5 (estimates averaged over years) and 4-6 (estimates from worst-case years) present estimates of one or more O₃-induced lung function decrements, and Figures 4-7 (estimates averaged over years) and 4-8 (estimates from worst-case years) present estimates of two or more decrements.

Figure 4-5. Percent of children estimated to experience one or more O₃-induced lung function decrements greater than 10, 15, or 20% for air quality adjusted to just meet the current and potential alternative standards (averaged over 2006 to 2010)



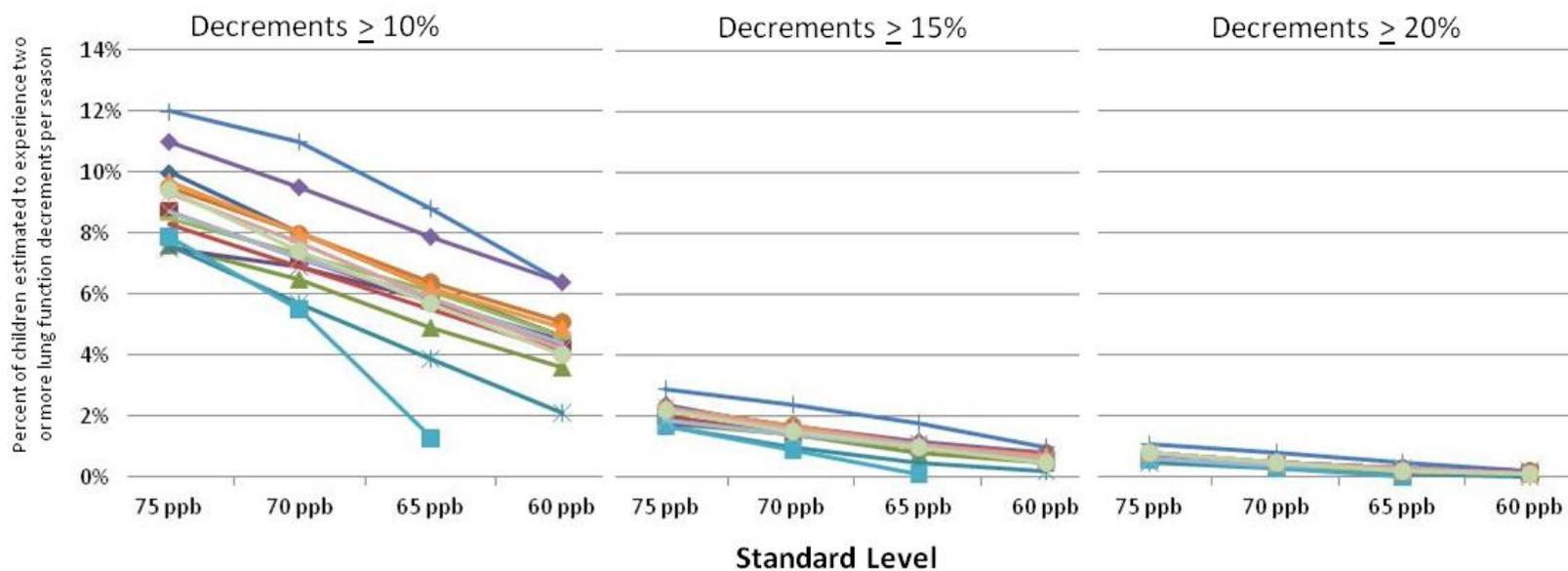
- ◆ Atlanta
- Baltimore
- ▲ Boston
- ✕ Chicago
- ✱ Cleveland
- Dallas
- ◆ Denver
- Detroit
- Houston
- ◆ Los Angeles
- New York
- ▲ Philadelphia
- ✕ Sacramento
- ✱ St Louis
- Washington

Figure 4-6. Percent of children estimated to experience one or more O₃-induced lung function decrements greater than 10, 15, or 20% for air quality adjusted to just meet the current and potential alternative standards (worst-case year from 2006 to 2010)



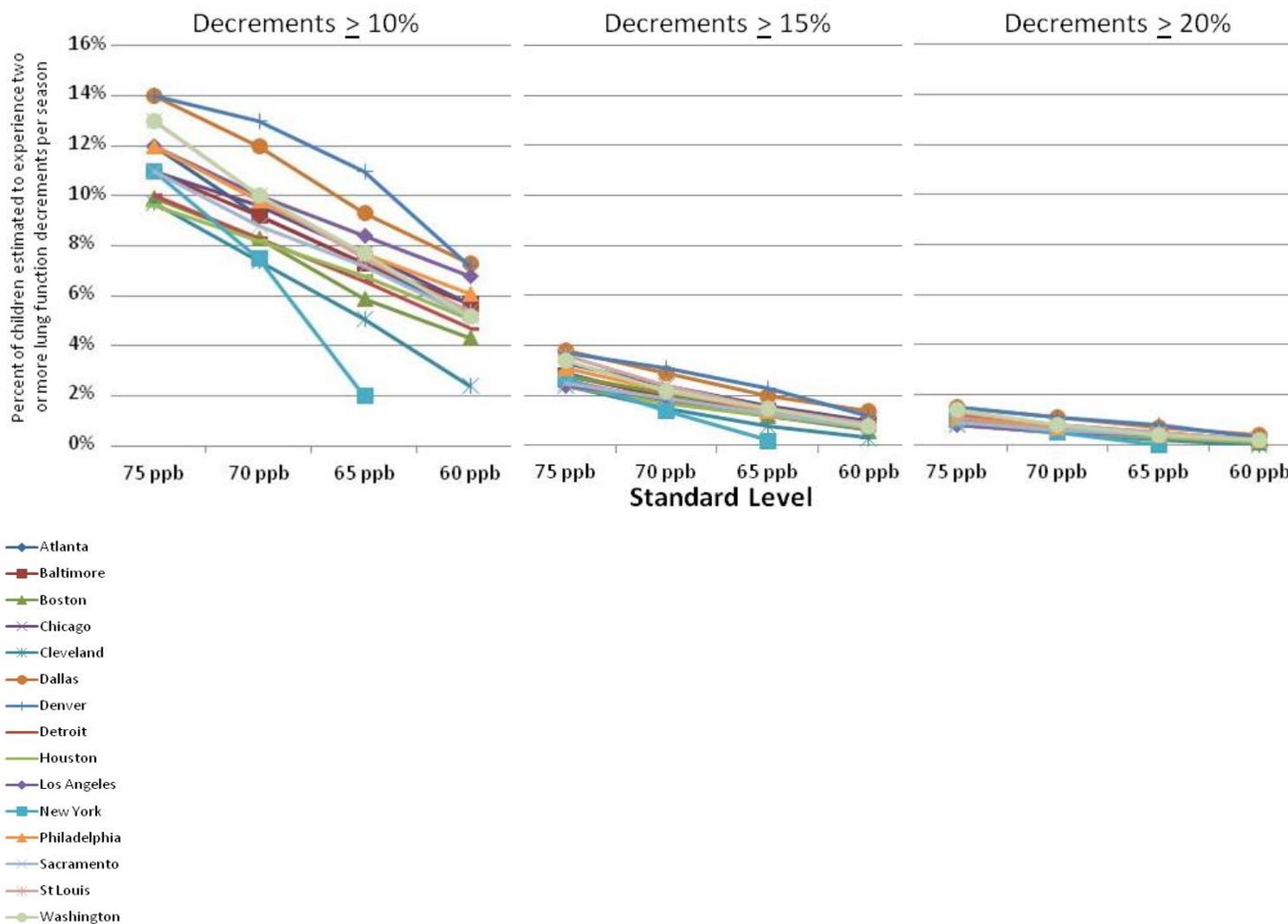
- Atlanta
- Baltimore
- Boston
- Chicago
- Cleveland
- Dallas
- Denver
- Detroit
- Houston
- Los Angeles
- New York
- Philadelphia
- Sacramento
- St Louis
- Washington

Figure 4-7. Percent of children estimated to experience two or more O₃-induced lung function decrements greater than 10, 15, or 20% for air quality adjusted to just meet the current and potential alternative standards (averaged over 2006 to 2010)



- ◆ Atlanta
- Baltimore
- ▲ Boston
- ✕ Chicago
- ✱ Cleveland
- Dallas
- ◆ Denver
- Detroit
- Houston
- ◆ Los Angeles
- New York
- ▲ Philadelphia
- ✕ Sacramento
- ✱ St Louis
- Washington

Figure 4-8. Percent of children estimated to experience two or more O₃-induced lung function decrements greater than 10, 15, or 20% for air quality adjusted to just meet the current and potential alternative standards (worst-case year from 2006 to 2010)



As illustrated above in Figures 4-5 to 4-8, adjusting air quality to just meet progressively lower potential alternative standard levels consistently reduces the percent of children estimated to experience potentially adverse lung function decrements. These results reflect the consistent reductions in the highest ambient O₃ concentrations upon air quality adjustment (section 3.2.1; U.S. EPA, 2014, chapter 4).²⁰ Based on Figures 4-5 to 4-8 and the associated details described in the HREA (U.S. EPA 2014, chapter 6), we take note of the following with regard to specific potential alternative standard levels:

1. For an O₃ standard level of 70 ppb:
 - a. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow approximately 11 to 17% of children in urban case study areas, including asthmatic children, to experience one or more O₃-induced lung function decrements $\geq 10\%$ (approximately 6 to 27% reduction, relative to current standard) per season. Summing across case study areas, these percentages correspond to approximately 260,000 asthmatic children experiencing approximately 1 million total occurrences of O₃-induced lung function decrements greater than or equal to 10%.
 - b. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow approximately 6 to 11% of children, including asthmatic children, to experience two or more O₃-induced lung function decrements $\geq 10\%$ (approximately 8 to 30% reduction, relative to current standard).
 - c. In the worst-case years, a standard with a level of 70 ppb is estimated to allow approximately 14 to 20% of children, including asthmatic children, to experience one or more O₃-induced lung function decrements $\geq 10\%$, and approximately 7 to 13% to experience two or more such decrements.
 - d. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow approximately 2 to 4% of children, including asthmatic children, to experience one or more O₃-induced lung function decrements $\geq 15\%$, and approximately 1 to 2.5% of children to experience two or more such O₃-induced decrements. In the worst-case years, approximately 3 to 5% of children are estimated to experience one or more O₃-induced lung function decrements $\geq 15\%$, and approximately 1 to 3% are estimated to experience two or more such decrements.
 - e. A standard with a level of 70 ppb is estimated to allow 2% or fewer children to experience any O₃-induced lung function decrements $\geq 20\%$, even in the worst-case years. Approximately 1% or fewer children are estimated to experience two

²⁰As discussed in section 3.2.3.1, the impact of the dose threshold in the lung function risk model is that O₃-induced FEV₁ decrements result primarily from exposures on days with average ambient O₃ concentrations above about 40 ppb (US EPA, 2014, section 6.3.1, Figure 6-9).

or more O₃-induced lung function decrements $\geq 20\%$, even in the worst-case years.

2. For an O₃ standard level of 65 ppb:

- a. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow approximately 3 to 15% of children, including asthmatic children, to experience one or more O₃-induced lung function decrements $\geq 10\%$ (approximately 20 to 77% reduction, relative to current standard). Summing across urban case study areas, these percentages correspond to approximately 190,000 asthmatic children experiencing almost 750,000 total occurrences of O₃-induced lung function decrements $\geq 10\%$.
- b. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow approximately 1 to 9% of children, including asthmatic children, to experience two or more O₃-induced lung function decrements $\geq 10\%$ (approximately 20 to 80% reduction, relative to current standard).
- c. In the worst-case years, a standard with a level of 65 ppb is estimated to allow approximately 4 to 18% of children to experience one or more O₃-induced lung function decrements $\geq 10\%$, and approximately 2 to 11% to experience two or more such decrements.
- d. On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow approximately 3% or less of children to experience one or more O₃-induced lung function decrements $\geq 15\%$, and approximately 2% or less of children to experience two or more such O₃-induced decrements. In the worst-case years, approximately 4% or less of children are estimated to experience one or more O₃-induced lung function decrements $\geq 15\%$, and up to approximately 2% are estimated to experience two or more such decrements.
- e. A standard with a level of 65 ppb is estimated to allow less than 1.5% of children to experience any O₃-induced lung function decrements $\geq 20\%$, even in the worst-case years. A standard with a level of 65 ppb is estimated to allow less than 1% of children to experience two or more O₃-induced lung function decrements $\geq 20\%$, even in the worst-case years.

3. For an O₃ standard level of 60 ppb:

- a. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to allow approximately 5 to 11% of children, including asthmatic children, to experience one or more O₃-induced lung function decrements $\geq 10\%$ (approximately 35 to 77% reduction, relative to current standard). Summing across urban case study areas, these percentages correspond to approximately 140,000 asthmatic children experiencing approximately 500,000 total occurrences of O₃-induced lung function decrements $\geq 10\%$.

- b. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to allow approximately 2 to 6% of children to experience two or more O₃-induced lung function decrements $\geq 10\%$ (approximately 40 to 70% reduction, relative to current standard).
- c. In the worst-case years, a standard with a level of 60 ppb is estimated to allow approximately 5 to 13% of children to experience one or more O₃-induced lung function decrements $\geq 10\%$, and approximately 2 to 7% to experience two or more such decrements.
- d. A standard with a level of 60 ppb is estimated to allow less than about 3% of children to experience any O₃-induced lung function decrements $\geq 15\%$ and less than 1% to experience decrements greater than 20%, even in the worst-case years. A standard with a level of 60 ppb is estimated to allow less than 1.5% of children to experience two or more O₃-induced lung function decrements $\geq 15\%$ and less than 0.5% to experience two or more decrements $\geq 20\%$, even in the worst-case years.

In further considering these exposure estimates, we take note of the associated uncertainties, as discussed in more detail in section 3.2.2 of this PA. In addition to the uncertainties in exposure estimates noted above, these include the relative lack of exposure-response information for key at-risk populations (i.e., children and asthmatics), since most controlled human exposures studies are conducted in healthy adults. Section 4.6 (below) discusses the implications of estimates of the occurrence of O₃-induced lung function decrements for potential alternative standard levels.

4.4.2.3 Risk-Based Considerations: Epidemiology-Based Mortality and Morbidity

The epidemiology-based risk assessments presented in the HREA (U.S. EPA, 2014, chapter 7) provide estimates of total mortality, respiratory hospital admissions and emergency department visits, and asthma exacerbations associated with short-term O₃ concentrations. The HREA also presents estimates of respiratory mortality associated with long-term²¹ concentrations. In evaluating these risk estimates, we consider the following question:

²¹Estimates of respiratory mortality associated with long-term O₃ concentrations are based on the study by Jerrett et al. (2009). Consistent with the O₃ metric used in the study, risk estimates are based on seasonal averages of 1-hour daily max O₃ concentrations.

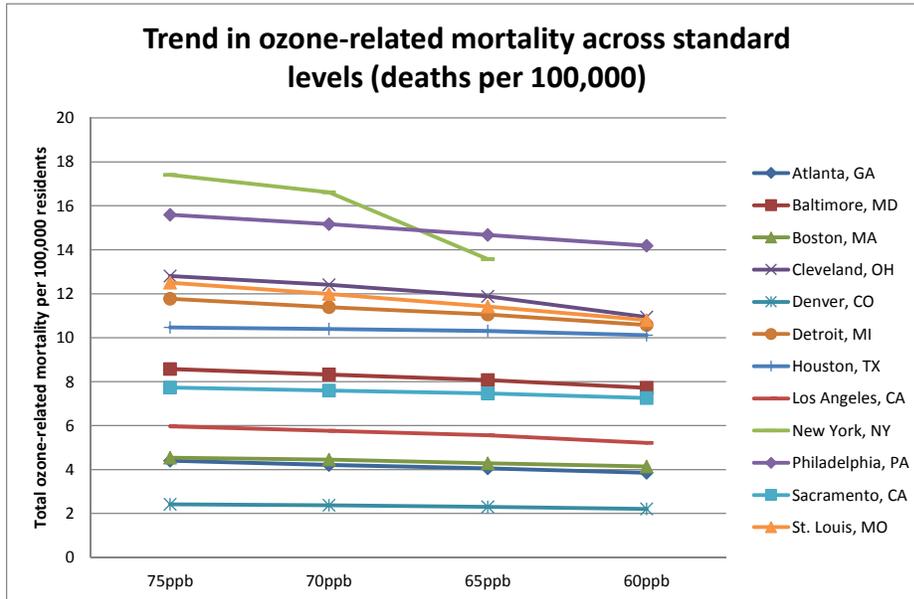
- **To what extent are potential alternative standards with revised levels estimated to decrease O₃ health risks, compared to the current standard, and what are the nature and magnitude of the health risks remaining for each alternative standard level evaluated?**

As discussed in more detail in section 3.2.3.2 of this PA, in considering this question we are mindful that the model-based approach used to adjust air quality in the HREA has important implications for risk estimates developed by applying concentration-response relationships from epidemiologic studies (section 3.2.1). In particular, given the use of linear concentration-response relationships, risk estimates are equally influenced by decreasing high O₃ concentrations and increasing low O₃ concentrations following air quality adjustment, when the increases and decreases are of equal magnitude. This and other uncertainties associated with risk estimates are discussed in section 3.2.3.2.

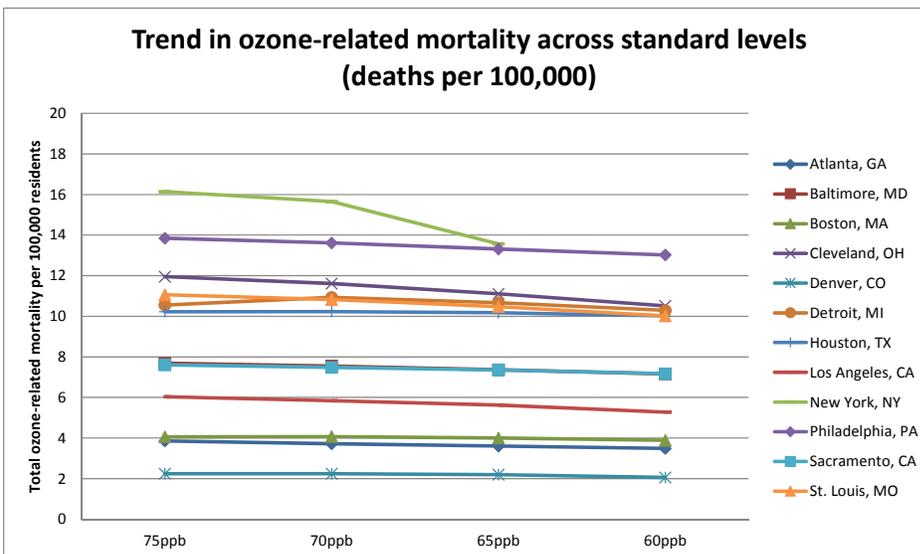
Key results from the HREA (U.S. EPA, 2014, chapter 7) are summarized below for estimates of total mortality associated with short-term O₃ concentrations (Figures 4-9 and 4-10) and respiratory hospital admissions associated with short-term O₃ concentrations (Figure 4-11). The other morbidity effects evaluated in the HREA (i.e., respiratory emergency department visits and asthma symptoms associated with short-term concentrations) exhibit patterns across standard levels that are similar to those reported for total mortality and respiratory hospital admissions (U.S. EPA, 2014, chapter 7).

Figure 4-9. Estimates of Total Mortality Associated with Short-Term O₃ Concentrations in Urban Case Study Areas (Air Quality Adjusted to Current and Potential alternative standard levels) – Total Risk

2007 Simulation year



2009 Simulation year



The risk estimates presented in Figure 4-9 above are based on applying linear concentration-response relationships to the full distributions of daily 8-hour “area-wide” O₃ concentrations. However, as in section 3.2.3.2 we note the ISA conclusion that there is less certainty in the shape of concentration-response functions for area-wide O₃ concentrations at the lower ends of warm season distributions (i.e., below about 20 to 40 ppb depending on the O₃

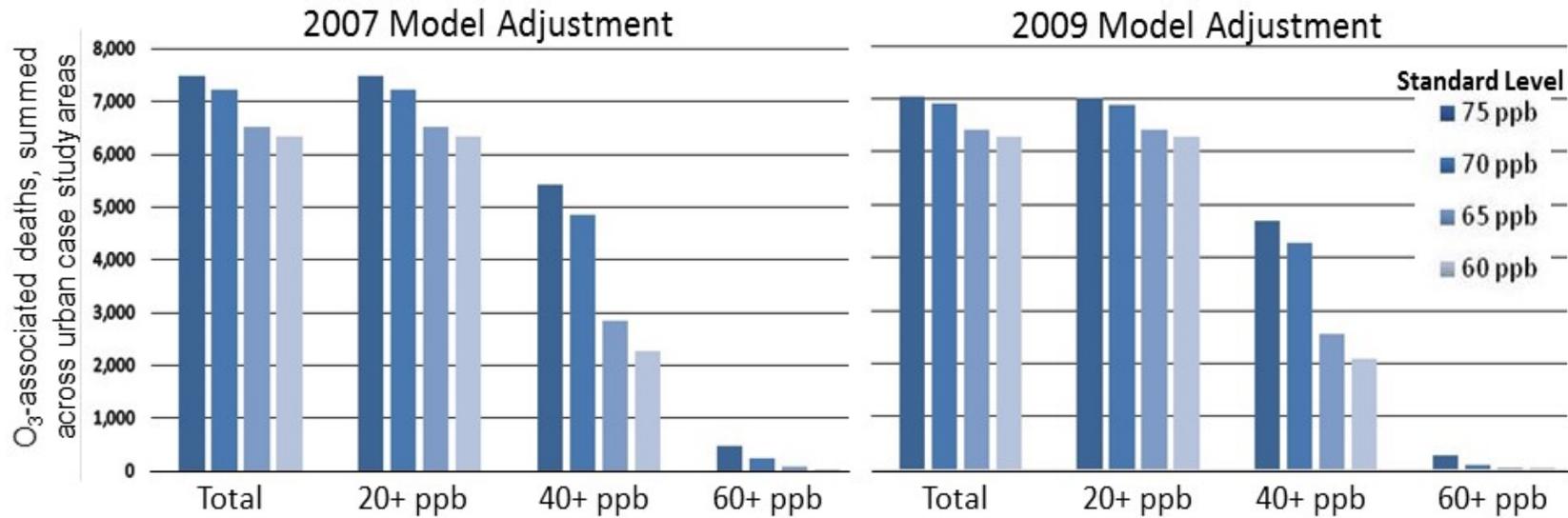
metric, health endpoint, and study population) (U.S. EPA, 2013, section 2.5.4.4). We also recognize that for the range of health endpoints evaluated, controlled human exposure and animal toxicological studies provide greater certainty in the increased incidence, magnitude, and severity of effects at higher exposure concentrations (discussed in sections 3.1.2.2 and 3.1.4.2 of this document).²² Thus, in addition to considering estimates of total O₃-associated risks, we also consider the extent to which risks are associated with days with higher, versus lower, area-wide O₃ concentrations.

Figure 4-10 presents estimates of O₃-associated deaths, summed across urban case study areas, for days with area-wide concentrations at or above 20, 40, and 60 ppb. As discussed in more detail in section 3.2.1 of this document, daytime O₃ concentrations in the upper portions of the distributions of area-wide concentrations tend to decrease upon adjustment to meet lower potential alternative standard levels, while concentrations in the lower portions of these distributions tend to increase. As a result, lower standard levels are estimated to be more effective at reducing deaths associated with the upper portions of these distributions of ambient O₃ concentrations than deaths associated with the full distributions.²³

²²As discussed in section 3.1.4.2, as ambient concentrations increase the potential for exposures to higher O₃ concentrations also increases. Thus with increasing ambient concentrations, controlled human exposure and animal toxicological studies provide greater certainty in the increased incidence, magnitude, and severity of O₃-attributable effects.

²³The relatively small proportion of O₃-associated deaths attributable to days with area-wide concentrations of 60 ppb or greater reflects the relatively small proportion of days with such elevated area-wide concentrations.

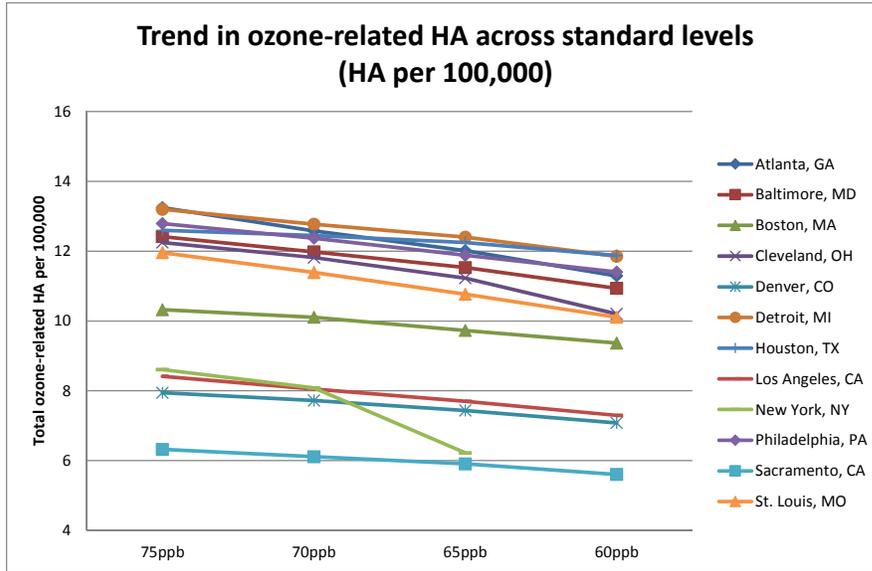
Figure 4-10. Estimates of O₃-Associated Deaths Attributable to Full Distribution of 8-Hour Area-Wide O₃ Concentrations and to Concentrations at or above 20, 40, or 60 ppb - Deaths Summed Across Urban Case Study Areas²⁴



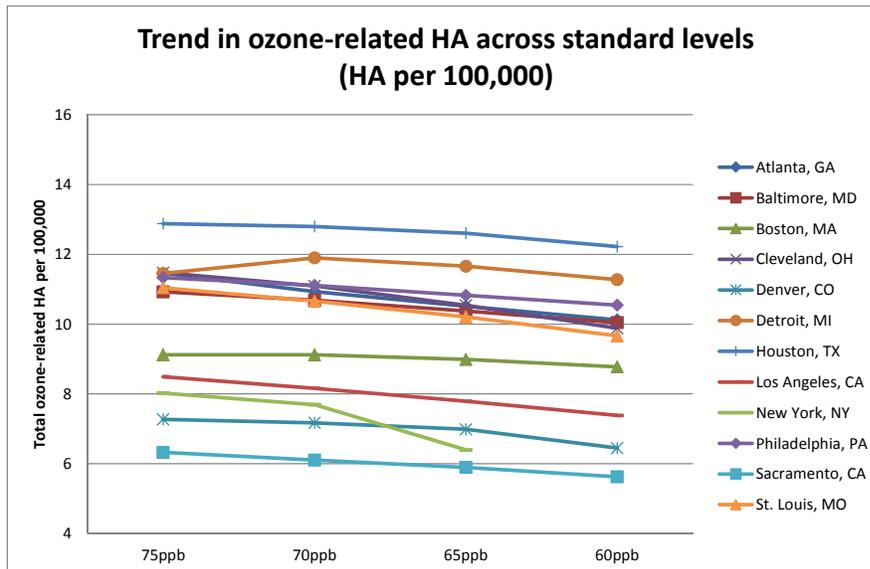
²⁴As discussed in section 4.3.3 of the HREA, the model-based air quality adjustment approach used to estimate risks associated with the current and alternative standards was unable to estimate the distribution of ambient O₃ concentrations in New York City upon just meeting an alternative standard with a level of 60 ppb. Therefore, the total number of deaths indicated for the 60 ppb standard level in Figure 4-10 reflects the 60 ppb estimates for all urban case study areas except New York City. For New York City, the estimated number of O₃-associated deaths for the 65 ppb standard level was assumed.

Figure 4-11. Estimates of Respiratory Hospital Admissions Associated with Short-Term O₃ Concentrations in Urban Case Study Areas (Air Quality Adjusted to Current and Potential alternative standard levels) – Total Risk

2007 Simulation year



2009 Simulation year

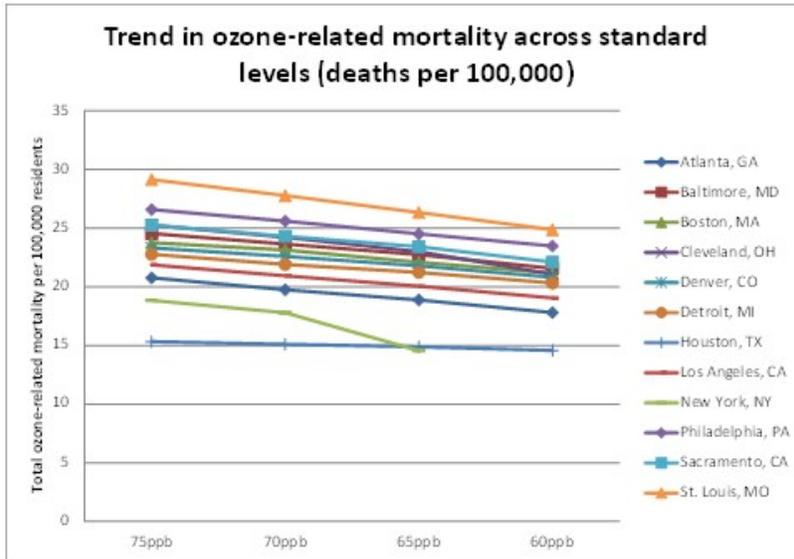


Key results from the HREA (U.S. EPA, 2014, chapter 7) are summarized in Figure 4-12 below for estimates of respiratory mortality associated with long-term O₃ concentrations, based on the study by Jerrett et al. (2009). As discussed in section 3.2.3.2 of this PA, Jerrett et al. (2009) reported that when seasonal averages of 1-hour daily maximum O₃ concentrations ranged from 33 to 104 ppb, there was no statistical deviation from a linear concentration-response relationship between O₃ and respiratory mortality across 96 U.S. cities (U.S. EPA, 2013, section 7.7). However, the authors reported “limited evidence” for an effect threshold at an O₃ concentration of 56 ppb (p=0.06). In communications with EPA staff (described in Sasser, 2014), the study authors indicated that it is not clear whether a threshold model is a better predictor of respiratory mortality than the linear model, and that “considerable caution should be exercised in accepting any specific threshold.” Consistent with this communication, the HREA estimated respiratory mortality associated with long-term O₃ concentrations based on the linear model from the published study, and in a series of sensitivity analyses with models that included thresholds ranging from 40 to 60 ppb (U.S. EPA, 2014, Figure 7-9).

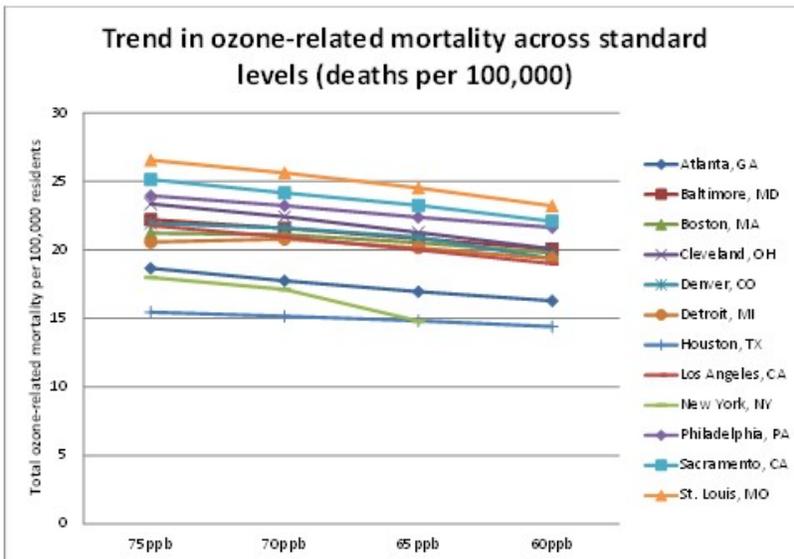
Figure 4-12 presents estimates of total O₃-associated respiratory deaths, based on a linear concentration-response relationship. As discussed for the current standard (section 3.2.3.2), HREA sensitivity analyses indicate that, if a threshold exists between 40 and 60 ppb, the number of respiratory deaths associated with long-term O₃ concentrations could potentially be considerably smaller than indicated by the no threshold model (U.S. EPA, 2014, Figure 7-9).

Figure 4-12. Estimates of Respiratory Mortality Associated with long-term O₃ Concentrations in Urban Case Study Areas (Air Quality Adjusted to Current and Potential alternative standard levels) – Total Risk

2007 Simulation Year



2009 Simulation Year



Based on Figures 4-9 to 4-12 and the associated details described in the HREA (U.S. EPA 2014, chapter 7), we take note of the following for an O₃ standard level of 70 ppb:

1. Total mortality associated with short-term O₃ concentrations:

- a. Across urban case study areas, risks are estimated to decrease by up to approximately 5% for a standard level of 70 ppb, compared to the current standard. Risk reductions are estimated consistently for the model year with generally higher O₃-associated risks (2007). In the year with generally lower risks (2009), a standard level of 70 ppb results in either no change or more modest reductions in estimated risks in most urban case study areas. In one area (Detroit) for the 2009 model year, O₃-associated mortality is estimated to increase by approximately 4%, compared to the current standard (see section 3.2.3.2 for further discussion of increased risk estimates following air quality adjustment²⁵).
 - b. When summed across urban case study areas, a standard level of 70 ppb is estimated to reduce O₃-associated deaths by approximately 4% (2007 model year) and 2% (2009 model year), compared to the current standard. For area-wide concentrations at or above 40 ppb, a standard level of 70 ppb is estimated to reduce O₃-associated deaths by approximately 10% (2007 model year) and 9% (2009 model year). For area-wide concentrations at or above 60 ppb, a standard level of 70 ppb is estimated to reduce O₃-associated deaths by approximately 50% (2007 model year) and 70% (2009 model year).²⁶
2. Respiratory hospital admissions associated with short-term O₃ concentrations: Compared to the current standard, changes in total risk estimated for a standard level of 70 ppb are similar to the changes in total risks estimated for total mortality (U.S. EPA, 2014, chapter 7).
 3. Respiratory mortality associated with long-term O₃ concentrations: A standard level of 70 ppb reduces total risk, compared to the current standard. Across urban case study areas, risks are estimated to decrease by up to approximately 6%. These risk reductions are estimated most consistently for the model year with generally higher O₃-associated risks (2007). In the year with generally lower O₃ concentrations (2009), a standard level of 70 ppb results in smaller reductions in estimated risks in most urban case study areas. In one area (Detroit) for the 2009 model year, O₃-associated mortality is estimated to increase by approximately 1%, compared to the current standard.

Based on Figures 4-9 to 4-12 and the associated details described in the HREA (U.S. EPA 2014, chapter 7), we take note of the following for an O₃ standard level of 65 ppb:

1. Total mortality associated with short-term O₃ concentrations:
 - a. Across most urban case study areas, risks are estimated to decrease by up to approximately 9% for a standard level of 65 ppb, compared to the current

²⁵As discussed in more detail above (section 3.2.3.2), because of the influence of the entire distribution of ambient O₃ concentrations on total risk estimates, the impacts of adjusting air quality to just meet potential alternative standards are more modest, and are less directionally consistent across urban case study areas, than observed for exposures of concern or O₃-induced lung function decrements.

²⁶These results reflect the fact that increases in area-wide O₃ concentrations upon air quality adjustment occur primarily at relatively low concentrations (i.e., on days with initial O₃ concentrations in the range of 10 to 40) (U.S. EPA, 2014, section 4.3.3.2 and appendix 7B, section 9.6).

standard. In one area (New York City), risks are estimated to decrease by up to approximately 22%.²⁷ These risk reductions are estimated most consistently for the model year with generally higher O₃-associated risks (2007). In the year with generally lower risks (2009), a standard level of 65 ppb results in smaller reductions in estimated risks in most urban case study areas. In one area (Detroit) for the 2009 model year, O₃-associated mortality is estimated to increase by approximately 1% compared to the current standard.

- b. When summed across urban case study areas, a standard level of 65 ppb is estimated to reduce O₃-associated deaths by approximately 13% (2007 model year) and 9% (2009 model year), compared to the current standard. For area-wide concentrations at or above 40 ppb, a standard level of 65 ppb is estimated to reduce O₃-associated deaths by approximately 47% (2007) and 46% (2009). For area-wide concentrations at or above 60 ppb, a standard level of 65 ppb is estimated to reduce O₃-associated deaths by over 80% (2007 and 2009 model years).
2. Respiratory hospital admissions associated with short-term O₃ concentrations: Compared to the current standard, changes in total risk estimated for a standard level of 65 ppb are similar to the changes in total risk estimated for total mortality (U.S. EPA, 2014, chapter 7).
3. Respiratory mortality associated with long-term O₃ concentrations: A standard level of 65 ppb reduces total risk, compared to the current standard. Across most urban case study areas, risks are estimated to decrease by up to approximately 10%. In one area (New York City), risks are estimated to decrease by up to approximately 24%. Risk reductions are estimated across all urban case study areas and in both model years evaluated, with larger reductions estimated for 2007 (i.e., the model year with generally higher O₃-associated risks).

Based on Figures 4-9 to 4-12 and the associated details described in the HREA (U.S. EPA 2014, chapter 7), we take note of the following for an O₃ standard level of 60 ppb:

1. Total mortality associated with short-term O₃ concentrations:
 - a. A standard level of 60 ppb is estimated to reduce total risk, compared to the current standard, in all urban case study areas. Across urban case study areas, risks are estimated to decrease by up to approximately 14%. Estimated risk reductions are larger for the model year with generally higher O₃-associated risks (2007).
 - b. When summed across urban case study areas, a standard level of 60 ppb is estimated to reduce O₃-associated deaths by approximately 15% (2007 model year) and 11% (2009 model year), compared to the current standard. For area-wide concentrations at or above 40 ppb, a standard level of 60 ppb is estimated to

²⁷ Because of the approach to adjusting air quality in New York (and Los Angeles), which differed from other urban case study areas (U.S. EPA, 2014, sections 4.3.3.1, 4.5), the HREA notes less overall confidence in results for these areas.

reduce O₃-associated deaths by almost 60% (2007 and 2009 model years). For area-wide concentrations at or above 60 ppb, a standard level of 60 ppb is estimated to reduce O₃-associated deaths by over 95% (2007 and 2009 model years).

2. Respiratory hospital admissions associated with short-term O₃ concentrations: Compared to the current standard, changes in total risk estimated for a standard level of 60 ppb are similar to the changes in total risk estimated for total mortality (U.S. EPA, 2014, chapter 7).
3. Respiratory mortality associated with long-term O₃ concentrations: A standard level of 60 ppb reduces total risk, compared to the current standard. Across urban case study areas, risks are estimated to decrease by up to approximately 17%. Risk reductions are estimated across all urban case study areas and in both model years evaluated, with larger reductions estimated for 2007 (i.e., the model year with generally higher O₃-associated risks).

In further considering these risk estimates, we take note of the associated uncertainties, as discussed in more detail in section 3.2.3.2 of this PA. In particular, these include (1) the national representativeness of urban case study areas in terms of the O₃ response to reductions in NO_x emissions; (2) the representativeness of risk changes based primarily on reductions in NO_x emissions versus changes that could be achieved with better-optimized emissions reduction strategies; (3) the shape of the concentration-response function at lower ambient concentrations, including the potential for a threshold in the association between long-term O₃ and respiratory mortality; (4) the presence of unexplained heterogeneity in effect estimates between locations; (5) the potential for exposure measurement errors; and (6) the possibility for reductions in risk associated with reductions in PM and/or NO₂ resulting from control of NO_x.

4.5 CASAC ADVICE AND PUBLIC COMMENTERS' VIEWS ON ALTERNATIVE STANDARDS

As discussed in section 3.3, staff recognizes that decisions regarding the weight to place on various types of evidence, exposure/risk information, and associated uncertainties reflect public health policy judgments that are ultimately left to the Administrator. To help inform those judgments with regard to the range of alternative primary O₃ standards appropriate for consideration, CASAC has provided advice to the Administrator based on their reviews of the O₃ ISA, HREA, and PA. This section summarizes the advice provided by CASAC regarding potential alternative standards, as well as the views expressed at the CASAC meetings by public commenters.

In the fall of 2011, rather than revising the O₃ NAAQS as part of the reconsideration process, EPA elected to coordinate further proceedings on the reconsideration rulemaking with the current ongoing periodic review. Accordingly, in this section we briefly describe CASAC advice from the reconsideration of the 2008 final decision on the level of the standard, as well as

CASAC advice received during the current review as it pertains to potential alternative standards.

Consistent with their advice in 2008, CASAC reiterated during the reconsideration its support for an 8-hour primary O₃ standard with a level ranging from 60 to 70 ppb, combined with the current indicator, averaging time, and form. Specifically, in response to EPA's solicitation of their advice during the reconsideration, the CASAC letter (Samet 2010) to the Administrator stated:

CASAC fully supports EPA's proposed range of 0.060 – 0.070 parts per million (ppm) for the 8-hour primary ozone standard. CASAC considers this range to be justified by the scientific evidence as presented in the Air Quality Criteria for Ozone and Related Photochemical Oxidants (March 2006) and Review of the National Ambient Air Quality Standards for Ozone: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper (July 2007).

Similarly, in response to EPA's request for additional advice on the reconsideration in 2011, CASAC reaffirmed their conclusion that “the evidence from controlled human and epidemiological studies strongly supports the selection of a new primary ozone standard within the 60 – 70 ppb range for an 8-hour averaging time” (Samet, 2011). CASAC further concluded that this range “would provide little margin of safety at its upper end” (Samet, 2011, p. 2).

In the current review of the Second Draft PA, as noted above, CASAC concurred with staff's conclusions that it is appropriate to consider retaining the current indicator (O₃), averaging time (8-hour average) and form (three-year average of the 4th highest maximum daily 8-hour average). With regard to level, CASAC stated the following (Frey, 2014, p. ii to iii):

The CASAC further concludes that there is adequate scientific evidence to recommend a range of levels for a revised primary ozone standard from 70 ppb to 60 ppb. The CASAC reached this conclusion based on the scientific evidence from clinical studies, epidemiologic studies, and animal toxicology studies, as summarized in the Integrated Science Assessment (ISA), the findings from the exposure and risk assessments as summarized in the HREA, and the interpretation of the implications of these sources of information as given in the Second Draft PA.

The CASAC acknowledges 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. The CASAC advises that, based on the scientific evidence, a level of 70 ppb provides little margin of safety for the protection of public health, particularly for sensitive subpopulations.

Thus, our policy advice is to set the level of the standard lower than 70 ppb within a range down to 60 ppb, taking into account your judgment regarding the desired

margin of safety to protect public health, and taking into account that lower levels will provide incrementally greater margins of safety.

The public commenters who expressed the view that the current primary O₃ standard is not adequate (section 3.3) also submitted comments that supported revising the level of the primary O₃ standard. Several of these commenters expressed the view that the level should be revised to the lower end of the range of 70 to 60 ppb, or in some cases to a level below 60 ppb. The basis for these commenters' views on the level of the standard is generally reflected in the rationale given by CASAC for their advice, and is discussed in section 3.3 of this PA. Public commenters who expressed the view that revision of the current standard is not necessary did not provide any provisional views on alternative levels that would be appropriate for consideration should the Administrator consider revisions to the standard. These views are also discussed in section 3.3 of this PA.

4.6 STAFF CONCLUSIONS ON ALTERNATIVE PRIMARY STANDARDS FOR CONSIDERATION

Staff's consideration of alternative primary O₃ standards builds upon our conclusion, discussed in section 3.4, that the overall body of evidence and exposure/risk information call into question the adequacy of public health protection afforded by the current standard, particularly for at-risk populations. We further conclude that it is appropriate in this review to consider alternative standards that would increase public health protection, compared to the current standard.

As discussed in sections 4.1 to 4.3 above, in the current review we conclude that it is appropriate for the Administrator to consider retaining O₃ as the indicator for the standard that protects against exposures to ambient O₃ and other photochemical oxidants (section 4.1), and to consider retaining the current averaging time (section 4.2) and form (section 4.3) for the primary O₃ standard. For a primary O₃ standard that is defined in terms of the current indicator, averaging time, and form, we reach the conclusion that, depending on the public health policy judgments made by the Administrator, the scientific evidence and exposure/risk information available in this review support considering alternative O₃ standard levels from 70 down to 60 ppb. The basis for this conclusion is discussed in detail in section 4.4 of this PA, and is summarized in this section.

Below, we summarize our approach to considering the scientific evidence and exposure/risk information, and the specific evidence and information that supports the range of levels from 70 to 60 ppb. In doing so, we focus particularly on the evidence and information as it relates to the upper (70 ppb), middle (65 ppb), and lower (60 ppb) portions of this range. Key exposure/risk information is summarized in Tables 4-4, and 4-5, and Figure 4-13.

Table 4-4 Summary of Estimated Exposures of Concern for Potential Alternative O₃ Standard Levels of 70, 65, and 60 ppb in Urban Case Study Areas²⁸

Benchmark Level	Alternative Standard Level (ppb)	Average % Children Exposed ²⁹	Number of Children (5 to 18 years) [Number of Asthmatic Children] ³⁰	Average % Reduction from Current Standard	% Children - Worst Year and Worst Area
One or more exposures of concern per season					
≥ 70 ppb	70	0.1-1.2	94,000 [10,000]	73	3.2
	65	0-0.2	14,000 [2,000]	95	0.5
	60	0 ³¹	1,400 [200] ³²	100	0.1
≥ 60 ppb	70	3.3-10.2	1,176,000 [126,000]	46	18.9
	65	0-4.2	392,000 [42,000]	80	9.5
	60	0-1.2	70,000 [8,000]	96	2.2
Two or more exposures of concern per season					
≥ 70 ppb	70	0-0.1	5,400 [600]	95	0.4
	65	0	300 [100]	100	0
	60	0	0 [0]	100	0
≥ 60 ppb	70	0.5-3.5	320,000 [35,000]	61	9.2
	65	0-0.8	67,000 [7,500]	92	2.8
	60	0-0.2	5,100 [700]	100	0.3

²⁸ As illustrated above in Figures 4-1 to 4-4, all alternative standard levels evaluated in the HREA were effective at limiting exposures of concern at or above 80 ppb. Therefore, Table 4-4 focuses on exposures of concern at or above the 70 and 60 ppb benchmark concentrations.

²⁹ Estimates for each urban case study area were averaged for the years evaluated in the HREA (2006 to 2010). Ranges reflect the ranges across urban case study areas.

³⁰ Numbers of children exposed in each urban case study area were averaged over the years 2006 to 2010. These averages were then summed across urban case study areas. Numbers are rounded to nearest thousand unless otherwise indicated.

³¹ Estimates smaller than 0.1% were rounded to zero.

³² As discussed in section 4.3.3 of the HREA, the model-based air quality adjustment approach used to estimate risks associated with the current and alternative standards was unable to estimate the distribution of ambient O₃ concentrations in New York City upon just meeting an alternative standard with a level of 60 ppb. Therefore, for the 60 ppb standard level the numbers of children and asthmatic children reflect all of the urban case study areas except New York.

Table 4-5 Summary of Estimated Lung Function Decrements for Potential Alternative O₃ Standard Levels of 70, 65, and 60 ppb in Urban Case Study Areas

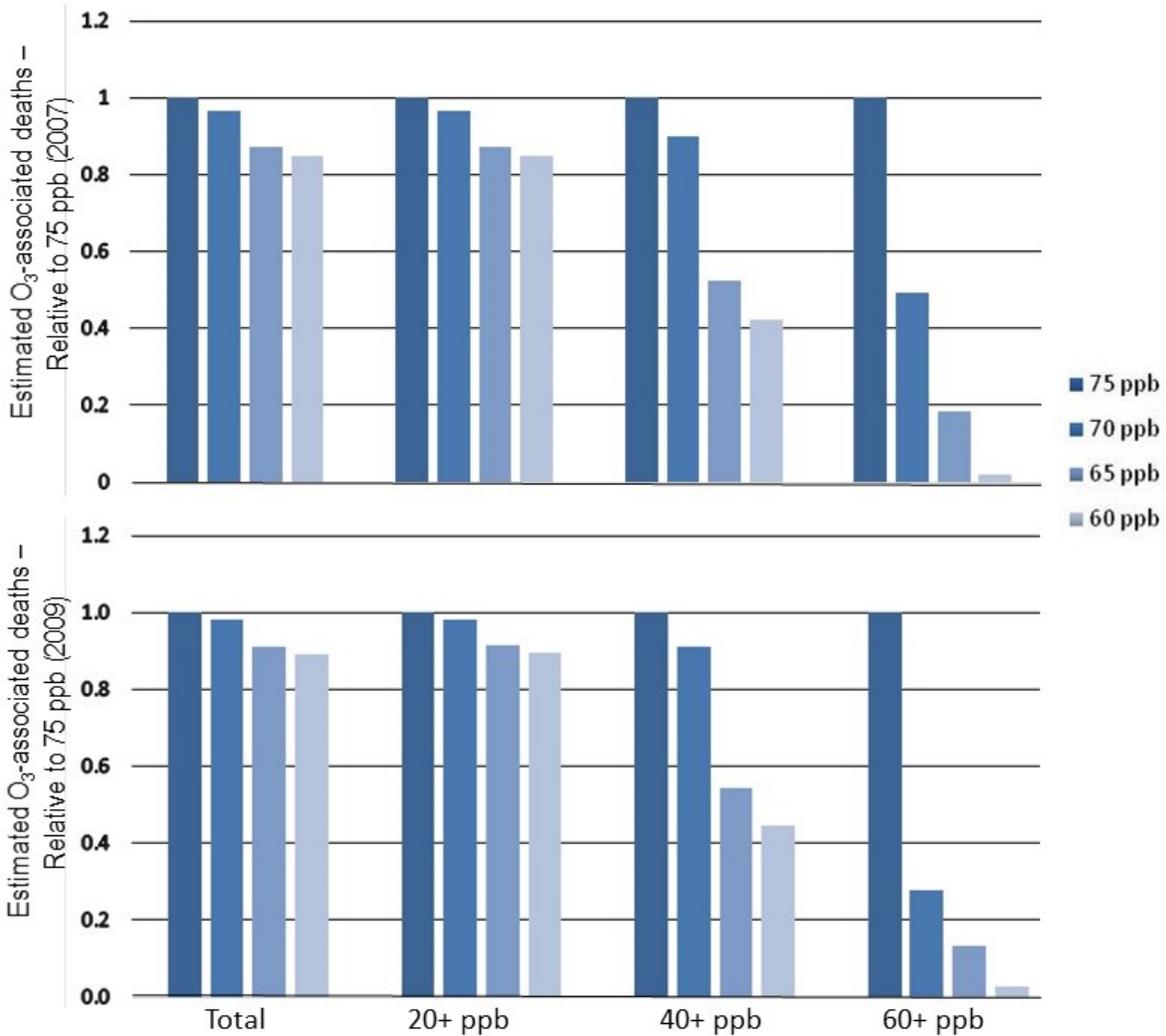
Lung Function Decrement	Alternative Standard Level	Average % Children ³³	Number of Children (5 to 18 years) [Number of Asthmatic Children] ³⁴	Average % Reduction from Current Standard	% Children Worst Year and Area
One or more decrements per season					
≥ 10%	70	11-17	2,527,000 [261,000]	15	20
	65	3-15	1,896,000 [191,000]	31	18
	60	5-11	1,404,000 [139,000] ³⁵	45	13
≥ 15%	70	2-4	562,000 [58,000]	26	5
	65	0-3	356,000 [36,000]	50	4
	60	1-2	225,000 [22,000]	67	3
≥ 20%	70	1-2	189,000 [20,000]	32	2.1
	65	0-1	106,000 [11,000]	59	1.4
	60	0-1	57,000 [6,000]	77	0.7
Two or more decrements per season					
≥ 10%	70	5.5-11	1,414,000 [145,000]	17	13
	65	1.3-8.8	1,023,000 [102,000]	37	11
	60	2.1-6.4	741,000 [73,000]	51	7.3
≥ 15%	70	0.9-2.4	276,000 [28,000]	29	3.1
	65	0.1-1.8	168,000 [17,000]	54	2.3
	60	0.2-1.0	101,000 [10,000]	71	1.4
≥ 20%	70	0.3-0.8	81,000 [8,000]	34	1.1
	65	0-0.5	43,000 [4,000]	66	0.8
	60	0-0.2	21,000 [2,000]	83	0.4

³³ Estimates in each urban case study area were averaged for the years evaluated in the HREA (2006 to 2010). Ranges reflect the ranges across urban case study areas.

³⁴ Numbers of children estimated to experience decrements in each study urban case study area were averaged over 2006 to 2010. These averages were then summed across urban case study areas. Numbers are rounded to nearest thousand unless otherwise indicated. As discussed above, for the 60 ppb standard level the numbers of children and asthmatic children included in Table 4-5 reflect all of the urban case study areas except New York.

³⁵As discussed in section 4.3.3 of the HREA, the model-based air quality adjustment approach used to estimate risks associated with the current and alternative standards was unable to estimate the distribution of ambient O₃ concentrations in New York City upon just meeting an alternative standard with a level of 60 ppb. Therefore, for the 60 ppb standard level the numbers of children and asthmatic children reflect all of the urban case study areas except New York.

Figure 4-13. Estimates of O₃-Associated Deaths Attributable to Full Distributions of 8-Hour Area-Wide O₃ Concentrations and to Concentrations at or above 20, 40, or 60³⁶ ppb O₃ - Deaths Summed Across Urban Case Study Areas and Expressed Relative to a Standard with a Level of 75 ppb



³⁶As discussed in section 4.3.3 of the HREA, the model-based air quality adjustment approach used to estimate risks associated with the current and alternative standards was unable to estimate the distribution of ambient O₃ concentrations in New York City upon just meeting an alternative standard with a level of 60 ppb. Therefore, the total number of deaths indicated for the 60 ppb standard level in Figure 4-10 reflects the 60 ppb estimates for all urban case study areas except New York City. For New York City, the estimated number of O₃-associated deaths for the 65 ppb standard level was assumed.

Summary of approach to reaching conclusions on alternative standard levels

In this PA, our approach to reaching conclusions on alternative standard levels focuses on the evidence from controlled human exposure and epidemiologic studies, as assessed in the ISA (U.S. EPA, 2013), and the exposure and health risk analyses presented in the HREA (U.S. EPA, 2014). This approach is discussed in detail in Chapter 1 (section 1.3), and is summarized below.

As an initial matter, we note that controlled human exposure studies provide the most certain evidence indicating the occurrence of health effects in humans following exposures to specific O₃ concentrations. Consistent with this, CASAC concluded 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, 2014, p. 5). As discussed above and in section 3.1.2.1, controlled human exposure studies have reported a variety of respiratory effects in healthy adults following exposures to O₃ concentrations of 60, 72,³⁷ or 80 ppb, and higher. The largest respiratory effects, and the broadest range of effects, have been studied and reported following exposures of healthy adults to 80 ppb O₃ or higher, with most exposure studies conducted at these higher concentrations. Exposures to O₃ concentrations of 80 ppb or higher have been reported to decrease lung function, increase airway inflammation, increase respiratory symptoms, result in airway hyperresponsiveness, and decrease lung host defenses in healthy adults.

Most of these effects have also been reported in healthy adults following exposures to O₃ concentrations below 80 ppb.³⁸ Exposures to O₃ concentrations of 72 ppb have been reported to decrease lung function and increase respiratory symptoms, a combination that meets the ATS criteria for an “adverse” response (section 3.1.3). Exposures to O₃ concentrations of 60 ppb have been demonstrated to decrease lung function, with decrements in some people large enough to be judged an abnormal response by ATS, and which CASAC has indicated could be adverse to people with lung disease.³⁹ In addition, as discussed in section 3.1.3, such a decrease in mean lung function meets the ATS criteria for an adverse response given that a downward shift in the distribution of FEV₁ would result in diminished reserve function, and therefore would increase risk from further environmental insult. Exposures to O₃ concentrations of 60 ppb have also been reported in one study (Kim et al., 2011) to increase airway inflammation, which provides a

³⁷ As noted above, for the 70 ppb exposure concentration Schelegle et al. (2009) reported that the actual mean exposure concentration was 72 ppb.

³⁸ Airway hyperresponsiveness and reductions in lung host defense have not been evaluated following exposures to O₃ concentrations below 80 ppb. The extent to which these respiratory effects occur following lower exposure concentrations is not clear from the available evidence, though we have no basis for concluding that an exposure concentration of 80 ppb reflects an effects threshold.

³⁹ In their advice to the Administrator based on the second draft PA, the CASAC indicated that “60 ppb is an appropriate exposure of concern for asthmatic children” (Frey, 2014).

mechanism by which O₃ may cause other more serious respiratory effects (e.g., asthma exacerbations).

Given the evidence for respiratory effects from controlled human exposure studies, we consider the extent to which standards with revised levels would be estimated to protect at-risk populations against exposures of concern to O₃ concentrations at or above the health benchmark concentrations of 60, 70, and 80 ppb (i.e., based on HREA estimates of one or more and two or more exposures of concern). In doing so, we note that, due to individual variability in responsiveness, only a subset of people who experience exposures at or above the three benchmark concentrations can be expected to experience associated health effects, and that available data are not sufficient to quantify that subset of people. We view the health effects evidence as a continuum with greater confidence and less uncertainty about the occurrence of adverse health effects at higher O₃ exposure concentrations, and less confidence and greater uncertainty as one considers lower exposure concentrations (discussed in more detail in section 3.2.2).

While there is greater uncertainty regarding the occurrence of adverse health effects at lower concentrations, we also note that the controlled human exposure studies that provided the basis for benchmark concentrations have not evaluated responses in populations at the greatest risk from exposures to O₃. Thus, the effects reported in healthy adults at each of the benchmark concentrations may underestimate effects in these at-risk groups. Compared to the healthy people included in most controlled human exposure studies, members of at-risk populations, including lifestages, (e.g., asthmatics, children) are at greater risk of experiencing adverse effects. In considering the health evidence within the context of drawing conclusions on potential alternative standard levels, we balance concerns about the potential for adverse health effects, especially in at-risk populations, with our increasing uncertainty regarding the likelihood of such effects following exposures to lower O₃ concentrations.

With respect to the lung function decrements that have been evaluated in controlled human exposure studies, we consider the extent to which standards with revised levels would be estimated to protect healthy and at-risk populations against O₃-induced lung function decrements large enough to be adverse in some people (based on quantitative risk estimates in the HREA). As discussed in section 3.1.3, although some experts would judge single occurrences of moderate responses to be a “nuisance,” especially for healthy individuals, a more general consensus view of the adversity of moderate lung function decrements emerges as the frequency of occurrence increases. Repeated occurrences of moderate responses, even in otherwise healthy individuals, may be considered to be adverse, since they could well set the stage for more serious illness (61 FR 65723). For the purpose of estimating potentially adverse lung function decrements in active, healthy people, in the 2008 review the CASAC panel indicated that a focus on the mid to upper

end of the range of moderate (i.e., FEV₁ decrements $\geq 15\%$) functional responses is appropriate. However, for children and adults with lung disease, FEV₁ decrements $\geq 10\%$ could lead to respiratory symptoms, would likely interfere with normal activities for many individuals, and therefore could be adverse. Large (i.e., FEV₁ decrements $\geq 20\%$) lung function decrements would likely interfere with normal activities for most people with lung disease and would increase the likelihood that they would seek medical attention. In the current review, CASAC judges that an FEV₁ decrement $\geq 15\%$ is an appropriate surrogate for adverse health outcomes in active healthy adults, while a decrement $\geq 10\%$ is a scientifically relevant surrogate for adverse health outcomes for people with asthma and lung disease (Frey, 2014). In reaching conclusions on alternative standard levels, we consider the extent to which standards with revised levels would be estimated to protect healthy and at-risk populations against one or more, and two or more, moderate (i.e., FEV₁ decrements $\geq 10\%$ and $\geq 15\%$) and large (i.e., FEV₁ decrements $\geq 20\%$) lung function decrements.

In evaluating the epidemiologic evidence within the context of drawing conclusions on potential alternative standard levels, we consider the extent to which available studies have reported associations with emergency department visits, hospital admissions, and/or mortality in locations that would likely have met potential alternative standards with levels below 75 ppb (based on analyses presented in section 4.4.1). In evaluating the epidemiologic evidence in this way, we consider both multicity and single-city studies, recognizing the strengths and limitations of each. Specifically, multicity studies evaluate large populations and provide greater statistical power than single-city studies; multicity studies reflect O₃-associated health impacts across a range of diverse locations, providing spatial coverage for different regions across the country and reflecting differences in exposure-related factors that could impact O₃ risks; and multicity studies afford a greater possibility of generalizing to the national population. In contrast, while single-city studies are more limited than multicity studies in terms of statistical power and geographic coverage, conclusions linking air quality in a specific area with health effect associations in that same area can be made with greater certainty for single-city studies (i.e., compared to multicity studies reporting only multicity effect estimates).

We also consider the epidemiologic evidence within the context of epidemiology-based risk estimates. Compared to the weight given to HREA estimates of exposures of concern and lung function risks (sections 4.4.2.1 and 4.4.2.2, above), and the weight given to the evidence (section 4.4.1), we place relatively less weight on epidemiologic-based risk estimates. In doing so, we note 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. Our 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 locations, the potential for exposure measurement errors, and uncertainty in the interpretation of the shape of concentration-response functions at lower O₃ concentrations (U.S. EPA, 2014, section 9.6). The HREA also concludes that lower confidence should be placed in the results of the assessment of respiratory mortality risks associated with long-term O₃ exposures, primarily because that analysis is based on only one study (even though that study is well-designed) and because of the uncertainty in that study about the existence and level of a potential threshold in the concentration-response function (U.S. EPA, 2014, section 9.6).

In considering the epidemiology-based risk estimates, we focus on the extent to which potential alternative O₃ standards with levels below 75 ppb are estimated to reduce the risk of O₃-associated mortality (based on the HREA results summarized in section 4.4.2.3).⁴⁰ As discussed in section 3.4 for the current standard, we consider estimates of total risk (i.e., based on the full distributions of ambient O₃ concentrations) and estimates of risk associated with O₃ concentrations in the upper portions of ambient distributions. A focus on estimates of total risks would place greater weight on the possibility that concentration-response relationships remain linear over the entire distribution of ambient O₃ concentrations, and thus on the potential for mortality and morbidity to be affected by changes in relatively low O₃ concentrations. A focus on risks associated with O₃ concentrations in the upper portions of the ambient distribution would place greater weight on the uncertainty associated with the shapes of concentration-response curves for O₃ concentrations in the lower portions of the distribution. Given that both types of risk estimates could reasonably inform a decision on standard level, depending on the weight placed on uncertainties in the occurrence and the estimation of O₃-attributable effects at relatively low O₃ concentrations, in reaching conclusions we consider what both types of estimates indicate with regard to potential alternative levels.

Staff conclusions on the range of levels appropriate for consideration

Using the approach discussed above to consider the scientific evidence and exposure/risk information, we reach the conclusion that it is appropriate for the Administrator to consider alternative primary O₃ standard levels from 70 to 60 ppb. The basis for this conclusion is discussed in detail in sections 4.4.1 and 4.4.2 above, and is summarized below.

With regard to controlled human exposure studies, we consider the lowest O₃ exposure concentrations at which various effects have been evaluated and statistically significant effects reported. We also consider the potential for reported effects to be adverse, including in at-risk populations and lifestages. As discussed in section 3.1.2.1, controlled human exposure studies

⁴⁰ Differences in estimated respiratory morbidity risks between potential alternative standard levels are similar to the differences estimated for total mortality associated with short-term O₃ concentrations.

provide evidence of respiratory symptoms combined with lung function decrements (an “adverse” response based on ATS criteria) in healthy adults following exposures to O₃ concentrations as low as 72 ppb, and evidence of potentially adverse lung function decrements and airway inflammation following exposures to O₃ concentrations as low as 60 ppb. Although some studies show that respiratory symptoms also develop during exposures to 60 ppb O₃, the increase in symptoms has not been reported to reach statistical significance by the end of the 6.6 hour exposure period (Adams 2006; Schelegle et al. 2009). Thus, while significant increases in respiratory symptoms combined with lung function decrements have not been reported following exposures to 60 ppb O₃, this combination of effects is likely to occur to some degree in healthy adults with 6.6-hour exposures to concentrations below 72 ppb, and also are more likely to occur with longer (i.e., 8-hour) exposures.⁴¹

With regard to the lowest exposure concentration shown to cause respiratory effects (i.e., 60 ppb), we note that most controlled human exposure studies have not evaluated O₃ concentrations below 60 ppb. Therefore, 60 ppb does not necessarily reflect an exposure concentration below which effects such as lung function decrements and airway inflammation no longer occur. This is particularly the case given that controlled human exposure studies were conducted in healthy adults, while people with asthma, including asthmatic children, are likely to be more sensitive to O₃-induced respiratory effects. In support of this, some epidemiologic panel studies, which can include members of at-risk groups such as children and outdoor workers, have found respiratory effects at ambient concentrations lower than 60 ppb (section 3.1.2.1).

With regard to other O₃-induced effects, we note that airway hyperresponsiveness and impaired lung host defense capabilities have been reported in healthy adults engaged in moderate exertion following exposures to O₃ concentrations as low as 80 ppb, the lowest concentration evaluated for these effects. As discussed in section 3.1.2.1, these physiological effects have been linked to aggravation of asthma and increased susceptibility to respiratory infection, potentially leading to increased medication use, increased school and work absences, increased visits to doctors’ offices and emergency departments, and increased hospital admissions. These are all indicators of adverse O₃-related morbidity effects, which are consistent with, and provide plausibility for, the adverse morbidity effects and mortality effects observed in epidemiologic studies.

⁴¹ In addition, CASAC observed that, “adverse health effects in young healthy adults occur with exposures to 72 ppb of ozone for 6.6 hours” and that “It is the judgment of CASAC that if subjects had been exposed to ozone using the 8-hour averaging period used in the standard, adverse effects could have occurred at [a] 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, 2014, p. 5).

Based on consideration of the above evidence, we conclude that available controlled human exposure studies support a level no higher than 70 ppb as the upper end of the range for consideration in the current review. In reaching this conclusion, we note that 70 ppb is just below the O₃ exposure concentration reported to result in lung function decrements and respiratory symptoms in healthy adults (i.e., 72 ppb), a combination of effects that meet ATS criteria for an adverse response. In addition, while 70 ppb is well below the 80 ppb exposure concentration shown to cause potentially adverse respiratory effects such as airway hyperresponsiveness and impaired host-defense capabilities, these effects have not been evaluated at exposure concentrations below 80 ppb and there is no reason to believe that 80 ppb represents a threshold for such effects.

We further conclude that the evidence from controlled human exposure studies⁴² supports considering alternative O₃ standard levels at least as low as 60 ppb. Potentially adverse lung function decrements and pulmonary inflammation have been demonstrated to occur in healthy adults at 60 ppb. Thus, 60 ppb is a short-term exposure concentration that may be reasonably concluded to elicit adverse effects in at-risk groups. Pulmonary inflammation, particularly if experienced repeatedly, provides a mechanism by which O₃ may cause other more serious respiratory morbidity effects (e.g., asthma exacerbations) and possibly extrapulmonary effects. As discussed in section 3.1.2.1, the physiological effects reported in controlled human exposure studies down to 60 ppb O₃ have been linked to aggravation of asthma and increased susceptibility to respiratory infection, potentially leading to increased medication use, increased school and work absences, increased visits to doctors' offices and emergency departments, and increased hospital admissions.

We further note that the range of alternative levels from 70 to 60 ppb is supported by evidence from epidemiologic studies and by exposure and risk estimates from the HREA. This evidence and exposure/risk information indicate that a level from anywhere in the range of 70 to 60 ppb would be expected to result in important public health improvements over the current standard. In particular, compared to the current standard a revised standard with a level from 70 to 60 ppb would be expected to (1) more effectively maintain short- and long-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard; (2) reduce the occurrence of exposures of concern to O₃ concentrations that result in respiratory effects in healthy adults (at or above 60, 70, and 80 ppb); (3) reduce the occurrence of moderate-to-large O₃-induced lung function decrements; and (4) reduce the risk of O₃-associated mortality and

⁴² As discussed in sections 3.1.2.1 and 4.4.1 above, panel studies also provide supporting evidence for these conclusions.

morbidity, particularly the risk associated with the upper portions of the distributions of ambient O₃ concentrations.

In reaching a conclusion on whether it is appropriate to consider alternative standard levels below 60 ppb, we note the following:

- While controlled human exposure studies provide evidence for O₃-induced respiratory effects following exposures to O₃ concentrations as low as 60 ppb, they do not provide evidence for adverse effects following exposures to lower concentrations. On this issue, CASAC concurred that 60 ppb O₃ is an appropriate and justifiable scientifically based lower bound for a revised primary standard, based upon 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 (Adams 2006; Schelegle et al., 2009; Brown et al. 2008; Kim et al., 2011), with limited evidence of adverse effects below 60 ppb” (Frey, 2014, p. 7).
- Based on the HREA results, meeting an O₃ standard with a level of 60 ppb would be expected to almost eliminate exposures of concern to O₃ concentrations at or above 60 ppb. To the extent lower exposure concentrations may result in adverse health effects in some people, a standard level of 60 ppb would be expected to also reduce exposures to O₃ concentrations below 60 ppb.
- U.S. and Canadian epidemiologic studies have not reported O₃ health effect associations based primarily on study locations likely to have met a standard with a level of 60 ppb.
- In all of the urban case study areas evaluated, a standard with a level of 60 ppb would be expected to maintain long-term O₃ concentrations below those where a key study indicates the most confidence in a linear concentration-response relationship with respiratory mortality.

Beyond the above considerations, we also note the HREA estimates indicating that meeting an O₃ standard with a level of 60 ppb would result in important reductions in the risk of O₃-induced lung function decrements and O₃-associated mortality and morbidity. Although some risk is estimated to remain based on these metrics, even with a level of 60 ppb, we have decreasing confidence in further public health improvements with levels below 60 ppb. We reach this conclusion because, as noted above, at a level of 60 ppb virtually no one in the population would be expected to experience exposures to O₃ concentrations at or above 60 ppb under conditions demonstrated in controlled human exposure studies to result in respiratory effects, and because epidemiologic studies have not reported O₃ health effect associations based primarily on study locations likely to have met a standard with a level of 60 ppb. Given all of the above considerations we conclude that, compared to standards with levels from 70 to 60 ppb, the extent to which standards with levels below 60 ppb could result in further public health improvements

becomes notably less certain. Therefore, we conclude that it is not appropriate in this review to consider standard levels below 60 ppb.

The range of levels from 70 to 60 ppb corresponds to the range of levels recommended for consideration by CASAC, based on the available evidence and information (Frey, 2014). While CASAC further offered the “policy advice” to set the level below 70 ppb, based on margin of safety considerations, the Committee acknowledged 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, 2014). Therefore, we note that our conclusions on the appropriate range for alternative primary O₃ standard levels are consistent with CASAC conclusions that the scientific evidence and exposure/risk information supports consideration of levels from 70 to 60 ppb, and that the ultimate identification of a standard that protects public health with an adequate margin of safety will reflect policy judgments that are explicitly reserved for the Administrator (section 1.2.1).

The following sections summarize the specific scientific evidence and exposure/risk information as they relate to revised O₃ standards with levels from the upper (70 ppb), middle (65 ppb), and lower (60 ppb) portions of the range of 70 to 60 ppb.

O₃ standard level of 70 ppb

A level of 70 ppb is below 80 ppb, an O₃ exposure concentration that has been reported to elicit a range of respiratory effects that includes airway hyperresponsiveness and decreased lung host defense, in addition to lung function decrements, airway inflammation, and respiratory symptoms. A level of 70 ppb is also below the lowest exposure concentration at which the combined occurrence of respiratory symptoms and lung function decrements have been reported (i.e., 72 ppb), a combination judged adverse by the ATS (section 3.1.3). A level of 70 ppb is above the lowest exposure concentration demonstrated to result in lung function decrements large enough to be judged an abnormal response by ATS and above the lowest exposure concentration demonstrated to result in pulmonary inflammation (i.e., 60 ppb).

Compared to the current standard, the HREA estimates that a revised O₃ standard with a level of 70 ppb would reduce exposures of concern to O₃ concentrations of 60, 70, and 80 ppb in urban case study areas, with such a standard level estimated to be most effective at limiting exposures at or above the higher health benchmark concentrations and at limiting multiple occurrences of such exposures. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow only up to about 1% of children (i.e., ages 5 to 18) to experience exposures of concern at or above 70 ppb (73% reduction, compared to current standard), and far less than 1% to experience two or more such exposures (95% reduction, compared to current standard). In the worst-case location and year (i.e., location and year with the largest exposure

estimate), about 3% of children are estimated to experience one or more exposures of concern at or above 70 ppb, and less than 1% are estimated to experience two or more. A standard with a level of 70 ppb is estimated to allow far less than 1% of children to experience exposures of concern at or above the 80 ppb benchmark concentration, even in the worst-case year (Table 4-4).⁴³

An O₃ standard with a level of 70 ppb is estimated to allow about 3 to 10% of children, including asthmatic children, to experience one or more exposures of concern at or above 60 ppb in a single O₃ season. As noted above, CASAC advised EPA that 60 ppb is an appropriate exposure of concern with respect to adverse effects on people with asthma, including children (Frey, 2014, p. 6, 8). Compared to the current standard, this reflects about a 46% reduction, on average across the urban case study areas. A standard with a level of 70 ppb is estimated to allow about 1% to 4% of children to experience two or more exposures of concern at or above 60 ppb. In the worst-case location and year, a standard set at 70 ppb is estimated to allow about 19% of children to experience one or more exposures of concern at or above 60 ppb, and 9% to experience two or more such exposures (Table 4-4).

Compared to the current standard, the HREA estimates that a revised O₃ standard with a level of 70 ppb would also reduce O₃-induced lung function decrements in children. A level of 70 ppb is estimated to be most effective at limiting the occurrences of moderate and large lung function decrements (i.e., FEV₁ decrements $\geq 15\%$ and $\geq 20\%$, respectively), and at limiting multiple occurrences of O₃-induced decrements. On average over the years 2006 to 2010, a standard with a level of 70 ppb is estimated to allow about 2 to 4% of children in the urban case study areas to experience one or more moderate O₃-induced lung function decrements (i.e., FEV₁ decrement $\geq 15\%$), which would be of concern for healthy people, and about 1 to 2.5% of children to experience two or more such decrements (approximately 30% reduction, compared to the current standard). In the worst-case location and year, up to 5% of children are estimated to experience one or more O₃-induced lung function decrements $\geq 15\%$, and up to 3% are estimated to experience two or more such decrements. A standard set at 70 ppb is estimated to allow about 2% or fewer children to experience large O₃-induced lung function decrements (i.e., FEV₁ decrement $\geq 20\%$), and to allow about 1% or fewer children to experience two or more such decrements, even in the worst-case years and locations (Table 4-5).

On average over the years 2006 to 2010, an O₃ standard set at 70 ppb is estimated to allow about 11 to 17% of children in the urban case study areas to experience one or more moderate O₃-induced lung function decrements (i.e., FEV₁ decrement $\geq 10\%$), which could be

⁴³ As noted above, due to interindividual variability, children (or adults) exposed at these levels will not necessarily experience health effects; the information available for some health effects is not sufficient to quantify the numbers of children in the urban case study areas who might experience these effects.

adverse for people with lung disease. This reflects an average reduction of about 15%, compared to the current standard. A standard with a level of 70 ppb is also estimated to allow about 6 to 11% of children to experience two or more such decrements (17% reduction, compared to current standard). In the worst-case location and year, a standard set at 70 ppb is estimated to allow about 20% of children in the urban case study areas to experience one or more O₃-induced lung function decrements \geq 10%, and 13% to experience two or more such decrements (Table 4-5).

Compared to the current standard, a revised standard with a level of 70 ppb would also more effectively maintain short-term ambient O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard. In particular, the single-city study by Mar and Koenig (2009) reported positive and statistically significant associations with respiratory emergency department visits in children and adults in a location that likely would have met the current O₃ standard over the entire study period but violated a revised standard with a level of 70 ppb or below. Thus, none of the single-city studies evaluated in section 4.4.1 provide evidence for O₃ health effect associations in locations meeting a standard with a level of 70 ppb or below. While this analysis does not provide information on the extent to which the reported O₃-associated emergency department visits would persist upon meeting an O₃ standard with a level of 70 ppb, or on the extent to which standard levels below 70 ppb could further reduce the incidence of such emergency department visits, it suggests that a revised O₃ standard with a level at or below 70 ppb would require reductions in the ambient O₃ concentrations that provided the basis for the health effect associations reported by Mar and Koenig.

As discussed above, compared to single-city studies, there is greater uncertainty in linking air quality concentrations from individual study cities to multicity effect estimates. With regard to multicity studies, we note that Dales et al. (2006) reported significant associations with respiratory hospital admissions based on air quality in 11 Canadian cities, most of which would likely have met the current standard over the entire study period but violated a revised standard with a level of 70 ppb or below over at least part of that period (Table 4-1). This analysis suggests that while the current standard would allow the ambient O₃ concentrations in most of the study locations that provided the basis for the association with hospital admissions, a revised O₃ standard with a level at or below 70 ppb would require reductions in those ambient O₃ concentrations. As with the study by Mar and Koenig, this analysis does not provide information on the extent to which the reported O₃-associated hospital admissions would persist upon

meeting an O₃ standard with a level of 70 ppb, or on the extent to which standard levels below 70 ppb could further reduce the incidence of such hospital admissions.⁴⁴

With regard to long-term O₃ concentrations, we evaluated the “long-term” O₃ metrics reported to be associated with mortality or morbidity in recent epidemiologic studies (e.g., seasonal averages of 1-hour or 8-hour daily max concentrations). Compared to the current standard, a revised standard with a level of 70 ppb would be expected to reduce the risk of respiratory mortality associated with long-term O₃ concentrations, based on information from the study by Jerrett et al. (2009), though we note the HREA conclusion, discussed above, that lower confidence should be placed in respiratory mortality risk estimates based on this study (U.S. EPA, 2014, section 9.6). In addition, a standard with a level of 70 ppb would be expected to more effectively maintain long-term O₃ concentrations below those where the study by Jerrett et al. (2009) indicates the most confidence in the reported association with respiratory mortality. Specifically, air quality analyses indicate this to be the case in 9 out of the 12 urban case study areas for a level of 70 ppb, compared to 6 out of 12 areas for the current standard. Finally, a revised standard with a level of 70 ppb would be expected to reduce long-term O₃ concentrations based on the types of metrics that have been reported in recent epidemiologic studies to be associated with respiratory morbidity (i.e., seasonal averages of daily maximum 8-hour concentrations).

In further considering the potential implications of epidemiology studies for alternative standard levels, we note estimates of total mortality associated with short-term O₃ concentrations.⁴⁵ As discussed above, we consider estimates of total risk (i.e., based on the full distributions of ambient O₃ concentrations) and estimates of risk associated with O₃ concentrations in the upper portions of ambient distributions. With regard to total risk we note that, when summed across urban case study areas, a standard with a level of 70 ppb is estimated to reduce the number of deaths associated with short-term O₃ concentrations by about 4% (2007) and 2% (2009), compared to the current standard.⁴⁶ Based on a national modeling analysis, the majority of the U.S. population would be expected to experience reductions in such risks upon reducing precursor emissions.

⁴⁴ In addition, for the other multicity studies identified in Table 4-1 (Cakmak et al., 2006; Stieb et al., 2009; Katsouyanni et al., 2009), and for the study by Bell et al. (2006) (for the 30 ppb cut point) (Table 4-2), the majority of study locations would likely have met a standard with a level of 70 ppb.

⁴⁵ As discussed above, compared to the weight given to the evidence and to HREA estimates of exposures of concern and lung function risks, we place relatively less weight on epidemiologic-based risk estimates.

⁴⁶ A standard with a level of 70 ppb is also estimated to reduce respiratory mortality associated with long-term O₃ concentrations in urban case study areas. However, given uncertainties associated with these risk estimates, as discussed above, we give them limited weight.

Compared to the total risk estimates noted above, an O₃ standard with a level of 70 ppb is estimated to be more effective at reducing the number of deaths associated with short-term O₃ concentrations at the upper ends of ambient distributions. Specifically, for area-wide O₃ concentrations at or above 40 ppb, a standard with a level of 70 ppb is estimated to reduce the number of deaths associated with short-term O₃ concentrations by about 10% compared to the current standard. In addition, for area-wide concentrations at or above 60 ppb, a standard with a level of 70 ppb is estimated to reduce O₃-associated deaths by about 50% to 70% (Figure 4-13).

As discussed above, CASAC concluded that there is adequate scientific evidence to consider a range of levels for a primary standard that includes an upper end at 70 ppb. However, CASAC differentiated its advice from the conclusions in the second draft PA by also advising that a level of 70 ppb would provide little margin of safety for protection of public health, particularly for sensitive subpopulations (Frey, 2014, p. 8). In particular, CASAC stated that:

At 70 ppb, there is substantial scientific certainty of a variety of adverse effects, including decrease in lung function, increase in respiratory symptoms, and increase in airway inflammation. Although a level of 70 ppb is more protective of public health than the current standard, it may not meet the statutory requirement to protect public health with an adequate margin of safety (Frey, 2014, p.8).

However, the committee also acknowledged 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, 2014).

In summary, compared to the current standard, a revised O₃ standard with a level of 70 ppb would be expected to (1) reduce the occurrence of exposures of concern to O₃ concentrations that result in respiratory effects in healthy adults (at or above 60 and 70 ppb) by about 45 to 95%, almost eliminating the occurrence of multiple exposures at or above 70 ppb; (2) reduce the occurrence of moderate-to-large O₃-induced lung function decrements (FEV₁ decrements \geq 10, 15, 20%) by about 15 to 35%, most effectively limiting the occurrence of multiple decrements and decrements \geq 15, 20%; (3) more effectively maintain short- and long-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard;⁴⁷ and (4) reduce the risk of O₃-associated mortality and morbidity, particularly the risk associated with the upper portions of the distributions of ambient O₃ concentrations.

⁴⁷ Though epidemiologic studies also provide evidence for O₃ health effect associations in locations likely to have met a standard with a level of 70 ppb, as discussed below for lower standard levels.

O₃ standard level of 65 ppb

Next, we consider a standard with a level of 65 ppb. A level of 65 ppb is well below 80 ppb, an O₃ exposure concentration that has been reported to elicit a range of respiratory effects that includes airway hyperresponsiveness and decreased lung host defense, in addition to lung function decrements, airway inflammation, and respiratory symptoms. A standard level of 65 ppb is also below the lowest exposure concentration at which the combined occurrence of respiratory symptoms and lung function decrements has been reported (i.e., 72 ppb), a combination judged adverse by the ATS (section 3.1.3). A level of 65 ppb is above the lowest exposure concentration demonstrated to result in lung function decrements large enough to be judged an abnormal response by ATS, where statistically significant changes in group mean responses would be judged to be adverse by ATS, and which the CASAC has indicated could be adverse in people with lung disease (i.e., 60 ppb). A level of 65 ppb is also above the lowest exposure concentration at which pulmonary inflammation has been reported in healthy adults (i.e., 60 ppb).

Compared to the current standard and a revised standard with a level of 70 ppb, the HREA estimates that a standard with a level of 65 ppb would reduce exposures of concern to the range of O₃ benchmark concentrations analyzed (i.e., 60, 70, and 80 ppb). The HREA estimates that meeting a standard with a level of 65 ppb would eliminate exposures of concern at or above 80 ppb in the urban case study areas. Such a standard is estimated to allow far less than 1% of children in the urban case study areas to experience one or more exposures of concern at or above the 70 ppb benchmark level, even in the worst-case years and locations, and is estimated to eliminate the occurrence of two or more exposures at or above 70 ppb (Table 4-4).

In addition, on average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow between 0 and about 4% of children (including asthmatic children) in urban case study areas to experience exposures of concern at or above 60 ppb, which CASAC has indicated is an appropriate exposure of concern for people with asthma, including children. This reflects an 80% reduction (on average across areas), relative to the current standard. A standard with a level of 65 ppb is estimated to allow less than 1% of children to experience two or more exposures of concern at or above 60 ppb (> 90% reduction, compared to current standard). In the worst-case location and year, about 10% of children are estimated to experience one or more exposures of concern at or above 60 ppb, with about 3% estimated to experience two or more such exposures (Table 4-4).

Compared to the current standard and a revised standard with a level of 70 ppb, the HREA estimates that a standard with a level of 65 ppb would also reduce the occurrence of O₃-induced lung function decrements. A level of 65 ppb is estimated to allow about 4% or less of children to experience moderate O₃-induced FEV₁ decrements $\geq 15\%$ (50% reduction, compared

to current standard), even considering the worst-case location and year. Such a standard is estimated to allow about 2% or less of children to experience two or more such decrements. A standard set at 65 ppb is estimated to allow about 1% or less of children to experience large O₃-induced lung function decrements (i.e., FEV₁ decrement \geq 20%), even in the worst-case year and location (Table 4-5).

On average over the years 2006 to 2010, a standard with a level of 65 ppb is estimated to allow about 3 to 15% of children to experience one or more moderate O₃-induced lung function decrements (i.e., FEV₁ decrement \geq 10%), which CASAC has indicated could be adverse for people with lung disease. This reflects an average reduction of about 30%, relative to the current standard. A standard with a level of 65 ppb is also estimated to allow about 1 to 9% of children in the urban case study areas to experience two or more such decrements (37% reduction, compared to current standard). In the worst-case location and year, a standard set at 65 ppb is estimated to allow up to about 18% of these children to experience one or more moderate O₃-induced lung function decrements \geq 10%, and up to 11% to experience two or more such decrements (Table 4-5).

With regard to O₃ epidemiologic studies we note that, compared to a standard with a level of 70 ppb, a revised standard with a level of 65 ppb would more effectively maintain short-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard. In particular, Katsouyanni et al. (2009) reported statistically significant associations with mortality based on air quality in 12 Canadian cities, most of which would likely have met a standard with a level of 70 ppb over the entire study period but violated a revised standard with a level of 65 ppb or below over at least part of that period (Table 4-1). This analysis suggests that while the current standard or a standard with a level of 70 ppb would allow the ambient O₃ concentrations in most of the study locations that provided the basis for the association with mortality in this study, a revised O₃ standard with a level at or below 65 ppb would require reductions in those ambient O₃ concentrations. As discussed above for a level of 70 ppb, this analysis does not provide information on the extent to which O₃-associated mortality would persist upon meeting an O₃ standard with a level of 65 ppb, or on the extent to which standard levels below 65 ppb could further reduce the incidence of this mortality.⁴⁸

With regard to long-term O₃ concentrations, as for 70 ppb (above) we evaluate the “long-term” O₃ metrics reported to be associated with mortality or morbidity in recent epidemiologic

⁴⁸ For the other multicity studies identified in Table 4-1 (Cakmak et al., 2006; Stieb et al., 2009; Katsouyanni et al., 2009 (for hospital admissions)), and for the study by Bell et al. (2006) (for the 30 ppb cut point) (Table 4-2), the majority of study locations would have met a standard with a level of 65 ppb.

studies (e.g., seasonal averages of 1-hour or 8-hour daily max concentrations). Compared to the current standard or a revised O₃ standard with a level of 70 ppb, a revised standard with a level of 65 ppb would be expected to further reduce the risk of respiratory mortality associated with long-term O₃ concentrations, based on information from the study by Jerrett et al. (2009).⁴⁹ In addition, a standard with a level of 65 ppb would be expected to more effectively maintain long-term O₃ concentrations below those where the study by Jerrett et al. (2009) indicates the most confidence in the reported association with respiratory mortality. Specifically, air quality analyses indicate this to be the case in 10 out of the 12 urban case study areas for a level of 65 ppb, compared to 6 out of 12 areas for the current standard and 9 out of 12 for a standard with a level of 70 ppb (Table 4-3). Finally, a revised standard with a level of 65 ppb would be expected to further reduce long-term O₃ concentrations based on the types of metrics that have been reported in recent epidemiologic studies to be associated with respiratory morbidity (i.e., seasonal averages of daily maximum 8-hour concentrations).

In further considering the potential implications of epidemiology studies for alternative standard levels, we note estimates of total mortality associated with short-term O₃.⁵⁰ As discussed above, we consider estimates of total risk (i.e., based on the full distributions of ambient O₃ concentrations) and estimates of risk associated with O₃ concentrations in the upper portions of ambient distributions. With regard to total risk we note that, when summed across urban case study areas, a standard with a level of 65 ppb is estimated to reduce the number of deaths associated with short-term O₃ exposures by about 13% (2007) and 9% (2009), compared to the current standard.⁵¹ For area-wide concentrations at or above 40 ppb, a standard level of 65 ppb is estimated to reduce O₃-associated deaths by almost 50% compared to the current standard, when summed across cities. For area-wide concentrations at or above 60 ppb, a standard level of 65 ppb is estimated to reduce O₃-associated deaths by more than 80% (Figure 4-13).

As discussed above, although CASAC concluded that the scientific evidence supports considering standard levels as high as 70 ppb, it also concluded that a level of 70 ppb would provide little margin of safety (Frey, 2014, p. 8). In support of its policy advice that the level should be set below 70 ppb, CASAC noted that an alternative standard with a level of 65 ppb would further reduce, though not eliminate, the frequency of lung function decrements $\geq 15\%$

⁴⁹ Though as discussed above, we note the lower confidence we place in these risk results (U.S. EPA, 2014a, section 9.6).

⁵⁰ As discussed above, compared to the weight given to the evidence and to HREA estimates of exposures of concern and lung function risks, we place relatively less weight on epidemiologic-based risk estimates.

⁵¹ A standard with a level of 65 ppb is also estimated to reduce respiratory mortality associated with long-term O₃ concentrations in urban case study areas. However, given uncertainties associated with these risk estimates, as discussed above, we give them limited weight.

and would lead to lower frequency of short-term premature mortality (i.e., compared to a standard with a level of 70 ppb) (Frey, 2014, p. 8).

In summary, compared to a standard with a level of 70 ppb, a revised standard with a level of 65 ppb would be expected to (1) further reduce the occurrence of exposures of concern (by about 80 to 100% compared to the current standard), decreasing exposures at or above 60 ppb and almost eliminating exposures at or above 70 and 80 ppb; (2) further reduce the occurrence of FEV₁ decrements ≥ 10 , 15, and 20% (by about 30 to 65%, compared to the current standard); (3) more effectively maintain short- and long-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard;⁵² and (4) further reduce the risk of O₃-associated mortality and morbidity, particularly the risk associated with the upper portion of the distribution of ambient O₃ concentrations.

O₃ standard level of 60 ppb

We next consider a standard with a level of 60 ppb. A level of 60 ppb is well below the O₃ exposure concentration that has been reported to elicit a wide range of potentially adverse respiratory effects in healthy adults (i.e., 80 ppb). A level of 60 ppb is also below the concentration where the combined occurrence of respiratory symptoms and lung function decrements was observed, a combination judged adverse by the ATS (i.e., 72 ppb, discussed in section 3.1.3). A level of 60 ppb corresponds to the lowest exposure concentration demonstrated to result in lung function decrements that are large enough to be judged an abnormal response by ATS, that meet ATS criteria for adversity based on a downward shift in the distribution of FEV₁, and that the CASAC indicated could be adverse in people with lung disease. A level of 60 ppb also corresponds to the lowest exposure concentration at which pulmonary inflammation has been reported in controlled human exposure studies.

Based on the HREA analyses of O₃ exposures of concern, a standard with a level of 60 ppb is estimated to eliminate exposures of concern at or above the 70 and 80 ppb benchmark concentrations and to be more effective than the higher standard levels at limiting exposures of concern at or above 60 ppb. On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to allow between 0 and about 1% of children, including asthmatic children, in urban case study areas to experience exposures of concern at or above 60 ppb, which CASAC indicated is an appropriate exposure of concern for asthmatic children. This reflects a 96% reduction (on average across areas), compared to the current standard. A standard with a level of

⁵²Though epidemiologic studies also provide evidence for O₃ health effect associations in locations likely to have met a standard with a level of 65 ppb, as discussed below for a level of 60 ppb.

60 ppb is estimated to allow virtually no children to experience two or more exposures of concern at or above 60 ppb. In the worst-case location and year, about 2% of children are estimated to experience exposures of concern at or above 60 ppb, with far less than 1% estimated to experience two or more such exposures (Table 4-4).

Based on the HREA analyses of O₃-induced lung function decrements, a standard with a level of 60 ppb would be expected to be more effective than a level of 70 or 65 ppb at limiting the occurrence of O₃-induced lung function decrements. A standard with a level of 60 ppb is estimated to allow about 2% or less of children in the urban case study areas to experience one or more moderate O₃-induced FEV₁ decrements \geq 15% (almost 70% reduction, compared to current standard), and about 1% or less to experience two or more such decrements (3% in the location and year with the largest estimates). A standard set at 60 ppb is estimated to allow about 1% or less of children to experience large O₃-induced lung function decrements (i.e., FEV₁ decrement \geq 20%), even in the worst-case locations and year (Table 4-5).

On average over the years 2006 to 2010, a standard with a level of 60 ppb is estimated to allow about 5 to 11% of children in the urban case study areas to experience one or more moderate O₃-induced lung function decrements that CASAC indicated could be adverse for people with lung disease (i.e., FEV₁ decrements \geq 10%). This reflects an average reduction of about 45%, compared to current standard. A standard with a level of 60 ppb is also estimated to allow about 2 to 6% of children in these areas to experience two or more such decrements (51% reduction, compared to current standard). In the worst-case location and year, a standard set at 60 ppb is estimated to allow up to about 13% of children to experience one or more moderate O₃-induced FEV₁ decrements \geq 10%, and 7% to experience two or more such decrements (Table 4-5).

With regard to O₃ epidemiologic studies we note that, compared to a standard with a level of 70 or 65 ppb, a revised standard with a level of 60 ppb would more effectively maintain short-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard. Specifically, in all of the U.S. and Canadian epidemiologic studies evaluated, the majority of study cities had ambient O₃ concentrations that would likely have violated a standard with a level of 60 ppb. Thus, none of the U.S. and Canadian epidemiologic studies analyzed provide evidence for O₃ health effect associations when the majority of study locations would likely have met a standard with a level of 60 ppb (Tables 4-1 and 4-2). As discussed above, while this analysis does not provide information on the extent to which the O₃-associated morbidity or mortality would persist upon meeting an O₃ standard with a level of 60 ppb, it suggests that a revised O₃ standard with a level of 60 ppb would require reductions in the ambient O₃ concentrations that provided the basis for those health effect associations.

With regard to long-term O₃ concentrations, compared to the current standard or a revised O₃ standard with a level of 70 or 65 ppb, a revised standard with a level of 60 ppb would be expected to further reduce the risk of respiratory mortality associated with long-term O₃ concentrations, based on information from the study by Jerrett et al. (2009).⁵³ In addition, a standard with a level of 60 ppb would be expected to more effectively maintain long-term O₃ concentrations below those where the study by Jerrett et al. (2009) indicates the most confidence in the reported association with respiratory mortality. Specifically, air quality analyses indicate this to be the case in all of the urban case study areas evaluated at a level of 60 ppb, compared to 6 out of 12 areas for the current standard, 9 out of 12 for a standard with a level of 70 ppb, and 10 out of 12 for a standard with a level of 65 ppb (Table 4-3). Finally, a revised standard with a level of 60 ppb would be expected to further reduce long-term O₃ concentrations based on the types of metrics that have been reported in recent epidemiologic studies to be associated with respiratory morbidity (i.e., seasonal averages of daily maximum 8-hour concentrations).

In further considering the potential implications of epidemiology studies for alternative standard levels, we note estimates of total mortality associated with short-term O₃ concentrations.⁵⁴ As discussed above, we consider estimates of total risk (i.e., based on the full distributions of ambient O₃ concentrations) and estimates of risk associated with O₃ concentrations in the upper portions of ambient distributions. With regard to total risk we note that, when summed across urban case study areas, a standard with a level of 60 ppb is estimated to reduce the number of deaths associated with short-term O₃ exposures by about 15% (2007) and 11% (2009), compared to the current standard (Figure 4-13).⁵⁵ For area-wide concentrations at or above 40 ppb, a standard with a level set at 60 ppb is estimated to reduce O₃-associated deaths by almost 60% compared to the current standard. For area-wide concentrations at or above 60 ppb, a standard level of 60 ppb is estimated to reduce O₃-associated deaths by over 95% compared to the current standard (Figure 4-13).

Relative to the current standard, or alternative O₃ standards with levels of 70 or 65 ppb, CASAC stated the following:

The frequency of lung function decrements and premature mortality from short-term exposure to ozone decreases even further when the alternative standard is lowered to 60 ppb (Frey, 2014, p.8).

⁵³ Though as discussed above, we note the lower confidence we place in these risk results (U.S. EPA, 2014a, section 9.6).

⁵⁴ As discussed above, compared to the weight given to the evidence and to HREA estimates of exposures of concern and lung function risks, we place relatively less weight on epidemiologic-based risk estimates.

⁵⁵ A standard with a level of 60 ppb is also estimated to reduce respiratory mortality associated with long-term O₃ concentrations in urban case study areas. However, given uncertainties associated with these risk estimates, as discussed above, we give them limited weight.

CASAC also concluded that “the recommended lower bound of 60 ppb would certainly offer more public health protection than levels of 70 ppb or 65 ppb and would provide an adequate margin of safety” (Frey, 2014, p. ii).

In summary, compared to a standard with a level of 70 or 65 ppb, a revised standard with a level of 60 ppb would be expected to (1) further reduce the occurrence of exposures of concern (by about 95 to 100% compared to the current standard), almost eliminating exposures at or above 60 ppb; (2) further reduce the occurrence of FEV₁ decrements \geq 10, 15, and 20%, (by about 45 to 85% compared to the current standard); (3) more effectively maintain short- and long-term O₃ concentrations below those present in the epidemiologic studies that reported significant O₃ health effect associations in locations likely to have met the current standard;⁵⁶ and (4) further reduce the risk of O₃-associated mortality and morbidity, particularly the risk associated with the upper portion of the distribution of ambient O₃ concentrations.

4.7 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

It is important to highlight the uncertainties associated with establishing standards for O₃ during and after completion of the NAAQS review process. Research needs go beyond what is necessary to understand health effects, population exposures, and risks of exposure for purposes of setting standards. Research can also support the development of more efficient and effective control strategies. In this section, we highlight areas for future health-related research, model development, and data collection activities to address these uncertainties and limitations in the current body of scientific evidence.

As has been presented and discussed in the ISA, particularly chapters 4 through 7, the scientific body of evidence informing our understanding of health effects associated with long- and short-term exposures to O₃ has been broadened and strengthened since the O₃ NAAQS review completed in 2008. Still, we have concluded that O₃ health research needs and priorities have not changed substantially since the 2007 O₃ Staff Paper (EPA 2007). Key uncertainties and research needs that continue to be high priority for future reviews of the health-based standards are identified below:

(1) An important aspect of risk characterization and decision making for air quality standard levels for the O₃ NAAQS is the characterization of the shape of exposure-response functions for O₃, including the identification of potential population threshold levels. Recent controlled human exposure studies of measurable lung function effects provide evidence for a smooth dose-response curve without evidence of a threshold for exposures between 40 and 120

⁵⁶As discussed above, these studies do not provide information on the extent to which O₃ health effect associations would persist following reductions in ambient O₃ concentrations in order to meet a standard with a level of 60 ppb.

ppb O₃ (US EPA, 2013, Figure 6-1). Considering the importance of estimating health risks in the range below 80 ppb O₃, additional research is needed to evaluate responses in healthy and especially people with asthma in the range of 40 to 70 ppb for 6-8 hour exposures while engaged in moderate exertion.

(2) Similarly, for health endpoints reported in epidemiologic studies such as hospital admissions, ED visits, and premature mortality, an important aspect of characterizing risk is the shape of concentration-response functions for O₃, including identification of potential population threshold levels. Most of the recent studies and analyses continue to show no evidence for a clear threshold in the relationships between O₃ concentrations commonly observed in the U.S. during the O₃ season and these health endpoints, though evidence indicates less certainty in the shape of the concentration-response curve at the lower end of the distribution of O₃ concentrations. However, there continues to be heterogeneity in the O₃-mortality relationship across cities (or regions), including effect modifiers that are also expected to vary regionally, which are sources of uncertainty. Additionally, whether or not exposure errors, misclassification of exposure, or potential impacts of other copollutants may be obscuring potential population thresholds is still unknown.

(3) The extent to which the broad mix of photochemical oxidants and more generally other copollutants in the ambient air (e.g., PM, NO₂, SO₂, etc.) may play a role in modifying or contributing to the observed associations between ambient O₃ and various morbidity effects and mortality continues to be an important research question. Ozone has long been known as an indicator of health effects of the entire photochemical oxidant mix in the ambient air and has served as a surrogate for control purposes. A better understanding of sources of the broader pollutant mix, of human exposures, and of how other pollutants may modify or contribute to the health effects of O₃ in the ambient air, and vice versa, is needed to better inform future NAAQS reviews.

(4) As epidemiologic research has continued to be an important factor in assessing the public health impacts of O₃, 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.

(5) Recent animal toxicological evidence, combined with limited evidence from controlled human exposure studies of cardiovascular morbidity and epidemiologic studies of cardiovascular mortality, have provided evidence of both direct and indirect effects on the cardiovascular system. However, additional work will need to examine biologically plausible mechanisms of cardiovascular effects, expand upon preliminary evidence from controlled human

exposure studies, address inconsistencies observed in epidemiologic studies of cardiovascular morbidity, and determine the extent to which O₃ is directly implicated or works together with other pollutants in causing adverse cardiovascular effects in both at-risk and the general populations.

(6) Most epidemiologic studies of short-term exposure effects have employed time-series or case-crossover study designs and have been conducted in large populations. These study designs remain subject to uncertainty due to use of ambient fixed-site data serving as a surrogate for ambient exposures, and to the difficulty of determining the impact of any single pollutant among the mix of pollutants in the ambient air. Measurements made at stationary outdoor monitors have been used as independent variables for air pollution, but the accuracy with which these measurements actually reflect subjects' exposure is not yet fully understood. Also, additional research is needed to improve the characterization of the degree to which discrepancy between stationary monitor measurements and actual pollutant exposures introduces error into statistical estimates of pollutant effects in epidemiologic studies.

(7) Recent studies of "long-term" O₃ often evaluate associations with daily maximum concentrations, averaged over the O₃ season. Research is needed to better understand the extent to which health effects associated with such long-term metrics are attributable to long-term average concentrations versus the repeated occurrence of daily maximum concentrations.

(8) Improved understanding of human exposures to ambient O₃ and to related copollutants is an important research need. Population-based information on human exposure for healthy adults and children and at-risk populations, including people with asthma, to ambient O₃ concentrations, including exposure information in various microenvironments, is needed to better evaluate current and future O₃ exposure models. Such information is needed for sufficient periods to facilitate evaluation of exposure models throughout the O₃ season.

(9) Information is needed to improve inputs to current and future population-based O₃ exposure and health risk assessment models. Collection of time-activity data over longer time periods is needed to reduce uncertainty in the modeled exposure distributions that form an important part of the basis for decisions regarding NAAQS for O₃ and other air pollutants. Research addressing energy expenditure and associated breathing rates in various population groups, particularly healthy children and children with asthma, in various locations, across the spectrum of physical activity, including sleep to vigorous exertion, is needed.

(10) An important consideration in the O₃ NAAQS review is the characterization of background levels. There still remain substantial uncertainties in the characterization of 8-hour daily max O₃ background concentrations. Further research to improve the evaluation of the global and regional models which have been used to characterize estimates of background levels

would improve understanding of the role of non-U.S. anthropogenic emissions on O₃ levels over the U.S.

4.8 SUMMARY OF STAFF CONCLUSIONS ON PRIMARY STANDARD

In this section, we summarize our conclusions regarding the primary O₃ standard. These conclusions are informed by our consideration of the available scientific evidence as assessed in the ISA, air quality/exposure/risk information assessed in the final HREA, recommendations and advice received from CASAC, and comments received from members of the public.

As an initial matter in this PA, staff concludes that reducing ambient O₃ concentrations to meet the current standard will provide important improvements in public health protection. This initial conclusion is based on (1) the strong body of scientific evidence indicating a wide range of adverse health outcomes attributable to exposures to O₃ concentrations found in the ambient air and (2) estimates indicating decreased O₃ exposures and health risks upon meeting the current standard, compared to recent air quality. Strong support for this conclusion is provided by the available health evidence, and by HREA estimates of exposures to O₃ concentrations shown to result in respiratory effects in healthy adults (exposures of concern \geq 60, 70 and 80 ppb); O₃-induced lung function risks (FEV₁ decrements \geq 10, 15 and 20%); and O₃-associated mortality and morbidity risks.

Staff further concludes that the O₃-attributable health effects estimated to be allowed by air quality that meets the current primary standard can reasonably be judged important from a public health perspective. This conclusion is based on consideration of the scientific evidence assessed in the ISA, including controlled human exposure studies reporting abnormal or adverse respiratory effects following exposures to O₃ concentrations below the level of the current standard and epidemiologic studies indicating associations with morbidity and mortality for air quality that would meet the current standard. This conclusion is also based on the HREA estimates of exposures of concern, lung function risks, and morbidity and mortality risks; on advice received from CASAC in their review of draft versions of the PA; on CASAC advice received in previous reviews; and on consideration of public comments. Staff reaches the overall conclusion that the available health evidence and exposure/risk information calls into question the adequacy of the public health protection provided by the current standard.

Given this conclusion regarding the adequacy of the current standard, staff also reaches conclusions for the Administrator's consideration regarding the elements of alternative primary O₃ standards that could be supported by the available evidence and exposure/risk information. In reaching conclusions about the range of potential alternative standards appropriate for consideration, staff is mindful that the Act requires primary standards that, in the judgment of the Administrator, are requisite to protect public health with an adequate margin of safety. The

primary standards are to be neither more nor less stringent than necessary. Thus, the Act does not require that primary NAAQS be set at zero-risk levels, but rather at levels that reduce risk sufficiently to protect public health with an adequate margin of safety.

The degree of public health protection provided by any NAAQS results from the collective impact of the elements of the standard, including the indicator, averaging time, level, and form. Staff's conclusions on each of these elements are summarized below.

- (1) **Indicator:** It is appropriate to continue to use O₃ as the indicator for a standard that is intended to address effects associated with exposure to O₃, alone or in combination with related photochemical oxidants. Based on the available information, staff concludes that there is no basis for considering any alternative indicator at this time. Meeting an O₃ standard can be expected to provide some degree of protection against potential health effects that may be independently associated with other photochemical oxidants, even though such effects are not discernible from currently available studies indexed by O₃ alone. Staff notes that control of ambient O₃ levels is generally understood to provide the best means of controlling photochemical oxidants, and thus of protecting against effects that may be associated with individual species and/or the broader mix of photochemical oxidants.
- (2) **Averaging time:** It is appropriate to consider continuing to use an 8-hour averaging time for the primary O₃ standard.
 - (a) Staff concludes that an 8-hour averaging time remains appropriate for addressing health effects associated with short-term exposures to ambient O₃. An 8-hour averaging time is similar to the exposure periods evaluated in controlled human exposure studies, including recent studies reporting respiratory effects following exposures to O₃ concentrations below the level of the current standard. In addition, epidemiologic studies provide evidence for health effect associations with 8-hour O₃ concentrations, as well as with 1-hour and 24-hour concentrations. A standard with an 8-hour averaging time (combined with an appropriate standard form and level) would also be expected to provide substantial protection against health effects attributable to 1- and 24-hour exposures.
 - (b) Staff also concludes that a standard with an 8-hour averaging time can provide protection against respiratory effects associated with longer term O₃ exposures. Analyses in the HREA show that as air quality is adjusted to just meet the current

or alternative 8-hour standards, most study areas are estimated to experience reductions in respiratory mortality associated with long-term O₃ concentrations. In addition, analyses in this PA indicate that just meeting an 8-hour standard with an appropriate level would be expected to maintain long-term O₃ concentrations (i.e., seasonal average of 1-hour daily max) below those where a key study indicates the most confidence in the concentration-response relationship with respiratory mortality. In considering other long-term O₃ metrics evaluated in recent health studies, analyses in the HREA indicate that the large majority of the U.S. population lives in locations where reducing NO_x emissions would be expected to decrease warm season averages of daily 8-hour ambient O₃ concentrations, a long-term metric used in several recent studies reporting associations with respiratory morbidity. Taken together, these analyses suggest that a standard with an 8-hour averaging time, coupled with the current 4th-highest form and an appropriate level, could provide appropriate protection against the long-term O₃ concentrations reported to be associated with respiratory morbidity and mortality.

- (3) **Form:** For an 8-hour O₃ standard with a revised level (as discussed below), it is appropriate to consider retaining the current form, defined as the 3-year average of the annual 4th-highest daily maximum concentration. Staff notes that this form was selected in 1997 and 2008 in recognition of the public health protection provided, when coupled with an appropriate averaging time and level, combined with the stability provided for implementation programs. The currently available evidence and exposure/risk information does not call into question these conclusions from previous reviews.
- (4) **Level:** The available scientific evidence and exposure/risk information provide strong support for considering an O₃ standard with a revised level in order to increase public health protection. Staff concludes that it is appropriate in this review to consider a revised standard level within the range of 70 ppb to 60 ppb, reflecting the judgment that a standard set within this range could provide an appropriate degree of public health protection and would result in important improvements in protection for at-risk populations and lifestyles.

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5 ADEQUACY OF THE CURRENT SECONDARY STANDARD

This chapter presents staff's considerations and conclusions regarding the adequacy of the current secondary O₃ NAAQS. In doing so, we pose the following overarching question:

- **Does the currently available scientific evidence- and exposure/risk-based information, as reflected in the ISA and WREA, support or call into question the adequacy and appropriateness of the protection afforded by the current secondary O₃ standard?**

In addressing this overarching question, we pose a series of more specific questions, as discussed in sections 5.1 through 5.5 below. We consider the nature of O₃-induced effects, including the nature of the exposures that drive the biological and ecological response and related biologically relevant exposure metrics (section 5.1); the scientific evidence and exposure/risk information, including that for associated ecosystem services, regarding (a) tree growth, productivity and carbon storage (section 5.2), (b) crop yield loss (section 5.3), (c) visible foliar injury (section 5.4), and (d) other welfare effects (section 5.5). Section 5.6 describes advice and recommendations received from CASAC. In section 5.7, we revisit the overarching question of this chapter and present staff conclusions on the adequacy and appropriateness of the current secondary standard.

5.1 NATURE OF EFFECTS AND BIOLOGICALLY RELEVANT EXPOSURE METRIC

- **Does the current evidence alter our conclusions from the previous review regarding the nature of O₃-induced welfare effects?**

As discussed further below, the current body of O₃ welfare effects evidence confirms and strengthens the conclusions reached in the last review on the nature of O₃-induced welfare effects. Ozone's phytotoxic effects were first identified on grape leaves in a study published in 1958 (Richards et al., 1958). In the more than fifty years that have followed, extensive research has been conducted both in and outside of the U.S. to examine the impacts of O₃ on plants and their associated ecosystems, since "of the phytotoxic compounds commonly found in the ambient air, O₃ is the most prevalent, impairing crop production and injuring native vegetation and ecosystems more than any other air pollutant" (U.S. EPA, 1989, 1996). Recent studies, assessed in the ISA, together with this longstanding and well established vegetation effects literature, further contribute to the coherence and consistency of the vegetation effects evidence.

In assessing the strength of the evidence, it is important to note that different types of studies can provide different types of information, each with different associated uncertainties

(U.S. EPA, 2013, Chapter 9, section 9.2). Controlled chamber studies are the best method for isolating or characterizing the role of O₃ in inducing the observed plant effects, and in assessing plant response to O₃ at the finer scales (U.S. EPA, 2013, Chapter 9, section 9.3). Recent controlled studies have focused on a variety of plant responses to O₃ including: 1) the underlying mechanisms as they relate to growth, productivity and carbon storage including: reduced carbon dioxide uptake due to stomatal closure (U.S. EPA 2013, section 9.3.2.1); 2) the upregulation of genes associated with plant defense, signaling, hormone synthesis and secondary metabolism (U.S. EPA 2013, section 9.3.3.2); 3) the down regulation of genes related to photosynthesis and general metabolism (U.S. EPA 2013, section 9.3.3.2); 4) the loss of carbon assimilation capacity due to declines in the quantity and activity of key proteins and enzymes (U.S. EPA, 2013, section 9.3.5.1); and 5) the negative impacts on the efficiency of the photosynthetic light reactions (U.S. EPA, 2013, section 9.3.5.1). As described in the ISA, these new studies “have increased knowledge of the molecular, biochemical and cellular mechanisms occurring in plants in response to O₃”, adding “to the understanding of the basic biology of how plants are affected by oxidative stress...” (U.S. EPA, 2013, p. 9-11). The ISA further concluded that controlled studies “have clearly shown that exposure to O₃ is causally linked to visible foliar injury, decreased photosynthesis, changes in reproduction, and decreased growth” in many species of vegetation (U.S. EPA 2013, p. 1-15).

Such effects at the plant scale can also be linked to an array of effects at larger spatial scales. For example, recent field studies at larger spatial scales, together with previously available evidence, support the controlled exposure study results and indicate that “ambient O₃ exposures can affect ecosystem productivity, crop yield, water cycling, and ecosystem community composition” (U.S. EPA, 2013, p. 1-15; Chapter 9, section 9.4).

The ISA summarizes the coherence across the full range of effects, from the least serious to the most serious, as follows (U.S. EPA, 2013, p. 1-8):

The welfare effects of O₃ can be observed across spatial scales, starting at the subcellular and cellular level, then the whole plant and finally, ecosystem-level processes. Ozone effects at small spatial scales, such as the leaf of an individual plant, can result in effects along a continuum of larger spatial scales. These effects include altered rates of leaf gas exchange, growth, and reproduction at the individual plant level, and can result in broad changes in ecosystems, such as productivity, carbon storage, water cycling, nutrient cycling, and community composition.

Based on its assessment of this extensive body of science, the ISA determined that, with respect to vegetation and ecosystems, a causal relationship exists between exposure to O₃ in ambient air and visible foliar injury effects on vegetation, reduced vegetation growth, reduced

productivity in terrestrial ecosystems, reduced yield and quality of agricultural crops and alteration of below-ground biogeochemical cycles (U.S. EPA 2013, Table 1-2). Additionally, the ISA determined that a likely to be causal relationship exists between exposures to O₃ in ambient air and reduced carbon sequestration in terrestrial ecosystems, alteration of terrestrial ecosystem water cycling and alteration of terrestrial community composition (U.S. EPA, 2013, Table 1-2). With regard to the relationship between O₃ and radiative forcing and climate change, the ISA determined that there is a causal relationship between changes in tropospheric O₃ concentrations and radiative forcing, and likely to be a causal relationship between changes in tropospheric O₃ concentrations and effects on climate (U.S. EPA, 2013, p. 1-13, and Table 1-3). From this set of effects that the ISA has concluded to be causally or likely causally related to O₃ in ambient air, we focus the discussion in the PA primarily on: 1) impacts on tree growth, productivity and carbon storage; 2) crop yield loss; 3) visible foliar injury. Each of these discussions also includes where appropriate, a discussion of any known or anticipated impacts that such individual plant or species level effects could have at larger scales, including ecosystems, and on associated ecosystem services.

In considering the available vegetation effects evidence, we make note of several important contextual features that frame our understanding of the science and how it informs our evaluation of the adequacy of the protection afforded by the current secondary NAAQS. First, we acknowledge that under natural conditions, a variety of factors can either mitigate or exacerbate the predicted O₃-plant interactions and are recognized sources of uncertainty and variability. These include: 1) multiple genetically influenced determinants of O₃ sensitivity; 2) changing sensitivity to O₃ across vegetative growth stages; 3) co-occurring stressors and/or modifying environmental factors (U.S. EPA, 2013, section 9.4.8).

Second, we acknowledge that the species that have been studied for O₃ sensitivity represent only a fraction of the tens of thousands of plant species that grow in the U.S. (USDA NRCS, 2014)¹, and that these species were typically selected because of their commercial importance (e.g., commodity crop or timber species) or because of observed O₃-induced visible foliar injury in the field. Of the species known to be sensitive to O₃ for foliar injury, 66 species have been identified on National Park Service (NPS) and U.S. Fish and Wildlife Service lands² and a subset of these are used in the USFS biomonitoring program (discussed in section 5.4 below). A number of these species have also been identified as important to tribal cultural practices (see Appendix 5-A). Appendix 7J of the 2007 Staff Paper showed that no state in the

¹ USDA NRCS. 2014. The PLANTS Database (<http://plants.usda.gov>, 3 January 2014). National Plant Data Team, Greensboro, NC 27401-4901 USA.

² See <http://www2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf>

lower 48 states had less than seven known O₃-sensitive plant species, with the majority of states having between 11 and 30 (see Appendix 7J-2 in U.S. EPA, 2007). We would not expect this information to have changed since the previous review because there has been very little change in the list of sensitive species and the occurrence of any of these plant species within a state would not be expected to change. With respect to agricultural species, a number of important commodity crops such as soybean and additional fruit and vegetable species such as lettuce have been shown to be sensitive to O₃ for either foliar injury or yield loss (U.S. EPA, 2013, section 9.4.4.1; Abt Associates, Inc., 1995).

Third, we acknowledge that out of the group of species known to be sensitive to O₃, we have chosen to focus primarily on species for which we have robust exposure-response (E-R) functions for biomass loss and yield loss using the W126 form (i.e., 11 tree and 10 crop species) in order to be able to quantitatively relate predicted changes in O₃ to predicted changes in plant exposures, responses and associated risks.³ However, while we recognize that this small group represents only a fraction of all species known or anticipated to be sensitive to O₃ in the U.S., we also note, as did CASAC, that among the studied species, there is a fairly large range of O₃ sensitivities represented, so that it could be reasonable to assume that other non-studied species might have sensitivities that fall within or near this range. Specifically, CASAC states “[i]t should not be assumed that species of unknown sensitivity are tolerant to ozone. It 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, 2014, p. 11).

Fourth, we acknowledge that in addition to the well-studied effects of biomass loss in trees and crops and visible foliar injury in bioindicator plants that we can quantify, numerous other more subtle and less easily observed effects occur along the continuum of spatial scales that lead to ecosystem effects. While these effects are more difficult to quantify, we acknowledge that any secondary standard set to protect the public welfare against the known and quantifiable adverse effects to vegetation should also consider the anticipated, but currently unquantifiable, potential adverse effects on vegetation, ecosystems and associated services.

Finally, we further acknowledge that in light of the above, when considering the available evidence, we seek to find the right balance between placing weight on the associated uncertainties and limitations of the evidence and placing weight on its well-established strength, coherence and consistency. In so doing, we note that CASAC, in commenting on section 6.7 which describes key uncertainties and future research areas, states that “[w]hile these scientific research priorities will enhance future scientific reviews of the ozone primary and secondary

³ There is an E-R function available for a 12th tree species (cottonwood), but this E-R function is considered less robust because it is based on the results of a single gradient study (Gregg et al., 2003).

standards, we also make clear that there is sufficient scientific evidence, and sufficient confidence in the available research results, to support the advice we have given above for this review cycle of the primary and secondary standards” (Frey, 2014, p. iv).

- **Does the current evidence continue to support a cumulative, seasonal exposure index as a biological-relevant and appropriate metric for assessment of the evidence and exposure/risk information?**

In this review, the ISA assessment of the full body of currently available evidence stated the following regarding biological indices (U.S. EPA, 2013, p. 2-44):

The main conclusions from the 1996 and 2006 O₃ AQCDs [Air Quality Criteria Documents] regarding indices based on ambient exposure remain valid. These key conclusions can be restated as follows:

- *ozone effects in plants are cumulative;*
- *higher O₃ concentrations appear to be more important than lower concentrations in eliciting a response;*
- *plant sensitivity to O₃ varies with time of day and plant development stage;*
- *quantifying exposure with indices that cumulate hourly O₃ concentrations and preferentially weight the higher concentrations improves the explanatory power of exposure/response models for growth and yield, over using indices based on mean and peak exposure values.*

The long-standing body of available evidence upon which these conclusions are based, provides a wealth of information on aspects of O₃ exposure that are important in influencing plant response. Specifically, a variety of “factors with known or suspected bearing on the exposure-response relationship, including concentration, time of day, respite time, frequency of peak occurrence, plant phenology, predisposition, etc.,” have been identified (U.S. EPA, 2013, section 9.5.2). In addition, the importance of the duration of the exposure and the relatively greater importance of higher concentrations over lower in determining plant response to O₃ have been consistently well documented (U.S. EPA, 2013, section 9.5.3). Much of this evidence was assessed in the 1996 Criteria Document (CD) (U.S. EPA, 1996), while more recent work substantiating this evidence is assessed in the subsequent 2006 CD and 2013 ISA.

Understanding of the biological basis for plant response to O₃ exposure led to the development of a large number of “mathematical approaches for summarizing ambient air quality information in biologically meaningful forms for O₃ vegetation effects assessment purposes ...” (U.S. EPA, 2013, section 9.5.3), including those that cumulate exposures over some specified period while weighting higher concentrations more than lower (U.S. EPA, 2013, section 9.5.2). As with any summary statistic, these exposure indices retain information on

some, but not all, characteristics of the original observations. As discussed in greater detail in section 6.2 below, the 1996 CD contained an extensive review of the published literature on different types of exposure-response metrics, including comparisons between metrics, from which the 1996 Staff Paper built its assessment of forms appropriate to consider in the context of the secondary NAAQS review. The result of these assessments was a decision by the EPA to focus on cumulative, concentration-weighted indices, which were recognized as the most appropriate biologically based metrics to consider in this context, with attention given primarily to two cumulative, concentration weighted index forms: SUM06 and W126. The SUM06 index is a threshold-based approach described as the sum of all hourly O₃ concentrations greater or equal to 0.06 ppm observed during a specified daily and seasonal time window (U.S. EPA, 2013, section 9.5.2). The W126 index is a non-threshold approach described as the sigmoidally weighted sum of all hourly O₃ concentrations observed during a specified daily and seasonal time window, where each hourly O₃ concentration is given a weight that increases from 0 to 1 with increasing concentration (Lefohn et al., 1988; Lefohn and Runeckles, 1987; U.S. EPA, 2013, section 9.5.2).

In both the 1997 and 2008 reviews, the EPA concluded that the risk to vegetation comes primarily from cumulative exposures to O₃ over a season or seasons⁴ and proposed, as one policy alternative, a secondary standard set in terms of such a form: SUM06 (61 FR 65716) and W126 (72 FR 37818) in the 1997 and 2008 reviews, respectively. Although in both reviews the policy decision was made to set the secondary standard to be identical to a revised primary standard (with an 8-hour averaging time), the Administrator, in both cases, also concluded, consistent with CASAC advice, that a cumulative, seasonal index was the most biologically relevant way to relate exposure to plant growth response (62 FR 38856, 73 FR 16436). Similarly, in the 2010 proposed reconsideration of the 2008 decision, the EPA proposed to conclude that O₃ exposure indices that cumulate differentially weighted hourly concentrations are the best candidates for relating exposure to plant growth responses and proposed as the only policy option to set the secondary standard in terms of one such form, the W126 (75 FR 2938). This approach of establishing a secondary standard that was separate and distinct from the primary standard and in particular using a cumulative seasonal exposure index such as W126 received strong support from CASAC in both 2008 and 2010 reviews (Henderson, 2006, 2008; Samet, 2010), as it has again in this review, as discussed in section 5.6 below.

An alternative to using ambient exposure durations and concentrations to predict plant response has been developed in recent years, primarily in Europe, i.e., flux models. While

⁴ In describing the form as “seasonal”, the EPA is referring generally to the growing season of O₃-sensitive vegetation, not to the seasons of the year (i.e., spring, summer, fall, winter).

“some researchers have claimed that using flux models can be used to better predict vegetation responses to O₃ than exposure-based approaches...” because flux models estimate the ambient O₃ concentration that actually enters the leaf (i.e., flux or deposition) (U.S. EPA, 2013, p. 9-114), it is important to note that “[f]lux calculations are data intensive and must be carefully implemented” (U.S. EPA, 2013, p. 9-114). Further, “[t]his uptake-based approach to quantify the vegetation impact of O₃ requires inclusion of those factors that control the diurnal and seasonal O₃ flux to vegetation (e.g., climate patterns, species and/or vegetation-type factors and site-specific factors)” (U.S. EPA, 2013, p. 9-114). In addition to these data requirements, each species has different amounts of internal detoxification potential that may protect species to differing degrees. This balance between O₃ flux and detoxification processes has been termed the “effective flux”. Accordingly, the “models have to distinguish between stomatal and non-stomatal components of O₃ deposition to adequately estimate actual concentration reaching the target tissue of a plant to elicit a response” and “ultimately the ‘effective’ flux” (U.S. EPA, 2013, pp. 9-114). The lack of detailed species- and site-specific data required for flux modeling in the U.S. and the lack of understanding of detoxification processes have continued to make this technique less viable for use in vulnerability and risk assessments at the national scale in the U.S. (U.S. EPA, 2013, section 9.5.4).

Therefore, consistent with the ISA conclusions regarding the appropriateness of considering cumulative exposure indices that preferentially weight higher concentrations over lower for predicting O₃ effects of concern based on the long-established conclusions and long-standing supporting evidence described above, and in light of continued CASAC support, we continue to focus on the aspects of ambient O₃ exposures that have biological relevance and the biologically relevant exposure indices or metrics that have been designed in light of this consideration, i.e., cumulative concentration-weighted indices. In addition, given the lack of any information in the current review to the contrary, we therefore again conclude that the current evidence, as in recent reviews, continues to support a cumulative, seasonal exposure index as a biologically relevant and appropriate metric for assessment of the evidence and exposure/risk information, and in particular, the W126 cumulative, seasonal metric (U.S. EPA, 2013, section 2.6.6.1, section 9.5.2). Such a metric, as stated above, has an “explanatory power” that is improved “over using indices based on mean and peak exposure values” (U.S. EPA, 2013, section 2.6.6.1, p. 2-44). Thus, as in the WREA, discussions of the effects evidence and exposure/risk results in sections 5.2 through 5.5 of this PA are provided in terms of the W126 index, where available.

- **What paradigm is being used to consider which of the known or anticipated O₃-induced effects have the potential to be adverse to the public welfare?**

The Clean Air Act (CAA), in section 109 (b) (2) requires that “[a]ny national secondary ambient air quality standard... shall specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air.” The “criteria” referred to in this text are defined earlier in CAA section 108 (a) (2) which states in part that “[a]ir quality criteria for an air pollutant shall accurately reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on public health or welfare which may be expected from the presence of such pollutant in the ambient air, in varying quantities.” Thus, while the criteria include “all identifiable effects”, Congress directed the EPA to establish the secondary NAAQS based on the Administrator’s judgement of what is requisite to protect against “adverse effects” in the context of the public welfare. However, the CAA does not provide specific standards for determining what constitutes an effect that is adverse to the public welfare leaving these determinations instead to the “judgment of the Administrator”. As stated above in section 1.1, the PA is intended to help “bridge the gap” between the Agency’s scientific assessments presented in the ISA and REAs, which constitute “the criteria” and the judgments required of the EPA Administrator regarding whether it is appropriate to retain or revise the NAAQS.⁵ In the context of the secondary standard, the PA thus serves the function of translating the information assessed in both the ISA and WREA into the public welfare policy context. In order to do this, the PA applies a specific approach or paradigm (see also section 1.3.2. above), that guides staff’s consideration and interpretation of the available information and which then informs staff conclusions regarding policy options that are appropriate for the Administrator to consider. The following discussion describes the evolution of this paradigm throughout the last several reviews and into the current review.

In the 1997 secondary O₃ NAAQS review, a policy-relevant distinction was made between the terms “injury” and “damage”. Specifically, O₃-induced “injury” to vegetation was defined as encompassing all plant reactions, including reversible changes or changes in plant metabolism (e.g., altered photosynthetic rate), altered plant quality or reduced growth. In contrast, “damage” was defined to include only those injury effects that reach sufficient magnitude as to also reduce or impair the intended use or value of the plant, thus potentially being adverse to the public welfare. In published scientific literature, on the other hand, the terms “adverse”, “injury” or “damage” continue to be used interchangeably. The early O₃ NAAQS reviews focused primarily on O₃-induced effects at the individual and species level. In such

⁵American Farm Bureau Federation v. EPA, 559 F. 3d 512, 521 (D.C. Cir. 2009); Natural Resources Defense Council v. EPA, 902 F. 2d 962, 967-68, 970 (D.C. Cir. 1990).

cases, examples of vegetation effects that were also classified as damage included reductions in aesthetic values (e.g., visible foliar injury in ornamental species or occurring in valued natural landscapes such as national parks) and tree growth/biomass and crop yield losses (i.e., in terms of weight, number, quality, appearance, or size of harvestable crop or timber species). In the context of evaluating effects on single plants or species grown in monocultures such as managed forests, this construct continues to remain useful (73 FR 16492/96).

In subsequent reviews, however, the scientific literature linking O₃ effects on plants or species to effects at the community or ecosystem level continued to increase. As a result, more recent reviews have considered a more expansive construct or paradigm of what appropriately constitutes O₃ “damage” to extend beyond that of the individual or species level. A number of these broader paradigms have been discussed in the literature (72 FR 37890; Hogsett et al., 1997; Young and Sanzone, 2002). Thus, in the 2008 review, the Administrator, while continuing to express support for relying on a definition of “adverse” discussed in section IV.A.3 of the proposal (72 FR 37889-37890) that embeds “the concept of ‘intended use’ of the ecological receptors and resources that are affected”, also supported applying “that concept beyond the species level to the ecosystem level” (73 FR 16496). In so doing, the Administrator took note of “a number of actions taken by Congress to establish public lands that are set aside for specific uses that are intended to provide benefits to the public welfare, including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment of future generations” (73 FR 16496). Thus, this paradigm recognized that the significance to the public welfare of O₃-induced effects on sensitive vegetation growing within the U.S. can vary depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located. Accordingly, any given O₃-related effect on vegetation and ecosystems (e.g., biomass loss, crop yield loss, visible foliar injury) may be judged to have a different degree of impact on the public welfare depending, for example, on whether that effect occurs in a Class I area, a city park, or commercial cropland. In the 2010 proposed reconsideration, the Administrator proposed to place the highest priority and significance on vegetation and ecosystem effects to sensitive species that are known to or are likely to occur in federally protected areas such as national parks and other Class I areas, or on lands set aside by states, tribes and public interest groups to provide similar benefits to the public welfare (75 FR 3023/24). Effects occurring in such areas would likely have the highest potential for being classified as adverse to the public welfare, due to the expectation that these areas need to be maintained in a more pristine condition to ensure their intended use is met. In contrast, in that proposal, the Administrator considered it less clear the degree to which O₃ vegetation impacts potentially predicted to occur in areas and on species that are already heavily managed

to obtain a particular output (such as commodity crops or commercial timber production), would impair the intended use at a level that would be judged adverse to the public welfare and also noted that these species would likely receive some protection for a standard set to provide protection in areas set aside to be maintained in a more pristine condition (75 FR 3024).

In the current review, we revisited the appropriateness of using this paradigm and whether the available information supported any further evolution. In so doing, we noted the ISA text, which states that “[o]n a broader scale, ecosystem services may provide indicators for ecological impacts. Ecosystem services are the benefits that people obtain from ecosystems (UNEP, 2003)” (U.S. EPA, 2013, Preamble, p. 1xxii) and the ISA list of a number of ecosystem services that can be affected by O₃-induced effects on plants and ecosystems, including decreased productivity, decreased carbon sequestration, altered water cycling, and altered community composition (U.S. EPA, 2013, Figure 2-2, pp. 2-36; Figure 9-1, p. 9-3). We further noted that other recent EPA documents have already incorporated this concept. For example, the recent review of the secondary NAAQS for oxides of nitrogen and sulfur recognized that changes in ecosystem services may be used to aid in characterizing a known or anticipated adverse effect to public welfare and that an evaluation of adversity to the public welfare might consider the likelihood, type, magnitude, and spatial scale of the effect, as well as the potential for recovery and any uncertainties relating to these conditions (77 FR 20232). Similarly, the EPA document, *Ecological Benefits Assessment Strategic Plan*, includes a definition of ecological goods and services used for the purposes of benefits assessment that EPA has relied upon in regulatory impact analyses for previous rulemakings. This definition states that ecological goods and services are the “outputs of ecological functions or processes that directly or indirectly contribute to social welfare or have the potential to do so in the future”...and that “[s]ome outputs may be bought and sold, but most are not marketed” (U.S. EPA, 2006b).

After considering this information, and given the accepted use of these concepts and their clear applicability to the secondary NAAQS review, we concluded that while it is still appropriate to apply the paradigm used in the 2010 reconsideration that takes into account the variation in public welfare significance of O₃-related vegetation effects when evaluating the potential adversity of the currently available evidence, there is also sufficient support for an expansion of this paradigm to explicitly include consideration of impacts to ecosystem goods and services. Doing so can help clarify the relationship between predicted O₃-induced vegetation effects and anticipated impacts on public welfare benefits received from those impacted species or ecosystems, and, as was done in the WREA, clarify how those services might be expected to change under air quality scenarios representing the current and potential alternative secondary standards (U.S. EPA, 2014a, chapter 5). The expansion of this paradigm to include ecosystem

goods and services brings with it a number of additional considerations. Specifically, when considering the potential public welfare benefits from these goods and services, it is important to note that they can accrue across a range of dimensions, including spatial, temporal, and social, and these likely will vary depending on the type of effect being characterized. For example, ecosystems can cover a range of spatial scales, and the services they provide can accrue locally or be distributed more broadly such as when crops are sold and eaten locally and/or also sold in regional, national and world markets. Ecosystem services can likewise be realized over a range of temporal scales from immediate up to long term (e.g., the removal of air pollutants that have a short-term impact on human health but are also climate forcers with long atmospheric lifetimes, such that their removal may have immediate as well as long-term benefits). The size of the societal unit receiving benefits from ecosystem services can also vary dramatically. For example, a national park can provide direct recreational services to the thousands of visitors that come each year, but also provide an indirect value to the millions who may not visit but receive satisfaction from knowing it exists and is preserved for the future (U.S. EPA, 2014a, chapter 5, section 5.5.1).

We thus conclude that it is appropriate for the Administrator, in specifying what “level of air quality” for a pollutant “is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air,” to evaluate the scientific evidence regarding these effects in the context of the most recent paradigm discussed above. This paradigm integrates the concepts of: 1) variability in public welfare significance given intended use and value of the affected entity such as an individual species; 2) relevance of associated ecosystem services to public welfare; 3) variability in spatial, temporal, and social distribution of ecosystem services associated with known and anticipated welfare effects. In so doing, we recognize that there is no bright-line rule delineating the set of conditions or scales at which known or anticipated effects become adverse to public welfare. Thus, the evidence and exposure/risk information discussed in this chapter will be further evaluated in Chapter 6 using the concepts incorporated in this paradigm to help inform the Administrator’s judgments with respect to the adversity of the effects to the public welfare and what is considered requisite protection.

5.2 FOREST TREE GROWTH, PRODUCTIVITY AND CARBON STORAGE

Trees merit consideration from a public welfare perspective because they provide many services that people value, including aesthetic value (also discussed in section 5.4 below), food, fiber, timber, other forest products, habitat, recreational opportunities, climate regulation, erosion control, air pollution removal, hydrologic and fire regime stabilization (U.S. EPA, 2014a, section

6.1, Figure 6-1, section 6.4, Table 6-13). One source identifies as many as 1,497 native tree species growing in the lower 48 of the U.S.⁶ Ozone has been shown to be phytotoxic to a number of important U.S. tree species with respect to growth, productivity, and carbon storage, including for cumulative exposures that have occurred under recent U.S. air quality. This section includes a discussion of the policy-relevant evidence and weight-of-evidence conclusions discussed in the ISA (section 5.2.1) and the exposure/risk results, including both quantitative and qualitative results for these effects, as well as associated ecosystem services (section 5.2.2) as described in the final WREA (U.S. EPA, 2014a). Important uncertainties and limitations in the available information are also discussed in each section. These discussions highlight the information we consider relevant to answering the overarching question and associated policy-relevant questions included in this section.

5.2.1 Evidence-based Considerations

- **To what extent has scientific information become available that alters or substantiates our prior conclusions of O₃-related effects on forest tree growth, productivity and carbon storage and of factors that influence associations between O₃ concentrations and these effects?**

Research published since the 2006 CD substantiates prior conclusions regarding O₃-related effects on forest tree growth, productivity and carbon storage. The ISA states that “previous O₃ AQCDs concluded that there is strong evidence that exposures to O₃ decreases photosynthesis and growth in numerous plant species” and that “[s]tudies published since the 2008 review support those conclusions” (U.S. EPA, 2013, p. 9-42). The recent studies that support the previous conclusions come from a variety of different study types that cover an array of different species, effects endpoints, levels of biological organization and exposure methods and durations. As stated in Chapter 1, and above, the documentation of O₃-induced species-specific responses across multiple lines of evidence, and over the full range of levels of biological organization highlights and strengthens the consistency and coherence of the evidence available in this review.

The previously available strong evidence for trees includes robust exposure-response (E-R) functions for seedling biomass loss in 11 species developed under the National Health and Environmental Effects Research Laboratory-Western Ecology Division (NHEERL-WED) program. This series of experiments used open-top-chambers (OTC) to study seedling growth response for a single growing season under a variety of O₃ exposures (ranging from near

⁶ USDA, NRCS. 2014. The PLANTS Database (<http://plants.usda.gov>, 7 July 2014). National Plant Data Team, Greensboro, NC 27401-4901 USA.

background to well above current ambient concentrations) and growing conditions (U.S. EPA 2013, section 9.6.2, Lee and Hogsett, 1996). The evidence from these studies shows that there is a wide range in sensitivity across the studied species in the seedling growth stage over the course of a single growing season, with some species being extremely sensitive and others being very insensitive, or alternatively quite tolerant, over the range of cumulative O₃ exposures studied (See Figure 5-1, below).

In addition, field-based studies of species growing in natural stands have compared observed plant response across a number of different sites and/or years when exposed to varying ambient O₃ exposure conditions only. For example, a study conducted in forest stands in the southern Appalachian Mountains found that the cumulative effects of ambient levels of O₃ decreased seasonal stem growth (measured as a change in circumference) by 30-50% for most of the examined tree species (i.e., tulip poplar, black cherry, red maple, sugar maple) in a high O₃ year in comparison to a low O₃ year (McLaughlin et al., 2007a). The authors also reported that high ambient O₃ concentrations can increase whole-tree water use and in turn reduce late-season streamflow (McLaughlin et al., 2007b) (U.S. EPA, 2013, p. 9-43). This study used ambient O₃ conditions found at several different sites to create the variation in O₃ exposures.

Because trees and other perennials are long lived, it is important to consider the potential for impacts beyond a single year. Limited evidence in previous reviews reported that vegetation effects from a single year of exposure to elevated O₃ could be observed in the following year. For example, growth affected by a reduction in carbohydrate storage in one year may result in the limitation of growth in the following year. Such “carry-over” effects have been documented in the growth of some tree seedlings and in roots (U.S. EPA, 2013, section 9.4.8; Andersen, et al., 1997). In the current review, additional field-based evidence expands our understanding of the consequences of single and multi-year O₃ exposures in subsequent years. A number of studies were conducted at a planted forest at the Aspen Free-Air Carbon Dioxide Enrichment (FACE) site in Wisconsin. These studies, which occurred in a field setting more similar to natural forest stands than OTC studies, observed tree growth responses when grown in single or two species stands within 30-m diameter rings and exposed to ambient and above ambient conditions over a period of ten years. Some researchers similarly recognized the potential for carry-over effects when they observed that the effects of O₃ on birch seeds (reduced weight, germination, and starch levels) could lead to a negative impact on species regeneration in subsequent years, and that the effect of reduced aspen bud size might have been related to the observed delay in spring leaf development. These effects suggest that elevated O₃ exposures have the potential to alter carbon metabolism of overwintering buds, which may have subsequent effects in the following year (Darbah, et al., 2008, 2007; Riikonen et al., 2008; U.S. EPA, 2013, section 9.4.3). Other studies found that, in addition to affecting tree heights, diameters, and main

stem volumes in the aspen community, elevated O₃ over a 7-year study period was reported to increase the rate of conversion from a mixed aspen-birch community to a community dominated by the more tolerant birch, leading the authors to conclude that elevated O₃ may alter intra- and inter-species competition within a forest stand (Kubiske et al., 2006; Kubiske et al., 2007) (U.S. EPA, 2013, section 9.4.3). These studies confirm earlier FACE results showing large decreases in growth for aspen over a 6-7 year period when exposed to elevated O₃ (King et al., 2005) and that yearly biomass loss cumulated over that timeframe.

In addition to individual studies, recent meta-analyses have quantified the effect of O₃ on trees across large numbers of studies. In particular, a recent meta-analysis (Wittig, et al., 2007) indicates a relationship between O₃ concentrations in the northern hemisphere and stomatal conductance and photosynthesis, which decrease growth (U.S. EPA, 2013, section 9.4.3.1; Wittig et al., 2007).⁷ This analysis reported that recent O₃ concentrations in the northern hemisphere are decreasing stomatal conductance (13%) and photosynthesis (11%) across tree species. It also found that younger trees (<4 years) were affected less by O₃ than older trees (Wittig, et al., 2007). A second meta-analysis, Wittig, et al. (2009), which quantitatively compiled peer-reviewed studies from the past 40 years, found that ambient O₃ concentrations reported in those studies significantly decreased annual total biomass growth (7%) across the 263 studies (U.S. EPA, 2013, section 9.4.3.1). The ISA states that this meta-analysis demonstrates the coherence of O₃ effects across numerous studies and species that used a variety of experimental techniques, and these results support the conclusion of the previous CD that exposure to O₃ decreases plant growth. Other meta-analyses have examined the effect of O₃ exposure on root growth and generally found that O₃ exposure reduced carbon allocated to roots. For example, Grantz et al. (2006) found that O₃ exposure reduced the ratio between the relative growth rate of the root and shoot by 5.6% (U.S. EPA, 2013, pp. 9-45 to 9-46).

In our consideration of the recent studies discussed above, in combination with the entire body of available evidence, we note that the recent scientific literature further strengthens and contributes to the consistency and coherence of the evidence base by substantiating and expanding prior conclusions regarding O₃-related effects on tree growth, productivity and carbon storage, including mixed species forest stands and the ecosystems and services that derive from them, as discussed more fully below. We also note that the ISA concludes that the currently available evidence supports causal determinations regarding O₃ effects on tree growth and productivity and the associated effects of altered carbon allocation to below ground tissues, rates of leaf and root production, turnover and decomposition that can alter below-ground

⁷ Meta-analysis allows for the objective development of a quantitative consensus of the effects of a treatment across a wide body of literature.

biogeochemical cycles, as well as the likely to be a causal relationship with reduced carbon sequestration and alteration of terrestrial community composition and water cycling (U.S. EPA, 2013, Table 2-2; 9-19). Finally, we note that except for the recent limited information on cottonwood in the ISA (U.S. EPA, 2013, section 9.6.3.3), there has not been an expansion in the number of tree species for which we have E-R functions, so only 12 species have available E-R functions for use in quantitative exposure and risk analyses and for predicting tree seedling response under a range of O₃ exposure conditions/scenarios. While these 12 species represent only a small fraction (0.8%) of the total number of native tree species in the contiguous U.S. (1,497), this small subset includes eastern and western species, deciduous and coniferous species, and species that grow in a variety of ecosystems and represent a range of tolerance to O₃ (Figure 5-1 below, U.S. EPA 2013, section 9.6.2; U.S. EPA, 2014a, section 6.2, Figure 6-2, Table 6-1). The CASAC states in their letter to the Administrator on the second draft PA, that while “[t]here is considerable uncertainty in extrapolating from the 12 forest tree species to all forest tree species in the U.S...[i]t is scientifically justifiable to extrapolate from the known E-R curves, assuming that they are representative of the un-sampled population” (Frey, 2014, p. 15).

As we further consider the results from the quantitative exposure and risk analyses, described below and in the WREA (U.S. EPA, 2014a), that in addition to the quantifiable portion of risks associated with the robust information on tree species, it is also reasonable to consider, based on the long-standing evidence and recent CASAC advice, the anticipated risks to other tree species that have not had their sensitivity to O₃ studied in a robust quantifiable way but that potentially have O₃ sensitivities that fall within the range for known species (see U.S. EPA, 2007, Table 7J-1 in Appendix 7J and Table 7J-2).

- **To what extent have important uncertainties in the evidence identified in the last review been reduced and/or new uncertainties emerged?**

As stated above, the ISA concludes that the new evidence confirms, strengthens and expands our understanding of O₃ effects on plants. Much of this new evidence is focused on the molecular and genetic level, providing important new mechanistic information that in some cases enhances our understanding of the complexity of the O₃-plant response. This information has, in general, reduced overall uncertainties at the subcellular and cellular scales (U.S. EPA, 2013, section 9.3.6).

Other recent information has also reduced some associated uncertainties regarding O₃ impacts at the whole plant, species, and ecosystem scales. Importantly, one key uncertainty related to the potential broader applicability of OTC-generated tree seedling E-R functions to estimate biomass loss under different (i.e., field) O₃ exposure conditions has been significantly reduced (U.S. EPA, 2013, section 9.6). Using recent field-based information available in the

current review, we conducted an analysis comparing OTC data with FACE data for one crop and one tree species (U.S. EPA, 2013, section 9.6.3.2). One comparison was done using soybean OTC data from the National Crop Loss Assessment Network (NCLAN)⁸ and more recent field-based data from the SoyFACE experiment, as discussed in section 5.3 below. The second was done using aspen seedling OTC data from the NHEERL-WED studies and more recent field-based data from the Aspen FACE study site. The result of the aspen analysis showed very close agreement between the biomass loss predictions based on OTC data and Aspen FACE observations, even when comparing the results of experiments that used different exposure methodologies, different genotypes, locations, and durations. The soybean analysis showed similar agreement between the OTC data and the SoyFACE experiment. Based on this analysis, the ISA concluded that “[o]verall, the studies at the Aspen FACE experiment were consistent with many of the open-top chamber (OTC) studies that were the foundation of previous O₃ NAAQS reviews” and that “[t]hese {recent} results strengthen the understanding of O₃ effects on forests and demonstrate the relevance of the knowledge gained from trees grown in OTC studies” (U.S. EPA 2013, p. 2-38, Section 9.6.3). The ISA additionally notes that with respect to aspen, “the function based on one year of growth was applicable to subsequent years” (of the six-year dataset) (U.S. EPA, 2013, section 9.6.3.2). This result is significant in that it shows that at least for this species, the seedling E-R function was able to predict responses beyond the seedling growth stage. While recognizing that some uncertainties remain for E-R functions for some individual species for which the database is relatively less robust, taken together, this information substantially reduces uncertainties associated with use of the tree seedling OTC-derived E-R functions to predict the response of tree seedlings in field settings and in some cases beyond the seedling growth stage. This information in combination with results from recent meta-analyses, as discussed above, reduces the uncertainties associated with potential impacts of other experimental factors on the O₃-plant response. Thus, in the current review, we have greater confidence than in the last review in using these E-R functions to estimate tree growth response outside the chamber setting (U.S. EPA, 2013, section 9.6.2; U.S. EPA, 2014a, section 6.2).

Several uncertainties are specific to studying or modeling O₃ impacts on trees, and derive from the long lifespan of trees, which can range from decades to centuries. Because most studies are designed to take place within an annual or 2-3 year timeframe, typically information is available for only a small fraction of the lifetime of a tree. Given this reality, one uncertainty

⁸ The NCLAN program was conducted from 1980 to 1987 at five different locations across the US. At each site, open top chambers were used to expose plants to O₃ treatments that represented the range of concentrations that occur in different areas of the world. The NCLAN focused on the most important U.S. agricultural crops (Heagle et al., 1989; <http://www.ars.usda.gov/Main/docs.htm?docid=12462>).

that remains is the degree to which exposures in a single year or over multiple years affect trees over the longer term. However, as discussed above, recent studies from the Aspen FACE site have reduced this uncertainty by providing additional evidence that demonstrates that exposures in one year have the potential to cause effects in a subsequent year (carry-over effects) and that the annual effects from exposures over multiple years have the potential to compound (U.S. EPA, 2013, 9.4.3, pp. 9-42 to 9-47). Such effects, when they cumulate from one or more years of elevated O₃ exposures, can lead to more serious longer-term impacts on growth, reproduction, recruitment, and competitive interactions within forest stands, and at larger spatial scales (U.S. EPA, 2013, p. 1-8), which would also have ramifications for any associated ecosystem services. In recognition of this recent evidence, the current CASAC Panel advised that “[a] 2% biomass loss is an appropriate scientifically based value to consider as a benchmark of adverse impact for long-lived perennial species such as trees, because effects are cumulative over multiple years” and stated that in its “scientific judgment, it is appropriate to identify a range of levels of alternative W126-based standards that includes levels that aim for not greater than 2% RBL for the median tree species” (Frey, 2014, p. 14). The CASAC further states that it “considers it significant that a similar value of 1% - 2% for tree seedling biomass loss was recommended previously by a consensus meeting of experts on ecological effects of ozone (Heck and Cowling, 1997)” (Frey, 2014, p. 14).

A related uncertainty comes from the limited evidence showing that sensitivity to O₃ can vary over the lifespan of trees and that this variation in growth-stage sensitivity is species-specific. For example, some species have been shown to be more sensitive during younger growth stages (i.e., seedling/sapling) while other species may be more sensitive as adults. Though a few studies have examined tree growth beyond the seedling stage (e.g., aspen) and in some species has been measured for both seedling and mature trees within a species (e.g., red oak), for most studied tree species it remains uncertain to what degree effects observed during one growth stage can be extrapolated to other growth stages. An analysis in the WREA comparing seedling to adult tree biomass loss, discussed in 5.2.2 below, informs our consideration of this remaining uncertainty (U.S. EPA, 2014a, section 6.2.1.1).

These uncertainties are taken into account when we consider how much weight to put on predictions of risks for known effects and how precautionary it is appropriate to be in light of the potential for cumulative effects from multiple year exposures that could reasonably be anticipated to occur, based on the evidence above.

- **To what extent does currently available evidence suggest locations where the vulnerability of sensitive species, ecosystems and/or their associated services to O₃-related effects on tree growth, productivity and carbon storage would have special significance to the public welfare?**

A number of different types of locations provide services of special significance to the public welfare. These services can flow in part or entirely from the vegetation that grows there (see also discussion under section 5.1 above). With respect to forested lands, the WREA notes that there are approximately 751 million acres of forest lands in the U.S., one third of which (250 million acres) is federally owned (U.S. EPA, 2014a, p. 5-15). In order to identify what types of forest locations have special significance from a public welfare perspective, it is first useful to consider the types of services that can flow from forested areas, and more specifically, from forested areas with trees that are sensitive to O₃. Some sensitive tree species provide public welfare benefits based on their cultural significance, and some lands are important to the public welfare for their cultural value. For example, tribal lands, federally designated Class I areas, non-Class I national parks and wilderness areas, and other areas set aside to provide similar public welfare benefits, are valued for their cultural services such as outdoor recreation and aesthetics. Appendix 5A includes a table listing known O₃-sensitive species, including some trees that have been identified as having cultural importance to some tribes (U.S. EPA, 2014a, section 6.4.2). Locations where these species are growing and are used by tribes to support cultural practices would thus be potentially vulnerable to impacts from elevated cumulative O₃ exposures, which could result in the loss of those associated cultural services, including those associated with sensitive tree species. Class I areas and other parks have also been afforded special federal protection to preserve services such as a healthy natural environment that provides for the enjoyment of these resources unimpaired for current and future generations, sustainable native plant and wildlife populations, and unique recreational opportunities. As mentioned above, 66 O₃-sensitive species have been identified on NPS and U.S. Fish and Wildlife Service lands).⁹ Other forested lands, both public and private, where trees are grown for timber production could also be at risk, especially in a single timber species stand that is sensitive to O₃ (i.e., Ponderosa pine) (see WREA section 6.3 and section 5.2.2 below). Urban forests provide a number of important services to the public, such as air pollution removal, cooling of the heat island effect, and beautification (U.S. EPA, 2014a, section 6.6.2). These

⁹ See <http://www2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf>

urban forests have also been recognized as important to environmental justice communities.¹⁰ Because urban forests can include O₃-sensitive trees (e.g., black cherry), O₃ exposures have the potential to reduce the services they provide. The WREA analysis of five urban case study areas, discussed below, quantified the O₃ impacts on air pollution removal and carbon sequestration in those urban areas (WREA, sections 6.6.2 and 6.7; section 5.2.2 below). Black cherry, for example, was one of the top ten occurring species in four of the five case study areas.

The above types of forested lands have clearly designated purposes or intended uses that help define the types of services that might be recognized as important from a public welfare perspective. In addition, other services provided by trees are potentially extremely valuable, but limited information is available to quantitatively value the extent of these services. Perhaps one of the most significant of these ecosystem services is climate regulation, which provides widespread and long-lasting public welfare benefits that the ISA determined is likely being compromised by the phytotoxic effects of O₃ on tree growth, productivity and carbon storage. By reducing the amount of carbon taken up by plants, more CO₂ is allowed to remain in the atmosphere where it potentially exacerbates the effects of climate change. In contrast to the location-specific discussion of services above, this service is potentially important to the public welfare no matter in what location the sensitive trees are growing, or what their intended current or future use. In other words, the benefit exists as long as the tree is growing, regardless of what additional functions and services it provides.

In addition to identifying forested locations that provide ecosystem services that are important to the public welfare, we must also consider to what extent there is the potential for O₃ to affect sensitive tree species growing on those lands to a degree sufficient to affect the public welfare. In so doing we first note that not all tree species are equally sensitive to O₃ and thus not equally vulnerable to current ambient O₃ exposures or those anticipated under various air quality scenarios. In further considering the degree to which O₃-induced impacts to ecosystem services associated with such trees might be expected to occur, we first focused on the 12 species of trees for which we have E-R functions. While all of these species provide goods and services that are important to the public welfare, not all species are equally sensitive to O₃ under recent ambient exposure conditions or conditions projected for adjusted air quality. Table 5-1 below (modified from WREA Table 6-13), provides a more detailed description of the ecosystem services provided by each of these species that benefit the public welfare. For the purposes of this

¹⁰ See <http://www.fs.fed.us/research/urban/environmental-justice.php> and Federal Interagency Working Group on Environmental Justice. (2011). Community-Based Federal Environmental Justice Resource Guide. August. Available at <http://www.epa.gov/environmentaljustice/resources/publications/interagency/resource-guide.pdf>

discussion we have ordered the species in the table to go in descending order from most to least sensitive (based on their predicted relative biomass loss (RBL) at a W126 of 15 ppm-hrs).

Table 5-1. O₃-Sensitive Trees, Their Uses and Relative Sensitivity

Tree Species	O ₃ Effect	Role in Ecosystems and Public Welfare Uses
Eastern Cottonwood¹¹ <i>Populus deltoides</i>	Biomass loss	Containers, pulp, and plywood Erosion control and windbreaks Quick shade for recreation areas Beaver dams and food
Black Cherry <i>Prunus serotina</i>	Biomass loss, Visible foliar injury	Cabinets, furniture, paneling, veneers, crafts, toys Cough remedy, tonic, sedative Flavor for rum and brandy Wine making and jellies Food for song birds, game birds, and mammals
Eastern White Pine <i>Pinus strobus</i>	Biomass loss	Commercial timber, furniture, woodworking, and Christmas trees Medicinal uses as expectorant and antiseptic Food for song birds and mammals Used to stabilize strip mine soils
Quaking Aspen <i>Populus tremuloides</i>	Biomass loss, Visible foliar injury	Commercial logging for pulp, flake-board, pallets, boxes, and plywood Products including matchsticks, tongue depressors, and ice cream sticks Valued for its white bark and brilliant fall color Important as a fire break Habitat for variety of wildlife Traditional native American use as a food source
Yellow (Tulip) Poplar <i>Liriodendron tulipifera</i>	Biomass loss, Visible foliar injury	Furniture stock, veneer, and pulpwood Street, shade, or ornamental tree – unusual flowers Food for wildlife Rapid growth for reforestation projects
Ponderosa Pine <i>Pinus ponderosa</i>	Biomass loss, Visible foliar injury	Lumber for cabinets and construction Ornamental and erosion control use Recreation areas Food for many bird species, including the red-winged blackbird, chickadee, finches, and nuthatches

¹¹ The E-R function for cottonwood is considered less robust because it is based on the results of a single gradient study (Gregg et al., 2003).

Tree Species	O ₃ Effect	Role in Ecosystems and Public Welfare Uses
Red Alder <i>Alnus rubra</i>	Biomass loss, Visible foliar injury	Commercial use in products such as furniture, cabinets, and millwork Preferred for smoked salmon Dyes for baskets, hides, moccasins Medicinal use for rheumatic pain, diarrhea, stomach cramps – the bark contains salicin, a chemical similar to aspirin Roots used for baskets Food for mammals and birds – dam and lodge construction for beavers Conservation and erosion control
Red Maple[^] <i>Acer rubrum</i>	Biomass loss	One of the most abundant and widespread in eastern U.S. Used for revegetation, especially in riparian buffers and landscaping, where it is valued for its brilliant fall foliage, some lumber and syrup production. Important wildlife browse food, especially for elk and white-tailed deer in winter, also leaves important food source for some species of butterflies and moths.
Virginia Pine <i>Pinus virginiana</i>	Biomass loss, Visible foliar injury	Pulpwood, strip mine spoil banks and severely eroded soils Nesting for woodpeckers, food for songbirds and small mammals
Sugar Maple <i>Acer saccharum</i>	Biomass loss	Commercial syrup production Native Americans used sap as a candy, beverage – fresh or fermented into beer, soured into vinegar and used to cook meat Valued for its fall foliage and as an ornamental Commercial logging for furniture, flooring, paneling, and veneer Woodenware, musical instruments Food and habitat for many birds and mammals
Loblolly Pine[*]	Biomass loss, visible foliar injury	Most important and widely cultivated timber species in the southern U.S. Furniture, pulpwood, plywood, composite boards, posts, poles, pilings, crates, boxes, pallets. Also planted to stabilize eroded or damaged soils. It can be used for shade or ornamental trees, as well as bark mulch. Provides habitat, food and cover for white-tailed deer, gray squirrel, fox squirrel, bobwhite quail and wild turkey, red-cockaded woodpeckers, and a variety of other birds and small mammals. Standing dead trees are frequently used for cavity nests by woodpeckers.
Douglas Fir <i>Pseudotsuga menziesii</i>	Biomass loss	Commercial timber Medicinal uses, spiritual and cultural uses for several Native American tribes Spotted owl habitat Food for mammals including antelope and mountain sheep
*Sensitivity categories added by EPA staff but not based on official designations.		

Sources: USDA-NRCS, 2013; Burns, 1990; Hall and Braham, 1998. [^]Red maple information from http://www.na.fs.fed.us/pubs/silvics_manual/volume_2/acer/rubrum.htm. *Loblolly pine use information from <http://www.ncsu.edu/project/dendrology/index/plantae/vascular/seedplants/gymnosperms/conifers/pine/pinus/australes/loblollypine/html>.

While we recognized that there are important ecosystem services provided by those species that are less sensitive to O₃, those species would likely receive less benefit from additional protection below the current standard. In contrast, the other species would likely see improvements in their associated ecosystem services, some significantly, from an improvement in air quality. However, at the highest end of the known sensitivity spectrum, there are different issues that must be considered when evaluating the usefulness of this information in answering the above questions. The E-R function that is available for cottonwood is based on the results of a single gradient study (Gregg et al., 2003) and is considered less robust than the other E-R functions developed in OTCs. That combined with its apparent extreme response to O₃ prompted CASAC to advise the Administrator to not place too much emphasis on cottonwood in the review of the secondary standard (Frey, 2014, p. 10). As a result, we have decided it would not be appropriate to use the cottonwood biomass loss estimates when considering what levels of W126 should be considered protective of median species biomass loss (see Table 5C-3).

However, in this discussion of ecosystem services, we believe it is important to include cottonwood, given the many ecosystem services cottonwood provides (see Table 5-1 above), and several unique features that potentially make it and its associated ecosystem goods and services particularly vulnerable to impacts from O₃. Specifically we note that cottonwood: 1) is often found growing along streams in riparian zones under well watered conditions that make it more susceptible to injury than species growing in areas that experience drier conditions in conjunction with higher O₃ exposures; 2) can be the only tree species growing in certain types of ecosystems, thus providing important habitat for some organisms; 3) is fast growing and used commercially for pulpwood, manufacturing furniture and as a possible source for energy biomass (Burns and Hankola, 1990); 4) has provided limited, though still uncorroborated, evidence of the potential for the existence of extremely sensitive plant species which can reasonably be anticipated to exist and that could be impacted at similar cumulative exposures. With regard to the latter, we observe that CASAC also expressed the view that it “should be anticipated that there are species of vegetation that are highly sensitive to ozone that do not have E-R functions, and others that are insensitive. It is scientifically justifiable to extrapolate from the known E-R curves, assuming that they are representative of the un-sampled population” (Frey, 2014, p. 16). We also note that upon revisiting the available literature in the ISA following CASAC’s review of the second draft WREA and PA, we found two studies on a related European species (*Populus nigra*) that showed that this species had an O₃ sensitivity that appears similar in magnitude to the U.S. cottonwood (*Populus deltoides*) based on its response for other growth endpoints as compared with the response of the other study species (Bortier, et al., 2000; Novak, et al., 2007) (U.S. EPA, 2006, AX9, pp. 91, 240; U.S. EPA, 2014a, Table 6-5).

This additional limited evidence of *Populus* seedling/sapling growth response, though not directly comparable to the U.S. study (Gregg et al., 2003) with respect to species, exposure methods, measurement endpoints and exposure values, does, in our judgment, lend some support to the observed magnitude of the reported U.S. cottonwood response.

In addition to the information provided here on these 12 species, we note that there are many other species of trees with known or suspected O₃-sensitive vegetation, such as those included in the 66 species identified on NPS and US Fish and Wildlife Service lands),¹² species used in the USFS biomonitoring network, and various ornamental and agricultural species (i.e., Christmas trees, fruit and nut trees) that currently provide ecosystem services important to the public welfare, but whose vulnerability to impacts from O₃ on tree growth, productivity and carbon storage has not been sufficiently characterized to allow it to directly inform our quantitative assessments (U.S. EPA, 2014a, Chapter 6; Abt Associates, 1995). However, as noted by CASAC, the anticipated impacts on these and other unstudied species should not be ignored or assumed insignificant. It is more likely that the range of O₃ sensitivities found in the studies tree species likely reflects the range of O₃ sensitivities in all tree species.

Other factors that should be taken into account when considering the potential degree to which O₃ might affect the ecosystem service flows from forested ecosystems are 1) the type of stand or community in which the sensitive species is found (i.e., single species versus mixed canopy); 2) the role or position the species has in the stand (i.e., dominant, sub-dominant, canopy, understory); 3) the O₃ sensitivity of the other co-occurring species (O₃ sensitive or tolerant); 4) environmental factors (drought or well watered conditions, other stressors).

In light of the above discussion, it is clear that there are numerous locations where the vulnerability of O₃-sensitive tree species to impacts from O₃ on tree growth, productivity and carbon storage and their associated ecosystems and services could have special significance to the public welfare. Confirmation that the American public values healthy forests is provided in the WREA, which shows that Americans are willing to pay to protect forests from the damaging effects of air pollutants (U.S. EPA, 2014a, Chapter 5, pp. 5-16). Data provided by the National Survey on Recreation and the Environment (NSRE) indicates that Americans have very strong preferences for the non-use values of existence, bequest, and option services related to forests. Studies (Haefele et al., 1991, Holmes and Kramer, 1995) assess willingness-to-pay (WTP) for spruce-fir forest protection in the southeastern U.S. from air pollution and insect damage and confirm that the non-use values held by the survey respondents were in fact greater than the use or recreation values. The results of this survey showed that median household WTP was

¹² See <http://www2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf>

estimated to be roughly \$29 (in 2007 dollars) for the minimal protection program and \$44 for the more extensive program. After decomposing their value for the extensive program into use, bequest, and existence values, the results were 13 percent for use value, 30 percent for bequest, and 57 percent for existence value (See U.S. EPA, 2014a, Table 5-6). These services may be at risk in areas where O₃-sensitive trees are found.

- **To what extent does the available evidence indicate the occurrence of O₃-related effects on forest growth, productivity and carbon storage attributable to cumulative exposures lower than previously established or that might be expected to occur under the current standard?**

The evidence base available in this review, as in the previous review, indicates that O₃-induced effects on tree growth, productivity and carbon storage can occur across a range of cumulative exposures, including those lower than previously established and that would be expected to occur under the current standard. In reaching this determination, we first consider the 11 tree seedling species for which robust E-R functions have been developed from the extensive evidence base of O₃-induced growth effects that was also available and relied upon in the previous review. Each of these species were studied in OTCs, with most species studied multiple times under a wide range of exposure and/or growing conditions, with separate E-R functions developed for each species/exposure condition/growing condition scenario combination or case. Using all the information available from these multiple study cases (52 cases in all), a robust composite E-R function was developed for all species combined and separate individual composite functions were derived for each species using cases that were available on individual species. These species-specific composite E-R functions have been successfully used to predict tree seedling species biomass loss response over a range of cumulative exposure conditions. Figure 5-1A, B below, which includes the 11 robust composite E-R functions available in the last review and the E-R for cottonwood (also described in U.S. EPA 2013, section 9.6.2 and U.S. EPA, 2014a, section 6.2, Table 6-1 and Figure 6-2), illustrates the appreciable variability in sensitivity that exists across the 12 studied species, and shows that biomass loss can occur over a wide range of cumulative exposures, including those previously established. This figure further shows that for some species biomass loss would be predicted to occur at very low cumulative exposures that can occur under air quality conditions that meet or are below the current standard (see Table 5-2 below). While we put less emphasis on cottonwood (as explained above), we do note that in answering the question above, it does provide limited recent evidence of the potential for effects of a greater magnitude and at lower cumulative exposures to occur than those considered in the last review and at exposures that would be allowed by the current standard. To the extent that such effects could be anticipated,

the cumulative exposures that could be allowed by the current secondary standard would not be protective.

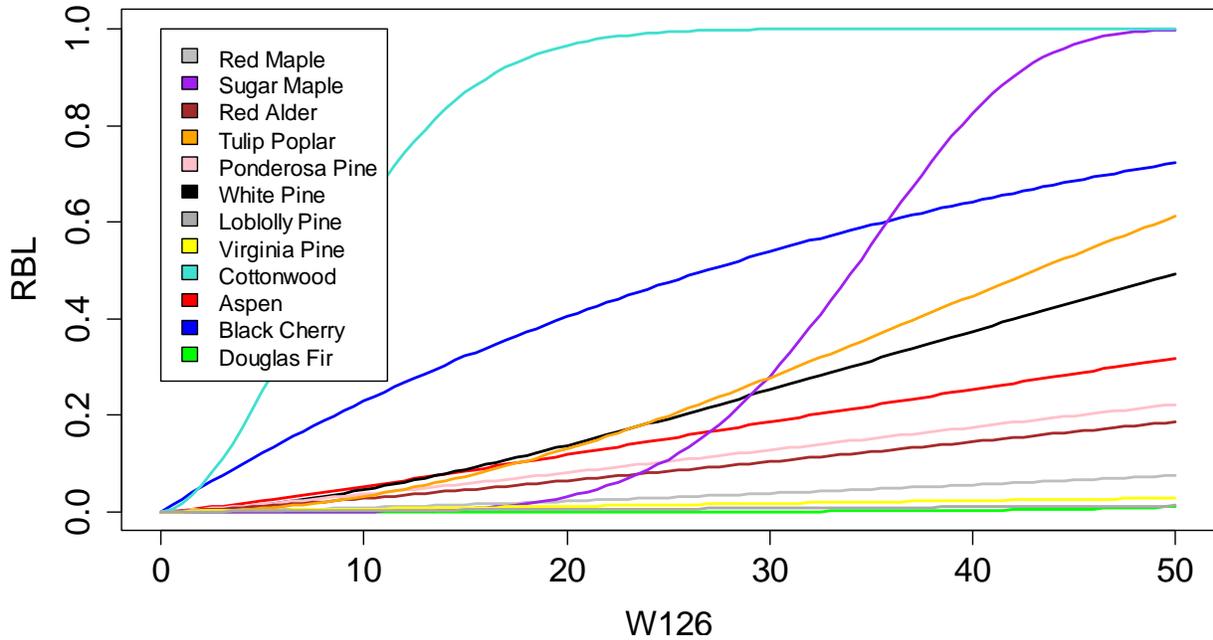
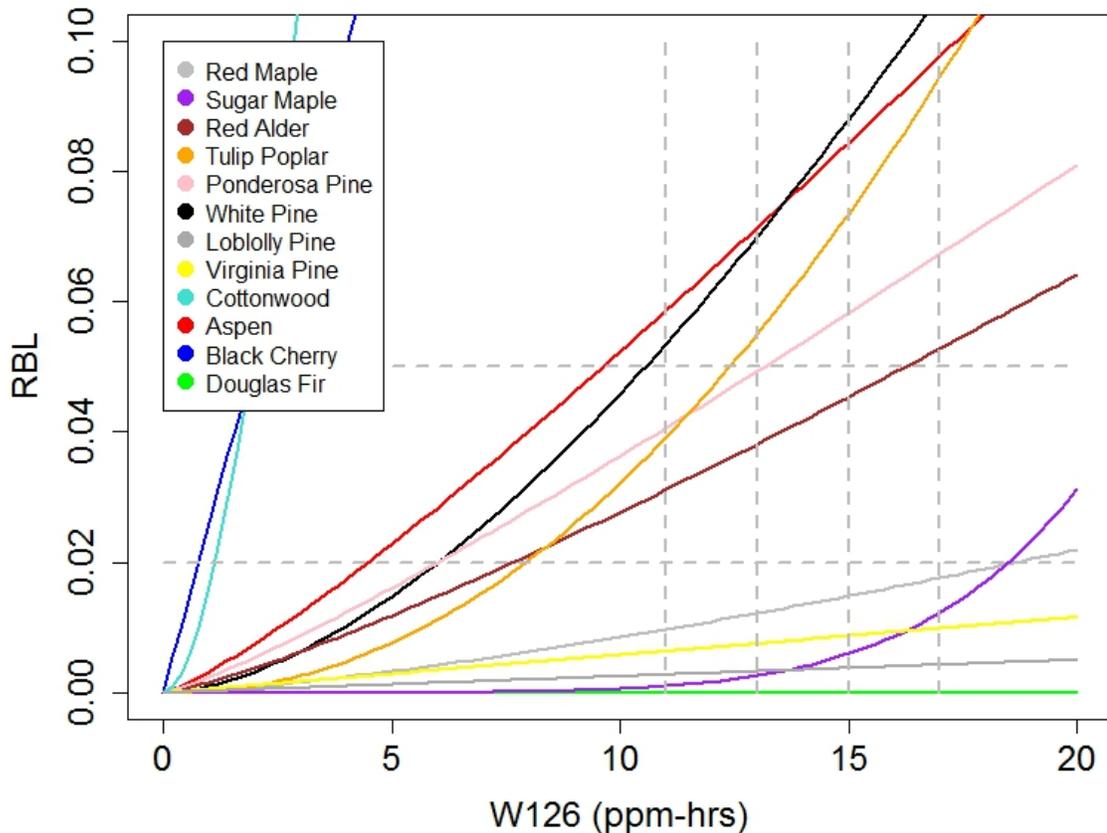


Figure 5-1. A) Relative biomass loss in seedlings for 12 studied species using composite functions in response to seasonal O₃ concentrations in terms of seasonal W126 index values, Y-axis scale for RBL values represents 0% up to 100% (U.S. EPA 2014a, Figure 6-2).



B) Expanded view of relative biomass loss in seedlings for 12 studied species using composite functions in response to seasonal O₃ concentrations in terms of lower range of seasonal W126 index values, Y-axis scale for RBL values represents 0% up to 10% (U.S. EPA 2014a, Figure 6-2).

In further answering the question above, we note CASAC’s advice that a 6% median RBL is unacceptably high, and that the 2% median RBL is an important benchmark to consider. Based on the information above, the median RBL is at or below 2% at the lowest W126 level assessed, 7 ppm-hrs. As the W126 level is incrementally increased, median RBL also increases incrementally, so that at W126 index values of 9, 11, 13, 15, 17, 19 and 21, the median RBL increases to 2.4%, 3.1%, 3.8%, 4.5%, 5.3%, 6.0% and 6.8%, respectively. Based on air quality analyses of 2009-2011 (Appendix 2B), there are approximately 342, 199, 92, 43, 24, 9, 3 and 0 monitors with 3-year average W126 index values above 7, 9, 11, 13, 15, 17, 19 and 21 ppm-hrs when meeting the current standard. We note that these counts of monitors are based on those meeting the current standard and that there are many monitors for the 2009-2011 period that do not meet the current standard and also are above the W126 values of 7-21 ppm-hrs.

We also consider it informative to examine the individual species responses and RBL over the same W126 range. We first note, based on Figure 5-1(B) above that over the range of 7 to 17 ppm-hrs, 5 species maintain RBLs of less than 2%. These more tolerant species include

Douglas fir, loblolly pine, Virginia pine, sugar maple and red maple. Two of these species (red maple and sugar maple) are estimated to have RBL levels above 2% at a W126 of 21. Black cherry, the most sensitive of the remaining six species, has RBL ranging from 35.57% at W126 of 17 down to 16.67% at the W126 index value of 7 ppm-hrs.

Additional evidence of the potential for O₃-induced effects on tree seedling growth, productivity and carbon storage occurring under air quality scenarios allowed by the current standard is shown in Table 5-2 below. Specifically, all monitor sites in Table 5-2 have 3-year 8-hour average values that meet the current standard, ranging from 67 to 75 ppb, have 3-year average W126 index values that are above 15 ppm-hrs, and are located in Class I areas. Across these 22 Class I areas, the highest single-year W126 index values for these three-year periods ranged from 17.4 to 29.0 ppm-hrs. In 20 of the areas, distributed across eight states (AZ, CA, CO, KY, NM, SD, UT, WY) and four regions (west, southwest, west/north central and central), this range was 19.1 to 29.0 ppm-hrs, exposure values for which the corresponding median species RBL estimates equal or exceed 6%, which CASAC termed “unacceptably high”. In addition, given that other environmental factors can influence the extent to which O₃ may have the impact predicted by the E-R functions in any given year, we also note that the highest three year periods, that include these highest annual values for the 21 areas, are at or above 19 ppm-hrs, ranging up to 22.5 ppm-hrs (which the median species RBL estimate is above 7%). Additionally, the highest three-year average W126 index value for each of the 22 areas (during periods meeting the current standard) was at or above 19 (ranging up to 22.5 ppm-hrs) in 11 areas, distributed among five states in the west and southwest regions (U.S. EPA, 2014c, Table 5-2, Appendix 5B).

In addition, as data permit, Table 5-2 shows the studied tree species that are found in each of these Class I areas. Quaking aspen and ponderosa pine are two tree species that are found in many of these 22 parks and have a sensitivity to O₃ exposure that places them near the middle of the group for which E-R functions have been established. In the areas where ponderosa pine is present, the highest single year W127 index values ranged from 18.7 to 29.0 and the highest 3-year average W126 values in which these single year values are represented ranged from 15 to 22.5, with these three-year values above 19 ppm-hrs in eight areas across five states. The ponderosa pine RBL estimates for 29 and 22.5 ppm-hrs are approximately 12% and 9%, respectively. In areas where quaking aspen is present, the highest single year W127 index values ranged from 19.2 to 26.7 ppm-hrs and the highest 3-year average W126 values in which these single year values are represented ranged from 15.0 to 22.2, with values above 19 ppm-hrs in eight areas across five states. The quaking aspen RBL estimates for 26.7 and 22.2 ppm-hrs are approximately 16% and 13%, respectively. Based on this, we note growth effects associated with

exposure concentrations occurring during periods where the current standard is met in many of these Class I areas. On the basis of such information, Table 5-2 provides evidence of the potential for significant growth loss in locations where ambient conditions meet the current standard.

Table 5-2. O₃ concentrations in Class I areas during period from 1998 to 2012 that met the current standard and where three-year average W126 index value was at or above 15 ppm-hrs.*

Class I Area	State / County	Design Value (ppb)*	3-year Average W126 (ppm-hrs)* (# ≥ 19 ppm-hrs, range)	Annual W126 (ppm-hrs)* (# ≥ 19 ppm-hrs, range)	Number of 3-year Periods
Bandelier Wilderness Area QA, DF, PP	NM / Sandoval	70-74	15.8-20.8 (2, 20.0-20.8)	12.1-25.3 (4, 19.2-25.3)	8
Bridger Wilderness Area QA, DF	WY / Sublette	69-72	15.1-17.4	9.9-19.2 (1, 19.2)	5
Canyonlands National Park QA, DF, PP,	UT / San Juan	69-73	15.0-20.5 (2, 19.8-20.5)	9.9-24.8 (5, 19.3-24.8)	9
Carlsbad Caverns National Park PP	NM / Eddy	69	15.0-15.3	8.6-26.7 (1, 26.7)	3
Chiricahua National Monument DF, PP	AZ / Cochise	69-73	15.7-18.0	13.2-21.6 (2, 19.3-21.6)	7
Grand Canyon National Park QA, DF, PP	AZ / Coconino	68-74	15.6-22.2 (7, 19.2-22.3)	11.3-26.7 (7, 19.8-26.7)	12
John Muir Wilderness Area QA, DF, PP	CA / Inyo	71-72	16.5-18.6	10.1-25.8 (2, 23.9-25.8)	3
Lassen Volcanic National Park DF, PP	CA / Shasta	75	15.3	13.6-18.7 (1, 18.7)	1
Mammoth Cave National Park BC, C, LP, RM, SM, VP, YP	KY / Edmonson	74	15.9	12.5-22.5 (1, 22.5)	1
Mesa Verde National Park DF	CO / Montezuma	67-73	15.5-21.0 (2, 19.0-21.0)	10.7-23.6 (4, 19.7-23.6)	10
Mokelumne Wilderness Area DF, PP	CA / Amador	74	17.6	14.8-22.6 (1, 22.6)	1
Petrified Forest National Park	AZ / Navajo	70	15.7	12.9-19.2 (1, 19.2)	1
Pinnacles National Monument	CA / San Benito	74	15.1	13.1-17.4	1
Rocky Mountain National Park QA, DF, PP	CO / Boulder	73-75	15.1-19.3 (1, 19.3)	9.5-25.1 (5, 20.7-25.1)	6
	CO / Larimer	74	15.0-18.3	11.1-25.8 (3, 19.1-25.8)	3
Saguaro National Park DF, PP	AZ / Pima	69-74	15.4-18.9	11.0-23.1 (3, 20.0-23.1)	6
Sierra Ancha Wilderness Area DF, PP	AZ / Gila	72-75	17.9-22.4 (3, 20.2-22.4)	14.8-27.5 (4, 20.3-27.5)	4
Superstition Wilderness Area PP	AZ / Maricopa	75	22.4 (1, 22.4)	14.5-28.6 (2, 27.4-28.6)	1

	AZ / Pinal	73-75	18.7-22.5 (2, 20.9-22.5)	14.8-29.0 (3, 22.6-29.0)	3
Weminuche Wilderness Area ^{QA, DF, PP}	CO / La Plata	70-74	15.0-19.1 (1, 19.1)	10.9-21.0 (2, 20.8-21.0)	5
West Elk Wilderness Area ^{QA, DF}	CO / Gunnison	68-73	15.6-20.1 (1, 20.1)	12.9-23.9 (3, 21.1-23.9)	8
Wind Cave National Park ^{QA, PP}	SD / Custer	70	15.4	12.2-20.6 (1, 20.6)	1
Yosemite National Park ^{QA, DF, PP}	CA / Tuolumne	73-74	20.7-20.8 (2, 20.7-20.8)	19.7-22.1 (4, 19.7-22.1)	2
Zion National Park ^{QA, DF, PP}	UT / Washington	70-73	17.8-21.1 (2, 20.3-21.1)	14.9-24.2 (5, 19.3-24.2)	4
<p>*Based on data from http://www.epa.gov/ttn/airs/airsaqs/detaildata/downloadaqsdta.htm (US EPA, 2014c). W126 values are truncated after first decimal place. Superscript letters refer to species present for which E-R functions have been developed. QA=Quaking Aspen, BC=Black Cherry, C=Cottonwood, DF=Douglas Fir, LP=Loblolly Pine, PP=Ponderosa Pine, RM=Red Maple, SM=Sugar Maple, VP=Virginia Pine, YP=Yellow (Tulip) Poplar. Sources for presence of species include U.S. Department of Agriculture databases in 2014 http://www.fs.fed.us/foresthealth/technology/nidrm2012.shtml, http://plants.usda.gov, http://www.wilderness.net/printFactSheet.cfm?WID=583</p>					

In answering the above question, we note that less information is available from field-based studies (e.g., FACE, gradient) due to the absence of robust E-R functions, the limited range of exposure scenarios evaluated, and unavailability of study exposures in terms of daily 8-hour averages.

Taken together, the information described above provides consistent and coherent evidence that O₃-induced impacts on tree seedling growth, productivity and carbon storage are occurring at cumulative exposures allowed by the current standard. In particular, this information provides clear evidence of the potential for significant growth loss in Class I locations where ambient conditions meet the current standard.

5.2.2 Exposure/Risk-based Considerations

The WREA presents a number of quantitative analyses of exposure and risk related to tree growth, productivity and carbon storage intended to inform our consideration of exposure and risk associated with the current and potential alternative standards (Table 5-3 below; U.S. EPA, 2014a, Chapter 6).

Table 5-3. Exposure, risk and ecosystem services analyses related to tree growth, productivity and carbon storage.

	Species Level Effects	Ecosystem Level Effects	Ecosystem Services
WREA estimates^A	Derivation of median biomass loss values from individual species E-R functions Comparison of tree seeding growth to that of mature trees	Percent of total geographic area ^B with annual relative biomass loss above 2% Number of assessed Class I areas with annual relative biomass loss above 2%	<ul style="list-style-type: none"> • Economic surplus to timber producers and consumers (WREA, Table 6-12) • Carbon storage, nationally (WREA, Table 6-19) • Carbon storage, in 5 urban areas (WREA, Table 6-21) • Air pollutant removal in 5 urban areas (WREA, Table 6-22)
^A See WREA Chapter 6 (U.S. EPA, 2014a).			
^B The total geographic area includes only the contiguous U.S.			

The relevant quantitative exposure and risk analyses for tree biomass loss, productivity and carbon storage include:

- 1) Species-specific and median biomass loss estimates from composite functions.
 - 2) National-scale assessments for: a) basal area weighted relative biomass loss for tree seedlings; b) timber production; c) carbon sequestration.
 - 3) Case study-scale assessments for: a) carbon sequestration; b) air pollution removal.
- **For what air quality scenarios were exposures and risks estimated? What approaches were used to estimate W126 exposures for those conditions? What are associated limitations and uncertainties?**

Quantitative exposure and risk analyses were conducted to evaluate the effects on tree growth, productivity and carbon storage, and associated ecosystem services, that would be predicted under five air quality scenarios (recent ambient, just meeting the current standard, and W126 potential alternative standards of 15, 11, and 7 ppm-hrs). Table 5-5 summarizes the methodology used to develop the quantitative estimates for each of the five air quality scenarios. In general, this methodology involved two steps. The first is derivation of the average W126 index value (across the three years) at each monitor location. This value is based on unadjusted data for recent conditions and adjusted concentrations for the four other scenarios. The development of adjusted concentrations was done for each of 9 regions independently (see U.S. EPA, 2014a, section 4.3.4.1). In the second step, national-scale spatial surfaces (W126 index values for each 12 x 12 km² grid cell from the Community Multi-scale Air Quality (CMAQ) model) were created using the monitor-location values and the Voronoi Neighbor Averaging (VNA) spatial interpolation technique (details on the VNA technique are presented in U.S. EPA, 2014a, Appendix 4A).

Table 5-4. Summary of methodology by which national surface of 3-year average W126 index values was derived for each air quality scenario.

Scenario	Development of W126 index values for Each Air Quality Scenario	
	Monitor-location-specific calculations and any model-based adjustment	Derivation of national surface of average W126 index values
Recent Conditions (2006-2008)	An annual W126 index value is calculated for each year at each monitor location, using the highest 3-month period. A location-specific 3-year W126 was calculated by averaging annual W126 index values from 3 consecutive years which may have used different 3-month periods.	The VNA method is applied to the monitor-location average W126 index values to create a national distribution of W126 index values within model grid-cells for each scenario.
Current Standard	2006-2008 hourly O ₃ concentrations at each monitor location are adjusted ^A to create a three year record of O ₃ concentrations that just meets the current standard (see WREA, section 4.3.4). This results in air quality at other monitors well below the level of the controlling monitor. A seasonal W126 index value is calculated for each year at each monitor location using the same 3-month period for each year (which is the highest as a 3-yr average and is highest in at least one of the years). A location-specific average is derived from these three index values.	
Average W126 Index of 15 ppm-hrs	First, hourly O ₃ concentrations were adjusted to just meet the current standard. Second, hourly O ₃ concentrations at each monitor location, within each modeling region, are adjusted to create a record for which the highest location-specific average index value in the region (the controlling location) just meets the scenario target index value.	
Average W126 Index of 11 ppm-hrs	A seasonal W126 index value is calculated for each year (of 2006-2008 period) at each monitor location, using the same 3-month period for each year (which is the highest in at least one of the years). A location-specific average is derived from these three index values.	
Average W126 Index of 7 ppm-hrs		
^A The model-based adjustment approach is based on regional emission reduction scenarios at monitor sites followed by spatial interpolation for broader spatial coverage. See WREA, chapters 3 and 4, and Appendix 4A.		

During the recent conditions period (2006 through 2008), the average W126 index values (across the three-year recent conditions period) at the monitor locations ranged from below 5 ppm-hrs to 48.6 ppm-hrs (U.S. EPA 2014a, Figure 4-4 and Table 4-3). Across the nine modeling regions, the maximum average W126 index values ranged from 48.6 ppm-hrs in the west region down to 6.6 ppm-hrs in the northwest region. After adjusting the 2006-2008 data to just meet the current standard in each region, the region-specific maximum values range from 18.9 ppm-hrs in the west region to 2.6 ppm-hrs in the northeast region (U.S. EPA, 2014a, Table 4-3). After application of the VNA technique to the current standard scenario monitor location values, the average W126 index values were below 15 ppm-hrs across the national surface with the

exception of a very small area of the southwest region (near Phoenix) where the average W126 index values was near or just above 15 ppm-hrs. Thus, it can be seen that application of the interpolation method to estimate W126 index values at the centroid of every 12 x 12 km² grid cell rather than only at each monitor location results in a lowering of the highest values.

- **What are the nature and magnitude of exposure- and risk-related estimates for tree growth, productivity, and carbon storage under recent conditions or conditions remaining upon meeting the current standard? To what extent are these exposures and risks important from a public welfare perspective?**

In answering the above question, the WREA performed a number of different assessments to estimate the exposures and risks predicted under the five air quality scenarios across a range of spatial scales. These assessments include those for individual species response as well as the median species response for studied species ranging from the county scale up to estimations of exposures and risks to ecosystem services associated with forests at the urban, park and national scales.

Before conducting the exposure and risk assessments, the WREA examined three approaches for characterizing the median response, as shown in Figure 5-2 below (U.S. EPA, 2014a, section 6.2.1.2 and Figure 6-5). These approaches use the 11 robust E-R functions for tree seedlings from the OTC research and the cottonwood E-R function. For some species, only one study was available (e.g., red maple), and for other species there were as many as 11 studies available (e.g., ponderosa pine). The first approach plotted the median (red line) of all 52 tree seedling studies available (across the 12 species). In this first approach, species with multiple studies would be represented more than once in the median. The second approach characterized the median (green line) by combining the composite E-R functions, when available for species with multiple studies, with the E-R functions for species with a single study available¹³ for each of the 12 tree species. In this second approach, each species is represented only once in the median. The third approach used a stochastic sampling method to randomly select a single E-R function from the studies available for each of the 12 species. The process was repeated 1,000 times (grey lines), and the median value was plotted for biomass loss values of 1% to 7%, and 10% (red dots; the bar associated with each median point denotes the 25th and 75th percentile values). This third approach illustrates the effect of within-species variability on estimates of the median response. The median W126 index values are similar when using the first two approaches; however, the median value is higher when within-species variability is included (U.S. EPA, 2014a, section 6.2.1.2). Across these three approaches, the median seasonal W126 index value for which a two percent biomass loss is estimated in seedlings for the studied species

¹³ For some species, only one study was conducted so that E-R function was used.

ranges between approximately 7 and 14 ppm-hrs. Using the green line, the seasonal W126 index value for which a two percent biomass loss is estimated in seedlings for the median of the composite functions for the 12 studied species is approximately 7 ppm-hrs. After reviewing these three approaches, the CASAC stated “[t]he Monte Carlo analysis (red dots, Figure 5-2) should not be used in evaluating the effect of ozone on RBL of tree seedlings. This analysis overemphasizes the species for which relatively few E-R functions are available, is biased toward the few less sensitive response functions available for some individual species, makes unsupported assumptions regarding the representativeness of available response functions, and confounds intra- and inter-species variability in unquantifiable ways. We favor using a measure of central tendency of the data, specifically the median across species (the green line in Figure 5-2). This analysis provides the median of best available estimates within each species, and the median across species with all species treated equally” (Frey, 2014, p. 14). Given this advice, in selecting an approach for use in later analyses, we have chosen to use the green line because the approach that generated it incorporates all the information in a way that gives equal weight to each studied species without losing any of the available data.

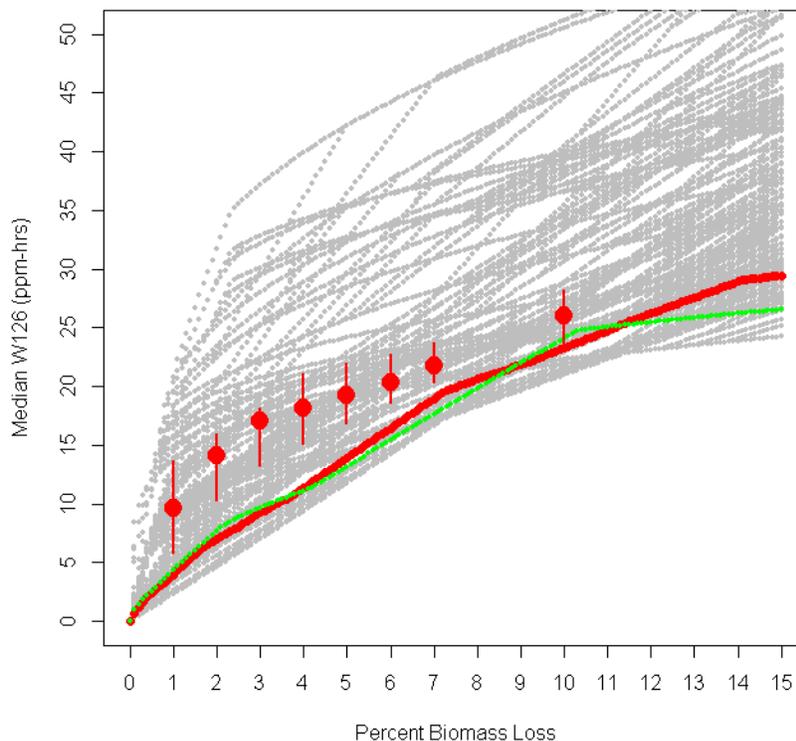


Figure 5-2. Relationship of tree seedling percent biomass loss with seasonal W126 index. (From U.S. EPA 2014a, Figure 6-5)

The WREA used the E-R functions for 12 species described above with information on the distribution of those species across the U.S., and average W126 exposure estimates to

estimate relative biomass loss for each of the studied species for each national air quality scenario (U.S. EPA, 2014a, section 6.2.1.3 and Appendix 6A). For example, the estimates of relative biomass loss of ponderosa pine for air quality adjusted to just meet the current standard are illustrated in Figure 5-3 below. While relative biomass loss below 2% is estimated for most areas where this species is found, estimates in some areas of the southwest fall above 2% biomass loss (U.S. EPA 2014a, Figure 6-8 and Appendix 6A).

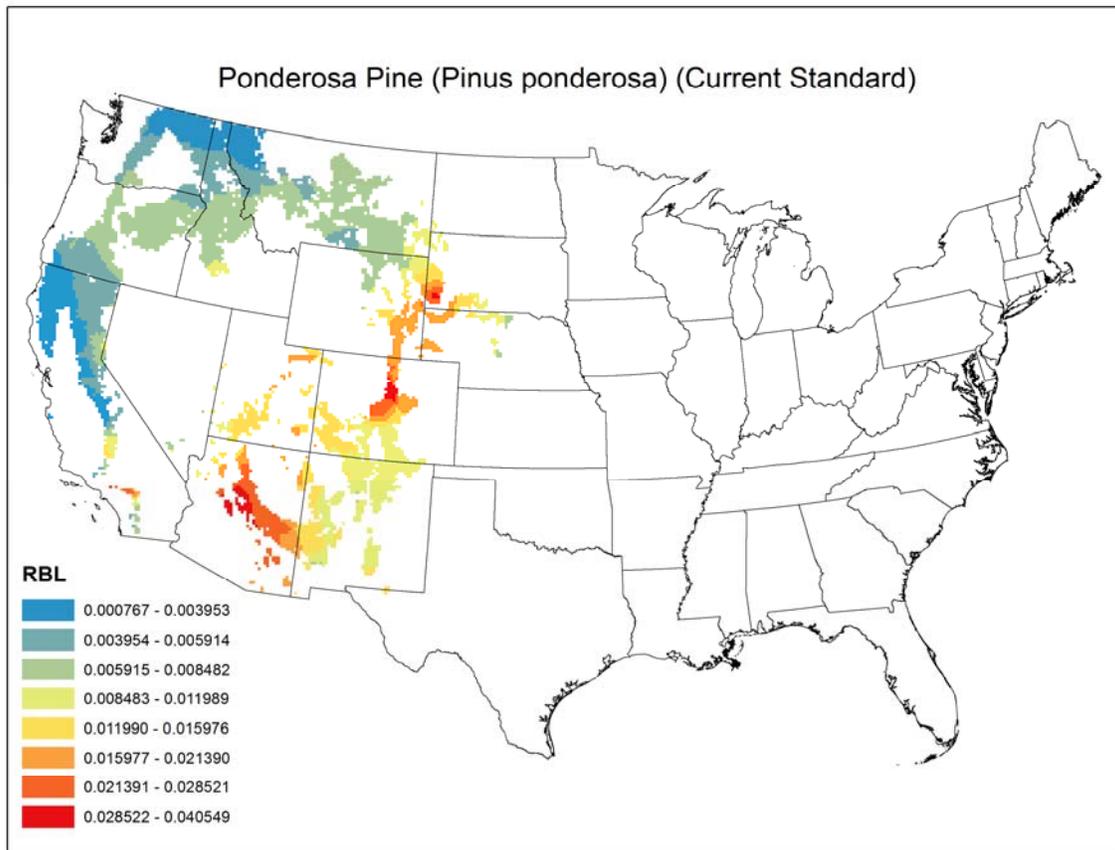


Figure 5-3. Relative biomass loss of Ponderosa Pine for air quality adjusted to just meet the current standard (U.S. EPA 2014a, Figure 6-8).

The WREA also developed national-scale estimates of O₃ biomass ecosystem-level impacts considering the 12 studied species together (U.S. EPA 2014a, section 6.8, Table 6-25). This was done using the species-specific biomass loss E-R functions, information on prevalence of the studied species across the U.S., and a weighting approach based on proportion of the basal area within each grid cell that each species contributes. The RBL values for multiple tree species were weighted by their basal area and combined into a weighted RBL value (wRBL). The wRBL is intended to inform our understanding of the potential magnitude of the ecological effect that could occur in some ecosystems. Specifically, the more basal area that is affected in a given

ecosystem, the larger the potential ecological effect. A wRBL value for each grid cell is generated by weighting the RBL value for each studied tree species found within that grid cell by the proportion of basal area it contributes to the total basal area of all tree species within the grid cell, and then summing those individual wRBLs. The percent of total basal area that exceeds a 2% weighted relative biomass loss in the recent conditions scenario is 10.1% (U.S. EPA 2014a, Table 6-25). Based on the average W126 index values estimated for the air quality scenario just meeting the current standard across the contiguous U.S., the WREA estimates 0.2% of the total geographic area to have a wRBL above 2% based on the E-R functions for the 11 tree species and 0.8% based on 12 tree species (U.S. EPA 2014a, Table 6-25). We recognize that these estimates are likely biased low as there may be other unstudied O₃-sensitive tree species in some areas that are also being impacted at those levels. Further, this analysis does not take into account the effects of competition, which could further increase biomass loss in O₃-sensitive species.

In addition, the WREA characterized the number of counties where there would be one or more studied tree species showed a 2% biomass loss (U.S. EPA, 2014a, Table 6-7), which is shown in Table 5-5 below. This is consistent with CASAC advice that “rather than focusing solely on the median relative biomass loss (RBL), the number of counties containing sensitive tree species that are expected to have growth loss of greater than 2% should be quantified.” (Frey, 2014, p. 11). These data are presented as the number of U.S. counties in which any of the 12 studied tree species exceeds 2% RBL, further categorized by the number of studied species that exceed that benchmark for each of the five air quality scenarios using 3-year average W126 index values. In addition, this table provides the total number of counties (out of 3,109 total counties) for each exposure scenario with at least one species exceeding 2% RBL and the number of counties where the median of the composite functions for each species exceeds 2% RBL. The maximum number of species that exceed 2% RBL in any one county is five species, which only occurs under recent O₃ conditions. After meeting the current standard, the maximum number of species in any one county is four. Because cottonwood and black cherry are highly sensitive species and to provide a reference for the effect of these species, the data are also presented excluding cottonwood and excluding cottonwood and black cherry.

This information shows that a number of counties have more than one O₃-sensitive species growing in it, potentially together in the same forest stands, whose RBLs are above 2%. Under recent conditions, the proportion of total counties of 3,109, with 1 or more species with an RBL greater than 2% is 89% (2,761 counties) for the scenario inclusive of cottonwood and black cherry. When air quality is adjusted to just meet the current standard, that proportion dropped to 74% (2,313 counties). When air quality is adjusted to just meet a 3-year average W126 index

value of 15, 11 and 7 ppm-hrs, the proportion is 73%, 72% and 71%, respectively. For median RBL values, under recent conditions, 72% of the counties have median RBLs above 2%. When air quality is adjusted to the current standard, that proportion drops to 22% and further decreases to 20% for air quality adjusted to just meet a 3-year average W126 level of 7 ppm-hrs.

Given CASAC's advice to put less emphasis on cottonwood, we focus on the rows of this table that excluded cottonwood. Under recent air quality conditions, the proportion of counties with 1 or more species with an RBL greater than 2% is 78% (2,418 counties). As air quality is adjusted to just meet the current standard and the alternative W126 index value of 7 ppm-hrs, this number drops to 62% and 58%, respectively. In addition, under recent conditions, 52% of the counties have median RBLs above 2%. When air quality is adjusted to the current standard, that proportion drops to 8% and further decreases to 6% for air quality adjusted to just meet a 3-year average W126 level of 7 ppm-hrs.

Table 5-5 also provides information on the influence of black cherry on the estimates and shows that black cherry is a very sensitive species that is widespread in the Eastern U.S. We note that of the 1,929 counties estimated to have 1 or more species with an RBL greater than 2% when meeting the current standard, 1,805 of those counties are estimated to have black cherry as the only specie estimated to experience this level of biomass loss. With respect to median RBL values, of the 239 counties estimated to have a median RBL above 2% when meeting the current standard, 203 of those counties have a RBL above 2% because of the presence of black cherry.

Table 5-5. Number of Counties with Tree Species Exceeding 2% Relative Biomass Loss.

Number of species exceeding 2% RBL	Number of Counties (3,109 Total)				
	Recent Conditions	75 ppb	15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
All 12 tree species with E-R functions					
5	134	-	-	-	-
4	387	3	3	-	-
3	765	24	22	14	5
2	882	994	981	972	924
1	593	1,292	1,273	1,238	1,277
0	348	796	830	885	903
Total counties exceeding	2,761	2,313	2,279	2,224	2,206
Counties exceeding for the median species	2,237	685	670	651	627
11 tree species with E-R functions excluding cottonwood					
5	15	-	-	-	-
4	180	-	-	-	-
3	680	3	3	-	-
2	933	46	32	14	5
1	610	1,880	1,857	1,818	1,812
0	691	1,180	1,217	1,277	1,292
Total counties exceeding	2,418	1,929	1,892	1,832	1,817
Counties exceeding for the median species	1,604	239	221	204	172
10 tree species with E-R functions excluding cottonwood and black cherry					
5	-	-	-	-	-
4	15	-	-	-	-
3	187	-	-	-	-
2	856	29	15	2	1
1	920	95	72	19	8
0	1,131	2,985	3,022	3,088	3,100
Total counties exceeding	1,978	124	87	21	9
Counties exceeding for the median species	666	36	18	6	2

We also consider WREA estimates (quantitative and qualitative) of effects on several ecosystem services. First, impacts on growth related to cumulative O₃ exposure values in federally designated Class I areas were derived from an average wRBL value (discussed above) for 145 of the 156 Class I areas (U.S. EPA 2014a, section 6.8.1). Given established objectives for Class I areas (e.g., to maintain in perpetuity), effects in Class I areas may be considered to

have the potential to adversely affect the intended use of the ecosystem, e.g., to leave them unimpaired and preserve them for the enjoyment of future generations. Under recent conditions, this analysis estimates that 13 Class I areas have wRBL values above 2%. Further, this analysis estimates that based on average W126 index values estimated for the air quality scenario just meeting the current standard, 2 of the 145 Class I areas assessed would be expected to have multiple-species, wRBL values above 2% (U.S. EPA 2014a, Table 6-26). However, we recognize that this analysis is limited to the 12 studied tree species, and therefore could underestimate other O₃-sensitive species without E-R functions.

The WREA also presents national-scale estimates of the effects of biomass loss on timber production and agricultural harvesting, as well as on carbon sequestration. The WREA used the O₃ E-R functions for tree seedlings to calculate relative yield loss (equivalent to biomass loss) across the trees' entire life spans. Because the forestry and agriculture sectors are related and trade-offs occur between the sectors, the WREA also calculated the resulting market-based welfare effects of O₃ exposure in the forestry and agriculture sectors.¹⁴ In the analyses for commercial timber production, based on the 3-year average W126 index values estimated for the air quality scenario just meeting the existing standard, RYL estimates for timber were below one percent with the exception of the Southwest, Southeast, Central, and South regions (U.S. EPA, 2014a, section 6.3, Table 6-9) (see U.S. EPA, 2014a, Table 6-8 for clarification on region names). At the current standard the highest yield loss occurs in upland hardwood forests in the South Central and Southeast regions at over 3% per year and in Corn Belt hardwoods at just over 2% loss per year. Relative yield losses for timber remain above one percent for the 3-year average W126 scenarios for 15 and 11 ppm-hrs in parts of the Southeast, Central, and South regions, and for the 7 ppm-hrs scenario in the Southeast and South regions (U.S. EPA, 2014a, section 6.3, Table 6-9). In addition, relative yield losses for timber were above two percent in parts of the Southeast and Central U.S. after just meeting the existing standard as well as in the 15 ppm-hrs and 11 ppm-hrs scenario.

In addition to estimating changes in forestry and agricultural yields, the WREA presents estimated changes in consumer and producer/farmer surplus associated with the change in yields. Changes in biomass affect individual tree species, but the overall effect on forest ecosystem productivity depends on the composition of forest stands and the relative sensitivity of trees

¹⁴ The WREA used the Forest and Agricultural Sector Optimization Model with Greenhouse Gases (FASOMGHG). FASOMGHG is a national-scale model that provides a complete representation of the U.S. forest and agricultural sectors' impacts of meeting alternative standards. FASOMGHG simulates the allocation of land over time to competing activities in both the forest and agricultural sectors. FASOMGHG results include multi-period, multi-commodity results over 60 to 100 years in 5-year time intervals when running the combined forest-agriculture version of the model.

within those stands. Economic welfare impacts resulting from just meeting the existing and alternative standards were largely similar between the forestry and agricultural sectors -- consumer surplus, or consumer gains, generally increased in both sectors because higher productivity under lower O₃ concentrations increased total yields and reduced market prices. Comparisons are not straightforward to interpret due to market dynamics. For example, because demand for most forestry and agricultural commodities is not highly responsive to changes in price, there were more examples for which producer surplus (i.e., producer gains) declines.¹⁵ In some cases, lower prices reduce producer gains more than can be offset by higher yields. The increase in consumer welfare is much larger than the loss of producer welfare resulting in net welfare gains in the forestry sector nationally. The national-scale analysis of carbon dioxide (CO₂) sequestration estimates more storage under the current standard compared to recent conditions (U.S. EPA 2014a, Appendix 6B, Table B-10). In considering the significance of the potential climate and ecosystem service impact, we also note the large uncertainties associated with this analysis (see U.S. EPA 2014a, Table 6-27).

We additionally consider the WREA estimates of tree growth and ecosystem services provided by urban trees over a 25-year period for five urban areas based on case-study scale analyses that quantified the effects of biomass loss on carbon sequestration and pollution removal (U.S. EPA 2014a, sections 6.6.2 and 6.7).¹⁶ The urban areas included in this analysis represent diverse geography in the Northeast, Southeast, and Central regions, although they do not include an urban area in the western U.S. Estimates of the effects of O₃-related biomass loss on carbon sequestration indicate the potential for an increase of somewhat more than a million metric tons of CO₂ equivalents for average W126 index values associated with meeting for the current standard scenario as compared to recent conditions. Somewhat smaller additional increases are estimated for the three W126 scenarios in comparison to the current standard scenario (U.S. EPA 2014a, section 6.6.2 and Appendix 6D).

In addition to the quantitative assessments discussed above, qualitative assessments for some ecosystem services, were also conducted, such as commercial non-timber forest products and recreation (U.S. EPA, 2014a, section 6.4), aesthetic and non-use values (U.S. EPA, 2014a, section 6.4), increased susceptibility to insect attack and fire damage (U.S. EPA, 2014a, sections 5.3 and 5.4, respectively). Other ecological effects that are causally or likely causally associated with O₃ exposure such as terrestrial productivity, water cycle, biogeochemical cycle, and

¹⁵ See Chapter 6, Section 6.3 of the WREA for a discussion of economic welfare and consumer and producer surplus.

¹⁶ The WREA used the i-Tree model for the urban case studies. i-Tree is a peer-reviewed suite of software tools provided by USFS.

community composition (U.S. EPA 2013, Table 9-19) were not directly addressed in the WREA due to a lack of sufficient quantitative information.

There is substantial heterogeneity in plant responses to O₃, both within species, between species, and across regions of the U.S. The O₃-sensitive tree species are different in the eastern and western U.S. -- the eastern U.S. has far more species. Ozone exposure and risk is somewhat easier to assess in the eastern U.S. because of the availability of more data and the greater number of species to analyze. In addition, there are more O₃ monitors in the eastern U.S. but fewer national parks (U.S. EPA, 2014a, chapter 8).

- **What are the uncertainties associated with both quantitative and qualitative information?**

Several key limitations and uncertainties, which may have a large impact on both overall confidence and confidence in individual analyses, are discussed here. Despite these uncertainties, the overall body of scientific evidence underlying the ecological effects and associated ecosystem services evaluated in the WREA is strong, and the methods used to quantify associated risks are scientifically sound (Frey, 2014). Key uncertainties associated with the assessment of impacts on ecosystem services at the national and case-study scales, as well as across species, U.S. geographic regions and future years include those associated with the interpolated and adjusted O₃ concentrations used to estimate W126 exposures in the WREA air quality scenarios and those associated with the available seedling E-R functions.

The WREA identifies sources of uncertainty for the W126 estimates for each air quality scenario and qualitatively characterizes the magnitude of uncertainty and potential for directional bias (U.S. EPA, 2014a, Table 4-5). These sources of uncertainty are described in more detail in the WREA Chapter 4 and summarized below.

An important large uncertainty in the analyses is the assumed response of the W126 concentrations to emissions reductions needed to meet the existing standard (U.S. EPA, 2014a, section 8.5.1). We note that any approach to characterizing O₃ over broad geographic areas based on concentrations at monitor locations will convey inherent uncertainty. The model-based adjustments, based on U.S.-wide emissions reductions in oxides of nitrogen (NO_x), do not represent air quality distributions from an optimized control scenario that just meets the current standard (or target W126 index values for other scenarios), but rather characterize one potential distribution of air quality across a region when all monitor locations meet the standard (U.S. EPA 2014a, section 4.3.4.2).¹⁷ An additional uncertainty comes from the creation of a national

¹⁷ Because our analyses used U.S.-wide NO_x emissions reductions to simulate just meeting the existing standard independently in each region, there are broad regional reductions in O₃ even in meeting standards in urban areas when targeting a few high-O₃ urban monitors for reductions. However, the assumption of broad regional or national NO_x reductions are not unreasonable given EPA regulations such as the NO_x SIP Call program

W126 surface using the VNA technique to interpolate recent air quality measurements of O₃. In general, spatial interpolation techniques perform better in areas where the O₃ monitoring network is denser. Therefore, the W126 estimated in the rural areas in the West, Northwest, Southwest, and West North Central with few or no monitors (Figure 2-1) are more uncertain than those estimated for areas with denser monitoring. Additionally, the surface is created from the three-year average at the monitor locations, rather than creating a surface for each year and then averaging across years at each grid cell; the potential impact of this on the resultant estimates is considered in the WREA (U.S. EPA, 2014a, Appendix 4A).

Because the W126 estimates generated in the air quality analyses are inputs to the vegetation risk analyses for biomass loss, any uncertainties in the air quality analyses are propagated into the those analyses (U.S. EPA 2014a, section 8.5). In its letter to the Administrator following its review of the second draft WREA CASAC notes that:

“The currently reported finding of only small differences in risk between just meeting the current standard and a W126-based level of 15 ppm-hrs must not be interpreted to mean that just meeting the current standard will be as protective as meeting a W126-based standard at 15 ppm-hrs. There are two key factors that must be considered when making this comparison. First, air quality was simulated in the Second Draft WREA based on the magnitude of across-the-board reductions in NO_x emissions required to bring the highest monitor down to the target level. Meeting a target level at the highest monitor requires substantial reductions below the targeted level through the rest of the region. This artificial simulation does not represent an actual control strategy and may conflate differences in control strategies required to meet different standards and different targets. As a result, there may be a number of monitors that meet the current standard but would not meet an alternative W126 standard. Second, and equally important, the current form of the standard is much less biologically relevant for protecting vegetation than is a seasonal, peak weighted index such as the W126, which was designed to measure the cumulative effects of ozone exposure.” (Frey, 2014, pp. 11-12).

With regard to the robust seedling E-R functions, the description of Figure 5-2 above provides some characterization of the variability of individual study results and the impact of that on estimates of W126 index values that might elicit different percentages of biomass loss in tree seedlings (U.S. EPA, 2014a, section 6.2.1.2). Even though the evidence shows that there are additional species adversely affected by O₃-related biomass loss, the WREA only has E-R functions available to quantify this loss for 12 tree species. This absence of information only

implemented to help areas meet the 1997 O₃ standard resulting in substantial reductions in power plant NO_x emissions from states across the eastern U.S., and the multitude of onroad and offroad mobile source rules that will lead to reduction in NO_x from these sources across the country in future years.

allows a partial characterization of the O₃-related biomass loss impacts in trees associated with recent O₃ index values and with just meeting the existing and potential alternative secondary standards. In addition, there are uncertainties inherent in these E-R functions, including the extrapolation of relative biomass loss rates from tree seedlings to adult trees and information regarding within-species variability. The overall confidence in the E-R function varies by species based on the number of studies available for that species. Some species have low within-species variability (e.g., many agricultural crops) and high seedling/adult comparability (e.g., aspen), while other species do not (e.g., black cherry). The uncertainties in the E-R functions for biomass loss and in the air quality analyses are propagated into the analysis of the impact of biomass loss on ecosystem services, including provisioning and regulating services (U.S. EPA, 2014a, Table 6-27). The WREA characterizes the direction of potential influence of E-R function uncertainty as unknown, yet its magnitude as high, concluding that further studies are needed to determine how accurately the assessed species reflect the larger suite of O₃-sensitive tree species in the U.S. (U.S. EPA, 2014a, Table 6-27).

Another uncertainty associated with interpretation of the WREA biomass loss-related estimates concerns the potential for underestimation of compounding of growth effects across multiple years of varying concentrations. Though tree biomass loss impacts were estimated using air quality scenarios of 3-year average W126 index values, the WREA also conducted an analysis to compare the impact of using a variable compounding rate based on yearly variations in W126 exposures to that of using a W126 index value averaged across three years. The WREA compared the compounded values for each region, except for the South. In these examples, one species was chosen that occurred within that region. Air quality values associated with just meeting the existing standard of 75 ppb were used. Within each region the WREA analysis used both the W126 index value at each monitor in the region for each year and the three-year average W126 index value using the method described in Chapter 4. The results show that the use of the three-year average W126 index value may underestimate RBL values slightly. However, it should be noted that the approach does not account for moisture levels or other environmental factors that could affect biomass loss (U.S. EPA, 2014a, section 6.2.1.4 and Figure 6-14). In considering these results, we note that in these regions and in all three years, the three-year average W126 index value is sometimes above and sometimes below the individual year W126 index value.

In the national-scale analyses of timber production, agricultural harvesting, and carbon sequestration, the WREA used the FASOMGHG model, which includes functions for carbon sequestration, assumptions regarding proxy species, and non-W126 E-R functions for three crops. However, FASOMGHG does not include agriculture and forestry on public lands, changes in exports due to O₃ into international trade projections, or forest adaptation. Despite

the inherent limitations and uncertainties, the WREA concludes that the FASOMGHG model reflects reasonable and appropriate assumptions for a national-scale assessment of changes in the agricultural and forestry sectors due to changes in vegetation biomass associated with O₃ exposure (U.S. EPA, 2014a, sections 6.3, 6.5, 6.6, and 8.5.2).

In the case study analyses of five urban areas, the WREA used the i-Tree model, which includes an urban tree inventory for each area and species-specific pollution removal and carbon sequestration functions. However, i-Tree does not account for the potential additional VOC emissions from tree growth, which could contribute to O₃ formation. Despite the inherent limitations and uncertainties, the WREA concludes that the i-Tree model reflects reasonable and appropriate assumptions for a case study assessment of pollution removal and carbon sequestration for changes in biomass associated with O₃ exposure (U.S. EPA, 2014a, sections 6.6.2, 6.7, and 8.5.2).

The overall effect of the combined set of uncertainties on confidence in the interpretation of the WREA results is difficult to quantify. Due to differences in available information, the degree to which each analysis was able to incorporate quantitative assessments of uncertainty differed. Despite these uncertainties, the overall body of scientific evidence underlying the ecological effects and associated ecosystem services evaluated in the WREA is strong, and the methods used to quantify associated risks are scientifically sound (Frey, 2014).

5.3 CROP YIELD LOSS

This section considers the current evidence and exposure/risk information to inform consideration of the adequacy of the protection provided by the current standard from known and anticipated adverse welfare effects of O₃ related to crop yield and other associated effects. Crops warrant consideration from a public welfare perspective because they provide food and fiber services to humans. This section includes a discussion of the policy-relevant science and weight-of-evidence conclusions discussed in the ISA (section 5.3.1) and the exposure/risk results (section 5.3.2) described in the final WREA. Important uncertainties and limitations in the available information are discussed under the related question below. These discussions highlight the information we consider relevant to answering the overarching question and associated policy-relevant questions included in this section.

5.3.1 Evidence-based Considerations

Ozone can interfere with carbon gain (photosynthesis) and allocation of carbon. As a result of decreased carbohydrate availability, fewer carbohydrates are available for plant growth, reproduction, and/or yield. For seed-bearing plants, these reproductive effects will culminate in

reduced seed production or yield. The detrimental effect of O₃ on crop production has been recognized since the 1960s, and current O₃ concentrations in many areas across the U.S. are high enough to cause yield loss in a variety of agricultural crops including, but not limited to, soybeans, wheat, cotton, potatoes, watermelons, beans, turnips, onions, lettuces, and tomatoes. Increases in O₃ concentration may further decrease yield in these sensitive crops while also causing yield losses in less sensitive crops (U.S. EPA 2013, section 9.4.4). The ISA concluded that the evidence is sufficient to determine that there is a causal relationship between O₃ exposure and reduced yield and quality of agricultural crops (U.S. EPA 2013, Table 2-2).

- **To what extent has scientific information become available that alters or substantiates our prior conclusions regarding O₃-related crop yield loss and of factors that influence associations between O₃ levels and crop yield loss?**

In general, the vast majority of the new scientific information has substantiated our prior conclusions regarding O₃ crop yield loss. On the whole, this evidence supports previous conclusions that exposure to O₃ decreases growth and yield of crops. The ISA describes average yield loss reported across a number of meta-analytic studies have been published recently for soybean wheat, rice, semi-natural vegetation, potato, bean and barley (U.S. EPA 2013, section 9.4.4.1). Further, several new exposure studies continue to show decreasing yield and biomass in a variety of crops with increased O₃ exposure (U.S. EPA 2013, section 9.4.4.1, Table 9-17). Research has linked increasing O₃ concentration to decreased photosynthetic rates and accelerated aging (U.S. EPA 2013, section 9.4.4) in leaves, which are related to yield. Recent research has highlighted the effects of O₃ on crop quality. Increasing O₃ concentration can also decrease nutritive quality of grasses and macro- and micro-nutrient concentrations in fruits and vegetable crops (U.S. EPA 2013, section 9.4.4). The findings of these studies did not change our understanding of O₃-related crop loss since the last review and little information has emerged on factors that influence associations between O₃ levels and crop yield loss.

- **To what extent have important uncertainties identified in the last review been reduced and/or new uncertainties emerged?**

Important uncertainties have been reduced regarding crop E-R functions, especially for soybean. In general, the ISA reports consistent results across exposure estimation techniques and across crop varieties. Figure 5-4 below illustrates the composite E-R functions for the 10 crop species assessed in the WREA (U.S. EPA, 2014a, Figure 6-3).

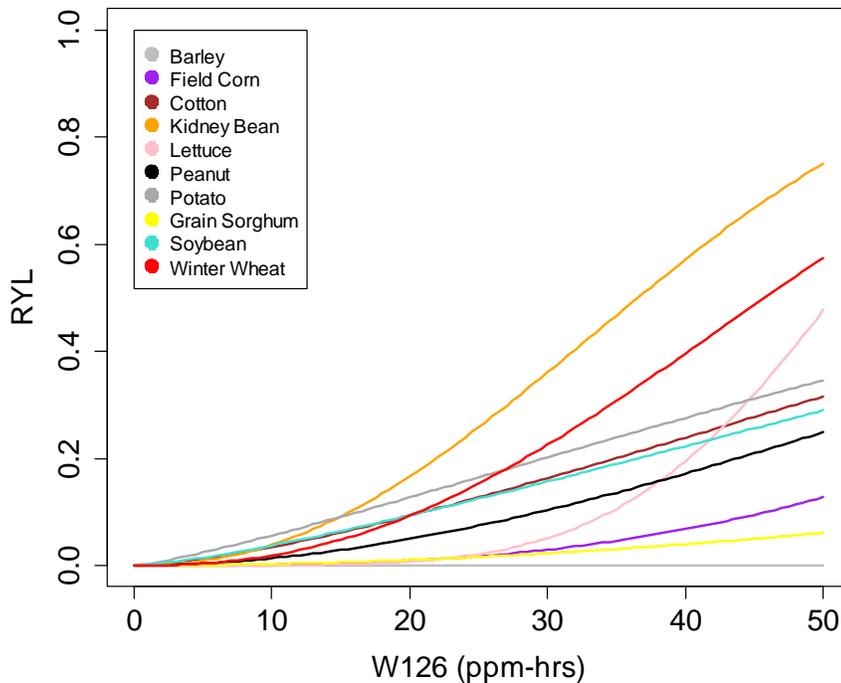


Figure 5-4. Relative yield loss in crops using the composite functions for 10 studied species in response to seasonal O₃ concentrations in terms of seasonal W126 index values, Y-axis scale for RYL values represents 0% up to 100% (U.S. EPA 2014a, Figure 6-3).

Two important uncertainties have been reduced regarding the E-R functions for yield effects of O₃ in crop species, especially for soybean. First, in the last several reviews, the extent to which E-R functions developed in OTC predicted plant responses in the field and under different exposure conditions was not clear. In this review, the ISA included an analysis comparing OTC data with field-based data for one crop and one tree species (U.S. EPA, 2013, section 9.6.3.2). The crop comparison was done using soybean OTC data from NCLAN and field-based data from SoyFACE. The NCLAN program, which was undertaken in the early to mid-1980s, assessed multiple U.S. crops, locations, and O₃ exposure levels, using consistent methods, to provide the largest, most uniform database on the effects of O₃ on agricultural crop yields (U.S. EPA 1996; U.S. EPA 2006; U.S. EPA 2013, sections 9.2, 9.4, and 9.6, Frey, 2014,

p. 9).¹⁸ The SoyFACE experiment was a chamberless field-based exposure study in Illinois that was conducted from 2001 – 2009 (U.S. EPA 2013, section 9.2.4). Yield loss in soybean from O₃ exposure at the SoyFACE field experiment was reliably predicted by soybean E-R functions developed in NCLAN (U.S. EPA, 2013, Section 9.6). This analysis supports the robustness and use of the E-R functions developed in NCLAN to predict relative yield loss to O₃ exposure in a realistic agricultural setting.

A second area of uncertainty that was reduced is that regarding the appropriateness of applying the NCLAN E-R functions to more recent cultivars that are currently being grown. Because recent studies continue to find yield loss levels in crop species studied previously under NCLAN that reflect the earlier findings, the ISA concluded that there has been little new evidence that crops are becoming more tolerant of O₃ (U.S. EPA, 2006a; U.S. EPA 2013). This is especially evident in the research on soybean. In a meta-analysis of 53 studies, Morgan et al. (2003) found consistent deleterious effects of O₃ exposures on soybean from studies published between 1973 and 2001. Further, Betzelberger et al. (2010) recently utilized the SoyFACE facility to compare the impact of elevated O₃ concentrations across 10 soybean cultivars to investigate intraspecific variability of the O₃ response. The E-R functions derived for these 10 current cultivars were similar to the response functions derived from the NCLAN studies (Heagle, 1989), suggesting there has not been any selection for increased tolerance to O₃ in more recent cultivars. The 2013 ISA reported comparisons between yield predictions based on data from cultivars used in NCLAN studies, and yield data for modern cultivars from SoyFACE (U.S. EPA, 2013, section 9.6.3). They confirm that the average response of soybean yield to O₃ exposure has not changed in current cultivars. Thus, staff concludes that at least for soybean, uncertainties associated with use of the NCLAN generated E-R functions to estimate biomass loss in recent cultivars has been reduced.

- **To what extent does the available evidence indicate the occurrence of O₃-related effects on crop yield loss attributable to cumulative exposures at lower ambient O₃ concentrations than previously established or to exposures at or below the level of the current standard?**

Little scientific evidence has emerged to indicate a lower W126 index value for cumulative exposures that can affect crop yield than previously established. However, as

¹⁸ The NCLAN protocol was designed to produce crop exposure-response data representative of the areas in the U.S. where the crops were typically grown. In total, 15 species (e.g., corn, soybean, winter wheat, tobacco, sorghum, cotton, barley, peanuts, dry beans, potato, lettuce, turnip, and hay [alfalfa, clover, and fescue]), accounting for greater than 85 percent of U.S. agricultural acreage planted at that time, were studied. Of these 15 species, 13 species including 38 different cultivars were combined in 54 cases representing unique combinations of cultivars, sites, water regimes, and exposure conditions. Crops were grown under typical farm conditions and exposed in open-top chambers to ambient O₃, sub-ambient O₃, and above ambient O₃.

discussed below, CASAC has provided a target benchmark protection for crop yield loss that can help better focus a discussion of the level of exposure that O₃ related effects on crops can occur (levels of concern). Currently available evidence supports effects on crop yield at cumulative exposures at and below the level of the current standard. As described above, the new evidence has strengthened the basis for using the information from the E-R functions.

Based on the 10 robust E-R functions (i.e., barley, lettuce, field corn, grain sorghum, peanut, winter wheat, cotton, soybean, potato and kidney bean) described in the ISA and additionally analyzed in the WREA (Figure 5-4), Table 5C-3 shows that for the CASAC recommended target benchmark protection level of 5% for median crop relative yield loss (RYL), W126 index values ranging from 7 to 17 ppm-hrs are protective. However, when individual species are considered over this same range, the proportion of crops protected varies from 5/10, 6/10, 7/10, 9/10, 10/10, and 10/10 at the W126 levels of 17, 15, 13, 11, 9, and 7 ppm-hrs. To the extent a given species is judged as having particular importance to the public welfare, breaking the information down by species can be helpful. For example, less than 5% yield loss was estimated for soybeans at the W126 index value of 12 ppm-hrs (U.S. EPA 2014a, Figure 6-3). Four of the studied crop species (barley, lettuce, field corn, and grain sorghum) are more tolerant, with RYL under 1% over the W126 range from 7 to 17 ppm-hrs. Peanut also remained under 4% RYL over the same W126 range. Other species differed regarding the W126 level at which RYL reached or fell below 5%. Specifically, for winter wheat, cotton, soybean, kidney bean and potato, the relevant W126 index values at which RYLs were below 5% are 15, 13, 11, 11, and 9 ppm-hrs.

Where the current evidence on crop yield loss is not in terms of parts per billion concentrations over a specific exposure period such as eight hours, assessing whether O₃ concentrations associated with meeting the current standard would allow crop yield effects is more complex. In order to characterize the O₃ exposures associated with crop yield loss in terms of seasonal W126 index and to consider the extent to which such index values might be expected to occur in agricultural locations that meet the current standard, we evaluated two agricultural counties in Kansas using O₃ monitoring data from EPA's Air Quality System (AQS) combined with the E-R function for soybeans. Sedgwick and Sumner counties both met the level of the 3-year, 8-hour standard of 75 ppb in 2009-2011, but both still had a maximum annual W126 level of 19 ppm-hours in 2011. At that annual W126 index value, soybean yield loss would be predicted to be 9% in those counties in that year.

- **To what extent does currently available evidence suggest locations where the vulnerability of sensitive species, ecosystems and/or their associated services to O₃-related crop yield loss would have special significance to the public welfare?**

During the previous NAAQS reviews, there were very few studies that estimated O₃ impacts on crop yields at large geographical scales (i.e., regional, national or global). Recent modeling studies of the historical impact of O₃ concentrations found that increased O₃ generally reduced crop yield, but the impacts varied across regions and crop species (U.S. EPA, 2013, Section 9.4.4.1). The largest O₃-induced crop yield losses were estimated to occur in high-production areas exposed to elevated O₃ concentrations, such as the Midwest and the Mississippi Valley regions of the United States. Among crop species, the estimated yield loss for wheat and soybean were higher than rice and maize. Additionally, satellite and ground-based O₃ measurements have been used to assess yield loss caused by O₃ over the continuous tri-state area of Illinois, Iowa, and Wisconsin. The results indicate that O₃ concentrations during the assessed period reduced soybean yield, which correlates well with the previous results from FACE- and OTC-type experiments (U.S. EPA 2013, section 9.4.4.1).

Thus, the recent scientific literature in the ISA continues to support the conclusions of the 1996 and 2006 CDs that ambient O₃ concentrations can reduce the yield of major commodity crops in the U.S. and to support the use of crop E-R functions based on OTC experiments. Agricultural areas that would be likely to have the most significance to the public welfare would be those high production areas for sensitive crops that also are exposed to high O₃ concentrations, such as areas in the Midwest and Mississippi Valley regions.

5.3.2 Exposure/Risk-based Considerations

Two main analyses are conducted in the WREA to estimate O₃ impacts related to crop yield. Annual yield losses are estimated for 10 commodity crops and these estimates are then additionally used to estimate O₃ impacts on producer and consumer economic surpluses (Table 5-6 below; U.S. EPA, 2014a, sections 6.2, 6.5).

Table 5-6. Exposure, risk and ecosystem services analyses related to crop yield.

	Crop-level impact^B	Agri-Ecosystem Services^C
WREA estimates^A	Annual Relative Yield Loss for Corn, Cotton, Potato, Sorghum, Soybean, Winter Wheat	Economic surplus to crop producers and consumers
^A See Chapter 4 WREA; ^B See Section 6.2 WREA; ^C See Sections 6.4 and 6.5 WREA		

- **For what air quality scenarios were exposures and risks estimated? What approaches were used to estimate W126 exposures for those conditions? What are associated limitations and uncertainties?**

The WREA crop analyses described here were performed for five air quality scenarios using the methodology summarized in Table 5-4 above. In general, this methodology is identical to the air quality scenarios for the biomass loss analyses and have the same uncertainties and limitations summarized in section 5.2.2 above. These air quality scenarios described in more detail in the WREA (U.S. EPA, 2014a, chapter 4 and Appendix 4A).

- **What is the nature and magnitude of the cumulative exposure- and risk-related estimates for crop yield loss associated with remaining upon simulating just meeting the current O₃ standard? What are the uncertainties associated this information?**

The WREA presents estimates of crop yield loss for the five air quality scenarios described above using 10 robust E-R functions for commodity crops that are grown across the U.S. (U.S. EPA, 2014a, section 6.5). The largest reduction in O₃ induced crop yield loss occurs when moving from the recent conditions scenario to that for just meeting the current standard (U.S. EPA, 2014a, section 6.5). In the analyses for agricultural harvest, the largest estimates of yield changes also occur when comparing the recent conditions scenario to that for the current standard. Under recent conditions, the West, Southwest, and Northeast regions generally have the highest yield losses. For the 3-year average W126 scenarios, relative yield losses for winter wheat¹⁹ are less than one percent. For soybeans, yield losses for these scenarios range from just above 1 percent to below one percent (U.S. EPA 2014a, section 6.5). However, when evaluated at the county level, 99% of soybean producing counties (1,718) have greater than 5% yield loss under recent conditions, while no counties show yield loss at or above this level when air quality is adjusted to just meet the current standard (U.S. EPA, 2014a, section 6.5).

The WREA estimates of O₃-attributable percent yield loss based on 3-year average W126 index values estimated after just meeting the current standard are relatively small (0.0 – 2.72%) across the 10 crop species analyzed, U.S. EPA 2014a, section 6.5, Appendix 6B). In considering these estimates, we recognize the significant uncertainties associated with several aspects of the analyses. Because the W126 estimates generated in the air quality analyses are inputs to the vegetation risk analyses for crop yield loss, any uncertainties in the air quality analyses are propagated into the those analyses (U.S. EPA 2014a, Table 6-27, section 8.5).

¹⁹ Among the major crops, because winter wheat and soybeans are more sensitive to ambient O₃ levels than other crops we focus on these crops for this discussion.

- **To what extent are the exposures and risks remaining upon simulating just meeting the current O₃ standard important from a public welfare perspective?**

From a public welfare prospective, the O₃ attributable risks to crops estimated for conditions that just meet the current standard are small. However, it is unclear how much weight to put on these results given the multiple areas of uncertainty associated with these estimates as discussed in the WREA and summarized above, including those associated with the model-based adjustment methodology and those associated with projection of yield loss at the estimated O₃ concentrations (U.S. EPA, 2014a, Table 6-27, section 8.5). In addition we note that while having sufficient crop yields is of high public welfare value, important commodity crops are typically heavily managed to produce optimum yields. Given all of the inputs that go into achieving these yields, such as fertilizer, herbicides, pesticides, and irrigation, it is difficult to determine at what point O₃-induced yield loss creates an adverse impact for the producer in the way of requiring increased inputs in order to maintain the desired yields. In contrast, based on the economic theory of supply and demand, increases in crop yields would be expected to result in lower prices for affected crops and their associated goods, which would benefit consumers. However, due to pre-existing market forces and subsidies, it is not clear that such benefits would be realized by the consumer. Given these competing impacts on producers and consumers, it is unclear how to determine what type of effect may be adverse to the public welfare. In considering this issue, CASAC states that “calculation of consumer and producer surpluses is a useful contribution to quantification of welfare effects. However, this national-level approach does not adequately account for negative effects on individual farmers and forest owners in high-ozone areas...” (Frey, 2014, p. 10). Instead, CASAC states that “[a] county scale is appropriate for assessing crop yield loss. Calculating producer and consumer surpluses at national or large region scales does not provide adequate protection. Farmers growing sensitive crops in high ozone locations can be considered a ‘sensitive population’ for welfare impacts, and crop yields under these conditions should be protected.” (Frey, 2014, pp. 14 – 15). The final WREA includes a county-level analysis in Appendix 6B finding that 99 percent of soybean-producing counties, for example, exceed 5% yield loss under recent conditions, while no counties have relative yield losses above 5% for any crop after adjusting air quality scenarios to just meet the current standard.

- **What are the ecosystem services potentially affected by O₃-related crop yield loss and to what extent are they important from a public welfare perspective? To what degree can the magnitude of the O₃ effect on these services be qualitatively or quantitatively characterized?**

The WREA presents national-scale estimates of the effects of biomass loss on timber production and agricultural harvesting, which supply provisioning services of food and fiber, as

well as on carbon sequestration (U.S. EPA 2014a, section 6.5). Because the forestry and agriculture sectors are related and trade-offs occur between the sectors, the WREA also calculated the resulting market-based welfare effects of O₃ exposure in the forestry and agriculture sectors. Overall effect on agricultural yields and producer and consumer surplus depends on the (1) ability of producers/farmers to substitute other crops that are less O₃ sensitive, and (2) responsiveness, or elasticity, of demand and supply (U.S. EPA, 2014a, sections 6.5, 8.2.1.3). Estimated O₃-attributable economic welfare impacts on agricultural sectors associated with air quality conditions adjusted to just meet the existing and potential alternative W126 standard levels were largely similar between the forestry and agricultural sectors. Estimates of consumer surplus, or consumer gains, were generally higher under those conditions (compared to recent conditions) in both sectors because higher productivity under lower O₃ concentrations increased total yields and reduced market prices (U.S. EPA 2014a, Table 6-18). Because demand for most forestry and agricultural commodities is not highly responsive to changes in price, generally producer surplus, or producer gains, decline. For agricultural welfare, annualized combined consumer and producer surplus gains were estimated to be \$2.6 trillion for model-based adjustment to meet the current standard. Combined gains were essentially unchanged in comparisons of the current standard scenario to the average W126 scenario for 15 ppm-hrs, but gains increased by \$21 million for the W126 scenario for 11 ppm-hrs and \$231 million for the W126 scenario for 7 ppm-hrs. In some cases, lower prices reduce producer gains more than can be offset by higher yields (U.S. EPA, 2014a, Table 6-18).

The WREA discusses multiple areas of uncertainty associated with these estimates (also summarized above), including those associated with the model-based adjustment methodology as well as those associated with projection of yield loss at the estimated O₃ concentrations (U.S. EPA, 2014a, Table 6-27, section 8.5).

5.4 VISIBLE FOLIAR INJURY

Visible foliar injury resulting from exposure to O₃ has been well characterized and documented over several decades of research on many tree, shrub, herbaceous, and crop species (U.S. EPA, 2013, 2006, 1996, 1984, 1978). The significance of O₃ injury at the leaf and whole plant levels depends on an array of factors, and therefore, it may be difficult to quantitatively relate visible foliar injury symptoms to other vegetation effects such as individual tree growth, or effects at population or ecosystem levels (U.S. EPA, 2013, p. 9-39). Visible foliar injury by itself, however, is an indication of phytotoxicity due to O₃ exposure and can impact the public welfare through damaging or impairing the intended use of the affected entity or the service it provides. For example, ways by which O₃-induced visible foliar injury may impact the public welfare include: 1) visible damage to ornamental species used in landscaping or leafy crops

(spinach, lettuce, tobacco) that affects the economic value, yield, or usability of that plant (U.S. EPA 2007, section 7.4.1; Abt Associates, Inc., 1995); 2) visible damage to plants with special cultural significance (e.g., those used in tribal practices); 3) visible damage to species occurring in natural settings valued for their scenic beauty and/or recreational appeal, including in areas specially designated for more protection (e.g., federal Class I areas) (73 FR 16490). Given limitations in the available information pertaining to the first two categories,²⁰ the discussions of the evidence and exposure/risk information in sections 5.4.1 and 5.4.2 below focus primarily on what is known about visible foliar injury that has been shown to occur in natural settings valued for their scenic beauty and/or recreational appeal.

At the time of the last review, the following was known:

- 1) Ozone causes diagnostic visible injury symptoms on studied bioindicator species.
- 2) Soil moisture is a major confounding effect that can decrease the incidence and severity of visible foliar injury under dry conditions and vice versa.
- 3) The most extensive dataset regarding visible foliar injury incidence across the U.S. was that collected by the USFSFHM/FIA Program.
- 4) Visible foliar injury incidence was considered to be widespread in both the eastern and western U.S. based on staff analyses of county level air quality data and USFS biomonitoring data which showed that for each year within a four year period (2001 – 2004) the percentage of counties having a biosite with visible foliar injury ranged between 11-30% at an 8-hour average annual level of 0.074 ppm (U.S. EPA, 2007, section 7.6.3.2).

In the remainder of this section, we consider how the currently available evidence and exposure/risk information informs our understanding of the relationship that exists between visible foliar injury and exposures to O₃ in ambient air and consideration of the adequacy of protection provided by the current standard. The policy-relevant evidence and weight-of-evidence conclusions drawn from the ISA are discussed in section 5.4.1, and the exposure/risk and associated ecosystem services estimates from the WREA, are discussed in section 5.4.2. Important uncertainties and limitations in each type of available information are also discussed in these two sections.

²⁰ Qualitative information regarding potential cultural impacts of O₃-induced visible foliar injury is noted in section 5.5 and Appendix 5-A).

5.4.1 Evidence-based Considerations

- **To what extent has scientific information become available that alters or substantiates our previous conclusions of O₃-related visible foliar injury and of factors that influence associations between O₃ exposures or concentrations and visible foliar injury?**

Recent research continues to build and substantiate the previous conclusions and findings drawn from several decades of research on many tree, shrub, herbaceous, and crop species (U.S. EPA, 2013, 2006, 1996, 1984, 1978) that O₃-induced visible foliar injury symptoms are well characterized and considered diagnostic on certain bioindicator plant species. Diagnostic usage for these plants has been verified experimentally in exposure-response studies, using exposure methodologies such as continuous stirred tank reactors (CSTRs), open-top chambers (OTCs), and free-air fumigation (FACE). Although 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, experimental and observational evidence has clearly established a consistent association of the presence of visible injury symptoms with O₃ exposure, with greater exposure often resulting in greater and more prevalent injury (U.S. EPA 2013, section 9.4.2). This new research includes: 1) controlled exposure studies conducted to test and verify the O₃ sensitivity and response of potential new bioindicator plant species; 2) multi-year field surveys in several National Wildlife Refuges (NWR) documenting the presence of foliar injury in valued areas; 3) ongoing data collection and assessment by the USFS FHM/FIA program, including multi-year trend analysis (U.S. EPA 2013, section 9.4.2). These recent studies, in combination with the entire body of available evidence, thus form the basis for the ISA determinations of a causal relationship between ambient O₃ exposure and the occurrence of O₃-induced visible foliar injury on sensitive vegetation across the U.S. (U.S. EPA 2013, p. 9-42).

With regard to evidence from controlled exposure studies, a recent study of 28 plant species confirmed prior findings of O₃ causing predictable diagnostic visible foliar injury symptoms on some species of plants. This study selected 28 plant species, most of which grow naturally throughout the northeast and midwest US, including in national parks and wilderness areas, that were suspected of being O₃-sensitive, and exposed them to four different O₃ concentrations (30, 60, 90, and 120 ppb) in CSTR chambers (Kline et al., 2008). Two experiments were conducted in each year of the study (2003 and 2004). Plants were exposed to O₃ for 7 hours a day, five days a week over the course of each experiment. Specifically, in 2003, the first experiment lasted from July 14 to August 21 and included 29 days of O₃ exposure and the second from September 9 to 30 and included 16 exposure days. In 2004, the first experiment was conducted from July 13 to August 10 with 21 O₃ exposure days and the second from August

27 to September 24, including 21 days of O₃ exposure. Though the exposures were cumulative over the course of the study, exposures were reported only in terms of the target exposure concentration for each experiment. The study reported O₃-induced responses in 12, 20, 28 and 28 of the 28 tested species at the 30, 60, 90 and 120 ppb exposure concentrations²¹, respectively. Based on their findings, the authors suggest that American sycamore, aromatic sumac, bee balm, buttonbush, common milkweed, European dwarf elderberry, New England aster, snowberry and swamp milkweed would make the most useful bioindicator species. Some of these species are native to Class 1 areas (discussed further below). The staff additionally concludes that given that the exposure protocol was designed to create a continuous exposure level, not a fluctuating one, this study shows that O₃-induced foliar injury can occur from 7-hour exposures repeated over multiple days at O₃ concentrations that are below the 75 ppb level of the current standard.²² While this type of controlled study provides clear evidence of cause and effect, it also has limitations. The authors, recognizing this cautioned that “extrapolation of these CSTR results to the field must be done carefully, since CSTR/greenhouse conditions ... are not representative of natural environmental conditions” (Kline et al., 2008).

A string of recently published multi-year field studies provide a complimentary line of field-based evidence by documenting the incidence of visible foliar injury symptoms on a variety of O₃-sensitive species over multiple years and across a range of cumulative, seasonal exposure values in several eastern and midwestern NWRs (U.S. EPA 2013, section 9.4.2.1; Davis and Orendovici, 2006; Davis, 2007a, b; Davis, 2009). Some of these studies also included information regarding soil moisture stress using the Palmer Drought Severity Index (PDSI). While environmental conditions and species varied across the four NWRs, visible foliar injury was documented to a greater or lesser degree at each site. As discussed further below, visible foliar injury incidence in these types of areas has greater significance to the public welfare.

- **To what extent have important uncertainties identified in the last review been reduced and/or new uncertainties emerged?**

The studies mentioned above also provide additional information regarding an important uncertainty identified in the previous review, i.e., the role of soil moisture in influencing visible foliar injury response (U.S. EPA 2013, section 9.4.2). These studies confirm that adequate soil moisture creates an environment conducive to greater visible foliar injury in the presence of O₃

²¹ Two of the target exposure levels, 30 and 60 ppb, fall below the level of the current standard (75 ppb). The mean exposure concentrations achieved in the CTSRs for the 30 ppb target level for each year and study were 27.9, 26.3, 27.1, and 29.3 ppb and for the 60 ppb target level were 56.6, 55.8, 57.9, and 59.0 ppb, for 2003 study 1, 2003 study 2, 2004 study 1, and 2004 study 2, respectively.

²² The current standard is met when the 3-year average of the 4th highest daily maximum 8-hour average concentrations is at or below 75 ppb.

than drier conditions. As stated in the ISA, “[a] major modifying factor for O₃-induced visible foliar injury is the amount of soil moisture available to a plant during the year that the visible foliar injury is being assessed ... because lack of soil moisture generally decreases stomatal conductance of plants and, therefore, limits the amount of O₃ entering the leaf that can cause injury” (U.S. EPA, 2013, p. 9-39). As a result, “many studies have shown that dry periods in local areas tend to decrease the incidence and severity of O₃-induced visible foliar injury; therefore, the incidence of visible foliar injury is not always higher in years and areas with higher O₃, especially with co-occurring drought (Smith, 2012; Smith et al., 2003)” (U.S. EPA, 2013, p. 9-39). This “...partial ‘protection’ against the effects of O₃ afforded by drought has been observed in field experiments (Low et al., 2006) and modeled in computer simulations (Broadmeadow and Jackson, 2000)” (U.S. EPA, 2013, p. 9-87). In considering the extent of any protective role of drought conditions, however, the ISA also notes that other studies have shown that “drought may exacerbate the effects of O₃ on plants (Pollastrini et al., 2010; Grulke et al., 2003)” and that “[t]here is also some evidence that O₃ can predispose plants to drought stress (Maier-Maercker, 1998)”. Accordingly, the ISA concludes that “the nature of the response is largely species-specific and will depend to some extent upon the sequence in which the stressors occur” (U.S. EPA, 2013, p. 9-87). Such uncertainties associated with describing the potential for foliar injury and its severity or extent of occurrence for any given air quality scenario due to confounding by soil moisture levels make it difficult to identify an appropriate degree of annual protection (as well as ambient O₃ exposure conditions that might be expected to provide that protection).

- **To what extent does the available evidence indicate the occurrence of O₃-related visible foliar injury attributable to cumulative exposures at lower ambient O₃ concentrations than previously established or to exposures at or below the level of the current standard?**

Recently available evidence confirms the evidence available in previous reviews that visible foliar injury can occur when sensitive plants are exposed to elevated O₃ concentrations in a predisposing environment (i.e., adequate soil moisture (U.S. EPA, 2013, section 9.4.2)). Recent evidence also continues to indicate the occurrence of visible foliar injury at cumulative ambient O₃ exposures previously established. Since the 2006 O₃ CD, results from several multi-year field surveys and experimental screenings of O₃-induced visible foliar injury on vegetation also show that visible foliar injury can occur under conditions where the annual 8-hour average O₃ concentrations are at or below the level of the current standard, as discussed here. Limited information exists regarding the incidence of visible foliar injury occurring in areas that have design values that meet the current 3-year average 8-hour standard.

To facilitate comparison with other studies reporting foliar injury response to W126 cumulative exposures, we obtained air quality data from the EPA's AQS database for monitors in each study location and calculated the 12-hr W126 index values and obtained the maximum 4th highest 8-hour average values for a subset of the most recent years included in each study (Table 5-7). As the shaded rows in Table 5-7 below show, in the years 2002/2003 and 2004 in the Cape Romain NWR in South Carolina, and the Seney NWR in Michigan, respectively, the 4th highest daily maximum 8-hour average O₃ concentrations were at or below the level of the current standard. We additionally note that the Cape Romain site met the current standard of 75 in every 3-year period during the study and has consistently met the standard from 2001 to 2012.²³ Under these air quality conditions, three species (i.e., winged sumac, Chinese tallow tree, and wild grape) exhibited O₃-induced stipple. In 2002, 32% of the examined wild grape plants, 20% of the winged sumac plants, and 4.6% of the Chinese tallow tree plants, respectively, were symptomatic (Davis, 2009). At the same time, the 12-hour W126 index value was 20 ppm-hrs. In 2003, when air quality was somewhat improved, foliar injury declined, with only 13.3% of wild grape showing O₃ stipple at a maximum 4th highest 8-hour of 74 ppb and a W126 index value of 11 ppm-hrs. The PDSI values were 0.27 and 2.45 in 2002 and 2003, respectively. These values show that 2003 was a wetter year than 2002, though 2002 would have been considered within the normal soil moisture range.

At the Seney NWR site, by comparison, the annual W126 level was similar in 2004 to that at Cape Romain in 2003, and the annual 8-hour average level was below that of the current standard, though the 3-year average design values were above that of the current standard for that year. Not surprisingly, given the lower O₃ air quality in 2004, the Seney study reported injury ranging from about 2% on common milkweed to about 6% on spreading dogbane. Though this study does not provide the PDSI values, the authors provided some discussion of a possible relationship stating that "the incidence of ozone injury on spreading dogbane, but not other species, was weakly, but not significantly, related to the drought index (PDSI)...However this relationship was too weak to be used for predictive purposes" (Davis, 2007b). The authors then conclude that "[n]evertheless, the threshold SUM06 ozone level needed to induce stipple on sensitive plants within the Seney refuge is likely 5000 ppb-hrs under the environmental conditions of these surveys" (Davis, 2007b). On the basis of the above, the staff concludes that these studies confirm that visible foliar injury has been shown to occur in the field at W126 index values ranging down to 10 ppm-hrs and provide limited evidence that such foliar injury

²³ Design values (concentrations in the form of the standard) for this monitoring site during this period are presented in the file available at: <http://www.epa.gov/airtrends/values.html> (US EPA, 2014d).

can occur in areas with special public welfare significance during periods that meet the current standard.

Table 5-7. Visible foliar injury incidence in four National Wildlife Refuges.

Name/ Site #/ Ref. ^A	Year ^B	4 th highest daily maximum 8-hour average	12-hour W126	% Plants with visible injury
Cape Romain NWR, South Carolina / 450190046 (Davis, 2009)	2002	0.075 ppm	20 ppm-hrs	5 - 32
	2003	0.074 ppm	11 ppm-hrs	3 - 13
Moosehorn NWR, Maine/ 230090102 (Davis, 2007a)	2002	0.1 ppm	24 ppm-hrs	0 - 17
	2003	0.083 ppm	22 ppm-hrs	0 - 13
	2004	0.082 ppm	14 ppm-hrs	3 - 10
Seney NWR, Michigan/ 261530001 (Davis, 2007b)	2002	0.083 ppm	11 ppm-hrs	0 - 13
	2003	0.076 ppm	15 ppm-hrs	1 - 6
	2004	0.074 ppm	10 ppm-hrs	2 - 6
Brigantine NWR, New Jersey / 340010005/ (Davis and Orendovici, 2006)	2001	0.095 ppm	39 ppm-hrs	0 - 45
	2002	0.092 ppm	53 ppm-hrs	0 - 4
	2003	0.085 ppm	36 ppm-hrs	0 - 4
^A Studies (cited above) reported exposures in terms of SUM06 form. EPA staff, using AQS data for the same monitors, calculated exposures in terms of the current 8-hour and W126 forms: http://www.epa.gov/ttn/airs/airsaqs/ (US EPA, 2014b)				
^B Only recent years with available W126 data were included in the table.				

By far the most extensive field-based dataset of visible foliar injury incidence is that obtained by of the USFS FHM/FIA biomonitoring network program. A trend analysis of data from the sites located in the Northeast and North Central U.S. for the 16 year period (1994-2009) (Smith, 2012) provides additional evidence of foliar injury occurrence in the field as well as some insight into the influence of changes in air quality and soil moisture on visible foliar injury and the difficulty inherent in predicting foliar injury response under different air quality/soil moisture scenarios (Smith, 2012; U.S. EPA 2013, section 9.2.4.1). In this study ambient exposures were expressed in terms of the SUM06 cumulative index coupled with a measure of the number of peak hourly concentrations above 100 ppb (N100). Soil moisture estimates were generated using both the PDSI and the plant moisture availability index (MI). Foliar injury was expressed in terms of the biosite index (BI)²⁴. The authors observed that over this 16-year

²⁴ Biosite index (BI) is the average score (proportion of leaves with injury [“amount”] x mean severity of symptoms on injured leaves [“severity”]) for each species averaged across all species on the biosite multiplied by 1,000.

period, “injury indices have fluctuated annually in response to seasonal ozone concentrations and site moisture conditions. Sites with and without injury occur at all ozone exposures but when ambient concentrations are relatively low, the percentage of uninjured sites is much greater than the percentage of injured sites; and regardless of ozone exposure, when drought conditions prevail, the percentage of uninjured sites is much greater than the percentage of injured sites” (Smith, 2012). The authors further note that while “both site moisture and ozone exposure play a role in foliar injury expression ... the interplay among these three factors is unique for each year and possibly each site. Extreme moisture deficits decrease foliar injury, ... [and] ... [i]n no year do high ozone exposures override the controlling effect of site moisture, although at the other end of the scale, injury severity is minimized under conditions of low ozone exposure regardless of site moisture conditions. This implies a necessary threshold of ozone exposure for injury to occur....” “In a similar analysis, Rose and Coulston (2009) reported a high percentage of biosites with injury across the Southern region in 2003, a year when SUM06 values >10 ppm-h were widespread at the same time that the land area was in moisture surplus or balance.” Thus, Rose and Coulston (2009) also “found evidence that it is the co-occurrence of sufficient moisture and elevated ozone that determine whether injury occurs to bioindicator plants, not ozone exposure alone.” Regarding the role of peak O₃ concentrations (>100 ppb O₃), Smith (2012) reported that over the 16-year period concentrations above 100 ppb have declined, and that this “... may account for the observed decrease in the severity of ozone-induced foliar injury to ozone sensitive bioindicator plants in eastern forests.” They also note that “[t]here is no compelling evidence, however, that moderate ozone concentrations, as reflected in seasonal mean SUM06 data, are on the decline” and “[t]his may explain why injury continues to be detected on many of the same sites every year” (Smith, 2012). The authors thus conclude that, “[a]lthough it is reasonable to remain concerned about long-term impacts of ozone pollution on our forest ecosystems, the findings of this biomonitoring survey point to a declining risk of probable impact on eastern forests over the 16-year period from 1994 to 2009” (Smith, 2012).

In a similar assessment of the USFS FHM/FIA data in the West, six years (2000 to 2005) of biomonitoring data for O₃ injury were evaluated for the three coastal states of California, Oregon and Washington (Campbell et al., 2007; U.S. EPA 2013, section 9.4.2.1). Campbell et al., 2007 found that “...ozone injury occurs frequently (25 to 37 percent of sampled biosites) in California forested ecosystems demonstrating that ozone is present at phytotoxic levels.” This study concluded that, “in California, an estimated 1.3 million acres of forest land and 596 million cubic feet of wood are at moderate to high risk to impacts from ozone. However, [m]ore years of data are needed to discern any trends” (Campbell et al, 2007). Though this study does not discuss the role of soil moisture in describing the results, the criteria used to select the biomonitoring sites include one that considers soil conditions. The best sites are identified as

those with low drought potential and good fertility. Thus, given the relatively high O₃ concentrations that occur in California and the likelihood that many of the biomonitoring sites occur in areas that have sufficient soil moisture, the high percentage of sampled biosites with foliar injury is not unexpected.²⁵

These recent studies continue to provide evidence of O₃-induced foliar injury occurring in many areas across the U.S. and augment our understanding of O₃-related visible foliar injury and of factors that influence associations between O₃ exposures or concentrations and visible foliar injury such as soil moisture.

- **To what extent does currently available evidence suggest locations where the vulnerability of sensitive species, ecosystems and/or their associated services to O₃-related visible foliar injury would have special significance to the public welfare?**

As mentioned above, federally designated Class I areas are afforded stringent protections under the 1977 amendments to the CAA. The CAA gives federal land managers of Class I areas “the responsibility to protect all air quality related values (AQRVs)...from deterioration.... In order to determine if deterioration is occurring, baseline AQRVs must be established” (Davis, 2009). Because of this need and the significance of these areas, studies often focus on these sites. For example, a study by Kohut (2007) was undertaken to assess the risks of O₃-induced visible foliar injury on O₃-sensitive vegetation in 244 parks managed by the NPS (U.S. EPA, 2013, pp. 9-40 to 9-41, U.S. EPA, 2014a, pp. 7-19 to 7-20). Kohut (2007) concluded that the risk of visible foliar injury was high in 65 parks (27 percent), moderate in 46 parks (19 percent), and low in 131 parks (54 percent). Thus, while this study suggests that there may be a reason for concern in as much as 46% of the parks, there were a number of important limitations associated with this study (described in footnotes 8 and 9 below) that weakened this conclusion. Given the importance of this kind of assessment, the WREA used Kohut (2007) as the conceptual basis for the subsequent WREA screening-level assessment, though numerous modifications were made to the approach to make it applicable to the context of this O₃ NAAQS review (see section 5.4.2 below).

In addition, as described above, several recently published studies (U.S. EPA 2013, section 9.4.2.1; Davis and Orendovici, 2006; Davis, 2007a,b; Davis, 2009, Kohut, 2007) were conducted in federally protected areas including federally designated Class I areas such as national parks. These studies confirm that visible foliar injury has been observed in these areas under annual air quality conditions with ambient concentrations at or below the level of the

²⁵ Staff additionally notes that a large proportion of O₃ monitoring sites in California did not meet the current standard during the study period (see: <http://www.epa.gov/airtrends/values.html>) (US EPA, 2014d).

current standard and at W126 index values within the CASAC range recommended in past reviews. This evidence continues to suggest that O₃-sensitive species and their associated ecosystems and services continue to remain vulnerable to visible foliar injury incidence in areas that have been afforded special protection by Congress and that have special significance to the public welfare.

5.4.2 Exposure- and Risk-based Considerations

The WREA presents a number of analyses considering air quality conditions associated with increased prevalence of visible foliar injury and potential associated welfare impacts (see Table 5-8 below, U.S. EPA, 2014a, Chapter 7). An initial analysis included the development of benchmark criteria reflecting different prevalence of visible foliar injury in conjunction with different W126 exposures and in some cases, soil moisture conditions. These criteria were then used in a screening-level assessment to characterize potential risk of foliar injury incidence under 2006-2010 conditions in 214 national parks. The last analysis was a case study assessment on three national parks, which also provides limited characterization of the associated ecosystem services. Despite the limitations and uncertainties associated with these analyses, and recognizing that the recent air quality conditions in most cases (prior to any model-based adjustment) did not meet the current standard, we believe that they help inform our understanding of the relationship between soil moisture and foliar injury incidence, as well as provide limited support for our conclusions regarding risk of visible foliar injury incidence under air quality conditions likely to meet the current standard in areas of special significance to the public welfare.

Table 5-8. Exposure, risk and ecosystem services analyses related to visible foliar injury.

	Ecosystem Level Effects	Ecosystem Services
WREA estimates	<p>Proportion of FHM/FIA biosites with foliar injury incidence at various W126 index values and soil moisture levels</p> <p>Percent of 214 national parks exceeding various W126 benchmarks derived from FHM/FIA biosite analysis ^A</p>	<p>Case study of 3 national parks characterized impacts using available visitor and use data, including monetary data for activities and visitor expenditures:</p> <ul style="list-style-type: none"> • Utilized Willingness-to-Pay studies for scenic impairment; • Assessed the overall cover of sensitive species; • Compared sensitive species cover to trails and overlooks; and, • Estimated percent of park area with O₃ concentrations above different W126 index values averaged over three consecutive years.
<p>^A The screening-level assessment of 214 national parks additionally included observations based on the model-based adjustments to just meet the current standard and targets for the three W126 scenarios (discussed below) but did not conduct a full analysis using these data.</p>		

- **For what air quality scenarios were exposures and risks estimated? What approaches were used to estimate W126 exposures for those conditions? What are associated limitations and uncertainties?**

Three types of foliar injury analyses were performed in the WREA and are considered below. They include an analysis using USFS FHM/FIA biosite data, a screening-level assessment in 214 national parks, and case studies of three national parks. The analysis of USFS biosite data was done using O₃ concentrations estimated for a national-scale surface of concentrations (at a 12 x 12 km² grid cell resolution in contiguous U.S.) using interpolation methodology applied to concentrations at O₃ monitor locations (U.S. EPA, 2014a, section 4.3.2, Appendix 4A). The analysis of USFS FHM/FIA data and the screening-level analysis using W126 benchmarks derived from these data used surfaces for each year from 2006 through 2010 (U.S. EPA, 2014a, Appendix 4A, section 4.2). In the National Park case study analyses, observations related to air quality were made for five air quality scenarios by the methodology summarized in Table 5-4 above.²⁶

The W126 index values in the individual years from 2006 to 2010 at monitors ranged from less than 5 ppm-hrs up to above 48 ppm-hrs (U.S. EPA, 2014a, Figure 4-4 and Table 4-3).

²⁶ In general, this methodology involved two steps. The first is derivation of the average W126 value (across the three years) at each monitor location. This value is based on unadjusted data for recent conditions and adjusted concentrations for the 4 other scenarios. The development of adjusted concentrations was done for each of 9 regions independently (see U.S. EPA, 2014a, section 4.3.4.1). In the second step, national-scale spatial surfaces (W126 values for each model grid cell) were created using the monitor-location values and the VNA spatial interpolation technique (details on the VNA technique are presented in U.S. EPA, 2014a Appendix 4A).

Concentration estimates varied appreciably across the five years with the median index values across grid cells ranging from a low of 5.5 ppm-hrs in 2009 up to 11 ppm-hrs in 2006 (U.S. EPA, 2014a, Appendix 4A, section 4.2). During the recent conditions period (2006 through 2008), the average W126 index values (across the three-year recent conditions period) at the monitor locations ranged from below 10 ppm-hrs to 48.6 ppm-hrs (U.S. EPA 2014a, Figure 4-4 and Table 4-3). After adjusting the 2006-2008 data to just meet the current standard in each region, and subsequent application of the VNA technique to the current standard scenario monitor location values, the average W126 index values were below 15 ppm-hrs across the national surface with the exception of a very small area of the southwest region (near Phoenix) where the average W126 index values was near or just above 15 ppm-hrs. A lowering of the highest values occurred with application of the interpolation method as a result of estimating W126 index values at a 12 x 12 km² grid resolution rather than at the exact location of a monitor. This indicates one uncertainty associated with this aspect of the approach to estimating W126 index values for the adjusted air quality just meeting the current standard. Other uncertainties are summarized in section 5.2.2 above.

- **What are the nature and magnitude of the cumulative exposure- and risk-related estimates for visible foliar injury under recent conditions or conditions meeting the current O₃ standard?**

As an initial matter, we consider the analysis of the biomonitoring site data from the USFS FHM/FIA Network, described in section 7.2 of the WREA.²⁷ Using this dataset and associated data for soil moisture during the sample years along with ambient air O₃ concentrations based on monitoring data from 2006 to 2010 and spatial interpolation methodology (as described above), the proportion of biosites with any foliar injury are observed to increase with increasing annual W126 index values up to specific values after which there is little change in proportion of affected biosites with higher W126 index values (see Figure 5-5 below; U.S. EPA, 2014a, section 7.2, Figure 7-10). The proportion of biosites metric is derived by first ordering the data (across biosites and sample years) by W126 index value estimated for that biosite and year. Then for each W126 index value the proportion of biosites exceeding the selected biosite index value for all observations at or below that W126 index value is calculated. The WREA repeated this using a biosite index value greater than zero, indicating presence of any foliar injury (USFS, 2011).

When looking only at presence or absence of foliar injury (“any injury”) with the exception of 2008, the proportion of biosites across all W126 index values with foliar injury

²⁷ Data were not available for several western states (Montana, Idaho, Wyoming, Nevada, Utah, Colorado, Arizona, New Mexico, Oklahoma, and portions of Texas).

exceeds 15 percent; in 2006, it exceeds 20 percent, while in 2008 the proportion of biosites with foliar injury across all W126 index values was just below 15 percent (U.S. EPA, 2014a, section 7.2.3, Figure 7-9). When categorized by moisture levels, the data demonstrate a distinct pattern. In general, the WREA concludes that the results of these foliar injury analyses demonstrate a similar pattern –the proportion of biosites showing the presence of any foliar injury (biosite index >0) increases from zero to about 20% (Figure 5-5 below). This increase occurs with increasing W126 index values up to approximately 10 ppm-hrs for any foliar injury (biosite index >0), with little change in proportion of biosites with any injury at higher W126 index values. The data for biosites during normal moisture years are very similar to the dataset as a whole, with an overall proportion of close to 18 percent for presence of any foliar injury. Among the biosites with a relatively wet season (average Palmer Z => 1), the proportion of biosites showing injury is much higher and the relationship with annual W126 index value is much steeper. Much lower proportions of biosites are reached for the any injury category at biosites with relatively dry seasons (average Palmer Z < -1.24), potentially indicating that drought may provide some protection from foliar injury as discussed in the ISA (U.S. EPA, 2014a, section 7.2.3, Figures 7-10). This information provides insight into the relationship between soil moisture and foliar injury and the issue of whether drought provides protection from foliar injury. Thus, there is relatively little change in the proportion of biosites beyond a W126 index value of 10 ppm-hrs. There are two important observations that can be made from these analyses: (1) the proportion of biosites exhibiting foliar injury rises rapidly at increasing W126 index values below approximately 10 ppm-hrs, and (2) there is relatively little change in the proportions above W126 index values of approximately 10 ppm-hrs.

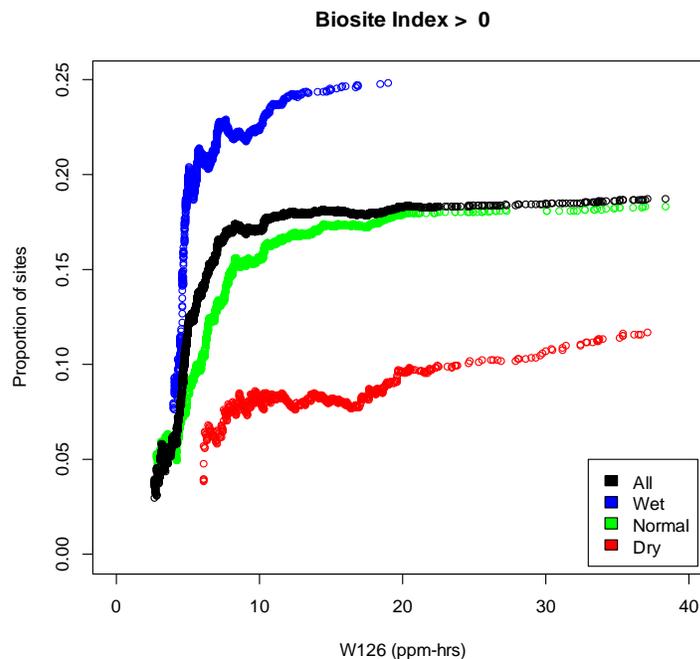


Figure 5-5. Cumulative proportion of biosites with any foliar injury present, by moisture category (U.S. EPA 2014a, Figure 7-10).

We additionally consider the WREA screening-level assessment in 214 parks in the contiguous U.S. that employed benchmark criteria developed from the above analysis (Table 5-9).^{28, 29} For example, annual O₃ concentrations corresponding to a W126 index value of 10.46 ppm-hrs represents the O₃ exposure concentration where the slope of exposure-response relationship changes for FHM biosites. The WREA refers to this as the “base scenario” benchmark. Above this index value, the percentage of FHM biosites showing foliar injury remains relatively constant. The W126 benchmarks across the five scenarios range from 3.05 ppm-hrs (five percent of biosites, normal moisture, any injury) up to 24.61 ppm-hrs (10% of biosites, dry, any injury). For the scenario of 10% biosites with injury, W126 index values were approximately 4, 6, and 25 ppm-hours for wet, normal and dry years, respectively. The national-

²⁸ The parks assessed here include lands managed by the NPS in the continental U.S., which includes National Parks, Monuments, Seashores, Scenic Rivers, Historic Parks, Battlefields, Reservations, Recreation Areas, Memorials, Parkways, Military Parks, Preserves, and Scenic Trails.

²⁹ The WREA applied different foliar injury benchmarks in this assessment after further investigation into the benchmarks applied in Kohut (2007), which were derived from biomass loss rather than visible foliar injury. Kohut cited a threshold of 5.9 ppm-hrs for highly sensitive species from Lefohn et al. (1997), which was based on the lowest W126 estimate corresponding to a 10% growth loss for black cherry. For soil moisture, Kohut (2007) qualitatively assessed whether there appeared to be an inverse relationship between soil moisture and high O₃ exposure.

scale screening-level assessment includes 42 parks with O₃ monitors and a total of 214 parks with O₃ exposure estimated from the interpolated O₃ surface for individual years from 2006 to 2010 (U.S. EPA, 2014a, Appendix 7A). These data were combined with lists from the NPS of the parks containing O₃-sensitive vegetation species (NPS, 2003, 2006). Based on NPS lists, 95 percent of the parks in this assessment contain at least one O₃-sensitive species. This analysis for recent air quality conditions, estimates that 58 percent of parks exceeded the benchmark criteria corresponding to the base scenario (W126>10.46 ppm-hrs, 17.7 percent of biosites, all moisture categories, any injury) for at least three years in the period from 2006 to 2010 (U.S. EPA, 2014a, section 7.3.2). Based on model-based adjustments to meet the current standard, none of the 214 parks have average W126 index values that would exceed the annual benchmark criteria for the base scenario (W126 >10.46 ppm-hrs) (U.S. EPA, 2014a, section 7.3.3.3).

Table 5-9. Benchmark criteria for O₃ exposure and relative soil moisture used in screening-level assessment of parks (from U.S. EPA 2014a, Table 7-6).

Scenario	Description	W126 Benchmark (in ppm-hrs)		
		Wet (Palmer Z ≥1)	Normal Moisture (Palmer Z between -1.25 and 1)	Dry (Palmer Z < - 1.25)
Base	17.7% of all FHM biosites showed any injury (higher W126 index values have a relatively constant percentage of FHM biosites showing injury)	10.46 (soil moisture not considered)		
5% of biosites	5% of FHM biosites showed any injury, reflects soil moisture categorization	3.76	3.05	6.16
10% of biosites	10% of FHM biosites showed any injury, reflects soil moisture categorization	4.42	5.94	24.61
15% of biosites	15% of FHM biosites showed any injury, reflects soil moisture categorization	4.69	8.18	N/A
20% of biosites	20% of FHM biosites showed any injury, reflects soil moisture categorization	5.65	N/A	N/A

Lastly, we consider the WREA case study analysis that focused on characterizing the ecosystem services potentially associated with visible foliar injury in three specific national parks (case study assessment). The parks included were Great Smoky Mountains National Park (GRSM), Rocky Mountain National Park (ROMO), and Sequoia/Kings National Parks (SEKI). For each park, the potential impact of O₃-related foliar injury on recreation (cultural services) was considered in light of information on visitation patterns, recreational activities and visitor expenditures. For example, visitor spending in 2011 exceeded \$800 million, \$170 million and

\$97 million dollars in GRSM, ROMO and SEKI, respectively. This assessment also included percent cover of species sensitive to foliar injury and focused on the overlap between recreation areas within the park and elevated W126 concentrations. Ozone concentrations in GRSM have been among the highest in the eastern U.S. In the recent conditions scenario, the grid cells representing 44 percent of GRSM had three year average W126 index value above 15 ppm-hrs. After adjustments to just meet the current standard, no grid cell had a three-year average W126 index value above 7 ppm-hrs. In the recent conditions scenario for ROMO, three-year average W126 index values for all grid cells were above 15 ppm-hrs. In the current standard scenario, values for 59 percent of the park were below 7 ppm-hrs. For SEKI, three-year average W126 index values for all grid cells were above 15 ppm-hrs in the recent conditions scenario, but dropped below 7 ppm-hrs for the current standard scenario (U.S. EPA, 2014a, section 7.4).

In summary, these analyses indicate that O₃ concentrations in U.S. national parks in recent years correspond to W126 index values at which some foliar injury may occur, with variation associated with relative soil moisture conditions. None of the 214 parks assessed are estimated to exceed the annual benchmark criteria for the base scenario (W126 >10.46 ppm-hrs) after adjusting air quality to meet the current standard. Although adjusted scenarios to just meet the current standard indicate substantial reductions in three-year average W126 index values estimated by the VNA approach, some individual year values may range higher. The case study analysis of three parks indicates the potential for appreciable ecosystem services impact associated with foliar injury. While these analyses indicate the potential for foliar injury to occur under conditions that meet the current standard, the extent of foliar injury that might be expected under such conditions is unclear from these analyses.

- **To what extent are the exposures and risks remaining upon simulating just meeting the current O₃ standard important from a public welfare perspective?**

The screening level assessment, as described above, indicates that risk of visible foliar injury is likely to be lower in most national parks after simulating just meeting the current standard. Based on the national-scale analysis, visible foliar injury would likely continue to occur at lower O₃ exposures, including some sensitive species growing in areas (e.g., National Parks and other Class I areas) that may provide important cultural ecosystem services to the public. Staff notes that such occurrences might reasonably be considered to have some importance from a public welfare perspective, as discussed in section 5.1 above.

- **What are the ecosystem services potentially affected by visible foliar injury, to what degree can the magnitude of these effects be qualitatively or quantitatively characterized, and to what extent are they important from a public welfare perspective?**

The ecosystem services most likely to be affected by O₃-induced foliar injury are cultural services, including aesthetic value and outdoor recreation. Aesthetic value and outdoor recreation depend on the perceived scenic beauty of the environment. Many outdoor recreation activities directly depend on the scenic value of the area, in particular scenic viewing, wildlife-watching, hiking, and camping. These activities and services are of significant importance to public welfare as they are enjoyed by millions of Americans every year and generate millions of dollars in economic value (U.S. EPA, 2014a, Chapter 5, Chapter 7). These aesthetic values are at risk of impairment because of O₃-induced damage directly due to foliar injury. Other ecosystem services that have also been found to be associated with O₃-sensitive plants include those that fall under the categories of provisioning. For example, several tribes have indicated that many of the known confirmed O₃-sensitive species (including bioindicator species) are culturally significant (see Appendix 5-A). Although data are not available to explicitly quantify these negative effects on ecosystem services, several qualitative analyses conducted in the WREA are summarized below.

To assess the effects of visible foliar injury on recreation, the WREA reviewed the NSRE, as well as the 2006 National Survey of Fishing, Hunting, and Wildlife-Associated Recreation (FHWAR) and a 2006 analysis done for the Outdoor Industry Foundation (OIF). According to the NSRE, some of the most popular outdoor activities are walking, including day hiking and backpacking; camping; bird watching; wildlife watching; and nature viewing. Participant satisfaction with these activities can depend on the quality of the natural scenery, which can be adversely affected by O₃-related visible foliar injury. According to the FHWAR and the OIF reports, the total expenditures across wildlife watching activities, trail-based activities, and camp-based activities are approximately \$230 billion dollars annually. While the WREA could not quantify the magnitude of the impacts of O₃ damage to the scenic beauty and outdoor recreation, the existing losses associated with current O₃-related foliar injury are reflected in reduced outdoor recreation expenditures (U.S. EPA, 2014a, section 7.1).

The WREA also assessed O₃ impacts on cultural ecosystem services related to foliar injury at three national parks – Great Smoky Mountains National Park, Rocky Mountain National Park, and Sequoia/Kings National Parks - by considering information on visitation patterns, recreational activities and visitor expenditures. The analysis included percent cover of species sensitive to foliar injury and focused on the overlap between recreation areas within the park and elevated W126 concentrations. All three of these park units are in areas that are known

to have high ambient O₃ concentrations, have vegetation maps, and have species that are considered O₃-sensitive. Using GIS, the NPS vegetation maps were compared to the national O₃ surface to illustrate where foliar injury may be occurring, particularly with respect to park amenities such as trails (U.S. EPA, 2014a, section 7.4).

Great Smoky Mountains National Park is prized, in part, for its rich species diversity. The large mix of species includes 37 O₃-sensitive species and many areas contain several sensitive species. With 3.8 million hikers using the trails every year and hikers' WTP over \$266 million for that activity, even a small benefit of reducing O₃ damage in the park could result in a significant economic value. Ozone concentrations in Great Smoky Mountains National Park have been among the highest in the eastern U.S. – at times twice as high as neighboring cities such as Atlanta (U.S. EPA, 2014a, p. 7-52). Unlike Great Smoky Mountains National Park, sensitive species cover in Rocky Mountain National Park is driven by a few O₃-sensitive species (7 species) and most notably by Quaking Aspen. This is significant in that many of the visitors to Rocky Mountain National Park visit specifically to see this tree in its fall foliage. Given 1.5 million hikers in Rocky Mountain National Park and their \$70 million WTP for the hiking experience, even a small improvement in the scenic value could be economically significant (U.S. EPA, 2014a, section 7.4.2, p. 7-56). Sequoia/Kings National Parks is home to 12 identified sensitive species. Again, although the EPA is not able to quantify the impact of this scenic damage on hiker satisfaction for hikers in Sequoia/Kings National Parks and their \$26 million WTP for the experience, even a small improvement in the scenic value could be economically significant ((U.S. EPA, 2014a, section 7.4.3, p. 7-63).

- **What are the uncertainties associated with this information and what is the level of confidence associated with those estimates?**

Uncertainties associated with these analyses are discussed in the WREA, sections 7.5 and 8.5.3, and in WREA Table 7-24. As discussed in the WREA (section 8.5.3), evaluating soil moisture is more subjective than evaluating O₃ exposure because of its high spatial and temporal variability within the O₃ season, and there is considerable subjectivity in the categorization of relative drought. The WREA generally concludes that the spatial and temporal resolution for the soil moisture data is likely to underestimate the potential of foliar injury that could occur in some areas. In addition, there is lack of a clear threshold for drought below which visible foliar injury would not occur. In general, low soil moisture reduces the potential for foliar injury, but injury could still occur, and the degree of drought necessary to reduce potential injury is not clear. Due to the absence of biosite injury data in the Southwest region and limited biosite data in the West and West North Central regions, the benchmarks applied may not be applicable to these regions. The WREA applied the benchmarks from the national-scale analysis to a screening-level analysis

of 214 national parks and case studies of three national parks. Therefore, uncertainties in the foliar injury benchmarks are propagated into the national park analyses.

There are also important uncertainties in the estimated O₃ concentrations for the different air quality scenarios evaluated (U.S. EPA, 2014a, section 8.5), as discussed earlier in this section. These uncertainties only apply to the national park case studies because these are the only foliar injury analyses that rely on the air quality scenarios, but any uncertainties in the air quality analyses are propagated into the those analyses. Additional uncertainties are associated with the national park case studies. Specifically, there is uncertainty inherent in survey estimates of participation rates, visitor spending/economic impacts, and willingness-to-pay. These surveys potentially double-count impacts based on the allocation of expenditures across activities but also potentially exclude other activities with economic value. In general, the national level surveys apply standard approaches, which minimize potential bias. Other sources of uncertainty are associated with the mapping, including park boundaries, vegetation species cover, and park amenities, such as scenic overlooks and trails. In general, the WREA concludes that there is high confidence in the park mapping (U.S. EPA, 2014, Table 7-24).

5.5 OTHER WELFARE EFFECTS

In addition to the welfare effects discussed in the previous sections, there is evidence of other O₃ effects, such as those related to climate impacts that we consider here. In this section, the WREA national-scale analyses of the effects of insect damage to forests related to elevated O₃ exposures are considered in section 5.5.1, and a case study-scale characterization of the effect community composition changes on forest susceptibility and fire regulation in California is considered in section 5.5.2. As above, these sections, where possible, consider the WREA information regarding risk remaining under adjusted conditions just meeting the current standard and associated uncertainties (U.S. EPA 2014a, section 8.5). Chapters 5, 6, and 7 of the WREA also qualitatively assessed additional ecosystem services, including regulating services such as hydrologic cycle and pollination; provisioning services such as commercial non-timber forest products; and cultural services with aesthetic and non-use values. The information associated with these latter effects is insufficient to inform the target protection of the standard. The effects of O₃ on climate are also considered in section 5.5.3 below, drawing primarily on the evidence presented in the ISA (U.S. EPA 2013, chapter 10).

5.5.1 Forest Susceptibility to Insect Infestation

Ozone in ambient air can contribute to increased susceptibility of some forests to infestation by some chewing insects, including the southern pine beetle and western bark beetle (U.S. EPA 2013, chapter 9; U.S. EPA 2014a, sections 5.3.3 and 5.4). These infestations can

cause economically significant damage to tree stands and the associated timber production. The WREA described the potential impacts of this effect on timber markets (U.S. EPA 2014a, section 5.4). In the short-term, the immediate increase in timber supply that results from the additional harvesting of damaged timber depresses prices for timber and benefits consumers. In the longer-term, the decrease in timber available for harvest raises timber prices, potentially benefitting producers. The USFS reports timber producers have incurred losses of about \$1.4 billion (2010\$), and wood-using firms have gained about \$966 million, due to beetle outbreaks between 1977 and 2004. It is not possible to attribute a portion of these impacts resulting from the effect of O₃ on trees' susceptibility to insect attack; however, the losses are embedded in the estimates cited and any welfare gains from decreased O₃ would positively impact the net economic impact. However, it is important to note that CASAC clarified that spatial association is not causation, even though expert opinion relates O₃-exposure to bark beetle infestation (Frey, 2014, p. 12).

To provide some quantitative estimates related to insect infestation-related risks, the WREA reported the estimates of 3-year average W126 index values in areas estimated to be at risk of greater than 25% timber loss (high loss) due to pine beetle infestation. This was done for all six WREA air quality scenarios. For example, for the recent conditions scenario, approximately 57 percent of the at-risk area has W126 estimates above 15 ppm-hrs, with the percentage dropping to approximately five percent in the current standard scenario (U.S. EPA 2014a, section 5.4).

5.5.2 Fire Regulation

Evidence indicates that fire regime regulation may also be negatively affected by O₃ exposure (U.S. EPA 2013, chapter 9; U.S. EPA 2014a, section 5.3.3). For example, Grulke et al. (2008) reported various lines of evidence indicating that O₃ exposure may contribute to southern California forest susceptibility to wildfires by increasing leaf turnover rates and litter, increasing fuel loads on the forest floor. According to the National Interagency Fire Center, in the U.S. in 2010 over 3 million acres burned in wildland fires and an additional 2 million acres were burned in prescribed fires. From 2004 to 2008, Southern California alone experienced, on average, over 4,000 fires per year burning, on average, over 400,000 acres per fire. The California Department of Forestry and Fire Protection (CAL FIRE) estimated that losses to homes due to wildfire were over \$250 million in 2007 (CAL FIRE, 2008). In 2008, CAL FIRE's costs for fire suppression activities were nearly \$300 million (CAL FIRE, 2008).

The WREA developed maps that overlay the mixed conifer forest area of California with areas of moderate or high fire risk defined by CAL FIRE and with recent W126 concentrations

and surfaces adjusted to just meet existing and alternative standards. The highest fire risk and highest O₃ concentrations overlap with each other, as well as with significant portions of mixed conifer forest. In the recent concentrations scenario, over 97 percent of mixed conifer forest area has average W126 index values over 7 ppm-hrs with a moderate to severe fire risk, and 74 percent has average W126 index values over 15 ppm-hrs with a moderate to severe fire risk. The scenario for air quality adjusted to just meet the current standard, almost all of the mixed conifer forest area with a moderate to high fire risk shows a reduction in O₃ to below a W126 index value of 7 ppm-hrs (average across three years of scenario). In the scenario for an average W126 index value of 15 ppm-hrs, all but 0.18 percent of the area has average index values below 7 ppm-hrs, and for the W126 scenarios of 11 and 7 ppm-hrs, all of the moderate to high fire threat area has estimated average W126 index values below 7 ppm-hrs (U.S. EPA 2014a, section 5.3.3, Figure 5-3). However, it is important to note that CASAC clarified that spatial association is not causation, but expert opinion relates O₃-exposure to fire risk (Frey, 2014, p. 12).

5.5.3 Ozone Effects on Climate

Tropospheric O₃ is a major greenhouse gas, third in importance after carbon dioxide (CO₂) and methane (CH₄). While the developed world has successfully reduced emissions of O₃ precursors in recent decades, many developing countries have experienced large increases in precursor emissions and these trends are expected to continue, at least in the near term (U.S. EPA 2013, section 10.3.6.2). Projections of radiative forcing due to changing O₃ over the 21st century show wide variation, due in large part to the uncertainty of future emissions of source gases (U.S. EPA 2013, section 10.3.6.2). In the near-term (2000-2030), projections of O₃ radiative forcing range from near zero to +0.3 W/m², depending on the emissions scenario (U.S. EPA 2013, section 10.3.6.2; Stevenson et al., 2006). Reduction of tropospheric O₃ concentrations could therefore provide an important means to slow climate change in addition to the added benefit of improving surface air quality (U.S. EPA, 2013, section 10.5).

It is clear that increases in tropospheric O₃ lead to warming. However the precursors of O₃ also have competing effects on the greenhouse gas CH₄, complicating emissions reduction strategies. A decrease in CO or VOC emissions would enhance OH concentrations, shortening the lifetime of CH₄, while a decrease in NO_x emissions could depress OH concentrations in certain regions and lengthen the CH₄ lifetime (U.S. EPA, 2013, section 10.5).

Abatement of CH₄ emissions would likely provide the most straightforward means to address O₃-related climate change since CH₄ is itself an important precursor of background O₃ (West et al., 2007; West et al., 2006; Fiore et al., 2002). A set of global abatement measures identified by West and Fiore (2005) could reduce CH₄ emissions by 10% at a cost savings, decrease background O₃ by about 1 ppb in the Northern Hemisphere summer, and lead to a

global net cooling of 0.12 W/m². West et al. (2007) explored further the benefits of CH₄ abatement, finding that a 20% reduction in global CH₄ emissions would lead to greater cooling per unit reduction in surface O₃, compared to 20% reductions in VOCs or CO (U.S. EPA, 2013, section 10.5).

Important uncertainties remain regarding the effect of tropospheric O₃ on future climate change. To address these uncertainties, further research is needed to: (1) improve knowledge of the natural atmosphere; (2) interpret observed trends of O₃ in the free troposphere and remote regions; (3) improve understanding of the CH₄ budget, especially emissions from wetlands and agricultural sources, (4) understand the relationship between regional O₃ radiative forcing and regional climate change; and (5) determine the optimal mix of emissions reductions that would act to limit future climate change (U.S. EPA, 2013, section 10.5).

The IPCC has estimated the effect of the tropospheric O₃ change since preindustrial times on climate to be about 25-40% of the anthropogenic CO₂ effect and about 75% of the anthropogenic CH₄ effect (IPCC, 2007). There are large uncertainties in the radiative forcing estimate attributed to tropospheric O₃, making the effect of tropospheric O₃ on climate more uncertain than the effect of the long-lived greenhouse gases (U.S. EPA, 2013, section 10.5).

Radiative forcing does not take into account the climate feedbacks that could amplify or dampen the actual surface temperature response. Quantifying the change in surface temperature requires a complex climate simulation in which all important feedbacks and interactions are accounted for. As these processes are not well understood or easily modeled, the surface temperature response to a given radiative forcing is highly uncertain and can vary greatly among models and from region to region within the same model (U.S. EPA, 2013, section 10.5).

As discussed in section 5.2 above, O₃ exposure is associated with reduced forest tree growth, productivity, and carbon storage. Therefore, reducing O₃ exposure would potentially increase carbon storage in O₃-sensitive trees, which could also have climate effects.

5.5.4 Additional Effects

Recent information available since the last review considers the effects of O₃ on chemical signaling in insect and wildlife interactions. Specifically, studies on O₃ effects on pollination and seed dispersal, defenses against herbivory and predator-prey interactions all consider the ability of O₃ to alter the chemical signature of VOCs emitted during these pheromone-mediated events. The effects of O₃ on chemical signaling between plants, herbivores and pollinators as well as interactions between multiple trophic levels is an emerging area of study that may result in further elucidation of O₃ effects at the species, community and ecosystem-level (U.S. EPA, 2013, p. 9-98).

5.6 CASAC ADVICE

This section discusses CASAC advice regarding the adequacy of the existing secondary standard with respect to the 2008 review, the 2010 reconsideration of the 2008 review, and most recently its advice in this review, initiated in September 2008, in its letter to the Administrator on the second draft WREA and PA. To give an overview, following the 2008 decision to revise the secondary standard by setting it identical to the revised primary standard, CASAC conveyed additional advice to the Administrator regarding that decision. Shortly after that, several petitioners filed suit challenging the decision and in September 2009, the EPA announced its intention to reconsider the 2008 standards, issuing a notice of proposed rulemaking in January 2010 (75 FR 2938). Soon after, the EPA solicited CASAC review of that proposed rule and in January 2011 solicited additional advice. This proposal was based on the scientific and technical record from the 2008 rulemaking, including public comments and CASAC advice and recommendations. As further described in section 1.2.2 above, the EPA in the fall of 2011 did not promulgate final rulemaking in that process but decided to coordinate further proceedings on the reconsideration rulemaking with this ongoing periodic review.

More specifically, in April 2008, the members of the CASAC Ozone Review Panel sent a letter to EPA stating that “[i]n our most-recent letters to you on this subject - dated October 2006 and March 2007 - ... the Committee recommended an alternative secondary standard of cumulative form that is substantially different from the primary Ozone NAAQS in averaging time, level and form — specifically, the W126 index within the range of 7 to 15 ppm-hours, accumulated over at least the 12 ‘daylight’ hours and the three maximum ozone months of the summer growing season” (Henderson, 2008). The letter continued:

The CASAC now wishes to convey, by means of this letter, its additional, unsolicited advice with regard to the primary and secondary Ozone NAAQS. In doing so, the participating members of the CASAC Ozone Review Panel are unanimous in strongly urging you or your successor as EPA Administrator to ensure that these recommendations be considered during the next review cycle for the Ozone NAAQS that will begin next year ... The CASAC was also greatly disappointed that you failed to change the form of the secondary standard to make it different from the primary standard. As stated in the preamble to the Final Rule, even in the previous 1996 ozone review, “there was general agreement between the EPA staff, CASAC, and the Administrator, ... that a cumulative, seasonal form was more biologically relevant than the previous 1-hour and new 8-hour average forms (61 FR 65716)” for the secondary standard.....Unfortunately, this scientifically-sound approach of using a cumulative exposure index for welfare effects was not adopted...

In response to the EPA's solicitation of their advice on the Agency's proposed rulemaking as part of the reconsideration, CASAC conveyed their support for the proposed approach as follows (Samet, 2010).

CASAC also supports EPA's secondary ozone standard as proposed: a new cumulative, seasonal standard expressed as an annual index of the sum of weighted hourly concentrations (i.e., the W126 form), cumulated over 12 hours per day (8am to 8pm) during the consecutive 3-month period within the ozone season with the maximum index value, set as a level within the range of 7 to [1]5 ppm-hours. This W126 metric can be supported as an appropriate option for relating ozone exposure to vegetation responses, such as visible foliar injury and reductions in plant growth. We found the Agency's reasoning, as stated in the Federal Register notice of January 19, 2010, to be supported by the extensive scientific evidence considered in the last review cycle. In choosing the W126 form for the secondary standard, the Agency acknowledges the distinction between the effects of acute exposures to ozone on human health and the effects of chronic ozone exposures on welfare, namely that vegetation effects are more dependent on the cumulative exposure to, and uptake of, ozone over the course of the entire growing season (defined to be a minimum of at least three months).

In its advice offered early in the current review, based on the updated scientific and technical record since the 2008 rulemaking, CASAC indicated that a conclusion that the current standard is inadequate to protect vegetation and ecosystems is "warranted" although it stated that the foundation needs to be broadened beyond analysis focused on Class I areas and trees to include "effects on sensitive crops, trees in regions outside of Class I areas, and additional ecosystem impacts" (Frey and Samet, 2012, p. 2). The Panel additionally endorsed the first draft PA discussions and conclusions on biologically relevant exposure metrics, stating that "the focus on the W126 form is appropriate" and that "there is a strong justification made for using a cumulative and weighted exposure standard for welfare effects (i.e., the W126)..." (Frey and Samet, 2012, p. 2).

In its letter dated June 26, 2014, CASAC again concluded that "the current secondary standard is not adequate to protect against current and anticipated welfare effects of ozone on vegetation..." (Frey, 2014, p. iii) and that "the form of the standard should be changed from the current 8-hour form to the cumulative W126 index and... that the discussion provides an appropriate and sufficient rationale" (Frey, 2014, p. 12). CASAC then further states that

"[t]hus, based on identification of known or anticipated ozone effects that are adverse to public welfare, taking into account the weight of evidence for causality of exposure to ozone and adverse welfare effects as given in Table 2-4 of the *Integrated Science Assessment*; results of the Second Draft WREA with regard to assessment of relative

biomass loss for tree species, foliar injury, and crop yield loss; and the breadth of adverse welfare effects for ecosystem services, foliar injury, and crop loss, the CASAC recommends that the secondary standard for ozone be revised as follows: (1) ozone should be the indicator; (2) the form and summation time of the standard should be the W126 index summed over the highest three-month interval during a year, based on accumulation over the 08:00 a.m. – 08:00 p.m. daytime 12-hour period; and (3) the level of the standard should be between 7 ppm-hrs and 15 ppm-hrs. These recommendations are based on scientific evidence of adverse effect associated with the presence of ozone in ambient air. Note that these levels are based on an annual form of the standard.” (Frey, 2014, p. 15).

With respect to the averaging time, CASAC additionally states that it “does not recommend the use of a three-year averaging period. We favor a single-year averaging period, which will provide more protection for annual crops and for the anticipated cumulative effects on perennial species. The scientific analyses considered in this review, and the evidence upon which they are based, are from single-year results. If a 3-year averaging period is established, then the upper limit will need to be reduced to protect against one-year ozone peaks” (Frey, 2014, p. 13).

5.7 STAFF CONCLUSIONS ON ADEQUACY OF SECONDARY STANDARD

This section presents staff conclusions for the Administrator to consider in deciding whether the existing secondary O₃ standard is adequate and whether it should be retained or revised. Our conclusions are based on consideration of the assessment and integrative synthesis of information presented in the ISA, as well as our analyses of air quality distributions; analyses in the WREA; and the comments and advice of CASAC and public comment on earlier drafts of this document and on the ISA and WREA, as discussed above. Taking into consideration the responses to specific questions discussed above, we revisit the overarching policy question for this chapter:

- **Does the currently available scientific evidence and exposure/risk information, as reflected in the ISA and WREA, support or call into question the adequacy and/or appropriateness of the protection afforded by the current secondary O₃ standard?**

As an initial matter, we note that the CAA does not require that a secondary standard be protective of all effects associated with a pollutant in the ambient air, but only those considered adverse to the public welfare (as described in section 1.3.2 above). In helping inform the Administrator’s judgments with respect to the adversity of the effects to public welfare, we have considered the scientific evidence and risk/exposure information in light of the paradigm used in the last review that takes into account the variation in public welfare significance of O₃-related

vegetation effects when evaluating the potential adversity of the currently available evidence. As discussed in Section 5.1, this paradigm recognized that the significance to the public welfare of O₃-induced effects on sensitive vegetation growing within the U.S. can vary depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located. Accordingly, any given O₃-related effect on vegetation and ecosystems (e.g., biomass loss, crop yield loss, visible foliar injury) may be judged to have a different degree of impact on the public welfare depending, for example, on whether that effect occurs in a Class I area, a city park, or commercial cropland. In the 2010 proposed reconsideration, the Administrator proposed to place the highest priority and significance on vegetation and ecosystem effects to sensitive species that are known to or are likely to occur in federally protected areas such as national parks and other Class I areas, or on lands set aside by states, tribes and public interest groups to provide similar benefits to the public welfare (75 FR 3023/24), recognizing that effects occurring in such areas would likely have the highest potential for being classified as adverse to the public welfare, due to the expectation that these areas need to be maintained in a more pristine condition to ensure their intended use is met.

In addition, there is also sufficient support to explicitly include consideration of impacts to ecosystem goods and services. Although ecosystem services were not explicitly considered in the Administrator's decision in the last review, they were recognized as an important category of public welfare effects (73 FR 16492). The CASAC letter also provides support for this approach. The inclusion of ecosystem goods and services in this paradigm brings with it a number of additional considerations. Specifically, when considering the public welfare benefits from these goods and services, it is important to note that they can accrue across a range of dimensions, including spatial, temporal, and social, and these likely will vary depending on the type of effect being characterized. For example, ecosystems can cover a range of spatial scales, and the services they provide can accrue locally or be distributed more broadly such as when crops are sold and eaten locally and/or also sold in regional, national and world markets. Ecosystem services can likewise be realized over a range of temporal scales from immediate up to long term (e.g., the removal of air pollutants that have a short-term impact on human health but are also climate forcers with long atmospheric lifetimes, which the removal of may have immediate as well as long-term benefits). The size of the societal unit receiving benefits from ecosystem services can also vary dramatically. For example, a national park can provide direct recreational services to the thousands of visitors that come each year, but also provide an indirect value to the millions who may not visit but receive satisfaction from knowing it exists and is preserved for the future (U.S. EPA, 2014a, chapter 5, section 5.5.1).

We thus recognize the usefulness of evaluating the scientific evidence regarding these effects in the context of the most recent paradigm discussed above. This paradigm integrates the concepts of: 1) variability in public welfare significance given intended use and value of the affected entity, such as individual species; 2) relevance of associated ecosystem services to public welfare; and 3) variability in spatial, temporal, and social distribution of ecosystem services associated with known and anticipated welfare effects. In so doing, we recognize that there is no bright-line rule delineating the set of conditions or scales at which known or anticipated effects become adverse to public welfare. Thus, the evidence and exposure/risk information discussed in this chapter will be further evaluated in Chapter 6 in light of the concepts incorporated in this paradigm to help inform the Administrator's judgments with respect to the potential adversity of the effects to the public welfare.

With respect to the scientific evidence, the longstanding evidence base on the phytotoxic effects of O₃ demonstrates that O₃-induced effects that occur at the subcellular and cellular levels, at sufficient magnitudes propagate up to larger spatial scales. The ISA summarizes the coherence across the full range of effects, from the least serious to the most serious, as follows (U.S. EPA, 2013, p. 1-8):

The welfare effects of O₃ can be observed across spatial scales, starting at the subcellular and cellular level, then the whole plant and finally, ecosystem-level processes. Ozone effects at small spatial scales, such as the leaf of an individual plant, can result in effects along a continuum of larger spatial scales. These effects include altered rates of leaf gas exchange, growth, and reproduction at the individual plant level, and can result in broad changes in ecosystems, such as productivity, carbon storage, water cycling, nutrient cycling, and community composition.

Many of the recent studies evaluated in this review have focused on and further increased our understanding of the molecular, biochemical and physiological mechanisms that explain how plants are affected by O₃, in the absence of other stressors, particularly in the area of genomics (U.S. EPA, 2013, Chapter 9, section 9.3). These recent studies, in combination with the extensive and long-standing evidence, have further strengthened the coherence and consistency of the entire body of research, so that our confidence in the supporting science is stronger than in the previous review.

Based on its assessment of the strength of the science, the ISA determined that the relationship that exists between exposure to O₃ in ambient air and visible foliar injury effects on vegetation, reduced vegetation growth, reduced productivity in terrestrial ecosystems, reduced yield and quality of agricultural crops and alteration of below-ground biogeochemical cycles (U.S. EPA 2013, Table 1-2) is causal. Additionally, the ISA determined that a likely to be causal

relationship exists between exposures to O₃ in ambient air and reduced carbon sequestration in terrestrial ecosystems, alteration of terrestrial ecosystem water cycling and alteration of terrestrial community composition (U.S. EPA, 2013, Table 1-2).

Recent studies also continue to provide strong and consistent evidence that adverse vegetation effects are attributable to cumulative seasonal O₃ exposures. On the basis of the entire body of evidence in this regard, the ISA concludes that “quantifying exposure with indices that cumulate hourly O₃ concentrations and preferentially weight the higher concentrations improves the explanatory power of exposure/response models for growth and yield, over using indices based on mean and peak exposure values” (U.S. EPA, 2013, p. 2-44). Thus, as in other recent reviews, the evidence continues to provide a strong basis for concluding that it is appropriate to judge impacts of O₃ on vegetation, related effects and services, and the level of public welfare protection achieved, using a cumulative, seasonal exposure metric, such as the W126-based metric. In addition, CASAC has consistently since the 1997 review expressed support for the use of such a metric as the most appropriate form for the secondary NAAQS. In its most recent letter on the second draft PA, CASAC states that it “concur[s] with the justification in this section that the form of the standard should be changed from the current 8-hr form to the cumulative W126 index and finds that the discussion provides an appropriate and sufficient rationale” (Frey, 2014, p. 12). Thus, based on the consistent and well-established evidence described above, we conclude that the most appropriate and biologically relevant way to relate O₃ exposure to plant growth, and to determine what would be adequate protection for public welfare effects attributable to the presence of O₃ in the ambient air, is to characterize exposures in terms of a cumulative seasonal form, and in particular the W126 metric.

Accordingly, in considering the current evidence and exposure/risk information with regard to the adequacy of public welfare protection it affords, we have considered both the evidence of vegetation and welfare impacts in areas of the U.S. likely to have met the current standard, as well as air quality information regarding W126 index values in such areas. In evaluating the adequacy of the current secondary standard, we first considered O₃ effects on tree growth, productivity and carbon storage and associated ecosystems and services. Recent studies confirm and extend the evidence of O₃-related effects on tree growth, productivity and carbon storage. Analysis of existing data conducted by the EPA staff and discussed in the ISA has substantially reduced the uncertainty associated with using OTC E-R functions to predict tree growth effects in the field, as described in section 5.2.1 above (U.S. EPA, 2013, section 9.6.3.2). The median of the composite E-R functions (green line), (U.S. EPA, 2014a, Figure 6-5, section 6.2.1.2) shows RBL for tree seedlings. We note CASAC’s advice that a 6% median RBL is unacceptably high, and that the 2% median RBL is an important benchmark to consider. The

median RBL is at or below 2% at the lowest W126 level assessed, 7 ppm-hrs. As the W126 level is incrementally increased, median RBL also increases incrementally, so that at W126 index values of 9, 11, 13, 15, 17, 19 and 21, the median RBL increases to 2.4%, 3.1%, 3.8%, 4.5%, 5.3%, 6.0% and 6.8%, respectively. Based on air quality analyses of 2009-2011 (Appendix 2B), there are approximately 342, 199, 92, 43, 24, 9, 3 and 0 monitors with 3-year average W126 index values above 7, 9, 11, 13, 15, 17, 19 and 21 ppm-hrs when meeting the current standard. We note that these counts of monitors are based on those meeting the current standard and that there are many monitors for the 2009-2011 period that do not meet the current standard and also are above the W126 values of 7-21 ppm-hrs.

We also consider it informative to examine the individual species responses and RBL over the same W126 range. We first note, based on Figure 5-1 (B) above that over the range of 7 to 17 ppm-hrs, 5 species maintain RBLs of less than 2%. These more tolerant species include Douglas fir, loblolly pine, Virginia pine, sugar maple and red maple. Two of these species (red maple and sugar maple) are estimated to have RBL levels above 2% at a W126 of 21. Black cherry, the most sensitive of the remaining six species, has RBL ranging from 35.57% at W126 of 17 down to 16.67% at the W126 index value of 7 ppm-hrs.

In addition, we also consider the growth effects associated with exposure concentrations at or below that of the current standard in Class I areas. Specifically, we found that there were 22 Class I areas that had monitor sites that have design values that meet the current standard, ranging from 67 to 75 ppb, and have 3-year average W126 index values that are above 15 ppm-hrs between the years of 1998 and 2012 (Table 5-2). Across these 22 Class I areas, the highest single-year W126 index values for these three-year periods ranged from 17.4 to 29.0 ppm-hrs. In 20 of the areas, distributed across eight states (AZ, CA, CO, KY, NM, SD, UT, WY) and four regions (west, southwest, west/north central and central), this range was 19.1 to 29.0 ppm-hrs, exposure values for which the corresponding median species RBL estimates equal or exceed 6%, which CASAC termed “unacceptably high”. In addition, given that other environmental factors can influence the extent to which O₃ may have the impact predicted by the E-R functions in any given year, we also note that the highest three year periods, that include these highest annual values for the 21 areas, are at or above 19 ppm-hrs, ranging up to 22.5 ppm-hrs (for which the median species RBL estimate is above 7%). Additionally, the highest three-year average W126 index value for each of the 22 areas (during periods meeting the current standard) was at or above 19 (ranging up to 22.5 ppm-hrs) in 11 areas, distributed among five states in the west and southwest regions (U.S. EPA, 2014c, Table 5-2, Appendix 5B).

In addition, quaking aspen and ponderosa pine are two tree species that are found in most of these 22 parks and have a sensitivity to O₃ exposure that places them near the middle of the

group for which E-R functions have been established. In the areas where ponderosa pine is present, the highest single year W127 index values ranged from 18.7 to 29.0 and the highest 3-year average W126 values in which these single year values are represented ranged from 15 to 22.5, with these three-year values above 19 ppm-hrs in eight areas across five states. The ponderosa pine RBL estimates for 29 and 22.5 ppm-hrs are approximately 12% and 9%, respectively. In the areas where quaking aspen is present, the highest single year W127 index values ranged from 19.2 to 26.7 ppm-hrs and the highest 3-year average W126 values in which these single year values are represented ranged from 15.0 to 22.2, with values above 19 ppm-hrs in eight areas across five states. The quaking aspen RBL estimates for 26.7 and 22.2 ppm-hrs are approximately 16% and 13%, respectively. Based on this, we predict growth effects associated with exposure concentrations at or below that of the current standard for most of these Class I areas. On the basis of such information, Table 5-2 provides evidence of the potential for significant growth loss in locations where ambient conditions meet the current standard. Based on this evidence, we note the occurrence in Class I areas, during periods where the current standard is met, of cumulative seasonal O₃ exposures of a magnitude that might reasonably be concluded to be important to public welfare.

Recent studies have provided additional evidence on tree biomass or growth effects associated with multiple year exposures in the field, including the potential for cumulative and carry-over effects. For example, a number of studies were conducted at a planted forest at the Aspen FACE site in Wisconsin where some researchers observed that the effects of O₃ on birch seeds (reduced weight, germination, and starch levels) could lead to a negative impact on species regeneration in subsequent years, and that the effect of reduced aspen bud size may have been related to the observed delay in spring leaf development. These effects suggest that elevated O₃ exposures have the potential to alter carbon metabolism of overwintering buds which may have subsequent effects in the following year. Other studies found that, in addition to affecting tree heights, diameters, and main stem volumes in the aspen community, elevated O₃ over a 7-year study period was reported to increase the rate of conversion from a mixed aspen-birch community to a community dominated by the more tolerant birch, leading the authors to conclude that elevated O₃ may alter intra- and inter-species competition within a forest stand (U.S. EPA, 2013, section 9.4.3).

While it is not possible at this time to identify the extent or magnitude of such effects in the field under exposures that may be associated with the current standard, their occurrence, on federal lands with special protections might reasonably be concluded to be an important public welfare consideration. We note here that the CASAC “concur[s] that biomass loss in trees is a relevant surrogate for damage to tree growth that affects ecosystem services such as habitat provision for wildlife, carbon storage, provision of food and fiber, and pollution removal.

Biomass loss may also have indirect process-related effects such as on nutrient and hydrologic cycles. Therefore, biomass loss is a scientifically valid surrogate of a variety of adverse effects to public welfare” (Frey, 2014, pp. 9-10).

In regard to the WREA analyses for risks for associated ecosystem services, we note that the WREA presents estimated changes in consumer and producer/farmer surplus associated with the change in forestry and agricultural yields. Changes in biomass affect individual tree species differently, and the overall effect on forest ecosystem productivity depends on the composition of forest stands and the relative sensitivity of trees within those stands. Economic welfare impacts resulting from just meeting the existing and alternative standards were largely similar between the forestry and agricultural sectors -- consumer surplus, or consumer gains, generally increased in both sectors because higher productivity under lower O₃ concentrations increased total yields and reduced market prices. Comparisons are not straightforward to interpret due to market dynamics. The national-scale analysis of carbon dioxide (CO₂) sequestration estimates more storage under the current standard compared to recent conditions, with somewhat smaller additional increases for the three W126 scenarios in comparison to the current standard scenario (U.S. EPA 2014a, Appendix 6B, Table B-10).

We additionally consider the WREA estimates of tree growth and ecosystem services provided by urban trees over a 25-year period for five urban areas based on case-study scale analyses that quantified the effects of biomass loss on carbon sequestration and pollution removal (U.S. EPA 2014a, sections 6.6.2 and 6.7).³⁰ The urban areas included in this analysis represent diverse geography in the Northeast, Southeast, and Central regions, although they do not include an urban area in the western U.S. Estimates of the effects of O₃-related biomass loss on carbon sequestration, for example, indicate the potential for an increase of somewhat more than a million metric tons of CO₂ equivalents for average W126 index values associated with meeting the current standard scenario as compared to recent conditions. Somewhat smaller additional increases are estimated for the three W126 scenarios in comparison to the current standard scenario (U.S. EPA 2014a, section 6.6.2 and Appendix 6D).

In considering the significance of these WREA analyses of risks for the associated ecosystem services for timber production, air pollution removal, and carbon sequestration, we note the large uncertainties associated with these analyses (see U.S. EPA 2014a, Table 6-27), and the potential to underestimate the response at the national scale. Thus, while we note that it is appropriate to consider predicted and anticipated impacts to these services in determining the adequacy of the protection afforded by the current standard, we also note that we place limited

³⁰ The WREA used the i-Tree model for the urban case studies. i-Tree is a peer-reviewed suite of software tools provided by USFS.

weight on the absolute magnitude of the risk results for these ecosystem service endpoints in light of these significant associated uncertainties.

In reaching conclusions regarding support for the adequacy of the current secondary standard provided by the currently available information on O₃-induced effects on trees and associated ecosystem services we note that: 1) there is robust evidence supporting the causal relationship between cumulative O₃ exposures and effects on tree growth, productivity, and carbon storage (U.S. EPA, 2013) and causal and likely to be causal relationships for several associated ecosystem services; 2) the tree seedling E-R functions evidence, which has been strengthened, demonstrates variability in sensitivity to O₃ across species; 3) estimated median RBLs are at or above 6%, a key CASAC benchmark, in several areas when air quality was at or below that of the current standard; 4) growth effects associated with exposure concentrations are predicted to occur in several Class I areas based on air quality from 1998-2012 that was at or below that of the current standard; 5) impacts from single year exposures can carry over to the subsequent year and/or cumulate over multiple years with repeated annual exposures; 6) evidence from both recent controlled chamber mechanism studies and field based exposure studies support earlier findings from OTC studies; and 7) WREA analyses show that O₃-induced biomass loss can impact ecosystem services provided by forests, including timber production, carbon storage, and air pollution removal, even when air quality is adjusted to just meet the current standard. Given the above, and noting CASAC views described above, staff concludes that the current evidence/risk information calls into question the adequacy of the public welfare protection afforded by the current standard from the known and anticipated adverse effects associated with O₃-induced impacts on tree growth, productivity and carbon storage, including the associated ecosystem services assessed in this review, and therefore it is appropriate to consider revision to provide increased protection.

With respect to crops, the detrimental effect of O₃ on crop production has been recognized since the 1960s, and recent O₃ concentrations in many areas across the U.S. are high enough that they might be expected to cause yield loss in a variety of agricultural crops including, but not limited to, soybeans, wheat, potatoes, watermelons, beans, turnips, onions, lettuces, and tomatoes (U.S. EPA, 2013, section 9.4.4). In general, the vast majority of the new scientific information confirms prior conclusions that exposure to O₃ can decrease growth and yield of crops. Recent research has highlighted the effects of O₃ on crop quality. Increasing O₃ concentration decreases nutritive quality of grasses, and decreases macro- and micro-nutrient concentrations in fruits and vegetable crops (U.S. EPA 2013, section 9.4.4). Recent studies continue to find yield loss levels in crop species studied previously under NCLAN that reflect the earlier findings. There has been little published evidence that crops are becoming more tolerant of O₃ (U.S. EPA, 2006a; U.S. EPA 2013). This is especially evident in the research on

soybean. The 2013 ISA reported comparisons between yield predictions based on data from cultivars used in NCLAN studies, and yield data for modern cultivars from SoyFACE (U.S. EPA, 2013, section 9.6.3). They confirm that the average response of soybean yield to O₃ exposure has not changed in current cultivars. In addition, satellite and ground-based O₃ measurements have been used to assess yield loss caused by O₃ over the continuous tri-state area of Illinois, Iowa, and Wisconsin. The results showed that O₃ concentrations reduced soybean yield, which correlates well with the previous results from FACE- and OTC-type experiments (U.S. EPA, 2013, section 9.4.4.1). Thus, the recently available evidence, as assessed in the ISA, continues to support the conclusions of the 1996 and 2006 CDs that ambient O₃ concentrations can reduce the yield of major commodity crops in the U.S.

The currently available evidence, as assessed in the ISA, continues to support the use of E-R functions for crops based on OTC experiments. Further, important uncertainties have been reduced regarding the E-R functions for crop yield loss, especially for soybean. In general, the ISA reports consistent results across exposure techniques and across crop varieties (U.S. EPA 2013, section 9.6.3.2). Soybean, which is the second-most planted field crop in the U.S.,³¹ would be predicted to have no more than 5% RYL at a W126 index value of 12 ppm-hrs, based on the E-R function. Staff analyses of recent monitoring data (2009-2011) indicate that O₃ concentrations in multiple agricultural areas in the U.S. that meet the current standard correspond to W126 index levels above 12 ppm-hrs. With regard to crops, CASAC states that it “concur[s] that another important surrogate for damage that is adverse to public welfare is crop loss. Crops provide food and fiber services to humans. Evaluation of market-based welfare effects of ozone exposure in forestry and agricultural sectors is an appropriate approach to take into account damage that is adverse to public welfare” (Frey, 2014, p. 10). However, as we describe in section 5.3 above, determining at what point O₃-induced crop yield loss becomes adverse to the public welfare is still unclear, given that it is heavily managed with additional inputs that have their own associated markets and that benefits can be unevenly distributed between producers and consumers. We further note that a standard set to provide requisite protection for trees could also potentially achieve appropriate protection for commodity crops.

In reaching conclusions regarding support for the adequacy of the current secondary standard provided by the currently available information on O₃-induced effects on crops, we note that 1) there is clear and robust evidence supporting the causal relationship between cumulative O₃ exposures and effects on crop yields and quality (U.S. EPA, 2013); 2) the crop E-R functions evidence, which has been strengthened, demonstrates variability in sensitivity to O₃ across species; 3) evidence from both recent controlled chamber mechanism studies and field based

³¹ <http://www.ers.usda.gov/topics/crops/soybeans-oil-crops/background.aspx>

exposure studies support earlier findings from OTC studies; 4) evidence continues to show that crops, and in particular soybean, has not become more tolerant of O₃ (U.S. EPA, 2013, section 9.6.3, 9.4.4.1); 5) WREA analyses show that O₃-induced crop yield loss can impact producer and consumer surpluses and the interaction between agriculture and timber production.

Given the above, and noting CASAC views described above as well as the difficulty in assessing adversity to public welfare of these effects, staff concludes that the current evidence/risk information calls into question the adequacy of the public welfare protection afforded by the current standard from the known and anticipated adverse effects associated with O₃-induced impacts on crop yields and associated services assessed in this review, and therefore, it is appropriate to consider revision to provide increased protection.

With respect to foliar injury, visible foliar injury surveys are used by the federal land managers to assess potential O₃ impacts in Class I areas (USFS, NPS, FWS, 2010). Given this focus on visible foliar injury, O₃-induced impacts have the potential to impact the public welfare in scenic and/or recreational areas on an annual basis. Visible foliar injury is associated with important cultural and recreational ecosystem services to the public, such as scenic viewing, wildlife-watching, hiking, and camping, that are of significance to the public welfare and enjoyed by millions of Americans every year, generating millions of dollars in economic value (U.S. EPA 2014a, section 7.1). In addition, several tribes have indicated that many of the known confirmed O₃-sensitive species (including bioindicator species) are culturally significant (see Appendix 5-A). We further note that CASAC “concur[s] that visible foliar injury can impact public welfare by damaging or impairing the intended use or service of a resource. Visible foliar injury that is adverse to public welfare can include: visible damage to ornamental or leafy crops that affects their economic value, yield, or usability; visible damage to plants with special cultural significance; and visible damage to species occurring in natural settings valued for scenic beauty or recreational appeal” (Frey, 2014, p. 10).

New research on visible foliar injury includes: 1) controlled exposure studies; 2) multi-year field surveys; and 3) USFS FHM/FIA biomonitoring program surveys and assessments. In addition to supporting the ISA causal determination, these studies also address some uncertainties identified in the previous review (i.e., influence of soil moisture on visible foliar injury development) and further show that visible foliar injury can occur under conditions where 8-hour average O₃ concentrations are or would be expected to be below the level of the current standard (e.g., Kline et al., 2008, as discussed in section 5.4.1 above). Incidence of visible foliar injury symptoms on O₃-sensitive vegetation has also been documented in the field in federally protected areas that have met the current standard. Importantly, these O₃-induced vegetation effects have been identified by the federal land managers as a diagnostic tool for informing conclusions regarding potential ozone impacts on potentially sensitive AQRVs and were found

in Class I areas that have particular public welfare significance in light of direction from Congress that these areas as merit a high level of protection (75 FR 3023/3024).

The studies mentioned above also provide additional information regarding the role of soil moisture in influencing visible foliar injury response (U.S. EPA 2013, section 9.4.2). These studies confirm that adequate soil moisture creates an environment conducive to greater visible foliar injury in the presence of O₃ than drier conditions. As stated in the ISA, “[a] major modifying factor for O₃-induced visible foliar injury is the amount of soil moisture available to a plant during the year that the visible foliar injury is being assessed ... because lack of soil moisture generally decreases stomatal conductance of plants and, therefore, limits the amount of O₃ entering the leaf that can cause injury” (U.S. EPA, 2013, p. 9-39). As a result, “many studies have shown that dry periods in local areas tend to decrease the incidence and severity of O₃-induced visible foliar injury; therefore, the incidence of visible foliar injury is not always higher in years and areas with higher O₃, especially with co-occurring drought (Smith, 2012; Smith et al., 2003)” (U.S. EPA, 2013, p. 9-39). This “...partial ‘protection’ against the effects of O₃ afforded by drought has been observed in field experiments (Low et al., 2006) and modeled in computer simulations (Broadmeadow and Jackson, 2000)” (U.S. EPA, 2013, p. 9-87). In considering the extent of any protective role of drought conditions, however, the ISA also notes that other studies have shown that “drought may exacerbate the effects of O₃ on plants (Pollastrini et al., 2010; Grulke et al., 2003)” and that “[t]here is also some evidence that O₃ can predispose plants to drought stress (Maier-Maercker, 1998)”. Accordingly, the ISA concludes that “the nature of the response is largely species-specific and will depend to some extent upon the sequence in which the stressors occur” (U.S. EPA, 2013, p. 9-87). However, such uncertainties associated with describing the potential for foliar injury and its severity or extent of occurrence for any given air quality scenario due to confounding by soil moisture levels make it difficult to identify an appropriate degree of protection (as well as ambient O₃ exposure conditions that might be expected to provide that protection).

We note the WREA analyses of the nationwide dataset (2006-2010) for USFS FHM/FIA biosites described in section 5.4.2 above, including the observation that the proportion of biosites with injury varies with soil moisture conditions and O₃ W126 index values (U.S. EPA 2014a, Chapter 7, Figure 7-10). The evidence of O₃-attributable visible foliar injury incidence occurring in USFS FHM/FIA biosites shows that the proportion of biosites showing foliar injury incidence increases steeply with W126 index values up to approximately 10 ppm-hrs. At W126 index levels greater than approximately 10 ppm-hrs, the proportion of sites showing foliar injury incidence is relatively constant. The air quality assessment discussed above identified Class I areas with recent air quality that met the current standard but were above a W126 index value of

15 ppm-hrs (Table 5-2). There were 22 Class I areas in this table and most of these areas had 3-year average W126 index values above 15 ppm-hrs for multiple 3-year periods. Given evidence of the potential occurrence of visible foliar injury at W126 index values of this magnitude, we note the ecosystem services that are at risk of impairment because of O₃-induced damage directly due to foliar injury, though data is not available to explicitly quantify these negative effects. Therefore, staff concludes that air quality levels that are at or below the level of the current standard may allow levels of visible foliar injury incidence to occur in areas of special significance to the public welfare.

In reaching conclusions regarding support for the adequacy of the current secondary standard provided by the currently available information on O₃-induced visible foliar injury we note that: 1) many species of native plants, including trees, have been observed to have visible foliar injury symptoms in both OTC and field settings, some of which have also been identified as bioindicators of O₃ exposure by the USFS; 2) visible foliar injury has been identified by the federal land managers as a diagnostic tool for informing conclusions regarding potential O₃ impacts on potentially sensitive AQRVs (USFS, NPS, FWS, 2010); 3) visible foliar injury incidence can occur for some species at very low cumulative exposures, but due to confounding by soil moisture and other factors, it difficult to predictively relate a given O₃ exposure to plant response; and 4) WREA analyses show that based on USFS biosite data, the proportion of biosites showing foliar injury incidence drops when W126 index values drop below approximately 10 ppm-hrs. However, we note that, with respect to visible foliar injury, we are unaware of any guidance for federal land managers regarding at what spatial scale or what degree of severity visible foliar injury might be sufficient to trigger protective action based on this potential impact on AQRVs. Further, there does not appear to be any consensus in the literature in this regard, and CASAC, while identifying target percent biomass loss and yield loss benchmarks for tree seedlings and commodity crops, respectively, did not provide a similar recommendation for this endpoint. Likewise, as in previous reviews, the ISA notes the difficulty in relating visible foliar injury symptoms to other vegetation effects such as individual plant growth, stand growth, or ecosystem characteristics (U.S. EPA, 2013, section 9.4.2, p. 9-39) and further noted that the full body of evidence indicates that there is wide variability in this endpoint, such that although evidence shows visible foliar injury can occur under very low cumulative O₃ concentrations, "...the degree and extent of visible foliar injury development varies from year to year and site to site..., even among co-members of a population exposed to similar O₃ levels, due to the influence of co-occurring environmental and genetic factors" (U.S. EPA 2013, section 9.4.2, p. 9-38).

Given the above, and taking note of CASAC views, we recognize foliar injury as an important O₃ effect which, depending somewhat on severity and spatial extent, may reasonably

be concluded to be of public welfare significance, especially when occurring in nationally protected areas. However, we also note the uncertainties associated with describing the potential for foliar injury and its severity or extent of occurrence for any given air quality scenario due to confounding by soil moisture levels and the difficulty in determining what degree of visible foliar injury incidence is likely to occur under different air quality conditions, and in particular on lands with special public welfare significance. We therefore conclude that the current standard may not adequately protect the public welfare from the known and anticipated adverse effects associated with O₃-induced impacts on visible foliar injury and associated services assessed in this review. Therefore, it may be appropriate to consider revising the standard to provide increased public welfare protection, though it is uncertain to what degree these O₃-induced impacts on visible foliar injury would be appropriately judged as important and adverse from a public welfare perspective.

The information for other welfare effects, including those with causal or likely causal relationships with O₃ (e.g., alteration of ecosystem water cycling, changes in climate), is limited with regard to our ability to consider potential impacts of air quality conditions associated with the current standard, although the WREA provides some perspective on this issue with regard to susceptibility to insect attack and fire regime change. We note, however, the importance of these effects categories to the public welfare.

As noted in section 1.3.2 above, our general approach to informing the Administrator's judgments recognizes that the available welfare effects evidence demonstrates a range of O₃ sensitivity across studied plant species and documents an array of O₃-induced effects that extend from lower to higher levels of biological organization. These effects range from those affecting cell processes and individual plant leaves to effects on the physiology of whole plants, species effects and effects on plant communities to effects on related ecosystem processes and services. Given this evidence, it is not possible to generalize across all studied species regarding which cumulative exposures are of greatest concern, as this can vary by situation due to differences in exposed species sensitivity, the importance of the observed or predicted O₃-induced effect, the role that the species plays in the ecosystem, the intended use of the affected species and its associated ecosystem and services, the presence of other co-occurring predisposing or mitigating factors, and associated uncertainties and limitations. At the same time, the evidence also demonstrates that though effects of concern can occur at very low exposures in sensitive species, at higher cumulative exposures those effects would likely occur at a greater magnitude and/or higher levels of biological organization and additional species would likely be impacted. It is important to note, however, due to the variability in the importance of the associated ecosystem services provided by different species at different exposures and in different locations, as well as differences in associated uncertainties and limitations, that, in addition to the magnitude of the

ambient concentrations, the species present and their public welfare significance are essential considerations in drawing conclusions regarding the significance of public welfare impact.

Therefore, in developing conclusions in this final PA, we note the complexity of judgments to be made by the Administrator regarding the adversity of known and anticipated effects to the public welfare and are mindful that the Administrator's ultimate judgments on the secondary standard will most appropriately reflect an interpretation of the available scientific evidence and exposure/risk information that neither overstates nor understates the strengths and limitations of that evidence and information.

Given all of the above, we reach the conclusion that the available evidence and exposure and risk information call into question the adequacy of public welfare protection provided by the current standard, and provides support for consideration of revisions to the current secondary standard to provide increased public welfare protection. More specifically, staff concludes that it is appropriate for the Administrator to consider revision of the current secondary O₃ standard to increase protection against O₃-attributable tree biomass loss, crop yield loss, and visible foliar injury, and their associated services, and particularly for those effects associated with cumulative, seasonal exposures that occur in Class I and similarly protected natural areas.

In reaching conclusions on options for the Administrator's consideration, we note that the final decision to retain or revise the current secondary O₃ standard is a public welfare policy judgment to be made by the Administrator, based on her judgment as to what degree of protection would be requisite (i.e., neither more nor less stringent than necessary) to protect the public welfare from any known or anticipated adverse effects. This final decision will draw upon the available scientific evidence for O₃-attributable welfare effects, and on quantitative analyses of vegetation and ecosystem exposures and associated risks to vegetation, ecosystems and their associated services, and judgments about the appropriate weight to place on the range of uncertainties inherent in the evidence and analyses. In making this decision, the Administrator will also need to weigh the importance of these effects and their associated ecosystem services in the overall context of public welfare protection.

Based on the considerations described in the sections above and summarized below, we therefore conclude that the currently available evidence and exposure/risk information call into question the adequacy of the public welfare protection provided by the current standard and provides support for considering potential alternative standards to achieve increased public welfare protection, especially for sensitive vegetation and ecosystems in federally protected Class I and similar areas. In this conclusion, we give particular weight to the evidence indicating the occurrence in Class I areas that meet the current standard of cumulative seasonal O₃ exposures associated with estimates of tree growth impacts of a magnitude that are reasonably considered important to public welfare.

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6 CONSIDERATION OF ALTERNATIVE SECONDARY STANDARDS

Chapter 5 reached the conclusion that the available evidence and exposure and risk information call into question the adequacy of public welfare protection provided by the current standard, and that it is appropriate for the Administrator to consider revising the current secondary standard to provide increased public welfare protection against O₃-attributable effects on tree biomass loss, crop yield loss, and visible foliar injury, and their associated services, particularly for those effects associated with cumulative, seasonal exposures, to the extent these effects are judged adverse to the public welfare. Given that conclusion, this chapter describes the staff evaluation of the available body of evidence, and exposure, risk and air quality information with regard to support for consideration of alternative standards, as articulated by the following overarching question:

- **What alternative secondary standards are supported by the currently available scientific evidence, exposure/risk information and air quality analyses?**

To assist us in interpreting the currently available scientific evidence and the results of recent quantitative exposure/risk analyses to address this question, we have focused on a series of more specific questions in sections 6.1, 6.2 and 6.3 below. We consider both the scientific and technical information available at the time of the last review and information newly available since the last review which has been critically analyzed and characterized in the ISA. Specifically, we consider the currently available scientific evidence and technical information in the context of decisions regarding the basic elements of the NAAQS: indicator (section 6.1); averaging time and form (section 6.2); and level (section 6.3). CASAC advice on potential alternative standards is described in section 6.4 and staff conclusions on potential alternative standards are discussed in section 6.5. Section 6.6 summarizes staff conclusions on the adequacy of the current standard and the alternative standards that are appropriate for the Administrator to consider. Key uncertainties in this review and areas in which future research and data collection would better inform the next review are identified in section 6.7.

6.1 INDICATOR

With regard to the selection of an appropriate indicator for alternative secondary standards, we consider the following question.

- **Does the information available in this review continue to support O₃ as the indicator for ambient air photochemical oxidants?**

In the last review of the air quality for O₃ and other photochemical oxidants and of the O₃ standard, as in other prior reviews, the EPA focused on a standard for O₃ as the most appropriate surrogate for ambient photochemical oxidants. Ozone is a long-established surrogate for ambient photochemical oxidants, among which it is by far the most widely studied with regard to effects on welfare and specifically on vegetation. The information available in this review adds to our understanding of the atmospheric chemistry for photochemical oxidants and O₃ in particular (as described in the ISA, sections 3.2 and 3.6, and summarized in section 2.2 in this document). The 1996 Staff Paper noted that the database on vegetation effects is generally considered to raise concern at levels found in the ambient air for O₃ and, therefore, control of ambient O₃ levels has previously been concluded to provide the best means of controlling other photochemical oxidants of potential welfare concern (U.S. EPA, 1996b, p. 277). In the current review, while the complex atmospheric chemistry in which O₃ plays a key role has been highlighted, no alternatives to O₃ have been advanced as being a more appropriate surrogate for ambient photochemical oxidants. Ozone continues to be the only photochemical oxidant other than nitrogen dioxide that is routinely monitored and for which a comprehensive database exists (U.S. EPA, 2013, section 3.6). Thus, staff concludes that O₃ remains the appropriate pollutant indicator for use in a secondary NAAQS that provides protection for public welfare from exposure to all photochemical oxidants.

6.2 FORM AND AVERAGING TIME

In considering potential forms and averaging times alternative to that of the current secondary standard (i.e., 4th highest daily maximum 8-hour average, averaged over 3 years), we address several specific questions.

- **To what extent does the currently available information provide support for considering forms different from that of the current secondary standard?**

In characterizing the current evidence, the ISA states that “[n]o recent information is available since 2006 that alters the basic conclusions put forth in the 2006 and 1996 O₃ AQCDs” with regard to biologically relevant exposure indices (U.S. EPA, 2013, section 2.6.6.1, p. 2-43). Based on the current state of knowledge and the best available data assessed in this review, the ISA therefore concludes that exposure indices that cumulate and differentially weight the higher hourly average concentrations over a season and also include the mid-level values continue to offer the most scientifically defensible approach for use in developing response functions and in defining indices for vegetation protection. Quantifying exposures with indices that cumulate hourly O₃ concentrations and preferentially weight the higher concentrations improves the

explanatory power of exposure/response models for growth and yield, over using indices based on mean and peak exposure values (U.S. EPA, 2013, section 2.6.6.1). These conclusions are based on the available body of evidence which provides a wealth of information, compiled over several decades, on the aspects of O₃ exposure that are most important in influencing plant response. As discussed in the ISA, the importance of the duration of the exposure and the relatively greater importance of higher concentrations (over lower concentrations) in determining plant response to O₃ have been well documented (U.S. EPA, 2013, section 9.5.3). Building on this research, other work has focused on developing “mathematical approaches for summarizing ambient air quality information in biologically meaningful forms for O₃ vegetation effects assessment purposes ...” (U.S. EPA, 2013, section 9.5.2, p. 9-99), including those known as cumulative, concentration weighted forms (i.e., SUM06, W126). Much of this work was completed by the mid-1990s, and was summarized in the 1996 Criteria Document (CD) (U.S. EPA, 1996a, section 5.5).

On the basis of this longstanding and extensive evidence demonstrating that the risk to vegetation comes from cumulative seasonal exposures, the EPA in the 1997 and 2008 reviews, as well as in the 2010 proposed rulemaking to reconsider the 2008 decision, recognized the importance of cumulative, seasonal exposures as a primary determinant of plant responses to O₃ in ambient air (61 FR 65741-42; 62 FR 38878; 72 FR 37893, 37896, 37900, 37904; 73 FR 16488-90, 16493-94; 75 FR 3000, 3010, 3012). For example, in the 1996 notice of proposed rulemaking, the Administrator recognized that the scientific evidence supported the conclusion that “a cumulative seasonal exposure index is more biologically relevant than a single event or mean index” (61 FR 65742). In the 2008 review, CASAC recognized that an important difference between the effects of short-term exposures to O₃ on human health and the effects of O₃ exposures on welfare is that “vegetation effects are more dependent on the cumulative exposure to, and uptake of, O₃ over the course of the entire growing season” (Henderson, 2006, p. 5). In that review, the CASAC O₃ Panel members were unanimous in supporting the final Staff Paper recommendation that “protection of managed agricultural crops and natural terrestrial ecosystems requires a secondary Ozone NAAQS that is substantially different from the primary ozone standard in averaging time, level and form” (Henderson, 2007, p. 3). Accordingly, in both the 1997 and 2008 reviews as well as the 2010 reconsideration, the Administrator proposed a secondary standard with a cumulative seasonal form as an appropriate policy option (61 FR 65742-44; 72 FR 37899-905; 75 FR 3012-3027).

In considering which exposure index was best suited for use as a form for the secondary O₃ NAAQS, the 1996 CD and 1996 Staff Paper evaluated a variety of different types of forms. These documents noted that a number of forms (e.g., the one event, mean and unweighted cumulative SUM00) are unable to reliably predict plant response because they either ignore the

role of duration or ignore the disproportionate impact of higher concentrations by weighting all concentrations equally (U.S. EPA, 1996b, p. 224). Other forms that were considered at that time included multicomponent forms which take into account many other relevant factors (e.g., plant growth stage, predisposition from earlier exposures). Of all the different exposure forms, these multicomponent forms consistently predict plant response best. However, due to being species-specific and highly complex, they were not considered suitable for more general application in the context of standard setting (U.S. EPA, 1996b, pp. 224-225). On the other hand, concentration-weighted forms that take into account the role of duration and concentration perform almost as well as the multicomponent forms. These forms include several threshold forms (e.g., SUM06, AOT60) and sigmoidally weighted cumulative indices (e.g., W126¹) (U.S. EPA, 1996a, pp. 5-84 to 5-136; U.S. EPA, 1996b, pp. 223-227). Given that these cumulative concentration-weighted forms were able to similarly predict plant response on the datasets for which they were evaluated (e.g., NCLAN), it was not possible to distinguish between them on this basis. Partly as a result, CASAC deliberations in 1995 did not produce a consensus on which cumulative concentration-weighted form would be best suited for a secondary NAAQS. As discussed further in 6.3 below, a workshop held in January of 1996 provided a consensus recommendation on the SUM06 form as appropriate for use in secondary standards, while also recognizing that a W126 form could also be appropriate (Heck and Cowling, 1997). Subsequent to this, the final 1996 Staff Paper and 1996 proposal notice both identified the SUM06 form as appropriate to consider and propose, respectively (U.S. EPA, 1996b, p. 285, 61 FR 65716). In selecting the SUM06 form that imposed a threshold despite the lack of scientific evidence for a discernible threshold for O₃-related vegetation effects across the range of studied species, the EPA noted that it had the benefit of not including concentrations that were considered at the time to be within the range of background, which was considered to be an important feature (U.S. EPA, 1996b, pp. 223-227).

In the subsequent review, the form of the standard was revisited in light of continued evidence that there remained a lack of discernible threshold for vegetation effects in general, and newer estimates of O₃ concentrations associated with background sources were lower than in the previous review such that their inclusion was less of a concern. On these bases, the 2007 Staff Paper recommended consideration of the W126 index as the basis for the form of a distinct secondary standard (U.S. EPA, 2006, pp. 9-11 to 9-15 and pp. AX9-159 to AX9-187; U.S. EPA, 2007, pp. 7-15/16). The EPA then proposed two options for the secondary standard, one of

¹ The W126 is a non-threshold approach described as the sigmoidally weighted sum of all hourly O₃ concentrations observed during a specified diurnal and seasonal exposure period, where each hourly O₃ concentration is given a weight that increases from 0 to 1 with increasing concentration (Lefohn et al, 1988; Lefohn and Runeckles, 1987; U.S. EPA, 2013, section 9.5.2).

which was to adopt a cumulative, seasonal standard based on the W126 index, while the other option was a secondary standard identical to the proposed revised primary standard (72 FR 37818). The CASAC Panel in that review expressed preference for the W126 index (Henderson, 2006). In deciding to reconsider the 2008 decision, the Administrator noted that past arguments or reasons for not moving to a cumulative, seasonal form, with appropriate exposure periods, were not based on disagreement over the biological relevance of the cumulative, seasonal form, or the recognized disadvantages of an 8-hour standard in measuring and identifying a specified cumulative, seasonal exposure pattern but were based on concerns over whether the EPA had an adequate basis to determine an appropriate level for a cumulative, seasonal secondary standard (75 FR 3019). Having reached the conclusion that such a level could be identified from within the range of levels proposed, the Agency proposed to set a secondary NAAQS in terms of a cumulative, seasonal standard form based on the W126 function (75 FR 2938). The CASAC also stated its support for this proposal, noting that it found the Agency's reasoning to be supported by the extensive scientific evidence considered in the last review (Samet, 2010).

In this review, we conclude that specific features associated with the W126 index still make it the most appropriate and biologically relevant cumulative concentration-weighted form for use in the context of the secondary O₃ NAAQS review. In particular, the W126 index does not apply an arbitrary exposure threshold below which concentrations are not included. Given the acknowledged variability in vegetation sensitivity, including evidence that some species are sensitive at very low cumulative exposures, and the continued lack of evidence of an exposure threshold for effects above a W126 index of zero, such a feature is scientifically justifiable and desirable. Thus, we conclude that the W126 form is best matched to the evidence associated with vegetation effects, as well as addressing the policy-relevant issue of how to weight exposures associated with background sources.

- **To what extent does the currently available information provide support for consideration of a cumulative seasonal form derived as a sum of weighted O₃ concentrations over daylight hours (8:00 am to 8:00 pm) and over the consecutive 3-month period having the highest sum within the O₃ season?**

As discussed in Chapter 5, mechanistic studies, including those recently assessed in this review, provide biological plausibility for the conclusions reached in the ISA that O₃-induced effects on plants are cumulative, that higher concentrations appear to be more important in eliciting a response than lower concentrations, and that plant sensitivity to O₃ can vary with time of day (U.S. EPA, 2013, p. 2-44). In particular, studies have shown that plants take up and respire gases through openings in their leaves, called stomata, which in general, are most open during daylight hours in order to allow sufficient CO₂ uptake for use in carbohydrate production

through the light-driven process of photosynthesis. Ozone, when present in sufficient amounts, is taken up along with the CO₂, where it and its derivatives can inhibit photosynthesis, leading to reduced carbohydrate production needed for growth, reproduction and repair (U.S. EPA, 2013, section 9.3.6; 75 FR 3013). Since plants are photosynthesizing during daylight hours and continue to grow throughout their growing season, the effects of repeated O₃ exposures continue to accumulate, both on a diurnal and seasonal basis. Thus, for vegetation, the element of “averaging time” has more appropriately been considered in terms of relevant exposure periods – diurnal and seasonal -- over which exposures are cumulated, or summed.

In the EPA’s consideration of such exposure periods in both the 1997 and 2008 reviews, and the 2010 reconsideration, the EPA identified the 12-hour daylight period from 8:00 am to 8:00 pm as appropriately capturing the diurnal window with most relevance to the photosynthetic process (61 FR 35716; 72 FR 37900; 75 FR 2938, 3013). In so doing, the EPA recognized, as did CASAC, that in some parts of the country this period may not include all daytime hours or all exposures of importance to vegetation, thus potentially underestimating the impact of O₃ at those sites (72 FR 37900-01; 75 FR 3013-14; Henderson, 2007, p. 3, pp. C-22-23). The evidence available in this review continues to provide support for focusing on the daylight hours, since for the majority of plants, the diurnal conditions of maximum O₃ uptake occur mainly during the daytime hours (U.S. EPA, 2013, section 9.5.3.2). This evidence shows that, in general, (1) plants have the highest stomatal conductance during the daytime; (2) atmospheric turbulent mixing is greatest during the day in many areas; and (3) the high temperature and high light conditions that occur during the day and that typically promote the formation of tropospheric O₃ also promote physiological activity in vegetation (U.S. EPA, 2013, section 9.5.3.2).

In addition, as in past reviews, we have also considered the evidence available from a number of studies that have reported O₃ uptake at night in some species (U.S. EPA, 2013, section 9.5.3.2). Typically the rate of stomatal conductance at night is much lower than during the day. Across the studies discussed in the ISA, nocturnal conductance ranged from negligible to 25% of daytime values (U.S. EPA, 2013, section 9.5.3.2), and, in some studies, varied by season and drought conditions. However, many of these studies did not link the night-time flux to measured effects on plants, making it difficult to know in those studies whether the impacts on the plant from nocturnal exposures are greater or less than those from similar daytime exposures, and whether or not they should be considered as separate impacts or as additive or synergistic with impacts from the preceding or subsequent daytime exposure.

Further, there are also uncertainties associated with the extent of the occurrence of high exposure to O₃ at night. For significant nocturnal stomatal flux and O₃ effects to occur, a susceptible plant with nocturnal stomatal conductance and low defenses must be growing in an

area with relatively high nighttime O₃ concentrations (often high elevation sites) and appreciable nocturnal atmospheric turbulence. It is unclear how many areas there are in the U.S. where these atmospheric conditions occur. It may be possible that these conditions exist in mountainous areas of southern California, front-range of Colorado and the Great Smoky Mountains of North Carolina and Tennessee. However, more information is needed in locations with high nighttime O₃ to assess the local O₃ patterns, micrometeorology and responses of potentially vulnerable plant species (U.S. EPA, 2013, section 9.5.3.2).

In consideration of the uncertainties that remain regarding the importance and extent of nocturnal exposures associated with plant uptake, and whether and how they might be incorporated into a national index, we conclude that it is appropriate to continue to focus on the 12-hour daylight exposure period of 8:00 am to 8:00 pm. We note that available monitoring data indicates that the daily increase in O₃ concentrations generally does not begin until after 8:00 am (U.S. EPA, 2013, section 3.6.3.2). In regard to this staff conclusion on an appropriate diurnal exposure period, CASAC states that “[a]ccumulation over the 08:00 a.m. – 08:00 p. m. daytime 12-hour period is a scientifically acceptable and recommended means of generalizing across latitudes and seasons” (Frey, 2014a, p. 13).

With regard to identification of the seasonal period over which to cumulate exposures, we note that a plant is vulnerable to O₃ pollution as long as it has foliage and is physiologically active (U.S. EPA, 2013, section 9.5.3, p. 9-112), i.e., during its growing season. The length of vegetative growing seasons varies depending on the type or species of vegetation and where it grows. For example, as discussed in the ISA, annual crops are typically grown for periods of two to three months while perennial species may be photosynthetically active longer, up to 12 months each year for some species (U.S. EPA 2013, section 9.5.3, p. 9-112). In general, the period of maximum physiological activity and thus potential O₃ uptake for vegetation coincides with some or all of the intra-annual period defined as the O₃ season, which can vary on a state-by-state basis (U.S. EPA, 2013, Figure 3-24, p. 3-83). This is because the high temperature and high light conditions, which can vary geographically, typically promote the formation of tropospheric O₃, as well as physiological activity in vegetation (U.S. EPA, 2013, section 9.5.3, p. 9-112).

The exposure periods used in studies of O₃ effects on vegetation reflect this understanding, with crop studies typically using shorter seasonal exposure periods and studies of longer lived trees and other perennial vegetation often extending for the entire annual growing season or in some cases over multiple growing seasons. Specifically, the ISA notes that “[m]ost of the crop studies done through NCLAN had exposures less than three months with an average of 77 days. Open-top chamber studies of tree seedlings, compiled by the EPA, had an average exposure of just over three months or 99 days. In more recent FACE experiments, SoyFACE

exposed soybeans for an average of approximately 120 days per year and the Aspen FACE experiment exposed trees to an average of approximately 145 days per year of elevated O₃, which included the entire growing season at those particular sites” (U.S. EPA, 2013, section 9.5.3.2, p. 9-112). Further, the U.S. Forest Service and federal land managers have typically used the 6 months from April through September as the accumulation period (U.S. EPA, 2013, section 9.5.3.2, p. 9-112). However, despite the possibility that plants may be exposed to ambient O₃ longer than 3 months in some locations, the ISA notes that “[t]he exposure period in the vast majority of O₃ exposure studies conducted in the U.S. has been much shorter than 6 months...” and “there is generally a lack of exposure experiments conducted for longer than 3 months” (U.S. EPA, 2013, section 9.5.3.2, p. 9-112). As a result, analyses of effects in terms of the W126 exposure index have typically defined the index in terms of a 3-month exposure period or at least in terms of periods shorter than 6 months (e.g., SoyFACE, Aspen FACE) (U.S. EPA, 2013, p. 9-112).

In the current review, the EPA conducted a new analysis to further inform the consideration of the most appropriate seasonal accumulation period (U.S. EPA, 2013, section 9.5.3). This analysis calculated and compared the 3- and 6-month maximum W126 index values for over 1,200 AQS and CASTNET EPA monitoring sites for the years 2008-2009. The two accumulation periods were found to be highly correlated metrics (U.S. EPA, 2013, Figure 9-13; section 9.5.3). The analysis indicates that in the U.S., W126 cumulated over 3 months and W126 cumulated over 6 months could be proxies of one another, as long as the period in which daily W126 is accumulated corresponds to the seasonal maximum. Therefore, it is expected that either statistic will predict vegetation response equally well. Given the above information, and in particular the results of the EPA analysis showing the maximum 3-month period is highly correlated with the longer 6-month maximum period, we again conclude that it is appropriate to continue to focus on the consecutive 3-month period with the highest cumulative exposure value within the monitored O₃ season as the seasonal exposure period with most relevance to vegetation. Given its review of the available science, CASAC also expressed support for this seasonal period, stating that “[t]he Second Draft PA makes a very strong case, consistent with previous CASAC judgment, for changing the secondary metric to the W126 averaged over the highest three-month interval” (Frey, 2014a, p. 13).

6.3 LEVEL

In considering potential levels for alternative secondary standards, we again find it useful to note that the protection provided by the secondary standard derives from the combination of all elements of the standard (indicator, form, averaging time, and level). Thus, in light of the discussions in section 6.2 above, we first consider what level or range of levels can reasonably be

judged to provide a requisite degree of public welfare protection when combined with a W126 index form of cumulatively weighted concentrations from 8:00 am to 8:00 pm over a maximum consecutive 3-month period. In addition to considering the information in the context of a single growing season, we also consider it in the context of a form for this W126 metric averaged across three consecutive growing seasons for reasons discussed in section 6.2 above.

In the discussion below, we turn first to consideration of the currently available scientific evidence as assessed and characterized in the ISA. We then consider the WREA findings with regard to vegetation, ecosystem effects and services estimated for different air quality scenarios. We additionally take note of important uncertainties and limitations in the evidence and exposure/risk analyses, as well as considerations related to interpreting these impacts in light of the additional policy considerations described in the adversity paradigm. Lastly, we take note of judgments to be made by the Administrator in drawing conclusions regarding effects and risks that represent adverse effects to public welfare. In so doing, we identify key considerations with regard to the currently available evidence, exposure/risk information and associated uncertainties in identifying the range of levels that may be appropriate to consider for a cumulative seasonal secondary standard. Such levels are described in section 6.5 below, which describes staff conclusions regarding alternative secondary standards appropriate to consider in this review.

- **What does the currently available evidence indicate with regard to the range of W126-based index values that may provide protection from vegetation effects of O₃?**

In answering this question, we first consider quantitative evidence for O₃ exposure effects on plant growth, productivity and related endpoints. In so doing, we draw primarily on the robust E-R functions developed in OTC studies for tree seedling and crop species as described in the ISA (U.S. EPA 2013, section 9.6), and as used in the WREA exposure and risk analyses (U.S. EPA, 2014b, section 6.2), and discussed in Chapter 5 of this document (Figures 5-1 and 5-4). It is important to note that these functions are used to provide estimates of growth and yield reduction in tree seedlings and crops that might be expected to result from exposure over a single growing season to various O₃ concentrations expressed in terms of a W126 index. We also consider the available evidence and exposure/risk information for visible foliar injury.

As a point of clarification, we note that CASAC commented that it “concur[s] that relative biomass loss for tree species, crop yield loss, and visible foliar injury are appropriate surrogates of a wide range of damage that is adverse to public welfare.” (Frey, 2014a, p. 10). While we agree that effects at the individual tree, crop, or other plant species level, in and of themselves, can be directly related to effects on the public welfare when they occur to a sufficient degree on lands with an intended use that can be affected by O₃-induced vegetation effects (e.g., timber

production, AQRVs in Class I areas), we also caution that not all predicted effects on vegetation occur to such a degree or occur on lands within this category. Thus, in considering the predicted effects on studied tree and crop species under various W126 exposures, we are mindful of the need to further determine under what conditions they can be considered surrogates for impacts that are important in the public welfare policy context.

Table 6-1 below presents estimates of relative biomass and yield loss for the 11 and 10 studied species of tree seedlings and crops, respectively, for which we have robust E-R functions developed in OTC studies, for a single growing season exposure to a number of W126 index values. In this table, we have included observations related to median and individual species relative biomass loss in tree seedlings and relative crop yield loss, at the target benchmark levels of 2% and 5%, respectively. These benchmarks are consistent with the 2% and 5% benchmarks for tree seedlings and crops, respectively, as advised by CASAC in this review (Frey, 2014a; section 6.4 below), and with values given focus in the 1996 expert consensus workshop. We have also included information on the number of studied species with estimates below other benchmarks that may also be of interest (i.e., 5%, 10%, and 15% for trees and 10% for crops). CASAC has placed most emphasis on the median species response in recommending a range of scientifically supportable levels. For example, CASAC noted that “[i]n our scientific judgment, it is appropriate to identify a range of levels of alternative W126-based standards that includes levels that aim for not greater than 2% RBL for the median tree species” (Frey, 2014a, p. 14).” CASAC also recognizes that as a policy matter the Administrator may find it useful to also consider information related to individual species responses, to the degree that they have special significance to the public welfare, when selecting an appropriate level or range of levels. Specifically, CASAC states that “[a]s a policy recommendation, separate from its advice above

Table 6-1. Tree seedling biomass loss and crop yield loss estimated for O₃ exposure over a season.

W126 value for exposure period	Tree seedling biomass loss ^A		Crop yield loss ^C	
	Median Value	Individual Species	Median Value	Individual Species
21 ppm-hrs	Median species w. 6.8% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 7.7 % loss ^D	≤ 5% loss: 4/10 species >5, <10% loss: 3/10 species >10, <20% loss: 3/10 species
19 ppm-hrs	Median species w. 6.0% loss ^B	≤ 2% loss: 3/11 species <5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 6.4 % loss ^D	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10, <20% loss: 2/10 species
17 ppm-hrs	Median species w. 5.3% loss ^B	≤ 2% loss: 5/11 species <5% loss: 5/11 species ≤ 10% loss: 9/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 5.1 % loss ^D	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10, <20% loss: 2/10 species
15 ppm-hrs	Median species w. 4.5% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 6/11 species ≤ 10% loss: 10/11 species >30% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species
13 ppm-hrs	Median species w. 3.8% loss ^B	≤ 2% loss: 5/11 species <5% loss: 7/11 species <10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species
11 ppm-hrs	Median species w. 3.1% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 8/11 species ≤ 10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 9/10 species >5, <10% loss: 1/10 species
9 ppm-hrs	Median species w. 2.4% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: all species
7 ppm-hrs	Median species w. ≤2% loss ^B	≤ 2% loss: 7/11 species ≤ 5% loss: 10/11 species >15% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: all species

A Estimates here are based on the 11 E-R functions for tree seedlings described in WREA, Appendix 6F and discussed in section 5.2.1, with the exclusion of cottonwood. See CASAC comments (Frey, 2014a).

B This median value is the median of the composite E-R functions for 11 tree species in the WREA, Appendix 6F (also discussed in section 5.2.1).

C Estimates here are based on the 10 E-R functions for crops described in Appendix 6F and discussed in section 5.3.1.

D This median value is the median of the composite E-R functions for 10 crops from WREA, Appendix 6F (also discussed in section 5.3.1).

regarding scientific findings, the CASAC advises that a level of 15 ppm-hrs for the highest 3-month sum in a single year is requisite to protect crop yield loss, but that lower levels provide additional protection against crop yield loss. Furthermore, there are specific economically significant crops, such as soybeans, that may not be protected at 15 ppm-hrs but would be protected at lower levels” (Frey, 2014a, p. iii).

From Table 6-1, we see that median tree species biomass loss is at or below 2% only at the lowest W126 index value assessed, 7 ppm-hrs. As the W126 index value is incrementally increased, median RBL also increases incrementally, so that at W126 index values of 9, 11, 13, 15, and 17, the median RBL increases to 2.4%, 3.1%, 3.8%, 4.5%, and 5.3%, respectively. Thus over the W126 range of 7 to 17 ppm-hrs, median species biomass loss ranges from approximately 2% to approximately 5%.

We also believe it is informative to examine the individual species responses and RBL over the same W126 range (7 to 17 ppm-hrs). We first note, based on Figure 5-1 (B) in chapter 5, that over this range, five species maintain RBLs of less than 2%. These more tolerant species include Douglas fir, loblolly pine, Virginia pine, sugar maple and red maple. Thus, little additional protection would be achieved for these species below the W126 index value of 17 ppm-hrs. Two of these species (red maple and sugar maple) would only exceed 2% RBL at 21 ppm-hrs. In contrast, black cherry, the most sensitive of the remaining six species, has RBL ranging from approximately 36% at 17 ppm-hrs down to approximately 17% at 7 ppm-hrs. Thus, given that the magnitude of predicted black cherry RBL could be judged adverse over this range, it is not clear to what extent this information informs the selection of an appropriate level (Table 6-1; U.S. EPA, 2014b, section 6.2, Appendix 6A), though clearly protection would be expected to be greater at lower W126 index values. We further note that CASAC based their recommendation of an appropriate W126 level by considering the median tree species RBL of no more than 2%, but some levels within CASAC’s recommended range allow for the possibility for individual species RBL to go much higher.

Because Table 6-1 was updated in this final PA to deemphasize cottonwood, based on staff’s understanding of CASAC advice in that regard, we note that the CASAC advice based on the numbers of species protected to no more than 2% RBL and median RBL values for tree seedlings associated with various W126 levels, as shown in the in the second draft PA table (U.S. EPA, 2014a), is no longer consistent in some cases with the revised Table 6-1. For example, in commenting on the version of Table 6-1 in the second draft PA CASAC states that “[t]able 6-1 presents the RBL results for individual species for different levels of W126. This table demonstrates that a range of 7 ppm-hrs to 15 ppm-hrs will protect against RBL of 2% for at

least 5 of the 12 species”² (Frey, 2014a, p. 14). In addition, CASAC states that “[t]he CASAC does not support a level higher than 15 ppm-hrs. For example, at 17 ppm-hrs, the median tree species has 6% relative biomass loss.... These levels are unacceptably high” (Frey, 2014a, p. iii). While we continue to place weight on CASAC’s scientific judgments that a 6% median RBL is unacceptably high, and that the 2% median RBL is an important benchmark to consider, we also note that the updated median RBL for a W126 level of 17 ppm-hrs is now 5.3%.

We further note that CASAC does not provide additional clarification regarding its views on the acceptability of median tree species RBL levels between 2% and 6%, beyond noting that values closer to the lower end of the range (W126 index value of 7 ppm-hrs) would provide greater protection for more sensitive tree species, and that the levels within CASAC’s recommended range allow for the possibility for individual species RBL to go much higher than 2% and 6%. Given the nature of this input, we then considered the RBL information available for the remaining five species (i.e., eastern white pine, aspen, tulip poplar, ponderosa pine, red alder) to further inform our evaluation of the additional protection that potentially could be achieved at different W126 levels within the range being considered. We thus note that at the W126 index value of 17 ppm-hrs, one species (eastern white pine) has RBL above 10% and one species (red alder) has RBL of 5.3% (below 6%) while the other three fall between approximately 6.7% and 9.8%. At the W126 index value of 15 ppm-hrs, two (i.e., red alder and ponderosa pine) of the five species fall below 6% RBL, while the remaining 3 species have RBLs that range from 7.4% to 8.8%. At the W126 index value of 13 ppm-hrs, three species (i.e., tulip poplar, ponderosa pine, red alder) fall below 6% RBL, while the remaining two have RBLs of 7.0% and 7.1%. At the W126 index value of 11 ppm-hrs, all five species have RBLs below 6%. Taken together with the more tolerant species, the proportion of the studied tree species with RBLs below 6% are 6/11, 7/11, 8/11, and 10/11 at W126 index values of 17, 15, 13, and 11 ppm-hrs, respectively. To the extent the focus is placed on different % RBL benchmarks and the proportion of studied trees protected at those levels, as well the expected impacts to associated ecosystem services, in regard to the identification of the appropriate level or range of levels, this information may be appropriate to consider.

With respect to crops, based on the 10 robust E-R functions (i.e., barley, lettuce, field corn, grain sorghum, peanut, winter wheat, cotton, soybean, potato and kidney bean) described in the ISA and additionally analyzed in the WREA (Figure 5-4), Table 6-1 shows that for the CASAC recommended target benchmark protection level of 5% for median crop relative yield loss (RYL), W126 index values ranging from 7 to 17 ppm-hrs are protective. However, when

² We note that the updated table shows that a range of 7 ppm-hrs to 17 ppm-hrs will protect against RBL of 2% for at least 5 of the 11 species.

individual species are considered over this same range, the proportion of crops protected varies from 5/10, 6/10, 7/10, 9/10, 10/10, and 10/10 at the W126 levels of 17, 15, 13, 11, 9, and 7 ppm-hrs. To the extent a given species is judged as having particular importance to the public welfare, breaking the information down by species can be helpful. For example, less than 5% yield loss was estimated for soybeans at the W126 index value of 12 ppm-hrs (U.S. EPA 2014, Figure 6-3). Four of the studied crop species (barley, lettuce, field corn, and grain sorghum) are more tolerant, with RYL under 1% over the W126 range from 7 to 17 ppm-hrs. Peanut also remained under 4% RYL over the same W126 range. Other species differed regarding the W126 level at which RYL reached or fell below 5%. Specifically, for winter wheat, cotton, soybean, kidney bean and potato, the relevant W126 index values at which RYLs were below 5% are 15, 13, 11, 11, and 9 ppm-hrs. As noted in Chapter 5, the significance of these predicted RYLs to the public welfare could be informed by the recognition that crops are heavily managed to obtain the desired yield, and the extent to which yield reductions in any specific crop in a particular location are considered adverse to public welfare could depend on a number of economic factors, including crop prices, crop substitution, and the welfare importance of relative changes in consumer and producer surplus.

With respect to considering visible foliar injury, and its ability to inform selection of an appropriate target level or range of levels for public welfare protection, we first recognize its value as a long-standing and well-established bioindicator of O₃ exposure, as described in the ISA (U.S. EPA 2013, section 9.4.2). In addition to the role of visible foliar injury as an indicator, we note that the aesthetic aspects of visible foliar injury itself have the potential to be important to public welfare (as described in section 5.4). CASAC also “concur[s] that visible foliar injury can impact public welfare by damaging or impairing the intended use or service of a resource. Visible foliar injury that is adverse to public welfare can include: visible damage to ornamental or leafy crops that affects their economic value, yield, or usability; visible damage to plants with special cultural significance; and visible damage to species occurring in natural settings valued for scenic beauty or recreational appeal” (Frey, 2014a, p. 10). In this regard, we first note that several tribes have identified a number of O₃ sensitive species that are important to their cultural practices (Appendix 5A). These species have many cultural uses such as food, medicines, dyes, tools/textiles, spiritual, and commercial. In addition, visible foliar injury has been identified by the federal land managers (FLMs) as a diagnostic tool for informing conclusions regarding potential ozone impacts on potentially sensitive AQRVs (USFS, NPS, FWS, 2010), and the evidence shows that injury has been documented in such areas under recent air quality conditions.

Despite its recognized importance to the public welfare as a general matter, we are unaware of any injury benchmarks or criteria that have been identified by the FLMs as to what extent and/or severity of observed foliar injury warrants protection efforts. In considering CASAC comments in this regard we note that while it states that “[a] level below 10 ppm-hrs is required to reduce foliar injury” (Frey, 2014a, p. iii), CASAC does not provide any additional information regarding the public welfare significance of this degree of injury or what an appropriate target benchmark or range of benchmarks would be for foliar injury in relation to what could be considered adverse to the public welfare. Given that there is substantial variability in this endpoint, such that “the degree and extent of visible foliar injury development varies from year to year and site to site ... even among co-members of a population exposed to similar O₃ levels, due to the influence of co-occurring environmental and genetic factors” (U.S. EPA, 2013, p. 9-38), staff recognizes the lack of a consistent or generally predictable relationship between particular W126 exposures and visible foliar injury incidence. We additionally note uncertainty in what can be concluded from foliar injury in relation to plant health, productivity and ecological function as “it is not presently possible to determine, with consistency across species and environments, what degree of injury at the leaf level has significance to the vigor of the whole plant” (U.S. EPA, 2013, p. 9-39). However, we do recognize the Congressional mandate, provided in the CAA amendments of 1977 that establish additional protections for Class I areas. The 1997 Consensus Workshop Report (Heck and Cowling, 1997) discussed below, noted the potential for visible foliar injury to occur at very low levels. CASAC also stated that “[v]isible foliar injury is even more sensitive than RBL of 2%, with W126 index values below 10 ppm-hrs required to reduce the number of sites showing visible foliar symptoms” (Frey, 2014, p. 14). We further note that the information discussed here regarding incidence of visible foliar injury does not include information regarding the severity of the observed symptoms and the degree to which the public welfare impacts from different severity benchmarks might vary. Thus, there is additional uncertainty regarding the potential variability in the severity of the symptoms across species and locations and to what degree this would affect the public welfare significance of these effects so that the appropriate range of W126 index values to protect against this effect is difficult and complicated to identify.

In further considering the available information pertaining to the question above, we additionally recognize conclusions that have been drawn by expert committees with regard to these endpoints (i.e., tree seedling growth, crop yields and visible foliar injury). For example, in their review of staff documents during the O₃ NAAQS review completed in 1997, the CASAC O₃ panel members expressed a wide range of opinions on aspects of the evidence important to consider in judging the adequacy of the O₃ secondary standard and in considering the form and level that would be appropriate for a secondary O₃ standard (Wolff, 1996). Subsequent to

CASAC meetings in 1995 on this topic, a consensus-building workshop sponsored by the Southern Oxidant Study group was held on the topic of the O₃ secondary standard in January 1996 (Heck and Cowling, 1997). This workshop was attended by 16 scientists with backgrounds in agriculture, managed forest, natural systems, and air quality, all of whom were leaders in their fields and whose research formed the basis of much of the research examined in the 1996 Criteria Document. These scientists expressed their judgments on what standard level(s) would provide vegetation with protection from O₃-related adverse effects that would be adequate, in their view.^{3,4} As the 1997 workshop publication indicates, the scientists at the 1996 workshop also reached consensus views regarding the types of exposures that were important in eliciting plant response and the types of metrics that were best at predicting these responses (Heck and Cowling, 1997). Before coming to agreement on daily and seasonal durations and forms pertinent to a distinct secondary standard, the participants discussed and identified endpoints to consider for natural, forest and agricultural ecosystems.⁵ With regard to form of the standard, participants concurred with either the SUM06 or W126 metrics, with consensus finally reached for SUM06, with some qualification regarding implications for a threshold. The participants identified the ranges they felt should be considered for each of three endpoints. Overall, the SUM06 values ranged from 8 to 20 ppm-hrs corresponding to W126 index values ranging from 5 to 17 ppm-hrs, based on the EPA analysis focused on conditions in NCLAN studies.⁶ This overall range reflected ranges for each of the three endpoints, with the following considerations (Heck and Cowling, 1997).

- Crops (yield reductions): SUM06 of 15-20 ppm-hrs (13 to 17 ppm-hrs, W126). This range was recognized to generally consider <10% yield loss in more than 75% of species.
- Trees (growth effects): SUM06 of 10-16 ppm-hrs (7 to 14 ppm-hrs, W126). This range was recognized to generally consider 1-2% per year growth reduction; in so

³ At the time of the workshop, the secondary O₃ standard being reviewed by EPA was a 1-hour average of 0.12 ppm (identical to the primary standard at that time). In 1997, EPA concluded the review by revising both standards to a longer averaging time of 8 hours with a level of 0.08 ppm (62 FR 38856).

⁴ The workshop publication describes the primary objective for the workshop as having been to assemble knowledgeable scientists to develop a group consensus on “various critical components associated with a possible revised secondary ozone standard” (Heck and Cowling, 1997).

⁵ For natural ecosystems, they focused on foliar injury as an indicator. For forest ecosystems, they concluded the data did not support selection of an indicator of effects on forest structure or function. As a result, they identified two indicators pertinent to the systems: growth effects on seedlings from species of natural forest stands (1-2% per year reduction), and growth effects on seedlings and saplings from tree plantations (1-2% per year reduction). For agricultural systems, the participants focused on protection against crop yield reductions, with their acknowledgment of high uncertainties at 5% leading them to a crop yield endpoint of 10% yield reduction (Heck and Cowling, 1997).

⁶ During the last review, W126 index values corresponding to the SUM06 values cited in the report were estimated using the NCLAN crop loss data, a key dataset considered by workshop participants (see Appendix 7B of 2007 Staff Paper; Appendix 6A of this document).

doing, the group identified a need to consider the potential for year-to-year compounding of impacts in long-lived perennial species.

- Visible Foliar Injury: SUM06 of 8 to 12 ppm-hrs (5 to 9 ppm-hrs, W126).

Since the publication of 1996 workshop report and conclusion of the 1997 NAAQS review, the evidence base has continued to expand as described in the 2006 CD and ISA (U.S. EPA, 2006; U.S. EPA, 2013). With regard to tree growth effects and crop yield reductions, results of additional studies conducted in the field have confirmed the tree seedling biomass loss and crop yield loss E-R relationships derived from earlier studies that used OTC (U.S. EPA 2013, section 9.6).

In the 2008 review, CASAC provided comments related to a cumulative seasonal secondary standard in the context of their comments on the draft and final Staff Papers and on the final decision (Henderson, 2006; Henderson, 2007; Henderson, 2008). In all instances, they conveyed support for establishment of a distinct secondary standard with a cumulative seasonal form. While the EPA, in the 2007 Staff Paper and 2007 notice of proposed rulemaking, recognized a broader range of W126 index values as appropriate for consideration with regard to a distinct secondary standard, the CASAC Panel focused on a range they described as approximately equivalent to that identified by the 1996 workshop participants (Henderson, 2007, pp. 3, C-27).⁷ In the CASAC Panel 2006-2007 advice on levels for such a standard, their suggestion was a focus on levels for a W126 index approximately equivalent to a SUM06 range of 10 to 20 ppm-hrs (Henderson, 2006, 2007, 2008), which they estimated in 2007 to be a range from 7 (or 7.5) to 15 ppm-hrs. Based on their consideration of the information available in that review (with regard to potential magnitude of effects across multiple years), the CASAC Panel further advised that “[i]f multi-year averaging is employed to increase the stability of the secondary standard, the level of the standard should be revised downward to assure that the desired threshold is not exceeded in individual years” (Henderson, 2007, p. 3). The CASAC advice provided on the 2010 proposed reconsideration and in this review is summarized in section 6.4 below.

In considering the evidence briefly summarized above in the context of levels for a W126-based standard, we recognize that given the different types of O₃-induced effects, genetic variability within and between species, and environmental modifiers of effects that also contribute to variability, it is not feasible to identify a range of cumulative seasonal exposures from the vegetation effects evidence which would provide a consistent degree of protection for

⁷ Appendix C of the March 26, 2007 CASAC letter (Henderson, 2007) used a 2001 ambient concentration dataset and other factors, rather than study data considered in the 1996 workshop, in estimating an “equivalency” between the two indices.

all species. Thus, in our consideration of the evidence, we note the importance of considering several dimensions that pertain to judgments regarding public welfare significance. For example, we take note of the usefulness of considering the cumulative seasonal exposure at which the median species response or the majority of the species' responses are expected to be below minimal response benchmarks of interest and at which only a very few species' responses are expected to exceed more substantial response benchmarks. Before articulating such considerations with regard to specific benchmarks and index values, we first consider the WREA findings in the context of the following question.

- **What are the nature and magnitude of risks to vegetation estimated for the average W126 index scenarios evaluated in the WREA, and what is the magnitude of risk reduction from risks estimated for air quality conditions estimated for the current standard?**

The WREA provides a characterization of ambient O₃ exposure and its relationship to ecological effects, and estimates of the resulting impacts to several ecosystem services. In considering the question posed above, we focus particularly on WREA estimates related to O₃ effects on plant biomass and associated ecosystem services effects. The WREA analyses provide information on the geographical extent of the effects of O₃ exposure on plant biomass for different air quality scenarios. We also note the relationships among effects on individual plants to other ecosystem components and functions, such as carbon sequestration and air pollutant removal (U.S. EPA 2013, section 9.4.3.4; U.S. EPA, 2014b, sections 6.6 and 6.7), as well as market responses to changes in timber and agricultural production (U.S. EPA, 2014b, sections 6.3 and 6.5). We additionally recognize the potential for O₃ to impact other biomass-related responses, such as the supply of non-timber forest products and other ecosystem responses for which we have primarily qualitative characterizations of impacts (U.S. EPA, 2014b, chapter 5).

We turn first to the WREA estimates for a range of effects related to biomass loss, which are based on application of the robust E-R functions for seedlings of 11 tree species described in the ISA (U.S. EPA, 2013, section 9.6.2) and the WREA (U.S. EPA, 2014b, Appendix 6F)⁸. First, we note (as considered above) the range of responses for the individual species for which robust E-R functions have been developed. These eleven species vary appreciably in sensitivity of growth reduction (in terms of relative biomass loss, or RBL) in response to O₃ exposure.

⁸ There is an E-R function available for a 12th tree species (cottonwood), but this E-R function is considered less robust because it is based on the results of a single gradient study (Gregg et al., 2003). That combined with its apparent extreme response to O₃ prompted CASAC to advise the Administrator to not place too much emphasis on cottonwood in the review of the secondary standard (Frey, 2014, p. 10). As a result, while we do include cottonwood in some of the analyses, we have decided it would not be appropriate to put less weight on the cottonwood biomass loss estimates when considering what levels of W126 should be considered protective of median species biomass loss.

Based on the 11 individual tree species with robust seedling E-R functions, six of the 11 species show 2% seedling biomass loss at a W126 index value below 8 ppm-hrs and in the other five species at a W126 index value above 18 ppm-hrs. Within the group of six more sensitive species, the most sensitive is black cherry (see Figure 5-1B).

In Appendix 6F, the WREA presents individual and median response across the studied tree and crop species (U.S. EPA 2014, Appendix 6F). This appendix includes an analysis of the median of the composite exposure-response (E-R) functions for tree seedlings and crops. Specifically, Tables 6F-1 and 6F-2 provide estimates of the relative loss for trees and crops respectively at various W126 index values using the composite E-R functions for each species. The median of the composite functions is calculated for all 12 tree species as well as for the 11 tree species excluding cottonwood. The median of the composite functions for all 12 tree species and all 10 crop species is consistent with the green line shown in Figures 6-5 and 6-6 (U.S. EPA 2014, section 6.2.1.2. Tables 6F-3 and 6F-4 provide estimates of the number of species for trees and crops respectively that would be below various benchmarks (e.g., 2% biomass loss for trees) at various W126 index values. Based on the median composite E-R function developed for the 11 tree species depicted in WREA Table 6F-1, median tree species biomass loss ranges from less than 1.5% to 5.3% over the W126 index value range of 7 to 17 ppm-hrs (U.S. EPA, 2014b, Appendix 6F).

We additionally consider the WREA estimates of overall ecosystem-level effects from biomass loss considering the studied species together (U.S. EPA 2014, section 6.8). The WREA analysis used the species-specific biomass loss E-R functions, information on prevalence of the studied species across the U.S., and a weighting approach based on proportion of the basal area within each grid cell that each species contributes. The WREA analyses use information from the individual and median E-R functions for tree seedlings to provide information on the geographical extent of the effects of O₃ exposure on growth reduction for different air quality scenarios. It provides information on the location and number of species affected, as well as information about the estimated effects in Class I areas. We note that some of these analyses continue to include cottonwood and where this is the case, it is so noted. In the WREA analyses, the largest reductions in O₃ concentrations occur when air quality is adjusted from recent conditions to meeting the current standard. Smaller changes in O₃ concentrations occur when air quality is adjusted for the W126 air quality scenarios for 15, 11, and 7 ppm-hrs (average across three years), relative to meeting the current standard.

A weighted RBL value for each grid cell is generated by weighting the RBL value for each studied tree species found within that grid cell by the proportion of basal area it contributes to the total basal area of all (unstudied and studied) tree species within the grid cell, and then

summing those individual weighted RBLs. Table 6-2 below describes the percent of assessed geographic area with RBL exceeding 2% for 11 species based on the average W126 index values estimated for five air quality scenarios. Under recent conditions, 7.6 % of the total geographic area has a wRBL above 2% while just meeting the current standard across the contiguous U.S., the WREA estimates 0.2% of the total geographic area to have a weighted relative biomass loss above 2% for the 7 ppm-hrs scenario (Table 6-2 below; U.S. EPA 2014, Table 6-25). In the W126 air quality scenarios for 15, 11, and 7 ppm-hrs (average across three years), the percent of total area having weighted relative biomass loss greater than two percent was 0.2 percent, 0.1 percent and <0.1 percent, respectively (Table 6-2 below; U.S. EPA 2014, Table 6-25). In considering these estimates, however, we note that the values for percentages of basal area include many grid cells in which none of the 11 studied species are found, and thus these values are likely to be low. In addition, the ecosystem level impacts from O₃-induced effects on biomass loss in each grid cell would also depend on the interaction between the studied species with known O₃-sensitivities and the other species that are also contributing to the total basal area which have unstudied O₃-sensitivities. Given these and other potential uncertainties and limitations associated with this analysis (U.S. EPA, 2014b, section 6.8), which were also commented on by CASAC (Frey, 2014a, p. A-40), we thus conclude that this analysis does little to inform the nature and degree of risk likely to be experienced by O₃-sensitive species growing in mixed-species forests, which are wide-spread in the eastern U.S. These values may be more appropriate for western forests which more often are composed of a single species (i.e., ponderosa pine, aspen forests).

Table 6-2. Percent of assessed geographic area exceeding 2% weighted relative biomass loss in WREA air quality scenarios.

Percent of total area with wRBL>2%	-----Air Quality Scenarios -----				
	Recent Conditions (2006-2008)	Conditions just meeting the current standard ^A	W126 index scenarios ^B		
			15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
Using all 12 Species	10.8%	0.8 %	0.7 %	0.5 %	0.2 %
Using 11 species (excluding cottonwood)	7.6%	0.2%	0.2%	0.1%	<0.1%

^A This analysis uses air quality values that are estimated per model grid cell using the W126 index value assigned to the grid cell based on application of the VNA method to the monitor-location W126 index values that are the average at that location across the 3 years of W126 index values for the adjusted dataset that just meets the current standard (4th highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb).

^B The national distribution of W126 index values within model grid-cells for each scenario reflects model-based adjustment of 2006-2008 O₃ concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA, 2014b, section 4.3.4.1 and Appendix 4A).

To further inform this issue, the WREA characterized the number of counties where the median RBLs were above 2% (U.S. EPA, 2014b, Table 6-7), as shown in Table 5-5. Given CASAC's advice to put less emphasis on cottonwood, we focus on the rows of this table that excluded cottonwood. Under recent conditions, 52% of the counties have median RBLs above 2%. When air quality is adjusted to the current standard, that proportion drops to 8% and further decreases to 6% for air quality adjusted to just meet a 3-year average W126 level of 7 ppm-hrs. With respect to median RBL values, of the 239 counties (8% of counties) estimated to have a median RBL above 2% when meeting the current standard, 203 of those counties have a RBL above 2% because of the presence of black cherry. Thus, as also discussed above in Section 6.2, given the magnitude of estimated RBL for black cherry over the entire range assessed, it is not clear to what extent the information for black cherry informs the selection of an appropriate level.

In addition, the WREA also characterized the number of counties where one or more individual studied tree species showed a 2% biomass loss (U.S. EPA, 2014b, Table 6-7), as also shown in Table 5-5. This is consistent with CASAC advice that "rather than focusing solely on the median relative biomass loss (RBL), the number of counties containing sensitive tree species that are expected to have growth loss of greater than 2% should be quantified" (Frey, 2014a, p. 11). The maximum number of species that exceed 2% RBL in any one county is five species, which only occurs under recent O₃ conditions. After meeting the current standard, the maximum number of species in any one county is four. This information shows that a number of counties have more than one O₃-sensitive species growing in it, potentially together in the same forest stands, whose RBLs are above 2%. Given CASAC's advice to put less emphasis on cottonwood, we focus on the rows of this table that excluded cottonwood. Under recent air quality conditions, the proportion of counties with 1 or more species with an RBL greater than 2% is 78% (2,418 counties). As air quality is adjusted to just meet the current standard and the alternative W126 index value of 7 ppm-hrs, this number drops to 62% and 58%, respectively. We note that of the 1929 counties estimated to have 1 or more species with an RBL greater than 2% when meeting the current standard⁹, 1805 of those counties are estimated to have black cherry as the only species estimated to experience this level of biomass loss. Thus, as also discussed above in Section 6.2, given the magnitude of estimated RBL for black cherry over the entire range

⁹ Excluding cottonwood, as discussed above.

assessed, it is not clear to what extent the information for black cherry informs the selection of an appropriate level. We next consider the wRBL estimates from the WREA analysis of 145 (of the 155) federally designated Class I areas for which there was sufficient information regarding O₃-sensitive species (U.S. EPA, 2014b, section 6.8.1, Table 6-26, Appendix 6E). These 145 parks had at least one O₃-sensitive tree species for which an E-R function for RBL was available. Using the E-R functions for the species found within each park, the WREA calculated an average wRBL value for each park for the 3-year average W126 index values estimated in those locations for the current standard and three W126 air quality scenarios. Under conditions adjusted to just meet the current standard, the average wRBL in 2 of the 145 parks is estimated to be above 2%, as presented in Table 6-3 below. These two parks are Badlands National Park, driven by sensitivity of cottonwood, and Wind Cave National Park, driven by sensitivity of ponderosa pine. We compare this estimate to those for the W126 scenarios. For the W126 scenarios of 15 and 11 ppm-hrs, the estimated weighted RBL is greater than 2% in these same two of the 145 parks, while it is greater than 2% in only 1 park (Wind Cave) for the 7 ppm-hrs scenario.

Table 6-3. Number of Class I areas (of 145 assessed) with weighted relative biomass loss greater than 2%.

	-----Air Quality Scenarios -----			
	Conditions just meeting the current standard ^A	3-Year Average W126 index scenarios ^B		
		15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
Number of Class I areas with wRBL>2%	2	2	2	1

^A The wRBL is estimated per model grid cell (in which there are any of the 12 studied species) from W126 index value assigned to the grid cell based on application of the VNA method to the monitor-location W126 index values that are the average at that location across the 3 years of W126 index values for the adjusted dataset that just meets the current standard (4th highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb).

^B The national distribution of W126 index values within model grid-cells for each scenario reflects model-based adjustment of 2006-2008 O₃ concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA, 2014b, section 4.3.4.1 and Appendix 4A).

The WREA estimates of crop yield loss for the modeled air quality scenarios are summarized in Table 6-4 below (details are provided in U.S. EPA 2014, section 6.5.1 and Appendix 6B). For the recent air quality conditions scenario, the means for all crops were less than 5% loss across all states. Crop yield loss estimates for all states were also less than 5% in the air quality scenario representing conditions just meeting the current standard (U.S. EPA, 2014, section 6.5.1 and Appendix 6B).

Table 6-4. Estimated mean yield loss (and range across states) due to O₃ exposure for two important crops.

Crop	----- Air Quality Scenarios -----				
	Recent Conditions (2006-2008)	Conditions just meeting the current standard ^A	Average W126 index scenarios ^B		
			15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
Corn	<5% ^C (0.01-0.88)	<5% (0.0-0.01)	<5% (0.0-0.01)	<5% (0.0 – 0.0)	<5% (0.0 – 0.0)
Soybean	<5% (0.69-8.30)	<5% (0.01 – 1.39)	<5% (0.01 – 1.13)	<5% (0.01 – 0.75)	<5% (0.01 – 0.59)

^A The crop yield loss is estimated per grid cell (and per FASOMGHG region) from W126 index value assigned to the cell based on application of the VNA method to the monitor-location W126 index values that are the average at that location across the 3 years of W126 index values for the adjusted dataset that just meets the current standard (4th highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb).

^B The national distribution of W126 index values within grid cells for each scenario reflects model-based adjustment of 2006-2008 O₃ concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA 2014 section 4.3.4.1 and Appendix 4A).

^C Mean yield loss is the mean across modeling units. The range presented in parentheses below the mean represents the minimum and maximum estimates across modeling units (U.S. EPA 2014, Appendix 6B).

The WREA also analyzes market responses to changes in timber and agricultural production (U.S. EPA, 2014b, sections 6.3 and 6.5). As explained above, however, comparisons of the WREA’s air quality scenarios for the national-scale estimates of timber production and consumer and producer surpluses are not straightforward to interpret due to market dynamics. Estimates for the recent conditions and current standard scenarios are compared to the three W126 scenarios. In general, substantially greater economic surpluses (approximately \$51 billion) are estimated from the comparison of the recent conditions (2006-2008) scenario to the current standard scenario. The vast majority of these economic surpluses are estimated for agricultural production. Differences of the average W126 scenarios from the current standard scenario are much smaller (U.S. EPA 2014, Appendix 6B).

Because increases in timber production represent increased tree growth and concurrent carbon sequestration, we also consider WREA estimates of the potential increase in carbon storage that potentially could occur for different air quality scenarios (U.S. EPA 2014, section 6.6.1). Comparisons of the W126 scenarios to the current standard scenario with regard to carbon sequestration estimates do not indicate an appreciable difference for the W126 scenario of 15 ppm-hrs beyond that achieved by just meeting the current standard. The majority of the enhanced carbon sequestration potential resulting from increases in forest biomass is predicted to occur for the W126 scenarios of 11 and 7 ppm-hrs. Over 30 years, the current standard scenario

projection is 89,184 million metric tons of CO₂ equivalents (MMtCO₂e).¹⁰ The WREA estimates additional sequestration potential of 13, 593 and 1,600 MMtCO₂e, for the W126 scenarios of 15, 11 and 7 ppm-hrs, respectively, as compared to the current standard (U.S. EPA 2014, Table 6-18). We also take note of the relatively smaller estimates for carbon sequestration associated with improved crop yields (over 30 years) in the agricultural sector, which indicate little difference among the different W126 scenarios, beyond that achieved by just meeting the current standard.

We additionally consider the WREA estimates for five urban areas of how reduced growth of O₃-sensitive trees in urban forests may affect the ecosystem services of air pollutant removal and carbon sequestration (U.S. EPA, 2014b, sections 6.6.2 and 6.7 and Appendix 6D). With regard to air pollutant removal, the WREA estimated metric tons of carbon monoxide, nitrogen dioxide, ozone and sulfur dioxide removed under the W126 scenarios. In considering these estimates we note the general assumptions made to estimate order of magnitude effects of O₃ removal by trees on O₃ concentrations in the five urban areas and the associated uncertainties (U.S. EPA 2014, sections 6.7 and 6.9 and Appendix 6D). Estimates for all five case study areas indicate increased pollutant removal from the recent conditions scenario to just meeting the current standard scenario, with much smaller differences between the current standard and the three W126 scenarios (Table 6-5 below). The largest difference in carbon sequestration is between the existing conditions scenario and the current standard scenario (Table 6-5). In addition to the small differences in W126 index values among the three W126 air quality scenarios relative to the current standard for these five areas, only 2 or 3 tree species were able to be assessed in each city. Therefore, these results may underestimate the overall impacts in these areas and nationally, although other areas of uncertainty (recognized below) may tend to contribute to the opposite potential (U.S. EPA 2014, Table 6-27).

¹⁰ 1 MMtCO₂e is equivalent to 208,000 passenger vehicles or the electricity to run 138,000 homes for 1 year as calculated by the EPA Greenhouse Gas Equivalencies Calculator (updated September 2013 and available at <http://www.epa.gov/cleanenergy/energy-resources/calculator.html>).

Table 6-5. Estimated effect of O₃-sensitive tree growth-related impacts on the ecosystem services of air pollutant removal and carbon sequestration in five urban case study areas.

Case Study Area	----- Air Quality Scenarios -----				
	Recent Conditions (2006-2008)	Conditions just meeting the current standard ^A	Average W126 index scenarios ^B		
			15 ppm-hrs	11 ppm-hrs	7 ppm-hrs
	Air Pollutant Removal (metric tons, CO, NO₂, O₃, SO₂)				
Atlanta	33,000	35,800	35,800	36,000	36,300
Baltimore	8,500	9,200	9,200	9,200	9,200
Chicago	355,000	359,000	359,000	361,000	365,000
Syracuse	1,500	1,700	1,700	1,700	1,700
Tennessee urban	474,000	511,000	511,000	516,000	522,000
	Carbon Storage (million metric tons of CO₂ equivalents, cumulative over 25 years)				
Atlanta	1.2	1.32	1.32	1.32	1.34
Baltimore	0.5	0.57	0.57	0.57	0.57
Chicago	16.9	17.05	17.05	17.10	17.21
Syracuse	0.14	0.17	0.17	0.17	0.17
Tennessee urban	18.0	19.67	19.67	19.89	20.16
^A Results are derived from estimates per model grid cell (in which there are any of the 12 studied species) from W126 index value assigned to the grid cell based on application of the VNA method to the monitor-location W126 index values that are the average at that location across the 3 years of W126 index values for the adjusted dataset that just meets the current standard (4 th highest daily maximum 8-hour concentration, averaged over 3 consecutive years of 75 ppb). ^B The national distribution of W126 index values within model grid-cells for each scenario reflects model-based adjustment of 2006-2008 O ₃ concentrations at monitoring sites such that the average W126 index at the controlling location in each of the modeling regions just meets the scenario target index value, followed by application of the VNA interpolation methodology (see U.S. EPA, 2014b, section 4.3.4.1 and Appendix 4A).					

With regard to foliar injury, we take note of the WREA analyses of the nationwide dataset (2006- 2010) for USFS/FHM biosites described in section 5.4.2 above, including the observation that the proportion of biosites with injury varies with soil moisture conditions and O₃ W126 index values (U.S. EPA 2014, Chapter 7, Figure 7-10). The evidence of O₃-attributable visible foliar injury incidence occurring in USFS/FHM biosites shows that the proportion of biosites showing foliar injury incidence increases steeply with W126 index values up to approximately 10 ppm-hrs. At W126 index levels greater than approximately 10 ppm-hrs, the proportion of sites showing foliar injury incidence is relatively constant.

In reflecting across the range of W126 index values evaluated in various WREA analyses, we first note the substantial reductions in biomass-related risks estimated for air quality

adjusted to just meet the current standard scenario. Additional incremental risk reductions are estimated across the W126 scenarios, although these risk reductions are substantially smaller.

In considering the WREA estimates here, we take note of uncertainties in the adjusted estimates. Adjustments were made to recent air quality to reflect just meeting the current standard and three W126 levels. These adjustments were based on air quality modeling simulations reflecting across-the-board reductions in NO_x emissions required to bring the highest monitor down to the target level in different regions of the country. In some areas, meeting a target level at the highest monitor in the region had the effect of substantially reducing concentrations below the targeted level in other parts of the region. This adjustment approach is not meant to represent an actual control strategy but to provide an approximation of the spatial variability of O₃ across an area when just meeting the current standard and three W126 levels.

We also note potential uncertainties in the extent to which the results for each modeled air quality scenario represent cumulative seasonal O₃ exposures that would be expected to occur across the three years represented in each scenario. In general, each scenario is represented by a dataset of 3-year average W126 index values across the national modeling area. Thus, the results estimated for the various analyses that use these scenarios do not reflect any year-to-year variability that would be expected in single year results. Rather, they reflect average estimates for the three year period modeled. Analyses in the WREA describe the potential for the WREA estimates to underestimate cumulative biomass-related effects in perennial species (as noted in sections 6.2 and 5.2.2 above and described in detail in U.S. EPA, 2014b, chapter 6, 6.2.1.4). This potential for underestimation is recognized in the context of the uncertainties associated with other aspects of the different analyses in section 6.9 of the WREA (e.g., U.S. EPA, 2014b, Table 6-27). We additionally note that the WREA compounding analyses do not take into account other variables that can affect the magnitude of these effects in the field. In considering this information discussed above in the context of identifying levels appropriate to consider for a W126-based standard, we take note of additional associated uncertainties as discussed under the following question.

- **What are important uncertainties and limitations in the evidence and exposure/risk analyses?**

In considering the evidence and exposure/risk information summarized above and the weight to place on this information, we are mindful of the uncertainties and limitations associated with several key aspects of this information. We first consider the uncertainties associated with the evidence underlying the tree seedling and crop E-R functions, given the importance of these functions for many of the ecosystem service analyses described in the

WREA. Several key uncertainties associated with this information are listed below and described in more detail in the WREA (U.S. EPA, 2014b).

- Uncertainty regarding the extent to which the subset of studied tree and crop species encompass the total number of O₃ sensitive species in the nation and to what extent it is representative of U.S. vegetation as a whole, given that information is available for only a small fraction of the number of total species of trees and crops grown in the U.S. (U.S. EPA, 2013, section 9.6, U.S. EPA, 2014b, Table 6-27).
- Uncertainties regarding intra-species variability due to the different numbers of studies that exist for different species so that the weight of evidence is not the same for each species. Those species with more than one study show variability in response and E-R functions. The potential variability in less well-studied species is, however, unknown (U.S. EPA, 2013, pp. 9-123/125, U.S. EPA, 2014b, section 6.2.1.2, and Table 6-27).
- Uncertainty regarding the extent to which tree seedling E-R functions can be used to represent mature trees since seedling sensitivity has been shown in some cases to not reflect mature tree O₃ sensitivity in the same species (U.S. EPA, 2013, section 9.6, U.S. EPA, 2014b, section 6.2.1.1 and Tables 6-5 and 6-27).
- Uncertainty in the relationship of O₃ effects on tree seedlings (e.g., relative biomass loss) in one or a few growing seasons to effects that might be expected to accrue over the life of the trees extending into adulthood (U.S. EPA, 2013, pp. 9-52/53, U.S. EPA, 2014b, section 6.2.1.4 and Table 6-27).
- Uncertainties associated with estimating the national scale ecosystem-level impacts using weighted relative biomass loss (U.S. EPA, 2014b, section 6.8, and Table 6-27).
- Uncertainties associated with potential biomass loss in federally designated Class I areas (U.S. EPA, 2014b, section 6.8. and Table 6-27).

Turning to consideration of the air quality conditions estimated for the various air quality scenarios, we take note of the following uncertainties associated particularly with estimates of O₃ exposures in rural areas nationally. These are described more completely in chapter 4 of the WREA (see for example, U.S. EPA, 2014b, section 4.4) and summarized in chapter 8 of the REA (U.S. EPA, 2014b, section 8.5).

- Uncertainties in O₃ exposures due to a lack of rural monitors, especially in the western U.S. and at high elevation sites.
- Uncertainties associated with the method (VNA) used to interpolate monitor values to estimate W126 index values in locations without monitors.
- Uncertainties in adjusted estimates of O₃ concentrations associated with meeting the current standard and potential alternative W126-based standards.

Numerous ecosystem services assessments were described in the WREA. These assessments relied heavily on models, which also relied on the inputs of the tree seedling and crop E-R functions and adjusted air quality estimates. Thus, including the uncertainties from the

first two categories discussed above, additional uncertainties associated with the ecosystem services models include the following.

- Uncertainties associated with use of the i-Tree model to estimate pollution removal and carbon storage in five urban area case studies, including uncertainties in the base inventory of city trees, the functions used for air pollution removal and carbon storage (U.S. EPA, 2014b, sections 6.6.2, 6.7, and Table 6-27).
- Uncertainties associated with use of the FASOMGHG model for national timber and crop production, including use of median E-R functions for crops in FASOM and crop proxy and forest type assumptions to fill in where there was insufficient data (U.S. EPA, 2014b, sections 6.3, 6.5, 6.6.1, and Table 6-27).
- Uncertainties associated with use of the FASOMGHG model to estimate national scale carbon sequestration, including those associated with the functions for carbon sequestration (U.S. EPA, 2014b, sections 6.2.1.1, 6.6.1, and Table 6-27).

In addition, the WREA estimates the incidence and of O₃-induced visible foliar injury, both at the national and national park scales. Numerous uncertainties are associated with these assessments and include the following.

- Uncertainties associated with our understanding of the number and sensitivity of O₃ sensitive species (U.S. EPA, 2014b, sections 7.2.1, 7.5 and Table 7-22).
- Uncertainties associated with spatial assignment of foliar injury biosite data to 12x12 km grids (U.S. EPA, 2014b, sections 7.2.1, 7.5 and Table 7-22).
- Uncertainties associated with availability of biosite sampling data in some locations in the western U.S. (U.S. EPA, 2014b, sections 7.2.1, 7.5 and Table 7-22).
- Uncertainties associated with soil moisture threshold for foliar injury (U.S. EPA, 2014b, sections 7.2.2, 7.2.3, 7.5 and Table 7-22).
- Uncertainties associated with spatial resolution of soil moisture data, time period for soil moisture data, drought categories and the combination of soil moisture and biosite data (U.S. EPA, 2014b, sections 7.3.3.2, 7.5 and Table 7-22).
- Uncertainties associated with O₃ exposure data of vegetation and recreational areas within parks (U.S. EPA, 2014b, sections 7.4, 7.5 and Table 7-22).
- Uncertainties associated with surveys of recreational activities (U.S. EPA, 2014b, sections 7.1.1.2, 7.5 and Table 7-22).

Additionally, there is uncertainty associated with the extent to which the endpoints and associated risk estimates considered above represent effects reasonably judged adverse in the context of public welfare. Despite these uncertainties, the overall body of scientific evidence underlying the ecological effects and associated ecosystem services evaluated in the WREA is strong, and the methods used to quantify associated risks are scientifically sound (Frey, 2014b).

All of these uncertainties are important to considerations below in the context of target levels of protection with regard to weight to be placed on various lines of evidence and assessment results.

- **Are there other aspects of the form that affect consideration of the welfare protection provided by the level of the cumulative seasonal standard?**

Although cumulative, seasonal exposure indices of interest for vegetation effects are often expressed in terms of a single season, we recognize that it can also be appropriate to consider a form that is evaluated over a multiple-year period, such as three years (U.S. EPA, 2007; 72 FR 37901; 75 FR 3021). The current form of the secondary standard is a 3-year average, and we recognize that the protection provided by the secondary standard derives from the combination of all elements of the standard (indicator, form, averaging time(s), and level). Thus, we find it appropriate to evaluate the protection that might be afforded by a form limited to a single year or one that is based on evaluation of exposures across multiple years. Although cumulative, seasonal exposure indices of interest for vegetation effects are often expressed in terms of a single season, we recognize that it can also be appropriate to consider a form that is evaluated over a multiple-year period, such as three years (U.S. EPA, 2007; 72 FR 37901; 75 FR 3021). Accordingly, this discussion explores the information relevant to consider in conjunction with the above identification of the W126 index form, 12-hour daylight averaging time and maximum consecutive 3-month seasonal exposure period, and the subsequent discussion on level below, when considering support in the current information for single and/or multiple-year options.

We additionally take note of advice from CASAC on this topic in the current and prior reviews. Specifically, in this review, CASAC stated that it “does not recommend the use of a three-year averaging period for the secondary standard. We favor a single-year period for determining the highest three-month summation which will provide more protection for annual crops and for the anticipated cumulative effects on perennial species. The scientific analyses considered in this review, and the evidence upon which they are based, are from single-year results. If, as a policy matter, the Administrator prefers to base the secondary standard on a three-year averaging period for the purpose of program stability, then the level of the standard should be revised downward such that the level for the highest three-month summation in any given year of the three-year period would not exceed the scientifically recommended range of 7 ppm-hrs to 15 ppm-hrs” (Frey, 2014a, p. iii).

In considering an annual form of a standard, we particularly take note of O₃-induced vegetation effects that can occur as a result of a single year’s exposure. These include visible foliar injury symptoms, growth reduction in annual and perennial species, and yield loss in annual crops. The following discussion considers these effects, in the context of their potential public welfare significance, and in regard to the extent to which a W126-based standard with an

annual form or one based on evaluation across multiple years may be able to provide appropriate protection

In the case of foliar injury, the ISA notes that the full body of evidence indicates that there is wide variability in this endpoint, such that although evidence shows visible foliar injury can occur under very low cumulative O₃ concentrations, "...the degree and extent of visible foliar injury development varies from year to year and site... even among co-members of a population exposed to similar O₃ levels, due to the influence of co-occurring environmental and genetic factors" (U.S. EPA 2013, section 9.4.2, p. 9-38). In addition, the WREA assessment of foliar injury showed the difficulty and complexity associated with identifying W126 index values that would consistently provide appropriate protection on an annual basis for this endpoint. We thus conclude that there is limited information to discern between the level of protection provided by an annual form or a 3-year average form of a W126 standard for this endpoint, and that a multiple year form could be considered to provide a more consistent target level of protection for this endpoint, given likely fluctuations in annual O₃ and soil moisture conditions.

In the case of annual commodity crops, the overall welfare effect of annual changes in yields due to O₃ exposures is not straightforward. As noted above, determining at what point O₃-induced crop yield loss becomes adverse to the public welfare is still unclear, given that it is heavily managed with additional inputs that have their own associated markets and that benefits can be unevenly distributed between producers and consumers. We thus conclude that there is limited information to discern between the level of protection provided by an annual form or a 3-year average form of a W126 standard for this endpoint. As with foliar injury, we thus conclude that it is appropriate to consider a level of protection for annual commodity crops that would be achieved, on average, using a multiple year form, to provide a more consistent target, given likely fluctuations in environmental and economic conditions.

In contrast to impacts on annual species that accrue in the single growing season in which the O₃ exposures occur, annual effects in perennial species can be "carried over" into the subsequent year where they affect growth and reproduction (U.S. EPA, 2013, pp. 9-43 to 9-44 and p. 9-86). In addition, when these effects occur over multiple years due to elevated O₃ exposures across several years, they accumulate and potentially compound, increasing the potential for effects at the ecosystem level and associated ecosystem services that may be of significance to the public welfare.

Effects from elevated O₃ years on perennial plants, when they occur over several years, can be propagated up to higher spatial scales where they can contribute to effects on ecosystem services, e.g., alteration of below-ground biogeochemical cycles, and alteration of both above- and below- ground terrestrial community composition and terrestrial ecosystem water cycling

(U.S. EPA, 2013, Table 9-19). Ozone has also been shown to affect plant reproduction in numerous ways (U.S. EPA, 2007, 7.3.3.3; U.S. EPA, 2013, 9.4.3.1). These effects, when they occur at sufficient magnitude for a single species, may result in impaired recruitment and loss of the species from the stand or community. This has the potential to change the community composition and biodiversity. If these effects occur in multiple plant species and/or over multiple years, they can result in a reduction in the productivity and carbon sequestration of terrestrial ecosystems. Such ecosystem-related effects and others discussed in the ISA may be considered to reflect impacts of critical O₃ exposures over the longer term. We additionally note that as compared to intermittent (or single year) critical O₃ exposures, multiple years of such exposures might be expected to result in larger impacts on forested areas, e.g., increased susceptibility to other stressors such as insect pests, disease, co-occurring pollutants and harsh weather, due to the potential for compounding or carry-over effects on tree growth.

Given the above, we find it reasonable to conclude that the public welfare significance of the effects that can accumulate as a result of multiple-year O₃ exposures have the potential to be greater and more certain than those that are realized in an individual year. Thus, to the extent that the focus for public welfare protection is on long-term effects that occur in sensitive tree species in natural forested ecosystems, including in federally protected areas such as Class I areas or on lands set aside by states, tribes and public interest groups to provide similar benefits to the public welfare, a cumulative seasonal standard that evaluates exposures across multiple years (in combination with an appropriate level) might be a more appropriate match to provide the requisite protection for those O₃-related effects on vegetation that when accumulated across years, are potentially significant and adverse to the public welfare.

Additionally, we address the potential for cumulative impacts on biomass loss over a 3-year period versus a 1-year period. First it is important to note that the WREA analyses that characterize plant biomass and associated ecosystem services effects, discussed above in this section, are based on a 3-year average. The WREA analysis examined the potential for biomass loss estimates based on a 3-year average W126 index value to underestimate the cumulative impact on growth based on the biomass loss that would be predicted in each of the 3 years, based on the yearly W126 index values. The results show that the use of the three-year average W126 index value may underestimate RBL values slightly. However, it should be noted that the approach does not account for moisture levels or other environmental factors that could affect biomass loss (U.S. EPA, 2014b, section 6.2.1.4 and Figure 6-14). In considering these results, we note that in these regions and in all three years, the three-year average W126 index value is sometimes above and sometimes below the individual year W126 index value.

In addition to the vegetation effects considerations described above, there are other policy-relevant factors that can be useful to consider. For example, under a standard with a

single year form, a monitor may be judged to meet the standard based on a single year of data, while under a standard with a form requiring evaluation over a multi-year period, a monitor is not judged to have met the standard until a complete multi-year record is available. For a W126-based potential standard, the multi-year form identified for consideration in the last review was the average cumulative seasonal metric over three consecutive years (75 FR 3027). Such a multi-year form remains appropriate to consider to provide stability to an alternative secondary standard, just as the multi-year form provides for the current standard (average over three years of annual fourth-highest daily maximum 8-hour average O₃ concentrations).¹¹ In considering the issue of stability in the context of such a form, we first note the inter-annual variability of seasonal W126 index, which is not unexpected given the logistic weighting function and also inter-annual variability in meteorological conditions that contribute to O₃ formation (see Appendix 2C). The staff analysis in Appendix 2C describes the variability in annual W126 index values in relation to variability in the 3-year average, which indicates that a standard based on an annual W126 index would be expected to have a lower degree of year-to-year stability relative to a standard based on a form that averages seasonal indices across three consecutive years. A more stable standard can be expected to contribute to greater public welfare protection by limiting year-to-year disruptions in ongoing control programs that would occur if an area was frequently shifting in and out of attainment due to extreme year-to-year variations in meteorological conditions. This greater stability in air quality management programs thus facilitates achievement of the protection intended by a standard. In light of this relationship, we conclude that a 3-year average form has the desirable feature of providing greater stability in air quality management programs and thus facilitating the achievement of the protection intended by a standard. Thus, we recognize the public welfare benefits of having a standard of a 3-year average form.

CASAC has asked that the PA quantify the ratio of the 3-year average of the highest three-month summations in each year to the highest three-month summation in the highest year within that same 3-year average period. This information is provided in a technical memorandum titled “Relationship between W126 annual values and three-year averages” (EPA-HQ-OAR-2005-0172) and in the analyses included in Appendix 2C. In the technical memorandum, an analysis summarized the relationship between annual W126 index values and the three-year averages of the annual values based on 2007-2009 air quality data. Based on the air quality data, 79 percent of counties meeting a three-year average W126 index value of 13 ppm-hrs would also not have annual W126 index values above 15 ppm-hrs. In addition, in terms

¹¹ See *ATA III*, 283 F. 3d at 374-75 (recognizing programmatic stability as a legitimate consideration in the NAAQS standard-setting process).

of county-years (i.e., the number of counties times the number of years in the analysis), 93 percent of the county-years meeting a three-year average W126 index value of 13-ppm-hrs would also meet an annual W126 index value of 15 ppm-hrs.

In addition, Appendix 2C compared annual W126 index values to three-year average W126 index values for 2008-2010 air quality data. It concluded that the data analysis “shows that the inter-annual variability in the annual W126 index tends to decrease with decreasing W126 levels. Thus, it is expected that reductions in NO_x emissions will not only result in lower 3-year average W126 levels, but also result in less inter-annual variability associated with annual W126 levels.” Appendix 2C also concludes that the inter-annual variability in the W126 index increases and decreases along with the three-year average.

These analyses suggest that meeting a 3-year average W126 index value of 13 ppm-hrs would mean that for most years and monitoring sites the annual W126 index value would be below 15 ppm-hrs. In addition, the relationship between 3-year average W126 index values and annual W126 index values is dynamic and varies with the three-year average W126 index value and will continue to change in the future with changing pollution levels.

Accordingly, in considering all elements for a revised standard, including level and form, we note that a standard with a form that averages across three years can also control for year-to-year variability and individual year concentrations. The appropriate level and form combination will depend on which effects endpoints are considered to warrant additional public welfare protection and what is considered to be the requisite range of target levels of protection. In articulating these objectives it may be appropriate to evaluate the nature of the O₃ induced effects and their significance or importance to the public welfare, as well as the role that year-to-year exposure variability can play in public welfare impacts.

- **What considerations may be important to the Administrator’s judgments on the public welfare significance of O₃ associated vegetation effects that may be expected under air quality conditions associated with different levels for a seasonal cumulative standard?**

Our consideration of this question is intended to provide a public welfare context for consideration of the evidence and exposure/risk information discussed above, which includes the nature and magnitude of observed and predicted effects at various levels of cumulative seasonal exposures. We also note the importance of considering information in an integrated manner, rather than focusing only on results from any one analysis. For example, we find it appropriate, in considering the evidence with regard to seedling growth reduction (or biomass loss), to consider the WREA estimates of affected area based on tree basal area together with estimates of individual species responses based simply on the evidence-based E-R functions, and in light of other potential impacts summarized above. In so doing in section 6.5 below, we take into

account considerations relevant to public welfare policy judgments required of the Administrator, such as those described here.

As recognized in sections 1.3.2 and 5.1, the Clean Air Act specifies that secondary standards specify a level of air quality that is requisite to protect against known or anticipated adverse effects to public welfare. In the Administrator's judgment as to the standards that would be requisite (i.e., neither more nor less stringent than necessary) to protect the public welfare under the Act, she may consider a number of factors including 1) what should be considered to constitute an adverse effect to the public welfare; 2) the nature and magnitude of the effects and the risks that remain after meeting the level of the current standard; and, 3) what is necessary to achieve the requisite (no more and no less) degree of public welfare protection. In the 2008 decision by which the current standard was established, the Administrator considered these factors in judging the previously existing standard to not provide the requisite public welfare protection. At that time the Administrator found that the exposure- and risk-based analyses available in that review indicated that adverse effects to vegetation would be predicted to occur under air quality conditions associated with just meeting the then-current standard. The effects identified were "visible foliar injury and seedling and mature tree biomass loss in O₃-sensitive vegetation" (73 FR 16496). In so noting, the Administrator indicated that he believed that "the degree to which such effects should be considered to be adverse depends on the intended use of the vegetation and its significance to public welfare" (73 FR 16496). With regard to consideration of intended use, the Administrator took note of the specific uses of public lands set aside by Congress and intended to provide benefits to the public welfare, "including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment for future generations" such as Class I areas (73 FR 16496). The Administrator also recognized areas set aside by states, tribes and public interest groups with the intent "to provide similar benefits to the public welfare, for residents on State and Tribal lands, as well as for visitors to those areas" (73 FR 16496).¹²

In the Administrator's judgments in the 2008 review, he did not identify specific criteria or benchmarks or a specific level of protection from adverse environmental effects to public welfare judged to be requisite under the Act.¹³ As noted above, the scientists at the 1996

¹² In considering areas that have not been afforded such special protection, ranging from vegetation used for residential or commercial ornamental purposes, such as land use categories that are heavily managed for commercial production of commodities such as agricultural crops, timber and ornamental vegetation, the Administrator indicated his expectation that protection of sensitive natural vegetation and ecosystems might be expected to also provide some degree of additional protection for heavily managed commercial vegetation (73 FR 16496).

¹³ In remanding the 2008 decision on the secondary standard back to the EPA, the Court of Appeals for the D.C. Circuit determined that EPA did not specify what level of air quality was requisite to protect public welfare

workshop identified ranges of cumulative seasonal index values (e.g., in terms of SUM06 or W126) in the context of considering a degree of protection for vegetation effects defined in terms of relative yield loss in crops and relative biomass loss in tree seedlings. Considering this information in the context of a secondary standard entails policy judgments by the Administrator with regard to the degree that impacts exceeding these or other benchmarks and other effects should be judged adverse to the public welfare. In considering levels for a W126-based secondary standard that may be appropriate to consider, we recognize that the statute requires that a secondary standard be protective against only those known or anticipated O₃ effects that are “adverse” to the public welfare, not all identifiable O₃-induced effects. Thus, we recognize both the importance of scientific consensus statements that have been made regarding vegetation-related endpoints and O₃ exposure levels that might protect against such key endpoints and the importance of placing such conclusions in the context of consideration of the public welfare more broadly.

As discussed in section 5.1 and recognized by the EPA in prior reviews, staff recognizes the importance of a more expansive construct or paradigm that addresses what constitutes adverse effects of O₃ to public welfare. In so doing, we also recognize several aspects or dimensions of vegetation effects for consideration within this paradigm. These include the likelihood, type, magnitude, and spatial scale of the effect, as well as the potential for recovery and any uncertainties relating to these conditions (77 FR 20231). As in the last review, we also continue to recognize that the public welfare significance of O₃-induced effects on sensitive vegetation growing within the U.S. can vary, depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located. Any given O₃-related effect on vegetation and ecosystems (e.g., biomass loss, foliar injury), therefore, may be judged to have a different degree of impact on the public welfare depending, for example, on whether that effect occurs in a Class I area, a city park, or in commercial cropland. In the 2008 review, the Administrator judged it appropriate that this variation in the significance of O₃-related vegetation effects should be taken into consideration in judging the level of ambient O₃ that is requisite to protect the public welfare from any known or anticipated adverse effects (73 FR 16496). For example, in considering visible foliar injury and seedling and mature tree biomass loss in O₃-sensitive vegetation expected under alternative air quality scenarios, the Administrator noted that “the degree to which such effects should be considered to be adverse depends on the intended use of the vegetation and its significance to the public welfare” (73 FR 16496). Further, the rulemaking

from adverse public welfare effects or explain why any such level would be requisite, as described in section 1.2.2 above. *Mississippi*, 744 F.3d at 272-73.

notice stated that “[i]n considering what constitutes a vegetation effect that is adverse from a public welfare perspective, the Administrator believes it is appropriate to continue to rely on the definition of ‘adverse,’ ... that imbeds the concept of ‘intended use’ of the ecological receptors and resources that are affected, and applies that concept beyond the species level to the ecosystem level” (73 FR 16496). The notice went on to state that “[i]n so doing, the Administrator has taken note of a number of actions taken by Congress to establish public lands that are set aside for specific uses that are intended to provide benefits to the public welfare, including lands that are to be protected so as to conserve the scenic value and the natural vegetation and wildlife within such areas, and to leave them unimpaired for the enjoyment of future generations” (73 FR 16496). Such public lands that are protected areas of national interest include national parks and forests, wildlife refuges, and wilderness areas.

We also consider effects on ecosystem services in considering adversity to public welfare. For example, the WREA has evaluated the economic value of ecosystem services affected by O₃ and how those services might be expected to change under different air quality scenarios representing the current and potential alternative standards (U.S. EPA, 2014b, chapters 6 and 7).

Lastly, we recognize several important considerations in evaluating levels of protection and levels for a cumulative seasonal W126-based standard including: the extent of areas expected to be affected nationwide and the magnitude of those effects; the extent of effects in areas of national significance; the extent to which these impacts might be judged significant from a public welfare perspective and associated uncertainties in the information. Accordingly, we recognize that the range of alternative standard levels that may be appropriate to consider differs based on the weight placed on different aspects of the evidence and on different aspects of the quantitative exposure/risk information, and the associated uncertainties, as well as on public welfare policy decisions regarding the public welfare significance of the effects considered and the approaches for considering benchmarks for growth or biomass loss and other vegetation effects of O₃. As described in chapter 1, our objective is to identify the range of policy options supported by the current evidence- and exposure/risk-based information and with consideration of the role of the Administrator’s public welfare judgments. In so doing, we recognize support for consideration of a broad range of W126 index values, which we discuss in section 6.5, with recognition of the different judgments that might provide support for different parts of such a range.

6.4 CONSIDERATION OF PROTECTIVENESS OF REVISED PRIMARY STANDARD

In staff consideration of the primary standard in chapter 4, staff concludes it is appropriate to consider alternative primary standards of the same form and averaging time as the current primary standard and a lower standard level within the range of 60 to 70 ppb. Thus, although the discussion in this chapter, with regard to the secondary standard, indicates the appropriateness of considering an alternative secondary standard with a cumulative, seasonal form, we also recognize that, to the extent that the Administrator may find it effective to control air quality using the same form for both the primary and secondary standards, it may be practical to consider the extent to which a standard in the form of the primary standard might be expected to also reduce and provide protection from cumulative seasonal exposures of concern. For example, if a clear and robust relationship was found to exist between 8-hour daily peak O₃ concentrations and cumulative, seasonal exposures, the averaging time and form of the current standard might be concluded to have the potential to be effective as a surrogate. In response to this, we ask the following question:

- **What does the available information indicate with regard to protection of welfare from cumulative O₃ exposures that might be afforded by alternative secondary standards based on the form of the current standard (a 3-year average of 4th highest 8-hour average concentrations)?**

Addressing this point, the ISA describes the results of a recent focus study that examined the diel¹⁴ variability in O₃ concentrations in six rural areas between 2007 and 2009 (U.S. EPA, 2013, pp. 3-131 to 3-133). The ISA reported that “[t]here was considerable variability in the diel patterns observed in the six rural focus areas” with the three mountainous eastern sites exhibiting a “generally flat profile with little hourly variability in the median concentration and the upper percentiles”, while the three western rural areas demonstrated a “clear diel pattern to the hourly O₃ data with a peak in concentration in the afternoon similar to those seen in the urban areas”, which was especially obvious at the San Bernardino National Forest site, 90 km east of Los Angeles at an elevation of 1,384 meters (U.S. EPA, 2013, p. 3-132). Thus, while the western sites that are influenced by upwind urban plumes may have increased cumulative seasonal values coincident with increased daily 8-hour peak O₃ concentrations, this analysis indicates that, in sites without such an urban influence (the eastern sites in this analysis), such a relationship does not occur (U.S. EPA, 2013, section 3.6.3.2). Thus, the lack of such a relationship indicates that in some locations, O₃ air quality patterns can lead to elevated cumulative, seasonal O₃ exposures without the occurrence of elevated daily maximum 8-hour average O₃ concentrations (U.S. EPA,

¹⁴ involving a 24-hr period

2013, section 3.6.3.2). Further, staff notes that the prevalence and geographic extent of such locations is unclear, since as in the last review, there continue to be relatively fewer monitors in the West, including in high elevation remote sites. In considering the findings of this analysis, we additionally recognize, however, that the cumulative seasonal values for the eastern rural sites, where cumulative seasonal O₃ concentrations appear to be relatively less related to daily maximum 8-hour concentrations, are lower in general than those of the western, urban-influenced sites.

In addition to the focus study described in the ISA (U.S. EPA, 2013, section 3.6.3.2), we considered analyses of air quality monitoring data and air quality modeling analyses. Chapter 2 of this document characterizes recent monitoring data on O₃ air quality in rural areas. While approximately 80% of the O₃ monitoring network is urban focused, about 120 rural monitors are divided among CASTNET, NCore, and portable ozone monitors (POMs) sites (Chapter 2, pp. 2-2 to 2-3, Figure 2.1). Specifically, as stated in chapter 2 “[a]lthough rural monitoring sites tend to be less directly affected by anthropogenic pollution sources than urban sites, rural sites can be affected by transport of O₃ or O₃ precursors from upwind urban areas and by local anthropogenic sources such as motor vehicles, power generation, biomass combustion, or oil and gas operations” (U.S. EPA, 2013, section 3.6.2.2). In addition, O₃ tends to persist longer in rural than in urban areas due to lower rates of chemical scavenging in non-urban environments. At higher elevations, increased O₃ concentrations can also result from stratospheric intrusions (U.S. EPA, 2013, sections 3.4, 3.6.2.2). As a result, O₃ concentrations measured in some rural sites can be higher than those measured in nearby urban areas (U.S. EPA, 2013, section 3.6.2.2). These known differences between urban and rural sites suggest that there is the potential for 8-hour daily peak O₃ concentrations and cumulative, seasonal exposures to not correlate well in those areas. However, while these metrics may not be directly correlated, reductions in NO_x emissions that occur in urban areas to attain primary standards would also have the effect of reducing downwind, rural concentrations over the season.

In addition, as was done in both the 1997 and 2008 reviews, staff has analyzed relationships between O₃ levels in terms of the current averaging time and form and a W126 cumulative form, based on recent air quality data. One analysis describes the W126 index values and current standard design values at each monitor for two periods: 2001-2003 and 2009-2011 (e.g., Appendix 2B, Figures 2B-2 and 2B-3). This shows that between the two periods, during which broad scale O₃ precursor emission reductions occurred, O₃ concentrations in terms of both metrics were reduced. There is a fairly strong, positive degree of correlation between the two

metrics (Appendix 2B).¹⁵ Focusing only on the latter dataset (2009-2011), it can be seen that at monitors just meeting the current standard (3-year average fourth-highest daily maximum 8-hour average concentration equal to 0.075 ppm), W126 index values (in this case 3-year averages) varied from less than 3 ppm-hrs to approximately 20 ppm-hrs (Appendix 2B, Figure 2B-3b). At sites with a 3-year average fourth-highest daily maximum 8-hour average concentration at or below a potential alternative primary standard level of 70 ppb, 3-year W126 index values were above 17 ppm-hrs at no monitors, above 15 ppm-hrs at one monitor, and above 13 ppm-hrs at 8 monitors. At sites with a 3-year average fourth-highest daily maximum 8-hour average concentration at or below a potential alternative primary standard level of 65 ppb, 3-year W126 index values were above 13 ppm-hrs at no monitors, above 11 ppm-hrs at three monitors, and above 7 ppm-hrs at 9 monitors. The majority of these monitoring sites are located in the West and Southwest and include the states of Arizona, California, Colorado, Nevada, New Mexico, and Utah. At sites with a 3-year average fourth-highest daily maximum 8-hour average concentration at or below a potential alternative primary standard level of 60 ppb, 3-year W126 index values were at or below 7 ppm-hrs at all monitors.

An additional analysis presents the data for sets of recent 3-year periods back to 2006 – 2008 and indicates that among the counties with O₃ concentrations that met the current standard, the number of counties with 3-year W126 index values above 15 ppm-hrs ranges from fewer than 10 to 24 (Appendix 2B, Figure 2B-9). In general during this longer period, W126 index values above 15 ppm-hrs and meeting the current standard were pre-dominantly in Southwest region. As the first analysis in Appendix 2B (for the 2001-2003 and 2009-2011 periods) indicates, monitors in the West and Southwest tend to have higher W126 index values relative to their design values than do monitors in other regions. This pattern is noteworthy because the Southwest region has a less dense monitoring network than regions in the Eastern U.S. (see Figure 2-1), so that the extent to which this pattern occurs throughout these regions is uncertain. Although single-year W126 index values were not separately analyzed in this analysis of the monitor data, it indicates appreciable variation in cumulative, seasonal O₃ concentrations among monitor locations meeting different levels of a standard of the current form.

Analyses of the WREA air quality scenarios indicate the potential for O₃ precursor emission reductions achieving O₃ concentrations that just meet different 8-hour standards to produce a significant reduction in 3-year W126 index values. For example, for the current standard scenario, nearly all adjusted monitors are at or below an estimated 3-year average W126 index value of 15 ppm-hrs (as summarized in section 5.2.2 and described in U.S. EPA, 2014b,

¹⁵ Appendix 2B additionally observes that the program implemented for reducing precursor emissions, especially NO_x, appears to have been an effective strategy for lowering both design values and W126 index values.

Table 4-1). Those monitors above 15 ppm-hrs would be limited to large urban areas in the southwestern U.S. (i.e., Phoenix, Los Angeles and Denver). When meeting a 4th highest 8-hour average scenario of 70 ppb averaged across 3 years, nearly all monitors in the U.S. would meet a 3-year W126 index value of 11 ppm-hrs, though some monitors in the southwest would remain between 11 and 15 ppm-hrs. At 65 ppb, all locations are at or below 11 ppm-hrs. Thus, similar to the monitoring analysis, the modeling analysis generally indicates reductions in W126 levels with reduced O₃ concentrations in terms of the current standard averaging time and form. This suggests that depending on the level for a standard of the current averaging time and form, a degree of welfare protection may be afforded. The extent to which such protection provides adequate public welfare protection additionally depends on the level of protection identified by the Administrator as requisite to protect the public welfare from any known or anticipated adverse effects. In so noting, however, we recognize the importance of also considering uncertainties in both the model-based adjustment analyses and those based on monitoring data. These uncertainties, including those related to monitor coverage, the extent to which recent data can be expected to describe future relationships, and modeling approaches¹⁶, among others, should be kept in mind when assessing the strength of this apparent relationship.

6.5 CASAC ADVICE

In our consideration of potential alternative standards, in addition to the evidence-based, risk/exposure-based, and air quality information discussed above, we also consider the advice and recommendations of CASAC in EPA's proposed 2010 reconsideration of the 2008 decision, as well as comments received in the current review, in the context of its review of the ISA, and the WREA and PA. Some of this advice on specific aspects of the evidence and exposure/risk information has already been discussed in the relevant sections above. This section specifically considers CASAC's scientific advice on the appropriate form, averaging times and level(s) associated with a secondary standard and other related science and policy advice. We have additionally considered public comments received to date, some of which have suggested a lack of new information to support a distinct secondary standard and others that urge the consideration of a secondary standard with a cumulative seasonal form using the W126 metric and a level within the range of 7 to 15 ppm-hrs.¹⁷

¹⁶ One uncertainty associated with the modeling approach, as noted in Chapter 5, relates to the lowering of the highest monitored values as a result of the application of the interpolation method used to estimate W126 index values at the centroid of every 12 X 12 km² grid resolution, rather than only at the exact location of a monitor.

¹⁷ Public comment received thus far in this review are in the docket EPA-HQ-OAR-2008-0699, accessible at www.regulations.gov.

In response to the EPA's solicitation of CASAC's advice on the Agency's proposed rulemaking as part of the reconsideration,¹⁸ CASAC conveyed their support for a secondary standard distinct from the primary standard, stating that it "also supports EPA's secondary ozone standard as proposed: a new cumulative, seasonal standard expressed as an annual index of the sum of weighted hourly concentrations (i.e., the W126 form), cumulated over 12 hours per day (8am to 8pm) during the consecutive 3-month period within the ozone season with the maximum index value, set as a level within the range of 7 to [1]5 ppm-hours. This W126 metric can be supported as an appropriate option for relating ozone exposure to vegetation responses, such as visible foliar injury and reductions in plant growth. We found the Agency's reasoning ... to be supported by the extensive scientific evidence considered in the last review cycle. In choosing the W126 form for the secondary standard, the Agency acknowledges the distinction between the effects of acute exposures to ozone on human health and the effects of chronic ozone exposures on welfare, namely that vegetation effects are more dependent on the cumulative exposure to, and uptake of, ozone over the course of the entire growing season (defined to be a minimum of at least three months). In this proposal, the Agency is responding to the clear need for a secondary standard that is different from the primary standard in averaging time, level and form" (Samet, 2010, p. i-ii).

In advice offered in the current review, which considers an updated scientific and technical record since the 2008 rulemaking, the CASAC reiterated its earlier conclusions regarding the appropriate form and averaging times for a secondary O₃ NAAQS at several points in its letter to the Administrator. In stating the basis for its conclusion, CASAC notes that "[i]n reaching its scientific judgment regarding the indicator, form, summation time, and range of levels for a revised secondary standard, the CASAC has focused on the scientific evidence for the identification of the kind and extent of adverse effects on public welfare" (Frey, 2014a, p. iii), and further that "[t]hese recommendations are based on scientific evidence of adverse effect associated with the presence of ozone in ambient air" (Frey, 2014a, p. 15). On this basis, CASAC reached its conclusions on the appropriate form for the secondary standard stating "[t]he CASAC supports the scientific conclusion in the Second Draft PA that the current secondary standard is not adequate to protect against current and anticipated welfare effects of ozone on vegetation. We recommend retaining the current indicator (ozone) but establishing a revised form of the secondary standard to be the biologically relevant W126 index accumulated over a

¹⁸ The reconsideration proposal included a proposed new cumulative, seasonal secondary standard, expressed as an index of the annual sum of weighted hourly concentrations (the W126 index), cumulated over 12 hours per day during the consecutive 3-month period within the O₃ season with the maximum index value, averaged over three years, set within a range of 7 to 15 ppm-hrs (75 FR 3027).

12-hour period (8 a.m. – 8 p.m.) over the 3-month summation period of a single year resulting in the maximum value of W126” (Frey, 2014a, p. iii).

In addition, we take note of the scientific advice provided by CASAC regarding its scientific judgments regarding appropriate target benchmarks of protection and the range of W126 index values that in its scientific judgment provides appropriate protection for these benchmarks. CASAC states that “[a] 2% biomass loss is an appropriate scientifically based value to consider as a benchmark of adverse impact for long-lived perennial species such as trees, because effects are cumulative over multiple years” and “[c]rop loss appears to be less sensitive than these other indicators, largely because of the CASAC judgment that a 5% yield loss represents an adverse impact, and in part due to more opportunities to alter management of annual crops” (Frey, 2014a, p. 14).

Given these benchmarks, CASAC provided further advice regarding an appropriate range of W126 levels that it considered appropriately protective. Specifically, “[t]he CASAC recommends that the level associated with this form be within the range of 7 ppm-hrs to 15 ppm-hrs to protect against current and anticipated welfare effects of ozone. The CASAC does not support a level higher than 15 ppm-hrs. For example, at 17 ppm-hrs, the median tree species has 6% relative biomass loss, and the median crop species has over 5% yield loss. These levels are unacceptably high” (Frey, 2014a, p. iii)¹⁹. CASAC further noted that “[w]ith compounding over the harvest cycle or life span of these species, this will result in considerably greater cumulative RBL as discussed above. For the more sensitive tree seedlings, a value closer to the lower end of the range (7 ppm-hrs) would be more appropriate. The level of 7 ppm-hrs is the only level analyzed for which the relative biomass loss for the median tree species is less than or equal to 2 percent. At 7 ppm-hrs, 7 of the 12 analyzed species have relative biomass loss of less than 2%” (Frey, 2014a, p. 14).

CASAC further noted 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. Most of the analyses found effects below 15 ppm-hrs (many at 10 or even 7 ppm-hrs)” (Frey, 2014a, p. 12).

CASAC also recognized that there were policy choices left to the Administrator with respect to determining an appropriate level of protection. In so doing “[t]he CASAC acknowledges that the choice of a level within the range recommended based on scientific evidence is a policy judgment under the statutory mandate of the Clean Air Act. Specifically, the

¹⁹ As noted in Section 6.3, the numbers for RBL for the median tree species have been updated between the second and final PA to deemphasize cottonwood, based on staff’s understanding of CASAC advice in that regard. We note that CASAC advice based on what is shown in Table 6-1 is no longer consistent in some cases with the revised table, and in particular with regard to median tree seedling RBL values.

Clean Air Act grants discretion to the Administrator to specify a standard that is ‘requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of [the] pollutant in the ambient air’ ... (Frey, 2014a, p. iii). In addition, CASAC also offered its policy advice regarding selection of an appropriate level within its scientifically recommended range, stating that “[a]s a policy recommendation, separate from its advice above regarding scientific findings, the CASAC advises that a level of 15 ppm-hrs for the highest 3-month sum in a single year is requisite to protect crop yield loss, but that lower levels provide additional protection against crop yield loss. Furthermore, there are specific economically significant crops, such as soybeans, that may not be protected at 15 ppm-hrs but would be protected at lower levels. A level below 10 ppm-hrs is required to reduce foliar injury. A level of 7 ppm-hrs is protective of relative biomass loss for trees and offers additional protection against crop yield loss and foliar injury. Therefore, 7 ppm-hrs is protective of ecosystem services. Thus, lower levels within the recommended range offer a greater degree of protection of more endpoints than do higher levels within the range” (Frey, 2014a, p. iii).

Additionally, in regard to the 3-year average option discussed in the second draft PA, CASAC thus notes that “[i]f, as a policy matter, the Administrator prefers to base the secondary standard on a three-year averaging period for the purpose of program stability, then the level of the standard should be revised downward such that the level for the highest three-month summation in any given year of the three-year period would not exceed the scientifically recommended range of 7 ppm-hrs to 15 ppm-hrs. ...The final Policy Assessment should quantify the ratio of the three-year average of the highest three-month summations in each year to the highest three-month summation in the highest year. This ratio should be used to determine what downward adjustment from the three-month summation in one year recommended here is needed if a three-year form is selected” (Frey, 2014a, pp. iii and iv).²⁰

Finally, we note that in commenting on the significance of the uncertainties associated with the evidence and exposure and risk analyses that remain, CASAC concludes that “[w]hile these scientific research priorities will enhance future scientific reviews of the ozone primary and secondary standards, we also make clear that there is sufficient scientific evidence, and sufficient confidence in the available research results, to support the advice we have given above for this review cycle of the primary and secondary standards” (Frey, 2014a, p. iv).

²⁰ See Section 6.4 and Chapter 2 for more discussion on the relationship between one-year and three-year average W126 index values.

6.6 STAFF CONCLUSIONS ON ALTERNATIVE STANDARDS

Staff's consideration of alternative secondary O₃ standards builds on our conclusion from section 5.7 above that the body of evidence, in combination with the results of the WREA analyses, calls into question the adequacy of the current secondary standard and provides support for consideration of alternative standards. In sections 6.1 to 6.3 above, we consider how the currently available scientific evidence and exposure/risk information informs staff conclusions regarding the basic elements of the NAAQS: indicator (6.1), form and averaging time (6.2), and level (6.3). In so doing, we consider both the information available at the time of the last review and information newly available since the last review that has been critically analyzed and characterized in the 2013 ISA. As an initial matter, with regard to the indicator, we conclude that based on the available science it is still appropriate to continue to use measurements of O₃ in accordance with federal reference methods as the indicator to address effects associated with exposure to ambient O₃ alone or in combination with related photochemical oxidants.

In considering alternative standards, staff has considered the available body of evidence as comprehensively assessed in the ISA, the risk and exposure information presented in the WREA, and CASAC advice and public comment in this review with regard to support for consideration of options that are different from the current standard, as articulated by the following overarching question:

- **To what extent does the currently available scientific evidence- and exposure/risk-based information, as reflected in the ISA and WREA, support consideration of alternatives to the current O₃ standard to provide increased protection from ambient O₃ exposures?**

In considering potential forms alternative to that of the current standard, we note that the form for the current secondary standard is the 4th highest daily maximum 8-hour average, averaged over three years. As discussed in chapter 5 and section 6.2 above, the longstanding evidence regarding the fundamental aspects of O₃ exposure that are directly responsible for inducing vegetation response indicates that plant response to O₃ is driven by the cumulative exposure to O₃ during the growing season (U.S. EPA, 2013, section 2.6.6.1). This cumulative exposure depends on both the total duration of the exposure (from repeated O₃ episodes) and the concentrations of those exposures (higher concentrations having a disproportionate impact as compared to lower concentrations). On the basis of this longstanding and extensive evidence, the ISA concludes that exposure indices that cumulate and differentially weight the higher hourly average concentrations over a season and also include the mid-level values offer the most scientifically defensible approach for use in developing response functions and in defining indices for vegetation protection (U.S. EPA, 2013, section 2.6.6.1).

CASAC advice in the 2008 review and on the 2010 proposed reconsideration also recognized that the nature of the exposures relevant to vegetation response is well described by a cumulative seasonal form and has supported the use of such a form for a secondary O₃ standard (Henderson, 2006; Samet, 2010). The current CASAC O₃ Panel has expressed similar views. We also note that on the basis of the evidence and exposure/risk information available in the two previous reviews, and in consideration of CASAC advice, the Administrator has recognized the importance of protecting vegetation from cumulative, seasonal exposures and proposed such a form as an appropriate, reasonable policy option (61 FR 65741-44; 72 FR 37899-905; 75 FR 3012-3027).

Thus, in considering alternative forms of the standard we conclude that it is reasonable and appropriate to consider a cumulative, concentration-weighted form to provide protection against cumulative, seasonal exposures to O₃ that are known or anticipated to harm sensitive vegetation or ecosystems. Such a form is specifically designed to focus on the kind of O₃ exposures that have been shown to cause harm to vegetation and would have a distinct advantage over the form of the current standard in characterizing air quality conditions potentially of concern for vegetation and in more directly demonstrating that the desired degree of protection against those conditions was being achieved.

In considering the appropriate index for a cumulative seasonal form, we recognize that a number of different cumulative concentration weighted indices have been developed and have been evaluated in the scientific literature and in past NAAQS reviews in terms of their ability to predict vegetation response and their usefulness in the NAAQS context (U.S. EPA, 2006, pp. 9-11 to 9-15 and pp. AX9-159 to AX9-187; U.S. EPA, 2007, pp. 7-15/16). While these various forms have different strengths and limitations, as noted in the ISA (U.S. EPA, 2013, section 9.5), the W126 index²¹ has some important advantages over other non-sigmoidally weighted cumulative indices. For example, given the lack of a discernible threshold for vegetation effects in general, we recognize the fact that the W126 metric does not have a cut-off in its weighting scheme (down to about 30 ppb below which the weighting factor is effectively zero), such that it includes consideration of potentially damaging lower O₃ concentrations. Additionally, the W126 metric adds increasing weight to hourly concentrations from about 40 ppb to about 100 ppb, an important feature because “as hourly concentrations become higher, they become increasingly likely to overwhelm plant defenses and are known to be more detrimental to vegetation” (U.S.

²¹ The W126 is a non-threshold approach described as the sigmoidally weighted sum of all hourly O₃ concentrations observed during a specified diurnal and seasonal exposure period, where each hourly O₃ concentration is given a weight that increases from 0 to 1 with increasing concentration (Lefohn et al, 1988; Lefohn and Runeckles, 1987; U.S. EPA, 2013, section 9.5.2).

EPA, 2013, p. 9-104). We additionally take note of CASAC advice in the 2008 review and on the 2010 proposed reconsideration recommending the use of the W126 index for a cumulative seasonal form for a secondary O₃ standard (Henderson, 2006; Samet, 2010). Similarly, the current CASAC O₃ Panel has indicated that a focus on a W126 form is appropriate (Frey, 2014a, p. iii). Therefore, on the basis of the strength of the evidence and advice from CASAC, we conclude that the W126 index is the most appropriate cumulative seasonal form to consider in the context of the secondary O₃ NAAQS review.

We next turn to the exposure periods – diurnal and seasonal – over which the W126 index would be summed in any given year. As discussed in section 6.2 above, the currently available information continues to provide support for a definition of the diurnal period of interest as the 12-hour period from 8:00 am to 8:00 pm (U.S. EPA, 2013, section 9.5.3). In prior reviews, the EPA has identified the 12-hour period from 8:00 am to 8:00 pm as appropriately capturing the diurnal window with most relevance to the photosynthetic process (72 FR 37900; 75 FR 3013), and CASAC has generally supported the 12-hour daylight period (Henderson, 2006, 2007). In light of the continued support in the evidence base and no evidence on this issue differing from that in previous reviews, we again conclude that it is appropriate to use the 12-hour period from 8:00 am to 8:00 pm to cumulate daily O₃ exposures. On this basis, we conclude that the 12-hour daylight window (8:00 am to 8:00 pm) represents the portion of the diurnal exposure period that is most relevant to predicting or inducing plant effects related to photosynthesis and growth and thus is an appropriate diurnal period to use in conjunction with a W126 cumulative metric.

With regard to a seasonal period of interest, the current evidence base continues to provide support for a seasonal period with a minimum duration of three months (U.S. EPA, 2013, section 9.5.3). We note that a plant is vulnerable to O₃ pollution as long as it has foliage and is physiologically active (U.S. EPA, 2013, section 9.5.3, p. 9-112), i.e., during its growing season. The exposure periods used in studies of O₃ effects on vegetation reflect this understanding and typically focus on study periods of 3-6 months. Included in the currently available evidence is a new analysis that compared 3- and 6-month maximum W126 index values for over 1,200 AQS and CASTNET EPA monitoring sites for the years 2008-2009 that found that the two accumulation periods were highly correlated (U.S. EPA, 2013, section 9.5.3, Figure 9-13). Thus, although we recognize that the selection of a single seasonal time period over which to cumulate O₃ exposures for a national standard necessarily represents a balance of factors, given the significant variability in growth patterns and lengths of growing season among vegetative species growing within the U.S., we conclude it is appropriate to identify the seasonal W126 index value as that derived from the consecutive 3-month period within the O₃ season with the highest W126 index value. We note that such a 3-month exposure period was also

supported by CASAC in advice provided during the last review and the 2010 proposed reconsideration (Henderson, 2006; Samet, 2010).

With regard to form, we additionally consider the period of time over which a cumulative seasonal W126-based standard should be evaluated. In so doing, we have considered the support for both a single year form and a form averaged over three years (section 6.2). We note comments from CASAC on this matter, in particular their comment in the current review that “[t]he CASAC does not recommend the use of a three-year averaging period for the secondary standard. We favor a single-year period for determining the highest three-month summation which will provide more protection for annual crops and for the anticipated cumulative effects on perennial species. The scientific analyses considered in this review, and the evidence upon which they are based, are from single-year results” (Frey, 2014a, p. iii).

We recognize that there are a number of O₃-induced effects that have the potential for public welfare significance within the annual timeframe. These effects mainly include reduced crop yields and visible foliar injury, as noted in section 6.2 above. There are uncertainties associated with these effects that make it difficult to determine the degree of annual protection needed to protect the public welfare from any known or anticipated adverse effects. There are also annual effects in perennial species that may result from a single year exposure and can be “carried over” into the subsequent year where they affect growth and reproduction (U.S. EPA, 2013, pp. 9-43 to 9-44 and p. 9-86). When such annual effects due to elevated O₃ exposures occur over multiple years, they have the further potential to be compounded, increasing the potential for effects at larger scales (e.g., population, ecosystem), including effects on associated services that may be of significance to the public welfare. These ecosystem services effects can include alteration of below-ground biogeochemical cycles, and alteration of both above- and below-ground terrestrial community composition and terrestrial ecosystem water cycling (U.S. EPA, 2013, Table 9-19) and reductions in productivity and carbon sequestration in terrestrial ecosystems. We additionally note that multiple consecutive years of critical O₃ exposures might be expected to result in larger impacts on forested areas (e.g., increased susceptibility to other stressors such as insect pests, disease, co-occurring pollutants and harsh weather) than intermittent occurrences of such exposures due to the potential for compounding or carry-over effects on tree growth.

Given the above, we conclude that the public welfare significance of the effects that can occur as a result of three-year O₃ exposures are potentially greater than those associated with a single year of such exposure. Thus, to the extent that the focus for public welfare protection to be afforded by the secondary O₃ standard is on long-term effects that occur in sensitive tree species in natural forested ecosystems, including federally protected areas such as Class I areas or on lands set aside by States, Tribes and public interest groups to provide similar benefits to the

public welfare, a standard with a form that evaluates the cumulative seasonal index across multiple years might be considered to provide a more appropriate match to the nature of O₃-related effects on vegetation upon which the secondary O₃ standard is focused. In considering such forms, we focus on one that averages the W126 index values across three years, as discussed in section 6.2 above.

In addition to the vegetation effects considerations described above, there are other policy-relevant factors that can be useful to consider. For example, under a standard with a single year form, a monitor may be judged to meet the standard based on a single year of data, while under a standard with a form requiring evaluation over a multi-year period, a monitor is not judged to have met the standard until a complete multi-year record is available. For a W126-based potential standard, the multi-year form identified for consideration in the last review was the average cumulative seasonal metric over three consecutive years (75 FR 3027). Such a multi-year form remains appropriate to consider to provide stability to an alternative secondary standard, just as the multi-year form provides for the current standard (average over three years of annual fourth-highest daily maximum 8-hour average O₃ concentrations).²² In considering the issue of stability in the context of such a form, we first note the inter-annual variability of seasonal W126 index, which is not unexpected given the logistic weighting function and also inter-annual variability in meteorological conditions that contribute to O₃ formation (see Appendix 2C). The staff analysis in Appendix 2C describes the variability in annual W126 index values in relation to variability in the 3-year average, which indicates that a standard based on an annual W126 index would be expected to have a lower degree of year-to-year stability relative to a standard based on a form that averages seasonal indices across three consecutive years. A more stable standard can be expected to contribute to greater public welfare protection by limiting year-to-year disruptions in ongoing control programs that would occur if an area was frequently shifting in and out of attainment due to extreme year-to-year variations in meteorological conditions. This greater stability in air quality management programs thus facilitates achievement of the protection intended by a standard. In light of this relationship, we conclude that a 3-year average form has the desirable feature of providing greater stability in air quality management programs and thus facilitating the achievement of the protection intended by a standard. Thus, we recognize the public welfare benefits of having a standard of a 3-year average form.

Thus, to the extent that the greater emphasis is placed on protecting against effects associated with multi-year exposures and maintaining more year-to-year stability of public

²² See *ATA III*, 283 F. 3d at 374-75 (recognizing programmatic stability as a legitimate consideration in the NAAQS standard-setting process).

welfare protection, we conclude that it is appropriate to consider a secondary standard form that averages the seasonal W126 index values across three consecutive years. We conclude that such a 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. Further, such a form might be concluded to contribute to greater stability in air quality management programs, and thus, greater effectiveness in achieving the desired level of public welfare protection, than that that might result from a single year form.

Turning to consideration of an appropriate range of levels for a W126-based standard, we first note that our general approach to informing these judgments recognizes that the available evidence demonstrates a range of O₃ sensitivity across studied plant species and documents an array of O₃-induced effects that extend from lower to higher levels of biological organization. These effects range from those affecting cell processes and individual plant leaves to effects on the physiology of whole plants, species effects and effects on plant communities to effects on related ecosystem processes and services. Given this evidence, it is not possible to generalize across all studied species regarding which cumulative exposures are of greatest concern, as this can vary by situation due to differences in exposed species sensitivity, the importance of the observed or predicted O₃-induced effect, the role that the species plays in the ecosystem, the intended use of the affected species and its associated ecosystem and services, the presence of other co-occurring predisposing or mitigating factors, and associated uncertainties and limitations. At the same time, the evidence also demonstrates that though effects of concern can occur at very low exposures in sensitive species, at higher cumulative exposures those effects would likely occur at a greater magnitude and/or higher levels of biological organization and additional species would likely be impacted. It is important to note, however, that due to the variability in the importance of the associated ecosystem services provided by different species at different exposures and in different locations, as well as differences in associated uncertainties and limitations, that, in addition to the magnitude of the ambient concentrations, both the species present and their public welfare significance are essential considerations in drawing conclusions regarding the significance or magnitude of public welfare impact.

Therefore, in developing conclusions in this PA, we take note of the complexity of judgments to be made by the Administrator regarding the adversity of known and anticipated effects to the public welfare and are mindful that the Administrator's ultimate judgments on the secondary standard will, as appropriate, reflect an interpretation of the available scientific evidence and exposure/risk information that neither overstates nor understates the strengths and limitations of that evidence and information.

As described above in section 5.1, we employ a paradigm to assist in putting the available science and exposure/risk information into the public welfare context. This paradigm has

evolved over the course of the O₃ NAAQS reviews and has also been informed by similar constructs developed for other secondary NAAQS reviews. As discussed in Section 5.1, this paradigm recognizes that the significance to the public welfare of O₃-induced effects on sensitive vegetation growing within the U.S. can vary depending on the nature of the effect, the intended use of the sensitive plants or ecosystems, and the types of environments in which the sensitive vegetation and ecosystems are located. Accordingly, any given O₃-related effect on vegetation and ecosystems (e.g., biomass loss, crop yield loss, visible foliar injury) may be judged to have a different degree of impact on or significance to the public welfare depending, for example, on whether that effect occurs in a Class I area, a city park, or commercial cropland. In the last review, the Administrator placed the highest priority and significance on vegetation and ecosystem effects to sensitive species that are known to or are likely to occur in federally protected areas such as national parks and other Class I areas, or on lands set aside by states, tribes and public interest groups to provide similar benefits to the public welfare (75 FR 3023-24; 73 FR 16496), recognizing that effects occurring in such areas would likely have the highest potential for being classified as adverse to the public welfare, due to the expectation that these areas need to be maintained in pristine or near pristine conditions to ensure their intended use is met. This approach also includes consideration of impacts to ecosystem goods and services. Although ecosystem services were not explicitly considered in the Administrator's decision in the last review, they were explicitly recognized as an important category of public welfare effects and they have an obvious relationship to consideration of intended use (73 FR 16492). In employing this approach, we note the support for it provided by CASAC advice in this review (Frey, 2014a).

In considering potential levels for an alternative standard based on the W126 metric, we focus the discussion primarily on: 1) impacts on tree growth, productivity and carbon storage; 2) crop yield loss; and 3) visible foliar injury. With respect to tree growth, we find it useful to consider the summary of relative biomass loss estimates in Table 6-1 above and the WREA risk/exposure estimates discussed in Section 6.3 and Appendix 6F. In Table 6-1, we take note of the different index value estimates with regard to the number of studied species below different response benchmarks, as well as with regard to the median response. We additionally consider the WREA estimates regarding: (1) percent of assessed geographic area exceeding 2% weighted relative biomass (Table 6-2); (2) number of assessed Class I areas with tree seedling weighted relative biomass loss estimates above 2% (Table 6-3); and (3) the percent median biomass loss across counties for different air quality scenarios (Table 5-5). Further, we note other WREA estimates for effects on ecosystem services related to public welfare, such as carbon sequestration and air pollutant removal. With respect to crop yield loss, we note the summary of crop yield loss estimates in Table 6-1 and the WREA risk/exposure estimates discussed in

Section 6.3 and Appendix 6F, which include individual species and median response. We also note information available on visible foliar damage to species occurring in natural settings, such as federal Class I areas, and the analyses in the WREA evaluating biosite data and several benchmarks of injury as summarized in section 5.4.2.

In focusing on trees and their associated ecosystem services, we first note that the studied tree species vary widely in their sensitivity to O₃-induced relative biomass loss. We thus find it informative to consider both median species values and individual species responses and RBL over the same W126 range. We note CASAC's advice regarding RBL levels, specifically their emphasis on a benchmark of median relative tree biomass loss at or below 2% and their view that a 6% median relative biomass loss is "unacceptably high". From Table 6-1 we see that median tree species biomass loss is at or below 2% only at the lowest W126 level assessed, 7 ppm-hrs. As the W126 level is incrementally increased, median RBL also increases incrementally, so that at W126 index values of 9, 11, 13, 15, 17 and 19, the median RBL increases to 2.4%, 3.1%, 3.8%, 4.5%, 5.3% and 6.0%, respectively. Thus over the W126 range of 7 – 17 ppm-hrs, median species biomass loss ranges from approximately 2% to approximately 5%.

We next take note of the number of individual species' RBLs that fall below those same benchmarks assessed for median species values. We also note the value of additionally characterizing the RBL estimates in comparison to higher loss levels such as 10% or 15%, especially for individual tree species. Based on Figure 5-1 (B) in Chapter 5, and as shown in Table 6-1, for W126 values at or below 17 ppm-hrs, the RBLs for each of 5 species is less than 2%. Thus, over the full range of alternative levels considered, the same level of protection relative to the 2% benchmark is achieved for these species. We therefore turn our attention to the remaining 6 studied species to see if additional information might be available to help inform consideration of an appropriate degree of protection. Specifically, we consider the RBL information available for the other species (i.e., eastern white pine, aspen, tulip poplar, ponderosa pine, red alder, and black cherry) to further inform our evaluation of the additional protection that potentially could be achieved at different W126 levels within the range identified. We note that, for W126 levels of 17 to 7 ppm-hrs, biomass loss decreases for these individual species with decreasing W126 levels such that at the W126 level of 17 ppm-hrs, five species have RBL above 6% while at the W126 level of 7 ppm-hrs, one species (black cherry) has an RBL above 6%. Taken together with the more tolerant species, the proportion of the studied tree species with RBLs below 6% are 6/11, 7/11, 8/11, and 10/11 at W126 index values of 17, 15, 13, and 11 ppm-hrs, respectively.

In consideration of other benchmark levels, 9/11 studied tree species have a predicted RBL below 10% at the W126 level of 17 ppm-hrs, while 10/11 species have a predicted RBL

below 10% for W126 levels of 15 to 7 ppm-hrs. In addition, 10/11 studied tree species have a predicted RBL below 15% for W126 levels of 17 to 7 ppm-hrs. We note that black cherry, the most sensitive of the 11 species, has RBLs ranging from approximately 36% at W126 index value of 17 down to approximately 17% at the W126 index value of 7 ppm-hrs. Thus, the predicted RBL for black cherry remains above 15% for W126 levels of 17 to 7 ppm-hrs, and it is not clear to what extent those predicted RBL values might inform consideration of the level of protection achieved for different W126 exposures within this range (Table 6-1; U.S. EPA, 2014b, section 6.2, Appendix 6A).

To further inform this issue, the WREA also characterizes the number of counties where the median RBLs were greater than 2% (U.S. EPA, 2014b, Table 6-7), as shown in Table 5-5. When air quality is adjusted to the current standard, 8% of the counties have median RBLs greater than 2%. That proportion drops to 7% for air quality adjusted to just meet a 3-year average W126 level of 15 ppm-hrs and to 6% for air quality adjusted to just meet a 3-year average W126 level of 7 ppm-hrs. Of the 239 counties (8% of counties) estimated to have a median RBL above 2% when meeting the current standard, 203 of those counties have a RBL greater than 2% because of the presence of black cherry. Thus, as also discussed above in Section 6.2, given the large magnitude of estimated RBL for black cherry over the entire range assessed, it is not clear to what extent the information for black cherry informs consideration of the overall level of protection achieved across the identified range.

In considering the potential magnitude of the ecosystem impact of reduced biomass in trees, we focus on the WREA estimates of weighted RBL for the W126 air quality scenarios (U.S. EPA, 2014b, section 6.8), focusing particularly on impacts in Class I areas. For the current standard and the three W126 scenarios (15 ppm-hr, 11 ppm-hr, and 7 ppm-hr), the percent of total national land-area having weighted RBL greater than 2% was 0.2%, 0.2%, 0.1% and <0.1%, respectively (Table 6-2; U.S. EPA 2014, Table 6-25). In addition, the WREA estimates indicate weighted RBL greater than 2% in 1-2 of 145 assessed nationally protected Class I areas for the current standard and all three W126 scenarios. To the extent that emphasis is given to such estimates for nationally protected Class I areas and for appreciable percentages of forested areas nationwide, a W126 index value extending up to 17 ppm²³ may be appropriate to consider.

The WREA provides qualitative and semi-quantitative information regarding the types and potential magnitude of O₃ impacts on ecosystem services. In noting the potential ecosystem

²³ While the WREA analyses did not include an air quality scenario for 17 ppm-hrs, the data suggests that, to adjust air quality to meet a W126 index value of 17 ppm-hrs, additional emissions reductions would have been needed relative to just meeting the current standard. Therefore, because the air quality scenarios for meeting the current standard and meeting a W126 index value of 15 ppm-hrs both indicate weighted relative biomass loss less than or equal to 2% in 143 of 145 assessed nationally protected Class I areas, the same would be true for an air quality scenario for just meeting a W126 index value of 17 ppm-hrs.

services benefits related to reductions in tree biomass loss resulting from just meeting potential alternative W126-based standards, we recognize, in particular, that impacts on climate regulation can reasonably be concluded to be potentially significant from a public welfare perspective and carbon sequestration has been identified as a potentially important tool for managing anthropogenic impacts on climate. The WREA estimates the potential increase in carbon storage that potentially could occur for different air quality scenarios (U.S. EPA 2014, section 6.6.1). Comparisons of the W126 scenarios to the current standard scenario with regard to carbon sequestration estimates do not indicate an appreciable difference for the W126 scenario of 15 ppm-hrs beyond that achieved by just meeting the current standard. The majority of the enhanced carbon sequestration potential in forests over time is predicted to occur for the alternative W126 scenarios of 11 and 7 ppm-hrs. Over 30 years, the current standard scenario projection is 89,184 million metric tons of CO₂ equivalents (MMtCO₂e).²⁴ The WREA estimates additional sequestration potential of 13, 593 and 1,600 MMtCO₂e for the W126 scenarios of 15, 11 and 7 ppm-hrs, respectively, as compared to the current standard (U.S. EPA 2014, Table 6-18). We additionally consider the WREA estimates for five urban areas of how reduced growth of O₃-sensitive trees in urban forests may affect air pollutant removal (U.S. EPA, 2014b, sections 6.6.2 and 6.7 and Appendix 6D). Estimates for all five case study areas indicate increased pollutant removal from the recent conditions to just meeting the current standard, with much smaller differences between the current standard and the three W126 scenarios (Table 6-5). However, we additionally take note of significant uncertainties and limitations associated with WREA estimates related to carbon sequestration and air pollution removal. Thus, we note that an identification of the requisite protection for forest trees and their associated ecosystem services would likely involve policy judgments regarding the appropriate weight to place on potential impacts to the public welfare with respect to estimated effects on the ecosystem services of carbon storage and urban air pollution removal associated with tree growth, as well as on the uncertainties associated with this information.

With respect to crops, we focus on the 10 robust E-R functions (barley, lettuce, field corn, grain sorghum, peanut, winter wheat, cotton, soybean, potato and kidney bean) described in the ISA and additionally analyzed in the WREA (Figure 5-4). We also note CASAC's advice regarding a recommended target benchmark protection level of 5% for median crop relative yield loss (RYL) and that, as shown in Table 6-1, W126 index values ranging from 7 to 17 ppm-hrs

²⁴ 1 million metric tons of carbon dioxide equivalents (MMtCO₂e) is equivalent to 208,000 passenger vehicles or the electricity to run 138,000 homes for 1 year as calculated by the EPA Greenhouse Gas Equivalencies Calculator (updated September 2013 and available at <http://www.epa.gov/cleanenergy/energy-resources/calculator.html>).

are estimated to have median crop RYL of less than or equal to approximately 5%. Given this, it is not clear to what extent this information informs the selection of an appropriate level.

When individual species are considered over this same range, the proportion of crops protected varies from 5/10, 6/10, 6/10, 9/10, 10/10, and 10/10 at the W126 levels of 17, 15, 13, 11, 9, and 7 ppm-hrs. To the extent a given species is judged as having particular importance to the public welfare, breaking the information down by species can be helpful. For example, less than 5% yield loss was estimated for soybeans at the W126 index value of 12 ppm-hrs (U.S. EPA 2014, Figure 6-3). Four of the studied crop species (i.e., barley, lettuce, field corn, and grain sorghum) are more tolerant, with RYL under 1% over the W126 range from 7 to 17 ppm-hrs. Peanut also remained under 4% RYL over the same W126 range. Other species differed regarding the W126 level at which RYL reached or fell below 5%. Specifically, for winter wheat, cotton, soybean, kidney bean and potato, the relevant W126 index values at which RYLs were below 5% are 15, 13, 11, 9 and 7 ppm-hrs. As noted in Chapter 5, and in early discussions in this chapter, the significance of these predicted RYLs to the public welfare could be informed by the recognition that crops are heavily managed to obtain the desired yield and the potential adversity to public welfare from yield reductions in any specific crop in a particular location would depend on a number of economic factors, including crop prices, crop substitution, and the welfare importance of relative changes in consumer and producer surplus. We also note that these crop species would likely receive some protection from a standard set, for example, to provide protection against tree biomass loss, such as in areas set aside to be maintained in a more pristine condition (75 FR 3024).

Visible foliar injury has been identified by the FLMs as a diagnostic tool for informing conclusions regarding potential ozone impacts on potentially sensitive AQRVs (USFS, NPS, FWS, 2010), indicating that such O₃-induced impacts might be considered to have the potential to impact the public welfare in scenic and/or recreational areas during years they occur. We take note of the WREA analyses of the nationwide dataset (2006-2010) for USFS/FHM biosites described in section 5.4.2 above, including the observation that the proportion of biosites with injury varies with soil moisture conditions and O₃ W126 index values (U.S. EPA 2014, Chapter 7, Figure 7-10). These analyses also show that foliar injury incidence increases steeply with W126 index values up to approximately 10 ppm-hrs. At W126 index levels greater than that, little or no further increase in proportion of sites showing foliar injury occurs.

With respect to visible foliar injury, we are unaware of any guidance for federal land managers regarding at what spatial scale or what degree of severity visible foliar injury is sufficient to trigger protective action based on this potential impact on AQRVs. Further, there does not appear to be any consensus in the literature regarding severity of foliar injury and risks to plant functions or services, and CASAC, while identifying target percent biomass loss and

yield loss benchmarks for tree seedlings and commodity crops, respectively, did not provide a similar recommendation for this endpoint. Likewise, as in previous reviews, the ISA notes the difficulty in relating visible foliar injury symptoms to other vegetation effects such as individual plant growth, stand growth, or ecosystem characteristics (U.S. EPA, 2013, section 9.4.2, p. 9-39) and further noted that the full body of evidence indicates that there is wide variability in this endpoint, such that although evidence shows visible foliar injury can occur under very low cumulative O₃ concentrations, "...the degree and extent of visible foliar injury development varies from year to year and site to site..., even among co-members of a population exposed to similar O₃ levels, due to the influence of co-occurring environmental and genetic factors" (U.S. EPA 2013, section 9.4.2, p. 9-38). Given this, it is not clear to what extent this information informs the selection of an appropriate level.

On the basis of all the considerations described above, including the evidence and exposure/risk analyses, and advice from CASAC, we conclude that a range of W126 index values appropriate for the Administrator to consider extends from 7 to 17 ppm-hrs. In so doing, however, we note, as recognized above, the role of judgments by the Administrator in such decisions. In selecting the range identified here, we primarily consider the evidence- and exposure/risk-based information for cumulative seasonal O₃ exposures represented by W126 index values (including those represented by the WREA average W126 scenarios) associated with biomass loss in studied tree species, both in and outside areas that have been afforded special protections. We note CASAC's advice that a 6% median RBL is unacceptably high, that the 2% median RBL is an important benchmark to consider, that for the lower W126 value of 7 ppm-hrs that the median tree species biomass loss is at or below 2%, and that for the upper value of 17 ppm-hrs the median tree biomass loss is below 6%²⁵. We also note the estimates indicating that a W126 level of 17 ppm-hrs reduces the percent of total nationwide land-area having weighted RBL greater than 2% to 0.2% (Table 6-2) and the number of Class I areas with weighted RBL greater than 2% to 2 of the 145 assessed nationally protected Class I areas.

We also note that tree biomass loss can be an indicator of more significant ecosystem-wide effects which might reasonably be concluded to be significant to public welfare. For example, when it occurs over multiple years at a sufficient magnitude, it is linked to an array of effects on other ecosystem-level processes, such as nutrient and water cycles, changes in above and below ground communities, carbon storage and air pollution removal (U.S. EPA, 2014b, Figure 5-1), that have the potential to be adverse to the public welfare.

Thus, in staff's view, the evidence- and exposure/risk-based information relevant to tree biomass loss and the associated ecosystem services important to the public welfare support

²⁵ We note that a W126 index value of 19 ppm-hrs is estimated to result in a median RBL value of 6%.

consideration of a W126-based secondary standard with index values within the range of 7-17 ppm-hrs. We consider such a range for a potential alternative cumulative seasonal W126-based standard, averaged over three years, based on our analysis of the small effect of year to year variability on the cumulative biomass loss associated with multiple years of exposure, and the benefits of improved stability of the W126 standard when evaluated using the 3-year average form. Lastly, we are mindful of the policy judgments required of the Administrator with regard to the public welfare significance of identified effects and the requisite level of protection, as well as the appropriate weight to assign the range of uncertainties inherent in the evidence and analyses.

While we additionally recognize foliar injury as an important O₃ effect which, depending on severity and spatial extent, most particularly in nationally protected areas such as Class I areas, may reasonably be concluded to be of public welfare significance, we take note of the appreciable variability in this endpoint, as summarized in chapter 5 and section 6.3 above, which poses challenges to giving it primary emphasis in identifying potential alternative standard levels. Similarly, we give less emphasis to consideration of crop yield loss in our consideration of potential standard levels here and in section 6.3 above, noting the median estimates of approximately 5% or lower for W126 index levels at and below 17 ppm-hrs. We also note the range of factors affecting annual crop yields, including those related to the role of management strategies as recognized in sections 5.3 and 6.2 above which complicate the identification of a degree of impact that can be considered adverse to the public welfare.

We further recognize the role of policy judgments by the Administrator, as described above, in identifying a target level of protection for the secondary O₃ standard. For example, to the extent effects associated with cumulative multi-year exposures are judged important to the public welfare, more weight may be placed on such effects, as well as the role that year-to-year exposure variability can play in realizing the potential public welfare impacts.

Lastly, we also conclude that, to the extent the Administrator finds it useful to consider the public welfare protection that might be afforded by a revised primary standard, this is appropriately judged by evaluating how the cumulative seasonal W126-based exposure metric is affected by attainment with such a revised primary standard. For example, comparison of the air quality conditions expected to result from a revised primary standard, with those conditions expressed in terms of W126 exposures, to the W126 levels concluded to provide the desired level of public welfare protection could inform a judgment of whether a secondary standard set identical to a revised primary standard would be expected to achieve the level of public welfare protection concluded to be requisite under the Act. In this type of evaluation, such as through the overlap analyses discussed in section 6.4 above, staff further concludes it is important to take into account associated uncertainties, including those associated with the limited monitor

coverage in many rural areas, including those in the west and southwest and at high elevation sites.

6.7 SUMMARY OF CONCLUSIONS ON THE SECONDARY STANDARD

Staff conclusions are informed by our consideration of the available scientific evidence as assessed in the ISA, the air quality/exposure/risk information in the WREA, advice from CASAC in this review and in prior reviews, and public comment in this review.

Staff conclusions on policy options that are appropriate for the Administrator's consideration in making decisions on the secondary standards for O₃, together with supporting conclusions from sections 5.7 and 6.5 above, are briefly summarized below. In reaching conclusions on alternative standards to provide requisite protection for public welfare effects associated with ambient O₃ exposures, staff has considered these standards in terms of the basic elements of the NAAQS: indicator, form, averaging time, and level. In drawing these conclusions, we are mindful that the Act requires secondary standards to be set so that, in the Administrator's judgment, they are requisite to protect public welfare from known or anticipated adverse effects, such that the standards are to be neither more nor less stringent than necessary. Thus, the Act does not require that NAAQS be set at zero-risk or background levels, but rather at levels that reduce risk sufficiently to protect public welfare from adverse effects.

- (1) Staff concludes, based on the combined consideration of the body of evidence and the results from the quantitative exposure/risk assessment, that the available evidence and exposure/risk information call into question the adequacy of the public welfare protection provided by the current standard and that it is appropriate to consider revising the standard to provide greater public welfare protection.
- (2) With regard to indicator, staff concludes that it is appropriate to continue to use O₃ as the indicator for a standard that is intended to address welfare effects associated with exposure to O₃, alone or in combination with related photochemical oxidants. Based on the available information, staff concludes that there is no basis for considering an alternative indicator at this time.
- (3) With regard to averaging time and form, staff concludes that it is appropriate to consider a revised secondary standard in terms of the cumulative, seasonal, concentration-weighted form, the W126 index. With regard to definition of the W126 index for this purpose, staff makes the additional conclusions:
 - a. It is appropriate to consider the consecutive 3-month period within the O₃ season with the maximum index value as the seasonal period over which

to cumulate hourly O₃ exposures. Staff notes that the maximum 3-month period generally coincides with maximum biological activity for most vegetation, making the 3-month duration a suitable surrogate for longer growing seasons.

- b. It is appropriate to cumulate daily exposures for the 12-hour period from 8:00 am to 8:00 pm, generally representing the daylight period during the 3-month period identified above.
 - c. It is appropriate to consider a form that averages W126 index values across three consecutive years. Staff concludes it is appropriate to consider this form in conjunction with appropriate levels in order to provide the desired degree of public welfare protection from O₃ effects across multiple years.
- (4) With regard to a target level of protection for a revised standard, staff concludes that it is appropriate to give consideration to a range of levels from 17 ppm-hrs to 7 ppm-hrs, expressed in terms of the W126 index averaged across three consecutive years.
- a. To the extent the Administrator finds it useful to consider the extent of public welfare protection that might be afforded by a revised primary standard, staff concludes that public welfare protection is appropriately judged through the use of the cumulative seasonal W126-based metric.

Staff additionally notes that, consideration of the support provided by the information available in this review will depend on public welfare policy judgments by the Administrator regarding the protection of public welfare. This range reflects staff judgment that a standard set within this range could provide an appropriate degree of public welfare protection.

6.8 KEY UNCERTAINTIES AND AREAS FOR FUTURE RESEARCH AND DATA COLLECTION

Staff believes it is important to highlight key uncertainties and recommendations for welfare-related research, including model development and data gathering, associated with secondary standards for O₃. Based on items highlighted in chapter 9 of the ISA, chapters 5 and 6 herein, and CASAC advice, we have identified the following areas for future research and data collection based on key uncertainties, research questions and data gaps that have been highlighted in this review of the secondary standard. The first research area addresses the key uncertainties associated with the extrapolation to plant species and environments outside of specific experimental or field study conditions. The second area of research pertains to the

assessment of the impact of O₃ on other welfare effects categories such as climate, ecosystem components, and whole ecosystem structure and function. A third area of research would support the development of approaches, tools, or methodologies useful in characterizing O₃ exposures in rural, remote, high elevation and/or complex terrain areas and in characterizing ecosystem services and their importance to the public welfare. These three areas are described below.

With regard to the first research area we note that while there have been five decades of research regarding O₃ effects on plants and much information has been compiled in previous reviews, a number of key uncertainties remain. For example, while national visible foliar injury surveys can indicate how widespread O₃ effects may be within the U.S., there remain uncertainties associated with estimating the risk to vegetation of differing amounts of O₃-induced visible foliar injury over the plant's leaf area and the relationship between relative soil moisture and the incidence and severity of foliar injury in sensitive species, as well as the extent to which different degrees of visible foliar injury can impact ecosystem services (e.g., tourism). Research to better characterize the relationship between O₃, soil moisture and foliar injury and to determine if there is an injury threshold or quantifiable relationship between these factors could help inform policy. Additionally, research to understand the connection between O₃-related foliar injury and other physiological effects and ecosystem services could also be useful. We further note that while this review relied on the robust E-R functions that are available for 11 tree and 10 crop species, there are tens of thousands of plant species in the U.S. (USDA, NRCS, 2014),²⁶ 66 of which have also been identified as O₃ sensitive on National Park Service and US Fish and Wildlife Service lands²⁷. Research on additional tree as well as non-tree species that would support the development of robust E-R functions would improve our understanding of the full range of response of plant species to O₃ and our understanding of the overall risk to vegetation. For example, studies using large numbers of native plant species across regions where those species are indigenous, might be expected to reduce uncertainties associated with extrapolating plant response for a given level of O₃ using composite response functions across differing regions and climates. Studies focused on fruits and vegetables might assist in reducing uncertainties associated with O₃ effects on agriculture. Particular focus is suggested on organically grown vegetables that may receive less intensive management than conventionally grown crops. Recent studies indicate that watermelons may be particularly sensitive to O₃

²⁶ USDA, NRCS. 2014. The PLANTS Database (<http://plants.usda.gov>, 3 January 2014). National Plant Data Team, Greensboro, NC 27401-4901 USA.

²⁷See <http://www2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf>

exposure (U.S. EPA, 2013, section 9.4.4.1) and older studies indicate grapes, honeydew melon, lemons and oranges may also be O₃ sensitive (Abt Associates Inc., 1995).

Some new information has emerged linking effects on tree seedlings with larger trees and similarities in results between exposure techniques (U.S. EPA 2013, section 9.6). Uncertainties remain in this area as well as uncertainties in extrapolating from O₃ effects on juvenile to mature trees and from trees grown in the open versus those in a closed forest canopy in a competitive environment. The relationship between nocturnal exposures and plant uptake and response is also an important subject for further research.

With respect to the second research area pertaining to the impact of O₃ on other welfare effects categories such as climate, ecosystem components, and whole ecosystem structure and function, uncertainties that remain in extrapolating individual plant response spatially or to higher levels of biological organization, including ecosystems, could be informed by research that explores and better quantifies the nature of the relationship between O₃, plant response and multiple biotic and abiotic stressors, including those associated with the ecosystem services that would be affected (e.g., hydrology, productivity, carbon sequestration). Because these uncertainties are multiple and significant due to the complex interactions involved, new research will likely require a combination of manipulative experiments with model ecosystems, community and ecosystem studies along natural O₃ gradients, and extensive modeling efforts to project landscape-level, regional, national and international impacts of O₃.

Uncertainties associated with projections of the effects of O₃ on the ecosystem processes of water, carbon, and nutrient cycling, particularly at the stand and community levels might be addressed through research on the effects on below ground ecosystem processes in response to O₃ exposure alone and in combination with other stressors. These below-ground processes include interactions of roots with the soil or microorganisms, effects of O₃ on structural or functional components of soil food webs and potential impacts on plant species diversity, changes in the water use of sensitive trees, and if the sensitive tree species is dominant, potential changes to the hydrologic cycle at the watershed and landscape level. Research on competitive interactions under different O₃ exposures might improve our understanding of how O₃ may affect biodiversity or genetic diversity. Such research could be strengthened by modern molecular methods to quantify impacts on diversity. More tools and research would improve our understanding of relationships between O₃ exposure and stressors such as insect infestations, plant diseases, drought and potential stressors from climate change. It is also important to understand how such interactions may affect ecosystem services such as CO₂ sequestration; food and fiber production; wildlife habitat and water resources.

With respect to the research areas related to the development of approaches, tools, or methodologies useful in characterizing O₃ exposures and the relationship between O₃-induced

effects and associated ecosystem services and public welfare in a policy context, we note that one of the most important uncertainties in this review is the characterization of air quality in rural areas where there is limited monitoring. More comprehensive monitoring in these areas would reduce uncertainties associated with O₃ exposures in many rural areas. Areas of particular uncertainty include protected natural areas in the western U.S, including those at high elevation, as well as those downwind of recently expanded oil and gas development areas. Uncertainties associated with quantifying exposure in areas with and without monitors might be addressed through additional work on interpolation methods and air quality models that are tailored to estimating cumulative seasonal exposures, as well as improved model capabilities that use more refined spatial grids and are better able to handle O₃ movement in complex terrain.

Uncertainties related to characterizing the potential public welfare significance of O₃-induced effects and impacts to associated ecosystem services could also be informed by research, such as research intended to clarify the relationship between O₃ exposure and fire risk and O₃ exposure and forest susceptibility to bark beetle infestation. Research relating known O₃ ecological effects such as reproductive effects to effects on production of non-timber forest products and research to characterize public preferences including valuation related to non-use and recreation for foliar injury could also help inform consideration of the public welfare significance of these effects.

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APPENDIX 2A

SUPPLEMENTAL AIR QUALITY MODELING ANALYSES OF BACKGROUND OZONE

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1. Introduction

One of the aspects of ozone that is unusual relative to the other pollutants with National Ambient Air Quality Standards (NAAQS) is that, periodically, in some locations, an appreciable fraction of the observed ozone results from sources or processes other than local and regional anthropogenic emissions of ozone precursors (Fiore *et al.*, 2002). Any ozone formed by processes other than the chemical conversion of local or regional ozone precursor emissions, such as nitrogen oxides (NO_x) or volatile organic emissions (VOC), is generically referred to as “background” ozone. As part of this review of the ozone NAAQS, EPA completed an extensive review of the known aspects of background ozone and summarized the findings in the Integrated Science Assessment (ISA) in March 2013 (USEPA, 2013). The purpose of this appendix is to present the results from supplemental air quality modeling analyses related to background ozone that were completed by EPA subsequent to the ISA. While these updated analyses use a recent base year (2007) and consider an alternative modeling methodology which can better account for non-linear ozone chemistry in some conditions, the results are largely consistent with previous determinations about the magnitude of background ozone contributions across the U.S.

Away from the surface, ozone can have an atmospheric lifetime on the order of weeks. As a result, background ozone can be transported long distances at heights above the boundary layer and, when meteorological conditions are favorable, be available to mix down to the surface and add to the total ozone loading from non-background sources. Generically, background ozone can originate from natural sources of ozone and ozone precursors, as well as from far upwind manmade emissions of ozone precursors. Natural sources of ozone precursor emissions such as wildfires, lightning, and vegetation can lead to ozone formation by chemical reactions with other natural sources¹. Another important natural component of background is ozone that is naturally formed in the stratosphere through interactions of UV light with atomic oxygen (O₂). Stratospheric ozone can periodically mix down to the surface at high concentrations, especially at higher altitude locations. The manmade portion of the background includes any ozone formed due to anthropogenic sources of ozone precursors emitted far away from the local area (e.g., international emissions). Finally, both biogenic and international anthropogenic emissions of methane, which can be chemically converted to ozone over relatively long time scales, can also contribute to global background ozone levels.

The precise definition of background ozone can vary depending upon context, but it generally refers to ozone that is formed by sources or processes that cannot be influenced by actions within the jurisdiction of concern. In the first draft policy assessment document (EPA, 2012), EPA presented three specific definitions of background ozone: natural background, North American background, and U.S. background. Natural background (NB) was the narrowest definition of background and it was defined as the ozone that would exist in the absence of any manmade ozone precursor emissions. The other two previously-established definitions of background presume that the U.S. has little influence over anthropogenic emissions outside our continental or domestic borders. North American background (NAB) is defined as that ozone that would exist in the absence of any manmade ozone precursor

¹ Ozone formed through reactions between natural emissions and local anthropogenic emissions (e.g., biogenic VOC with man-made NO_x) is generally not considered to be background ozone.

emissions inside of North America. U.S. background (USB) is defined as that ozone that would exist in the absence of any manmade emissions inside the United States. It is important to note that **each of these three definitions of background ozone requires photochemical modeling simulations** to estimate what the residual ozone concentrations would be were the various anthropogenic emissions to be removed.

As noted in the first draft policy assessment, EPA has revised several aspects of our methodology for estimating the change in health risk and exposure that would result from a revision to the ozone NAAQS. First, risk estimates are now based on total ozone concentrations as opposed previous reviews which only considered risk above background levels. Second, EPA is now using air quality models to estimate the spatial patterns of ozone that would result from attaining various levels of the NAAQS, as opposed to simplistic rollback techniques that required the estimation of a background ozone “floor” beyond which the rollback would not take place. Both of these revisions have had the indirect effect of obviating the need for estimating background ozone levels as part of the ozone risk and exposure assessment (REA). Regardless, EPA expects that a well-founded understanding of the fractional contribution of background sources and processes to surface ozone levels will be valuable towards informing policy decisions about the NAAQS. Section 2 of this document will describe the supplemental air quality modeling simulations that have recently been completed by EPA to bolster our understanding of background ozone. Section 3 will present the results from the updated analyses and provide estimates of average background ozone levels, and how they can vary in time and space across the U.S. Based on the same modeling, Section 4 will consider the entire spectrum of variable background ozone levels with special emphasis on areas and times in which background can approach or exceed the level of the NAAQS. Section 5 will utilize the supplemental air quality modeling estimates to determine the relative importance of specific components of background ozone. Section 6 will present estimates of the overall fraction of ozone that is estimated to result from background sources or processes in each of the 12 urban case study areas in the epidemiology study based analyses in Chapter 7 of the Risk and Exposure Assessment (REA) (EPA, 2014) based on the updated modeling. Finally, Section 7 will conclude with a limited analysis of how background ozone levels impact longer-term ozone metrics that may be important from a welfare perspective (i.e., W126).

2. Description of modeling methodologies

As noted above, air quality models are typically used to estimate background ozone as it is quite difficult to measure directly. Without special monitoring, it is impossible to determine how much of the ozone measured by a monitor originated from sources that are considered background. Even the most remote monitors within the U.S. can periodically be affected by U.S. anthropogenic emissions. Previous modeling studies have estimated what background levels would be in the absence of certain sets of emissions by simply comparing the ozone differences between a base model simulation and a control simulation in which emissions were removed. This basic approach is often referred to as “zero out” modeling or “emissions perturbation” modeling. Examples of zero out modeling include the three major studies summarized in the ISA (Zhang et al., 2011; Emery et al., 2012, Lin et al., 2013). It is important to note that the specific concepts of NB, NAB, and USB are all explicitly tied to zero-out modeling, as those definitions are based on estimating what remains *in the absence of* specific sets of man-made emissions.

EPA has conducted and will describe updated air quality modeling for a 2007 base year that employs a regional air quality model nested within a coarser-scale global chemical transport model to estimate NB, NAB, and USB levels when the respective manmade emissions are zeroed. This modeling is described in detail in section 2a.

While the zero-out approach has traditionally been used to estimate background ozone levels, the methodology has some acknowledged limitations. First, from a policy perspective, the purely hypothetical and ultimately unrealizable zero manmade emissions scenarios have limited application in this regard. Secondly, the assumption that background ozone is what is left after specific emissions have been removed within the model simulation can be misleading in locations where ozone chemistry is highly non-linear. Depending upon the local composition of ozone precursors, NO_x emissions reductions can either increase or decrease ozone levels in the immediate vicinity of those reductions. For those specific urban areas in which NO_x titration of ozone can be significant, zero-out modeling can result in inflated estimates of background ozone when these NO_x emissions are completely and unrealistically removed. Paradoxically, in certain times and locations in a zero-out scenario there can be more background ozone than actual ozone within the model (EPA, 2014).

A separate modeling technique attempts to circumvent these limitations by apportioning the total ozone within the model to its contributing source terms. This basic approach is referred to as “source apportionment” modeling. While source apportionment modeling has not been previously used in the context of estimating background ozone levels as part of an ozone NAAQS review, it has frequently been used in other regulatory settings to estimate the “contribution” to ozone of certain sets of emissions (EPA 2005, EPA 2011). The source apportionment technique provides a means of estimating the contributions of user-identified source categories to ozone formation in a single model simulation. This is achieved by using multiple tracer species to track the fate of ozone precursor emissions (VOC and NO_x) and the ozone formation caused by these emissions. The methodology is designed so that all ozone and precursor concentrations are attributed to the selected source categories at all times without perturbing the inherent chemistry. The zero out modeling attempts to determine what ozone be in the absence of background sources. The source apportionment modeling attempts to determine how much of the modeled ozone has resulted from background sources. EPA has conducted and will describe new source apportionment modeling that employs a regional air quality model nested within a coarser-scale global chemical transport model to assess the contributions of boundary conditions and other potential background sources (e.g., wildfires, biogenic emissions, and Canadian/Mexican emissions). This modeling is described in detail in section 2b.

a. 2007 GEOS-Chem/CMAQ zero-out modeling:

In order to provide estimates of the overall fraction of ozone that is estimated to result from background sources in each of the 12 REA urban study areas, EPA conducted new modeling that utilized the same model base year (2007) as was used in the ozone modeling that inform the risk and exposure analyses (EPA, 2014, Appendix 4b). The EPA modeling used a model configuration similar to that of Emery (2012), in that it nested a regional-scale (12 km) air quality model inside a global air quality model

simulation with a much coarser horizontal grid resolution (2.0 by 2.5 degrees). Figure 1a shows a map of the model domain.

The global scale simulation utilized the GEOS-Chem model, version v8-03-02, except for the chemistry package which was from version v8-02-01. The emissions estimates used in the 2007 base year modeling were aggregated from a variety of sources, starting with the global Emissions Database for Global Atmospheric Research (EDGAR) emission inventory. These initial estimates were then improved by utilizing various area-specific inventories, such as the 2005 National Emissions Inventory (NEI) for the U.S. portions of the domain, and available inventories for Asia, Canada, Europe, and Mexico. In addition to the anthropogenic estimates, emissions were specified for a variety of background sources including: lightning NO, soil NO_x, wildfires, and biogenic VOC emissions. The wildfire data is from the Global Fire Emissions Database (GFED). The biogenic VOC estimates were simulated by the Model of Emissions of Gases and Aerosols from Nature (MEGAN) version 2.1. The meteorological data is based on the Goddard Earth Observing System Model, Version 5 (GEOS-5) analysis fields. More information on the global simulation is available within Henderson *et al.* (2013). This reference also provides a broad evaluation of the ability of this specific GEOS-Chem configuration to provide accurate lateral boundary conditions of ozone to finer-scale regional simulations. Using satellite retrievals from the Tropospheric Emissions Spectrometer (TES), Henderson *et al.* (2013) concluded that the GEOS-Chem ozone prediction biases and errors are generally within TES uncertainty estimates. For instance, for the ozone season month of August, model predictions are within plus or minus 20 percent of the satellite estimates between nearly 80 percent of the time, with slightly better performance along the southern boundary.

The lateral boundary conditions from the global model were then used as inputs for a 12 km horizontal resolution, CMAQ version 4.7.1, model simulation. Four scenarios were modeled: 1) a 2007 base case simulation which was the basis of the air quality modeling performed for the 2nd draft ozone REA and is described in more detail in Appendix 4b of EPA (2014), 2) a natural background run with anthropogenic ozone precursor emissions² removed in both the global and regional models, 3) a North American background run with anthropogenic ozone precursor emissions removed across North America (global and regional model simulations), and 4) a U.S. background run with anthropogenic ozone precursor emissions removed over the U.S (global and regional model simulations). Detailed analyses of EPA's 2007 zero out modeling results are provided in sections 3 through 6 of this appendix.

An operational model performance evaluation was completed for surface ozone in the 2007 base simulation as described separately (EPA 2014, Appendix 4b). For the purposes of this analysis, EPA assessed the model ability to reproduce measured daily maximum 8-hour (MDA8) ozone values and seasonal mean MDA8 ozone concentrations for the period April to October 2007. As noted earlier, the base year modeling in this analysis used climatological monthly-average wildfire emissions which are not

² In the global model all ozone precursor species were removed (i.e., VOC, NO_x, CO), except for methane which was reset to pre-industrial levels to reflect natural contributions. In the regional modeling, the methane levels were left unchanged.

intended to capture discrete events from specific fires that occurred in 2007, so perfect correlation between observations and model predictions should not be expected. Figure 1b provides a density scatterplot of the observed and predicted daily 8-hour ozone peaks paired in space and time for the 2007 CMAQ base. As can be seen, the majority of pairs line up along the 1:1 line. There is a tendency for the model to overestimate site-days with low 8-hour ozone peaks, and underestimate the site-days with higher peak ozone values. Modeled 8-hour ozone peak concentrations exhibited relatively small bias and error compared to the observations. The average bias in MDA8 ozone estimates was 3.5 ppb. Figure 1c depicts the spatial bias patterns in MDA8 ozone at all sites that measured valid ozone data for at least 100 days during the April-October period. CMAQ overestimations are greatest along the Gulf Coast region, along the Atlantic coastline, and over the central U.S. The majority of underestimated seasonal mean MDA8 occurs within southern California. The model performance for the 2007 base simulation is equivalent or better than typical state-of-the-science photochemical model performance recently reported in the literature (Simon et al, 2012).

Certainly some remote monitoring locations are more affected by background sources than other locations in the network. However, this and numerous other analyses have shown that even the most remote ozone monitoring locations in the U.S. are periodically affected by U.S. manmade emissions. In this analysis we carefully assess model performance to ensure that model error does not influence the characterization of background ozone. As noted in the recent ISA (EPA, 2013), there is greater confidence in the ability of the model to predict mean contributions from background sources rather than individual events. Beyond the statistical analyses summarized in the previous paragraph and in appendix 4b of the 2nd draft ozone REA (EPA, 2014), it is valuable to attempt to diagnose the model ability to account for background ozone within the simulation. EPA assessed whether any correlation existed between daily model biases and daily background ozone estimates. Figure 1d shows that at high-elevation sites (i.e., sites more than 1km above sea level) the highest estimates of natural background ozone tend occur on days with greatest overestimation. Conversely, the site-days with the lowest natural background estimates tend to occur when the model underestimates the observed daily peaks at these sites. This relationship between background estimates and simulation bias appears to be constrained to the mountainous portion of the Western U.S. Figure 1d also shows that estimates of natural background ozone greater than 60 ppb are associated with large over-predictions. However, based on the relatively low model bias and the general lack of correlation between daily bias values and background estimates, EPA believes that these model estimates can be used to help characterize background ozone levels over the U.S. Although the highest background estimates should be considered with caution.

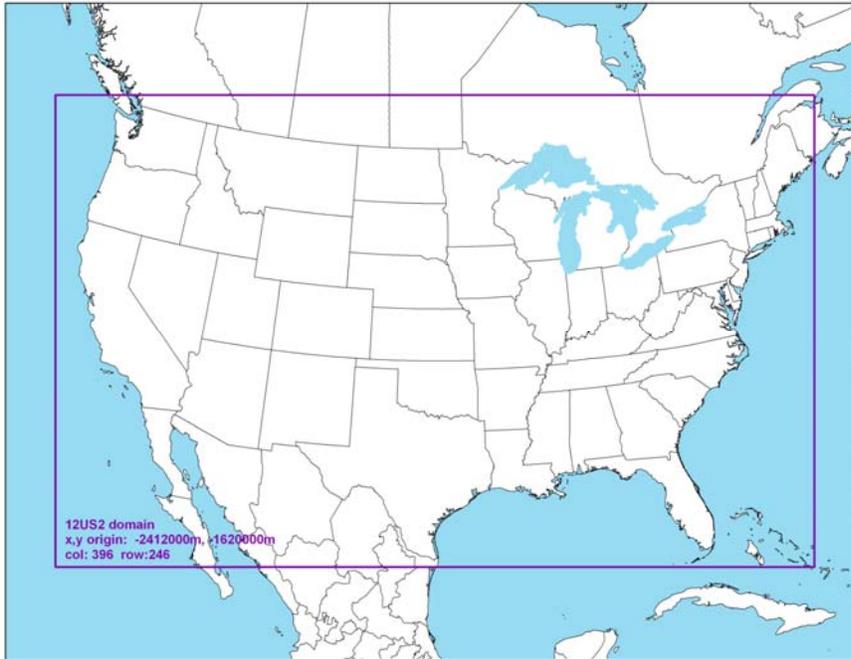


Figure 1a. Modeling domain used in 2007 CMAQ and CAMx modeling.

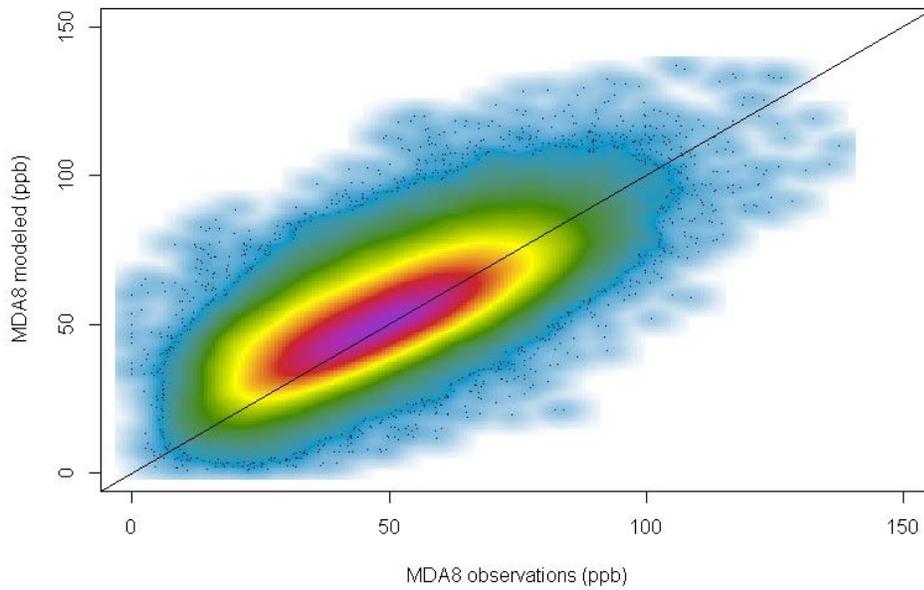


Figure 1b. Density scatterplot comparing CMAQ base daily peak 8-hour ozone predictions against observed 8-hour ozone peaks paired in space and time for all sites during April-October 2007.

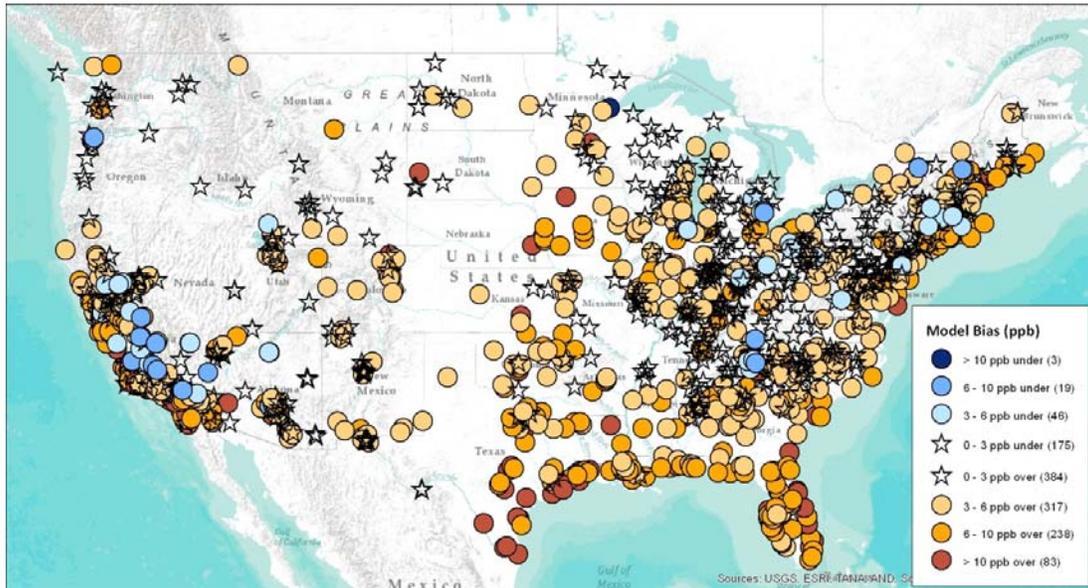


Figure 1c. Bias in seasonal mean (April-October) maximum daily 8-hour ozone predictions in the 2007 CMAQ base simulation.

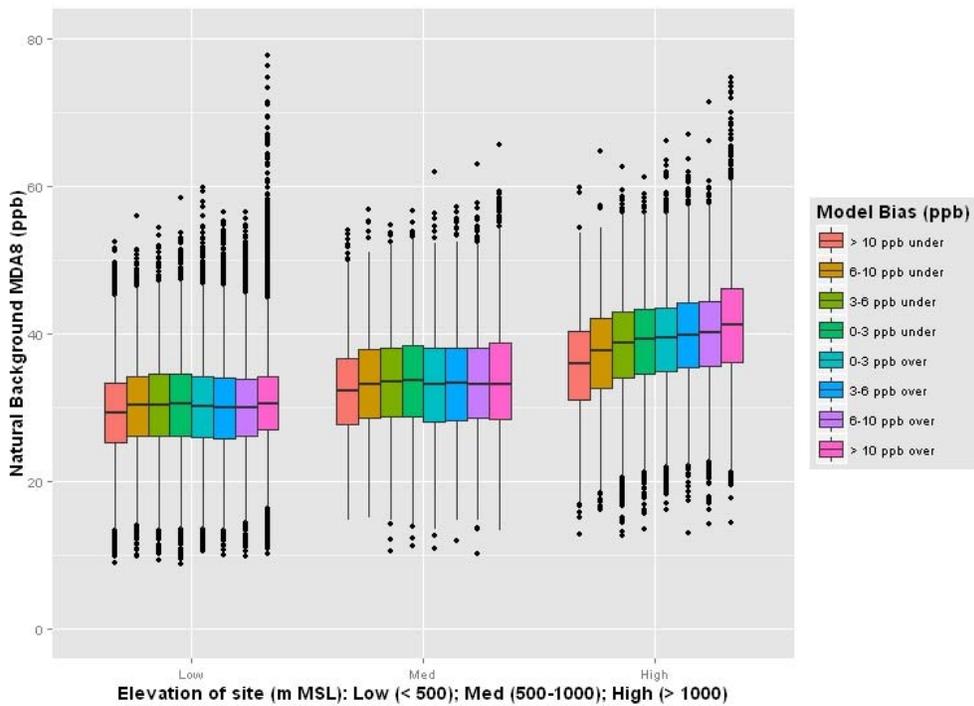


Figure 1d. Relationship between CMAQ estimations of MDA8 natural background ozone and daily model biases.

b. 2007 GEOS-Chem/CAMx source apportionment modeling:

The same global modeling described above was used to assign lateral boundary conditions to the regional-scale (12 km) CAMx v5.0 source apportionment simulations. Wherever possible, the emissions and meteorological inputs in the CAMx modeling were chosen to mimic the 2007 base CMAQ simulation described earlier. Figure 1a shows a map of the model domain.

As with the CMAQ base case, a limited operational model performance evaluation was also completed for surface ozone in the 2007 base simulation. For the purposes of this analysis, EPA assessed the model ability to reproduce measured daily maximum 8-hour (MDA8) ozone values and seasonal mean MDA8 ozone concentrations for the period April to October 2007. Figure 2a provides a density scatterplot of the observed and predicted daily 8-hour ozone peaks paired in space and time for the 2007 CAMx base simulation. As can be seen, the majority of pairs line up along the 1:1 line. Again, there is a tendency for the model to overestimate site-days with low 8-hour ozone peaks and underestimate the site-days with higher peak ozone values. Modeled 8-hour ozone peak concentrations exhibited relatively small bias and error compared to the observations. The average bias in MDA8 ozone estimates was 3.5 ppb. Figure 2b depicts the spatial bias patterns in MDA8 ozone at all sites that measured valid ozone data for at least 100 days during the April-October period. CAMx overestimations are greatest along the Gulf Coast region, along the Atlantic and Pacific coastlines, and within the southeastern U.S. The majority of underestimated seasonal mean MDA8 occurs in California away from the coastline.

The apportionment tools in CAMx utilized here to estimate the contribution of background sources are well-established and have previously been peer-reviewed (UNC, 2009). EPA used the Anthropogenic Precursor Culpability Assessment (APCA) tool in this analysis. The APCA tool attributes ozone production to manmade sources whenever ozone is determined to result from a combination of anthropogenic and biogenic emissions (Environ, 2011). The APCA methodology defines natural ozone as the production resulting from the interaction of biogenic VOC with biogenic NO_x emissions. Eleven separate source categories were tracked in the source apportionment analysis, including five boundary condition terms and six in-domain sectors:

- Boundary condition terms:
 - Northern edge
 - Eastern edge
 - Southern edge
 - Western edge
 - Top boundary

- In-domain sectors:
 - U.S. anthropogenic emissions
 - Point sources located within the Gulf of Mexico
 - Category 3 marine vessels outside State boundaries
 - Climatologically-averaged wildfire emissions

- Biogenic emissions
- Canada/Mexico emissions (only those sources within the domain)

It should be noted that the source apportionment modeling conducted here does not allow for replication of natural background because of the construct of boundary conditions. The boundary conditions for our applications can include ozone and/or ozone precursors that were originally generated by natural sources, as well as ozone produced from far upstream anthropogenic emissions (e.g., Asia). It is not possible to disentangle these two terms. Instead, the source apportionment modeling is primarily used to help estimate background into the U.S., which is assumed to be the contributions from nine of the modeled sectors; that is, everything except U.S. anthropogenic emissions and point sources located within the Gulf of Mexico.

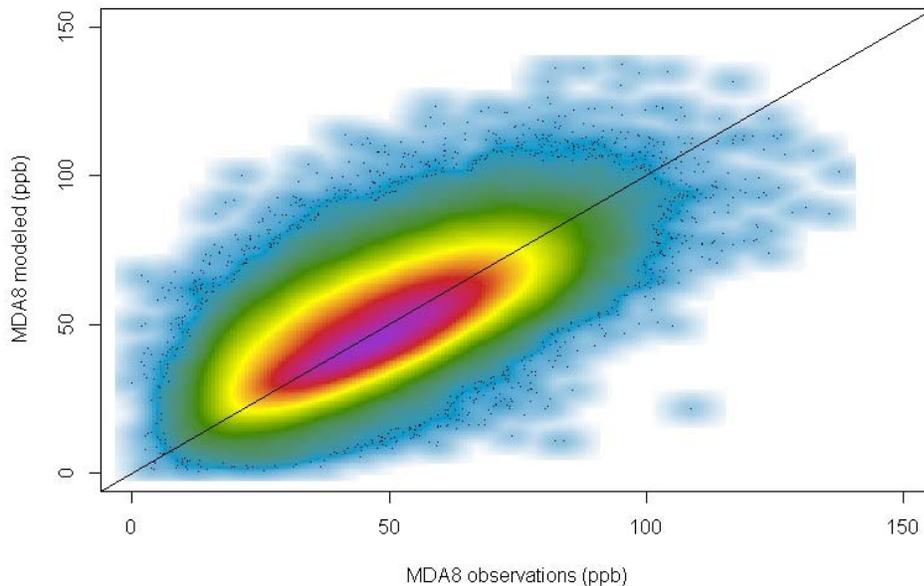


Figure 2a. Density scatterplot comparing CAMx base daily peak 8-hour ozone predictions against observed 8-hour ozone peaks paired in space and time for all sites during April-October 2007.

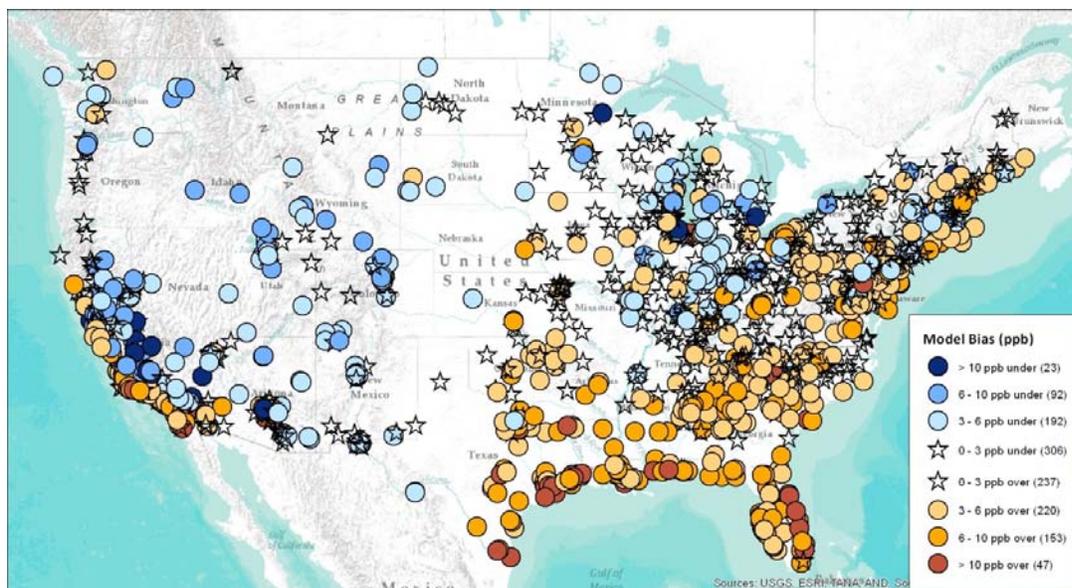


Figure 2b. Bias in seasonal mean (April-October) maximum daily 8-hour ozone predictions in the 2007 CAMx base simulation.

3. Estimates of seasonal-average background ozone levels

This section of the appendix provides estimates of seasonal average background ozone levels over the U.S. As noted in the introduction and as discussed in detail in the ISA, background ozone values can vary significantly in space and time. There can be atypical episodes of higher background ozone concentrations amidst the routine days that drive seasonal average background. The highest background episodic concentrations are typically associated with stratospheric intrusions or wildfires. These background “events” can be difficult to model as they require event-specific model inputs. The primary goal of the EPA modeling was to estimate the seasonal average background concentrations between April and October 2007. Previous analyses have shown that this is the period in which average background levels are highest (Zhang et al., 2011). This section of the appendix focuses on seasonal mean levels of background. (Section 4 will consider the upper range of possible background ozone.)

The analysis focus on the maximum daily 8-hour ozone average in ppb. This metric is referred to as MDA8. This section will first present model estimates of seasonal mean ozone levels in the base simulation. This will be followed by estimates of NB, NAB, and USB from the CMAQ zero out modeling. After discussing the magnitudes of background levels, the section switches to a consideration of the relative percentage of background to total ozone across the U.S. This portion of the text will utilize both the CMAQ zero out and CAMx source apportionment modeling.

Figure 3a displays the 2007 base case, CMAQ model-predicted, seasonal mean (April-October) MDA8 ozone concentrations in grid cells with active monitoring locations over the U.S. The model results are shown at the monitoring site level as opposed to in the default gridded format to foster subsequent site-level estimates of background magnitudes. Each grid cell containing an Air Quality System (AQS) ozone monitor that was collecting valid data in 2007 was identified and the model

background estimates were extracted for those grid cells and displayed accordingly. The base predictions are provided for context to allow easier interpretation of the following plots which isolate specific background levels. As can be seen, most of the U.S. experiences seasonal mean MDA8 ozone levels greater than 50 ppb in the base case simulation. The median value over the 1,294 monitoring locations is 52.5 ppb.

Figure 3b provides an estimate of what seasonal-average MDA8 would be in a natural background scenario, using the 2007 EPA zero out modeling. Again, in this GEOS-Chem/CMAQ simulation, all anthropogenic ozone precursor emissions were removed from both the global and regional simulations, and methane levels were adjusted to pre-industrial levels in the global simulation. As shown, natural background ozone levels range from approximately 15-35 ppb with the highest values occurring over the higher-elevation sites in the western U.S. The median value over these locations is 24.2 ppb, and more than 50 percent of the sites have natural background levels of 20-25 ppb. The highest modeled estimate of seasonal average, natural background, MDA8 ozone is 34.3 ppb at the high-elevation CASTNET site (Gothic) in Gunnison County, CO.

Figures 3c and 3d show the same information for the North American and U.S. background scenarios. In these model runs, all anthropogenic ozone precursor emissions were removed from the U.S., Canada, and Mexico (NAB scenario) and then only the U.S. (USB scenario). The figures show that there is not a large difference between the NAB and USB scenarios. Seasonal mean MDA8 NAB and USB ozone levels range from 25-50 ppb, with the most frequent values estimated in the 30-35 ppb bin. The median seasonal mean background levels are 31.5 and 32.7 ppb (NAB and USB, respectively). Again, the highest levels of background are predicted over the intermountain western U.S. Locations with NAB and USB concentrations greater than 40 ppb are confined to Colorado, Nevada, Utah, Wyoming, northern Arizona, eastern California, and parts of New Mexico. Similar to NB, the highest NAB and USB levels were modeled to occur at the Gothic CO site (46.7/47.7). This remote rural site is located 2,926 meters (9,600 feet) above mean sea level and should not be considered representative of background ozone at lower-altitude, more-populated regions. The high USB and NAB values along the Gulf Coast are most likely due to model biases.

Absolute model estimates of various background definitions are useful, but they can be influenced by any local biases and errors in the modeling. A separate way to look at the role of background in seasonal mean ozone levels is to consider the fractional contribution of NB, NAB, and USB to total ozone at each location. Considering the proportional role of background allows for an informative comparison between the two modeling approaches without having to account for the differences in base case biases and errors. Figures 4a, 4b, and 4c show the estimated fractional contribution of NB, NAB, and USB to total seasonal average MDA8 ozone levels at the monitoring locations from the CMAQ zero out modeling. The modeling estimates that approximately 35-80 percent of the seasonal average MDA8 ozone at monitoring locations is due to natural background sources. A majority sites have NB fractions between 40 and 60 percent. The mean natural background proportion over all sites is 47 percent. That is, when all global anthropogenic emissions are removed and global methane levels in GEOS-Chem are restored to pre-industrial levels, seasonal average MDA8 levels are reduced by approximately half. The fractional proportions of NAB and USB are very similar. In both

cases, most sites have background fractions that range from 50 to 80 percent. The mean NAB fraction (to seasonal mean MDA8) is 63 percent. The mean USB fraction is 66 percent.

As noted in the introduction, the advantage of the source apportionment modeling is that all of the modeled ozone is attributed to various source terms and thus this approach is not affected by the confounding occurrences of background ozone values exceeding the base ozone values as can happen in the zero out modeling (i.e., background proportions > 100%). Consequently, one would expect the fractional background levels to be lower in the source apportionment methodology as a result of removing this artifact. It is also important to remember that the terms NB, NAB, and USB are explicitly linked to the zero out modeling approach. (USB is the ozone that would exist in the absence of U.S. anthropogenic emissions.) In contrast, the source apportionment modeling performed here provides estimates the amount of MDA8 ozone that is attributable to U.S. anthropogenic emissions relative to total base model ozone. Figure 4d shows the relative contribution from ***sources other than U.S. anthropogenic emissions*** to total seasonal mean MDA8 ozone based on the 2007 source apportionment modeling. The fractional contribution fields between CMAQ zero out USB estimates and CAMx source apportionment estimates of source other than U.S. anthropogenic emissions are quite similar. The spatial patterns in Figures 4c and 4d are consistent, with the highest fractional contributions from sources other than U.S. anthropogenic emissions occurring along U.S. borders and over the intermountain western States. The source apportionment modeling estimates that approximately 40-80% of the seasonal average MDA8 ozone at monitoring locations is due to sources other than manmade ozone precursor emissions from the U.S. A majority of sites have non-U.S. fractions between 40 and 70 percent. The mean proportion attributable to international and natural sources over all sites is 59 percent. Despite the differences in the methodologies this is very similar to the mean USB estimate of 66 percent from the zero out modeling.

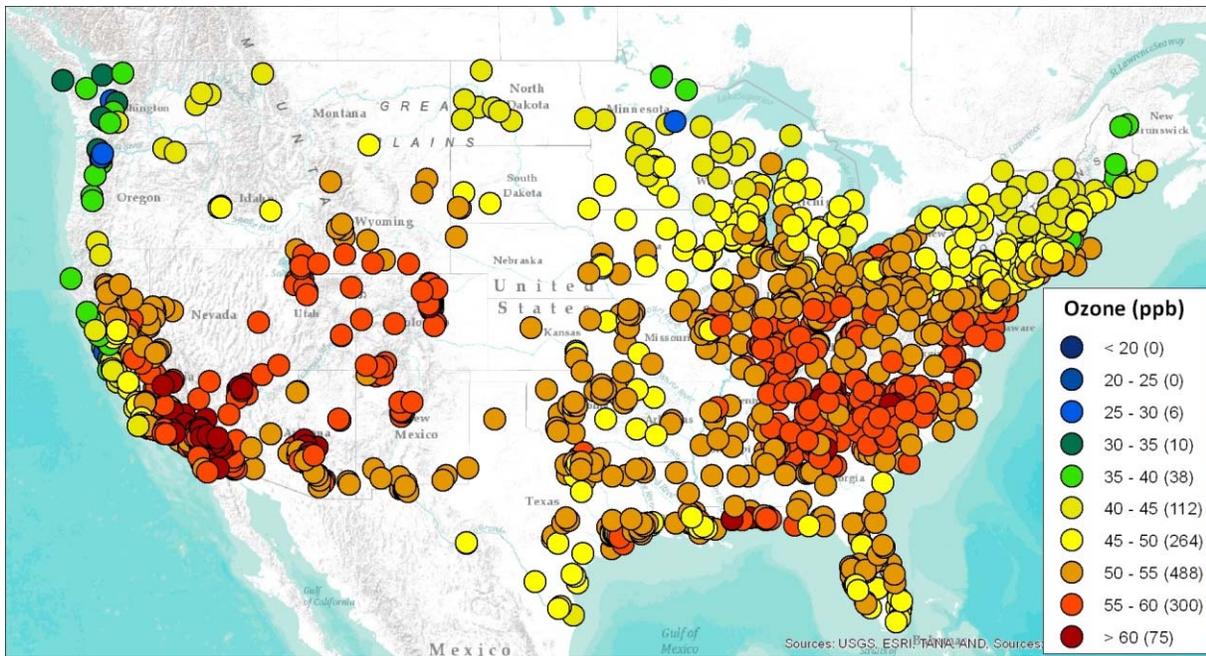


Figure 3a. April-October average MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ base simulation.

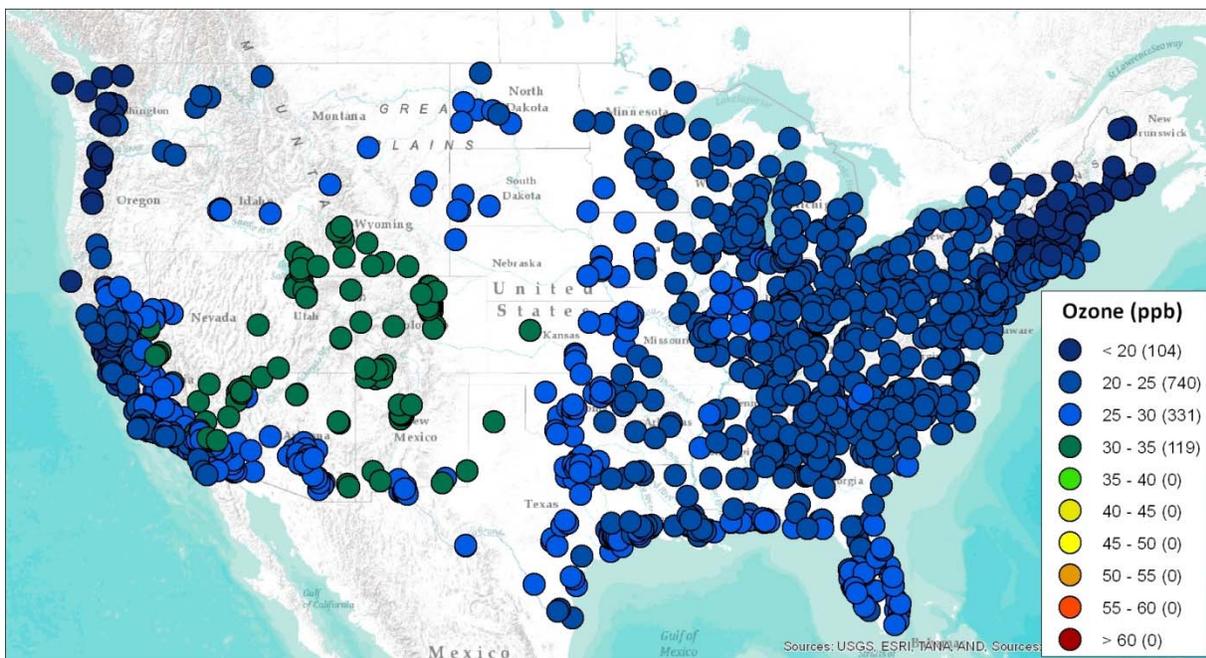


Figure 3b. April-October average *natural background* MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ zero out simulation.

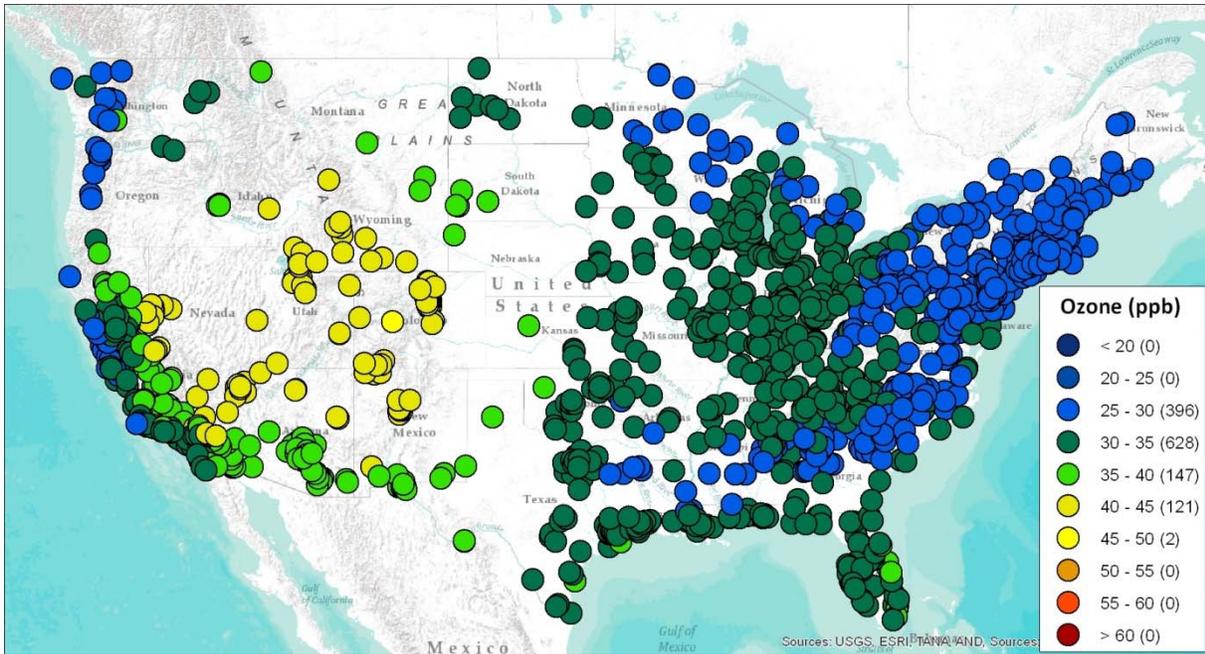


Figure 3c. April-October average North American background MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ zero out simulation.

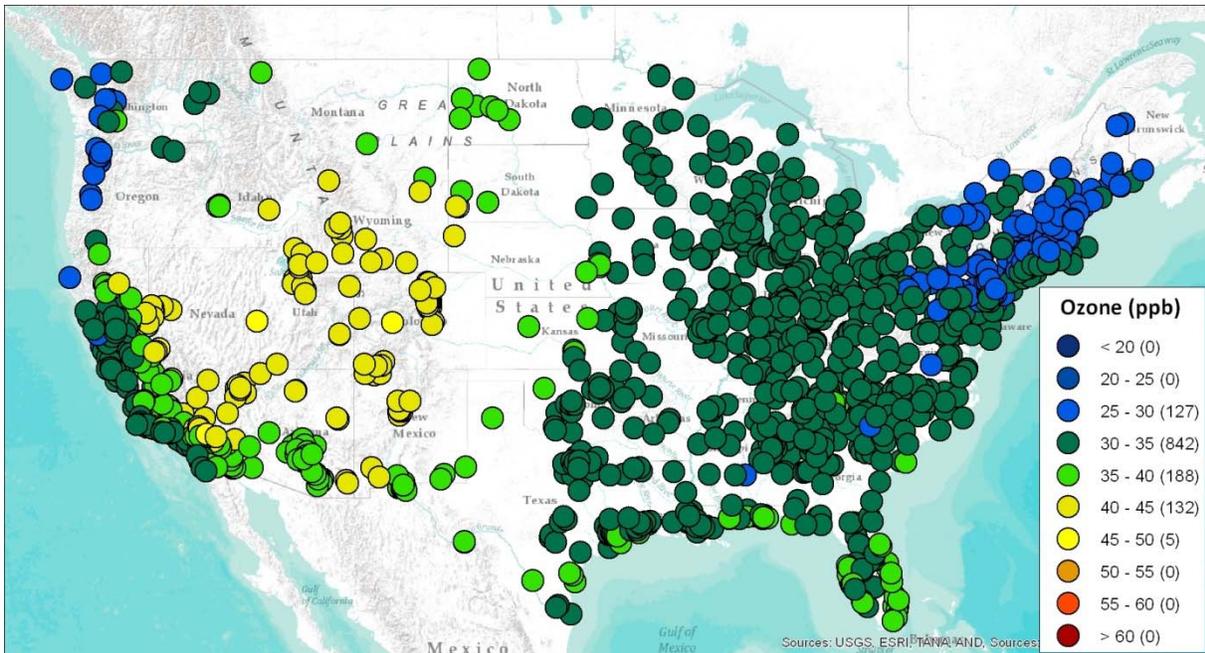


Figure 3d. April-October average United States background MDA8 ozone (ppb) at monitoring locations across the U.S. as estimated by a 2007 CMAQ zero out simulation.

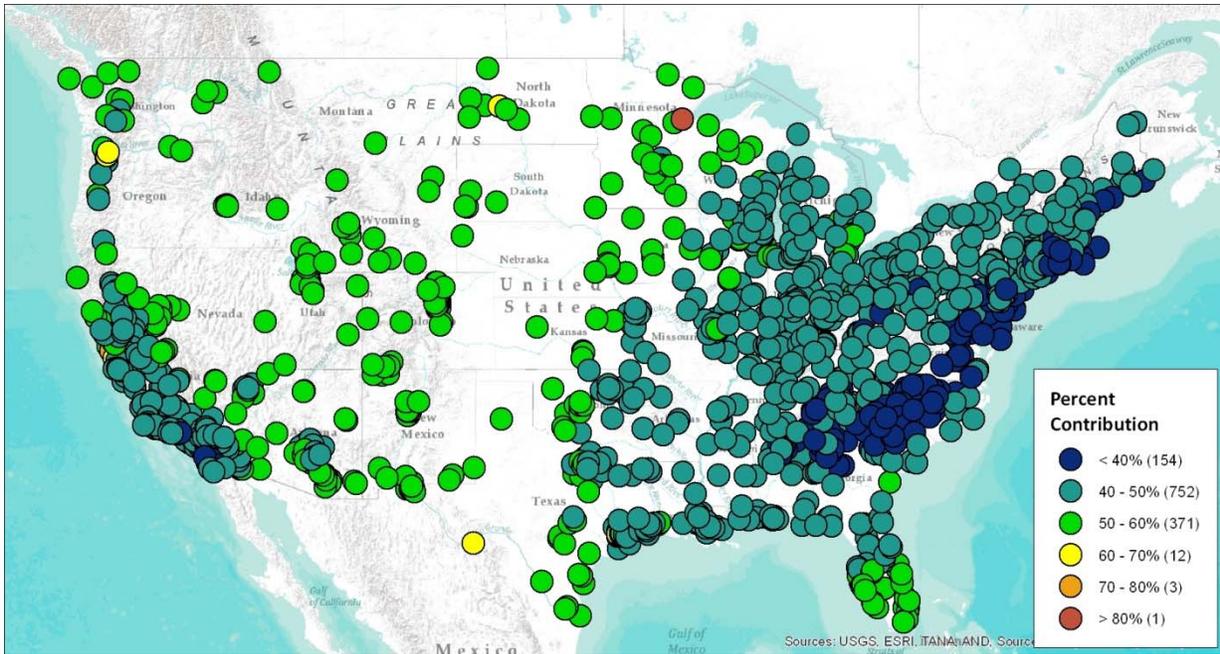


Figure 4a. Ratio of *natural background* to total April-October average MDA8 ozone at monitoring locations across the U.S. as estimated based on 2007 CMAQ simulations.

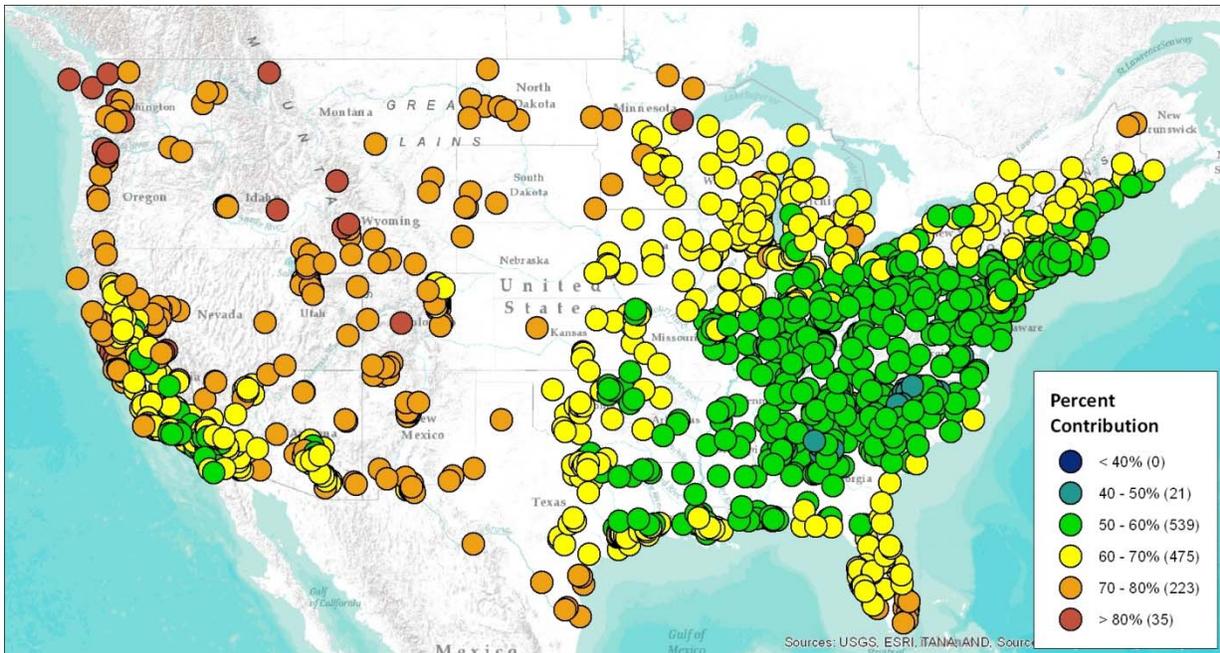


Figure 4b. Ratio of *N. American background* to total April-October average MDA8 ozone at monitoring locations across the U.S. as estimated based on 2007 CMAQ simulations.

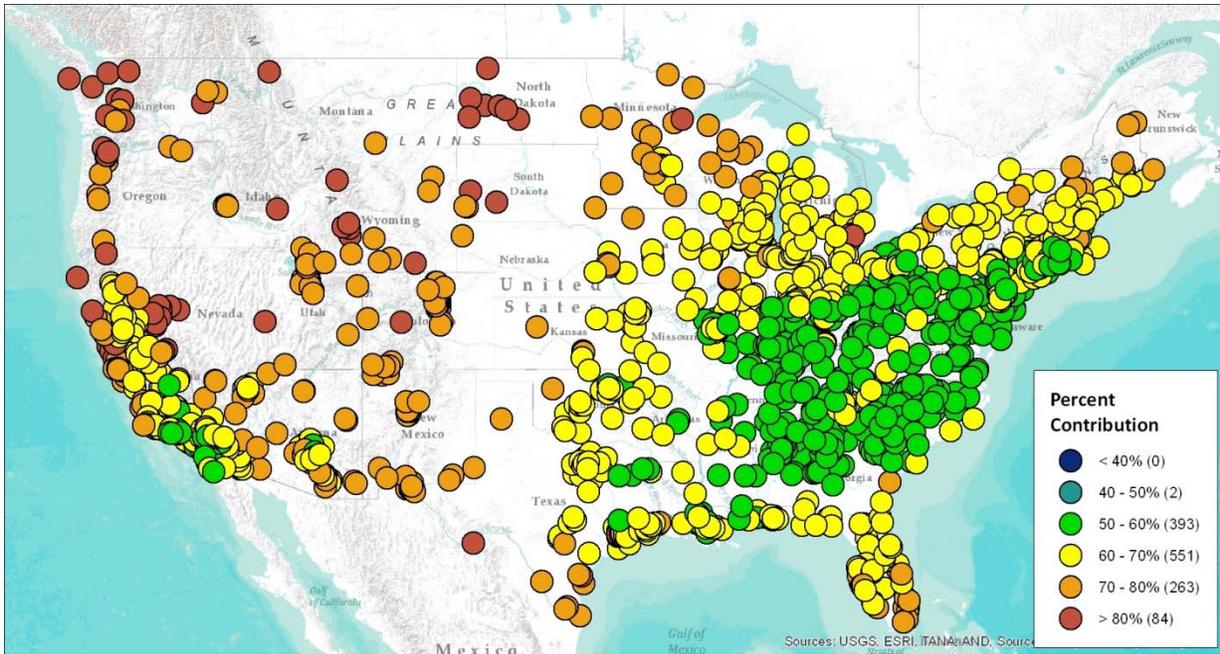


Figure 4c. Ratio of *U.S. background* to total April-October average MDA8 ozone at monitoring locations across the U.S. as estimated based on 2007 CMAQ simulations.

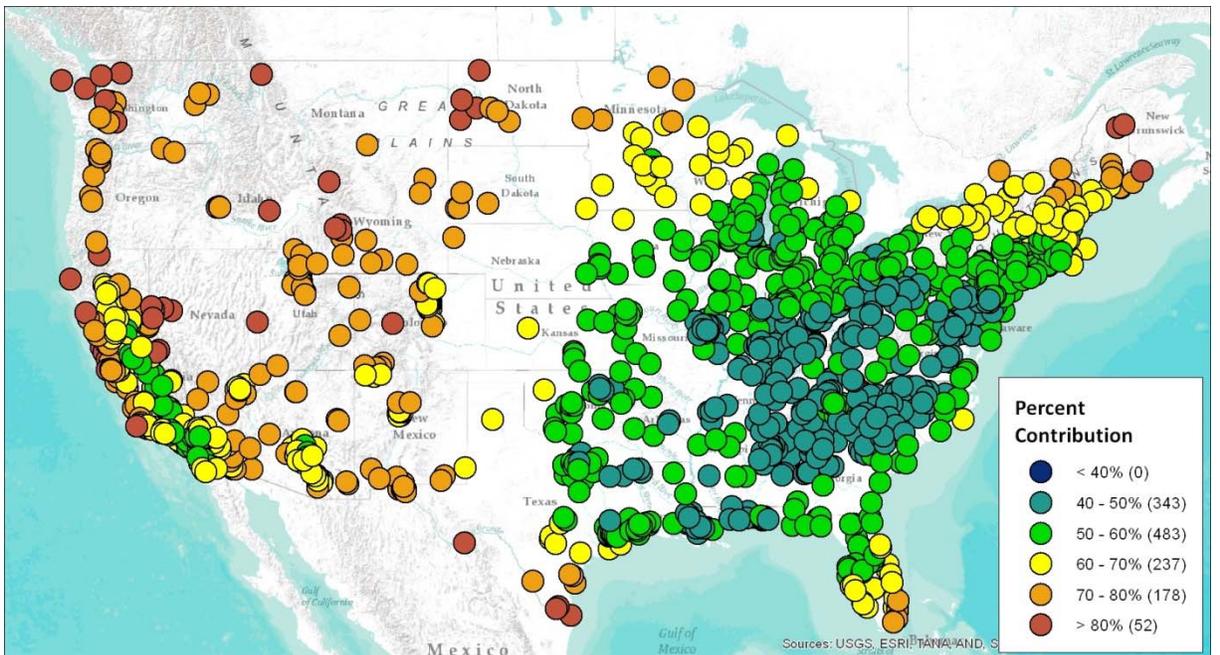


Figure 4d. Ratio of *sources other than U.S. anthropogenic emissions* to total April-October average MDA8 ozone at monitoring locations across the U.S. as estimated by a 2007 CAMx source apportionment simulation.

4. Distributions of background ozone levels

As a first-order understanding, it is valuable to be able to characterize seasonal mean levels of background ozone. However, it is well established that background levels can vary substantially from day-to-day. From an implementation perspective, the values of background ozone on possible exceedance days is a more meaningful distinction. The first draft policy assessment (EPA, 2012) considered this issue in detail, via summaries of the existing 2006 zero out modeling (Henderson et al., 2012), and concluded that “results suggest that background concentrations on the days with the highest total ozone concentrations are not dramatically higher than typical seasonal average background concentrations.” Based on this finding, the 1st draft policy assessment determined that “anthropogenic sources within the U.S. are largely responsible for 4th highest 8-hour daily maximum ozone concentrations.” This portion of the appendix will consider the entire spectrum of variable background ozone levels with special emphasis on days in which base model ozone concentrations approach or exceed the level of the NAAQS.

The 2007 modeling agrees with the finding from the previous 2006-based modeling analyses that the highest modeled ozone site-days tend to have background ozone levels similar to mid-range ozone days. Figures 5a-5c show the distribution of April-October MDA8 background levels (NB, NAB, and USB, respectively) from the CMAQ zero out runs. As noted in section 2, zeroing out emissions can remove the effects of local NO_x titration and result in modeled background values that are higher than the base model ozone. The “box and whisker” plots shown in these figures display four key features of the distributions:

- a. the median concentration (black horizontal line) per bin,
- b. the inter-quartile range (blue colored box) which represents the 25th-75th percentile range in values within the distribution,
- c. the “whiskers” (dark gray vertical lines with top and bottom whiskers) which represent the range of values within 1.5 times the inter-quartile range, and
- d. the “outliers” (gray points) which are any values outside the whiskers.

As can be seen in Figure 5a, natural background values do not vary greatly as a function of the base modeled ozone. Recall that the seasonal average natural background MDA8 ozone values were modeled to range from 15-35 ppb across the U.S. with a median value of 24 ppb. The highest values were at the high-elevation sites in the western U.S. Based on the distributional analysis, the 75th percentile values are on the order of 30 ppb. Natural background levels exceeding 40-45 ppb are considered to be statistical outliers, due to their infrequency. Figure 5b shows the same type of distributions but for NAB instead of NB. NAB values are generally 6-12 ppb higher than their NB counterparts, due to the affect of higher global methane values and the influence of anthropogenic emissions from Asia. It was previously reported (in section 3) that the median seasonal average NAB MDA8 values were 31.5 ppb. Based on the distributions, it can be seen that 75th percentile values are

approximately 40 ppb; it is rare for NAB MDA8 values to exceed 50-55 ppb. NAB values are constant in magnitude once the base ozone exceeds 50 ppb indicating that the higher base ozone values are driven by non-NAB sources (i.e., North American emissions). Finally in Figure 5c, the USB MDA8 distributions by base model MDA8 are shown. The results are similar to NAB.

Figure 5d shows the results from the source apportionment modeling of non-U.S. anthropogenic source contributions to MDA8 ozone (i.e., the nine source apportionment categories other than U.S. anthropogenic emissions and Gulf of Mexico point sources). This non-counterfactual approach is expected to give a better indication of background levels at low concentrations. At low levels, almost all of the ozone is determined to be from background origins. The CAMx modeling shows that contributions from non-U.S. anthropogenic emissions peak when base ozone ranges from 45-55 ppb and then drop off slightly at higher base MDA8 values. The source apportionment modeling of non-U.S. impacts (similar to USB) indicates slightly lower background levels than the zero out modeling. The 75th percentile values are generally less than 35 ppb, compared to 40 ppb in the zero out modeling. It is rare to have background impacts greater than 55ppb. Interestingly, when base model MDA8 ozone exceeds 70 ppb, it is rare to have background impacts greater than 45 ppb in the CAMx source apportionment modeling.

Figures 6a-6d show the equivalent plots as 5a-5d, but use background fractions (background MDA8 / base MDA8) as the dependent variable instead of the absolute background concentrations. These plots show the same effect; that is, the proportional relative contribution of background sources and processes decreases as peak ozone increases. For natural background (Figure 6a), the median fractions drop from 50% background for values between 45-50 ppb to only 35% background for base MDA8 values between 70-75 ppb. For NAB and USB (Figures 6b and 6c), the median fractions drop from 70% background for values between 45-50 ppb to only 45% background for base MDA8 values between 70-75 ppb. The source apportionment modeling (Figure 6d) estimates less of a proportional role of non-U.S. anthropogenic emissions. In that modeling, the median fractions drop from 65% background for values between 45-50 ppb to only 35% background for base MDA8 values between 70-75 ppb. A key observation, as noted in the first draft policy assessment document, is that the relative importance of background decreases on days most likely to violate the NAAQS. An additional policy-relevant finding from the distributional analyses is that the relative role of background sources would be increased if the level of the NAAQS were lowered. At 60 ppb, the modeling suggests that the median fractional contribution from background is 45-55 percent, but there can be cases where background comprises 80-90 percent of the total ozone.

Many of the cases when background ozone is estimated to contribute in large proportions to relatively high ozone days may be eligible for consideration as exceptional events, but again, this modeling is not designed to resolve specific events that occurred in 2007. While there is greater confidence in the model's ability to predict mean contributions from background sources than from individual events, it is also useful to briefly consider the upper end of the background ozone

distributions. Figure 7 shows the 95th percentile³ USB estimates from the zero out modeling. The 95th percentile MDA8 USB ozone levels range from 35-60 ppb, with the most frequent values residing in the 35-40 and 40-45 ppb bins. The median 95th percentile background USB ozone level is 42.0 ppb. As with the seasonal mean MDA8 USB, the highest levels of high background days (i.e., 95th percentile days) are observed over the intermountain western U.S. At these locations, 95th percentile USB levels can exceed 50 ppb. Background values at the 95th percentile end of the distribution are 4-12 ppb higher than the mean background values at the same locations.

³ During the April-October period, there were 214 days of modeling results. Thus, the 95th percentile values represent approximately the 10th highest days from the distribution.

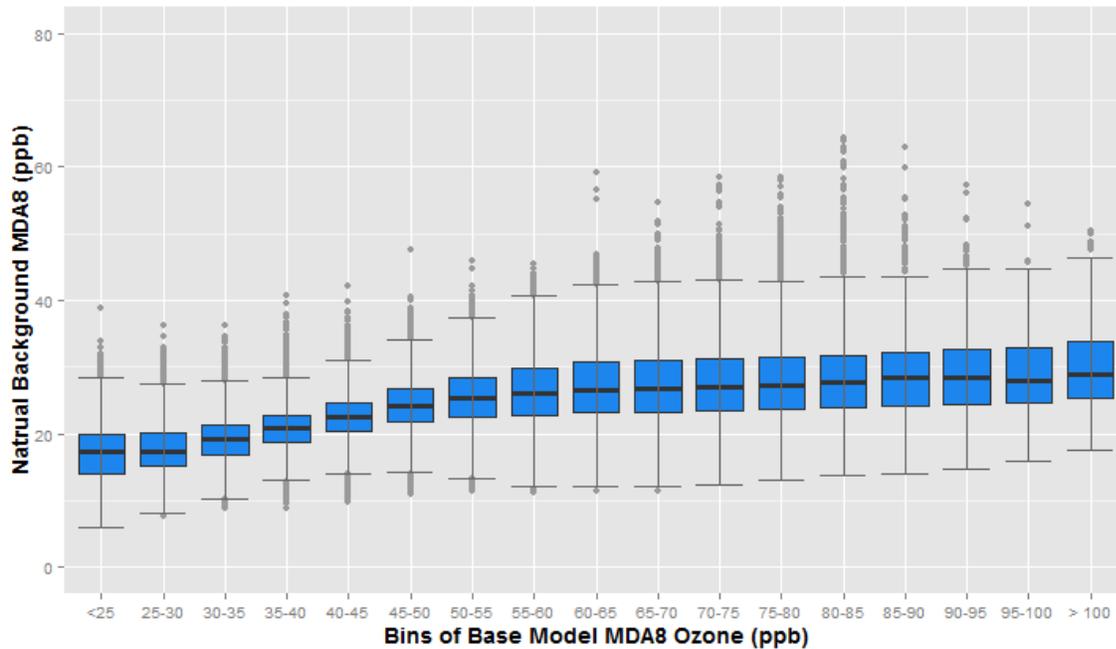


Figure 5a. Distribution of *natural background* MDA8 ozone (ppb) at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

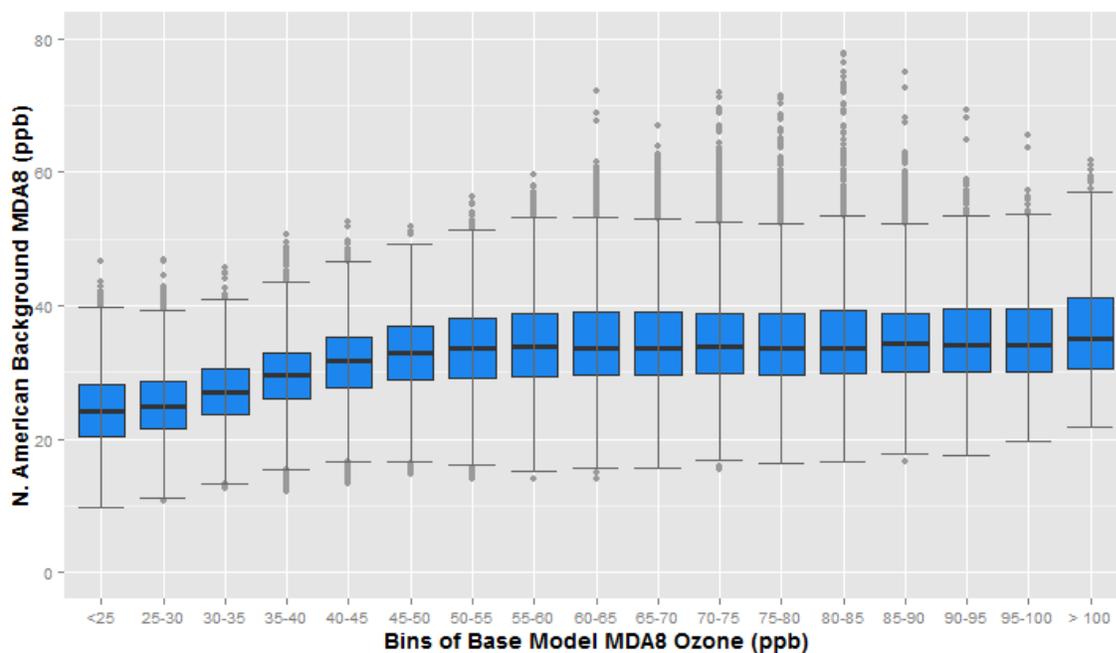


Figure 5b. Distribution of *N. American background* MDA8 ozone (ppb) at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

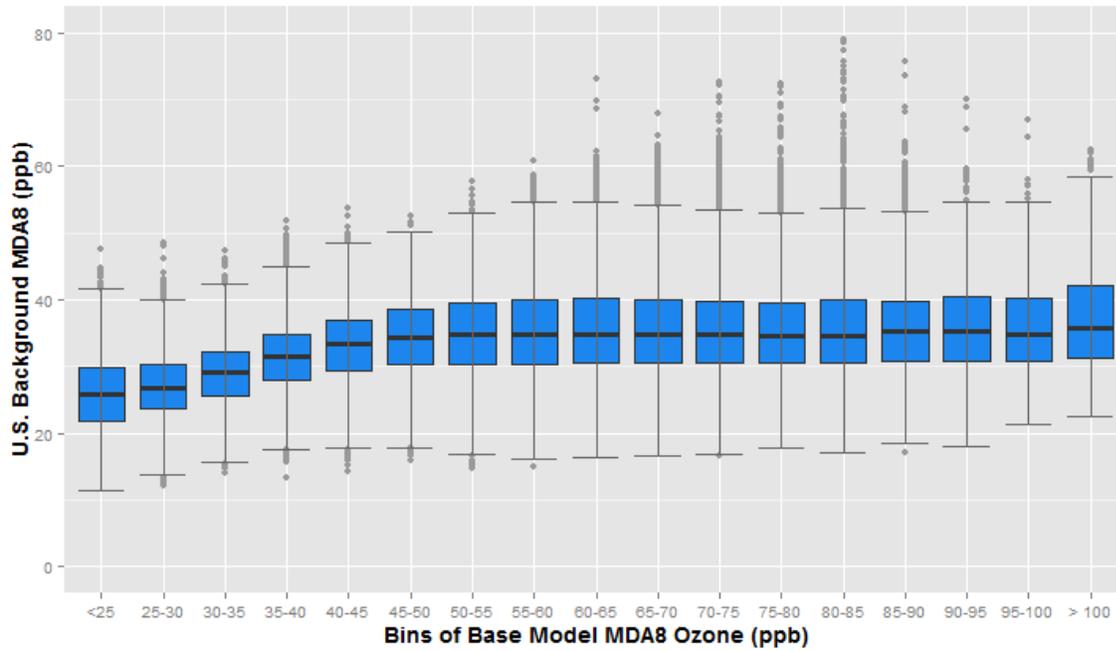


Figure 5c. Distribution of *U.S. background* MDA8 ozone (ppb) at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

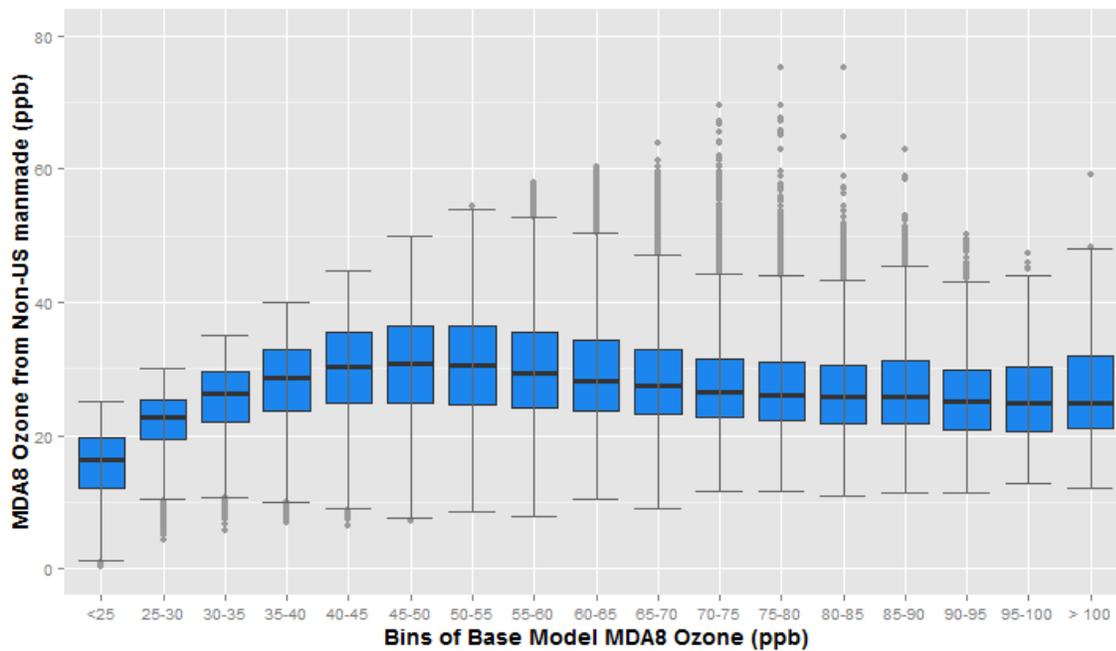


Figure 5d. Distribution of MDA8 ozone contributions from non-U.S. manmade sources (ppb) at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CAMx simulations.

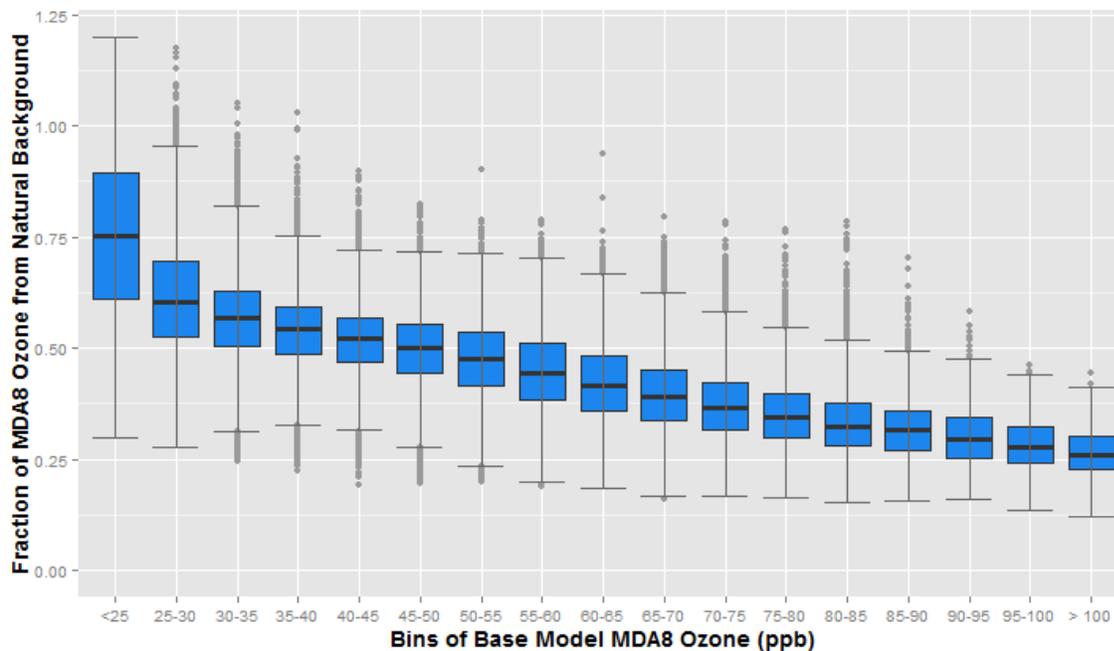


Figure 6a. Distribution of *natural background* MDA8 ozone fractions at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

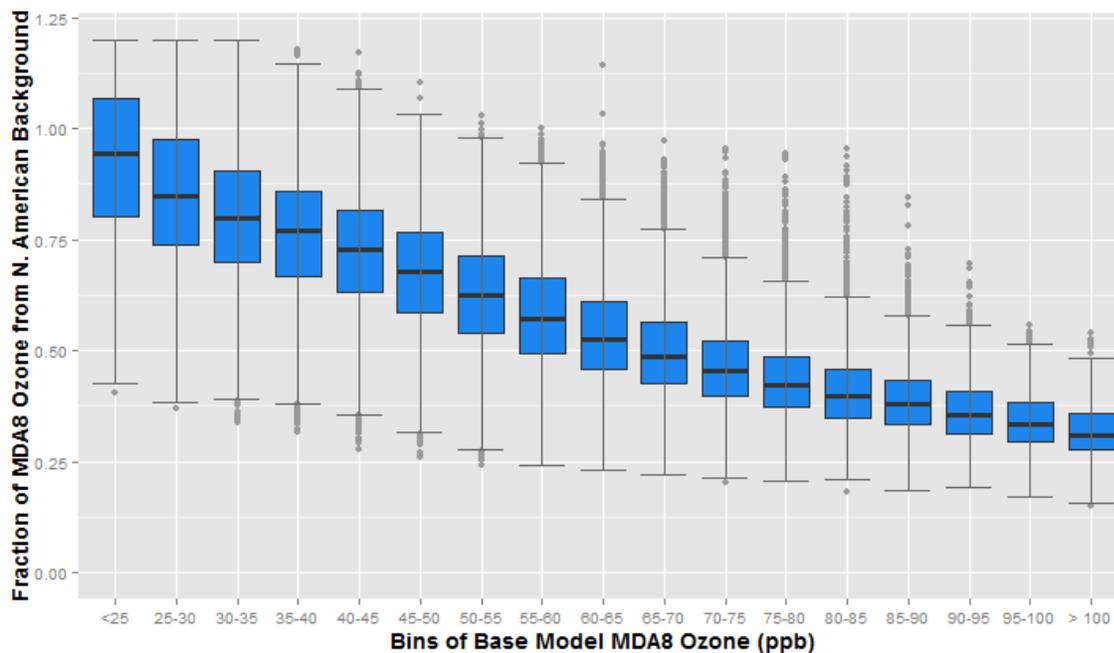


Figure 6b. Distribution of *N. American background* MDA8 ozone fractions at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

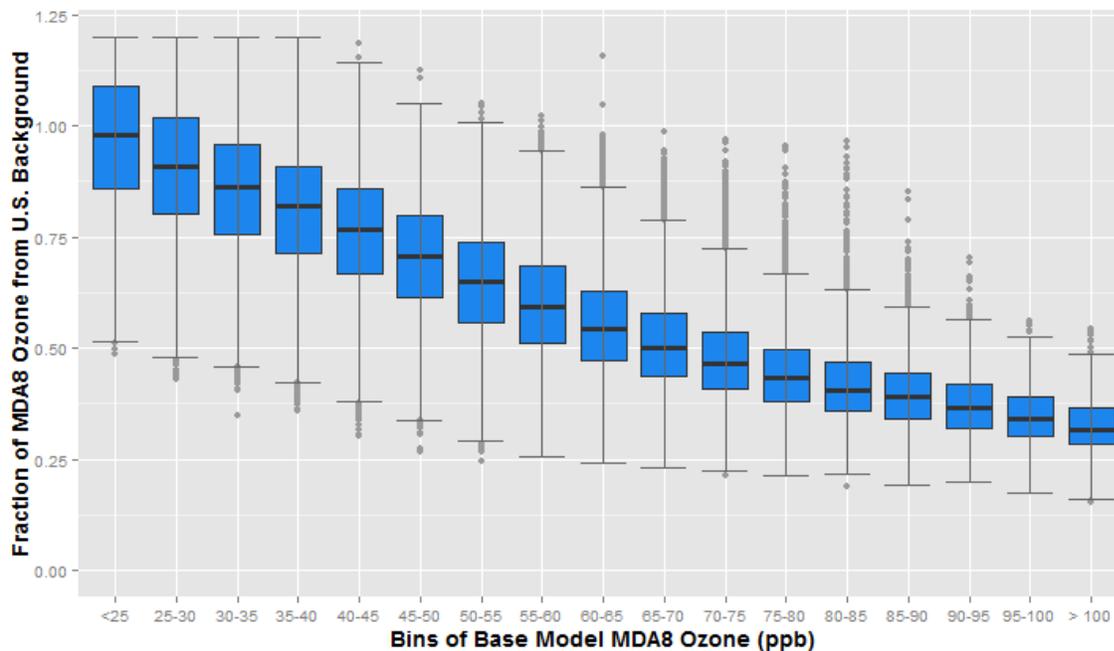


Figure 6c. Distribution of *U.S. background* MDA8 ozone fractions at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by 2007 CMAQ simulations.

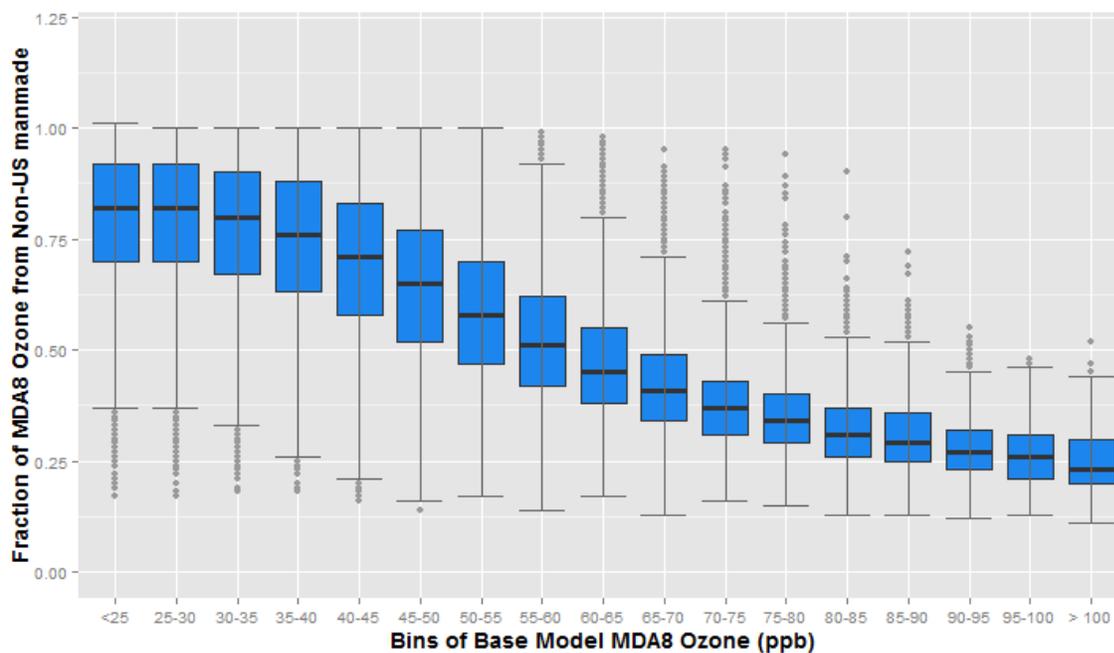


Figure 6d. Distribution of MDA8 ozone fractions *from non-U.S. anthropogenic sources* at monitoring locations across the U.S. (Apr-Oct), binned by base modeled site-day MDA8, as estimated by the 2007 CAMx simulation.

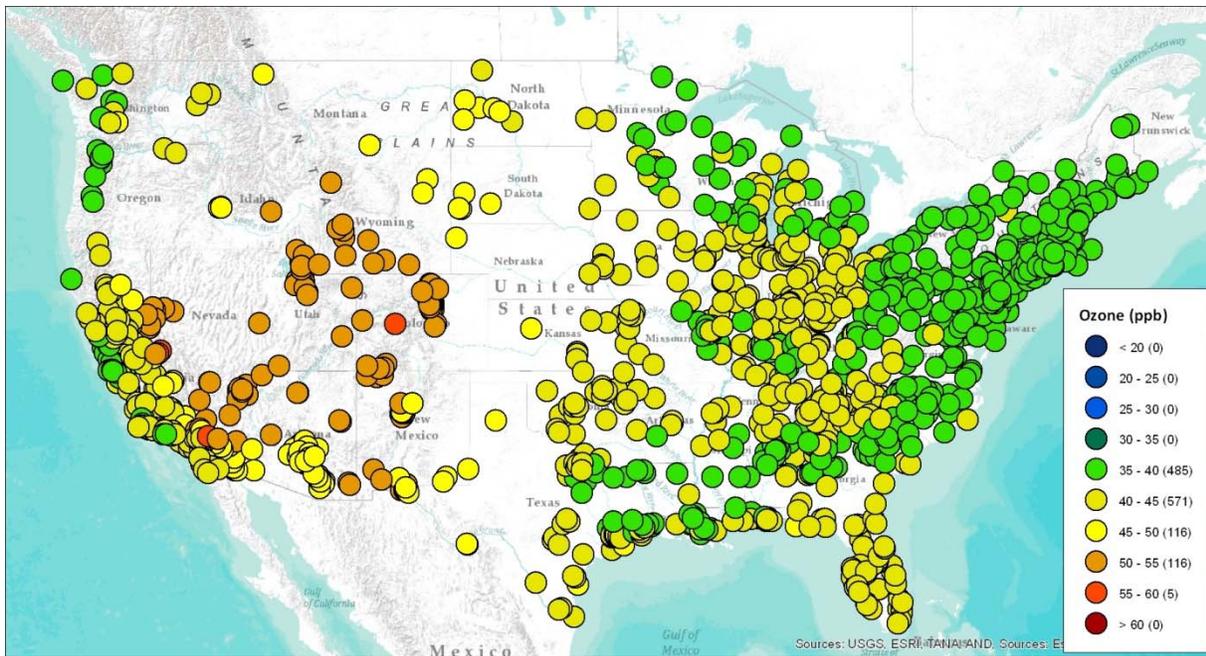


Figure 7. April-October 95th percentile *United States background MDA8 ozone (ppb)* at monitoring locations across the U.S. as estimated by a 2007 CMAQ base simulation.

5. Contribution of various processes and sources to total background ozone

This section will utilize the supplemental 2007 air quality modeling estimates to determine the relative importance of specific elements of background ozone. Comparing the differences between the three zero out scenarios can provide some information about the role of certain sets of emissions. Figure 8a compares the NAB (zero out North American manmade emissions) and USB (zero out U.S. manmade emissions) scenarios. The difference between these two runs is the inclusion of anthropogenic emissions within the Canada and Mexico portions of the modeling domain. These emissions contribute less than 2 ppb to the seasonal mean MDA8 ozone levels over most of the U.S. There are 70 sites, near an international border, where the modeling estimates Canadian/Mexican seasonal average impacts of 2-4 ppb. While not shown, the modeled peak single day impacts from these specific international emissions sources can approach 25 ppb (e.g., San Diego, Buffalo NY). Figure 8b compares the NB (zero out all manmade emissions and reset GEOS-Chem methane values to pre-industrial levels) to the NAB. The difference between these two runs is the inclusion of global methane emissions related to recent human activity as well as anthropogenic emissions outside of North America. These emissions are estimated to contribute 6-15 ppb to seasonal mean ozone levels over the U.S. The most frequent bin is the 8-10 ppb increase. It is not possible via these runs to parse out what fraction of this change is due to international emissions as opposed to methane emissions, but the ISA summarized existing modeling (Zhang *et al.*, 2011) that suggested that the rise in methane from pre-industrial levels to present-day levels led to increases in seasonal average ozone levels of 4-5 ppb. The greatest impacts from these sources occurs over the western U.S., where international emissions would be expected to have the largest impacts.

Figures 9a-9g show the fractional contribution to total seasonal mean MDA8 values of individual source sectors that were tracked in the CAMx source apportionment modeling. Figure 9a shows the impact from the regional model boundary conditions. The ozone entering the model domain via the boundary conditions could have a variety of origins including: a) natural sources of ozone and ozone precursors (including methane) emanating from outside the domain, b) anthropogenic sources of ozone precursors (including methane) from international emitters, and c) some fraction of U.S. emissions (natural and anthropogenic) which are exported and then re-imported into the domain via synoptic-scale recirculation. Thus, one should not presume that the boundary condition contribution is directly tied to any particular background definition. At most locations, boundary conditions contributed 40-60 percent of the total MDA8 seasonal mean at sites across the U.S. The highest proportional impacts from the boundary conditions (the top boundary contributes negligibly) are along the coastlines and the intermountain West.

Figure 9b shows the source apportionment contribution (to seasonal mean MDA8) from the most significant sector that was tracked: U.S. anthropogenic ozone precursor emissions. Again the most common outcome at an individual site was that 40-60% of the seasonal mean ozone values originated from U.S. anthropogenic emissions. The locations with smaller fractional contributions (e.g., 10-20 percent) from U.S. sources are generally located in places where ozone values are typically low such as the Pacific Northwest. Figures 9c-9g display the fractional contributions from the other five in-domain sectors listed in section 2. The impacts from these sectors are briefly summarized below:

- Biogenic emissions:
 - Most frequent bin: 3-5 percent
 - Highest site-specific contribution: 10-20 percent
 - Region with greatest impacts: Great Plains states where soil NO_x emissions are large
- Climatologically-average fire emissions:
 - Most frequent bin: 0-1 percent
 - Highest site-specific contribution: 3-5 percent
 - Region with greatest impacts: California, Kansas/Oklahoma region
- Within-domain Canadian/Mexican manmade emissions:
 - Most frequent bin: 0-1 percent
 - Highest site-specific contribution: 10-20 percent
 - Region with greatest impacts: Sites along international borders (NY, VT, CA, AZ, TX)
- Category 3 marine vessels outside U.S. territorial waters:
 - Most frequent bin: 0-1 percent
 - Highest site-specific contribution: 10-20 percent
 - Region with greatest impacts: Coastal sites (especially southern CA)
- Gulf of Mexico point sources⁴:
 - Most frequent bin: 0-1 percent
 - Highest site-specific contribution: 1-3 percent
 - Region with greatest impacts: Sites in southeast TX and southern LA

⁴ This sector was also included as part of U.S. anthropogenic source impacts in Figure 9b, but is broken out separately in Figure 9g.

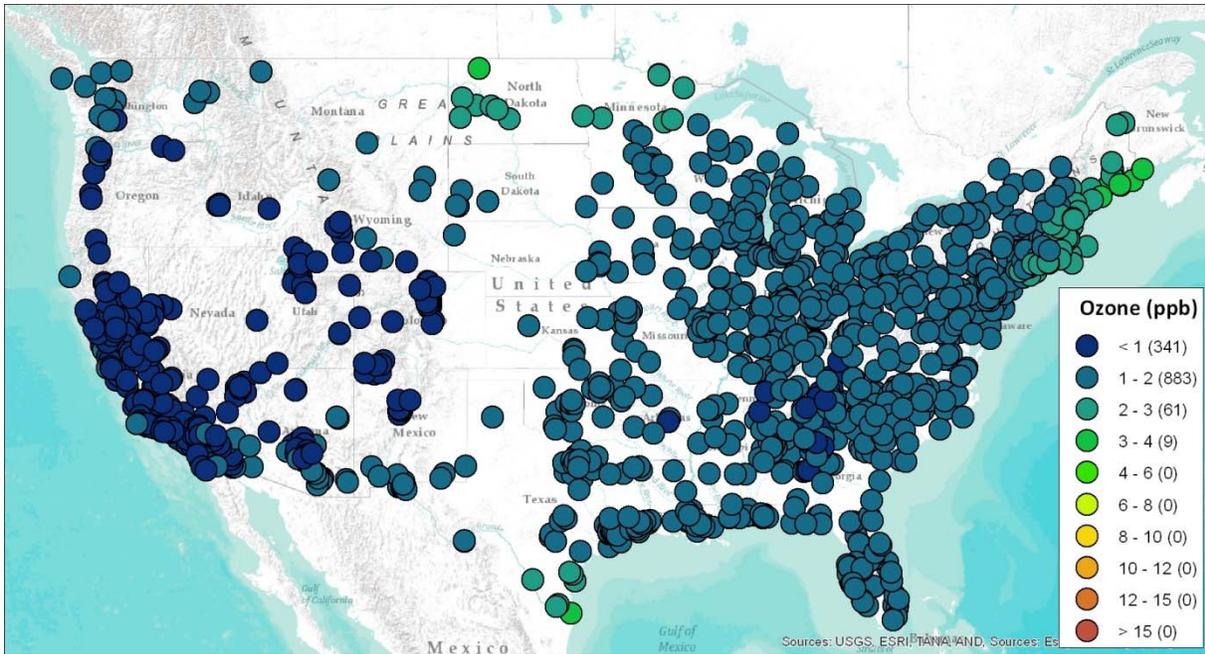


Figure 8a. Difference in April-October average MDA8 ozone (ppb) at monitoring locations across the U.S. between the USB scenario and the NAB scenario. The difference between these two runs isolates the impact of within-the-domain anthropogenic emissions from Canada and Mexico.

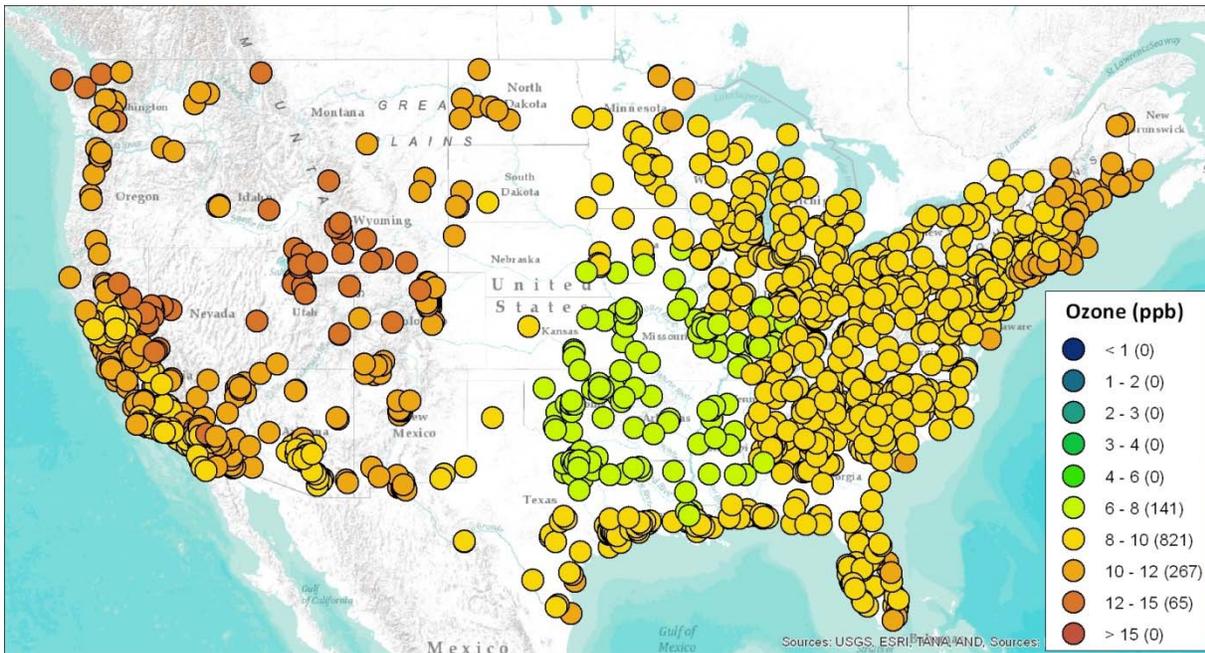


Figure 8b. Difference in April-October average MDA8 ozone (ppb) at monitoring locations across the U.S. between the NAB scenario and the NB scenario. The difference between these two runs isolates the impact of the rise in global methane emissions from the pre-industrial and anthropogenic emissions from outside North America.

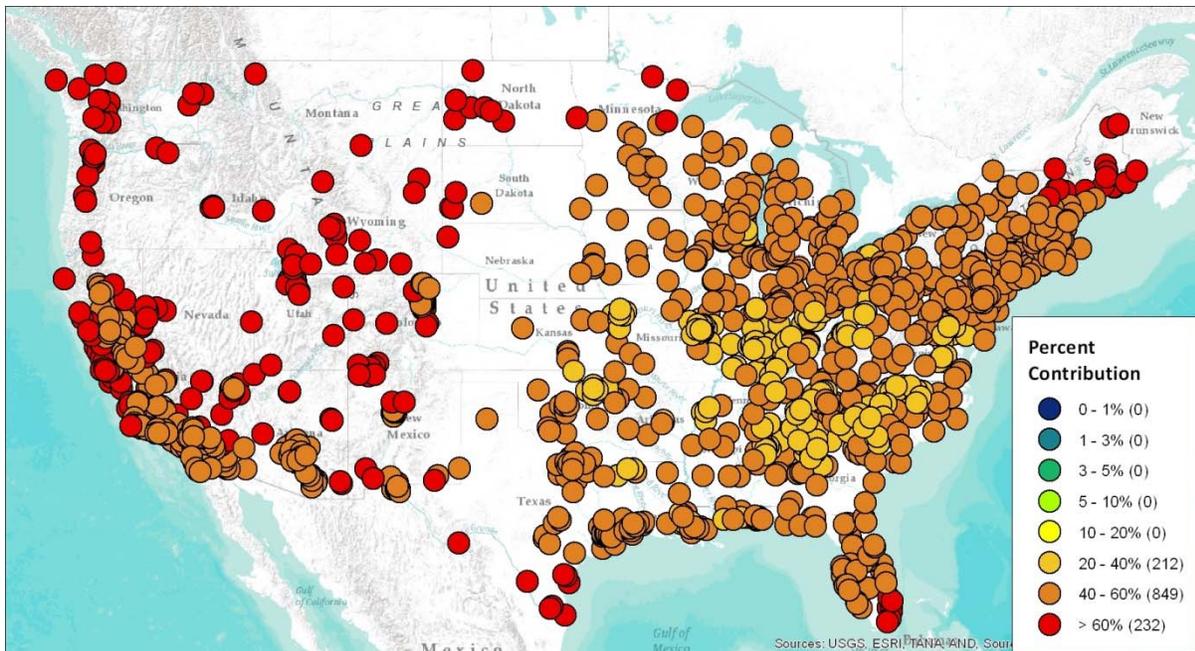


Figure 9a. Percentage of April-October average MDA8 ozone that is apportioned to *boundary conditions* as estimated at monitoring locations by a 2007 CAMx simulation.



Figure 9b. Percentage of April-October average MDA8 ozone that is apportioned to *U.S. anthropogenic sources* as estimated at monitoring locations by a 2007 CAMx simulation.

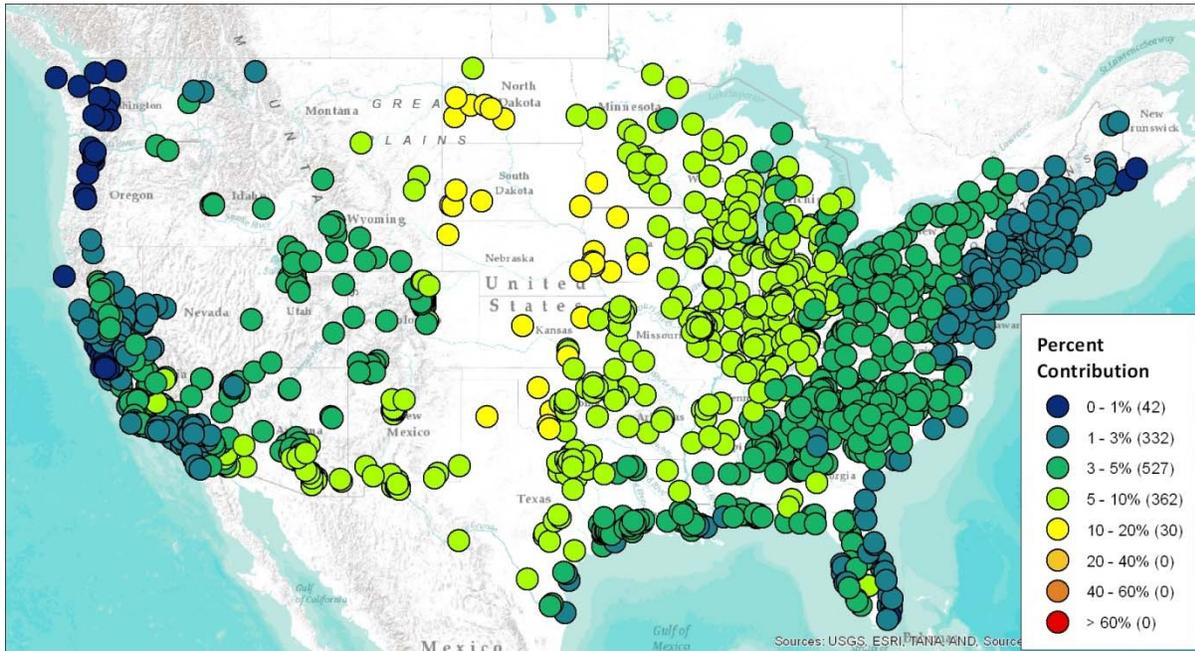


Figure 9c. Percentage of April-October average MDA8 ozone that is apportioned to *purely biogenic emissions* as estimated at monitoring locations by a 2007 CAMx simulation.



Figure 9d. Percentage of April-October average MDA8 ozone that is apportioned to *climatological fire emissions* as estimated at monitoring locations by a 2007 CAMx simulation.

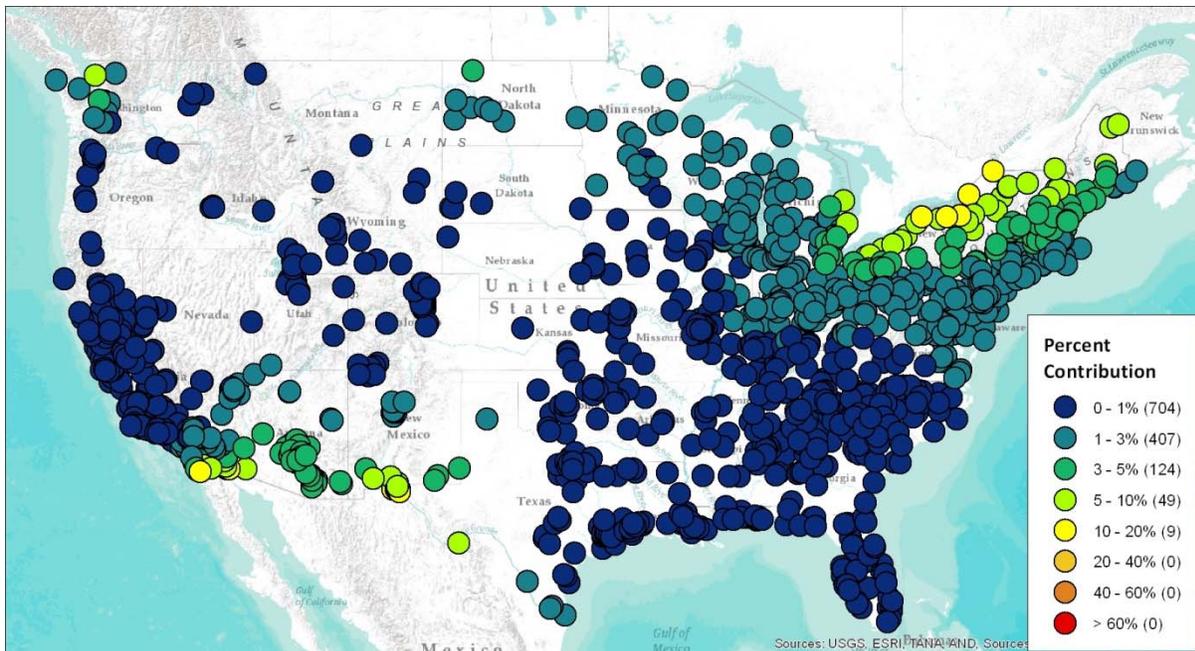


Figure 9e. Percentage of April-October average MDA8 ozone that is apportioned to *anthropogenic emissions from in-domain Canadian and Mexican sources* as estimated at monitoring locations by a 2007 CAMx simulation.

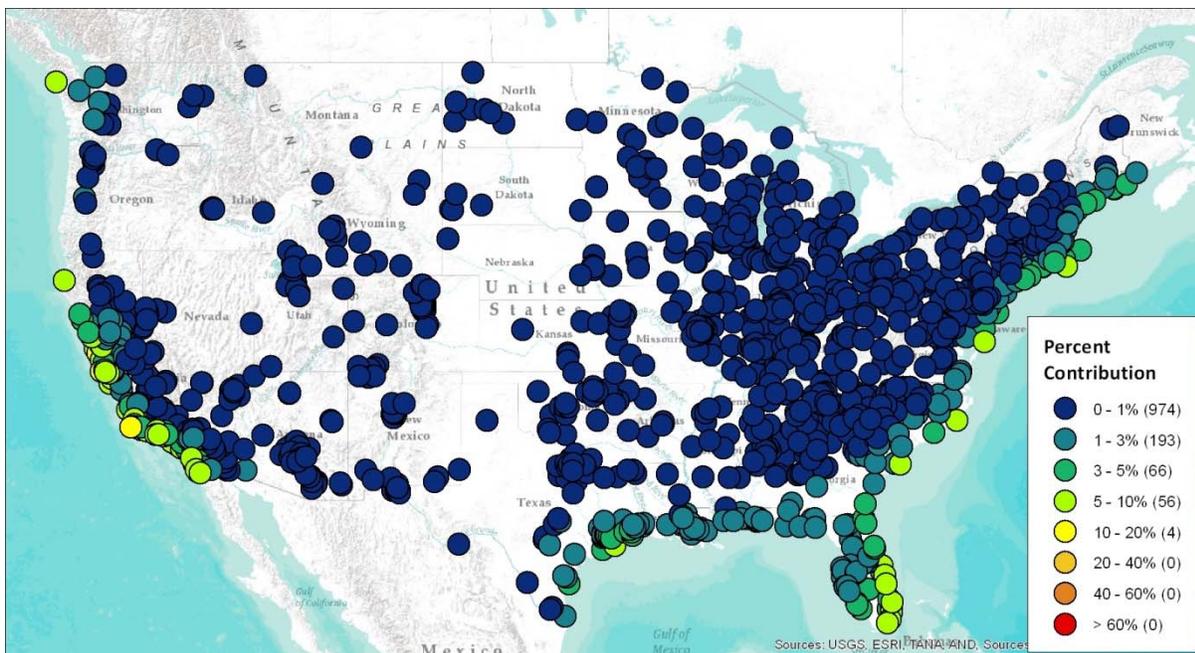


Figure 9f. Percentage of April-October average MDA8 ozone that is apportioned to *Category 3 marine vessel emissions beyond U.S. territorial waters* as estimated at monitoring locations by a 2007 CAMx simulation.



Figure 9g. Percentage of April-October average MDA8 ozone that is apportioned to *Gulf of Mexico point sources* as estimated at monitoring locations by a 2007 CAMx simulation.

6. Estimates of the fractional background contribution to total ozone in 12 specific areas

This penultimate section of the appendix presents estimates of the overall fraction of ozone that is estimated to result from background sources or processes based on the updated modeling in each of the 12 urban case study areas in the epidemiology study based analyses in Chapter 7 of the Risk and Exposure Assessment (REA). Tables 1a-1c summarize the CAMx-estimated fractional contributions of sources other than U.S. anthropogenic emissions to total ozone in each of the 12 areas. Table 1a shows that the fractional contributions from sources other than anthropogenic emissions within the U.S. to seasonal mean MDA8 levels can range from 43 to 66 percent across these 12 urban areas. These fractions are consistent with the national ratios summarized in section 3, although the urban fractions of background tend to be smaller than at rural sites. As shown in section 4, the fractional contributions from background are smaller on days with high modeled ozone (i.e., days that may exceed the level of the NAAQS). Table 1b provides the fractional contributions from these non-U.S. sources, only considering days in which base model MDA8 ozone was greater than 60 ppb. As expected, the fractional background contributions are less and range from 31 to 55 percent. Rather than taking the fractions of the seasonal means (as in Table 1a), Table 1c displays the mean and median daily MDA8 background fractions. These metrics may be more appropriate for application to health studies, but as can be seen the fractional contribution to backgrounds calculated via this approach are very similar to the Table 1a calculations. For completeness sake, although EPA expects the source apportionment results to provide a more realistic estimate of fractional background values, for completeness, we also provide USB fractions based on zero out modeling for the 12 cities (see Table 1d). The results are similar to the source apportionment findings (compare against Table 1a), but the zero out technique provides slightly higher background proportions.

All days, CAMx	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	59.3	54.4	43.0	48.9	47.3	39.1	48.5	51.1	45.4	48.7	46.4	49.8
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.3	25.9	26.2	25.7	31.3	23.3	27.0	29.1	24.5	24.2	29.7	24.3
Fractional contribution from background	0.43	0.48	0.61	0.52	0.66	0.60	0.56	0.57	0.54	0.50	0.64	0.49

Table 1a. April-October average MDA8 ozone, average MDA8 ozone from sources other than U.S. manmade emissions, and the fractional contribution of these background sources in the 12 REA urban study areas, as estimated by a 2007 CAMx simulation.

Only days w/ base MDA8 > 60 ppb	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	74.0	75.3	70.7	72.0	67.5	68.9	70.3	74.4	74.1	74.0	68.3	70.0
Model MDA8 seasonal mean from emissions other than U.S. anthropogenic sources	25.4	23.7	24.4	25.4	37.3	24.4	28.0	31.9	23.5	22.9	32.1	25.4
Fractional contribution from background	0.34	0.31	0.35	0.35	0.55	0.35	0.40	0.43	0.32	0.31	0.47	0.36

Table 1b. Average MDA8 ozone, average MDA8 ozone from sources other than U.S. manmade emissions, and the fractional contribution of these background sources in the 12 REA areas, as estimated by a 2007 CAMx simulation using site-days in which base MDA8 ozone exceeded 60 ppb.

	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Mean of daily MDA8 background fractions	0.46	0.53	0.68	0.58	0.69	0.64	0.59	0.61	0.61	0.56	0.67	0.52
Median of daily MDA8 background fractions	0.43	0.51	0.73	0.54	0.69	0.66	0.59	0.60	0.63	0.54	0.66	0.49

Table 1c. Fractional contribution of non-U.S. manmade emissions sources in the 12 REA urban study areas, as estimated by a 2007 CAMx simulation using means and medians of daily MDA8 fractions.

All days, CMAQ	ATL	BAL	BOS	CLE	DEN	DET	HOU	LA	NYC	PHI	SAC	STL
Model MDA8 seasonal mean	58.6	55.6	45.2	51.8	57.1	43.5	49.4	54.8	47.7	50.5	51.9	52.6
Model MDA8 seasonal mean from USB emissions	30.0	29.9	28.5	31.6	42.2	31.7	33.0	33.3	29.1	29.4	34.4	32.0
Fractional contribution from background	0.51	0.54	0.63	0.61	0.74	0.73	0.67	0.61	0.61	0.58	0.66	0.61

Table 1d. April-October average MDA8 ozone, average MDA8 ozone from USB, and the fractional contribution of these background sources in the 12 REA urban study areas, as estimated by two separate 2007 CMAQ simulations.

7. Background ozone and W126

As discussed in section 5 of the second draft policy assessment, EPA is considering the adequacy of the current secondary standard to protect against welfare effects. One metric that has been considered previously as a potential cumulative seasonal index is the W126 metric. The W126 index is a sigmoidally weighted sum of all hourly O₃ concentrations observed during a specified daily and seasonal time window, where each hourly O₃ concentration is given a weight that increases from 0 to 1 with increasing concentration (Lefohn et al, 1988). The weights are defined such that values of 0.060 ppm get a weight of ~0.3; 0.070 ppm values get a weight of ~0.6; and 0.085 ppm values get a weight of ~0.9. The remainder of this section uses the 2007 zero out modeling to conduct a limited assessment of the role of background ozone on W126 levels over the U.S.

The analysis of background influence on W126 is not as detailed as the analyses related to seasonal mean MDA8 ozone. Instead of considering impacts at every monitoring location, EPA assessed NB, NAB, and USB influences at four sample locations: Atlanta GA, Denver CO, Farmington NM, and Riverside CA. Each of these four locations had relatively high observed values of W126 in 2010-2012. Atlanta is an urban area in the Eastern U.S. with high primary ozone design values but relatively low levels of seasonal background ozone. Riverside and Denver also have high primary ozone design values but are in the Western U.S. where background ozone levels are generally higher. Farmington NM was chosen as a site that has relatively lower primary ozone design values along with its relatively high W126 levels. The varying characteristics of each of these locations perhaps allows broader national extrapolation of the 4-site results.

In previous EPA reviews of the O₃ NAAQS, the influence of background ozone was estimated according to a counterfactual (i.e., how much ozone would exist in the absence of certain sets of emissions). In the current review, EPA is supplementing the counterfactual assessment with analyses that estimate the fraction of the existing ozone that is due to background sources. This has important ramifications for assessing the influence of background on W126 concentrations, because of the non-linear weighting function used in the metric which emphasizes high ozone hours (e.g., periods in which ozone is greater than ~60 ppb). As an example, consider a sample site in the intermountain western U.S. region with very high modeled estimates of U.S background (e.g., seasonal mean USB of 45 ppb with some days as high as 65 ppb). Even at this high background location, the calculated annual W126 values in the USB scenario are quite low, on the order of 3 ppm-hrs. Most sites in the domain where background levels are lower than the location cited above will have even smaller background W126 estimates, on the order of 1 ppm-hrs, which is consistent with values mentioned in past reviews (USEPA, 2007). Using the counterfactual scenarios, background ozone has a relatively small impact on W126 levels across the U.S.

However, because of the non-linear weighting function used in the W126 calculation, the sum of the W126 from the USB scenario and the W126 resulting from US anthropogenic sources will not equal the total W126. In most cases, the sum of those two components will be substantially less than total W126. As a result, EPA believes it is more informative to estimate the fractional contribution of background ozone to W126 levels. The 5-step methodology for assessing the fractional influence of

background ozone to annual W126 levels in the four locations is described below. The fractional influence methodology essentially places higher weights on background fractions on days that are going to contribute most substantially to the yearly W126 value.

- Step 1a: Calculate the MDA8 ozone values from the base and the three zero out modeling scenarios at each grid cell containing a site in an area.
- Step 1b: Calculate the W126 daily index for the base model scenario.
- Step 2: For each site, find the three months with highest summed W126 daily indices.
- Step 3: Normalize the daily MDA8 values in the base, NB, NAB, and USB scenarios by the corresponding W126 daily index from the base scenario.
- Step 4: Calculate the average W126-weighted MDA8 values over the three month period for each of the four scenarios (base, NB, NAB, USB).
- Step 5: Calculate the NB/Base, NAB/Base, and USB/Base ratios based on step 4 outputs. These values represent an estimate of the fractional influence of background ozone on modeled W126 levels.

Figure 7a shows the estimated fractional influence of the three background definitions on W126 levels in Atlanta, Denver, Farmington, and Riverside. Based on this limited assessment, natural background sources are estimated to contribute 29-50% of the total modeled W126 with the highest relative influence in the intermountain western U.S. (e.g., Farmington NM) and the lowest relative influence in the eastern U.S. (e.g., Atlanta). U.S. background is estimated to contribute 37-65% of the total modeled W126. Figure 7b compares the relative influence of background on W126 versus seasonal mean MDA8 ozone. The proportional impacts of background are slightly less for the W126 metric than for seasonal mean MDA8 (discussed in section 2.4.2), because of the weighting function that places more emphasis on higher ozone days when background fractions are generally lower.

There are several caveats associated with this analysis. First, only the zero out modeling was used to assess the fractional influence of background sources on W126. The source apportionment approach estimated slightly smaller relative contributions for seasonal mean MDA8 levels, so from that perspective the zero out estimates could represent the high end of background influence on W126. Additionally, the methodology used for this analysis relies on daily MDA8 values as a surrogate (the data were readily available) for the 8a-8p time period relevant to the W126 metric. The key conclusion from this cursory analysis is that background ozone may comprise a non-negligible portion of current W126 levels across the U.S. This fractional influence is greatest in the intermountain western U.S. and are slightly smaller than the seasonal mean MDA8 metric. In the counterfactual cases, when non background sources are completely removed, the remaining W126 levels are low (< 3 ppm-hrs).

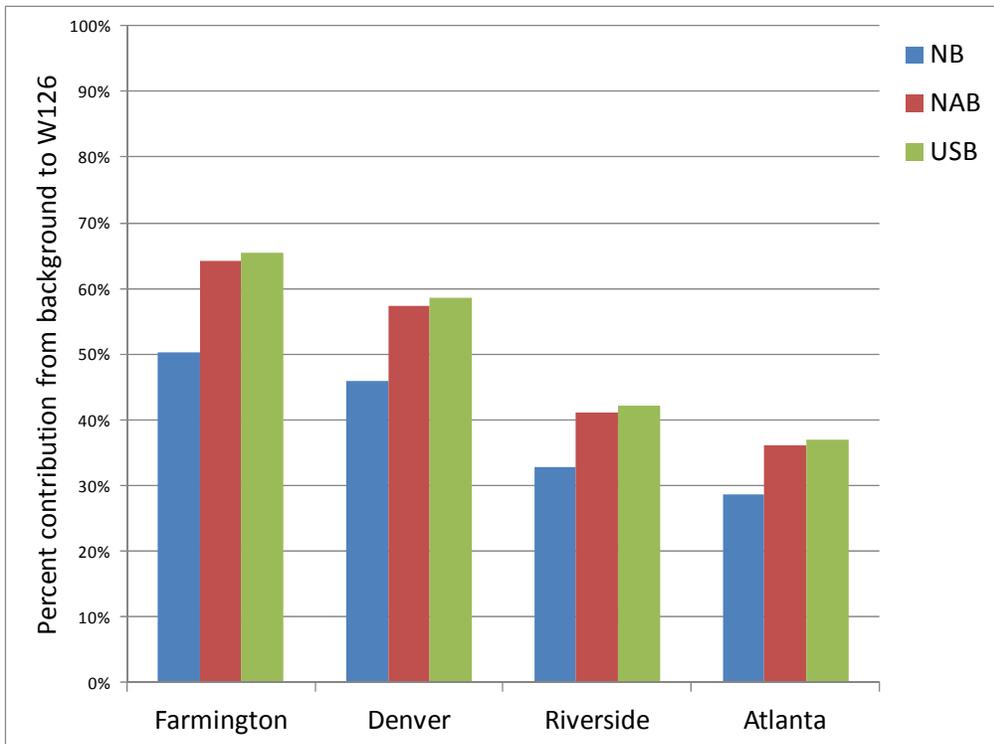


Figure 7a. Fractional contribution of background sources to W126 levels in four sample locations. Model estimates based on 2007 CMAQ zero out modeling.

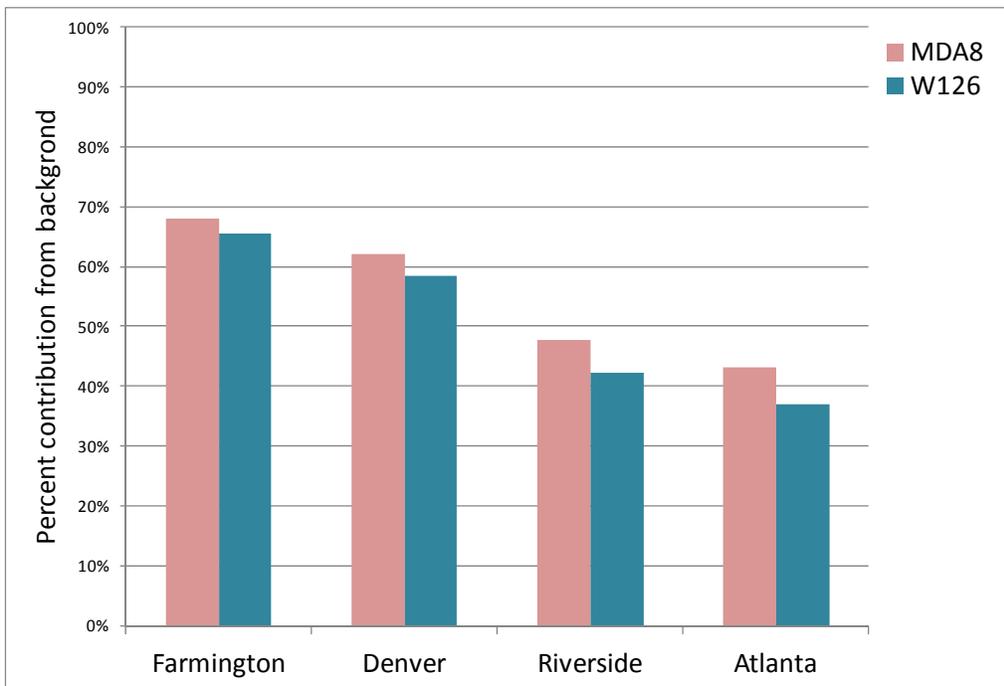


Figure 7b. Fractional contribution of U.S. background to seasonal mean MDA8 ozone and W126 levels in four sample locations. Model estimates based on 2007 CMAQ zero out modeling.

8. Summary

The precise definition of background ozone can vary depending upon context, but it generally refers to ozone that is formed by sources or processes that cannot be influenced by local control measures. Background ozone can originate from natural sources of ozone and ozone precursors, as well as from upwind manmade emissions of ozone precursors. In order to help further characterize background ozone levels over the U.S., EPA has completed additional air quality modeling analyses subsequent to the 1st-draft policy assessment. As shown above, the results are largely consistent with previous determinations about the magnitude of background ozone contributions across the U.S.

For a variety of reasons, it is challenging to present a comprehensive summary of all the components and implications of background ozone. In many forums the term “background” is used generically and the lack of specificity can lead to confusion as to what sources are being considered. Additionally, it is well established that the impacts of background sources can vary greatly over space and time which makes it difficult to present a simple summary of background ozone levels. Further, background ozone can be generated by a variety of processes, each of which can lead to differential patterns in space and time, and which often have different regulatory ramifications. Finally, background ozone is difficult to measure and thus, typically requires air quality modeling which has inherent uncertainties and potential errors and biases. Even with all of these complexities in mind, EPA believes the following concise and step-wise summary of background ozone is appropriate as based on previous modeling exercises and the more recent EPA analyses summarized herein.

- The most fundamental definition of background is “natural background” (NB). NB ozone is that which is produced by processes other than manmade emissions. Examples of sources of natural background include: stratospheric ozone intrusions, wildfire emissions, and biogenic emissions from vegetation and soils. To date, NB ozone has been estimated to be that ozone that would exist in the absence of anthropogenic ozone precursor emissions worldwide. Modeling analyses have shown that NB levels can vary in time and space. As shown in Section 3, April-October average NB levels range from approximately 15-35 ppb with the highest values in the spring and at higher-elevation sites.
- More expansive definitions of background include North American background (NAB) and U.S. background (USB). These definitions represent the ozone that originates from sources and processes other than North American or U.S. anthropogenic sources. Sources of NAB and USB include all the same sources of natural background, plus manmade ozone precursors emitted outside the North America or the U.S. Modeling analyses have shown that NAB and USB background levels can vary in time and space. As discussed in Section 3, seasonal mean NAB and USB background levels range from approximately 25-45 ppb with the highest values in the spring and at higher-elevation sites. USB levels are slightly higher than NAB, usually by less than 2 ppb.

- Estimates of seasonal mean background ozone levels are valuable in terms of a first-order characterization, however because levels can vary significantly from day-to-day, it is also instructive to consider the distribution of daily model estimates of background ozone over a season. Typically, model background is slightly higher in the April-June period than in the later portion of the ozone season (July-October) (EPA, 2012). More importantly, the modeling shows that the days with highest ozone levels, on average, have similar background levels to days with lower values. As a result, the proportion of total ozone that has background origins is smaller on high ozone days (e.g., days > 70 ppb) than the more common lower ozone days that drive seasonal means. Section 4 provides information about the distribution of background ozone fractions. Based on the source apportionment modeling, it is shown that U.S. anthropogenic emissions typically comprise the majority of the total ozone on site-days with base modeled ozone MDA8 values greater than 60 ppb.
- While it is important to recognize that most high ozone days (i.e., potential exceedance days) are estimated to be driven predominantly by non-background emissions, the recent EPA modeling also shows times and locations in which background contributions are estimated to approach 60-80 ppb. As described in Sections 4 and 6 of this document, these occurrences are relatively infrequent. While the modeling was not expressly developed to capture these types of events, ambient observations have also shown relatively rare events where background ozone sources (wildfires, stratospheric intrusions) have overwhelmingly contributed to an ozone exceedance. From a policy perspective, these background events must be viewed in the context of their relative infrequency and the existing mechanisms within the Clean Air Act (e.g., exceptional event policy, 179B international determinations) that help ensure States are not required to control for events that are inherently outside their ability to influence. While background ozone levels can approach and periodically exceed the NAAQS at some locations, these conditions are not a constraining factor in the selection of a NAAQS. The Clean Air Act requires the NAAQS to be set at a level requisite to protect public health and welfare. Case law makes it clear that attainability and technical feasibility are not relevant considerations. In previous reviews, EPA assessed the proximity of potential levels to peak background levels as a *secondary consideration* between levels where health and welfare was protected.
- Section 5 shows that the contributions to background are multi-dimensional. Daily peak 8-hour ozone values over the U.S. are a function of local and regional anthropogenic emissions, anthropogenic emissions from outside the U.S. (including shipping emissions), natural and anthropogenic methane emissions, wildfire emissions, and purely natural sources. While local and regional controls are still considered to be the most effective at reducing local ozone levels, any measures to reduce the international contributions or methane-induced background will also be valuable.
- In previous ozone NAAQS reviews, EPA estimated risk from exposure only to ozone concentrations above background. In the first drafts of the REA and PA for the current ozone

review, EPA estimated risk from exposure to total measured ozone concentrations, which include those concentrations from background sources. EPA will continue to provide estimates of risk from exposure to total ozone, consistent with CASAC advice, in the second draft policy assessment. The recent EPA modeling was completed to assist in determining, in a limited sense, the risk attributable to background ozone. The fractional values of background contributions in the 12 REA study areas (43-66 percent) could be used as first order approximations of the risk due to ozone background.

9. References

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APPENDIX 2B

MONITORING DATA ANALYSIS OF RELATIONSHIPS BETWEEN CURRENT STANDARD AND W126 METRIC

Presented here are monitoring data analyses evaluating relationship between ozone (O₃) concentrations in the averaging time and form of the current secondary standard (3-year average of the annual 4th highest daily maximum 8-hour concentrations, in parts per billion), and a three-year W126 metric (3-year average of the annual maximum 3-month sum of weighted daytime concentrations, in parts per million-hours). We also consider the responsiveness of these two metrics to historical changes in air quality related to ozone precursor emissions.

For this analysis, we chose to examine monitoring data from a base period (2001-2003) as well as a recent period (2009-2011). The base period was chosen to represent air quality conditions before the implementation of the 1997 national ambient air quality standard (NAAQS) for O₃ (0.08 ppm). In 2004, EPA designated 113 areas as nonattainment for the 1997 standard, which required many areas to begin precursor emissions control programs for the first time. At about the same time, EPA began implementation of the NO_x Budget Trading Program under the NO_x State Implementation Plan, also known as the “NO_x SIP Call¹,” which required summertime reductions in NO_x emissions from power plants and other large sources throughout the Eastern U.S. These programs were successful in reducing peak O₃ concentrations, especially in the Eastern U.S., and as a result only 8 of the original 113 nonattainment areas were still violating the 1997 O₃ NAAQS during the 2009-2011 period.

Hourly O₃ concentration data were retrieved from EPA’s Air Quality System (AQS) database² for both periods, and used to calculate design values for the current standard as well as 3-year average W126 values for both periods. The procedures for calculating design values for the current standard from hourly O₃ concentration data are described in 40 CFR Part 50, Appendix P, and the procedures for calculating the 3-year average W126 values are described in section 4.3.1. of the 2nd draft Welfare Risk and Exposure Assessment (WREA). There were 838 monitoring sites with sufficient data to calculate these values for both periods. In order to identify regional patterns in the relationships, these sites were grouped into the nine NOAA

¹ <http://www.epa.gov/airmarkets/progsregs/nox/sip.html>

² EPA’s Air Quality System (AQS) database is a national repository for many types of air quality and related monitoring data. AQS contains monitoring data for the six criteria pollutants dating back to the 1970’s, as well as more recent additions such as PM_{2.5} speciation, air toxics, and meteorology data. At present, AQS receives hourly O₃ monitoring data collected from nearly 1,400 monitors operated by over 100 state, local, and tribal air quality monitoring agencies.

climate regions (Karl and Koss, 1984) used in the WREA. Figure 2B-1 presents a map of these regions, which are color-coded to match the scatter plots in the subsequent figures.

Figures 2B-2a, 2B-2b, 2B-3a and 2B-3b show scatter plots of the design values for the current standard (x-axis) versus 3-year average W126 values (y-axis) for the base period and recent period, respectively. Most monitors in the U.S. both exceeded the current standard of 75 ppb and a three-year average W126 value of 15 ppm-hrs during the base period. During the recent period, both the design values and 3-year average W126 values were much lower, and there also appears to be less scatter between the two metrics. In both periods, the highest design values and W126 values occurred in the West region which includes California. Finally, it is worth noting that monitors in the Southwest and West regions tend to have higher W126 values relative to their design values than in other regions.

Figure 2B-4 shows a scatter plot of the design values for the current standard for the base period (x-axis) versus for the recent period (y-axis), while Figure 2B-5 shows this same relationship based on the 3-year average W126 values. The relationship between the two periods appears to be fairly linear for both metrics, indicating that larger decreases in these metrics tended to occur at monitors with higher base values. Figures 2B-6 and 2B-7 show design values for the current standard and 3-year average W126 values, respectively, compared to the unit changes in those values between the base period and recent period. Figures 2B-6 and 2B-7 show the difference between each point and the one-to-one lines in Figures 2B-4 and 2B-5, respectively. In particular, these figures highlight that there were some monitors where design values for the current standard and/or W126 values increased. However, those monitors also tended to have lower base values, and were mostly located outside of areas subject to emissions controls under the 1997 standard.

Finally, Figure 2B-8 compares the unit change in design values (in ppb; x-axis) to the unit change in 3-year average W126 values (in ppm-hrs; y-axis). This figure shows that in most locations, the current standard metric and the W126 metric exhibit similar responses to changes in precursor emissions. In particular, the NO_x SIP Call, which was implemented in the states east of the Mississippi River, was effective at reducing both design values and W126 values at nearly all monitors in the Eastern U.S. The relationship was much more variable in the remaining regions, where emissions control programs were mostly local and limited to areas which were violating the NAAQS.

Based on this analysis of ambient monitoring data, we can make the following general conclusions about the relationship between the design value metric for the current O₃ standard and the 3-year average W126 metric:

1. There is a fairly strong, positive degree of correlation between the two metrics.

2. Monitors in the West and Southwest regions tend to have higher W126 values relative to their design values than in other regions.
3. Reducing precursor emissions, especially NO_x, is an effective strategy for lowering both design values and W126 values. In particular, regional control programs such as the NO_x SIP call are effective at reducing both metrics over a broad area.

In addition, Figure 2B-9 examines the number of counties with 8-hour design values meeting the current standard and 3-year average W126 index values greater than 15 ppm-hrs. Most of these counties were located in the Southwest region of the country. There were no counties in any of the studied 3-year periods that had design values less than or equal to 65 ppb and 3-year average W126 index values greater than 15 ppm-hrs.

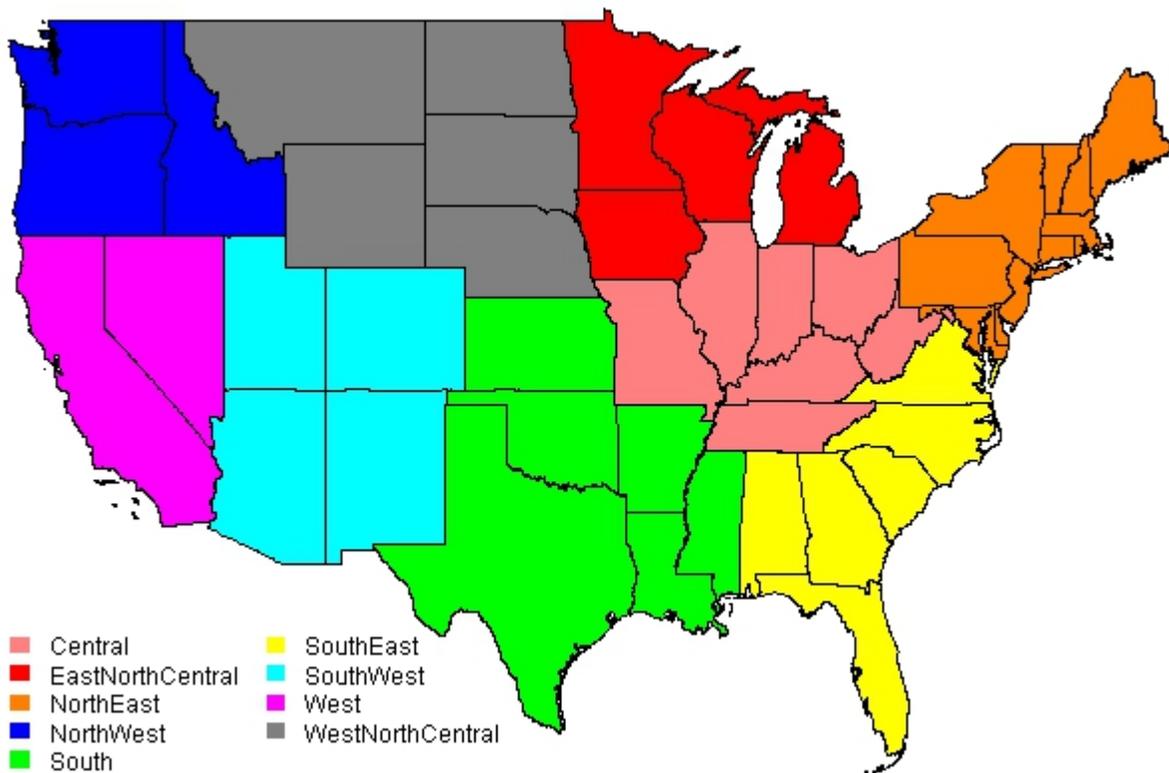


Figure 2B-1. Map of the 9 NOAA climate regions (Karl and Koss, 1984), color coded to match the subsequent scatter plots.

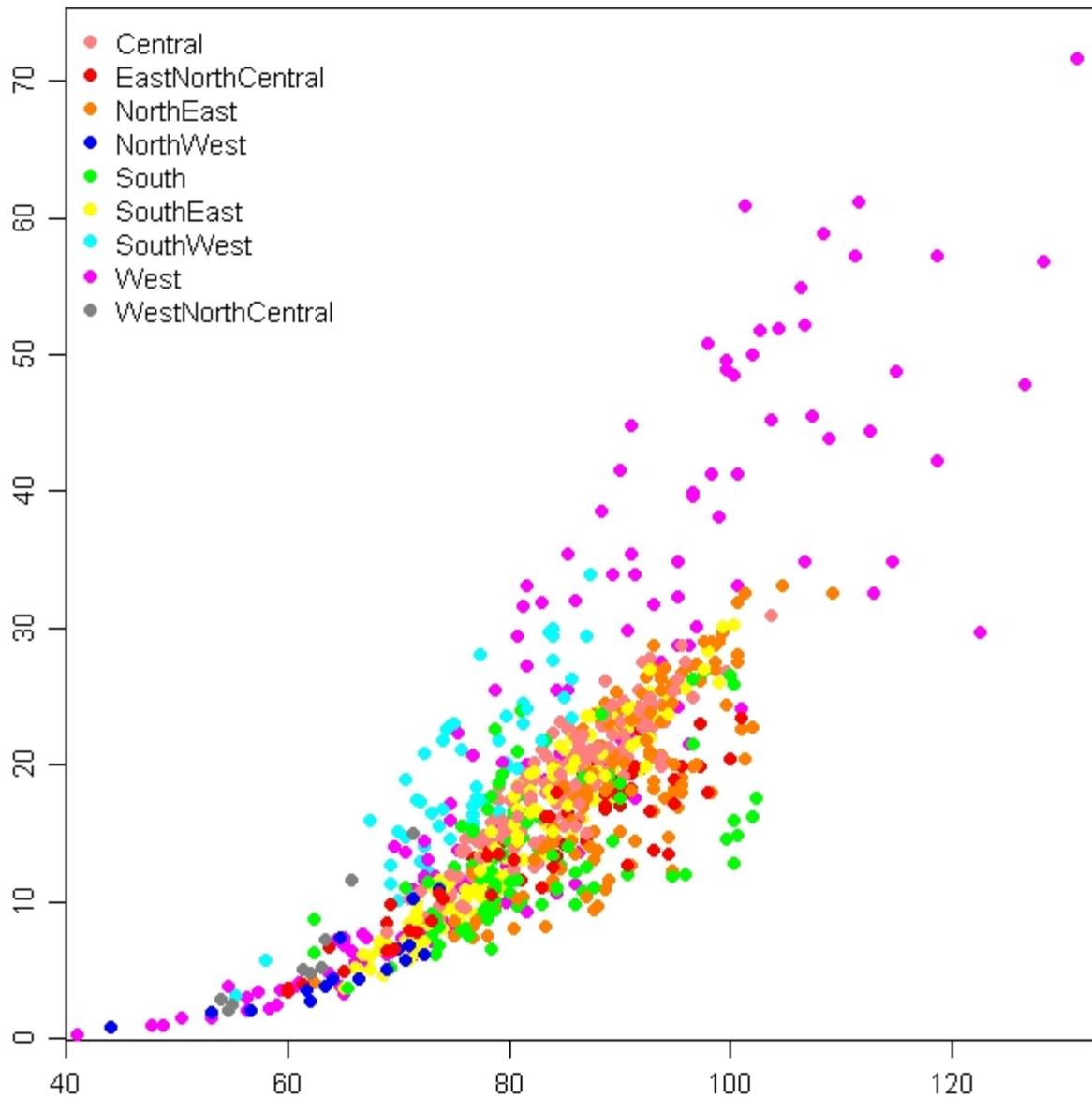


Figure 2B-2a. Design values for the current O₃ standard in ppb (x-axis) versus 3-year average W126 values in ppm-hrs (y-axis) based on ambient monitoring data for 2001-2003.

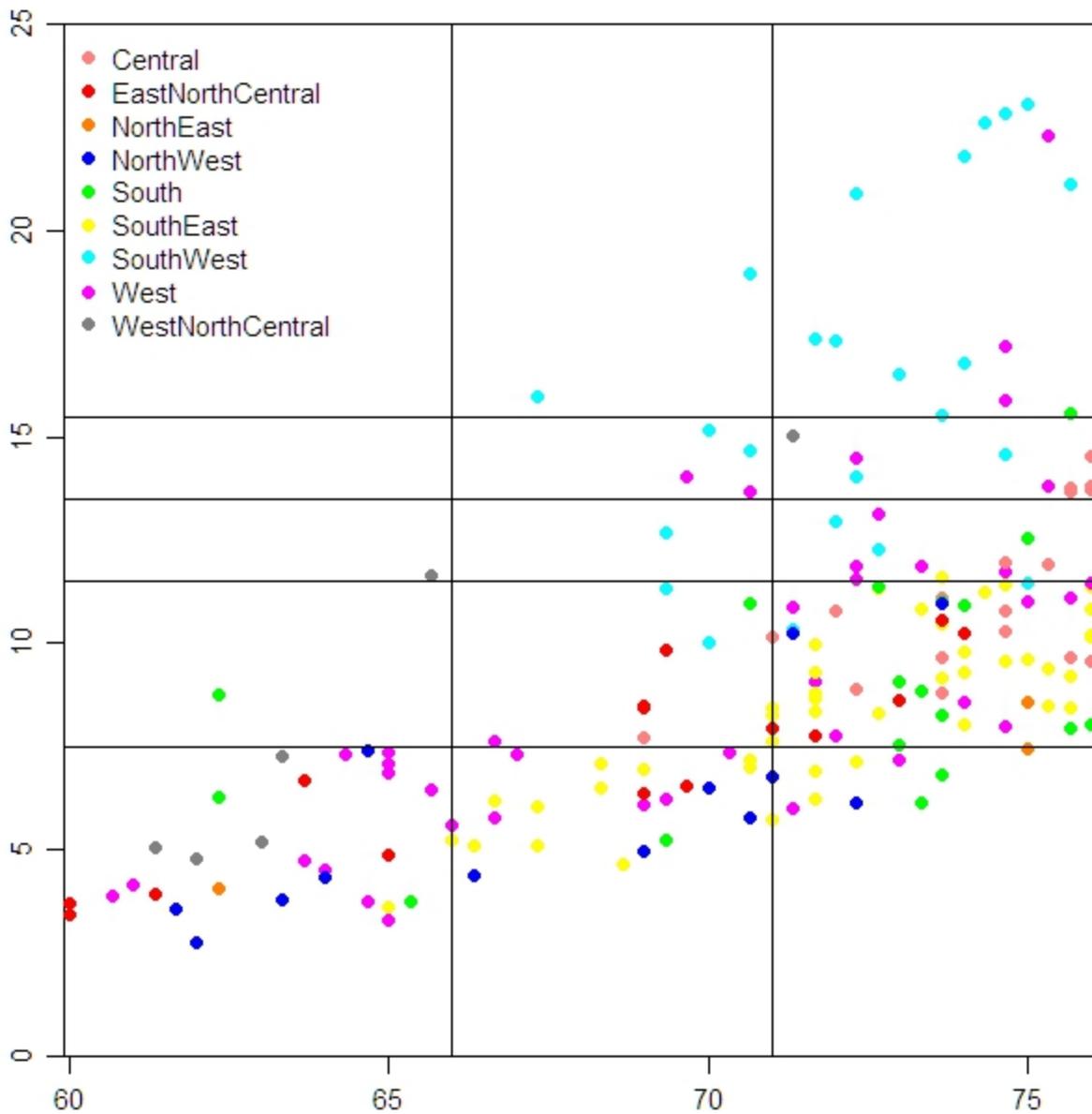


Figure 2B-2b. Design values for the current O₃ standard in ppb (x-axis) versus 3-year average W126 values in ppm-hrs (y-axis) based on ambient monitoring data for 2001-2003 with a focus on monitors with 2001-2003 design values below 75 ppb.

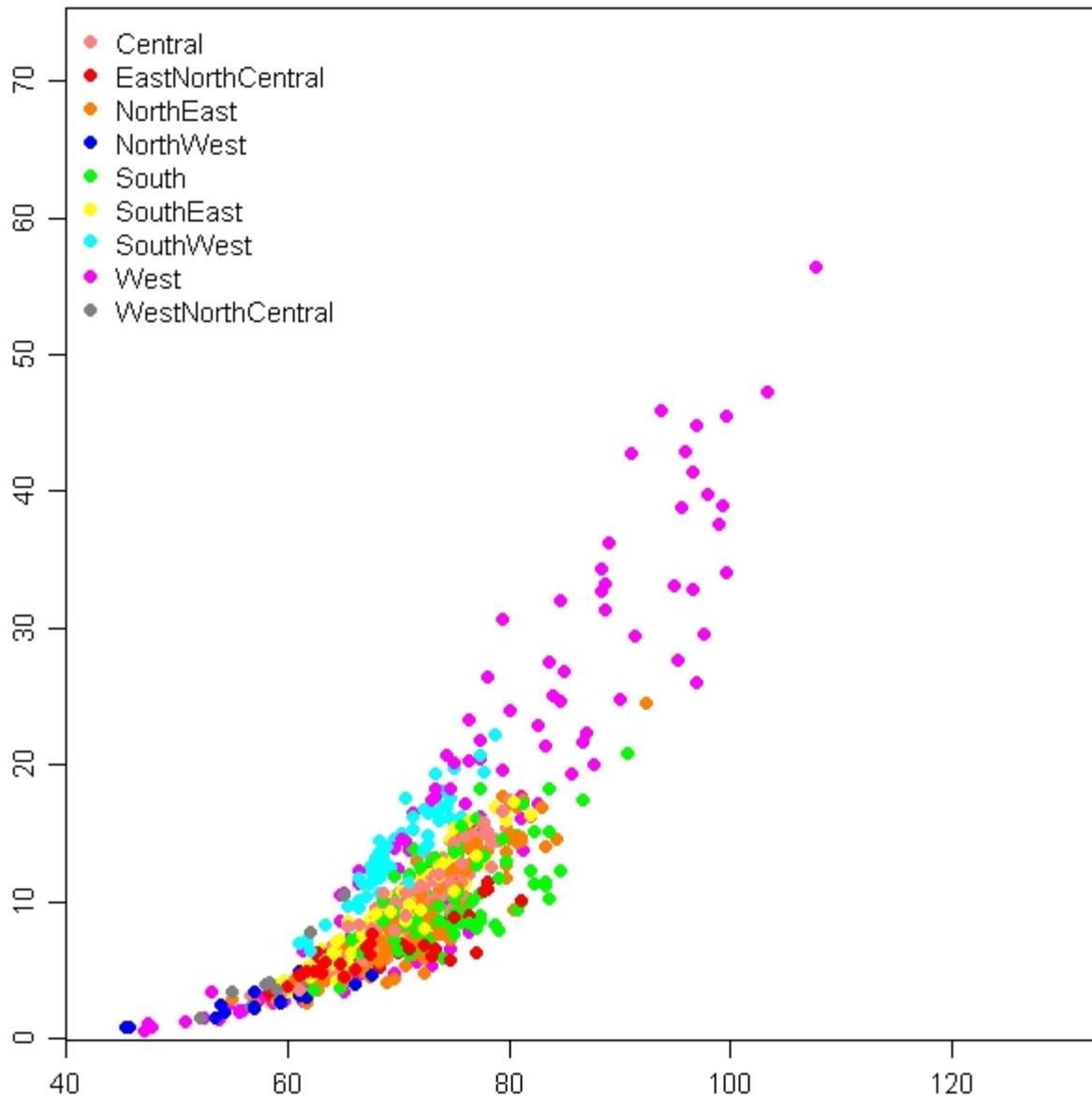


Figure 2B-3a. Design values for the current O₃ standard in ppb (x-axis) versus 3-year average W126 values in ppm-hrs (y-axis) based on ambient monitoring data for 2009-2011.

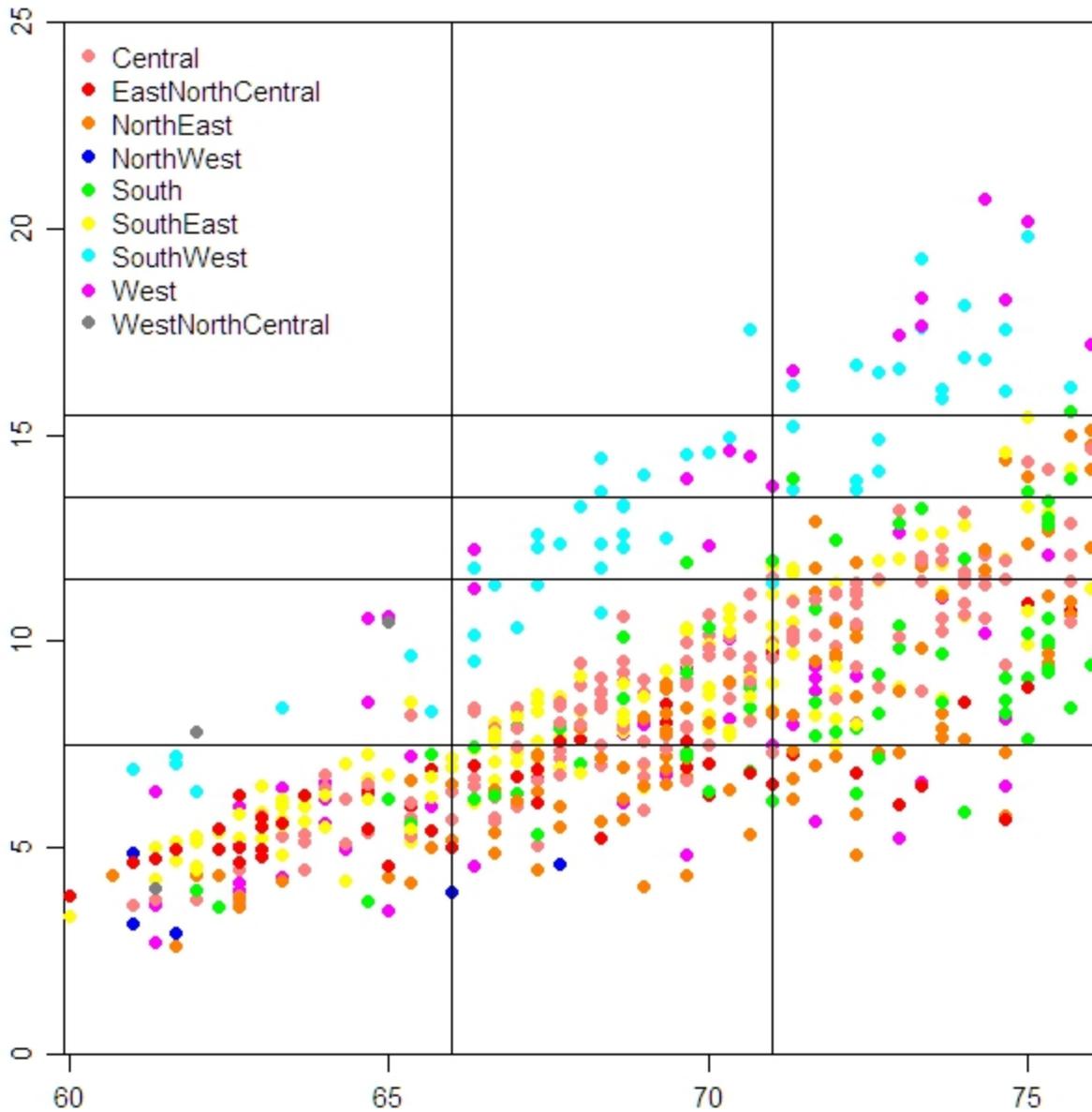


Figure 2B-3b. Design values for the current O₃ standard in ppb (x-axis) versus 3-year average W126 values in ppm-hrs (y-axis) based on ambient monitoring data for 2009-2011 with a focus on monitors with 2009-2011 design values below 75 ppb.

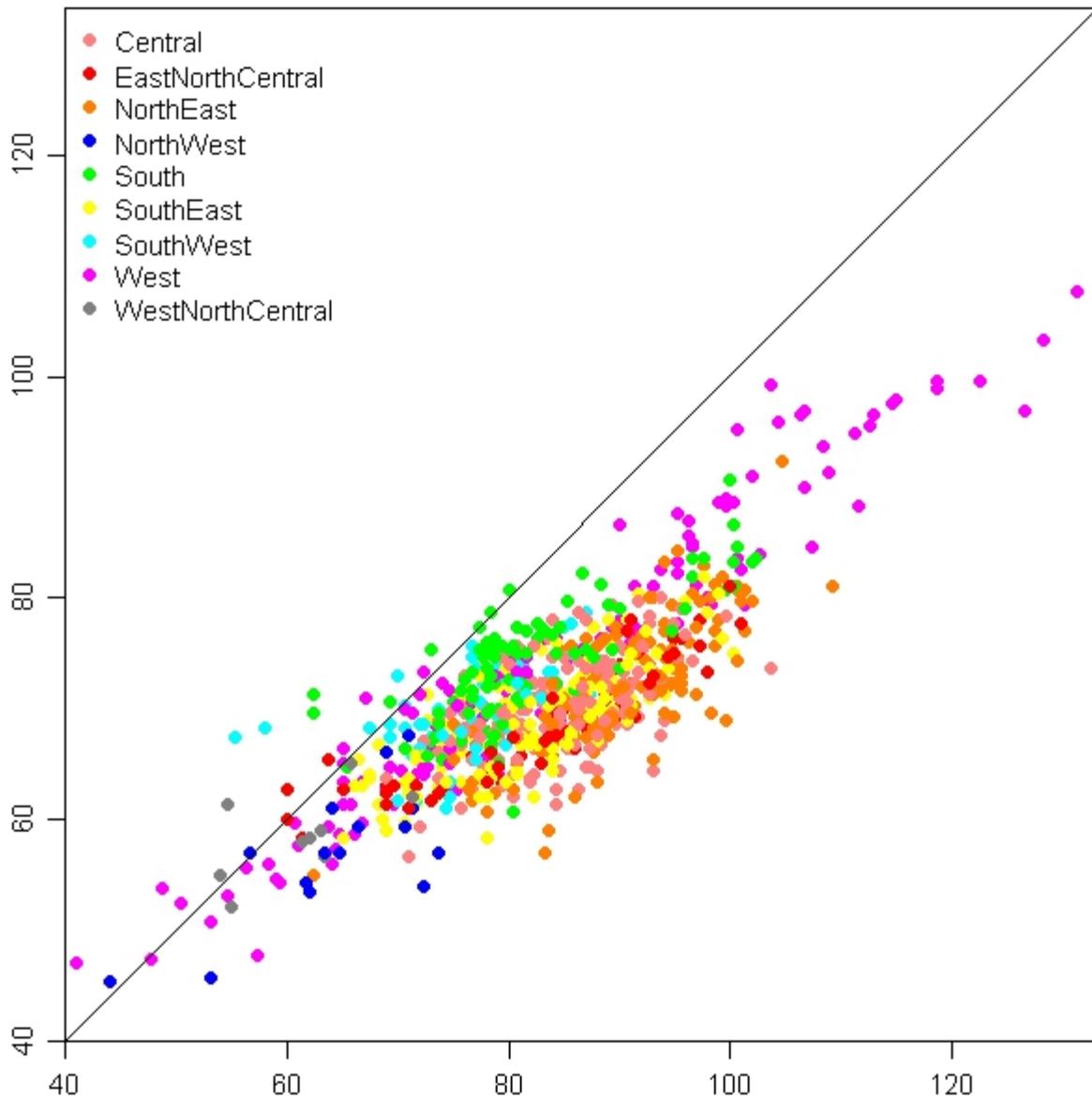


Figure 2B-4. Design values for the current O₃ standard in ppb based on ambient monitoring data for 2001-2003 (x-axis) versus 2009-2011 (y-axis).

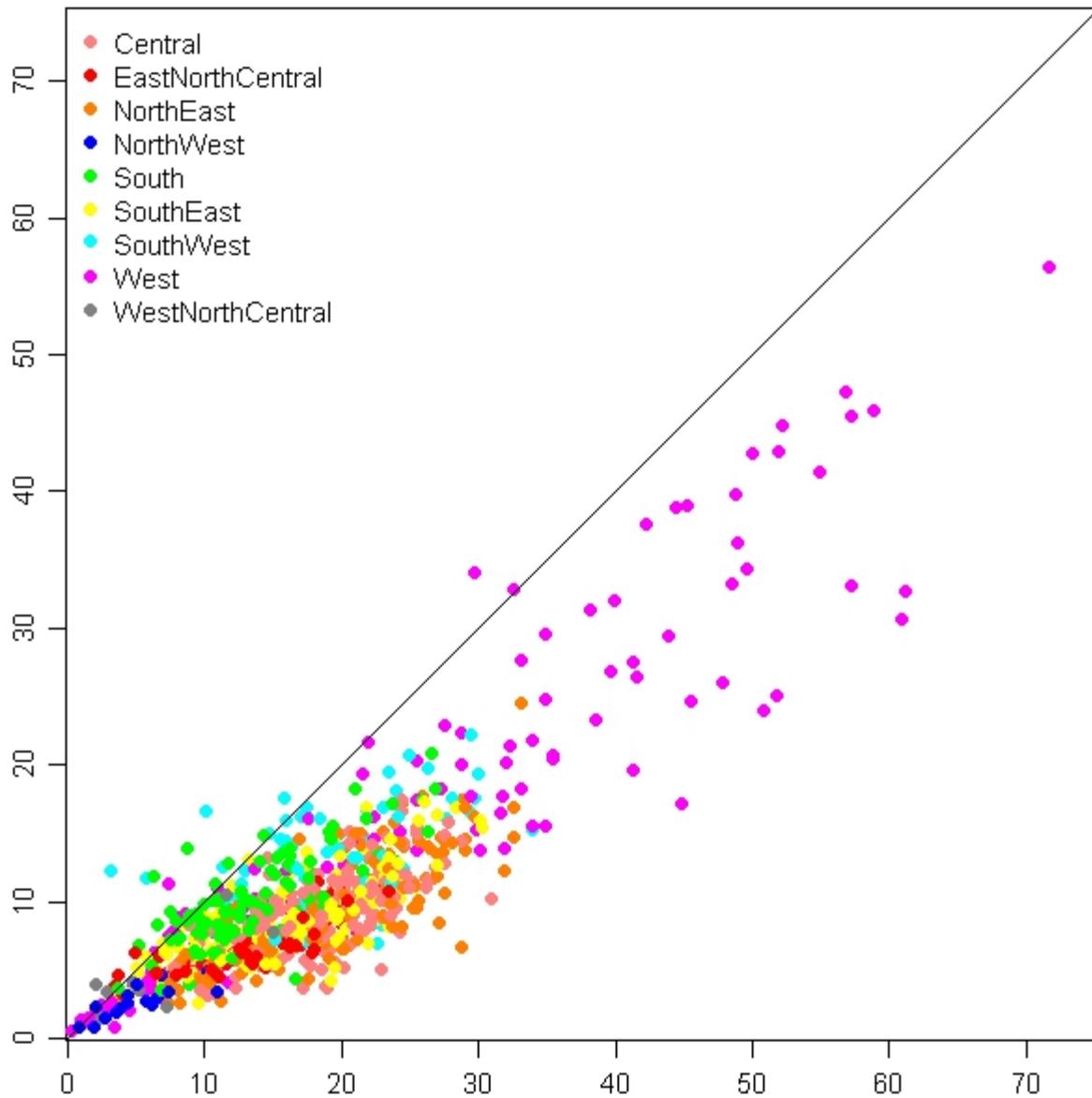


Figure 2B-5. Three-year average W126 values in ppm-hrs based on ambient monitoring data for 2001-2003 (x-axis) versus 2009-2011 (y-axis).

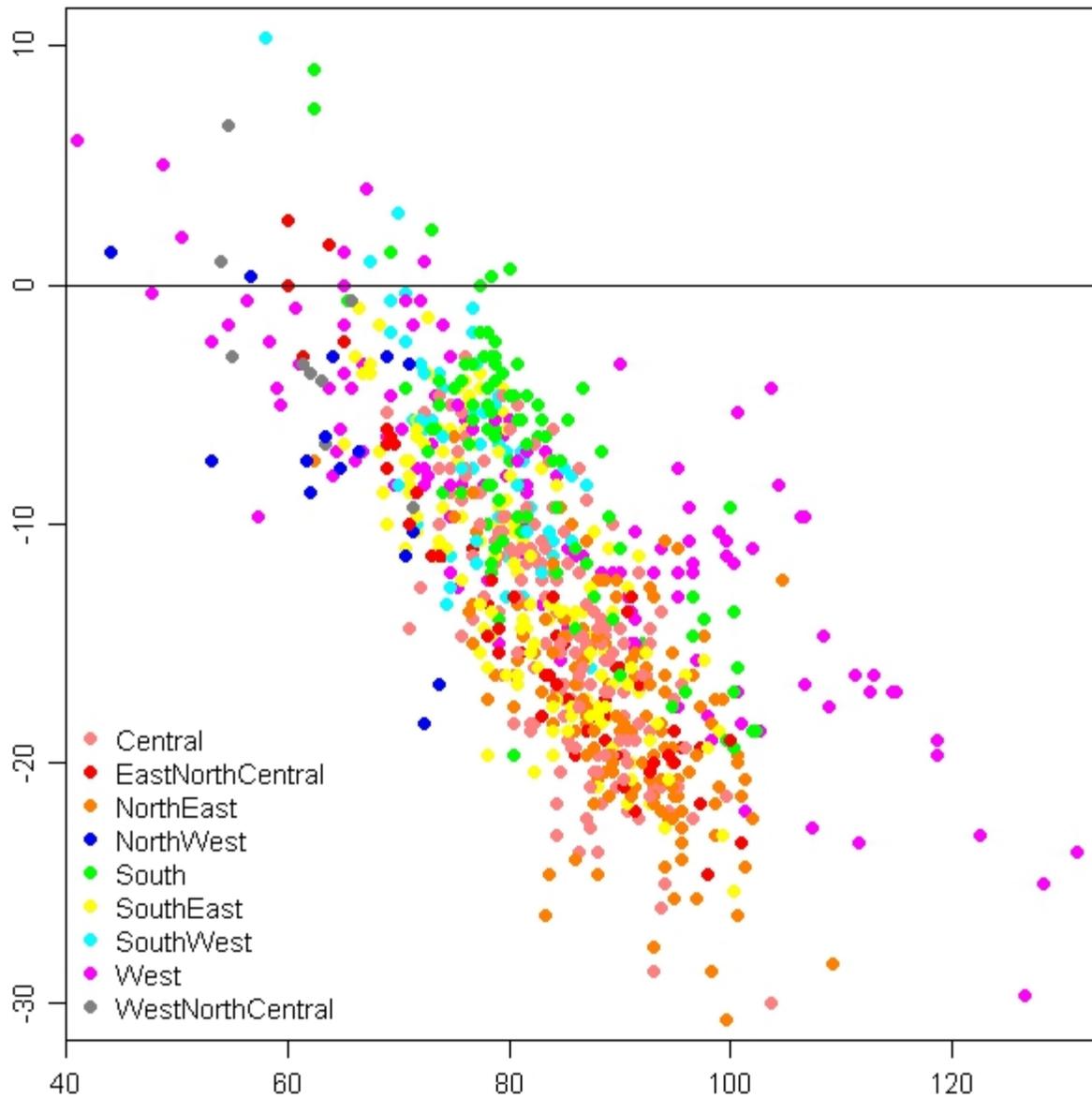


Figure 2B-6. Design values for the current O₃ standard in ppb based on ambient monitoring data for 2001-2003 (x-axis) versus unit (ppb) change in design values from 2001-2003 to 2009-2011 (y-axis).

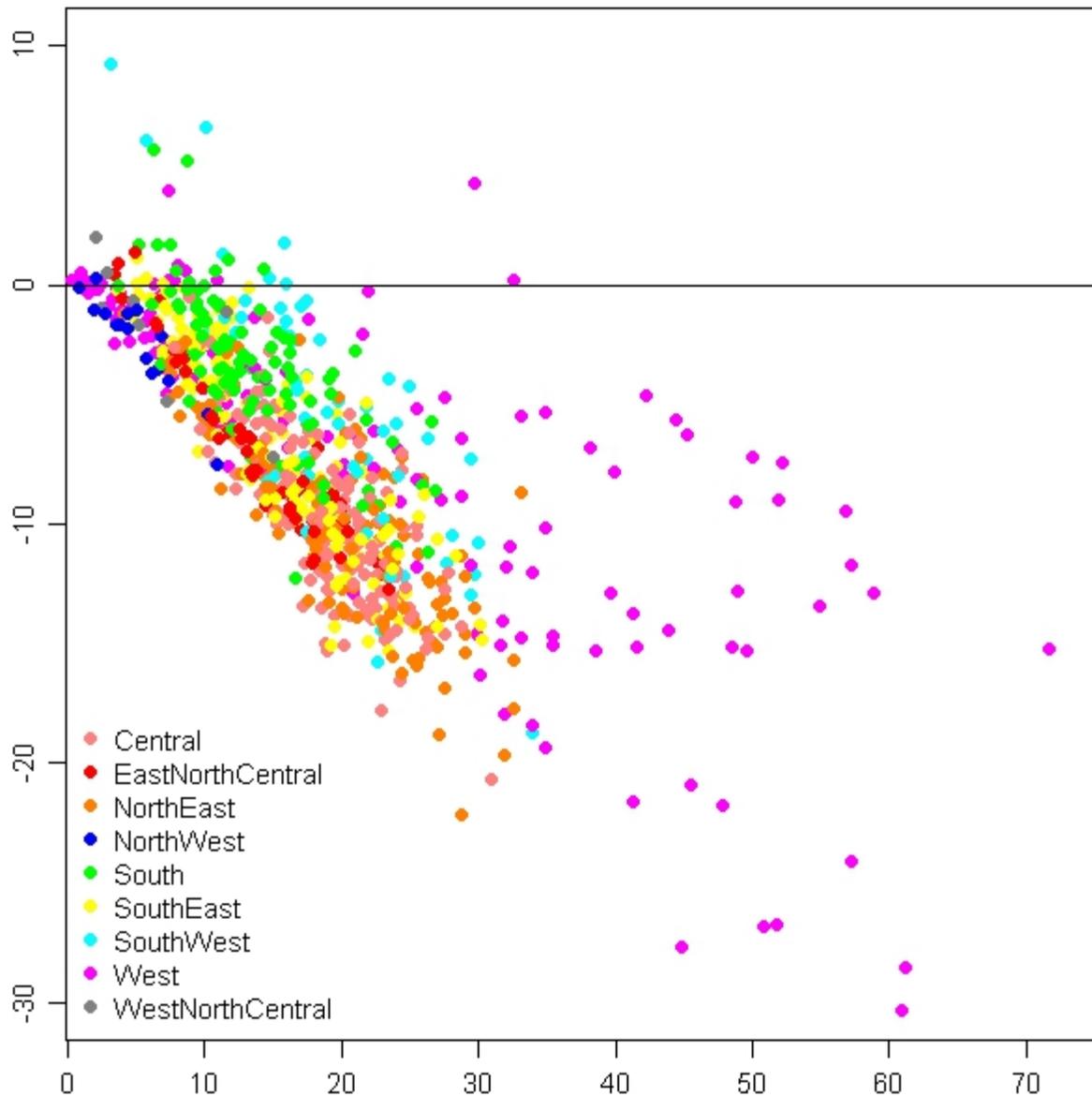


Figure 2B-7. Three-year average W126 values in ppm-hrs based on ambient monitoring data for 2001-2003 (x-axis) versus unit (ppm-hr) change in 3-year average W126 values from 2001-2003 to 2009-2011 (y-axis).

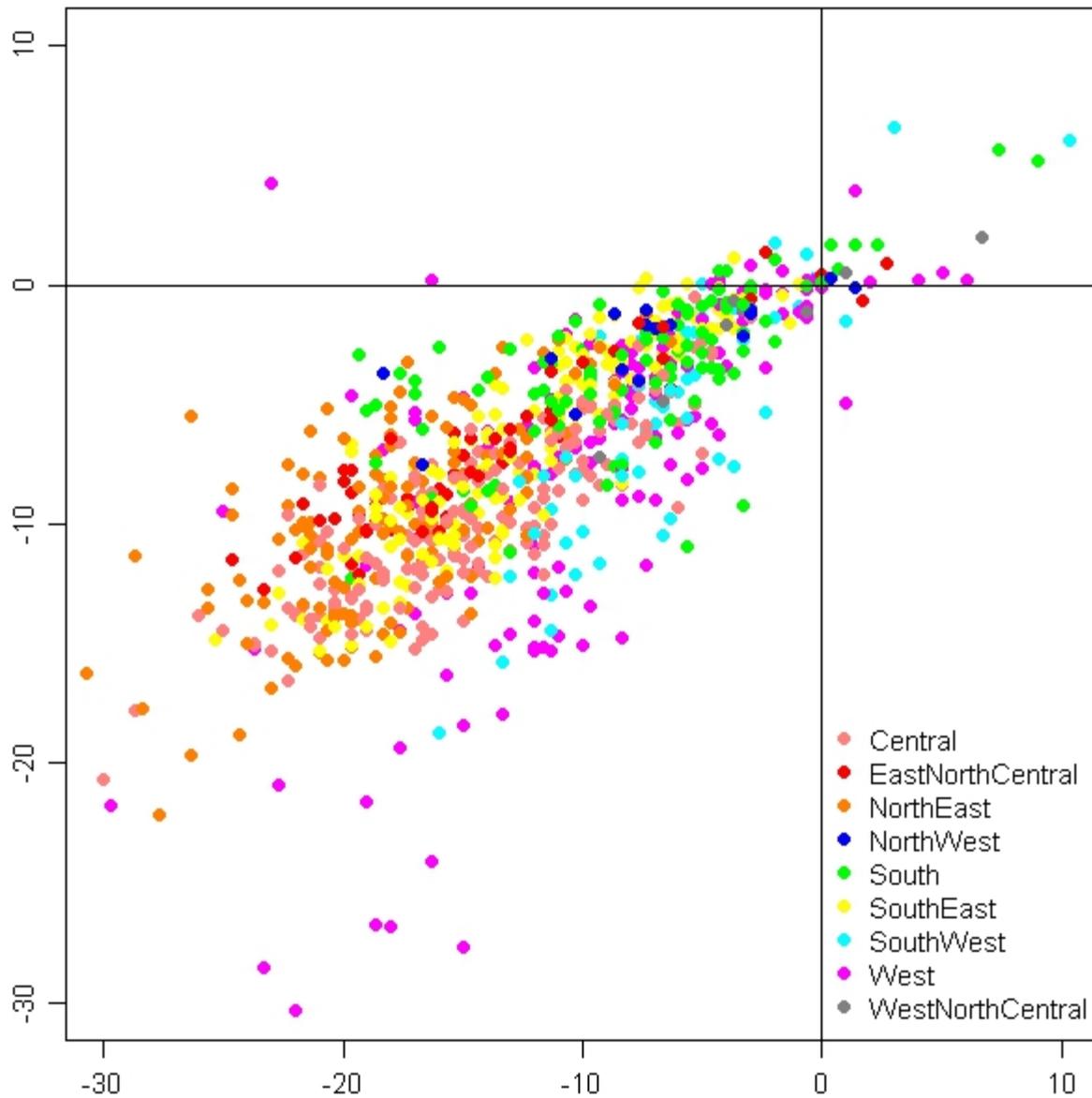
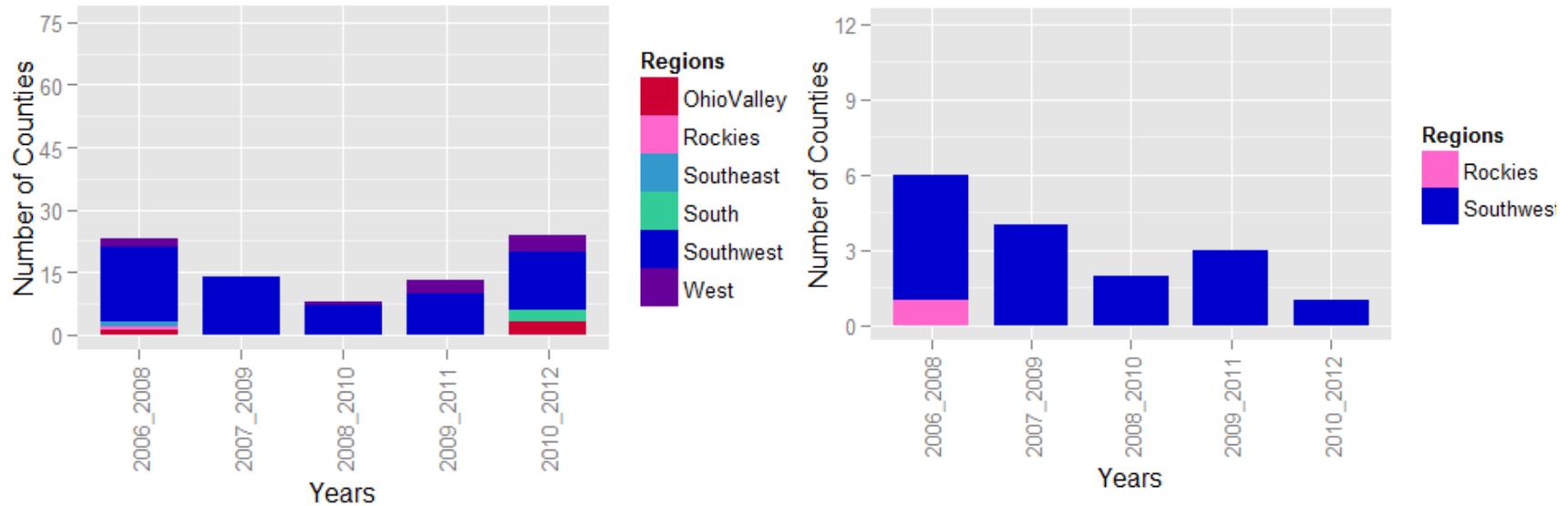


Figure 2B-8. Unit (ppb) change in design values for the current O₃ standard from 2001-2003 to 2009-2011 (x-axis) versus unit (ppm-hr) change in 3-year average W126 values from 2001-2003 to 2009-2011 (y-axis).

1



2

3 **Figure 2B-9. Number of counties where the 8-hour design value is meeting the current standard and 3-year average W126**
 4 **index value is greater than 15 ppm-hrs (left), and number of counties where the 8-hour design value is less than**
 5 **or equal to 70 ppb and 3-year average W126 index value is greater than 15 ppm-hrs (right)³.**
 6

7

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8 Karl, T.R. and Koss, W.J., 1984: "Regional and National Monthly, Seasonal, and Annual Temperature Weighted by Area, 1895-1983." Historical Climatology
 9 Series 4-3, National Climatic Data Center, Asheville, NC, 38 pp.

³ No counties in any of the studied 3-year periods were at or below a 3-year average of 4th highest daily maximum 8-hour averages of 65 ppb and also above a 3-year W126 index value of 15 ppm-hrs.

APPENDIX 2C

INTER-ANNUAL VARIABILITY IN W126 INDEX VALUES: COMPARING ANNUAL AND 3-YEAR AVERAGE METRICS (2008-2010)

2C.1 OVERVIEW

This appendix describes an analysis comparing values for a single-year or annual W126 metric to a W126 metric averaged over three consecutive years. The purpose of this analysis is to compare values based on a 3-year average of annual W126 indices to values based on a single annual W126 index. The deviations of the annual W126 index values in 2008, 2009, and 2010 from the 2008-2010 average W126 index values are presented.

2C.2 GENERAL DATA PROCESSING

The air quality data for this analysis originated from EPA's Air Quality System (AQS) data base, the official repository of ambient air measurements. The data used in this analysis consisted of W126 index values calculated from hourly ozone concentrations measured at 1082 ozone monitors nationwide. Ozone monitors must have submitted data to AQS for at least 75% days in their required ozone monitoring season in 2008, 2009, and 2010 to be included in the analysis.

2C.3 RESULTS & CONCLUSION

The figure below shows a scatter plot of the deviations in the annual W126 index from the 3-year average by monitor. The solid curves represent the average deviation in a moving window along the x-axis for each year. From this figure, it is apparent that the highest annual W126 index value occurred in 2008 for most monitoring locations, the lowest annual W126 index value occurred in 2009 for most monitoring locations, and the 2010 W126 index value was generally somewhere in between. It is also apparent that the inter-annual variability in the W126 index increases along with the 3-year average. For monitors with 3-year average W126 values near 15 ppm-hrs, the average deviation was +3.5 ppm-hrs in 2008 and -3.8 ppm-hrs in 2009. This represents a 1-year swing of -7.3 ppm-hrs.

The model-based air quality adjustments in the 2nd draft of the O₃ Welfare REA show that reducing NO_x emissions is effective for reducing 3-year average W126 levels. In Appendix 2B, the analyses based on ambient monitoring data also show that large-scale reductions in NO_x emissions are associated with lower W126 levels. Finally, the data analysis presented in this appendix shows that the inter-annual variability in the annual W126 index tends to decrease with decreasing W126 levels. Thus, it is expected that reductions in NO_x emissions will not only

result in lower 3-year average W126 levels, but also result in less inter-annual variability associated with annual W126 levels.

The W126 index is based on a logistic weighting function that increases the weights assigned to hourly ozone concentrations very rapidly. Hourly ozone concentrations of 50 parts per billion are given a weight of about 10% while concentrations of 80 parts per billion are given a weight of nearly 90%. The annual W126 index is calculated as a 3-month sum of weighted ozone concentrations during daylight hours, which amounts to a sum of roughly 1100 weighted hourly concentrations. Thus, even a modest change in the average daily ozone level may have a significant impact upon the annual W126 index. Since ozone formation is heavily influenced by meteorology, the inter-annual variability in meteorological conditions tends to cause a large inter-annual variability in the W126 index.

In conclusion, this evaluation indicates the extent to which a form for the secondary ozone standard that averages the annual W126 index values over three consecutive years might be expected to account for the annual variability in this index since the 3-year period would be expected to include year(s) below as well as above the 3-year average.

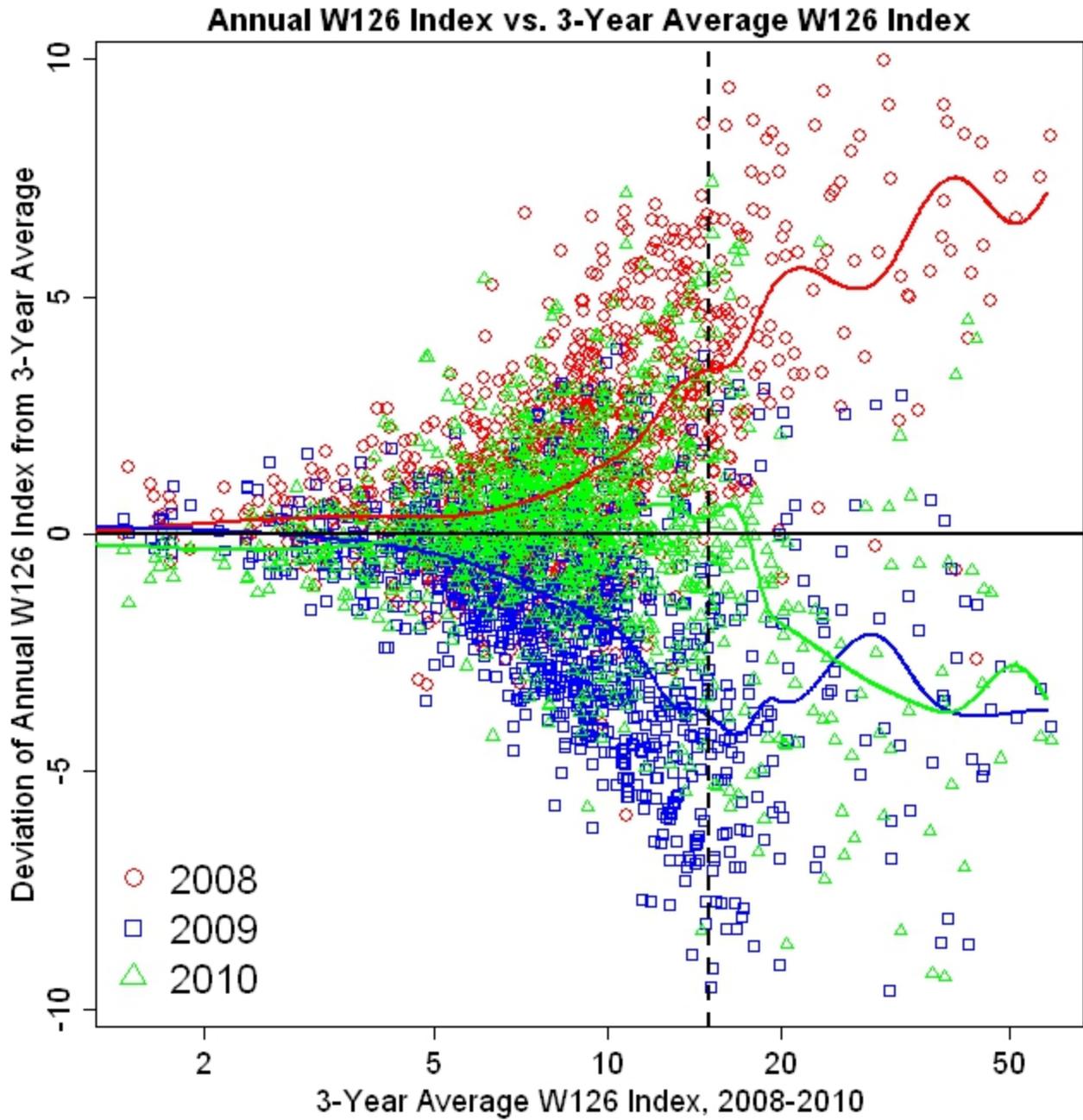


Figure 2C-1. Deviation of the annual W126 index values in 2008, 2009, and 2010 (y-axis) from the 3-year average W126 index value (x-axis).

APPENDIX 3A

RECENT STUDIES OF RESPIRATORY-RELATED EMERGENCY DEPARTMENT VISITS AND HOSPITAL ADMISSIONS

Hospital Admissions for All Respiratory Causes

The APHENA study (APHENA is for Air Pollution and Health: A European and North American Approach) analyzed air pollution and health outcome data from existing Canadian, European, and U.S. multi-city studies and examined the influence of varying model specification to control for season and weather (Katsouyanni et al., 2009). The U.S.-based portion of the APHENA study utilized the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) cohort which, for the Katsouyanni et al. (2009) analysis, comprised respiratory hospital admissions among individuals 65 years of age and older from 14 US cities with O₃ data from 1985-1994 (7 cities had summer only O₃ data). For the year round analysis, Katsouyanni et al. (2009) reported consistently positive, and statistically significant in models with 8 degrees of freedom per year (U.S. EPA, 2013, section 6.2.7.2), associations between 1-hour O₃ concentrations and respiratory hospital admissions across the datasets from the U.S., Canada, and Europe (U.S. EPA 2013, Figure 6-15).¹ In co-pollutant models adjusting for PM₁₀, O₃ effect estimates remained positive, though effect estimates were somewhat attenuated in the U.S. and European datasets, possibly due to the PM sampling schedule (U.S. EPA 2013, Figure 6-15). Effect estimates for the warm season were larger than for the year-round analysis in the Canadian dataset, but generally similar in magnitude to the year-round analysis in the U.S. and European datasets.

Several additional multicity studies examined respiratory disease hospital admissions in Canada and Europe. Cakmak et al. (2006) reported a statistically significant increase in respiratory hospital admissions in 10 Canadian cities (4.4% increase per 20 ppb increase in 24-hour average O₃, 95% CI: 2.2, 6.5%). In analyses of potential effect modifiers of the O₃-respiratory hospital admission relationship, individuals with an education level less than the 9th grade were found to be at greater risk. Dales et al. (2006) reported a 5.4% (95% CI: 2.9, 8.0%) increase in neonatal respiratory hospital admissions for a 20 ppb increase in 24-hour average O₃

¹The study by Katsouyanni et al. (2009) evaluated different statistical models. Although the investigators did not identify the model they deemed to be the most appropriate for comparing the results across study locations, they did specify that “overall effect estimates (i.e., estimates pooled over several cities) tended to stabilize at high degrees of freedom” (Katsouyanni et al., 2009). In discussing of the results of this study, the ISA focused on models with 8 degrees of freedom per year (US EPA, 2012a, section 6.2.7.2).

concentrations in 11 Canadian cities from 1986 to 2000. In contrast, Biggeri et al. (2005) did not detect an association between short-term O₃ exposure and respiratory hospital admissions in four Italian cities from 1990 to 1999.

In addition to the large multi-city studies discussed above, several smaller-scale studies have also reported associations with total respiratory hospital admissions. Specifically, Lin et al. (2008) reported a positive association between O₃ and pediatric (i.e., <18 years) respiratory admissions in an analysis of 11 geographic regions in New York state from 1991 to 2001, though results were not presented quantitatively. In co-pollutant models with PM₁₀, the authors reported that region-specific O₃ associations with respiratory hospital admissions remained relatively robust.

Cause-Specific Hospital Admissions

With regard to cause-specific respiratory outcomes, the limited evidence available in the last review indicated that the strongest findings were for ambient O₃ associated asthma and chronic obstructive pulmonary disease (COPD) respiratory hospital admissions (U.S. EPA 2013, 6.2.7.2). Since the last review, a few additional studies have investigated cause-specific respiratory admissions (i.e., COPD, asthma, pneumonia) in relation to O₃ exposure (Medina-Ramon et al, 2006; Yang et al., 2005; Zanobetti and Schwartz, 2006; Silverman and Ito, 2010).

Medina-Ramon et al. (2006) examined the association between short-term ambient O₃ concentrations and Medicare hospital admissions for COPD among individuals ≥ 65 years of age for COPD in 35 cities in the U.S. for the years 1986-1999. The authors reported an increase in COPD admissions for lag 0-1 day in the warm season for a 30 ppb increase in 8-h max O₃ concentrations. The authors found no evidence for such associations in cool season or in year round analyses. In a co-pollutant model with PM₁₀, the association between O₃ and COPD hospital admissions remained robust. In Vancouver from 1994-1998, a location with low ambient O₃ concentrations (U.S. EPA, 2013, Table 6-26), Yang et al. (2005) reported a statistically non-significant increase in COPD admissions per 20 ppb increase in 24-hour average O₃ concentrations. In two-pollutant models with every-day data for NO₂, SO₂, CO, and PM₁₀, O₃ risk estimates remained robust, though not statistically significant (U.S. EPA, 2013, Figure 6-20; Table 6-29). In addition, Wong et al. (2009) reported increased O₃-associated COPD admissions during periods of increased influenza activity in Hong Kong.

The ISA assessed a study that evaluated asthma-related hospital admissions in New York City (U.S. EPA, 2013, section 6.2.7.2) (Silverman and Ito, 2010). This study examined the association of 8-hour max O₃ concentrations with severe acute asthma admissions (i.e., those admitted to the Intensive Care Unit [ICU]) during the warm season in the years 1999 through

2006 (Silverman and Ito, 2010)). The investigators reported positive associations between O₃ and ICU asthma admissions for the 6- to 18-year age group for a 30 ppb increase in max 8-hour average O₃ concentrations, but little evidence of associations for the other age groups examined (<6 years, 19-49, 50+, and all ages). However, positive associations were observed for each age-stratified group and all ages for non-ICU asthma admissions, but again the strongest association was reported for the 6- to 18-years age group. In two-pollutant models, O₃ effect estimates for both non-ICU and ICU hospital admissions remained robust to adjustment for PM_{2.5}. In an additional analysis, using a smooth function, the authors examined whether the shape of the concentration-response curve for O₃ and asthma hospital admissions (i.e., both general and ICU for all ages) is linear. When comparing the curve to a linear fit line, the authors found that the linear fit was a reasonable approximation of the concentration-response relationship between O₃ and asthma hospital admissions, but the limited data density at relatively low O₃ concentrations contributes to uncertainty in the shape of the concentration-response relationship at the low end of the distribution of O₃ concentrations (U.S. EPA, 2013, Figure 6-16).

In contrast to COPD and asthma, the evidence for pneumonia-related admissions was less consistent. Medina-Ramon et al. (2006) examined the association between short-term ambient O₃ concentrations and Medicare hospital admissions among individuals ≥ 65 years of age for pneumonia. The authors reported an increase in pneumonia hospital admissions in the warm season for a 30 ppb increase in 8-hour max O₃ concentrations, with no evidence of an association in the cool season or year round. In two-pollutant models restricted to days for which PM₁₀ data was available, the association between O₃ exposure and pneumonia hospital admissions remained robust. In contrast, Zanobetti and Schwartz (2006) reported a decrease in pneumonia admissions for a 20 ppb increase in 24-hour average O₃ concentrations in Boston for the average of lags 0 and 1 day.

The magnitude of associations with respiratory-related hospital admissions may be underestimated due to behavioral modification in response to forecasted air quality (U.S. EPA, 2013, section 4.6.6). Recent studies (Neidell and Kinney, 2010; Neidell, 2009) conducted in Southern California demonstrates that controlling for avoidance behavior increases O₃ effect estimates for respiratory hospital admissions, specifically for children and older adults. This study shows that on days where no public alert warning of high O₃ concentrations was issued, there was an increase in asthma hospital admissions. Although only one study has examined averting behavior and this study is limited to the outcome of asthma hospital admissions in one location and time period (i.e., Los Angeles, CA for the years 1989-1997), it does provide preliminary evidence indicating that some epidemiologic studies may underestimate associations

between O₃ and health effects by not accounting for behavioral modification when public health alerts are issued.

Emergency Department Visits for All Respiratory Causes

A large single-city study conducted in Atlanta by Tolbert et al. (2007), and subsequently reanalyzed by Darrow et al. (2011) using different air quality data and evaluating associations with different metrics, provides evidence for associations between short-term exposures to ambient O₃ concentrations and respiratory emergency department visits. Tolbert et al. (2007) reported an increase in respiratory emergency department visits for a 30 ppb increase in 8-hour max O₃ concentrations during the warm season. In copollutant models with CO, NO₂, and PM₁₀, limited to days in which data for all pollutants were available, associations between O₃ and respiratory emergency department visits remained positive, but were attenuated. Darrow et al. (2011) reported the strongest associations with respiratory emergency department visits for 8-hour daily max, 1-hour daily max, and day-time O₃ exposure metrics (all associations positive and statistically significant), while positive, but statistically non-significant, associations were reported with 24-hour average and commuting period exposure metrics. In addition, a negative association was observed when using the night-time exposure metric (U.S. EPA, 2013, Figure 6-17). The results of Darrow et al. (2011) suggest that averaging over nighttime hours may lead to smaller O₃ effect estimates for respiratory emergency department visits due to dilution of relevant O₃ concentrations (i.e., the higher concentrations that occur during the daytime); and potential negative confounding by other pollutants (e.g., CO, NO₂) during the nighttime hours (U.S. EPA, 2013, section 6.2.7.3)

Cause-Specific Emergency Department Visits

In evaluating asthma emergency department visits in an all-year analysis, a Canadian multi-city study (Stieb et al., 2009) reported that 24-hour O₃ concentrations were positively associated with emergency department visits for asthma at lag 1 and lag 2. Though the authors did not present seasonal analyses, they stated that no associations were observed with emergency department visits in the winter season, suggesting that the positive associations reported in the all-year analysis were due to the warm season (Stieb et al., 2009). In addition to asthma, the authors reported that O₃ was positively associated with COPD emergency department visits in all-year analyses, but that associations with COPD visits were statistically significant only for the warm season (i.e., April-September).

Several single-city studies have also provided evidence for positive associations between asthma emergency department visits and ambient O₃ concentrations. Ito et al. (2007) reported positive and statistically significant associations with asthma emergency department visits in New York City during the warm season, and an inverse association in the cool season, for a 30 ppb increase in 8-hour max O₃ concentrations. In two-pollutant models with PM_{2.5}, NO₂, SO₂, and CO, the authors found that O₃ risk estimates were not substantially changed during the warm season (U.S. EPA, 2013, Figure 6-20; Table 6-29).

Strickland et al. (2010) examined the association between O₃ exposure and pediatric asthma emergency department visits (ages 5-17 years) in Atlanta using air quality data over the same years as Darrow et al. (2011) and Tolbert et al. (2007), but using population-weighting to combine daily pollutant concentrations across monitors. Strickland et al. (2010) reported an increase in emergency department visits for a 30 ppb increase in 8-hour max O₃ concentrations in an all-year analysis. In seasonal analyses, stronger associations were observed during the warm season (i.e., May-October) than the cold season. In co-pollutant analyses that included CO, NO₂, PM_{2.5} elemental carbon, or PM_{2.5} sulfate, Strickland et al. (2010) reported that O₃ risk estimates were not substantially changed. The authors also examined the concentration-response relationship between O₃ exposure and pediatric asthma emergency department visits and reported that positive associations with O₃ persist at 8-hour ambient O₃ concentrations (3-day average of 8-hour daily max concentrations) at least as low as 30 ppb.

In a single-city study conducted in Seattle, WA, Mar and Koenig (2009) examined the association between O₃ exposure and asthma emergency department visits for children (< 18) and adults (≥ 18). For children, positive and statistically significant associations were reported across multiple lags, with the strongest associations observed at lag 0 and lag 3. Ozone was also found to be positively associated with asthma emergency department visits for adults at all lags, except at lag 0. The slightly different lag times for children and adults suggest that children may be more immediately responsive to O₃ exposures than adults (Mar and Koenig, 2009).

In addition to the U.S. single-city studies discussed above, a single-city study conducted in Alberta, Canada (Villeneuve et al., 2007) provides support for the findings from Stieb et al. (2009), but also attempts to identify those lifestages at greatest risk for O₃-associated asthma emergency department visits. Villeneuve et al. reported an increase in asthma emergency department visits in an all-year analysis across all ages with associations being stronger during

the warmer months. When stratified by age, the strongest associations were observed in the warm season for individuals 5-14 and 15-44. These associations were not found to be confounded by the inclusion of aeroallergens in age-specific models.

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APPENDIX 3B: AMBIENT O₃ CONCENTRATIONS IN LOCATIONS OF HEALTH STUDIES

Annual 4th highest daily maximum O₃ concentrations for all U.S. monitors operating during the 1975 – 2010 period were retrieved from EPA's AQS database. These data were used to calculate O₃ design values for the 2008 8-hour O₃ NAAQS of 0.075 parts per million (ppm) according to 40 CFR part 50, Appendix P. Design values were calculated for each O₃ monitor and each 3-year period between 1975-1977 and 2008-2010 whenever sufficient data were available.

Ozone Design Values in Study Locations

Ozone monitors were matched to 200 health study locations on a case-by-case basis, using the following guidelines:

- 1) Areas defined by a Metropolitan Statistical Area (MSA) were matched with O₃ monitors by incorporating all of the monitors located in within the MSA boundaries.
- 2) Areas not represented by a MSA were matched to monitors by incorporating all of the monitors in the county central to location of the health study area.
- 3) In some cases, EPA staff made judgment calls. For example, EPA staff matched the Los Angeles, CA study area to the Los Angeles-Long Beach-Santa Ana, CA MSA defined by Los Angeles County, CA and Orange County, CA, while the Long Beach, CA study area was matched to Los Angeles County, CA and the Santa Ana, CA study area was matched to Orange County, CA.

In some cases, EPA staff matched two or more study areas to the same county or MSA. In other cases, a study area was matched to a MSA and another study area was matched to a county within the same MSA. For each 3-year period, the area design value was determined by the monitor reporting the highest design value in the county or MSA. This has two implications for the design values:

- 1) Design values are sensitive to changes in the monitoring network. The addition or discontinuation of O₃ monitors in an area may cause increases or decreases in the design value trend.
- 2) Only valid design values are reported. According to 40 CFR Part 50, Appendix P, design values greater than the level of the NAAQS (0.075 ppm) are always valid, while design values less than or equal to 0.075 ppm must have 75% annual data completeness in order to be valid. This may cause anomalies in the design value trend. For example, a monitor may report a valid design value based on as few as 12 days of data, or a monitor with less than 75% annual data completeness may have valid design values in some 3-year periods and invalid design values in others.

We have identified design values for the U.S. O₃ epidemiologic studies identified in Sections 3.1.4.2 and 3.1.4.3 of the second draft Policy Assessment (see Tables 3D-1 to 3D-4). For each study, design values were identified for the cities evaluated and for the years over which the study was conducted. These design values are reported in tables A-1 to A-22 of the Wells et al, 2012 memo “Analysis of Recent U.S. Ozone Air Quality Data to Support the O₃ NAAQS Review and Quadratic Rollback Simulations to Support the First Draft of the Risk and Exposure Assessment”.

Table 3B-1. Number of Study Cities from Multicity Epidemiologic Studies of Hospital Admissions and Emergency Department Visits Using Short-Term O₃ Metrics with 3-Year Averages of Annual 4th Highest Daily Maximum 8-hour O₃ Concentrations ≤ 75 ppb¹

Study	Location	Endpoint ²	% Increase (95% CI) ³	# Study Cities ≤ 75 ppb over entire study period
All-year				
Medina-Ramon et al. (2006)	36 U.S. cities	COPD HA	All year: 0.24 (-0.78, 1.21) Warm season: 1.63 (0.48, 2.85)	4
		Pneumonia HA	All year: 1.81 (-0.72, 4.52) Warm Season: 2.49 (1.57, 3.47)	
Katsouyanni et al. (2009)	14 U.S. cities	Respiratory HA	All Year: 2.38 (0.00, 4.89) Warm Season: 2.14 (-0.63, 4.97)	2
	12 Canadian Cities	Respiratory HA	All year: 2.4 (0.51, 4.40) Warm Season: 4.1 (1.4, 6.8)	10
Dales et al. (2006)	11 Canadian cities	Respiratory HA	All year: 5.41 (2.88, 7.96)	7
Stieb et al. (2009)	7 Canadian cities	Asthma ED	All year: 3.48 (0.33, 6.76)	5
		COPD ED	All year: 4.03 (-0.54, 8.62) Warm season: 6.76 (0.11, 13.9)	
Cakmak et al. (2006)	10 Canadian cities	Respiratory HA	All year: 4.38 (2.19, 6.46)	7

¹ For U.S. study areas, we used EPA’s Air Quality System (AQS) (<http://www.epa.gov/ttn/airs/airsaqs/>) to identify 8-hour O₃ concentrations. For Canadian study areas, we used publically available air quality data from the Environment Canada National Air Pollution Surveillance Network (<http://www.etc-cte.ec.gc.ca/napsdata/main.aspx>). We followed the data handling protocols for calculating design values as detailed in 40 CFR Part 50, Appendix P.

²HA stands for hospital admissions; ED stands for emergency department visits.

³Ozone effect estimates are taken from Table 6-28 in the ISA (U.S. EPA, 2013a).

Table 3B-2. Number of Study Cities from Multicity Epidemiologic Studies of Mortality Using Short-Term O₃ Metrics with 3-Year Averages of Annual 4th Highest Daily Maximum 8-hour O₃ Concentrations ≤ 75 ppb

Study	Location	Endpoint	% Increase (95% CI) ⁴	# Study Cities ≤ 75 ppb over entire study period
All-year				
Schwartz (2005)	14 U.S. cities	Non-accidental mortality	0.76 (0.13, 1.40)	1
Bell et al. (2007)	98 U.S. communities	Non-accidental mortality	0.64 (0.34, 0.92)	6
Bell and Dominici (2008)	98 U.S. communities	Non-accidental mortality	1.04 (0.56, 1.55)	6
Katsouyanni et al. (2009)	90 U.S. cities	Non-accidental mortality	3.02 (1.10, 4.89)	6
		Respiratory mortality	2.54 (-3.32, 8.79) for <75; 1.10 (-6.48, 9.21) for 75+	
		Cardiovascular mortality	3.83 (-0.16, 7.95) for < 75; 2.30 (-1.33, 6.04) for 75+	
Bell et al. (2004)	95 U.S. communities	Non-accidental mortality	1.04 (0.54, 1.55)	6
Katsouyanni et al. (2009)	12 Canadian cities	Non-accidental mortality	0.73 (0.23, 1.20)	8
		Respiratory mortality	0.13 (-1.60, 1.90); -0.60 (-2.70, 1.60) for 75+	
		Cardiovascular mortality	0.87 (-0.35, 2.10) for <75; 1.1 (0.10, 2.20) for 75+	
Warm Season				
Schwartz (2005)	14 U.S. Cities	Non-accidental mortality	1.00 (0.30, 1.80)	1
Zanobetti and Schwartz (2008a)	48 U.S. cities	Non-accidental mortality	1.51 (1.14, 1.87)	4
Zanobetti and Schwartz (2008b)	48 U.S. cities	Non-accidental mortality	1.60 (0.84, 2.33)	4
		Respiratory mortality	2.51 (1.14, 3.89)	
		Cardiovascular mortality	2.42 (1.45, 3.43)	

⁴Ozone effect estimates are taken from Tables 6-42 and 6-53 in the ISA (U.S. EPA, 2013a).

Medina-Ramon and Schwartz (2008)	48 U.S. cities	Non-accidental mortality	1.96 (1.14, 2.82)	4
Franklin and Schwartz (2008)	18 U.S. communities	Non-accidental mortality	1.79 (0.90, 2.68)	1
Katsouyanni et al. (2009)	90 U.S. cities	Non-accidental mortality	3.83 (1.90, 5.79)	6
		Respiratory mortality	4.40 (-2.10, 11.3); 4.07 (-4.23, 13.0) for 75+	
		Cardiovascular mortality	6.78 (2.70, 11.0) for <75; 3.18 (-0.47, 6.95) for 75+	
Bell et al. (2004)	95 U.S. communities	Non-accidental mortality	0.78 (0.26, 1.30)	6
Katsouyanni et al. (2009)	12 Canadian Cities	Non-accidental mortality	0.42 (0.16, 0.67)	8

Table 3B-3. Number of Study Cities from Single-City Epidemiologic Studies Using Short-Term O₃ Metrics with 3-Year Averages of Annual 4th Highest Daily Maximum 8-hour O₃ Concentrations < 75 ppb

Study	Location	Age	Endpoint	% Increase (95% CI) ⁵	# Study Cities ≤ 75 ppb over entire study period
All-year					
Strickland et al. (2010)	Atlanta	Children	Asthma ED visits	6.38 (3.19, 9.57)	0
Warm season					
Ito et al. (2007)	New York City	All	Asthma ED visits	16.9 (10.9, 23.4)	0
Darrow et al. (2011)	Atlanta	All	Respiratory ED visits	2.08 (1.25, 2.91)	0
Tolbert et al. (2007)	Atlanta	All	Respiratory ED visits	3.90 (2.70, 5.20)	0
Strickland et al. (2010)	Atlanta	Children	Asthma ED visits	8.43 (4.42, 12.7)	0
Silverman and Ito (2010)	New York City	6 to 18 years	Asthma HA	28.2 (15.3, 41.5)	0
		All	Asthma HA	12.5 (8.27, 16.7)	0
Mar and Koenig (2009)	Seattle, WA	18+	Asthma ED visits	19.1 (3.00, 40.5)	1

⁵Ozone effect estimates are taken from Table 6-28 in the ISA (U.S. EPA, 2013a).

Table 3B-4 Number of Study Cities from Epidemiologic Studies Using Long-Term O₃ Metrics with 3-Year Averages of Annual 4th Highest Daily Maximum 8-hour O₃ Concentrations > 75, 70, 65, or 60 ppb

Study	Number of Cities	Study Period	Number (Percent) of Cities with Maximum conc >75	Number (Percent) of Cities with Maximum conc >70	Number (Percent) of Cities with Maximum conc >65	Number (Percent) of Cities with Maximum conc >60
Islam et al. 2008, 2009	11 ⁶	1994-2003	11 (100%)	11 (100%)	11 (100%)	11 (100%)
Jerrett et al. 2009	94 ⁷	1977-2000	91 (97%)	92 (98%)	93 (99%)	94 (100%)
Lin et al. 2008	26 ⁸	1991-2001	24 (92%)	24 (92%)	26 (100%)	26 (100%)
Meng et al. 2010	7	1997-2002	7 (100%)	7 (100%)	7 (100%)	7 (100%)
Moore et al. 2008	8	1980-2000	8 (100%)	8 (100%)	8 (100%)	8 (100%)
Salam et al. 2009	11 ⁹	1992-2005	12 (100%)	12 (100%)	12 (100%)	12 (100%)
Zanobetti & Schwartz 2011	105	1985-2006	100 (95%)	104 (99%)	104 (99%)	104 (99%)

⁶ Study authors included 12 cities in their analyses, air quality data that met completeness criteria described above were available for 11 cities

⁷ Study authors included 96 cities in their analyses, air quality data that met completeness criteria described above were available for 94 cities

⁸ Study authors included 27 cities in their analyses, air quality data that met completeness criteria described above were available for 26 cities

⁹ Study authors included 12 cities in their analyses, air quality data that met completeness criteria described above were available for 11 cities

Relationship between average 24-hour and highest 8-hour O₃ concentrations for cities analyzed by Bell et al. (2006)

Bell et al. (2006) reported associations between mortality and 24-hour average O₃ concentrations (i.e., averaged across monitors in cities with multiple monitors) in a multi-city study of 98 U.S. cities. Positive associations persisted in a series of analyses that restricted O₃ concentrations to those below various cut points (cut points ranged from 5 to 60 ppb in 5 ppb increments). To facilitate consideration of these cut point analyses for the second draft of the O₃ Policy Assessment, so as to match the form and averaging time of the existing primary standard, we evaluated the relationship between 24-hour average O₃ concentrations, averaged across monitors in cities with multiple monitors, and the highest 8-hour daily maximum O₃ concentrations among the individual monitors in each city.

EPA staff retrieved daily 24-hour average and 8-hour maximum O₃ concentrations reported to EPA by monitors in the 98 study areas defined in Bell et al. (2006) during the 1987-2012 period from EPA's Air Quality System (AQS) database. Next, EPA staff obtained the study area boundaries from the published study (Bell et al., 2006) and used them to determine which O₃ monitoring sites were associated with each study area. The 24-hour average O₃ concentrations were averaged spatially across all available monitors within each study area on each day where monitoring data were collected. Next, days where the area-wide 24-hour average concentration (i.e., averaged spatially across monitors in areas with multiple monitors) was greater than 60 ppb were removed from the data. Based on the data remaining (i.e., with 24-hour average concentrations of 60 ppb or below), the annual 4th highest 8-hour daily maximum concentrations were identified for each study area and for each year from 1987-2012 (Table 3D-3). This process was repeated by further removing days with area-wide 24-hour average concentrations greater than 55 ppb, 50 ppb, etc., down to 5 ppb, and re-calculating the same statistics after each removal. The resulting dataset consisted of the annual 4th highest 8-hour daily maximum concentrations for all study areas.

Table 3B-5 Number of Study Cities with 4th Highest 8-hour Daily Maximum Concentrations Greater Than the Level of the Current Standard and Potential Alternative Standards For Various Cut-Point Analyses Presented in Bell et al. (2006)¹⁰

	Cut-point for 2-day moving average across monitors and cities (24-h avg)									
	20	25	30	35	40	45	50	55	60	All
Number (%) of Cities with 4th highest >75 (any year; 1987-2000)	0 (0%)	0 (0%)	12 (12%)	52 (53%)	77 (79%)	88 (90%)	93 (95%)	94 (96%)	94 (96%)	94 (96%)
Number (%) of Cities with 4th highest >70 (any year; 1987-2000)	0 (0%)	3 (3%)	31 (32%)	77 (79%)	86 (88%)	93 (95%)	94 (96%)	94 (96%)	95 (97%)	95 (97%)
Number (%) of Cities with 4th highest >65 (any year; 1987-2000)	0 (0%)	10 (10%)	58 (59%)	84 (86%)	93 (95%)	94 (96%)	94 (96%)	94 (96%)	94 (96%)	94 (96%)
Number (%) of Cities with 4th highest >60 (any year; 1987-2000)	1 (1%)	36 (37%)	74 (76%)	93 (95%)	96 (8%)	97 (99%)	97 (99%)	97 (99%)	97 (99%)	97 (99%)

¹⁰ Study authors included 98 cities in their analyses, air quality data only available for 95

Relationship between average and highest 8-hour daily maximum O₃ concentrations for New York City, as analyzed by Silverman and Ito (2010)

EPA staff retrieved daily maximum 8-hour O₃ concentrations for the 13 monitors in the New York City area used in the Silverman and Ito (2010) study for April-August of 1999-2006 from the AQS database. Next, EPA staff spatially averaged these concentrations across monitors for each day during this period, and then paired them with the highest 8-hour daily maximum value reported across the 13 monitors on each day.

Next, the range of observed average daily maximum 8-hour concentrations was broken into 5 ppb increments. The number of days where the area-wide average daily maximum 8-hour concentration fell within the increment and the number of days where one or more monitored 8-hour daily maximum values were greater than 75, 70, 65 and 60 ppb were recorded for each 5 ppb increment. These numbers are summarized in Table 3D-4.

Table 3B-6 Summary statistics for Observed O₃ Concentrations in the New York City Area, April – August 1999 – 2006

	2-day moving average across monitors (ppb)								
	11 to 20 (62 days)	21 to 25 (92 days)	26 to 30 (178 days)	31 to 35 (206 days)	36 to 40 (236 days)	41 to 45 (196 days)	46 to 50 (153 days)	51 to 55 (111 days)	56 to 60 (71 days)
Days > 75 ppb	0	0	1	0	1	2	9	15	20
Days > 70 ppb	0	0	1	4	1	12	17	23	30
Days > 65 ppb	0	0	1	6	5	18	37	42	45
Days > 60 ppb	0	0	2	7	12	39	67	61	53

Relationship between average and highest 8-hour daily maximum O₃ concentrations for Atlanta, as analyzed by Strickland et al. (2010)

For our assessment of the Strickland et al. (2010) study, based in the Atlanta metropolitan area, we retrieved 8-hour daily maximum concentration data for 4 of the 5 monitors used in the study during the study period (May-October, 1993-2004) from the AQS database. The 5th monitor was a part of the Southeastern Aerosol Research and Characterization (SEARCH) network, which does not report data to EPA. EPA staff calculated the area-wide average of the 8-hour daily maximum concentrations for each day, and compared to population-weighted average concentrations obtained from the author. The correlation between the arithmetic average values and the population-weighted average values was very high (R = 0.985), thus EPA staff deemed the arithmetic average to be a suitable surrogate for the population-weighted average used in the study. Finally, 3-day moving averages were calculated from the daily area-wide average values (matching the air quality metric used in the study), and paired with the highest monitored 8-hour daily maximum value occurring during each 3-day period.

Next, the range of observed average daily maximum 8-hour concentrations was broken into 5 ppb increments. The number of days where the area-wide average daily maximum 8-hour concentration fell within the increment and the number of days where one or more monitored 8-hour daily maximum values were greater than 75, 70, 65 and 60 ppb were recorded for each 5 ppb increment. These numbers are summarized in Table 3D-5.

Table 3B-7 Summary statistics for Observed O₃ Concentrations in the Atlanta Area, April – August 1999 – 2006

	3-day moving average across monitors (ppb)										
	26-30 (75 days)	31-35 (144 days)	36-40 (165 days)	41-45 (210 days)	46-50 (235 days)	51-55 (244 days)	56-60 (272 days)	61-65 (234 days)	66-70 (169 days)	71 to 75 (124 days)	76 to 80 (106 days)
Days > 75	0	0	2	2	10	24	53	80	89	87	87
Days > 70	0	0	6	6	20	49	81	111	107	96	95
Days > 65	1	0	8	19	38	75	118	147	133	106	100
Days > 60	1	2	15	33	68	115	152	173	147	116	102

Relationship between annual and highest 1-hour daily maximum O₃ concentrations for 12 study areas, as analyzed by Jerrett et al. (2009)

The Jerrett et al. (2009) study used a long-term metric based on seasonal averages of 1-hour daily maximum O₃ concentrations to evaluate associations between respiratory mortality and long-term or repeated exposures to O₃. Authors divided study cities into quartiles based on these seasonal averages of 1-hour daily O₃ concentrations. Using AQS, we identified the 3-year averages of annual 4th highest daily maximum 8-hour O₃ concentrations in study cities during the study period. Table 3D-6 presents the means and maximums of these concentrations over the study period.

In addition, for the 12 urban case study areas included in the epidemiology-based risk assessment of the 2nd draft of the Health REA we identified the seasonal averages of 1-hour daily maximum concentrations (i.e., the O₃ metric evaluated by Jerrett et al., 2009) for air quality adjusted to the current and alternative standards. Specifically, for adjusted air quality “quarterly” averages of 1-hour concentrations for April-June and July-August were calculated for each area and year. The quarterly values were considered to be valid if valid daily maximum 1-hour values were available for at least 75% of the days in the quarter. The two quarterly values were then averaged, as was done by Jerrett et al. (2009) to generate the long-term metric used in the study. This process was repeated for the various model-based adjustment scenarios in each of the 12 study areas. Summary statistics based on this seasonal average of daily O₃ concentrations are presented in Table 3D-7 for recent air quality and for air quality adjusted to just meet the current and alternative standards.

Table 3B-8 Three-Year Averages of Annual 4th Highest Daily Maximum 8-hour O₃ Concentrations in 94¹¹ Study Areas Examined in Jerrett et al. (2009)

	City	Mean over study period	Max over study period
Cities in the lowest quartile of average exposure ¹²	Charleston, WV	81	99
	Chicago, IL	103	114
	Colorado Springs, CO	62	66
	Corpus Christi, TX	82	89
	Detroit, MI	95	103
	Flint, MI	83	91
	Ft. Lauderdale, FL	74	79
	Kansas City, MO	87	97
	Lansing, MI	81	90
	Madison, WI	82	102
	Minneapolis, MN	74	80
	New Orleans, LA	86	99
	Orlando, FL	79	82
	Portland, OR	81	91
	Providence, RI	110	124
	Salinas, CA	68	74
	San Antonio, TX	85	92
	San Francisco, CA	88	96
	San Jose, CA	91	103
	Seattle, WA	78	88
Tacoma, WA	78	88	
Vallejo, CA	74	82	
Wichita, KS	75	81	
Cities in the highest three quartiles of average exposure ¹³	Charleston, SC	79	90
	Charlotte, NC	97	112
	Chattanooga, TN	90	97
	Cincinnati, OH	101	119
	Cleveland, OH	98	108
	Columbia, SC	85	109
	Columbus, OH	93	103
	Dallas/Ft Worth, TX	106	118
	Dayton, OH	95	122
	Denver, CO	83	91

¹¹ Jerrett et al. (2009) examined 96 MSAs; this analysis included the 94 cities that met data completeness criteria described above, after linking monitors to MSAs (see lines 10-28, above).

¹² Based on visual inspection of Figure 1 in Jerrett et al. (2009)

¹³ Based on visual inspection of Figure 1 in Jerrett et al. (2009)

El Paso, TX	85	96
Evansville, IN	93	100
Fresno, CA	112	123
Gary, IN	91	105
Greely, CO	69	75
Greensboro, NC	89	100
Greenville, SC	86	94
Harrisburg, PA	94	103
Houston, TX	121	140
Huntington, WV	94	103
Indianapolis, IN	93	103
Jackson, MS	79	98
Jacksonville, FL	81	87
Jersey City, NJ	106	118
Johnstown, PA	90	107
Kenosha, WI	101	114
Knoxville, TN	91	97
Lancaster, PA	94	101
Las Vegas, NV	80	85
Lexington, KY	88	99
Little Rock, AR	86	107
Los Angeles, CA	193	248
Memphis, TN	94	103
Milwaukee, WI	103	117
Nashville, TN	94	106
Nassau, NY	NA ¹⁴	NA
New Haven, CT	116	136
New York City, NY	118	129
Newark, NJ	90	105
Norfolk, VA	91	101
Oklahoma City, OK	86	93
Philadelphia, PA	117	136
Phoenix, AZ	86	96
Pittsburgh, PA	101	123
Portland, ME	106	117
Portsmouth, NH	92	104
Racine, WI	102	124
Raleigh, NC	90	104
Reading, PA	99	114
Richmond, VA	94	104
Riverside, CA	196	245
Roanoke, VA	83	95

¹⁴ Air quality data did not meet completeness criteria described above

Rochester, NY	89	99
Sacramento, CA	110	118
San Diego, CA	121	141
Shreveport, LA	83	88
South Bend, IN	90	102
Springfield, MA	102	115
St Louis, MO	105	122
Steubenville, OH	82	99
Syracuse, NY	85	96
Tampa, FL	85	91
Toledo, OH	93	108
Trenton, NJ	112	124
Tucson, AZ	76	82
Ventura, CA	118	132
Washington, DC	105	116
Wilmington, DE	103	116
Worcester, MA	92	102
York, PA	95	107
Youngstown, OH	93	103

Table 3B-9 Long-Term O₃ Concentrations in 12 Urban Case Study Areas (Using the O₃ Metric Evaluated by Jerrett et al., 2009) for Recent Air Quality and Air Quality Adjusted to Meet Standard Levels of 75, 70, 65, and 60 ppb

	Air Quality Adjusted to:	2006 (Adj Yrs 2006-2008)	2007 (Adj Yrs 2006-2008)	2008 (Adj Yrs 2008-2010)	2009 (Adj Yrs 2008-2010)	2010 (Adj Yrs 2008-2010)
Atlanta	Recent	65	63	57	50	56
	75	53	52	53	47	52
	70	50	49	49	44	49
	65	47	46	46	42	46
	60	45	44	44	40	44
Baltimore	Recent	60	59	57	52	60
	75	54	54	53	49	55
	70	52	51	51	48	53
	65	49	49	48	46	50
	60	46	46	46	44	48
Boston	Recent	49	50	46	45	49
	75	48	49	49	45	48
	70	46	47	48	44	48
	65	44	45	46	43	46
	60	43	43	44	41	44
Cleveland	Recent	51	52	53	49	54
	75	49	50	51	47	51
	70	47	48	48	45	48
	65	45	45	45	43	45
	60	41	41	41	40	42
Denver	Recent	63	63	63	58	60
	75	62	61	63	58	60
	70	60	59	62	58	58
	65	58	58	59	56	55
	60	53	53	53	51	50
Detroit	Recent	50	54	51	48	52
	75	50	52	N/A	N/A	N/A
	70	48	50	51	49	52
	65	47	49	49	47	50
	60	45	46	46	45	47
Houston	Recent	53	48	47	47	46
	75	48	46	47	48	46
	70	47	45	46	47	46
	65	46	44	45	46	45
	60	45	43	43	44	44
Los Angeles	Recent	65	61	64	62	57
	75	58	59	60	60	58
	70	55	56	57	58	56
	65	52	53	54	54	53
	60	N/A	N/A	N/A	N/A	N/A
New York City	Recent	53	54	55	48	55
	75	47	47	51	47	51
	70	N/A	N/A	N/A	N/A	N/A
	65	N/A	N/A	N/A	N/A	N/A
	60	N/A	N/A	N/A	N/A	N/A
Philadelphia	Recent	56	59	57	51	58
	75	51	52	54	49	54
	70	49	50	51	47	52
	65	47	48	49	45	49
	60	45	46	47	43	47
Sacramento	Recent	66	59	65	61	55
	75	55	50	54	51	48
	70	52	48	51	49	46
	65	50	46	49	47	44
	60	47	44	46	44	42
Saint Louis	Recent	58	58	52	51	55
	75	53	53	51	50	54
	70	50	51	50	48	52
	65	47	48	48	46	49
	60	44	45	45	43	46

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Appendix 5A

Ozone-Sensitive Plant Species^A Used by Some Tribes*		
<small>*(Based on Feedback from 3 Tribes)</small>		
Common Name (other common names)	Scientific Name	Confirmed bioindicator species
Red alder (Oregon alder, Western alder)	<i>Alnus rubra</i>	Y
Speckled alder (Tag alder, Gray alder, Hoary alder)	<i>Alnus rugosa (Alnus incana)</i>	Y
Groundnut (Wild bean, American potato bean)	<i>Apios americana</i>	Y
Spreading Dogbane (Common dogbane)	<i>Apocynum androsamifolium</i>	Y
Common milkweed	<i>Asclepias syriaca</i>	Y
New England Aster	<i>Aster novae-angliae</i> <i>Symphotrichum novae-angliae</i>	
Green ash	<i>Fraxinus pennsylvanica</i>	
Twinberry	<i>Lonicera involucrate</i>	Y
Bee-balm	<i>Monarda didyma</i>	
Virginia creeper	<i>Parthenocissus quinquefolia</i>	Y
Jack pine	<i>Pinus banksiana</i>	Y
Lodgepole pine	<i>Pinus contorta</i>	
White pine	<i>Pinus strobus</i>	
Black poplar (Balsam poplar)	<i>Populus balsamifera trichocarpa</i>	
Quaking aspen (Trembling aspen)	<i>Populus tremuloides</i>	Y
Black cherry	<i>Prunus serotina</i>	Y
Choke cherry	<i>Prunus virginiana</i>	
Douglas fir	<i>Pseudotsuga menziesii</i>	
Allegheny blackberry (Common blackberry)	<i>Rubus allegheniensis</i>	Y
Thimbleberry	<i>Rubus parviflorus</i>	Y
Cutleaf coneflower (Coneflower, Golden glow)	<i>Rudbeckia laciniata</i>	Y
Pussy willow	<i>Salix discolor</i>	
Shinning willow	<i>Salix lucida</i>	
American elder (White elder)	<i>Sambucus canadensis</i>	Y
Red elderberry	<i>Sambucus racemosa</i>	Y
Sassafras	<i>Sassafras albidum</i>	
Goldenrod	<i>Solidago altissima</i>	
Huckleberry	<i>Vaccinium membranaceum</i>	Y
Wild grape	<i>Vitis spp.</i>	
European wine grape	<i>Vitis vinifera</i>	Y

^ASpecies included in this list are identified in one or more of the following sources:
 1) SP 2007 (www.2.nature.nps.gov/air/Pubs/pdf/flag/NPSozonesensppFLAG06.pdf)
 2) NPS O₃ Bioindicators 2006 (www.nature.nps.gov/air/Pubs/bioindicators/index.cfm)
 3) Kline et al., 2008; 4) Davis, 2007/ 2009; 5) Flagler, et al., eds., 1998
 6) USDA FS FHM/FIA: Ozone Bioindicator Sampling and Estimation (www.nrs.fs.fed.us/fia/topics/ozone/pubs/pdfs/ozone%20estimation%20document.pdf) and Ozone Injury in West Coast Forests: 6 Years of Monitoring (2007).

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APPENDIX 5B: CLASS I AREAS BELOW CURRENT STANDARD AND ABOVE 15 PPM-HRS

This appendix identifies Class I areas that might have W126 index values above 15 ppm-hrs allowed by the current standard based on an analysis of recent O₃ monitoring data. Table 5B-1 provides all monitoring sites from 1998-2002 that were at or below 75 ppb (3-year average of 4th highest maximum 8-hour average), at or above 15 ppm-hrs (3-year average), and located in counties with Class I areas. For each year that met these 3-year requirements, we also provide the maximum annual 8-hour O₃ concentration (in ppb) and W126 index value (in ppm-hrs).

Table 5B-1 Examples of Counties Containing Class I Areas where Recent 3-Year O₃ Concentrations were Below 75 ppb and 3-Year Average W126 Index Values were Above 15 ppm-hrs

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
400380011	1998-2000	1998	70	67	15.88	14.71	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	1998-2000	1999	70	72	15.88	16.57	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	1998-2000	2000	70	71	15.88	16.36	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2002-2004	2002	71	74	15.70	14.45	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2002-2004	2003	71	71	15.70	18.07	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2002-2004	2004	71	70	15.70	14.57	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2003-2005	2003	71	71	16.64	18.07	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2003-2005	2004	71	70	16.64	14.57	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2003-2005	2005	71	72	16.64	17.29	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2004-2006	2004	72	70	16.56	14.57	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2004-2006	2005	72	72	16.56	17.29	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2004-2006	2006	72	74	16.56	17.81	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2005-2007	2005	71	72	16.36	17.29	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2005-2007	2006	71	74	16.36	17.81	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2005-2007	2007	71	67	16.36	13.98	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2006-2008	2006	69	74	16.37	17.81	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2006-2008	2007	69	67	16.37	13.98	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2006-2008	2008	69	68	16.37	17.32	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2010-2012	2010	73	71	18.06	13.21	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2010-2012	2011	73	75	18.06	19.33	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400380011	2010-2012	2012	73	74	18.06	21.65	Chiricahua National Monument	AZ	Cochise	Chiricahua National Monument
400510081	2008-2010	2008	69	74	15.61	22.20	Flagstaff Middle School	AZ	Coconino	Grand Canyon National Park
400510081	2008-2010	2009	69	66	15.61	11.38	Flagstaff Middle School	AZ	Coconino	Grand Canyon National Park
400580011	2008-2010	2010	69	69	15.61	14.89	Flagstaff Middle School	AZ	Coconino	Grand Canyon National Park
400510081	2006-2008	2008	70	74	19.29	22.20	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400510081	2007-2009	2008	68	74	15.38	22.20	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400510081	2007-2009	2009	68	66	15.38	11.38	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	1998-2000	1998	73	72	18.74	18.23	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	1998-2000	1999	73	76	18.74	21.27	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	1998-2000	2000	73	71	18.74	16.74	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	1999-2001	1999	72	76	17.64	21.27	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	1999-2001	2000	72	71	17.64	16.74	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
400580011	1999-2001	2001	72	70	17.64	14.91	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2000-2002	2000	73	71	19.47	16.74	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2000-2002	2001	73	70	19.47	14.91	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2000-2002	2002	73	79	19.47	26.78	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2001-2003	2001	74	70	21.79	14.91	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2001-2003	2002	74	79	21.79	26.78	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2001-2003	2003	74	73	21.79	23.70	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2002-2004	2002	74	79	22.29	26.78	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2002-2004	2003	74	73	22.29	23.70	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2002-2004	2004	74	72	22.29	16.41	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2003-2005	2003	74	73	19.98	23.70	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2003-2005	2004	74	72	19.98	16.41	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2003-2005	2005	74	79	19.98	19.84	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2004-2006	2004	73	72	19.39	16.41	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2004-2006	2005	73	79	19.39	19.84	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2004-2006	2006	73	70	19.39	21.92	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2005-2007	2005	72	79	20.24	19.84	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2005-2007	2006	72	70	20.24	21.92	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2005-2007	2007	72	69	20.24	18.95	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2006-2008	2006	70	70	19.29	21.92	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2006-2008	2007	70	69	19.29	18.95	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2007-2009	2007	68	69	15.38	18.95	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2010-2012	2010	72	69	17.90	14.89	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2010-2012	2011	72	74	17.90	18.45	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400580011	2010-2012	2012	72	73	17.90	20.34	Grand Canyon National Park, The Abyss	AZ	Coconino	Grand Canyon National Park
400700101	2007-2009	2007	75	76	22.45	24.95	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2007-2009	2008	75	78	22.45	27.52	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2007-2009	2009	75	72	22.45	14.89	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2008-2010	2008	73	78	20.29	27.52	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2008-2010	2009	73	72	20.29	14.89	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2008-2010	2010	73	70	20.29	18.45	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2009-2011	2009	72	72	17.90	14.89	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2009-2011	2010	72	70	17.90	18.45	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2009-2011	2011	72	76	17.90	20.35	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2010-2012	2010	74	70	21.41	18.45	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
400700101	2010-2012	2011	74	76	21.41	20.35	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
400700101	2010-2012	2012	74	78	21.41	25.44	Tonto NM	AZ	Gila	Sierra Ancha Wilderness Area
401320051	2007-2009	2009	75	70	22.48	14.51	Rio Verde	AZ	Maricopa	Superstition Wilderness Area
401340081	2007-2009	2008	75	78	22.48	27.49	Rio Verde	AZ	Maricopa	Superstition Wilderness Area
401397061	2007-2009	2007	75	79	22.48	28.65	Rio Verde	AZ	Maricopa	Superstition Wilderness Area
401701191	2010-2012	2010	70	68	15.79	12.96	Petrified Forest National Park, South Entrance	AZ	Navajo	Petrified Forest National Park
401701191	2010-2012	2011	70	69	15.79	15.16	Petrified Forest National Park, South Entrance	AZ	Navajo	Petrified Forest National Park
401701191	2010-2012	2012	70	73	15.79	19.26	Petrified Forest National Park, South Entrance	AZ	Navajo	Petrified Forest National Park
401900211	1998-2000	1998	73	77	15.55	18.60	22nd & Craycroft	AZ	Pima	Saguaro National Park
401910181	1998-2000	1999	73	73	15.55	16.53	22nd & Craycroft	AZ	Pima	Saguaro National Park
401910281	1998-2000	2000	73	77	15.55	15.52	22nd & Craycroft	AZ	Pima	Saguaro National Park
401900211	2001-2003	2002	73	77	15.53	16.01	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2001-2003	2003	73	78	15.53	23.14	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2006-2008	2007	74	73	18.98	17.24	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2006-2008	2008	74	74	18.98	20.01	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2007-2009	2007	71	73	16.10	17.24	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2007-2009	2008	71	74	16.10	20.01	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2007-2009	2009	71	67	16.10	11.04	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2008-2010	2008	69	74	15.47	20.01	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2008-2010	2009	69	67	15.47	11.04	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2008-2010	2010	69	68	15.47	15.36	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2010-2012	2010	71	68	16.84	15.36	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2010-2012	2011	71	75	16.84	17.36	Saguaro Park	AZ	Pima	Saguaro National Park
401900211	2010-2012	2012	71	71	16.84	17.79	Saguaro Park	AZ	Pima	Saguaro National Park
401910111	2001-2003	2001	73	69	15.53	12.73	Saguaro Park	AZ	Pima	Saguaro National Park
401910181	2006-2008	2006	74	76	18.98	21.54	Saguaro Park	AZ	Pima	Saguaro National Park
402130011	2007-2009	2007	75	77	22.52	24.59	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2007-2009	2008	75	80	22.52	29.02	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2007-2009	2009	75	70	22.52	14.81	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2008-2010	2008	74	80	20.87	29.02	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2008-2010	2009	74	70	20.87	14.81	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2008-2010	2010	74	72	20.87	18.79	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2009-2011	2009	73	70	18.75	14.81	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2009-2011	2010	73	72	18.75	18.79	Queen Valley	AZ	Pinal	Superstition Wilderness Area
402180011	2009-2011	2011	73	78	18.75	22.66	Queen Valley	AZ	Pinal	Superstition Wilderness Area

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
600500021	2010-2012	2010	74	75	17.68	15.56	201 Clinton Road, Jackson	CA	Amador	Mokelumne Wilderness Area
600500021	2010-2012	2011	74	70	17.68	14.87	201 Clinton Road, Jackson	CA	Amador	Mokelumne Wilderness Area
600500021	2010-2012	2012	74	78	17.68	22.61	201 Clinton Road, Jackson	CA	Amador	Mokelumne Wilderness Area
602701011	2008-2010	2008	72	77	17.19	25.85	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2008-2010	2009	72	70	17.19	15.55	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2008-2010	2010	72	69	17.19	10.16	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2009-2011	2009	71	70	16.54	15.55	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2009-2011	2010	71	69	16.54	10.16	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2009-2011	2011	71	75	16.54	23.92	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2010-2012	2010	72	69	18.69	10.16	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2010-2012	2011	72	75	18.69	23.92	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
602701011	2010-2012	2012	72	73	18.69	22.00	Death Valley National Monument Near Nevares Springs Access	CA	Inyo	John Muir Wilderness Area
606900031	2005-2007	2005	74	71	15.18	13.11	Pinnacles National Monument, SW of East Entrance Station	CA	San Benito	Pinnacles National Monument
606900031	2005-2007	2006	74	78	15.18	17.44	Pinnacles National Monument, SW of East Entrance Station	CA	San Benito	Pinnacles National Monument
606900031	2005-2007	2007	74	75	15.18	14.99	Pinnacles National Monument, SW of East Entrance Station	CA	San Benito	Pinnacles National Monument
608900071	2008-2010	2009	75	74	15.31	13.66	Anderson - North Street	CA	Shasta	Lassen Volcanic National Park
608900091	2008-2010	2010	75	74	15.31	15.33	Anderson - North Street	CA	Shasta	Lassen Volcanic National Park
608930031	2008-2010	2008	75	83	15.31	18.72	Anderson - North Street	CA	Shasta	Lassen Volcanic National Park
610900051	2009-2011	2009	74	77	20.72	21.80	251 S Barretta, Sonora, CA 95370	CA	Tuolumne	Yosemite National Park
610900051	2009-2011	2010	74	72	20.72	20.58	251 S Barretta, Sonora, CA 95370	CA	Tuolumne	Yosemite National Park
610900051	2009-2011	2011	74	74	20.72	19.78	251 S Barretta, Sonora, CA 95370	CA	Tuolumne	Yosemite National Park
610900051	2010-2012	2010	73	72	20.84	20.58	251 S Barretta, Sonora, CA 95370	CA	Tuolumne	Yosemite National Park
610900051	2010-2012	2011	73	74	20.84	19.78	251 S Barretta, Sonora, CA 95370	CA	Tuolumne	Yosemite National Park
610900051	2010-2012	2012	73	75	20.84	22.14	251 S Barretta, Sonora, CA 95370	CA	Tuolumne	Yosemite National Park
801300111	2000-2002	2000	73	72	15.11	14.06	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2000-2002	2001	73	71	15.11	13.18	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2000-2002	2002	73	78	15.11	18.09	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2003-2005	2003	75	82	16.61	23.91	South Boulder Creek	CO	Boulder	Rocky Mountain National Park

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801300111	2003-2005	2004	75	68	16.61	9.57	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2003-2005	2005	75	76	16.61	16.35	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2004-2006	2004	75	68	17.01	9.57	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2004-2006	2005	75	76	17.01	16.35	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2004-2006	2006	75	82	17.01	25.11	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2008-2010	2008	73	76	16.11	20.77	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2008-2010	2009	73	73	16.11	12.57	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2008-2010	2010	73	72	16.11	14.98	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2009-2011	2009	73	73	16.13	12.57	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2009-2011	2010	73	72	16.13	14.98	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2009-2011	2011	73	76	16.13	20.82	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2010-2012	2010	74	72	19.34	14.98	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2010-2012	2011	74	76	19.34	20.82	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
801300111	2010-2012	2012	74	76	19.34	22.20	South Boulder Creek	CO	Boulder	Rocky Mountain National Park
805199911	1998-2000	1998	73	71	20.18	21.13	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	1998-2000	1999	73	77	20.18	23.98	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	1998-2000	2000	73	73	20.18	15.43	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	1999-2001	1999	73	77	18.40	23.98	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	1999-2001	2000	73	73	18.40	15.43	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	1999-2001	2001	73	70	18.40	15.80	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2000-2002	2000	71	73	18.01	15.43	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2000-2002	2001	71	70	18.01	15.80	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2000-2002	2002	71	71	18.01	22.82	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2001-2003	2001	71	70	18.90	15.80	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2001-2003	2002	71	71	18.90	22.82	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2001-2003	2003	71	73	18.90	18.07	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2002-2004	2002	70	71	17.95	22.82	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2002-2004	2003	70	73	17.95	18.07	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2002-2004	2004	70	67	17.95	12.96	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2003-2005	2003	69	73	15.82	18.07	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2003-2005	2004	69	67	15.82	12.96	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2003-2005	2005	69	69	15.82	16.42	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2004-2006	2004	68	67	15.60	12.96	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2004-2006	2005	68	69	15.60	16.42	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2004-2006	2006	68	70	15.60	17.40	Gothic	CO	Gunnison	West Elk Wilderness Area

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805199911	2005-2007	2005	68	69	16.38	16.42	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2005-2007	2006	68	70	16.38	17.40	Gothic	CO	Gunnison	West Elk Wilderness Area
805199911	2005-2007	2007	68	65	16.38	15.31	Gothic	CO	Gunnison	West Elk Wilderness Area
806710041	2005-2007	2005	72	75	18.78	17.93		CO	La Plata	Weminuche Wilderness Area
806710041	2005-2007	2006	72	74	18.78	20.82		CO	La Plata	Weminuche Wilderness Area
806710041	2005-2007	2007	72	69	18.78	17.58		CO	La Plata	Weminuche Wilderness Area
806710041	2006-2008	2006	70	74	18.10	20.82		CO	La Plata	Weminuche Wilderness Area
806710041	2006-2008	2007	70	69	18.10	17.58		CO	La Plata	Weminuche Wilderness Area
806710041	2006-2008	2008	70	69	18.10	15.91		CO	La Plata	Weminuche Wilderness Area
806710041	2008-2010	2008	71	69	15.07	15.91		CO	La Plata	Weminuche Wilderness Area
806710041	2008-2010	2009	71	71	15.07	10.94		CO	La Plata	Weminuche Wilderness Area
806710041	2008-2010	2010	71	74	15.07	18.36		CO	La Plata	Weminuche Wilderness Area
806710041	2009-2011	2009	74	71	16.80	10.94		CO	La Plata	Weminuche Wilderness Area
806710041	2009-2011	2010	74	74	16.80	18.36		CO	La Plata	Weminuche Wilderness Area
806710041	2009-2011	2011	74	77	16.80	21.09		CO	La Plata	Weminuche Wilderness Area
806710041	2010-2012	2010	73	74	19.16	18.36		CO	La Plata	Weminuche Wilderness Area
806710041	2010-2012	2011	73	77	19.16	21.09		CO	La Plata	Weminuche Wilderness Area
806710041	2010-2012	2012	73	69	19.16	18.02		CO	La Plata	Weminuche Wilderness Area
806900071	2008-2010	2010	74	77	18.31	19.12	Fort Collins - West	CO	Larimer	Rocky Mountain National Park
806900111	2008-2010	2008	74	76	18.31	21.63	Fort Collins - West	CO	Larimer	Rocky Mountain National Park
806900111	2008-2010	2009	74	73	18.31	14.17	Fort Collins - West	CO	Larimer	Rocky Mountain National Park
806900071	1999-2001	1999	74	74	15.05	11.16	Rocky Mountain National Park, Long's Peak	CO	Larimer	Rocky Mountain National Park
806900071	1999-2001	2000	74	78	15.05	25.82	Rocky Mountain National Park, Long's Peak	CO	Larimer	Rocky Mountain National Park
806900071	1999-2001	2001	74	70	15.05	8.16	Rocky Mountain National Park, Long's Peak	CO	Larimer	Rocky Mountain National Park
806900071	2004-2006	2004	74	73	15.57	16.23	Rocky Mountain National Park, Long's Peak	CO	Larimer	Rocky Mountain National Park
806900071	2004-2006	2006	74	76	15.57	18.53	Rocky Mountain National Park, Long's Peak	CO	Larimer	Rocky Mountain National Park
806999911	2004-2006	2005	74	78	15.57	16.20	Rocky Mountain National Park, Long's Peak	CO	Larimer	Rocky Mountain National Park
808301011	1998-2000	1998	70	68	16.37	12.90	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	1998-2000	1999	70	69	16.37	14.17	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	1998-2000	2000	70	73	16.37	22.04	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	1999-2001	1999	69	69	15.66	14.17	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	1999-2001	2000	69	73	15.66	22.04	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park

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808301011	1999-2001	2001	69	65	15.66	10.77	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2000-2002	2000	69	73	17.51	22.04	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2000-2002	2001	69	65	17.51	10.77	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2000-2002	2002	69	70	17.51	19.72	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2001-2003	2001	67	65	16.00	10.77	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2001-2003	2002	67	70	16.00	19.72	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2001-2003	2003	67	67	16.00	17.50	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2002-2004	2002	68	70	16.34	19.72	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2002-2004	2003	68	67	16.34	17.50	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2002-2004	2004	68	69	16.34	11.79	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2003-2005	2003	70	67	16.96	17.50	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2003-2005	2004	70	69	16.96	11.79	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2003-2005	2005	70	76	16.96	21.59	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2004-2006	2004	73	69	19.02	11.79	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2004-2006	2005	73	76	19.02	21.59	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2004-2006	2006	73	74	19.02	23.68	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2005-2007	2005	73	76	21.00	21.59	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2005-2007	2006	73	74	21.00	23.68	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2005-2007	2007	73	70	21.00	17.73	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2006-2008	2006	71	74	18.36	23.68	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2006-2008	2007	71	70	18.36	17.73	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2006-2008	2008	71	69	18.36	13.67	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
808301011	2007-2009	2007	69	70	15.58	17.73	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2007-2009	2008	69	69	15.58	13.67	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
808301011	2007-2009	2009	69	69	15.58	15.35	Mesa Verde National Park, Resource Management Area	CO	Montezuma	Mesa Verde National Park
2106105011	2006-2008	2006	74	71	15.99	12.55	Mammoth Cave National Park, Houchin Meadow	KY	Edmonson	Mammoth Cave National Park
2106105011	2006-2008	2007	74	82	15.99	22.58	Mammoth Cave National Park, Houchin Meadow	KY	Edmonson	Mammoth Cave National Park
2106105011	2006-2008	2008	74	70	15.99	12.83	Mammoth Cave National Park, Houchin Meadow	KY	Edmonson	Mammoth Cave National Park
3501510051	2004-2006	2004	69	65	15.30	8.65	5ZR on BLM Land bordering residential area outside Carlsbad	NM	Eddy	Carlsbad Caverns National Park
3501510051	2004-2006	2005	69	67	15.30	10.55	5ZR on BLM Land bordering residential area outside Carlsbad	NM	Eddy	Carlsbad Caverns National Park
3501510051	2004-2006	2006	69	76	15.30	26.71	5ZR on BLM Land bordering residential area outside Carlsbad	NM	Eddy	Carlsbad Caverns National Park
3501510051	2005-2007	2005	69	67	15.33	10.55	5ZR on BLM Land bordering residential area outside Carlsba	NM	Eddy	Carlsbad Caverns National Park
3501510051	2005-2007	2006	69	76	15.33	26.71	5ZR on BLM Land bordering residential area outside Carlsbad	NM	Eddy	Carlsbad Caverns National Park
3501510051	2005-2007	2007	69	66	15.33	8.72	5ZR on BLM Land bordering residential area outside Carlsbad	NM	Eddy	Carlsbad Caverns National Park
3501510051	2006-2008	2006	69	76	15.07	26.71	5ZR on BLM Land bordering residential area outside Carlsbad	NM	Eddy	Carlsbad Caverns National Park
3501510051	2006-2008	2007	69	66	15.07	8.72	5ZR on BLM Land bordering residential area outside Carlsba	NM	Eddy	Carlsbad Caverns National Park
3501510051	2006-2008	2008	69	67	15.07	9.78	5ZR on BLM Land bordering residential area outside Carlsbad	NM	Eddy	Carlsbad Caverns National Park
3504310011	2000-2002	2001	72	69	17.19	12.17		NM	Sandoval	Bandelier Wilderness Area
3504310011	2000-2002	2002	72	74	17.19	19.62		NM	Sandoval	Bandelier Wilderness Area
3504310011	2001-2003	2001	71	69	17.37	12.17		NM	Sandoval	Bandelier Wilderness Area
3504310011	2001-2003	2002	71	74	17.37	19.62		NM	Sandoval	Bandelier Wilderness Area
3504310031	2000-2002	2000	72	75	17.19	23.51		NM	Sandoval	Bandelier Wilderness Area
3504310031	2001-2003	2003	71	76	17.37	25.38		NM	Sandoval	Bandelier Wilderness Area
3504310011	1999-2001	2001	72	69	17.87	12.17	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310011	2002-2004	2002	74	74	20.86	19.62	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310011	2002-2004	2004	74	71	20.86	17.73	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
3504310011	2003-2005	2004	74	71	20.08	17.73	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310011	2004-2006	2004	73	71	17.75	17.73	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	1999-2001	1999	72	76	17.87	18.14	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	1999-2001	2000	72	75	17.87	23.51	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2002-2004	2003	74	76	20.86	25.38	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2003-2005	2003	74	76	20.08	25.38	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2003-2005	2005	74	75	20.08	17.03	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2004-2006	2005	73	75	17.75	17.03	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2005-2007	2005	73	75	17.50	17.03	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2005-2007	2007	73	71	17.50	17.05	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2006-2008	2007	70	71	15.87	17.05	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504310031	2006-2008	2008	70	65	15.87	12.15	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504390041	2004-2006	2006	73	72	17.75	19.26	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504390041	2005-2007	2006	73	72	17.50	19.26	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
3504390041	2006-2008	2006	70	72	15.87	19.26	2ZR Site moved from Rio Rancho City Hall to senior center	NM	Sandoval	Bandelier Wilderness Area
4603301323	2005-2007	2005	70	70	15.49	13.56	Wind Cave National Park, Visitor Center	SD	Custer	Wind Cave National Park
4603301323	2005-2007	2006	70	73	15.49	20.63	Wind Cave National Park, Visitor Center	SD	Custer	Wind Cave National Park
4603301323	2005-2007	2007	70	69	15.49	12.29	Wind Cave National Park, Visitor Center	SD	Custer	Wind Cave National Park
4903701011	1998-2000	1998	73	71	19.80	19.78	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	1998-2000	1999	73	73	19.80	20.25	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	1998-2000	2000	73	76	19.80	19.36	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2001-2003	2001	70	66	18.94	9.91	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2001-2003	2002	70	72	18.94	22.12	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2001-2003	2003	70	74	18.94	24.80	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2002-2004	2002	72	72	20.50	22.12	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2002-2004	2003	72	74	20.50	24.80	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
4903701011	2002-2004	2004	72	72	20.50	14.57	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2003-2005	2003	71	74	18.59	24.80	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2003-2005	2004	71	72	18.59	14.57	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2003-2005	2005	71	69	18.59	16.40	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2004-2006	2004	70	72	16.59	14.57	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2004-2006	2005	70	69	16.59	16.40	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2004-2006	2006	70	70	16.59	18.80	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2005-2007	2005	70	69	17.66	16.40	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2005-2007	2006	70	70	17.66	18.80	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2005-2007	2007	70	72	17.66	17.78	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2006-2008	2006	71	70	18.10	18.80	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2006-2008	2007	71	72	18.10	17.78	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2006-2008	2008	71	71	18.10	17.71	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2007-2009	2007	70	72	16.07	17.78	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2007-2009	2008	70	71	16.07	17.71	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2007-2009	2009	70	68	16.07	12.72	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2010-2012	2010	69	68	15.01	13.87	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2010-2012	2011	69	69	15.01	14.23	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4903701011	2010-2012	2012	69	72	15.01	16.93	Canyonlands National Park, Island in the Sky	UT	San Juan	Canyonlands National Park
4905301301	2006-2008	2006	71	72	21.12	24.29	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2006-2008	2007	71	71	21.12	19.37	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2006-2008	2008	71	72	21.12	19.69	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2007-2009	2007	70	71	18.01	19.37	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2007-2009	2008	70	72	18.01	19.69	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2007-2009	2009	70	68	18.01	14.96	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2008-2010	2008	70	72	18.46	19.69	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2008-2010	2009	70	68	18.46	14.96	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2008-2010	2010	70	72	18.46	20.73	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2009-2011	2009	70	68	17.85	14.96	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2009-2011	2010	70	72	17.85	20.73	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2009-2011	2011	70	72	17.85	17.86	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2010-2012	2010	73	72	20.34	20.73	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2010-2012	2011	73	72	20.34	17.86	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
4905301301	2010-2012	2012	73	75	20.34	22.42	Zion National Park, Dalton's Wash	UT	Washington	Zion National Park
5603599911	1998-2000	1998	72	71	17.25	16.02	Pinedale	WY	Sublette	Bridger Wilderness Area

Monitor ID #	Years (3-year average)	Year (annual)	Max 8-hour (ppb) (3-year average)	Max 8-hour (ppb) (annual)	W126 (3-year average)	W126 (annual)	Monitor Site Name	State	County	Name of Class I Area Located in County
560359911	1998-2000	1999	72	72	17.25	16.88	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	1998-2000	2000	72	73	17.25	18.86	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	1999-2001	1999	71	72	16.68	16.88	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	1999-2001	2000	71	73	16.68	18.86	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	1999-2001	2001	71	69	16.68	14.31	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2000-2002	2000	71	73	17.46	18.86	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2000-2002	2001	71	69	17.46	14.31	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2000-2002	2002	71	72	17.46	19.21	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2001-2003	2001	70	69	16.63	14.31	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2001-2003	2002	70	72	16.63	19.21	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2001-2003	2003	70	70	16.63	16.36	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2002-2004	2002	69	72	15.16	19.21	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2002-2004	2003	69	70	15.16	16.36	Pinedale	WY	Sublette	Bridger Wilderness Area
560359911	2002-2004	2004	69	65	15.16	9.93	Pinedale	WY	Sublette	Bridger Wilderness Area

APPENDIX 5C: EXPANDED EVALUATION OF RELATIVE BIOMASS AND YIELD LOSS

This appendix expands to range W126 index values evaluated for relative biomass and yield loss. Specifically, Tables 5C-1 and 5C-2 below provide estimates of the relative loss for trees and crops respectively at various W126 index values using the composite E-R functions for each species for each integer W126 index value between 7 ppm-hrs and 30 ppm-hrs. The median of the composite functions is calculated for all 11 tree species excluding cottonwood. These tables also provide estimates of the number of species for trees and crops respectively that would be below various benchmarks (e.g., 2% biomass loss for trees) at various W126 index values. Table 5C-3 provides an expansion of Table 6-1 to reflect each integer W126 index value between 7 ppm-hrs and 23 ppm-hrs.

Table 5C-1 Relative Biomass Loss for Eleven Individual Tree Seedlings and Median at Various W126 Index Values

W126	Douglas Fir	Loblolly	Virginia Pine	Red maple	Sugar maple	Red Alder	Ponderosa Pine	Aspen	Tulip Poplar	Eastern White Pine	Black Cherry	Median (11 species)	Number of Species ≤ 2%	Number of Species ≤ 5%	Number of Species ≤ 10%	Number of Species ≤ 15%
30	0.1%	0.8%	1.7%	3.8%	28.1%	10.4%	12.8%	18.6%	27.7%	25.2%	53.8%	12.8%	3	4	4	6
29	0.0%	0.7%	1.7%	3.6%	23.7%	10.0%	12.3%	17.9%	26.1%	24.0%	52.6%	12.3%	3	4	5	6
28	0.0%	0.7%	1.6%	3.5%	19.9%	9.6%	11.8%	17.2%	24.5%	22.8%	51.4%	11.8%	3	4	5	6
27	0.0%	0.7%	1.6%	3.3%	16.4%	9.2%	11.4%	16.5%	23.0%	21.6%	50.1%	11.4%	3	4	5	6
26	0.0%	0.7%	1.5%	3.1%	13.4%	8.8%	10.9%	15.8%	21.4%	20.5%	48.8%	10.9%	3	4	5	7
25	0.0%	0.6%	1.4%	3.0%	10.9%	8.4%	10.4%	15.2%	19.9%	19.3%	47.5%	10.4%	3	4	5	7
24	0.0%	0.6%	1.4%	2.8%	8.7%	8.0%	10.0%	14.5%	18.4%	18.2%	46.2%	8.7%	3	4	7	8
23	0.0%	0.6%	1.3%	2.7%	6.9%	7.6%	9.5%	13.8%	17.0%	17.1%	44.8%	7.6%	3	4	7	8
22	0.0%	0.6%	1.3%	2.5%	5.3%	7.2%	9.0%	13.1%	15.6%	15.9%	43.3%	7.2%	3	4	7	8
21	0.0%	0.5%	1.2%	2.4%	4.1%	6.8%	8.6%	12.4%	14.3%	14.9%	41.9%	6.8%	3	5	7	10
20	0.0%	0.5%	1.2%	2.2%	3.1%	6.4%	8.1%	11.8%	13.0%	13.8%	40.3%	6.4%	3	5	7	10
19	0.0%	0.5%	1.1%	2.1%	2.3%	6.0%	7.6%	11.1%	11.8%	12.7%	38.8%	6.0%	3	5	7	10
18	0.0%	0.5%	1.0%	1.9%	1.7%	5.7%	7.2%	10.4%	10.6%	11.7%	37.2%	5.7%	5	5	7	10
17	0.0%	0.4%	1.0%	1.8%	1.2%	5.3%	6.7%	9.8%	9.4%	10.7%	35.6%	5.3%	5	5	9	10
16	0.0%	0.4%	0.9%	1.6%	0.9%	4.9%	6.3%	9.1%	8.4%	9.7%	33.9%	4.9%	5	6	10	10
15	0.0%	0.4%	0.9%	1.5%	0.6%	4.5%	5.8%	8.4%	7.4%	8.8%	32.2%	4.5%	5	6	10	10
14	0.0%	0.4%	0.8%	1.4%	0.4%	4.2%	5.4%	7.8%	6.4%	7.9%	30.4%	4.2%	5	6	10	10
13	0.0%	0.3%	0.8%	1.2%	0.3%	3.8%	4.9%	7.1%	5.5%	7.0%	28.6%	3.8%	5	7	10	10
12	0.0%	0.3%	0.7%	1.1%	0.2%	3.5%	4.5%	6.5%	4.7%	6.2%	26.7%	3.5%	5	8	10	10
11	0.0%	0.3%	0.6%	1.0%	0.1%	3.1%	4.1%	5.9%	3.9%	5.4%	24.8%	3.1%	5	8	10	10
10	0.0%	0.3%	0.6%	0.9%	0.1%	2.8%	3.6%	5.2%	3.2%	4.6%	22.9%	2.8%	5	9	10	10
9	0.0%	0.2%	0.5%	0.7%	0.0%	2.4%	3.2%	4.6%	2.6%	3.9%	20.9%	2.4%	5	10	10	10
8	0.0%	0.2%	0.5%	0.6%	0.0%	2.1%	2.8%	4.0%	2.0%	3.2%	18.8%	2.0%	5	10	10	10
7	0.0%	0.2%	0.4%	0.5%	0.0%	1.8%	2.4%	3.4%	1.5%	2.6%	16.7%	1.5%	7	10	10	10

Table 5C-2 Relative Yield Loss for Ten Individual Crop Species and Median at Various W126 Index Values

W126	Barley	Lettuce	Field Corn	Grain Sorghum	Peanut	Cotton	Soybean	Winter Wheat	Potato	Kidney Bean	Median (10 species)	Number of Species ≤ 5%	Number of Species ≤ 10%	Number of Species ≤ 20%	Number of Species > 5% and ≤ 10%	Number of Species > 10% and ≤ 20%
30	0.1%	5.1%	2.9%	2.3%	10.4%	16.3%	15.7%	22.5%	20.2%	36.1%	13.0%	3	4	7	1	3
29	0.0%	4.4%	2.7%	2.1%	9.7%	15.6%	15.0%	21.0%	19.4%	34.0%	12.4%	4	5	8	1	3
28	0.0%	3.7%	2.4%	2.0%	9.1%	14.9%	14.4%	19.5%	18.7%	31.9%	11.8%	4	5	9	1	4
27	0.0%	3.1%	2.2%	1.9%	8.6%	14.1%	13.7%	18.0%	18.0%	29.8%	11.2%	4	5	9	1	4
26	0.0%	2.6%	1.9%	1.7%	8.0%	13.4%	13.1%	16.6%	17.2%	27.8%	10.6%	4	5	9	1	4
25	0.0%	2.1%	1.7%	1.6%	7.4%	12.7%	12.5%	15.3%	16.5%	25.8%	10.0%	4	5	9	1	4
24	0.0%	1.7%	1.5%	1.5%	6.9%	12.0%	11.8%	14.0%	15.7%	23.9%	9.4%	4	5	9	1	4
23	0.0%	1.4%	1.3%	1.4%	6.4%	11.3%	11.2%	12.7%	15.0%	22.0%	8.8%	4	5	9	1	4
22	0.0%	1.1%	1.2%	1.3%	5.9%	10.6%	10.6%	11.5%	14.2%	20.1%	8.2%	4	5	9	1	4
21	0.0%	0.9%	1.0%	1.1%	5.4%	10.0%	10.0%	10.4%	13.5%	18.4%	7.7%	4	7	10	3	3
20	0.0%	0.7%	0.9%	1.0%	5.0%	9.3%	9.4%	9.3%	12.7%	16.6%	7.1%	5	8	10	3	2
19	0.0%	0.6%	0.8%	0.9%	4.5%	8.7%	8.8%	8.3%	12.0%	15.0%	6.4%	5	8	10	3	2
18	0.0%	0.4%	0.7%	0.8%	4.1%	8.0%	8.2%	7.3%	11.3%	13.4%	5.7%	5	8	10	3	2
17	0.0%	0.3%	0.6%	0.8%	3.7%	7.4%	7.6%	6.4%	10.5%	11.9%	5.1%	5	8	10	3	2
16	0.0%	0.2%	0.5%	0.7%	3.3%	6.8%	7.0%	5.6%	9.8%	10.5%	4.4%	5	9	10	4	1
15	0.0%	0.2%	0.4%	0.6%	2.9%	6.2%	6.4%	4.8%	9.1%	9.2%	3.9%	6	10	10	4	0
14	0.0%	0.1%	0.3%	0.5%	2.6%	5.6%	5.9%	4.1%	8.4%	7.9%	3.3%	6	10	10	4	0
13	0.0%	0.1%	0.2%	0.5%	2.2%	5.0%	5.3%	3.5%	7.7%	6.8%	2.8%	6	10	10	4	0
12	0.0%	0.1%	0.2%	0.4%	1.9%	4.5%	4.8%	2.9%	7.0%	5.7%	2.4%	8	10	10	2	0
11	0.0%	0.0%	0.2%	0.3%	1.6%	3.9%	4.3%	2.3%	6.3%	4.7%	2.0%	9	10	10	1	0
10	0.0%	0.0%	0.1%	0.3%	1.4%	3.4%	3.8%	1.9%	5.6%	3.8%	1.6%	9	10	10	1	0
9	0.0%	0.0%	0.1%	0.2%	1.1%	2.9%	3.3%	1.5%	4.9%	3.0%	1.3%	10	10	10	0	0
8	0.0%	0.0%	0.1%	0.2%	0.9%	2.5%	2.8%	1.1%	4.3%	2.4%	1.0%	10	10	10	0	0
7	0.0%	0.0%	0.0%	0.1%	0.7%	2.0%	2.3%	0.8%	3.6%	1.8%	0.8%	10	10	10	0	0

Table 5C-3 Tree Seedling Biomass Loss and Crop Yield Loss estimated for O₃ exposure over a Season.

W126 index value for exposure period	Tree seedling biomass loss ^A		Crop yield loss ^C	
	Median Value	Individual Species	Median Value	Individual Species
23 ppm-hrs	Median species w. 7.6% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤ 10% loss: 8/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 8.8 % loss ^D	≤ 5% loss: 4/10 species >5,<10% loss: 1/10 species >10,<20% loss: 4/10 species >20: 1/10 species
22 ppm-hrs	Median species w. 7.2% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 8.2 % loss ^D	≤ 5% loss: 4/10 species >5,<10% loss: 1/10 species >10,<20% loss: 4/10 species >20: 1/10 species
21 ppm-hrs	Median species w. 6.8% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 4/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 7.7 % loss ^D	≤ 5% loss: 4/10 species >5,<10% loss: 3/10 species >10,<20% loss: 3/10 species
20 ppm-hrs	Median species w. 6.4% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >40% loss: 1/11 species	Median species w. 7.1 % loss ^D	≤ 5% loss: 5/10 species >5,<10% loss: 3/10 species >10,<20% loss: 2/10 species
19 ppm-hrs	Median species w. 6.0% loss ^B	≤ 2% loss: 3/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 6.4 % loss ^D	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10,<20% loss: 2/10 species
18 ppm-hrs	Median species w. 5.7% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 7/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 5.7 % loss ^D	≤ 5% loss: 5/10 species >5,<10% loss: 3/10 species >10,<20% loss: 2/10 species
17 ppm-hrs	Median species w. 5.3% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 5/11 species ≤ 10% loss: 9/11 species ≤ 15% loss: 10/11 species >30% loss: 1/11 species	Median species w. 5.1 % loss ^D	≤ 5% loss: 5/10 species >5, <10% loss: 3/10 species >10,<20% loss: 2/10 species
16 ppm-hrs	Median species w. 4.9% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 6/11 species ≤ 10% loss: 10/11 species >30% loss: 1/11 species	Median species w. 4.4 % loss ^D	≤ 5% loss: 5/10 species >5,<10% loss: 4/10 species >10,<20% loss: 1/10 species
15 ppm-hrs	Median species w. 4.5% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 6/11 species ≤ 10% loss: 10/11 species >30% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species

14 ppm-hrs	Median species w. 4.2% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 6/11 species ≤ 10% loss: 10/11 species >30% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species
13 ppm-hrs	Median species w. 3.8% loss ^B	≤ 2% loss: 5/11 species <5% loss: 7/11 species <10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 6/10 species >5, <10% loss: 4/10 species
12 ppm-hrs	Median species w. 3.5% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 8/11 species ≤ 10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 8/10 species >5, <10% loss: 2/10 species
11 ppm-hrs	Median species w. 3.1% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 8/11 species ≤ 10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 9/10 species >5, <10% loss: 1/10 species
10 ppm-hrs	Median species w. 2.8% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 9/11 species <10% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: 9/10 species >5, <10% loss: 1/10 species
9 ppm-hrs	Median species w. 2.4% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 10/11 species >20% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: all species
8 ppm-hrs	Median species w. 2.0% loss ^B	≤ 2% loss: 5/11 species ≤ 5% loss: 10/11 species >15% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: all species
7 ppm-hrs	Median species w. ≤2% loss ^B	≤ 2% loss: 7/11 species ≤ 5% loss: 10/11 species >15% loss: 1/11 species	Median species w. ≤5% loss ^D	≤ 5% loss: all species

A Estimates here are based on the 11 E-R functions for tree seedlings described in WREA, Appendix 6F and discussed in section 5.2.1, with the exclusion of cottonwood. See CASAC comments (Frey, 2014).

B This median value is the median of the composite E-R functions for 11 tree species in the WREA, Appendix 6F (also discussed in section 5.2.1).

C Estimates here are based on the 10 E-R functions for crops described in Appendix 6F and discussed in section 5.3.1.

D This median value is the median of the composite E-R functions for 10 crops from WREA, Appendix 6F (also discussed in section 5.3.1).

United States
Environmental Protection
Agency

Office of Air Quality Planning and Standards
Health and Environmental Impacts Division
Research Triangle Park, NC

Publication No. EPA-452/R-14-006
August 2014
