

**U.S. Environmental Protection Agency
Clean Air Scientific Advisory Committee (CASAC)
Ozone Review Panel
Public Meeting**

**EPA Presentation of Revisions
to Draft Ozone Integrated Science Assessment**

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**Raleigh, NC
September 11, 2012**

Timeline for Ozone ISA

- 1st Draft – March 2011
- CASAC meeting – May 19-20, 2011
- 2nd Draft – September 2011
- CASAC meeting – January 9-10, 2012
- 3rd Draft – June 2012
- • CASAC meeting – September 11-13, 2012
- Final ISA – December 2012 target



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Chapters in Draft Ozone ISA

Preamble

Legislative and Historical Background

1. Executive Summary
2. Integrative Summary
3. Atmospheric Chemistry and Ambient Concentrations
4. Exposure to Ambient Ozone
5. Dosimetry and Mode of Action
6. Integrated Health Effects of Short-term O₃ Exposure
7. Integrated Health Effects of Long-term O₃ Exposure
8. Populations Potentially at Increased Risk for O₃-related Health Effects
9. Environmental Effects: O₃ Effects on Vegetation and Ecosystems
10. The Role of Tropospheric O₃ in Climate Change and UV-B Effects



Response to CASAC Comments

Major ISA Revisions

Particular attention was given to several important points raised by CASAC:

- Integration of evidence across scientific disciplines
- Causal determination for short-term O₃ exposure and cardiovascular effects
- Characterization of potentially at-risk populations
- Discussion of background ozone concentrations

Integration of Evidence

- Chapter 2 – Integrative Summary
 - Enhanced integration of health effects evidence across scientific disciplines and health endpoints
 - Synthesized epidemiologic evidence on differences across exposure metrics on risk estimates, regional heterogeneity in risk estimates, and concentration-response relationship
- Chapter 4 – Exposure to Ambient Ozone
 - Added discussion on long-term exposure information and implications for epidemiologic studies
 - Added population-concentration maps using data summarized in Chapter 3
 - Added time-activity information which is linked to subsequent chapters
- Chapters 6 and 7 – Health Effects of Short- and Long-term Ozone Exposure
 - Increased discussion of recent evidence with consideration of key findings from previous reviews
 - Added details regarding exposure assessment methods and measurement error issues with linkages to Chapter 4 and discussed their potential influence on heterogeneity of results among studies



Cardiovascular Effects with Short-term O₃ Exposure

- Recommendation:
 - “In the CASAC’s opinion, the evidence from toxicological, human clinical, and epidemiological studies of short-term ozone exposure all support upgrading the causal determination for cardiovascular effects from ‘suggestive of a causal relationship’ to ‘likely to be causal relationship.’”
- Carefully reconsidered the weight of evidence for the causal determination relying on EPA’s framework for causal determinations, and clearly articulated the scientific basis for decision to retain “suggestive of a causal relationship” conclusion.
 - Strong toxicological evidence from a small body of recent and past studies provides potential biologically plausible mechanisms, but of questionable translation to human responses: reflex responses, vascular oxidative stress and inflammation
 - Controlled human exposure: small number of studies provide inconsistent results
 - Epidemiologic evidence:
 - Consistent, positive associations between short-term O₃ exposure and cardiovascular mortality
 - Inconsistent findings for cardiovascular morbidity (e.g., heart rhythm, physiological biomarkers, and hospital admissions or emergency department visits)

Cardiovascular Morbidity: Epidemiologic results

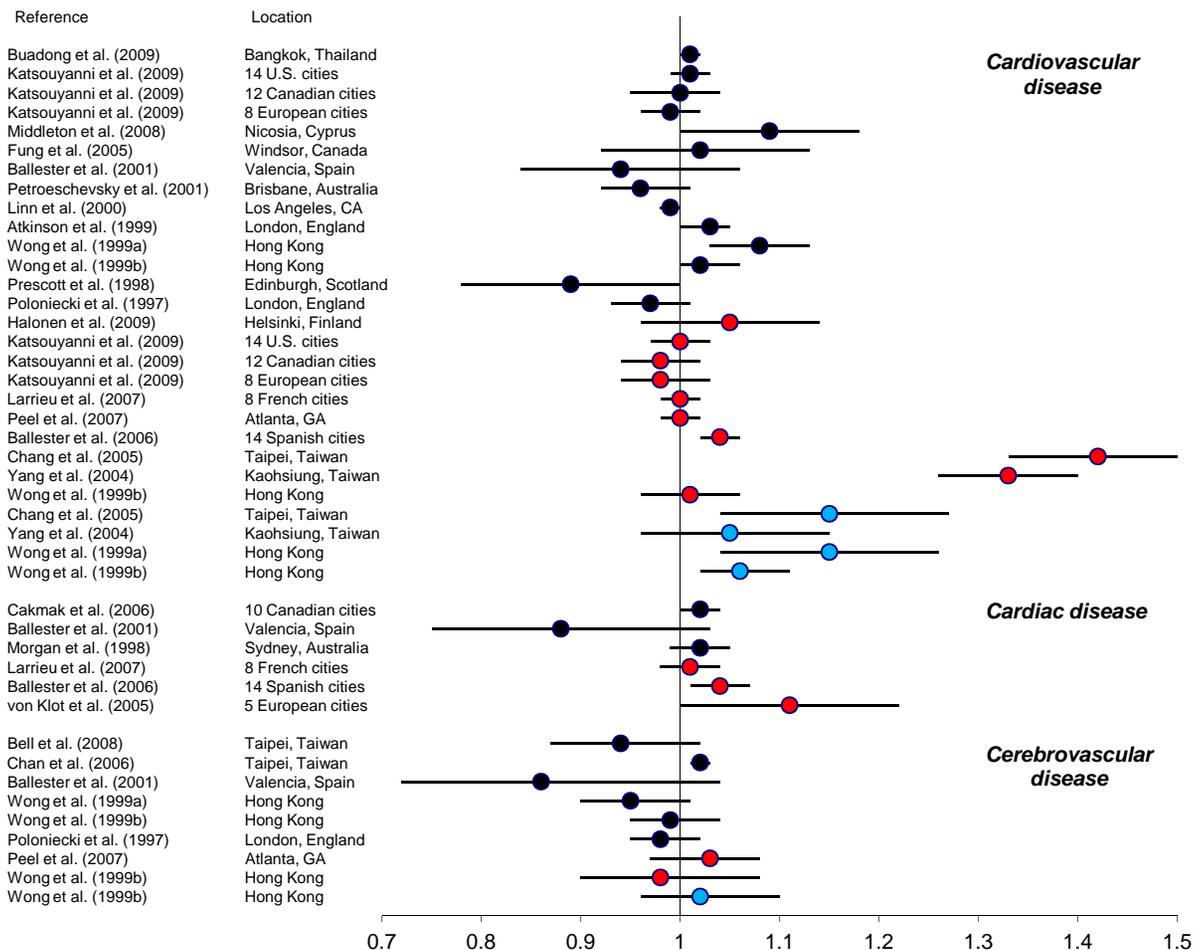


Figure 6-21. Odds ratio (95% CI) per increment ppb increase in ozone for over all cardiovascular ED visits or HAs.

Note: Increase in O₃ standardized to 20 ppb for 24-h avg period, 30 ppb for 8-h avg period, and 40 ppb for 1-h avg period. Ozone concentrations in ppb. Seasons depicted by colors – black: all year; red: warm season; light blue: cold season. Age groups of study populations were not specified or were adults with the exception of Fung et al. (2005), Wong et al. (1999b), and Prescott et al. (1998), which included only individuals aged 65+.

Cardiovascular Morbidity: Epidemiologic results (cont)

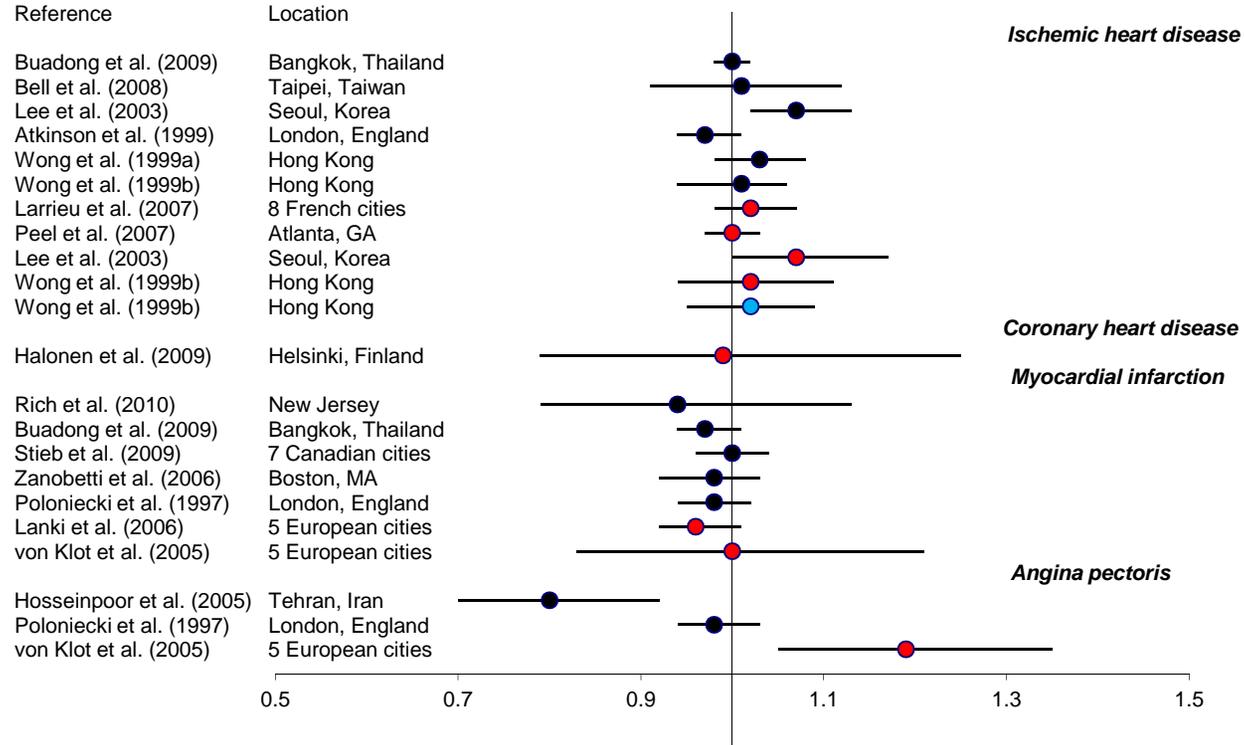


Figure 6-23 Odds Ratio (95% confidence interval) per increment ppb increase in ozone for ischemic heart disease, coronary heart disease, myocardial infarction, and angina pectoris ED visits or HAs.

Note: Increase in O₃ standardized to 20 ppb for 24-h averaging period, 30 ppb for 8-h averaging period, and 40 ppb for 1-h averaging period. Ozone concentrations in ppb. Seasons depicted by colors: black: all year; red: warm season; light blue: cold season. Age groups of study populations were not specified or were adults with the exception of Wong et al. (1999a) and Atkinson et al. (2006a), which included only individuals aged 65+.

Characterization of potentially at-risk populations or lifestages

<p>Adequate evidence</p>	<p>There is substantial, consistent evidence within a discipline to conclude that a factor results in a population or lifestage being at increased risk of air pollutant-related health effect(s) relative to some reference population or lifestage. Where applicable this includes coherence across disciplines. Evidence includes multiple high-quality studies.</p>
<p>Suggestive evidence</p>	<p>The collective evidence suggests that a factor results in a population or lifestage being at increased risk of an air pollutant-related health effect relative to some reference population or lifestage, but the evidence is limited due to some inconsistency within a discipline or, where applicable, a lack of coherence across disciplines.</p>
<p>Inadequate evidence</p>	<p>The collective evidence is inadequate to determine if a factor results in a population or lifestage being at increased risk of an air pollutant-related health effect relative to some reference population or lifestage. The available studies are of insufficient quantity, quality, consistency and/or statistical power to permit a conclusion to be drawn.</p>
<p>Evidence of no effect</p>	<p>There is substantial, consistent evidence within a discipline to conclude that a factor does not result in a population or lifestage being at increased risk of air pollutant-related health effect(s) relative to some reference population or lifestage. Where applicable this includes coherence across disciplines. Evidence includes multiple high-quality studies.</p>

Characterization of potentially at-risk populations or lifestages

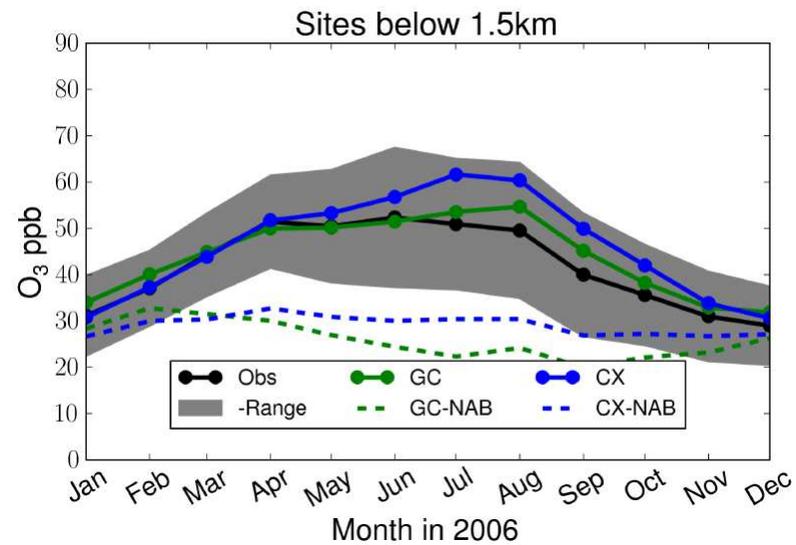
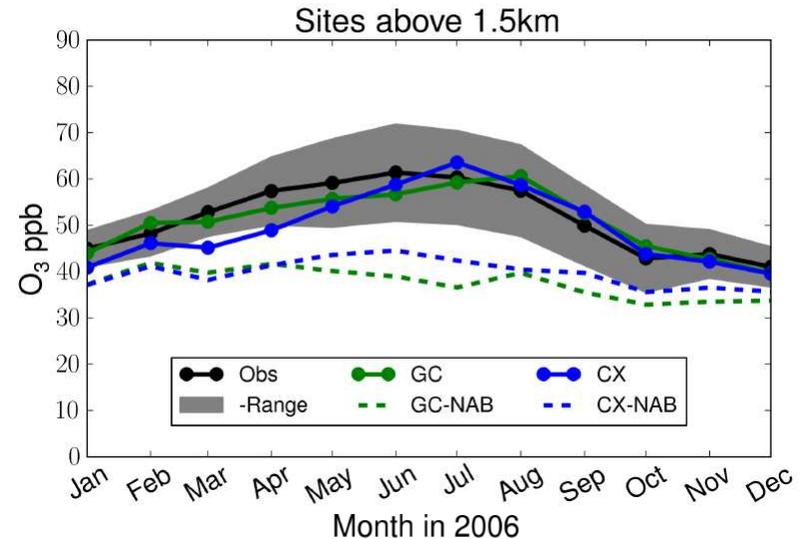
Evidence Classification	Potential At Risk Factor
Adequate evidence	<ul style="list-style-type: none"> Asthma (Section 8.2.2) Children, Older Adults (Section 8.3.1) Diet (Section 8.4.1) Outdoor workers (Section 8.4.4)
Suggestive evidence	<ul style="list-style-type: none"> Genetic factors (Section 8.1) Sex (Section 8.3.2) SES (Section 8.3.3) Obesity (Section 8.4.2)
Inadequate evidence	<ul style="list-style-type: none"> Influenza/Infection (Section 8.2.1) COPD, CVD, Diabetes (Sections 8.2.3 – 8.2.5) Hyperthyroidism (Section 8.2.6) Race/ethnicity (Section 8.3.4) Smoking (Section 8.4.3) Air conditioning use (Section 8.4.5)
Evidence of no effect	–

New Analyses of Ozone Background Concentrations

Simulations by a regional scale model (CX for CAMx) were considered in addition to results from a global model (GC for GEOS-Chem) for monthly mean daily maximum 8-hr average O₃ concentration.

Results show little difference even though the resolution of the regional model is 12 x 12 km and global model is 50 x 50 km indicating that factors in addition to model resolution need to be considered in interpreting the results.

New material (Fig. 3-14)



New Analyses of Ozone Background Concentrations

New Table 3-1: Comparison of Zhang et al. (2011) and Emery et al. (2012) base case models with measurements at CASTNET sites

Region	CASTNET		GEOS-Chem		CAMx	
	Spring	Summer	Spring	Summer	Spring	Summer
California (5) ^a	58 ± 12 ^b	69 ± 14 ^b	52 ± 11 ^b ; 0.52 ^c 38 ± 7 ^d ; 37±6 ^e	66 ± 18 ^b ; 0.22 ^c 37 ± 9 ^d ; 35±9 ^e	50 ± 10 ^b ; 0.50 ^c	66 ± 13 ^b ; 0.30 ^c 42 ± 6 ^d
West (14)	54 ± 9	55 ± 11	53 ± 7; 0.30 42 ± 6; 41±6	55 ± 11; 0.12 40 ± 9; 38±9	49 ± 8; 0.39 40 ± 7	57 ± 10; 0.33 41 ± 8
North Central (6)	47 ± 10	50 ± 12	47 ± 8; 0.52 33 ± 6; 30±7	51 ± 14; 0.44 27 ± 7; 24±7	45 ± 11; 0.63 30 ± 6	54 ± 13; 0.48 31 ± 5
Northeast (5)	48 ± 10	45 ± 14	45 ± 7; 0.44 33 ± 7; 29±6	45 ± 13; 0.47 24 ± 7; 18±6	46 ± 11; 0.53 30 ± 5	53 ± 14; 0.54 27 ± 6
Southeast (9)	52 ± 11	52 ± 16	51 ± 7; 0.42 32 ± 7; 29±7	54 ± 9; 0.21 29 ± 10; 28±9	54 ± 9; 0.56 33 ± 6	61 ± 12; 0.45 30 ± 6

^aValues in parentheses after each region name refer to the number of sites.

^bShown are seasonal (spring, summer) MDA8 O₃ concentration means (ppb ± standard deviation).

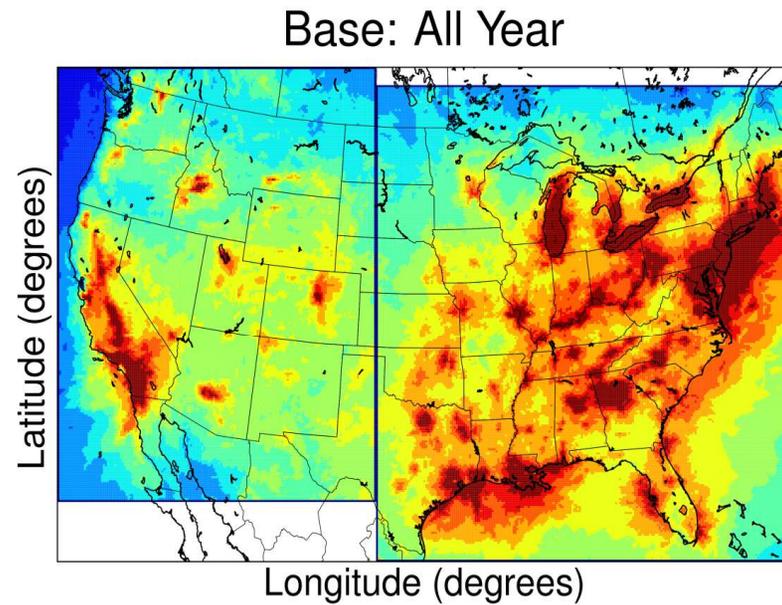
^cShown are mean R² of all individual model-measurement pairs at individual CASTNET sites.

^dUS background mean MDA8 O₃ concentrations (ppb ± standard deviation) are shown beneath the base case means.

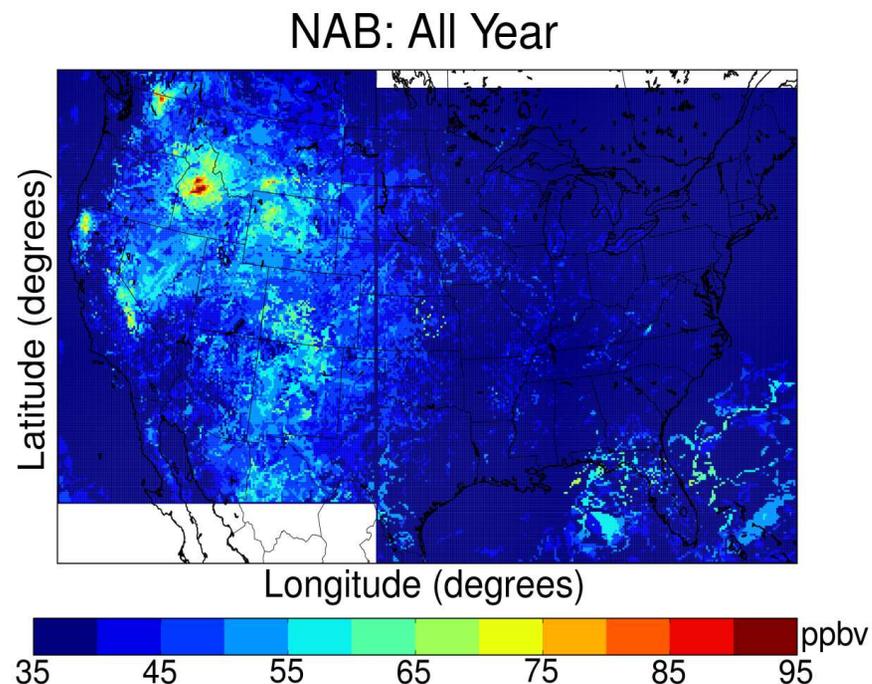
^eNA background mean MDA8 O₃ concentrations (ppb ± standard deviation).

Source: Data from [Zhang et al. \(2011\)](#) and [Emery et al. \(2012\)](#).

Upper panel: 99th percentile (4th highest) daily maximum 8-hr average O₃ concentrations calculated by CAMx for the base case (i.e., including all natural and anthropogenic sources everywhere in the world).



Lower panel: North American background concentrations on the same days as the 4th highest concentrations predicted by the base model



New material (Fig. 3-16)

Response to CASAC Comments

Additional ISA Revisions

There were many other revisions in response to CASAC, including:

- Added flow diagrams to the Preamble
- Removed Continuum of Respiratory Effects figures from Chapters 1 and 2
- Added human activity patterns (e.g., time spent outdoors, breathing rates) to Chapter 4 and activity levels used in experimental studies to Chapters 5 and 6
- Characterized the potential for effects from O₃ versus O₃ reaction products in Chapter 5 with consultation and contribution from an outside expert
- Added discussion to Chapter 6 on the time course respiratory effects and the effect of prior ambient exposures on responses in controlled human studies
- Added rationale to Chapter 7 explaining the grouping of reproductive and developmental outcomes (including infant mortality) discussions within long-term O₃ exposure effects
- Added discussion on future trends in tropospheric O₃; expanded discussion of the IPCC Representative Concentration Pathways scenarios; incorporated radiative forcing from O₃ precursor emissions; and UV-B shielding discussion revised to be more concise with clear conclusions in Chapter 10
- Added causal determination tables to end of Chapters 9 and 10



General Charge to CASAC

Please comment on the adequacy of these and other changes to the chapters and recommend any revisions to improve the discussion of key information. Please recommend any revisions that may further improve the clarity of discussion.