Responses to CASAC Questions on the Ozone PA from Consultant Dr. Sonja Sax

Questions from Dr. Corey Masuca

1) 2.1. Ozone and Photochemical Oxidants in the Atmosphere

How sound science is this mechanism of ozone transfer between the stratosphere and the troposphere?

> I am not sufficiently familiar with these transport mechanisms to adequately answer this question.

2) 2.3.1 Ambient Air Monitoring Requirements and Monitoring Networks

While a number of types of sites are mentioned in this section such as PAMS, NCore, CASTNET, National Park Service (NPS), and Special Purpose Monitors (SPMs), what about Near Road Monitoring Sites, especially for NOy?

> I agree that any data or information from the Near Road monitors should be included and discussed.

3) 2.3.2 Data Handling Conventions and Comparisons for Determining Whether Standards Are Met

There is a reference to the hourly concentrations being utilized to compute 8-hour averages. Is this short-term 8-hour rolling average consistent with short-term actual and scientific studies?

> This is an important question that has been raised by others (e.g., Dr. Lange). In general, I don't think there is clear agreement between how the NAAQS design values are calculated (for determining whether an area is in compliance with the NAAQS) and how exposures are evaluated in the epidemiological literature. In addition, there is also some discordance between how exposures are evaluated in controlled human exposure studies and animal studies, and how the NAAQS is determined from monitoring stations. My colleagues and I discuss this and related issues in the following publication:
>
> Goodman, JE; Sax, SN; Lange, SS; Rhomberg, LR. 2015. "Are the Elements of the Proposed Ozone National Ambient Air Quality Standards Informed by the Best Available Science?" *Reg. Tox. Pharmacol.* 72(1):134-140.

4) 2.4.3 Diurnal Patterns

While this section refers diurnal patterns of relative ozone concentrations between day and night, are these diurnal patterns solely (although mostly are) attributable to temperature? What about stagnant weather conditions? What about the effects on topography/geography in determining diurnal patterns?

> EPA does a good job in summarizing information related to not only diurnal patterns, but also regional patterns where specific weather conditions (such as stagnant weather patterns) and/or topography could impact ozone concentrations (i.e., based on the selection of examples provided

as in Figures 2-10 and 2-11). While additional discussion could be included to specifically address these points, the examples provided show a good range of different conditions that could impact ozone concentrations spatially and regionally.

5) Background Ozone

There, in general appears to be a lot of discussion about background ozone concentrations from transport and natural sources. However, are most salient ozone concentrations more localized and from anthropogenic sources?

I appreciate that EPA includes a thorough discussion of background sources of ozone because in some regions of the country, background ozone levels can be a significant contributor to overall ozone levels, and this makes it challenging to meet increasingly more stringent NAAQS. Understanding when and how much background ozone contributes to overall ozone levels will help in attainment of the NAAQS.

This section references the utilization of photochemical grid models due to the lack of ability to characterize the origins of ozone and the ability to estimate the magnitude of background ozone. However, how predictable are these photochemical models, especially given the highly photolytic and relative instability of ozone in the atmosphere?

I am not sufficiently familiar with the photochemical models to comment on this question.

This section mentions that international emissions sources via transport mostly originate from anthropogenic sources. However, is there a possibility that there can be international transports from non-anthropogenic/biogenic sources?

I agree that it is possible for international transport of biogenic precursors to also contribute, but it may be more difficult to evaluate the contributions from these sources.

Also, this section noticeably leaves out non-international, interstate transport of ozone.

I agree that this is an important issue that should be addressed.

6) 2.5.1.6 Pre-Industrial Methane

There is a whole section devoted to long-lasting atmospheric methane. However, what is the importance of methane with respect to the formation of and consideration of ozone? Is a discussion on methane warranted?

I am not sufficiently familiar with all the details regarding ozone chemistry, if it does play a significant role in ozone formation, then I think it is appropriate for methane to be discussed.

Questions from Dr. Sabine Lange

Air Quality

1) *Multiple ozone chemistry analyses (e.g. Downey et al., 2015; Simon et al., 2012) have demonstrated that in an area where peak daily ozone concentrations have decreased over time, over the same period of time the lowest daily ozone concentrations have also decreased (due to the NOx disbenefit aspect of ozone chemistry). An example is provided in Figure 1. What are your thoughts about the change of annual average ozone concentrations (which tend to be the focus of epidemiology studies) with decreases in annual peak ozone concentrations?*

   EPA does acknowledge that "Reductions of NOX emissions are expected to result in a compressed O3 distribution, relative to current conditions" (Draft Ozone PA, pg. 2-4), and it looks like that is what is shown in Dr. Lang's Figure 1. As Figure 1 shows, however, this also means that there will be more days that experience somewhat higher ozone concentrations, although potentially no days with levels that exceed very high concentrations. With regards to how these changes should be interpreted for epidemiology studies, I think the larger issue of how ambient levels relate to actual personal exposures of ozone and how this impacts exposure measurement error in the epidemiology studies is a more critical issue. In the PA, EPA acknowledge this important source of uncertainty, and is one reason provided for not conducting the "epidemiology-based" risk assessment.

Epidemiology

2) *Is an epidemiology study with higher statistical power (sample size) innately more protected against problems of confounding, error, and bias, than an epidemiology study with lower statistical power (sample size)?*

   The issue of statistical power is separate from issues related to confounding, errors and bias. You can have a very large study that has serious confounding issues if these are not controlled for (or are unmeasured). Similarly, large studies can be prone to selection bias, exposure measurement errors, etc. Sample size (or statistical power) will affect whether you are able to "detect" an effect, and is only one aspect of study quality (larger sample sizes are preferred), but is separate from other issues of study quality, which are associated with the study design, execution, and analyses methodology. That is, poor study design, execution or poor methodology can lead to errors and biases.

3) *In section 3.3.3 (Exposure Concentrations Associated with Effects) and section 3.3.4 (Uncertainties in the Health Effects Evidence), the EPA notes that the epidemiology studies are generally assessing the associations between ambient ozone and specific health outcomes and are not investigating the details of the exposure circumstances eliciting these effects (e.g. pg 3-40 and pg 3-43). Do you think that this statement is correct? If so, is this statement generally true of air pollution epidemiology studies, or is it peculiarly specific to ozone? If it is not specific to ozone, then should this caveat always be considered when evaluating exposure concentrations associated with these types of epidemiology studies?*

I agree with this statement – the ambient data, whether from fixed-site monitors or from modeling data are only surrogates of the actual personal exposures and any differences contribute to exposure measurement errors. This statement is true for all air pollution studies, not only ozone, and this caveat should be included for other air pollution epidemiology studies.

Exposure-Response Modeling

4) *In section 3.4.4 (Key Uncertainties) of this PA, the EPA notes that "In recognition of the lack of data for some at risk groups and the potential for such groups, such as children with asthma, to experience lung function decrements at lower exposures than healthy adults, both models generate nonzero predictions for 7-hour concentrations below the 6.6-hour concentrations investigated in the controlled human exposure studies." Is assuming a lack of threshold in an exposure-response relationship a standard method for considering potential at-risk populations that may not have been characterized in an exposure-response assessment?*

   This approach does not make sense to me. If asthmatics are truly more susceptible to the effects of ozone, then it might be that the threshold for effects might be lower, but not zero. Although data are limited, the data that are available do not indicate that asthmatics are more susceptible than non-asthmatics to the effects of ozone. In fact, data are inconsistent, with some studies indicating effects in asthmatics at elevated ozone exposures, but others showing no effects. For example, no effects on lung function were observed in asthmatics compared to non-asthmatics at exposures to 400 ppb for 2 hours (Alexis *et al*., 2000) and 200 ppb for 2 hours (Mudway *et al*., 2001).

   Alexis, N; Urch, B; Tarlo, S; Corey, P; Pengelly, D; O'Byrne, P; Silverman, F. 2000. "Cyclooxygenase metabolites play a different role in ozone-induced pulmonary function decline in asthmatics compared to normals." *Inhal. Toxicol.* 12(12):1205-1224.

   Mudway, IS; Stenfors, N; Blomberg, A; Helleday, R; Dunster, C; Marklund, SL; Frew, AJ; Sandstrom, T; Kelly, FJ. 2001. "Differences in basal airway antioxidant concentrations are not predictive of individual responsiveness to ozone: A comparison of healthy and mild asthmatic subjects." *Free Radic. Biol. Med.* 31(8):962-974.

5) *The EPA also notes in this section that there is a lack of information about the factors that make people more susceptible to ozone-related effects, and that the risk assessment could therefore be underestimating the risk. However, the exposure-response model used to estimate the risk of lung function decrements uses those people in the health population with a greater response to ozone than the mean response (i.e. that fraction of the people in controlled human exposure studies who had FEV1 responses >10%, 15%, or 20%). Does this method already include consideration for more susceptible people in the population?*

   The controlled human exposure studies that form the basis of the exposure-response model are based on exposure circumstances that are highly unlikely to occur in the general population, and in particular in susceptible population groups (i.e., heavily exercising individuals exposed to elevated concentrations of ozone over extended periods of time). Only outdoor workers are likely to experience the exposure conditions in these studies. In addition, the results clearly indicate that only a small percentage of the study

volunteers (although generally healthy adults) had a statistically significant response to ozone, and as noted by Dr. Lange, these responders likely represent people that are more susceptible to ozone (particularly at lower ozone concentrations). Therefore, I agree that the model already represents a very conservative estimation of ozone effects that are likely to be protective of sensitive population groups.

Questions from Dr. James Boylan

Chapter 2 – Air Quality

- *Is the discussion on O_3 and Photochemical Oxidants in the Atmosphere (Section 2.1) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Sources and Emissions of O_3 Precursors (Section 2.2) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Ambient Air Monitoring and Data Handling Conventions (Section 2.3) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Ozone in Ambient Air (Section 2.4) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Background O_3 (Section 2.5) accurate and complete? If not, what additional information needs to be included?*

Overall, for the PA, the discussion of Air Quality seemed accurate and complete. As noted by other CASAC members, inclusion of state-to-state transport of ozone and ozone precursors could be included, in addition to a discussion of potential international transport of non-anthropogenic precursors of ozone.

Chapter 3 – Review of the Primary Standard

- *Is the discussion on Exposure and Risk Conceptual Model and Assessment Approach (Section 3.4.1) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Population Exposure and Risk Estimates for Air Quality Just Meeting the Current Standard (Section 3.4.2) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Population Exposure and Risk Estimates for Additional Air Quality Scenarios (Section 3.4.3) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Key Uncertainties (Section 3.4.4) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Public Health Implications (Section 3.4.5) accurate and complete? If not, what additional information needs to be included?*

In general, the overall summary of the Review of the Primary Standard was adequate for the PA. The modeling of the Population and Exposure Risk Assessment (which I only briefly reviewed) appeared to be very extensive and thorough, although I did not see any of the actual estimates of the individual exposures, which would have been interesting to see. Also, it would be interesting to compare the modeled estimates to actual personal

exposure studies to provide some validation for the model (I did not see any discussion of model validation).

Appendix 3C – Air Quality Data Used in Population Exposure and Risk Analyses

- *Is the discussion on Urban Study Areas (Section 3C.2) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Ambient Air Ozone Monitoring Data (Section 3C.3) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Comprehensive Air Quality Model with Extensions (CAMx) (Section 3C.4.1) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Evaluation of Modeled Ozone Concentrations (Section 3C.4.2) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Air Quality Adjustment to Meet Current and Alternative Air Quality Scenarios (Section 3C.5) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Interpolation of Adjusted Air Quality using Voronoi Neighbor Averaging (Section 3C.6) accurate and complete? If not, what additional information needs to be included?*
- *Is the discussion on Results for Urban Study Areas (Section 3C.7) accurate and complete? If not, what additional information needs to be included?*

In general, these sections of the PA appeared to be adequate and complete. I did not review in detail to assess whether the information is accurate, as that would be beyond my available time and expertise. The results and discussion seemed appropriate.