Oral Statement to Clean Air Scientific Advisory Committee on the *Integrated Science Assessment for Ozone and Related Photochemical Oxidants, External Review Draft*

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My name is Randy Mandel, and I help lead Ramboll's Ecology Practice. My comments today pertain to the Draft 2019 Integrated Science Assessment for Ozone and Related Photochemical Oxidants - Appendix 8 "Ecological Effects". More detailed comments have also been submitted in writing to EPA.

I have over 30 years of experience in restoration ecology and ecophysiology and specialize in site-specific native plant ecology. I've published multiple articles and work internationally as a subject matter expert.

The Draft 2019 Ozone ISA Appendix 8 considers substantially more information than the Final 2013 Ozone ISA. Notwithstanding, there are a number of critical deficiencies in the 2019 analysis. In general, the Draft 2019 Ozone ISA oversimplifies its summarized evidence and has a number of technical inaccuracies, which, in turn, have the overall effect of making it difficult to ascribe if, and at what level, there is specific potential harm for the purposes of setting standards. These inaccuracies and over-simplifications will be described in these comments. Additionally, the draft fails to substantiate several of its causal effects determinations. Finally, there are many confounding factors that affect the validity of the conclusions EPA has drawn. For example, analysis of the reduced plant growth and biomass fails to consider plant age and how the proportion of the plant that uptakes tropospheric ozone changes with age, affecting biomass production.

EPA's justification for a causal relationship between ozone and foliar injury is not robust. The exact parts per million level of ozone that causes foliar injury is difficult to determine due to incomplete/ insufficient data summaries within the document as well as the inherent variation in ozone sensitivity between species, populations, and phenotype. EPA should clarify the variability of results in association with phenotype and metabolic pathways. Furthermore, EPA does not properly assess the degree to which their results can reasonably be extrapolated to other plants and species. EPA should also better emphasize the lack of information connecting foliar injury with additional effects on plant or ecosystem health.

In regards to reduced plant growth and biomass, confounding effects of plant age were not adequately considered. Specifically, plant age increases the percent of the plant that is woody and, thus, the proportion of the plant that is actively involved in internal water conductance and atmospheric gas exchange. Therefore, increased variance in plant age distribution likely creates a larger spread in a plant's response to ozone – a fact that is not fully considered within the analysis.

Also, the plant reproduction section is largely based on agronomic data. Native and agronomic plants have differing responses to ozone, and extrapolation of crop data to all species leads to further uncertainly when these conclusions are applied to native plants.

EPA's section on increased tree mortality is substantiated, but the specific response of different species to ozone is unclear from the presentation of the information. Furthermore, the statistical significance of

the findings is low in some cases, likely attributed to small sample sizes and differences between populations.

The section on the effects of ozone on herbivore growth and reproduction uses a range of studies with tenuous extrapolation to all species. EPA's arguments would be better supported by a more detailed consideration of how herbivore growth and reproduction vary by species and whether the distribution of impacts also varies by species.

There is a lack of substantiation of the ozone effects on plant-insect signaling. Extrapolation of data from a single species increases uncertainty – there is a need for a greater variety of organismic diversity. This analysis is not well supported. EPA should consider if there is sufficient information to include this in the Final Ozone ISA, particularly since it was not included in 2013 Ozone ISA.

Carbon sequestration within natural ecosystems is extremely complex and depends on the soil microbia and fungi, moisture, and other factors. The causal relationship between ozone and carbon sequestration is difficult to estimate because these relationships are very intricate. This, in turn, serves to complicate the determination of ozone impacts, similarly affecting the next three end points (below-ground biogeochemical cycles, alternation of terrestrial community composition, and ecosystem water cycling).

The effect of synergetic carbon-water-organismic relationships on the below-ground biogeochemical cycles is not properly considered, thereby leading to potential misinterpretation of effect.

The below-ground biogeochemical cycle considerations are relevant, as mycorrhiza influence how long plants will leave their stoma open. Further, analysis of below-ground biogeochemical cycling would benefit from considering the inter-relationship with other reproduction methods (i.e. seed production) and terrestrial community composition (i.e. invasive species). As with other causality factors, this makes it difficult to ascertain the possible ozone concentration that may cause potential impact.

Finally, the influence of ozone on ecosystem water cycling is poorly represented within both the 2013 and 2019 ISAs. Specifically, the ISAs do not consider the confounding influence of the difference between dry, intermediate, and wetland ecological communities and how the ecological system affects the length of time plants will keep their stoma open and, therefore, their susceptibility to ozone.

I have submitted written comments that expand on the above points to help provide more specific information. Thank you for your time today and considering my concerns.