

Oral Testimony Before the CASAC Ozone Review Panel on the First Draft Ozone REA

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Thank you for the opportunity to speak today on behalf of the American Petroleum Institute. These comments will focus on the general approach used in the first draft REA (US EPA, 2012) for estimating health effects associated with short-term ozone exposures based on the epidemiology evidence. Overall, EPA should restrict the risk assessment to respiratory morbidity outcomes and directly calculate risk reductions using BenMAP. If it does present risk estimates for mortality, EPA must fully acknowledge the uncertainty and variability in these estimates, which could include a risk estimate of zero based on the fact that evidence is not strong enough to support a causal association.

There is a substantial amount of uncertainty in the underlying epidemiology studies that form the basis of the core risk estimates. EPA should restrict its evaluation to respiratory morbidity because this is the only endpoint that it determined was "causal."

EPA notes that mortality is the most important endpoint, even though it classified it as only "likely causal." Evaluation of health endpoints with a "likely causal" classification, if included at all, should be clearly qualified as being much more uncertain. Endpoints that are only suggestive, including cardiovascular morbidity and mortality effects, should not be considered in the risk assessment. The evidence is not supportive of these health endpoints.

Another important issue relates to the way that EPA used BenMAP to estimate health effects. EPA generated two BenMAP model runs to calculate "total" ozone-related risks down to an ozone concentration of zero, one for recent ozone ambient levels and one for rolled-back levels that meet the current standard. As EPA acknowledged, there is uncertainty in extrapolating health risks from exposures that go beyond the ozone levels that were measured in the epidemiology studies. EPA presented a second set of analyses in which "total" risks are modeled down to the lowest measured level (LML) in the epidemiology studies. This method is more appropriate in that it accounts for some of the uncertainty in the shape of the concentration-response function, particularly outside the range of available data, and it reflects a more realistic scenario than one in which there is, on average, a zero level of ozone exposure. EPA needs to acknowledge that this level of ozone is unattainable. Still, EPA should calculate the difference in risks for current or alternative ozone standards directly and consider other lower ozone bounds for calculating risks. Also, in applying the LML bounds, further uncertainty is introduced from the use of surrogate LMLs, and this uncertainty needs to be fully accounted for.

In addition, to determine the risk reduction resulting from attainment of the current ozone standard, EPA simply calculated the difference between the "total" risk point estimate calculated for current ambient ozone concentrations and the rolled-back ozone concentrations. In doing so, EPA did not generate confidence bounds for any of the risk reductions (because these are based on point estimates). These risk reductions are misleading because there is no way to judge how uncertain they are without confidence bounds.

If EPA is considering mortality endpoints, an important issue that EPA needs to address is the unexplained heterogeneity in the effect estimates from multi-city studies. Although EPA is using city-specific mortality estimates in the risk assessment, the variability is minimized by the Bayesian approach that is applied to these estimates. Instead, EPA should at a minimum use regionally adjusted, rather than nationally adjusted, estimates. In addition, EPA should use multi-pollutant models because of evidence of confounding effects from PM, indicating a reduction in risk when PM is included in the model. If confounding is not accounted for in the risk assessment, effect estimates will be overstated.

Overall, EPA also needs to consider quantitatively more fully the uncertainty in the underlying epidemiology studies and how this uncertainty impacts the risk estimates. It is not sufficient to discuss these issues qualitatively. A rigorous sensitivity analysis that addresses all sources of uncertainty is the only way to determine if risk estimates can be relied on to inform the level of the NAAQS.

In conclusion, EPA should restrict the risk assessment to respiratory morbidity outcomes because these are the only ones EPA has concluded are causal. In addition, EPA should directly calculate risk reductions using BenMAP and include confidence bounds around these estimates. Lastly, if EPA presents risk estimates for mortality it must more fully acknowledge the uncertainty and variability in these estimates.

References

US EPA. 2012. "Health Risk and Exposure Assessment for Ozone (First External Review Draft)." EPA 452/P-12-001. 474p., July.