**Oral Comments on the First External Review Draft of the "Health Risk and Exposure Assessment for Ozone" Prepared for the CASAC Ozone Panel**

**By**
**Jon M. Heuss**
**Air Improvement Resource, Inc.**

**September 11, 2012**

I am Jon Heuss of Air Improvement Resource, Inc. (AIR). We are reviewing the draft REA[1] for the Alliance of Automobile Manufacturers. We identified many issues with the third draft ISA and provided detailed comments to the Agency[2] that serve as the scientific support for these comments.

The REA list a number of goals. We have comments on each. The first listed goal is to provide estimates of the number of people with $O_3$ exposures with moderate or greater exercise above benchmark levels. This calculation, however, is not directly a measure of risk of adverse effects or risk to public health. In addition, there are three ways in which the estimates in the draft are biased high. For example, we document that the APEX model predicts more elevated ventilation rate occurrences than observed in the real world data from Brochu et al. 2006.[3] In 1996, EPA acknowledged that the risk analysis allowed more high ventilation rates (hence greater risk) than would actually occur in the populations of interest. The 2007 Langstaff memo on uncertainty also acknowledged that the values produced by the ventilation rate algorithm exhibit an excessive degree of variability.

A second way relates to how EPA defines moderate or greater exercise over 8 hours. The REA follows the approach begun in 1996 of defining EVR's between 13 and 27 as moderate.[4] Risks are calculated for individuals with daily 8-hour average EVR greater than 13 using response functions developed from chamber study data conducted at a significantly higher EVR, ~ 20. Ted Johnson showed the EPA algorithm predicts that the 95$^{th}$ percentile 8-hour EVR is between 14 and 15 while the EVR used in the clinical studies of 20 is about the 99$^{th}$ percentile.[5] Thus, the resulting headcounts and risks are

---

[1] US EPA. 2012. "Health Risk and Exposure Assessment for Ozone (First External Review Draft)." EPA 452/P-12-001. 474p., July.
[2] J. M. Heuss and G. T. Wolff, Review and Critique of the U. S. Environmental Protection Agency's Third External Review Draft of the Integrated Science Assessment for Ozone and Related Photochemical Oxidants, Air Improvement Resource, Inc. Report prepared for The Alliance of Automobile Manufacturers, August 20, 2012. Docket No. EPA-HQ-ORD-2011-0050-0044.
[3] Ibid., at page 18.
[4] REA, supra note 1, at page 5-16.
[5] T. Johnson and J. Capel, "Background Information on EVR Sequence Statistics, September 25, 2007, Attachment 2 to Comments of the American Petroleum Institute on National Ambient Air

unreasonably high.

A third way is that ozone at breathing height is lower than ozone at measurement height. This was acknowledged in the last review but not evaluated in the health RA even though the analogous difference between ozone at plant height and ozone at measurement height was evaluated in the welfare RA. The sensitivity to these three factors should be evaluated in the next draft.

The second listed goal is to provide estimates of the number of people with various FEV1 decrements. This calculation is necessary but not sufficient to estimate risk of adverse effects since it does not include estimates of lung function decrements accompanied by respiratory symptoms, as the American Thoracic Society Guidelines recommend. As noted in the draft, the risks (which we point out are biased high) are similar to those estimated in the last review. We urge EPA to include the new McDonnell et al., 2012[6] concentration-response function model in the next draft and discuss how the results relate to adversity.

The third goal is to provide estimates of the potential magnitude of premature mortality and/or selected morbidity health effects. Rather than using EPA's preferred positive associations, we urge the Agency to explore the full range of associations in the literature. If this is done it will become apparent, as Koop and Tole pointed out in 2004:[7]

> …when model uncertainty is accounted for in the analysis, measures of uncertainty associated with these point estimates became very large. Indeed they became so large that the hypothesis that air pollution has no effect on mortality is not implausible.

The fourth and fifth goals are to understand the influence of various inputs and assumptions on the risk estimates and the uncertainties in those estimates. This is laudable and important. In addition to the issues for the clinical studies, the REA should evaluate the wide range of positive and negative associations in individual cities and the temporal and spatial variations in the combined associations in multi-city studies. For example, in the regional analysis by Bell and Dominici, 2008 only two of seven regions have positive and statistically significant ozone/mortality associations while two have small negative associations. In another example, in the Medina-Ramon et al., 2006 study of 36 U. S. cities the individual-city associations for COPD hospital admissions in the summer ranged from –30 % to +40 % for a 0.030 ppm increase in 8-hour ozone. The individual-city associations for pneumonia hospital admissions ranged from –15% to +

---

Quality Standards for Ozone, Proposed Rule, October 9, 2007, Docket No. EPA-HQ-OAR-2005-0172-12158-1.1

[6] W. McDonnell, P. Stewart, M. Smith, C. Kim, and E. Schelegle, "Prediction of lung function response for populations exposed to a wide range of ozone conditions." *Inhal. Toxicol.,* 24(10), 619-633. (2012).

[7] G. Koop and L. Tole,"Measuring the Health Effects of Air Pollution: to What Extent Can We Really Say that People are Dying from Bad Air," *J. of Environmental Economics and Management*, 47, 30-54. (2004).

20% for a 0.030 ppm increase in 8-hour ozone. The combined associations for the two categories were positive in the warm season, but were negative in the cold season and not significant over all year. By switching the baseline analysis for the Medina-Ramon study to the all-year result and exploring the spatial and temporal differences, the limitations of the epidemiologic risk assessment will become apparent.

The sixth goal is to understand the national mortality burden associated with ozone. Rather than use the Bayesian combined estimates from Bell et al., 2004, a variety of other possible approaches and baselines should be considered. If the individual city results from Bell et al., 2004 are considered, the pattern includes both positive and negative cities, which is not biologically plausible. If the regional combined results from Bell and Dominici, 2008 or Smith et al., 2009 are considered, the ozone mortality impact varies dramatically across the country. If the combined results from the APHENA study for all-cause mortality with PM controlled are considered, none of the 16 model combinations had a positive and statistically significant association in the United States.[8] With these other possible choices for the baseline, the futility of estimating a national mortality burden becomes apparent.

In addition, the existence of a substantial threshold for the first physiological effects in controlled studies is not consistent with EPA's assumption that the dramatically more severe effects suggested by some epidemiological studies have no threshold. Such an assumption is not consistent with either the general principles of toxicology or the specific findings of ozone toxicological studies.

[8] AIR comments, supra note 2, at pp. 25-26.