



March 26, 2014

Statement of Stewart Holm before CASAC regarding Long-term
Ozone Exposure and Mortality
American Wood Council

Thank you for the opportunity to speak today. I am Stewart Holm, Chief Scientist at the American Wood Council. I am here speaking to you today to provide highlights on why EPA's current 75 ppb ozone NAAQS provides sufficient protection of public health.

My comments today focus on three main areas: 1) EPA's conclusions in the 2013 Integrated Science Assessment (ISA) as it relates to the draft EPA Risk and Exposure Assessment, 2) data limitation issues, and 3) consideration of influencing factors in evaluation of the data set.

EPA concluded in 2006 that evidence for respiratory mortality from long-term ozone exposure was "suggestive" of causation, but in the 2013 Integrated Science Assessment (ISA), EPA concluded that the association with respiratory mortality was likely to be causal, whereas evidence for all-cause and cardiovascular mortality were deemed to be suggestive of causation (US EPA, 2006, 2013). As discussed in more detail in my written comments, the evidence for respiratory mortality is limited, not consistent across studies, and not consistent with evidence for total mortality.

Also, studies of long-term exposures and respiratory mortality are too limited to make a causal determination for respiratory mortality. Compared to studies of short-term ozone exposures and mortality, there are only a few studies that have evaluated mortality associated with long-term ozone exposures. Most studies that assessed cause-specific mortality have focused on cardiopulmonary mortality; I am only aware of three that have evaluated respiratory mortality on its own: Abbey *et al.* (1999), Jerrett *et al.* (2009), and Lipsett *et al.* (2011). Only the studies by Abbey *et al.* (1999) and Jerrett *et al.* (2009) were referenced in the ISA, and EPA appeared to base its likely causal conclusion for respiratory mortality only on Jerrett *et al.* (2009).

Jerrett *et al.* (2009) evaluated total, respiratory, and cardiovascular mortality risks associated with long-term ozone exposure in single- and two-pollutant models (with PM_{2.5}) in the American Cancer Society cohort. Jerrett *et al.* (2009) reported small increases in mortality, particularly respiratory mortality, but the results were inconsistent across the mortality endpoints evaluated. For example, Jerrett *et al.* (2009) did not observe an association between ozone exposure and all-cause mortality or other cause-specific mortality outcomes. Notably, in their two-pollutant models that controlled for PM_{2.5}, Jerrett *et al.* (2009) found statistically significant *decreased* risks between long-term ozone exposure and all-cause and cardiovascular-related mortality. These

findings raise questions regarding the respiratory mortality findings on which EPA relied in the REA to calculate long-term risks.

In addition, this study has several shortcomings in its design and implementation. For example, it did not fully consider meteorological factors, such as temperature, or lifestyle factors such as smoking history where follow up on initial status is necessary, which may be independently linked to mortality risk. There also are significant uncertainties associated with the use of a single ozone value, averaged over both space and time, to represent ozone exposures for the entire population within each metropolitan area when we know it is much more variable.

Finally, EPA acknowledged that environmental temperature and region of the country were significant modifiers of the reported ozone associations with respiratory mortality. Jerrett *et al.* (2009) reported a lack of statistically significant associations between ozone and mortality in the Northeast and Industrial Midwest, which had the most respiratory deaths, and in Southern California, which had the highest ozone concentrations. This lack of correlation suggests that something other than ozone is affecting mortality risks and further highlights a lack of clear support for ozone-induced respiratory mortality and the proposed causal classification.

The findings by Jerrett *et al.* (2009) were not supported by the other two epidemiology studies that specifically evaluated respiratory mortality and long-term ozone exposures. Abbey *et al.* (1999) conducted a study of mortality in the Seventh-day Adventists cohort in California. The authors reported that non-malignant respiratory, cardiopulmonary, and all-cause mortality were not significantly increased with long-term ozone exposures. Lipsett *et al.* (2011) investigated the association between ozone and mortality, including respiratory mortality, in a cohort of female teachers in California. For respiratory mortality, the authors reported no significant association with ozone.

Overall, EPA's conclusions in the 2013 ozone ISA regarding long-term ozone exposure and respiratory mortality including its "causal" classification are not supported by the limited available evidence or the lack of association with total mortality. Thus, respiratory mortality risks from long-term ozone exposure should not be evaluated and quantified in the REA.

References

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