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Testimony to the EPA Clean Air Science Advisory Committee (CASAC) Ozone Review Panel for the Reconsideration of the National Ambient Air Quality Standard
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Bruce Copley, Ph.D.
ExxonMobil Biomedical Sciences, Inc.

My name is Bruce Copley. I am an epidemiologist with ExxonMobil Biomedical Sciences, Inc. I would like to thank EPA and CASAC for the opportunity to address the Committee today. My remarks focus on whether or not there is adequate scientific support for the theory that current levels of ambient ozone cause acute cardiovascular mortality and morbidity. Based on data from the 2008 review, EPA concluded that the data support this causality conclusion. The most current studies on the health effects of ozone--excluded from recent deliberations on the ozone NAAQS discussion--clearly do not support this causality conclusion. I will summarize the findings from these studies and also address why EPA's acute mortality risk estimates are not accurate and, thus, do not justify lowering the current ozone standard.

First, new scientific evidence clearly demonstrates that in contrast to EPA's conclusion, the ozone acute mortality association is significantly confounded by exposure to other pollutants.

I note that this new scientific evidence is especially critical to answering EPA charge question number 6, which asks *"to what extent the effects observed in epidemiology studies should be attributed specifically to ozone alone?"*. In a Health Effects Institute (HEI) sponsored study, Katsouanni et al. (2009) reported that when PM_{10} was included in a two-pollutant models, the "all-cause mortality" for ozone in the U.S., using data from the National Morbidity, Mortality and Air Pollution Study (NMMAPS), was reduced by 68-88% depending on the age group considered. Also, the cardiovascular mortality attributed to ozone was reduced by 155% (Table 24).

Similarly, using data from NMMAPS, Smith and Switzer (2009) reported a 22-33% reduction in the ozone mortality coefficient when PM_{10} was included in a two-pollutant model. This study also indicated regional heterogeneity of effect that suggests that a derived single national risk

estimate is unreliable for standard-setting. Franklin and Schwartz (2008) reported that including sulfate PM in a two-pollutant model decreased the ozone mortality by 33% and that the ozone association was not statistically significant.

Second, a large body of new scientific information clearly suggests that, in contrast to EPA's conclusion, current levels of ozone do not cause cardiovascular morbidity.
This new scientific evidence is highly relevant to answering EPA charge question 9.
Question 9 asks CASAC to provide their perspective on the occurrence and public health importance of various ozone induced health effects, including cardiovascular morbidity and mortality at alternate ozone standards. Numerous new epidemiology studies report no association between current ambient ozone levels in the U.S. and cardiovascular hospital admissions. These studies include those focusing on cardiovascular morbidity alone (such as those reported by Szyskowicz (2008), Symons et al. (2006), Villeneuve et al. (2006), Wellenius et al. (2005), Tolbert et al. (2007), Zanobetti and Schwartz (2006), and Katsuyanni et al. (2009)), as well as those focusing on combined cardiovascular and respiratory causes (Tolbert et al 2007, Zanobetti and Schwartz, 2006).

As noted by the HEI Review Committee in their commentary on the findings from the study by Katsouyanni, this marked lack of an association for cardiovascular morbidity provides little coherence for the ozone-cardiovascular mortality association. The Katsouyanni study also demonstrated that the risk estimates used for standard-setting are particularly sensitive to the specification of statistical models.

In both the Risk and Exposure Assessment (REA) and the Regulatory Impact Assessment (RIA) prepared by EPA for the ozone NAAQS reconsideration, EPA adopted the theory that the ozone-mortality association was causal and also estimated the risks of ozone exposure and the benefits of reducing this pollutant, based on single-pollutant model results. The new scientific data cast doubt on these assumptions and approaches. In order to facilitate the most scientifically sound decision on the ozone NAAQS, a thorough assessment of all relevant scientific data, including new scientific information, is required. Again, we would like to thank EPA and CASAC for the opportunity to provide these comments.

References

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