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appearing on behalf of the American Petroleum Institute

**regarding the PM_{2.5} epidemiology evaluation in the
Integrated Science Assessment for Particulate Matter First External Review Draft (December 2008)**

Docket No. EPA-HQ-ORD-2007-0517

April 1, 2009

Thank you for the opportunity to speak and provide written comments. I am Dr. Julie Goodman, an epidemiologist and board-certified toxicologist at Gradient Corporation, an environmental consulting firm in Cambridge, Massachusetts.

In the 2004 Air Quality Criteria Document, US EPA concluded that exposure to ambient PM caused or was associated with a wide variety of health effects, and that no threshold had been identified below which these health effects occur. Based on a review of several recent epidemiology studies, I conclude that the ISA has not adequately demonstrated that studies published since then: (1) demonstrate that PM_{2.5} causes additional health effects not identified in the last review; (2) provide reduced uncertainties or stronger evidence for the previously identified effects; (3) provide evidence that risk estimates for the previously identified effects have increased since the last review; or (4) provide further information on the possibility that these effects occur at lower levels than previously identified.

US EPA's framework for causality described in Chapter 1 of the ISA places too much weight on ecological epidemiology studies and too little weight on their uncertainties, including confounders, measurement error, exposure misclassification, and model uncertainty. In addition, the ISA does not adequately consider weak associations, the lack of consistency of observed associations, the lack of specificity of exposures and health effects, and the preponderance of non-statistically significant findings.

Chapter 2 of the ISA briefly reviews studies that are discussed in more detail in Chapters 6 and 7, which focus on studies of short- and long-term PM_{2.5} exposures, respectively. These three chapters do not accurately portray the epidemiological data as a whole and present a biased portrayal of the weight of evidence. For example, the majority of studies reported either null or weakly positive findings. In other cases, weakly positive findings became non-significant when adjusted for confounders. Several studies

did not have information on co-pollutants or other factors that may have been associated with exposure and/or outcome, such that reported associations were likely biased away from the null. Exposure misclassification (which could have biased results in either direction) was perhaps the biggest shortcoming of many of the studies considered by US EPA, as almost all studies used measurements from central monitors as surrogates for personal exposures, and other studies did not actually measure exposures at all. For some studies, risk estimates across cities were heterogeneous, yet they were pooled for an overall risk estimate, which is scientifically inappropriate. In the short-term exposure studies, risk estimates were often sensitive to the various lag times investigated. Many long-term exposure studies used the Cox proportional hazard model, which likely led to biased estimates because model assumptions were not always met. More importantly, most long-term exposure studies had few exposures below 15 $\mu\text{g}/\text{m}^3$, so they were not informative regarding risks below the current NAAQS.

Finally, studies relied on to assess concentration-response relationships (discussed in Chapters 2 and 8) are not sufficient for concluding a linear, no-threshold model. The evaluation of the studies in the ISA does not fully consider the limitations of these studies and does not assess other studies that do not support a linear model. Thus, the analysis presented in this 1st Draft should be discarded and replaced with a more comprehensive evaluation of the studies conducted for potential use in the 2nd draft. Proper inclusion and analysis of all the appropriate data may not allow EPA to conclude a linear, no-threshold model is the most appropriate for assessing either PM morbidity or mortality. Without defensible validation of such a model, no conclusions can be drawn as to whether there is a continued impact of morbidity and mortality as hypothetical future ambient PM concentrations approach zero.

In conclusion, the ISA does not provide sufficient scientific evidence to suggest $\text{PM}_{2.5}$ causes health effects at levels below the current NAAQS.