Comments on the *Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment*

Julie E. Goodman, Ph.D., DABT
Gradient Corporation, Cambridge, MA

on behalf of the American Petroleum Institute

May 7, 2009

Chapter 3 of the *Particulate Matter National Ambient Air Quality Standards: Scope and Methods Plan for Health Risk and Exposure Assessment* (PM REA Scope and Plan) describes the planned health risk assessment. The risk assessment will focus primarily on fine particles ($PM_{2.5}$), but also on thoracic coarse particles ($PM_{10-2.5}$), and will generate a core set of risk estimates for several combinations of urban study areas and air quality scenarios. Single and multi-factor sensitivity analyses will be conducted to generate alternative sets of risk estimates for a subset of these scenarios.

When conducting the risk assessment, it is critical that US EPA base their core set of risk estimates on plausible exposure scenarios and robust epidemiological data. That is, the core set of risk estimates should not be based on an overly conservative set of assumptions, but on the factors that are most likely indicative of the true risk of individuals in the population. The uncertainty and sensitivity analyses should then be used to address more conservative and worst-case scenarios.

To begin, US EPA must choose the most biologically plausible concentration-response curves. In the case of $PM_{2.5}$, the first draft PM Integrated Science Assessment (ISA) states that a threshold is not apparent based on ecological epidemiology studies; however, there is a scientifically valid reason, based on the mode of action, that the health effects of interest are actually of a threshold nature. One must conclude that the concentration-response function in epidemiology studies relates not to actual linearity in humans but rather to an artifactual flattening that is most likely attributable to population heterogeneity and exposure misclassification, two factors that are evident throughout the $PM_{2.5}$ epidemiology literature. Because of this, a threshold model should be used for the core set of risk estimates. Other concentration-response models can be assessed in the sensitivity and uncertainty analyses.

In addition, only health endpoints for which $PM_{2.5}$ is deemed "causal" by US EPA's definition should be considered in the core risk assessment. In general, US EPA's causality framework understates

uncertainties – including confounders, measurement error, exposure misclassification, and model uncertainty – and does not give them consistent or sufficient weight in the evaluation of epidemiology studies. Because there is already a certain degree of uncertainty in a "likely to be a casual relationship," and even more uncertainty in a "suggestive of a causal relationship," it is inappropriate to use endpoints with these classifications in the risk assessment. For example, the first draft PM ISA states that epidemiology studies do not consistently report associations between PM exposure and reproductive or developmental outcomes, yet still classifies the data as "suggestive." Unless the data for reproductive and developmental effects become consistent and robust, these health endpoints should not be considered in the risk assessment.

Finally, US EPA should not conduct a quantitative risk assessment of thoracic coarse particles. The first draft PM ISA does not identify any health effect endpoint categories as having sufficient support for a "causal" or "likely to be" causal classification and, again, this "suggestive" evidence is simply not robust enough to contribute to a risk assessment.

In closing, the core risk estimates in the PM REA should be based on threshold models and "causal" endpoints. Other less plausible scenarios should be addressed in the sensitivity and uncertainty analyses.