WHAT TYPES OF PM2.5 (OR ASSOCIATED POLLUTANTS) MAY BE MORE (OR LESS) LIKELY TO CAUSE PREMATURE MORTALITY? AN UPDATE, INCLUDING NEW PEER REVIEWED LITERATURE

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Abstract

Should all types of PM2.5 be seen as equally harmful? Early (~1990-1995) statistical associations between PM2.5 and/or PM10 mass levels and premature mortality, in the absence of information about the toxicity of specific PM types, suggested this possibility. But the Clean Air Scientific Advisory Committee (1996), the National Research Council of the National Academy of Science (1998), and the New England Journal of Medicine (2000) all have called for EPA to use toxicology to help identify the most potentially harmful components of the hundreds of compounds in the PM mix, in order that the PM constituents which cause the most harm be preferentially regulated.

New, more sophisticated statistical studies are now becoming available, some funded by EPA, and some from Europe. Much toxicology has also been done, although little new toxicology has been done on the most common constituents of ambient PM2.5. Much of the new work, as well as some toxicology, suggests that some constituent(s) of vehicular emissions may have more importance than previously thought, and that other emissions may have less importance than previously thought. New insights from these research results may also help understand difficult-to-resolve findings from earlier studies, as well.

The U.S. Department of Energy has proposed that EPA initiate a "structured regulatory toxicology program" designed at the outset to help understand which PM2.5 constituents may be most harmful, or of negligible harm, at ambient levels. Such a program would compare many different PM2.5 components or mixtures in rodent inhalation models, at the same specific and rising multiples of ambient concentrations, against specific health endpoints. Those PM2.5 components which cause significant adverse effects at low multiples of ambient concentrations would be seen as more likely to cause similar adverse effects in humans, than those PM2.5 components which fail to cause adverse effects until high multiples of ambient concentrations.