

**Soile Tapio**  
Helmholtz Zentrum München  
Institute of Radiation Biology  
Oberschleissheim, Germany

**Richard Wakeford**  
Dalton Nuclear Institute  
University of Manchester  
Manchester, United Kingdom

**Lydia Zablotska**  
Department of Epidemiology  
and Biostatistics  
University of California San Francisco  
San Francisco, California

**Steven E. Lipshultz**  
Department of Pediatrics  
Leonard M. Miller School of Medicine  
University of Miami  
Miami, Florida

on behalf of all authors of the paper

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## Is Ambient PM<sub>2.5</sub> Sulfate Harmful?

<http://dx.doi.org/10.1289/ehp.1205873>

Lepeule et al. (2012) associated reduced PM<sub>2.5</sub> (particulate matter  $\leq 2.5 \mu\text{m}$  in aerodynamic diameter) with decreased mortality over almost four decades. Because the sulfate/PM<sub>2.5</sub> ratio dropped among six localities but the PM<sub>2.5</sub> mortality coefficient did

not “substantially” increase, the authors concluded that sulfate must be “about as toxic” as average PM<sub>2.5</sub>. In a two-pollutant world, perhaps.

When a single source emits several PM<sub>2.5</sub> species, and a specific species is emitted from several sources, chemical-specific associations might not reflect inherent toxicity but rather status as a marker of harmful coemissions (Grahame and Hidy 2007; Mostofsky et al. 2012). Furthermore, because total PM<sub>2.5</sub> is often associated with adverse health outcomes, association of a constituent representing a large portion of total mass (e.g., sulfate) may occur unrelated to any inherent toxicity (Mostofsky et al. 2012).

Toxicological studies have not indicated adverse health effects from sulfate per se (Schlesinger and Cassee 2003). However, reducing a unit of black carbon (BC) increased life expectancy 4–9 times more than reducing a unit of PM<sub>2.5</sub> (Janssen et al. 2011). Evidence from both toxicological and human panel studies with accurate subject exposure consistently has linked BC with adverse cardiovascular health outcomes (Grahame and Schlesinger 2010). Metals and other emissions from older steel plants are particularly toxic (Dye et al. 2001).

Substantial reductions in BC and polycyclic aromatic hydrocarbons from diesel engines and coke ovens, various metals from steel plants, and nickel and vanadium from residual oil have occurred over the time frame examined by Lepeule et al. (2012). Sulfur was coemitted by all of these sources. Because less abundant but more toxic PM<sub>2.5</sub> species were also substantially reduced over this period, changes in the sulfate/PM<sub>2.5</sub> ratio as applied to mortality might reflect toxicity of coemissions, not of sulfate. Is sulfate inherently toxic or merely a coemission of harmful PM species?

Researchers must use models that include many relevant PM<sub>2.5</sub> species to successfully parse adverse health effects of each (Grahame and Hidy 2007). BC (and to a lesser extent nickel) remains consistently associated with adverse health outcomes when increasingly sophisticated models—all including 18 PM<sub>2.5</sub> species—are used; however, sulfate associations become negative and insignificant (Mostofsky et al. 2012).

Further, subject exposure measures must be reasonably accurate; associations found with accurate exposure may not be found when central monitor concentrations are proxies for exposure across a metropolitan area (Suh and Zanobetti 2010).

Human panel studies can examine effects of PM<sub>2.5</sub> species with more accurate subject exposure. Schwartz et al. (2005) found consistent associations for measures of heart rate variability with BC, but fewer associations

for PM<sub>2.5</sub>. In that study, the authors used an algorithm separating BC from PM<sub>2.5</sub> and found no associations with the PM<sub>2.5</sub> remainder (termed “secondary PM<sub>2.5</sub>” by the authors), which would include both secondary sulfate and its reaction products.

Any conclusions regarding sulfate toxicity are premature until consistent results from advanced models (Mostofsky et al. 2012), which are able to examine many chemical species and incorporate good exposure measures, are available and are congruent with toxicology.

*The authors declare they have no actual or potential competing financial interests.*

**Thomas Grahame**  
U.S. Department of Energy  
Washington, DC  
E-mail: [thomas.grahame@hq.doe.gov](mailto:thomas.grahame@hq.doe.gov)

**Richard Schlesinger**  
Pace University  
New York, New York

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## Is Ambient PM<sub>2.5</sub> Sulfate Harmful? Schwartz and Lepeule Respond

<http://dx.doi.org/10.1289/ehp.1205873R>

Grahame and Schlesinger make two arguments against the conclusions of our paper (Lepeule et al. 2012). Regarding their first point, we argued that if sulfates are non-toxic—and the fraction of particles that are