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## Secondary Sulfate Effects?

doi:10.1289/ehp.10293

Maynard et al. (2007) related mortality with ambient particulate black carbon (BC) and sulfate (SO<sub>4</sub>). They also associated SO<sub>4</sub> in Boston, Massachusetts, with “secondary,” distant “coal combustion.” Their correlation of 0.44 between BC and SO<sub>4</sub>, however, suggests the importance of local source(s) of SO<sub>4</sub>, including vehicle exhaust (especially diesel).

Centrally monitored BC poorly characterizes exposure to traffic emissions and results in exposure misestimation, causing distorted or undetected health associations (Ito et al. 2004). Maynard et al. (2007) address this deficiency by geocoding BC estimates to a decedent’s residence; a central monitor characterizes SO<sub>4</sub> exposure. In their single-pollutant models, BC and SO<sub>4</sub> are associated with daily mortality, but in multipollutant models, only BC retains significance.

The association of mortality with SO<sub>4</sub> is inconsistent. In a review of toxicologic studies, Schlesinger and Cassee (2003) suggested that SO<sub>4</sub> is benign. In an *in vivo* study of PM<sub>2.5</sub> (particulate matter ≤ 2.5 μm in aerodynamic diameter) components, Seagrave et al. (2006) found that lung toxicity and inflammation correlated with vehicular but not secondary particles, including SO<sub>4</sub>. However, vehicular emissions are consistently associated with cardiac or other end points (Grahame and Schlesinger 2007). How does secondary SO<sub>4</sub> cause mortality, if harmful biological mechanisms have not been found? Or could the mortality association with SO<sub>4</sub> of Maynard et al. (2007) be linked with other local PM sources?

Maynard et al. (2007) cited mortality associations for coal and traffic tracers reported by Laden et al. (2000), but other study findings differ. In a reanalysis, Schwartz (2003) found that their traffic variable remained significant, but their regional “coal” variable became insignificant. We (Grahame and Hidy 2004) noted that among the six cities described by Laden et al. (2000), Boston had the lowest levels of the “coal tracer” (selenium); only Boston had a significant association with an apparent coal source. Similarly, only in Boston was an association with SO<sub>4</sub> significant. Finding that local residual oil sources emitted over half the Se and SO<sub>4</sub> in Boston, these authors (Grahame and Hidy 2004) concluded from toxicology that these results represented effects of residual oil emissions.

SO<sub>4</sub> is elevated near major roadways. Reponen et al. (2003) showed an SO<sub>4</sub> gradient that declined based on distance from a midwestern freeway; Riediker et al. (2004) found that among highway-related factors, only the “speed changing” factor, which included emissions from accelerating diesel engines, showed elevated SO<sub>4</sub>. These findings are relevant to the monitoring conditions of Maynard et al. (2007). Other Boston studies show commingling between SO<sub>4</sub> and vehicular indicators. Clarke et al. (2000), for example, reported that loadings of BC are as high in an SO<sub>4</sub> indicator as in the traffic indicator. Because coal plants emit virtually no BC (Edgerton E, Mueller P, Monroe L, Jansen J, Waid C, unpublished data), high BC in urban SO<sub>4</sub> indicators reflects both SO<sub>4</sub> and BC from diesels in an SO<sub>4</sub> factor, derived from measurements near a thoroughfare. The SO<sub>4</sub>–BC correlation described by Maynard et al. (2007) thus suggests that SO<sub>4</sub> associations reflect statistical comingling of vehicular and coal emissions.

Association of harm from traffic emissions, but not SO<sub>4</sub> (or secondary aerosols generally), has been found in panel and epidemiology studies that *a*) precisely measure exposure to local vehicular emissions, and *b*) test effects of secondary PM<sub>2.5</sub>–SO<sub>4</sub> compared with local emissions. Health end points in such studies include long-term mortality, heart rate variability reduction, ST-segment depression, cardiac effects, vasoconstriction, increased blood pressure, or morbidity [reviewed by Grahame and Schlesinger (2007)]. Kodavanti et al. (2005) found no effects only with the highest SO<sub>4</sub> concentrations.

We suggest that findings of harmful exposure to secondary SO<sub>4</sub> per se are tenuous until physiologic mechanisms are identified that support toxicity near ambient concentrations.

*T.G. declares no competing financial interests. G.M.H. has research grants from EPRI (for preparation of a NARSTO multipollutant air quality management assessment) and the Southern Company Services Inc. (for data analysis of atmospheric chemistry measurements related to the southeastern aerosol research and characterization experiment, and supporting preparation of the NARSTO multipollutant air quality management assessment).*

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## REFERENCES

- Clarke RW, Coull B, Reinsch U, Catalano P, Killingsworth C, Koutrakis P, et al. 2000. Inhaled concentrated ambient particles are associated with hematologic and bronchoalveolar lavage changes in canines. *Environ Health Perspect* 108:1179–1187.
- Grahame TJ, Hidy GM. 2004. Using factor analysis to attribute health impacts to particulate pollution sources. *Inhal Toxicol* 16(suppl 1):143–152.
- Grahame TJ, Hidy GM. 2007. Pinnacles and pitfalls for source apportionment of potential health effects from airborne particle exposures. *Inhal Toxicol* 19:727–744.
- Grahame TJ, Schlesinger RB. 2007. Health effects of airborne particulate matter: do we know enough to consider regulating specific particle types or sources? *Inhal Toxicol* 19:457–481.
- Ito K, Xue N, Thurston G. 2004. Spatial variation of PM<sub>2.5</sub> chemical species and source-apportioned mass concentrations in New York City. *Atmos Environ* 38:5269–5282.
- Kodavanti UP, Schladweiler MC, Ledbetter AD, McGee JK, Walsh L, Gilmour PS, et al. 2005. Consistent pulmonary and systemic responses from inhalation of fine concentrated ambient particles: roles of rat strains used and physicochemical properties. *Environ Health Perspect* 113:1561–1568.
- Laden F, Neas L, Dockery D, Schwartz J. 2000. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environ Health Perspect* 108:941–947.
- Maynard D, Coull BA, Gryparis A, Schwartz J. 2007. Mortality risk associated with short-term exposure to traffic particles and sulfates. *Environ Health Perspect* 115:751–755.
- Reponen T, Grinshpun S, Trakumas S, Martuzevicius D, Wang Z-M, LeMasters G, et al. 2003. Concentration gradient patterns of aerosol particles near interstate highways in the greater Cincinnati airshed. *J Environ Monit* 5:557–562.
- Riediker M, Devlin R, Griggs T, Herbst M, Bromberg P, Williams R, et al. 2004. Cardiovascular effects in patrol officers are associated with fine particulate matter from brake wear and engine emissions. *Part Fibre Toxicol* 1:2; doi:10.1186/1743-8977-1-2 [Online 9 December 2004].
- Schlesinger RB, Cassee F. 2003. Atmospheric secondary inorganic particulate matter: the toxicological perspective as a basis for health effects risk assessment. *Inhal Toxicol* 15:197–235.
- Schwartz J. 2003. Daily deaths associated with air pollution in six U.S. cities and short-term mortality displacement in Boston. In: *Revised Analyses of Time-Series Studies of Air Pollution and Health*. Boston:Health Effects Institute, 219–226.
- Seagrave JC, McDonald JD, Bedrick E, Edgerton ES, Gigliotti AP, Jansen JJ, et al. 2006. Lung toxicity of ambient particulate matter from southeastern U.S. sites with different contributing sources: relationships between composition and effects. *Environ Health Perspect* 114:1387–1393.

## Secondary Sulfate Effects: Schwartz Responds

doi:10.1289/ehp.10293

I am not convinced by the argument of Grahame and Hidy in their letter. In fact, I find it unsupportable by modern atmospheric science and statistics. First, they argue that much of the sulfates in Boston, Massachusetts, derive locally, rather than from long-range transport, and that diesel exhaust is a probable major source. Sulfates, a major source of fine particles in Boston and other East Coast communities, derive primarily from coal-burning power plants, and this clearly may be uncomfortable for the electric utility industry and the Department of Energy; however, it is

equally clear that there is little doubt about it among atmospheric scientists. For example, Salmon et al. (1997) reported that sulfate concentrations at a monitoring station in a limited-access conservation area in a rural part of western Massachusetts were almost identical to the sulfate concentrations in Boston, whereas the elemental carbon concentrations, which are markers of traffic, particularly the diesel exhaust Grahame and Hidy postulate as the source of sulfates in Boston, were quite different. The sulfate levels in Boston cannot simultaneously be driven by local traffic and still be identical to those in a distant rural area.

This similarity in sulfates persists to later periods. The U.S. Environmental Protection Agency's Technology Transfer

**Table 1.** Sulfate concentrations by year in Springfield and Boston, Massachusetts.

Year/city	No. of days	Mean ( $\mu\text{g}/\text{m}^3$ )
2001		
Boston	117	3.08
Springfield	117	3.04
2002		
Boston	122	2.98
Springfield	122	2.51
2003		
Boston	124	3.03
Springfield	124	2.00
2004		
Boston	125	3.08
Springfield	125	3.01
2005		
Boston	106	3.19
Springfield	106	3.17

Network (U.S. EPA 2007) provides data on sulfate levels measured on the same days in Springfield, Massachusetts, at the western end of the state, and Boston, 95 miles to the east (Table 1). Apparently, truck traffic is almost identical in these cities.

Hourly sulfate monitors are operated in St. Louis, Missouri, Boston, and elsewhere. Hourly concentrations peak in the afternoon after the boundary layer has broken up, and transported sulfates can mix down to ground level. In contrast, concentrations of BC, a marker of diesel exhaust, peak near 0600 hours.

Regarding Grahame and Hidy's second point, in our study (Maynard et al. 2007) we reported that the sulfate effect was no longer significant when we controlled for BC. However, because there were far fewer days with sulfate and because the coefficient was similar to the coefficient described by Laden et al. (2000), we stated that our finding strengthened the evidence for an association with mortality. We see nothing in Grahame and Hidy's letter to lead us to change that view. Yes, the association is not statistically significant. But judgments about associations are made by combining evidence across studies, not on single studies. And in a meta-analysis, adding a study with a positive coefficient of similar size to previous studies increases the significance of the association when the cities are all combined. Thus our statement that this result adds to the support for a sulfate association

remains the most reasonable interpretation. Finally, Grahame and Hidy make much of the change in effect size for sulfate when controlling for BC. Although this does indicate that there is some correlation of the measures, this correlation is to be expected, because common weather patterns drive some of the day-to-day variation in most pollutants. It does not imply that sulfate in Boston comes to a substantial extent from diesel engines.

*The author declares he has no competing financial interests.*

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#### REFERENCES

- Laden F, Neas L, Dockery D, Schwartz J. 2000. Association of fine particulate matter from different sources with daily mortality in six U.S. cities. *Environ Health Perspect* 108:941–947.
- Maynard D, Coull BA, Gryparis A., Schwartz J. 2007. Mortality risk associated with short-term exposure to traffic particles and sulfates. *Environ Health Perspect* 115:751–755.
- Salmon LG, Cass GR, Pederen DU, Durant JL, Gibb R, Lunts A, et al. Determination of Fine Particle Concentration and Chemical Composition in the Northeastern United States, 1995. Boston:Northeastern States for Consolidated Air Use Management.
- U.S. EPA (U.S. Environmental Protection Agency). 2007. Air Quality System Technology Transfer Network. Available: <http://www.epa.gov/ttn/airs/airsaqs/> [accessed 5 October 2007].