Lipfert *et al.* (*2*) of 1980 sulfate mortality data has already considered the available Inhalable Particle Network dichotomous sulfate dataset and a computer model simulated sulfate dataset. Both are free of sulfate artifact, but still give sulfate coefficients that have elasticities not significantly different from those we reported.

Similarly, Lipfert and Morris doubt the validity of our results "as smoking, diet, water hardness, and migration were not accounted for. . . ." However, these factors are only of concern if they confound the analysis by covarying strongly with sulfate measurements (*3*). Again, the cross-sectional analysis by Lipfert *et al.* (*2*) has already shown that smoking and water hardness are not significantly correlated with sulfates. More important, after these factors (and migration) were incorporated into their cross-sectional mortality regressions (which we believe are often overspecified), the 1980 sulfate coefficient was still significant and not statistically different from a sulfate coefficient produced by a model without such factors. Moreover, a further analysis of the 1980 dataset, which considered more than 300 Standard Metropolitan Statistical Areas (SMSAs) and more complete model specifications (for example, smoking, migration, racial mix, health care, industrial mix, and climate), also confirms the robustness of the sulfate coefficient that we reported in our original research (*4*).

Lipfert *et al.* have also considered the other pollutants they mention, concluding that automotive related pollutants (ozone, lead, and carbon monoxide) did not yield significant positive coefficients and could be eliminated from consideration (*2*). In the end, after addressing the data and model specification factors they now raise in their letter, Lipfert *et al.* reported a sulfate mortality elasticity ranging from 2.8 to 13%, which coincides well with our originally estimated range of 4 to 9%. Thus, the 1980 sulfate mortality coefficient we reported in our work has actually proven quite insensitive to the factors raised by Lipfert and Morris.

Lipfert and Morris also express concerns about potential biases in "ecological" studies. We tested our results for potential biases by conducting several sensitivity analyses, including region-specific mortality data regressions on spatially averaged mortality and pollution data (*1*). None of these analyses suggested the presence of significant confounding of variables. Lipfert and Morris raise the possibility that the weak relationship found between total suspended particulate matter (TSP) and human mortality can be accounted for by the fact that central monitor measurements are less representative of regional TSP than they are of regional sulfates. That is why we also considered multiple site SMSA averages of pollution measurements during our sensitivity analyses. We found no appreciable changes (*1*). The fine particle component is the portion that can most readily enter the thorax and is therefore thought to be the most pernicious part of TSP. On this basis, the U.S. Environmental Protection Agency (EPA) changed from a TSP to an inhalable particle standard in 1987 (*5*). Moreover, of the fine particles, the acidic aerosol (of which sulfates are a major component) is thought to be of such special health concern that the EPA Clean Air Scientific Advisory Committee has recommended that the agency consider whether acidic aerosols should be added to the agency's list of criteria pollutants.

In summary, considering all the subsequent analyses and biological plausibility, it is more likely than ever that our results are a product of a valid relationship between sulfate air pollution and human health effects.

George D. Thurston
New York University Institute of Environmental Medicine, Long Meadow Road, Tuxedo, NY 10987
Haluk Özkaynak
Department of Environmental Health, Harvard School of Public Health, 655 Huntington Avenue, Boston, MA 02115

REFERENCES

1. H. Özkaynak and G. D. Thurston, *Risk Anal.* **7**, 449 (1987).
2. F. W. Lipfert, R. G. Malone, M. L. Daum, N. R. Mendell, C-C. Yang, "A statistical study of the macroepidemiology of air pollution and total mortality" (Report 52122, Brookhaven National Laboratory for the U.S. Department of Energy, Upton, NY, 1988).
3. J. S. Evans, T. Tosteson, P. L. Kinney, *Environ. Int.* **10**, 55 (1984).
4. J. S. Evans, H. Özkaynak, B. Burbank, in preparation.
5. Particulate matter with an aerodynamic diameter less than or equal to a nominal 10 micrometers (PM_{10}); U.S. Environmental Protection Agency, *Fed. Reg.* **52**, 24639 (1 July 1987).
6. "An acid aerosol issue paper" (EPA/600/8-88/005F, Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Washington, DC, 1989).

Erratum: The last sentence of the abstract of the report "Induction of type I diabetes by Kilham's rat virus in diabetes-resistant BB/Wor rats" by D. L. Guberski *et al.* (15 Nov., p. 1010) should have read, "This model of diabetes may provide insight regarding the interaction of viruses and autoimmune disease."

Erratum: Table 3 in the report "Latitudinal and longitudinal oscillations of cloud features on Neptune" by Lawrence A. Sromovsky (1 Nov., p. 684) contained errors. In column 4, the two values for inferred shear should have been −0.8 and −13.8 m/(s-deg), respectively. The corresponding text in column 1 on page 686 should have read, "the advection model is quantitatively upheld for the DS2 motions, but not for the short-period component of the GDS motions. They both agree on phase lags, although the GDS is not in good quantitative agreement on amplitudes. Relative to a smooth shear profile obtained from a simple polynomial fit to the observed wind speeds as a function of latitude (*11*), the shear derived from the advective model amplitude ratio is almost the same for DS2, but a ninth as large for the GDS."