Table 1. Regional coefficients for Narragansett, Rhode Island aerosol of 3 through 8 August 1979.

Signature	Type of weighting		
	Subjective*	Sample variance†	Effective variance‡
New England	0.21 ± 0.30	0.16 ± 0.21	
Boston	0.17 ± 0.04	0.17 ± 0.05	0.24 ± 0.05
New York City	0.06 ± 0.09	0.07 ± 0.04	
Washington	0.00 ± 0.10	0.00 ± 0.08	
Interior	0.29 ± 0.10	0.28 ± 0.12	0.41 ± 0.09

*Weights according to our original article. Uncertainties directly from SAS output. †Uncertainties calculated according to (*8*), with variance of sample only. ‡Insignificant sources eliminated according to procedures of (*6*). Uncertainties calculated according to (*8*), with variances in both sample and signatures.

volves either the variance of the receptor sample or the variances of sample and signatures combined with the source strengths (*8*), but not the variances of the signatures alone. This latter weighting is without precedent in the literature. Thurston and Laird thus do not follow the state-of-the-art procedures outlined in a paper of which Thurston was a co-author (*7*). Our Table 1 shows three apportionments of the same sample: our original version based on subjective weightings, one with the proper inverse-variance weightings (from the receptor sample only), and one based on effective-variance weighting (*7, 8*). The stability and meaningfulness of the coefficients are evident in three ways: the results from inverse-variance weighting agree closely with those from subjective weighting, the sense of the results from the more rigorous effective-variance treatment agrees with both our other answers, and the 85 percent confidence intervals around the Boston and Interior coefficients from all three of our treatments exclude zero. We do not see how Thurston and Laird can make such strong assertions about lack of meaning on the basis of the 95 percent confidence level when the opposite answer is available at the 85 percent level. To us, it seems clear that the midwestern coefficient of this sample is nearly double the northeastern coefficient, no matter how they are calculated, and that both are known accurately.

The sweeping rejection by Thurston and Laird of apportionment data based on confidence intervals alone is a classic example of the dangers of depending upon a single statistical argument and its subjective interpretation at the expense of all other knowledge. For a complex system such as the atmosphere and its aerosol, this approach is particularly risky. To be sure, uncertainties of individual regional coefficients are large—we have estimated some to be as great as 30 percent even for the most significant coefficients. But this hardly justifies discarding the entire technique. Abundant data of other types are available with which to counter the conclusion of Thurston and Laird, and they emphasize the reliability of regional coefficients. For example, time-series of regional coefficients correlate tightly with large-scale meteorology (*1*), long-term regional apportionments of sulfate for the Northeast agree well with those derived from transport models and directional studies (*1*), and the validity of our midwestern coefficients has been verified in the 1983 Cross-Appalachian Tracer Experiment (*9*), during which every pulse of perfluorocarbon tracer gas released from Dayton, Ohio, and later sensed near our two sampling sites in New England was preceded by a pronounced maximum of midwestern aerosol (*2, 10*). When all the evidence is considered, it is clear that our tracer system is functioning properly and that regional coefficents are highly meaningful.

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Smoking and Longevity

In response to the communication by J. E. Enstrom (Letters, 31 Aug., p. 878), we would like to make the following comments. We did not question that the survey by Enstrom and Godley (*1, 2*) was representative of the U.S. population. We did question its coding of lifetime nonsmokers. Enstrom asserts his confidence in his procedures and notes that our methods yielded higher smoker-nonsmoker longevity differences than Enstrom and Godley reported (*2*); however, both results are within appropriate limits for such studies.

We draw different conclusions than Enstrom does from the literature he cites. For example, the authors of the three-state Amish study (*3*) reported very similar nonaccidental death rates for Amish men and women age 40 and above, as have the authors of other studies of nonsmoking populations (*4*). These studies show neglible differences in life expectancy between nonsmoking men and women. The Alameda County data of Wingard (*5*) and of Enstrom (*6*) are not directly comparable with our data (*7*) for two reasons: (i) their data is for individuals age 30 to 69, and ours is for individuals age 30 to 105; (ii) classifications that merge continuous and ex-smokers can not be equated with classifications of continuous smokers only. Wingard (*5*) pointed out that her merged classification of continuous and ex-smokers created certain anomalies in the estimates of male-female mortality risk. Our conclusion from the evidence is that smoking explains at least half and perhaps 80 to 90 percent of the male-female mortality difference after age 30.

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Erratum: In the listing of recipients of the National Medal of Science (News and Comment, 8 Mar., p. 1183), the affiliation of Helmut E. Landsberg was incorrect. He is emeritus professor at the University of Maryland.