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Particulate Air Pollution and Daily Mortality in Steubenville, Ohio

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Particulate air pollution has been associated with daily mortality in London, England, both in the smog episodes of the 1950s and at the lower pollution levels of the late 1960s and early 1970s. Replicating these findings in the United States has been difficult, because particulates are usually sampled every sixth day. Replication, particularly with a gravimetric measure of particulates, is important in assessing the causality of the relation. Daily measurements of total suspended particulates by high volume gravimetric sampler are available for the Steubenville, Ohio, metropolitan area. These were matched to daily mortality counts from the detail mortality tapes of the National Center for Health Statistics. Deaths of residents which occurred outside the Steubenville Standard Metropolitan Statistical Area were excluded. Because of the much smaller population, the average total number of deaths per day in the Steubenville Standard Metropolitan Statistical Area over the 11-year period 1974–1984 was about 1% of the deaths in a typical London winter. Despite this reduced statistical power, total suspended particulate count was significantly associated with increased daily mortality in Poisson regression analyses controlling for season and temperature. An increase in particulates of 100 $\mu g/m^3$ was associated with a 4% increase in mortality on the succeeding day. Associations with sulfur dioxide were not significant after adjustment for particulates. The relation appeared to continue at levels well below the current National Ambient Air Quality Standard. *Am J Epidemiol* 1992;135:12–19.

air pollutants, environmental; air pollution; mortality

Editor's note: *For a discussion of this paper and for the authors' response, see pages 20 and 23, respectively.*

Extremely high levels of air pollution were associated with considerable excess mortality in episodes in Donora, Pennsylvania, in 1948 (1), London, England, in 1952 (2), and the Meuse Valley, Belgium, in 1930 (3). These air pollution episodes were characterized by high levels of particulates, sulfur dioxide, and moisture. High levels of aerosol acidity may also have been present.

More common levels of air pollution may also be associated with early mortality. Analyses of data from London (4–6) have shown an association with mortality across a broad range of pollution values, with no evidence

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of a threshold. The relation with particles was independent of sulfur dioxide, but not vice versa (6). Similar findings have been reported in New York City (7, 8). These studies have used Coefficient of Haze or airport visibility as a proxy for particulate level. The London data also used an optical measure (British Smoke shade) rather than a gravimetric measure of particulate concentration. This measure was nonlinearly related to total suspended particulates (9). These differences in measurement make comparisons with current monitored particulate levels difficult. They also make it difficult to replicate the finding of a stronger relation with particles than with sulfur dioxide, since different particle measures are used in each study.

Studies using direct gravimetric measurement of particulate concentration in the United States have been limited by the every-sixth-day sampling schedule for particulate monitors. However, particulates and sulfur dioxide have been measured daily in Steubenville, Ohio, by the North Ohio Valley Air Authority from 1974 onward. These data were combined with daily mortality counts in the Steubenville Standard Metropolitan Statistical Area in an attempt to replicate the results from London.

MATERIALS AND METHODS

Daily deaths in the Steubenville Standard Metropolitan Statistical Area were read from the annual detail mortality tapes of the National Center for Health Statistics for the years 1974–1984. Total population in the 1980 US Census was 163,099, including 26,400 in Steubenville and 91,564 in the remainder of Jefferson County, Ohio, plus 71,535 in Brooke and Hancock counties in West Virginia (10). Deaths due to accidents (*International Classification of Diseases*, Ninth Revision, codes ≥800) were excluded, as were all deaths of residents which occurred outside of the Standard Metropolitan Statistical Area. The mean number of deaths per day after the exclusions was approximately three, which did not allow further breakdown by specific cause. This is consistent with previous studies (4–8) in which total mortality has been associated with ambient air pollution.

Numbers of deaths per day were matched to daily measurements (8:00 a.m. to 8:00 a.m.) of total suspended particulates and sulfur dioxide from a monitor at the offices of the North Ohio Valley Air Authority in a former school in a residential neighborhood of Steubenville. The air quality record of this site is remarkably complete—99.9 percent data capture for daily total suspended particulate concentrations for the study period. Other monitoring sites in the region sampled on the every-sixth-day schedule. This station was routinely audited by the Ohio Environmental Protection Agency and participated in quality assurance checks by the federal Environmental Protection Agency. As one of the monitoring sites contributing to the Harvard Six Cities Study, this monitor was routinely audited by the Harvard University quality assurance staff, in addition to an independent contractor (11).

Sulfur dioxide was measured hourly by continuous monitor from 1974 onward. However, reports from the field indicated some initial difficulty with the new continuous monitoring system. To test this, we correlated the 24-hour average sulfur dioxide concentrations from the continuous monitor with the sulfur dioxide concentrations from the integrated gas bubbler sampler which was run until 1978. The correlation was 0.42 in 1974, rising to 0.90 in 1977. This suggests that the gas bubbler data were more reliable in earlier years. Therefore, the sulfur dioxide exposure variable was defined as the bubbler data until 1978 and the 24-hour average of the hourly continuous monitor concentrations afterward. As a sensitivity check, the 24-hour averages of the continuous monitor were also used for the entire time period. In 1987, the Environmental Protection Agency adopted a 10-μm cutoff aerodynamic diameter as the basis for the inhalable particulate ambient air quality standard (12). A series of co-located total suspended particulate and inhalable partic-

ulate samplers were operated in Steubenville in 1984 and 1985 and indicated a mean inhalable:total suspended particulate ratio of 0.51 (13). Daily data on mean 24-hour temperature and dew point temperature were obtained from the nearest National Oceanic and Atmospheric Administration weather station at the Pittsburgh, Pennsylvania, airport approximately 20 km to the east.

Mortality is a rare event which is classically modeled as a Poisson process. The number of events per day in a population this size does not permit a Gaussian approximation. Figure 1 shows the distribution of mortality counts in Steubenville during this period. The distribution is slightly skewed, and the empirical variance (3.08) is quite close to the mean (3.07), as in a Poisson distribution.

Mortality rates may fluctuate annually because of viral epidemics, population changes, and other factors unrelated to air pollution exposures. To model these annual fluctuations, we treated year as a random effect in the analyses. The use of random effects specification provides a parsimonious model for year-to-year variation. In addition, each year of data can be treated as a replicate study so that robust variance estimates can be calculated (see below). We tested for the possibility of a time trend separately. The impact of weather on mortality can be highly nonlinear, with only extreme conditions producing any effect. To test the correct specification of weather in our models, we examined nonparametrically smoothed plots of mortality versus temperature and dew point temperature. Once a model for weather was developed, daily mortality counts were regressed on weather, indicators for season, and the previous day's total suspended particulate count. Both current and prior day's pollution and weather were considered.

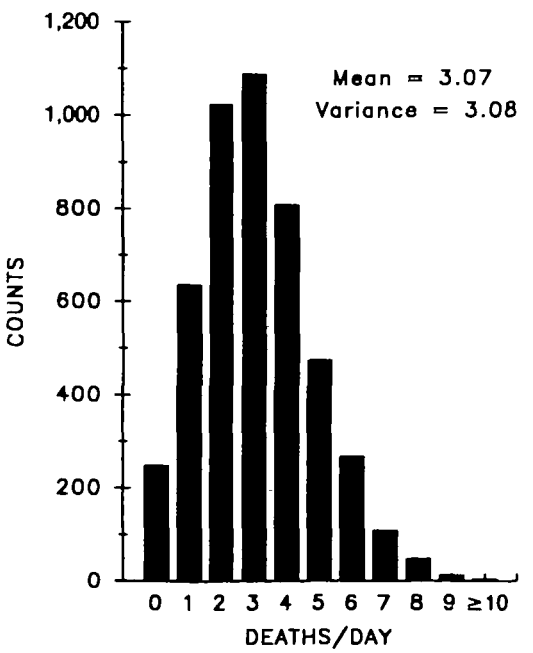

FIGURE 1. Distribution of counts of deaths per day in Steubenville, Ohio, 1974–1984.

The Poisson model was estimated using the generalized estimating equations of Liang and Zeger (14, 15). In these models, as in a classic Poisson model, we assume $\log[\mathbf{E}(\mathbf{Y})] = \mathbf{X}\beta$, where $\mathbf{X}$ is the matrix of covariates and $\mathbf{E}$ denotes the expected value. In addition, we assume an exchangeable covariance structure

$$\mathrm{cov}(\mathbf{Y}_{ik}, \mathbf{Y}_{jl}) = \mathbf{A}^{1/2}[\sigma_e^2 I + \sigma_w^2 \delta_{kl}]\mathbf{A}^{1/2},$$

where $\mathbf{Y}_{ik}$ is the ith observation in the kth year, δ is the Kronecker delta, and $\mathbf{A}$ is the Poisson variance of $\mathbf{Y}$ under the assumption of independence, which is equal to the expected value of $\mathbf{Y}$. The Liang and Zeger approach allows the calculation of robust variance estimates, which give unbiased hypothesis tests even if the covariance is misspecified. The Liang and Zeger approach incorporates the covariance structure in the estimation of the regression coefficients as well as their variances, giving more efficient estimates of the parameters. All regressions were also repeated assuming an autoregressive covariance structure and using a fixed effects model for each year. Because of the computational complexity, models were initially developed using simple Poisson regression, and final models were then estimated using the Liang and Zeger methodology.

TABLE 1. Percentile distributions and mean values of variables in a study of particulate air pollution, Steubenville, Ohio, 1974–1984

Variable	No. of days	%					Mean
		10	25	50	75	90	
Deaths/day	4,018	1	2	3	4	5	3.07
TSP* ($\mu g/m^3$)	4,016	36	56	91	139	209	111
Sulfur dioxide (ppb)*	3,927	7	12	23	38	55	28
Daily mean temperature (°C)	4,018	−3.9	2.2	11.1	18.9	22.8	10.1
Daily mean dew point temperature (°C)	3,937	−10.0	−3.9	3.9	12.2	16.7	3.6

* TSP, total suspended particulates, ppb, parts per billion.

RESULTS

Table 1 illustrates the distribution of mortality, air pollution, and weather values in Steubenville during the 4,018 days in the analysis. Data on total suspended particulates were missing on only two study days; sulfur dioxide data were missing on 91 days, mean dew point temperature on 81 days, and mean temperature on no days.

Initial analyses of weather variables using nonparametric smoothing showed a nonlinear relation between mortality and both temperature and dew point temperature. Mortality appeared to be higher on very hot and humid days. In Poisson regression models exploring weather dependence, neither temperature nor dew point temperature was a significant predictor of mortality after control for seasonal variation. The previous day's temperature and dew point were likewise insignificant, as were quadratic transformations of these variables. When indicator variables were created for hot days (24-hour mean temperature >21.1°C), humid days (24-hour mean dew point temperature >18.3°C), and hot, humid days (both conditions), only hot, humid days were a significant predictor of mortality. Humid days were almost significant, whereas hot days were not ($p > 0.3$). Hence, the effect seems to be an increase in mortality on humid days when the high dew point temperature keeps the temperature high even at night.

In simple Poisson regressions controlling for weather and season, previous day's total suspended particulates was a significant predictor of daily mortality ($\beta = 0.000352 \pm 0.000111$ (standard error)). Exclusion of all weather and season variables did not substantially change the total suspended particulate association ($\beta = 0.000300 \pm 0.000108$). A variable representing a linear time trend was highly insignificant ($p > 0.4$). Use of the random effects model for year increased the estimated coefficient of total suspended particulates ($\beta = 0.000381 \pm 0.000082$) (table 2). The standard errors are based on the robust variance estimates. The relative risk of 1.04 for particulates is estimated for a 100-$\mu g/m^3$ change in total suspended particulate concentration. The intercept is the logarithm of the baseline mortality rate ($e^{1.039} = 2.83$ deaths/day) with all other variables set to zero.

When an autoregressive covariance structure was specified, no significant autocorrelation was found, and there was little change in the estimated effect of total suspended particulates ($\beta = 0.000352 \pm 0.000111$). Treating year as a fixed effect similarly had little impact on the estimated effect of total suspended particulates ($\beta = 0.000318 \pm 0.000126$), and all of the annual indicator variables were insignificant.

To illustrate the association between number of deaths per day and total suspended particulate counts, we ranked the days by total suspended particulate level and divided them into quartiles. These quartiles were then used in the Poisson regression instead of a continuous particulate variable. Plotted in figure 2 is the relative risk of death for each quartile versus the mean total sus-

TABLE 2. Poisson regression analysis of daily mortality and total suspended particulate count, with year treated as a random effect, Steubenville, Ohio, 1974–1984

Variable	Coefficient (SE*)	t_{robust}	RR* (95% CI*)
Intercept	1.039 (0.015)	69.60	
Winter†	0.101 (0.023)	4.36	1.11 (1.06–1 16)
Spring†	0.044 (0.021)	2.08	1.04 (1.00–1.09)
Hot and humid‡	0.0609 (0.0346)	1.76	1.06 (0.99–1.14)
TSP* (100 $\mu g/m^3$)§	0.0381 (0.0082)	4.66	1.04 (1.02–1.06)

* SE, standard error; RR, relative risk; CI, confidence interval; TSP, total suspended particulates.
† Indicator for winter or spring months
‡ Average temeprature >21 1°C and average dew point temperature >18.3°C.
§ Effect of a 100-$\mu g/m^3$ increase in the previous day's mean.

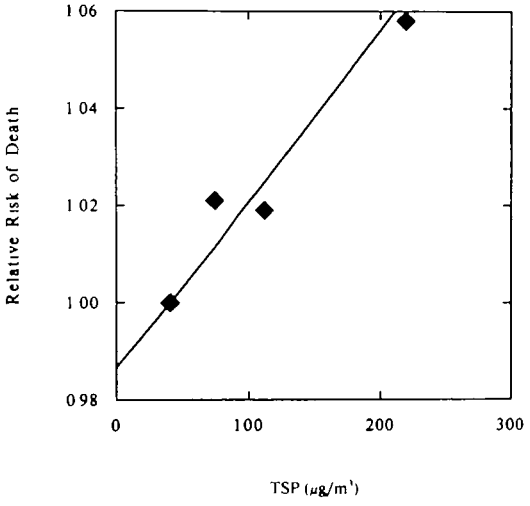

FIGURE 2. Relative risk of death in Steubenville, Ohio, by quartile (♦) of total suspended particulate (TSP) concentration, after controlling, via regression, for the other factors in table 2 The relative risk in the lowest quartile was taken as 1.0. The line shows the relative risk of death as predicted by the regression model that treats total suspended particulates as a continuous covariate, after controlling for season, weather, and year of study.

pended particulate count for that quartile. The relative risk in the lowest quartile was taken as 1.0. Also plotted is the predicted relative risk of death versus total suspended particulates as a continuous covariate, based on the random effects model above.

When sulfur dioxide was used as the exposure measure, a significant association with mortality was also found ($\beta = 0.0104 \pm 0.0042$). The association was less significant than the association with particulates. When both sulfur dioxide and particulates were included in the model simultaneously, particulates remained significant ($\beta = 0.000300 \pm 0.000128$), with little reduction in its estimated coefficient, while sulfur dioxide was insignificant ($\beta = 0.0059 \pm 0.0048$), with a substantial attenuation in the estimated effect. When the sulfur dioxide measurements were taken from the hourly monitor for 1974 to 1978, sulfur dioxide was insignificant without particulates in the model.

DISCUSSION

The finding that hot, humid days were associated with daily total mortality in Steubenville is consistent with previous analyses. Schimmel and Murawski (16) identified and corrected for specific heat wave episodes, determined by maximum temperature, in their analysis of daily mortality in New York City. Daily deaths have been fitted to a nonlinear, exponential function of temperature by Buechley et al. (17) and Marmor (18). Kalkstein (19) analyzed more detailed weather data and mortality using cluster analysis. The cluster characterized by hot and humid days was the only weather pattern consistently associated with excess mortality in US cities. Although there were strong associations of daily mortality with season and weather in this study, the association with air pollution was not confounded by these variables. The effect estimate for total suspended particulates increased modestly (17 percent) upon inclusion of season and weather variables in the model.

While total suspended particulates and

sulfur dioxide were each associated with increased mortality when considered separately, only total suspended particulate count was associated with mortality when both pollutants were considered jointly. Earlier studies have also suggested a stronger association of mortality with particulate rather than sulfur dioxide air pollution.

During the December 1952 air pollution episode in London (20), there were an estimated 4,000 excess deaths in the metropolitan area, including 2,000 excess deaths in London County. British Smoke measurements of particulates had a maximum 24-hour average of about 4,000 $\mu g/m^3$; sulfur dioxide had a peak of 1.5 parts per million (ppm) and average values of 0.95 ppm. Almost exactly 10 years later, in December 1962, there was a similar air pollution episode (21). Sulfur dioxide concentrations peaked at about 1.5 ppm and averaged about 0.80 ppm during the episode. Particulate levels were about one fifth of those of the 1952 episode, but total excess deaths in this episode were only estimated to be 350 in London County (21). Thus, while sulfur dioxide concentrations were similar to those in the 1952 episode, particulate concentrations and excess deaths in the 1962 episode were only one fifth of those seen earlier. This suggests that excess death is associated with particulate air pollution rather than sulfur dioxide air pollution.

Schwartz and Marcus (6) have recently reexamined the association of daily particulate and sulfur dioxide air pollution with the record of daily mortality in London for the winters of 1958–1972 originally compiled by Macfarlane (22). Daily mortality had a highly significant positive association with both particulate and sulfur dioxide pollution. The particulate association was independent of sulfur dioxide concentrations, while the sulfur dioxide association was severely diminished after control for particulates.

Other authors have looked to New York City data for evidence of a linkage between daily mortality and air pollution which is more relevant to the US experience. Daily total mortality was compared with sulfur dioxide and Coefficient of Haze, an optical measure of particulate concentrations. Glasser and Greenburg (23) reported that deviations from expected mortality for the winter months (October-March) during the 5-year period 1960–1964 increased monotonically with Coefficient of Haze and sulfur dioxide. In cross-stratified and multivariate analyses, a stronger association was found with sulfur dioxide. Schimmel and Murawski (16), Kalkstein (19), and Schimmel and Greenburg (24), and more recently, Ozkaynak et al. (8) have elaborated on these analyses for New York City data for the period 1963–1972.

Schimmel and Murawski (16) estimated that 2.8 percent of premature deaths could be attributed to air pollution during this 10-year period. This estimate of premature deaths is the product of the estimated effect of smoke shade times the average smoke shade concentrations, 2.14 CoHs. Over the period considered (1963–1972), there was a sharp reduction in mean sulfur dioxide concentrations from 0.183 ppm in 1963–1966 to 0.059 ppm in 1970–1972. On the other hand, the mean Coefficient of Haze remained relatively stable over the period, changing only from 2.07 for 1963–1966 to 2.13 for 1970–1972. The percentage of excess deaths attributed to air pollution differed little over the period, although sulfur dioxide concentrations decreased by a factor of three. In these analyses, the regression coefficient for mortality versus sulfur dioxide was considerably reduced when the particulate measure was included in the model, while the particulate regression coefficient was only slightly reduced after control for sulfur dioxide.

Even with the small number of expected deaths per day in the Steubenville Standard Metropolitan Statistical Area, there was a statistically significant association between number of deaths per day and mean total suspended particulates averaged over the 24 hours ending at 8:00 a.m. An increase of 100 $\mu g/m^3$ was associated with an estimated increase in deaths per day of 4 percent.

In the reanalysis of London mortality versus air pollution, Schwartz and Marcus (6)

reported a nonlinear association between particles and mortality, with a steeper exposure-response association at lower air pollution concentrations. Based on the regression over all years, the daily death rate would be estimated to increase by 12.0 deaths per day between 50 and 150 μg of smoke per cubic meter and 8.2 deaths per day between 150 and 250 μg of smoke per cubic meter. With a mean daily mortality rate of 295.0 deaths per day, these increases would imply 4.1 percent and 2.8 percent increases, respectively, for a 100-$\mu g/m^3$ increase in British Smoke shade.

Wichmann et al. (25, 26) examined the effect on total mortality of a 5-day air pollution episode which affected parts of West Germany in January 1985. The 24-hour mean sulfur dioxide concentrations reached a maximum of 0.24 ppm, and suspended particulates, measured by beta attenuation, reached a maximum 24-hour mean of 600 $\mu g/m^3$. Total mortality was estimated to increase from 214.9 deaths per day to 231.9 deaths per day during the air pollution episode, i.e., an increase of 8 percent. The mean particulate concentration during the period increased by approximately 200–250 $\mu g/m^3$. In a control area not affected by the pollution episode, deaths per day increased by only 2 percent during the same periods. If we assume a log-linear association, then these results would indicate about a 3.2 percent increase in daily mortality associated with each 100-$\mu g/m^3$ increase in suspended particulates.

In summary, the Steubenville, London (6), and New York City (16) analyses concur in finding suspended particulates, and not sulfur dioxide, to be the principle air pollution correlate with mortality. In addition, there is striking quantitative concurrence in the relative increase in total mortality versus particulates between different studies. While no epidemiologic study can prove causation, the finding of such similar results in several different populations substantially increases the weight of evidence for a causal association between particulate air pollution and daily mortality.

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Invited Commentary: Particulate Air Pollution and Daily Mortality

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In their 1979 review of the health effects of particulate air pollution, Holland et al. (1) concluded that short-term increases in deaths became discernible when concentrations of suspended particulates (as assessed by the British Smoke method) exceeded about 500 $\mu g/m^3$ as a 24-hour average, with the simultaneous presence of concentrations of sulfur dioxide exceeding about 700 $\mu g/m^3$. Similarly, rounded figures of 500 $\mu g/m^3$ for each of these pollutants were quoted by the World Health Organization (2) as levels above which excess mortality might be expected among the elderly or chronically ill. Such statements were not meant to imply any specific threshold. They indicated the point at which, in relatively simple statistical analyses, a role of air pollution began to emerge against the background of much larger climatic and other influences.

Since that time, as the authors of the present paper (3) have pointed out, the application of multiple regression techniques to long series of data from both London (4) and New York (5) have illustrated the possible role of air pollution over a wider range of concentrations, generally with the regression on some measure of suspended particulates being more significant, in a statistical sense, than that on measures of sulfur dioxide. A curious feature has been the steeper slope of mortality-smoke relations at low smoke concentrations as compared with high smoke concentrations (4). In view of the very substantial effects of low temperatures, influenza epidemics, and other illness factors in producing excess deaths during the winter months in the United Kingdom (6), it may be worth considering whether the models developed exerted sufficient control over these important variables.

A series of studies of daily mortality in relation to air pollution have also been conducted in Athens, Greece, in recent years. Initial findings (7) indicated a relation with sulfur dioxide rather than smoke, extending down to relatively low concentrations, although for later years (8) associations were demonstrated with each measured pollutant, that with smoke then being more pronounced. In the Athens data, day-to-day changes in mortality were seen to be more significant for respiratory illnesses than for other conditions (9).

The investigation in Steubenville, Ohio (3), has broken new ground in examining data from a very small population in which the average number of deaths per day was only three, and where, in comparison with former levels in London, concentrations of both particulates and sulfur dioxide have been low throughout. In these circumstances, treating the deaths in terms of a Poisson distribution seems wholly appropriate, but clearly there is little opportunity to explore otherwise interesting aspects such as the age or cause distribution of excess deaths or the way in which peaks in mortality might be linked with environmental factors in a time series approach.

The Poisson assumption tests used by the authors should have been presented. Since the

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mean and variance for the overall distribution of deaths per day are close to equality, overdispersion was probably not a problem. However, it may still occur within the modeling process, and the possible need to allow for extra variation should have been discussed. If such overdispersion were present in the modeling because the mean/variance relation was not truly Poisson in the conditional distributions, then standard errors would be underestimated and the analysis would appear more sensitive than it really was. It is also traditional to remove general trends before investigating the cross-correlation of time series. Modeling does this up to a point, but the authors did not really clarify the degree to which this was achieved. A full plot of the raw data series would have been very helpful, and its absence is to be regretted.

Finally, the results using quartile groupings of the particulate pollutant variable need a bit more discussion. The association with mortality is due entirely to results from the pollutant categories including the high and low extremes. It is possible that there are some very influential data points leading to a general trend estimate not truly representative of the whole data set. There are standard techniques for investigating this which the authors no doubt used. They should have presented the results of this part of the investigation; without them it is difficult to feel confident that the modeling is appropriate and the conclusions justified.

However, the present results (3) indicate, as others have done (4), an association with one of the measured pollutants that is of smaller magnitude than that of climatic or other seasonal effects. Consistency in this respect with the several other studies referred to could add weight to the possibility of a causal role, although one of the other postulates put forward by Bradford Hill (10), biologic plausibility, needs consideration. The tacit assumption with respect to acute effects of air pollution on mortality is that the pollution provides the "last straw," precipitating death in terminally ill people whose cardiorespiratory systems are already severely compromised. Clearly, such information as is available from experimental inhalation studies involving normal, or less severely impaired, subjects does not provide an adequate lead on mechanisms—although, with respect to sulfur dioxide, the maximum exposures in Steubenville that might be inferred from the summarized data in table 1 (3, p. 15) fall far short of any reported to have detectable effects (11). There is virtually no experimental evidence related to nonspecific total suspended particulates, but any acid aerosol component would also have been likely to fall far below concentrations reported to have positive effects in experimental studies (11).

The comment that should perhaps be added to the present findings, as it was to studies in London (12), is that extrapolation to situations differing substantially from those in which the investigation was done should be undertaken with caution. It is not just a matter of how particulates were measured, whether by reflectance (British Smoke) or gravimetrically (total suspended particulates). There can never be any general "equivalence" between such assessments, since they are based on wholly different properties of the material. The nature of suspended particulates varies widely with the types of sources and can never be fully specified. The composition of particulates today, whether in Steubenville or elsewhere, will differ greatly from that in London in the 1950s, which was dominated by the tarry carbonaceous emissions from coal fires. Whether some other types of particulates may be of greater or lesser toxicity remains an open question.

Caution is also required with respect to the temporal distribution of any of the pollutants during the day. It may be that transient peaks are more important with respect to acute effects than average exposures over 24 hours, and that is certainly the implication from experimental studies with sulfur dioxide (13). The relation between transient peak values and daily mean values is another feature that varies with the nature of the sources, the ratio between the two being relatively small for "area" sources (such as the domestic chimneys

that once pervaded all British cities) but greater where just a few large point sources are dominant. While this might imply that a more appropriate measure would, in each circumstance, show a stronger relation between air pollution and health outcome, it would reconcile apparent differences in the absolute values associated with adverse effects, with peak exposures experienced from point sources being much closer to those from area sources than 24-hour averages would suggest.

Since the study area formed part of the intensive Harvard Six Cities Study (14), many of these additional features of the pollution scene will have been documented and referred to in other papers, allowing assessment of relevance to other locations.

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The Authors' Response to Waller and Swan

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More than 20 years have passed since the Clean Air Act of 1970 was passed and National Ambient Air Quality Standards were established to protect the US public from the adverse health effects of air pollution. The focus of regulatory control in the 1970s was control of sulfur and particulate pollution. The primary epidemiologic evidence suggesting an adverse health effect of particulate air pollution was based on daily mortality and daily air pollution measurements collected in London, England, in the 1960s (1, 2). In their commentary, Waller and Swan (3) noted that, using simple statistical analyses, investigators concluded that increased mortality could not be observed at concentrations below about 500 $\mu g/m^3$ for a 24-hour average. Ware et al. (4) interpreted these same analyses as showing a dose response down to the lowest levels observed. Recently, more sophisticated statistical analysis (5) has indeed suggested that an association between increased mortality and higher particulate and sulfur pollution could be found in the London data down to the lowest observed concentrations.

Nevertheless, the epidemiologic evidence for such an association has been downplayed in the US regulatory debate because of parochial attitudes. First, it is claimed that air pollution in the United States is not like that found in London. Second, it is claimed that people in the United States have different life-styles, medical delivery systems, etc., from their English cousins and therefore may not be exposed in the same fashion or have the same response to air pollution. There is an underlying feeling that American air quality standards should be based on studies of US citizens using US exposure methods.

Remarkably, there is considerable evidence that particulate air pollution was also associated with increased mortality in New York City in the 1960s (6) and recently in Santa Clara (7) and Los Angeles (8) counties, California. These studies have been discounted because they are based on an optical measure of particulate concentrations rather than the gravimetric measurement method mandated by the Environmental Protection Agency (EPA).

The Steubenville study (9) breaks new ground in presenting associations based on the EPA's reference method for particulate air pollution at the time the data were measured. Statistically significant associations were found even though the population was small, with only an average of three deaths per day. If this association is real, similar results should readily be found in other large US cities. Unfortunately, the number of communities with complete daily records of particulate concentrations measured by EPA-approved reference methods is very small. In fact, the motivation for even attempting such an analysis in the relatively small town of Steubenville, Ohio, was the availability of a long record of daily total suspended particulate measurements. In almost all other communities, such analysis is not possible, because local air pollution agencies sample particulates at a frequency of once every 6 days in compliance with EPA recommendations. While such intermittent sampling may

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be sufficient to estimate annual mean values and even the maximum concentration for the year, it is not adequate for time series analysis of health effects.

Given the small number of deaths per day, the Steubenville data are limited in the information which they can provide. Waller and Swan (3) have described several areas in which additional analyses are required. Specifically, cause of death should be investigated for guidance on mechanisms of action. Likewise, exposure to particulate pollution must be differentiated to identify the specific averaging times associated with excessive mortality and the specific chemical component of the mix of particulate pollutants which might be identified as the causative agent.

Waller and Swan make several specific comments regarding the Steubenville analysis. They first note the importance of low temperature and influenza epidemics in producing excess deaths in the winter months in London, i.e., periods when pollution was also high. In Steubenville, the highest concentrations of particulate pollution were observed during the summer, so that confounding by winter epidemics or low temperatures is unlikely. Extremely high temperatures were associated with increased mortality in Steubenville and were included in the mortality models.

Second, they note that there might be overdispersion of the residuals from the Poisson model of daily mortality. Following McCullagh and Nelder (10), we can estimate the overdispersion parameter as the ratio of the chi-square statistic to the number of residual degrees of freedom. For the model of daily mortality versus total suspended particulates, this was somewhat less than 1.0, suggesting no overdispersion.

Third, Waller and Swan suggest that the association may be due entirely to very influential data points at the high and low extremes. Our interpretation of the relative risk by quartile of exposure is that this association is found across the entire range of exposures.

Finally, Waller and Swan suggest that biologic plausibility as defined by Bradford Hill (11) needs to be considered. While biologic plausibility is one of nine criteria Hill proposed for assessing causation, his specific comment was:

> It will be helpful if the causation we suspect is biologically plausible. But this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day (11, p. 298).

In the case of particulate air pollution and mortality, there is general agreement that at high concentrations such as those seen during the 1952 London smog episode (12), particulate air pollution will cause death. The question is whether it is plausible that these effects extend down to concentrations currently observed in American cities.

We suggest that the dose-response association between total mortality and daily particulate air pollution found in Steubenville provides evidence for a significant health effect even at concentrations at or below the National Ambient Air Quality Standard for particulate matter. This association is consistent with results of daily mortality analyses in the United Kingdom and the United States. Further study is required to define mechanisms and specific agents. Nevertheless, as Bradford Hill concluded in his lecture,

> All scientific work is incomplete—whether it be observational or experimental. All scientific work is liable to be upset or modified by advancing knowledge. That does not confer upon us a freedom to ignore the knowledge we already have, or to postpone the action that it appears to demand at a given time (11, p. 300).

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