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Reexamination of London, England, Mortality in Relation to Exposure to Acidic Aerosols During 1963–1972 Winters

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Air pollution epidemiology since the 1950s has been able to demonstrate that increases in daily mortality in London, England, were associated with elevated concentrations of index air pollutants, i.e., British Smoke (BS) and sulfur dioxide (SO_2). In this work, we reanalyze that portion of the 1958–1972 winter mortality-pollution record for which daily direct acid aerosol measurements were made at a central site in London (St. Bartholomew's Medical College). The purposes of these exploratory analyses are to examine the dataset for indications of a relationship between acid aerosol pollution and human mortality and to compare any noted associations with those for other pollution variables. It is found that the log of acid aerosol concentrations is more strongly associated with raw total mortality in bivariate analyses than is BS or SO_2, despite the fact that acid data are available from only one central site (versus seven disperse sites for BS and SO_2). The logarithmic nature of the exposure side of the H_2SO_4-mortality relationship implies a saturation model of pollution effects, possibly due to multiday pollution harvesting influences on a susceptible subpopulation. Moreover, mortality-pollution cross-correlation analyses indicate that mortality effects usually follow pollution in time, supporting a causal relationship between the two. The apparent advantage of H_2SO_4 over BS in predicting total raw mortality is consistent with the hypothesis that it is the portion of particulate mass of greater health significance and may also allow the development of London mortality results which are more easily transferable to other environments than is the case for existing BS results.

Introduction

Air pollution epidemiology since the 1950s has been able to demonstrate that increases in daily mortality in London, England, were associated with elevated concentrations of index air pollutants, i.e., British Smoke (BS) and sulfur dioxide (SO_2). For example, the record of London winter mortality and air pollution during the period from 1958–1972 has been studied extensively using various statistical approaches. Martin and Bradley (*1*) first investigated the winter of 1958–1959, separately examining the associations of total mortality, bronchitis mortality, and pneumonia mortality with the daily mean of seven London County sites' BS and SO_2 measurements. To control for slow moving fluctuations in mortality (e.g., due to influenza epidemics), Martin and Bradley used daily deviations from a 15-day moving mean of mortality. They noted that, as pollution increased, there was a slight falling off of the rate of increase in mortality. The authors concluded that both BS and SO_2 were significantly correlated with total mortality, with the highest reported association being with the logarithm of BS. Dawson and Brown (*2*) reanalyzed the 1958–1959 data using more refined time series regression models, also finding that the logarithm of pollution, or a saturation model, provided the best fit of the data.

The 1958–1972 composite pollution-mortality record was first compiled by Macfarlane (*3*), who presented time series plots indicating the seasonal covariance of daily total mortality and pollution in Greater London during this period. Mazumdar, Schimmel, and Higgins (*4,5*) analyzed the winter data from this period using three types of analyses, finding an association between BS and total daily mortality, but no threshold for effects was indicated by the regression of mortality on a quadratic model. Indeed, a decline in BS regression coefficients was found with increasing BS levels, a result consistent with Dawson's saturation model. Similarly, Ostro (*6*) found higher regression coefficients for lower BS (< 150 $\mu g/m^3$). Ostro suggested several possible reasons for this, including the

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possibility that lower BS may contain a higher proportion of toxics such as sulfuric acid, or that the pattern may simply be a fit of an S-shaped toxicological dose-response curve.

Advanced time series models have recently been applied to the 1958–1972 winter mortality data set by both Shumway et al. (*7*) and Schwartz and Marcus (*8*). In the former analysis, the best model for the combined data of all years involved the regression of mortality prefiltered to remove autocorrelation (in a manner similar to a 15-day moving average) on temperature lagged 2 days before mortality and the logarithm of the same-day levels of either BS or SO_2. The authors concluded that pollution acts positively and instantaneously on mortality, with no evidence of a threshold of effect.

Schwartz and Marcus studied each of 14 winters separately (to remove the time trend of falling mortality in the first few years), as well as collectively for years 1965–1972. They considered same-day pollution and meteorological variables as predictors of 15-day moving average mortality deviations in a linear model, although they also noted that there is a curvilinear relationship between pollution and both raw mortality and deviations from the 15-day moving average of mortality. They suggested that this may be an artifact of autocorrelation in the pollution exposures and, thus, on extremely high pollution days, responders may have been depleted by responses on preceding days of moderate pollution. Also, avertive behavior by the population was suggested as a possibility, as well as the fact that the particulate mass/BS ratio may have differed on high pollution versus low pollution days. It was found that BS had a consistent relationship with mortality independent of SO_2, which was less reliably associated with mortality in their linear models.

Although aerosol acidity levels were not routinely measured during the London fog episodes in the 1950s and early 1960s, acidity levels have long been suspected as a causal factor in the mortality associated with these episodes. Original reports on the December 1952 London fog (*9–11*) mention sulfur trioxide and/or its reaction product (i.e., sulfuric acid mist) as probable important smog constituents, although no direct measurements were available. Aside from the deleterious effects of sulfuric acid suspected at the time, anecdotal evidence also pointed toward acidic pollution as a causal agent. For example, unexpected livestock respiratory morbidity and deaths at the Smithfield Club's show at Earl's Court, London, from December 5–12, 1952, were reported during that smog episode (*12*). Interestingly, it was noted at the time that only the prize cattle were affected, or those whose stalls would have been cleaned out most often, thereby removing urine, an important source of acid neutralizing ammonia gas (Dr. Lynne Reid, Boston Children's Hospital, personal communication, 1987). This hypothesis is supported by the chamber work of Collumbine et al. (*13*), who reported that "enough ammonia could be generated from the excreta of . . . animals to neutralize an otherwise lethal concentration of acid mist." Other pollutants, such as BS and SO_2, although known to be elevated at the time, would be expected to affect livestock more evenly, since ammonia would not neutralize those pollutants, and their effects alone would not explain the impact specificity to the prize cattle. Doctors thought enough of sulfuric acid (H_2SO_4) as a health threat at the time that bottles of ammonia with wicks were placed in hospital wards during pollution episodes in an effort to neutralize the acids (Dr. David Bates, University of British Columbia, personal communication, 1987). Commins and Waller's summary of their intermittent direct aerosol acidity measurements during fog episodes (commencing in 1957) indicates that the highest recorded central London acid aerosol hydrogen ion (H^+) concentrations, expressed as H_2SO_4, were a 347 $\mu g/m^3$ H_2SO_4 24-hr mean and a 678 $\mu g/m^3$ 1-hr mean during the December 1962 episode (*14*). These data demonstrate that extreme aerosol acidity levels did occur during such fog episodes in London and, in conjunction with the anecdotal evidence, lend support to the hypothesis that acidic aerosol pollution was a causal agent in the reported London fog-mortality associations.

The only known field epidemiologic investigation in which sulfuric acid was measured directly and was implicated as the causative agent for human health effects took place in Yokkaichi, Japan, in the 1960s (*15*). During a period of about 8 years, when there were high emissions of H_2SO_4 mists from a titanium dioxide pigment plant, there was an excess of 600 cases of respiratory morbidity in downwind populations. Acid concentrations and the incidence of lung disease declined with distance from the plant. Furthermore, lung disease incidence dropped precipitously after sulfuric acid emission controls were applied. Unfortunately, the actual H_2SO_4 concentration data were severely limited, and most inferences of exposure were obtained from litmus paper measurements collected at various locations in the residential district around the plant. The reported associations are, however, strongly suggestive of a causal relationship between acid aerosol exposure and respiratory disease incidence.

Recent work on the mortality and morbidity effects of sulfates also indicates health effects of ambient exposures to acidic sulfate species. In their original study of Southern Ontario (*16*), Bates and Sizto found a significant correlation ($p < 0.001$) between acute hospital admissions for respiratory disease and daily O_3 and SO_2 concentration and ambient temperature, with 24- and 48-hr lags of the environmental data. However, in their subsequent analysis (*17*), using available every sixth-day 24-hr sulfate (SO_4^{2-}) ion data, they found SO_4^{2-} to be the index most highly correlated with both total respiratory and asthma admissions in summertime. Sulfate was probably only a surrogate for the acidic components within it in this case, since the relationship observed was more statistically

significant than that of irritants such as ozone and sulfur dioxide, known to be more potent than ammonium sulfate, which is a more neutral SO_4^{2-} compound and the species most commonly present in the atmosphere. Also, in a recent cross-sectional study of U.S. mortality rates, Ozkaynak and Thurston (*18*) presented an analysis of 1980 mortality in relation to available alternative aerosol pollutant surrogates that supports the importance of sulfate aerosols to human health effects. In addition to sulfate concentrations, the analysis considered annual average concentrations of total suspended particulate matter (TSP), inhalable particulate matter (IP), and fine particulate matter (FP). It indicated that the levels of significance for pollutant indices as predictors of mortality rate were ordered SO_4^{2-} > FP > IP > TSP, with only FP and SO_4^{2-} being statistically significant. It is suspected that, in these analyses, the FP and SO_4^{2-} measures may be acting as surrogates for the acidic aerosols they comprise.

Published animal and human volunteer studies support the observational epidemiologic evidence indicating acidic aerosols to be causative agents in respiratory health effects. Schlesinger has summarized the known effects of inhaled acids on respiratory tract defense mechanisms, based primarily on animal studies (*19*). He found that acids may impair these defenses, possibly increasing susceptibility to chronic lung disease. Both Schlesinger (*19*) and Lippmann (*20*) noted that the effects of cigarette smoke and H_2SO_4 were found to be the same after either single or intermittent exposures, supporting the hypothesis that H_2SO_4 exposures may have a role in the development of chronic bronchitis.

Utell (*21*) has critiqued available human exposure studies for acid aerosols, reporting that they demonstrate the importance of acidity to the irritant potency of sulfates as related to the alteration of lung function. He noted the importance of host factors such as airway ammonia or whether the subject is an extrinsic asthmatic. Utell also reported the lack of evidence for lung function alterations at more typical ambient H_2SO_4 levels (below 100 $\mu g/m^3$) but pointed out that this may be because no investigators had performed clinical tests at such low concentrations. While as yet circumstantial, these results provide further supportive evidence of the potential for acidic aerosols to adversely affect human respiratory functions. In view of these and various other studies, Lippmann (*20*) has speculated that the active agent, and the particulate pollutant with potentially the highest level of significance, would be the hydrogen ion concentration (H^+).

In this work, we reanalyze that portion of the 1958–1972 winter mortality-pollution record for which daily direct acid aerosol measurements were made at a central site in London (St. Bartholomew's Medical College). The purpose of these exploratory analyses is to examine the data set for indications of a relationship between acid aerosol pollution and human mortality. Of special interest is to characterize any noted acid-mortality associations and to compare them with mortality associations for other pollution variables in the data set.

London Air Pollution and Human Mortality Data Collection

The data considered in these analyses include pollution and mortality records collected in Greater London during winter periods (November 1–February 29) beginning in November 1963 and ending in February 1972. The air pollution data were compiled from one of two sources. First, BS and SO_2 data (in $\mu g/m^3$) were compiled by Macfarlane (*3*) as daily means of seven sites run by the London County Council and spatially distributed throughout London County. It is important to note that during the period under consideration these daily pollution samples were collected for 24-hr periods ending at noon of the day for which they are reported and therefore include 12 hr of information about the previous day's pollution (*4*). A second data set of BS, SO_2, and aerosol acidity (H^+, calculated as $\mu g/m^3$ sulfuric acid) was also compiled for one central London site run by the Medical Research Council Air Pollution Research unit at the St. Bartholomew's Medical College. These pollution samples were collected from 3:00 A.M. of the day for which they are reported to 3:00 A.M. the next day during the winter of 1963–1964, but were collected from midnight-to-midnight of each sampling day from April 1, 1964, onward in order to directly coincide with mortality records. More details regarding the central site location and the collection procedures for the pollution observations are presented by Ito and Thurston (*22*), as well as by Commins (*23*) and Commins and Waller (*14*). Figure 1 displays the Greater London Metropolitan Area, showing the seven sites used to compute the mean BS and SO_2 data (*24*), as well as the St. Bartholomew's Medical College central site.

The Greater London mortality data, as compiled by Macfarlane (*3*), was obtained from the London General Register Office for winter periods beginning in 1958 and for all days commencing in April 1965. Total mortality, respiratory mortality, and cardiovascular mortality were all compiled daily during these periods, but only total mortality is considered in this work. The Greater London population was fairly stable during the period considered in this research (1963–1972), averaging about 8 million people. For example, in 1958 the Greater London population was 8.2 million, dropping only 5% to 7.8 million by 1970.

Pollution and Total Mortality Data Analyses

The pollution and mortality data for each of the nine winters of data were combined into one data set for analysis. This procedure is acceptable in this case

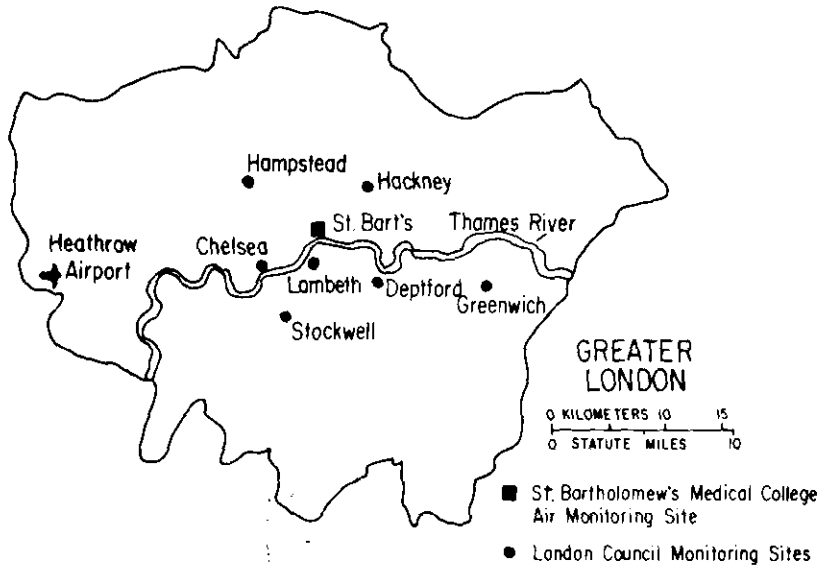

FIGURE 1. Historical London, England, air pollution monitoring sites.

because the period under study, late 1963 to early 1972, is subsequent to the implementation of the London smoke control zones (1961–1963) (*25*) and is therefore a period of fairly constant average winter pollutant concentrations. Prior to combining the data, each year's total mortality data was also prefiltered using a filter of the form applied by Shumway et al. (*7*). As can be seen in Figure 2, this filter weights the mortality data in a manner very similar to the calculation of deviations from a 15-day moving average of mortality, except that it eliminates the undesirable short-term cyclical fluctuations (ripples) in the running mean deviations filter. However, this calculation cannot be made for the first and last 9 observations for each winter, and these 18 data observations were therefore trimmed from each winter's data set. The data for the nine winters were then concatenated in time series order, but with 15 missing observations for each variable placed between each winter in order to prevent false autocorrelations between the end of one winter and the beginning of the next. The resulting data set comprised a total of 1041 observations, of which 120 were the added missings, for a final total of 921 observations of daily pollution, total mortality, and filtered total mortality data for the nine-winter data set.

The means and standard errors of key variables in the data set considered are displayed in Table 1. Values for the logs of the pollution variables are presented along with the raw variables because, as discussed previously, the logs have been shown in several previous analyses to be more strongly related to mortality than the raw observations. As can be seen in Table 1, the means and medians of the log transformed variables are closer to one another than is true for the raw observations, a product of the fact that the pollution variables are roughly log-normally distributed. The BS mean at the central site (measured at St. Bartholomew's) is 10% lower than the mean of the BS observations at seven London sites, but the SO_2 mean is approximately one-third higher, on average, than the mean of SO_2 concentrations at seven sites.

Table 2 shows the bivariate correlations of the central site and mean of seven-site pollution variables with the two mortality variables, both on the same day as the pollution readings and the day following. A comparison of the same-day BS correlations would appear to indicate that the mean of the measurements at the seven sites provides a better index of exposure than the central-site measurements. However, it is seen that the central site BS correlations with each mortality measure improve for the following day and are approximately equal to the same-day seven-site mean BS correlations in those cases. Conversely, the seven-site

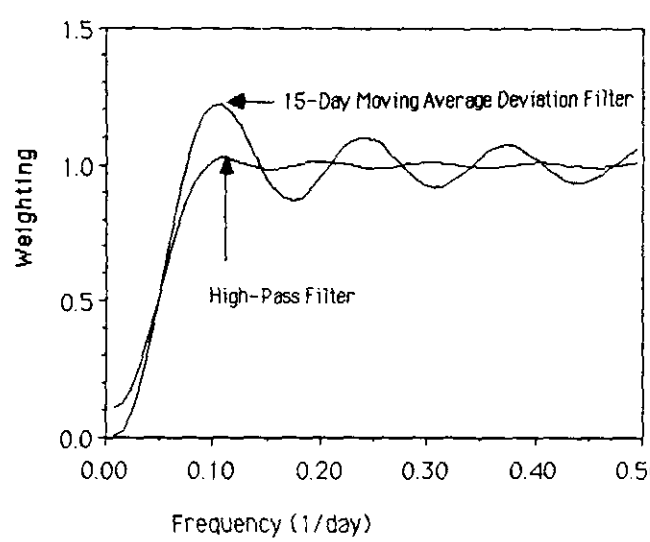

FIGURE 2. Frequency/response of the high-pass filter as compared to the frequency/response of a 15-day moving average deviation filter.

Table 1. Means, medians, and standard errors of select pollution, meteorological, and mortality data for nine winters, 1963–1972.[a]

Variable	Mean	Median	SE of mean
Seven-site British Smoke[b], $\mu g/m^3$	100.3	76.0	2.8
Log of seven-site BS[b]	4.37	4.33	0.02
Central-site BS[c], $\mu g/m^3$	90.1	68.0	2.3
Log of central-site BS[c]	4.28	4.22	0.02
Seven-site sulfur dioxide[b], $\mu g/m^3$	239.5	209.0	4.2
Log of seven-site SO_2[b]	5.37	5.34	0.46
Central-site SO_2[c], $\mu g/m^3$	317.7	282.0	5.3
Log of central-site SO_2[c]	5.65	5.64	0.48
Central-site H_2SO_4[c], $\mu g/m^3$	6.5	5.2	0.2
Log of central-site H_2SO_4[c]	1.64	1.65	0.02
Temperature, °C at 9 A.M.	5.47	5.40	0.12
Relative humidity, % at 9 A.M.	81.0	82.0	0.3
Total daily mortality[c]	281.1	271.0	1.5
Filtered total daily mortality[c]	0.00	−1.24	0.6

[a] $n = 921$.
[b] Sampled noon of prior day to noon of observation day.
[c] Sampled midnight of prior day to midnight of observation day.

BS correlations decline for the next-day mortality cases. This apparent inconsistency is explained by the fact that the central-site data coincide in time with the mortality records, whereas the seven-site mean pollution sample period preceded the daily mortality period by 12 hr. Thus, the central-site analysis has revealed the true time lag in the pollution-mortality association that had been hidden in prior analyses because of the way in which the seven-site mean pollution data had been collected. Similarly, the mean of seven-site SO_2 correlation with the mortality indices drops off when the next day mortality is employed, while the central-site correlation remains strong. Interestingly, the central-site SO_2 variables show a slightly stronger correlation with the mortality indices than do the mean of seven-site variables. Overall, once the time differences in the pollution sampling period are accounted for, there appears to be a lag in the association of pollution with mortality (i.e., pollution precedes mortality), and the central site SO_2 and BS data appear roughly comparable to the mean of seven-site data as indices of exposure.

The log of H_2SO_4 measured at the central site is much more strongly correlated with raw total daily mortality than any of the other variables listed in Table 2, especially when it is correlated with the next-day mortality ($r = 0.31$). It is also clear from the results in Table 2 that the logarithm transformation helps the acid-mortality association more than is true for BS or SO_2. For the filtered mortality variable, however, the H_2SO_4 correlation is weakened versus raw total mortality [e.g., $r = 0.19$ for log (H_2SO_4) with next-day filtered mortality]. Thus, the St. Bartholomew's College H_2SO_4 measurements appear to be correlated with Greater London mortality, especially before the mortality data are filtered for slow moving fluctuations. The superiority of the log of H_2SO_4 concentration versus the raw H_2SO_4 data in correlations with total mortality agrees with the previous analyses of British Smoke-total mortality associations. This may imply that a saturation of mortality effects is indeed occurring over 2 or more days and that a cumulative effect of several episode days may be more relevant than modeling a single day effect alone.

Figure 3 shows time series plots of the daily wintertime total mortality and filtered total mortality. Although the filtered total mortality has largely removed slow-moving fluctuations in the mortality data, the winters of 1967–1968 and 1969–1970 are still slightly nonstationary, due to major influenza epidemics in those years (*3*). It may be necessary to also (or instead) control for these effects by considering an influenza epidemic dummy variable in subsequent regression analyses of these data, as has been suggested by Wyzga (*26*). Also, Roth, Wyzga, and Hayter (*27*) have noted that such filtering methods to control for autocorrelations can inadvertently affect the mortality-pollution relationship indicated by subsequent multivariate methods. Thus, when the data filtering influences the pollution-mortality relationship, as is apparently the case for the H_2SO_4-mortality correlation, then the sensitivity of results to alternative autocorrelation-reducing methods and filters deserves further investigation.

Figure 4 displays four scatter plots of mortality versus log-scale acid aerosol concentrations. Figures 4*a* and 4*b* display the effects of filtering on the entire nine-winter data set. There is considerable random scatter in Figure 4*a* for values above 400 deaths per day, which, as can be seen in Figure 3, are all associated with the influenza epidemics in the winters of 1967–1968 and

Table 2. Bivariate Pearson correlations of central-site and seven-site mean pollution variables with same-day and following-day raw and filtered total mortality for 1963–1972 London winters.[a]

Variable	Raw total mortality		Filtered total mortality	
	Same day	Next day	Same day	Next day
Seven-site British Smoke	0.14	0.09	0.22	0.10
Central-site British Smoke	0.11	0.12	0.15	0.17
Seven-site sulfur dioxide	0.13	0.11	0.19	0.13
Central-site sulfur dioxide	0.17	0.17	0.20	0.21
Central-site sulfuric acid	0.20	0.21	0.14	0.16
Log(seven-site BS)	0.15	0.10	0.24	0.12
Log(central-site BS)	0.11	0.14	0.14	0.22
Log(seven-site SO_2)	0.12	0.10	0.18	0.14
Log(central-site SO_2)	0.17	0.17	0.21	0.21
Log(central-site H_2SO_4)	0.29	0.31	0.17	0.19

[a] $n = 921$.

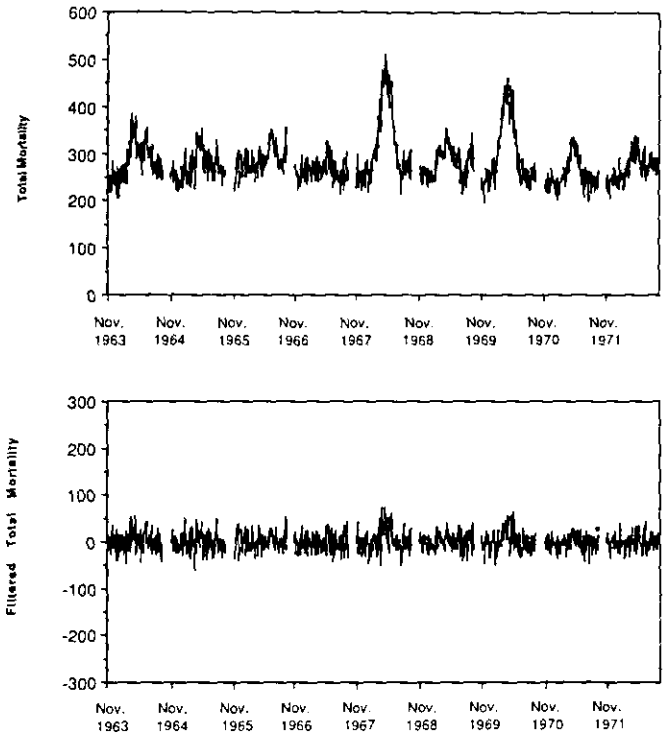

FIGURE 3. Time series plots of raw and filtered total daily mortality for Greater London winters, 1963–1972 (n = 921).

1969–1970. The mortality filtering reduces this scatter, as shown in Figure 4*b*, but the overall relationship is weakened at the same time (r = 0.17 versus 0.29). This weakening effect can be seen more clearly in Figures 4*c* and 4*d*, which are plots of the raw and filtered mortality for a winter (1965–1966) without a major influenza epidemic (see Fig. 3). In this case, the raw mortality versus log of acidity in Figure 4*c* exhibits a linear trend (without the influenza scatter), and the filtering seems to be of little help to the residuals, but clearly weakens the pollution-mortality relationship in Figure 4*d* in the process of removing shared long-wave fluctuations in the data. Thus, the advantage provided by the filtering of data (i.e., suppression of extraneous influenza effects) are at the same time counterbalanced by its indiscriminate suppression of other, perhaps causal, slow-moving associations between mortality and pollution indices.

Figure 5 shows, for the 1965–1966 winter, the effects of the log transform on the acid aerosol-total mortality relationship. This year is of interest because it is both free of major influenza epidemic effects and is a winter for which there are no extreme acid aerosol values that might overly influence the results for one winter of data (n = 102, maximum daily H_2SO_4 = 24 μg/m^3). Figure 5*a* clearly displays a saturation of effects, apparently leveling off at about 10 μg/m^3 H_2SO_4 in these data. Total mortality versus a log scale plot of acid aerosol, as plotted in Figure 5*b*, indicates the improved linear fit afforded by this transformation of the pollution data.

Figure 6 displays a time series plot of the 1965–1966 winter aerosol acidity and raw total mortality data for the period (November 10, 1965–February 19, 1966, n = 102). For this winter, of all the central site and seven site mean pollution variables considered in this work (i.e., BS, SO_2, and H_2SO_4), the maximum correlation found with either mortality index was that between the log of H_2SO_4 and the following day's total raw

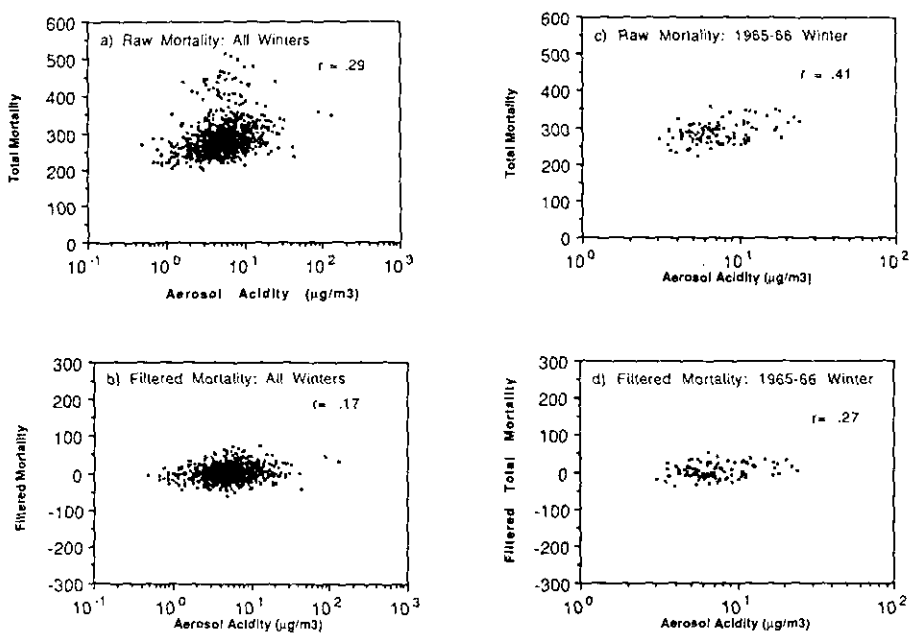

FIGURE 4. Scatter plots of raw and filtered total daily London mortality versus log scale acid aerosol concentrations for all winters (n = 921) amd for the winter of 1965–1966 (n = 102).

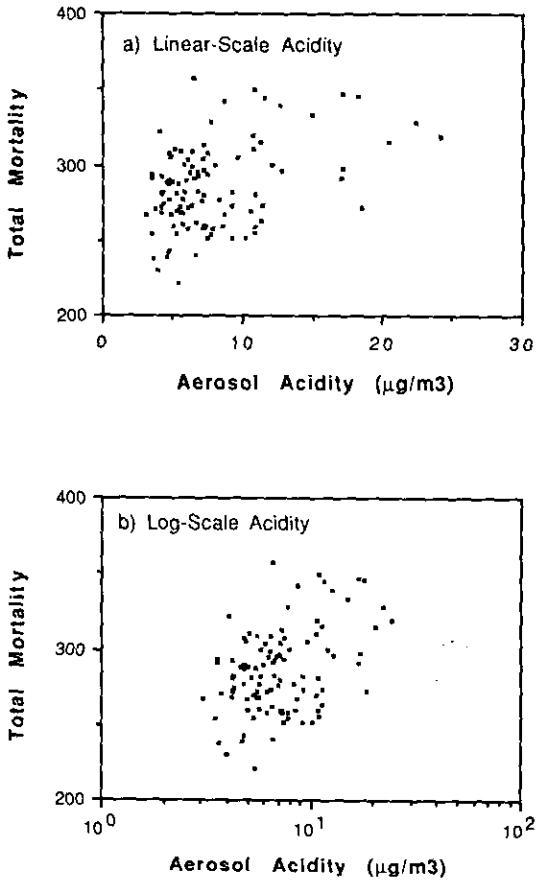

FIGURE 5. Daily total raw mortality versus log and linear scale aerosol acidity concentrations for the London winter of 1965–1966 (n = 102).

mortality (r = 0.52). Moreover, it is visually apparent from this plot that the mortality peaks are well correlated with the acid peaks and generally follow them in time. Thus, in this winter period having no dramatic outlier pollution concentrations or major influenza epidemics to complicate the data, the overall air pollution-mortality association is more clearly evident.

Figure 7 displays the autocorrelation of certain key variables discussed previously. The autocorrelation is defined as the Pearson correlation of a given variable's observations with its value on days 1, 2, 3, etc., subsequent or previous. It is immediately apparent from these plots that raw total mortality shows the greatest extent of autocorrelation out to 15 days. This is no doubt at least in part due to the previously discussed slow moving influences on mortality evidenced in Figure 3. The log of central site acid (as H_2SO_4) shows the autocorrelation pattern most similar to raw total mortality. The filter's effect on total mortality autocorrelation is also apparent, and essentially all autocorrelation has been removed from this variable. The log of the mean of seven-site BS and the central-site BS look very similar to each other, exhibiting less autocorrelation than the H_2SO_4 data and both showing secondary peaks at 7 and 14 days lag. This indicates that there is a day-of-week effect on BS, with each day being more correlated with the same weekday 1 and 2 weeks ago/ahead than other surrounding days in the previous/following 2 weeks. This day-of-week effect is likely a product of BS concentrations being tied closely to day-to-day particulate emission variations. Acid concentrations, conversely, have no apparent day-of-week effects. This is consistent with the hypothesis that atmospheric acidity is a complex quantity, likely being dependent on such nonemission variables as catalytic salt availability for sulfur dioxide conversion and ammonia availability for acid neutralization.

Figure 8 presents the cross-correlations of mortality over time with key pollution and meteorological variables. At day zero (k = 0), these plots show the bivariate correlation of each variable with mortality, but the values plotted to the right of day zero indicate the correlation of that variable with mortality on the following days (up to 15 days later). Conversely, the values to the left of day zero are the correlations of the variable with mortality on the previous days (up to 15 days before). Higher absolute correlations (either positive or negative) after time zero than before time zero imply that the mortality follows the variable considered in time, providing one method to test for causality. The log of H_2SO_4, for example, is better correlated with both indices of mortality considered on the next day than on the same day the pollution occurred, and the area under the curve left of time zero is smaller than to the right of time zero. Indeed, a paired t-test of the differences between the cross-correlation on the days prior and the days following indicates that the total raw mortality-log of H_2SO_4 correlations on days following are significantly higher than those on days

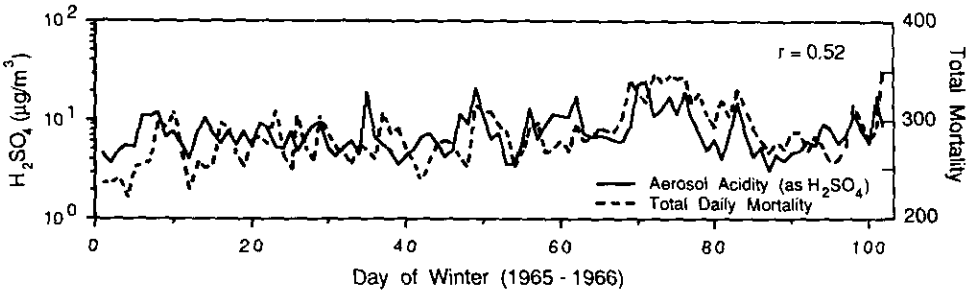

FIGURE 6. Time series plot of Greater London daily total raw mortality and aerosol acidity (as H_2SO_4) for the winter of 1965–1966 (n=102). The correlation coefficient noted is that for the log of acidity with the next day's mortality.

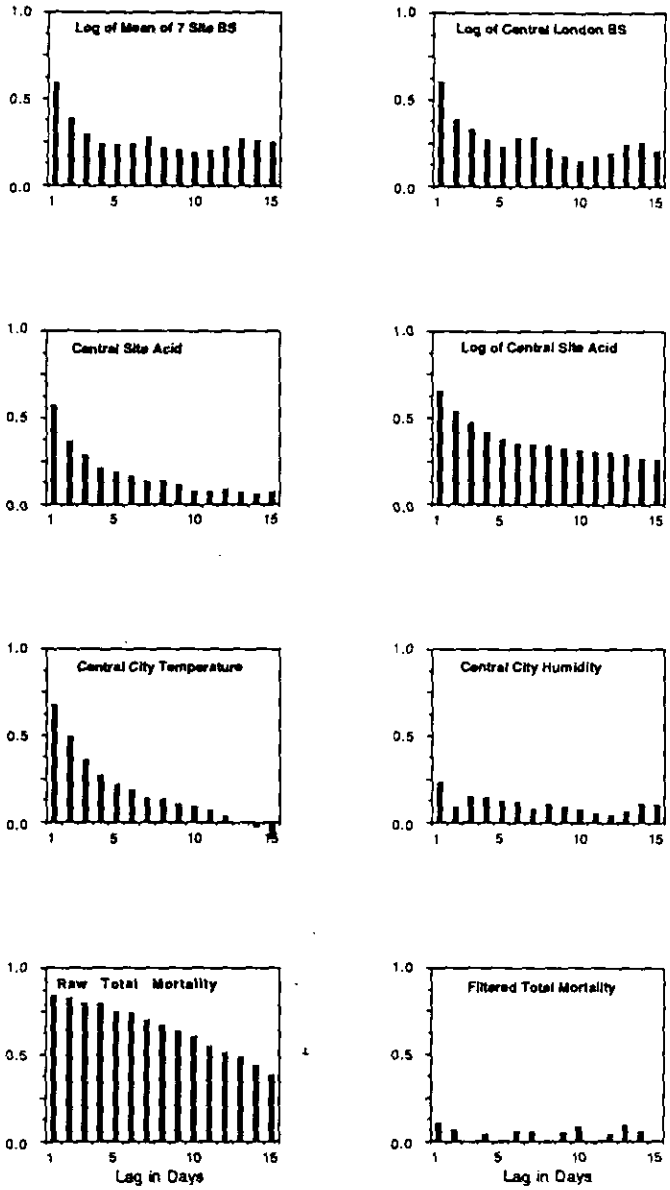

FIGURE 7. Autocorrelations of key London pollutant, weather, and mortality variables during 1963–1972 winters (n = 921).

prior (p < 0.001). Similarly, temperature shows a larger (negative) correlation with mortality on days after time zero, the maximum negative correlation occurring 2 to 3 days after time zero. This agrees with Shumway's regression result that his best mortality model considered the temperature 2 days prior to the mortality observation (*7*).

The two BS variables presented in Figure 8, like H_2SO_4, also show higher mortality correlations on days following than on days prior, and the previously noted 7 and 14 day-of-week effects on BS autocorrelations are also evident in the BS-mortality cross-correlations. It is interesting to again note, in the case of the seven-site mean BS versus the central site BS cross-correlations, that the former shows the same-day effect to be largest, while the latter shows a 1-day lag to the maximum pollution-mortality relationship. Central city humidity does not show an effect similar to the other variables: All correlations (except same day) are small. If any trend is discernible, it is that high mortality precedes high humidity. Overall, it appears that these cross-correlation plots imply that increased mortality follows both pollution exposures and low temperatures in wintertime, a result which lends support to the hypothesis of causal relationships between these variables and human mortality.

Conclusions and Future Plans

This paper has briefly summarized past analyses of the 1958–1972 wintertime London mortality and then proceeded to consider the possible role of acid aerosols in the previously reported air pollution-mortality associations. It was found that the log of acid aerosol concentrations were more strongly associated with raw total mortality in bivariate analyses than was British Smoke or sulfur dioxide, despite the fact that acid data were reported from only one central site. However, once total mortality was prefiltered for slow-moving autocorrelations, the acid relationship appeared to be roughly similar in strength to that for BS and SO_2, once differences in pollution collection periods were accounted for. Moreover, the cross-correlation results indicated a roughly 1-day lag in pollution effects on mortality, and a 2- to 3-day lag in temperature effects, as well as overall trends for mortality to follow high pollution and low temperature values. As such, there appears to be evidence for both an association and a causality relationship between elevated pollution (including H_2SO_4) and mortality which are worthy of further research to quantify.

The apparent logarithmic nature of the H_2SO_4-mortality relationship supports the saturation model of pollution effects hypothesized by Dawson and Brown (*2*). This may be due to avertive behavior, especially since Macfarlane reports that episode warnings were publicized at the time of high pollution (*28*). Alternatively, Waller reported that, at moderate humidities (< 85%), all London acid droplets were in the submicron size range while, at higher relative humidities, acids were also found in the larger (nonrespirable) particle size range (*29*). Thus, not all of the measured acids during fog episodes would necessarily be respirable, reducing their health effects from that implied by the total H_2SO_4 concentration. Most likely, however, is that the saturation of effects is due to the harvesting of the pollution susceptible population on prior moderate pollution days. This would concur with the characterization of the affected population as "a susceptible group of patients whose life expectation, judging from their pre-existing diseases must, even in the absence of fog, have been short" (*10*). Thus, the entire curve of pollution effects would be expected to plateau as found here, but would likely rise again at some higher, as yet unspecified, level of pollution when the healthy population began to be severely affected by

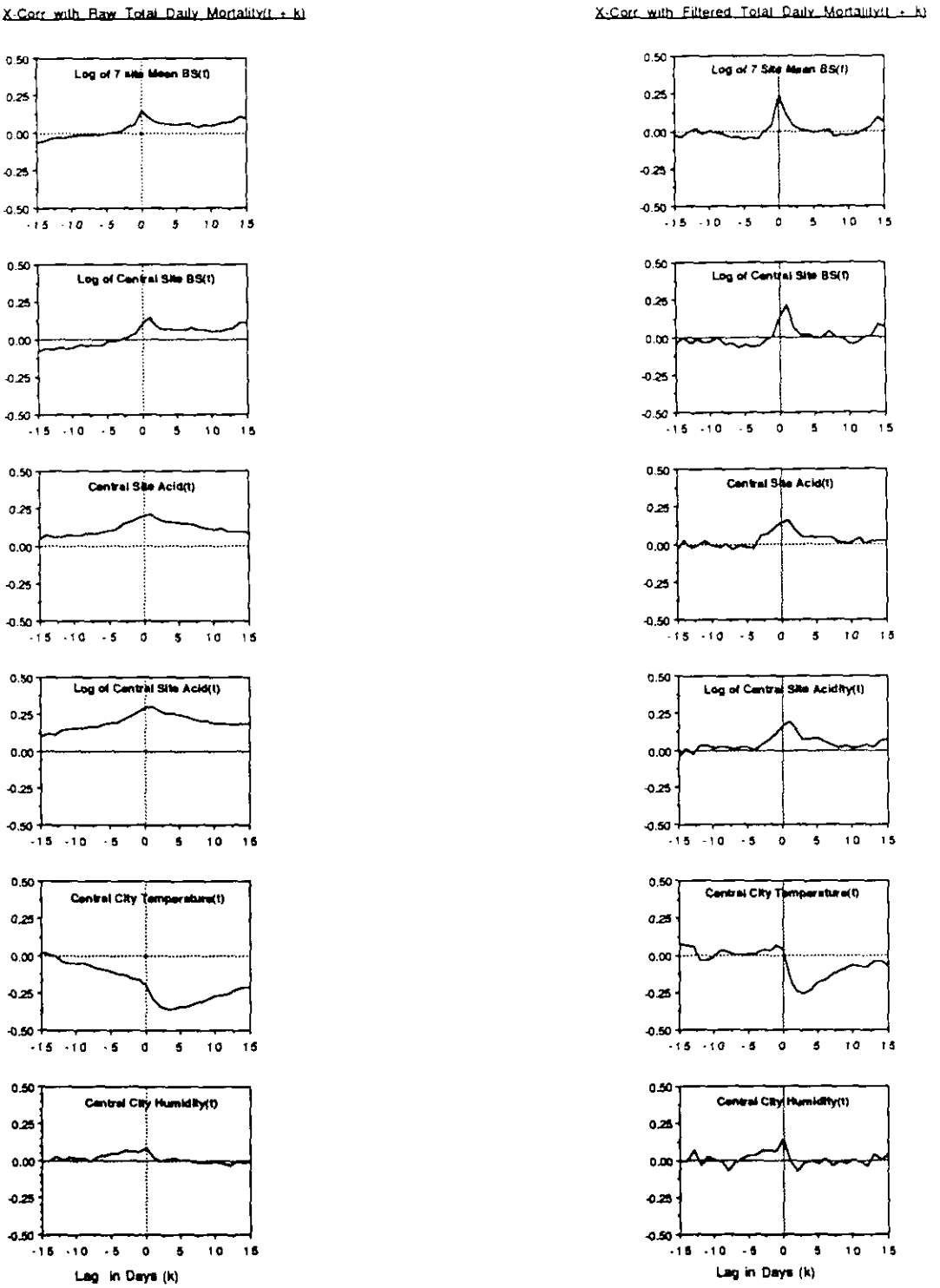

FIGURE 8. Cross-correlations (X-Corr) of key London pollutant and weather variables with raw and filtered total daily mortality, 1963–1972 winters ($n = 921$).

the pollution exposures. This implies that examination of, for example, the period of probably much higher H_2SO_4 exposures in the 1950s might well show the start of a second rising mortality trend at levels above the maximum level considered here (134 $\mu g/m^3$).

Future work will attempt to quantify the H_2SO_4-human mortality relationship implied by the analyses presented here. This is important because results for acid aerosols are directly applicable to other environments. British Smoke, in contrast, is an index of particulate mass which depends on the blackness of that

matter, and therefore varies in meaning as a pollutant index over time at a single site and (especially) between sites. Future research in this regard is expected to employ multivariate time series regression approaches to assess both the nature and size of the acid aerosol-mortality relationship indicated in this exploratory analysis of the London wintertime dataset. Furthermore, as part of this work, we expect to investigate the effects of various methods of controlling for autocorrelations in the data (e.g., via alternate prefilters or controlling regression variables) on the ability of such statistical models to discern and quantify an acid aerosol influence on human mortality.

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