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TO MORTALITY IN NEW YORK CITY"

A Critique

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Inge F. Goldstein, Dr. P.H.,
Columbia University School of Public Health, New York, N. Y. 9506450

Martin Goldstein, Ph. D.
Belfer Graduate School of Science Yeshiva University
New York, N. Y. 7027000

Leon Landovitz, Ph. D.
City University of New York Graduate Center
New York, N. Y. 9506449

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A Critique

Inge F. Goldstein, Dr. P.H., Martin
Goldstein, Ph.D., and Leon Landovitz, Ph.D.

In a paper in the May, 1976 issue of the Journal of Occupational Medicine entitled "The Relation of Air Pollution to Mortality" ¹ Schimmel and Murawski (SM) report new results in an ongoing study of the health effects of air pollution they and associates have been engaged in for some years. ^{2,3} The conclusion of this paper, based on their analysis of regression of daily mortality from New York City for the period 1963-72 on the pollution variables sulfur dioxide (SO_2) and smoke-shade taken at one aerometric station situated in Harlem, is that although there is some excess in daily mortality attributable to SO_2 , on the basis of the regression, SO_2 itself is actually harmless, but is associated with other as yet unidentified pollutants that are the real cause of the excess mortality.

We have previously criticized an earlier paper of this series ⁴ for what we felt are serious methodological weaknesses. As SM, in their new paper and in their response to our critique, do not deal adequately with the criticisms we have made, we feel it is necessary to repeat and amplify them. In addition, we would like to make some further criticisms of SM's procedure some of which we have outlined in a letter to the editor ⁵. In balance, we conclude that the uncertainties and ambiguities in SM's work do not justify the conclusions they have reached.

We will divide up our criticisms into four categories:

- 1) The use of a single air pollution station to represent the entire metropolitan area.

2) Methodological weaknesses in the linear multiple regression analysis

3) Handling of meteorological variables

4) Other criticisms

1. Use of a single air pollution monitoring station.

The most serious drawback of SM's study is the use of a single, centrally located air pollution measuring station to represent the pollution exposure of the population of a large metropolitan area. The errors and uncertainties introduced into SM's analysis due to this come in many ways and have not been adequately dealt with by them.

A) As one example, a crucial step in SM's reasoning is their belief that smokeshade, a measure of particulates, did not change over the same period of time in which sulfur dioxide decreased by 70%; on page 332 SM state "Our study has not shown a significant change in premature death estimates over a ten-year period in New York City despite a marked reduction in SO₂ levels with smokeshade remaining virtually the same." They were misled by the data from the Harlem station. The levels of smokeshade may not have shown a significant change at the Harlem station but decreased over the whole city from 1969 to 1974 by approximately 40% (This calculation was made from data published by the Department of Air Resources in New York City).

B) SM perform a regression of city-wide mortality on pollution variables measured at the Harlem station. As we have published earlier⁵ and pointed out in our previously published critique⁴ of an earlier paper by SM³, the correlation

coefficients for pollution measured at different stations average about 0.5 for SO_2 and 0.4 for smokeshade with a range from -0.6 to +0.9. Additional analyses and investigations of the problem of representativeness of the levels of air pollution in New York City by the forty-station aerometric network are described in two forthcoming papers in the Journal of Atmospheric Environment.^{7,8}

In answer to our previous criticism of SM for using one station for estimating pollution over the entire metropolitan area, SM refers in (1) to an unpublished appendix available from them on request, giving their reasons for having chosen to rely on pollution data from a single station only.

We have received from them this unpublished Appendix (F) and wish to comment on it as follows:

SM points out correctly that data from the entire forty-station aerometric network has been available only for half the period covered by their ten-year study, and even during the years data from the network was available it was very often incomplete. Thus, it would not have been possible to do their ten-year study using the network data.

Our point however, was not that they should necessarily have used the network data for what they wanted to do, but rather that the network data, incomplete as it is, reveals such poor correlation in daily levels of pollution between the central station used by them and the other stations of the network that the conclusions of a study based only on one station are subject to very serious uncertainties.

SM note, also correctly, in their Appendix, that "it would, however, be difficult to establish what would be the appropriate weights to be used in

combining the measures....taking into account population weighting, accounting for normal day-to-day mobility of the population and the fact that many deaths do not occur in the home," to which we would like to add the fact that demographic variables, and therefore mortality rates, would also confound the weighting according to area. We only fail to understand why SM think this is a disadvantage of a study based on the forty stations of the network, but not more seriously a disadvantage of using only one.

In their Appendix SM examine mathematically the errors of using a single station on the basis of what they call a simplified model. On the basis of their mathematical examination they estimate that the overall excess mortality in New York City as calculated by them from a regression of mortality on pollution at the central station can be in error as a result of the reliance on one station by $\pm 40\%$. In this estimate they have used data published by us on the correlation coefficients between the stations of the New York City network (Goldstein)⁵.

We believe that among the "simplifications" introduced by them in their estimation is one so serious it vitiates their analysis.

As their analysis is unpublished, we summarize it briefly:

SM assume that a more correct independent pollution variable would be the city-wide average P rather than the Harlem station measure of sulfur dioxide P_1 . They and we have pointed out drawbacks to their assumption (see above) but it does not strike us as an unreasonable one for an approximate calculation.

They call the regression coefficient they would have obtained if it had been possible to use the city-wide average P and the regression coefficient they

have actually obtained using only the Harlem station α_1 . The "correct" and the "observed" mortality estimates are thus:

$$\Delta M = \alpha \bar{P}$$

$$\Delta M^+ = \alpha_1 \bar{P}_1$$

The "correct" regression equation is

$$y = \alpha \sum x_j + \epsilon \quad (1)$$

where y is the city-wide mortality variate measured about its mean, and x_j is the pollution variate in area j measured about its mean.

The regression equation used in SM's study is $y = \alpha_1 x_1 + \epsilon$

In both equations ϵ represents the deviation from the regression.

The least squares estimate of α_1 is

$$\frac{\overline{x_1 y}}{\delta^2(x_1)}$$

(using an obvious notation) (2)

To obtain the error estimate SM substitute equation (1) in equation (2), but with the omission of the error term ϵ . This omission is presumably made for further simplification on the assumption that the error in so doing is negligible. Our point is that the omission is not negligible, but serious.* The result of their substitution is:

$$\alpha = \frac{x_1 (\alpha \sum x_j)}{\delta^2(x_1)} = \frac{\sum r_j \delta(P_j)}{\delta(P_1)} \quad (4)$$

where

$$r_j = \frac{x_1 x_j}{\delta(x_1) \delta(x_j)}$$

pollution between station j and station 1.

* correlation coefficient of

* An Appendix with mathematical details is available upon request from the authors.

For their estimate of the error in their mortality estimate they use the average r found by us (about 0.5) and the assumption that $\delta_j(P)$ is proportional to P_j for each district. Combining these estimates and including further estimates of their errors gives the $\pm 40\%$ figure referred to earlier.

Inspection of equation (4) reveals that it is asserting that the regression coefficient of a dependent variable (city-wide mortality) on one independent variable (city-wide pollution) can be calculated exactly from the regression coefficient on a second independent variable (pollution at the Harlem station) and knowledge of the correlation coefficient between the two independent variables. This absurd conclusion is a result of the neglect of the error term ϵ in equation (1). Its absurdity can be visualized easily by a vector argument: it is equivalent to the assertion that if we have 3 non-coplanar vectors, and know the angles between the first and second, and between the second and third, we can calculate exactly the angle between the first and third. It should be obvious that the most we can calculate are upper and lower limits for the unknown angle.

SM's upper and lower limits of $\pm 40\%$ do not arise because their analysis requires such upper and lower limits but because they have estimated the errors in the quantities that appear in equation (4). Their equation (4) does not require any such limits, as the correlation coefficients and standard deviations are available for many days of the study period; if they were used, an exact value of α would follow from the use of equation (4). This is of course an impossible result.

C) SM use as a test of the self-consistency of their analysis and results an earlier result obtained by Schimmel and Greenburg³, who made a comparison

of regression coefficients for various categories of mortality on air pollution for the whole city and on pollution in a "special area", a portion of the city surrounding the Harlem measuring station.

In Appendix F these earlier results are referred to and the statement made that "these estimates for the immediate surrounding data were approximately the same as those for the city as a whole on a proportionate basis." The Table in Reference #3 shows ratios of the regression coefficient for city-wide mortality to special area mortality (corrected for the population ratios) to vary from .04 (for respiratory diseases regressed on same day SO_2 concentrations) to 1.29 (coronary heart disease regressed on same day smokes shade concentrations) a difference by a factor of over 30. Both of these, it is to be noted, are conditions in which pollution effects on mortality might plausibly be expected, and they are the two largest contributions to SM's estimated premature deaths.

2. The Use of a Linear Multiple Regression Analysis.

SM used a linear multiple regression analysis to relate daily mortality in various categories to three explanatory variables: temperature, and two pollution measures: SO_2 and smokes shade. There are four criticisms we would like to make of this analysis as applied to the problem and data in question:

A) The use of a linear regression when there is more than one independent variable is of course a straightforward extension of this approach when there is one independent variable. When the explanatory variables (two pollution variables and temperature) are highly correlated, as is the case here, the regression coefficients found are uncertain in meaning, and fail to represent properly the actual relation between dependent and explanatory variables. SM are

aware of this but fail to recognize the shadow of uncertainty it casts on their conclusions. Statistical methods for dealing with multicollinearity of the independent variables are available but have not been employed by them in this study.

B) No consideration is given to sources of error in the explanatory variables themselves. In two papers ^{7,8} we discuss the poor correlation between pollution measured at pairs of stations, in New York City, and express our opinion that this poor correlation is probably not primarily due to experimental errors which are known to exist in the measurement but rather due to local meteorological conditions and the proximity of local sources of pollutants that make pollution at a given station a poor representation of pollution in the surrounding area. In turn this means that in effect the measured pollution, if regarded as a measure of population exposure, is subject to considerable experimental error. Matters are made worse when data from one station are taken as representative of a large metropolitan area (see point 12 below).

C) Although SM mention the reasonable probability that there is a threshold effect in the relation between mortality and pollution, they fail to deal with it. This could have been done by the use of non-linear terms in the regression.

D) SM failed to consider the possibility, widely suggested by other researchers, that sulfur dioxide and particulates might have a synergistic effect on health. To take interaction between the explanatory variables into account they could have investigated for example whether the product of sulfur dioxide and particulate measure ($P_1 \times P_2$ in their notation) correlates with mortality better than does either independently. Comparing two London fog episodes, during the

second of which mortality and particulates were only 1/6 of their values in the first episode but sulfur dioxide levels were the same, SM takes the difference to be evidence that sulfur dioxide has no effect on mortality. The facts are equally consistent with a synergistic model. The London episodes do not rule out either possibility. (In fact, a third explanation for the drastic reduction in mortality during the second London episode has been proposed, and is mentioned by SM; the medical authorities in London, after the experience with the first episode, advised people, in particular the aged and sick, to stay indoors and to avoid exertion. In addition, there were certain other differences between the episodes. Duration, humidity, prevalence of central heating differed substantially. All of these make a comparison of the two episodes much more complicated than is implied by SM.)

The failure of SM to take into account interaction between the independent variables, non-linear effects, the disregard of demonstrated large experimental errors in the independent variables, and the failure to use statistical techniques to deal with multi-collinearity of the independent variables introduce serious questions about the partitioning out of the effects of air pollution between the two pollutants. This partitioning of the effects between sulfur dioxide and smokeside is crucial to SM's conclusions. As pointed out in our previous critique the regression coefficients fluctuate widely in magnitude and sign, frequently with an error far larger than the coefficient itself. Taking these fluctuations in the regression coefficients and the size of their associated errors into account, we conclude that no statistically significant difference between the contribution of sulfur dioxide and smokeside to mortality for the period 1963-72 has been demonstrated by them.

3. Meteorological Variables

A) The complicating effect of weather both as having a direct effect on health or a synergistic one when combined with air pollution, and, simultaneously, as being responsible for changes in levels of air pollution, is not properly corrected for. In Dr. Schimmel's earlier paper (with Dr. Greenburg) he mentions having considered six other weather variables such as humidity, minimum and maximum temperature, and others, and concludes, "But mean daily temperature alone was used in the regression since the other weather variates had only second-order effects when temperature and pollution were held constant, as shown by partial correlation coefficients". This conclusion that all other weather variables may be disregarded is dubious when one takes into account some facts about the relationships between air pollution and weather, and weather and health. For example, humidity in the summer during a heat wave probably has a totally different effect on health from humidity in the winter.

Another example of the complicated relationship can be seen when looking at relative humidity and its relationship to air pollution. A very high level of relative humidity can be associated with precipitation, or just with very humid weather. Precipitation is known to cleanse the air of pollutants, whereas high levels of humidity without rain could very well occur with high levels of pollutants. Increased humidity is known to speed up the reaction between particulates and sulfur dioxide to form sulfates and sulfuric acid aerosols, both of which have been implicated in adverse health effects. Dr. Schimmel's failure to take these weather factors into account properly may lead either to an overestimate or underestimate of mortality due to air pollution.

It is our opinion that the best approach to deal with this problem of

separation between air pollution and weather in their effects on health is to use an epidemiologic method rather than statistical techniques. The Environmental Epidemiology Research Unit at Columbia University School of Public Health has been engaged in a study relating morbidity to air pollution. In this study we used data from the forty-station aerometric network of New York City as the source of air pollution data representing the local areas surrounding the stations. Two areas, Harlem and Bedford-Stuyvesant, were chosen for study. They are almost identical in social and demographic variables and may be presumed to have similar weather conditions, but according to data of the aerometric network, differ in day-to-day variations in pollution levels. To monitor health effects, daily visits for respiratory illnesses to hospital emergency rooms in these geographical areas have been chosen as dependent variables, thus controlling for the effect of weather and other possible confounding variables by epidemiologic design⁸.

B) The manner in which SM deal with the remaining weather variable, temperature, raises many questions, as they themselves point out repeatedly. They assume a linear dependence on temperature although, as they note, others have used non-linear models, which would seem to have a sounder scientific basis. Among the surprising results of their analysis are "that increased mortality is to be associated with the locally warmer days, even in the winter," and that "in July-August, the SO₂ share [of premature deaths from respiratory disease] reaches a level of -24% [for a regression on a 5-day pollution total][This] is to be taken as a statistical artifact of the relation between the SO₂ variable and the temperature variable".

C) The most serious weakness in their treatment of the temperature variable

appears, in our opinion, in their treatment of "heat waves". SM exclude "heat waves" from their analysis on the grounds that mortality in such period is due solely to an enhanced effect of temperature, there being "little excess in air pollution". In view of the increased electric demand expected during heat waves for air-conditioning purposes, it would be surprising if this were true. Indeed, elsewhere in their paper SM state "....[A] secondary peak in SO₂ has gradually developed during the last years of the study during the July-August period....The explanation for this appears to be the fact that the local utility has been carrying peak loads during the summer because of air conditioning and burning as much, and even more, fuel than during peak winter loads". (P. 325). Examination of Table 2 of SM reveals that in a majority of the "heat waves" excluded by them, the excess air pollution was appreciable. In 4 of the episodes the pollution dropped rather than rose, but in six out of the remaining eight episodes the percentage increase for at least one pollutant was larger than the percentage increase in total mortality (32-65%). This is not a claim that heat stress is not a major factor in causing excess in total mortality, but only that a possible significant effect of air pollution has been arbitrarily excluded from the analysis.

Such exclusion would probably not have been required if the dependence on temperature had not been assumed to be linear. SM state "The behavior of the temperature variable appears to be extremely complex in all seasons of the year. We have not fully understood its influence on our estimates of air pollution." (P. 331) We concur.

4. Other Criticisms

Other criticisms are as follows:

A) SM state as though it is an established fact that "air pollution mainly

affects those with pre-existing disease". This is possibly a reasonable inference for the acute effects of pollution, which are all that SM's study is designed to investigate. Chronic deleterious effects on people who would otherwise have been healthy will not be observed by this approach, but cannot therefore be ruled out when decisions such as the relaxation of standards are under consideration.

B) The use of daily mortality statistics to assess the adverse health effects of daily levels of air pollution is a poor choice of variables. In the author's own words, "It would thus appear that if the linear model has any validity, the estimates of excess deaths is an appropriate measure of the adverse health effects of air pollution". (P. 320) This statement constitutes one of the bases of SM's conclusion. We think they should not have extrapolated from conclusions based on a mortality study, which they admit is subject to considerable uncertainty in its conclusions about mortality, to conclusions about acute health effects generally.

C) There have been significant changes during their study in the demographic profile of the population of the City of New York - with an increasing proportion of the population in the lower and upper age brackets and in the lower socio-economic strata. These would be expected to be more susceptible to the effects of pollutants. Thus, the population on which the regression is based has not been stable.

In order to compensate for these changes, either (1) the mortality variate should have been adjusted for the above-mentioned effect in some clearly defined way, or (2) mortality by age group data should have been used. It would also have been of interest to find the effect of socio-economic composition of New York

City's population which has undergone changes during the study period.

D) The direct connection between SO_2 and the sulfates derived from it by atmospheric oxidation, together with the mounting evidence that sulfates are injurious to health, is a further unavowed confounding factor in establishing a relation between mortality and ambient SO_2 .

E) SM, after stating the conclusion they have drawn from their own study that SO_2 is not of itself a contributor to excess mortality, quote as supporting evidence statements by Swedish and British Medical Authorities and a recent review prepared at the request of the National Air Conservation Commission that SO_2 and smokes shade are pollution indicators and not necessarily the agents causing illness. SM's study, whatever its weaknesses, was an experimental study that looked for health effects of SO_2 and concluded they were absent. The various authorities quoted as supporting the same view were expressing the reasonable and cautious opinion that in the absence of evidential proof of harmful effects of SO_2 and smoke-shade it is best for the time being not to jump to the conclusion that they have toxic effects; their "conclusions" is thus not based on evidence but on an absence of evidence. It is therefore inappropriate for SM to cite this as supporting evidence for their own conclusion.

CONCLUSIONS

In view of the cumulative uncertainties in SM's analysis, it is not surprising to find, on inspection of Table VII of their paper, that the regression coefficients fluctuate wildly in magnitude, and the standard error often exceeds the regression coefficient itself. Such results cannot be said to inspire confidence in the firmly stated conclusions reached by SM on the basis of them.

SM state their conclusion as follows: "One of the major objectives of the study was to determine whether the sharp reduction in SO₂ levels which has occurred in New York has been attended by a concomitant reduction in adverse health effects. Our results have failed to confirm such a reduction." We feel the methodological weaknesses of SM's study are so severe that no such conclusion may safely be drawn.

We would like to make one extraneous comment in closing. In criticizing SM's conclusions we are not taking a position on the question of whether standards for SO₂ emissions should be relaxed at the present time. We recognize the complexities of the combination of health, economic, and political factors involved, and claim no special expertise that allows us to make any recommendation. We have concerned ourselves solely with the question: has SM's study established that SO₂ is innocuous? We suspect we and SM are in complete agreement that there is no sound experimental evidence from epidemiologic studies of the relation between air pollution and health to support the opposite conclusion either. We probably disagree with SM on the weight to be assigned to other kinds of evidence; e.g., toxicological or clinical studies, and strongly disagree on the interpretation and weight they give to their own study in reaching their conclusions. Decisions on standards will have to be made taking all relevant factors into account, of which health effects are only one; our sole point in this note is to state our view that SM's study has failed to establish a case for the harmlessness of SO₂.

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- 3) Schimmel H., and Murawski T. J. "SO₂ - Harmful Pollutant or Air Quality Indicator?". J.A.P.C.A. 25 (7): 739-4, 1975.
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