Letters

Air Pollution in Los Angeles

In "Carbon monoxide: Association of community air pollution with mortality" (16 Apr., p. 265), Hexter and Goldsmith report data showing that carbon monoxide (CO) is associated with increased mortality in Los Angeles County. They state that mortality follows a cyclic pattern, with maxima occurring in winter. By use of multiple regression technique they show a statistically significant association between observed ambient CO levels and mortality.

All primary contaminants found in Los Angeles smog (that is, pollutants which are emitted directly to the atmosphere, such as CO, sulfur dioxide, hydrocarbons, particulates, and nitric oxide) have a similar, cyclic concentration pattern, with maxima in winter. Nitrogen dioxide, an intermediate, secondary contaminant formed by photochemical oxidation of nitric oxide, also demonstrates a cyclic pattern of wintertime maxima because the less effective wintertime irradiation precludes completion of the photochemical reaction to form ozone and other more stable reaction products. During photochemical ozone formation the nitrogen dioxide disappears. The subsequent photochemical reaction products, such as ozone, which are formed during the reaction between nitrogen dioxide and hydrocarbon also have a cyclic concentration pattern, with the maxima occurring during the summer months when conditions for photochemical reaction are most favorable.

Our studies of data for the period corresponding to Hexter and Goldsmith's study, 1962–1965, show high degrees of correlation between ambient concentrations of CO and certain other atmospheric contaminants. For example, the coefficients of correlation between CO and nitric oxide, or nitrogen dioxide, are significant at a level of more than 99.9 percent ($P < .001$). Thus it is probable that nitric oxide or nitrogen dioxide would also have shown a significant correlation with mortality if the concentrations of these contaminants had been used by Hexter and Goldsmith in their model instead of CO. To single out CO when discussing atmospheric contaminant concentrations as associated with mortality is perhaps an oversimplification of this relationship, and may actually constitute a disservice to the community by emphasizing the relation between CO concentration and mortality while ignoring other suspects of equal or even greater statistical significance.

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In Hexter and Goldsmith's report one finds the statement, "The MSE [mean square for residuals] for the model with carbon monoxide is 192.0, a reduction of 36 percent [37.1 percent according to table 1] from the variance of the original observations." The reader must be familiar with multiple regression analysis and study table 1 to realize that the model *without* CO "explains" only 0.4 percent less (36.7 compared with 37.1 percent according to table 1) of the original variance.

The rationale behind the model is left largely unclear. Since all variables shown (in fig. 1: daily deaths, maximum temperature, and carbon monoxide concentration) were cyclic and of similar period there appears to be no reason for removal of the first few Fourier components of the dependent variable (daily deaths). Certainly one would have expected the independent environmental variables to "explain" a greater percentage of the original variance had those data not been removed. It also raises the question, what is the cause of the cyclic (seasonal) variation in daily deaths if it is not the cyclic variation of the environmental variables? The statement "In Los Angeles, there is a secular trend both for total mortality and for carbon monoxide concentration" also misleads rather than clarifies. The reader must work out for himself that the population of Los Angeles County also increased during this period and allow for the possibility that the mortality *rate* actually declined as the average carbon monoxide concentration increased. Since high daily deaths were associated with low temperatures, might not minimum or mean temperatures have been better temperature variables? Is not the effect of temperature on homoiotherms modified by relative humidity, wind velocity, and daytime illumination? One must admit the possibility that the reduction of variance achieved by carbon monoxide was due simply to its correlation with one or more of these unconsidered parameters. The relationships of carbon monoxide concentration to wind speed and inversion height (in turn related to stratus cloud, relative humidity, and illumination) are certainly systematic.

Since automobiles are the principal source of carbon monoxide in Los Angeles and since automobiles produce both carbon monoxide and traffic fatalities, might not this circumstance (rather than toxicity) be the source of the claimed association between carbon monoxide concentration and total mortality? (M. C. MacCracken, private communication).

This reader, at least, is left with the impression that all sorts of functions of the independent variables were cranked through the analysis and that that model was retained which maximized the regression coefficient for carbon monoxide, the whole exercise being performed only to "document" an a priori conclusion of the authors.

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Mosher *et al.* and Ellsaesser raise a number of important questions concerning our report. Both ask the key question, might not some other factor be responsible for the association observed between carbon monoxide concentration and excess mortality?

The "hidden variable" problem is always present in any statistical analysis, and there is always the possibility that the reduction in variance achieved by introducing CO was due to correlation with one or more unconsidered factors.

Mosher *et al.* state that the correlation of CO with nitric oxide (NO) and with nitrogen dioxide (NO_2) are both significant beyond the 99.9 percent level. From this they infer that nitric oxide or nitrogen dioxide would also have

shown a significant correlation with mortality. However, oxidant is also correlated with CO ($P < .00001$) but showed no association with mortality when substituted for CO or when added to the regression with CO. Our attention was directed to CO in preference to other primary pollutants because it is present in concentrations known to affect oxygen transport in the body. But we agree that NO, NO_2, sulfur dioxide, and the meteorological variables cited by Ellsaesser and others should be examined.

Minimum temperature can be ruled out; when substituted for maximum temperature in our regressions the mean square for residuals (MSE) and t for the CO coefficient were increased. Mean temperature gave a small decrease in MSE and in t, but this does not affect our conclusions.

Ellsaesser questions the consistency of our statement concerning reduction in variance with Table 1. The R^2 in the table is the ratio of the regression sum of squares to the total sum of squares, here $163,895/441,313 = .3714$. The reduction in variance for the regression is $(302.27 - 191.98)/302.27 = .3649$. These differ because MSE, a more conservative measure, takes into account the reduction in degrees of freedom resulting from the addition of terms to the regression.

Ellsaesser emphasizes that the model without CO "'explains' only" 0.4 percent less of the original variance than the model with CO. As stated in our report, the estimated contribution to mortality for Los Angeles County associated with CO may be a difference of as many as 11 deaths in one day, all other factors being equal. We do not think that contribution is trivial.

Ellsaesser is correct in stating that one would expect the regressor variables to "explain" a greater percentage of the original variance if the Fourier components of the dependent variable were not removed. But cyclic variables with the same periodicity will exhibit a nonzero correlation even if otherwise totally unrelated. To avoid this spurious association it was necessary to add Fourier terms to the regression. Otherwise, daily mortality in Los Angeles, uncorrected for the cyclic components, would be expected to exhibit a highly significant correlation with CO concentrations in Paris, hog prices in Quebec, maximum temperatures in Buenos Aires, or similar cyclic phenomena.

The observation that the mortality rate may have declined during the time that CO levels were increasing is correct but irrelevant. Analysis based on rates is both unnecessary and impossible—impossible because the required denominator data, classified by age, race and sex, are not available between census years, and unnecessary because analysis of the relationship between CO concentration and total mortality does not require knowledge of the denominator. We included trend terms to allow for population growth, changes in population structure, and secular changes in mortality. From the nature of regression analysis the trend terms provide a close fit at the beginning and end of the study period. Analysis of residuals for the model with trend, cyclic variation, and temperature removed showed no significant serial correlation, and a nonparametric test for fit (the "runs" test) was also negative. This indicates that no systematic deviation, such as would be caused by inadequate allowance for population changes, is present.

Ellsaesser suggests that automobile fatalities might be the source of the association. We looked at specific causes using the same regressions and found that most of the excess mortality is attributable to cardiovascular disease. The association of CO with automobile fatalities is actually negative; this is predictable, since CO concentrations are lowest on weekends when automobile accidents and fatalities are highest, although other factors also contribute.

With regard to Ellsaesser's final comment, perhaps we did not make our procedure sufficiently clear. We did indeed test a number of models before arriving at the regressions shown in our report. However, the testing was done *before* adding the CO and oxidant variables. The second of the three models shown provides an estimate of daily mortality which may be used in other applications of interest to us; development of this model gave us the opportunity to test CO and oxidant with little additional effort. We certainly had grounds for suspecting that CO and oxidant contribute to mortality. We found our suspicions confirmed for CO but not confirmed for oxidant.

We are well aware that demonstration of an association does not prove cause and effect. However, the maximum daily average for CO concentration observed in our study, 20 ppm, is sufficient to convert at equilibrium ap-

proximately 3 percent of the blood hemoglobin to carboxyhemoglobin, with reduction in the oxygen-carrying capacity of the blood. Since the pollutant data are basin averages, some individuals were exposed to substantially higher levels.

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American in Japan

I have just returned from my 20th or so trip to Japan to find on my desk Chalmers Johnson's review (5 Feb., p. 467) of my book *The Emerging Japanese Superstate*. . . . It seems to me that I am almost obligated to quote and comment on one remarkable and characteristic paragraph of the review (*italics are mine*). "This book seems to be inspired by Kahn's *two* visits to Japan during which he gave lectures at Kyoto-Sangyo University (*which he apparently does not know is more of a Japanese Rand Corporation than a university*) and where he had several meetings of several members of *one segment* of the Japanese establishment who understandably were quite pleased when Kahn *went home and wrote down what they told him* without doing any further checking."

1) I have been making about two or three trips a year to Japan since 1965. Each of these trips was for the dual purpose of information gathering and information giving and, as a result, I have had a great deal of contact with every aspect of Japanese society, including students, laborers, union officials, all the political parties, and so on.

2) Kyoto-Sangyo University is a university. It has none of the characteristics of a "Japanese Rand Corporation" other than those any university may possess. I happen to be (along with Arnold Toynbee, Raymond Aron, and Hubert Humphrey) a member of its academic advisory board and have been at the university many times.

3) As is clearly pointed out, the first chapter of the book summarizes not only the talks I gave at Kyoto University but also the argumentation in the book. It would not have been possible to get this argumentation from what was then my audience.

4) . . . As I also bring out in the book, we did a serious study in the mid-1960's on long-range Japanese prospects. The argument of the book