Public Health

BRONCHITIS AND THE ACIDITY OF URBAN PRECIPITATION

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The incidence of bronchitis is uncommonly high in Britain, especially in urban areas during winter; and it has been conjectured that air pollution is at least partly responsible. In this paper I report a correlation between bronchitis mortality and the acidity of urban precipitation.

The mortality-rates for bronchitis were calculated from the Registrar General’s annual statistical reviews for the whole populations of fifty-three county and Metropolitan boroughs in England, Scotland, and Wales over the five-year period 1950-54, and plotted on scatter diagrams versus air-pollution data from single stations and periods within these boroughs. These latter data were extracted from table 8 of the 27th report of the Department of Scientific and Industrial Research on the investigation of atmospheric pollution (1955), which provides figures for the monthly atmospheric deposit of tar, ash, other combustible matter and sulphate per unit area in urban districts, and also gives the pH of the precipitation. Of all these, only the last two showed any obvious relationship with the bronchitis death-rates, and a more detailed statistical treatment was then carried out, using in this case averages of all the pH and sulphate values given for the winter months September to March in each of the fifty-three boroughs. To obtain a more normal frequency distribution, and also a more linear regression, a logarithmic transformation of the bronchitis death-rates and of the sulphate deposits was employed.

The logarithmic correlation coefficient (r) for bronchitis and winter pH of precipitation amounted to −0.49, a value significant at the 1% level. The corresponding coefficient for bronchitis mortality and winter sulphate deposit, which is no doubt related to the production of sulphur dioxide from fuel combustion, amounts to +0.32, a value significant only at the 5% level.

To test further the above relationships, partial correlations of bronchitis mortality with the pH of winter precipitation and with the sulphate deposit were carried out, in order to eliminate the effects of any correlation between the two environmental variables. Following this procedure, the coefficient of correlation (first-order) between bronchitis mortality and pH of winter precipitation fell only slightly to −0.45, a value still significant at the 1% level. In contrast, the correlation with sulphate deposit declined considerably, the first-order coefficient of +0.24 being no longer significant even at the 5% level. It is therefore unnecessary to consider this last correlation further.

The partial regression equation relating bronchitis mortality (b, as deaths per 100,000 of population per year) to the acidity of winter precipitation (a, as pH) is b = 192 (0.838)a. This relation is shown graphically in the chart, from which it may also be seen that the logarithmic average of the annual urban death-rate from bronchitis is 82 per 100,000 persons, or about 1.8 times the rural death-rate for the same period. In those areas where rain pH is below 4, and hence strongly acid, the death-rate reaches 100, while it is only about 60 in those urban areas with the least acid rainfall. The rural death-rate for the same period is considerably lower even than this, at 46 annually per 100,000 of population; so acidity would not appear to be the only factor involved in the increased urban death rate, if in fact the acidity correlation is taken as truly causal. While such an interpretation must of course be regarded with caution, it seems not unreasonable to infer that long-continued exposure to acid aerosols might increase the likelihood of bronchial inflammation. In this connection it must be borne in mind that during dry weather the aerosol droplets will be considerably more acid than the ultimate raindrops in which they are precipitated, and may well exhibit pH values below 2 at times.

Even in predominantly rural areas the rainfall may be quite strongly acid. For instance, in the English Lake District rain pH averages less than 4.5, and goes below 4 on occasions, owing to atmospheric supply of sulphuric acid with easterly winds from the great industrial areas (Gorham 1958a). Surprisingly, however, in the industrial cities themselves hydrochloric acid seems likely to be the chief cause of low pH values (Gorham 1958b). The effects of both these strong mineral acids on bronchial tissues might well repay detailed investigation.

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REFERENCES
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Strontium 90 and Malignant Disease

A report in the New York Times of Sept. 20 refers to new work on the important question of whether or not there is a threshold level of 90Sr in bone, below which no bone cancer or leukaemia is induced. The work is that of M. P. Finkel at the Argonne National Laboratory in Lemont, Illinois, and it is described in detail in Science, in an issue which has not yet reached us. 90Sr was injected in varying doses into 810 mice, and the incidence of tumours and length of life in this group was compared with those of an uninjected control series. According to the New York Times, the results suggested that no damage was done by small doses—in other words, there was a threshold in mice. From observations in cats and dogs, however, Miss Finkel estimates that 90Sr is probably seven to fourteen times more harmful to man than to mice, because of his greater size and other factors. Her conclusion is that the threshold for man is so much higher than present fallout levels that fallout is extremely unlikely to induce even one bone tumor or one case of leukaemia.

On the assumptions that no threshold exists and that weapon tests stop this year, the recent United Nations report estimated that fallout radiation may produce 400 to 2000 cases of leukaemia a year. Assuming a threshold of 400 rem, the U.N. Committee’s figure for fallout leukaemia dropped to zero.

1. See Lancet, Aug. 16, 1958, pp. 355, 360

The relation between bronchitis mortality and acidity of precipitation in urban areas. M gives the logarithmic mean death-rate.