To the Editor

Respiratory illness in British schoolchildren and atmospheric smoke and sulphur dioxide

SIR—In a recent issue of the *Journal of Epidemiology and Community Health* Melia *et al* have reported on the analyses of a cross sectional¹ and longitudinal² study of the possible associations of atmospheric smoke and sulphur dioxide with respiratory illness in British school children.

Since my colleague, Raphael Toeplitz, and his associates are conducting an epidemiological monitoring programme in the vicinity of a large new coal-fired power plant and are collecting a somewhat comparable set of data in areas with varying likelihood of pollution exposures, we have studied and discussed these reports with unusual care and interest. Two questions have been of particular concern in our analysis; the evidence that exposures to one or a combination of these pollutants or changes in such exposures are associated with alterations in the incidence or prevalence of respiratory disease indicators in children, and the logical approach to the interpretation of trend data for respiratory conditions in relationship to changes in pollution exposures.

We are disappointed in the analysis of the first aspect and confused by the logical approach used for the second topic.

Specifically as to the first point, no apparent effort has been made to consider the joint effects of the two pollutants. The regression analysis represented by tables 5 and 6 of the cross sectional study,¹ although labelled to show that they referred to winter mean levels of smoke and SO₂, actually only show data for summer and autumn levels. In discussing the apparent association of respiratory illness and levels of smoke the citation of the "air quality standards of the World Health Organisation" is misleading. WHO has proposed criteria, not standards, and the joint presence of both smoke and SO₂ is proposed. Since they do not deal with the pollutants in combination, it is hardly reasonable to say the results "conflict with" the WHO review.

In the second paper on the longitudinal findings² by testing only whether in areas with decreases in pollution there is or is not a decrease in respiratory problems they have chosen an artificial criterion which we believe to be inappropriate for reasons discussed below.

(A) The treatment of the dependent data set gives equal weight to each of a heterogeneous set of answers (mother's self administered reporting of the child's morning cough, day or night cough, colds going to the chest, whether the child's chest ever sounded wheezy or whistling, and the number of attacks of asthma or

bronchitis experienced during the past 12 months). Surely, from the large number of respiratory surveys available, it would have been possible to have weighted these various responses by the likelihood of chronicity, impairment of lung function, or expected numbers of school absences. This over-simplification is confounded in both articles by the method of presentation; in the first we are given data only on one or more positive answers, or three or more; in the second paper we are given data only on the change in the number of positive answers.

(B) In the longitudinal study² the dependent variable increase, no change or decrease in the number of these conditions is distressingly heterogeneous. The largest proportion of children have no change, but this category may represent no change in children with from zero to six conditions. Surely the greater the number of conditions a child may have, the greater the likelihood of a decrease in the number of conditions and vice versa. Thus children with no change in a high number of conditions should be considered separately from no change in children with none.

Furthermore, children with six conditions in one year and five in the next are grouped together with children having one condition in one year and none in the following year; correspondingly, those with no conditions in the first year and six in the second are grouped with those who only increase from one to two or from zero to one. These are such heterogeneous groups that there surely must be more informative ways to present such data.

(C) There is no a priori reason to assume that if pollution is associated with increased respiratory conditions that a decrease in one year in pollution would necessarily be associated with a decrease in respiratory conditions in the same year.

(D) If the association shown in the first article¹ is present then in the high pollution area there would be more respiratory conditions during the first period; in subsequent periods, with no inherent change in risks, due to regression to the mean, we could expect a greater decrease in this high sector than in locations with lower initial frequencies. This problem has recently been treated by Davis³ and by Chinn and Heller⁴ from the authors' own unit. In the first article, as in that by Chinn and Heller, there is a demonstrable trend with age, which is not mentioned in the analyses of the second article.² Although the exact analysis of data such as they are dealing with may require normally distributed data, the use of a weighted score for respiratory conditions would permit the calculation of means, standard deviations, and correlation coefficients between the first and subsequent data sets. As shown in the articles by Davis³ and by Chinn and Heller,⁴ these suffice, along

with other accessible statistics, for the estimation of the contribution of the age trend and the regression contribution to the longitudinal change of a "high" subset which is being followed.

(E) Even if all of the analyses had been free of any defect the narrowness of the hypothesis being tested in the second paper—namely, that in the year when pollution decreases there is a decrease in the number of respiratory conditions which is greater in areas with the greater decrease in pollution, cannot justify the categorical conclusion that, "... the evidence suggests that the levels of pollution measured during the national study were not harmful to health." In the light of the findings in the first article¹ such a sweeping generalisation is unsupportable.

Other workers have subsequently reported significant but small declines in a longitudinal study of pulmonary function in third, fourth, and fifth grade children with increases in 24 hour TSP and mean SO₂ in episodes in a range of approximately 0 to 275 µg/M³.⁵ A large scale French study in its cross sectional component found a statistical association between "upper respiratory tract" symptoms in children and sulphur dioxide over a range of daily means of 22 to 85 µg/M³ (specific method) or 13 to 127 µg/M³ (acidimetric method).⁶

Surely the importance of childhood respiratory disease and the high stakes and long term planning necessary in reducing pollution which may or may not be related to childhood pulmonary disease deserves a more straightforward and logical treatment of these complex problems. Since the data collected are of great value and most of the suggested approaches seem feasible, we urge that a more convincing approach be made to its analysis.

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Dr Melia and coauthors reply:

The letter of Professor Goldsmith and Mr Toeplitz raises several points that ought to have been clear from our original texts.^{1 2} Clearly all the results could not be presented in detail but those published do, we believe, give a fair representation of the results as a whole.

The first point concerning tables 5 and 6¹ arises because we did not make it clear enough that summer and autumn referred to the season of the interviews. The pollution values used were winter levels, as stated. Analyses with a smoke × SO₂ product term—to test for synergistic types of effect—showed nothing. We should have mentioned that.

The remaining points will be answered using the identifying letters of Goldsmith and Toeplitz.

(A) Weighting to combine several variables into one is inevitably controversial. Our position is that the simplest approach will generally be the most interpretable and a count (equal weights of one) is certainly easy to understand. Without general agreement about coding the severity of respiratory measures, we might have been severely criticised had we followed the suggestion of Goldsmith and Toeplitz.

(B) The table of individuals according to their initial counts of respiratory conditions in 1973 and the changes which occurred by 1974 may help to clarify this point. The children generated paired data which essentially required a form of McNemar's test for the analysis.⁷ This uses the elements falling on either side of the "no change" column to generate a proportion for analysis on the logistic scale. Admittedly this means combining several different types of cell—for example, 6-5, 4-3, and so on—but these are not as heterogeneous as Goldsmith and Toeplitz feared. Furthermore, as may be seen from the table, individual analysis of the groups defined by the number of initial symptoms would have been impractical because of the small numbers concerned.

(C) Goldsmith and Toeplitz have a point. Nevertheless, if such changes had been found they would have been evidence for the cause and effect

Number of children who had a decrease, no change, or an increase in number of respiratory conditions between 1973 and 1974, by the number of conditions reported in 1973

Change in number of respiratory symptoms by 1974						
No of symptoms in 1973	Reduction of 2 or more	Reduction of 1	No change	Increase of 1	Increase of 2 or more	Total
0	0	0	516	63	13	592
1	0	66	64	12	5	147
2	19	14	21	11	3	68
3	11	5	9	2	1	28
4	9	2	1	2	1	15
5 or 6	1	3	3	0	0	7
Total	40	90	614	90	23	857