Correspondence

Coal and the lung

SIR,—The interest produced by Dr Anthony Seaton’s editorial “Coal and the lung” is demonstrated by the letters you have already received from various authorities in this field and by the criticisms they offered. Dr Seaton’s replies to these criticisms do not entirely satisfy me; perhaps I may be allowed to explain why.

Professor AL Cochrane’s comments (November 1983, p 877) depend on the fact that a self-selected sample constituting less than 30% of a defined population offers a large possibility of inbuilt bias. Dr Seaton seems to postulate that the remaining 70% would be more disabled than those examined, and that the effect Love and Miller reported would therefore be an underestimate. What is the evidence for this? Is it not equally possible that those not examined included disproportionately more of those fit enough to seek a change of employment, so that the effect was overestimated? Surely we must rely on the basic principles of epidemiology—“Whatever the size of sample that is being approached in the survey, it is essential that as near 100% as possible of the potential respondents participate.”

No final answers can come from samples which do not meet this requirement.

It should also be noted that Love and Miller adjusted for the effects of smoking by an additive factor, which would work if a particular total dose of cigarettes had the same effect no matter at what age it was reached. I think other evidence suggests that this assumption is too simple, and that some remaining effect of smoking could still be affecting the results.

It is pleasing to find that Dr Seaton too finds it difficult to remember which of the rather numerous papers from his institute contain what information. The reference he gives to Rogan et al in Inhaled Particles III pp 883–4 does not exist. A paper with the same title and authors appeared in the British Journal of Industrial Medicine. If this is what he is referring to, he says it claims that “even in those with category 0 radiographs it has been shown that higher dust exposures are associated with lower levels of FEV1.” But this is not so. In this paper a multiple regression equation fitted to the data on men in all radiological categories showed a significant dependence of FEV1 on dust exposure, smoking, age, height, and weight. Another, including also sitting height, was used to predict the FEV1 of an average man in each radiological category. The absence of marked differences between the predicted and observed means was used to deduce that radiological category did not in itself affect lung function. It was not demonstrated that among the 3005 men in category 0 FEV1 was related to dust exposure, as Dr Seaton implies.

Inhaled Particles III (1971), pp 883–94, contains a paper by Rae et al on chronic bronchitis and dust exposure in coalminers, which does not discuss FEV1 at all but shows a rather rough correlation between dust exposure and percentage with chronic bronchitis in men with category 0 radiographs in one out of four age groups, the 35–44 age group. In the non-smokers the tendency is in fact for a negative correlation in the other three of the four age groups, and in the 35–44 age group it is only the high proportion in the men with the highest dust exposure that generates any appearance of a trend. The authors comment that a much larger sample is needed before firm conclusions could be drawn, and warn that their estimates of dust exposure include “rather speculative calculations.”

I hope that Professor Cochrane will not “now concede that his careful studies have been superseded.”

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This letter was sent to Dr Seaton, who replies below.

SIR,—Thank you for giving me the opportunity of replying to Dr Oldham’s letter. Before doing so I have discussed his points with Dr Michael Jacobsen, who has been responsible for much of the planning and statistical analysis of the Coal Board’s research.

In my original article (April 1983, p 241) I referred to the paper by Love and Miller cited by Dr Oldham as showing that coalmine dust exposure accelerates the age related decline in FEV1. In his letter in November Professor Cochrane criticised that work on the grounds that the sample of men studied did not include those who had left the collieries concerned before the surveys were made. In reply I pointed out (p 878) that the authors had discussed this very problem and I suggested that any bias is likely to have resulted in an understatement rather than an overstatement of the effect of dust. Dr Oldham asks for evidence to support this conjecture. I have sent him a preliminary report of the more recent work to which I referred, which shows that miners who had left the collieries we have been studying before retirement tended to have slightly lower age standardised levels of FEV1, and showed a somewhat more, rather than less, severe average dust related decrement in level of FEV1, than men who remained at the mines throughout the study period.

Love and Miller also anticipated Dr Oldham’s second point, about the way that they chose to adjust for the effect of smoking. They reported that they had tested the validity of an assumption implicit in their analysis, that age related rates of change in FEV1 were the same among non-smokers and various smoking categories. They concluded that there was no evidence in their data to contraindicate the approach used. Soutar and colleagues also considered
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PD Oldham

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