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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[1] 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."[2] No final answers can come from samples which do not meet this requirement.

It should also be noted that Love and Miller[1] 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*.[3] 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 FEV_1." 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 FEV on dust exposure, smoking, age, height, and weight. Another, including also sitting height, was used to predict the FEV 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 FEV 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,[4] which does not discuss FEV 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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[1] Love RG, Miller BG. Longitudinal study of lung function in coalminers. *Thorax* 1982;**37**:193–7.
[2] Alderson M. *An introduction to epidemiology*. London: Macmillan, 1976.
[3] Rogan JM, Attfield MD, Jacobsen M, Rae S, Walker DD, Walton WH. Role of dust in the working environment in development of chronic bronchitis in British coal miners. *Br J Ind Med* 1973;**30**:217–26.
[4] Rae S, Walker DD, Attfield MD. Chronic bronchitis and dust exposure in British coalminers. In: Walton WH. ed. *Inhaled particles III*. Old Woking, Surrey: Unwin, 1971:883–96.

**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 FEV_1. 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 between the surveys when the measurements 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[1] 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 FEV_1 and showed a somewhat more, rather than less, severe average dust related decrement in level of FEV_1 than men who remained at the mines throughout the study periods.

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 FEV_1 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[1] also considered

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this matter. These authors note that in the context of their (cross sectional) analysis of levels of FEV_1 (as distinct from rate of loss in FEV_1 with age) allowance for an age dependent smoking effect explained more of the total variability in their data than a simple additive model. They add, however, that the relationship between dust exposure and FEV_1 was hardly altered by the way that smoking was taken into account. The latter finding is consistent with the results of other work,[2] which show that the significant effect of dust exposure on FEV_1 in coalminers who smoke is statistically indistinguishable from the dust effect evident in miners who do not smoke.

Dr Oldham is right in his assumption that the paper by Rogan *et al* which I mentioned is that published in the *British Journal of Industrial Medicine* (his reference 3) (and I am sorry about the error in my earlier lists of references). He is right also to point out that the analyses described in that paper did not, on their own, demonstrate the association between dust exposure and FEV_1 separately in the 3005 men with category 0. However, Dr Jacobsen, a coauthor of the paper, has in subsequent work on the same data confirmed the significant relationships between dust exposure and FEV_1 in the 3005 men in category 0 (as well as in 2272 with no bronchitic symptoms, as first reported in 1973[3]). The negative gradient is evident and unlikely to be explicable by chance factors ($p < 0.01$) even in a small subgroup consisting of 346 life long non-smokers with radiographs classified as category 0/– or 0/0 who did not report having persistent cough and phlegm. A publication is being prepared giving details of these more recent studies of the material first reported by Rogan and his colleagues.[3]

I did not mention respiratory symptoms in my article or in the reply to comments, but since Dr Oldham raises the matter it is worth making two observations. Firstly, he chooses to discuss only one of three tables that show an association between dust exposure and the chronic cough and sputum syndrome in the two younger age groups studied by Rae *et al* (reference 4 cited by Dr Oldham). Secondly, both of the comments that he quotes from the paper are in the context of warnings that the apparent absence of the association in older men should not be interpreted as indicating that the effect of dust on symptom prevalence is minimal or non-existent.

Of course, no single report or study can provide the final answer to complex questions of this kind, and Dr Oldham is right to remind me of the caution required when attempting to draw conclusions from data subject to the selection effects so common in studies of industrial groups. In 1966 Professor Cochrane wrote "It is, I think, possible that if we could measure dust dosage more accurately then the correlation with respiratory symptoms and ventilatory function would be much more exact."[3] Those more accurate measures of dosage have become available during the last 14 years as a result of the Coal Board's research. I was hoping that Professor Cochrane and his colleagues might now agree that the evidence accumulated vindicates his suggestion of 18 years ago. The correlations are now much more exact.

There is a danger that legitimate debate on the minutiae of individual reports will obscure an important point I was anxious to make. This was that a lot of new information is available now about relationships between direct measures of miners' exposures to dust and risks to their health, including the risk of developing disabling airways obstruction. That information has provided a rational basis for dust control strategies designed to protect the men's health.

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[1] Soutar CA, Hurley JF, Gurr DC. Relationship between dust exposure and lung function in miners and ex-miners. In: *Proceedings of the VIth International Conference on Pneumoconiosis, Bochum 1983*. Geneva: International Labour Office (in press).
[2] Jacobsen M. Occupational health risk assessment. In: Hester RE, ed. *Industry and the environment in perspective*. London: Royal Society of Chemistry, 1983:179–89. (Special Publication No. 46.)
[3] Cochrane AL. Chronic bronchitis and occupation. *Br Med J* 1966;i:858–9.

Persisting "asthma" in tropical pulmonary eosinophilia

SIR,—We were very interested in the report by Dr DA Jones and his colleagues (September 1983, p 692) of tropical pulmonary eosinophilia simulating classical asthma in a 37 year old Indian man.

We met the same problem in a 4 year old Indian girl who had been resident in France for one year. She was referred to our paediatric chest clinic with the diagnosis of asthma of two months' duration. After four months of oral bronchodilator treatment only partial improvement had been achieved and our attention was drawn to the unusual intensity of inspiratory crackles audible in the chest and to a leucocytosis varying from $8{\cdot}2$ to $22{\cdot}4 \times 10^9$/l with 24–60% of eosinophils. The total serum immunoglobulin E concentration was 940 IU/ml (PRIST Phadebas). The chest radiographs were reviewed and found to show diffuse bilateral miliary mottling, predominant in the mid and lower zones. The filarial fluorescent antibody (FFA) test gave a positive result with a titre of 1/12 800. Specific dynamic compliance was decreased (48 ml/cm H_2O/l at functional residual capacity; normal = 68).[1] Intradermal skin test responses to common inhaled allergens were negative.

During treatment with diethyl carbamazine in a total dose of $2{\cdot}1$ g (123 mg/kg) over 22 days, the serum IgE concentration peaked at 19 900 IU/ml and was still raised at 1100 IU/ml one year later. One month after completion of treatment the result of the FFA test was positive with a titre of 1/200, but specific dynamic compliance had returned to normal values. Chest radiographs showed clearing of the mottling six months after treatment had started, although blood eosinophilia persisted ($1{\cdot}1 \times 10^9$/l). Dyspnoea disappeared within a few weeks of starting treatment with diethyl carbamazine and bronchodilator drugs were discontinued. There has been no recurrence of symptoms during the three years that have followed.

Tropical pulmonary eosinophilia is exceedingly rare in

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