Correspondence

Coal and the lung

SIR,—The interesting editorial (April 1983, p 241) on coal and the lung by Dr Anthony Seaton brings us up to date with current theories on the development of benign simple pneumoconiosis and massive fibrosis. There would be general agreement with his statement "Much of the variability in prevalence of pneumoconiosis in different parts of Britain is likely to be related to differences in total exposure to dust and in the composition of that dust."

I must, however, disagree with his views on coal dust, emphysema, and disabling airway obstruction. He states that "The effect of the highest dust exposures, two or three times that which man would now be exposed to if he worked continuously at the highest allowable levels for the whole of his working life, was roughly comparable with the average effect of being a cigarette smoker." Although there is a clear relationship between measured exposure to respirable dust and both the FEV and the decline in FEV, it does not necessarily follow that high dust exposures are responsible for the emphysema in coalminers. When there are high levels of pollution with dust there are likely to be high levels of nitrous fume from shotfiring in the mine air, especially in poorly ventilated areas.[1]

Many postmortem studies over the past 50 years have reported an excess of emphysema in coal miners.[2–5] The recent pathological study from Cardiff of miners and non-miners dying of ischaemic heart disease confirmed that this excess occurs even after age and smoking habits are taken into account.[6] In their earlier studies the Cardiff group found a very high incidence of emphysema in 247 coalminers compared with their matched non-miners.[7,8] The extent of the anatomical emphysema was closely related to a reduced FEV during life, but was not related to the radiological category of pneumoconiosis in either their study or an earlier study.[9] Thus it seems improbable that the dust alone is responsible for emphysema except perhaps in cases of massive fibrosis with scar emphysema. Although "distensive," dust-pigmented centrilobular ("focal") emphysema is often a feature of the lungs of coal and carbon workers[10] it is not an essential feature since it is not always or uniformly present and it is also found in the general population.[11]

There is no animal work to suggest or confirm that high dust levels cause emphysema, but extensive animal toxicological studies have shown that exposure to low levels of fumes such as occur in mines results in emphysema.[12–15] Furthermore, there is good evidence that acute poisoning from nitrous fumes may occur in coalminers[16] and chronic poisoning associated with emphysematous changes is not uncommon.[17,18]

There is no good reason for stating that coal dust alone causes emphysema but there is good evidence that carbon dust[15] or coal dust may act as a carrier of fume. Both tobacco smoke and mine air contain nitrous fumes and the carbon particles of tobacco smoke and the coal dust of mine air may be important transporters of concentrated fume to the terminal airways of the lungs. It is encouraging that the level of dust in the mines is diminishing but more needs to be done to discourage cigarette smoking in miners and to reduce the nitrous fume content of mine air.

The hypothesis that dust causes emphysema in coalminers may not be entirely wrong, since dust can be a contributory factor as a carrier of fume. Whatever the cause, it is true that coalminers suffer emphysema more often and to a greater degree than non-miners, and arrangements for benefit for the more seriously disabled should be simplified.

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SIR,—Although I agree with most of Dr Anthony Seaton's editorial in the April issue and admire the work of his institute, I must object to his claim that the question of whether exposure to coal dust increases a man's risk of developing emphysema and airways obstruction has been finally answered by the paper by Love and Miller.[1]

The main criticism of Love's paper arises from the nature of the material available to the authors. Ideally, to answer the question in which they were interested, an 11 year follow up study of a well defined population of miners and ex-miners would be necessary. This was not available, so they made the best use they could of the three cross-sectional studies of working miners. However, the brute fact remains that only 1677 of the original 6191 were re-examined. After we have allowed for reasonable exclusions the re-examination rate is 28·7%. The chance of this population being representative of the original population is small and the possibility of inbuilt bias large.

Two other points can be made. The authors themselves only claim that all their variables, including "dust exposure," explained 6% of the variance and their results do not seem compatible with the Medical Research Council's careful epidemiological studies summarised at the Royal Society of Medicine meeting.[2] I think that we shall have to wait some time for the final answer.

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SIR,—Unfortunately a number of unwarranted inferences are likely to be drawn from Dr Anthony Seaton's editorial "Coal and the lung" (April 1983, p241). It is clear that coal mining is associated with a reduction in ventilatory capacity and that the latter is independent of the presence of coalworkers' pneumoconiosis.[1 2] Dr Seaton quotes a paper that states that in the past the highest dust exposure had roughly the same effect on FEV_1 as cigarette smoking.[3] Such a comparison is misleading. Unfortunately the National Coal Board questionnaire records current smoking habits and not pack years. To compare the effects of maximal dust exposure to those of average cigarette consumption is inappropriate, and it would be more relevant to compare the rate of decline in the FEV_1 in the most dust exposed to that of the heaviest smokers. Moreover, airways obstruction develops in only 13–15% of cigarette smokers.[4] Thus the paper by Love and Miller[3] compares the effects of cigarette smoking, which induces significant obstruction in a minority, with that of dust, which induces minor obstruction in the vast majority of those with prolonged exposure.[2 5] This is a specious and misleading comparison.

Similarly, Love and Miller deliberately selected older subjects with a long history of dust exposure.[3] Since it is known that cigarette smoking reduces life expectancy by about 11 years, and also leads to premature disability,[6] then the loss to the study from death and disability will have been significant. In contrast, there is no evidence to suggest that dust is similarly lethal, and indeed in one prospective study of coalminers, in which cigarette smoking had a significant effect on the standardised mortality ratio, no discernible effect was apparent from increasing years spent underground.[7] The population studied by Love and Miller was therefore composed of a survivor group resistant to the effects of smoking.

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***These letters were sent to Dr Seaton, who replies below.

SIR,—Dr Kennedy has often made the point that he believes nitrogen dioxide to be an important cause of emphysema in coalminers, though readers of his paper on the subject[1] will not, I think, be wholly convinced by the case he made. Our recent work has shown a relationship between measured exposure to respirable coalmine dust in life and the risk of having centriacinar emphysema post mortem in men who also showed a fibrotic reaction to dust. In addition, it has been possible to show a similar relationship between risk of emphysema and lung coal content. In both cases the risk was reduced with higher levels of quartz and other silicates in the dust. This work has recently been submitted for publication. We do not have figures for exposure to oxides of nitrogen in this study, but we have recently investigated the relationship between exposure to these oxides, respiratory symptoms, and FEV_1 in coalminers.[3] We find that current levels of the gases, as Dr Kennedy would wish, are low and we are unable to demonstrate any relationship between exposure to oxides of nitrogen and respiratory symptoms or FEV_1.

I do not dispute that in the 1960s there were times when dangerously high concentrations of nitrogen dioxide may have followed shotfiring, and vigilance is clearly necessary to ensure that this does not recur. Whether the present low levels of nitrogen dioxide in coalmines play any part in the causation of "emphysema" must remain an open question. If they do, the effect on FEV_1 from our recent studies seems to be so small as to be undetectable—in any case it is less than the effects of cigarette smoking and coal dust exposure.[2]