

## Editorial

# Update on lung disease in coalminers

The best known disabling chronic lung diseases of coalminers are progressive massive fibrosis, a wholly occupational disease, and chronic airflow obstruction, a partial one. This editorial briefly reviews recently completed studies that have furthered our understanding of progressive massive fibrosis and have enabled direct estimates to be made of the risk of the disease in relation to exposure to measured concentrations of respirable dust. The influence of exposure to quartz on pneumoconiosis in coalminers and the importance of irregular opacities are considered. The evidence that airflow obstruction in coalminers, long the subject of debate, does result from exposure to respirable dust, and that dust related lung functional impairment may be severe in some individuals is also discussed.

Much of the work quoted here is based on British Coal's pneumoconiosis field research and other studies carried out by the Institute of Occupational Medicine. The purpose of this research programme is to provide quantitative information on the risks of respiratory disease in relation to airborne dust, on which preventive measures may be based. The main strength of this research and the reason it is quoted extensively here lies in its extraordinarily detailed long term programme of measurements of airborne dust concentrations, which, linked to similarly detailed, and compatible, occupational records enable precise estimates of individual cumulative exposures to respirable dust to be calculated. By this means the demonstration of dust exposure/medical response relations has advanced our knowledge of the qualitative and quantitative effects of airborne dust.

### **Progressive massive fibrosis**

Progressive massive fibrosis (complicated pneumoconiosis) of coalworkers is associated with breathlessness,<sup>1</sup> impaired lung function,<sup>2,3</sup> and increased mortality.<sup>4-7</sup> In the coal industry the strategy of prevention of progressive massive fibrosis has been based on the observation by Cochrane that the condition is much more likely to occur in men who already have simple pneumoconiosis than in those without.<sup>8</sup> Since the risk appeared to be predominantly in men with category 2 or 3 simple pneumoconiosis, and much less in men with category 1, dust control measures have

been directed towards reducing the likelihood of a man developing category 2 simple pneumoconiosis in the hope that this would also reduce the incidence of progressive massive fibrosis. Airborne dust control regulations intended to prevent category 2 simple pneumoconiosis are based on estimates of dust/disease relations established by British Coal's pneumoconiosis field research.<sup>9,10</sup>

These dust control measures have been successful in reducing the prevalence of both simple pneumoconiosis and of progressive massive fibrosis. Although new cases of both these do still occur, the prevalence of category 1 or greater simple pneumoconiosis and of progressive massive fibrosis in the current workforce of a selection of British collieries in 1984 were 1.5% and 0.1%, respectively, compared with 8.3% and 0.8% in 1962.<sup>11</sup> Whereas selective loss of men<sup>12</sup> partly due to contraction of the industry has contributed to this change, there is strong evidence also of a decline in incidence of new cases of simple pneumoconiosis.<sup>13</sup> This represents a considerable achievement in disease prevention.

The estimates of risk of simple pneumoconiosis were based on studies of working miners, and since men may leave work on account of ill health, it was not known until recently whether the estimates properly represented the risks in all miners, including those who leave the industry. New information on this has been provided by a follow up study of British miners who were working in the industry in the 1950s and were restudied 22 years later whether or not they were still working in mining. Men who left the industry did indeed have higher frequencies of pneumoconiosis than men who stayed,<sup>12,14</sup> confirming that ill health was one reason for their leaving. The relations, however, between cumulative exposure to respirable dust and risk of simple pneumoconiosis were similar in men who left and men who stayed,<sup>14</sup> confirming the validity of the previous estimates of risk.<sup>10</sup>

This study also showed that men may develop progressive massive fibrosis for the first time after leaving the industry,<sup>10</sup> whereas simple pneumoconiosis appears not to alter much on aggregate after exposure to dust has ceased.<sup>12</sup> In this and another study<sup>15</sup> the risk of men with category 1 simple pneumoconiosis developing progressive massive

fibrosis appears more substantial than shown in previous work.<sup>8,16</sup> Furthermore, it has been pointed out that whereas the incidence of progressive massive fibrosis is low among miners with little or no simple pneumoconiosis, the large size of the group means that it contributes about half of the cases of progressive massive fibrosis that do occur.<sup>17</sup> This evidence suggests that the scientific basis for the prevention strategy (based on reducing the incidence of category 2 simple pneumoconiosis) is not as reliable as was thought.

A direct estimate of the risk of progressive massive fibrosis in relation to measured exposure to respirable dust would enable a prevention programme to be more soundly based, and fortunately a study of this condition in British miners has just been completed. Much information on the factors influencing the development of progressive massive fibrosis has been published in report form,<sup>15</sup> and from this work an estimate of risk in relation to cumulative exposure to respirable dust has been made.<sup>18</sup> A further study is planned of the reasons why this estimate indicates that the risk of PMF in response to a range of exposures to mixed respirable dust is slightly greater than the previously estimated risks of developing category 2 simple pneumoconiosis.<sup>10</sup> When fully understood, this new information may lead to a review of the prevention strategy.

### **Quartz and pneumoconiosis**

The average concentrations of quartz in the respirable dust in British coalmines are generally less than 10%, and in these conditions quartz has not been shown to influence the risk of simple pneumoconiosis over and above the risk attributable to exposure to mixed dust.<sup>10</sup> There are some exceptions to this which suggest, not surprisingly, that quartz may be important when its concentrations in the airborne dust are much higher than the average. For instance some men in one colliery where quartz concentrations in the coalface dust were unusually high as a result of adverse mining conditions have developed an unusually rapidly progressive pneumoconiosis.<sup>19</sup> Another study of men whose simple pneumoconiosis progressed unusually rapidly, in many cases to progressive massive fibrosis, indicated that exposure to quartz had contributed to their disease.<sup>10,20</sup> The role of quartz is being further investigated by attempts to distinguish by the chest radiographic appearances between quartz related and coal related progressive massive fibrosis<sup>21</sup> and by a study in progress of exposure/response relations in miners who have been exposed to unusual proportions of quartz in the respirable dust. Quantitative information on safe levels of airborne quartz when the por-

portion of quartz in the dust is high is urgently needed to aid decisions on dust control limits.

### **Irregular opacities**

The ILO scheme for recording the chest radiographic appearances of pneumoconiosis distinguishes between small opacities that are rounded in shape and those that are irregular.<sup>22</sup> Until recently, studies of coalworkers' pneumoconiosis have considered only rounded opacities, though there is now evidence that irregular opacities are also related to dust exposure.<sup>23-25</sup> These opacities differ from rounded opacities in that they are related to an impairment of lung function,<sup>23,26,27</sup> and have been shown to be related to the presence of emphysema and fibrosis in necropsy material.<sup>28</sup>

Since disease prevention strategy is based on the relations of small rounded opacities with dust exposure, this new information on irregular opacities could have important implications. Further studies of irregular opacities are in progress.

### **Chronic airflow obstruction**

The longstanding debate on "occupational bronchitis" (or "industrial bronchitis") refers to the chronic inflammatory or degenerative effects of dust on the lung, excluding pneumoconiosis. These processes are usually assessed by such features as symptoms of chronic bronchitis, measurements of lung function, and necropsy studies of emphysema or mucous gland hypertrophy.

An important chapter in the debate has been completed by a series of studies in which features of chronic inflammation or degeneration of the lung in several different industrial populations have been compared with quantitative estimates (based on detailed measurements) of lifetime individual exposures to airborne dust. These, summarised in a recent review,<sup>29</sup> include several separate cross sectional studies of British coalminers,<sup>30-32</sup> South African goldminers,<sup>33</sup> and United States<sup>34</sup> and West German<sup>35</sup> coalminers. All confirm that exposure to respirable dust is related to a reduction in lung function, even after allowing for the effects of smoking. Additionally, two longitudinal studies of change of lung function have shown that exposure to dust accelerates the loss of function occurring with age.<sup>36,37</sup> It would take the most determined sceptic to refuse to acknowledge this weight of evidence that exposure to dust impairs lung function. Another study has shown that symptoms of chronic bronchitis are also related to dust exposure.<sup>38</sup>

The next chapter in the debate, on whether the effects of dust are ever severe enough to be disabling,

is being written at present. There is already some evidence for an affirmative answer. A small group of British coalminers has been identified who showed severe effects of dust exposure on their lung function, a deficit of the order of 600 ml of FEV<sub>1</sub> for a moderately high lifetime exposure to dust.<sup>39</sup> Severe effects of dust are confirmed by a large mortality study of British miners,<sup>7</sup> which showed that death from respiratory disease was related to lifetime exposure to respirable dust, not only for pneumoconiosis but also for the certified causes "chronic bronchitis and emphysema." Whereas the diagnostic difficulties of death certification are well recognised, it is highly probable that the causes of death in the latter categories were from chronic inflammatory and degenerative conditions.

Thus there is already some good evidence that the chronic inflammatory and degenerative effects of dust on the lung may occasionally be severe enough to cause disability and premature death. Studies in other industrial populations might help to confirm these results. Important questions which remain to be answered include the further characterisation of these effects of dust in terms of clinical features, pathology, and pathogenesis, and what measures are required to prevent men from being disabled.

In general, dust exposure appears to affect the forced vital capacity at least as much as the FEV<sub>1</sub>,<sup>29 31 40</sup> a different pattern of abnormality from the classic airflow obstruction induced by smoking. This suggests an element of a restrictive lung defect as well as airflow obstruction. Some workers have suggested that airflow limitation occurs in the upper airways, but their data also show a parallel reduction of FEV<sub>1</sub> and FVC.<sup>40</sup> Men, however, who show severe effects of dust exposure,<sup>31 39</sup> most of whom have smoked, have severe airflow obstruction, and in many cases are clinically indistinguishable from men whose disease is entirely attributable to smoking.

There is an incomplete picture of the underlying pathological abnormalities which account for these functional defects. A necropsy study has shown that emphysema is more common in miners than in a comparable population of non-miners,<sup>41</sup> confirming the conclusions of an earlier much criticised study,<sup>42</sup> and another has shown that the likelihood of the presence of centrilobular emphysema in coalminers' lungs is related to exposure to respirable dust.<sup>43</sup> Yet the pattern of the dust related functional defect would suggest that one or more other processes occur in addition to the emphysema, to account for the restrictive component of the defect. Perhaps such processes should be sought in the region of the respiratory bronchiole, where abnormalities have been described in workers exposed to mineral dusts other than coal.<sup>44</sup>

It is probable that dust control measures designed to prevent pneumoconiosis also aid the prevention of occupational bronchitis. Studies in progress will enable the estimation of exposure/response relations for lung function which will help to review the adequacy of present dust control measures. A minority of individuals who are extremely susceptible to the effects of dust, however, may be difficult to protect by general measures, and it would be helpful to identify these individuals before they have developed severe functional impairment. It has already been shown that miners with unusually rapid loss of function may be identified by serial spirometric measurements, before they become frankly abnormal,<sup>45</sup> and the best adjunct to general dust control measures would seem to be serial measurements of lung function in all men in dusty occupations to identify and advise those who appear to be suffering from an unusually rapid decline of function.

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