

Most of the men in the study had been employed elsewhere before starting work at the factory; only 40% started before age 30. Of the 13 men in the post-1950 group who had possible asbestosis, five had worked in the cotton industry. One of these had been a stripper and grinder for 20 years and was considered to have reached the possible asbestosis category within eight years of first employment at the factory studied, the only sign within 10 years (Table 2). Another man who was certified as suffering from asbestosis after 12 years in the factory had previously been a chemical worker for 20 years; asbestosis was not confirmed at death. It seems likely, therefore, that some of the signs observed in this study were at least partly attributable to previous employment in other dusty occupations.

The association between smoking and signs of asbestosis (Table 6) is in agreement with Weiss (1971) who found pulmonary fibrosis in the chest radiograph in 40% of smokers and 24% of non-smokers among workers exposed to chrysotile asbestos.

The data have been analysed in terms of a family of exposure measures, an inherent feature of which is that there is the equivalent of an exponential decline in the amount of active material. This could arise as a result of both elimination of dust and a reduction in activity of the dust remaining. Beattie and Knox (1961) determined the mineral content after death in lungs of workers from the factory, and found no evidence of a decline in the first eight years after exposure had stopped. However, they took no account of changes in dust level; those who had survived for a period after leaving the factory were, on average, probably first exposed about 15 years earlier than those who died while still employed, and 70% of the men in the study had been employed before 1932. There is evidence that trace metals are leached from chrysotile asbestos *in vivo* (Morgan *et al.*, 1971; Morgan *et al.*, 1973). This leaching, and other changes within the lungs, may reduce the hazard of the remaining material. The range of measures was introduced because it was felt that the simplest measure, that of cumulative exposure, might not be completely satisfactory because of its inability to allow for the possibility of development of disease after exposure has ended. Crepitations appeared to develop in five men while they were not exposed, but in two of these the change could have been caused by observer differences. The changes in the other three men could have been attributable to the transient nature of the sign or to reasons other than asbestos exposure.

Because so few of the men have been retired for even five years, we are unable to discriminate between the different dose-response relationships. Data of this type from other sources would provide

clues to the most appropriate value of the half-life time of dust elimination. If elimination were slow, then cases of asbestosis would be first diagnosed and certified in men who left the industry many years earlier; if this were not the case, then the use of cumulative dose would be reasonably valid. It is known that an excess incidence of lung cancer and mesothelioma occurs in men many years after the end of exposure (Newhouse, 1973) but information is lacking on the diagnosis of asbestosis in former asbestos workers.

There are several reasons why the models might be inappropriate. First, if the diagnostic procedures were not uniform over the period 1961-73, then some of the recorded new cases could be attributable to a change in diagnostic criteria rather than to true clinical changes. The factory medical officer was replaced during this period and, in addition, lung function testing was introduced at about the same time. Second, the assumption that the amount of dust deposited in the lungs is a fixed proportion of the airborne concentration would not be valid if there had been changes in the particle size in the airborne dust cloud during the period 1951-72. In view of the changes in dust levels over this period, a change in the size distribution would not be surprising; however we have no relevant data.

A dose-response relationship between morbidity and asbestos exposure has been considered in two other studies. McDonald *et al.* (1974) studied several facets of disease, death, radiological changes, pulmonary function changes and respiratory symptoms and related these to the cumulative exposure measured as millions of particles per cubic foot times years (mpcf-yr). They concluded that there was a 1% risk of acquiring clinically significant disease for an exposure between 100 and 200 mpcf-yr. Some of their observations were based on a complete cohort but others only on current employees. Weill *et al.* (1975) considered lung function measurements and irregular small opacities on the chest radiographs and found little evidence of a dose-response relationship below 100 mpcf-yr. Using the results of simultaneous sampling with the impinger and fibre counting method they equated this dose with 200 f-yr/cm³. Their observations were based on current employees only, and for some, exposure had started only shortly before the study.

The results of the present study are disappointing in that it is not possible to draw any definite conclusions on the effects of the present 2f/cm³ standard. Comparison of the present results with those given earlier (British Occupational Hygiene Society, 1968; Berry, 1973) shows that there is a higher prevalence of crepitations at any dose than was observed previously. However, in view of the doubt that

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