

Occupational health factors in the United Kingdom

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Asbestos dust, like the dust of many other basic industrial materials, can be a hazard to the health of people exposed to it in high concentrations. It is important that the risk should be clearly understood and that proper controls be exercised over the mining, manufacture and industrial use of the material.

An intensive study of this industrial hygiene problem has been carried out in the United Kingdom, where the most advanced government regulations for its control have recently been introduced. The Asbestos Industry Regulations were first introduced as long ago as 1931, and the pattern of control that has subsequently developed, culminating in the Asbestos Regulations 1969, has been an example to the rest of the world. This is because the British experience has shown that Regulations, where they have been properly applied, can be effective.

Three diseases have been associated with exposure to asbestos fibres. Asbestosis, a type of pulmonary fibrosis, is the result of inhaling high concentrations of fibrous asbestos dust, usually over many years, and a dose-response relationship has been established, i.e. the concentration of dust and duration of exposure determine the length of the interval between first exposure and the first recognisable signs of this disease. In 1968 the British Occupational Hygiene Society published standards for chrysotile asbestos dust, on the basis of which concentrations of less than 2 fibres/ml, averaged over 3 months, would be expected to reduce the risk of having early clinical signs of asbes-

tosis to less than 1%, over a working lifetime of 50 years.

Lung cancer has been associated with industrial asbestos exposure. From clinical observation over the past 40 years it would appear that this complication is more likely to occur in the presence of severe asbestosis. Controls which are successful in reducing the incidence of asbestosis should therefore reduce the associated risk of lung cancer. American epidemiological work indicates the increased carcinogenic effects from smoking for asbestos workers. Asbestos insulation workers in New York who smoke appear to have a greater chance of dying from lung cancer than insulation workers who are non-smokers exposed to the same levels of dust.

Mesothelioma is a malignant tumour of the pleura or peritoneum. Medical opinion is uncertain with regard to the dose-response relationship in mesothelioma, though it may be contracted as a result of a much lower level of dosage than required for asbestosis. By no means all mesothelioma cases seem to be associated with asbestos. Evidence in Britain suggests that in about 10-15% of cases where the information has apparently been complete, no history of exposure to asbestos has been recorded. A recent survey of cases reported in Canada, however, could only trace an association with asbestos in 20%. In those cases where asbestos exposure has been authenticated, the period which elapses between first exposure and the onset of symptoms may be between 20 and 50 years.

Asbestosis can result from exposure

to all commercially utilised types of asbestos, but is confined to those persons occupationally exposed to substantial amounts of asbestos dust.

In Britain the number of confirmed new cases was 134 in 1969. This disease is more prevalent in workers employed in the insulation industry and in shipyards where the Asbestos Industry Regulations 1931 did not apply.

Where mesothelioma has been found to be associated with asbestos, the association has been mainly with crocidolite (blue asbestos). There are far fewer cases who have been exposed exclusively to chrysotile (this may account for the different incidence in the UK and Canada, which is the world's largest chrysotile producer). There are at present no authenticated cases of exposure to amosite or anthophyllite. The number of confirmed cases in Britain each year is about 60.

Mesothelioma has attracted considerable attention in recent years since it was first reported in association with exposure to N.W. Cape crocidolite in 1959. Some of these and some subsequent cases reported in London were of "neighbourhood" and domestic as opposed to occupational origin and it was assumed that these exposures had been to very slight concentrations of asbestos dust. Subsequent work, including dust measurements under conditions simulating some of the exposures described, has shown that these cases could well have been examples of considerable exposure. For example, in 1930 or earlier, a London woman brushing down her husband's dusty

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could well have been exposed to a far higher concentration of dust than has been permitted since 1931 in the factory where he was employed.

In South Africa more cases have now come to light in crocidolite mining areas of the N.W. Cape, but none have been confirmed among people exposed in the crocidolite mining areas of the Transvaal. No authenticated cases have been found among people exposed exclusively to amosite, which is mined in the same areas of the Transvaal and which has physical characteristics very similar to those of Transvaal crocidolite. This difference may be because blue fibre from the N.W. Cape is about 3 times finer in diameter than Transvaal blue and therefore more likely to reach the lung depths and penetrate to the pleura because of its sharp needle-like structure. Chrysotile fibres, which are long and curly, are intercepted in the bifurcations of the broncheolar subdivisions and amosite and crocidolite from the Transvaal do not penetrate as deeply because of their larger fibre diameters. This is a possible explanation of the means whereby certain fibres reach the pleura, but does not explain how the peritoneum is involved, except

the diaphragm. There is no clinical or experimental evidence associating peritoneal mesothelioma with ingestion.

It is important to realise that the cases of asbestosis and mesothelioma being reported today are the result of exposure many years ago. There is a long latent period between first exposure and the first recognisable symptoms. These cases date back to a time when controls were not as effective as they are today in Britain. The Asbestos Regulations 1969 are now applied not only to asbestos factories, but to any occupation in which employees are exposed to concentrations of asbestos dust liable to endanger their health. Approximately 40 to 50% of the cases being diagnosed now are the result of work in the insulation contracting industry, which can involve very dusty jobs carried out in confined and badly ventilated spaces. Insulation work is subject to the Asbestos Regulations 1969, but was not adequately covered by the 1931 Regulations.

Acceptable hygiene standards are clearly set out in Technical Data Note 13 issued by H.M. Factory Inspectorate in the Dept. of Employment. There are many occupations to

which the substantive provisions of the Regulations would not apply, because they do not give rise to dust above the recommended levels. Examples of these might be the handling of asbestos-cement sheets, the fitting of most asbestos-based packings and jointings, and the bonding of brake and clutch linings. The range of such applications is being steadily extended as more products, particularly in asbestos textiles, are produced in dust-suppressed grades.

Thus the essence of the British approach is to impose controls where dust levels could be dangerous to operatives and to restrict the use of crocidolite to essential purposes. Controls work. It has been shown in the asbestos textile factory where we ourselves work that the implementation of measures for dust prevention and personal protection since the early 1930s has reduced the incidence of lung cancer among our asbestos workers wholly employed since then to a level no higher than that expected for the population as a whole. Epidemiological studies at another British factory have shown no excess mortality among asbestos workers involved only in low or moderate exposure to asbestos dust.

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