

Most of the asbestos found in our millipore filter samples was present as single fibres or as ultimate fibrils (Figs. 2 and 3). These could be estimated by counting except that some were obliterated by other contaminants. There were, however, some complex agglomerates (Figs. 4 and 5) and it is impossible to decide in any meaningful way how many particles such masses represent. Such a mass tends to break up when the filter is extracted with acetone and there are far more fibres near an agglomerate than in the rest of the sample. Agglomerates might also break up when meeting surface active substances in the lung.

These observations are offered as an indication that asbestos is a contaminant in the air of cities and as an explanation of the asbestos bodies that have been found in the lungs of persons not industrially exposed to asbestos. The concentration of fibres is small and there is no indication whether the fibres are pathologically significant.

Asbestos fibres are removed from the lung by a mechanism (BOTHAM and HOLT, 1968) that involves the production and eventual fragmentation of asbestos bodies. Evidence from animal experiments suggests that once a fibre is coated it is no longer pathogenic but that the uncoated fibres produce the pathological effects. Since the rate of coating depends on the nature of the fibre, chrysotile (and glass) fibres being coated more quickly than crocidolite and amosite, fibres of the amphiboles are potentially more dangerous. Asbestos fibres in the lung are coated in the cytoplasm of macrophages and giant cells, but if several fibres are retained by the same cell only one is usually coated. Moreover, the number of macrophages in an area of lung is limited. Thus, while low concentrations of isolated fibres are likely to have minimal pathological significance, a high local concentration produced by the break up of an agglomerate of fibres, might represent a potential risk.

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