

Experimental asbestosis

The new work in experimental asbestosis—after an interval of many years—is producing some interesting results. It is now possible to carry out quantitative inhalation experiments in animals. By this means Wagner and his colleagues (1965) and Marris *et al.* (1966) have shown that chrysotile produces less fibrosis than amosite or crocidolite for the same dose by inhalation and this appears to be due to the much more rapid elimination of the chrysotile. Figure 2 shows the results of giving equal doses of chrysotile, crocidolite, amosite, and finely powdered glass to rats by inhalation and then following the rate of elimination for two months after the end of exposure.

Chrysotile is much more rapidly eliminated than the other three dusts. The reason for this more rapid elimination is not yet clear but it may be related to solubility. These findings in experimental asbestosis accord with the observations on man by Beattie and Knox (1961) and by Nagelschmidt (1965) that lungs with severe asbestosis often contain very little mineral compared with other types of pneumoconiosis.

The importance of particle length on the development of fibrosis is still not solved but work by Wagner (1957, 1961, 1965), Holt *et al.* (1964), and Davis (1964) in several animal species has shown that fibrosis of the lungs is produced by fine particles or very short fibres. Long fibres may still be more fibrogenic for a given mass than short ones (Klosterkötter, 1965) but critical experiments to test this in lungs have not yet been achieved. We need experiments of the type King and Nagelschmidt developed to study the effects of mass, surface area, and number in experimental silicosis (Zaidi *et al.*, 1956). How the asbestos produces fibrosis is still uncertain; it appears to be less toxic to the phagocytes in tissue culture than quartz. Pernis (1965) suggests that it may be a direct stimulus to the fibroblasts. Its action on the phagosomes in the phagocytes, which have been shown to be damaged by silica (Nash *et al.*, 1966), is now being investigated.

The nature of the asbestos body—a term now agreed internationally to be more appropriate than the asbestosis body (U.I.C.C., 1965)—has been elegantly investigated by Davis (1964) with the electron microscope, but its importance, if any, in the

pathogenesis of the fibrosis remains obscure. Ferritin—an iron protein complex with about 20 per cent iron—is probably an

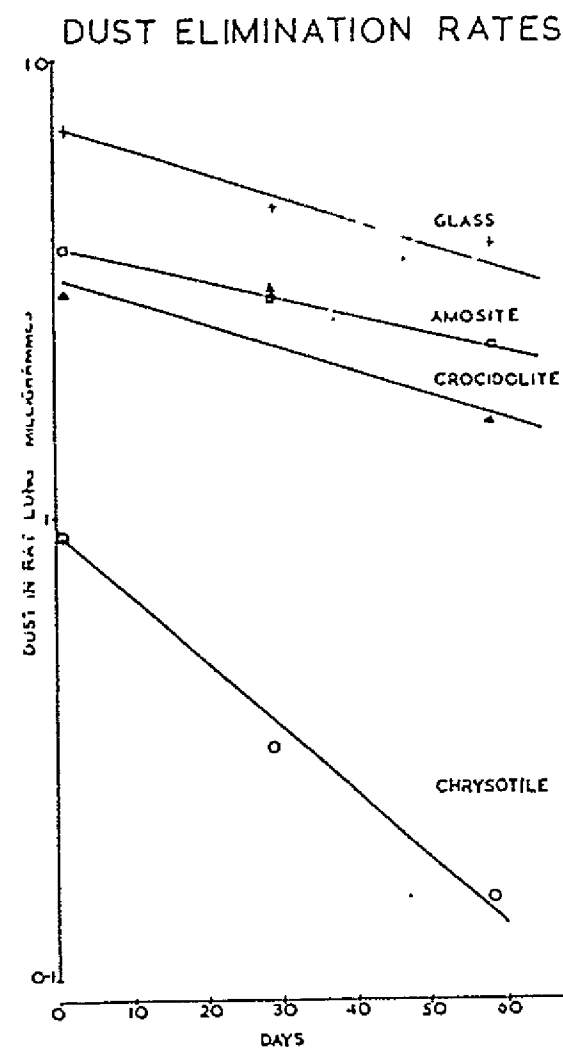


FIG. 2
The weight of asbestos dust and powdered glass in rats' lungs after 30 days' exposure to dust clouds of similar respirable mass concentration, and the subsequent loss of dust over the next 58 days. Each point is the mean of 8 rats (equal numbers of male and female).

important component of iron in the formation of the chest radiograph of other types of pneumoconiosis in the rat. It is of less interest in the rat but of greater interest in man (1966) and there is a great deal of interest in the tissues and spots.

Experimental carcinogenesis. At the time of the association between a well established, little as an experiment in man. Many investigations report by Wagner and to produce mesothelial asbestos. The earlier work and specific pathogen. Twenty mg. of asbestos mesothelial tumours in chrysotile, amosite, extraction of its material—a survival experiment know whether there a tumours produced by has produced the least expectedly high and investigate the effects of the dust inhalation asbestos, therefore, at acts is not yet known. has adsorbed on it. It also takes up oil from other sources (H clear whether these oil nor is it yet clear what animal experiments.