

Once recently, the same authors (1968) again confirmed the non-conformity between the cytotoxic, hemolytic and fibrogenic properties of different types of asbestos. Klosterkotter (1968) also established that the decrease of lactic acid in the tissue culture which contacted with different samples of asbestos does not correspond to the fibrogenic ability of the latter. Unlike Vigliani and Pernis (1968) Schlipkotter established that the hemolytic activity of four forms of asbestos is identical. Polyvinylpyridine-N-oxide (PVN-O) retards the hemolytic activity of amphibolic asbestos; however, it does not affect the activity of fibrosis. Vigliani and Pernis (1968) showed that PVN-O and EDTA turned out to be incapable of protecting the macrophage from cytotoxic action of asbestos.

On the other hand, in the experiments of Klosterkotter (1968) a certain decrease in fibrosis is achieved on subcutaneous introduction of PVN-O.

In the carefully presented work of Roe and Harington (1968) with the aid of histochemical and enzyme methods it was shown that chrysotile, as quartz, exerts a damaging action more actively than crocidolite and amosite. At the same time the macrophages do not lose their capability for transfer of asbestos particles. It is sufficient to say that even 18 months after subcutaneous introduction, they can be revealed in the pleura. Szymczykiewicz, Wozniak (1966) showed that the degree of oxygen intake (per 1 mg of dry tissue) in the culture of the isolated lungs corresponds to the degree of fibrogenicity of the dust of quartz, calcined and noncalcined chrysotile asbestos.

Thus, in spite of the contradictions in the data of the various authors, it is possible to draw the conclusion that asbestos exerts a definite damaging action on macrophages by changing their exchange and changing the rate of increase in and nature of the development of these cells.