

The next major question in the mechanism of the action of asbestos dust is its interaction with the cells of the organism. It has been established that even the long asbestos particles are absorbed by one or several macrophages (Vigliani, Pernis, 1964). In this case the authors did not reveal substantial changes in the macrophages.

According to our observations in conjunction with A. A. Gerasimenko (1966), during the introduction of chrysotile-asbestos into the peritoneum of white mice the number of morphologically changed and, probably, denatured macrophages is for certain 2 times higher than during the introduction of coal dust. There is reason to believe that the damage of macrophages can trigger a complex chain of immunological reactions. Both our works (F. M. Kogan, A. A. Gerasimenko, G. I. Bunimovich, 1967) and observations of Z. S. Repnitskaya, 1967, N. T. Butkin and M. V. May, Medeka, 1960, Gambini, 1961, etc., testify to this. However, in this article we do not analyze the immunological aspects of the problem of the action of asbestos, but only focus our attention on the fate of macrophages.

In conjunction with A. P. Darinovskaya (1966) we succeeded in showing that under the effect of chrysotile-asbestos (and to a somewhat lesser degree serpentine) in twenty-four hours the growth of cells and their migration in the organ culture of the lungs of the human embryo is retarded.

Later, Vigliani and Pernis (1968) came to the conclusion that the dust of chrysotile-asbestos possesses hemolytic activity, damaging erythrocytes; around filaments there is observed an accumulation of young fibroblasts; Davis (1964) is inclined to examine these fibroblasts as a phase of evolution of the macrophage. In this connection we must give special attention to the fact that the same Vigliani and Pernis (1964), denying the cytotoxic action of asbestos, established that in the culture of fibroblasts which contacted with asbestos, the collagen content in albumin is 2 times higher than in controls.