at a concentration of 69 mg/m^3 revealed on the average in the lungs of rats 0.98 mg of chrysotile-asbestos (at the same time, fiberglass - 8 mg) and established a three times more rapid elimination of chrysotile after the stopping of the dust.

Consequently, chrysotile-asbestos is eliminated well, but the remaining comparatively small quantity, by virtue of the high fibrogeneous nature of the dust, is sufficient for the development for fibrosis. The major portion of the dust is driven out through the bronchi. The part which fell in the interstice at the level of the alveolar bronchioles is removed by the lymph flow. However, the lymphatic drainage of alveoli is not able to remove the dust deposited in emplysematous vesicles formed in connection with the obliteration and obstruction of the alveolar passages.

Thus the prolonged accumulation of this dust is apparently the reason for the developing process of fibrosis.

So we have established that both fine-fibered asbestos and granulated serpentine are capable of causing active fibrosis. Subsequently, this capability of short-fibered asbestos was confirmed by Wagner (1958), Yoshikava (1960), Holt, et al., (1964), Donna, Cappa (1967).

The fact that the decisive role is not played by the fibrous structure and hardness of asbestos is borne out by the fact that fiberglass (hardness according to Mohs scale 5-7) does not possess any expressed fibrogenous action, in spite of its more than 8 times more gradual elimination from the lungs (Shepers, 1955, Matytskaya, 1954, Szmay, 1959, Sadkovskaya, 1959, Pushkin, 1962). Davis (1963, 1967) showed that only the few filaments are introduced inside the cells of the alveolar epithelium, a fact which also does not confirm the mechanical hypothesis of the action of asbestos.