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Progress Report on Asbestos Experiments

December 9, 1938

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In reply to your letter of the 6th, I may say that any progress report of experiments on asbestos dust will necessarily be extremely brief.

In the original experiment, undertaken with the grant from the asbestos industry, animals have now been exposed to the very finely ground asbestos fiber for a period of 20 months. Concentrations of atmospheric dust ranging from 125 to 160 million particles per cubic foot of air have been maintained in the dusting room. Animals killed at intervals throughout this period have failed to exhibit any gross evidence of fibrosis. Minute asbestosis bodies have been observed since the second month but cellular reaction is still limited to widely scattered microscopic foci of early fibrous tissue. This is at variance with the experience in a previous experiment where concentrations of only 39.5 to 55.6 million particles per cu.ft. of air had already produced gross evidence of fibrosis in the lungs after 15 months exposure.

As previously reported, the assumption that grinding asbestos fiber to an extremely fine state of subdivision would accelerate the development of reaction in the tissues has not been justified. On the contrary such treatment seems to have had the opposite effect and almost destroyed their capacity to irritate the lungs. This has led to the hypothesis that the

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irritation from asbestos dust is not of chemical but is more probably of a mechanical nature.

Several collateral observations appear to support this view. In a series of intravenous injection experiments with four different varieties of asbestos, ground so that none of the particles exceeded 3μ in diameter, one gram doses failed to produce fibrous changes in any organ of the body after one years time. Since free silica introduced in this manner invariably does cause fibrosis of a characteristic nature and since such reaction is inversely proportional to the size of the injected particles, it seems logical to infer that the reaction to asbestos is of a different nature.

Chrysotile asbestos continues to give trouble, we have never been able to inject the full dose nor to keep an animal alive for more than 7 days thereafter. Either the physical or the chemical properties of this mineral render it unsuitable for injection into the veins. Three other forms, crocidolite, amosite and amphibole have produced no ill effects.

The reaction to inhaled asbestos, unlike that to crystalline silica, is not a progressive one in experimental animals. In a previous experiment 8 months exposure followed by removal of the animals to a normal atmosphere for 2 or 3 years resulted in retrogression rather than progression of pulmonary changes. The asbestos fibers were present in the lungs in very appreciable numbers and they became coated with the

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material responsible for the formation of the asbestos body. They were still present at the end of the observation period but no fibrosis had appeared. Perhaps one might agree with Dr. Leary that the coating on the fibers is protective and that because of it they lose their capacity to irritate the tissues. These observations continue to suggest a mechanical form of irritation which is manifested only in the lungs because only this organ moves enough in normal function to be so irritated.

We are attempting to check these hypotheses by a new inhalation experiment with long-fibered crysotile. Another dust room has been assigned to asbestos and a suitable apparatus has been perfected for breaking up this material and generating a dust containing a high percentage of fibers. Concentrations of 120 million per cubic foot of air are being maintained in the dust room. A group of 100 guinea pigs, 20 rats, 20 mice and 6 cats have now been exposed a little over one month. The time of course is much too short to anticipate results of any kind.

We are also making further checks by injecting short and long fibered asbestos directly into both lungs of a group of animals. To ascertain the influence of active motility on the part of the lung, we have induced an artificial pneumothorax on one side to put the organ at rest; the other lung continues to move normally with respiration. If our assumption is correct, we may expect that the long fiber material will produce fibrosis only on the side where active motion occurs; in the collapsed lung the long fiber should have little effect. The finely ground

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material should produce no reaction in either lung.

If, as anticipated, the long fiber inhalation experiment gives rise to pulmonary fibrosis we shall follow the development of the changes in x-ray films as originally proposed when the short fiber inhalation test was undertaken. However there is little point in undertaking this phase of the work until the monthly autopsy samples begin to reveal definite evidence of pulmonary change.

(signed) LEROY U. GARDNER

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