EXHIBIT P-810

UNION ASBESTOS & RUBBER COMPANY

INTEROFFICE CORRESPONDENCE

December 19, 1938

TO Mr. C. Mosier FROM Mr. R. E. Cryor

Attached hereto please find the report and correspondence which you recently handed me referring to the asbestos dust experiment being conducted by Dr. Gardner at Saranac Lake.

In the present report I find most of the information is further in confirmation of previous results as reported in April of this year.

When this experimental work started in March of 1937, Dr. Gardner outlined the objectives as being two-fold:

1. To observe by x-ray examination any tendency towards development of fibrosis in the lungs of experimental animals subjected to high concentrations of asbestos dust over prolonged time intervals.

2. To determine if possible whether the irritation that develops from asbestos dust in the lung tissues is a result of chemical affects, or mechanical action.

To date the results seem to point definitely to mechanical action as being the cause of fibrosis. This means simply that the shape and size of the particles that accumulate in the lungs appears to be of more importance as a source of irritation than the chemical composition of the particles. Dr. Gardner has found, contrary to what was expected, that the finer particles cause less irritation than the coarser particles. Animals exposed to high concentrations of very fine dust for twenty months have not developed fibrosis of lung tissues, whereas in previous experiments with coarser particles of asbestos dust, fibrosis developed in 15 months exposure. This appears to be an important point and something heretofore not known.

Most of the experimental results seem to indicate that asbestos dust is probably less likely to produce fibrosis of the lungs than other forms of silica or silicate dust.

There is some evidence to show that crysotile fiber may be more harmful than other varieties of asbestos that have been tested including Amosite. This is not conclusive at this time however.

To further check the mechanical theory as the source of irritation injections of coarse dust are being made

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into both lungs of each of a group of animals. With these animals one lung has been artificially collapsed, and thus rendered immobile. The other lung functions normally. If the mechanical theory is correct, presumably, fibrosis will occur in the lung that moves and will not develop in the collapsed lung.

Further experiments are now under way to get additional information on the effects of asbestos dust in different particle sizes and possibly this may lead to some definite conclusions as to where the limits of the hazard, in respect to particle size begin and end.

My personal opinion is that if this work is continued it will likely establish a background of information that will be of considerable assistance in designing equipment and protective devices for eliminating the industrial hazard. If it can be established that only a certain range of particle size and only definite concentrations of dust are harmful, our problem will have been simplified to a considerable extent.

R. E. Cryor:IS

R&C

R. E. Cryor of Union Asbestos & Rubber to C. Mosier on Gardner's Asbestos Dust Experiments (1938) Published by AsbestosClaims.Law
ACL Doc 13133 · Page 2 of 7 asbestosclaims.law/library/13133/2

JOHNS-MANVILLE CORPORATION

TWENTY-TWO EAST FORTIETH STREET

NEW YORK, N.Y.

EXECUTIVE OFFICES

December 14, 1938

Mr. L. J. Silverman
Secretary-Treasurer
Union Asbestos & Rubber Company
310 South Michigan Avenue
Chicago, Illinois

Re: Dr. Gardner -
Asbestos Dust Experiments

Dear Lew:

I am enclosing herewith a copy of a further progress report from Dr. Gardner, dated December 9, 1938.

With kindest regards, I am

Sincerely yours,

Vandiver Brown

Vandiver Brown,
General Attorney

Enclosure
VB: k

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Progress Report on Asbestos Experiments

December 9, 1938

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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ACL Doc 13133 · Page 5 of 7
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asbestosclaims.law/library/13133/5

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 100 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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ACL Doc 13133 · Page 6 of 7 **asbestosclaims.law/library/13133/6**

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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ACL Doc 13133 · Page 7 of 7
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asbestosclaims.law/library/13133/7