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PLAINTIFF'S EXHIBIT 03967.01

CONFIDENTIAL

Asbestos, Que.
March 19, 1949.

George K. Foster

Saranac Report

NOTED V. B.

The report on experimental asbestosis has been reviewed. The experiments and resulting conclusions are summarized in the attached report, to which I have added my own comments and recommendations.

The recommendations are for the industry as a whole. Several points need further clarification; when these have been discussed with the Saranac people I shall submit another report.

Kenneth W. Smith, M.D.

KWS/h.n.

PLAINTIFF'S EXHIBIT WV-000836

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SUMMARY OF FINDINGS

A. Various forms of asbestos fibres produce typical fibrosis in the lungs of some animals.

B. Fibrosis is produced by the mechanical and not the chemical action of the fibre.

C. Fibrosis is produced by the long fibres (20-50 microns) and not by the short fibres (under 20 microns).

D. Asbestosis was produced in an atmosphere with a total dust count of 138 M.P.P.C.F. of which only 0.6% were 10 microns or longer.

E. As the concentration of long fibres in the air is increased, the fibrosis develops in a shorter time.

F. Fibrosis will not progress after removal from dust to normal air.

G. Asbestosis bodies are formed around fibres to prevent further irritation.

H. Aluminum therapy is useless in asbestosis.

I. Asbestos dust causes no significant change in the course of pulmonary tuberculosis.

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COMMENTS

1. Tissue and animal susceptibility was noted in the experiments. Individual susceptibility to the dust among our employees has been noted for many years.

2. We always have believed that the action of the dust was mechanical and not chemical. If there was an added chemical effect, I believe that we would see countless cases of asbestosis because we have had hundreds of men exposed to high concentrations of dust.

3. The theory of the action only by long fibres of 20-50 microns is, at first, hard to accept because:

   (a) I personally have heard the late Dr. Gardner say that probably the most dangerous lengths of fibre for the production of asbestosis are those of 5 to 15 microns in lengths

   (b) In Dr. Gardner's letter to Mr. Sabourin of Feb.23,1944, he states: "I believe that the true hazard in asbestos plants is proportional to the amount of small, individual uncrushed fibres in the atmosphere. I suspect that the reason only one sees little or no typical asbestosis in the mines and mills is because there is probably too little such fibre in the air of these plants. Conversely the reason for the typical disease in the fabricating plants is the fact that the processes there open the bundles of asbestos fibres and tear them into lengths short enough to be inhaled in dangerous quantities".

   (c) In the U.S. Public Health Bulletin #241 entitled "A Study of Asbestosis in the Asbestos Textile Industry" it is stated that the medium length of fibres range from 7 to 16 microns in the plants studied. Dust counts around the mixing pickers and cards showed the lowest percentage of fibre and the shortest lengths of fibre and it was among these men (pickers and carders) that the greatest number of asbestosis cases occurred.

However, in spite of these three statements, I would prefer to believe in the action only of the long fibres and accept these recent Saranac findings because:

I - The dust counts in our mills show 65% - 80% of the fibres are 5 microns or less in length and rarely 1% are 20 microns or longer, and we have relatively few asbestosis cases.

II - The recent Saranac studies have shown that asbestosis will develop in an atmosphere of 138 M.P.P.C.F. total count of which only 0.6% were fibres longer than 10 microns. We have had many areas in our mills where such counts could have been found.

III - Saranac also has shown that, logically, as the concentration of long fibres is increased, more fibrosis develops. The fibre deposits in the Thetford area are of longer fibre than the Asbestos deposit. Although there is no data today, we might assume that the dust in the Thetford Mills has a higher percentage of the longer fibres than that in the Asbestos Mills. This might account in part for the greater number of asbestosis cases seen in the Thetford Plants.

IV - The relatively small number of asbestosis cases occurring in the asbestos mines and mills as compared with the textile plants might be due in part to the percentage of fibres in the industrial atmosphere. Dust counts in various textile plants show up to 26% fibre while counts in our mills show only 4%-5% fibre.

4. That the fibrosis of asbestosis will not progress after removal of the animal from dust to normal air can easily be believed. I have seen many men who, when transferred to non-dusty jobs, have improved physically and whose x-rays have shown no progression.

5. We always have believed that the asbestosis bodies are not evidence of disease, but simply the fact that fibre has been inhaled and the body has shown defensive action in an effort to neutralise the irritation.

6. I still believe that asbestos dust will not aggravate a pre-existing tuberculosis; also that the course of tuberculosis will not be altered in a man who has worked for several years in our mills as long as he has no x-ray evidence of asbestosis. However, at the moment I can neither accept nor deny the statement that asbestosis has no effect on superimposed tuberculosis.

The Saranac experiments have shown that asbestos is not an inert dust like gypsum, nor is it a very active dust like quarts. They have shown that concurrent asbestosis and tuberculosis produce more fibrosis than does either disease alone. Also it has been shown that tubercle bacilli have been found in the asbestotic fibrosis which is not the usual site for this infection. These experiments have shown that there may be some connection between non-tuberculous infection and asbestosis.

I believe that this is a fact for, although I do not think our mill workers have more respiratory complaints than the average man, I have seen several cases where pneumonia has developed on a pre-existing asbestosis. Such cases are extremely difficult to treat, show a very slow recovery and considerable fibrosis in the x-ray. The local doctors have stated that pneumonia in an employee who has worked for many years in the dust often has a fatal termination.

In view of the probable detrimental effect of asbestosis on superimposed pneumonia, I believe that the relationship of tuberculosis developing on a well-established asbestosis should have further study. Although this relationship has not been shown in the recent experiments, my opinions must be neutral until more cases of the disease among our employees have been studied.

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Recommendations

I - The air in the Asbestos and Thetford plants should be sampled and studied for fibre lengths, and these findings correlated with the experimental and human studies at Saranac.

II - Asbestosis can be controlled if:-

(a) Every effort is made to lower the dust concentration in the working area of the employee, and
(b) transfers to non-dusty jobs are effected before any disability develops, probably as soon as there are significant x-ray changes.

III- Tuberculosis would not be a problem if:-

(a) Every active case is isolated and not allowed to work;
(b) non-dusty jobs are found for apparently arrested cases;
(c) routine case finding, examination of all contacts, etc. (an adequate tuberculous control program) was carried out.

IV - We should have an opinion from the Saranac Laboratory on the association of asbestosis and cancer. There is a world-wide effort to find the cause and cure for cancer. The medical literature is full of suggested causes and asbestos has been mentioned as a very definite possible cause. Although in actual practice we have seen no cases of cancer of the lung among our employees, asbestos is an irritant and as such might play some role in the development of carcinoma. An expert opinion on this relationship would be worth while.