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February 28, 1944.

Mr. Ivan Sabourin
Aldred Building
Montreal, P.Q.,
Canada

Dear Mr. Sabourin:

I have your letter of February 22 asking for comments on the relationship between asbestosis and susceptibility to tuberculosis. This question cannot be answered decisively from a study of post mortem material alone. Medical problems of this nature are usually solved on the basis of probabilities and it is this matter of probabilities on which we need further evidence in the case of tuberculosis associated with asbestosis.

Merewether in his "Memorandum on Asbestosis" Tubercle [illegible]er and December, 1933 and January, 1934) admits that "the data on [illegible]ject are meager." He reports that of 374 workers while at work, [illegible] showed signs of active tuberculosis and that the British Silicosis [illegible]Asbestos Medical Board in its first periodic examination of workers in this industry found only one case of asbestosis and tuberculosis in 1512 examinations. On the other hand the English post mortem material at that time revealed 35.7% of the cases complicated by tuberculosis. (Subsequently he appears to have found more cases if Dr. Riddell is correctly informed). In the publication cited Merewether concludes:

> "The evidence therefore, points to the hypothesis that at present the added risk to asbestos workers from pulmonary tuberculosis is limited and definitely less than in workers exposed to free silica; virulent massive infections will as in the general population, cause death; lesser ones will result in more widespread fibrosis than would appear in cases amongst the general population and slight infections, in common with many other affections, are likely to precipitate death in cases of advanced asbestosis where they wouldn't do so in the general population." The underscoring is mine but I agree with these views.

When Dr. Merewether was in this country last year he visited me for several days and we spent most of our time discussing these problems. He still accepts our experimental work which proves that there is no specific susceptibility to tuberculosis induced by asbestosis and he, like ourselves, is anxious to accumulate comparative statistics from various parts of the world to establish the general clinical experience.

As I indicated in my last letter the Quebec experience is not yet analyzed; there is only an impression that most of the asbestos workers die of tuberculosis without taking into account the excessive prevalence of such infection among the population of the Province. This impression needs verification by careful analysis of morbidity and mortality statistics collected from the employees of the asbestos industry, from their families not exposed to asbestos dust and from other communities in the Province of Quebec. Such data should then be compared with similar statistics for asbestos workers in other countries. If there is a specific occupational predisposition to tuberculosis it will become apparent on such analysus just as it has for silicosis. Wherever silicosis is being created, the high mortality from tuberculosis cannot be hidden.

I do not believe that there is much tuberculosis among asbestos workers in the United States and suspect that the excess mortality from this cause in England is probably due to the low living standards of industrial workers. Possibly selection of cases coming to autopsy has influenced the picture. American studies have thus far failed to demonstrate enough tuberculosis among asbestos workers to cause comment. The U. S. Public Health Service Study of Asbestosis in the Asbestos Textile Industry in North Carolina, (Bulletin 241), issued in 1938, hardly mentioned tuberculosis, dismissing the subject with the statement [illegible]age 51 that "there were only 2 cases of active, pulmonary tuberculosis group" (541 employees). Shull, (Radiology 27, No. 3, 279-292: [illegible]examined 71 of 100 cases discharged from the plants where the [illegible] Health Service made its study 15 months later and found 8 cases complicated by tuberculosis of slight to moderate extent but only 2 were active. These two subsequently died of this cause and in 2 others the infection later became active. Shull states in his conclusions that "asbestosis does not predispose to tuberculosis." Presumably he infers, although he does not say so, that the 4 cases of active disease among 541 workmen represented accumulated disabilities when the examination program was inaugurated. Donnelly (J. Ind. Hyg. and Tox. 18, No. 4, 222-228:1936) also examined 151 discharged employees including the 100 reported by Shull. He gives more details on the cases of tuberculosis. One of them had worked in the asbestos industry for 5 months and the others 15 months. The remaining 2 had worked 6 and 10 years respectively and their infections might have been aggravated by exposure to asbestos dust. He points out, however, that an incidence of 2 cases among 151 industrial employees is no higher than in any other industrial group. McPheeters, (J. Ind. Hyg. and Tox., 18, 229-239: 1936) likewise examined and reported on these cases and he is in entire agreement with the other reporters. In his conclusions he states, "No activating or aggravating effect of asbestos dust or asbestosis or tuberculosis was observed." Finally I believe I told you that I myself, had just returned from North Carolina where I had reviewed chest films of some 100 employees of the local asbestos industry and saw only one that showed any indication of adult type pulmonary tuberculosis. This was completely

healed. I commented on the fact to the members of the Medical Board of the State Industrial Commission with whom I was conferring and the Chairman, Dr. Vestal, told me that tuberculosis was a rare finding among asbestos workers although common enough in other industrial workers in their state. It would therefore, appear that in North Carolina asbestosis does not favor or predispose to tuberculosis.

I have just now completed a review of 125 films for an asbestos fabricating plant in another state. In this plant the amount of severity of the asbestosis on the first examination in 1930 was well marked, approximately 40% of the group showing definite X-ray evidence of the disease. Nevertheless, the total adult type tuberculosis rate was only 5%; four of these cases occurred in persons with asbestosis and 2 of them were considered probably active.

A second examination was made upon 67 of the original group after an interval of 5 years during which they remained at work. Now the incidence of asbestosis had increased to 62% but the tuberculosis rate had fallen to 2.9%. A third examination was done on 57 of the original group who still remained in employment after a further interval of 3 to 4 years; in this group the incidence of asbestosis was over 80% but that for tuberculosis only 3.5%. In spite of excessive amounts of severe asbestosis the tuberculosis rate remained within the limits usually found in American industrial populations.

The behavior of the tuberculosis in these people was not unusual. For example one man, a spinner for 13 years, was 31 years old when first examined. He had at that time an early non-specific dust reaction and evidence of old tuberculosis in the top of his lungs. In two succeeding examinations his dust reaction steadily increased to reach first stage asbestosis 9 years after he was first seen. His tuberculosis however, remained apparently uninfluenced. Most of the others behaved in the same manner. These people had already been exposed to asbestos dust for periods of 3 to 18 or more years when they were first examined and the three subsequent examinations during the following 9 years give us a good opportunity to follow what might have developed. No new case of tuberculosis appeared. If it had not by that time the chances were not very great that they would later.

After they left the asbestos industry we do not know what happened to most of them but on a few there are insurance records that indicate the cause of death. Two died of tuberculosis. One had had his infection when he was examined in 1930 at the age of 50. At that time he had an early asbestosis after weaving for 10 years and evidence of associated chronic tuberculosis. He was advised to leave which he did and died of his infection about 3 years later. The second man aged 32 had been a weaver for 12 years and had first stage asbestosis but no evidence of tuberculosis at the time. He left his job after the

first examination for reasons not stated. Three years later he was reported to have died of tuberculosis in a sanitorium. Such cases could occur anywhere.

It is possible that if we had follow-ups on more of these cases after they left the asbestos industry we would find more persons who subsequently contracted tuberculosis and died of it. However, the evidence now available indicates no such tendency. Animal experiments, contrary to those upon silicosis, disclose no theoretical reasons for suspecting that asbestosis creates any special susceptibility to the tubercle bacillus of Koch.

I am becoming more and more convinced that that the high tuberculosis rates reported in English autopsy statistics are due to causes other than occupation. I believe that the same must hold true in Quebec where the amount of severity of the asbestos dust reaction is very much less than it is in the fabricating plants in the United States.

I believe I have covered the other points raised in letter and will therefore not prolong this discussion further.

Very truly yours,

Leroy U. Gardner, M. D.
Director

LUG:RH

cc to Mr. Vandiver Brown