PLAINTIFF'S EXHIBIT

MET 363

October 24, 1939

Mr. W. G. Carlisle, President,
Toledo Vitrified Brick Company,
1220 Madison Avenue,
Toledo, Ohio.

Dear Mr. Carlisle:

I have your letter of October 17 and will try to make clear what the present position with regard to dust particles is. It has been proved that particles of dust that gain entrance into the lung must be not larger than 10 microns in size and all dust counts that are made by the Public Health Service, the United States Bureau of Mines, the Saranac Laboratory, the Harvard School of Public Health, and ourselves are based on a total count of particles under 10 microns. It is true that most of these particles will be around 3 microns in many industrial dust exposures. There is some dispute as to what may be the action, if any, of particles of ultra-microscopic size but this does not have much practical importance.

Our clinical observations are based upon many types of exposure for which the 10 million particle count is a fool rule. It is not by any means a measurement of precision but it is an index that is very useful both in enabling the investigator to have an adequate conception of the actual silica hazard in a given place; also it forms a useful means of checking the value of dust exhaust equipment or other dust control measures that may be set up.

With respect to free silica, that is silica existing in a free or uncombined state, it is the accepted opinion among the investigators in this country, England, South Africa, and Canada, that only free silica causes silicosis, and that combined silica, such as magnesium silicate, sodium silicate, or any other chemical combination, does not cause the disease. As far as we know, only one silicate dust, namely asbestos, has been shown to produce a typical characteristic fibrosis of the lungs which we call asbestosis. Asbestosis pathologically is quite different from silicosis and can be differentiated from it either by X-ray or by microscopic examination of pathological specimens.

Generally speaking, a dust is dangerous in proportion to the amount of free silica which it contains. Another factor, however, enters into this picture. It has been shown that where certain substances may be present in the dust along with the silica, these substances may tend to affect the action of the silica in the lungs, either retarding it or accelerating it. Both metallic aluminum and aluminum oxide, for instance, are credited (at least on the basis of animal experimentation) with being able to inhibit the action of silica entirely. Substances present in the dust of iron ore mines and in some granite works apparently retard the action of silica. At least this is the only explanation which can be offered for the fact that in these certain industries, silicosis develops much more slowly than would

be expected if the silica content of the dust were the only governing factor. On the other hand, some of the British observers conclude that certain alkalis when present in the dust along with the free silica would greatly intensify its action. These observations were made with respect to soap powder dusts. However, these latter findings have not been substantiated and are still open to question.

I enclose a couple of reprints which you may find of some interest. I will be glad to hear from you further if I can be of any service.

Sincerely yours,

A. J. Lanza, M.D.
Assistant Medical Director