PLAINTIFF'S EXHIBIT

UC-127

UNION CARBIDE CORPORATION

270 PARK AVENUE, NEW YORK, N.Y. 10017

MID.CAL DEPARTMENT

June 7, 1967

Dr. T. J. Hall Union Carbide Europa A.A. 40, Rue Du Rhone 1211 Geneve 3 Suisse

Dear Tom:

I have reviewed the report, "Asbestos As A Health Hazard In The United Kingdom", prepared by I. C. Sayers, and in general I find that it is reasonably accurate. I do not attach very much importance to the data on the incidence of asbestosis and death from asbestosis and cancer because these are merely tabulations of events and do not take into consideration the numbers of people who were subjected to the possibility of the event occurring.

We had been interested in the possibility that the short fiber Coalinga product might have a greater hazard than the more conventional forms of asbestos because sub-micron silica has been known to cause a rapidly progressive silicosis after exceedingly brief exposure. We were concerned about whether the Coalinga material with its exceedingly fine fibril diameter might have a similar effect in causation of asbestosis. We therefore made some preliminary studies in which the material was injected into the belly cavity of guinea pigs, rats and rabbits and also was injected intratrachealy by a method which distributes the aspestos throughout the lungs of rats. The materials injected were the standard fiber, a refined fiber and a long fiber obtained from Johns Manville for purposes of comparison. In the injection study the Coalinga refined fiber produced the most severe reaction in the belly cavity, whereas the

standard fiber and the Johns Manville fiber were essentially the same and less severe. In the injection study in the lung the crude fibers caused a more severe lesion, the refined fiber was intermediate and the Johns Manville fiber was least reactive. The only conclusion we can draw from this crude test is that it is possible that our Coalinga product may be more halfardous to use than long fiber asbestos in that it may induce the disease, asbestosis, at an early time after exposure.

The question was raised whether the 5 million particles per cubic foot were suill valid as a Threshold Limit Value. I maintain that this value is still correct in terms of preventing the disease, asbestosis. There is no evidence of asbestosis occurring among people who have worked in an environment where the concentration was kept within the Threshold Limit Value. It is probable that the 5 million particles per cubic foot will not be acceptable for the prevention of mesothelioma. I have no idea what concentration might be effective in preventing this disease and I would wonder whether even a limit of 1 million particles per cubic foot would be effective in this regard.

Finally, I would give my unquestionable support to the request of the Pneumoconiosis Research Council for trace element analysis of the standard sample of asbestos. By so doing we would develop techniques which could be applied to our product from King City and which would be useful in making comparisons with the standards.

I hope this will be helpful to you, if you have any further questions please let me know.

C. H. Wern M. Will

Associate Medical Director

C. U. Dernehl, M.D. ed