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SUPPLEMENTARY PROGRESS REPORT

ON

FIBROUS DUST STUDIES

for

**Johns-Manville Corporation
PPG Industries
Pittsburgh Corning Corporation
Raybestos-Manhattan, Inc.**



INDUSTRIAL HEALTH FOUNDATION, INC.

225 N. MICHIGAN

ANN ARBOR, MICHIGAN 48106

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INDUSTRIAL HEALTH FOUNDATION, INC.
A NON-PROFIT ORGANIZATION FOR THE ADVANCEMENT OF HEALTHY WORKING CONDITIONS IN INDUSTRY

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by

**Paul Gross, M.D.
Director of Pathology Research**

September, 1974

**Daniel C. Braun, M.D.
President**

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In a recent publication, "Asbestos-Induced Intrathoracic Tissue Reaction," we reported the results of our investigation that was sponsored in part by the industries listed on the title page and in part by the National Institutes of Health. It was based on a total of 192 survivors out of a total of 430 rats and 150 survivors out of a total of 239 hamsters, all of which had been injected intrapleurally. The animals not included in this report, equivalent to about 55% of the injected rats and 37% of the injected hamsters, were excluded because they had died too early of epidemics of pulmonary disease. By "too early" is meant prior to the time when the first tumor could be expected to appear.

The animals injected intratracheally with asbestos suffered much greater mortality rates than the animals injected intrapleurally. This was largely attributable to the necessity of performing multiple intratracheal injections in order to introduce a sufficiently high dosage of dust into the lungs. A dosage greater than 1.75 mg was often lethal.

However, in a previous investigation lung cancers have been produced in rats as early as 17 months after asbestos exposure. At about that time (in 1969) an epidemic of pneumonia decimated the animals to a degree which forced Dr. Gross to replace the dead animals with new ones in order to obtain meaningful results. In June, 1971, these new animals were transferred to the Medical University of South Carolina under the general care of Dr. Carllick, D.V.M. About six months after this transfer, an epidemic of pneumonia caused by mycoplasma killed most of the animals injected intratracheally with amosite. The result of this high mortality

among these animals was that no conclusions could be drawn. Although no lung cancers had developed in any of these animals, the animals injected intrapleurally suffered a lower mortality and definitive conclusions were possible. These conclusions were incorporated in a report published in October, 1973.

Microscopically, the lungs of the animals were characterized by extensive acute pneumonia which largely overshadowed the changes caused by the different types of asbestos dust. There was no discernible difference in the character of the lesions associated with the various types of asbestos or their modifications. No pulmonary tumors were seen. However, because of the overwhelming nature of the associated pneumonia, no definitive conclusion is possible with regard to the differences in effect (if any) on the pulmonary tissues of the various types and modifications of asbestos. Because the tracheobronchial tree of rodents will tolerate only very small doses of asbestos dust suspensions, many injections are required to attain proper dosage. As a result of this study, it has become painfully apparent that an investigation requiring the lungs to be loaded with a considerable asbestos dust burden by means of numerous intratracheal injections is doomed to failure. This failure is assured by the fact that each intratracheal injection is associated with infective organisms that will ultimately cause fatal pneumonia.

Nevertheless, although the results of the intratracheal injections do not allow for conclusions, the results of the intrapleural injections suggest that amosite is no less cancerogenic than crocidolite and that chrysotile is

appreciably less carcinogenic than either amosite or crocidolite. It can also be concluded that the pathogenicity of all three types of asbestos decreases when they are heated to 900° to 1000° C for two hours, and that the trace-metal theory of the pathogenicity of asbestos must be abandoned.

Aside from the conclusions pertaining to the cancerogenic potential of the different types of asbestos, this investigation has demonstrated, as pointed out in the accompanying reprint, "Asbestos-Induced Intrathoracic Tissue Reactions," that cells other than mesothelial cells also may give rise to cancers when materials are injected into the chest cavity. This demonstration casts doubt on the propriety of labeling all intrathoracic tumors as mesotheliomas. Inasmuch as many of the intrathoracic tumors are fibrosarcomas, and since fibrosarcomas arise spontaneously in rats, the validity of using the intrapleural injection technique in rats as tests for carcinogenicity becomes highly dubious.