file: Asbestos Literature References

INDUSTRIAL HYGIENE FOUNDATION
OF AMERICA, Inc.

ASBESTOS BIOEFFECTS RESEARCH FOR INDUSTRY

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Subject: Summary of Needs and Plans for Asbestos Research

The purpose of this session of the Industrial Hygiene Foundation Management Planning Meeting, held in the Hunt Room of the Webster Hall Hotel on July 19, was (1) to help develop a better concept of the asbestos industry's environmental and occupational health research needs and what is being done to meet them and (2) to provide the U. S. Public Health Service Surgeon General with a summary report and any recommendations which may be developed from the meeting to aid in setting, clarifying, and reaching sound and realistic health conservation goals in the national interest.

The meeting was sponsored by the Foundation's Policy and Planning Committee.

Those attending the meeting were:

Name	Company
Bowman, H.M.	Thiokol's Reaction Motors Division
Colwell, M.O.	Aluminum Company of America
Cralley, L.J.	U.S. Public Health Service
Davison, E.K.	Davison Sand & Gravel Company
deTreville, R.T.P.	Managing Director of IHF
Edwards, F.H.	Owens-Corning Fiberglas Corporation
Grant, L.B.	Pittsburgh Plate Glass Company
Gross, Paul	Director of IHF Research Laboratory
Hatch, T.F.	University of Pittsburgh
Hazard, W.G.	Owens-Illinois Glass Company
Minard, David	University of Pittsburgh
Schrenk, H.H.	Secretary, IHF Board of Trustees
Smith, F.W.	Mine Safety Appliances Company
Smith, K.W.	Johns-Manville Corporation
Smyth, H.F.	Mellon Institute
Wright, G.W.	St. Luke's Hospital

Dr. J. C. McDonald Montreal, Quebec	Dr. Arthur J. Vorwald Detroit, Michigan
Dr. Leo Noro Helsinki, Finland	Dr. J. C. Wagner Penarth, Glamorgan, Wales
Dr. Sherman S. Pinto Tacoma, Washington	Dr. George W. Wright Cleveland, Ohio
Prof. Paul Sadoul Nancy, France	Dr. Philip Enterline Montreal,Quebec
Dr. K. W. Smith New York, New York	Dr. Philippe Duval Noranda, Quebec
Dr. J. A. Vidal Montreal, Quebec	Dr. Eugene Pendergrass Philadelphia, Pa.
Prof. E. C. Vigliani Milan, Italy	Mr. K. W. Nelson Salt Lake City, Utah

In the course of the QAMA Meeting, much of "what is known" was reviewed and discussed. Dr. Wright found the meeting very valuable from a personal standpoint, chiefly regarding discussions of the very pertinent questions, "What we need to know" and "what is unknown."

For example, he stated that the word "asbestos" is as general a term as "bacteria" and that it is not possible to translate data from one type to another. The types vary with origin and chemical composition and almost never exist in a "pure" form. It is very difficult to categorize an exposure environment because of the need to capture, identify, recognize, and quantitate the material we now call "asbestos". During processing, asbestos, which is not indestructible, may undergo dissolution or change in form. Is the material redisseminated identical to the original, or does it differ?

Dr. Wright indicated that it had been sobering to realize the extent to which asbestos is distributed in various forms in the general population.

he said, the loose thinking concerning asbestos exposure was demonstrated recently in Pennsylvania when the occurrence of mesothelioma in a three-year old child was attributed to the father's working in industry under the union category of an asbestos worker. Actually, this union category title covers many individuals who have no occupational exposure to asbestos.

At this point Dr. deTreville called on Dr. Lewis J. Cralley to provide a brief report on U.S. Public Health Service, Division of Occupational Health plans.

Dr. Cralley stated that he would like to cover briefly three areas of U.S. Public Health Service, Division of Occupational Health interest.

(1) A prospective longitudinal epidemiological study is now under way in four industry exposure categories:

(a) Asbestos textiles

(b) Asbestos friction products

(c) Asbestos cement products

(d) Asbestos insulating materials

The aim is to cover at least 2500 workers in each category with a wide range of exposure from office to production worker and with built-in controls. Exposures are being defined in detail including factors other than asbestos. In addition, the past history of plants regarding asbestos exposure is being recorded in an attempt to develop retrospective studies designed to compliment those of a prospective nature. Detailed information on each worker is being developed using a questionnaire approach, which includes occupations, hobbies, and smoking history. These studies will be carried out over the

Doll's study is that early data indicates that industrial hygiene control of dust exposures will reduce the incidence of lung cancer in asbestos workers.

Dr. deTreville then requested Dr. Gross to provide a detailed description of IHF's Fibrous Dust Research Proposal.

Dr. Gross said that Thomson[1] has found that about 30% of the people that had died in hospitals in Capetown (500 consecutive autopsies) had "asbestos bodies" in the lungs. The same author[2] had found a similar prevalence of "asbestos bodies" among the hospital deaths in Miami, Florida. Cauna, Totten and Gross[3] examined the lungs of 100 non-selected hospital deaths for which autopsy permission had been obtained and found that 40% of these had "asbestos bodies" in their lungs. Ian Webster[4] recently reported a 47% prevalence in Johannesburg.

There is, at present, a reasonable doubt that "asbestos bodies" are specific indicators of the inhalation of asbestos fibers. Such bodies have been found in coal miners and talc workers where they have been called

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(1) Thomson, J.G., et al. Asbestos as Modern Urban Hazard. S. Afr. Med. J. 37:77-81, 1963.

(2) Thomson, J.G., et al. Asbestos as an Urban Air Contaminant. Arch Pathol. 81:458-464, May 1966.

(3) Cauna, Dzidra; Totten, Robert S.; and Gross, Paul. Asbestos Bodies in Human Lungs at Autopsy. JAMA 192:371-373, May 3, 1965.

(4) Webster, Ian. Report of progress quoted in the Annual Report of the Pneumoconiosis Research Unit of the South African Council for Scientific and Industrial Research, 1965.

which indicated a greatly increased risk of lung cancer among British workers exposed to asbestos dust while fabricating the asbestos.

2. In a recent investigation in the laboratory of the Industrial Hygiene Foundation, lung cancers were found in rats exposed for 18 months to high concentrations of chrysotile dust.[9] Because rats exposed to chrysotile dust by other investigators failed to develop lung cancer, there is a good probability that the chrysotile dust that produced lung cancer was significantly different from that which did not have this effect. It is possible that this difference is caused by a coating of nickel steel alloy applied to the asbestos dust particles during the continuous hammer milling process used for rendering the dust respirable. This method has not been used by previous investigators. Nickel has been found capable of producing lung cancer in trace amounts.

Although asbestos and glass are both silicates, the industrial health experience with workers of these two materials has been vastly different. No pulmonary disease has been reported in workers exposed to glass dust.

Nevertheless, the dust of filamentous glass has been labeled as highly dangerous in the lay press by association with asbestos. It is a fact that although the effect of flake glass dust on the lungs of animals has been reported, no similar study has been made of the dust of filamentous glass which does not have an associated resin or filler.

It is proposed to examine the response of animals' lungs to different inorganic fibrous materials of contrasting chemical composition, and to characterize the differences and similarities in the response.

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(9) Gross, P. and deTreville, R.T.P. Experimental Asbestosis: Studies on the Progressiveness of the Pulmonary Fibrosis Caused by Chrysotile Dust. To be published.

These materials will be injected intratracheally into hamsters under ether anesthesia. Previous experience has demonstrated that asbestos bodies will form in response to chrysotile dust within five months. The animals will therefore be held for six months and then killed. Asbestos bodies will be sought in lung juice as well as in the lung tissues.

The lungs will be expanded with buffered formalin under a head of 10 cm. of water. Blocks removed from these lungs will be sectioned at 6 μ after paraffin impregnation. These sections will be stained by different methods and photographed sequentially to provide means for studying more completely the tissue reaction to these various materials. Search for the so-called asbestos bodies will be made using routinely stained sections, and sections stained for iron. In addition, unstained cleared sections will be examined under dark-field conditions for asbestos bodies. The latter glow when examined in this manner.

The difference in tissue reaction between the synthetic chrysotile dust and that which is known to be fibrogenic as well as carcinogenic will point to the contribution to pathogenicity made by one or more of the various trace substances which may be associated with chrysotile asbestos dust as a result of processing.

The development of "asbestos bodies," better termed Ferruginous bodies, to dusts other than asbestos would point to the non-specificty of these structures and to the desirability of collecting such "asbestos bodies" from human lungs in order to subject them to electron-probe analysis. Such

III. Because of the interest of a number of companies in biologic investigations similar to those planned for chrysotile asbestos, but using instead, amosite and crocidolite, an additional investigation is proposed to be undertaken concurrently.

As in the proposal dealing with chrysotile asbestos, the purpose of this supplemental, concurrent investigation is to attempt to pinpoint the cause of the biologic activity of all three main types of asbestos dusts: whether this lies in the silicates per se, in the associated trace metals, or in the associated hydrocarbons.

Because neither amosite nor crocidolite is available as a pure synthetic material, the investigation proposed for these two types of asbestos is necessarily different from that proposed for chrysotile. Nevertheless, in order to make the investigation complete, it is desirable to include also chrysotile in the new investigation since different methods are involved.

Inasmuch as the biologic activity of asbestos seems to have two components: (a) inflammatory— responsible for asbestosis, and (b) neoplastic— responsible for lung cancer and mesothelioma, it is advisable to investigate both of them. This can readily be accomplished within the four year span contemplated for this investigation.

In order to determine whether the biologic activity of asbestos resides in the silicate, in the associated trace metals, or in the associated hydrocarbons, attempts will be made to remove the trace metals and the hydrocarbons from their respective silicates and to compare the biologic activities of such "purified" asbestos samples from which these associated materials have been removed.

3. the dust minus the hydrocarbons (solvent extraction). Here, it is anticipated that the extraction may not be 100% complete.
4. the dust minus trace metals
5. the dust minus trace metals then subjected to solvent extraction to remove most of the hydrocarbons

In order to investigate the inflammatory component of the various (15) batches of asbestos dust, these will be injected intratracheally into rats and hamsters. The lungs of these animals are expected to show definitive changes within one year after the intrapulmonary introduction of the dusts.

In order to explore the lung cancer aspect of the neoplastic component of the biologic activity of asbestos dust, additional rats will be injected with the same batches of dust as above and allowed to live out their lives. Most of the animals, however, will have died before the end of the third year.

The other (mesotheliomatous) aspect of the above neoplastic component will be investigated by injecting the 15 different batches of asbestos dust within the pleural cavity of rats and hamsters and allowing them to live out their lives. Most of these animals will also have died before the end of the third year.

For the investigation of the inflammatory component of asbestos dust, a large enough number of animals will be put on test to allow sampling at intervals throughout the year and still have an adequate number remaining to represent the 12 month old lesion.

The final report covering the entire investigation will be issued toward the end of the fourth year. This report will probably be in the form of several manuscripts suitable for publication.