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7/5/98: Dr. Hans Weil: EPA Hearings

Undated memo (to Paul Kotin?) whines about Selikoff excluding "us" from a portion of the NYAS Conference (prob. 1978)

2/16/72: EPA hearings testimony notes that an excess risk of lung cancer in asbestos workers was "established in the late 1940's and early 1950's." (p.2)

Paul Kohn

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from the desk of **Hans Weill, M.D.**

I consider this action to keep us off the formal portion of the NYAS Asbestos Conf. a flagrant example of poor management, undoubtedly politically motivated. I'm considering what responses/alternatives are open to us.

Hans

EPA Hearings on Proposed Emission Control Standards for Asbestos,

Los Angeles, February 16, 1972

Statement of Dr. Hans Weill, Tulane University School of Medicine
New Orleans, Louisiana

My name is Hans Weill and I am a specialist in Pulmonary Disease and Professor of Medicine at Tulane University School of Medicine. For the past ten years I have been actively engaged in the investigation of pulmonary effects of industrial and community environmental inhalants, including organic and inorganic dusts and toxic irritant gases. For the past two years, we have been carrying out a comprehensive investigation of workers engaged in the manufacture of asbestos cement and other asbestos-containing construction products. I appreciate the opportunity to provide this statement, following which I will be pleased to attempt to clarify any further questions which you may have concerning the biological effects of asbestos exposure.

Because of the evidence indicating an association between dose of asbestos exposure and its adverse biological effects, it is not surprising that these reactions are most easily recognized in industrial populations where this exposure is highest. It has been appreciated for one-third of a century that excessive occupational exposure to asbestos dust can result in diffuse pulmonary fibrosis or asbestosis. Recent evidence strongly suggests that this hazard can be controlled by reducing industrial dust levels and there is general agreement that even lower community levels of asbestos

fiber are not likely to be associated with a fibrosing effect on the lungs.

That an excess risk of bronchogenic carcinoma of the lungs occurs in workers exposed to asbestos was established in the late 1940s and early 1950s. This risk is not the same for all types of asbestos exposure and seems to be associated, perhaps synergistically, with at least one other factor, cigarette smoking. Of great importance to the purposes of this hearing is the more recent demonstration that reduced asbestos exposure in the plants results in not only a lower risk of developing pulmonary fibrosis but also is associated with a marked fall in mortality for cancer of the lung. This evidence that the increased risk of developing pulmonary neoplasms can be controlled by adequate dust suppression in the plant comes from two separate studies in the United Kingdom and leads to guarded optimism, not only in relation to the industrial asbestos exposures but, by extrapolation, to the tentative conclusion that the lower general environmental asbestos levels do not constitute a significant risk to the population in terms of the development of lung neoplasms.

If there seems little likelihood that asbestosis or bronchogenic carcinoma of the lung can result from general environmental levels of asbestos fiber, what can be said about the rare but important tumor called mesothelioma which may affect the lining of the chest or abdominal cavities? This complex and interesting story has been evolving during the past decade with interest stemming initially from the reported association of industrial

and geographic proximity to the crocidolite mines in the Northwest Cape of South Africa and the appearance of these tumors. Exposures were not quantitated but were likely to have been quite heavy. Confusion resulted from the observation that similar mines producing blue asbestos in the Transvaal section of South Africa were not associated with cases of mesothelioma in the exposed occupational and geographic populations. While a few cases of mesothelioma have been reported in the surrounding environs of a plant using primarily crocidolite in the United Kingdom, emissions were uncontrolled and likely to have been considerable. In a survey of mesothelioma in Scotland recently reported, it was difficult to separate occupational from residential exposure since most of those working in the asbestos industry lived close to the industrial sites, and only one male case of mesothelioma occurred out of 51 individuals who did not have occupational exposure and who lived within one-half mile of shipbuilding, docks, or asbestos factories. It can be readily appreciated that the evidence supporting an association between mesothelioma and non-occupational residential exposure to asbestos is quite limited, particularly when contrasted to available data which suggests little association between general community asbestos exposure and the development of mesothelioma.

The Mayo Clinic recently reviewed 37 cases of mesothelioma which had been seen during the previous 22 years. In only ten of these was there

a definite or probable occupational exposure to asbestos, while in 27 no history of environmental or occupational exposure was detected. None of these patients lived in major industrial or metropolitan areas and a non-occupational exposure to asbestos fiber seems highly unlikely. The most precise definition and quantitation of asbestos exposure is at present possible only for an industrial environment where significantly lower asbestos dust levels have recently been demonstrated to be associated with a normal or only slightly increased risk for pulmonary or pleural malignancies. If confirmed by continuing studies, this ultimately provides strong evidence that the minimal asbestos exposures in urban communities will have no adverse measurable biologic effects.

Since today our attention is being directed to the portion of the proposed emission standards which prohibits visible emissions of particulate matter from asbestos-containing tailing dumps, let us examine the most pertinent scientific evidence concerning the risk associated with environmental exposures in areas geographically proximate to asbestos mines. Last month at these hearings in New York, Dr. J. C. McDonald of McGill University testified in some detail about the investigations which he has supervised on the Quebec chrysotile mining and milling industries and the Canadian mesothelioma survey. Because of the importance of these results to the hearings, I would like to briefly summarize the appropriate portions of these studies. In the study of mortality in the chrysotile producing industry of Quebec, a very high follow-up of deaths was achieved which presented strong evidence

that even heavy exposure in this industry carried only a modest risk of lung cancer and even a smaller risk of malignant mesothelioma. Again, the evidence that low and moderate asbestos dust levels are not associated with increased risk of developing pulmonary or pleural malignancies in the workers has strong implications concerning that risk in the non-occupationally exposed population. The asbestos-containing tailings dumps in the United States are in chrysotile mines, underlining the importance of Dr. McDonald's findings to this deliberation.

Results of an epidemiologic survey of malignant mesothelioma in Canada are also of interest. Although an association between the development of these tumors and occupational exposure (usually not mining or milling) was found in 1/4th of the cases, after exclusion of occupational and domestic exposures there was no case of mesothelioma occurring in an individual who had a history of ever living within 20 miles of an asbestos mine or mill. This encouraging data concerning chrysotile asbestos health effects, which has been substantiated in chrysotile mining and milling in other portions of the world should be evaluated in view of the facts that all of the asbestos mines and mills in this country produce chrysotile fiber and over 90% of the asbestos used in manufacturing and other utilization is also this fiber type. Strong consideration must be given to the possibility that some or all of the biological effects of asbestos may result from something other than the fiber itself, possibly a contaminant introduced in processing or manufacturing.

This and other unresolved questions will hopefully be answered as the result of intensive research being conducted on this problem.

Because of my interest in maintaining the health of workers and the population in general, I support vigorous efforts in controlling the emission of actual or potential injurious inhalants, even when doses which have been demonstrated to have an adverse biological effect are of a different order of magnitude than those likely to be encountered in the general environment. I, therefore, support most of the provisions of these proposed emission standards but would suggest that present evidence does not indicate a significant risk from the environmental levels of chrysotile asbestos surrounding these mines. While the technology is being developed to control these emissions, continued health surveillance will provide data which hopefully will confirm these present tentative conclusions.