FILE NAME: Union Carbide (UC)

DATE: 1984

DOC#: UC102

DOCUMENT DESCRIPTION: Letters from Mt. Sinai to the EPA with Cover Letter to the ORC Asbestos Task Force

National Place, Suite 911
1331 Pennsylvania Avenue, N.W.
Washington, D.C. 20004
202-737-6330

Organization Resources Counselors, Inc.

Memorandum

February 7, 1985

To: ORC Asbestos Task Force

From: Darrell K. Mattheis

Subject: Letter from Dr. Arthur Langer, Associate Director, Environmental Sciences Laboratory, Mt. Sinai, to Dr. John A. Moore, EPA, Concerning the NRDC Petition for a Prohibition on the Use of Asbestos in Auto and Truck Brakes

Thanks to John Marsh, I recently received a copy of a very interesting letter as described above. I think that Dr. Langer's opinion, as formulated in his letter to Dr. Moore, will be of interest to the members of this Task Force. Enclosed is a copy of the "Langer Letter".

Darrell K. Mattheis

DKM/jt

Enclosure

RECEIVED
FEB 12 1985
T. A. LINCOLN, M.D.

02873

Copies of two letters from Dr. Arthur Langer, Associate Director, Environmental Sciences Laboratory, Mount Sinai Medical Center to Dr. John A. Moore, EPA Assistant Administrator, concerning the petition of the NRDC for a "Prohibition on Continued Use of Asbestos in Automobile Brakes" are enclosed.

It is interesting that Dr. Langer considers the documentation in the petition to be "mindless and pure rubbish."

More importantly, however, it is noteworthy that Dr. Langer recognizes the significant differences in risk produced by different fibre types and industrial processes.

This is the first time I have seen a statement by a member of the Mt. Sinai group that such differences exist. I don't know whether it represents a consensus among the Mt. Sinai scientists; on the basis of a recent conversation with Dr. Selikoff, I doubt it, but the letter might be useful in the "chrysotile defense" and in cases where Dr. Langer is a witness.

Sincerely,

C28734

Comments C File # /
MOUNT SINAI MEDICAL CENTER .

6.

THE MOUNT SINAI MEDICAL CENTER

ONE GUSTAVE L. LEVY PLACE • NEW YORK, N.Y. 10029

Mount Sinai School of Medicine • The Mount Sinai Hospital

Environmental Sciences Laboratory
Cummings Basic Sciences Building
10 East 102 Street
New York, New York 10029
(212) 650-6173

October 11, 1984

Assistant Administrator for Pesticides
and Toxic Substances
Environmental Protection Agency
637 East Tower - TS788
401 M Street, S.W.
Washington, D.C. 20460

Dear Jack:

I have just read the NRDC petition for a prohibition on continued use of asbestos in automobile brakes, sent to me by Steve Shapiro. The documents are mindless and pure rubbish. My major concern with the documents is that data are invoked to support their own argument taken from several of my own papers.

Do you really have to answer this petition?

Sincerely,

Arthur M. Langer, M.D.
Environmental Sciences Laboratory

AML:jm
cc: Dr. Steven Shapiro

Steve — Thanks for the documents!
Art

02875

THE MOUNT SINAI MEDICAL CENTER
ONE GUSTAVE L. LEVY PLACE • NEW YORK, N.Y. 10029
Mount Sinai School of Medicine • The Mount Sinai Hospital

December 7, 1984

Environmental Sciences Laboratory
Cummings Basic Sciences Building
80 East 102 Street
New York, New York 10029
(212) 650-6173

John A. Moore, DVM
Assistant Administrator for Pesticides
and Toxic Substances
Environmental Protection Agency
637 East Tower - TS788
401 M Street, S.W.
Washington, D.C. 20460

DEC 16 1984
Office of the Assistant Administr...
For Pesticides and Toxic Substanc...

Dear Dr. Moore:

It has been made known to me that my written remarks of October 11, 1984 concerning the Natural Resources Defense Council's Petition for a "Prohibition on Continued Use of Asbestos in Automobile Brakes" will enter, with other documentation, into the rule-making docket along with the original petition. I am therefore requesting that this letter be substituted as a formal critique.

My concerns regarding the petition are many, essentially focusing on lack of insight and on an unscholarly approach to the problem:

- The NRDC claims that both epidemiological and pathological reports "have shown only recently that asbestos is one of the largest single causes of environmental human cancer in the United States." This statement does not accurately reflect the literature. In support of this statement the petition cites HHS Secretary Califano who reported that approximately 17% of American cancer deaths over the next several decades will be "linked" to asbestos. There are approximately five hundred thousand cancer deaths per year in the United States which would roughly translate into 85,000 cancer deaths per year "linked to asbestos." Immediately following this statement is an attribution to Nicholson and co-workers who have estimated that cancer deaths in the United States from asbestos will number some 8,500 to 10,000 per year over the next 20 years. The authors of this document fail to reconcile this order of magnitude difference in these figures; fail to resolve the proportion of excess cancers attributable to workplace exposures; have not considered that if 8,500 cancer deaths will occur among 10-15 million "exposed" workers in different occupational settings, 85 thousand asbestos "linked" deaths means that everyone in the general population is at the same level of risk. It seems farfetched;

- In their discussion of mesothelioma and lung cancer, the NRDC authors use data from studies of insulation workers in their descrip-

C2876

tion of increased risk of asbestos diseases in exposed populations. The nature of the fiber exposure, fiber type, fiber dimensionality and fiber concentration are significantly different when compared to workers maintaining and repairing brake pads. Data for insulation workers may have little bearing for brake workers. These data are uncritically accepted as data applicable for all workers exposed to any variety of asbestos fiber;

- The assertion that all asbestos fiber types are carcinogenic in humans is correct. However, not all fiber types produce human mesotheliomas (no mesotheliomas have been reported amongst humans exposed to anthophyllite fiber in the workplace) and not all of the asbestos fiber types produce the same attack rates for bronchogenic carcinoma. While it is true that the three major varieties of asbestos used in the United States induce both types of malignant tumors in experimental animals, industrial manipulation may alter outcome;

- There is an attribution to a study conducted by my colleagues here in the Environmental Sciences Laboratory which revealed excess cancer mortality "in workers with short-term exposure--one month or less of employment in an asbestos plant." This citation appears at the end of a paragraph which states that "People living in the area of asbestos mines, mills, factories and even shipyards and family members of asbestos workers have become victims of mesotheliomas despite what we would be considered light exposure to the dust." The asbestos plant in Paterson, New Jersey is believed, by many (including Seidman of the American Cancer Society) to have had workplace exposures approaching 100f/ml. Nicholson occasionally uses a figure of 35, I have used a figure of 50 but it is generally conceded the exposure levels were very high indeed. One month's exposure during the second World War translates into a 500-1,000f/ml exposure day (10-hour shifts during the second World War). Using a 20-day work month this computes to a 10,000-20,000f/ml exposure month. This would correspond to approximately a 2-5 year exposure in the occupational setting at the present 2f/ml level. No one would consider this a "trivial" exposure. No one would consider this a "bystander" or "environmental" exposure;

- In the very next paragraph, on page 8 of the petition, the work of Harries, carried out in the Devonport shipyards of Great Britain, is used to support their argument that mesotheliomas may occur from "bystander" exposures. Harries and colleagues showed that the tearing out of asbestos fiber on shipboard results in aerosols which may approach 1,000f/ml in the immediate environment! Fiber measurements made during tear-out operations, aboard ship, have shown tens of f/ml of air in areas removed from the actual site of rip-out. Again, this is included to support the argument of the petitioners that "indirect occupational exposure" was sufficient to produce mesotheliomas many years later. These indirect measurments are considerably greater than those encountered in most workplaces today. Additionally, the fiber types encountered in

the Paterson insulation plant was amosite and the fiber encountered by Harries in the Devonport shipyard was predominantly crocidolite. The mesothelioma frequency for both these fiber types exceeds those reported for chrysotile in every study the world over. We are dealing with mesothelioma risk for different fiber types. Although chrysotile asbestos does produce human mesothelioma one cannot use the experience of workers exposed to crocidolite and amosite as a "measure of risk for chrysotile exposure;"

- Although asbestos fiber released from automobile brakes may increase the asbestos levels encountered in urban air the report of a mesothelioma in a toll collector is not evidence of an etiological association;

- I will support the contention of the petitioners that there are operations encountered during brake maintenance and repair which release "dangerous levels of asbestos during brake servicing;"

- The general case for risk to non-occupationally exposed persons is based on risk assessment models. For example, Nicholson (1983) has provided the Environmental Protection Agency with a document entitled "Asbestos Health Effects Update." Using this model a general scenario is generated for both lung cancer and mesothelioma and extrapolations are made concerning risk to populations for which no exposure data exist. Eleven studies provide lung cancer data whereas four studies provide the mesothelioma data base. The lung cancer studies provide fractional increases of lung cancer risk per unit of exposure but these fractional risks vary considerably depending on fiber type and industry. So it is with the mesothelioma data set which is based on a number of fiber types integrated to yield a single risk model. The question to be raised is whether data for crocidolite, amosite and mixed fiber types (the insulation worker data) may be used for chrysotile. This, coupled with the relatively poor workplace fiber measurements, yields a model which may be used only in its broadest context. These shortcomings were raised in a recent NRC document (1984) where ranges of risk were provided rather than a single value. Again, the mesothelioma data (I stress mesothelioma because segments of the population will live long and this will be the important disease on the basis of time) is based on insulation workers (mixed fiber), chrysotile textiles, amosite insulation, and asbestos cement product manufacture. The lowest values exist for chrysotile exposure in textile plants. Therefore, all of the asbestos deaths projected into the future are based on risk assessment for mixed fibers in a range of circumstances. The variance associated with these projections are large. The numbers of deaths attributable to asbestos brake pad maintenance amounts to some 500 per year over the next several decades. This value may be lower or even greater than given. We simply do not know;

- The only pleural mesothelioma described in a brake repair worker, in which only chrysotile asbestos fiber was observed in the pulmon-

UCC 004208

ary tissues, was reported by myself. I believe this working population to be at risk but I also believe this risk to be less than encountered with other fiber types in other work environments;

- Where are the data to show that substitute friction product materials are "safe"? What happens to e.g., aramid fiber when sheared from a brake surface?;

- Why is "sintered metal" compositions considered safe? Sintered hard metal materials are now implicated in pulmonary disease (hard-metal disease).

I agree with the petitioners that there are many instances where brake maintenance and repair subjects workers to asbestos fiber exposure which increases their neoplastic risk. There is no question that this occurs and that the risk is measurable. I similarly believe that there are no data to support that such risk exists for the general population. Exposure to the general population may occur and that the control of asbestos emissions should be instituted. However, we have no data to support the contention that it represents a "public health hazard." I believe it would be prudent to prevent such exposures from occurring to the general population even though there are no data to support that such exposures are hazardous. The federal courts have noted that one may rely on "general data" in making certain decisions. Therefore, if this petition is to be considered as a "general approach" to the asbestos problem then, presumably, the Environmental Protection Agency will base its decision on courtroom law. As it now stands, one can only appeal for the decision to be based on the concept of "prudence" rather than science. The decision should be made on the social basis and not a pseudo-scientific one.

This issue is a very important one and should not be approached in an unscholarly fashion. My strongest objections focus on the misuse of data and the uncritical use of these data in supporting certain contentions. It is a form of quasi science which we, as environmental scientists, cannot afford or allow to go unchallanged as it reflects upon all of us and our work.

Very truly yours,

Arthur M. Langer, Ph.D.
Associate Professor of Mineralogy
Associate Director, Environmental
Sciences Laboratory

AML:jm

C2873