UCC

UNION CARBIDE

INTERNAL CORRESPONDENCE

METALS DIVISION

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To (Name) Mr. E. A. Piersall
Division UCC - Metals
Location Grand Junction, CO

Date October 31, 1978
Originating Dept. "Calidria" Asbestos
Answering letter date

Copy to Messrs. R. G. Beverly (w/e)
G. J. Hanks, Jr. "*
E. W. Kantz "*
J. L. Myers "
File

Subject Regulation of Taconite Tailings as Asbestos

Attached, for your information, is more on the EPA actions to regulate all mineral fibers as though they were asbestos. So far we have been able to convince their RECRA people that asbestos is already heavily regulated under NESHAPS and should not be also placed under RECRA. There are problems, however, in that asbestos is on the "priority list" that came out of the EPF/EPA court settlement and it is conspicuous by its absence.

Best regards,

Harrison B. Rhodes

HBR/rmm
Attachment

*J/M letter 10/9/78 only

A20704

4.3.5.2

Johns-Manville
Sales Corporation

Ken Caryl Ranch
Denver, Colorado 80217
(303) 979-1000

October 9, 1978

Mr. Joseph Padgett, Director
Strategies and Air Standards Division
U.S. Environmental Protection Agency
Research Triangle Park, NC 27711

RE: Roy Albert's June 28, 1978 Recommendation Regarding the Regulation of Taconite As An Asbestos Cancer Hazard

Dear Mr. Padgett:

EPA's Carcinogen Assessment Group recently sent us a copy of Philip Cook's June 1, 1978 "DRAFT REPORT - ASSESSMENT OF POTENTIAL ENVIRONMENTAL HEALTH HAZARD ASSOCIATED WITH AIR-BORNE MINERAL FIBERS EMITTED DURING TACONITE ORE PROCESSING." In addition, we received a copy of Roy Albert's June 28, 1978 memorandum to you, setting forth his recommendation that EPA should regulate taconite as an asbestos cancer hazard.

A copy of Johns-Manville's comments on Dr. Cook's draft report and Roy Albert's recommendation is attached for your careful consideration.

We are distressed that Roy Albert, as Chairman of EPA's Carcinogen Assessment Group, would use Dr. Cook's draft report as a basis for recommending that taconite be regulated as an asbestos-cancer hazard. Not only does Dr. Cook's report present a superficial and incomplete assessment of the present state of knowledge, the assessment also contains various inaccuracies which we have noted in our comments. In addition, we question the propriety of relying on any health hazard assessment prepared by one who is trained as a physical inorganic chemist, rather than by one with appropriate scientific credentials.

Furthermore, we disagree with Roy Albert's conclusions regarding the potential health effects of short fibers. There is both human and animal evidence to show that short fiber lengths of asbestos are not fibrogenic or carcinogenic. This evidence is not "suggestive" or "indecisive" as characterized by Roy Albert. Our attached comments refer to the various animal studies which clearly demonstrate that these short fibers are biologically inactive. In addition, McDonald's study of the Homestake Mine found no excess of bronchogenic cancer, or any other tumor, in this population exposed to short fibers. As indicated in our comments, although the

NIOSH study did report an excess of bronchogenic cancer in a smaller cohort of this population, that study is so plagued with scientific inaccuracies that it is being repeated.

It is our hope that EPA would undertake a more complete and objective assessment of the potential environmental health hazards associated with airborne mineral fibers emitted during taconite ore processing before making any decision with respect to the possibility of regulation.

It is our understanding that short fibers of the cummingtonite-grunerite series of minerals are commonplace in the general mining industry. Their presence in taconite ore is not unique. Therefore, if EPA were to accept Roy Albert's recommendation on its face, much of the mining industry would needlessly be regulated as posing an asbestos-cancer hazard.

We would be glad to discuss any questions which you may have regarding the enclosed comments.

Very truly yours,

Richard P. Carter, Manager
Government Affairs
Health, Safety & Environment Department

Enc.

RPC/lc

cc: S. Gage
S. Jellinek
W. Barber
M. James
R. Carton
R. Albert
D. Goodwin

bcc: P. Kotin, M.D.
J. Autry
J. P. Leineweber
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B. J. Pigg

A20706

JUN 2 6 1978

ASSESSMENT OF POTENTIAL [illegible]

SUBJECT: Asbestos Cancer Hazard

FROM: Roy E. Albert, Chairman
Carcinogen Assessment Group

TO: Joseph Padgett, Director
Strategies and Air Standards Division

This is in response to your memorandum of May 3, 1978 requesting CAG assistance in determining whether EPA should regulate taconite as an asbestos cancer hazard. In our opinion the answer should be in the affirmative. The attached report by Dr. Cook summarizes the present state of knowledge. In essence, there is suggestive evidence that short fiber lengths of asbestos may not be fibrogenic or carcinogenic so far as mesothelioma is concerned but the evidence is not decisive and there is no evidence one way or the other regarding the effect of fiber length on the induction of bronchogenic cancer which is the main health hazard.

Attachment [illegible]
Duluth, Minnesota

cc: S. Gage
S. Jellinek
W. Barber
M. James
R. Carton
E. Anderson
CRU/2 copies
CAG Reading

EAnderson/dj/6/27/78

A20707

ASSESSMENT OF POTENTIAL ENVIRONMENTAL HEALTH HAZARD ASSOCIATED WITH AIRBORNE MINERAL FIBERS EMITTED DURING TACONITE ORE PROCESSING

DRAFT REPORT

June 1, 1978

Philip H. Cook, Ph.D.

ENVIRONMENTAL RESEARCH LABORATORY-DULUTH
U.S. Environmental Protection Agency
6201 Congdon Boulevard
Duluth, Minnesota 55804

fibers* in airborne dust derived from mining, milling, and use of asbestos are known to cause asbestosis, lung cancer, pleural and peritoneal mesothelioma, and gastrointestinal tract cancer in man. Chrysotile and the amphiboles crocidolite and amosite are associated with all these diseases while anthophyllite mined in Finland is associated with an excess lung cancer risk and high incidence of pleural thickening and calcification (1). Non-occupational exposure to asbestos in the neighborhoods of industrial sources (2) or from household contact with asbestos workers (3) leads to increased risk for mesothelioma.

Fibrous amphibole minerals released from rock during non-asbestos mining operations pose a less well-defined risk of disease when inhaled. Since little or no epidemiological or animal study data exists which can be applied specifically to each occurrence, risk assessment often depends on comparison of physical and chemical properties to asbestos and on comparison of airborne fiber concentrations to concentrations associated with asbestos disease.

Excess cancer and pneumoconiosis deaths have occurred for workers exposed to aerosols of talc contaminated with tremolite, anthophyllite, and chrysotile (4). A study of underground miners in a gold mine with grunerite fibers has demonstrated a three-fold excess of respiratory cancer mortality (5) but a subsequent study (6) of a larger cohort from the same mine (not limited to underground workers) indicates no excess of respiratory cancer deaths.

What basis exists for associating asbestos health hazards with mineral fibers released by taconite mining and beneficiation? Because of their unique structure, chrysotile fibers are certain to be identical morphologically, structurally, and chemically to various chrysotile fibers released by chrysotile asbestos mining and milling. Comparison of amphibole fibers to known amphibole asbestos materials is more difficult because the various amphibole minerals are formed with both fibrous (asbestiform) and non-fibrous habits. It is also probable that a gradation exists between extremely fibrous and non-fibrous amphibole occurrences.

A further complication is the propensity of amphibole minerals to cleave, or split along preferred crystal planes, when crushed and thus produce particles with length to diameter ratios in excess of 3:1. Amphibole fibers produced from amphibole asbestos such as amosite may also result from cleavage, however. No biological data exists to show different response to samples of amphibole fibers which differ only in subtle internal crystal defect structures or in crystal face orientations although these properties may be related to the asbestiform nature of the mineral sample.

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*The term "fiber" in this discussion refers simply to any free particle with a length to width ratio equal or greater than 3:1.

A current popular theory among asbestos health experts is that any long, thin durable fiber introduced into tissue will induce malignant neoplasms. Stanton (7) implanted 17 fibrous glasses of different dimensional distributions into the pleurae of rats and observed that fibers less than or equal to 1.5 μm in diameter and greater than 8 μm in length yielded the highest probability of pleural sarcomas. Extrapolation of these results to fiber carcinogenesis in man is subject to consideration of the influence of fiber dimension on aerodynamic behavior, penetrability, and clearance from the lung; i.e., some of the potentially more carcinogenic fiber sizes may have greatly diminished abilities to reach target tissue. The generally accepted greater cancer risk associated with crocidolite fiber inhalation as compared to chrysotile fiber inhalation may result primarily from greater lung retention of crocidolite fibers.

Asbestos aerosols invariably have a wide range of fiber lengths and widths. Even animal experiments can not provide absolute effect/fiber size relationships since asbestos samples of uniform fiber size have not been prepared. Rats intraperitoneally injected with short chrysotile fibers (95% less than 5 μm in one test and 99% less than 3 μm in a second test) incurred approximately a 40% incidence in tumors, in a study (8) which provides the closest association between short fibers and carcinogenesis. Pulmonary tissues of asbestos workers contain many more short fibers than long with an even shorter fiber size distribution in pleural tissue (9).

The Environmental Protection Agency's animal tests of amphibole fibers obtained from Reserve Mining Company (RMC) will not be completed until 1980. These tests involve intratracheal installation and intrapleural injection of RMC amphibole fibers and amosite fibers in rats (10). The amphibole fiber samples used are representative of fibers found in air samples from Silver Bay but not typical of all amphibole fibers present. In vitro tests of RMC amphibole, amosite, crocidolite, chrysotile, anthophyllite, tremolite, and a non-fibrous grunerite indicate that the RMC amphibole is lytic to sheep erythrocytes and depresses rabbit alveolar macrophage cellular viability similarly to the amphibole asbestos samples whereas the non-fibrous grunerite sample is not active. Cytotoxic effect in such in vitro tests has been linked to the ability to cause fibrosis but is not necessarily associated with carcinogenesis.

Mineral fibers with structures and surface properties entirely different than the asbestos minerals can be carcinogenic. Palygorskite, a fibrous clay mineral, induced as many mesotheliomas, following intraperitoneal injection in rats, as chrysotile (11). The extraordinary incidence of mesotheliomas in a Turkish village (12) is now thought to be a result of zeolite fibers released into the environment by erosion and use of volcanic tuff (13).

Given the chemical, crystallographic, and morphological similarity of RMC amphibole fibers to asbestos fibers, it is most likely that inhalation exposure to high concentrations of these fibers would produce asbestos disease. It is important to note that at this time no evidence of fibrosis in RMC employees with more than 20 years employment is found by pulmonary function studies and chest radiographs (14). These studies can not be extended to conclude that increased risk of lung cancer and mesothelioma is absent but indicate that exposures to fibers are considerably lower than those experienced by asbestos workers in the past and associated with asbestosis and cancer. NIOSH spot dust inspections for mineral fibers indicate time weighted average fiber concentrations in RMC working areas are far below the occupational health standards for asbestos and even further below the concentrations asbestos workers were exposed to decades ago.

The community air amphibole fiber concentrations in Silver Bay, Minnesota have now been well characterized by transmission electron microscope analysis of air samples collected on membrane filters. Amphibole fiber concentrations at three sites in residential Silver Bay were determined from continuous air monitoring to average approximately 125,000 fibers per cubic meter for the years 1974, 1975, and 1976 (15). The electron microscope analysis was performed with a technique essentially identical to the EPA provisional method published in August 1977 (16) and was in excellent agreement with results reported independently for 12 air samples by the Minnesota Department of Health and Mt. Sinai School of Medicine Environmental Sciences Laboratory. Three other laboratories participating in the interlaboratory comparison each used inferior analysis techniques and produced data which failed to correlate with any other laboratories' results (17). Air samples recently collected by Region V during and after a prolonged shutdown of Reserve Mining Company are currently being analyzed and appear to show very low amphibole fiber concentrations in the Silver Bay air during the shutdown period. Although stack emission controls being installed should greatly reduce fiber concentrations, fugitive dust from the on-land tailings basin to begin operation in 1980 pose an unknown addition to amphibole fiber concentrations in the air of the Silver Bay area.

The dose response equation for human inhaled fiber exposure is not well established and the existence of an exposure threshold, below which increased cancer risk is zero, is unknown. Amphibole fiber concentrations in the air near the taconite processing facility at Silver Bay, Minnesota are probably at least 1,000 times lower than past concentrations in the asbestos industry which led to high incidence of asbestos disease. Evaluation of the environmental hazard, however, requires consideration of factors such as 24 hour per day exposure, exposure initiation at infancy, potentially longer period of accumulated exposure, and acceptable environmental health risk versus acceptable occupational health risk. A compounding problem for assessing cancer risk at lower exposure levels is the increased lapse time between initial exposure and onset of disease (18).

The new available monitoring, health, and mineralogical information should not change EPA's originally stated concern for the long-term health of persons living in the vicinity of taconite processing facilities which handle ore highly contaminated with amphibole or chrysotile fibers. Emissions to the environment of such fibers should be controlled with best available technology. Such controls are planned for installation at Reserve Mining Company. It should be noted that, while the 8th Circuit Court decision of March 14, 1975 stated:

> "The best that can be said is that the existence of this asbestos contaminant in air and water gives rise to a reasonable medical concern for the public health. The public's exposure to asbestos fibers in water and air creates some health risk. Such a contaminant should be removed,"

the findings of fact of the United States District Court in the Reserve Mining Trial were not reversed or modified by the 8th Circuit Court. These findings of fact included:

1. "Many of these fibers are morphologically and chemically identical to amosite asbestos and an even larger number are similar to amosite asbestos."

2. "Exposure to these fibers can produce asbestosis, mesothelioma, and cancer of the lung, gastrointestinal tract, and larynx."

3. "The discharge into the air substantially endangers the health of the people of Silver Bay and surrounding communities as far away as the eastern shore in Wisconsin."

4. "The present and future industrial standard for a safe level of asbestos fibers in the air is based on the experience related to asbestosis and not to cancer. In addition its formulation was influenced more by technological limitations than health considerations."

5. "The exposure of a non-worker populace cannot be equated with industrial exposure if for no other reason than the environmental exposure, as contrasted to a working exposure, is for every hour of every day."

6. "While there is a dose-response relationship associated with the adverse effects of asbestos exposure and may be therefore a threshold exposure value below which no increase in cancer would be found, this exposure threshold is not now known."

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COMMENTS BY JOHNS-MANVILLE CORPORATION ON THE JUNE 1, 1978 "DRAFT REPORT - ASSESSMENT OF POTENTIAL ENVIRONMENTAL HEALTH HAZARD ASSOCIATED WITH AIRBORNE MINERAL FIBERS EMITTED DURING TACONITE ORE PROCESSING" BY PHILIP M. COOK, Ph.D.

Initially, the title of the draft report leads the reader to believe that the report will provide an assessment of the potential environmental health hazards associated with airborne mineral fibers emitted during taconite ore processing. However, the report is no more than a collection of references from which the implication is made that there may be a health hazard associated with such emissions. In addition, it is incongruous why an individual with Dr. Cook's background was chosen to prepare a document of this nature. Dr. Cook's training as a physical inorganic chemist in no way prepares or qualifies him to evaluate potential health hazards that might be present. Furthermore, it is likely that Dr. Cook is not without bias in his assessment of this situation, since he was the principal technical advisor to the federal government during portions of the Reserve Mining trial in 1973. This incidentally took place only one year after Dr. Cook received his doctorate from the University of Wisconsin.

Set forth below are comments relating to specific paragraphs of Dr. Cook's draft report.

Page 1, paragraph 1. In essence, this paragraph is an all-encompassing statement which implies that any type or level of exposure to asbestos fibers can lead to an increased risk of disease. Dr. Cook makes no attempt whatsoever to place the various types of exposure, i.e. occupational, para-occupational, and non-occupational in proper perspective as far as exposure levels and potential and actual risk of disease are concerned. Manifestations of asbestos-related diseases have not been associated with low level exposures to asbestos fibers, as is implied by Dr. Cook. The occurrence of asbestos-related diseases is dose related in terms of fiber levels and years of exposure.

There is increasing evidence to indicate that even in para-occupational situations, those who have developed asbestos diseases did receive high doses to fibers. The first paragraph erroneously implies that household exposures to asbestos have been minimal in the dose-relationship concept. It is likely that these exposures were substantial. As recognized by Selikoff and others, the impregnation of drapes, rugs, furniture, etc. with asbestos fibers and the constant resuspension of fibers in the respirable range creates an exaggerated hazard. These household exposures provide an opportunity for repetitively high, short peak exposures due to the shaking out of work clothes. Lacking specific dust counts

over the appropriate time period, any conclusion that these exposures were minimal is totally unacceptable. Once asbestos is carried home by the workmen, it accumulates in the home; and its presence in the home is likely to become permanent. For example, it gets into the rugs, from which it becomes resuspended by movements such as brushing and walking. In consequence, family members are getting a 24-hour a day, 7-day a week exposure, relatively speaking, rather than an interval exposure. Furthermore, in the home environment, an exaggerated opportunity is present for co-factors to be operating, such as smoking and other household pulmonary insults.

At the New York Academy of Sciences June 1978 Science Week Conference, E. Cuyler Hammond, reported on a study which he conducted with Dr. Selikoff and others which was aimed at finding out if exposure to small amounts of asbestos will lead to any long-term harmful effects. This study traced the fates of 5,550 men who lived in the community near an asbestos plant in Paterson, New Jersey between 1942 and 1954. The researchers stated that it was safe to assume that people living in that community were exposed to asbestos. Samples of settled dust collected from the attics of houses near the factory still contained appreciable numbers of amosite asbestos fibers. However, the researchers failed to find any unusual incidence of cancer among these asbestos-exposed people. This is evidence that a low-level exposure to asbestos has not led to any increased incidence of malignancies.

Page 1, paragraph 2. The unqualified statements in paragraph 1 are followed in the second paragraph by a greatly over-simplified statement on risk assessment by Dr. Cook, which in contrast includes a statement which implies that fiber concentrations are important in relationship to the risk of disease. As Dr. Cook has noted, the physical, chemical, and morphological properties of fibers are extremely important in comparing incidence of disease from one location to another. However, it is extremely important that other factors, which may have influenced the occurrence of disease, also be taken into account. An example of this is the relatively large difference in the incidence of disease which is found in miners and millers of asbestos fiber, as compared with insulation workers, which indicates that co-factors may play an important part.

Page 1, paragraph 3. We are at a loss to appreciate the relevance of the reference in the third paragraph to the study by Kleinfeld, et al, on exposures to asbestos in the talc mining and milling industry. These were not exposures to short "asbestos-like" fibers of the type encountered in taconite operations, but represented exposures to fibers of the type which have been shown in other situations to be capable of producing disease when present in high enough concentrations. While it is agreed that occupational exposures to tremolite and anthophylite of sufficient dose and duration can produce asbestos disease, we do not see how this is relevant for inclusion in an assessment of the potential environmental health hazards associated with airborne mineral fibers emitted during taconite ore processing.

A20803

Reference is then made to two studies of the Homestake miners who have been exposed to cummingtonite-grunerite fibers of relatively short dimensions.

The first paper reports the research efforts of Drs. Gillam, Dement, Lemen, Wagoner, and their associates at NIOSH. The authors of this study reported an excess of both lung cancer and pneumoconiosis in this population. They attributed the excess to the presence of a form of asbestos in cummingtonite-grunerite ore. The NIOSH group totally dismissed the possibility that this excess was caused by other potential lung cancer hazards present in the mine, such as diesel exhaust, arsenic and radon daughters. Cigarette smoking habits were ignored. The increase in fibrosis was erroneously attributed to asbestos, rather than to free crystalline silica, known to be present in the mine in substantial quantities.

The Gillam, et al paper is fatally flawed due to its failure to understand the problems inherent in studying a small cohort. The authors' claim of excessive rates in the asbestos-related malignant and non-malignant disease categories is clearly based on poor and incomplete data analyses. Even if the claim were based on valid analyses, data to incriminate asbestos, rather than one or a combination of other coexisting materials in the causation of cancer, are entirely lacking. To ascribe the excess of non-malignant respiratory disease to asbestos and ignore the known exposure to high levels of free crystalline silica in the past, confirmed by the frequent diagnosis of silicosis on the death certificates, borders on irresponsibility. To ignore the potential for carcinogenic and co-carcinogenic effects resulting from the mixed exposure to silica dust, arsenic fumes and particles, blasting powder fumes and radon daughters, and to arbitrarily ascribe all of their excess of cancer to asbestos particles is manifestly irresponsible and has no justification in the methodology of science. As a result of the serious concerns raised by Johns-Manville and others regarding the NIOSH methodology and conclusions, this study is currently being repeated by NIOSH.

As Dr. Cook properly notes, a subsequent study by McDonald of a much larger cohort from the same mine indicates no excess of respiratory cancer deaths. The McDonald study has greater scientific credibility due to its inclusion of a much larger population sample. Finally, it should be noted that neither NIOSH or the McDonald study found any excess of mesothelioma or gastrointestinal tumors.

Since the McDonald study did not find any excess incidence of bronchogenic cancer, mesothelioma, or gastrointestinal tumors, and since the Homestake Mine environment included fibers virtually all of which were less than 5 micrometers in length, one can conclude that long-term exposure to amphibole fibers with such physical dimensions, at least at the dose levels reported, does not induce the development of asbestos-related disease.

A20807

Page 1, paragraphs 4 and 5. In these two paragraphs, Dr. Cook implies that it is extremely difficult to extrapolate from the known biological activity of chrysotile and various amphibole fibers to minerals which may be similar but not identical in crystalline form or chemical composition. While this may be true, based on the work of Dr. Stanton and others, it is the physical dimensions of the fiber that is of primary importance, whereas subtle differences in chemistry and physics are of secondary importance.

Page 2, paragraph 1. In the first paragraph of the second page, Dr. Cook refers to the work of Dr. Stanton and characterizes his findings by stating that "fibers less than or equal to 1.5 μ in diameter and greater than 8 μ in length yielded the highest probability of pleural sarcomas." Dr. Cook has characterized Dr. Stanton's findings in a rather incomplete manner, as Dr. Cook's characterization leaves one with the impression that fibers less than 8 micrometers in length can yield tumors in these animal studies. However, such a characterization is not supported by the findings of Dr. Stanton and others.

For example, Stanton's fibrous glass studies show a high correlation between the probability of tumor production and the size of fibers; the probability rising as the numbers of fibers thinner than 1.5 microns and longer than 8.0 microns is increased. Stanton's data show that the low tumor yield group and especially the zero yield group were treated with glass fibers that were either virtually 100% less than 8 microns in length or were thicker than 1.5 microns. In reporting the results of his studies, Stanton stated:

> "The results of these experiments raised at least two points that merit discussion, the relationship of fiber dimension to mechanisms of carcinogenesis and the relevance of findings to human exposure. An earlier paper proposed that the fibers at the lower range of optical visibility (i.e. diameters $< 1.5\mu$) that were exceptionally short (i.e. lengths $< 5\mu$) might account for the carcinogenicity of several types of glass and asbestos fibers (1). This hypothesis was not supported by subsequent data (2), which along with that of the present report indicates that the fine diametered fibers that are very long, are carcinogenic and probably become more carcinogenic as their length increases. The negligible carcinogenicity of short fibers is perhaps related to the histological observation that virtually all coarse and fine diametered fibers with lengths of 8μ or less are efficiently entrapped by phagocytes. Many of the smallest particles are transported to regional lymph nodes, but even those that are not seem completely sequestered within the cytoplasmic limits of macrophages and foreign-body giant cells at th- site of implantation. Similar observations have been made with chrysotile fibers in vitro (8)."[1]

A20806

[1]Stanton, M. F., Laynard, M., Miller, M., May, M., West, E.: Carcinogenicity of Fibrous Glass: Pleural Response in the Rat in Relation to Fiber Dimension. J. Nat'l. Cancer Inst., 58:587-603 (1977).

Using fibrous glass samples of unusual specificity placed directly into the pleura of hamsters, Smith has obtained information quite similar to that reported by Stanton. Smith confirms Stanton's findings as to the strong relationship between length of the fibers and tumor development. However, Smith found no tumors with a sample in which 98% of the fibers were 10 microns or less in length.

Animal studies by Pott, Wagner, Davis and others have also reported the induction of mesothelioma following the introduction of long, thin, durable fibers of various kinds into the pleura or peritoneal cavities. All of these researchers have induced tumors in animals by this method using a variety of durable fibers such as asbestos, fibrous glass, ceramic fibers, aluminum whiskers, and other materials, whenever sufficient numbers of long, very thin fibers were introduced. All of these researchers have been unable to induce tumors when only short and/or thick fibers are introduced.

In the middle of the first paragraph, Dr. Cook implies that long fibers, which have carcinogenic potential, have greatly diminished abilities to reach target tissue. This inference by Dr. Cook is contrary to findings of long fibers in both animals and man in all areas of the lung following inhalation exposures to long fibers. On the other hand, short fibers are quickly removed from the lung by the mucociliary and macrophage system. The only case where Dr. Cook's inference could be true is in the case of long chrysotile fibers which are curly and present a larger cross-section. This larger cross-section causes the fibers to be trapped in the bronchial system, rather than permitting them to penetrate to the distal portions of the lung.

Page 2, paragraph 2. In this paragraph, Dr. Cook continues with his short fiber hypothesis. With regard to the study by Pott, Hugh and Friedrichs referred to by Dr. Cook, it has now been established that the sample used in that experiment contained a sufficient quantity of long fibers (greater than 10 micrometers in length) to induce mesothelioma. This, together with the massive doses of fibers which were used, can easily account for the tumors which were found in the animals. The reference to the work of Bignon, presented at the National Bureau of Standards in 1977, is another example of the presence of short fibers in the target tissue, with the unfounded presumption that they were responsible for the disease. This is extremely poor logic, in that the mere presence of an agent does not necessarily mean that it was responsible for biological changes. Again, the significance of the presence of long fiber in the pleural tissue of these workers is neglected by Dr. Cook.

Page 2, paragraph 3. In the third paragraph, Dr. Cook refers to the work now being carried out by Dr. Pelekar using amphibole fibers obtained from the Reserve Mining Company. First of all, the fact that these fibers cause hemolosis of sheep erythrocytes

and depress the activity of rabbit alveolar macrophages in no way can be related to their biological activity in the whole animal. There have been many attempts to make such blanket correlations, but they have failed. Dr. Cook does correctly state that the cytotoxic effect in in vitro tests have not been associated with carcinogenesis.

Unfortunately, Dr. Cook fails to note that the fiber samples being used in these tests are not at all representative of the fibers which are emitted from the Reserve Mining Company's taconite processing plant. These fibers were produced from a geological curiosity found by IITRI personnel during a very thorough exploration of the Reserve Mining pit. This sample represents an almost asbestiform occurrence of the mineral cummingtonite, which is normally carried through the processing system. It was ground to produce a sample which contained fibers, 85% of which were less than 5 micrometers in length. The remaining 15% contained fibers up to 100 micrometers in length. By comparison, the airborne fibers from Reserve's taconite processing plant contained probably less than 1% of fibers greater than 5 micrometers in length. To the best of our knowledge, there are no reports of fibers in excess of 10 micrometers in length. Therefore, the fibers being tested by EPA, as referred to by Dr. Cook, are not representative of actual human exposure to amphibole fibers emitted from the Reserve Mining Company's taconite processing operations. Since these fibers are not representative of human exposures, the data which ultimately will be forthcoming from these tests will not be useful in assessing the potential environmental health hazard associated with airborne mineral fibers emitted during taconite ore processing at the Reserve Mining Company's operations or elsewhere. It is interesting to note that Dr. Cook only refers to Reserve Mining's taconite operations as if there were no other taconite processing operations in this country. Based on the title of his draft report, one assumes he would have examined emissions from other taconite operations.

Page 2, paragraph 4. In the final paragraph on page 2, Dr. Cook refers to two studies in which minerals other than asbestos have produced mesothelioma. However, neither of these studies are supportive of Dr. Cook's short fiber hypothesis, since in both studies sufficient concentrations of long, thin fibers were present to produce mesotheliomas.

Page 3, paragraph 1. We are assuming that in the first sentence of this paragraph, Dr. Cook is referring to the amphibole fibers which were specially obtained for EPA's studies. However, as previously indicated, these fibers are not representative of actual human exposure to taconite emissions.

Dr. Cook is correct in noting that at this time there is no evidence of fibrosis in Reserve employees with more than 20 years' employment. Dr. Cook further notes that these studies cannot be extended to conclude that increased risk of lung cancer and mesothleioma is

absent. While Dr. Cook is correct in this regard, he attributes this solely to the low exposures to fibers. While Dr. Cook may be correct, he totally ignores the possibility that the fibers to which the Reserve employees were exposed are too short to induce fibrosis. Dr. Cook also fails to note that in other populations occupationally exposed to asbestos for periods of less than 20 years, an excess incidence of both lung cancer and mesothelioma have been reported. It is irresponsible for Dr. Cook to fail to even consider the possibility that the dimensions of the fibers to which the Reserve employees are exposed are such as to render them biologically inactive, regardless of the level of exposure.

Page 3, paragraph 2. In the second paragraph on page 3, Dr. Cook is guilty of not being objective about his work. In effect, he is saying that anyone who gets results which agree with him is doing good work, whereas others are using "inferior analytical techniques." There is considerable debate still going on regarding the reproducability of analyses of environmental fiber concentrations. EPA is well aware of the problems in this area, and has reached the conclusion that it is impossible to regulate fiber emissions into the air without a reliable analytical method. Experts in this field generally agree that the Environmental Sciences Laboratory at Mt. Sinai in New York and Dr. Cook's laboratory in Duluth generally obtain higher fiber counts than other laboratories. Regardless of the accuracy of Dr. Cook's reported findings, we are unable to determine the relevance of the statements he makes in this paragraph to an assessment of potential environmental health hazards associated with airborne mineral fibers emitted during taconite ore processing.

Page 3, paragraph 3. In the last paragraph on page 3, Dr. Cook makes the statement that "the dose response equation for human-inhaled fiber exposure is not well established..." To the contrary, the data do show a dose-response relationship with regard to asbestos exposures, even though the precise level at which there will be no adverse impact on morbidity and mortality is not known. However, we believe that a critical review of the best available evidence indicates that a time-weighted average health standard of 2 F/cc will not have any adverse impact on the morbidity or mortality of individuals occupationally exposed to asbestos. As Dr. Cook recognizes, the amphibole fiber concentrations in the air near the taconite processing facility at Silver Bay, Minnesota are far less than the current OSHA permissible exposure level of 2 F/cc.

Dr. Cook then goes on to imply that there may indeed be a health hazard due to the consideration of such factors as 24-hour per day exposure, etc. However, there is no foundation upon which to even base an implication of a possible hazard. Two paragraphs earlier, Dr. Cook notes that there is no evidence of fibrosis in Reserve Mining Company employees with more than 20 years' employment. Yet, most of these individuals received a 24-hour per day exposure, if one includes the time spent at home and otherwise in the

neighborhood of the facility. The literature, if showing nothing else, shows that background concentrations of asbestos are unrelated to any increase of disease. In addition, there is no evidence of any excess disease from non-occupational and non-para-occupational exposures to asbestos. As we previously indicated, Cuyler Hammond's recently delivered paper at the New York Academy of Sciences meeting reported no unusual incidence of cancer among people who lived in neighborhoods near an asbestos plant in Paterson, New Jersey, while at the same time the employee population at that plant have exhibited a high incidence of asbestos-related disease.

Page 4. The last page contains many citations from the original Reserve Mining trial and the decision from the U.S. Circuit Court of Appeals. While these may represent findings by the District Court and Appeals Court, they cannot be taken as scientifically factual. It is disturbing to think that EPA, or any other government agency, would even consider making health hazard assessments based on court decisions, rather than scientifica data. Since when has our judicial system become the source of complex scientific determinations?

In summary, we have read with interest this as well as previous publications of Dr. Cook. In manuscripts that describe his analytical studies and other research, we respect his qualifications. We are at a loss, however, to understand or appreciate his qualification to prepare a critical review or assessment of a situation in which "Environmental Health Hazard" is the shibboleth by which regulatory action is proposed. Equally astounding is our information that Roy Albert has used this Draft Report as the basis for the conclusion that a health hazard has been demonstrated incidental to taconite ore processing.

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