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May 31, 1966

TO: H. M. Ball
W. P. Gibbons
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G. H. Martens, Jr.
K. V. Lindell
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F. E. Dutcher
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F. H. May, Jr.
R. F. Orth
J. L. Allen
W. F. Goodwin
R. F. Knapp
G. D. Murray
C. L. Scheckler
Yvan Couture - Asbestos
T. S. Patterson - Toronto DH
Carl Thompson (Hill and Knowlton)

BACKGROUND FACTS
ON THE
ENVIRONMENTAL HEALTH ASPECTS OF ASBESTOS

Each of us is exposed, each day, to the possibility of questions from within or outside the company regarding the various aspects of asbestos and public health. As you know, we developed a "position statement" (in the form of questions and answers) at the time of Dr. Selikoff's March 1 publicity release. Now that several additional attacks on asbestos, from other sources, have occurred, it seems well to up-date it.

Accordingly, we worked with Hill and Knowlton and Dr. Kenneth Smith to produce the attached statement that is recommended for use for

(Con't.)

the time being.

As you will see, this statement does not contain any new "ammunition" for use in rebutting criticism; there simply has not yet been time to develop such ammunition from the literature search phase of our current activity. However, the wording is up-dated to reflect the two most recent public attacks. We'll update and re-issue as additional facts come to our attention.

If you have queries from the press, please refer them to me - or in my absence, to Bill Raines, Director of Public Relations - or Jon Allen, Manager of Press Relations.

F. J. Solon, Jr.

fjs/eb
att.

INTERIM POSITION STATEMENT REVISED 5/31/66
(For use in reply to possible queries)

ENVIRONMENTAL HEALTH ASPECTS OF ASBESTOS

The following may be attributed to Dr. Kenneth W. Smith, Medical Director of Johns-Manville Corporation.

GENERAL COMMENT

No one really knows what causes cancer. Therefore, any medical testimony which points a finger at a potential suspect is understandably of interest and concern to the public.

As an asbestos producer, Johns-Manville conducts medical examinations and maintains thorough records on the health of its employees. J-M records do not indicate that cancer occurs any more frequently among the employees at the company's asbestos mines and asbestos-processing plants than among the general population.

With regard to claims of exposure of the general public to the inhalation of asbestos fibers, as a result of their use in ceiling and floor tile, brake linings, etc., very little scientific evidence is available. Asbestos is not one, but a variety of different minerals. It is important that objective scientific studies be made to determine whether any of the various types of asbestos present potential health hazards to miners, workers, users or the general public.

To that end, Johns-Manville and the Asbestos Textile Institute have been cooperating since 1963 with the U. S. Public Health Service in a study of the asbestos industry. The study is being headed by Dr. Lewis Cralley of the Public Health Service.

Johns-Manville is a member of the Quebec Asbestos Mining Associatio which is also sponsoring a program of medical research on possible effects of asbestos on workers who mine and process it.

(Con't.)

COMMENT IN REGARD TO DR. SELIKOFF'S FINDINGS

Reference (by Dr. Irving J. Selikoff) to an elevated incidence of cancer among those exposed to asbestos dust is based on limited reports relating to a relatively small group of workers who install and/or remove a variety of insulation materials, including some which contain asbestos.

COMMENT IN REGARD TO THE ARTICLE IN THE MAY ISSUE OF THE ARCHIVES OF PATHOLOGY (A.M.A.)

Recent articles speculating about asbestos as an air contaminant call for emphasis on the scientific limitations by the authors themselves.

For example, the article just published in the May issue of ARCHIVES OF PATHOLOGY of the American Medical Association refers to finding "scanty" asbestos in some of 500 autopsies performed. Yet, the authors specifically state that none of the subjects had "pulmonary disability and all died from other causss." The authors do pose some questions they say deserve study.

HILL AND KNOWLTON, INC.
Public Relations Counsel
150 EAST FORTY-SECOND STREET
NEW YORK 17, N. Y.
OXFORD 7-5600

CARL THOMPSON
Vice President

October 7, 1966

Mr. F. J. Solon, Jr.
Vice President
Johns-Manville Corporation
22 East 40th Street
New York, N.Y. 10017

Dear Jack:

The reference to the symposium I mentioned yesterday by phone came from Gershon Fishbein's Environmental Health Letter which said as follows:

"ASBESTOS, PNEUMOCONIOSIS SEMINARS COMING UP:

"Much concerned with the problem of asbestos and its effects on workers, the Public Health Service's Division of Occupational Health will sponsor a seminar on the subject Dec. 7-8 in Cincinnati to bring up to date some of the information which has been accumulating recently.

"Of particular concern is the reported increased incidence of mesothelioma, associated with extensive exposure to asbestos fibers. The Pennsylvania Department of Health is reportedly investigating some clusters of cases in neighborhoods near factories using asbestos or fibers."

Since this has received public distribution, I see no reason not to have our Washington office check further and am asking them to do so.

The other item about the Pennsylvania Department of Health is of significance to you and we know that there have been some recent references to studies done around Lancaster, Pa.

Sincerely,

Carl Thompson

CT:sdd

Draft 8/12/66

Asbestos and Human Health

The march of modern industry brought with it many previously unsuspected health hazards to those opposed to various substances. To achieve maximum protection to workers, industrial medicine has become an important part of industry, a distinct branch of medical practice, and a function of public health authorities at all levels of government. Industry has a continuing role in protecting the health and safety of its workers and the users of its products.

In the asbestos industry -- mining and fabricating -- an industrial health hazard is asbestosis, a recognized disease of the lungs known to medicine for 40 years. Asbestosis is discussed in some detail elsewhere in this brief. At this point it is important to note the following facts:

. Modern methods of industrial hygiene in mines and factories have reduced asbestosis to the point where it is no longer considered a common health hazard for workers.

. Asbestosis only occurs after prolonged and excessive occupational exposure, which is now generally controlled.

. Because of its long history, the medical literature on asbestosis is extensive and both prevention and treatment are well understood.

Recently, however, there have been published, in medical journals and the lay press as well, suggestions that lung cancer may be associated with exposure to asbestos.

The implications of these suggestions are naturally of great concern to the asbestos industry. The industry is concerned with the health of its own employees -- asbestos miners and fabricating workers.

In addition, there is concern also for workers in customer industries -- insulators and construction workers who might use asbestos. Finally, the suggestions involve the health of the general public, however slight may be their exposure and however remote may be the possibility that such exposure might lead to disease.

In the past 20 years, the condition of lung cancer has received increasing medical and public attention because the reported mortality from lung cancer has increased substantially in numbers. It is generally conceded, however, that at least a part of this reported increase is apparent rather than real: That is, it results from expanded knowledge of cancer, tremendously improved diagnostic techniques and procedures, and what a prominent and respected medical journal has called greater "lung cancer consciousness" among doctors. Furthermore, even part of the real lung cancer increase is due to more people living longer, as a result of the greatly decreased mortality from other diseases, particularly infectious diseases. Fewer people die young of tuberculosis for example; they survive to reach cancer-prone old age.

In any case the reported increase in lung cancer cases has resulted in serious and laudable efforts to identify the cause or causes. It is natural and rational that all substances inhaled into the lungs should be among prime suspects. Cigarette smoking has received most of the attention and blame, although general air pollution has also been widely accused. Uranium mining apparently increases the risk of lung cancer. In the search for factors that may be involved, asbestos dust was inevitably added to the list of substances to be studied.

Unfortunately, the tendency, particularly among laymen, has been to equate investigation with incrimination, and there are some

who tend to believe a suspect is guilty until proven innocent -- not innocent until proven guilty.

A few hundred years ago it was generally believed that maggots were spontaneously generated by the heat of the sun, and that the sun rotated around the earth. (Repeated observations seemed to confirm these beliefs.)

A little over a hundred years ago it was believed that cholera and yellow fever were caused by the miasma -- or bad air -- from swamps. Fifty years ago pellagra was thought to be an infectious disease. The observations on which these medical beliefs were based were relatively sophisticated for their time, but the conclusions were as erroneous as the theory of the sun spinning around the earth ! These theories could not be confirmed (by experimentation) and were eventually disproved by experimentation.

Suggestions associating lung cancer with asbestos have neither been confirmed nor disproven by experimentation, nor by many observations of asbestos workers. Without such support, the subject should be regarded with scepticism at the present time.

The fact that the question has been raised, however, has resulted in increased studies to collect evidence necessary to make a determination of what the facts are.

I. What is Asbestos?

Any attempt to assess the effect of asbestos on health is complicated by the fact that asbestos is not a single chemical or physical entity. "Asbestos" is a name given to a group of minerals, not to one particular mineral. The two major groups of these minerals, pyroxene asbestos and amphibole asbestos, comprise various types; chrysotile, crocidolite, amosite, anthophyllite, tremolite, actinolite, brucite and picrolite.

In the United States chrysotile, amosite and crocidolite are used for different kinds of products, but chrysotile is by far the most common. About XX% of the asbestos used in this country is chrysotile, principally from mines in Canada.

Each of these types of asbestos differs from the others, chemically and physically. To add to the complexity, there are at least five different varieties of chrysotile, each with a distinct chemical composition, so that the product of one Canadian mine is not identical with the product of another Canadian mine.

In recent years some of the writers on asbestosis have become aware that there is significance in the different types of asbestos, and they have attempted to specify the type to which asbestosis victims may have been exposed.

Such differentiation has not been as clearly observed by those who theorize on the asbestos-lung cancer association. Their writings tend to speak of "asbestos" generally, ignoring the very real and important differences among the various types, and indeed, disregarding the distinct possibility that the subjects of their studies may not have been exposed to asbestos of any type whatsoever.

Each of the three most important types of asbestos, chrysotile, amosite and crocidolite, exhibit distinguishing characteristics.

. Chrysotile - By far the leading producers are Canada and the Soviet Union. Smaller deposits are found in Rhodesia, China and the United States. Chrysotile, a pyroxene asbestos, occurs where areas of serpentine rock have been cracked by earth movement and subsequently reacted by hot volcanic waters under intense pressure. This reaction transforms the granular serpentine into fibrous chrysotile. Chemically, chrysotile is a hydrous magnesium silicate with a positive electrical charge. Because of this positive charge chrysotile can be attacked by acids. Commercial fibers contain small varying amounts of iron and titanium.

. Crocidolite - The bulk of the world's supply comes from South Africa, although there are small mines in Australia. It is an amphibole asbestos, occurring in iron-rich sedimentary rock. Chemically, crocidolite is a ferrous sodium silicate with a negative electrical charge. It is characterized by a very deep blue color.

. Amosite - The Transvaal district of South Africa is the only place where amosite is found. It is a member of the amphibole asbestos family, occurs in the same type of rock as does crocidolite, and has the same negative electrical charge. Chemically, amosite is a ferrous magnesium silicate.

With these basic differences it might be expected that the three types of asbestos would vary in biological effect, and numerous observations confirm the expectation. There seems to be broad agreement that chrysotile, the most common type in use in the United States, is least likely to produce asbestosis as a result of over-exposure.

There is no proof that any of the three types of asbestos produce lung cancer, in animals or in man. But in any case it should be remembered that data that may implicate, say, crocidolite can not be assumed to apply to amosite, still less to chrysotile.

Wagner ( ) experimented with all three types of asbestos on various laboratory animals, and found that crocidolite produced the most severe reactions and that amosite was more than five times as irritating as chrysotile. He reported, in 1963:

"With chrysotile dust it was possible to produce severe lesions in the lungs of guinea-pigs, slight fibrosis in monkeys, while no significant effect was observed in two rabbits. Amosite dust causes marked asbestosis in all three kinds of animals. Lesions occur more rapidly in guinea-pigs exposed to this dust than in those exposed to chrysotile. There is an indication that the disease is progressive in monkeys and rabbits. Similar pathology to that which occurred in a monkey exposed to chrysotile for 22 months was observed in a monkey exposed to amosite for four months. The impure crocidolite dust caused severe disease in guinea-pigs, and the rate of respiratory infection among animals exposed to this dust was more marked than with the other types. It is possible that this may be due to the high quartz content of the dust.

"Finally, asbestos bodies could be demonstrated in the lungs of animals exposed to all three types of asbestos dust. These were scanty in the animals exposed to chrysotile and usually segmented. Only occasional fibres were observed. In contrast to this, the asbestos bodies in the animals that were dusted with amosite were plentiful, and segmentation was rare; fibres were extremely numerous and far outnumbered the bodies.

Following further investigation, Dr. Wagner offered an explanation for this observation at a medical symposium ( ) in 1965:

"A method of producing asbestos dust clouds has been devised and an animal inhalation experiment carried out to test it... The elimination rate of Rhodesian chrysotile has been found to be three times greater than that of the amosite and crocidolite, which suggests an explanation for the previously observed reduced fibrogenicity of this dust. The reason for the difference in the elimination rate remains to be determined."

At the same meeting Schepers ( ) expressed doubt whether any of the reported lung cancer cases claimed to be associated with previous asbestosis could be identified with chrysotile exposure:

> "My first impression is that there now is less certainty that asbestos inhalation is associated with pulmonary neoplasia than there was ten or twenty years ago. Perhaps this is due to the greatly reduced dust exposures. Asbestos may, after all, prove to be carcinogenic only in overwhelming dosage. Thus the high prevalence of neoplasia which was reported several decades ago may be a function of the severity of exposure rather than an indication of high carcinogenic potency. I suspect that in final analysis the carcinogenicity of asbestos will be rated as of low order...
>
> "...Finally, there is the question of whether inhalation of chrysotile is associated with neoplasia. On critically reviewing the work histories of eleven cases of lung cancer in chrysotile workers, I find that all of these had, at one time or another, also been exposed to other forms of asbestos, mainly amosite or crocidolite. Their predominant exposure was to chrysotile, but since there is such strong evidence incriminating amosite as a carcinogen, the fact that these men also had been exposed to amosite is disruptive of a theory of carcinogenicity of chrysotile per se." ( )

II. What is Asbestosis?

Asbestosis, first described ~~in 1924~~ one of the years ago, is one of a class of diseases called the pneumoconioses, ~~resulting~~ diseases which result when excessive quantities of dust are inhaled, over a long period of time. True asbestosis is characterized by the presence of particles of asbestos lodged in the lung, so that the surrounding lung tissue becomes fibrous. Clinical symptoms are not always present, but in its severe form asbestosis is accompanied by breathing difficulty and in such cases the victim may be disabled.

Of great relevance to the alleged link between asbestosis and lung cancer is the fact that diagnoses of asbestosis itself are still uncertain. Despite the undeniable existence of the disease, and its extensive literature, even the most experienced physicians have difficulty in distinguishing asbestosis from other pneumoconioses and even from completely unrelated chest diseases.

The situation is made more complicated by the fact that pneumoconioses are sometimes caused by a mixture of various dusts, such as ~~cement, carbon or silica~~ silica coal cement or asbestos. If a lung disease patient has been exposed to mixed dust, lives in an air-polluted city and is a heavy smoker, how accurately can the specific cause of his disease be determined?

Much more common than asbestosis is silicosis, caused by the inhalation of stone dust. Silicosis has been proven to be associated with tuberculosis. On the other hand, all evidence to date supports the view that there is no association between asbestosis and tuberculosis. Furthermore, silicosis is progressive, while asbestosis is static. The disease only gets worse through continued exposure to asbestos dust. Finally, there is a difference of opinion among scientists as to whether

the lesions induced by asbestos in experimental animals is identical with human asbestosis or merely resembles the human disease.

The difficulty of diagnosing asbestosis was pointed out by Sander ( ) in 1955, who cited some of the confusing symptoms and reported the varying diagnoses made by a group of physicians in examining X-ray films:

> "All these examples I assume represent patients who present themselves to their physicians with symptoms of dyspnea, with a history of some exposure to asbestos dust, and with chest films of the character I have mentioned. How easy it is to fall into the trap of a gunshot misdiagnosis! Unless other possible causes for the x-ray changes are considered first and a detailed past occupational history is evaluated, along with the character and extent of the most recent dust exposure, another worker who actually needed reassurance will be told that his lungs are full of asbestos dust and that he should quit his trade! From that point on, his symptoms usually increase to a marked degree, and he has developed what to him is a real disability. In my experience, such doctor-induced disability is commoner in some areas than is the disability from the disease itself...
>
> "...A number of my films from the textile mill in North Carolina for which I am consultant, representing cases which I had classified as 'early or first-stage asbestosis,' were called 'essentially negative' by a number of physicians present who had had long experience with this disease. The reverse also was true; some films I had called negative which others thought represented first-stage asbestosis. The same disagreement was found with the other films which were presented. It was our final conclusion that it is impossible to clearly define a first-stage case and that it can be called 'essentially negative' one day and 'first stage' the next by the same reader. This was not new. It has been emphasized repeatedly by Pendergrass, beginning in 1938."

A similar experience was reported in 1960 by Williams and Hugh-Jones ( ), who took 38 X-ray films of asbestosis patients, and mixed in at random films from nine patients with other chest diseases, and six normal chest films. The films were then shown to two panels of radiologists and chest specialists who were asked to interpret them. The authors wrote:

"There were several surprising findings in this study. One was the extent of the disagreement between the observers in the diagnosis of the films from the certified cases of asbestosis. These patients had all been seen and certified by the pneumoconiosis medical panels and had initially been selected by us as classical examples of the disease. Yet when the same films were viewed in the present trial the number thought to show changes consistent with the diagnosis of asbestosis varied from 40 to 80% approximately according to the observer...

"...In conclusion it is apparent, from the observers' findings in the group of films from other pulmonary diseases, that the radiological appearances in asbestosis are by no means specific, and may be confused with such widely differing diseases as chronic bronchitis and emphysema, sarcoidosis, and other pneumoconioses. Presumably if other interstitial fibroses such as scleroderma, lymphangitis carcinomatosa, xanthomatosis, or beryllium granuloma had been included in this group the confusion would have been even greater."

The situation has been summarized by Demy, 1962 ( ):

"...there is no specific radiologic (x-ray) diagnosis of pulmonary asbestosis, and the picture of expertness and infallibility with which lawyers like to introduce their particular expert medical witness is a legal fiction to impress the jury. Lawyers should be introduced to the Second Aphorism of Hippocrates, who, writing on ancient medicine, stated 'Whoever, having undertaken to speak or write on medicine, have first laid down...some hypothesis to their argument... are clearly mistaken in what they say.'"

Since the diagnosis of asbestosis is so often questionbale, a diagnosis of asbestosis accompanied by lung cancer must be even more questionable, since diagnosis of primary lung cancer is still an uncertain art.

Another feature of asbestosis which negates the lung cancer association is the non-progressive nature of the lesions of asbestosis. The malignant feature of cancer is its ability to spread, while asbestotic fibroses are limited to the area of the included asbestos particle.

Experiments conducted by Vorwald et al ( ) revealed that the fibroses produced in animals by exposure to asbestos dust do not spread or become worse. The authors said in 1951:

"Although in man asbestosis is a chronic disease with diffuse pulmonary fibrosis which requires years to develop, it is possible to reproduce in one or more species of animal characteristic tissue changes which are similar to the lesions of human asbestosis...

"...Long asbestos fibers are essential in the production of the peribronchiolar fibrosis; short fibers are incapable of producing this reaction...

"...The mode of action of the long asbestos fiber in the production of asbestosis is primarily mechanical rather than chemical in nature...

"...Established experimental asbestosis ceases to progress on discontinuance of dust exposure.

"The experimental investigation shows, in fact, that on discontinuance of exposure there was an appreciable clearing of the mature pulmonary lesions, due to contraction of the fibrous tissue."

Gross and Smith ( ) compared the non-progressive nature of asbestosis with progressive silicosis, suggesting that the explanation might be found in the contrast in shape between the long asbestos fiber and the round silicon particle. Their paper published in 1959, observed:

"In contrast to silicosis, asbestosis is not a progressive disease. Basically silicosis owes its characteristic progressiveness to the fact that silica particles deposited in one site, may be transported by tissue fluid (generally as edema fluid) to uninvolved portions of the lung to initiate new lesions. Asbestos fibers, in contrast, cannot pass the intricate barrier presented by the lattice work of tissue fibers as readily as particles of the same diameter which are substantially spherical. Also, since asbestos has a very considerable solubility, dissolution of the fibers offers a plausible explanation for the paucity of mineral found in asbestotic scar tissue. Both of these factors prevent the transport of significant amounts of asbestos fibers from their site of deposition to new locations and therefore explain the non-progressiveness of asbestosis."

The cancer-causing potential of a substance is usually tested through animal experimentation in which a clear dose-time relationship is demonstrated. Animals exposed to heavy doses of the carcinogen over long periods of time usually develop cancers. Those more lightly exposed -- either in time or dose -- may show no signs of malignancies.

Asbestos has not produced lung cancer in laboratory animals. Furthermore, there seems to be no clear or consistent dose-time relationship for asbestosis in humans.

Kleinfeld et al ( ) have remarked on this lack of correlation between exposure and pulmonary disease:

"The study group comprised 56 asbestos workers. They were selected on the basis of having had an exposure to asbestos dust, as asbestos insulators, for 14 years or more and no previous occupational dust exposure...

"...In general, the correlation between the degree of pulmonary function impairment and the clinical and roentgenographic findings was poor for the exposed group as a whole and no consistent correlation could be found between the duration of exposure and lung function impairment in this group. The lack of a good correlation between the degree of pulmonary function impairment and the clinical, roentgenographic, and environmental findings observed in the present study has also been reported by Bader et al."

The comment of Gross ( ) in 1963 on lack of evidence is appropriate.

"...In recent years, the association of pulmonary carcinoma with asbestosis has been the topic of many publications. It seems only proper to expect the same rigorous criteria for the establishment of etiologic relationship between pulmonary cancer and asbestos as have been demanded for the proof that pulmonary cancer is caused by excessive cigarette smoking."

III. What is an "Asbestos Body?"

Asbestosis is characterized by the presence in the lung of inhaled particles of asbestos. Although asbestosis is difficult to diagnose, in true cases of asbestosis, the inhaled particles (golden brown in color, symmetrical, club-shaped, up to 5 microns thick and 50 microns long) will form tiny tissue growths called "asbestos bodies."

Apart from the problem of what type of asbestos may comprise any given "asbestos body," the term has gradually been extended in use to apply to any similar lesions found in the area of the lung which form around a foreign body.

It is known that many cases of pneumoconiosis are of mixed origin. Nevertheless some clinicians and pthologists who have reported the presence of "asbestos bodies" have not employed chemical analysis. This loose identification has even been applied to particles found in the lungs of subjects with no known exposure to asbestos whatsoever.

Cauna et al ( ) in 1965 reported the difficulty of identifying asbestos, as opposed to other particles, in a study of autopsied human lungs:

> "The problem of absolute identification of asbestos in asbestos bodies is a difficult one, and to our knowledge no successful histochemical procedure has been devised. Chemical examination of lung tissues for acid insoluble residues yields a value for total silica that might represent asbestos or other silicates. No chemical determinations were done in these cases. An attempt was made to identify the element silicon in asbestos bodies (from a known case of asbestosis) by electron probe analysis. Although iron was readily identified, no silicon was encountered. The bodies seen in talcosis are identical to those in asbestosis and are thought to be the result of contamination with tremolite, another fibrous silicate. It is possible that the bodies seen in our material are associated with tremolite."

In a December 1965 symposium on asbestos, three experts discussed the same problem of identifying an "asbestos body." Vorwald ( ) stated:

> "In view of my experimental and clinical studies of pneumoconiosis with special reference to asbestos, and from the standpoint of the clinical pathologist, looking at tissue from post-mortem material of the lungs, it would appear that a real problem is: what is an asbestos fiber? It seems to me that we must perfect techniques where we can specifically identify an asbestos fiber as asbestos, distinct from other fibers present in the lungs that are not asbestos.
>
> "Let us also recognize that not all bodies that we see in the lungs, coated or uncoated, contain an asbestos fiber. There are many bodies, to wit, the graphite body, which is coated with ferratin and iron, which is segmented; the bodies seen in coal-worker's pneumoconiosis, the bodies seen in tremolite talc workers. I have seen the rouleux formation of degenerated red blood corpuscles which, under ordinary hematoxylin-eosin stains, often appear as segmented asbestos bodies.

And Holt ( ) added:

> "Again, assuming that everything that looks like an asbestos body is an asbestos body seems completely fallacious because in the literature you will find photographs of, for example, talc bodies. The center is not black. I suppose that if there is a talc hazard this must be one of the most serious ones because every woman when she powders her nose, inhales very large concentrations of talc and I should be extremely surprised if there were not talc bodies in the lungs of women in general."

The criticism of Schepers ( ) was directed at recent studies which have been given attention in the lay press:

"I am frankly astonished at the facility with which asbestos bodies have been recovered from lungs at Cape Town and at Miami, since it has always been difficult to demonstrate asbestos in lungs that have been long and thoroughly exposed to the dust and that might even have well-established pulmonary fibrosis. I believe I am quoting correctly the consensus of experience of pathologists in the U.S.A. that the findings of asbestos bodies is both rare and, in diseased lung tissue, always significant. The superabundance of cases in Cape Town and Miami therefore is a mystery. The suggestion that this may be the result of abrasion of automobile brakes does not correlate with the prevalence of automobiles or local driving habits. There are more automobile brakes abraded per linear mile in New York City than in either Cape Town or Miami. If this is the factor, I am sure Drs. Selikoff and Churg and other pathologists would have long since reported on this phenomenon. For the time being, and despite this interesting presentation by Dr. Thompson, I am obliged to continue to believe that the prevalence of asbestos bodies is a function of occupational exposure to asbestos dust. I have examined hundreds of lungs of persons who never have been employed in asbestos industries and none of these has shown asbestos bodies. Likewise I have examined the lungs of thousands of experimental control animals, who breathed the same air as do the human residents in normal cities. Again, asbestos bodies have never been detected in a single mouse, rat, hamster, guinea pig, rabbit, cat, dog or monkey, unless such an animal had previously been deliberately exposed to respirable asbestos dust. When I see my first unexposed subject with asbestos bodies, I shall of course revise my scepticism."

IV. What is an "Asbestos Worker?"

The opinion of Schepers is that the "prevalence of asbestos bodies is a function of the occupational exposure to asbestos dust." Obviously asbestos miners and workers in asbestos fabricating plants fit this category.

It might be assumed that an "Asbestos Worker," the name applied to a member of the International Association of Heat and Frost Insulators and Asbestos Workers, is also occupationally exposed to asbestos dust. This may be true in some cases, but it is not categorically true.

Members of the so-called "Asbestos Workers" Union work with a wide variety of materials including different types of asbestos. Certain insulating materials contain amosite fiber and a magnesium silicate base; others contain a mixture of amosite and chrysotile fiber; still other insulating materials contain a mixture of these two fibers with calcined diatomaceous earth which contains a fair percentage of cristobalite. In recent years fiber glass has competed with rock wool and rock cork. Since workers tend to specialize in the installation of certain materials, it is possible that many "asbestos workers" have never been in contact with any type of asbestos fiber whatsoever.

Most reports linking asbestos to lung cancer have been based on studies made among these "asbestos workers," who install various roofing and insulating materials. And in only a minority of lung cancer cases has there been reported concurrent findings of "asbestosis" or "asbestos bodies" in the lungs.

xxdxxx

The comment of Isselbacher et al, ( ) made in 1953, still applies today:

"Few reported cases of lung cancer related to industrial asbestos exposures provide data on the character and quantity of dust exposure. This is a serious deficit in exact study of etiologic correlation."

V. The Health of Asbestos Miners and Fabricating Plant Employees.

In the United States and Canada, reports suggesting an above-average incidence of lung cancer with asbestos have not come from studies conducted among those groups most heavily exposed. On the contrary, the exposure of workers in the insulating trades, where such reports have originated, may be considered comparitively moderate at most, and complicated by exposure to other dusts and other factors. This anamolous situation would suggest that slight, or moderate exposure to asbestos is more dangerous than heavy exposure -- a premise that is dubious on the face of it.

So far as is known, employees of asbestos fabricating plants in the United States, with a positive exposure to asbestos, and a positive tendency to develop asbestosis, are no more likely to develop lung cancer than the general population.

Lanza ( ) commented in 1952:

> "...as some of the mines and fabricating plants have been working extensively for over thirty years, it would seem that if cancer was a frequent complication of exposure to asbestos dust, this fact would have become evident long ago, especially as the medical supervision of asbestos workers is both thorough and comprehensive."

Although no definitive study of the health of American asbestos factory workers has been published as yet, such a study is now being carried out by the U. S. Public Health Service.

The health of Canadian asbestos miners, however, has been studied, and the results show that the lung cancer rate among these workers is not significantly higher than among the general population. The study was published in 1958 by Braun and Truan: ( )

"According to the findings in this study, the mortality rate from lung cancer does not appear to increase with length of exposure or with degree of exposure, a fact which presents strong evidence against the carcinogenicity of asbestos.

"Comparison of the experience among the asbestos miners with that of various segments of the unexposed, comparable population shows that the observed number of deaths among the miners is not significantly greater than the expected number. The rate for proved cases among the asbestos miners (25.3 per 100,000) compares well with the rate of 22.5 per 100,000 for the rest of the Province, and 20.8 per 100,000 for adult males throughout the Dominion of Canada...

"...On the basis of what are believed to be complete and reliable data, it seems fair to conclude that the asbestos miners in the Province of Quebec do not have a significantly higher death rate from lung cancer than do comparable segments of the general population.

"Furthermore, the death rate from lung cancer in the areas contiguous to the asbestos operations is comparable to that in areas widely scattered throughout the Province of Quebec and is lower than in some urbanized areas within the Province."

Add - quotes re epidemiological data -

VI. Chemical, Physical and Biological Tests With Asbestos

In an effort to identify known carcinogens, all types of asbestos have been subjected to exhaustive chemical analyses. Minute traces of some suspect substances have been found, but in such infinitesimal quantity that no significance is attributed to them by scientists studying the question.

In 1965 Goldblatt and Goldblatt ( ) commented on the fact that the statistical observations on which the asbestos-lung cancer theory is based are not supported by chemical or biological experimentation. The authors said:

> "'But at no stage in all these impressive researches was any clue obtained which might have offered any support to the possibility that asbestos could act as a carcinogen. There is no reliable criterion by which one can anticipate carcinogenicity and, as is well known, relatively minute changes in the structure of a chemical carcinogen are sufficient to diminish or eliminate carcinogenic action.
>
> "'If asbestos is indeed to be regarded as a carcinogen, the need is felt to demonstrate some property which can be regarded as something more than inertness.'"

At the 1965 Symposium Schepers ( ) reported the results of animal experimentation. His work shows that chrysotile asbestos is neither carcinogenic of itself in rats, nor does it promote the effect of a known carcinogen, beryllium, when that substance is administered with asbestos.

Said Schepers:

"In a study completed a few years ago at Saranac Lake, we proceeded as follows. We used laboratory bred albino rats, which, in previous experiments with beryllium sulfate, beryllium oxide and beryllium fluoride, had yielded abundant pulmonary carcinomata. (See Progress in Tumor Research, Karger, Basle, 1962). Two groups of rats were started respectively on exposures to chrysotile dust and beryllium sulfate dust. A third group served as an undosed control. After six months of exposure the beryllium group was split into two halves. One group continued in the beryllium environment. Another group was now exposed to chrysotile dust for six months. The original chrysotile group was dealt with in identical manner, i.e. one half was continued on chrysotile and the other half transferred to beryllium exposure. All exposures were terminated after a year. The prevalence of pulmonary neoplasia was statistically contrasted for each group. In essence the following was found:

Group 1. Undosed controls: No neoplasia.
Group 2. Chrysotile exposure only: No neoplasia.
Group 3. Beryllium exposure only: About 1/4 of the animals developed pulmonary neoplasia.
Group 4. Beryllium followed by chrysotile: About 1/5 developed pulmonary neoplasia.
Group 5. Chrysotile followed by beryllium. About 1/10 developed pulmonary neoplasia.

"One may infer the following from this experiment:

a. The animal species is normally not pulmonary carcinoma prone, but possesses a latent potential toward neoplasia when adequately challenged.

b. Beryllium sulfate is a potent carcinogen when inhaled for six or more months at a daily aerosol concentration of 12 gamma per cubic foot of air.

c. Chrysotile evinced no carcinogenic potential in this animal species.

d. Chrysotile did not act as a co-carcinogen with beryllium sulfate.

e. The lower incidence of neoplasmata in Groups 4 and 5 does not necessarily imply that chrysotile acts as an anti-carcinogen, but may perhaps be explained by the lesser exposure period to the beryllium and the shorter available induction time for pulmonary carcinogenesis."

Dr. Schepers had previously commented sceptically on the reports of "asbestos bodies" found in the lungs of persons with no history of exposure, and had discounted the theory that automobile brakes might release harmful quantities of asbestos dust into the air.

Dr. Kenneth W. Smith ( ) Medical Director, Johns-Manville Corporation, presented the results of tests which disprove this theory:

"In our Research Center we are continually testing our brake linings and those of other companies on various types of automobiles and trucks. When we first heard of the supposed association of the disintegration of the brake lining with the asbestos bodies in lung juice, we immediately started a program of pulling the wheels off these test automobiles and examining the brake lining dust. Various samples were examined after 5,000 miles of driving, 10,000, 15,000 miles, etc. There was ample dust for examination, but in not one of these samples of our own manufacture, or samples of brake lining made by other manufacturers in this country, could we identify a chrysotile fiber. As far as we are concerned it is impossible for any of our type brake lining, manufactured in this country, to disintegrate and liberate chrysotile asbestos fibers which can be inhaled to form asbestos bodies. This is quite understandable because chrysotile asbestos fiber disintegrates when exposed to the tremendous heat created by the friction of the brake drum on the brake lining material."

VII. Summary

The theory that lung cancer is caused by exposure to asbestos has not been proven and should be regarded with scepticism at the present time. The following facts tend to cast doubt on the theory:

1. Asbestos is not one substance but many substances, differing in chemical and physical composition.

2. A recognized disease of the lungs, asbestosis, is non-progressive and has no clear dose-time relationship.

3. It is not certain whether asbestosis can be induced in laboratory animals.

4. Asbestosis is difficult to diagnose and is frequently confused with other diseases caused by dust inhalation.

5. Exposure to asbestos has not produced lung cancer in laboratory animals.

6. It is not certain that so-called "asbestos bodies" sometimes found in lung cancer victims are actually asbestos; many other substances cause similar lesions.

7. In the United States and Canada, the occupational groups most heavily exposed to asbestos dust, such as miners and fabricating plant workers, have no significantly different rate of lung cancer than is found in the general population.

8. The so-called "asbestos workers" in the insulating and building trades work with a variety of different materials, which may or may not include asbestos. In any case they are exposed to various types of dust, and for the most part their exposure to asbestos dust is certainly moderate compared to the exposure of miners or asbestos fabricating plant employees.

9. Only a very small part of the population is occupationally exposed to asbestos. There can be no possible correlation between this exposure and the reported increase in lung cancer cases in recent years.

December 9, 1968

REPORT OF MEETING
December 3, 1968
Public Relations Sub-Committee
Environmental Health Task Force

Present:

F. J. Solon, Jr. - Chairman
W. P. Raines
M. M. Swetonic
Bert Goss - Hill & Knowlton
Carl Thompson - Hill & Knowlton

Newsletter For Mount Sinai Project

It was agreed that the money for producing the newsletter ought to come out of the basic research program budget. The mechanics of writing, approving, printing and distributing the newsletter were discussed. Hill and Knowlton agreed to prepare an outline of these mechanics --- including costs --- that could be used as a sort of check list for a meeting with Jobe and Dr. Selikoff to iron out any difficulties. Matt Swetonic was assigned the task of checking with Hibbert Printing Company to see whether final printing and mailing stages of the program could be handled through the J-M Advertising & Public Relations production department's art costs.

J-M

Business Week Article

Various avenues of approach to Business Week for the placement of a favorable story on the Mt. Sinai program were discussed. The decision was made to approach the Labor Editor, rather than go higher in the organization. Carl Thompson of H & K outlined the specific approach and pointed out the pros and cons in the move.

It was decided that any attempt to place a favorable article in Contractor News should be delayed until after the Business Week approach is concluded since the latter is the big circulation book and we should go at it first.

San Francisco Department of Public Works

Discussed the letter received by Dr. Selikoff from the San Francisco Department of Public Works requesting information on the potential dangers of asbestos to the general public. Needs further investigation. Solon will talk with Burt Jobe.

Jack Solon recommended strongly, it was agreed to by all members of the committee, that J-M set up a team of J-M people, knowledgable in the field of asbestos and health, who would be ready at a moment's notice to fly to trouble spots like San Francisco and "move in" on the situations.

O'Toole and The Washington Post

Tom O'Toole of the Washington Post contacted Dr. Selikoff about a series of articles examining a wide range of environmental health problems, including possible health dangers associated with asbestos, beryllium, coal dust, radiation. It is believed the series is designed as a boost for the Health & Safety Bill in the next Congress. O'Toole interviewed Selikoff on December 2, an article appeared in the Washington Post December 4 - a damaging article basically concerned with dockyard workers.

Ralph Nader

Nader has occasionally mentioned asbestos in his listing of potentially harmful dusts. Should he at some time in the future decide to crusade against asbestos, it was decided that more could be gained from sitting down and talking with the man than by taking the General Motors route. H & K sources indicate that Nader is a reasonable man and can be approached on that basis. It was agreed, however, that he should not be contacted unless he takes on Johns-Manville first.

Manville Situation (Addendum to November 6 minutes)

Dr. Merrill recently remembered that Dr. Demy of Somerset Hospital did indeed approach him in 1966 for work history records and that he did not follow through by providing them. This is, of course, in direct contrast to the statement that we made to the Messenger-

contacts between J-M medical representatives and the outside scientific community, especially in the Manville area.