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POSITION PAPER

ON THE

HEALTH ASPECTS OF ASBESTOS

ADVERTISING AND PUBLIC RELATIONS DEPARTMENT
JOHNS-MANVILLE CORPORATION

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I. INTRODUCTION

The march of modern industry brought with it many previously unsuspected health hazards to those exposed 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 more than 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 health 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.

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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 part of this reported increase results from expanded knowledge of cancer and tremendously improved diagnostic procedures, especially for lung cancer. Furthermore, with the greatly improved health of the average individual, people are living longer today. The average life span in 1920 was 54 years; today it has increased to about 70 years. With this increased longevity, more people now are developing many more chronic, disabling diseases than they did years ago. For example, fewer people today die at an early age from tuberculosis or rheumatic fever. They survive to develop chronic heart disease, arthritis and cancer -- diseases typical of older age groups.

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

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most of the attention and blame, although general air pollution is also considered a factor. 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 proved innocent -- not innocent until proved guilty.

For example, 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.

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 in their time, but the conclusions were as erroneous as the theory of the sun spinning around the earth! These theories could not be confirmed and were eventually disproved by experimentation and experience.

Suggestions associating lung cancer with asbestos have neither been confirmed nor disproved by experimentation, nor by many observations of asbestos miners and fabricating 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.

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II. 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 95 percent 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

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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.

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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.

Wagner experimented with all three types of asbestos on various laboratory animals, and found that crocidolite produced the most severe asbestosis 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."

The point made by all these experts is: data that may tend to associate one type of asbestos with some disease cannot be assumed to apply to other types as well.

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III. WHAT IS ASBESTOSIS?

Asbestosis, first described more than 40 years ago, is one of a class of diseases called the pneumoconioses -- 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 the diagnosis of asbestosis itself still is difficult. Despite undeniable evidence of the disease and recent extensive reporting in the medical literature, the great majority of physicians cannot diagnose asbestosis. In relation to other disease entities, asbestosis occurs so rarely that it is not on the teaching curriculum in medical colleges and the average doctor has no exposure to a patient with this condition either in his training or in his practice.

The situation is made more complicated by the fact that pneumoconioses are sometimes caused by a mixture of various dusts, such as 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?

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Much more common than asbestosis is silicosis, caused by the inhalation of the dusts of crystalline quartz silica (sand dust). Silicosis has been proved 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 most medical authorities observe that asbestosis is static. The disease gets worse only through continued exposure to asbestos dust. Finally, there is a difference of opinion among scientists as to whether the lesion 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."

Since the diagnosis of asbestosis is so often questionable, 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 casts doubt on the lung cancer association is that most specialists observe that the lesions of asbestosis are non-progressive. 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:

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"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.

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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."

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IV. 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," this 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 pathologists who have reported the presence of "asbestos bodies" have not employed the necessary identifying procedures -- chemical analysis, gas chromatography, electron microscopy. This loose identification has been applied even 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."

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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. Thomson, I am obliged to continue to believe that the prevalence of asbestos bodies is a

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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."

Because of the presence of other materials in the air today, Gross suggested in 1966 that the term "asbestos body" should no longer be used:

"The pressing question today is the following one: Are there materials other than asbestos, that are of filamentous character and manufactured today, that are part of ambient urban air, and that will stimulate alveolar phagocytes to produce asbestos bodies? These materials could probably be classed mostly as plastics and would, most likely, have one important feature in common, namely a biologic 'inertness.'...

"Because of the present uncertainty regarding the specificity of the asbestos body, the term 'ferritin' body was suggested. This term was criticized because the composition of ferritin has not been adequately characterized. Perhaps the use of the term 'ferruginous' body will serve the purpose until it can be shown that these structures are specific for asbestos fibers."

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V. 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.

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."

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VI. THE HEALTH OF ASBESTOS MINERS AND FABRICATING PLANT EMPLOYEES

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.

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"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."

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VII. 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 1956 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:

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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."

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VIII. SUMMARY

The theory that lung cancer is caused by exposure to asbestos has not been proved 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. Asbestosis is difficult to diagnose and is frequently confused with other diseases caused by dust inhalation.

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

5. In the United States and Canada, occupational groups 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.

6. 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 potential exposure to asbestos dust is certainly moderate compared to that of miners or asbestos fabricating plant employees.

7. 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.

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