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	December 6, 1977	See Below	
Subject		From	Location and mail code
"Medical Aspects of Occupational Exposure to Asbestos" by Dr. Hilton C. Lewinsohn		J. G. Heil	VF PLYC 2B

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The attached article should be reviewed. It points out on page 8 that an employee with diagnosed case of asbestosis can still look forward to working for ten to fifteen years and may live five to ten years after finishing work. The disease process may not appreciably shorten life in present day circumstances.

On Page 16 the effects of fiber exposure are discussed. "If this cumulative exposure were spread over 30 years, this would mean that concentrations less than 1.5 to 2 fibers/cc would cause no reduction in FVC (Pulmonary function test); and that concentrations greater than 7 fibers/cc would usually lead to a reduction in FVC as well as X-ray changes in 10% or more of workers."

Dr. Lewinsohn is a recognized expert in the field of asbestos related disease because of his research/studies made in England with asbestos textile workers.

MEDICAL ASPECTS OF OCCUPATIONAL EXPOSURE TO ASBESTOS

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(Talk to Members of Friction Materials Standards Institute, Inc. Annual Meeting on June 22, 1977)

A. Introduction

It always pays to define "asbestos", even when it seems that it is unnecessary to do so because of the sophistication of the audience being addressed. "Asbestos", is a generic term for a variety of hydrated silicate minerals which have one common attribute, namely, the ability to be separated into relatively soft, silky fibers. Although the name is ordinarily associated with those varieties which have technologic importance, it is applicable to all minerals which fit the above description. The term "asbestiform minerals" is perhaps most descriptive.

There are two main classes depending upon their crystal structure, namely, serpentine and amphiboles. The sole member of the serpentine class is chrysotile asbestos which comprises nearly 95% of world production. There are five asbestiform varieties of amphibole, namely, crocidolite, amosite, anthophyllite, tremolite and actinolite.

The uses of asbestos are many and the physico-chemical properties of the different varieties determine their commercial importance. The medical complications resulting from exposure to asbestos are also related to the physical and chemical properties of this fibrous mineral species.

B. The "Asbestos Diseases" and Other Conditions Associated with Asbestos Exposure

1. Benign, non-disabling conditions

(a) Asbestos corns, warts or callosities

Workers handling raw asbestos fiber as it arrives from the mines, often get splinters in their hands. These splinters may cause an inflammatory reaction which eventually subsides leaving a hard, thickened, raised area of skin with a central core of fibrous tissue. There is no information available to indicate whether this mode of asbestos penetration can lead to subsequent malignant change in distant organs and skin cancer has not been recorded as a complication of asbestos warts. The skin continually renews itself and the corns eventually merely mark the spot where fibers once were.

(b) Asbestos Bodies

In Belfast about one in five of elderly men coming to autopsy had a sufficient number of asbestos bodies in his lungs for these to be detected by examining one or two microscopic sections.[1]

Thomson[2], in 1964, reported on investigations which began in Cape Town, South Africa, in 1960. These investigations were intended to determine the extent to which the ordinary urban dweller is exposed by occupation or environment to the inhalation of asbestos. Over 25% of the lungs of 500 consecutive

autopies on subjects of 15 years and over showed asbestos bodies by the method used. Thomson started a similar study in Miami, Florida, in 1961 while exchange professor there. The overall positive findings were remarkably similar to those in Cape Town. In 85% of the positive cases the bodies were scanty, were not associated with pulmonary changes and were regarded as the result of contamination of the urban atmosphere. In 6% of all the males examined the bodies were numerous and were presumably of occupational origin.

I have classified asbestos bodies under benign non-disabling conditions because they are not in themselves indicative of disease. Asbestos-bodies are encapsulated fibers probably inert and indicative of asbestos exposure. The implications of Thomson's findings will be discussed later in this paper. According to Selikoff,[3] asbestos bodies do not appear randomly distributed among the general population of New York but are, to an important extent, occupationally related.

Asbestos fibers are not all coated and converted into bodies. Uncoated fibers may persist for many years after exposure to asbestos ceases.[4]

It would appear that asbestos bodies contain inactivated fibers and that the uncoated fibers are the ones associated with disease causation. Other mineral

fibers can form similar bodies and some people prefer the term ferruginous bodies.[5] Glass fiber, silicon carbide, filamentous aluminum silicate and fibrous talc could be confused with coated asbestos fibers.

Pooley[6] has found that asbestos bodies in mesothelioma cases from 4 different countries were associated with amphibole exposure and that asbestos bodies detected in these lungs were all derived from amphibole fibers. The importance of this will become apparent later.

(c) Pleural Plaques, Pleural Fibrosis and Pleural Calcification

The lungs are invested by a thin layer of connective tissue known as the pleura. This membrane covers the outer surface of each lung and is then reflected on itself in the midline to cover the inner surface of the chest wall. A potential space exists between the two layers.

Inhalation of asbestos dust results in very characteristic changes in the pleura which can be regarded as an index to exposure. Pleural changes may also occur after infections such as pneumonia and pleurisy, tuberculosis, injury to the chest wall and exposure to commercial talcs.

Pleural plaques are well-defined areas of pleural thickening which are found on the domes of the diaphragm, along the rib margins and in the gutter which runs along the margin of the vertebral column - the paravertebral gutter. These may be recognized on X-rays but often

are not seen and are primarily discovered at autopsy.

Pleural plaques or pleural thickening may become calcified. Pleural calcification has been described by Kiviluoto in Finland.[7] These studies drew attention to the widespread presence of calcification of the pleura in communities exposed to asbestos dust. Kiviluoto reported on the finding of pleural calcification among people in a rural community living in the vicinity of two open cast anthophyllite asbestos mines. Burilkov and Michailova recently suggested that the fibrous mineral sepiolite, which is present in concentrations of up to 5% of soil in certain parts of Bulgaria with high rates of pleural calcification, might be responsible for non-occupational pleural calcifications in various countries.[8] Gibbs[9] has studied the epidemiology of pleural calcification and found that pleural calcification among Quebec miners was not due to exposure to chrysotile itself, but to dusts produced during mining operations. The distribution of cases within the Quebec industry showed that exposure to dusts responsible for pleural calcification occurred mainly in Thetford Mines and were therefore associated with local geological formations. Talc and mica in the Thetford area were considered the most likely agents responsible for pleural calcification in Quebec chrysotile workers.

Are these pleural changes truly benign or should

they be considered as precursors of malignant changes? This question cannot yet be answered adequately, but in Gibbs' study, persons with definite and suspected pleural calcification showed no overall excess mortality when compared with all persons employed in the Quebec asbestos mining and milling industry born between 1891 and 1920. Elmes[10] found pleural plaques in 25% of cases of mesothelioma studied by him in Belfast. He stated that when the exposure was mixed, i.e. to more than one type of fiber, the presence of pleural plaques in a population seemed to indicate a level of exposure capable of producing mesotheliomas. Edge[11] found that shipyard workers with pleural plaques who had mixed exposure to asbestos (without evidence of pulmonary fibrosis) had a 2.5 times increased risk of developing lung cancer when compared with the general population.

Leathart[12] described the lung function results in 181 asbestos workers and concluded that asbestosis was usually, but not always, associated with lung function defects while pleural calcification alone had no effect on lung function. Becklake[13] showed that, while pleural calcification alone appeared to have no adverse effect on lung function, diffuse pleural thickening was accompanied by a reduction in certain parameters of pulmonary function.

Pleural changes have been known to be associated with asbestosis for a long time, but changing circumstances

in industry have reduced dust concentrations in factories and intermittent exposure elsewhere. The typical asbestosis is becoming less common and less severe than it used to be and has become replaced by a very slowly progressive disease in the lungs, while the well recognized pleural changes are still occuring to the same extent. These pleural changes have become "more obvious" because the underlying lung disease is minimal. There may well be some instances where pleural changes occur in the absence of underlying lung disease.

2. Asbestos Diseases

(a) Asbestosis

Asbestosis is a fibrosis or scarring of the lungs resulting from the inhalation of respirable asbestos fibers in high concentrations, usually for a prolonged period of time. Some authors include the associated thickening of the visceral pleura in this definition.

The dependent (lower) parts of the lung are affected first and the process progresses as the years go by, even after exposure ceases. The diagnosis of asbestosis depends on;

(1) history of exposure
(2) finding of fine end-inspiratory crackles at the lung bases on auscultation with a stethoscope
(3) clubbing of the fingers (not an essential diagnostic sign)
(4) X-ray changes

(5) lung function changes indicative of restriction of ventilation or impairment of gas exchange.

The ILO U/C International Classification of Radiographs of Pneumoconiosis (1971) has been developed for epidemiologic purposes and is descriptive, not diagnostic. A diagnosis of asbestosis can only be made by examining the worker, the X-ray, the lung function tests and the occupational history. Other respiratory diseases such as chronic bronchitis, emphysema, asthma and certain chronic lung diseases can be mistaken for asbestosis.

The severity and progression of asbestosis appears to depend on the amount of asbestos retained in the lung. From the time symptoms are first noted most workers can continue to work for 10 to 15 years and may live another 5 to 10 years after finishing work, usually having had to reduce the work-load in gradual stages because of increasing shortness of breath. Asbestosis is unusual under the age of 50. Other conditions leading to the necessity for light work and early retirement may precede asbestosis in this age group. As mentioned earlier, improving industrial conditions over the past 20 years have resulted in a less severe form of asbestosis than was seen in the 1930's, 1940's and 1950's. This disease process may not appreciably shorten life in present day circumstances.

(b) Asbestos Cancer

According to Gilson,[14] it was about fifty years after the commercial exploitation of asbestos began that lung cancer was first thought to be caused by the dust (1935), about another ten years before this was generally thought probable (1945), and a further ten before it was finally established in the asbestos textile industry (Doll 1955).

Lung cancer complicates 50-60% of asbestosis cases resulting from exposure to conditions more than 30 to 40 years previously. In some sections of the asbestos industry the effect of improvements in dust control on the excess mortality of lung cancer appears to be a dramatic reduction of this complication in parallel with the reduction of asbestosis incidence. The most striking contrast, according to Gilson, is between the low risk in chrysotile miners and millers in Quebec born between 1891 and 1920, and the high risk in the insulation workers where exposures have been to a mixture of chrysotile and amosite in the U.S.A., or to these two and crocidolite in the U.K.

Asbestos fibers have a large surface area and readily allow adsorption of other materials. Lung cancers have been blamed on substances adsorbed on to the fiber such as trace metals, cigarette smoke and hydrocarbons from other sources. Cigarette smoke is important and the interaction of cigarettes and

asbestos exposure has been well documented by Selikoff.[15] Non-smoking asbestos workers rarely get lung cancer. The types of lung cancer in smoking asbestos workers do not differ in their effects from primary lung cancers in other people. The majority of affected individuals die within a year of diagnosis. Although lung cancer is usually associated with underlying asbestosis, some authorities believe that this is not always the case. The risk of premature death from malignant chest disease seems to be confined to those with high dust exposure. Asbestosis usually no longer kills because improved dust conditions have resulted in a "milder" form of disease. Less mortality from asbestosis occuring after longer periods of exposure has resulted in survival of workers through the long latent period of lung cancer.

(c) Mesothelioma

The association between exposure to asbestos and diffuse malignant mesothelioma first attracted wide attention through the publication of a series of South African cases by Wagner, Sleggs and Marchand (1960).[16] Subsequent comparisons between cases of this pathologically controversial condition and control patients have confirmed a statistically significant association with asbestos. Wagner et al also showed that neighborhood or community exposure could be associated with this malignant tumor.

Exposure in some cases may be of brief duration and there is a long lapsed period (latent interval) between first exposure and diagnosis or death. This lapsed period may be from 20 to 40 years or more. Disease diagnosed today probably had its causation in working conditions which prevailed between 20-40 years ago, or longer.

The tumor affects the pleura, grows slowly, doesn't spread readily to other parts of the body and it kills by slowly compressing the lung and vital structures associated with it. Peritoneal tumor is less common and is similar in its effects. The tumor can occur from about the age of 35 onwards but more than 50% do not develop until after age 60. Cigarette smoking does not seem to be a causative factor.

According to a review of the epidemiology of mesothelioma from estimates of incidence presented by Alison and Corbett McDonald at the XVIII International Congress on Occupational Health in Brighton, in 1975, the incidence of mesothelial tumors is extremely high in three situations: among insulators, among those who work in or live in cities with shipyards and among those who work in or live in certain cities with large asbestos plants.

The most definite association with mesothelioma is following exposure to crocidolite fibers from the Cape Province and Transvaal in South Africa and from

Western Australia. McDonald and McDonald concluded that there are indications in most types of exposure of a gradient in the mesothelioma inducing potential of asbestos fiber with crocidolite being the most hazardous, amosite less hazardous and chrysotile least hazardous. McDonald[17] has recently demonstrated that cases from the chrysotile mines in Quebec, Canada, were due to crocidolite. Crocidolite was used for the Canadian army respirators and this fiber was processed at the site of the major Canadian chrysotile mine. Crocidolite was similarly used in gas masks in Britain. The Canadian gas-mask workers experience exactly parallels that recently reported by Dr. J. S. P. Jones[18] and colleagues of some 1,600 persons employed, 1939-45, on the same process using Australian crocidolite in Nottingham, England. McDonald calculates that the risk of mesothelioma after crocidolite exposure in the circumstances described would appear to be at least 50 times greater than that associated with chrysotile production.

There is no evidence that the general public is at risk of developing mesotheliomas from the fibers measurable in the ambient air. Occupational histories are deficient in those studies which have attempted to correlate environmental measurements with mesothelioma incidence. The correlations between increasing

utilization of asbestos during and since World War II are more reliable, indicating the likelihood of occupational risk rather than public health risk.

C. The Issues at Stake

There appear to be three major issues at stake at the present time, viz:

(1) Can asbestos products be manufactured safely?

If so,

(i) is there any risk to users of asbestos-containing products and,

(ii) do low levels of exposure constitute a public health risk?

(2) Can substitutes be found for asbestos?

(3) Who is going to provide the answers and make the decisions?

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1. Manufacturing of Asbestos Product

The uses of asbestos are myriad. Many of the uses of asbestos are probably unnecessary and continue because traditions die hard. It cannot be replaced by suitable substitutes as yet in many areas.

There is satisfactory evidence in the world literature to indicate that asbestosis is a dose-response related disease. There is, furthermore, adequate evidence that a dose-response also exists for the carcinogenic properties of asbestos. The fibrogenic (ability to produce lung fibrosis)

effect and the carcinogenic (cancer producing) effect of asbestos appear to be similar for all varieties in commercial use. The scientific opinion with regard to the proposed gradation of effect attributed to crocidolite, amosite, chrysotile, tremolite and anthophyllite in the production of diffuse malignant mesothelioma of the pleura or peritoneum, is divided. Although most observers believe that crocidolite, particularly that from Australia and the N.W. Cape Province of South Africa, is the most dangerous fiber, that amosite holds an intermediary position and that chrysotile presents the least hazard, some authorities do not accept this thesis. Having weighed the evidence presented by both schools of thought, I am of the opinion that crocidolite has greater mesothelioma producing potential than amosite or chrysotile and that its use should be strictly curtailed. In the United Kingdom the use of crocidolite has virtually disappeared and no raw fiber has been imported or handled in production since 1970.

In reviewing the literature it is obvious that advances in the control of asbestos manufacture did not proceed at the same pace in the industrialized world. The Asbestos Industry Regulations, 1931, which came into effect in the United Kingdom in 1933, preceded the rest of the world by approximately 40 years (or more). Although it is difficult, if not impossible, to make comparisons of working conditions in different countries for the same type of industry, there are indications that the health experiences are different

in such countries due to the time lag between introduction of comparable control measures. One group of workers in a South African factory described by Collins[19] in 1967, worked in totally uncontrolled conditions. This paper is not suitable for statistical analysis, but the description given by Collins of conditions, in what he calls "an asbestos refinery", is horrifying. He states "The dust within the building resembled a dense fog, and could be seen escaping into the atmosphere through the entrance. Jets of dust escaped like steam from faults in the conduction systems between mills and cyclones, and dust lay thick on every beam and projecting surface."

The insulation workers of the United States are perhaps the best studied and most widely quoted groups in present medical literature due to the prolific publication of results by the Environmental Sciences Department at Mount Sinai Hospital in New York. Chrysotile asbestos miners and millers in Quebec have been equally well studied by McDonald.[20] Nicholson[21] demonstrates quite clearly the problems which exist in attempting to define dust exposures for insulation workers where adequate dust measurements are lacking, while McDonald has been able to utilize information provided by the asbestos mining industry to derive a meaningful "Dust Index" for chrysotile miners in Quebec.

The best documented study of asbestos workers (textiles) with regard to medical and dust-measurement data is that of the British Occupational Hygiene Society's Sub-Committee on

Asbestos Standards which was published in 1968 and recommended a cumulative standard of 100 fiber/cc years for chrysotile asbestos.[22] In 1970 NIOSH reported that records of dust concentrations between 1930 and 1967 in one asbestos textile factory, and between 1948 and 1968 in another, were assembled in the Pennsylvania Department of Health. In a report presented at the Western Industrial Health Conference by Howard Ayer, it was disclosed that, using lung function as the most sensitive indicator of asbestos health effect, it appears that cumulative exposures below 50 fiber/cc years cause no reduction in FVC, and exposures greater than 200 fiber/cc years are usually associated with reduction in FVC. If this cumulative exposure were spread over 30 years, this would mean that concentrations less than 1.5 to 2 fibers/cc would cause no reduction in FVC; and that concentrations greater than 7 fibers/cc would usually lead to a reduction in FVC as well as X-ray changes in 10% or more of workers.

The present standard in the U.S.A., and most of the world, is 2 fibers/cc and is based on the BOHS Standard for chrysotile. In the United Kingdom and certain other countries crocidolite is dealt with more stringently because of its association with mesothelioma. In October 1975 OSHA proposed a tightening of the standard to 0.5 fibers/cc and in December 1976 NIOSH recommended that it be 0.1 fiber/cc.

The argument regarding the adequacy of the standard is dependent upon the "no safe threshold for a carcinogen" theory. There is qualitative evidence that the 1931 Asbestos Industry

Regulations in the U.K. had the effect of markedly reducing the incidence of asbestosis and similarly reducing the excess deaths from lung cancer in the same factory studied by the BOHS.[23] Some residual effect is still being seen in this factory because dust levels were still relatively high in many areas until very recently. Bearing in mind that the BOHS Standard was not published until 1968, that new Asbestos Regulations were not made in the United Kingdom until 1969 and that the 2 fibers/cc standard was not officially applied there until 1970, it is obvious that no conclusions can as yet be drawn with regard to the level of risk still attached to working in conditions in total compliance with this standard.

Having attempted to review the main issues regarding asbestos manufacture, the answer to the question posed is obviously, that we do not know for sure, but the evidence is pointing towards the conclusion that, when adequately controlled the risk of asbestosis and lung cancer can be reduced to virtually nil. Dr. Roach[24] has summed up the situation as follows:

> "A problem arises when it is appreciated that there is no exposure which can be said to be absolutely free of risk. There is no single threshold exposure held in common by everyone. There is, consequently, this gradually increasing risk in relation to exposure. The application of dust control to meet a TLV, an MAC, MAK value, or other similar hygiene standard will limit and control the risk but is unlikely to reduce it to zero. It has to be remembered that asbestos is very widely used and brings real benefits to the community at large. A standard could be made so stringent that the cost of dust control is prohibitive, that the production and use of asbestos ceases to be economic, production and use is discontinued and the associated benefits are lost. The benefits gained by reducing the risk of

asbestosis through reducing air contaminant exposure have to be weighed against the possible loss of direct and indirect benefits to the community from the use of the material."

He goes on later to remark:

"The air quality attained in industry in different countries does differ and, no doubt, will continue to differ. A wealthy country can afford to spend more money on air-contaminant control. Also, a country very conscious of the slightest risks to which its workers may be exposed through their occupation may be expected to have different standards from one which is not, where other health risks may be so much the greater.

The benefits to the community from the use of inexpensive asbestos products have in some measure to be weighed in the balance against the benefits to the health of the workers that would accrue by reducing asbestos dust exposure."

The further comments of Dr. Roach are of interest and I quote:

"To derive hygiene standards for an air contaminant which provide a known degree of protection against a health hazard, it is necessary to have a body of data showing the amount of air contaminant to which people are exposed and the corresponding effects or lack of them in the people. It is also necessary to have a grasp of the consequences to industry and users of limiting and controlling emissions of the contaminant. Our present information is very imprecise, particularly in terms of the practical consequences of specific hygiene standards. In developing recommendations for a hygiene standard, the British Occupational Hygiene Society Sub-committee found that knowledge of the relationship between exposure and risk was not the greatest area of uncertainty. A much more difficult and contentious problem was to decide on what, in fact, was an acceptable level of dust control.

More information is needed, for example, on the expense of dust control. Where this is done by ventilation it is important to know what is the minimum amount and what kind of local exhaust ventilation and dilution ventilation is necessary to achieve a given degree of air cleanliness in a work place, since costs tend to climb as the cube of the air flow.

Research is needed to determine the balance between local and general ventilation which produces a specified degree of control at minimum cost. By setting down the capital

> cost, installation cost, running and maintenance costs, it becomes possible to grasp more firmly the consequences of adopting particular hygiene standards. This kind of information is needed throughout the field of asbestos dust control so as to be able to weigh up the costs of achieving high air cleanliness.
>
> This does not reduce the choice of an air quality standard to a mathematical equation, nor does it avoid the need to exercise wise judgment in the choice of standard. However, the judgment can become a little less arbitrary than at present."

The second question posed under the above heading is in regard to the risk to users of asbestos containing products. Asbestos is used throughout industry and until recently, outside of the manufacturing industry, users took few, if any precautions. Because asbestosis is dose-related no immediate health hazard was apparent from this cause in users of asbestos products. The exception to this rule is in the insulation industry where the upsurge of cases became marked in the late 1950's and early 1960's, probably as a result of the increase in asbestos usage under poor conditions during World War II. The process of spraying asbestos onto girders of high-rise buildings, spraying asbestos on the interior of buildings for heat and sound insulation and the extensive use of this process in naval ship-building programs was probably one of the most hazardous uses ever. Mechanical operations such as the sawing, drilling or abrading of asbestos products will create dust and power tools create more dust than hand tools. The quantity of dust produced will also depend on the amount of asbestos in the product and the nature of other components. Most demolition processes, where asbestos-based

products are being removed, are likely to give off considerable amounts of dust.

It has been shown that brief exposure to crocidolite asbestos can result in development of mesothelioma. Users of asbestos products are usually exposed intermittently and accumulate a smaller dose of dust in the same period of time as workers continuously exposed in manufacture of asbestos products. Mesothelioma may occur in the absence of asbestosis. Mesothelioma has occurred in plumbers, carpenters, electricians, etc., who were exposed in the vicinity of insulation workers or others using asbestos. It has also been reported in persons who have lived in close proximity to crocidolite mines and mills and factories or building sites at which crocidolite asbestos was used. From all the epidemiological surveys there are between 5-30% of cases of mesothelioma in which no evidence of exposure to asbestos can be found. It has been shown that nearly everyone who lives in an urban community has some amphibole asbestos fibers in their lungs.

There has been a great deal of controversy as to whether asbestos brake-linings constitute a health risk in terms of exposure of brake service mechanics. The epidemiological surveys conducted on this population have been carried out very recently by the Selikoff group, and no other epidemiological evidence is available as yet. There appears to be some evidence of radiologic changes in brake-service mechanics in the group studied by Selikoff[25] but no evidence of frank disease.

At the Annual American Industrial Hygiene Conference last

month, NIOSH presented data showing that the use of proper work practices would reduce the asbestos exposure of brake service mechanics to an extremely low level. (Approximately 1/20th of the presently permissible OSHA level.) NIOSH will shortly issue a technical bulletin outlining acceptable work practices. The recommended work practices will be almost identical to instructions provided by Raybestos-Manhattan to all friction material customers for the past 2-1/2 years.

There is obviously some risk attached to the use of certain asbestos containing products, but many give off no dust and others, once incorporated in machinery, etc., never again see the light of day. Great care should always be taken in the use of asbestos and materials containing it and the dust levels should always be below the minimum required. The main non-industrial use of asbestos is in do-it-yourself building materials. There are also some domestic products which contain asbestos, such as some electrical appliances. There is negligible risk of fibers being dispersed from domestic products in normal use provided they are in good condition.

To prevent the misuse of asbestos products warning labels should always be affixed and work practices advised.

In answer to the third question it should suffice to say that there is no published epidemiological evidence to support the hypothesis of a possible danger to the general public. The biological effects of asbestos have always manifested themselves in individuals or groups of individuals exposed to dust concentrations many orders of magnitude greater than

levels measurable in the general environment. One major area of concern is the ingestion of fibers from water carried in asbestos cement pipes or from the filtration of wines, beers, spirits, beverages, etc., through chrysotile asbestos filters. This concern arises mainly because of the demonstration of an excess incidence of gastro-intestinal cancer in certain heavily exposed asbestos insulation workers and heavily exposed textile workers. It is interesting that the textile workers studied by the BOHS do not exhibit a similar excess mortality from GI cancer. If the use of chrysotile asbestos filters is discontinued this would be a retrograde step. The ingestion of chrysotile asbestos and other types of fibers in experimental animals has failed to produce mesotheliomas. From human evidence, only people with a severe exposure to asbestos dust have contracted peritoneal mesotheliomas and these tumors have not been found in any of the asbestos mining areas except those mining crocidolite, in spite of the very heavy dust exposure especially in those exposed to chrysotile.

2. Asbestos Substitutes

The major health problem associated with asbestos exposure is mesothelioma. As has been stated earlier in this paper, asbestosis can be controlled and lung cancer appears amenable to similar controls, but because the latent period between first exposure and diagnosis of mesothelioma is long, and this malignant tumor's association with asbestos exposure is a recent discovery, sufficient time has not yet elapsed to determine the level of dust capable of producing this response. Some evidence

has emerged that mesothelioma is dose related, but more time is needed to determine this dose. The gradation of effect previously discussed becomes important in the context of prevention. The reasons for this gradation of effects are of importance and may determine the feasibility of using other fibrous materials as substitutes for asbestos.

The theory developed by Timbrell[26] can explain the gradations in biologic potential of the various types of asbestos. He suggests that long fibers are preferentially deposited in the respiratory bronchioles at bifurcations and that this may explain why fibrosis tends to be associated first with respiratory bronchioles and with long fibers. He also suggests that the characteristic "rectilinear" shape of amphibole fibers compared to the "curly" morphology of chrysotile fibers, allows the amphiboles to penetrate to deeper parts of the lung more efficiently than chrysotile fibers. A reasonable theory has thus been proposed to explain the reason for the development of mesotheliomas. It is based upon the ability of certain types of fiber to penetrate deeper into the lung than others and to reach the pleural cavity by direct penetration.

Substitutes for asbestos are being sought although for most purposes none have been found as yet. Other fibrous minerals are being tried among them glass fibers and mineral wools. Animal experiments indicate that if such fibers were capable of reaching the pleura, i.e. had the same physical characteristics as amphibole asbestos fibers, they could produce mesotheliomas. The available evidence depends upon the implantation into the

pleural cavity by open surgical techniques, of the various materials tested to date. Both in Europe and in the United States epidemiological studies have failed thus far to demonstrate any carcinogenic hazard to workers in the man-made mineral fiber industry. This is a field of very active research and should hopefully provide answers in the near future which would prevent a repetition of the asbestos tragedy.

3. Who is going to decide?

(a) Scientific Opinion

"As long as there is any airborne asbestos dust in the work environment, there may be some small risk to health. Nevertheless exposure up to certain limits can be tolerated for a lifetime without incurring undue risks." (Roach)[24]

"With this discouraging picture of inadequate knowledge of risk, ill-defined exposure information, and limited enforcement of existing levels before us, one may well ask of what value a TLV is for asbestos, or for any carcinogen. Should such materials be banned from use in all forms? Asbestos is extensively used in industry for insulation, for inclusion in plastics and other products, for reinforcing high-stress materials. At present, nearly 1 million tons are used annually in the United States. For some uses, as in brake linings, it is difficult to find a replacement. A societal decision to ban the use of asbestos would create serious, if not insurmountable, difficulties. Moreover, we would still face control problems posed by the large quantities of asbestos in current use. Our only recourse at this time is to limit human exposures to asbestos and other similarly recognized carcinogens to the lowest possible levels, with existing technology.

In the case of asbestos, a TLV can serve a purpose: Recognizing that it is, in fact, a Risk Limitation Value, however ill-defined that risk might be, it can serve to mandate implementation of available technology and rule out the small fraction of work processes in which available technology fails to keep up with the major portion of the industry. More, however, is required than the specification of a number. The specification of work practices and engineering controls offers an essential supplement to a numerical TLV if the latter is used at all. Application

of many economically and technically feasible procedures can reduce exposure to levels much below existing numerical values. These should be mandated. Moreover, such procedures can be specified with joint government-union-management cooperation and can be monitored much more readily than can dust concentrations. TLVs and work-practice standards should be reviewed frequently with a view to achieving continued reduction in worker exposures. The specification of a proposed TLV can serve as a stimulus for the development of new engineering-control methods or to rule out marginal processes that cannot be controlled. As new engineering developments evolve, the lowering of a minimal standard can be undertaken along with the specification of additional protective work procedures.

To a limited extent, this has taken place in the asbestos industry." (Nicholson).[21]

"Threshold and dose response are only two of the components in decision-making in environmental control and regulation. In addition to scientific data, with all of its present limitations, public health responsibility must incorporate "prudence" as a factor in judgment. This invokes such issues as "cost/benefit ratios" and "risk", as recently reviewed by Falk. The cost/benefit ratio, at best an elusive attainment, must clearly delineate the "cost to whom" and "benefit to whom." The quantitative contribution to this equation must virtually be entirely derived from data on man. The concept of "risk" - the summation of threshold and dose response - when applied to population, is indispensably but not exclusively based on human as well as on experimental data. Laboratory contribution to "risk" encompasses the entirely tenable concept of threshold as well as dose response when addressed to the subject of the conference: the hazards of environmental agents to man." (Kotin).[27]

(b) Trade Unions

"In the past, risk assessment has been largely the domain of academic, industrial, and government scientists who have usually waited as long as possible to share their information with workers. This discussion will focus on the need for risk assessment to be a process continually going on at two levels, Federal and local.

The need for Federal involvement in the standard-setting process is obvious, with tasks including carcinogenic risk assessment itself, standard-setting, enforcement, and when necessary, further modifications if workers are not being adequately protected. The need for worker involvement has only more recently been recognized. During this Federal standard-setting process the involvement of workers or their

representatives is critical to the design of an adequate standard, monitoring, and medical surveillance. Once set, workers must have an active and informed role at the local level in assuring that the standard is enforced." (Wolfe).[28]

"To reiterate, the most difficult decisions to be made by government will not be scientific in nature. Social and moral decisions will be made that can channel and shape the development of our control technology, which itself will become a major determinant of our future welfare. In this process, labor's contribution is unique.

Alone among American publics, the worker is most exposed to environmental insult both in the community and in the shop, while being most vulnerable to the economic consequences of control. He and his institutions are of necessity, therefore, in a position of forced objectivity. Thus his is a critical voice to be heeded.

The participation of organized labor is not automatic. A positive effort must be made, an effort I call "positive public advocacy." This is an essential government responsibility, involving the public in decision-making processes such as the assessment of environmental risk." (Samuels).[29]

(c) Government and its Agencies

"Because the Federal government has provided for a National Cancer Plan under the leadership of the Director of the National Cancer Institute, it must be this Federal agency that provides overall leadership for an effective integrated national program for prevention and control of occupational cancer. A splintering of responsibility for research and training can work only to the detriment of the worker. The NCI cannot retreat from its responsibility to provide regulatory agencies with information concerning risk of exposure to specific chemical, physical, and parasitic agents demonstrated to induce tumors." (Lassiter-OSHA).[30]

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Ed Hatton

Attached are three articles related to the health risks versus exposure levels of asbestos. The A/C cement currently complies with the OSHA limits of 2 f/cc

Please call if you have any questions

Tim Dail

7/26/79

More power to you when you ship
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Remarks of Philip E. Enterline, Ph.D.
Professor of Biostatistics, Graduate School of Public Health
University of Pittsburgh

at

Asbestos Information Association
Third Annual Industry-Government Conference

September 8-9, 1976

Dr. Enterline is Professor of Biostatistics, Graduate School of Public Health, University of Pittsburgh. He received his undergraduate education at Westminster College, and his postgraduate degrees at the American University.

Dr. Enterline is a recognized expert in the fields of biostatistics and industrial epidemiology. He has published a number of distinguished papers. Prior to joining the Univeristy of Pittsburgh faculty, Dr. Enterline served in a variety of responsible positions in the U.S. Public Health Service. He has a broad background of experience on health advisory committees and has done extensive consulting work for government, academia and private industry.

Listening to Alex Cross reminded me of a grand tour I did of England back in 1962 when I first got in the "asbestos business." I'll never forget a young statistician who was assigned to orient a group of us about the problem in England and what was going on, and throughout his entire presentation he talked about the "asbestosis" factories in England. The man next to me said, "Oh, how I wish we could get him to stop saying that!"

I was asked today to talk about a project I've been doing for the Asbestos Information Association, and I'll spend a little bit of time talking about it, not much time, because I haven't gotten into it very deeply. This is something Guy Gabrielson asked me if I would undertake and is being done with the help of one of my graduate students. The question we're trying to answer is, "What did we know about the association between asbestos and cancer at various points in time as we go back through history? What did the experts think about the assosiation in 1935, 1940, 1945, etc.? At any point in time what was the expert opinion? What did writers think about what they were seeing?"

This is an interesting kind of project because it requires doing something that to my knowledge has never been done. It means reading not only the summaries of the various articles, but the introductions and the discussions, and if it's a paper given at a meeting, the discussions of the paper from the floor and the questions and answers, if they're published. Also, all the editorials that have been written about asbestos and cancer, textbooks, letters to the editor, and questions and answers in the leading medical journals. Then try to form some kind of opinion about what was believed at these various points in time.

The game plan is that we'll have one or two graduate students do the actual screening of the literature (photographing the pages that are relevant) and then have a biostatistician and an epidemiologist independently review this material and make some decisions about what points in time were certain things known. I want to emphasize that we're not trying to find out when should we have known; we're trying to find out when did we know. When did the writers of the various periods realize what they were dealing with? That's something a little different than an ordinary literature review.

To give you a sampling of what we find when we do this, I'll show you a couple of things we've abstracted which illustrate a number of points about this particular project.

The first slide is a writing from the bulletin of the American Society for the Control of Cancer and was written in 1943. I'm going to talk only about asbestos and lung cancer in order to narrow this thing down somewhat.

Slide #1

1943
Bull. Am. Soc. Cont. of Cancer

> "Asbestosis cancer of the lung is the most recent newcomer among the occupational cancers of this organ. First described in 1935, there are now 18 cases of this industrial cancer on record observed among asbestos workers in England, Germany, and the United States. The latter contributed five cases. Inasmuch as the asbestos industry is most extensively developed in this country asbestosis cancer of the lung has for us a special hygienic and sociologic significance.

Now this is interesting because it's based upon reports on an association of asbestosis and bronchogenic carcinoma at autopsy, and it is written by Bill Heuper whom some of you surely know. He wrote a great deal about occupational cancer. Now let me show you a writing in 1961. This slide is from the Journal of the American Medical Association. That's a very prestigious journal, and this writing is even more prestigious because this happens

to come from a section of that journal that answers questions that are asked by the medical profession. If you're a practicing physician and want the latest information on a particular topic, you write to the Journal. They refer your letter to someone they consider a leading expert and that person will then reply in the columns of the journal to your question.

Slide #2

> "There is no epidemiological evidence that silicosis, resulting from undue exposure to free silica, produces cancer of the lung. Of the several silicates, asbestos is held in suspicion, especially in Great Britain. In the American literature there is no evidence that there is a relationship between asbestosis and lung cancer. The reason for the difference between the English and American experience is not apparent. It may be due to a difference in the type of asbestos fiber or to the total dosage."

As you might expect, in 1961 there was considerable correspondence which followed this, however, the writer stuck to his opinion and defended it on the grounds that there was no epidemiologic evidence and points out in subsequent commentary in the Journal that without epidemiologic evidence no picture of a disease would really be complete. This is interesting because what is happening in 1961 is applying a different set of rules to decide if there is an association as compared to 1943. In other words, the rules were changing at the same time knowledge was developing.

This type of commentary is not indexed and calls for reading lots of journals, many of them weeklies. My student has read all issues of the Lancet (which is probably the leading medical journal in the world), the British Medical Journal, and the Journal of the American Medical Association back to 1935 and has picked out a lot of material that I can't present here today. So far we've gotten up through 1961. After that it really gets tough because the

volume of literature exploded after that date, particularly after the 1964 conference in New York. I'm simply giving you some idea of the type of project that Guy has launched us upon, what this project involves, and where we are at present. Incidentally, we've located some very humerous stuff. For example, in 1949 Ovaltine was being advertised to prevent pneumonociosis and asbestosis. So we've come a long way since 1949.

Let me now proceed to something quite different -- and this is stimulated by an article I saw in this morning's paper. It's the matter of how much disease will be produced from exposure to asbestos. We've been working on a model to predict how much disease is produced with continuous exposure at any given fiber level over any given period of time. For example, we could think of workers who start at age 20 and who work for 50 years at 2 fibers per cc. How much disease would we expect to occur? This is really the important question, I think. What will happen to people if we take certain actions on their behalf at this point in time?

First, to review rapidly some elements of carcinogenesis needed to develop a model, we know that if you're exposed to a carcinogin, you won't get cancer tomorrow, but that there will be a peak incidence at some future date and thereafter it tapers off. This distribution seems to follow a mathematical function called a log normal.

Another important notion for modeling the effects of asbestos exposure is that for low dose rates the time it takes for cancer to appear will be very long. On the other hand, if you have a very high dose rate, cancer will appear rather quickly. I'm thinking now about groups of people, say 100,000 people exposed at different levels. In other words, there is a relationship between

the intensity of exposure and the latent period. Thus, a population would have to live a long time to experience a full response to exposures at very low levels.

Now if we add to this the assumption that this dose-response relationship is linear, we can develop the family of response curves. The linear dose-response idea means simply that every increment of dose causes a like increment in response -- a straight-line relationship. That has some acceptance as far as cancer is concerned. It's one that you may not like, however, because it means that any little bit of asbestos will produce some kind of response given enough people and enough time. If you talk about a threshhold limit, the idea is that a little bit won't produce anything -- that you have to have a certain amount - 2 fibers, 5 fibers, etc., before you produce any disease. I don't think it really works that way. I think that any little bit does produce something.

Here is the output of the model, and is kind of the end product.

Slide #3

Cumulative Relative Risks for Respiratory Cancer
at Seven Levels of Continuous Exposure

Exposure Level (f/cc)	20 Year	40 Year	60 Year
1/2	1.000	1.000	1.003
1	1.000	1.003	1.021
2	1.000	1.026	1.098
4	1.005	1.154	1.361
8	1.050	1.684	2.075
16	1.390	3.400	3.702
32	3.291	7.861	7.032

Unfortunately, in the interest of time, I've had to skip some intermediate steps and assumptions but could provide these to anyone interested. This says that where the cumulative relative risk is carried to three decimal places, no effect of asbestos on the death rate from lung cancer can be detected until 60 years of exposure at 1/2 fiber, 40 years at 2 fibers, and 20 years at 4 fibers. One reason for this is the assumed long latent period for low doses. After 60 years, for example, increased lung cancer would appear; but, of course, for persons entering the labor force in their 20's, other causes of death are pretty important at this point.

Now that we know what the risk is at various exposure levels, we could calculate absolute number of cancers if we knew how many people were exposed. I went back to something the Asbestos Information Association did some time ago and made a slide of it. Here are quotes from Irving Selikoff about how many people are exposed and how many will die from asbestos-related diseases.

Slide #4

Dr. I. J. Selikoff . . . . Predictions

	No. of Workers	Description of Cohort	Total Deaths from Asbestosis Lung Cancer & Mesothelioma
'ticle in Wall Street 'ournal -- 6/8/72 udies by Dr. Irving ikoff . . . Lead Him Predict..."	250,000	All workers currently Employed in the Asbestos Industry	85,000*
'icle in the New York imes...6/13/72 'e Days Later) our experiences.... 's a Guide...."	500,000	All workers currently and previously employed in the Asbestos Industry	170,000
stimony at Toxic bstances Hearing - r studies indicate.."	1,000,000	All workers currently, previously, and who will be employed in the Asbestos Industry in this country	340,000

*Actual Quoted Prediction was 95,000, But this included deaths from GI Cancer.

CTD010971

This is essentially the material that was quoted in this morning's paper and apparently assumes that all persons exposed have been and will be exposed like men in his studies -- mostly asbestos insulators. We hope that isn't so. What we'd really like to know is how many men would be affected by asbestos if exposures were at 4 fibers, 2 fibers, etc., since that is what the government is deciding.

Let's just take one number. Let's take the first number, 250,000 -- that's all workers currently employed in the asbestos industry. One question we might ask is how many of these people would you expect to die of lung cancer if there was no asbestos around. Let's say they never had been and never would be exposed to asbestos. Of course, some people would still die of lung cancer because lung cancer has lots of causes -- cigarettes, air pollution, etc. Also, you have to remember that things can happen to you other than dying of lung cancer. You could get hit by a truck or have a heart attack (some people are going to have a heart attack when they read some of this stuff).

I estimate that if there was no asbestos exposure, in a group of 250,000 men there would be 12,500 dying of lung cancer. Now, what if these men were exposed continuously to half a fiber for 60 years? From Slide #3, we can estimate that 12,537 would die of lung cancer (1.003 x 12,500). If these 250,000 men were all exposed continuously to two fibers, 12,762 would die; if exposed to four fibers, 17,012 would die, and if exposed to eight fibers, 25,937 would die of lung cancer. This is after 60 years. Looked at another way, 250,000 men exposed for 60 years to half a fiber will produce 37 lung cancer deaths, etc.

The problem then comes down to these excess deaths and how many there are at these different exposure levels. To simply deny that there are any excess deaths I don't think is ever going to get any place, but I think to recognize how many we're talking about may have some meaning. Given improvements in industrial hygiene, I'm sure we're not talking about excesses of the magnitude Dr. Selikoff predicts. Of the 500,000 he predicts will enter the industry or will be exposed during the remainder of the century, a 2 fiber standard at 40 years of continuous exposure would increase deaths from 25,000 to 25,650.

Let me show you my last slide which is a check on how well my model actually predicts.

Slide #5

Relative Risk Predicted by Continuous Exposure Model at 15 f/cc Compared with Relative Risk Reported in Study of U.S. Insulation Workers

Years from Onset of Exposure	Predicted Relative Risk	Observed Relative Risk (Adjusted)
10	1.0	0.0
10-14	1.6	2.2
15-19	2.8	3.4
20-24	4.3	3.1
25-29	5.4	4.5
30-34	5.7	6.4
35-39	5.4	5.4
40-44	4.8	4.9
45-49	4.1	3.6
50+	3.4	3.8

Does my model predict Selikoff's finding? These data are from Selikoff's study of 17,000 insulation workers. Selikoff has undoubtedly produced some of the most magnificent epidemiological data I've seen, and I think this 17,000 insulation workers study is truly a great study. You see there is a very good correspondence. The model predicts pretty much what Selikoff observed. Notice, for example, that Selikoff observed less and less cancer as time went on. That's what the model predicted too. Notice that his peak occurred for men who were exposed 30-34 years ago, and I get exactly the same peak.

To summarize, it is possible based upon the epidemiological data now available to predict what will happen if various actions are taken at this time in regard to asbestos exposure and if various standards are set. This should be helpful in deciding where you want to set this thing. I think it would be sticking our heads in the sand to say that nobody will ever be affected at 2 fibers or at any other finite level.

ASBESTOS