PLAINTIFF'S EXHIBIT
UC-1299

ASBESTOS: "THE HISTORY, THE PRESENT, THE FUTURE"

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Introduction:

One cannot take liberties in assembling historical facts except in the choice of reference sources. My task is to place the historical facts of the asbestos saga into their true perspective in such a manner that the current policies regarding its use and regulation assume a logical consistency and that future trends may be predicted. It is not easy to unravel the intertwined and tangled web of medical, industrial hygiene, experimental, epidemiologic and political factors involved.

"Asbestos" is a generic term used to describe the fibrous varieties of a number of mineral silicates. Two main groups of rock-forming minerals produce fibrous forms, namely the serpentines and the amphiboles. The fiber types with which people are most likely to come into contact as a result of their use in industry are:

(a) serpentines - chrysotile

(b) amphiboles - crocidolite
amosite
authophyllite
actirolite
tremolite

Amosite is an acronym for Asbestos Mines of South Africa and is mineralogically known as cummingtonite - grunnerite asbestos.

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"Asbestos" is also used to refer to the industrial products containing one of the many varieties of the raw material. It is important to refer to a product by name since the asbestos content and fiber variety in it will differ from item to item. Asbestos cement products for building purposes use mainly chrysotile and take the greatest tonnage of asbestos. The potential exposure of workers manufacturing asbestos-containing products may be very different from persons handling and using them. Henry Walton has reviewed this topic very adequately.

The activities in which there is a potential risk of occupational exposure to asbestos dust are of almost infinite variety, and monitoring methods must be adapted to suit. The broad categories into which such activities may be sub-divided are:

(a) Primary production: mining, milling, transportation

(b) Manufacture of asbestos-based products:
- asbestos cement
- friction materials
- textiles

(c) Product usage
- insulating
- fitting friction materials

(d) Disposal of asbestos containing wastes

(e) Non-asbestos industry; asbestos as incidental contaminant:
- talc contaminated by tremolite
- taconite ore

(f) Para-occupational exposure and neighborhood exposure:
- asbestos on work clothes brought into home

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THE HISTORY

The first report in the English medical literature of a death resulting from pulmonary fibrosis due to the inhalation of asbestos dust was reported by Dr. Montague Murray in 1900. A second case was reported in England in 1924 and 1926 by Cooke. He described the unusual and characteristic "asbestosis bodies." In 1927 Oliver reported two cases, to be followed by McDonald and Seiler. In 1928 Simson reported two cases in South Africa. Other cases were then reported by Wood and Page, Haddow and Wood, and Gloyne. In the United Kingdom the comprehensive report of Merewether and Price appeared in 1930 and resulted in promulgation of the Asbestos Industry Regulations, 1931. In Britain, the statutory medical examination of asbestos workers by Silicosis Medical Boards, on an initial and periodic basis, was introduced under the provisions of the Silicosis and Asbestosis (Medical Arrangements) Scheme, 1931 (SR & O, 1931, No. 341). In addition, asbestosis became a compensable disease under the provisions of the "Asbestos Industry (Asbestosis) Scheme, 1931.

The first report of asbestosis cases in the United States was made by Pancoast, et. al. in 1917. They reported 17 cases of fibrosis in a Pennsylvania plant. Further cases were reported by Mills (1930), Donnelly (1933), and Lynch and Smith (1931). North Carolina industries figure prominently in the U.S. epidemiologic history with reports by Shull (1936) and Dressen, et.al. (1938) being added to that of Donnelly. These investigators reported cases of respiratory impairment and various degrees of "asbestosis" among the groups of workers investigated. Dressen's study on behalf of the U.S. Public Health Service formed the basis for the U.S. threshold limit value

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of 5 million particles per cubic foot of air (MPPCF) which remained in effect until 1972 when an updated standard was promulgated by the Occupational Safety and Health Administration.

According to Gilson, 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 by epidemiologic evidence that asbestos workers in tne textile industry in Britain had an excess death rate from this disease. Buchanan (1964) found that approximately 60% of cases of certified asbestosis cases in Britain died of lung cancer. Unfortunately, no smoking histories were obtained, an important ommission. In the United States, the first detailed epidemiolgic investigation of asbestos and cancer was reported by Selikoff, et. al. (1964). They studied the mortality of 632 asbestos insulation workers exposed to asbestos dust for 20 years or longer, and found a significant excess of cancer of the lung. In 1968, Selikoff and co-workers demonstrated that asbestos workers who smoked were at far greater risk of lung cancer than those who did not. Selikoff also noticed a slignt excess incidence of gastro-intestinal cancers in his population of insulation workers. This was also reported in a population of insulation workers in Belfast (Elmes) and from a group of asbestos workers in London (Newhouse). The original cohorts studied by Merewether and others (Knox, et. al.) in Britain, exposed mainly to chrysotile, did not show this excess of GI cancers.

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In 1960, Wagner, et. al. published their findings which associated diffuse malignant mesothelioma of the placera with exposure to asbestos. The population which they studied lived in the North-west Cape Province of South Africa where crocidolite was mined. According to Dement, "the first mesothelioma in an asbestos worker was observed by Leicher in 1954." However, Wagner, et. al.'s paper clearly demonstrated the association and since the 1960's this rare tumor has been recognized and diagnosed more frequently. For most reported series, the proof of asbestos exposure is lacking in from 10% - 60% of cases. In the crocidolite mining areas, in shipyards where crocidolite and amosite were used in substantial amounts and in certain selected occupations, such as gas-mask manufacture in Britain in World War II, this tumor appears to occur more commonly than in mining areas or manufacturing operations involving the use of chrysotile asbestos.

Perhaps the most worrisome aspect of the reports by Wagner, et. al. (1960) and Newhouse and Thomson (1965) is the association between mesothelioma and domestic contact with asbestos workers by their families, and the possibility of "neighborhood" exposure as the result of living in the near proximity of mines, mills or factories. Workers whose jobs do not directly involve asbestos may be casually or individually exposed if they are unprotected and enter the asbestos-work environment. Although some investigators have characterized these exposures, that is domestic, neighborhood and indirect occupational exposure, as minimal, there is good reason to believe that they were in fact fairly heavily exposed over prolonged periods of time, although in terms of occupational exposures in the same era (20-40 years ago), they could be regarded as relatively "minimal." Not all studies of populations

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living in the vicinity of asbestos plants have been able to identify a health hazard and in the U.S.A. Hammond was unable to demonstrate an excess of cancer or mesothelioma deaths in a community living near a dusty plant in New Jersey when he compared it to a similar community without any asbestos plants in its midst.

Before attempting to assess the present situation with regard to asbestos and health, it is necessary to define and briefly discuss two items which are freqently referred to in the medical literature. Histologic examination of the lungs of asbestosis cases revealed the presence of unusual beaded, rod-like particles which were originally called asbestosis bodies because it was thought that they only occurred in the presence of the disease. It was later demonstrated that they could be found in the sputum and the lung tissue of persons who had been exposed to asbestos but not necessarily suffering from asbestosis, and so they were then called asbestos bodies. These bodies may also be found in the lungs of the general population, because asbestos is ubiquitous in our environment. These bodies are iron-containing, and since some investigators believe any durable fiber which enters the lung may become the core of such a body, the term "ferruginous bodies" is preferred. There is considerable evidence that most ferruginous bodies are in fact asbestos bodies and the latter term is more commonly used. Asbestos bodies, therefore, are an index to exposure but not diagnostic of disease. It has been demonstrated that their frequency in lung tissues increases with exposure so that asbestos workers' lungs contain numerous asbestos bodies, workers in industry (blue collar) have less than asbestos workers but more than white collar workers, and so forth.

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Thickening of the pleura or the formation of pleural plaques has been observed in asbestos exposed populations. Pleural plaques have been associated with non-occupational as well as occupational exposure to asbestos. When plaques are bilateral, they are commonly recognized as a useful indicator of asbestos exposure. They have been reported in rural populations in a number of countries, including Czechoslovakia, Bulgaria, Finland, Greece and Turkey. In Finland they have been attributed to anthophylite exposure, affective persons living near asbestos mines, but not associated with asbestosis, excess cancer deaths or mesothelioma in such "environmentally" exposed populations. Pleural plaques used to be regared merely as an index to past asbestos exposure and without disabling health effects. However, it has become clear from recent studies, such as those conducted over many years on workers in the British Naval Dockyards, that persons with these lesions may subsequently be more prone to developing pulmonary fibrosis, i.e., asbestosis, or may have an increased risk of developing mesothelioma.

THE PRESENT

WHAT ARE THE PATHOLOGICAL EFFECTS OF EXPOSURE IN MAN?

Exposure to asbestos at work or elsewhere may result in five conditions:

1. The presence of asbestos in tissues without disease - e.g., asbestos bodies in the general population.

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2. The presence of asbestos in the tissues causing benign changes - e.g., skin warts, pleural plaques.

3. The presence of asbestos in the tissues and the development of malignant mesothelioma of the pleura or peritoneum.

4. Asbestos in the lungs with tissue damage and the development of lung cancer.

5. Asbestos present with potentially fatal damage to the lungs (pulmonary fibrosis or asbestosis), but no cancer.

There are two other conditions which have been associated with exposure to asbestos. These two conditions have been listed separately, because at the present time, there is insufficient published information regarding the presence or absence of asbestos in human tissue in these cases and the association has been established purely on epidemiological grounds.

The two other conditions which have been detected in greater numbers among asbestos workers than would be expected from a similar sample of the general population are:

a. Cancer of the gastro-intestinal system involving esophagus, stomach and colon and rectum.

b. Cancer of the larynx.

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WHAT IS THE EVIDENCE FOR STATING THAT ASBESTOS MAY BE PRESENT IN TISSUE WITHOUT DISEASE?

Examination of material from random autopsy series in several cities has revealed the presence of asbestos in lung tissue. The frequency of this finding depends upon the diligence of the search. When digested lung tissue is examined, prevalence approaches 100%. These findings can occur in the absence of any asbestos associated diseases.

WHAT ARE THE BENIGN CHANGES IN TISSUES FOUND IN THE PRESENCE OF ASBESTOS AND WHAT ARE THEIR SIGNIFICANCE IN TERMS OF PROGNOSIS?

Warts on the fingers and hand, and a discrete reaction involving the parietal pleura, usually in more than one place and referred to as pleural plaques, are often found in people who have been occupationally exposed to asbestos. Pleural plaques are usually a radiographic diagnosis in an otherwise healthy person. Pleural plaques may calcify. Pleural plaques have also been described in people exposed by living in the vicinity of certain mines or tilling soil with a high asbestos fiber content.

The above conditions are not in themselves disabling, although the pleural plaques may indicate a level of exposure sufficient to progress to more serious disease. The effect of pleural plaques on pulmonary function, although detectable in population studies, is modest and is mainly seen as small reductions in lung volumes. By contrast, X-ray changes may be very striking, particularly in the presence of calcification.

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Although the presence of pleural plaques alone does not appear to cause symptoms of disability, there is some evidence that they affect prognosis. They have been associated by some authorities with increased incidence of lung cancer and malignant mesothelioma has been reported as developing in the cells at the edge of the plaque.

WHAT ARE THE CURRENT VIEWS REGARDING MALIGNANT MESOTHELIOMA?

1. Epidemiologic evidence indicates a gradation of effect related to fiber type. Crocidolite, particularly fiber from the North West Cape Province of South Africa and from Western Austrialia, is considered to be the type of fiber most frequently associated with mesothelioma. Cnrysotile is considered to be least likely to cause it and amosite has been allocated an intermediate status. Although anthophyllite has been associated with asbestosis, pleural plaques and lung cancer, no cases of mesothelioma have been attributed to it.

2. Cigarette smoking does not seem to be a causative factor.

3. Exposure 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 had its causation in working conditions between 20 to 40 years ago or longer.

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4. The tumor affects the pleura, grows slowly, doesn't spread readily and kills by slowly compressing, first the lung on one side, and then the vital structures in the center of the chest or the lung on the other side. Peritoneal tumor is less common and is similar in its effects.

5. The tumor can occur from about the age of 35 onwards, but more than 50% do not develop until over the age of 60.

6. Domestic or neighborhood exposure has resulted in the development of this disease.

7. Mesothelioma is not uniquely associated with asbestos exposure and in most reported series a small proportion (15%-30%) cannot be related to asbestos.

WHAT ARE THE FEATURES OF ASBESTOS-ASSOCIATED MALIGNANT DISEASE OF THE LUNGS?

1. The risk of premature death from malignant chest disease seems to be confined to those with high dust exposure, though sometimes of brief duration.

2. Asbestosis is no longer an inevitably fatal conditon because improved dust conditions have resulted in a "milder" form of disease, or in fact a sub-clinical entity which is not always recognized. Less mortality from asbestosis occurring after longer periods of exposure to lower concentrations of dust than in past years has resulted in survival of workers through the long latent period of lung cancer.[13, 14]

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3. The interaction of cigarettes and asbestos exposure as risk factors is of great importance. Non-smoking asbestos workers rarely get lung cancer.[14, 15]

4. The primary lung cancers in smoking asbestos workers do not differ in their effects from primary lung cancers in other people, and the results of treatment do not differ either.[5]

5. Although lung cancer is usually associated with asbestosis, some authorities believe that this is not necessarily so.

DOES ASBESTOS HAVE OTHER CARCINOGENIC PROPERTIES?

1. Cancer of the gastro-intestinal tract involivng oesophagus, stomach, colon and rectum, has been reported in excess in asbestos insulation workers and other asbestos workers.

2. An association has been found in some reported studies between an excess incidence of cancer of the larynx and asbestos exposure. The effect of alcohol and smoking is a confounding variable in these studies.

3. At present there is insufficient published information regarding the presence or absence of asbestos in human tissue in these conditions and the association has been established purely on epidemiologic grounds. Evidence presently accumulating is tending to weaken the view that exposure

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to asbestos causes alimentary tract cancer. Large excesses continue in the studies where they were originally reported, but in other new studies no such excesses have been found.

4. It has recently been sugested that there may be an association between non-Hodgkin's lymphoma of the alimentary tract and exposure to asbestos. The debate on this matter continues.

WHAT IS ASBESTOSIS AND HOW DOES IT AFFECT THE EXPOSED INDIVIDUAL?

1. Asbestosis is a fibrosis or scarring of the lungs and includes the associated thickening of the visceral pleura, but not that of the parietal pleural.

2. The lower (dependent) parts of the lungs are affected first progressing as the years go by even after exposure ceases.

3. A diagnosis of asbestosis can only be made by examining the worker, all the available x-ray films, the pulmonary function tests made over a period of time and the complete occupational history.

4. Other respiratory diseases such as chronic bronchitis, emphysema, asthma and certain chronic lung diseases can be mistaken for asbestosis.

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5. From the time symptoms are first noted, most workers can continue with light work for 10 to 15 years and may live another 5 to 10 years after finishing such work.

6. Asbestosis is unusual under the age of 50. Other conditions leading to the necessity for light work and retirement may precede it in this age group.

7. Improving industrial conditions over the past 20 years have resulted in a type of asbestosis less severe than in the 1930's, 1940's and 1950's. At the present time life expectancy may not be appreciably shortened by this disease.

C. THE FUTURE

There appear to be three major issues at stake affecting the future of asbestos, viz:

(1) Can asbestos products be manufactued 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 Products

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 of its uses.

There is satisfactory evidence in the world literature to indicate that the development of asbestosis is dose-related. There is, furthermore, good 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 but more and more credence is being given to it. 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, a few authorities still do not accept this thesis. Having weighed the evidence presented by both schools of thought, a recent British Governmental Study is of the opinion that crocidolite and amosite have greater mesothelioma producing potential than chrysotile and that their use should be strictly curtailed. In the United Kingdom the import of amosite and crocidolite fiber has virtually ceased.

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In reviewing the literature it is obvious that advances in the control of asbestos manufacture did not proceed at the same place 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 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 studies 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. Nicholson 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

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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 an Asbestos Standard for Chrysotile, which was published in 1968 and recommended a cumulative standard of 100 fiber/cc years for chrysotile asbestos. 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 appeared that cumulative exposures below 50 fiber/cc years caused no reduction in FVC, and exposures greater than 200 fiber/cc years were 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. is 2 fibers/cc which came into effect in 1976 and is based on the BOHS Standard for chrysotile. In the United Kingdom a revised guidance note published by HSE formally lays down the new control limits for asbestos in the workplace which HSC agreed <u>should be adopted with effect from 1 January 1983.</u>

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The control limits are:

For dust consisting of or containing any crocidolite (blue asbestos).	0.2 fibre/ml when measured as a time-weighted average over any 4-hour period.
For dust consisting of or containing any amosite (brown asbestos) but not crocidolite.	0.5 fibre/ml when measured as a time-weighted average over any 4-hour period.
For dust consisting of or containing other types of asbestos (eg chrysotile - white asbestos) but not crocidolite or amosite.	1 fibre/ml when measured as a time-weighted average over any 4-hour period.

It is stressed in the guidance note that these control limits do not represent safe levels which once attained make further improvements in dust control unnecesssary - there remains the statutory duty to reduce exposure to lower levels if reasonably practicable.

The procedures for sampling are set out in more detail than in earlier editions of the guidance note. Persons developing an air sampling strategy, it says, need to be aware of all asbestos processes at the workplace and should have adequate information, instruction and training for the task. A new section in the note outlines acceptable techniques for sampling and analysis of bulk materials for use with the Approved Code of Practice 'Work with asbestos insulation and asbestos coating'. HSC are considering further measures for the control of exposure to asbestos dust. In addition the methods of measurement are under review.

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

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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. The BOHS Standard was published in 1968, and new Asbestos Regulations were made in the United Kingdom in 1969 and the 2 fibers/cc standard was officially applied there in 1970.

Many uncontrollable variables make it difficult to calculate whether this standard is adequate. Sampling methods are far from standardized, the use of static sampling results in the BOHS study cannot be adequately related to personal sampling results in surveys, insufficient time has elapsed since the adoption of the present standard, manufacturing methods have constantly varied over the years and a wide range of statistically derived dose-response curves makes it questionable whether it is rational to employ a single control limit throughout industry. For large sections of industry, such as asbestos cement and tiles (which are the largest users of asbestos fibers), and for miscellaneous uses, and for persons who utilize asbestos products, no dose-response data have been collected.

The British report previously referred to concludes as follows:

> 105 In the light of our terms of reference (paras 1 and 2) we conclude as follows:
>
> (a) we have not found any material which was available to the Advisory Committee on Asbestos which on reconsideration would have altered the conclusion in our previous report;

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(b) data which have become available subsequently lead us to alter the conclusion of our previous report in so far as they relate to the control limit in the workplace as follows:

(i) amosite imports have effectively ceased and chrysotile is at present for practical purposes the only type of raw asbestos fibre imported into the UK;

(ii) the evidence that asbestos fibre causes alimentary tract cancer in man is less convincing than in 1979;

(iii) the case that amosite is more dangerous than chrysotile has strengthened in respect of both peritoneal and pleural mesothelioma. We recommend that formal prohibition of the manufacture and importation of new products made of amosite and crocidolite should be considered;

(iv) the range in the slopes of the dose-response relationships for lung cancer and chrysotile exposure has widened since our report of 1979. At one extreme the risk associated with the manufacture of textiles in South Carolina may have been greater than has been previously reported in relation to any other chrysotile application, while at the other extreme the presence of any increased risk with increasing exposure in a large cohort of men who manufactured brake linings is questionable.

106 We consider that subsequent evidence has supported the view expressed in 1979 that in respect of lung cancer the relationship of exposure to mortality is linear.

107 Subsequent evidence has supported our previous view that peritoneal mesothelioma for practical purposes never, and pleural mesothelioma rarely, has occurred in man in relation to exposure to chrysotile alone.

108 In view of the fact that all forms of asbestos can be carcinogenic, further improvement in control should be made as advances in engineering make them reasonably practicable and the use of all types of asbestos should be curtailed as safer and effective substitutes become available.

109 Steps should be taken to ensure that measurements of exposure to asbestos in the workplace are made and recorded in such a way that it is possible to determine what benefits (if any) have occurred as a result of improvements in control of dust.

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110 We draw attention to the fact that little epidemiological information is available about the effects of work in the asbestos cement industry where chrysotile only has been used. As this is now the largest single section of the asbestos industry in the UK, further research should be carried out at the earliest opportunity.

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 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."

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> 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 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.

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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 carried out by the Selikoff group, appear to detect some evidence of radiological changes in brake-service mechanics, but no evidence of frank disease.

At the Annual American Industrial Hygiene Conference in 1977, 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).

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

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dust levels should always be below the minimum required. The main non-industrial use of asbestos is in do-it-yourself bulding 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.

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 mortality from GI cancer. 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.

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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 or 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 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

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penetrate deeper into the lung than others and to reach the pleural cavity by direct penetration.

Substitutes for asbestos are being sought. 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 cold 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?

The human body is able to deal with small doses, or low levels of exposure to cancer-causing materials, with no adverse long-term effect, according to some authorities.

> "The previling scientific view is that human cells are equipped with mechanisms that prevent unrestrained reproduction. Exposure to carcinogens somehow causes some cells to become asocial and reproduce abnormally, eventually invading other parts of the body. A few scientists are of the opinion that one molecule of a carcinogen, acting on a single cell, could eventually result in cancer. Since it has not been proved positively

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> that this does not happen, they argue that all human contact with carcinogens should be prevented wherever possible.
>
> On the other hand, however, daily experience with naturally occurring carcinogens suggests that there is probably a threshold below which there is no harmful biological effect, at least for most of us. For example, the ultraviolet rays in sunlight are known to be potent carcinogens, but most people do not contract skin cancer from being out in the sun. Moreover, our bodies contain trace amounts of a number of elements which are known carcinogens. Recent experiments have shown that certain hormones, such as the female sex hormone estrogen, are also carcinogenic."
>
> (Monsanto. Chemical Facts Bulletin)

Nothing in life is risk-free and society, weighing the known scientific facts in the balance ultimately will decide what risks to take. It is important that society is made aware of improvement in industry and its products to enable it to compare the risks taken thirty years ago with those of today. No unnecessary exposure is justificable and should be avoided as far as is reasonably practicable. Before condemning useful products or useful materials on the basis of incomplete evidence, the consequences of their disappearance from daily use need to be considered.

A major unresolved issue is whether asbestos poses a threat to the public health. A number of events have recently brought this matter to the forefront of the public scene. It has been shown that the mesothelioma rate in Connecticut has increased rapidly in the past ten years and this has been attributed to the increased usage in parallel periods allowing for the time lag for development of the disease. The discovery of asbestos in waste dumps, asbestos in dry wall spackling compounds, asbetos in ember ashes and asbestos

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in hair dryers, has all been publicized and proclaimed as a public health risk. It is at present impossible to determine whether, in the case of consumer products, any hazard whatever exists, but because of the "no safe level for carcinogen theory" the general public must be protected from any unnecessary exposure.

Waste dumps, asbestos emissions from asbestos manufacturing plants, brake lining and clutch-facing wear products and asbestos in public buildings and schools are all examples of sources of low-level dust exposure of varying degree. It is virtually impossible, by means of the best available technology, to reach inter-observer agreement on ambient air levels of asbestos in differing circumstances and even duplicative sampling by two laboratories produces widely differing results. The one exception is the measurement of asbestos in schools with sprayed walls or ceilings. No definite epidemiological studies have been done, and are unlikely to be done, which correlate the minute levels of asbestos in the general environment with the incidence of asbestos-related disease and those which have been attempted are either incomplete in detail or inconclusive in their findings. Even the Connecticut data can be readily faulted because the diagnostic confirmation of mesotheliomas in the Tumor Registry is weak and because occupational history details are unknown for the cases, thus making it impossible at this stage to determine whether they were primarily of occupational or environmental origin.

Many issues remain unresolved including the level at which to set a hygiene standard below whch everybody will be safe. The general public,

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exposed to trace amounts of asbestos in the general environment, does not appear to be at risk, but the added effect of unnecessary exposure from asbestos-containing products in daily use has prompted vigorous demands for the abolition of those products and the banning of asbestos in some instances. This is *not* a scientific issue as no data exists upon which to form a reasoned opinion and will be decided by the mechanisms which society has devised in the form of government bodies and regulatory agencies. Asbestos will continue to be present in the air we breathe and water we drink as it has been since time immemorial. Nobody breathes pure air.

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