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August 30, 1972

To: Asbestos Study Committee

Subject: "Health Hazards of Asbestos", by J. C. Gilson

Enclosed is an article "Health Hazards of Asbestos" by J. C. Gilson.

Mr. I. H. Weaver, Chairman of the Committee, felt this was a good overview of the entire asbestos health situation as it now stands, and suggested that it be distributed to members of the Committee.

EWD/erc
Enclosure

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[redacted]

P-FMSI- 0023

[redacted]

MEMO

from the desk of

I.H. WEAVER

I think the attached article gives a good overview of the entire asbestos/ health situation at it now stands. I suggest you distribute copies to the other members of the Asbestos Study Committee.

Ike

Health hazards of asbestos

J. C. GILSON*

Inhaled fibres of asbestos can cause fibrosis of the lungs and two kinds of cancer. Protection of asbestos workers calls for monitoring and controlling their working environment and linking these records with records of their health

HISTORICAL

Asbestos was the first inorganic fibre to be used in composites. More than 4000 years ago clay pots in Finland were strengthened by adding anthophyllite fibres[1]. In classical times asbestos cloth was used to preserve the ashes of the eminent. The oldest known piece of asbestos cloth from the New World, dating from about 1740, is a small purse made of tremolite in the Sir Hans Sloane collection of minerals in the British Museum (Natural History).

The modern asbestos industry is about 100 years old, starting nearly simultaneously in Canada and the USSR, but it was not until 30 years later that the first medical reports appeared in France and England, indicating that there might be a specific type of damage to the lungs following inhalation of the dust. By the late 1920s it was clear from surveys made in this country and in USA[2,3] that a high proportion of older workers in the asbestos textile industries were becoming severely disabled by a specific type of chest disease due to the dust. This was named asbestosis.

DISEASES CAUSED BY ASBESTOS DUST

Research carried out in the 1930s, supported by the greatly expanded investigations during the last 15 years into the types of disease caused by asbestos, now provides a much clearer picture of the specific hazards and how damage to health can be avoided in the future. Table 1 lists the diseases.

Table 1 Diseases caused by asbestos dust

Asbestosis	Fibrosis of lungs
Cancer	*Bronchial lung)* Mesothelioma
Asbestos Corns	Skin

Asbestosis

In asbestosis the dust causes scarring and thickening of the tissues of the lung. The two parts particularly affected are the finest air passages (respiratory bronchioles) where they branch into the terminal air sacs (the alveoli), and the surface of the lung (pleura). The thickening of the tissues induced by the asbestos dust affects the function of the lung in three ways. The volume when fully inflated at the end of a full inspiration is less than normal. The tissues become stiffer than normal so that the compliance is reduced. The thickening in the alveolar walls, when the disease is extensive, also reduces the gas transfer for oxygen, so that the blood leaving the lung is no longer fully saturated with this gas. The transfer of carbon dioxide from the blood to the air in the lungs is not, however, appreciably affected. The reduced oxygen transfer is partly compensated for by an increase in the frequency of breathing so that the subject notices breathlessness on slight exertion. These alterations of lung function are used to assist in diagnosis of asbestosis and measure the severity of the damage.

Asbestosis takes a number of years to develop, even under very dusty conditions, but once established it is a progressive disease not materially affected by avoiding further dust exposure. The less the dust exposure, the longer interval before the onset of disease, the less its severity; and the less chance of being affected. This dose response relationship is used to fix the acceptable dust levels – threshold limit values – for those working with asbestos. The article by Holmes in this issue describes how these standards are applied in practice. Prevention by avoiding exposures to a dangerous quantity of dust is an essential step towards the safe use of asbestos.

Asbestos cancers

Some years after the recognition of asbestosis as an important problem in the asbestos textile industry, articles began to appear in the medical journals[4] suggesting an association between asbestosis and lung cancer. A survey in 1955 firmly established that those who had worked in the asbestos textile industry before the improvements in dust control, introduced in the 1930s, had a 10-fold excess risk of developing lung cancer[5]. The survey, however, also indicated that the improvement in dust control introduced at a particular factory after 1933 had very materially reduced the risk. Later surveys at this factory have confirmed this[6].

The lung cancers seen in asbestos workers are similar to those caused by cigarette smoking. Recent research indicates that there is likely to be a synergistic effect of cigarette smoking and exposure to asbestos dust[7]. The precise quantitative inter-relationship between asbestos, cigarettes, and other factors is not fully established, but present evidence indicates that those who smoke cigarettes and are exposed to asbestos dust have a risk of developing lung cancer at least fifty times greater than non-smokers who are not exposed to asbestos dust.

In the last 15 years there has been much new information about the link between exposure to asbestos and another previously very rare type of cancer affecting the

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surface of the lung and the gut[8]. Reports of these mesotheliomas as they are called have increased steeply over the last 10 years. There is general agreement in most industrialized countries that there has been a real increase of this form of cancer[9]. In about 80% of cases there is a history of exposure to asbestos dust at some time in the past. A feature of these tumours is the long interval between first exposures to asbestos dust and the detection of the cancer. It is rarely less than 20 years and may be up to 50 or more years. In some instances the exposure to the dust has been short, only a few months, but the highest incidence of tumours has occurred in those most heavily exposed to asbestos dust. Cigarette smoking seems to play no part in these tumours, but some research workers think there may be other co-factors present as well as asbestos[10].

The only other specific injury caused is the formation of asbestos corns on the fingers when the fibres lodge in the skin. The removal of the fibre usually cures the corn and no cancers of the skin relatable to asbestos have been reported.

PRACTICAL IMPLICATIONS OF THE BIOLOGICAL EFFECTS OF ASBESTOS

Inhalation of the fibre

For all practical purposes the risk from asbestos is limited to inhalation of the fibres. Thus control of the airborne dust levels and their monitoring by instruments, which will measure that part of the dust which can gain access to the deeper parts of the lung, is an essential step in the safe use of all types of asbestos. Although asbestos fibres can be ingested in minute amounts in beverages which have been filtered through asbestos, or water supplies[11], there is no firm evidence that such tiny traces have any ill-effects. Feeding massive doses of asbestos to animals has so far failed to produce any mesotheliomas or other cancers.

Size and shape of fibres

Recent research has helped to clarify the probable influence of fibre length and diameter in producing asbestosis and the bronchial cancers. The fibrosis is thought to be caused principally by the fibres between about 5 and 100 μm in length. Fibres much larger than this in the environment settle out quickly and are not inhaled. Further size separation occurs in the air passages, the important size parameter being fibre diameter since it is this dimension rather than fibre length that governs the falling speed of the fibres. Thus fibres greater in diameter than about 2 μm (these also tend to be the longest) mostly fall or impact in the upper respiratory tract and are carried away with the sputum. In the narrow airways any long fibres remaining are deposited by interception and fibres longer than about 100 μm seldom reach the finest bronchioles. This means that for the control of asbestosis, and probably bronchial cancers, the dose of fibres between about 5 μm and 100 μm in length and up to about 2 μm in diameter is the fraction of the dust which has to be measured.

For the mesotheliomas the evidence about the biologically important size is much less complete. Using information from many sources, such as the aerodynamic behaviour of fine fibres, the size and shape of fibres which are retained in the lungs of animals and man following exposure to different types of asbestos, and the epidemiological studies of the incidence of these tumours in man, for different types of fibre, it seems probable that the important fibres are likely to be those which are straight, small in diameter (up to about 1 μm) and perhaps 10 μm in length. It is not yet known whether the ultra-fine fibres only visible under the electron microscope are biologically important. Such fibres are present in large numbers in the lungs of those who have been exposed to asbestos, but their combined mass is extremely small. It is too early yet to use this information to establish with confidence a separate dust standard to prevent the development of these mesotheliomas. This is a field of intensive research at the present.

Some of the new evidence suggests that the size and shape of the fibre are more important than its chemical composition, provided it is relatively insoluble. It may be that extremely fine fibres of many different materials can penetrate cells without immediately killing them, but once inside can damage the mechanisms of cell divisions. An implication of this view is that care should be taken to avoid exposure to dusts of all types of fibre less than 0·5 μm diameter and several micrometres in length.

Types of asbestos and occupations within the industry

The last 10 years have shown the importance to health of the type of asbestos inhaled and the occupation of the workers within the industry. Earlier medical reports did not differentiate between one type of asbestos and another, and most of the surveys were concerned with asbestos textile workers. A full assessment of the risks would ideally be based on studies of workers only exposed to each type of fibre and in all the operations within the industry in which this fibre was used. In practice the information is much less complete than this. Exposures to one type of fibre have usually occurred only in the mining and fibre separating. This work usually takes place in countries where the medical records are scanty and the labour turnover is rapid. A notable exception is in the chrysotile mines in Quebec where a very comprehensive survey has just been completed[12–14]. In the manufacturing countries several types of fibre are often mixed together or have been processed concurrently so that employees have been exposed to several types of fibre in unknown quantities. Past records of dustiness are rarely available for relating to the incidence of the diseases. Thus the current assessment of the relative risks in the past from different types of fibre and occupations is based on information which is far from complete. Use can be made of experiments in animals, especially rats, because most of the diseases seen in man can be produced in these animals. New information is rapidly accumulating which may give a clearer indication of the way in which different types of asbestos produce their biological effects.

There is general agreement that asbestosis and bronchial cancers can be caused by all types of commercially used asbestos (amosite, anthophyllite, chrysotile, and crocidolite) if the dust is inhaled in sufficient quantities, but it now seems likely that the risk from chrysotile may be less than with the other types of fibre. There is also evidence that the risk is lowest in mining and increases along the fibre separating and manufacturing processes. This is thought to be due to the higher proportion of airborne dust consisting of respirable fibres able to penetrate into the deepest part of the lung. In practice this means that the cleaner the fibre and the more completely it is separated into individual

fibres and small bundles, the greater the risk. There is also good evidence of a dose response relationship for asbestosis and bronchial cancers and thus, if the dust levels are kept within the new standards, the risks of asbestosis and bronchial cancers in the future should be very small.

The risk of developing mesotheliomas has a different relation to fibre type. It is probably highest with crocidolite and lowest with chrysotile. No cases clearly related to anthophyllite alone have been reported, despite careful search. The risk with amosite probably lies between crocidolite and chrysotile. The evidence for a dose response relationship is less clear in the case of mesotheliomas and hence the threshold limit value is more difficult to assess. In the Asbestos Regulations 1969[15] the standard for crocidolite is set at one-tenth of that for other types of asbestos.

COMPARISON OF OCCUPATIONAL RISKS

What is the magnitude of the risk of developing ill-health from asbestos? No single index provides a satisfactory measure of injury to health.

Thus the excess risk of death before a specified age may be a useful index for those diseases causing sudden or rapid death, but it is an inappropriate index for diseases causing a long period of disability but little shortening of life. The cancers associated with asbestos exposure fall into the first group and asbestosis the second group. In different occupational groups comparisons of mortality are easier to make than those of illness. But even for mortality the comparisons are not straightforward. For example, the more the selection is limited to a definable group with a high past exposure, the worse the risk will appear. Allowance has also to be made for the effects of age, length of exposure, and how long the occupational group has been followed.

When such allowances are made the excess mortality from 'all causes' in groups of workers heavily exposed to the more damaging types of asbestos dusts in the past is closely comparable to that of coalminers who have developed the severer form of coalworkers' pneumoconiosis or that of deep-sea fishermen who have the highest rates of accidental deaths of any occupation. However, the excess mortality from 'all causes' in these occupational groups is less than that of male smokers of twenty cigarettes and more a day compared with non-smokers.

This is the position for deaths from 'all causes', but deaths from specific causes, such as mesotheliomas, asbestosis, or lung cancers relative to that of the general public, are of course proportionately much more increased. This is because mesotheliomas and asbestosis are extremely rare in those who have not worked with asbestos. Confusion sometimes occurs between the proportion of individuals exposed who develop a disease and the excess risk of a particular disease in exposed individuals compared with the general public. The first may be relatively small and the second extremely high.

THE FUTURE

As in many occupational diseases proof of the efficacy of new preventive measures, including the validity of the current threshold limit values, can only come from linking information of three types — the manufacturing process and the types of fibre and other materials used; the measurements of exposure; the medical records of those exposed. Computers now make it easy to store this information, but we still need the foresight and administration to see that it is achieved. If it is not done we may still in 20 years or so be in no better position to answer important questions which are at present unanswerable because of the paucity of past records.

CONCLUSION

The recent increase in the number of cases of asbestosis and other diseases related to past exposure to asbestos is the result of relatively heavy exposures to the dust, particularly in parts of the asbestos industry not covered by the 1931 Asbestos Regulations.

Much new information about the biological effects of asbestos has been acquired recently. The new Asbestos Regulations 1969 based on this information, if correctly applied, should greatly reduce the risks in the future.

The selection of which types of asbestos to use in new processes should take into account their biological effects if the risk of dust exposure is likely to occur during manufacture or in the use of the products.

When working with extremely fine fibres of any material which may become airborne, caution is needed. An examination of the possible biological effects is required if the product is to be widely used and risks of damage to health are to be avoided in the future.

Proof of the efficacy of present preventive measures depends on much better record keeping than has been the case in the past.

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