



REPORT

Asbestos exposure and disease: notes for medical practitioners

This pamphlet has been developed from the Asbestos Advisory Committee's report to the Minister of Labour on medical and industrial issues.

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The report has been modified for publication and distribution as educational material concerning asbestos exposure and disease for doctors by Dr C Walls of the Occupational Safety and Health Service, Department of Labour.

SUMMARY

Significant exposure to any type of asbestos fibre presents a health hazard. This may lead to an interstitial fibrosis of the lung (asbestosis) or a cancer. Occupationally-related diffuse malignant mesothelioma (a particular type of cancer) is associated with exposure to crocidolite (blue asbestos) and amosite (brown asbestos) and, less commonly, from exposure to chrysotile (white asbestos).

It would appear that asbestos-related disease follows a dose-response relationship and that there is a threshold of fibre burden in the lung for the development of cancer.

The present Workplace Exposure Standard for asbestos is considered adequate for the protection of workers exposed to asbestos dust if they are exposed to that level of asbestos throughout their working life. The Standard takes no account of previous exposures. A medical surveillance system is recommended for those exposed during work.

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A National Asbestos Disease Register and an Asbestos Exposure Register together with two asbestos medical panels (the National Asbestos Medical Panel and the Asbestos Radiological Panel) have been established. The Occupational Safety and Health Service of the Department of Labour (OSH) is the organisation responsible for maintaining these registers and panels and can be contacted for information at:

The Asbestos Registers,
PO Box 3705,
Wellington.

DEFINITIONS

In the context of this pamphlet, the following words are used with the ascribed meanings:

Alveolus	The terminal air sac of the lung.
Asbestos	Naturally occurring fibrous hydrated silicates belonging to the serpentine and amphibole groups.
Asbestosis	A diffuse interstitial fibrosis of the lung resulting from exposure to asbestos.
Bronchus	An air passage in the lung.
Cancer of the lung	A malignant tumour of the bronchus.
Carcinogen	A substance capable of initiating changes in the body leading to cancer.
Hazard	The potential of a substance, material or process to affect adversely human health.
Mesothelioma	A malignant tumour usually arising from the lining of the chest cavity or the abdomen. Occasionally it may arise from the membrane surrounding the heart.
Pericardium	The fibrous sac surrounding the heart and base of the great vessels.
Peritoneum	The membrane lining the abdominal cavity. It is derived from the same type of embryological tissue as the pleura.
Pleura	The membrane lining the chest cavity and the lungs.
Pleural effusion	A collection of fluid in the pleural cavity
Pleural plaque	A non-malignant, localised mass of tissue arising from the pleura.

Pleural thickening

A more diffuse, non-malignant, mass of tissue arising from the pleura.

Risk

The mathematical chances of someone developing an adverse consequence from exposure to a particular substance, material or process, over a known period of time.

Increased risk

That the risk of developing an adverse consequence in the group to which the term is applied is higher than in the general population.

Significant risk

The exposure that has the potential to cause asbestos-related disease. Such exposure takes into account:

- The work history of the exposed person;
- Any high, short-term exposures;
- The length of time of exposure; and
- The levels of asbestos dust to which the person was exposed.

Workplace Exposure Standard

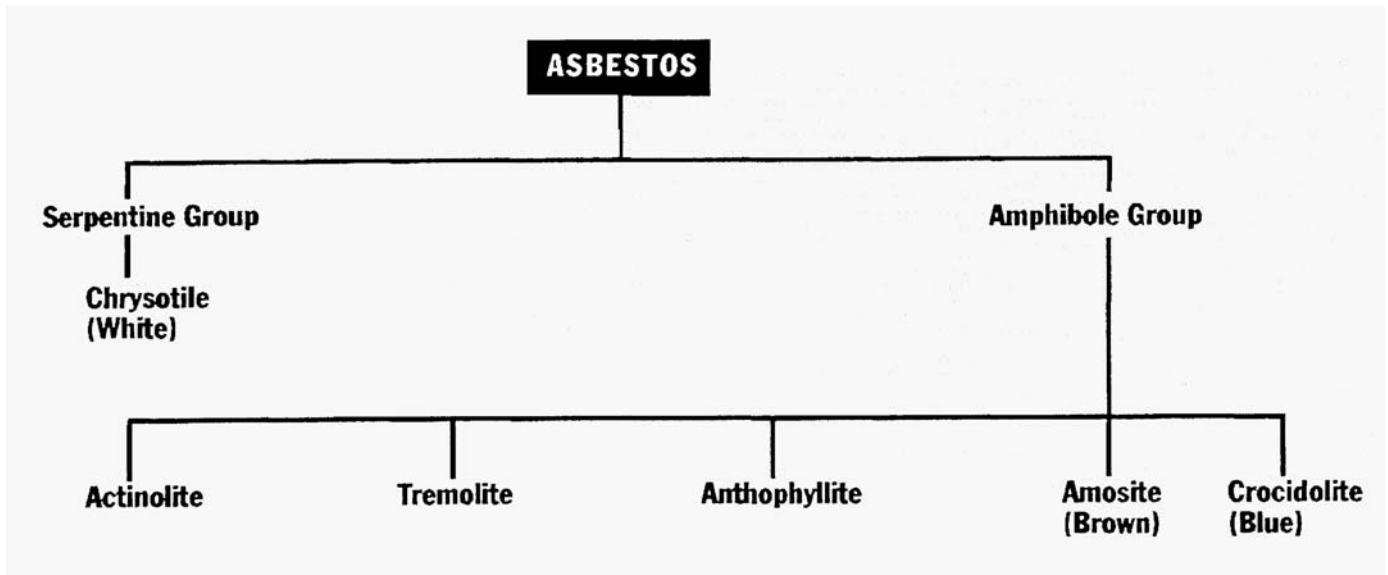
That level of a hazard which the majority of workers can be exposed throughout their working life in a normal working week without suffering an ill-health effect. Workplace Exposure Standards are guidance values only and have no legal authority **except** in the case of asbestos, where the Asbestos Regulations 1983 give these values legal force. The common abbreviation is **WES**.

HISTORY OF ASBESTOS USE

Asbestos has a long history of use. It was probably first used about 2500 years ago in Finland to strengthen clay pots. In classical times, the Greeks used it to weave shrouds for the bodies of the great who were to be burnt on funeral pyres.

Its widespread use in industry began about 1880, with the exploitation of large chrysotile deposits in Canada and Russia. Initially, its main use was in the textile industry to produce incombustible products and improved materials for gland packings.

Because of its relative cheapness and unique properties, it has had a wide variety of uses, the commonest of which were in asbestos cement products such as pipes, roofing sheets and wall boards; in insulation; and in friction products.



TYPES OF ASBESTOS

Asbestos is not a single chemical or geological entity, but a term used to describe naturally occurring fibrous hydrated silicates. There are six common varieties (see fig. 1).

Amosite and crocidolite (the amphiboles) are chain silicates. Their fibres are straight, needlelike structures which may split longitudinally to produce very fine fibrils.

By contrast, chrysotile fibres tend to be longer, softer and curlier. This is important from the viewpoint of lung dynamics as this property has the effect of increasing their diameter and making it less likely that they will reach the smaller airways.

HISTORY OF HEALTH EFFECTS OF ASBESTOS

The information in this section is largely derived from a paper detailing the history of the knowledge of the health effects of asbestos by Murray¹.

Although fibrosis of the lung was first recognised as being associated with asbestos exposure in 1899 in the United Kingdom by Dr Montague Murray at Charing Cross Hospital, he did not report on this until 1906. This association was largely ignored because of the greater problem at that time of pulmonary tuberculosis.

In the 1920s, a greater interest developed in fibrosis of the lungs amongst asbestos workers. A comprehensive study on the health effects of asbestos was carried out in the United Kingdom by Merewether (HM Medical Inspector) and Price

(an engineering inspector colleague) between 1928 and 1930. This study led to the first-ever asbestos regulations, the UK Asbestos Regulations of 1931, which came fully into force in 1933.

During the 1930s there was continued interest in asbestosis. In the late 1930s a few papers described cancer of the lungs in asbestos workers. It is perhaps relevant to note the greater importance of pulmonary tuberculosis as a cause of morbidity and mortality at that time, and this association was not pursued.

In 1947, Merewether, now the Chief Medical Inspector of Factories in the UK, initiated a study to determine whether lung cancer was more common in asbestos workers. This led to a study by Doll, published in 1955, which showed that lung cancer was a specific hazard of certain asbestos workers, and that for those who had worked for at least 20 years in places where they were exposed to asbestos dust, the risk was 10 times that experienced by the general population. The role of smoking was not reported.

Little attention was paid to this discovery. This may have been due to the fact that the major dust problem at that time in the UK was coal dust, and that relatively few people were involved in the asbestos industry. Moreover, the major cause of lung cancer for the population at large was recognised as being cigarette smoking.

In 1960, a paper by Wagner *et al* in South Africa was published which showed, for the first time, an association between exposure to crocidolite (blue asbestos) and the development, many years later, of the rare tumour-diffuse malignant mesothelioma. This led to great interest in asbestos-related health problems throughout the world, followed by a flood of scientific papers and great media interest.

A major problem that followed from these

associations between asbestos and disease was what should be the allowable concentration of asbestos fibres in the workplace. The existing workplace standard in the UK had been developed to control asbestosis, and there was no information about the levels of asbestos fibres to which those who developed mesothelioma had been exposed. An arbitrary decision was made that the standard for crocidolite should be one-tenth the standard for other forms of asbestos.

The debate on this issue continues.

ASBESTOS USE IN NEW ZEALAND

In New Zealand, the use of asbestos has been restricted to chrysotile, amosite and small amounts of crocidolite.

Chrysotile has for many years been the most commonly used fibre and today accounts for some 95% of the world production. Importation of amosite and crocidolite in its raw fibrous state has not been permitted into New Zealand since 1984. These varieties may, of course, be encountered in buildings, principally as insulation or as fire barriers where they were applied before the ban took effect.

Asbestos has also been used extensively in the railway workshop industry, the building industry, the shipping industry, the sawmilling industry, and in the asbestos cement industry in Christchurch and Auckland. Workers unloading asbestos cargo onto the wharves, fitters, electricians, boiler workers, carpenters, brake repairers and others were commonly exposed. There were no asbestos textile industries developed in New Zealand, so that the New Zealand asbestos experience does not include the very high asbestos dust levels encountered in this industry.

Mesothelioma as a consequence of exposure to asbestos in New Zealand was discussed in a letter to the editor of the *New Zealand Medical Journal* by Glass². Up to 1989, 174 cases had been notified to the Cancer Registry.

Asbestosis and lung cancer have not been effectively notified to the appropriate government departments, and the extent of this condition is unknown. However, a case control study in New Zealand by Glass *et al* indicated that overall, asbestos-related occupations were found to be associated with elevated risks of cancer in three sites—the lung, pleura and peritoneum—and that the risks of cancer of these three sites were highest among the group of machinery fitters, plumbers, welders, boiler makers, metal moulders, metal platers and electricians³.

Of particular note in New Zealand was the prevalence of markers of asbestos exposure and disease in two particular sites where asbestos cement products were manufactured. These were the Fletcher plant in Mandeville Street, Christchurch, and the James Hardie plant in Great South Road, Penrose, Auckland.

The New Zealand Asbestos Regulations were first promulgated in 1978 and were amended in 1983. As a result of the Advisory Committee's recommendations, new regulations will be written in 1992 and a code of practice developed. The New Zealand Workplace Exposure Standard for asbestos has been kept constantly under review and has been revised twice since 1978 in order to provide greater protection for those who may be exposed to asbestos.

ASBESTOS-RELATED DISEASES

In general terms, the chances of developing asbestos-related disease depend on four main factors. These are:

1. The type and size of asbestos fibre inhaled;
2. The number of fibres inhaled;
3. The length of time in which that inhalation continued;
4. Other individual factors.

Reaction of the Body to Asbestos

Exposure to varying amounts of asbestos is almost universal in so called "developed" societies. Apart from occupational exposures, however, the amounts will generally be very small. Post mortem examinations on people who have died from unrelated causes, nonetheless, reveal the presence of asbestos fibres in the lungs of the great majority of people. Therefore, it appears that it is not the presence of asbestos that causes disease, but the dose of fibres received.

The body may react to the presence of asbestos in different ways. These different reactions appear generally to be dose-related.

Asbestos may be present in body issues, such as the lungs, without causing any disease or alteration of function.

Asbestos may cause localised tissue reactions which rarely have any effect on function and for which there is no evidence at present that they may

progress to actual disease. These are referred to as **asbestos-related conditions** and indicate exposure to asbestos in the past. These conditions are not at the moment thought to act as predictors of disease, although there is debate about this.

Such reactions may be seen in the skin and the pleura (the lining membrane of the lung). So-called asbestos "warts" are caused by asbestos fibres penetrating the skin and causing a reaction. Benign pleural effusions occur in a small percentage of asbestos workers, usually less than 20 years after the initial exposure to high levels of asbestos. In most instances, the effusion resolves spontaneously⁴.

Pleural plaques are a common manifestation of exposure to asbestos. They occur as localised nodular lesions, most often on the parietal pleural. Their relatively discrete nature and location accounts for the virtual absence of decrements of lung function in people in whom they are found. Calcification may occur with time, but does not necessarily imply an enlargement of the lesion⁴.

Pleural thickening is a relatively common manifestation in which there is a pleural reaction. Extensive pleural thickening may lead to a reduction in lung function by interfering with ventilation⁴.

Asbestos can cause illnesses and these are referred to as **asbestos-related diseases**.

Inhaled asbestos may induce tissue damage in the lungs, resulting eventually in the development of a progressive diffuse interstitial fibrosis called **asbestosis**. This may be associated with the pleural changes described above. It only occurs in those who have been exposed to considerable concentrations of asbestos over a long period of time. This disease can progress even though exposure to asbestos has stopped.

Bronchogenic (lung) carcinoma is associated with exposure to asbestos. The degree of association varies with the type of asbestos fibre morphology, concentration and type of exposure. Asbestos textile workers have a higher relative risk than those exposed to asbestos in other ways, though this industry was not present in New Zealand. There is also evidence that lung cancer only develops in association with pre-existing inflammation and subsequent fibrosis (asbestosis), but this remains a matter of debate⁴.

Asbestos may also induce malignant changes in pleura and peritoneum (the lining tissue of the abdomen and intestines) causing diffuse malignant **mesothelioma**. This malignant tumour arises many years after exposure to asbestos. The

average latency period between the first exposure and the diagnosis of mesothelioma is 35 to 40 years, with most deaths occurring in people over 60 years of age⁴.

In the vast majority of people there is a past history of exposure to crocidolite (blue asbestos) or amosite (brown asbestos). A recent study does support the view, however, that chrysotile (white asbestos) exposure may cause mesothelioma⁵.

There is no association between mesothelioma and smoking history.

In addition it has been suggested that asbestos exposure may also produce malignant change in other parts of the body, particularly the larynx and upper gastro-intestinal tract. The evidence, however, appears equivocal and the matter is still under debate.

FIBRE TYPE, DOSE AND DISEASE

Fibre Characteristics

When considering the potential health hazard of asbestos, it is important to recall that asbestos is not a single chemical or mineral substance. As discussed in the earlier section, there are two main types of asbestos: serpentine (chrysotile) and the amphiboles (crocidolite and amosite).

Chrysotile is a magnesium silicate, white in colour. The fibres are relatively easily separated from the parent ore and form bundles which are soft and curly. The best-quality fibres may be up to 5 cm long, which make it useful for weaving. However, any form of mechanical treatment tends to break them, resulting in thinner and shorter fibres which may be in the respirable range. The magnesium which is present in the outer layer of the fibre may leach out in the body fluids, resulting in an unstable structure. This may lead to the dissolution of the fibre in the body, digestion by macrophages and so reduction in the body burden of asbestos dust.

Crocidolite is an iron-sodium silicate, blue in colour. The fibres are straight and rigid and may split longitudinally to produce fine fibrils. Because of their outer lining, they are relatively resistant to acids and to body fluids. This means that they may survive unchanged in the body for at least 40 years and the finest fibrils may migrate through the lung to the pleura.

Amosite is an iron magnesium silicate, grey brown in colour. The fibres are harsh and tend to be longer than the other types of asbestos, making it

excellent for heat insulation. As with crocidolite the fibres may survive unchanged in the body for many years.

Inhalation of Asbestos Fibres

The eventual fate of asbestos fibres which are inhaled depends very largely on their physical characteristics.

The larger fibres are filtered out in the nose and throat.

Smaller fibres pass into the bronchi. Because of the way the inspired air spins in vortices as it passes into the lungs, many inspired fibres are thrown outwards and caught on the sticky mucous lining of the airways. These fibres are then carried upwards in the mucous and, in due course, expectorated or swallowed.

The fibres that escape this defence mechanism must be light enough to remain in suspension and short enough not to get caught in the smaller airways. The majority of the inspired longer curlier chrysotile fibres are, therefore, eliminated by this mechanism and do not reach the alveolus.

Although this filtering mechanism favours the chances of the smaller fibres (less than 10 microns in length) reaching the lung tissue, it is possible for larger fibres, particularly if they pass obliquely or longitudinally through the airways, to reach the alveolus.

When dust or an asbestos fibre reaches an alveolus, it is normally removed by a scavenger cell to the tissue surrounding the alveolus. If the asbestos fibre is too big, the cell may rupture and the fibre may initiate a tissue reaction leading to the formation of scar tissue or many years later to a cancer.

The biological activity of fibres increases with fibre length. The threshold for potential cancer causation is probably about 3 microns and peaks at 10 microns. It then declines, probably because the longer fibres are prevented from penetrating deeply into the lung tissue.

Fibre Type and Disease

ASBESTOSIS

There is little evidence to suggest that the risk of developing asbestosis is affected by exposure to different fibre types. It is probable that the type of work done is more relevant, as in some industries using asbestos there is inherently more dust. In the textile industry, based exclusively overseas, for example there is more likely to be a higher

concentration of asbestos fibres in the workplace air than in the asbestos cement industry which is New Zealand's industrial experience. This may help to explain the low incidence of asbestosis recorded in New Zealand statistics.

The evidence suggests that the risk of developing asbestosis is closely linked to the intensity and duration of exposure.

LUNG CANCER

Exposure to airborne asbestos fibres leads in some circumstances to lung cancer. Due to the relatively long latent period between exposure and the onset of clinically manifest disease those people with lung cancer at present are reflecting their degree of exposure in the past. Frequently the concentration of fibre to which they have been exposed is unknown, but was almost certainly higher than the concentrations to be found at work today.

The important issue thus arises as to whether a carcinogenic threshold exists for asbestos. This is particularly important when considering the practical situation of short-lived exposure to asbestos in buildings.

There are two schools of thought⁶. According to the "no threshold" hypothesis, one molecule of carcinogen, or one fibre, by damage to the biological material, can change the cell into a cancerous cell. This one cell then multiplies and eventually produces cancer. This theory has obvious implications for those exposed environmentally to asbestos.

On the other hand, the "threshold" hypothesis recognises the possibility of repair and the existence of a complex defence mechanism in the body which copes with individually deviated cells, such as cancer cells spontaneously appearing throughout the lifetime. Cancer can only develop, if and when the defence mechanism is overcome by large numbers of such cells created by a large number of "hits"-that is, when the "threshold" of tolerance is overstepped.

Browne reviewed eight epidemiological studies in which there were lower levels of exposure under which no excess of cancers is found. These results support the threshold hypothesis.

Most authorities agree that the evidence is compelling from both animal studies and human epidemiology that fibres shorter than 5 microns do not cause fibrosis or cancer. It seems generally to be agreed that both fibrogenicity and carcinogenicity are mediated through damage and repair mechanisms.

Although in the past a linear relationship between dose and response in respect of asbestos-related lung cancer has been proposed, there is good evidence for a threshold response, and a carcinogenic dose, which may in fact be higher than the fibrogenic

dose. Since Weil proposed this view in 1979, there has been a growing number of studies which suggest a threshold and which have demonstrated that lung cancer rates have not been raised in low exposure situations.

Further, recent studies have found positive evidence that the lung cancer risk was only increased in the presence of radiological or pathological evidence of asbestosis⁷.

MALIGNANT MESOTHELIOMA

It was in 1960 that the first cases of mesothelioma were reported as occurring in those mining crocidolite in South Africa. Since then, a large number of studies have confirmed that occupational mesothelioma is associated with exposure to crocidolite and with a lesser risk from exposure to amosite. Rogers *et al* now suggest chrysotile exposure alone may be sufficient to cause mesothelioma⁵.

The size of the fibre is also of importance. The results of animal studies suggest that mesotheliomas are caused by fibres longer than 5 microns. Unpublished work, presented at the International Conference of Occupational Health in Montreal 1990, suggests that, in humans, the greatest risk of mesothelioma is associated with exposure to crocidolite fibres equal to or greater than 10 microns in length, and that there is a clear dose relationship in the initiation of these cancers.

ASBESTOS EXPOSURE AND SMOKING

The exact relationship between the effects of asbestos exposure and cigarette smoking remains a matter of debate.

It should be noted that asbestos itself is

carcinogenic, and a clear risk exists for anyone working with asbestos. Exposure to asbestos increases the risk of lung cancer in both smokers and non-smokers, with the highest risk being in those with exposure to both agents (see table 1). Asbestos-related lung cancer may occur at exposure levels which are too low to cause asbestosis, and as with other asbestos-related medical disorders, has a latency period of at least 15 years from first exposure.

In this situation, where the two effects are multiplied, smoking can effectively be ignored when considering the cause of disease for industrial compensation. This concept is clearly accepted by the Accident Compensation Corporation as they have granted compensation quite correctly to workers who developed lung cancer after significant asbestos exposure despite their co-existing heavy smoking. By recognising this state of affairs, medical practitioners can be of great help to their patients by facilitating their claims for rightful compensation.

THE ASBESTOS REGISTER

Surveillance of Asbestos Workers

Today, most workers exposed to asbestos work in the asbestos removal industry. Other industry groups that may suffer exposure include people exposed to in situ asbestos such as technicians running cable through false ceilings in commercial buildings and workers demolishing or renovating older buildings. Surveillance during these persons' working lives may be of value because it enables:

1. The documentation of exposure, and changes on X-ray and/or lung function testing for compensation purposes; and

Table 1: Relative Risks and Interactions of Asbestos Workers and Lung Cancer⁸

	Relative Risk			
	Hammond <i>et al</i>	Liddell <i>et al</i>	Selikoff <i>et al</i>	Berry <i>et al</i>
Non-exposed non-smoker	1.0	1.0	1.0	1.0
Asbestos exposure alone	5.2	3.0	17.5	10.0
Smoking alone	10.6	4.9	7.0	11.7
Smoking and asbestos exposure combined	52.2	8.2	32.7	25.7

2. The opportunity to discuss with workers their fears relating to past exposure and to reinforce the safe working practices essential to the safe handling of asbestos.

The Asbestos Regulations 1983 and the new regulations currently being prepared will require medical examinations every three years for designated workers.

A surveillance medical examination should include a full review of the medical history including the occupational history, a physical examination and other procedures as considered appropriate by the examining occupational or chest physician.

Surveillance of People Exposed to Asbestos in the Past

The National Asbestos Medical Panel believes that there is no good evidence that any medical surveillance of people exposed to asbestos in the past provides benefit in terms of the prevention of asbestos-related diseases to those surveyed.

Surveillance remains a matter that the person and their doctor should decide on an individual basis.

The panel does recommend that those people who in retrospect decide that they have had an exposure to asbestos have a baseline chest X-ray and base line lung function tests consisting of Forced Expiratory Volume (1 sec) and Vital Capacity. These could act as a reference if these people were to develop respiratory symptoms at a later date or request further ongoing surveillance.

DIAGNOSTIC PROCEDURES

Medical Procedures for People not Covered by the Regulations

A detailed occupational history is crucial if a doctor is to recognise that a medical disorder is related to previous exposure to asbestos. As most asbestos-related medical disorders present 20 to 40 years after initial exposure, the occupational history must include details of exposure to asbestos in any form, in all jobs the worker has ever had.

The most important investigations in the assessment of a patient with suspected asbestos-related medical disorder are a chest X-ray (ideally both a PA

and a lateral film) and lung function tests. Chest X-ray abnormalities will be present for most asbestos-related medical disorders prior to the development of the major clinical respiratory symptoms, and are an essential feature of the diagnosis of non-malignant asbestos-related diseases. The National Medical Advisory Panel will use the ILO international classification to record the nature and the extent of any radiological abnormalities.

Lung function tests are essential in the assessment of the degree of respiratory impairment of patients with asbestos-related medical disorders. The simplest useful measurements of lung function are the forced vital capacity (FVC) and the forced expiratory volume in one second (FEV1) and it is important that the test is done properly, with maximal effort. The lung function results must be interpreted in association with the level of breathlessness to determine respiratory impairment.

It is thus recommended that all patients forwarded to the Asbestos Medical Register for consideration should have both a chest X-ray and spirometry. If there is difficulty undertaking either or both these investigations, they will be organised by OSH.

THE WORKPLACE EXPOSURE STANDARD (WES)

The purpose of the workplace standard is to establish a minimum standard of asbestos exposure for the protection of workpeople. This standard assumes that most people exposed to this level of asbestos fibre or less throughout their working life (normal working hours) will not suffer any adverse health effects from exposure to the asbestos.

The Workplace Exposure Standard should not be regarded as marking the boundary between safety and disease. Every effort should be made to reduce air borne concentrations to the lowest possible level. The importance of good housekeeping practices should also be noted. Unless asbestos waste products are carefully disposed of during work, their later removal may significantly increase airborne concentrations.

In order to be certain that the workplace standard is not exceeded, there must be appropriately trained people to take environmental air samples, and a laboratory with suitable equipment. Inquiries about such tests and their costs can be made to OSH and the DSIR (shortly to become a Crown Research Institute).

The present Workplace Exposure Standard for asbestos is based on the measurement of asbestos fibres collected by using a personal sampler with a membrane filter. At present the gazetted standard is⁹:

Actinolite, anthophyllite, chrysotile and tremolite

- (1) An average concentration over any four-hour period of 1 fibre per millilitre of air; or
- (2) A maximum concentration over any 10-minute period of 6 fibres per millilitre of air.

Crocidolite and amosite

A concentration over any four-hour period of 0.1 fibres per millilitre of air.

(Though actinolite, anthophyllite and tremolite were not used in New Zealand, they are included in the standard for completeness and because the exact constituent nature of many processed asbestos products imported into New Zealand is unknown.)

RESPIRATORY PROTECTIVE EQUIPMENT

The Asbestos Advisory Committee noted that, in 1984, the Department of Health issued a list of respiratory protective equipment approved for use by those working with asbestos. OSH is updating the standards for respiratory equipment with respect to utilising testing results from overseas, especially from Worksafe in Australia, and will publicise this.

Good occupational hygiene practice requires that all practical efforts be made to prevent asbestos dust from entering the air of the workplace. In circumstances where it is impracticable to prevent asbestos from entering the atmosphere, suitable respiratory protective equipment must be worn.

This section provides guidance on the suitable types of respiratory equipment available for this purpose. Suppliers of this equipment will be able to provide evidence that the device being purchased or used has been approved to an acceptable standard as set out in this document.

General

Respirators to protect against asbestos can be grouped into three main types:

1. Half facepiece air purifying.
2. Full facepiece air purifying, including powered air purifying.
3. Air line respirator, including self-contained breathing apparatus.

There are two ways of providing personal respiratory protection against contaminants such as asbestos:

1. Purifying the air taken from the working environment; or
2. Supplying the person with good-quality air from outside the working environment.

The three main factors that influence the degree of protection afforded by a respirator are:

- the filter type,
- the respirator face fit, and
- the pressure within the respirator.

A respirator described as "positive-pressure" is one in which the air pressure inside the facepiece is positive (or greater) than the air pressure outside during exhalation and inhalation, because air is supplied.

A "negative-pressure" respirator is one where the air pressure inside the facepiece during inhalation is negative (or less) than the air pressure outside.

Respirator Protection Factors

Respirator Protection Factors (RPFs) are used to assist in the correct selection of respirators according to the characteristics of the hazards involved, the capabilities and limitation of the respirator, and the fit of the respirator on the person.

The RPF is a measure of the degree of protection provided by a respirator to a wearer. This is defined as the ratio of the concentration of air contaminant outside the respirator to that inside the respirator. To assist with this decision, Appendix 1 (p.14) contains a list of asbestos levels likely to be encountered in different work activities.

Respirators Approved by the Director-General of Health for Use With Asbestos

The following types of respirators are approved by the Director-General of Health for use when working with, or handling, asbestos as provided for under Regulation 15 (2) of the Asbestos Regulations 1983.

Respirators are arranged in three classes representing three different respirator protection factors (RPFs). The choice of factors depends on the mode of operation of the device.

The RPF is that assigned to the respirator by the Standard or approving authority, as the case may be. The RPF depends upon the device being properly fitted and the wearer being instructed in performing facial fit checks in accordance with AS 1715:1991 *Selection, Use and Maintenance of Respiratory Protective Equipment*.

Selection of Appropriate Respiratory Protective Equipment

The degree of respirator protection required for asbestos work is determined by the nature of the work, the type of asbestos, the work method, and the potential for exposure to dust.

Because of the variability of contaminant and the unpredictability of asbestos levels, people requiring respirator protection for asbestos work are encouraged to use the highest level of protection while taking into account economic and practicability factors (AS 1715:1991 provides further advice).

In selecting the correct equipment, the following factors need to be considered:

1. The maximum levels of asbestos dust likely to be encountered.
2. The RPFs of the chosen respiratory equipment.
3. The nature of the work the wearer is to undertake.
4. Personal requirements of the wearer, e.g. facial hair, glasses.

NOTE: The degree of protection is governed by the type of filter and facepiece type, and the effectiveness of the individual facial seal each time the device is put on. Significant leakage will occur if facial hair passes under the seal.

Standards for Respirators

All respirators which fall into the three classes by general description must comply fully with one of the following standards

- NIOSH/MSHA (National Institute for Occupational Safety and Health/Mine Safety and Health Administration, USA)
- CEN (European Committee for Standardisation)
- AS 1716:1991 (Standards Australia).

To maintain consistent quality, each of these standards refers to ongoing quality assurance programmes which facilitate compliance with performance standards. In addition, the relevant statutory authority in the country of origin will generally approve devices on the understanding that this process is adopted.

Each standard states that, to comply with the standard, assembled respirators shall consist of components which have been tested as a system. The use of components other than those tested as a system is to be discouraged because performance and efficiency may be compromised.

Classes of Respirator

CLASS I

Type of Respirator

Half facepiece respirators, negative-pressure, disposable, or replaceable particulate filter devices (these devices will have a minimum of Class P1 or Dust/Mist filters).

Devices must comply with NIOSH/MSHA, CEN or AS 1716:1991.

Job

Simple short-term bulk sampling. Hand work only on materials such as asbestos cement, or gaskets containing asbestos.

Assigned Respirator Protection Factor

Up to 10 X Workplace Exposure Standard.

CLASS II

Type of Respirator

A. Full facepiece negative-pressure particulate respirators with Class P3 or HEPA (High-Efficiency Particulate Air) filters.

B. Full facepiece powered air purifying particulate respirator with Class P3 or HEPA filters.

Devices must comply with NIOSH/MSHA, CEN or AS 1716:1991.

Job

Effective wet stripping of asbestos. Power tool use on asbestos cement and similar products; clearance monitoring or entry into asbestos removal work area. Moderately dusty work.

Assigned Respirator Protection Factor

100 X Workplace Exposure Standard.

CLASS III

Type of Breathing Apparatus

Full facepiece airline positive-pressure respirator with a tight-fitting facepiece operated in continuous, or pressure demand, mode with filtered air. This class also includes positive-pressure self-contained breathing apparatus. The purity, quality and quantity of the air supply shall comply with Appendix A of AS 1715:1991. The air intake to the compressor must be sited in an uncontaminated atmosphere.

Devices must comply with NIOSH/MSHA, CEN or AS 1716:1991.

Job

Dry removal of asbestos, ineffective wet stripping. Asbestos work in confined spaces.

Assigned Respirator Protection Factor

1000 X Workplace Exposure Standard.

SYNTHETIC MINERAL FIBRES

The production and use of synthetic mineral fibres (SMF) – previously known as man-made mineral fibres – has increased considerably in the last 30-40 years. They are frequently employed as an asbestos substitute.

Fibre Types

The term "synthetic mineral fibre" (SMF) includes a range of fibres used in a variety of industries.

Rockwool and fibreglass are used in thermal and acoustic insulation.

Ceramic fibres are used in high temperature insulation such as furnaces.

Continuous filament is used as a reinforcement in various materials such as cement, textiles and paper products.

Special-purpose fibres are manufactured for specific purposes such as in the aerospace industry. These are often of a very small diameter and known as superfine glass fibres.

Fibre Characteristics

SMFs are amorphous in character - which is in contrast to naturally occurring fibres (such as asbestos), which are crystalline in structure. They therefore do not split longitudinally into fibrils of smaller diameter, but may break transversely into smaller segments.

However, those fibres that mimic the physical characteristics of asbestos fibres and are of considerable durability in the human body may prove in the future to give rise to similar health effects as asbestos.

Respiratory Effects Following the Inhalation of SMFs

The physical similarity between synthetic mineral fibres and asbestos has raised the question whether their inhalation might result in adverse health effects. This concern has stimulated considerable research in the last 15 years and was reflected in the terms of reference of the Asbestos Advisory Committee.

The respiratory effects of SMFs are considered in three groups:

IRRITATION

Those exposed to excessive amounts of dust at work, are more likely to develop symptoms of respiratory irritation than those who work in an environment in which no dust was produced.

If workers inhale SMFs at work, these could well cause respiratory irritation which would result in the development of cough and sputum.

FIBROTIC LUNG DISEASE

The Asbestos Advisory Committee noted that there was no epidemiological or animal evidence to suggest that fibrotic lung disease might result from exposure to SMFs.

LUNG CANCER

The Asbestos Advisory Committee noted the findings from two large epidemiological studies of over 40,000 production and maintenance workers who had been exposed to SMFs^{10,11}.

Workers who produced fibrous wool using rock or slag were reported in the United States study to have a 30% increase in respiratory cancer 20 years or more after first exposure¹⁰. In the European study, a 40% increase was reported¹¹.

Workers who produced fibrous wool from glass were reported as having a much smaller increase in respiratory cancer - about a 10% increase in each study.

There was no excess of respiratory cancer reported among the small group of workers who produced glass filament.

Doll, in a review of these studies, concluded that there had been an occupational hazard of lung cancer among workers who produced rock/slag wool and that there may have been a hazard among workers producing glass wool¹². He agreed with previous researchers that, given the reported low levels of exposure, a greater excess of lung cancer would not be observed even if the hazard being dealt with were asbestos, and suggested that perhaps the exposure estimates were too low.

Animal experiments have indicated that on a fibre-to-fibre basis, SMFs are less carcinogenic than asbestos¹³. The explanation for this appears to be that, on average, SMFs are less durable than asbestos and, as mentioned above, tend to break across the fibre rather than longitudinally.

It has been suggested that the explanation for the excess of cancer reported among the workers producing SMFs from rock/slag wool might be due to contaminants in the feed stock, furnace fumes or past exposure to asbestos¹³.

In the absence of any detailed dose-response relationships, the lung cancer risks associated with various types of exposure cannot be estimated. However, current exposures to mean levels of 0.2 respirable fibres or less per ml of air seem unlikely to cause any detectable excess in lung cancer rates¹².

A Workplace Standard for Synthetic Mineral Fibres

It is prudent to act on the assumption that exposure to SMFs may increase the risk of lung cancer among the workforce. OSH considers that on the basis of present knowledge, the following Workplace Exposure Standard, when implemented, would provide adequate protection for workers.

For synthetic mineral fibres:

1 fibre/ml of air (time-weighted average) and 5mg/m³ inspirable dust.

OCCUPATIONAL SAFETY AND HEALTH SERVICE (OSH)

The Occupational Safety and Health Service of the Department of Labour is charged with the responsibility of providing information and management advice to people at work and the public at large on such matters as asbestos and other occupational hazards.

The local branches of OSH are listed in Appendix 2 (pp.15-16) and can be contacted if you have problems with hazards at work including asbestos. If the branch is unable to help, they will refer the problem to OSH's experts who will provide advice.

If you have specific enquiries concerning asbestos or the operation of the asbestos registers write to:

The Registrar,
Asbestos Disease and Exposure Registers
P.O.Box 3705
Wellington

CONCLUSIONS

All asbestos fibre types may produce asbestosis and lung cancer. The greatest risk of mesothelioma is associated with exposure to crocidolite and with a lesser risk from exposure to amosite and chrysotile.

All asbestos-related disease appears to follow a dose-response relationship. It is the view of the National Asbestos Medical Panel that there is a threshold of fibre burden in the lung for the development of cancer. In establishing the diagnosis, it is essential that there is a complete history, including a full occupational history which provides details of exposure to asbestos.

In the light of present knowledge, the present New Zealand asbestos Workplace Exposure Standard is appropriate to prevent asbestos-related disease in those entering an industry where they will suffer exposure for the first time.

An Asbestos Exposure Register has been established for workers who have been exposed to asbestos in the past.

A National Asbestos Medical Panel and a National Asbestos Radiological Panel have been established to aid in the diagnosis of and determining the incidence of asbestos-related conditions and diseases.

The Occupational Safety and Health Service of the Department of Labour (OSH) is the organisation responsible for establishing, maintaining, and funding both the register and the panels.

The medical examination procedures stated in the Asbestos Regulations 1983 provide adequate surveillance for workers exposed to asbestos.

It would be prudent to act on the assumption that exposure to synthetic mineral fibres (SMFs) may increase the risk of lung cancer among the workforce.

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APPENDIX 1: ASBESTOS LEVELS ASSOCIATED WITH TYPICAL MATERIALS AND ACTIVITIES

Respirable dust testing carried out in New Zealand during the past 10 years allows some generalised comments to be made on dust levels normally found in common industrial situations. The figures in the table below are for **guidance only**. It is important to realise that abnormal conditions may lead to higher (or lower) levels than those indicated.

In the table > means "more than", < means "less than". All figures are expressed as respirable fibre per millilitre of air (f/ml).

NOTE: BECAUSE THE LEVEL OF FIBRE IN AIR CANNOT BE ACCURATELY ASSESSED IN EACH CASE, AND DIFFERENCES IN OPERATION MAY LEAD TO HIGHER LEVELS THAN QUOTED, THE LEVEL OF RESPIRATORY PROTECTION SHOULD ALWAYS BE ASSESSED ON THE HIGH SIDE OR WORST CASE SCENARIO.

	<i>Typical value</i>	<i>Extremes likely to be encountered</i>
Insulation		
Removal of moulded laggings	< 2	0-10
Chrysotile millboard, cutting, etc	1-2	0-20
Handling asbestos cloth	<1	0-2
Handling asbestos string	< 2	0-2
Removal of woven laggings	< 2	0-10
Sprayed amosite, removal wet	5-20	up to 100**
Sprayed amosite, removal dry	-	up to 300
Sprayed crocidolite, removal dry	5-20	up to 100**
Sprayed chrysotile, removal wet	5-20	up to 100**
Sprayed chrysotile, removal dry	-	probably 100
Stripping asbestos-covered wire etc.	<2	-
Cutting/sawing amosite-bearing insulation (Marinite, etc.)	0-2*	100
Asbestos cement products		
Cutting etc. dry (power tools)	0-2*	up to 20
Cutting etc. wet (power tools)	<1	up to 10
Construction work (outside)	<1	up to 10
Cutting A/C with hand tools	<1	1
Ambient air below sprayed insulation		
Chrysotile, amosite	usually 0.1	0.1
Crocidolite	usually 0.1	occasionally 0.2-1
Friction products		
Cutting, finishing, radius grinding etc.	normally 1	0-10
Changing filter bags	10	100
Handling friction materials (pads etc.)	<0.5	2
Dry sweeping	0-2	-
Handling raw asbestos		
Chrysotile, amosite	2 with care*	
General		
Handling talc (may contain minor tremolite)	< 2	-
Cutting gaskets	<2	-
Cutting greenstone (associated with tremolite)	<2	-
Handling/quarrying serpentine (with minor chrysotile)	<2	possibly up to 100 if conditions very dusty

* Assumes some form of extraction equipment

** To achieve low levels, extraction equipment in the room and good work practices will be required unless the insulation is well cemented