Asbestos and other Occupational Lung Diseases in New Zealand: 2012 Annual Report

1. ASBESTOS CEMENT PRODUCTS
2. TEXTURED COATINGS
3. FLOOR TILES, TEXTILES AND COMPOSITES
4. SPRAYED COATINGS ON WALLS, BEAMS/COLUMNS
5. ASBESTOS INSULATED BOARD
6. LAGGING
7. LOOSE ASBESTOS IN CEILING OR FLOOR CAVITY
Ministry of Business, Innovation and Employment (MBIE)
Hīkina – Whakatutuki Lifting to make successful

MBIE develops and delivers policy, services, advice and regulation to support economic growth and the prosperity and wellbeing of New Zealanders.

MBIE combines the former Ministries of Economic Development, Science + Innovation, and the Departments of Labour, and Building and Housing.

Front cover picture: Typical locations for the most common asbestos materials inside residential buildings.
Source: Health and Safety Executive: Asbestos kills: Protect yourself! Reproduced under the terms of the Click-Use Licence.

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Introduction

The Ministry of Business, Innovation and Employment’s two Asbestos Registers - the Disease Register and the Exposure Register - have been in existence since 1992. When established, they had the following objectives:

1. to raise awareness of asbestos-related disease nationally
2. to improve the radiological diagnosis of asbestos disease by using the ILO reference radiographs
3. to confirm that pleural plaques is a disease and not just a marker for asbestos exposure
4. to reinforce the importance of patient support groups.

Since these four objectives were defined, technological developments have seen HRCT (High Resolution Computed Tomography) superseding ILO X-ray classification.

The legacy of disease from working with and exposure to asbestos in New Zealand is continuing. Exposure is now largely confined to home renovators, demolition workers, carpenters, electrical and plumbing workers, building maintenance workers and asbestos removal workers.

The demolition, rebuild and renovations arising from the 2010-2011 Christchurch earthquakes have again brought asbestos exposure in the construction industry to the fore.

Worldwide, asbestos exposure still occurs at a high level; and a number of lessons can be learned as a result.

Firstly, there continues to be a general lack of awareness of the effects of inhaling dust at work, regardless of the type of dust. Secondly, too great an emphasis has been put on the nature of the dust and whether or not dust is a danger to health. Views that cement dust is safe and silica dust is unsafe; or blue asbestos is worse than white asbestos have distracted users from the real issue; that is, the need to remove all dust from workplace air.

Today, dust is recognised as a major problem in the workplace, both in terms of respiratory health and general health. While its solutions are not complex, they do involve identifying the source of the dust and containing it. At a personal level, there is also the need to provide comfortable and effective, fit-tested respiratory protection.

With a gradual reduction in cigarette smoking among the working population, the impact of dirty workplace air on respiratory health is likely to become clearer, with diseases such as welder’s lung, asthma, and chronic obstructive pulmonary disease (COPD) recognised more clearly for their relationship to work practices and procedures.

Dr W. I. Glass (Convenor)
Dr R. Armstrong
Dr D. Jones

National Asbestos Medical Panel
September 2013
Summary of asbestos-related changes

Compared with the previous year’s annual report:

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesothelioma</td>
<td>227</td>
<td>232</td>
<td>5</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>119</td>
<td>124</td>
<td>5</td>
</tr>
<tr>
<td>Asbestosis</td>
<td>285</td>
<td>294</td>
<td>9</td>
</tr>
<tr>
<td>Pleural Abnormalities</td>
<td>615</td>
<td>649</td>
<td>34</td>
</tr>
<tr>
<td>Total</td>
<td>1246</td>
<td>1299</td>
<td>53</td>
</tr>
</tbody>
</table>

Table 1: Categories of notified occupational lung diseases

<table>
<thead>
<tr>
<th>Year</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>2003</td>
<td>81</td>
</tr>
<tr>
<td>2004</td>
<td>84</td>
</tr>
<tr>
<td>2005</td>
<td>103</td>
</tr>
<tr>
<td>2006</td>
<td>99</td>
</tr>
<tr>
<td>2007</td>
<td>91</td>
</tr>
<tr>
<td>2008</td>
<td>99</td>
</tr>
<tr>
<td>2009</td>
<td>91</td>
</tr>
<tr>
<td>2010</td>
<td>90</td>
</tr>
</tbody>
</table>

Table 2: National cancer figures for mesothelioma
Part 1: Asbestos

What is asbestos?
Asbestos is a term used to describe naturally occurring fibrous hydrated silicates of which there are six common varieties.

Figure 1 shows the different types of asbestos.

![Asbestos Types Diagram](image)

**Figure 1: Types of asbestos**

Amosite (brown asbestos) and crocidolite (blue asbestos) have straight needle-like fibres which naturally split in their long axis, producing very fine fibrils (thin fibres). In contrast, chrysotile (white asbestos) has softer curlier hair-like fibres, which makes them useful for weaving, and they have been used to make asbestos cloth for centuries.

When these fibres are inhaled, they behave in different ways in the lungs.

Chrysotile (magnesium silicate) fibres slowly lose magnesium, which leaches into body fluids, making the fibre more easily digested by scavenger cells (macrophages). This reduces the body burden of the asbestos dust, and has led to the view that white fibres are less harmful than blue or brown fibres.

Crocidolite (iron-sodium silicate) fibres are straight and rigid and can split longitudinally. They are more resistant to body fluids and can survive unchanged in the body for up to 40 years or more. The fine fibrils can migrate through the lung tissue to the lung lining (pleura).
Amosite (iron magnesium silicate) fibres are longer, making it useful for insulation. Again, the fibres remain unchanged in the body for years.

**Asbestos awareness in New Zealand**

1938

The introduction to the *Report of the Interdepartmental Committee on Silicosis* refers to asbestos as follows:

“In the working of asbestos the dust produced gives rise to a pulmonary condition known as asbestosis, which, although different in its character from silicosis, causes a disease similar in some respects to it. Free silica is not associated with asbestos, yet the fibres produce a deadly pulmonary disease.”

1951

In the *Annual Report of the Department of Health*, Dr Garland, Director of the Division of Occupational Health, notes:

“*Asbestos is now being quarried in the Dominion, and the dust can be expected to cause a certain amount of lung damage unless proper precautions are taken.*”

1953

Notification of occupational diseases introduced. No specific identification of asbestos-related lung disease.

1960

*Occupational Health Notes on Diseases Arising from Occupation* published by the Department of Health:

“The fibrosis of asbestos is diffuse ... there is some statistical evidence for an increased incidence of bronchogenic carcinoma.”

1964

A New Zealand standard is set for asbestos fibres in air.

1968

Dr Copplestone, Assistant Director (Occupational Health) initiates a New Zealand asbestos survey.

1971

Dr Allingham, who succeeded Dr Copplestone, reports on the survey:

“207 workers surveyed. 101 workers x-rayed. 17 showed pleural changes (one case later confirmed as asbestosis).”

1977

Environmental Dust Laboratory established to identify asbestos fibres in air.

1978

Asbestos Regulations promulgated.

1981

The allowable concentration for asbestos fibres (other than crocidolite fibres) is reduced from 2 fibres/ml to 1 fibre/ml. Crocidolite remains at 0.2 fibres/ml over a 10-minute sampling period.

1982

Jim Butterworth, Auckland Secretary, New Zealand Engineers Union, expresses concern about the long-term effects of asbestos on workers at James Hardie factory.
1983

Interim Report of Workers at James Hardie (unpublished) T. Kjellstrom, F. Rennie. Of 353 workers x-rayed, 53 showed parenchymal (functional) and/or pleural changes. An increasing percentage of abnormalities found over time since first exposure.

Asbestos Regulations replace 1978 regulations.

A New Zealand Gazette notice reduces the air standard for crocidolite to 0.1 fibre/ml over 4 hours.

1984

Relative Cancer Risks from Exposure to Different Asbestos Fibre Types. J. Keir Howard, NZ Medical Journal 97; 646-9. In this review article, the point is made that:

"...considerable amounts of crocidolite were used in the past, particularly in insulation, and as New Zealand did not restrict the use of the amphiboles until well after most industrialised nations had done so, the use of blue asbestos continued for longer in this country."

Importation of blue and brown asbestos is banned in New Zealand.

1987

Asbestos Regulations 1983, Amendment No. 1, tightens provisions for medical examinations.

1988

Thelma Bell, widow of asbestosis victim Clarrie, organises a public meeting in Christchurch.

Barry Brown, Secretary of the South Island Labourers Union, arranges for Maevis Watson, Occupational Health Nurse, and Associate Professor Bill Glass from the Otago Medical School, to interview ex-Fletcher workers from the Mandeville Street plant.

Cate Brett – Christchurch Star – highlights the tragedy of asbestos use.

1989

Robin McKenzie, Engineer with NZED (New Zealand Electricity Department), is diagnosed as suffering from mesothelioma. In 1990, his lawyer took legal action against the Crown. Media coverage keeps up public concern.

1990

Review of Fletcher’s Mandeville Street Factory (unpublished). W.I. Glass. 103 workers contacted. 87 investigated. 47 cases of asbestos-related conditions (plus 7 probable cases). 12 deaths to date.

Hon. Bill Birch, Minister of Labour, establishes the Asbestos Advisory Committee in October to report on all aspects of asbestos exposure in New Zealand.

1991


“Case control studies based on New Zealand Cancer Registry showed that asbestos-related occupations were found to be associated with elevated risks of cancer in the lung, pleura and peritoneum. The risks of cancer in these
three sites were highest among the group comprising machinery fitters, plumbers, welders, boilermakers, metal moulders, metal polishers and electricians."

*Report of the Asbestos Advisory Committee* presented to the Minister in April. The Minister decides to implement the recommendations following Cabinet approval in August.

Tim Frederiksen establishes the Asbestos Victim and Support Group, which later became the Asbestos Disease Association of New Zealand.

1992

In March, a National Asbestos Register is established (recommendation 4 of the Report). This Register was to be in two parts: Part 1 for those who had been exposed, and Part 2 for those suffering from an asbestos-related disease. The Occupational Safety and Health Service of the Department of Labour is appointed to administer the registers.

ECNZ (Electricity Corporation of New Zealand) combines with the Department of Labour to publicise asbestos hazards.

An audit of floor sanders and work practices involving asbestos-backed vinyl sheeting in the Christchurch area is carried out by K.D. Sheat and published by the Department of Labour.

ACC lump sum entitlements for asbestos-related claims cease.

1993

The first Annual Report of the National Asbestos Registers confirms 199 cases of asbestos-related disease.

(March) Rights to initiate common-law claims in regard to asbestos-related conditions cease.

ECNZ establishes model asbestos surveillance programme of current and past employees.

Secondary cases of asbestos disease are found to occur in family members of asbestos workers. Their only exposure was to dust brought to the home from the workplace.

1994

The second Annual Report reviews and confirms a total of 462 cases of asbestos-related diseases.

Visit by Professor Margaret Becklake, International Asbestos Authority, to advise on the registers.

1995

*Asbestos Exposure and Disease: Notes for Medical Practitioners* is published by the Department of Labour.

1995/96

The third Annual Report confirms 535 cases.

1996

*The Management of Asbestos in the Non-Occupational Environment* is published by the Ministry of Health.
1996/97 The fourth Annual Report of the National Asbestos Register notes an increase in occurrence of mesothelioma cases.

1997 The Asbestos Medical Panel publishes its first report, based on a review of asbestos cement-exposed workers on the Exposure Register. 2257 notifications are reviewed. A positive finding is a non-specific association between cumulative asbestos exposure and a questionnaire diagnosis of asthma.


1999 Visit by Professor Corbett McDonald and Professor Alison McDonald to advise on updated information on mesothelioma.


2000 Mesothelioma in New Zealand, the second study by the National Asbestos Medical Panel, is published.


Lung Function Changes in Asbestos-Exposed Workers with Pleural Plaques, the third study by the National Asbestos Medical Panel, is published.


Occupational Safety and Health (Department of Labour) publishes the Review of Guidelines for the Management and Removal of Asbestos.


2010 Ed Grootogoed, Chairman of the Asbestos Diseases Association of New Zealand (Inc), passed away in May. Ed did much to highlight the asbestos tragedy in New Zealand, in particularly his concern at the dumping of asbestos and rebuilding on old dump sites.
2011

Professor Ken Takahashi from the University of Occupational and Environmental Health, Kitakyushu City, in Japan, visits New Zealand for the Federal ANZSOM conference in Wellington and spoke on *Global Mesothelioma Deaths Reported to the World Health Organisation between 1994 and 2008.*


2012

Dr Michael Donoghue, Medical Director of ALCOA World Alumina, is a guest speaker at the Update on Occupational Respiratory Disease meeting. He spoke on *Occupational Asthma in the Aluminium Smelters of Australia and New Zealand: 1991-2006.*

Important developments

The 2010 and 2011 Canterbury earthquakes

On Saturday 4th September 2010, a 7.1 magnitude earthquake struck the South Island, 40 kilometres west of Christchurch. Some buildings in Christchurch and surrounding areas were badly damaged, and a small number of people were injured.

On Tuesday 22nd February 2011, a 6.3 magnitude earthquake struck 10 kilometres south-east of Christchurch. It caused widespread damage across Christchurch, particularly the central city and eastern suburbs. The earthquake occurred at 12.51pm, a time when many people were working, having lunch or shopping in the city. Two buildings completely collapsed, and 185 people were killed.

Another 6.3 magnitude earthquake occurred on 13 June 2011, and affected buildings already damaged by the previous earthquakes. 46 people were injured.

The devastation wrought by these earthquakes and multiple aftershocks has given rise to the extraordinary increase in demolition and rebuilding that is still continuing strongly. Occupations most at risk of exposure to asbestos are demolition workers and people involved in building renovation.

The Ministry of Business, Innovation and Employment is actively engaged in ensuring exposure risks are minimised and is monitoring reporting of restricted work and the asbestos exposure register. These are two key indicators which the Ministry uses to inform its operational programmes.

Asbestos Exposure Notifications in Christchurch

The Asbestos Exposure Register exists to allow individuals who have been exposed to asbestos to register with the Exposure Register. While similar to the Asbestos Disease Register, the decision to register is an individual and voluntary decision. Nevertheless, trends in exposure registrations can indicate the presence of possible exposure risks.

A review of the exposure registrations for Christchurch for 2010, 2011 and 2012 indicated that demolition was causing some concern among the workforce.
Notifications to the Ministry of Business, Innovation and Employment (MBIE) (formerly the Department of Labour) of restricted work involving asbestos from 1 July 2012 to May 2013 were:

<table>
<thead>
<tr>
<th>Region</th>
<th>Number of notifications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Auckland</td>
<td>48 (approx.)</td>
</tr>
<tr>
<td>Wellington</td>
<td>40 (approx.)</td>
</tr>
<tr>
<td>Christchurch</td>
<td>560</td>
</tr>
</tbody>
</table>

Visit to the Asbestos Diseases Research Institute (ADRI), Sydney, Australia, January 2013

Dr Glass visited the Institute, meeting with the Director, Professor Nico van Zandwijk and other senior staff. Three important outcomes arose from this visit.

The first was an offer for New Zealand to join with Australia in a future annual Asbestos Awareness Week. The 2012 end-of-year awareness week in Australia focussed on the renovator – not just the male home renovator but also the wife, as a possible reason why there had been an increase in mesothelioma occurrence among women. With the renovation and repair of Christchurch homes arising from the Canterbury earthquakes, awareness of this issue is critical.

The second outcome was to learn of the research into mesothelioma by the Institute. In particular, the role of microRNAs and their impact in the regulation of gene expression and control of many normal cellular processes and disease states. Dr Glen Reid, who is leading this research, noted in the Annual Report:

“We have identified a number of MIRNAs that are over- or under-represented in mesothelioma cell lines and tumours. We are currently investigating the role of these microRNAs and we are investigating if we can exploit them as a novel target for treatment.”

The third outcome was the May 2013 visit of Dr Matthew Soeberg, a postdoctoral research fellow from Wellington, now working with the ADRI and focussing on the mesothelioma epidemic in Australia.

Update on Occupational Respiratory Disease, Wellington, June 2012

This one-day seminar celebrated 20 years of the Asbestos Disease Register and Asbestos Exposure Register. These two registers were established by the then-Department of Labour at the instigation of the Minister of Labour, Sir William (Bill) Birch, following the Report of the Asbestos Advisory Committee in April 1991.

The seminar acknowledged the role of the medical panel in keeping asbestos at the forefront of cancer-causing agents in the New Zealand workplace.
The programme covered a wide range of topics, which included monitoring isocyanate exposure, the increasing relevance of chronic obstructive pulmonary disease (COPD) in the workplace, radiology as a diagnostic tool, lung function testing in the workplace, enzyme exposure and the impact of the Canterbury earthquakes on occupational respiratory disease exposure to asbestos and silica.

The organisers acknowledged the generosity of Alcoa World Alumina Australia in sponsoring Dr Michael Donoghue, their international Director of Health and Chief Medical Officer, who gave the keynote address on smelter asthma.

**Asbestos Certificate of Competence Assessor Training**

Asbestos removal approval and supervision continues to involve a considerable amount of MBIE’s health and safety inspectorate time. As noted earlier, there was a marked upsurge in asbestos notifications following the Canterbury earthquakes of 2010 and 2011.

As part of the on-going up-skilling of the health and safety inspectorate, a three-day training programme was organised and presented in Christchurch in October 2012. The programme included the health effects of asbestos, sampling and testing, the purpose of the Asbestos Registers, construction site management requirements, the selection and use of PPE (personal protective equipment), a site visit and health monitoring of both the inspectors conducting assessments and the asbestos removal workers.

**Measuring silica-containing dust exposure of workers cutting/drilling linear board, a fibre-cement product**

This investigation was carried out by Kerry Cheung of the Centre for Public Health Research at Massey University, and Gerry Kalogeropoulos, Health and Safety Inspector for MBIE.

The method used was a real-time video exposure monitoring system which visually recorded the task being undertaken by the worker, at the same time recording on screen the dust levels as they varied with the task activity.

Recorded levels of the mixed fibre-cement dust indicated peaks of up to 120mg/m³ when using a skill saw, and an even higher peak when angle grinding. No New Zealand Workplace Exposure Standards exist for short term exposure levels (STELs) for this product, and the intermittent nature of the work activity was such that only an estimate of the TLV-TWA was possible, based on inhalable rather than respirable dust.

Nevertheless, the high peaks indicated the need for greater awareness of this hazard, the use of attached exhaust systems to tools, wearing effective fit-tested respiratory protection and systematically monitoring workers’ respiratory function when cutting or drilling this material.
Part 2: Review of asbestos-related disease notifications

Summary
This report reviews 1299 cases that were notified to the National Asbestos Medical Panel between March 1992 and July 2012. They include:

- 232 cases of mesothelioma
- 124 cases of lung cancer
- 294 cases of asbestosis
- 649 cases of pleural abnormalities.

The number of lung cancer cases reported is roughly half of the number of mesothelioma cases. This suggests that the taking of a lung cancer history is still dominated by the smoking factor, and that occupational factors are downplayed.

Repairing or renovating hundreds of earthquake-damaged properties is a major task for residential and commercial building owners. With the “housing stock” in Christchurch older than elsewhere in New Zealand, asbestos exposure for renovators is an important issue.

Mesothelioma notifications remain high, reflecting exposure in the 1960s and 1970s, and this trend is likely to continue for some years to come.
Results

Figure 2 shows the distribution of the four main diagnostic categories:

- lung cancer
- pleural disease
- asbestosis
- mesothelioma.

![Pie chart showing disease categories](image)

**Figure 2: Categories of disease 1992-2012**

What is noticeable is that pleural disease is the main category, with lung cancer clearly under-represented when compared with mesothelioma.
Figure 3 looks at occupations for the total number of notified asbestos disease cases during the period.

![Figure 3: Notified asbestos disease by occupation 1992-2012](image)

It is clear that carpenters, plumbers and electricians are together responsible for 67% of all cases. These ‘all purpose’ construction workers are an occupational category at risk particularly because, unlike asbestos cement workers, they are not always seen as being at obvious risk.

The non-occupational category refers to cases where an individual’s exposure was not work-related. This includes all cases resulting from secondary or environmental exposure, such as children brought up in the home of an asbestos worker, and people who washed asbestos-contaminated clothes. The ‘no known exposure’ category refers predominantly to mesothelioma cases where conclusive exposure histories were not available.

(Note: the time delay is often 40-50 years from exposure and has either been forgotten, never recognised, or not known by the surviving family member).
Mesothelioma

Figure 4: Distribution of mesothelioma by occupation

Mesothelioma - a rare cancer of the pleural membranes on the surface of the lungs - is strongly related to asbestos exposure. The panel reviewed 232 cases of mesothelioma, of which:

- 223 were Caucasian
- 6 were Maori
- 3 were identified as ‘Other’
- 217 were males, 15 were females
- the mean age at diagnosis was 67 years (range 35-85)
- the mean number of years since first exposure was 45 (range 12-74)
- the mean exposure index was 178 (range 1-780)
- there were 21 current smokers, 127 ex-smokers and 61 never-smokers (information for 23 cases was unavailable).

Asbestos processors, plumbers/fitters/laggers, and carpenters/builders, accounted for over 60% of all registered cases.
Over the period from 1954 to 2010, a total of 1,618 cases of mesothelioma have been registered. Figure 5 shows that the total number of cases continued to rise and in 2005 it exceeded 100 for the first time. However, the cases have remained in the 90s for the last two years, which equates to approximately two cases each week.

Mesothelioma is very much a disease of old age as Table 3 illustrates, with 49% of cases occurring to people aged 70 or over.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age group</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt; 50</td>
<td>50s</td>
</tr>
<tr>
<td>Female</td>
<td>13</td>
<td>26</td>
</tr>
<tr>
<td>Male</td>
<td>18</td>
<td>136</td>
</tr>
<tr>
<td>Total</td>
<td>31</td>
<td>162</td>
</tr>
</tbody>
</table>

Table 3: Mesothelioma occurrence by age range 1994-2010

Of the 1191 cases, 165 occurred to women and 1026 to men. As women are seldom employed directly in the asbestos-exposed workplaces, their exposure could be as a result of “secondary” exposure to dust brought home from work on the hair and clothes of family members.
Lung cancer

Figure 6: Distribution of lung cancer by occupation

Lung cancer is a cancer of some of the cells in parts of the lung, usually beginning in the lining of the airway.

The panel reviewed a total of 124 cases of lung cancer, of which:
- 118 were Caucasian
- 4 were Maori
- 1 was from a Pacific Island
- 1 was identified as ‘Other’
- 122 were males, 2 were females
- the mean age at diagnosis was 69 (range 42-86)
- the mean number of years since first exposure was 46 (range 17-63)
- the mean exposure index was 165 (range 12-565)
- there were 28 current smokers, 85 ex-smokers, 8 never smokers and 3 unknown.

Lung cancers are classified according to the type of cell affected. Histological classification revealed 55 cases of squamous cell carcinoma, 30 adeno, 15 small cell, 4 undifferentiated, 1 bronchiolar-alveolar, 4 large cell and 15 cases where classification was not stated. In addition, the tumour sites were as follows: 46 upper lobe, 46 lower lobe, 11 middle lobe, and 21 not stated.

The occupational distribution of lung cancer follows a similar pattern to both pleural plaques and asbestosis, in that plumbers, fitters, carpenters and asbestos processors account for most of the cases.
Asbestosis

Figure 7: Distribution of asbestosis notifications by occupation

Asbestosis is a fibrotic or scarring disease of the lung tissue. The disease develops slowly over many years from initial exposure. It can continue to develop after exposure to asbestos has ceased.

The panel reviewed a total of 294 cases of asbestosis, of which:

- 290 were Caucasian
- 2 were Maori
- 2 were from a Pacific Island
- 278 were males, 16 were females
- the mean age at diagnosis was 68 (range 37-86)
- the mean number of years since first exposure was 43 (range 15-71)
- the mean exposure index was 180 (range 10-720)
- there were 22 current smokers, 210 ex-smokers and 57 never smokers (accurate smoking histories were not available in 5 cases)
- radiological changes showed 175 cases with pleural plaques and/or pleural thickening.
Of the 294 asbestosis cases, 150 were categorised by ILO classification, and others were categorised on the basis of CT, HRCT or pathology, where available. With the recent trend to use HRCT, categorisation by ILO classification is less frequently done.

**Definition of asbestosis used in the register**

An important issue with this disease is ‘what criteria constitute a diagnosis of asbestosis?’ The main point of discussion is the difference between a clinical diagnosis of asbestosis, and a diagnosis suitable for use in a national database where the inclusion of patients with early disease is desirable.

The definition of Gilson\(^1\) in his review of asbestosis-related lung conditions in the ILO encyclopaedia has been chosen by the panel and is as follows:

(a) a history of significant exposure to asbestos dust rarely starting less than 10 years before examination  
(b) radiological features consistent with basal fibrosis (1/0 and above, ILO, 1980)  
(c) characteristic bilateral crepitations, and  
(d) lung function changes consistent with at least some features of the restrictive syndrome.

*Gilson* notes that not all criteria need to be met in all cases, but that (a) is essential, and (b) should be given greater weight than (c) or (d). However, occasionally (c) may be the sole sign. Further, he notes that although the restrictive syndrome is the most common pattern (about 40%), in about 10% of cases airway obstruction is the main feature, while in the remainder a mixed pattern is seen.
Of the 294 asbestosis cases:

- all had a significant exposure history, with a mean exposure index of 180 (range 10-720)
- mean latency was 42 years, with a range of 15-71 years
- most cases had an ILO rating 1/1 or greater where this rating was used, although this criterion is now seldom used
- detailed clinical examination results were not always available from the records, thus the presence of crackles was not measurable
- lung function changes are recorded in the register based on the availability of data either from respiratory laboratories, respiratory physicians, or occupational health nurses
- additional information from HRCT scanning has led to the recognition of some cases of asbestosis not covered by the Gilson criteria. As noted, HRCT diagnosis is now the norm.

**Pleural abnormalities**

Pleural abnormalities include pleural plaques, diffuse pleural thickening, chronic fibrosing pleuritis and pleural effusions. It does not include pleural disease occurring together with mesothelioma, lung cancer or asbestosis.

![Figure 9: Distribution of pleural abnormality notifications by occupation](image-url)
Of the 649 cases reviewed:
- 637 were Caucasian
- 8 were Maori
- 4 were from Pacific Islands
- all but 10 were males
- the mean exposure index was 162 (range 6-708)
- there were 62 smokers, 386 ex-smokers and 166 never smokers (accurate smoking histories were not available in 35 cases).

Discussion

Information recorded in the Disease Register under-estimates the total burden of asbestos-related disease in New Zealand. This is a consequence of the voluntary nature of the Register, lack of understanding of work as a factor in disease causation by the medical profession, and failure by the Cancer Registry to code occupation in their database.

However, the Register continues to serve an important purpose. There is now greater awareness of the work factor in disease than in 1992 when the Register was established. MBIE now has greater commitment to the importance of occupational illnesses; the Accident Compensation Corporation employs a greater number of occupational doctors, and there are increasing numbers of occupational nurses and safety officers in the private sector.

The Registers, part of the wider Notifiable Occupational Disease System (NODS) operated by MBIE have, in the view of the medical panel, played an important part in encouraging these developments.

Pleural plaques

One of the aims of the medical panel was to confirm the view that pleural plaques were not just a marker of exposure, but represented a disease state. The then-Department of Labour publication *Lung Function Changes in Asbestos Exposed Workers with Pleural Abnormalities* in 2000 indicated a clear dose-response pattern, including a reduction of FVC and FEV1 with increasing asbestos exposure, independent of smoking habit.

Reference has been made to the impact of an asbestos-related occupation on the health of a worker’s partner and children. Two cases of pleural plaques notified in the last few years illustrate two women developing widespread plaques in their early 70s. Their only exposure was to asbestos dust brought home on their husbands’ clothes which they washed. In one case the husband was an asbestos sprayer; in the other, a carpenter.

Asbestosis

The increasing use of HRCT has resulted in the identification of minor degrees of asbestosis often with few, if any, symptoms and no disability. It is possible that these individuals will have a better long-term outlook, although this is not yet established.

Lung cancer

The contribution of occupational asbestos exposure to the causation of lung cancer is well recognised as being under-estimated, and over-attributed to smoking among workers exposed to asbestos. One approach to this issue is to determine the ratio between mesothelioma and lung cancer on the grounds that most mesotheliomas are diagnosed and the majority are regarded as being caused by asbestos exposure at work. Various estimates of such a ratio have been suggested with a range from two to 10. Even if the lower ratio of
1:2 is taken – based on the mesothelioma cases diagnosed over 1994-2010, for example – some 2,382 cases of lung cancer due to asbestos exposure would have occurred, or approximately 148 a year. It is likely that this figure could be even higher.

**Mesothelioma**

Reported cases of mesothelioma have continued to rise in New Zealand over the past decade as shown in Figure 5, and based on the New Zealand Cancer Registry. It is of interest to note the mean exposure index for mesothelioma of 152 - as recorded by the panel - is not dissimilar to exposure indices for pleural plaques (162), lung cancer (162) and asbestosis (180). In other words, mesothelioma, like other asbestos-related conditions, is in general dose-dependent.

**Chronic obstruction pulmonary diseases (COPD) and asbestos exposure**

These conditions are now being recorded if present in individuals with an asbestos-related disease, as well as in those asbestos-exposed workers who have no confirmed asbestos-related lung or pleural disease. Over the past years 33% of reported cases of asbestos-related disease also had COPD, 40% among cases of pleural plaques, 45% among asbestosis cases, 80% among lung cancer cases and 0% among cases of mesothelioma. In addition, 16 cases that were referred to the Panel because of asbestos exposure but without classical asbestos-related conditions had COPD.

**Other asbestos issues**

**Building maintenance workers**

Great Britain’s Health and Safety Executive (HSE) has published two related guidance booklets. The first, *Introduction to Asbestos Essentials* (2001), is specifically aimed at building maintenance workers.

The second, *Asbestos Essentials Task Manual* (2008), is aimed at any worker who may come into contact with asbestos in the course of their work. Such workers include electricians, plumbers, computer installers, telecommunication engineers and others.

**Demolition workers**

The process of demolition is often carried out over a weekend, utilising casual labour. The presence of asbestos is not necessarily determined prior to a demolition, and as a consequence there may be no knowledge of exposure.

**Asbestos removal workers**

While the New Zealand Demolition and Asbestos Association, in association with the then-Department of Labour published *Guidelines for the Management and Removal of Asbestos* (2011), undoubtedly a proportion of asbestos removal workers would have been exposed to significant amounts of asbestos for short- or long-term periods, and some would have transported asbestos dust from work to home on their clothes, boots or body. The National Exposure Register does not identify this group specifically, but they could be the group at most risk, and be a source of asbestos disease in the decades ahead.

**Floor sanders**

In 1992, an audit of floor sanders and their working practices was carried out in Christchurch. This audit revealed a number of features:

- a failure to prevent spread of dust to other rooms
• lack of a thorough ‘clean up’ after sanding
• poor respiratory protection for sanders
• sanders taking asbestos dust home on their work clothes
• transfer of dust from sanding machines to vehicles
• careless disposal of sanding dust
• a general lack of understanding about asbestos.

Overseas studies confirmed the New Zealand experience, with high levels of asbestos dust measured in rooms during the sanding process. With the renovation of earthquake damaged homes in Christchurch, floor sanding will continue to pose a health risk.

**Brake lining repair workers**

Some brake linings still contain asbestos. Such work is often carried out in designated ‘small workplaces’. As such they are less likely to be visited by Health and Safety Inspectors, and more likely to have inadequate local exhaust ventilation. The work is intermittent and the dose may be long-term and low-level in contrast to demolition workers (high-level and short-term). Nevertheless these workers comprise an on-going ‘at-risk group’.

**Protecting workers’ families**

In 2002 NIOSH (the National Institute for Occupational Safety and Health in the United States) produced a document with the above title to re-emphasise the importance of protecting workers’ families from the take-home exposure of hazardous substances.

The Asbestos and Respiratory Disease Medical Panel has kept details of such cases as part of the on-going review of asbestos-related disease.

Three cases of mesothelioma have been reported where exposure was brought into the home. In one case, the individual washed her younger brother’s work clothes. He was an apprentice in the railway workshops where asbestos was used in both pipe insulation in the steam engines and noise insulation in the carriages. It was common for the apprentice boys to have ‘snowball’ fights with the asbestos.

In the second case, the individual would visit her father’s place of work, an asbestos cement factory, and play with the asbestos, as well as being exposed to asbestos dust being brought home. The investigating physician’s report was graphic:

> “Her father worked with asbestos and she recalls as a child visiting his work on multiple occasions and playing with asbestos dust. She can remember being covered in asbestos and being exposed to clouds of asbestos which would no doubt have been inhaled”.

In the third case, the father was a house builder and the brother a lino layer – both occupations providing exposure to asbestos dust which was again brought back into the home.

No other source of asbestos was found in any of these cases.

There was one case of asbestosis. This occurred in a woman who, when young, washed her father’s clothes. He worked in an asbestos cement manufacturing plant.
The remaining cases were pleural plaques. Two were males who were exposed in childhood to asbestos dust brought into the home by their fathers, both of whom worked in at an asbestos cement plant. In one case, the father died of lung cancer as a consequence of his work exposure to asbestos. In the other case it is recorded that “it was not uncommon for the workers to take their children in [to work] on Saturday morning, which was their ‘clean-up day’ – probably one of the dustiest situations – where the children could make ‘snow balls’ with the asbestos”.

In an unusual situation, the home of a worker and family was situated across the road from an asbestos cement manufacturing plant. He died of lung cancer and his wife was found to have pleural plaques in an X-ray carried out in later life. Here, there was a possibility that the proximity of the home to the plant resulted in environmental contamination within the home and its surroundings, resulting in sufficient exposure over many years to account for the two conditions.

**Environmental exposure and asbestos-related disease**

**Erionite**

Erionite is a naturally-occurring fibrous mineral. It has been listed as a Group 1 carcinogen by IARC (the International Agency for Research on Cancer) and animal studies suggest that it has a greater carcinogenic potential than crocidolite (‘blue’) asbestos. It first came to attention among people who lived in three villages in the Cappadocian region of Central Anatolia in Turkey. In this situation, erionite was excavated from local volcanic tuffs to create storage areas, and also for use in construction materials. Investigation showed that malignant mesothelioma, localised and diffused pleural thickening, and interstitial fibrosis were significantly increased, compared with unexposed villages.

A 2011 study on erionite exposure in North Dakota, USA, among workers in gravel pits and road works, indicated that out of 35 workers investigated, seven had either bilateral pleural changes or interstitial lung changes. Complex assessment of these seven related in two having bilateral pleural changes, most likely resulting from environmental exposure to dust containing erionite in the course of their work.

Another study on environmental exposure occurred in New Caledonia³. In this situation, studies had shown that the soil contained serpentine.

A case control study on malignant mesothelioma was carried out as a result of the high incidence of malignant mesothelioma. 109 cases recorded in the cancer registry of New Caledonia between 1984 and 2008 were investigated. The study concluded that serpentine on roads was the greatest environmental risk factor (odds ratio 495.0; 95% CI, 46.2-4679.7).
Part 3: Occupational respiratory disease

There has been little emphasis on the importance of occupational asthma, silicosis and work-related chronic obstructive respiratory disease in New Zealand until recent years. However, there is a historical base to occupational respiratory disease as referenced below:

1938

Silicosis: Report of Interdepartmental Committee, Bulletin No 57, Department of Scientific and Industrial Research, Wellington. This report noted that since the 1915 Miners’ Phthisis Act, 1,576 miners had been granted pensions as a result of silicosis; of those, 1,508 were described as gold miners and 68 as coal miners.

1940s

A record of tuberculosis morbidity in hospital nurses recorded in the book Challenge for Health by Dr F.S. McLean showed a morbidity rate per 1,000 nursing staff which ranged from 8.9 to 20.3, averaging 16.5 over the years 1943-1949.

1961

The Grey Valley Survey in which Dr Francis de Hamel surveyed 1,524 miners and ex-miners in the coal industry during 1958 revealed only 32 cases of pneumoconiosis, not all of whom had worked in New Zealand coal mines.

The 1990s

This period saw a significant output of publications on non-asbestos occupational respiratory disease, largely due to Professor Neil Pearce of the Centre for Public Health Research at Massey University, together with Dr David Fishwick and Lisa Bradshaw. Over this period occupational asthma was investigated nationally among farmers, sawmill workers, aluminium workers; at the same time respiratory symptoms, lung function and dust levels were measured in the wood industry, plywood mill workers, hairdressers, mussel openers, welders and quarry workers.

2000

In 2000 Dr Jeroen Douwes joined the Centre, accelerating the interest in sawmill workers, wood dust and respiratory effects.

Occupational asthma

In the Colt Lecture given by Anthony Newman Taylor at the Ninth International Symposium on Inhaled Particles at Cambridge University in 2001, Professor Taylor noted some key points about this disease:

- asthma is the most prevalent cause in the United Kingdom, and probably in the western world, of respiratory ill health during working life
- asthma consists of variable airflow limitation, reversibility and hyper-responsiveness of the airways
- asthma can be induced by direct toxic damage to the lining of the airways (irritant inducers) or the RADS phenomenon, or as a result of a specific hypersensitivity response to inhaled proteins and low molecular-weight chemicals
- asthma can be incited in persons who already have hyper-responsive airways when exposed to pollutants in the workplace air
- evidence is strong that the risk of developing occupational asthma is determined more by the level of exposure to the risk factor than by individual susceptibility
• asthma induced by work exposures carries a high risk of progression to chronic asthma
• there is evidence that the extent of occupational asthma can be reduced by reducing the airborne concentration of the causative agents.

The current programme of MBIE to investigate dirty workplace air is a practical response to the accumulating evidence relating to the causes of work-related asthma.

In New Zealand, a debate frequently occurs regarding work-aggravated asthma (WAA), and work-induced asthma (occupational asthma), largely related to whether a compensation claim is accepted or not. While this debate may be relevant to the issue of compensation, it is clearly not relevant to reducing and minimising the impact of asthma at work. This is because both work-aggravated and work-induced asthma are a consequence of exposure to dirty workplace air, and the outcome of both types of asthma is seen in lost time from work. The possibility is that what begins as asthma may in time, if exposure continues, lead to a lack of reversibility and the development of chronic obstructive pulmonary disease.

In confirming a case of occupational asthma, the Panel requires the following:
• a diagnosis of asthma
• asthma occurring for the first time at work (work-induced asthma) or asthma made worse at work (work-aggravated asthma)
• a work history of exposure to an asthmagen or to a substance that can irritate the respiratory tract
• supportive peak flow records indicating a temporal relationship between work and symptoms/peak flow
• evidence of reversibility.

Case study – smelter asthma
Smelter asthma has been reported since the 1960s with cases occurring predominantly in smelter potrooms, where the reduction of alumina to aluminium takes place. This occurs in electrolytic ‘pots’, hence the alternative name ‘potroom’ asthma. These ‘pots’ have a steel shell, heat insulating materials, a carbon cathode lining, a pool of molten aluminium metal, and an overlying cryolite bath containing dissolved alumina with a pre-baked carbon rod suspended in the bath.

The pots are arranged in lines (‘pot lines’) in rooms (‘pot rooms’), and a direct current of over 150,000A is passed through the pots. This process releases oxygen from the alumina, leaving behind the molten aluminium metal.

The pots are hooded with fume extraction, but sections of the hood have to be opened for maintenance and other activities, so gases, dusts and fumes from the process enter the workplace.

The symptoms of smelter asthma consist of difficulty in breathing, wheezing, chest tightness and cough, and diagnosis is confirmed by spirometry and a history. Symptoms rarely appear less than two weeks after first exposure, and more usually after several months. Initially, they disappear when away from work, reappearing when back in the workplace.

Over the period 1991-2000, the incidence of occupational asthma was investigated in seven aluminium smelters in Australia (six) and New Zealand (one). The number of new cases
varied from year to year, but with a general fall in incidence rate per 1,000 employees from as high as 9.46 in 1992 to less than 1 in 2004, 2005 and 2006.

The paper discusses limitations in the study, but concluded that the success was the result of a combination of interventions, including pre-placement medical examination with exclusion of those with a history of asthma from working in the potrooms, control and reduction of exposure to the dust and fumes, and the introduction of good respiratory protection programmes. While the authors cannot pinpoint any one particular intervention method, they acknowledge the value of a comprehensive approach using a hierarchy of control, including isolation, enclosure, ventilation, work practices, education, and personal protection.

With workplace dust identified as a major hazard requiring reduction and control in many New Zealand industries, the success of the approach used in the aluminium smelter industry can act as a valuable guideline.

**Case study: Extrinsic allergic alveolitis**

An investigation was undertaken into recovering natural oils from freeze-dried krill. Once the oils were extracted, the waste left over called “mark” remained in a fine powder. Over several weeks, four workers were investigated who developed upper and/or lower respiratory symptoms.

In two cases, a diagnosis of work related extrinsic allergic alveolitis was made. In another case, the worker initially developed nose and throat symptoms but then developed asthma. In a fourth case, occupational asthma was confirmed but X-ray examination resulted in a separate diagnosis of sarcoidisis disease. This episode indicated the importance of containing contaminated workplace air at source in order to prevent respiratory conditions which can have serious repercussions for the health and well-being of the exposed workers.

**Metal fume fever**

This condition, which commonly occurs to welders cutting or welding galvanised steel, as well as less commonly to workers exposed to other freshly-formed metal oxides produced during high temperature processes.

Metal fume fever is usually a self-limiting systemic condition occurring four to 12 hours after exposure and characterised by fever, muscle aches and pains, malaise, cough and a metallic taste in the mouth. Recovery usually takes 24 hours and unless the doctor asks about a patient’s occupation it is usually diagnosed as the common ‘flu’.

In 2004 the *Journal of Occupational and Environmental Medicine* published a paper on the inflammatory responses and oxidative stress in a group of automotive welders in Taiwan. The main findings of this relatively small cross-sectional study were:

1. higher values for average urine zinc, nickel and copper in full time welders than in a control group;
2. a correlation between average urine zinc, nickel and copper levels and hours of welding per week;
3. a finding that 17.7% of welders had experienced flu-like symptoms in the course of their career;
4. urine zinc levels were associated with white blood count, neutrophil count, eosinophil cancer and inter leukin-6, suggesting the inflammatory response might be mediated by cytokines;
5. the results confirm other studies that welding fume exposure is associated with systemic inflammatory responses, as indicated by increased levels of white blood cells and neutrophils, while the raised eosinophil levels might suggest an allergic mechanism where local injury results in inflammation with the release of a histamine-like substance; and
6. it was also noted that smoking was significantly associated with urine zinc and nickel levels.

Chronic obstructive pulmonary disease

Chronic obstructive respiratory disease (CORD), or chronic obstructive pulmonary disease (COPD), as it is now more commonly referred to, “is the fourth leading cause of death worldwide”34. It is defined as a condition with airflow limitation which is not fully reversible, is progressive and is associated with an abnormal inflammatory response of the lungs to noxious particles or gases. Historically, and still, the major cause is cigarette smoking. However, there is increasing evidence indicating that exposure to dusts, gases, and fumes at work are linked to the development of COPD. As a result, it is now recognised as an occupational disease in certain situations, with likely additive effects occurring between smoking and some workplace exposures.

Contaminants of air associated in studies with work-related COPD include: welding fumes, silica, coal, oil mist, Portland cement, cotton, grain and wood dusts35.

Diagnosis of COPD requires both a clinical and occupational approach and as far as the latter is concerned, a careful occupational history. That is, a chronological list of jobs, what the job entailed, which respiratory exposures occurred, to what extent and for how long.

As a consequence, the panel is now not only recording the presence of COPD in asbestos-diagnosed cases, but also in asbestos-exposed and non-asbestos exposed cases.

Again, in the rebuilding in Christchurch, occupations such as welding and concrete drilling, cutting and grinding could lead to increased exposure and health risk.

Silica and silicosis – one agent, many outcomes

Silica is the most common element found in the earth’s crust. When combined with other substances, silica is relatively harmless. Silicosis is a serious lung condition described by the ILO as an accumulation of crystalline silica dust in the lungs and the tissue reaction to its presence. It is caused by inhaling airborne crystalline silica dust in high concentrations over a period of time.

In a review of the outcome of silica exposure, Steenland36 noted “evidence in recent years indicates that silica causes lung cancer, and probably renal disease, in addition to its well-known relationship to silicosis” and, as indicated in the previous section, it can also result in Chronic Obstructive Pulmonary Disease.

While silicosis is rarely diagnosed in New Zealand, there is evidence that most New Zealand rocks contain some quartz, and with erosion and quarrying, the quartose sands tend to
contain a higher content of quartz than the parent material\textsuperscript{37}. This was re-affirmed in an extractive industry study where the dust measurements carried out by the Department of Labour showed that in 13\% of the air samples collected, levels of respirable quartz exceeded 0.2mg/m\textsuperscript{3} (the current New Zealand Workplace Exposure Standard).
Appendix 1

Contact details for the Asbestos and Occupational Respiratory Disease Registrar

Asbestos and Occupational Respiratory Disease Registrar
Ministry of Business, Innovation and Employment
PO Box 3705
Wellington 6140

Members of the National Asbestos Medical Panel

W.I. Glass ONZM, MBChB, DPH, DIH, FFOM (Lond), FAFOM (Hon), FFOM (I) (Convenor).

R. Armstrong MBChB (Hons), FRCP, FRACP.

D. Jones MBBS, MRCP (UK), FRACP.
Appendix 2

The National Asbestos Registers

The National Asbestos Registers were established in March 1992 in line with recommendations made to the Minister of Labour by the Asbestos Advisory Committee.

Formation of the Asbestos Advisory Committee

The Asbestos Advisory Committee was established in October 1990 to report to the Minister of Labour on issues relating to the health effects and use of asbestos in New Zealand, adequacy of controls and legislation, and clarification of the legal entitlements available for affected workers. This followed increasing public concern about past and present effects of asbestos on workers, former workers and their families.

Establishment of the National Asbestos Registers

Recommendation 4 of the Report of the Asbestos Advisory Committee to the Minister of Labour advised:

“That an asbestos medical register be established for people who have been significantly exposed to asbestos. OSH (formerly the Occupational Safety and Health Service of the Department of Labour, now the Ministry of Business, Innovation and Employment) should be the organisation responsible for establishing, maintaining and funding the medical register.

The medical register should be in two parts:

Part 1 – Those notified as having been exposed to asbestos;

Part 2 – Those notified as having an asbestos-related disease.

The system should allow movement of the name of a registered person from Part 1 to Part 2 of the register when indicated.

Notifications to Part 1 of the medical register were to be made by those who felt they had been exposed to asbestos, or by people acting on their behalf (and following consultation), such as an employer, union official, relative or friend.

Notification to Part 2 of the medical register would be done by medical practitioners.”

A Notifiable Occupational Disease System (NODS) was established in 1992 and the Asbestos Registers have been incorporated in that scheme. This was in accordance with recommendation 5 of the Asbestos Advisory Committee.
The Ministry, in association with Electricorp Production Ltd, undertook an extensive advertising campaign in March and April 1992. Advertisements were published in all the major newspapers and several trade magazines.

The interest generated as a result of this campaign ensured a high response rate for the Exposure Register. Notifications were made by individuals, trade unions, occupational health nurses, doctors, the Asbestos Diseases Association of New Zealand and by some larger companies.

Notifications were directed to branch offices of the Ministry or directly to the Registrar.

The Exposure Register

In recommendation 4, the committee envisaged that people wishing to be recorded on the asbestos exposure register would have their exposure assessed at an office of the Department. Only those people who were judged as having had ‘significant exposure’ would then be recorded on the register. However, the huge response from individuals exposed made it impractical to screen registrants in this fashion.

Once a person notifies MBIE that they have been exposed to asbestos, an asbestos exposure registration form is sent to them. The form collects information about the individual, their work exposure to asbestos and the state of their respiratory health.

When the completed form is returned to the registrar, the details are recorded on a database. The individual is then sent a copy of Asbestos – A Deadly Dust, which is dedicated to asbestos and its associated health problems. If the person indicates that they have a family doctor, the doctor is informed their patient has been included on the Asbestos Exposure Register, and is sent a copy of the Ministry’s booklet Asbestos Exposure and Disease: Notes for Medical Practitioners.
The Disease Register

Tenders for the National Asbestos Medical Panel were called for in 1991. A tender was accepted on 31 October 1991. The successful tender came from the group listed below:

Dr R. Armstrong, Professor R. Beasley, Dr J. Crane, Associate Professor W. Glass, Dr D. Jones, Dr N. Pearce.

Professor Beasley retired upon his appointment as Professor of Medicine at the Wellington Clinical School. Dr Crane joined the National Occupational Asthma Panel. Dr D. Fishwick joined the panel in 1997. He was subsequently appointed to a position in the United Kingdom. In 2008, Andrew Brant joined the Panel, later resigning to become Chief Health Officer at North Shore Hospital.

The first meeting of the panel was held in February 1992. Professor Glass was nominated as the panel’s convenor.

The following members were appointed to the National Asbestos Radiological Panel: Dr Paul White, Dr George Foote and Dr Graeme Anderson.

The Registrar from 1991 to 1996 was Craig Eades. From 1996 to 1998 the position was held by Nicola Holden, and later in the year by Andrea Eng until 1999 when Louisa Thomas was appointed.


Processes for registering people

Notifications for the register come from two major sources. The first is from doctors whose patients have been diagnosed or are suspected of having an asbestos-related disease. The second source of notification is from individuals. Once a notification is made to the Registrar,
and consent gained from the person concerned, relevant medical records and a full occupational history are obtained.

**Data collection**

The data collected includes a medical history, occupational history, chest x-ray, CT scan where available, lung function tests and pathology reports. On notification being received by the Registrar:

- An occupational health nurse visits the patient and conducts a health interview, a detailed occupational and social (including smoking) history.
- Relevant medical reports are obtained from general practitioners and physicians.
- A PA chest x-ray is obtained and read by a radiologist according to ILO (1980) guidelines. CTs are used where available, and on occasions requested.
- Lung function data is obtained from physicians’ reports or requested from respiratory laboratories. Where this is not possible, results are obtained from a test carried out by an occupational health nurse, using a portable spirometer.
- Pathology and post-mortem reports are reviewed where available.

![Exposure Index Calculation Formula](image)

This is calculated for each job and total exposure is the sum of each D.

**Figure 12: Exposure Index Calculation Formula**

**Data assessment**

The National Asbestos Medical Panel reviews the information obtained, calculates an exposure index (see overleaf) and correlates the medical data.

**Exposure Index**

An exposure index (D) was calculated from the product of years of asbestos exposure (A); intensity of exposure, using a 1-5 grading according to job category (B); and degree of exposure, using a 3-point grading (C).

Guidelines for calculating this index are shown as follows:

$$ D = A + B + C $$

A = Total years of exposure in any one job
B = Job intensity as follows:
   - Mining, milling and processing = 5
   - Boiler/lagging, rail carriages, shipyard, spraying insulation = 4
   - Asbestos cement products, construction, demolition, removal = 3
   - Electrical, friction products = 2
   - Loading, driving, environmental = 1

C = Degree of exposure (unprotected)
   - Continuous (>50% of work) = 3
   - Intermittent (20-50% of work) = 2
   - Minimal (<20% or occasional) = 1

D = A x B x C for each job
   Exposure index = sum of all Ds

**Medical data**

Relevant respiratory symptoms and signs are recorded from the medical histories, and lung function data is classified into restrictive, obstructive, mixed or normal. Pathology reports are used to confirm mesotheliomas and classify lung cancers.

**Classification of Diagnostic Categories**

On the basis of the foregoing, the cases were placed into a primary diagnostic category of:

- mesothelioma
- lung cancer
- asbestosis
- pleural abnormalities (plaques, diffuse bilateral pleural thickening and effusions)
- other cancers
- obstructive lung disease without x-ray changes.
Appendix 3

Ministry of Business, Innovation and Employment publications on asbestos


National Asbestos Registers, Annual Reports:
- 1992-1993
- 1994-1995
- 1995-1996
- 1996-1997
- 1997-1998
- 1999-2000
- 1999-2000
- 2000
- 2001
- 2002
- 2003
- 2004
- 2005
- 2006
- 2007


Out-of-Print Publications (limited availability)


Asbestos: A Deadly Dust. 50 Years of Asbestos Use in New Zealand. Reprint from Safeguard magazine, December 1991.

Appendix 4

References


