



Asbestos and Other Occupational Lung Diseases in New Zealand

1992 - 2008



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INTRODUCTION

THE 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.

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

Worldwide, asbestos exposure still occurs at a high level and there are a number of lessons than 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 whether or not dust is a danger to health. Concepts that cement dust is safe and silica dust unsafe, or blue asbestos is worse than white asbestos have distracted governments from the real issue of dirty workplace air.

Today this is recognised as a major problem, both in terms of respiratory and general health. However, its solutions are not complex. They involve putting in place good general and local exhaust ventilation, and providing comfortable and effective respiratory protection. While that sounds relatively simple, there needs to be a desire for clean workplace air.

So where to from here for the Registers? 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 recognised more clearly for their relationship to work practices and procedures.

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National Asbestos Medical Panel
July 2009

PART 1: REVIEW OF ASBESTOS-RELATED DISEASE NOTIFICATIONS

1.1 Summary

This report reviews 1125 cases that were notified to the National Asbestos Medical Panel between March 1992 and July 2008. They include:

- 212 cases of mesothelioma
- 107 cases of lung cancer
- 253 cases of asbestosis
- 553 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.

The transfer of asbestos from workers to the home is another emerging feature of asbestos-related disease in New Zealand. Family members are presenting with pleural changes or, rarely and tragically, mesothelioma.

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

Three Department of Labour publications have arisen from the registers. They are:

- *Respiratory Symptoms and Asbestos Dust Exposure* (1997)
- *Mesothelioma in New Zealand* (2000)
- *Lung Function Changes in Asbestos Exposed Workers with Pleural Abnormalities* (2000).

In addition, two special reports have been published:

- *Recent Advances in Asbestos-Related Disease* (Dr M Becklake, 1994)
- *The Epidemiology of Mesothelioma in Historical Context* (Doctors J C and A D McDonald, 1998)

1.2 Results

Figure 1 shows the distribution of the four main diagnostic categories:

- Lung Cancer
- Pleural Disease
- Asbestosis
- Mesothelioma.

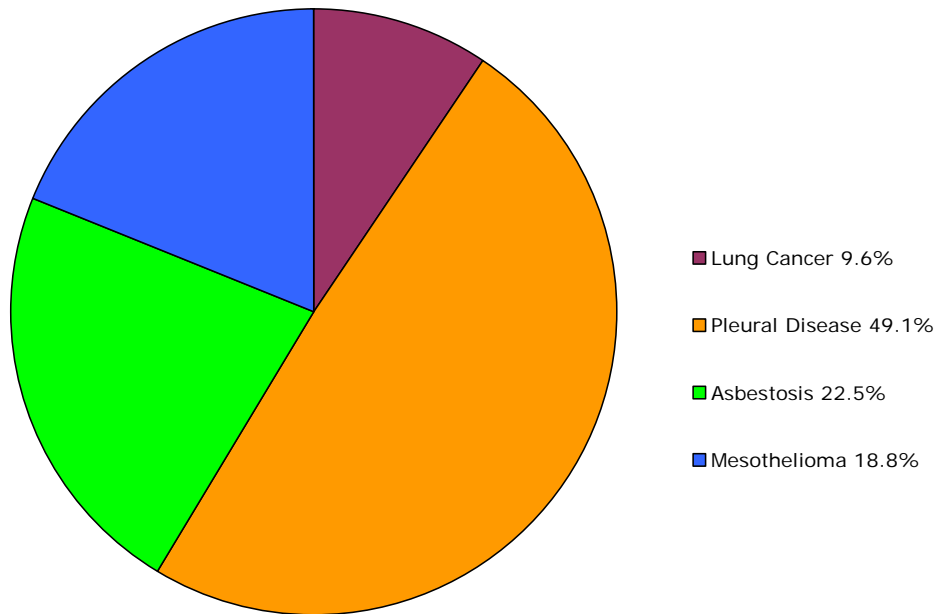


Figure 1: Categories of Disease 1992-2008

What is noticeable is that pleural disease is the main category with lung cancer clearly under-represented when compared with mesothelioma.

Figure 2 looks at occupations for the total number of notified asbestos disease cases during the period.

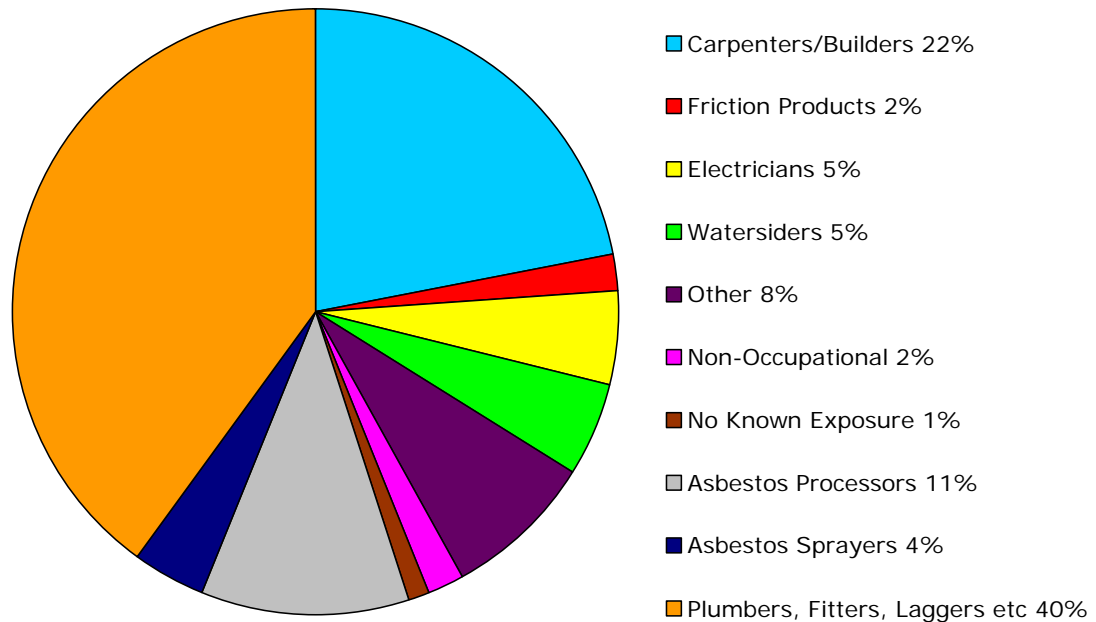


Figure 2: Notified Asbestos Disease by Occupation 1992-2008

It is clear that carpenters, plumbers and electricians are together responsible for 67 percent 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).

1.3 Mesothelioma

Mesothelioma - a rare cancer of the pleural membranes on the surface of the lungs - is strongly related to asbestos exposure. The panel reviewed 212 cases of mesothelioma, of which:

- 204 were Caucasian
- 6 were Maori
- 2 were identified as “Other”
- 199 were males, 13 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 19 current smokers, 116 ex-smokers and 59 never-smokers (information for 10 cases was unavailable).

The three occupations: asbestos processors, plumbers/fitters/laggers, and carpenters/builders, accounted for over 60 percent of all registered cases.

It has been noted that an asbestos exposure history may be lacking with mesothelioma cases. Our experience suggests that with patience and recognition of the range of likely exposures, it is often possible to obtain evidence of asbestos exposure. In one case the disease developed in a middle-aged woman living in a small rural town. It was revealed that as a teenager she had washed the clothes of an older brother who had been a railway workshop apprentice. Asbestos lagging was used in the repair and maintenance of the boilers, and apprentices were known to use asbestos in 'snowball fights'.

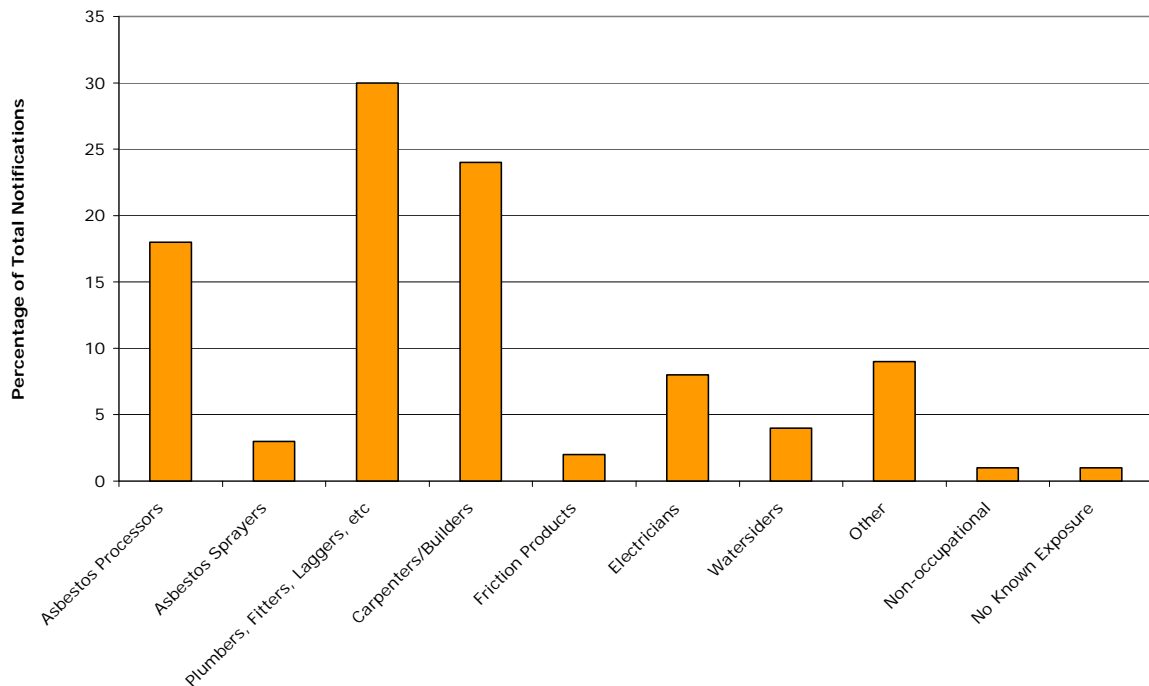


Figure 3: Distribution of Mesothelioma by Occupation

1.4 National Cancer Figures on Mesothelioma

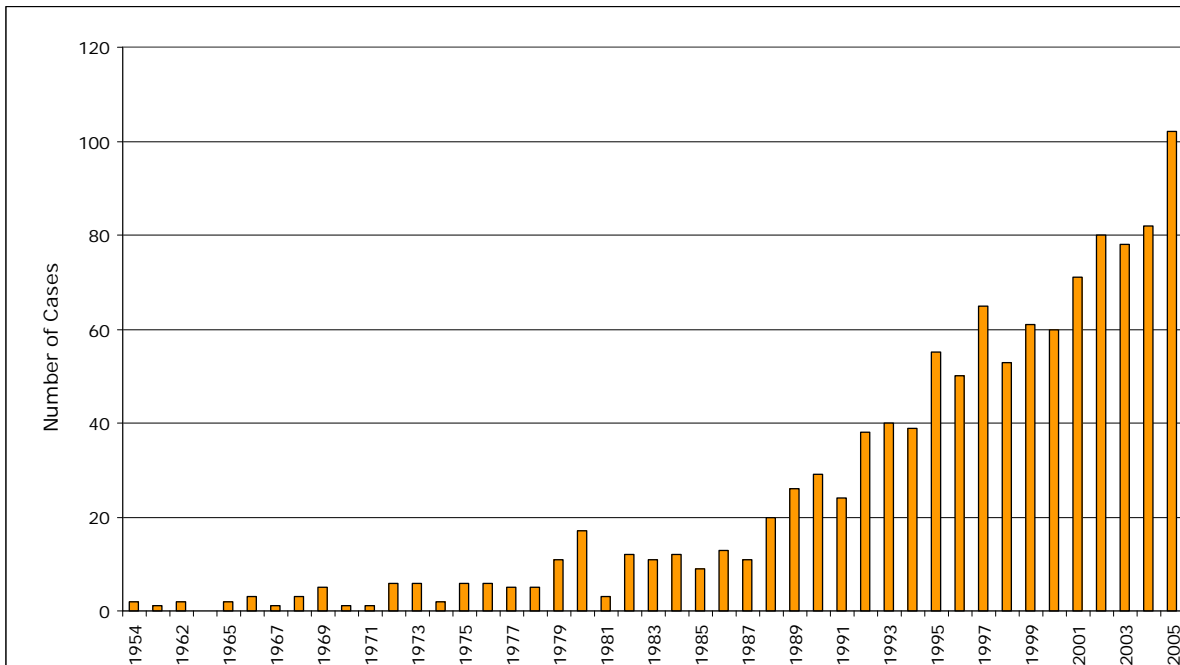


Figure 4: Number of Cases of Mesothelioma in New Zealand 1954 to 2005

Over the period 1954-2005 a total of 797 cases of mesothelioma have been registered. Figure 4 shows that the total number of cases continues to rise up to 2005 (the latest figures) and exceeds 100 for the first time.

Mesothelioma is very much a disease of old age as Table 1 illustrates, with over 50% of cases occurring to people aged 70 or over.

Age Group	Gender		Total
	Male	Female	
Under 20	0	0	0
20 to 44	6	10	16
45 to 69	335	42	377
70+	346	58	404
Total	687	110	797

Table 1: Mesothelioma Occurrence by Age Range

The male:female ratio is about 7:1 except in those under 45, where this gender ratio is reversed at 1:1.6. Of the 797 cases, 110 occurred to women and 687 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.

1.5 Lung Cancer

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 107 cases of lung cancer, of which:

- 101 were Caucasian
- 4 were Maori
- 1 was from a Pacific Island
- 1 was identified as "Other"
- 105 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 25 current smokers, 72 ex-smokers, 7 never smokers and 3 unknown.

Lung cancers are classified according to the type of cell affected. Histological classification revealed 52 cases of squamous cell carcinoma, 27 adeno, 12 small cell, 4 undifferentiated, 1 bronchiolar-alveolar, 3 large cell and 8 cases where classification was not stated. In addition, the tumour sites were as follows: 43 upper lobe, 39 lower lobe, 11 middle lobe, and 14 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. See Figure 5: Distribution of Lung Cancer by Occupation.

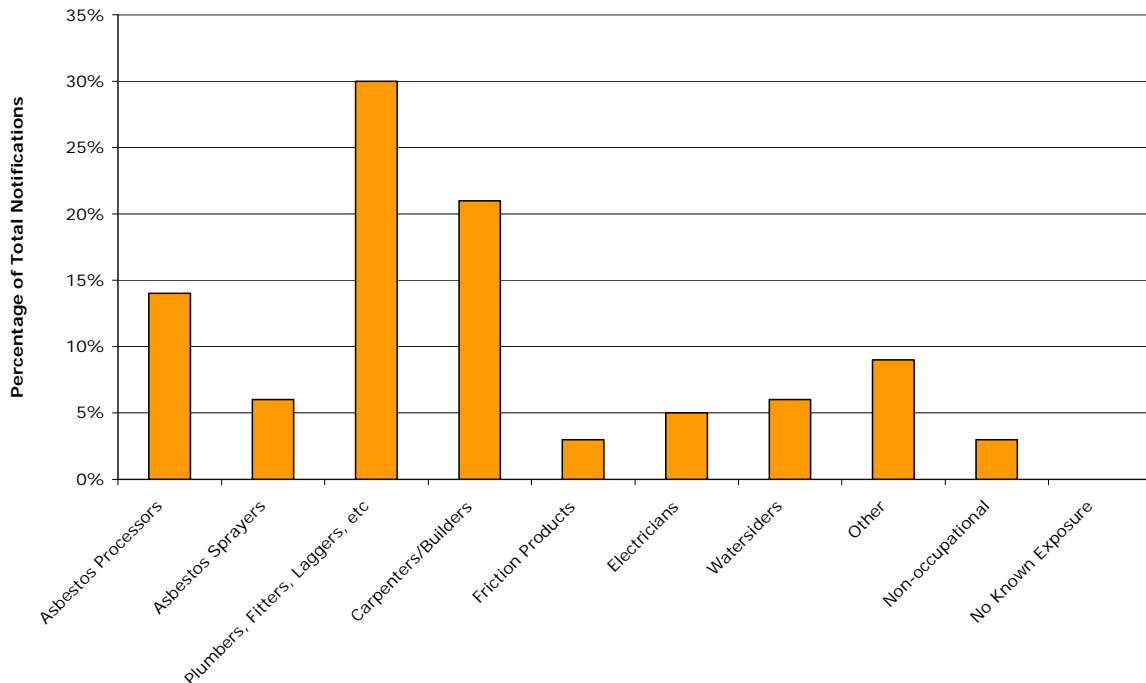


Figure 5: Distribution of Lung Cancer by Occupation

1.6 Asbestosis

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 253 cases of asbestosis, of which:

- 249 were Caucasian
- 2 were Maori
- 2 were from a Pacific Island
- 237 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 20 current smokers, 183 ex-smokers and 43 never smokers (accurate smoking histories were not available in 7 cases)
- Radiological changes showed 142 cases with pleural plaques and/or pleural thickening.

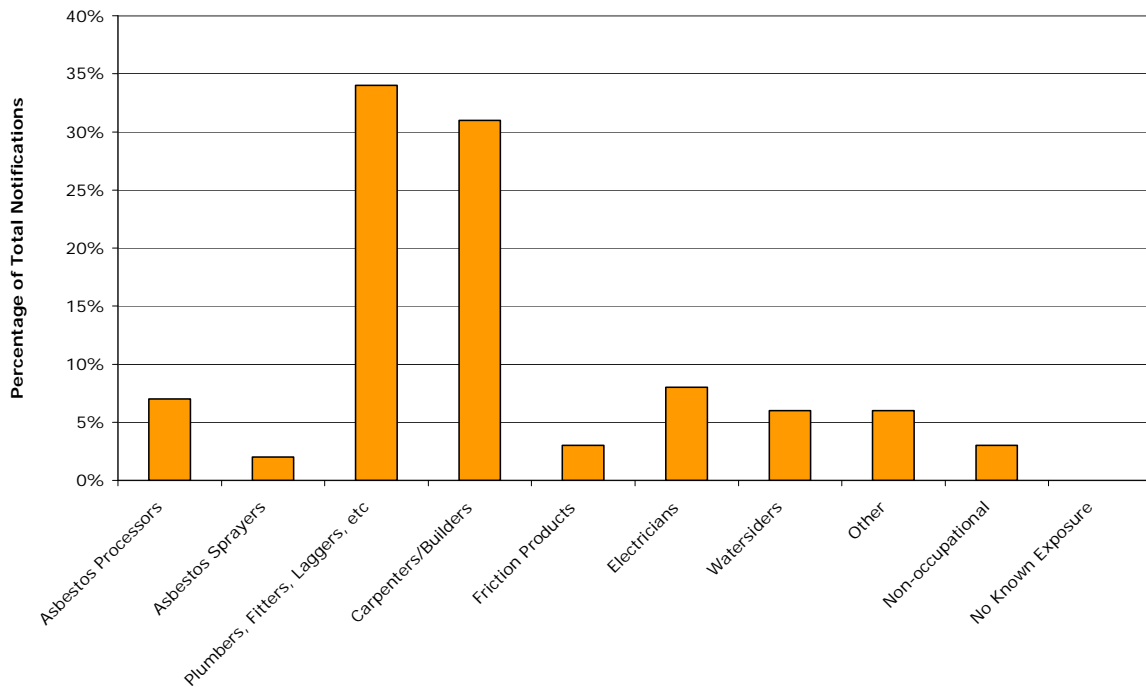


Figure 6: Distribution of Asbestosis Notifications by Occupation

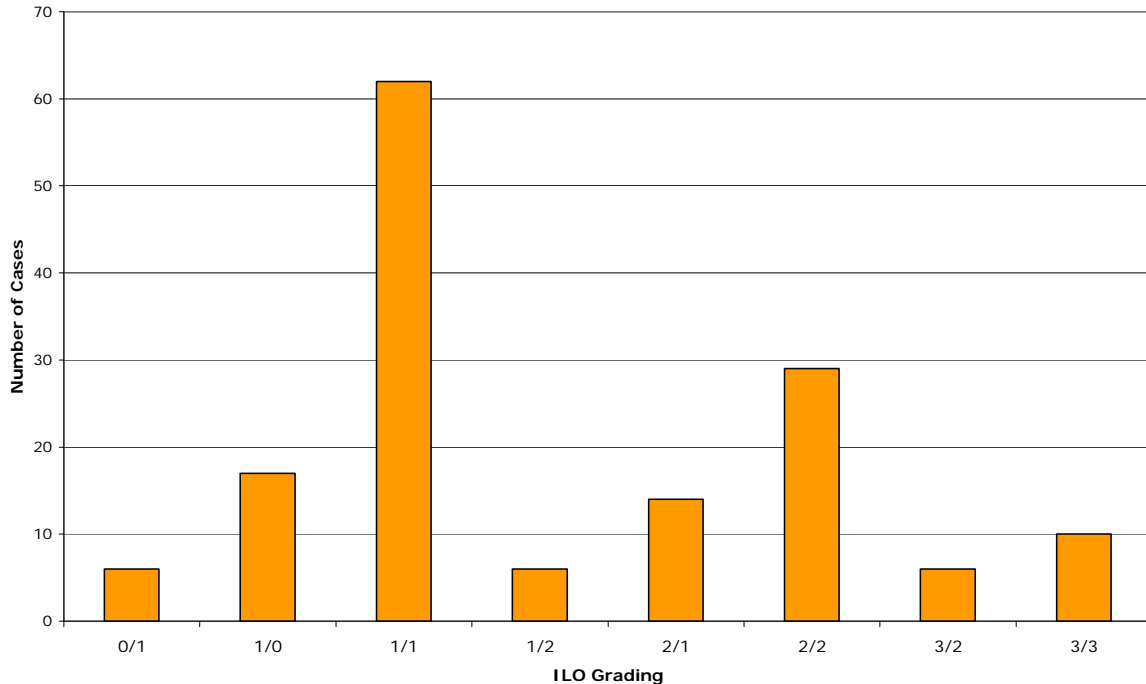


Figure 7: ILO Grading of Asbestosis Cases (n = 150)

Of the 253 asbestosis cases, 148 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.

1.6.1 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*^a 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
- (d) Lung function changes consistent with at least some features of the restrictive syndrome.

Gilson notes 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

^a Gilson JC: *Asbestosis, Encyclopaedia of Occupational Health and Safety, 1983 3rd edition, vol 1 pp 187-191*

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 253 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.

The lung function test numbers in the report confirm the classical restrictive lung function pattern picture does not dominate, with obstructive, mixed, and normal patterns occurring.

1.7 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.

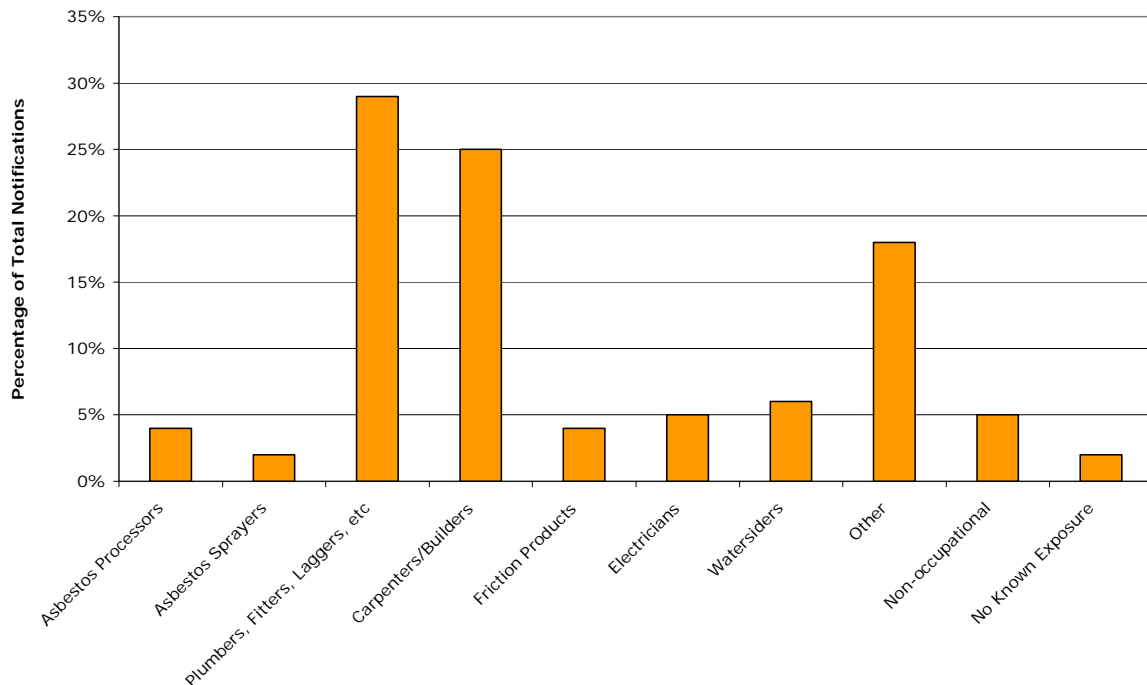


Figure 8: Distribution of Pleural Abnormality Notifications by Occupation

Of the 553 cases reviewed:

- 542 were Caucasian
- 7 were Maori
- 4 were from Pacific Islands
- All but 10 were males
- The mean exposure index was 162 (range 6-708)
- There were 52 smokers, 328 ex-smokers and 138 never smokers (accurate smoking histories were not available in 35 cases).

1.8 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 a useful purpose. There is now greater awareness of the work factor in disease than in 1992 when the Register was established. The Department of Labour 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 the Department of Labour have, in the view of the medical panel, played an important part in encouraging these developments.

1.8.1 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 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 was made earlier 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; the other, a carpenter.

1.8.2 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.

1.8.3 Lung Cancer

The contribution of occupational asbestos exposure to the causation of lung cancer is well recognised as being underestimated, 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 and, as noted by *Kjellstrom^b* can range from 1 to 10. Even if the lower ratio of 1:2 is taken – based on the mesothelioma cases diagnosed over 1994-2005, for example – some 1,594 cases of lung cancer due to asbestos exposure would have occurred, or approximately 145 a year. It is likely that this figure could be even higher.

1.8.4 Mesothelioma

Reported cases of mesothelioma have continued to rise in New Zealand over the past decade as was shown in Figure 4, 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.

1.8.5 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 year 33% of the 85 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, eight cases that were referred to the Panel because of asbestos exposure but without classical asbestos-related conditions, had COPD.

^b Kjellstrom T: *The Epidemic of Asbestos-Related Diseases in New Zealand*. International Journal Occupational - Environmental Health 2004, 102 June

PART 2: NON-ASBESTOS 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 non-asbestos occupational respiratory disease as referenced below:

- 1938** Silicosis: Report of Interdepartmental Committee, Bulletin No 57, Department of Scientific and Industrial Research, Wellington¹.
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. McLean² 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 Survey³ 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, to a large extent 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 nationally⁴ among farmers⁵, sawmill workers⁶ and aluminium workers⁷; at the same time respiratory symptoms, lung function and dust levels were measured in the wood industry^{8,9}, plywood mill workers^{10,11}, hairdressers¹², mussel openers¹³, welders^{14,15,16,17,18,19,20,21} and quarry workers²².
- 2000** In 2000 Dr Jeroen Douwes joined the Centre, accelerating the interest in sawmill workers, wood dust and respiratory effects.

2.1 Occupational Asthma

In the Colt Lecture given by Anthony Newman Taylor at the Ninth International Symposium on Inhaled Particles at Cambridge University in 2001²³, 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 the Department of Labour 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 respiratory 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.

2.2 Occupational Asthma Case Studies

Case 1

This case concerned a 44 year old woman who began work as a hospital cleaner, then in mid 2003 worked in theatre as a sterile technician for 15 hours a week. She was exposed to "Shipshape" (a scourer), and "Epizyme" (a washing fluid which was heated, forming a mist). Epizyme contained cellulose, amylase, lipase and proteinase.

Some five to six months after beginning work, she noticed sneezing, reddened eyes and a cough, worse during work days and better in the weekend.

Four months later her symptoms worsened with breathlessness, wheeze and back pain. She had a nine-month period off work with improvement of her lower respiratory tract symptoms after about three months, but not her upper respiratory tract symptoms.

On returning to work to the hospital her symptoms returned and remained for the next two years.

Epizyme was ultimately identified as a potential cause and removed, with a gradual improvement of her chest symptoms, but persistence of her upper respiratory tract symptoms. Her past history indicated no childhood asthma, hay fever or eczema. She was an ex-smoker of 20 years.

Investigations showed normal lung function and chest X-ray. There were no work-related serial peak flow tests available. A methacholine bronchial provocation test with a PC20 of 1.4mg/ml showed moderate hyper-responsiveness. Reviewing her history there was latency between her first exposure and development of her upper respiratory tract symptoms, and a further latency before her lower respiratory tract symptoms occurred – a pattern typical in asthma due to enzyme exposure.

There was a work-relatedness with her symptoms in the initial stage of her exposure, and the chemicals are known asthmagens. Her outcome to date has not been one of recovery as she now has a non-specific bronchial hyper-responsiveness as a result returning to work even though the enzymes have been removed.

Case 2

This case concerned a 54 year old woodworker who used western red cedar, macrocarpa and occasionally rimu and Oregon pine in the manufacture of timber doors and windows. In the first two to three years he had no health problems. Over a Christmas holiday, he developed upper respiratory tract symptoms with sneezing and itchy eyes which continued on his return to work, gradually involving his lower respiratory tract with cough and phlegm, breathing difficulties and "chills". He noted that the symptoms were worse at work, improving when away from work. He now finds that cold winds and exercise act as respiratory irritants.

In his past history he had no childhood asthma or hay fever and is a very modest ex-smoker with a pack per year history of two to three.

His spirometry was normal, his skin tests for atopy negative apart from a small reaction to house dust. No peak flow measurements were available.

The diagnosis was made from his history and his exposure to a known asthmagen (western red cedar) which contained plicatic acid, a sensitising agent. The slow development of his condition, first upper airway symptoms then lower airway symptoms, was consistent with such exposure.

Case 3

This case involving a 27 year old male was less clear cut. He had a previous history of working in the boat building industry and cabinet making, but without symptoms. He then began a boilermaking apprenticeship where he worked in a large, dusty factory and was exposed to welding fumes. After some two months he developed a mild cough and tiredness. This improved over the two week Christmas break with the symptoms returning when he recommenced work.

He then had a job on a fishing boat below deck, removing steel tanks by grinding the steel into strips. Beneath the steel there was polyurethane foam which was caught up in the hot grinding disc. There were significant fumes in this confined space, and a fan had been installed and a paper filter mask provided. He worked for two hours then had a break. On his second "shift" he was the only individual doing this job and the fan had been removed. He felt unwell, dizzy, and had a nasty taste in his mouth. He went on deck for a break before returning to complete the task over the remaining four hours. That evening he had a blocked nose, headache, flu-like symptoms, cough and was short of breath, finding it difficult to breathe. He saw his General Practitioner who recorded "moist chest sounds".

Investigations showed a normal X-ray, normal spirometry, no bronchial hyperactivity (methacoline challenge), peak flows unconvincing, positive atopic skin test, negative isocyanate IgE.

The respiratory physician raised the possibility of asthma, but this could not be confirmed.

2.3 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 career²⁴

4. Urine zinc levels were associated with white blood count, neutrophil count, eosinophal 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 eosinophal levels might suggest an allergic mechanism where local injury results in inflammation with the release of a histamine-like substance
6. It was also noted that smoking was significantly associated with urine zinc and nickel levels.

2.4 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”²⁵. 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 dusts²⁶.

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.

2.5 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 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 Steenland²⁷ 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 material²⁸. This was re-affirmed in the 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/m³ (the current New Zealand workplace exposure standard).

2.6 Indoor Air Quality (IAQ)

An article on particle emission characteristics of office printers²⁹ revealed that among 62 printers investigated, while 60% did not emit submicrometer particles, 40% did, among which 27% were high emitters. However, there is no data to link any harm to these exposures. Office-related respiratory conditions have been recognised for some years with pollutants entering the office environment from outdoor air, as well as pollutants arising from within the office environment. These include organic vapours (VOCs), fibres and inorganic gases. With increasing numbers of office workers, attention to indoor air quality is as important as attention to factory and outdoor (quarries, construction) air. The article makes the point that by identifying the high emitters and recognising emitting rates are printer-type specific and are affected by toner cartridge age, such contaminants of indoor air "can be reduced by a proper choice of printers".

2.7 Recent "New" Work-Related Occupational Respiratory Disease

In an editorial in *Occupational Environmental Medicine* *Kreiss*³⁰ looked into the future. She noted recent, "new", occupational respiratory diseases such as flock workers' lung - an interstitial lung disease - hypersensitivity pneumonitis associated with bio-contaminated synthetic metal working fluids, asthma associated with 3-amino-5-mercapto-1, 2, 4-triazole (AMT) in herbicide manufacture, and bronchiolites obliterans from flavouring chemicals used in popcorn plant workers.

Kreiss wrote of barriers to recognition of new respiratory diseases, including:

- Clinicians too readily attributing lung disease to smoking
- The lack of work-related symptoms in some diseases, i.e. occupational industrial diseases
- Long latency of some diseases
- Lack of knowledge by physicians of patients' workplaces and hazards

With regard to recognition of new diseases, she instanced the value of a national state-based surveillance system for occupational asthma.

As to the future and emerging causes of occupational lung disease, *Kreiss* cites asthma related to damp offices. "Building dampness can be considered an emerging cause of occupational lung disease because we are still unsure of causal agents ..."

In addition, *Kreiss* raises the issue of COPD and states: "Although 80% of COPD is attributable to smoking, most of the remainder is likely to have occupational

causes." She goes on to say that "control recommendations can be made, even when such relations are based on surrogate exposures and before specific aetiological agents or safe levels of exposure are known".

PART 3: ASBESTOS ISSUES AHEAD

3.1 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 asbestos

Overseas studies confirmed the New Zealand experience with high levels of asbestos dust measured in rooms during the sanding process.

3.2 Asbestos Removal Workers

This group of workers was very active in the late 1980s and the 1990s. Although the Department of Labour published the *Guidelines for the Management and Removal of Asbestos (1995)*, undoubtedly a proportion of such 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.

3.3 Brake Lining Repair Workers

Brake linings still contain asbestos. Such work is often carried out in designated 'small workplaces'. As such they are less likely to be inspected, 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 ongoing 'at-risk group'.

3.4 Building Maintenance Workers

It is of significance that the Great Britain 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.

3.5 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 no knowledge of exposure occurs.

APPENDIX 1: BACKGROUND TO THE 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 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 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 Occupation Disease System (NODS) was established in 1992 and asbestos registers have been incorporated in that scheme. This was in accordance with recommendation 5 of the Asbestos Advisory Committee.

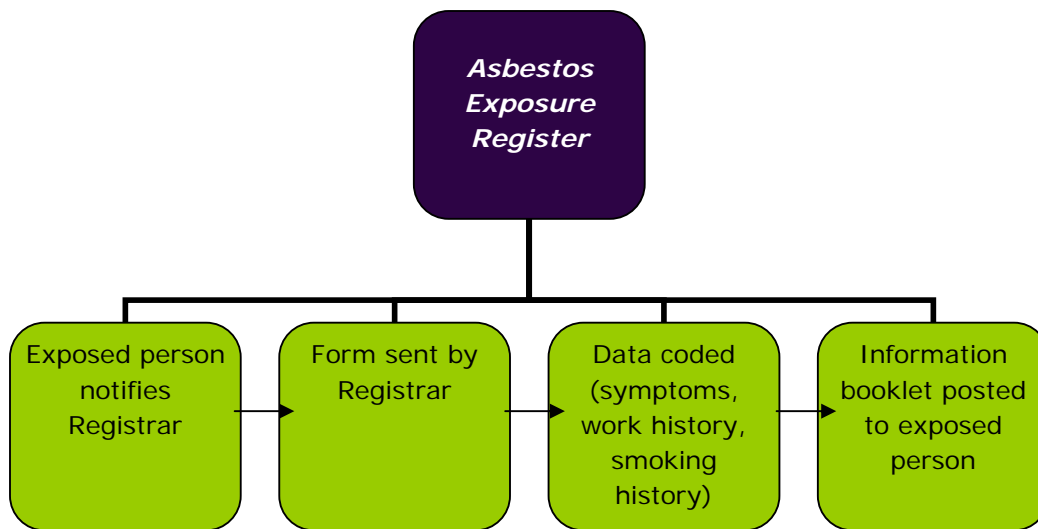


Figure 9: Asbestos Exposure Register Process

The Department of Labour, in association with Electricorp Production, 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 has ensured a high response rate for the exposure register. Notifications have been made by individuals, trade unions, occupational health nurses, doctors, the Asbestos Diseases Association of New Zealand and by some larger companies.

Notifications are directed to branch offices of the Department or directly to the Registrar.

In recommendation 4, the committee envisaged 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, although this has since been done.

Once a person notifies the Department of Labour they have been exposed to asbestos, an asbestos exposure registration form is sent. 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

Department's booklet *Asbestos Exposure and Disease: Notes for Medical Practitioners*.

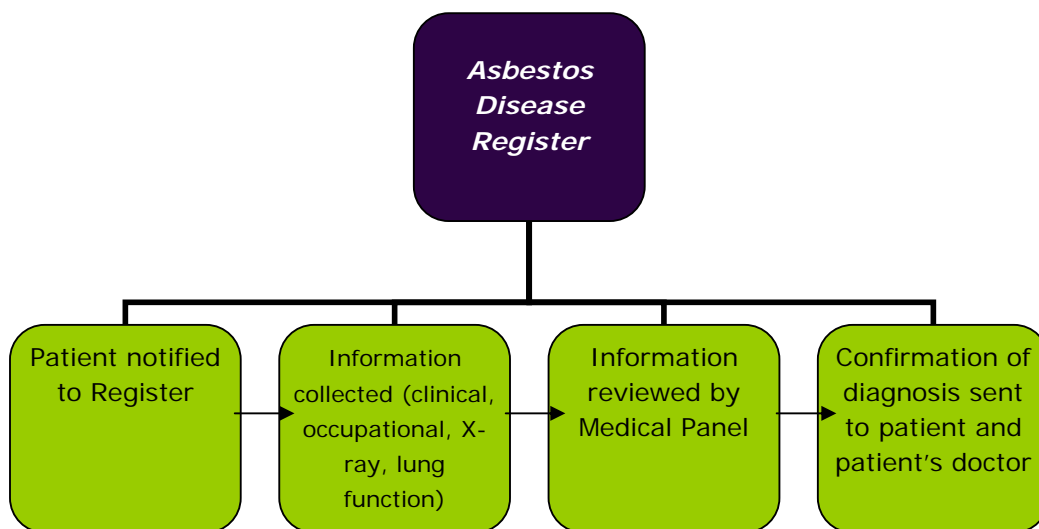


Figure 10: Asbestos Disease Register Process

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.

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.

In 2002 Jenny West was the Registrar, with Dougal McNeill Registrar in 2004 and Gail Abel in 2006, Justine Ward in 2007 and Anne Thomas in 2008.

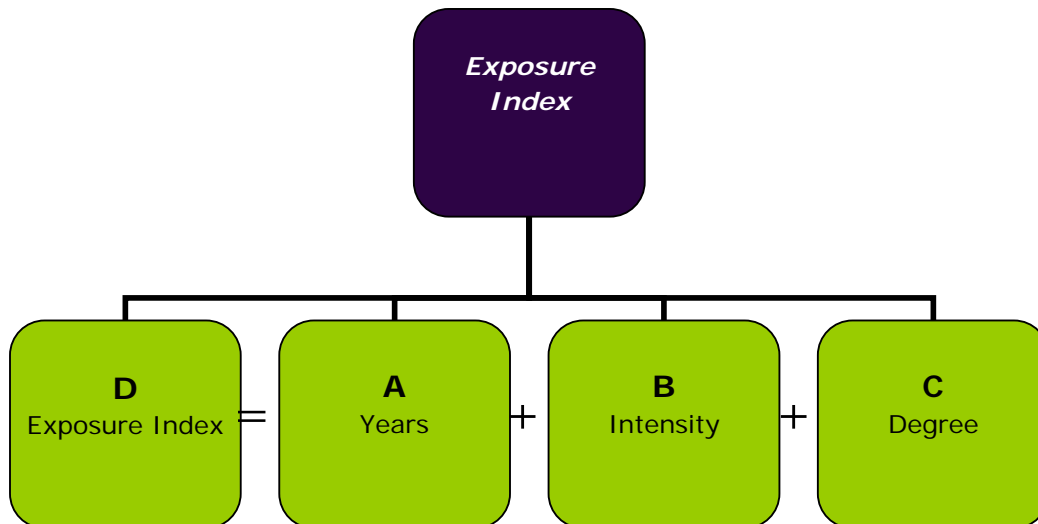
Processes for Registering People

Notifications for the register come from two major sources. The first is from doctors whose patients have been diagnosed or were 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.



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

Figure 11: Exposure Index Calculation Formula

Data Assessment

The National Asbestos Medical Panel reviewed the information obtained, calculated an exposure index (see overleaf) and correlated 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 below.

- 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 were recorded from the medical histories, and lung function data was classified into restrictive, obstructive, mixed or normal. Pathology reports were 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 2: ASBESTOS

What is Asbestos?

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

Figure 12 below shows the different types of asbestos.

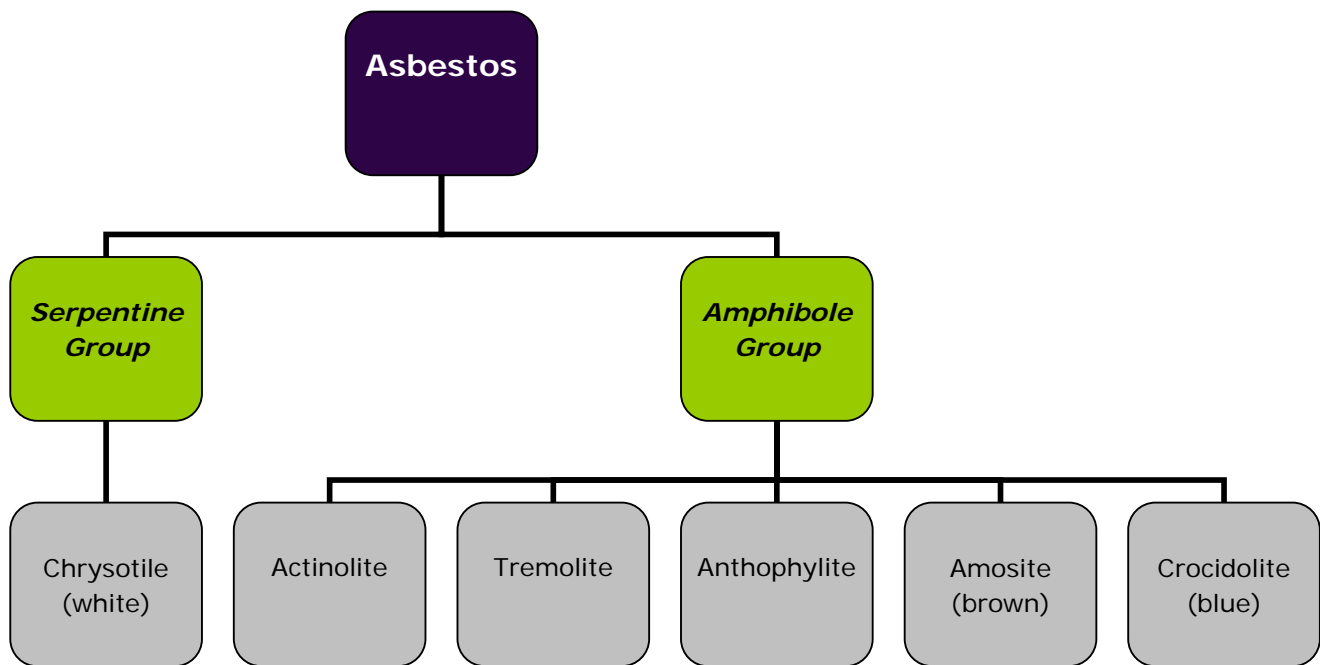


Figure 12: Types of Asbestos

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 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.

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) was reduced from 2 fibres/ml to 1 fibre/ml.

Crocidolite remained at 0.2 fibres/ml over a 10-minute sampling period.

- 1982** Jim Butterworth, Auckland Secretary, NZ Engineers Union, expresses concern about long-term effects of asbestos on workers at James Hardie factory.
- 1983** *Interim Report of Workers at James Hardie* (unpublished) Kjellstrom T and Rennie F. Of 353 workers x-rayed, 53 showed parenchymal and/or pleural changes. An increasing percentage of abnormalities found over time since first exposure.
- Asbestos Regulations replace 1978 regulations.
- Gazette* notice reduced 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 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) Glass W.I. 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** *Lung Cancer, Smoking and Exposure to Asbestos in New Zealand.* Glass W.I; Kawachi I., Pearce N. *Journal of Occupational Health Safety* 7 (1).
- "Case control studies based on NZ 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 and 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 was 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 was 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 was carried out by KD Sheat and published by OSH (Department of Labour).
- ACC lump sum entitlements for asbestos-related claims ceased.
- 1993** The first *Annual Report of the National Asbestos Registers* confirming 199 cases of asbestos-related disease.

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

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

Secondary cases of asbestos disease found to occur in family members of asbestos workers whose 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 registers.

1995 *Asbestos Exposure and Disease Notes for Medical Practitioners* published by Department of Labour.

1995/96 The third annual report confirms 535 cases.

1996 *The Management of Asbestos in the Non-Occupational Environment* published by 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 published its first report based on a review of asbestos cement-exposed workers on the exposure register. 2257 notifications were reviewed. A positive finding was a non-specific association between cumulative asbestos exposure and a questionnaire diagnosis of asthma.

1998 Health and Safety in Employment (Asbestos) Regulations 1998 promulgated.

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

Asbestos and the New Millennium published. Prepared by Lois Syret and Ed Grootegoed on behalf of The Asbestos Association of New Zealand.

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

Increased Mesothelioma Incidence in New Zealand. T Kjellstrom, P. Smartt. NZMJ, November.

Lung function changes in asbestos-exposed workers with pleural plaques, the third study by the National Asbestos Medical Panel.

2001 *Asbestos New Zealand 2001, Lois J Syret and A.H. Grootegoed.*

Occupational Safety and Health Review of Guidelines for the Management and Removal of Asbestos.

Asbestos Exposure in New Zealand. Bill Glass, New Ethicals, August.

2003 *The New Zealand National Asbestos Registers 1992 to 2001, Lessons Learned. Presented at Asbestos Symposium Seoul National University, Korea by Bill Glass.*

The Epidemic of Asbestos-related Diseases in New Zealand. Int. J. Occup – Environ Health 10.2 June 2004 Tord Kjellstrom.

2004 *Mortality and Morbidity in Asbestos Exposure in New Zealand: The Hidden Legacy of Asbestos Exposure. NZ Med J 117; 117: 1 P Smartt.*

APPENDIX 3: MISCELLANY

Currently Available Department of Labour Publications on Asbestos

1. *Talking Asbestos* (brochure) 1987 (Archived)
<http://www.osh.govt.nz/order/catalogue/archive/talkingasbestos.pdf>
2. *Asbestos Exposure and Disease: Notes for Medical Practitioners.* (Information Bulletin) 1992.
<http://www.osh.govt.nz/order/catalogue/pdf/asbestos.pdf>
3. *Guidelines for the Management and Removal of Asbestos*, 1995.
<http://www.osh.govt.nz/order/catalogue/pdf/asbmgmt.pdf>
4. *The Epidemiology of Mesothelioma in Historical Context.* J.C. McDonald and A.D. McDonald, 1998. Special Report. (Archived)
<http://www.osh.govt.nz/order/catalogue/archive/mesothelioma.pdf>
5. *Respiratory Symptoms and Asbestos Dust Exposure.* Occupational Health Report Series: No. 2, 1997.
<http://www.osh.govt.nz/order/catalogue/pdfs/ohrs002.pdf>
6. *Asbestos: A Deadly Dust. 50 Years of Asbestos Use in New Zealand.* Reprint from Safeguard magazine, December 1991.
7. *National Asbestos Registers, Annual Reports: 1992-1993, 1994-1995, 1995-1996, 1996-1997, 1997-1998, 1992-2004, 1992-2005, 1992-2007*
<http://www.osh.govt.nz/order/catalogue/524.shtml>
8. *Respiratory Health and Silica Dust Levels in the Extractive Industry.* Occupational Health Report Series: No. 9, 2003.
<http://www.osh.govt.nz/order/catalogue/pdfs/ohrs009.pdf>
9. *Report of the Asbestos Advisory Committee to the Minister of Labour.* Department of Labour, 1991.
<http://www.osh.govt.nz/order/catalogue/archive/asbestosreport.pdf>
10. *Flooring Contractors.* Occupational Health Report Series: No. 4, 2000.
<http://www.osh.govt.nz/order/catalogue/pdfs/occ4flooring.pdf>

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A. Brant MBChB, FRACP, MBA, PhD.

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⁵ *Asthma and Allergy in New Zealand Farmers*. M. Kimbell-Dunn, L.M. Bradshaw et al. *American Journal of Industrial Medicine* 1999; 35: 51-57.

⁶ *Asthma and Other Respiratory Symptoms in New Zealand Pine Processing Sawmill Workers*. J. Douwes, D. MacLean et al. *American Journal of Industrial Medicine* 2001; 39: 608-615.

⁷ *Potroom Asthma: New Zealand Experience and Follow-Up*. T.V. O'Donnell, B. Wellford and E.D. Coleman. *American Journal of Industrial Medicine* 1989; 15: 43-49.

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⁹ *Wood Dust Exposure in Small New Zealand Woodworking Companies*. D. McBride, K. Nelse. *The Journal of Occupational Health and Safety – Australia and New Zealand*, 2004; 20: 465-472.

¹⁰ *Respiratory Symptoms and Occupational Exposures in New Zealand Plywood Mill Workers*. W. Fransman, D. MacLean et al. *American Occupational Hygiene* 2003; 42: 287-295.

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- ¹⁴ *Chronic Interstitial Lung Disease in a Welder of Galvanised Steel.* W.I. Glass, D.B. Taylor, A.M. Donoghue. *Occupational Medicine* 1994; 44: 158-160.
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- ¹⁷ *Two Year Follow-Up of Pulmonary Function Values among Welders in New Zealand.* R. Erkinjuntti-Pekkanen, T. Slater et al. *Occupational and Environmental Medicine* 1999; 56: 328-333.
- ¹⁸ *Exposure to Fumes in Typical New Zealand Welding Operations.* E. Dryson, D. Rogers. *New Zealand Medical Journal* 1991; 23: 351-258.
- ¹⁹ *Respiratory Symptoms, Across-Shift Lung Function Changes and Lifetime Exposures of Welders in New Zealand.* D. Fishwick, L. Bradshaw et al. *Scandinavian Journal of Work and Environmental Health.* 1997; 23:351-358.
- ²⁰ *Changes in Work Practice after a Respiratory Health Survey among Welders in New Zealand.* T. Slater, R. Erkinjuntti-Pekkanen, D. Fishwick, L. Bradshaw, N. Pearce, S. Cheng, H. Armstrong, D. McLean. *New Zealand Medical Journal* 2000 July 28; 113 (1114): 305-8.
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- ²² *Respiratory Health and Silica Dust Levels in the Extractive Industry in New Zealand.* W.I. Glass, D. McLean et al. *Occupational Health Report Series*, No. 9 2003; 25.
- ²³ *Asthma and Work.* Anthony Newman Taylor. *Ann. Occupational Hygiene* 2002; 46: 563-574.
- ²⁴ *Inflammatory Responses and Oxidative Stress from Metal Fume Exposure in Automobile Welders.* Luo, Jiin-Chyuan John, Hsu, Kuang-Hung, Shen, Wu-Shiun. *Journal of Occupational And Environmental Medicine* 2009;51(1): 95-103.
- ²⁵ *Occupation and COPD.* D.C. Christiani. *Occupational and Environmental Medicine* 2005; 62: 215.

²⁶ *Occupational Contribution to the Burden of Chronic Obstructive Pulmonary Disease.* John Balmes. *Journal of Occupational and Environmental Medicine* 2005; 47: 154-160.

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